

The Eysenck personality factors: Psychometric structure, reliability, heritability and phenotypic and genetic correlations with psychological distress in an isolated Croatian population

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Received 15 February 2006; received in revised form 30 May 2006; accepted 14 June 2006

Available online 7 September 2006

Abstract

We report the psychometric structure of a Croatian translation of the Eysenck Personality Questionnaire-Revised (short-form), its correlations with psychological distress (General Health Questionnaire-30), its heritability, and personality–psychological distress genetic correlations. The setting is a large (≈ 1000), family-based sample of men and women from an isolated Croatian island. The neuroticism and extraversion traits and the lie scale showed good psychometric characteristics. The translated psychoticism scale was unsatisfactory in this sample. It had a very low internal consistency, probably due in part to heavily biased

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item responses. There were significant additive genetic contributions to variation in neuroticism, extraversion, and psychological distress. Psychological distress had a very high genetic correlation with neuroticism, and a moderate genetic correlation with extraversion.

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Keywords: Personality; Neuroticism; Extraversion; Depression; EPQ; GHQ; Heritability; Genetics; Croatia

1. Introduction

There is a growing consensus about the validity of human personality traits as important dispositions toward feelings and behaviours (Matthews, Deary, & Whiteman, 2003). Here we examine the Eysenck Personality Questionnaire-Revised, short-form, which includes the traits of neuroticism, extraversion and psychoticism, and a lie scale (Eysenck, Eysenck, & Barrett, 1985). Neuroticism and extraversion, especially, appear in most trait models of personality (Matthews et al., 2003). An important part of the validation of any trait-based model of personality and its associated measurement instrument is to investigate its applicability to other cultures. This tends to be done in two ways: emic and etic. Emic research typically uses the lexicon of the local culture to investigate the structure and content of the personality-related terms (Saucier & Goldberg, 2001). Etic research applies personality measures devised in one culture to new cultures and asks whether they show the same psychometric structure and reliability and validity (McCrae, 2001).

A large amount of etic research has been completed on the Eysenck Personality Questionnaire. The research has been done mostly on the original 90-item EPQ. Generally, its psychometric structure has been well-reproduced in at least 34 countries (Barrett & Eysenck, 1984; Barrett, Petrides, Eysenck, & Eysenck, 1998). Here we apply the 48-item short-form of the EPQ-Revised in a new setting.

There is great interest in discovering the genetic contributions to common, complex diseases (e.g., Davey Smith et al., 2005; Lohmueller, Pearce, Pike, Lander, & Hirschhorn, 2003). One such group of illnesses is states of anxiety and depression, which form a major cause of medical consultation and a large burden of morbidity in the population. Genetic contributions are likely to be polygenic, i.e. with many genes each contributing a small effect (Hirschhorn & Daly, 2005). Moreover, a likely useful route to discovering the genetic contributions to common disorders is to examine the genetic bases of quantitative traits which act as risk factors for them (Flint & Mott, 2001). Among students, Neuroticism from the Eysenck Personality Questionnaire-Revised correlated with total scores on the General Health Questionnaire-28 (which measures anxiety and depression) at 0.54 for men ($N = 347$) and 0.52 for women ($N = 550$) (Stewart, Ebmeier, & Deary, 2005). In the same samples the correlations with Extraversion were -0.26 and -0.21 , respectively. Thus, for states of low mood like anxiety and depression, the personality trait of neuroticism is a major target for investigation (Boomsma et al., 2000; Flint et al., 1995; Jardine, Martin, & Henderson, 1984; Kirk et al., 2000; Levinson, 2006; Middeldorp, Cath, Van Dyck, & Boomsma, 2005; Nash et al., 2004; Sham et al., 2000). The extensive review of twin and family studies conducted by Middeldorp et al. (2005) concluded that the comorbidity of anxiety and major depressive disorders was in part due to genetic factors associated with the personality trait of

neuroticism. A likely contributor is genetic variation influencing the serotonin transporter length polymorphism, but it has not been replicated in large studies (Willis-Owen et al., 2005). In the present study we shall examine the genetic correlation between personality traits and psychological distress.

