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Neurometric Correlates of Trauma Using the Brief Neurometric Battery (BNB)

A thesis submitted in partial fulfillment of the requirement
for the degree of Bachelor of Science in Neuroscience with Honors from
The College of William & Mary

by

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Williamsburg, VA
April 25, 2017
COMPLIANCE

Research approved by the Protection of Human Subjects Committee

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Abstract

Objective: The goal of this research was to determine the utility of a novel Brief Neurometric Battery (BNB) (Kieffaber et al., 2016) for measuring the neurometric correlates of a) experiences with sexual trauma and b) personality characteristics, and to assess the correlation of sexual trauma with event-related potentials derived through EEG.

Methods: The BNB testing battery uses a nested array of visual and auditory stimuli to elicit several event-related potentials (ERPs) and oscillatory activity in about 20 minutes. This subclinical study at the College of William & Mary used the BNB with college-aged women with past experiences of sexual violence. Participants’ experiences with sexual trauma were measured using the PTSD Checklist for DSM IV – Specific (PCL-S) and the Impact of Event Scale – Revised (IES-R), and personality traits were measured using a very brief ten-item personality measure.

Results: IES-R scores were significantly correlated with both P300 and vMMN ERPs. Grouping participants into “high” and “no stress” groups, a discriminant analysis using components vMMN, P300, and the C1 wave was significant, and achieved 78.9% accuracy in the reclassification of groups.

Conclusions: These results indicate that an ERP-based neurometric profile including the vMMN, C1 wave, and P300 ERPs may be useful for detecting neural changes associated with traumatic sexual experiences.

Significance: This study identified the use of a brief neurometric battery to elicit ERPs which generate a more complete and complex assessment of the manifestation of sexual trauma in the psychophysiology of the brain.
Since 2004, the United States National Institute of Mental Health has called for research to enhance the understanding of the link between violence and trauma and consequential mental health effects. Areas of focus include 1) clinical studies investigating the impact of stressful events on neurology and memory processing, 2) psychosocial and biological factors of trauma pathology and changes in these resulting from said trauma, 3) the development of assessment and screening measures, and 4) the translation of research into clinical applications (NIMH, 2004). This initiative refers to the expansion of academic and clinical understanding of posttraumatic stress disorder (PTSD). The NIMH defines PTSD as a disorder that develops as the result of a “shocking, scary, or dangerous” experience, including sexual violence or abuse. A more specific and extensive definition can be found in the Fifth Version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V, 2013) or abbreviated as such: “exposure to threatened death, serious injury or sexual violation” that “causes clinically significant distress or impairment of the individual’s social interactions, capacity to work or other important areas of functioning” (American Psychiatric Association, 2017). In order to allow for a diagnosis of PTSD, symptoms must be severe and impactful, and last more than one month; symptom categories include re-experiencing, avoidance, arousal/reactivity, and cognition/mood. This understanding of PTSD and its symptoms has lent itself to significant research opportunities, which have tended to focus on military experience-linked PTSD that may not be generally applicable to civilians (Brewin, Andrews, & Valentine, 2000; Xue et al., 2015) and to a lesser extent to childhood sexual abuse (CSA; Paolucci et al., 2001).
One area of research related to the NIMH directive is the Sexual Violence Research Initiative, founded by the World Health Organization (WHO) and Global Forum for Health Research (Australia) in 2000, and stemming from the Study on Women’s Health and Domestic Violence Against Women (WHO, 2017). This initiative has directed global interests around sexual violence and trauma with the South African Medical Research Council since 2006 (SVRI, 2015). Among the SVRI objectives that align directly with the NIMH initiatives are extending research and policy related to childhood sexual abuse (CSA) and the health effects of sexual violence, including trauma-related psychological disorders (SVRI, 2015).

The Sexual Violence Research Initiative and the World Report on Violence and Health (Jewkes & Garcia-Moreno, 2002) define sexual violence as “any sexual act, attempt to obtain a sexual act, unwanted sexual comments or advances, or acts to traffic, or otherwise directed, against a person’s sexuality using coercion, by any person regardless of their relationship to the victim, in any setting, including but not limited to home and work.” The DSM-V, too, includes a definition of “sexual violence” in the category of stressful events that could lead to PTSD, and includes “forced sexual penetration, alcohol/drug facilitated sexual penetration, abusive sexual contact,” and “noncontact sexual abuse” (Levin et al., 2014).

In a 2006 paper, Chivers-Wilson relays two important statistics. Firstly, according to the National Center for PTSD, one in three rape victims are affected by posttraumatic stress disorder (PTSD) in their lifetimes, with one source reporting 94% of survivors experiencing PTSD symptoms within two weeks of a rape (not accounting for the extended - one month - duration requirement of a diagnosis). Secondly, Creamer et al. (2001) determined that the lifetime prevalence for PTSD in women who have been sexually assaulted is 50% (Chivers-Wilson,
A study by Kilpatrick (2002) found that the lifetime prevalence of rape among adult women differs throughout the United States, ranging from 11.0% in the Mid-Atlantic region to 21.1% in the Mountain region. With one in five women in the United States sexually assaulted while in college (Kreb et al., 2007), the rates of sexual violence-linked PTSD that will affect this population now and into the future represents a significant enough public health concern to consider appropriate methods of recognizing, predicting, preventing, and treating PTSD in survivors.

