

FMRI network dynamics underpinning the impact of affective carry-over on cognitive control

Julian Gaviria ^{*a,b,c}. ORCID ID: 0000-0002-4266-1371

Gwladys Rey ^d. ORCID ID: 0000-0001-7192-1577

Matt L. Miller ^e. ORCID ID: 0000-0002-2377-6325

Thomas Bolton^f. ORCID ID: 0000-0002-2081-4031

Dimitri Van De Ville ^{g, h}. ORCID ID: 0000-0002-2879-3861

Patrik Vuilleumierⁱ. ORCID ID: 0000-0002-8198-9214

Authors

^aDepartment of Psychiatry, Amsterdam UMC; ^bAmsterdam Neuroscience, Amsterdam UMC; ^cVrije Universiteit, Amsterdam, The Netherlands; ^dMaster of Advanced Studies (MAS) in Neuropsychology, University of Geneva, Switzerland; ^eREACH Institute, Arizona State University, USA; ^fdepartment of Psychiatry, University of Geneva, Switzerland; ^gMedical image processing lab, Neuro-X Institute, Ecole polytechnique fédérale de Lausanne (EPFL), Switzerland; ^hDepartment of radiology and medical informatics, University of Geneva, Switzerland. ⁱLaboratory for behavioral neurology and Imaging of cognition, University of Geneva, Switzerland.

*Correspondence

Julian Gaviria Lopez. E-mail: jualgalo@gmail.com;

Keywords

Brain networks; emotions; dynamic functional connectivity (dFC); Task-rest association. Cognitive control

Abstract

Behavioral research has extensively documented the impact of affective states on cognitive control. Despite increasing interest in investigating how affect and emotion influence cognition, the functional neural architecture mediating these effects remains unresolved. Here, we examined how changes in brain network dynamics elicited by negative emotions modulate the subsequent recruitment of attentional processes at behavioral and neural levels. We collected functional magnetic resonance imaging (fMRI) BOLD activity in healthy humans during three sequential conditions (naturalistic movie watching, resting state, and cognitive control task) in either neutral or negative emotion contexts. We assessed fMRI data by using co-activation patterns (CAPs) analysis to characterize dynamic functional connectivity within whole-brain networks across experimental conditions, and then applied structural equation modelling to uncover their functional relationships. We found that neural markers of cognitive control featured by the frontoparietal control network (FPN) during cognitive control were modulated by prior occurrences of distinct activity patterns after negative emotions, involving (1) the salience (SN) and sensorimotor (SMN) networks during the emotion-eliciting event itself, and (2) the default mode (DMN), salience (SN), and sensorimotor (SMN) networks in a resting period following emotional elicitation. Further, these neural associations were reflected by concomitant changes in behavioral measures of cognitive control after negative emotion. Altogether, our results provide new insights into the brain dynamic functional organization supporting high-order cognitive processing and its regulation by negative emotions.

Introduction

Emotional episodes often spill over to subsequent mental state and cognitive processing, leading to adaptive changes across a range of domains from perception through to memory or attention¹⁻⁴, but also to detrimental effects such as rumination, decision biases, or mood disorders (see reviews⁵⁻⁷). These observations converge with the view that, rather than constituting distinct and independent systems, emotion and cognition involve intimately interconnected and highly interacting processes that largely overlap in their neural underpinnings⁸⁻¹¹. Thus, the functional architecture of emotion processing and cognitive control encompasses several shared networks of cortical and subcortical brain regions¹²⁻¹⁴. Nevertheless, the neural mechanisms of the relationship between emotion and cognition remain poorly understood.

A robust finding on emotion-cognition associations concerns changes in attention and executive control abilities induced by exposure to emotional information^{15,16}. Empirical evidence shows a differential influence of emotional valence on attentional conflict processing, whereby interference effects are more prominent in negative than positive mood, across various populations and different conflict paradigms¹⁷⁻²⁴. This is observed in response conflict situations, in which we must prioritize our thoughts and actions by enhancing the processing of relevant information and suppressing irrelevant information. Typical examples are the Stroop or Flanker paradigms²⁵, where negative affect increases conflict during response selection^{17,18}. However, although abundant research has been conducted on neural systems underpinning cognitive control outside emotional contexts (e.g., conflict monitoring theory²⁶ and outcome evaluation theory²⁷), less is known about their modulation by affective state.

Past research has generally highlighted the anterior cingulate cortex (ACC) as a key brain hub in detecting conflict signals and promoting subsequent behavioral changes²⁸. Accordingly, some neuroimaging studies found increased activation of dorsal anterior cingulate (dACC) areas when attentional conflict is exacerbated by negative affect²⁹. Beyond isolated brain regions, recent studies have explored intrinsic brain activity underlying cognitive control from a network perspective³⁰. The fronto-parietal control (FPN), ventral attention (VAN), and dorsal attention networks (DAN) have been postulated as a domain-general neural system underlying emotional influences on cognitive interference processing³¹.

Brain areas within the default mode network (DMN) also play an important role in both cognitive and affective functions, characterized by associations with the FPN and increased recruitment during emotionally intense experiences or negative moods. Moreover, the DMN has been linked to depressive rumination and cognitive deficits caused by inefficient top-down control of negative thoughts in psychopathological conditions. Despite these increasing insights into brain regions involved in the relationship between emotion and cognitive control (including DAN, VAN, FPN, and DMN), it is still unclear how brain activity patterns evoked during emotional episodes may trigger changes in cognitive control systems and how these changes persist over time to influence cognitive performance.