Here we examine a large sample of Croatian people living in a small, isolated island with relatively stable communities. The study of isolate populations aims to take advantage of increased genetic and environmental homogeneity compared with predominantly urban populations (Vitart et al., 2006). This can facilitate gene mapping but has the potential disadvantages of reducing the diversity of genetic influences and increasing the extent of shared environmental influences, which may be particularly important for personality traits. We apply the Eysenck Personality Questionnaire-Revised (short-form) to this new group. We examine its psychometric structure, internal consistency, sex differences, heritability, and phenotypic and genetic relationship to psychological distress.

2. Method

2.1. Sample

Adult subjects living in the villages of Komiza and Vis on the Croatian island of Vis were recruited in May 2003 and May 2004 for a large genetic study. Croatia has 15 Adriatic Sea islands with populations greater than 1000. The villages on the islands have unique population histories and have preserved their isolation from other villages and the outside world through many centuries. The history, demography and genetic structure of these villages have been investigated for more than 50 years (Rudan, Campbell, & Rudan, 1999; Rudan et al., 1999). Participants underwent a medical examination and interview, led by research teams from the Institute for Anthropological Research and the Andrija Stampar School of Public Health, Zagreb, Croatia. Informed consents, procedures and questionnaires were reviewed and approved by relevant ethics committees in Scotland and Croatia. All individuals over 18 years old and resident on the Island of Vis were invited to participate in this study. Volunteers attended an early morning clinic where fasting blood samples were collected and various physiological quantitative traits were measured. Blood samples were also collected for DNA extraction and plasma and serum samples were aliquoted and stored for future measurement of biochemical quantitative traits. They then completed a series of questionnaires relating to family and medical history as well as lifestyle and diet. As a part of the interview participants also completed the Eysenck Personality Questionnaire-Revised (short-form; EPQ-R) and the General Health Questionnaire 30 (GHQ). Seventy percent of the villages' adult population took part in the study, a total of 1030 individuals (427 men, 603 women), 9 of whom have no EPQ-R or GHQ data. The mean age was 56.1 years ($SD = 15.6$), and ranged from 18 to 93 years. Five hundred and eighty eight individuals could be placed in 125 pedigrees (the largest of which links 134 phenotyped individuals and has a depth of six generations). The following pairs have been phenotyped for personality traits: 210 parent–child, 128 full sibs, 12 half-sibs, 127 cousins, 36 grandparent–grandchild, and 119 avuncular. Thus, most of the information to estimate genetic parameters is derived from the observed resemblance between parents and offspring and from the observed variation between and within nuclear families.

2.2. Eysenck Personality Questionnaire-Revised (short-form)

This is a self-reported questionnaire (Eysenck et al., 1985). It has 48 items, 12 for each of the traits of neuroticism, extraversion, and psychoticism, and 12 for the lie scale. Each question has a binary response, ‘yes’ or ‘no’. For the present study the questionnaire was translated into Croatian. It was then back-translated independently. The back-translated (English) and original English versions were compared by IJD and IR (who is fluent in both Croatian and English) and two additional researchers (Lada Jamnicki, MD and Martina Niksic, PhD), a Scot with Croatian as a second language, and a Croat with English as a second language, who were not involved in the original translation of the items. This content-based checking and the item-level factor analysis (see below) provided clear support for scoring the neuroticism, extraversion and lie scale items as suggested by Eysenck et al. (1985). Each dichotomous item was scored 1 or 0, and each scale had a maximum possible score of 12 and minimum of zero.

2.3. General Health Questionnaire 30

This is a 30-item, self-reported questionnaire that asks about recent psychological distress (Goldberg & Williams, 1988). Each question has four response options. It was back-translated using the same method as the EPQ-R. This content-based checking and the item-level factor analysis (see below) provided clear support for using a total score based upon responses to all of the items. Each item was scored from 1 to 4, and the total GHQ score thus had a maximum possible score of 120 and minimum of 30.

