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Frequency and heritability of depression symptomatology in the second half of life: evidence from Danish twins over 45

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ABSTRACT

Background. Self-reported depressive symptoms among the elderly have generated considerable interest because they are readily available measures of overall well-being in a population often thought to be at special risk for mental disorder.

Method. The heritability of depression symptoms was investigated in a sample of 2169 pairs of Danish twins (1033 MZ and 1136 same sex DZ) ranging in age from 45 to over 95. Twins completed an interview assessment that identified symptoms of depression, which were scored on Affective, Somatic and Total scales.

Results. Overall heritability estimates (a^2) for the Affective ($a^2 = 0{\cdot}27$, (95% CI 0·22–0·32)). Somatic ($a^2 = 0{\cdot}26$, (0·21–0·32)), and Total ($a^2 = 0{\cdot}29$, (0·22–0·34)) scales were all moderate, statistically significant and similar to results from other studies. To assess possible variations in heritability across the wide age span, the sample was stratified into age groups in increments of 10 years. The magnitude of heritable influence did not vary significantly with age or sex. Somatic scale heritability tended to be greater for females than for males, though this difference was not statistically significant. The genetic correlation between the Affective and Somatic scales was 0·71, suggesting substantial common genetic origins.

Conclusions. Though the frequency of self-reported depressive symptoms increased with age in this sample, their heritability did not.

INTRODUCTION

Self-reported depressive symptoms among the elderly have generated considerable interest because they are readily available measures of overall well-being in a population often thought to be at special risk for mental disorder. Rates of clinical levels of such symptoms vary from sample to sample (Roberts & Vernon, 1983; O'Neil *et al.* 1986; Silberg *et al.* 1990), with reported rates ranging from 10 to 20% (Blazer & Williams, 1980; Gurland *et al.* 1983; Lindsay *et al.* 1989).

In spite of these estimates, the relative level of depression in older adults compared to those in other age groups is not known. Blazer (1989) and Newman (1989) both concluded that the incidence of clinically diagnosed depression declined after the age of 60, while the frequency of self-reported symptoms of depression tended to increase. Other studies have yielded similar results (Kessler *et al.* 1992), but most of these studies have not adequately sampled those older than 80. Some have speculated that the dissociation between frequency of clinical depression and endorsement of depression symptoms

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in older adults may simply reflect age-related increases in cognitive and somatic complaints that do not directly affect affective state (Zemore & Eames, 1979). Others have suggested that the depression symptoms in the elderly may be reactions to physical maladies (Berkman *et al.* 1986; Kennedy *et al.* 1990), or even a qualitatively distinct form of depression in which physical complaints are primary (Fogel & Fretwell, 1985; Newman *et al.* 1991). McGue & Christensen (1997) found a high level of genetic correlation (on the order of 0·80) between affective and somatic depressive symptoms in their sample of twins over age 75, suggesting that the two kinds of symptoms are at least linked.

Kendler *et al.* (1994) have suggested that depressive symptoms arise from an underlying hereditary temperamental characteristic in interaction with environmental experiences unique to the individual (as opposed to shared with other family members). Prior studies have reported heritability estimates ranging from 13% to 48%, with slightly higher estimates for women than for men (Kendler *et al.* 1986, 1994; Carmelli *et al.* 2000).

There is some evidence that the genetic contribution to depression increases in old age. Gatz *et al.* (1992) observed an overall heritability of 16% among community-dwelling older Swedish twins, but the heritability was higher in those over the age of 60 than in those younger than that. McGue & Christensen (1997) found a heritability of 34% in a similar sample of Danish twins 75 years of age and older. Carmelli *et al.* (2000) observed the heritability of depression to increase from 25% to 55% over a 10 year period in a sample of male twin military service veterans. These studies have had, however, moderate sized samples and limited age ranges, so leaving open the question of age-moderators of genetic influences on depressive state.

The purpose of this study was to investigate the frequency of depression symptoms and the extent and nature of changes with age in their heritability in a large sample of older twins ranging in age from 45 to over 95, drawn from the same relatively homogeneous population and evaluated in the same manner. Because both the frequency of depression symptoms and estimates of heritability may differ for women and men, we also stratified the sample by sex. Finally, we investigated the genetic relationship between affective and somatic depressive symptoms.

