Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity

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The Darwinian paradox of male homosexuality in humans is examined, i.e. if male homosexuality has a genetic component and homosexuals reproduce less than heterosexuals, then why is this trait maintained in the population? In a sample of 98 homosexual and 100 heterosexual men and their relatives (a total of over 4600 individuals), we found that female maternal relatives of homosexuals have higher fecundity than female maternal relatives of heterosexuals and that this difference is not found in female paternal relatives. The study confirms previous reports, in particular that homosexuals have more maternal than paternal male homosexual relatives, that homosexual males are more often later-born than first-born and that they have more older brothers than older sisters. We discuss the findings and their implications for current research on male homosexuality.

Keywords: fecundity; male homosexuality; pedigree analysis; reproductive success

1. INTRODUCTION

The determinants of homosexuality are often the topic of heated discussion, partly because of alleged moral implications, but also because of a genuine difficulty in explaining how genetic factors that lower male fecundity survive natural selection (Moran 1972; Bell & Weinberg 1978). Data are often the missing element in such discussions. Here, we provide novel empirical data on the fecundities of the families of homosexuals and heterosexuals, together with findings regarding possible determinants of human male homosexuality. We also show that the data contradict a number of hypotheses about the genetic basis of homosexuality. It should be noted that, in this argument, all researchers do not assume that homosexuals never reproduce but that they reduce, on average, their direct fitness.

At present, two lines of evidence point to genetic factors being partially associated with human male homosexuality. The first line comes from studies of the familiarity of homosexuality. Family studies of brothers and twins report that homosexuality is more common in brothers of homosexual subjects (Bailey & Zucker 1995). Hamer and co-workers (Hamer *et al.* 1993; Hamer & Copeland 1995), using pedigree analysis, have shown an elevated homosexuality rate in the maternal line of homosexuals. This finding has been taken to suggest the existence of X-linked genetic factors favouring male homosexuality. DNA linkage analyses, performed on homosexual brothers, have localized a candidate factor on the Xq28, a distal region of the short arm of the X-chromosome (Hamer *et al.* 1993; Hamer & Copeland 1995). These findings, however, have been difficult to replicate (Bailey *et al.* 1999; Rice *et al.* 1999).

The second, independent, line of evidence comes from studies of sexual differentiation of the brain during foetal life, which found that, in men, homosexual orientation correlates with late birth order and an excess of older brothers (Blanchard & Klassen 1996; Blanchard 1997). Blanchard (1997) hypothesized that late birth order and sibling sex ratio reflected the progressive immunization of some mothers to H–Y antigen, presented by the male foetus. After a number of pregnancies with male foetuses, the increasing effect of this maternal immunity reaction, according to the hypothesis, should reduce the sexual differentiation of the brain in succeeding male foetuses (Blanchard 1997). Blanchard *et al.* (1997) suggest that each additional older brother increases the odds of homosexuality in the next male born by *ca.* 33%.

The primary aim of our work was to replicate the above findings. Note that the two lines of evidence are not mutually exclusive. Therefore, we investigated both hypotheses concomitantly, using a family-tree analysis to compare families of homosexual and heterosexual males.

We also sought to analyse the possible persistence of genetic factors favouring homosexuality, which contradicts the expectation that natural selection would eliminate such factors. This Darwinian paradox has been the topic of several studies, but is still unresolved. Wilson (1975), attempting to solve the paradox, suggested a possible role for homosexuals as helpers in their families, increasing the fitnesses of their relatives and, thus, balancing their reduced direct fitness. However, both Muscarella (2000) and Bobrow & Bailey (2001) in recent empirical studies, showed that homosexuals do not act as helpers and do not give more financial or emotional resources to siblings than do heterosexual men. Other studies by Ř. Trivers (personal communication), Rice (1998) and Miller (2000) have suggested the partial penetrance of a candidate homosexual genetic factor and hypothesized that pleiotropic or sexually antagonistic effects, acting on personality, increase fecundity in non-homosexual carriers, thereby balancing the reduction in homosexual fecundity (Miller 2000). No supportive evidence or empirical data, however, has yet been shown.

