Inbreeding effects on pair fecundity and population persistence

ALEXANDRE ROBERT*, DENIS COUVET and FRANÇOIS SARRAZIN

UMR 5173 MNHN-CNRS ‘Conservation des espèces restauration et suivi des populations’, Muséum National d'Histoire Naturelle, 61 rue Buffon, 75005 Paris, France

Received 10 December 2003; accepted for publication 7 December 2004

Despite strong empirical evidence of the harmful effects of inbreeding on fecundity, spontaneous recessive deleterious mutations are generally considered as acting on survival only in evolutionary models and population viability analyses. In this study, we modelled a species with separate sexes to assess the effect of selection on fecundity in small populations on the risk of extinction. We showed that the impact of inbreeding on short-term fitness changes and that population dynamics are strongly influenced by phenotypic interactions among males and females during reproduction. In particular, population persistence was found to be highly sensitive to the level at which selection acts (i.e. individual vs. pair) and to asymmetry among sexes (in terms of mutation rates and mutational effects). © 2005 The Linnean Society of London, Biological Journal of the Linnean Society, 2005, 86, 467–476.


INTRODUCTION

In small populations subject to inbreeding depression (Charlesworth & Charlesworth, 1999; Hedrick & Kalinowski, 2000) and mutation accumulation (Lynch, Conery & Bürger, 1995), rapid genetic deterioration may affect population growth through a reduction in mean vital rates (such as survival and fertility rates). The impact of such reduction on population competitive ability and on extinction risk depends both on the efficiency of selection against deleterious mutations and on the demographic cost associated with this selection. Genetic models of inbreeding generally investigate the change in mutation frequencies and relative population viability as a function of the fractional decrease in overall individual fitness caused by each single mutation. In such models, the cost of inbreeding is generally defined solely in terms of the survival of inbred young relative to non-inbred young (Feldman & Christiansen, 1984; Brook et al., 2002), as juvenile survival is the demographic component for which the negative effects of inbreeding have been the most commonly documented (Ralls, Ballou & Templeton, 1988).

However, a variety of empirical studies have uncovered the noted effects of inbreeding on fecundity. In some laboratory studies, Drosophila experience more inbreeding depression in fertility than in viability (Simmons, Sheldon & Crow, 1978). Further, numerous studies on natural populations revealed significant inbreeding depression on traits related to reproduction in both plant and animal species (reviewed by Keller & Waller, 2002). In the field of conservation biology, recent in situ studies gave evidence of the effect of inbreeding on fecundity in endangered bird (Bouzat et al., 1998; Westermeier et al., 1998) and mammal (Meagher, Penn & Potts, 2000) species.

Although inbreeding effects on fecundity may contribute substantially to fitness reduction, the extent of selection on fecundity and its impact on population viability is still difficult to assess in sexual species. In particular, in most studies on inbreeding, the attributes used as indices of reproductive success (demographic traits such as hatchability rates, egg production, or biometric measures such as offspring growth, body weight . . .) do not allow clear discrimination between the respective roles of the parental

*Corresponding author. E-mail: arobert99@yahoo.fr
phenotypes and the offspring genotype (Falconer & Mackay, 1996; Roff, 1997; Frankham, Ballou & Briscoe, 2002). Furthermore, in dioecious or gynoec-choric species, where two individuals interact for the purposes of reproduction (through physiology and/or behaviour), there is no accurate parameter with which to define the reproductive performance of a pair of individuals as a function of the phenotype of each of these individuals. Such definition is of great importance to assess fitness evolution in a specific demographic context. In the field of conservation, accurate assessments of the way in which parental genotypes influence fecundity are required in threatened animal species with complex reproductive behaviour (e.g. parental care) where both the male and female contribute to various extents to reproductive success (Margulis, 1998).

