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Cognitive functions in depressive disorders: evidence from a population-based study

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ABSTRACT

Background. Most of the available evidence on the effects of depression is based on in- and outpatient samples focusing on individuals suffering from major depression. The aims of this study were to examine cognitive functioning in population-based samples and to determine whether cognitive performance varies as a function of depression subgroup.

Method. Population-based samples (aged 20–64 years) with major depression ($N=68$), dysthymia ($N=28$), mixed anxiety-depressive disorder ($N=25$) and minor depression ($N=66$) were examined on a variety of cognitive tasks (i.e. episodic memory, verbal fluency, perceptual-motor speed and mental flexibility). One hundred and seventy-five non-depressed individuals served as controls.

Results. The total group of depressed individuals showed impairments in tasks tapping episodic memory and mental flexibility. Of more interest, however, was the observation that the pattern of impairments varied as a function of depression subgroup: the major depression and mixed anxiety-depressive disorder groups exhibited significant memory dysfunction, whereas individuals with dysthymia showed pronounced difficulties in mental flexibility. Minor depression did not affect cognitive performance. Verbal fluency and perceptual-motor speed were not affected by depression.

Conclusions. These results indicate that persons with depressive disorders in the population exhibit cognitive impairments in tasks tapping episodic memory and mental flexibility and that cognitive impairment varies as a function of depressive disorder.

INTRODUCTION

Depressive disorder represents a public health problem with a lifetime prevalence varying between 10–25% for women and 5–12% for men (APA, 1994). Since Miller's (1975) early review, a number of studies have shown that both mild and severe forms of depression are associated with deficits on cognitive, motor, perceptual and communication tasks (Murphy *et al*. 1998). For example depression-related deficits have been documented in tasks tapping explicit memory (Bazin *et al*. 1994), verbal fluency (Ravnkilde *et al*. 2002), psychomotor speed (Austin *et al*. 1992) and attention (Landro *et al*. 2001).

It is of interest to note that episodic memory functioning appears as more vulnerable to the negative effects of depression as compared with other forms of memory (Ilsley *et al*. 1995). For example, studies have reported spared functions in tasks tapping implicit memory (Hertel & Hardin, 1990; Danion *et al*. 1995), semantic memory (Zakzanis *et al*. 1998) and short-term memory (Ilsley *et al*. 1995). In addition, two recent meta-analyses reported a significant stable association between depression and episodic memory impairments in both the young (Burt *et al*. 1995) and old (Kindermann & Brown, 1997), although the detrimental effect of depression on memory was greater among younger than older individuals. However, it is important to note that some studies have found no association between depression and episodic

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memory performance (e.g. Fossati *et al*. 1999; Grant *et al*. 2001). Possible explanations for these inconsistent findings may be differences across studies regarding the selection of participants, depression subtypes and treatment settings. Other potential sources for the mixed results are differences in the material used to measure memory performance (e.g. visual *v*. verbal), retention interval and the amount of effort during encoding and retrieval (e.g. recall *v*. recognition).

Recent neurobiological findings suggest that depressive disorder is associated with structural and functional changes in brain structures critical for episodic memory functioning. For example, research indicates an association between depressive disorder and reductions in the metabolic activity of the prefrontal cortex (Dolan *et al*. 1992; Drevets, 2000), and neural atrophy in limbic structures such as the hippocampus and amygdala have been reported (Sheline *et al*. 1998; McEwen & Magarinos, 2001).

Research focusing on other cognitive domains shows a mixed pattern of results. For example, Purcell *et al*. (1997) found that unipolar depression was related to impairments in motor speed and attention set-shifting tasks, while performance in tests of executive functioning, visual memory function, and cognitive speed was intact. In contrast with these findings, Grant *et al*. (2001) reported deficits in executive functioning in unmedicated depressed out-patients, whereas no impairments were observed in attention, psychomotor speed and episodic memory functioning as compared with a group of non-depressed. Other investigators have reported that major depression is associated with impairments in psychomotor speed (TMT-A) and mental flexibility (TMT-B), whereas verbal fluency proved to be unaffected (Austin *et al*. 1992).

