Research Article

Effectiveness of a Questionnaire Measure of Executive Function in Predicting Treatment Outcomes for Anxiety and Depression Following CBT -

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ABSTRACT

Previous research shows deficits in Executive Function (EF) in patients with anxiety and depression. Recent studies have shown that EF measured by neuro-imaging and neuropsychological tests predicts treatment outcomes for depression, but it is unclear whether they predict outcomes for anxiety. Neuro-imaging and neuropsychological tests are effective but intensive procedures that may not always be accessible to clinicians. Previous research has explored the viability of questionnaire measures of EF. A previous study suggests that the Revised Dysexecutive Questionnaire (DEX-R) predicts concurrent depression and anxiety; however, it is unclear how comorbidity influenced these results. The purpose of the current study was to investigate whether a questionnaire measure of EF could predict concurrent depression and anxiety and well as outcomes following treatment. A total of 206 psychiatric outpatients with major depression or anxiety disorders completed the DEX-R prior to Group Cognitive Behavioral Therapy (GCBT). They also completed anxiety and depression scales at pre-and post-treatment. Executive dysfunction predicted symptom severity for pre-treatment anxiety after controlling for comorbid depression, and for pre-treatment depression after controlling for comorbid anxiety. Symptom severity in anxiety was predicted by specific executive deficits in inhibition; symptom severity in depression was predicted by executive problems with volition and social regulation. DEX-R significantly predicted post-treatment symptoms of anxiety but not depression following treatment in GCBT. It was concluded that EF deficits are associated with both anxiety and depressive disorders and predict responsiveness to treatment for anxiety patients. Screening of psychiatric patients for EF and, where indicated, incorporation of neurocognitive training strategies into therapy, may improve treatment outcomes.

Keywords: Executive Function; Depression; Mood Disorders; Anxiety Disorders; CBT

INTRODUCTION

Executive Function (EF) is the higher order, self-regulatory, cognitive processes for monitoring and control of thought and behaviour, associated with the frontal lobes [1,2]. Executive deficits could result from damage to the frontal lobe, or disruptions to the connective pathways mediated by the frontal lobes [3]. Over the past two decades, research has investigated apparent similarities between symptoms of executive dysfunction and symptoms of clinical anxiety and depression. According to the Diagnostic and Statistical Manual of Mental Disorders 5th Edition [4], individuals with depression and anxiety typically present with deficits in motivation/volition, problem solving, planning, concentration, emotional stability, psychomotor disturbances and perseverative thoughts; all of these are fundamental deficits in EF [5,6]. Depressed individuals frequently exhibit hypoactivity in cortical regions of the frontal lobes and prefrontal and anterior cingulate, which has been linked with the lack of volition (or planned decisive action) that frequently accompanies depression [7]. Anxiety disorders have sometimes been characterized by problems with inhibition which is defined as the inability to inhibit a prepotent response [8]. For example, panic disorder is characterized by problems with ignoring stimuli [9], as is Obsessive Compulsive Disorder (OCD). Recent meta-analyses have confirmed that both anxiety and depression are associated with a wide range of significant deficits in EF.

A large meta-analysis [10] compared participants with major depressive disorder with healthy controls on neuropsychological measures of EF. Depression was reliably associated with significant impairments in all domains of EF measured ($d = 0.45$–0.58), including inhibition (of a habitual response), shifting (adapting to different sets of rules), updating (of relevant information in working memory), verbal working memory, visuospatial working memory, planning, and verbal fluency. Associations could not be accounted for by processing speed. Deficits were greater in patients with more severe depression, and those taking psychotropic medications. Some evidence suggested there were greater impairments in tasks requiring inhibition than other domains. Other meta-analyses have confirmed that major depression is associated with deficits across numerous domains of EF [11,12].

Recent meta-analyses have examined the relationship of EF with specific anxiety disorders. Snyder, Raiser and Heller [13] found that, compared with healthy controls, a diagnosis of OCD was associated with broad range of EF impairments including inhibition, shifting, updating, verbal and visuospatial working memory or planning (with most effect sizes d between 0.3 and 0.5). None of these associations could be accounted for by general motor slowness or by comorbid depression. Depression was a significant moderator on one task measure, the Stroop interference task; samples with comorbid depression had *less* severe problems inhibiting an established response on this task. Another meta-analysis [14] found that patients with OCD were significantly impaired in EF and a wide range of other cognitive domains, independent of symptom severity, medication status or co-morbid disorders. There seemed to be a larger deficit in planning ability than other domains.

Scott et al. [15] conducted a meta-analysis of 60 studies on neurocognitive deficits associated with Post-Traumatic Stress Disorder (PTSD). Compared to healthy controls, patients with PTSD had significant deficits in a wide range of cognitive abilities including EF ($d = .45$). A systematic review [16] of the role of EF in PTSD and, across 18 studies, found that, in comparison with trauma-exposed controls and healthy controls, participants with PTSD showed significantly impaired EF. There were greater impairments in EF for participants with comorbid depression.

