Medial PFC Damage Abolishes the Self-reference Effect

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Abstract

■ Functional neuroimaging studies suggest that the medial PFC (mPFC) is a key component of a large-scale neural system supporting a variety of self-related processes. However, it remains unknown whether the mPFC is critical for such processes. In this study, we used a human lesion approach to examine this question. We administered a standard trait judgment paradigm [Kelley, W. M., Macrae, C. N., Wyland, C. L., Caglar, S., Inati, S., & Heatherton, T. F. Finding the self? An event-related fMRI study. *Journal of Cognitive Neuroscience, 14,* 785–794, 2002] to patients with focal brain damage to the mPFC. The self-reference effect (SRE), a memory advantage conferred by self-related processing, served as a measure of intact self-processing ability. We found that damage to the mPFC abolished the SRE. The results demonstrate that the mPFC is necessary for the SRE and suggest that this structure is important for self-referential processing and the neural representation of self. ■

INTRODUCTION

When individuals contemplate information in relation to themselves—for example, daydream about future successes or reflect on a negative event that transpired at work—they engage in a cognitive process of self-reference. Several psychological studies suggest that self-referential processing, associated with the building of a self-concept, is valuable and may be evolutionarily adaptive. Self-referential processing plays a role in the consolidation of memory (Rogers, Kuiper, & Kirker, 1977) and in the perception of social cues (Lombardo et al., 2010; Eisenberger & Lieberman, 2004) and is more generally integral to emotional control, reappraisal, and effective psychotherapeutic interventions (Mansell, 2011). The ability to evaluate whether information is self-relevant is especially crucial in the social realm where deficiencies can have significant consequences for normal social interaction. For example, individuals with autism have a variety of self-referential processing deficits (i.e., self–other distinctions) that are thought to contribute to impairments in interpersonal functioning (Lombardo et al., 2010).

Neuroimaging research has implicated a network of brain regions, including the medial PFC (mPFC), across a variety of self-referential processing paradigms, from tasks engaging mind wandering to tasks requiring personality trait evaluation (Christoff, Gordon, Smallwood, Smith, & Schooler, 2009; Moran, Macrae, Heatherton, Wyland, & Kelley, 2006; Northoff et al., 2006; Schmitz, Kawahara-Baccus, & Johnson, 2004; Kelley et al., 2002). The mPFC has also been associated with the representation of value (Chib, Rangel, Shimojo, & O'Doherty, 2009) and emotional processing (Damasio, 1999) and might play a critical role in the representation of self-relevance or personally salient information (e.g., one's own name or hometown; Northoff & Panksepp, 2008; Schmitz & Johnson, 2007). Also, functional neuroimaging studies indicate that the mPFC is involved in the ability to represent and comprehend the mental states of others, known as "theory of mind" (ToM; e.g., Gallagher & Frith, 2003). Although these neuroimaging findings imply that the mPFC contributes to ToM abilities, neuropsychological evidence suggests that the mPFC region may not be critical. For example, two lesion studies found that patients with mPFC damage were unimpaired on both basic and higher-order ToM tasks, such as comprehending faux pas (Umeda, Mimura, & Kato, 2010; Bird, Castelli, Malik, Frith, & Husain, 2004). However, Umeda and colleagues (2010) found that patients with mPFC damage reported postmorbid changes in personality and social functioning. Interestingly, these patients endorsed "autistic" personality traits, which were associated with deficits in social interaction.

Returning to self-referential processing more specifically, this capacity appears to confer a special memory advantage, a phenomenon known as the "self-reference effect" (SRE; Symons & Johnson, 1997). For example, personality traits processed for self-relevance, that is, in relation to oneself (e.g., "Am I a generous person?") are better remembered than traits processed for other-relevance (e.g., "Is Sally a generous person?"; Rogers et al., 1977). Neuroimaging research has shown that the mPFC is active during this type of self-referential processing, that is, judging personality traits (Macrae, Moran, Heatherton, Banfield, & Kelley, 2004; Schmitz et al., 2004; Kelley et al., 2002). Moreover, mPFC activity is parametrically modulated by self-relevance (Moran et al., 2006), and its activity is predictive of subsequent memory for self-relevant traits (Macrae et al., 2004).