2.4. Statistical analyses

Factor analysis of the EPQ-R was done using the principal factors method in the SAS statistical package, ignoring the genetic relationships among the individuals. Tetrachoric correlations were used because of the binary response format of the questions. Factor analysis of the GHQ was done using the principal factors method in SPSS version 14. There are some missing data for EPQ-R and GHQ, and this is indicated in the numbers available for the analyses.

The genetic parameters (variance components and genetic correlations) were estimated by fitting univariate or bivariate linear mixed models (Lynch & Walsh, 1998) using the restricted maximum-likelihood method implemented in ASReml (Gilmour et al., ASReml User Guide Release 1.0, VSN International Ltd., Hemel Hempstead, UK). We use all phenotypic information efficiently by fitting the complete kinship matrix pertaining to the entire sample. If \mathbf{y} denotes the vector of traits observations, the univariate linear mixed model can be written in matrix notation as: $\mathbf{y} = \mathbf{X}\boldsymbol{\tau} + \mathbf{Z}\mathbf{u} + \mathbf{e}$, where $\boldsymbol{\tau}$ is the vector of fixed effects, \mathbf{X} the associated design matrix, \mathbf{u} the vector of the fitted random additive genetic effects, \mathbf{Z} the associated design matrix, and \mathbf{e} the vector of residual errors, with \mathbf{u} and \mathbf{e} distributed as multivariate normal densities with means 0 and variances to be estimated. To take account of the genetic relationships between all individuals in the sample we fit the numerator relationship matrix \mathbf{A} , which is equal to twice the kinship matrix (Lynch & Walsh, 1998). Hence, we assume that $\mathbf{u} \sim \text{MVN}(\mathbf{0}, \mathbf{A}\sigma_A^2)$. Therefore, all relationship data is retained with the full pedigree information used by fitting the full relationship matrix in the model. This model is equivalent to the standard polygenic ‘null’ model for QTL mapping

in complex pedigrees (Almasy & Blangero, 1998; George, Visscher, & Haley, 2000). The natural logarithm of the GHQ phenotype scores was used to improve normality. Sex and age were fitted as fixed effects (already explained above). In addition to the additive genetic effects, we also fitted a random ‘mother effect’ corresponding to the effect common to all individuals who share the same biological mother. This ‘mother effect’ is a composite of shared environment, maternal and dominance genetic effects as it is the full-sibs who contributed mostly to its estimate. When a mother effect was fitted, the full model was $y = X\tau + Zu + Z_m m + e$, with m the random effect associated with the mother and Z_m the matrix linking the phenotypes to the mother effects. We assumed that the different mother effects were uncorrelated.

The statistical significance of an estimated variance component was determined by a likelihood ratio test (LRT), in which the obtained likelihood for the full model was compared to the likelihood of the nested model, in which the variance component was constrained to be zero. Under the null hypothesis of no difference between the two models, the distribution of the LRT statistic is a 1/2:1/2 mixture of a point mass at zero and a χ^2 distribution with one degree of freedom (Self & Liang, 1987).

3. Results

Four orthogonally rotated factors from the principal axis factor analysis of the EPQ-R are shown in Table 1. The analysis resulted in the following first six eigenvalues: 8.63, 6.60, 4.57, 2.64, 2.32, 1.59. Thus, a scree slope criterion might suggest three or five factors. However, the Eysenck scale is so well-replicated at the item level that we extracted four. As will become clear, though, there are three reliable factors in this analysis. The items associated with the neuroticism and extraversion traits and lie factors all have high loadings on the expected factors, with almost no substantial cross-loadings on the other factors. The exception is the psychoticism factor. Fewer than half of its items have large (>0.50) loadings on the expected factor, and seven of the 12 items have their highest loadings on non-psychoticism factors. The Cronbach alpha (internal consistency) coefficients were as follows: neuroticism = 0.82; extraversion = 0.78; psychoticism = 0.26; and lie = 0.78. Therefore, the psychometric analyses show that the neuroticism, extraversion and lie scales perform well in this sample, but not the psychoticism scale.

The GHQ had a Cronbach alpha of 0.92. Principal axis factoring of the GHQ items in this sample reveals that the first six eigenvalues are: 8.97, 1.58, 1.04, 0.67, 0.50, 0.42. Therefore, the use of a single total score is well-supported here.

