DEDICATION

To my dog, Jelly, for being a buoy during the hard times.
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LIST OF ABBREVIATIONS

ANOVA: Analysis of variance

CAPS: Clinician-Administered PTSD Scale

DSM-IV: Diagnostic and Statistics Manual of Mental Disorders, 4th Edition

EEG: Electroencephalogram

GABA: Gamma-aminobutyric acid

ISI: Interstimulus interval

kHz: Kilohertz

MEG: Magnetoencephalogram

MIRECC: Mental Illness Research, Education, and Clinical Center at the W.G. Hefner VA Medical Center

MRI: Magnetic Resonance Imaging

msec: Milliseconds

OEF/OIF: Operation Enduring Freedom/Operation Iraqi Freedom

PCL-M: Posttraumatic Stress Disorder Checklist, Military Version

PTSD: Posttraumatic Stress Disorder

RPSQ: Rivermeade Post Concussion Symptoms Questionnaire

SD: Standard deviation

SSRI: Selective serotonin reuptake inhibitors

TBI: Traumatic Brain Injury
ABSTRACT

Selecting, processing, and interpreting stimuli is necessary for a healthy and autonomous person. A component of this process is sensory gating, or the ability to filter extraneous information in the environment and select what is important for subsequent processing. A common symptom of Posttraumatic Stress Disorder (PTSD) is sensory flooding, which is an overwhelming feeling of too many stimuli at once. Literature suggests that sensory gating deficiencies may underlie problems with sensory flooding in PTSD patients. Two groups of combat veterans from Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF), those diagnosed with PTSD and those not diagnosed with PTSD, underwent sensory gating testing using the paired-click paradigm while neuronal responses were recorded with magnetoencephalogram (MEG) technology. Results showed that the PTSD and control groups both had deficient sensory gating, demonstrated by high S2/S1 ratios, counter to previous studies’ findings. A significant laterality effect was found for the PTSD group for response amplitude to S1 and S2, with the left hemisphere showing larger responses in both cases. These findings suggest that either the controls are more similar to PTSD patients than what was expected, or that this study failed to capture true differences between the two groups. There also may be laterality differences in cognitive processing found in PTSD patients.
INTRODUCTION

Posttraumatic stress disorder is a type of anxiety disorder that develops following a traumatic event outside the realm of usual life. The disorder is characterized by three clusters of symptoms: re-experiencing the event, avoiding reminders of the trauma, and chronic physiological arousal (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). A common issue included in the physiological arousal cluster is sensory flooding, or an overwhelming feeling of fragmentation and confusion from too much information at one time.

Selecting, processing, and interpreting stimuli is a necessary ability of a healthy and autonomous person. A component of this process is sensory gating, which is the ability to filter the extraneous information in the environment and select what is important at the time for subsequent processing and interpretation (Mayer et al., 2009). A current topic in neurophysiological research is whether patients with PTSD have deficient sensory gating, leading to sensory flooding (Karl, Malta, & Maercker, 2006). If this mechanism is contributing to sensory flooding, then this provides a starting point for developing treatment to help patients.

Although PTSD research is still in its beginning stages, sensory gating is an important element to examine because it may underlie many day-to-day symptoms in these individuals, such as suppression of extraneous noise, attention, concentration, irritability, stress, and sleep disturbances. Much can be learned about the measure used to study sensory gating and discrepancies that have been found between healthy and deficient individuals.
It is specifically important to study the mental health of combat veterans. It is estimated that between 11 and 30 percent of veterans from Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) will be afflicted with PTSD (Goulding et al., 2009). Many of these veterans have already returned home since deployment, and a large number will do so in the coming years. It is necessary to better understand the role of sensory gating in this population.

The paired-click paradigm is commonly used to study sensory gating. Neutral sound pairs (S1 and S2) are presented in quick succession and EEG technology is capable of recording the neuronal responses elicited by these sounds. In theory, the amplitudes of the neuronal responses that are obtained from this task are representative of how an individual processes information (Karl et al., 2006). A commonly used method of indexing responses in sensory gating is by using the peaks of activity near particular latencies after stimulus onset. The dependent variables of interest are the amplitude and latency of these peaks, usually at 50, 100, and 200 msec poststimulus (Boutros, Korzyukov, Jansen, Feingold, & Bell, 2004). It takes the activation from the auditory stimuli about 20 milliseconds to reach the cortex and the first wave that is recorded is the P50 wave (Freedman et al., 1987). The P50 wave is named for its positive polarity and the fact that it occurs about 50 milliseconds after the onset of the auditory stimulus. The P50 wave is selected as the highest peak of activity between 40 and 80 milliseconds after the onset of the stimulus (Erwin & Buchwald, 1986). A range is used because the latency of the P50 response varies a few milliseconds per individual and per stimulus. When the P50 wave is averaged for all presentations of S1 and separately for all presentations of S2, they are called the grand averages of the evoked potentials. The ratio of the responses
(S2/S1) can be calculated with the grand averaged evoked potentials, which is a representation of the sensory gating ability of the individual. It is assumed that the response to S2 will be smaller than the response to S1, indicating that some filtering has occurred. Therefore, larger ratios indicate less filtering, or deficient sensory gating. It is also possible to examine the N100 and P200 peaks after the stimulus onset (Boutros, Korzyukov, Jansen, Feingold, & Bell, 2004), but the P50 occurs before conscious processing, and it is less susceptible to attentional manipulation, so the ratio of the P50 amplitude of S2 to S1 is the most typical measure of gating.

Sensory gating is commonly measured using electroencephalogram technology, or EEG. The goal of using this technology is to see levels of cellular activity in a noninvasive manner. The electrical transmission between neurons, the comparable level of activity of the neurons, and the location within the brain are all factors that can be investigated. The process of measuring EEG first involves placing a single electrode at the Cz location, which is at the vertex of the head, and a ground electrode on the forehead or by the ear. The EEG technician measures the scalp and uses abrasive gel to clean the spot where the electrode will be placed. The electrode is connected to wires on a computer with a program that records the electrical activity extracellularly. The participant is asked not to move around or talk unless directed to do so. The goal is to be able to register electrical activity of neurons, which show the temporal properties of brain circuits (Michel et al., 2004).

Another method of collecting sensory gating EEG is by using multiple channel recordings, which involves placing several other electrodes on the scalp in the same fashion. Some researchers believe that, like increasing the number of questions on a...
survey, multiple sites yield a more accurate response compared to only using one site (Clementz & Blumenfeld, 2001). The signal to noise ratio (the level of strength of the desired response relative to the background noise) is much smaller with only one electrode, whereas the background noise can be averaged and filtered with more EEG electrode sites.

There are some deficiencies in the EEG measurement process, regardless of the number of channels used. The technique registers cellular activity from an extracellular location (the scalp). This leads to interferences from many other simultaneous electrical signals in the brain and general noise from the environment (Hari et al., 2010).

Occasionally instead of using EEG, magnetoencephalogram (MEG) is used because there is much less distortion from internal noise (Hari et al., 2010). Instead of electrical signals, the MEG records magnetic signals from postsynaptic potentials and “virtual channels” inside the brain are triangulated using multiple electrodes. (Schwartz, Edgar, Gaetz, & Roberts, 2010). A structural MRI can then be mapped on top of the virtual channels to show what areas of the brain may be activated.

