ALTERNATIVE SEXUALITIES AND EVOLUTION

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ABSTRACT: Ward showed that stressing pregnant rats led to sons who showed decreased mounting of females and increased lordotic behaviour. There was no effect on female offspring. Herein we go beyond her work and suggest an evolutionary hypothesis for the spread of homosexuality in human males in populations. A new mutation passes down to an ancestral pregnant human female and thereby to her male foetus. This mutation, combined with stress due to crowding, leads to homosexuality in her son. If a male foetus has the gene but his mother experiences no stress during gestation, this son is heterosexual. If the mother is stressed but her male foetus does not have the gene, her son is once again heterosexual. That is, both genes and stress lead to homosexual sons. This mechanism leads to increased survival in grandchildren in times of starvation, since their homosexual uncle has no or few children to compete with them, and does not introduce female partners to the group. Families without the gene mostly starve to death. Thus the gene spreads in the population. In a variant of the model, starvation syndrome in a pregnant woman interacts with the filial gene to cause homosexuality in her son. The model leads to four predictions.

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The problem of eusociality in the Hymenoptera partially involves explication of the evolution of a nonreproductive female caste (Wilson, 1971; Holldobler and Wilson, 1990; Ferguson, 1993). Does a similar theoretical problem exist in our own species? Certain men and certain women refrain (or mostly refrain) from reproduction because they function sexually only (or almost only) with their own sex (homosexuality). Let us postulate that this trait is partially genetic. Assuming an initial mutation millions of years ago, how could this trait spread in populations? The problem is profoundly simple and clear. The purpose of this paper is to propose a sociobiological model solving this problem and thereby to show also that a gene may interact with prenatal stress to generate male homosexuality in humanity. Previous writers have considered genes and stress to be alternative hypotheses rather than possible co-determinants.

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HUMAN GENETIC STUDIES

Bailey and Pillard (1991) conducted the largest twin study of male sexual orientation to date. They studied monozygotic twins, dizygotic twins, adoptive brothers in families with an occurrence of homosexuality, and nontwin brothers of homosexuals. The concordance for homosexuality in monozygotic twins was 52%.
Dizygotic twins had a concordance of 22%. Bailey and Pillard were not surprised by a concordance with adoptive brothers of 11%, but they expected a higher concordance than 9.2% for nontwin brothers. They felt that this figure was lower than predicted by simple genetic hypotheses and by other studies. These concordances will be analyzed according to our views in the Discussion.

Hamer et al. (1993) used pedigree and DNA linkage analyses to investigate genetic influence on male sexual orientation in 114 families of homosexual men. Transmission was apparently sex-linked. Mothers pass the trait to their sons by one of their X-chromosomes. These authors were able to find the approximate location of the gene on the X-chromosome. The authors state that they have shown that "at least one subtype" of male homosexuality is partially genetic. Thus there may be other subtypes of homosexuality that are not influenced by this particular gene. They also state that the frequency of homosexuals in populations is about 2%.

These studies show that there is some genetic basis for human male homosexuality which makes possible evolutionary analyses.

EARLIER PROPOSED SOCIOBIOLOGICAL HYPOTHESES

Ruse (1988) summarizes the three main present sociobiological answers to the question of human male homosexuality.

First, G. E. Hutchinson (1959) proposed the balanced polymorphism hypothesis, which is also discussed in more detail in Wilson (1975). The most famous case of a balanced polymorphism in genetics is that of sickle-cell anaemia (see Dobzhansky et al., 1977, for a good discussion). Similarly, although human homosexuals have few or no children (and assuming that they are homozygotes), it is postulated that the heterozygotes are highly fit. Neither Hutchinson, Wilson, nor Ruse have proposed in what way the heterozygotes might be fitter. Sex-linkage as discovered by Hamer et al. (1993) excludes such an hypothesis.

The second proposal summarized by Ruse (1988) is kin selection (Hamilton, 1964; Wilson, 1975, 1978). Under certain conditions an individual may forego its own reproduction in order to help raise its own siblings. Siblings in diploid animals share one-half of their genes on average with each other. By helping siblings an individual may thus pass on a greater or an equal number of its genes to future generations as it would have had it produced its own children. There may be circumstances where the individual may judge itself a poor prospective parent and so instead turn to kin, but the precise circumstances which might be involved have not been specified. This explanation is the kin selection hypothesis of homosexuality. Part of the hypothesis states that homosexuals may attain successful positions in society as a means of helping relatives.