A systematic review of studies of panic disorder [17] did not find support for the presence of an impairment in EF, nor other areas of cognitive functioning; however, the findings were inconclusive due to small sample sizes in studies. Isolated studies investigating EF deficits associated with other anxiety disorders report mixed results. For instance, one study of Generalized Anxiety Disorder in later life [18], reported no deficits in EF associated with anxiety unless there was comorbid depression. Another study [19] reported deficits in EF associated with generalized social anxiety disorder. In summary, despite some mixed results, recent large meta-analyses confirm significant deficits in executive deficits associated with major depression and anxiety diagnoses of OCD and PTSD. Given its relationship with anxiety and depression, attention has turned to the role of executive dysfunction in predicting responsiveness to treatment.

Both pharmacological and psychological interventions have been demonstrated to be effective in treating depression [20-22] and anxiety disorders [23]. There have been two meta-analyses of

predictors of treatment response for depression. A meta-analysis of predictors of treatment outcomes (including pharmacological, psychological and care management) for depression [24], found that poor treatment outcomes were predicted by severity of baseline depression, EF deficits and presence of co-morbid anxiety. There were mixed results about whether the domain of response inhibition was a significant predictor. Another meta-analysis [25] examined whether six domains of EF extracted from eight studies predicted short-term effectiveness of pharmacological interventions. Only performance related to the planning and organisation tasks predicted treatment response; other domains including response inhibition did not. This is consistent with early research linking depression with problems in volition [7]. Using positron emission tomography with inpatients with major depression, Mayberg et al. [26] found that responsiveness to antidepressant medication was predicted by metabolism in the rostral anterior cingulate area of the brain which is deeply involved in action regulation [27]. Gyurak et al. [28] used magnetic resonance imaging to monitor frontoparietal activation in patients with major depression, and found that frontoparietal activation, during the response inhibition task (but not selective attention or working memory tasks) predicted later remission with antidepressant treatment. Overall these studies suggest that inhibition may be central in predicting treatment effectiveness for depression, and that volition (planning and organisation) may also play a role.

At present, research results are inconclusive regarding whether EF predicts treatment outcomes for anxiety disorders. Two studies of generalized anxiety disorder in later life found that pre-treatment EF did not predict treatment outcome, although changes over time in EF were associated with reductions in anxiety [29,30]. Johnco, Wuthrich and Rapee [31] found that pre-treatment cognitive flexibility did not predict outcomes in anxiety or depression following CBT for older participants. Similarly, EF did not predict treatment outcomes following CBT for participants with OCD [32], although non-recovered patients had lower pre-treatment social functioning than recovered patients. In summary, research has not been successful at predicting treatment outcomes for anxiety from pre-treatment EF.

The present study investigates whether EF predicts treatment outcomes following CBT for outpatients with diagnoses of depression and anxiety. Most studies of EF to date have used neurological imaging or neuropsychological tests of EF. Although highly effective, these intensive assessments may not always be available to clinicians in all settings. Previous research has investigated the utility of psychometric questionnaires including The Dysexecutive Questionnaire (DEX) [33,34] to measure executive dysfunction. Shaw, Oei and Sawang [35] investigated the factor structure of the DEX with community, depressed, anxious, and neurologically impaired samples and found that that a factor structure including factors of Inhibition, Volition, and Social Regulation, was superior to other factor models (DEX-R). Volition is the formation of intention and self-awareness [2]. Inhibition is the ability to inhibit a prepotent response in order to make a less automatic but task-relevant response [37]. Social regulation reflects awareness and concern for social rules [35]. Oei, Shaw and Healy [34] compared EF using the DEX-R in neurological, psychiatric patients and general community members. They found that patients with anxiety had greater EF deficits than all other groups and greater deficits in inhibition and volition; patients with depression reported greater executive dysfunction in volition than the community group. However, given the high co-morbidity between depression and anxiety disorders (50 to 60%) [36], it is

unclear how comorbidity influenced results reported by Oei et al. [34].

The current study investigates whether EF, measured by DEX-R, predicts treatment outcomes for anxiety and depression following CBT for outpatient with diagnoses of major depression or an anxiety disorder. A further aim is to investigate whether EF concurrently predicts symptom severity in depression and anxiety after controlling for comorbidity. We hypothesized that 1) after controlling for comorbidity, that executive dysfunction, would concurrently predict both anxiety and depression; 2) lower levels of pre-treatment executive dysfunction would predict better treatment outcomes for both anxiety and depression 3) that deficits in pre-treatment inhibition would predict both concurrent and post-treatment outcomes for anxiety and 4) that deficits in pre-treatment volition would predict both concurrent and post-treatment outcomes for user.

