

## Factors Associated with Higher Fecundity in Female Maternal Relatives of Homosexual Men

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DOI: 10.1111/j.1743-6109.2012.02785.x

### ABSTRACT

**Introduction.** Recent evidence suggests that sexually antagonistic genetic factors in the maternal line promote homosexuality in men and fecundity in female relatives. However, it is not clear if and how these genetic factors are phenotypically expressed to simultaneously induce homosexuality in men and increased fecundity in their mothers and maternal aunts.

**Aims.** The aim of the present study was to investigate the phenotypic expression of genetic factors that could explain increased fecundity in the putative female carriers.

**Methods.** Using a questionnaire-based approach, which included also the Big Five Questionnaire personality inventory based on the Big Five theory, we investigated fecundity in 161 female European subjects and scrutinized possible influences, including physiological, behavioral, and personality factors. We compared 61 female probands who were either mothers or maternal aunts of homosexual men. One hundred females who were mothers or aunts of heterosexual men were used as controls.

**Main Outcome Measures.** Personality traits, retrospective physiological and clinical data, behavior and opinions on fecundity-related issues were assessed and analyzed to illustrate possible effects on fecundity between probands and control females.

**Results.** Our analysis showed that both mothers and maternal aunts of homosexual men show increased fecundity compared with corresponding maternal female relatives of heterosexual men. A two-step statistical analysis, which was based on *t*-tests and multiple logistic regression analysis, showed that mothers and maternal aunts of homosexual men (i) had fewer gynecological disorders; (ii) had fewer complicated pregnancies; (iii) had less interest in having children; (iv) placed less emphasis on romantic love within couples; (v) placed less importance on their social life; (vi) showed reduced family stability; (vii) were more extraverted; and (viii) had divorced or separated from their spouses more frequently.

**Conclusions.** Our findings are based on a small sample and would benefit from a larger replication, however they suggest that if sexually antagonistic genetic factors that induce homosexuality in males exist, the factors might be maintained in the population by contributing to increased fecundity greater reproductive health, extraversion, and a generally relaxed attitude toward family and social values in females of the maternal line of homosexual men. **Camperio Ciani AS, Fontanesi L, Iemmola F, Giannella E, Ferron C, and Lombardi L. Factors associated with higher fecundity in female maternal relatives of homosexual men. J Sex Med 2012;9:2878–2887.**

**Key Words.** Female Fecundity; Male Homosexuality; Sexually Antagonistic Selection; Gay Men

### Introduction

Human sexual behavior spans a wide spectrum that ranges from complete heterosexuality to complete homosexuality, with all possible combi-

nations in varying frequencies. This is true across all human populations and all time periods [1–3]. A growing number of studies suggest that various genetic factors underlie human sexual behaviors. Heritable variations in sexual orientation support

the notion of an evolutionary maintenance mechanism. The role of genetic factors influencing homosexuality has been debated from an evolutionary perspective because the presence of these factors contradicts the Darwinian assumption that natural selection should progressively eliminate factors that reduce individual fecundity and fitness. Along this line, there is unanimous agreement that homosexuals reproduce significantly less than heterosexuals [4–7]. Notwithstanding, various avenues of research suggest genetic factors are partly associated with male homosexuality in our species [2,8–12].

The first set of evidence comes from family studies of biological brothers, adoptive brothers, and monozygotic twins that show that homosexuality is more common in brothers of homosexual subjects [8,13]. Hamer et al. studied homosexual brothers through DNA linkage analysis and found an increased rate of homosexuality in the maternal line. The researchers hypothesized that a putative genetic factor was located in the long arm of the X chromosome in the q28 region [2,10]. However, follow-up research has been unable to replicate these results.

This so-called Darwinian paradox has been the central theme of several studies [7]. In 1975, Wilson suggested a hypothesis based on kin selection [14]. He hypothesized that homosexuals could have an adaptive role as helpers in their families through affectionate and/or economic means, promoting the fitness of their close kin and thus balancing their own direct fitness loss. However, various researchers have failed to confirm the kin selection hypothesis [14], instead finding that homosexual men contribute no more than heterosexual men in terms of increased presence or economic or affective terms [15,16]. However, in a recent series of studies, Vasey et al. have reevaluated this hypothesis by observing a population of androphilic Samoan men, known as the fa'afafine [17–20]. LeVay suggested that homosexuality could be maintained by increased fecundity of female relatives. However, this suggestion remained untested [1]. King et al. found that homosexuals have larger families compared with heterosexuals, irrespective of the paternal and maternal lines, and in 2004, Camperio Ciani et al. found that women in the maternal line of homosexual subjects were significantly more fecund, having approximately one-third more offspring, than females in the maternal line of heterosexual men [21,22]. Significant differences were not found when comparing females from the paternal

line. The researchers also found that this effect applied to both homosexuals and bisexuals in the same manner [23]. Other studies confirmed that homosexual maternal female lineages are significantly more numerous, compared with heterosexual ones. Rahman et al. confirmed this finding, although reported that it only applied to Caucasian families [24].

