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Why Sex? and Why Only in Pairs?[1]

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Abstract. Understanding the purpose of sex remains one of the most important unresolved problems in evolutionary biology. The difficulty is not that there are too few theories of sex, the difficulty is that there are too many and none stand out. To distinguish between theories we suggest the following question: *Why are there no triparental species in which an offspring is composed of the genetic material of three individuals?* A successful theory should confer an advantage to biparental sex over asexual reproduction without conferring an even greater advantage to triparental sex. We pose our question in the context of two leading theories of sex, the (deterministic) mutational hypothesis that sex reduces the rate at which harmful mutations accumulate, and the red queen hypothesis that sex reduces the impact of parasitic attack by increasing genotypic variability. We show that the mutational hypothesis fails to provide an answer to the question because it implies that triparental sex dominates biparental sex, so the latter should never be observed. In contrast, we show that the red queen hypothesis is able to explain biparental sex without conferring an even greater advantage to triparental sex.

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1. Introduction

The breadth and variety of methods by which different species reproduce through sex is nothing short of remarkable. Nonetheless, sexual reproduction displays a stunning regularity.

> Each sexually produced offspring of any known species is produced from the genetic material of precisely *two* individuals. That is, sex is always *biparental*.

The obvious, but overlooked, question is, *Why?* In particular, why are there no triparental species in which an offspring is composed of the genetic material of three individuals?

Answering this question – and similar questions regarding quadriparental sex, etc. – is bound to shed light on the purpose of sex itself, one of the most important unresolved problems in evolutionary biology (see, e.g., Otto and Lenormand (2002) or Rice (2002) on the importance of this question). Indeed, a complete theory of sex must strike a delicate balance. On the one hand – as is well known – it must explain why genetic mixing is sufficiently beneficial so that biparental sex overcomes the twofold cost of males it suffers because an equally-sized asexual population would grow twice as fast (Maynard Smith 1978). On the other hand – and this point is central here – genetic mixing must not be so beneficial that a further increase in fitness would be obtained from even more of it through triparental sex.

Little or no attention has been paid to the possibility that a theory of biparental sex might inadvertently confer an advantage to triparental sex. Perhaps this is because one is tempted to dismiss triparental sex on the grounds that the associated costs — be they the cost of unproductive males or mating coordination costs — are

prohibitive. But, insofar as such arguments have been provided at all, they are unpersuasive. In particular, they fail to take into account the key point that any argument against the transition from biparental sex to triparental sex may be even more persuasive for ruling out the transition from asexual reproduction to biparental sex. Several such arguments are considered below.

The present paper considers whether either of the two leading theories for the maintenance of biparental sex is consistent with the absence of triparental sex. The first of these theories is the (deterministic) mutational hypothesis due to Kondrashov (1982, 1988). The second is the "red queen" hypothesis, of which several models have been proposed (see, e.g., Jaenike 1978; Hamilton 1980; and Hamilton et. al. 1990). Both the mutational and red queen hypotheses exploit the fact that sex generates genetic mixing, although they are in sharp disagreement about precisely why genetic mixing is advantageous. Roughly, the mutational hypothesis asserts that genetic mixing reduces the rate at which harmful mutations accumulate, while the red queen hypothesis asserts that it reduces the impact of parasitic attack by increasing genotypic variability.

We observe that there is a particular triparental sexual system that involves no additional cost of males relative to biparental sex. Under the mutational hypothesis this triparental system has a fitness advantage over biparental sex for all parameter values considered because it generates more genetic mixing. Moreover, this advantage can be substantial when the mutation rate is high enough so that biparental sex has a fitness advantage over asexual reproduction (i.e., high enough so that biparental sex overcomes its twofold cost of males). That is, if the mutational hypothesis is true, then either asexual reproduction has a fitness advantage over biparental sex (because the mutation rate is low) or triparental sex has a signif-

icant fitness advantage over biparental sex (because the mutation rate is high). The mutational hypothesis is therefore unable to simultaneously explain the presence of biparental sex and the absence of triparental sex.

On the other hand, we present a simplified red queen model that confers an overwhelming advantage to biparental sex over asexual reproduction but confers no advantage at all to triparental sex (or to quadriparental sex, etc.) over biparental sex. The red queen hypothesis therefore is not at odds with the presence of biparental sex and the absence of triparental sex.