Much research over the past 25 years has established the relationship between interpersonal violence and mental health outcomes, including long-term effects (Boney-McCoy & Finkelhor, 1995, 1996; Kessler et al., 1995; Kilpatrick et al., 2003; Resnick et al., 1993). However, research on sexual violence and PTSD related to adolescents is less conclusive (Kessler, Avenevoli, & Merikangas, 2001; Kilpatrick et al., 2003). In one study, Kilpatrick et al. (2003) found that girls in a nationwide survey met criteria for PTSD (DSM-III) at a rate of 6.3% (p. 695), and interpersonal violence “emerged as a strong predictor” of comorbidity to major depressive episodes and substance abuse/disorder (p. 699).

Research is occurring on university campuses nationwide relative to the experience of sexual violence by its young adult students. For example, the National Sexual Misconduct Campus Climate Survey conducted in October 2014 at the university of this research reported that 28% of the college’s women have experienced physical sexual misconduct (William & Mary Task Force for Preventing Sexual Assault & Harassment, 2015). Additional information in this survey revealed that 21% of rape survivors at the university had been raped more than once or multiple times, and a large portion of the rapes occurred while the survivor was 18-19 years old.
(41%). Rates varied by subgroup, including extracurricular involvement and sexual orientation. This highlights the importance of research relative to sexual violence and the experience of trauma in college-aged women for prevention and clinical services.

The consequences of sexual violence may manifest socially, behaviorally, biologically, developmentally, and psychologically (Chivers-Wilson, 2006). Social research has focused primarily on prevention, perceived social consequences of the event, and individual, clinical, and judicial systems of support, as these can greatly affect the probability of PTSD development (Dunmore et al., 1999; Chivers-Wilson, 2006).

Several authors have done comprehensive reviews of biopsychosocial research in this field, including the psychophysiological impacts of child abuse and neglect (Glaser, 2000), the developmental neurobiological impact of childhood stress and trauma (Teicher et al., 2002), the long-term effects of childhood abuse on the brain and neurobiology (Bremner, 2003), and connections between early abuse and changes in the limbic system, hippocampus, corpus callosum and hemispheric laterality (Teicher et al., 2006).

Biological research has been fairly extensive for adverse childhood experiences (ACEs; Anda et al., 2005) including sexual abuse. Research from Bremner et al. (2003; 2003), assessed the impact of CSA and PTSD on the dysregulation of the hypothalamic-pituitary-adrenal axis, neuroendocrine changes, and cortisol response. DeBellis et al. (1999) reported on biological stress symptoms and the strong relationship to childhood maltreatment, as well as negative HPA impacts in sexually abused girls (1994). Stein et al. (1997) discussed a link between reduced hippocampal volume and CSA in women.
Research conducted on the biological effects of sexual violence and psychological disorders have focused on adult survivors of childhood sexual abuse. Heim et al. (2000) found increased pituitary-adrenal and autonomic responses to stress, especially in those with current depression and anxiety symptoms. Psychological assessments of those impacted by sexual abuse and PTSD also produced significant results. Adults who had experienced abuse as a child had significantly lower scores on the Wechsler Memory Scale, Logical subcomponent, for short-term verbal memory but no difference for visual memory (Bremner et al., 1995). Additionally, attention and abstract reasoning/executive functioning assessments among children with maltreatment-related PTSD revealed poorer scores compared to healthy controls (Beers & DeBellis, 2002).

One way to measure both the biological and psychological components of violence-linked PTSD is through psychophysiological applications. Ito et al. (1998) conducted a study of hospitalized abused children and found higher levels of left hemisphere coherence and a reverse asymmetry stemming from a deficit in left cortical differentiation. Similarly, Teicher et al. (1994) used EEG coherence and MRI to reveal abnormal cortical development in survivors of childhood sexual abuse. Teicher et al. (1997) outlined four vulnerable neurobiological targets for those impacted by ACEs, CSA, and PTSD, including the hippocampus, amygdala, prefrontal cortex, and corpus callosum.

Bremner et al. (2003) used MRI and PET to assess hippocampal structure and activity related to memory for women with abuse experiences who either had or did not have PTSD, finding a 16% smaller volume in those with abuse and affected by PTSD compared to those with abuse but without PTSD. Women with abuse and PTSD projected a 19% smaller hippocampus
compared to non-abuse, non-PTSD controls. Likewise using MRI, Driessen et al. (2000) reported 16% smaller hippocampal volumes for women with borderline personality disorder and early trauma. This study also found a 7% smaller volume in the amygdala when compared to the healthy controls. Stein et al. (1997) reported unilateral left-sided hippocampal volume reductions of 5% in women impacted by sexual abuse as a child, which were not correlated with indices of explicit memory functioning. This research is supported by Navalta et al. (2006), which investigated memory recall, left-hemisphere processing, hemispheric integration, and inhibitory capacity problems in college-aged women with CSA experiences and found a strong graded association relative to duration of abuse and memory function.

Despite significant EEG, MRI, and PET research in the areas of sexual violence-related stress, little research has been done using the event-related brain potentials to assess low-level changes in processing. A paper by Metzger et al. (1999) found larger heart-rate responses (autonomic reactivity) and slower habituation of skin conductance in response to startle tones among women with current and lifetime sexual violence-linked PTSD, compared to a control group. This research hints to the possibility of using auditory ERPs, particularly the P50 - elicited by suppression in response to a second of paired click sounds - and MMN_{ISI} - the result of tones that occur at a different interstimulus interval - components in assessing PTSD. A meta-analysis in 2011 (Javanbakht, et al.), found large numbers of P50 studies reporting impaired sensory gating in those affected by PTSD.