Another study, which included a much larger sample size of homosexual males, has confirmed that homosexual men have a larger family size but only on the maternal side [25]. Notably, this study could not replicate King et al.'s findings of generalized increased fecundity [21]. Schwartz et al. recently suggested that fecundity in relatives of homosexuals is not limited to the maternal line [6]. However, while this study clearly shows a higher fecundity for mothers of homosexuals, it fails to distinguish fecundity between maternal aunts (which share an X chromosome with the subject) and maternal uncles (which do not share any X chromosome with the subject) or between maternal-sister cousins (which share the X chromosome with the subject) and maternal-brother cousins (which do not). Therefore, it has been difficult to assess differential fecundity in females from the maternal line. Other studies confirmed that the mothers of homosexuals have increased fecundity, while two further studies found independent evidence for maternal aunts' increased fecundity [25–27].

Taken together, these studies suggest the existence of genetic factors that partly influence male homosexuality, as the influence of increased fecundity in females might balance the fitness loss originating from homosexual males. The genetic model that could account for a balanced influence of sexual orientation in males and fecundity in females to maintain a stable frequency of homosexuals in the human population was investigated through a series of mathematical models based on single locus or multi loci [28,29]. Camperio Ciani et al. used an array of empirical data from various independent studies and showed that the most adequate model of maintaining homosexuality in human males is a two loci model with a sexually antagonistic selection mechanism, with at least one activating gene on the X chromosome and another one indifferently X-linked or autosomal [29]. The X chromosome in males is inherited only from the maternal line, which may be why homosexuals have more homosexual relatives from the maternal line and possibly why fecundity asymmetrically affects only the maternal line [20,24,25,29–31]. A sexually antagonistic model suggests that the same

set of alleles from one or more genes positively influences fitness in one sex (in this case, females) while negatively influencing the other.

The crucial research question is that at present, we do not know why homosexual males have more fecund maternal female relatives. We define the maternal female relatives as the mothers and maternal aunts of homosexuals, as carriers of putative genetic factors influencing male homosexuality (GFMH), following Camperio Ciani et al. [29]. We do not intend by this affirmation that homosexuality is just genetic but instead use the same term and operational definition that has been used in previous works.

We investigate herein the means by which phenotypic expression (physiological, emotional, or behavioral) increases fecundity. There are a reasonable number of causal connections suggesting that increased fecundity could be achieved either via differences in physiological fertility (such as a particular healthy condition and/or resistance to reproductive pathologies) or by promoting personality traits (such as high extroversion and low conscientiousness) that might increase sociosexuality. Additionally, sharing specific opinions or behaviors that promote sexuality or promiscuity may promote fecundity, either alone or in conjunction with physiological or personality traits. Hence, there are many potential candidates for inducing higher fecundity in the maternal female relatives of homosexuals [32–36].

### Aims

The aim of the present study is therefore to investigate the phenotypic expression of the previously hypothesized candidate genes that promote homosexuality in men and increase fecundity in the GFMH females.

### Methods

To compare GFMH with control females (mothers or maternal aunts of heterosexual men), we explored physiological factors that could increase fertility differences between the two groups that could also be associated with higher fecundity, according to the review of Hedon [32]. Such differences include stronger resistance to gynecological disorders, complicated pregnancies, pre- or peri-birth complications and spontaneous abortions [32]. We scrutinized the health problems most relevant to periods of fertility and those regarding reproduction such as endometriosis,

chlamydia, human papilloma virus (HPV), ovary dysfunctions (such as hyper- or hypo-ovulation, an-ovulation, and cysts), uterine dysfunctions (such as fibromas, malformations, reduced uterus, and other minor pathologies), tubal dysfunctions, pelvic infections (pelvic inflammatory disease [PID]), cancer, or other rare illnesses. All of these health problems may reduce fertility in mature females and may result in physiological expression of differential fecundity [32].

We also investigated personality profile differences that might indirectly induce higher fecundity using the BFQ inventory (Italian and French versions) based on the Big Five questionnaire [36–38].

Finally, we used a 10-point Likert scale to examine a set of relevant behaviors through attitude and opinion differences that could directly influence fecundity, such as aversion to contraceptives, disposition to having a larger number of sexual partners, or differences in personal opinions and individual values attributed in family, social, and sexual values. Such items included the importance of contraception (how important the subject rated contraceptive methods), number of sexual partners, importance of sex in the couple, importance of sexual intimacy, importance of romantic love in the couple, importance of a family, importance of family stability, the ideal number of children, care of children, importance of having children, importance of a child's education, importance of profession, importance of culture, and importance of social life.

