SENIOR HONORS THESIS: ANALYSIS OF BRAIN STRUCTURE IN A COMMUNITY SAMPLE OF WOMEN WITH POSTTRAUMATIC STRESS DISORDER AS A RESULT OF CHILD ABUSE EXPOSURE

LISA MARTORANO

Department of Biology, Wittenberg University, Springfield, OH 45501-0720 USA

In conjunction with Dr. Cathy Pederson, Dr. Stephanie Little and Dr. Robin Osborn D.O.
Abstract

The long term effects of child abuse can deter brain development and function in adult abuse survivors. This study attempts to make a positive correlation between participants with post-traumatic stress disorder (PTSD) secondary to child abuse and reduced volumes in the hippocampus, pituitary, and caudate nucleus. Participants were recruited through newspaper advertisement and were right handed females between 20 and 40 years of age. Women who matched the study criteria, based on phone interviews, were screened using a demographics questionnaire and a variety of psychological testing including the Childhood Trauma Questionnaire and Millon Multiaxial Clinician Inventory. Those accepted into the study took the Weschler Memory Scale, Wonderlic Personnel Test, Clinician Administered PTSD Scale, and a magnetic resonance image of their brain. Women were then placed into one of three groups: post traumatic stress disorder as a result of child abuse (n=21), child abuse without PTSD (n=18), and normal controls (n=21). Each MRI slice of a brain structure was traced three times using the 3DBrainStation. Averages were calculated and summed to determine total volume of each structure. Demographic matching between groups showed no differences in age, body mass index, education, alcoholic drinks per year, and pack years smoking (p>0.05). There was no significant difference between the groups in hippocampal (p=0.426 left, 0.547 right), pituitary (p=0.273) and caudate nucleus (p=0.622 left, 0.959 right) volumes. Furthermore, PTSD diagnosis did not influence structural volume. The results show that child abuse may not be a detrimental factor in altering brain structural development in a community sample of women with posttraumatic stress disorder.

Introduction

Child Abuse

Child abuse is a current and ongoing national epidemic. According to the Child Welfare Information Gateway (2007), child abuse may be defined as those actions which “result in death, serious physical or emotional harm, sexual abuse, or exploitation, or an act or failure to act which presents an imminent risk of serious harm.” Current statistics gathered from the National Child Abuse and Neglect Data System’s (NCANDS) comprehensive report, Child Maltreatment (2005), show the nationwide impact of child abuse. In 2005, 899,000 cases of child abuse and neglect were reported amongst the 50 states, Puerto Rico and the District of Columbia with children ranging from birth to three years of age being the most at-risk group for victimization. Child maltreatments types include: neglect (62.8%), physical abuse (16.6%), sexual abuse (9.3%) and emotional abuse (7.1%) (NCANDS 2005). Specifically, HIV-positive women have
been shown to comprise a large group of former victims of childhood sexual abuse (Myers et al. 2006). Further statistics show that white children account for 49.7% of abuse cases followed by African American (23.1%) and Hispanic children (17.4%). 50.7% of the victims were girls while the remaining 47.3% were boys (NCANDS 2005). The figures were based on reported investigations from Child Protection Services; thus, they do not accurately account for all nationwide instances of child abuse.

The impact of child abuse can be profound. In a community based study, Vranceanu et al. (2007) analyzed 100 women who presented to an inner-city women's health center and found that women who had been exposed to childhood maltreatment were less likely to have a social support network and more likely to experience stress in adulthood. Similarly, McCauley et al. (1997) showed that compared to adult women who never experienced child abuse, child abuse victims were more likely to experience depression, anxiety, somatization, interpersonal sensitivity, abuse drugs and alcohol, and also suffer from suicide attempts. In another study, a survey was given to 205 female patients at a primary care practice, and the results showed that those who had experienced emotional abuse and neglect experienced more depression, anxiety and somatization symptoms than controls (Spertus et al. 2003). Thus, the damage of child abuse can sustain itself through adulthood. Lang et al. (2006) conducted a self-report survey involving 222 adult female veterans about current health status and childhood maltreatment experiences. It was found that victims of emotional abuse experienced a decrease in physical functioning, increased bodily pain, and an increase in pain medication usage. Physical abuse, on the other hand, was correlated with overall poor health (Lang et al. 2006). These long term effects can accumulate and result in the development of post traumatic stress disorder.
Posttraumatic Stress Disorder

