The Genetic and Environmental Effects on Depressive Symptoms Among Older Female Twins

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The aim of the present study was to examine the contribution of genetic and environmental factors to depressive symptoms among older women. The participants were 102 monozygotic and 115 dizygotic female twin pairs aged 64 to 76 years. Depressive symptoms were assessed by the Center for the Epidemiologic Studies Depression Scale. The contribution of genetic and environmental effects was estimated for the constructed depressiveness factor and for the subscales which were depressed mood, psychomotor retardation, lack of wellbeing and interpersonal difficulties. Of the variance in depressiveness, shared environmental influences accounted for 39% and nonshared environmental influences 61%. For the subscales, 24% to 62% of the variance was explained by individual, and 13% to 23% by shared, environmental factors. Lack of wellbeing had its own moderate additive genetic effect explaining 30% of the variance. This study showed that in older women predominantly environmental factors underlay individual differences in depressiveness; however, the factors varied to some extent between dimensions measured by the subscales.

Depressive symptoms are relatively common in older populations (Haynie et al., 2001), especially among women (Piccinelli & Wilkinson, 2000; Sonnenberg et al., 2000; Takkinen et al., 2004). Compared to major depression based on clinical assessment, depressive symptoms often assessed by self-ratings represent the milder end of the continuum of depression (Kendler & Gardner, 1998). Studying the origin of individual differences in depressive symptoms is important, because the subclinical symptoms have received less attention, even though their prevalence is considerably higher than the prevalence of major depression. Studies comparing depressive symptoms and major depression suggest common genetic and environmental vulnerability factors and neurobiological substrates (Foley et al., 2001; van der Berg et al., 2001, 1999). Depressive symptom scores that lie even below a clinical cut-off can predict various psychiatric diagnoses (Zonderman et al., 1993). Depressive symptoms can decrease subjective wellbeing, increase the risk of suicidal ideation and attempts, and illnesses, and increase the use of hospital and outpatient medical services (see for a review Blazer, 2003). Given the multiple negative correlates of depressiveness, an interesting question which arises is the etiology of depressive symptoms, in particular as depressive symptoms may not be a homogeneous phenomenon but a representation of a multivariate factor with varied genetic and environmental sources underlying its dimensions.

A number of self-rating scales have been developed to assess depressive symptoms (McDowell & Newell, 1996). These scales usually include subscales to tap the different dimensions of depressiveness. In the present study, we use the Center for the Epidemiologic Studies Depression Scale (CES-D). The CES-D was designed to measure depressive symptoms in population surveys (Radloff, 1977). Though the scale is not designed for clinical diagnosis, it is based on symptoms of depression as seen in clinical cases. The scale is commonly used in studies among older populations. The emphasis in the scale is on the affective component of depressed mood. The CES-D has usually captured four factors which are depressed affect, positive affect, psychomotor retardation and interpersonal difficulties. According to Radloff

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(1977), the positive items form a separate dimension in the scale and are not merely the inverse of the negative items. Some studies have indicated that among older people lack of wellbeing rather than negative symptoms is a more salient feature of depression (Gatz et al., 1992). Moreover, in old age the lack of wellbeing factor and negative symptoms are suggested to have separate sets of predictors (Haynie et al., 2001).

Twin and family studies on depressive symptoms have generally shown rather low genetic impact and considerable environmental impact explaining individual differences in adulthood and old age (Clifford et al., 1984; Gatz et al. 1992; Jansson et al., 2004; Jardine et al., 1984; Kendler et al. 1994b; MacKinnon, et al., 1990; McGue & Christensen, 1997). Only a few studies have focused on the sources of individual variation in the subscales of depressive symptoms. Gatz et al. (1992) used CES-D total scale and subscales among 29- to 87-year-old twins. In the CES-D total scale, 16% of the variance was explained by genetic effects, 27% was accounted for by shared environmental effects and 55% by effects that were unique for an individual. Age had a very small effect in the models. For the subscales, environmental effects explained most of the variation except for psychomotor retardation, which had a slight genetic effect, the genetic effect being more evident among older twins.

Jang et al. (2004) recently reported the heritability in the subscales of CES-D and other depression scales among young adults. The results indicated genetic effect (between 18% and 35%) on the subscales that reflect physiologic functions, such as loss of appetite, loss of pleasure, and cognitions such as feelings of guilt, hopelessness and positive affect. Other symptoms that were associated with major depression, such as negative affect, nausea, headaches and tearfulness, were not heritable. In another study among young and middle-aged twins (Silberg et al.,1990), additive genetic effects explained 29% of the variance in depressive symptoms, shared environmental effects accounted for 13% and unique environmental effects the remaining 58% of variability in the total CES-D score. For the subscale scores of depressed mood and psychosomatic retardation only environmental effects were observed, while the subscales for interpersonal difficulties and lack of wellbeing had genetic effects accounting for 55% and 33% of variability, respectively. These findings indicate that there may be differences in the magnitude of genetic and environmental effects on different dimensions of depressive symptoms.

