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Pair-Bonding and the Evolutionary Trajectory of *Homo*: Disease Avoidance as an Adaptive Trait

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Abstract

As the ancestors of both the great apes and humans began to separate into two lineages, several distinctions emerged and solidified for the separate genera. It is suggested here that the sequelae to sexually transmitted diseases (STDs) and subsequent behavioral tendencies to avoid sexually transmitted diseases played an important role in forging the unique character of the Australopithecine/Homo line. In particular, the advantage of pair-bonding versus promiscuity in avoiding STDs would facilitate the crystallization of both the nascent nuclear family and the man-to-child affiliative bond. In addition, the unexpectedly small sexual dimorphism of Homo is suggested to be a partial consequence of replacing (physical) dominance acquisition as a reproductive strategy with the ability and motivation to form an on-going pair-bond. The capacity of males to send and the capacity of females to receive communication signals of male reliance and competence are suggested to be a key dynamic in the separation of the hominid line from the pongids.

Introduction

When humans and their nearest simian relatives are examined, two statements seem without serious challenge. First, humans and the great apes—the chimpanzees and the gorilla—have a recent common ancestry,[3] and second, humans and these great apes behave differently. This article attempts to complement the various extant theories that link the first and the second statements. That is, as humans and the great apes diverged from each other, they each developed their own behavioral repertoires, which resulted in descendants—over the millennia—to the present time. This article attempts to discuss the evolutionary effect of sexually transmitted diseases (STDs) upon (a) pair-bonding, (b) adult male provisioning, (c) the man-to-child affiliative bond, and (d) minimal sexual dimorphism.

Homo sapiens has two unique features which would be unexpected from a large, terrestrial primate: (i) sexual dimorphism is minimal and (ii) men systematically and actively provision particular women and their own children. In addition, the species harbors a wide array of sexually transmitted diseases (STDs) which can severely and adversely affect reproductive health of individuals and, by extension, of the group. This article attempts to lend insight into how these three features may have interacted with each other in our evolutionary history. The putative linkage of STDs with our phylogeny will be examined first. For a discussion and simulations/models of the relationship of STDs in humans and bio-cultural evolution, see Immerman (1986a, 1986b, 1988, 1992a, 1992b) and Immerman and Mackey (1997, 1999b, 1999c).

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[3] The literature and discussion on the exact phylogenetic relationship among *Homo*/*Australopithecus,* the various chimpanzees and the gorilla are impressive and sophisticated. However, for the purposes of this argument, the exact relationships are not germane. This argument accepts that humans and chimpanzees had a relatively recent common ancestor, and that common ancestor had a relatively recent shared ancestor with that of the gorilla. The argument put forward here is applicable to all of the phylogenetic permutations with which the authors are familiar.

STDs and Human Evolution

STDs represent unique phenomena in that the best prevention from individual infection—abstinence—is a formula for societal extinction. If everyone in a tribe or group were to engage in abstinence, then the tribe or group lasts only one additional generation. Even though all individuals who are born are guaranteed to have ancestors and an individual mortality, they are not equally guaranteed descendants. Hence, there is no guarantee for the perpetuity of the commonweal. If there is universal abstinence within a group, then there is no spread of infection; however, there would also be no children. If there are no children, then there is no survival of the tribe or community over generations. When the last childless person dies, the commonweal is extinct; e.g., the Shakers.[4]

Accordingly, sexual intercourse among its members is mandated for the survival of an intact, coherent social group. For all extant groups, there must have been continuous sexual interaction across the millennia. With this highly commonsensical mandate comes the potential of STDs.

Problems with STDs

Given that all organisms are subjected to diseases and parasites, why would STDs generate any unique problems? The source of the unique *sequelae* is not only the death of the host, but is also the dual threat (i) to the fertility of the host and (ii) to the viability of infants that are born. Plus, the viability of the clan or tribe itself is strongly related to the health of its infants.

It is unlikely, if not impossible, to evaluate the etiology of archaic sexually transmitted diseases and their *sequelae* (Zinzer 1963; McNeill 1976; cf. Krause 1992).[5] Over geological time intervals parasites, like any other organism, both arise and become extinct. Long-term adaptations to each other for both the host and the parasite can change the character of the host's response to the parasite and the parasite's effect upon the host. (See Ridley 1993 for a discussion on the long-term adaptations of host and parasite to each other.)

For example, in earlier eras, syphilis was much more of a fatal disease than it is currently (McNeill 1976). Both advanced medical treatments and

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[4] "Survival of the group" should in no way be construed as a variation on the theme of "group selection" as a/the replicator for Darwinian evolution. From this presentation's perspective, the gene—not the individual, not the population, not the meme—is the replicator which survives generation to generation. However, as both past and current events clearly and starkly illustrate, people do identify with a/their own cultural group, and more is the pity, do tend to eradicate alternative groups if given the chance. Examples include Cambodia, Rwanda, the Holocaust, pogroms, Stalin's vengeance upon the Ukrainians, influenza in Amazonia, the extermination of the Tasmanians, "ethnic cleansing" in Bosnia and Kosovo, *ad infinitum*. But, an individual's tribal totems may identify that person to those who would eradicate that individual—and his or her genes. If all of the members of a particular totem have been eliminated, whether by parasites, floods, colonial imperialism, *blitzkrieg*, or drought, then the group which is referenced by that totem, by definition, is gone. The unique genes or alleles shared by members of the obliterated group may live on in alternative individuals, e.g., captured "war brides," but the cultural entity whose members have been obliterated is no more. To the extent that a cultural entity invents or adopts customs, mores, or habits which prevent the replication of its own members' genes, that cultural entity can either extinguish itself or reduce its population enough to be easily overwhelmed by more prolific and predatory cultural/genetic competitors.

[5] Of course, the specific timing of the origin of STD-related infertility and infant morbidity/mortality in our ancestors is and may remain unknown (Long 1996). Nonetheless, it is suggested here that whenever STD-related infertility and infant morbidity/mortality did begin, an adaptive reaction would have also begun. A failure to adapt to STD-related pathologies would have led to decreased fertility. If the prevalence of the infertility were widespread, then the family, lineage, clan, or tribe wherein the high levels of STDs had occurred would be threatened with extinction. Given the demographic success of the current version of *Homo sapiens*, successful adaptations, by definition, had to have occurred.

It should be clearly noted that any extrapolation backwards in time would not necessitate a one-to-one correspondence between the characteristics of current STDs and those of their putative ancestors. Both the pathogens and their host have had numerous generations with which to (co)adapt to each other. In terms of modeling, it will be simply efficacious to utilize current consequences of STDs—which are known—rather than to speculate on past consequences—which are unknown and probably unknowable.

probably mutual adjustments between parasite and the host, i.e., humans, have attenuated its lethality. Accordingly, any current host/parasite interaction may not reflect the initial or even the intermediate interaction of by-gone years; especially when the unit of time used as a measuring rod is on the order of millions of years. However, as a general rule of thumb, parasites usually do not serve themselves well if they kill their host quickly. A host better benefits its parasites when the host stays alive and full of nutrients. STD parasites and their human or proto-human host would not be exceptions to this rule of thumb, nor could humans be considered transcendent to biological processes. Thus, although the ratio of archaic diseases' morbidity versus symbiosis is unlikely to be known or knowable, it will be argued below that current STDs do have an impact on fertility, infant mortality and infant morbidity. There is no reason to presume that archaic diseases would have been any more benign.

