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The Driving Behavior Survey as a Measure of Behavioral Stress Responses to MVA-related PTSD

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Abstract

Numerous treatments are available that address the core symptoms of posttraumatic stress disorder (PTSD). However, there are a number of related behavioral stress responses that are not assessed with PTSD measures, yet these behavioral stress responses affect quality of life. The goal of the current study was to investigate whether a recently developed measure of behavioral stress response, the Driving Behavior Survey (DBS), was sensitive to change associated with treatment among a group of participants diagnosed with PTSD. The DBS indexes anxious driving behavior, which is frequently observed among individuals with motor vehicle accident-related PTSD. Participants ($n = 40$) were racially-diverse adults (M age = 40.78, 63% women) who met diagnostic criteria for motor vehicle accident-related PTSD. Hierarchical linear modeling analyses indicated that participants who were assigned to a brief, exposure-based intervention displayed significant reductions on the DBS subscales relative to participants assigned to the waitlist control condition ($r = .41-.43$). Moreover, mediational analyses indicated that the observed reductions on the DBS subscales were not better accounted for by reductions in PTSD. Taken together, these findings suggest that the DBS subscales are sensitive to changes associated with PTSD treatment and can be used to augment outcome measures in PTSD treatment trials

Keywords

PTSD; stress response; exposure; treatment; driving; assessment

Posttraumatic stress disorder (PTSD) is a chronic and debilitating disorder with a lifetime prevalence rate of up to 25% (Hidalgo & Davidson, 2000). PTSD has a clear impact on

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health, health care service utilization, and general functioning. Although a variety of traumatic events can result in PTSD (e.g., sexual assault, combat, physical assault, natural disaster), motor vehicle accidents (MVAs) are believed to be the leading cause of PTSD in Western societies (e.g., Beck & Coffey, 2007; Blanchard & Hickling, 2004; Kessler, Sonnega, Bromet, & Hughes, 1995; Norris, 1992). Over three million Americans are injured in MVAs each year (Blanchard & Hickling, 2004). Prospective studies of injured MVA survivors have reported PTSD rates that range from 8% (Mayou, Bryant, & Duthie, 1993) to approximately 40% (Blanchard & Hickling, 2004), with a reasonable estimate being around 25% (see Blanchard & Hickling, 2004, for a review). MVA-related PTSD and associated difficulties, such as driving anxiety and avoidance, may persist for years (e.g., Mayou et al. 1993; Mayou, Tyndel, & Bryant, 1997).

Given the public health significance of MVA-related PTSD, it is important that treatments address both the core symptoms of PTSD as well as associated behavioral stress responses, such as driving anxiety and avoidance behaviors. Recognition of the importance of behavioral stress responses began with the “fight or flight” model proposed by Cannon in 1929. Over the last few decades, a number of different behavioral stress responses have been recognized, including the freeze response (Gray & McNaughton, 2000) as well as the attachment or “tend and befriend” (Taylor, 2006) response. Importantly, while playing an adaptive role in survival during a traumatic event, such behaviors commonly become overgeneralized to circumstances beyond the immediate acute stress environment and cause impairment (Lang et al., 2003). Unfortunately, such impairing behavioral stress responses are often overlooked in the PTSD and anxiety treatment literature.

Most mental health treatments target reductions of core PTSD symptoms. However, in many cases, it is behavioral stress responses that cause impairment (e.g. Cloitre, Miranda, Stovall-McClough, & Han, 2005) and, in turn, may lead an individual to seek treatment. It is assumed that behavioral stress responses will decrease with the alleviation of symptom frequency and intensity (Koenen, Goodwin, Struening, Hellman, & Guardino, 2003), although this assumption is rarely examined empirically.

MVA-related PTSD is an ideal target for the identification of specific behavioral stress responses that may cause impairment, as the trauma is directly associated with a discrete area of behavioral functioning (i.e., traveling on roadways). People who have experienced MVAs are likely to experience posttraumatic stress symptoms most acutely in the context of driving a vehicle. These types of behavioral responses have been noted in clinical writings (e.g., Beck & Coffey, 2005), and can include a number of nuanced anxiety-related behaviors such as driving significantly below the speed limit, refusing to make a right-hand turn, or becoming extremely angry at other drivers who are deemed to be unsafe. As previously described, these types of behavioral stress responses can negatively impact quality of life, but fall outside of specific PTSD symptoms. To date, there has been limited empirical work examining the impact of PTSD treatment on the frequency of behavioral stress responses that are specific to particular categories of traumatic events.