The aim of the present study was to examine the contribution of genetic and environmental factors to depressive symptoms among older women. We especially wanted to study in more detail the architecture of genetic and environmental effects underlying the different dimensions of depressiveness. We used structural equation modeling to analyze the common and specific genetic and environmental effects in the factor structure. This procedure allows for investigation of the proportions of genetic and environmental effects in the subscales of depressive symptoms within the factor structure of the whole scale and comparison of the alternative models.

Method

Sample

The study is part of the Finnish Twin Study on Aging (FITSA), a research program on the genetic and environmental effects on the disablement process in older women. The sample was drawn from a larger twin study, the Finnish Twin Cohort Study with 13,888 adult twin pairs at the baseline in 1975 (Kaprio & Koskenvuo, 2002; Kaprio et al., 1978). The zygosity of the twins was determined at the baseline by a validated questionnaire (Sarna et al., 1978). The twins were classified as monozygotic (MZ), dizygotic (DZ) or of uncertain zygosity (XZ). The method classified 92.7% of the pairs as MZ and DZ with 1.7% probability of misclassification.

The Finnish Twin Cohort Study included 1260 respondent female twin pairs born during the period 1924 to 1937 and first studied in 1975. Of this group, an invitation to take part in the present study was sent to 178 MZ, 212 DZ and 24 XZ twin pairs selected on the basis of age and zygosity. The inclusion criteria were willingness of both sisters of a twin pair to participate and self-reported ability to walk two kilometers and to travel independently to the laboratory. The reasons for nonparticipation were that one or both sisters were unwilling to participate (50 MZ, 51 DZ and 5 XZ pairs), had poor health (28 MZ, 52 DZ and 5 XZ pairs), or had died (2 MZ, 3 DZ and 1 XZ pairs). A total of 98 MZ, 106 DZ and 13 XZ twin pairs participated in the laboratory examination. The zygosity of XZ twins was determined by a battery of 10 highly polymorphic gene markers using DNA extracted from a venous blood sample. According to the results, 4 XZ pairs were classified as MZ and 9 as DZ.

Measures

Depressive symptoms were assessed by the Center for the Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). The total scale has 20 items. Four positively worded items were reverse-coded and they formed the subscale for lack of wellbeing. The subscales of depressed mood and psychomotor retardation included seven items each. The subscale of interpersonal difficulties had two items. Three participants had one item missing and one participant four items missing in the scale. The missing items were imputed using the mean of the individual's scale. Because of the different number of items in the scales, the mean scores for the total scale and four subscales were calculated. The scale has shown good reliability and validity also among older people (see Beekman et al., 1997; Gatz et al., 1993; Lewinsohn et al., 1997; Scott & Melin, 1998). The internal consistency of the total scale and

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the subscales was adequate in the present study (Cronbach's alpha, α = .88 for total scale, α = .84 for depressed mood, α = .75 for psychomotor retardation, α = .79 for lack of wellbeing, and α = .52 for interpersonal difficulties).

Because co-twins may have considerable social interaction, and this may cause bias in twin correlations and means, frequency of social contacts was studied. Social contact with the co-twin was determined by a single self-reported item: How often do you and your twin sister see or phone each other? (0 = never, 1 = less than once in a half year, 2 = about once a half year, 3 = about once a month, 4 = about once a week, 5 = daily or almost daily). About 90% of the members of a twin pair fully agreed on the frequency of social contact. The disagreeing pairs (10%) differed from each one point, except one pair that had two points difference. Because of the high agreement between the co-twins, a mean of the social contact was calculated within a twin pair. The distribution of the frequency of social contact was very skewed: 44% of the pairs contacted each other daily or almost daily. Therefore, the variable was dichotomized in the further analyses (1 = contacts once a week or less, 2 = contacts daily or almost daily).

Statistical Methods

The distributions of the subscales for depressed mood, psychomotor retardation and interpersonal difficulties were skewed. Logarithmic transformation was carried out for these scales and after this the distributions were adequate for the analysis. Means, intraclass correlations and correlations in the total and subscales of CES-D were calculated. The effects of the social contacts with the co-twin on depressive symptoms were studied by partial correlations and *t* tests.

After these preliminary analyses, a measurement model was constructed using the multisample method of LISREL software (Jöreskog et al., 1999). The mean scores of the subscales were used as observed variables. The use of four means instead of 20 items in the model was more practical given the present sample size. In the model, factor loadings, variances of the factors and residual variances were set equal for both twin sisters across the MZ and DZ groups. This was done to keep the structure of the measurement model similar in the MZ and DZ groups. In addition, covariances between corresponding components of twins were estimated freely in both the MZ and DZ group. The constructed model was fitted to the observed covariance matrix using Maximum Likelihood method. The fit of the model was evaluated using χ^2 test. Root mean square error of approximation (RMSEA; Steiger, 1990), comparative fit index (CFI; Hu & Bentler, 1995), and nonnormed fit index (NNFI; Bentler & Bonnet, 1980) were also used according to the rules suggested by Hu and Bentler (1999; RMSEA < 0.06, CFI > 0.95 and NNFI > 0.95). The means of the CES-D subscales were included in the measurement model to test that there were no mean differences between the MZ and DZ twins. The effect of age was also checked in the measurement model.

