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ORIGINAL ARTICLE

The genetic and environmental relationship between major depression and the five-factor model of personality

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Background. Certain personality traits have long been suspected to reflect an enduring vulnerability to major depression (MD) in part because of shared genetic risk factors. Although many have agreed that normative personality is well captured by the 'Big-Five' personality traits of Openness (O), Conscientiousness (C), Extraversion (E), Agreeableness (A) and Neuroticism (N), to date genetically informative studies have only examined the relationship between MD and N and E.

Method. Questionnaires were completed on a website, yielding a sample of 44 112 subjects including both members of 542 same-sex twin pairs. Personality was measured by the Big Five Inventory. Structural modeling was performed by Mx.

Results. Three of the big-five personality traits – O, E and A – had small phenotypic associations with risk for MD and small genetic correlations. Two traits – N and C – had stronger phenotypic associations (positive for N and negative for C) with the following estimates of the genetic correlation with MD: +0.43 for N and −0.36 for C. N and C were moderately negatively correlated. Controlling for N reduced the genetic correlation between C and MD more than controlling for C reduced the genetic correlation between N and MD.

Conclusions. A large proportion of the genetic risk for MD that is expressed via personality is captured by N, with a modest amount due to C, and small amounts from O, E and A.

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Introduction

A long tradition of research has examined the interrelationship between personality and major depression (MD) with a leading hypothesis being that certain personality traits reflect an enduring vulnerability to MD (Kendler *et al*. 1993*a*; Klein *et al*. 1993; Bagby *et al*. 1995; Enns & Cox, 1997). Given the substantial evidence that genetic factors contribute to both risk for MD (Kendler *et al*. 2006*a*; Sullivan *et al*. 2000) and to variation in personality (Loehlin, 1992; Loehlin *et al*. 1998), it is of particular interest to determine the genetic contribution to the covariation between personality and MD risk.

Some consensus has developed in the last 20 years that human personality can be well accounted for by five personality traits most commonly termed: Openness (O), Conscientiousness (C), Extraversion (E), Agreeableness (A) and Neuroticism (N) (McCrae, 1989; Digman, 1990). However, we are aware of genetically informative studies that have examined the relationship only between MD and neuroticism (N) (Kendler *et al*. 1993*a*, 2006*b*; Fanous *et al*. 2007) and extraversion (E) (Kendler *et al*. 2006*b*). The present study examines, for the first time to our knowledge, the genetic relationship between MD and all of the 'Big-Five' personality traits.

Method

Sample

As outlined in detail elsewhere (Kendler *et al*. 2009), participants in this study were part of data collected from 'Twins: an interactive personality test' from 1 July 2005 to 1 May 2008. This survey was designed as an interactive assessment tool for measures of personality, psychopathology, and substance use and dependence. The website permits any two people,

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regardless of whether they are twins or not, to compare their personalities and behaviors. Participants could take the survey as individuals. All participants were volunteers and were recruited over the world wide web. Potential respondents found out about the site via internet search engines, direct access to its address (http://www.outofservice.com/twins/), or through links from other sites.

Data collection was done with automated computerized administration, data entry and scoring. All participants received individualized feedback after completing the survey. The data presented in this article were collected using a non-commercial, advertisement-free website (www.outofservice.com) that contains personality measures as well as several games, quizzes, and questionnaires for entertainment purposes. Participants did not provide any identifying information and anonymity was assured. This research obtained exempt ethics approval at Virginia Commonwealth University.

We utilized a variety of quality control measures to assess the amount of duplicate or faked responses, or false twin pairs in the sample (Kendler *et al*. 2009). This included examining distributions of our personality measures and finding no excess of extreme scores, examining similarity of reported year of birth, height and weight in twin pairs, and asking and following up the small number of positive responses to an item in our questionnaire about duplicate entries. Consistent with other reports of internet samples (Gosling *et al*. 2004), these investigations suggest quite low levels of faked or duplicate data.

Zygosity was assessed by responses in both twin pairs to the three items found most discriminating when tested against DNA results in the Virginia Adult Twin Study of Psychiatric and Substance Use Disorders (VATSPSUD; Kendler & Prescott, 2006). Pairs, when the responses of the two twins were inconsistent, were eliminated from the study ($n=9$).

Assessments

Personality was assessed by the Big Five Inventory Personality Test (John & Srivastava, 1999), a 44-item scale that assesses O, C, E, A, and N. Responses to these items are recorded by a 5-point Likert scale: (1) strongly disagree, (2) disagree a little, (3) neither disagree nor agree, (4) agree a little, and (5) strongly agree. In the entire sample, these variables were relatively normally distributed with estimates of skewness ranging from -0.50 for A, to $+0.04$ for N.

