

# Fraternal Birth Order and the Maternal Immune Hypothesis of Male Homosexuality

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In men, sexual orientation correlates with an individual's number of older brothers, each additional older brother increasing the odds of homosexuality by approximately 33%. It has been hypothesized that this fraternal birth order effect reflects the progressive immunization of some mothers to Y-linked minor histocompatibility antigens (H-Y antigens) by each succeeding male fetus and the concomitantly increasing effects of such maternal immunization on the future sexual orientation of each succeeding male fetus. According to this hypothesis, anti-H-Y antibodies produced by the mother pass through the placental barrier to the fetus and affect aspects of sexual differentiation in the fetal brain. This explanation is consistent with a variety of evidence, including the apparent irrelevance of older sisters to the sexual orientation of later born males, the probable involvement of H-Y antigen in the development of sex-typical traits, and the detrimental effects of immunization of female mice to H-Y antigen on the reproductive performance of subsequent male offspring. The maternal immune hypothesis might also explain the recent finding that heterosexual males with older brothers weigh less at birth than heterosexual males with older sisters and homosexual males with older brothers weigh even less than heterosexual males with older brothers.

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**Key Words:** birth order; birth weight; homosexuality; anti-H-Y antibodies; H-Y antigen; maternal immune hypothesis; parity; sexual orientation.

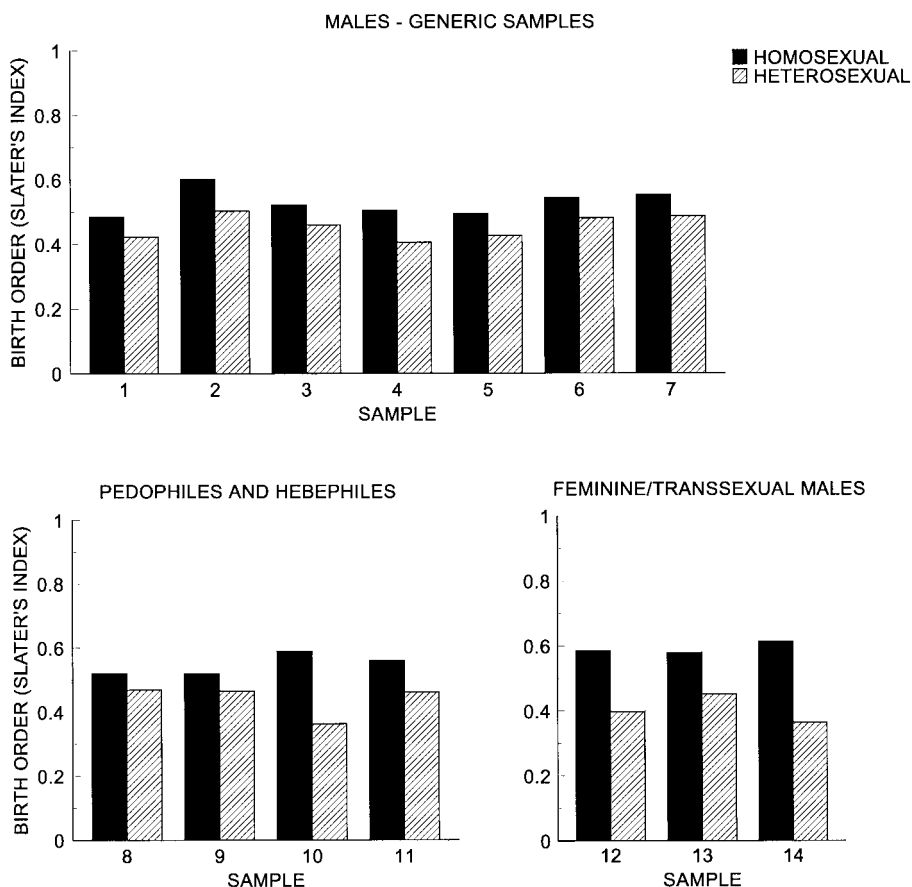
Researchers began asking at least 60 years ago whether older siblings increase or decrease the odds of homosexuality in later born children. Since 1989, the

author, his colleagues, and other researchers have investigated this question in subjects examined in recent years and in subjects examined decades ago; in groups collected in England, The Netherlands, Canada, and the United States; in psychiatric patients and in non-patient volunteers; in subjects examined in adulthood and in subjects examined in childhood; in transsexual subjects and in subjects contented with their assigned gender; and in men sexually attracted to adults as well as men attracted to children.

