Depression, cerebral atrophy, cognitive performance and incidence of dementia

Population study of 85-year-olds

SIGURDUR PÁLSSON, ÓLAFUR AEVARSSON and INGMAR SKOOG

Background Hospital-based studies suggest that depression in old age relates to organic brain changes.

Aims To determine whether these findings are confirmed in a population-based sample.

Method A population sample of non-demented 85-year-olds (227 mentally healthy and 62 with DSM–III–R depression were given a neuropsychiatric examination and computerised tomographic scans of the brain, and followed for three years.

Results On the Mini-Mental State Examination, those with a low educational level with major depression performed worse than the mentally healthy; this distinction was not evident among those who had received higher education. Measures of brain atrophy were similar in depressed and mentally healthy individuals. The three-year incidence of dementia was increased in those with early-onset major depression.

Conclusions Higher education may protect against cognitive symptoms in depressed individuals. The association between depression and cerebral atrophy in the elderly is not very strong. The higher incidence of dementia in those with early-onset major depression may be due to a longer lifetime duration of depression, emphasising the importance of detecting and treating depression in the community.

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A number of hospital-based brain-imaging studies report that depression in the elderly is associated with structural changes in the brain (Jacoby & Levy, 1980; Greenwald *et al*, 1997). It is not clear whether depression diagnosed in the general population is also related to structural brain changes. However, population-based studies report that cognitive impairment is common in elderly depressed individuals (Van Ojen *et al*, 1995). Cognitive impairment may also be secondary to depression (American Psychiatric Association (APA), 1987). Finally, depression has been suggested as a risk factor for the later development of dementia (Devanand *et al*, 1996), although this has not been shown consistently (Pálsson & Skoog, 1997). We examined depression in relation to cognitive function, incidence of dementia and brain atrophy measured by computed tomography (CT) in a population-based sample of non-demented 85-year-olds followed for three years.

METHOD

Subjects

This study is part of the Longitudinal Gerontological and Geriatric Population Studies in Gothenburg (Skoog *et al*, 1993*b*; Steen & Djurfeldt, 1993). In 1986–87, all 85-year-olds born between 1 July 1901 and 30 June 1902, registered for census purposes in Gothenburg, were invited to take part in a health survey. Both people living in the community and those in institutions were included. A neuropsychiatric examination was performed on a systematic subsample (n=494; 144 men and 350 women). This sample has been described in detail previously (Skoog *et al*, 1993*b*), and was representative with regard to gender, marital status, three-year mortality rate, institutionalisation and registration as a psychiatric out-patient or in-patient in Gothenburg (Skoog *et al*, 1993*b*). Individuals with dementia (n=147) or mental disorders other than depression according to DSM–III–R (APA, 1987) (schizophrenic/schizophreniform disorder, delusional disorder, psychosis not otherwise specified (NOS), phobic disorder, obsessive–compulsive disorder and generalised anxiety disorder) (n=58) were excluded, leaving 289 85-year-olds (62 depressed, 227 mentally healthy) for study.

A follow-up examination was performed three years later when the subjects were 88 years old (Aevarsson & Skoog, 1996). Out of the original sample of 289 non-demented, mentally healthy or depressed 85-year-olds, 62 had died and 72 refused further examinations, but 155 took part in the neuropsychiatric examination at the age of 88. Information regarding dementia on 112 of the individuals who had died or refused was obtained from medical records or other sources (Aevarsson & Skoog, 1996); no information was available on 22 subjects. Sufficient information for a diagnosis of dementia was thus obtained for 267 individuals (92.4% of the population at risk, 210 mentally healthy, 57 depressed). The number of new cases of dementia was 50 (11 men and 39 women). Of these, 33 were traced from the neuropsychiatric examination and 17 from case records or other information.

The study was approved by the Ethics Committee, Göteborg University. All participants (or their nearest relatives) gave their informed consent to participation in the study.

Examinations and diagnoses

The examinations included a physical examination performed by a geriatrician, an electrocardiogram, a chest X-ray, a battery of blood tests, CT scans of the brain and a neuropsychiatric examination performed by a psychiatrist (Skoog *et al*, 1993*b*; Steen & Djurfeldt, 1993).

The neuropsychiatric examination was semi-structured and included tests of mental functioning, including the Mini-Mental State Examination (MMSE; Folstein *et al*, 1975), ratings of psychiatric symptoms and signs during the preceding month and ratings of dementia symptoms (Skoog *et al*, 1993*b*; Aevarsson & Skoog, 1996). In a semi-structured telephone interview, informants close to the subjects were asked about symptoms and signs of dementia.