On this latter score, Ruse (1988) refers to Weinrich (1967), who summarized some of the anthropological literature. Homosexuals in other societies may for example be described as "decision-makers", "rich", "leaders", and sometimes even "chiefs". In Ruse's opinion, this elevated status is not really true of Western societies, so that homosexuals may have been selected for in the past but today are relicts from that past time.

Finally, Ruse (1988) states his own parental manipulation hypothesis. A parent may cause a child not to reproduce itself but to turn to sibling-raising so that the parent will thereby raise more offspring with its help. The main difference between kin selection and parental manipulation is that the son who displays kin selection will favour its own genes while in parental manipulation the son will favour its parents' genes to its own detriment.

ANIMAL STUDIES ABOUT STRESS

Ward (1972) stressed pregnant rats by restraining them in Plexiglas tubes with exposure to very bright light three times daily for forty-five minutes each time. The stressors were exerted from day 14 to day 21 of gestation. The prenatally stressed male offspring of these females copulated and ejaculated less with females in adulthood. A postnatally stressed control group of male offspring showed no such effect. The subjects were castrated and then injected with estrogen and progesterone. The prenatally stressed male rats showed three times as many lordotic (sexually receptive) responses when approached by normal adult males than did controls. Such prenatal stress had no effect whatever on females.

Ward in the same paper discusses the effects of stress on animals including effects on the adrenal glands. ACTH suppresses testosterone production by the testes but increases adrenal activity. (The "prenatal stress syndrome" is the demasculinization and femininization of behaviour in male offspring as a result of prenatal stress.) She sees this syndrome (hereafter called the Ward Effect) as a population control mechanism, which is of ecological interest. Her discussion ignores genes and selection entirely. It is a proximate and not ultimate treatment.

Ward (1974) summarized her earlier research. Then she proposed her rat research as a simple model that may help our understanding of human homosexuality. This discussion is not cited in later papers. Ward was largely silent about the issue of human homosexuality until she briefly discussed it in Ward (1984). Her views as expressed therein will be summarized at the end of this paper. In our opinion, the Ward Effect may indeed explicate homosexuality in humans, if it can be demonstrated that prenatal stress in humans has a similar effect as in rats.

Ward (1977) repeated the 1972 experiment, but after castration injected testosterone rather than estrogen and progesterone. The results were very similar to the previous 1972 study.

Ward and Weisz (1980) stressed pregnant rats and then measured plasma testosterone in both mothers and foeti. There was declining testosterone on days 18 and 19 after conception. In controls, testosterone was highest on these days. Thus in stressed animals

there was less testosterone at a critical time in development. This phenomenon may underly the effects of prenatal stress on the brains of foetal males. Ward was not very interested in brain effects. However, Ward (1984) mentioned that the aromatization of androgen to estrogen is needed for the normal masculinization of the brain. She notes lower levels of aromatization on days 18-20 of gestation. The relationship to behavioural development was not made clear.

Rhees and Fleming (1981) had three experimental groups. All treatments--Wardian stress, injection of ACTH, starvation--resulted in the Ward Effect. For our purposes, the starvation treatment (50% of the food given to controls) is salient.

A MODEL WITH TWO VARIANTS

A physiological definition of "stress" is given by Selye (1976). A Ward Effect in men would be somewhat different than in rats. Rats show only female behaviour while human homosexuals display both passive and active behaviour. Thus we define the Ward Effect in men as the presence of adult homosexuality as a result of the interaction between prenatal stress and a foetal gene in the development of the central nervous system. We are now ready to put forth our sociobiological hypothesis concerning humans:
An interplay between prenatal stress caused by crowding in the external environment or by starvation and by a gene in male foeti leads to adult homosexuality. This condition in the past allowed less competition for resources among these males' nieces and nephews (some of whom carried the same gene) so that more offspring survived than if these homosexuals had had their own children as well. Thus the gene spreads in populations. Now we elaborate.