In order to provide an overview of the results of the author's research program, the birth order data from each study have been converted into a common metric, namely Slater's Index (Slater, 1962). Slater's Index equals the number of siblings older than the subject divided by the subject's total number of siblings, that is,  $\text{older siblings}/(\text{older siblings} + \text{younger siblings})$ . This index cannot be calculated for only-children; for all other individuals, regardless of sibship size, it expresses birth order as a quantity between 0 and 1, where 0 corresponds to firstborn and 1 corresponds to lastborn. The results are shown in Fig. 1 (see also Table 1). In this figure, taller bars denote later births. The results show that, in every one of the 14 samples, the homosexual subjects were born later in their sibships than the heterosexual subjects.

One can compute, as a kind of simple meta-analysis, the probability that the same group would show the higher mean 14 of 14 times purely by chance. That probability is about 1 in 10,000. These data therefore establish beyond much doubt that homosexual males do, on average, have higher birth orders than comparable heterosexuals. Because the sexual orientation of a newborn boy cannot operate backward in time to affect his number of older siblings, this finding implies

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**FIG. 1.** Mean birth orders of homosexual and heterosexual subjects from studies in the Clarke research program on biodemographic correlates of sexual orientation. A brief description of the subjects in each sample is given in Table 1. The black bars represent the mean birth orders for homosexual subjects, and the light bars represent the mean birth orders for heterosexual subjects. Taller bars denote higher birth orders, that is, later births. *Hebephiles* are men who are most attracted sexually to pubescent children, as opposed to *pedophiles*, who are most attracted sexually to prepubescent children.

that the number of older siblings—or some factor associated with that—must affect the newborn boy's sexual orientation.

The foregoing conclusion immediately raises a number of further questions, for example: Is it really maternal or paternal age, rather than the number of older siblings, that influences sexual orientation in males? If it really is the number of older siblings, is it the number of older brothers, older sisters, or both? What is the relation between birth order and sexual orientation, if any, in females? Most of the relevant data have already been reviewed in detail elsewhere (Blanchard, 1997). In the following section, therefore, the answers to these questions are presented in summary form.

## FURTHER BIODEMOGRAPHIC ANALYSES

Older brothers increase the odds of homosexuality in later born males (Blanchard and Bogaert, 1996a, 1996b, 1998; Blanchard *et al.*, 2000; Blanchard, Zucker, Siegelman, Dickey, and Klassen, 1998; Bogaert, Bezeau, Kuban, and Blanchard, 1997; Ellis and Blanchard, 2001; Green, 2000; Jones and Blanchard, 1998; Robinson and Manning, 2000; see also Blanchard and Bogaert, 1997a; Purcell, Blanchard, and Zucker, 2000; Williams *et al.*, 2000; Zucker *et al.*, 1997). The best available data indicate that each additional older brother increases the odds of homosexuality by 33% (Blanchard and Bogaert, 1996b).

TABLE 1

Studies on Birth Order and Sexual Orientation in Males from the Clarke Research Program on Biodemographic Correlates of Sexual Orientation