Most of the available evidence focusing on the impact of depression on cognition is based on individuals suffering from major depression and little is known as to whether other depressive disorders (e.g. dysthymia) is associated with cognitive dysfunction. Also, in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (APA, 1994) depressive disorders were extended with two new research diagnoses: mixed anxiety-depressive disorder and minor depression. The DSM-IV field trial for mixed anxiety-depressive disorder suggested that this group experienced significant functional impairment (Zinbar *et al*. 1994). To the best of our knowledge, no research has specifically examined whether these depressive subgroups are related with cognitive dysfunction.

It is also important to highlight that most of the available evidence focusing on the effects of depression on cognition is based on in- and out-patient samples (e.g. Burt *et al*. 1995), and an important research goal therefore, is to determine whether persons with depressive disorders in the population also show cognitive impairments.

The present study investigated cognitive functioning in younger adults (20–64 years) collected from a population-based study on mental health in Stockholm County. The main aims were to examine whether: (*a*) depressed individuals differ from non-depressed healthy controls across a number of different cognitive domains including tests of episodic memory, verbal fluency, perceptual motor speed and mental flexibility; and to examine whether (*b*) cognitive performance varies as a function of depression subgroup (i.e. major depression, dysthymia, mixed anxiety-depressive disorder and minor depression).

METHOD

Subjects

All subjects in the current study were participants in the PART study, a population-based, longitudinal project on mental health, work and relations in Stockholm County. A random sample of 19 744 inhabitants aged 20–64 years who were Swedish citizens and living in Stockholm County, received a questionnaire by post. The questionnaire comprised questions regarding demography, life events, social support, working conditions, unemployment, health status, well-being and screening scales for mental disorders. The response rate was 53% ($N=$ 10 443). From the pool of questionnaire respondents, random samples of screening positive subjects (i.e. reporting many psychiatric symptoms) and screening negative subjects (i.e. reporting no psychiatric symptoms) were invited to a semi-structured psychiatric interview according to Schedules for Clinical Assessment in

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Table 1. *Demographic characteristics across study samples*

	Controls ($N=175$)	Major depression ($N=68$)	Dysthymia ($N=28$)	Mixed anxiety depressive disorder ($N=25$)	Minor depression ($N=66$)	Total depression group ($N=187$)	F/χ^2
Gender, *N* (%)							$\chi^2=14{\cdot}241$**
Male	89 (50·9)	17 (25·0)	11 (39·3)	13 (52·0)	30 (45·5)	71 (38·0)	
Female	86 (49·1)	51 (75·0)	17 (60·7)	12 (48·0)	36 (54·5)	116 (62·0)	
Education, *N* (%)							$\chi^2=7{\cdot}593$
University	52 (29·5)	21 (30·9)	7 (25·0)	6 (24·0)	14 (21·2)	48 (25·7)	
Secondary	67 (38·3)	21 (30·9)	10 (35·7)	13 (52·0)	23 (34·8)	67 (35·8)	
Primary	55 (31·4)	26 (38·2)	11 (39·3)	6 (24·0)	29 (43·9)	72 (38·5)	
Age, mean (S.D.)	43·8 (12·3)	45·7 (12·5)	43·2 (11·8)	43·2 (11·3)	42·7 (12·2)	43·9 (12·3)	$F=0{\cdot}568$

** $P<0{\cdot}01$.

Neuropsychiatry, SCAN (Wing *et al*. 1990). Here, additional information regarding met and unmet health care needs was obtained, blood samples for genetic analyses were collected, and a shorter neuropsychological test battery was administered including tests of episodic memory, verbal fluency and perceptual-motor speed. A more detailed description of the population and the methods applied has been provided elsewhere (Forsell *et al*. 2003).

Out of the total of 1093 persons interviewed, 212 fulfilled the criteria for the following mood-related diagnoses: major depression, dysthymia, bipolar syndromes, mixed anxiety-depressive disorder and minor depression according to the DSM-IV criteria. The study samples were subjected to a further selection according to a series of criteria. We excluded persons with neurological disease (i.e. epilepsy, $N=8$) and those with bipolar syndromes ($N=2$). In addition, 10 persons were excluded because of severe language difficulties, and one person was excluded because he had drunk alcohol before the testing session. Data were incomplete or missing for four persons, and hence the final study group consisted of 187 persons with the following DSM-IV diagnoses: major depression ($N=68$), dysthymia ($N=28$), mixed anxiety-depressive disorder ($N=25$) and minor depression ($N=66$). Across the depressed study samples 47 persons used psychopharmacological drugs (i.e. antidepressants, anxiolytics, sleep medication or neuroleptics).

