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Parsing Trait and State Effects of Depression Severity on Neurocognition: Evidence from a 26-Year Longitudinal Study

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Abstract

Cognitive dysfunction in mood disorders falls along a continuum, such that more severe current depression is associated with greater cognitive impairment. It is not clear whether this association reflects transient state effects of current symptoms on cognitive performance, or persistent, traitlike differences in cognition that are related to overall disorder severity. We addressed this question in 42 unipolar and 47 bipolar participants drawn from a 26-year longitudinal study of psychopathology, using measures of attention/psychomotor processing speed, cognitive flexibility, verbal fluency, and verbal memory. We assessed (a) the extent to which current symptom severity and past average disorder severity predicted unique variance in cognitive performance; (b) whether cognitive performance covaried with within-individual changes in symptom severity; and (c) the stability of neurocognitive measures over six years. We also tested for differences among unipolar and bipolar groups and published norms. Past average depression severity predicted performance on attention/psychomotor processing speed in both groups, and in cognitive flexibility among unipolar participants, even after controlling for current symptom severity, which did not independently predict cognition. Within-participant state changes in depressive symptoms did not predict change in any cognitive domain. All domains were stable over the course of six years. Both groups showed generalized impairment relative to published norms, and bipolar participants performed more poorly than unipolar participants on attention/psychomotor processing speed. The results suggest a stable relationship between mood disorder severity and cognitive deficits.

> Mood disorders are characterized by cognitive as well as affective disturbance. Metaanalyses have consistently reported deficits in attention, executive functions, and memory in both unipolar (Zakzanis, Leach, & Kaplan, 1998) and bipolar depression (Kurtz & Gerraty, 2009). These deficits appear to fall along a continuum, such that more severe depressive symptoms are associated with greater cognitive deficits (McClintock, Husain, Greer, & Cullum, 2010; also see meta-analyses by Christensen, Griffiths, MacKinnon, & Jacomb [1997] and McDermott & Ebmeier [2009]). However, the meaning of this association is not

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clear. At least two alternative interpretations are possible: a *state effects* hypothesis and a *trait-like relationship* hypothesis.

According to the *state effects* hypothesis, a correlation of current symptom severity with cognitive performance could mean that current depressive symptoms *cause* poor cognitive performance—that is, as an individual's symptoms become more severe, his or her performance becomes poorer. Consistent with this possibility, some studies have shown that rumination or sad mood induction has deleterious effects on cognition in non-clinical samples (e.g., Bartolic, Schefft, Glauser, & Titanic-Schefft, 1999), but others have not found this effect (e.g., Clark, Iversen, & Goodwin, 2001). Similarly, some have reported improved cognition upon recovery from unipolar or bipolar depressive episodes (Beats, Sahakian, & Levy, 1996; Malhi et al., 2007). However, these studies have conflicted regarding which domains show improvement upon remission, and generally have not taken practice effects into account. The one study to our knowledge that did control for practice effects found that impairments did not improve in any domain after depressive symptoms remitted (Reischies & Neu, 2000). Thus, evidence for the state effects hypothesis is inconclusive.

A second possible interpretation of correlations between depression severity and cognition is that individuals who tend to experience more severe, recurrent, and/or chronic depressive episodes show persistent (i.e., *trait-like*) cognitive deficits. This would produce a correlation between current depression severity and cognitive performance because individuals with greater "trait depression severity" are more likely to have high levels of symptom severity at any given sampling point (Judd et al., 1998), and consistently show poor cognition. In support of this possibility, two studies have reported greater cognitive dysfunction among unipolar and bipolar individuals with more recurrent depression (Basso & Bornstein, 1999; Robinson and Ferrier, 2006). Furthermore, deficits in attention and processing speed appear to be longitudinally stable in individuals with mood disorders (Bonner-Jackson, Grossman, Harrow, Rosen, & Goldberg, 2010; Burdick, Goldberg, Harrow, Faull, and Malhotra, 2006), although these studies did not examine effects of mood fluctuation over time.