The thesis that humans have been vulnerable to STDs for a long time can be inferred from the specificity of the pathogens; whether the pathogen is a virus, a bacterium, or a metazoan. For example, herpes simplex virus type 2 (HSV-2) is transferred sexually and generally occurs in the genital area. The time depth of HSV-2 in humans is deep. Sakaoka et al. (1994) suggest that the split between herpes simplex virus type 1, usually transmitted non-venereally, and HSV-2, usually transmitted venereally, had occurred several million years ago. (See Sakaoka et al. 1995 for a similar argument, and see Nahmias 1992 and Nahmias and Dowdle 1968 for an overview.) Thus, the split would have occurred during the tenure either of the latter Australopithecines or in archaic *Homo*.

Another example is the human papillomavirus. There are more than 70 known types of human papillomaviruses (HPVs) (Ong et al. 1996). Some human papillomaviruses can be passed venereally and can cause disease. For example, HPV types 16 and 18 are passed venereally and are causal agents in the development of cervical and vulvar cancer (zur Hausen 1996). Ong et al. (1993) suggest that the diversity between HPV types evolved over several million years. See Chan et al. (1992) for a similar argument and conclusion. Of additional interest, Bernard, Chan, and Delius (1994) argue that HPV type 13 (which exclusively infects the oral cavity) separated from its closest relative—the pygmy chimpanzee papillomavirus—approximately five million years ago.

Gonorrhea is caused by the bacteria *Neisseria gonorrhoeae*. McGee et al. (1990) infected both female baboons and female chimpanzees with *N. gonorrhoeae*. The gonococci attached to, damaged, and invaded the oviduct (fallopian tube) mucosa of the chimpanzees, but not the oviduct mucosa of the baboons, both *in vivo* and *in vitro*. The pattern of gonococcal infection in chimpanzees was identical to that in humans, whereas the pattern in baboons was like that in other mammals (in which the gonococci do not cause genital infections). Lucas et al. (1971) were able to infect male genitals of chimpanzees *in vivo*, whereas DiGiacomo et al. (1977) were unsuccessful in infecting the genital tract of baboons *in vivo*. Thus, gonococcal infection was possible in chimpanzees, as it is in humans, but gonococcal infection could not be demonstrated in baboons. McGee et al. (1990) suggest that these data indicate that hominoid susceptibility to gonococcal infection began during the evolutionary interval between the baboons and the chimpanzees. Framed a little differently, the susceptibility arose after the monkey-pongid split, but before the hominid-chimpanzee split. Hence, genital infection from gonorrhea was possible among the Australopithecines as well as among early *Homo*.

Lice are metazoan. Pubic lice (*Phthirus pubis*), head lice (*Pediculus humanus capitis*), and body lice (*Pediculus humanus humanus*) each specialize in a segment of the human body and are not interchangeable (Billstein 1990). Again, this specificity suggests an extended time frame back to their common ancestor.

Because STDs have not been reported in feral chimpanzees or gorillas (Lockhart, Thrall, and Antonovics 1996; Phillips-Conroy et al. 1994; Kraus, Brown, and Arko 1975; cf. Heldstab et al. 1981; Nunn, Gittleman, and Antonovics 2000), it is reasonable to infer that humans' unique and on-going vulnerability to STDs occurred after the pongid-hominid split.

STDs are not Benign Occurrences

Current problems related to human fertility which stem from contemporary STDs involve a myriad of pathogenic agents. There are more than 50 sexually transmitted organisms and syndromes recognized today (CDC 1990). These organisms include bacteria, viruses, ectoparasites, fungi and protozoa. The pathologies caused by these organisms include, but are not limited to, infertility, chronic pelvic pain, copulatory pain and ectopic pregnancy due to gonorrhea and chlamydia; anal, cervical, penile and vulvar carcinoma due to human papilloma virus; acquired immunodeficiency due to HIV; hepatitis and hepatic cancer due to hepatitis B virus; and life threatening fetal, neonatal and infant infections (i.e., syphilis, HIV, herpes simplex virus, and hepatitis B virus)(Aiken 1992; Holmes et al. 1990; McDermott, Steketee, and Wirima 1996; McDermott et al. 1993; Schulz, Cates, and O'Mara 1987; Villa 1997).

These current STDs present a wide range of consequences that can lower female fertility and neonatal viability. It is important to note that STDs would lower an individual's ability to replace themselves in proportion to the prevalence of the STDs. For example, if syphilis is contracted by a woman, the chances for miscarriage, infant death, stillbirth or prematurity are all increased (Schulz et al. 1990; Waugh 1990). Furthermore, if the mother is infected the chances are also substantial that the fetus will contract syphilis from the mother which would decrease the child's life chances: life chances which would include a reproductive history.

The proportions are not small. Untreated syphilis during pregnancy is passed to virtually 100% of infants: 50% result in prematurity or perinatal death (Schulz et al. 1990). In a 1917 (pre-antibiotic) study of 1000 syphilitic pregnancies, 8% ended in still-births, 23% in infant deaths and 21% of the infants had contracted syphilis; i.e., over half (52%) of the infants were severely affected. The corresponding numbers for the controls (n = 826) were 2%, 11%, and 0% respectively (or a total of 13%) (Schulz et al. 1990).

Examples of *sequelae* following congenital syphilis for the neonate include deafness, dental defects, bony lesions, eye lesions and nervous system lesions including mental retardation, obstructive hydrocephalus and seizure disorders. None of these conditions would seem to enhance an individual's reproductive history or an individual's desirability as a mate. Acquired immunodeficiency syndrome (AIDS), of course, is a fatal disease, without a known cure, and is also associated with higher infant and child mortality (Taha et al. 1995). See Haldane (1949), Barkow (1989), Graves and Duvall (1995) and Hamilton and Zuk (1982) for complementary discussions, and for examples from non-humans, see Sheldon (1993).

The current leading cause of infertility is pelvic inflammatory disease (PID) as caused by STDs (e.g., *C. trachomtis* and *N. gonorrhoeae*) (Aral, Mosher and Cates 1991; Weström and Märdh 1990; Harrison and Alexander 1990; Peterson, Galaid, and Cates 1990; Wolner-Haussen, Kiviat, and Holmes 1990; Moore and Cates 1990; Weström 1987; Moore and Spadoni 1984). Basically, these STDs can infect (salpingitis) and scar the fallopian tubes. Consequently, such afflicted women can have impaired conception or have a tubal (ectopic) pregnancy. Note that ectopic pregnancies represent the leading cause of maternal deaths during the first trimester in the U.S. (Herbertson and Storey 1991; JAMA 1995). Prior to effective and sterile surgical procedures, e.g., 1880, the predominant prognosis for an ectopic pregnancy was maternal death (72%-90% death, 28%-10% survival). The current prognosis is 99%+ survival and less than 1% death (Lurie 1992). A recent study in Sweden (Weström et al. 1992) indicated that occluded fallopian tubes significantly decreased the chances for a successful attempt at becoming pregnant, and, if pregnancy did occur, also (significantly) increased the chances of an ectopic pregnancy by at least a factor of six (Table 1).