Although a number of studies have examined the efficacy of treatment for MVA-related PTSD (e.g., Beck, Coffey, Foy, Keane, & Blanchard, 2009; Blanchard et al., 2003; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005; Taylor, Fedoroff, Koch, Thordarson, Fecteau, & Nicki, 2001), decrease in severity of formal, DSM-based symptoms have been the primary treatment outcome. Many of these studies have included secondary outcome measures, such as depression, anxiety, and global functioning (e.g., Beck et al., 2009; Blanchard et al., 2003; Taylor et al., 2001), but changes in domains of behavioral stress responses, such as driving-related anxiety and avoidance, have not been examined. Clapp and colleagues developed a brief and psychometrically sound self-report measure of driving-

related anxious behaviors called the Driving Behavior Survey (DBS; Clapp, Olsen, Beck, et al., 2011). This scale was developed to assess anxious driving behavior, operationalized as an increase, decrease, or general disorganization of behavior occurring as a consequence of anxiety during operation of a motor vehicle. The DBS is an ideal instrument to index behavioral stress responses because it assesses a broad range of specific problematic behaviors that are relevant to individuals diagnosed with MVA-related PTSD (e.g., Blanchard & Hickling, 2004; Mayou et al., 1997).

The DBS indexes three areas of behavioral stress responses in the domain of driving. The exaggerated safety/caution subscale (ESCB) captures excessively cautious behaviors believed to increase perceptions of control (e.g. driving far below the posted speed limit). These behaviors have long been noted in the clinical literature on anxious driving (e.g. Taylor & Koch, 1995) and may be categorized broadly under the umbrella of avoidance. As such, behavioral stress in this domain is thought to facilitate the long-term maintenance of anxiety through down-regulation of the anxious response (Clark, 1999; Salkovskis, 1991). Excessively cautious behaviors also are believed to increase the risk of another MVA due to the violation of normative driving behavior.

The anxiety-based performance deficits subscale (ABPD) focuses on driving performance deficits (e.g., difficulty staying in the correct lane) that are commonly discussed in the traffic and transportation safety literatures (e.g., Dula, Adams, Miesner, & Leonard, 2010; Kontogiannis, 2006; Matthews et al., 1998). Behaviors included within this subscale are thought to be a result of interference due to the increased cognitive load of anxious thoughts and behaviors (e.g. rumination or peripheral monitoring).

Lastly, the hostile/aggressive driving behaviors subscale (HAB) includes behavioral stress responses of “fight” and captures hostile behaviors. These behaviors receive limited attention in the literature despite the finding that trait-level anxiety has been associated with anger and aggressive violations (Deffenbacher, Huff, Lynch, Oetting, & Salvatore, 2000; Lucidi et al., 2010; Shahar, 2009; Ulleberg, 2002). Behaviors on this subscale (e.g. shouting, honking, and aggressive gesturing) are hypothesized to be a result of anxiety decreasing the threshold for aggressive responses to traffic stress (Clapp, Olsen, Beck, et al., 2011; Clapp, Olsen, Danoff-Burg, et al., 2011).

The current study draws from a randomized controlled trial examining the efficacy of a brief, exposure-based treatment for MVA-related PTSD, referred to as Written Exposure Therapy (WET). As reported elsewhere (Sloan, Marx, Bovin, Feinstein, & Gallagher, 2012), participants who were randomly assigned to WET exhibited significant reductions in PTSD symptom severity relative to participants assigned to a waitlist (WL) control condition. The goal of the present study was to examine whether changes resulting from WET generalized to changes in behavioral stress responses in the form of reduction in driving-related anxious behaviors. We expected that participants assigned to WET would show improvement in each of the DBS subscales relative to participants assigned to the control condition.

