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A population-based twin study of self-esteem and gender

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ABSTRACT

Background. Self-esteem (SE), a widely used construct in the social sciences, is usually conceptualized as a reflection of socialization and interpersonal experiences that may differ considerably between the genders.

Methods. The Rosenberg self-esteem scale was assessed at personal interview in both members of 3793 unselected twin pairs (1517 male–male, 856 female–female and 1420 male–female) from the population-based Virginia Twin Registry. Gender effects on SE were assessed by both analysis of variance and biometrical twin modelling.

Results. The mean SE score was slightly but significantly lower in women *v*. men, and in women who grew up with a male *v*. a female co-twin. Twin modelling suggested that: (*i*) individual differences in self-esteem in both men and women were best explained by genetic and individual-specific environment factors; (*ii*) heritability estimates were similar in women (32%) and in men (29%); and (*iii*) the same genetic factors that influenced SE in women also influenced SE in men. Analyses supported the validity of the equal environment assumption for SE. The heritability of SE cannot be explained by the moderate correlation between SE and symptoms of depression.

Conclusions. These results are inconsistent with prominent gender-related aetiological models for SE, which postulate that individual differences arise from socialization experiences both within and outside the home of origin which differ widely for the two genders. Instead, a significant proportion of the population variance in SE is due to genetically-influenced temperamental variables that are the same in men and women.

INTRODUCTION

Self-esteem (SE) is a psychological construct that has, since the time of William James, been widely employed in the field of mental health and in the social sciences more generally. Low levels of SE have been associated with a range of adverse outcomes including poor school performance (Brookover *et al*. 1964), substance abuse (Brehm & Back, 1968; Bry *et al*. 1982; Walitzer & Sher, 1996), eating disorders (Yates, 1989; Kendler *et al*. 1991), anxiety disorders (Ingham *et al*. 1986), major depression (Brown *et al*. 1986, 1990; Ingham *et al*. 1986) and poor general health (Hunter *et al*. 1981).

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Nearly all of the large literature on SE has assumed that the aetiology of this construct is to be found in the psycho-social domain. As summarized by Robson (1988) 'The self-concept was seen as being acquired through interaction with other people rather than being inborn'. Contrary to this perspective, we recently showed, in a study of female–female monozygotic (MZ) and dizygotic (DZ) twin pairs, that twin resemblance for SE was substantial and appeared to be due largely, or entirely, to genetic factors (Roy *et al*. 1996).

Men, on average, have modestly higher levels of SE than women (Skaalvik, 1986; Feingold, 1994). Prior studies have argued that gender differences arise because of different sources of SE in the two genders. In particular, inter-

personal relationships have been postulated to be central to SE in women while positive individuation, dominance and school/occupational success are considered to be particularly important sources of SE for men (Swap & Rubin, 1983; Miller, 1986). Nearly all studies of gender and SE have assumed that these differences arise from the varying pattern of socialization of males and females (Swap & Rubin, 1983; Miller, 1986; Eagly, 1987). That is, differences in SE arise because men and women are taught to derive self-value from different sources.

In this report, we extend our earlier genetic-epidemiological investigation of SE to include a large sample of male–male MZ and DZ twins and male–female DZ twins. Our major goal is to clarify, by the use of twin modelling techniques, the sources of individual differences in SE in men and women. In particular, we seek to determine if: (*i*) genetic factors are of similar importance in the aetiology of SE in the two genders; and (*ii*) whether the same or different genetic factors influence SE in men and women.

METHOD

The sample

The twins included in this report derive from two inter-related projects utilizing the population-based Virginia Twin Registry – formed from a systematic review of all birth certificates in the Commonwealth of Virginia. The female–female twin pairs were eligible to participate if they were white and born 1934–1971 and both members had previously responded to a mailed questionnaire, to which the individual response rate was ~ 64%. In our first interview completed in 1988–9, we assessed 92% of the eligible individuals ($N = 2163$), 90% face-to-face, the rest by telephone. Zygosity was determined blindly by standard questions (Eaves *et al.* 1989) photographs, and when necessary, DNA (Spence *et al.* 1988; Kendler *et al.* 1992).