Sample	Authors	Description of the sample	Number homosexual <sup>a</sup>	Number heterosexual <sup>a</sup>
1	Blanchard and Zucker (1994)	American volunteers from earlier study by Bell <i>et al.</i> (1981)	451	215
2	Zucker and Blanchard (1994)	American psychoanalytic patients from earlier study by Bieber <i>et al.</i> (1962)	85	61
3	Blanchard and Bogaert (1996a)	American volunteers interviewed by Alfred Kinsey and associates from 1938 to 1963 (see Gebhard and Johnson, 1979)	666	3305
4	Blanchard and Bogaert (1996b)	Canadian volunteers from two groups of 302 probands originally matched on year of birth	278	285
5	Blanchard <i>et al.</i> (1998)	British and American volunteers from earlier studies by Siegelman (1972, 1973, 1974, 1978, 1981)	311	188
6	Blanchard and Bogaert (1998)	American sex offenders against adults from earlier study by Gebhard <i>et al.</i> (1965)	128	156
7	Ellis and Blanchard (2001)	American and Canadian volunteers	168	893
8	Blanchard and Bogaert (1998)	American sex offenders against pubescents from earlier study by Gebhard <i>et al.</i> (1965)	59	121
9	Blanchard and Bogaert (1998)	American sex offenders against children from earlier study by Gebhard <i>et al.</i> (1965)	37	131
10	Bogaert <i>et al.</i> (1997)	Canadian pedophilic patients referred for assessment	63	55
11	Blanchard <i>et al.</i> (2000)	Canadian pedophilic and hebephilic patients referred for assessment.	60	141
12	Blanchard and Sheridan (1992)	Canadian outpatients referred for assessment of gender dysphoria (roughly, transsexualism); subsample matched on sibship size and decade of birth	117	117
13	Blanchard <i>et al.</i> (1995)	Canadian child and adolescent outpatients, matched on age at presentation, sibship size, and year of birth	156	156
14	Blanchard <i>et al.</i> (1996)	Dutch gender-dysphoric patients, adult and adolescent samples combined	100	76

<sup>a</sup> These are the numbers of male subjects for whom Slater's Index could be calculated, that is, those with at least one sibling. The total number of subjects in this table is somewhat less than the total number of males in these studies because only-children are not represented here and because—in studies in which the main conclusions were based on matched subsamples—the subsamples were the source of data for the present Table 1 and Fig. 1.

Older sisters have no effect on the odds of homosexuality in later born males (Blanchard and Bogaert, 1996a, 1996b, 1998; Blanchard *et al.*, 1998, 2000; Bogaert *et al.*, 1997; Ellis and Blanchard, 2001; Green, 2000; Jones and Blanchard, 1998; Robinson and Manning, 2000). Only one study that is inconsistent with this conclusion has been reported (Bogaert, 1998).

The relation between number of older brothers and male homosexuality is not an artifact of higher maternal or paternal age (Blanchard and Bogaert, 1996a, 1996b, 1997b, 1998; Blanchard and Sheridan, 1992; Bogaert *et al.*, 1997) or of birth interval (Blanchard and Bogaert, 1997c).

Neither older brothers nor older sisters affect the odds of homosexuality in later-born females (Blanchard, 1997; Blanchard *et al.*, 1998; Bogaert, 1997; Ellis and Blanchard, 2001). It therefore appears that females are invisible to the birth order phenomenon. Females

do not influence their siblings' sexual orientation, and their siblings do not influence theirs.

These findings have established a man's number of older brothers, or *fraternal birth order*, as the only environmental variable that has ever been shown to correlate reliably with sexual orientation. The relation between high fraternal birth order and homosexuality has been called the *fraternal birth order effect* (Blanchard, 1997).

## FRATERNAL BIRTH ORDER AND OTHER DIMENSIONS OF EROTIC ATTRACTION

It is clear that fraternal birth order—or some factor associated with it—affects the gender of persons to

whom a man is sexually attracted. Does fraternal birth order also affect the *age* of persons to whom a man is attracted? Results from the study by Blanchard *et al.* (2000) indicate that fraternal birth order does not affect the age of preferred erotic targets.

On the other hand, some previous research has suggested that fraternal birth order might correlate with sexual variations other than homosexuality (Lalumière, Harris, Quinsey, and Rice, 1998). That study was carried out on a highly heterogeneous sample of sex offenders, however, and it is not clear what those other variations might be. The findings of Blanchard *et al.* (2000) indicate that, if fraternal birth order does correlate with other sexual variations, pedophilia is not one of them. Establishing whether—or which—sexual variations besides homosexuality correlate with fraternal birth order could be important in discovering the mechanism of action behind this variable.

## QUANTITATIVE ISSUES

On several occasions, reporters from the popular media have asked the author whether his findings will incite homophobic parents to start aborting male fetuses after they have already produced two or three sons. It can readily be shown that the birth order effect is not strong enough for the purpose of family planning, whether by homophobic parents or by parents with some other agenda.