In sum, the correlation between current symptoms and cognition may reflect (a) a *state effect* of symptoms on cognition or (b) a *trait-like relationship* between "depressive disorder severity" and cognition. This distinction is not merely academic, but has implications for what neuropsychological studies of depression tell us about the etiology and maintenance of mood disorders. For instance, if correlations reported in the literature largely reflect state effects (e.g., lower processing speed results from fatigue associated with depressive episodes), then they may be mere artifacts of depressive symptoms, and minimally informative about underlying mechanisms. On the other hand, if these correlations reflect trait associations (e.g., poorer processing speed is a stable, trait-like deficit associated with more recurrent, chronic, or severe depressive disorders), they are more likely to be informative about the role of dysfunctional cognitive or brain systems in the pathogenesis of depression. No study to date has directly compared effects of current state to overall trait depression severity on cognition.

Previous studies of depression severity and cognition may have been limited by a reliance on a taxonic conceptualization of depression. For instance, nearly every study that has reported a correlation between depressive symptoms and cognitive performance has excluded individuals not meeting full criteria for a current MDE, implying a qualitative difference between individuals above and below the MDE threshold. However, many taxometric studies indicate that depression is a dimensional phenomenon (e.g., Slade & Andrews, 2005), and dimensional models of depressive symptoms have greater predictive validity than categorical models (Prisciandaro & Roberts, 2009). Therefore, restricting a study's sample to individuals currently experiencing an MDE may not fully capture the

relationship between symptom severity and cognitive functioning. Moreover, requiring that participants meet criteria for an MDE underrepresents individuals who experience only mild or infrequent symptoms. For these reasons, modeling the effects of the full range of depressive symptoms (from euthymic to severely depressed) on cognition may be a more valid approach to this question.

Another methodological challenge is that there are no established measures of "trait depression severity." One useful approach may be to sample current symptom severity across a number of prospective observations and derive an average depression severity score. This variable would capture the average of the waxing and waning of depressive symptoms over time, and thus would be higher both among individuals who experience more severe episodes and among individuals who experience more chronic or frequent episodes (Klein, 2008). Because this variable is based on multiple observations, it would be a *more reliable estimate* of overall mood disorder severity than a single observation of current symptom severity. Therefore, if the trait-like relationship hypothesis is correct, past average severity should be a stronger predictor of cognition than current symptom severity. In contrast, current severity (by definition) reflects current mood state, whereas past average severity does not. Therefore, if the state effect hypothesis is correct, then current symptom severity should be a stronger predictor of cognition than past average severity.

Using a 26-year longitudinal dataset, the current study examined the unique effects of state and trait depression severity on attention/psychomotor processing speed, cognitive flexibility, verbal fluency, and verbal memory. We investigated these effects in a sample of currently depressed or euthymic individuals with unipolar depression or bipolar disorder. (Individuals who were currently manic or psychotic were not included in order to reduce noise from factors other than depression that may relate to cognitive disturbance.) The primary aims of the study are to examine (a) the amount of unique variance in cognitive functioning explained by current versus past average depression severity; (b) whether state changes in depressive symptoms predict change in cognition over the course of six years; and (c) whether cognition shows a trait-like longitudinal stability over the course of six years.

Lastly, evidence conflicts regarding whether unipolar and bipolar individuals differ on cognitive function. Some comparisons have indicated poorer performance in bipolar depression on memory and executive tasks (e.g., Borkowska & Rybakowski, 2001), whereas others have found no differences between unipolar and bipolar depression (e.g., Sweeney, Kmiec, & Kupfer, 2000). One study even found poorer executive function in unipolar than bipolar depression (Taylor Tavares et al., 2007). We therefore compared these groups to each other and to published norms on cognitive performance.

Method

Participants

The present sample is drawn from the Chicago Follow-Up Study, a 26-year prospective research program studying severe psychopathology (Bonner-Jackson et al., 2010; Burdick et al., 2006; Harrow et al., 2000). Participants were recruited from inpatient psychiatric units and initially diagnosed by Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978) for an acute depressive or manic episode based on a structured interview. Participants were reassessed seven times after index hospitalization, at approximately 2, 4.5, 7.5, 10, 15, 20, and 26 year points. Beginning at the 15-year follow-up, a neuropsychological battery was administered.

The sample used for the present study consists of 89 individuals who participated in neuropsychological testing (42 unipolar and 47 bipolar). Participants were classified as bipolar in analyses if they a) were diagnosed with bipolar I disorder at index hospitalization (n=32) or b) met *DSM-IV* criteria for a past year hypomanic or manic episode at any follow-up point based on the Schedule for Affective Disorders and Schizophrenia (SADS, Endicott & Spitzer, 1978; n=15). A subset of 44 participants (19 unipolar and 25 bipolar) completed the neuropsychological battery at two time points, on average 6.4 years apart (SD = 2.4 years) and were therefore included in longitudinal analyses.