There were relatively large sex differences in neuroticism (Cohen’s $d = 0.49$) and GHQ (Cohen’s $d = 0.45$), with women scoring higher (Table 2). Men scored slightly higher on extraversion (Cohen’s $d = 0.17$). Women scored higher on the lie scale (Cohen’s $d = 0.37$). Age correlated strongly positively with the lie scale and less so with neuroticism and GHQ, and had a modest negative correlation with extraversion (Table 2).

The GHQ total score had a strong positive correlation with neuroticism, a modest negative correlation with extraversion, and a near-to-zero correlation with the lie scale (Table 2). There was a modest negative association in this sample between neuroticism and extraversion ($r = -0.27$, $p < 0.001$). Therefore, to check that this was not the source of the correlation between extraversion and GHQ a partial correlation analysis was conducted. The partial correlation between

Table 1

Principal factor analysis of the items in the Eysenck Personality Questionnaire-Revised, short-form

Item number ^a	Designated trait in the EPQ-R	Factor 1	Factor 2	Factor 3	Factor 4
1	N	0.77	−0.07	−0.08	−0.16
2	P	0.57	−0.08	−0.07	0.13
3	E	−0.02	−0.01	0.74	0.02
4	L	−0.03	−0.51	0.14	0.12
5	N	0.66	0.10	−0.18	0.23
6	P	0.13	0.00	−0.07	0.86
7	E	0.01	−0.05	0.75	−0.19
8	L	0.12	0.71	0.06	0.09
9	N	0.56	0.28	0.06	−0.03
10	P	0.17	0.33	0.12	0.73
11	E	−0.07	−0.07	0.66	0.22
12	L	0.07	0.60	0.01	0.01
13	N	0.53	0.04	−0.12	0.10
14	P	0.21	0.29	0.08	−0.22
15	E	−0.34	−0.06	0.68	−0.02
16	L	0.06	−0.57	0.08	−0.01
17	N	0.73	−0.05	−0.11	−0.13
18	P	0.02	−0.49	0.05	0.16
19	E	0.14	−0.12	0.50	−0.02
20	L	−0.10	0.78	0.09	0.06
21	N	0.76	−0.03	−0.00	−0.06
22	P	0.31	0.24	0.12	−0.30
23	E	−0.06	0.01	0.66	0.10
24	L	−0.15	0.74	0.08	−0.02
25	N	0.69	−0.18	−0.19	0.18
26	P	−0.28	−0.03	0.52	0.41
27	E	0.31	−0.15	−0.73	0.03
28	P	0.24	0.06	−0.00	0.54
29	L	−0.01	0.79	0.09	0.08
30	N	0.85	−0.02	−0.03	0.03
31	P	0.25	−0.09	0.12	0.12
32	E	−0.26	−0.05	0.64	0.59
33	L	0.03	0.65	0.05	−0.06
34	N	0.59	−0.16	−0.08	0.28
35	P	0.02	−0.07	0.05	0.23
36	E	0.01	0.20	0.60	0.03
37	L	−0.03	0.72	0.18	0.05
38	N	0.75	0.06	−0.12	−0.07
39	P	0.44	0.17	0.07	−0.10
40	L	−0.09	0.88	0.06	0.02
41	E	0.34	−0.19	−0.58	0.15
42	N	0.68	−0.05	−0.38	−0.03
43	P	0.03	−0.32	0.01	0.32
44	E	0.04	0.02	0.71	−0.04
45	L	−0.03	−0.62	0.04	0.02
46	N	0.49	0.03	−0.13	0.17
47	L	0.05	0.60	−0.00	−0.01
48	E	−0.24	0.23	0.63	0.16

Loadings shown in bold apply to those items that were intended to act as factor indicators on the EPQ-R.

^a The numbers attached to the items are the numbers given by Eysenck et al., 1985, p. 29, where the full content of each item in English is available.

