# Assault-related self-blame and its association with PTSD in sexually assaulted women: an MRI inquiry

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## Abstract

Sexual assault is a frequent interpersonal trauma, which often leads to post-traumatic stress disorder (PTSD). Among other postassault characteristics, self-blame attributions were suggested to play an important role in sexually assaulted individuals' coping and were consistently associated with PTSD in this population. The present study aimed to elucidate the neural underpinnings that may associate self-blame and PTSD in women who experienced sexual assault at adulthood, using structural and resting-state functional MRI. Thirty-eight sexually assaulted women and 24 non-exposed matched controls were studied (mean age: 25 years). Among the sexually assaulted participants, assault-related self-blame was negatively correlated with gray matter volume (GMV) bilaterally in the lingual gyrus and adjacent intracalcarine cortex. GMV in this cluster was also predicted by intrusion symptoms and negative social reactions. Resting-state functional connectivity (rs-FC) of this cluster with the left anterior temporal fusiform cortex significantly differed between PTSD and non-PTSD sexually assaulted participants, and was inversely correlated with intrusion symptoms and with peritraumatic dissociation. Finally, lingual cluster's GMV and rs-FC with the anterior fusiform mediated the association between self-blame and intrusion symptoms across sexually assaulted participants. These findings link assault-related self-blame, disrupted postassault recovery and the neural circuitry involved in the processing of traumatic memories.

Key words: self-blame; sexual assault; post-traumatic stress disorder; magnetic resonance imaging

## Introduction

Sexual assault is a well-established risk factor for various mental and physical health consequences, including post-traumatic stress disorder (PTSD) (Golding, 1994; Elliott *et al.*, 2004; Kilpatrick *et al.*, 2007; for a recent review see Dworkin *et al.*, 2017). Of the different postassault characteristics that were suggested to play a crucial role in victims' psychological coping (e.g. Boeschen *et al.*, 2001; Ullman *et al.*, 2007; Littleton, 2010), one of the most studied is self-blame attributions, which have consistently been found to correlate with PTSD symptomatology in this population (Frazier, 2000; Boeschen *et al.*, 2001; Filipas and

Ullman, 2006; Ullman *et al.*, 2007; Najdowski and Ullman, 2009). Importantly, self-blame attributions following sexual assault are often boosted by a stigmatizing social climate and victim-blaming reactions from various community systems and support providers (Campbell and Raja, 1999; Filipas and Ullman, 2001; Ahrens, 2006; Campbell *et al.*, 2009; Kennedy and Prock, 2016). Consequently, compared with victims of other traumatic experiences such as combat stress and car accident, in victims of sexual assault, trauma-related self-blame is highest, and its association with PTSD is the strongest (Moor and Farchi, 2011).

Several studies addressed the mechanism associating self-blame to post-traumatic symptomatology in sexually assaulted women. While some suggested that it involves re-experiencing sensations, maladaptive beliefs and global distress (Boeschen *et al.*, 2001; Koss *et al.*, 2002; Koss and Figueredo, 2004), others focused on coping strategies (Najdowski and Ullman, 2009). Yet, despite the great empirical support for an association between these phenomena, the pathway by which self-blame attributions contribute to post-traumatic symptoms is not well understood.

Many neuroimaging studies have utilized magnetic resonance imaging (MRI) to investigate the neural underpinnings of emotional and cognitive processes [such as emotion dysregulation (e.g. Powers *et al.*, 2017) and overgeneralized self-blame (e.g. Green *et al.*, 2012; Lythe *et al.*, 2015)] that are associated with the emergence of psychiatric disorders. Yet, no study assessed the neural traits associated with self-blame attributions in sexually assaulted individuals. Thus far, MRI studies of self-blame were conducted in healthy samples during fictional lab tasks (e.g. Seidel *et al.*, 2010; Nicolle *et al.*, 2011) or in depressed individuals (e.g. Green *et al.*, 2012), and pointed to the involvement of regions such as the subgenual cingulate cortex and amygdala. Only one study addressed the neural correlates of cognitive distortions, including self-blame, in explicitly traumatized individuals, most of them in a car accident (Daniels *et al.*, 2011). During trauma recall, distorted cognitions were correlated with blood–oxygen-level-dependent (BOLD) signal intensity in brain areas implicated in imagery, autobiographic memory recall, and motor preparation, most notably the visual cortex, somatosensory areas and basal ganglia. As these activations were also strongly correlated with re-experiencing, the authors concluded that cognitive distortions are linked with vivid imagery as it is elicited by trauma cues (Daniels *et al.*, 2011).

The goal of the present study was to elucidate the neural underpinnings that may associate assault-related self-blame and PTSD in sexually assaulted individuals. To this end, we examined whether self-blame attributions are associated with regional neuroanatomical alterations in gray matter volume (GMV) across the whole brain in women who were sexually assaulted during adulthood, and if so, whether and how such alterations are functionally related to PTSD. The latter was evaluated by assessing whether resting-state functional connectivity (rs-FC) patterns of regions showing self-blame-related GMV alterations differ between PTSD and non-PTSD participants, and whether these patterns are correlated with overall post-traumatic symptomatology and/or specific PTSD symptom clusters (for a similar approach see Ma *et al.*, 2012; Wang *et al.*, 2017). Finally, a mediation analysis was conducted to assess whether identified neural alterations mediate the association between self-blame and PTSD symptoms. Although we hypothesized that self-blame would be negatively correlated with GMV in regions previously implicated in self-blame and/or PTSD (e.g. anterior cingulate cortex, amygdala), in light of the scarcity of studies of structural alterations in women who were victims of sexual assault during adulthood, and lack of studies of the neural substrates of self-blame in this population, our analyses were exploratory.

