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**Homosexuality And Biological Factors:
Real Evidence -- None;
Misleading Interpretations: Plenty**

Dr. van den Aardweg explains why he believes the claims for a biological basis for SSA have little merit.

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In 1898, the Austrian empress Elizabeth was stabbed to death in Genova by 25-year-old Luigi Lucheni. The murderer was proud of his act, which he declared was “revenge for my life.” After turbulent years in prison, Lucheni hanged himself in 1910. A typical representative of the prevailing 19th century thinking on abnormal behavior, professor Mégevant performed an autopsy, investigating the brain to uncover the anomalies that were supposed to underlie the murderer’s “psychopathic disposition.” However, nothing out of the ordinary could be found; even Lucheni’s brain weight was standard. Disappointed, the professor put the head in a jar with formaldehyde and stored it in the cellar of the Institute for Forensic Medicine. A neuroanatomically normal psychopath, what a scientific riddle!

Yet the explanation of this criminal’s arrogant, merciless, and abnormally hostile personality was close at hand, provided one would pay attention to what he had to say himself about his psychological history. An illegitimate child, abandoned and cruelly abused and exploited by several foster “parents,” he was driven by frustration and embitterment. But psychogenesis had not been discovered by then, so to speak, and psychiatry was dominated by Kraepelin’s postulate: mental aberrations stem from abnormalities in the brain, which moreover are inherited. For criminal behavior, the variant was Lombroso’s theory of the deliquente nato, the born-that-way delinquent.

Perusing the research literature on homosexuality of the last 15-20 years, one recognizes the same 19th century mentality. The nonprofessional reader who is not able to read the rules will get the impression that there is no scientific doubt with respect to homosexuality’s biological causation; at least, that powerful constitutional predispositions have been ascertained. If you are not precisely born a homosexual, you will in any case possess some biological homosexual disposition, which in practice amounts to the same. And if science has not yet unearthed the definitive biological causes, it is in the process of doing so, because the experimental indications are piling up. So science would seem to support the notion of the omosessuale nato. [1]

By and large, this is the message conveyed by the majority of the reports in the professional magazines. If developmental-psychological factors are given some attention they are played down as of secondary importance at most; often no mention is made of them at all. Now what is the truth? First, that not a single genetic, physiological, anatomical, or neuroanatomical correlate

of homosexuality has been demonstrated. Secondly, that contrary to the impression they confer, precisely the studies of the last 15-20 years have made the existence of such correlates more unlikely than before. Thirdly, that these realities are either not perceived or purposely kept out of awareness because most academic publications on homosexuality are influenced or determined by the predominant gay ideology.

No Hormonal Correlates

The conclusion arrived at by Perloff in 1965 that no hormonal peculiarities had been demonstrated in homosexuals still holds today. In 1993, Byne and Parsons thus summarized their thorough expert analysis of the investigations on homosexuality and biologic factors, including hormones: "There is no evidence ... to substantiate a biologic theory." [2] And after 1993? Nothing remotely resembling proof of hormonal influences on homosexuality either. Yet a warmed-up version of the intersex (Zwischenstufen) theory of Magnus Hirschfeld, according to which male homosexuals have a hormone-induced feminized brain and lesbians a masculinized, continues being dished up as if founded in scientific fact. Prenatal androgen deficiency and excess (in homosexual men and women, respectively) are held responsible. [3] This view is however an undifferentiated programmatic sketch more than a testable theory. For what is meant, for instance, by a "feminized" male brain?

Does it mean that in some, as yet postulated, brain structure, the perceptual recognition center of "the feminine," the "image" of the female Gestalt has been substituted by the Gestalt of "the masculine"? That sounds rather fanciful (and what then caused the picture of the feminine in the homosexual pedophile to be substituted by that of "the boyish"? And so on for the other sexual "orientations"). Or does a "feminized" male brain mean that the boy's behavior is becoming feminized; or rather, that the boy's aggression drive is reduced, because lack of daring and of physical fighting spirit is much more tied to homosexuality than "femininity"? [4] In the latter case, the supposed brain anomaly contains nothing that spontaneously generates or inherently predisposes to homosexual desires. Reduced male aggression (and its counterpart, enhanced female aggression/tomboyishness) as a temperamental trait (the current term is "gender nonconformity") might then be considered at most an "indirectly predisposing," better still, a "pseudopredisposing" factor. In fact, it is the environment and the child's self-view which determine if such temperament plays a role in the genesis of homosexuality. In this variant of the sex-atypical brain theory, the origin of homosexuality itself is not accounted for; in principle it may be easily incorporated in a developmental-psychological view. It certainly does not justify the horrible notion of "gay children."