METHOD

Sample

The study sample was drawn from the participants of two population-based surveys spanning the population of Denmark: the Study of Middle-Aged Danish Twins (MADT), and the intake assessment of the Longitudinal Study of Aging Danish Twins (LSADT). These studies have both been described in detail previously (Gaist *et al.* 2000 for MADT; Christensen *et al.* 1999 for LSADT). Briefly, participants in the MADT and LSADT were identified in the Danish Twin Register (Kyvik *et al.* 1996), a nationwide population-based register that is continuously updated. The participants in the MADT were recruited from a random sample of 120 twin pairs from each of the 22 birth cohorts 1931–1952, with a participation rate of 83·1%. The participants in the LSADT were recruited from the pool of twins aged 70 and over in 1995, 1997, and 1999, with participation rates of 77% in 1995, 79% in 1997, and 72% in 1999. The sample resulting for this study consisted of 2169 pairs of Danish twins (1033 monozygotic (MZ) and 1136 same sex dizygotic (DZ)) ranging in age from 45 to over 95, and included the 406 twin pairs over age 75 used by McGue & Christensen (1997). Thus, that study was based on a subset of the sample in the present study. Only twin pairs of known zygosity with complete depression data were included in our analyses. Opposite sex twin pairs were not included because they were not sampled consistently in each age group.

The study thus makes use only of twin pairs. We have no way to assess the implications of this for the portion of the sample drawn from MADT, as only complete pairs were recruited in that survey. For the portion of the sample drawn from LSADT, which included both twin pairs and individual twins whose co-twin did not participate, there were small but significant differences in mean Affective and Total (but not Somatic) scale scores for both males and females, with singles reporting slightly higher (< 1 point) mean scores than members of pairs. The issue is

Table 1. *Items from the adapted form of the CAMDEX Depression Scale*

Affective scale
How often do you feel happy?
Do you feel sad, depressed, or miserable?
Are you happy and satisfied with your life at present?
Do you feel lonely lately?
How do you feel about your future?
Do you sometimes feel that life is not worth living?
Do you feel tense and do you worry more than usual?
Do you consider yourself a nervous person?
Do you feel worthless?
Somatic scale
Do you find it difficult to concentrate?
Do you sometimes feel that you think more slowly?
Do you find it more difficult to make decisions?
Do you find that you have lost energy recently?
Do you find it more difficult to cope with things?
Have you lost pleasure or interest in doing things?
Do you speak more slowly than usual?
Do you have extraordinarily long sleep?

Wording is a paraphrase of the actual interview item.
CAMDEX, Cambridge Mental Disorders of the Elderly Examination.

noteworthy because the most common reason for lack of co-twin participation in LSADT is death.

Procedure

The participants completed in-person interview assessments lasting approximately 1 h, usually in their residences. The interviews were conducted by interviewers employed by the Danish National Institute of Social Research and trained and monitored by David Gaist and Kaare Christensen, using the same procedures for all four waves (three LSADT and one MADT) of participants. Co-twins were interviewed independently by different interviewers. The interview included an assessment of health, diseases, medications, activities of daily living, cognitive functioning, and life circumstances and events, in addition to an adapted version of the depression section of the Cambridge Mental Disorders of the Elderly Examination (CAMDEX) (Roth *et al.* 1986).

