The Neural Correlates of Emotional Memory in Posttraumatic Stress Disorder

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Background: Posttraumatic stress disorder (PTSD) is marked by intrusive, chronic, and distressing memories of highly emotional events. Previous research has highlighted the role of the amygdala and its interactions with the hippocampus in mediating the effect of enhanced memory for emotional information in healthy individuals. As the functional integrity of these regions may be compromised in PTSD, the current study examined the neural correlates of emotional memory in PTSD.

Methods: We used functional magnetic resonance imaging and an event-related subsequent memory recognition paradigm to study amygdala and hippocampus activation in 18 individuals with PTSD and 18 trauma-exposed non-PTSD control participants.

Results: Memory enhancement for negative, relative to neutral, pictures was found across all subjects, without significant differences between groups. Relative to the trauma-exposed non-PTSD group, the PTSD group showed exaggerated amygdala activation during the encoding of negative versus neutral pictures. This effect was even more pronounced when the analysis included data from only pictures that were subsequently remembered 1 week later. In the PTSD group, degree of amygdala activation during the encoding of negative versus neutral pictures was positively correlated with hippocampal activation and current PTSD symptom severity. The PTSD group also showed exaggerated hippocampal activation in response to negative pictures that were remembered versus forgotten. Finally, hippocampal activation associated with the successful encoding of negative relative to neutral pictures was significantly greater in the PTSD group.

Conclusions: Exaggerated amygdala activation during the encoding of emotionally negative stimuli in PTSD is related to symptom severity and to hippocampal activation.

Key Words: Amygdala, emotion, hippocampus, magnetic resonance imaging, memory, neuroimaging, posttraumatic, stress disorders

Emotionally arousing stimuli or events are typically remembered better than neutral ones (1). This phenomenon can be beneficial, as it helps us to remember sources of potential danger and avoid them in the future. However, memories of highly emotional events can become intrusive, chronic, and extremely distressing, as in the case of individuals with posttraumatic stress disorder (PTSD). The mediating neuroanatomy of enhanced memory for emotional information has been studied extensively in rodents and healthy humans, but little is known about the neural correlates of this phenomenon in individuals with PTSD.

Neuroscience research has revealed that the effect of emotion on memory is mediated, at least in part, by activation in the amygdala and by interactions between the amygdala and hippocampus (2–9). These two structures are anatomically connected, and efferent projections from the amygdala to the hippocampus are thought to play a role in the emotional modulation of memory (3,10). While the amygdala appears to be responsible for assigning emotional significance to stimuli, the hippocampus assigns contextual meaning to them (11). Basic research has highlighted the role of norepinephrine and glucocorticoids in mediating the effects of emotion on memory via their actions on these two structures (2,12,13).

Importantly, amygdala activation has been shown to be increased in PTSD during the recollection of traumatic events and the viewing of trauma-unrelated emotionally negative stimuli (14–19). Furthermore, degree of amygdala activation is positively correlated with PTSD symptom severity (e.g., 17,18,20,21). Given this exaggerated amygdala activation, individuals with PTSD may show enhanced memory for novel emotional stimuli. Indeed, this behavioral finding has been documented in PTSD ([11,22–24]; but see [25,26]).

The effect of emotional arousal on memory may also depend on amygdala-hippocampal interactions (5,27). Although some studies have reported diminished hippocampal activation in PTSD (26,28–32), others have reported exaggerated hippocampal activation (31,33–38). The direction of hippocampal abnormalities in PTSD may depend on the type of task and/or analysis performed (39). Admon et al. (40) recently reported a delicate interplay between the amygdala and hippocampus in response to stressful life events. While prestress amygdala activation was associated with greater symptoms after stress, such symptoms also depended on the degree of hippocampal plasticity following stress, highlighting the need to study both structures in trauma-exposed individuals.

Recently, subsequent memory paradigms have highlighted the role of the amygdala and hippocampus in the emotional modulation of memory in healthy humans (6,41). In these paradigms, brain activation during encoding is analyzed based on whether items are later remembered or forgotten (42,43). The term difference due to memory (Dm) refers to greater brain activation for remembered than forgotten items and is thought to reflect successful encoding processes (44,45). In healthy individuals, amygdala and hippocampal/parahippocampal activation are associated with successful subsequent memory of emotional relative to neutral stimuli (41,43).

