Symptoms of Anxiety and Depression in Young Adults: Genetic and Environmental Influences on Stability and Change

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Etiological factors for stability and change in symptoms of anxiety and depression, including sex differences, are largely unexplored in young adults. Using biometric modeling and two-wave longitudinal data from 4393 Norwegian twins aged 18 to 31 we explored (i) heritabilities of symptoms of anxiety and depression, (ii) effects of genetic and environmental factors on the stability and change of such symptoms, and (iii) sex-specific effects. The phenotypic cross-time correlations for symptoms of anxiety and depression were estimated to .54 and .49 for males and females, respectively. The best fitting longitudinal model specified additive genetic and individual environmental influences and emerging effects from the shared environment for females only. For both males and females, long-term stability was mainly attributable to stable additive genetic factors, whereas change was essentially related to environmental influences. Minor time-specific genetic effects were indicated, and some stable variance was due to the individual environment. Additive genetic risk factors explained 87% and 68% of the phenotypic cross-time correlation for males and females, with the unique environment accounting for the remaining covariance. The results provide strong evidence for the temporal stability of genetic risk factors for symptoms of anxiety and depression in young adults, and substantial sex-specific influences on heritability, stability and change.

Young adulthood is hypothesized to be a particularly vulnerable period for the development of psychological problems (Burke et al., 1990) and the prevalence of mental disorders has been characterized as alarmingly high (Newman et al., 1996). Major cross-sectional community surveys have reported a 1-year prevalence of mental disorders in young adults ranging from 25% (Robins & Regier, 1991) to 37% (Kessler et al., 1994), depending on diagnostic criteria and age ranges used in the studies (Narrow et al., 2002). Comparable estimates have been shown in epidemiological surveys of the Nordic populations (Alto-Setälä et al., 2001; Kringlen et al., 2001), indicating that the prevalence rates of mental disorders in young adults range somewhere between 25% and 40% in industrialized countries. Young adulthood covers a highly transitional life period and insufficient resolution of the many adaptive challenges faced during this life stage may have serious implications and long-term consequences for the individual's subsequent achievements. During young adulthood, most people leave their childhood home, complete their education, establish a career and many take on family responsibilities and nurse small children. Emergence of mental disorders during this phase may therefore have more critical and lasting consequences than at other stages in life, and research indicates a considerable risk potential for accumulation of complicating factors, recurrences and future chronicity in young adults with mental health problems (Wittchen et al., 1998).

The most frequently diagnosed disorders in young adults are usually mood or anxiety disorders (Alto-Setälä et al., 2001), and subthreshold symptoms of anxiety and depression, rate among the most prevalent complaints seen in psychiatric and general medical practice overall (Kendler et al., 1986). Along with age, gender is a powerful predictor of such symptoms. A higher female preponderance in prevalence, incidence and morbidity risk for symptoms of anxiety and depression is one of the most widely documented findings in epidemiological research worldwide. Differences usually emerge some time during midpuberty, and by late adolescence, the female:male ratio usually approaches 2:1 which is the rate commonly seen in adults (Hankin et al., 1998).

Co-occurrence of anxiety and depression, both as symptoms and disorders, is another common finding

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and suggests a shared vulnerability. Several large scale epidemiological studies have indicated that comorbidity between the most common psychiatric disorders may be accounted for by two broad underlying factors reflecting externalizing (antisocial, substance abuse/dependence) and internalizing (anxious, depressive) disorders (Krueger, 1999). The internalizing factor has been shown to be divisible into two subdimensions, one referring to a combination of pervasive anxiety and sadness (anxious-misery), including major depression (MD), dysthymia, and generalized anxiety disorder (GAD), and one reflecting phobic avoidance (fear), including animal and situational phobia (Kendler et al., 2003; Krueger, 1999). The stability of this latent 3-factor structure has been found to be substantial (Vollebergh et al., 2001).

Previous research on etiological explanations of development, stability and change in risk factors for symptoms of anxiety and depression during young adulthood, and for the precipitous rise in gender differences is limited and advanced research on causal factors is needed.

Etiology

Mental health problems result from a complex interplay of multiple biological, social and psychological vulnerability factors conferring probabilistic risks. Influences from childhood events, concurrent psychosocial adversity, and genetic vulnerability are commonly assumed to be important causal factors throughout the lifespan, and all may have age-dependent and sex-specific effects (e.g., Kendler et al., 2002).

Substantial genetic risk factors have been found for virtually all psychiatric disorders that have been studied, including liability to symptoms of anxiety and depression, with heritability estimates commonly ranging between .3 and .5 (Kendler et al., 1986; Tambs et al., 1995), although varying somewhat according to age and sex (Scourfield et al., 2003), measurement instrument, and informants included in the study (Happonen et al., 2002). The remaining variance is usually explained by fluctuating nonshared environmental variance, and measurement error (e.g., Jardine et al., 1984).