On a larger scale, traumatic stress can lead to other conditions including posttraumatic stress disorder (PTSD). PTSD is an anxiety disorder which may be accommodated by neural changes in the frontal and limbic systems of the brain (Francati et al. 2007). According to the National Institute of Mental Health (2007), PTSD can develop in response to a terrifying or stressful event in which the victim experiences severe threats and/or physical damage. Fear is the main emotion aroused by PTSD and it can also stimulate activation of the hypothalamic-pituitary-adrenal axis (Nutt & Malizia 2004). PTSD symptoms include: persistent frightening thoughts or memories, flashbacks, hypersensitivity, avoidance of traumatic stimuli or social gatherings, feeling emotionally numb, and sleep difficulties (Francati et al. 2007). For victims of childhood sexual abuse, PTSD symptoms were more pronounced if the victim experienced intrafamilial abuse in comparison to being abused an extrafamilial perpetrator; further re-victimization as an adult led to a positive correlation in more PTSD-related symptoms including sexual problems (Myers et al. 2006). More specific systemic effects of abuse-related PTSD involve decreased cerebrospinal fluid (Rasmusson et al. 2006), altered thyroid activity (Friedman et al. 2005), and a reduced volume of the anterior cingulate cortex as compared with controls (Kitayama et al. 2006). In addition, PTSD can lead to dysfunction of the hippocampus which is implicated in the memory response (Bremner et al. 2003).

However, other research yields contradictory results. Twamley et al. (2004) compared the three groups of college students: PTSD and trauma, trauma only, and no-trauma in terms of neuropsychological functioning such as word production and execution, working memory, and mental aptitude. Apparently, there were no significant differences among the groups in any of the testing areas; this demonstrates that PTSD may not predispose the brain to
neuropsychological impairment (Twamley et al. 2004). Yet, when introducing alcohol as a variable in PTSD veterans, there was a significant decrease in verbal and visual memory, attention, and processing speed (Samelson et al. 2006).

In 1995, Kessler et al. compiled a National Comorbidity Survey based on the responses of 5877 people nationwide aged 15 to 54, finding that the prevalence of PTSD is 7.8%. On average, women are less likely to experience traumatic events than men (Tolin and Foa 2006), but have a higher incidence of PTSD (Tolin and Foa 2006). Such gender differences in relation to PTSD are seen in regional brain volumes and behavioral mechanisms for coping with PTSD (Nemeroff et al. 2005; Tolin and Foa 2006; Walker et al. 2004). Ullman and Filipas (2005) analyzed a community sample of college male and female college students regarding their sexual abuse history and coping strategies. The females reported greater incidence of sexual abuse and PTSD severity compared to males, and also experienced more distress in relation to the assault. It is interesting that the females were less likely to disclose abuse incidents, thereby internalizing their traumatic experience which could lead to the development of PTSD (Ullman and Filipas 2005). Other events that may lead to PTSD include natural disasters and combat exposure for men and rape and molestation for women (Kessler et al. 1995).

In studies with adults and children, PTSD has shown to result in various consequences to the body. De Bellis et al. (1999) compared maltreated children with PTSD to healthy controls. The maltreated group had greater levels of urinary free cortisol and catecholamines than control participants, thus correlating with severity and duration of PTSD. Also, the maltreated children with PTSD had smaller intracranial and cerebral volumes than matched controls; however, no significant differences in the hippocampus were found (De Bellis et al. 1999). Contrary, an adult study containing PTSD subjects and controls found no significant difference in intracranial...
volume, but showed a reduction in white matter/intracranial volume ratio, suggesting white matter atrophy (Villarreal et al. 2002).

