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PHENOTYPIC EVOLUTION AND PARTHENOGENESIS

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The argument has often been made that obligate unisexuality is an evolutionary dead end incapable of sustaining a lineage for very long periods on an evolutionary time scale or of spawning new phylogenetic groups (Darlington 1939; Stalker 1956; Muller 1964; Mayr 1970; Uzzell 1970; White 1973; Maynard Smith 1978). Such assertions are generally based on the opinions that the absence of recombination will eliminate the only mechanism by which a population can rid itself of deleterious mutations (Muller's ratchet) as well as severely restrict the potential for phenotypic evolution that is essential for coping with a variable environment.

The validity of the evolutionary dead end label is questionable. Many extant parthenogens have enormous population sizes, are much more geographically widespread than their bisexual relatives, and possess highly generalized phenotypes capable of existence under a diversity of environmental conditions. Most parthenogens that appear to be in danger of extinction owe their problems to genetic disruptions resulting from backcrosses to their bisexual parental species rather than to inferior competitive or coevolutionary abilities (M. Lynch, in prep.).

One could argue that current success is an inappropriate measure of evolutionary potential because it fails to consider the dynamic aspects of the selection process. However, several lines of evidence indicate that phenotypic evolution does not come to a complete standstill even under asexual parthenogenesis. Those populations of obligate parthenogens that have been examined biometrically exhibit levels of phenotypic variance comparable to that in their bisexual relatives (Oliver and Herrin 1976; Atchley 1977, 1978; Parker 1979). These results do not appear to be attributable to polyphyletic origins of the unisexual races or to meiotic parthenogenesis; they could be a consequence of a higher level of unisexual phenotypic sensitivity to environmental effects, a trend which would, however, be contrary to what is generally observed in parthenogens (M. Lynch, in prep.). Many studies have presented strong evidence of allelic and karyotypic evolution in unisexual lineages of monophyletic origin (Suomalainen 1961; Badino and Robotti 1975; Lokki et al. 1976; Saura et al. 1976; Parker 1979; Leslie and

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Vrijenhoek 1980; Ochman et al. 1980), and several positive responses to selection have been obtained with initially isogenic parthenogenetic lines (Banta 1939; Henslee 1966). Finally, a remarkably complete set of fossil data from the late Cenozoic suggests that *Melanoides tuberculata*, one of the few unisexual mollusks in existence today, has undergone evolutionary transitions just as dramatic as sexual species (Williamson 1981).

Although theoretical work signals the potential importance of Muller's ratchet in haploid unisexual populations (Felsenstein 1974; Maynard Smith 1978), there is as yet no direct evidence that a gradual accumulation of deleterious mutations results in a depression of fitness and ultimately in extinction of diploid unisexuals. Indeed, one study appears to provide a strong statement to the contrary. A parthenogenetic control line of *Drosophila mercatorum* that has been maintained for 19 yr (several hundred generations) has exhibited a gradual increase in parthenogenetic capacity despite the fact that it routinely has been maintained at a small size (50–100) and occasionally reduced to a single individual, precisely the conditions that would encourage the operation of Muller's ratchet (Templeton 1982). Results from competition experiments between bacterial strains differing only in level of mutator activity are also contrary to expectations under Muller's ratchet (Chao and Cox 1983). Leslie and Vrijenhoek (1980) have demonstrated, with hybridogenetic strains of the fish *Poeciliopsis*, an accumulation of mutations that are deleterious in the homozygous state, but the attributes of these mutant alleles in their normal heterozygous genetic background are not known.

Most characters upon which selection acts have a polygenic basis with multiple alleles segregating at many loci (Wright 1968). It is overly simplistic to classify polygenic mutations as inherently deleterious or beneficial as their influence on fitness critically depends upon the genetic background in which they arise and their fidelity to that background. Lande (1976*b*) has demonstrated for bisexual populations that polygenic mutation is a potent evolutionary mechanism that can maintain large amounts of genetic variance even in the face of intense selection. In principle, but with additional limitations, the same is true for unisexual populations.

In order to develop dynamic expressions for phenotypic evolution in unisexual populations under as biologically realistic conditions as possible, we have constructed phenotypic selection models that are directly coupled to the underlying polygenic system. By comparison of our results with similar derivations for bisexuality (Lande 1976*a*, 1976*b*, 1977) it becomes possible to quantify expected differences in rates of phenotypic evolution between unisexual and bisexual lineages, an element that has previously been missing from the evolution-of-sex controversy. The long record of successful application of phenotypic selection models, similar in principle to those employed here, by plant and animal breeders (Falconer 1981) supports the extension of such models to problems in evolutionary ecology (Lande 1976*a*, 1976*b*, 1982).

EVOLUTION IN OBLIGATE PARTHENOGENS

We start by assuming a population size that is effectively infinite so that genetic drift will not be an important evolutionary force. This is probably a reasonable

assumption for many parthenogenetic rotifers, nematodes, crustaceans, insects, and millipedes. Take a low density *Daphnia* population of 10^3 individuals/m² for example; the population size in a small 100×100 m pond would be 10^7 . We further assume that the parthenogenetic mechanism insures that the maternal genotype is transmitted to progeny intact with the exception of mutations. This is generally true of apomictic parthenogenesis as well as for many forms of meiotic parthenogenesis (Suomalainen 1950; Narbel-Hofstetter 1964; White 1973).

Under the additional assumptions of diploidy and a purely additive genetic basis for characters, the genotypic value of an individual is

$$g = \sum_{i=1}^n (a_i + a_{n+i})$$

where n is the number of loci contributing to the character, and a_i and a_{n+i} are the allelic effects of the two genes at locus i . The phenotype of an individual is then represented by

$$z = g + e$$

where e is the environmental contribution to the trait assumed to be normally distributed with variance, V_e , and uncorrelated with g , so that the conditional probability

$$p(z|g) \propto \exp \left[-\frac{(z - g)^2}{2V_e} \right].$$

In the following, we treat z as a normally distributed, independent character or linear combination of characters, but multivariate evolution can be treated in a similar manner as shown by Lande (1979). The normality assumption can be met for most metric characters by use of an appropriate scale transformation (Wright 1968).