Twin studies of both children (e.g., Rice et al., 2002) and adults (Kendler et al., 2003) have indicated a substantial shared genetic predisposition for the most common psychopathological conditions, and separate, although highly correlated genetic factors have been suggested for the internalizing subdimensions of anxious–misery and fear (Kendler et al., 2003). Accordingly, it is hypothesized that genetic factors predispose individuals to broad clusters of psychiatric disorders and subthreshold conditions, with distinct environmental stressors leading to the various forms of psychopathology to manifest (Kendler & Karkowski-Shuman, 1997). The personality trait of neuroticism (N) has been found to be a strong predictor of comorbidity among the internalizing disorders (Bienvenu et al., 2001; Hettema et al., 2006), and recent evidence from biometric studies suggests a considerable overlap between genetic factors for N and genetic factors associated with internalizing disorders (Hettema et al., 2006) such as MD (Fanous et al., 2002) and GAD (Hettema et al., 2004), as well as between N and subthreshold symptoms of anxiety and depression (Jardine et al., 1984).

Etiology of Stability and Change

Knowledge of factors causing psychological problems is important, and while both short-term environmental adversities (e.g., stressful life events) and long-term diatheses (e.g., genetic risk, psychosocial factors) are major causal factors in the development of mental ill-health, relatively little is known about how these factors act over time.

Genetic factors are often implicitly assumed to represent static influences throughout the lifespan, contributing to stability over time, whereas change is assumed to reflect factors residing in the environment. Genetic and environmental factors may, however, be involved in both stability and change. Some environmental events might represent proximal causal factors, largely generating time-specific effects. Others may have long-term effects due to lasting influences from past events, or due to being persistently or consistently experienced. Similarly, genetic influences do not necessarily imply immutability or stability. New genetic variance may emerge at any point in life, with different genes coming in to play at different developmental stages throughout the lifespan. Previous research on etiological factors for stability and change in symptoms of anxiety and depression is limited, and only a few studies have reported on the long-term contribution from genetic factors. Analyzing two-wave data from elderly male twins, Carmelli et al. (2000) showed that the stability of symptoms was attributable to continuity of genetic influences. Rijsdijk et al. (2003), studying a heterogeneous sample of female twins (aged 18–79), found the contribution of genetic factors to be considerable (73%), and estimated the cross-time correlations for genetic and environmental influences to be .69 and .22, respectively. None of these studies focused on populations of young adults, and both were restricted to one sex only, leaving sex-specific effects entirely unexplored. Evidence suggests, however, that the etiology of symptoms of anxiety and depression may differ according to both age and sex (Scourfield et al., 2003). Gillespie, Kirk, et al. (2004) studying an age heterogeneous sample of Australian twins (aged 20–96) found genetic innovations at age 20 to essentially explain continuity in anxiety and depression, but smaller age-dependent innovations were observed for female respondents at the ages of 30 (anxiety) and 40 and 70 (depression). Recently, Kendler et al. (2006) demonstrated both qualitative (different genes) and quantitative (different magnitude of effect) sex-specific genetic effects for MD in a large population-based sample of Swedish twins.

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The report was based on cross-sectional data, however, and did not include young adults.

In this study we apply longitudinal genetic analyses to self-reported symptoms of anxiety and depression. Longitudinal twin data offers a unique opportunity to estimate the effects of genetic and environmental risk factors, including the stability and change in such factors, and sex-specific effects. Relatively few studies have focussed exclusively on populations of young adults, and we have no knowledge of previous biometric studies exploring the stability and change in risk factors during this developmental juncture. Results indicative of sex-specific etiological effects also need further replication and confirmation. Using a population-based sample of 4393 young adult Norwegian twins aged 18 to 31, we aim to address these limitations in longitudinal research on symptoms of anxiety and depression in young adults by estimating (i) heritabilities of symptoms of anxiety and depression, (ii) the temporal stability of genetic and environmental risk factors, including (iii) sex-specific effects.

Method

Sample

The Norwegian Institute of Public Health Twin Study is an ongoing longitudinal study with a cohort sequential design. The database consists of population-based registry data that includes data from all like- and unlike-sexed twins born in Norway between 1967 and 1979 (N = 15,370), where both twins survived to age 3 (82%-89%) and their current addresses could be obtained. Twins were identified through information about plural births in the Medical Birth Registry of Norway. Questionnaire data were first collected in 1992 (T_1) from twins born 1967 to 1974 and in 1998 (T_2), a second questionnaire was sent to all twins born 1967 to 1979. Data for the present study come from both data collections (T_1 and T_2). Responses on T_1 were obtained from a total of 2570 complete pairs and 724 singletons, altogether 5864 individuals, representing an individual response rate of 74% and a pairwise response rate of 63%. On T_2, responses were obtained from 8045 twins, including 3334 complete pairs and 1377 singletons. The individual and pair-wise response rates were 63% and 53%, respectively. The combined T_1 and T_2 questionnaire samples include 9478 twins who responded to at least one of the questionnaires. About 80% of the twins participating at T_1 also participated at T_2 and the longitudinal sample consists of 4430 twins who participated at both data collections, including 1725 complete pairs and 980 single responders. The mean ages of the respondents were 21.73 (SD = 2.23) and 25.59 (SD = 3.67) for T_1 and T_2, respectively.