To determine the relative contribution of genetic and environmental effects to the estimated variance of the latent factors in the measurement model, structural equation modeling was used. Estimation of genetic effects is based on the comparison of the similarity of MZ pairs, who share all their genes in common by descent, with DZ pairs, who share on average 50% of their segregating genes. Further, MZ and DZ twins are expected to be equally susceptible to environmental influences relevant to depressive symptoms and that mating is random with respect to the trait under study (Posthuma et al., 2003). The total phenotype variance (V) is decomposed to three sources of variances: additive genetic (A), shared environmental (C), and nonshared environmental (E) effects. The ACE model was fitted to the latent factors of the measurement model. A and/or C component were dropped from the model and the nested models were compared by the difference in the χ^2 value (Bentler & Bonnet, 1980) and Akaike's Information Criterion (AIC; Akaike, 1987). A significant chi-square difference indicates that the reduction in the model significantly reduces the fit of the model to the data. In the AIC value comparison, a smaller value indicates better fit. The model comparison aimed to detect the most parsimonious model that fitted the data well and was theoretically interpretable.

To study the genetic and environmental effects on the latent factor and the four subscales simultaneously, the common-factor common pathway model was used (Rijsdijk & Sham, 2002). In this model the covariation between subscales is attributed to a single underlying 'phenotypic' latent variable called depressiveness in this study. The observed variance of the latent variable is decomposed into genetic and environmental components of variance called common A, common C and common E. In addition there are variable-specific genetic and environmental sources of variance called specific A, specific C and specific E. An alternative model, multifactor independent model, was also tested. In this model, the variance of the four subscales is decomposed into common A, common C and common E. The model also includes the variable-specific genetic and environmental sources (specific A, specific C and specific E). The schematic diagrams of these two models are shown in Figure 1.

Results

Descriptive Statistics of Depressive Symptoms

The means, standard deviations and correlations for MZ and DZ twins in depressive symptoms are shown in Table 1. The sum score in the total CES-D scale was 14. About a third of the participants scored 16 or more in the scale. Correlations between the subscales were high and indicated adequate factor loadings for the structural equation modeling. Twin pairs showed significant intraclass correlations in depressive symptoms.

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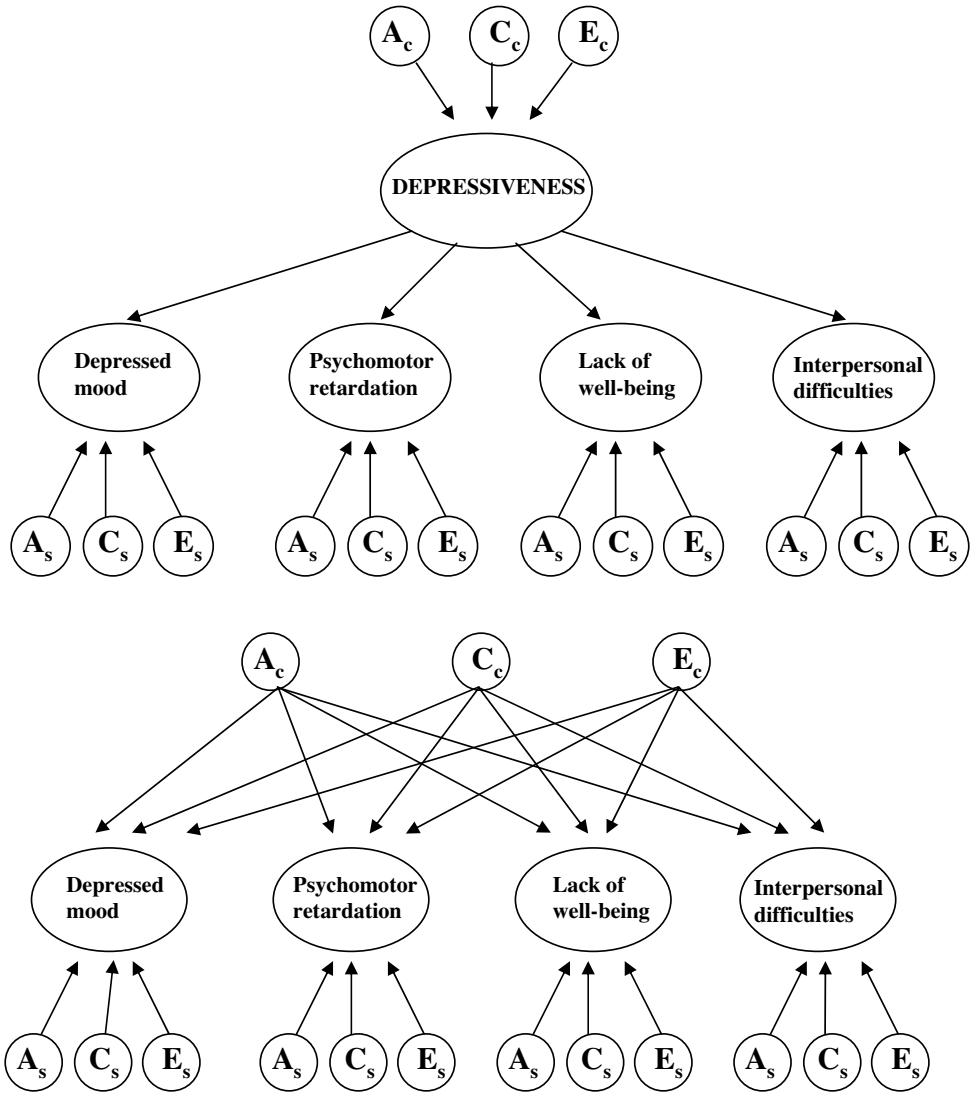