Let us assume that the prevalence of homosexuality in men with no older brothers is 2%. In other words, the probability ( $P$ ) of homosexuality for that group is 0.0200. That probability corresponds to odds of 0.0204, where odds are defined as  $P/(1 - P)$ . Let us further assume that the odds of homosexuality are increased 33% by each additional older brother (Blanchard and Bogaert, 1996b). One can then compute the odds of homosexuality for men with any number of older brothers, and these odds can be converted back into probabilities using the formula,  $P = Odds/(1 + Odds)$ . Table 2 shows the odds and probabilities of homosexuality for men with zero to five older brothers.

The table shows that the probability that a couple's son will be gay rises from 2% for their first son to 6% for their fifth son. That is a threefold increase. However, 94% of fifth sons will still be heterosexual. Therefore, couples who attempted to abort prehomosexual sons would, in the overwhelming majority of instances, be aborting preheterosexual sons.

TABLE 2

Odds and Probability of Homosexuality as a Function of Number of Older Brothers, Attributable Fraction for Men with Zero to Five Older Brothers, and Frequency Distribution of Older Brothers from Blanchard and Bogaert (1996b)

Older brothers	Odds	Probability (prevalence)	Attributable fraction	Frequency distribution
0	.0204	.0200	.0000	.5464
1	.0271	.0264	.2432	.2848
2	.0361	.0348	.4260	.1126
3	.0480	.0458	.5634	.0397
4	.0639	.0600	.6668	.0132
5+	.0849	.0783	.7445	.0033

The foregoing analysis raises the question, how strong *is* the fraternal birth order effect? One way of approaching this is to ask: Of all gay men, how many acquired their sexual orientation through the older brother route?

The difference in the prevalence of homosexuality among men with one older brother and the prevalence among men with no older brothers represents the increase in prevalence attributable to the fraternal birth order effect. This increase, divided by the (total) prevalence of homosexuality among men with one older brother, represents the proportion of gay men with one older brother who acquired their sexual orientation via the older brother route,  $(.0264 - .0200)/.0264 = .24$ . In other words, almost one-fourth of gay men with one older brother owe their sexual orientation to their fraternal birth order. In epidemiology, this quantity is called the *attributable fraction* or *attributable risk*. Table 2 shows the attributable fractions for men with zero to five older brothers. Cantor, Blanchard, Paterson, and Bogaert (in press) give the derivation of an equation that allows these attributable fractions to be calculated directly.

The epidemiological statistic called the *population attributable fraction* (or *population attributable risk*), applied to the present context, is the proportion of all gay men who acquired their sexual orientation via the older brother route. This is the sum of attributable fractions for gay men with 0, 1, 2, . . . ,  $n$  older brothers, weighted by the proportions of gay men who have 0, 1, 2, . . . ,  $n$  older brothers. In Table 2 (rightmost column), these weights are estimated by the frequency distribution of older brothers obtained by Blanchard and Bogaert (1996b) for their homosexual subjects. The weighted sum is therefore the cross-product of the last two columns in Table 2,  $(.0000 \times .5464) + \dots +$

(.7445  $\times$  .0033), which equals .15 or 15%. This means, in round numbers, that roughly one gay man in seven owes his sexual orientation to the fraternal birth order effect.

Cantor *et al.* (in press) showed that their equation for the attributable fraction, when solved for number of older brothers, could be used to calculate the number of older brothers for which the attributable fraction is .50—the point at which 50% of gay men owe their sexual orientation to fraternal birth order. Inspection of Table 2 shows that this quantity must lie somewhere between 2 and 3 older brothers. In fact, it is almost exactly 2.5 (Cantor *et al.*, in press). This means that, for men with 2.5 older brothers, older brothers equal all other causes of homosexuality combined.

## BIRTH WEIGHT AND THE TIMING OF THE FRATERNAL BIRTH ORDER MECHANISM

None of the data presented so far addresses the *timing* of the fraternal birth order effect. Does this etiologic factor operate during a man's prenatal development, or in his early childhood, or even later, in puberty or adolescence? The author's most recent finding bears on this question. This finding arose unexpectedly, in a study of sexual orientation, birth order, and birth weight.

