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Parthenogenetic Populations Can Remain Stable in Spite of High Mutation Rate and Random Drift

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Genotypic models [1, 2] imply that the accumulation of deleterious mutations is a mechanism which severely limits the evolutionary potential of parthenogenetic species. It is thought that high genomic mutation rate under weak selection will lead to genetic deterioration due to random fixation of deleterious mutations. This has been called "Muller's ratchet" [3]. For large populations where Muller's ratchet is unlikely to operate, the label of "evolutionary dead end" usually attached to reproduction by parthenogenesis has been shown to be untenable in phenotypic selection models [4]. Here we simulate weak stabilizing selection in small populations under the conditions for which Muller's ratchet is supposed to work, but we assume fitness to be determined by many quantitative characters. We demonstrate that, as expected, a high mutation rate may hinder the maintenance of an optimal genotype, but contrary to the "ratchet" analogy, we find that mean fitness reaches a stable stochastic equilibrium near the optimum.

We simulate a small population of obligately parthenogenetic organisms with discrete and non-overlapping generations. We start with N_e mothers. Each mother produces the same number of offspring, and each offspring carries a new mutation with probability μ. We assume that several (NDIM = dimension of the phenotypic space) quantitative characters $z = (z_1, \ldots, z_{\mathrm{NDIM}})$ contribute multiplicatively to total fitness $W(z)$ of each individual. Defining fitness as survival probability, we apply viability selection by deleting an individual with probability $(1 - W(z))$. After selection the next generation of N_e mothers is chosen randomly from the survivors. These processes are simulated by Monte Carlo methods: we do not evaluate any model equation numerically, but we simulate directly the stochastic events as occurring in natural populations.

The decrease in fitness with increasing distance $|z_i|$ of each character from its optimal value is modelled as a Gaussian function. By a proper scale transformation the fitness function can be treated with optimum value at zero and width σ_w. Then, the fitness $W(z)$ of an individual with phenotype z is given by

$$W(z) = W_0 \prod_{i=1}^{\mathrm{NDIM}} \exp\{-z_i^2/(2\sigma_w^2)\} = W_0 \exp\{-|z|^2/(2\sigma_w^2)\}$$

with $|z| = (\Sigma\, z_i^2)^{1/2}$ as the length (Euclidian norm) of the character vector z and W_0, the maximal fitness, is set equal to 1. Mutations occur with probability μ per genome per generation; any genomic mutation can affect each character. We assume the mutational effects on the genotypic value of a character to be normally distributed around its actual values g_i with variance m^2

$$p(g'_i) = (2\pi m^2)^{-1/2} \exp(-(g'_i - g_i)^2/2m^2) .$$

We let the phenotypic values z_i be normally distributed around the corresponding genotypic values g_i with variance V_e. If we consider a single character z_i, mutation may increase or decrease its value with the same probability. In a multidimensional space of the character vector, mutations on average increase the length for simple geometric reasons. The probability of "back mutations" (which is ignored in the genotypic models [1, 2]), or mutations which decrease the length of the character vector, is given [6] approximately by

$$p_{\mathrm{back}} = 1/2 - (2\pi)^{-1/2} \int_0^y \exp(-t^2/2)\mathrm{d}t$$

with $y = m\mathrm{NDIM}/(2|g|)$ and g as the genotypic equivalent to z.

With fitness 25 % below the optimal value (NDIM = 40, $\sigma_w = 3$, $m = 0.1$), less then 20 % of the mutations are backmutations. Therefore, near the optimum most mutations are deleterious.