Whether the neural correlates of the successful subsequent memory of emotional stimuli differ in individuals with PTSD is unclear. To date, only two studies have used subsequent emotional memory paradigms to examine amygdala and hippocampal activation in PTSD. Neither study explored a direct functional relationship...
between these structures. Dickie et al. (46) examined subsequent memory for fearful and neutral faces in PTSD. They reported a significant positive correlation between PTSD symptom severity and amygdala activation in response to successfully remembered fearful versus neutral faces. However, because all subjects had PTSD, whether those with PTSD differ from trauma-exposed individuals without the disorder is unknown. More recently, Thomaes et al. (37) used a subsequent memory paradigm to study nine individuals with PTSD associated with childhood abuse and multiple comorbid psychiatric disorders. Relative to the control group, the PTSD group showed significantly greater hippocampal activation in response to subsequently remembered deeply encoded negative words compared with a low-level baseline (but not when compared with neutral words). However, this study had a small sample size, used a trauma-unexposed control group, and reported no group differences in the amygdala.

In the current study, we examined the neural correlates of the emotional modulation of memory in PTSD. Specifically, we used functional magnetic resonance imaging (fMRI) to study amygdala and hippocampal function during the encoding of negative, compared with neutral, pictures in trauma-exposed individuals with and without PTSD. A recognition memory test was administered outside of the scanner 1 week later. We predicted that both groups would remember more negative pictures than neutral pictures, but given that PTSD is primarily a disorder of intrusive emotional memories, we predicted that the PTSD group would remember more negative versus neutral pictures than the trauma-exposed non-PTSD (TENP) group. Additionally, we predicted that the PTSD group would show exaggerated amygdala and hippocampal activation during the encoding of negative versus neutral pictures, especially those that were subsequently remembered. Our prediction of exaggerated amygdala activation in PTSD was based on similar previous findings in the literature (e.g., 47,48). Our prediction of greater hippocampal activation in PTSD was based on a previous finding (37) and on our reasoning that enhanced memory for negative versus neutral pictures in PTSD should be accompanied by greater hippocampal activation. Furthermore, based on the findings of subsequent memory studies (6,41), we predicted that fMRI signal changes in the amygdala and hippocampus would be positively correlated, especially in the PTSD group. Finally, based on previous findings (17,18,20,21,46), we predicted that fMRI signal changes in the amygdala would be positively correlated with symptom severity in the PTSD group. Due to limited prior evidence, we had no predictions regarding a correlation between hippocampal activation and symptom severity.

Methods and Materials

Participants

Participants were 42 individuals who reported experiencing criterion A traumatic events, including motor vehicle accidents, sexual or physical abuse, assault, and witnessing serious injury/death. According to the Clinician-Administered PTSD Scale for DSM-IV (CAPS) (49), 21 had current PTSD and 21 never had PTSD (TENP group). Participants were right-handed (50) with no history of head injury, neurological disorders, or other major medical conditions. No participants were pregnant or using psychotropic or cardiovascular medication at the time of study. Two PTSD participants were excluded from analyses due to excessive head movement during scanning. Three TENP participants were excluded due to below-chance memory performance. One PTSD participant failed to return for the memory test. The final sample consisted of 18 (3 male) in the PTSD group and 18 (6 male) in the TENP group (Table 1).

The Structured Clinical Interview for DSM-IV (51) was used to assess other Axis I disorders. Current comorbidity included major depression (n = 4 PTSD, n = 1 TENP), panic disorder (n = 2 PTSD), and specific phobia (n = 1 PTSD). The Partners Healthcare System (Boston, Massachusetts) Institutional Review Board approved this study. Written informed consent was obtained from each participant.

Materials

Stimuli consisted of 80 negative, 80 neutral, and 80 positive pictures selected from the International Affective Picture System (IAPS) (52). The negative and positive pictures did not differ on normative arousal ratings. Pictures within each type were divided into two separate sets (A and B); one set was used for encoding and the other as foils in the subsequent recognition test. Sets were counterbalanced across subjects in each group and were matched for valence and arousal ratings within each picture type (negative, neutral, positive).