### Participants

#### Inclusions

The participants in this study were 207 European females from various cities in northern Italy (Genova, Milano, Padova, and Pisa) and 9 females from France (Paris). The prerequisites for inclusion in the GFMH female group were being the mother or maternal aunt of a male older than 18 who was of definite sexual orientation (rated on the Kinsey scale) [39]. The GFMH females were contacted between 2005 and 2011 by the authors through gynecological counselors and associations of gay parents or through their own sons and nephews. The sons and nephews in this case were only those who responded to the Kinsey scale at levels 5 and 6 (5 meaning almost completely and 6 meaning completely homosexual) [39]. The authors contacted most of GFMH female subjects through indirect contacts and snowball sampling. The recruitment procedure for the control females

was identical to that of parents of homosexuals, and occurred in the same geographical region and was recruited through associations and social clubs. In this case, the sons and nephews were only those who responded at the Kinsey scale as 0 or 1 (0 completely or 1 almost completely heterosexual). No relatives of bisexuals were included in this study.

It is relevant to note that estimates referring to populations of units different from the survey units (i.e., in this case, the population of the mothers or aunts of homosexual subjects) is a complex issue and can lead to oversampling in large families (with higher fecundity than average). There is a tendency to oversample larger families due to their higher probability of being selected compared with smaller families. This tendency makes comparison with national statistics on family size inappropriate for two reasons. First, the national fecundity rate considers all females, including those with no sons, while mothers with no sons were ineligible for inclusion in this study. Second, national statistics that survey all families do not oversample large families as other partial sampling research designs do, and possible corrections meant to correct for the inverse probability of being selected introduce well-documented biases [40,41]. We confirm that these biases have no relevance in our study, owing to our procedure. The control sample was not from national statistics but instead was selected using the same methodology to reproduce the same possible biases and to guarantee the required internal validity of the comparisons.

All of the probands completed (i) a 142-item questionnaire designed specifically for this research; and (ii) an Italian or French version of the Big Five Questionnaire and personality inventory (BFQ, 132 items) [37,38]. Questionnaires were administered privately and anonymously and were mailed back to our laboratory. Each participant read, signed, and returned a detailed informed consent sheet. The research design was submitted and approved by the University General Psychology Department Ethical committee.

### Exclusions

Three questionnaires were invalid because their sons or nephews were adopted, and for the purpose of this research, all relations needed to be biological. An additional 22 probands were excluded from statistical analysis because relevant parts of the questionnaire were incomplete: 6 did not complete the first section regarding physical

health, opinions, and behaviors, and 16 did not complete section 2, the BFQ personality inventory test. Given these losses, the final sample consisted of 161 probands, with 61 in the GFMH group and 100 in the control group.

Of the GFMH group, 49 were mothers of homosexuals (including two who were also grandmothers of homosexuals and two who were also aunts of homosexuals). Twelve were aunts of homosexuals (of which two were also grandmothers of homosexuals). In the control group, 49 were mothers of heterosexuals (of which 16 were also maternal aunts of heterosexual males), and 51 were maternal aunts (of which 16 were mothers of heterosexual males). No proband in the control group reported any homosexual sons or maternal nephews.

### Measures

The first section of the self-report questionnaire concerned socio-anagraphical data, such as the number of biological offspring (both male and female), the relationship with the target subject (either homosexual or heterosexual), profession, and marital status.

A second section was devoted to the clinical history of the proband and included 45 yes–no questions on major general health disorders. These were followed by 37 yes–no questions associated with pre- and peri-natal problems.

Twenty-six questions regarded health problems most relevant to periods of fertility and reproduction such as endometriosis, Chlamydia infections, HPV, ovary dysfunctions (hyper- and hypo-ovulation, an-ovulation, and cysts), uterine dysfunctions (fibromas, malformations, reduced uterus size, and other pathologies), tubal dysfunctions, PID, cancer, or other rare illnesses.

The list included 11 questions on health problems that occurred during their pregnancies or childbirth, including all recorded complications such as spontaneous abortion, stillbirth, caesarean parturition, extended parturition, specific medical assistance during birth, and any other recalled complications.

All questions were compiled in a final frequency per subject defined as “complicated pregnancies.” This variable considers all pregnancy and parturition problems that might affect fertility and residual fecundity and was calculated for each proband relative scaled to the total number of offspring she produced.

The third section of the questionnaire investigated three questions regarding sexual behavior, such as the number of partners the proband had

during her fertile life, if she had ever decided to have a voluntary abortion (yes–no), and the importance given to contraception. Nine questions on opinions and desires were investigated using a 10-point Likert scale, described as follows: “Please indicate on this linear 10-point scale ranging from not relevant at all (1) to extremely relevant (10) . . .” the following items: (i) romantic love in the couple; (ii) sexual intimacy; (iii) family stability; (iv) maternity values; (v) children care; (vi) social life; and (vii) interest in professional activities, (viii) in culture, and (ix) in information.

Associated with the questionnaires designed for this study, we administered one of the widest validated personality questionnaires: the BFQ inventory (Italian and French versions), a 132-item questionnaire based on the Big Five theory that produces *T*-scores (mean 50, standard deviation [SD] 10) for five personality dimensions compared with a standardized population [37,38].

### Statistical Analyses

The small sample size and large number of variables to be analyzed caused two limitations in our study. First, it was not possible to conduct separate analyses of mother and maternal aunts. Both groups, however, have a common GFMH (although there was a 100% probability for mothers and only 75% for maternal aunts) and both showed increased fecundity. Hence, in this pilot study, we considered mothers and maternal aunts as a single group, possibly carrying the same genetic factors on the X chromosome. The second limitation was that we could not perform a multiple logistic regression analysis using all variables and instead used a two-step procedure.

