7

Pre-natal hormones? Stress? Immune attack?

Hormones

Many people have wondered if homosexuality is caused by exposure in the womb to unusual levels of male or female hormones. The theory is that if a male embryo is exposed to lower than normal levels of male hormones, or a female embryo to excess male hormones, the child may grow up homosexual. Such exposure to sex hormones may make lower animals bisexual. In this chapter we argue any such effect is small.

In normal development, it takes a natural surge of testosterone in the embryo to turn the female reproductive tracts into male sex organs. You could say that the default sexuality in the womb is female, and that, without the testosterone surge the embryo would remain female.

Treatments for medical conditions during pregnancy and certain rare hormonal conditions in humans have given researchers opportunity to study the effect of high or low levels of male and female hormones on the embryo in the womb and on later sexual orientation. We will look particularly at two of them.

Also, see Chapter Eight for detail of supposed effects on the brain.

Exposure to diethylstilbestrol

Between about 1940 and 1970, diethylstilbestrol, an artificial female sex hormone, was given to pregnant mothers at risk of miscarriage. (It is no longer administered because of increased risks of genital cancer in daughters and sons of these women.) The doses of diethylstilbestrol given to women in the study were very high: 5-250 mg. per day. In much later research, the children of these women were queried in detail about their sexual orientation in the previous eighteen months: fantasies, romantic/sexual daydreams, and many other detailed tests.[1] In two studies, there was slightly more lesbianism than in the controls (a normal comparison group), but two earlier studies found no difference in sexual orientation. A fifth study, the latest and most definitive[2] showed no difference. So, the girls were exposed to levels of female hormone far in excess of anything a fetus would naturally be exposed to, and, even at those very high levels, no effect was found.

A study of twenty boys,[3] exposed to diethylstilbestrol in the womb, showed that none had homosexual tendencies (though one of the non-exposed controls did). This suggests that pre-natal exposure to this hormone does not lead to homosexuality in men.

Adrenogenital syndrome

When girls are exposed to male hormones in the womb, one outcome is adrenogenital syndrome. You met adrenogenital syndrome in Chapter Five. To recapitulate briefly: in the development of a female fetus, the adrenal glands normally produce a hormone called cortisol which is involved in control of protein and carbohydrate metabolism. In adrenogenital syndrome, because of an enzyme deficiency, an androgen is produced instead. Girls exposed in the uterus to this hormone (at nine times the usual concentration) develop unusually large clitorises (more like miniature phalluses), and, if the condition is untreated, can grow up looking very masculine. These days, females with this condition are given life-long drug treatment to counteract the masculinising effect of the continuing androgen production. However, 40 years ago, girls with this condition were sometimes left untreated, and researchers have studied them to find effects on sexual orientation.

Earlier studies showed no effect on sexual orientation, but one study by sex researchers Money, Schwartz, and Lewis[4] came up with a

large group (37%) who were bisexual, but not lesbian, which seemed to show a large influence on sexual orientation. However, a survey of diabetic patients matched with the girls for age and hospital experience came up with identical levels of bisexuality. Unless we argue that diabetes also causes bisexuality, it would appear that common environmental factors in the two groups might have been responsible. These girls were frequently hospitalised[5] and subjected to much medical scrutiny and interviewed about their sexuality. The Money, Schwartz, and Lewis study has also been criticised for poor interviewing techniques, which over-estimated the bisexuality of the respondents.[6] For example, girls with this syndrome who are untreated are acutely embarrassed about themselves and often unwilling to talk about sex at all. In this study, they did not feel feminine and did not have boyfriends. Some of this may have been interpreted as bisexuality. In Chapter Three, we mentioned gender nonconformity as one of the strongest predictors of future homosexuality. These girls felt very different from their peers. They were particularly conscious of their excessive hairiness, which they said was the one thing they would like changed more than anything, even their deep voices.

