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Research report

Transient medial prefrontal perturbation reduces false memory formation

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ABSTRACT

Knowledge extracted across previous experiences, or schemas, benefit encoding and retention of congruent information. However, they can also reduce specificity and augment memory for semantically related, but false information. A demonstration of the latter is given by the Deese–Roediger–McDermott (DRM) paradigm, where the studying of words that fit a common semantic schema are found to induce false memories for words that are congruent with the given schema, but were not studied. The medial prefrontal cortex (mPFC) has been ascribed the function of leveraging prior knowledge to influence encoding and retrieval, based on imaging and patient studies. Here, we used transcranial magnetic stimulation (TMS) to transiently perturb ongoing mPFC processing immediately before participants performed the DRM-task. We observed the predicted reduction in false recall of critical lures after mPFC perturbation, compared to two control groups, whereas veridical recall and recognition memory performance remained similar across groups. These data provide initial causal evidence for a role of the mPFC in biasing the assimilation of new memories and their consolidation as a function of prior knowledge.

1. Introduction

Knowledge acquired across prior experiences, incorporated into schemas, can exert a strong influence on the processing of incoming information. Schemas provide an internal structure or scaffold for the assimilation of congruent information, enhancing memory for related, congruent information (Anderson, 1981; Bartlett, 1932). This enhancement occurs at the cost of memory for episodic detail resulting in reduced memory specificity (Friedman, 1979; Goodman, 1980). As every episode is partly consistent and partly inconsistent

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with prior knowledge, competing schematic and episodic elements are weighed to integrate new memories with existing knowledge (Alba & Hasher, 1983). As such, expectations raised by schemas during memory formation can proactively interfere with memory, causing intrusions of schema-consistent, but false information at retrieval (Tuckey & Brewer, 2003).

A striking example of strong schema-based expectations leading to false memory intrusions is provided by the Deese–Roediger–McDermott (DRM) paradigm (Roediger & McDermott, 1995). Studying lists of words that are semantically organized improves later recall compared to random lists of words (Bower, Clark, Lesgold, & Winzenz, 1969), but learning these lists can also provoke false memories for words that are congruent with the semantic schema but actually never studied. Here, the weighing of schematic memories is increased by study words ('rest', 'awake', 'pillow') that are semantically associated to a non-studied critical lure word ('sleep') that may therefore be more likely to intrude at retrieval (Roediger & McDermott, 1995; Stadler, Roediger, & McDermott, 1999).

The medial prefrontal cortex (mPFC) is proposed to play a critical role in leveraging prior knowledge to influence online encoding and retrieval. Whereas memory for specific episodes and general knowledge are thought to be supported by the medial temporal lobe and posterior neocortical areas, the mPFC and its connectivity with the medial temporal lobe supports the integration of novel memories into pre-existing networks of knowledge (Preston & Eichenbaum, 2013; van Kesteren, Ruiter, Fernández, & Henson, 2012). Indeed, previous animal and human studies have related medial prefrontal processing to a schema-congruent learning benefit (Liu, Grady, & Moscovitch, 2016; Tse et al., 2011; van Kesteren, Rijpkema, Ruiter, Morris, & Fernández, 2014; van Kesteren et al., 2013), as well as reduced memory specificity (Berkers, Klumpers, & Fernández, 2016; Xu & Südhof, 2013). The mPFC could play a role in evaluating the overlap of new memories with prior knowledge, thereby weighting the influence of schematic and episodic memory components. Thus, when encoding a list related to a 'sleep' schema, a strong weighting of prior knowledge in the processing of incoming experiences causes proactive interference during later retrieval. As a result, schema-based expectations promote recall of related words that had not been encountered before, resulting in faulty judgments about whether or not a semantically related word had been seen previously.

The mPFC has been implicated in memory encoding and retrieval after long-term consolidation (Takashima et al., 2006), but lesions to this region by themselves often do not lead to severe global impairments in declarative memory, in contrast with lesions to the medial temporal lobe (Melo, Winocur, & Moscovitch, 1999; Scoville & Milner, 1957). Specifically, damage to mPFC is reported to lead to an increase in confabulation (Schnider, 2003), and a reduction of the influence of schema on recognition memory (Spalding, Jones, Duff, Tranel, & Warren, 2015). In the DRM task, lesions situated in the mPFC covering Brodmann areas 32, 12 and 10 have been related to a reduction in false recall, whereas veridical recall remains unaffected (Warren, Jones, Duff, & Tranel, 2014). These results indicate that the mPFC contributes to false memory formation and retrieval. Lesion studies, however, have the caveat that they can be confounded by brain plasticity following damage, leading to modification of brain connections, reduced spatial specificity due to damage or disruption of additional brain regions, and reduced cognitive specificity due to global impairments in cognitive functioning. Imaging studies have linked activity during encoding to subsequent false recognition only, but not false recall, which might involve different processes (Staresina & Davachi, 2006). Furthermore, these studies have linked encoding activity to subsequent false recognition in regions across the lateral, dorsal and ventromedial prefrontal cortex (Cabeza, Rao, Wagner, Mayer, & Schacter, 2001; Kim & Cabeza, 2007; McDermott, Gilmore, Nelson, Watson, & Ojemann, 2017).