2. Triparental sex

Triparental sex will be said to occur when each cell of an offspring is composed of the genetic material of three parents. We will focus upon a particularly significant triparental system in which an offspring receives half of its genetic material from its mother and one-quarter from each of its two fathers. We refer to this reproductive system as $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$-triparental sex, or simply $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex. Before proceeding any further, let us address several possible arguments against any such triparental system.

First, there is the obstacle of developing the requisite genetic machinery for combining the genetic material of more than two parents. Providing a plausible and detailed microbiological mechanism through which triparental sex might operate is well beyond the scope of this paper. Nevertheless, it is noteworthy that, although triparental sex has never been observed in nature, triparental *recombination* is well known to occur in viruses,[5] where offspring DNA are routinely a combination of the DNA of two, three or more parents (e.g., Bresch 1959;, Stent 1963; and Munz et. al. 1983).[6] The presumption that nature could never adapt this viral triparental

[5] According to most biologists, viral recombination is not a form of sexual reproduction. There is even a question as to whether viruses are "alive."

[6] According to Bresch (1959), "In a 'triparental' cross, for instance, the [host] cells will be

recombination technology, or some other existing technology, into an advantageous triparental sexual mechanism becomes less and less plausible as the advantage of triparental sex over biparental sex grows. As we shall show, the mutational hypothesis implies that if biparental sex is to have an advantage over asexual reproduction, then triparental sex must have a significant advantage over both.[7]

Second, one might argue that the costs of coordinating the mating of three individuals over just two outweigh the potential benefits.[8] To be taken seriously, such an argument must carefully consider the additional benefits and coordination costs incurred not only in the transition from biparental to triparental sex, but also in the transition from asexual reproduction to biparental sex. A serious difficulty for any such argument is that while there are clearly significant additional coordination costs involved in the transition from asexual to biparental sex—e.g., a technology for locating mates must be developed and maintained—the ample empirical evidence for sperm competition (e.g., Parker 1970) implies that the additional coordination cost of triparental sex over biparental sex is negligible for a large number of species. Indeed, as the following quote from Birkhead (1998) highlights, the prevalence of sperm competition implies that biparental mating behavior routinely

infected by the [viral] phage types ab^+c^+, a^+bc^+, and a^+b^+c. In this case one finds triparental recombinants abc among the progeny, i.e., particles with a marker from each of the three parental types."

[7] One might conjecture that nature is incapable of developing any form of advantageous triparental sex since any mutation in that direction is bound to create a zygote that is not viable. But such a pessimistic view seems unwarranted, especially in light of the in vitro fertilization technique for humans recently approved for use in the U.K. Under this technique, the future child's mitochondrial DNA comes from a second woman so that the child will be free of an otherwise serious mitochondrial disease. The result is a fitter triparental child with 0.1% of its DNA from the second woman, a permanent change that will be passed down through the generations. While this is not the full triparental sex we consider here, it shows that additional mixing of human genetic material is by no means always fatal.

[8] We have not found any detailed or thorough analyses of the additional coordination costs of triparental over biparental sex, although an informal and very brief discussion on a related topic can be found in Power (1976).

brings together, within a single female, genetic material from multiple males.

> A common assumption about reproduction is that the spermatozoa in the vicinity of ova around the time of fertilization are from a single male. However, for a wide range of organisms, both internal and external fertilizers, this assumption is almost certainly wrong. It is wrong because among internal fertilizers, females typically copulate with more than one male during a single reproductive cycle, and among externally fertilizing animals, often several males simultaneously release spermatozoa near a spawning female. When the ejaculates from two or more males compete to fertilize the ova of a particular female, the process is referred to as sperm competition. Sperm competition is virtually ubiquitous and its biological consequences are considerable.

Sperm competition occurs, for example, in birds (Goetz et. al. 2003; and Parrott 2005), ants and bees (Holldobler and Wilson 1994), shrimp (Bilodeau et. al. 2004), snails (Evanno et. al. 2005), snakes (Garner et. al. 2002), tortoises (Roques et. al. 2004), fruit-flies (Bressac and Hauscheteck-Jungen 1996), and in polyspermic species such as the comb jelly where a female's egg may be penetrated by multiple sperm, one of which is "chosen" to fertilize it (Carre and Sardet 1984). In all these cases, which are by no means exhaustive, triparental sex—e.g., where the sperm of two distinct males fertilize a single egg—would entail negligible additional coordination costs over biparental sex.