As mentioned before, fear and the associated bodily response are important elements of PTSD. To investigate the effects of fear on salivary cortisol levels, Elzinga et al. (2003) exposed female participants to personalized trauma scripts. The study compared female child abuse victims with PTSD and without PTSD, showing that those with PTSD had higher levels of salivary cortisol levels during and after exposure to the trauma script. PTSD participants also had 122% higher cortisol levels than their non-PTSD counterparts (Elzinga et al. 2003). This stress response was also seen in other studies. Bremner et al. (2003) recruited adult male and female participants in addition to healthy controls to measure their cortisol levels when exposed to a cognitive challenge. The PTSD participants had a 61% greater surge in cortisol levels in anticipation of a cognitive challenge, and 46% increase during the challenge as compared to the control group (Bremner et al. 2003). Gender also plays a role in the cortical response for male PTSD participants demonstrated higher cortisol levels during the study than their female PTSD counterparts, which is interesting given that females are more likely to be affected by PTSD (Bremner et al. 2003).

Yet, when analyzing the following groups: PTSD and childhood sexual abuse, sexual abuse only, and controls Bremner et al. (2003) observed that women with PTSD-related childhood sexual abuse exerted lower levels of cortisol in the afternoon hours (12:00-8:00 pm) than matched controls. Bremner et al. (2003) also found that both PTSD and abuse and abuse only groups had a blunted adrenocorticotropin response to corticotropin-releasing factor. This points to the long term effects of PTSD on the brain, suggesting PTSD is associated with hypocortisolemia in the afternoon hours and a blunted response to adrenocorticotropin and
corticotropin-releasing factor. What accounts for the development of conditions like PTSD and the physiological effects of stress? This question can be examined more closely when looking at the brain itself.

**Stress & its Biochemical Impact on the Brain**

Child abuse may produce stress on the brain. One of the brain’s major stress pathways includes the hypothalamic-pituitary-adrenal (HPA) axis (De Bellis et al. 1999). When exposed to stress, the hypothalamus stimulates the release of corticotropin-releasing factor (CRF) which stimulates the release of adrenocorticotropin (ACTH) from the anterior pituitary gland. Notably, subjects who have experienced early life trauma tend to have increased levels of corticotropin-releasing factor (Nemeroff et al. 2005). This cascade then causes the adrenal cortex to release glucocorticoids to various regions of the brain, promoting behavioral responses to stress (De Bellis et al. 1999). Glucocorticoids are often known to exacerbate the stress response and have been shown to inhibit glucose uptake in the brain, leading to decreased energy stores for neurons to complete normal function and repair over prolonged episodes of stress (Sorrells and Sapolsky 2006). Although glucocorticoids can have pro-inflammatory effects, by and large they still remain anti-inflammatory in the periphery; thereby inducing negative effects for those who experience periods of traumatic stress (Sorrells and Sapolsky 2006). Upon glucocorticoid secretion, homeostasis is eventually reached through negative feedback inhibition by the hypothalamus, pituitary and hippocampus (De Bellis et al. 1999). For instance, Sapolsky et al. (1984) demonstrated that when glucocorticoid receptors were reduced in aged rats and Battelboro rats, increased levels of the glucocorticoid, corticosterone, correlated with a termination of the
stress response. These results further attest to the effects of hippocampal plasticity in response to glucocorticoid secretion.

Several studies have demonstrated the impact of the HPA axis in response to PTSD. Otte et al. (2007) administrated the cortical inhibitor, metyrapone, for women with PTSD to observe the effect on the HPA axis. It was shown that the HPA response to metyrapone was diminished in women with PTSD. This provides further support that when corticotropin-releasing factor activity is high, downregulation of the CRF receptors follows (Otte et al. 2007). A similar result was found when comparing PTSD-related comorbid dissociative disorder to healthy controls. It was demonstrated that this subgroup had a blunted stress response compared to healthy controls (Simeon et al. 2007). Once again this study provides evidence of a downregulation of the HPA axis in response to long term stress and associated stress disorders.

Hippocampus

Stress can likewise influence the development of the hippocampus which is implicated in learning and memory. It affects the hippocampus in numerous ways which include impairing hippocampal plasticity, facilitating neuron death, and halting the process of neurogenesis (Sapolsky 2003). Once the stress cascade begins, both the amygdala and hippocampus may be a target for glucocorticoid activation. Specifically, the hippocampus contains an increased number of glucocorticoid receptors (Sapolsky et al. 1984). A structure located within the hippocampus known as the dentate gyrus develops in the postnatal period and plays a role in granular cell proliferation (Gould and Tanapat 1999). Furthermore, this structure continues to develop into adulthood and may be sensitive to environmental changes. Thus, stressful events induce the
release of adrenal steroids which can in turn inhibit cellular proliferation in the dentate gyrus, leading to an altered structure of the hippocampus (Gould and Tanapat 1999).

