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The relations of cognitive, behavioral, and physical activity variables to depression severity in traumatic brain injury: Reanalysis of data from a randomized controlled trial

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Abstract

Objective—To explore the relations of cognitive, behavioral, and physical activity variables to depression severity among people with traumatic brain injury (TBI) undergoing a depression treatment trial.

Setting—Community

Participants—Adults (N=88) who sustained complicated mild to severe TBI within the past 10 years, met criteria for major depressive disorder, and completed study measures.

Design—Randomized controlled trial

Methods—Participants were randomized to cognitive-behavioral therapy (n=58) or usual care (n=42). Outcomes were measured at baseline and 16 weeks. We combined the groups and used regressions to explore the relations among theoretical variables and depression outcomes.

Main Measures—Depression severity was measured with the Hamilton Depression Rating Scale and Symptom Checklist-20. Theory based measures were the Dysfunctional Attitudes Scale

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(DAS), Automatic Thoughts Questionnaire (ATQ), Environmental Rewards Observation Scale (EROS), and the International Physical Activity Questionnaire (IPAQ).

Results—Compared to non-TBI norms, baseline DAS and ATQ scores were high and EROS and IPAQ scores were low. All outcomes improved from baseline to 16 weeks except the DAS. The ATQ was an independent predictor of baseline depression. An increase in EROS scores was correlated with decreased depression.

Conclusions—Increasing participation in meaningful roles and pleasant activities may be a promising approach to treating depression after TBI.

Keywords

brain injuries; depression; cognitive therapy; behavior therapy; surveys and questionnaires; physical activity

INTRODUCTION

Major depressive disorder (MDD) following TBI is increasingly recognized as a distinct,[1] highly prevalent[2] comorbid condition that is associated with greater disability after TBI. [3–5] Yet, depression is undertreated [2] and the field lacks solid evidence-based treatments for this important comorbidity.[6,7] One randomized controlled trial used physical exercise as a treatment for depression;[8] however, most depression treatment trials have used some variant of cognitive behavioral therapy (CBT).[7,9] The effect of these interventions has not been robust, compared to control groups. [7,9]

To inform future research and clinical care, we analyzed the results of a recently completed clinical trial from three theoretically distinct treatment perspectives commonly used to understand and improve depression: cognitive therapy, behavior therapy, and physical activity interventions. Cognitive therapy is founded upon the idea that negative automatic thoughts [10] and dysfunctional attitudes [11] are higher among people who are depressed. Moreover, reductions in negative cognitions are believed to mediate improvements in depression in the course of treatment.[12,13] Behavior therapy is based on the notion that a lack of rewarding activities causes depression.[14] Furthermore, from this perspective increasing engagement in rewarding behaviors, (e.g., increased pleasant or meaningful activities) should mediate treatment-related improvement in depression.[15] Regarding physical activity, research supports a link between the intensity and duration of physical activity and mood.[16] Research has shown that low physical activity [17,18] and high levels of sedentary behavior [19] increase ones risk for depression. Exercise and physical activity interventions have been found to reduce depressive symptoms [20] and can be an effective treatment for major depression. [21] Finally, there is a dose-response relation between increased energy expenditure and recovery from MDD. [22]

In this clinical trial, the effects of in-person CBT or telephone-administered CBT were compared to usual care for MDD after TBI.[23] Based on the classic CBT model, the intervention involved a behavior therapy aspect to increase pleasant, rewarding activities, including physical activity,[24] as well as a cognitive therapy component to identify and mitigate depressive thinking. We measured the effect of the trial on depression severity as

well as on cognitive therapy aspects (negative automatic thoughts, dysfunctional attitudes), behavior therapy aspects (environmental rewards), and physical activity aspects (time spent engaged in physical activity and sedentary behavior) at baseline and 16 weeks.