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Table 1. Estimated distribution of male homosexuals in the families of study subjects.

	maternal line		paternal line	
	N	homosexuals	N	homosexuals
male relatives of homosexual subjects	396	22[a]	593	12
male relatives of heterosexual subjects	370	0[b]	604	8

[a] Homosexual male relatives of homosexual subjects: expected (based on the paternal line) $(12/593 \times 396) = 8.01$; observed $= 22$; $\chi^2 = 8.92$; d.f. $= 1$; $p < 0.005$.
[b] Homosexual male relatives of heterosexual subjects: expected (based on the paternal line) $(8/604 \times 370) = 4.9$; observed $= 0$; $\chi^2 = 3.45$; d.f. $= 1$; not significant.

2. METHODS

We asked 98 homosexual and 100 heterosexual men, living in northern Italy, to fill in an anonymous questionnaire, in a private setting, following written instructions. Subjects reported their own sexual orientation and provided demographic information and sexual-orientation data on their siblings, first cousins, parents, aunts, uncles and grandparents.

Homosexual subjects were sampled using the targeted sampling methodology of Watters & Biernacki (1989) for accessing 'hidden' populations. Subjects were recruited from three associations for homosexual men and two discotheques. For comparison, the heterosexual control group was sampled from two after-work clubs and two discotheques, located in the same geographical region, controlling further for age of subjects. Sampling occurred at different times to ensure that subjects with different habits were represented. The tendency to over-sample larger families, which could have two or more homosexual siblings, was controlled by asking the composition of the family and the estimates were corrected by weighting the units with the inverse of their probability of selection. More generally, estimates referring to populations of units different from the survey units (i.e. the population of the grandmothers of the interviewed subjects) were corrected, when necessary, by weighting the units with the inverse of their probability of selection in the sample.

Accessing a 'hidden' population is difficult because no sampling frame exists and public acknowledgement of membership could be potentially prejudicial to the subjects; therefore, standard probabilistic sampling methods produce low response rates and unreliable responses (Heckathorn 1997).

Targeted sampling is a widely employed method for accessing hidden populations, in preference to snowball sampling (Goodman 1961) and other examples of chain-referral. All these procedures introduce well-documented biases (Spreen & Marius 1992), which we were aware of. We considered that these biases would have limited relevancy in this study, and our procedures were unlikely to be associated with such biases, owing to the fact that we accessed only demographic variables (such as number of siblings, uncles, etc.). Moreover, the control sample was selected, using the same methodology, to reproduce the same possible biases and to guarantee the internal validity of the comparisons.

The questionnaire was designed to yield the following information: size of the aforementioned relative classes, birth order and sex of siblings, and sexual orientation of all male relatives. The questionnaires showed that the two subject groups did not differ in age (mean homosexuals, 33.22 years; mean heterosexuals, 33.06 years; two-tailed Mann–Whitney tests $p = 0.903$), education ($p = 0.655$) or professional status ($p = 0.989$). The information provided was considered trustworthy owing to the anonymity of the questionnaire, its simplicity (e.g. recalling numbers of brothers and first cousins) and the lack of emotionally laden questions. Sexual orientation was self-reported, and confirmed by answers to five questions from the seven-point Kinsey scale on sexual self-identification, fantasy, attraction, imagination and personal behaviour (Kinsey *et al.* 1948).

Subject age was self-reported; level of education was in accordance with the Italian scholastic system; profession was separated into eight categories, devised by us. Six subjects reported information on parents and siblings only.

The homosexual frequency on the maternal line of homosexual subjects was compared with the expected frequency on the corresponding paternal line (the observed rate of homosexuality in paternal line multiplied by the number of relatives in the material line). This was preferred to a comparison with the relative frequency for the whole population, the estimate of which, in Italy, is provisional and unreliable (Barbaglio & Colombo 2001); in fact, population estimates are based only on those individuals that have declared their sexual orientation in ersus forms. Furthermore, the whole country population rate is not adequate for comparison with the rate of geographically restricted area.

3. RESULTS

According to subjects' reports, homosexuals have more homosexual relatives on the maternal than on the paternal pedigree line; families of heterosexuals do not share this feature (table 1).