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Although previous work has established a correlation between self-referential processing and the mPFC, it remains an open question whether the mPFC is critical for such processing. In regard to the SRE in particular, we predicted that if the mPFC is critical for this effect, then patients with mPFC damage should fail to show it. We tested this prediction in this study.

We studied six patients with mPFC damage. The mPFC patients were compared with 8 patients with brain damage outside the putative self-referential brain network (brain-damaged comparisons [BDC]; matched with the mPFC patients on age and education) and 15 healthy participants (normal comparisons [NC]). In a personality trait judgment task (see Methods), participants were instructed to judge personality traits in three encoding conditions: self (Does this trait describe you?), other (Does this trait describe Oprah Winfrey?), and case (Is this trait capitalized?). Subsequently, a recognition task was administered, in which a set of personality traits (90 old, 90 new) were presented one at a time, and participants were asked to make old/new judgments. A corrected recognition score (proportion of hits − proportion of false alarms) was calculated for each condition (self, other, case). The SRE was calculated by subtracting the corrected recognition score for traits presented in the other condition from the corrected recognition score for traits presented in the self condition (*self − other*).

METHODS

Participants

mPFC Group

Six patients with lesions to the mPFC (five bilateral and one unilateral right) were selected from the Cognitive Neuroscience Patient Registry of the University of Iowa's Department of Neurology (see Table 1 for demographic information of all participant groups). Patient groups were defined based on functional neuroanatomical criteria. Specifically, the mPFC group was chosen based on a previous study using a similar version of the self-referential processing task used in this study (Kelley et al., 2002). In the previous study, functional imaging results revealed activity in the mPFC in the right hemisphere with Montreal Neurological Institute (MNI) coordinates ($x = 10, y = 52, z = 2$; putatively corresponding to BA 10). All six subjects included in the mPFC group had lesions that overlapped with this right mPFC region (Figure 1).

BDC Group

Eight patients with brain damage were selected; their lesions involved cortices outside the putative self-referential processing networks, for example, default mode network or cortical midline structures (Buckner, Andrews-Hanna,

Table 1. Demographic and Neuropsychological Variables

	MPFC, n = 6	*BDC, n = 8*	*NC, n = 12*
Age (years)	58.6 (11.1)	55.7 (17.8)	70.1 (10.8)[a]
Education (years)	13.5 (1.5)	16.3 (2.7)	15.5 (3.0)
Sex	3M, 3F	4M, 4F	9M, 6F
Handedness	6R	8R	15R
Chronicity (years)	16.7 (8.6)	12.3 (12.8)	N/A
Laterality	5B, 1R	4B, 3L, 1R	N/A
WAIS-III: Verbal IQ	108.0 (20.1)	113.4 (12.3)	118.8 (12.2)
Wechsler Memory Scale-III: General Memory Index	100.4 (16.2)	97.7 (11.1)	N/A
Wechsler Memory Scale-III: Working Memory Index	109.8 (15.0)	108.1 (11.9)	110.4 (6.7)
Rey Auditory–Verbal Learning Test: Trial 5, 30-min recall	11.3 (2.8), 8.3 (2.4)	11.8 (2.4), 7.5 (3.9)	13.2 (1.4), 9.7 (3.3)
Complex Figure Test: 30-min recall	20.7 (7.4)	16.1 (7.1)	21.1 (5.3)
Token Test	43.8 (0.4)	41.6 (3.3)	N/A
Wide Range Achievement Test-Revised: Reading Standard Score	102.0 (13.1)	105.1 (10.3)	108.3 (8.1)
Beck Depression Inventory-II	4.0 (4.1)	4.9 (7.5)	3.1 (3.1)

Demographic and neuropsychological variables, means (*SD*s), are reported for all subjects, except for lesion-specific measures (laterality and chronicity), which are only reported for the patient groups. Chronicity is the time between lesion mPFC onset and experimental testing. There were no significant differences between groups for education, chronicity, or neuropsychological measures (ANOVA, each $p > .05$).

M = male; F = female; R = right; L = left; B = bilateral; N/A = not applicable.

[a]There was a significant difference between groups for age (ANOVA; $F(2, 26) = 4.3, p = .02$), as the NC group was older (see Results for statistical comparisons).