Gonorrhea, a cause of PID, has also been associated with increased chances of having stillbirths, prematurity (low birth weight, hence increased morbidity), prolonged labor, and spontaneous abortion (Brunham, Holmes, and Embree 1990; Corcoran and Ridgway 1994; Weström 1991). In addition, the infectious agents (e.g., chlamydia and gonorrhea) which cause PID also create a whole range of other neonatal problems: opthalmia, conjunctivitis, pneumonia, and arthritis (Gutman and Wilfert 1990).

Table 1. Reproductive history of women, who, after index laparoscopy were diagnosed as having abnormal (e.g., occluded) fallopian tubes [patients] or has having normal (i.e., symptom free) fallopian tubes [controls] (Adapted from Weström et al. 1992).

	Patients (*n* = 1732)		Controls (*n* = 601)		
Reproductive events	*n*	%	*n*	%	Z
Avoided pregnancy	370	21.4%	144	24.0%	1.33
Attempted pregnancy	1309	75.6%	451	75.0%	0.29
Became pregnant	*1100*	*84.0%*	*439*	*97.3%*	*7.34*
(first pregnancy ectopic)	(100)	(9.1%)	(6)	(1.4%)	(5.38)
No pregnancy occurred	*209*	*16.0%*	*12*	*2.7%*	
(examination found tubal infertility)	(188)	(14.4%)	(9)	(2.0%)	
(examination indicated non-tubal infertility)	(21)	(1.6%)	(3)	(0.7%)	
Not pregnant for unknown reasons	53	3.0%	6	1.0%	
Totals	1732	100.0%	601	100.0%	

Moore and Cates (1990) estimate that, after a single episode of PID, infertility resulted in 6% of the mild cases, 13% of the moderate cases and 30% of the severe cases. (Note that these figures arose even when treatment was available.) Each successive bout of PID doubles the chances of infertility. For example, Weström and Märdh (1990) reported that one episode of PID rendered 11% of the patients infertile, two episodes left 23% of the patients infertile, and three or more episodes resulted in 54% of the patients becoming infertile. See Tables 2 and 3 for additional examples of the adverse impact on fertility by repeated infections of STDs. The age of the woman is also relevant in terms of the infection's impact. Among those women who were seeking to achieve a pregnancy but had experienced one episode of acute PID, 12.6% in the age group of 15-24 years and 25% of the 25-34 year old cohort were either infertile from their infection or had an ectopic pregnancy (Weström and Märdh 1990). It should be reiterated that these statistics arise in a time and a society with readily available medical information, medical technology and inexpensive antibiotics. In the pre-antibiotic era, after being infected with gonorrhea, up to 70% of the women had tubal obstruction (Moore and Cates 1990). Holtz (1930) estimated that 1.3% of the PIDs were lethal. Mosher and Aral (1985) calculated that PID accounted for a third to a half of recent increases in infertility.

Table 2. Chances of a woman becoming sterile from gonorrhea by number of lifetime infections (adapted from McFalls and McFalls 1984).

Number of lifetime infections	Cumulative percent who become sterile	Percent who remain fertile	Total percent
1	14.1%	85.9%	100.0%
2	25.9%	74.1%	100.0%
3	36.4%	63.6%	100.0%
4	45.4%	54.6%	100.0%
5	52.9%	47.1%	100.0%
6	59.2%	40.8%	100.0%
7	65.5%	34.5%	100.0%
8	68.3%	31.7%	100.0%
9	77.5%	22.5%	100.0%
10 or more	77.5%	22.5%	100.0%

An infection of herpes simplex virus increases the chances of prematurity, low birth weight (again, strongly associated with infant mortality/morbidity) and spontaneous abortion. The maternal infection could also be transmitted to the infant. A herpes simplex virus infection in an infant can spread to multiple organs including the central nervous system, the lung, the liver, the adrenals, the eyes, the mouth, and the skin. When the virus affects multiple organs, there is a 60% mortality rate at one year. Of the survivors, 44% are left with permanent neurologic impairment (Stagno and Whitley 1990). Again, these statistics are from a time and a place where modern medical techniques were utilized.

Although these pathologies, *inter alia*, are not necessarily lethal to the nubile woman, the symptoms of these pathologies would tend to decrease the afflicted individual's level of competitiveness in attracting desirable mates. To the extent that these infections affect skin texture, body odors, genital secretions, general activity level and romantic tendencies (see Buss 1989, 1994 and Buss and Schmitt 1993 for examples), the chances for a successful impregnation are similarly decreased.

It is important to note that the number of sexual partners is the best (current) predictor of the chances of having a sexually transmitted disease (Allen et al. 1991; Aral, Mosher, and Cates 1990; Brunham and Plummer 1990; Hunter et al. 1994; Laumann et al. 1997; Moore and Cates 1990; Newell et al. 1993; Thrall, Antonovics, and Bever 1997; Weström and Märdh 1990). The greater the number of partners, the greater the chance for an infection. It should also be noted that there is an asymmetry in the rate of transmission between the sexes. The rate of transmission from male to female is greater than the rate from female to male. Although males are relatively less likely to become infected, once infected, they are contagious and able to infect additional partners. While current STDs rarely render the infected male

Table 3. Normative *sequelae* to conceptions of syphilitic mothers as a function of number of pregnancies (Kunitz 1972).

Pregnancy number	Expected result
1st	Miscarriage at fifth month
2nd	Stillbirth at eighth month
3rd	Live birth, but death from syphilis soon after birth
4th	Live birth, but the infant shows signs of syphilis weeks or months after birth
5th	Initially a healthy child who shows signs of syphilis after a few years
6th	A healthy child until the teen years, then shows signs of syphilis
7th	A healthy child who never shows signs of syphilis

sterile, once the male passes the infection to a woman, the woman's fertility (e.g., blocked Fallopian tubes, ectopic pregnancy) is threatened as is the success of a pregnancy (e.g., still births, etc.) (Howards 1995; Hook and Handsfield 1990; Joesoef et al. 1991). Thus, the male's fertility becomes effectively impaired in relation to and in proportion to the sterility of his partners.[6]

An illustration of the suggested dynamics would be the initial encounter of British sailors with the Maori of New Zealand. The Maori population had been isolated from most of the world and all of Europe for a very long time. Early English writers on the Maori noted a fairly unrestricted sexuality on the part of their women (Buck 1962; Crosby 1986). Early English sailors were also probably a good deal unrestricted. Venereal disease from the English sailors spread rapidly within the Maori population. In the 1850s, Francis D. Fenton, in the process of conducting a census of the Maori population, gathered data on 444 Maori wives. Of the 444, only 221 had any living children and 155 were completely barren. A colonial surgeon in the area noted that in a sample of 230 Maori women, 124 either had no children or had no living children (Crosby 1986). Crosby (1986) notes that there were many possible explanations for the Maori barrenness, e.g., infanticide, "but the worst villain in the tragedy was surely venereal disease. It kills parents, kills fertility, kills fetuses, kills children, and erases the desire for children" (p. 257). The notes from an early clinic in New Zealand (1837) recorded that 3.8% of the Maori were diagnosed with a venereal disease (Crosby 1986).

