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Brain Function Associated with Cooccurring Trauma and Depression Symptoms in College Students

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ABSTRACT
The goal of this pilot study was to determine if functional brain differences are present among individuals with high and low cooccurring trauma and depression symptoms. This pilot study examined how the P300 latency component of event-related potentials (ERPs), measured using electroencephalography (EEG) while participants performed a go/no-go task, might be associated with cooccurring self-report of trauma and depression symptoms in a sample of college students (N = 38). Alpha-corrected independent sample t tests revealed statistically significant differences in ERP P300 peak latencies between those in the high cooccurring trauma and depression symptoms group (n = 12) and those in the low group (n = 26) for all 3 midline electrode sites (Fz, Cz, and Pz). This pilot study provides preliminary evidence of differential brain functioning in individuals experiencing cooccurring trauma and depression symptoms. Accordingly, these findings support future research examining brain functioning in cooccurring symptoms.

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College students; cooccurring symptoms; depression symptoms; electroencephalography; event-related potentials; P300 latency; trauma symptoms

Depression and trauma symptoms are major global public health concerns, combining to cause an estimated economic burden of $125.4 billion per year (Centers for Disease Control and Prevention, 2013, 2015; Greenberg et al., 2003). When these symptoms cooccur, they result in greater functional impairment than when they are experienced individually (De Graaf, Bijl, Smit, Vollebergh, & Spijker, 2002; Kilpatrick et al., 2003). Cooccurrence of trauma and depression symptoms has been reported to exceed 50%, indicating that many individuals are affected by both of these symptom groups (Blanchard, Buckley, Hickling, & Taylor, 1998; Rytwinski, Scur, Feeney, & Youngstrom, 2013). This study focuses on cooccurrence because the research in this area is limited, despite the deleterious impact and pervasiveness of cooccurring trauma and depression symptoms.

Clinical research examining trauma and depression separately has indicated differences in brain function among individuals with these symptoms when compared to healthy controls (De Bellis, 2001; Javanbakht, Liberzon, Amirsadri, Gjini, & Boutros, 2011; Karl, Malta, & Maercker, 2006; Lobo et al., 2015; Paelecke-Habermann, Pohl, & Leplow, 2005;
Ruchsow et al., 2008; Tavares, Drevets, & Sahakian, 2003). Epidemiological research examining traumatized individuals has indicated that having one set of symptoms increases susceptibility for developing the other (Boney-McCoy & Finkelhor, 1995; Breslau, Davis, Peterson, & Schultz, 2000; Kilpatrick et al., 2003; O’Donnell, Creamer, & Pattison, 2004). Multiple studies have found that trauma symptoms are more likely to cooccur with depression symptoms than to occur alone (Blanchard et al., 1998; Boney-McCoy & Finkelhor, 1995; Breslau et al., 2000; Kilpatrick et al., 2003; O’Donnell et al., 2004; Rytwinski et al., 2013). Trauma and depression symptoms are also often associated with common consequences (e.g., anhedonia, insomnia, isolation), yet the mechanisms underlying their commonalities are largely unknown (Blanchard et al., 1998; Breslau et al., 2000; O’Donnell et al., 2004; Yarvis & Schiess, 2008).

Given that having one set of symptoms increases the susceptibility of developing the other, it is possible that cooccurring trauma and depression symptoms have an underlying shared vulnerability (Breslau et al., 2000; Kendler, Karkowski, & Prescott, 1999; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; O’Donnell et al., 2004). However, brain function has yet to be thoroughly examined as a shared vulnerability by examining symptoms in cooccurrence. Understanding the specific brain function related to these symptoms could be particularly important for college students, who commonly experience trauma and depression symptoms, but might not yet meet criteria for a psychiatric disorder (Boney-McCoy & Finkelhor, 1995; Copeland, Keeler, Angold, & Costello, 2007; Kilpatrick et al., 2003; Lewinshon, Rohde, & Seely, 1998; Read, Ouimette, White, Colder, & Farrow, 2011).