Zygosity

Zygosity information is not included in the Norwegian Medical Birth Registry, and initial zygosity assignment was based upon discriminant function analyses using seven questionnaire items in T_1 and the same seven and two additional items in T_2. Previous research on a different Norwegian twin sample showed these items to categorize correctly more than 97% of the twins (Magnus et al., 1983). Twenty-four micro-satellite markers were then genotyped on a subsample of 676 of the like-sexed pairs in the sample. Results from these markers were used as dependent variables in a discriminant analysis with the above mentioned questionnaire items as independent variables. Seventeen of the 676 pairs with DNA information were found to be misclassified by the questionnaire data and were corrected. Thus, the total number of expected misclassified pairs was estimated as 2.0% of the like-sexed pairs.

Measures

The SCL-25 (Hesbacher et al., 1980) is a widely used self-administered screening instrument for detecting psychological problems in nonpsychiatric settings. It was specifically designed to measure symptoms of anxiety and depression, and includes highly correlated scales for anxiety (10 items) and depression (15 items). The SCL anxiety and depression scales have similar reliability and validity (Koeter, 1992) and figures of sensitivity and specificity (Goldberg et al., 1976) as the General Health Questionnaire (GHQ; Goldberg & Williams, 1988). They were initially designed as 'state' measures, but a range of studies have demonstrated that common psychological symptoms display considerable temporal stability, to a large extent reflect stable or 'trait'-like aspects (e.g., Foley et al., 2001), and strongly predict prospective risk of disorder (Kendler et al., 1994). The SCL-5 used in the present study includes five items (two anxiety and three depression items) empirically selected from the SCL-25 that explain the maximally highest proportion of the variance of the two subscales and the total scores (Tambs & Moum, 1993). Development of the SCL-5 was based on SCL data from a sample of 5999 subjects (2993 men, 3006 women) participating in a health screening by the government Norwegian Health Screening Service in 1990. The alpha reliability for the short form questionnaire was .85 and the sum of the five selected items has been shown to correlate .92 with the global SCL-25 score.

Respondents completing the SCL-5 were asked to indicate if he/she during the last 14 days was bothered or distressed at all, was a little bit, quite much, or very much bothered by: (1) 'Feeling fearful', (2) 'Nervousness or shakiness inside', (3) 'Feeling hopeless about the future', (4) 'Feeling blue', and (5) 'Worrying too much about things'. Responses were scored using a scale 1 to 4, and the average score across items was used as a total index score (1–5). Alpha reliabilities for the present sample, based on imputed item values (see below), were estimated to .82 and .84 for T_1 and T_2 respectively. A multisample confirmatory factor analysis using the software program Mx (Neale et al., 1999) was conducted to further validate the scale and test for cross-sex measurement invariance. Due to the variables being standardized, the invariance of the factor loadings were tested. Minor, but significant sex differences were

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observed at T_1 ($\Delta\chi^2_5$ = 51.00, p = .00, AIC = 41.00). Item loadings ranged from .74 to .90 for females and .78 to .92 for males. No significant sex differences were observed at T_2 ($\Delta\chi^2_5$ = 8.22, p = .14, AIC = –1.78), and item loadings ranged from .72 to .89. Further psychometric information of the instrument is described elsewhere (Tambs & Moum, 1993).

Imputation

Imputation of missing values increased the total effective sample size from 5175 subjects (complete data on all five SCL items) to 5832 subjects at T_1, and from 7829 to 8004 at T_2, whereas the longitudinal material increased from 3870 to 4393 twins. The Expectation Maximization (EM) imputation option in SPSS 12.0.1 was used to impute missing values for each SCL-5 item using the remaining SCL items and seven selected items as matching variables. The EM procedure is a process of regression imputation based on the observed relationship between variables. Missing values are replaced iteratively until successful iterations are sufficiently similar, and yield a complete set of data. The matching variables were highly associated with the SCL score, one example being: 'Over the last month, have you suffered from nervousness (felt irritable, anxious, tense, or restless)?'. Regression analysis using the seven matching variables as predictors for the total SCL score yielded Multiple *R*'s of .73 and of .75 for the T_1 and T_2 data, respectively. To avoid the possibility of artefactual inflation of the twin correlations, imputation was carried out on an individual basis, thus ignoring the paired structure of the twin data. Data from respondents with more than five values missing on the total 12 selected items were excluded from the analyses. The same procedure was repeated for each measurement occasion.

Statistical Analyses

Initial estimates of the importance of genetic and environmental influences were calculated using polychoric cross-twin (i.e., correlations between $Twin_1$ and $Twin_2$ for each time point) and cross-twin cross-time correlations (i.e., correlations between $Twin_1$–T_2 and $Twin_2$–T_1) for the five zygosity groups. Having computed the correlations as a means of inspecting the covariance structure of the data, model-fitting analyses were conducted on raw ordinalized data by means of the maximum likelihood (ML) estimation procedure in Mx. The scales were polytomized into four categories (three thresholds), to include a minimum of 10% in each category. Variances were constrained to unity as they cannot be calculated when thresholds are estimated. The raw data approach provides better estimates and correct model fit statistics for nonnormally distributed data. The procedure also allows for preliminary testing of the basic assumptions concerning the homogeneity of response (thresholds) distributions within pairs, across sex, and zygosity. Ascertainment bias can also be tested by comparing the thresholds in complete and incomplete pairs (Neale & Eaves, 1993).

