Gerdina de Jong (Ed.)

Population Genetics and Evolution

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Quantitative Genetic Models for Parthenogenetic Species

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1 Introduction

Animal and plant breeders have successfully applied quantitative genetic models (Bulmer 1980; Falconer 1981) to describe the phenotypic selection of metric characters. The extension of these concepts to problems in evolutionary ecology (Lande 1976a,b, 1982) gives general dynamic expressions for the phenotypic evolution of polygenic characters, though at the expense of making confining assumptions such as infinite population size, additive loci, and restrictions on the form of selection functions. Therefore, to connect ecology with genetics, the framework of quantitative genetics is most suitable for questions where (1) the underlying genetic systems are so complex that we cannot hope to understand them locus by locus and, for that reason, we strive to obtain models that give reasonably good approximation, and (2) where the results of the theory seem to be at least qualitatively correct in spite of the unavoidable inherent simplifying assumptions. Most of the possible errors resulting from such approximation to reality may be irrelevant in comparative studies where the emphasis is on qualitative behaviour.

The need for genetic theory in ecology is self-evident when one tries to evaluate the performance of parthenogenetic species relative to sexual ones to gain insight into the complicated reproductive strategies of the freshwater zooplankton species with long periods of parthenogenetic reproduction but sexually produced resting eggs. Here, I will demonstrate the usefulness of quantitative genetic models for investigating such problems as the consequences of obligate and cyclical parthenogenesis for the evolutionary potential of species and for the evolution of specialist versus generalist strategies.

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2 Phenotypic Evolution of a Single Character

Let us first consider a single quantitative character of a diploid organism and assume that this character is determined by n diploid loci and that its genotypic value can be written as a summation over purely additive contributions of the 2 n allelic effects. (This assumption of additivity is necessary only for cyclical parthenogenesis in order to make the decomposition of genetic variance into a hidden and expressed component tractable; for obligate parthenogenetic species the genotypic value comprises all additive and non-additive contributions.) The per generation change in the genotype distribution of a population can be estimated when the non-genetic contributions to the phenotypic variance (e.g. developmental noise), the genotypic variance and the shape of the fitness function are known. Sexual and asexual populations differ in the realized genetic variance. In the case of sexual reproduction, Lande (1976a,b) calculated an equilibrium level of genetic variance maintained by a balance between input via mutation and recombination and output by selection. Lynch and Gabriel (1983) derived analogous formulas for phenotypic evolution under parthenogenesis and found the equilibrium to be:

$$\hat{V}_g = \frac{1}{2}(V_m + \sqrt{V_m (V_m + 4(V_e + V_w))}) \tag{1}$$

with V_w as the variance of the fitness function assumed to be Gaussian, V_e as the variance in developmental noise, and V_m as the per generation input of genetic variance. The number of loci is implicit in V_m, where the total input of mutation is summed over loci and alleles. The corresponding formula for sexual reproduction has identical structure but V_m is always multiplied by 2 n. Thus, the number of loci appears explicitly also (rewritten from Lande 1976):

$$\hat{V}_g = \frac{1}{2}(2\,n\,V_m + \sqrt{2\,n\,V_m (2\,n\,V_m + 4(V_e + V_w))}) \ . \tag{2}$$

An essential result of the models is that these equilibria (Eqs. 1 and 2) are independent of the optimal genotypic value so that a population under directional selection can approach its equilibrium level of genetic variance even if it is far from the optimal genotype. Lynch and Gabriel (1983) have shown that for most parthenogens this equilibrium level will be reached within a few hundred generations and have argued that the maintenance of this genetic variability is an important mechanism which renders rates of phenotypic evolution the same order of magnitude as bisexual organisms. For example, a change in the mean genotypic value as large as 5 phenotypic standard deviations is feasible within 100 generations. In general, higher genetic variability from sexual reproduction implies a more rapid evolution, but only if mutation rate per locus and the sensitivity of phenotypic development to environmental effects are equal for sexual and parthenogenetic reproduction. There are arguments suggesting higher mutation rates and lower environmental sensitivity in parthenogenetic species (Lynch 1985), but experimental evidence is lacking.

This study of phenotypic evolution under parthenogenesis may hold as an example of how methods of quantitative genetics can be applied to ecological questions and lead to new insights. Lynch and Gabriel (1983) have shown that the common assertion, that obligate parthenogenesis is an evolutionary dead end, is suspect.

