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Hippocampal volume in women victimized by childhood sexual abuse

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ABSTRACT

Background. Several prior studies have found reduced hippocampal volume in victims of psychological trauma with post-traumatic stress disorder (PTSD). We were interested to determine if this finding was evident in women who were victimized by severe sexual abuse in childhood.

Methods. In this study, hippocampal volume was measured using quantitative magnetic resonance imaging (MRI) in 21 women who reported being severely sexually abused in childhood and 21 sociodemographically similar women without abuse histories.

Results. Women who reported sexual victimization in childhood had significantly reduced (5% smaller) left-sided hippocampal volume compared to the non-victimized women. Hippocampal volume was also smaller on the right side, but this failed to reach statistical significance. Left-sided hippocampal volume correlated highly ($r_s = -0{\cdot}73$) with dissociative symptom severity, but not with indices of explicit memory functioning.

Conclusions. These findings, which are generally consistent with prior reports of reduced hippocampal volume in combat veterans with PTSD, suggest that diminished hippocampal size may be either a consequence of trauma exposure or a risk factor for the development of psychiatric complications following trauma exposure. The observed relationship between symptom severity and hippocampal volume suggests that mesial temporal lobe dysfunction may directly mediate certain aspects of PTSD and dissociative disorder symptomatology.

INTRODUCTION

Numerous studies have determined that exposure to child or adolescent sexual abuse (CSA) is a risk factor for the subsequent development of adult psychopathology (Herman *et al.* 1986; Burnam *et al.* 1988; Saunders *et al.* 1992; Mullen *et al.* 1993; Romans *et al.* 1995). Although CSA seems to be broadly associated with elevated risk for many psychiatric disorders including bulimia (Welch & Fairburn, 1993; Garfinkel *et al.* 1995), anxiety disorders (David *et al.* 1995; Mancini *et al.* 1995; Stein *et al.* 1996) and substance abuse (Triffleman *et al.* 1995), CSA is believed to be a dominant aetiological factor in the development of dissociative disorders (Spiegel & Cardeña, 1991; Nash *et al.* 1993; Irwin, 1994; Spiegel, 1994) and many forms of post-traumatic stress disorder (PTSD) (Bremner *et al.* 1993*b*; Wolfe *et al.* 1994; Zlotnick *et al.* 1996).

Although research is now being conducted into sociocultural and psychological factors that may influence vulnerability to (and resilience from) the negative psychiatric outcomes of criminal victimization in women (Norris, 1992; Rothbaum *et al.* 1992; Astin *et al.* 1993), little research has been conducted into neurobiological factors that may be of importance. In fact, it is only in the past several years that investigators have considered the possibility that severe emotional trauma may affect not only the psyche, but also the brain (Friedman *et al.* 1995).

Preclinical research conducted over the past

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decade has shown that experimental stressors (e.g. restraint stress or social stress) can result in functional and morphological changes within the hippocampus in rodents and primates (Sapolsky *et al.* 1985; 1990; Gould *et al.* 1994; Sapolsky, 1994). It is now generally accepted that stress-induced elevations of glucocorticoids augment the extracellular accumulation of excitatory amino acids (EAAs) such as glutamate (Moghaddam *et al.* 1994; Stein-Behrens *et al.* 1994), resulting in hippocampal damage which is evident both at the cytoarchitectural (i.e. reduced cell sprouting and neuronal cell death, particularly in the CA3 region) (Stein-Behrens *et al.* 1994) and functional (i.e. impaired learning and memory) (Luine *et al.* 1994; Alvarez *et al.* 1995; Bodnoff *et al.* 1995) levels.

These findings have led clinical investigators to hypothesize that exposure to traumatic stress might analogously affect hippocampal morphology and functioning in humans. In light of the well-established effects of hippocampal damage on memory systems in humans and other primates (Squire & Zola-Morgan, 1991; Miller *et al.* 1993; Zola-Morgan & Squire, 1993; Zola-Morgan *et al.* 1994; Alvarez *et al.* 1995), recent research efforts have focused on determining whether or not patients with PTSD have memory problems (Sutker *et al.* 1991; Bremner *et al.* 1993*a*, 1995*a*; Gurvits *et al.* 1993; Uddo *et al.* 1993; Sutker *et al.* 1995; Yehuda *et al.* 1995) and, in some studies, on whether or not they manifest radiological evidence of hippocampal damage (Bremmer *et al.* 1995*b*, 1997; Gurvits *et al.* 1996).

