

Evidence of Human Infertility

Documentary material for the webpage *Human origins: Are we hybrids?*

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If humans are of hybrid origin, particularly if they are derived from a cross between two rather distinct and genetically incompatible parents, then we might expect there to be lingering signs of sterility even in modern populations. While later-generation hybrids do tend to recover fertility under the influence of natural selection, their gametes can show traces of their origins for many generations. And in fact, if we examine data relating to human fertility, we find that it has been well known for decades that human sperm is abnormal (in comparison with that of the typical mammal).^a Sperm abnormalities are characteristic of hybrids (McCarthy, 2006, p. 18). Human spermatozoa are not of one uniform type as in the vast majority of all other types of animals. It is also interesting to note the more recent (1975) discovery that the gorilla shares this unusual characteristic with human beings (see Appendix).¹ Thus, Bedford *et al.*² say that "it has long been appreciated that from a morphological standpoint [*i.e.*, from the standpoint of their shape], the population of spermatozoa ejaculated by normal fertile

a. Reports of abnormal spermatogenesis in humans go back at least as far as von Ebner (1902) (165.2), who noticed that the developmental stages of the sperm cells are less regularly arranged in the seminiferous tubules of human beings than in those of other mammals.

men compares poorly with that of fertile males in domestic and laboratory species." Seuáñez *et al.*³ (1977) make an even stronger statement: "Human semen has long been known to differ from that of any other mammal [excepting the gorilla] in the high proportion of abnormal spermatozoa that it contains."⁴ (See Figure) These researchers found that in humans, on average, 18.4 percent of the spermatozoa are abnormal in shape; but in the common chimpanzee, just 0.2 percent (a 92-fold difference).⁵ In the pygmy chimpanzee, they observed no abnormally shaped spermatozoa at all.⁶ In a more extensive survey, Bedford⁷ examined spermatozoa from a wide variety of primates, and concluded that variability was the salient feature distinguishing human sperm from that of other members of Order Primates (his study did not include gorillas and was published prior to the discovery that gorillas share this trait with humans). He emphasizes that, although the spermatozoa

of other primates that we have studied were essentially homogeneous, human ejaculates are characterized by a heterogeneity, some features of which are visible in the light microscope. This heterogeneity is even more striking when viewed at the higher magnifications obtained with the electron microscope.^a In addition to marked variation in the shape of the nucleus,^b rather large vacuoles^c occur commonly within the nuclear chromatin.^d In contrast to the uniform nuclei of monkey spermatozoa, the nuclear chromatin of the human spermatozoon also presents a considerable variation from sperm to sperm ... These characteristics, as well as other pathological features involving the formation of the acrosome, the presence of residual cytoplasm of the spermatid^e at the neck, distortions in the arrangement of the mitochondria and variation in their number, *all testify to the disturbingly common occurrence of abnormally formed spermatozoa in*

a. Clermont (104.7,35) notes that microscopic examination of the lining of the seminiferous tubules in humans "suggests a haphazard arrangement of its germ cell components, which is not easily systematized. This is in sharp contrast with what can be observed in most mammalian species."

b. *nucleus*: the part of a cell that contains the chromosomes..

c. *vacuole*: an abnormal cavity within the substance of the germ cell. Guyer (223.45,41-45) found that vacuoles characterize the spermatozoa of hybrid pigeons.

d. *chromatin*: the chemical complex of DNA and protein of which chromosomes are composed.

e. *spermatid*: immature male reproductive cell.

the ejaculates of men of supposedly 'normal' fertility. [italics added]

There have been numerous claims of a correlation between the proportion of deformed sperm present in an individual's semen and the rate of spontaneous abortion in females fertilized with that semen.⁸ Even normal-looking human spermatozoa can be genetically deficient. In 1973, Bedford *et al.* reviewed the literature on this topic and analyzed ejaculates obtained from 44 human subjects (students, 22 to 30 years old):

It is clear that the ejaculates of men in this [American] society contain many spermatozoa which present a 'normal' morphology, but show marked differences in the structural character of the nuclear chromatin. Thus, the presence of an oval uniform head (type 1 — oval (normal) of Macleod, 1964,⁹ 1970¹⁰) cannot necessarily be presumed to indicate a normally formed nucleus. Furthermore, this variation in the structural quality of the human sperm nucleus *stands in very real contrast to the homogeneity observed in samples of mature spermatozoa from monkeys and rabbits.*¹¹ [italics added]

