Title

Visually mediated functioning improves following treatment of hoarding disorder.

Authors

J Zakrzewski, Jessica
A Gillett, Drew
R Vigil, Ofilio
et al.

Publication Date

2020-03-01

DOI

10.1016/j.jad.2019.12.030

Peer reviewed

HHS Public Access

Author manuscript

J Affect Disord. Author manuscript; available in PMC 2021 April 20.

Published in final edited form as:

J Affect Disord. 2020 March 01; 264: 310–317. doi:10.1016/j.jad.2019.12.030.

Visually Mediated Functioning Improves Following Treatment of Hoarding Disorder

Jessica J. Zakrzewski[1], **Drew A. Gillett**[1], **Ofilio R. Vigil**[2], **Lauren C. Smith**[2], **Kiya Komaiko**[2], **Chia-Ying Chou**[2], **Soo Y. Uhm**[2], **L. David Bain**[3], **Sandra J. Stark**[3], **Michael Gause**[4], **Gillian Howell**[3], **Eduardo Vega**[3], **Joanne Chan**[3], **Monika B. Eckfield**[5], **Janice Y. Tsoh**[2], **Kevin Delucchi**[2], **R. Scott Mackin**[2], **Carol A Mathews**[1]

[1]Department of Psychiatry, Center for OCD, Anxiety, and Related Disorders, College of Medicine, University of Florida, FL, USA

[2]Department of Psychiatry, University of California, San Francisco and San Francisco VA Medical Center, San Francisco, CA, USA

[3]Mental Health Association of San Francisco, San Francisco, CA, USA

[4]Sonoma County Community Development Commission, USA

[5]Department of Nursing, California State University, East Bay, CA, USA

Abstract

Background: Hoarding disorder (HD) is a highly debilitating psychiatric disorder that affects 2–6% of adults. Neuropsychological deficits in visual memory, detection, and categorization have been reported in HD. To date, no study has examined the relationship between neurocognitive functioning and treatment for HD. We aim to determine the association between neurocognitive functioning and treatment outcomes, as well as the impact of HD-specific treatment on cognitive functioning.

Methods: 323 individuals with HD were randomized to 20 weeks of peer- or clinician-led group behavioral treatment. 242 participants completed pre- and post-treatment neuropsychological testing covering eight neurocognitive domains. Rates of cognitive impairment (CI) were assessed for each neurocognitive domain. The association of baseline neurocognitive function on treatment response was examined using multiple regression. MANOVA and post-hoc tests were used to determine neurocognitive performance change pre- to post treatment.

Results: Sixty-seven percent of participants had CI on ≥1 cognitive domain. There was no significant effect of pre-treatment neurocognitive functioning on treatment outcome. Post-treatment improvements were observed in visual memory, visual detection, decision making,

Address Correspondence to: Carol A. Mathews, Department of Psychiatry, University of Florida, 100 S Newell Drive, L4-100, Gainesville FL 32610, 352-294-4927, fax 352-627-5015, carolmathews@ufl.edu, Scott Mackin, PhD, Department of Psychiatry, 401 Parnassus Avenue, San Francisco, CA 94143-0984, Scott.mackin@ucsf.edu.

Authors Jessica J. Zakrzewski, Ofilio Vigil, Carol A. Mathews, R. Scott Mackin Drew A. Gillett, Lauren C. Smith, Kiya Komaiko, Chia-Ying Chou, Soo Y. Uhm, L. David Bain, Sandra J. Stark, Michael Gause, Mark Salazar, Julian Plumadore, Gillian Howell, Eduardo Vega, Joanne Chan, Monika B. Eckfield, Janice Y. Tsoh, Kevin Delucchi all have no financial disclosures to declare.

All statements in this report, including its finding and conclusions are solely those of the authors and do not necessarily represent the views of the PCORI, its Board of Governors or Methodology Committee.

information processing speed, visuospatial processing, attention/working memory (p≤.001). Declines in performance were found in visual reaction time and categorization.

Limitations: This was a non-inferiority trial to examine two treatment types with no normative comparison group. Treatment seeking individuals are more likely to be insightful, motivated, and have other features which limit generalizability.

Conclusions: Patterns of cognitive impairment in HD are similar to previous reports. Pre-treatment neurocognitive functioning did not impact treatment response. Neuropsychological functioning improved across multiple domains following targeted treatment.

Keywords

Hoarding Disorder; Cognitive Impairment; Treatment; Cognitive Behavioral Therapy; Visual Functioning; Neurocognitive Functioning

1. Background

Hoarding disorder (HD) is one of the most common neuropsychiatric disorders in older adults, with reported prevalence rates of up to~ 6% in individuals over age 55 (Ayers et al., 2010; Cath et al., 2017; Grisham et al., 2006; Samuels et al., 2008). The core feature of HD is the persistent difficulty discarding objects which is often combined with excessive acquisition and leads to clutter over time. Significant clutter in turn leads to a variety of problems, including a high risk for falls, health code violations, house fires and difficulties with self-care (Ayers et al., 2014; Harris, 2010; Kim et al., 2001). Rates of cognitive impairment in specific domains also appear higher in this population than are demonstrated with normal aging and may contribute to functional impairment in these individuals (Ayers and Dozier, 2015; Frost and Hartl, 1996; Grisham et al., 2007; Mackin et al., 2011; Mackin et al., 2016; Tolin et al., 2011).

While the proposed cognitive-behavioral model for hoarding disorder (Frost and Hartl, 1996) posits that information processing and decision-making difficulties are key factors of hoarding disorder, the extant literature regarding the type and extent of cognitive impairment in HD is still inconsistent. To date, the cognitive domains that have been examined and the tests used to examine each to domain vary widely (Ayers et al., 2013; Mackin et al., 2016; Woody et al., 2014). Presently, only a few studies have examined the proportion of individuals whose impairment in a given cognitive domain reaches the threshold of clinically significant neurocognitive deficits (Ayers et al., 2010; Hwang et al., 1998; Mackin et al., 2016; Marx and Cohen-Mansfield, 2003; Tolin et al., 2014), and even fewer have examined neurocognition across multiple domains (Grisham and Baldwin, 2015; Mackin et al., 2011; Mackin et al., 2016).

A systematic review by Woody et al (2014) suggests that the cognitive domains of most interest in HD include indecisiveness and performance in unstructured sorting tasks, attention and working memory, categorization and organization, visual organization and planning, and visuospatial learning and memory. Similarly, a review by Grisham and Baldwin (2015) suggested that, across studies, there appear to be subtle changes in attention, memory (visual and verbal), and sorting/decision making tasks. However, neither of these

reviews included a meta-analysis, and in fact, for many cognitive domains, a sufficient number of studies does not yet exist for a meaningful meta-analysis to be conducted. The variability in the literature with regard to domains studied, tests used, and results obtained makes it difficult to identify neurocognitive domains in which consistent, objective differences between HD and controls are present. Additional data with more consistent test use within and across domains are thus still needed.