The assumption of no covariance between genotypic value and environmental effect is common to almost all quantitative genetic models for reasons of mathematical tractability. Its biological justification depends on the situation. There is no question that genotypes vary in their response to different environments, i.e., that genotype-environment interactions are important (Bulmer 1980). However, almost all information on the subject comes from genetically unique (and often inbred) lines of domesticated plants and animals rigidly maintained in distinct environments throughout their lives. Whether or not a genotype-environment interaction will translate into genotype-environment covariance in a natural environment will depend on the extent to which individuals with different genotypic values are able to assort into different subhabitats according to their average environmental effects. The problem is essentially one of scale: Populations of individuals that are immobile with respect to important environmental variation should not exhibit significant genotype-environment covariance. Moreover, even when genotype-environment interactions do appear to be important, they can sometimes be rendered negligible by an appropriate transformation of the scale of measurement. The effect of true positive genotype-environment covariance will be to enhance the response to directional selection since the genotypes closest to the projected optimum will have exaggerated phenotypes.

We have chosen to represent the operation of selection on the phenotypes by a Gaussian fitness function with an optimum phenotype, θ ,

$$W(z) \propto \exp \left[- \frac{(z - \theta)^2}{2V_w} \right].$$

$\sqrt{V_w}$ is a measure of the width of the fitness function and is inversely related to the intensity of selection operating on suboptimal phenotypes. Although the Gaussian fitness function defines a form of stabilizing selection, it can also be used to represent directional selection operating on populations whose mean phenotype ($\bar{z} = \bar{g}$) is far above or below the optimum. The fitness of genotype g is

$$W(g) \propto \int W(z) \cdot p(z|g) \cdot dz \propto \exp \left[- \frac{(\theta - g)^2}{2(V_e + V_w)} \right]. \quad (1)$$

Finally, all genes are subject to mutation each generation, giving a per-generation input of genetic variance into the population of

$$V_m = \sum_{i=1}^n (\mu_i m_i^2 + \mu_{n+i} m_{n+i}^2)$$

where μ and m^2 represent the mutation rate and variance of mutational effects. The genotype distribution of the progeny of individuals with genotypic value g in generation t is then

$$p[g(t+1)|g(t)] \propto \exp \left\{ - \frac{[g(t+1) - g(t)]^2}{2V_m} \right\}. \quad (2)$$

After the application of selection and mutation, the genotype distribution in the following generation becomes

$$p[g(t+1)] \propto \int p[g(t)] \cdot W(g) \cdot p[g(t+1)|g(t)] \cdot dg. \quad (3)$$

The distribution of $g(t)$ is always normal, since (3) is simply a convolution of normal distributions, and can be written as

$$p[g(t)] \propto \exp \left\{ - \frac{[g(t) - \bar{g}(t)]^2}{2V_g(t)} \right\} \quad (4)$$

where V_g is the genetic component of phenotypic variance for the character. Under the assumption of no genotype-environment covariance, V_g is the genotype-phenotype covariance which itself is the sum of the elements of the covariance matrix of allelic effects

$$V_g(t) = \text{Cov}_{g,z}(t) = \sum_{i=1}^{2n} \sum_{j=1}^{2n} \text{Cov}_{a_i, a_j}(t).$$

Upon substitution of (1), (2), and (4), equation (3) expands to

$$p[g(t+1)] \propto \int \exp \left(- \frac{1}{2} \left\{ \frac{[g(t) - \bar{g}(t)]^2}{V_g(t)} + \frac{[\theta - g(t)]^2}{V_e + V_w} + \frac{[g(t+1) - g(t)]^2}{V_m} \right\} \right) dg(t)$$

which upon integration becomes

$$p[g(t + 1)] \propto \exp \left[-\frac{1}{2} \cdot \frac{\left(g(t + 1) - \left\{ \theta - \left[1 - \frac{V_g(t)}{V_g(t) + V_e + V_w} \right] \cdot [\theta - \bar{g}(t)] \right\} \right)^2}{V_m + V_g(t) \cdot \left[1 - \frac{V_g(t)}{V_g(t) + V_e + V_w} \right]} \right].$$

This is itself a normal distribution with mean phenotype

$$\bar{g}(t + 1) = \bar{g}(t) + \left\{ \frac{V_g(t) \cdot [\theta - \bar{g}(t)]}{V_T(t) + V_w} \right\} \tag{5}$$

and genetic variance

$$V_g(t + 1) = V_g(t) + V_m - \frac{V_g^2(t)}{V_T(t) + V_w} \tag{6}$$

where $V_T(t) = V_g(t) + V_e$ is the total phenotypic variance. These are the basic recursion equations for evolutionary change of an asexual parthenogen exposed to Gaussian selection and mutation. Although we have derived (5) and (6) in terms of additive gene systems for later comparisons, they apply in principle to any obligately nonrecombinational unisexual system when V_m is the total per-generation input of variance in genotypic values via mutation and V_g is the total (including dominance and epistasis), rather than additive, expressed genetic variance.

Equation (6) shows that the genetic variance of a unisexual population is bounded away from zero by mutation. As long as that is true, then by (5), the mean phenotype must converge on the optimum, θ . Equation (6) also shows that an equilibrium level of genetic variance will be reached when the input via mutation is balanced by the output by selection,

$$\hat{V}_g = \frac{V_m + \sqrt{V_m[V_m + 4(V_e + V_w)]}}{2}. \tag{7}$$

Since \hat{V}_g is independent of the optimum phenotype, it can be reached well before evolutionary change has ceased, and it will even be maintained when θ is fluctuating so long as the width of the fitness function, V_w , remains roughly constant. Once the population has attained its equilibrium level of genetic variance, phenotypic evolution will proceed at the rate

$$\Delta \hat{g} = \left(\frac{\hat{V}_g}{\hat{V}_g + V_e + V_w} \right) \cdot (\theta - \bar{g}). \tag{8}$$

Equations (5) and (8) are identical to those derived by Lande (1976a, 1976b) for polygenic characters with a purely additive genetic basis in bisexual populations. Therefore, for such characters, differences in the rates of phenotypic evolution between unisexual and bisexual populations are to be found in the differences in equilibrium levels of genetic variance and the rate at which such variance can be acquired in unisexual populations. Lande (1976b, 1977) has shown for characters