Figure 1

A schematic diagram of common-factor common pathway model (top), and multifactor independent pathway model (bottom).
A = additive genetic effect, C = shared environmental effect, E = nonshared environmental effect, c = common effect, s = specific effect.

MZ correlations were about equal to DZ correlations in the total sum score and the subscales of depressed mood, psychomotor retardation and interpersonal difficulties. The pattern of correlations implied moderate shared environmental effect rather than genetic effects. In the subscale of lack of wellbeing, the MZ correlation was about twice as high as DZ correlation, indicating the presence of additive genetic effect.

The frequency of social contacts between the cotwins had very little effect on the correlations of depressed symptom scores between the sisters: the difference between intraclass depressive symptoms correlations and the partial correlations with the frequency of social contact varied between 0.0001 and 0.009. The frequency of social contacts was not related to the mean level of the CES-D total score or any of the subscales.

Measurement Model

According to the theoretical background of the scale, the measurement model included four subscales constructed of the mean scores of depressed mood, psychomotor retardation, lack of wellbeing and interpersonal difficulties. The factor of depressiveness was constructed of the four subscales. Each factor for Twin A and comparable factor for twin B was allowed to correlate. Multisample method was used in order to include MZ and DZ groups in the same analysis. The model fitted well to the data (Figure 2). The subscales, especially depressed mood and psychomotor retardation, showed substantial loadings to the factor of depressiveness. Because of the prominent influence of the latent factor depressiveness, the within-twin-pair correlations for depressed mood turned out to be nonsignificant, and these subscale-

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Table 1

Correlations, Means and Standard Deviations of the CES-D Total Scale and Subscales Among MZ (*N* of Pairs = 102) and DZ (*N* of Pairs = 115) Female Twins

	CES-D total scale	Depressed mood	Psychomotor retardation	Lack of wellbeing	Interpersonal difficulties	MZ mean (*SD*)	MZ intraclass *r*
CES-D total scale		.86	.81	.74	.42	0.70 (0.27)	.38
Depressed mood	.81		.69	.43	.31	0.37 (0.46)	.31
Psychomotor retardation	.81	.62		.34	.32	0.50 (0.41)	.35
Lack of wellbeing	.72	.34	.35		.23	1.11 (0.75)	.45
Interpersonal difficulties	.47	.41	.37	.14		0.17 (0.33)	.32
DZ mean (*SD*)	0.68 (0.26)	0.32 (0.37)	0.44 (0.38)	1.13 (0.75)	0.20 (0.37)		
DZ intraclass *r*	.34	.25	.32	.22	.34		

Note: All correlations significant at least at $p < .05$.
MZ correlations are given above the diagonal and DZ correlations are below the diagonal. Means and standard deviations are shown for raw, nontransformed scores. In calculating the correlations, log-transformed scores for depressed mood, psychomotor retardation and interpersonal difficulties were used.

specific correlations were set as zero in the further analysis (Table 2).

The assumption that there is no difference in means and variances between MZ and DZ twins was tested in the measurement model. First, the means were set to be equal for MZ and DZ twins, and second, the means were let to be unequal. The same procedure was carried out for variances. The observed nonsignificant χ^2 difference between the models indicated that means and variances did not differ significantly between the groups ($\chi^2[4] = 5.48$, $p = 0.24$ for means, and $\chi^2[5] = 4.78$, $p = 0.44$ for variances). The effect of age was added to the measurement model, but it had very low correlations to the factors (between –.13 and .11), and it was not significantly related to any of the factors in the model.

Genetic and Environmental Variance

The structural equation modeling for common-factor common pathway model started with testing ACE, AE, CE and E models for the factor of depressiveness (Table 3; models 2.1–2.4). Then, the subscales of psychomotor retardation, lack of wellbeing and interpersonal difficulties, which had turned out to have significant own within-twin correlations in the measurement model, were added to the model to test their genetic and environmental components (Table 3; models 3.1–5.4). The model comparison indicated that for the factor depressiveness, and the subscales psychomotor retardation and interpersonal difficulties, CE models fitted best. For lack of wellbeing, AE model fitted best.