Animal studies show the consequences of this complex stress system. As the stress cascade begins, Gould et al. (1997) found that there is a decrease in the number of proliferating granular cells in adult marmoset monkeys. Tanapat et al. (1998) further showed that upon exposure to male rat odor, rat pups experienced an increase in corticosterone levels, which was inversely correlated with H-thymidine labeled cells in the developing dentate gyrus. Similarly, stress at early ages can exacerbate hippocampal damage. Early administration of corticotropin-releasing factor may result in a loss of hippocampal CA3 neurons and impair memory function (Brunson et al. 2001). Long term glucocorticoid exposure leads to similar findings. Sapolsky et al. (1990) demonstrated that in young adult male vervet monkeys, year-long administration of glucocorticoids resulted in hippocampal damage to the CA2 and CA3 neurons such as dendritic atrophy, soma shrinkage, nuclear pyknosis, and cell layer irregularity. This observation can be attributed to the fact the glucocorticoids may increase the vulnerability of hippocampal neurons to damage through an unspecified catabolic mechanism, thus resulting in hippocampal toxicity (Sapolsky 1985). In cynomolgus monkeys, it appears that stress can result in the downregulation of glucocorticoid receptors in the hippocampus, which may further impair its ability to inhibit the HPA axis (Sapolsky et al. 1991).

It is evident that the hippocampus is prone to damage induced by stressful events. In a gender neutral study, 37 subjects who had experienced a traumatic event were assessed twice using Magnetic Resonance Imaging (Bonne et al. 2001). Participants were assessed one week and six months after the event. There was no significant difference between hippocampal volume at either time intervals for both of the groups (Bonne et al. 2001). However, this is not
always the case in more chronic cases of PTSD. A meta-analysis consisting of nine studies analyzed the following groups: adult subjects with chronic PTSD, healthy controls, and traumatized controls, thereby finding a significant reduction in hippocampal volume in the chronic PTSD group compared to the other two groups (Kitayama et al. 2005). This trend can also be seen in children. Maltreated children with a high severity of PTSD and increased cortisol levels were found to have a reduction in their hippocampal volume (Carrion et al. 2007).

More specific research involving child abuse also yielded similar results. Bremner et al. (1997) recruited adult survivors of child abuse diagnosed with PTSD and matched controls. The PTSD group had a 12% reduction in left hippocampal volume than the controls (Bremner et al. 1997). Twenty one adult females who had self-identified instances of child abuse also had a 5% reduction in left hippocampal volume, but no significant difference could be found for the right hippocampus (Stein et al. 1997). In a community sample, women with childhood sexual abuse and PTSD had a 19% smaller hippocampal volume than women without abuse or PTSD, and a 16% smaller hippocampal volume than abused women without PTSD (Bremner et al. 2003). However, in another community based study, no significant difference between the PTSD group and abuse and control groups in hippocampal volume were found (Pederson et al. 2004).

**Pituitary Gland**

Located at the base of the brain, the pituitary gland is primarily responsible for controlling hormonal functions in the body. But as part of the HPA axis, the pituitary gland may also be adversely affected by stress. A pediatric study (Thomas and De Bellis 2004) showed that for a group of children aged 4 to 17 years, those who had maltreated PTSD showed no significant difference in pituitary volume compared to healthy controls. However, when
narrowing the age groups, there were significant differences in pituitary volume with age for the PTSD group as compared to controls. Larger pituitary volumes were seen in the pubertal/postpubertal maltreated PTSD subjects compared to controls (Thomas & De Bellis 2004). Additionally, adult women with a history of child abuse displayed above normal adrenocorticotropin response when corticotropin-releasing factor was administered, thereby showing how stress can sensitize the anterior pituitary to CRF (Heim et al. 2001).