Thirdly, there is the "twofold cost of sex," namely, that a sexual population with a one to one ratio of (unproductive) males to females produces half as many offspring as an equally-sized asexual population (Maynard Smith 1978). The simple reason

for this is that every individual in the asexual population can reproduce whereas only half of the individuals in the sexual population – the females – can do so. One might then naturally expect $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex — involving two unproductive males and one female — to display a threefold cost of males relative to asexual reproduction. But, remarkably, $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex results in only a *twofold* cost. Put differently, $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ triparental sex involves *no additional cost of males* relative to biparental sex. We now explain why.

Because the cost of males is determined not by the ratio of males to females in each mating instance but, rather, by the *population* ratio of males to females, determining the population ratio is central. We therefore turn to Fisher's (1930) celebrated equilibrium argument. Applying Fisher's logic to $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex, we note first that the total reproductive value of all of the males in any generation is precisely equal to that of all of the females in that generation. This is because, under $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex, all of the females supply half of the genes of all future generations. But then the remaining half must be supplied by all of the males. Consequently, if the equilibrium sex ratio were not one, it would be evolutionarily advantageous to produce only offspring of the sex that is in short supply, pushing the sex ratio toward one.[9] We conclude that the equilibrium sex ratio must be one, and that each male therefore mates with two females and vice versa. But this means that the cost of males is twofold, precisely as in the case of biparental sex. That is, $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex entails no additional cost of males relative to biparental sex.[10]

[9] We maintain the usual assumption that offspring of either sex are equally costly to raise to maturity.

[10] In contrast, there is a threefold cost of males in a triparental population in which a mother and two fathers all contribute equally to the offspring, i.e., $\frac{1}{3}$-$\frac{1}{3}$-$\frac{1}{3}$ sex. In such a system, because all females supply only one-third of the genes of all future generations, Fisher's argument implies that there must be twice as many males as females, and hence a threefold cost of males. So although in comparison to $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex, the additional genetic mixing from $\frac{1}{3}$-$\frac{1}{3}$-$\frac{1}{3}$ sex yields additional fitness

Since $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex is not observed in nature, it must not have a fitness advantage over biparental sex. We now show that the mutational hypothesis is not consistent with this requirement.

3. The mutational hypothesis

A well known explanation for the maintenance of sex in large populations is Kondrashov's (1982, 1988) mutational hypothesis in which sex is advantageous because it halts the otherwise steady accumulation of harmful mutations. The first theory of this kind was due to Müller (1932, 1964), but relied upon a finite population.

Kondrashov's (1982) model is as follows. There is a population consisting of a continuum of individuals. Each individual consists of a single strand of DNA (i.e., individuals are haploid) that has infinitely many loci.[11,12] Mutations at all loci are equally harmful and an offspring's survival probability is determined entirely by the number of mutations in his genome. Specifically, an offspring with $i < K$ mutations survives with probability $s_i = 1 - (\frac{i}{K})^{\alpha}$. Offspring with K or more mutations are not viable. As individuals develop into adults, they independently receive additional mutations according to a Poisson distribution with mutation rate μ, where the probability that any particular locus receives a mutation is zero. These additional mutations do not affect survival, but may be passed on to one's offspring, affecting its survival.

Kondrashov compares the limiting fitnesses (survival probabilities) of two kinds of populations, one that reproduces asexually and one that reproduces biparentally.

benefits, once its additional 1.5-fold cost of males is taken into account, it has a lower overall fitness than $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex under all parameter values considered here.

[11] A locus is a location on a strand of DNA. Each locus contains a gene. A typical strand of DNA, also called a ploid, consists of many loci.

[12] The assumption that individuals are haploid is for simplicity only. The results are identical when individuals are diploid, i.e., consist of two strands of DNA.

For an asexual population, Kondrashov shows that after many generations the limiting fraction of offspring that survives in each generation is $e^{-\mu}$, regardless of the values of K and α. We refer the reader to Kondrashov (1982) for the details. As for a biparental population, we review Kondrashov's analysis here so that we may adapt it to a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ triparental population.

A biparental population is divided equally into males and females with identical distributions of mutations. Only pairs of individuals of opposite sex can produce an offspring which is equally likely to be male or female, and males are randomly matched to females prior to mating.[13]

The life-cycle is mutations-recombination-selection-mutations. That is, adults accumulate mutations, males and females are randomly matched and sexual reproduction occurs (recombination), fit offspring survive and become adults, adults accumulate mutations, etc. It is assumed that when recombination occurs, there is no linkage between loci. That is, the probability that an offspring receives a mutation from a particular locus of a parent's genome is independent of the locations and number of other mutations on that parent's genome.