However, the two comprehensive neurocognitive studies of HD that have been conducted (Mackin et al., 2011; Mackin et al., 2016) identified impairments primarily in visually mediated domains (visual memory, visual detection and visual categorization), in line with previous, more targeted work. While the available research suggests that visually mediated processes, including categorization, may be particularly problematic in HD, it is unclear if these deficits are a cause or an effect of the disorder and if they change following treatment.

The current standard of care for treatment of HD includes cognitive-behavioral therapy (CBT) in either individual or group format (Tolin et al., 2015; Williams and Viscusi, 2016). CBT for HD involves increasing motivation for treatment, graded exposure to non-acquiring, organizational training, practice in sorting and discarding possessions, and cognitive restructuring techniques (Steketee et al., 2010; Tolin et al., 2007). Clinician-led CBT reduces HD symptoms by 20–30%; our group has recently shown that peer-led treatment is equally effective (Mathews et al., 2018). Using the same sample examined in the currently study, Mathews et al. (2018) found that individuals with high levels of hoarding symptom severity at baseline and those who received assistance from family and friends post-treatment received the most benefit from therapy. While we know that type of facilitator does not affect treatment success, it is currently unknown whether treatment for HD impacts cognition, or whether cognitive dysfunction affects the effectiveness of hoarding treatment.

The aim of this study was to examine the relationship between neurocognitive functioning and HD-specific behavioral treatment outcome. Based on the results for Mackin et al (2011; 2016) which also looked across a broad number of domains, we hypothesized that individuals with HD would have impairments in visually mediated cognitive domains, including visual learning and memory, categorization, and speed of information processing, and that these impairments would be associated with a poorer treatment outcome. We also hypothesized that there would be no differences in observed relationships between neurocognition and treatment outcome by treatment type (e.g., clinician vs. peer-led). Finally, we hypothesized that visual categorization and speed of information processing, but not visual learning and memory, would improve following HD-specific treatment, which focuses on sorting, decision making, and decluttering techniques.

2. METHODS

2.1 Participants and Study Procedures

Three hundred and twenty-three participants with HD were included in the treatment study. Full protocol details and the primary treatment outcomes have been previously published (Mathews et al., 2018; Uhm et al., 2016). At baseline, participants completed a semi-structured clinical interview, self-report questionnaires and a comprehensive neurocognitive

assessment (Table 1). Following the 20-week treatment protocol, participants repeated the self-report questionnaires and underwent a second neurocognitive assessment. Participants were contacted for the post-treatment assessments regardless of whether they completed the treatment and were financially compensated for their participation.

2.2 Inclusion/exclusion

All participants met DSM-5 criteria for HD and had moderate to severe hoarding symptoms. Individuals were excluded if they had moderate or severe dementia (Montreal Cognitive Assessment score <17), were unable to consent to participation, or were unable to participate in treatment groups. If a participant had received individual or group treatment for HD in the year prior, they were also excluded, but not if they were receiving other forms of concurrent treatment such as medication management, or psychotherapy for non-hoarding related symptoms. Participants were not excluded if they had co-occurring psychiatric illnesses, were on medication, had active substance use, or psychosis.

2.3 Clinical and Self-Report Assessments

Hoarding symptom severity was assessed at baseline and post-treatment with the Saving Inventory-Revised (SI-R; (Frost et al., 2004). HD diagnosis was assigned using the Structured Interview for Hoarding Disorder (Nordsletten et al., 2013), and lifetime and current history of other psychiatric disorders were assessed using the Mini International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998). The Beck Depression Inventory-II (BDI) and the Beck Anxiety Inventory (BAI) were administered pre and post treatment (Beck and Steer, 1993; Beck et al., 1961). The Swanson, Nolan and Pelham Questionnaire (SNAP-IV) was used to assess symptoms of attention, concentration, hyperactivity and impulsiveness and was administered at baseline (Swanson et al., 2001).

2.4 Neurocognitive Assessment

Neurocognitive functioning was assessed following the clinical assessments and prior to randomization. Participants completed post-treatment cognitive assessments within three months following their last group therapy meeting (preferably within one month). The average time for an individual's treatment start date following assessment varied based on meeting participant needs for location and timing, but the average time between pre-treatment assessment and post treatment survey completion was eight months. A small minority of participants (N=19, 3%) completed the post assessment ≥15 months following their pre-assessment. The cognitive assessment was designed to cover a broad array of cognitive functioning domains, minimizing participant burden (for full list of the variables used for each domain by task, see Table 1). Estimates of full scale IQs were obtained using the National Adult Reading Test (NART), an oral word reading test, which has been shown to create a quick and reliable estimate of an individual's IQ (Nelson, 1982). Verbal memory and learning were assessed using an auditory list learning task, the Hopkins Verbal Learning Test- Revised (HVLT-R) (Shapiro et al., 1999). Visual learning and memory were assessed using the Brief Visuospatial Memory Test-Revised, which consists of a brief presentation of a series of simple visual figures that are then graphically reproduced by the participant and scored for both accuracy and location (Benedict et al., 1996). The Wechsler Adult Intelligence Test -IV (WAIS-IV) Block Design Task (Wechsler, 1999), which involves

manually manipulating blocks to reproduce designs of increasing difficulty, was used to assess visuospatial processing. Abstract reasoning was assessed using the WAIS-IV Matrix Reasoning task, which involves visual pattern matching. Information processing speed was assessed using two tasks, the Symbol Digit Modalities Test (SDMT), which involves timed replication of symbols using a numbered key legend (Smith, 2002), and the Stroop Test, which involves rapid recitation of colors printed in words that do not correspond to their ink color (Golden and Freshwater, 2002). Visual detection and perseveration were assessed using the Conners Continuous Performance Test- 2nd Edition (CPT), a computerized task which involves watching for a specific letter and responding via button press (Conners CK, 2000). Visual categorization and problem solving were assessed using the Delis-Kaplan Executive Functioning System (DKEFS) Sorting Task which involves physically sorting a set of cards based on the various features (words, sizes, colors) the cards possess (Delis, 2001b). The Iowa Gambling Task (IGT), a computerized tasks involving feedback and probability of winning based on card selection from various decks, was used to assess decision making and planning (Bechara et al., 1994).

On average, the neurocognitive assessment took 1.5 hours to complete and all tests were administered using standard protocols. To minimize possible learning effects from repeated administration of the same tasks, alternate forms were used for the post-treatment assessments when available. Three tests had validated, alternate forms that were used for post-treatment assessments- for the HVLT-R, BVMT-R, and the DKEFTS Sorting Task. All testing was administered by trained research staff, overseen by a licensed neuropsychologist. Research staff were blinded to participant group status and to co-occurring psychiatric conditions.