## Materials and methods

### Participants

Sixty-eight women aged 19–37 years enrolled in the present study: 38 victims of adult sexual assault and 30 non-exposed controls (NEC) matched for age, education level and handedness. Participants were recruited via advertisements in social media and in the Tel Aviv Rape Crisis Center. Inclusion criterion for the traumatized individuals was experiencing sexual assault(s) in the past 3 years and after the age of 18 years. In order to maintain the ecological validity of the study, and because many victims of adult sexual assault have histories of childhood sexual abuse (e.g. Elliott *et al.*, 2004), participants with additional experiences of earlier sexual assaults were not excluded. Although we tried to recruit both women and men to participate in the study, only women responded to our advertisements. NEC inclusion criterion was a lack of exposure to significant traumatic events. Exclusion criteria for both groups included MRI contraindications, a history of neurological disorder or any pervasive developmental disorder, head injury, fibromyalgia, any significant physical condition that is not being treated, and a frequent use of alcohol during the past 6 months. NEC participants were additionally excluded if they had a history of any psychiatric disorder. Prior to enrollment in the study, all participants were provided with full details about the study and gave informed written consent. The study was approved by the Sheba hospital Helsinki committee and the Tel Aviv University ethical board.

### Procedure

The study took place at the Strauss Center for Computational Neuroimaging at Tel Aviv University. The experimental session included a 40-min MRI scan followed by 45 min of filling out self-report questionnaires in a quiet, private room. Women in the Sexual Assault group were instructed to fill out the questionnaires with regard to their most recent experience of sexual assault, while NEC participants were instructed to fill them out with regard to a self-chosen difficult event, which they have experienced sometime along their lives. Upon completion, participants were debriefed and asked for their feelings, and finally thanked and compensated for their time.

### Measures

*Self-blame attributions.* Assault-related self-blame was assessed using the Rape Attribution Questionnaire (RAQ; Frazier, 2003). Of its five subscales, we analyzed only the items assessing characterological self-blame, which assigns the blame to the kind of person one is (e.g. 'things like this happen to people like me'), and is consistently considered to be psychologically maladaptive (Koss *et al.*, 2002; Hall *et al.*, 2003; Ullman *et al.*, 2007). The five items (each answered on a five-point scale) assessing this kind of self-blame were averaged to create a Self-Blame score. Women in the NEC group were given a modified version of the questionnaire, in which items were rephrased to assess participant's view of reasons for the occurrence of rape. Thus, for NEC participants, this score reflects rape-related victim-blaming attitudes.

*Psychological symptomatology.* Post-traumatic symptoms were assessed with the PTSD Checklist (PCL-5; Weathers *et al.*, 2013), a widely used self-report measure with good psychometric properties (e.g. Keane *et al.*, 2014; Bovin *et al.*, 2016). The PCL-5 includes 20 items that assess PTSD symptoms severity (on a five-point scale) during the past month. We calculated a total severity score as well as scores on its four subscales: Intrusion, Avoidance, Negative Alterations in Cognitions and Mood, and Alterations in Arousal and Reactivity. For some analyses, the

sexually assaulted women were divided into probable PTSD and non-PTSD subgroups according to a PCL-5 cutoff score of 31, which corresponds to diagnoses made with the Clinician-Administered PTSD Scale (Bovin *et al.*, 2016) and is in agreement with the PCL-S cutoff of 44, which was recommended for use with female civilian samples (Blanchard *et al.*, 1996; Hoge *et al.*, 2014). Depressive symptoms were assessed with the Patient Health Questionnaire—depression module (PHQ-9; Kroenke *et al.*, 2001) that consists of nine items, which measure the frequency (on a five-point scale) of experiencing depressive symptoms during the past 2 weeks. General distress was measured with the Brief Symptom Inventory (BSI; Derogatis and Melisaratos, 1983) that includes 53 items, which assess the degree (on a five-point scale) of psychological distress during the prior month. Total mean scores were calculated for the PHQ-9 and the BSI.

*Childhood trauma.* The 28-item Childhood Trauma Questionnaire (CTQ; Bernstein and Fink, 1998) was used to retrospectively measure the frequency (on a five-point scale) of traumatic experiences in childhood, including sexual abuse, physical abuse, emotional abuse, physical neglect and emotional neglect. Total CTQ score was calculated for each participant.

*Peritraumatic and postassault characteristics.* The Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Marmar *et al.*, 1997) was used to assess the degree of dissociation experienced during the assault. It contains eight statements describing dissociative experiences, which participants rate on a five-point scale. Negative social reactions following disclosure were assessed with the Social Reactions Questionnaire (SRQ; Ullman, 2000), which includes 48 items specifying responses often experienced by sexual assault victims, both positive and negative. Participants were asked to indicate (on a five-point scale) how often they received these responses from other people. Scores of all negative reactions items (e.g. 'pulled away from you') were averaged to create a Negative Reactions score. Perceived Social Support was measured with a single item, asking the participant to assess (on a five-point scale) the extent of the ongoing social support she is receiving.