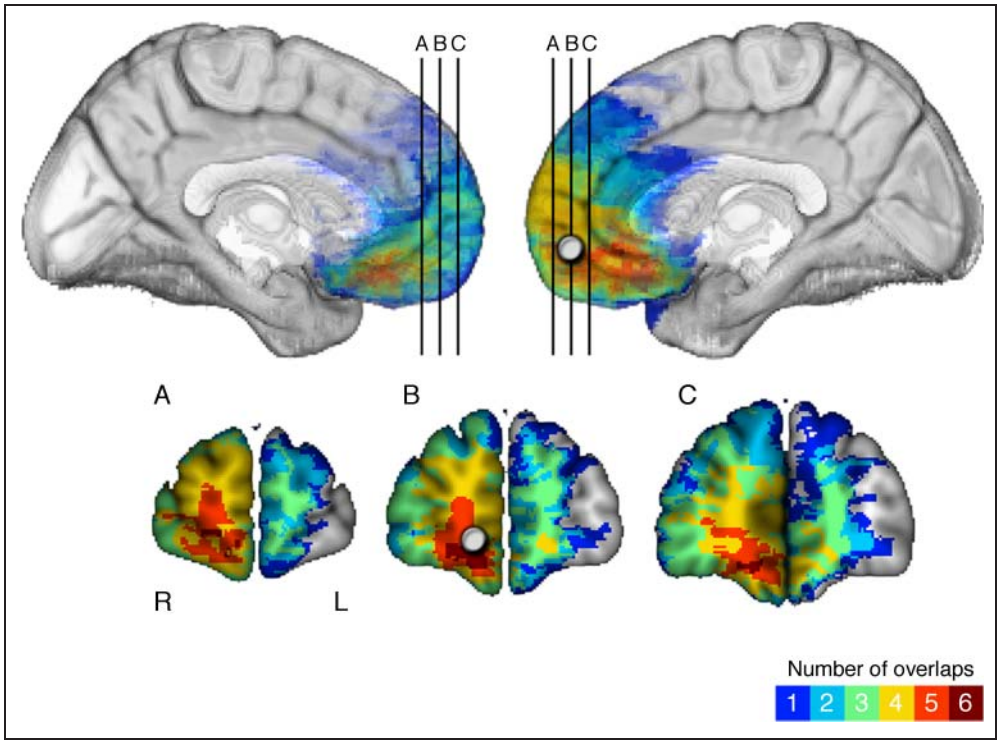

Figure 1. Lesion overlap map for the mPFC group ($N = 6$), displayed on the MNI template (MNI-152). The maximum overlap is 6 (see color scale). Midsaggital views are presented for both hemispheres. Coronal slices are in radiological convention (left on the right). A gray dot is plotted in the right hemisphere at the location of the ROI from Kelley et al. (2002), where neural activity was associated with encoding during self trials ($x = 10$, $y = 52$, $z = 2$). The ROI overlies the region of maximum lesion overlap for the mPFC group.

& Schacter, 2008; Northoff et al., 2006). The BDC group was chosen to match the mPFC group on average age and education. The lesions were bilateral in three cases (with damage primarily to medial and lateral occipital, temporal, and parietal regions) and unilateral in five cases (with damage to medial and lateral occipital, temporal, and insular regions; Figure 2).

General Inclusion Criteria

All patients met the standard inclusion criteria for the Iowa Patient Registry, including stable (nonprogressive), circumscribed brain lesions and no history of dementia or psychiatric disorder. All subjects were characterized neuropsychologically and neuroanatomically in the chronic epoch