**Caudate Nucleus**

The caudate nucleus is located within the basal ganglia and plays a role in regulating and filtering information (HOPES 2007). Yet, not much research is available showing damage to the caudate nucleus as a result of early life trauma. One study consisting of 265 healthy Australian men and women aged 18 to 70 showed that participants who had experienced more than two early traumatic life experiences experienced a reduction in the caudate nuclei when compared with those who had never experienced early traumatic experiences (Cohen et al. 2006). Bremner et al. (1997) found no significant difference in caudate nuclei volumes in adult survivors of childhood sexual abuse as compared to controls.

**Purpose of this study**

Based on previous research regarding the nature of stress and child abuse, it is imperative to study the long term effects of child abuse. The purpose of this research is to analyze the volumetric effects of child abuse on several areas of the brain including the hippocampus, pituitary gland and caudate nucleus. Furthermore, magnetic resonance imaging will help determine whether a significant difference in structure volume exists between the following
groups: PTSD and child abuse, child abuse without development of PTSD, and matched controls.

Methods

Participants

Sixty right-handed females, ages 20-40, were selected and placed into one of the following groups: child abuse with development of PTSD (N=21), child abuse without development of PTSD (N=18), and normal controls which had no history of abuse or PTSD (N=21). Participants in each group were matched based on demographic factors such as age, education level, alcohol consumption, nicotine use, and illicit drugs. However, participants were disqualified if they had a learning disability, psychological disorder, attention deficit disorder, metal implant, pregnancy, current drug use, or remained unconscious for longer than 5 minutes (Pederson et al. 2004).

Procedure

Participants were recruited through a newspaper advertisement in the *Springfield News Sun*. A telephone questionnaire was implemented to assess participants’ medical history including body mass index, current drug and alcohol consumption, and prescription usage as well as childhood history including family divorce rate, number of times one moved, and abuse history. Participants were disqualified if they were pregnant, had metal implantation or a pacemaker, a learning disability, a psychological disorder, and if they were current illegal drug users. If participants met the initial qualifications, they were invited to Wittenberg to sign an informed consent form which outlined their role in the study and discussed confidentiality. Next, they independently took various psychological tests, including: demographic questionnaire,
Childhood Trauma Questionnaire (CTQ; Bernstein & Fink 1988), Trauma Symptom Inventory (TSI; Briere 1995) and Millon Clinical Multiaxial Inventory—Third Edition (MCMI-III; Millon, Millon, & Davis 1994). To be considered for the normal control group, participants had to score T < 85 on all subscales of the MCMI-III. Control participants must have also scored a maximum 8 on the CTQ emotional abuse scale, 7 on the physical abuse scale, and 5 on the sexual abuse scale. Participants in the PTSD or abuse group were required to score in the Severe to Extreme category on the CTQ with minimum scores of 16 on the emotional abuse scale, 13 on the physical abuse scale, and 13 on the sexual abuse scale. Qualified participants further completed the Wechsler Memory Scale—3rd Edition (WMS; Wechsler 1997) and the Wonderlic Personnel Test (Wonderlic 1988). Participants were then interviewed by a psychologist using the Clinician Administered PTSD Scale (CAPS; Blake et al. 1988) for placement into one of the three groups: PTSD and child abuse, child abuse only, and control. In order to be placed into the PTSD group, participants met Criterion A of the DSM-IV-TR (American Psychiatric Association 2000) PTSD definition (Pederson et al. 2004).

Magnetic Resonance Imaging & Tracings

Each participant received a magnetic resonance imaging (MRI) of the brain on a 1.0 Tesla Siemens Magnatom (Vision) at Crystal Clear Imaging, Ltd. or 1.5 Tesla Siemens Magnatom at Mercy Medical Center (Springfield, Ohio). Participants consumed two or less alcoholic drinks daily three weeks prior to the MRI to minimize alcohol’s effect on the brain (Agartz et al. 1999). All tracings were performed on the 3D BrainStation. Tracings of the right and left hippocampus were performed using a sagittal cross-section of the brain. The pituitary gland was traced using a coronal cross-section, while the left and right caudate nuclei were
traced using a transaxial cross-section. The slice thickness was 1 mm in all cases. The average areas of each slice were then summed to get total brain structure volume. It is important to note that the total volume of the caudate nucleus was multiplied by two to account for every other slice measurement. Lastly, each participant received monetary compensation for their time at a rate of $10-15 per hour. The overall procedure received approval from Wittenberg’s Institutional Review Board.