The BSI, PCL, PHQ-9, CTQ and PDEQ were administered in their Hebrew versions, which have been found reliable and are extensively used (e.g. Hoffman *et al.*, 2016; Zerach and Magal, 2017), although only the Hebrew version of the BSI has been validated (Canetti *et al.*, 1994). We translated into Hebrew the RAQ and SRQ, and used back-translation and agreement between judges to validate the translation.

### Image acquisition

Participants underwent a T1-weighted structural scan followed by a resting-state fMRI scan in a Magnetom Prisma 3T scanner (Siemens, Germany) equipped with a 64-channel head coil. T1-weighted images were acquired in the axial plane using a 3D magnetization-prepared rapid gradient-echo sequence with the following parameters: $TR = 2530$ ms; $TE = 2.88$ ms; $TI = 1100$ ms; flip angle $= 7°$; matrix size $= 224 \times 224$; voxel size $= 1 \times 1 \times 1$ mm$^3$; iPAT $= 2$. The resting-state scan was carried out using a 2D BOLD echo planar sequence in an axial plane with the following parameters: $TR = 2000$ ms; $TE = 30$ ms; flip angle $= 75°$; matrix size $= 64 \times 64$; voxel size $= 3.5 \times 3.5 \times 3.5$ mm$^3$ and full brain coverage by acquiring 37 interleaved slices (no gaps); iPAT $= 2$ with fat suppression. For each participant, this sequence generated 200 volumes in a total of 6:40 min. During the resting-state scan,

participants were asked to relax, maintain their eyes closed, not to think of anything in particular and to stay awake.

### Image preprocessing

*Voxel-based morphometry.* For estimation of macroscopic volumetric regional tissue alterations, voxel-based morphometry (VBM, Ashburner and Friston, 2000) was performed with MATLAB (MathWorks, Natick, MA, USA) and SPM8 (Wellcome Department of Cognitive Neurology, London, UK; http://www.fil.ion.ucl.ac.uk/spm), using the optimized VBM protocol (Good *et al.*, 2001). Preprocessing included a segmentation of images into gray matter, white matter and cerebrospinal fluid, a high-dimensional DARTEL spatial normalization to MNI space and modulation for the non-linear components only, allowing for the analysis of GMV corrected for individual brain size. Lastly, images were smoothed with a Gaussian kernel of 8 mm. The resulting preprocessed gray matter maps were used for volumetric analyses.

*Resting-state functional connectivity.* rs-FC analyses were performed with the Functional Connectivity toolbox (CONN) v16 (Whitfield-Gabrieli and Nieto-Castanon, 2012) and SPM12. All preprocessing steps were conducted using CONN's default preprocessing pipeline for volume-based analyses on MNI-space. Following discarding of the first four volumes, preprocessing included slice time correction, realignment, normalization to MNI space and smoothing with a Gaussian kernel of 8 mm. The aCompCor strategy (Behzadi *et al.*, 2007) was used to extract signal noise from white matter and cerebrospinal fluid masks. Motion artifacts were further detected using the ART-based scrubbing procedure, using the default thresholds of global-signal $Z$ value $= 9$ and subject motion $= 2$ mm. Next, motion parameters, white matter and cerebrospinal fluid were entered as potential confounders and were regressed out from each participant's BOLD signal, as were four time points detected by the ART-based scrubbing procedure. Finally, data were filtered using a band-pass filter (0.008–0.09 kHz) and linearly detrended. Fisher-transformed bivariate correlations were used for analyses.

### Statistical analyses

Demographic and psychological measures were compared between Sexual Assault and NEC groups as well between the PTSD and non-PTSD subgroups using Student's *t*-tests for continuous variables and $\chi^2$ tests for categorical variables, performed with SPSS v20 (IBM Corp., Armonk, NY, USA). Associations between Self-Blame and all other psychological variables were assessed via two-tailed correlational analyses in the Sexual Assault group only. These and all following between-group comparisons and correlational analyses were considered significant at a false discovery rate (FDR)-corrected threshold of $P < 0.05$.

Self-Blame-related whole brain GMV alterations were assessed via regression analyses in SPM8 with age, total intracranial volume and CTQ as covariates of no interest, conducted separately for Sexual Assault and NEC groups. Clusters were considered significant at an FDR-corrected cluster level threshold of $P < 0.05$. Significant cluster's mean GMV was calculated for each participant and exported to SPSS. In addition, for further evaluation of the functional relevance of a cluster identified with volumetric alterations, anatomical locations of this cluster were extracted from SPM and saved for rs-FC analysis. In order to assess the relationship of self-blame-related GMV alterations

with psychological symptomatology and peri- and postassault factors among the Sexual Assault group, two-tailed correlation analyses were conducted between an identified cluster's GMV and PCL, PHQ-9, BSI, PDEQ, Negative Reactions and Perceived Social Support. Next, because high correlations between these variables were expected, a stepwise regression analysis including these variables and Self-Blame was performed in SPSS. Although the effect size for Self-Blame in the second regression is expected to be biased by the first regression (Kriegeskorte *et al.*, 2009), this was necessary to ensure that the correlation of Self-Blame with a cluster's GMV was not a spurious one (driven by the correlation of one of the other psychological variables with the cluster's GMV). In addition, based on the strong association that has been observed between distorted cognitions-related brain activity and intrusion characteristics (Daniels *et al.*, 2011), regressions were repeated with participants' Intrusion subscale score instead of the total PCL score. A secondary analysis assessed whether an identified cluster's GMV differed between PTSD, non-PTSD and NEC groups, using one-way ANOVA followed by a Tukey post hoc test.