Lifetime MD was assessed by self-report using a questionnaire validated with a Swedish mailed survey (Kendler *et al*. 1993*b*). This questionnaire contained an expanded version, adapted to self-report, of the section for MD from the Structured Clinical Interview for DSM-III-R (Spitzer *et al*. 1987). To reduce the problem of subjects learning that 'no' responses to probes reduced the length of the questionnaire, the key probe for MD ('Thinking back over your entire life, have you ever had a time when you were feeling depressed, down or sad most of the time for at least two weeks') was asked, along with other key probes, at the start of the questionnaire. For those who responded positively to this probe, later in the questionnaire, 11 additional questions were used to assess the remaining eight symptomatic criteria for MD 'for the time in your life when these feelings of depression were at the worst'. For example, separate items were used for appetite decrease/weight loss *versus* appetite increase/weight gain, and for hypersomnia *versus* insomnia. Three response options were used: 'most of the time', 'sometimes' and 'never'. For these analyses, only items scored as 'most of the time' were considered positive. In addition, to obtain DSM-IV criteria for MD (APA, 1994), subjects had to respond as 'sometimes' or 'most of the time' to the question 'did these feelings interfere with your daily tasks?'

Analyses

The goal of our twin analyses was to decompose the covariance in liability to MD and to the individual personality traits into its genetic and environmental components. We assumed that twin resemblance arises from two latent factors: (i) additive genes (A), contributing twice as much to the monozygotic (MZ) as to the dizygotic (DZ) twin correlation, and (ii) shared or 'common' environment (C), which contributes equally to the correlation in MZ and DZ twins. In addition to this 'common' environment, the model also contains individual-specific environment (E), that reflects measurement error and those environmental experiences which make members of a twin pair different.

Using the software package Mx (Neale *et al*. 2003), we fit models by the method of maximum likelihood to data from all same-sex twin pairs. [The number of available opposite-sex DZ pairs ($n=67$) was too small to give us any realistic power to model qualitative sex effects)]. We used Akaike's Information Criterion (AIC) (Akaike, 1987; Williams & Holahan, 1994) for model selection. The *lower* its value, the better is the balance between explanatory power and parsimony.

Analyzing four twin-zygosity groups enabled us to examine quantitative sex effects, i.e. whether the magnitude of genetic effects on MD and personality are the same in males and females. From the best-fit models, we were able to estimate correlations in the genetic and environmental risk factor for MD and

Table 1. *Results of model fitting and parameter estimates for bivariate twin models of the relationship between lifetime major depression and the personality traits of openness, conscientiousness, extraversion, agreeableness, and neuroticism*

Variable	Quantitative sex effects	Parameters	Δ df	Openness	Conscientiousness	Extraversion	Agreeableness	Neuroticism
AIC	Y	ACE	–	129.6	162.7	262.1	124.8	157.4
AIC	N	ACE	9	113.7	152.0	248.3	111.5	141.7
AIC	N	CE	12	135.4	153.5	257.9	118.7	147.3
AIC	N	AE	12	108.4*	146.7*	242.3*	105.7*	135.9*
a^2	Personality			**0.64**	**0.44**	**0.46**	**0.44**	**0.45**
95% CI				*0.56* to *0.69*	*0.35* to *0.52*	*0.37* to *0.53*	*0.35* to *0.52*	*0.37* to *0.53*
a^2	MD			**0.59**	**0.59**	**0.59**	**0.59**	**0.59**
95% CI				*0.46* to *0.71*	*0.46* to *0.71*	*0.46* to *0.71*	*0.46* to *0.71*	*0.46* to *0.71*
r_a				**+0.17**	**−0.36**	**−0.06**	**−0.18**	**+0.43**
95% CI				*+0.01* to *+0.32*	*−0.55* to *−0.17*	*−0.24* to *+0.13*	*−0.37* to *+0.02*	*+0.26* to *+0.59*
r_e				**+0.13**	**+0.09**	**−0.09**	**−0.05**	**+0.29**
95% CI				*−0.04* to *+0.30*	*−0.08* to *+0.25*	*−0.26* to *+0.07*	*−0.21* to *+0.12*	*+0.13* to *+0.43*

AIC, Akaike's Information Criterion (Akaike, 1987); A, additive genetic effects; C, common or shared environmental effects; E, unique environmental effects; a^2, heritability; r_a, genetic correlation; CI, confidence interval.

