

## FATAL SEXUALLY TRANSMITTED DISEASES (FSTDs), SUCH AS AIDS, SELECT FOR THE EVOLUTION OF MONOGAMY AND PROVIDE A MODEL FOR BACKGROUND EXTINCTION

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### ABSTRACT

Monogamous behavior may have a genetic component, in which case it can be subject to natural selection. The mechanism of selection may be the reduced fertility that correlates with STDs. AIDS is a fatal STD (FSTD) capable (without intervention) of severely reducing the human population. We show how, for any mating species, multiple FSTDs (statistical "runs") occurring within the characteristic time for population recovery, drive that species exponentially towards extinction. Runs of FSTDs occur on geological time scales, providing a general, quantitative model for background extinction rates, and a simpler alternative to the Red Queen hypothesis. It has been difficult to get people to change their risk behavior even when they have full knowledge of how HIV is spread. If we come to understand monogamy (and perhaps polygamy) as heritable traits, then new approaches for slowing the HIV epidemic become apparent.

*Keywords:* Sexually transmitted diseases, AIDS, HIV, monogamy, polygamy, genetics and evolution of mating behavior, background extinction, Red Queen hypothesis, punctuated equilibrium, genetic drift, population bottleneck, mitochondrial Eve.

"...even a slight degree of infertility, combined with those other causes which tend to check the increase of every population, would sooner or later lead to extinction. The diminution of fertility may be explained in some cases by the profligacy of the women.... A much more probable view is suggested by the analogy of the lower animals. The reproductive system can be shown to be susceptible to an extraordinary degree (though why we know not) to changed conditions of life; and this susceptibility leads both to beneficial and to evil results." Charles Darwin [32].

"In native theory, barrenness is the punishment of promiscuity; and vice versa, only persistent monogamy is rewarded by conception." Margaret Mead [103].

## 1. Introduction

Sexual contact with multiple partners is one of the leading correlates of multifactorial infertility in humans [6,7,24–26,50,68,136,150,154], probably due to sexually transmitted diseases (STDs) ([19,20,27,33,77,82,91,109,129,151]; however, cf. [159]). Since STDs can be transmitted between the internal reproductive systems of both female and male [14,99,146], in monogamous couples, the microbial flora of their reproductive tracts may establish an equilibrium and compatibility with each other which, moreover, may be stabilized by the immune system (cf. [157]). This is further suggested by the common occurrence of genital infections in initial sexual relationships, compared to a lower frequency in stable relationships of some duration, at least amongst couples who act monogamously [13]. Multiple sexual contacts promote the introduction of bacterial and viral strains alien to the reproductive tract of the individual, giving rise to repeated infections by bacteria and viruses that, under other circumstances, might be tolerated (cf. [130,140]). Such infections, whether latent [23] or disclosed, often lead to involuntary, indeed permanent infertility.

## 2. Monogamy and STDs

These observations lead us to propose the hypothesis that:

*Monogamy may be an evolutionary strategy to minimize the risk of reproductive tract infection and consequent infertility.*

Polygamy itself is a strategy for increased individual reproductive success [84], and, furthermore, in humans, has proven to correlate with an overall higher fertility rate [22]. Thus, a balance may be struck between monogamous and polygamous behavior (cf. [2,60,74]). Where this balance lies varies from species to species, probably depending on the sensitivity of the reproductive tract to infection, longevity of sexual partners, length of the breeding season and the number of times per season that breeding can occur. In *Homo sapiens*, given the possibility of continuous breeding from puberty to menopause, the implications of polygamy are great, in terms of involuntary infertility and reduced reproductive success. The sparse evidence available has been summarized [136]:

"Infertility often is relatively common in areas where marriages tend to be unstable and where many people have multiple sexual partners [126]. Marital instability, premarital intercourse, and brief consensual unions — all of which may increase the number of sexual partners — can lead to infertility by facilitating the spread of STDs [33,127,131]. At the same time, marital instability may be the result of infertility [114]. In some societies, women are divorced because of childlessness, or women leave infertile marriages hoping to conceive with a new partner [33,113,128]...

"Polygyny (more than one wife) may increase the likelihood of infertility ([66,114,126,132] [cf. [134]]), probably because STDs can be transmitted quickly among all wives in a polygynous marriage [however, cf. [75]]. At the same time, where polygyny exists, lack of offspring with one wife may encourage a man to take other wives [114]. A 1977 Kenyan survey found that, among women aged 45 to 49, 6 percent of cowives were childless, while only 2 percent of sole wives were childless [65]. (Women in

polygynous marriages may have coitus less often, which also could help to explain such a difference)....

"In the US... survey data suggest that infertility among young married women increased slightly between 1965 and 1976 [108]. Infertility, defined as no pregnancy after one or more years of intercourse without contraception, increased from 4.5 to 7.4 percent among those age 15 to 29, a statistically significant change [108]. Rates of STDs and pelvic inflammatory disease (PID) increased markedly during this period.... the results agree with clinicians' observations of increasing infertility problems among young US women [39,106]. These data suggest an impact of increasing STDs on infertility even where treatment is available."

High population densities are often correlated with reduced fertility [83]. This could derive from a shift from monogamous to polygamous behavior at higher population densities (cf. [53,55]). The result may be an increase in reproductive tract infections and thus reduced fertility (cf. [142]). Infections of the reproductive tract may thus be an alternative explanation to stress, which is commonly supposed to regulate fertility in dense populations ([28]; cf. [16,54]). Of course, stress and susceptibility to disease go hand in hand, so both may occur.

Even in species that do not have internal fertilization, monogamy occurs [118]. This could possibly be an evolutionary response to the potential vertical transfer of pathogens from one generation to the next via the gametes [110]. Some species pair for life [1], but have a short mating season. Such once-a-year mating may not suffice to produce an equilibrium of bacterial flora in the couple, but it would serve to minimize the acquisition of STDs. Different species may avoid reproductive tract infection by means other than monogamy (for instance, parthenogenesis: [15]), or by degrees of monogamous behavior [155]. For instance, a short breeding season may in itself reduce chances for polygamous behavior [158]. Monogamy may be especially important in reducing STDs in animals that breed in dense colonies. Even though some adultery occurs (reviewed in [36]), it tends to be between animals in neighboring nests, which should limit the spread of any STDs that might be present.

We do not wish to suggest that infections of the reproductive tract are the sole explanation of monogamous behavior. A number of other hypotheses have been proposed [9,11,29,35,81,101,117,133,155,158] that are not mutually exclusive, but few have been sufficiently studied (cf. [120]). Our hypothesis that STDs have played an important selective role in the evolution of animal mating systems, including that of humans, should be considered as an alternative or synergistic hypothesis. While potentially and actually pathogenic flora and fauna have been investigated in wild populations [18,51,63,124], there seems to be almost no literature on their STDs, either because they have not been looked for (cf. [138]), or because the selective pressure against behavior allowing their spread is so severe that they are not found. We are in the unusual situation that humans must, for now, provide a model for this question in other animals.