The second goal of the study was to demonstrate that the behavioral stress responses indexed by the DBS subscales are distinct symptoms from PTSD. To accomplish this goal, we conducted correlation analyses between the three subscales of the DBS and specific PTSD symptom clusters. We hypothesized that associations between the DBS subscales and the PTSD symptom clusters would be small to medium. Next, mediational analyses were conducted to evaluate whether changes on the three DBS subscales were fully accounted for by changes in PTSD symptoms. As we hypothesized that the DBS subscales index unique information, we did not expect to find that the changes on the DBS subscales would be fully accounted for by changes in PTSD symptoms.

Method

Participants

Participants were recruited from the Boston, MA area using flyer postings and public service announcements of the treatment study. To be included in the study, individuals needed to be at least 18 years old and have a primary diagnosis of PTSD related to an MVA (occurring at least three months prior). Exclusion criteria included current psychotic diagnosis, organic mental disorder, current substance dependence, unstable bipolar disorder, English illiteracy, and high risk for suicidal behavior (e.g., suicide attempt in past two months).

Researchers were contacted by 145 individuals interested in the study. Of these, 68 did not qualify based on an initial phone screen. The remaining 67 individuals presented for an initial assessment to determine study eligibility criteria. The Clinician Administered PTSD Scale (CAPS; Weathers, Keane, & Davidson, 2001) was administered at the initial in-person assessment to evaluate presence and severity of PTSD. The Structured Clinical Interview for DSM-IV Axis I Disorders with Psychotic Screen (SCID; Spitzer, Williams, Gibbon, & First, 1994) also was administered to evaluate other inclusion/exclusion diagnostic criteria. Of the 67 individuals who completed the initial assessment, three declined enrollment, two had significant cognitive impairment, five met diagnostic criteria for substance dependence, three met diagnostic criteria for psychosis, two met diagnostic criteria for current bipolar disorder that was unstable, and six did not meet current diagnostic criteria for PTSD. Forty-six individuals were ultimately found to satisfy study inclusion/exclusion criteria based on the in-person diagnostic assessment. Six of the 46 participants were not current drivers and therefore did not complete the DBS measure, resulting in a final sample of 40 participants in the current report (see Sloan et al., 2012 for details).

Of the 40 participants included in the current study, the mean age was 40.78 ($SD = 13.26$) and 25 (63%) were women. Racial background was diverse with 37.5% identifying as African-American, 30% Caucasian, 10% Hispanic, 2.5% Asian-American, and 15% as “other” or having a mixed racial background.

Approximately half of the individuals included in the current study ($n = 19$) were randomized to receive WET (see Sloan et al., 2012 for full details) and half ($n = 21$) were randomized to the minimal contact, WL control condition. Two participants discontinued treatment but presented for reassessment at six weeks post-randomization (or post-treatment for WET participants) and 18 weeks post randomization (or three month post-treatment for WET participants). In the larger RCT, a 30 week assessment was also included only for participants randomized to WET. We did not include the 30 week assessment in the current study because of our interest in examining group differences.

Measures

The *Driving Behavior Survey* (DBS; Clapp, Olsen, Beck, et al., 2011) was used to measure anxious driving behavior. This measure consists of 21-items that index the frequency of anxious driving behavior across three domains: exaggerated safety/caution behavior (ESCB), anxiety-based performance deficits (ABPD), and hostile/aggressive behavior (HAB). Items are rated on a 1 to 7 Likert-type scale with higher mean scores indicating greater frequency of anxious behavior. As previously noted, the DBS subscales have shown strong internal validity and consistency as well as convergent associations in prior research with both college and treatment-seeking samples (Clapp, Olsen, Beck, et al., 2011; Clapp, Olsen, Danoff-Burg, et al., 2011; Clapp, Baker, Litwack, Sloan, & Beck, in press). DBS subscales were calculated by averaging the scores across the seven items in each behavioral dimension. In the current sample, all three scales showed good to excellent internal

consistency ($\alpha = .85 - .93$) and good test-retest reliability between post treatment assessments ($r = .80 - .85$).

