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Males, Parthenogenesis, and the Maintenance of Anisogamous Sex

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The problem of the maintenance of anisogamous sex is addressed by considering the effect of fertilization on the fitness of parthenogenetic females when such fertilization yields inviable triploid progeny. We consider four types of parthenogenesis: (i) apomixis, (ii) homogametic amphimixis, (iii) heterogametic amphimixis, and (iv) homogametic automixis. Homozygous sexual populations are genetically stable if males or selection eliminate the excess females produced by heterozygous parthenogenetic genotypes. Homozygous parthenogenetic populations are stable if the parthenogenetic output of homozygotes exceeds that of heterozygotes. In turn, sex can only invade heterozygous parthenogenetic populations when sexual output of parthenogens is larger than their parthenogenetic output. The existence of interior stable equilibria generally requires the instability of at least one boundary and some degree of heterosis. In a two-locus model, we study the evolution of mechanisms protecting either sex or parthenogenesis in reproductively polymorphic populations. We find that males do not respond to the presence of parthenogenesis in such a way as to eliminate it, but parthenogenesis is subject to selective pressures increasing reproductive isolation, and thus the success of parthenogenesis. The results suggest that reproductively polymorphic populations are ephemeral.

1. Introduction

One of the major sources of embarassment for the theory of evolution is its apparent inability to explain the maintenance of anisogamous sex (Maynard Smith, 1978*a*; Bell, 1982). It is well known that sex is subject to a nominal two-fold fitness disadvantage over newly-arisen apomictic parthenogenesis, all other things being equal. This disadvantage, called the cost of meiosis, can be mitigated under some circumstances (e.g. Uyenoyama, 1984), but by themselves these mechanisms are not powerful enough to provide general protection to sex.

Some evolutionary biologists are inclined to see the emergence of parthenogenesis as a "cytological tour de force" (White, 1978), ineluctably giving rise to low fitnesses in new parthenogenetic lineages. This view renders the problem of the maintenance of sex trivial, in that parthenogenetic forms having viability and fertility comparable to that of their immediate sexual ancestors are considered impossible. In support of this view, the parthenogenetic output of tychoparthenogenetic species, which normally reproduce sexually but display occasional parthenogenesis, is subject to heavy embryo mortality (Templeton, 1982). If tychoparthenogenesis is indeed a primordial condition which has often led to ameiotic parthenogenesis through

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clonal selection, as supposed by Templeton (1982), the fitness disadvantage of emerging parthenogenesis could be greater than the cost of meiosis. On the other hand, this very difficulty perhaps suggests that this is not the route by which parthenogenesis has normally evolved (cf. Maynard Smith, 1978*a*). Another body of evidence suggests that parthenogenesis could emerge as fully functional macromutations (Darevsky, 1983; White 1970, 1973, 1978; Marshall & Brown, 1981; Uzzell, 1964; Uzzell & Darevsky, 1975). Although this type of mutation is considered rare, such parthenogens are expected to enjoy a sizeable advantage over their sexual ancestors. These mutations can also be triggered by hybridogenesis (e.g. Parker & Selander, 1976; Maynard Smith, 1978*a*,*b*; Cole, 1979; Dawley *et al.*, 1985). In a sense, these parthenogenetic forms are the ones that give a meaning to the "cost of meiosis".

It has been suggested that males could cause "reproductive wastage" (White & Contreras, 1979) to newly emerged parthenogens by inducing lethal triploidy to their unreduced eggs upon fertilization (Stalker, 1954; Henslee, 1966; Ikeda & Carson, 1973; White, 1973; Williams, 1975; Cuellar, 1977; White & Contreras, 1979; Dawley *et al.*, 1985). Intuitively, it seems reasonable to suppose that male fertilization of parthenogenetically produced eggs could protect sex against invasion by parthenogenesis. This protective role of males must also depend on the degree of reproductive isolation of parthenogenetic lineages from the parent sexual population. Regardless of the mutational process leading to parthenogenesis, we expect that early parthenogenetic variants would mate with the parent form(s) at some frequency (cf. Uyenoyama, 1984; Crew *et al.*, 1985).

In spite of the plausibility of this mechanism for the protection of sex, theoretical evolutionary biologists have apparently neglected its study. To correct this situation, we propose a collection of models from which we obtain the degree of reproductive isolation necessary for invasion by various types of parthenogenesis. We also consider the possibility that parthenogenetic variants could be affected by viability and fertility selection. In addition, we consider the subsequent evolution of traits that affect the success of the two modes of reproduction, including male fertilization potential, female mating behaviour, genital tract incompatibility and egg-surface exclusion of sperm.

2. One Locus Model

(A) MODEL SET-UP

In this model, summarized in Table 1, infinite population size, discrete generations, and a 1:1 sex ratio of fertilized ova are assumed. We take allele A_1 to confer sexual reproduction when homozygous and allele A_2 to confer parthenogenetic reproduction when homozygous. The proportion of females of genotype A_iA_j in the population of males and females is defined by P_{ij} and the proportion of A_iA_j males by Q_{ij}. Viability is affected by the parthenogenesis allele, a proportion 1-vu of males surviving to reproductive age when heterozygous, and a proportion 1-u when homozygous, with viabilities of 1-mn for heterozygous females and 1-m for homo-

TABLE 1

Selection scheme for one-locus model

	Male phenotypes		
Genotype	A_1A_1	A_1A_2	A_2A_2
Frequency	Q_{11}	Q_{12}	Q_{22}
Viability	1	$1-vu$	$1-u$
Virility	c	ce	cf
	Female phenotypes		
Genotype	A_1A_1	A_1A_2	A_2A_2
Frequency	P_{11}	P_{12}	P_{22}
Viability	1	$1-mn$	$1-m$
Fertility	1	$1-hs$	$1-s$
	Egg types		
Unred. unfert.	0	$(1-bX)d$	$1-aX$
Unred. fert.	0	dbX	aX
Red. unfert.	$1-X$	$(1-d)(1-gX)$	0
Red. fert.	X	$(1-d)gX$	0

Where: $X = c[Q_{11}+Q_{12}(1-vu)e+Q_{22}(1-u)f][P_{11}+P_{12}(1-mn)+P_{22}(1-m)]^{-1}$

zygous females. We also assume that genotype affects virility by a factor e in heterozygotes and by a factor f in homozygotes.

At reproduction, different male genotypes contribute to a sperm pool of size

$$c[Q_{11}+Q_{12}(1-vu)e+Q_{22}(1-u)f]$$

in proportion to their relative abundance. The size of the sperm pool thus varies with the sex ratio in the population. The fertilization rate of sexual females is in turn directly proportional to the size of the sperm pool, approximating the biological situations of monoparity and monogamy without excess males. Intuitively, we expect c, defined as virility, to be high in viable sexual populations. (This parameter is the object of a specific treatment in the two-locus model.) Population size is kept constant through normalization by w, the average fitness, explicitly defined in the recursion systems. The total frequency of matings under the assumption of random female choice by males is taken to be

$$[Q_{11}+Q_{12}(1-vu)e+Q_{22}(1-u)f].$$

The frequency of successful $P_{12}Q_{22}$ matings is then

$$cP_{12}(1-mn)Q_{22}f[P_{11}+P_{12}(1-mn)+P_{22}(1-m)]^{-1}.$$

The total frequency of all successful matings with A_1A_1 females is then

$$cP_{11}[Q_{11}+Q_{12}(1-vu)e+Q_{22}(1-u)f][P_{11}+P_{12}(1-mn)+P_{22}(1-m)]^{-1},$$

or $P_{11}X$, where X is the effective sex ratio.