Blanchard and Ellis (in press) studied 3229 adult, homosexual and heterosexual, men and women (the probands), whose mothers knew the sex of every child (or fetus) that they carried prior to the proband. Information on birth weight, maternal gravidity, and other demographic variables was reported on questionnaires completed by the probands' mothers. The results for the heterosexual subjects confirmed earlier reports that boys with older brothers weigh less at birth than boys with older sisters (Magnus, Berg, and Bjerkedal, 1985; Trotnow, Bregulla, and Flügel, 1976). Comparisons of the homosexual and heterosexual subjects showed that homosexual males with older brothers weigh even less at birth (about 170 g) than do heterosexual males with older brothers. In contrast, homosexual and heterosexual males with no older siblings, or older sisters only, do not differ in birth weight. These findings suggest that prior male pregnancies do influence the development of subsequent male fetuses; that this influence is felt to varying degrees by individual fetuses; and that those fetuses who

are most strongly affected by this process, as indicated by their lower birth weights, are also those most likely to be prehomosexual. They also establish below-average birth weight as the first characteristic to be linked with a specific developmental pathway to homosexuality. Finally, these findings suggest that the factors that determine this pathway—factors related to a mother's number of prior male pregnancies—operate before the individual's birth.

The birth weight data represent the last of the currently available epidemiological findings regarding birth order and sexual orientation. The question next arises: What can explain these findings?

## THE MATERNAL IMMUNE HYPOTHESIS

Blanchard and Bogaert (1996b) conjectured that the high fraternal birth order of homosexual men may reflect a maternal immune reaction, which is provoked only by male fetuses and which becomes stronger after each pregnancy with a male fetus. This hypothesis rests partly on the argument that a woman's immune system would appear the biological system most capable of "remembering" the number of male (but not female) fetuses that she has previously carried and of progressively altering its response to the next fetus according to the current tally of preceding males. They further theorized that the relevant fetal antigen might be one of the male-specific, Y-linked, minor histocompatibility antigens (e.g., Pierce *et al.*, 2000), often referred to collectively as H-Y antigen (for reviews, see Müller, 1996; Wolf, 1998). Because H-Y antigen is expressed by male fetuses but not by female fetuses, a woman's immune system could selectively recognize, remember, and respond to male fetuses.

Building on these facts and suppositions, Blanchard and Bogaert (1996b) formulated the maternal immune hypothesis, which can be summarized as follows: H-Y antigens are present on cell surfaces in males (but not in females) from a very early stage of fetal development (Epstein, Smith, and Travis, 1980; Krco and Goldberg, 1976; Shelton and Goldberg, 1984; White, Anderson, and BonDurant, 1987; White, Lindner, Anderson, and BonDurant, 1983). H-Y antigen from male fetuses sometimes gets into the mother's circulation and alerts her immune system. The mother produces antibodies to H-Y, which cross the placental barrier and enter the fetal brain. When that happens, these antibodies partly prevent the fetal brain from developing in the male-typical pattern, so that the

individual will later be attracted to men rather than women. The probability—or strength—of maternal immunization increases with each male fetus, therefore the probability of homosexuality increases with each older brother.

## INDIRECT EVIDENCE

Various lines of evidence that bear on the feasibility of the maternal immune hypothesis have been reviewed by Blanchard and Klassen (1997). H-Y antigen almost certainly plays some role or roles in the sexual differentiation of vertebrates (Wachtel, 1983) because it is usually present in the heterogametic and absent in the homogametic sex—in mammals, present in males and absent in females—and because it has been highly conserved throughout vertebrate evolution (Nakamura, Wachtel, Lance, and Beçak, 1987; Wachtel, Koo, and Boyse, 1975). The possible involvement of H-Y antigen in the development of sex-typical traits suggests that anti-H-Y antibodies produced by the mother and passed through the placental barrier to the fetus could affect subsequent sexual behavior in men.

However well H-Y antigen might fit the requirements of the immune theory, it cannot be relevant unless the immune systems of pregnant females recognize and react to it. Animal research (reviewed by Blanchard and Klassen, 1997) has clearly established that the maternal immune system does respond to fetal H-Y antigen. H-Y antigen has been hypothesized as the reason that male fetuses are more likely to provoke immune reactions in human mothers than are female fetuses (Blanchard and Klassen, 1997). Evidence of such reactions includes higher levels of antibodies, presumably of maternal origin, in the sera of male fetuses or their mothers and the finding that male fetuses are more likely than female fetuses to initiate maternal Rh immunization (see also Gualtieri and Hicks, 1985).

