f19 The perpetuation of sex < repair >

> It's not because asexual species don't appear. It's because they don't last.
> —Matthew Meselson.[1]

Outcrossing (cross-breeding of pairs of animals, or plants, of different strains but usually of the same breed) that sex allows appears to be a favored evolutionary strategy. Outcrossing, which requires finding a genetically distant mate, requires an expenditure of energy that asexual reproduction avoids. The problem therefore is to explain the perpetuation of sex in a species. What profit can it provide?

Patrick J. Keeling summarizes current wisdom when he writes: "No satisfactory explanation for the origin and evolution of sex in eukaryotes has been proposed that is based solely on the selective advantage (to organisms) derived from genetic shuffling, a primary consequence of meiosis." Explanations favor two themes: sex speeds the elimination of bad mutations, and sex speeds the spreading beneficial mutations. The former reason proffered by Alexey S. Kondrashov[2] and traceable back to A. Weismann,[3] is proven true so far only for yeast (for which sex is merely meiosis followed by shuffling and without sexual selection).[4] The latter is "The Red Queen Principle" of Leigh van Valen.[5] Proponents of this hold the main benefit of sex to be in shuffling the genome quickly, making it difficult for parasites in their catchup pursuit to immediately lock onto weaknesses. As the Red Queen tells Alice, in Charles Lutwidge Dodgson's *Through the looking-glass, and what Alice found there* by *Lewis Carroll*, "in this place it takes all the running you can do, to keep in the same place."[6]

In each cell of haploid organisms, there is only one copy of each gene. To repair errors that intracellular enzymes detect on a particular gene, haploid cells are needful of outcrossing. Thereby, the genes from two cells become physically available to each other at the same time, and with the chance that the errors on both are in different places. Then each cell can serve as a "floppy disk" chromosome to chromosome backup for the genes of the other. In this case of fusion-mediated outcrossing, after sex that creates a transient diploid stage in which repairs are made, the two cells simply split into daughter cells again.

In each cell of diploid organisms, there are two copies of each gene (except in the mature germ-line cells). Spare genes for genetic repair are readily available within the cell itself and recombination from its own material can replace outcrossing as a source of spare gene parts. Yet sex for advanced multicellular organisms is always found to involve recombination *and* outcrossing. Why?

Addressing physician's concerns, and not the longer view that we are the product of countless mutations, Richard E. Michod explains that outcrossing inhibits the expression of mutations. [7]

> Every diploid cell carries two copies of every gene, one from the mother and one from the father of the organism. But the protein-making machinery of the cell often requires just one good copy of a gene. If one copy of a gene has mutated, its nonmutated counterpart will then be dominant and the mutated gene recessive. The effect of the mutation will not be felt in the cell. Even though mutations are recessive, they can accumulate in germ cells and, therefore, in a population. In fact, the germ cells of all organisms carry many recessive mutations. What saves them from the potentially harmful consequences is that individuals who are not close relatives generally have mutations in different genes.

The level at which the accumulation of mutations equals their removal by natural selection will, at equilibrium, be a function of sex and population size. Consider:

> Harmful mutations decrease fitness.
>
> As these are acquired their effect will be additive.
>
> However, the effects of mutations are not necessarily independent of one another.

In that case, fitness can decreases faster (or slower) than simple addition would predicted; a phenomenon called *synergistic* (or *antagonistic*) *epistasis*.

Sexual recombination is a mechanism that can evolve to lessen epistasis.

The mutational deterministic hypothesis is that sex has evolved in response to synergistic epistasis (and would be selected against in the case of antagonistic epistasis).The test will be to find examples. The shyness of these in spite of the clear favoring of sex by nature might indicate that no sweeping generalizations are justifiable and examples will be for specific cases:

One neutral is Santiago F. Elena and Richard E. Lenski's 1997 study of pairs of mutations in *Esherichia coli* that exhibit epistasis in both directions and, for the population, epistasis is zero. Conclusion: sexual interactions that do occur between *E. coli* bacteria are not explainable in terms of the mutational deterministic hypothesis. [8]

Two favorable are:

The value of sex in the competition between host a parasite is clear for natural populations of a New Zealand freshwater snail *Potamopyrgus antipodarum*. Individuals of these snails can be, and be reproductively, sexual or asexual. According to Curtis M. Lively, the *P. antipodarum* frequency of sex is highest in lakes and habitats within lakes where parasites are most prevalent.

The sex card can also be played by a parasite. According to Andrew. F. Read, parasitic nematode larvae from rat hosts that have acquired immune protection develop into sexual adults more frequently than do those from unprotected hosts.