Environmental factors appear to override hormonal influences, according to another study;[7] no correlation was found between masculine behaviour in girls with adrenogenital syndrome and increased physical masculinisation. In still another study of the condition, lesbianism seemed to be associated with poor vaginal function, in which the girls doubted their femininity.[8]

A Swedish paper[9] found some effect on sexual orientation. Non-heterosexual orientation was reported by 20% of the sample which was significantly different from controls. Meyer-Bahlburg and others,[10] conclude that there is definitely some effect, but it is rather modest. In a quite thorough investigation, 31% of women had crushes on other women (but so did 14% of the controls), 9% expressed love for other women which was just significantly different from the controls, 11% had actually had sex with other women, but this was not significantly different from the controls. "Most women were heterosexual, but the rates of bisexual and homosexual orientation were increased above controls not only in women with classical CAH, but also in those women with a non-classic form of the syndrome, and the effect correlated with the degree of prenatal androgenization."[10] Boys can also have this condition.

They are simply exposed to more male hormones than usual. This might be expected to completely eliminate homosexuality. But, in a sample of thirty, one experienced homosexual attraction.[11] This level (3%) is not significantly different from the occurrence of homosexuality in the normal population. The sample is too small to say much more, except that exposure in the uterus to excess masculinising hormone clearly does not eliminate homosexual orientation in males.

These results disproved the theory of pre-natal exposure to excess hormones as an infallible cause of homosexuality. Exposure to excess androgen had no effect on boys, and a modest effect on girls. The girls were exposed in the womb to one of the strongest doses of male hormones known in the scientific record, but a minority became bisexual or lesbian. What, then, can possibly be producing lesbianism in females experiencing normal conditions in the womb? Not exposure to pre-natal hormones, it seems.

In a lesser known 1974 study of 18 young women in Soviet Russia who had adrenogenital syndrome, none showed the slightest trace of lesbianism or lesbian erotic fantasy.[12] The author attributed this to stricter mores in the Soviet Union. Regardless, it seems the result is sensitive to social setting.

Subsequent papers confirmed more masculine-type play as children, and somewhat less heterosexual interests, but remarkably, in view of the high level of male hormone exposure, in one study of 250 girls, 95% had no problems with female gender identity.[46] One conclusion would be that the effects of the hormones were remarkably small.

Finger ratios and sexual orientation

In 2000 Williams et al.[13] Californian researchers, published results which seemed to confirm hormonal influence on sexual orientation. They measured finger length ratios at a gay and lesbian fair, and found the ratio of index/ring finger was significantly more "masculine" in lesbians. Since people are born with these ratios, this seemed evidence that pre-natal hormones, mainly testosterone, were powerfully influencing sexual orientation.

Digit ratios could be measured using a photocopier—an easy laboratory test!—so an explosion of confirmatory studies followed, and were extended to males, but the results for the men turned out to collapse in a

mess of contradictory papers, (one contributing factor to finger lengths for the men was ethnicity), and as at 2010 only the lesbian results are firm enough to comment on.

We must emphasise that the connection between the finger length and lesbianism is actually weak. Van Anders and Hampson[14] could only explain 6-9% of the variance (i.e., explain 6-9% of the lesbianism using finger lengths). Put simply, that is a very weak effect. Also, heterosexuals with the same finger ratios outnumbered lesbians 60 to 1.[13]

In a rather tour-de-force experiment, Lutchmaya et al.[15] measured the fetal hormone levels directly in the amniotic fluid of pregnant women and then much later, after birth, measured the digit ratios in the children. This did not look at sexual orientation of course— too early for that—and they found a relationship between the hormone ratios and the digit ratios, but again rather modest. However this result was only just statistically significant and it needs replication. Seventy three percent of the explanation for the digit ratios was not the hormones.

Twin researchers Paul et al.[16] did a study to find the extent of genetic influence (as opposed to hormonal influence) on the finger-length ratio and concluded that 66% of the effects were genetic. This is above average, moderately strong, but much stronger than the effect of hormones. The conclusion then is that there is some genetic feature which influences this ratio and that is predominant. Hormone effects are secondary at best, according to the authors.

McFadden[17] found that the women's finger length ratios did not correlate with other supposed markers of prenatal hormone exposure, called otoacoustic emissions, fluctuating asymmetry and visio-spatial expertise. It rather seems whatever the explanation for the effect, it is not very likely to be hormones.

