CHAPTER 3.

WHY DON'T MEN BREAST-FEED THEIR BABIES?

The Non-Evolution of Male Lactation.

Section 1.

Today, we men are expected to share in the care of our children. We have no excuse not to, because we are perfectly capable of doing for our kids virtually anything that our wives can do. And so, when my twin sons were born in 1987,I duly learned to change diapers, clean up vomit, and perform the other tasks that come with parenthood.

The one task that I felt excused from was nursing my infants. It was visibly a tiring task for my wife. Friends kidded me that I should get hormone injections and share the burden. Yet cruel biological facts seemingly confront those who would bring sexual equality into this last bastion of female privilege or male cop-out. It appears obvious that males lack the anatomical equipment, the priming experience of pregnancy, and the hormones necessary for lactation. Until 1994, not a single one of the world's 4,300 mammal species was suspected of male lactation under normal conditions. The nonexistence of male lactation may thus seem to be a solved problem requiring no further discussion, and it may seem doubly irrelevant to a book about how the unique aspects of human sexuality evolved. After all, the problem's solution seems to depend on facts of physiology rather than on evolutionary reasoning, and exclusively female lactation is apparently a universal mammalian phenomenon not at all unique to humans.

In reality, the subject of male lactation follows perfectly from our discussion of the battle of the sexes. It illustrates the failure of strictly physiological explanations and the importance of evolutionary reasoning for understanding human sexuality. Yes, it's true that no male mammal has ever become pregnant, and that the great majority of male mammals normally don't lactate. But one has to go further and ask why mammals evolved genes specifying that only females, not males, would develop the necessary anatomical equipment, the priming experience of pregnancy, and the necessary hormones. Both male and female pigeons secrete crop "milk" to nurse their squab; why not men as well as women? Among seahorses it's the male rather than the female that becomes pregnant; why is that not also true for humans?

As for the supposed necessity of pregnancy as a primary experience for lactation, many female mammals, including many (most?) women, can produce milk without first being primed by pregnancy. Many male mammals, including some men, undergo breast development and lactate when given the appropriate hormones. Under certain conditions, a considerable fraction of men experience breast development and milk production even without having been treated hormonally. Cases of spontaneous lactation have long been known in male domestic goats, and the first case of male lactation in a wild mammal species has been reported recently.

Thus, lactation lies within the physiological potential of men. As we shall see, lactation would make more evolutionary sense for modern men than for males of most other mammal species. But the fact remains that it's not part of our normal repertoire, nor is it known to fall within the normal repertoire of other mammal species except for that single case reported recently. Since natural selection evidently could have made men lactate, why didn't it? That turns out to be a major question that cannot be answered simply by pointing to the deficiencies of male equipment. Male lactation beautifully illustrates all the main themes in the evolution of sexuality: evolutionary conflicts between males and females, the importance of confidence in paternity or maternity, differences in reproductive investment between the sexes, and a species' commitment to its biological inheritance.

As the first step in exploring these themes, I have to overcome your resistance to even thinking about male lactation, a product of our unquestioned assumption that it's physiologically impossible. The genetic differences between males and females, including those that normally reserve lactation for females, turn out to be slight and labile. This chapter will convince you of the feasibility of male lactation and will then explore why that theoretical possibility normally languishes unrealized.

Our sex is ultimately laid down by our genes, which in humans are bundled together in each body cell in twenty-three pairs of microscopic packages called chromosomes. One member of each of our twenty-three pairs was acquired from our mother, and the other member from our father. The twenty-three human chromosome pairs can be numbered and distinguished from each other by consistent differences in appearance. In chromosome pairs 1 through 22, the two members of each pair appear identical when viewed through a microscope. Only in the case of chromosome pair 23, the so-called sex chromosomes, do the two representatives differ, and even that's true only in men, who have a big chromosome (termed an X chromosome) paired with a small one (a Y chromosome). Women instead have two paired X chromosomes.

What do the sex chromosomes do? Many X chromosome genes specify traits unrelated to sex, such as the ability to distinguish red and green colors. However, the Y chromosome contains genes specifying the development of testes. In the fifth week after fertilization human embryos of either sex develop a "bipotential" gonad that can become either a testis or an ovary. If a Y chromosome is present, that bet-hedging gonad begins to commit itself in the seventh week to becoming a testis, but if there's no Y chromosome, the gonad waits until the thirteenth week to develop as an ovary.

That may seem surprising: one might have expected the second X chromosome of girls to make ovaries, and the Y chromosome of boys to make testes. In fact, though, people abnormally endowed with one Y and two X chromosomes turn out most like males, whereas people endowed with three or just one X chromosome turn out most like females. Thus, the natural tendency of our bet-hedging primordial gonad is to develop as an ovary if nothing intervenes; something extra, a Y chromosome, is required to change it into a testis.

It's tempting to restate this simple fact in emotionally loaded terms. As the endocrinologist Alfred Jost put it, "Becoming a male is a prolonged, uneasy, and risky venture; it is a kind of struggle against inherent trends towards femaleness." Chauvinists might go further and hail becoming a man as heroic, and becoming a woman as the easy fallback position. Conversely, one might regard womanhood as the natural state of humanity, with men just a pathological aberration that regrettably must be tolerated as the price for making more women. I prefer merely to acknowledge that a Y chromosome switches gonad development from the ovarian path to the testicular path, and to draw no metaphysical conclusions.