Mark Ridley

His textbook *Evolution* illuminates how we have arrived at the diversity of life that we currently observe.[9]

In humans, a twenty-year-old man's genome is copied about 40 times by puberty and is copied again every sixteen days thereafter. By her fetus late-stage, a human female has, with about 33 cell divisions behind them, her lifetime supply of eggs. **Mark Ridley** reminds us that "When a thirty-year-old man breeds with a thirty-year-old woman, his DNA has been copied 430 times against her 33." Bereft of the cytoplasm that houses protective enzymes, such as catalase or superoxide dismutase in somatic (non-germ) cells, sperm are on their own. Lacking repair and self-destruct mechanisms, so vulnerable to DNA damage by a variety of environmental factors, sperm are about a week in the epididymis being remodeled in preparation for ejaculation and in the female reproductive tract up to six days swimming around searching for an egg. "With about thirteen times as many errata in his DNA, about 185 of the 200 copying mistakes in each human conception may come from the sperm." Of these, most are benign but "on average, 2 to 20 damaging errors are heritable by offspring." Also, "about 50 percent of [human] conceptions have a botched number of chromosomes." [10]

Right now (but for how long given the pace of research worries M. A. Surian in 2002) a barrier to virgin birth (embryos with two male or two female genomes) in mammals are epigenetic contributions that must be present from both parents (or the embryo develops abnormally and is aborted).[11] These epigenetic contributions before zygote formation are genes in the sperm imprinted by the male and genes in the oocyte imprinted by the female that in the embryo are the ones, otherwise indistinguishable in a pair, that are, in fact, switched on. Nearly 50 mammalian imprinted genes have been identified so far and do battle to determine a baby's size—the mother's for small and the father's for big.

On the bright side, Ridley writes: "Sexual reproduction, which operates according to Mendel's principles of inheritance, has the advantage of redistributing the parents' mutations among the offspring. In effect, a toss of the coin determines whether any particular gene will be 'allowed' into

each embryo. Meiosis is the fateful cell division in which each gene, whether perfect or mutated, has only a 50-50 chance of making its way into a gamete—a particular sperm or egg. On average, if a male or female with one harmful DNA mutation produces eight gametes, four will have the flaw and four will be free of error. When the sperm and eggs of two parents—both of whom have one harmful mutation—are combined to form eight new organisms, four offspring on average will have one harmful mutation and two will have two, but the remaining two will have no mutations at all. The life-form carries on."

In an essay, *Sex, Errors, and the Genome*, 2001, Ridley suggests that for species, which normally reproduce sexually, cloning is not an option as the proportion of bad mutations in succeeding clone generations will soon reduce their populations to extinction.[12]

But for a clone originated (***Figure f19.1***) and (also) reproducing organism, cloning can be successful for the parent *is* "perfect" and reproduction has the safeguard that many of the offspring will have genetic variation. So a change in the environment, not so drastic as to eliminate all, is countered by some "imperfect" clones being then the chosen "perfect" ones that continue. This, for medicine and agriculture, is a well known curse!

What Ridley does not suggest is that sexual reproduction, and a penchant for outbreeding, guarantees that in a large population a large number of recessive genes will be expressed and those that test well for quality will accumulate extensively sub rosa in a species' population. When a changed environment causes a numbers pinch, inbreeding, by circumstance, exposes a plethora of recessive genes (mostly not those that result in "bad teeth, cross eyes, and an unnatural interest in banjo playing," as Nicholas P. Money facetiously delivers it)[13] that can turn the environmental clampdown into a bottleneck (a time of rapid speciation) for the species. Forced to unveil its store of recessive genes, the sexually reproducing species may emerge radiant.

To be a successful reproductive option, a caveat is, cloning must be profligate. However, sexual reproduction makes profligate reproduction not an option for the female parent of higher life especially when nurturing enters the picture. So sex is not better than cloning which easily wins the numbers game. Yet higher life exists and for it are the complications of sex. For evolutionists, given the absence of purpose to evolution, this begs the question: Why was there a need for sex in the first place? ❑

Figure f19.1

Dolly and Ian Wilmut

Even in death, Dolly is the pretty one. Cloned by Wilmut from an adult cell, Dolly is now on display at the Royal Museum in Edinburgh. Her skin is exhibited pickled, tanned, washed and stretched over a fibreglass frame. Pink paint touches up her nose and her yellow eyes are glass replicas. Plasticine replaces her soft tissue and muscles. Andrew Kitchener, curator of birds and mammals at the National Museums of Scotland, confides: "Dolly was extremely bouffant-looking after the process, so we had to matt her fleece up a bit." [14]