We have performed two additional waves of telephone interviews completed in 2001 (92·5%) and 1898 (87·7%) of the original sample, respectively. The mean ($\pm$S.D.) of months between the first and third interviews was $61{\cdot}3 \pm 5{\cdot}1$. In the third interview, at which time SE was assessed, we interviewed both members of 854 pairs, 497 of whom were monozygotic (MZ), 354 of whom were dizygotic (DZ) and 3 of whom had unknown zygosity. The mean age of the participating twins in the third wave of interviews was $34{\cdot}6 \pm 7{\cdot}5$ years and ranged from 22 to 59.

The male–male and male–female twin pairs were ascertained in a separate study beginning in 1993. Twins were eligible for this study if: one or both twins were successfully matched, they were a member of a multiple birth which included at least one male, were Caucasian, and were born between 1940 and 1974. Of 9378 twins eligible for study, 6864 were interviewed (73%), 1184 refused (12·6%), 354 did not agree within the study time limit (3·8%), 851 could not be located (9·1%) and 125 were deceased or too ill to be interviewed (1·3%). Thus, of 8402 twins contacted and available for participation, 81·7% were successfully interviewed.

From this sample, we have 851 male–male MZ pairs, 647 male–male DZ twins and 1404 opposite-sex (OS) male–female DZ pairs with complete data on SE. In addition, this sample contained 10 triplets in whom we could interview only two members, 14 triplets where we interviewed all three members and one complete set of quadruplets all of whom co-operated. From these higher-order multiple births, we formed, for these analyses, a total of 58 additional twin pairs: 3 female–female MZ, 3 female–female DZ, 13 male–male MZ, 12 male–male DZ and 27 male–female DZ. Excluded from these analyses were twins whose co-twin had not participated.

At the time of interview, subjects in the male–male/male–female study ranged in age from 18 to 60 years, with a mean age of 35·1 (S.D. = 9·2). Most interviews were conducted by telephone, but ~ 5% of subjects were interviewed in person because of personal preference, residing in an institutional setting (usually jail or prison), or not having telephone service. Zygosity was determined by an algorithm based on standard questions, validated against the zygosity diagnoses (based on DNA and photographs) in the female–female sample. Application of the algorithm to this male sample was validated by analysis of 15 highly-informative DNA polymorphisms in a random sample of 184 twin pairs. The algorithm classified 177 pairs correctly, an error rate of 3·8%.

All interviews were conducted blind to information about the co-twin. Written informed consent was obtained prior to face-to-face interviews and verbal assent prior to phone interviews. Of the total of 3811 twin pairs available from both studies, usable SE data was available from both members of 3793 pairs (99·5%).

SE was assessed by the 10-item Rosenberg SE Scale (Rosenberg, 1965) on which responses varied on four-point scale from strongly agree to strongly disagree. However, due to a clerical error, one of the items was deleted from the interview with the male–male and male–female twins ('At times I think that I am no good at all'). Therefore, the nine-item version was used for all twins in these analyses. If fewer than 50% of the SE items were missing (which occurred in only 12 twins, of whom 10 were missing only a single item), we extrapolated their total scale score from the answered items.

Statistical analysis

Measures of 'childhood similarity' and 'current frequency of contact' were obtained from the twins. Each twin was asked how often they shared the same room, had the same playmates, were dressed alike, and were in the same classes at school (Loehlin & Nichols, 1976). They were also asked how often they were currently in contact with their co-twin (Kendler *et al.* 1986). We tested the equal environment assumption – that MZ and DZ twins were equally correlated for exposure to environmental variables that influenced SE – by attempting to predict the absolute value of the twin difference in SE, controlling for age and zygosity, by measures of the similarity of their childhood and adult environments.

Prior to twin modelling, we regressed out, from an individual twin's SE score, the effect of age, gender, and, within gender, whether the twin had a same *v.* opposite-sex co-twin. The models fitted to these data begin with the standard sources of variance found in all such twin models: additive genes (A), common or familial environment (C) and individual-specific environment (E) (see Kendler, 1993; and Neale & Cardon, 1992) for a more detailed description). In addition, by comparing estimates for A, C and E in male–male and female–female pairs, we can address the question of whether these estimates are gender-dependent. That is, does the relative importance of genetic or environmental risk factors for SE differ in men and women? Also, by analysing jointly all five twin-zygosity groups (male MZ, male DZ, female MZ, female DZ and opposite-sex DZ), we can address an additional question: to what extent do the same genetic and/or environmental factors influence SE in men *versus* women? It is possible, for example, that familial factors are important in influencing SE in both men and women – but the familial factors are entirely different. In that case, SE would be significantly correlated in male DZ pair and female DZ pairs, but not in opposite sex DZ pairs.