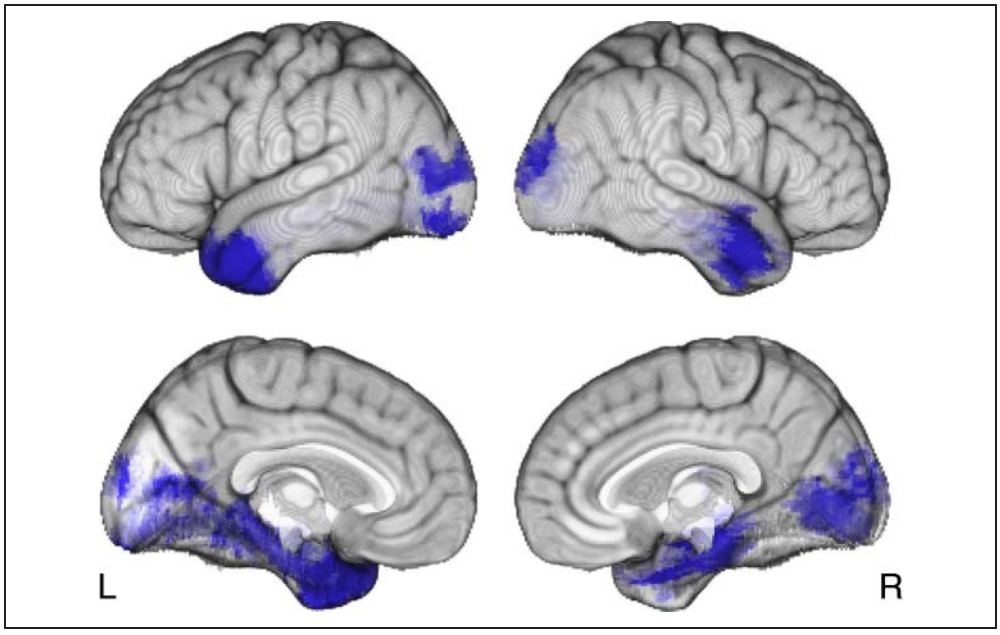

Figure 2. Lesion coverage map for the BDC group ($N = 8$), displayed on the MNI template (MNI-152). The map was thresholded at a value of 1 (represented in blue) so that all areas of lesion in the BDC group would be represented. The lesions primarily affect the occipital and temporal cortices and do not overlap with the mPFC.

(>3 months post onset of lesion) according to the standard protocols of Benton Neuropsychology Laboratory (Tranel, 2009) and the Laboratory of Human Neuroanatomy and Neuroimaging (Frank, Damasio, & Grabowski, 1997; Damasio & Damasio, 1989).

NC Group

Fifteen healthy adults with no history of psychiatric or neurological illnesses were studied. The participants in this NC group were not explicitly matched to brain-damaged patient groups on demographic factors (see Results). All participants gave informed consent according to a protocol approved by the institutional review board of the University of Iowa.

Detailed Neuroanatomical Description of the mPFC Group

The functional imaging literature (e.g., Kelley et al., 2002) has suggested an anatomo-functional entity that corresponds broadly to the mPFC as a topographic ensemble (i.e., both ventral and dorsal components of the mPFC). Accordingly, the patients selected for our study had damage that encompassed the mPFC, as described above (see Figure 1). Many of these patients have damage that extends into the ventromedial PFC. We note that the ROI from Kelley et al. (2002; Figure 1) is really at the boundary between ventromedial PFC and dorsomedial PFC, in a location maximally covered by our sample. Thus, our mPFC patient sample was appropriate for testing the hypothesis we derived from the functional imaging literature, and the fact that the maximal overlap in the mPFC lesions was centered squarely on the coordinates published by Kelley et al. provides definitive support for this claim.

Task Procedure

Personality Trait Judgment Paradigm (SRE Task)

A set of 270 trait adjectives (normed from Anderson, 1968) was selected and counterbalanced for syllable number, word length, and valence (135 negative traits, 135 positive traits). We used a slightly modified version of the trait judgment task used in Kelley et al. (2002; e.g., changed the person used for the other condition). There were three different conditions in which participants were asked to make trait judgments: (1) self (e.g., Does this trait describe you?), (2) other (e.g., Does this trait describe Oprah Winfrey?), and (3) case (e.g., Is this trait capitalized?). On each trial, a fixation cross was presented for 500 msec, followed by simultaneous presentation of a cue denoting the condition (e.g., self) and a trait adjective. Subjects made yes/no responses for each trial. RT was not collected, as RTs in brain-damaged participants tend to be highly variable and not reliably informative. Detailed instructions and practice blocks were given before the experimental task to assure that the subjects understood and were comfortable with the task. Two blocks consisting of 45 trials each (15 self, 15 other, and 15 case) were given. The order of the trials was randomized, and the trait adjectives for each condition were counterbalanced. To assess the SRE, subjects performed an unexpected recognition memory task completed after a 15-min retention interval. In the recognition task, subjects were presented with 180 trait adjectives, including 90 "old" (from the encoding trials) and 90 "new" traits. A fixation was presented before each word for 500 msec. Next, subjects were presented with the trait adjectives, one at a time, for 2000 msec each. Subjects were instructed to make "yes" or "no" responses based on whether they remembered the word from before.