A similar process occurred in Siberia. Small pox and venereal diseases were introduced into the local groups by the Ostyak, Tungus, Yakut and the Samoyed (Crosby 1986; Donner 1954; cf. the Chukchi [Bogoras 1901]). Of these, the Tungus practiced sexual hospitality with strangers: "A woman is not food—she does not decrease" (Shirokogoroff 1979:72). The two diseases decimated the local populations.

[6] This biological fact of asymmetry in gender transmission and gender susceptibility to infertility would help lay the foundation for the cultural trend of the double standard/asymmetry in latitude of sexual behavior between the two genders.

It should also be noted that, unlike some other diseases (e.g., measles), STDs are not dependent upon a large, dense population to remain within the host population. STDs are more dependent upon the numbers of sexual partnerships and the frequency of matings for their persistence rather than upon the numbers which comprise the population (Thrall and Antonovics 1997; Thrall, Antonovics, and Wilson 1998; see Ewald 1993 for an over-view on the evolution of virulence). Said differently, STDs can exist and be maintained over generations within a small population. The consensual view is that early *Homo* was a hunting and gathering primate that lived in small mobile bands or tribes. Accordingly, such demographics would not preclude STDs from chronically impacting upon the social and behavioral dynamics of the tribes. See Pennington and Harpending (1991) for an example of a proposed relationship among the Herero of Botswana between a low population level and STDs.

Lessened Sexual Dimorphism in *Homo*

Sexual dimorphism is less pronounced in *Homo sapiens* than would be expected given our generally agreed upon ecological heritage as (i) a large, (ii) terrestrial primate, which is (iii) non-obligate monogamous. Dominance displays by human males that are based on their own physical attributes also seem to be substantially restricted. This article seeks to address one avenue by which expected dominance displays by human males would have been selected against and thereby reduced (see Immerman 1986a, 1992b, 1993 for a complementary discussion).

Although dominance, as a construct, has a rich history with variegated definitions, this article has a narrow focus. A dominance display is defined here as a behavior or a physical characteristic on the part of one adult male which is directed at other adult males to allow differential access to breeding females. Successful dominance displays by an adult male would enhance that adult male's access to breeding females. Unsuccessful dominance displays or lack of dominance displays would decrease the male's access to breeding females (see Ellis 1995 and Dewsbury 1982 for examples of the relationship—sometimes stark and sometimes slight—between increased male dominance and increased reproductive success).

Parameters of Sexual Dimorphism in Primates

Although there are exceptions, sexual dimorphism[7] (e.g., by weight; Hall 1985) tends to be greater in (semi)terrestrial primates than in arboreal primates (e.g., baboon [*Papio* 185] vs. spider monkey [*Ateles* 94], gorilla [*Pan gorilla* 219] vs. langurs [*Presbytis* 107]). Again with exceptions, larger primates tend to have more sexual dimorphism than smaller primates (e.g., orangutan [*Pongo pygmaeus* 199] versus night monkey [*Aotus trivirgatus* 102]). Sexual dimorphism also tends to be lesser or nonexistent in primates which tend to be monogamous (e.g., gibbon [*Hylobates* 104] versus chimpanzee [*Pan troglodytes* 121]; marmoset [*Callithrix jacchus* 95] versus macaque [*Macaca* 149]). The argument is that, when males exchanged the harder-to-scan world of the trees for the easier-to-scan world of the ground, they were better able to assert dominance and have multiple sexual partners. Indeed, terrestrial primates are more prone to be polygynous than are arboreal primates (Jolly 1985; Hrdy 1999). Accordingly, after it was freed from problems of fissile tree limbs and incessant gravity, additional male size would be advantageous in creating dominance for the larger male and in creating submission in the smaller male (see Fleagle 1988; Martin, Willner, and Dettling 1994; McHenry 1991; Richard 1985 for examples and discussion). Hence, more effective male-to-male dominance displays/aggression could then be translated to multiple partners which would lead to a greater number of descendants who, in turn, would pass on the genetic material underpinning the physical attributes of the "successful" display. The same argument would apply to increased canine size and enhanced piloerection or other display items (e.g., manes) which could be used to gain dominance and, thereby, to gain access to more sexual partners, and, hence, to sire more descendants.

[7] By convention, the sexual dimorphism ratio is computed by setting the female value at 100 and setting the male value in relation to the female value.

There are three givens that apply here: (a) *Homo*'s predecessor *Australopithecus* did exhibit a large degree of sexual dimorphism by size (Hall 1985; Plavcan and van Schaik 1997); (b) *Homo*, compared to *Australopithecus*, gradually increased in size (Hall 1985; Aiello 1994); and (c) *Homo* became exclusively terrestrial. From these three givens a not unreasonable, inferred assumption would be that *Homo* would follow the basic trend of maintaining or increasing sexual dimorphism. However, sexual dimorphism decreased (Arsuaga 1997; *Economist* 1994; Lewin 1987; Lockwood et al. 1996; McHenry 1991).

In terms of height, the sexual dimorphism of contemporary humans is 107 (SD 1.5; n = 93 [societies]; i.e., women are 94% the height of men) (Alexander et al. 1979). The human canine is virtually (sexually) isomorphic, and piloerection is not a functional human trait. In terms of weight, the sexual dimorphism of (U.S.) humans is 130. Since the linear correlation between the weight of primate males and the sexual dimorphism of their species is significant (r_p = .569; n = 47; $p < .01_{\text{two-tailed}}$) (Hall 1985), then the sexual dimorphism of human males could be predicted from their weight. When the "sexual dimorphism ratio" is predicted from the average man's weight, the predicted value is a sex ratio for male-to-female of 187.4. This predicted value overestimates the actual value of 130 by 1.55 standard deviations.[8]

In other words, humans are far more sexually isomorphic than would be expected by the ecological circumstance of their phylogeny. It is argued here that there were selective pressures against dominance displays in early *Homo*, and that an excellent candidate for one such agent that generated the negative selection is sexually transmitted disease (STD). A complementary candidate (from Hrdy 1999) is that larger mothers—once terrestrial—were positively selected because of the sheer advantage that size has in manipulating the environment for herself and her children.

STDs and Selection Against Dominance Displays

If, as is reasonable to assume, the dynamics of STDs were operative for extended eras—albeit not necessarily the specific and current diseases—then there would be selective advantages in traits which vitiate the frequency or incidence of the dynamics. The argument presented here is that selection against dominance displays (e.g., size) in hominid males was one such consequence to the threats of STDs.