**Event-related potentials**

An event-related potential (ERP) averages together the electrical activity pertaining to a specific mental event recorded from electroencephalography (EEG; Bokura, Yamaguchi, & Kobayashi, 2001; Picton et al., 2000). The ERP waveforms have multiple components that reflect different stages and levels of processing (Bradley et al., 1997). The largest and most commonly studied component, the P300, is the task-relevant stimulus that reflects a cognitive component (Bradley et al., 1997). This large and positive wave peaks approximately 300 ms after the onset of a stimulus related to the task (Duncan et al., 2009). The measure of P300 latency is thought to reflect the timing involved in an individual’s decision to respond or withhold his or her response to a stimulus during a cognitive processing task (Shucard, McCabe, & Szymanski, 2008). This is an effective component to study cognitive processes because it is sensitive to conditions where cognition is impaired, and deviations in this
component have been shown to reflect deficits in cognitive processing (Duncan et al., 2009).

**Response inhibition**

Inhibition is an extremely important aspect of executive functioning and can be evaluated in a simple experimental task known as the go/no-go task in which participants are asked to differentially respond to stimulus presentations (Kiefer, Marzinzik, Weisbrod, Scherg, & Spitzer, 1998). During standardized inhibitory go/no-go tasks, participants are told to respond to a target stimulus (go condition), and to refrain from responding to a nontarget stimulus (no-go condition). Evaluating ERPs produced during a go/no-go cognitive task is an effective way to evaluate inhibitory processes due to their high temporal resolution (Bokura et al., 2001).

**EEG and ERPS in trauma and depression symptoms**

Research in the area of brain function in depression and trauma symptoms is emerging. Much of the research examining these symptoms has evaluated depression or trauma symptoms separately, even though these symptoms are often experienced jointly. This pilot study aims to fill these gaps by examining cooccurring trauma and depression symptoms.

Shorter P300 latencies were reported in studies that detected differences between individuals with depression symptoms and healthy controls (Bruder et al., 2002; Bruder et al., 2009). Multiple studies, including two meta-analytic reviews, one comparing traumatized and nontraumatized individuals and a second examining posttraumatic stress symptoms, found longer P300 latencies associated with trauma symptoms (Javanbakht et al., 2011; Karl et al., 2006; Lobo et al., 2015; Shucard et al., 2008). Because shorter P300 latencies have been seen with depression symptoms and longer P300 latencies have been seen with trauma symptoms, we sought to determine if these latency differences would be observable in individuals with cooccurring trauma and depression symptoms.

**This study**

We hypothesize that individuals with cooccurring trauma and depression symptoms will have different P300 latencies in comparison to individuals not experiencing these cooccurring symptoms. Specifically, we hypothesize that during the go/no-go task there will be shorter P300 latencies associated with depression symptoms (Bruder et al., 2002; Bruder et al., 2009) and longer P300 latencies associated with trauma symptoms in the cooccurring high group as compared to the low group (Javanbakht et al., 2011; Karl
et al., 2006; Lobo et al., 2015; Shucard et al., 2008). Our work examining these symptoms in cooccurrence represents a logical next step in this line of research.

**Method**

**Participants**

The participants in this pilot study included 38 college students (ages 18–20), who were recruited through the university subject pool over the course of two semesters, between January 2015 and December 2015. These participants were recruited for an Institutional Review Board (IRB)-approved study assessing trauma and depression symptoms using electroencephalography.

**Procedure**

Each participant signed an IRB-approved informed consent. This pilot study consisted of three surveys: a demographics survey, the Achenbach Adult Self-Report (ASR; Achenbach, 2009), and the Trauma Symptom Checklist (TSC–40; Briere & Runtz, 1989). Directly after these assessments were completed in random order, a Food and Drug Administration-approved 64-channel Biosemi EEG system with ActiveTwo Pin-type electrodes was used to assess brain activity during the go/no-go cognitive task. None of the participants withdrew from the study and there were no adverse events. The principal investigator, the second author, who is a licensed psychologist, was present to monitor clinical safety. To ensure confidentiality, identifiers were removed, including participant names, date of birth, and date of assessment. Further, data were secured through the use of an encrypted, firewalled, password-protected computerized data collection and management system, the Collaborative Informatics and Neuroimaging Suite, which is compliant with the Health Insurance Portability and Accountability Act (A. Scott et al., 2011).