To elucidate the functional mechanisms underlying these phenomena, it is essential to better characterize the temporal aspects of emotional influences on cognitive control and the dynamics of functional connectivity (FC) among brain networks, in addition to their neuroanatomical correlates. Abundant empirical evidence shows that emotion processing may produce “online” modulations of attentional networks for emotion-eliciting events themselves, either by facilitating attention orienting

to emotional information³² or by distracting cognitive resources^{33–35} when emotional cues occur at the same time as task-relevant stimuli. These effects have been linked to direct projections from limbic brain regions, such as the amygdala, to both low-level sensory cortical areas and higher-level fronto-parietal areas^{36,37}. In addition, however, more sustained changes in brain activity and connectivity during perceptual or cognitive tasks are also observed when emotions precede these tasks, including delayed effects on face processing³⁸, selective attention³⁹, memory², or pain perception and empathy⁴⁰. Moreover, it has been shown that transient emotional episodes can induce long-lasting “carry-over” effects on the intrinsic functional networks (IFNs) at rest, e.g., **after** watching negative movies^{41–44} or perceiving aversive smells⁴⁵. Such emotional carry-over effects at rest include modulations of DMN activity and its connectivity with other IFNs⁴⁶, possibly accounting for emotion-induced or mood-dependent shifts in cognitive functions⁴⁷. Accordingly, affective processes imply dynamic fluctuations in brain state that unfold across multiple brain networks, which are not only engaged during emotion-eliciting events but also active before and after these events, and not only emotion-specific but recruited by various non-emotional processes or tasks. Changes in network dynamics evoked by emotional experiences might therefore also influence their engagement during subsequent cognitive performance and account for affective influences on behavior.

To investigate these issues and identify the neural mechanisms of affective influences on cognitive control, we recorded fMRI-BOLD activity in an emotion induction paradigm where we compared brain activity during both active cognitive processing and passive resting state conditions following exposure to either neutral or negatively valenced movies. This allowed us to examine how sustained emotional carry-over effects on brain state^{41,44} could influence neural and behavioral indices of cognitive control. Importantly, to assess the functional dynamics of reciprocal influences between

different brain network active at different points in time, we deployed a recently developed signal processing method to obtain quantitative temporal parameters, as well as anatomical characteristics of dynamic functional connectivity (dFC) across the whole brain. Specifically, we leveraged a co-activation patterns (CAPs) approach^{48,49} without any predefined seed region of interest (ROI) or functional brain parcellation. It permitted us to track dynamic fluctuations of network activity over time in a data-driven manner with voxel-wise resolution. We then probed functional relationships between CAPs using a Bayesian structural equation modeling (BSEM) approach. By characterizing transient brain-wide CAPs modulated as a function of emotion and subsequent cognitive control demands, and defining their reciprocal relationships over time, we could test three complementary hypotheses about the influence of emotions on cognition. Namely, that emotional effects on brain activity patterns during the cognitive control task might be related to (1) changes in dFC expressed in intrinsic/spontaneous activity at rest following emotion induction; (2) changes in dFC evoked during emotional appraisal itself; and (3) changes in behavioral performance measured during the cognitive control task.

Based on previous research, we expected that negative emotions would induce changes in distributed brain circuits, including the DMN typically expressed in resting state⁵⁰, as well as the SN, DAN and/or executive control networks (FPN), typically recruited by cognitive control demands⁵¹⁻⁵³. More critically, we predicted a significant relationship between the modulation of brain CAPs observed during negative movies and during resting state after negative movies as well as the subsequent impact of negative emotion on brain activity during the cognitive control tasks.

Materials and Methods

Participants

Commented [pv1]: Make sure to add the various clarifications / justifications asked by the reviewer as we describe in the rebuttal

A cohort of 87 participants was initially contacted and asked to fill out an online screening questionnaire. Among them, we selected thirty-two French-speaking female volunteers (mean age = 23.2 ± 4.3) based on the following inclusion criteria: No history of neurological and psychiatric diseases, right-handedness, no medication or drug intake, regular menstrual cycle, known hormonal phase, and no contraceptive method (to rule out hormonal effects on emotional processing⁵⁴ and functional connectivity^{55,56}). Only female participants were recruited because previous pilot testing ($N=28$) suggested stronger emotional induction in women, compared to males, particularly with the movie clips used here^{54,57}. In subsequent analyses, 8 participants were excluded due to head movement during scanning (>1.5 mm in all axes) and/or low performance in the cognitive task ($>20\%$ of trials without response). The final sample therefore consisted of 24 subjects. Participants provided written informed consent according to the cantonal research ethics committee (CCER), University of Geneva. Scanning was scheduled for each participant in the early stage of the menstrual follicular phase, when the levels of estradiol and progesterone hormones are moderate^{57,58}. Nicotine and caffeine consumption were prohibited 10 hours before scanning.

Experimental design

Participants initially practiced on two cognitive control tasks used in subsequent fMRI sessions (30 trials from the Flanker task and 30 from the Stroop task). Next, subjective affective state was assessed by the French version of the positive and negative Affect Schedule (PANAS). Participants then underwent fMRI scanning for approximately 60 minutes (**Figure 1A**). Two experimental contexts were sequentially presented in counterbalanced order across participants, involving different affective inductions with short movies. One movie condition induced emotions with negative valence (negative context), and the other movie condition was neutral (neutral context). These two experimental sessions were separated by a pause of about 20 minutes.

The full sequence of the paradigm (see **Figure 1A**) comprised 5 different blocks, given once in the neutral context and once in the negative context, in the following order: first short movie clip [“MOVIE1” condition (5 min)] followed by a resting period [“REST1” condition (5 min)], then second movie clip [“MOVIE2” condition (5 min)] with the same valence as the first, followed by a cognitive control task block [“TASK” condition (~5 min)]. Finally, there was a second resting period [“REST2” condition (5 min)], which corresponding data was analyzed elsewhere⁴⁷. The present study will focus only on REST1, MOVIE2, and TASK blocks. Individual ratings of the emotional content of each movie clip, as well as another measure of subjective affective state (PANAS) were obtained again at the end of each context.