2.5 Analysis

To assess any potential limitations to the generalizability of the results of this study due to differential dropout, paired t-tests and Pearson's chi-square tests were performed to evaluate group differences between treatment completers and dropouts on demographic variables. We then performed three primary analyses to address the three core aims of this study (rates of cognitive impairment, impact of neurocognitive functioning on treatment response, and changes in cognitive function following HD-specific treatment). Mean scaled scores for key outcome measures were used (see Table 1) in all three analysis when appropriate, including the third, omnibus analysis, which compared functioning across tests, to allow for equivalent comparisons of performances across measures. However, raw scores were used to examine pre-to-post individual test performances to: 1) preserve maximum variability of performance and 2) allow for assessment of potentially relevant variables within cognitive domains that do not have scaled scores available. As the outcomes examined in both scaled scores and raw scores did not differ substantially, only raw scores are reported for pre-to-post scores.

Given the broad inclusion criteria for the parent treatment study, sensitivity analyses were also performed for each aim to remove any effects of neurological insult or active drug use on the neurocognitive assessments. In these analyses, individuals with the following were excluded: drug use within the last two months and/or significant history of head injury or brain trauma. We also performed additional analyses that included baseline hoarding

symptom severity in the omnibus model. These analyses were exploratory only as hoarding symptom severity and neurocognitive functioning are likely to be highly related to one another, and thus changes in one independent of the other would be difficult to assess within the same model in the current sample. The effect of treatment on hoarding symptom severity has been previously reported (Mathews et al., 2018).

2.6.1 Rates of Cognitive Impairment and Global CI Burden—Cognitive Impairment (CI) was defined as performance equal to or below 1.5 standard deviations below normative data for age and education matched peers on any given measure (scaled score <6). A global CI burden score was created by coding individuals = 1 if they met CI criteria for any given test and summing their scores across all tests (total range = 0–10). Rates of CI and impact of global CI burden on treatment outcome were determined by chi-square tests and frequency rates.

2.6.2 Prediction of Treatment Outcome—The relationship between neurocognitive function and treatment response was examined using multiple regression. Change in hoarding severity (SI-R score) pre-to post-treatment was used as the primary outcome measure for treatment response. We first used mean scaled scores for baseline performance on all neurocognitive measures as predictor variables. We then examined CI in each domain and global CI burden as predictors of treatment outcome. Treatment response was examined for the entire sample, regardless of treatment assignment, as well as for each treatment group (clinician-led and peer-led) separately.

2.6.3 Changes in Neurocognitive Performance Pre- to Post-Treatment—Change in performance pre-to post-treatment was examined for all cognitive tests simultaneously using a two-factor group assignment (clinician vs. peer-led), pre- to post-assessment-by-test repeated measures MANOVA. As this test of main effects eliminates individuals with partial data using listwise deletion, resulting in a loss of information, post-hoc paired t-tests in the entire sample were then conducted for each individual cognitive test using raw scores to maximize the available information and examine the full range of performance variability. Initially, hoarding severity was added as a covariate to this model, however it explained all pre-to-post performance change and was excluded in order to examine the degree of pre-to-post change differences in individuals. In addition to examining the standard outcome variables for each measure, we also examined the DKEFS Sorting Task, which measures visual categorization, in more depth, including not only total correct and incorrect sorts, but also time to first sort and the proportion of correct over attempted sorts (Table 1). We examined these outcomes as visual categorization has been consistently reported to show impairment in HD, and our previous work suggests that HD is associated with more subtle impairments in this domain than can be identified using traditional scale scores (Mackin et al., 2016). We examined only set one of the DKEFS Sorting Task for these detailed analyses because the alternative DKEFS form that was used for set two in the post-treatment assessment is not as well validated, and performance on set two shows more variability and less reliability (Delis, 2001a).

3. RESULTS

3.1 Participant Demographics and Clinical Characteristics

Of the 323 randomized participants, 318 completed at least a portion of the initial pre-treatment neurocognitive assessment, and 243 completed at least some portion of the follow-up assessment. Baseline characteristics of the study participants as well as individuals who dropped out of treatment are shown in Table 2. Demographics did not vary significantly by treatment type (peer or clinician led treatment groups). The majority of participants (61%) met criteria for at least one psychiatric disorder, with mood (50%) and anxiety disorders (48%) being the most common (for a full description of comorbidity within this sample see (Archer et al., 2018)). The overall mean change in SI-R scores (the primary treatment outcome measure) was 17.7 points, with 37% of individuals who received clinician led treatment, and 36% of those who received peer led treatment having post treatment scores below the clinical cutoff for HD (<42 on the SI-R) (for a full description of treatment outcomes see (Mathews et al., 2018)). A total of 67 individuals were in the sensitivity analyses due to drug use or significant neurologic history. These individuals did not differ from included participants on any measure other than an increase in anxiety (sensitivity inclusion mean BAI=16.4, SD=11.6, exclusion mean BAI = 20.9, SD=12.3, t= −2.74, p=0.006). Because the samples did not differ substantially on the majority of the clinical variables examined, we present the primary analyses here, which includes all participants, including those individuals with significant drug or neurologic history. The exceptions, in which we present results from the sensitivity analyses for comparison to the primary analyses, are specifically noted as such.

3.2 Levels of Cognitive Impairment

Sixty-one percent of participants exhibited CI on at least one cognitive test at baseline (number of tests impaired mean (M)=1.5; median (MDN)=1.0; standard deviation (SD)=1.7; range=0–9). Individuals who had impairment on at least one test did not differ in age, gender, race, education, hoarding severity, depression symptoms, or anxiety symptoms from those who did not. Individuals who fell in the impaired range on at least one task had lower National Adult Reading Test (NART) scores (which provides a proxy for IQ) (t=3.17, p=0.002, *M diff*= 2.47) and were more likely to be participants who were excluded in the sensitivity analysis due to a significant neurological history (X^2=6.93 p=0.008). The highest rates of CI were found in visual memory (23%), verbal learning (18%) and memory (17%), processing speed (SDMT 15%, Stroop CW 8%), visual detection (36%) and perseveration (30%) (Figure 1). There were no differences in global CI scores between treatment groups (X^2=5.58, p=0.35). Twenty-five percent of the sample had a global CI score of 1, indicating impairment in one cognitive test, 16% had a score of two, 10% had a score of three, 7% had a score of four, and 4% had a score of five or more. The sensitivity analysis, which examined the reduced data set, excluding the 67 participants with a significant drug, alcohol, or neurological history, demonstrated no significant group differences (X^2=1.68, p=0.996) on the number of domains in which individuals scored in the CI range.

3.3 Relationship between baseline neurocognitive functioning and treatment outcome

When all of the neurocognitive tests were included in a single model to assess the effect of cognitive performance on hoarding specific treatment outcome, pre-treatment cognitive performance did not predict treatment response (R^2=0.10 F(15, 148)=1.06, p=0.395). The global cognitive impairment (CI) score was also not a significant predictor of treatment response (R^2=0.003, F(1, 224)=0.66, p=0.419).