In order to explore the functional significance of a cluster with Self-Blame-related volumetric alterations to the manifestation of PTSD, this cluster was used as 'seed' and assessed for its rs-FC with all cortical and subcortical brain regions, anatomically defined with the Harvard-Oxford atlas implemented in CONN. Mean temporal correlations between the BOLD signals from the VBM-identified cluster and each region for each participant were calculated and exported to SPSS. Two-tailed *t*-tests were used to detect regions whose rs-FC with the seed cluster were significantly different between the PTSD and non-PTSD groups. For these regions, we computed across the Sexual Assault group the correlations between the rs-FC with the seed cluster and psychological symptomatology measures, peri- and postassault characteristics, and the seed cluster's GMV. In order to rule out the possible effects of childhood trauma and age on these associations, correlations were calculated while controlling for CTQ and age. For comparison purposes, these rs-FC values and correlations were also computed in the NEC group.

Finally, in order to assess whether Self-Blame-related GMV alterations and associated rs-FC patterns may account for the relations between self-blame and post-traumatic symptom severity, we conducted a mediation analysis using PROCESS (Hayes, 2013) for SPSS, controlling for CTQ and age. Given the previous evidence of strong association between cognitive distortions and intrusion, analyses were conducted for both total PCL score and the Intrusion subscale. Mediation was established if the bias corrected 95% confidence interval for the completely standardized indirect effect did not include zero and thus considered significant.

## Results

### Sample characteristics

Out of the 68 recruited participants, 6 NEC participants were excluded from analysis (2 due to neurological abnormalities discovered after scanning, and four due to above-cutoff PCL scores). Thus, the final sample consisted of 38 Sexual Assault and 24 NEC participants. Among these, one participant from the Sexual Assault group experienced anxiety inside the scanner and thus completed only the structural scan. One participant from each Sexual Assault and NEC groups had ADHD. The use of psychotropic medications was reported by seven participants in the Sexual Assault group and one in the NEC group. Among

the sexually assaulted women, mean time since last assault was 18.3 months (SD 10.0; range 2–36). Twenty-five participants (66%) in the Sexual Assault group scored above the 31 PCL-5 cutoff.

No differences were found in demographic variables between the Sexual Assault and NEC groups, as well as between the PTSD and non-PTSD subgroups (Table 1). As expected, scores on the PCL and its subscales, BSI and PHQ-9 differed both between the NEC and Sexual Assault groups and between the Sexual Assault subgroups. CTQ scores were higher in the Sexual Assault group, but did not differ between the PTSD and non-PTSD subgroups. Peri- and postassault psychosocial measures were also similar between the Sexual Assault subgroups, except for Self-Blame which was higher in the PTSD subgroup (Table 1).

### Associations between self-blame attributions, psychological symptomatology, childhood trauma and peri- and postassault characteristics

Self-Blame was strongly and positively associated with total PCL score ($r = 0.67$, $P < 0.001$) and its subscales: Intrusion: $r = 0.60$, $P < 0.001$, Negative Alterations in Cognitions and Mood: $r = 0.57$, $P < 0.001$, and Alterations in Arousal and Reactivity: $r = 0.55$, $P = 0.002$, but not with Avoidance ($r = 0.41$, $P = 0.105$). Self-blame was also positively correlated with PHQ-9 ($r = 0.60$, $P < 0.001$), BSI ($r = 0.55$, $P = 0.002$) and CTQ ($r = 0.51$, $P = 0.008$).

### Voxel-based morphometry

Among the Sexual Assault participants, Self-Blame was negatively associated with GMV in a medial occipital cluster, mostly comprising of the bilateral lingual gyrus and intracalcarine cortex (BA17/18, cluster size: $k = 1335$, peak MNI coordinates: $x = 0$, $y = -66$, $z = 6$, $t_{max} = 4.71$, $P = 0.005$; Figure 1). The cluster's GMV was negatively correlated with total PCL score and subscales (except Avoidance), as well as with BSI and Negative Reactions (Table 2). There were no positive associations between Self-Blame and GMV. When examined in NEC participants, victim-blame attributions were not associated with GMV decreases or increases.

A stepwise regression analysis among the Sexual Assault participants confirmed that Self-Blame was the strongest predictor of lingual cluster's GMV [$F(1, 36) = 19.99$, $P < 0.001$, $\beta = -0.603$], significantly explaining 36% of its variance ($R^2_{Adjusted} = 0.35$). Negative Reactions were also found to be a significant predictor, explaining an additional 9.1% of lingual GMV variance [$F_{change}(1, 34) = 5.65$, $P = 0.023$, $\beta = -0.328$]. In contrast, PCL, PHQ-9, BSI, PDEQ and Perceived Social Support did not predict GMV in the identified cluster. However, when Intrusion subscale was added to the regression, it became the strongest predictor [$F(1, 36) = 27.74$, $P < 0.001$, $\beta = -0.665$] significantly explaining 44% of variance in lingual GMV in Sexual Assault participants ($R^2_{Adjusted} = 0.43$), with Self-Blame significantly explaining additional 6.4% of variance [$F_{change}(1, 34) = 4.43$, $P = 0.043$, $\beta = -0.318$]. None of the variables significantly predicted GMV in the identified lingual cluster in the NEC group.