METHOD

Participants

The sample consisted of 206 outpatients with a primary diagnosis of either anxiety (71.4%) or major depression (28.6%) referred to a private psychiatric hospital for Group Cognitive Behavioral Therapy (GCBT). Inclusion criteria were a single primary diagnosis of either anxiety or depression. No patients with a diagnosis of psychosis were included. All diagnoses were made by psychiatrists according to the DSM-IV-TR [38] prior to referral. No psychiatric patient was excluded on the basis of medication status. Anxiety diagnoses included panic disorder (39.3%), generalized anxiety disorder (20.9%), Post-Traumatic Stress Disorder (PTSD) (10.1%), and other anxiety disorders (1%). They were 64.5% female and 35.5% male with an average age of 43.5 years. For 94.9% of participants, English was the primary language. Post-treatment data was available for 144 outpatients who attended the final CBT session (69.9%). There were no significant differences between completers and non-completers on age (F [1, 204] = 1.97, p = .162), gender (F [1, 204] = 0.79, p = .376) educational level (F [1, 204] = 0.27, p = .602), nor pre-treatment anxiety (F [1, 204] = 1.59, p = .209), depression (F [1, 204] = 0.94, p = .335 or EF DEX-R (F [1, 204] = 2.37, p = .125).

Measures

All measures were written in English.

Dysexecutive functioning: EF was assessed pre-treatment using the 15-item revised Dysexecutive Questionnaire (DEX-R) [35]. Item responses range from 0 (*never*) to 4 (*very often*) with higher scores indicating greater dysfunction. Total DEX-R had a Cronbach's α of .87. EF subscales of Inhibition (e.g. ability to inhibit responses, confabulation, impulsivity), Volition (e.g. planning problems, apathy and lack of drive) and Social Regulation (e.g. lack of concern for social rules) were calculated after Shaw et al. [35]. Internal consistency was good for Inhibition (α = .79) and Volition (α = .81). Internal consistency as measured by Cronbach's alpha was low for Social Regulation (α = .56). However, given low scores on Cronbach's alpha are common for scales with few items, we also calculated mean inter-item correlation for Social Regulation (r = .39); this was within the recommended range [39].

Depression and anxiety symptomatology: Two well established and validated scales were utilized: the Zung Self-Rating Depression Scale (SDS) [40] and the Beck Anxiety Inventory (BAI) [41]. The Zung SDS has demonstrated acceptable reliability, and predictive

validity as a screening measure for diagnosis of depression [42]. It, correlates well (0.69) with the treating physician's global ratings of depressed outpatients during treatment [43]. The BAI is a well-established measure demonstrated to have sound psychometric properties including reliability, concurrent and construct validity [41]. For the current sample, the SDS had a Cronbach's α of .84 for the pre-treatment scores and .90 for post-treatment scores; Cronbach's α for the BAI was .94 for both pre-treatment scores and post-treatment scores.

Procedure: Ethical clearance was received from The University of Queensland and Toowong Private Hospital. As in our previous papers [34,35], participants were recruited from an outpatient psychiatric unit at the Toowong Private Hospital. All patients were referred to the CBT Unit for GCBT and gave informed written consent to participation in the study prior to their involvement. The GCBT programs have been described in previous publications [44-46]. Each group involved, on average, eight patients. Treatment consisted of eight 3.5-hour sessions over 4 weeks. The manualized GCBT program for anxiety comprised psycho-education, self-monitoring, relaxation exercises, cognitive restructuring, interoceptive and in vivo exposure, problem solving, maintenance and relapse prevention. The manualized GCBT program for depression covered psycho-education, self-monitoring, relaxation, behavioral activation, cognitive monitoring and restructuring, problem solving, maintenance and relapse prevention. The anxiety and depression programs are documented in unpublished workbooks by Oei at Toowong Private Hospital. All groups were facilitated by a clinical psychologist with over 20 years of GCBT experience and a psychiatric nurse with extensive experience in GCBT. Participants completed DEX-R, anxiety and depression scales prior to treatment and anxiety and depression scales after GCBT.

Statistical Analyses

We first conducted a MANOVA to compare pre-treatment differences on anxiety, depression and DEX-R scores for patients with an anxiety diagnosis compared with patients with a depression diagnosis; this was then repeated substituting DEX-R subscales. To determine whether executive dysfunction could predict symptom severity in anxiety and depression after taking into account comorbidity, we conducted a series of stepwise Hierarchical Multiple Regression Analyses (HMR) through SPSS, using either pre-treatment anxiety or pre-treatment depression as the outcome variable, as described by Tabachnick and Fidell [47]. When pre-treatment anxiety was the outcome variable, the order of entry of predictor variables was as follows: Step 1 pre-treatment depression; Step 2 primary diagnostic category (anxiety versus depression); Step 3 executive dysfunction; Step 4 two-way product terms involving executive dysfunction and diagnostic category. When pre-treatment depression was the outcome variable, pre-treatment depression was substituted at Step 1, and pre-treatment anxiety was substituted at Step 2. We centred each variable before calculating product terms to avoid problems of multicollinearity associated with utilizing product terms in regression [48].