Individuals live for a single generation. Let q_i denote the common fraction of males and females in a given generation with i mutations after selection. After mutations arrive according to the Poisson process, the fraction of males and females with i mutations is

$$q_i' = e^{-\mu} \sum_{j=0}^{i} q_j \frac{\mu^{i-j}}{(i-j)!}. \tag{1}$$

[13]Because the two sexes are completely symmetric, Kondrashov did not in fact divide his population into males and females. Instead, he assumed that any two individuals can mate, leading to a simpler model with identical results. But to provide a unified treatment of biparental and triparental sex, we introduce males and females now because they will be needed when we consider $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ triparental sex, where the roles of the two sexes are not symmetric.

Now, because no two matched individuals have more than one mutation in total at each locus, the frequency with which an offspring from parents having n and m mutations has i mutations is $\binom{n+m}{i}(\frac{1}{2})^{n+m-i}(\frac{1}{2})^{i}$, because, at any locus the offspring is equally likely to inherit the content of the mother's or the father's locus, independently of what occurs at any other locus. Consequently, the fraction of offspring having i mutations after recombination is,

$$q_i'' = \sum_{n+m \geq i} q_n' q_m' \binom{n+m}{i} \left(\frac{1}{2}\right)^{n+m},$$

and half of these offspring are male and half are female. Finally, since offspring with $i < K$ mutations survive with probability s_i and offspring with K or more mutations do not survive, the fraction of males and females with $i < K$ mutations after selection is,

$$q_i''' = \frac{s_i q_i''}{s_0 q_0'' + ... + s_{K-1} q_{K-1}''}, \tag{2}$$

where $s_0 q_0'' + ... + s_{K-1} q_{K-1}''$ is the fitness of the population, or equivalently, the fraction of surviving offspring, male or female. The equilibrium distribution of mutations is characterized by the additional condition that $q_i = q_i'''$ for $i = 0, 1, ..., K-1$, from which one can also obtain the population's equilibrium fitness.

We now adapt Kondrashov's biparental analysis to a triparental $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual population, again divided equally into males and females (by Fisher's 1930 equilibrium argument). As in the biparental case, the life cycle is mutations-recombination-selection-mutations, and we again let q_i denote the common fraction of males and females with i mutations after selection. As before, after mutations arrive, the fraction of males and females with i mutations is q_i' given by equation (1).

Consider a triparental match in which the mother has m mutations and the

two fathers have n total mutations. The offspring can have i mutations if for some $m' \leq m$ and some $n' \leq n$, it receives m' from the mother and n' from the fathers, where $m'+n' = i$. Analogous to biparental recombination, at any locus, the offspring inherits the contents of the mother's locus with probability one-half and inherits the content of a father's locus with probability one-quarter, independently of what occurs at any other locus. Therefore, because the three parents have no more than one mutation in total at each locus, the frequency, with which their offspring have i mutations is,

$$r^i_{m,n} = \sum \binom{m}{m'} \binom{n}{n'} \left(\frac{1}{2}\right)^m \left(\frac{1}{4}\right)^{n'} \left(\frac{3}{4}\right)^{n-n'},$$

where the sum is over $m' \leq m$ and $n' \leq n$ such that $m' + n' = i$.[14] Since half the offspring are male and half are female, the fraction of male and female offspring having i mutations after recombination is,

$$q''_i = \sum_{n+m \geq i} q'_m \left(\sum_{j=0}^{n} q'_j q'_{n-j} \right) r^i_{m,n}.$$

Finally, the fraction of males and females having $i < K$ mutations after selection is q'''_i, which as before, is related to q''_i through equation (2).

The equilibrium distribution of mutations is again characterized by the additional condition that $q_i = q'''_i$ for $i = 0, 1, ..., K-1$, from which one can also obtain the population's equilibrium fitness.

Let us now compare the equilibrium fitness of a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual population with

[14] Analogous to biparental recombination, at any locus, the offspring's gene comes from the mother with probability one-half and from each of the two fathers with probability one-quarter, independently of what occurs at any other locus.

that of a biparental population. The values of $\alpha = 1, 2, \infty$ and $K = 5, 20, 60, 80$ considered here are taken from the literature (Kondrashov (1982), Howard (1994)).