Bremner *et al.* (1995*b*) showed that male combat veterans with PTSD had reduced MRI-derived right-sided hippocampal volume compared to control subjects and, moreover, that certain aspects of their memory deficit were correlated with hippocampal volume. The finding of reduced hippocampal volume in male combat veterans with PTSD has recently been replicated by Gurvits and colleagues (1996), who found a bilateral effect. Most recently, Bremner *et al.* (1997) were able to demonstrate a 12% reduction in left-sided hippocampal volume in a mixed sample of men and women who experienced abuse in childhood.

In the present study, we used MRI-based measurements to assess hippocampal volume in women who experienced a common form of psychological trauma – childhood sexual abuse (CSA) (Breslau *et al.* 1991; Anderson *et al.* 1993; Resnick *et al.* 1993; Kessler *et al.* 1995), in comparison to non-victimized control women. We hypothesized that women who experienced CSA would have smaller hippocampi than the non-abused subjects.

In a prior study, Bremner *et al.* (1995*b*) found that right-sided hippocampal volume correlated negatively with certain aspects of short-term memory functioning. However, Gurvitis *et al.* (1996) found no relationship between hippocampal volume and memory functioning. In the present report we also examine the relationship between verbal explicit memory functioning and hippocampal volume to test the hypothesis that these will be negatively correlated in the abused subjects.

METHOD

Subjects

Subjects were 21 adult females who reported having experienced severe childhood sexual abuse (CSA) and 21 adult females who had no history of abuse (nCSA). All subjects gave informed, written consent to participate. Women with CSA and nCSA comparison subjects were recruited for this study using notices posted in waiting rooms in several community women's health care clinics. The notices outlined the nature of our research programme and indicated that we were seeking women with histories of childhood sexual abuse as well as women without abuse histories. An honorarium was offered for participation. Potential subjects were asked to leave their name and phone number on an answering machine, and one of the investigators (C.H.) contacted them by telephone to determine preliminary their eligibility for participation.

A 20–30 min telephone interview determined the presence or absence of severe CSA, which we defined as the report to the investigator of attempted or completed vaginal or anal penetration occurring between a child 14 years of age or younger and a perpetrator who was at least 5 years older than the child. We did not attempt to corroborate the subject's self-report with external evidence of abuse and neglect (e.g. child protective agency records, reports of contemporary informants) owing, in part, to the difficulty in obtaining such data – in the minority

of cases where such external data are available at all (Finkelhor & Dziuba-Leatherman, 1994). We did, however, decide *a priori* to mitigate the chances of miscategorizing subjects by excluding persons who reported 'recovering' their memories of abuse in the context of psychotherapy (Williams, 1994; Pope & Hudson, 1995; Bremner *et al.* 1996). In actuality, though, none of the subjects reported a sustained period of total amnesia for knowing that they had been sexually abused.

Non-abused controls denied exposure to CSA or other traumata including witnessing a death, being assaulted, being physically abused, or being sexually abused in childhood or adulthood. Non-victimized controls were also required to be free of current Axis I pathology; this resulted in the exclusion of three otherwise-eligible nCSA subjects (one with obsessive–compulsive disorder, one with generalized anxiety disorder and one with major depressive disorder).

Subjects were ruled ineligible to participate if they had a history of head injury requiring any rehabilitation or hospitalization for longer than an overnight stay, or a history of seizures (except for $\leqslant 2$ febrile seizures in childhood) or other neurological disorders. One CSA subject who was completing a 10-day course of oral prednisone for asthma was excluded, in case this might affect her cognitive functioning (Newcomer *et al.* 1994). One grossly obese subject (weight $>$ 300 pounds) was also excluded.

At the time of study, three CSA subjects took psychoactive mediations: one took amitriptyline 75 mg/day; one took haloperidol 1 mg/day; and one took trazadone 100 mg/day and alprazolam 0·5 mg p.r.n. Two nCSA subjects took psychoactive medications at bedtime for sleep: one took amitriptyline 50 mg and one took lorazepam 0·5 mg. Since none of these medications would be expected to influence hippocampal or total brain volume, these subjects were included.

Subjects currently abusing alcohol or other substances were excluded. Prior course of substance (ab)use was documented during the diagnostic interview, and the Michigan Alcohol Screening Test – short version (SMAST; Selzer *et al.* 1975) and Drug Abuse Screen Test (DAST; Skinner, 1982) were administered to reflect the overall severity of lifetime drug and alcohol use. SMAST scores were 7·43 (S.D. 7·12) in the CSA subjects *v.* 0·52 (S.D. 1·40) in the nCSA subjects ($S = 582$, $z = -3{\cdot}68$, $P < 0{\cdot}0002$). DAST scores were 6·57 (S.D. 5·08) in the nCSA subjects *v.* 1·86 (S.D. 2·13) in the nCSA subjects ($S = 583$, $z = -3{\cdot}32$, $P < 0{\cdot}002$).

