



Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction

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Abstract

Most of the available evidence on neuropsychological functioning in anxiety disorders is based on clinical samples, investigating persons affected by obsessive–compulsive disorder. Knowledge is sparse regarding cognitive functions in other types of anxiety disorders.

The aim of this study was to examine whether persons diagnosed with an anxiety disorder show neuropsychological impairments relative to healthy controls in tasks tapping episodic memory, verbal fluency, psychomotor speed, and executive functioning. Population-based samples comprising individuals affected by panic disorder with and without agoraphobia or agoraphobia ($n = 33$), social phobia ($n = 32$), generalised anxiety disorder ($n = 7$), obsessive–compulsive disorder ($n = 16$), and specific phobia ($n = 24$) were compared with healthy controls ($n = 175$) in test performance. Overall, the total anxiety disorder group exhibited significant impairments in episodic memory and executive functioning. Separate analyses on the respective anxiety subgroup indicated that panic disorder with and without agoraphobia, and obsessive–compulsive disorder were related to impairments in both episodic memory and executive functioning. In addition, social phobia was associated with episodic memory dysfunction. Verbal fluency and psychomotor speed were not affected by anxiety. Specific phobia and generalised anxiety disorder did not affect neuropsychological functioning

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1. Introduction

Anxiety disorders are common with the prevalence varying between 2% and 5% in population-based studies (APA, 1994). During the past decade, an increasing number of studies addressing neuropsychological functioning in anxiety disorders have been published. However, the majority of this research has focused on the less

prevalent obsessive–compulsive disorder (OCD), whereas less attention has been paid to the other *diagnostic and statistical manual of mental disorders* (DSM-IV) (APA, 1994) defined anxiety disorders, such as panic disorder (PD) and social phobia (SP).

Research focusing on cognitive functioning in OCD has demonstrated episodic memory impairments for both non-verbal (Dirson et al., 1995; Savage et al., 1996, 1999) and verbal information (Savage et al., 2000; Zitterl et al., 2001). Also, reliable performance deficits have been obtained in tasks tapping executive functioning (Martinot et al., 1990; Purcell et al., 1998a,b; Veale et al., 1996; Head et al., 1989), although some re-

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search has reported normal executive functioning among persons affected by OCD (Boone et al., 1991; Christensen et al., 1992; Zielinski et al., 1991).

As noted above, only a few studies have addressed neuropsychological functioning in other DSM-IV defined anxiety disorders, and available evidence presents a mixed pattern of results. Regarding episodic memory, Lucas et al. (1991) reported that persons with PD exhibit reliable performance impairments for both visual and verbal information. In a similar vein, Asmundson et al. (1995) reported that both PD and SP were associated with significant recall deficits for verbal information, although individuals with PD performed as well as controls for visually based information. In addition to these findings, some research has established significant executive dysfunction in PD (Cohen et al., 1996). In contrast to these observations, other investigators found no evidence of an episodic memory dysfunction in PD, and this was true for both verbal and visual stimuli (Gladsjo et al., 1998). Also, Purcell et al. (1998a,b) compared groups of OCD patients, PD, and major depression with healthy controls across a number of cognitive domains. The results indicated that only OCD patients exhibited impairments in executive functioning, attention, and episodic memory, whereas the PD and major depression groups performed as well as the healthy controls. It is highly likely that these inconsistent findings are a function of methodological differences between the studies regarding selection of participants, patient status, materials used in the memory tasks, and memory performance assessment (e.g. recall vs. recognition).

Evidence from neurobiological studies suggests that brain structures subserving episodic memory functioning are affected in anxiety disorders. For example, positron emission tomography studies demonstrate abnormal blood flow in medial temporal lobe including amygdala and hippocampus in symptom provoked SP patients (Tillfors et al., 2001), and an involvement of the hippocampal and parahippocampal areas in PD (Bisaga et al., 1998). In addition, magnetic resonance imaging study reported abnormalities in temporal lobe structures in PD subjects (Vythilingam et al., 2000). Further, neuroimaging studies in patients with OCD suggest a frontal-subcortical circuit involvement (Kwon et al., 2003).