Experimental Design

Functional magnetic resonance imaging data were gathered while participants viewed the stimuli, which were presented in an event-related paradigm via the stimulus-presentation program MacStim (MacStim 3.2.1; Darby, White Ant Occasional Publishing; West Melbourne, Australia) using a Sharp Notevision6 (XG-NV6XU) LCD projector (Osaka, Japan). Each full-color PICT file was presented for 5 seconds followed by a fixation cross ranging in duration from 1 to 11 seconds. The duration of each of the two functional scans was 8 minutes, 8 seconds. Immediately following the scan, participants completed the Beck Depression Inventory (BDI) to quantify depressed mood (53).

One week later, participants returned to the laboratory. At this visit, they completed a surprise recognition memory test outside of the scanner. Participants viewed all 240 pictures (sets A and B), only half of which they saw during fMRI scanning. Participants indicated whether they had seen the picture during scanning (old) or whether it was a novel picture that they had not previously seen (new). They also reported their confidence in each selection on a three-point scale. After completing the memory test, participants

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**Table 1. Demographic and Psychometric Data**

<table>
<thead>
<tr>
<th></th>
<th>PTSD Mean</th>
<th>PTSD SD</th>
<th>PTSD Range</th>
<th>TENP Mean</th>
<th>TENP SD</th>
<th>TENP Range</th>
<th>r(34)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>28.2</td>
<td>7.8</td>
<td>19–46</td>
<td>26.2</td>
<td>4.5</td>
<td>20–36</td>
<td>.914</td>
<td>.369</td>
</tr>
<tr>
<td>Education</td>
<td>15.0</td>
<td>2.3</td>
<td>10–18</td>
<td>16.8</td>
<td>1.6</td>
<td>14–20</td>
<td>2.765</td>
<td>.009</td>
</tr>
<tr>
<td>CAPS Current</td>
<td>57.7</td>
<td>15.8</td>
<td>34–85</td>
<td>3.6</td>
<td>5.9</td>
<td>0–24</td>
<td>13.620</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>CAPS Lifetime</td>
<td>86.1</td>
<td>12.0</td>
<td>66–105</td>
<td>11.3</td>
<td>8.8</td>
<td>0–33</td>
<td>21.254</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>BDI</td>
<td>15.3</td>
<td>12.0</td>
<td>0–41</td>
<td>1.6</td>
<td>1.7</td>
<td>0–5</td>
<td>4.810</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

BDI, Beck Depression Inventory; CAPS, Clinician Administered PTSD Scale; PTSD, posttraumatic stress disorder; TENP, trauma-exposed non-PTSD.
again viewed the 120 pictures they originally saw during scanning and rated each picture on 9-point Likert scales of arousal (1 = calm, 9 = excited) and valence (1 = negative, 5 = neutral, 9 = positive).

fMRI Procedures

Scans were obtained from a Symphony/Sonata 1.5 Tesla whole body scanner (Siemens Medical Systems, Iselin, New Jersey) with a three-axis gradient head coil. After shimming procedures were performed (54), high-resolution, three-dimensional, magnetization-prepared rapid acquisition gradient-echo scans (repetition time/echo time/flip angle = 2730 msec/3.39 msec/7°; slice thickness = 1.33 mm) were collected. Functional magnetic resonance imaging (blood oxygen-level dependent [BOLD]) (55) images were acquired using a gradient echo T2*-weighted sequence (repetition time/echo time/flip angle = 2000 msec/40 msec/90°). Functional images were collected in 26 coronal slices angled perpendicular to the anterior commissure–posterior commissure line (slice thickness = 4 mm, skip = 1 mm; voxel size = 3.1 × 3.1 × 4 mm).