Depressive disorders (38 major depressive disorders (MDS), 22 dysthymic disorders and two with depression NOS) during the last month were diagnosed according

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to DSM–III–R (APA, 1987), as described previously (Skoog *et al*, 1993*a*).

Early-onset depressive disorder was defined as depression where the first onset occurred before the age of 65, and late-onset depression as depression where the first episode occurred after that age, based on information from the subjects themselves and from medical records from general hospitals, psychiatric hospitals and out-patient clinics in the area. There were 12 cases of early-onset (11 MDS, one dysthymic) and 50 of late-onset depression (27 MDS, 21 dysthymic, 2 depression NOS) at age 85. Among the early-onset cases, five were identified by history, two by case records, and five by both sources.

Cognitive function

Cognitive function was measured by the MMSE, a simple instrument measuring global cognitive function, with a maximum score of 30. If individual items could not be completed because of physical disability, the subjects were regarded as not testable. If individual items were refused, zero scores were given.

Computed tomography

A systematic subsample of 269 non-demented 85-year-olds was invited to undergo a CT scan of the head; 136 accepted. The participants and non-participants were similar with respect to a number of factors (Skoog *et al*, 1994), and their mean MMSE values did not differ significantly (27.9 (s.d. 2.3) *v.* 27.5 (s.d. 2.0); P=0.171). One CT scan was excluded for technical reasons, and 25 individuals who had a CT scan were excluded because they suffered from a mental disorder other than depression, leaving 110 individuals (21 depressed, 89 mentally healthy).

All CT scans were performed without contrast enhancement, and with 10-mm continuous slices, on a Philips Tomoscan 310 or on a General Electric 8800. The scans were examined by two experienced radiologists, who were blind to the results of the neuropsychiatric examination. The occipital, parietal, frontal and temporal lobes were categorised using a three-point scale (normal, mild and moderate or severe) according to the estimated extent of brain atrophy (De Leon *et al*, 1980). The following linear distances were determined, using a transparent metric ruler as described by De Leon *et al*: (a) the bifrontal span of the lateral ventricle; (b) the width of the lateral ventricles at the head of the caudate nucleus; (c) the sum of the separate widths of the left and right Sylvian fissures; (d) the minimum width of the bodies of the lateral ventricles at the waist; and (e) the width of the third ventricle. Ratios for (a) to (d) were determined by dividing the values obtained by the width of the brain at the level of the measurement, giving the following ratios: (f) bifrontal ratio, (g) bicaudate ratio, (h) Sylvian fissure ratio, and (i) sella media ratio. The rating procedure was carried out separately for the cortical and ventricular studies.

Statistical analysis

Fisher's exact test, the χ^2 test and the permutation t-test were used to test the hypothesis of no differences between the groups (Cox & Hinkley, 1974). The Spearman rank correlation coefficient was used to test correlations (r) (Cox & Hinkley, 1974); two-tailed tests were used. The incidence (I) of dementia was based on person–years at risk as described previously (Aevarsson & Skoog, 1996) and computed as:

$$I = \frac{\text{subjects affected in the interval}}{\text{sum of risk years}}.$$

RESULTS

Cognitive performance in relation to depression at the age of 85

Twenty-four 85-year-olds (six depressed and 18 mentally healthy) could not be assessed by the MMSE because of handicaps such as paresis, blindness or hearing loss, leaving 265 (56 depressed and 209 mentally healthy) for study. Individuals with MDS, but not those with dysthymic disorder, had a lower mean MMSE score than the mentally healthy (Table 1). Early-onset (n=11) and late-onset depressives (n=45) had similar mean MMSE scores (27.0 (s.d. 1.7) *v.* 27.1 (s.d. 2.9), P=0.901).

When the sample was divided into those with six years or less of compulsory education (only one subject had less than six years' education) and in those with more than six years' education, MDS was found to be related to a lower score on the MMSE only in those with the lower level of education (Table 1). The proportion of subjects with depression did not differ between the educational levels (six years' education or less: 20.2% (95% CI 14.7–26.7) (n=38/188); more than six years' education: 23.2% (95% CI 13.9–34.9) (n=16/69); P=0.607)).