3.4 Pre-to-post change in neurocognitive performance following treatment

The omnibus repeated measures MANOVA test to examine differences in neurocognitive performance by group, time point, and test did not show a significant main effect of treatment group (F(13,144)=0.827, p=0.631, η^2=0.069). There were significant differences between cognitive tests, with a moderate effect size (F(13,144)=24.418, p<0.001, η^2=0.688) and differences across time point with a small to moderate effect size (F(1,156)=6.360, p=0.013, η^2=0.039). There were also significant differences in time by test with a small to moderate effect size (F(13,144)=5.924, p<0.001, η^2=0.348) indicating pre- and post-test performance differences for individual neurocognitive tests. Sensitivity analysis found the same pattern of results, with nearly identical effect sizes (data not shown). When baseline HD severity was added as a covariate to the rmMANOVA, the pre-to-post assessment differences in neurocognitive functioning were no longer significant in the omnibus model (data not shown).

We next assessed the pre-to post-treatment differences by cognitive domain. Eighty-six of the participants were missing data on at least one test in either the pre-or post-assessment batteries. We thus conducted post-hoc paired sample t-tests using raw scores for to assess pre- vs post-test performance for each individual test. The same pattern of results was found in the post-hoc tests generated by the MANOVA (which used scaled scores and only included individuals with complete neurocognitive data) and the raw score t-tests, with minimal differences that could be accounted for by the change in sample size (data not reported).

Visual memory, visuospatial processing, working memory, processing speed, visual detection, and decision making and planning all showed significant increases in performance pre- to post treatment (Table 3). Visual learning, visual detection reaction time, and visual categorization and problem solving all showed significant performance declines pre- to post treatment. The sensitivity analyses (which excluded individuals with significant neurological history or drug use) found similar patterns of performance change, although the changes in decision making and planning were no longer significant; however, one aspect of visual categorization and problem solving (number of incorrect sorts) did improve to a significant level with in the sensitivity analysis, indicating that these measures may be sensitive to neurological insult (Table 3).

In the more nuanced examination of categorization ability using time to first sort and proportion of correct over attempted sorts in both sets of the DKEFS Sorting Task, we found significant increases in the time to first sort and decreases in the proportion of correct sorts pre-to post-treatment. When examining just the first set of the two sorts administered during

each visit, we found no significant change in the overall number of correct over attempted sorts. However, we did find a significant improvement in the proportion of correct over attempted sorts in this first set in the pre to post comparison, with four additional sorts being completed in the first set alone from pre to post treatment.

3.5 Comparison of change in block design and DKEFS sorting tasks

Contrary to our hypothesis and the results of our previous work (Mackin et al., 2011; Mackin et al., 2016), we found an overall decrease in performance in visual categorization and problem solving pre-to post-treatment using the same DKEFS Sorting Task. As our previous work identified patterns of early dysfunction followed improvement in scores for sorting and categorization (Mackin et al., 2016), we next examined the DKEFS Sorting Task trial by trial and compared it to the WAIS Block Design. We examined time to first sort for the first 10 trials of set one of the DKEFS sorting task pre-to post-treatment to determine whether speed of categorization changed with hoarding treatment. Next, to determine whether any observed changes in reaction time were specific to categorization, we examined the pre-to post-treatment changes in time to completion of the WAIS Block Design task for each trial as a control, as the Block Design is a timed task that also involves visual processing. We hypothesized that participants who improved with hoarding specific treatment would show trial by trial slowing on the DKEFS, but not on the Block Design, reflective of the increased focus on decision making, sorting, and discarding in the treatment process.

As seen in Figure 2, participants showed significant slowing across nearly all trials pre-to post-treatment on the DKEFS as well as on the overall time taken ($t(243)=-4.3$, $p<0.001$). This pattern was not seen in the Block Design; although there was an increase in the overall time taken ($t(240)=-2.9$, $p=0.005$) this effect was driven by slowing on the last two trials only, which are significantly harder than earlier trials, and which fewer people complete.

4. Conclusions

This study is the first to examine the relationships between neurocognitive status and hoarding-specific treatment in a large sample of individuals with HD. As was found by Mackin et al (2016), a large proportion of participants had evidence of clinically significant impairment in at least one cognitive domain, and many showed impairments in multiple domains. Contrary to our hypothesis, neurocognitive status at baseline did not predict treatment outcome. Instead, we saw improvement in neurocognitive function across multiple domains following HD-targeted treatment, independent of treatment type. Specifically, visually mediated processes, including visual memory, visuospatial processing, and visual detection, improved following treatment, while verbal learning and memory, abstract reasoning and decision making and planning (when controlling for neurological injury and drug use) showed no change with treatment. The ability to learn abstract designs decreased post-treatment, as did reaction times for sorting and visual detection, while time to complete a visuospatial processing task did not change. As expected, when hoarding severity was included in the model, pre-to-post variability in neurocognitive performance was no longer significant. Additional studies in larger samples would be required to determine whether

hoarding-specific treatment has an effect on neurocognitive functioning independent of hoarding symptom severity. As reported by Kyrios et al, hoarding severity and hoarding-related cognitions and fears about decision-making are strongly correlated. While in cognitive behavioral model, cognitions and decision-making are psychological rather than neurocognitive constructs, are nevertheless likely to be correlated with or related to underlying neurocognitive changes, as previously demonstrated (Carbonella and Timpano, 2016; Moshier et al., 2016; Phung et al., 2015).

However, our findings are in line both with the cognitive behavioral model of hoarding disorder, as previously outlined by Frost and Hartl, and with previous research examining executive functioning changes in depressed older individuals following both supportive or problem solving therapy, which found that increases in performance were linked to decreases in depression severity (Beaudreau et al., 2015; Mackin et al., 2014). Taken together with the previous work on depression, our findings indicate that targeted treatment of psychiatric complaints and/or participant in treatment can also have an effect on cognitive outcomes.

Unexpectedly, we found mean declines in performance for three visually-mediated cognitive domains, visual learning, visual detection reaction time, and visual categorization, all of which have been consistently reported to be impaired in HD (Ayers et al., 2013; Grisham et al., 2010; Wincze et al., 2007; Woody et al., 2014). For visual detection and visual categorization, the declines in performance appear to be due at least in part to an increase in reaction time pre-to post-treatment. The increase in reaction time for visual detection and for the visual categorization task could potentially be a direct effect of HD treatment. The cognitive behavioral treatment approach used in the parent study emphasizes sorting and discarding as an active component of the therapy and may encourage participants to slow down and consider their choices, thus increasing their reaction times, as was seen on the time to first sore on the DKEFS sorting task in our analyses. This is consistent with previous work that indicates that on this task, increase in sort time is typically thought to reflect lower impulsivity (Delis, 2001a).