**Trauma symptom checklist**

The TSC–40 is a 40-item self-report measure that assesses trauma symptoms, which occur on a spectrum (Briere & Runtz, 1989). This measure is used routinely in healthy individuals who have subclinical trauma symptoms. Individuals report symptoms associated with trauma and do not indicate a specific traumatic event. In a validation study of 667 maltreated young adults conducted by the author of the TSC–40, less than 4% of the sample fell within the clinical range (Briere, 1996b). Hence, this is an appropriate measure for assessing trauma symptoms in healthy populations and is sensitive to differences in scores that occur in a nonclinical range.
This measure uses a 4-point Likert response scale ranging from 0 (never) through 3 (often) to address symptom occurrence and frequency during the past month. The TSC–40 has six subscales that assess different aspects of symptom clusters seen in individuals who have experienced trauma: sexual problems, sexual abuse trauma index, sleep disturbance, dissociation, anxiety, and depression. This measure has been shown to have high internal consistency with Cronbach’s alpha ranging from .89 to .91 (Briere, 1996a). We used the TSC–40 total score, which is a summary score of all of the items on the TSC–40, as our measure of trauma symptoms.

**Achenbach adult self-report**

Our measure of depression symptoms was the Anxious/Depressed scale of the ASR (Achenbach, 2009). The ASR is a standardized and well-validated 126-item measure. The Anxious/Depressed scale of the ASR has been used in multiple studies as a measure of depression symptoms (Reef, Diamantopoulos, Van Meurs, Verhulst, & Van Der Ende, 2009; T. J. L. Scott, Heil, Higgins, Badger, & Bernstein, 2009). This scale is based on *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM–IV]; American Psychiatric Association, 1994) diagnostic criteria for major depressive disorder (Reef et al., 2009; T. J. L. Scott et al., 2009). The ASR Anxious/Depressed scale has been shown to have stable psychometric properties and has a similar number and scope of items as stand-alone depression measures, such as the Beck Depression Inventory (BDI; Beck, Ward, & Mendelson, 1961; T. J. L. Scott et al., 2009). The ASR Anxious/Depressed scale and BDI have also been shown to have high convergent validity (Achenbach & Rescorla, 2003). This questionnaire was scored and evaluated based on the recommendations in the ASR manual for this scale and the percentile cutoff scores designated for the clinical range (Achenbach & Rescorla, 2003).

**EEG and Go/No-Go task**

Participants completed a 12-min standardized auditory go/no-go task while the EEG was being recorded (Falkenstein, Koshlykova, Kiroj, Hoormann, & Hohnsbein, 1995). In the go/no-go task, the participant heard two tones that were either the same frequency or different frequencies for a total of 50 stimuli presentations. For this task, participants must inhibit their response when they hear two tones of the same frequency (low–low), and respond by pressing the spacebar when they hear tones of different frequencies (low–high). In our pilot study, we are focusing on the go stimuli of the go/no-go task, as this condition has been found in the literature to have more consistent differences between healthy individuals and individuals with depression and trauma symptoms (Bruder et al., 2002; Bruder et al., 2009; Karl et al., 2006; Lobo et al., 2015; Tenke, Kayser, Stewart,
& Bruder, 2010). Additionally, research has indicated that the go condition reveals more specific differences in individual functioning as seen in a meta-analysis by Lobo and colleagues (2015), where longer go latencies were associated with posttraumatic symptomology, whereas no-go latencies displayed a nonspecific effect that was not related to posttraumatic symptomology. Due to the differences that the go component has revealed in individuals displaying these symptoms individually (i.e., shorter latencies in depression, and longer latencies associated with posttraumatic symptoms and hyperarousal), this component was chosen to examine cooccurrence of these symptoms (Bruder et al., 2002; Bruder et al., 2009; Karl et al., 2006; Lobo et al., 2015; Tenke et al., 2010).