Framed a little differently, the males who successfully utilized dominance displays to gain access to multiple sexual partners would relatively increase their chances of becoming infected with a STD. Once infected, they were at increased probability to infect (numerous) females, which, in turn, would increase both the chances of female sterility and/or infant morbidity/mortality. In other words, more effective dominance displays would have led to increased rates of infection and sterilization, without necessarily leading to more impregnations, and would have led to more sickly or dead infants rather than to healthy progeny. That is, the dominance displays which led to more sexual partners would not necessarily lead to more progeny who would have progeny of their own.

Those males with less (effective) dominance displays would successfully copulate with fewer females, and, thereby, have a reduced risk of infection. Those infection-free copulations would have an enhanced likelihood to result in healthy pregnancies and healthy infants. Hence, the less dominant male would be less likely to spread a STD infection. This male, *ceteris paribus*, would have a relatively increased level of fertility and would contribute to a lower incidence of infant morbidity/mortality: relatively fewer partners, but relatively more healthy descendants. Of course, the same argument would apply to the female. Her acceptance of a non-dominant male as a sexual partner is intuitively counter-productive in the short term: "I have just copulated with an ineffectual partner who would sire comparatively ineffectual offspring." However, in the long term,

[8] Using a similar method for data from Plavcan and van Schaik (1997), a similar result occurred. The correlation (r_p) of .389 was significant (n = 86; $p < .01_{\text{two-tailed}}$). The predicted sexual dimorphism ratio was 155, or 1.15 standard deviations larger than the actual male-to-female ratio.

(i) conceptions will predominate over sterility, (ii) live infants will predominate over stillbirths, (iii) healthy children will predominate over sickly children, and (iv) live mothers will predominate over dead mothers. Said differently, there is no advantage accrued to the woman, in terms of number of offspring, if she has only one partner or if she has multiple partners. Multiple partners may be (quantitatively) advantageous for the male, but not for the female: a definite asymmetry exists here. The trade-off for the female is one of perceived genetic benefits for her offspring (an exemplar of the group has fathered her child) versus increased chances of being infected with a sexually transmitted disease (the exemplar of the group, who has many other sexual partners, has just sterilized her).

Men Systematically and Actively Provision Particular Women and Their Own Children

A cross-cultural universal is that men will gather resources—food is an excellent example—from outside the perimeter of their camp/village and then return to the camp/village and share that resource with particular women (wives) and particular children (the men's children). The pattern is highly predictable (Hewlett 1992; HRAF 1949; Lamb 1987; Mackey 1985, 1986, 1996) and occurs across societal structures and across ecologies (Murdock 1957, 1967); examples include the Yanomamo (Chagnon 1977), China (Chance 1984), Tibet (Ekvall 1968), the Tiwi of Australia (Hart and Pilling 1960), the Dani of New Guinea (Heider 1979), Japan (Norbeck 1976), Australian aborigines (Tonkinson 1978), and the Yuqui of Amazonia (Stearman 1989). The provisioning is not totally exclusive. Systematic food sharing has been ritualized in many, if not all, societies. Rarely can a hunter claim a large kill for only his own family (Coon 1971; Lee 1982; Tonkinson 1978). But, within these contexts, it is a universal that a man provides singular attention to the woman/women to whom he is married and to the legitimate children that he has fathered in terms of protection and provisioning. (See HRAF 1949; Malinowski 1927 and Hendrix 1996 for theoretical discussions; and Brown's 1991 presentation of human universals.)

When resources are not forthcoming from a prospective groom, brides are difficult to acquire (Cashdan 1993) and wives are difficult to keep (Betzig 1989). When the pattern of male provisioning does break down across the overall society (e.g., the Ik; Turnbull 1972), the breakdown signals an overall societal disintegration and is a focused topic of the ethnographer's analysis.

Adult males' active and systematic food sharing—i.e., when (i) the men procure food from outside of the group's perimeter, and (ii) they then return to the group for sharing with females and their young—is not a primate trait. Generally, adult male primates do not procure food and then return to the adult females and their young to give away that food. The adult male primates may allow a shared feeding from the same source, or may relinquish food to a "begging" female; e.g., among chimpanzees (Boesch 1994; Boesch and Boesch 1989; de Waal 1997, 1998; de Waal and Lanting 1997; Goodall 1986; Nishida and Hosaka 1996; Stanford 1996; Teleki 1973; Parish 1996). However, these adult males do not leave the perimeter of the troop, obtain food, and then return to the troop to give the food to adult females, who might then give it to their young.

While not a primate characteristic, food sharing by adult males does occur in many—if not most—bird species, especially if the species tends toward monogamy (Kleiman 1977), and in the canids: wolves (Mech 1966; Mowat 1963; Murie 1944), coyotes (Dobie 1949; McMahan 1976; Ryden 1974; Young and Jackson 1951), jackals (Lawick and Lawick-Goodall 1971; Moehlman 1980), hunting dogs (Kuhme 1965), and foxes (Alderton 1994). (See King 1980; Mackey 1976, 1996; and Thompson 1978 for discussions.) For example, the adult male wolf will catch prey, return to the den, and give the food via regurgitation to the mother wolf and her/his pups for their consumption (Mech 1970).[9] Of further interest, these canids also tend toward facultative monogamy and toward minimal sexual dimorphism (Kleiman 1977). An argument can be made that convergent evolution has occurred between adult male canids and the adult male *Homo* (see Mackey 1976, 1996 for expanded discussion on putative

convergent evolution). However, one important difference between the canids and *Homo* is that there is often only one mating (alpha) pair in a canid pack, whereas each man and each woman can expect to mate/marry and have children.

An Independent Man-to-Child Affiliative Bond

Perspectives on Fathering prior to 1976

Until 1976, academics or professional perspectives on (U.S.) fathers were fairly simple. Mothers were responsible for the social, behavioral, emotional and cognitive development of the young child. Fathers were to assist mothers, both directly and indirectly, and were to be a (primary) breadwinner or provider who would develop resources with which the mother could nurture his/her children. An example of this maternal primacy is the Foss series (1961-1969) *Determinants in Infant Behavior*. There are over 600 references cited in the series. Of those 600 references, 91 refer directly to "mother/maternal" whereas only one citation refers directly to "father/paternal," and this citation is to Itani's study on the macaque monkey.

As he developed his theory of attachment, John Bowlby (1961, 1969, 1979) introduced the term "monotropy." Monotropy suggested that an infant needed a stable mother figure with whom to attach and thereby form the basis of trust and intimacy for later adult relationships (see Schaffer and Emerson 1964, who argued that an adult was needed for such attachment, but the adult could be male or female; however, Bowlby's influence was clearly more potent in academia than was Schaffer and Emerson's). Margaret Mead (1949:185-190) coined the phrase that motherhood is a biological necessity, whereas fatherhood was a social invention (often quoted as a "social accident;" e.g., Parke and Sawin 1977). The popular press generally reflected the academic paradigm. For reviews of popular culture's view of U.S. fathers, see Demos (1986), Griswold (1993), LaRossa (1997) and LaRossa et al. (1991). A father as a "nurturer" was viewed as essentially irrelevant in his young child's life (cf. Freud 1964; while currently out of style, it may have been prescient).