The main objective of this research was to extend our knowledge on neuropsychological functioning in persons affected by an anxiety disorder in a population. In contrast to most previous research addressing this topic, the present investigation is based on population-based samples of persons meeting the DSM-IV criteria for an anxiety disorder. We compared different anxiety subgroups with non-anxious healthy controls across a number of different tasks tapping various cognitive domains including episodic memory for verbal information, verbal fluency, psychomotor speed, and executive

functioning. In this way, we were able to examine cognitive functioning in population-based samples of persons suffering from an anxiety disorder, and to determine whether cognitive performance varied as a function of anxiety subgroup.

2. Methods

The participants were selected from the PART-study, an ongoing population-based, longitudinal project on mental health, work and relations in Stockholm County. In short, an extensive questionnaire comprising questions regarding demography, life events, social support, working conditions, unemployment, health status, well-being, and screening scales for mental disorders were mailed to a random sample of 19,742 inhabitants aged 20–64 years, Swedish citizens, and registered in Stockholm County. In total, 10,441 persons responded (53%). From the pool of questionnaire respondents, random samples of persons reporting many psychiatric symptoms ($n = 884$), and persons reporting no psychiatric symptoms ($n = 209$) were interviewed using the Schedules for Clinical Assessments in Neuropsychiatry (SCAN) (Wing et al., 1990), which gives DSM-IV diagnoses. Clinically experienced psychiatrists and psychologists conducted the interviews. In connection with the SCAN interview a comprehensive neuropsychological test battery was administered including tests of episodic memory, verbal fluency, perceptual-motor speed, and mental flexibility.

Of the 1093 interviewees, 136 fulfilled the criteria of at least one anxiety disorder according to DSM-IV. Persons affected by posttraumatic stress disorder ($n = 4$), and anxiety due to somatic disease ($n = 1$) was excluded due to low numbers. Of the remaining 131 persons, 19 were excluded for harming co-morbid neurological diseases ($n = 6$), psychotic episodes ($n = 2$), intoxication ($n = 2$), bad eyesight ($n = 3$), obvious language problems ($n = 3$), not been sleeping the night before the test session ($n = 2$) and missing cognitive data ($n = 1$). Thus, the final study sample consisted of 112 individuals fulfilling the criteria for one or more DSM-IV anxiety disorders. Due to low numbers the persons were grouped together according to the clinical impressions. The first group ($n = 33$) was formed by individuals suffering from PD with and without agoraphobia ($n = 30$), or agoraphobia only ($n = 3$). Persons suffering from SP that did not fulfil the criteria for some of the disorders in the first group formed the second group ($n = 32$). The third group ($n = 7$) was comprised by persons diagnosed with generalized anxiety disorder (GAD) who met no criteria for belonging to any of the groups above. The fourth group ($n = 16$) was formed of those with OCD who do not belong to groups above. Finally, the fifth group ($n = 24$) consisted of those with specific phobia only. It

Table 1
Demographic characteristics across study samples

	Controls (<i>n</i> = 175)	Panic disorder with and without agoraphobia (<i>n</i> = 33)	Social phobia (<i>n</i> = 32)	Generalised anxiety disorder (<i>n</i> = 7)	Obsessive-compulsive disorder (<i>n</i> = 16)	Specific phobia (<i>n</i> = 24)	The total group of anxiety disorders (<i>n</i> = 112)	<i>F</i> ration, χ^2	<i>p</i> value
<i>Gender</i>	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	$\chi^2 = 16.180$	0.006
Male	89 (50.9)	7 (21.8)	10 (31.3)	2 (28.6)	4 (25.0)	8 (33.3)	31 (27.7)		
Female	86 (49.1)	26 (78.8)	22 (68.8)	5 (71.4)	12 (75.0)	16 (66.7)	81 (72.3)		
<i>Education</i>	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	$\chi^2 = 7.707$	0.657
University	52 (29.9)	7 (21.2)	5 (15.6)	2 (28.6)	3 (18.8)	7 (29.2)	24 (21.4)		
Secondary	67 (38.5)	12 (36.4)	14 (43.8)	2 (28.6)	4 (25.0)	8 (33.3)	40 (35.7)		
Primary	55 (31.6)	14 (42.4)	13 (40.6)	3 (42.9)	9 (56.3)	9 (37.5)	48 (42.9)		
<i>Age</i>	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>F</i> = 2.282	0.047
	43.9 (12.3)	43.6 (13.0)	38.0 (12.3)	41.7 (11.8)	35.7 (13.7)	43.2 (11.1)	40.7 (12.6)		