Here, we sought to investigate further the possible role of the anterior part of the mPFC in the false memory effect in a non-clinical setting using a temporary experimental perturbation of mPFC processing that obviates the confounding effect arising from neural plasticity. We used offline transcranial magnetic stimulation (TMS) to transiently perturb mPFC processing before participants performed the DRM-task. Previous studies have shown that by stimulating the dorsolateral prefrontal cortex it is possible to manipulate memory encoding of both verbal (Javadi & Walsh, 2012; Javadi, Cheng, & Walsh, 2012; Sandrini, Cappa, Rossi, Rossini, & Miniussi, 2003) and non-verbal material (Epstein, Sekino, Yamaguchi, Kamiya, & Ueno, 2002; Floel et al., 2004). However, to our knowledge, there has not been any brain stimulation study reported to date investigating the role of the mPFC in memory formation. Following studies that have perturbed deeper regions in the mPFC using an angled, figure-of-eight coil (Klucharev, Munneke, Smidts, & Fernández, 2011; Rushworth, Hadland, Paus, & Sipila, 2002), we aimed to perturb processing in a more anterior region of the mPFC (Brodmann area 10) with a continuous theta-burst stimulation (cTBS) protocol. This repetitive TMS protocol has been shown to induce a transient decrease in neural excitability for up to an hour (Huang, Edwards, Rounis, Bhatia, & Rothwell, 2005; Huang, Rothwell, Chen, Lu, & Chuang, 2011). Our focus was on the possibility of altering the probability of 'false memories' induced by the DRM protocol. Following mPFC perturbation or a control stimulation procedure, participants encoded and recalled DRM-lists, followed by a recognition test. Based on prior literature, we predicted that cTBS directed at the mPFC would result in a reduction in false recall and/or recognition of critical lures compared to a behavioral and stimulation control.

2. Materials and methods

2.1. *Participants*

The present study was part of a larger study with multiple tasks. For one task not reported here there was a gender-specific hypothesis, the results of which will be reported elsewhere. Therefore, all participants included were female. Fifty-one participants were pre-screened for contraindications for participation in a TMS-study and were found to fit the criteria for inclusion (no history of participant or family of epilepsy, prior head trauma, medication, implanted

neurostimulators, cochlear implant, pacemaker or intracardial wires, intake of psycho-active substances). Ten participants were excluded: one participant was excluded because we could not measure the active leg motor threshold (see below), two participants experienced discomfort during stimulation, two participants withdrew participation, two participants were on medication, and one participants had an excessive intake of alcohol (7 units) in the 24 h before returning for the second stimulation session, and two participants were excluded because the achieved stimulation intensity differed substantially in relation to the target stimulation intensity (less than 70% of target intensity, see Procedure Intake Session below for specifics about the method used to determine stimulation intensity). This resulted in 42 participants being included in the two stimulation groups (21 in the mPFC stimulation group and 21 in the Cz stimulation group). Furthermore, 46 age-matched participants were included in a group receiving no stimulation (behavioral control group).

Participants have been shown to be able to suppress false memories when they have knowledge about the false memory effect (McCabe & Smith, 2002). In a debriefing questionnaire administered after completing the experiment (see Supplemental Materials & Methods), participants were asked whether they knew prior to or during the task that the word lists were created as to induce false memories for words that had not been presented. Participants that answered that they knew about the purpose of the task before, or while listening to the word lists were then excluded from analysis. Overall, data from 28 subjects were excluded according to these criteria. These exclusions were unevenly distributed across participants (4 out of 21 for the group that received stimulation to the mPFC, 7 out of 21 from the group that received stimulation to the Cz, and 18 out of 46 participants in the behavioral control group), which might be due to random sampling effects since the same questionnaire and instructions were used for all participants. This resulted in a total of 59 remaining participants: 17 in the mPFC stimulation group (experimental group), 14 participants in the Cz stimulation group (TMS control group), and 28 participants in the behavioral control group. For one participant in the latter group, data for the recognition task was lost due to a technical error, but the recall data was included. As previous studies have reported a strong relation between performance on the DRM task and sleep (Diekelmann, Born, & Wagner, 2010; Payne et al., 2009), data was also obtained on 'Sleep Quality' in the night before the experimental session, and the current state of 'Drowsiness' and 'Feeling Rested' (see Supplemental Materials & Methods for the questionnaire, and Table 1 for specifics of the three groups). All participants received payment or course credits for their contribution. All participants had normal or corrected-to-normal vision and provided written informed consent according to the guidelines of the local ethics committee (Commissie Mensgebonden Onderzoek region Arnhem-Nijmegen, The Netherlands).

