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## **Associations between residual depressive symptoms, cognition, and functioning in patients with euthymic bipolar disorder: results from the FACE-BD cohort**

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1 **Associations between residual depressive symptoms, cognition, and functioning in**  
2 **patients with euthymic bipolar disorder : results from the FACE-BD cohort**

3 Social functioning in euthymic bipolar disorder

4

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9

1 **Abstract**

2 **Background:** The relationship between residual depressive symptoms, cognition, and  
3 functioning in euthymic patients with bipolar disorders (BD) is a subject of debate.

4 **Aims:** Our objective was to assess whether cognition mediates the association between  
5 residual depressive symptoms and functioning in euthymic patients with BD.

6 **Methods:** We included 241 adults with euthymic BD in a multicenter cross-sectional study.  
7 We used a battery of tests to assess six cognition domains. A path analysis was then used to  
8 perform a mediation analysis of the relationship between residual depressive symptoms,  
9 cognitive components, and functioning.

10 **Results:** Only verbal and working memory were significantly associated with better  
11 functioning. Residual depressive symptoms were associated with poorer functioning. No  
12 significant relationship was found between residual depressive symptoms and any cognitive  
13 component.

14 **Conclusions:** Cognition and residual depressive symptoms appear to be two independent  
15 sources of variation in the functioning of euthymic patients with BD.

16 **Declaration of interest:** None.

17

18 **Keywords:** bipolar disorders; cognition; verbal memory; euthymia; attention; social  
19 functioning; executive functions; speed processing; working memory; principal component  
20 analysis, path analysis, mediation analysis

21

## 1 INTRODUCTION

2 Bipolar disorders (BD) are highly disabling (1) and prevalent (2). More than half the  
3 individuals with BD experience significant functional impairment in several domains, such as  
4 family and social life and work, outside the acute phases of the illness (3). In the last decade,  
5 the focus of research in BD has changed from clinical remission to functional recovery (3).  
6 However, the sources of high variation observed in the psychosocial functioning of  
7 individuals with BD are still poorly understood. Cognitive impairment is an important  
8 determinant of functional impairment in BD (4). Because the strength of association between  
9 poor social functioning and cognitive impairment in BD is similar to that seen in  
10 schizophrenia (5), it seems crucial to characterize the underlying architecture of cognitive  
11 performance in BD.

12 Previous studies investigating multiple cognitive areas in BD have usually focused on  
13 domains *a priori* that were subjectively defined and selected. However, the validity of these  
14 domains is questionable, as it relies on the assumption that the latent organization of human  
15 cognition is similar in persons with BD and healthy controls. **This crucial assumption has**  
16 **received few experimental evidence (6). It thus remains possible that** the same  
17 neuropsychological tests might evaluate different cognitive competencies in these two groups  
18 of participants if there is a discrepancy in how cognitive measures relate to one another  
19 between individuals with and without BD. This lack of equivalence between the constructs  
20 could lead to artifacts in the observed differences in cognitive test performances. A few  
21 studies have explored the latent cognitive structure in BD and found that cognitive  
22 functioning was best described by multi-factorial models (6-8). However, **some of these**  
23 **studies** included symptomatic patients (7, 8). Here, we have measured performance for a  
24 broad range of cognitive domains in euthymic BD using a comprehensive battery of  
25 neuropsychological tests. We have examined the component structure of BD cognitive



1 processes using a Principal Component Analysis, which allows the data-driven reduction of  
2 multiple cognitive measures, avoids arbitrary and *a priori* categorization of several tests into  
3 domains, and results in a reliable estimate of underlying cognitive constructs in BD.