There would merely be temperamentally placid boys and "wild" girls, the vast majority of them growing up as normal heterosexuals.

The crucial question however is: What is the evidence for a link between this (or other) behavioral traits and prenatal, or whichever other, hormonal or brain irregularities? The alternative explanation, habit formation and self-view by rearing and other social influences, is certainly not less likely. Mama's boys and/or boys with "psychologically absent" fathers tend to be overdomesticated, so to speak, and it is precisely these parent-child factors that have incontestably been shown to be associated with male homosexuality. [5]

Fathers' girls and girls whose personality was not much shaped by their mother, and girls with other defeminizing childhood background factors may adopt more "masculine" or boy-like attitudes and habits. Anyhow, specific parent-child and peer group interactions have been amply demonstrated, while the hormonal-neuronal explanation has precious little to offer but speculations. There are no indications that homosexuals have suffered hormonal deviations before or after birth, their hormonal system is normal and in agreement with their biologic sex.

The evidence proposed by the proponents of the feminized/masculinized brain theory is limited to a few hardly relevant observations: the female lordosis reflex in male rats after testosterone deprivation (which reflex however is not indicative of their sexual drive); the possibly enhanced prevalence of lesbian tendencies in women suffering from congenital adrenal hyperplasia or CAH (who have been exposed to prenatal androgen hormones) [6]; and a few contradictory data regarding finger length ratios.

Regarding CAH, the majority of these women are heterosexual, so that their supposed brain masculinization would affect only a minority. If lesbianism would indeed be relatively frequent among these patients (the data are not conclusive [7]), it is hard to see why that would argue for a hormonal cause or even predisposition in healthy lesbians who are hormonally normal and whose genitals are not semi-masculinized like in these CAH patients. A psychological explanation of lesbianism in girls with "unfeminine" genitals and the various traumatic experiences associated with it is more realistic than a physiological explanation. For feelings of feminine inferiority are practically inevitable in girls who suffer from such a condition, and that is how a lesbian development often starts.

With respect to men with disturbances leading to prenatal androgen insensitivity or deficiency (and who are therefore believed to possess "feminized" brain centers), no connection with homosexuality has been found. [8] This has been the usual outcome of the older studies on homosexuality in persons who really have some aberration of the sex hormones or sex-chromosomes, too: they do not become psychosexually aberrant. According to some authors their sexuality may be somewhat rudimentary, "infantile," underdeveloped, though, and this is understandable. [10]

Do homosexuals have a 2D:4D (index finger: ring finger) ratio like the one typical of the opposite sex? It has been declared this "suggests" sex-atypical prenatal hormones and brain formation. But the phenomenon is in all likelihood no more than a peculiar artifact, like others of that kind, [11] so we had better forget about it.

In all, the periodically launched "promising" leads of hormonal correlates of homosexuality have invariably proven dead ends; there is a history of nearly 90 years to illustrate this point. It is at odds with scientific prudence to make the gigantic leap from (otherwise, not sufficiently studied) observations with rats to the complicated level of human sexuality. It is time the criticism of Byne (1995, p. 337) gets through to psychiatrists, psychologists and other professionals who sometimes tend to be overly impressed with reported biologic indications. Byne says there are too many

"...hasty interpretations, based on limited sample sizes, shaky methodologies, and extremely limited knowledge about functions of particular brain structures and even less knowledge about the biological substrates of the mind."

In other words, there is much amateur speculation instead of serious science. He explains:

“Attempts to prove that gay men have feminized gonatropin responses [12] were made decades after strong evidence suggested that the brain mechanism regulating the response does not differ between men and women” and “It required 25 studies to convince some that testosterone levels in adulthood do not reveal sexual orientation” (p. 336; see also Byne, 1997).

As long as a suspect’s guilt has not been proven, he must be treated as innocent. One may personally believe homosexual persons must have hormonal or neuroanatomical peculiarities, but scientifically there is no reason not to consider them physically normal and healthy (brain evidence: below).