Behavioral Analysis

Hits (number of pictures correctly remembered 1 week later), misses, false alarms, correct rejections, and the sensitivity index (d’) for each picture type (negative, neutral, positive) were calculated for each participant. The d’, a signal detection statistic, was included because it takes into account both hits and false alarms (d’ = Z[hit] – Z[false alarm]). Hit rate and d’ data were analyzed using two separate 2(group: PTSD, TENP) × 3(picture-type: negative, neutral, positive) analyses of variance (ANOVAs). However, preliminary analysis of behavioral data revealed no significant differences on any of the memory measures between positive and neutral pictures either between or within diagnostic groups. Additionally, positive pictures were reported as significantly less arousing than negative pictures, despite initial matching of pictures based on IAPS normative ratings. Given this, we limit our reported fMRI analyses to comparisons between negative and neutral pictures only. Hit rate and d’ data were then analyzed using two separate 2(group: PTSD, TENP) × 2(picture-type: negative, neutral) ANOVAs.

fMRI Data Analysis

Image preprocessing and statistical analyses were performed using SPM2 (http://www.fil.ion.ucl.ac.uk/spm/Welcome Department of Imaging Neuroscience, London, United Kingdom). Functional data were motion-corrected, coregistered to subjects’ anatomical images, spatially normalized into a standard stereotaxic space (Montreal Neurological Institute [MNI]), and spatially smoothed using a 4-mm Gaussian kernel. The BOLD responses were modeled as events convolved with the canonical hemodynamic response function in SPM2. For each condition (negative, neutral, positive), all trials were averaged to estimate BOLD responses. The voxelwise statistical parametric maps resulting from the clusters of significantly activated voxels. Extracted data were then submitted to independent sample t tests (in SPSS; SPSS Inc, Chicago, Illinois) to determine whether fMRI signal changes differed between PTSD and TENP groups. This method of analysis (21) creates an unbiased functional region of interest (ROI) and thus permits the use of a .05 significance threshold for the subsequent (two-tailed) between-group comparisons on extracted data. This method also benefits from the ability to use a larger sample size when creating the functional ROI. Whole-brain voxelwise between-group analyses were also conducted, and those results were nearly identical to those reported herein. Additionally, whole-brain voxelwise analyses confirmed that BOLD signal changes in our a priori regions of interest were indeed in the same direction in both groups, thus further supporting the use of this method of analysis.

For all contrasts in which activation was found in both amygdala and hippocampus, bivariate correlations were run between the extracted values from these regions within groups to test our hypotheses concerning the relationship between activation of these two structures. Extracted values from the amygdala and hippocampus were also tested for correlations with CAPS and BDI scores within groups.

Statistics

The voxelwise statistical parametric maps resulting from the one-sample t tests (main effect of condition) were inspected for activations in a priori regions of interest. Given our strong, directional a priori hypotheses, we used a significance threshold of p < .001, uncorrected (z score ≥ 3.09) for activations in these regions. Because the procedure of correcting p values based on the region size is biased toward finding significance in relatively small structures, we chose to employ the above-stated constant significance threshold (17,48). For regions about which we had no a priori prediction, we used a more conservative constant significance threshold of p < .00001, uncorrected (z score ≥ 4.27) (31,48).

Results

Valence/Arousal Ratings

Two separate 2(group: PTSD, TENP) × 2(picture type: negative, neutral) ANOVAs were used to analyze valence and arousal ratings. Valence. The main effect of picture type was significant [F(1,34) = 89.77, p <.001]. Negative pictures were rated signifi-
cantly lower in valence than neutral pictures. The main effect of group and the picture type by group interaction were not significant (p's > .33).

**Arousal.** The main effect of picture type was significant \[F(1,34) = 109.45, p < .001\]. Negative pictures were rated significantly more arousing than neutral pictures. The main effect of group and the picture type by group interaction were not significant (p's > .15).

**Memory Performance**

Two separate 2(group: PTSD, TENP) × 2(picture type: negative, neutral) ANOVAs were used to analyze hits and d'. With regard to hits, the main effect of picture type was significant \[F(1,34) = 71.85, p < .001\]. Negative pictures were remembered significantly better than neutral pictures (Figure S1 in Supplement 1). The main effect of group and the picture type by group interaction were not significant (p's > .25). The same pattern of results was found when assessing d': only the main effect of picture type was significant \[F(1,34) = 9.299, p = .004\] (all other p's > .80).

There were no significant correlations between memory measures (hits or d' for either valence category) and CAPS or BDI scores.