We suppose that spermatozoa retain to some degree the ability to penetrate parthenogenetic eggs, a fraction a for homozygous A_2A_2 mothers and a fraction b for heterozygous A_1A_2 mothers, inducing lethal triploidy. These parameters effectively represent the degree of isolation of the parthenogenetic lineage. A_2A_2 females are obligate parthenogens, while a proportion d of total heterozygous output of eggs is unreduced. The viability of A_1A_1, A_1A_2 and A_2A_2 eggs is taken to be 1, $1-hs$ and $1-s$ without loss of generality. The proportion d can also be interpreted as a dominance parameter, $d=1$ for dominance of parthenogenetic reproduction or $d=0$ for dominance of sexual reproduction. The proportion of potentially parthenogenetic eggs killed by males is the product of the probability of mating and the probability of inducing triploidy in unreduced eggs. Then, the parthenogenetic output of homozygotes is $(1-s)\,(1-aX)$ and that of heterozygotes is $(1-hs)\,(1-bX)d$. If the haploid ova of heterozygous females can be fertilized more easily than their unreduced eggs, the probability of reduced egg fertilization in mated heterozygous females is defined as g, $g \geq b$. If the compatibility with spermatozoa does not depend on egg ploidy, $g = b$. The sexual output of heterozygotes is then $(1-hs)\,(1-d)gX$. Table 1 gives the complete selection scheme.

Three types of parthenogenesis are recognized (Blackwelder & Shepherd, 1981). In ameiotic or apomictic parthenogenesis, meiosis is bypassed and the progeny are genetically identical to the mother. In amphimictic parthenogenesis, either an extra genome complement is withheld during meiosis or the follicular genome doubles before meiosis, resulting in a Hardy-Weinberg genotype distribution for heterozygous maternal loci. If females are homogametic, only females are produced. If they are heterogametic, two-thirds of the progeny are female and one-third are male, assuming lethal *WW* caryotype. In automictic parthenogenesis, meiosis is complete but the gametic genome doubles, resulting in complete homozygosity. This type of parthenogenesis is a successful method of eliminating male progeny for homogametic females, but not for heterogametic females. Therefore, the latter are not considered here.

(B) ANALYSIS

The recursion systems used throughout the analysis are presented in the appendix. We first performed a boundary stability analysis on the sexual boundary, with $\text{Freq}\{A_1\} \approx 1$ and with $X \approx c$, and on the parthenogenetic boundary, with $\text{Freq}\{A_1\} \approx 0$ and $X \approx 0$. We chose independent perturbation magnitudes for each genotype frequency, unless otherwise specified. We obtained the conditions for local asymptotic convergence to the sexual boundary and the corresponding condition for local stability of the parthenogenesis boundary. In addition, we considered the stability of reproductive mode, which may not be affected by the presence of either allele, depending on dominance d. The system thus allows two types of polymorphism: genetic and reproductive. The sexual boundary is characterized by an effective sex ratio equal to that of the corresponding sexual population, regardless of the presence of the parthenogenesis allele. Similarly, the parthenogenetic boundary satisfies the condition of absence of males, or $X = 0$. This latter condition does not,

however, apply to heterogametic amphimixis, where the minimal effective sex ratio is

$$c[Q_{12}^*(1-vu)e+Q_{22}^*(1-u)f][P_{12}^*(1-mn)+P_{22}^*(1-m)]^{-1}$$

where * denotes frequencies at equilibrium. Indeed, in heterogametic amphimixis, females continuously produce males by meiotic segregation though they are useless or even deleterious. The results of the perturbation analysis are collected in Table 2.

TABLE 2

Conditions for boundary stability, one-locus model

Stability of homozygous sexual populations

Apomixis

$$c\{2-(1-vu)e+(1-hs)(1-mn)[2bd-(1-d)g]+\phi[1+(1-hs)(1-mn)[2bd-(1-d)g]+(1-s)(1-m)a]\}>2[1+\phi](1-hs)(1-mn)d$$

Homogametic amphimixis

$$c\{2-(1-vu)e+(1-hs)(1-mn)[2bd-(1-d)g]+\phi[1+(1-hs)(1-mn)[bd-(1-d)g/2]+(1-s)(1-m)a]\}$$
$$>2(1-hs)(1-mn)d+\phi[(1-hs)(1-mn)d+(1-s)(1-m)]$$

Heterogametic amphimixis

$$c\{4-2(1-vu)e+(1-hs)(1-mn)[3bd-2(1-d)g]+[\phi/4][6-(1-vu)e-(1-u)f+(1-hs)(1-mn)[3bd-(1-d)g+3(1-s)a]\}$$
$$>3(1-hs)(1-mn)d+[\phi/4][(1-hs)(1-mn)d+(1-s)(1-m)]$$

Automixis

$$c\{2-(1-vu)e+(1-hs)(1-mn)[2bd-(1-d)g]+\phi[1+(1-s)(1-m)a]\}$$
$$>2\{(1-hs)(1-mn)d+\phi(1-s)(1-m)\}$$

Stability of sexual reproduction

Apomixis

$$c[\phi+\beta]>2\{[1+\phi](1-hs)(1-mn)(1-bc)d+[\chi+\beta](1-s)(1-m)(1-ac)\}$$

Homogametic amphimixis

$$c[3\phi+4\beta]>[8+4\phi](1-hs)(1-mn)(1-bc)d+[8\chi+8\beta+2\phi](1-s)(1-m)(1-ac)$$

Heterogametic amphimixis

$$c[3\phi+4\beta]>[24+6\phi](1-hs)(1-mn)(1-bc)d+[24\chi+12\beta+3\phi](1-s)(1-m)(1-ac)$$

Automixis

$$c\phi>4(1-hs)(1-mn)(1-bc)d+[4\chi+4\beta+2\phi](1-s)(1-m)(1-ac)$$

Stability of homozygous parthenogenesis

Apomixis

$$2[1+\phi](1-s)(1-m)>[1+2\phi](1-hs)(1-mn)d$$

TABLE 2 (*continued*)

Homogametic amphimixis

$$2(1-s)(1-m) > (1-hs)(1-mn)d$$

Heterogametic amphimixis

$$[8+3\phi](1-s)(1-m)[(1-m)-(1-u)fac/2]$$
$$>4cf(1-u)(1-hs)(1-mn)(1-d)g+(1-hs)(1-mn)d[4(1-m)-2(1-u)fbc]$$
$$+\phi\{cf(1-u)+cf(1-u)(1-hs)(1-mn)(1-d)g+(1-hs)(1-mn)d[(1-mn)-(1-u)fbc/2]\}$$

Automixis

$$[2+\phi](1-s)(1-m) > (1-hs)(1-mn)d$$

Stability of parthenogenesis

Apomixis

$$2[P^*_{12}(1-hs)(1-mn)d+P^*_{22}(1-s)(1-m)][P^*_{12}(1-mn)+P^*_{22}(1-m)] > \theta cP^*_{12}(1-hs)(1-mn)(1-d)g$$