is worth noting that 49 persons diagnosed with an anxiety disorder also fulfilled the criteria for other psychiatric diagnoses: 34 of the participants had a concomitant depressive disorder, and 15 had alcohol dependence/abuse. Regarding psychopharmacological drug use, 17 anxiety persons used antidepressive drugs, 6 used anxiolytics, 7 were taking sleeping medication, and 2 persons used neuroleptics.

The control group consisted of 175 persons. None of the persons in the control group fulfilled the criteria for a psychiatric disorder, or were 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. Three control persons were excluded in the TMT-B task due to completion failure. The PART study was approved by the ethics committee at Karolinska Institute and informed consent was obtained from each participant.

The demographic characteristics for the control group and the anxiety disorder samples are presented in Table 1. The six study groups differed significantly on chronological age, ($F(5,281) = 2.28, p = .047$) and gender ($\chi^2(5, n = 287) = 16.18, p = .006$), but not on level of education ($\chi^2(10, n = 286) = 7.71, p = .657$).

2.1. Neuropsychological tests

2.1.1. Episodic memory

The episodic information to-be-remembered consisted of 32 neutral 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.

2.1.2. Verbal fluency

The Word Association Test was used as a test of letter fluency (Benton and 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 in one minute, beginning with each of the target letters.

2.1.3. Perceptual-motor speed and executive functioning

The trail-making test (TMT) was used to assess perceptual-motor speed and executive function (Reitan, 1959; Reitan and Davidson, 1974), and 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 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 told to connect the circles as fast as they could. The examiner immediately pointed out the first error observed, 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, the examiner recorded accuracy scores and completion time.

2.2. Procedure

Trained co-workers at the data collection centre administered the neuropsychological test battery. Participants were tested individually in one session that took approximately 25 min to complete. The test battery was always administered before the SCAN-interview. 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 a rate of 3 s/word. Immediately after presentation of the last word in the list, participants were asked to free recall orally as many words as possible from the list. The examiner recorded each subjects 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 per category were allowed and the examiner recorded the responses. Finally the perceptual-motor speed and executive function tests (TMT-A and 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.

To examine whether test order affected cognitive performance, a 4 (Test order) \times 6 (Study group) factorial ANOVA was performed on each cognitive task. There were no effects of test order or study group by test order

interaction, thus the data were collapsed across this variable ($F_s < 1$).

The mean performance across cognitive tasks and study groups is presented in Table 2. The main data analyses were made in two steps. First, we examined the total group of persons affected by an anxiety disorder ($n = 112$) and compared these with healthy controls in order to determine whether anxiety in general exhibit negative effects on cognitive performance. This was followed by five separate analyses of covariance (ANCOVAs) for each of the anxiety subgroups.

Also, in order to examine whether concomitant depression (Burt et al., 1995; Veijel, 1997) or alcohol dependence/abuse (Nixon et al., 1987; Delin and Lee, 1992) affected the obtained results, additional analyses were performed. In the first set of these analyses we excluded persons meeting the DSM-IV criteria for depression, and in the second set we excluded persons with alcohol dependence/abuse according to the DSM-IV. Moreover, in order to investigate whether psychopharmacological drug use affected the obtained results we divided the total group of anxiety persons in psychopharmacological drug users and non-users and compared these two groups with the healthy controls. For all analyses, age and gender were entered as covariates.

