

## Research article

# Shared regulatory systems in memory suppression and fear extinction: An ALE and MACM meta-analysis

Yuhao Zhao<sup>a,b</sup>, Tianyuan Zhang<sup>a,b</sup>, Ruizi Xiong<sup>a,b</sup>, Xingnan Cui<sup>a,b</sup>, Justin C. Hulbert<sup>c</sup>, Peiduo Liu<sup>a,b</sup>, Wenjing Yang<sup>a,b,\*</sup>

<sup>a</sup> Faculty of Psychology, Southwest University (SWU), Chongqing, 400715, China

<sup>b</sup> Key Laboratory of Cognition and Personality (SWU), Ministry of Education, Chongqing, 400715, China

<sup>c</sup> Program in Neuroscience, Bates College, Lewiston, ME, USA

## ARTICLE INFO

## Keywords:

Inhibitory control

Memory control

Fear extinction

Activation likelihood estimation (ALE)

Meta-analytic connectivity modeling (MACM)

## ABSTRACT

Memory suppression and fear extinction both reduce the influence of past experiences on current cognition and behavior, but they are typically studied within separate research traditions. The present study examined whether these two processes recruit partially overlapping neural systems involved in the regulation of unwanted memories and previously threat-associated cues. We conducted a preregistered coordinate-based meta-analysis of fMRI studies using activation likelihood estimation (ALE) and meta-analytic connectivity modeling (MACM). The analysis included 63 experiments involving 1452 participants and focused on contrasts commonly used to index retrieval suppression in the Think/No-Think paradigm and threat-cue processing during extinction learning. ALE revealed convergent activation in bilateral inferior frontal gyrus/anterior insula and right inferior parietal lobule. MACM further showed that these regions were embedded within broader executive-control and attention-related coactivation networks. These findings suggest that memory suppression and fear extinction partially converge within broader control- and attention-related systems involved in controlling task-relevant memory and threat representations. At the same time, partly distinct medial temporal and affective-learning patterns point to important domain-specific differences between intentional episodic retrieval suppression and extinction-based threat-safety updating. Overall, the findings help bridge research on memory control and fear extinction by identifying partially overlapping regulatory systems while preserving important theoretical distinctions between the two processes. This framework highlights how control- and attention-related neural systems may contribute to the regulation of unwanted memories and conditioned fear responses through their interactions with memory, threat-learning, and contextual updating processes.

## 1. Introduction

The ability to flexibly regulate the influence of past threat experiences is essential for adaptive behavior and emotional well-being [1]. Fear extinction provides a central model for understanding this capacity: when a previously threatening cue is repeatedly encountered without an aversive outcome, conditioned fear responses gradually diminish Pavlov [2]. Traditionally, extinction has been understood as a form of new safety learning that does not erase the original fear memory but modifies how threat associations are expressed in a given context. Neurobiological accounts have therefore emphasized interactions among the amygdala, hippocampus, and ventromedial prefrontal cortex in encoding threat-safety contingencies, contextual modulation, and extinction

memory [3–6]. More recent evidence, however, suggests that extinction also engages broader prefrontal, cingulo-opercular, and attentional-control systems, indicating that the attenuation of conditioned fear may involve not only associative updating but also regulatory control over reactivated threat-related representations [7,8].

During extinction learning, a previously threatening cue can still elicit fear because it reactivates stored memories of prior aversive experiences [9,10]. Thus, extinction may involve not only the formation of new safety associations but also the regulation of reactivated fear-related memories. Memory, as a central component of emotional experience, provides one pathway through which emotional responses may be regulated [11]. Accordingly, a growing body of research has examined how the control of memory processes influences emotional

\* Correspondence to: Faculty of Psychology, Southwest University (SWU), Beibei, Chongqing 400715, China.

E-mail address: [yangwenjing@swu.edu.cn](mailto:yangwenjing@swu.edu.cn) (W. Yang).

<https://doi.org/10.1016/j.bbr.2026.116483>

Received 3 April 2026; Received in revised form 12 August 2026; Accepted 15 September 2026

Available online 30 September 2026

0166-4328/© 2026 Elsevier B.V. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

functioning [11–13]. The Think/No-Think (TNT) paradigm is a well-established experimental framework for studying the intentional control of memory retrieval, in which participants are instructed to either retrieve or suppress unwanted memories [14–18]. In the TNT paradigm, participants first learn cue–target associations and are later instructed either to retrieve or to suppress the target when presented with the cue [19]. Repeated suppression typically leads to a decrease in subsequent recall, a phenomenon known as suppression-induced forgetting. Many studies have shown that memory control not only reduces the accessibility of unwanted memories but also diminishes the emotional responses associated with them [11,20–23]. Neuroimaging evidence further indicates that memory suppression engages prefrontal and cingulo-opercular control regions, including the dlPFC and vlPFC, which interact with medial temporal and affective-memory systems such as the hippocampus and amygdala during the regulation of unwanted memories [24,4,25]. Deficits in the ability to control unwanted memories have been linked to affective disorders such as depression and PTSD, which are characterized by intrusive threat memories and disturbances in emotional regulation Stramaccia et al. [26].

Collectively, these findings raise the possibility that memory control processes may contribute to some aspect of fear extinction [7]. This view aligns with everyday experience: for instance, a person who has recovered from a car accident may still feel fear when hearing the distant sound of braking tires. Overcoming this reaction may involve not only recognizing that the sound no longer signals personal danger, but also regulating the retrieval and emotional impact of the associated traumatic memory. Consistent with this idea, several researchers have proposed that memory inhibition may represent one functional component contributing to fear extinction, alongside other mechanisms such as safety learning, contextual modulation, and prediction-error-based updating [27]. This view is further supported by animal and human evidence implicating partially overlapping prefrontal-hippocampal and fronto-limbic pathways in both fear extinction and memory control. Key regions implicated across studies include the infralimbic cortex and nucleus reuniens in rodents, the hippocampus and amygdala, and in humans the right dorsolateral and ventrolateral prefrontal cortex, dorsal ACC/pre-SMA, and anterior insula. These regions may contribute to the regulation of threat-related memory representations through interactions between control-related cortical systems and medial temporal or affective-learning systems [7].

Taken together, these observations raise the possibility that fear extinction learning and memory control may recruit partially overlapping regulatory systems that support the control of reactivated memory and threat-related representations. Within this broader framework, we focus on inhibitory regulation as one potential functional component that may contribute to extinction learning, while recognizing that extinction also depends on safety learning, contextual modulation, and predictive updating [24]. From a clinical standpoint, although exposure-based interventions are grounded in extinction principles, they often show high relapse rates, practical implementation challenges, and limited long-term efficacy [28–32]. Identifying how memory control processes relate to fear extinction may therefore inform the development of cognitively grounded therapeutic approaches. Specifically, if inhibitory regulation contributes to the regulatory processes supporting extinction learning, strengthening memory-control capacities through targeted cognitive training may represent one promising avenue for improving emotional regulation and reducing the recurrence of maladaptive fear.

In the present study, we aimed to examine whether fear extinction and memory control recruit partially overlapping neural systems involved in regulating threat-related memory representations. To this end, we analyzed two established paradigms: the fear conditioning and extinction paradigm, focusing on the extinction learning phase, and the Think/No-Think (TNT) task, focusing on direct suppression conditions. The extinction learning phase was selected because conditioned threat cues continue to reactivate previously acquired threat-related

representations while conditioned fear responses gradually diminish. Thus, extinction learning may involve not only the acquisition of new safety associations but also the regulation of reactivated threat-related memories. The TNT direct suppression condition was selected because it provides the most direct experimental model of intentional memory control while minimizing potential confounds associated with alternative strategies such as attentional diversion or thought substitution. Building on theoretical accounts suggesting that memory-control processes may contribute to fear regulation [7], we examined the conjunction between No-Think > Think and CS+ > CS− contrasts. This analysis was designed to identify potential convergence in neural systems involved in the regulation of threat-related representations across the two paradigms. As a complementary analysis, we also examined the conjunction between Think > No-Think and CS− > CS+. Whereas the former contrast is commonly associated with memory retrieval, the latter has been linked to safety-signal processing and safety learning during extinction. This complementary analysis was included to characterize neural systems associated with retrieval- and safety-related processing and to provide a broader context for interpreting the specificity of the primary conjunction findings.

Based on these considerations, we expected memory suppression and fear extinction to show partial neural convergence within control- and attention-related cortical systems, particularly in prefrontal and parietal regions that have been implicated in the regulation of memory retrieval, threat processing, and task-relevant representations [8,24,33]. We also examined whether medial temporal and affective-learning regions, including the hippocampus and amygdala, would show overlapping or domain-specific patterns across the two paradigms, given their central roles in episodic memory, contextual modulation, and fear learning [7, 11]. Finally, we used MACM to determine whether the regions showing cross-domain convergence were embedded within broader large-scale networks associated with cognitive control, attentional orienting, and the regulation of threat-related representations. To minimize potential confounds related to developmental or clinical factors, only studies involving healthy adult participants were included.

## 2. Methods

### 2.1. Literature search and article selection

The meta-analysis was prospectively registered on the Open Science Framework (OSF; <https://doi.org/10.17605/OSF.IO/B24VN>) and conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [34]. The analytical procedures closely followed the preregistered protocol, with only minor differences from the initial plan, which are described in the Supplementary Materials 1.

Systematic literature searches were conducted in PubMed, Web of Science, and ProQuest (coverage through June 2025). Detailed search strategies are provided in Supplementary Table S1. Additional relevant studies were identified through backward citation tracking of included articles and prior meta-analyses [8,13,35].

The inclusion and exclusion criteria for the literature are as follows: (1) Publication Type: Only original research articles published in peer-reviewed English-language journals were considered. Empirical studies were included, while reviews, meta-analyses, and case reports were excluded. (2) Study Population: Only studies involving healthy adults without any clinically diagnosed psychiatric disorders were included. Studies focusing on clinical or subclinical populations were excluded. When studies included both patient and control groups, only data from healthy control participants were considered. (3) fMRI Methodology: Only neuroimaging studies employing whole-brain fMRI and reporting activation coordinates in a standard anatomical reference space—either Talairach or Montreal Neurological Institute (MNI)—were included. Studies were required to present results from whole-brain analyses. Studies based solely on region-of-interest (ROI) analyses were excluded.

unless whole-brain data were also reported. (4) Task Paradigm: Studies were included if they employed either the TNT paradigm for memory control or the fear conditioning and extinction paradigm for fear extinction, and reported whole-brain general linear model (GLM) analyses of relevant experimental contrasts. For TNT studies, both emotionally laden and neutral stimuli were included to ensure sufficient data coverage and enhance the robustness of the meta-analytic results. (5) Coordinate Availability Criteria: Only studies that reported activation coordinates for individual experimental conditions were included. Studies were considered eligible if they reported contrast-based activation results, such as “No-Think > Think” or “CS+ > CS-”. In the context of fear extinction, CS+ refers to a conditioned stimulus previously paired with an aversive outcome, whereas CS- denotes a non-conditioned stimulus that was never associated with the aversive event.

To safeguard analytical validity, multiple methodological biases were systematically mitigated throughout this meta-analysis: (1) Selection bias was controlled by excluding clinical/subclinical populations (e.g., trauma-exposed individuals below PTSD thresholds) while including only medication-naïve healthy adults, precluding neurodevelopmental, pathological, and stress-response confounds; (2) Dimensionality bias was addressed through auditing demographic distributions (age/sex), auditing task-design specificity (direct suppression / extinction learning), multiple comparison correction protocols, and reporting completeness; (3) Thresholding bias was controlled by requiring validated multiple comparison correction methods (FDR/FWE cluster-level correction), while accepting uncorrected voxel-level findings exclusively at stringent thresholds ( $p < 0.001$ ). (4) Duplicate data bias was precluded via cross-verification of sample descriptors, authorship, and experimental protocols—excluding cohorts with participant overlap; (5) Temporal bias was controlled through publication year documentation and methodological evolution tracking; (6) Extraction bias was mitigated by dual-researcher independent coordinate extraction with inter-rater validation.

## 2.2. Activation likelihood estimation (ALE) meta-analysis

In the present study, we conducted ALE meta-analyses using the GingerALE software (version 3.0.2; available at <http://brainmap.org/ale/>; [36]). The analyses were based on reported activation coordinates from eligible studies and included four contrast analyses and a conjunction analysis assessing the overlap between the memory control and fear extinction domains.