We used a parallel procedure to test if pre-treatment DEX-R scales predicted treatment outcomes in depression and anxiety following GCBT. We first checked for change in depression and anxiety scores between pre and post-treatment using ANOVAs. We used HMR to test whether DEX-R scales would predict post-treatment anxiety after controlling for pre-treatment anxiety and primary diagnosis, and whether DEX-R scales would predict post-treatment depression after

controlling for pre-treatment depression and primary diagnosis.

RESULTS

Preliminary Analyses

Missing values (< 5% for each variable) were scattered randomly throughout the data so did not pose any methodological problems [49]. Several DEX-R scales, depression and anxiety scales had a significant positively skew ($Z > 1.96$) and were kurtosed, which is not uncommon with clinical data [50]. However multiple regression assumes normality [47], so we transformed the data. Analyses of transformed data yielded the same pattern of results as the untransformed data, so the untransformed analyses were reported.

Comparison of Anxious and Depressed Groups on Clinical Measures

We conducted MANOVAs to check differences in pre-treatment scores for patients with an anxiety diagnosis compared with patients with depression. The anxious group had higher scores than the depressed group on anxiety ($F [1, 203] = 58.20$, $p < .001$), depression ($F [1, 203] = 24.70$, $p < .001$), total DEX-R ($F [1, 203] = 25.05$, $p < .001$) and DEX-R subscales of Inhibition ($F [1, 203] = 17.46$, $p < .001$), Volition ($F [1, 203] = 27.37$, $p < .001$), but not Social Regulation ($F [1, 203] = 0.70$, $p = .405$).

Executive Dysfunction as a Predictor of Pre-Treatment Anxiety

Table 1: Means, Standard Deviations and Inter-correlations between variable.

Variables	Mean (SD)	n	1.	2.	3.	4.	5.	6.	7.
1. Pre-treatment anxiety	23.49 (13.25)	206	-						
2. Post-treatment anxiety	15.79 (12.21)	143	.62 ***	-					
3. Pre-treatment depression	52.73 (9.48)	206	.61 ***	.56 ***	-				
4. Post-treatment depression	46.17 (11.05)	144	.45 ***	.70 ***	.69 ***	-			
5. DEX-R total score	24.39 (10.30)	206	.52 ***	.45 ***	.54 ***	.48 ***	-		
6. DEX-R Inhibition	8.18 (4.61)	206	.47 ***	.28 **	.36 ***	.27 **	.85 ***	-	
7. DEX-R Volition	13.76 (5.82)	206	.50 ***	.47 ***	.64 ***	.55 ***	.91 ***	.59 ***	-
8. DEX-R Social Regulation	2.46 (1.97)	205	.15 *	.23 **	.09	.20 **	.57 ***	.36 ***	.40 ***

* $p < .05$ ** $p < .01$ *** $p < .001$
Table 1 shows means, standard deviations and bivariate correlations. There were significant correlations between DEX-R scales, anxiety and depression measures.

Table 2: DEX-R as a Predictor of Concurrent Anxiety.

Step	Predictor Variables Added	Anxiety Score					
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Depression Score	0.57 [0.41; 0.74]	0.08	.41 ***	.37 ***	.38	123.08 ***
2.	Diagnostic Group	-9.49 [-12.82; -6.16]	1.69	-.32 ***	.45 ***	.08	30.87 ***
3.	DEX-R	0.24 [0.09; 0.39]	0.08	.19 **	.48 ***	.03	11.59 **
4.	DEX-R x Diagnosis	-0.43 [-0.75; -0.11]	.16	-.14 *	.50	.02	6.85 *

* $p < .05$ ** $p < .01$ *** $p < .001$

Tables 2 and 3 show HMR, using total DEX-R to predict concurrent anxiety, after controlling for depression. Pre-treatment depression scores at Step 1 and diagnostic category at Step 2 both predicted pre-treatment anxiety scores ($p < .001$). At Step 3, inclusion of the DEX-R total scores improved prediction of pre-treatment anxiety scores ($p = .001$). There were further improvements to the model with inclusion of 2-way product terms at Step 4 ($p = .010$). The variables which accounted for unique variance in anxiety scores were depression scores ($\beta = .41$, $p < .001$), diagnostic category ($\beta = -.32$, $p < .001$), DEX-R total scores ($\beta = .19$, $p = .002$) and DEX-R X Diagnosis ($\beta = -.41$ $p = .010$). So, higher anxiety scores were predicted by an anxiety diagnosis, higher scores on DEX-R and the interaction term. Figure 1 graphs the significant two-way interaction. There is a steeper gradient for the relationship between DEX-R and anxiety for patients with an anxiety diagnosis than for patients with a depression diagnosis, meaning that higher levels of DEX-R predict proportionately higher levels of anxiety for anxiety patients than for depression patients.