Next, the best-fitting submodels were combined in a final model (Figure 3). The final model fitted well to the data. The percentages of common and specific genetic and environmental influence for the CES-D factors are shown in Table 4. The model indicated moderate shared environmental effect on the factor depressiveness and the subscales depressed mood, psychomotor retardation and interpersonal difficulties. For the lack of wellbeing, there was in addition to environmental effects a moderate additive genetic effect. The common contribution provided through the factor depressiveness to the subscale depressed mood was considerable, leaving only some nonshared environmental effect specific to depressed mood. The common contribution provided through the factor depressiveness to psychomotor retardation was also considerably high. In the subscales lack of wellbeing and interpersonal difficulties, the specific effects had a more extensive role than the common effects.

The multifactor independent pathway model was tested as an alternative model. The parameters that were nonsignificant were excluded one by one from the model, starting from the smallest value. The model with all paths significant is presented in Figure 4. Similar to the previous model, this model has common C and common E factors for all four subscales. The contribution is higher to the subscales depressed mood and psychomotor retardation than to lack of wellbeing and interpersonal difficulties. The combinations of the variable-specific A, C and E in each subscale are also similar to the previous model.

Table 2

Within Mono-(MZ) And Dizygotic (DZ) Twin Pair Correlations for Measurement Model Factors Depressiveness, Depressed Mood, Psychomotor Retardation, Lack of Wellbeing and Interpersonal Difficulties

Measurement model factor	MZ twin pairs (*n* = 102)	DZ twin pairs (*n* = 115)
Depressiveness	0.35	0.41
Psychomotor retardation	0.40	0.31
Lack of wellbeing	0.39	0.16
Interpersonal difficulties	0.30	0.27

Note: For the subscale depressed mood, the twin-pair correlations were provided through the factor depressiveness.

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Table 3

The Fit and Comparison of the Nested Models for Factor Depressiveness, and the Subscales Depressed Mood, Psychomotor Retardation, Lack of Wellbeing and Interpersonal Difficulties

Model	$\chi 2$	*df*	RMSEA	AIC	NNFI	CFI	Model comparison	$\Delta\chi 2$ (*df*)
1 Measurement model	62.32	54	0.038	98.32	0.98	0.98		
2 Nested models for the factor depressiveness								
2.1 Depressiveness ACE	111.23	62	0.086	125.23	0.88	0.90		
2.2 Depressiveness AE	116.09	63	0.089	128.09	0.89	0.88	2.2 vs. 2.1	4.86 (1)*
2.3 Depressiveness CE	111.23	63	0.084	123.23	0.89	0.90	2.3 vs. 2.1	0 (1) NS
2.4 Depressiveness E	138.11	64	0.104	148.11	0.83	0.85	2.4 vs. 2.1	26.87 (2)**
3–5 Nested models for the subscales under depressiveness CE								
3.1 Psychomotor retardation ACE	99.78	60	0.079	117.78	0.91	0.92		
3.2 Psychomotor retardation AE	100.17	61	0.077	116.17	0.91	0.92	3.1 vs. 3.2	0.39 (1) NS
3.3 Psychomotor retardation CE	100.08	61	0.077	116.08	0.91	0.92	3.3 vs. 3.1	0.30 (1) NS
3.4 Psychomotor retardation E	264.39	62	0.174	279.39	0.00	0.04	3.4 vs. 3.1	63.61 (2)**
4.1 Lack of wellbeing ACE	91.07	60	0.069	109.07	0.92	0.93		
4.2 Lack of wellbeing AE	91.07	61	0.068	107.07	0.92	0.93	4.2 vs. 4.1	0 (1) NS
4.3 Lack of wellbeing CE	95.20	61	0.072	111.20	0.92	0.92	4.3 vs. 4.1	4.13 (1)*
4.4 Lack of wellbeing E	111.23	62	0.086	125.23	0.88	0.90	4.4 vs. 4.1	20.16 (2)**
5.1 Interpersonal difficulties ACE	94.55	60	0.073	112.55	0.92	0.92		
5.2 Interpersonal difficulties AE	96.43	61	0.074	112.43	0.92	0.92	5.2 vs. 5.1	1.88 (1) NS
5.3 Interpersonal difficulties CE	94.51	61	0.071	110.51	0.92	0.93	5.3 vs. 5.1	0.04 (1) NS
5.4 Interpersonal difficulties E	111.23	62	0.086	125.23	0.88	0.91	5.4 vs. 5.1	16.68 (2)**

Note: NS = nonsignificant, * $p < .05$, ** $p < .001$
For the subscale depressed mood, the environmental influences were provided through the factor depressiveness.

Compared to the common-factor common pathway model, the multifactor independent pathway model had a slightly bigger AIC value indicating worse fit. Therefore, the final model with the best fit is the common-factor common pathway model.

Discussion

The present study showed that about two thirds of the influence on depressive symptoms is due to environmental variation that is unique to each individual. The results are congruent with the previous findings that environmental factors explained most of the variance in depressive symptoms in older people (Gatz et al. 1992; Jansson et al., 2004; McGue & Christensen, 1997). Familial influence also plays a role, though in a smaller scope. For lack of wellbeing, that is, the revised scale for positive mood, the familial influence appeared to be genetic in origin. For depressed mood, psychomotor retardation and interpersonal difficulties, that is, negative mood, in turn, the familial influence was due to the shared environment, for instance parenting style and early social experiences, and peer effects in common or shared life events (see Romanov et al., 2003; Rose et al., 2003). The influence varied in the subscales of depressive symptoms indicating differences in the sources of variance in different aspects of mood.