### 2.2.1. Single-dataset analysis

Four separate ALE meta-analyses were conducted to identify convergent activation patterns for each contrast of interest: “No-Think > Think”, “Think > No-Think”, “CS+ > CS-”, and “CS- > CS+”. For the memory control domain, activation coordinates were extracted from “No-Think > Think”, indexing inhibitory suppression, and “Think > No-Think”, indexing retrieval-related processing in the TNT paradigm. For the fear extinction domain, activation foci were derived from “CS+ > CS-”, reflecting the processing of threat cues during extinction learning, and “CS- > CS+”, reflecting the learning and expression of safety signals. All coordinates were converted into MNI space [37] and analyzed using the ALE algorithm [38], with statistical significance assessed via 1000 permutations and cluster-level FWE correction ( $p < 0.05$ , cluster-forming threshold  $p < 0.001$ ; [39, 40]).

### 2.2.2. Conjunction and contrast analyses

We performed conjunction analysis using GingerALE 3.0.2 to identify brain regions commonly activated in both the memory control and fear extinction conditions. Specifically, the conjunction focused on the contrasts “No-Think > Think” and “CS+ > CS-”, representing inhibitory processes during retrieval suppression and conditioned fear regulation, respectively. First, we conducted separate ALE analyses for each dataset and applied a voxel-level threshold of  $p < 0.001$  with 10,000

permutations and a minimum cluster size of 200 mm<sup>3</sup> to correct for multiple comparisons. We then computed a conjunction map by taking the voxel-wise minimum of the two thresholded ALE images. The resulting map highlighted regions of significant activation convergence shared across both conditions [41].

In addition to the primary conjunction between “No-Think > Think” and “CS+ > CS-”, we also examined the conjunction between “Think > No-Think” and “CS- > CS+” as a complementary analysis. This analysis was conducted to characterize patterns of activation convergence associated with retrieval- and safety-related processing and to provide a broader context for interpreting the specificity of the primary conjunction findings. Full methodological details, including preprocessing steps, null distribution generation, and anatomical labeling procedures, are provided in the Supplementary Material 2.

## 2.3. Meta-analytic connectivity modeling (MACM)

We employed MACM to identify task-specific coactivation patterns associated with predefined ROIs. MACM is a data-driven approach that estimates functional connectivity based on the consistent coactivation of brain regions across a large corpus of task-based neuroimaging studies [42,43]. The underlying assumption of this method is that brain regions are functionally connected if they are consistently coactivated across multiple independent experiments.

To implement the analysis, we defined ROIs and conducted a meta-analytic search using the BrainMap Functional database, followed by ALE to identify consistent coactivation patterns. We first defined ROIs based on the results of the ALE conjunction analysis. All significant conjunction clusters were selected, and a 12-mm cubic ROI [44] was centered on the peak coordinate of each cluster. For each ROI, we then conducted an independent meta-analytic search in the BrainMap Functional database (<http://www.brainmap.org>) to extract task-related coactivation foci. The search was restricted to studies categorized as “Activations only” under the “Experiments” field, conducted in the context of “Normal Mapping,” and involving “Normals” as participants. This procedure enables the identification of statistically robust coactivation patterns for each ROI across an independent sample of task-based neuroimaging studies. To characterize the overall coactivation profile of each predefined ROI, we then merged all foci extracted from its constituent clusters into a single dataset, which was used to represent the coactivation pattern associated with the entire ROI. To ensure the independence of MACM results across ROIs, studies that overlapped between different ROIs were excluded prior to subsequent analysis. Retrieved coordinates were exported and converted into MNI space for subsequent analysis [45,46].

We conducted MACM analyses separately for three ROIs identified in the ALE conjunction analysis: (1) the left inferior frontal gyrus (270 experiments, 4704 subjects, 3760 activation foci), (2) the right inferior frontal gyrus (417 experiments, 6528 subjects, 6872 foci), and (3) the right inferior parietal lobule (93 experiments, 1293 subjects, 1516 foci). After removing duplicate experiments that were shared across ROIs, the final combined dataset included 715 unique experiments and 10,696 activation foci. Each dataset was analyzed independently using GingerALE version 3.0.2, with the same statistical thresholds applied as in the primary ALE analyses: a voxel-level threshold of  $p < 0.001$ , cluster-level FWE correction at  $p < 0.05$ .

## 2.4. Network-based correlation analysis

To quantitatively characterize the large-scale functional architecture of the identified coactivation patterns, the results of the MACM analysis were mapped onto canonical large-scale resting-state networks (RSNs) [41,44, 47]. Specifically, we utilized the seven-network cortical parcellation proposed by B. T. Thomas Yeo and colleagues Thomas Yeo et al. [48], which was further refined in the atlas developed by Konrad Schaefer and colleagues Schaefer et al. [49]. These canonical RSNs

include the visual network (VN), somatomotor network (SMN), dorsal attention network (DAN), ventral attention network (VAN), frontoparietal control network (FPCN; also referred to as the executive control network, ECN), default mode network (DMN), and limbic network (LN). In addition to the seven-network parcellation, we also employed the finer-grained network subdivision derived from the *Schaefer2018\_100Parcels\_17Networks* template, which provides a more detailed delineation of large-scale functional systems.

Following established protocols [44], we assessed network correspondence by calculating both relative and absolute overlap between the aggregated MACM map and RSN templates ([41]; Huang, 2023). Spatial overlap between the MACM-derived coactivation pattern and each RSN was quantified using two complementary metrics. First, the spatial overlap proportion (SOP) was used to characterize relative network distributions, defined as the proportion of overlapping voxels between the MACM coactivation pattern and a given RSN relative to the total number of voxels in the MACM map. Second, the Dice similarity coefficient was calculated to quantify absolute spatial overlap between the two maps. The definitions and computational procedures for SOP and Dice coefficient followed previous studies [44,50,51]. In the formulas, ( $X_i$ ) denotes the  $i$ th MACM-derived coactivation pattern and ( $Y_j$ ) represents the  $j$ th RSN template.

As a supplementary analysis, the ECN was further subdivided into three subnetworks—ControlA (ContA), ControlB (ContB), and ControlC (ContC)—based on the Schaefer2018 functional parcellation. Each ECN subnetwork mask was individually compared with the aggregated coactivation map using the same SOP and Dice coefficient procedures in order to further characterize the subnetwork-level distribution of the MACM-derived functional connectivity pattern. This approach allows the MACM-derived coactivation pattern to be interpreted within the framework of established large-scale functional brain systems.

$$SOP_{(ij)} = \frac{X_i \cap Y_j}{X_i}$$

$$Dice_{(ij)} = \frac{2|X_i \cap Y_j|}{X_i \cup Y_j}$$

### 3. Results

Of the 2326 publications initially identified, 1452 participants across 63 independent subject groups from 36 studies were included in the final meta-analysis. Details of the study selection process are presented in Fig. 1, while Tables 1 and 2 provide an overview of the included studies and the demographic characteristics of the participant samples, respectively. Comprehensive activation coordinates and detailed slice images are provided in the Supplementary Materials 2&4.

#### 3.1. Independent analyses of memory control

The *No-Think > Think* contrast revealed 11 significant clusters, with peak activations observed in the bilateral anterior insulae, inferior frontal gyrus (IFG), superior frontal gyrus (SFG), supplementary motor area (SMA) and pre-SMA, dACC, superior/middle frontal gyri (MFG) and inferior parietal lobule (IPL). Conversely, the *Think > No-Think* analysis identified 6 clusters. The medial temporal lobe (MTL) showed bilateral involvement, where activations primarily involved the parahippocampal gyrus and extended into the hippocampus. Additional activations were located in the posterior cingulate cortex and cuneus (see Fig. 2).

(a) Formal conjunction analysis between *No-Think > Think* and *CS+ > CS-*, conducted to identify regions showing statistically significant cross-domain convergence between retrieval suppression and threat-cue processing during extinction. The conjunction revealed overlapping activation in bilateral IFG/anterior insula and right IPL. (b–e) Independent ALE meta-analyses for each contrast, shown to characterize the activation patterns within each domain and to provide context for interpreting the formal conjunction results: (b) *No-Think > Think*, indexing retrieval suppression during memory control; (c) *CS+ > CS-*, reflecting threat-cue processing during fear extinction; (d) *Think > No-Think*, reflecting retrieval-related processing; and (e) *CS- > CS+*, reflecting safety-signal processing during extinction. Importantly, panels (b–e) represent independent domain-specific ALE results and should not be interpreted as evidence of formal cross-domain overlap.

SMA, supplementary motor area; dACC, dorsal anterior cingulate cortex; IFG, inferior frontal gyrus; SFG, superior frontal gyrus; IPL, inferior parietal lobule; dmPFC, dorsomedial prefrontal cortex; CS+,

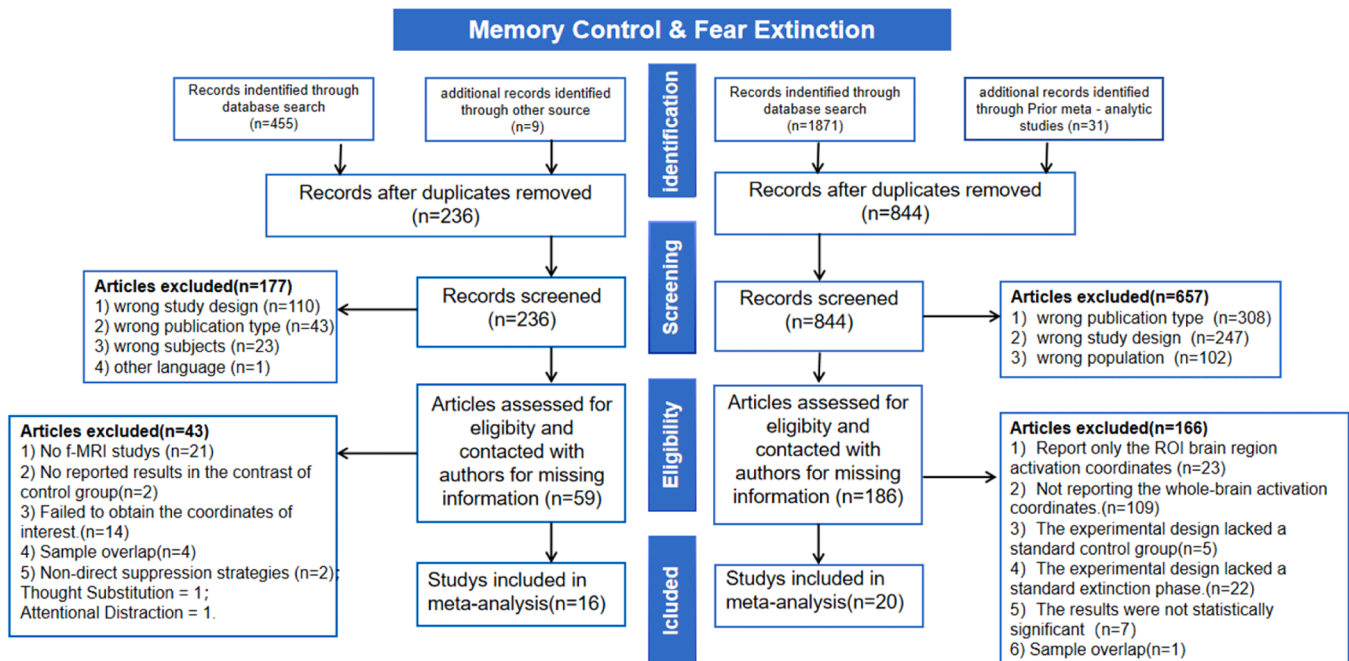


Fig. 1. Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) flow diagram for the procedure of study identification.