The following seven studies are the only studies to date that have investigated sensory gating deficits in PTSD patients using the paired-click paradigm. Most of the research concludes that there are larger S2/S1 ratios in the PTSD samples, indicating deficient gating, compared to the control samples (Ghisolfi et al., 2004; Gillette et al., 1997; Holstein et al., 2010; Hunter et al., 2011; Neylan et al., 1999; & Skinner et al., 1999). However, one study found no sensory gating differences between Vietnam War nurses with PTSD and controls (Metzger et al., 2002).
Gillette et al. (1997) investigated the suppression of the P50 response in four groups: 10 Vietnam combat veterans with PTSD, five alcohol dependents without PTSD or combat exposure, five controls with combat exposure, and five controls with no combat exposure and no alcohol dependence. All participants were male, age-comparable, and free of medications other than acetaminophen or ibuprofen. The study consisted of the standard paired-click paradigm in three blocks with different interstimulus intervals (ISIs): 250, 500, and 1000 msec. S2/S1 ratio results showed a significant effect of group and ISI, and no group x ISI interaction. The PTSD group had a significantly larger S2/S1 ratio, denoting less gating, than all comparison groups (p<.05), and there were no significant differences between the comparison groups. The groups produced lower S2/S1 ratios at 250 msec ISI compared to 1000 msec ISI, and lower S2/S1 ratios at 500 msec ISI compared to 1000 msec ISI. There was no significant difference of ratio between groups at the 250 and 500 ISIs. This shows that S2/S1 ratios were lower at shorter intervals. P50 latency and amplitude in response to S1 was not significantly different between groups or ISIs, and there was no group x ISI interaction. Interestingly, at the 250 ISI, the S2/S1 ratio for the PTSD group was positively and significantly correlated with the participants’ Clinician Administered PTSD Scale (CAPS) score on re-experiencing intensity. This study was one of the first to propose the lack of sensory gating in veterans with PTSD, and to show that the S2/S1 ratio decreased with a shorter ISI. Importantly, this study showed that this quantitative measure of sensory gating is reflective of a symptom’s intensity.

Neylan et al. (1999) conducted a study of sensory gating deficiencies in 15 male Vietnam combat veterans with PTSD and 12 male controls with no combat history.
Patients with PTSD were recruited if they had no history of substance abuse in the past year, were not diagnosed with any other mental disorders, and were free of medications, except for four subjects who were taking selective serotonin reuptake inhibitors (SSRIs). Control participants were recruited if they were medically healthy, not taking medication, had no neurological or Axis I disorders, and were not substance abusers. The PTSD group and the control group were comparable in level of education, but the PTSD group was slightly older in average age than the control group (PTSD 49.6 ± 2.3 years, control 45.3 ± 6.5 years, two-tailed $t, p = 0.048$). A standard paired-click paradigm was employed, with 500 msec between pairs, and intertrial interval varying between 7 and 8 seconds. Results showed that the S2/S1 ratio was significantly larger in the PTSD group (mean = .408, SD = .126) compared to the control group (mean = .213, SD = .126, two-tailed $t, p = .024$). The P50 response amplitudes to S1 were not significantly different between groups. This study suggests that the sensory impairment is not in the response amplitude of S1, but rather in the gating of repeated stimuli.

Skinner et al. (1999) conducted a study of sensory gating in a sample of 10 male combat veterans with PTSD. Two participants had a history of alcohol dependence, so comparison groups included five male alcoholic controls, five male combat-exposed controls, and five male normal controls. The sample also included nine female rape victims that had PTSD, and nine female normal controls. All participants were age- and education- matched and were free of medication other than acetaminophen or ibuprophen. Participants were tested with three blocks of the paired-click paradigm with different ISIs for each block (250, 500, and 1000 msec), and blocks were randomly ordered. Pairs were presented every five seconds. At the 250 msec ISI, the male PTSD
group had a higher S2/S1 ratio compared to each male comparison group (PTSD vs. controls, \( t = 7.54, p < 0.01 \); PTSD vs. alcoholics, \( t = 3.56, p < 0.05 \); PTSD vs. controls, \( t = 5.77, p < 0.01 \)). Sensory gating was not significantly different for the male groups at 500 or 1000 msec ISIs, despite numerical differences. At the 250 msec ISI, the female PTSD group had larger S2/S1 ratios compared to the female control group (\( t = 13.82, p < 0.01 \)). For male participants, there was no group difference in P50 response amplitude to S1 or P50 response latency to S1. The same was true for female participants. Importantly, this study only found sensory gating differences at the 250 msec ISI, which is shorter than what is commonly used. Also importantly, there was no gender difference between male controls and female controls at any of the ISIs, and no gender difference in percent response at each ISI between male and female PTSD participants.

Metzger et al. (2002) conducted a study of 24 female Vietnam nurses with current PTSD and 24 female Vietnam nurses never having PTSD, using the paired-click paradigm. This population is informative because, of all trauma populations that have been studied (veterans from Vietnam, Korea, World War II; childhood sexual abuse victims; rape victims), nurses have the largest magnitude responses to traumatic imagery while having comparable scores to other non-combat related PTSD patients on the CAPS (Orr & Roth, 2000). Also, nurses also have very high levels of intellectual functioning, considering that 50 percent had obtained a college degree, and 80 percent had remained in the nursing field post-service. These findings counter the idea that individuals with PTSD have generally impaired cognitive functioning, because nurses were still able to perform at a high level in their careers. In Metzger et al.’s study, the paired-click paradigm was composed of 75 pairs of clicks, 1 msec in duration, with 250 msec between
the clicks, and 5 seconds between trials. In contrast to previous findings, results showed that the PTSD group did not significantly differ from the control group in the S2/S1 ratio and did not differ in response amplitude or latency to either S1 or S2. As a next step, the researchers classified each participant by “gater” (S2/S1 < 0.40) or “non-gater” (S2/S1 > 0.40). In both groups, 38 percent of the participants were classified as “non-gaters” (9 of 24 PTSD, 9 of 24 control), resulting in no difference between groups. This was the first major study to disagree with previous sensory gating research on individuals with PTSD.

Ghisolfi et al. (2004) conducted a study of 12 individuals who were previously diagnosed with PTSD related to urban violence (four men, eight women), which included a variety of situations including armed assault, physical abuse, torture, rape, and threatening work accident. The study also included 12 individuals with schizophrenia (four men, eight women), and 24 healthy controls (eight men, 16 women). Controls could not be taking medication besides nicotine and oral contraceptives, but all participants with schizophrenia were taking atypical antipsychotics. Participants with PTSD were taking a variety of antidepressants, anxiolytics, and mood medications, with some participants taking two or more medications simultaneously. Psychiatric comorbidities in the individuals with PTSD included depression and impulsive behavior disorder. All groups were balanced for age and gender. The paired-click paradigm employed 1 msec duration clicks, with 500 msec between clicks in the pair and 5 seconds between trials. Results showed that the PTSD group had significantly larger S2/S1 sensory gating ratios compared to the control group, but not compared to the schizophrenia group. The response amplitude to S2 was significantly larger in the schizophrenia group compared to the control group, but not in comparison to the PTSD group. P50 amplitude and latency
in response to S1 was not significantly different between groups. There were no gender differences. This study is significant because it provides evidence that individuals with PTSD, even when the cause of the disorder is varied, produce significantly larger sensory gating ratios compared to healthy controls. However, other variables in the study, like the lack of control in medication use, dosage, and psychiatric comorbidity with the participants’ PTSD, must be taken into consideration when forming conclusions about this study.