In the first step of the data analysis, we compared one by one all fecundity variables, fertility disorders, behavior, attitudes, opinions, and personality traits using either the two group averages and standard deviations with *t*-test statistics (for continuous variables) or the two group frequencies with Chi-squared statistics (for categorical variables). In particular, for the *t*-test analyses, we recomputed average estimates of the *t*-test *P* values with nonparametric bootstraps (based on 1,000 samples). These additional analyses were performed to account for the relatively small sample size and to assess the robustness of the parametric *t*-test results. Subsequently, in the second step, only statistically significant variables (*P* < 0.05), of the previous one by one comparison, were entered as predictors in a multiple logistic regression with GFMH as the dependent variable in the model.

This allowed us to reduce the number of variables in the multiple logistic regression to a feasible number and to scale and rank the combined effect size of each significant variable, which was not possible with *t*-test statistics. Because GFMH is a dichotomous variable (0/1), we modeled this data as a logistic regression based on generalized linear models (GLMs) [42]. The logistic regression analysis allowed us to evaluate how and to what extent the two groups differ in terms of the selected predictors. More precisely, for each predictor in the logistic regression model, we computed its effects size with respect to GFMH according to the following equation:

$$\text{Effect Size source} = 100 * (\text{Dsource} / \text{Dnull}) [43]$$

where *D*source and *D*null denote the deviance attributable to the predictor and the null deviance (i.e., the deviance for a regression model with a constant term with no predictors), respectively. Notice that in case of GLMs, the term deviance replaces that of variance for standard linear regressions [42]. The effect size statistic represents the percentage of deviance explained by a dependent variable attributable to a predictor in the logistic regression model. In other words, the value of the effect size for a dependent variable represents the sensitivity of that dependent variable for the selected predictor. Finally, to estimate the 95% confidence interval of the effect size estimated, we recomputed average estimates of the effect size of the *z*-statistics by again using nonparametric bootstraps (based on 1,000 samples).

### Results

As shown in Table 1, the GFMH and the control group did not differ in professional status. There

**Table 1** Chi-square analysis of sociodemographic differences between GFMH females and control females

N	Control (100)	GFMH (61)	$\chi^2$	<i>P</i>
<b>Profession</b>				
Homemaker, no career	56	26	10.35	0.07
Teacher	11	6		
Social work	3	2		
Business	8	6		
Employee	11	18		
Manual labor	11	3		
<b>Marital status</b>				
Married vs. all others	84–16	42–19	4.26	<0.05
Divorced vs. all others	8–92	13–48	4.80	<0.05
Widowed vs. all others	7–93	6–55	0.12	0.73

Significant differences *P* < 0.05 in bold

**Table 2** T-test analysis of fecundity differences between GFMH and control females

	Control			GFMH			t	P
	N	Mean	SD	N	Mean	SD		
Mothers	49	<b>2.1</b>	0.68	49	<b>2.51</b>	0.89	-2.54	<b>0.01</b>
Maternal aunts	51	<b>1.47</b>	1.01	12	<b>2.33</b>	1.09	-2.6	<b>0.01</b>
Total	100	<b>1.78</b>	0.91	61	<b>2.48</b>	0.94	-4.64	<b>0.0001</b>

Significant differences  $P < 0.05$  in bold

were significantly more divorced or separated women in the GFMH group, while the difference in widows was not significant. Consequently, there were fewer married women in the GFMH group. Hence, we introduced the variable divorced-separated to the regression analysis while excluding the married variable as complementary. The two groups did not differ with respect to age (controls: mean age 53.6, SD 5.73; GFMH: mean age 55.1, SD 5.89) at a Welch two sample  $t$ -test ( $t_{(120,9)}$ ,  $P = 0.384$ ).

We found a significant difference in fecundity of GFMH females compared with control females (Table 2).

The  $t$ -test analysis of all possible differences in the two female groups showed relevant differences in the two distributions.

All results presented in Tables 3–5 were confirmed by nonparametric bootstrap analysis. In particular, the statistical results ( $P$  values) obtained using the two different analyses (standard  $t$ -test and  $t$ -test based on bootstrap samples) were highly correlated  $r = 0.96$  ( $P > 0.01$ ).

As shown in Table 3, the probands in the GFMH group are less exposed to pre-parturition problems and are significantly less exposed to gynecological complications during their fertile life.

General disorders are shown as a numeric variable consisting of the sum of 46 yes–no questions for each female regarding the presence of general health disorders between the ages of 15 and 45. Gynecological disorders are shown as a numerical variable consisting of the sum of 26 yes–no questions regarding gynecological disorders.