Of interest, the influential developmental psychologist Harry Harlow (1971)—generalizing from his work with the rhesus macaque—created the syllogism: (i) Men like to be with women; (ii) women like to be with their children; therefore (iii) men are found with children as a derivative effect. That is, because women are proximate to children and men like to be proximate to women, then men become proximate to children. Adams (1960) had earlier presented the same theme, and Smuts and Gubernick (1992) resurrected the thesis.

However, more recent empirical work has argued for an independent man-to-child affiliative bond (Hewlett 1992; Lamb 1976; Mackey 1985, 1996; see Blankenhorn 1995 and Popenoe 1996 for reviews of the literature). In gist, men, both in the absence as well as in the presence of women, assume nurturing duties plus "play"/interact with their children in a caretaking mode. These parenting behaviors are directed both at their daughters and at their sons. Such parenting behaviors are found systematically, cross-culturally, and in non-trivial numbers; i.e., fathering behaviors are predictable across cultural boundaries, and cannot be considered as a form of error variance. For example, in 23 cultures studied (i) in public places, (ii) away from the domicile (e.g., parks, markets, playgrounds), (iii) during daylight hours, and (iv) when men are not precluded from being with children by local cultural norms (e.g., working hours), then over a fifth (20.9%, SD = 5.81) of the children who were with adults were with (at least) one man, but no woman was with them (Mackey 1996, 2001). This incidence of such man-child dyads, well away from their domicile and with no women proximate, would not be predicted from any of the studied primate species. Because of the marked size and power differentials between men and children, the implication is strong that the men are

[9] It may be useful to contrast this canid set of behaviors with the isolate tiger, leopard or cheetah wherein only the mother nurtures her young. When it comes to rearing offspring, the adult male is totally irrelevant. Also contrast the canid set of behaviors with the social lion and the social hyena. Adult male lions and hyenas are considered a threat to the mother's young (Guggisberg 1963; Kruuk 1972; Lawick and Lawick-Goodall 1971; Rasa 1986; Rudnai 1973; Schaller 1972). In addition, neither adult male lions nor adult male hyenas return with food to relinquish for their young.

with their children because they—the men—choose to be. Fieldwork and a cottage industry of anecdotes strongly suggest that the men choose the association because they enjoy being with their own children and "like" them. Another third (34.7%) of the children were with (at least) one man and (at least) one woman. The rest of the children were with (at least) one woman, but no man was with them (Table 4).

Discussion

If a STD were to be introduced within an intact tribe of early humans, then those females who were infected could expect an increased loss of their descendants by (i) loss of potential children from the mothers' ectopic pregnancies (and their death), (ii) loss of potential children from sterility via STDs, (iii) offspring wastage, i.e., spontaneous abortion or stillbirths, from infected mothers, and (iv) loss of children via neo-natal deaths. In addition, *sequelae* from STD infections would lower any surviving child's attractiveness when he or she reached maturity. Conversely, those females who avoided infections would not be burdened with these traits which adversely affect their reproductive success.

Of course, in a pongid group—chimpanzees and gorillas—virtually all the mature females will mate with the dominant male or males. Thus, it would be difficult to avoid an infection once the STD was introduced. And, indeed, models which presume universal mating within a tribe/troop which, in turn, harbors a STD indicate that nearly all females become infected, and fairly quickly so (see Immerman and Mackey 1999a for examples).

Nonetheless, one scenario would, over generations, very slowly allow females to systematically avoid a STD infection. Namely, if a newly matured female were to mate with a non-dominant male, then a pregnancy could ensue with a decreased chance of infection. The youth and sexual inexperience of the female somewhat protects her from infection. The lack of dominance by the male, hence a commensurate lack of mating opportunities for him, somewhat protects him from infection.

At this point, Fisher's (1983) *Sex Contract* becomes a germane construct (see Immerman and Mackey 1999a for complementary discussion). To wit: Fisher argues that (Australopithecine/archaic *Homo*) women exchanged relative sexual exclusivity with a man for his unique, systematic, reliable provisioning and protecting of her and her (also his) children. Such an early negotiation seems reasonable. The non-dominant male cannot offer his dominance as an inducement to the female for her to have sexual intercourse with him. However, an alternative inducement—food would be a likely candidate—may serve the same function to obtain sexual activity. Over time, as the exchange became more common for the participants, particular males might favor other particular females who would agree to the exchange, and, thereby, a nascent, if inchoate, pair-bonding would also gain momentum.

The exchange would benefit both genders. Females increased the quality of their diet. Males had an increased opportunity to sire offspring. For both genders, their genetic tendencies would thereby become (over)represented in subsequent generations.

Given the longevity of large sexual dimorphism, there is no reason to believe that sexual exclusivity would be aligned with the reciprocal agreement. The dominant males would still have copulated with preferential females at preferred times (during ovulation).

Nonetheless, over time females would learn to exploit a new resource, a new source of very nutritious food: the male. As a consequence, the female had a new problem to solve in her reproductive strategy.

That is, dominant males were "dominant" because previous ancestral females defined them as such. If a female were to maximize her chances for descendants, then the female needed her sons to mimic the "dominant" features that were attractive to females. Similarly, she needed her daughters to find dominant males sexually attractive.

On the other hand, the female will have neither sons nor daughters if she is weakened or sickened due to a poor diet and dies. The balance between short-term health (ontogeny) and long term descendants (phylogeny) was probably delicate indeed. It is suggested here that, however slowly the shift occurred, "diet" won out.

Table 4. Associations (in percentages) among (all) children and adult groups in 23 cultures. The data are from times that were discretionary for men (adapted from Mackey 2001).

Culture	Number of Children	Adult group			Total % of children
		Men-only	Women-only	Men and Women	
Israel	2139	31.9%	53.5%	14.6%	100.0%
Iceland	1694	29.0%	39.6%	31.4%	100.0%
Morocco	1398	28.5%	56.8%	14.7%	100.0%
India	1104	28.5%	43.0%	28.5%	100.0%
Brazil-urban	542	24.4%	46.4%	29.2%	100.0%
Taiwan	2790	23.8%	56.8%	19.4%	100.0%
Ireland	1852	22.9%	36.5%	40.6%	100.0%
Japan	1336	22.9%	38.3%	38.8%	100.0%
Brazil-rural	549	22.8%	54.3%	22.9%	100.0%
Kenya	748	21.9%	49.2%	28.9%	100.0%
China	2162	21.3%	20.2%	58.5%	100.0%
Senufo (Ivory Coast)	1132	21.0%	41.5%	37.5%	100.0%
Hong Kong	164	20.7%	32.3%	47.0%	100.0%
Austria	132	20.5%	37.1%	42.4%	100.0%
Sri Lanka	1973	20.5%	64.7%	14.8%	100.0%
United States	14,692	20.5%	43.65%	35.85%	100.0%
Virginia	8953	17.5%	43.5%	39.0%	100.0%
Iowa	639	18.6%	46.6%	34.8%	100.0%
Texas	873	21.8%	44.3%	33.9%	100.0%
Kentucky	309	30.7%	44.7%	24.6%	100.0%
NE + CA	2561	18.8%	33.2%	48.0%	100.0%
California	1357	15.6%	49.6%	34.8%	100.0%
Ivory Coast	1642	17.4%	67.4%	15.2%	100.0%
London	397	17.4%	42.3%	40.3%	100.0%
Lima, Peru	490	17.4%	52.2%	30.4%	100.0%
Spain	1058	16.8%	31.5%	51.7%	100.0%
Mexico	1355	14.2%	50.2%	35.6%	100.0%
Paris	485	8.7%	22.9%	68.4%	100.0%
Karaja (Brazil)	399	8.3%	40.8%	50.9%	100.0%
Mean		20.9%	44.4%	34.7%	100.0%
SD		5.8%	11.9%	14.5%	