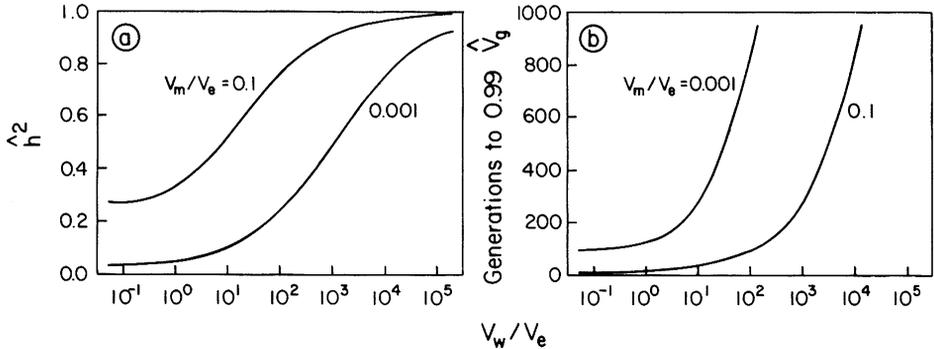


FIG. 1.—*a*, Equilibrium levels of heritability ($= \hat{V}_g/V_T$) for polygenic characters in clonal populations as a function of the per-generation mutational input of genetic variance (V_m), environmental variance (V_e), and the width of the fitness function ($\sqrt{V_w}$). The intensity of selection decreases with increasing V_w . *b*, Time needed for single incipient clones to attain 99% of their equilibrium levels of expressed genetic variance.

with an additive genetic basis in bisexual populations that

$$\hat{V}_g = \frac{2nV_m + \sqrt{2nV_m[2nV_m + 4(V_e + V_w)]}}{2}$$

This is identical in form to (7) except that the equilibrium genetic variance in bisexual populations depends on the effective number of segregating units ($2n$ in a diploid). In obligate apomicts the entire genome is transmitted as a single, linked unit. Thus, the relative evolutionary rates of well-established unisexual and bisexual populations depend critically upon the number of loci contributing to the character, the mutation rate per locus, and the sensitivity of phenotypic development to environmental effects. The rate of phenotypic evolution in a unisexual population would actually exceed that of an otherwise comparable bisexual population if $V_{m,unisex} > 2nV_{m,bisex}$. In addition, a relatively low sensitivity to environmental effects would tend to elevate the equilibrium heritability (\hat{V}_g/V_T) of a unisexual population (fig. 1*a*), thereby increasing the efficiency of the selective process.

Two sets of circumstances suggest that these conditions may often be met in unisexual populations. First, based on their mode of origin, there is reason to believe that the mutation rate of many unisexual species may be elevated relative to that of their bisexual ancestors. There is substantial cytogenetic and electrophoretic evidence that many obligate parthenogens owe their origin to hybridization events, and several successful syntheses of clones via hybridization of bisexual parental species have been accomplished in the laboratory (Harrison and Peacock 1926; Astaurov 1969; Schultz 1973; White et al. 1977; O'Rourke 1979). In fact, as yet there is no good evidence that any extant parthenogenetic vertebrate has arisen by any other mechanism. Experiments with *Drosophila* have repeatedly demonstrated that mutator activity is increased up to 10 times upon interspecific hybridization (Thompson and Woodruff 1978). This may be primarily a meiotic effect, but numerous parthenogens rely on automictic systems. Second,

ecological and biogeographic data for parthenogenetic species from a diversity of phylogenetic groups provide support for the idea that, compared to their bisexual relatives, obligate parthenogens tend to have highly generalized genotypes, i.e., phenotypes that are relatively insensitive to environmental variation (M. Lynch, in prep.).

Lande's (1976a) analysis of data from *Drosophila*, mice, and maize indicates that $V_m/V_e \approx 1 - 5 \times 10^{-3}$ for bisexual species. Therefore, 10^{-3} and 10^{-1} would appear to be reasonable limits of this parameter for parthenogenetic species, based on the arguments above. Figure 1a illustrates the equilibrium levels of heritability for different values of V_w/V_e for these two extremes of V_m/V_e . Both mutation and environmental variation limit the effectiveness with which clonal selection can eliminate genetic variance, the first by introducing new genetic variation each generation following selection, the second by reducing the ability of the selective process to discriminate between genotypes. Even under extremely intense selection (low values of V_w/V_e), the heritabilities of obligately parthenogenetic populations will be maintained at levels (0.03–0.26) that are within the range of values commonly observed in bisexual populations (Falconer 1981).

Since an incipient clone has, by definition, zero genetic variance at the outset, it is necessary to examine the rate at which the equilibrium level is approached. We have determined, by iteration, the number of generations that it takes clones with $V_m/V_e = 10^{-3}$ and 10^{-1} to attain 99% of their equilibrium levels of genetic variance (fig. 1b). Except for cases of very weak selection (the percent selective mortality at $V_w/V_e = 10^3$ is 0.1 and 0.5 for V_m/V_e of 0.001 and 0.1, see below), the times to convergence are quite rapid, generally < 1,000 and frequently < 200 generations. Under strong selection and $V_m/V_e = 10^{-1}$, precisely the conditions that would generate a rapid rate of phenotypic evolution, the equilibrium level of genetic variance is reached in as few as 10 generations. Since the majority of parthenogens reproduce at least annually, and some such as rotifers and cladocerans may have up to 50 generations in a single year, most extant obligate parthenogens should be close to their equilibrium levels of genetic variance and should exhibit considerable evolutionary potential. Indeed, depending on the rate of input of variance by mutation and the intensity of selection, a change in \bar{g} , the mean genotypic value, by as much as five phenotypic standard deviations in as few as 100 generations is feasible under obligate parthenogenesis (fig. 2), provided that the population is large and/or fecund enough to bear the selective load.