Homogametic amphimixis

$$2[P^*_{12}(1-hs)(1-mn)d+P^*_{22}(1-s)(1-m)][P^*_{12}(1-mn)+P^*_{22}(1-m)]$$
$$> \theta cP^*_{12}[(1-hs)(1-mn)(1-d)g+1/4]$$

Automixis

$$2[P^*_{12}(1-hs)(1-mn)d+P^*_{22}(1-s)(1-m)][P^*_{12}(1-mn)+P^*_{22}(1-m)]$$
$$> \theta cP^*_{12}[(1-hs)(1-mn)(1-d)g+1/2]$$

Where the parameters are as defined in Table 1, except:

$\phi = [P_{12}-Q_{12}]/Q_{12}$

$\chi = Q_{12}/Q_{22}$

$\beta = [P_{22}-Q_{22}]/Q_{12}$

$\theta = [Q_{11}+Q_{12}(1-vu)e+Q_{22}(1-u)f][Q_{11}+Q_{12}+Q_{22}]^{-1}$.

and P_{ij}, Q_{ij} are perturbation frequencies.

The conditions for stability of homozygous sexual populations show that sex can resist parthenogenetic invasion if the viability or fertility of parthenogens is depressed, or if males have copulatory access to parthenogenetic females. The stability criterion of sex under ameiotic and homogametic parthenogenetic perturbation states that the parthenogenetic allele is eliminated if the final fitness of parthenogenetic individuals, combining their sexual and parthenogenetic output, is less than that of the sexual form. If triploidy is the only selective pressure associated with parthenogenesis, sex is stable if at least half the parthenogenetic eggs are destroyed. On the other hand, the parthenogenesis allele can be retained in a sexual population if parthenogenesis is recessive, and heterosis compensates for the wastage of parthenogenetic eggs.

The corresponding local stability criterion for parthenogenesis allele fixation depends strictly on the relative viability and fertility of heterozygous and homozygous apomictic lineages. Homogametic amphimictic heterozygotes must be twice as fit as their homozygous counterparts in order to invade, and automictic heterozygotes cannot invade, since ϕ is large. The retention of sexual parameters in the condition for fixation of heterogametic amphimixis reflects the retention of males in such parthenogenetic populations, even though their effect is potentially deleterious to females. The stability of the parthenogenetic boundary under small sexual perturbations is generally insured by the stability of homozygous parthenogenesis. Generally, sex can invade heterozygous female populations if sex is dominant over parthenogenesis. While the conditions for male invasion are more easily met in homogametic amphimictic and automictic heterozygous populations, the admissibility of these heterozygote populations depends on extreme viability or fertility depression of homozygote parthenogenetic boundaries if males contribute to the stability of the sexual boundary.

Critical points of the system are genotype frequency values for which

$$\Delta P_{ij} = 0 \quad \text{and} \quad \Delta Q_{ij} = 0, \qquad \Delta P_{ij} = P_{ij}' - P_{ij}.$$

We reduced the system to the conditions

$$\Delta P_{11} = 0, \qquad \Delta Q_{22} = 0 \quad \text{and} \quad \Delta P_{12} + P_{22} - \Delta Q_{12} - \Delta Q_{22} = 0,$$

which is a necessary and sufficient set of conditions for the existence of critical points. From the recursion system, we find two trivial critical points at Freq $\{A_1\} = 1$ and Freq $\{A_1\} = 0$. The other critical points require:

$$\begin{aligned}
X^* &= \{P_{12}^*(1-hs)(1-mn)d + P_{22}^*(1-s)(1-m)\} \\
&\quad \times \{P_{12}^*(1-hs)(1-mn)[bd-(1-d)g(1-\delta)] \\
&\quad + P_{22}^*(1-s)(1-m)a - P_{11}^*\}^{-1} \\
&= \{P_{12}^*(1-hs)(1-mn)d + P_{22}^*(1-s)(1-m)\} \\
&\quad \times \{P_{11}^*\alpha + P_{12}^*(1-hs)(1-mn)[bd+(1-d)g\alpha] + P_{22}^*(1-s)(1-m)a\}^{-1} \\
&= \{P_{12}^*(1-hs)(1-mn)d + P_{22}^*(1-s)(1-m)\} \\
&\quad \times \{P_{11}^*(1-2\gamma) + P_{12}^*(1-hs)(1-mn)[(1-d)g(1-\gamma) - bd] \\
&\quad - P_{22}^*(1-s)(1-m)a\}^{-1}
\end{aligned}$$

where:

$$\delta = [Q_{12}^*(1-vu)e + 2Q_{22}^*(1-u)f][8Q_{22}^*(Q_{11}^* + Q_{12}^*(1-vu)e + Q_{22}^*(1-u)f)]^{-1}$$

$$\alpha = [1 - 2Q_{11}^* - 2Q_{12}^*2Q_{22}^*][2Q_{11}^* + 2Q_{12}^* + 2Q_{22}^*]^{-1}$$

$$\gamma = [2Q_{11}^* + Q_{12}^*(1-vu)e][16Q_{11}^*(Q_{11}^* + Q_{12}^*(1-vu)e + Q_{22}^*(1-u)f]^{-1}.$$

This system can be reduced to:

$$P_{11}^*/P_{12}^*(1-hs)(1-mn)(1-d)g = [Q_{12}^*(1-vu)e/4Q_{22}^* + 1/2]\theta^{-1} - 1$$

$$P_{22}^*(1-s)(1-m)a + bd/(1-d)g$$
$$= [Q_{12}^*(1-vu)e/2Q_{22}^* + 1][1/2 - 1/8(Q_{11}^* + Q_{12}^* + Q_{22}^*)$$
$$-2Q_{11}^* + Q_{12}^*(1-vu)e/32Q_{11}^*(Q_{11}^* + Q_{12}^*(1-vu)e + Q_{22}^*(1-u)f)]\theta^{-1}$$
$$-2Q_{11}^* + Q_{12}^*(1-vu)e/32Q_{11}^*(Q_{11}^* + Q_{12}^*(1-vu)e + Q_{22}^*(1-u)f)$$

where θ is as defined in Table 2.

These results show that critical surfaces are either points or manifolds along which X^* remains constant for all genotype frequencies. It also suggests that frequency perturbations that do not change the value of X^* may simply move the system on a critical manifold. Actually, $\Delta X = 0$ is a necessary but not sufficient condition for the existence of a critical point for the complete system. We write ΔX in the compact form:

$$\Delta X = c\{[Q_{11}' + Q_{12}'(1-vu)e + Q_{22}'(1-u)f][P_{11} + P_{12}(1-mn) + P_{22}(1-m)]$$
$$-[Q_{11} + Q_{12}(1-vu)e + Q_{22}(1-u)f][P_{11}' + P_{12}'(1-mn) + P_{22}'(1-m)]\}$$
$$\times\{[P_{11} + P_{12}(1-mn) + P_{22}(1-m)][P_{11}' + P_{12}'(1-mn) + P_{22}'(1-m)]\}^{-1}.$$

This expression admits one trivial solution at $Q_{11} + Q_{12} + Q_{22} = 0$, which corresponds to the parthenogenetic boundary. The expression is a quadratic form, yielding a maximum of two distinct solutions for $\Delta X = 0$. Since $\Delta X = 0$ is a necessary but not sufficient condition for critical points of the system, the number of solution planes satisfying the expression for X^* should not exceed two, discounting trivial solutions. It may happen that disjoint subsets of a manifold defined by $\Delta X = 0$ are actual solutions to X^*. Considering this, a repellor $\Delta X = 0$ manifold defines only unstable X^* solutions, either sources or saddles. On the other hand, if a $\Delta X = 0$ manifold is an attractor, population trajectories are expected to stay on the manifold, so that there is at least one attractor of unknown nature on $\Delta X = 0$. In the case of ΔX stability, the manifold could define any number of attractors satisfying X^*.