The control group consisted of 175 persons and was formed by selecting those who were found to have no symptoms at a pathological level in the SCAN interview. None of the persons in the control group was taking psychopharmacological drugs and all were in good physical health. All participants had sufficient visual and auditory capabilities to manage the sensory demands of the study. The PART study was approved by the ethics committee at the Karolinska Institute and informed consent was obtained from each participant.

The demographic characteristics for controls and depression groups are presented in Table 1. A one-way analysis of variance (ANOVA) regarding chronological age showed that the five groups did not differ with respect to age ($F(4,357)=0{\cdot}57$, $P>0{\cdot}05$). Level of education was equal across groups ($\chi^2(8, N=361)=7{\cdot}42$, $P>0{\cdot}50$), whereas the distribution of gender differed reliably over study samples ($\chi^2(4, N=362)=14{\cdot}24$, $P<0{\cdot}01$) as shown by χ^2 tests.

Cognitive tests

Episodic memory

The episodic information to be remembered consisted of 32 words. The words belonged to eight taxonomic categories (e.g. vehicles, toys, kitchen utensils) with four subordinates each (e.g. train, doll, spoon). The items used were highly typical of their category, according to norms established by Nilsson (1973). Subjects were instructed to remember as many words as possible but were not informed about the possibility to organize the words.

Verbal fluency

The Word Association Test was used as a test of letter fluency (Benton & Hamsher, 1989). The test consists of three word-finding trials, using the letters F, A and S. The subjects were instructed to produce as many words as possible

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in 1 min, beginning with each of the target letters.

Perceptual-motor speed and mental flexibility

The trail-making test was used to assess perceptual-motor speed (Reitan, 1959; Reitan & Davidson, 1974), it was given in two parts, A and B. For both parts, subjects were presented with a white sheet of paper on which circles were distributed. In part A, the circles were numbered from 1 to 25 and participants were asked to draw lines to connect the 25 circles in the correct order (i.e. 1–2–3 … 25). In part B, the circles contained numbers from 1 to 13 and letters from A to L. The subjects were instructed to connect the consecutively numbered and alphabetically lettered circles, by alternating between the two sequences (i.e. 1–A–2–B … L–13). In both tests, subjects were requested to connect the circles as fast as they could. The first error observed was immediately pointed out by the examiner, and the subject was required to correct the error. Thereafter, the subject could continue in the proper sequence. From the second error onward the subject was not corrected, and performance time was unlimited. For both parts, accuracy scores and completion time were recorded by the examiner.

Procedure

The cognitive test battery was administered by trained co-workers at the data collection centre for the PART-study. The test battery was always administered before the SCAN-interview. All participants were tested individually in one session that took approximately 25 min to complete. The test session started with a questionnaire concerning health status. The examiner gathered information regarding sensory functioning (vision and hearing), neurological diseases, migraine, sleep apnoea, concussion of the brain, epilepsy, meningitis and tick-borne encephalitis (TBE). Information regarding drug intake and mother tongue was also collected.

This was followed by the verbal fluency test (FAS). After completion of this test, the participants were presented with the episodic memory test. The examiner read the entire list of 32 words aloud at the rate of one word every 3 s. Following presentation of the last item in the list, participants received an immediate free recall test. Participants recalled orally during a period of 3 min. The examiner recorded each subject's unique recall order verbatim. This task was followed by a cued recall test. In this test, the eight taxonomic names were provided as retrieval cues. The examiner read each category name aloud, one by one, and subjects were asked to recall as many words as possible belonging to each category. Twenty seconds were allowed per category and the responses were recorded by the examiner. Finally the tests of perceptual-motor speed (TMT-A) and mental flexibility (TMT-B) were administered.

Four different test orders were created and approximately 250 subjects were randomly assigned to each presentation order. In addition, for the episodic memory task, four different and scrambled orders of the 32 nouns were prepared, yielding four unique word lists, which were counterbalanced across test order. The category names provided in the cued recall test were presented in two different orders and counterbalanced across test order as well.

RESULTS

In order to examine whether test order affected cognitive performance, a 4 (Test Order) $\times 5$ (Study group) factorial ANOVA was performed on each cognitive task. None of these analyses revealed any main effect of test order or any reliable study group by test order interaction and thus data were collapsed across this variable (Fs < 1).