The research of Bremner et al. (1995) discussed above regarding verbal and visual memory hindrances and changes in hippocampal volume allow the consideration of the P300, vMMN, C1 wave, and MMN_{FREQ} components; the P300 measures working memory (Johnson et
al., 2013), the vMMN measures visual memory, and the MMN$_{\text{FREQ}}$ measures auditory memory (Kieffaber et al., 2016). Based on the research provided, we would expect to see differences elicited for mismatch negativity using the above components, but a lesser impairment in visual memory than verbal memory (Samuelson, 2011). Further, Orr et al. (1998) found that women with a history of PTSD from sexual abuse showed larger physiologic reactions to abuse-related imagery but not nonabuse-related imagery. The battery used in this study hopes to derive further conclusion about the distinction of nonabuse-related imagery as insignificant.

Beers & DeBellis (2002) relayed a cognitive deficit in executive function in those affected by sexual violence-linked PTSD, which can be measured by the P300 and N2pc components (Brydges et al., 2014). The P3 peak is associated with updating of working memory. According to a meta-analysis in 2011 (Javanbakht, et al.), a common finding is that P300 responses are increased to trauma-related stimuli in those with PTSD.

The purpose of this research was to expand upon the current literature of psychophysiological measures and neuroimaging techniques with an ERP-based approach from a tool that is characterized by high temporal resolution, low cost, and low invasiveness (Gayle et al., under review). Further, this research intended to assess the possibility of developing a neurometric profile of college-aged women affected by sexual violence. This study used the novel Brief Neurometric Battery (BNB) (Kieffaber et al., 2016) to detect cognitive differences in a subclinical population of women affected by sexual violence-linked posttraumatic stress disorder. Previously, the BNB has been shown to distinguish age-related changes in brain function (Kieffaber et al., 2016), assess a subclinical correlation with the Adult Autism Spectrum Quotient (Gayle, Osborne, & Kieffaber, under review), and distinguish children with Autism
Spectrum Disorder (Gayle et al., under review). This research attempts to answer questions about how ERPs, elicited by the BNB, may be related to the diagnostics of sexual violence-linked PTSD.

Method

Participants

Participants were recruited through two methods: via an online system to receive credit within an introductory course at William & Mary, and on a volunteer basis through flyering and contacting support groups and social networks for those who have experienced sexual trauma. Some participants self-referred, and recruitment was nonsystematic. All research assistants agreed to confidentiality in addition to completing mandated ethical trainings, and no identifying information was provided to research assistants or included in the study beyond the informed consent document, which was kept separate from data, demographics, and questionnaire responses. 33 female students between the ages of 18 and 23 participated in the study. Three participants were removed from analysis due to incomplete collection of data (n=2), or excessive EEG artifact (n=1). Of the remaining 30 participants included in analysis, the demographics were as follows: average age 19.9 years (SD 1.27); three left-handed; two with previous concussions; and three with clinical diagnoses of PTSD within the last year. Two self-identified as Asian, two as Hispanic-Latino-Pacific Islander, one as HLPI-Caucasian, one as American Indian/Alaska Native, and one as Arab-Caucasian. 18 disclosed a history of psychiatric conditions for themselves or their family, with an additional participant who was not sure.
Measures

All assessments were self-report questionnaires available to the public. No information about the results of the questionnaires nor diagnoses were provided to the participants. All participants received resources to support and counseling services at the conclusion of the experiment and upon request.

Impact of Events Scale - Revised (IES-R; Weiss & Marmar, 1997). This assessment is a 22-item scale assessing how “distressing” a trauma-inducing-event (sexual violence) was for the participant within the last seven days. The IES-R is built on a three-factor model assessing Intrusion, Avoidance, and Hyperarousal. Each item was scored on a Likert scale of 0-4 from “not at all” to “extremely,” with total scores able to range from 0 to 88. A study of PTSD in motor vehicle accident survivors (Beck, Grant, Read, Clap, Coffey, Miller, & Palyo, 2007) found this scale had “specific cross-measure agreement in the assessment of PTSD symptomatology” (p.10), citing it as “one of the most widely-used self-report measures within the trauma literature” (abstract). The article also outlines that the Avoidance subscale may not reflect accurate scores of emotional numbing as a method of avoidance, as “many of the items reflect active avoidance” (p.11). The authors state that the Avoidance subscale may not “fit as closely with the DSM criteria for PTSD” (p.11) compared to the Intrusion and Hyperarousal subscales. Creamer et al. (2003) found high internal consistency (alpha=0.96), and proposed that “the IES-R may be sensitive to a more general construct of traumatic stress in those with lower symptom levels” (abstract). A total score of 33 gave a diagnostic sensitivity of 0.91 and specificity of 0.82 (Creamer et al., 2003; Beck et al., 2009) when compared with the general
PTSD Checklist. Of the three participants in the present study that disclosed a clinical PTSD diagnosis (all within the last year), two met the criteria for probable diagnosis and one did not. The cutoff score of 33 was used in our analyses, described later.