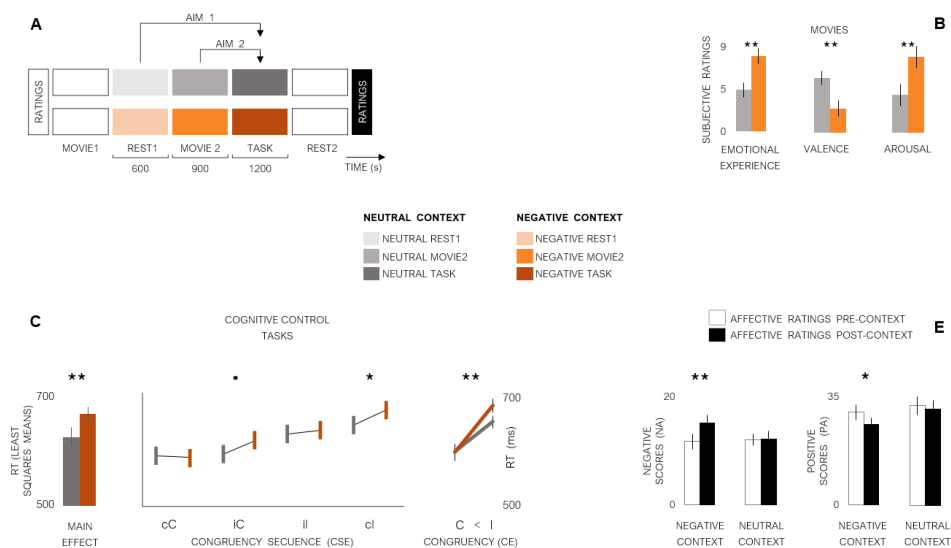


Figure 1. Paradigm design. **A.** Two experimental contexts (neutral and negative) were presented in two functional magnetic resonance imaging (fMRI) runs in a single day. The order of the two contexts was counterbalanced across participants. Each context consisted of 1) a first movie clip (MOVIE, 5 min.) followed by a rest period (REST1, 5 min.), then a second movie clip (MOVIE2, 5 min) of the same valence as the first followed by a cognitive control task (TASK, 5 min) and finally a second rest period (REST2). Colored blocks indicate experimental conditions of interest, with arrows indicating the main target of our analyses (see Figure 3). **B.** Subjective affective ratings of the movies obtained after fMRI scanning using unipolar Likert scales, assessing emotional experience, valence, and arousal felt for each movie clip. **C.** Behavioral performance in cognitive control trial types in each cognitive task, including congruent (C) and incongruent (I) trials that could be preceded by either the same or opposite condition (“c” or “i” trials), in a semi-random but balanced order. Left.

Commented [pv2]: Change the x axis of congruency graph as discussed

Global comparison of mean reaction time (RT) in negative (orange color) and neutral (gray color) contexts, pooled across conditions. Middle. RTs for correct responses on all trial types (congruent or incongruent conditions, preceded by the same or different condition), showing a significant attentional conflict on incongruent trials and consistent emotion-induced increases except for the easiest congruent trials. Right. A reliable attentional interference (congruency effect, CE) was found after exposure to both negative and neutral movies, but enhanced in the former “negative” context, as compared with the latter “neutral” context. **D.** Mean scores of two independent unipolar dimensions of affect: negative affect (NA) and positive affect (PA), before (pre-induction score) and after (post-induction score) each experimental run (negative and neutral context). Whiskers stand for standard deviation of means. * $p < .1$; * $p < .05$; ** $p < .01$.

Our paradigm design allowed us to address two main research aims. First, fMRI activity recorded during “REST1” allowed probing how negative emotions elicited by “MOVIE1” produce sustained changes in intrinsic brain network states (i.e., emotional carry-over or inertia), which could already be observed at rest^{41,47} and then impact subsequent task performance^{38,59}. Second, emotion induction by “MOVIE2” allowed us to strengthen negative affect further and examine how this modulates brain networks recruited by cognitive control demands in the “TASK” condition. Our nested repeated measures design therefore enabled us to test the impact of both negative and neutral contexts on brain dynamics and its consequence for cognitive control, in the same participants. The final “REST2” condition allowed measuring the sustained effects of both negative affect and cognitive effort on intrinsic brain network activity, but this block is not relevant to cognitive task performance in our current study and not reported here [see⁴⁷ for results on this part]. Detailed information on movies, affective ratings (PANAS), and behavioral tasks is given in SI, “Supplementary methods” section.

Cognitive control tasks

To test cognitive control performance in different affective contexts, we used two classic tasks with attentional interference, the Stroop and Flanker paradigms. Both tasks have been validated by previous behavioral studies^{17,18,60}. Each task (**Figure S1A**) comprised 80 trials (40 congruent and 40 incongruent). Each trial lasted 1s, followed by an inter-trial interval with a central fixation cross randomly jittering from 2 to 4.9s. Both tasks were counterbalanced across affective context

conditions and across participants. Each task combined congruent (C) and incongruent (I) trials in which a central target (name in Stroop, number in Flanker) was presented for a binary classification response (male/female for Stroop, odd/even for Flanker), together with a distractor corresponding to either the same (C) or opposite (I) response. In addition, a given trial could be preceded by either the same or opposite condition (C or I trials), in a semi-random but balanced order. This resulted in 4 trial types, allowing subsequent behavioral analysis according to both current compatibility (indicated by upper-case letters “C” and “I”) and compatibility of the preceding trial (indicated by lower-case letters “c” and “i”) (see **Figure S1B** for a schematic description). Sequential effects in trial order make iC and cI trials more difficult than cC or iI as both require a shift in the stimulus-response link⁶⁰.

The attentional control systems engaged by both paradigms have been extensively investigated in past research at both behavioral and brain levels, and shown to involve similar areas in fronto-parietal cortex, insula, and subcortical regions, generalizing across a variety of conflict monitoring conditions^{61–63}. Also, previous studies reported a high sensitivity of both tasks to negative emotional priming^{1,17,64}. Additionally, both paradigms offer the possibility to present many trials in short sessions, with minimal learning, and yield different measures including not only for the resolution of attentional conflict but also post-conflict adaptation across successive trials^{65,66}. Here, our selection and design of these two cognitive control tasks was carefully guided by extensive pilot testing outside fMRI, which allowed us to ensure robust and similar interference effects in both tasks, with and without emotional modulation, as directly replicated during the fMRI study (see “Results” section and **Figure S1C**). Using two different tasks also allowed us to avoid systematic order confound due to habituation / practice effects when comparing performance in two different affective contexts. Consequently, they were counterbalanced across conditions and participants (half performed the Stroop task in negative context and the Flanker task in neutral context, and vice versa for the other half). Detailed information about the two tasks is provided in the SI “Supplementary Methods” section. These tasks (Flanker or

Stroop) were always given after the second movie ("MOVIE2" condition) in each experimental context (see **Figure 1A**), since exerting control demands during the cognitive task prior to the first movie could have modified emotion regulation and emotion experience during this movie.