The mean performance across cognitive tasks and study groups is summarized in Table 2. In the first set of analyses we compared the total group of depressed individuals ($N = 187$) with healthy controls, in order to determine whether depressive illness in general exerts negative effects on the specific cognitive task. This was followed by separate ANCOVAs for each of the depressive subgroups. Finally, to examine whether psychopharmacological drug use affected the obtained results, we divided the total group of depressed persons into one group with medication and one group without medication. Gender was entered as covariate in all analyses.

Effects of depression on cognitive function

Episodic memory

The ANCOVA performed on episodic memory performance showed that persons affected by

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Table 2. *Cognitive performance across study samples*

Cognitive task	Controls ($N=175$)		Major depression ($N=68$)		Dysthymia ($N=28$)		Mixed anxiety depressive disorder ($N=25$)		Minor depression ($N=66$)		Total depression group ($N=187$)	
	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Episodic memory												
Free recall	14·1	4·7	12·9*	4·7	13·3	4·8	12·2+	4·6	13·6	5·5	13·1*	5·0
Cued recall	16·6	4·9	14·6**	5·0	15·4	5·1	13·9*	4·8	16·2	4·9	15·2**	5·0
Verbal fluency												
F	15·8	4·7	15·0	6·3	15·9	4·9	16·6	6·4	16·4	5·4	15·9	5·8
A	12·8	4·9	13·0	5·7	11·9	4·7	12·7	5·1	13·2	5·2	12·8	5·3
S	16·4	5·0	16·0	6·1	15·7	4·7	16·6	6·1	16·8	5·6	16·3	5·7
Total	45·0	12·8	43·9	16·8	43·5	13·2	46·0	15·7	46·5	14·4	45·1	15·3
Motor/speed												
TMT A†	23·9	0·9	23·9	0·7	24·0	0·0	24·0	0·0	23·9	0·4	23·9	0·5
TMT A (s)	38·1	15·1	41·6	15·2	41·9	18·2	40·6	10·7	39·1	15·3	40·6	15·2
TMT B†	23·5	2·4	23·0	4·1	23·6	1·6	23·8	0·8	23·9	1·1	23·5	2·6
TMT B (s)	76·3	32·7	85·7	40·8	96·2**	45·1	82·1	22·8	79·3	38·0	84·5*	38·7

† Number of correctly connected circles (maximum = 24).
* $P<0·05$; ** $P<0·01$.
+ $P<0·1$.

depression remembered fewer words in both free ($F(1,358)=6·50$, mean squared error (MSE) = 22·22, $P<0·05$) and cued ($F(1,358)=9·66$, MSE = 24·03, $P<0·01$) recall as compared with the healthy controls. Also, persons with major depression performed more poorly than non-depressed controls in both free ($F(1,239)=6·65$, MSE = 21·07, $P<0·05$) and cued ($F(1,239)=10·39$, MSE = 24·10, $P<0·01$) recall. Persons suffering from mixed anxiety-depressive disorder had a tendency to perform less well than non-depressed controls in free recall ($P<0·06$), and recalled fewer words than controls in the cued ($F(1,196)=6·65$, MSE = 23·87, $P<0·05$) recall task. Dysthymia and minor depression did not affect episodic memory performance (Fs < 1).

Verbal fluency

There were no reliable differences between the total group of depressed individuals or between any of the depression subgroups and the non-depressed control group on the measures of verbal fluency (Fs < 1).

Perceptual-motor speed and mental flexibility

The ANCOVA performed on the TMT-A and TMT-B accuracy scores and the TMT-A completion time revealed no significant differences between the total group of depressed individuals or between any of the depression subgroups and the healthy controls (Fs < 1). The analyses of TMT-B completion time showed that the total group of persons with depression used significantly more time to complete the TMT-B ($F(1,352)=4·73$, MSE = 1290·60, $P<0·05$) than non-depressed controls. Likewise, the ANCOVA on dysthymia showed that these persons used reliably more time to complete the TMT-B ($F(1,198)=7·90$, MSE = 1204·18, $P<0·01$) than controls. Major depression, mixed anxiety-depressive disorder and minor depression did not affect the completion time performance on the TMT-B (Fs < 1).