4 Among clinical factors, residual depressive symptoms have been reported to be the  
5 strongest predictor of functional impairment (9) and quality of life (10) in euthymic BD. They  
6 are also associated with lower adherence to medication in BD (11). In contrast, residual  
7 hypomanic symptoms have no impact on functioning in euthymic BD (4, 9, 12-14). Whereas  
8 residual hypomanic symptoms are not related to cognition (15), it is less clear whether  
9 subthreshold depressive symptoms negatively affect neuropsychological performance in  
10 euthymic BD or not. The relationship between residual depressive symptoms and cognition is  
11 mixed, showing a small impact of subthreshold depression on only a few cognitive  
12 components, such as verbal memory, speed, and executive function, but not for others (16). A  
13 few studies have explored the role of cognition in mediating the relationship between  
14 depressive symptoms and functioning in euthymic BD, leading to inconsistent results. One  
15 study showed that verbal memory partially mediated the relationship between subthreshold  
16 depressive symptoms and functional outcome in euthymic people with BD (17). However, the  
17 sample size was not large enough to include other cognitive moderators in the model. In  
18 contrast, another study reported that cognition did not mediate the relationship between  
19 depressive symptoms and social competencies in BD (18). Again, only one global  
20 neurocognitive composite score was included as the cognitive moderator in the model, due to  
21 the limited sample size. There have been no studies, to date, that have investigated  
22 simultaneous mediation between sub-depressive symptoms and functioning by multiple  
23 cognitive components in BD. In this study, the cognitive domains obtained with PCA were  
24 entered in a path analysis model, as potential mediators between residual depressive  
25 symptoms and functioning.

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## **METHODS**

### **Study design and recruiting network characteristics**

This multicenter, cross-sectional study included patients recruited into the FACE-BD (FondaMental Academic Centers of Expertise for Bipolar Disorders) cohort by a French national network of nine BD Expert Centers (Bordeaux, Créteil, Grenoble, Marseille, Monaco, Montpellier, Nancy, Paris and Versailles). This network was set up by the FondaMental Foundation ([www.fondation-fondamental.org](http://www.fondation-fondamental.org)) and funded by the French Ministry of Research and the French Ministry of Health to build an infrastructure and provide resources to follow clinical cohorts and comparative-effectiveness research on a representative patient population.

### **Participants**

BD was diagnosed based on a structured clinical interview that assessed the DSM-IV-R criteria. Outpatients of 18 to 65 years of age with type I, II, or not otherwise specified (NOS, including cyclothymia) BD were eligible. All patients were euthymic when they were tested according to the DSM-IV-R criteria, with a Montgomery Åsberg Depression Rating Scale (19) (MADRS) of less than 10 and a Young Mania Rating Scale (20) (YMRS) of less than 10. Exclusion criteria were a history of neurological or sensory disorders, dyslexia, dysorthographia, dyscalculia, dysphasia, dyspraxia, language delay, substance-related disorders in the previous month, and electroconvulsive therapy in the past year. The ethics committee (Comité de Protection des Personnes Ile de France IX) approved the study protocol on January 18, 2010. Although the committee waived the requirement for written informed consent, the patients received a letter informing them of the study and asking whether they agreed to participate.

1

## 2 **Assessment tools**

### 3 *Clinical assessments*

4       The age at onset; number of previous mixed, hypomanic, manic, and major depressive  
5 episodes; subtype of BD; and history of psychotic symptoms were recorded. We used the  
6 yes/no format for recording whether the patient was taking lithium carbonate, anticonvulsants,  
7 antipsychotics, antidepressants, or anxiolytics at the time of the evaluation. Finally, three  
8 socio-demographic characteristics were collected: sex, age, and educational level.

9       Psychosocial functioning was measured using the Functioning Assessment Short Test  
10 (FAST), which covers six functioning domains: autonomy, occupational functioning,  
11 cognitive functioning, financial issues, interpersonal relationships, and leisure time (21). The  
12 higher the score, the greater the disability. FAST has good internal consistency (Cronbach  
13 alpha between 0.87 and 0.96) and test-retest reliability (Pearson correlation coefficient  
14 between 0.90 and 0.97; intraclass correlation coefficient between 0.90 and 0.98, [see](#)  
15 [Supplemental References S1 for a bibliography of FAST psychometrical properties](#)).