Data Analysis

SPSS for Windows version 15.0 (SPSS Inc., 2006) was used for all statistical tests. Demographic characteristics, CTQ abuse scores, total hippocampal volume, total pituitary volume, and total caudate nuclei volume were assessed with Univariate analyses of variance (ANOVA). T-tests were further implemented to detect mean differences among the three groups.

Results

Tracings

Sample tracings of each structure are shown below. For each structure, two views are shown with the structure highlighted by the red arrow. The images are from a single participant and do not represent a uniform version of the structure as it varies by individual.
Figure 1. A sagittal MRI image cross-section of the brain representing lateral (left) and medial (right) views of the hippocampus (red arrow).

Figure 2. A coronal MRI image cross-section of the brain representing anterior (left) and posterior (right) views of the pituitary gland (red arrow).
Demographic Characteristics

First, demographic characteristics for the sixty participants are reported in Table 1. There are no significant differences among the three groups in terms of demographics ($p > 0.05$), thereby showing that all three groups are well-matched. The demographics are as follows: age ($F(2,59) = 1.302, p = 0.280$), body mass index ($F(2,59) = 2.977, p = 0.059$), years of education ($F(2,59) = 1.649, p = 0.201$), drinks/year ($F(2,59) = 1.298, p = 0.281$), pack years ($F(2,59) = 0.528, p = 0.592$), and the Wonderlic Personnel Test ($F(2,59) = 1.919, p = 0.156$).

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>ABUSE</th>
<th>CONTROL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\bar{x}$</td>
<td>SD</td>
<td>$\bar{x}$</td>
</tr>
<tr>
<td>Age</td>
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<td>5.2</td>
<td>26.6</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>27.1</td>
<td>6.0</td>
<td>27.3</td>
</tr>
<tr>
<td>Years of Education</td>
<td>14.0</td>
<td>2.0</td>
<td>14.6</td>
</tr>
</tbody>
</table>
When introducing the variable of abuse, there were significant differences among the three groups in terms of the severity of abuse experienced \((p < 0.001)\). The results of the Childhood Trauma Questionnaire (Bernstein & Fink 1988) are presented in Table 2 with emotional abuse \((F(2,59) = 49.815, p = 0.000)\), physical abuse \((F(2,59) = 17.156, p = 0.000)\), and sexual abuse \((F(2,59) = 11.142, p = 0.000)\).

### Table 2. Type of Abuse (CTQ)

<table>
<thead>
<tr>
<th>Type of Abuse</th>
<th>PTSD</th>
<th>ABUSE</th>
<th>CONTROL</th>
</tr>
</thead>
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<td></td>
<td>(\bar{x})</td>
<td>SD</td>
<td>(\bar{x})</td>
</tr>
<tr>
<td>Emotional Abuse</td>
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<td>4</td>
<td>15</td>
</tr>
<tr>
<td>Physical Abuse</td>
<td>13</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>Sexual Abuse</td>
<td>13</td>
<td>8</td>
<td>15</td>
</tr>
</tbody>
</table>

In this study, the hippocampus, pituitary gland and caudate nucleus were compared amongst the following groups: PTSD and abuse, abuse only and control. Table 3 shows no significant difference between groups for the left \((F(2,59) = 0.866, p = 0.426)\) or right \((F(2,59) = 0.609, p = 0.547)\) hippocampus. There was also no significant difference between the three groups when comparing the percentage of total brain volume in the left \((F(2,59) = 0.093, p = \)
0.911) and right \((F(2,59) = 0.215, p = 0.807)\) hippocampus. Table 4 presents the pituitary gland \((F(2,59) = 1.329, p = 0.273)\) and its percentage of total brain volume \((F(2,59) = 1.779, p = 0.178)\). Similarly, there is no reported significance between the groups in terms of pituitary structure. Lastly, table 5 presents the left caudate nucleus \((F(2,59) = 0.479, p = 0.622)\) and right caudate nucleus \((F(2,59) = 0.041, p = 0.959)\) along with the percentage of total brain volume for the left \((F(2,59) = 0.261, p = 0.771)\) and right caudate nucleus \((F(2,59) = 2.194, p = 0.121)\).