In table 1, it should be noted that the higher total number (N) in the paternal line than in the maternal line naturally emerges from the following. (i) The presence of one more kin class in the paternal line (sons of paternal uncles); while in the maternal line the corresponding kin class (sons of maternal uncles) could not be included because the members of this kin class do not share the X chromosome with our subjects. (ii) The fact that the number of fathers classified in the paternal line is higher than the number of brothers classified in the maternal line, as expected in a population with low general fecundity, such as the studied population from northern Italy. We also included fathers in the analysis, as they could, in principle, be homosexual; homosexuals are reproductive and can transmit genetic factors (Moran 1972; Bell & Weinberg 1978).

It should also be noted that we did not compare homosexuals' and heterosexuals' estimates, since homosexuals may have reported a higher frequency of homosexuality among their relatives (Bailey *et al.* 1999). It is unknown whether homosexuals over-estimate, heterosexuals under-estimate, or both biases occur.

Regarding the relationship between birth order and sexual orientation, we found: (i) a birth-order distribution of

Table 2. Reported fecundities of subjects' relatives from the maternal and paternal lines (*p*-Value calculated using the Mann–Whitney test; n.s., not significant.)

relative class	likelihood of sharing X chromosome	homosexuals			heterosexuals			
		N	mean fecundity	s.d.	*N*	mean fecundity	s.d.	*p*
mothers	1	98	2.69	1.30	100	2.32	1.05	0.02
first-borns' mothers	1	32	1.94	0.88	52	1.77	0.61	n.s.
maternal aunts	0.75	95	1.98	0.98	121	1.51	0.97	0.001
maternal uncles	0.25	114	1.75	0.91	117	1.73	0.94	n.s.
maternal grandparents	0.5	91	3.55	2.57	100	3.39	1.85	n.s.
sons and daughters of maternal grandparents[a]	0.25–1	307	2.17	0.85	338	1.83	0.72	0.001
paternal aunts	0	111	1.75	1.07	129	1.94	1.13	n.s.
paternal uncles	0	134	1.75	0.95	135	1.67	0.94	n.s.
paternal grandparents	0	88	4.03	2.72	99	3.81	2.43	n.s.
sons and daughters of paternal grandparents (excluding father)[b]	0	239	1.77	0.85	264	1.80	0.89	n.s.

[a] Cumulative fecundity of mothers, maternal aunts and maternal uncles.
[b] Cumulative fecundity of paternal aunts and paternal uncles.

homosexuals that was significantly different (less first borns, more second, third and fourth borns) from that of heterosexuals, both in our sample ($\chi^2 = 9.89$, d.f. $= 3$, $p < 0.05$) and in the whole population of northern Italy ($\chi^2 = 11.03$, d.f. $= 3$, $p < 0.02$), as reported in census data from the Italian Bureau of Statistics (ISTAT 1951–1981); (ii) an excess of males among older siblings in homosexuals (observed: 69 brothers, 45 sisters; expected: 57 brothers, 57 sisters; $\chi^2 = 5.06$, d.f. $= 1$, $p < 0.05$).

We then estimated the importances of the excess of homosexuals in the maternal line and the excess of older brothers and other possible biological factors. A multiple linear regression and a logistic regression are carried out, using a stepwise selection method, to identify biological factors that might explain the observed variability in sexual orientation, measured on the seven-point Kinsey scale (Kinsey *et al.* 1948) and then dichotomized. Both models selected the same factors; therefore, only outcomes from linear regression are reported. The first predictor, selected by the stepwise method, was 'number of homosexual relatives in maternal line', accounting for 14% of the variance in sexual orientation (beta $= -0.489$, $R^2 = 0.14$ ($p < 0.001$), partial $R^2 = 0.12$). The second predictor selected, 'number of previous male brothers', explains 6.7% of the residual variance (beta $= -0.141$, $R^2 = 0.067$ ($p = 0.001$), partial $R^2 = 0.078$). Predictors not selected were: number of homosexual relatives in the paternal line, birth order, total number of brothers and age. The above model explains a limited 20% of the sexual-orientation variance ($R^2 = 0.207$, corrected $R^2 = 0.195$).