No Genetic Proof

Despite numerous suggestions to the contrary, the last fifteen years of renewed research led even behavioral geneticists in favor of a genetic explanation of homosexuality to the conclusion that genetic factors for homosexual inclinations as such do not exist. This interesting fact hardly gets the attention it deserves. The other remarkable point is that in consequence, current genetic speculations focus on predisposing factors of a non-sexual nature. As a result, it is implicitly admitted that the prime and decisive causes lie in the person’s life history. The indirect evidence for these conclusions has come from twin studies, the direct from the exploration of genetic linkage.

Concordance percentages in volunteer studies vary from 25-66 for monozygotic (MZ) twins, roughly two times the percentages for dizygotics (DZ). [13] This is quite dissimilar from the picture in the case of uncontested genetic factors like the color of the eyes, certain diseases, etc. Apart from the fact that volunteer studies do not adequately represent the total population of homosexuals with twins (see further on), these results are not proof of the genetic determination of homosexuality. First, because only half of the co-twins of the MZ homosexual index persons in these groups were also homosexual. Secondly, because the average concordance of DZ male homosexuals in volunteer studies is 20%, whereas the rate of homosexuality among non-twin brothers of male homosexuals “hovered closely around 9%.” [14] DZ twin brothers of homosexuals are genetically not more similar than other brothers, so the finding that DZ twins of male homosexuals are twice as often homosexual as the average brother of a homosexual man challenges a genetic explanation. Both the higher concordance in MZ than in DZ pairs and the higher incidence in DZ twins as compared with non-twin siblings point to a psychological (environmental) explanation. Very regrettably, the psychological dimension has been virtually neglected in all of these studies, except for an occasional observation like the footnote by Bailey and Pillard (1995, note 34):

We found in both our male and female studies that discordant MZ twins also reported quite different childhood experiences. ... the homosexual twins reported more sex-atypical behavior... (“Sex-atypical behavior” is the concept of gender nonconformity we dealt with above).

Why did an observation like that did not lead to collecting detailed developmental-psychological data of these subjects of identical genetic make-up regarding their relationships with parents and

peers and self-image in relation to their co-twin? Anyhow, the observation of Bailey and Pillard is satisfactorily explained by the psychology of twins. Their self-view is shaped by intense comparison with their co-twin (and by their being compared to each other by their environment); either they feel “identical” (want to be and act like their alter ego) or they overemphasize their differences, e.g., with respect to their virility or femininity. [15] Thirdly, 11% of adoptive brothers of homosexual males are reported to be homosexual, too. [16] This finding, which neither genetic nor perinatal hormones can account for, casts more than a little doubt on the genetic explanation of the homosexuality of the biological sons, thus on the whole genetic hypothesis.

However, concordance rates in volunteer samples appear to be inflated, since homosexuality-concordant twins, especially MZ twins, are as a rule overrepresented. [17] Therefore, samples from twin registers are considered more representative. [18] Bailey et al. (2000) found 3 out of 27 MZ male homosexuals from the Australian twin register to be concordant (11%), versus 0 out of 16 same-sex dizygotics (0%) and 2 of 19 opposite-sex dizygotics (12%). Of 22 female MZ twins, 3 (14%) were concordant, versus 0 of 16 same-sex dizygotics (0%) and 2 of 19 opposite-sex dizygotics (12%). This was no “statistically significant support for the importance of genetic factors,” which the reader who inspects the simple numbers given above may readily see. Significantly, though, it has subsequently been attempted to squeeze as much “heredity” as possible out of these obvious data by applying more “flexible” (and thus more debatable) criteria for “homosexuality” and using a “heredibility” formula.