The enticing idea that prenatal hormones are fixing one's sexual orientation in stone proves only to be a quite weak effect.

Other pre-natal hormone effects

Knickmeyer[18] used the same system as Lutchmaya et al., waited until the children were born, and observed their play. They found no link between pre-natal hormone levels and children's play whether gender-typical or atypical.

A more recent paper[19] also looking at sex hormones in amniotic fluid, similarly waited until the children were born and observed at 13 months the tendencies to play with gender-typical or atypical toys. This could be taken as a rough indication of future SSA. Though there were very clear gender-linked preferences for gender appropriate toys, this was totally unrelated to previous sex-hormone levels—except for progesterone, which makes no biological sense and which the authors themselves rejected. However there was a link with family structure—a large number of elder brothers suppressed masculine preference in boys. There was also a strong influence of more elder sisters—they promoted more feminine play in boys. A similar effect was seen for girls— an excess of elder sisters was linked to less feminine play. But these are social effects and much stronger than any prenatal sex-hormone effects. These social effects are not completely consistent with those in Chapter Three and more work is needed.

So prenatal sex hormone exposure did not even affect gender-typical play very much.

Adult exposure to sex hormones

Do sex hormone drugs given to adults have any effect on sexual orientation?

It was long believed that homosexuals had lower levels of testosterone (male hormone), or higher levels of estrogen (female hormone) in their bodies, and that lesbians had higher levels of testosterone and lower estrogen levels. The corrective step appeared to be administration of counter-balancing doses of whatever hormone was necessary. But it didn't work. Male homosexuals given male hormones only became more sexually active, not more heterosexual. So doctors experimented with doses of estrogen in the thirties to see if they stimulated androgen feedback responses. The father of computer science, Alan Turing, arrested for homosexual activities, was required to take estrogen. It had no apparent effect.[20] Courts ordering men to undergo hormonal treatment to change their orientation eventually stopped the practice as it became clear it was ineffective.

In the literature, as reviewed by New York hormone expert Meyer-Bahlburg, three studies suggested testosterone levels were lower in male homosexuals, twenty found levels in homosexuals were the same

as in heterosexuals, and two found elevated levels in homosexuals.[21] Another reviewer of the biomedical literature, from the Netherlands, Louis Gooren, remarks, "Not only have the best designed studies failed to find differences in hormone levels between homosexuals and heterosexuals, but…the scientific principles of endocrinology do not make that plausible."[3] Nor, he commented has it ever "been reported that sexual orientation underwent a shift induced by the change of levels of androgens and estrogens."

On the other hand, there is plenty of evidence that hormonal therapy raises or inhibits existing sex drive. Rates of sexual fantasy and orgasm more than tripled in one group of men being treated with androgen for very low levels of testosterone.[22] This is one of the strongest effects on record for heightened libido. A similar test of women on estrogen replacement therapy showed about a 20% increase in libido compared with controls.[23] When they are given to combat advanced breast cancer androgens also increase libido in women.[24] Some drugs decrease libido. Oral contraceptives tend to lower sex drive by about 30%, according to one study.[25] But, even in those cases, habits and mental attitudes can overrule. Even with chemical castration recommended for some sex offenders, some criminal sexual behaviour persisted because of mental habits that had been established. In one classic study, in which men were treated with estrogens and anti-androgens,[26] some criminal sexual behaviour continued even though sexual activity dropped to about 25% of normal, and interest to about 60%. Even physical castration has equivocal effects for many offenders. For some, sexual fantasy and performance decrease quite rapidly: in one study of 2500 sex offenders, repeated offences fell from 50% to 3.5%—but a small minority continued to be as sexually active as ever.[27] It is still possible for castrated men, paraplegics, or eunuchs to have mental orgasms.[28] But generally apathy sets in.