The fact that increases in reaction time and poorer performance were observed for the learning, categorization, and detection tasks, but not for a block construction task, which also has a reaction time component but does not involve decision making, suggests that previous poor performance, motivation, or larger visual processing changes are unlikely to account for the observed changes. Similarly, although it is possible that learning effects caused some of the observed improvements in neurocognitive functioning, such effects would be expected to occur globally, affecting all cognitive domains to a similar degree, which was not seen in our data. In addition, we found that when individuals with a history of significant neurological insults and/or drug use were excluded from our sample, the number of incorrect sorts decreased significantly pre to post treatment, and that decision making and planning improvements were no longer significant. These findings indicate that cognitive improvements following treatment cannot simply be related to wide variability in our sample due to inclusion criteria and are more likely to be an effect of treatment.

As individuals with HD learn to discard more effectively in the context of treatment, they may take longer to complete tasks requiring visual discrimination/detection, which could then result in decreased performance on visual learning and categorization tasks. Previous work examining decision making strategies with feedback in HD found that individuals with HD took longer to make decisions and that this increase latency did not improve overall performance or strategy over time (Pushkarskaya et al., 2017; Pushkarskaya et al., 2018), further supporting the idea that therapies targeted at changes in decision making skill may further increase performance latencies.

4.4 Limitations

This study has several limitations. First, there was no control group for performance comparisons. All comparisons were done against normative data and used to convert performance to standard scores, which have limited generalizability. Second, this is a treatment seeking sample of individuals with clinical levels of HD. These individuals are likely more insightful, motivated, and have other features which differentiate them from other samples in previous research. Other health measures were not included in sensitivity analyses and have unknown contributions to CI and individual neurocognitive performance. Previous treatment attempts and medications were not taken into consideration for inclusion. Finally, due to the lack of control sample, we are unable to reliability calculate practice or learning effects from pre-to post-treatment for each domain. Pre to post changes should be viewed with these possible effects in mind, however, due to the clinical makeup and treatment seeking nature of this sample, it is unclear how such effects would be comparable to published normative samples.

4.5 Conclusions & Future Directions

This study supports the previously identified patterns of CI in individuals with HD, and confirms previous work suggesting that executive functioning deficits in individuals with HD are primarily visually mediated (Mackin et al., 2016). This study also reveals new observations with regard to the slowing of response times specifically during categorization tasks following treatment, which may be a direct function of the treatment process. Future research should explore whether changes in categorization-specific response times can be used as a predictor of treatment outcome and/or as an indicator of effective engagement in the treatment process. This study demonstrates that pre-existing neurocognitive dysfunction does not affect treatment outcome, and, most importantly, that visually mediated neurocognitive processes improve with hoarding specific treatment.

Acknowledgements & Disclosures

This work was supported through a Patient-Centered Outcomes Research Institute (PCORI) Assessment of Prevention, Diagnosis, and Treatment Options Program Award (number CE- 1304–6000). The funder had no role in the design and conduct of the study; collection, management, analysis and interpretation of the data; preparation, review or approval of the manuscript or decision to submit the manuscript for publication.

5. REFERENCES

Archer CA, Moran K, Garza K, Zakrzewski JJ, Martin A, Chou C-Y, Uhm SY, Chan J, Gause M, Salazar M, Plumadore J, Smith LC, Komaiko K, Howell G, Vigil O, Bain D, Stark S, Mackin RS,

Eckfield M, Vega E, Tsoh JY, Delucchi KL, Mathews CA, 2018. Relationship between symptom severity, psychiatric comorbidity, social/occupational impairment, and suicidality in hoarding disorder. Journal of Obsessive-Compulsive and Related Disorders.

Ayers CR, Dozier ME, 2015. Predictors of hoarding severity in older adults with hoarding disorder. International psychogeriatrics 27, 1147–1156. [PubMed: 25115688]

Ayers CR, Ly P, Howard I, Mayes T, Porter B, Iqbal Y, 2014. Hoarding severity predicts functional disability in late-life hoarding disorder patients. Int J Geriatr Psychiatry 29, 741–746. [PubMed: 24343998]

Ayers CR, Saxena S, Golshan S, Wetherell JL, 2010. Age at onset and clinical features of late life compulsive hoarding. Int J Geriatr Psychiatry 25, 142–149. [PubMed: 19548272]

Ayers CR, Wetherell JL, Schiehser D, Almklov E, Golshan S, Saxena S, 2013. Executive functioning in older adults with hoarding disorder. Int J Geriatr Psychiatry 28, 1175–1181. [PubMed: 23440720]

Beaudreau SA, Rideaux T, O'Hara R, Arean P, 2015. Does cognition predict treatment response and remission in psychotherapy for late-life depression? The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry 23, 215–219. [PubMed: 25441055]

Bechara A, Damasio AR, Damasio H, Anderson SW, 1994. Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 50, 7–15. [PubMed: 8039375]

Beck A, Steer R, 1993. Beck Anxiety Inventory Harcourt Assessment Inc, San Antonio.

Beck AT, Ward CH, Mendelson MM, Mock JJ, Erbaugh JJ, 1961. An inventory for measuring depression. Archives of General Psychiatry 4, 561–571. [PubMed: 13688369]

Benedict RHB, Schretlen D, Groninger L, Dobraski M, Shpritz B, 1996. Revision of the Brief Visuospatial Memory Test: Studies of normal performance, reliability, and validity. Psychological Assessment 8, 145–153.

Carbonella JY, Timpano KR, 2016. Examining the Link Between Hoarding Symptoms and Cognitive Flexibility Deficits. Behavior Therapy 47, 262–273. [PubMed: 26956657]

Cath DC, Nizar K, Boomsma D, Mathews CA, 2017. Age-Specific Prevalence of Hoarding and Obsessive Compulsive Disorder: A Population-Based Study. The American Journal of Geriatric Psychiatry 25, 245–255. [PubMed: 27939851]

Conners CK, e., 2000. Conners' Continuous Performance Test II: Computer Program for Windows Technical Guide and Software Manual. Multi-Health Systems, Tonawanda, NY.

Delis D, Kaplan E and Kramer J, 2001a. The Delis-Kaplan Executive Function System: Technical Manual. The Psychological Corporation., San Antonio.

Delis D, Kaplan E and Kramer J, 2001b. Delis–Kaplan Executive Function System. The Psychological Corporation, Harcourt Brace & Co., San Antonio, TX.

Frost R, Hartl T, 1996. A cognitive-behavioral model of compulsive hoarding. Behav Res Ther 34, 341–350. [PubMed: 8871366]

Frost RO, Steketee G, Grisham J, 2004. Measurement of compulsive hoarding: saving inventory-revised. Behaviour Research and Therapy 42, 1163–1182. [PubMed: 15350856]

Golden C, Freshwater S, 2002. Stroop Color Word Test: Revised Examiner's Manual. Stoelting Co, Wood Dale, IL.