And, lo!, the magic formula turns the defeat for the genetic explanation into a victory so that henceforth what was evidently “no support for genetic factors” can be sold as modest “support” (Kirk et al., 2000)! Such handling of the raw numbers borders on what the French call “statistical massage”; it is at any rate no test of the power of a genetic versus a non-genetic model. [19] The same is true of the interpretation in a similar study that the “[homo] sexual orientation was substantially influenced by genetic factors.” [20]

In this case too, the simple numbers tell the tale better than sophisticated calculations based on a speculative model [21]: Two of 10 MZ homosexual men had a homosexual twin brother (20%) vs. 4 of a combined group of 28 male DZ twin pairs and pairs of non-twin brothers one of whom was a homosexual (14%). Four of 9 female MZ pairs were concordant (44%) vs. 8 of a combined group of 28 female DZ twins plus non-twin sisters one of whom was a lesbian (29%). This indicates a slight preponderance of MZ concordance, not significant statistically though. In a non-random sample of never-married twins from the Minnesota Twin Registry, which seems to contain the majority of the twins of this State, Hershberger (1997) found hereditability coefficients that were mildly consistent with genetic influences for lesbians, not for male homosexuals. [22]

In sum, MZ concordance becomes lower the more representative the samples; at the same time, the difference between MZ and DZ concordances becomes less convincing. [23] The more important conclusion, however, is that the genetic hypothesis has become increasingly less plausible and seems engaged in a rearguard action. For no theorists of genetic influences can be found any more who believe in the existence of a “gay gene” proper. The view of the role of genes underwent a silent, but very significant change: no longer the prime determinants, they now function at most as predisposing factors. In short, the decisive cause(s) of homosexuality are not hereditary. Even Hamer, the man who in 1993 caused the media stir with his “near-discovery” of the gay gene [24] admits:

We do not expect to find (in the future) a gene that is the same in every gay man ... just one that is correlated to sexual orientation. [25]

Unclearly as it is worded, he seems to hint at predisposing factors. Bailey theorizes in the same direction after finding that childhood gender nonconformity was (to a degree) compatible with a genetic statistical model while homosexual feelings were not. [26] But the case for the genetic origin of gender nonconformity is far from strong either. Wasn't it Bailey himself who previously had noticed that it was this very item of gender nonconformity which distinguished the homosexual from the heterosexual twin in MZ pairs discordant for homosexuality? [27]

Dramatically decreasing genetic evidence from modern twin research was on the one side, while on the other, the search for a genetic linkage came to a dead end. The well-known 1993 finding of Hamer, et al., did indeed not demonstrate the existence of a single gene, because it was not shown that the highly selective group of homosexual men showing a moderate correlation between DNA markers and a region of the X chromosome shared a particular molecular sequence. [28] The supposed genetic factor thus might have been any physical or temperamental resemblance with the mother (from whom the X chromosome is inherited). The whole thing was, after all, a storm in a tea cup. Subsequent analysis and research vindicated the verdict by the famous French authority in the field, Jerome Lejeune, that the methodological defects of the investigation were so serious that "were it not for the fact that this study is about homosexuality, it would probably never have been accepted for publication." [29]

A first replication by the same team with a small group reported a barely significant confirmation for homosexual men, not for lesbians [30]; the calculations of the team were, however, rejected by the statistician experts. [31] And an independent Canadian team failed to uncover a link between male homosexuality and the X chromosome in a larger sample. [32] So much for the direct exploration of the genes. Circumstantial evidence is sometimes deduced from familial and pedigree findings. It has long been known that homosexuality occurs relatively more frequently in certain families and pedigrees, but genetic explanations are implausible in view of the erratic way it is distributed within these families and pedigrees: "We never found a single family in which homosexuality was distributed in the obvious pattern that Mendel observed." [33]

And this statement by Hamer is even an understatement. On the aforementioned higher correlation in lesbian propensities between lesbians and their mothers than between them and their sisters, [34] he comments: "The rate was a whopping 33 percent, meaning that the daughter of a lesbian had a one-in-three chance of also being a lesbian. Genetically speaking, this result was impossible." [35] Psychologically not so, however. [36] Many specific personality-shaping habits are transmitted from one generation to the next by learning. This may explain varied familial phenomena a genetic hypothesis cannot. It is therefore arbitrary to present a possibly somewhat elevated occurrence of male homosexuality among maternal relatives as evidence for genetic influences, as has been done in a recent publication [37] (Fortunately the authors admit that it is "still possible" to attribute their data to "culturally, rather than genetically, inherited traits"). [38]