The longitudinal analyses used here extend the basic univariate model by decomposing the variance of SCL scores at each measurement occasion *and* the covariance between occasions, into genetic and environmental sources. This enables investigation of whether the same latent genetic and environmental factors contribute to the observed variance in the observed trait over time. Thus, heritability (a^2), and the environmental variance components, c^2, and e^2, can be estimated for each measurement occasion, and by standardizing the A, C, and E matrices, the genetic, common environmental and individual environmental cross-time correlations can be estimated. Furthermore, genetic and environmental contributions to the phenotypic cross-time correlation may be calculated. These multivariate models are more statistically powerful than univariate models and therefore provide more precise estimates of the latent effects on the phenotypes (Schmitz et al., 1998).

Tests for various sex-specific effects, which can be either qualitative or quantitative, were also conducted. Quantitative sex effects refer to sex differences in the magnitudes of the genetic and environmental variance components, whereas qualitative effects refer to sex differences in the sets of genes (or common environments)

Table 1

SCL Index Scores

	N	Frequency distribution, % (*N*)				*M*	*SD*	*p*
		I	II	III	IV			
T1								
Males	2751	42.2 (1162)	20.0 (550)	23.9 (658)	13.8 (381)	1.32	0.47	< .01
Females	3081	29.0 (893)	19.4 (597)	28.8 (886)	22.9 (705)	1.45	0.50	
T2								
Males	3423	48.1 (1648)	19.6 (671)	19.2 (658)	13.0 (446)	1.29	0.44	< .01
Females	4581	35.8 (1638)	17.9 (818)	25.7 (1177)	20.7 (948)	1.41	0.49	

Note: I = Ordinal category 1, score 1.

II = Ordinal category 2, score 1.2.

III = Ordinal category 3, score 1.4, 1.6.

IV = Ordinal category 4, score 1.8 or higher.

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Table 2

Twin Correlations for Symptoms of Anxiety and Depression at Time Points 1 and 2: Within-Twin Cross-Time Correlations, Cross-Twin Correlations, and Cross-Twin Cross-Trait Correlations by Zygosity

	Within-twin cross-time	Cross-twin: Scl-5 T_1	Cross-twin: Scl-5 T_2	Cross-twin cross-time
MZ_m	**.55** (.48–.61)	**.61** (.53-.68)	**.51** (.42–.59)	**.46** (.37–.53)
MZ_f	**.50** (.44–.56)	**.63** (.56–.69)	**.49** (.43–.55)	**.38** (.30–.44)
DZ_m	**.55** (.47–.62)	**.26** (.14–.28)	**.25** (.13–.36)	**.22** (.11–.32)
DZ_f	**.48** (.40–.55)	**.35** (.24–.44)	**.38** (.30–.46)	**.18** (.10–.27)
DZ_u	**.53** (.48–.58)	**.24** (.16–.32)	**.16** (.09–.23)	**.19** (.12–.25)

that influence trait variation. Qualitative sex differences are observed as a parameter estimate, the correlation between additive genetic influences in males and females (r_g) that is significantly less than 1.0. To test for sex differences, the fits are compared between increasingly constrained hierarchical models that parameterize qualitative, then quantitative and then no sex effects (Neale & Maes, 2003).

The data were analyzed using correlated factor models (Neale & Cardon, 1992) in which the variance from each measure is decomposed separately into its genetic and environmental components, and the correlations across components are estimated. The fit of the full model (ACE with quantitative and qualitative sex effects) was compared to several nested submodels. When using ML analysis of raw data, no overall measure of fit is obtained. However, the difference in –2 log likelihood (–2LL) between the models is distributed as χ^2, allowing the relative fit of submodels against the saturated model to be tested using the χ^2 difference test ($\Delta\chi^2_{df}$). A nonsignificant χ^2 value is regarded as compatible with the data, whereas a significant value ($p < .05$) implies a more poorly fitting model and suggests that the model should be rejected. Models with fewer parameters are preferred if they do not provide a substantial significant deterioration in fit. To select the best fitting model, however, we used the Akaike Information Criterion (AIC), which provides a summary index of both parsimony and fit ($\Delta\chi^2$–$2\Delta df$; Akaike, 1987) rather than applying a significant difference in fit as a criterion. The model with the highest negative AIC value was preferred over models having either smaller negative values or positive ones.

Results

Descriptive Statistics and Tests of Threshold Homogeneity

The frequency distribution for each of the ordinal categories is shown in Table 1. In addition, Table 1 shows the means and variances based on full-score distributions prior to recoding. There were significant mean differences in the symptom levels across sexes. Females scored on average, higher than males ($p < .001$) at both measurement occasions. For both males and females, symptom scores were significantly reduced from T_1 to T_2 ($p < .01$). Age was found to be basically uncorrelated with symptom scores, with correlation estimates of .02 and –.04 at T_1 and T_2, respectively.