The primary analyses revealed similar improvement in depression severity in the usual care versus combined CBT groups on the 17-item version of the Hamilton Depression Rating Scale (HAMD-17)[25] and the Depression Symptom Checklist-20 (SCL-20).[26] The total sample demonstrated an overall decrease of 5.9 points on the HAMD-17. Approximately one-third demonstrated a 50% decrease on the HAM-D, and two-thirds no longer met criteria for MDD at 16 weeks.[23] There were no between-groups differences on any of the cognitive, behavioral or physical activity variables at baseline or 16 weeks.

We wanted to examine how these theoretically-based variables were related to depression severity in our sample of people with MDD and TBI. First, we compared mean scores of these variables at baseline to the best available normative data. In this depressed group we hypothesized that at baseline, negative automatic thoughts, dysfunctional attitudes, and sedentary behaviors would be higher than population means while environmental rewards and physical activity would be lower than average, compared to available norms. Second, we hypothesized that at baseline negative automatic thoughts, dysfunctional attitudes, and sedentary behavior would be positively correlated with depression severity while pleasant activities and physical activity would be inversely correlated with depression severity. Third, we examined whether baseline values of these theoretical variables were predictors of change in depression severity over time. Based on prior research,[27,28] we hypothesized that high levels of dysfunctional attitudes would be associated with less improvement in depression treated with CBT. Fourth, we hypothesized that improvement in the theoretical variables (less negative thinking and sedentary behavior and greater pleasant activities and physical activity) would be associated with improvement in depression severity over the course of the study.

METHODS

Procedures

The study was based at the University of Washington (UW) and conducted between December 2008 and December 2012. The Human Subjects Review Boards at the UW and all the recruitment sites approved the study.

The study coordinator contacted persons who indicated interest in the study. Interested persons underwent preliminary screening for probable MDD using the Patient Health Questionnaire-9 (PHQ-9)[29] and basic inclusion and exclusion criteria. Those who had PHQ-9 scores of at least 10 and met other eligibility criteria were invited to participate in secondary screening that included: release of medical information to confirm history of TBI and the depression module of the Structured Clinical Interview for DSM-IV (SCID).[30] Those who met criteria for MDD and other eligibility criteria underwent informed consent procedures. All screening, baseline and outcome assessments were conducted over the telephone by trained study staff kept unaware of randomization status.

Study Sample

Participants were recruited regionally and nationally from community and clinical settings serving persons with TBI. Specifically, subjects were recruited through the University of Washington affiliated medical centers (Seattle, WA), St. Luke's Rehabilitation Institute (Spokane, WA), Moss Rehabilitation Institute (Philadelphia, PA), and University of Alabama at Birmingham (Birmingham, AL). We also recruited participants through TBI-related websites, TBI support groups, TBI clubhouses, a study website, newsletters and email listservs.

Inclusion criteria were: at least 18 years of age; hospitalized within the past 10 years for a complicated mild to severe TBI as defined by a Glasgow Coma Scale (GCS)[31] score of 3–12 or documented intracranial abnormalities on imaging or had post-traumatic amnesia of at least 7 days.[32] If TBI diagnosis was based exclusively on GCS, participants were excluded if there was evidence that alcohol intoxication could have confounded the exam (blood alcohol levels exceeded 199 mg/dL).[33] Participants met DSM-IV criteria for current MDD and had to be fluent English speakers who resided in the United States.

Exclusion criteria were: no stable home or regular access to a telephone; lifetime history of schizophrenia; current psychotic disorder, suicidal intent, or alcohol or drug dependence (within the past month) based on the MINI International Neuropsychiatric Interview;[34] currently in psychotherapy for depression; planning to start an antidepressant or psychotherapy for depression within the 16-week study period; new or unstable dose of antidepressant medication (within 6, and 4 weeks, respectively); and significant cognitive impairment as demonstrated by impaired performance on two or more of the following four tests administered via telephone: Digit Span (excluded if below the 5th percentile),[35] Hopkins Verbal Learning Test,[36] (excluded if below the 1st percentile) and Oral Trailmaking Tests A and B,[37] (excluded if below the 1st percentile).