Table 3 shows that when DEX-R sub-scales (instead of total DEX-R) were entered at Step 3, this still significantly improved prediction of concurrent anxiety (F [3, 199] = 6.42, $p < .001$) but inclusion of product terms at Step 4 made no further improvement (F [3, 196] = 2.10, $p = .102$). The variables accounting for unique variance in pre-treatment anxiety scores were depression scores ($\beta = -.45$, $p < .001$), diagnostic category ($\beta = .41$, $p < .001$) and DEX-R Inhibition ($\beta = .23$, $p = .001$). So, higher pre-treatment anxiety scores were predicted by an anxiety diagnosis and higher scores on DEX-R Inhibition.

Executive Dysfunction as a Predictor of Pre-Treatment Depression

Table 4 shows the HMR using total DEX-R to predict depression scores after controlling for anxiety scores. At Step 1, anxiety scores predicted pre-treatment depression ($p < .001$) but diagnostic category at Step 2 did not ($p = .417$). Inclusion of whole-scale DEX-R at Step 3 improved the model ($p < .001$) but inclusion of product terms at Step 4 made no further improvement ($p = .358$). The variables which accounted for unique variance in depression scores were anxiety scores ($\beta = .46$, $p < .001$) and DEX-R total score ($\beta = .31$, $p < .001$). So, higher pre-treatment depression scores were predicted by higher scores on DEX-R.
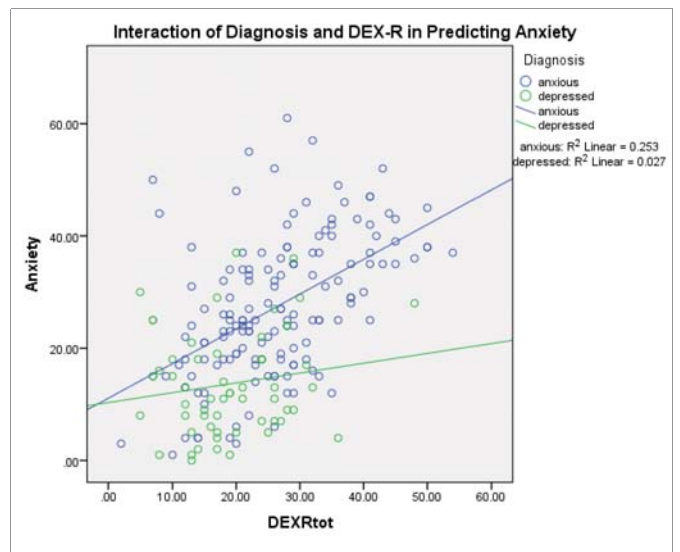

Figure 1: Interaction of DEX-R and Diagnostic Category in Prediction of Anxiety

Table 4: DEX-R as a Predictor of Concurrent Depression.

Step	Predictor Variables Added	Depression Score					
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Anxiety Score	0.33 [0.23; 0.42]	0.05	.46 ***	.37 ***	.38	123.08 ***
2.	Diagnostic Group	0.15 [-2.57; 2.86]	1.38	.01	.37 ***	.00	0.66
3.	DEX-R	0.28 [0.17; 0.40]	0.06	.31 ***	.44 ***	.07	24.15 ***
4.	DEX-R x Diagnosis	0.12 [-0.13; 0.37]	.13	.05	.44	.00	0.89
* p < .05		** p < .01		*** p < .001			

Table 5 shows the HMR for depression scores using the DEX-R subscales. Inclusion of DEX-R subscales at Step 3 significantly improved prediction of pre-treatment depression ($p < .001$), but inclusion of the product terms at Step 4 made no further improvement ($p = .176$). The variables which accounted for unique variance in depression scores were DEX-R Volition ($\beta = .58$, $p < .001$), anxiety score ($\beta = .40$, $p < .001$), and DEX-R Social Regulation ($\beta = .13$, $p = .014$). So, higher depression scores were predicted by higher scores in DEX-R Volition, and lower scores on DEX-R Social Regulation.

Evaluation of GCBT Treatment Outcomes

We tested for reductions in depression and anxiety symptomatology following GCBT. Means and standard deviations for pre and post measures for anxiety and depression are displayed in Table 1. There were significant reductions in anxiety (t [142] = 8.66, $p < .001$), and depression (t (143) = 7.60, $p < .001$). As there was no control group in the clinical setting, we used a pre-post bias correction to calculate effect size recommended by Morris and De Shon [51], resulting in medium effect sizes for anxiety ($d = 0.53$) and depression, ($d = 0.43$).

DEX-R Scales as Predictors of Treatment Outcomes for Anxiety

Table 6 and 7 show HMR using DEX-R scales to predict post-treatment anxiety, after controlling for pre-treatment anxiety.

Table 3: DEX-R Subscales as Predictors of Concurrent Anxiety.