Model fitting was performed using Mx (Neale, 1994) applied to the variance covariance matrices by the method of maximum likelihood. The best fitting model was chosen using Akaike's information criteria (Akaike, 1987; Williams & Holahan, 1994). We then present parameter estimates from the best fitting model, where a^2 and e^2 equal the proportion of variance in SE scores due to additive genetic effects and individual specific (or unique) environment, respectively. The parameter r_g is the correlation in the additive genetic effects on SE in males and females. If $r_g = 1$, then the genetic factors which influence SE in males and females are the same. If $r_g = 0$, then the genetic factors which influence SE in males are unrelated to the genetic factors which influence SE in females.

Differences in mean values of SE were determined by *t* test and analysis of covariance. While our twin analyses were restricted to twin pairs on whom we had SE measures from both members, our analyses of mean scores were performed on all available twins.

RESULTS

Means

The sample sizes, means and S.D.s of the SE scores, standardized against the entire sample, for the six gender-zygosity groups were: female MZ ($N = 1065$) $0{\cdot}06 \pm 1{\cdot}06$; female DZ ($N = 779$) $-0{\cdot}02 \pm 1{\cdot}08$; male MZ ($N = 1727$) $+0{\cdot}14 \pm 0{\cdot}98$; male DZ ($N = 1317$) $0{\cdot}01 \pm 0{\cdot}99$; female members of OS–DZ pairs ($N = 1427$) $-0{\cdot}20 \pm 1{\cdot}00$; male members of OS–DZ pairs ($N = 1428$) $+0{\cdot}02 \pm 1{\cdot}00$. An analysis of covariance, controlling for the effect of age, found that

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Table 1. *Model-fitting results for self esteem in male–male, female–female and male–female twin pairs*

Model	χ^2	df	AIC	a^2_m	c^2_m	e^2_m	a^2_f	c^2_f	e^2_f	r_{mf}
I	18·3	8	+2·3	0·29	0·00	0·71	0·33	0·00	0·66	+0·88
II	18·3	10	−1·7	0·29	0·00*	0·71	0·33	0·00*	0·66	+0·88
III	18·7	11	−3·3	0·28	0·00*	0·72	0·32	0·00*	0·66	1·00*
IV	26·5	13	+0·5	0·30†	0·00*	0·70†	0·30†	0·00*	0·70†	1·00*

* Constrained to value of zero or unity.
† Constrained to be equal in males and females.

the effects of zygosity ($F = 11{\cdot}92$, df = 1/7672, $P = 0{\cdot}0006$), gender ($F = 30{\cdot}50$, df = 1/7672, $P = 0{\cdot}0001$) and same *v*. opposite sex twin pairs ($F = 8{\cdot}64$, df = 2/7672, $P = 0{\cdot}0002$) were all significant. A follow-up analysis of this last result indicated that the significant difference was due to lower SE scores in females from OS *v*. SS pairs ($F = 16{\cdot}95$, df = 1/7672, $P = 0{\cdot}0001$). No such difference was seen in males ($F = 0{\cdot}05$, df = 1/7672, $P = 0{\cdot}82$). While these differences are statistically significant, they were small in magnitude. The mean difference in SE in males and females was 0·13 S.D. units, gender accounting for only 0·4% of the variance in the total sample SE scores. In female DZ twins, the gender of the co-twin accounted for 0·7% of the variance in SE scores.

Individual differences – twin modelling

Twin analyses are predicated on the assumption that the trait-relevant environmental experiences are similarly correlated in MZ and DZ twin pairs. We tested this by predicting within-pair similarity in SE separately in male–male and female–female pairs from the within-pair similarity in childhood environments and the frequency of current contact as adults. None of the results of these four analyses was statistically significant.

After partialling the effects of age, gender and zygosity, the product-moment correlations (and sample sizes of twin pairs) for SE in the five twin zygosity groups were: MZ male twins +0·30 ($N = 859$), DZ male twins +0·11 ($N = 658$), MZ female twins +0·35 ($N = 500$), DZ female twins +0·16 ($N = 356$) and male–female DZ twins +0·13 ($N = 1420$).

As outlined in Table 1 we began by fitting a full model (model I) which allowed for separate estimates of a^2 and e^2 for males and females and for a variable correlation in the genetic and environmental risk factors in the genders. This model fit the data relatively well and produced estimates of a^2 that were quite similar in males (0·29) and females (0·33). Common environment was estimated to be 0·00 in both males and females. The correlation in genetic factors which influence SE in males and females was estimated to be high ($r_g = +0{\cdot}88$).