Fertility selection can be defined as a type of selection in which differences in fitness between the genotypes result from the differing abilities of mating pairs to produce offspring. Despite strong biological evidence for the existence of selection on fecundity acting as a property of mating pairs in various taxa (De Nettancourt, 1977; Ober et al., 1983; Schaap et al., 1984), relatively little work has been done on fertility selection (but see Bodmer, 1965; Pollak, 1978; Feldman, Christiansen & Liberman, 1983; Clark & Feldman, 1986; Vekemans, Schierup & Christiansen, 1998) and these aspects are generally neglected in models of inbreeding and population viability analyses. Some models of population viability (especially in the field of conservation; see Mills & Smouse, 1994) have taken into consideration the deleterious effect of inbreeding on reproduction, and recent versions of generic packages for population viability analysis such as VORTEX (Lacy, 2000) allow one to consider such effects. However, most models focus on overall population properties (such as the mean inbreeding coefficient) or individual characteristics (such as individual inbreeding coefficients) to assess the effect of genetic deterioration, rather than considering selection depending on the association between male and female genotypes. In this study, we used a modelling approach integrating demographic and genetic processes, and focused on species with separate sexes, to assess the extent of selection on fecundity in small inbred populations and its effect on the risk of extinction. We showed that phenotypic interaction among males and females (at the pair level) during reproduction is an essential factor to consider for assessing the impact of inbreeding on short-term fitness changes and population dynamics. In particular, we found population persistence is highly sensitive to (1) the relationship between the reproductive performance of a given pair of individuals and the intrinsic individual qualities; (2) the asymmetry among sexes (in terms of mutation rates and mutational effects).

**METHODS**

**Population Dynamics**

We used a two-sex individual-based model approach to consider a dioecious semielparous life-cycle (non-overlapping generations). In each time-step, males and females were drawn randomly and paired according to their social mating system (monogamy, polygyny or polyandry). In the case of monogamy, males and females were paired one to one and each individual could mate only once. In the case of polygyny, males could mate several times and the number of females per male was random and unrestricted. A symmetrical model was assumed for polyandry. All adults died after reproduction, that is, fecundity was the only parameter of fitness. The expected basic fecundity per pair (i.e. in the absence of inbreeding effects) was \( P_0 \). However, due to the effects of inbreeding that caused some differences among individual and pair qualities, the expected fecundity varied from one pair to the other. Hence, a specific fecundity \( P_{ij} \) was attributed to each pair \((i, j)\). The computation of \( P_{ij} \) is described below.

Demographic stochasticity resulted first from the drawing of the number of offspring of each reproducing pair \((i, j)\) from a Poisson distribution of expected value \( P_0 \) and second from the random determination of the sex of each individual according to a 1 : 1 sex ratio.

Initially, \( N_0 \) individuals were present in the population. In each year, population size was truncated to the carrying capacity \( K \), independently of the genotypes of individuals in order to keep constant selective coefficients. An alternative method for density dependence was considered by assuming a linear decrease of fecundity with increasing population size, where the mean individual fecundity at time \( t \) is given by \( P_{ij} = 2 + (P_{max} - 2)(1 - N_0/K) \), \( P_{max} \) being the basic pair fecundity in the absence of density dependence. As no qualitative effect of the system of density dependence was detected, results are only presented for the truncation method.

**Genetic Characteristics**

The genome of each individual was explicitly represented as a series of \( L \) different diploid loci. We considered two alleles per locus, \( A \) being the wild-type allele and \( a \) an unconditionally deleterious partially recessive allele. The relative fitnesses of the \( AA \), \( Aa \) and \( aa \) genotypes at a given locus were 1, 1 \( - hs \) and 1 \( - s \), respectively, where \( s \) is the selection coefficient and \( h \) is the dominance coefficient of \( a \).

The initial frequency of deleterious alleles was \( q_0 \) and new deleterious mutations stochastically occurred.
in every generation, with an average of $U$ genomic mutations per generation (Poisson distribution), corresponding to a per locus rate $u$, with $U = 2Lu$. We assumed that each new deleterious mutation that occurred in the population was unique. Reverse mutation was not considered.