3. Results

3.1. Effects of anxiety on cognitive function

3.1.1. Episodic memory

The ANCOVA performed on episodic recall showed that persons affected by an anxiety disorder remembered fewer words in both free ($F(1,282) = 11.43, p = .001$) and cued ($F(1,282) = 10.59, p = .001$) recall as compared with healthy controls. Separate analyses of the respective anxiety diagnosis showed that persons affected by PD with and without agoraphobia, or agoraphobia, recalled significantly fewer words as compared with healthy controls in both free ($F(1,203) = 8.66, p = .004$) and cued ($F(1,203) = 5.31, p = .022$) recall. Also, persons with SP performed worse than controls in both free ($F(1,202) = 7.75, p = .006$) and cued ($F(1,202) = 7.30, p = .007$) recall tasks. Persons suffering from OCD had a tendency to remember fewer words than controls in free recall ($F(1,186) = 3.29, p = .071$) and remembered significantly fewer words in cued recall ($F(1,186) = 3.98, p = .048$). Specific phobia and GAD did not affect episodic memory performance ($F_s < 1$).

3.1.2. Verbal fluency

The ANCOVA performed on verbal fluency showed that individuals affected by SP generated reliable fewer words beginning with letter F ($F(1,203) = 6.57,$

Table 2
Neuropsychological performance across study samples

Cognitive task	Controls (<i>n</i> = 175)		Panic disorder with and without agoraphobia (<i>n</i> = 33)		Social phobia (<i>n</i> = 32)		Generalised anxiety disorder (<i>n</i> = 7)		Obsessive– compulsive disorder (<i>n</i> = 16)		Specific phobia (<i>n</i> = 24)		The total group of anxiety disorders (<i>n</i> = 112)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Episodic memory</i>														
Free recall	14.1	4.7	12.2**	4.8	12.6**	5.6	12.7	1.7	13.1	3.4	14.1	5.5	12.9***	4.9
Cued recall	16.6	4.9	14.9*	4.8	14.7**	5.7	14.9	2.5	14.8*	4.9	16.0	5.6	15.1***	5.1
<i>Verbal fluency</i>														
F	15.8	4.7	15.7	5.2	13.4*	5.4	16.3	4.4	13.9	4.2	17.3	6.6	15.1	5.5
A	12.8	4.9	13.2	4.7	11.7	5.2	14.1	4.1	12.0	4.0	13.9	4.9	12.8	4.7
S	16.4	5.0	17.1	5.5	15.7	6.3	16.6	4.2	15.	3.8	17.8	7.3	16.6	5.9
Total	45.0	12.8	46.0	13.3	40.8	15.2	47.0	9.7	41.5	10.6	48.8	17.0	44.5	14.4
<i>Motor/speed</i>														
TMT A ^a	23.9	0.9	24.0	0.0	24.0	0.0	23.9	2	23.9	0.3	24.0	0.0	24.0	0.1
TMT A (s)	38.0	15.1	39.2	14.0	38.0	11.2	46.0	15.5	40.7	11.1	35.5	11.2	38.7	12.4
TMT Ba	23.5	2.4	22.6	4.0	23.6	2.1	24.0	0.0	24.0	0.0	23.7	1.7	23.4	2.6
TMT B (s)	73.8	32.7	86.1*	35.4	75.5	33.2	78.3	31.4	83.9**	22.6	80.6	28.8	81.1**	31.4

Statistically significant results are displayed in a bold format.

^a Number of correctly connected circles (max 24).

* *p* < .05.

** *p* < .01.

*** *p* < .001.

$p = .011$) than controls. Also, persons affected by SP had a tendency to generate fewer words totally in FAS ($F(1,203) = 2.75, p = .099$). Verbal fluency proficiency was of equal size in the total group of persons affected by anxiety, and in PD and GAD groups, and in specific phobias as compared with healthy controls ($F_s < 1$).