16

### 17 *Battery of cognitive tests*

18 Experienced psychologists administered the tests in a fixed order. Testing lasted a total of 120  
19 minutes, including a 5 to 10-minute break. The standardized test battery complied with the  
20 recommendations issued by the International Society for Bipolar Disorders (22). It included  
21 11 tests and evaluated the following six cognitive domains:

- 22 - motor speed with the digit symbol coding and symbol search subtests from the Wechsler  
23 Adult Intelligence Scale (WAIS) version III;
- 24 - attention with the Conners' Continuous Performance Test II (CPT-II) (23) and the Trail  
25 Making Test (TMT) (24);

- 1 - executive functions with the Stroop color and word test and verbal fluency (25) ;
  - 2 - verbal memory with the California Verbal Learning Test (CVLT) (26);
  - 3 - working memory with the digit span subtest from the WAIS III and the spatial span subtest
  - 4 from the Wechsler Memory Scale version III; and
  - 5 - intellectual functioning with the vocabulary and matrix reasoning subtests from the WAIS-
  - 6 III
- 7 Some of the current cognitive data obtained with this battery have been published previously
- 8 (27).

9

## 10 **Statistical analyses**

11

### 12 *Principal Component Analysis (PCA)*

13 PCA is a powerful statistical method for identifying the underlying organization of multiple

14 variables like cognitive measures and allows data reduction necessary to avoid Type I errors

15 from multiple comparisons. The data set for the PCA included 22 raw cognitive variables:

16 number of correct answers for the digit symbol coding and symbol search tests, percentages of

17 omissions and commission errors for the CPT, reaction time (ms) for hits in the CPT, time (s)

18 to complete the TMT-A & B, number of responses in the color, word, and color-word

19 conditions of the Stroop test, number of correct words for phonemic and semantic verbal

20 fluency, number of recalled words in the immediate recall, short, and long delay free recall of

21 the CVLT, number of total correct recognized words for the CVLT, span lengths for the

22 forward and backward conditions of the spatial and digit span tests, number of correct

23 answers for matrix reasoning, and total score for vocabulary.

24 Sampling adequacy was evaluated using the Kaiser-Meyer-Olkin (KMO) measure (28) for the

25 overall cognitive data set and Bartlett's test of sphericity (29). The number of components to

1 be extracted in the PCA was determined by Horn's parallel analysis (30). This method  
2 contrasts eigenvalues produced through a parallel PCA on 1000 random datasets, with the  
3 same number of variables and observations as the observational dataset, to generate  
4 eigenvalues for components that are adjusted for sample error-induced inflation. Adjusted  
5 eigenvalues  $> 1$  indicate dimensions to retain.

6 We ran a PCA on cognitive variables followed by an Oblimin rotation. The rotation was  
7 performed to simplify the component structure. We used an oblique rotation because the  
8 cognitive components were believed to be correlated with each other. In PCA, the usual  
9 standard for sample size is a participant-to-variable ratio  $> 5$  (31), and therefore required 110  
10 participants for the current study.

11

#### 12 *Path and mediation analysis*

13 Zero-order correlations between MADRS, cognitive components, FAST scores, age,  
14 sex, and education were calculated using Pearson correlation coefficients. A path analysis was  
15 performed using MADRS, cognitive components, and FAST scores to test whether cognitive  
16 components mediated the relationship between residual depressive symptoms and  
17 functioning. The model tested in the path analysis did not include the YRMS score, as we  
18 expected it to correlate with neither cognition nor functioning. The model allowed the residual  
19 variances of the cognitive components scores to be correlated. Age, sex, and education were  
20 used as covariates in the model.

21 Analyses were performed using the *lavaan* package of *R* statistical software version 3.3  
22 with the Maximum Likelihood estimation method. Linear regression analyses were conducted  
23 to evaluate the relationships among the variables and were indexed using standardized path  
24 coefficients. Because the FAST total score is usually not normally distributed in euthymic BD

1 (32), we used a nonparametric bootstrapping of the standard errors with 2000 iterations for the  
2 correlation and SEM analyses.

3 The fit between the model and the data was assessed using four indices: the chi-square  
4 goodness-of-fit statistic ( $\chi^2$ ), comparative fit index (CFI), root-mean-squared error of  
5 approximation (RMSEA), and standardized root mean square residual (SRMR).