There were no significant threshold liability differences within pairs, or between monozygotic (MZ) and dizygotic (DZ) twins when comparing like-sexed twins. However, constraining the threshold distributions to be equal for males and females yielded a significant deterioration in fit for both T_1 ($\Delta\chi^2_9 = 5.32$, $p = .81$, AIC = –12.68), and T_2 ($\Delta\chi^2_9 = 11.95$, $p = .22$, AIC = –6.05), indicating sex differences in the distribution of symptom scores (as also indicated by descriptive statistics). Consequently, the thresholds in males and females were allowed to vary across sexes for further analyses. The thresholds in complete and incomplete twin pairs could be constrained to equality without any significant deterioration in fit, indicative of no significant co-operation bias.

Twin Correlations

The phenotypic cross-time correlation was estimated to be .54 (95% confidence interval [CI] .49–.59) and .49 (95% CI .43–.54) for males and females, respectively. Table 2 displays the polychoric within-twin cross-time correlations, the cross-twin correlations and the cross-twin cross-time correlations by zygosity with 95% CI.

Table 3

Model Fitting Results

Sex effect	Model	–2LL	$\Delta\chi^2$	Δdf	p	AIC
I	1. ACE	35,124.43				
	2. $AC_f E$	35,124.28	-0.15	4	—	–8.15
	3. AE	35,134.17	9.74	5	.08	–0.26
	4. CE	35,261.35	136.91	8	.00	120.91
	5. E	35,708.79	584.36	13	.00	558.36
II	6. ACE	35,131.67	7.24	5	.20	–2.76
	7. $AC_f E$	**35,133.91**	**9.48**	**9**	**.40**	**–8.52**
	8. AE	35,152.18	27.74	10	.00	7.74
III	9. ACE	35,153.67	29.24	11	.00	7.24
	10. AE	35,153.67	29.24	14	.01	1.24

Note: Quantitative sex effects refer to sex differences in the magnitudes of the genetic and environmental factors, whereas qualitative sex-specific effects refer to sex differences in the sets of genes influencing trait variation. The best fitting model is shown in bold.

I = Nonscalar (general) sex limitation (qualitative and quantitative sex differences)

II = Scalar (common) sex limitation (quantitative sex differences)

III = No sex limitation (no sex differences)

–2LL = 2 × log likelihood

AIC = Akaike Information Criterion. Best fitting model in bold.

C_f = Effects from C are estimated for females only

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Table 4

Parameter Estimates and Correlations Between Male and Female Additive Genetic Factors (r_g) with 95% Confidence Intervals for the Full and Best Fitting Models

Model	Time 1							Time 2						
	a^2m	c^2m	e^2 m	a^2f	c^2f	e^2f	r_g	a^2m	c^2m	e^2m	a^2f	c^2f	e^2f	r_g
Full	.57	.02	.42	.51	.00	.49	.59	.48	.03	.49	.22	.26	.52	.36
Best fitting*	.55		.45	.50		.50	1.00	.50		.50	.30	.19	.51	1.00

Note: a^2 indicates heritability whereas c^2 and e^2 indicates the shared and nonshared environmental variance components.
* AC_1E with quantitative sex effects. Effects from shared environmental factors are estimated for females at T_2 only.

Consistently greater cross-twin resemblance in MZ than DZ twins, suggested a significant contribution from genetic factors at both time points, and significantly greater cross-twin cross-time correlations in MZ than in DZ twins, indicated genetic contributions to the long-term stability in symptoms of anxiety and depression. Co-twin similarity for DZ unlike-sexed (DZU) and same-sexed pairs was indicative of sex-specific genetic effects.

Bivariate Modeling

The model fitting results are shown in Table 3.

The full model (model 1) against which the nested submodels were compared, was an ACE model allowing for both qualitative and quantitative sex differences (nonscalar, or general sex limitation model). Pathways from A and C were sequentially eliminated to test for significant contribution of additive genetic and shared environmental effects. Based on the correlation analyses that only indicated effects from shared environmental factors for females at T_2, an ACE model (model 2) specifying estimation of the C parameter for females at T_2 only was estimated. This model fitted the data well, suggesting that familiar resemblance for symptoms of anxiety and depression could be explained solely by additive genetic effects at T_1, but not for females at T_2. This model yielded a negative $\Delta\chi^2$ value (–.15). In general, the full model is an optimal model in terms of –2LL. However, due to a necessary constraint (cross-time correlations for C-effects were set equal across sex), which was not specified in this reduced model, the $\Delta\chi^2$ value turned out negative. The CE and E models (models 4 and 5) were both firmly rejected by the $\Delta\chi^2$-test (136.91_6, $p < .001$; 584.36_8, $p < .001$), indicating significant influences from additive genetic effects. An ACE model specifying only quantitative genetic effects ($r_g = 1$; model 7) and specifying estimation of the C parameter for females at T_2 only, fitted best of all models in terms of AIC and p values, whereas the models with no sex differences (models 9 and 10) were firmly rejected by the χ^2-test.