3.1.3. Perceptual-motor speed and executive functioning

The ANCOVA conducted on TMT-A and TMT-B accuracy scores and TMT-A completion time revealed no significant differences between the total group of anxiety persons or for any of the anxiety subgroups and controls ($F_s < 1$). The analyses on TMT-B completion time showed that persons affected by an anxiety disorder needed significantly more time to complete the TMT-B ($F(1,277) = 8.38, p = .004$) as compared with healthy controls. Likewise, persons affected by PD with and without agoraphobia, or agoraphobia needed more time to complete the TMT-B ($F(1,199) = 5.28, p = .023$) than controls. In addition, persons diagnosed with OCD were slower than controls in their performance on TMT-B completion time ($F(1,182) = 5.74, p = .018$). Persons diagnosed with SP, GAD or specific phobia showed no reliable effects on TMT-B completion time ($F_s < 1$) as compared with healthy controls.

In addition, we performed additional multivariate analyses (MANCOVAs), with age and gender as covariates and adjustment for multiple testing (i.e. Bonferroni correction). These analyses showed the same pattern of findings as that obtained with the ANCOVA procedure.

3.2. Effects of co-morbid depression, alcohol dependence/abuse, and psychopharmacological drug use on cognitive functions

In order to examine whether a concomitant depressive disorder ($n = 34$) or alcohol dependence/abuse ($n = 15$) affected the obtained results, we excluded the participants meeting these criteria and subsequently performed a new series of ANCOVAs. These analyses revealed an identical pattern of results indicating reliable episodic memory deficits in both the total group of anxiety persons and anxiety subgroups. However, exclusion of participants with alcohol dependency/abuse resulted in that the significant effects of PD and OCD on TMT-B completion time disappeared suggesting that obtained results on TMT-B may be related to alcohol dependence/abuse.

In the final set of analyses, we investigated whether psychopharmacological drug use affected the obtained results. Here, the total anxiety group was divided into those using medication ($n = 23$), not using medication ($n = 89$), and were then compared with the group of healthy controls. The ANCOVA on free recall showed a significant group effect ($F(2,281) = 6.42, p = .002$). Post hoc analyses revealed that both medicated

(mean = 11.78, SD = 4.3) and unmedicated (mean = 13.16, SD = 5.0) anxiety persons recalled fewer words than controls (mean = 14.08, SD = 4.7), although the two anxiety groups did not differ. In a similar vein, the ANCOVA on cued recall performance showed a reliable group effect ($F(2,281) = 5.36, p = .005$). Post hoc analyses revealed that both medicated (mean = 14.52, SD = 5.0) and unmedicated (mean = 15.20, SD = 5.1) anxiety persons performed equally well in the cued recall task. Unmedicated persons recalled significantly fewer words than the healthy controls (mean = 16.59, SD = 4.9) whereas medicated persons had a non-significant tendency to remember fewer words than the controls. Likewise, the ANCOVA on TMT-B completion time revealed a significant group effect ($F(2,276) = 4.37, p = .013$). The subsequent post hoc analyses revealed that medicated anxiety persons (mean = 88.13, SD = 33.5) used more time to complete the TMT-B as compared with unmedicated anxiety persons (mean = 79.25, SD = 30.7) and the controls (mean = 73.75, SD = 27.3), whereas the two later groups did not differ.

4. Discussion

The main objective of this study was to examine the effects of anxiety disorders on cognitive functioning. This was accomplished by comparing individuals diagnosed with an anxiety disorder according to DSM-IV criteria with a group of non-anxious controls across a number of tasks tapping different cognitive domains. In contrast to most previous research, the participants in this work were collected from a population-based study including many non-treated anxious persons. In general, the main results indicate that anxiety disorders are associated with episodic memory dysfunction. The pattern of this impairment was similar for all anxiety subgroups, with reliable deficits in both free and cued recall of episodic information. In addition, our results suggest executive dysfunction (TMT-B) in persons diagnosed with PD, and OCD. In contrast, anxiety did not affect verbal fluency and psychomotor speed (TMT-A). Persons suffering from a specific phobia and GAD showed no cognitive dysfunction.