Again this shows that there is no significant difference in the caudate nucleus as is seen with the hippocampus and pituitary gland.

| Table 3. Hippocampal Volumes for PTSD & Abuse, Abuse Only and Control Groups. |
|---------------------------------|--------|--------|--------|--------|--------|--------|--------|
|                                | PTSD   |        | ABUSE  |        | CONTROL|        |
|                                | \(\bar{x}\) | SD     | \(\bar{x}\) | SD     | \(\bar{x}\) | SD     | \(p\) Value |
| Left Hippocampus               | 2938.9 | 486.7  | 2760.0 | 422.1  | 2912.5 | 439.3  | 0.426     |
| Right Hippocampus              | 3174.6 | 425.3  | 3050.6 | 395.4  | 3047.3 | 437.9  | 0.547     |
| Left Hippocampus % Brain       | 0.25   | 0.05   | 0.25   | 0.05   | 0.26   | 0.04   | 0.911     |
| Right Hippocampus % Brain      | 0.27   | 0.05   | 0.28   | 0.04   | 0.27   | 0.04   | 0.807     |

| Table 4. Pituitary Gland Volumes for PTSD & Abuse, Abuse Only and Control Groups. |
|---------------------------------|--------|--------|--------|--------|--------|--------|--------|
|                                | PTSD   |        | ABUSE  |        | CONTROL|        |
|                                | \(\bar{x}\) | SD     | \(\bar{x}\) | SD     | \(\bar{x}\) | SD     | \(p\) Value |
| Pituitary                      | 895.7  | 190.7  | 868.9  | 193.1  | 966.7  | 202.7  | 0.273     |
| Pituitary % Brain              | 0.08   | 0.01   | 0.08   | 0.02   | 0.09   | 0.02   | 0.178     |
Table 5. Caudate Nuclei Volumes for PTSD & Abuse, Abuse Only and Control Groups.

<table>
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<th>ABUSE</th>
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<td>Left Caudate</td>
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<tr>
<td>Right Caudate</td>
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<td>Left Caudate %</td>
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<td>0.04</td>
<td>0.4</td>
</tr>
<tr>
<td>Right Caudate %</td>
<td>0.4</td>
<td>0.04</td>
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Independent Sample t-tests

Independent sample t-tests were performed to further analyze the differences among the groups. Assuming equal variances the following results were gathered in Table 6 for PTSD versus control: left hippocampus ($t(40) = 0.184, p = 0.855$), right hippocampus ($t(40) = 0.956, p = 0.345$), pituitary gland ($t(40) = -1.168, p = 0.250$), left caudate ($t(40) = 0.454, p = 0.653$) and right caudate ($t(40) = -0.139, p = 0.890$). Thus, there is no significant difference among the groups for each brain structure as a result of independent sample t-test analysis. Likewise, Table 7 shows the results for abuse only versus control: left hippocampus ($t(37) = -1.100, p = 0.278$), right hippocampus ($t(37) = 0.024, p = 0.981$), pituitary gland ($t(37) = -1.535, p = 0.133$), left caudate ($t(37) = -0.553, p = 0.584$) and right caudate ($t(37) = -0.304, p = 0.763$). This cross-comparison also reveals no significant differences among the abuse only and control groups.

Table 6. PTSD & Abuse vs. Control Summary of Brain Areas.

<table>
<thead>
<tr>
<th>Brain Structure</th>
<th>Group</th>
<th>N</th>
<th>$t$</th>
<th>Significance</th>
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<td>-----------------</td>
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</tr>
<tr>
<td>Hippocampus (L)</td>
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<td>-1.535</td>
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Discussion

In this study we determined that there is no significant difference for the following brain areas: hippocampus, pituitary and caudate nucleus when comparing PTSD and abuse, abuse only and control participants. There is also no significant difference in terms of the percentage of total brain volume for these structures. Notably, the results seem to parallel the findings in Twamley et al. (2004) with the notable exception that the current study focuses predominately on structural differences rather than neuropsychological functional differences as a result of PTSD. PTSD may therefore not play a major role in altering long term structural volumes in a community sample of women with abuse-related PTSD.