Holstein et al. (2010) conducted a sensory gating study involving 23 patients with PTSD related to various types of trauma, including sexual assault, physical assault, car accident, rape, abuse, and combat exposure. Thirteen of the 23 PTSD participants were taking antidepressants, anxiolytics, or atypical psychotics. Eighteen controls were matched for age and gender and were not taking any medications. The paired-click paradigm consisted of 70 pairs of clicks that were 1 msec in duration, 500 msec apart, and had an intertrial interval of 6 to 10 seconds. Their measure of suppression was calculated as such: \( |1 - \frac{\text{amplitude}_{S2}}{\text{amplitude}_{S1}}| \times 100\% \). It is more common for sensory gating studies to calculate sensory gating by using S2/S1, but Participants also completed the Hopkins Symptom Checklist (SCL-90-R) (Parloff, Kelman, & Frank 1954). Results showed that the PTSD group showed significantly less P50 suppression than the control group. Analysis of stimulus type revealed that the response amplitude to S1 was not significantly different between groups, but the response amplitude to S2 was significantly higher in the PTSD group. There were no differences between groups in regards to response latencies. A negative correlation was found between the suppression ratio and the indices of the SCL-90-R, indicating that higher psychopathological
symptoms were associated with a lower sensory gating ability. However, this correlation accounted for low variance (< 14%).

Hunter et al. (2011) is the only study to date that has conducted a sensory gating study of combat veterans with PTSD with MEG technology. Participants were seven Vietnam combat veterans with PTSD and ten matched controls based on age, sex, and level of education who had no exposure to combat. Diagnoses were established with the Clinician-Administered PTSD Scale for DSM-IV (CAPS), and several patients with PTSD had comorbid depression or substance abuse disorder in remission for a minimum of six months. All participants included in the study were free of medications. The paired-click paradigm consisted of 150 pairs of clicks, each click being 3 msec in duration, with 500 msec between clicks in the pair, and 7 to 11 seconds between trials. The task was completed in a MEG device, followed by a structural MRI scan where participants had no task. P50 responses were selected from the position of Heschl’s gyrus, the location of the primary auditory cortex, in the left and right auditory cortices. Results showed that the S2/S1 ratio was significantly larger in the PTSD group compared to the control group only in the right hemisphere, indicating a lateralized deficit. The left hemisphere did not show a significant group difference. There was a significant difference between the response amplitudes to S1 and S2 in both PTSD and control groups, and in hemisphere, where the response amplitude to S1 was significantly larger than the response amplitude to S2, except for the right hemisphere in the PTSD group. This indicates that the changing S2 value is the driving force behind the S2/S1 ratio. Interestingly, results showed a significant positive correlation, $r(6) = 0.87, p = 0.02$, between CAPS hyperarousal scores and response amplitude to S1, indicating that
participants with larger responses to S1 tended to have worse symptoms. There was also a significant negative correlation, $r(7) = -0.86, p = 0.01$, between the right hemisphere S2/S1 ratio and total score on the PSTD Symptom Severity Scale (PSS-SR), indicating that better gating is associated with worse symptoms. These two correlations are counterintuitive, because it would be expected that sensory gating deficits would be associated with worse symptoms, and not the other way around. Hunter et al.’s proposed explanation is that what is seen as a sensory gating deficiency could actually be a protective mechanism and reduces the severity of symptoms. Studies on symptom severity in PTSD and sensory gating are sparse, so more research is needed to be able to draw more informed conclusions.

While there is not much research on sensory gating in PTSD patients, there are multiple decades of research on sensory gating in schizophrenia patients. While PTSD and schizophrenia are separate disorders, sensory flooding is one symptom that overlaps between them, so the schizophrenia population can be a useful reference for emerging research on PTSD. In a meta-analysis of 58 studies reporting P50 S2/S1 ratios and amplitudes for schizophrenia patients and healthy individuals, results showed that schizophrenia patients had larger mean amplitude responses to S2 in 52 studies (89.66%) and a larger mean ratio in 56 studies (96.55%) (Chang, Arfken, Sangla, and Boutros, 2011). These findings suggest several things. First, the schizophrenia population almost always shows deficient sensory gating when compared to controls, so the filtering mechanism likely underlies some sensory aspects of the disorder. Secondly, the larger response amplitude to S2 in schizophrenia patients provides support that the S2 value is
driving the larger S2/S1 ratio in patients with sensory gating deficiencies, which is an assumption made when using the S2/S1 ratio as a measure of filtering.

There is also little empirical evidence suggesting where, specifically, sensory gating is occurring. The neural auditory pathway includes the inferior colliculus, medial geniculate nucleus of the thalamus, and auditory cortex. Current reports suggest that the sensory gating response is mediated by the auditory cortex, the thalamus, and contributions from the prefrontal cortex dampening the response to S2 (Korzyukov et al., 2007; Mayer et al., 2009).

Mayer et al. (2009) conducted a study using a variation of the paired-click paradigm, where pairs of identical or non-identical clicks were presented to participants to examine the cortical and subcortical regions of the brain that are active during sensory gating. Participants were in a functional MRI device during the presentation of the clicks. The assumption was that there would be more blood flow in areas of the brain that were active during gating. Results showed that a large network of brain structures was involved in the gating of stimuli. Specifically, key areas included the bilateral auditory cortex, bilateral prefrontal cortex, and the right ventral lateral nucleus of the thalamus. No hippocampal activation was seen during the presentation of either type of clicks. In addition, they found that the left auditory cortex may play a role in selecting the stimuli that should be gated or receive subsequent processing. Mayer et al. (2009) confirmed results from Korzyukov et al. (2007), which showed that the frontal lobe, and specifically the prefrontal cortex, is the main mediator of sensory gating. Korzyukov et al. also saw that the P50 was generated in the temporal lobes, but more specificity was not described.
As previously described, Hunter et al. (2011) selected P50 responses from Heschl’s gyrus in the left and right auditory cortices in order to determine laterality differences between PTSD patients and controls, and results were successful in determining that P50 responses were gated at this location, and the S2/S1 ratio was significantly larger in the PTSD group only in the right hemisphere, indicating a lateralized deficit. The left hemisphere did not show a significant group difference.

The reasons behind the proposed sensory gating deficiencies in the PTSD population are varied. However, there is an emerging hypothesis about failure to learn safety. According to Rothbaum and Davis (2003), in individuals with specific phobias, fearful reactions will only occur in the presence of the fearful stimuli. However, patients with PTSD have a much flatter generalization gradient, meaning that they do not discriminate as much between situations that may cause stress versus those that may not. For patients with PTSD, fear and stress are displayed even in the presence of safety cues.

