

Does Pre-natal Hormonal Exposure Make You Homosexual?

Many people have wondered if same-sex attraction (SSA) is fixed or influenced 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 the boy may become homosexual, or a female embryo to excess male hormones, the girl may grow up lesbian. Such exposure to sex hormones may make lower animals bisexual.

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 (and the action of another hormonal substance that inhibits the normal development of female organs), the embryo would remain female. Normally, a trace of a female sex hormone, estradiol, is also needed for growth in the uterus of both male and female babies.

A popular recent theory is the "maternal immune hypothesis". It argues that decreased male hormones in the fetus arising from an immune attack from the mother, predispose to SSA..

The maternal immune hypothesis²⁴ 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. The first child is untouched, but the mother has an immune reaction, and subsequent Rh positive children are severely attacked by the antibodies of the mother, and neurological damage is one of the consequences. The specific hypothesis for SSA is that the mother reacts to the maleness of the first boy and creates antibodies. These, like any other maternal antibodies, penetrate the placenta and enter any subsequent fetus. If the fetus is a boy, the hypothesis supposes a specific attack on his developing neural tissue - a male-specific attack. It also claims some less specific attack that lowers the birth weight²⁵. The resulting boy is supposed to be predisposed to SSA. (This hypothesis does not try to explain SSA in a first-born. Since many with SSA are first born or have sisters only, the maternal immune hypothesis can only attempt to explain 17% or less of total SSA²⁶ (Cantor *et al.* 2002)).

According to the theory, the antibodies in the mother increase with each subsequent pregnancy of a male hence the possibility of SSA also increases constantly. We will examine this later in this chapter and conclude it is highly speculative. Some facts definitely conflict with it.

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 at four of them.

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.) Research on female guinea pigs had shown a link between synthetic female hormone and sexual behavior, though not in an expected direction. It led to increased masculine sexual behavior in female guinea pigs. It also led - more predictably - to decreased masculine behavior in male rats. Researchers wondered if the same effects might be found in humans.

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 definitive² 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 SSA 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 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 masculinizing effect of the continuing androgen production. However, forty 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 Lewis⁴ came up with a large group (37 percent) 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. These girls were frequently hospitalized and subjected to much medical scrutiny and interviewed about their sexuality. The diabetes caused them to have poor lubrication, difficulty coming to orgasm, and sexual repulsion⁵. This only happened with type II diabetes which has a strong lifestyle origin. The Money, Schwartz, and Lewis study has also been criticized 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, by default. 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 behavior in girls with adrenogenital syndrome and increased physical masculinization. In still another study, lesbianism seemed to be associated with poor vaginal function, in which the girls doubted their femininity.⁸

The latest paper by Meyer-Bahlburg and others⁴⁰, is the latest word so far and concludes that there is definitely some effect, but it is rather modest: 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 Congenital Adrenal Hyperplasia, but also in those women with a non-classic form of the syndrome, and the effect correlated with the degree of prenatal androgenization".⁴⁰

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 the sample of thirty, one experienced homosexual attraction.⁹ This level (3%) is not significantly different from the incidence of homosexuality in the normal population. The sample is too small to say much more, except that exposure in the uterus to excess masculinizing 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 study in 1974, of 18 young women in Soviet Russia who had adrenogenital syndrome, none showed the slightest trace of lesbianism or lesbian erotic fantasy¹⁰ The authors 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. One conclusion would be that the effects of the hormones were remarkably small.

Androgen Insensitivity Syndrome

What would happen if a chromosomal male (XY) was not exposed to any androgen at all in the womb? If anything hormonal was going to produce homosexuality in a male child, you would think this would be it. But instead the child develops as a girl, with a heterosexual orientation. The fetus remains feminine because the testosterone surge does not occur. Androgen insensitivity syndrome is a rare condition in which the cells in the developing male fetus lack the receptors for the hormone testosterone, meaning that, however much is produced by the testes, the cells will not respond to it. Children are, therefore, born with external female genitalia, but have testes in the abdominal cavity. However, the cells do seem to be sensitive to female sex hormones, because breasts and typical female shape develop at puberty (under the influence of traces of female sex hormones produced by the testes!), but, because there are no ovaries, there are no menses, and the condition is usually discovered only at that point. These people otherwise are brought up as females. They develop a female gender identity and a sexual attraction toward men. Their patterns of sexual arousal and kinds of erotic imagery are indistinguishable from normal females.⁴

A rare condition with similar effects is called 17alpha-hydroxylase deficiency. The absence of a particular enzyme in the production pathway for male hormones means that neither the adrenal glands nor the testes produce any male hormone at all. Such a person looks like a girl when born. Without intervention, he also grows up indistinguishable from a girl.¹¹

These conditions emphasise the very large effects of social conditioning!

Maternal Stress

In rats, researchers have found a link between maternal stress and demasculinizing effects in the sexual behavior 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 effect in humans during the Second World War. If mothers underwent heavy stress, he found no heterosexuality in their young offspring, 25 percent bisexuality, and 35 percent homosexuality. The remainder were too young to know what their preferences were.¹²

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 criticized for not interviewing the mothers.¹² Three other studies on humans did not find any effect. The latest and most sophisticated of the three studies, although it found no correlations with stress for boys, did find a relatively strong correlation between homosexual fantasy and childhood gender non-conformity¹² (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.