MRI data acquisition

Anatomical and functional whole-brain MRI data were acquired with a 3T scanner (Siemens TIM Trio) at the Brain & Behavior Laboratory, University of Geneva. A multiband-sequence was implemented, with a voxel size of 2mm isometric and a TR of 1.3s (see SI "Supplementary Methods" section for further description of both data acquisition and preprocessing).

FMRI brain analysis.

The present study focused on whether emotion-induced changes in brain networks recruited by high cognitive control demands ("TASK" condition) can be related to (1) the preceding modulation of brain networks engaged during the emotion-inducing events ("MOVIE2" condition), and/or (2) the changes in intrinsic network activity measured at rest ("REST2" condition). Accordingly, our approach comprised two complementary aims (graphically depicted in **Figure 1A**): 1) To link changes in CAPs following affective induction ("REST1" condition) with those observed during the cognitive control load ("TASK" condition); 2) To link changes in CAPs observed during affective induction itself ("MOVIE2" condition) to those observed during the cognitive control load ("TASK" condition). Please note we chose to have a second movie induction phase (MOVIE2) after the resting state condition and just before the task condition, rather than only one single affective induction phase (MOVIE1), in order to ensure robust emotion effects on task performance and avoid their progressive attenuation during the intervening rest period.

To these aims, our fMRI and behavioral data were analyzed using two successive stages. First, we performed a CAP-based analysis of dynamic functional connectivity to identify brain networks differentially engaged during each experimental condition. Second, we performed a Bayesian structural equation modeling (BSEM) analysis on functional relationships among brain CAPs active in various conditions, either directly between them or indirectly mediated by relevant behavioral measures, such as reaction time during cognitive control. These stages are outlined below.

Stage 1: Dynamic functional connectivity analysis (CAPs identification)

To examine changes in IFNs as a function of experimental conditions, we implemented a co-activation pattern (CAP) methodology^{67,68}. This approach provides a measure of dynamic, time-varying functional connectivity by decomposing fluctuations of the fMRI-BOLD signals into multiple spatial patterns that reflect the instantaneous organization and reorganization of networks co-activated over time. Unlike static connectivity analysis based on correlations by averaging over long periods, dFC analysis with CAPs accounts for the BOLD temporal variability by representing instantaneous brain configurations at single time points (fMRI volumes) and then sorting these configurations in a few dominant patterns through clustering^{48,69}. Given its single-volume temporal resolution, the CAP approach implies a minimal set of assumptions, constraints, and transformations of fMRI data⁴⁹. The single-volume temporal resolution also enables the exclusion of time points corrupted by noise without consequences for neighbor volumes⁷⁰. This cleaning strategy improves outcomes compared to classical analytical strategies that are more dependent on the time-series of fMRI volumes. Furthermore, recent research has offered insights into the neuronal origin of the signal fluctuations characterized by the CAPs⁷¹.

CAPs generation

Corrupted frames were first identified (framewise displacement > 5mm) and excluded from voxel-wise brain signals recorded across all time frames (1392 vols. TR= 1300 ms). Following motion correction, all fMRI volumes kept for analysis underwent k-means clustering to be separated into K different clusters (CAPs) in an agnostic fashion. Namely, neither the affective “context” (i.e., neutral, negative) nor the task “condition” (i.e., rest, movie, task) labelling were included in this stage. After clustering, volumes assigned to the same cluster were averaged, resulting in maps representing the spatial configuration of a given CAP. Each of these CAPs were then normalized by the standard error (within cluster and across fMRI volumes) to generate Z-statistic maps, which quantify the degree of significance to which the CAP map values (for each voxel) deviate from zero. Importantly, this approach defined not only the spatial configurations of dynamic brain networks (CAP maps), but also temporal metrics to quantify dFC variability over time, by computing the *occurrences*⁷² of each CAP in each condition. Occurrences are defined as the sum of fMRI frames (time-points) assigned to a given CAP among all the retained frames, across the entire scanning duration.

Our CAP analysis was computed with the “tbCAPs” toolbox⁴⁸. Here we implemented a seed-free method that classifies all time points (except the corrupted ones) with similar spatial distributions of activity, according to the k-means clustering algorithm. This approach extends the seed-based approach used in other studies^{44,46,69} to a data-driven, whole-brain analysis without restricting analysis to a limited set of networks. Neither aprioristic functional parcellations nor specific brain regions of interests (ROIs) were implemented in these analyses.

To determine the number of clusters used to define the CAPs, a consensus resampling-based algorithm⁷³ identified the number and membership (consensus) of reliable CAPs within our dataset. Parameters used to calculate the “consensus rate” between all pairs of samples included 80% of item resampling, a maximum k of 12, and 50 resamplings, with Euclidean indices as the distance

Commented [pv3]: Is this what you meant? Not just assessing?

measurement. The consensus values for different cluster solutions (0= samples are never clustered together across consensus folds; 1= samples are always clustered together) yielded K=5 as the optimal number of clusters. Subsequently, different K-means solutions were examined to identify partitions (i.e., CAPs) with the greatest consistency and reliability across different clustering scenarios. The results of this inter-clustering similarity analysis are described in **Figure S3**. A graphical description of our methodological procedure is found in **Figure 2**.