Parameter estimates and correlations between genetic effects in males and females (r_g) for the full and best fitting models are given in Table 4. Constraining of the parameter estimates (model 7) to be equal in males and females, produced a significant deterioration in fit for T_2 ($\Delta\chi^2_1 = 10.59$, $p = .001$), but not for T_1 ($\Delta\chi^{22}_1 = 1.04$, $p = .31$). We then tested if the genetic and environmental parameter estimates could be set equal over time, for males and females separately. This resulted in a significantly reduced fit for females ($\Delta\chi^{22}_1 = 10.98$, $p = .001$) but not for males ($\Delta\chi^2_1 = 0.98$, $p = .32$).

In the best fitting model (Figure 1), the cross-time correlations between the latent additive genetic factors were high, and estimated to .89 (95% CI .79–.99), whereas the individual environment contributed less to stability over time with respective correlations for males and females of .15 (95% CI .03–.27) and .31 (95% CI .22–.40). A significant deterioration in fit was found when constraining the cross-time correlation for individual environmental effects to be equal for males and females ($\Delta\chi^2_1 = 5.08$, $p = .02$).

Analysis of the phenotypic cross-time correlation revealed that additive genetic factors accounted for 87% and 68% of the correlation for males and females, respectively, whereas the individual environment explained the remaining 13% and 32%.

Modeling based on imputed and unimputed data yielded consistent results.

Discussion

The findings from this study indicate that liability to symptoms of anxiety and depression are quite stable, and that the temporal stability of symptoms largely is attributable to stable genetic factors. Genes are thus contributing to preserve individual differences in symptomatology throughout young adulthood, and essentially, the same genetic influences are operating over time. However, some time-specific genetic variance was implicated, such that genetic factors, although primarily representing a source of continuity, also represent a minor source of change.

In contrast, individual environmental influences accounted for approximately half of the phenotypic variance at each assessment, but the effects were mostly transitory. This indicates that the substantial effects from immediate life circumstances generally do not produce long-lasting effects or enduring changes on liability to symptoms of anxiety and depression, and suggests that the environmental effects are not generally cumulative. Nevertheless, some individual-specific experiences do seem to exert a sustaining influence, and these long-lasting effects from past or

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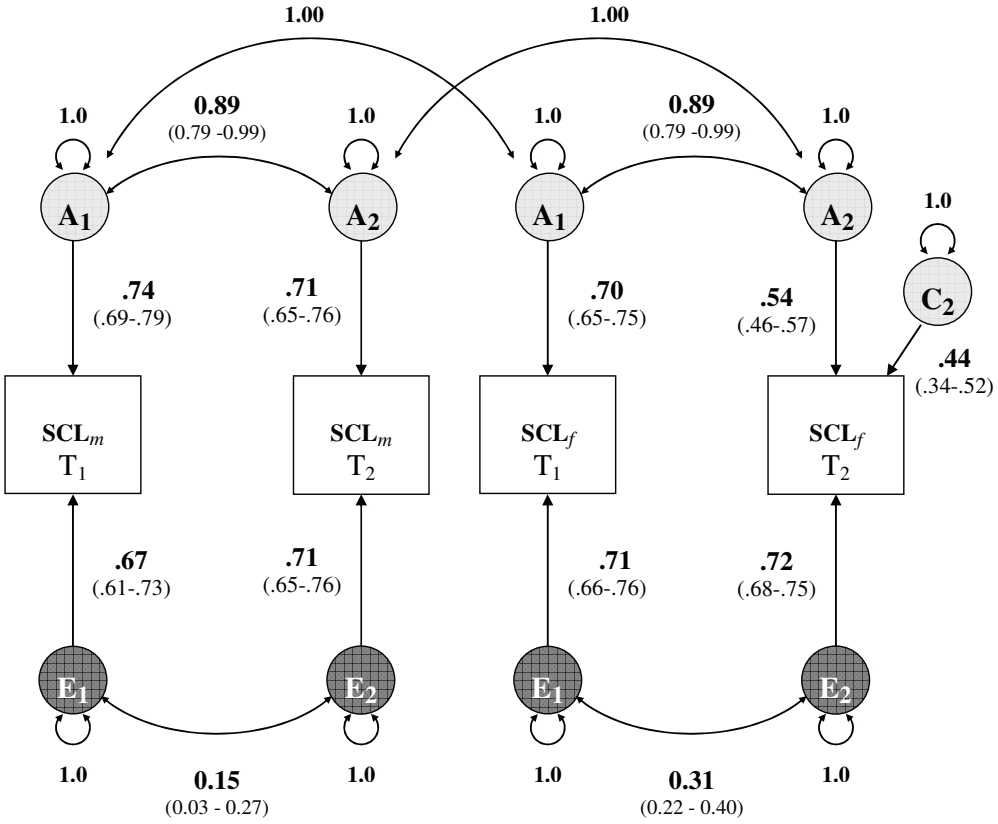

Figure 1

The magnitude of each path is shown in the figure, and should be squared to equal the proportion of variance in the observed variable accounted for by the latent factors.

persistent events were found to be significantly more common in females than in males.

Stable effects from shared environmental factors were altogether negligible. This is in agreement with previous studies (e.g., Rijsdijk et al., 2003), suggesting that the shared environment generally do not contribute to similarity between co-twins over time.

