Genetic Simplex Modeling of Eysenck's Dimensions of Personality in a Sample of Young Australian Twins

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The relative stability and magnitude of genetic and environmental effects underlying major dimensions of adolescent personality across time were investigated. The Junior Eysenck Personality Questionnaire was administered to over 540 twin pairs at ages 12, 14 and 16 years. Their personality scores were analyzed using genetic simplex modeling which explicitly took into account the longitudinal nature of the data. With the exception of the dimension lie, multivariate model fitting results revealed that familial aggregation was entirely explained by additive genetic effects. Results from simplex model fitting suggest that large proportions of the additive genetic variance observed at ages 14 and 16 years could be explained by genetic effects present at the age of 12 years. There was also evidence for smaller but significant genetic innovations at 14 and 16 years of age for male and female neuroticism, at 14 years for male extraversion, at 14 and 16 years for female psychoticism, and at 14 years for male psychoticism.

Eysenck has argued extensively for a trait theory of personality in which personality dimensions are best explained as stable constructs across the life span (Eysenck & Eysenck, 1980; Eysenck & Eysenck, 1985). An important question is the degree to which the observed phenotypic stability in personality is determined by genetic and environmental effects.

McCrae and Costa (1990) found that beyond the age of 30, the means of cross-sectional and longitudinal measures of personality show little or no change, with retest correlations ranging from .55 to .85. Their conclusion is supported by Conley (1984) in their extensive review of longitudinal studies of intelligence, personality and social attitudes. They demonstrated that personality can be regarded as a relatively stable construct across the adult lifespan. Watson and Clark (1984) in a review of 55 studies examining measures of negative affect, similar to Eysenck's Neuroticism Scale, found that retest correlations were typically highest (> .80) in the first five months but then declined and levelled off (.50–.60) over the next 5 to 6 years. More recently, Kirk and colleagues (2000) analyzed data from the Australian Twin Registry to examine the retest correlations between the EPQ (Eysenck & Eysenck, 1975) and EPQ-R Neuroticism Scale scores (Eysenck et al., 1985) over a 19-year interval, as well as the retest correlations between long and short versions of the EPQ-R Neuroticism Scale (Eysenck et al., 1985) over a ten-year interval. Despite differences in administration procedures (e.g., questionnaire vs. interview), the 19-year retest correlations ranged from .53 to .87. These correlations are slightly higher than those reported by Ormel and Rijsdijk (2000), who used a Dutch neuroticism scale based on the Maudsley Personality Inventory (Eysenck, 1959) to report retest correlations over a 16-year interval ranging from .40 to .73. Taken together, the above correlations not only contradict Mischel's conclusion that measures of consistency in personality rarely produce retest correlations above .30 (Eysenck & Eysenck, 1985), but also demonstrate convincingly that measures of personality, such as neuroticism, are indeed stable constructs.

With regard to genetic continuity, Eaves and Eysenck (1976) have reported that variation within subjects over a two-year period between tests of neuroticism was due entirely to environmental factors specific to individuals. Despite methodological limitations (Eaves et al., 1998; Lake et al., 1999), Eaves and colleagues (1989) have reported very high genetic correlations between adult and juvenile measures of extraversion and neuroticism, .84 and .44 respectively. The same authors also investigated the stability of gene expression in measures of adult personality taken at different ages. They predicted that relatives of the same degree will correlate less as the age difference increases because the phenotype is to some extent affected by age-specific gene expression. In the case of twins, the same genes are always being expressed because they

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are identical in age. Dizygotic twins will correlate more highly than siblings, who in turn will correlate more highly than parents and offspring. In other words, 'the effects of development result in greater differences in gene expression between relatives who are further apart in age' (Eaves et al., 1989, p. 195). Based on a small cross-sectional sample of 178 families comprising twins, nuclear families, extended kinships and adoptive families, the authors calculated that the decline in the genetic component of the covariance between relatives for every 10 years' difference in age was only 3% and 12% for extraversion and neuroticism respectively. The authors concluded that (i) there was little to support the idea that different genes were expressed at different ages in adults; (ii) the effects were strongest for neuroticism and extraversion; and (iii) any apparent change in adult gene expression was more likely to be a function of reinforcement augmenting earlier inherited personality differences.

Viken and colleagues (1994) analyzed extraversion and neuroticism scores measured on nearly 15,000 male and female Finnish twins, aged between 18 and 53 years, who were tested on two occasions six years apart. They found little evidence of new genetic contributions to individual differences beyond age 30. This was in contrast to significant new environmental effects which emerged at every age. The authors argued that longitudinal instability is best explained in terms of events or innovations which are capable of producing long-lasting changes in personality.

Although most of the reviewed reports are limited to cross-sectional analyses, they are consistent with a longitudinally stable genetic contribution underpinning variation in adult personality. If longitudinal data are available, the degree to which variation in a complex behaviors is caused by stable and enduring effects, versus those which are transient, can be more precisely modeled using a simplex design (Boomsma et al., 1989; Eaves et al., 1986; Neale & Cardon, 1992). The chief advantage of this model fitting approach is the partitioning of environmental and genetic variation at each time-point into genetic and environmental effects unique to each occasion versus the contribution of genetic and environmental effects transmitted from previous time-points (Eaves et al., 1986).

Aim

As previously mentioned, there appears to be high genetic continuity in adult personality, at least with respect to neuroticism and extraversion (Eaves et al., 1989). With the exception of Viken and colleagues (1994), most of the research on genetic continuity in personality has been based on data sets which were either longitudinal but genetically uninformative (Conley, 1984; Watson & Clark, 1984; Ormel & Rijsdijk, 2000), or genetically informative but cross-sectional (Eaves et al.,1989). Moreover, these studies have focused on adult samples and much less is known about the genetic stability in measures of adolescent personality. Therefore, the aim of this paper is to fit simplex models to longitudinal measures of adolescent neuroticism, psychoticism, extraversion and lie. This will determine whether there are quantitative or qualitative changes in the magnitude of genetic effects over time.