The most intriguing result derives from the pedigree analysis of fecundity. Table 2 shows that maternal relatives of homosexuals have higher fecundity than maternal relatives of heterosexuals; this difference does not appear in the paternal line.

Especially noteworthy are the differences between the groups formed by maternal aunts and mothers of homosexuals and heterosexuals. A potential confounding variable is that, owing to male birth-order effects, homosexuals may belong, on average, to slightly larger families than

heterosexuals. Excluding mothers of non-first-born homosexuals from the analysis is a conservative way of controlling for this effect (conservative because birth order is only tenuously associated with homosexuality; see above).

Fecundities of grandparents of homosexuals and heterosexuals are not significantly different, whereas the difference in the fecundities of sons and daughters of maternal grandparents is highly significant (table 2).

4. DISCUSSION

The results confirm the existence of previously proposed biological predictors that partly explain male homosexuality: (i) the prevalence of homosexuals in the maternal line, suggesting possible genetic factors in the X-chromosome (Hamer *et al.* 1993); and (ii) the number of older brothers of homosexuals, suggesting a possible maternal immunization effect (Blanchard & Klassen 1996; Blanchard 1997). Stepwise regression analysis shows that both variables have a limited effect, explaining *ca.* 20% of the variance in sexual orientation; the maternal effect, however, is weighted relatively greater (14%) than the number of older brothers (6.7%). The fact that birth order and total number of brothers did not enter the regression means that these variables are already explained by the number of older brothers.

Another result is that females in the maternal lines of homosexuals are significantly more fecund than those in the maternal lines of heterosexuals. No significant differences are found, however, when comparing paternal relatives of homosexuals and heterosexuals. It should be noted that higher fecundity in mothers of homosexuals is predicted by the immunization hypothesis of Blanchard (1997). In fact, multiple male births are necessary for immunization to happen, and this occurs only in highly fecund mothers in our society. It could be argued that aunts might present higher fecundity owing to familiarity effects. Familiarity effects do not, however, explain why higher fecundity should be present only in the maternal line and not in aunts of the paternal line. Furthermore, higher fecundity, while not significant (possibly owing to the small

sample size), is also found in mothers whose first son is homosexual, where the immunization hypothesis could not apply and higher fecundity should not be expected.

The observed differences in fecundity are compatible, at present, with either physiological or behavioural causes (e.g. lower abortion rate or increased ability to find mates, respectively), or both. The results in tables 1 and 2 suggest that genetic factors, transmitted in the maternal line, both increase the probability of being homosexual in males and increase fecundity in females. Grandparents do not show significant differences in fecundity. Note that if the grandmother is the carrier of the factors (likelihood of 0.5), she should show a higher fecundity, but if the grandfather is the carrier (likelihood of 0.5), we do not expect him to show higher fecundity. Therefore, a significant difference between the fecundities of grandparents of homosexuals and heterosexuals is not expected.

We have already mentioned that the hypothesis of genetic factors favouring homosexuality contradicts the expectation that natural selection would eliminate such factors, creating a paradox. Our data resolve this paradox by showing that there might be, hitherto unsuspected, reproductive advantages associated with male homosexuality. Existing genetic models of homosexuality are unable, however, to accommodate our results. A simple one-locus genetic model may predict the persistence of homosexuality, if homosexuals are assumed to be homozygous for an allele that is beneficial to heterozygotes (MacIntyre & Estep 1993; McKnight 1997; Werner 1998).

Such a model, however, does not explain the differences between paternal and maternal pedigree lines, nor does it explain the preferential maternal inheritance of homosexuality. Instead, both features can be found in one-locus models of genes residing on sex chromosomes (Rice 1998). Specifically, the assumption of an X-linked allele beneficial to female fecundity, but detrimental to male fecundity, would fit the pattern in our data. The existence of X-linked genetic factors associated with male homosexuality has been suggested previously from DNA-linkage analysis of homosexual brothers (Hamer *et al.* 1993; Hamer & Copeland 1995; for dissenting views, see Risch *et al.* 1993; Rice *et al.* 1999). The model, however, faces another difficulty. Based on our data, the X-linked allele should substantially enhance female fecundity, while being, at most, slightly detrimental to overall male fecundity (note that, in table 2, the fecundity of maternal uncles of homosexuals is not depressed, despite the fact that five of these individuals were childless homosexuals or bisexuals). The model would, thus, predict that the allele is very common (Rice 1998), whereas homosexuality is uncommon (Kinsey *et al.* 1948; Whitam *et al.* 1993; Hamer & Copeland 1995; Barbaglio & Colombo 2001).