The original 21 items from CAMDEX were supplemented with an additional 11 items to provide more comprehensive assessments of both depression history and current affective state. McGue & Christensen (1997) factor-analysed the resulting 21 items that related to current depression symptomatology, and selected a two-factor solution comprising 17 items. Nine of the selected items loaded primarily on the first factor, and could be characterized as affective symptoms reflecting lack of well-being. Eight of the selected items loaded primarily on the second factor. These items reflected cognitive difficulties, slowing, and lack of energy. The remaining four items did not load significantly on either factor and were dropped. Factor analysis of these same items on the current sample revealed that they performed in the same manner, so we made use of them in the same way for the current study. The items were scored as Affective, Somatic and Total scales, again in the same manner as in McGue & Christensen. The items from each scale are given in Table 1. Internal consistency reliability estimates were 0·78 for the Affective scale, 0·80 for the Somatic scale, and 0·86 for the Total scale for this sample. Two-year stability in scale scores was 0·63 for the Affective scale, 0·54 for the Somatic scale, and 0·64 for the Total scale in a subsample of 1733 individuals who have provided the second wave of data for LSADT. Estimates for the means were not adjusted for the clustering of twin pairs in the sample, as our focus in this large sample was on the estimates of the means themselves and not results of statistical tests of differences in means. It has been shown that the primary effect of clustering is on the standard errors and not parameter estimates (Collins & Hern, 1991).

The characteristics of the sample and resulting scale means and standard deviations are given in Table 2. Because the scores were highly positively skewed, they were log-transformed prior to analysis of twin similarity. Unlike normalizing transformations like the Blom transformation, the log transformation has the advantage of preserving phenotypic variance associated with age and sex, while reducing scale skewness. Moreover, the log transformation has the advantage that the transformed scores for individuals within the sample are not dependent on those of others within the sample. As we had no *a priori* hypothesis that the moderating effect of age would be linear (or even monotonic), we categorized the sample into five age groups: 45–50, over 50 to 60, over 60 to 70, over 70 to 80, and over 80. Prior to analysis of twin similarity, the scale scores were further adjusted to eliminate age and sex effects (McGue & Bouchard, 1984) by subtracting an age-sex-group-specific mean

Table 2. *Characteristics of the Danish Twin Sample*

Measure	Age group 1 (45–50)	2 (> 50–60)	3 (> 60–70)	4 (> 70–80)	5 (> 80)	Total
Number of pairs						2169
MZ males	62	153	118	117	21	471
MZ females	64	146	118	184	50	562
DZ males	63	141	106	167	19	496
DZ females	56	134	99	290	61	640
Males						
Affective scale score, mean	10·7	10·3	10·5	10·7	11·5	10·5
S.D.	2·4	1·8	2·2	2·3	2·7	2·2
Somatic scale score, mean	8·7	8·7	9·2	10·0	11·6	9·2
S.D.	1·8	1·6	2·1	2·6	3·8	2·1
Total scale score, mean	19·4	19·0	19·6	20·6	23·1	19·7
S.D.	3·8	3·0	3·9	4·4	6·0	3·8
Females						
Affective scale score, mean	10·6	11·0	11·0	11·6	12·2	11·2
S.D.	2·1	2·6	2·5	3·0	3·5	2·7
Somatic scale score, mean	8·7	9·1	9·3	10·1	11·2	9·6
S.D.	1·5	2·0	2·2	2·7	3·4	2·3
Total scale score, mean	19·3	20·0	20·2	21·6	23·4	20·8
S.D.	3·1	4·0	4·3	5·0	6·1	4·5

MZ, monozygotic; DZ, dizygotic.

from each log-transformed score. All analyses of twin similarity in this study were based on the log-transformed, age-sex adjusted scores.

To estimate the heritability of the individual depression scales by age group and sex, we analysed the twin data with biometric models following standard biometric procedures (Neale & Cardon, 1992). Thus, we assumed that the total variance in a scale could be decomposed as follows:

$$V = A + C + E, \quad (1)$$

where A represents the variance contributed to the total by additive genetic effects, C represents the variance contributed to the total by environmental factors shared by reared-together twins (and thus sources of their behavioural similarity), and E represents the variance contributed to the total by environmental factors not shared by reared-together twins (and thus sources of their behavioural dissimilarity). Assuming that shared environmental effects contribute equally to the similarity of MZ and DZ twins, the expected twin covariances are given by

$$\begin{aligned} &A + C \text{ for MZ twins} \quad \text{and} \\ &1/2\ A + C \text{ for DZ twins.} \end{aligned} \quad (2)$$

We estimated the variance components from the observed twin variances and covariances using the method of maximum likelihood with the Mx software package (Neale, 1994). We began by fitting the same biometric model to the twin data for each scale and each age-sex group. We evaluated each model on the basis of model fit (that is, whether it had a non-significant χ^2 goodness of fit statistic) and parsimony (that is, none of the parameters in the model could be deleted without a significant increase in the χ^2 statistic). The Akaike information criterion (AIC $= \chi^2 - 2df$); (Akaike, 1983) provides a summary index of both fit and parsimony; models that have large negative AIC values are preferred over models with smaller negative or positive AIC values.