Comparison of GMV in the identified cluster between PTSD, non-PTSD and NEC groups revealed a significant difference [$F(2, 59) = 4.42$, $P = 0.016$; Figure 2A]. The PTSD subgroup had decreased lingual GMV compared with the non-PTSD subgroup ($P = 0.014$), with NEC not different from either subgroup ($P = 0.188$ and $P = 0.341$, respectively).

**Table 1.** Demographic and psychological characteristics of the sample

| Measures | Sexual assault (n = 38) | NEC (n = 24) | P | PTSD (n = 25) | Non-PTSD (n = 13) | P |
|---|---|---|---|---|---|---|
| Age, years | 25.31 ± 4.46 | 25.06 ± 4.01 | 0.826 | 24.72 ± 3.89 | 26.42 ± 5.39 | 0.271 |
| Handedness | | | | | | |
| Right | 29 (76%) | 18 (75%) | .906 | 17 (68%) | 12 (92%) | 0.095 |
| Left | 9 (24%) | 6 (25%) | | 8 (32%) | 1 (8%) | |
| Income | | | | | | |
| Below median | 28 (74%) | 19 (79%) | .749 | 19 (76%) | 9 (69%) | |
| Similar to median | 7 (18%) | 3 (13%) | | 4 (16%) | 3 (23%) | 0.784 |
| Above median | 2 (5%) | 2 (8%) | | 1 (4%) | 1 (8%) | |
| Education | | | | | | |
| High school | 9 (24%) | 5 (20%) | .757 | 7 (28%) | 2 (15%) | |
| Some academic | 16 (42%) | 12 (50%) | | 11 (44%) | 5 (39%) | 0.534 |
| B.A. | 10 (26%) | 4 (17%) | | 6 (24%) | 4 (31%) | |
| M.A. or above | 3 (8%) | 3 (13%) | | 1 (4%) | 2 (15%) | |
| PCL Total | 37.11 ± 17.56 | 8.29 ± 7.08 | **<0.0001** | 46.96 ± 12.03 | 18.15 ± 8.39 | **<0.0001** |
| Intrusion | 8.92 ± 4.98 | 1.88 ± 2.31 | **<0.0001** | 11.00 ± 4.05 | 4.92 ± 4.17 | **<0.001** |
| Avoidance | 4.68 ± 2.37 | 1.58 ± 1.98 | **<0.0001** | 5.56 ± 1.69 | 3.00 ± 2.65 | **<0.001** |
| Negative Alterations in Cognitions and Mood | 12.5 ± 7.11 | 3.04 ± 2.76 | **<0.0001** | 16.44 ± 5.25 | 4.92 ± 2.50 | **<0.0001** |
| Alterations in Arousal and Reactivity | 11.00 ± 6.69 | 1.79 ± 2.21 | **<0.0001** | 13.96 ± 6.11 | 5.31 ± 3.20 | **<0.0001** |
| PHQ-9 | 12.32 ± 3.57 | 6.87 ± 3.23 | **<0.0001** | 15.44 ± 5.46 | 6.31 ± 5.15 | **<0.0001** |
| BSI | 1.55 ± 0.37 | 0.80 ± 0.25 | **<0.0001** | 1.90 ± 0.62 | 0.82 ± 0.65 | **<0.0001** |
| CTQ | 63.82 ± 23.10 | 40.25 ± 11.16 | **<0.0001** | 67.36 ± 24.23 | 57.00 ± 19.86 | 0.097 |
| PDEQ | 24.82 ± 7.97 | 13.79 ± 5.32 | **<0.0001** | 26.44 ± 8.33 | 21.69 ± 6.42 | 0.041 |
| Self-Blame | 2.81 ± 1.10 | 1.55 ± 0.55 | **<0.0001** | 3.19 ± 1.08 | 2.07 ± 0.73 | **0.001** |
| Negative Reactions | 1.30 ± 0.64 | 0.55 ± 0.26 | **<0.0001** | 1.41 ± 0.66 | 1.09 ± 0.56 | 0.076 |
| Perceived Social Support | 3.40 ± 1.33 | 3.42 ± 1.53 | 0.476 | 3.16 ± 1.34 | 3.85 ± 1.21 | 0.066 |