Table 2

Sex differences and correlations with age and GHQ for the EPQ-R traits of neuroticism and extraversion and the lie scale

Trait or GHQ	Male ^a	Female ^b	<i>t</i> for sex difference	Correlation with age ^c	Correlation with GHQ ^d
Neuroticism	4.4 (3.2)	6.0 (3.3)	−7.5*	0.18*	0.54*
Extraversion	8.5 (2.6)	8.0 (2.8)	2.6*	−0.23*	−0.23*
Lie	8.0 (3.0)	9.0 (2.5)	−5.9*	0.50*	0.01
GHQ	55.4 (10.1)	60.3 (11.9)	−6.9*	0.17*	–

Columns 2 and 3 are mean (SD).

^a *N* = 408–422.

^b *N* = 575–596.

^c *N* = 983–1000.

^d *N* = 978–995.

* *p* < 0.01.

extraversion and GHQ was $r_{E-GHQ-N} = -0.22$ ($N = 957$, $p < 0.001$). In a linear regression model of GHQ, neuroticism and extraversion contributed significant independent variance to GHQ: 30% and 3.3%, respectively (both $p < 0.001$). When the regression analysis was repeated for each sex separately, the results were very similar.

The proportion of the total variance contributed by additive genetic effects was significant for all traits except psychoticism, as follows: neuroticism = 0.24; extraversion = 0.41; and GHQ = 0.18 (Table 3). A ‘mother effect’ was also fitted but was non-significant for all three measures, with estimated associated variance components contributions of 0.08 (SE = 0.09; $p = 0.17$) for extraversion, 0.024 (SE = 0.1, $p = 0.42$) for neuroticism, and 0.045 (SE = 0.09, $p = 0.3$) for GHQ. Similar estimates were obtained using a maximum likelihood method implemented in

Table 3

Additive genetic contributions to variation in EPQ-R neuroticism and extraversion, and GHQ

Trait or GHQ	Covariates	Mean effect of covariate (SE)	Additive genetic heritability (SE)	<i>p</i> -Value for LRT ^a
Neuroticism Mean = 5.38	Age Sex ^b	0.04 (0.007) 1.57 (0.21)	0.24 (0.11)	0.02
Extraversion Mean = 8.21	Age Sex	−0.04 (0.005) −0.42 (0.17)	0.41 (0.10)	<0.00001
ln (GHQ) Mean = 4.05	Age Sex	0.002 (0.0003) 0.08 (0.01)	0.18 (0.10)	0.04
Neuroticism	Age Sex ln (GHQ)		0.118 (0.11)	0.15
Extraversion	Age Sex ln (GHQ) EPQ-R-N		0.39 (0.11)	0.0001

^a The null hypothesis in the likelihood ratio tests is that of no additive genetic effects (or defined h^2).

^b Sex effect given as female versus male.

Table 4

Bivariate genetic analysis of EPQ-R (neuroticism, extraversion) and GHQ

Trait	Covariates	Genetic correlation (SE)	Phenotypic correlation (SE)	Environmental correlation
ln (GHQ) and neuroticism	Age, sex	0.91 (0.26)	0.52 (0.02)	0.42
ln (GHQ) and extraversion	Age, sex	−0.37 (0.26)	−0.27 (0.03)	−0.24
Extraversion and neuroticism	Age, sex	−0.41 (0.24)	−0.22 (0.03)	−0.14
ln (GHQ) and extraversion	Age, sex, neuroticism	−0.23 (0.45)	−0.19 (0.03)	−0.18

SOLAR (Almasy & Blangero, 1998). Therefore, subsequent results are from models that did not fit mother effects. For neuroticism (EPQ-R-N), the variance component attributable to additive genetic effects became non-significant after adjustment for GHQ, suggesting that these two measures are influenced by shared genes. On the other hand, for extraversion the additive variance component is still highly significant even after adjustment for GHQ or neuroticism, suggesting a non-shared genetic contribution. In agreement with this observation, when a bivariate analysis was performed, there was a very high genetic correlation between neuroticism and GHQ, $r_g = 0.91$, and modest genetic correlations between extraversion and GHQ, $r_g = -0.37$, and between neuroticism and extraversion, $r = -0.41$ (Table 4).