Sample characteristics

We screened 267 people. One hundred were eligible, consented, underwent the baseline assessment and were randomized (n=40 to telephone CBT, 18 to in-person CBT, and n=42 to usual care control). Participants were on average 45.8 (SD 13.3) years old; 63% were male, 90% non-Hispanic White, and 71% had completed at least some college (see Table 1). Mean time since injury was 3.33 (SD 2.72) years and 31% had a severe TBI. Average HAM-D scores at baseline (17.5 [SD 3.9]) were in the moderately depressed range.[38] Outcome data were obtained on 88 participants at 16 weeks.

Thirty-eight percent reported a history of MDD prior to their TBI, 34% had 2 or more prior major depressive episodes, and 53% reported their current depressive episode had lasted at least one year. Psychiatric comorbidities included current dysthymic disorder (12%), alcohol abuse (10%), substance abuse (4%), and a history of PTSD (18%). Forty percent of those in usual care and 55% in the CBT group reported receiving antidepressant treatment or counseling (e.g., rehabilitation counseling) outside the study during the trial.

Randomization

We used “choice- or equipoise-stratified” randomization.[39] Participants chose from three randomization options: 1) telephone CBT versus in person CBT versus UC; 2) in person CBT versus UC; or 3) telephone CBT versus UC. Randomization was computer-generated and stratified on TBI severity (complicated mild/moderate versus severe) and randomization option choice. See primary outcome paper for details.[23]

Measures

Demographic and injury-related characteristics were obtained from participant screening interviews and medical records reviews. The Mini International Neuropsychiatric Interview (MINI)[34] was used to rule out bipolar disorder and psychosis and to determine the presence or absence of comorbid substance abuse or dependence prior to the baseline assessment.

Depression severity was measured at baseline and 16 weeks with two measures. The HAM-D-17 is a widely-used clinician-rated measure of depression severity during the past week.[25] Rating scales are unique to each item. The total score can range between 0–53 points, with higher scores indicating more severe depression. The SCL-20 is a self-report measure on which respondents indicate how much they were distressed by 20 depression symptoms over the past week on a scale from 0 (not at all) to 4 (extremely).[26] The total score on the SCL-20 ranges from 0–80; higher scores represent more severe depression. Both instruments have been shown to have strong measurement properties in this sample.[40] We used a structured version of the HAM-D for improved reliability.[41]

Automatic negative depressive thoughts were measured with the Automatic Thoughts Questionnaire (ATQ),[10] a 30-item questionnaire that measures how often negative depression-related thoughts or “self-talk” occurred during the past week, using a scale from 1 (not at all) to 5 (all the time). Scores range from 30–150 with higher scores reflecting greater negative self-talk. Prior studies have shown that the ATQ is reliable, valid and sensitive to change with treatment.[42]

Depression-related dysfunctional attitudes were measured with the Dysfunctional Attitudes Scale (DAS Form A),[11] a 40-item questionnaire asking respondents to indicate how they think “most of the time”. The DAS is thought to measure beliefs or assumptions characteristic of depressed persons. Respondents indicate agreement with these attitudes toward life on a scale from 1 (totally agree) to 7 (totally disagree). The total score can range from 40–280 and higher scores correspond to greater dysfunctional attitudes. The DAS has good internal consistency, test-retest reliability and good construct validity, including moderately strong correlations with depression.[11] The DAS has been shown to normalize with cognitive therapy. [12]

Response-contingent environmental rewards were measured with the Environmental Reward Observation Scale.(EROS)[43], which consists of 10 items that measure how much life experiences and events are pleasurable or satisfying rated on a 1 (strongly disagree) to 4 (strongly agree) scale. Exemplar items are: “A lot of activities in my life are pleasurable” and “I wish that I could find more hobbies that would bring me a sense of pleasure” (reverse scored). The measure has good internal consistency, test-retest reliability and convergent

validity compared to pleasant events measures.[43] The EROS improves with behavior therapy for depression.[44]