RESULTS

Scale means

Fig. 1 shows the mean Affective and Somatic scale scores by age group for males and females. As also observed by Newman (1989), the means for this sample increased almost monotonically with age for both scales in both sexes. The rate of increase was minimal prior to Age Group 3 (the 60s), and then more rapid. The mean scale scores increased more rapidly for males than for

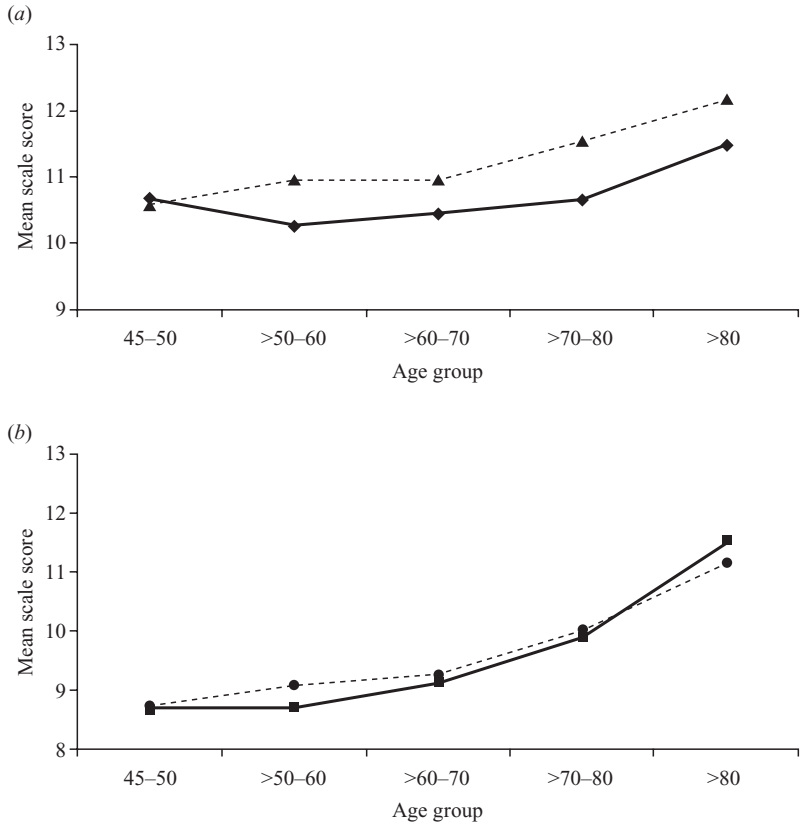

FIG. 1. Depression scale means by age group: (*a*) Affective scale (◆, male; ▲, female); (*b*) Somatic scale (■, male; ●, female).

females, so that by Age Group 5 (over 80), the scores for males had reached those for females. For the Affective scale, two-way analysis of variance revealed significant ($P < 0{\cdot}001$) mean differences by age group and sex, as well as a significant ($P < 0{\cdot}05$) age × sex interaction. For the Somatic scale, mean differences by age group were significant ($P < 0{\cdot}001$), and there was a significant ($P < 0{\cdot}05$) age × sex interaction. Mean differences by sex were not significant, however.

To evaluate the potential impact of contact between members of the twin pairs on level of depression symptomatology, we compiled data from 526 study participants who also provided data regarding the frequency with which they get together and speak on the telephone. Twins' get-together scores correlated 0·88, and phone-contact scores correlated 0·90, establishing the reliability of these measures. Using double-entered MZ twin pairs, we regressed Twin 1's Total scale score on Twin 2's, and compared the results to regressions including each contact score and the products of contact score and Total scale score. None of the added regression coefficients was significant, and the overall significance levels of the regressions decreased with the inclusion of the additional terms. Thus, we concluded that frequency of contact had no significant effect on twin similarity for depression symptomatology.