Since there are only two admissible $\Delta X = 0$ manifolds in the interior, the stability of one such manifold requires that at least one boundary be unstable, generally the sexual one. If the parthenogenetic boundary is stable, and the sexual unstable, and both manifolds arise in the interior, then an unstable $\Delta X = 0$ manifold is expected to separate the parthenogenetic boundary and the stable manifold. The parametric values required are $(1-hs)(1-mn) \gg bc$, $d < c(1-d)g$, $(1-s)(1-m) \ll ca$ and $(1-vu)e \gg 1$. As mentioned before, these conditions call for an unrealistic degree of heterosis. If the parthenogenetic boundary is also unstable, all stable solutions are expected to lie on the only admissible $\Delta X = 0$ manifold. The additional condition is then $(1-s)(1-m) < (1-hs)(1-mn)d$. Obviously, stable interior solutions exist for a very narrow parameter range which is biologically implausible, considering the degree of heterosis required. For this reason, reproductively polymorphic populations may be considered exceptional.

We tried to obtain explicit conditions for the stability of X^*, first using independent genotype perturbations, then by a Jacobian analysis. Unfortunately, the stability conditions for X^* depend on the magnitude of genotype frequencies. Even under all justifiable simplifications, the results are too complex to be readily interpreted. The only important result from this analysis is that $\Delta X = 0$ admits no solution if

$$(1 - hs)(1 - mn)d > cb$$

and if

$$(1 - s)(1 - m) > ca.$$

Then, the parthenogenetic boundary is globally stable. We turned to numerical analysis for further resolution of the interior dynamics, discussed in section 3.

3. Numerical Analysis

In order to explore more completely the internal dynamics of these systems we performed a numerical exploration of the evolution of apomictic parthenogenesis, as described in the one-locus model, using iterative calculation of solution trajectories. Three initial conditions were used: (i) almost entirely sexual, (ii) reproductively polymorphic, and (iii) almost entirely homozygotic parthenogenetic populations. Numerical solutions were calculated for a maximum of 1000 generations or until allelic frequency and sex ratio changes fell below 10^{-12}. Oscillatory behaviour was sought using change in sign of allelic frequency change or of sex ratio change as criteria.

For most parameters, three different values were used in all possible combinations, except for mn and m for which a single value was used. Additional numerical solutions were calculated in the absence of viability and fertility selection on females in conjunction with a wider range of virility parameters.

Tables 3 and 4 summarize the results of all these numerical solutions. While Table 3 gives some results for relatively low values of the male virility parameter, c, in realistic situations we expect c to be at its highest possible value, from the results of the two-locus model. Table 4 gives the runs made for high values of c in the absence of selection on females.

In these runs, the sexual boundary was stable in 21% of the cases where convergence occurred, with elimination of allele A_2 in 16% of the convergent cases. (This means that in 5% of the cases, reproduction remained exclusively sexual, but the allele for parthenogenesis was retained at some frequency.) Reproductively polymorphic populations were noted in 14% of the cases, although we do not know if these results were in fact due to attractor sets located in the interior of the state-space. Such results arose when (i) neither allele was completely dominant, (ii) when the reduction in net viability of unreduced eggs due to fertilization is less than two-fold, and (iii) and when parthenogenetic homozygotes were subject to a substantial fitness reduction. In effect, reproductive polymorphism requires net heterozygote advantage, an unlikely condition in actual populations. Simultaneous stability of two equilibria was noted in 35% of the cases. This situation arises only when the average

TABLE 3

Numerical analysis of apomictic parthenogenesis

(a) Selection on females†

Population at convergence	Initial conditions: Homo. partheno.¶	Reprod. poly.‖	Sexual††
Homo. partheno.	12 939	10 920	7389
Poly. partheno.	4 374	4 362	2952
Reprod. poly.	0	396	2568
Poly. sexual	0	303	393
Homo. sexual	0	1 122	4779
Non-convergent§	2 916	2 580	1593

(b) No selection on females‡

Population at convergence	Initial conditions: Homo. partheno.¶	Reprod. poly.‖	Sexual††
Homo. partheno.	11 664	11 664	9234
Poly. partheno.	5 832	5 832	4260
Reprod. poly.	0	0	1212
Poly. sexual	0	0	558
Homo. sexual	0	0	1722
Non-convergent§	0	0	510

† $m = mn = 0{\cdot}1$ $a = b = d = g = c = ce = cf = hs = s = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$.
‡ $hs = s = mn = m = 0{\cdot}0$ $a = b = d = g = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$, $c = ce = cf = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9, 0{\cdot}95, 0{\cdot}99, 1{\cdot}0$.
§ ΔX or $\Delta A_1 > 10^{-12}$ after 1000 generations.
¶ $P_{11} = Q_{11} = 0{\cdot}0$, $P_{12} = 0{\cdot}001$, $Q_{12} = 0{\cdot}0001$, $P_{22} = 0{\cdot}9989$, $Q_{22} = 0{\cdot}0$.
‖ $P_{11} = 0{\cdot}05$, $Q_{11} = 0{\cdot}0$, $P_{12} = Q_{12} = 0{\cdot}45$, $P_{22} = 0{\cdot}05$, $Q_{22} = 0{\cdot}0$.
†† $P_{11} = Q_{11} = 0{\cdot}4999$, $P_{12} = Q_{12} = 0{\cdot}0001$, $P_{22} = Q_{22} = 0{\cdot}0$.

TABLE 4

Numerical analysis of apomictic parthenogenesis

No selection on females† $c = 0{\cdot}95/0{\cdot}99/1{\cdot}0$:

Population at convergence	Initial conditions: Homo. partheno.§			Reprod. poly.¶			Sexual‖		
Homo. partheno.	1944/	1944/	1944	1944/	1944/	1944	1578/	1290/	888
Poly. partheno.	972/	972/	972	972/	972/	972	672/	480	432
Reprod. poly.	0/	0/	0	0/	0/	0	60/	180/	930
Poly. sexual	0/	0/	0	0/	0/	0	132/	144/	138
Homo. sexual	0/	0/	0	0/	0/	0	420/	438/	462
Non-convergent‡	0/	0/	0	0/	0/	0	54/	384/	66

† $hs = s = mn = m = 0{\cdot}0$, $a = b = d = g = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$, $ce = cf = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9, 0{\cdot}95, 0{\cdot}99, 1{\cdot}0$.
‡ ΔX or $\Delta A_1 > 10^{-12}$ after 1000 generations.
§ $P_{11} = Q_{11} = 0{\cdot}0$, $P_{12} = 0{\cdot}001$, $Q_{12} = 0{\cdot}0001$, $P_{22} = 0{\cdot}9989$, $Q_{22} = 0{\cdot}0$.
¶ $P_{11} = 0{\cdot}05$, $Q_{11} = 0{\cdot}0$, $P_{12} = Q_{12} = 0{\cdot}45$, $P_{22} = 0{\cdot}05$, $Q_{22} = 0{\cdot}0$.
‖ $P_{11} = Q_{11} = 0{\cdot}4999$, $P_{12} = Q_{12} = 0{\cdot}0001$, $P_{22} = Q_{22} = 0{\cdot}0$.

effect of fertilization upon unreduced eggs over the two parthenogenetic genotypes exceeds one-half (i.e., $a+b>1$). In all these cases of simultaneous stability, the parthenogenetic boundary is always stable. In Table 4, all populations which started from reproductively polymorphic initial conditions converged towards the parthenogenetic boundary.