Physical activity was measured with the International Physical Activity Questionnaire (IPAQ 7-Day short version)[45] that assesses minutes and hours per day spent engaging in moderate, vigorous, and walking activities. The IPAQ has been validated against accelerometer data, performs at least as well as other self-report measures[45] and is sensitive to change.[46] Data were processed according to guidelines (available at: http://www.ipaq.ki.se/ipaq.htm). For the total score, we derived total metabolic equivalent (MET)-minutes per week by adding the MET-minutes spent in moderate, vigorous, and walking activity per week.

Sedentary activity was measured by a single question that is part of the IPAQ and has demonstrated acceptable reliability and validity.[47] The question asks, “During the last 7 days, how much time did you usually spend sitting on a weekday?” Total sedentary activity is expressed in hours of sitting per week.

CBT-TBI Intervention

We developed a manualized version of CBT for people with TBI (CBT-TBI) based on Drs. Simon and Ludman’s eight-session structured telephone CBT protocol for primary care patients.[48] The CBT-TBI therapy manual was used for both in-person and telephone counseling. Based on our prior TBI telephone counseling studies,[49,50] we reorganized the material into 12 sessions delivered over 16 weeks and instituted other adaptations to make the protocol more accessible for people with cognitive impairment. See the primary outcome paper for details.[23]

Usual Care

Patients assigned to usual care were notified by phone of their depression status and were encouraged to continue using the rehabilitation and primary care services available to them. Patients were free to self-refer to mental health services outside the study, and a list of local mental health resources was provided to the patient.

Data Analyses

Since the treatment and control groups achieved similar improvement on the primary analysis, we combined the data from both treatment and control groups for this study. We used descriptive statistics to compare baseline theoretical (cognitive, behavioral and physical activity) variables to existing normative data. To determine whether at baseline theoretical variables were significant correlates of depression, we used univariate and stepwise linear regressions to examine the relations between theoretical variables and measures of depression severity at baseline. To examine whether theoretical variables predicted improvement in depressive symptoms over the course of the study, we computed regressions using baseline theoretical variables to predict depression severity at 16 weeks after controlling for baseline depression. We used a similar approach to evaluate whether the theoretical variables functioned like mediators of depression improvement. Step-wise regressions used a forward selection approach ($p<.05$ to enter, $p>.10$ to leave). Due to the

large number of regressions, we focused on results that were consistent based on the HAM-D and the SCL-20.

RESULTS

Theoretical variables at baseline and end of trial (Hypothesis 1)

At baseline, cognitive, behavioral, and physical activity variables were different from nondepressed, non-TBI norms in the expected directions (see Table 2). The study sample was 1.47 SD higher on the ATQ, 2.81 SD higher on the DAS, and 1.55 SD lower on the EROS compared to existing normative data from nondepressed samples.[43,51] Baseline physical activity was slightly below U. S. norms[52] while median hours of sedentary behavior (sitting) was 6 hours per week day in the sample versus 4 hours in the normative sample.[47]

Over the course of the trial ATQ, EROS, and IPAQ MET variables improved, but did not completely normalize. Surprisingly, mean DAS scores increased somewhat from baseline to the end of the trial, indicating a slight worsening in dysfunctional attitudes over time. Median sitting time remained unchanged over the course of the trial.

Relations between theoretical variables and depression severity at baseline (Hypothesis 2)

The first set of regression analyses addresses the question of what baseline cognitive, behavioral and physical activity variables are significantly related to depression severity at baseline (see Table 3). Based on the univariate regressions, the only baseline variables associated with initial depression severity on both the HAMD and the SCL-20 were higher negative automatic thoughts (ATQ) and lower environmental rewards (EROS). In the stepwise model, only the ATQ was an independent predictor of both depression measures.