**Table 3** T-test analysis of the clinical history differences between GFMH and control females

	Control			GFMH			t	P
	N	Mean	SD	N	Mean	SD		
General disorders between the ages of 15 and 45	99	0.44	0.77	60	0.26	0.54	1.69	0.09
Gynecological disorders	99	<b>0.39</b>	0.62	61	<b>0.22</b>	0.42	1.99	<b>0.04</b>

Significant differences  $P < 0.05$  in bold

**Table 4** T-test analysis pregnancy disorder in relation to fecundity differences between GFMH and control females

	Control		GFMH		t	P
	N	Mean	N	Mean		
Complicated pregnancies	90	<b>0.25</b>	60	<b>0.08</b>	2.90	<b>0.004</b>
Complicated parturitions	90	0.22	60	0.23	-0.06	0.94
Natural parturitions	90	0.87	60	0.81	0.99	0.31
Caesarians	90	0.14	60	0.23	-1.23	0.22
Spontaneous abortions	90	0.18	60	0.09	1.68	0.09

Significant differences  $P < 0.05$  in bold

Table 4 shows that compared with control females, GFMH females have significantly fewer complicated pregnancies based on 11 yes–no questions.

The retrospective comparison of attitudes and opinions regarding fecundity-related behaviors (Table 5) held by GFMH shows that members of this group are significantly less concerned with the importance of having children ( $P = 0.03$ ), with romantic love within the couple ( $P = 0.005$ ), and with the importance of social life ( $P = 0.04$ ) and family stability ( $P = 0.006$ ). Furthermore, they showed a more relaxed attitude toward family values.

Table 6 shows  $t$ -scores for each personality trait. The only personality trait that significantly differs between GFMH and control females is extraversion.

### Logistic Model

Table 7 shows the results of the multiple logistic regression model and the average estimation of the effect size of each predicting variable, in terms of deviance proportion explained, and 95% confidence intervals based on the 1,000 replication bootstrap analysis. The total effect size of the logistic regression model is 20.08, and its 95% confidence interval is between 10.80 and 28.92.

### Discussion

Our results suggest that GFMH females have significantly higher levels of fecundity. This is true

**Table 5** I-test analysis of behavior, attitudes, and opinions differences between GFMH and control females

	Control			GFMH			<i>t</i>	<i>P</i>
	N	Mean	SD	N	Mean	SD		
Contraception	97	3.80	1.37	59	3.79	1.17	0.03	0.97
Number of partners	99	2.26	3.17	56	3.56	5.04	-1.63	0.10
Ideal n. of children	99	2.97	2.29	61	2.91	1.96	0.18	0.85
Importance of career	99	6.82	2.61	61	7.08	2.09	-0.67	0.50
Importance of family	86	9.31	1.28	40	8.77	1.57	1.88	0.06
Importance of having children	99	<b>8.45</b>	2.07	61	<b>7.77</b>	1.82	2.18	<b>0.03</b>
Importance of sexual intimacy	98	8.88	1.59	61	8.36	1.74	1.91	0.05
Care for children	99	9.47	1.18	61	9.44	0.90	0.19	0.84
Romantic love within the couple	98	<b>9.28</b>	1.29	61	<b>8.59</b>	1.60	2.85	<b>0.005</b>
Importance of culture	98	8.53	1.49	60	8.21	1.41	1.32	0.18
Importance of children's education	99	9.57	1.01	60	9.56	0.76	0.06	0.94
Importance of social life	99	<b>8.24</b>	1.59	61	<b>7.72</b>	1.57	2.02	<b>0.04</b>
Importance of family stability	98	<b>9.32</b>	1.23	61	<b>8.72</b>	1.41	2.75	<b>0.006</b>
Importance of sex in the couple	98	4.02	1.26	61	3.80	0.89	1.27	0.20

Significant differences  $P < 0.05$  in bold

for both mothers and maternal aunts, even when sampled independently. We found that GFMH females produce more offspring during their lifetimes, which also confirms previous results that report that mothers and aunts of homosexual men (GFMH) have significantly higher levels of fecundity than maternal female relatives of heterosexual men. The fecundity rates and differences between

groups found in this study are indeed very similar to previous studies on homosexual maternal line fecundity [22,23,25–28,30]. Rahman et al. proposed that increased fecundity applied only to the maternal line in white families and not in others. This discrepancy could be due either to higher fecundity of non-whites or other causes. Thus, research with other populations, non-Caucasian, larger samples, and the inclusion of further maternal kin is needed [24].

**Table 6** T-test analysis of BFQ personality traits differences between GFMH and control females

	Control			GFMH			<i>t</i>	<i>P</i>
	N	Mean	SD	N	Mean	SD		
Extraversion	95	<b>50.5</b>	11.1	58	<b>54.7</b>	11.5	-2.19	<b>0.02</b>
Agreeableness	95	53.3	10.7	58	55.5	13.9	-1.03	0.30
Conscientiousness	95	50.3	10.5	58	52.3	11.6	-1.05	0.29
Neuroticism	95	55.3	10.9	58	58.6	11.5	-1.74	0.08
Openness	95	46.5	11.0	58	46.9	11.6	-0.18	0.85

Significant differences  $P < 0.05$  in bold

**Table 7** Logistic regression of the significant predicting variables adopting a generalized linear model

	% Effect size (bootstrap mean)	% Effect size (bootstrap 95 CI)
Complicated pregnancies	4.12	0.94–7.99
Importance of couple romantic love	4.11	0.38–8.80
Extraversion	2.79	0.06–7.03
Importance of children	2.54	0.07–6.68
Divorced-separated	2.41	0.02–6.72
Gynecological disorders	2.00	0.04–4.95
Importance of family stability	1.13	0.01–3.73
Importance of social life	0.93	0.01–3.20
Total	20.08	10.80–28.92

For each predicting variables are shown nonparametric bootstrap mean and nonparametric bootstrap 95% confidence interval effect sizes. Bootstrap statistics were based on 1,000 samples.