Neuropsychological Variables

All patients were tested on various neuropsychological measures, including intelligence (WAIS-III: Verbal IQ), general and working memory (Wechsler Memory Scale-III: General Memory Index and Working Memory Index), verbal memory (Rey Auditory–Verbal Learning Test: Trial 5 and 30-min delayed recall), visuospatial memory (Complex Figure Test: 30-min delayed recall), language (Multilingual Aphasia Examination Token Test), reading ability (Wide Range Achievement Test-Revised: Reading Standard Score), and general mood (Beck Depression Inventory-II). These neuropsychological variables were measured to allow the investigation of potential confounds in the SRE due to group differences in intelligence, memory, language, reading ability, or mood (see Table 1).

Data Analysis

SRE Calculation

We calculated the SRE for each subject. The SRE was calculated based on the methods used in Kelley et al. (2002), which were consistent with standard measures of memory recognition. First, proportion of hits (correct recognition) and proportion of false alarms (FA; incorrect recognition of new words) were calculated for each condition. For example, a hit for the self condition corresponded to the correct recognition of a trait that the subject encoded in relation to themselves during the self condition ("I am organized."). Second, the proportion of hits from the self condition (e.g., self hits/30) minus the proportion of false alarms (pFA = FA/90) was subtracted from the proportion of hits from the other condition minus the proportion of false alarms. The SRE calculation is illustrated by the following equation: (p*self* hits − pFA) − (p*other* hits − pFA).

Lesion Analysis Procedures

Lesion Mapping Procedures

All subjects underwent structural scanning procedures. MRIs were acquired in a 1.5-T General Electric Sigma scanner

with a 3-D spoiled gradient recall sequence yielding 1.5-mm contiguous T1-weighted coronal cuts. If subjects were unable to undergo MRI scanning, CT data were collected. Lesion maps were generated using the MAP-3 method (Fiez, Damasio, & Grabowski, 2000; Frank et al., 1997), in which the boundaries of the lesions of a given subject are visually identified on MRI or CT scans and manually transferred onto a normal reference brain (PC local standard space, resolution = 0.94 × 0.94 × 1.6 mm) based on the delineation of homologous anatomical landmarks. Lesion delineation and transfer were done using Brainvox (Frank et al., 1997). Lesion overlap maps (NMaps) were created by summing the 3-D MAP-3 binary lesion mask for each subject within the mPFC patient group ($n = 6$).

RESULTS

SRE Analysis

In support of the main prediction, there was a significant effect of group for the SRE ($F(2, 26) = 6.16$, $p = .007$), and the mPFC group showed a significantly lower SRE than the BDC (rank sum = 24, $p = .005$) and NC (rank sum = 33, $p = .01$) groups (Figure 3). These effects could be specifically attributed to recognition deficits in the self condition for the mPFC group (mPFC $M = 0.15$, BDC $M = 0.33$, NC $M = 0.30$), as recognition performance was approximately equivalent across all groups in both other (mPFC $M = 0.14$, BDC $M = 0.17$, NC $M = 0.16$) and case (mPFC $M = -0.01$, BDC $M = -0.02$, NC $M = 0.04$) conditions. In fact, the SRE was virtually abolished in the mPFC group, as the patients recognized virtually the same numbers of traits for the self versus other conditions. There were no significant group differences in neuropsychological factors such as intelligence, general memory, language, and mood (each $p > .05$, for all contrasts) that could have accounted for these results (see Table 1). Although there were no significant differences between patient groups (mPFC and BDC) in any demographic variables (age, education, or chronicity; each $p > .05$), the NC group was significantly older than both the mPFC (rank sum = 37, $p = .026$) and BDC (rank sum = 63, $p = .036$) groups (see Table 1). However, the significant effect of group for the SRE remained even after controlling for age and education ($F(3, 24) = 3.08$, $p = .035$), suggesting that these results were not due to differences in demographic factors.