Baseline theoretical variables as predictors of change in depression severity (Hypothesis 3)

The next set of regressions examined the extent to which baseline cognitive, behavioral or physical activity variables predicted change in depression severity from baseline to 16 weeks. There were no consistent predictors of change in depression severity across both measures. For example, as baseline MET scores increased, HAM-D scores decreased (B= −0.30, p=.004), but the same relation was not found between METs and the SCL-20.

Change in theoretical variables as predictors of change in depression (Hypothesis 4)

The third set of regressions was used to identify cognitive, behavioral or physical activity variables that changed in relation to change in depression severity. The univariate regressions indicated that decreases in automatic thoughts as well as increases in environmental rewards were significantly related to decreased depression severity on both outcome measures. In the forward step-wise models, only increased environmental rewards was related to decreased depression severity on both measures. However, there was a non-significant trend for decreased sitting time to be related to decreased depression severity on both the HAM-D and SCL-20.

Reported regressions did not include treatment group assignment in the models. However, we conducted follow-up regression analyses in which we examined whether there was an interaction between the theoretical variables and treatment condition when predicting depression severity outcomes. None of the models predicting HAMD-17 or SCL-20 outcomes revealed a significant interaction effect between the DAS, ATQ or EROS and treatment condition (data not shown).

DISCUSSION

To our knowledge this is the first study to examine evidence for the utility of cognitive, behavioral and physical activity models as ways to explain and potentially target treatment for MDD in people with TBI. We observed interesting similarities and differences compared to what has been found among people without TBI.

Negative thoughts, dysfunctional attitudes, and depression in TBI

Consistent with prior research,[53] we found evidence for the relevance of negative automatic thoughts as measured by the ATQ in people with TBI and MDD. ATQ scores were elevated in people with TBI and MDD compared to normative data.[51] The ATQ was also significantly related to baseline depression severity based on both HAMD-17 and SCL-20 scores. In the step-wise regression model, the ATQ was the only independent predictor of baseline depression severity for both measures. While none of the theoretical measures predicted post-treatment depression severity, change in ATQ scores was significantly and consistently related to change in depression severity from baseline to 16 weeks. These results support the notion that treatment aimed at reducing negative automatic thoughts may facilitate alleviation of MDD in this population. Based on the work of Malec and colleagues,[54] automatic negative thoughts about the severity of TBI-related impairments may be particularly important to target in interventions aimed at reducing depression severity after TBI.

In contrast to the pattern of results from the ATQ, the relations between the DAS and depression outcomes did not fit the cognitive therapy model of depression. Participants reported extremely high DAS scores at both time points. CBT did not influence DAS scores relative to controls, and mean DAS scores worsened slightly over the course of the trial. Of all the theory-based measures we used, the DAS has the most evidence for moderating outcomes.[55] However, high baseline DAS scores did not predict less improvement in depression severity at the end of the study. A decrease in dysfunctional attitudes was related to improvement in depression severity across both measures. However, the relation of change in DAS scores to change in depression severity did not remain significant in the step-wise regression models.

We can only speculate about why the ATQ and DAS had dissimilar relations with depression severity. While both measures tend to covary with depression severity in treatment trials,[56] the DAS demonstrates greater stability over time.[57] One reason may be that on the DAS, respondents are asked to describe the way they think most of the time, whereas on the ATQ individuals indicate how often the thought occurred to them over the last week. Next, the DAS items form two factors: perfectionism and need for approval.[58] It may be that for