Other parameters of importance in the system are allelic dominance, d, and probability of fertilization of unreduced eggs, a and b. Tables 5, 6 and 7 summarize the numerical analyses of these parameters for high values of c. For these Tables, *with* selection on females, 61% of the convergent cases displayed stability of sexual reproduction, and 59% completely eliminated allele A_2. Sixteen percent of the cases converged towards the sexual boundary from reproductively polymorphic initial conditions, 15% completely eliminating allele A_2. Reproductively polymorphic populations still made up 26% of the convergent cases, all from sexual initial conditions. Simultaneous stability of two equilibria was noted in 85% of the cases of convergence.

TABLE 5

Numerical analysis of apomictic parthenogenesis

(a) Selection on females† $d = 0{\cdot}1/0{\cdot}5/0{\cdot}9$

	Initial conditions								
Population at convergence	Homo. partheno.¶			Reprod. poly.‖			Sexual††		
Homo. partheno.	1944/	1458/	729	1503/	1236/	717	243/	267/	189
Poly. partheno.	0/	486/	972	0/	474/	972	0/	36/	270
Reprod. poly.	0/	0/	0	0/	0/	0	480/	525/	534
Poly. sexual	0/	0/	0	66/	0/	0	132/	18/	0
Homo. sexual	0/	0/	0	591/	186/	123	1332/	1305/	1194
Non-convergent§	243/	243/	243	27/	291/	375	0/	36/	0

(b) No selection on females‡ $d = 0{\cdot}1/0{\cdot}5/0{\cdot}9$

	Initial conditions								
Population at convergence	Homo. partheno.¶			Reprod. poly.‖			Sexual††		
Homo. partheno.	1944/	1944/	0	1944/	1944/	0	810/	1080/	288
Poly. partheno.	0/	0/	1944	0/	0/	1944	0/	0/	912
Reprod. poly.	0/	0/	0	0/	0/	0	354/	372/	384
Poly. sexual	0/	0/	0	0/	0/	0	216/	66/	0
Homo. sexual	0/	0/	0	0/	0/	0	366/	318/	216
Non-convergent§	0/	0/	0	0/	0/	0	198/	108/	144

† $m = mn = 0{\cdot}1$, $c = 0{\cdot}9$, $a = b = g = ce = cf = hs = s = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$.
‡ $hs = s = mn = m = 0{\cdot}0$, $a = b = g = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$, $c = 0{\cdot}99, 1{\cdot}0$, $ce = cf = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9, 0{\cdot}95, 0{\cdot}99, 1{\cdot}0$.
§ ΔX or $\Delta A_1 > 10^{-12}$ after 1000 generations.
¶ $P_{11} = Q_{11} = 0{\cdot}0$, $P_{12} = 0{\cdot}001$, $Q_{12} = 0{\cdot}0001$, $P_{22} = 0{\cdot}9989$, $Q_{22} = 0{\cdot}0$.
‖ $P_{11} = 0{\cdot}05$, $Q_{11} = 0{\cdot}0$, $P_{12} = Q_{12} = 0{\cdot}45$, $P_{22} = 0{\cdot}05$, $Q_{22} = 0{\cdot}0$.
†† $P_{11} = Q_{11} = 0{\cdot}4999$, $P_{12} = Q_{12} = 0{\cdot}0001$, $P_{22} = Q_{22} = 0{\cdot}0$.

TABLE 6

Numerical analysis of apomictic parthenogenesis

(a) Selection on females† $b = 0{\cdot}1/0{\cdot}5/0{\cdot}9$:

Population at convergence	Initial conditions: Homo. partheno.¶			Reprod. poly.‖			Sexual††		
Homo. partheno.	1377/	1377/	1377	1164/	1146/	1146	240/	216/	243
Poly. partheno.	486/	486/	486	483/	486/	477	279/	27/	0
Reprod. poly.	0/	0/	0	0/	0/	0	429/	642/	468
Poly. sexual	0/	0/	0	27/	18/	21	39/	57/	54
Homo. sexual	0/	0/	0	270/	294/	336	1164/	1245/	1422
Non-convergent§	324/	324/	324	243/	243/	207	36/	0/	0

(b) No selection on females‡ $b = 0{\cdot}1/0{\cdot}5/0{\cdot}9$:

Population at convergence	Initial conditions: Homo. partheno.¶			Reprod. poly.‖			Sexual††		
Homo. partheno.	1296/	1296/	1296	1296/	1296/	1296	810/	624/	744
Poly. partheno.	648/	648/	648	648/	648/	648	648/	264/	0
Reprod. poly.	0/	0/	0	0/	0/	0	228/	546/	338
Poly. sexual	0/	0/	0	0/	0/	0	48/	96/	138
Homo. sexual	0/	0/	0	0/	0/	0	120/	270/	510
Non-convergent§	0/	0/	0	0/	0/	0	90/	144/	216

† $m = mn = 0{\cdot}1$, $c = 0{\cdot}9$, $a = d = g = ce = cf = hs = s = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$.
‡ $hs = s = mn = m = 0{\cdot}0$, $a = d = g = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9$, $c = 0{\cdot}99, 1{\cdot}0$, $ce = cf = 0{\cdot}1, 0{\cdot}5, 0{\cdot}9, 0{\cdot}95, 0{\cdot}99, 1{\cdot}0$.
§ ΔX or $\Delta A_1 > 10^{-12}$ after 1000 generations.
¶ $P_{11} = Q_{11} = 0{\cdot}0$, $P_{12} = 0{\cdot}001$, $Q_{12} = 0{\cdot}0001$, $P_{22} = 0{\cdot}9989$, $Q_{22} = 0{\cdot}0$.
‖ $P_{11} = 0{\cdot}05$, $Q_{11} = 0{\cdot}0$, $P_{12} = Q_{12} = 0{\cdot}45$, $P_{22} = 0{\cdot}05$, $Q_{22} = 0{\cdot}0$.
†† $P_{11} = Q_{11} = 0{\cdot}4999$, $P_{12} = Q_{12} = 0{\cdot}0001$, $P_{22} = Q_{22} = 0{\cdot}0$.

Without selection on females, 22% of the cases resulted in stability of the sexual boundary, elimination of allele A_2 occurring in 18% of the cases. Reproductively polymorphic populations were obtained 21% of the time, all from sexual initial conditions. Dual stable equilibria occurred in 43% of the convergent cases.