The *Clinician-Administered PTSD Scale* (CAPS; Weathers et al., 2001) was used to establish PTSD diagnosis related to the index MVA and as a measurement of PTSD symptom severity. The CAPS consists of the 17 cardinal symptoms of PTSD defined by the *DSM-IV* (American Psychiatric Association, 1994), with clinicians rating the frequency and intensity of each symptom on a 0-4 Likert-type scale. For the current study, symptoms with frequency ratings 1 and intensity ratings 2 were counted towards determining PTSD diagnostic status (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). Individuals meeting *DSM-IV* symptom criteria and having a total CAPS severity score of at least 40 received a formal PTSD diagnosis (Weathers et al., 2001). CAPS scores demonstrate strong psychometric properties with one-week test-retest reliability ranging between .90 and .96 (Weathers et al., 2001). As described by Sloan and colleagues (2012) inter-rater reliability for PTSD diagnosis in this sample was excellent ($\kappa = .94$). In addition to total score, scores for criteria B (re-experiencing), C (avoidance and numbing), and D (hypervigilance) were calculated by summing the frequency and intensity for all the symptoms in each cluster.

Treatment

WET consisted of 5 weekly sessions in which participants were instructed to write about their index trauma event with as much emotion and detail as possible. The first session was approximately one hour in duration and consisted of providing psychoeducation about PTSD along with a treatment rationale. The role of avoidance of reminders of the trauma event (both thoughts and behaviors) was emphasized within the psychoeducation. Participants were also informed that engaging in these avoidance behaviors contributes to the maintenance of PTSD symptoms. The rationale for confronting trauma memories was then presented, and WET was described as one method for confronting such memories. Participants were then given general instructions for writing about the trauma event as well as specific instructions for the first session. These instructions were read to the participants, and then the printed instructions were left with the participants while 30 minutes of writing alone was completed. The therapists returned to the room after 30 minutes, checked in with the participants about how the writing session went for them, and then encouraged the participants to allow themselves to have whatever thoughts, feelings or images that came to mind if they found themselves thinking about the trauma event during the course of the upcoming week. Aside from this general instruction, no assignments were given to the participant to complete in-between sessions. The remaining four sessions consisted of the participants completing 30 minutes of writing about the traumatic event. Instructions for each writing session varied slightly. Consistent with the first day of writing, the therapists returned to the room after 30 minutes to let participants know it was time to stop, and checked in with them regarding how the writing sessions went. Therapist contact time with participants was generally 10 minutes per session.¹

Data Analytic Strategy

Our first hypothesis was that participants assigned to the treatment condition would display significant reductions in DBS subscales following treatment relative to participants assigned to the WL condition. To test this hypothesis, hierarchical linear modeling (HLM; Raudenbush & Bryk, 2002) was conducted for each of the three DBS subscales (ABPD, ESCB, & HAB). A random slopes model was specified in which Time (level 1) and the Condition $\times$ Time interaction was included as a predictor. Time was coded in terms of the wave of measurement (baseline: 0; 6-weeks post-randomization: 1; 18-weeks post-

¹The WET protocol is available upon request from the corresponding author.

randomization: 2) and was modeled as a random effect in order to allow for the possibility that trajectories of change varied between individuals.

The level 1 and level 2 models can therefore be written as:

Level-1 Model

$$y_{ij} = \beta_0 + \beta_1 * (\text{Time}_{ij}) + e_{ij}$$

Level-2 Model

$$\begin{aligned}\beta_0 &= \gamma_{00} + v_0 \\ \beta_1 &= \gamma_{10} + \gamma_{11} * (\text{Condition}_j) + v_1\end{aligned}$$

HLM models were conducted using HLM 6.0 (Raudenbush, Bryk, & Congdon, 2004). The coefficient of interest was γ_{11} , which represented the Condition $\times$ Time interaction, and indicated the significance of differential effects between treatments over time. Effect sizes for the Condition $\times$ Time interactions were calculated by converting the t-ratios into

correlations using the following formula: $r = \sqrt{t^2 / (t^2 + df)}$ (Raudenbush & Bryk, 2002). Our prediction would be supported if we observed significant Time $\times$ Condition effects.

The second goal of the study was to demonstrate that the behavioral stress responses indexed by the DBS subscales were distinct from PTSD symptoms, as indexed by the CAPS. To accomplish this goal, we conducted correlational analyses between the DBS subscales and PTSD symptom severity utilizing data from the 6-week and 18-week assessment periods. These assessment periods were selected in order to increase the range of scores on the DBS subscales and CAPS given the restricted range of scores on these two measures at the baseline assessment. The increased range on the measures increased the power of the analyses and increased the likelihood that the observed associations reflect the true relationship between the variables. We expected to observe small to medium associations between the DBS subscales and the PTSD symptom clusters on the CAPS.