For populations that have attained the optimum phenotype in stable environments, there is one clear advantage to unisexuality. Because inferior genotypes are not generated by recombination, there will be less of a load on a unisexual populations. The mean fitness of individuals in a population is

$$\bar{W}(t) = \int p[g(t)] \cdot W(g) \cdot dg$$

$$\propto \frac{1}{[V_g(t) + V_e + V_w]^{1/2}} \cdot \exp \left\{ - \frac{[\theta - \bar{g}(t)]^2}{2[V_g(t) + V_e + V_w]} \right\}. \quad (9)$$

For equilibrium populations with $\bar{g} = \theta$ this reduces to

$$\bar{W} \propto (\hat{V}_g + V_e + V_w)^{-1/2},$$

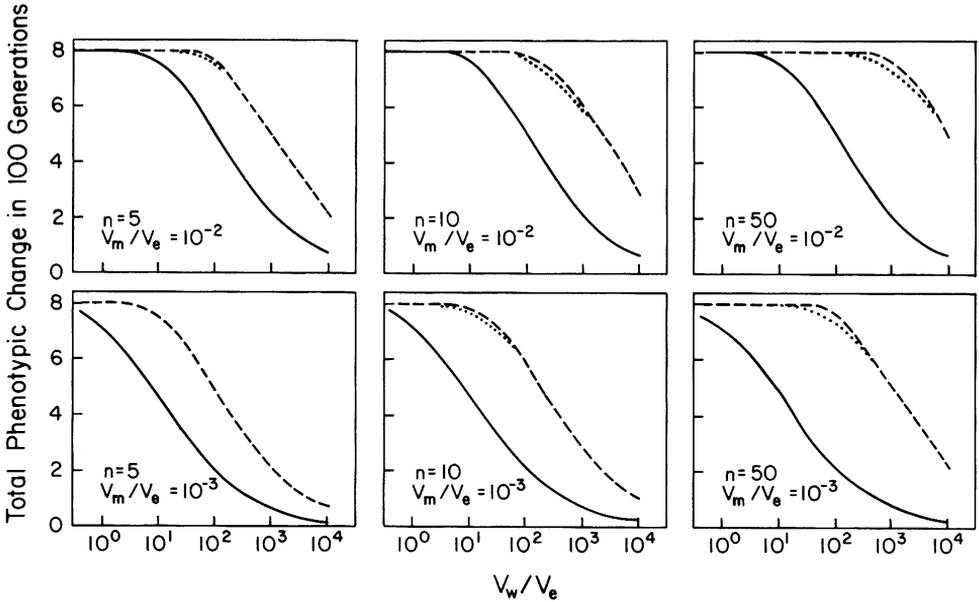


FIG. 2.—Total phenotypic change in environmental standard deviations ($\sqrt{V_e}$) for obligate bisexuals and cyclical parthenogens with a periodicity of sex < 50 generations (dashed lines) and obligate unisexuals (solid lines) over a 100-generation period for various combinations of the number of loci (n), rate of input of genetic variance via mutation (V_m), environmental variance (V_e), and width of the fitness function ($\sqrt{V_w}$). Intensity of selection decreases with increasing V_w . All populations had attained their equilibrium levels of genetic variance and were set at $\theta - \bar{g} = 8$ at the beginning of the 100-generation run. The dotted lines give the selection response for cyclical parthenogens engaging in sex once every 100 generations.

giving a proportion of selective deaths of

$$1 - \frac{\bar{W}}{W(\theta)} = 1 - \left\{ \frac{V_w}{\hat{V}_g + V_e + V_w} \right\}^{1/2}.$$

This quantity is the mean proportional reduction of fitness of individuals in an equilibrium population relative to the optimal phenotype.

HIDDEN GENETIC VARIANCE

Since mutation cannot be prevented, selection operating on a clonal population is actually a continuous process of altering the covariance relationships between the effects of mutant alleles and nonalleles in ways that favor specific phenotypes. This is especially true for unisexual populations since the individual acts as a linkage group from which new mutations cannot be purged. With variable allelic effects and multiple loci, a given phenotype can be encoded in a multitude of different ways. Therefore, even under extremely intense selection for a narrow

range of phenotypes, we can anticipate a large amount of genetic variance in a clonal population that will be hidden by negative covariances between the effects of alleles and nonalleles.

To investigate this problem, it is necessary to re-examine the clonal genotype at the level of the gene. First, we examine the joint distribution of allelic effects after a generation of mutation and selection. Under the assumption that mutations are randomly and normally distributed, the initial genotype distribution of the population prior to selection on the first mutants will be multivariate normal

$$p(a_1, \dots, a_{2n}) \propto \exp \left(-\frac{1}{2} \sum_{i=1}^{2n} \sum_{k=1}^{2n} S_{ik} a'_i a'_k \right)$$

where a'_i and a'_k are the additive effects of alleles i and k measured as deviations from their means \bar{a}_i and \bar{a}_k , and S_{ik} is the ik th element of the inverse of the covariance matrix of allelic effects. Rewriting the Gaussian fitness function as

$$W(a_1, \dots, a_{2n}) \propto \exp \left[-\frac{1}{2} \frac{(\sum_{i=1}^{2n} a'_i)^2}{V_e + V_w} \right]$$

with the optimal phenotype, $\theta = 0$, the convolution of $p(a_1, \dots, a_{2n})$ and $W(a_1, \dots, a_{2n})$ gives the joint allelic distribution following selection

$$p'(a_1, \dots, a_{2n}) \propto \exp \left[-\frac{1}{2} \sum_{i=1}^{2n} \sum_{k=1}^{2n} \left(S_{ik} + \frac{1}{V_e + V_w} \right) a'_i a'_k \right]$$

which is itself multivariate normal. Although the covariance structure changes, multivariate normality is thereafter maintained by mutation and Gaussian selection. This insures that the regressions of individual allelic effects on phenotypes will all be linear and homoscedastic (Kendall and Stuart 1979) so that the hidden genetic variance in a population can be estimated following a procedure introduced by Lande (1977).

The dynamic expression for the covariance between allelic effects can be written

$$\text{Cov}_{a_i, a_j}(t + 1) = [\text{Cov}_{a_i, a_j}(t)]_w + \delta_{ij} \mu_i m_i^2 \tag{10}$$

where $[\text{Cov}_{a_i, a_j}(t)]_w$ is the covariance between effects of alleles i and j in the previous generation after selection, and $\delta_{ij} = 1$ if $j = i$ and 0 if $j \neq i$. The allelic effect-phenotype regression before selection is

$$a'_i(t) = \frac{\text{Cov}_{a_i, z}(t)}{V_T(t)} \cdot [z - \bar{z}(t)] + \epsilon_{i,z} \tag{11}$$

where $\epsilon_{i,z}$ is the independent and homoscedastic residual element. Taking the cross products of $a'_i(t)$ and $a'_j(t)$ and averaging over all individuals gives

$$\text{Cov}_{a_i, a_j}(t) = \frac{\text{Cov}_{a_i, z}(t) \cdot \text{Cov}_{a_j, z}(t)}{V_T(t)} + \overline{\epsilon_{i,z} \cdot \epsilon_{j,z}}. \tag{12}$$