Data were processed using the EEGLab and ERPLab plug-ins designed for MATLAB following the methods outlined by Lopez-Calderon and Luck on the ERPLab Toolbox Web site (Lopez-Calderon & Luck, 2014). These EEG data were segmented into epochs surrounding the stimuli presentations, starting 200 ms before the stimulus, and lasting until 800 ms after the stimulus onset for each stimulus presentation. A series of uniform steps were completed on the continuous EEG data sets to remove bad channels, electrical noise, and non-brain-wave artifacts (e.g., muscle activity, eye movement, and blinks) that contaminate the brain signal. The steps that were used to analyze the data were based on the ERPLab Toolbox tutorial, which states the best practices for analyzing EEG and ERP data (Lopez-Calderon & Luck, 2014). Artifact-free ERPs were created using ERPLab to examine the P300 component of the ERP waveform for three electrode sites: Fz (frontal), Cz (central), and Pz (parietal). The P300 component of the ERP and the midline electrode sites were chosen based on their widespread use in the literature.

**Low and high symptom groups**

We designated low and high trauma and depression symptom groups based on the recommended clinical cutoffs found in the literature for our measures (Birrer, Michael, & Munsch, 2007; Reynolds & Brewin, 1998). The ASR recommended a cutoff at the 69th percentile and the TSC–40 recommended a cutoff score of 30 or greater for the clinical range (Achenbach & Rescorla, 2003; Briere, 1996a; Silvern et al., 1995). Participants were only placed in the high cooccurring group if they met or exceeded both the trauma and depression symptom cutoff scores. If a participant only met one cutoff (i.e., trauma or depression), that participant went into the low group along with participants who were below the clinical cutoff on both measures. We then examined the peak latency of the P300 ERP component within the midline electrode sites (Fz, Cz, and Pz), occurring between 280 and 580 ms after the stimuli presentations within these two groups (Dodin & Nandrino, 2003).
**Results**

Analyses included the entire sample \( (N = 38) \). The age \( (M = 18.39, SD = 0.59) \), gender (males = 7.9%, females = 92.1%), and race (White = 81.6%, non-White = 18.4%) of our participants were the same as the overall population of undergraduates taking introductory psychology at a college in the southeastern United States. The age, gender, language, and race differences between participants in our high and low cooccurring groups were not statistically significant so they were not included as covariates in the analyses. Visual inspection of the scatterplot for the relation among trauma symptoms and depression symptoms indicated that this relationship was linear. A zero-order correlation was used to verify and statistically examine this linear relationship. The correlation between trauma and depression symptoms was high and statistically significant, \( r(38) = .702, p < .001 \). College students with more trauma symptoms had higher depression symptom scores. The correlation between depression and trauma symptoms has a corresponding \( R^2 \) value of 0.49, which does not exceed the multicolinearity cutoff of \( R^2 = .80 \) (Marsh, Dowson, Pietsch, & Walker, 2004).

The average total trauma symptoms score \( (M = 26.42, SD = 16.02) \) and depression symptoms score for the entire sample \( (M = 12.13, SD = 6.81) \) were both below the clinical range and what is expected for a college sample (Briere, 1996a). We had 14 participants who qualified for the depression symptom high group by exceeding the cutoff percentile of 69 for the clinical range established by the ASR \( (M = 19.57, SD = 2.65, range = 16–24) \), and 24 participants who fell within the boundaries of the depression symptom low group \( (M = 7.97, SD = 4.15, range = 2–15) \). We had 16 participants who qualified for the trauma symptom high group by exceeding the cutoff score of 30 established by the TSC \( (M = 43.38, SD = 7.95, range = 31–55) \), and the remaining 22 participants were placed in the trauma symptom low group \( (M = 14.09, SD = 5.39, range = 6–23) \). After creating these groups, there were 12 participants who qualified for the cooccurring high symptom group by scoring in the clinical range on both measures, and the remaining 26 fell in the low group. As would be expected, the cooccurring high group displayed significantly higher trauma symptoms \( (M = 43.38, SD = 7.95) \) than the low group \( (M = 14.09, SD = 5.39) \), as well as higher depression symptoms \( (M = 19.57, SD = 2.65) \) than the low group \( (M = 7.97, SD = 4.15) \). A chi-square test of independence indicated no significant differences for gender between our high and low cooccurring symptoms groups, \( \chi^2(N = 38) = .005, p = .946 \). Because gender differences were not found, they were not included as covariates in the analyses.