We start with a population at the optimum that carry no mutations. After several generations the frequency of the class without mutations decreases and finally reaches zero by random drift: Muller's ratchet has clicked one notch (Fig. 1). The accumulation of mutations continues at its predicted [5] rate (Fig. 2a) and is accompanied by a decrease in average fitness. Contrary to expectations, however, the effect of additional mutations on mean popula-

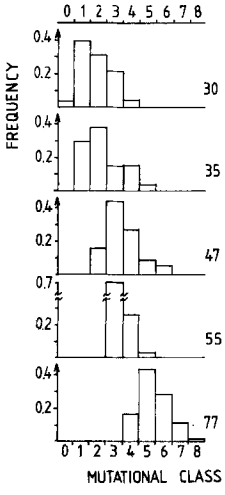
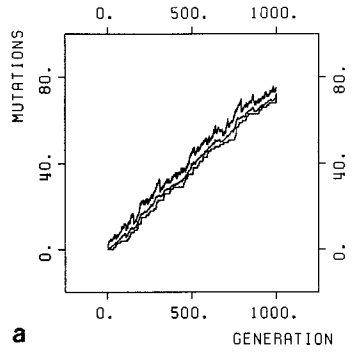
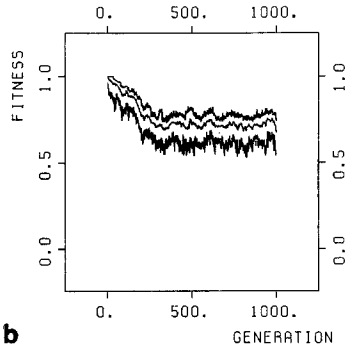


Fig. 1. Operating Muller's ratchet. Loss of genotypes with a minimal number of mutations is demonstrated by the frequency distribution of genotypes classified according to the number of mutations. The statistics are taken after selection and before randomly choosing the mothers of the next generation. The actual generation number is given on the right (NDIM = 40, $m = 0.1$, $\mu = 0.1$, $N_e = 50$, $\sigma_w = 3$)





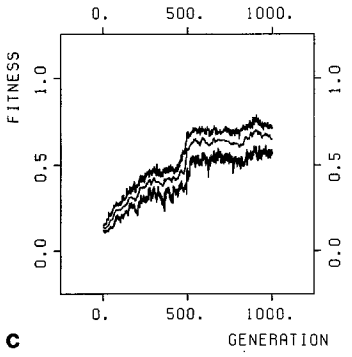


Fig. 2. a) Continuous accumulation of mutations. Maximum, minimum, and average number of mutations per genotype are shown as a function of generation number. b) Equilibration of the phenotypic distribution of a population starting at the optimum. Time courses of maximum, minimum, and average fitness of phenotypes are shown. c) Equilibration of the phenotypic distribution of a population as in (b), but starting far away from the optimum. Note the population approaches the same stochastic equilibrium as in (b)

tion fitness levels off (Fig. 2b). That this is a stable stochastic equilibrium is demonstrated by populations which start far from the optimum and drift back to the same level (Fig. 2c).

These simulations can be understood by a detailed analytical approach [6]: drift keeps the distribution of genotypic values in the population compact preventing them from being widely dispersed on the whole space. The input of variance by mutation is balanced by the loss of variance due to drift, which results in a bounded distribution of phenotypes near its optimum.

Therefore, in this model we can distinguish two different modes of stability: either Muller's ratchet does not operate (e.g., under strong selection) and the optimal class is always reproduced by direct replication, or Muller's ratchet operates in the vicinity of the optimum, but the phenotypic distribution reaches a stochastic equilibrium. These two modes of stability in asexual populations are comparable to the distinction between direct replication and stochastic replication in polynucleotides [7]. The evolutionary efficacy of parthenogenesis is shown by the values of mean population fitness at this stochastic equilibrium which are similar to predictions [8] for bisexual populations under stabilizing selection and random drift.

These results suggest that the influence of high genomic mutation rate and random drift on mean fitness of parthenogenetic populations strongly depends on the topology of the adaptive landscape. If each mutation reduces fitness, genetic deterioration is inevitable [1 – 3]. However, deleterious mutations have only little impact if many characters contribute to fitness and genomic mutations reduce fitness only in the statistical average: if selection is too weak to maintain a specific optimal genotype, random drift balances the input of mutations.

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