Consider a crowded ancestral human population consisting of kin groups within a spatial structure. Normally there might be a very minor tendency in females in the direction of the Ward Effect through pleiotropy or other mechanism. When a female possesses a mutation on one of her X-chromosomes which is passed on to her daughter through her ovum, this mutation in the adult daughter is passed on to half of her sons. When prenatal stress arises, the foetal gene interacts with the stress to generate the Ward Effect. Her son is homosexual. This mutation is not expressed when there is no emotional and physical stress. It only expresses itself during periods of stress for example due to crowding. The son does not reproduce. His sisters do reproduce and half of them pass down the mutation to half of their children of either sex. By this latter generation, hunger will be a factor if the crowding causes a shortage of resources including food. These newest offspring will survive the shortage, since they do not have their uncle's children with which to compete.

Hunger may also have been caused by long-term famines lasting twenty or thirty years. We postulate the existence of such famines in the past and predict that research will confirm the existence of such past famines in for example Africa, Europe, and Asia.

Not only nieces and nephews would be saved. Assuming that the average food requirements for survival of individuals is just above what on average is available to these individuals in fact, a sacrifice on the homosexual's part of two offsring would feed also several more

distant relatives and perhaps additional people who are tied to his kin group by bonds of reciprocal altruism. A homosexual who sacrificed two offpring would have to save four nieces and nephews, but the above argument shows that in some circumstances at least he would save many more people, relatives and otherwise. Further, we can expect a bearer of a homosexual gene to compete for nutritional resources for his family, thus assuring that the resources saved by his non-reproduction would benefit those closest to home.

But in reproductive lines where the mutation is not passed down, there will be more children competing within the family for food. Most offspring would starve. Thus the mutation is preferentially spread through time and increases in frequency in the population. Although the mother's emotional stress may initially have been caused by crowding, we postulate that any severe emotional stress during pregnancy will today interact with the male foetal gene to produce homosexuality.

A variant takes place in an environment of extreme food shortage. It cannot be said that the people involved suffered from Selye's stress syndrome, because Selye (1976) does not mention either hunger or starvation. For starvation, we propose the terms "dystrophic syndrome" or simply "starvation syndrome". Once again there is the same mutation which promotes the Ward Effect, but which operates under starvation conditions. (See Rhees and Fleming, 1981.)

Women carrying the mutation have half of their sons become homosexual and so there is less competition for food among the grandchildren. Thus more survive than in other families where competition is greater. The mutation is sex-linked (following Hamer et al., 1993).

R. B. Lee (verbal communication) has told the author that he observed only one clearly homosexual man amongst a total of three thousand !Kung San he studied. Lee (1979) reported that North Ghana agriculturalists lost three times as much weight in lean periods each year as did the hunter-gatherer San. The frequency of homosexuality in the San may be close to the mutation rate. Since hunger may be greater in agriculturalists if the above comparison has generality, it may be that greater hunger in farmers would lead to higher frequencies of homosexuality in such people than in hunter-gatherer societies. (For the model to work, agriculturalists too must have undergone periodic lengthy famines.)

The author predicts that agricultural societies should have more homosexuals per capita than hunter-gatherers do, assuming lengthy famines and their effects on human populations.

DISCUSSION

In an early paper (Ferguson, 1976), we argued that evolutionary theory can make predictions. Indeed, these models lead to predictions. It is hard to test evolutionary models directly. Nevertheless, if a gene has been selected to interact with prenatal stress thus generating homosexuality in the past, this very mechanism should still exist today. This latter assertion is testable. Remember that both stress or starvation and the mutation must be present together if homosexuality is to be generated.

1. During wars, populations may exist in conditions of reduced food supply. We predict that more homosexuals are born at these times, for example in Holland during the Second World War. Similarly, severe droughts in Northern Africa in recent years would lead us to expect an expanding homosexual population in these areas.

2. Anorexia and bulimia are medical conditions in which food intake is decreased. We predict that anorexic and bulimic women whose menses have not stopped have more homosexual sons than other women.

3. The above examples deal with starvation syndrome. Now we turn to emotional (and consequent physical) stress as in the crowding example. We predict that women emotionally stressed during pregnancy have more homosexual sons than do other women, granting also that their sons have inherited the homosexual gene from them.