All subjects were determined by history and, where appropriate, by review of medical records or the administration of ancillary tests, to be free of significant medical illness. Educational level and socio-economic (Hollingshead, A.B. – Four factor index of social status. Unpublished manuscript, 1975, Yale University) were recorded. Two of the 21 CSA subjects and three of the 21 nCSA subjects were left-handed.

Psychiatric assessment

All subjects were evaluated using a version of the Structured Clinical Interview for DSM-IV (SCID; First *et al.* 1995) and a separate interview for DSM-IV Dissociative Disorders (SCID-D; Steinberg *et al.* 1990; Bremner *et al.* 1993*b*). The Clinician-Administered PTSD Scale (CAPS; Blake *et al.* 1995) was used to assess PTSD severity in the abuse victims. The Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986), a widely-used self-report measure of dissociative symptoms, and the Beck Depression Inventory (BDI; Beck *et al.* 1961), a widely-used self-report measure of depressive symptoms, were also administered.

To assess intellectual functioning the subjects were administered five subtests of the Weschler Adult Intelligence Scale–Revised (WAIS-R; Wechsler, 1981): the vocabulary, similarities, picture completion, block design and digit symbol tests. Explicit memory performance was assessed using the California Verbal Learning Test (CVLT; Delis *et al.* 1987). Detailed results will be presented in a separate, future publication; but the relationship of several chief measures of intellectual and memory functioning with hippocampal volume will be examined here.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) scans were conducted by experienced personnel on a Siemens Magnetom SP63 Helicon SE2 at 1.5 Tesla field strength. A T1-weighted coronal localizer scan was used to ensure that the entire hippocampus was being imaged. This

was followed by a T1-weighted sagittal sequence (BW = 89 Hz, TR = 550 ms, TE = 14 ms, Flip angle = 65°; thickness = 5 mm, FOV = 230 mm, matrix = 256 × 256). The volumetric acquisition used a T2-weighted Turbo Spin Echo (TURBOSE) sequence (TR = 4000 ms, TE = 90 ms, thickness = 4 mm, interslice gap = 0·4 mm, matrix = 335 × 512@0·75 FOV, FOV = 260 mm (195 mm@0·75 FOV), acquisitions = 2, echo train = 12, BW = 130 Hz). The orientation of the volumetric acquisition was in the oblique coronal plane, with slices taken perpendicular to the hippocampus as determined from the parallel sagittal slices as described above. Excellent definition of the hippocampus was made possible by the superb in-plane resolution of these images. Scans were read clinically by a board-certified neuroradiologist (B.M.) to rule out identifiable pathology (e.g. tumour, MS plaques).

Hippocampal volumetric analyses

MR images were transferred to a computerized system (Allegro Software Version 5.1.1., ISG Technologies, Mississauga, ON, Canada) for outlining the volumes of interest (VOIs), reconstructing 3-D images, and computing volume measurements. The oblique coronal slices from the TURBOSE sequence were used to reconstruct the hippocampus. The 'index' slice was that slice demonstrating the mamilliary bodies. One slice posterior to this was used as the first slice to outline L/R hippocampus VOI. Seven slices and corresponding L/R hippocampal VOIs were then examined moving posterior to anterior up to the fornix. All VOIs were traced manually using a mouse-driven cursor, separately for the right and left sides. Computer algorithms were then used to reconstruct right and left hippocampal volumes, in mm^3. For use as a reference factor, a 'standardized' brain volume was computed by producing a total brain VOI from the 'index' slice. This produced a brain slice 4 mm in thickness with the circumference varying from patient to patient. Volumetric analysis were conducted by an experienced research associate (M.G.T.) who was blind to diagnostic status. The manual tracing were conducted twice, on separate occasions (test–retest reliability, intra-class correlation coefficient (ICC) = 0·67 for left hippocampus; ICC = 0·71 for right hippocampus) and the volumes reported here are averages of these measurements.