Twin correlations

The twin intraclass correlations for the three log-transformed and age-sex-corrected depression scale scores are given in Table 3. The MZ correlations generally exceeded the corresponding DZ correlations, and many differed by approximately a factor of 2, which is the amount of difference predicted by an additive genetic model without shared environmental effects. These observations implicate heritable effects on

Table 3. *Twin intraclass correlations for sex-adjusted, log-transformed depression scale scores by age group and sex*

	Age group					
	1 (45–50)	2 (> 50–60)	3 (> 60–70)	4 (> 70–80)	5 (> 80)	All
Affective scale						
MZ						
Males	0·23	0·17	0·32	0·33	0·32	0·27
Females	0·41	0·18	0·23	0·32	0·34	0·28
All	0·32	0·18	0·27	0·32	0·34	0·28
DZ						
Males	0·38	0·04	0·13	0·16	−0·03	0·14
Females	0·15	0·06	0·16	0·08	0·19	0·10
All	0·27	0·05	0·14	0·10	0·14	0·12
Somatic scale						
MZ						
Males	0·15	0·15	0·28	0·24	−0·09	0·20
Females	0·35	0·18	0·37	0·28	0·41	0·29
All	0·25	0·17	0·32	0·26	0·27	0·25
DZ						
Males	0·10	0·27	−0·08	0·08	0·19	0·09
Females	0·12	0·22	0·10	0·14	−0·06	0·12
All	0·09	0·24	0·01	0·12	−0·01	0·11
Total scale						
MZ						
Males	0·23	0·20	0·34	0·34	0·03	0·28
Females	0·38	0·17	0·37	0·33	0·45	0·32
All	0·31	0·18	0·36	0·34	0·34	0·30
DZ						
Males	0·29	0·17	0·08	0·09	0·20	0·14
Females	0·11	0·15	0·06	0·12	0·06	0·11
All	0·21	0·16	0·07	0·11	0·08	0·12

MZ, monozygotic; DZ, same sex dizygotic.
Table 3 gives numbers of twin pairs.

the depression scales, replicating the observations in McGue & Christensen (1997). Unlike their sample, however, this sample did not show higher MZ correlations for the Somatic scale than for the Affective scale. In fact, the reverse was true. As they observed, the correlations for females did tend to be higher than those for males.

Univariate biometric modelling

We investigated the heterogeneity of our parameter estimates across age groups by fitting 4 types of models with varying levels of constraint to the data for each scale. The first model was without constraint. For the second set of models, we constrained the genetic variance, the non-shared environmental variance, and the genetic and non-shared environmental variances together to be equal across age groups (we termed these absolute constraints). We also constrained the relative influence of genetic factors (i.e. heritability) to be equal across age groups (which we termed a relative constraint). For the third set of models, we applied the same set of absolute and relative constraints over sex, and for the fourth set of models we constrained estimates over both age and sex.

Our initial biometric modelling analyses included the C parameter for shared environmental variance, but we eliminated it from further analysis because it was 0 for all but two of the scale/age group combinations and near 0 in the other two cases, and its elimination did not significantly increase the χ^2 statistics of model fit. In no case could we eliminate the A parameter for genetic variance without causing a significant increase in the χ^2 test statistic relative to models that included the A parameter. Thus, we report test statistics and parameter estimates for the AE models only, subject to varying constraints.

Table 4. *Test statistics for AE models for the three depression scales varying across age and sex*