Grisham JR, Baldwin PA, 2015. Neuropsychological and neurophysiological insights into hoarding disorder. Neuropsychiatr Dis Treat 11, 951–962. [PubMed: 25897231]

Grisham JR, Brown TA, Savage CR, Steketee G, Barlow DH, 2007. Neuropsychological impairment associated with compulsive hoarding. Behav Res Ther 45, 1471–1483. [PubMed: 17341416]

Grisham JR, Frost RO, Steketee G, Kim HJ, Hood S, 2006. Age of onset of compulsive hoarding. J Anxiety Disord 20, 675–686. [PubMed: 16112837]

Grisham JR, Norberg MM, Williams AD, Certoma SP, Kadib R, 2010. Categorization and cognitive deficits in compulsive hoarding. Behav Res Ther 48, 866–872. [PubMed: 20542489]

Harris J, 2010. Household hoarding and residentile fires, Int Congr Appl Psychol, Melbourne, Australia.

Hwang JP, Tsai SJ, Yang CH, Liu KM, Lirng JF, 1998. Hoarding behavior in dementia. A preliminary report. The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry 6, 285–289. [PubMed: 9793576]

Kim HJ, Steketee G, Frost RO, 2001. Hoarding by elderly people. Health Soc Work 26, 176–184. [PubMed: 11531193]

Mackin RS, Arean PA, Delucchi KL, Mathews CA, 2011. Cognitive functioning in individuals with severe compulsive hoarding behaviors and late life depression. Int J Geriatr Psychiatry 26, 314–321. [PubMed: 21319334]

Mackin RS, Nelson JC, Delucchi K, Raue P, Byers A, Barnes D, Satre DD, Yaffe K, Alexopoulos GS, Arean PA, 2014. Cognitive outcomes after psychotherapeutic interventions for major depression in older adults with executive dysfunction. The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry 22, 1496–1503. [PubMed: 24378255]

Mackin RS, Vigil O, Insel P, Kivowitz A, Kupferman E, Hough CM, Fekri S, Crothers R, Bickford D, Delucchi KL, Mathews CA, 2016. Patterns of Clinically Significant Cognitive Impairment in Hoarding Disorder. Depress Anxiety 33, 211–218. [PubMed: 26474146]

Marx MS, Cohen-Mansfield J, 2003. Hoarding behavior in the elderly: a comparison between community-dwelling persons and nursing home residents. International psychogeriatrics 15, 289–306. [PubMed: 14756164]

Mathews CA, Mackin RS, Chou CY, Uhm SY, Bain LD, Stark SJ, Gause M, Vigil OR, Franklin J, Salazar M, Plumadore J, Smith LC, Komaiko K, Howell G, Vega E, Chan J, Eckfield MB, Tsoh JY, Delucchi K, 2018. Randomised clinical trial of community-based peer-led and psychologist-led group treatment for hoarding disorder. BJPsych open 4, 285–293. [PubMed: 30083381]

Moshier SJ, Wootton BM, Bragdon LB, Tolin DF, Davis E, Dimauro J, Diefenbach GJ, 2016. The relationship between self-reported and objective neuropsychological impairments in patients with hoarding disorder. Journal of Obsessive-Compulsive and Related Disorders 9, 9–15.

Nelson H, 1982. The National Adult Reading Test (NART): Test Manual. MFER-Nelson, Windsor.

Nordsletten AE, Fernández de la Cruz L, Pertusa A, Reichenberg A, Hatch SL, Mataix-Cols D, 2013. The Structured Interview for Hoarding Disorder (SIHD): Development, usage and further validation. Journal of Obsessive-Compulsive and Related Disorders 2, 346–350.

Phung PJ, Moulding R, Taylor JK, Nedeljkovic M, 2015. Emotional regulation, attachment to possessions and hoarding symptoms. Scand J Psychol 56, 573–581. [PubMed: 26183596]

Pushkarskaya H, Tolin D, Ruderman L, Henick D, Kelly JM, Pittenger C, Levy I, 2017. Value-based decision making under uncertainty in hoarding and obsessive- compulsive disorders. Psychiatry Res 258, 305–315. [PubMed: 28864119]

Pushkarskaya H, Tolin DF, Henick D, Levy I, Pittenger C, 2018. Unbending mind: Individuals with hoarding disorder do not modify decision strategy in response to feedback under risk. Psychiatry Res 259, 506–513. [PubMed: 29154203]

Samuels JF, Bienvenu OJ, Grados MA, Cullen B, Riddle MA, Liang KY, Eaton WW, Nestadt G, 2008. Prevalence and correlates of hoarding behavior in a community-based sample. Behav Res Ther 46, 836–844. [PubMed: 18495084]

Shapiro AM, Benedict RH, Schretlen D, Brandt J, 1999. Construct and concurrent validity of the Hopkins Verbal Learning Test-revised. The Clinical neuropsychologist 13, 348–358. [PubMed: 10726605]

Sheehan DV, Lecrubier Y Fau - Sheehan KH, Sheehan Kh Fau - Amorim P, Amorim P Fau - Janavs J, Janavs J Fau - Weiller E, Weiller E Fau - Hergueta T, Hergueta T Fau - Baker R, Baker R Fau - Dunbar GC, Dunbar GC, 1998. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. Journal of Clinical Psychiatry 59, 22–33.

Smith A, 2002. Symbol Digit Modalities Test. Western Psychological Services Los Angeles, CA.

Steketee G, Frost RO, Tolin DF, Rasmussen J, Brown TA, 2010. Waitlist-controlled trial of cognitive behavior therapy for hoarding disorder. Depress Anxiety 27, 476–484. [PubMed: 20336804]

Swanson JM, Kraemer HC, Hinshaw SP, Arnold LE, Conners CK, Abikoff HB, Clevenger W, Davies M, Elliott GR, Greenhill LL, Hechtman L, Hoza B, Jensen PS, March JS, Newcorn JH, Owens EB, Pelham WE, Schiller E, Severe JB, Simpson S, Vitiello B, Wells K, Wigal T, Wu M, 2001. Clinical

relevance of the primary findings of the MTA: Success rates based on severity of ADHD and ODD symptoms at the end of treatment. Journal of the American Academy of Child and Adolescent Psychiatry 40, 168–179. [PubMed: 11211365]

Tolin DF, Frost RO, Steketee G, 2007. Buried in treasures: Help for compulsive acquiring, saving, and hoarding. Oxford University Press, New York.