As one reviewer of the literature on hormones and libido comments,

The available literature suggests that humans have not escaped completely from the endocrinological control of sexual behavior and that humans are similar in certain ways to the other mammals. On the other hand it is also obvious that social learning plays an extremely important role in human sexual behavior.[24]

The placebo effect

Hormonal effects are often small compared with the effects of mental attitudes. People who think a treatment is going to work often show improvement even though the treatment is proven ineffective. This is called the placebo effect. Placebos are inactive substances, without physical curative effects, which are often used in drug trials. For this reason, double-blind trials are now the rule when drugs are being tested: neither the patient nor the researcher knows who received the placebo and who received the prescribed drug until afterwards. Studies of the effect of drugs on libido are subject to a strong placebo effect—people who believe the treatment will raise libido often show increased sex drive, suggesting that state of mind is one of the most powerful influences on human sexuality.

One researcher of the effect of hormones on libido (Brown-Sequard, in Paris) was notorious many years ago for insisting that a preparation of monkey testicles had revolutionised his sex life. Only much later did researchers learn that the testicles had been accidentally prepared in such a way that any sex hormones had been thoroughly eradicated. The effect was all in the mind. "Very many suggested effects on libido are anecdotal, and doubtful, and may arise from increases in general well-being," says one researcher in the field.[29]

Maternal stress

In rats, researchers have found a link between maternal stress and demasculinising effects in the sexual behaviour of male offspring. The mother's stress leads to a delayed testosterone surge in male rats. An East German researcher, Dorner, claimed to have found a similar stress effect in humans during the Second World War. If mothers underwent a lot of stress, he found no heterosexuality in their young offspring, 25% bisexuality, and 35% homosexuality. The remainder were too young to know what their preferences were.[10]

These were spectacular results, but the study appears to be maverick. Other studies on rats could not find the effect, and stress in human mothers delays the testosterone surge much less markedly than in rats. Dorner has also been criticised for not interviewing the mothers.[30] Three other studies on humans did not find any effect.[30] A later and more sophisticated study, although it found no correlations with

stress for boys, did find an unsurprising relatively strong correlation between homosexual fantasy and childhood gender non-conformity[30] (see Chapter Three). Curiously, in this study, there was a moderate correlation for girls between maternal stress and lesbianism, which made no sense to the authors. Girls are not exposed to a pre-natal testosterone surge, so a delayed surge makes no sense in this context.

The latest and biggest survey[31] basically concludes that there is a weak effect for boys and a more significant effect for girls. A similar survey for the stressful effects of an historic Dutch famine could find no effects.[32] In no case can the effects be described as overwhelming, which is why it has been so hard to establish. It is another minor factor in the development of homosexuality for a few people.

The Maternal Immune Hypothesis —the "anti-boy" antibody

Another popular recent theory to explain homosexuality is the "maternal immune hypothesis". It argues that an immune attack on the fetus by the mother predisposes to SSA.

This section will conclude that the hypothesis is much too speculative.

The maternal immune hypothesis[33] is that a male fetus may cause an immune reaction in the mother, rather similar to the development of Rhesus sensitivity in an Rh negative mother with an Rh positive baby. In this syndrome the first child is untouched, but the mother has an immune reaction, and any subsequent Rh positive children are severely attacked by the mother's antibodies, and may suffer neurological damage. The SSA hypothesis is that the mother reacts to the maleness of the first boy and creates antibodies that—like other maternal antibodies—penetrate the placenta and enter any subsequent male fetus, attacking developing brain tissue, particularly male-specific brain sites. Some researchers think lower birth weight is another result of this hypothesis.[34] The new-born boy is supposed to be predisposed to SSA. However this hypothesis does not try to explain SSA in a first-born and can be calculated to explain only 17% or less of total SSA.[35]

According to the theory, the antibodies in the mother increase with each male child, raising the likelihood of SSA with each subsequent birth.

There are several major problems with the theory:

One: immunological attack by the mother probably creates more frequent schizophrenia or autism[47] but neither was found in people with SSA when surveyed.[48]

Two: the original finding of an excess of older brothers in men with SSA is now looking increasingly doubtful. Many large samples cannot find the effect.