μ	$K = 5$	$K = 20$	$K = 60$	$K = 80$	α
1	2.1	1.0	0.4	0.3	∞
	2.3*	1.6*	0.7*	0.5*	2
	2.0*	1.4*	0.6*	0.5*	1
2	4.8	3.0	1.4	1.1	∞
	4.8	4.2	2.3	1.8	2
	4.4*	4.1	2.2	1.7	1
3	7.4	5.6	2.8	2.2	∞
	7.1	7.0	4.3	3.6	2
	6.7	7.1	4.3	3.6	1
4	9.8	8.6	4.5	3.6	∞
	9.3	10.1	6.6	5.6	2
	8.8	10.2	6.7	5.7	1
6	14.2	15.3	8.9	7.3	∞
	13.2	16.7	11.8	10.2	2
	12.6	16.8	12.0	10.4	1
8	17.8	22.7	14.4	11.9	∞
	16.5	23.7	17.7	15.5	2
	15.9	23.7	18.0	15.7	1

Table 1: % Advantage of Triparental Sex

Table 1 shows the advantage of $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex over biparental sex. Each entry in the table is the percentage amount by which the equilibrium fitness of a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual population exceeds that of a biparental population for a particular vector of parameters, (μ, K, α). Because the only cost to sex in Kondrashov's model is the cost of males, there is no cost to $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex over biparental sex. Consequently, each entry is also the percentage amount by which the growth rate of the triparental population exceeds that of the biparental population. An asterisk indicates that biparental sex fails to overcome its twofold cost relative to asexual reproduction in that cell.

Every entry in Table 1 is positive, indicating that a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual population

always grows faster than a biparental population. Moreover, when biparental sex overcomes its twofold cost — indicated by cells without asterisks — the advantage to triparental sex can be substantial. For example, with intermediate selection (i.e., $\alpha = 2$) and a mutation rate of 2, a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ population grows between 1.8% and 4.8% faster than a biparental population, implying a relative doubling time of between 14 and 39 generations. The mutational hypothesis therefore does not provide an explanation for both the presence of biparental sex and the absence of triparental sex.

Also, the higher is the mutation rate, the larger is the advantage to $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex. With intermediate selection, for example, a mutation rate of 3 is already high enough to imply that a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual population grows 3.6% to 7.1% faster than a biparental population, implying a relative doubling time of between 10 and 20 generations. Thus, in contrast to the literature (Kondrashov 1988; Charlesworth 1990; and Howard 1994), not only do low mutation rates – e.g., below 1 or 2 – constitute evidence against the mutational hypothesis, but *high* mutation rates too constitute evidence against it. And indeed, genomic mutation rate estimates of between 3 and 6 have been found, for example, in chimpanzees (Keightley and Eyre-Walker 2000).

To permit a direct comparison with the literature, Table 1 provides relative *equilibrium* fitnesses of triparental and biparental populations. However, to further illustrate the inability of the mutational hypothesis to explain the absence of triparental sex, we also establish that a small fraction of triparental females introduced into an equilibrated biparental population will eventually take over.

An equilibrated biparental population is seeded with a small fraction of females each possessing one copy of a dominant triparental gene for $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual reproduction. Their distribution of mutations is that of the biparental population. Males

can mate with biparental and triparental females. The triparental gene is expressed only in females, although males can pass it on to male and female offspring, the latter then reproducing triparentally through $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex.

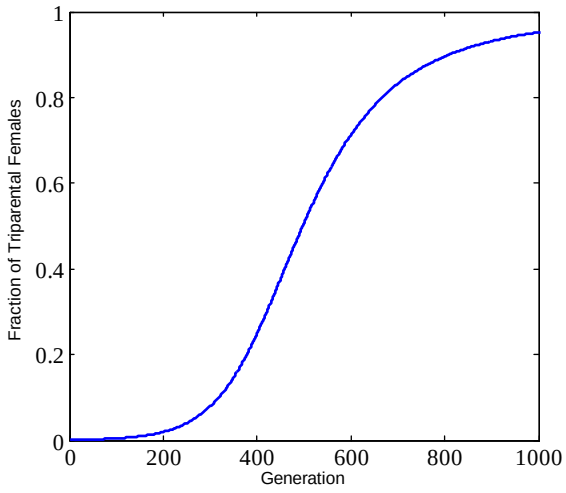

Figure 1: $f = 0.001$, $\mu = 3$, $K = 20$, $\alpha = 2$

In all runs, the fraction of triparental females – i.e., those with at least one copy of the triparental gene – increases with each generation, and *the biparental population is driven to extinction.* A particular example of one of our runs is shown in Figure 1, where f denotes the initial number of females, as a fraction of the population, possessing a single copy of the triparental gene. In contrast, when a triparental sexual population is in equilibrium, biparental sex fails to successfully invade.