## 6 7 8 **RESULTS**

### 9 **Participants**

10 We included 241 patients. Table 1 reports their sociodemographic and clinical  
11 characteristics and Table 2 the results of the battery of cognitive tests. No patient had more  
12 than 5% missing cognitive data; the missing cognitive data were estimated using a  
13 multivariate imputation by chained equations in the *mice* package of *R*.

### 14 15 **PCA**

16 The KMO measure for the overall cognitive data set was 0.85 and Bartlett's test of  
17 sphericity was significant ( $X^2(231) = 2295, p < 0.001$ ), both indicating good factorability of  
18 the cognitive data. Horn's parallel analysis showed that five components should be retained,  
19 as their adjusted eigenvalue was above 1 (see Supplemental Table S2). The 5-component  
20 structure explained 61% of the variance (see Supplemental Table S3). All component loadings  
21 were greater than 0.4, and all communalities were higher than 0.3 (see Table 3). The first  
22 component consisted of all measures of the CVLT and was designated "verbal memory." The  
23 second component bundled TMT, CPT omissions, symbol search, vocabulary, symbol coding  
24 and semantic verbal fluency and was designated "speed of processing and verbal knowledge."  
25 The third component included all measures of spatial and digit spans, with matrix reasoning,  
26 and was designated "working memory and problem-solving." The fourth component consisted

1 of all measures of the Stroop test, with phonemic verbal fluency, and was designated “verbal  
2 fluency and inhibition.” The final component included CPT reaction time and commission  
3 and was designated “visual sustained attention.”

#### 4 5 **Path and mediation analyses**

6 Supplemental Table S4 reports the zero-order correlations between the variables  
7 included in the model. The path analysis model allowed correlations between the residual  
8 variances of all cognitive components, except “visual sustained attention” which was not  
9 correlated with any other cognitive components.

10 The path analysis model is shown in Figure 1. We represent neither covariances  
11 between cognitive components, nor the regressions on covariates, to enhance readability.

12 Path analysis requires at least 15 participants for each variable (33). We included ten  
13 variables in the model and therefore required at least 150 participants. There were 0.8%  
14 missing data, which were handled using the full information maximum likelihood estimation.  
15 The four patterns of missingness are reported in Supplemental Table S5 and the covariance  
16 coverage matrix of missing data in Supplemental Table S6.

17 The model provided a good fit for the data, as suggested by the nonsignificant chi-  
18 square goodness-of-fit statistic ( $X^2(4) = 7.5$ ,  $p = 0.113$ ), a CFI greater than 0.95 (0.989), an  
19 RMSEA not significantly larger than 0.05 (RMSEA = 0.06, one-sided  $P$  value of the test of  
20 the null hypothesis RMSEA = 0.05, 0.329), and SRMR lower than 0.08 (0.017).

21 The model explained 30% of the variance in functioning. Altogether, the analysis  
22 revealed the following relationships between the variables (Figure 1): a significant positive  
23 association between MADRS and FAST, a significant negative association between “verbal  
24 memory” cognitive component and FAST, and a significant negative association between the  
25 “verbal fluency and inhibition” cognitive component and FAST. We found no other  
26 significant associations, and in particular, MADRS was not significantly associated with any

1 cognitive component, thus showing that cognitive component scores did not mediate the  
2 relationship between MADRS and FAST. Estimated standardized path coefficients for all  
3 variables included in the path analysis model (including covariates) and residual correlation  
4 coefficients between cognitive components are reported in Supplemental Table S7.

5  
6

## 7 **DISCUSSION**

8       Here, we first used a PCA to identify the underlying architecture of cognitive processing  
9 in euthymic patients with BD. We then used a path analysis to evaluate whether cognition  
10 mediated the relationship between residual depressive symptoms and functioning.