So humans produce a second type of spermatozoa: normal in overall shape, but with an abnormal nucleus. But the problems don't stop here. These authors go on to say that

even a normally shaped human sperm nucleus may often be abnormal at the molecular or ultrastructural level. Moreover, genetic paternal defects are often expressed by early death in the embryo in the pre- or peri-implantation period,¹² an event that would not be detected in the course of normal clinical observation ... These considerations, the high incidence of spontaneous abortion in this society (15 to 20% of all pregnancies¹³) and the possibility of a greater fetal loss at an earlier stage of human development^{14,a} suggest that the subject of sperm-related embryonic pathology in man is worthy of further basic and clinical study.¹⁵

So human sperm doesn't just look bad. It *is* bad. Spermatic abnormalities result in inviable offspring. Such findings effectively exclude the possibility

a. This compares with a rate of only about 4% in macaque monkeys (273.1; 430.43).

that the abnormal spermatozoa of human males arose through natural selection. Natural selection is for advantageous characteristics, ones that aid in reproduction, not hamper it. How could deformed, dysfunctional spermatozoa be advantageous?^b

Before it was known that gorilla semen also contained a large percentage of abnormal spermatozoa, the observation that human spermatozoa are highly variable in form was often explained as an adverse consequence of insulating the testicles with clothing. But when Martin *et al.*¹⁶ found similar abnormalities in gorilla semen, this explanation lost plausibility (i.e., gorillas don't wear pants). Moreover, recent studies have directly demonstrated that human semen abnormalities are unaffected by clothing and other lifestyle factors affecting scrotal temperature.¹⁷

It's hard to account for *Homo*'s inconsistent sperm production, without assuming that humans had a recent hybrid origin. The data otherwise seem entirely anomalous and paradoxical. The observed infertility of human beings was obviously puzzling to R. V. Short:

To agriculturalists accustomed to conception rates in cattle following a

b. Semen quality can also be measured in terms of chromosome counts. Whereas gorillas and humans have an incidence of slightly more than 1% diploid sperm, the chimpanzee has none (334.58,152). A diploid germ cell contains twice as many chromosomes as it should. If a diploid spermatozoon fertilizes a normal egg, the resulting offspring will be a triploid individual having three chromosomes of each type rather than the normal two. Human triploids abort early in the course of development. Even if only a single chromosome is triploid (trisomy), the consequences are grave (176.3). And yet, "Trisomy is the most commonly identified chromosome abnormality in man, occurring in approximately 5% of all clinically recognizable pregnancies. Almost all trisomies spontaneously abort, and by the time of birth only two types — namely trisomy 21 [Down syndrome] and trisomies involving the sex chromosomes — occur in appreciable frequency" (Hassold *et al.*, (231.81,253). In all such cases, an extra chromosome is present in the spermatozoon or in the egg. A five-percent rate for trisomic conceptions therefore indicates that about 2.5 percent of all human gametes contain an extra chromosome, which means (the rules of meiosis) that an additional 2.5 percent of them lack a chromosome, which brings the total proportion of aneuploid gametes to five percent. Of course, the rate of trisomy observed in "clinically recognizable pregnancies" would underestimate the frequency of aneuploid gametes (if a gamete lacked one or more large chromosomes, development might terminate soon after fertilization and pass unrecognized). Recent studies have avoided this problem by directly examining the chromosomal content of individual spermatozoa. Such studies indicate that at least *ten percent* of the gametes produced by a typical human male are aneuploid — a rate far in excess of that in laboratory animals (334.59). Some investigators suggest that rates of aneuploidy are even higher. Boué *et al.* estimate that one human conception in every two has an incorrect number of chromosomes (74.89,15a). Variation in gametic chromosome counts is characteristic of interspecific hybrids (57.6; 89.5; 183.3; 206.2; 208.9; 223.4; 291.1; 328.3; 334.65; 536.8; 588.6; 602.83).