In an attempt to present the long known [39] and recently well-replicated [40] phenomenon that homosexual men (not women) have relatively more older brothers than heterosexual men as an indication of the biological cause of male homosexuality, a far-fetched theory has been invented. Mothers of male homosexuals might progressively produce an "antibody" to male fetuses every

time they are pregnant with a boy, which in turn would eventually feminize the developing brain of the younger male embryos (The theory has only relevance for 15% of the male homosexuals, viz., those with more older brothers). [41] Physiological anti-boy mechanisms have never been demonstrated, however, and the fully speculative status of the feminized male brain has already been described. Why not try a psychological explanation? Already in 1937 psychiatry professor Schultz pointed to the impact of the position of the “nice little brother” (liebe Brüderchen) among his older brothers on his psychosexual development. [42]

No Neuroanatomical Correlates

Like professor Mégevant a century ago, present-time brain researchers have never really been awarded in their quest for unambiguous brain anomalies in homosexuals. E.g., an initial report of larger inter-hemispheric fiber bundles in homosexual men could not be replicated. [43] Nor was there a convincing reason to explain LeVay’s 1991 over-publicized observation of a smaller hypothalamic nucleus (INAH3) in some homosexual men who had died of AIDS in comparison with heterosexual intravenous drug users as evidence of a feminized brain center. Differences between the groups other than the homosexuality variable might have caused the effect: procedures of tissue preparation, length of the disease period, previous occurrence of other venereal diseases, or medication. A replication by Byne et al. (2001), hailed by some as “proof” of a “homosexual brain center” [44] has in fact made that explanation even more unlikely. In a small group of homosexual men who had died of AIDS they found a trend for the ratio of INAH3-volume to brain weight to be smaller than that ratio for deceased heterosexual men who were drug users. The trend was not significant statistically, hence strictly speaking, the difference is not uncontestable. Byne suspects that since the brain weights of the heterosexual men with AIDS were much lower than both those of the HIV-negative heterosexual men and the homosexuals with AIDS, the trend,

“... may reflect the superior health care received by the homosexual male group compared to the heterosexual male group with AIDS, all of whom were intravenous drug users.” [45]

Nor does he exclude that histological preparation caused the relative shrinkage of INAH3 in the homosexuals:

“Since some New York hospitals have a preponderance of HIV+ patients who are gay men, while others have a preponderance of HIV+ patients who are drug users, the homosexual and heterosexual patients tended to come from different institutions, and therefore, there were likely variations in autopsy and fixation procedures that were confounded with sexual orientation.”

For these reasons, he believes his second finding is the more reliable and important one: the nuclei of the homosexuals contained as many neurons as those of the heterosexual men. That is, 60% more neurons than the female nucleus. This is the more interesting because INAH3 seems the only brain-anatomical structure which is sexually dimorphic. [46] In sum: no evidence for the “wrongly put on nerves” (like the strings of a guitar) the poet Dante ascribed to homosexuals! [47]

Conclusions

The main conclusion is obvious if we keep our eyes on the interesting factual observations in the reports of the last few decades and let our sight not be obscured by the biology-biased interpretations they are wrapped in. No bodily correlates of homosexuality have been demonstrated. Like with the monster of Loch Ness, there are periodic claims that a biologic factor has been spotted, but upon closer inspection, the claims evaporate. [48] This renders any discussion of whether a determinate correlate would be a cause, an effect, or an insignificant byproduct of another homosexuality-connected variable superfluous.

But there is more. Whereas constitutional theories seem increasingly speculative, they are only the psychological correlates of homosexuality that are well-established. The highest correlations have systematically been found for what is currently designated as childhood and adolescent gender nonconformity: lack of integration in the boyhood/girlhood world and feelings of not belonging to the same-sex world. [49]

This syndrome has been established in clinical as well as nonclinical samples, in various countries and over several generations. Significantly, it is also recognized by authors who prefer to believe in biological theories (Hamer, LeVay, Bailey). The second-highest correlations exist with the finding of defective relations with the same-sex parent; the third-highest with maternal dominance/overprotection for the homosexual man, and with varied father factors for the lesbian. [50] Empirically, then, a psychological explanation is the most realistic.