11       We found five underlying components involved in cognition in euthymic BD. Two  
12 components were derived from individual variables that were relatively homogeneous and  
13 specific regarding modality: the “verbal memory” component was derived only from CVLT  
14 measures and the “visual sustained attention” only from CPT measures. The remaining three  
15 components were more heterogeneous. The “verbal fluency and inhibition” component  
16 consisted of verbal responses sometimes involving inhibition (phonemic verbal fluency and  
17 color/word condition of the Stroop test) and sometimes not (color and word condition of the  
18 Stroop test). The “speed of processing and verbal knowledge” component contained a  
19 combination of visuospatial and verbal variables. The “working memory and problem  
20 solving” component bundled non-verbal reasoning and working memory measures. Greater  
21 variability within cognitive components has already been reported in BD and was interpreted  
22 as a decrease in the differentiation of previously discrete cognitive processes through a  
23 decline in neural connectivity (8). The number of extracted cognitive components is similar to  
24 that of previous studies which found five (6) or six underlying dimensions in cognition for  
25 BD (7). The labels used in these previous studies to describe the cognitive dimensions were  
26 similar to those applied in the current study, consisting of verbal memory, speed of



1 processing, working memory, executive functions, verbal knowledge, and attention  
2 dimensions. This suggests a relative stability and reliability of the method we used to uncover  
3 the underlying cognitive components in BD.

4         There was a negative association between residual depressive symptoms and  
5 functioning: individuals with more pronounced depressive symptoms had a poorer social  
6 functioning, which was not explained by age, sex, lower education, or poorer cognition. This  
7 finding is in accordance with previous cross-sectional (13, 17, 18, 32) and longitudinal studies  
8 (9, 14), showing that subclinical depressive symptoms in bipolar disorders are the main  
9 predictors of poor functional outcome, particularly work functioning (34). The present study  
10 included only euthymic patients, based on stringent criteria: the mean score for depression  
11 was very low but similar to previous studies exploring social functioning in euthymic BD (9,  
12 13, 17, 32). However, the data show that functional impairment may be associated with even  
13 very low residual depressive symptoms, measured with a scale that was not specifically  
14 designed to assess subsyndromal depressive symptoms.

15         Among the five cognitive components found in this study, only two were positively  
16 associated with functioning: “verbal memory” and “verbal fluency and inhibition.” Patients  
17 with better verbal memory, verbal fluency and inhibition also had a better social functioning.  
18 These results are consistent with several prior reports indicating that verbal memory (14) and  
19 inhibitory control (Stroop Colour Word Test) (35) were more highly associated with  
20 functioning than other cognitive functions. That functioning was more highly related to  
21 residual depressive symptoms than cognition could be explained by the auto evaluation  
22 method we used to measure everyday functioning. Self-reported measures of social  
23 functioning may be influenced less by objective cognitive performance and more by  
24 depressive symptomatology, due to a pessimistic subjective appraisal of oneself and one's  
25 environment. **This influence might be particularly important for low FAST scores (better**

1 functioning), like in our sample, where the distribution of FAST scores was skewed on the  
2 right. In contrast, the reverse may be true for performance-based measures of functioning and  
3 real-world functional milestones in BD, which may be influenced more by objective cognitive  
4 performance and less by depression (5, 12).

5 No associations between residual depressive symptoms and cognitive components were  
6 significant, showing that cognition does not mediate the relationship between subclinical  
7 residual depressive symptoms and functioning. This result is in accordance with a previous  
8 study reporting that perceived cognitive impairment and subclinical residual depressive  
9 symptoms are two independent sources of variation in the functioning of individuals with BD  
10 (36). However, this mediation might occur for higher levels of depressive symptoms, as the  
11 impact of depressive symptoms on cognition varied according to the clinical response after  
12 treatment (37).

13 Our model explained only 30% of the variance in functioning, supporting a role for  
14 other factors that were not measured here. Previous studies have suggested that functioning  
15 may be impaired when sleep is persistently disrupted (38), when social cognition is impaired  
16 (13), and when episode density is high (35). The mean level of functioning in participants  
17 recruited in this study corresponds to moderate functional difficulties (3). It is in the range  
18 (from 6 to 29) of those found in studies exploring the relationship between cognition and  
19 functioning in euthymic BD (13, 32, 36). This consistency supports the general applicability  
20 of our findings to patients with euthymic BD, provided the same assessment tools are used.

