the memory is believed to be real by the patient. In false memory syndrome, a person's relationship and identity are focused on a traumatic memory that objectively has not happened. The syndrome is characterized when a person's whole identity and behavior are focused on a traumatic memory that is inaccurate. The syndrome is destructive when the patient avoids confronting any evidence that could contradict the memory. Therapists that use risky practices are likely to cause false memory syndrome. Eye-Movement Desensitisation and Reprocessing (EMDR) may play a role in creating false memories. EMDR therapy is used as a common treatment of Post Traumatic Stress Disorder symptoms by having the patient recall a traumatic event while performing lateral eye movements. However, EMDR has come to popularity quicker than it was validated for efficiency which limits the effects of EMDR that are known. EMDR may be detrimental to memory. Eye movements underline the quantity and quality of memory and EMDR has been shown to decrease the vividness of an autobiographical memory which could amplify false memories. False memories showing the same functions in the brain as real memories cause the question of whether memories are reliable.

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Topic: AS13 Emotion, Memory and Cognition

LOSS OF A5-NICOTINIC RECEPTORS AND SLEEP DEPRIVATION CAUSE AGE AND SEX-SPECIFIC MEMORY DEFICITS

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Mice lacking the α5-subunit of the nicotinic acetylcholine receptors (ACNA5 mice), have decreased pyramidal activity in the prefrontal cortex, and altered behaviours related to the PFC and hippocampus, where similar networks exist. Sleep deprivation (SD) has been shown to affect these networks causing increased activation of SST interneurons and suppression of hippocampal pyramidal neurons, leading to impaired hippocampus-related memories. Here we examined whether male and female ACNA5 mice of different ages exhibit altered hippocampus-dependent memory performance, and whether this is affected by SD. Adolescent and adult WT and ACNA5 mice of both sexes were examined in the object location task (OLT). SD was assessed by keeping animals awake for 5 hours though gentle handling after the first trial of OLT and memory performance was evaluated at the end of SD. We found that young male animals of both genotypes and young female WT mice showed the expected preference for the object in the novel location, with SD having no effect. In contrast, young female ACNA5 animals behaved similarly towards both objects, indicating an impaired location memory. In adult mice, only WT animals that were allowed to sleep exhibited preference for the object in the novel location. WT sleep deprived mice displayed impaired memory performance, as did adult ACNA5 animals, of both sexes. These findings reveal a significant age and sex-specific impact of the chronic absence of α5AChRs on location memory. As adults, both male and female ACNA5 animals exhibit impaired memory compared to WT controls. But female mutant mice appear more sensitive to the α5AChR deletion, as they are already impaired at earlier ages. The effect of sleep deprivation was also age-dependent as it did not affect adolescents but only adult WT animals, whose memory performance was reduced to the level of ACNA5 mice.

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TOWARDS UNDERSTANDING THE NEURAL UNDERPINNINGS OF ASSOCIATIVE MEMORY: A TES-EEG STUDY

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Associative memory (AM) represents an ability to bind unrelated information into meaningful units and encode them as distinct memories. AM has been the function of interest in many non-invasive transcranial electrical stimulation (tES) studies aiming to maximize the potential for memory modulation by varying stimulation loci, frequency, and amplitude. In the current study, we aimed to capture the tES modulation potential of AM performance when tailoring the stimulation protocols to the individual brain rhythms. By matching the stimulation frequency to the frequency of each subject's AM task-induced electrophysiological (EEG) activity in theta spectrum (4-8 Hz), we developed two types of personalized oscillatory protocols: theta-modulated otDCS and transcranial alternating current stimulation (tACS), which we administered alongside the constant transcranial direct current stimulation (tDCS) and a sham condition in the single-blind cross-over experiment. To comparatively assess the effects of different tES protocols delivered over the posterior parietal cortex, we tested the recognition and recall ability of the 42 healthy young adults on paired-associate paradigms after each of four conditions. During AM assessment participant's EEG activity was recorded. Group-level comparisons of each active tES condition against sham did not show differences in AM task performance either on recognition or cued-recall. However, data showed variability in performance depending on the task and the outcome measures. To explore the potential sources of variability in effect expression, analysis of the function-relevant neurophysiological markers is necessary. Therefore, behavioral results will be accompanied by features of underlying brain activity extracted from the EEG signals. Apart from introducing a novel approach to probing AM with personalized tES, this well-powered, multi-protocol, multi-task, and multi-measure study produced a comprehensive dataset that allows exploration of factors that could uncover different patterns in responsiveness to tES, as well as the insight into how neurophysiological changes reflected on the behavioral level.

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