**fMRI Results**

**Negative Versus Neutral.** Collapsing across groups, the negative versus neutral comparison revealed significant activation in both the amygdala and hippocampus bilaterally and in other unpredicted regions (Table S1 in Supplement 1). Functional magnetic resonance imaging data were extracted from significant functional activations (ROIs) in the amygdala and hippocampus. Independent sample t tests on the differences scores of the extracted data revealed significantly greater right amygdala activation in the PTSD compared with the TENP group (Figure 1).

In the PTSD group, significant positive correlations were found between BOLD signal increases in the right hippocampus (MNI = 22, −28, −6) and in both the left \(r(16) = .564, p = .015\; [\text{MNI} = −18, −4, −14]\) and right \(r(16) = .513, p = .029\; [\text{MNI} = 18, −4, −12]\; Figure 2A) amygdala. In the TENP group, no such correlations were found between BOLD signal in the right hippocampus and either the left \(r(16) = .037, p = .883\) or right \(r(16) = −.005, p = .983\) amygdala. Additionally, PTSD subjects' current CAPS scores were positively correlated with activation in the left \(r(16) = .563, p = .015; \text{Figure 2B}\) but not right \(r(16) = .339, p = .169\) amygdala. Analogous correlations were not found in the TENP group [left:

**Negative > Neutral: PTSD Group Only**

![Figure 1](image1.png)  
**Figure 1.** The functional image on the left displays activation in the right amygdala (Montreal Neurological Institute = 26, −2, −18, z = 4.52) in the negative > neutral condition collapsing across groups. The bar graph on the right shows magnetic resonance signal change in this region relative to fixation baseline by diagnostic status. Fix, fixation baseline; Neg, negative; Neut, neutral; PTSD, posttraumatic stress disorder; TENP, trauma-exposed non-PTSD.

![Figure 2](image2.png)  
**Figure 2.** (A) Correlation between blood oxygen-level dependent activation in the right hippocampus (Montreal Neurological Institute [MNI] = 22, −28, −6) and right amygdala (MNI = 18, −4, −12) in the negative > neutral contrast in the posttraumatic stress disorder group only \(r = .513, p = .029\). (B) Correlation between blood oxygen-level dependent activation in the left amygdala (MNI = −26, −4, −22) and current Clinician-Administered PTSD Scale for DSM-IV scores in the negative > neutral contrast in the posttraumatic stress disorder group only \(r = .563, p = .015\). CAPS, Clinician-Administered PTSD Scale for DSM-IV; PTSD, posttraumatic stress disorder.

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Negative Remembered Versus Neutral Remembered. Collapsing across groups, we found significantly greater activation in response to subsequently remembered negative pictures than to subsequently remembered neutral pictures in the left hippocampus and bilateral amygdala. Extracted values in these functionally defined ROIs were submitted to two separate independent sample t tests. Activation in the right \( t(34) = 2.122, p = .041 \) and left \( t(34) = 2.053, p = .048 \) amygdala was significantly greater in the PTSD group than the TENP group (Figure 3). In the PTSD group only, a significant positive correlation was found between activation in the left hippocampus and the left amygdala \( r(16) = .614, p = .007; \) Figure 4A). The analogous correlation was not found in the TENP group \( r(16) = .082, p = .746 \). No significant correlations were found between activation in these NegR versus NeutR ROIs and CAPS or BDI scores in the PTSD group.

Negative Remembered Versus Negative Forgotten. Collapsing across groups, we found significantly greater activation in response to negative pictures that were subsequently remembered than to those that were forgotten in the right amygdala and hippocampus bilaterally. No between-group differences in amygdala activation were found. Right hippocampal activation was greater in the PTSD group than in the TENP group \( t(34) = 1.919, p = .064 \).

Discussion
Our subsequent recognition memory paradigm revealed the expected memory enhancement for negative versus neutral pictures after a 1-week retention interval. Contrary to our hypotheses,
this effect was not significantly greater in the PTSD group than the TENP group. This finding is contrary to previous reports of better memory for emotional versus neutral material in PTSD (11,22–24) but is consistent with the findings of Bremner et al. (26), who found no evidence for enhanced memory for emotional versus neutral word pairs in PTSD. Our 1-week retention interval may not have been long enough to reveal behavioral between-group differences (both groups remembered over 80% of the negative stimuli after 1 week). Additionally, group differences in memory may be more likely to emerge when the stimuli are more directly related to participants’ traumatic events. However, a lack of behavioral differences between groups may actually make the between-group differences in brain activation more interpretable.