A still more recent study¹³ repeated Dorner's study with people in the most stressed German cities, but found no increased rates of homosexuality.

However the latest and biggest survey²⁷ basically concludes that there is a small weak effect for boys and a more significant effect for girls. In no case can the effects be described as overwhelming, which is why the link has been so hard to establish. At most it is another minor factor and can be explained psychologically.

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.¹⁴ 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.¹⁵ 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.¹⁶ 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 percent increase in libido compared with controls.¹⁷ Androgens also increase libido in women when they are given to combat advanced breast cancer.¹⁸ Some drugs decrease libido. Oral contraceptives tend to lower sex drive by about 30 percent, according to one study.¹⁹ But, even in those cases, habits and mental attitudes can overrule. Even with chemical castration recommended for some sex offenders, some criminal behavior persisted because of mental habits that had been established. In one classic study, in which men were treated with estrogens and anti-androgens,²⁰ some criminal sexual behavior continued even though sexual activity dropped to about 25 percent of normal, and interest to about 60 percent. 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 percent to 3.5 percent - but a small minority continued to be as sexually active as ever.²¹ It is still possible for castrated men, paraplegics, or eunuchs to have mental orgasms.²² 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."¹⁸

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. Because of the placebo effect, 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. 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 revolutionized 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 his 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.²³

Finger Ratios and Sexual Orientation

In 2000 Williams et al.,³⁴ 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 length 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 collapsed in a chaos of contradictory papers, and as at this date only the lesbian results are firm enough to comment on.

We must emphasise that the connection between the finger length and lesbianism is actually fairly weak. Van Anders and Hampson³⁶ could only explain 6-9% of the variance. In English rather than tech-ese, that is a weak effect. For any finger length ratio chosen, lesbians in the population are outnumbered by heterosexuals 60 to 1. It seems having a “masculine” finger-length ratio only influences women towards SSA in a small minority of cases, at best.

In a rather tour-de-force experiment, Lutchmaya et al (2004)³⁷ measured the fetal hormone levels directly in the amniotic fluid and then much later, after birth, measured the digit ratios. 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: 73% of the explanation for the digit ratios was not the hormones.

Twin researchers Paul *et al.*³⁸ did a study to find the extent of genetic influence (as opposed to hormonal influence) and concluded that 66% of the effects influencing the digit ratio 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. Hormones are secondary at best, according to the authors.

We do have to ask – are the lesbians aware their bodies are more masculine than they would like and does this influence their ideas about their sexual orientation? It’s certainly a good possibility. So the hormonal influence idea is a little shaky on those grounds alone.

Prenatal hormones fixing one’s sexual orientation in stone is an enticing idea but the effect is weak.

Pre-natal hormone effects

A recent paper (van de Beek, van Goozen, et al. 2008⁴²) is one of a few which checks the hormonal state of the fetus by actual analysis of the amniotic fluid. They then waited until the children (both boys and girls) 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.

The results were that though there were very clear gender-linked preferences for gender appropriate toys, this was totally unrelated to fetal sex-hormone levels – except for progesterone in boys which made them more masculine and makes no biological sense, according to the authors.

However there was a link with elder brothers – the more elder brothers the more masculine preference was suppressed – a kind of Fraternal Birth Order effect. But there was also a definite influence of more elder sisters – they promoted more feminine play in boys. A similar effect was seen for girls: the more elder sisters the less feminine play. These seem to be social effects and much stronger than any prenatal sex-hormone effects.

The Maternal Immune Hypothesis – the “antiboy” antibody

According to the hypothesis the mother’s immune reaction is against unique male proteins, and targets the fetal brain causing homosexuality. But we have to ask – if the attack is against male-specific targets then the testes must also be attacked. 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 included in the last: testicular dysgenesis (the testes do not develop). These conditions are multifactorial in origin, 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.²⁸ This is interesting because for hypospadias, levels of testosterone are low right through pregnancy to the post-natal period. The testes in the foetus under 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.

Early work suggested males with SSA statistically experience puberty a few months earlier than males with OSA²⁹ (Blanchard & Bogaert, 1996a). This contradicted any maternal immune attack hypothesis; you would expect puberty to be later if the functions of the testis were impaired. Later and better work with much larger samples shows the difference in puberty is statistically insignificant (Savin-Williams and Ream 2006.³⁹) Again, this would not be expected 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.³²

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.

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.³⁰ However the known better verbal fluency in males with SSA³¹ 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 & Liderman, 1994)³³, with a sample of 17,283 mother and son pairs, tested whether enhanced autoimmunity in the mother 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.)⁴¹

We have to conclude that there are several layers of hypothesis moving the Maternal Immune Hypothesis from the "speculative" to the "very speculative". For a much more thorough treatment please consult (Whitehead 2007)⁴³.

Summary

Although there are some pre-natal hormonal effects having a major effect on sexual behavior for lower animals, there is no 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 – arguably a psychological construct. Sex drugs do increase or lower sex drive, but that appears to be about all.

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

We leave the last word to several researchers in the field.

"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."¹⁰

“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.”³⁵

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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