Statistical analysis

Sociodemographic and symptom comparisons between CSA and nCSA subjects were made using Student's *t* tests or Wilcoxon Rank Sum tests, where appropriate. Given the unidirectional nature of our hypotheses (i.e. that the CSA subjects would have smaller hippocampi than the nCSA subjects) we used one-tailed statistical tests for the analyses involving hippocampal volume. Analysis of variance with repeated measures (ANOVAR) was used: between-groups effects were abuse status (i.e. CSA or nCSA) and the repeated within-subjects effect was side (i.e. left or right). Dunnett's test was applied to test for between-groups differences on a *post hoc* basis when a main or interactional effect was seen at the $P < 0{\cdot}10$ (two-tailed, which would correspond to $P < 0{\cdot}05$ using a one-tailed test) level.

Although the merits of using a standardized brain region as a volumetric reference factor continue to be debated (Arndt *et al.* 1991; Wang & Jernigan, 1994), we also conducted the analyses covarying for the total brain slice VOI (as described above). Symptom severity scores (e.g. DES) were often non-parameterically distributed, so associations with hippocampal volume were tested using the Spearman correlation coefficient (r_s). A total of 15 correlations were tested, so the *P* value for a significant result was reduced to 0·003 (i.e. 0·05/15); only correlations meeting this level of significance are reported. Data are expressed as mean (S.D.).

RESULTS

Characteristics of the subjects

Sociodemographic, body morphometric, and IQ parameters

Women who reported severe sexual abuse during childhood (CSA) were similar to non-abused comparison subjects (nCSA) on all indices of sociodemographic status, body morphometrics, and intellectual functioning (Table 1).

Diagnostic assessment

Fifteen of the 21 CSA subjects (71·4%) met DSM-IV criteria for a current diagnosis of PTSD, and 15 of 21 (71·4%) met criteria for a

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Table 1. *Characteristics of CSA and nCSA subjects**

	CSA ($N = 21$)	nCSA ($N = 21$)
Age (years)	32·0 (6·3)	30·2 (6·4)
Education (years)	13·0 (3·0)	13·7 (1·9)
SES (Hollingshead)	34·3 (10·5)	36·9 (9·8)
Height (inches)	65·0 (2·9)	65·9 (2·0)
Weight (lbs)	159 (47)	149 (29)
Body Mass Index (kg/m²)	26·1 (7·0)	24·3 (5·2)
Handedness (% right)	90·5%	85·7%
WAIS-R Vocabulary Subscale	12·0 (2·4)	11·5 (3·1)
WAIS-R Similarities Subscale	10·5 (1·7)	10·5 (1·7)
WAIS-R Picture Completion Subscale	9·1 (2·0)	9·1 (2·5)
WAIS-R Digit-Symbol Substitution Subscale	10·6 (2·5)	11·9 (2·4)

* Values are expressed as mean (S.D.). Student's t test, 2-tailed, all P values NS.

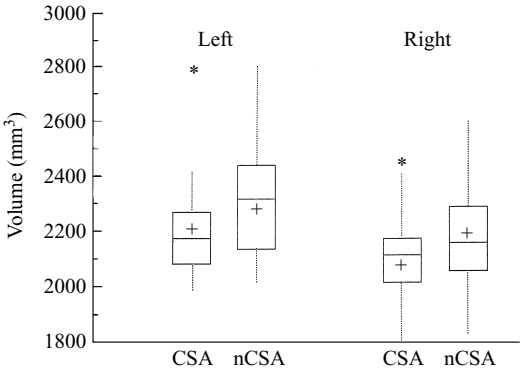

FIG. 1. Boxplots of MRI-derived hippocampal volumes (mm³) in 21 women with severe childhood sexual abuse (CSA) and 21 non-abused women (nCSA). Boxes include the interquartile range (i.e. 25th to 75th percentile) with the horizontal line within the box indicating the group median (+ indicates the group mean). The vertical lines extending from the boxes show the range of the data within twice the interquartile range, and * represents outliers beyond this range.

dissociative disorder (1 met criteria for dissociative amnesia, 5 for dissociative identity disorder, and 9 for dissociative disorder NOS); 13 subjects had both PTSD and a dissociative disorder. Six of 21 CSA subjects (28·6%) met criteria for a current diagnosis of major depression. Other current co-morbid disorders in the CSA subjects were social phobia ($N = 1$) and obsessive-compulsive disorder ($N = 1$).

Psychiatric symptom severity

In the CSA subjects, CAPS subscale scores were as follows: re-experiencing 11·8 (S.D. 10·3), range 0–31; numbing 22·9 (S.D. 15·5), range 0–48; arousal 23·6 (S.D. 11·6), range 6–43. Mean total CAPS score was 58·2 (S.D. 34·8), range 11–122. CAPS scores could not be obtained for the nCSA subjects who, by definition, had not experienced severe trauma.