Note: Uncorrected P-values are displayed, FDR-corrected $P<0.05$ are marked in bold.
BSI, Brief Symptom Inventory; CTQ, Childhood Trauma Questionnaire; NEC, non-exposed controls; PCL, PTSD Checklist; PDEQ, Peritraumatic Dissociative Experiences Questionnaire; PHQ-9, Patient Health Questionnaire – depression module.
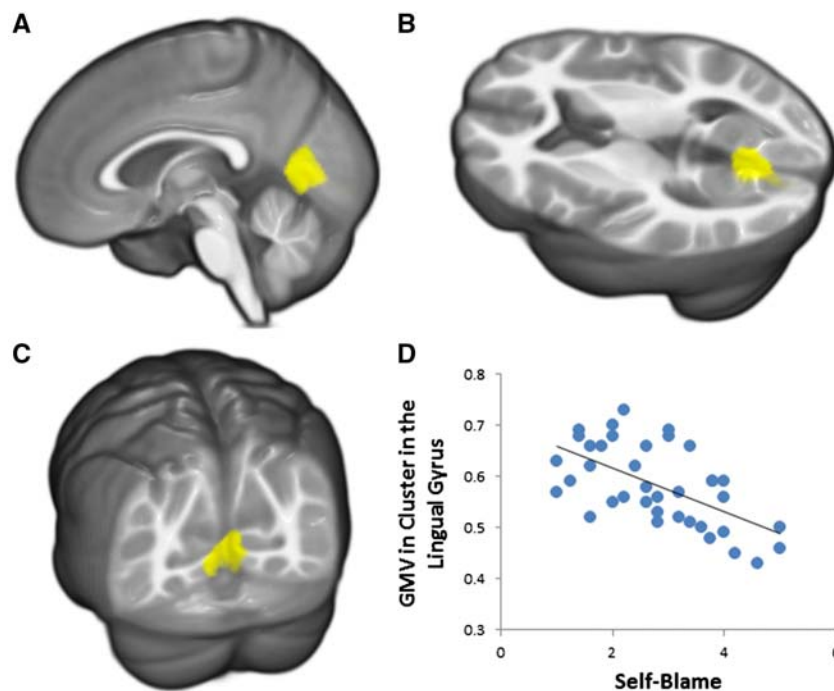


**Fig. 1.** GMV reduction associated with assault-related self-blame in sexually assaulted women. (A–C) Location of identified cluster in the lingual gyrus negatively correlated with Self-Blame, superimposed on the study T1 template. (D) Mean GMV in the identified cluster extracted for each participant plotted against Self-Blame scores.
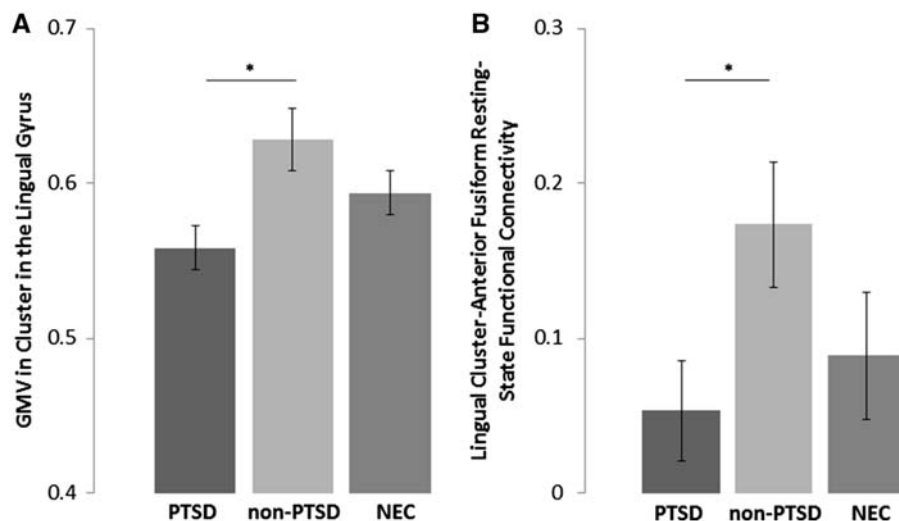
**Table 2.** Correlations between the GMV of the self-blame-related lingual cluster and its resting-state functional connectivity with the anterior fusiform cortex with psychological symptomatology and peri-and postassault characteristics

|  | Lingual GMV | | Lingual-fusiform rs-FC | |
|---|---|---|---|---|
|  | Sexual assault | NEC | Sexual assault | NEC |
| PCL—total | −0.57*** | 0.16 | −0.27 | 0.07 |
| Intrusion | −0.67*** | 0.09 | −0.39* | −0.03 |
| Avoidance | −0.15 | −0.15 | −0.29 | 0.15 |
| Negative alterations in cognitions and mood | −0.48** | 0.27 | −0.28 | −0.08 |
| Alterations in arousal and reactivity | −0.42* | 0.22 | −0.01 | 0.21 |
| PHQ-9 | −0.32 | 0.20 | −0.08 | 0.05 |
| BSI | −0.53** | 0.04 | 0.05 | 0.12 |
| PDEQ | −0.21 | −0.20 | −0.43** | 0.27 |
| Negative reactions | −0.51** | −0.09 | −0.21 | 0.18 |
| Perceived social support | 0.12 | −0.09 | −0.04 | −0.02 |

*P-value <0.05 is FDR-corrected.
**P-value <0.01 is FDR-corrected.
***P-value <0.001 is FDR-corrected.



**Fig. 2.** Between group comparisons of GMV in the self-blame-related lingual cluster and of its resting-state functional connectivity with the anterior fusiform. (A) Lingual cluster's GMV in PTSD, non-PTSD and NEC. (B) Lingual-fusiform functional connectivity in PTSD, non-PTSD and NEC. *P<0.05.

### Resting-state functional connectivity

Comparing rs-FC strength of the lingual cluster with other brain regions between the PTSD and non-PTSD subgroups revealed a significant difference in the lingual cluster rs-FC with the anterior division of the left temporal fusiform cortex [$t(35) = −2.22$, $P = 0.033$]. rs-FC between these regions was lower in the PTSD compared with the non-PTSD subgroups (Figure 2B). NEC did not differ from either subgroup ($P = 0.509$ and $P = 0.188$, respectively).