single artificial insemination of 75%, the low fertility of our own species must come as something of a surprise, and requires an explanation. Normal human semen is notorious for showing an extremely high proportion of morphologically abnormal spermatozoa, often in excess of 40%, whereas the other primates (except the gorilla) have remarkably uniform spermatozoa. This suggests that a high proportion of human spermatozoa may be genetically defective, and would produce an abnormal embryo if they were capable of fertilizing the egg. There is circumstantial evidence to suggest that we do suffer from an extremely high incidence of early embryonic mortality. Thus, Hertig¹⁸(1975) examined the uteri of 210 fertile, married women who were having hysterectomies for unspecified reasons; he recovered 34 embryos aged 1-17 days, of which only 21 were judged to be normal, giving an embryonic mortality rate of 38% by the time of the first missed menstrual period.^a French and Bierman¹⁹ (1962) were able to follow 3,084 presumed pregnancies on the Hawaiian island of Kauai, from the time of the first missed menstrual period, and they showed that 23.7% of them failed to result in a live birth. Chromosomal studies of spontaneous abortions show that the abnormality rate is highest for early abortions, reaching 50% at 8 weeks gestational age, and declining to less than 5% by the end of the 20th week (Alberman & Creasy, 1975²⁰). One is inevitably forced to the conclusion that we are an extremely infertile species, and have been at least for the last two centuries.^{21,b}

a. Post-implantation death is often seen in hybrid embryos as, for example, in crossing goats with sheep (328.61,698).

b. According to Behrman and Patton (67.1,1), one married couple in five is unable to conceive.

Appendix: Infertility in the Gorilla

It is now well established that gorillas suffer from infertility far more often than is the case in other mammals. Thus, Martin *et al.* found that in the gorilla "Spermatozoa are markedly pleomorphic [i.e., variable in form] within any individual semen sample ... Other pongid species, and indeed other anthropoids examined are remarkably uniform in size and appearance."^{22,a} Seuánez *et al.* found that 16 percent of the spermatozoa in gorilla semen are abnormal in shape (this was in comparison with 18.3 percent in humans).^{23,b} This study involved only two gorillas, but Gould and Kling²⁴(1982) examined semen from a much larger sample and found that four of the 15 gorillas examined produced no sperm at all.^c In the remaining 11 individuals, an even higher proportion of abnormal sperm was evident than that reported by Seuánez and his colleagues (the exact percentages of abnormal spermatozoa reported were: 60, 40, 45, 60, 60, 40, 51, 90, 85, 80, and 30). A wild-caught gorilla examined by Platz *et al.*²⁵ showed 92.5 percent abnormal sperm. Many of these spermatozoa had abnormally shaped nuclei. Raphael *et al.* (1989) evaluated four male gorillas and found, in all four cases, that semen volumes were low, that 85 to 95 percent of the spermatozoa were abnormal, and that few or none of the spermatozoa actually present were motile.²⁶

If anything, gorillas are even less fertile than human beings. They commonly suffer from a condition known as *testicular atrophy* (or *hypoplasia*), which is seen in many

a. These authors measured dimensions (head length, head width, midpiece length, principal piece length, and total length) of spermatozoa in a variety of primates. Their results indicate that, when compared with other primates, the spermatozoa of humans and gorillas are about ten times more variable in form (334.5, Table 1).

b. The two samples examined by these authors were both taken from individuals that had fathered offspring. The semen examined may therefore have been of higher quality than that of the typical gorilla. Nevertheless, they found that 27 percent of the human, and 29 percent of the gorilla, spermatozoa were abnormal in some way or another (if not in shape, then at least with respect to other features, such as staining, cytoplasmic features, maturity, etc.).

c. McKenny *et al.*(1944) reported that smears from the testes of a 15-year-old captive gorilla showed no spermatozoa (328.7). Brandt (1972) records another case in which a gorilla failed to produce spermatozoa (81.6).