There is also the possibility that attending to all stimuli, whether redundant or irrelevant, is a developed mechanism in combat because it enhances chances of survival. It must be considered that some neurophysiological mechanisms that would be considered problematic in a normal situation are actually imperative for survival in other situations. The functionality of a sensory gating “deficit” is very plausible.

Finally, there has been an established relationship between low hippocampal volume and vulnerability for PTSD post-combat. In a study of PTSD among monozygotic twins, low hippocampal volume showed to be a genetic risk factor for PTSD after combat exposure, but not a genetic risk factor for depression, which is the most highly comorbid mental health disorder associated with PTSD (Stander et al.,
2014). The relationship between depression and PTSD will be discussed in more detail at a later point. While the hippocampus has not been shown to be the generator of P50 responses in humans (Grunwald et al., 2003; Korzyukov et al., 2007), the proposition that sensory gating deficiencies are due to physiological changes is likely.

**Hypotheses**

In this study, two groups of combat veterans from Operation Enduring Freedom/Operation Iraqi Freedom were included: those who had been diagnosed with PTSD, and those who had not. All participants listened to paired clicks, described in detail at a later point, while MEG technology was able to record their neurological responses from the left and right auditory cortices.

The first objective of this study was to determine the sensory gating ability for the two groups. The second objective was to determine if any sensory gating discrepancies were due to laterality differences. Variables of interest were the P50 response amplitude and latency to S1 and S2, and the S2/S1 ratio in the left and right auditory cortices. First, it was hypothesized that the PTSD group and control group would have no differences in response amplitude or latency to S1 in either hemisphere. Second, the PTSD group would have smaller response amplitude to S2 compared to the control group in both hemispheres, indicating that there was less suppression of the response to redundant stimuli. There should be no differences between response latency to S2 between groups. Third, the PTSD group would have larger S2/S1 ratios (indicating poorer gating) in both hemispheres compared to the control group. Next, the response amplitude to S2 should be the driving force behind the S2/S1 ratio, since the S1 value should be constant. There
should be no laterality difference for this association. Finally, S1 amplitude and latency values should not be correlated in either hemisphere.

The third objective of this study was to determine the level of symptom severity that both groups were experiencing and whether symptom severity was associated with the sensory gating ratio. Participants completed the Posttraumatic Stress Disorder Checklist, Military version (PCL-M) at the beginning of their scheduled testing session. Higher scores on the PCL-M should be positively associated with S2/S1 ratios, indicating that higher levels of symptom severity are related to more deficient sensory gating ability.
METHOD

Participants

Participants were 16 combat veterans from Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) drawn from a database at the Mental Illness Research, Education, and Clinical Center (MIRECC) at the W.G. Hefner VA Medical Center in Salisbury, North Carolina. All participants completed a diagnostic interview (Structured Clinical Interview for DSM Disorders, DSM-IV edition) at the W.G. Hefner VA Medical Center and allowed further contact for research participation opportunities. Exclusion criteria included ferrous metal, including pacemakers and other implants, due to their interference with magnetic scanning procedures. Standard dental fillings were acceptable but other orthodontic implants were excluded. Other exclusion criteria included individuals with traumatic brain injury, chronic pain, neurological disorders, or active substance abuse disorders. Participants could not be taking anticonvulsants, benzodiazepines, or GABA agonists. Participants also had to be capable of complying with task instructions during scans. Each participant received $250 in compensation for participation and cost of travel to and from Wake Forest Baptist Medical Center, where the study took place.

A total of 16 participants were included in this study, six that were diagnosed with PTSD and ten controls that had no psychiatric disorders. Given the small sample size, demographics between groups were as homogeneous as possible. All participants were male and right-handed. The PTSD group had an average age of 43.3 years and average education of 13 years. The control group had an average age of 38.1 years and average education of 14 years.
Procedure

Participants were scheduled for imaging at the Wake Forest Baptist Health Medical Center to undergo structural magnetic resonance imaging (MRI), magnetoencephalogram (MEG), and electroencephalogram (EEG), which required approximately five hours of participation. Upon arrival, participants gave consent to participate in the study and then completed two questionnaires, the Posttraumatic Stress Disorder Checklist – Military Version (PCL-M) (VA National Center for PTSD, 1993) and the Rivermeade Post Concussion Symptoms Questionnaire (RPSQ) (Eyres, Carey, Gilworth, Neumann, & Tennant, 2005).

The PCL-M is a 17-item questionnaire consisting of questions about problems and complaints that combat veterans commonly have in response to significant stress, including but not limited to physiological and psychological issues such as heart pounding, sweating, disturbing memories, difficulty concentrating, hyper-alertness, and emotional numbness. Instructions are to mark an answer on a scale of 1 to 5 to indicate how much the issue has bothered the participant in the past month, 1 being not at all and 5 being extremely bothersome. Results are scored in a three-category algorithm, with a score of 17 to 33 indicating low posttraumatic symptoms, 34 to 43 indicating moderate posttraumatic symptoms, and 44 to 85 indicating high posttraumatic symptoms. In this study, the PTSD group averaged 49.83, SD = 12.51, placing them in the high posttraumatic symptoms category; all participants scored in the high posttraumatic symptoms category except for one participant, who placed in the low posttraumatic symptoms category. The control group averaged 19.2, SD = 2.62, placing them in the
low posttraumatic symptom category. All participants in the control group scored in the low symptom category.

The RPSQ is a 16-item questionnaire consisting of symptoms that are commonly reported after head injury or accident. Participants are asked to rate how severe the problem has been for them currently, compared to before the accident, on a 0 to 4 scale where 0 indicates that they do not experience the symptom and 4 indicates that it is a severe problem. The first three questions, the “RPSQ-3,” consist of only physiological symptoms, including headaches, dizziness, and nausea or vomiting. These questions are associated with the early symptom cluster in diagnosing head injury severity, and a high score in this section indicates close monitoring of the individual and early medical attention. The last 13 questions, the “RPSQ-13,” including fatigue, frustration, concentration, and restlessness, are associated with later clusters of symptoms and have a greater impact on psychosocial functioning and overall lifestyle. Although this questionnaire mostly applies to individuals with concussion, many of the problematic symptoms of PTSD overlap, and this information can be useful. Out of a possible 12 points on the RPSQ-3, the PTSD group averaged 4.6 and the control group averaged 2.4, indicating that participants in both categories did not consider the symptoms to be a severe problem. Out of a possible 52 points on the RPSQ-13, the PTSD group averaged 32.2, indicating moderate issues with psychosocial symptoms. The control group averaged 9.3, indicating few issues with the same symptoms.