Step	Predictor Variables Added	Anxiety Score					
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Depression Score	0.62 [0.43; 0.81]	0.10	.45 ***	.37 ***	.38	122.34 ***
2.	Diagnostic Group	-9.98 [-12.39; -5.58]	1.73	-.31 ***	.45 ***	.08	30.48 ***
3.	DEX-R Inhibition	0.65 [0.29; 1.00]	0.18	.23 ***	.49 ***	.05	6.42 ***
	DEX-R Volition	-0.06 [-0.42; 0.30]	0.18	-.03			
	DEX-R Social Regulation	0.09 [-0.66; 0.84]	0.38	.01			
4.	DEX-R Inhibition x Diagnosis	-0.23 [-1.09; 0.63]	0.44	-.03	.50 ***	.02	2.10
	DEX-R Volition x Diagnosis	-0.35 [-1.03; 0.32]	0.34	-.07			
	DEX-R Social Reg x Diagnosis	-0.98 [-2.67; 0.72]	0.86	-.06			
* p < .05		** p < .01		*** p < .001			

For both regressions, inclusion of pre-treatment anxiety at Step 1 significantly contributed to the prediction of post-treatment anxiety ($p < .001$) but inclusion of diagnostic group at Step 2 did not ($p = .363$). Table 6 shows that inclusion of DEX-R total score at Step 3 improved prediction of post-treatment anxiety ($p = .011$), but inclusion of the interaction term at Step 4 did not ($p = .993$). At Step 4, pre-treatment anxiety ($\beta = .50$, $p < .001$), and DEX-R total score ($\beta = .20$, $p = .011$), accounted for significant unique variance in post-treatment anxiety. That is, better treatment outcomes in anxiety were predicted by lower levels of pre-treatment anxiety and lower DEX-R total score. Table 7 shows that when DEX-R subscales were used instead of DEX-R total score at Step 3, DEX-R subscales improved prediction of pre-treatment anxiety ($p = .003$), but inclusion of product terms at Step 4 did not. Variables accounting for unique variance in post-treatment anxiety were pre-treatment anxiety ($\beta = .48$, $p < .001$), DEX-R Volition ($\beta = .24$, $p = .008$) and DEX-R Social Regulation ($\beta = .16$, $p = .020$). That is, better treatment outcomes for anxiety were predicted by lower levels of pre-treatment anxiety, DEX-R Volition and DEX-R Social Regulation.

DEX-R Scales as Predictors of Treatment Outcomes for Depression

Tables 8 and 9 show HMR, using DEX-R scales, to predict post-treatment outcomes for depression scores. For both regressions, inclusion of pre-treatment depression at Step 1 significantly

Table 5: DEX-R Subscales as Predictor of Concurrent Depression.

| Step | Predictor Variables Added | Depression Score | | | | | |
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Anxiety Score	0.29 [0.20; 0.38]	0.04	.40 ***	.37 ***	.38	122.34 ***
2.	Diagnostic Group	1.13 [-1.34; 3.60]	1.25	.05	.37 ***	.00	0.67
3.	DEX-R Inhibition	-0.19 [-0.44; 0.06]	0.13	-.09	.55 ***	.18	26.95 ***
	DEX-R Volition	0.94 [0.74; 1.15]	0.11	.58 ***			
	DEX-R Social Regulation	-0.76 [-1.26; -0.27]	0.25	-0.16 **			
4.	DEX-R Inhibition x Diagnosis	0.48 [-0.10; 1.06]	0.30	.10	.55 ***	1.6	1.67
	DEX-R Volition x Diagnosis	0.06 [-0.40; 0.53]	0.24	.02			
	DEX-R Social Reg x Diagnosis	-0.97 [-2.12; 0.18]	0.58	-.09			

* $p < .05$ ** $p < .01$ *** $p < .001$

Table 6: DEX-R as a Predictor of Post-Treatment Anxiety.

| Step | Predictor Variables Added | Post Treatment Anxiety Score | | | | | |
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Pre Treatment Anxiety	0.49 [0.33; 0.65]	0.08	.50 ***	.38 ***	.38	81.80 ***
2.	Diagnostic Group	-1.09 [-5.70; 3.52]	2.33	-.04	.38 ***	.00	0.83
3.	DEX-R	0.25 [0.06; 0.44]	0.10	.20 *	.40 ***	.03	6.65 *
4.	DEX-R x Diagnosis	0.00 [-0.43; 0.43]	0.22	.00	.40 ***	.00	0.00

* $p < .05$ ** $p < .01$ *** $p < .001$

Table 7: DEX-R Subscales as Predictors of Post-Treatment Anxiety.