Although the slope of (11) is not altered by selection, the phenotype distribution $p[z - \bar{z}(t)]$ is, so that

$$[\text{Cov}_{a_i, a_j}(t)]_w = \frac{\text{Cov}_{a_i, z}(t) \cdot \text{Cov}_{a_j, z}(t)}{V_T(t)} \cdot \frac{[V_T(t)]_w}{V_T(t)} + \frac{\epsilon_{i:z} \cdot \epsilon_{j:z}}{\epsilon_{i:z} \cdot \epsilon_{j:z}} \quad (13)$$

Substituting (12) into (13) and back into (10) yields

$$\text{Cov}_{a_i, a_j}(t + 1) = \text{Cov}_{a_i, a_j}(t) - \frac{k(t) \cdot \text{Cov}_{a_i, z}(t) \cdot \text{Cov}_{a_j, z}(t)}{V_T(t)} + \delta_{ij} \mu_i m_i^2 \quad (14)$$

where $k(t) = 1 - \{[V_T(t)]_w/V_T(t)\}$ is the proportional reduction of phenotypic variance due to selection, which for a Gaussian fitness function is equal to $V_T(t)/[V_T(t) + V_w]$.

Equation (14) is the general recursion expression for the elements of the covariance matrix of allelic effects for a population reproducing entirely by clonal propagation. Under the assumptions of equal mutability of all loci all of the diagonal elements of this matrix will be equal to each other, as will all of the off-diagonal elements. As noted earlier, the expressed genetic variance is equal to the sum of all the elements of this covariance matrix. The sum of the diagonal elements

$$V_{g^t}(t + 1) = 2n \cdot \text{Cov}_{a_i, a_i}(t + 1) \quad (15)$$

is the total amount of allelic variance in the population and the sum of the off-diagonal elements

$$V_{gh}(t + 1) = 2n(2n - 1) \cdot \text{Cov}_{a_i, a_j}(t + 1) \quad (16)$$

is the amount by which V_g differs from V_{g^t} because of covariance between allelic and nonallelic effects.

Noting that

$$\text{Cov}_{a_i, z}(t) = \text{Cov}_{a_i, a_i}(t) + (2n - 1)\text{Cov}_{a_i, a_j}(t)$$

and substituting (14) in (15) and (16), we obtain the recursion equations for the total variance and covariance of allelic effects

$$V_{g^t}(t + 1) = V_{g^t}(t) + V_m - \frac{V_g^2(t)}{2n[V_g(t) + V_e + V_w]}, \quad (17)$$

$$V_{gh}(t + 1) = V_{gh}(t) - \frac{(2n - 1)V_g^2(t)}{2n[V_g(t) + V_e + V_w]}. \quad (18)$$

These equations show that, although the expressed genetic variance of a clonal lineage will be maintained at an equilibrium by a balance between selection and mutation (eq. [7]), the total amounts of allelic variance and covariance will not. Under purely clonal reproduction, $V_{gh}(t)$ will continuously decrease over time, the rate depending upon the number of effective loci, the levels of expressed genetic variance and environmental variance, and the intensity of selection. $V_{g^t}(t)$ will continuously increase. Moreover, since V_{gh} is by definition zero when a clone initially arises, it is always negative. Therefore, the negative of V_{gh} is the amount

of genetic variance in a clonal population hidden in the form of negative correlations between permanently linked alleles. V_{gt} can thus be thought of as the potential amount of genetic variance available to a clonal population were these linkages to be broken.

Equations (17) and (18) formalize Muller's (1964) supposition about the accumulation of mutant alleles under unisexuality. Mutant alleles do indeed continuously accumulate in ameiotic parthenogens, but for polygenic characters a considerable amount of this variation can be permanently masked. There must be some biological limits to the extent that the polygenes can be altered and still remain functional, i.e., an upper limit to the amount of genetic variance that can be hidden. However, these limitations may be very high and reached only after the passage of considerable evolutionary time when large numbers of loci contribute to characters. If this true, then the amount of hidden genetic variance in a clonal population would provide a measure of its age. In some cases such variation might be quantified by forced backcrossings of the parthenogenetic females with isogenic lines of their bisexual relatives or by conventional techniques in molecular biology.

EVOLUTIONARY CONSEQUENCES OF PERIODIC SEX

A number of organisms have cyclically parthenogenetic or heterogonic life cycles, i.e., alternating periods of unisexual and bisexual reproduction. Gall wasps of the family Cynipidae typically follow a single generation of unisexuality with one of bisexuality. Most rotifers, digenetic trematodes, aphids, and cladocerans have an indefinite number of unisexual generations interspersed with single generations of sex. The same is true of many plants and lower animals that propagate vegetatively. For these organisms, the storage of hidden genetic variance during the unisexual phase has important implications for the rate of phenotypic evolution following a bout of recombination.

In the following we assume that, in a sexual generation, clonal selection operates prior to recombination and mutation, and that mating is random, an assumption that is at least approximately true in cladocerans (Lynch 1983a, 1983b). Under random mating all of the covariances between alleles in uniting gametes become zero; this accounts for $2n \cdot n$ of the $2n(2n - 1)$ off-diagonal elements in the covariance matrix of allelic effects. The remaining $2n(n - 1)$ off-diagonal elements account for the covariances of allelic effects within gametes, these being reduced by recombination by the factor $(1 - r_{ij})$ where r_{ij} is the recombination frequency between the i th and the j th loci. Under free recombination $r_{ij} = 0.5$. Taking r to be equal over all loci, after a generation of sex the hidden genetic variance is reduced to

$$V_{gh}(t + 1) = (1 - r) \cdot \frac{(n - 1)}{(2n - 1)} \cdot [V_{gh}(t)]_w \quad (19)$$

where $[V_{gh}(t)]_w$ is the hidden genetic variance in generation t following clonal selection and before recombination. Thus, for polygenic characters with large effective numbers of loci, hidden genetic variance can be reduced by as much as

75% by a single generation of recombination. All of this variance is immediately converted to expressed genetic variance which is then exposed to clonal selection. For cyclical parthenogens with long phases of unisexuality, massive amounts of hidden genetic variance may be released following a bout of recombination, leading to a sudden increase in the response to selection.