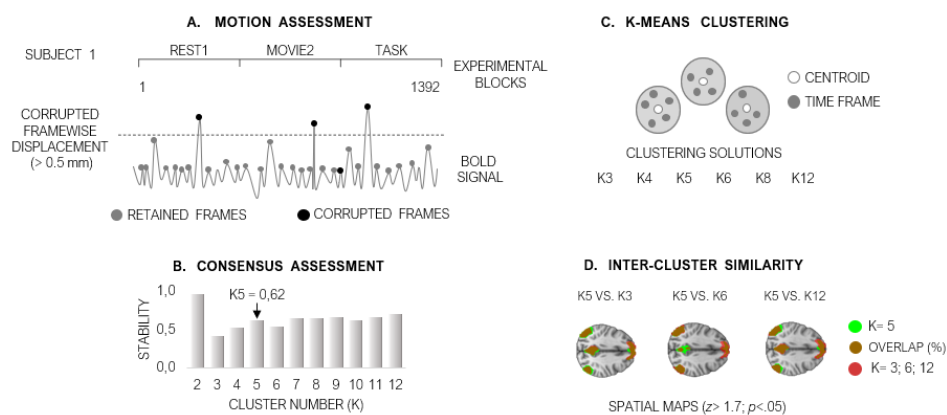


Figure 2. CAPs analysis pipeline of fMRI data (stage1). **A.** Assessment of corrupted frames across subjects and experimental blocks (1392 fMRI volumes) underwent k-means clustering to be separated into K different co-activation patterns (CAPs). Different K-means solutions were examined. **B.** Consensus assessment. **C.** Frames were compared in order to identify partitions (i.e., CAPs) with the greatest consistency and reliability across different clustering scenarios. Results of this inter-clustering similarity analysis are described in **Figure S3**.

Stage 2: Bayesian structural equation modeling (BSEM)

Multivariate BSEM was employed to examine the associations between particular dFC patterns, as this approach is adequate to deal with uncertainty in data due to high variability or small sample size⁷⁴. Hence it yields more interpretable results even when small to moderate sample sizes are modeled with fairly complex structural equation models^{75,76}. First, we examined the potential links between CAPs present during the “REST1” condition (reflecting affective “carry-over” following emotional

induction) and CAPs recruited during the cognitive control task ("TASK" condition). This analysis aimed at testing whether emotional effects on cognitive control networks could be predicted by changes in spontaneous dFC at rest. Second, we examined the links between CAPs modulated by negative emotional induction (vs neutral induction) in the "MOVIE2" condition and CAPs engaged during the cognitive control task ("TASK" condition) directly following emotion induction. This second analysis allowed testing whether emotional effects on cognitive control networks could be predicted by dFC changes evoked during emotional movies. For these analyses, the number of occurrences of each CAP resulting from the brain activation pattern analysis in stage 1 was entered in our model as directly observed variables.

Additionally, we assessed the relationship of brain changes with behavioral performance in the cognitive task by testing for any mediation effect of behavioral variables (using RTs from each trial type: cC, iC, cl, il) on the expression of different brain activation patterns, in both the negative and neutral emotional contexts. The multivariate mediation analyses modeled the brain CAPs occurrences in "REST1" (**Figure S4A**) and "MOVIE2" (**Figure S4B**) conditions as predictors. CAPs occurrences of the "TASK" condition were considered as outcomes variables. Additionally, the behavioral indices from the cognitive control task were modeled as indicators of a single latent variable (RT TASK), representing the underlying task performance measured by these indices. RT TASK was then tested as a mediator of the observed relationship between the predictors and outcomes. Please note, we used RT as a mediator here rather than a target measure because our main goal was to identify associations between brain networks over time based on emotional context. We aimed to relate changes in their dynamics across different stages during and after emotional events. Therefore, our BSEM primarily focused on brain-to-brain effects rather than brain-to-behavior and tested how emotion-dependent associations between networks were reflected by corresponding differences in behavior, using RT as a combined marker of the emotional impact on cognitive control.

A graphical illustration of these two analyses is shown in **Figure S4**. All the BSEMs were conducted in R⁸⁵ with the Blavaan⁷⁷ and Bayestest⁷⁸ packages. Further information of the BSEM methodology is reported in SI, “Supplementary Methods” section.

Results

Emotion induction

We found significant differences between subjective affective ratings before and after the negative emotional context evoked by movies [negative scores, NA: $t(23) = 6.97$; $p < .01$; positive scores, PA: $t(23) = 4.82$; $p < .05$; **Figure 1D**]. In contrast, the “pre” and “post” affective scores did not differ in the neutral context [NA: $t(23) = 1.7$; $p = \text{n.s.}$; PA: $t(23) = 2.10$; $p = \text{n.s.}$; **Figure 1D**]. These data confirm a shift in the affective state of participants during the negative context condition, subsequent to the experimentally induced emotions with audiovisual clips.

Moreover, as expected, the affective valence (Likert scale from 1 to 9) of movie contents was rated much lower for “negative” than “neutral” clips [$F(1, 72) = 317$; $p_{FDR} < .05$; **Figure 1B**], while there was no difference between the two movies from the same emotional context (neutral movies 1 vs 2, $F(1, 72) = 0.22$; $p_{FDR} = \text{n.s.}$; negative movies 1 vs 2, $F(1, 72) = 0.22$; $p_{FDR} = \text{n.s.}$). Arousal was also rated higher for negative compared to the neutral movies [$F(1, 72) = 288.21$; $p_{FDR} < .05$; **Figure 1B**], with no differences between movies from the same emotional condition [neutral, $F(1, 72) = 0.41$; $p_{FDR} = \text{n.s.}$; negative, $F(1, 72) = 0.16$; $p_{FDR} = \text{n.s.}$]. Likewise, subjective emotional experience during movies showed more intense and more negative ratings in the negative than neutral condition [$F(1, 72) = 384.44$; $p_{FDR} < .05$; **Figure 1B**].

Behavioral performance and affective modulation of cognitive control tasks.

In keeping with preliminary pilot testing, behavioral performance during fMRI confirmed an effective standardization of our two attentional tasks (Flanker and Stroop). Accuracy and response times (RTs) showed no significant difference between tasks in any trial type (no main effect of task, nor interaction of task x trial type in detailed analyses; see full results in the SI “Supplementary Results” section). Moreover, this RT pattern across trial types was similar regardless of the emotional induction (see SI “Supplementary Results” section, **Table S1**, and **Figure S1C**). These results ensure a reliable comparison of cognitive control performance across conditions with no task confound, and no habituation or learning effects. All behavioral and imaging data were therefore pooled across the two tasks.