If maternal anti-H-Y antibodies influence future sexual orientation by acting on the fetus, they most likely do so by diverting the sexual differentiation of the fetal brain from the male-typical pathway. The fetal brain is potentially accessible to anti-H-Y antibodies because the blood-brain barrier is not completely developed during prenatal life. Therefore the plausibility of the maternal immune hypothesis rests on the answers to two questions: (a) How likely is it that brain tissue would be a target of anti-H-Y antibodies; and (b) Could anti-H-Y antibodies affect sexual differenti-

ation in the brain without affecting differentiation in other sex-dimorphic organs, in particular, the genitalia? The first question is answered by evidence, reviewed by Blanchard and Klassen (1997), that H-Y antigen is strongly represented on the surfaces of brain cells. The second question is addressed by evidence that, in mice, testes can develop in the complete absence of H-Y antigen (McLaren, Simpson, Tomonari, Chandler, and Hogg, 1984). It is therefore plausible that the genitalia could develop normally in human males whose H-Y antigen is only partly bound by anti-H-Y antibodies.

The only relevant animal study also supports the notion that exposure to anti-H-Y antibodies *in utero* could affect subsequent sexual behavior in males. Singh and Verma (1987) immunized two groups of female mice to H-Y antigen by injecting them intraperitoneally with male spleen cells. One group was from an inbred and one from an outbred strain. Two groups of female control mice from the same strains were injected with saline solution. All four groups were then allowed to mate and give birth to their litters. When the male pups from these litters had grown to maturity, the investigators studied their reproductive performance. Only 10% of the males born to the immunized females “performed successful mating” (p. 151) compared with 100% of the males born to the control females; these percentages were the same for the inbred and the outbred animals. Singh and Verma did not, unfortunately, give any further description of the “very poor reproductive performance” (p. 151) of the sons of immunized mothers; this could represent a lack of interest in estrous females, incompetence at mounting in the correct position, or a failure to intromit a sufficient number of times to ejaculate. The behavioral deficit appears to have been somewhat independent of gonadal abnormalities; the inbred males were described as nearly sterile but the outbred males were not.

## CAN THE MATERNAL IMMUNE HYPOTHESIS ALSO EXPLAIN THE BIRTH WEIGHT FINDINGS?

One might conjecture that a mild maternal immune response to Y-linked antigens produces a slightly reduced birth weight, whereas a stronger immune response produces a markedly reduced birth weight as well as an increased probability of homosexuality.

Thus, in retrospective studies, males with older brothers weigh less at birth than males with older sisters, and homosexual males with older brothers weigh less yet. In other words, the findings of Blanchard and Ellis might represent a kind of dosage effect.

The plausibility of this explanation is supported by animal studies of maternal immunization to paternal antigens, which have shown that such immunization can affect fetal weight, placental weight, or both. Both increases and decreases in conceptus weight have been obtained, depending on the experimental procedure (e.g., Gentile, Malan Borel, Angelucci, Miranda, and Margni, 1992; Lu and Dawson, 1986; Saji, Nakamura, Wakao, and Negoro, 1980). It has also been shown that the same procedure can produce a decrease in fetal weight along with an increase in placental weight (Zuckermann and Head, 1985). This last result is particularly relevant to the human data because all three available studies of birth weight—including that by Blanchard and Ellis—indicate that prior male pregnancies decrease fetal weight for subsequent males, whereas the one relevant study of placental weight in humans suggests that prior male pregnancies increase placental weight for subsequent males (Vernier, 1975). Vernier suggested that the larger placentae of newborn boys with older brothers are related to progressive maternal immune reactions to Y-linked antigens, and Zuckermann and Head proposed that placental size may increase in the face of immunologic attack as a compensatory response that mitigates damage to the fetus.

The maternal immune hypothesis could also explain why the author found no difference in birth weight between firstborn homosexual and heterosexual men or between homosexual and heterosexual men who had older sisters only. This hypothesis implies that homosexual men with no older brothers must have acquired their sexual orientation by other means, for example, polymorphic “gay genes” (e.g., Hamer, Hu, Magnuson, Hu, and Pattatucci, 1993; Hu *et al.*, 1995). Their birth weights would not, therefore, show signs of maternal immune attack.