Effects of psychopharmacological drug use on cognitive function

Finally, in order to investigate whether psychopharmacological drug use affected the obtained results, we performed a series of additional one-way ANCOVAs, with gender as covariate. In the set of analyses we divided the total group of

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depressed individuals into those using psychopharmacological drugs ($N=47$) and those individuals without drugs ($N=140$) and compared these groups with the healthy controls. The results indicated that both medicated (mean = 12·55, S.D. = 5·14) and unmedicated depressed individuals (mean = 13·29, S.D. = 4·94) performed less well than controls (mean = 14·08, S.D. = 4·70) in free recall, although the two depressed groups did not differ ($F(2,357)=3·83$, MSE = 22·21, $P<0·05$). In cued recall, healthy controls remembered more words (mean = 16·59, S.D. = 4·92) than the medicated (mean = 14·79, S.D. = 4·84) and unmedicated (mean = 15·31, S.D. = 5·06) depressed groups, although the latter two groups did not differ, ($F(2,357)=5·09$, MSE = 24·06, $P<0·01$). The ANCOVA on TMT-B completion time revealed a main effect of group ($F(2, 351)=3·39$, MSE = 1286·83, $P<0·05$) indicating that medicated depressed persons were slower (mean = 91·16, S.D. = 36·20) in completing the TMT-B than unmedicated individuals (mean = 82·36, S.D. = 39·33), who in turn were slower than the group of healthy controls (mean = 76·17, S.D. = 32·67).

DISCUSSION

The main purpose of this research was to examine the effects of depressive disorders on cognitive functioning. This was accomplished by comparing population-based samples of adults with a depression diagnosis with healthy controls across a number of cognitive tasks. In agreement with earlier findings (e.g. Burt *et al.* 1995; Veiel, 1997), the results of the present study indicate that depressive disorders in general are associated with deficits in both free and cued recall of episodic information. Also, persons diagnosed with a depressive disorder exhibited impairments in tasks tapping mental flexibility replicating earlier findings (Austin *et al.* 1992). In contrast, depressed individuals showed no impairments in perceptual speed and verbal fluency. Of more interest, however, was the observation that the pattern of these impairments varied as a function of depression subgroup: major depression and mixed anxiety-depressive disorder were related to significant impairments in the memory tests, whereas individuals with dysthymia showed significant impairments in speed when solving the TMT-B test. Minor depression did not differ from healthy controls in any cognitive task. However, it is worth noting that these results should be treated with caution, as the small sample size of particularly the dysthymia and mixed anxiety-depressive subgroups may have limited our ability to obtain significant results.

As noted, previous research has yielded inconclusive results regarding the nature of the episodic memory deficits observed in depression. For instance, some investigators report that impairments primarily reflect a general retrieval deficit (Massman *et al.* 1992; Ilsley *et al.* 1995), whereas others report that depressed individuals perform selectively worse in recognition tasks, suggesting that the memory deficit observed in depression is related to encoding impairments (Kindermann & Brown, 1997). The present free-recall data demonstrated that the total sample of depressed individuals, the major depression and mixed anxiety-depressive disorder groups, exhibited significant memory dysfunction. However, when taxonomic category cues were provided as retrieval support for recall of organizable words, all groups showed an improvement in performance relative to free recall. Importantly, the magnitude of the recall improvement was equally large across depression groups and controls. This pattern of results suggests that the ability to utilize retrieval cues is well preserved in depression. Also, the finding that the depressed individuals did not selectively improve recall performance following retrieval support suggests that access problems alone may not account for the episodic memory impairment observed in depression. Rather, our results indicate that encoding problems play an important role in depression-related memory deficits (e.g. Kindermann & Brown, 1997).

Mixed anxiety-depressive disorder and minor depression have been included as two new research diagnoses in the DSM-IV and it is not yet known whether these diagnoses are associated with cognitive impairments. The present findings suggest that persons diagnosed with mixed anxiety-depressive disorder show substantial episodic memory dysfunction, comparable to that observed in major depression. This outcome supports earlier clinical observations of considerable functional impairments in this depressive subgroup and that the mixed anxiety-depressive disorder merits inclusion in the DSM

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as a syndrome (Barlow & Campbell, 2000). This issue is further elaborated by Tyrer (2001) who argues that mixed anxiety-depressive disorder should be conceptualized as a syndrome, cothymia, and not as a subsyndromal disorder as defined in the DSM-IV. It is also interesting to note that the typical sex distribution of higher prevalence rates among females than males in depressive disorders was absent in this subgroup. Here, equal numbers of men ($N=13$) and women ($N=12$) were diagnosed with mixed anxiety-depressive disorder. However, the numbers are small and prevalence rates need to be confirmed in future epidemiological work.