But there's more to a man than testes alone. A penis and prostate gland are among the many other obvious necessities of manhood, just as women need more than ovaries (for instance, it helps to have a vagina). It turns out that the embryo is endowed with other bipotential structures besides the primordial gonad. Unlike the primordial gonad, though, these other bipolar structures have a potential that is not directly specified by the Y chromosome. Instead, secretions produced by the testes themselves are what channel these other structures toward developing into male organs, while lack of testicular secretions channels them toward making female organs.

For example, already in the eighth week of gestation the testes begin producing the steroid hormone testosterone, some of which gets converted into the closely related steroid dihydrotestosterone. These steroids (known as an-drogens) convert some all-purpose embryonic structures into the glans penis, penis shaft, and scrotum; the same structures would otherwise develop into the clitoris, labia minora, and labia majora. Embryos also start out bet-hedging with two sets of ducts, known as the Mullerian ducts and Wolffian ducts. In the absence of testes, the Wolffian ducts atrophy, while the Mullerian ducts grow into a female fetus's uterus, fallopian tubes, and interior vagina. With testes present, the opposite happens: androgens stimulate the Wolffian ducts to grow into a male fetus's seminal vesicles, vas deferens, and epididymis. At the same time, a testicular protein called Mullerian inhibiting hormone does what its name implies: it prevents the Mullerian ducts from developing into the internal female organs.

Since a Y chromosome specifies testes, and since the presence or absence of the testes' secretions specifies the remaining male or female structures, it might seem as if there's no way that a developing human could end up with ambiguous sexual anatomy. Instead, you might think that a Y chromosome should guarantee 100 percent male organs, and that lack of a Y chromosome should guarantee 100 percent female organs. In fact, a long series of biochemical steps is required to produce all those other structures besides ovaries or testes. Each step involves the synthesis of one molecular ingredient, termed an enzyme, specified by one gene. Any enzyme can be defective or absent if its underlying gene is altered by a mutation. Thus, an enzyme defect may result in a male pseudohermaphrodite, defined as someone possessing some female structures as well as testes. In a male pseudoher-maphrodite with an enzyme defect, there is normal development of the male structures dependent on enzymes that act at the steps of the metabolic pathway before the defective enzyme. However, male structures dependent on the defective enzyme itself or on subsequent biochemical steps fail to develop and are replaced either by their female equivalent or by nothing at all. For example, one type of pseudohermaphrodite looks like a normal woman. Indeed, "she" conforms to the male ideal of female pulchritude even more closely than does the average real woman, because "her" breasts are well developed and "her" legs are long and graceful. Hence cases have turned up repeatedly of beautiful women fashion models not realizing that they are actually men with a single mutant gene until genetically tested as adults.

Since this type of pseudohermaphrodite looks like a normal girl baby at birth and undergoes externally normal development and puberty, the problem isn't even likely to be recognized until the adolescent "girl" consults a doctor over failure to begin menstruating. At that point, the doctor discovers a simple reason for that failure: the patient has no uterus, fallopian tubes, or upper vagina. Instead, the vagina ends blindly after two inches. Further examination reveals testes that secrete normal testosterone, are programmed by a normal Y chromosome, and are abnormal only for being buried in the groin or labia. In other words, the beautiful model is an otherwise normal male who happens to have a genetically determined biochemical block in his ability to respond to testosterone. That block turns out to be in the cell receptor that would normally bind testosterone and dihydrotestos-terone, thereby enabling those androgens to trigger the further developmental steps of the normal male. Since the Y chromosome is normal, the testes themselves form normally and produce normal Mullerian inhibiting hormone, which acts as in any man to forestall development of the uterus and fallopian tubes. However, development of the usual male machinery to respond to testosterone is interrupted. Hence development of the remaining bipotential embryonic sex organs follows the female channel by default: female rather than male external genitalia, and atrophy of the Wolffian ducts and hence of potential male internal genitalia. In fact, since the testes and adrenal glands secrete small amounts of estrogen that would normally be overridden by androgen receptors, the complete lack of those receptors in functional form (they are present in small numbers in normal women) makes the male pseudohermaphrodite appear externally super-feminine.

Thus, the overall genetic difference between men and women is modest, despite the big consequences of that modest difference. A small number of genes on chromosome 23, acting in concert with genes on other chromosomes, ultimately determine all differences between men and women. The differences, of course, include not just those in the reproductive organs themselves but also all other post-adolescent sex-linked differences, such as the differences in beards, body hair, pitch of voice, and breast development.

The actual effects of testosterone and its chemical derivatives vary with age, organ, and species. Animal species differ greatly in how the sexes differ, and not only in mammary gland development. Even among higher anthropoids— humans and our closest relatives, the apes—there are familiar differences in sexual distinctiveness. We know from zoos and photos that adult male and female gorillas differ obviously at a long distance by the male's much greater size (his weight is double the female's), different shape of head, and silver-haired back. Men also differ, though much less obviously, from women in being slightly heavier (by 20 percent on the average), more muscular, and bearded. Even the degree of that difference varies among human populations: for example, the difference is less marked among Southeast Asians and Native Americans, since men of those populations have on the average much less body hair and beard development than in Europe and Southwest Asia. But males and females of some gibbon species look so similar that you couldn't distinguish them unless they permitted you to examine their genitals.

In particular, both sexes of placental mammals have mammary glands. While the glands are less well developed and nonfunctional in males of most mammal species, that degree of male underdevelopment varies among species. At the one extreme, in male mice and rats, the mammary tissue never forms ducts or a nipple and remains invisible from the outside. At the opposite extreme, in dogs and primates (including humans) the gland does form ducts and a nipple in both males and females and scarcely differs between the sexes before puberty.