Our findings do not support theories assuming enduring effects of early experiences on mental health with limited opportunities for change in adult life. Rather, our results are consonant with most previous behavioral genetic research on symptoms of depression (Rijsdijk et al., 2003), anxiety and depression (Gillespie, Kirk, et al., 2004), and closely associated personality traits such as neuroticism and negative emotionality (e.g., Gillespie, Evans et al., 2004; McGue et al., 1993). These collective results suggest a stable liability to symptoms, primarily explained by genetic factors and considerable, but mainly transient influences from environmental factors and events (Loehlin, 1992).

The stability of individual differences and the continuity of etiological factors for symptoms of anxiety and depression also during young adulthood are of particular note when keeping in mind the biological, psychological, and social changes occurring during this developmental period, and the fact that young adulthood involves more life-changing roles, demographic diversity, instability, and identity decisions than any other life stage (Arnett, 2000). A minor, but interesting paradox concerns age and symptom scores not being correlated, although symptom scores dropped significantly over time. The drop was very small, however, and despite attrition analyses not having shown substantial recruitment bias on mental health in our sample (Harris et al., in preparation), the drop could be due to some minor biased attrition or a change in the extreme scorers over time (Kirk et al., 2000).

Heritability and Correlation Estimates

Our heritability estimates are consistent with previous estimates of joint anxiety and depression measures, or measures of psychological distress (Rijsdijk et al., 2003; Tambs et al., 1995), but on the high side compared to a majority of reported estimates for MD (e.g., Kendler et al., 2006) and symptoms of depression (e.g., Agrawal et al., 2004). In a two-wave study of adult female twins (aged 18–79, mean age 47.7), Rijsdijk et al. (2003) reported heritabilities of 44% and 51% for the total GHQ score. The contribution from genetic factors to the phenotypic cross-time correlation was estimated to be between 65% and 73%.

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The corresponding estimate in our study was 68%, whereas the male estimate was 87%. Similar estimates have also been shown for subjective well-being, which is closely and negatively correlated with symptoms of depression (Nes et al., 2006). Also, Gillespie, Kirk, et al. (2004) found genetic innovations at age 20 to essentially explain continuity in anxiety and depression throughout life. Although heritability estimates are sample statistics, estimates for depressive symptoms and MD obtained from methodologically rigorous twin studies have been shown to be highly similar across studies, indicative of no major differences in European or European-derived populations (Kendler et al., 2006). Minor inconsistencies between our results and previous studies may partly be attributable to our sample being decidedly younger and more homogenous in age than comparable studies (e.g., Gillespie, Kirk, et al., 2004; Kendler et al., 2006), partly due to our use of a joint measure of symptoms of anxiety and depression. A correlation of .75 between the subscales for anxiety and depression (SCL-25, Hesbacher et al., 1980), and a pervasive comorbidity rate for anxiety and depression generally observed, suggest however, that 'pure' cases may be unrepresentative.

Sex-Specific Effects

Descriptive analyses showed that the female respondents reported significantly more symptoms than their male counterparts, and biometric modeling indicated several notable sex differences in etiology. First, the nonshared environment accounted for a larger proportion of the phenotypic cross-time correlation in females than in males. Second, the environmental risk factors were significantly more stable for females, and thirdly, there were considerable sex differences in the magnitude of genetic and environmental influences at T_2. Lastly, modeling results suggested both qualitative and quantitative sex-specific genetic effects.

Environmental effects explained significantly more of the temporal stability on liability to symptoms of anxiety and depression in females than in males, and nonshared environmental factors were to a larger extent stable, suggesting a special female susceptibility to long-term effects from past events. Previous cross-sectional studies suggest the likelihood of different susceptibility by sex from certain life events and point to gender-based differential responses to psychological distress, with women being more vulnerable than men to the effects of undesirable events in their proximal social networks and interpersonal relationships (e.g., Maciejewski et al., 2001) and men being more influenced by occupationally related or law-/violence-related difficulties (Kendler et al., 2001). On average, women have been found to be more sensitive to the effects of low levels of social support (Kendler et al., 2005), and particularly at risk for crises involving children, housing and reproductive difficulties (Nazroo et al., 1997). Our study adds important information concerning the continuity of environmental effects. Unfortunately, we were not able to explore to what extent these effects represent long-term effects from past events, persistent or consistently occurring factors in the environment, nor to identify the specific experiences accounting for the observed effects. Our results suggest however, a differential stability of environmental effects in young adult males and females.

Significantly higher male than female heritability for liability to symptoms of anxiety and depression was indicated at T_2. Higher male than female heritability of depressive symptoms has previously been reported for adolescents (e.g., Eley & Stevenson, 1999; Rice et al., 2002), whereas higher female heritability for liability to MD has been reported for middle-aged and elderly twins (Kendler et al., 2006). Apparent inconsistencies might be attributable to different assessment procedures (self-ratings, diagnostic interviews), and somewhat differing phenotypes under study (categorical versus continuous measures), and substantial age differences between the samples investigated. The study by Kendler et al. (2006) included twins aged 42 and older, and the etiology of liability to psychological distress symptoms in older age groups are likely to differ from that in young adults. Evidence from epidemiological, clinical and longitudinal studies indicates that depression arising after age 50 may, at least partly, have unique etiological factors (e.g., Krishnan, 2002).