4. A red queen model

A second major class of theories for the maintenance of sex is the class of red queen theories. These explain sex as a way for a host organism to maintain parity in

the race against parasites (e.g., Jaenike 1978, Hamilton 1980, and Hamilton et. al. 1990).

Our purpose in this section is to present a red queen model in which biparental sex has an overwhelming advantage over asexual reproduction but in which triparental sex has no advantage over biparental sex. In particular, biparental sex will strictly dominate triparental sex if the latter entails even an arbitrarily small additional cost.

Red queen theories are idealizations of the following scenario in nature. A typical parasite reproduces very frequently within a host, undergoing subtle random mutations with each successive generation. Occasionally, these mutations create a parasitic offspring that is capable of bypassing the host's defense mechanisms. The parasite is then able to rapidly multiply within the host, with the aim of exiting the host and spreading throughout the host population. The rapid multiplication within the host often results in the host's death. The parasite will spread throughout the host population, killing those that it infects. But it can only infect individuals whose defense mechanisms are sufficiently similar to that which it "evolved" to defeat. In particular, if all members of the host population have identical DNA sequences (i.e., identical "genotypes"), as can be the case for an asexual species, the entire population may be killed off since all its members rely on the same susceptible defense mechanism. In contrast, if there is sufficient genetic variation within the host population, as is the case for a sexual species, then only a fraction of individuals may be susceptible to the parasite.

In a nutshell then, the essence of this class of red queen theories is this. The absence of genetic variation can render an asexual species extremely susceptible to attack from parasites, whereas the genetic variation created by a sexually reproduc-

ing species provides protection, making it far less susceptible. It is less susceptible because the distribution of genotypes created by sexual reproduction is sufficiently spread out that a parasitic attack on any one genotype, or on any small range of genotypes, affects only a small fraction of the population. Moreover, unlike in an asexual population, genotypes that are killed off by the parasite can re-emerge as offspring in the next generation of a sexual population via genetic recombination thereby maintaining the overall genetic diversity of the population. The broad conclusion from this class of models is that when a parasite is sufficiently virulent and its attacks are not too infrequent, a sexually producing species can be more successful than an asexual species, even after accounting for the twofold cost of males.

In the remainder of this section we present a highly simplified red queen model that captures the features described above and that gives an overwhelming advantage to biparental sex over asexual reproduction. But, unlike the mutational hypothesis, it gives no further advantage to triparental sex. The important general insight upon which this conclusion is based is that the time-dynamics of population genetics implies that, *whether a species is biparental or triparental (or beyond), the limit distribution of its genotypes is the same* (Perry, Reny, and Robson 2007). As a result, multi-parental sex with three or more parents will not yield any fitness advantage over biparental sex.

Consider an infinite population of haploid individuals whose genomes have four loci, A, B, C, D. Each locus can be occupied by one of two alleles, a or a' in locus A; b or b' in locus B; c or c' in locus C; and d or d' in locus D.[15] Thus, (a, b', c', d) and (a', b', c, d) are two of the sixteen possible genotypes that might comprise an

[15] Recall that each locus on a strand of DNA marks the location of a particular gene. An allele is one of several variations of a gene.

individual in this population.[16]

At each date (generation) $t = 1, 2, ...$ all individuals in the population always reproduce either asexually, biparentally, or triparentally. We will consider each possibility in turn. But regardless of the sexual system that is in place, once every N generations there is a probability $\varepsilon > 0$ that a parasitic attack will occur, killing all individuals of a randomly chosen genotype in the current population.[17] For simplicity, we will suppose that each genotype in the current population is equally likely to be killed conditional on the occurrence of an attack.[18] Individuals live for a single generation.

Let us first consider the fate of an asexual population. Because there are finitely many (indeed, 16) possible genotypes, each one will, with probability one, be the target of a parasitic attack at some date. Moreover, once all individuals of a particular genotype are killed, that genotype will be extinct forever since, under asexual reproduction, the offspring of the remaining distinct genotypes are identical to their parents. Consequently, an asexual species will become extinct with probability one, regardless of the initial distribution of genotypes in the population.