PTSD Symptom Checklist - Specific (PCL-S; Weathers, Herman, Huska, & Keane, 1993). This 17-item questionnaire, built from the criteria for PTSD in the DSM-IV, utilized a five-point Likert scale to determine PTSD symptom severity relevant to a specific traumatic experience. The participant was asked to respond to how “troublesome” the event (of sexual violence) was for the individual within the last month, from “not at all” to “extremely.” The PCL-S (DSM-IV) was selected for this research because of a clinician interpretation requirement for the more updated PCL-5 (DSM-V), and because the Specific scale is largely similar in structure to the nonspecific PCL-5 (U.S. Department of Veterans Affairs, 2017). According to Creamer, Bell, & Failla (2003), the IES-R and PTSD Checklist are highly correlated. Research by Blanchard et al. (1996) corroborated the original study’s findings with a female population of recently traumatized motor vehicle accident victims and sexual assault survivors, and used a cutoff score of 44 as opposed to 50 found in the original article. Further, the researchers found the overall diagnostic was validated against the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995), but individual items were less correlated.

Brief Neurometric Battery (BNB; Kieffaber, Okhravi, Hershaw, & Cunningham, 2016). The BNB was proven viable as a way to elicit eight ERPs and five oscillatory measures through an initial study published in 2016. In another study by Gayle, Osborne, & Kieffaber (under review) using the Brief Neurometric Battery, each trial consists of “(1) a standard or deviant (frequency or ISI) tone, (2) a compound visual stimulus set and (3) an auditory paired-click
stimulus (on a subset of trials).” Previous research used the BNB in the cognitive assessment of aging (Kieffaber et al., 2016), and in young children and adults with autism spectrum disorders (Gayle et al, under review; Gayle, 2016). ERPs elicited include P300, P50 suppression (reported as “P50 Diff” meaning the difference in amplitudes from the P50 elicited by two paired clicks), visual mismatch negativity (vMMN), inter-stimulus interval mismatch negativity (MMN_{ISI}), frequency mismatch negativity (MMN_{FREQ}), C1 wave, error-related negativity/positivity (ERN and P_{e}), and N2pc. The N2pc was not analyzed in this study due to technical challenges.

**Experimental Design**

The task in this study was based on the brief neurometric battery as published in 2016 by Kieffaber et al. The BNB, presented through MATLAB (Mathworks Inc., USA) begins with two periods of resting-state EEG recordings from 60 seconds with eyes closed and 60 seconds with eyes open. Task instructions appeared next, followed by a trial period for participants to become comfortable with the task. The task involves attending to the visual and auditory stimuli as presented, and pressing one of two keys on the keyboard when a noted visual cue is produced. Approximately 20 minutes of stimuli were presented. A task schematic is presented in Figure 1. Information on the frequency of stimuli can be found in Table 2.

Error-related negativity (ERN) and positivity (P_{e}) ERP components were derived by comparing correct and incorrect responses. Task feedback messages were displayed between trials to encourage accuracy and speed.
Auditory

A series of “standard” tones were presented with an interstimulus interval of 2600 ms. Standard tones were 500 Hz sinusoidal tones with a 100 ms duration. Standard tones – 1000 Hz, 100 ms duration – were replaced with “deviant” tones at a ~16% exchange rate in order to elicit the frequency-MMN ERP component. Another ~16% of standard tones were presented after a 1300 ms interstimulus interval, to elicit the interval-MMN ERP component. Auditory click pairs (square-wave tones, 1 ms each, 250ms ISI) were integrated within the standard interval to elicit the P50 ERP component. All tones were presented binaurally through pneumatic headphones (3M E-A-RTONE™ 3a) inserted prior to the start of the study.

Visual

Visual stimuli were presented for 250 ms against a black background. Presentation relative to auditory stimuli varied between 100 and 950 ms after conclusion of audio. Stimuli – an “X” or “O” and “1” or “2” – were presented laterally to the fixation point, which was continuously displayed. The task instruction was to attend to the visual stimuli – either numbers or letters, as designated – and respond using two pre-selected keys on the computer keyboard. Key-stimulus assignment was randomized across participants. Letters or numbers were randomly assigned to be the deviant stimulus, and an equal appearance of each within the set across 15% of trials was intended to elicit an oddball P300 ERP. Lateral position of the target was pseudorandomized to produce the N2pc ERP component, which was not analyzed in this study.

A rectangular sine grating in the upper or lower quarter of the screen was displayed with the display of the task-relevant stimuli in order to elicit the C1 wave ERP component. Phase grating was adjusted by 18 pixels per screen refresh, giving the illusion of movement. Spatial
frequency was 0.0083 cycles per pixel (120 pixels per cycle). Grating was oriented either horizontally or vertically, one occurring randomly at a higher frequency (87% of trials) to elicit the visual-MMN ERP component.

**Procedure**

All data was collected at the William & Mary Cognitive Psychophysiology Lab (CPL). The university’s Protection of Human Subjects Committee reviewed all questionnaires, forms, and materials before the study was conducted (approval PHSC-2016-11-08-11564-pdkieffaber), and campus psychologists were alerted to the presence of the study to be available to provide additional support as needed. The time from the start of the questionnaires to completion of the final components of the study was, on average, just over two hours, but ranged from two hours to eight days due to delays created by technical challenges. The questionnaires were always completed within an hour before the task, except for three exceptions, as explained above for study time.