Participants who were psychotic (n=11) or who met more than two *DSM-IV* criteria for a manic episode (n=5) according to the SADS at the time of neuropsychological assessment were excluded.¹ The sample size of 89 reported above reflects these exclusions.

Measures

Diagnostic assessment—Depressive symptoms over the previous two weeks were assessed at each follow-up using the composite depressed mood and behavior subscore from the Katz Adjustment Scales (KAS; Katz and Lyerly, 1963). The KAS is a 55-item self-report instrument that assesses domains of distress and adjustment, and was the precursor to the SCL-90. KAS depression severity at the time of neuropsychological assessment was strongly correlated with a past month depression severity score derived from the SADS, r(87) = .75, p < .001.

We computed "past average depression severity" by averaging each participant's KAS depression severity ratings over all follow-ups prior to the first neuropsychological assessment. The primary aim of the study involved comparing the current and past average depression variables' predictive ability in regression analyses. To ensure the validity of this comparison, we assessed whether these variables differed in mean level or distribution. The two variables had similar means (current: 15.0; past average: 14,0; t(78) = -.27, *ns*) and distributions (current: SD = 4.1, skewness = 0.8, $SE_{skew} = 0.3$; past average: SD = 5.0, skewness = 1.4, $SE_{skew} = 0.3$).

Neuropsychological battery—The neuropsychological battery consisted of tests falling into four domains. To compute domain composite scores, we converted raw scores from each cognitive measure to *z*-scores; reversed the direction of these, as appropriate, so that positive numbers indicated better performance; and summed the resultant *z*-scores as follows:

- *Attention/Psychomotor Processing Speed:* a) Digit Symbol from the Wechsler Adult Intelligence Scale–Revised (WAIS-R; Wechsler, 1981) and b) Trail Making Test part A (Reitan, 1979)
- Cognitive Flexibility: a) Percent perseverative errors and b) categories completed from the Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1993)²
- Verbal Fluency: The C-F-L phonemic fluency task (Benton & Hamsher, 1976)

¹The current investigation did not assess effects of manic symptoms (although an important topic; Kurtz and Gerraty, 2009), because few individuals were manic at the time of testing. ²Trails B, considered a measure of attentional set-shifting (Bowie & Harvey, 2006), was not included in the cognitive flexibility

²Trails B, considered a measure of attentional set-shifting (Bowie & Harvey, 2006), was not included in the cognitive flexibility composite due to weak correlations with other constituents of the composite. Nonetheless, analyses using a flexibility composite including Trails B yielded results very similar to those presented here.

Verbal Memory: a) List A trials 1–5 total, b) short delay free recall, c) long delay free recall, and d) discriminability from the California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987)

Intercorrelations among the constituent measures of each composite ranged from .47 to .84 (all p's < .001). The Information subtest from the WAIS was used as an estimate of premorbid intellectual functioning (DeQuardo, Goldman, Tandon, McGrath-Giroux, & Kim, 1995).

Data Analysis

Analyses were completed using SPSS 18.0. Alpha was set at .05 (two-tailed) for all analyses.

To identify differences between the sample and the normal population, we converted raw scores to *z*-scores based on published norms for each neurocognitive measure, summed these *z*-scores to form composites, and then compared each group's normed scores to the population mean (i.e., zero) using single sample *t*-tests. We assessed differences between unipolar and bipolar individuals using a series of linear regressions in which each cognitive domain was regressed on covariates (age, gender, education) and polarity (unipolar or bipolar).

To evaluate the effects of current and past average depression severity on cognition, we first ran separate linear models in which each cognitive composite was regressed on either current or past average KAS depression severity (and covariates). Then, to determine whether either predictor had unique effects on cognition over and above the effects of the other, we regressed each composite onto current and past average depression simultaneously. Significant unique effects of past average, but not current, depression in these models would suggest that cross-sectional associations between depression and cognition arise largely from trait depression severity. Conversely, significant unique effects of current, but not past average, depression would provide support for the state effects hypothesis. To test whether polarity moderated the results, each of these regression models included a second block containing polarity and the interaction of current and/or trait depression with polarity. Significant interactions were followed up using analyses of simple slopes. All predictors were mean-centered.