21

22 Limitations of our study include the cross-sectional design, which precludes the  
23 assessment of causality and the direction of potential causal links. Another limitation is the  
24 lack of inclusion of social cognitive tasks in the assessment. We did not compare the  
25 cognitive architecture found in the current sample of BD patients to a control group. Finally,

1 the sample size was not large enough to test a more complex model that includes other  
2 variables of the illness (type of BD, number of previous episodes, age at onset, and history of  
3 psychosis) and medication. **The time since the last mood episode might also be an important**  
4 **factor lacking in the present study.** These variables might have also had an effect on  
5 functioning in euthymic BD.

6 Our findings have important implications for future clinical studies. Individuals with  
7 BD who respond to treatment may nevertheless continue to experience residual depressive  
8 and cognitive symptoms, leading to difficulties in functioning. First, it seems crucial to  
9 improving the assessment and the characterization of residual depressive symptoms, with for  
10 example specific scales. Treatment approaches should possibly include the cognitive  
11 performance improvement and the full residual depressive symptoms remission as important  
12 targets to obtain functional recovery in BD. The optimal method to treat residual depressive  
13 symptoms is not clear, but they may be targeted by the use of mood stabilizers effective in the  
14 treatment of depressive polarity. Evidence for the efficacy of pharmacological and  
15 psychological interventions that target cognitive deficits in BD is still preliminary, despite  
16 promising avenues such as functional remediation (39). Among cognitive dimensions for  
17 cognitive remediation in euthymic BD, our data suggest that verbal memory and fluency, and  
18 inhibition may be choice targets.

19 In summary, this study provides further evidence that cognitive impairments in specific  
20 dimensions are a core feature of BD. This study also suggests that cognition is a separable  
21 dimension from depressive symptoms that persist during the inter-episodic period of BD.  
22 Verbal memory and fluency and Stroop test performance were particularly associated with  
23 functioning in our sample of euthymic BD and should be assessed in future studies focusing  
24 on functional outcome in BD.

25

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2

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11

12

### 13 **Figure Legends**

14 **Figure 1.** Mediation Model. Rectangles represent the observed measured variables. Arrows  
15 showing the free regression weight are drawn between variables. Values are the standardized  
16 path coefficients. The squared multiple correlation ( $R^2$ ) value for the dependent variable  
17 appears in the upper right corner of each rectangle. Covariates and covariation between the  
18 cognitive components are not drawn to increase readability but were indeed included in the  
19 model.

20 \*\* $0.01 > p > 0.001$ , \*\*\* $p < 0.001$ .