Methods

Subjects

Data were collected in three waves as part of ongoing studies into the development of melanocytic naevi (moles) at the ages of 12 and 14 years, and of cognition at age 16. The clinical protocols of these studies have been described in detail elsewhere (Evans et al., 2001; McGregor et al., 1999; Wright & Martin, in press; Zhu et al., 1999). Twins were enlisted by contacting the principals of primary schools in the greater Brisbane area, media appeals and word of mouth. Informed consent was obtained from all participants and parents prior to testing. Twins were tested as closely as possible to their 12th, 14th and 16th birthdays.

Measures and Imputation

At each interview at ages 12, 14 and 16 years, twins, co-twins and their siblings were asked to complete the full 81-item Junior Eysenck Personality Questionnaire (JEPQ; Eaves et al., 1989; Eysenck, 1972; Eysenck & Eysenck, 1975), which assesses the three major dimensions of personality: psychoticism (P; 17 items), extraversion (E; 24 items) and neuroticism (N; 20 items). In addition, the questionnaire contained the 20-item Lie (L) Scale which is a measure of social desirability. The JEPQ was scored on a three-point scale (*yes, don't know* and *no*) with *don't know* responses recorded as missing. The imputation option in PRELIS 2.20 (Jöreskog & Sörbom, 1992) was used to impute missing values using sex and the full number items within each personality dimension. The same procedure was repeated at each wave. This approach substitutes values for the missing values from other cases with similar response patterns and no missing values in the matching variables from other cases, provided that the variance in the values from the other cases is acceptable (see Jöreskog & Sorbom, 1992). In order to avoid the possibility of artifactual inflation of twin correlations, imputation was carried out on an individual basis ignoring the paired structure of the data. Raw cumulative scores were then calculated for each personality dimension across age and sex. Summary statistics appear in Table 1.

Zygosity

Zygosity in the same-sex twin pairs was diagnosed by typing nine highly polymorphic DNA microsatellite markers (AmpFLSTRR Profiler PlusT, Applied Biosystems, Foster City, CA) and three blood groups (ABO, MNS, Rh). The probability of dizygosity given concordance for all markers in our panel, was $< 10^{-4}$. In

Table 1

Age Norms: Number of Complete Responses (*N*), Means (μ), Standard Deviations (δ) and Cronbach Alphas (α) for the Dimensions of Psychoticism, Extraversion, Neuroticism, and Lie, Based on Twins Aged 12, 14 and 16 Years

	Psychoticism 17 items				Extraversion 24 items				Neuroticism 20 items				Lie 20 items			
Age	*N*	μ	δ	α	*N*	μ	δ	α	*N*	μ	δ	α	*N*	μ	δ	α
Males																
12 yrs	673	3.59	7.73	.70	672	19.11	11.81	.71	675	10.10	21.27	.73	674	8.37	15.84	.72
14 yrs	574	4.28	8.25	.74	574	19.26	13.25	.72	575	9.08	23.65	.74	576	6.38	12.88	.71
16 yrs	525	3.93	5.96	.73	522	19.53	13.14	.72	524	8.71	21.12	.73	525	6.28	11.22	.70
Females																
12 yrs	669	1.94	4.00	.68	668	18.23	15.69	.72	672	10.91	23.00	.73	674	9.62	15.98	.72
14 yrs	579	2.26	4.47	.74	579	18.89	15.24	.72	582	10.56	23.63	.74	582	7.78	14.78	.72
16 yrs	570	2.27	3.78	.73	565	19.35	17.21	.73	570	11.20	20.05	.73	570	7.29	12.69	.71

50 twin pairs where DNA was not available, zygosity was judged by similarity of appearance. The number of complete twin pairs and singletons by zygosity is shown in Table 2. For psychoticism, neuroticism, extraversion and lie there were 326, 331, 326 and 326 twin pairs respectively who had complete data on all three occasions.

Genetic and Longitudinal Analyses

General, common and scalar sex-limitation univariate genetic models were fitted to the data by the method of Maximum Likelihood in Mx using continuous data methods (Neale, 1999). Multivariate models were then fitted to the data. These make use of the additional information in the cross-correlations between relatives for different traits or same traits measured at different times. Although the Cholesky decomposition allows us to determine the extent to which genetic and environmental influences are shared in common by a trait measured at different time points, it is limited in so far as it does not take full advantage of the time-series nature of the data (i.e., that causation is unidirectional through time; Boomsma et al., 1989). Our solution was to fit simplex models which are described in detail elsewhere (Evans et al., 2001; Evans et al., 2002; Gillespie et al., 2004).

For each simplex design in this study, the covariance part of the model consisted of 16 parameters (i.e., three innovations [ζ] and two transmission coefficients [β] for each source of variance [A, C and E]). The models also included measurement error (ε) parameters which influence the phenotypes at each age but which are not transmitted to subsequent ages. These were constrained equal at all three ages. Because the simplex models were fitted to continuous data, three means (constrained equal within twin pairs) were specified for each personality dimension measured at ages 12, 14 and 16 years. In order for the model to be identified, the factor loadings on the observed variables from the latent factors were set to one, and the variance of the innovation terms estimated.

Results

In order to correct for skewness, square-root transformations were required for the dimensions of psychoticism and extraversion. Maximum Likelihood correlations between personality scores measured at 12, 14 and 16 years appear in Table 3. These correlations range from moderate to large. Male retest correlations range from .43 to .64 and the female correlations range from .36 to .67. The highest were between scores measured closer in age.

Mean and Variance Homogeneity

For each dimension of personality there were no significant differences in means and variances either within twin pairs or across zygosity. We also found no significant sex differences in the variances. There were, however, significant sex differences in the means for psychoticism at ages 12, 14 and 16 years (males higher), extraversion at age 12 (males higher), neuroticism at ages 14 and 16 (females higher), and lie at ages 12, 14 and 16 years (females higher).

Twin-Pair Correlations

Maximum Likelihood twin-pair polychoric correlations and 95% confidence intervals for the JEPQ

Table 2

The Number of Twin Pairs with Complete Personality Data by Zygosity at 12, 14 and 16 Years

	12 yrs	14 yrs	16 yrs
MZFF	129	112	133
MZMM	124	104	116
DZFF	109	93	75
DZMM	116	99	69
DZFM	192	170	152
Total	670	578	545

Note: Numbers are approximate and averaged across P, E, N and L scales and do not include incomplete twin pairs.

