LETTER TO THE EDITOR

Reproductive Wastage and the Evolution of Genetic Systems

An outstanding problem for evolutionary biology is the maintenance of anisogamous sex in the face of the ostensible twofold reproductive advantage accruing to parthenogenetic females that do not produce male offspring (Williams, 1975; Maynard Smith, 1978; Bell, 1982). Conventional solutions are sought in terms of cryptic advantages to sex involving variable environments, genetic load, and DNA damage (Rose & Redfield, 1988). An alternative point of view is that efficient parthenogenesis is not intrinsically deleterious, just difficult to achieve directly by a single mutation (White, 1978, p. 294; Uyenoyama, 1984). In addition, it has been proposed that new parthenogens face the problem of reproductive wastage, in which sexual males fertilize parthenogenetic eggs and thereby induce lethal triploidy (White & Contreras, 1979; Lynch, 1984; Krieber & Rose, 1986). In effect, parthenogenetic variants could be excluded by reproductive wastage extrinsic to their genetic or evolutionary efficiency when free from fertilization.

An interesting feature of the analysis of reproductive wastage by Krieber & Rose (1986) was that numerical solutions of the model equations rarely gave polymorphism of genetic systems. In most cases, sex was fixed or parthenogenesis was fixed. However, this was not an analytical result. Hickey & Rose (1988) suggested that population dynamic models like those of Rose (1983) or Tremblay & Rose (1985) might capture the essential features of this evolutionary interaction. Given the absence of genetic exchange between parthenogenetic and sexual forms, their evolutionary interaction becomes like that of two competing populations, complicated by the reproductive wastage that occurs when they mate.

Let x represent the density of sexual form and y represent the density of the parthenogenetic derivative. Let $r(x)$ be the density-dependent reproductive output of the sexual form, with $q(y)$ the analogous function for the parthenogenetic form. Let $c_1(y)$ represent the competitive effect of the parthenogen on the sexual form and $c_2(x)$ represent the competitive effect of the sexual form on the parthenogen. Let $f_1(t)$ represent the effect of reproductive wastage on the sexual form and $f_2(x)$ represent the effect of reproductive wastage on the parthenogen. We assume that r and q are strictly decreasing functions, with $r(0) > 0$, $q(0) > 0$, and K_i such that $r(K_1) = q(K_2) = 0$. We also take all c_i and f_i to be strictly increasing, taking on zero values when their arguments are zero. With these definitions, our model becomes

$$\mathrm{d}x/\mathrm{d}t = x[r(x) - c_1(y) - f_1(y)] \tag{1a}$$

$$\mathrm{d}y/\mathrm{d}t = y[q(y) - c_2(x) - f_2(x)]. \tag{1b}$$

The analysis of this model is straightforward, because it has the mathematical form of a two-dimensional competition model with strong density-dependence. As for all two-dimensional continuous-time dynamical systems, there are no strange

attractors, or chaotic aperiodic dynamics. In addition, there are no limit cycles; all asymptotic states are equilibria, with stability determined by patterns of isocline intersections (Hirsch & Smale, 1974, p. 271). The general condition for invasion of parthenogenetic forms in initially sexual populations is

$$q(0) > c_2(K_1) + f_2(K_1) \tag{2}$$

with the interpretation that the intrinsic rate of increase of the parthenogen has to be sufficiently great relative to the competitive and reproductive wastage effects of the sexual form. This much is intuitively obvious.

The interest comes when one considers the problem of the eventual fate of the sexual/parthogenetic mixture, given successful invasion by the parthenogen. We begin with the Lotka–Volterra case:

$$dx/dt = x[a_1 - b_1 x - c_1 y - f_1 y] \tag{3a}$$

$$dy/dt = y[a_2 - b_2 y - c_2 x - f_2 x]. \tag{3b}$$

Here the a_i and b_i parameters have simple interpretations. The a_i parameter is the intrinsic rate of increase for species i growing in an ecological vacuum, normally given as "r" or "m". The b_i parameter reflects density limitation of growth rates. In terms of the classic logistic equation, $b_i = r_i / K_i$, so that, as species density i approaches K_i, $a_i - b_i x_i$ goes to zero. With a model of this kind, invasion of parthenogenetic forms requires

$$a_2/[c_2 + f_2] > K_1 = a_1/b_1 \tag{4a}$$

while fixation of the parthenogenetic form, given invasion, requires

$$a_2/b_2 = K_2 > a_1/[c_1 + f_1]. \tag{4b}$$

Consider the following assumptions. Suppose that sexual males are always in excess, so that sexual females do not lack mates when sexual males mate with parthenogens. Then $f_1 = 0$. In addition, we will assume that competition between sexual and parthenogenetic forms is relatively even, so that we can take all b_i and c_i to have the same value. Then the conditions for invasion and fixation of parthenogenesis become