An ordinary stepwise linear regression analysis showed that a low score on the MMSE was related to depression (B=−1.15; s.e.=0.57; P=0.047; f=4.07), but not to any of the linear ventricular measures.

Brain atrophy at the age of 85 in relation to depression

There were no differences between depressed and mentally healthy 85-year-olds regarding CT linear ventricular measurements (Table 2). There were no significant associations between depression and frontal (r=−0.07, P=0.692), temporal (r=0.00, P=0.758), parietal (r=0.03, P=0.814) or occipital (r=−0.03, P=0.899) cortical atrophy. There was no difference as regards CT measurements between early- and late-onset cases of depression. When the sample was divided into those with six years' education or less and those with more than six years' education, depression was found to be related to a larger Sylvian fissure ratio in those with the higher level of education (n=6, 222.0 (s.d. 68.9) *v.* 166.7 (s.d. 26.6); P=0.013), but not to any other CT measures in any of the groups.

Depression at the age of 85 in relation to the incidence of dementia between the ages of 85 and 88

During the three-year follow-up, 37 out of 210 (17.6%) mentally healthy and 13 out of 57 (22.8%) depressed 85-year-olds became demented. The incidence of dementia (based on person–years at risk) between the ages of 85 and 88 in mentally healthy

Table 1 Mean Mini-Mental State Examination score in mentally healthy and depressed 85-year-olds

	n	Mean (s.d.)
More than 6 years' education		
Mentally healthy	53	28.7 (1.2)
All depressed	16	28.6 (1.2)
Major depressive syndrome	11	28.3 (1.2)
Dysthymic disorder	4	29.8 (0.5)
6 years' education or less		
Mentally healthy	150	27.5 (2.0)
All depressed	38	26.5 (2.8)*
Major depressive syndrome	22	26.0 (3.3)*
Dysthymic disorder	15	27.3 (1.7)

*P < 0.05 compared with mentally healthy individuals. No information on educational level of 8 subjects.

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Table 2 Ventricular linear measurements on CT scans of the brain in mentally healthy and depressed 85-year-olds

	Mentally healthy (n=89)		All depressed (n=21)			Major depression (n=10)			Dysthymic disorder (n=10)		
	Mean	(s.d.)	Mean	(s.d.)	P	Mean	(s.d.)	P	Mean	(s.d.)	P
Bicaudate ratio	16.7	(3.6)	16.5	(2.6)	0.885	16.3	(2.3)	0.793	15.9	(2.8)	0.650
Bifrontal ratio	37.8	(4.2)	38.0	(4.6)	0.837	38.2	(4.4)	0.706	36.8	(4.2)	0.578
Sella media ratio	26.0	(3.7)	26.5	(4.1)	0.359	27.2	(4.0)	0.238	25.4	(4.1)	0.851
Sylvian fissure ratio	17.6	(3.9)	19.4	(5.4)	0.217	19.7	(6.4)	0.548	19.8	(4.8)	0.172
Third ventricle width (mm)	8.6	(1.6)	9.1	(1.9)	0.311	9.1	(1.9)	0.378	8.6	(1.4)	0.907

P-values between depressed and mentally healthy individuals.

Table 3 Incidence of dementia between age 85 and 88 in relation to depression at age 85

Status at age 85	All n	Years at risk	No. of cases dementia	Incidence per 1000 person–years			
				95% CI	OR[1]	95% CI[1]	P[1]
Mentally healthy	210	489	37	76	(54–103)		
Any depression	57	121	13	107	(58–177)	1.5	(0.8–2.9)	0.255
Early-onset[2]	11	22	4	184	(53–408)	2.8	(0.9–8.6)	0.069
Late-onset[2]	46	99	9	91	(42–165)	1.2	(0.6–2.6)	0.614
Major depressive disorder[3]	34	69	10	144	(71–249)	2.1	(1.0–4.3)	0.055
Early-onset	10	19	4	208	(60–452)	3.2	(1.0–10.2)	0.036
Late-onset	24	50	6	120	(45–242)	1.7	(0.7–4.1)	0.287
Dysthymia	22	49	3	61	(13–169)	0.8	(0.2–2.7)	0.718

1. OR=odds ratio compared to mentally healthy; P is compared to mentally healthy; CI=confidence interval.
2. Early-onset v. late-onset: OR=2.3 (95% CI 0.6–8.2); P=0.202.
3. Early-onset MDS v. late-onset MDS: OR=1.9 (95% CI 0.5–7.8); P=0.346.

individuals and in depressed individuals did not differ significantly (Table 3). The results were similar for men and women, and for the two educational groups.