An independent samples \( t \) test was conducted to compare the P300 latency component of the ERP for three midline electrode sites (Fz, Cz, and Pz) in the cooccurring high and low groups. Table 1 presents the
peak P300 latencies to the go stimuli for the cooccurring high and low groups. There was a significant difference in P300 latency for the Fz site between the cooccurring high ($M = 498.78$, $SD = 81.64$) and low groups ($M = 402.63$, $SD = 86.53$), $t(1, 36) = -3.239$, $p = .003$, $d = 1.142$. There was a significant difference in P300 latency for the Cz site between the cooccurring high ($M = 343.87$, $SD = 60.93$) and low groups ($M = 407.51$, $SD = 93.97$), $t(1, 31.632) = 2.498$, $p = .018$, $d = .742$. There was a significant difference in P300 latency for the Pz site between the cooccurring high ($M = 328.90$, $SD = 26.45$) and low groups ($M = 370.51$, $SD = 74.73$), $t(1, 34.615) = 2.518$, $p = .017$, $d = .804$ (see Figure 1).

To control for family-wise error rate after multiple comparisons, we employed a Bonferroni-corrected alpha of .017 (Bland & Altman, 1995). After correcting our alpha level, one of our significant findings (i.e., Cz go latency $p = .018$) was no longer significant. We then conducted a power analysis with G*Power (Erdfelder, Faul, & Buchner, 1996) to determine the critical cutoff scores needed to detect an effect given our sample size and Bonferroni-corrected alpha. The critical cutoffs for our pilot study were .89 or larger for Fz, .61 or larger for Cz, and .54 or larger for Pz. Our effect sizes

### Table 1. Peak P300 Latencies and Standard Deviations.

<table>
<thead>
<tr>
<th></th>
<th>High group</th>
<th>Low group</th>
<th>$p$ value</th>
<th>Effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Go Fz</td>
<td>498.77</td>
<td>402.63</td>
<td>$.003**</td>
<td>$d = 1.142$</td>
</tr>
<tr>
<td>Cz</td>
<td>343.87</td>
<td>407.51</td>
<td>$.018*</td>
<td>$d = .742$</td>
</tr>
<tr>
<td>Pz</td>
<td>328.90</td>
<td>370.51</td>
<td>$.017**</td>
<td>$d = .804$</td>
</tr>
</tbody>
</table>

Note: Peak P300 latencies and standard deviations over midline electrode sites (Fz, Cz, and Pz) for the Go condition in the high and low groups for cooccurring trauma and depression symptoms. *$p \leq .05$. **$p \leq .017$ Bonferroni-corrected alpha.

Figure 1. Peak P300 latencies with standard error bars, over the midline electrode sites (frontal [Fz], central [Cz], and parietal [Pz]) for the go stimuli in the high and low groups for cooccurring trauma and depression symptoms. *$p \leq .05$. **$p \leq .017$ Bonferroni corrected alpha.
for Fz, Cz, and Pz exceeded the critical cutoffs indicating that we had sufficient power to detect an effect in our pilot study (Cohen, 1992; Table 1).