To test whether changes in current symptom severity predicted changes in neurocognition in the subset of participants who participated in neuropsychological testing at two time points, we computed change scores from the first to the second time point for current KAS depression severity and for the four cognitive composites. We regressed the change score for each neuropsychological measure on the change score for KAS depression and covariates (gender, education, change in age between time points). Polarity and a depression by polarity interaction term were included in a second block.

Finally, we assessed the longitudinal stability of cognitive performance using Pearson correlations from the first to the second time point.

Missing data—Not all individuals had mood ratings from every follow-up. We included participants in the current vs. past average severity analyses only if they had KAS data from at least four follow-ups (i.e., one from the neuropsychological follow-up and at least three from previous follow-ups). Seventy-nine participants met this criterion (38 unipolar and 41 bipolar); these participants had data from a mean of 4.9 follow-ups (SD = 0.7) spanning a mean of 16.2 years (SD = 3.9).

Additionally, not all individuals completed every neuropsychological test. Domain composites were only computed if an individual had complete data for all tests comprising that composite. Individuals who had data for some domains but not others were included in analyses of domains for which they had sufficient data.

Results

Demographic and Clinical Characteristics

Characteristics of the sample are presented in Table 1. Unipolar and bipolar participants were similar on most demographic and clinical variables, but bipolar participants were more likely to have a history of psychosis, to be taking lithium or antipsychotics, and to be taking psychiatric medications in general at the time of neuropsychological testing.

Group Differences on Neurocognition

Both groups of participants performed more poorly than published population norms on all four cognitive domains (Figure 1). The one exception to this pattern was that attention/ psychomotor processing speed was significantly lower than the population mean for bipolar, but not unipolar, participants. Consistent with this, bipolar participants showed poorer attention/psychomotor processing speed than unipolar participants, but the groups did not differ on other cognitive measures.³

Trait versus State Effects of Depressive Symptoms

Past average versus current depression severity in predicting neurocognition

—When run in separate models, higher levels of both current and past average depression severity predicted poorer attention/psychomotor processing speed (Table 2). When entered in the same model, the effect of past average severity remained significant, whereas that of current severity did not. Furthermore, past average depression predicted cognitive flexibility at a trend level, while current depression did not. When entered into the same model, neither current nor average depression significantly predicted flexibility.

However, analyses revealed that effects of average depression on cognitive flexibility were moderated by diagnosis. Follow-up analyses indicated that average depression severity predicted poorer cognitive flexibility among unipolar, but not bipolar, individuals, both alone and when controlling for current depression severity. Taken together with the fact that both groups showed impairment in flexibility relative to published norms (Figure 1), this suggests that bipolar participants show poor flexibility regardless of disorder severity, but only those unipolar participants with more severe depressive illness demonstrate poor cognitive flexibility.⁴

Within-individual effects of state symptom fluctuation—Within-individual change in depressive symptoms did not predict change in any cognitive domain, independently or in interaction with polarity (Table 3).

Stability of neurocognition—All four cognitive domains were stable over the course of six years (Table 3).

³When history of psychosis, lithium use, or antipsychotic use were entered as covariates, the effect of polarity on attention/ psychomotor processing speed dropped to trend. When controlling for antidepressant use, the effect of polarity remained significant. ⁴Lithium use was negatively associated with current depression, and antidepressant use was positively associated with current and past average depression. Nonetheless, when lithium, antidepressant, or antipsychotic use were included in analyses as covariates, the pattern of results for attention/psychomotor processing speed was similar, though slightly weaker, and the pattern for cognitive flexibility was identical.

Discussion

Although there is a growing literature on neurocognitive function in mood disorders, research to date has not clearly disentangled trait from state relationships between depression and neurocognition. Using data from a 26-year longitudinal study, we found that unipolar and bipolar individuals with higher past average depression severity showed greater decrements in attention/psychomotor processing speed and (among unipolar individuals) cognitive flexibility, over and above the effects of current symptom severity. In contrast, current symptom severity did not predict cognitive deficits independent of those associated with past average severity. Moreover, performance in all four cognitive domains was stable over the course of six years, and did not covary with within-individual symptom fluctuation.