As noted above, a number of studies have addressed memory functioning in OCD, whereas evidence is sparse regarding episodic memory functioning in other anxiety disorders. Previous research on OCD has demonstrated strong evidence of episodic memory deficits for non-verbal information, although some recent findings also suggest episodic memory deficits for verbal information in OCD, replicating the present findings (Savage et al., 2000; Zitterl et al., 2001). More interesting, however, was the finding that the group composed of PD individuals and the SP group showed the largest episodic mem-

ory impairments corroborating some earlier research (Lucas et al., 1991; Asmundson et al., 1995), although others have failed to establish memory deficits in PD (Gladsjo et al., 1998; Purcell et al., 1998a,b).

It is important to note that a closer inspection of the free and cued recall data across study samples suggests that the ability to utilize semantic cues in anxiety is intact. The magnitude of the gain from free to cued recall was of equal size across anxiety subgroups and controls. This pattern of results suggests that the episodic memory impairment primarily is related to encoding rather than to retrieval problems. Also, the observation that anxiety persons did not selectively improve recall performance when retrieval cues were provided further suggests that the episodic memory deficits occur during acquisition rather than at retrieval.

Previous findings concerning TMT performance in anxiety disorders display a mixed pattern of results. For example, two early studies (Martinot et al., 1990; Aronowitz et al., 1994) demonstrated that OCD patients were significantly slower than controls, whereas Cohen et al. (1996) reported normal completion time in OCD, but significant dysfunction in SP. In contrast to that finding, there are reports of intact psychomotor speed and executive functioning (TMT-A and -B) in patients diagnosed with SP and PD (Asmundson et al., 1995; Gladsjo et al., 1998). To the extent that the TMT-A taps psychomotor-speed, attention, and co-ordination, the present findings suggest that these abilities are intact in anxiety disorders. Executive functioning as measured by the TMT-B was impaired in the total group of anxiety disorders, in the PD group, and in OCD. Research has indicated that anxiety is related to divided attention deficits (e.g., Mor et al., 2002). Given that divided attention is an important prerequisite for completion of the TMT-B, impairments in divided attention capacity may contribute to the observed performance decrement in TMT-B. However, when comorbidity was controlled for in both the OCD and PD group, the effects disappeared, suggesting that concomitant alcohol abuse mediated the observed anxiety-related deficits in executive functioning.

Overall, verbal fluency was not affected by anxiety disorders, although the total FAS-score proved marginally significant in SP. Thus, the present findings suggest preserved lexical retrieval in anxiety disorders in the population (Boone et al., 1991; Zielinski et al., 1991).

As noted above, GAD was unrelated to cognitive dysfunction. However, it is important to note that relatively few persons were diagnosed with GAD in the present population ($n = 7$). Small sample sizes affect the probability to obtain reliable group differences. Thus, future research is required to assess the replicability of this finding.

Specific phobias frequently co-occur with another anxiety diagnosis and are associated with less distress and less interference in everyday life than the comorbid

main diagnosis (APA, 1994). Not surprisingly, since individuals with only a specific phobia diagnosis were included in the study sample, the present finding indicated no impairments in any of the cognitive tasks. Across tasks, individuals diagnosed with specific phobia performed equally well as controls.

Comorbidity among psychiatric disorders is highly prevalent. Over one-half of patients in psychiatric treatment receive more than one diagnosis (Wolf et al., 1988). In the present study samples, 49 of the totally 112 anxiety individuals, had a DSM-IV defined comorbid depressive disorder or alcohol dependence/abuse. It is well established that depression exhibits a negative effect on episodic memory functioning (Burt et al., 1995; Veijel, 1997). For example, in our previous work, we demonstrated episodic memory dysfunction in persons with depressive disorders collected from a population-based sample (Airaksinen et al., 2004). An important observation in the present study was that exclusion of participants suffering from a concomitant depressive disorder, alcohol dependence/abuse, or were using psychopharmacological drugs did not affect the overall pattern of results on episodic memory. This outcome strongly suggests that anxiety disorders exhibit a unique negative influence on episodic memory functioning.