In contrast with these findings, the second research diagnosis, minor depression, showed no influence on cognitive functioning. Across tasks, individuals diagnosed with minor depression performed equally as well as controls. However, it is important to note that this outcome may have been related to the fact that the cognitive tests used in present study were not sensitive enough to detect potential cognitive dysfunction in minor depression.

Both accuracy scores and completion time for the TMT-A were of equal magnitude across all studied groups. To the extent that the TMT-A taps perceptual-motor speed, attention and coordination, the present results suggest that these abilities were unaffected by depressive disorder thus corroborating some earlier findings (cf. Grant *et al.* 2001). However, in the TMT-B, a different pattern of results was observed. Compared with the TMT-A, the TMT-B poses further demands on mental flexibility (i.e. set shifting) in managing more than one stimulus category at a time and in shifting the course of an ongoing activity. It involves semantic and working memory functions (Baddeley, 1992; Lezak, 1995). Replicating previous findings (Austin *et al.* 1992; Ravnkilde *et al.* 2002), the total group of depressed individuals was slower in completing the TMT-B, although accuracy scores were unaffected. Interestingly, the analysis across the different subgroups revealed that only persons diagnosed with dysthymia demonstrated reliable increments in completion time, whereas individuals diagnosed with major depression, mixed anxiety-depressive disorder and minor depression used similar amounts of time to healthy controls to complete the test. As noted earlier, evidence is sparse regarding the relationship between dysthymia and cognitive functioning. The present results suggest that dysthymia is unrelated to episodic memory dysfunction, which is in congruence with an earlier report (Yee & Miller, 1994). However, when dysthymic persons were required to shift conceptually between numerical and alphabetical order, severe impairments were observed, suggesting that the disorder is associated with pronounced difficulties in mental set shifting (Marshall *et al.* 1997).

There were no effects of depressive disorder on verbal fluency proficiency. Irrespective of depressive disorder, individuals generated as many words as controls. To the extent that verbal letter fluency taps cognitive operations such as lexical retrieval and semantic memory (Lezak, 1995) the present findings suggest that these functions are spared among depressed individuals in the population, replicating earlier findings based on in- and out-patient samples (Wolfe *et al.* 1987; Ilsley *et al.* 1995).

Previous research indicates that psychotropic medication intake may influence cognitive performance (e.g. Amado-Boccara *et al.* 1995). In the present sample, 47 of the 187 depressed persons used psychotropic drugs of various types (i.e. antidepressants, anxiolytics, sleep medication and neuroleptics). In order to minimize the potential of drug use confounding the results, comparisons were made between patients with and without medication. These results indicated that psychopharmacological drug use was unrelated to episodic memory performance, whereas mental flexibility, as tapped by the TMT-B, was significantly affected. This outcome suggests that some cognitive functions may be more susceptible to the negative repercussions of drug use and this needs further exploration in future research.

Before concluding it is important to highlight the high dropout rate in this study. By Swedish standards the participation rate of 53% in this study is low. In other studies on health outcomes in Stockholm County aimed at random population samples the expected participation would be approximately 60–65%. We regard this difference as being due to the subject of our investigation since many of those approached refused participation for this reason. The questionnaire took about one hour to complete and it is likely that persons severely affected by depression

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would often decline participation due to the nature of the disorder. The persons who answered the questionnaire, and whom were selected for interview, had a higher income and a higher education, were more often female, and were more often born in the Nordic countries than those who did not participate. Although the depression diagnoses were based on established DSM-IV criteria and the controls did not suffer from any psychiatric disorder the results might have been affected by the dropout rate. It is most likely that the associations we found are an underestimation of the true effects of depression on cognition in the population.

The strength of the study is that it is population-based, involves a large number of untreated persons and replicates studies showing that the majority of people with psychiatric disorders receive no professional treatment (Shapiro *et al*. 1984; Kessler *et al*. 1994). The fact that depressed persons who have not sought care have a cognitive dysfunction may have both human (Bremner *et al*. 2000) and economic (Bushnell & Bowie, 1995) repercussions for society.

To summarize, the current study clarifies previous uncertainties by showing that cognitive dysfunction varies as a function of depressive disorder. Specifically, major depression and mixed anxiety-depressive disorder were associated with greater cognitive impairments than dysthymia, whereas minor depression proved to be unrelated to cognitive performance.

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