Dissociative Experience Scale scores were 17·8 (S.D. 13·6), range 4–63 in the CSA subjects *v.* 4·0 (S.D. 3·6), range 0–11 in the nCSA subjects (Wilcoxon Rank Sum (S) = 636·5, $z = -4{\cdot}65$, $P < 0{\cdot}0001$). Three CSA subjects but none of the nCSA subjects had DES scores $\geqslant 30$ – a cut-off score indicative of clinically significant dissociative symptomatology (Carlson *et al.* 1993).

Beck Depression Inventory scores were 16·1 (S.D. 12·9), range 3–46 in the CSA subjects *v.* 6·2 (S.D. 4·6), range 0–17 in the nCSA subjects ($S = 574{\cdot}5$, $z = 3{\cdot}09$, $P < 0{\cdot}002$).

Hippocampal volumes

Right-sided hippocampal volumes were 2097 (S.D. 169) mm³ in the CSA subjects *v.* 2160 (S.D. 210) mm³ in the nCSA subjects (i.e. 2·9% smaller in the CSA subjects). Left-sided hippocampal volumes were 2194 (S.D. 181) mm³ in the CSA subjects *v.* 2307 (S.D. 193) mm³ in the nCSA subjects (i.e. 4·9% smaller in the CSA subjects). ANOVARs revealed a main effect of abuse status ($F_{1,40}$- = 2·94, $P < 0{\cdot}05$, 1-tailed), a significant main effect of side (larger on the left; $F_{1,40} = 19{\cdot}85$, $P < 0{\cdot}0001$), but no significant abuse status × side interaction ($F_{1,40} = 0{\cdot}80$, $P =$ NS). *Post-hoc* testing indicated that CSA and nCSA subjects did not differ significantly in right-sided hippocampal volumes (Dunnett's T test, (T) = 1·69, mean significant difference, (M.S.D.) = 99·0, $P =$ NS, 1-tailed), but that CSA subjects had significantly smaller left-sided hippocampal volumes than nCSA subjects (T = 1·69, M.S.D. = 97·1, $P < 0{\cdot}05$, 1-tailed) (Fig. 1). The finding

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remained significant when either the reference brain slice volume or SMAST score was used as a covariate, or if right-handed subjects only were included.

Relationship to alcohol use history

There were no significant correlations observed between SMAST scores and either total, right-sided, or left-sided hippocampal volume. As a more stringent test of the possibility that hippocampal volume might be related to alcohol abuse, we also compared hippocampal volumes among CSA ($N = 11$) and nCSA ($N = 21$) subjects with SMAST scores $\leqslant 5$; such scores are indicative of the absence of a history of alcohol use problems (Selzer *et al.* 1975). As for the sample as a whole, the two groups exhibited a main effect of diagnosis ($F_{1,30} = 3{\cdot}50$, $P < 0{\cdot}04$, 1-tailed), with *post-hoc* testing revealing significantly smaller hippocampi on the left side ($T = 1{\cdot}70$, $P < 0{\cdot}05$, 1-tailed).

Relationship between hippocampal volume and psychiatric symptom severity

DES scores correlated significantly ($r_s = -0{\cdot}73$, df = 20, $P < 0{\cdot}0002$) with left-sided hippocampal volumes in the CSA subjects. Although modest correlations (i.e. in the range of 0·4–0·5) were noted between several of the CAPS subscales and both total and left-sided hippocampal volumes, these fell below our stringent P level of 0·003 after adjusting for multiple tests. Beck Depression Inventory scores did not correlate significantly with either right, left, or total hippocampal volume in either CSA or nCSA subjects.

Relationship between hippocampal volume and abuse characteristics

There were no significant correlations between hippocampal volume and either age at onset of abuse or duration of abuse in the CSA subjects. As an index of abuse severity, the number of perpetrators was examined in relation to hippocampal volume; no significant correlations emerged.

Relationship between hippocampal volume and memory functioning

Memory test results of the two groups will be presented in detail in a separate, future report. We wish to note here, however, that there were no significant differences between groups on explicit memory functioning and no correlations in either CSA or nCSA subjects between hippocampal volume and any of the measures of learning and memory on the California Verbal Learning Test.