We have examined the evolutionary dynamics of cyclically parthenogenetic systems by computer simulation using values of n from 5 to 50, $V_e = 1$, V_m from 0.001 to 0.1, V_w from 0.1 to 10,000.0, r from 0.0 to 0.5, and sex every 2 to 100 generations. Starting from a single clone, the populations always evolve to stable equilibrium cycles of expressed and hidden genetic variance that are independent of θ and the recombination rate. In the absence of any simple analytical solution for the equilibrium cycles, we assumed that the populations had reached their equilibrium levels of genetic variance when the relative change per cycle was $< 10^{-14}$. The time necessary to reach this point increases with the number of loci, and decreases with the frequency of sex, the recombination rate, and the intensity of selection (fig. 3). For most reasonable values of the genetic and environmental parameters, cyclically parthenogenetic populations starting from single clones attain 99% of their equilibrium genetic variance within 500–10,000 generations.

Once the equilibrium cycles of genetic variance were established, we set all of the mean phenotypes equidistant from θ and followed their subsequent convergence on the optimum phenotype. A general principle that emerges from this work is that, for additive genetic systems that have attained their equilibrium level of genetic variance, the long-term rate of phenotypic evolution is approximately independent of the frequency of sex so long as the characteristics of the fitness function remain constant during the cycle. The release of hidden genetic variance following sex elevates the heritability in cyclical parthenogens (fig. 4) and allows them to periodically attain much higher per-generation rates of phenotypic evolution than can ever be experienced under obligate bisexuality, but in the long term this periodic advantage is balanced by the cost of not producing recombinants during the unisexual phase (fig. 5).

The amount of phenotypic evolution over a number of generations actually increases with the frequency of sex, but the differences between most cyclical parthenogens and obligate bisexuals are negligible as can be seen in figure 2. The additional time required for a cyclical parthenogen to evolve to a specific phenotype never exceeded the length of a single cycle. Only when the cycle length approached 100 generations (which probably rarely occurs for most extant cyclical parthenogens) did we begin to detect a slight reduction in phenotypic evolution at intermediate selection intensities when calculations were carried out to four digits (fig. 2). However, even in these cases, the long-term level of adaptation (measured as the geometric mean of \bar{W} , eq. [9]) over a number of generations was always lowest for bisexuality and increased to a maximum for the cycle length equal to the selective period. This measure of adaptation is directly related to the probability of survival of a population (Lande 1976*b*).

Thus, at least for situations in which the selection pressures do not change significantly over time scales equal to the entire life cycle, advantages of continuous sex over cyclical parthenogenesis are not to be found in either the rate of

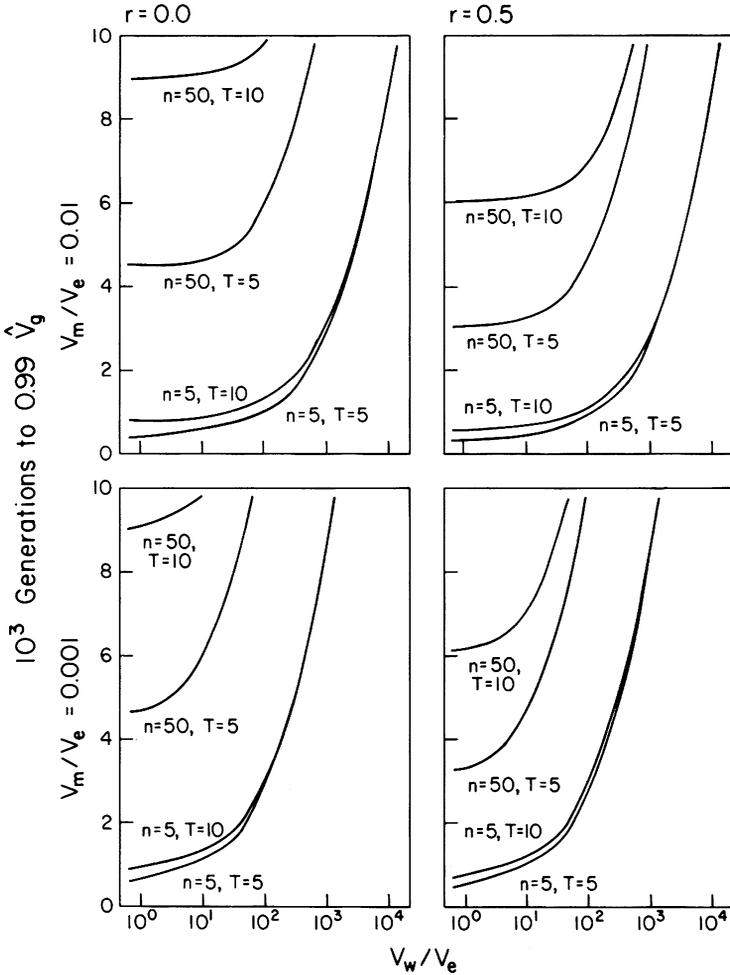


FIG. 3.—Total number of generations (unisexual plus bisexual) required for populations of cyclical parthenogens initiated from a single clone to reach 99% of their equilibrium levels of expressed genetic variance. Values are given for complete linkage ($r = 0.0$) and free recombination ($r = 0.5$) under various combinations of number of loci (n), periodicity of sex (T), input of genetic variance via mutation (V_m), environmental variance (V_e), and width of the fitness function ($\sqrt{V_w}$).

phenotypic evolution or the probability of extinction. We have not exhaustively examined the consequences of variable environments, but for almost all cases in which we allowed θ to vary linearly, quadratically, or sinusoidally with time, we found that the geometric mean \bar{W} was highest for the longest cycle length and lowest for obligate bisexuality regardless of the relative positions of the mean phenotypes at the end of the selective period. These results help explain why only a single generation of sex but one or more consecutive generations of unisexuality comprise the life cycles of almost all cyclical parthenogens, and why most cyclical