Among sexually assaulted participants, the lingual-fusiform rs-FC was inversely correlated with Intrusion ($r = −0.39$, $P = 0.021$), but not with other symptomatology measures (Table 2). Among the peri- and postassault characteristics, a negative correlation was observed with PDEQ ($r = −0.43$, $P = 0.009$). Finally, lingual-fusiform rs-FC was positively albeit non-significantly correlated with the lingual cluster's GMV ($r = 0.28$, $P = 0.104$). In contrast, among the NEC group, lingual-fusiform rs-FC was not significantly correlated with any of the assessed variables (Table 2).

### Mediation analysis of the association between self-blame and post-traumatic symptoms

This analysis revealed that the lingual gyrus GMV and lingual-fusiform rs-FC did not mediate the relationship between Self-Blame and total PCL score (95% CI=−0.016–0.214), but did mediate the relationship between Self-Blame and Intrusion score (95% CI=0.003–0.219).

### Discussion

This is the first study to assess brain alterations associated with self-blame attributions in individuals who were sexually assaulted. We found self-blame-related GMV alterations bilaterally in the lingual gyrus and adjacent intracalcarine cortex (referred to here as 'the lingual cluster'). GMV in this cluster was also predicted by intrusion and negative social reactions. Resting-state functional connectivity of this cluster with the left anterior temporal fusiform cortex (referred to here as 'lingual-

fusiform rs-FC') was found to significantly differ between the PTSD and non-PTSD Sexual Assault subgroups, and was inversely correlated with intrusion and with peritraumatic dissociation. Finally, lingual cluster's GMV and lingual-fusiform rs-FC mediated the association between self-blame and intrusion symptoms in the Sexual Assault group. Taken together, these results provide preliminary support for the involvement of the lingual gyrus and neighboring intracalcarine cortex in the neural pathway associating self-blame attributions and post-traumatic symptomatology related to intrusive memories.

Specifically, as previously reported (e.g. Frazier, 2000; Filipas and Ullman, 2006), we found that self-blame correlated strongly with psychological symptoms at adulthood, including PTSD and depression symptoms and general distress, as well as with childhood trauma. These findings support the central role assigned to self-blame in postassault coping and reinforce views that emphasize the explicit addressing of self-blame attributions in the treatment of this population (Moor and Farchi, 2011).

Our whole-brain analysis revealed that among women in the Sexual Assault group, assault-related self-blame was negatively correlated with GMV in a cluster mostly consisting of the lingual gyrus bilaterally. Importantly, although high correlations were observed between self-blame-related GMV and all the other psychological measures we assessed, a stepwise regression analysis revealed that GMV in the lingual cluster was strongly predicted by self-blame and intrusion symptoms, and, to a lesser extent, by negative social reactions. These findings are in agreement with Daniels *et al.*'s (2011) observation that among traumatized individuals, cognitive distortions were correlated with heightened activity during trauma recall in several structures including the lingual gyrus and adjacent regions, and that these activations were strongly associated with re-experiencing during the recall.

Previous neuroimaging studies provide support for an involvement of the lingual gyrus in retrieval and processing of negatively valenced memories (Taylor *et al.*, 1998; Kompus *et al.*, 2009; Kross *et al.*, 2009; Burianova *et al.*, 2010), and, among individuals with PTSD, in processing and imagery of the trauma memory (Rauch *et al.*, 1996; Bremner *et al.*, 1999, 2004; Huber *et al.*, 2001; Yang *et al.*, 2004). Furthermore, regional cerebral blood flow in this region was correlated with flashbacks intensity (Osuch *et al.*, 2001). These results suggest that the lingual gyrus plays a role in both voluntary and involuntary trauma recall. Our finding of intrusion being a potent predictor of lingual GMV among the sexually assaulted participants is consistent with these studies and extends the current functional literature to the realm of structural changes.

Although the present study cannot answer questions of causality, several lines of evidence suggest that the observed lingual GMV alterations may be an acquired rather than a pretraumatic characteristic. Evidence from animal studies show that both stress and high-dose glucocorticoid exposure facilitate structural reorganization in multiple cortical areas including the visual cortex (Maya Vetencourt *et al.*, 2008; Liston and Gan, 2011; Spolidoro *et al.*, 2011). In humans, ongoing atrophy in occipital regions among veterans with PTSD was associated with an increase in PTSD symptoms (Cardenas *et al.*, 2011), and lower volume and thickness of the lingual gyrus was found in adults who experienced childhood sexual abuse or witnessed domestic violence (Tomoda *et al.*, 2009, 2012). Further support for the hypothesis that the assault had a causal role in the alterations in the lingual cluster derives from the current observation that in NEC participants, lingual GMV was not predicted by any of the

psychological variables we assessed. The fact that lingual GMV differed between the PTSD and non-PTSD subgroups and that the NEC group was in between the two Sexual Assault subgroups, suggest that the observed alterations may reflect both vulnerability and resilience among assaulted women.