Upon arrival for the study, participants provided informed written consent and were told of the opportunity to ask questions presently, throughout, and after the conclusion of the study. Participants were then given a set of questionnaires to complete, which included a demographic survey, the PCL-S, and the IES-R. Participants were asked to complete questionnaires by considering only events of physical/sexual violence/trauma that they had personally experienced. Participants were asked to mark an additional option, “N/A” (not applicable) for all questions on the IES-R and PCL-S if the participant had not experienced an event of sexual violence. Questionnaires typically took 10-25 minutes to complete. One participant took the questionnaires
twice to correct responses based on further explanation of the criteria, and the second
questionnaire is used in the analysis.

Participants were then given both a hearing and vision test. Participants were asked to
stimulate the scalp with a sanitized brush, and skin was cleansed with a PDI Electrode Prep Pad
(PDI Healthcare) or alcohol wipe. Participants were measured and fitted for a fabric cap with 72
Ag-AgCl sintered electrodes, and electrode Cz was positioned equidistant between the nasion
and inion and at the midpoint between the ears. The 64 electrodes on the net of the cap were
filled with conducting gel (ECI Electro-Gel). A ground electrode was placed in the center of the
forehead at the nasion, a reference electrode was placed at the tip of the nose, and an additional
eight electrodes were applied, with gel, to the cleansed skin areas around the eyes in order to
collect mastoid recordings as well as oscillatory movements (EOG) for removal from the data.
All functioning electrodes were recorded at levels below 20kΩ, and those that were not were
recorded below the appropriate level were removed during data analysis. Then, participants
completed the BNB procedure, as described above.

**EEG Recording**

Stimulus presentation for the experiment was presented through MATLAB (Mathworks,
Inc., USA) across approximately 20 minutes, resulting in 400 total trials. EEG data were
recorded at resting and active visual states for 60 seconds each prior to the presentation of
task-related visual or auditory stimuli. Data were recorded continuously at 1000 samples per
second while the participant remained in an electrically-shielded Faraday chamber. Data were
recorded through a high-impedance DBPA-1 Sensorium bio-amplifier (Sensorium Inc., Charlotte, VT).

**EEG Data Analysis**

Data were stored on a local network and analyzed using EEGLAB (Delorme & Makeig, 2004) and ERPLAB (Lopez-Calderon & Luck, 2014). All participants’ trials were visually reviewed by a research assistant to remove channels and segments of data impacted by excessive artifacts. Channels with excessive artifact (M=7.5) were interpolated using a spherical spline. A high-pass IIR Butterworth filter of 0.5 Hz prior to ocular artifact identification, including blinks and horizontal eye movements, removed via independent component analysis; segments of the data with sufficient artifact were cut from the data and impacted events were not included in the analysis. Data were segmented into frames of -200 to 1000 ms around each stimulus event. Data were baseline-corrected based on the 200 ms pre-stimulus interval, except for P50 data, which were baseline-corrected based on a 100 ms pre-stimulus interval. An additional high-pass filter of 10Hz was applied to the segments of data relevant to the P50 ERP analysis (Dalecki, Croft, & Johnstone, 2011).

Segmented data were averaged over trials by event type. ERP difference waveforms were created, as were grand average waveforms and topographies (Figures 2, 3, and 4). These informed latency intervals for mean amplitude measurements. See Table 1 for information on component latencies.

Rather than selecting only one of the 72 channels used to make the EEG recordings, a principal components analysis (PCA) was used to reexpress the multielectrode EEG data in
terms of only a few latent signals or factors based on the spatial covariance between electrode sites, sometimes called “virtual electrodes” (Spencer et al., 1999). Component scores obtained from the PCAs permit the expression of the activity of the virtual electrodes as a function of time. Scaled to match the microvolt scaling of the input data, these PCA time-courses were used to obtain component measurements (Kieffaber & Hetrick, 2005).

**Statistical Analysis**

All statistical analyses were performed using SPSS version 22 (SPSS Inc., Chicago, IL). Relationships between trauma and ERPs was first assessed by computing the correlations IES-R score, PCL-S score, and ERP components. Treating PTSD symptomatology as a categorical variable, participants were grouped into “no stress” and “high stress” groups using an IES-R cutoff of 33 (Creamer et al., 2003, Beck et al., 2009). Participants were grouped into the stress category “Low” if they presented an IES-R total score equal to 0 (n=10), and into the “High” category if they presented an IES-R score equal to or above 33 (n=9). A multivariate analysis of variance (MANOVA) was used to determine whether the collection of ERPs (vMMN, C1, MMN_{ISI}, MMN_{FREQ}, P300, P50 Diff, and ERN) were significantly different between Low and High stress categories. Univariate analyses, Pearson correlations, and linear regression analyses were conducted to assess PTSD-related differences in ERP measurements.

In order to complement the MANOVA, a discriminant analysis with two groups and three ERP predictors was also performed, as well as an exploratory 2x7 analysis using all ERP components. Power analysis for the 3-component discriminant function analysis with two groups and three predictor variables determined a sufficient sample size (n=20) using an alpha of .05, a
power of .80, and a large effect size ($f=40$) (Faul, Erdfelder, Lang, & Buchner, 2007; Gayle, under review). The discriminant analysis attempted to determine whether group membership could be predicted using a neurometric profile consisting of the vMMN, P300, and C1 wave component.