Although much of the results across abuse-related research projects vary, the results presented for the hippocampal tracings are consistent with earlier results from our lab (Pederson et al. 2004). We did not find significant differences between groups for the pituitary gland, which is inconsistent with the results presented in Thomas & De Bellis (2004). This inconsistency may be due to differences in age and time since trauma between children and adults. Lastly, the caudate nucleus results are consistent with the findings in Bremner et al. (1997) which support the notion that the caudate nucleus acts a control region in the brain. Although there is a wide variation in findings, this study provides one of the first instances for comparison of brain structures in women affected by child abuse and PTSD in a community setting.

Several factors may account for these variations in results. Given that we sampled participants from a community setting, it is likely that the participants were functional enough to partake in daily activities such as raising a family and occupational duties. Thus, while 39 of the participants experienced adverse events growing up, the introduction of positive life experiences
possibly reverses previous stress on the developing brain. Stein *et al.* (1997) mentions that the brain’s own neural plasticity may play a role in sustaining brain function as one matures. Additionally, one’s genetic disposition can sensitize or desensitize her to stress (Carrion *et al.* 2007). The brain itself may also have inherent mechanisms for coping with traumatic stress. For abuse-related PTSD participants, cortisol release could be suppressed as a means to preserve internal brain energy for times of immediate and traumatic stress (Bremner *et al.* 2003). The release of cortisol and its effect on brain area volume could have played a role in the study, which may account for why no significant difference was found between abuse-related PTSD and the control group.

Additionally, the limitations of our study must be considered. First, the data gathered from the phone questionnaires and psychological tests were based upon self report. For instance, police records were not sought, nor were family members interviewed to verify instances of child abuse. Thus, the issue of integrity among participants remains questionable; however, the use of multiple tests and the aid of a psychologist helped to verify consistency among the study's participants and their associated group placement. Also, the sample size was somewhat small for a community setting. Increasing sample size would enable one to better establish a solid trend of the effects of child abuse in community-based samples. The MRI in itself limited participants on the basis of pregnancy, pacemakers, and other metal objects. One participant, in particular, was able to get through the psychological testing, but experienced anxiety when it was time to get the MRI. Thus, MRI may induce fear in participants which may affect the study outcome.

This study is largely based upon the assumption that PTSD-related trauma is a major consequence of child abuse; thereby this condition plays a large role in the recruitment of participants for the study. Yet, trauma affects individuals differently. The base rates of PTSD in
participants may vary among participants and it is difficult to determine whether stress is related to the child abuse or additional events in one's life (Tolin and Foa 2006). Given that PTSD did not significantly affect brain structural volume, this variable may in fact be unrelated to structural changes in a community sample. Furthermore, the participants were functional members of society so the threshold severity of PTSD in the clinical and community settings may differ.

The strength of this study is that it allows for comparison between not just PTSD and control groups, but also between PTSD and abuse only groups which adds a level of control for the abuse history found in the PTSD group. This enables one to gain more insights on the effects of abuse and how the severity of abuse can influence areas of the brain. In summary, the hippocampus, pituitary gland, and caudate nuclei were not significantly different between our PTSD, abuse only, and normal control groups. Further research utilizing imaging technology still needs to be completed to generate a consistent trend and assess whether child abuse affects adult brain development. Much of the literature utilizes inpatient participants for studies involving PTSD-related child abuse. The area of community settings needs to be given further attention as it may help medical providers and researchers to understand a larger sector of the abuse survivor population. Gender studies could also be implicated in understanding brain structural changes as a result of child abuse exposure. A cross-comparison of male and female abuse victims in a community sample would serve to provide a greater understanding of the effects of abuse on the brain. More importantly, the means of obtaining abuse histories and establishing a consistent definition of abuse needs to be given further attention. Overall, we hope to attain further neurobiological information on the impacts of child abuse and assess the implications for brain development in adulthood.
Acknowledgements

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References