2.2. *Procedure Intake Session*

TMS protocols were applied using a biphasic pulse configuration using a MagVenture MC-B70 Butterfly coil connected to a MagPro-X100 stimulator (MagVenture, Farum, Denmark). This coil uses two angled windings (inner diameter: 27 mm, outer diameter 97 mm) to increase the effectiveness of stimulating relatively deep brain areas. Stimulation intensity was defined by measuring active motor thresholds during the intake session. Specifically, the toe/leg representation in the primary motor cortex (Cz) is located at a comparable depth level in the interhemispheric wall compared to the target location in the mPFC (see also Klucharev et al., 2011). Therefore, stimulation intensity was defined as 80% of the measured active leg motor threshold. Further, to ensure that stimulation intensity was within established safety guidelines (Rossi, Hallett, Rossini, & Pascual-Leone, 2009), the active motor threshold was also obtained for the right hand as a reference. As such, if 80% of the active leg motor threshold exceeded 120% of the active hand motor threshold, the intensity was adjusted as to fall below 120% of the active hand motor threshold (in each stimulation group one participant was excluded where the achieved stimulation intensity with this procedure was lower than 70% of target intensity, see Participants). Electrodes were placed on both the first dorsal interosseous (FDI) muscle of the right hand (belly-tendon montage), and the tibialis anterior (TA) muscle of the right leg (placed collaterally at approximately 1/3 of the length of the muscle from the top). The electromyography-signal was measured at a 1 kHz sampling rate and bandpass filtered at 1–1000 Hz using an EKIDA DC amplifier (Ekida GmbH). During stimulation of the lateral hand area of the primary motor cortex, the active motor threshold was established as the minimum stimulation intensity of single pulses that produced

Table 1 – Information on age, stimulation parameters used and sleep characteristics on the night prior to the experimental session for the three groups. All subjects were female. Descriptive statistics are reported as means with standard error of mean (S.E.M.) in brackets. Sleep Quality, Feeling Rested and Drowsiness were rated by participants on a six-point scale. Stimulation parameters are reported as a percentage of maximum stimulation output. The last column reports F-statistics on group differences (*p*-value between brackets).

	Behavioral control N = 28	MPFC stimulation N = 17	Cz stimulation N = 14	Group differences
Age (yrs)	21.71 (.53)	21.59 (.57)	23.29 (.65)	2.104 (.13)
Sleep Quality	5.86 (.25)	5.29 (.19)	5.00 (.23)	.873 (.42)
Drowsiness	2.96 (.24)	2.94 (.35)	2.86 (.40)	.028 (.97)
Feeling Rested	4.39 (.22)	4.71 (.17)	4.64 (.33)	.539 (.59)
AMT hand (%)	N.A.	26.94 (1.09)	25.07 (1.17)	1.352 (.25)
AMT leg (%)	N.A.	36.88 (1.53)	35.56 (2.27)	.328 (.57)
TMS Intensity (%)	N.A.	28.35 (1.10)	26.43 (1.42)	1.193 (.28)

a liminal EMG response in 50% of trials during isometric contraction of the FDI muscle. The active motor threshold was similarly defined for the midline toe/leg area of primary motor cortex (Cz) during isometric contraction of the TA muscle.

Coil position for the repetitive stimulation was established by drawing locations using the 10–20 system on a tightly-worn swim cap. The target stimulation was delivered to target the mPFC, whereas control stimulation was delivered to target the midline toe/leg representation of the primary motor cortex (Cz). First, the Cz was outlined with a removable marker as half the distance from left to right tragus, as well as from nasion to inion. Second, the mPFC stimulation site was defined as two-thirds the distance from vertex to nasion (see Fig. 1). Therefore, the coil was positioned tangentially to the skull, and oriented with a 90° angle with reference to the sagittal midline, with the handle either pointing to left-lateral or right-lateral direction (counterbalanced across subjects). This orientation is optimal for inducing an electrical field in the lateral-medial direction, perpendicular to the cortical layers in the medial wall of the prefrontal cortex (Laakso, Hirata, & Ugawa, 2013). The stimulation protocol consisted of a 40 sec continuous theta-burst train (Huang et al., 2005), which consists of bursts of three pulses administered at a rate of 50 Hz, that are themselves repeated at a rate of 5 Hz. The stimulation protocol was administered over either mPFC or Cz location (see Table 1 for stimulation parameters of the two stimulation groups).