4. Discussion

The neuroticism and extraversion scales had good psychometric characteristics, but not the psychoticism scale, which was not used further here. Sex differences and associations with age were as expected for neuroticism and extraversion. Indeed, despite the translation of the items, even the means and SDs were similar to the UK values for neuroticism and extraversion (Eysenck et al., 1985). The additive genetic contributions to these traits were within the range of those found in the literature (Matthews et al., 2003). The genetic correlation between neuroticism and psychological distress measured using the GHQ was very high.

Genetic isolates have been very valuable for the mapping of rare genetic diseases and are believed to offer equal advantages for unravelling the genetics of more common complex diseases. These populations have been shown to exhibit increased levels of linkage disequilibrium, which is favourable for gene mapping (Service et al., 2006). In addition, preliminary genetic studies using genetic markers in the communities on the Eastern Adriatic islands in Dalmatia have indicated reduced genetic diversity within the island populations surveyed in comparison to the Croatian general population (Vitart et al., 2006). In tandem with these studies and to provide a foundation for gene mapping studies, it is important to study the distribution of quantitative traits that underline common complex disease and the performance of standard instruments used to measure these traits.

The heritability estimates found here are remarkably congruent with other estimates for extraversion and neuroticism (Bouchard, 2004; Keller, Coventry, Heath, & Martin, 2005; Lake, Eaves, Maes, Heath, & Martin, 2000). Despite this being a population isolate, the heritability estimates for the neuroticism and extraversion EPQ-R components are similar to those published from more diverse populations. This suggests that the potential disadvantages of using isolates in

behavioural research are small. Family-based studies provide an upper limit for the heritability since they may include a component in the resemblance between relatives which is due to shared environment. The latter can be more readily estimated in adoption or twin studies, especially where the latter are reared apart (McGue & Bouchard, 1998; Stoolmiller, 1999). Our attempts to fit mother effect which presumably is a proxy for shared household effect suggested that this effect is low. This is in agreement with twins studies of much larger sample size (Eaves, Heath, Neale, Hewitt, & Martin, 1998; Keller et al., 2005). Interestingly, these large scale studies additionally underlined a significant non-additive genetic contribution to variation underlying personality traits, most likely due to additive by additive epistasis. Our present study is too small to unravel such effects. The high genetic correlation between neuroticism and psychological distress was in close agreement with the review of data from other cultures where it is found that neuroticism has a high genetic correlation with anxiety and depression (Middeldorp et al., 2005). Our results also suggested that the correlation between the additive genetic effects is much larger than the correlation between the environmental effects.

The trait of neuroticism is the most-studied risk factor for anxiety and depression. In this sample we found that extraversion added significant additional variance to psychological distress. The correlations between GHQ and neuroticism (positive) and extraversion (negative) here are similar to those in a large UK sample of students (Stewart et al., 2005). In addition to extraversion's being a replicated negative correlate of psychological distress, independent of neuroticism, it was found here to have a negative genetic correlation with GHQ. Therefore, genetic studies of psychological distress should focus on extraversion in addition to neuroticism as a risk factor.

There are some reasons why the psychoticism items might not have performed well here. First, the distribution of responses was suboptimal. For half of the items, one of the two responses received fewer than 10% endorsements: Q6 = 7%; Q10 = 2%; Q18 = 4.6%; Q22 = 8.1%; Q26 = 7.2%; and Q39 = 3.6%. Second, some of the psychoticism questions were inappropriate. For example, one of the questions is about insurance, and there was no insurance in Croatia at that time. It should also be noted that Heath and Martin's (1990) large, multivariate genetic analysis found problems with the coherence of the psychoticism scale's items.

Acknowledgements

The field work was funded by a Medical Research Council Human Genetics Unit research grant to AW, AC, IR and Andrew Carothers. We thank Lada Jamnický and Martina Niksic for their help with back-translation of the questionnaires from Croatian to English, and Sue Brotherstone for her help with ASReml. We thank Geoff Der for assistance with the factor analysis of the EPQ-R. IJD is the recipient of a Royal Society-Wolfson Research Merit Award. VV is a Medical Research Council fellow.

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