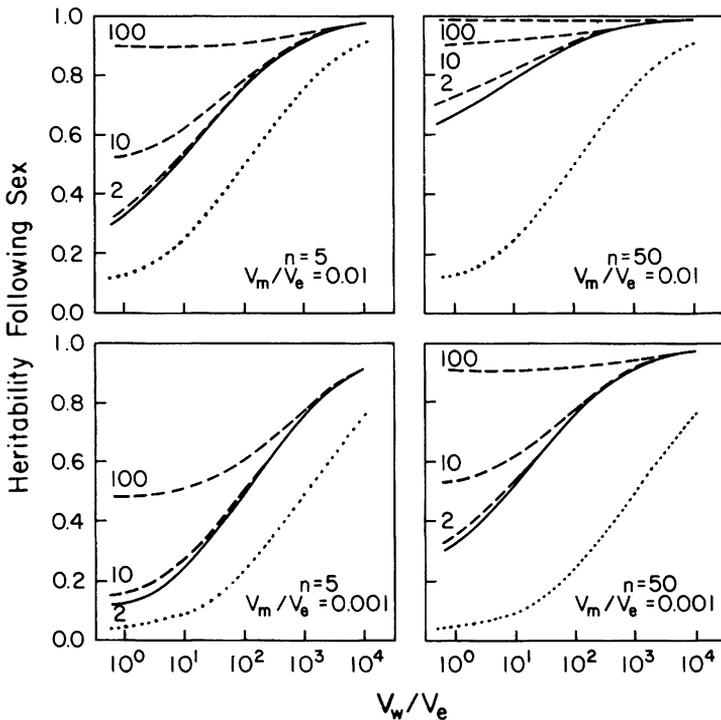


FIG. 4.—Equilibrium heritability in the generation following sex for cyclical parthenogens with sex every 2, 10, and 100 generations (dashed lines) as a function of width of the fitness function ($\sqrt{V_w}$), number of loci (n), rate of input of genetic variance via mutation (V_m), and environmental variance (V_e). Equilibrium levels of heritability for obligate bisexuals (solid line) and obligate unisexuals (dotted lines) are given for reference.

parthenogens initiate their annual phases with sexually rather than parthenogenetically produced, diapausing propagules. The results also make it difficult to explain the rarity of cyclical parthenogenesis on the basis of optimization criteria, and lend support to the argument that extremely stringent cytogenetic barriers, early evolutionary bottlenecks, and subsequent interference from secondarily derived obligate parthenogens prevent most bisexual populations from making the transition to cyclical parthenogenesis (White 1973; Lynch 1983*b*).

DISCUSSION

The models that we have presented for unisexual evolution indicate that the “evolutionary dead end” label is untenable. Although for purely additive systems obligate parthenogens will always evolve at slower rates than otherwise similar bisexuals (with the same number of loci, rate and distribution of effects of polygenic mutations), most unisexual populations that have been established for more than a few hundred years should nonetheless contain sufficient genetic variance for metric characters to generate observable rates of phenotypic evolu-

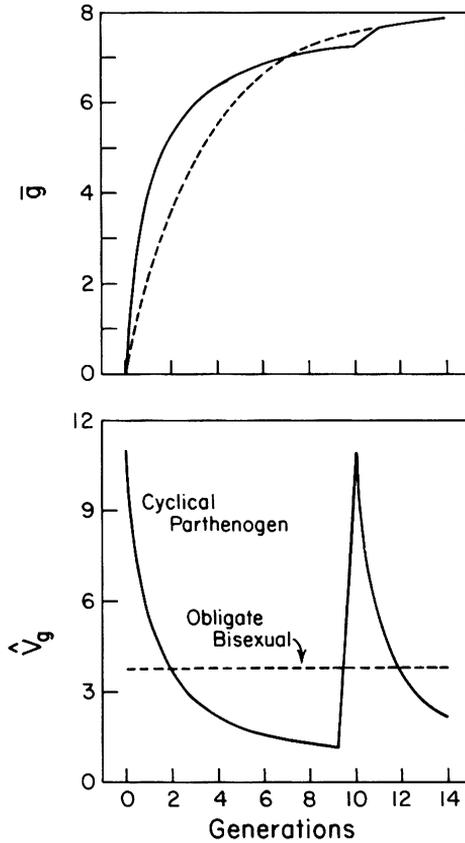


FIG. 5.—Dynamics of phenotypic evolution for a cyclical parthenogen with sex every 10 generations (solid line) and an obligate bisexual (dashed line) exposed to a Gaussian fitness function with $V_w = 10$ and $\theta - \bar{g}$ initially equal to 8.0. $V_m = 0.01$ and $V_e = 1.0$. \bar{g} = mean phenotypic value and \hat{V}_g = level of expressed genetic variance.

tion. If anything, we have overemphasized the disadvantage of obligate parthenogenesis by focusing on purely additive systems. Dominance and epistasis are the rule rather than the exception for polygenic systems (Wright 1968), but only in the case of functionally ameiotic parthenogenesis do these components of genetic variance contribute much to the correlation between relatives and hence to the rate of phenotypic evolution. If, in addition, mutation rates are commonly elevated and environmental sensitivity depressed in obligate parthenogens as indirect evidence suggests, then it seems likely that rates of phenotypic evolution under this mode of life may frequently approach or even exceed those under bisexuality.

An especially potent mechanism for phenotypic evolution arises when ameiotic parthenogenesis is periodically interrupted by a generation of sex. The periodic release of hidden genetic variance by the hatching of sexually produced propagules allows a population of cyclical parthenogens to rapidly adjust to potentially

new selective pressures. Such adjustments are important, for example, in intermittent *Daphnia* populations that are annually exposed to vertebrate and invertebrate predators that select against large and small prey sizes, respectively, and whose densities are highly dependent on climatic conditions at the onset of the growing season.

Because of the extremely severe cytogenetic and ecological barriers that must be overcome for a successful transition from bisexuality to cyclical parthenogenesis (Lynch 1983*b*), those few cyclical parthenogens now known to exist may be products of former genetic revolutions, descendants of single and extremely rare individuals that suddenly and nearly perfectly met the challenges of cyclical parthenogenesis. Once this transition has been made, the cyclical parthenogens may themselves be capable of generating secondary genetic revolutions. When selection is intense, mutation rates high, and sex infrequent, a change in \bar{g} as much as 3 or 4 phenotypic standard deviations can occur in a single generation following sex. Take the following realistic values for a cyclical parthenogen engaging in sex every 10 generations: $V_m/V_e = 0.001$, $n = 50$, $\theta - \bar{g} = 8.0$, and $V_w/V_e = 1.0$ giving 36% selective mortality and $\hat{V}_g = 1.165$ in the generation following sex. The average phenotypic standard deviation over the life cycle is 1.20 and $\Delta\bar{g} = 2.94$ in the first generation following sex (by eq. [5]), giving $\Delta\bar{g}/\sqrt{V_T} = 2.45$. Thus, cyclical parthenogenesis provides a feasible mechanism for quantum evolution that does not depend on macromutation.