Our main analysis of behavioral performance focused on RTs, as they provide the most sensitive and commonly used measure in these tasks⁷⁹. However, qualitatively similar results were observed for accuracy (see detailed description in the SI “Supplementary Results” section). In accordance with our pilot validation, a linear mixed model (LMM) ANOVA analysis with the factors “task”, “trial congruency”, “trial sequence”, and “emotion context” confirmed an absence of differences between Stroop and Flanker tasks [main effect of task: $F(1, 154) = 1.52$; $p = \text{n.s.}$]. Importantly, there was a robust congruency effect (CE) in both tasks [slower RT with incongruent “I” vs. congruent “C” distractors on current trial: $F(1, 154) = 40.11$; $p < .0001$], regardless of the affective induction (no interaction, $F(1, 154) = 1.79$; $p < 0.5$). CE effects did not differ when both tasks were directly compared [“I > C FLANKER” vs. “I > C STROOP”: $t(23) = 1.73$; $p = \text{n.s.}$; **Figure S1C**]. Additionally, the congruence sequence (CSE) effect was also significant [$F(1, 161) = 43.96$; $p < .0001$. **Table S1**], reflecting an influence of the preceding trial on the current one (“cC”, “iC”, “il”, and “cl”) as classically reported on such tasks⁸⁰. Again, there was no difference in CSE when contrasting trials from both tasks [“cC” trials: $\beta = 12.68$; $t(1.6)$; $p_{\text{FDR}} = \text{n.s.}$; “iC” trials: $\beta = -14.96$, $t(-1.95)$; $p_{\text{FDR}} = \text{n.s.}$; “il” trials: $\beta = 14.17$; $t(1.8)$; $p_{\text{FDR}} = \text{n.s.}$; “cl” trials: $\beta = 7.1$; $t(0.9)$; $p_{\text{FDR}} = \text{n.s.}$; **Figure S1C**], and there was no task by congruence

sequence interaction ($F(1,154) = 1.79$; $p = \text{n.s.}$). These results fully accord with previous behavioral work on attentional conflict and sequence effects associated with strategic attentional control, and confirm that these are generalizable across experimental tasks^{61,80,81}.

More critically for the objectives of our study, we then directly compared performance in the two different affective contexts. Results showed a significant main effect of emotion manipulation, with generally longer RTs when the task was performed following the negative compared to the neutral movie [$F(1,161) = 7.75$; $p < .01$, **Figure 1C**]. Further, although a strong congruency (CE) effect was seen in both emotional manipulations condition [$I > C_{\text{NEUTRAL}}$: $\beta = -48.9$; $t = -8.22$; $p_{\text{FDR}} < .0001$; $CI(-2.13_{\text{lower}}, -1.22_{\text{upper}})$; $d = -1.67$; $I > C_{\text{NEGATIVE}}$: $\beta = -58.5$; $t = -9.84$; $p_{\text{FDR}} < .0001$; $CI(-2.48_{\text{lower}}, -1.54_{\text{upper}})$; $d = -2.01$], it was significantly greater when the task was performed in the negative compared to the neutral context [Main effect of emotion on CE: $F(1,161) = 7.77$; $p < .01$; **Figure 1C**]. Likewise, we found a significant congruence sequence effect (CSE) in both emotional contexts [$F(1,161) = 46.15$; $p < .0001$, **Table S1**], which was amplified in the negative context for the two most difficult trials: namely, “iC” trials [$iC_{\text{neutral task}}$ vs. $iC_{\text{negative task}}$: $\beta = -14.9$; $t = -2.01$; $p_{\text{FDR}} = .07$; $CI(-1.18_{\text{lower}}, -0.01_{\text{upper}})$; $d = -0.58$; **Figure 1C**], and “cl” trials [$cl_{\text{neutral task}}$ vs. $cl_{\text{negative task}}$: $\beta = -24.88$; $t = -3.38$; $p_{\text{FDR}} < .01$; $CI(-1.57_{\text{lower}}, -0.37_{\text{upper}})$; $d = -0.97$. (**Figure 1C**)]. No significant effect of emotional manipulation was observed on trials with low conflict adaptation (i.e., “cC” and “il”. **Figure 1C**). Accordingly, there was a reliable interaction between the factors of emotional context and trial sequence [Main effect of emotion on CSE: $F(1,161) = 6.82$; $p < .01$], indicating an amplification of cognitive conflict adaptation following exposure to negative emotions.

Taken together, our behavioral data demonstrate a consistent modulation of cognitive control performance in response to our negative emotional induction, as evidenced by 1) a general slowing of behavioral performance (i.e., main effect of emotion. **Figure 1C, left**); 2) an amplification of

attentional conflict (larger interference effect [$I > C$], **Figure 1C, right**); and 3) a higher cost of strategic adaptation with greater influence of the preceding trial on the current one (**Figure 1C, middle**) - in the negative compared to the neutral task condition. These data not only show that selective attention control was successfully manipulated during our cognitive tasks, but also confirm a robust negative affect-related impact on executive function and conflict adaptation across different paradigms^{17,18}.

Standard GLM analysis of fMRI during cognitive control tasks.

We verified that cognitive control demands in the task blocks activated brain networks reported in previous studies using the same tasks^{26,64,80,82-84}. To do so, we compared fMRI activity during the main attentional conditions of both tasks (Stroop and Flanker tasks combined) using a standard event-related GLM implemented with SPM12⁸⁵. As expected, we found robust increases due to attentional interference ($I > C$ trials) in several brain areas involved in conflict monitoring and selective attention. These activations encompassed posterior parietal areas, the dorsolateral prefrontal cortex, and extrastriate visual areas bilaterally ($p_{FWE} < .05$; corrected at cluster level), overlapping with the dorsal frontoparietal network (dFPN)⁸⁶. Detailed results are shown in **Table S2** and **Figure S2**. These neural data converge with our behavioral results to confirm a reliable engagement of attentional control systems during the cognitive task blocks.