**Table 1**

Characteristics of the 20 fMRI studies included in the fear extinction meta-analysis.

| Authors                 | n   | Males (%) | mean age | materials of CS\UCS                          | Reinforcement rate during conditioning (%) | Immediate extinction? | Same Environment in Conditioning-Extinction Phases? | Correction Level              |
|-------------------------|-----|-----------|----------|----------------------------------------------|--------------------------------------------|-----------------------|-----------------------------------------------------|-------------------------------|
| Faul et al. [52]        | 40  | 55        | 24.55    | face\electric stimulation                    | 50%                                        | no(1 day)             | no                                                  | $p_{\text{corr}} < 0.05$ FWE  |
| Gramsch et al. [53]     | 24  | 46        | 29.02    | geometrical figures \rectal pain stimulation | 80%                                        | yes                   | yes                                                 | $p_{\text{uncorr}} < 0.001$   |
| Hagedorn et al. [54]    | 60  | 50        | 23.9     | geometrical figures \electric stimulation    | 62.50%                                     | no                    | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Hennings et al. [55]    | 24  | 38        | 21       | pictures\electric stimulation                | 50%                                        | no                    | no                                                  | $p_{\text{corr}} < 0.05$ FWE  |
| Hermann et al. [56]     | 46  | 100       | 23.4     | lights\electric stimulation                  | 62.50%                                     | yes                   | no                                                  | $p_{\text{corr}} < 0.05$ FWE  |
| Hollandt et al. [57]    | 16  | 19        | 23.4     | face\electric stimulation                    | 60%                                        | no(1 day)             | yes                                                 | $p_{\text{uncorr}} < 0.001$   |
| Holt et al. [58]        | 17  | 100       | 34.2     | lights\electric stimulation                  | 60%                                        | no(1 day)             | no                                                  | $p_{\text{corr}} < 0.05$ MC   |
| Kattoor et al. [59]     | 19  | 71        | 24.06    | geometrical figures \rectal pain stimulation | 75%                                        | yes                   | yes                                                 | $p_{\text{uncorr}} < 0.001$   |
| Kinner et al. [60]      | 64  | 50        | 23.67    | lights\electric stimulation                  | 62.50%                                     | no(1 day)             | no                                                  | $p_{\text{corr}} < 0.05$ FWE  |
| Levar et al. [61]       | 70  | 100       | 21.75    | geometrical figures \electric stimulation    | 33%                                        | yes                   | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Maier et al. [62]       | 17  | 41        | 31       | pictures\electric stimulation                | 50%                                        | no(1 day)             | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Merz et al. [63]        | 15  | 100       | 24.9     | geometrical figures \electric stimulation    | 62.50%                                     | yes                   | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Pohlack et al. [64]     | 118 | 66        | NA       | colors\electric stimulation                  | 50%                                        | yes                   | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Ridderbusch et al. [65] | 100 | 54        | 33.1     | faces\electric stimulation                   | 60%                                        | no(1 day)             | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Sehlmeyer et al. [66]   | 32  | 38        | 23.6     | faces\acoustic white noise                   | 25%                                        | yes                   | yes                                                 | $p_{\text{uncorr}} < 0.001$   |
| Seo et al. [67]         | 19  | 22        | 29.3     | lights\electric stimulation                  | 62.50%                                     | no(1 day)             | no                                                  | $p_{\text{corr}} < 0.05$ FWE  |
| Spohrs et al. [68]      | 51  | 100       | 22.8     | geometrical figures \thermal stimulation     | 50%                                        | no(1 day)             | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Spoormaker et al. [69]  | 16  | 100       | 25       | geometrical figures \electric stimulation    | 50%                                        | yes                   | yes                                                 | $p_{\text{uncorr}} < 0.005$   |
| Visser et al. [70]      | 79  | 31        | 22.1     | pictures\electric stimulation                | 46%                                        | no(2 day)             | yes                                                 | $p_{\text{corr}} < 0.05$ TFCE |
| [71]                    | 93  | 33        | 32.93    | face\electric stimulation                    | 60%                                        | no(1 day)             | yes                                                 | $p_{\text{corr}} < 0.05$ FWE  |
| Total/mean              | 920 | 60.7      | 25.98    |                                              | 56%                                        |                       |                                                     |                               |

Abbreviations: CS, conditioned stimulus; UCS unconditioned stimulus;  $p_{\text{uncorr}}$  = uncorrected  $p$ -value for multiple comparisons;  $p_{\text{corr}}$  = corrected  $p$ -value for multiple comparisons; FWE = with family-wise error correction; TFCE = Threshold-Free Cluster Enhancement; MC = Monte Carlo-corrected; fMRI=functional magnetic resonance imaging, NA= Not available.

conditioned stimulus previously paired with an aversive outcome; CS−, non-conditioned stimulus never associated with the aversive outcome.

### 3.2. Independent analyses of fear extinction

For the CS+ > CS− contrast, seven significant clusters emerged, involving the bilateral anterior insulae, ventral striatum, rostral anterior cingulate cortex, and bilateral IFG. In the reverse contrast CS− > CS+, seven clusters were identified. Significant activation was observed in the left MTL, involving the parahippocampal gyrus and extending into both the hippocampus and the amygdala. Other activations were observed in the bilateral posterior cingulate cortex, orbital gyrus, and angular gyrus (see Fig. 2).

(a) Descriptive spatial comparison of medial temporal lobe (MTL) activation patterns derived from the independent ALE meta-analyses of *Think* > *No-Think* and CS− > CS+. The memory-control map (*Think* > *No-Think*; red) showed bilateral hippocampal/parahippocampal involvement, whereas the fear-extinction map (CS− > CS+; blue) showed a more left-lateralized MTL pattern. These independent ALE maps are presented to characterize the spatial distribution of MTL

involvement across the two domains.

(b) Results of the direct ALE contrast analysis, (*No-Think* > *Think*) > (CS+ > CS−), conducted to identify regions showing significantly greater activation convergence for memory suppression than for threat-cue processing during fear extinction. Significant differences were observed predominantly in right-lateralized frontal regions and the right precuneus.

SFG, superior frontal gyrus; MFG, middle frontal gyrus; CS+, conditioned stimulus previously paired with an aversive outcome; CS−, non-conditioned stimulus never associated with the aversive outcome.

### 3.3. Shared activation patterns across memory control and fear extinction

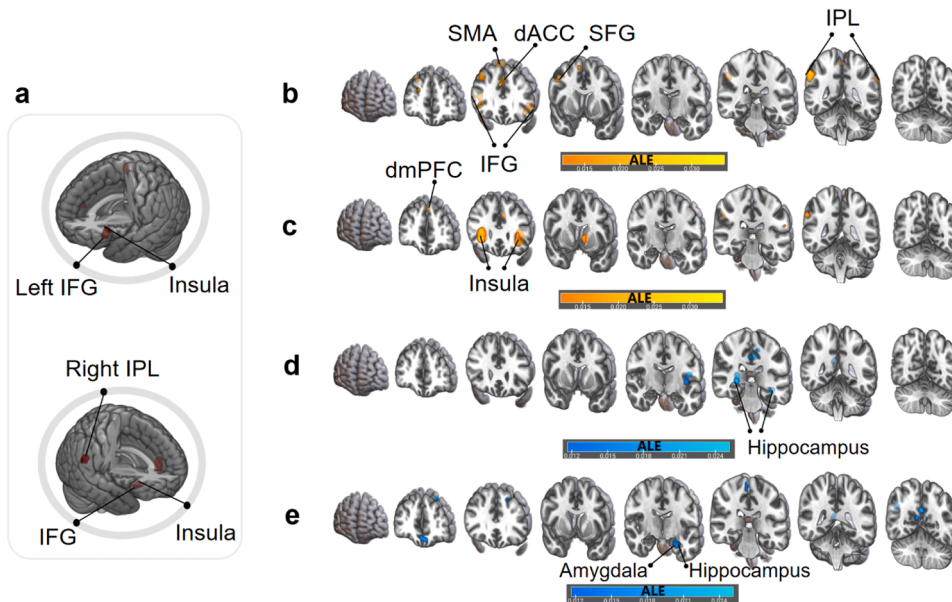
To identify shared neural activation across memory control and fear extinction, a formal conjunction analysis was performed (see Fig. 2 and Supplementary Fig. S6). This analysis revealed three significant clusters in the *No-Think* > *Think* ∩ CS+ > CS− contrast: (1) the left anterior insula and inferior frontal gyrus, (2) the right anterior insula and inferior frontal gyrus, and (3) the right inferior parietal lobule. A fourth cluster was also identified in the conjunction analysis, centered in the left dorsal

**Table 2**

Characteristics of the 16 fMRI studies included in the memory control meta-analysis.

| Authors               | n   | Males (%) | mean age | Cue-target materials | Nature of the cue-target materials | Suppression Strategy | Test Phase with SP/IP Probes | Correction Level              |
|-----------------------|-----|-----------|----------|----------------------|------------------------------------|----------------------|------------------------------|-------------------------------|
| Benoit, Anderson [72] | 24  | 58        | 25       | word pairs           | Neutral - Neutral                  | inhibitory control   | SP & IP                      | $p_{\text{uncorr}} < 0.001$   |
| Benoit et al. [73]    | 18  | 33        | 25       | word pairs           | Neutral - Neutral                  | inhibitory control   | SP & IP                      | $p_{\text{corr}} < 0.05$ FWE  |
| Benoit et al. [73]    | 16  | 50        | 22       | word-picture         | Neutral - Neutral                  | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Depue et al. [74]     | 16  | 63        | 20       | face - picture       | Neutral - Negative                 | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Depue et al. [74]     | 21  | 52        | 21.5     | face - picture       | Neutral - Neutral                  | inhibitory control   | IP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Gagnepain.2014        | 24  | 52        | 22.3     | word - picture       | Neutral - Neutral                  | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Gagnepain et al. [75] | 24  | 33        | 26.5     | face - picture       | Neutral - Negative & Neutral       | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Hulbert et al. [76]   | 18  | 36        | 26.5     | word pairs           | Neutral - Neutral                  | inhibitory control   | SP & IP                      | $p_{\text{corr}} < 0.05$ GRFT |
| Levy, Anderson [77]   | 18  | 44        | 25.5     | word pairs           | Neutral - Neutral                  | inhibitory control   | SP & IP                      | $p_{\text{corr}} < 0.05$ FWE  |
| Liu et al. [78]       | 21  | 100       | 21.6     | face - picture       | Neutral - Negative                 | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Mary et al. [79]      | 175 | NA        | 39       | word - picture       | Neutral - Neutral                  | inhibitory control   | SP & IP                      | $p_{\text{corr}} < 0.05$ FWE  |
| Meyer.2022            | 33  | 49        | 24.85    | picture pairs        | Neutral - Negative                 | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Sacchet et al. [80]   | 16  | 50        | 31.7     | word pairs           | Neutral - Negative & Neutral       | inhibitory control   | SP & IP                      | $p_{\text{corr}} < 0.05$ FWE  |
| Smith et al. [81]     | 48  | 48        | 29.8     | face - picture       | Neutral - Negative                 | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FDR  |
| Wei.2021              | 26  | 42        | 23.41    | picture pairs        | Neutral - Neutral                  | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FWE  |
| Yang et al. [20]      | 20  | NA        | 20.56    | word pairs           | Neutral - Negative & Neutral       | inhibitory control   | SP                           | $p_{\text{corr}} < 0.05$ FDR  |
| Total/mean            | 518 | 51        | 25.17    |                      |                                    |                      |                              |                               |

Abbreviations: SP, Same Probe test; IP, Independent Probe test;  $p_{\text{uncorr}}$  = uncorrected  $p$ -value for multiple comparisons;  $p_{\text{corr}}$  = corrected  $p$ -value for multiple comparisons; FWE = with family-wise error correction; FDR = with false discovery rate correction; fMRI=functional magnetic resonance imaging, NA= Not available.

**Fig. 2.** ALE activation patterns for memory control and fear extinction.

anterior cingulate cortex, corresponding to Brodmann area 32. However, due to its limited spatial extent (2 voxels), this cluster did not meet the minimum size threshold for subsequent MACM and was excluded from further connectivity and network analyses. Therefore, three conjunction-derived clusters located in the bilateral inferior frontal gyri and right inferior parietal lobule were selected as ROIs for follow-up analyses. Detailed coordinates and statistical values for all clusters are provided in Supplementary Table S2&S3. No significant clusters were identified for the reverse conjunction contrast  $Think > No-Think \cap CS- > CS+$ .

### 3.4. Differential activation between memory control and fear extinction

To facilitate comparison across domains, Fig. 3a presents the MTL patterns derived from the independent ALE analyses of memory control

and fear extinction, whereas Fig. 3b shows the direct contrast between the  $No-Think > Think$  and  $CS+ > CS-$  maps.

Direct contrast analyses were conducted in both directions for the suppression/threat-processing contrasts and for the retrieval/safety-processing contrasts. For the comparison  $(No-Think > Think) > (CS+ > CS-)$ , six significant clusters were identified, primarily involving right-lateralized frontal regions, including the superior and middle frontal gyri, precentral gyrus, inferior frontal gyrus, and medial frontal gyrus, together with the right precuneus (see Fig. 3b and Supplementary Table S3). No significant clusters were identified for the reverse contrast,  $(CS+ > CS-) > (No-Think > Think)$ .

For the comparison  $(Think > No-Think) > (CS- > CS+)$ , a significant cluster was identified in the right occipital lobe, with peak activation in the lingual gyrus and cuneus (BA 17). No significant clusters were found for the reverse contrast,  $(CS- > CS+) > (Think > No-Think)$ .