**Results**

A 2x8 MANOVA revealed a significant relationship between the Low and High stress groups, $[F(7, 11) = 3.602, p=.048, \text{Wilk}'s \lambda = .339]$. When measurements were entered into an exploratory discriminant analysis, ERP profiles including the vMMN, P300, MMN$_{\text{FREQ}}$, MMN$_{\text{ISI}}$, C1 wave, ERN, and P50 Difference components significantly predicted group membership (High and Low), $\text{Wilk}'s \lambda = .339, \chi^2 (7) = 14.589, p = .042$, *Canonical Correlation*: .813. In this discriminant analysis, 94.7% of original grouped cases were correctly reclassified, and 68.4% of participants were correctly classified using “leave one out” cross validation. In light of the reduced statistical power associated with the high number of predictors relative to the number of subjects, a second discriminant analysis was performed using just the best three predictors from the first analysis - vMMN, P300, and C1 wave. This discriminant function analysis led to the significant prediction of group membership (High and Low), $\text{Wilk}'s \lambda = .586, \chi^2 (3) = 8.277, p = .041$, *Canonical Correlation*: .643, with 78.9% of original grouped cases were correctly reclassified, and 73.7% of participants were correctly classified using “leave one out” cross validation.

Linear regression analysis of only High and Low IES-R total scores (n=19) with the ERP components showed no significance. Linear regression with all IES-R scores (n=30) showed no
overall significance, but showed significance with vMMN ($r = .439, p = .008$) and P300 ($r = .331, p = .037$). Linear regressions were non-significant with PCL-S scores ($n=30$).

**Discussion**

The primary aim of the current research was to utilize low-level EEG procedures in generating a neurometric profile of those impacted by sexual violence-related PTSD relative to a control population. The measure, the Brief Neurometric Battery, in this study uses a nested array of stimuli to derive multiple components in a very short period of time (approximately 20 minutes). Demonstrated significance in generating similar low-level profiles in the cognitively aged and those impacted by ASD (Kieffaber et al., 2016; Gayle, under review), paralleled the current research which successfully developed a multivariate neurometric profile related to sexual violence-linked PTSD.

The vMMN component, proven valuable to the neurometric profile of the High stress group, reveals a response to directional changes in visual stimuli, requiring the integration of visual stimuli into memory. A reduced visual mismatch negativity for the PTSD-positive group could indicate a difficulty integrating visual stimuli into memory, or suggest that high levels of stress coincide with reduced sensitivity to changes. It is important to note that vMMN alterations are aligned with the literature (described above) on memory disturbance and reduced hippocampal volume in those affected by trauma.

The C1 wave, denoting activation of the visual cortex, was larger for those in the high-stress group, which may suggest an increased sensitivity to visual input. Further, the combined understanding of the vMMN and P300 predict a heightened response to visual stimuli
but a less sensitive memory for visual stimuli, meaning that high stress may be associated with
the allocation of more neural resources to visual input at any given moment and fewer resources
allocated to the comparison of current stimuli with prior history or expectations (e.g., memory).
This conclusion parallels the hypervigilance observed in those with PTSD resulting from
neurotransmitter alterations (Southwick et al., 1999), who are constantly unsettled and may
interpret all stimuli as new, never relying on memory to distinguish deviance.

Finally, the P300 component showed a strong positive waveform, interpreted as a larger
amplitude for participants with high PTSD symptoms. This is consistent with the meta-analysis
by Johnson et al. (2013), which produced higher P3a amplitudes for those impacted by PTSD.
Unlike the present research, Johnson et al. reports a decreased P3b amplitude for those impacted
by PTSD when viewing neutral stimuli. Because of the neutrality of the stimuli in the BNB, the
large P3 amplitude elicited in the present research could require further investigation of this
discrepancy.

While these three components make up the 3-component - vMMN, C1 wave, P300 -
discriminant analysis profile of those with PTSD based on this research, we will consider the
implications of the other significant variables from the 7-component exploratory analysis. In the
P50 measure, a larger difference for the high stress group (greater suppression of the second
sound) parallels the visual response to hypervigilance: in being so connected with the stimuli of
the present (as evidenced by the C1 wave), it is likely that this attention greatly impacts attention
to closely following stimuli. The first stimulus remains in processing longer, since it does not
appear to be integrated into the memory as easily (reduced vMMN). The elicited MMN_{isi}
represents an increased response to unexpected auditory inputs. This parallels the research of
Metzger et al. (1999), which found larger heart rate and slower habituation to startle tones, and it, too, connects with the theory of hypervigilance presented above.

**Limitations**

There are several limitations to our study. The study group may not be representative of all those who have experienced sexual trauma or PTSD, as our participants were recruited from a college campus, and subpopulations most affected by violence and trauma, including people of color (namely Native Americans) (Perry, 2004), incarcersted persons (Beck et al., 2013), and LGBTQ+ individuals, especially transgender persons (National Sexual Violence Resource Center, 2015), could not be shown to make up the bulk of the study. The data found in this study may not be generalizable across sexes and genders, as this study focused solely on the collection of data from female survivors of sexual violence and cannot account for the varied data on PTSD impacts in males (Hanna & Grant, 1997; Kessler et al., 1994; Kilpatrick et al., 2003, Teicher et al, 2003). Additionally, not all participants included in the study with “high PTSD” by the IES-R scale had clinical diagnoses of PTSD, and not all of those with clinical diagnoses of PTSD were categorized into a high-symptom group.