3 Consequences of Cyclical Parthenogenesis

From the same study some unexpected consequences for cyclical parthenogenesis also follow. Under parthenogenesis there is a continuous accumulation of hidden genetic variance which can be quantified by a calculation of the covariances between allelic effects. According to Lynch and Gabriel (1983), up to 75% of this hidden genetic variance can be converted into expressed genetic variance within one generation after sex; the minimum value is 50% since covariances between all genes on different chromosomes are immediately broken up. Therefore, massive amounts of hidden genetic variance may be released when a cyclical parthenogenetic species interrupts long periods of unisexuality by a bout of recombination. Dramatic responses to selection may then occur within a few generations. The mean value of the genotype distribution may jump several phenotypic standard deviations in a single generation after sex. In the long term, the rate of evolution under cyclical parthenogenesis is essentially the same for frequencies of sexual reproduction from once per generation to once per 100 generations. Figure 1 shows the changes of genotype distributions during evolution under bisexuality (Fig. 1a) and cyclical parthenogenesis (Fig. 1b,c). For cyclical parthenogens, the genotype distribution after each bout of sexual recombination is broader than under continuous sexual reproduction due to the expression of hidden

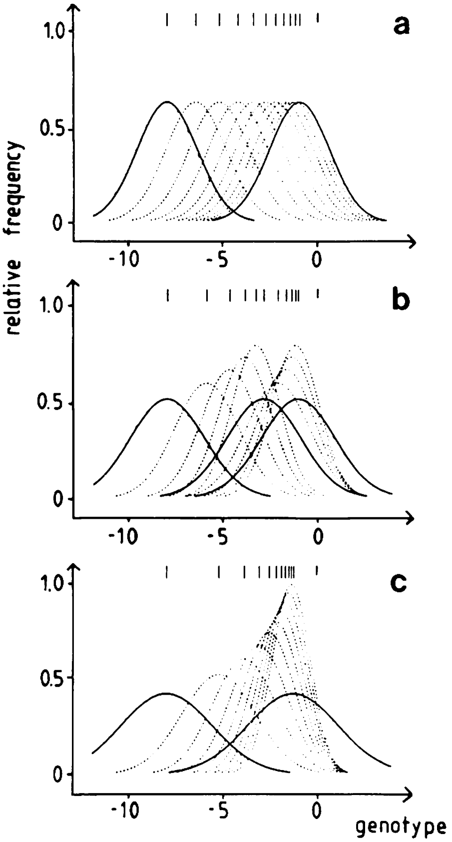

Fig. 1a–c. Comparison of the change in the genotype distribution during evolution of sexual and cyclical parthenogenetic species. **a** Sexual reproduction each generation. **b** Sexual reproduction every five generations. **c** Sexual reproduction every ten generations. Mean phenotype values are marked on the x-axis (optimal value at 0)

genetic variance. This may explain why resting eggs are produced sexually: if the optimal genotype of the next season is unpredictable, a broad genotype distribution in resting eggs provides the population with enough variability to track fluctuations in the environmental conditions. The nearly optimal clones can use the reproductive advantage of parthenogenesis in a two-fold way during times of more constant environment: they grow faster because they do not produce males, and they avoid the production of less fit offspring as it would necessarily happen with recombination. Therefore, cyclical parthenogenesis may guarantee high performance.

4 Muller's Ratchet

An argument often made about parthenogenesis is that deleterious mutations would accumulate by "Muller's Ratchet" (Muller 1964). Felsenstein (1974) and Maynard Smith (1978) evaluated Muller's verbal arguments with detailed genetic model studies and gave precise conditions under which Muller's ratchet can operate. If mutations reduce fitness only slightly, and if the effective population size is small, there is a chance that all individuals in the optimal class (with fewest mutations) die or fail to reproduce. Gabriel and Wagner (1988) constructed a phenotypic model as an alalogue to the studies of Felsenstein and Maynard Smith to see if Muller's arguments hold when fitness is determined by several independent quantitative characters, each with a polygenic basis. This quantitative genetic model also gives conditions for the fitness and the effective population size under which Muller's ratchet operates. Without mutations, the optimal genotype class gets lost by random drift and Muller's ratchet advances, always deleting the class with the fewest mutations. In this model each mutation decreases the fitness with high probability, but, in contrast to Felsenstein and Maynard Smith, back-mutations are not totally ignored. The surprising result is that even under a continuously operating Muller's ratchet, the mutational load of the population is limited: as soon as the population's mean fitness has decreased to a certain value, the accumulation of further mutations no longer has an effect on the population mean fitness. The main reason for the difference between the phenotypic model and Felsenstein's and Maynard Smith's models is that in the former, back-mutations become more frequent with increasing distance to the optimum due to the geometry of the multidimensional fitness function. This provides a limit for the decrease of fitness comparable to the expected mean fitness of a sexual population under random drift as approximated by Lande (1976b). The model of Gabriel and Wagner (1988) shows that Muller's ratchet may hinder the survival of the optimal genotype in a parthenogenetic species, but, nevertheless, the population can stay in a stochastic equilibrium near the optimum. This can, therefore, be interpreted as a phenotypic analogue for a transition from direct replication to stochastic replication in polynucleotides (Swetina and Schuster 1982). Under high genomic mutation rates, stochastic replication may have similar effects as recombination. In this case, it seems to be of only minor importance for performance whether reproduction is sexual or parthenogenetic.