Dörner et al. (1980) have already provided some confirmation. They found that significantly more male homosexuals were born in Germany during the war years than in a similar span of time before the war and in a similar span of tlme after the war. Naturally the war was a highly stressful time for German pregnant women as elsewhere. They lost husbands and sons, were raped, bombed and so on. Dörner et al. (1983) extended and confirmed their earlier findings. Ellis et al. (1988) confirmed Dörner and his colleagues. They found that stress in the second trimester of pregnancy correlated with later homosexuality in sons. Ellis and co-workers also discovered that more stress during the year before pregnancy also so correlated, which is a unique result. These studies are a confirmation of our hypothesis.

Bailey et al. (1991) critiqued and tested the stress hypothesis. Among their criticisms, they state that human adrenal response is less extreme than in nonhuman animals. They also declare that the Ward Effect is not a good model. They write that subject rats should show masculine sexual behaviour with other males, not only lordosis. This points out a difference in the situation in rats and humans. The Ward Effect in rats involves only femininization and demasculinization, but in men there is dominant male behavior directed towards other men as well. This difference suggests that the Ward Effect in the two species is analogous and not homologous in origin. However, we stand by our hypothesis, and continue to use the term "Ward Effect" in humans even if the two cases turn out to be only analogous.

In the empirical part of their paper, Bailey et al. found no more stress in the mothers of homosexuals than in the mothers of nonhomosexuals. But they admit a flaw in their approach. The greatest stressor in the women studied was moving house, which does not compare with the intensity of stress in Dörner's studies.

Ward (1984) changed her (1974) view of her Ward Effect as a model for human homosexuality. She considered Dörner's application of her findings to humanity to be premature. She pointed out that rodents and primates differ in the mechanisms of sexual differentiation. Ward concluded that her rodent work may have no clinical significance. Dörner however read Ward (1977) and immediately formed the hypothesis that stress in mothers causes homosexuality in man. This was effectively confirmed as indicated above and the very differences between the mechanisms in man and in rodents greatly strengthen the credibility of his findings.

4. LeVay (1991) found that a nucleus in the anterior hypothalamus is larger in heterosexual men than in heterosexual women and larger in heterosexual men than in homosexual men. In both cases, the male heterosexual nucleus was twice the size of the other. This result is controversial. However, Kerchner and Ward (1992) found that a nucleus in the medial preoptic area of the rat hypothalamus in prenatally stressed male animals was smaller than in nonstressed rats. Conceivably the former rats were homosexual. LeVay wrote in his paper that this region of the rat brain is comparable to the nucleus he studied in people. We predict that more neuroanatomical correlates of homosexuality will be found and that LeVay's work will be confirmed, and a partial genetic basis found for his results.

It is possible to take another look at the results of Bailey and Pillard (1991). The concordance of 52% for monozygotic twins indicates a great deal of environmental influence as well as genetic. This concordance is the highest of all concordances in the study because the twins are genetically identical and according to our model share the same intrauterine environment with very similar prenatal stress. The concordance for dizygotic twins (22%) is lower because on average the twins will share only half their genes, so that half of the time only one twin will have the gay gene, although they will share the same intrauterine environment. Thus this concordance (22%) should be about half that of identical twins, which is approximately so. Bailey and Pillard are surprised that with nontwin brothers the concordance is only 9.2%. Yet these brothers will have the same gay gene as the proband only half of the time, and further they will not share the same intrauterine environment. The proband would have experienced prenatal stress, yet it is highly likely that his nontwin brother did not. Finally, Bailey and Pillard consider the concordance with adoptive brothers of 11% to be expected. We find this concordance to be extremely high. True, mothers who give up their babies for adoption might often experience high levels of stress while pregnant. But also needed to produce the concordance is a frequency of gay genes in the population of at least 11%. Remember Hamer et al.'s (1993) estimated frequency of only 2%. We suggest that the frequency of homosexuality in adoptive sons in adopting families showing no homosexual members be recorded. If that frequency is also 11%, we will be confronted with a higher frequency of gay genes in the population than expected. Another factor potentially involved is that adoptive children may be differently treated than biological children by parents and siblings, thus somehow leading to the high concordance for homosexuality.

Obviously human homosexuality is not wholly genetic. Not only do we have to consider the complexities of individual upbringing, but also culture is influential. Gene-culture coevolution (Lumsden and Wilson, 1981) might shed some light on these phenomena.