This aspect of cyclical parthenogenesis takes on broader significance when one considers that the majority of well-studied obligate parthenogens are imperfectly reproductively isolated from their bisexual ancestors (M. Lynch, in prep.). Frequently, fertilization of a normally parthenogenetic female results in an immediate conversion of her progeny to bisexuality. Often these progeny have extraordinary phenotypes and are either inviable or infertile, the two-headed and two-trunked progeny resulting from the backcrosses of parthenogenetic lacertid lizards with their bisexual parental species being an extreme example (Darevsky 1966). In other cases, some or all of the progeny are functional bisexuals (Haskins et al. 1960; Narbel-Hofstetter 1962; Benazzi-Lentati 1966; Schultz 1969; Rössler and DeBach 1972; Templeton 1982). Such events may lead to local extinctions of parthenogenetic species and/or an assimilation of many of the genes previously hidden in the parthenogens into the bisexual race (see Vrijenhoek [1979] for a suggestive example in *Poeciliopsis*). The release of hidden genetic variance may be especially dramatic in cases of introgression between obligate parthenogens and related bisexual species when the obligate parthenogens have been isolated for hundreds or even thousands of generations. Thus, although these kinds of events may be extremely rare, over evolutionary time they may have played a very significant role in organic evolution by periodically providing populations with the genetic variance necessary for rapid and radical evolutionary change.

This is not a mechanism that can be discredited on the basis of the rarity of parthenogenesis. Almost all well-studied animal species show some potential for parthenogenesis (White 1973), and many taxonomic groups that do not currently have unisexual representatives may have had them in the past. Indeed, partheno-

genesis may be much more common in nature than is currently believed since it is generally only sought when a population with an extremely biased sex ratio is located. Most importantly, this is an evolutionary mechanism that is highly amenable to short-term experimental investigation with many unisexual-bisexual complexes of invertebrates.

For the above reasons, it is important that future investigations with parthenogenetic species measure the components of phenotypic variance (V_g and V_e), the rate at which new genetic variance is generated via mutation (V_m), and the amount and rate of accumulation of hidden genetic variance (V_{gh}). The estimation of V_g and V_e is particularly straightforward for clonally reproducing populations, since in the absence of genotype-environment covariance, maternal effects, or common family environment they are respectively equal to the between- and within-clone variance.

There are two ways to estimate V_m . If it can be verified that a population is at equilibrium with respect to expressed genetic variance (either by a periodic analysis of V_g or by a comparison of V_g from several populations) and the width of the fitness function ($\sqrt{V_w}$) can be determined, then V_m can be directly determined from natural populations by use of equation (7). If this is not possible, then V_m can be determined in the laboratory by isolating multiple lines from a single clone, maintaining them without selection, and periodically measuring the between-line variance (V_g). As can be seen from equation (6), in the absence of selection and with initial expressed genetic variance equal to zero,

$$V_g(t) = V_m \cdot t,$$

so that V_m can be estimated from the slope of a regression of V_g on t . Since V_m is such a critical component of phenotypic selection models and also independent of the selection function, many additional opportunities, such as the estimation of the response to alternative fitness functions, arise once an estimate of V_m is available.

Cyclical parthenogens offer opportunities for measuring the rate of accumulation of hidden genetic variance and hence for testing several assumptions of the model. From equation (19) it can be seen that, for traits with large effective numbers of loci, the increase in the level of expressed genetic variance following a generation of random mating will be

$$\Delta V_g \approx \left[1 - \frac{(1-r)}{2} \right] [V_{gh}(t)]_w$$

so that

$$[V_{gh}(t)]_w \approx \frac{2\Delta V_g}{1+r}.$$

Thus, even if r is unknown, absolute limits can be put on $[V_{gh}(t)]_w$ by setting $r = 0$ and 0.5. Furthermore, a periodic analysis of ΔV_g with a controlled population could be used to test equation (18). Such a test would be most easily performed with a laboratory population denied sex and subjected to constant selection long

enough to attain its equilibrium level of expressed genetic variance for obligate parthenogenesis. A regression of ΔV_g , determined by periodically mating a random subset of the otherwise unisexual base population, on t should be linear according to (18).

Finally, since the dynamics of genetic variance are independent of the optimal phenotype under stabilizing selection approximated by a Gaussian fitness function, a periodic determination of V_g following a phase of sex in a cyclical parthenogen could be used to estimate the width of the fitness function, and hence the intensity of selection, by the solution of (6). Since V_m is small in comparison to V_g , it can be ignored without significantly altering the short-term solution of (6).

SUMMARY

Using phenotypic selection models directly coupled to the polygenic system, we examine the validity of the common assertion that obligate parthenogenesis is an evolutionary dead end. As is true for bisexual organisms, an equilibrium level of genetic variance is attained under obligate parthenogenesis when the input via mutation is balanced by the output via selection. We show that for most parthenogens this equilibrium will be reached within a few hundred generations. When the ability of ameiotic parthenogens to utilize the dominance and epistatic components of genetic variance and possible elevated mutation rates and reduced levels of environmental sensitivity in parthenogens are accounted for, it becomes clear that the rate of phenotypic evolution of well-established parthenogens may often approach or even exceed that under obligate bisexuality.

We also examine the consequences of periodic sex. By exploiting hidden genetic variance released by sex, cyclical parthenogenesis periodically allows much higher rates of phenotypic evolution than can ever be attained under obligate bisexuality. In the long run, rates of phenotypic evolution are approximately independent of the frequency of sex for populations that have attained their equilibrium levels of genetic variance. It is suggested that cyclical parthenogenesis is rare not because of any inherent disadvantages, but because of the extremely stringent requirements necessary for the transition to and maintenance of such a complex life cycle. The possibility that occasional backcrosses between parthenogens and their bisexual parental species may play an important role in organic evolution is explored, and techniques are suggested for the future analysis of parthenogenetic systems.

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