Tolin DF, Frost RO, Steketee G, Muroff J, 2015. Cognitive behavioral therapy for hoarding disorder: a meta-analysis. Depress Anxiety 32, 158–166. [PubMed: 25639467]

Tolin DF, Villavicencio A, Umbach A, Kurtz MM, 2011. Neuropsychological functioning in hoarding disorder. Psychiatry Res 189, 413–418. [PubMed: 21764138]

Tolin DF, Witt ST, Stevens MC, 2014. Hoarding disorder and obsessive-compulsive disorder show different patterns of neural activity during response inhibition. Psychiatry Res 221, 142–148. [PubMed: 24389161]

Uhm SY, Tsoh JY, Mackin RS, Gause M, Chan J, Franklin J, Eckfield M, Salazar M, Vigil O, Bain D, Stark S, Vega E, Delucchi KL, Mathews CA, 2016. Comparison of a peer facilitated support group to cognitive behavior therapy: Study protocol for a randomized controlled trial for hoarding disorder. Contemporary clinical trials 50, 98–105. [PubMed: 27444427]

Wechsler D, 1999. Wechsler Abbreviated Scale of Intelligence. Psychological Corporation, San Antonio, TX.

Williams M, Viscusi JA, 2016. Hoarding Disorder and a Systematic Review of Treatment with Cognitive Behavioral Therapy. Cognitive behaviour therapy 45, 93–110. [PubMed: 26795499]

Wincze JP, Steketee G, Frost RO, 2007. Categorization in compulsive hoarding. Behav Res Ther 45, 63–72. [PubMed: 16530724]

Woody SR, Kellman-McFarlane K, Welsted A, 2014. Review of cognitive performance in hoarding disorder. Clin Psychol Rev 34, 324–336. [PubMed: 24794835]

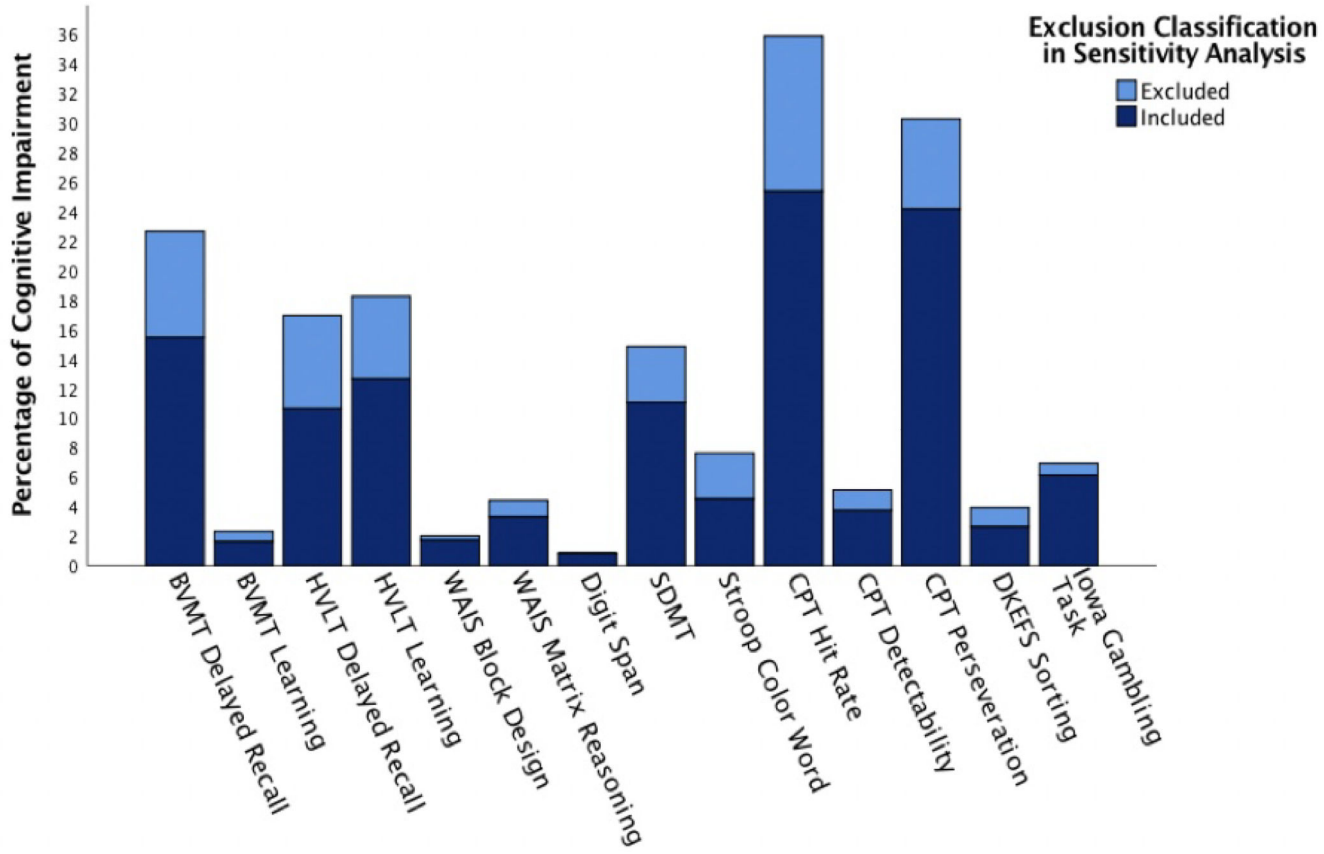
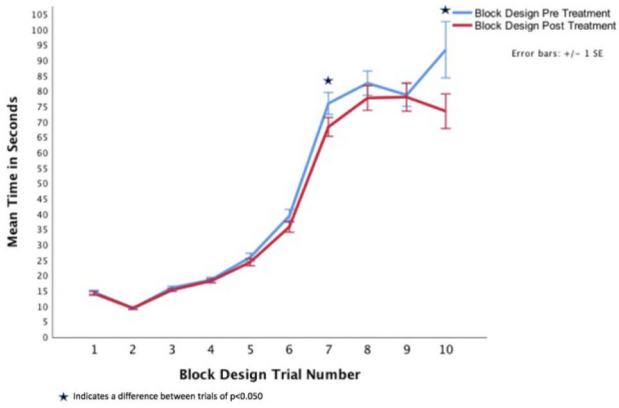


Figure 1 :

Rates of Cognitive Impairment in the Overall Treatment Sample. The full bars represent the entire treatment sample. The lighter blue represents the number of people excluded in the sensitivity analysis and the darker blue represents those who met sensitivity inclusion criteria.