2.3. DRM task

In the DRM task, participants are presented with lists of 15 semantically associated words that are all related within each list to an omitted critical lure word. At free recall and recognition, these critical lures are then often falsely recalled and recognized (critical intrusions) as having been part of the studied word list (Roediger & McDermott, 1995). Twenty-two English DRM lists were selected based on reported norms from a larger set of 36 lists (Stadler et al., 1999). Specifically, lists were selected that in at least 30% of all participants had evoked false recall of the critical lures. The lists were translated into Dutch. An initial pilot study ($N = 6$) was performed to exclude lists that did not produce false recall of critical lures. The lists were read out by the experimenter (R.M.W.J.B.) at a rate of one word per second, followed immediately by a prompt for the recall of the list words. Four lists were rejected that did not prompt false recall of critical lures in the pilot subjects. Next, the resulting eighteen lists were read out by the experimenter at a speed of one word per second and recorded using the free audio editing program Audacity (http://audacity.sourceforge.net). From each list, the first, eighth and tenth words were included for the recognition test (54 old items), intermixed with a further 18 critical lures and 54 semantically unrelated lures taken from the discarded DRM lists.

2.4. Procedure experimental session

The stimulation protocol was administered while the participants remained seated in a comfortable chair in the stimulation lab. Next, participants were relocated to a behavioral lab, explained the purpose of the task through verbal and written instructions (see SI Materials & Methods) and given a headset to wear (mean delay between TMS and task: 349.09 sec, S.E. = 11.59 sec). The audio files for each of the eighteen lists were played in randomized order to the participants through a headset. After each list, the participants were instructed to verbally recall as many words as possible from the list (see SI: Materials and Methods). Recall was self-paced, there was no time limit, and participants pressed a button to hear the next list when they thought they would not be able to recall any more words from the list. Recall was scored by the experimenter present in the same room, but also recorded with a microphone throughout, and scored by a secondary rater who was blind to group membership of participants.

After completion of recall for all eighteen lists, instructions for the recognition task were given and the task was started

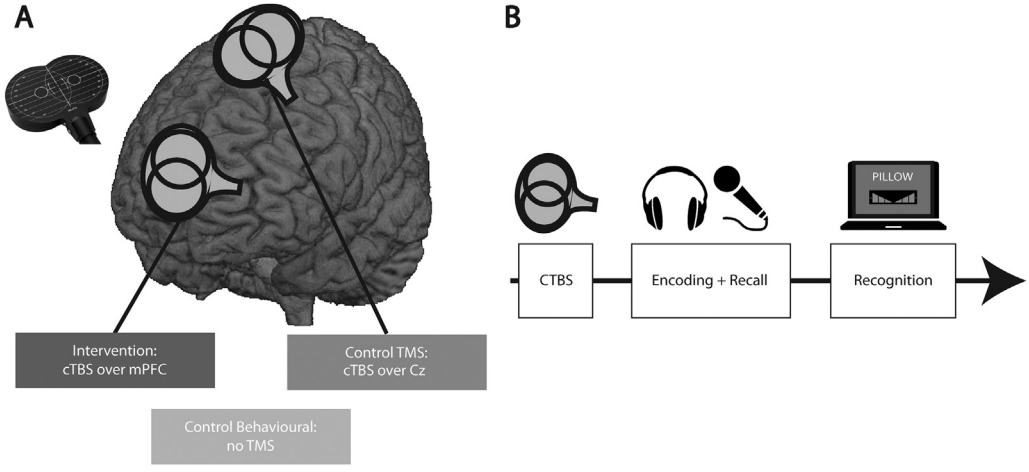

Fig. 1 – Experimental procedures. A. The three groups consisted of an intervention group receiving stimulation over mPFC, a stimulation control group receiving stimulation over Cz, and a behavioral control group. B. The experimental session started with administration of cTBS using TMS. Next, participants listened to and immediately recalled the word lists. Lastly, they performed a recognition task on a computer.

(see Fig. 1). In the recognition task, the 54 old items, 54 unrelated lures and 18 critical lures were presented in randomized order. Each word was presented on screen in white font against a gray background, with a rating scale containing six answer options and their corresponding keyboard buttons displayed ([A]: 'certainly old', [S]: 'probably old', [D]: 'perhaps old', [J]: 'maybe new', [K]: 'probably new', [L]: 'certainly new'). This task was also self-paced, and after pressing a button the response was visible for a further 2 sec. Each trial was then separated by a 3 sec interval. Lastly, participant completed the debriefing questionnaires.

2.5. Analysis

Recall performance was scored manually by the experimenter (R.M.W.J.B.) during the session. An independent rater who was blind to group membership also rated recall scores by listening to the audio recordings for a subset of participants ($N = 41$, including all participants in stimulation conditions except two where audio files failed to record). Inter-rater reliability was assessed using interclass correlation (ICC) and was found to be consistent for recall of studied items (ICC = .964, $p < .001$) and recall of critical intrusions (ICC = .963, $p < .001$). The experimenter's scores were used with confidence hereafter. Recall was defined as mean proportion recalled of the total amount: studied items as words recalled compared to all items presented, and critical items as critical intrusions recalled compared to all critical lures. Recognition performance was characterized using d′. At low confidence levels, there were significant differences between the amount of 'old' and 'new' responses for lures and critical lures. Therefore, responses were collapsed across all confidence levels (high = 'certainly' old or new, middle = 'probably' and low = 'maybe'). The corrected recognition rate used the proportion of hits from all studied items. Similarly, corrected critical endorsement rate used the proportion of critical items endorsed versus all critical items, and the false alarm rate used the proportion of unrelated lures endorsed versus all unrelated lures. Rates of 1 were replaced with 1 − [1/(2 * N)] before calculating d′, with N equal to the total number of trials (Macmillan & Creelman, 2005). Qualitative sleep ratings on the six point Likert scale were converted to a quantitative scale from 1 till 6. Statistical analyses were performed using IBM SPSS statistics (version 21). ANCOVA tests were used to test group effects on the dependent measures, while controlling for the rated 'Sleep Quality', 'Drowsiness' and 'Feeling Rested'. If significant group effects were found, follow-up comparisons were performed using post hoc independent samples t-tests (two-tailed).