Four out of 11 (36%) people with early-onset depression and nine out of 46 (20%) with late-onset depression became demented during the three-year follow-up. The incidence of dementia (based on person–years at risk) between the ages of 85 and 88 was higher in those with early-onset MDS than in the mentally healthy (Table 3).

DISCUSSION

Depression and cerebral atrophy

This is the first survey to examine the relationship between depression diagnosed in the general population and measures of cerebral atrophy. Other brain imaging studies have included patients recruited from hospitals or out-patient departments with more severe depression, and more 'difficult' cases. In common with other population studies (Copeland *et al*, 1992), the majority of depressed people in our study were not known to the medical profession, and only 19% were treated with antidepressants (Skoog *et al*, 1993*a*). Most hospital-based studies report that depressed patients have more ventricular enlargement and higher indices of cortical atrophy than controls (Elkis *et al*, 1995), especially among the elderly (Jacoby & Levy, 1980; for review see Pálsson & Skoog, 1997). In our study, the width of the ventricles and the degree of cortical atrophy did not differ between depressed people and controls. The more generalised cerebral atrophy found in depressed patients in hospital-based studies might reflect a more severe or chronic illness, often including subjects with organic brain disorders. However, even in these studies, the differences between depressed individuals and controls are often subtle (Elkis *et al*, 1995; Pálsson & Skoog, 1997). Our findings suggest that if there is an association between depression and cerebral atrophy in the elderly, it is not very strong.

Depression as a risk factor for dementia

Evidence in the literature as to whether depression is a risk factor for dementia is contradictory. One prospective study reports an association (Devanand *et al*, 1996), while others report no association (Copeland *et al*, 1992; Henderson *et al*, 1997). None of these studies gives the age at onset of depression. The meta-analyses of case–control studies by Jorm *et al* (1991) suggested that both early- and late-onset depression were associated with an increased risk of dementia. In our study, both early-onset and late-onset depressed people showed mild cognitive impairment, but only the small group of early-onset MDS had an increased risk of developing dementia. This supports the finding by Speck *et al* (1995) that depression which had begun 10 or more years before the onset of Alzheimer's disease was a stronger risk factor for dementia than onset of depression within 10 years of the onset of Alzheimer's disease. Depression is associated with disturbances in the hypothalamic–pituitary–adrenal axis with increased cortisol secretion (O'Brien, 1997). It is possible that those with early-onset MDS have a longer lifetime duration of depression than those with late-onset MDS, and

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thus have a longer exposure to increased levels of glucocorticoids, which may lead to brain damage, especially in the hippocampus (O'Brien, 1997). Our findings, and those of others (Copeland *et al*, 1992; Henderson *et al*, 1997), suggest that if there is also an increased risk of dementia in late-onset depressives, the association is not very strong.

Depression and cognitive impairment

In line with the results of studies (Van Ojen *et al*, 1995) of younger samples of elderly individuals, we found that depressed 85-year-olds had mild cognitive impairments, measured with the MMSE. We further noted that this cognitive dysfunction was only found in those with major depressive syndrome and in the group with the lower level of education. It is possible that depressed individuals with higher education may compensate better for the associated cognitive dysfunction than those with lower education; or alternatively, that higher education protects against cognitive dysfunction in depression. Our results are noteworthy because of the relatively high educational level in the Swedish population. Almost all subjects had at least six years of schooling, and none was illiterate. In our study, cognitive dysfunction in depressed individuals was not found to be related to structural brain changes, which is contrary to the finding of Greenwald *et al* (1997), who reported that widening of the third ventricle correlated with poorer performance on the MMSE in depressed individuals. Cognitive impairment is a well-established symptom of depression (APA, 1987). Considering that the large majority of depressed individuals had late-onset depression, which was not related to later development of dementia, our findings suggest that the cognitive dysfunction noted in our study is secondary to the depression and not related to organic brain changes or incipient dementia. The findings emphasise the importance of not misdiagnosing elderly patients with late-onset depression as having incipient dementia.

