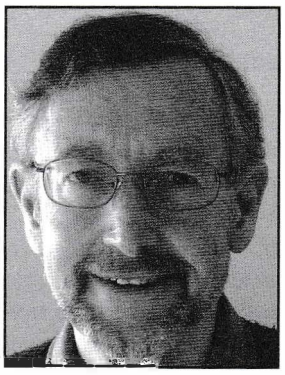
Latest Twin Study Confirms Genetic Contribution To SSA Is Minor

As in previous studies, identical twins usually differ for SSA.

By N.E. Whitehead, Ph.D.

Twin studies are favorites of mine because of the potential light they throw on the origins of same-sex attractions (SSA). The latest one (Santtila et al., 2008) is three times larger than any previous study – in fact, larger than all the rest put together.



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Does this latest study teach us something new? Quick answer: No. It confirms the best recent studies, which tell us that genetic factors are minor; non-genetic factors are major.

The paper's title is "Potential for Homosexual Response is Prevalent and Genetic." This implies to the average reader that homosexuality is sometimes hidden, but commonly occurring, and that it is predominantly genetic. But we shall see this title is not representative of the study's actual findings.

This is the fifth systematically sampled twin study to look at SSA independently in men and women. Of the four previous studies, two were from Australia (Buhrich, Bailey & Martin, 1991; Bailey, Dunne & Martin, 2000), and two were from the USA (Hershberger, 1997; Bearman & Bruckner, 2002).

This latest study is from Finland. Using the very centralized records typical of Scandinavian states, they assembled a large, genuinely random sample of twins (6,001 female individuals and 3,152 males) for a study that was primarily on aggression. With that constraint, they were permitted only two questions about SSA: "What same-sex sexual contact have you had in the last year?" and (in essence) "If there was no prospect of anyone finding out, and you were sexually propositioned by someone of the same sex you liked, what would be your chances of accepting?"

Before we go further, let's address one small difficulty. Unfortunately, different studies use different measures for SSA. Some ask for total number of partners – this one asked only the frequency of contacts in the last year. Other surveys ask the frequency of same-sex fantasy. This one asked respondents to fantasize (perhaps for the first time) about what sexual contact with a same-sex partner might be like. The authors then say this is measuring "potential homosexuality," but you and I would probably conclude that such a measure is fairly clearly indicating something other than SSA. This measure obviously would include bisexual people, and casts the net so wide, that it also could well be testing for something like novelty, curiosity, or sensation-seeking, rather than actual sexual orientation. In this study, 32.8% of men and 65.4% of women replied "yes" to that question about fantasy, in contrast to 3.1% of men and 1.2% of women who described themselves as actually homosexually active.

The results were:

Activity	Genetic	Shared Environment	Non-shared
Men	27% (2.7-38)	0% (0-18)	73% (62-85)
Women	16% (8.3-24)	0% (0-3.6)	84% (76-91)

Potential Genetic		Shared Environment	Non-shared
Men	37% (12-47)	0% (0-19)	63% (53-73)
Women	46% (32-52)	0% (0-11)	54% (48-60)

Table 1. Relative influence of various factors for the Santtila and Sandnabba (2008) data. Error ranges in parentheses are the 95% (2 sigma) error range.

The table shows that the estimated genetic contributions are a few tens of percent, but that the error ranges (in brackets) are quite large and this could possibly mean the genetic contribution is zero. This is exactly the same as has been found previously. They also show that the non-shared environmental contribution (i.e. environmental factors particular to the individual) greatly predominate – in other words they are the largest group of causes of SSA.

Are genetic contribution results of say 27% important? No. In the twin studies world the influence would be classified as weak to modest. And any influence is indirect – it is likely to be something like an innate tendency to be very sensitive to the opinions of others. However, even this weak or modest genetic contribution is probably greatly overstated.

Twin study researchers usually involve the siblings of identical twin subjects as much as possible, because they are genetically related to the same degree as fraternal twins, hence like substitute fraternal twins. This sibling/twin comparison is very interesting because it tests for any special twin environment. For example, did the twins influence each other to be SSA? Or did the genetic similarity between the identical twins and siblings cause some lesser SSA in the siblings also? In this case, the siblings were tested along with the identical twins and fraternal twins and the results were meaningless-- i.e., they did not yield results compatible with genetic influence in SSA. Although the authors do not specify exactly what the problem was, it must have been severe ("...attempts at fitting uni-variate and bi-variate extended-family scripts for categorical data were not successful...." which is scientese for the explanation I give above). This would usually be enough to destroy a study of genetic influ-

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