The effect of sex-biased mutation rates on population dynamics was assessed by assuming equivalent mutational effects for both sexes, with sex-specific probabilities of occurrence of new mutations $U_{\text{male}}$ and $U_{\text{female}}$, with $U_{\text{male}} = 2U/(2 - \text{asym})$ and $U_{\text{female}} = 2U - U_{\text{male}}$, asym being a coefficient quantifying the degree of asymmetry between male and female mutation rates for a constant mean mutation rate $U$. We assumed in all cases that $U_{\text{male}}$ was higher than or equal to $U_{\text{female}}$ (see Discussion). Values of genetic parameters $s$, $h$, and $U$ used in most figures corresponded to average values commonly assumed for nearly additive mildly deleterious mutations (Drake et al., 1998; Lynch et al., 1999). We assumed multiplicative interactions for fitness and free recombination of all loci.

**RELATIONSHIP BETWEEN PAIR GENOTYPE AND PAIR FECUNDITY**

As mentioned previously, in the absence of an inbreeding effect, the expected basic fecundity of each pair is $P$. However, as different mutation loads may be carried by the different males and females that pair, we investigated different scenarios with respect to the interaction of parental phenotypes and its effect on pair fecundity.

1. **Computation of $P_i$ as a function of the global individual qualities $w_i$ and $w_j$**
   
   We assumed that the quality of an individual reflected simply its genetic load. The respective numbers $\text{het}$ and $\text{hom}$ of heterozygous and homozygous $a$ mutations present within the $L$ loci of the individual $i$ determined its relative individual quality ($w_i$) given by $w_i = (1 - hs)^{\text{het}} \cdot (1 - s)^{\text{hom}}$. The fecundity of the pair $(i, j)$ was then given by $P_{ij} = P \cdot f(w_i, w_j)$, $f$ being a simple function of $w_i$ and $w_j$ (multiplicativity, additivity, arithmetic mean, geometric mean, or harmonic mean).

2. **Computation of $P_i$ as a function of the genotypes of $i$ and $j$ at each locus**
   
   The fecundity of the pair $(i, j)$ was given by $P_{ij} = Pw_iw_j$ with $w_i = (1 - hs)^{\text{het}} \cdot (1 - s)^{\text{hom}}$ where $\text{Het}$ and $\text{Hom}$ are the number of loci contributing to a fractional reduction of $hs$ and $s$, respectively. These numbers $\text{Het}$ and $\text{Hom}$ were simply counted by the program, for each pair $(i, j)$, as functions of the intrapair dominance coefficient $h$, the intrapair dominance coefficient $H$ and the respective genotypes of parents over the $L$ loci considered, using the rules given in Table 1 for intrapair complete recessivity ($H = 0$) and complete dominance ($H = 1$).

<table>
<thead>
<tr>
<th>Genotype of parent $i$</th>
<th>Genotype of parent $j$</th>
<th>$H = 0$</th>
<th>$H = 1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>AA</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Aa</td>
<td>AA</td>
<td>1</td>
<td>$1 - hs$</td>
</tr>
<tr>
<td>aa</td>
<td>AA</td>
<td>1</td>
<td>$1 - s$</td>
</tr>
<tr>
<td>Aa</td>
<td>Aa</td>
<td>$1 - hs$</td>
<td>$1 - hs$</td>
</tr>
<tr>
<td>aa</td>
<td>Aa</td>
<td>$1 - s$</td>
<td>$1 - s$</td>
</tr>
<tr>
<td>aa</td>
<td>aa</td>
<td>$1 - s$</td>
<td>$1 - s$</td>
</tr>
</tbody>
</table>

3. **Computation of $P_i$ with sex-biased mutational effects**

When mutations were assumed to affect one sex only, the relative intrinsic quality of an individual $i$ of the sex affected by mutations was given by $w_i = (1 - hs)^{\text{het}} \cdot (1 - s)^{\text{hom}}$. The relative qualities of all individuals of the other sex were assumed to be equal to one.

In the case of sexually antagonistic mutational effects, the relative intrinsic quality of an individual $i$ of the sex negatively affected by mutations was computed as $w_i = (1 - hs)^{\text{het}} \cdot (1 - s)^{\text{hom}}$ and the relative intrinsic quality of an individual $j$ of the sex positively affected by mutations was computed as $w_j = (1 - hs)^{\text{het}} \cdot (1 - s)^{\text{hom}}$.