Models	χ^2	df	*P*	AIC
Affective scale				
No constraints	22·93	20	0·29	−17·07
Absolute constraint on A over age	32·05	28	0·27	−23·95
Absolute constraint on E over age	29·98	28	0·36	−26·02
Absolute constraint on A and E over age	46·50	36	0·11	−25·51
Relative constraint on age	30·36	28	0·35	−25·64
Absolute constraint on A over sex	23·69	25	0·54	−26·31
Absolute constraint on E over sex	35·97	25	0·07	−14·03
Absolute constraint on A and E over sex	55·45	30	0·00	−4·55
Relative constraint on sex	23·79	25	0·53	−26·21
Absolute constraint on A over age and sex	33·27	29	0·27	−24·73
Absolute constraint on E over age and sex	40·56	29	0·08	−17·44
Absolute constraint on A and E over age and sex	75·55	38	0·00	−0·45
Relative constraint on age and sex	**30·37**	**29**	**0·40**	**−27·63**
Somatic scale				
No constraints	40·75	20	0·00	0·75
Absolute constraint on A over age	46·28	28	0·02	−9·72
Absolute constraint on E over age	93·73	28	0·00	37·73
Absolute constraint on A and E over age	176·94	36	0·00	104·94
Relative constraint on age	47·29	28	0·01	−8·71
Absolute constraint on A over sex	45·37	25	0·01	−4·63
Absolute constraint on E over sex	54·32	25	0·00	4·32
Absolute constraint on A and E over sex	68·76	30	0·00	8·76
Relative constraint on sex	48·20	25	0·00	−1·80
Absolute constraint on A over age and sex	51·43	29	0·01	−6·57
Absolute constraint on E over age and sex	93·82	29	0·00	35·82
Absolute constraint on A and E over age and sex	188·63	38	0·00	112·63
Relative constraint on age and sex	**45·22**	**29**	**0·03**	**−12·78**
Total scale				
No constraints	17·36	20	0·63	−22·64
Absolute constraint on A over age	22·91	28	0·74	−33·09
Absolute constraint on E over age	26·07	28	0·57	−29·93
Absolute constraint on A and E over age	39·41	36	0·32	−32·59
Relative constraint on age	22·14	28	0·78	−33·86
Absolute constraint on A over sex	18·41	25	0·83	−31·59
Absolute constraint on E over sex	24·23	25	0·51	−25·77
Absolute constraint on A and E over sex	32·50	30	0·35	−27·50
Relative constraint on sex	18·53	25	0·82	−31·47
Absolute constraint on A over age and sex	24·32	29	0·71	−33·68
Absolute constraint on E over age and sex	27·72	29	0·53	−30·29
Absolute constraint on A and E over age and sex	47·63	38	0·14	−28·37
Relative constraint on age and sex	**22·35**	**29**	**0·81**	**−35·65**

Absolute constraint refers to constrained variance parameter; relative constraint refers to constrained heritability; AIC is Akaike Information Criterion; best-fitting model is shown in boldtype. See text for discussion.

The variances of scale scores varied considerably from age group to age group and Twin 1 and Twin 2 variances were not always similar, decreasing the degree to which the models fit. The no constraint model did fit well for the Affective and Total scales, however, and, for both the Affective and Somatic scales, the model applying relative constraints on age and sex provided the best balance between model fit and parsimony as measured by the AIC. Even for the Somatic scale for which all the models fit less well, the model applying relative constraints on age and sex fit significantly better than any other.

Heritability of the Somatic scale was slightly higher in females, but recognition of the difference did not improve model fit. For the Affective scale, no sex difference was apparent. The model fit statistics for the models tested are shown in Table 4. The heritability estimates for the general model and for the best-fitting model for each scale are shown in Table 5, along with the 95% CIs for the heritabilities from the best-fitting model. The heritability estimates were as follows: 0·27 for the Affective scale (95% CI 0·22–0·32), 0·26 for the Somatic scale (95% CI (0·21–0·32) and 0·29 for the Total scale (95% CI (0·22–0·34)).