As reliance upon male provisioning occurred, two competitions were simultaneously affected: (lessened) male-male competition via physical dominance and (enhanced) female-female competition (see Lovejoy 1981 for a complementary discussion).

Male-Male Competition

Although there are variations upon the theme and each variation can have a rich context, male-male competition is simple: (i) males tussle, often ritualistically, and (ii) the winner gets the female(s). However, as females biased toward the exchange of provisioning for sexual activity, male-male tussling would become increasingly vacuous and unproductive. The winner won nothing. Progeny would be increasingly sired by providers—who could be of modest size—not by "alpha" males—who were often of immodest size.

Female-Female Competition

In terms both of willingness to copulate and of available sperm, dominant males are not a finite resource. They are essentially infinite. Female-female competition is valueless along this dimension. However, high quality food is finite and valuable. The initial ante for the female was granting sexual access. The ante increased to (the male's perceptions of) her sexual exclusivity, and the dynamics of "paternal certainty" were introduced into the species' reproductive strategy. Females who more successfully negotiated the exchange of sex for food had a dietary advantage versus those females who were less successful. Better diets enhanced survival. Poorer diets diminished survival.[10]

Female Reproductive Calculus

From what was probably a fairly simple choice for her female ancestors, a qualitatively different strategy had unfolded for this transitional female. For eons, her ancestors merely had to notice which male out-tussled his competitors. Such a winner would sire the females' children.

Her descendants would have a much more complex evaluation to make. She, in competition with other females, had to shift from what the male's physical dominance had achieved in the past—winning the tussle—to what a man would do in the future—be able and be willing to share valuable resources with her (and her children).

Again, it is useful to note that the female, who would exchange sex for resources from a non-dominant male, would also exchange sex for dominance from the dominant male(s). Thus, any forging of a pair-bond system within the species would be very slow to congeal (Appendix A). However, once the

[10] Both the small brain plus the lack of artifacts (which would indicate otherwise) of any Australopithecine/archaic *Homo* suggest a minimal cultural quiver: symbolic or material. Any learned adaptations that may have been passed down as a tradition were probably done non-linguistically, non-verbally. Analyses of teeth and bones indicate an omnivorous diet gleaned from the African savannas. Several Australopithecine species existed co-terminously. Arguably, several early *Homo* species also were contemporary with each other.

Our lineal ancestors, and perhaps others, had developed a primitive, yet—by definition— effective, lithic technology. But, the question can be asked: "What good is a rock with a sharp edge in the savanna, or even in the forest?" A thrown rock may chase away competitors, but reducing the size of the rock by removing flakes also removes percussion power. A wide, blunt rock is a more effective hammer on, for example, nuts, than a narrow, sharp one. The enhanced efficiency of removing meat from a bone with a rock rather than one's teeth *cum* fingers seems problematic. What is not problematic is the ability of the sharpened edge of a rock to smash open long bones that contain marrow. Bone marrow is rich in nutrients, especially fats.

While larger and faster carnivores, such as lions, leopards, and hyena packs, may enjoy a prohibitive advantage in hunting and/or appropriating scavenged kills, they would still have difficulty freeing the marrow from the bone's casing. Here our ancestors with their sharpened rocks had an advantage. If our primate homologues are used as a frame of reference, any obtained marrow would be eaten by the procurer. That procurer would then experience an enriched diet that would be otherwise hard to replicate from the savanna.

It is difficult to imagine the much smaller female out-muscling the larger male in initially attaining the marrow. What is less difficult to imagine is the female exchanging sexual favors in return for the marrow (see Fisher 1983 for a discussion). The exchange would be especially appealing to a non-dominant male who might otherwise experience less access to females than the established dominant (alpha) males.

pair-bonding/facultative monogamy became an option within a tribe/troop, the advantages of a pair-bond/monogamous union in avoiding the deleterious consequences of STDs (upon reproductive success) would be relatively quick and decisive. A woman who forms a pair-bond with a man has reduced chances (i) of ectopic pregnancy, (ii) of infertility caused by PID, and (iii) of infirm offspring. In addition, both the man and the woman of the pair-bond, within their own adult lives, have reduced chances of being harmed by the negative consequences of STDs.

Furthermore, any of the other three options—(1) promiscuous male and promiscuous female, (2) promiscuous male and pair-bonding female, and (3) pair-bonding male and promiscuous female—are far more likely to have reduced reproductive success (the dyad of the promiscuous male and the pair-bonding female has a slight advantage compared to the other two, but is still much more vulnerable than is the dyad in which both partners are pair-bonded or monogamous (Immerman and Mackey 1999a; and see Appendix B).

Thus, the physical features of male dominance display which, heretofore, would have led to multiple partners and increased numbers of descendants, were now selected against. Male dominance displays had become aligned with STDs, with infected partners and with reduced numbers of descendants. Non-dominance by the male had become aligned with enhanced health, enhanced provisioning, and more descendants. The large degree of sexual dimorphism that signaled dominance of the alpha-male became markedly reduced. His copulatory success had become the inverse of his reproductive success.

Shifts in Mating Communications

The nubile human female was now faced with a reproductive strategy more complex than any of her distant ancestors and any of her simian cousins. She was to find a man—a mate—who, not only could gather resources and provide protection for her, her children, and their resources, but would also willingly share those resources with her and her children. A man who could provision and protect, but chose not to do either would be of minimal use to the woman and her children. The preferred man/husband had to be both able and willing to provision and protect (see Buss 1989; Lovejoy 1981, and Symons 1979 for complementary discussions on the selectivity of mating partners).

Framed differently, there was a shift in mating communication systems. Heretofore, the male who could display dominance within the male hierarchy, and thereby achieve a dominant status, would be attractive to fertile females. The females would "read" his high dominance status and thus accept him as a mating partner. With the advent of pressures from STDs upon fertility, any STD avoidance behaviors made the communication systems far more subtle. The female needed to avoid STDs (that is, avoid multiple sexual partners), plus, at the same time, she needed reliable protection and provisioning for herself and her children. Consequently, she needed to attract a man, who not only was dominant enough to generate resources and protection, he was also willing to share his time and treasure in the nurturing of her and his children.