3. Results

3.1. Recall

Participants freely recalled on average more than half of all studied words (mean proportion correct: .57, S.E. = .01), and falsely recalled over a third of all critical intrusions (mean proportion: .36, S.E. = .03). 'Sleep Quality' as rated by participants for the night before the experimental session correlated positively with false recall [$\rho(59) = .320$, $p = .013$], whereas the rated 'Drowsiness' correlated negatively [$\rho(59) = -.266$, $p = .042$] with correct recall. There were, however, no significant group differences in these sleep parameters (see Table 2).

There was no effect of stimulation group on true recall performance following stimulation, while controlling for the effect of the sleep parameters [$F(2, 53) = .379$, $p = .687$, Partial Eta Squared = .014]. However, there were significant group differences in recall of critical intrusions [$F(2, 53) = 3.271$, $p = .046$, Partial Eta Squared = .110]. Specifically, the experimental group receiving TMS to the mPFC recalled fewer critical intrusions than the TMS control group (mean difference: .16, $p = .022$; cohen's $d = .67$) and the behavioral control group (mean difference: .12, $p = .041$; cohen's $d = .38$), while there were no differences in recall of critical intrusions between the two control groups (mean difference: .04, $p = .55$; cohen's $d = .26$, see Table 2 and Fig. 2). For comparison purposes, the effects on recall for the entire group, including those subjects that realized what the purpose of the task was, were not significant for true nor false recall [true recall: $F(2, 81) = .500$, $p = .608$, Partial Eta Squared = .014; false recall: $F(2, 81) = 1.796$, $p = .172$]. Thus, stimulation of the mPFC decreased false recall of critical intrusions for DRM-lists encoded after stimulation in those subjects that were naive to the purpose of the task.

The direction of the coil in the stimulation groups did not have an effect on true recall (mPFC stimulation group, mean

Table 2 – Group performance for recall and recognition tasks. Descriptive statistics of proportions and d′ are reported as means with S.E.M. in brackets.

		Behavioral control $N = 28$[a]	mPFC stimulation $N = 17$	Cz stimulation $N = 14$
Recall	Studied items total	.57 (.02)	.55 (.02)	.58 (.02)
	Critical intrusions total	.37 (.04)	.29 (.04)	.42 (.05)
	Time to complete (sec)	1108.10 (63.15)	1052.75 (51.03)	1169.26 (67.57)
	Serial position (#)[b]	6.57 (.32)	6.70 (.34)	7.21 (.38)
Recognition	Hits	.79 (.02)	.77 (.02)	.79 (.03)
	False alarms	.18 (.04)	.17 (.02)	.14 (.03)
	Critical intrusions	.84 (.03)	.81 (.05)	.88 (.03)
	D′ studied items	1.83 (.14)	1.78 (.12)	2.08 (.12)
	D′ critical intrusions	2.13 (.14)	2.07 (.19)	2.43 (.19)

[a] Data for the recognition task was missing for one participant.
[b] Data for some participants was not included due to missing audio-recordings (included data for Behavioral group: $N = 24$; Cz group: $N = 13$, mPFC group: $N = 16$).