Additionally, an article by Schalinski et al. (2015) noted that the time passed since the event or events may impact physiological responses to stress, but the summation of adversities or varied types of trauma led to stronger responses regardless of this time lapse, as measured in cortisol levels in the hair. The present research did not ask the participants at what age - or how long ago - they experienced the trauma which was surveyed in the study. Similarly, participants were not asked the relationship between themselves and the perpetrator of the sexual violence,
nor were they asked the duration of the abuse; in support of this, research by McLean et al. (2014) suggested that the characteristics of the perpetrator and the duration of abuse were found unrelated to the severity of PTSD experienced. Participants were not asked to answer the questionnaire multiple times if they experienced violence of multiple types or counts, under the presumption that the experience of PTSD may be a culmination of all experiences or stressors (Cloitre et al., 2009), and differentiation of thoughts or impacts would be difficult for participants.

This study also did not request information on resilience or protective factors for the experience of PTSD, including intelligence (Cook et al., 2008), positive coping strategies, bypassing fear response, counseling support, and social support (NIMH, 2004; Dunmore et al., 1999), which may have effects on emotional stability and the regulation of emotions, processing of errors, and managing of auditory and visual memory. Participants had a high prevalence (n=18) of psychiatric and psychologic conditions in family history, including immediate family and the participants themselves. Additionally, a majority of participants were on medication at the time of the experiment (n=18), ranging from birth control to antidepressants and autoimmune medications. Because PTSD is often accompanied by psychologic or psychiatric factors including depression, substance abuse, and anxiety disorders (Kessler et al., 1994; 1995; Kilpatrick et al., 2003), and because these factors were largely impacting the study’s population, this could not be controlled for in analyses unlike in other EEG/ERP studies.
Implications

This study has several implications and should serve to direct future research in the field, and contribute to the bank of research that informs public policy around sexual violence and trauma. This research, particularly the varied IES-R and PCL-S scores reported by those impacted by trauma, provides context for the variety of situations and conditions that those affected by sexual violence may be managing. Because of the varied severity of PTSD symptoms, it is reasonable to assume that participants in this study, and outside of it, would benefit greatly from various treatment methods. This research could be supplemented with an analysis of treatment programs and the mediating effects on PTSD symptom severity, particularly the vMMN and P300, as correlated with the IES-R. Additionally, this research serves as a starting point from which to analyze revictimization and the subsequent likelihood of violence or stress resulting from trauma. As noted in the limitations of this study, the research did not ask the number, variety, or duration of experiences of sexual violence; because of this there is no way to analyze the cognitive psychophysiological effects of revictimization and compare ERPs or PTSD symptomatology scores. Najdowski & Ullman (2011) reported that a survey of women demonstrated more maladaptive and adaptive coping strategies among those who were revictimized within the year-long study. Future research would do well to continue this analysis and associated resources, particularly in relation to the vMMN and P300.

This research and others that supplement it need to further investigate the relationship of emotional expression, regulation, and awareness for survivors of sexual violence. While this research is unable to explain revictimization, perhaps the correlated vMMN and P300 could
provide a springboard from which to understand the psychological and neurological factors that increase the vulnerability of survivors to revictimization, such as numbing to threat cues, reduced risk perception, or even hypervigilance (Lalor & McElvaney, 2010; National Sexual Violence Resource Center, 2012).

This research also emphasizes not only the field of research on sexual violence, but also on other life stressors that extend across the lifetime, or begin at an early age (Sherin et al., 2011). Research in the domains of health psychology and public health would benefit from additional neuroscientific research on life stressors and related PTSD—including discrimination/harassment, drug abuse/disorder, poverty, immigration status, and social networks—that can begin in early development and continue to impact health throughout the life course (Anda et al., 2006; National Research Council & Committee on Population, 2013). Research initiatives could be expanded to identify correlations between this relationship and physiological data present in the literature focusing on the HPA axis, impacted neurological and psychological development, the neuroendocrine system, and brain potentials (ERPs). Sociopsychological research could look into the impacts of cumulative trauma within and across the domains described above, as begun in the literature (Follette et al., 1996; Sherin & Nemeroff., 2011).

Future research could, as well, investigate not just the prevalence of stressful events, but any discrepancy between event severity and an individual’s perception of such stress, and the correlations of this with PTSD. All PTSD assessments in our questionnaire were self-report measures, and as such relied on the individual’s perception of the event and their symptomatology since. It is possible that a more objective measure of the events studied here could elicit significant correlations with ERPs if controlling for the individual’s perception of
their PTSD symptomatology and instead assessing the neurobiological impacts of the trauma, not the outwardly communicated response to it (or vice versa, controlling for the severity and measuring only the participant’s perception).

This paper contributes to the literature around sexual violence-linked PTSD in college-aged women, and serves to inform the assessment and treatment processes through an understanding of the implications of affected vMMN, P300, and C1 wave ERP components. This paper also supports the claims of the Brief Neurometric Battery (BNB) in eliciting low-level processing through ERPs as measured in EEG.
References


with and without early childhood sexual abuse and posttraumatic stress disorder (PTSD).

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http://doi.org/10.1016/j.clinph.2016.01.023


doi:10.1002/jts.20610


https://www.bjs.gov/content/pub/pdf/aic02.pdf


doi:10.1017/S0048577299981180


### Tables

Table 1.

*ERP component latencies.*

<table>
<thead>
<tr>
<th>ERP</th>
<th>Latency</th>
</tr>
</thead>
<tbody>
<tr>
<td>vMMN</td>
<td>125-275 ms</td>
</tr>
<tr>
<td>C1 wave</td>
<td>20-75 ms</td>
</tr>
<tr>
<td>P300</td>
<td>300-750 ms</td>
</tr>
<tr>
<td>MMNisi</td>
<td>150-250 ms</td>
</tr>
<tr>
<td>P50</td>
<td>-40-100 ms</td>
</tr>
<tr>
<td>MMNfreq</td>
<td>110-210 ms</td>
</tr>
<tr>
<td>ERN</td>
<td>-50-100 ms</td>
</tr>
<tr>
<td>Pe</td>
<td>100-350 ms</td>
</tr>
</tbody>
</table>
Table 2.