| Step | Predictor Variables Added | Post Treatment Anxiety Score | | | | | |
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Pre Treatment Anxiety Score	0.47 [0.31; 0.63]	0.08	.48 ***	.38 ***	.38	81.80 ***
2.	Diagnostic Group	-1.54 [-6.12; 3.05]	2.32	-.06	.38 ***	.00	0.83
3.	DEX-R Inhibition	-0.26 [-0.71; 0.20]	0.23	-.09	.43 ***	.06	4.82 **
	DEX-R Volition	0.49 [0.13; 0.85]	0.18	.24 **			
	DEX-R Social Regulation	1.11 [0.18; 2.04]	0.47	.16 *			
4.	DEX-R Inhibition x Diagnosis	0.84 [-0.21; 1.89]	0.53	.13	.43 ***	.02	1.39
	DEX-R Volition x Diagnosis	-0.47 [-1.26; 0.33]	0.40	-.10			
	DEX-R Social Reg x Diagnosis	-0.96 [-3.07; 1.15]	1.07	-.07			

* $p < .05$ ** $p < .01$ *** $p < .001$

Table 8: DEX-R as a Predictor of Post-Treatment Depression.

| Step | Predictor Variables Added | Post Treatment Depression Score | | | | | |
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Pre-Treatment Depression Score	0.71 [0.53; 0.88]	0.09	.61 ***	.47 ***	.48	122.53 ***
2.	Diagnostic Group	-1.86 [-5.51; 1.79]	1.84	-.08	.47 ***	.00	0.99
3.	DEX-R	0.12 [-0.06; 0.29]	0.09	.10	.48 ***	.01	2.14
4.	DEX-R x Diagnosis	-0.16 [-0.52; 0.20]	.18	-.06	.48 ***	.00	0.74

* $p < .05$ ** $p < .01$ *** $p < .001$

Table 9: DEX-R Subscales as Predictors of Post-Treatment Depression.

| Step | Predictor Variables Added | Post Treatment Depression Score | | | | | |
		B [95% CI]	SEB	β	R² (adj)	Δ R²	F Change
1.	Pre Treatment Depression Score	0.71 [0.50; 0.92]	0.10	.61 ***	.47 ***	.48	122.53 ***
2.	Diagnostic Group	-2.39 [-6.14; 1.35]	1.89	-.10	.47 ***	.00	0.99
3.	DEX-R Inhibition	-0.16 [-0.56; 0.23]	0.20	-.06	.49 ***	.03	2.22
	DEX-R Volition	0.15 [-0.25; 0.55]	0.20	.08			
	DEX-R Social Regulation	0.88 [0.07; 1.69]	0.41	.15 *			
4.	DEX-R Inhibition x Diagnosis	-0.23 [-1.15; 0.69]	0.47	-.04	.48 ***	.01	0.55
	DEX-R Volition x Diagnosis	-0.06 [-0.76; 0.63]	0.35	-.01			
	DEX-R Social Reg x Diagnosis	-0.76 [-2.58; 1.06]	0.92	-.06			

* $p < .05$ ** $p < .01$ *** $p < .001$

contributed to prediction of post-treatment depression ($p < .001$) but inclusion of diagnostic group at Step 2 did not ($p = .322$). Inclusion of neither total DEX-R nor DEX-R subscales at Step 3 significantly improved the model, nor did inclusion of the interaction terms at Step 4. However, examination of β-weights reveals that DEX-R Social Regulation predicted significant unique variance in post-treatment depression ($\beta = .15$, $p = .034$), as did pre-treatment depression ($\beta = .61$, $p < .001$). That is better treatment outcomes in depression were

predicted by lower pre-treatment scores in depression and social regulation problems.

DISCUSSION

This study aimed to test whether EF, as measured by DEX-R, was related to concurrent symptom severity of depression and anxiety after controlling for comorbidity, and whether it could predict treatment outcomes following GCBT for outpatients with depression and anxiety disorders. Consistent with our hypothesis and previous research [10,13], executive dysfunction predicted concurrent symptom severity in anxiety after controlling for comorbid depression, and concurrent depression after controlling for comorbid anxiety. Analyses using subscales were consistent with hypotheses that, after controlling for comorbidity, problems in inhibition would be concurrently associated with anxiety, and problems with volition associated with depression.

Consistent with our predictions, DEX-R significantly predicted reductions in anxiety following participation in a GCBT. However, contrary to predictions, DEX-R did not predict treatment outcomes for depression. The prediction of outcomes from DEX-R subscales also differed from hypotheses. We will first discuss the pattern DEX-R of subscales in the prediction of concurrent anxiety and depression, then in the prediction of treatment outcomes.

Consistent with our hypothesis and previous literature [9], after controlling for comorbid depression, severity of pre-treatment anxiety was predicted by executive deficits in inhibition. Also, consistent with our hypotheses, after controlling for comorbid anxiety, depression scores were predicted by executive problems with volition; this is consistent with previous reports that patients with depression have difficulties in organization and initiating goal-directed activities [7]. This suggests that findings by Oei et al. [34] that patients with anxiety had greater deficits in Volition as well as Inhibition may have been due to comorbid depression. The current study shows that, after controlling for comorbidity, problems with volition are associated with depression but not anxiety.