This study also provides a first tentative answer to understanding phenotypic expression of sexually antagonistic genetic factors in females that induce higher fecundity and influence homosexuality in males.

Provided that these results can be confirmed with a larger data sample, phenotypic expression of genetic factors that lead to homosexuality in men and increase fecundity in women [2,7–12,18–20,22,23,25–27,29–31,44] may be mediated by a heterogeneous combination of physiologically superior gynecological and reproductive health and, possibly, a psychological phenotype consisting in a relaxed attitude toward family and social relationships and increased extraversion and tendency to divorce or separate.

We found that GFMH females have fewer gynecological problems during their fertile years and significantly fewer complicated pregnancies, despite having been pregnant more frequently than the control women. This factor alone could explain a higher fecundity. The relative absence of gynecological and pregnancy problems is one of the highest predictors of increased fecundity [32].

The retrospective behavioral investigation identified a rather large number of significantly

predictive variables showing that GFMH females have fewer concerns regarding family and social values. This might seem counterintuitive to the predictions of high fecundity in this group. However, decreased concern should not be misinterpreted as meaning reduced care, but rather as a relaxed attitude. A woman who does not rate family stability, social relationships, and children as highly important issues does not necessarily dislike their family and social relationships. On the contrary, we interpret this factor as meaning that she is less concerned by such issues. Children, romantic love within the couple, family stability, and social life have less relative importance for GFMH females compared with the control females. This finding may be related to their increased frequency of divorce or separation.

Personality completes the frame. Extraversion is the only trait that significantly differed between GFMH and controls, with the GFMH being significantly more extraverted. From an evolutionary perspective, personality has an adaptive value in natural selection processes, including reproductive success [34–36]. According to evolutionary psychologists, subjects who have high scores in extraversion and openness and low scores in conscientiousness are more likely to pursue short-term mating, have shorter marital relationships, and show increased fecundity [33,34]. A correlation was found between high fecundity and high extraversion, openness and agreeableness, and low levels of neuroticisms and conscientiousness [45–47]. However, while GFMH females were more extraverted, they showed a nonsignificant increase in conscientiousness than controls, did not differ in neuroticism, and did not have significantly more sexual partners [48]. This final point should be noted because a preliminary study suggested that GFMH females had more sexual partners during their lifetimes. Consequently, we hypothesized that the increased number of sexual partners could lead to increased fecundity. However, while a reanalysis of the initial GFMH sample along with a larger-sized sample confirmed a trend toward a higher number of sexual partners, the difference was not significant. Thus, promiscuity and the number of sexual partners do not reflect phenotypic expression.

Phenotypic expression is not the sole contributor to increased fecundity in the GFMH. Studies on Samoan androphilic males suggest that homosexuals might help more offspring in their family through increased *avuncularity* (uncle-like behavior) [18,19]. *Avuncularity* may act as a form of kin

selection for homosexual males and could increase fecundity in female kin. Thus, this phenomenon may positively impact sexually antagonistic genes expression that accounts for increased fecundity in GFMH females.

### Limitations

Our study has a similar sample size to comparable psycho-medical studies dealing with homosexual subjects [1,10,22,31,49]. Still, the GFMH group is rather small in number ( $N = 61$ ), and the high number of variables examined did not allow us to analyze mothers and maternal aunts separately for factors other than fecundity. To further explore the differences among GFMH females, our results should be confirmed and qualified with larger samples.

One limitation of our study is that we did not sample females in the paternal line. This was due to economic constraints. However, previous studies implicated both the X chromosome and the maternal line, which led us to focus our limited resources on maternal females, while leaving the investigation of paternal relatives to future research [2,10,22,29].

The risk of a sampling bias is always present in this sort of study [22]. The questionnaire was very long (142 items plus 132 items for personality assessment) and addressed sensitive issues. Moreover, the target sample consisted of a rather sensitive group. Thus, we encountered a high number of refusals to participate in the study, and the data collection phase lasted longer than 5 years. This could influence the results by sampling only certain profiles. To reduce this possible effect, we took particular care in targeting participants for inclusion and used an identical procedure for the control group [6,22]. An alternative interpretation of our data is that the mothers of homosexual males who responded had spent a significant portion of their life with a homosexual son, which could have progressively changed their opinions and emotions compared with mothers of heterosexuals. It is possible that our results might be showing either a reporting bias or a statistical artifact by virtue of women having cues of their male children's reproductive viability, which might affect personal attitudes and opinions. However, because personality traits stabilize after the age of 18 and are mostly unaffected by social environments, a son reproductive viability influence is rather unlikely, as it is unlikely a reporting bias regarding own fertility and gynecological health [36,38]. However, an effect might exist on life issue

opinions. Admittedly here, if subjects might bias their response, at present we cannot anticipate the bias direction. This concern should be addressed in further studies. A further limitation is that with our method we could not investigate any neural correlate of behavioral differences that have been shown important in sexual behavior and arousal [50].