interspecific hybrids. "Indeed, testicular hypoplasia^d and infertility is found throughout the literature on adult gorillas. Most often an aetiological [i.e., causative] factor for this infertility remains unknown" (Benirschke and Adams²⁷). This condition has been consistently observed, not just in captive specimens, but in free-living individuals as well. Although various workers who have reported testicular atrophy in gorillas have deemed it an abnormal or pathological state, it seems actually to be *characteristic* of gorillas because all reports describing gorilla testes in any detail concur in the observation of atrophy. As early as 1937, Koch²⁸ observed that the seminiferous tubules of a gorilla, Bobby, then residing at the Berlin Zoo, were atrophied and sclerotic.^a Wislocki²⁹ (1942) compared the testes of a wild-shot gorilla with those of a chimpanzee, stating they were

from a mountain gorilla conservatively estimated to have weighed 450 lbs and to have been fully adult. The testes were exceedingly small, the pair weighing 36 grams ... In the chimpanzee the seminiferous tubules are large and densely filled with maturing stages of the germinal cells, including spermatids and spermatozoa. *In the gorilla, on the contrary, the tubules are few and quite small and are widely separated from one another by fields of stroma and interstitial cells.* Nevertheless the tubules are active and contain both spermatids and spermatozoa. The presence of these cells indicates that the gorilla's testes in question are functionally active. *The testes may not have reached their maximum growth or full activity, however, for in that event one would have anticipated more abundant spermatogenesis than is actually present.*" [italics added]

Another case of testicular atrophy, this time in a captive gorilla, was reported four years later by Clarke.³⁰ In 1954, Steiner dissected the corpse of a lowland gorilla. Although the animal was well-nourished and physically mature, Steiner found that "the reproductive tract was not only underdeveloped, but abnormal."³¹ He observed that the "testes were highly abnormal, showing a severe atrophy and sclerosis of the seminiferous tubules" and that the seminal vesicles contained no spermatozoa.^{32,b} In a separate paper on the same individual, Steiner *et al.* (1955) found that "Microscopically, the testes showed a virtually complete atrophy and sclerosis of the seminiferous tubules and a great relative, if not actual, increase in the number of interstitial cells of Leydig in all areas; an unquestionable increase, represented by large solid sheets, was present in many places."^{33,a}

d. *hypoplasia*: a condition of arrested development in which an organ or part remains below the normal size or in an immature state.

a. *sclerosis*: hardening of tissue, especially from overgrowth of fibrous tissue or increase in the interstitial tissue that separates the seminiferous tubules.

b. In general, he observes, "Small external genitalia appear to be a constant finding in the gorilla, both wild and captive."

a. These authors state (538.4,5) that "no male [gorilla] in captivity is known to have reached sexual maturity, although the other three species of anthropoid apes have reproduced under similar living conditions." Of course, some captive gorillas have exhibited an ability to breed since this statement was made (1955). But even in 1981, Dixson (140.1) noted that "only three of the thirteen male gorillas in British collections are of proven breeding ability." The infertility of captive gorillas is such a problem

In 1961 Inoue *et al.* examined a wild-caught 40-year-old male and stated that "both the penis and the testicles are remarkably small — the latter are sparrow's-egg-sized."³⁴ In the same year Hosakawa commented on the abundance of interstitial tissue present in the testicles of a 40-year-old male caught in the wild the year before.³⁵ The next year Hall-Craggs³⁶ examined the testicles of two more wild gorillas, an adolescent and an adult. He found that the testicles were very small, that they contained large masses of interstitial cells separating the seminiferous tubules, and that the epididymis contained no spermatozoa. He further states that "The large proportion of testicular interstitial tissue found in captive animals is present in the two free gorillas, and is therefore *a normal finding*" (italics added). An additional case of testicular atrophy was reported by Antonius *et al.*³⁷ These authors state that "Each of the testes were about 3.8 cm in length^b ... Microscopic examination revealed that one testis was slightly more atrophic than the other. Interstitial cells occupied about half the bulk of both testes ... Although some tubules contained a few spermatogonia [i.e., immature germ cells], in most there was no spermatogenesis. Those free of spermatogonia were lined with Sertoli's cells alone." Recently, Munson and Montali (1990) reported yet another case of testicular "degeneration" in a gorilla.³⁸ Moreover, in referring to available records at Washington's National Zoological Park, these authors stated that in both males and females "infertility was a major problem in all gorillas."³⁹ Böer (1983) also reported "severe testicular atrophy" in a captive gorilla.⁴⁰ Foster and Rowley (1982) performed testicular biopsies on six wild-caught gorillas and found that spermatozoa were absent in all six.⁴¹ Dixson (1981) examined the testicles of three gorillas, two orangutans, and one chimpanzee. He found that