Our structural equation model confirmed the factor structure of the CES-D. The latent factor depressiveness was underlying the four subscale scores. In the design of the scale the emphasis was on the affective component, depressed mood (Radloff, 1977). Our findings are in line with this as the depressiveness factor explained practically all of the variation in the subscale depressed mood. The variation in the subscales for negative mood was entirely explained by environmental effects, whereas positive mood was explained by genetic and environmental affects that were unique to an individual. Silberg et al. (1990) also found that positive mood and interpersonal difficulties

Table 4

Percentage of Genetic and Environmental Effects in the Final Model

	Common			Specific		
	A	C	E	A	C	E
Depressiveness				–	39	61
Depressed mood	–	27	42	–	–	31
Psychomotor retardation	–	24	38	–	13	24
Lack of wellbeing	–	8	12	30	–	49
Interpersonal difficulties	–	7	11	–	23	59

Note: A = additive genetic; C = shared environmental; E = nonshared environmental effect.

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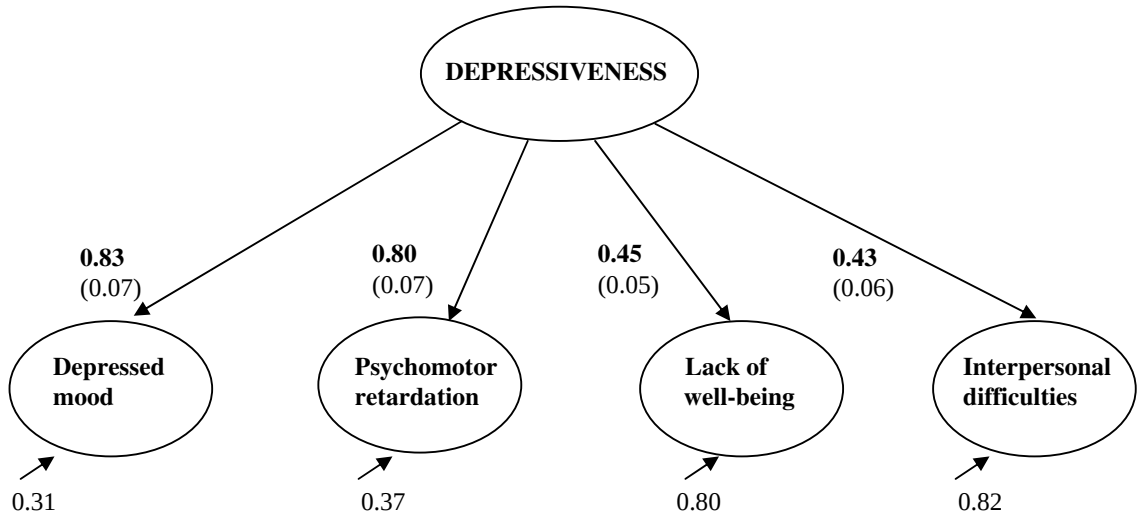
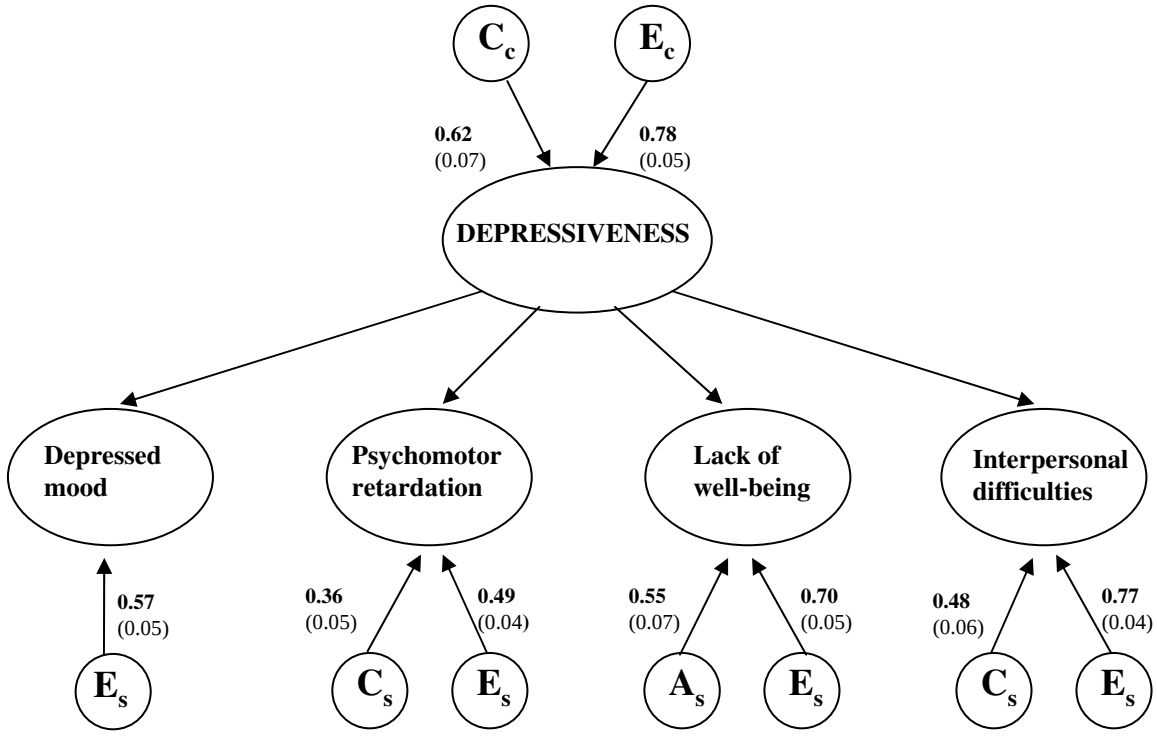