Table 5. *Parameter estimates for general and best-fitting models*

Age group	Males			Females		
	Phenotypic variance	General model a^2	Best-fitting model a^2	Phenotypic variance	General model a^2	Best-fitting model a^2
Affective scale						
1 (45–50)	0·46	0·32	0·27	0·45	0·38	0·27
2 (> 50–60)	0·38	0·16	0·27	0·54	0·18	0·27
3 (> 60–70)	0·48	0·32	0·27	0·56	0·25	0·27
4 (> 70–80)	0·48	0·32	0·27	0·60	0·28	0·27
5 (> 80)	0·53	0·37	0·27	0·59	0·35	0·27
	95% CI for a^2 in best-fitting model: 0·22–0·32					
Somatic scale						
1 (45–50)	0·32	0·18	0·26	0·30	0·34	0·26
2 (> 50–60)	0·32	0·24	0·26	0·47	0·23	0·26
3 (> 60–70)	0·44	0·21	0·26	0·47	0·36	0·26
4 (> 70–80)	0·59	0·22	0·26	0·58	0·30	0·26
5 (> 80)	0·73	0·01	0·26	0·67	0·31	0·26
	95% CI for a^2 in best-fitting model: 0·21–0·32					
Total scale						
1 (45–50)	0·62	0·32	0·29	0·61	0·36	0·29
2 (> 50–60)	0·56	0·23	0·29	0·74	0·20	0·29
3 (> 60–70)	0·71	0·33	0·29	0·75	0·34	0·29
4 (> 70–80)	0·71	0·30	0·29	0·78	0·32	0·29
5 (> 80)	0·76	0·16	0·29	0·72	0·39	0·29
	95% CI for a^2 in best-fitting model: 0·22–0·34					

a^2, Heritability; general model lets all parameters vary freely; best-fitting models allow constraints that do not cause significant increases in χ^2 statistic from general model.

Bivariate analysis

The correlations between Affective and Somatic scores within individual persons and between one twin and another (cross-twin correlations) can help to clarify the relationship between the two characteristics within an individual. These correlations are shown in Table 6. Consistent with previous studies (Berkman *et al*. 1986; Kessler *et al*. 1992; McGue & Christensen, 1997), there was a substantial within-person correlation between the two scales for both males and females. If the relationship between two scales is primarily due to non-shared environmental effects, then both the MZ and DZ correlations should be near 0. On the other hand, if the relationship is primarily due to genetic effects, the MZ cross-twin correlation should be higher than the DZ correlation and should approach the within-person correlation. The data in Table 6 suggest some genetic involvement in the relationship, though they also suggest non-shared environmental influences.

We fit several bivariate Cholesky models (Neale & Cardon, 1992) to the observed twin data to assess formally the genetic correlation between the Affective and Somatic scales. The model that provided the best balance between fit and parsimony equated the genetic and environmental correlations across the age and sex groups. The genetic correlation between the scales in this model was 0·71 (95% CI 0·61–0·81), and the environmental correlation was 0·44

Table 6. *Within person and cross-twin correlations between Affective and Somatic scales*

Age group	Within person	Cross-twin	
		MZ	DZ
Males			
1 (45–50)	0·52	0·12	0·11
2 (> 50–60)	0·44	0·12	0·11
3 (> 60–70)	0·54	0·24	−0·02
4 (> 70–80)	0·47	0·21	0·00
5 (> 80)	0·56	0·12	0·01
Females			
1 (45–50)	0·50	0·17	−0·05
2 (> 50–60)	0·46	0·10	0·02
3 (> 60–70)	0·52	0·30	0·02
4 (> 70–80)	0·52	0·20	0·08
5 (> 80)	0·51	0·32	−0·15

The cross-twin correlation is the correlation between the Affective score of the first member of a twin pair and the Somatic score of the second member (MZ, monozygotic; DZ, dizygotic).

(95% CI 0·40–0·48). The bivariate heritability, or the proportion of the phenotypic correlation that can be accounted for by common genetic effects, averaged 0·38 across the age and sex groups. This suggests that genetic effects that act on both scales account for some portion of the phenotypic variance. In addition, each scale does appear to have independent influences, both genetic and environmental. Though this model fit the data the best, its fit did not differ significantly from those of several of the others tested, including models equating only the genetic correlation and models equating the genetic variance as well as the genetic and environmental correlations. The parameter estimates resulting from those models were very similar to those reported here.