A considerable decrease in female heritability at T_2 was associated with complementary increases in shared environmental influences. Shared environmental factors include both lifelong and currently shared experiences. Results did not indicate significant effects from lifelong shared experiences on symptoms of anxiety and depression, but suggest that currently shared experiences are influencing symptomatology for females at the second assessment. Support for the etiological significance of shared environmental factors on anxiety and depression has been scarce, and our finding of an emerging effect in females only, is somewhat at odds with the majority of studies which converge on finding that environmental influences are substantial, but almost exclusively of the nonshared variety (Kendler et al., 1992). However, Kendler et al. (2002) found that twin resemblance in social phobias was almost entirely due to family environment in females, and in males to be essentially attributable to genetic factors. Tambs & Moum (1993) have also reported sex-specific sibling environment in a large sample of Norwegian nuclear families and twins, and the mere existence of such an association suggests at least a small effect of common environmental factors.

Less than one unit of AIC constituted the basis for selecting the best fitting model (model 7) to the AC_fE model allowing for both quantitative and qualitative sex differences (model 2). Thus, we

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cannot dismiss the existence of qualitative sex-specific genetic effects. Correlations between the male and female genetic factors (r_g) were estimated to be .63 (95% CI .33-.93) and .80 (95% CI .39–1.00) for time points 1 and 2 (model 7), and these estimates are very similar to genetic cross-sex correlations for MD which have previously been estimated to be .63 in the largest published twin study to date (Kendler et al., 2006). Failure to provide evidence for qualitative sex-specific genetic effects in many studies may be related to limited statistical power due to insufficient sample size. Very large sample sizes are needed to reliably detect sex-specific familial transmission for ordinalized and dichotomous data scores (Neale et al., 1994).

Limitations

These results should be interpreted in the context of several methodological limitations.

Cronbach's α for the SCL-5 index was estimated to be .82 and .84 for the T_1 and T_2 data. In twin studies, the individual environmental effect also subsumes measurement error, and estimates of familial resemblance are therefore proportionally deflated by decreasing reliability. However, estimated contributions to the co-variation between measurements, from genetic and environmental factors, are not affected unless measurement errors at the two assessments are correlated.

The use of continuous short-form scales with sound psychometric properties, like SCL-5, may provide greater statistical power than examination of categorical diagnoses, and can be regarded as complementary to analyzing formal diagnoses. Despite the obvious benefits of economy, reliability and statistical power, we examined only current symptoms (past 14 days) rather than lifetime prevalence, and the construct explored is not entirely the same as the diagnostic categories of, for example, MD and GAD. Very few people seem to experience time-limited symptoms with a majority fluctuating across threshold and sub-threshold levels over time such that classification of stability by means of only categorical diagnoses may fail to capture the majority of cases which display persistent symptoms across the life span (Merikangas et al., 2005).

Questionnaire data may to some degree reflect the reporting behavior of symptoms rather than the actual occurrence of symptoms, and genetic effects on reporting behavior have been suggested (Kendler & Karkowski-Shuman, 1997). A large population-based follow-up survey of differential symptom reporting found no evidence indicating that the presentation of depressive symptoms differ by gender (Bogner & Gallo, 2004), and there is little support for sex differences in the recall of particular symptoms (Kendler et al., 2001).

Longitudinal studies are subjected to ascertainment bias and inevitable dropout, and twin studies are particularly vulnerable as co-operation is required from pairs. The individual response rates (74% and 63%) were lower than optimal, but tests of the homogeneity of thresholds did not reveal significant differences between complete and incomplete twin pairs, thus indicating that volunteering behavior was not correlated with the target variables.

Estimates of stability and change based on two-wave studies may have limited reliability as observations over alternative time periods might yield different results, and only linear change trajectories can be measured. Also, we used longitudinal correlated factor models, and our results reflect the limitations specified in these particular models.

Finally, the results were obtained from a sample of young adult Norwegians, and may not extrapolate to other age cohorts or ethnic groups.

Conclusion

Our study extends the previous research literature on symptoms of anxiety and depression by directly quantifying the contribution from genetic and environmental risk factors to the stability and change in symptomatology in young adults, and by estimating sex-specific effects. Based on the current and previous findings, the liability to symptoms of anxiety and depression in childhood, adolescence, and adult life is primarily characterised by stable genetic influences and transitory environmental effects. The results also add important information on sex-specific differences in the magnitude of etiological effects, and their stability over time.

Our approach offers a broad understanding of the etiological processes underlying mean-level stability of symptoms of anxiety and depression in young adults, but raises a need for more detailed investigation of individual change processes and of the relevance of specific life experiences for stability in symptomatology. Developmental pathways might be reliant on interplay among several different influences. More refined understanding of the heterogeneity and specificity of individual responses to environmental factors, and of the specific etiological mechanisms for stability and change in young adult males and females, is important for understanding the development and continuity of psychopathology, and for efforts aiming to design intervention strategies and treatment programs.

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