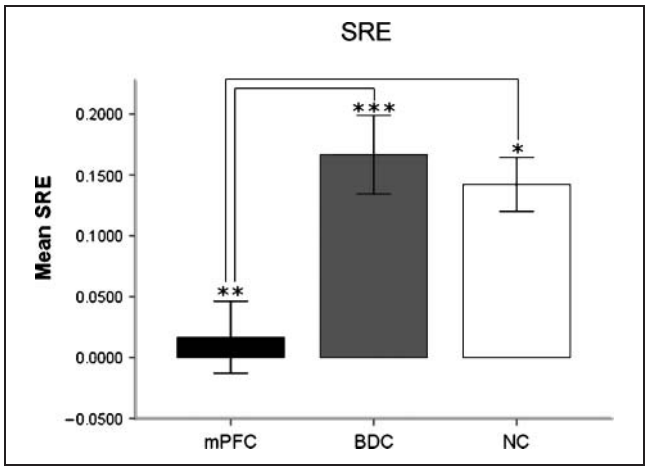

Figure 3. SRE group averages. The average SRE is significantly lower in the mPFC group than in both the BDC and NC groups. *$p = .01$; **$p = .007$; ANOVA, ***$p = .005$, pairwise comparisons. Error bars correspond to 1 *SEM*.

Lesion Volume and SRE

To address the potential contribution of lesion volume to the main effect of Group for SRE, we examined the correlation between total lesion volume and SRE for both patient groups (BDC and mPFC; each group had an extreme outlier removed for this analysis). The correlation between Lesion Volume and SRE was not significant (Kendall's $\tau = -0.36$, $p = .11$). In a subsequent within-group analysis, there was no significant correlation between Lesion Volume and SRE in either the mPFC (Kendall's $\tau = 0$, $p = 1$) or BDC (Kendall's $\tau = -0.18$, $p = .62$) group.

DISCUSSION

The results support the hypothesis that the mPFC plays a critical role in mediating the self-reference advantage in memory, and they are consistent with neuroimaging studies that have pointed to the importance of the mPFC in the SRE (Moran et al., 2006; Northoff et al., 2006; Macrae et al., 2004; Schmitz et al., 2004; Kelley et al., 2002). They are also compatible with the claim that the mPFC may facilitate the representation and the detection of self-relevance (Northoff & Panksepp, 2008; Schmitz & Johnson, 2007). Our study focused specifically on the interaction between self and memory, as opposed to self-processing in general. Thus, our study does not address the broader issue of whether the mPFC would be necessary for self-processing independent of memory. Future studies could investigate the critical role of the mPFC region in other types of self-processing (e.g., self-agency).

Although the mPFC has been the focus of numerous studies on the self, debate remains regarding the unique role of the mPFC in self-processing (Lou, Luber, Stanford, & Lisanby, 2010; Legrand & Ruby, 2009). Functional neuroimaging research has implicated a network of subcortical and cortical structures in self-referential processing, including, most consistently, the posterior cingulate cortex (PCC) and inferior parietal lobule (IPL; e.g., Lou et al., 2010; Northoff et al., 2006; Northoff & Bermpohl, 2004). For example, the PCC was implicated in an intracranial EEG study, where activity in the PCC was specific to the processing of self-relevant stimuli (Dastjerdi et al.,

2011). In a TMS study, disruption of IPL activity during self-referential processing reduced the normal SRE (Lou et al., 2010). Together, these studies suggest that (in addition to the mPFC) the medial and lateral parietal cortices may contribute to self-processing. Future investigations could examine whether the IPL or the PCC are necessary and/or sufficient for self-referential processing.

The ability to detect and encode information for self-relevance might contribute not only to the formation of a self-concept but also more broadly to psychological and social functioning. Across a variety of psychopathological conditions and personality disorders, self-referential processing appears to be dysfunctional, making it a major target for psychotherapy (Mansell, 2011). Recent research in patients with autism provides evidence for an association between aberrant mPFC activity and both self-referential processing deficits and impaired social functioning (Lombardo et al., 2010). Our results may also help to explain well-documented social impairments in patients with damage to the ventromedial PFC region (e.g., Anderson, Barrash, Bechara, & Tranel, 2006), as such damage could disrupt the normal functioning of self-referential processing network and the neural representation of self (Northoff & Panksepp, 2008; Schmitz & Johnson, 2007; Damasio, 1999).

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