The resultant model from multiple logistic regression explains a data deviance of only approximately 20%, which is somehow small. We did not expect to find very high effect sizes in our study due to the constraints of its design. A retrospective questionnaire, no matter how detailed, could never account for all choices, facts, and experiences that influence a person's decision to have or not have a child at any given time in life and hence might have enriched the model significance. Nevertheless, even a phenotypic frame with small effect sizes, when projected in a longitudinal context, such as a 30 years human's fertile life, could significantly influence the fecundity increase of GFMH females.

## Conclusions

With this type of limited data, we cannot directly derive a causal connection between the hypothetical sexually antagonistic autosomal or X-chromosome-linked genetic factors and health, behavior, and personality. Our tentative interpretation of the data is that females from the maternal line of homosexual men, who share X-chromosome-linked genetic factors with them, are more fecund, and this is relatively associated with better reproductive health, a socio-sexually relaxed attitude and extraverted personality frame. Functional genomics studies could further investigate how these traits, and maybe others, might affect the fecundity phenotype of maternal female relatives of homosexuals.

## Acknowledgment

We all wish to thank R. de Santis, president of AGEDO (Italian Association of Parents of Homosexuals), for her constant support and promotion of the aims of this research in all possible instances to increase our sample size and overcome prejudices and large difficulties.

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*Conflict of Interest:* None.

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## References

- 1 LeVay S. Queer science. The use and abuse of research into homosexuality. Cambridge, MA: The MIT Press; 1996.
- 2 Hamer D, Copeland P. The science of desire. New York: Simon and Schuster; 1995.
- 3 Camperio Ciani A. Darwin ed il paradosso dei geni omosex. *Darwin* 2005;56:36–45.
- 4 Bell A, Weinberg M. Homosexualities: A study of diversity among men and women. New York: Simon & Schuster; 1978.
- 5 Moran P. Familial effects in schizophrenia and homosexuality. *Aust N Z J Psychiatry* 1972;6:116–9.
- 6 Schwartz G, Kim RM, Kolundziji AB, Rieger G, Sanders AR. Biodemographic and physical correlates of sexual orientation in men. *Arch Sex Behav* 2010;39:93–109.
- 7 Jannini EA, Blanchard R, Camperio Ciani A, Bancroft J. Male homosexuality: Nature of culture. *J Sex Med* 2010;7:3245–53.
- 8 Bailey J, Zucker K. Childhood sex-typed behavior and sexual orientation: A conceptual analysis and quantitative review. *Dev Psychol* 1995;31:43–55.
- 9 Blanchard R, Bogaert A. Homosexuality in men and number of older brothers. *Am J Psychiatry* 1996;153:27–31.
- 10 Hamer D, Hu S, Magnuson V, Hu N, Pattatucci A. A linkage between DNA markers on the X chromosome and male sexual orientation. *Science* 1993;261:321–7.
- 11 Miller E. Homosexuality, birth order, and evolution: Toward an equilibrium reproductive economics of homosexuality. *Arch Sex Behav* 2000;29:1–34.
- 12 Mustanski B, DuPree M, Nievergelt C, Bocklandt S, Schork N, Hamer DH. A genome-wide scan of male sexual orientation. *Hum Genet* 2005;116:272–8.
- 13 Bailey J, Pillard R. A genetic study of male sexual orientation. *Arch Gen Psychiatry* 1991;48:1089–96.
- 14 Wilson E. Sociobiology: The new synthesis. Cambridge, MA: Harvard University Press; 1975.