From Table 5, we see that increasing the allelic dominance of parthenogenetic reproduction has relatively little effect on the stability of the sexual boundary, while it decreases the number of cases of polymorphic sexual populations. Its major effect lies on the parthenogenetic boundary, where it promotes the stability of heterozygous parthenogenetic populations at the expense of homozygous parthenogenetic populations. The effect of increasing b, or increasing the proportion of unreduced eggs killed by successful mating, shown in Table 6, is to reduce the stability of the parthenogenetic boundary, particularly heterozygote female populations, to the benefit of the sexual boundary. The effect of increasing a is similar to that of increasing b, but it is not sufficient to make the sexual boundary stable. Particularly evident in part (b) of Table 7, its prime effect is to create two stable equilibria, whether or not one lies on the sexual boundary. Parameter values giving rise to

TABLE 7

Numerical analysis of apomictic parthenogenesis

(a) Selection on females† $a = 0{\cdot}1/0{\cdot}5/0{\cdot}9$

	Initial conditions								
Population at convergence	Homo. partheno.¶			Reprod. poly.‖			Sexual††		
Homo. partheno.	1377/	1377/	1377	1191/	1167/	1098	675/	12/	12
Poly. partheno.	486/	486/	486	483/	483/	480	120/	93/	93
Reprod. poly.	0/	0/	0	0/	0/	0	648/	765/	126
Poly. sexual	0/	0/	0	21/	21/	24	30/	48/	72
Homo. sexual	0/	0/	0	258/	285/	357	702/	1257/	1872
Non-convergent§	0/	0/	0	234/	231/	228	12/	12/	12

(b) No selection on females‡ $a = 0{\cdot}01/0{\cdot}5/0{\cdot}9$

	Initial conditions								
Population at convergence	Homo. partheno.¶			Reprod. poly.‖			Sexual††		
Homo. partheno.	1296/	1296/	1296	1296/	1296/	1296	1512/	522/	144
Poly. partheno.	648/	648/	648	648/	648/	648	432/	246/	234
Reprod. poly.	0/	0/	0	0/	0/	0	0/	774/	336
Poly. sexual	0/	0/	0	0/	0/	0	0/	0/	282
Homo. sexual	0/	0/	0	0/	0/	0	0/	0/	900
Non-convergent§	0/	0/	0	0/	0/	0	0/	402/	48

† $m = mn = 0{\cdot}1$, $c = 0{\cdot}9$, $b = d = g = ce = cf = hs = s = 0{\cdot}1$, $0{\cdot}5$, $0{\cdot}9$.
‡ $hs = s = mn = m = 0{\cdot}0$, $b = d = g = 0{\cdot}1$, $0{\cdot}5$, $0{\cdot}9$, $c = 0{\cdot}99$, $1{\cdot}0$, $ce = cf = 0{\cdot}1$, $0{\cdot}5$, $0{\cdot}9$, $0{\cdot}95$, $0{\cdot}99$, $1{\cdot}0$.
§ ΔX or $\Delta A_1 > 10^{-12}$ after 1000 generations.
¶ $P_{11} = Q_{11} = 0{\cdot}0$, $P_{12} = 0{\cdot}001$, $Q_{12} = 0{\cdot}0001$, $P_{22} = 0{\cdot}9989$, $Q_{22} = 0{\cdot}0$.
‖ $P_{11} = 0{\cdot}05$, $Q_{11} = 0{\cdot}0$, $P_{12} = Q_{12} = 0{\cdot}45$, $P_{22} = 0{\cdot}05$, $Q_{22} = 0{\cdot}0$.
†† $P_{11} = Q_{11} = 0{\cdot}4999$, $P_{12} = Q_{12} = 0{\cdot}0001$, $P_{22} = Q_{22} = 0{\cdot}0$.

convergence on either boundary conformed to the boundary conditions derived analytically.

Additional numerical solutions were done with parameter values of $a = c = ce = cf = g = 1{\cdot}0$, $b = hs = mn = m = 0{\cdot}0$, $d = 0{\cdot}1$ and $s = 0{\cdot}95$, $0{\cdot}99$ and $0{\cdot}995$. As expected, these particular numerical solutions apparently converged to a unique asymptotically stable reproductively polymorphic equilibrium, fixation of parthenogenesis being eliminated as an attractor state by a high level of selection against the homozygotic parthenogen.

All of these results strongly support our analytical results with respect to the conditions required for reproductive polymorphism and the effect of parametric variation on the stability of the boundaries. No numerical solution gave rise to oscillatory behaviour, though we cannot exclude the possibility of limit cycles in this system.

In all of the numerical work just discussed, a maximum of two stable equilibria were found, which is a corollary of the maximum of two distinct critical manifolds predicted earlier. We conducted a last numerical search to test this conclusion. For

each parameter value, initial conditions were taken according to an increment schedule of 0·2 for each genotype frequency, as long as males and both alleles were present in the initial population. In effect, we tested convergence properties under a wide range of initial conditions. Parametric values of a, b and d were generated according to a similar schedule, while other parameters were kept at 1·0. Runs were made when parameters values ensured stability of both sexual and parthenogenetic boundary. For the sake of speed of execution, we took $P_{11} = Q_{11}$, which is true after one generation, but creates a bias in favour of the parthenogenetic basin of attraction. From our previous analysis, there cannot be any interior attractors under these conditions. In fact, all population trajectories converged to either one boundary or the other, sex in 91 cases, parthenogenesis in 4090. These additional results support our contention about the existence of a maximum of two disjoint sets of critical points.

4. Two-Locus Model

In this formulation, we suppose that parthenogenetic functions are carried at one locus, say A, and that other functions under study are carried at another locus, say B. Recombination distance between the two loci is taken as r, as usual. Since the full recursion systems for the various types of parthenogenesis are extremely lengthy, we do not give them here; they can be obtained by expansion of the one-locus model. To verify them, we derived the boundary stability conditions for allele A_1, which where found to be identical to the results of the one-locus model with $r = 0$. All other parameters in the two-locus model are as defined in the previous one-locus model.

(A) EVOLUTION OF VIRILITY

The arguments presented so far indicate that parthenogenesis can be eliminated rapidly from sexual populations, providing that males kill the excess of females produced by parthenogens by the induction of lethal triploidy. If parthenogenetic variants appear that could overcome viability, fertility or mating selection pressures, and establish themselves in a population, clonal selection would increase their fertility and viability.

Reproductively polymorphic populations can revert to sexual reproduction only if males become more effective in the presence of parthenogenesis. In order to study a conditional increase in viability or virility of males in the presence of parthenogenesis, we attribute to locus B the determination of virility and male viability. We take c to be the virility and viability of B_1B_1 males, $ce(1-vu)$ that of heterozygotes B_1B_2 and $cf(1-u)$ that of homozygotes B_2B_2, as defined in the previous model.