Model II differed from model I in setting both common environmental parameter estimates to zero. The fit of the model did not change at all and the AIC improved, indicating that we had no evidence that common environment significantly impacted on SE.

Model III differed from model II in setting the correlation in genetic risk factors for SE in males and females to unity. The fit changed very little and the AIC further improved. In these data, we could not reject the hypothesis that the genetic influences on SE were the same in males and females.

In model IV, we constrained the estimates of a^2 and e^2 to be the same in males and females. The fit of the model took a substantial jump and the AIC worsened considerably. Although the heritability estimates for SE were similar in males and females in this sample, we could reject the hypothesis that they were equal.

Model III then, was the best fitting model, and estimated the heritability of SE in males and females (with 95% confidence intervals) to be: 29% (23–34) and 32% (25–39), respectively.

DISCUSSION

Our major goal in this paper was to use a genetic-epidemiological design to clarify the relationship between the genetic risk factors for SE in men and women. Our results were clear. As we had previously demonstrated in women (Roy *et al*. 1995), twin resemblance for SE was

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due entirely to genetic factors with a heritability of ~ 30%. The role of genetic factors in the aetiology of SE was slightly greater in females than males, but the difference was modest and only detectable because of the large sample sizes. In neither men nor women could we detect evidence for an effect of the family environment on twin resemblance for SE. Most importantly, the genetic risk factors for SE appeared to be the same in men and women.

Using a sample that was population based and many times larger than prior studies (Feingold, 1994), we replicated previous evidence for a gender difference in SE. A meta-analysis, based on 27 studies published from 1984 to 1992, found a mean effect size for the impact of gender on SE of 0·16 (Feingold, 1994) – very close to the 0·13 found in this report. Men appear to have, on average, higher SE than women. However, this gender difference is very small and accounts for under one-half of 1% of the total population variation for SE.

In addition to the main effect of gender on SE, we found that the impact of gender on self-esteem varied with the gender of the co-twin. In DZ twins, the SE score of a male is apparently unrelated to the gender of his co-twin while the SE score of a female is lower if her co-twin is male than if her co-twin is female. Again the effect size is quite small, ~ 1/6th of a standard deviation, accounting for under 1% of the total variance in SE. However, these results suggest that growing up with a male co-twin has a subtle adverse effect on a female's SE.

Finally, we also found an impact of zygosity on SE. Controlling for gender of twin and co-twin, the mean SE score for MZ twins was 0·16 S.D. units higher than that seen for DZ twins. We are unaware of any similar previously published finding. This difference could arise in two ways. First, the effect might be present in the population, reflecting perhaps a greater sense of 'specialness' experienced by MZ twins or the impact of the emotional bond between MZ twins which is usually closer than that seen with DZ twins. Alternatively, the effect might arise through selection bias. In these analyses, we only examined twins in which both members co-operated, as only in such pairs could zygosity be assigned with confidence. However, SE was significantly lower in twins whose co-twin refused to participate in our study ($F = 12{\cdot}30$, df = 1/8667, $P = 0{\cdot}0005$), suggesting an inverse relationship between SE and co-operativity. Given that SE is more strongly correlated in MZ than in DZ twins, our requirement that both members of a pair participate will more effectively screen out individuals with low SE in MZ than in DZ twins (Kendler & Holm, 1985). While we cannot easily determine the extent to which the SE difference in zygosities is 'real', its impact is likely to be limited. MZ twins do not appear to differ from DZ twins in the major personality dimensions (Rutter & Redshaw, 1991), level of self-report psychiatric symptoms (Kendler *et al.* 1995) or in hospital admission rates for major affective and psychotic disorders (Kendler *et al.* 1996).

What is inherited?

The measure we have used for SE – the Rosenberg scale – reflects 'global self-approval' rather than, for example, competency in specific life roles (Brown *et al.* 1990). As Rosenberg himself has argued (Rosenberg *et al.* 1995), this scale is particularly 'affective' in nature, thereby raising the question of whether global SE can be meaningfully differentiated from depression or dysthymia. Since symptoms of depression are heritable (e.g. Jardine *et al.* 1984; Kendler *et al.* 1994), could our evidence for genetic effects on SE result simply from the correlation between SE and depression?