21 MADRS: Montgomery Åsberg Depression Rating Scale

22 FAST: Functioning Assessment Short Test

23

24

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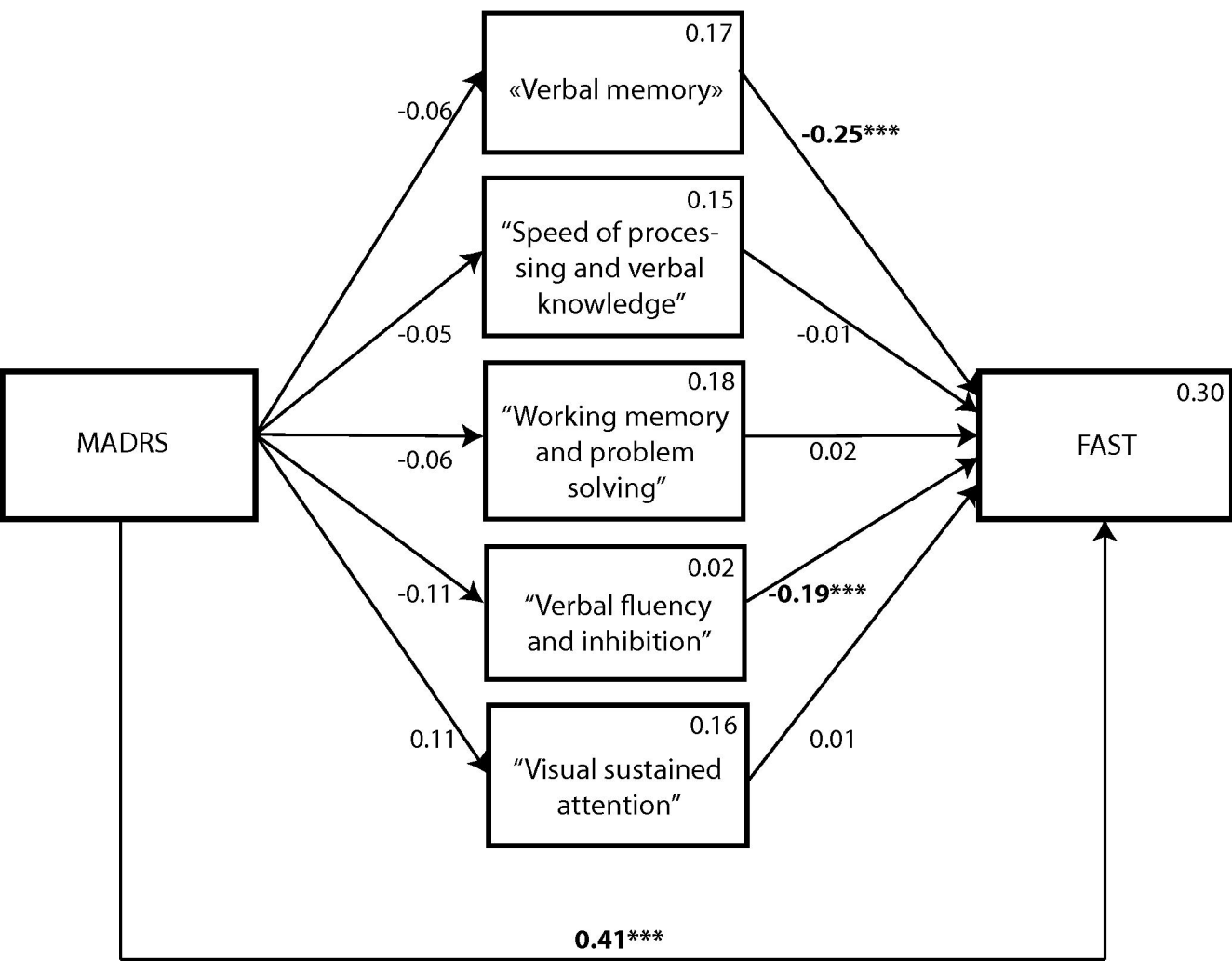
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- 10

Cognitive component scores



**Table 1.** Participant socio-demographical and clinical characteristics:

<b>Variable</b>	<b>Mean</b>	<b>SD</b>	<b>Range</b>
Age (years)	41	11.3	19-65
Educational level (years)	14.5	2.7	7-22
Age at onset (years)	25	9.5	12-60
Number of mixed episodes	0.2	0.7	0-8
Number of hypomanic episodes	2.8	5.3	0-30
Number of manic episodes	1.4	1.9	0-10
Number of major depressive episodes	5.2	5.4	0-30
MADRS [0-60]	4	3.4	0-10
YMRS [0-60]	1.1	2	0-10
FAST [0-72]	16.8	13.2	0-64
<b>Percentage</b>			
Sex (percentage of males)	40.2		
Diagnosis	55.6 (Type 1)	29.5 (Type 2)	14.9 (NOS)
History of psychosis	43.6		
Antidepressant	26		
Lithium Carbonate	25		
Anticonvulsant	32.7		
Antipsychotic	28.4		

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MADRS: Montgomery Åsberg Depression Rating Scale