These findings are consistent with a trait-like association between overall mood disorder severity and neuropsychological impairment, whereas state changes in depressive symptoms appear to have minimal influence on cognitive performance. This suggests that previous reports of cross-sectional relationships between current symptom severity and cognition (e.g., McDermott & Ebmeier, 2009) were not due to current clinical state, but instead reflect that individuals prone to more severe, chronic, or recurrent episodes show more severe stable cognitive deficits. This conclusion is consistent with findings that some individuals with mood disorders show cognitive impairments even during periods of euthymia (e.g., Kurtz & Gerraty, 2009).

There are at least two potential explanations for this trait-like association between disorder severity and cognitive impairment. First, impaired attention/psychomotor processing speed and cognitive flexibility may be pre-existing dimensional risk markers for mood disorders. Findings of cognitive impairment in healthy first-degree relatives of unipolar (Christensen, Kyvik, & Kessing, 2006) and bipolar probands (Bora, Yucel, & Pantelis 2009) provide some evidence for this possibility. Accordingly, our findings may be relevant to hypotheses regarding cognitive endophenotypes for mood disorders (Glahn, Bearden, Niendam, & Escamilla, 2004).

Alternatively, stable cognitive deficits may be enduring "scars" of mood episodes. That is, there may be a dose-response relationship between time spent depressed and cognitive impairment (Grant, Thase, & Sweeney, 2001). Levels of neurotrophic factors are lower and cortisol levels higher during depressive episodes than during euthymia, suggesting possible mechanisms for this scar effect (Lin, 2009; Steiger & Holsboer, 1997). Indeed, evidence indicates that cortisol levels are negatively correlated with cognitive performance in individuals with depression (Gomez et al., 2009).

Unipolar and bipolar participants showed generalized impairment across all cognitive domains, consistent with prior reports (Zakzanis, Leach, & Kaplan, 1998; Kurtz & Gerraty, 2009). Bipolar participants demonstrated poorer attention/psychomotor processing speed than unipolar participants, consistent with some previous studies (e.g., Borkowska & Rybakowski, 2001). However, this difference may be due to the greater use of antipsychotic drugs and lithium in the bipolar group, as both of these medications are associated with motor slowing (Goldberg & Chengappa, 2009). Moreover, most deficits were within one standard deviation of published norms–a level of decrement frequently seen even in healthy individuals (Binder, Iverson, & Brooks, 2009).

Several limitations should be taken into account in interpreting this study. First, the independent variables were related to potentially confounding factors such as history of psychosis and medication use. However, covariate analyses suggested that the significant effects of depression severity on cognition were not due to these factors. Second, it is possible that non-participation in follow-up visits was systematically related to clinical state,

such that individuals were less likely to participate when more severely depressed. This may have led to an underestimate of past average depression severity for some participants. Third, although our past average depression severity measure was sensitive to both chronicity and episode severity, it did not allow us to distinguish whether our findings were primarily driven by chronicity, episode severity, or both. Fourth, our study lacked a healthy comparison group. We were able to compare our participants to published test norms; however, differences among domains may reflect differences among norming samples. Finally, although we assessed multiple domains, our battery was not comprehensive; future studies might assess whether our findings generalize to additional domains such as working memory.

The study also had a number of strengths, stemming from the use of a 26-year prospective dataset. First, this allowed us to derive a measure of past average depression severity based on multiple prospective observations of depressive symptoms. This measure is likely more valid than a one-time retrospective report (Ben-Zeev, Young, & Madsen, 2009). Second, we were able to assess the stability of cognitive measures over multiple assessments, as well as the relationship between within-individual changes in depression and changes in cognition. We were therefore uniquely able to compare "trait-like" and "state-like" effects of depression severity on neurocognition in the same sample. Third, we were able to reclassify 16 individuals diagnosed as having unipolar depression at the index hospitalization as bipolar, based on identification of hypomanic or manic episodes at later follow-up points. It should be noted that we were only able to reclassify participants who experienced these episodes within a year prior to a follow-up point; this may have prevented us from reclassifying some bipolar individuals. Nonetheless, this stands in contrast to cross-sectional studies of unipolar depression, which cannot assess whether individuals initially diagnosed as "unipolar" go on to develop manic symptoms.