The problem is not solved by assuming that the allele leads only rarely to homosexuality, since, in this case, the allele should also be prevalent in heterosexuals' families and, thus, could not explain differences in fecundity. Finally, it has been hypothesized (Miller 2000) that homosexuality may result from the accumulation, in a single individual, of many alleles, each of which is, by itself, beneficial, but whose cumulative effect is to favour homosexuality. This may account for persisting rare homosexuality, but not for differences between paternal and maternal family lines or the preferential maternal inheritance of

homosexuality. This model, however, may be combined with the previous one to obtain a polygenic model where one or more alleles are X-linked. Such a model may, in principle, reproduce our findings, but a full analysis of this possibility is beyond the scope of the present paper.

Finally, we emphasize that over 79% of the variance in male sexual orientation in our sample remains unaccounted for by the factors of an excess of maternal homosexual kin and number of older brothers. This is consistent with theoretical and empirical studies, which show that individual experiences are a powerful determinant of human sexual behaviour and self-identity (Churchill 1967; Enquist *et al.* 2002; for other species, see D'Udine & Alleva 1983; Hogan & Bolhius 1994). Indeed, it is still possible that the higher incidence of homosexuality in the maternal line results from culturally, rather than genetically, inherited traits. In many societies, such as in northern Italy, mothers spend a lot of time with children of both sexes, especially during the early years, which are critical for the development of sexual identity and orientation (Bailey & Zucker 1995; Smith *et al.* 1998). This suggests that the mother and the mother's family could be the preferential source of some of a child's behaviour and attitudes (Cavalli-Sforza & Feldman 1981; Cavalli-Sforza *et al.* 1982), including traits relevant to the child's future sexual preferences and behaviour. One may also speculate about co-evolution between genes and culture (Cavalli-Sforza & Feldman 1981; Boyd & Richerson 1985; Laland *et al.* 1995). For instance, a genetic mutation with the characteristics described above may not be advantageous when fecundity is high and limited only by the availability of resources, as in most traditional societies, but may spread more readily if fecundity is low and resources plentiful, as found in industrialized societies, such as Italy.

In conclusion, it is clear that our findings, if confirmed by further research, are only one piece in a much larger puzzle on the nature of human sexuality.

REFERENCES

Bailey, J. M. & Zucker, K. J. 1995 Childhood sex typed behavior and sexual orientation: a conceptual analysis and quantitative review. *Dev. Psychol.* **31**, 43–56.

Bailey, J. M., Pillard, R. C., Dawood, K., Miller, M. B., Farrer, L. A., Tivedi, S. & Murphy, R. L. 1999 A family history study of male sexual orientation using three independent samples. *Behav. Genet.* **29**, 79–86.

Barbaglio, M. & Colombo, A. 2001 *Omosessuali moderni. Gay e lesbiche in Italia*. Bologna: Il Mulino.

Bell, A. P. & Weinberg, M. 1978 *Homosexualities: a study of diversity among men and women*. New York: Simon & Schuster.

Blanchard, R. 1997 Birth order and sibling sex ratio in homosexual versus heterosexual males and females. *A. Rev. Sex Res.* **8**, 27–67.

Blanchard, R. & Klassen, P. 1996 H-Y antigen and homosexuality in men. *J. Theor. Biol.* **185**, 373–378.

Bobrow, D. & Bailey, M. J. 2001 Is male homosexuality maintained via kin selection. *Evol. Hum. Behav.* **22**, 361–368.

Boyd, R. & Richerson, P. J. 1985 *Culture and the evolutionary process*. University of Chicago Press.

Cavalli-Sforza, L. L. & Feldman, M. 1981 *Cultural transmission and evolution*. Princeton University Press.

Cavalli-Sforza, L. L., Feldman, M. W., Chen, K. H. & Dornbusch, S. M. 1982 Theory and observation in cultural transmission. *Science* **218**, 19–27.