Overall, the demographics of the groups were as expected for patients with PTSD and control individuals. There was a high discrepancy between the groups in descriptive symptoms, on both the PCL-M and the RPSQ.
After completing the questionnaires, participants were escorted to the MEG lab, where they underwent simultaneous MEG and EEG data collection. A CTF Systems, Inc. MEG 2005 neuromagnetometer system was used in this study. The MEG had 275 sensors surrounding the head, spaced by 22.4 mm on average, which is able to triangulate points of activity within the brain. The entire apparatus is housed in a magnetically shielded space. After being introduced to the scanner and the space, participants had their scalps cleaned and fitted for EEG electrodes by a certified medical technician. Once sitting in the MEG, they were then given a threshold hearing test to determine the level of sound necessary for the presentation of the acoustic stimuli. Participants underwent ten minutes of baseline recording with their eyes open. They were instructed to try to minimize excessive blinking and movement of the head and body. They were then introduced to the sensory gating task (described below) and instructed to silently count the pairs of clicks that they heard to ensure that they were paying attention to the tones. Throughout the scanning procedure, participants were monitored with audio and video feeds.

The sensory gating task consisted of 66 pairs of 20 msec, 1 kHz tones presented 500 msec apart, from onset of the first to onset of the second, with 3 to 6 seconds between pairs. The first tone was denoted “S1” and the second tone denoted “S2.” The 66 pairs of tones were first presented monaurally to the left ear, and then again to the right ear. Each presentation of the task took approximately 10 minutes. After each presentation the participants were asked how many pairs of tones they counted.

Lastly, participants were escorted to the MRI scanner where a high-resolution three-dimensional structural image of the brain was created with axial, coronal, and sagittal images. Participants’ heads were gently restrained to reduce movement. The
The purpose of the inclusion of the MRI structural scan was to be able to visually map the triangulation of points from the MEG onto structures of interest in the brain. The structural image added to the accuracy of the points, due to individual differences in each participant’s brain structure.

The MEG procedure acquired neuronal activity across the duration of the task, and then collapsed it over the 66 trials to create a single waveform of activity, called a source series (Figure 1), for a specific region of interest in the brain. The source series shows the average activity occurring 500 msec after the onset of S1, and the overlapped average activity occurring 500 msec after the onset of S2. The left and right auditory cortices were selected for this study due to their importance in the neural pathway for auditory processing. For the right ear, the left auditory cortex was selected, and for the left ear, the right auditory cortex was selected. In order to select the source series from these specific areas of interest, a spatial filter weighed out the activity in all other areas not included in the region of interest.

**Figure 1.** Source series showing the amplitude of neuronal responses over time in milliseconds. The blue line indicates the response to S1 and the red line indicates the response to S2.
The P50 response to S1 was selected from the averaged waveform by identifying the N100 peak, which is the absolute maximum amplitude between 30 and 120 msec after the onset of S1, and then selecting the largest opposite polarity peak amplitude preceding it. The P50 response to S2 was identified in the same manner. The amplitude and latency of the P50 response to S1 and S2 was recorded. The sensory gating ratio was calculated by dividing the average P50 response amplitude to S2 by the average P50 response amplitude to S1.

Five dependent values (S1 amplitude, S1 latency, S2 amplitude, S2 latency, and S2/S1 ratio) were recorded for each participant, separately for two source series (the left ear presentation to the right auditory cortex, and the right ear presentation to the left auditory cortex), totaling ten values per participant.

**Analyses**

A mixed 2 x 2 repeated measures analysis of variance (ANOVA) with a between groups factor of diagnosis (control and PTSD) and a within participants factor of hemisphere (left hemisphere auditory cortex and right hemisphere auditory cortex) was used to examine the P50 response amplitude to S1, P50 response latency to S1, P50 response amplitude to S2, P50 response latency to S2, and S2/S1 sensory gating ratio. Age was included as a covariate due to the difference in audition and other developmental changes across the lifespan.

Two-tailed bivariate Pearson correlations were conducted between the left hemisphere response amplitude to S1 and the left hemisphere ratio; the left hemisphere response amplitude to S2 and the left hemisphere ratio; the right hemisphere response amplitude to S1 and the right hemisphere ratio; and the right hemisphere response
amplitude to S2 and the right hemisphere ratio. Two-tailed bivariate Pearson correlations were also conducted between the right hemisphere ratio and PCLM total score, and the left hemisphere ratio and PCLM total score.
RESULTS

One control participant was excluded from left hemisphere analyses because his S2/S1 ratio was an outlier in the data set, being more than ten times the average of any other S2/S1 data point.

All statistical analyses were performed with SPSS Statistical Software version 22.

Demographics

There were no significant differences of age or level of education between the PTSD and control groups.

S1 Amplitude

Results showed that there was no significant difference in P50 response amplitude to S1 between groups or across hemispheres. There was also no group x hemisphere interaction and no group x age interaction. However, means initially showed a numeric trend of slightly larger responses in the left hemisphere compared to the right hemisphere in both the control and PTSD groups (Table 1).

Table 1

Means (standard deviations) for P50 response amplitude and latency to S1 and S2 and S2/S1 ratio in both hemispheres.

<table>
<thead>
<tr>
<th></th>
<th>Amplitude (µV)</th>
<th>Latency (msec)</th>
<th>Ratio Scores</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>S1</td>
<td>S2</td>
<td>S1</td>
</tr>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P50 R</td>
<td>0.00704 (0.00349)</td>
<td>0.00519 (0.00263)</td>
<td>55.0 (12.4)</td>
</tr>
<tr>
<td>P50 L</td>
<td>0.00861 (0.00583)</td>
<td>0.00521 (0.00304)</td>
<td>52.5 (12.5)</td>
</tr>
<tr>
<td>PTSD Group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P50 R</td>
<td>0.00726 (0.00461)</td>
<td>0.00468 (0.00200)</td>
<td>53.0 (12.1)</td>
</tr>
<tr>
<td>P50 L</td>
<td>0.01090 (0.00463)</td>
<td>0.00852 (0.00282)</td>
<td>66.9 (12.5)</td>
</tr>
</tbody>
</table>
No significant differences. Results showed that there was no significant difference in P50 response latency to S1 between groups or across hemispheres. There was also no group x hemisphere interaction and no group x age interaction. However, means showed a numerical trend of slightly longer latencies in the left hemisphere compared to the right hemisphere in only the PTSD group, and almost no numerical differences in the control group (Table 1).

**S2 Amplitude**

Results showed that there was a significant group x hemisphere interaction for P50 response amplitude to S2 $F(1, 12) = 4.925, p < 0.05, \eta^2 = .291$. There were no main effects of group, hemisphere, or group x age interaction.
Upon further analysis, a one-way ANOVA showed that there was a significant difference between hemispheres in the PTSD group, $F(1, 5) = 10.492, p < 0.05, \eta^2 = 0.677$, with the left hemisphere having larger response amplitude to S2 compared to the right hemisphere (means: left = 0.00852 µV, SD = 0.00282; right = 0.00468 µV, SD = 0.0020). There were no significant differences in response amplitude to S2 between hemispheres in the control group.

Independent t-tests showed that there were group differences in response amplitude to S2 for the left hemisphere approaching significance ($p = 0.054$), with the PTSD group having larger response amplitudes to S2 compared to the control group (Table 1). There were no group differences in response amplitude to S2 for the right hemisphere.
Figure 4. Average response amplitude to S2 by hemisphere and group. Significant differences were found between hemispheres for the PTSD group.