Using longitudinal data on the course of mood disorders over 26 years, we demonstrated that past average depression severity predicted impairment in attention/psychomotor processing speed among unipolar and bipolar individuals, and in cognitive flexibility among unipolar individuals, over and above effects of current symptoms. These findings highlight the need for treatments that target neurocognitive symptoms of mood disorders. Efforts to develop treatments for cognitive dysfunction in schizophrenia (e.g., the CNTRICS initiative; Carter & Barch, 2007) may therefore also be relevant to individuals with severe mood disorders.

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Sarapas et al.



Figure 1.

Neurocognitive performance among individuals with unipolar and bipolar depression. Values are z-scores representing performance compared to published norms (with standard errors). *p < .05, ***p < .001.

Table 1

Sample Characteristics

	Unipolar (n=42)	Bipolar (n=47)	Unipolar vs. Bipolar
Demographics			
Age at Assessment (SD)	39.2 (4.9)	38.7 (5.3)	t(87)=0.43, ns
Female	59.5%	53.2%	$\chi^2(1)=0.36$, ns
Caucasian	66.7%	76.6%	$\chi^2(1)=1.08$, ns
Years of Education (SD)	13.8 (1.8)	13.5 (1.9)	t(87)=0.71, ns
Clinical Variables			
Current Depression (SD)	14.4 (5.2)	15.4 (5.0)	<i>t</i> (87)=-0.94, <i>ns</i>
Past Average Depression (SD)	14.4 (3.8)	15.2 (4.3)	<i>t</i> (77)=-0.81, <i>ns</i>
History of Psychosis	23.8%	48.9%	$\chi^2(1)=6.00, p<.05$
Age at Index Hospitalization (SD)	22.9 (3.3)	22.8 (4.0)	<i>t</i> (87)=0.09, <i>ns</i>
Current Medication			
Antidepressant	23.8%	27.7%	$\chi^2(1)=0.17$, ns
Lithium	7.3%	32.6%	$\chi^2(1)=8.45, p<.01$
Antipsychotic	4.8%	19.1%	$\chi^2(1)=4.24, p<.05$
Any Psychotropic	38.1%	67.4%	$\chi^2(1)=7.57, p<.01$
Premorbid Intellectual Function			
WAIS Information, Scaled (SD)	11.9 (3.1)	12.2 (2.9)	t(84)=-0.45, ns

Note. "Current Depression" = depression at neuropsychological testing based on KAS depressed mood and behavior score (range: 9 to 36); "Past Average Depression" = mean KAS depression ratings from all follow-ups prior to neuropsychological testing.

Table 2

Total and Unique Effects of Current and Trait Depressive Symptoms on Cognitive Performance

	Current Depression		Past Average Depression	
Domain	Total Effect	Unique Effect	Total Effect	Unique Effect
Attention/Speed	26*	14	32 **	27*
Cognitive Flexibility ^a	14	06	20+	17
Unipolar			32 **	36*
Bipolar			03	03
Fluency	13	08	14	10
Verbal Memory	11	06	13	10

Note. Values are β coefficients from linear regression. "Total Effect" = effects of current (or past average) depression in separate linear models; "Unique Effect" = effects of current and past average depression when entered into same model. For each coefficient reported as significant in the table, the overall model was also significant.

^{*a*}Analyses revealed a polarity by past average depression interaction for Cognitive Flexibility, for both Total Effect [β =.42, t(70)=2.54 p<.05] and Unique Effect [β =.59, t(68)=2.61, p<.05]. Coefficients from analyses of simple slopes among unipolar and bipolar individuals are therefore presented.

 $^{+}p < .10,$

^rp<.05,

p < .01.

Table 3

Longitudinal Stability of Cognitive Domains and Relationship with Fluctuation in Depressive Symptoms

	Prediction by Change in Depression	Correlation T1 – T2
Attention/Speed	β=.20, <i>t</i> (36)=1.33, <i>ns</i>	<i>t</i> (39)=.59, <i>p</i> <.001
Cognitive Flexibility	$\beta =26$, $t(34) = -1.56$, ns	<i>1</i> (37)=.36, <i>p</i> <.05
Fluency	β =.13, t(36)=0.85, ns	<i>t</i> (39)=.75, <i>p</i> <.001
Verbal Memory	β=.14, <i>t</i> (33)=0.14, <i>ns</i>	<i>t</i> (36)=.73, <i>p</i> <.001