Conclusions

Our pilot study sought to evaluate how cooccurrence of trauma and depression symptoms might be related to P300 latency. We found that individuals with high cooccurring trauma and depression symptoms significantly differed from those with low symptoms at all three midline electrode sites. These results support previous research on P300 latency in trauma and depression (Bradley et al., 1997; Bruder et al., 2002; Bruder et al., 2009; Duncan et al., 2009; Javanbakht et al., 2011; Karl et al., 2006; Lobo et al., 2015; Shucard et al., 2008; Tenke et al., 2010).

We found shorter P300 latencies at the central (Cz) and parietal (Pz) electrode sites, as well as longer P300 latencies at the frontal electrode site (Fz) in our high cooccurring trauma and depression symptom group when compared to the low group. These results are supported by past studies that have also shown P300 reductions at midline-central sites in individuals with depression symptoms (Bruder et al., 2002; Bruder et al., 2009; Ruchsow et al., 2008; Tenke et al., 2010) and longer P300 latencies in individuals with trauma symptoms (Javanbakht et al., 2011; Karl et al., 2006; Lobo et al., 2015; Shucard et al., 2008). Our findings point to observable differential resource allocation in individuals with cooccurring trauma and depression symptoms, as opposed to individuals in the low group.

Our high group was comprised of individuals who met or exceeded the recommended cutoffs for both trauma and depression symptoms. Rather than having a “super control” low group, we chose a low group that was made up of individuals who were either low on both, low only for trauma, or low only for depression. Our goal in having a heterogeneous low group for comparison in our pilot study was to strengthen the ecological validity of our findings of measurable differences in P300 latency in the high group. Future research should aim to identify other dimensional aspects of trauma and depression symptoms as a way of targeting the specific processes that drive changes in cortical activation.

Implications

These pilot findings suggest that those with cooccurring trauma and depression symptoms have P300 latencies that might differ from individuals experiencing only trauma or depression symptoms. However, given that this study examined symptoms and not disorders, one cannot conclude that individuals with comorbid disorders differ from those with just one or the other. The literature has shown that those with depression symptoms have characteristic differences in
cognitive functioning indicated by shorter P300 latencies, whereas those with trauma symptoms have characteristically longer P300 latencies. These findings suggest that the processes contributing to shorter P300 latencies in depression, and longer P300 latencies in trauma symptoms might be combined in individuals who are experiencing cooccurring symptoms. Further, the findings suggest that trauma symptoms might be more closely associated with the frontal region, whereas depression symptoms might be more associated with the central and parietal regions. This falls in line with past research that has indicated that individuals who have experienced traumatic events have greater difficulties within their frontal brain systems exhibited by deficits in functioning in the prefrontal and medial frontal brain systems (Koenen et al., 2001; Shin et al., 2004; Williams et al., 2006). Research has shown that the responsivity of the medial prefrontal cortex is “inversely associated” with trauma symptom severity (Shin, Rauch, & Pitman, 2006). Prolonged P300 latencies are seen in cases of decreased cognitive ability (Picton, 1992). Accordingly, the longer P300 latencies displayed at the frontal electrode site in our cooccurring symptoms group could be a consequence of these frontal deficits displayed by individuals experiencing trauma symptoms. In contrast, shorter P300 latencies to novel stimuli have been reported for midcentral sites in individuals with depression compared to healthy control participants (Bruder et al., 2002; Bruder et al., 2009; Tenke et al., 2010). The differences between our cooccurring symptoms high and low groups point to potential cognitive changes in the midcentral regions in individuals experiencing depression symptoms (Tenke et al., 2010).

The results of this study correspond to findings of frontal deficits associated with trauma, as well as the multiple studies indicating shorter P300 latencies for the midcentral sites. In sum, these findings suggest that the processes contributing to longer P300 latencies for trauma symptoms and shorter P300 latencies for depression symptoms are combined in individuals experiencing cooccurring symptoms. This is important to consider in future research as many psychological disorders, such as depression and anxiety, are commonly comorbid. Our findings suggest a distinct difference between brain function in those with and without cooccurring symptoms. The findings of this study support future research investigating these disorders in isolation, as well as in comorbidity, in an attempt to isolate the deficits unique to each disorder.