## PATHWAYS AND PITFALLS FOR FUTURE RESEARCH AND THEORY

It is possible that fraternal birth order has additional effects on development that are not sex-dimorphic in nature as well as effects that appear sex-dimorphic but

are not truly so. Such effects represent potential research directions in their own right, but they also represent possibilities for error or confusion in future work on the relation of fraternal birth order and sexual orientation. Some possible issues are considered in the closing section of this article.

### *IQ and Fluctuating Asymmetry*

A few studies have suggested that fraternal birth order increases the risk of mental retardation in later born males (Ackerman, Goolsby, and Paal, 1988; Flannery and Liederman, 1994; Lord, 1992). The available evidence is rather weak, but it is consistent with the finding that fraternal birth order is positively correlated with fluctuating asymmetry in adult subjects (Lalumière, Harris, and Rice, 1999). Fluctuating asymmetry is considered by many researchers to be evidence that the development of the fetus was perturbed by noxious factors in the prenatal environment (e.g., Leary and Allendorf, 1989). These findings underscore the possibility that fraternal birth order might have nonspecific effects on fetal development as well as a specific effect on sexual orientation. One would expect the correlation between the specific and nonspecific effects to be low, since they would be related only indirectly, but the correlation could be strong enough to affect the results of some investigations.

### *Birth Weight*

The mean birth weight of newborn girls is typically 100–150 g lower than that of newborn boys. Therefore the finding that prehomosexual males with older brothers weigh less at birth than preheterosexual males with older brothers might be interpreted as additional evidence that the former were feminized *in utero*. That interpretation would not be warranted at this time. The lower birth weight of homosexual males with older brothers might reflect some growth-inhibiting process that has nothing to do with the causes of the ordinary difference in mean birth weight between girls and boys.

### *2D:4D Ratio*

Williams *et al.* (2000) found that men with more older brothers tend to have shorter index fingers relative to their ring fingers (i.e., low 2D:4D ratios). The 2D:4D ratio is a sex-dimorphic character, with lower ratios typical of males. The sex difference is estab-

lished early in fetal development and is thought to result from the higher androgen levels produced by the male gonad. Williams *et al.* therefore hypothesized that male fetuses who have been preceded by other males in the same womb “see” more androgen during crucial stages of development. Their interpretation is consistent with other evidence that supports the emerging hypothesis that male homosexuality may result from hyper- rather than hypoandrogenization of the fetus (e.g., Bogaert and Hershberger, 1999; Lalumière, Blanchard, and Zucker, 2000; McFadden and Champlin, 2000; Robinson and Manning, 2000).

The reliability of the finding of Williams *et al.* (2000) has not yet been established. The only replication attempt known to the present author failed to confirm it (Robinson and Manning, 2000). Even if this intriguing finding does prove reliable, that would not necessarily mean that males with high fraternal birth orders see more androgen, or if they do, that the extra androgen is the reason for their increased likelihood of homosexuality.

In the first place, it is conceivable that male fetuses who have been preceded by prior male pregnancies “see” more of something, but what they see is anti-H-Y antibody, not testosterone. Flaherty, Zimmerman, and Wachtel (1979) found that cell surface changes involving H-Y and components of the major histocompatibility complex, changes that are induced by testosterone but not by estradiol, are also induced in the presence of anti-H-Y antibody. This may be physiologically relevant, because changes involving the major histocompatibility complex at the cell surface could have effects on embryonic development. The main point here is that anti-H-Y antibody might produce certain “faux” androgen effects.

In the second place, it is possible that male fetuses who follow prior male pregnancies do see more androgen. That extra androgen may not, however, represent the cause of homosexuality in later male fetuses but rather the fetuses’ attempt to resist it. It is possible, in other words, that male fetuses whose sexual differentiation is threatened by anti-H-Y antibody attempt to compensate by producing more testosterone. This last possibility also suggests a shift in perspective on the problem posed by homosexuality for evolutionary biology and evolutionary psychology. The correct question might not be why the multiparous mother starts producing essentially infertile sons, but rather what prevents her from producing more of them.

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