We derived the conditions for invasion and for fixation of allele B_2 in populations of arbitrary sex ratio ($0 \leqq X \leqq c$), for all types of parthenogenesis. The complete conditions are quite complex, depend on the magnitude of genotype frequencies, and differ somewhat between types of parthenogenesis. Under the additional

assumption of a significant sexual segment in the population, i.e. $X > 0(\varepsilon)$ for apomixis and homogametic amphimixis, or $X > f(1-u)/(1-m)$ for heterogametic amphimixis, these conditions shrink to an identical simple result. As shown in Table 8, the condition for invasion of allele B_2 is simply superiority of B_1B_2 over B_1B_1. The condition for its fixation is in turn B_2B_2 homozygote superiority, $(1-u)f > (1-vu)e$, in the presence of sexual reproduction. These boundary conditions have precisely the same form as those for selection on an allele in a conventional diallelic single-locus model. This very familiar result states that as long as males can reproduce, selection is expected to increase their fitness. This result also shows that males do not exhibit a conditional response to the presence of parthenogenesis. This prevents return to exclusive sexual reproduction once parthenogenesis has invaded, in that there is no increase in the intensity of selection for male virility upon invasion of parthenogenesis. Below, we will present additional results which indicate that this finding is not exceptional.

TABLE 8

Conditions for boundary stability, two-locus model

Invasion of male virility variant B_2, *all types of parthenogenesis*

$$(1-vu)e > 1$$

Invasion of male avoidance, genital tract incompatibility to sperm, and general egg surface exclusion variants B_2

Apomixis, homogametic amphimixis and automixis

$$4(1-hs)(1-mn)[1-(1-op)bc]d > c\{7-(1-op)-2(1-op)(1-hs)(1-mn)(1-d)g\}$$

Heterogametic amphimixis

$$3(1-hs)(1-mn)[1-(1-op)bc]d > c\{7-(1-op)-2(1-op)(1-hs)(1-mn)(1-d)g\}$$

Invasion of parthenogenetic egg surface exclusion variants B_2

Apomixis, homogametic amphimixis and automixis

$$2(1-hs)(1-mn)[1-(1-op)bc]d > c\{3-(1-hs)(1-mn)(1-d)g\}$$

Heterogametic amphimixis

$$3(1-hs)(1-mn)[1-(1-op)bc]d > c\{7-(1-op)-2(1-op)(1-hs)(1-mn)(1-d)g\}$$

Where $(1-op)$ = proportion of matings avoided or neutralized by B_1B_2 females.
† All conditions require $X > 0$.

(B) EVOLUTION OF MALE AVOIDANCE, FEMALE GENITAL TRACT INCOMPATIBILITY TO SPERM AND GENERAL EGG SURFACE EXCLUSION

We suppose that these mechanisms afford protection to parthenogenetic output at the expense of the sexual output of heterozygous females. In the previous model, we assumed independence of parthenogenetic functions and male functions. We now consider that locus A is responsible for both female reproductive status and pleiotropic effects on male functions, as in the one-locus model. Locus B is responsible for mating behaviour or physiological compatibility of eggs with sperm. B_1B_1

homozygotes are taken to show normal response to males, while for other genotypes a proportion of matings are aborted for behavioural or physiological reasons, *op* in B_1B_2 heterozygous females and *p* in B_2B_2 females. We assume that allelic variation at locus *B* does not cause pleiotropic effects in males. In finite population models with monoparity, pre-mating avoidance and post-mating imcompatibility would differ in that males being rejected are still available to the obligately sexual females, but are "spent" by post-mating incompatibility. In a finite population, this could affect the size of the sperm pool, but the two phenomena are indistinguishable in an infinite population model.

We derived the conditions for the invasion of allele B_2 affecting sexual compatibility under the assumption of non-negligible sexual reproduction. These conditions, given in Table 8, state that allele B_2 is eliminated if A_1A_2 heterozygote females are predominantly sexual. This allele can invade if parthenogenesis is at least somewhat dominant and the total output of diploid eggs from A_1A_2 females exceeds that of the sexual genotypes. In the neighbourhood of the parthenogenetic boundary, mating failure appears as a second order term, indicating that clonal selection for increased viability and fertility is more important than secondary protection of parthenogenetic output from males.

(C) EVOLUTION OF PARTHENOGENETIC EGG SURFACE EXCLUSION AND RESISTANCE TO TRIPLOIDY

In the preceding section, the variants faced depressed fertilization rates, regardless of their reproductive mode. Now we will consider cases in which the depression in fertilization affects only unreduced eggs, either through surface exclusion or resistance to triploidy. As before, the effects of these mechanisms may differ in a finite population model, particularly when one is interested in the prediction of the distribution of ploidies in a parthenogenetic populations, but both mechanisms are indistinguishable in an infinite population model.

In both cases, we supposed that a proportion *op* of unreduced eggs from B_1B_2 mothers and a proportion *p* of unreduced eggs from B_2B_2 mothers would survive successful matings. The conditions for invasion by allele B_2 in predominantly sexual populations are given in Table 8. Variants can invade if parthenogenesis is present in the population. Close to the parthenogenetic boundary, we find conditions identical to those of the preceding analysis. In pure sexual populations, such mutations have no effect on their carriers. The conditions for invasion by parthenogenetic egg surface exclusion and triploidy resistant forms are more easily met than those causing mating failure, and we suppose that they are a major force in the incipient evolution towards complete parthenogenesis in reproductively polymorphic populations.

This analysis of the two-locus model indicates, above all, that once parthenogenesis successfully invades sexual populations, whether or not proceeds immediately to fixation, sex is doomed. This happens because males do not react evolutionarily to the presence of parthenogenesis, while parthenogenetic females are subject to continuous selection pressures to minimize the effect of males.

Furthermore, stable polymorphic populations are highly improbable, given the one-locus results, and highly ephemeral if they arise at all. The sole possible exception is partially recessive automixis, because inbreeding depression alone can then cause the severe viability and fertility selection on homozygote forms required for instability of the parthenogenetic boundary. Otherwise, the analytical results suggest that if parthenogenesis can invade, it will normally eliminate sex.

5. Discussion

Our analysis shows that the classical two-fold advantage of parthenogenesis does not have to be mitigated by severe viability or fertility depression to explain the maintenance of sex. We do not challenge the extensive evidence supporting the view that early stages of parthenogenesis initially suffer low fitness, especially in the case of automixis (Templeton, 1982; Uyenoyama, 1984). On the other hand, there is some evidence supporting the saltatory origin of efficient parthenogenesis (Cuellar, 1977; Maynard Smith, 1978*a*), with the additional feature that these "hopeful monsters" do not have the goldschmidtian problem of finding a mate. On our view, their problem is one of avoiding willing mates. Indeed, even when parthenogens are fully functional, our results show that male fertilization inducing triploidy can readily eliminate the parthenogenetic variants from sexual populations. Our conclusion is that the combination of cytological inefficiency with the egg wastage due to male fertilization is sufficient to explain the rarity of parthenogenesis, in the absence of any additional selective advantage accruing to sexual recombination (cf. Maynard Smith, 1978*a*; Bell, 1982). Therefore, we conclude that there is no outstanding problem left to account for in the evolutionary maintenance of anisogamous sex. This assertion has been frequently made before for theories of the evolution of sex which assume that it conveys one or another type of adaptive benefit (Rose & Redfield, in press). However, our theory is different in that (i) it does not require any selection mechanisms which have not already been directly observed, (ii) the parametric conditions required for our evolutionary mechanism are not restrictive, and (iii) particular instances of the maintenance of sex in the face of parthenogenesis have been independently attributed to the selective factors that we study (Darevsky, 1958; Maslin, 1962; Lowe & Wright, 1966; Lowe *et al*, 1970; White, 1973; Williams, 1975; Parker & Selander, 1976; Cuellar, 1977; Cole, 1979; White & Contreras, 1979; Dawley *et al.*, 1985).