Dynamic functional connectivity (dFC).

The first step of our dFC analysis was to compute co-activation patterns (CAPs) consistently expressed during the entire scanning period. Unlike standard methods for estimating brain networks as "static" systems with averaged connectivity estimates, the CAPs capture dynamic patterns of functional coordination that fluctuate over time. Moreover, this data-driven approach enabled us to define the configurations of time-varying connectivity between regions (i.e., CAPs) which are not

Commented [pv4]: Can we also say that they confirm that both tasks engaged similar / same network?

Commented [JG5R4]: I think it's better as it is. Cf., response to question 16.

captured by traditional methods based on aprioristic compartmentalization of brain areas (i.e., functional atlas). We leverage the advantages of this approach to identify emotion-responsive brain networks that dynamically fluctuated across conditions and then examine their reciprocal relationships as well as their link to behavioral performance.

The “consensus” algorithm (see pipeline in **Figure 2**) indicated K=5 as the optimal number of CAP solutions, including four overlapping with functionally distinct networks and one more diffuse, presumably corresponding to noise artifacts. Subsequently, the consistency and reliability of these networks (i.e., CAPs) were validated with further k-means solutions (k=3, k=4, k=5, k=6, k=8, k=12). This intercluster similarity analysis (measured by voxel-wise Pearson distance between maps) revealed that the first four CAPs had the greatest spatial consistency across different K-means solutions. Namely, the four CAPs from the reference clustering (k=5) showed very high overlap (mean=90%) with similar CAPs resulting from different k-means solutions (**Figure S3**). Notably, the anatomical spatial configuration of these four CAPs resembled several well-known intrinsic functional networks (**Figure 3A**) as described in previous studies⁹³. Each of them also showed different rates of occurrences in the different experimental conditions (**Figure 3B**. See statistics in **Tables S3, S4**). We therefore selected the four most consistent maps as networks of interest for subsequent analysis, and disregarded the fifth noise-related map.

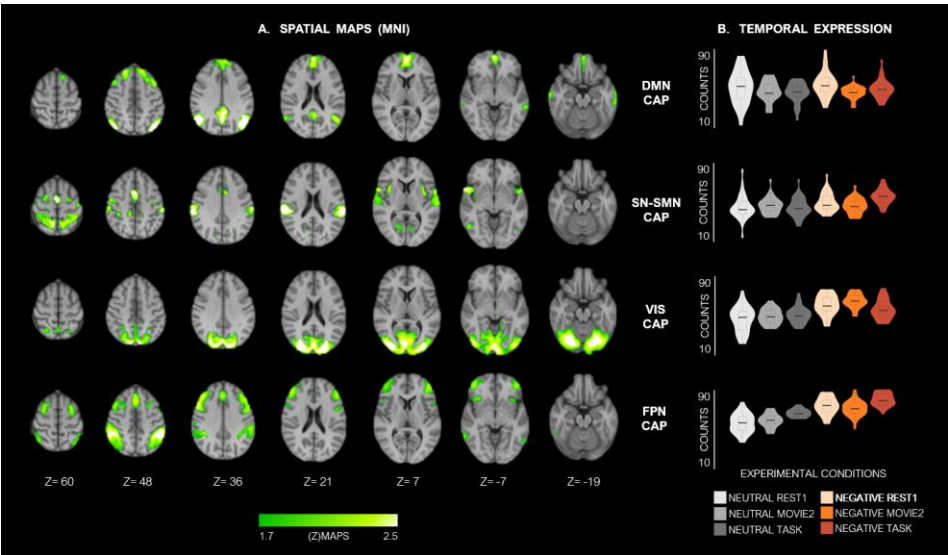


Figure 3. Brain maps depict the four co-activation patterns identified by our data-driven analysis of dFC. **A.** The spatial maps of each CAP are illustrated with a threshold of $[z > 1.7]$ ($p < .05$); green color code]. Further anatomical information on peak regions is provided in **Tables 1, 2, 3, and 4** respectively. **B.** Violin plots depicting the median (horizontal black lines), the interquartile range (box markers); and the probability density (vertical histograms) of each CAP expression (occurrences) across all experimental conditions (color coded). Descriptive statistics of the temporal occurrences are found in **Table S3**.

The first CAP overlapped with default mode-related areas [(DMN)⁸⁷. See peak coordinates in **Table 1**], including the medial prefrontal cortex (MPFC), posterior cingulate cortex (PCC), and inferior parietal lobe (IPL). This DMN_{CAP} was mainly expressed in the “REST1” condition, which accounted for a significant variance across experimental conditions. However, the occurrence rates of DMN_{CAP} did not differ significantly between the two affective contexts (**Figure 3B**, full statistics in **Tables S3 and S4**).

| DMN _{CAP} | | | | |
|--------------------|----|----|----|-----|
| REGION (MNI) | X | Y | Z | K> |
| Frontal_Mid_Orb_L | 41 | 53 | -8 | 360 |

| | | | | |
|----------------------|-----|-----|-----|-------|
| Frontal_Inf_Orb_R | 51 | 39 | -14 | 144 |
| Frontal_Inf_Orb_L | 49 | 33 | -12 | 488 |
| Frontal_Inf_Tri_L | -53 | 23 | 6 | 96 |
| Frontal_Sup_Medial_R | 1 | 59 | 14 | 54432 |
| Temporal_Mid_R | 67 | -15 | -18 | 3904 |
| Temporal_Mid_L | -65 | -23 | -14 | 6696 |
| Precuneus_L | 1 | -61 | 32 | 11952 |
| Angular_R | 53 | -63 | 34 | 13576 |
| Angular_L | -43 | -71 | 34 | 18048 |
| Cerebellum_Crus2_R | 39 | 77 | -40 | 4944 |
| Cerebellum_Crus2_L | -39 | -73 | -42 | 3296 |

Table 1. Brain regions contributing to DMN_{CAP}. Peak MNI coordinates at $z > 1.7$ ($p < .05$) across all participants are reported for all regions.