YMRS: Young Mania Rating Scale

FAST: Functioning Assessment Short Test

NOS: Not Otherwise Specified

**Table 2.** Participant neuropsychological performance:

<b>Function</b>	<b>Test</b>	<b>Variable</b>	<b>Mean (percentile)</b>	<b>SD (percentile)</b>	<b>Mean (raw)</b>	<b>SD (raw)</b>
Motor speed	Digit / Symbol Coding Symbol Search		43.1	25.8	70.2	15.2
			56.2	28	33.7	7.5
Attention	Continuous Performance Test	Omissions	43.1	27.9	2.2%	4.6%
		Commissions	53.6	30.1	30.8%	20.2%
		Reaction Time	52	32.6	402.5 ms	84.2 ms
	Trail Making Test	Part A	49.6	25.7	35 s	13 s
Part B		45.5	28.3	104.1 s	15.9 s	
Executive functions	Stroop Test	Word	45.8	22.8	104.1	15.9
		Color	37.4	25.2	73	12.9
		Color/Word	47	29.8	43.1	11.1
	Verbal Fluency	Phonemic	48.2	31.3	24.1	7.3
		Semantic	39.9	27.9	31.7	8
Verbal memory	California Verbal Learning Test	Immediate Recall	46.8	32.5	56.4	9.9
		Short Delay Free Recall	46.9	30	11.9	2.9
		Long Delay Free Recall	44.8	30.3	12.2	2.7
		Total Recognition	52.8	26.3	15.1	1.2
Working memory	Spatial Span	Forward	46.1	26.5	8.3	1.9
		Backward	43.9	26	7.4	1.8
	Digit Span	Total	43.6	25.9		
		Forward			9.4	2
		Backward			6.4	2.3
Intellectual functioning	Matrix Reasoning Vocabulary		45.8	29	18.3	4.5
			61.9	28	42.9	10.2

Table 3. Component loadings and communalities for the cognitive variables

Variable	Component loadings					Communality
	1	2	3	4	5	
CVLT Long Delay Free Recall	<b>0.94</b>	0.03	-0.06	0.04	0.02	0.88
CVLT Short Delay Free Recall	<b>0.92</b>	0.03	-0.03	0.01	0.01	0.85
CVLT Immediate Recall	<b>0.84</b>	0.02	0.05	0.09	-0.05	0.78
CVLT Total Recognition	<b>0.73</b>	-0.15	0.06	-0.23	0.07	0.55
TMT B	-0.13	<b>-0.69</b>	-0.16	0.03	-0.08	0.66
CPT Omissions	0.26	<b>-0.65</b>	0.17	0.04	0.02	0.37
Symbol Search	0.1	<b>0.65</b>	0.06	0.17	-0.1	0.62
Vocabulary	-0.03	<b>0.62</b>	0.04	0	0.25	0.44
Symbol Coding	0.17	<b>0.61</b>	0.05	0.21	-0.15	0.65
TMT A	-0.07	<b>-0.52</b>	-0.2	-0.03	0.16	0.46
Verbal Fluency Semantic	0.03	<b>0.46</b>	0.13	0.24	0	0.41
Spatial Span Backward	-0.02	0.16	<b>0.74</b>	-0.09	-0.03	0.61
Spatial Span Forward	0.01	0.04	<b>0.73</b>	-0.02	-0.06	0.55
Digit Span Backward	0.08	-0.04	<b>0.69</b>	0.21	0.06	0.62
Digit Span Forward	-0.08	-0.14	<b>0.65</b>	0.35	0.17	0.57
Matrix Reasoning	0.18	0.3	<b>0.56</b>	-0.35	0.01	0.62
Stroop Word	-0.04	0.08	0.01	<b>0.77</b>	0.02	0.63
Stroop Color	0.13	0.08	0.1	<b>0.69</b>	-0.06	0.62
Stroop Word/Color	0.11	0.09	0.18	<b>0.56</b>	-0.16	0.5
Verbal Fluency Phonemic	0.09	0.27	-0.18	<b>0.41</b>	0.34	0.39
CPT Reaction Time	-0.03	-0.13	0	-0.09	<b>0.86</b>	0.78
CPT Comissions	-0.07	-0.14	-0.05	-0.06	<b>-0.83</b>	0.74