In both cases, the expected fecundity of the pair $(i, j)$ was given by $w_{ij} = Pw_iw_j$.

In all cases, population and individual fitnesses were expressed relative to the average population fitness at time 0. Extinction occurred when population size was equal to zero. Population viability was investigated in different scenarios by using Monte Carlo simulations in which 1000 population trajectories were drawn.

**RESULTS**

**OVERALL INDIVIDUAL QUALITIES AND PAIR REPRODUCTIVE PERFORMANCE**

Figure 1 presents a comparison of mean times to extinction ($t_{ext}$) obtained using various functions to compute the relative reproductive performance of pairs. Here, individuals were assumed to interact with each other as a function of their ‘overall’ quality (i.e. the relative fecundity $w_{ij}$ of the pair $(i, j)$ was simply a function of the genetic loads $w_i$ and $(w_j)$. A strong interaction between the function used and population size was observed, with $t_{ext}$ being much more sensitive.
to the variation in population size for ‘additivity’ and ‘multiplicativity’ than it was for the ‘mean’ functions.

**INTRAPAIR INTERACTIONS OCCURRING AT THE LOCUS LEVEL: COMPARISON BETWEEN DOMINANCE AND RECESSIVITY**

Here we examined more complex relationships among parental genetic loads, in which the contribution of a given locus $k$ to the relative quality of the pair $(i,j)$ depended on the interaction among parental genotypes at this particular locus ($w_{ik}$ and $w_{jk}$). The parameter $H$ was used to define a recessivity/dominance relationship between the contributions of the genotypes of both parents at each locus, as a function of $s$ and $h$. In the case of dominance, a deleterious mutation at the locus $k$ contributed to the reduction of the fecundity of the pair if it was carried by one or both parents. In the case of recessivity, a deleterious mutation affected pair fecundity only if present in both parents (see Table 1).

For a given value of $h$, Figure 2 indicates that short-term extinction risk (i.e. in the scenario in which mutation rates were high) was reduced when parental genotypes interacted recessively, compared with dominantly. On the contrary, when extinction occurred on longer time scales on average (i.e. when considering low mutation rates), dominance was advantageous relative to recessivity. When examining changes in population relative fitness, recessivity (both at intra- and interindividual levels) was always beneficial in the short run and became more detrimental than dominance in the long run. In these examples, populations with different mutation rates exhibited different extinction patterns because the mutation rate primarily determined the time to extinction. Similar differences were obtained by comparing species with different basic growth rates for the same mutation rate (data not shown). In such cases, species with a high reproductive potential became extinct in the long run and dominance was advantageous, while slow
Growing species always became extinct rapidly and recessivity was advantageous.

**Effects of Male–Female Asymmetry**

In order to assess the impact of asymmetry in mutational effects, we compared situations in which deleterious mutations were neutral when carried by males and deleterious when carried by females, with situations in which mutations had the same deleterious effect on pair fecundity in males and females (mutational effects were assumed to interact multiplicatively at the pair level). As for the recessivity/dominance relationship, short- and long-term extinction probabilities were antagonistically affected, with the former case being advantageous in the short run and detrimental in the long run. Species with low reproductive potential were therefore more affected if mutations acted on both sexes while the reverse was true in fast-growing species (Fig. 3). Similar patterns were obtained by comparing populations with different mutation rates and equivalent growth rates (data not shown): mutations acting on both sexes were more deleterious for high mutation rates leading to short-term extinction, and more beneficial for low mutation rates leading to longer-term extinction.

The effect of sexually antagonistic selection was investigated by considering that each homozygous mutation had a negative effect $s$ in one sex (i.e. inducing a reduction in pair fecundity in a proportion $s$) and a positive effect $s'$ (inducing an increase in pair fecundity), with $s' \leq s$. The effect of $s'$ on extinction is presented in Figure 4, for a constant value of $s$, indicating that mutations had a maximal impact for intermediate values of $s/s'$ except when population size was very low, for which the maximal impact was obtained when $s/s'$ tended towards infinity (see Discussion).