Taking into account the aforementioned functional data implicating the lingual gyrus in visual imagery of the trauma, together with findings of altered lingual gyrus activity in acutely and non-acutely traumatized individuals compared with NEC (New *et al.*, 2009; Nilsen *et al.*, 2016), the following cascade may be suggested. Exposure to trauma initiated structural and functional alterations of the lingual cluster; in assaulted women who were highly preoccupied with self-blame attributions, repeated engagement in visual imagery of the trauma might have excessively activated the lingual gyrus, thus further contributing to the structural alterations. The structural and functional alterations led, in turn, to elevation in intrusive memories.

This scenario is supported by our finding that the PTSD subgroup had lower levels of rs-FC between the lingual cluster and the left anterior temporal fusiform cortex, compared with the non-PTSD subgroup. Anatomically, these occipital and anterior temporal cortices are directly connected via the inferior longitudinal fasciculus (Catani *et al.*, 2003). The human anterior fusiform is considered a multimodal area, which, together with other temporal and inferior parietal regions, supports social cognition, language and mental simulations (Binder and Desai, 2011), and has been demonstrated to be crucially and selectively important for the processing and retrieval of semantic information (Binney *et al.*, 2010; Mion *et al.*, 2010).

The fusiform cortex has been implicated in the processing of trauma-related stimuli, and was reported to be hypoactivated in individuals with PTSD compared with non-PTSD during exposure to trauma reminders and retrieval of emotionally valenced stimuli (Bremner *et al.*, 1999, 2003; Shin *et al.*, 2001). Furthermore, such hypoactivation predicted subsequent increases in PTSD symptoms in patients with acute stress disorder (Cwik *et al.*, 2017). Thus, reduced lingual-fusiform rs-FC in the PTSD subgroup may reflect alterations in the neural circuitry involved in the processing of traumatic memories, and specifically, deficiencies in the narration of trauma memory and in the assignment of personal meaning, which are important processes in post-traumatic recovery (Cloitre *et al.*, 2011; Peri and Gofman, 2014). In contrast, more synchronized activity between these regions may enhance one's ability to integrate representations of the traumatic memory into her autobiographical knowledge, and to reevaluate distorted cognitions by exploring the meaning of the assault.

This interpretation is in line with the dual representation theory of PTSD and its revised model (Brewin *et al.*, 1996, 2010; Brewin, 2001, 2003), which conceptualize PTSD as a failure to associate low-level sensory representations of the traumatic event with the abstract declarative ones. As a result, the sensory representations are poorly contextualized, and involuntarily reactivated as flashbacks. In contrast, in non-PTSD individuals, the traumatic event is integrated with its autobiographical context, and semantic memory processes are activated to produce meaningful interpretations of the event (Brewin *et al.*, 2010). Brewin *et al.* further hypothesized that these processes are subserved by areas in the ventral visual stream, one of which is the fusiform cortex.

The hypothesis that coordinated activity of the lingual and fusiform regions is associated with improved narrated memory for the assault is supported by the negative correlations of the lingual-fusiform rs-FC with intrusive symptoms and with peritraumatic dissociation in the Sexual Assault group. This is

because theoretical and empirical accounts have consistently associated both unintentional re-experiencing and peritraumatic dissociation with fragmented and disorganized trauma memory (Ehlers and Clark, 2000; Murray *et al.*, 2002; Engelhard *et al.*, 2003; Halligan *et al.*, 2003).

Our findings that intrusion symptoms are mediated by lingual gyrus GMV and lingual-fusiform rs-FC may have therapeutic implications. Although a causal pathway cannot be determined, our findings suggest that plasticity in the lingual gyrus or in its functional connectivity with the anterior fusiform may lead to amelioration of intrusion symptoms, and, possibly, that such plasticity may be enhanced via changes to individuals' social cognitions associated with the assault. This suggestion is in accordance with Hauner *et al.* (2012) finding that successful exposure therapy is characterized by decreased lingual gyrus responsiveness to phobogenic images, and that post-therapy activity in this structure predicted later therapeutic outcomes. Future studies could examine the psychological and neural effects of the use of psychotherapeutic (such as exposure) and neurofeedback techniques to target the function of the lingual gyrus and its functional connectivity with the anterior fusiform in sexually assaulted individuals.

Our study has several limitations. As mentioned above, given the cross-sectional design of this study, alternative explanations for the order of observed effects cannot be ruled out. For example, it is possible that lingual GMV is a pre-trauma characteristic that influences both self-blame attributions and intrusion symptoms. Another limitation concerns the sample. A minority of the traumatized participants were using psychotropic medications, which might have influenced MRI indices of structure and function. Nonetheless, repeating our analyses after excluding these participants yielded the same pattern of results, with the main findings remaining unchanged. Last, all psychological and psychosocial measures relied on self-report, and could have been influenced by demand characteristics. In addition, we did not assess participants' memory characteristics and thus could not test this aspect of our hypothesis.

Despite these limitations, this study provides important preliminary converging evidence for the relevance of the ventral occipital cortex to both assault-related self-blame and post-traumatic re-experiencing in sexually assaulted individuals, and advocates for additional exploration of this relationship. By incorporating neuroimaging methods into the study of postassault self-blame, new insights about the determinants of victims' coping can be established and better-informed therapeutic techniques can be promoted, for the benefit of this population. Future studies may reveal whether the present findings can be generalized to victims of other traumatic experiences characterized with high levels of self-blame, such as following a car accident. Meanwhile, the present study provides evidence for the neuroanatomical injury associated with self-blame attributions, and emphasizes the need for a change in current social stand toward sexually assaulted individuals.

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