We could examine this issue because we also assessed in our twin sample symptoms of depression using items from the SCL-depression scale (Derogatis *et al.* 1973), which we have previously shown to be heritable (Kendler *et al.* 1994). The correlation between symptoms of depression and SE was negative and highly significant ($r = -0{\cdot}46$, $P < 0{\cdot}00001$). We regressed out the effect of depressive symptoms from each twin's SE score and then re-fitted the models outlined in Table 1 to these 'depression-corrected' SE scores. The results differed little from those presented above. Model III was again the best-fitting model and the estimated heritability (with 95% confidence intervals) of 'depression-corrected' SE was only slightly lower than found using the uncorrected SE value: males 28% (22–37) and females 29% (22–35). These results suggest that the genetic influences on SE are largely independent of those that influence depressive symptoms.

Integration

The results of twin modelling – which examines the sources of individual differences – suggest that the genetic factors that influence SE are the same in men and women. However, men and women have significantly different mean SE scores. While these two findings might appear contradictory, they are not. Assume, for example, that menstruation consistently impacts in a modest, negative manner on SE. Since virtually all women but no men experience menstruation, its effect would be detected in a comparison of mean values but not in a study of individual differences. (The same pattern of findings could be produced by pervasive cultural differences in the socialization of boys and girls.) While women have slightly lower average SE than men, the same genetic factors cause men and women to differ from the level of SE typical for their gender. SE might, in this pattern of results, resemble height and weight. While there are large, biologically driven, differences in the mean height and weight of men and women, the genetic factors which influence individual differences in these two physical traits are apparently the same in men and women (Maes *et al*. 1997).

At least one alternative explanation of these findings, however, is plausible. Let us return to our example of menstruation. Assume that menstruation has, on average, a slight negative impact on SE. However, the SE reducing effect of menstruation differs among women, in part because of genetic differences. These genetic influences on SE (which would be unique to women) would be of such small overall impact (remembering that the total gender difference accounts for $< 1\%$ of variance in SE), they would be undetectable in twin analyses (Martin *et al*. 1978).

In addition to a general effect of gender on mean levels of SE, however, we also have evidence for a more specific effect: female twins have lower SE if they have grown up with a male *v*. with a female co-twin. This result suggests that a particular aspect of the familial environment – gender of the co-twin – can significantly influence SE. However, magnitude of this effect is also very small ($< 1\%$ of the variance). Power analyses in twin studies have shown limited ability, even with large sample sizes, to detect familial environmental effects accounting for less than 5% of the variance (Martin *et al*. 1978). It is, therefore, not surprising that our twin modelling detected no evidence for an impact of family environment on SE.

Implications

Our results are inconsistent with the hypothesis that SE is largely environmental in origin. SE does not emerge solely through interaction with other people, but rather is substantially influenced by genetic factors, probably acting via temperamental characteristics. Indeed, the results obtained for SE – moderate heritability and absence of detectable familial–environmental factors – are very similar to those found for most human personality traits (Eaves *et al*. 1989; Loehlin, 1992).

Our findings also do not support the hypothesis that features of the rearing environment that are consistent across members of a sibship, such as socio-economic class, parental rearing style or religious orientation, have a crucial impact on SE in adulthood. The following scenario, for example, while in accord with much of the prior work on gender differences in SE, is particularly inconsistent with our findings:

> Some parents and communities emphasize strict gender roles in their children, encouraging their sons to be active and assertive, thereby fostering the development of positive SE, while rearing their daughters to be passive and submissive, thereby fostering relatively low SE. Other parents and communities, by contrast, are more egalitarian in the treatment of their offspring, raising sons and daughters in ways that impact similarly on their development of SE.

Finally, our results suggest that the factors that influence SE in men and women are likely to be far more similar than different. At least with respect to the impact of genetic and temperamental factors on SE, these findings are not consistent with the large body of work suggesting vital differences in the nature of SE in men and women (Swap & Rubin, 1983; Miller, 1986; Eagly, 1987).

Limitations

These results should be interpreted in the context of two potentially significant methodological limitations. First, our sample is limited to a

specific ethnic/cultural group (Caucasians in the southeastern United States). It cannot be assumed that our findings on gender differences in SE would extrapolate to other ethnic/cultural groups.

Secondly, our study is based entirely on twins who may not be representative of the entire population with respect to SE. However, the magnitude of the gender difference seen in this sample is congruent with those detected in a range of non-twin populations (Feingold, 1995).

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