Furthermore, belief in a causal contribution of some (mostly unspecified) biologic variable, which is shared by many professionals who view homosexuality basically as a psychological phenomenon, is purely hypothetical. I think Schultz-Hencke, one of the coryphées of German psychiatry, was right when he wrote as far back as 1932: Homosexuality and every correlate of it is “psychologically explicable, without leaving a remainder.” [51] Even the unboyishness of many prehomosexual boys may rather be seen as an effect of intra-family factors, habit formation, and self-concept than as temperamental. [52] And certainly is all talk of “gay children” irresponsible, not only morally, but also scientifically. There is nothing intrinsically “gay” in either the biological or the psychological nature of children, nothing that spontaneously would push them to homoerotic feelings. The theoretical improbability of the existence of physiological correlates specific for homosexuality may appear more clearly if homosexual and heterosexual pedophilia, transvestism, exhibitionism etc. are taken into account (curiously, this is almost never done). For either specific hormonal, hormonal-brain or other factors are postulated for each of them, or they are regarded as “environmentally” caused. The first option is wild, the second challenges the biologic co-causation of homosexuality, because on what grounds should homosexuality be the exception, since the desires of pedophiles, etc. have the same characteristics as those of homosexuals (exclusiveness, obsessiveness)?

Proven Psychological Variables Ignored

Methodologically, it is a pity that most of the reviewed studies did not include the psychological variables of proven validity as to their relation with homosexuality. The more so since their results are mostly used as arguments for a (biologic) theory. But what is the value of a theory based on research which left out some of the most important variables? Notably the various collections of MZ and DZ twins might have yielded rich data had thorough psychological examinations been conducted of the childhood/ adolescence background, parental and peer factors, self-

view, and neurotic emotionality. [53] That is equally true of studies on familial or pedigree clustering and the more-brothers phenomenon in a subgroup of male homosexuals. This missed opportunity points to either ignorance of the psychology of homosexuality or unwillingness to give it the credit it deserves (or both).

Gay Activists Dominate Research

Whence this 19th century step-motherly treatment of psychology by our present-day professors Mégevant? It is because with few exceptions they are gay persons wedded to the gay ideology. They are the Weinbergs, LeVays, Hamers, Baileys, Hershbergers etc., who openly admitted that biological roots of homosexuality favor social acceptance of the gay agenda (and right they are). It is in their interest to be single-mindedly biology-biased. And since the gay ideology has become the party line in the official establishment of the human sciences, inclusive of most professional journals, all findings “support” homosexuality’s biologic origin and mental normality or at least “suggest” it. Free research and free thinking is taboo as soon as it seems to threaten the gay cause. The ideologically distorted science thus produced and sponsored profoundly misleads the public. On a deeper level, it is often motivated not by thirst for the truth, but by the wish to rationalize or justify the normality sought by so many persons who are committed to a sexually abnormal lifestyle.

End Notes

1. This misrepresentation of the present state of research is imitated by not a few authors who apparently accept it without critical examination. A painful example is the contention of Serra (2004) that there would be “a coherent complex of observations indicating with sufficient strength that ... a (causal) biological component may not be excluded and which even suggest that this has an appreciable weight” (p. 232). That boils down to suggesting the existence of the omosessuale nato, though Serra’s formulation is vague. I mention this example because Father Serra is a retired professor of genetics of the Gregoriana University in Rome and a honorary member of the Papal Academy for Life. His misleading article in the Jesuit periodical *La Civiltà Cattolica* will probably make some impression in certain Catholic circles.
2. P. 228. Unlike the authors who blithely dream up physiological “explanations” without solid expertise in this area, Byne is an authority in the field of psychiatric neuroanatomy, Parsons in psychiatric genetics (both at the New York institute of Psychiatry).
3. E.g., Mustanski et al., 2002; Hershberger & Segal, 2004. They quote Meyer-Bahlburg (2001) although this author gives no evidence on hormonal or brain peculiarities of homosexuals, only on the psychosexual development of women with a chromosomal disturbance (classic CAH). According to some (not all) studies they manifest more lesbian inclinations than other women; yet their “prenatal hormonal milieu does not dictate a bisexual or lesbian outcome” and “few consider themselves lesbians” (p. 163).
4. Research data: van den Aardweg, 1986, chpt. 15; Freund & Blanchard, 1987; Hockenberry & Billingham, 1987.
5. As for the habit-formation explanation of boyish aggressiveness and daring or the lack of it, a comparison of the behavior of boys from families of working men with boys from academic

families is instructive. Boys from the latter families are generally “softer,” more “feminine” if we prefer this psychological term, less physically aggressive. Also, compare boys from slums with boys from middle-class families.