The second method that we used to demonstrate that the DBS subscales capture distinct responses from the PTSD measure was to conduct mediational analyses. We expected that changes in each of the three DBS subscales would not be fully mediated by changes in PTSD symptom severity. The mediation analyses used condition (WL: 0, WET: 1) as the predictor, baseline to 6-week post randomization changes in CAPS scores (PTSD symptom change) as the mediator, and baseline to 18-week post randomization changes in DBS subscales as the outcome in order to maintain the temporal precedence of the hypothesized mediator.

Results

Sensitivity to Treatment

The results of the HLM analyses are presented in Table 1, and the summary statistics for the DBS subscales as a function of condition and time are presented in Table 2. As expected, a significant Condition $\times$ Time interaction was observed for all three DBS subscales. Consistent with our predictions, participants assigned to WET reported significantly greater decreases on all three DBS subscales relative to participants assigned to the WL condition.

Although significant treatment effects were observed, relative increases in DBS subscales were noted among individuals assigned to the control condition. A series of post-hoc *t*-tests exploring the significance of within-group DBS subscale change from pre-treatment to 18 weeks post randomization were conducted to examine if the previously described HLM results were driven by increases in the scores of the WL condition. The full results of the six planned *t*-tests can be found in Table 3. The WET condition displayed reliable reductions on all three DBS subscales ($p = .01-.03$).

DBS subscales distinction from PTSD symptom severity

Table 4 provides summary statistics for PTSD symptom severity as measured by the CAPS as a function of time and condition and Table 5 displays the correlations of the DBS subscales and PTSD symptom clusters as a function of time. As expected, the correlational analyses indicated small to moderate associations between the DBS subscales and the PTSD symptom severity scales. Specifically, we found that at 6 weeks post-baseline the HAB scale displayed moderate positive correlations with Cluster B, C, and D PTSD symptoms but these associations were not significant at 18 weeks post-baseline. The ESCB scale only displayed a significant positive association with Cluster B PTSD symptoms at 6 weeks post baseline. The ABPD scale did not display significant association with PTSD symptom clusters at 6 weeks post-baseline; however, at 18 weeks post-baseline the ABPD scale was significant associated with greater PTSD symptom severity as well as greater Cluster B, and Cluster D PTSD symptom severity.

As expected, the results across the three DBS subscales indicate that there is likely a small to medium relationship with PTSD symptomology. Although the pattern of significant findings differed between the two time points, none of the correlations between DBS subscales and PTSD symptom severity were significantly differed between time points ($p > .25$).

We next conducted mediational analyses to demonstrate that the decreases observed for each of the three DBS subscales were not fully accounted for by decreases in PTSD symptoms. Multiple regression analyses were run for each of the three DBS subscales to investigate if the traditional prerequisites for mediation (Baron & Kenny, 1986) were met, followed by an estimate of the indirect effect through the bootstrapping method (Preacher & Hayes, 2008; $N = 5000$ samples). For the ABPD subscale, there was a significant relationship between treatment condition and ABPD ($b = 1.37$, $SE_b = .51$, $t = 2.71$, $p = .01$), although there was not a significant relationship between ABPD and CAPS change ($b = 0.01$, $SE_b = .01$, $t = 1.27$, $p = .21$) and treatment condition remained significant after adding CAPS change in a combined model ($b = 2.45$, $SE_b = .86$, $t = 2.84$, $p < .01$). Further, the bootstrapped estimate of the indirect effect was not significant ($b = -1.08$, $SE_b = .68$, CI $[-2.43 - 0.25]$). Thus, change in ABPD subscale was not fully mediated by change in PTSD symptom severity.

For the ESCB subscale, there was a significant relationship between treatment condition and ESCB ($b = 1.48$, $SE_b = .50$, $t = 2.99$, $p < .01$). However, there was not a significant relationship between ESCB and CAPS change ($b = 0.016$, $SE_b = .01$, $t = 1.62$, $p = .11$) and treatment condition remained significant after adding CAPS change in a combined model ($b = 2.34$, $SE_b = .86$, $t = 2.74$, $p < .01$). Further, the bootstrapped estimate of the indirect effect was not significant ($b = -0.82$, $SE_b = .66$, CI $[-2.17 - 0.44]$), which indicates that the change observed for the ESCB subscale was not fully mediated by changes in PTSD symptom severity.