DISCUSSION

In this study of adult women who reported being victims of severe childhood sexual abuse (CSA) we found that hippocampal volumes – most discernibly on the left side – were reduced compared to those of demographically, educationally, and intellectually-comparable women without abuse histories. We also found that within the CSA subjects the severity of dissociative and, to a lesser extent, other post-traumatic stress disorder (PTSD) symptoms correlated significantly with left hippocampal volume. These findings support a possible relationship between hippocampal dysfunction and post-traumatic psychiatric symptoms.

Although our observations of reduced hippocampal volume in female CSA victims are generally consonant with similar findings in three other studies of psychologically traumatized subjects (Bremner *et al.* 1995*b*; 1997; Gurvits *et al.* 1996), several caveats apply. First, it must be recognized that our sample of CSA victims was biased toward including those with self-identified severe sexual abuse; this is reflected in the high rates of PTSD and dissociative disorders diagnosed in our sample. Consequently, our findings are unlikely generalizable to all CSA victims in the community. Secondly, it should be noted that our sample of CSA victims had increased alcohol and drug use severity compared to our control subjects. In our study, prior alcoholism severity did not appear to account for the hippocampal volumetric findings, as smaller left-sided hippocampal volume was evident even in CSA subjects without any significant history of alcohol use problems. Nevertheless, given the evidence that individuals exposed to trauma are at increased risk for alcohol abuse (see Stewart, 1996 for review) and that longstanding alcohol abuse can lead to hippocampal (and other brain) volume loss (Pfefferbaum *et al.* 1988; Di Sclafani *et al.* 1995) it will be important in future studies to carefully control for this factor.

A critical question raised by our findings is

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whether reduced hippocampal size can be caused by severe childhood emotional trauma. Although our original rationale for conducting this study was based on the hypothesis that severe psychological trauma would cause hippocampal 'damage' – as is known to occur secondary to stress in several rodent and primate models (Sapolsky *et al*. 1985, 1990; Sapolsky, 1994; Stein-Behrens *et al*. 1994) – it is important to recognize that our data do not directly test this hypothesis, and we remain uniformed about the extent to which it is true. It is indeed possible that abuse experiences constitute a psychological stressor of sufficient magnitude to result in hippocampal 'atrophy'. An alternative hypothesis which deserves serious consideration, though, is that hippocampal differences might have been present prior to the trauma, and that such differences might somehow predispose the individual to the development of psychiatric complications of trauma (e.g. PTSD). Given the information that genetic factors are important in the development of combat-related PTSD (Goldberg *et al*. 1990), we must strongly consider the possibility that a constitutional (perhaps genetic) abnormality in hippocampal development – which could be a risk factor for the development of psychiatric symptoms in the face of exposure to psychological trauma – might be the basis for our findings.

How are we to explain our finding of hippocampal volume reduction in the absence of explicit memory dysfunction? It is possible, given the relatively young age of our subjects, that subtle differences in memory may have been undetectable at present, but might be discernible with more difficult memory tasks or become more apparent as ageing progresses. It is also possible that changes in hippocampal morphometry as detected by MRI may be a relatively insensitive indicator of more meaningful hippocampal functional or metabolic changes. These may be better detected in future studies using techniques such as magnetic resonance spectroscopy (Dager & Steen, 1992; Hennig *et al*. 1992), positron emission tomography (Squire *et al*. 1992; Rauch *et al*. 1996; Schacter *et al*. 1996), or functional magnetic resonance imaging (Breiter *et al*. 1996). We must also consider the possibility that the magnitude of hippocampal volume reduction (5%) found here is so negligible that memory impairment does not result, or, alternatively, that the hippocampal insult may have transpired early enough in life that adequate functional comensation has occurred.

Finally, though, we must seriously entertain the likelihood, on the basis of our data, that some forms of hippocampal dysfunction may not result in explicit in explicit memory dysfunction, *per se*. In this context, we might speculate that the site and/or nature of the hippocampal abnormality seen here spares explicit 'memory' in the conventional sense, but somehow disrupts brain systems responsible for a range of metamemory functions such as the ability to integrate one's own recollections into a cohesive narrative, and the capacity to understand these remembrances in the context of one's 'self'. In other words, the hippocampal abnormality may directly mediate many aspects of the phenomena known as dissociation (Krystal *et al*. 1995). If this was true, then dissociative symptoms such as traumatic amnesia and intrusive recollections might be better conceptualized as dysfunction within systems that monitor memory and regulate access to memory in emotionally-charged contexts. A challenge to progress in this field will be to elucidate the neuroanatomical substrates of these systems and to determine how, and why, they may fail in response to overwhelming psychological trauma.

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