S2 Latency

Results showed that there were no significant differences in the P50 response latency to S2 between groups or across hemispheres. There was also no group x hemisphere interaction or group x age interaction. However, means showed a numerical trend of slightly longer latencies in the left hemisphere compared to the right hemisphere in only the PTSD group, and almost no numerical differences in the control group (Table 1). This finding is congruent with the results for the P50 response latency to S1.

S2/S1 Ratio

Results showed that there were no significant differences in S2/S1 ratio between groups or across hemispheres. There was also no group x hemisphere interaction or group x age interaction. However, means for the control group showed a numerical trend for
Figure 5. Average response latency to S2 by hemisphere and group. No significant differences.

better gating in the left hemisphere compared to the right hemisphere, and means for the PTSD group showed a numerical trend for better gating in the right hemisphere compared to the left hemisphere (Table 1).

Since there were no significant differences in the S2/S1 ratio, but significant differences in the response amplitude to S2, a further analysis was conducted for the response amplitude to S1. A one-way ANOVA showed that there was a significant difference in response amplitude to S1 between hemispheres for the PTSD group, $F(1, 5) = 9.541, p < 0.05, \eta^2 = 0.656$, with larger responses in the left hemisphere compared to the right hemisphere.
Results showed a significant negative correlation between response amplitude to S1 in the right hemisphere and the S2/S1 ratio in the right hemisphere, $r(16) = -0.601, p < 0.05$. This indicates that higher S1 amplitudes are associated with lower ratios (better gating) in the right hemisphere. However, results showed no significant correlations between the response amplitude to S2 in the right hemisphere and the S2/S1 ratio in the right hemisphere. Taken together, these results show that the response amplitude to S1 is the driving force behind the ratio.

**Left Hemisphere Correlations**

Results showed a significant negative correlation between response amplitude to S1 in the left hemisphere and the S2/S1 ratio in the left hemisphere, $r(15) = 0.524, p <
0.05. This indicates that higher S1 amplitudes are associated with lower ratios (better gating) in the left hemisphere. However, results showed no significant correlations between the response amplitude to S2 in the left hemisphere and the S2/S1 ratio in the left hemisphere. Taken together, these results show that the response amplitude to S1 is the driving force behind the ratio. This finding is consistent with results in the right hemisphere.

**Symptomology Correlations**

Results showed that PCLM score and the S2/S1 ratios for the left and right hemispheres were not significantly correlated.
DISCUSSION

The results of this study suggest that there is not a significant difference in sensory gating ability between the combat veterans with PTSD compared to combat veterans without PTSD used in this study. The hypothesis that the PTSD group would have significantly larger S2/S1 ratios in both hemispheres compared to the control group was not supported. There were no group differences in S2/S1 ratio between groups or between hemispheres.

Although there were no significant group differences, both the PTSD and control groups had deficient gating seen in the very high S2/S1 ratios in both hemispheres compared to previous studies. In one study of sensory gating in PTSD patients, Metzger et al. (2002) classified his participants into “gaters,” or those with S2/S1 ratios under 0.40, and “nongaters,” or those with S2/S1 ratios over 0.40. If applying these standards to the present study, only three out of 16 participants overall would qualify as “gaters.” Rather than the PTSD group not showing the expected deficiency, the means actually showed that not only was there a deficiency in the PTSD group, but there was also a deficiency in the control group as well.

Means also suggest that there was a slight laterality difference, although it was not statistically significant. All of the participants in this study experienced paired clicks presented monaurally in the left ear first, followed by the right ear. Since there was a slight difference between hemispheres for both groups, this suggests that time may be a factor in sensory gating ability, with the control participants showing habituation and the PTSD participants showing fatigue. Numerical trends suggest that the control group benefited from this time difference, since the average S2/S1 ratio was higher in the first
presentation, denoting poorer gating, but lower in the second presentation, denoting better gating. However, numerical trends show that the PTSD group had slightly lower S2/S1 ratios during the first presentation, but higher ratios during the second presentation. A possible explanation for this is that the control group habituated after more trials, while more trials were more taxing for the PTSD group. The task may be more difficult for the PTSD group the longer in duration that it became.

There has not been any previous research where the paired-click paradigm was presented monaurally to combat veterans with PTSD. However, Gillette et al. (1997) employed three randomized blocks with different ISIs (250, 500, and 1000 msec) but did not find habituation or facilitation from the beginning to the end of the presentation.

Hunter et al. (2011) also found significant laterality differences for their PTSD patients, but it was in the opposite direction than the trend found in the present study. Hunter et al.’s results showed that the PTSD group had significantly larger S2/S1 ratios compared to the control group in the right hemisphere, but that the ratios in the left hemisphere were comparable between groups. In the present study, the larger ratios were seen in the left hemisphere for the PTSD group.

There were significant laterality effects found in the response amplitudes to S1 and S2. The hypothesis that there would be no significant group differences for the response amplitude to S1 was not supported. Although there were no group differences, analyses showed a significant laterality effect for the PTSD group, indicating that there were larger responses in the left hemisphere compared to the right hemisphere. The hypothesis that there would be significant group differences for the response amplitude to S2 was not supported. However, there was a significant laterality effect again in the left
hemisphere compared to the right hemisphere for the PTSD group, with larger responses in the left hemisphere compared to the right. This means that the PTSD group started with larger responses in the left hemisphere at S1 and maintained larger responses in the left hemisphere at S2.

The hypotheses about response latencies to S1 and S2 were supported. There were no significant differences between groups or between hemispheres for the response latency to S1 and no significant differences between groups or between hemispheres for the response latency to S2. The hypotheses about the association between response latency and amplitude were also supported. The latency and amplitude of responses were also not significantly correlated with each other, for both S1 and S2. This indicates that the size of the response and the time it takes for a response to occur are not different based on the diagnosis of PTSD. The size of the response and when it occurs are also not related.

The hypotheses about the correlations between the S2/S1 ratios and the S1 and S2 values were not supported. It was expected that the response amplitude to S2 would be the value changing the ratio. However, for both the left and right hemispheres, only the S1 values were significantly associated with the S2/S1 ratio. More specifically, since the S1 value was the denominator, the S1 values were negatively driving the ratio, so the larger the amplitude of the response to S1, the smaller the ratio.

The hypotheses about the correlation between symptoms and S2/S1 ratios were not supported. Symptom severity and S2/S1 ratio were not significantly correlated with each other. Previous studies of sensory gating in PTSD patients have found varying results. Although Gillette et al. (1997) and Holstein et al. (2010) found positive
correlations with symptomology and S2/S1 ratios, their results were slightly different. Gillette et al. found that the S2/S1 ratio was significantly positively associated with the CAPS score for re-experiencing symptoms, meaning that higher ratios were associated with worse re-experiencing symptoms. Holstein et al. found that the S2/S1 ratio was significantly negatively associated with SCL-90-R indices, meaning that higher ratios were associated with more psychopathological symptoms. Studies need to further investigate the relationship of PTSD symptoms and sensory gating performance variables.