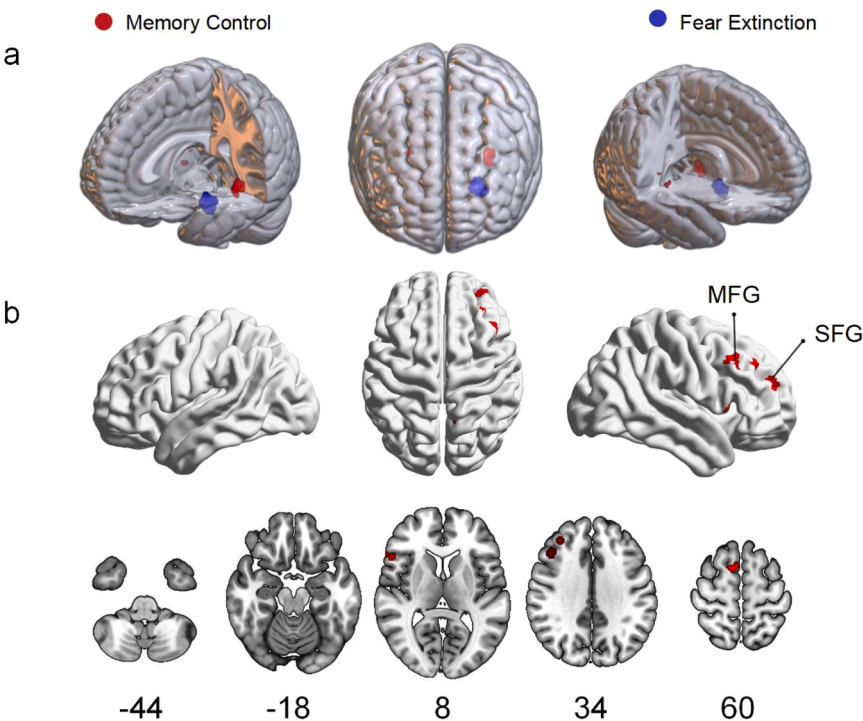


Fig. 3. Domain-specific medial temporal patterns and differential activation between memory control and fear extinction.

Neither the direct contrast nor the formal conjunction analysis revealed significant clusters within the MTL. To further characterize MTL involvement, we therefore descriptively compared the spatial

patterns observed in the independent *Think* > *No-Think* and *CS-* > *CS+* ALE analyses (Fig. 3a). Memory control exhibited bilateral recruitment of the parahippocampal gyrus and hippocampus. In contrast, fear

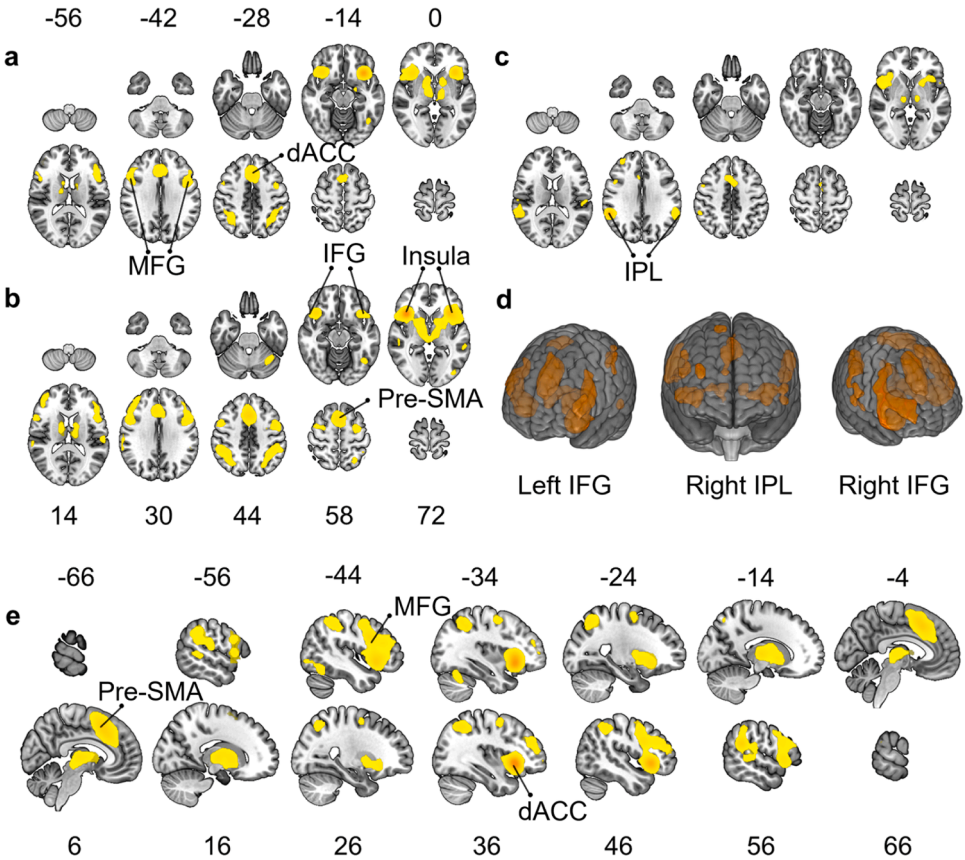


Fig. 4. Meta-analytic connectivity modeling results for the three conjunction-defined regions of interest (ROIs).

extinction activation was localized to the left parahippocampal gyrus, with an extent reaching the hippocampus and amygdala. Although these effects did not survive formal conjunction testing, the partially overlapping spatial patterns may suggest convergent involvement of MTL regions across domains, while also indicating distinct spatial distributions within the hippocampal–parahippocampal complex.

### 3.5. Coactivation maps of conjunction-derived regions

MACM revealed distinct yet overlapping coactivation profiles for the three regions identified in the conjunction analysis. For the left anterior insula and IFG, significant coactivation was observed with the bilateral IFG, ACC, thalamus, striatum, bilateral IPL, fusiform gyri, MFG, and cerebellum. The right anterior insula and IFG exhibited a comparable coactivation pattern, involving the bilateral IFG, ACC, thalamus, IPL, fusiform gyri, MFG, and cerebellar regions. Coactivation associated with the right IPL included the bilateral IFG and insulae, ACC, SMA, thalamic ventral nuclei, putamen, superior temporal gyri, medial frontal gyri, and precuneus (see Fig. 4).

To characterize the aggregate coactivation profile across all three regions, we combined the results from the individual MACM datasets, which had been independently generated for each ROI and comprised a total of 715 non-overlapping experiments and 11,571 participants (see

Methods). The conjunction-level MACM map, obtained by merging these datasets and applying identical ALE parameters, revealed a widespread coactivation network that encompassed the bilateral inferior frontal gyri, anterior insulae, anterior cingulate cortex, thalamus, putamen, inferior and superior parietal lobules, fusiform gyri, medial and middle frontal gyri, supplementary motor area, and cerebellum. These findings indicate a common task-based coactivation architecture broadly consistent with the profiles identified at the level of individual ROIs, while also suggesting convergent functional connectivity patterns associated with both memory control and fear extinction processes (see Fig. 4 and Supplementary Table S4).

The three ROIs were identified from the formal conjunction analysis of *No-Think* > *Think* and *CS+* > *CS-*, and MACM was conducted to characterize their broader task-based coactivation patterns. (a) Coactivation map associated with the left IFG. (b) Coactivation map associated with the right IFG. (c) Coactivation map associated with the right IPL. (d) Surface renderings of the MACM results: the left, middle, and right panels show coactivation patterns for the left IFG, right IPL, and right IFG, respectively. (e) Sagittal views of the aggregated MACM coactivation pattern across the three conjunction-defined ROIs.

Pre-SMA, pre-supplementary motor area; dACC, dorsal anterior cingulate cortex; IFG, inferior frontal gyrus; MFG, middle frontal gyrus; IPL, inferior parietal lobule.

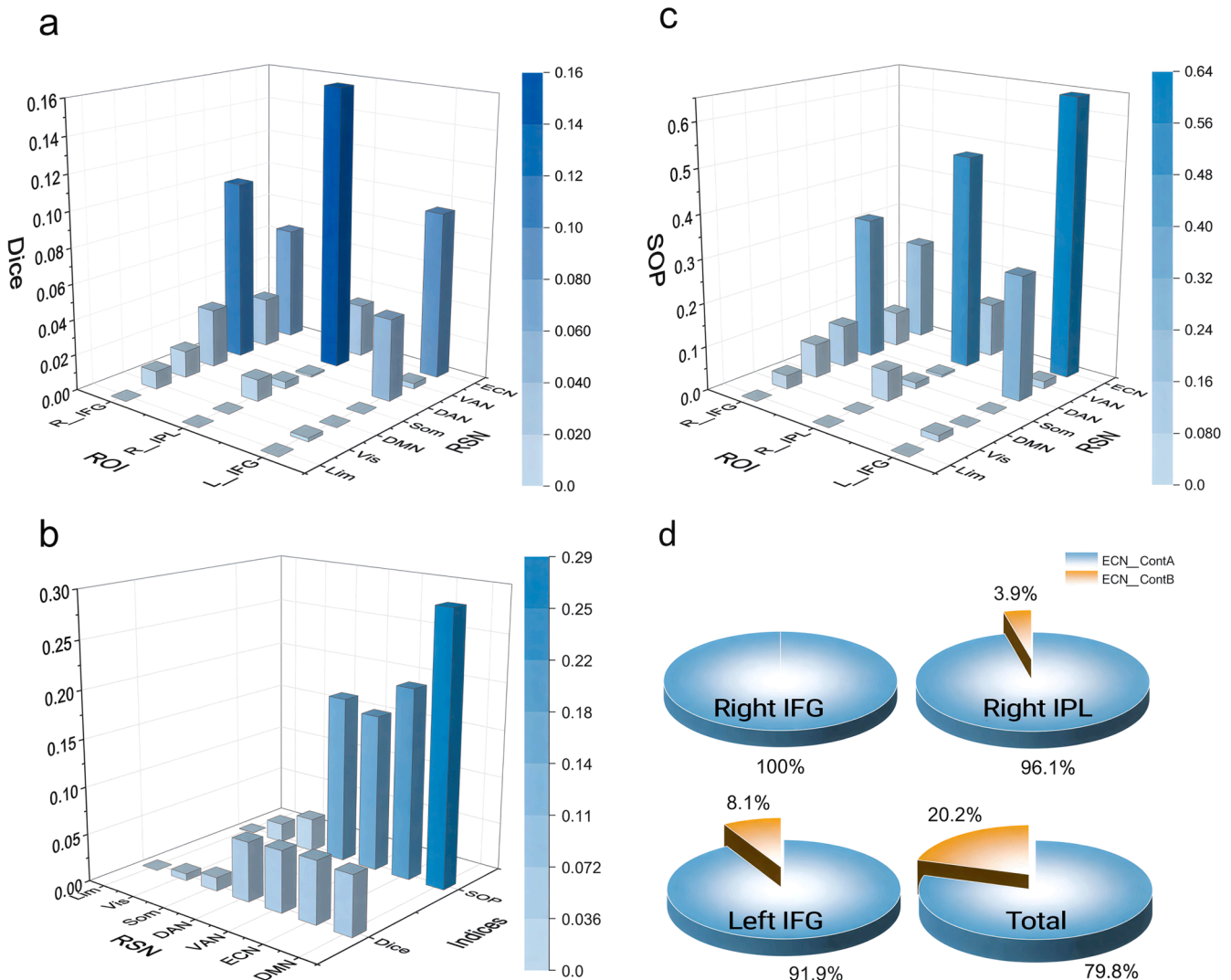


Fig. 5. Network-based characterization of MACM-derived coactivation patterns.

### 3.6. Schaefer atlas-based profiling of conjunction-derived coactivation networks

To delineate the large-scale functional network architecture associated with the conjunction-identified regions, we assessed the spatial convergence between their meta-analytic coactivation maps and the Schaefer2018\_100Parcels\_17Networks atlas (MNI152 space). This analysis was performed at two levels: (1) the canonical seven-network framework to capture broad functional affiliations, and (2) the tripartite subdivision of the ECN into ContA, ContB, and ContC, to characterize more fine-grained connectivity profiles (see Fig. 5).

At the seven-network level, the left IFG demonstrated the strongest spatial correspondence with the ECN (SOP = 0.641; Dice = 0.094), followed by moderate alignment with the DAN (SOP = 0.283; Dice = 0.046). Within the ECN, overlap was primarily localized to the ContA subdivision (SOP = 0.590; Dice = 0.186), with negligible involvement of ContB or ContC. The right IFG and anterior insula showed the highest convergence with the DAN (SOP = 0.328; Dice = 0.102), along with more limited coactivation with the ECN (SOP = 0.230; Dice = 0.065). Within the ECN, overlap was exclusively attributable to ContA (SOP = 0.223; Dice = 0.129), with no measurable contribution from ContB or ContC. The right IPL exhibited its greatest overlap with the VAN (SOP = 0.493; Dice = 0.16), and secondary correspondence with the ECN (SOP = 0.124; Dice = 0.03), again localized primarily to the ContA subdivision (SOP = 0.112; Dice = 0.063). Finally, coactivation patterns for all three seed regions showed minimal or no spatial convergence with the limbic network. Additionally, no appreciable overlap was observed between the right IPL and the visual network, or between the left IFG and the somatomotor network.