Limitations

Some limitations to this pilot study include that it is a cross-sectional study of depression and trauma symptoms. Therefore, we cannot examine causality. It is also possible that the relationship between our study variables would differ depending on the type of trauma experienced. In addition, we assessed trauma and depression symptoms, not clinical posttraumatic stress disorder.
(PTSD) and depression per se; studies of disorders could have differing results from this study focused on symptoms. Although our pilot sample was sufficiently powered, it was a relatively small sample that was limited in age, gender, and racial and ethnic diversity. Given that the symptoms assessed are commonly seen in college students, the results might generalize to other college students between the ages of 18 and 20. However, this study had a narrow age range and the college student sample recruited through the university subject pool represents a convenience sample. The student population for this study is predominantly female (62%) and lacking in ethnic diversity (18%), which resulted in a predominantly White female sample. These factors limit the generalizability these findings. Future research should examine a more expansive age range, as well as use random sampling techniques to obtain a more representative sample in age, gender, and racial and ethnic diversity.

An additional limitation of this study regards the severity of trauma and depression symptoms displayed by our sample. Among our sample of 38, only 12 individuals exceeded the clinical cutoff criteria for both the trauma symptom and depression symptom measures. However, among the 12 individuals that qualified for the cooccurring group, a good variability of scores was displayed falling well into the clinical range of scores on both measures. A further limitation of this study is the fact that these measures only assess trauma and depression symptoms, not the corresponding DSM diagnoses of PTSD and major depressive disorder.

To expand on these findings and address limitations of this study, future studies would benefit from implementing diagnostic measures to examine not just symptomatology, but comorbid PTSD and major depressive disorder diagnoses. These studies should aim to include a larger sample with individuals who have clinical comorbid PTSD and depression, as well as individuals experiencing only PTSD or depression, and finally a healthy control group. Comparing brain functioning among individuals with cooccurring symptoms, single diagnoses, and healthy controls might allow for a greater understanding of the factors contributing to differential brain functioning within these disorders. A longitudinal study of these disorders should provide the greatest insight into their development. Due to the prevalence of PTSD and depression in the military, a suggested future research endeavor would be for researchers to work with the military to examine a large sample of individuals prior to deployment, with follow-up measures on return from deployment, and yearly after return for a period of 5 years. This study would need to control for traumatic brain injury by excluding any individuals who experience brain damage during deployment that could affect P300 findings. Future research in this area holds promise to provide insight into differential brain function underlying depression and trauma symptoms,
both when these symptoms are experienced in isolation and in cooccurrence.

**Future directions**

Trauma and depression symptoms are frequently cooccurring mental health problems. Our pilot findings of significantly shorter P300 latencies (Cz and Pz) associated with depression symptoms and significantly longer latencies (Fz) associated with trauma symptoms in our high cooccurring symptoms group pull together findings from past studies examining either depression or trauma symptoms individually, but not in cooccurrence. These results provide support for future studies looking at shared neural mechanisms over time and in a variety of cognitive tasks, including both visual and auditory cognitive tasks. These P300 differences were present when examining trauma and depression symptoms together, highlighting the importance of examining cooccurring symptoms. Additional research should examine interaction effects (e.g., type of trauma, severity of depression) in a larger sample over time as a way to identify related factors that might influence functional brain changes. Research on cooccurring symptoms is important for shedding light on possible risk and resilience factors that might not be present when studying either set of symptoms individually. This pilot study highlighted that brain functioning might be different in individuals with cooccurring symptoms when compared to individuals experiencing only depression or only trauma symptoms. If this pilot study is replicated and expanded, particularly related to the age range and diversity of the sample, the results could contribute to our understanding of cooccurring symptoms.

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**References**


during traumatic imagery in male and female Vietnam veterans with PTSD. *Archives of General Psychiatry, 61,* 168. doi:10.1001/archpsyc.61.2.168