Consider next the fate of a biparental sexual population in which males and females mate randomly and both parents contribute half of their genetic material to the offspring. Specifically, suppose that in any mating instance the alleles in two of the offspring's loci come from its mother and the other two alleles come from its father, with all six possibilities being equally likely, and that the offspring is equally

[16] The extension to any number of alleles and loci is straightforward. One can also allow individuals to be diploid, or triploid, etc., rather than haploid without changing the results.

[17] Our results would be unchanged if most of the time only a fraction of individuals with the chosen genotype were killed, so long as there is at least a small positive probability that all of them are killed.

[18] Our conclusion would not change, for example, if the conditional probabilities were instead proportional to a genotype's representation in the population.

likely to be male or female.[19]

The dynamics of this sexual population are more interesting. Let us suppose, for a moment, that there is no possibility of a parasitic attack, i.e., that $N = \infty$. Then the population dynamics are deterministic because the population is infinite. Indeed, if $q^t_{(i,k,j,l)}$ is the date-t fraction of individuals in the population with genotype $(i,j,k,l) \in \{a,a'\} \times \{b,b'\} \times \{c,c'\} \times \{d,d'\}$, then we can compute the date-$(t+1)$ fraction, $q^{t+1}_{(i,j,k,l)}$, of such individuals as follows. Since the probability is one-sixth that in any particular match the female will contribute her first pair of alleles to an offspring and the male will contribute his second pair, the fraction of offspring who receive (i,j) as their first pair of alleles from their mother and (k,l) as their second pair from their father is one-sixth the fraction of matches of females whose first pair of alleles is (i,j) with males whose second pair is (k,l), that is $\sum_{i',j'k',l'} q^t_{(i,j,k',l')} q^t_{(i',j',k,l)}/6$. Repeating this for all the possible combinations in which the two parents can contribute alleles i,j,k and l, we obtain $q^{t+1}_{(i,k,j,l)} = (\sum_{i',j'k',l'} q^t_{(i,j,k',l')} q^t_{(i',j',k,l)} + \sum_{i',j'k',l'} q^t_{(i,j',k,l')} q^t_{(i',j,k',l)} + \sum_{i',j'k',l'} q^t_{(i,j',k',l)} q^t_{(i',j,k,l')})/3$. Thus, starting from any initial distribution of genotypes one can straightforwardly trace out the dynamics of the population's genotype distribution.

It is well-known that the distribution of genotypes in the above dynamical system converges to so-called *linkage equilibrium* regardless of the initial distribution (see, e.g. Christiansen 1999). In linkage equilibrium, the fraction of individuals of any particular genotype is the product of the population frequencies with which each allele occurs.[20] So, if we let p_i denote the fraction of the population's alleles that are equal to $i \in \{a,a'\}$, and let p_j denote the fraction of the population's alleles that are

[19] Equiprobable recombination events are not necessary. It would suffice to assume merely that the probability is less than one that the offspring receives any two alleles from the same parent.

[20] Without loss of generality, it is assumed that, as here, each allele is specific to a single locus.

equal to $j \in \{b, b'\}$, and similarly for p_k and p_l for alleles at loci C and D, then the limiting population frequency of genotype (i, j, k, l) is $p_i p_j p_k p_l$. It is easy to verify that this distribution is indeed an equilibrium of the above dynamical system, and it is unique by the global convergence result.

Importantly, the rate of convergence to linkage equilibrium is very fast, exponential in fact. Consequently, returning now to the case in which $N < \infty$, if N is not too small, the distribution of genotypes will be very close to linkage equilibrium prior to the first attack. We can now describe the dynamics of the biparental population.

For N not too small, the distribution of genotypes will be approximately in linkage equilibrium just prior to an attack, i.e., genotype (i, j, k, l) will occur with frequency close to $p_i p_j p_k p_l$. When an attack occurs, all individuals of one genotype, say (a, b, c, d), will be eliminated. But because the population was close to linkage equilibrium, all alleles remain present, e.g., allele a occurs in the still-present genotype (a, b', c, d) which made up a positive fraction, approximately $p_a p_{b'} p_c p_d$, of the pre-attack population. The distribution of alleles, however, is no longer the same, e.g., the new relative frequency of allele a to allele a' has fallen to approximately $(1 - p_b p_c p_d) p_a / p_{a'}$. Consequently, during the next N generations the population will converge approximately to its new linkage equilibrium before the next attack.