*Trial counts for stimulus presentation, A) visual stimuli and B) auditory stimuli.*

<table>
<thead>
<tr>
<th>A) Visual Trial Type</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>High probability targets (standard)</td>
<td>350</td>
</tr>
<tr>
<td>Low probability targets (deviant)</td>
<td>50</td>
</tr>
<tr>
<td>High probability distractors (standard)</td>
<td>350</td>
</tr>
<tr>
<td>Low probability distractors (deviant)</td>
<td>50</td>
</tr>
<tr>
<td>Left target</td>
<td>200</td>
</tr>
<tr>
<td>Right target</td>
<td>200</td>
</tr>
<tr>
<td>Sine grating at top</td>
<td>200</td>
</tr>
<tr>
<td>Sine grating at bottom</td>
<td>200</td>
</tr>
<tr>
<td>Sine grating in standard direction</td>
<td>350</td>
</tr>
<tr>
<td>Sine grating in deviant direction</td>
<td>50</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B) Auditory Trial Type</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard Tone (Hz)</td>
<td>310</td>
</tr>
<tr>
<td>Deviant Tone (Hz)</td>
<td>60</td>
</tr>
<tr>
<td>Deviant ISI</td>
<td>60</td>
</tr>
<tr>
<td>Paired Clicks</td>
<td>90</td>
</tr>
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</table>
Table 3.

Means and standard errors for BNB metrics.

<table>
<thead>
<tr>
<th>BNB Metric</th>
<th>Control: IES-R=0 Mean (SE)</th>
<th>PTSD: IES-R&gt;33 Mean (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>vMMN</td>
<td>-.66 (.27)</td>
<td>-.82 (.52)</td>
</tr>
<tr>
<td>C1 wave</td>
<td>1.20 (.52)</td>
<td>2.96 (.46)</td>
</tr>
<tr>
<td>P300</td>
<td>4.95 (1.06)</td>
<td>7.20 (1.11)</td>
</tr>
<tr>
<td>MMNisi</td>
<td>-.51 (.42)</td>
<td>-1.48 (.56)</td>
</tr>
<tr>
<td>P50 Diff</td>
<td>.19 (.07)</td>
<td>.35 (.13)</td>
</tr>
<tr>
<td>MMNfreq</td>
<td>-.47 (.54)</td>
<td>-1.04 (.15)</td>
</tr>
<tr>
<td>ERN</td>
<td>.08 (.50)</td>
<td>.26 (.11)</td>
</tr>
</tbody>
</table>

Table 4.

Discriminant analysis structural matrix values for A) three-component analysis (alpha=.05, power=.80, effect size $f=.40$, minimum sample size $= 20$, and B) exploratory seven-component analysis.

A) Structural ERP Value

<table>
<thead>
<tr>
<th>ERP</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>vMMN</td>
<td>0.752</td>
</tr>
<tr>
<td>C1 wave</td>
<td>0.724</td>
</tr>
<tr>
<td>P300</td>
<td>0.422</td>
</tr>
</tbody>
</table>

B) Structural ERP Value

<table>
<thead>
<tr>
<th>ERP</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>vMMN</td>
<td>0.453</td>
</tr>
<tr>
<td>C1 wave</td>
<td>0.436</td>
</tr>
<tr>
<td>P300</td>
<td>0.254</td>
</tr>
<tr>
<td>MMNisi</td>
<td>-0.244</td>
</tr>
<tr>
<td>P50 Diff</td>
<td>0.189</td>
</tr>
<tr>
<td>MMNfreq</td>
<td>-0.139</td>
</tr>
<tr>
<td>ERN</td>
<td>0.032</td>
</tr>
</tbody>
</table>
Figure 1. A) Task version of one sample schematic of 4 trials in which the target stimuli are the triangle and the square and the distractor stimuli are the diamond and the circle. Each trial consisted of one visual presentation and up to two auditory presentations. This graphic shows different directions (horizontal v. vertical) of the sine grating, while the BNB version in this study displayed left-movement and right-movement differences in the sine grating. B) Timing of a single trial. The duration of each trial was 2600 ms, and both type and presence/absence of auditory stimuli varied between trials.
Figure 2. Mismatch negativity waveforms and peak latencies. A) vMMN difference waveform, 125-275 ms. B) MMNfreq difference waveform, 110-210 ms. C) MMNisi difference waveform, 150-250ms.
Figure 3. ERP waveforms and peak latencies. A) Grand average P300 difference waveform. Peak amplitude was measured between 300 and 750 ms. B) Grand average C1 raw waveform. Peak amplitude was measured between 20 and 75 ms.
Figure 4. ERP waveforms and peak latencies. A) Grand average ERN raw waveform. Peak amplitude for ERN was measured between -50 and 100 ms, and for P_e between 100 and 350 ms. B) Grand average P50 raw waveforms for sound 1 (S1) and sound 2 (S2). P50 suppression was measured between -40 and 100 ms.
Figure 5. Means and standard error for ERP components.

Figure 6. Values produced in structural matrices of discriminant analyses.