- 15 Bobrow D, Bailey J. Is male homosexuality maintained via kin selection? *Evol Hum Behav* 2001;22:361–8.
- 16 Muscarella F. The evolution of homoerotic behavior in humans. *J Homosex* 2000;40:51–78.
- 17 Vasey PL, Pocock DS, VanderLaan DP. Kin selection and male androphilia in Samoan fa'afafine. *Evol Hum Behav* 2007;28:159–67.
- 18 Vasey PL, VanderLaan DP. Avuncular tendencies in Samoan fa'afafine and the evolution of male androphilia. *Arch Sex Behav* 2008;39:821–30.
- 19 Vasey PL, VanderLaan DP. Maternal and avuncular tendencies in Samoa: A comparative study of women, men and fa'afafine. *Hum Nat* 2009;20:269–81.
- 20 VanderLaan DP, Vasey PL. Male sexual orientation in independent Samoa: Evidence for fraternal birth order and maternal fecundity effects. *Arch Sex Behav* 2011;40:495–503.
- 21 King M, Green J, Osborn D, Arkell J, Hetherington J, Pereira E. Family size in white gay and heterosexual men. *Arch Sex Behav* 2005;34:117–22.
- 22 Camperio Ciani A, Corna F, Capiluppi C. Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. *Proc Biol Sci* 2004;271:2217–21.
- 23 Camperio Ciani A, Iemmola F, Blecher S. Genetic factors increase fecundity in female maternal relatives of bisexual men as in homosexuals. *J Sex Med* 2009;6:449–55.
- 24 Rahman Q, Collins A, Morrison M, Orrells J, Cadinouche K, Greenfield S, Begum S. Maternal inheritance and familial fecundity factors in male homosexuality. *Arch Sex Behav* 2007;37:962–9.
- 25 Blanchard R, Lippa RA. Birth order, sibling sex ratio, handedness, and sexual orientation of male and female participants in a BBC Internet research project. *Arch Sex Behav* 2007;36:163–76.
- 26 Turner WJ. Homosexuality, type 1: An Xq28 phenomenon. *Arch Sex Behav* 1995;24:109–34.
- 27 Bailey JM, Pillard RC, Dawood K, Miller MB, Farrer LA, Trivedi S, Murphy RL. A family history study of male sexual orientation using three independent samples. *Behav Genet* 1999;29:79–86.
- 28 Gavrilets S, Rice W. Genetic models of homosexuality: Generating testable predictions. *Proc Biol Sci* 2007;273:3031–8.
- 29 Camperio Ciani A, Cermelli P, Zanzotto G. Sexually antagonistic selection in human male homosexuality. *PLoS ONE* 2008;3:e2282.
- 30 Iemmola F, Camperio Ciani A. New evidence of genetic factors influencing sexual orientation in men: Female fecundity increase in the maternal line. *Arch Sex Behav* 2009;38:393–9.
- 31 Vasey P, VanderLaan DP. Birth order and male androphilia in Samoan fa'afafine. *Proc Biol Sci* 2007;274:1437–42.
- 32 Hedon B. Fertility and sterility: A current overview. *Proceedings of the 15th World Congress on Fertility and Sterility*. Philadelphia, PA: Taylor and Francis; 1995.
- 33 Buss D. Evolutionary personality psychology. *Annu Rev Psychol* 1991;42:459–91.
- 34 Buss D. Evolutionary psychology: A new paradigm for psychological science. *Psychol Inq* 1991;6:1–30.
- 35 Solomon E, Berg L, Martin D, Villee C. *Biologia*. Napoli, Italy: EdiSES; 1997.
- 36 Camperio Ciani A, Capiluppi C, Veronese A, Sartori G. The adaptive value of personality differences revealed by small island population dynamics. *Eur J Pers* 2007;21:3–22.
- 37 Caprara G, Perugini M. Personality described by adjectives: The generalizability of the Big Five to the Italian lexical context. *Eur J Pers* 1994;8:357–69.
- 38 McCrae R, Costa P. Personality trait structure as a human universal. *Am Psychol* 1997;52:509–16.
- 39 Kinsey AC, Martin CE, Pomeroy WB. *Sexual behavior in the human male*. Philadelphia, PA: Saunders; 1948.
- 40 Goodman LA. Snowball sampling. *Ann Math Statist* 1961;32:148–17.
- 41 Spreen M, Marius H. Rare populations, hidden populations and link-tracing designs: What and why? *Bull Methodol Sociol* 1992;6:34–58.
- 42 McCullagh P, Nelder J. *Generalized linear models*. 2nd edition. Philadelphia, PA: Taylor and Francis; 1989.
- 43 Nakagawa I, Cuthill C. Effect size, confidence interval and statistical significance: A practical guide for biologists. *Biol Rev* 2007;82:591–605.
- 44 Vasey P. Homosexual behavior in primates: A review of evidences and theory. *Int J Primatol* 1997;16:173–204.
- 45 Jokela M, Alvergne A, Pollet TV, Lummaa V. Reproductive behavior and personality traits of the Five Factor Model. *Eur J Pers* 2011;77:213–30.
- 46 Jokela M, Keltikangas-Järvinen L. Adolescent leadership and adulthood fertility: Revisiting the “central theoretical problem of human socio-biology.” *J Pers* 2009;77:213–30.
- 47 Eaves LJ, Martin NG, Heath AC, Hewitt JK, Neale MC. Personality and reproductive fitness. *Behav Genet* 1990;20:563–8.
- 48 Rahman Q. Homosexuality and other markers of human sexual orientation: The association between the fraternal birth order effect in male. *Biol Lett* 2005;1:393–5.
- 49 Karger S, Klucken T, Werhrum S, Zimmerman M, Schienle A, Walter B, Vaiti D, Stark R. Neural activation toward erotic stimuli in homosexual and heterosexual males. *J Sex Med* 2011;8:3132–43.
- 50 Buss DM. Paternity uncertainty and the complex repertoire of human mating strategies. *Am Psychol* 1996;51:161–2.

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