5 Optimal Breadth of Adaptation

Another important factor for the performance of a species is its ability to adapt to changing environmental conditions. A first step towards a quantitative genetic theory for the evolution of the breadth of adaptation has been taken by Lynch and Gabriel (1987a,b) who have developed a theory of the expression of the response of individual genotypes to density-independent gradients of environmental factors. The fitness function had often been treated as fixed, and therefore, independent of evolution, but in this approach the fitness function is treated as a performance curve (tolerance curve or norm of reaction) which itself can change during the evolutionary process. The model has incorporated the following environmental variabilities: spatial variance ($V_{\varphi s}$), within generation temporal variance ($V_{\varphi tw}$), and between generation temporal variance ($V_{\varphi tb}$). Breadth ($\sqrt{g_2}$) and optimal setting (g_1) of the maximum of the performance curve of each individual are treated as quantitative characters on a scale of an environmental variable (φ) so that the fitness of an individual is determined by

$$w(z_1, z_2 \mid \varphi) = \frac{1}{\sqrt{2\pi z_2}} \exp\left\{-\frac{(z_1-\varphi)^2}{2z_2}\right\}, \tag{3}$$

where z_1 and z_2 are the phenotypic values corresponding to the genotypic values g_1 and g_2. The difference between genotypic and phenotypic values may, in this context, be called developmental noise. It is trivial to see that the optimal g_1 is identical with the mean value of the environment, but the question is how to predict the optimal genotypic value of breadth of adaptation dependent on given variabilities of the environment. This can be done by optimization of the expected geometric mean fitness (Lynch and Gabriel 1987b). After a scale transformation so that the mean value of the environmental state φ is 0, an approximation formula for the expected geometric mean fitness is

$$w(g_1, g_2) \cong \frac{1}{\sqrt{2\pi(V+V_{\varphi s})}} \exp\left\{-\frac{1}{2}\left[\frac{g_1^2 + V_{\varphi tb} + V_{\varphi tw}\ 1 + \frac{V_{\varphi s}}{V}}{V+V_{\varphi s}}\right]\right\} \tag{4}$$

with

$$V = g_2\left(1-\frac{1}{K}\right) + V_{e1}$$

$$K = \frac{g_2(g_2+V_{e1})}{V_{e2}} + 2$$

and with V_{e1} and V_{e2} as the variances of the developmental noise on g_1 and g_2.

The parameters of this theory can be extracted from experimental data by a maximum likelihood procedure as demonstrated by Gabriel (1987). The theory confirms what is intuitively expected: temporal variability of the environment selects for more broadly adapted genotypes, and the within-generation heterogeneity is more important than the between generation component. But, on the other hand, the theory produces results which are at least at first counterintuitive. Spatial heterogeneity selects for more broadly adapted genotypes only when operating in conjunction with certain patterns of temporal variance: if the within-generation temporal variance is much less than the between-generation temporal variance, spatial heterogeneity can select for a *reduction* of the breadth of adaptation. The formulas of Lynch and Gabriel are results of an optimality approach for infinite populations and do not tell how fast evolution takes place and how much better sexual populations adapt than parthenogenetic or cyclic parthenogenetic populations. Below, I present some simulation results with mutation and selection on finite populations. I did not find differences in the optimal breadth of adaptation between sexual and parthenogenetic species.

6 Evolution of Generalism and Specialism

The simulations start with an effective number of parents for which the allelic contributions to the genetic values of the individuals' performance curve (as the setting of the maximum and as the variance) are stored. The first step is the production of offspring, by free recombination in the case of sexual reproduction. Each offspring carries a new mutation according to the genomic mutation rate. The phenotypic values are calculated by adding developmental noise to the genetic values. Individual fitness is calculated from these phenotypic values. The next generation of parents is created by applying viability selection, which is a function of the environmental state for each individual (see Eq. 3). From the surviving parents the new effective population (N_e = 250) is chosen randomly. Presented here are simulations performed with the following parameters: 20 diploid loci per character; mutation probability per genome is 0.1; average widths (square root of the variance) of mutational effect are 0.1 for g_1 and 1.0 for g_2 on a relative scale so that the developmental noise on g_1 is 1; as shown by Lynch and Gabriel (1987) the effect of developmental noise on g_2 is of minor importance, and therefore, is set at zero in this study. The mutational effects on the genotypic values g_1 are assumed to be normally distributed around its actual values, and for g_2 to be according to a beta distribution of the second kind (see Lynch and Gabriel 1987a).