MZFF/MM = monozygotic female/male twin pairs; DZFF/MM = dizygotic female/male twin pairs; DZFM = dizygotic opposite sex twin pairs.

Table 3

Test–Retest Correlations for the JEPQ Dimensions

	Males (N = 531–687)											
	Psychoticism			Extraversion			Neuroticism			Lie		
	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs
12 yrs		.51	.45		.57	.49		.57	.44		.54	.44
14 yrs	.45		.53	.56		.63	.53		.63	.52		.60
16 yrs	.37	.47		.47	.66		.45	.59			.44	.64
	Females (N = 571–688)											

Note: Female correlations appear below the diagonal.

dimensions appear in Table 4. For each dimension, a model which constrained the MZ and DZ twin-pair correlations to zero was significantly worse than a model in which all twin-pair correlations were allowed to vary, indicating significant familial aggregation for each JEPQ dimension. With the exception of neuroticism at 16 years, a model which then constrained the MZ to equal the DZ twin-pair correlations (same and opposite sex) also deteriorated significantly for each dimension. This suggests that additive genetic effects were the likely sources of familial aggregation. Finally, a model which constrained equal the same and opposite sex dizygotic twin pair correlations did not provide a good fit to the lie data at 12 and 14 years of age, which suggests possible sex limitation.

Univariate Analysis

The best-fitting univariate models are summarized in Table 5.

Psychoticism

A common-effects sex limitation model provided the best fit to the psychoticism data at ages 12 and 14 years. At age 14 years, the best fitting univariate model also included a shared environmental component that explained 11% of the variance. At age 16 years, the male and female parameters could be constrained equal, and additive effects accounted for 41% of the variance.

Extraversion

For extraversion, the male and female genetic and environmental parameters at each age could be constrained equal without any significant deterioration in model fit. Additive genetic effects explained between 41% and 47% of the total variance.

Neuroticism

For neuroticism at age 12, an AE common-effects sex-limitation model provided the best fit to the data.

Table 4

Maximum Likelihood Twin Pair Polychoric Correlations and their 95% Confidence Intervals (CI) for Psychoticism, Extraversion, Neuroticism and Lie

	Psychoticism						Extraversion					
	12 yrs		14 yrs		16 yrs		12 yrs		14 yrs		16 yrs	
	r	95% C.I.	r	95% C.I.	r	95% C.I.	r	95% C.I.	r	95% C.I.	r	95% C.I.
MZFF	.47	0.33–0.59	.55	0.40–0.67	.42	0.28–0.54	.39	0.23–0.52	.45	0.29–0.59	.46	0.31–0.58
MZMM	.51	0.37–0.61	.64	0.51–0.74	.46	0.31–0.57	.41	0.25–0.54	.52	0.36–0.65	.43	0.27–0.57
DZFF	.07	–0.13–0.27	.32	0.12–0.49	.13	–0.15–0.37	.33	0.15–0.48	.26	0.06–0.44	.28	0.06–0.48
DZMM	.16	–0.03–0.34	.37	0.18–0.53	.29	0.06–0.47	.29	0.11–0.44	.09	–0.10–0.29	.16	–0.07–0.38
DZFM	.11	–0.02–0.24	–.07	–0.22–0.08	.02	–0.13–0.18	.12	–0.02–0.25	.23	0.09–0.36	.14	–0.02–0.29
	Neuroticism						Lie					
	12 yrs		14 yrs		16 yrs		12 yrs		14 yrs		16 yrs	
	r	95% C.I.	r	95% C.I.	r	95% C.I.	r	95% C.I.	r	95% C.I.	r	95% C.I.
MZFF	.36	0.20–0.50	.50	0.34–0.62	.25	0.09–0.41	.53	0.40–0.64	.54	0.39–0.66	.58	0.46–0.68
MZMM	.52	0.37–0.63	.56	0.41–0.68	.48	0.33–0.61	.44	0.29–0.57	.44	0.27–0.58	.43	0.27–0.57
DZFF	.12	–0.07–0.30	.19	–0.02–0.37	.27	0.05–0.47	.43	0.26–0.57	.36	0.18–0.53	.30	0.08–0.49
DZMM	.27	0.09–0.43	.29	0.10–0.46	.41	0.20–0.59	.35	0.17–0.50	.38	0.20–0.54	.36	0.14–0.55
DZFM	.06	–0.08–0.20	.14	–0.01–0.28	.10	–0.07–0.25	.15	0.01–0.29	.09	–0.06–0.24	.23	0.07–0.37

Note: MZFF/MM = monozygotic female/male twin pairs; DZFF/MM = dizygotic female/male twin pairs; DZFM = dizygotic opposite sex twin pairs.

Twin pair correlations based on separate male and female means and variances.

Table 5

Summary of Best-Fitting Univariate Models with Standardized Variance Components

	Af	*Cf*	*Ef*	*Am*	*Cm*	*Em*
Psychoticism						
2 yrs	.41		.59	.47		.53
14 yrs	.35	.11	.54	.41	.11	.48
16 yrs	.41		.59	.41		.59
Extraversion						
12 yrs	.41		.59	.41		.59
14 yrs	.47		.53	.47		.53
16 yrs	.44		.56	.44		.56
Neuroticism						
12 yrs	.28		.72	.53		.47
14 yrs	.48		.51	.48		.51
16 yrs	.36		.64	.36		.64
16 yrs		.30	.70		.30	.70
Lie						
12 yrs	.22	.32	.46	.18	.23	.60
14 yrs	.34	.17	.49	.10	.31	.59
16 yrs	.50		.50	.50		.50

Note: *Am Cm Em* & *Af Cf Ef* = male and female model parameters.

Estimates of additive genetic variance were almost double for males at 53% versus 28%. At ages 14 and 16 years, the model could be constrained equal across sex. At age 16, however, there was insufficient power to choose between an AE or CE model.