6. Meyer-Bahlburg, 2001. Byne & Parsons (1993) make it clear how unconvincing the masculinized-brain hypothesis is to account for this otherwise not conclusively demonstrated phenomenon (p. 232).

7. See note 2, above.

8. Byne & Parsons, 1993, p. 232.

9. E.g., the older study of Raboch & Nedoma, 1958.

10. Züblin, 1957. Interestingly, Züblin remarked that the weak sexuality of these physically abnormal men seems strongly determined by their need to “behave like other men.” Meyer-Bahlburg (2001) points to the rudimentary sexual drive of women with CAH.

11. Mustanski et al., 2002.

12. Gonadotropins: hormones working on the sexual glands. Feminized gonadotropin responses: responses comparable to those of the female physiological cycle.

13. Of 56 American male MZ pairs, 59 (29%) were concordant, against 12 (22%) of 54 DZ pairs (Bailey & Pillard, 1991); of 20 British male and female MZ pairs, 5 (25%) were concordant, against 3 (12%) of 25 DZ pairs. The difference was not significant (King & McDonald, 1992). Of 38 American male MZ pairs, 25 (66%) were concordant, against 7 (30%) of 23 DZ pairs (Whitam et al., 1993). Of 71 American female MZ pairs, 34 (48%) were concordant, against 6 (16%) of 37 DZ pairs (Bailey et al., 1993).

14. Bailey & Pillard, 1995, p. 136.

15. I know a few such cases. The homosexual twin of these MZ pairs had viewed himself (and was seen by his parents) as the weaker of the two or was mother’s boy (the other one, father’s boy). Farber (1981) described two MZ sisters reared apart, one of whom a lesbian, the other heterosexual. In contrast with her co-twin, the lesbian had a conflict-ridden relation with her foster mother and a strong attachment to her foster-father, whom she imitated. Psychology gives the clues!

16. Bailey & Pillard, 1995, note 30. Homosexuality seems to be relatively frequent in adoptive children in general, which has to do with many of those children’s liability to feeling not belonging (less valuable) in comparison with their biological siblings.

17. The phenomenon of “concordance-dependent ascertainment bias,” which was responsible for the suspect 100% MZ concordance (against 11.5% DZ concordance; or, under a broader definition of homosexuality, 42.3% DZ concordance) in the male group of Kallmann (1952). The figure of Kallmann raises some questions, by the way. A favorite disciple of psychiatrist Ernst Rüdin, the highest Nazi authority on the medical aspects of “racial hygiene” and a zealous advo-

cate of forced sterilization of the mentally disturbed and “psychopaths,” Kallmann, like Rüdin, saw twin research as a means to improve the diagnosis of family members of “racially inferior” persons. He called for the sterilization of schizophrenics and many of their seemingly healthy family members who allegedly carried the postulated sick recessive gene, estimating that this made necessary the sterilization of about 5% of the population (!). Probably not by coincidence, he found extremely high concordance rates for MZ schizophrenics. What did he originally, before his flight to the U.S., have in mind for homosexuals? (Müller-Hill, 1984; Blondet, 1995).

18. It is not clear, though, how representative because the volunteer effect cannot be ruled out. Only about half of the twins invited for the study eventually participated. In addition, the register itself is a volunteer register which may contain no more than 10-20% of the Australian MZ and DZ twins (Kirk et al., 2000, note 39).

19. Heritability formulas are statistics to estimate the part of score variance that might fit a proposed heredity model. Besides being based on assumptions which are susceptible of debate, heritability coefficients are not measurements of genetic influence, merely quantifications of the degree obtained observations are compatible with a postulated genetic model. It does not really enhance the plausibility of heritability coefficients for personality traits that according to their reckonings viewpoints on the death penalty, abortion on demand, and even a virtue like “humility” are “50%” genetically determined (Excellent analyses: Whitehead & Whitehead, 1999). *Another source of confusion flows from the use of proband-wise concordance percentages in stead of the usual pair-wise percentages. The proband formula overestimates “real” concordance, yielding genetically-biased results. Proband-wise formula: $2(++): [2(++)+-] \times 100\%$; pair-wise formula: $(++) : N \times 100\%$.

20. Kendler et al., 2000, p. 1843. The sample came from a U.S. national survey, but is not a representative of homosexuals with twins, nor can the volunteer factor be excluded.