No qualitative difference between monogamous and polygamous mating systems was detected, so results are presented for monogamy only in Figures 1, 2, 3 and 4.

The effect of sex-biased mutation rates on population dynamics was examined for mutations with large additive effects. In all cases, monogamy led to more rapid extinction than did polygamy for demographic reasons (Fig. 5A; Legendre et al., 1999). Furthermore, our analysis showed that the degree of asymmetry between male and female mutation rates influenced the mean time to extinction in the case of polygamy. If the mutation rate was assumed to be higher in males than in females, an increasing degree of asymmetry led to a reduction of population viability in the case of polygyny, whereas it led to an increase of viability in the case of polyandry. This impact was due to an effect of the sex-specific mutation rates on the variance in reproductive output of the population, resulting in either increased or decreased extinction risk through demographic stochasticity. It occurred only in species in which the variance in the contribution of individuals to the next generation was unequal in the two sexes. In the case of monogamy, the degree of asymmetry between male and female mutation rates had no impact on population viability (see Discussion).

As the impact of the degree of asymmetry in mutation rates was highly dependent on mutation parameters, a comparison between completely symmetrical ($\text{asym} = 0$) and completely asymmetrical ($\text{asym} = 1$)

![Figure 3](image_url)  
**Figure 3.** Effect of male–female asymmetries in mutational effects on extinction rates. $N_0 = K = 25$; $q_0 = 0.0$; $s = 0.05$; $U = 0.1$; $L = 1000$; $\text{asym} = 0$. Abbreviations as in Fig. 1.

![Figure 4](image_url)  
**Figure 4.** Impact of sexually antagonistic mutation effect on the mean time to extinction. $s$ is the deleterious effect of homozygous mutations in females and is constant in all situations; $s'$ is the beneficial effect of homozygous mutations in males. $N_0 = K$; $P = 9$; $q_0 = 0.0$; $s = 0.05$; $h = 0.35$; $U = 0.1$; $L = 1000$; $\text{asym} = 0$. Abbreviations as in Fig. 1.
CONTRIBUTION OF PARENTAL INBREEDING TO REPRODUCTIVE SUCCESS

Individuals are generally assumed to interact as a function of their ‘overall’ quality, especially when the total reproductive fitness is the criterion used to characterize individuals. In Figure 1, the reproductive performance of pairs only depend on the global relative performance of each parent, uncovering the importance of the male–female fitness interaction to assess short- and long-term population persistence. As the ‘mean’ functions (geometric, arithmetic, harmonic) reduce the effective selection coefficient of each mutation, selection is less efficient, leading to more mutation accumulation and increased long-term extinction risk relative to the ‘multiplicative’ and ‘additive’ scenarios. Yet, due to a weaker demographic cost due to inbreeding, our results indicate that (1) deleterious mutations affecting reproduction may reduce population viability considerably, and (2) the quantitative impact of these processes on population dynamics varies substantially with the assumptions made concerning the way in which parental phenotypes interact and the degree of asymmetry between sexes (in terms of the probability of occurrence and the effect of mutations). This suggests that such interactions should be considered explicitly in population models to avoid underestimations of the impact of inbreeding on evolutionary and demographic changes.

DISCUSSION

Although theoretical fertility selection models exist (Bodmer, 1965; Pollak, 1978; Feldman et al., 1983; Clark & Feldman, 1986), this approach is rarely used to assess short-term population persistence in relation to inbreeding. Our study focussed on the interaction between inbreeding and reproduction, and its consequences on population dynamics, rather than on inbreeding itself. Our results indicate that (1) deleterious mutations affecting reproduction may reduce population viability considerably, and (2) the quantitative impact of these processes on population dynamics varies substantially with the assumptions made concerning the way in which parental phenotypes interact and the degree of asymmetry between sexes (in terms of the probability of occurrence and the effect of mutations). This suggests that such interactions should be considered explicitly in population models to avoid underestimations of the impact of inbreeding on evolutionary and demographic changes.
Sex biases in the effects of inbreeding

Different investments in reproduction may induce different sensitivities to inbreeding in males and females, in terms of fecundity decrease and offspring viability (Margulis, 1998). In humans, recent results suggest that in inbred populations, the reproductive output of a given pair depends primarily on the inbreeding coefficient of the woman rather than the degree of inbreeding of the man or the degree of relatedness between spouses (Ober, Hyslop & Hauck, 1999). This trend is confirmed by similar results in rodents (Margulis & Altmann, 1997).