χ^2 (54) = 62.32, p = .204, RMSEA = 0.038, CFI = 0.98, NNFI = 0.98

Figure 2

The completely standardized solution of the measurement model.
Path coefficients (standard errors) for the factor depressiveness and four subscales depressed mood, psychomotor retardation, lack of wellbeing and interpersonal difficulties are shown.



χ^2 (60) = 64.04, p = .337, RMSEA = 0.025, AIC = 88.04, CFI = 0.98, NNFI = 0.98

Figure 3

The path diagram of the common-factor common pathway model with additive genetic (A), shared environmental (C), and nonshared environmental (E) effects explaining the variance of the factor depressiveness and four subscales depressed mood, psychomotor retardation, lack of wellbeing and interpersonal difficulties.
Standardized path coefficients (standard errors) of the model selected as best explaining the present data are presented. c = common effect, s = specific effect.

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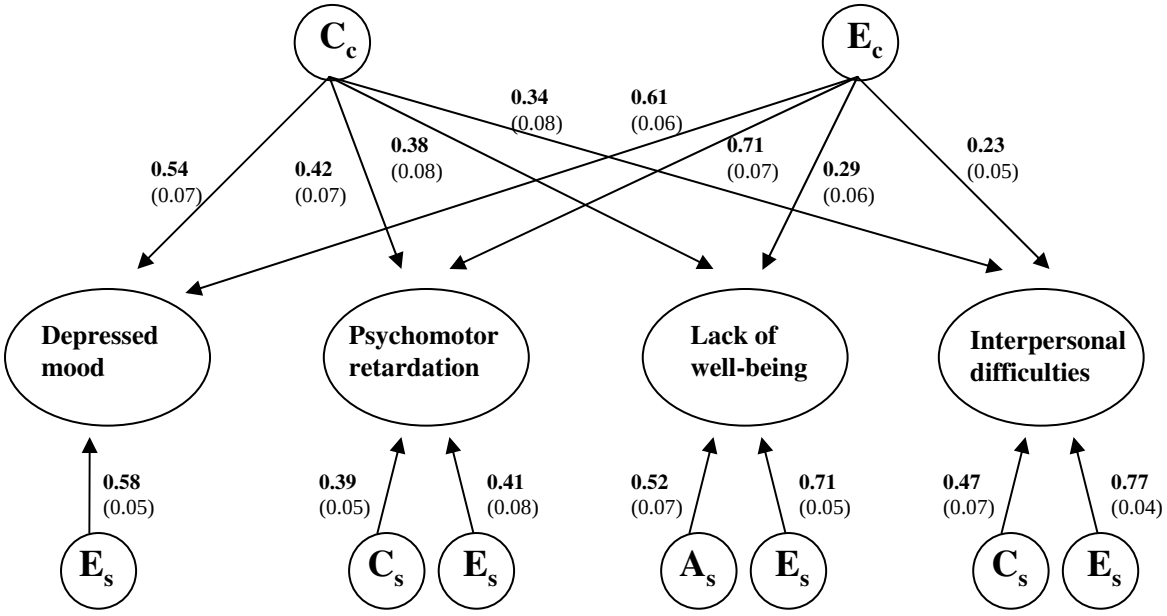

χ^2 (57) = 59.72, p = .300, RMSEA = 0.021, AIC = 89.72, CFI = 0.98, NNFI = 0.99

Figure 4

The path diagram of the multifactor independent pathway model with additive genetic (A), shared environmental (C), and nonshared environmental (E) effects explaining the variance of the factor depressiveness and four subscales depressed mood, psychomotor retardation, lack of wellbeing and interpersonal difficulties.
Standardized path coefficients (standard errors) of the model selected as best explaining the present data are presented. c = common effect, s = specific effect.

had moderate genetic effects, whereas depressed mood and psychomotor retardation were explained by only environmental effects. The results of our study indicate that the positive mood subscale has its own different etiology giving support to the earlier suggestion that its content cannot be interpreted only as an inverse to negative mood (Radloff, 1977; Zich et al., 1990).