the testes from the orang-utans and chimpanzee were apparently normal whereas those from the gorillas exhibited varying degrees of atrophy. In 'Guy', a silverback which had lived for over thirty years at the London Zoo, the testes were extremely small, measuring 27 X 18 mm (right) and 28 X 19 mm (left). The corresponding weights including the atrophic epididymes were only 6.85 grammes and 4.4 grammes respectively. The entire genital tract of this male was removed, yet it was not possible to identify with certainty either the prostate or seminal vesicles. Sections of the testes exhibited a diffuse nodular appearance, the seminiferous tubules were totally degenerate and fibrosed and none of the stages of spermatogenesis could be identified. Fine structural observations, using the electron microscope, revealed that the Leydig cells, which normally produce testosterone, were

that even expensive technical procedures such as in-vitro fertilization and gamete intrafallopian transfer have been deemed worthwhile (291.4; 322.2), and captive populations are still not self-sustaining (186.6; 456.7).

b. These testicles were of the same size as those of another captive gorilla, "Samson," examined by Short (509.15,8).

non-functional. 'Jojo', a much younger male which had died at Chester Zoo, exhibited a similar testicular picture. In the third gorilla, 'Oban' from the Yerkes Regional Primate Research Center, the testes had a more normal appearance. The seminiferous tubules were small and surrounded by large amounts of interstitial tissue, but this is not an indication of abnormality, since both Wislocki and Hall-Craggs have described these features in material from wild-shot gorillas. However, in 'Oban' sloughing and degeneration of the germinal epithelium was apparent and mature spermatozoa could not be identified in either the seminiferous tubules or epididymis. The causes of testicular atrophy in captive gorillas are not known.^{42,a}

Descriptions of testicular atrophy in gorillas are peculiarly reminiscent of accounts describing the testicles of the common mule and of other sterile, or partially sterile, hybrids. In comparing the testes of the mule with those of the horse, Makino⁴³ found that in the mule "the seminiferous tubules of the testes show a remarkable decrease in diameter and contain a very small number of germ cells. In the majority of tubules, only one or two layers of such germ cells line their inner wall, and most of the cells found there are spermatogonia [i.e., germ-cell precursors]. The tubular lumina [i.e., the interior of the seminiferous tubules] are empty, or sometimes contain a mass of degenerating cell debris. In striking contrast to the shrinkage of the seminiferous tubules, the interstitial tissue shows a remarkable development between the widely separated tubules." Other researchers paint a similar picture of hybrid testes, in which seminiferous tubules are small, the interstitial tissue overdeveloped, and the germ cells degenerate.⁴⁴

a. Although Steiner *et al.* (538.4) suggested testicular atrophy may be due to malnutrition, Dixson and his colleagues (140.8) countered this supposition by noting that the most normal of the three sets of gorilla testicles they had examined belonged to "Oban," who was extremely malnourished after suffering from a gastrointestinal disorder for several years.

NOTES:

1. (334.5)
2. (66.7,24)
3. (507.2,345)
4. See also (65.3).
5. (507.2, Table 1). See also (66.7).
6. (507.2, Table 1)
7. (65.3,104-105). See also (65.1,448).
8. (53.8; 184.4; 256.6; 328.1; 447.6; 473.8)
9. (327.9)
10. (327.8)
11. (66.7,24-25)
12. (73.53; 73.56)
13. (602.75)
14. (233.5)
15. (66.7,27)
16. (334.5)
17. (303.4; 412.93; 588.83)
18. (233.45)
19. (188.6)
20. (21.5)
21. (509.1,12)
22. (334.5, Legend to Plate 3)
23. (507.2,345)
24. (206.9)
25. (432.7)
26. (452.45)
27. (67.4,144)
28. (271.6)
29. (602.65,286-287)
30. (104.2)
31. (537.3,146)
32. (537.3,156-157)
33. (538.4,7)
34. (249.3,33)
35. (242.5)
36. (230.2)
37. (29.1)
38. (393.3,102)
39. (393.3,102)
40. (73.8)
41. (187.6,122)
42. (140.1,140-141); See also (140.8).

43. (330.5,225)

44. (57.6; 64.2; 64.5; 223.45; 242.3; 252.4; 269.98,518; 318.6,500; 330.7; 444.1; 539.1; 539.2,892; 539.22; 540.9; 542.3,360; 596.3)