At the level of the locus, some mutations may affect unconditionally the reproductive performance in male and female (this may be expected for mutations affecting the general vigour of individuals, or some reproductive behaviour), whereas some others may decrease specifically the reproductive performance in one sex only by altering sex-specific functions. Various genetic or physiological mechanisms can contribute to induce sex-biased mutational effects. For instance, deleterious mutations at sex-linked loci (especially recessive or partially recessive ones) are strongly expressed in the hemizygous state in the heterogamic sex and are thus subject to stronger counter-selection than are deleterious mutations in autosomal genes (Haldane, 1927). Furthermore, numerous ecological studies have reported that some traits (such as coloration and behaviour) can be selected discordantly in males and females (Endler, 1980; Forsman, 1995) and there is evidence that the genomes contain a significant frequency of sexually antagonistic alleles (Rice, 1992, 1998).

Focusing on autosomal mutations only, our results suggest that, if mutations have antagonistic effects on male and female fecundity (with a deleterious effect \( s \) on one sex and a beneficial effect \( s' \) on the other sex), the mean time to extinction is related mainly to the ratio \( s/s' \) (Fig. 4). For \( s/s'=1 \), mutations tend to accumulate rapidly but have effectively no impact on population dynamics when fixed. When \( s/s' \) is high, the global effect of each mutation on population dynamics is maximal, but selection acts to impede mutation accumulation. Mutations have a maximal impact for intermediate values of \( s/s' \) (see Fig. 4 for \( N = 100 \)) for which the fixation load is maximum (the value of \( s/s' \) that minimizes \( t_{\text{eq}} \) is related to the effective size of the population \( N_e \). It is expected to be approximately equal to 0.8/\( N_e \); Lande 1994). If effective population size is low relative to \( s \) (i.e. \( N_e < 0.8/s \); see Fig. 4 for \( N = 25 \)), mutation accumulation has a maximal impact when \( s' = 0 \).

**Sex biases in mutation rates**

The evidence for sex-biased mutation rates is not new (Haldane, 1947), but it has been strengthened in recent times (Crow, 1997; Drake et al., 1998) in studies involving various taxa (especially *Mus musculus* and *Homo sapiens*). Although the causes of this bias vary depending on the type of mutation considered (nucleotide substitution, gain, loss), the bias is attributed largely to greater numbers of germline cell divisions in males. The mutation rate of evolutionary importance is of course the average over the two sexes, but any asymmetry among male and female may have some short-term demographic consequences. Our results suggest that the impact of male-biased mutation rates on extinction can be substantial for mutations of large effect acting on a short time-scale (i.e. with high \( sh \)).

The new mutations occurring in the adult germlines have no effect on the reproductive output of these adults, but will affect the fecundity of their offspring. In our program, any new mutation is unique, all mutations arise independently from each other, and sex assignment is random. Therefore, the reported effect of mutation rate asymmetry on extinction is not an artefact of the model structure; it is due simply to the effect of the asymmetry on the intergenerational variance in the mean reproductive potential of individuals (regardless of their sex) due to newly arisen mutations. At a given generation, the variance in the number of new mutations \( \text{mut} \) transmitted to the offspring can be written as (see Appendix for details):

\[ \text{var(mut)} = 0.25(N(U_m + U_f) + \text{[male covariance term]} + \text{[female covariance term]}). \]

In this expression, the covariance terms represent the non-independence in the numbers of new mutations transmitted during distinct reproduction events (for example, if a male mates with several females at a given generation, the numbers of new mutations transmitted during these reproductive events are not independent, so the male covariance term is higher than zero).