Additional to our hypotheses, after controlling for comorbid anxiety, severity of pre-treatment depression was also predicted by lower scores on social regulation. Social Regulation reflects a lack of concern in social situations. So, higher levels of depression symptomatology were predicted by higher levels of social concern, rather than indifference to social rules typical of executive dysfunction. There is little previous literature examining the relationship between concern for social rules and depression. Oei et al. [34] reported no significant differences in Social Regulation between depression patients and a community sample. Perhaps the relationship between depression and high social concern in this study reflects social discomfort. Zahn et al. [52] found that depression patients in remission retrospectively reported high levels of self-disgust, guilt and shame. Collazzoni et al. [53] found that humiliation differentiated clinically depressed subjects, from a carefully matched non-clinical sample. It is possible then that the relationship between social regulation and depression in this study may reflect social discomfort and embarrassment.

In this study higher pre-treatment anxiety scores were predicted, not only by higher scores on DEX-R, but also by the interaction of DEX-R and diagnosis. That is, higher levels of executive dysfunction predicted proportionately higher levels of anxiety for anxiety patients than for depression patients. This pattern of results can be interpreted through considering differences in scores on clinical measures for patients with a primary diagnosis of anxiety versus depression. Patients with an anxiety diagnosis had higher scores than depression patients, on anxiety, depression, DEX-R total score, and problems with Inhibition. This suggests that patients with a primary diagnosis of anxiety were more likely to have a secondary diagnosis of depression than vice versa. This is consistent with previous findings that depression can develop as a secondary issue to anxiety [54] and to mild to moderate traumatic brain injury [55].

Analyses using DEX-R scales to predict treatment outcomes in anxiety showed some departures from hypotheses. Consistent with hypotheses, total DEX-R scores predicted treatment outcomes in symptom severity for anxiety. However, contrary to predictions, and previous literature linking inhibition problems with anxiety [9], Volition and Social Regulation rather than Inhibition predicted poorer treatment outcomes in anxiety. Why might this be? Problems with response inhibition are common for patients with anxiety. However, CBT teaches patients with anxiety to better manage their automatic responses. So perhaps GCBT helped treat problems with inhibiting responses common to anxiety and executive dysfunction, by enabling patients to respond in more flexible ways to aversive stimuli. In the current sample, depression was higher for patients with an anxiety diagnosis than for patients with a depression diagnosis. Perhaps comorbid depression associated with volition problems predicted poor outcomes[1]. Patients with comorbid depression may not have had issues related to depression addressed within the time frame given that the GCBT program was intended primarily to address issues associated with anxiety. Deficits in planning and initiating associated with lower scores in in Volition may have been associated with poorer participation in group activities and homework for these outpatients, which affected treatment outcomes.

Regressions using DEX-R scales to predict treatment outcomes in depression also showed some unexpected results. Contrary to hypotheses, neither total DEX-R score, nor problems with Volition predicted post-treatment depression scores. The sample for this study included psychiatric outpatients with major depressive disorder. As lack of volition is a key symptom of depression [7], patients would have had major deficits in volition. Therefore, eight sessions of GCBT over four weeks of GCBT may have been insufficient to produce significant change, which may have effected prediction of treatment outcomes by DEX-R. Although this explanation seems plausible, further vigorous research is needed. The only DEX-R subscale which predicted treatment outcomes in depression was Social Regulation, with poorer treatment outcomes predicted by greater deficits in social awareness characteristic of executive dysfunction. Perhaps a lack of concern about group rules impeded participation in GCBT.

This study investigated executive dysfunction measured by the DEX-R questionnaire with psychiatric outpatients diagnosed with anxiety or depression. Executive dysfunction predicted both concurrent anxiety and depression after controlling for comorbidity. Problems in inhibiting responses predicted concurrent anxiety; problems with Volition and Social Regulation predicted concurrent depression. Greater EF problems in Volition and Social Regulation predicted poorer treatment outcomes for anxiety. Strengths of the study included a real-world sample and a longitudinal data set. Weaknesses included reliance on self-report measures, which are limited by individuals' insight into their own abilities. A further

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1 Indeed when regressions were repeated controlling for depression as well as anxiety at Step 1, Volition no longer predicted treatment outcomes.

limitation is that no control group was utilized.

The findings suggest that screening patients with anxiety for executive dysfunction may predict receptivity to CBT. Social regulation was relevant for treatment outcomes for both patients with depression, and patients for anxiety. Checking social regulation scores prior to CBT may identify individuals who may have difficulty participating in a group and may benefit more from individual therapy. Individuals with executive dysfunction may benefit from incorporation of neurocognitive training strategies along with traditional approaches in order to improve therapeutic gains. Neurocognitive training has been found to improve EF and social ability in brain injured samples and schizophrenic patients [56,57]. Future research could test the effectiveness of incorporating neurocognitive training into CBT for individuals with anxiety diagnoses following pre-treatment screening for EF deficits.

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