Three: if the attack is against male-specific targets then the testes should also be attacked since there are a lot more male-specific targets there. Attack on the testes would result in impairment of fertility in males with SSA. One would be likely to detect increases in four conditions which usually group together—poor semen quality, hypospadias (somewhat feminine deformation of the penis), and cryptorchidism (undescended testes). These three conditions are usually summed up in the following inclusive category: testicular dysgenesis (the testes do not develop). These conditions have many causes, and birth weight is also low. However none of them is known to be associated with homosexuality.

In fact, individuals with hypospadias have slightly increased psychological levels of masculinity.[36] This is interesting because for hypospadias, levels of testosterone are low right through pregnancy to the post-natal period. Is it really possible that the testes in the fetus under supposed immune attack by the mother can still produce levels of testosterone high enough to avoid hypospadias, but low enough to produce SSA? This doesn't make sense. Orchitis (inflammation of the testes) would be a symptom of generalised immune attack on maleness but neonatal orchitis is much less common in males than homosexuality is.

Work with large samples of adolescents shows there is no difference in age of puberty between SSA and OSA people.[37] But one would expect a later puberty if the functions of the testes are impaired by maternal immune attack.

An attack on "maleness" should particularly affect development of male genitalia in any fetus which is later SSA-prone. But the opposite

has been found. From the data gathered by Kinsey, penile lengths were statistically 0.8 cm longer for males with SSA than males with OSA.[38]

The biggest unanswered question is: if there is no attack on the testes which have the largest congregation of male-specific targets, why would there be on the brain? The best interpretation is that no such attack takes place.

Four: People with SSA do not show evidence of impaired brain function which would result from maternal attack on the male brain.

Attack on fetal male brain neurology has also been supposed from previous studies to manifest itself in learning difficulties, but in reading and writing rather than arithmetic.[39] However the known better verbal fluency in males with SSA[40] and the fact that they are not known for learning difficulties, argues that homosexuality is not a result of any supposed anti-male immune attack.

The most definitive study to date (Flannery and Liderman, 1994),[41] with a sample of 17,283 mother and son pairs, tested whether enhanced autoimmunity in the mother (a possible measure of attack on the fetus) was associated later in the child with cerebral palsy, mental retardation, seizures, articulation disorder, reading or arithmetic disability, verbal or performance aptitude deficits and ADHD. After controlling for birth factors, enhanced autoimmunity did not correlate with the above neurological problems. This large survey contradicted earlier surveys with poorer control, which gave rise to the idea of such a link (Gualtieri and Hicks, 1985).[42] Later work shows that the immune reaction was connected with later schizophrenia, autism, and depression, but not changed sexuality.[49] It seems we can add homosexuality to the list of conditions not related to maternal immune attack. A much more thorough criticism of the maternal immune hypothesis is given elsewhere.[43]

We have to conclude that there are several layers of hypothesis moving the maternal immune hypothesis from the "speculative" to the "very speculative" and there is evidence against each.

Summary

Although there are some pre-natal hormonal effects on sexual behaviour for lower animals, there is not convincing evidence for such an effect on sexual orientation in humans. The studies examining the effects of high doses of female hormones to pregnant women are particularly

informative because these are very high doses and any hormonal effects on sexual orientation should show up clearly. But the result is a dubious effect on women and no effects on men. Any effects on sexual orientation appear to be better explained in terms of gender non-conformity—a psychological construct. Sex hormones do increase or lower sex drive, but that appears to be about all.

The maternal immune hypothesis seems very speculative, and needs much more evidence before it is taken more seriously.

We leave the last word to several researchers in the field. James[44] summarises the evidence for effects of prenatal hormone exposure on subsequent sexual orientation as "weak".

In summary, the evidence from prenatal endocrine disorders and from the offspring of hormone-treated pregnancies suggests that hormones may contribute to, but do not actually determine, the course of sexual orientation in individuals with an abnormal sex steroid history during prenatal life.[3]

"At this time, the literature does not support a causal link between hormones and homosexuality."[12]

Also, "In clinical practice numerous patients are encountered with gross abnormalities of their hormonal profiles. As a rule this does not impact on their gender identity or sexual orientation."[45]

So, not only your genes didn't make you do it, it seems your hormones didn't either. In sexual orientation, the strongest stimulation appears to come from the mind and the environment.

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