To further characterize the common large-scale network affiliations across all three ROIs, a combined meta-analytic coactivation map was generated and compared against the canonical seven-network atlas. This conjunction-level MACM map comprised 3913 suprathreshold voxels ( $Z \geq 3.09$ ). Among all networks, the highest Dice coefficient was observed for the ECN (Dice = 0.0650), followed closely by the VAN (Dice = 0.0647), DMN (Dice = 0.0623), and DAN (Dice = 0.0621). Minimal convergence was observed with the somatomotor (Dice = 0.014) and visual networks (Dice = 0.007), and no overlap was found with the limbic network. In terms of SOP, the DMN showed the strongest correspondence (SOP = 0.2870), followed by the ECN (SOP = 0.2011), DAN (SOP = 0.1761), and VAN (SOP = 0.1648). Only minimal overlap was observed with the somatomotor (SOP = 0.0348) and visual networks (SOP = 0.0197), and no overlap was detected with the limbic network.

The MACM maps were generated from the three ROIs identified by the formal conjunction analysis and were compared with canonical resting-state networks (RSNs) to characterize their large-scale network affiliations. (a) Dice coefficients quantifying absolute spatial overlap between each ROI-specific MACM map and seven canonical RSNs: limbic (Lim), visual (Vis), default mode network (DMN), somatomotor (Som), dorsal attention network (DAN), ventral attention network (VAN), and executive control network (ECN). (b) SOP and Dice coefficients quantifying the correspondence between the aggregated MACM map, combined across all three ROIs, and the seven canonical RSNs. (c) Spatial overlap proportion (SOP) between each ROI-specific MACM map and the same seven RSNs. (d) Relative distribution of ECN overlap across its subdivisions for each ROI-specific MACM map and the aggregated map, further characterizing the subnetwork-level organization of the observed ECN correspondence. ContA, Control A; ContB, Control B.

## 4. Discussion

In this study, we conducted a comprehensive meta-analysis to examine whether fear extinction and memory control recruit partially overlapping neural systems involved in the regulation of threat-related

memory representations. Drawing on activation data from 1452 participants in the ALE analysis, we identified convergent activation in bilateral IFG, anterior insula, and right IPL. Subsequent MACM analyses, based on functional coactivation data from 11,571 participants, revealed that these regions are embedded within broader control- and attention-related networks, particularly executive control and ventral attention systems. Together, these findings suggest partial neural convergence within cortical systems involved in cognitive control and attentional regulation. Within this broader regulatory architecture, inhibitory regulation may represent one functional component shared across memory suppression and fear extinction, alongside salience detection, attentional orienting, task-set maintenance, and general cognitive control [8].

The ALE analysis revealed convergent activation in the bilateral IFG, anterior insula, and right IPL, regions frequently implicated in inhibitory control, salience processing, attentional reorienting, and broader cognitive control processes [82–84]. The IFG-insula complex has been associated with domain-general control function, including response inhibition, salience detection, task-set updating, and emotion regulation [84,85]. These interpretations are broadly consistent with theoretical accounts suggesting that control-related cortical systems interact with medial temporal and affective-memory systems during the regulation of unwanted memories and threat-related representations [7]. In the context of memory suppression, these regions may contribute to the intentional control of retrieval by maintaining suppression goals, detecting intruding memory representations, and coordinating control over episodic retrieval processes ([24]; Engen et al., 2018). In the context of fear extinction, similar regions may support the regulation of responses to previously threat-associated cues by integrating salience detection, attentional orienting, and the updating of threat-related representations [8]. The IPL, which has been linked to attentional shifting and episodic retrieval, may further contribute to the monitoring and reorienting of attention when task-relevant memory or threat representations become active [86,87]. Together, these regions may support the flexible allocation of control and attention when memory or threat-related representations become task relevant. In this sense, the observed frontoparietal–insular convergence may provide a plausible neural context in which inhibitory regulation contributes to both memory suppression and fear extinction.

Although fear extinction and memory control showed partial convergence within control- and attention-related regions, they also exhibited certain domain-specific differences. Memory control elicited stronger engagement of a right-lateralized frontoparietal network—including the superior, middle, and medial frontal gyri, pre-cuneus, and precentral gyrus—which may reflect the greater demands associated with deliberate retrieval suppression, goal maintenance, and the monitoring of unwanted memory intrusions [16,25,88]. Compared with fear extinction, which often unfolds through repeated cue exposure and gradual updating of threat–safety associations, memory control requires participants to intentionally implement a suppression goal when memory cues are encountered. In this sense, memory suppression resembles explicit forms of emotion regulation, such as cognitive reappraisal, in that both involve the deliberate recruitment of control-related prefrontal systems to modulate memory or emotional responses [11,13,27]. Our results also refine prior meta-analytic findings on fear conditioning and extinction [35]. The relatively limited dlPFC involvement observed in the present analysis may reflect differences in paradigm selection and contrast definition. Whereas broader conditioning-related meta-analyses may include tasks that strongly engage expectancy, working memory, and decision-related processes, our focus on classical Pavlovian extinction and the CS+ > CS− contrast may have emphasized regions more closely related to salience processing, threat-cue evaluation, and extinction-related regulatory demands, including the IFG/anterior insula. Thus, rather than identifying the IFG as a uniquely inhibitory node, the present findings suggest that fear extinction and memory control recruit partially overlapping regulatory systems while

differing in the extent to which they engage deliberate frontoparietal control processes.

Regarding the MTL, neither the direct contrast nor the formal conjunction analysis revealed significant clusters, suggesting that evidence for cross-domain MTL convergence was weaker than that observed in frontoparietal-insular regions. Nevertheless, independent domain-specific analyses showed MTL involvement in both paradigms, but with distinct spatial profiles: memory control showed more bilateral and posterior hippocampal-parahippocampal recruitment, whereas fear extinction showed a more left-lateralized anterior MTL pattern extending toward the amygdala (see Supplementary Table S2 for details). These patterns may reflect different representational demands, with memory control relying more on the regulation of episodic retrieval processes and fear extinction relying more on the reactivation, contextual modulation, and updating of affective threat representations [89, 90, 91]. The involvement of the amygdala in fear extinction, but not in the present memory-control contrast, may partly reflect the predominance of neutral materials in current TNT paradigms. Indeed, studies using emotionally negative stimuli have reported amygdala modulation during memory suppression (e.g., [92–95]), suggesting that amygdala-centered affective systems may also become targets of inhibitory control when emotional content is involved. Thus, the MTL findings suggest that memory control and fear extinction engage domain-relevant but partly distinct representational processes within the MTL. Future TNT studies using fear-related or trauma-relevant materials may help clarify how memory-control processes interact with amygdala-centered affective systems.

To characterize how the conjunction-derived regions were integrated into larger functional networks, we applied MACM and quantified network correspondence using both SOP and Dice coefficients, providing complementary indices of their large-scale functional affiliations [44]. At the network level, the MACM results showed that the bilateral IFG/anterior insula were primarily associated with the ContA subdivision of the ECN, which includes dlPFC regions involved in goal maintenance, task-set control, and proactive regulation [22,96,97]. In contrast, the right IPL demonstrated stronger correspondence with the VAN, which is implicated in detecting salient or unexpected events and supporting stimulus-driven shifts in attention [22,98]. In the context of memory control, ECN engagement may reflect the deliberate, effortful maintenance of suppression goals and the proactive inhibition of retrieval [22], while VAN involvement may facilitate the detection and reorienting of attention when unwanted memories become active [16, 24]. During fear extinction, interaction between these networks may support the coordination of control and attention as individuals process salient threat cues, update threat–safety expectations, and regulate conditioned fear responses [99,100]. The involvement of the DMN in the coactivation pattern may further reflect internally directed processes, such as autobiographical retrieval, self-referential evaluation, or contextual representation, which could contribute to memory and fear regulation under some task conditions [101]. More broadly, these network-level findings suggest that inhibitory regulation operates in coordination with salience detection, attentional orienting, threat-expectancy updating, and autonomic/interoceptive processes during emotional responding [102,103]. Thus, the suppression of unwanted memories and the regulation of conditioned fear may depend on dynamic cooperation among executive-control, attention-related, and internally oriented systems that support inhibitory regulation, together with interactions involving subcortical structures and broader physiological states.

From a theoretical standpoint, the present findings provide partial support for the retrieval-stopping model [7], which proposes that fear extinction and memory control may involve interactions among prefrontal, medial temporal, and affective-learning systems during the regulation of threat-related memory representations. Our findings suggest that fear extinction may not depend solely on associative relearning, but may also recruit control- and attention-related processes that

help regulate the expression of reactivated threat memories. At the same time, the observed overlap may also be compatible with broader frameworks of extinction learning, including predictive-learning accounts emphasizing threat-expectancy updating, contextual-processing accounts highlighting the role of medial temporal systems, and memory-modification perspectives focusing on changes in the expression of previously acquired representations [104–106]. This interpretation complements traditional associative learning accounts while remaining compatible with broader frameworks emphasizing predictive learning, contextual updating, and memory modification. Thus, inhibitory regulation may represent one functional component within a broader set of mechanisms supporting adaptive emotional learning. Future research could combine fear extinction and TNT paradigms to examine whether behavioral regulation and neural engagement covary across these contexts. Such work may help clarify when overlapping prefrontal, insular, parietal, and medial temporal systems specifically support inhibition and when they reflect broader processes such as attentional orienting, salience detection, threat-expectancy updating, or contextual modulation. More broadly, comparing regulatory processes across extinction, reappraisal, and memory suppression may help determine how externally and internally driven emotional responses are controlled, thereby informing theoretical models of emotional learning and regulation-based interventions.

From a translational perspective, these findings may motivate future research examining whether memory-control or broader cognitive-control capacities could contribute to extinction-based interventions, which can be accompanied by relapse and limited long-term efficacy in some clinical contexts [29–32,107]. The observed overlap in control- and attention-related systems may provide a basis for generating hypotheses about how regulatory processes relate to maladaptive threat memories and fear responses. However, these translational implications remain speculative, as the present coordinate-based meta-analysis does not directly identify intervention targets or evaluate treatment efficacy. Future prospective and intervention studies will be needed to determine whether memory-control capacities or related prefrontal regulatory functions are meaningfully associated with extinction learning, relapse, or exposure-based treatment outcomes in clinical populations.

## 5. Limitations

Several limitations should be noted. First, the present meta-analysis focused on the classical fear conditioning/extinction paradigm and the TNT paradigm. Although these paradigms provide well-established models of extinction learning and intentional memory suppression, they do not fully capture more cognitively mediated forms of fear regulation, such as explicit reappraisal, expectancy modulation, predictive learning, or context-dependent memory updating. Moreover, the *No-Think* > *Think* and *CS+* > *CS-* contrasts differ in their cognitive demands and should not be treated as equivalent measures of inhibition. Future studies combining extinction and TNT procedures within the same participants, particularly with fear-related or trauma-relevant materials, would help clarify how memory-control processes interact with extinction-related learning and affective regulation.

Second, although the analyses met recommended standards for ALE meta-analysis [40], the number of available experiments was still modest, which may have reduced sensitivity to subtle or spatially variable effects, especially in medial temporal and affective-learning regions. In addition, coordinate-based ALE relies on reported peak coordinates rather than whole-brain statistical maps, which necessarily limits anatomical precision and may obscure finer spatial distinctions between the two paradigms. MACM also depends on the coverage and sampling characteristics of the BrainMap database, which may introduce database-related biases.

Finally, the present findings cannot fully dissociate inhibitory regulation from related processes such as salience detection, attentional engagement, task-set maintenance, threat-expectancy updating, and

broader cognitive control. Therefore, the observed convergence should be interpreted as evidence for partially overlapping regulatory systems rather than as a direct marker of inhibition-specific mechanisms. Caution is also warranted when generalizing these findings to clinical populations, as only healthy adults were included. Future work integrating neuroimaging with behavioral, physiological, and computational measures in both healthy and clinical samples will be needed to determine when these overlapping regulatory systems specifically support inhibitory regulation and when they reflect broader regulatory or learning processes.

## 6. Conclusions

The present ALE and MACM meta-analysis suggests that memory suppression and fear extinction recruit partially overlapping control- and attention-related neural systems, particularly bilateral IFG/anterior insula and right IPL. These regions were embedded within broader executive-control and attention-related coactivation networks, suggesting that both paradigms may draw on domain-general control and attentional processes when individuals suppress unwanted memories or regulate responses to previously threat-associated cues. At the same time, the findings preserve important distinctions between the two processes: memory suppression primarily involves deliberate control of episodic retrieval, whereas fear extinction additionally depends on threat-safety updating, contextual modulation, and emotional regulation. Together, these results help bridge research on memory control and fear extinction by identifying partially overlapping regulatory systems while highlighting the need for future neuroimaging, behavioral, and physiological studies to clarify how control-related processes contribute to the attenuation of unwanted memories and conditioned fear responses.