We found no difference between early- and late-onset cases regarding cognitive performance and brain atrophy, which contradicts the results of other studies, which report that elderly individuals with late-onset depression perform worse on cognitive tests (Van Ojen *et al*, 1995) and show more changes on brain imaging (Greenwald *et al*, 1997) than early-onset cases. One reason why we got different results may be the great age of our sample; another may be that we used different methods to obtain information regarding onset of depression. In most studies, onset is determined by information from the participants themselves, whereas we also included information from case records. This might have reduced the influence of under-reporting the individuals with incipient dementia and poor memory. Our finding that 11/12 early-onset cases had MDS suggests that severe depressive episodes are more likely to be remembered.

Methods

Some factors relating to method have to be considered. First, linear measurements and subjective ratings are rather crude measures of cerebral atrophy, which may be one reason why we failed to detect a relationship between brain atrophy and depression. Another possibility is that CT is less sensitive than magnetic resonance imaging. However, Elkis *et al* (1995), in their meta-analysis of 61 studies, reported that the imaging method was not systematically related to the magnitude of the study effect. It is thus not likely that our findings could be explained by the imaging method used.

A second limitation is that the statistical power was too weak to detect small differences for some of the analyses (due to small numbers in some of the subgroups). It may be that a larger sample would have shown a significant association between, for example, depression and some of the brain atrophy measurements. However, the number of subjects studied in the CT and incidence studies is similar to, or larger than, those in most previous studies.

Third, although the sample was drawn from the general population, the response rate at baseline was only 63% (still quite a high response rate in this age group). The comparison between responders and non-responders showed, however, that the sample investigated could be considered representative of its population base (Skoog *et al*, 1993*b*). Further, only about half of those invited to have a CT scan accepted. However, this is not a low response rate for such an examination, considering that this is a population study. Furthermore, those who participated in the CT examinations did not differ from those who did not participate with regard to a number of factors, including scores on the MMSE (Skoog *et al*, 1994).

Fourth, it has to be emphasised that differential survival may be of importance in studies at this advanced age, and this may be relevant for depression, especially for early-onset depression, as depression is associated with an increased mortality rate throughout life. This factor may have decreased the association between early-onset depression and the incidence of dementia, as fewer individuals with early-onset depression survive to an age at which dementia is common. Furthermore, it may also decrease the possibility of finding an association between depression and brain atrophy, if the combination of these is associated with an increased mortality rate. In theory, if factors associated with an increased risk for dementia increase survival in subjects with early-onset depression, they may contribute to the relationships reported here. However, we believe that this explanation is unlikely.

In summary, we could not corroborate the common opinion that depression in the elderly is mainly a result of the structural brain changes that occur with ageing. We believe that the cognitive dysfunction in elderly depressed individuals, noted in this and other studies, is mainly a secondary phenomenon of depression. Individuals with a low level of education may be more prone to this consequence of depression than those with a high level of education. We also found that early-onset, rather than late-onset, MDS was associated with an increased incidence of dementia in 85-year-olds. This is possibly due to a longer duration of depression during life, maybe related to longer exposure to increased cortisol levels. Whether or not treatment of depression in younger years may prevent development of dementia in old age has yet to be elucidated. In light of the low treatment rate of depression, our findings further emphasise the importance of detecting and treating depression in the community.

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REFERENCES

Aevarsson, O. & Skoog, I. (1996) A population-based study on the incidence of dementia disorders between 85 and 88 years of age. *Journal of the American Geriatric Society*, **44**, 1455–1460.

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

Copeland, J. R. M., Davidson, I. A., Dewey, M. E., et al (1992) Alzheimer's disease, other dementias, depression and pseudodementia: prevalence, incidence and three-year outcome in Liverpool. *British Journal of Psychiatry*, **161**, 230–239.

Cox, D. R. & Hinkley, D. V. (1974) *Theoretical Statistics*. London: Chapman & Hall.

De Leon, M. J., Ferris, S. H., George, A. E., et al (1980) Computed tomography evaluations of brain–behaviour relationships in senile dementia of the Alzheimer's type. *Neurobiology of Aging*, **1**, 69–79.

Devanand, D. P., Sano, M., Tang, M.-X., et al (1996) Depressed mood and the incidence of Alzheimer's disease in the elderly living in the community. *Archives of General Psychiatry*, **53**, 175–182.

Elkis, H., Friedman, L., Wise, A., et al (1995) Meta-analysis of studies of ventricular enlargement and cortical sulcal prominence in mood disorders. Comparisons with controls or patients with schizophrenia. *Archives of General Psychiatry*, **52**, 735–746.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) "Mini-Mental state": a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, **12**, 189–198.