Since the concordance for homosexuality in human male monozygotic twins is only about 50% (Bailey and Pillard, 1991), there is clearly room for environmental influence.

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REFERENCES

Bailey, J. M., and Pillard, R. C. A genetic study of male sexual orientation. Archives of General Psychiatry 48: 1089-1096, 1991.

Bailey, J. M., Willerman, L., and Parks, C. A test of the maternal stress theory of human male homosexuality. Archives of Sexual Behavior 20: 277-293, 1991.

Dobzhansky, Th., Ayala, F. J., Stebbins, G. L., and Valentine, J. W. Evolution. San Francisco: W. H. Freeman, 1977.

Dörner, G., Geier, Th., Ahrens, L., Krell, L., Münx, G., Sieler, H., Kittner, E. and Müller, H. Prenatal stress as possible aetiogenetic factor of homosexuality in human males. Endokrinologie 75: 365-368, 1980.

_____, Schenk, B., Schmiedel, B. and Ahrens, L. Stressful events in prenatal life of bi- and homosexual men. Endocrinology 81: 83-87, 1983.

Ellis, L., Ames, M. A., Peckham, W., and Burke, D. Sexual orientation of human offspring may be altered by severe maternal stress during pregnancy. Journal of Sex Research 25: 152-157, 1988.

Ferguson, A. Can evolutionary theory predict? American Naturalist 110: 1101-1104, 1976.

Ferguson, T. A. Y. Sudden eusociality in the Hymenoptera. Evolutionary Theory 10: 183-186, 1993.

Hamer, D. H., Hu, S., Magnuson, V. L., Hu, N., and Pattatucci, A. M. L. A linkage between DNA markers on the X-chromosome and male sexual orientation. Science 251: 321-327, 1993.

Hamilton, W. D. The genetical evolution of social behaviour. Journal of Theoretical Biology 7: 1-57, 1964.

Holldobler, B., and Wilson, E. O. The Ants. Cambridge: Harvard University Press, 1990.

Hutchinson, G. E. A speculative consideration of certain possible forms of sexual selection in man. American Naturalist 93: 81-91, 1959.

Kerchner, M., and Ward, I. L. SDN-MPOA volume in male rats is decreased by prenatal stress, but is not related to ejaculatory behavior. Brain Research 581: 244-251, 1992.

Lee, R.B. The !Kung San. Cambridge: Cambridge University Press, 1979.

LeVay, S. A difference in hypothalamic structure between heterosexual and homosexual men. Science 253: 1034-1037, 1991.

_____. The Sexual Brain. Cambridge, Mass.: MIT Press, 1993.

Lumsden, C. J., and Wilson, E. O. Genes, Mind and Culture. Cambridge, Mass.: Harvard University Press, 1981.

Rhees, R. W., and Fleming, D. E. Effects of malnutrition, maternal stress, and ACTH injections during pregnancy on sexual behavior of male offspring. Physiology and Behavior 27: 879--882, 1981.

Ruse, M. Homosexuality: A Philosophical Inquiry. Oxford: Blackwell, 1988.

Selye, H. The Stress of Life. 2nd ed. New York: McGraw-Hill, 1976.

Ward, I. L. Prenatal stress feminizes and demasculinizes the behavior of males. Science 175: 82-84, 1972.

_____. Sexual behavior differentiation: Prenatal hormonal and environmental control. In Sex Differences in Behavior, Friedman, R. C. et al. (eds.). New York. John Wiley, 1974, pp. 3-17.

_____. Exogenous androgen activates female behaviour in noncopulating prenatally stressed male rats. Journal of Comparative and Physiological Psychology 91: 465-471, 1977.

_____. The prenatal stress syndrome: Current status. Psychoneuroendocrinology 9: 3-11, 1984.

_____, and Weisz, J. Maternal stress alters plasma testosterone in fetal males. Science 207: 328-329, 1980.

Weinrich J. D. Human Reproductive Strategy. Unpublished PhD Thesis, Harvard University, 1976.

Wilson, E. O. The Insect Societies. Cambridge, Mass: Harvard University Press, 1971.

_____. Sociobiology. Cambridge, Mass.: Harvard University Press, 1975.

_____. On Human Nature. Cambridge, Mass.: Harvard University Press, 1978.