people living with TBI, issues around self-criticism, unmet needs for achievement, dependency upon others, and needing approval are particularly salient and contribute to persistently high scores on the DAS. Dysfunctional attitudes are characterized as "maladaptive, inflexible and extreme assumptions by which the self or world is judged."[p. 1531][13] Common TBI-related cognitive impairments, especially in the domain of executive dysfunction, could lead to more extreme agreement with DAS items. These impairments might not inflate ATQ scores, which essentially ask respondents to recall how many times they had a particular thought in the past week. Finally, DAS scores may have not improved or worsened slightly over the study period in part because people with TBI may have difficulty with the metacognitive demands of cognitive therapy such as self-observation as well as analyzing and refuting dysfunctional thinking patterns. Perhaps giving people with TBI more time, more cognitive accommodations, or other therapeutic techniques will improve outcomes from cognitive therapy. Other therapeutic techniques include acceptance and commitment therapy (ACT)[59] and mindfulness based CBT (MBCBT).[60] These therapies seem promising because they rely on concepts such as awareness, acceptance, and compassion rather than on trying to challenge dysfunctional beliefs. Preliminary MBCBT trials among people with TBI and symptoms of depression suggest this treatment may be efficacious.[61–63]

Environmental rewards and depression in TBI

One of the most compelling findings had to do with the relation between depression and environmental rewards. Lower scores on the EROS were correlated with higher depression severity at baseline, and greater increases in EROS scores were related to greater improvement in depression severity over the course of the trial. In fact, change in EROS scores emerged as the only significant independent predictor of depression improvement in the stepwise regression model.

These results provide preliminary support for a behavioral understanding of depression after TBI[14] and imply that helping people engage in meaningful, enjoyable roles and activities on a routine basis may be an effective way of treating MDD. Recent observational[64,65] and treatment studies[66] also can be interpreted to support a behavioral approach to understanding and treating depression after TBI. For example, in a longitudinal study that employed structural equation modeling, Schonberger and colleagues[65] demonstrated that functional changes 1–6 months after TBI predicted anxiety and depression one year after injury. The authors interpreted the results to mean that improving a person's capacity to lead a satisfying life despite disabilities may reduce psychiatric illness after TBI. Bombardier and colleagues[66] argued that a telephone intervention reduced depressive symptoms in persons with TBI because it focused on helping people resume meaningful social and occupational roles.

In summary, these results suggest that behaviorally-oriented therapies such as behavioral activation[24,67] represent a promising approach to treating depression after TBI, but may require further adaptations for this population. In the CBT trial upon which this study is based,[23] we addressed behavioral activation first and emphasized it more than cognitive distancing and restructuring techniques. Nevertheless, we were unable to demonstrate

greater improvement in environmental rewards in the treatment versus control group at 16 weeks, and scores on the EROS remained below average based on available normative data. Research is needed on methods to enhance resumption of pleasant or meaningful daily activities in this population.

Physical activity and depression in TBI

Research in non-TBI samples has indicated that low physical activity [17,18] and high levels of sedentary behavior [19] are associated with greater depressive symptoms. Similarly, we found that self reported physical activity was low and sedentary behavior was high in this sample compared to normative data. However, we found no evidence that physical activity measures were related to depression severity at baseline and only inconsistent evidence that physical activity was predictive of depression severity at 16 weeks. There was little evidence for any consistent or meaningful relation between change in physical activity measures and change in depression over the course of the trial. The absence of a relation may be partly attributable to the fact that the study did not explicitly focus on improving physical activity except to the extent that this may have been an aspect of the behavioral activation plan. It should be noted that in a trial that sought to improve depression through improved physical activity in persons with TBI, significantly reduced depression was found among people who exercised at least 90 minutes per day.[8]

Limitations

Before concluding, key limitations of the study should be highlighted. The sample was relatively small and consisted of people with MDD who enrolled in a treatment trial. Participants were mostly male, Caucasian and middle-aged. Therefore, these findings may not be generalizable to the broader population of people with TBI and depression. We lack information on the magnitude of a clinically significant change in DAS, ATQ and EROS measures as well as whether the Beta scores in Table 3 represent clinically meaningful relations. This limits our ability to judge how meaningful changes on these measures are. Our study lacked non-depressed TBI controls and relied on comparisons between our participants and existing normative data on the DAS, ATQ, EROS and IPAQ. As a result we cannot be certain that the differences we observed between study participants and normative data are due to depression and not to TBI or other characteristics that might differ between our sample and normative groups. Future studies that assess theoretical variables such as dysfunctional attitudes in TBI survivors with and without comorbid depression could help elucidate the extent to which these variables are related to depression versus other TBI-related factors such as cognitive impairment. Since in our treatment trial CBT did not result in greater improvement in these theoretical variables compared to the control group, we combined both treatment and control groups for this study. Therefore, we necessarily assumed that the relations between depression severity and the theoretical variables were similar in the treatment and control conditions. Follow-up regression analyses in which group was included in the models predicting depression severity supported this assumption.