Lie

A common-effects sex-limitation model provided the best fit to the lie data at 12 and 14 years. For males and females alike, familial aggregation was best explained by a combination of additive genetic and shared environmental effects. Estimates of familial aggregation, which were best explained by a combination of additive genetic and shared environmental effects, were slightly higher among females and ranged from 51% to 54% of the variance. The male estimate was 41% at both ages. At age 16, the genetic and environmental components of variance could be constrained equal across sex, and additive genetic effects explained 50% of the variance.

Multivariate Analysis

Because of the strong directional evidence of non-scalar sex limitation for a number of the dimensions at each age, male and female data were analyzed separately. Although not modeled, the analyses also included twins from opposite-sex dizygotic twin pairs in order to improve the estimates of means and variances. Model fitting results are shown in Tables 6, 7, 8 and 9. Multivariate analyses began by fitting Cholesky decompositions to the time-series data using continuous data methods. For all dimensions except for lie, an AE Cholesky provided the most parsimonious fit to the data as judged by the least significant change in log-likelihood.

Female multivariate heritability estimates ranged from 38% to 47% for psychoticism, 40% to 45% for extraversion, 27% to 44% for neuroticism and from 52% to 53% for lie. For males, the estimates were 44% to 55% for psychoticism, 40% to 51% for extraversion, 43% to 55% for neuroticism and from 41% to 47% for lie.

Additive genetic and nonshared environmental latent factor correlations were then calculated and these appear in Table 10. For each dimension, the correlations between the latent additive genetic factors underpinning variation at each time-point were very high and this finding was consistent across sex. The nonshared environmental factor correlations were all small to moderate and decreased with increasing time intervals, suggesting that most of the nonshared environmental variance was time-specific. There were

Table 6

Multivariate Model Fitting Results for Psychoticism

	Psychoticism									
	Females					Males				
	−2LL	*df*	*Δ2LL*	*Δdf*	*p*	*−2LL*	*df*	*Δ2LL*	*Δdf*	*p*
Cholesky										
ACE	2732.23	1797				3015.98	1751			
AE	2732.23	1803	0.00	6	1.00	3017.35	1757	1.38	6	.97
CE	2744.83	1803	12.60	6	.05	3025.52	1757	9.55	6	.15
E	2808.05	1809	75.82	12	***	3113.27	1763	97.29	12	***
Simplex										
ACE	2732.63	1799	0.40	2	.82	3016.25	1753	0.27	2	.87
AE	**2732.79**	**1804**	**0.16**	**5**	**1.00**	3017.76	1758	1.51	5	.91
Drop ζ_{a3}	2767.29	1805	34.50	1	***	**3017.94**	**1759**	**0.18**	**1**	**.67**
Drop ζ_{a2}	2763.48	1805	30.68	1	***	3032.88	1759	15.12	1	***
Drop ζ_{a2-3}	2812.47	1806	79.68	2	***	3034.34	1760	16.58	2	***
CE	2744.97	1804	12.34	5	*	3026.21	1758	9.96	5	.08
E	2968.12	1809	235.49	10	***	3113.27	1763	97.02	10	***

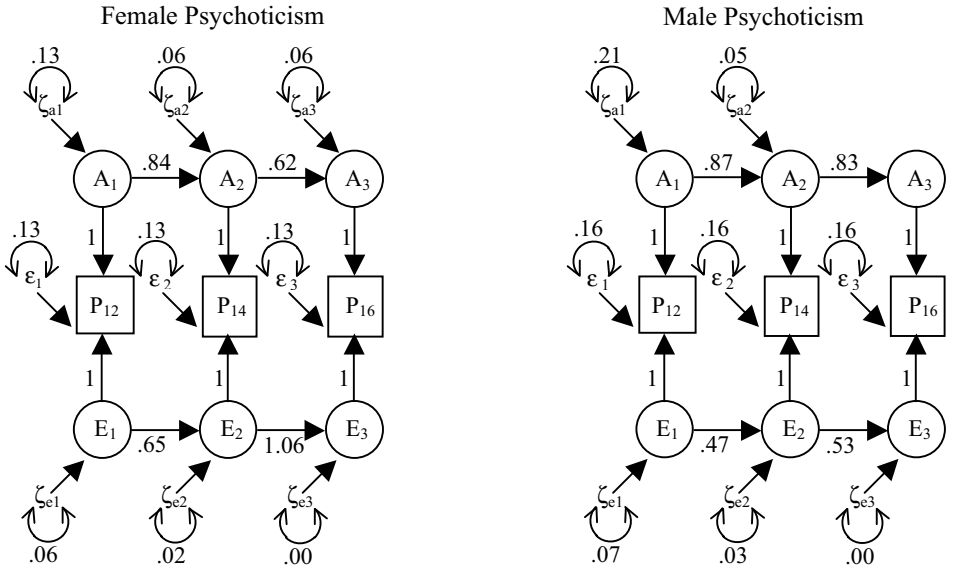

Figure 1

Best fitting genetic simplex model for female and male psychoticism.

$P_{12–16}$ = psychoticism 12–16 yrs

$A_{1–3}$, $E_{1–3}$ = additive genetic and nonshared environmental effects

$\zeta_{a1–3}$, $\zeta_{e1–3}$ = additive genetic innovations and nonshared environmental innovations

$\varepsilon_{1–3}$ = error parameters 12–16 yrs

double/single headed arrows = variance components/path coefficients

some large nonshared environmental correlations (e.g., lie or neuroticism at 14 and 16 years) which suggests that some aspects of the unique environment are stable across time.