The overall biparental dynamics is therefore as follows. Beginning approximately from linkage equilibrium, all individuals of a random genotype are killed by a parasitic attack. All alleles remain present, however. The population, with its new distribution of alleles, converges after N generations to its new approximate linkage equilibrium. The next attack occurs, killing all individuals of a random genotype, and so on. Thus, a biparental population survives forever.[21]

[21] It is possible that the population fraction of some particular allele tends to zero along the

Clearly then, biparental sex has an overwhelming advantage over asexual reproduction. But what about triparental sex? The key observation, and this observation holds very generally, is that the dynamics are unaffected by whether sexual reproduction is biparental or $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual. This is because:

> For any given distribution of alleles, the distribution of genotypes in a $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sexual population converges, at an exponential rate, to the same linkage equilibrium distribution as in a biparental population.[22] $(*)$

To get a sense of this convergence result, let us suppose that $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex works as follows. There are equal populations of males and females and each female is randomly matched with two males (each male mates twice). In each mating instance there are two males and one female, and the alleles in two of the offspring's loci come from its mother and the other two alleles come, one each, from the two fathers, with all twelve possibilities being equally likely.

While analysis of the triparental dynamics would take us too far afield (see Perry, Reny, and Robson 2007), let us show that the biparental population's linkage equilibrium distribution is also a linkage equilibrium distribution of the triparental population. Suppose then that the triparental population begins with the biparental population's linkage equilibrium distribution of genotypes. It suffices to show, by symmetry, that the fraction of triparental offspring with genotype (a, b, c, d) is $p_a p_b p_c p_d$. One way that this offspring can be produced is if the mother contributes

path. But the fact remains that the species survives forever.

[22] See Perry, Reny, and Robson (2007) who show that this convergence result holds for any number of alleles, any number of loci, any number of ploids, and any ploid-symmetric distribution over recombination events such that the probability that an offspring receives any two alleles from the same parent is less than one (i.e., there is imperfect linkage between loci).

ab and the first father contributes c and the second father contributes d. The fraction of mothers whose AB loci contain ab is $p_a p_b$, the fraction of fathers whose C locus contains c is p_c, and the fraction of fathers whose D locus contains d is p_d. Hence, the fraction of triparental matches of this kind is $p_a p_b p_c p_d$. There is a one-sixth probability that the mother in this triparental match contributes ab, and a one-half probability that the first father contributes c and the second contributes d. Hence, this one way of producing the offspring (a, b, c, d) has probability $p_a p_b p_c p_d/12$. Since there are twelve equiprobable ways of producing this particular offspring, the resulting fraction of offspring with this genotype is $p_a p_b p_c p_d$, exactly as in the previous generation.

Hence, because the red queen dynamics depend only on the derived sequence of linkage equilibria, and because by $(*)$ the linkage equilibria are the same whether sex is biparental or $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ triparental, the population growth rate will be the same with either sexual system. So, biparental sex can dominate asexual reproduction, but $\frac{1}{4}$-$\frac{1}{4}$-$\frac{1}{2}$ sex can never dominate biparental sex. Consequently, in contrast to the mutational model, biparental sex dominates triparental sex here if the latter involves even an arbitrarily small extra cost.

Finally, let us address two further issues. First, one might wonder what would happen if the number of generations between attacks were random. As before, an asexual population goes extinct with probability one. But the biparental and triparental populations can never go extinct so long as their populations have at least four distinct genotypes, because if the number of genotypes is ever reduced to three, sexual recombination ensures that the next generation consists of at least four. Furthermore, we expect that there remains no advantage of triparental sex over biparental sex given their shared tendencies toward linkage equilibrium.

Second, one might wonder whether an asexual species can successfully invade a biparental population. To ensure that it cannot, one may need to allow the interactions between the host and parasite to be more frequent. Then, the biparental (or triparental) population need not arrive approximately at linkage equilibrium between successive parasitic attacks. Nevertheless, we would not expect triparental sex to have any advantage over biparental sex, but further study here would be welcome.

5. Discussion

There are rich returns to addressing the question: "Why is sex never triparental?"

Under the mutational hypothesis, triparental sex always dominates biparental sex and high genomic mutation rates only serve to increase this advantage. With all three options available, either asexuality would be best or triparental sex would be best. Accordingly, biparental sex should not be observed.

In contrast, there is a ray of hope with the red queen hypothesis. Using a deliberately simplified red queen model, we have shown that biparental sex can have even an overwhelming advantage over asexuality, yet there is no further gain from more than two parents.

These results demonstrate that those who ask "why sex?" should also ask "...and why only in pairs?" Answering the second question can distinguish between otherwise equally plausible answers to the first.

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