Similar findings emerged for the HAB subscale. Specifically, a significant relationship between treatment condition and HAB was observed ($b = 1.32$, $SE_b = .50$, $t = 2.62$, $p = .01$), although there was not a significant relationship between HAB and CAPS change ($b =$

0.012, $SE_b = .01$, $t = 1.25$, $p = .22$) and treatment condition remained significant after adding CAPS change in a combined model ($b = 2.33$, $SE_b = .86$, $t = 2.70$, $p = .01$). Moreover, the bootstrapped estimate of the indirect effect was not significant ($b = -1.00$, $SE_b = .75$, CI [-2.49 - 0.48]).

Taken together, the mediation analyses indicated that decreases in the three DBS subscales for the WET condition were not significantly mediated by reductions in PTSD symptoms. These findings, in combination with the correlational analyses, underscore that the three DBS subscales index behavioral stress responses that are not fully captured by PTSD symptoms.

Discussion

The aim of this study was to determine whether domains of anxious driving behavior, as assessed by the DBS subscales, served as treatment-sensitive indices of behavioral stress responses among individuals with MVA-related PTSD. Whereas cognitive-behavioral interventions demonstrate reliable improvements in formal symptoms of PTSD, many studies do not exhibit improved functioning across a range of life domains (e.g., Frisch, Cornell, Villanueva, & Retzlaff, 1992; Schnurr et al., 2003). This has generated a growing interest in more comprehensive assessments of post-trauma functioning (Frueh, Turner, Beidel, & Cahill, 2001; Holowka & Marx, 2012). Data from the present study support the utility of anxious driving behavior as one such indicator of behavioral stress responses in individuals presenting with MVA-related PTSD. Consistent with our hypotheses, significant reductions in exaggerated safety, performance deficits, and hostile/aggressive driving behaviors were noted among the WET condition relative to the WL condition at 6- and 18-weeks post-randomization. This finding is critical in supporting the validity of the DBS as a measure sensitive to treatment-related change.

It is also important to demonstrate that the behavioral stress response indexed by the DBS subscales is distinct from PTSD symptom severity. As we hypothesized, observed associations between the DBS subscales and the PTSD symptom clusters were small to medium, suggesting that the DBS subscales are related yet distinct constructs from PTSD symptoms. Moreover, findings from the mediation analyses support the contention that the reduction on the DBS subscales observed for the treatment participants were not better accounted for by reductions in PTSD symptom severity. It is also worth noting that no DBS subscale evidence a reliable association with PTSD symptom clusters. Taken together, the correlational and the mediational analyses support the distinction of the DBS subscales from PTSD symptom measurement.

Given negative traffic outcomes associated with driving anxiety (Dula et al., 2010; Matthews et al., 1998; Shahar, 2009; Taylor & Koch, 1995) and elevated levels of anxious driving behavior among individuals with MVA-related PTSD (Clapp et al., in press), the present data support the clinical relevance of anxious driving within this population, as well as the treatment sensitivity of the measure.

Evidence for radiating effects of the intervention in this study is particularly noteworthy given that the WET protocol does not specifically address driving-related impairment through *in vivo* exposures or stress management skills (e.g. relaxation or mindfulness). Although some have noted the importance of skills-based training for individuals presenting with driving anxiety (e.g., Taylor, Deane, & Podd, 2002; 2008), these findings support the notion that focusing on a primary disorder is beneficial for reducing potentially impairing behavioral responses beyond the formal symptoms of the target disorder.

Reduction in anxious driving behavior concurrent with the remission of formal PTSD symptoms is consistent with the hypothesized relation between state-level anxiety and problematic driving behavior (Clapp, Olson, Beck, et al., 2011). These data suggest that psychoeducation and reductions in PTSD symptomatology positively impact specific domains of anxious driving behavior. Future study should evaluate the extent to which formal skills training and/or *in vivo* exposures enhance effects noted in this study.