Genetic simplex models were then fitted to the data, and model fitting results are also shown in Tables 6, 7, 8 and 9. When compared to the ACE Cholesky, the change in log-likelihood with 2 degrees of freedom associated with the ACE simplex did not deteriorate significantly for any of the personality dimensions. Further analyses revealed that an AE simplex provided the best fit to the data in each case. In other words, there was no evidence of shared environmental effects. Exceptions were for female extraversion, as well as male and female lie, for which there was insufficient power to choose between either an AE or CE simplex model. Since the primary aim was to investigate qualitative and quantitative changes in genetic effects over

Table 7

Multivariate Model Fitting Results for Extraversion

	Extraversion									
	Females					Males				
	–2LL	*df*	*Δ2LL*	*Δdf*	*p*	*–2LL*	*df*	*Δ2LL*	*Δdf*	*p*
Cholesky						Cholesky				
ACE	4066.24	1791				ACE	3743.40	1747		
AE	4068.66	1797	2.43	6	.88	AE	3744.49	1753	1.09	6	.98
CE	4073.17	1797	6.93	6	.33	CE	3760.61	1753	17.21	6	**
E	4141.57	1803	75.33	12	***	E	3812.39	1759	68.99	12	***
Simplex						Simplex					
ACE	4066.59	1793	0.35	2	.84	ACE	3743.40	1749	0.00	2	1.00
Drop ζ_{a3}	4066.59	1794	0.00	1	1.00	AE	3744.56	1754	1.16	5	.95
Drop ζ_{a2}	4066.89	1794	0.30	1	.58	Drop ζ_{a3}	**3744.56**	**1755**	**0.00**	**1**	**1.00**
Drop $\zeta_{a2–3}$	**4066.89**	**1795**	**0.30**	**2**	**.86**	Drop ζ_{a2}	3756.73	1755	12.17	1	***
AE	4068.79	1798	2.20	5	.82	Drop $\zeta_{a2–3}$	3756.73	1756	12.18	2	***
CE	4073.21	1798	6.62	5	.25	CE	3760.62	1754	17.22	5	**
E	4141.57	1803	74.98	10	***	E	3812.39	1759	68.99	10	***

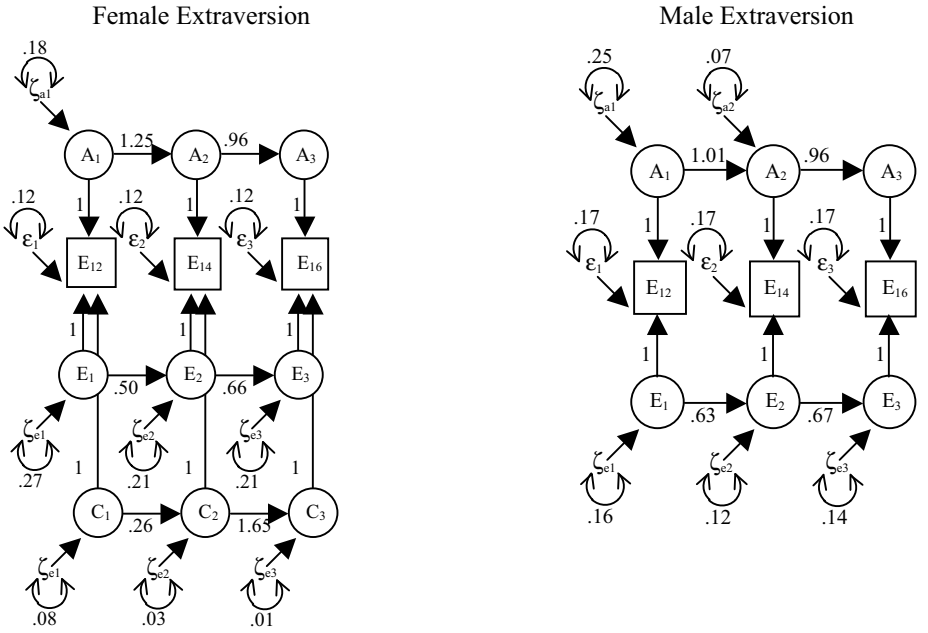

Figure 2

Best fitting genetic simplex model for female and male extraversion.
E_{12-16} = extraversion 12–16 yrs
A_{1-3}, E_{1-3}, C_{1-3} = additive genetic and nonshared and shared environmental effects
ζ_{a1-3}, ζ_{e1-3}, ζ_{c1-3} = additive genetic innovations, nonshared and shared environmental innovations
ε_{1-3} = error parameters 12–16 yrs
double/single headed arrows = variance components/path coefficients

time, the effect of dropping additive genetic innovations at 14 and 16 years was then investigated for each model. The best-fitting genetic simplex models are illustrated in Figures 1, 2, 3 and 4.

Psychoticism

For psychoticism, the largest genetic innovation was at age 12 for males and females alike. For males, the genetic innovation at 16 years could be dropped from the final model. The nonshared environmental innovations were all small relative to the size of the error parameters.

Extraversion

For male and female extraversion, the best-fitting models included large genetic innovations at age 12 years which contributed to variation at ages 14 and 16. The female genetic innovations at 14 and 16 years could be dropped from the final model, whereas for males, only the genetic innovation at 16 years could be removed. Unlike psychoticism, the nonshared environmental innovations were larger relative to the error terms for females, whereas the reverse was observed for males.

Neuroticism

For neuroticism, the largest genetic innovations were again at age 12 years for males and females alike. The female genetic innovation at 16 years could be removed from the model, whereas the genetic innovations at 14 and 16 years could not be removed from the male model.

Lie

For males and females alike the genetic innovations at 14 and 16 years could be removed from the final models.

Discussion

Univariate

Consistent with previous research findings for personality, familial aggregation for each dimension was significant and explained approximately 30% to 50% of the total variance at each age. Contrary to previous reports investigating the junior EPQ which were based on smaller sample sizes (Eaves et al., 1989), these results suggest that common-effect sex-limitation models provide a more parsimonious fit for most of the personality dimensions: psychoticism (12 and 14 years); neuroticism (12 years); and lie (12 and 14 years). In other words, it appears that while the same latent genetic effects in males and females contribute to variation in the JEPQ dimensions, their relative contributions differ across sex.