There are several ways of interpreting these results. If the recordings of the responses in each individual are accurate, then it could mean that the combat veterans with PTSD are more similar to the combat veterans without PTSD than was expected for this study. It could be that the PTSD patients do not actually have the traumatic stress that is being represented by the PCLM, or that the control group has a significant level of traumatic stress and does not display symptoms, or does not report the symptoms. Although the PCLM results showed that the PTSD group scored in the high level category and the control group scored in the low level, many control participants still had some symptoms of PTSD without the formal diagnosis of the disorder.

This could also possibly reveal that combat veterans without a formal diagnosis of PTSD still have sensory gating deficiencies. Both combat veterans with PTSD and combat veterans without PTSD have experienced severe trauma, so it’s possible that the sensory gating deficiency is coming from somewhere other than the disorder. Although previous studies have produced results showing that control groups without PTSD have adequate sensory gating (Ghisolfi et al., 2004; Gillette et al., 1997; Holstein et al., 2010;
Hunter et al., 2011; Metzger et al., 2002; Neylan et al., 1999; Skinner et al., 1999), this study solely included veterans from OEF/OIF, so the circumstances of their combat experience cannot necessarily be equated with that of other veterans, because while there are multiple decades of research that has led to an understanding of PTSD from the Vietnam war, etc., research on veterans from the OEF/OIF is much newer. It is also difficult to make generalizations about veterans with PTSD and patients with PTSD related to other traumatic events altogether. As discussed previously, hypervigilance to a complex combat environment could have been a development that was beneficial for a certain set of circumstances, and the issue is upon return to a safe environment. For patients with PTSD related to another traumatic event, like abuse, this is probably not the case, so sensory gating research may be examining different things between these populations. Stander et al. (2014) has stated that, “The military is a unique context,” and that, “Unique stressor events are likely to have unique predictors and adjustment outcomes” and it is important to examine PTSD patients keeping this in mind (p. 94).

It is also possible that the timing of the paired-click paradigm was inadequate to measure the group differences that were expected. Studies have previously employed multiple levels of intertrial intervals in one study, and results seem to suggest that gating is best examined at shorter rather than longer ITIs. Skinner et al. (1999) found significant group differences of S2/S1 ratio between the PTSD group and control group when using a 250 msec ITI, but no differences between groups at ITIs of 500 and 1000 msec. Gillette et al. (1997) found no group differences of average S2/S1 ratios between 250 and 500 msec, but they do report more gating at shorter ITIs. While it must be acknowledged that other studies were able to find group differences using a 500 msec ITI, it is possible that
changing the ITI for this study would have shown more gating across participants. It should also be noted that the duration of the stimuli in the present study were 20 msec, whereas the duration of the stimuli in the previously discussed research ranged from 1 to 3 msec. Aspects of the stimuli may also play a part in the gating mechanism.

There are several limitations to the present study. The hearing threshold test used to determine decibel level for the presentation of clicks was not uniform for all participants. While it is typical for the sound level to be a set number of decibels above hearing threshold to eliminate hearing ability differences, this did not occur in the present study. However, using age as a covariate produced no significant effects of age, so confidence that hearing ability across the lifespan was not a major factor is high.

While the number of participants included in this study was comparable to other studies employing PTSD patients, a larger sample size would have been beneficial for this study. Since symptoms of PTSD, as with any disorder, can vary a great deal across patients, it is necessary to have a representative sample before forming conclusions about the entire population of interest. It is suggested that doubling the sample size would be beneficial for any subsequent replications.

It would have also been beneficial to record more exact sequence variables for each participant for their deployment, return, diagnosis of PTSD, and duration of PTSD. The only time variable that was known for these participants was that they served in OEF/OIF, which could have been any time since October 2001 to present, and that they had since returned and were subsequently diagnosed with PTSD. It would be valuable to know how long participants were home from service before they began to have symptoms emerge, the amount of time between when they first started experiencing
symptoms and the time that they were diagnosed with PTSD, and the amount of time since their diagnosis. The “honeymoon phase” is a well-known phenomenon where veterans returning home post-deployment have suppressed perceptions and demonstration of PTSD symptoms due to expectations of what life will be like once returning to a normal life (Stander et al., 2014). Generally, PTSD symptoms are delayed about six months post-deployment due to this effect. One possible explanation for the control participants’ unexpected high S2/S1 ratios in the present study is that if they, in fact, do have PTSD, the more obvious symptoms are suppressed but their physiological reactions are already abnormal.

Although the PCLM is a common way of quantifying symptoms in the diagnosis of PTSD, another survey that may have added detail to the demographic picture of the patients in this study is the addition of CAPS information. The PCLM asks patients how often they are bothered by the following symptoms in the past month only, using a five-point Likert scale where 1 means, “Not at all” and 5 means, “Extremely.” This type of survey quantifies symptom severity by asking how often this bothers the patient, but variables such as how often these symptoms occur, how long they last, and their intensity are other factors that should be considered. For instance, asking a patient with PTSD how often they were bothered by disturbing dreams of a stressful military experience from the past, and receiving the answer that it bothered them a moderate amount (3 out of 5), could mean that the patient is moderately bothered by one dream per night or one dream every week. The CAPS survey is more thorough, asking about the frequency and intensity of symptoms for the past month, week, and across the lifetime. For instance, one question asks whether the patient has ever had unpleasant dreams about the traumatic
experience, what happens in the dream, how much distress it causes the patient, if the patient wakes up, and how long it takes to go back to sleep. Although brevity is important when conducting research so as to not fatigue the participants, other research that has included PTSD patients has used the CAPS survey, and this information could be valuable if the present study were to be repeated. The assumption that the sensory gating paradigm is being employed to understand more about hyperarousal symptoms in PTSD patients can’t be taken for granted; more correlational work needs to be conducted to see how symptom severity is associated with sensory gating deficits. The present study did not find a significant correlation between sensory gating ability and symptom severity, but perhaps with different, more thorough measures, this can be examined more closely.

Another aspect of PTSD that must be considered is that it has a high comorbidity rate with depression, anxiety, panic disorder, conduct disorder, personality disorder, and substance abuse (Stander et al., 2014). There is an abundance of research on PTSD and depression, since these occur most frequently together. Several hypotheses exist about the nature of their co-occurrence, the most supported being that PTSD causes depression and insomnia, and that PTSD and depression share common risk factors and vulnerabilities. Although patients in the current study were screened to have no other mental health disorders, Najavits et al. (2009) has found that depression is so prevalent that it is almost impossible to exclude it from the sample of interest in clinical research. The relationship of depression and sensory gating ability is not well known. However, it is important to consider that this disorder may play into sensory gating results.

Two studies have focused on sensory gating deficits in patients with depression. Jiang et al. (2006) studied the sensory gating abilities of patients with first-episode
depression using the paired-click paradigm. Results showed that, similar to other psychiatric disorders with inhibition problems, the depression patients had significantly larger S2/S1 ratios compared to controls, suggesting a sensory gating deficit. However, symptom severity was not significantly correlated with sensory gating performance. In a follow-up study, Wang et al. (2009) studied patients with treatment-resistant depression and non-treatment-resistant depression with the same paired-click paradigm and found that both groups had sensory gating deficiencies, but that the treatment-resistant depression patients had more severe deficits. In contrast to Jiang et al., results showed a significant positive correlation of the 17-item Hamilton Rating Scale for Depression scores and the S2/S1 ratio in the treatment-resistant depression patients.