In fact, this conclusion is so obvious that our mathematical results are perhaps of greater value where the evolution of parthenogenesis itself is concerned. One of our most important findings is that once wholly parthenogenetic populations are established they are evolutionarily stable with respect to sexual invasion in the absence of any ecological differentiation between sexual and parthenogenetic forms. This stability is an immediate corollary of the role of males in the genetic stability of sex. The key feature of the results is that the role of males in maintaining sex is frequency-dependent: when sex is more common, there are more males, and so parthenogenetic females face a greater risk of fertilization. Conversely, when sex is less common, there are fewer males, and parthenogenetic females will not be

fertilized as often. In addition, when males are rare, parthenogenetic females will make it more difficult for sexual females to find mates. The sex ratio of smaller populations is subject to marked random fluctuations, and the number of males can therefore fall below the minimum required to maintain sex. Thus, our findings readily explain the frequent appearance of parthenogenesis in isolated populations, compared with pandemic populations (Bell, 1982).

Another significant finding is that once parthenogenesis successfully invades a population, sex is expected to disappear under most circumstances. We discount the maintenance of reproductively polymorphic populations on the basis of two results: their existence requires heterosis with respect to the alleles determining reproductive mode, and subsequent evolution of parthenogenetic resistance to fertilization will act to increase their isolation from males, thereby decreasing the magnitude of the fertilization cost of parthenogenesis. Automixis is an exception to this rule, particularly when males have a negligible role in the stability of sex: homozygote lineages are completely inbred, and heterozygote lineages are impossible to maintain without some form of sexual reproduction. Interestingly, all tychoparthenogenetic species are automictic (Templeton, 1982). In such cases, we propose that tychoparthenogenesis may be a stable state that does not result, by itself, in functional parthenogenesis. Otherwise, we predict that reproductive polymorphism due to segregating allelic variation, as opposed to non-genetic developmental contingencies, will rarely be found.

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APPENDIX

Recursion Systems for Single-locus Model

(i) *Apomictic parthenogenesis.* We define P_{ij}' as the frequency of A_iA_j females in generation $t+1$. The recursion system for apomixis is

$$wP_{11}' = c[Q_{11}+Q_{12}(1-vu)e/2][P_{11}/2+P_{12}(1-hs)(1-mn)(1-d)g/4]$$
$$\times[P_{11}+P_{12}(1-mn)+P_{22}(1-m)]^{-1}$$

$$wQ_{11}' = wP_{11}'$$

$$wP_{12}' = c[P_{11}Q_{12}(1-vu)e/4+P_{11}Q_{22}(1-u)f/2+P_{12}Q_{11}(1-hs)(1-mn)(1-d)g/4$$
$$\times P_{12}Q_{12}(1-hs)(1-mn)(1-d)(1-vu)ge/4+P_{12}Q_{22}(1-hs)(1-mn)(1-d)$$
$$\times(1-u)gf/4]$$
$$\times[P_{11}+P_{12}(1-mn)+P_{22}(1-m)]^{-1}+P_{12}(1-hs)(-mn)(1-bX)d$$

$$wQ_{12}' = wP_{12}'-P_{12}(1-hs)(1-mn)(1-bX)d$$

$$wP_{22}' = c[Q_{12}(1-vu)e/8+Q_{22}(1-u)f/4]P_{12}(1-hs)(1-mn)(1-d)g$$
$$\times[P_{11}+P_{12}(1-mn)+P_{22}(1-m)]^{-1}+P_{22}(1-s)(1-m)(1-aX)$$

$$wQ_{22}' = wP_{22}'-P_{22}(1-s)(1-m)(1-aX)$$

where w is the average fitness of the population

$$w = X[P_{11}+P_{12}(1-hs)(1-mn)(1-d)g]+P_{12}(1-hs)(1-mn)(1-bX)d$$
$$+P_{22}(1-s)(1-m)(1-aX).$$

We know that homogametic amphimixis and automixis differ from apomixis only by the allocation of offspring produced parthenogenetically by heterozygotes into homozygous sexual and homozygous parthenogenetic female genotypes. In heterogametic amphimixis, this allocation also produces male genotypes. Under the assumption of random mating, the proportion of sexually-derived genotypes is the same for all types of parthenogenesis. We therefore derive the corresponding recursion systems from that of apomixis. We define $Y_{ij} = wQ_{ij}'$ in the above recursion system, where Y_{ij} is the frequency of sexually-derived genotypes A_iA_j in any type of parthenogenesis.

(ii) *Homogametic amphimictic parthenogenesis.* The recursion system for homogametic amphimixis is

$$wP_{11}' = Y_{11} + P_{12}(1-hs)(1-mn)(1-bX)d/4$$

$$wQ_{11}' = Y_{11}$$

$$wP_{12}' = Y_{12} + P_{12}(1-hs)(1-mn)(1-bX)d/2$$

$$wQ_{12}' = Y_{12}$$

$$wP_{22}' = Y_{22} + P_{22}(1-s)(1-m)(1-aX) + P_{12}(1-hs)(1-mn)(1-bX)d/4$$

$$wQ_{22}' = Y_{22}.$$

(iii) *Automictic parthenogenesis.* For automixis, the recursion system is

$$wP_{11}' = Y_{11} + P_{12}(1-hs)(1-mn)(1-bX)d/2$$

$$wQ_{11}' = Y_{11}$$

$$wP_{12}' = Y_{12}$$

$$wQ_{12}' = Y_{12}$$

$$wP_{22}' = Y_{22} + P_{22}(1-s)(1-m)(1-aX) + P_{12}(1-hs)(1-mn)(1-bX)d/2$$

$$wQ_{22}' = Y_{22}.$$

where w is as defined for apomixis.

(iv) *Heterogametic amphimictic parthenogenesis.* Retaining Y_{ij} as previously defined, the recursion system for heterogametic amphimixis is

$$wP_{11}' = Y_{11} + P_{12}(1-hs)(1-mn)(1-bX)d/8$$

$$wQ_{11}' = Y_{11} + P_{12}(1-hs)(1-mn)(1-bX)d/16$$

$$wP_{12}' = Y_{12} + P_{12}(1-hs)(1-mn)(1-bX)d/4$$

$$wQ_{12}' = Y_{12} + P_{12}(1-hs)(1-mn)(1-bX)d/8$$

$$wP_{22}' = Y_{22} + P_{22}(1-s)(1-m)(1-aX)/2 + P_{12}(1-hs)(1-mn)(1-bX)d/8$$

$$wQ_{22}' = Y_{22} + P_{22}(1-s)(1-m)(1-aX)/4 + P_{12}(1-hs)(1-mn)(1-bX)d/16$$

where w is now

$$w = X[P_{11} + P_{12}(1-hs)(1-mn)(1-d)g] + 3[P_{12}(1-hs)(1-mn)(1-bX)d + P_{22}(1-s)(1-m)(1-aX)]/4.$$