The total sample size required to reject a false ACE model with no sex limitation when a common-effects sex-limitation model was true with 80% power at the

Table 8
Multivariate Model Fitting Results for Neuroticism

	Neuroticism									
	Females					Males				
	–2LL	*df*	*Δ2LL*	*Δdf*	*p*	*–2LL*	*df*	*Δ2LL*	*Δdf*	*p*
Cholesky										
ACE	10424.88	1803				10016.48	1753			
AE	10425.31	1809	0.42	6	1.00	10019.03	1759	2.55	6	.86
CE	10432.45	1809	7.57	6	.27	10029.21	1759	12.73	6	.05
E	10473.31	1815	48.43	12	***	10116.28	1765	99.79	12	***
Simplex										
ACE	10424.95	1805	0.07	2	.96	10016.88	1755	0.40	2	.82
AE	10425.34	1810	0.39	5	1.00	**10021.26**	**1760**	**4.38**	**5**	**.50**
Drop ζ_{a3}	**10426.57**	**1811**	**1.23**	**1**	**.27**	10058.59	1761	37.32	1	***
Drop ζ_{a2}	10468.64	1811	43.30	1	***	10037.87	1761	16.61	1	***
Drop ζ_{a2-3}	10472.41	1812	47.07	2	***	10080.47	1762	59.21	2	***
CE	10432.45	1810	7.50	5	.19	10032.32	1760	15.44	5	**
E	10663.09	1815	238.14	10	***	10650.99	1765	634.11	10	***

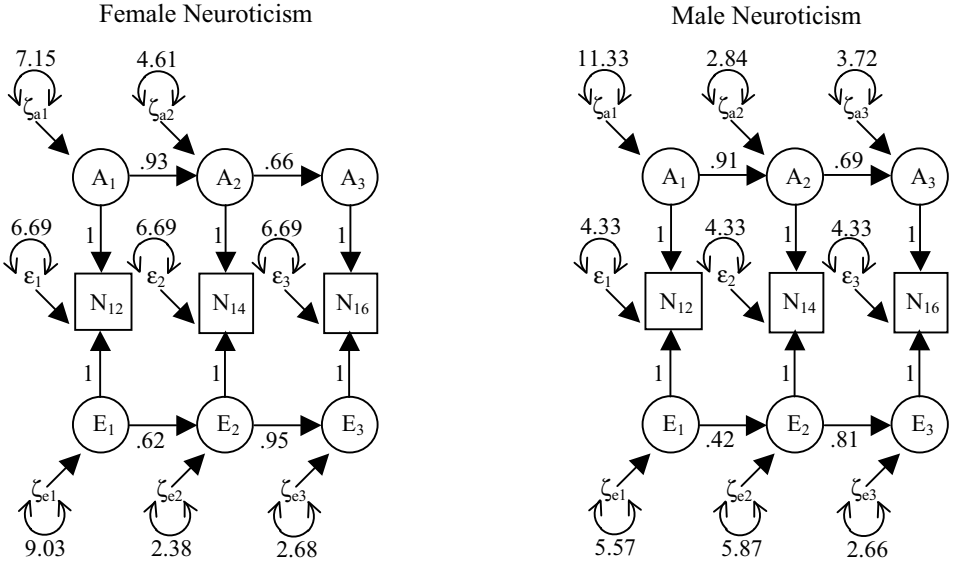

Figure 3
Best fitting genetic simplex model for female and male neuroticism.
$N_{12–16}$ = neuroticism 12–16 yrs
$A_{1–3}$, $E_{1–3}$ = additive genetic and nonshared environmental effects
$\zeta_{a1–3}$, $\zeta_{e1–3}$ = additive genetic innovations and nonshared environmental innovations
$\varepsilon_{1–3}$ = error parameters 12–16 yrs
double/single headed arrows = variance components/path coefficients

.05 significance level, and with 3 degrees of freedom, was 345, 429 and 632 for psychoticism at 12, 14 and 16 years respectively, which is well below the current sample size. Similar sample size estimates were calculated for the remaining personality dimensions confirming that there was sufficient power.

Multivariate

The fit of the ACE simplex models provided a better explanation of the time-series data in so far as the fit was no worse than the corresponding Cholesky decompositions with 2 degrees of freedom to test the change in log-likelihood. Moreover, the AE simplex models fitted the data well for each dimension of adolescent personality. Exceptions were female extraversion as well as male and female lie for which there was insufficient power to choose between either an AE or CE Cholesky. The additive genetic factor correlations based on the Cholesky decompositions revealed that

Table 9

Multivariate Model Fitting Results for Lie

	Lie									
	Females					Males				
	–2LL	*df*	*Δ2LL*	*Δdf*	*p*	*–2LL*	*df*	*Δ2LL*	*Δdf*	*p*
Cholesky										
ACE	9544.67	1805				9214.61	1754			
AE	9553.27	1811	8.60	6	.20	9220.97	1760	6.36	6	.38
CE	9548.40	1811	3.73	6	.71	9216.13	1760	1.52	6	.96
E	9680.95	1817	136.29	12	***	9298.23	1766	83.62	12	***
Simplex										
ACE	9546.27	1807	1.60	2	.45	9215.11	1756	0.50	2	.78
Drop ζ_{a2-3}, ζ_{c2-3}	**9546.35**	**1809**	**0.08**	**2**	**.96**	**9215.11**	**1758**	**0.00**	**2**	**1.00**
AE	9553.87	1812	7.60	5	.18	9222.75	1761	7.64	5	.18
CE	9550.19	1812	3.92	5	.56	9218.03	1761	2.92	5	.71
E	9680.95	1817	134.68	10	***	9298.23	1766	83.12	10	***

the latent factors were all very highly correlated across time points, which is consistent with a pleiotropic model of gene action whereby the same genes explain variation across different time-points.

It is difficult to imagine that genetic variation in personality is completely determined by the age of 12 years. Smaller genetic innovations were observed at 14 and 16 years, most notable for male neuroticism at 14 and 16 years, as well as female neuroticism at 14 years. These smaller genetic innovations potentially hint at age-specific genetic effects related to developmental or hormonal changes during puberty and psychosexual development.