21. The authors use the proband-wise concordance formula, overestimating MZ twin resemblance; in this text, pair-wise percentages are given.

22. With reference to this “moderate consistency” with a genetic model, see the contradictory finding of Pattatucci and Hamer (1995) that the highest correlation concerning lesbian interests was not between the lesbians and her sisters, but between the lesbians and their mothers. See also the failure of Hu et al. (1995) to find markers for a gene for lesbianism.

23. We cannot rule out the hypothesis that MZ concordance for homosexuality (and for other features) in former days was indeed higher than at present. It may be that the MZ children of former generations were more than at present reared and viewed as being identical, whereas MZ children of recent generations are more treated as distinctive individuals, their differences being emphasized in stead of their similarities. Examination of the relative proportions of MZ and DZ twins in non-Western cultures might help clarifying this issue.

24. Hamer et al., 1993.

25. Hamer & Copeland, 1994, p. 198.

26. Bailey et al., 2000.

27. Bailey & Pillard, 1995, footnote 34.
28. Byne, 1994.
29. Lejeune wrote this to me (1993) in response to my question about his opinion on Hamer's article in Science. Lejeune was a great and erudite scientist, the discoverer of the gene causing Down syndrome.
30. Hu et al., 1995.
31. Risch et al., 1993.
32. Rice et al., 1999.
33. Hamer & Copeland, 1994, note 47.
34. Pattatucci & Hamer, 1995.
35. Hamer & Copeland, 1994, p. 191.
36. The finding must be repeated before it can be generalized. It is certainly relevant in connection with the debate on parenting and adoption by lesbian couples.
37. Camperio-Ciani et al., 2004. This is a rather shoddy study. "Measurement" of the homosexual inclinations of the relatives consisted in the opinion of the interviewed homosexuals themselves (The tendency of self-defensive homosexuals to project homosexuality in others is a well-known phenomenon). Besides, the informants were volunteers, so that the results may be an artifact. Otherwise, the authors emphasize that only 20% of the variance of pedigree sexual orientation could be accounted for by the genetic hypothesis.
38. Ibidem, p. 2220. "Culturally inherited" sounds strange. Why not: "Transferred by habits of rearing and education"? For example, male-female role imbalances which clearly stem from habit can be observed in certain families; maternal overprotection can sometimes be traced back for several generations, not to speak of personality shaping world views or beliefs.
39. E.g., the study of Lang, 1936.
40. Bogaert, 2003. Statistically, the probability that a boy in certain families with more brothers becomes a homosexual increases 38% with each older brother. In view of the increasing rarity of families with a series of brothers, this familial factor will have affected few future homosexuals in Western society.
41. Bogaert, 2003. Bogaert's 15% nicely accords with that of Lang, 1936, who estimated 10-20%.
42. Homosexual men with more brothers not seldom felt inferior to them, were more overprotected, treated in a softer way.
43. Lasco et al., 2002.

44. In his book, Bailey (2003) misunderstood a communication of Byne to him as a confirmation of LeVay's finding. He euphorically writes he would like to invest big money in Byne's research (if he had it, of course), probably in the hope that this scientist will come up with the ardently desired biological proof. The scientific quality of Byne's publications indicates that funding him is not a bad idea, indeed, but: will the outcome make Bailey cheerful?

45. Letter of July 20, 2005, to this author. The next quote is from this letter, too.

46. Byne et al., 2001, p. 90.

47. *Inferno*, XV, verse 114: *li mal protesi nervi*.

48. One of the recent one-day butterflies: the Swedish discovery of feminized body odor preferences of homosexual men. Evidence for a genetic cause of homosexuality, or for the sense of humor of the authors?

49. A survey of the studies until the eighties: van den Aardweg, 1986, Table 13.1; for later studies: e.g., Bem, 1996.

50. van den Aardweg, 1986, Table 15.1 and 27.5; Fisher & Greenberg, 1996, p. 137.

51. "restlos psychologisch erklärbar" (p. 300).

52. The analysis of the evidence concerning the specific "femininity" or nonaggressiveness in prehomosexual boys and "masculine" tendencies in some prelesbian girls is a chapter in itself. Here I can merely state my conclusion.

53. Earlier in the text I recalled Bailey's observation that homosexuality-discordant MZ male twins differed in boyhood gender nonconformity.

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