On the other side of the coin, the successful male needed to communicate to females both (i) his ability to share and to nurture as well as (ii) his motivation to share and to nurture. Accordingly, the emphases in communication systems shifted from physical and behavioral displays toward psychological and behavioral traits. The successful man communicated to women his capacity and willingness to nurture. The successful woman could "read" the man's communications with accuracy and then act upon her "read."

Of course, such shifts in communication strategies lend themselves to counter-strategies. An increase in the subtlety in "false" advertising on the part of men would be continuously matched by the increase in the subtlety on the part of women's ability to discern the genuine from the ersatz. As the cultural phenomena of courtship ritual and marriage were superimposed upon the biological phenomenon of pair-bonding, the bride's kin groups entered into the evaluation of the prospects of the prospective grooms and the grooms' own families. Depending

upon the culture, such entry by the woman's kin group currently varies from mere advisory to vetoes to mandates (Van den Berghe 1979; Stephens 1963).

Man-to-Child Affiliative Bond

For the man, a woman offers sexual gratification. Between the man and the woman, there is a commonsensical *quid pro quo*: provisioning and protection exchanged for sexual exclusivity. Her children are another matter. *Prima facie*, the children offer the man nothing. They extract food. They interfere with adult copulations. They are asexual. They are noisy and annoying. Pongid males and the males of other terrestrial primates generally tolerate the young of the tribe (at least his young), but leave nurturing to the mothers (Goodall 1986; Smuts et al. 1986; Taub 1984). Yet, human males—men—are clearly fond of their children, nurture them, play with them and are quite willing to provision and protect them. All of those activities are at a cost to him, either economically or psychologically. Why would our male ancestors break with their primate history? The imperative to procreate more children rather than tend to already existing ones seems more reproductively efficient.

It is argued here that those males who did have a rudimentary affiliative bond toward (his/her) children—"liked" them—would have a clear reproductive advantage over those men who did not "like" (his/her/any) children. If sexual exclusivity were becoming a norm, then the children of the wife also tended to be the children of the husband. His nurturing of her children was also the nurturing of his children; i.e., his genes, his inclusive fitness. Framed a little differently, the father's emotion of affection for his children would be the drivewheel to allow paternal certainty to be a fruitful and effective strategy. Just as a husband's jealousy may reduce his wife's straying, his nurturing of his own (very altricial) children would increase the man's chances for his own grandchildren. There is a measure of intuition which infers that, for someone to nurture other people and to give them treasure, it is much more palatable if those people were liked by the dispenser. Any other combination of a giver-receiver relationship seems a non-viable competitor.

Conclusion

As the Australopithecines evolved into *Homo* a great number of features and feedback loops were undoubtedly operating, and perhaps operating simultaneously. Brain size was increasing, ovulation was being hidden, continuous sexual receptivity on the part of females was being instituted, the foundation for grammatical language was being laid, female mammary glands were increasing, body hair was decreasing, childhood was being lengthened, the pervasiveness of cultural traditions was being extended, *inter alia*.[11]

This article focuses only on four such features and their subsequent inter-dependent feedback loops: (i) human susceptibility to sexually transmitted diseases; (ii) pair-bonding or facultative monogamy; (iii) a reduced sexual dimorphism; and (iv) preferential paternal provisioning through a man-to-child affiliative bond. Selection of males, by females, for their psycho-behavioral traits of reliable provisioning, rather than for dominance displays based on physical attributes, would have reduced for males the reproductive advantage via their possession of an enhanced size or other physical/behavioral facets of dominance. Selection for psycho-behavioral traits which form the basis for pair-bonding, rather than multiple sexual partners, would have reduced the risks to fertility as a consequence of STDs. Selection by the female for those psycho-behavioral traits in men, which allowed for a man-to-child affiliative bond to emerge and to solidify, increased the viability of their children. Whether these four features were more of a cause or more of an effect in *Homo*'s separation

[11] For reviews of data/theory on the evolution of human behavior, see Chagnon and Irons (1979), Crawford, Smith, and Krebs (1987), Cronk, Chagnon, and Irons (2000), Dunbar, Knight and Power (1999), and Lockard (1980). For theories and discussions on bio-cultural feedback loops across generations, see Barkow (1980, 1989), Durham (1979, 1982, 1991), Boyd and Richerson (1982, 1983, 1985), and Lumsden and Wilson (1982, 1985); cf. Brown's (1991) presentation of human universals.

from chimpanzees-gorillas-Australopithecines is well beyond the scope of this analysis. Similarly, the integration of the thick and complex overlay of cultural forms and myth systems onto these behavioral tendencies is also beyond the focus of this analysis. However, that these four features are deeply embedded in the core of the human condition and are diagnostic of what it means to be human does not appear to be problematic.

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Appendix A. The spread of females who favor reciprocity (sex for food), over generations, versus reciprocity-neutral females, over generations, in a community after a STD has been introduced (from Immerman and Mackey 1999a).

	Percent of the band's daughters by type of female		
Generation Number	Females who favor reciprocity	Females who are reciprocity neutral	Total % females
1	16.7%[a]	83.3%	100.0%
2	17.0%	83.0%	100.0%
3	17.3%	82.7%	100.0%
4	17.6%	82.4%	100.0%
5	18.0%	82.0%	100.0%
10	19.7%	80.3%	100.0%
20	23.6%	76.4%	100.0%
50	37.9%	62.1%	100.0%
100	65.7%	34.3%	100.0%
150	85.6.%	14.4%	100.0%
200	94.9%	5.1%	100.0%
250	98.3%	1.7%	100.0%

[a] Arbitrarily chosen at 1 in 6.

Appendix B. In the context of an STD infestation, the loss of descendants by (i) loss of potential children from ectopic pregnancies, (ii) loss from sterility via STDs, (iii) offspring wastage [k = .2] from infected mothers and (iv) pre-pubescent mortality [k = .1] across all reproductive strategies (adapted from Immerman and Mackey 1999a).

Analysis of descendants' life-chances	Dyad Type (n =120 conceptions per dyad)			
	Bonding-male and Bonding-female	Bonding-male and Promiscuous-female	Promiscuous male and Bonding-female	Promiscuous male and Promiscuous female
Loss from ectopic pregnancies	3.330	21.013	20.165	21.229
Loss from STD sterility	.000	25.946	24.343	26.313
Loss from offspring wastage (k = .2 of infected women)	.000	14.776	14.513	14.839
Loss from pre-pubescent mortality (k = .1 of remaining children)	11.667	5.867	6.098	5.762
Total lost descendants	14.997	67.602	65.119	68.143
Percentage of n	12.497%	56.335%	54.266%	56.786%
Number of generative offspring	105.003	52.398	54.881	51.857
Percent of all potential children who are generative offspring	87.503%	43.665%	45.734%	43.214%
Mean number of daughters	2.562	1.279	1.339	1.265