The simplex models fitted to female extraversion and to the male and female lie data provide an interesting insight into the cultural effects on developing

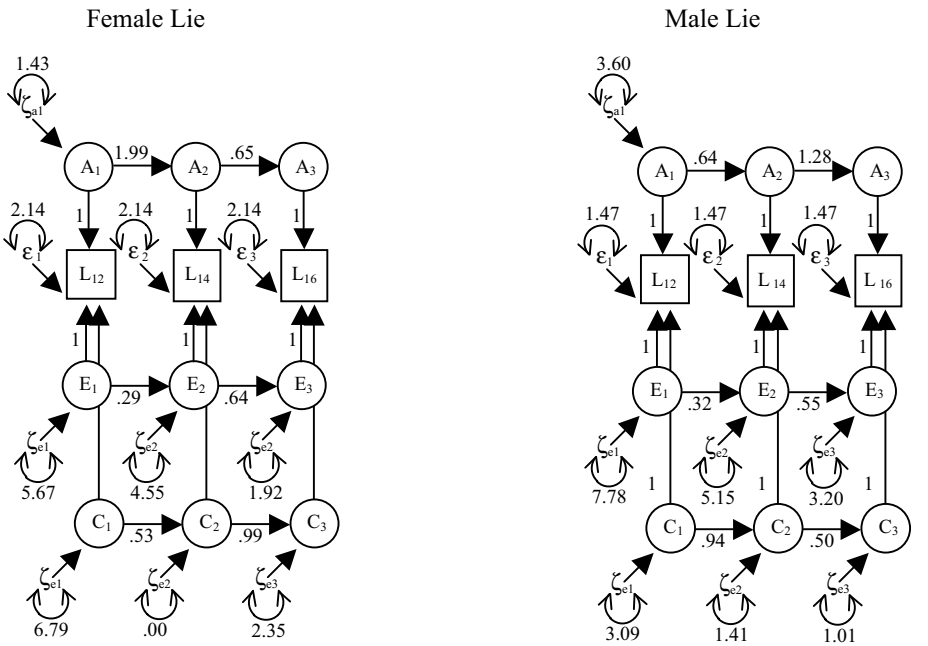

Figure 4

Best fitting genetic simplex model for female and male Lie.

$L_{12–16}$ = lie 12–16 yrs

$A_{1–3}$, $E_{1–3}$, $C_{1–3}$ = additive genetic and nonshared and shared environmental effects

$\zeta_{a1–3}$, $\zeta_{e1–3}$, $\zeta_{c1–3}$ = additive genetic innovations, nonshared and shared environmental innovations

$\varepsilon_{1–3}$ = error parameters 12–16 yrs

double/single headed arrows = variance components/path coefficients

Table 10

Additive Genetic (above diagonal) and Nonshared Environmental (below diagonal in italics) Latent Factor Correlations for the Four Dimensions of Personality

	Females											
	Psychoticism			Extraversion			Neuroticism			Lie		
	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs
12 yrs		.77	.61		.87	.84		.76	.68		.80	.69
14 yrs	*.22*		.64	*.33*		.92	*.40*	.94		*.21*		.76
16 yrs	*.20*	*.33*		*.17*	*.45*		*.36*	*.41*		*.16*	*.49*	
	Males											
	Psychoticism			Extraversion			Neuroticism			Lie		
	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs	12 yrs	14 yrs	16 yrs
12 yrs		.85	.90		.89	.91		.86	.79		.89	.87
14 yrs	*.17*		.94	*.30*		1.00	*.24*		.74	*.26*		.77
16 yrs	*.06*	*.16*		*.18*	*.33*		*.12*	*.53*		*.14*	*.48*	

personalities. Because extraversion is a measure of sociability and lie is in part a measure of social conformity, the shared environmental simplex structures might therefore reflect the impact of schooling or home on social development, the effects of which may disappear once schooling is complete. The female extraversion simplex model is at odds not only with the males, but also with the univariate analyses for which there was clear evidence of no cultural effects. The substantial shared environmental component for lie is consistent with previous analyses of Australian adolescent twins (Macaskill et al., 1994). Although the multivariate modeling did not enable a clear choice between the competing AE and CE model for lie, this trend appears to emerge because the univariate analyses revealed significant cultural effects at ages 12 and 14 years which were no longer evident at 16. Indeed, twin studies based on lie scores from adult populations have consistently found no evidence of shared environmental effects (Eaves et al., 1989; Gillespie et al.,2001). Therefore, the current results suggest that cultural effects causing variation in lie may only be present during early adolescence.

Limitations

Our results need to be interpreted in the context of important limitations. There were several instances where there was insufficient power to choose between AE and CE simplex models (female extraversion, male and female lie). The power to reject a false saturated ACE simplex model based on the female neuroticism data with 80% power, at a significance level of .05 with 2 degrees of freedom, when the ACE Cholesky is true would require a sample size of 56,330 subjects. When based on the female psychoticism data, the sample size required is 32,468 subjects. Similarly large sample sizes would be required to reject with 80% confidence ACE simple models for the remaining dimensions of personality.

Questions of power also highlight the limitation of fitting simplex models restricted to three time-points which could, in part, be overcome with additional measurement waves. When data are limited to three time points, a common genetic factor model will also provide a very good fit when compared to the genetic simplex model. Therefore, although a number of models were fitted, the current approach was by no means exhaustive.

Ormel and Rijsdijk (2000) have argued that simplex models cannot adequately describe the data and suggest that their 'mixed' model represents the best-informed hypothesis. Their model includes a common 'trait' factor (T) and 'state' simplex (S) structures. Longitudinal stability is conceptualized as arising from carry-over or transmission effects and an immutable common-factor effect. They fitted this model to the data based on a random population sample of 296 adult subjects who completed a Dutch version of the neuroticism scale on five occasions over a 16-year period. Their findings were equivocal: both the mixed and simplex models provided comparable fits to the data. A common-factor model fitted the data poorly but by their own estimates, there was insufficient power to detect potential trait effects. Although their results were not based on genetically informative data, as more data points become available, this method represents an ideal extension to the current simplex design. Other possible modeling strategies include biometric growth models (see Neale & McArdle, 2000), but these are beyond the scope of the current analyses, and besides, more than three time-points are required.

Conclusion

Despite the above limitations, the current time-series data based on three time-points provided an opportunity to fit simplex models and test explicit hypotheses of genetic continuity for the dimensions of adolescent

personality. Most of the genetic variation in measures of adolescent personality at 14 and 16 years could be explained by large innovations and genetic variation already present at 12 years. There was, however, evidence for smaller but significant genetic innovations at 14 and 16 years for male and female neuroticism, at 14 years for male extraversion, at 14 and 16 years for female psychoticism, and finally, at 14 years for male psychoticism. The same data are also ideal for fitting univariate and multivariate linkage models to detect QTLs of significant effect. DNA is now available from many of these same twins in the current study including their siblings and parents.