Our findings of exaggerated amygdala activation in the PTSD group relative to the TENP group during the encoding of negative versus neutral pictures (especially those that were subsequently remembered) add to the existing literature documenting exaggerated amygdala responses in PTSD (e.g., [14,15,17]; for a review see [56]). Additionally, our correlation between amygdala activation during the encoding of negative stimuli and symptom severity in the PTSD group parallels that of Dickie et al. (46). Positive correlations between symptom severity and amygdala activation have also been reported in previous studies (17,18,20,21).

The degree of hippocampal activation in both the negative versus neutral and negative remembered versus neutral remembered contrasts did not differ between groups. Similarly, Tomaes et al. (37) did not find enhanced hippocampal activation in response to negative words in PTSD when using neutral pictures as a comparison condition. However, our finding of a significant positive correlation between amygdala and hippocampal activation in these contrasts only in the PTSD group supports the idea of exaggerated functional connectivity between these two structures in PTSD. Additionally, hippocampal activation was greater in the PTSD group than the TENP group when directly assessing remembered (vs. forgotten) negative pictures.

Consistent with previous research (41), greater bilateral amygdala activation was found in response to the negative Dm than to the neutral Dm in both groups pooled together. However, this amygdala activation did not differ between diagnostic groups. This lack of a between-group difference may help account for the lack of a behavioral difference between groups. However, we did find greater hippocampal activation in PTSD subjects than in TENP subjects in response to the negative Dm versus neutral Dm contrast. Splitting subjects by diagnosis revealed that only the PTSD group displayed significant hippocampal activation in response to this contrast. This finding lies in contrast to that of Dolcos et al. (41), who...
reported enhanced hippocampal activation in response to the emotional Dm versus neutral Dm in a sample of healthy individuals. However, Dolcos et al. (41) used both positive and negative pictures and a cued-recall task and a 45-minute retention interval, whereas the current study used a recognition test and a 1-week retention interval. These paradigm differences may have accounted for differences in results, as these two types of memory are believed to be mediated by different regions within the medial temporal lobe, with recall requiring more hippocampal involvement than recognition (57).

Limitations and Conclusions

In the current study, we excluded individuals who were taking psychiatric medications and who had current comorbid disorders other than depression and anxiety disorders. While comorbid depression in the PTSD group may be viewed as a limitation, given the high comorbidity rates between PTSD and depression (58), excluding PTSD patients with depression would have severely compromised external validity. Nearly all analyses remained significant when data from participants with comorbid depression were temporarily removed. In addition, in the PTSD group, amygdala activation was correlated with CAPS scores but not BDI scores. Nevertheless, future studies of emotional memory in PTSD should employ psychiatric control groups. It is also important to note that the restricted range of CAPS scores in the TENP group could explain the lack of significant correlations with BOLD signal changes in this group. Finally, using the main effect of condition maps to define functional ROIs cannot reveal areas of functional activation in which the direction of effects differ between the PTSD and TENP groups. However, whole-brain voxelwise analyses confirmed that BOLD signal changes in our a priori regions of interest were indeed in the same direction in both groups, thus supporting use of this method.

In conclusion, we document for the first time that amygdala activation related to the subsequent memory of negative, trauma-unrelated versus neutral pictures is exaggerated in PTSD. Additionally, enhanced hippocampal activation in PTSD is related to the successful encoding of negative pictures, and functional coupling between the amygdala and hippocampus during the encoding of negative pictures also appears exaggerated. Together, these findings point toward an interaction between these two structures in the pathophysiology of this disorder.

That these functional abnormalities were not accompanied by relatively enhanced memory for negative versus neutral stimuli suggests that fMRI measures may be more sensitive to group differences than behavioral measures in this paradigm. Because our comparison group was exposed to criterion A trauma but did not have PTSD, trauma exposure cannot account for the group differences reported herein. Future studies using longitudinal or twin designs should attempt to determine whether these functional abnormalities are acquired characteristics of PTSD or rather represent risk factors for the development of PTSD after psychological trauma.

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Supplementary material cited in this article is available online.


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