The interrelation between the positive and negative mood is interesting. Subjective wellbeing is not a single dimension, but composed of dimensions of positive and negative affect (Diener et al., 1985; Emmons & Diener, 1985). These dimensions have shown to be independent of each other and have different correlates (Baker et al., 1992; Stallings et al, 1997; Watson, 1988). On the other hand, strong negative correlations have also been found, for instance between satisfaction with life and depression, neuroticism and suicide risk (see Hayes & Joseph, 2003; Koivumaa-Honkanen et al., 2000; Lewis et al., 1999). In unraveling the ambiguous relationship between positive and negative, the results of the present study provide some interesting aspects. According to the results, negative and positive affect are correlated and part of the environmental factors underlying them are common for the subscales of lack of wellbeing and the rest of the CES-D scale. However, the genetic source of variance is characteristic only of lack of wellbeing and not of the subscales measuring negative mood in the rest of the scale.

The subscale lack of wellbeing was not only different from the other CES-D subscales in terms of its sources for individual differences, it also had highest mean scores and biggest variance of the four subscales in this sample. The result goes along with the previous findings that the lack of wellbeing is a relatively big contributor to the overall depression (Haynie et al., 2001), and maybe more so among older compared to younger people (Gatz et al., 1992). The high prevalence of lack of wellbeing may also increase the likelihood to find genetic influence.

The present study focused on depressive symptoms in older female twins. The inclusion of only older females means that care should be taken in generalizing the findings to men and other age groups. Studying depressive symptoms only among older women is, however, well-grounded as depressive symptoms are more prevalent among women than men (Piccinelli & Wilkinson, 2000; Sonnenberg et al., 2000; Takkinen et al., 2004). In the present study approximately a third of the sample scored 16 or more in the scale, a validated cut-off for increased risk. High prevalence of depressive symptoms has also been reported earlier among Finnish populations (Heikkinen et al., 1995, 2002; Varjonen, et al., 1998). It is noteworthy that in

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the current study mean scores of negative affect were approximately the same in magnitude as found in previous studies in other countries (Gatz et al., 1992; Haynie et al., 2001). Thus, the higher prevalence of depressive symptoms in Finland than other countries is probably primarily explained by higher scores in the subscale of the lack of wellbeing indicating lower positive mood in our population.

There is a great interest in searching for genetic susceptibility to mood disorders, and to find genetic markers for depression. The previous studies on the sources of variation in depressive symptoms measured by CES-D among older people have usually found only small or no genetic influences (Gatz et al., 1992; Jansson et al., 2004; Silberg et al., 1990). Studies that focused on major depression report genetic influences up to 50% (Johansson et al., 2001; Sullivan et al., 2000). Heterogeneity in the genetic and environmental effects on the different dimensions of major depression has been reported (Kendler et al., 1994a). In the present study, genetic influence was found only in one subscale, lack of wellbeing. The finding suggests that there may be a bigger chance to find genes for happiness and wellbeing than for depressed mood. It is known that subjective wellbeing shares genes with some personality traits (Bergeman et al., 1991; Eid et al., 2003). Moreover, animal models on the traits related to depressive symptoms, such as neuroticism and anxiety, provide evidence of gene–environment interaction (see Bakshi & Kalin, 2000; Holmes, 2001). More research on genetic correlations may help in detecting the genetic background of wellbeing and positive mood.

Are there means to enhance positive mood or reduce the lack of wellbeing? Lack of wellbeing seems to be the biggest reason for scoring high in the depression scale for older women, and besides the large proportion of environmental effects that are unique to each individual, there is a moderate additive genetic effect. Lykken and Tellegen (1996) concluded after finding correlations suggesting a high nonadditive genetic effect (interactions between different genes — epistasis) in subjective wellbeing that trying to be happier may be as futile as trying to be taller. Too much pessimism may, however, be groundless. In several studies, positive affect and subjective wellbeing have been mainly due to additive genetic effects and support for nonadditivity is scarce (Bergeman et al., 1991; Harris et al., 1992; Roysamb et al., 2002). Moreover, the proportion of genetic influence does not exceed half of the total variance. Many environmental factors unique to an individual affect wellbeing in addition to genes. On the other hand, possible gene–environment interaction enables intervention with the effects that are due to genetic variation. Until now, unfortunately, very little has been known of gene–environment interaction in positive affect.

In terms of depressed mood or negative affect, the sources lie partly in shared environment (such as early home environment, school and peers, and shared life events), the behavioral patterns that have been learned there and partly in the experiences unique to an individual. These environmental factors may include learned helplessness, emotional abuse and neglect during childhood, lack of social support and stress related to social relationships, and adverse life events (Blazer, 2003; Piccinelli & Wilkinson, 2000). Psychological or psychosocial approaches as well as other nonpharmacological treatment that take into account environmental factors might be worth trying, especially as medical treatment has been shown to be less successful in treating milder depressive disorders (see Blazer, 2003). It is important that further research take into account the interaction, origin and specific characteristics of positive and negative mood within depressive symptomatology.

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