Greenwald, B. S., Kramer-Ginsberg, E., Bogerts, B., et al (1997) Qualitative magnetic resonance imaging findings in geriatric depression. Possible links between later-onset depression and Alzheimer's disease? *Psychological Medicine*, **27**, 421–431.

Henderson, A. S., Korten, A. E., Jacomb, P. A., et al (1997) The course of depression in the elderly: a longitudinal community-based study in Australia. *Psychological Medicine*, **27**, 119–129.

Jacoby, R. J. & Levy, R. (1980) Computed tomography in the elderly 3. Affective disorder. *British Journal of Psychiatry*, **136**, 270–275.

Jorm, A. F., van Duijn, C. M., Chandra, V., et al (1991) Psychiatric history and related exposures as risk factors for Alzheimer's disease: a collaborative re-analysis of case–control studies. *International Journal of Epidemiology*, **20** (suppl. 2), S43–S47.

O'Brien, J. T. (1997) The 'glucocorticoid cascade' hypothesis in man. Prolonged stress may cause permanent brain damage. *British Journal of Psychiatry*, **170**, 199–201.

Pálsson, S. & Skoog, I. (1997) The epidemiology of affective disorders in the elderly: a review. *International Clinical Psychopharmacology*, **12** (suppl. 7), S3–S13.

Skoog, I., Nilsson, L., Landahl, S., et al (1993a) Mental disorders and the use of psychotropic drugs in an 85-year-old urban population. *International Psychogeriatrics*, **5**, 33–47.

—, —, Palmertz, B., et al (1993b) A population-based study of dementia in 85-year-olds. *New England Journal of Medicine*, **328**, 153–158.

—, Palmertz, B. & Andreasson, L.-A. (1994) The prevalence of white-matter lesions on computed tomography of the brain in demented and non-demented 85-year-olds. *Journal of Geriatric Psychiatry and Neurology*, **7**, 169–175.

Speck, C. E., Kukull, W. A., Brenner, D. E., et al (1995) History of depression as a risk factor for Alzheimer's disease. *Epidemiology*, **6**, 366–369.

Steen, B. & Djurfeldt, H. (1993) The gerontological and geriatric population studies in Gothenburg, Sweden. *Zeitschrift für Gerontologie*, **26**, 163–169.

Van Ojen, R., Hooijer, C., Bezemer, D., et al (1995) Late-life depressive disorder in the community I. The relationship between MMSE score and depression in subjects with and without psychiatric history. *British Journal of Psychiatry*, **166**, 311–315.

CLINICAL IMPLICATIONS

- The association between depression and cerebral atrophy in the elderly is not very strong.
- Elderly people with depression in the community, mainly those with low education, show cognitive dysfunction, which is possibly secondary to the depression and not related to structural brain changes or incipient dementia. These findings should alert doctors not to misdiagnose elderly people with late-onset depression as having incipient dementia.
- Early-onset major depression is associated with an increased incidence of dementia, which may be related to a longer lifetime duration of depression. In the light of the low treatment rate of depression, our findings further emphasise the importance of detecting and treating depression in the community. Whether treating depression earlier may prevent the development of dementia in old age has to be elucidated.

LIMITATIONS

- Linear measurements and subjective ratings on CT are crude indicators of cerebral atrophy, possibly too crude to detect subtle differences in brain atrophy between depressed and mentally healthy individuals.
- Differential survival may influence the results of studies of the very old. This may be especially relevant for early-onset depression, as depression is associated with an increased mortality rate throughout life. Differential survival may also have decreased the association between depression and brain atrophy, if the combination of these is associated with an increased mortality rate.
- The statistical power may have been too weak to detect small differences for some of the analyses, owing to small numbers in some of the subgroups. However, the number of subjects studied in the CT and incidence studies is similar to, or larger than, that in most previous studies.

SIGURDUR PÁLSSON, MD, ÓLAFUR AEVARSSON, PhD, INGMAR SKOOG, PhD, Institute of Clinical Neuroscience, Department of Psychiatry, Sahlgrenska University Hospital, S-413 45, Göteborg, Sweden

Correspondence: Sigurdur Pálsson, MD, Department of Psychiatry, The University Hospital, Landspítalinn, PO Box 10, 121 Reykjavík, Iceland. Tel: +354 56 01 000 or +354 564 560 1718; Fax: +354 56 01 720; e-mail: sigpp@rsp.is

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