It is important to note the high dropout rate in this study; only 53% of the randomly selected individuals participated. We regard the low participation rate 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 possible that persons severely affected by psychiatric disorders would often refuse participation due to the nature of the disorder. The individuals who answered the questionnaire and who were selected to the interview had a higher education, higher income, were more often female and born in the Nordic countries than those who did not participate. It is highly likely that the present findings have been affected by the high dropout rate, such that the obtained effects of anxiety on cognition are an underestimation of the true effects of anxiety on cognition in the population.

In summary, this study extends previous research by indicating that anxiety disorders are associated with reliable impairments in episodic memory and executive functions in population-based samples.

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References

- Airaksinen E, Larsson M, Lundberg I, Forsell Y. Cognitive functions in depressive disorders: evidence from the population-based study. *Psychological Medicine* 2004;34:83–91.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Washington, DC: American Psychiatric Association; 1994.
- Aronowitz BR, Hollander E, Decaria C, Cohen L, Saoud JB, Stein D, et al. Neuropsychology of obsessive compulsive disorder. Preliminary findings. *Neuropsychiatry Neuropsychology and Behavioral Neurology* 1994;7:81–6.
- Asmundson GJG, Stein MB, Larsen DK, Walker JR. Neurocognitive functions in panic disorder and social phobia patients. *Anxiety* 1995;1:201–7.
- Benton L, Hamsher KD. Multilingual aphasia examination. Iowa City, Iowa: AJA Associates; 1989.
- Bisaga A, Katz JL, Antonini A, Wright CE, Margouloff C, Gorman JM, et al. Cerebral glucose metabolism in women with panic disorder. *American Journal of Psychiatry* 1998;155:1178–83.
- Boone KB, Ananth J, Philpott L, Kaur A, Djenderedjian A. Neuropsychological characteristics of nondepressed adults with obsessive–compulsive disorder. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology* 1991;4:96–109.
- Burt DB, Zembor MJ, Niederehe G. Depression and memory impairment: a meta analysis of the association, it's pattern, and specificity. *Psychological Bulletin* 1995;117:285–305.
- Christensen KJ, Won Kin S, Dysken MW, Maxwell Hoover K. Neuropsychological performance in obsessive–compulsive disorder. *Biological Psychiatry* 1992;31:4–18.
- Cohen LJ, Hollander E, DeCaria CM, Stein DJ, Simeon D, Liebowitz MR, et al. Specificity of neuropsychological impairments in obsessive–compulsive disorder: a comparison with social phobic and normal control subjects. *Journal of Neuropsychiatry and Clinical Neurosciences* 1996;8:82–5.
- Delin CR, Lee TH. Drinking and the brain: current evidence. *Alcohol and Alcoholism* 1992;27:117–26.
- Dirson S, Bouvard M, Cottraux J, Martin R. Visual memory impairment in patients with obsessive–compulsive disorder: a controlled study. *Psychotherapy and Psychosomatics* 1995;63:22–31.
- Gladso JA, Rapaport MH, McKinney R, Lucas JA, Rabin A, Oliver T, et al. A neuropsychological study of panic disorder: negative findings. *Journal of Affective Disorders* 1998;49:123–31.
- Head D, Bolton D, Hymas N. Deficit in cognitive shifting ability in patients with obsessive–compulsive disorder. *Biological Psychiatry* 1989;25:929–37.
- Kwon JS, Kim JJ, Lee DW, Lee JS, Lee DS, Kim MS, et al. Neural correlates of clinical symptoms and cognitive dysfunctions in obsessive–compulsive disorder. *Psychiatry Research* 2003;122:37–47.