Estimates in bold and 95% confidence intervals in italic.

* Best-fit model.

personality. For example, a genetic correlation of unity between MD and a particular personality dimension would mean that the same genetic risk factors contribute to variation in the personality trait and liability to MD. An individual-specific environmental correlation of zero would mean that the environmental risk factors for MD and that particular personality dimension were independent of one another.

Results

The sample contained 44112 completed questionnaires with unique user codes of which 65.3% were female, 85.4% aged ⩾18 years, and 72.0% Caucasian. The lifetime prevalence of DSM-IV (APA, 1994) MD in this sample was 32%. The bi-serial correlation between lifetime MD and the individual personality traits were: O +0.14; C −0.23; E −0.16; A −0.15 and N +0.43.

Our twin modeling included 542 same-sex twin pairs of the following composition: 364 female MZ, 80 female DZ, 77 male MZ, and 21 male DZ. Table 1 depicts the results of bivariate twin model fitting. We began with a full ACE model that allowed for quantitative sex effects. Next we fitted an ACE model without quantitative sex effects. The AIC improved with this step for all the bivariate models for personality and MD, indicating that no significant differences are observed in any parameters of the models between males and females. Next, we compared the fits of a CE and an AE model. For all of the analyses, the CE model produced deterioration in the AIC compared to the ACE model. By contrast, the AE model consistently produced an improvement in the AIC, and proved the overall best-fit model. Table 1 depicts the heritability estimates for the personality dimensions and MD from the AE model, along with the 95% confidence intervals (CIs). The heritability of the big-five personality dimensions ranged from 0.44 for conscientiousness and agreeableness to 0.64 for openness. Despite our model sample size, the CIs of these estimates were reasonably tight as would be expected given the quantitative nature of these variables (Neale *et al*. 1994). In all models, the heritability of MD was estimated at 0.59 and this was known less precisely as expected given that this is a dichotomous trait (Neale *et al*. 1994).

Table 1 also depicts the estimates from the best-fit AE models for r_a and r_e along with their CIs. The absolute value of the genetic correlation between personality and MD was strongest for N (+0.43) followed by C (−0.36). Weaker genetic correlations were observed between MD and A (−0.18), O (+0.17) and E (−0.06). CIs for the genetic correlations between MD, and A and E included zero.

For N, A and O, r_e was in the same direction as r_a but weaker. For C and MD, while the genetic correlation was negative, the environmental correlation was positive. Only for E was the environmental correlation larger than the genetic correlation. However, only for N and MD did the CIs for the environmental correlation exclude zero.

In the entire sample, four of the 10 inter-correlations between the five personality traits were ⩾0.30: A and C (+0.35), A and N (−0.34), N and C (−0.31), and N and E (−0.30). The negative phenotypic correlation

between N and C raises the question of whether the genetic correlation between C and MD results in part from the shared variance between N and C. To address this question, we compared the results of bivariate Cholesky decomposition models (N/MD and C/MD) with trivariate models (C/N/MD and N/C/MD). In this context, these models can be best understood as the twin modeling equivalent of a multiple regression analysis. Comparing the best-fitting results from these models (all of which were AE), allowed us to determine that 53% of the genetic effect of N on MD persisted after accounting for the effects of C. By contrast, 29% of the genetic effect of C on MD persisted after the effects of N were taken into account.

Comment

The goal of this paper was to provide a comprehensive view, from a genetic epidemiological perspective, of the association between lifetime MD and normative personality variation. Our results indicate that with respect to their level of association with MD, the big-five personality traits can be divided into two groups. The first group contains three traits: O, E and A. All of them had small phenotypic associations with risk for MD – positive for O, and negative for E and A. Estimates of the genetic correlation (r_a) from the best-fit twin model were also quite small ($+0.17$, -0.06 and -0.18, respectively). In our moderate-sized twin sample, two of these correlations (for E and A) were not statistically significant, while the 95% CIs for the r_a for MD and O just barely excluded zero. These results suggest that genetic factors which influence levels of O, E and A have at most a quite small impact on risk for MD.