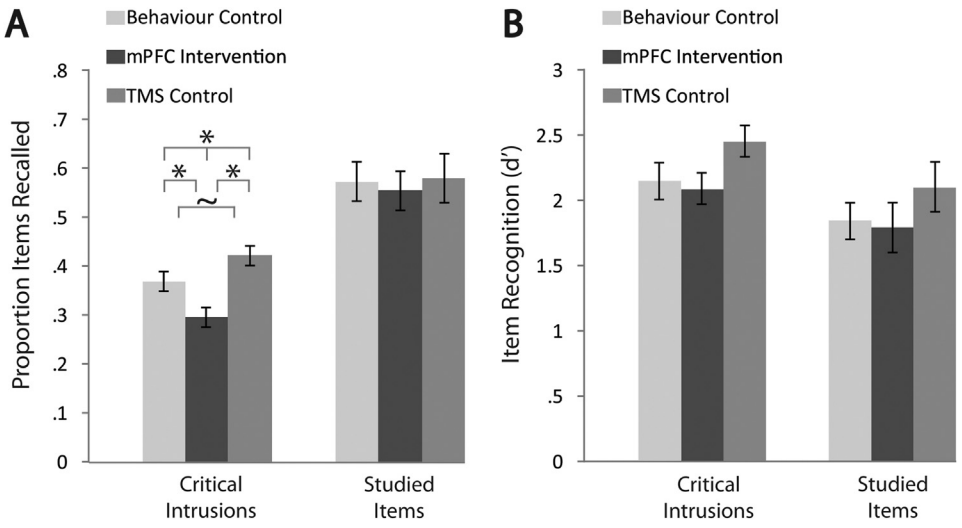

Fig. 2 – Performance on the DRM task. A. Free recall performance on critical intrusions and studied items for the intervention group and two control groups. B. Overall recognition performance on critical intrusions and studied items for the intervention group and two control groups. Error bars indicate S.E.M.; *$p < .05$; ~$p > .1$.

difference left-right handle orientation: .06, $p = .12$, Cz stimulation group, mean difference left-right: .02, $p = .62$) nor false recall performance (mPFC stimulation group, mean difference left-right: .20, $p = .92$, Cz stimulation group, mean difference left-right: 1.04, $p = .61$). Furthermore, there were also no differences in the time participants took to recall the lists [$F(2, 53) = .641$, $p = .530$], or the serial order in which the critical intrusion was recalled [$F(2, 47) = .817$, $p = .447$].

3.2. *Recognition*

Participants were better than chance in recognizing studied items [mean d′ studied items: 1.88, S.E. = .08, *vs* a chance level of d′ = 0, $t_{(57)} = 23.15$, $p < .001$], but also more likely than chance to endorse critical lures as studied [mean d′ endorsed critical intrusions: 2.19, S.E. = .10, versus a chance level of 0, $t_{(57)} = 22.53$, $p < .001$]. There were no group effects on recognition of studied items [$F(2, 52) = .972$, $p = .385$, Partial Eta Squared = .036] or critical intrusions [$F(2, 52) = .983$, $p = .381$, Partial Eta Squared = .036].

4. Discussion

This study set out to test the hypothesis that the mPFC is involved in evaluating the congruency of new memories with prior knowledge, thereby weighting the influence of schematic and episodic memory components. This hypothesis predicts that when encoding a list of semantically related words in the DRM paradigm ('rest', 'awake', 'pillow'), the weighting of schematic memory components in memory inferences is increased along with the probability of falsely recalling non-studied words of the same schematic category (i.e., the non-studied critical lure – 'sleep'). Experimental perturbation of the brain region presumed to support this process, the mPFC, should therefore induce a decrease in false memory formation. The results are that the application of an inhibitory TMS protocol to the mPFC before studying DRM lists indeed induced a decrease in false recall of critical lures for the lists studied after stimulation, whereas recognition and veridical recall performance remained unaffected compared to control groups. This pattern of results is in line with previous patient studies (Warren et al., 2014), and provides initial causal evidence for a specific role of the mPFC at the intersection of prior knowledge, memory formation, and memory specificity.

Certain procedural features and limitations of the study should be noted. First, the use of temporary interruption of the mPFC using a cTBS protocol applied via TMS obviates issues associated with brain plasticity that can arise with permanent lesions and thus the study of patients with frontal lobe damage. However, this approach rests on the assumption that cTBS has inhibitory effects on synaptic plasticity up to an hour in the medial wall of the prefrontal cortex (Hayward et al., 2007; Huang et al., 2005). Particular uncertainty surrounds the depth at which stimulation had an effect on cortical excitability. Of the lesion extent reported in Warren et al. (2014), it is unlikely that BA regions 32 and 12 were targeted, but plausible that BA region 10 in the anterior mPFC was reached, explaining the behavioral effects reported here. Further simulation studies should formally model physiological effects of stimulation using this specific coil and stimulation site and empirically verify these with functional neuroimaging studies in order to optimize the current design further.

Second, because encoding of the word lists was immediately followed by recall, it is not possible based on the current study to differentiate between encoding or recall processes. Future studies investigating the role of the mPFC in schema-based false memory inferences should temporally separate encoding and recall, and pinpoint effects on list encoding and recall separately. Third, we did not find any group effects on false recognition, most likely due to ceiling effects that are also reported previously (Warren et al., 2014). Fourth, the cTBS

was applied to the mPFC in the experimental group, with the coil placed in close proximity to peripheral facial muscles, pain and sensory receptors. However, in the stimulation control group, the coil was placed relatively distant from peripheral musculature and receptors. Therefore, the cTBS protocol could have induced stronger muscle contractions when applied to the mPFC, with potentially stronger sensations or discomfort, pain and emotional distress in this condition. These effects could confound task effects, although they would induce a general impairment rather than the specific impairment reported here. However, muscle contractions were observed to be limited, and over the course of the experiment only six participants from the two stimulation groups reported some minor experiences of discomfort, pain or stimulation of eyes and ears (four following mPFC stimulation, two following Cz stimulation). This relatively low level of discomfort compared to lateral prefrontal stimulation could be due to the fact that the medial facial muscles are smaller than lateral facial muscles.