In conclusion, not only did the PTSD patients in the present study exhibit poor sensory gating (large S2/S1 ratios), but the control group also exhibited poor sensory gating. If it is assumed that the paired-click paradigm is adequately representing the ability to inhibit redundant stimuli in the environment, then these results are suggesting that there are similarities between the combat veterans with PTSD and combat veterans without PTSD that is not apparent at this time. Possible explanations could be that the control group is not experiencing typical symptoms, not reporting symptoms, the diagnostic tools being used are not capturing the symptoms, or the symptoms have yet to fully develop. It is also possible that the PTSD group does not have the level of traumatic stress that they are reporting, although this is less likely because the S2/S1 ratios between groups would be smaller. It is also possible that PTSD is not associated with sensory gating deficiencies and there is something about the combat exposure itself that is causing the deficiencies that is not understood yet. Another possible explanation is that
the performance of veterans from OEF/OIF cannot be equated with other veterans’
performance from other wars because the nature of their service is different and only
recently studied. It also always must be questioned if the sample is representative of the
population at hand and if there are any comorbidities that could be present in the sample
that may affect results. If it is not assumed that the paired-click paradigm is adequately
measuring sensory gating differences, then there are several other types of questions to
consider. The specifics in this study did not match to other previously discussed studies,
most notably in the duration of the stimuli and the possibility that the ISI was too long.
Future directions for this study must take these possibilities into serious consideration.

The laterality differences found in this study should also be taken into
consideration. Only the PTSD group showed larger responses to S1 and S2 in the left
hemisphere, which could indicate structural or developmental differences. This study
employed MEG technology, whereas it is more common to use EEG technology in
sensory gating research. An advantage in this study is that it is possible to examine
specific regions of interest in the brain with MEG technology, while this is either not
possible or more heavily distorted with other technologies.

PTSD research of combat veterans is only now coming to the forefront. Hopefully
more studies on a similar topic to this one will eventually lead to a better understanding
of symptoms, biological markers of vulnerabilities for this disorder, and improved
treatment.
REFERENCES


APPENDIX

<table>
<thead>
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<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
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<tbody>
<tr>
<td>1</td>
<td>Not at all</td>
<td>A little bit</td>
<td>Moderately</td>
<td>Quite a bit</td>
<td>Extremely</td>
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</tbody>
</table>

**Instructions:** Below is a list of problems and complaints that veterans sometimes have in response to stressful life experiences. Please read each one carefully, mark the answer to indicate how much you have been bothered by that problem *in the last month.*

1. Repeated, disturbing memories, thoughts, or images of a stressful military experience from the past?
   - 1 2 3 4 5

2. Repeated, disturbing dreams of a stressful military experience from the past?
   - 1 2 3 4 5

3. Suddenly acting or feeling as if a stressful military experience were happening again (as if you were reliving it)?
   - 1 2 3 4 5

4. Feeling very upset when something reminded you of a stressful military experience from the past?
   - 1 2 3 4 5

5. Having physical reactions (e.g. heart pounding, trouble breathing, or sweating) when something reminded you of a stressful military experience from the past?
   - 1 2 3 4 5

6. Avoiding thinking or talking about a stressful military experience from the past or avoid having feelings related to it?
   - 1 2 3 4 5

7. Avoid activities or situations because they remind you of a stressful military experience from the past?
   - 1 2 3 4 5

8. Trouble remembering important parts of a stressful military experience from the past?
   - 1 2 3 4 5

9. Loss of interest in things that you used to enjoy?
   - 1 2 3 4 5
10. Feeling distant or cut off from other people?
   1  2  3  4  5

11. Feeling emotionally numb or being unable to have loving feelings for those close to you?
   1  2  3  4  5

12. Feeling as if you future will somehow be cut short?
   1  2  3  4  5

13. Trouble falling or staying asleep?
   1  2  3  4  5

14. Feeling irritable or having angry outbursts?
   1  2  3  4  5

15. Having difficulty concentrating?
   1  2  3  4  5

16. Being “super alert” or watchful on guard?
   1  2  3  4  5

17. Feeling jumpy or easily startled?
   1  2  3  4  5
After a head injury or accident some people experience symptoms that can cause worry or nuisance. We would like to know if you now suffer any of the symptoms below. Because many of these symptoms occur normally, we would like you to compare yourself now with before the accident. For each symptom listed below please circle the number that most closely represents your answer.

0 = not experienced at all
1 = no more of a problem
2 = a mild problem
3 = a moderate problem
4 = a severe problem

<table>
<thead>
<tr>
<th>Symptom</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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<tr>
<td>Headaches</td>
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<td>Feelings of dizziness</td>
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<tr>
<td>Nausea and/or vomiting</td>
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<td>Noise sensitivity (easily upset by loud noise)</td>
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<td>Sleep disturbances</td>
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<td>Fatigue, tiring more easily</td>
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<td>Being irritable, easily angered</td>
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<td>Feeling depressed or tearful</td>
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<td>Feeling frustrated or impatient</td>
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<td>Forgetfulness, poor memory</td>
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<td>Poor concentration</td>
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<td>Taking longer to think</td>
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<td>Blurred vision</td>
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<td>Light sensitivity (easily upset by bright light)</td>
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<tr>
<td>Double vision</td>
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<td>Restlessness</td>
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Are you experiencing any other difficulties? Please specify, and rate as above.

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<tr>
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CURRICULUM VITAE

ELIZABETH T. COMITZ

BORN: October 25, 1989 in Alexandria, Virginia

UNDERGRADUATE STUDY: James Madison University
B.A., Psychology
B.A., Interdisciplinary Philosophy
August 2008 – May 2012

GRADUATE STUDY: Wake Forest University
M.A., Psychology
August 2012 – Present

SCHOLASTIC AND PROFESSIONAL EXPERIENCE:

Research Assistant, Wake Forest Baptist Health Medical Center Neurobiology and Anatomy Lab
June 2013 – Present

Research Assistant, Wake Forest University Psychophysiology Lab
August 2012 – Present

Research Assistant, James Madison University Sleep and Actigraphy Lab
August 2011 – May 2012

Research Assistant, James Madison University Cognition and Critical Thinking Lab
August 2011 – May 2012

Teaching Assistant, James Madison University

Introduction to Philosophy
Ancient Greek Philosophy
August 2011 – January 2012

Campus Campaign Coordinator, Teach For America
August 2010- April 2011

VOLUNTEER EXPERIENCE:

Volunteer, Wake Forest Baptist Health Medical Center, Adult Inpatient Psychiatric Unit
March 2014 – Present
Volunteer, World Food Program  
April 2007 – May 2009

PROFESSIONAL SOCIETIES:

Member, Society for Psychophysiological Research  
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President, National Society of Collegiate Scholars, James Madison University  
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Wake Forest University Graduate Scholarship, 2012-2013

Phi Beta Kappa Scholar, 2011

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PUBLICATIONS AND PRESENTATIONS:


