The Contagion Mechanism for the Origin of Sex

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The origin of sex is one of the least understood aspects of the evolution of sex. Recently it has been proposed that sex arose as a result of a contagious genetic element which acted so as to foster its own transmission. Two continuous-time deterministic models of this mechanism are analysed. The first model lacks population regulation. With this assumption, sex is always established in the population, predominating if the sexual form is viable. The second model includes population regulation, giving rise to evolutionary dynamics in which sex need not be established in the population. In addition, with the second model, when the sexual form is viable, it need not fix. Thus, the contagion mechanism can give rise to sex, but need not do so.

1. Introduction

The problem of the origin of sex is one of the major remaining puzzles in evolutionary theory. Reasonable headway has been made with other aspects of the evolution of sex: the origin of anisogamous sex (e.g. Parker, Baker & Smith, 1972; Charlesworth, 1978), the maintenance of anisogamous sex (e.g. Maynard Smith, 1971, 1976; Charlesworth, 1980; Rose, 1982), sex-role allocation (e.g. Charnov, Maynard Smith & Bull, 1976), and so on (Williams, 1975; Maynard Smith, 1978). But no particularly satisfying evolutionary model is available for the origin of sex (Maynard Smith, 1978, p. 9). It has been suggested that sex might be established because of the advantages provided by diploidy with directional dominance, in that recessive deleterious alleles will have little effect in heterozygous condition (Maynard Smith, 1978, pp. 7–9). A major problem for this theory is that there are many sexual species, such as those of the *Chlamydomonus* genus, with only a transitory diploid phase, and which therefore ostensibly do not receive significant fitness benefits from such directional dominance. Moreover, this is thought to be the primitive condition among sexual eukaryotes, so that it is directly relevant to theories of the origin of sex (Maynard Smith, 1978, p. 9).

Hickey (1982) has provided an alternative hypothesis. He suggests that parasitic transposons which caused sex in their hosts would foster their own spread, and thus the spread of sex, even if such parasitism depresses host fitness. Since this sex need be no more complex than would be required to allow the spread of the transposable element, it represents a fairly plausible starting point for the evolution of an initially simple sexual system. Evidently, this theory could also postulate plasmids with such "contagious" sex-inducing effects. Thus, Hickey (1982) alludes to the evidence that bacterial conjugation appears to have evolved from selection for the transfer of the genes of conjugative plasmids, rather than their hosts (Willetts & Shurray, 1980). In sum, Hickey has provided a plausible and novel contagion mechanism for the origin of sex from a primitive condition of asexuality.

This contagion mechanism should act with greatest ease in unicellular organism without somatic tissues. Among such organisms, the transmission of contagious sex-inducing parasites could proceed by mechanisms involving extrusion of cell walls and the formation of cytoplasmic bridges, resembling the transmission mechanism of the *Escherichia coli* F-factor (Goodenough & Levine, 1974, pp. 395–399). Moreover, it is among such unicellular organisms that sex must have arisen most commonly, since it seems to be polyphyletic among the Protozoa and Algae, but not among the Metazoa. Thus explicit mathematical models of the origin of sex by contagion can be usefully cast in terms of the ordinary differential equation models known to work so well with microbial community dynamics (Vandermeer, 1969), in which contagion becomes a kind of predation process.

Models of this kind are presented here, and analysed with a view to assessing the cogency of Hickey's (1982) contagion mechanism. It is found that it can indeed have the effects conjectured, but not necessarily as straightforwardly as might at first be hoped.

2. Model I: Sexual Contagion without Density Limitation

In models of the microbial sex-contagion process, it is reasonable to assume that sex-inducing genetic elements, or "sex-factors", arise very infrequently, with their immediate fate being determined by their population dynamics, without change in their fitness effects. The sole exception would be mutations causing loss of function, and reversion to a sexuality. Thus, let x_1 represent the population density of the asexual microbe and x_2 represent the population density of the sexual microbe. Let w_1 and w_2 give the intrinsic rates of increase of microbes of types 1 and 2, respectively. It will be assumed that types 1 and 2 are evenly mixed throughout the

local medium, with the probability of contact being a homogeneous linear function of the product of their densities, with a constant probability of conversion of type 1 to type 2 upon contact. Finally, it will be assumed that the sex-inducing genetic element loses its function with a probability of u. With these assumptions, the following pair of ordinary differential equations provide a suitable model:

$$\dot{x}_1 = w_1 x_1 + u x_2 - c x_1 x_2 \tag{1}$$

$$\dot{x}_2 = w_2 x_2 - u x_2 + c x_1 x_2 \tag{2}$$

where $\dot{x}_i = dx_i/dt$, c gives the contagion efficiency, and all parameters are nonzero, with u and c strictly positive.

The analysis of the system of equations (1) and (2) proceeds straightforwardly (see Freedman, 1980). The system has isoclines as follows:

$$\dot{x}_1 = 0 \quad \text{iff} \quad x_2 = \frac{w_1 x_1}{c x_1 - u} \tag{3}$$

$$\dot{x}_2 = 0 \quad \text{iff} \quad x_2 = 0 \quad \text{or} \quad x_1 = \frac{u - w_2}{c}. \tag{4}$$

These isoclines give the following system equilibria:

$$\text{E1:} \quad (x_1, x_2) = (0, 0) \tag{5}$$

$$\text{E2:} \quad (x_1, x_2) = \left(\frac{u - w_2}{c}, \frac{w_1(w_2 - u)}{w_2 c} \right). \tag{6}$$

Equilibrium E1 is always admissible, but equilibrium E2 is admissible only if $u > w_2$ and $w_1 w_2 < 0$. The system of equations (1) and (2) has Jacobian matrix

$$\mathbf{J}(x_1, x_2) = \begin{pmatrix} w_1 - c x_2 & u - c x_1 \\ c x_2 & w_2 - u + c x_1 \end{pmatrix}, \tag{7}$$

giving system eigenvalues of w_1 and $w_2 - u$ for E1, from equations (5) and (7), so that E1 is locally unstable unless w_1 is strictly negative. As such parameter values are incompatible with the existence of a population within which sex could originate, it will be assumed henceforward that $w_1 > 0$, so that E1 is never locally stable. When E2 is admissible, it has eigenvalues given by $\lambda \pm$ in

$$2\lambda \pm = w_1 u / w_2 \pm \sqrt{(w_1 u / w_2)^2 + 4 w_1 (w_2 - u)}. \tag{8}$$

Local asymptotic stability occurs if and only if $w_2 < 0$.

Assuming that $w_1 > 0$, the system of equations (1) and (2) may be readily characterized in terms of its global dynamics. If $u \leq w_2$, $\dot{x}_2 \geq 0$, with $\dot{x}_2 = 0$ only if $u = w_2$ and $x_1(0) = 0$. Assuming $u < w_2$,

$$\dot{x}_2 = [w_2 - u]x_2 + cx_1x_2$$

$$\geq r_1x_2,$$

for some $r_1 > 0$. Thus $x_2(t) \geq x_2(0) e^{r_1 t}$, so that, for $x_2(0) > 0$,

$$\lim_{t \to \infty} x_2(t) = \infty$$

Meanwhile, equation (1) gives $\dot{x}_1 < 0$ for $x_1 > u/c$ and $\dot{x}_1 > 0$ for $x_1 < u/c$, when x_2 is arbitrarily large. Thus, we have x_1 converging to a small neighbourhood of u/c. If we define $\rho_2 = x_2/(x_1 + x_2)$, then it is clear that $\rho_2 \to 1$ as $t \to \infty$. If $u = w_2$, then $\dot{x}_2 = cx_1x_2$. If $x_1(0) = 0$, then $x_2(t) = x_2(0)$. Otherwise, note that E2 is equivalent to E1. Thus, the system has no locally stable equilibria. If $x_2(0) = 0$, $x_1(t) \to \infty$ as $t \to \infty$. If both $x_i(0) > 0$, we have $x_2(t) > 0$ for all t. From equation (1), we again find that $\dot{x}_1 > 0$ for $x_1 < u/c$. Thus, if $r_2 = \min(x_1(t = 0), u/c)$, we have $\dot{x}_2 \geq r_2x_2$, so that $x_2(t) \to \infty$ as $t \to \infty$, as before. Again, for arbitrarily large $x_2(t)$, we have x_1 converging to u/c, so that $\rho_2 \to 1$ and $t \to \infty$.

If $u > w_2$, then the global dynamics are most readily understood in terms of the isocline phase-portraits shown in Fig. 1. There are two major cases: $w_2 \geq 0$ and $w_2 < 0$. In the first case, the $\dot{x}_2 = 0$ isocline is entirely to the left of the $\dot{x}_1 = 0$ isocline. Since, with systems of this kind, trajectories
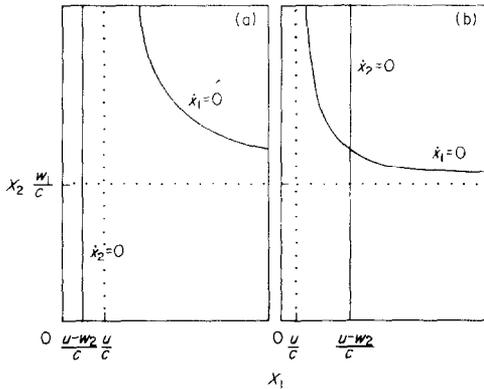

FIG. 1. Phase-plane portraits of the system of equations (1) and (2). The graphed solid lines give the indicated isoclines. The dotted lines give the asymptotes of the $\dot{x}_1 = 0$ curve. (a) For $w_2 \geq 0$, the x_2 isocline is entirely to the left of the x_1 isocline. Sex fixes. (b) For $w_2 < 0$, the x_2 isocline intersects the x_1 isocline, defining a global asymptotically stable equilibrium at the unique intersection point. There is asexual/sexual polymorphism.

beginning in the interior of the first quadrant remain away from the edges for finite times (Albrecht *et al.*, 1974), the flow due to $\dot{x}_1$ necessarily takes the trajectory to the right of the $\dot{x}_2 = 0$ isocline and keeps it there. Thus, $x_2(t) \rightarrow \infty$ as $t \rightarrow \infty$, as before, and the same analysis as that for the $u \le w_2$ case shows that $\rho_2 \rightarrow 1$ as $t \rightarrow \infty$.

In the second case, the system of equations (1) and (2) has the same form as equations (3.9) of Harrison (1979), wherein it is shown that for $x_1 > u/c$, the system asymptotically converges to E2. For $0 < x_1 \le u/c$, $\dot{x}_1 = w_1 x_1 + [u - cx_1] x_2 \ge w_1 x_1 > 0$, so that $x_1(t)$ enters the region $x_1 > u/c$ after a finite period of time, providing $x_1(0) > 0$. Thus the system globally converges to E2 when $w_2 < 0$, and ρ_2 does not approach unity.

The biological interpretation of the dynamics of the system of equations (1) and (2) is that, providing $w_2 > 0$, sex will spread through the entire population. But, if the sexual form of the microbe is not viable on its own, the population will remain polymorphic, with both sexual and asexual forms. Note that in *either* case, sex is established in the microbial population.

3. Model II: Sexual Contagion with Density Limitation

An important feature of the dynamics of Model I was indefinite population growth of the sexual form while the asexual form remained at finite equilibrium levels. These dynamics arose because the model assumed the absence of density-dependent population regulation. Relaxing this assumption will prevent unbounded population growth of either form, and thus might affect the evolutionary outcome. In particular, it might be expected that population regulation could mitigate the spread of sexual contagion.

If it is assumed that population regulation conforms to the logistic pattern and that mutations destroying sexual function are sufficiently infrequent, then the following pair of equations are a reasonable model:

$$\dot{x}_1 = w_1 x_1 - d_1 x_1^2 - c_1 x_1 x_2 \tag{9}$$

$$\dot{x}_2 = w_2 x_2 - d_2 x_2^2 + c_2 x_1 x_2. \tag{10}$$

It is assumed that $d_i > 0$, $i = 1, 2$, $w_1 > 0$, and $c_1 > 0$. When the sexual form is inviable, $w_2 \le 0$. Since forms 1 and 2 are closely related, they will have between-type density-dependent growth limitation effects of about the same magnitude as within-type effects. If the sexual form is sufficiently sensitive to such effects, it is possible to have $c_2 \le 0$. If both w_2 and c_2 are not positive, $\dot{x}_2 < 0$ for all (x_1, x_2) and $x_2(t) \rightarrow 0$ as $t \rightarrow \infty$. If $w_2 > 0$ and $c_2 = 0$, $x_2(t) \rightarrow w_2/d_2 = k_2 > 0$ as $t \rightarrow \infty$, while $x_1(t) \rightarrow (w_1 - c_1 k_2)/d_1 > 0$ as $t \rightarrow \infty$. The two nontrivial cases arise when (i) $w_2 > 0$ with $c_2 < 0$, and (ii) $c_2 > 0$.

In the first case, the model of equations (9) and (10) is isomorphic with the Lotka–Volterra two-species competition model with density-dependent population regulation (e.g. Freedman, 1980, pp. 147–151). The behaviour of this system is well-understood, as summarized in Fig. 2. The sexual form will go to fixation globally only if $w_2/d_2 > w_1/c_1$ and $w_2/-c_2 \geq w_1/d_1$ simultaneously. The biological interpretation of this condition is that the
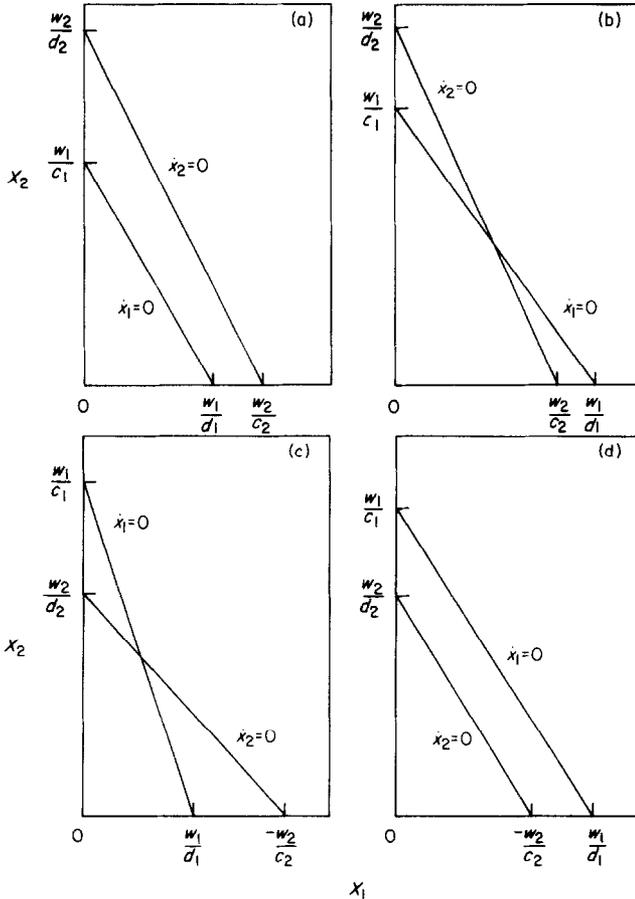

FIG. 2. Phase-plane portraits of the system of equations (9) and (10) system when $w_2 > 0$ and $c_2 < 0$ simultaneously. The graphed solid lines give the indicated isoclines. (a) The system trjaectory proceeds to the global asymptotically stable equilibrium given by $(x_1, x_2) = (0, w_2/d_2)$. Sex fixes. (b) The equilibrium given by $(x_1, x_2) = (w_1/d_1, 0)$ is a local asymptotically stable equilibrium. The asexual population successfully resists sexual invasion. (c) The unique internal equilibrium given the isocline intersection is globally asymptotically stable. Polymorphism. (d) The equilibrium of (b) is globally asymptotically stable. Sex is not established.

sexual form should not be too depressed in fitness, while having high contagiousness and low sensitivity to within-type density effects.

If $w_2/d_2 > w_1/c_1$ but $w_1/d_1 > w_2/-c_2$, then sexuality cannot increase in frequency when initially rare in a population close to ecological equilibrium, even though uniform asexuality is not globally asymptotically stable. In this case, the asexual population cannot be invaded by the sexual form, chiefly because the sexual form is either not sufficiently contagious or is more sensitive to between-type than within-type competition.

If $w_2/d_2 < w_1/c_1$ and $w_2/-c_2 > w_1/d_1$, then the population achieves a global asymptotically stable polymorphism, with both forms present. The sexual factor is sufficiently contagious, but the sexual form is too sensitive to within-type density-dependent population growth inhibition.

In the second case, $c_2 > 0$, the analysis is not as completely standard. The system has the following isoclines:

$$\dot{x}_1 = 0 \quad \text{when} \quad x_1 > 0 \quad \text{if} \quad x_2 = \frac{w_1}{c_1} - \frac{d_1}{c_1} x_1 \tag{11}$$

and

$$\dot{x}_2 = 0 \quad \text{when} \quad x_2 > 0 \quad \text{if} \quad x_2 = \frac{w_2}{d_2} + \frac{c_2}{d_2} x_1. \tag{12}$$

These isoclines give rise to a unique equilibrium

$$(x_1^*, x_2^*) = \left(\frac{d_2 w_1 - c_1 w_2}{c_1 c_2 + d_1 d_2}, \frac{w_2 d_1 + c_2 w_1}{c_1 c_2 + d_1 d_2} \right), \tag{13}$$

though this equilibrium is not always in the first quadrant. The generalized Jacobian matrix for this system is

$$\mathbf{J}(x_1, x_2) = \begin{pmatrix} w_1 - 2d_1 x_1 - c_1 x_2 & -c_1 x_1 \\ c_2 x_2 & w_2 - 2d_2 x_2 + c_2 x_1 \end{pmatrix}. \tag{14}$$

As shown in Fig. 3, there are four distinct and generic isocline configurations. In two of these, there is no admissible interior equilibrium. When $w_2/d_2 > w_1/c_1$, the configuration of Fig. 3(a), the edge equilibrium given by $(x_1, x_2) = (0, w_2/d_2)$ has eigenvalues $-w_2$ and $w_1 - c_1 w_2/d_2$. Thus, this equilibrium is locally asymptotically stable when $w_2/d_2 > w_1/c_1$. The other edge equilibrium, $(x_1, x_2) = (w_1/d_1, 0)$, has eigenvalues $-w_1$ and $w_2 + c_2(w_1/d_1)$, giving instability. In Fig. 3(a) it is clear that x_2 increases for $x_2 \leq w_2/d_2$, while x_1 decreases for $x_2 > w_1/c_1$. Thus the system moves toward the x_2 axis. Near the x_2 axis, x_2 decreases for $x_2 > w_2/d_2$, bringing the system-state close to some local neighbourhood of the $(0, w_2/d_2)$ equilibrium. Thus this equilibrium is globally asymptotically stable. This
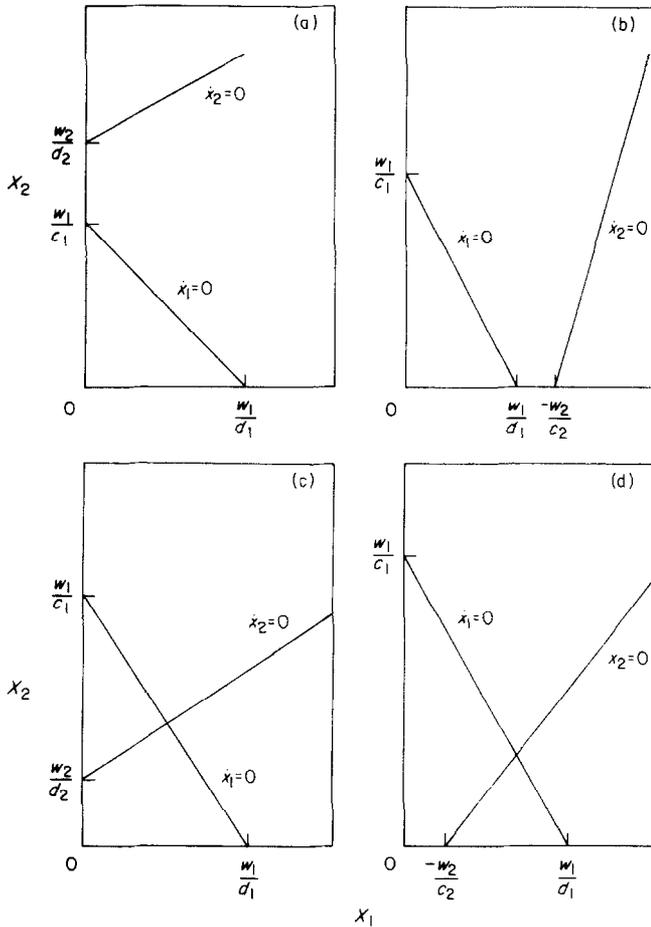
FIG. 3. Phase-plane portraits of the system of equations (10) and (11) when $c_2 > 0$. The graphed solid lines give the indicated isoclines. (a) The equilbrium given by $(x_1, x_2) = (0, w_2/d_2)$ is globally asymptotically stable. Sex fixes. (b) The equilibrium given by $(x_1, x_2) = (w_1/d_1, 0)$ is globally asymptotically stable. Sex is not established. (c), (d) The unique internal equilibrium given by the isocline intersection is globally asymptotically stable. Polymorphism.

behaviour arises because the parameters specify a fairly fit sexual form, which the asexual form is highly liable to be converted to.

If $w_2 < 0$ and $-w_2/c_2 > w_1/d_1$, the only equilibrium other than the origin, which is always unstable, is that given by $(w_1/d_1, 0)$. Unlike the preceding case, now $w_2 + c_2(w_1/d_1)$ is negative, giving local asymptotic stability. By a parallel argument to that just given, it is easy to show that this equilibrium is the unique global attractor for this system configuration. Biologically,

this case involves an inviable sexual form with a sex-factor of insufficient success in converting asexual forms.

In the remaining two configurations, there are no stable edge equilibria. The $(0, w_2/d_2)$ equilibrium, which occurs only when $w_2 > 0$, is unstable since its associated Jacobian matrix has a positive eigenvalue, $w_1 - c_1(w_2/d_2)$, because $w_1/c_1 > w_2/d_2$. The $(w_1/d_1, 0)$ equilibrium, which is always present, is unstable because its Jacobian matrix has the positive eigenvalue $w_2 + c_2(w_1/d_1)$. But, in both these cases, the interior equilibrium is admissible, with associated Jacobian matrix eigenvalues given by $\lambda \pm$ in

$$2\lambda \pm = -(d_1 x_1^* + d_2 x_2^*) \pm [(d_1 x_1^* + d_2 x_2^*)^2 - 4x_1^* x_2^* (c_1 c_2 + d_1 d_2)]^{1/2}. \quad (15)$$

Since $c_1 c_2 + d_1 d_2$ is strictly positive, the real parts of $\lambda \pm$ are strictly negative, guaranteeing local asymptotic stability of the interior equilibrium. Global asymptotic stability follows from the fact that, if χ_i is defined equal to $x_i - x_i^*$, then

$$V(\chi_1, \chi_2) = c_2 \chi_1 - c_2 x_1^* \ln\left(\frac{\chi_1 + x_1^*}{x_1^*}\right) + c_1 \chi_2 - c_1 x_2^* \ln\left(\frac{\chi_2 + x_2^*}{x_2^*}\right) \quad (16)$$

is a Liapanov function for (x_1^*, x_2^*) as given by equation (13) (cf. Freedman, 1980, pp. 37–40), as may be readily checked. The biological significance of this result is that, even with a viable sexual form and sexual contagion, as in the configuration of Figure 3(c), the sexual form need not fix.

Finally, it should be noted that model II accords a greater structural advantage to the sexual form than model I, in that disabling mutations of the contagious sex-factor are not allowed. Yet in spite of this, sex may fix less readily.

4. Conclusion

The foregoing analysis shows that contagious sex-factors could give rise to sex in intially asexual populations, as Hickey (1982) suggested. They could, in some cases, even eliminate asexual forms. But neither is a necessary consequence of the existence of a contagious sex-factor, because with some models, under some parametric regimes, the sexual form fails to become established. In particular, the addition of density-dependent population regulation was shown to mitigate or forestall the spread of a contagious sex factor. The significance of these results is *not* that they provide any clear empirical expectations for patterns of sexuality among living things. The models discussed here are no more than special cases of a broad range of possibly relevant models. Rather, the diversity of evolutionary behaviour produced by these simple models shows that further

theoretical analysis is warranted, because it is not the case that the contagion mechanism has only one possible effect.

In addition to the discovery that a contagious sex-factor may or may not successfully invade an asexual population, it was also found that evolution could produce a population polymorphic for sexuality *versus* asexuality. With this polymorphism maintained by the contagion effect, evolution could then foster the spread of sexuality by means of a group selection mechanism in favour of a sexual subpopulation which underwent more rapid increases in mean fitness than the corresponding asexual subpopulation (Fisher, 1958; Crow & Kimura, 1965; Maynard Smith, 1968, 1978). Thus one possibility is that the contagion mechanism is only a mediate factor in some cases of the establishment of sex.

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