## Author contributions

YZ and WY conceived the study. YZ, TZ, RX, and XC collected and analyzed the data. YZ performed formal analysis, data curation, visualization, and software implementation, and prepared the first draft of the manuscript. WY, JH, and PL contributed to manuscript review and editing. WY and PL supervised the project and secured funding. All authors read and approved the final manuscript.

## CRediT authorship contribution statement

**Yuhao Zhao:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Tianyuan Zhang:** Validation, Supervision, Software, Methodology. **Ruizi Xiong:** Visualization, Validation, Data curation. **Xingnan Cui:** Supervision, Software. **Justin C. Hulbert:** Writing – review & editing. **Peiduo Liu:** Writing – review & editing. **Wenjing Yang:** Writing – review & editing, Supervision, Funding acquisition, Conceptualization.

## Ethics approval

This study is a meta-analysis based on previously published fMRI research and does not involve the collection of new human or animal data. Therefore, ethical approval was not required, and no new informed consent procedures were necessary. All original studies included in the analyses reported compliance with ethical standards and institutional review procedures.

## Declaration of Generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work we used ChatGPT in order to

improve the language and readability. After using this tool, we reviewed and edited the content as needed and take full responsibility for the content of the published article.

## Transparency and preregistration

This study was preregistered [<https://doi.org/10.17605/OSF.IO/B24VN>], and all meta-analytic coordinates are publicly available. While the core theoretical framework and methodology remain consistent with the original protocol, the specific neuroanatomical hypotheses were refined in the Introduction to align with the latest retrieval-stopping models [7]. Furthermore, we incorporated the ProQuest database as an additional source to ensure a more comprehensive literature search.

## Funding

This research was supported by the National Natural Science Foundation of China (No. 32271112) and the Fundamental Research Funds for the Central Universities (No. SWU2209239).

## Declaration of Competing Interest

None.

## Acknowledgements

This research was supported by the National Natural Science Foundation of China (No. 32271112) and the Fundamental Research Funds for the Central Universities (No. SWU2209239).

## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.bbr.2026.116483](https://doi.org/10.1016/j.bbr.2026.116483).

## Data availability statement

The data supporting this study are available on request from the corresponding author.

## References

- [1] G.J. Quirk, D. Mueller, Neural Mechanisms of Extinction Learning and Retrieval, *Neuropsychopharmacology* 33 (1) (2008) 56–72, <https://doi.org/10.1038/sj.npp.1301555>.
- [2] P.I. Pavlov, Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex, *Ann. Neurosci.* 17 (3) (2010) 136–141, <https://doi.org/10.5214/ans.0972-7531.1017309>.
- [3] J.A. Gottfried, R.J. Dolan, Human orbitofrontal cortex mediates extinction learning while accessing conditioned representations of value, *Nat. Neurosci.* 7 (10) (2004) 1144–1152, <https://doi.org/10.1038/nn1314>.
- [4] M.R. Milad, R.K. Pitman, C.B. Ellis, A.L. Gold, L.M. Shin, N.B. Lasko, M.A. Zeidan, K. Handwerker, S.P. Orr, S.L. Rauch, Neurobiological Basis of Failure to Recall Extinction Memory in Posttraumatic Stress Disorder, *Biol. Psychiatry* 66 (12) (2009) 1075–1082, <https://doi.org/10.1016/j.biopsych.2009.06.026>.
- [5] J.C. Motzkin, J. Hiser, I. Carroll, R. Wolf, M.K. Baskaya, M. Koenigs, L.Y. Atlas, Human ventromedial prefrontal cortex lesions enhance the effect of expectations on pain perception, *Cortex* 166 (2023) 188–206, <https://doi.org/10.1016/j.cortex.2023.04.009>.
- [6] D. Schiller, J.W. Kanen, J.E. LeDoux, M.-H. Monfils, E.A. Phelps, Extinction during reconsolidation of threat memory diminishes prefrontal cortex involvement, *Proc. Natl. Acad. Sci.* 110 (50) (2013) 20040–20045, <https://doi.org/10.1073/pnas.1320322110>.
- [7] M.C. Anderson, S.B. Floresco, Prefrontal-hippocampal interactions supporting the extinction of emotional memories: the retrieval stopping model, *Neuropsychopharmacology* 47 (1) (2022) 180–195, <https://doi.org/10.1038/s41386-021-01131-1>.
- [8] M. Picó-Pérez, M. Alemany-Navarro, J.E. Dunsmoor, J. Radua, A. Albajes-Eizaguirre, B. Vervliet, N. Cardoner, O. Benet, B.J. Harrison, C. Soriano-Mas, M. A. Fullana, Common and distinct neural correlates of fear extinction and cognitive reappraisal: A meta-analysis of fMRI studies, *Neurosci. & Biobehav. Rev.* 104 (2019) 102–115, <https://doi.org/10.1016/j.neubiorev.2019.06.029>.