Although anxious driving behavior is known to be diagnostically complex and independent of motor vehicle trauma (Ehlers, Hofmann, Herda, & Roth, 1994; Taylor & Deane, 2000; Taylor et al., 2002), these behaviors appear well-suited to function as treatment sensitive indices of behavioral stress responses for individuals with MVA-related PTSD. In particular, the excessive caution and hostility/aggression subscales measure broader categories of behavioral stress responses, avoidance and fight, which are intended to be captured by PTSD symptoms C1-C2 (i.e. avoidance of thoughts and feelings, avoidance of things) and D2 (i.e. irritable or aggressive) respectively. However, as evidenced by the mediational analyses in this study, these subscales seem to measure a unique aspect of functioning that we believe provide greater clinical utility for the index trauma of MVA. Furthermore, the DBS may be well-suited to capture behavioral stress responses related to other types of index traumas. For example, aggressive driving, as measured with the hostility/aggression subscale, has been noted as relevant among individuals with combat-related PTSD, particularly veterans who have experienced combat in the recent conflicts in Iraq and Afghanistan where troops were often attacked while driving (Kuhn, Drescher, Ruzek, & Rose, 2010). Further examination of anxiety-related driving behaviors among returning veterans, in particular the hostility/aggression subscale, and consideration of such behaviors as a target for treatment is warranted.

A number of factors should be considered in the interpretation of these data. First, the relatively small sample limits strong interpretation of several effects. Research incorporating larger trauma samples with a greater range of symptom severity (e.g., mild to severe symptomatology) will provide more precise estimates of associations between PTSD symptoms and domains of anxious driving behavior. Second, evaluation of anxious driving behavior was restricted to self-reported scores as assessed with the DBS subscales. Although the DBS measure demonstrates strong psychometric properties (e.g. Clapp, Olsen, Beck, et al., 2011) and there is concordance between self-report and behavioral measures of anxious driving (Taylor et al., 2007), utilization of behavioral assessment would strengthen the conclusions. Third, evaluation of anxious driving behavior in the current study was limited to active motorists. A handful of participants in the larger clinical trial were excluded from the current study given the absolute absence of driving at the initial assessment, which they reported was the direct result of their MVA. Although some of these individuals resumed driving at later assessments, their data could not be utilized in the current study. An additional area of behavioral stress responses to an MVA that would more comprehensively capture the anxiety of these individuals and may be worthy of future consideration is “back-seat driving” behaviors of individuals who travel as passengers in motor vehicles or use other modes of transportation (e.g., public transit).

In summary, the DBS shows promise as a measure of trauma-specific behavioral stress responses for individuals with MVA-related PTSD. Results of the present study indicate reductions in exaggerated safety, performance deficits, and aggressive driving behaviors that may place survivors at increased accident risk. Consistent with efforts to identify alternative indicators of behavioral stress responses and impairment, data suggest anxious driving behaviors are related, but conceptually distinct, from formal PTSD symptoms, and responsive to PTSD treatment. Continued research is encouraged to identify and develop additional measures of trauma-specific behavioral stress responses.

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Highlights

- Anxiety during operation of a motor vehicle may impact driving performance
- Excessive safety, performance deficits, and aggressive driving domains were assessed
- Persons with posttraumatic stress disorder evidence reliable reductions in each domain
- The reduction in anxious driving was not accounted for by reductions in posttraumatic stress symptoms
- Anxious driving behavior shows promise as behavioral stress responses

Table 1

Hierarchical linear modeling estimated effects for condition, time, and condition $\times$ time interaction for each subscale in the Driving Behavior Survey

DBS Subscales	<i>B</i>	<i>SE</i>	<i>t</i>	<i>df</i>	<i>P</i>	<i>r</i>
ABPD						
Intercept	2.22	0.18	12.49	39	<.001	
Time	0.41	0.22	1.90	38	.64	
Time $\times$ Condition	-0.63	0.22	-2.91	38	<.001	0.43
ESCB						
Intercept	4.49	0.22	20.62	39	<.001	
Time	0.27	0.19	1.46	38	.15	
Time $\times$ Condition	-0.60	0.22	-2.75	38	.01	0.41
HAB						
Intercept	2.96	0.17	16.92	39	<.001	
Time	0.27	0.19	1.42	38	.17	
Time $\times$ Condition	-0.62	0.22	-2.88	38	.007	0.42

Note: DBS = Driving Behavior Survey; ABPD = anxiety-based performance deficits; ESCB = excessive safety/caution behavior; HAB = hostile aggressive behavior