Conclusions

This study offers some support for the utility of a cognitive-behavioral model of depression among people with TBI. Hypothetical cognitive, behavioral and physical activity correlates

of depression are elevated among people with TBI and depression. None are indicators of poor prognosis for improvement over a 16-week period. Our results suggest behavioral activation, which is designed to reduce avoidance and improve engagement in pleasant and meaningful activities in daily life, may be an effective means of treating people with TBI and MDD. Put in broader terms, interventions that help the person recover from the psychosocial effects of TBI and resume important roles and enjoyable activities should facilitate recovery from MDD. The data suggest that there also may be a role for cognitive therapy aimed at reducing negative automatic thoughts. One place to start may be challenging overestimates of TBI-related impairments.[54] Given that our trial of CBT for MDD failed to improve environmental rewards or negative automatic thoughts more than usual care, the field needs treatment development research to discover more powerful ways to conduct behavioral activation and cognitive therapy in people with TBI. To be effective, behavioral and cognitive therapy interventions may need to be longer, more frequent or more intensive than in standard protocols, and may require more intensive adjunctive case management to help patients overcome the often multiple practical and biopsychosocial barriers to recovery.[23] The feasibility and efficacy of ACT and MBCT also deserve exploration as alternatives to classical CBT.

Effectiveness studies also are needed to discover how we can increase the impact of existing interventions. Stepped-care or combined treatment models should be explored. In stepped-care, patients choose between several potentially effective therapies (e.g. behavioral activation, cognitive therapy and/or antidepressants). Outcomes are carefully monitored and treatment is intensified or switched to an alternative approach if they do not achieve remission from their depression after an adequate trial (e.g., 6–8 weeks).[68] More research is needed to achieve a ‘personalized’ approach of targeted initial treatment based on baseline demographic and clinical variables. The efficacy of combined medical and psychotherapeutic treatments for MDD after TBI also merits research since thus far the efficacy of unimodal approaches to treating MDD in this population has not been robust.

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Table 1

Demographic and Injury Characteristics of Participants

Variable	All Randomized Subjects N=100	Randomized to CBT N=58	Randomized to Usual Care N=42
Age Mean (SD)	45.8 (13.3)	45.4 (14.1)	46.3 (12.4)
Sex – n (%) male	63 (63%)	34 (59%)	29 (69%)
Race/Ethnicity– n (%)			
Non-Hispanic White	90 (90%)	52 (90%)	38 (90%)
Other	10 (10%)	6 (10%)	4 (10%)
Education – n (%)			
Less than high school	12 (4%)	7 (3%)	5 (5%)
GED	8 (8%)	5 (9%)	3 (7%)
High school diploma	10 (10%)	8 (14%)	2 (5%)
Tech/Vocational	7 (7%)	5 (9%)	2 (5%)
Some college	45 (45%)	26 (45%)	19 (45%)
Undergraduate degree	16 (16%)	9 (16%)	7 (17%)
Graduate degree	10 (10%)	3 (5%)	7 (17%)
Marital Status – n (%)			
1 – Single/Never married	30 (30%)	18 (31%)	12 (29%)
2 – Married/Partnered	24 (24%)	11 (19%)	13 (31%)
3 - Divorced	39 (39%)	24 (41%)	15 (36%)
4 - Separated	5 (5%)	3 (5%)	2 (5%)
5 - Widowed	1 (1%)	1 (2%)	0 (0%)
6 - Other	1 (1%)	1 (2%)	0 (0%)
Cause of Injury – n (%)			
Motor vehicle	38 (28%)	24 (41%)	14 (33%)
Fall	34 (34%)	18 (31%)	16 (38%)
Assault/Blunt	8 (8%)	6 (10%)	2 (5%)
Bicycle	5 (5%)	3 (5%)	2 (5%)
Pedestrian	8 (8%)	4 (7%)	4 (10%)
Other	7 (7%)	3 (5%)	4 (9%)
TBI Severity			
Complicated Mild-Moderate	69 (69%)	40 (69%)	29 (69%)
Severe	31 (31%)	18 (31%)	13 (31%)
Years since TBI mean (SD)	3.33 (2.72)	3.41 (2.84)	3.21 (2.58)