$$a_2/[b + f_2] > a_1/b \quad \text{and} \quad a_2/b > a_1/b, \tag{5}$$

respectively. These conditions might be clearer if we define a reproductive wastage parameter

$$w = a_2/b - a_2/[b + f_2] > 0 \tag{6a}$$

and note that $a_i/b = K_i$, giving invasion and fixation conditions

$$K_2 - w > K_1 \quad \text{and} \quad K_2 > K_1, \tag{6b}$$

respectively. Since w is positive, whenever the invasion condition is met, fixation is ensured. If we relax the assumption that $f_1 = 0$, then there is a second wastage parameter which appears in the fixation condition as

$$K_2 > K_1 - w_2$$

where w_2 is positive, only enhancing the tendency to fix upon successful invasion. This is the same type of pattern that was found numerically in Krieber & Rose (1986).

With nonlinear forms for the model functions, the results become more complex. If we take $f_1(y) = 0$ and $f_2(x) = fx^2$ or $f_2(x) = fx/[x+1]$ and otherwise retain the Lotka–Volterra assumptions, then with uniform competition within and between sexual and parthenogenetic forms, there are no stable interior equilibria. If the parthenogenetic form can invade, it will fix. In these two cases, the Lotka–Volterra results are qualitatively preserved. There are other cases, however, where they are not. If we take $c_1(y) = c_1 y^2$, $c_2(x) = c_2 x^2$, $f_1(y) = 0$, $f_2(x) = fx^2$ and again preserve the Lotka–Volterra assumptions otherwise, there are asymptotically stable equilibria in the interior, allowing polymorphism of genetic systems. Nonetheless, there are still parameter values for this model which lead to fixation of parthenogenesis upon successful invasion. It is not inevitable that parthenogenesis will fix if it invades, in these nonlinear models, though it remains a major possibility in all types of model and an inevitability for some types.

In conclusion, there does appear to be some tendency for parthenogenesis and sex to be mutually exclusive, as suggested by Krieber & Rose (1986), but it is not a mathematically ineluctable tendency. That this lack of stable polymorphism should arise is a natural result of the fact that the fertility loss imposed on parthenogens by sexuals decreases as the frequency of sexuals decreases. One possibility which should be mentioned is that if the origin of parthenogenesis is associated with some associated genetic effect on ecological role, perhaps by pleiotropy or linkage, then polymorphism of the two reproductive systems could arise from ecological differentiation. The above models allow for this by the possibility of having small values for the c_i functions. Under these conditions, our analyses have found opportunities for coexistence of the two forms. It remains the case for all these population-dynamic models that reproductive wastage can forestall the invasion of parthenogenesis, suggesting that sex is not maintained by adaptive benefits alone, at least in some cases.

Department of Biology,
Dalhousie University,
Halifax, Nova Scotia,
Canada B3H 4J1

DIANE E. EDMONDS
MICHAEL R. ROSE†

† Present address and correspondence: Department of Ecology and Evolutionary Biology, School of Biological Sciences, University of California, Irvine, CA 92717, U.S.A.

REFERENCES

BELL, G. (1982). *The Masterpiece of Nature.* Berkeley: University of California Press.
HICKEY, D. A. & ROSE, M. R. (1988). In: *The Evolution of Sex: An Examination of Current Ideas* (Levin, B. R. & Michod, R. E., eds). Sunderland, Mass.: Sinauer Assoc.
HIRSCH, M. W. & SMALE, S. (1974). *Differential Equations, Dynamical Systems, and Linear Algebra.* New York: Academic Press.
KRIEBER, M. & ROSE, M. R. (1986). *J. theor. Biol.* **122,** 421.
LYNCH, M. (1984). *Quart. Rev. Biol.* **59,** 257.
MAYNARD SMITH, J. (1978). *The Evolution of Sex.* Cambridge: Cambridge University Press.
ROSE, M. R. (1983). *J. theor. Biol.* **101,** 137.
ROSE, M. R. & REDFIELD, R. J. (1988). *Evolutionary Theory,* in press.
TREMBLAY, C. & ROSE, M. R. (1985). *Theor. Pop. Biol.* **28,** 359.
UYENOYAMA, M. K. (1984). *Evolution* **38,** 87.
WHITE, M. J. D. (1978). *Modes of Speciation.* San Francisco: W. H. Freeman.
WHITE, M. J. D. & CONTRERAS, N. (1979). *Evolution* **33,** 85.
WILLIAMS, G. C. (1975). *Sex and Evolution.* Princeton, N.J.: Princeton University Press.