DISCUSSION

This study consisted of the analysis of twin resemblance for depression symptomatology in a sample of Danish twins aged 45 and older. Its strengths include the large sample size and age range and the consistency of instruments and administration procedures. The analyses revealed that depression is moderately heritable among older people. The remainder of the variance in depression is due to non-shared environmental circumstances. Thus, though the frequency of depression symptoms in the second half of life is somewhat heritable as it is in the first half, it remains primarily the result of circumstances and characteristics unique to each individual. Our data also did not reveal any differences in the heritability of depressive symptoms across the age range of our sample. As that range extended from 45 to over 95, it encompassed the full range that could be considered practically available for research analysis. Thus, though the frequency of depression symptoms increases with age, the increase appears to be the result of increasing occurrence of circumstances that instill depression. This appears to be true for both men and women, as our data showed no difference in the heritability of depression between men and women.

Like McGue & Christensen's (1997) more limited sample, our data revealed substantial common genetic effects on the Affective and Somatic scales. That is, the genetic correlation was 0·71. Though it did differ significantly from 1·0, a large portion of the heritable variance could be attributed to a single common factor. This corroborates their belief that the association between the two scales is fundamental.

Though our study was not designed to address alternative mechanisms for the association between affective and somatic symptoms of depression, our results do provide some data regarding mechanisms that have been suggested. Because of the wide age range of our sample, the evidence for a common genetic factor and the stability of our heritability estimates suggest that the association between the scales is more than simply a direct effect of deteriorating physical health due to age on both somatic and affective symptoms, as hypothesized by Murrell, *et al*. (1983) for example. This will be the subject of further investigations in future, making use of illness, medication and physical and cognitive functioning data included in the sample assessment. At the same time, our analysis does not eliminate the possibility that the association reflects a core depressive syndrome in which somatic symptoms not directly associated with health complaints are primary, (as hypothesized by Newman *et al*. 1991), though the association could also reflect a core depressive syndrome in which affective symptoms lead to somatic symptoms. The particular somatic and/or affective symptoms causing the increased depression with age are apparently not heritable, however, as the heritability of both affective and somatic symptoms is stable with age.

This study had three major limitations. First, it is subject to the well-known limitations of its twin research design, particularly the equal-environments assumption (Plomin *et al*. 1990). This refers to the assumption that any greater similarity in MZ than DZ twins must be the result of their greater genetic similarity, rather than to any greater similarity in their environments. This assumption draws concern because of the possibility that parents treat identical twins more similarly than they do fraternal twins, a possibility that is probably not particularly relevant in a sample of elderly twins who presumably have lived independently for many years. In addition, examination of data regarding frequency of contact between twins, both by phone and in person, revealed no

relationship between frequency of contact and depression symptomatology. Of course, recent or current contact does not necessarily index common experience over a lifetime, so that the findings from this study should be replicated in other samples using different methodologies.

The second major limitation is the cross-sectional nature of the research design. Cohort and period differences can increase the variance from age group to age group in this design without reflecting any increase in genetic variance. Such differences may possibly underlie the large and inconsistent differences in variance from age group to age group in this sample, and these differences could conceivably mask differences in heritability with age. It is worth noting that the one longitudinal study of depression in the elderly to date (Carmelli *et al.* 2000) did report an increase in heritability of depression symptoms with age, though the sample was considerably smaller than ours. The LSADT study providing data for this analysis involves several waves of data collection from the same participants, so future longitudinal analyses will be possible. Such analyses should also seek to clarify the interrelationship between the Affective and Somatic scales in use.

A third limitation is the dependence of the results on time-specific depressive symptoms. That is, co-twins were only considered to be similar to the extent they report similar levels of depressive symptoms at the same time. Thus, the heritability of reliable variance in depressive symptoms at some time during the second half of life may be considerably higher than we were able to measure. To provide perspective in evaluating the potential impact of this, we note that the 2-year stability in CAMDEX scale scores was 0·63 for the Affective scale, 0·54 for the Somatic scale and 0·64 for the Total scale in a subsample of 1733 individuals who have provided the second wave of data for LSADT.

Finally, this study is also limited in its assessment of depression due to its self-report format. The CAMDEX was selected for use in this study because it had been previously translated and used successfully in Danish populations. Still, we have no evidence of the nature of the relationship between the Danish version of the CAMDEX and clinician-diagnosed depression so the clinical significance of the depression scores we used is unknown.

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