- Lucas JA, Telch MJ, Bigler ED. Memory functioning in panic disorder: a neuropsychological perspective. *Journal of Anxiety Disorders* 1991;5:1–20.
- Martinot JL, Allilaire JF, Mazoyer BM, Hantouche E, Huret JD, Lewgaut-Demare F, et al. Obsessive–compulsive disorder: A clinical, neuropsychological and positron emission tomography study. *Acta Psychiatrica Scandinavica* 1990;82:233–42.
- Mor N, Winquist J. Self-focused attention and negative affect: a meta-analysis. *Psychological Bulletin* 2002;128:638–62.
- Nilsson L-G. Category norms for verbal material. Tech Rep. No. 135. Uppsala, Sweden: Department of Psychology, University of Uppsala; 1973.
- Nixon SJ, Kujawski A, Parsons OA, Yohman JR. Semantic (verbal) and figural memory impairment in alcoholics. *Journal of Clinical and Experimental Neuropsychology* 1987;9:311–22.
- Purcell R, Maruff P, Kyrios M, Pantelis C. Cognitive deficits on obsessive–compulsive disorder on test of frontal–striatal function. *Biological Psychiatry* 1998;43:348–57.
- Purcell R, Maruff P, Kyrios M, Pantelis C. Neuropsychological deficits in obsessive–compulsive disorder. A comparison with unipolar depression, panic disorder and, normal controls. *Archives of General Psychiatry* 1998;55:415–23.
- Reitan RM. Manual for administration of neuropsychological test batteries for adults and children. Indianapolis, Indiana: Author, 1959.
- Reitan RM, Davidson LA. Clinical neuropsychology: current status and applications. New York: Wiley; 1974.
- Savage CR, Keuthen NJ, Jenike MA, Brown HD, Baer L, Kendrick AD, et al. Recall and recognition memory in obsessive–compulsive disorder. *Journal of Neuropsychiatry and Clinical Neurosciences* 1996;8:99–103.
- Savage CR, Baer L, Keuthen NJ, Brown HD, Rauch SL, Jenike MA. Organizational strategies mediate nonverbal memory impairment in obsessive–compulsive disorder. *Biological Psychiatry* 1999;45:905–16.
- Savage CR, Deckersbach T, Wilhelm S, Rauch SL, Baer L, Reid T, et al. Strategic processing and episodic memory impairment in obsessive–compulsive disorder. *Neuropsychology* 2000;14:141–51.
- Tillfors M, Furumark T, Marteinsdottir I, Fischer H, Pissiota A, Lngström B, et al. Cerebral blood flow in subjects with social phobia during stressful speaking tasks: a PET study. *American Journal of Psychiatry* 2001;155:1220–6.
- Veale DM, Sahakian BJ, Owen AM, Marks IM. Specific cognitive deficits in tests sensitive to frontal lobe dysfunction in obsessive–compulsive disorder. *Psychological Medicine* 1996;26:1261–9.
- Vejjel HOF. A preliminary profile of neuropsychological deficits associated with major depression. *Journal of Clinical and Experimental Neuropsychology* 1997;19:587–603.
- Vythilingam M, Anderson ER, Goddard A, Woods SW, Staib LH, Charney DS, et al. Temporal lobe volume in panic disorder – a quantitative magnetic resonance imaging study. *Psychiatry Research: Neuroimaging Section* 2000;99:75–82.
- Wing JK, Babor T, Brugha TE, Burke J, Cooper JE, Giel R, et al. SCAN. Schedules for clinical assessment in neuropsychiatry. *Archives of General Psychiatry* 1990;47:589–93.
- Wolf AW, Schubert DSP, Patterson MB, Grande TP, Brocco KJ, Pendleton L. Associations among major psychiatric diagnoses. *Journal of Consulting and Clinical Psychology* 1988;56:292–4.
- Zielinski CM, Taylor MA, Juzwin KR. Neuropsychological deficits in obsessive–compulsive disorder. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology* 1991;4:110–26.
- Zitterl W, Urban C, Linzmayer L, Aigner M, Demal U, Semler B, et al. Memory deficits in patients with DSM-IV obsessive–compulsive disorder. *Psychopathology* 2001;34:113–7.