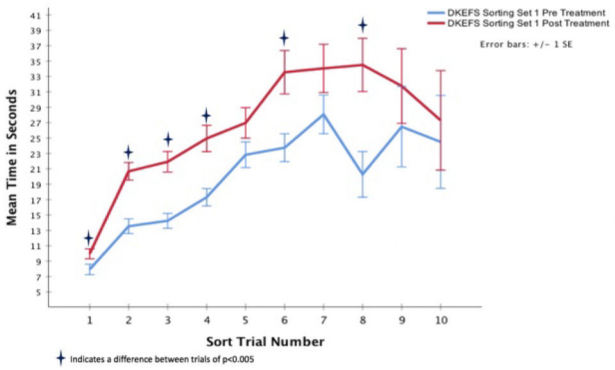


Figure 2:

Trial Comparisons of WAIS Block Deign Task and the DKEFS Sorting Task Pre and Post Treatment. The graph on the right is the trial by trial time performance for the Block Design task with blue indicating pre-treatment performance times and red indicating post-treatment. The right figure is the same trial by trial performance times for the Sorting Task pre and post treatment. Significance levels indicated below are the minimum significance seen in comparison testing.

Table 1:

Neurocognitive Battery Administered Pre/Post Treatment

Domain	Scaled Score	Raw Score
Estimated IQ	National Adult Reading Test (NART) Estimated Full Scale IQ	NART Number of Correct Words
Verbal Learning & Memory	Hopkins Verbal Learning Test (HVLT) Learning Score	HVLT Learning Score
	HVLT Delayed Recall Score	HVLT Delayed Recall Score
Visual Learning & Memory	Brief Visuospatial Memory Test- Revised (BVMT) Delayed Recall Score	BVMT Delayed Recall Score
	BVMT Learning Score	BVMT Learning Score
Visuospatial Processing	Wechsler Adult Intelligence Test - IV (WAIS-IV) Block Design Task Total Score	Block Design Total Score
Abstract Reasoning	WAIS-IV Matrix Total Score	Matrix Total Score
Attention/Working Memory	WAIS-IV Digit Span Total Score	Digit Span Total Score
Information Processing Speed	Symbol Digit Modalities Test (SDMT) Total Correct Score	SDMT Total Correct Score
	Stroop Color Word Score	Stroop Color Word Score
Visual Detection & Perseveration	Conners Continuous Performance Test- 2nd Edition (CPT) Hit Reaction Time	CPT Hit Reaction Time
	CPT Detectability Total Correct	CPT Detectability Total Correct
	CPT Variability Score	CPT Variability Score
	CPT Perseverations Total	CPT Perseverations Total
Visual Categorization & Problem Solving	Delis-Kaplan Executive Functioning System (DKEFS) Sorting Task Total Correct Sorts (Both Sets)	DKEFS Sorting Time of 1st Sort
		DKEFS Sorting Total Correct Sorts
		DKEFS Sorting Total Incorrect Sorts
		DKEFS Sorting Total Incorrect Sorts
		DKEFS Sorting Correct Over Attempted (All)
		DKEFS Sorting Correct Over Attempted (Set 1)
Decision Making & Planning	Iowa Gambling Task (IGT) Net Total Score	IGT Net Total Score

Table 2.

Baseline Participant Demographics

	Total Pre-Treatment Sample (N=318)	**Treatment Complete (*N*=243)**	**Treatment Drop-Outs (*N*=75)**	**Treatment to Drop-Out Comparison *t/x²*; *p*-value**
Gender (% female)	74.4	77.0	65.3	5.27; 0.072
Mean age (SD)	59.6 (10.5)	60.0 (10.5)	57.9 (10.4)	1.53; 0.127
Mean Education (SD)	15.3 (2.3)	15.5 (2.3)	14.7 (2.4)	2.73; 0.007**
Race (% white)	60.6	62.3	56.2	0.88; 0.349
NART Est. Mean IQ (SD)	115.8 (6.5)	116.8 (5.7)	112.7 (7.9)	4.78; <0.0001***
Mean SI-R Total (SD)	65.5 (11.7)	64.9 (11.7)	67.5 (11.3)	−1.69; 0.093
Mean SNAP Total (SD)	16.3 (13.2)	15.5 (12.8)	18.3 (14.5)	−1.57; 0.117
Mean BAI Score (SD)	13.6 (9.4)	16.7 (12.0)	19.4 (10.9)	−1.74; 0.083
Mean BDI Score (SD)	15.9 (14.1)	19.3 (12.9)	20.5 (12.5)	−0.74; 0.461

* significant at <0.05

** significant at <0.01

*** significant at <0.001

Table 3.

Pre to Post Treatment Neurocognitive Raw Score Performance

Domain	Test	N	Pre-Mean (SD)	Post-Mean (SD)	*t; p*-value
Verbal Learning & Memory	HVLT Learning	241	25.7 (4.7)	25.6 (4.9)	0.25; 0.803
	HVLT Delayed Recall	241	9.2 (2.3)	9.2 (2.2)	−0.13; 0.899
Visual Learning & Memory	BVMT Delayed Recall	242	8.2 (3.0)	8.8 (2.8)	−4.0; <0.001 *** †
	BVMT Learning	242	4.5 (2.1)	4.0 (2.0)	3.0; 0.003 ** †
Visuospatial Processing	Block Design	242	37.5 (11.8)	40.2 (12.5)	−5.3; <.001 *** †
Abstract Reasoning	Matrix	205	17.5 (4.7)	17.9 (4.5)	−1.9; 0.063
Attention/Working Memory	Digit Span	189	19.2 (3.8)	19.7 (4.0)	−2.1; 0.037 * †
Information Processing Speed	SDMT	234	49.7 (10.2)	51.4 (9.7)	−3.8; <0.001 *** †
	Stroop Color Word	240	37.8 (9.3)	39.5 (9.4)	−4.2; <0.001 *** †
Visual Detection/Perseveration	CPT Hit RT	232	460.9 (62.6)	471.3 (66.2)	−3.1; 0.002 ** †
	CPT Detectability	232	0.82 (0.39)	0.95 (0.45)	−5.3; <0.001 *** †
	CPT Variability	232	9.9 (9.1)	9.1 (9.9)	1.1; 0.266
	CPT Perseverations	67	2.5 (7.8)	1.9 (9.7)	0.4; 0.677
Visual Categorization & Problem Solving	DKEFS Time 1st Sort	244	7.8 (7.3)	19.0 (14.0)	−15.3; <0.001 *** †
	DKEFS Total Correct Sorts	243	9.3 (2.5)	8.7 (2.8)	3.8; <0.001 *** †
	DKEFS Total Incorrect Sorts	243	2.9(2.9)	2.5 (2.8)	1.6; 0.112 †
	DKEFS Correct Over Attempted (All)	243	0.80 (0.15)	0.81 (0.16)	−0.7; 0.481
	DKEFS Correct Over Attempted (Set 1)	244	0.79 (0.20)	0.85 (0.18)	−4.4; <0.001 ***
Decision Making & Planning	IGT Net Total Score	182	20.7 (29.6)	25.9 (32.2)	−2.0; 0.045 *

* significant at <0.05

** significant at <0.01

*** significant at <0.001

† significant in sensitivity analysis group at <0.050