Note: All comparisons were non-significant by Fisher Exact or Mann-Whitney

Table 2

Depression and Predictor Variable Outcomes

Variable	Baseline	16 Weeks	Norms
Depression measures			
HAM-D-17 – mean (SD)	17.6 (4.0)	11.9 (6.3)	Mild (8–13) to moderate (14–18) depression [38]
HAM-D-17 response n (%)		28 (32%)	---
SCL-20 mean (SD)	1.95 (0.53)	1.23 (0.70)	---
SCL-20 response n (%)		33 (38%)	---
MDD negative on SCID		58 (67%)	---
Predictor Variables			
ATQ – mean (SD)	79.8 (26.7)	63.1 (26.3)	52.9 (18.2)[51]
DAS Total – mean (SD)	194 (36.0)	209 (30.0)	119.0 (26.7)[51]
EROS – mean (SD)	21.8 (4.8)	24.7 (5.2)	29.4 (4.9)[43]
IPAQ MET minutes/week (median)	2396	2859	2514
N (%) with low physical activity	47 (48%)	36 (41%)	15.9%
N (%) with moderate physical activity	9 (9%)	11 (13%)	22.1%
N (%) with high physical activity	42 (43%) *	41 (47%) **	62.0%[52]
IPAQ sitting hours/weekday (median)	6	6	4 hrs/weekday[47]

† Significance by Fisher Exact or Mann-Whitney.

Unknowns

* n=2;

** n=12

Table 3

Regressions examining the relationships between cognitive, behavioral and physical activity predictors and depression outcomes

	Predicting Baseline DV, Using Baseline IV's				**Predicting 16 week DV, Controlling for Baseline DV, Using Baseline IV's**				**Predicting 16 week DV, Controlling for Baseline DV, Using Change in IV's**			
	Univariate		**Stepwise**		**Univariate**		**Stepwise**		**Univariate**		**Stepwise**	
HAM-D	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*
ATQ	0.05	**.001**	0.05	**.001**	0.03	.312			0.11	**<.001**		
DAS	0.00	.881			0.02	.402			−0.05	.009		
EROS	−0.18	**.037**			0.01	.943			−0.52	**<.001**	−0.52	**<.001**
MET*1000	0.02	.730			−0.23	**.021**	−0.30	**.004**	0.03	.773		
SITTING	0.17	.118			−0.22	.205	−0.39	**.029**	0.43	**.010**		
SCL-20	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*	*B*	*Sig.*
ATQ	0.01	**<.001**	0.01	**<.001**	0.01	.158			0.02	**<.001**	0.01	**<.001**
DAS	−0.00	**.001**			0.00	.262			−0.01	**<.001**		
EROS	−0.04	**<.001**			−0.01	.736			−0.06	**<.001**	−0.03	**.035**
MET*1000	−0.01	.180			−0.01	.181			0.00	.830		
SITTING	0.03	**.039**			−0.03	.157			0.05	.650	0.03	**.050**

Predictors with values in gray are not in the stepwise regression models

DV=dependent variable, i.e., measures of depression; IV=independent variable