- [9] J. Chen, Z. Fang, X. Zhang, Y. Zheng, Z. Chen, How fear memory is updated: from reconsolidation to extinction? *Neurosci. Bull.* 41 (6) (2025) 1054–1084, <https://doi.org/10.1007/s12264-025-01367-7>.
- [10] S. Dural, E. Gür, H. Çetinkaya, Testing the memory reconsolidation hypothesis in a fear extinction paradigm: the effects of ecological and arbitrary stimuli, *Learn. & Behav.* 50 (3) (2022) 417–432, <https://doi.org/10.3758/s13420-022-00536-2>.
- [11] H.G. Engen, M.C. Anderson, Memory control: a fundamental mechanism of emotion regulation, *Trends Cogn. Sci.* 22 (11) (2018) 982–995, <https://doi.org/10.1016/j.tics.2018.07.015>.
- [12] B.E. Depue, A neuroanatomical model of prefrontal inhibitory modulation of memory retrieval, *Neurosci. Biobehav. Rev.* 36 (5) (2012) 1382–1399, <https://doi.org/10.1016/j.neubiorev.2012.02.012>.
- [13] W. Liu, N. Peeters, G. Fernández, N. Kohn, Common neural and transcriptional correlates of inhibitory control underlie emotion regulation and memory control, *Social. Cogn. Affect. Neurosci.* 15 (5) (2020) 523–536, <https://doi.org/10.1093/scan/nsaa073>.
- [14] M.C. Anderson, K.N. Ochsner, B. Kuhl, J. Cooper, E. Robertson, S.W. Gabrieli, G. H. Glover, J.D.E. Gabrieli, Neural systems underlying the suppression of unwanted memories, *Science* 303 (5655) (2004) 232–235, <https://doi.org/10.1126/science.1089504>.
- [15] B.E. Depue, M.T. Banich, T. Curran, Suppression of emotional and nonemotional content in memory: effects of repetition on cognitive control, *Psychol. Sci.* 17 (5) (2006) 441–447, <https://doi.org/10.1111/j.1467-9280.2006.01725.x>.
- [16] W. Yang, H. Jia, Q. Feng, D. Wei, J. Qiu, J.C. Hulbert, Functional connectivity between right-lateralized ventrolateral prefrontal cortex and insula mediates reappraisal's link to memory control, *J. Affect. Disord.* 290 (2021) 316–323, <https://doi.org/10.1016/j.jad.2021.04.057>.
- [17] W. Yang, K. Zhuang, P. Liu, Y. Guo, Q. Chen, D. Wei, J. Qiu, Memory suppression ability can be robustly predicted by the internetwork communication of frontoparietal control network, *Cereb. Cortex* 31 (7) (2021) 3451–3461, <https://doi.org/10.1093/cercor/bhab024>.
- [18] Y. Yan, J.C. Hulbert, K. Zhuang, W. Liu, D. Wei, J. Qiu, M.C. Anderson, W. Yang, Reduced hippocampal-cortical connectivity during memory suppression predicts the ability to forget unwanted memories, *Cereb. cortex (New. Y. N. Y.)* 33 (8) (2023) 4189–4201, <https://doi.org/10.1093/cercor/bhac336>.
- [19] D. Nardo, M.C. Anderson, Everything you ever wanted to know about the Think/No-Think task, but forgot to ask, *Behav. Res. Methods* 56 (4) (2024) 3831–3860, <https://doi.org/10.3758/s13428-024-02349-9>.
- [20] Wenjing Yang, Liu Qi, Jia Hui, The effect of active forgetting on negative emotion and its cognitive mechanism, *J. Psychol. Sci.* 47 (5) (2024) 1069–1079, <https://doi.org/10.16719/j.cnki.1671-6981.20240505>.
- [21] P. Gagnepain, J. Hulbert, M.C. Anderson, Parallel regulation of memory and emotion supports the suppression of intrusive memories, *J. Neurosci.* 37 (27) (2017) 6423–6441, <https://doi.org/10.1523/JNEUROSCI.2732-16.2017>.
- [22] K.J. Hunt, L.K. Knight, B.E. Depue, Related neural networks underlie suppression of emotion, memory, motor processes as identified by data-driven analysis, *BMC Neurosci.* 24 (1) (2023) 44, <https://doi.org/10.1186/s12868-023-00812-5>.
- [23] Y. Wang, Z. Zhu, J. Hu, D. Schiller, J. Li, Active suppression prevents the return of threat memory in humans, *Commun. Biol.* 4 (1) (2021) 609, <https://doi.org/10.1038/s42003-021-02120-2>.
- [24] M.C. Anderson, M. Crespo-Garcia, S. Subbulakshmi, Brain mechanisms underlying the inhibitory control of thought, *Nat. Rev. Neurosci.* 26 (7) (2025) 415–437, <https://doi.org/10.1038/s41583-025-00929-y>.
- [25] J.R. Wessel, M.C. Anderson, Neural mechanisms of domain-general inhibitory control, *Trends Cogn. Sci.* 28 (2) (2024) 124–143, <https://doi.org/10.1016/j.tics.2023.09.008>.
- [26] D.F. Stramaccia, A.-K. Meyer, K.M. Rischer, J.M. Fawcett, R.G. Benoit, Memory suppression and its deficiency in psychological disorders: a focused meta-analysis, *J. Exp. Psychol. General.* 150 (5) (2021) 828–850, <https://doi.org/10.1037/xge0000971>.
- [27] A. Bachfischer, I.M. Harris, Controlling unwanted memories: a conceptual review grounded in the process model of emotion regulation, *Psychon. Bull. Rev.* (2025), <https://doi.org/10.3758/s13423-025-02745-y>.
- [28] A. Chalkia, N. Schroyens, L. Leng, N. Vanhasbroeck, A.K. Zenses, L. Van Oudenhove, T. Beckers, No persistent attenuation of fear memories in humans: a registered replication of the reactivation-extinction effect, *Cortex* 129 (2020) 496–509, <https://doi.org/10.1016/j.cortex.2020.04.017>.
- [29] M.G. Craske, N. Treanor, C.C. Conway, T. Zbozinek, B. Vervliet, Maximizing exposure therapy: an inhibitory learning approach, *Behav. Res. Ther.* 58 (2014) 10–23, <https://doi.org/10.1016/j.brat.2014.04.006>.
- [30] T.E. Davis, J. Brennan, Specific phobia, fear, and the autism spectrum in children and adolescents: adapting OST for ASD, *Clin. Child. Fam. Psychol. Rev.* 27 (2) (2024) 509–522, <https://doi.org/10.1007/s10567-024-00475-6>.
- [31] R.J. Jacoby, J.S. Abramowitz, Inhibitory learning approaches to exposure therapy: A critical review and translation to obsessive-compulsive disorder, *Clin. Psychol. Rev.* 49 (2016) 28–40, <https://doi.org/10.1016/j.cpr.2016.07.001>.
- [32] A. Koskina, I.C. Campbell, U. Schmidt, Exposure therapy in eating disorders revisited, *Neurosci. & Biobehav. Rev.* 37 (2) (2013) 193–208, <https://doi.org/10.1016/j.neubiorev.2012.11.010>.
- [33] X. Lin, D. Chen, J. Liu, Z. Yao, H. Xie, M.C. Anderson, X. Hu, Observing the suppression of individual aversive memories from conscious awareness, *Cereb. cortex (New. Y. N. Y.)* 34 (6) (2024) bhac080, <https://doi.org/10.1093/cercor/bhac080>.
- [34] D. Moreau, B. Gamble, Conducting a meta-analysis in the age of open science: Tools, tips, and practical recommendations, *Psychol. Methods* 27 (3) (2022) 426–432, <https://doi.org/10.1037/met0000351>.
- [35] M.A. Fullana, A. Albajes-Eizaguirre, C. Soriano-Mas, B. Vervliet, N. Cardoner, O. Benet, J. Radua, B.J. Harrison, Fear extinction in the human brain: a meta-analysis of fMRI studies in healthy participants, *Neurosci. Biobehav. Rev.* 88 (2018) 16–25, <https://doi.org/10.1016/j.neubiorev.2018.03.002>.
- [36] S.B. Eickhoff, D. Bzdok, A.R. Laird, F. Kurth, P.T. Fox, Activation likelihood estimation meta-analysis revisited, *NeuroImage* 59 (3) (2012) 2349–2361, <https://doi.org/10.1016/j.neuroimage.2011.09.017>.
- [37] J.L. Lancaster, D. Tordesillas-Gutiérrez, M. Martínez, F. Salinas, A. Evans, K. Zilles, J.C. Mazziotta, P.T. Fox, Bias between MNI and Talairach coordinates analyzed using the ICBM-152 brain template, *Human. Brain Mapp.* 28 (11) (2007) 1194–1205, <https://doi.org/10.1002/hbm.20345>.
- [38] P.E. Turkeltaub, G.F. Eden, K.M. Jones, T.A. Zeffiro, Meta-analysis of the functional neuroanatomy of single-word reading: method and validation, *NeuroImage* 16 (3) (2002) 765–780, <https://doi.org/10.1006/nimg.2002.1131>.
- [39] V.I. Müller, E.C. Cieslik, A.R. Laird, P.T. Fox, J. Radua, D. Mataix-Cols, C. R. Tench, T. Yarkoni, T.E. Nichols, P.E. Turkeltaub, T.D. Wager, S.B. Eickhoff, Ten simple rules for neuroimaging meta-analysis, *Neurosci. & Biobehav. Rev.* 84 (2018) 151–161, <https://doi.org/10.1016/j.neubiorev.2017.11.012>.
- [40] S.B. Eickhoff, A.R. Laird, P.M. Fox, J.L. Lancaster, P.T. Fox, Implementation errors in the GingerALE Software: description and recommendations, *Human. Brain Mapp.* 38 (1) (2017) 7–11, <https://doi.org/10.1002/hbm.23342>.
- [41] S.B. Eickhoff, D. Bzdok, A.R. Laird, C. Roski, S. Caspers, K. Zilles, P.T. Fox, Coactivation patterns distinguish cortical modules, their connectivity and functional differentiation, *NeuroImage* 57 (3) (2011) 938–949, <https://doi.org/10.1016/j.neuroimage.2011.05.021>.
- [42] S.B. Eickhoff, S. Jbabdi, S. Caspers, A.R. Laird, P.T. Fox, K. Zilles, T.E.J. Behrens, Anatomical and functional connectivity of cytoarchitectonic areas within the human parietal operculum, *J. Neurosci.* 30 (18) (2010) 6409–6421, <https://doi.org/10.1523/JNEUROSCI.5664-09.2010>.
- [43] R. Langner, C. Rottschy, A.R. Laird, P.T. Fox, S.B. Eickhoff, Meta-analytic connectivity modeling revisited: Controlling for activation base rates, *NeuroImage* 99 (2014) 559–570, <https://doi.org/10.1016/j.neuroimage.2014.06.007>.
- [44] R. Gan, Y. Qiu, J. Liao, Y. Zhang, J. Wu, X. Peng, T.M. Lee, R. Huang, Mapping the mentalizing brain: An ALE meta-analysis to differentiate the representation of social scenes and ages on theory of mind, *Neurosci. Biobehav. Rev.* 167 (2024) 105918, <https://doi.org/10.1016/j.neubiorev.2024.105918>.
- [45] S.K. Meier, K.L. Ray, J.C. Mastan, S.R. Salvage, D.A. Robin, Meta-analytic connectivity modeling of deception-related brain regions, *PLOS ONE* 16 (8) (2021) e0248909, <https://doi.org/10.1371/journal.pone.0248909>.
- [46] G. Papitto, A.D. Friederici, E. Zaccarella, The topographical organization of motor processing: An ALE meta-analysis on six action domains and the relevance of Broca's region, *NeuroImage* 206 (2020) 116321, <https://doi.org/10.1016/j.neuroimage.2019.116321>.
- [47] Y. Huang, J. Huang, L. Li, T. Lin, L. Zou, Neural network of metaphor comprehension: an ALE meta-analysis and MACM analysis, *Cereb. Cortex* 33 (21) (2023) 10918–10930, <https://doi.org/10.1093/cercor/bhad337>.
- [48] B.T. Thomas Yeo, F.M. Krienen, J. Sepulcre, M.R. Sabuncu, D. Lashkari, M. Hollinshead, J.L. Roffman, J.W. Smoller, L. Zöllei, J.R. Polimeni, B. Fischl, H. Liu, R.L. Buckner, The organization of the human cerebral cortex estimated by intrinsic functional connectivity, *J. Neurophysiol.* 106 (3) (2011) 1125–1165, <https://doi.org/10.1152/jn.00338.2011>.
- [49] A. Schaefer, R. Kong, E.M. Gordon, T.O. Laumann, X.-N. Zuo, A.J. Holmes, S. B. Eickhoff, B.T.T. Yeo, Local-global parcellation of the human cerebral cortex from intrinsic functional connectivity MRI, *Cereb. Cortex* 28 (9) (2018) 3095–3114, <https://doi.org/10.1093/cercor/bhx179>.
- [50] J. Li, R. Zhang, S. Liu, Q. Liang, S. Zheng, X. He, R. Huang, Human spatial navigation: Neural representations of spatial scales and reference frames obtained from an ALE meta-analysis, *NeuroImage* 238 (2021) 118264, <https://doi.org/10.1016/j.neuroimage.2021.118264>.
- [51] Y. Tie, L. Rigolo, I.H. Norton, R.Y. Huang, W. Wu, D. Orringer, S. Mukundan, A. J. Golby, Defining language networks from resting-state fMRI for surgical planning—A feasibility study, *Human. Brain Mapp.* 35 (3) (2014) 1018–1030, <https://doi.org/10.1002/hbm.22231>.
- [52] L. Faul, D. Stjepanović, J.M. Stivers, G.W. Stewart, J.L. Graner, R.A. Morey, K. S. LaBar, Proximal threats promote enhanced acquisition and persistence of reactive fear-learning circuits, *Proc. Natl. Acad. Sci.* 117 (28) (2020) 16678–16689, <https://doi.org/10.1073/pnas.2004258117>.
- [53] C. Gramsch, J. Kattoor, A. Icenhour, M. Forsting, M. Schedlowski, E.R. Gizewski, S. Elsenbruch, Learning pain-related fear: neural mechanisms mediating rapid differential conditioning, extinction and reinstatement processes in human visceral pain, *Neurobiol. Learn. Mem.* 116 (2014) 36–45, <https://doi.org/10.1016/j.nlm.2014.08.003>.
- [54] B. Hagedorn, O.T. Wolf, C.J. Merz, Cortisol before extinction generalization alters its neural correlates during retrieval, *Psychoneuroendocrinology* 136 (2022) 105607, <https://doi.org/10.1016/j.psyneuen.2021.105607>.
- [55] A.C. Hennings, M. McClay, J.A. Lewis-Peacock, J.E. Dunsmoor, Contextual reinstatement promotes extinction generalization in healthy adults but not PTSD, *Neuropsychologia* 147 (2020) 107573, <https://doi.org/10.1016/j.neuropsychologia.2020.107573>.
- [56] A. Hermann, R. Stark, M.R. Milad, C.J. Merz, Renewal of conditioned fear in a novel context is associated with hippocampal activation and connectivity, *Social. Cogn. Affect. Neurosci.* 11 (9) (2016) 1411–1421, <https://doi.org/10.1093/scan/nsw047>.
- [57] M. Hollandt, A. Wroblewski, Y. Yang, I.C. Ridderbusch, T. Kircher, A.O. Hamm, B. Straube, J. Richter, Facilitating translational science in anxiety disorders by