In the case of monogamy, each individual mates once only, so the covariance terms are zero for both males and females, and \[ \text{var(mut)} = 0.25N(U_m + U_f) = 0.5NU. \] Consequently, for a given value of the mean mutation rate \( U \), \( \text{var(mut)} \) is not affected by the asymmetry between \( U_m \) and \( U_f \).

In the case of polygyny, the female covariance term is always zero and the male covariance term is equal to or higher than zero because each male can mate more than once. Because the male covariance term is an increasing function of \( U_m \), \( \text{var(mut)} \) increases with \( U_m \) for a given value of \( U \). The symmetrical pattern occurs in the case of polyandry, in which \( \text{var(mut)} \) decreases with an increase of \( U_m \) for a given value of \( U \).

This effect of the degree of asymmetry among sex mutation rates on the variance in the overall number of new mutations transmitted to the offspring contributes to the intergenerational (or interannual) variance in population growth. An increase in this variance elevates the risk of stochastic extinction, whereas a reduction in this variance has the opposite effect (Lande, 2002).

Despite the existence of theoretical work on fertility selection (Bodmer, 1965; Pollak, 1978; Feldman et al., 1983; Clark & Feldman, 1986) and on the impact of genetic deterioration on the viability of small populations (Hedrick, 1994; Lande, 1994; Lynch et al., 1995; Charlesworth & Charlesworth, 1999), the risk of extinction caused by deleterious mutations affecting reproduction in a specific context is still difficult to assess. In some species, reproductive compensation mechanisms (see Ober et al., 1999) may have negative effects on the purging of mutations affecting fecundity (Overall, Ahmad & Nichols, 2002), leading to a more rapid accumulation of mutations of mild effect in small populations, and to an increase in the frequency of severe genetic disorders. Furthermore, mating behaviour, which may be extremely variable from one species to another, may have a strong impact on risk of extinction (Moller, 2000; Bessa-Gomes et al., 2003), and may interact strongly with genetic processes. Hence, further multidisciplinary and specific work is required in this field of research. In particular, incorporating additional constraints into our model, such as assortative mating (Kondrashov & Shpak, 1998) or mate retention depending on breeding success (Dubois & Cézilly, 2002) may be valuable in the assessment of the impact of breeding behaviour on the viability of small inbred populations.

ACKNOWLEDGEMENTS

We are grateful to Isabelle Olivieri for helpful suggestions during the preparation of this paper.

REFERENCES


REFERENCES


© 2005 The Linnean Society of London, Biological Journal of the Linnean Society, 2005, 86, 467–476


APPENDIX

At a given generation, the overall number of new mutations transmitted to the offspring (mut) is equal to half the sum of the number of new mutations occurring in males that effectively reproduce plus half the sum of the number of new mutations occurring in females that effectively reproduce.

\[
mut = 0.5 \left( \sum M_i(x) + \sum M_j(U_j) \right)
\]

with \(N\) being the total number of mating events, \(M_i(x)\) being the number of mutations occurring in the individual \(i\) with \(x\) expected new mutations per generation.

(Poisson drawing), and \( U_m \) and \( U_f \) being, respectively, the male and female mutation rates.

The variance in \( \text{mut} \) is given by

\[
\text{var}(\text{mut}) = \text{var}\left(0.5\left[\sum_i^N [M_i(U_m)] + \sum_j^N [M_j(U_f)]\right]\right)
\]

\[
= 0.25\left[\text{var}\left(\sum_i^N [M_i(U_m)]\right) + \text{var}\left(\sum_j^N [M_j(U_f)]\right)\right]
\]

\[
\text{var}(\text{mut}) = 0.25\left[\sum_i^N [\text{var}(M_i(U_m))] + \sum_{ij}^N [\text{cov}(M_i(U_m), M_j(U_f))] + \sum_k^N [\text{cov}(M_k(U_m), M_k(U_f))]\right].
\]

This expression can be written:

\[
\text{var}(\text{mut}) = 0.25(NU_m + \text{male covariance term} + NU_f + \text{female covariance term}).
\]

Thus,

\[
\text{var}(\text{mut}) = 0.25(N(U_m + U_f) + \text{male covariance term} + \text{female covariance term}).
\]