Churchill, W. 1967 *Homosexual behavior among males: a cross-cultural and cross-species investigation*. Englewood Cliffs, NJ: Prentice-Hall.

D'Udine, B. & Alleva, G. E. 1983 Early experience and sexual preferences in rodents. In *Mate choice* (ed. P. Bateson), pp. 311–327. Cambridge University Press.

Enquist, M., Ghirlanda, S., Wachtmeister, C.-A. & Lundqvist, D. 2002 An ethological theory of attractiveness. In *Advances in visual cognition. I. Facial attractiveness* (ed. G. Rhodes & L. A. Zebrowitz), pp. 127–151. Westport, CT: Ablex Press.

Goodman, L. A. 1961 Snowball sampling. *Ann. Math. Stat.* **32**, 148–170.

Hamer, D. H. & Copeland, P. 1995 *The science of desire*. New York: Simon & Schuster.

Hamer, D. H., Hu, S., Magnuson, V. L., Hu, N. & Pattattucci, A. M. 1993 A linkage between DNA markers on the X chromosome and male sexual orientation. *Science* **261**, 321–327.

Heckathorn, D. D. 1997 Respondent-driven sampling: a new approach to the study of hidden populations. *Soc. Probl.* **44**, 174–199.

Hogan, J. A. & Bolhius, J. J. 1994 *Causal mechanisms of behavioural development*. Cambridge University Press.

ISTAT, 1951–1981 *Annali ISTAT: mortalità, natalità, fecondità, comportamenti riproduttivi*. Rome: Istituto Nazionale di Statistica.

Kinsey, A. C., Martin, C. E. & Pomeroy, W. B. 1948 *Sexual behavior in the human male*. Philadelphia: W. B. Saunders.

Laland, K. N., Kumm, J. & Feldman, M. W. 1995 Gene–culture co-evolutionary theory: a test case. *Curr. Anthropol.* **36**, 131–156.

MacIntyre, F. & Estep, K. W. 1993 Sperm competition and the persistence of genes for male homosexuality. *Biosystems* **31**, 223–233.

McKnight, J. 1997 *Straight science: homosexuality, evolution and adaptation*. New York: Routledge.

Miller, E. M. 2000 Homosexuality, birth order and evolution: toward an equilibrium reproductive economics of homosexuality. *Arch. Sex. Behav.* **29**, 1–34.

Moran, P. A. P. 1972 Familial effects in schizophrenia and homosexuality. *Aust. N. Z. J. Psychiatry* **6**, 116–119.

Muscarella, F. 2000 The evolution of homoerotic behavior in humans. *J. Homosex.* **40**, 51–77.

Rice, G., Anderson, C., Risch, N. & Ebers, G. 1999 Male homosexuality: absence of linkage to microsatellite markers at Xq28. *Science* **284**, 665–667.

Rice, W. 1998 Sex chromosomes and the evolution of sexual dimorphism. *Evolution* **38**, 735–742.

Risch, N., Squires-Wheeler, E. & Keats, B. J. B. 1993 Male sexual orientation and genetic evidence. *Science* **262**, 2063–2065.

Smith, P. K., Cowie, H. & Blades, M. 1998 *Understanding children's development*. Oxford: Blackwell.

Spreen, M. & Marius, H. 1992 Rare populations, hidden populations and link-tracing designs: what and why? *Bull. Methodol. Sociol.* **6**, 34–58.

Watters, J. K. & Biernacki, P. 1989 Targeted sampling options for the study of hidden populations. *Soc. Probl.* **36**, 416–430.

Werner, D. 1998 Sobre a evolução e variação cultural na homosexualidade masculina. In *Masculino, feminino, plural* (ed. J. M. Pedro & M. P. Grossi), pp. 99–129. Florianópolis, Brazil: Mulheres.

Whitam, F. L., Diamond, M. & Martin, J. 1993 Homosexual orientation in twins: a report on 61 pairs and three triplet sets. *Arch. Sex. Behav.* **22**, 187–206.

Wilson, E. O. 1975 *Sociobiology: the new synthesis*. Cambridge, MA: Harvard University Press.