- adjusting extinction training in the laboratory to exposure-based therapy procedures, *Transl. Psychiatry* 10 (1) (2020) 110, <https://doi.org/10.1038/s41398-020-0786-x>.
- [58] D.J. Holt, G. Coombs, M.A. Zeidan, D.C. Goff, M.R. Milad, Failure of Neural Responses to Safety Cues in Schizophrenia, *Arch. General. Psychiatry* 69 (9) (2012) 893, <https://doi.org/10.1001/archgenpsychiatry.2011.2310>.
- [59] J. Kattoor, E.R. Gizewski, V. Kotsis, S. Benson, C. Gramsch, N. Theysohn, S. Maderwald, M. Forsting, M. Schedlowski, S. Elsenbruch, Fear Conditioning in an Abdominal Pain Model: Neural Responses during Associative Learning and Extinction in Healthy Subjects, *PLoS ONE* 8 (2) (2013) e51149, <https://doi.org/10.1371/journal.pone.0051149>.
- [60] V.L. Kinner, O.T. Wolf, C.J. Merz, Cortisol increases the return of fear by strengthening amygdala signaling in men, *Psychoneuroendocrinology* 91 (2018) 79–85, <https://doi.org/10.1016/j.psyneuen.2018.02.020>.
- [61] N. Levar, J.M.C. Van Leeuwen, N.A.J. Puts, D. Denys, G.A. Van Wingen, GABA Concentrations in the Anterior Cingulate Cortex Are Associated with Fear Network Function and Fear Recovery in Humans, *Front. Human. Neurosci.* 11 (2017) 202, <https://doi.org/10.3389/fnhum.2017.00202>.
- [62] S. Maier, A. Szalkowski, S. Kamphausen, E. Perlov, B. Feige, J. Blechert, A. Philipsen, L.T. Van Elst, R. Kalisch, O. Tüscher, Clarifying the role of the rostral dmPFC/dACC in fear/anxiety: learning, appraisal or expression? *PLoS ONE* 7 (11) (2012) e50120 <https://doi.org/10.1371/journal.pone.0050120>.
- [63] C.J. Merz, A. Hermann, R. Stark, O.T. Wolf, Cortisol modifies extinction learning of recently acquired fear in men, *Social. Cogn. Affect. Neurosci.* 9 (9) (2014) 1426–1434, <https://doi.org/10.1093/scan/nst137>.
- [64] S.T. Pohlack, F. Nees, M. Ruttorf, L.R. Schad, H. Flor, Activation of the ventral striatum during aversive contextual conditioning in humans, *Biol. Psychol.* 91 (1) (2012) 74–80, <https://doi.org/10.1016/j.biopsycho.2012.04.004>.
- [65] I.C. Ridderbusch, A. Wroblewski, Y. Yang, J. Richter, M. Hollandt, A.O. Hamm, H.-U. Wittchen, A. Ströhle, V. Arolt, J. Margraf, U. Lueken, M.J. Herrmann, T. Kircher, B. Straube, Neural adaptation of cingulate and insular activity during delayed fear extinction: a replicable pattern across assessment sites and repeated measurements, *NeuroImage* 237 (2021) 118157, <https://doi.org/10.1016/j.neuroimage.2021.118157>.
- [66] C. Sehmeyer, U. Dannlowski, S. Schöning, H. Kugel, M. Pyka, B. Pfeleiderer, P. Zwitterlood, H. Schifflbauer, W. Heindel, V. Arolt, C. Konrad, Neural correlates of trait anxiety in fear extinction, *Psychol. Med.* 41 (4) (2011) 789–798, <https://doi.org/10.1017/S0033291710001248>.
- [67] J. Seo, K.N. Moore, S. Gazecki, R.M. Bottary, M.R. Milad, H. Song, E.F. Pace-Schott, Delayed fear extinction in individuals with insomnia disorder, *Sleep* 41 (8) (2018), <https://doi.org/10.1093/sleep/zsy095>.
- [68] J. Spohrs, M. Ulrich, G. Grön, M. Prost, P.L. Plener, J.M. Fegert, L. Bindila, B. Ahler, Fear extinction learning and anandamide: an fMRI study in healthy humans, *Transl. Psychiatry* 11 (1) (2021) 161, <https://doi.org/10.1038/s41398-020-01177-7>.
- [69] V.I. Spoomaker, A. Sturm, K.C. Andrade, M.S. Schröter, R. Goya-Maldonado, F. Holsboer, T.C. Wetter, P.G. Sämann, M. Czisch, The neural correlates and temporal sequence of the relationship between shock exposure, disturbed sleep and impaired consolidation of fear extinction, *J. Psychiatr. Res.* 44 (16) (2010) 1121–1128, <https://doi.org/10.1016/j.jpsychires.2010.04.017>.
- [70] R.M. Visser, J. Bathelt, H.S. Scholte, M. Kindt, Robust BOLD responses to faces but not to conditioned threat: challenging the amygdala's reputation in human fear and extinction learning, *J. Neurosci.* 41 (50) (2021) 10278–10292, <https://doi.org/10.1523/JNEUROSCI.0857-21.2021>.
- [71] A. Wroblewski, M. Hollandt, Y. Yang, I.C. Ridderbusch, A. Pietzner, C. Szeska, M. Lotze, H.-U. Wittchen, I. Heinig, A. Pittig, V. Arolt, K. Koelbeck, C. A. Rothkopf, D. Adolph, J. Margraf, U. Lueken, P. Pauli, M.J. Herrmann, M. H. Winkler, J. Richter, Sometimes I feel the fear of uncertainty: how intolerance of uncertainty and trait anxiety impact fear acquisition, extinction and the return of fear, *Int. J. Psychophysiol.* 181 (2022) 125–140, <https://doi.org/10.1016/j.ijpsycho.2022.09.001>.
- [72] R.G. Benoit, M.C. Anderson, Opposing mechanisms support the voluntary forgetting of unwanted memories, *Neuron* 76 (2) (2012) 450–460, <https://doi.org/10.1016/j.neuron.2012.07.025>.
- [73] R.G. Benoit, J.C. Hulbert, E. Huddleston, M.C. Anderson, Adaptive top-down suppression of hippocampal activity and the purging of intrusive memories from consciousness, *J. Cogn. Neurosci.* 27 (1) (2015) 96–111, [https://doi.org/10.1162/jocn\\_a.00696](https://doi.org/10.1162/jocn_a.00696).
- [74] B.E. Depue, G.C. Burgess, E.G. Willcutt, L. Ruzic, M.T. Banich, Inhibitory control of memory retrieval and motor processing associated with the right lateral prefrontal cortex: evidence from deficits in individuals with ADHD, *Neuropsychologia* 48 (13) (2010) 3909–3917, <https://doi.org/10.1016/j.neuropsychologia.2010.09.013>.
- [75] P. Gagnepain, R.N. Henson, M.C. Anderson, Suppressing unwanted memories reduces their unconscious influence via targeted cortical inhibition, *Proc. Natl. Acad. Sci.* 111 (13) (2014), <https://doi.org/10.1073/pnas.1311468111>.
- [76] J.C. Hulbert, R.N. Henson, M.C. Anderson, Inducing amnesia through systemic suppression, *Nat. Commun.* 7 (1) (2016) 11003, <https://doi.org/10.1038/ncomms11003>.
- [77] B.J. Levy, M.C. Anderson, Purging of Memories from Conscious Awareness Tracked in the Human Brain, *J. Neurosci.* 32 (47) (2012) 16785–16794, <https://doi.org/10.1523/JNEUROSCI.2640-12.2012>.
- [78] W. Liu, N. Kohn, G. Fernández, Dynamic Transitions between Neural States Are Associated with Flexible Task Switching during a Memory Task, *J. Cogn. Neurosci.* 33 (12) (2021) 2559–2588, [https://doi.org/10.1162/jocn\\_a.01779](https://doi.org/10.1162/jocn_a.01779).
- [79] A. Mary, J. Dayan, G. Leone, C. Postel, F. Fraisse, C. Malle, T. Vallée, C. Klein-Peschanski, F. Viader, V. De La Sayette, D. Peschanski, F. Eustache, P. Gagnepain, Resilience after trauma: The role of memory suppression, *Science* 367 (6479) (2020) eaay8477, <https://doi.org/10.1126/science.aay8477>.
- [80] M.D. Sacchet, B.J. Levy, J.P. Hamilton, A. Maksimovskiy, P.T. Hertel, J. Joormann, M.C. Anderson, A.D. Wagner, I.H. Gotlib, Cognitive and neural consequences of memory suppression in major depressive disorder, *Cogn. Affect. Behav. Neurosci.* 17 (1) (2017) 77–93, <https://doi.org/10.3758/s13415-016-0464-x>.
- [81] R. Smith, A. Alkozei, J. Bao, W.D.S. Killgore, Successful goal-directed memory suppression is associated with increased inter-hemispheric coordination between right and left frontoparietal control networks, *Psychol. Rep.* 121 (1) (2018) 93–111, <https://doi.org/10.1177/0033294117723018>.
- [82] C.M. Dodds, S. Morein-Zamir, T.W. Robbins, Dissociating inhibition, attention, and response control in the frontoparietal network using functional magnetic resonance imaging, *Cereb. Cortex* 21 (5) (2011) 1155–1165, <https://doi.org/10.1093/cercor/bhq187>.
- [83] J. Long, X. Song, Y. Wang, C. Wang, R. Huang, R. Zhang, Distinct neural activation patterns of age in subcomponents of inhibitory control: A fMRI meta-analysis, *Front. Aging Neurosci.* 14 (2022) 938789, <https://doi.org/10.3389/fnagi.2022.938789>.
- [84] H. Namkung, S.-H. Kim, A. Sawa, The Insula: An Underestimated Brain Area in Clinical Neuroscience, Psychiatry, and Neurology, *Trends Neurosci.* 41 (8) (2018) 551–554, <https://doi.org/10.1016/j.tins.2018.05.004>.
- [85] I. Molnar-Szakacs, L.Q. Uddin, Anterior insula as a gatekeeper of executive control, *Neurosci. & Biobehav. Rev.* 139 (2022) 104736, <https://doi.org/10.1016/j.neubiorev.2022.104736>.
- [86] O. Numssen, D. Bzdok, G. Hartwigsen, Functional specialization within the inferior parietal lobes across cognitive domains, *eLife* 10 (2021) e63591, <https://doi.org/10.7554/eLife.63591>.
- [87] J. Choi, H. Cho, J.-S. Choi, I.Y. Choi, J.-W. Chun, D.-J. Kim, The neural basis underlying impaired attentional control in problematic smartphone users, *Transl. Psychiatry* 11 (1) (2021) 129, <https://doi.org/10.1038/s41398-021-01246-5>, <https://www.nature.com/articles/s41398-021-01246-5>.
- [88] J. Long, L. Peng, Q. Li, L. Niu, H. Dai, J. Zhang, K. Chen, T.M. Lee, M. Huang, R. Zhang, Acute stress impairs intentional memory suppression through aberrant prefrontal cortex activation in high trait ruminators, *Int. J. Clin. Health Psychol.* 24 (2) (2024) 100463, <https://doi.org/10.1016/j.ijchp.2024.100463>.
- [89] S. Ghetti, S.A. Bunge, Neural changes underlying the development of episodic memory during middle childhood, *Dev. Cogn. Neurosci.* 2 (4) (2012) 381–395, <https://doi.org/10.1016/j.dcn.2012.05.002>.
- [90] W. Le Duc, C.R. Butler, G.P.D. Argyropoulos, S. Chu, C. Hutcherson, A.C. Ruocco, R. Ito, A.C.H. Lee, Hippocampal damage disrupts the latent decision-making processes underlying approach-avoidance conflict processing in humans, *PLOS Biol.* 23 (2) (2025) e3003033, <https://doi.org/10.1371/journal.pbio.3003033>.
- [91] M. Alexandra Kredlow, R.J. Fenster, E.S. Laurent, K.J. Ressler, E.A. Phelps, Prefrontal cortex, amygdala, and threat processing: implications for PTSD, *Neuropsychopharmacology* 47 (1) (2021) 247–259, <https://doi.org/10.1038/s41386-021-01155-7>, <https://www.nature.com/articles/s41386-021-01155-7>.
- [92] B.E. Depue, T. Curran, M.T. Banich, Prefrontal Regions Orchestrate Suppression of Emotional Memories via a Two-Phase Process, *Science* 317 (5835) (2007) 215–219, <https://doi.org/10.1126/science.1139560>, <https://www.science.org/gdoi/10.1126/science.1139560>.
- [93] Y. Liu, W. Lin, C. Liu, Y. Luo, J. Wu, P.J. Bayley, S. Qin, Memory consolidation reconfigures neural pathways involved in the suppression of emotional memories, *Nat. Commun.* 7 (1) (2016) 13375, <https://doi.org/10.1038/ncomms13375>.
- [94] A.-K. Meyer, R.G. Benoit, Suppression weakens unwanted memories via a sustained reduction of neural reactivation, *eLife* 11 (2022) e71309, <https://doi.org/10.7554/eLife.71309>.
- [95] W. Yang, P. Liu, K. Zhuang, D. Wei, M.C. Anderson, J. Qiu, Behavioral and neural correlates of memory suppression in subthreshold depression, *Psychiatry Res. Neuroimaging* 297 (2020) 111030, <https://doi.org/10.1016/j.psychres.2020.111030>.
- [96] M.K.P. Joyce, S. Uchendu, A.F.T. Arnsten, Stress and Inflammation Target Dorsolateral Prefrontal Cortex Function: Neural Mechanisms Underlying Weakened Cognitive Control, *Biol. Psychiatry* 97 (4) (2025) 359–371, <https://doi.org/10.1016/j.biopsycho.2024.06.016>.
- [97] L. Brown, L.K. White, W. Makhoul, M. Teferi, Y.I. Sheline, N.L. Balderston, Role of the intraparietal sulcus (IPS) in anxiety and cognition: opportunities for intervention for anxiety-related disorders, *Int. J. Clin. Health Psychol.* 23 (4) (2023) 100385, <https://doi.org/10.1016/j.ijchp.2023.100385>.
- [98] S.Y. Coll, E. Marti, N. Doganci, R. Prak, Functional connectivity of the dorsal and ventral attention network and its role in attentional disengagement, *Brain Behav.* 15 (10) (2025) e70868, <https://doi.org/10.1002/brb3.70868>.
- [99] C.A. Cushing, Y. Peng, Z. Anderson, K.S. Young, S.Y. Bookheimer, R.E. Zinbarg, R. Nusslock, M.G. Craske, Broadening the scope: Multiple functional connectivity networks underlying threat conditioning and extinction, *Imaging Neurosci.* 2 (2024) imag-2-00213, [https://doi.org/10.1162/imag\\_a.00213](https://doi.org/10.1162/imag_a.00213), [https://direct.mit.edu/imag/article/doi/10.1162/imag\\_a.00213/122335/Broadening-the-scope-Multiple-functional](https://direct.mit.edu/imag/article/doi/10.1162/imag_a.00213/122335/Broadening-the-scope-Multiple-functional).
- [100] W. Zhang, Y. Huang, Y. Mei, J. Wang, H. Dou, Y. Lei, Executive functions modulate conditioned fear extinction and reinstatement: the differential roles of shifting, updating, and inhibition, *Int. J. Clin. Health Psychol.* 25 (2) (2025) 100581, <https://doi.org/10.1016/j.ijchp.2025.100581>.
- [101] B.E. Depue, J.M. Orr, H.R. Smolker, F. Naaz, M.T. Banich, The organization of right prefrontal networks reveals common mechanisms of inhibitory regulation

- across cognitive, emotional, and motor processes, *Cereb. Cortex* 26 (4) (2016) 1634–1646, <https://doi.org/10.1093/cercor/bhu324>.
- [102] S. Battaglia, C. Nazzi, T.B. Lonsdorf, J.F. Thayer, Neuropsychobiology of fear-induced bradycardia in humans: progress and pitfalls, *Mol. Psychiatry* 29 (12) (2024) 3826–3840, <https://doi.org/10.1038/s41380-024-02600-x>.
- [103] S. Garofalo, M.E. Maier, G. di Pellegrino, Medial frontal negativity signals unexpected omission of aversive events, *Sci. Rep.* 4 (2014) 4816, <https://doi.org/10.1038/srep04816>.
- [104] J. Björkstrand, T. Agren, A. Frick, J. Engman, E.M. Larsson, T. Furmark, M. Fredrikson, Disruption of memory reconsolidation erases a fear memory trace in the human amygdala: an 18-month follow-up, *PLoS One* 10 (7) (2015) e0129393, <https://doi.org/10.1371/journal.pone.0129393>.
- [105] L.F. Gruss, A. Keil, Sympathetic responding to unconditioned stimuli predicts subsequent threat expectancy, orienting, and visuocortical bias in human aversive Pavlovian conditioning, *Biol. Psychol.* 140 (2019) 64–74, <https://doi.org/10.1016/j.biopsycho.2018.11.009>.
- [106] S. Maren, K.L. Phan, I. Liberzon, The contextual brain: implications for fear conditioning, extinction and psychopathology, *Nat. Rev. Neurosci.* 14 (6) (2013) 417–428, <https://doi.org/10.1038/nrn3492>.
- [107] A. Santos-Mayo, S. Moratti, How fear conditioning affects the visuocortical processing of context cues in humans: evidence from steady state visual evoked responses, *Cortex* 183 (2025) 21–37, <https://doi.org/10.1016/j.cortex.2024.11.002>.