Figure 2 shows the evolution of sexual and parthenogenetic populations after a drastic reduction (by a factor of 100) of the between-generation temporal variance of the environment so that a smaller breadth of adaptation is optimal (g_1 is assumed here to be at its optimum). Both sexual and parthenogenetic populations converge to the new optimal value but the sexual population is much faster due to the higher genetic variance under sexual reproduction.

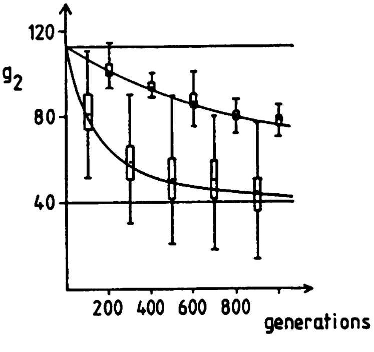

Fig. 2. Population response to a change in the optimal breadth of adaptation. *Upper curve:* evolution under parthenogenesis; *lower curve:* under sexual reproduction. The *boxes* give the quartiles, *vertical bars* give the total range of results. *Horizontal lines* are the optimal values

When these and similar simulations are performed until convergence has been obtained, it is possible to estimate the equilibrium levels of genetic variances. These values can be approximately compared with the predictions of Eqs. (1) and (2) by the following simple calculation. To reduce the problem to the estimation of genetic variance under phenotypic evolution of a single character, one allows only one character to vary. If one keeps the breadth of adaptation ($\sqrt{g_2}$) fixed, the fitness function varies with the optimal value (g_1) in such a way that $g_2 + V_{\varphi s}$ corresponds to the variance of the fitness function V_w in Eqs. (1) and (2). [With $V_{e2} = 0$, it follows from Eq. (3) that $w(g_1, g_2)$ is proportional to $\exp(-g_1^2/2(g_2 + V_{e1} + V_{\varphi s}))$; according to Lynch and Gabriel (1983) the corresponding genic fitness $w(g)$ for a single character is proportional to $\exp(-g^2/2(V_e+V_w))$.] To make these comparisons, simulations were performed for several variabilities of the environment until equilibria at the corresponding optimal g_2 values were attained. The calculations are in good agreement with simulation results for asexual populations. For sexual populations, however, the expected values are always somewhat higher (up to 40%) than the simulation results. One possible explanation of this effect may be found in the criticism of Turelli (1984) and Bürger (1986 and this Vol.). They argue that under certain circumstances the maintained genetic variance may be smaller than predicted by Lande. In the face of finite populations, this may be more important for sexual than for asexual reproduction. To get an estimate for the genetic variance of g_2 one can fix the g_1 value and see how the geometric mean fitness changes with variation of g_2. This again gives the corresponding fitness curve for application of Eqs. (1) and (2). Figure 3 shows that such curves are characteristically asymmetrical, but for the application of Eqs. (1) and (2), the fitness curve for g_2 should be Gaussian. Therefore, by calculating variances from these curves, one can get only rough estimates of V_w. However, these calculations still agree quite well for parthenogenesis, and they differ by much less than one order of magnitude for sexual reproduction. Therefore, rough estimates of expected genetic variances of the g_2 are possible by this procedure also.

The asymmetry of the expected geometric mean fitness $w(g_1, g_2)$ with respect to g_2 (see Fig. 3) may have another important implication: If one compares the fitness values at lower and higher g_2 values but with the same distance to the optimal g_2, the fitness is more reduced by lowering the breadth of adaptation than by increasing g_2.