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References

Boomsma, D. I., Martin, N. G., & Molenaar, P. C. (1989). Factor and simplex models for repeated measures: Application to two psychomotor measures of alcohol sensitivity in twins. *Behavior Genetics, 19,* 79–96.

Conley, J. J. (1984). The hierarchy of consistency: A review and model of longitudinal findings on adult individual differences in intelligence, personality and self-opinion. *Personality and Individual Differences, 5,* 11–25.

Eaves, L. J., & Eysenck, H. J. (1976). Genetic and environmental components of inconsistency and unrepeatability in twins' responses to a neuroticism questionnaire. *Behavior Genetics, 6,* 145–160.

Eaves, L., Eysenck, H. J., & Martin, N. G. (1989). *Genes, culture, and personality: An empirical approach*. London: Academic Press.

Eaves, L. J., Heath, A. C., Neale, M. C., Hewitt, J. K., & Martin, N. G. (1998). Sex differences and non-additivity in the effects of genes on personality. *Twin Research, 1,* 131–137.

Eaves, L. J., Long, J., & Heath, A. C. (1986). A theory of developmental change in quantitative phenotypes applied to cognitive development. *Behavior Genetics, 16,* 143–162.

Evans, D. M., Frazer, I. H., Boomsma, D. I., & Martin, N. G. (2001). Developmental genetics of red cell indices during puberty: A longitudinal twin study. *International Journal of Human Genetics, 1,* 41–53.

Evans, D. M., Gillespie, N. A., & Martin, N. G. (2002). Biometrical genetics. *Biological Psychology, 61,* 33–51.

Eysenck, H. J. (1959). *Manual for the Maudsley Personality Inventory*. London: University of London Press.

Eysenck, H. J., & Eysenck, M. W. (1985). *Personality and individual differences: A natural science approach*. New York: Plenum Press.

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual for the Eysenck Personality Questionnaire (adult and junior)*. San Diego, CA: Digits.

Eysenck, M. W., & Eysenck, H. J. (1980). Mischel and the concept of personality. *British Journal of Psychology, 17,* 191–204.

Eysenck, S. B. G. (1972). *Junior Eysenck Personality Inventory*. EdITS/Educational and Industrial Testing Service, PO Box 7234, San Diego, CA 92167.

Eysenck, S. B. G., Eysenck, H. J., & Barrett, P. (1985). A revised version of the Psychoticism scale. *Personality and Individual Differences, 6,* 21–30.

Gillespie, N. A., Kirk, K. M., Evans, D. M., Heath, A. C., Hickie, I. B., & Martin, N. G. (2004). Do the genetic or environmental determinants of anxiety and depression change with age? A longitudinal study of Australian twins. *Twin Research, 7,* 39–53.

Gillespie, N. G., Johnstone, S., Boyce, P., Heath, A. C., & Martin, N. G. (2001). The genetic and environmental relationship between the Interpersonal Sensitivity Measures (IPSM) and the personality dimensions of Eysenck and Cloninger. *Journal of Personality and Individual Differences, 31,* 1039–1051.

Jöreskog, K. G., & Sorbom, D. (1992). *New features in LISREL 8*. Chicago, IL: Scientific Software International.

Kirk, K. M., Birley, A. J., Statham, D. J., Haddon, B., Lake, R. I., Andrews, J. G., & Martin, N. G. (2000). Anxiety and depression in twin and sib pairs extremely discordant and concordant for neuroticism: Prodromus to a linkage study. *Twin Research, 3,* 299–309.

Lake, R. I. E., Eaves, L. J., Maes, H. M., Heath, A. C., & Martin, N. G. (1999). Further evidence against the environmental transmission of individual differences in Neuroticism from a collaborative study of 45850 twins and relatives on two continents. *Behavior Genetics, 30,* 223–233.

Macaskill, G. T., Hopper, J. L., White, V., & Hill, D. J. (1994). Genetic and environmental variation in Eysenck Personality Questionnaire scales measured on Australian adolescent twins. *Behavior Genetics, 24,* 481–491.

McCrae, R. R., & Costa, P. T. (1990). *Personality in adulthood*. NewYork: Guilford.

McGregor, B., Pfitzner, J., Zhu, G., Grace, M., Eldridge, A., Pearson, J., Mayne, C., Aitken, J. F., Green, A. C., & Martin, N. G. (1999). Genetic and environmental contributions to size, color, shape, and other characteristics of melanocytic naevi in a sample of adolescent twins. *Genetic Epidemiology, 16,* 40–53.

Neale, M. C. (1999). *Mx: Statistical modeling* (5th ed.). Richmond, VA: Department of Psychiatry, Medical College of Virginia.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for Genetic Studies of Twins and Families*. *NATO ASI Series*. Dordrecht, the Netherlands: Kluwer Academic.

Neale, M. C., & McArdle, J. J. (2000). Structured latent growth curves for twin data. *Twin Research, 3,* 165–177.

Ormel, J., & Rijsdijk, F. V. (2000). Continuing change in neuroticism during adulthood structural modelling of a 16-year, 5-wave community study. *Personality and Individual Differences, 28,* 461–478.

Viken, R. J., Rose, R. J., Kaprio, J., & Koskenvuo, M. (1994). A developmental genetic analysis of adult personality: Extraversion and neuroticism from 18 to 59 years of age. *Journal of Personality and Social Psychology, 66,* 722–730.

Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96,* 465–490.

Wright, M., & Martin, N. (in press). The Brisbane Adolescent Twin Study: Outline of study methods and research projects. *Australian Journal of Psychology*.

Zhu, G., Duffy, D. L., Eldridge, A., Grace, M., Mayne, C., O'Gorman, L., Aitken, J. F., Neale, M. C., Hayward, N. K., Green, A. C., & Martin, N. G. (1999). A major quantitative-trait locus for mole density is linked to the familial melanoma gene CDKN2A: A maximum-likelihood combined linkage and association analysis in twins and their sibs. *American Journal of Human Genetics, 65,* 483–492.