Table 2

Summary statistics for the Driving Behavior Subscale scores as a function of condition and time

Time-point	Measure	WET		Waitlist	
		Mean	SD	Mean	SD
Baseline	ABPD	2.27	1.50	2.22	1.25
	ESCB	4.68	1.31	4.16	1.72
	HAB	3.17	1.48	2.80	1.15
6 Weeks Post Baseline	ABPD	2.05	1.00	2.99	1.61
	ESCB	4.31	1.81	5.03	1.07
	HAB	2.49	1.33	3.58	1.40
18 Weeks Post Baseline	ABPD	1.79	0.78	3.10	2.00
	ESCB	3.95	1.63	4.91	1.48
	HAB	2.44	1.12	3.39	1.77

Note: WET = Written Exposure Therapy, $n = 21$; Waitlist $n = 19$, SD = standard deviation; ESCB = excessive safety/caution behavior; ABPD = anxiety-based performance deficits; HAB = hostile aggressive behavior

Table 3

Results from post-hoc t-tests of Driving Behavior Subscales change from pretreatment to 18-week follow up in each condition

Condition	DBS Subscales	<i>t</i>	<i>df</i>	<i>p</i>
WET	ABPD	2.31	18	0.03
	ESCB	2.81	18	0.01
	HAB	2.33	18	0.03
WL	ABPD	-1.84	20	0.08
	ESCB	-1.52	20	0.14
	HAB	-1.53	20	0.14

Note. WET = Written Exposure Therapy; WL = waitlist; DBS = Driving Behavior Survey; ABPD = anxiety-based performance deficits; ESCB = excessive safety/caution behavior; HAB = hostile aggressive behavior

Table 4

Descriptive statistics for the PTSD severity scores as a function of condition and time

Time-point	Measure	WET		Waitlist	
		Mean	SD	Mean	SD
Baseline	CAPS Total	60.79	15.50	69.10	18.59
	Cluster B	15.05	5.50	17.67	4.91
	Cluster C	25.05	7.71	28.48	10.19
	Cluster D	20.68	6.48	22.95	6.98
6 Weeks Post Baseline	CAPS Total	18.95	11.74	71.10	18.07
	Cluster B	7.37	4.36	18.76	5.42
	Cluster C	6.58	5.61	30.57	10.40
	Cluster D	5.00	3.94	21.76	7.73
18 Weeks Post Baseline	CAPS Total	10.26	8.99	55.19	25.32
	Cluster B	3.16	3.91	14.71	4.85
	Cluster C	3.42	3.59	22.14	14.54
	Cluster D	3.68	3.64	18.33	9.13

Note: PTSD = posttraumatic stress disorder; WET = Written Exposure Therapy, $n = 21$; Waitlist $n = 19$, SD = standard deviation; CAPS = Clinician Administered PTSD Scale

Table 5

Correlations between DBS subscales and PTSD severity scores at 6- and 18-week post baseline assessment

	6 Weeks Post Baseline			18 Weeks Post Baseline		
	ABPD	ESCB	HAB	ABPD	ESCB	HAB
CAPS Total	0.17 (.31)	0.31 [†] (.06)	0.41 ^{**} (<.01)	0.34 [*] (.03)	0.23 (.15)	0.25 (.11)
Cluster B	0.25 (.12)	0.33 [*] (.04)	0.50 ^{**} (<.01)	0.32 [*] (<.05)	0.24 (.14)	0.30 [†] (.06)
Cluster C	0.14 (.40)	0.26 (.11)	0.32 [*] (<.05)	0.29 [†] (.07)	0.16 (.33)	0.18 (.28)
Cluster D	0.11 (.50)	0.29 [†] (.07)	0.32 [*] (.01)	0.36 [*] (.02)	0.28 [†] (.08)	0.28 [†] (.08)

Note. All results listed as: $r(p)$

DBS = Driving Behavior Survey; PTSD = posttraumatic stress disorder; ABPD = anxiety-based performance deficits; ESCB = excessive safety/caution behavior; HAB = hostile aggressive behavior; CAPS = Clinician Administered PTSD Scale

[†]
 $p < .10$

^{*}
 $p < .05$

^{**}
 $p < .01$