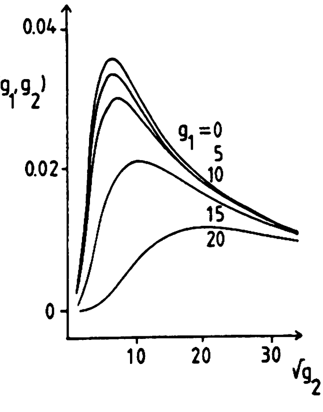

Fig. 3. The expected geometric mean fitness as a function of the realized breadth of adaptation. The curves with lower fitness are calculated for deviations of g_1 (the setting of the maximum of the performance curve) from the mean environmental state

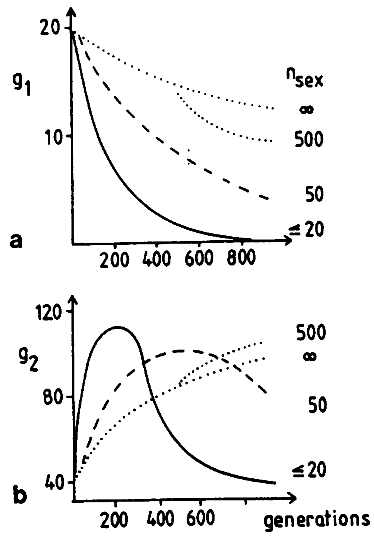

Fig. 4a,b. Evolution to a new mean environmental state (new value 0, old value 20) at various frequencies of sexual reproduction (n_{sex}). The optimal breadth of adaptation ($\sqrt{g_2}$) is identical for both environmental states. **a** Time course of the change in g_1 (setting of the maximum of the performance curve). **b** Corresponding time course of g_2 which is temporarily increased during the evolution of g_1

This higher fitness difference causes a stronger selection. Therefore, evolutionary increases of breadth of adaptation may be faster than reductions.

Figure 3 also demonstrates that the maximum of $w(g_1, g_2)$ occurs always at higher g_2 values when g_1 is not at its optimum (which is chosen to be 0). This becomes important if the environment shifts to a new mean value and the evolution of g_1 (the optimal setting of the maximum of the performance curve) is slow. Let us assume that the optimal breadth of adaptation ($\sqrt{g_2}$) is identical for the old and the new environmental mean value so that only g_1 has to be adapted. But as long as g_1 is not optimal, the fitness is higher for increased g_2 values due to the shape of $w(g_1, g_2)$. Therefore, the evolution to the new g_1 state is accompanied by a temporary increase in g_2. The breadth of adaptation returns to its optimal value when g_1 evolves near its new optimum. This is shown in Fig. 4a and b for parthenogens and cyclical parthenogens with various frequencies of sex. When sexual reproduction occurs more often than once per 25 generations, there is no difference between sexual and parthenogenetic species. At frequencies lower than every 30 generations, a difference between cyclical parthenogens and bisexuals appears. This is in contrast to the study on the evolution of a single quantitative trait where small differences appear at frequencies higher than 100. The reason for this is the small population size which reduces the hidden genetic variance stored during asexual reproduction.

7 Conclusions

The three models presented here demonstrate how concepts of quantitative genetics can be applied fruitfully to problems in evolutionary ecology; here, several qualitative and quantitative predictions are made concerning the performance of both obligate and cyclically parthenogenetic species as compared to sexually reproducing species.

The study of the evolution of a single quantitative character shows that under parthenogenesis a high degree of genetic variability can be maintained, similar in amount to that produced by sexual reproduction, by a balance between input via mutation and output via selection. Therefore, rates of evolution comparable even to sexually reproducing species are possible. Cyclical parthenogenesis has to be considered as a very powerful reproductive strategy because (1) it provides the population with a broader genetic variance after a sexual cycle than there would be under continuous sexual reproduction, (2) the long-term rates of evolution are equal under cyclical parthenogenesis and sexuality and (3) the advantage of faster population growth can be used during asexual reproduction. This model on a single quantitative character refutes the common assertion that parthenogenesis is an evolutionary dead end. A second disadvantage often associated with parthenogenesis, the accumulation of deleterious mutations by Muller's ratchet, is also found to be questionable if fitness is determined by several quantitative characters with a polygenic basis. If back-mutations are not excluded, the mutational load of parthenogenetic species is limited to a level where the mean population fitness is similar to a sexual population under random drift. A further argument conjectured against parthenogenesis is a reduced flexibility in fluctuating environments. A study of the evolution of the breadth of adaptation shows that the optimal breadth of adaptation in temporally and spatially heterogeneous environments is independent of reproductive strategy. The performance of parthenogenetic species is expected to be high even under non-constant environmental conditions. Evolution to a new optimal breadth or to a new optimal setting of the performance curve is indeed faster under sexuality, but this is important only for fast and large changes in the mean environmental state or in temporal or spatial variances. Long-term rates of evolution are identical for cyclical parthenogenesis and permanent sexual reproduction, but under the restriction that the effective population size is large compared to the frequency of sex.

These new and surprising results of the models presented may demonstrate that the application of quantitative genetic models – even with all its inherent simplifications – is a powerful and promising approach to problems in evolutionary ecology.

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