The results reported here build and expand on previous studies that highlight a role of the mPFC in integrating novel experiences with existing schematic knowledge. The extent of overlap between novel experiences and schema determines the manner in which new experiences are encoded. If the overlap between schema and novel experiences is high, mnemonic processing relies more on schematic components rather than episodic memory components. Studies in animals (Tse et al., 2011), and human lesion (Spalding et al., 2015) and functional imaging studies (Bein, Reggev, & Maril, 2014; van Kesteren et al., 2013, 2014; Liu et al., 2016) have related processing in the mPFC to a learning benefit for information that is congruent with prior knowledge. However, the exact localization of these effects within the mPFC remains unprecise, as imaging studies have implicated both ventral (van Kesteren et al., 2013, 2014) and rostromedial aspects of the mPFC (Bein et al., 2014; Brod, Lindenberger, Werkle-Bergner, & Shing, 2015). False memories have been linked to the mPFC in human lesion studies (Warren et al., 2014) and functional imaging studies, although precise localization of these effects in the mPFC is also lacking (Cabeza et al., 2001; Kim & Cabeza, 2007; McDermott et al., 2017).

Anatomical evidence from rodents and non-human primates indicate that the mPFC is connected, through the thalamus, with the medial temporal lobe and posterior representational areas (Aggleton & Brown, 1999; Aggleton, Desimone, & Mishkin, 1986; Amaral & Cowan, 1980; Cassel et al., 2013; DeVito, 1980; Hoover & Vertes, 2012; Hsu & Price, 2007; Van Der Werf, Jolles, Witter, & Uylings, 2003; Vertes, Hoover, Do Valle, Sherman, & Rodriguez, 2006; Vertes, Hoover, Szigeti-Buck, & Leranth, 2007). Recently, Xu and Südhof demonstrated in mice that the anatomical circuit including the hippocampus, nucleus reuniens of the thalamus and the mPFC determines the specificity of memory encoding (Xu & Südhof, 2013). Functional imaging studies in humans have also shown that the mPFC is functionally connected to regions in the medial temporal lobe and temporo–parietal association cortices (Andrews-Hanna, Reidler, Sepulcre, Poulin, & Buckner, 2010; Greicius, Krasnow, Reiss, & Menon, 2003; Saleem, Kondo, & Price, 2008; Uddin et al., 2010; Öngür & Price, 2000), potentially also mediated by the thalamus (Thielen, Takashima, Rutters, Tendolkar, & Fernández, 2015). Therefore, this brain region can function as a higher-order convergence zone of perceptual and mnemonic information. The anterior aspect of the mPFC, which was likely targeted here, has specifically been shown to connect both anatomically (Liu et al., 2013) and functionally to the medial temporal lobes in various task contexts (Bein et al., 2014; Benoit, Gilbert, & Burgess, 2011; Berkers et al., 2016; Brod, Lindenberger, Wagner, & Shing, 2016; Liu et al., 2016). In such a functional network, the medial temporal lobe is typically implicated in episodic memory (Nyberg, Mclntosh, Houle, Nilsson, & Tulving, 1996; Schacter & Wagner, 1999), whereas the angular gyrus and temporal regions are involved in storing higher-order conceptual knowledge (Bonner, Peelle, Cook, & Grossman, 2013; Bonnici, Richter, Yazar, & Simons, 2016; Price, Bonner, Peelle, & Grossman, 2015; Wagner et al., 2015). The mPFC, in turn, has been found to be involved in leveraging prior knowledge for making novel inferences (Zeithamova, Dominick, & Preston, 2012), simulating the future (Benoit, Szpunar, & Schacter, 2014; Benoit et al., 2011) and guiding decision-making (Bechara, Damasio, Tranel, & Damasio, 1997; Venkatraman, Rosati, Taren, & Huettel, 2009; Wang, Rogers et al., 2014, Wang, Luo et al., 2014). Therefore, the mPFC might be ideally positioned to weigh the relevance of schematic elements for the current situation, mediating concurrent benefits in schema-congruent learning and schema-based memory inferences. In the context of a list of semantically associated words, schematic elements are weighed more heavily compared to detailed episodic components, at the same time potentially promoting recall (Bower et al., 1969) and false schema-based memory inferences (Roediger & McDermott, 1995).