The second group of personality traits – N and C – had stronger phenotypic associations for risk for lifetime MD that was positive for N and negative for C. Estimates of the genetic correlation from our best-fit twin model were statistically significant and moderate: $+0.43$ for N and -0.36 for C. These findings indicate that genetic factors which influence N and C have an appreciable overlap with those factors impacting on risk for MD. N and C had a moderate inverse phenotypic correlation in our sample. Using bivariate and trivariate Cholesky decomposition analyses – which function here as a genetic form of multiple regression – we found that the strength of the genetic relationship between C and MD declined much more with the inclusion of N in the model than the relationship between N and MD declined when C was included in the model. Thus a substantial proportion of the relationship between C and MD resulted from the shared variance with N. Put another way, the proportion of the variance in C that was *not* shared with N was only quite modestly related to risk for MD. In sum, our results suggest that a large proportion of the genetic risk for MD which is expressed via personality is captured by N, with only modest amounts resulting from C and even less from the other three personality traits that make up the big five.

The results from this study are reassuringly similar to those previously obtained in a very large longitudinal Swedish study (Kendler *et al*. 2006*b*). The best-fit models applied to that sample estimated the genetic correlation between MD and N to be $+0.46$ in women and $+0.47$ in men *versus* our estimate of $+0.43$. The parallel estimates from the Swedish sample for the genetic correlation between MD and E were -0.10 and -0.15 compared to our estimate of -0.06. Our estimated heritability for MD (59%) is somewhat higher than that obtained in the most recent meta-analysis (37%) although the CIs of these two estimates nearly overlap (Sullivan *et al*. 2000). Our heritability estimates for four of the big-five factors (C, E, A, N) – within the narrow range of 44–46% – are well within the range commonly found for personality traits in prior general population studies (e.g. Tellegen *et al*. 1988; Bouchard, 1993; Riemann *et al*. 1997). Our heritability estimate for O (59%) is somewhat higher than that typically seen although not so different from an estimate for O of 53% found in a German twin sample (Riemann *et al*. 1997).

Given personality assessment by self-report is easy, cheap and does not require a trained interviewer, some have argued that gene finding for MD might profitably employ personality as an 'intermediate phenotype'. Our results suggest that if this strategy is to be pursued for MD, N is far and away the best personality trait to assess. Investigators have already 'voted with their feet' in this regard, with a number of efforts published and underway to detect risk genes for N (e.g. Fullerton *et al*. 2003; Neale *et al*. 2005; Kuo *et al*. 2007; Shifman *et al*. 2008). Our findings provide evidence that a modest proportion of genes which influence C might also impact on risk for MD, but the personality dimensions of O, A and E are probably not profitable to pursue as endophenotypes for MD.

Limitations

These results should be interpreted in the context of four potential methodological limitations. First, while diverse, the sample of subjects completing the web-based assessment is unlikely to be entirely representative of the general population. However, a recent review of the psychological literature suggests that fears about the unrepresentativeness of web-based samples are exaggerated (Gosling *et al*. 2004). Second, this was a cross-sectional study and we cannot rule

out that part of the relationship between MD and personality resulted from transient state effects of current levels of depression or scar effects of prior episodes. A prior longitudinal twin study of MD and N performed in the VATSPSUD revealed a 'scar' effect but this accounted for a quite small proportion of the total covariance between the two traits (Kendler *et al*. 1993*a*). Third, although we set a high threshold for each individual symptomatic criterion (requiring it to be present for at least 2 weeks 'most of the time'), and required depression-impairment, the lifetime prevalence of MD in our sample was higher than that seen in most epidemiologic samples. However, these rates for MD are not out of keeping with those found in the VATSPSUD (34.4% in women, 28.5% in men), or in the Christchurch longitudinal sample where 37% of individuals met criteria for MD at least once by the time of early adulthood (Wells & Horwood, 2004). Our sample is relatively young and both the non-twin and twin proportions of the sample were predominantly female (64.8% and 82.1%, respectively) which also would increase rates of MD. Finally, the rates of MD could be upwardly biased by self-selection into the website or because the anonymous method of assessment encourages more accurate responding in the presence of social desirability biases (Kissinger *et al*. 1999; Garb, 2007). Fourth, we could not verify the identity of the twin pairs, so it was possible that subjects who were not twins could have participated. As noted above, several checks of the data did not suggest high rates of faking. Most convincingly, we included in our survey questions about height and weight and obtained the following correlations: height MZ +0.90, DZ +0.51; weight MZ +0.87, DZ +0.47. These results are quite comparable with those found in prior twin studies as exemplified by the results from a recent epidemiological study of over 3300 Swedish twin pairs: height MZ +0.93, DZ +0.53; weight MZ+0.87, DZ +0.44 (Silventoinen *et al*. 2008). We also examined similarity of reported date of birth and found only a small number of normally distributed disagreements with no evidence for an excess of widely divergent birthdates. From these data, we conclude that the proportion of 'faux' twin pairs in our sample is quite small.

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Declaration of Interest

None.

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