According to this account, perturbation of the mPFC should not only decrease the propensity for making false schema-based memory inferences, but also impair recall performance by abolishing schema-benefits for veridical recall. Here, we found no effects on veridical recall, nor were they found in patients with mPFC lesions (Warren et al., 2014). This lack of impaired veridical memory could be explained by the construction of DRM lists, which is done by selecting the strongest semantic associates of specific critical lure words (Roediger & McDermott, 1995; Stadler et al., 1999). Therefore, the study words are not selected on basis of the strength of their mutual semantic associations, although they are associated indirectly by virtue of the connecting critical lure word. For instance, the word list with the critical lure word 'black', contains study words with a strong semantic link to the critical lure word such as: 'white', 'coal', 'night' and 'funeral'. However, here the semantic link among the study words themselves is only indirect. As such, false recall of critical lures is strongly affected by schema memory components, whereas veridical recall is relatively more dependent on episodic memory. In such a scenario, lesions or experimental perturbation of the mPFC should indeed more strongly affect false memory for critical lures rather than veridical memory of studied words.

The stimulation protocol used is beneficial for stimulating deeper brain regions, but comes with the necessary side-effect of stimulating regions nearer the convexity. For instance, the frontal pole is likely affected by the protocol, which has been implicated in future-thinking and prospective memory

(Burgess, Scott, & Frith, 2003; Okuda et al., 2003), and encoding future goals (Tsujimoto, Genovesio, & Wise, 2011). Indeed, the encoding task likely involved a prospective component, as subjects knew at encoding that they would be tested later on the word lists. However, if a down-regulation of prospective future retrieval goals were the primary mechanism driving the selective effects on false memory recall found here, similar deficits would have been found on veridical recall. Furthermore, due to the increased diameter of the coil windings, stimulation probably also affected dorsolateral prefrontal cortices, which have been widely implicated in the top-down deployment of attention (Buschman & Miller, 2007) and working memory maintenance (Curtis & D'Esposito, 2003). One could speculate that working memory maintenance was required to keep words online when encoding the word lists, and it is only when several words are kept online simultaneously that the common theme of the list can be detected. In such a case, down-regulation of working memory maintenance would prevent participants from detecting the common theme, which would prevent false inferences of having encoded the critical lure. Although plausible initially, it has been shown that a reduction in working memory capacities is actually associated with an increase in false memories on the DRM-task (Peters, Jelicic, Verbeek, & Merckelbach, 2007) and misinformation paradigms (Zhu et al., 2010). Furthermore, down-regulation of working memory maintenance would probably also affect veridical recall of the correct words, rendering this mechanism an unlikely explanation for the reported effects.

Another alternative explanation within the schema framework can be made for the reported effects. It could be that the basic deficit underlying the selective effect on false recall are explained by a decreased ability to access, activate or construct prior knowledge while encoding word lists. Whereas retrieval of semantic information is typically related to more lateral regions of the prefrontal cortex in imaging studies (Badre & Wagner, 2002; Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997; Wagner, Paré-Blagoev, Clark, & Poldrack, 2001), stimulation of mPFC could have reduced the access to prior knowledge while encoding novel word lists. Indeed, while it can be argued that a down-regulation of the mPFC reduced the influence of prior knowledge on novel memory encoding, future studies should elucidate whether the critical task of the mPFC is to access or construct schematic themes while encoding new information, or evaluating already accessed schematic components while encoding new information.

Further work is necessary developing methods of stimulating the medial surface of the prefrontal cortex. Such a more focused protocol would be of interest to investigate schema-based memory processing. Previous brain stimulation studies of memory have only targeted parietal cortex (Bonnì et al., 2015; Wang, Rogers et al., 2014, Wang, Luo et al., 2014), primary sensory cortex (Waldhauser, Braun, & Hanslmayr, 2016) or lateral prefrontal cortex (Epstein et al., 2002; Floel et al., 2004; Hanslmayr, Matuschek, & Fellner, 2014; Javadi & Walsh, 2012; Javadi et al., 2012; Sandrini et al., 2003). This study adds to these approaches by documenting a protocol for off-line stimulation of the mPFC which does not suffer from the practical limitations of earlier protocols. For instance, earlier studies targeting the mPFC have used the double cone coil (Hayward et al., 2007; Klucharev et al., 2011), which has a specific geometry that limits placement such that only midline regions can be targeted, and the lateral windings cover a significant portion of the lateral prefrontal cortex. With a more conventional, smaller figure-of-8 coil it might be possible to more specifically stimulate midline regions. This methodology should, crucially, be validated using formal modeling and measurement of physiological effects of stimulation using functional neuroimaging.

In sum, as seminal studies on false memories have shown (Loftus, 1996), our memory is not like a book that can be written and read out in a rote manner. Rather, it is a generative, reconstructive process which relies on episodic memory components and inferences based on our prior knowledge or schemas to reconstruct prior experiences and simulate future scenarios. Memory distortions are prevalent even in people with superior autobiographical memory (Patihis et al., 2013). Therefore, it is of utmost importance to understand how these memory distortions arise and how the brain is involved in their construction. Our results provide initial evidence that an experimental stimulation intervention targeting the mPFC can causally decrease the influence of schema on memory formation.

Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.cortex.2016.12.015.

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