A second CAP encompassed regions from both the sensorimotor (SMN)⁸⁸ and salience networks (SN)⁸⁹, including the insula, cingulate cortex, SMA, and pre- and post-central cortices (**Figure 3A**; see peak coordinates in **Table 2**). Occurrences of this SMN-SN_{CAP} were more frequent during the “TASK” than during “REST1” or “MOVIE2” conditions. This difference was significant only in the negative context. (**Figure 3B**, **Tables S3**, **S4**).

| REGION (MNI) | SN-SMN _{CAP} | | | |
|-----------------|-----------------------|-----|----|-------|
| | X | Y | Z | K> |
| Cuneus_L | -9 | -85 | 32 | 168 |
| Cuneus_R | 17 | -75 | 26 | 680 |
| Occipital_Mid_R | 43 | -77 | 16 | 408 |
| Calcarine_R | 21 | -63 | 6 | 2768 |
| Temporal_Mid_L | -49 | -67 | 2 | 1344 |
| Temporal_Sup_R | 63 | -27 | 20 | 35576 |
| Temporal_Mid_R | 53 | -63 | -2 | 2048 |
| Temporal_Sup_L | -61 | -31 | 18 | 31624 |

Commented [pv6]: Should probably go in the suppl mat!?

Commented [JG7R6]: Moving these tables to the main manuscript was suggested by reviewer 3. Cf. Question 53.

| | | | | |
|-------------------|-----|-----|----|-------|
| Postcentral_L | -45 | -15 | 56 | 2048 |
| Precentral_R | 49 | -9 | 52 | 2064 |
| Insula_R | 39 | -17 | -4 | 80 |
| Supp_Motor_Area_R | 1 | -3 | 54 | 16224 |
| Insula_R | 39 | -1 | -6 | 80 |
| Frontal_Mid_L | -35 | 43 | 28 | 240 |

Table 2. Brain regions contributing to SN-SMN_{CAP}. Peaks MNI coordinates reported at $z > 1.7$ ($p < .05$) across all participants are shown for all regions.

The third CAP overlapped with the visual system (VIS)⁹⁰, comprising occipital and posterior parietal areas (**Figure 3A**; see peak coordinates in **Table 3**), and its occurrences were predominantly driven by the “negative MOVIE2” condition. However, VIS_{CAP} was also generally more present in the negative context (**Figure 3B**, **Tables S3** and **S4**).

| REGION (MNI) | VIS _{CAP} | | | K> |
|--------------------------|--------------------|-----|-----|-------|
| | X | Y | Z | |
| Precuneus_L | -2 | -54 | 54 | 4890 |
| Cuneus_R | 4 | -84 | 24 | 8860 |
| Middle occipital gyrus_L | -22 | -88 | 24 | 16322 |
| Middle occipital gyrus_R | 24 | -88 | 26 | 12059 |
| Lingual gyrus_R | 20 | -68 | -4 | 11148 |
| Lingual gyrus_L | -18 | -70 | -4 | 10556 |
| Parietal_Sup_L | -6 | -64 | 2 | 4576 |
| Parietal_Sup_R | -16 | -76 | 52 | 4410 |
| Temporal_Inf_R | 51 | -56 | -20 | 2167 |
| Temporal_Mid_R | 50 | -72 | 8 | 2060 |
| Parietal_Inf_L | -32 | -59 | 50 | 784 |
| Fusiform_R | 27 | -67 | -16 | 5296 |

Table 3. Brain regions contributing to VIS_{CAP}. Peaks MNI coordinates reported at $z > 1.7$ ($p < .05$) across all participants are shown for all regions.

Finally, a fourth CAP implicated fronto-parietal brain regions typically engaged during goal-directed cognition and executive functions [(FPN)⁹¹ **Figure 3A, Table 4**]. Occurrences of this FPN_{CAP} fluctuated between both affective contexts, with more presence in the “negative TASK” condition (**Figure 3B, Tables S3 and S4**).

These four CAPs were then used to address our two main questions concerning the emotional modulation of cognitive control, namely, whether changes in the functional interplay of brain networks at rest (hypothesis 1) or during movie watching (hypothesis 2) may predict a differential recruitment of cognitive control systems in the affective (vs neutral) contexts.

| REGION (MNI) | FPN _{CAP} | | | |
|----------------------|--------------------|-----|-----|--------|
| | X | Y | Z | K> |
| Parietal_Inf_R | 47 | -43 | 50 | 24896 |
| Parietal_Inf_L | -45 | -45 | 50 | 23608 |
| Precuneus_L | -9 | -69 | 52 | 56 |
| Temporal_Inf_L | -57 | -61 | -10 | 1104 |
| Temporal_Inf_R | 63 | -49 | -10 | 2576 |
| Frontal_Mid_L | -45 | 31 | 28 | 29424 |
| Frontal_Mid_R | 47 | 33 | 30 | 435264 |
| Frontal_Sup_Medial_R | 3 | 27 | 42 | 8368 |
| Frontal_Inf_Oper_R | 59 | 15 | 10 | 56 |
| Frontal_Inf_Oper_R | 51 | 15 | 6 | 192 |
| Insula_L | -33 | 19 | -4 | 776 |
| Insula_R | 35 | 23 | -6 | 1576 |

Table 4. Brain regions contributing to FPN_{CAP4}. Co-active peaks (MNI coordinates) reported at $z > 1.7$ ($p < .05$) across all participants are shown for all conditions.

Association of affective scores with CAPs

We also tested whether occurrences of these relevant CAPs were associated with behavioral measures of subjective emotional state (PANAS). We found that higher occurrences of DMN_{CAP} observed in the “REST1 ” condition were associated to more negative affect scores following emotional induction in the negative context [Rho= 0.52; 95% CI(0.25lower, 0.79upper); ROPE: 0.0%; Figure 4A]. This correlation was not significant in the neutral context [Rho= 0.12; 95% CI(-0.32lower, 0.40upper); ROPE: 59%; Figure 4A]. Occurrences of $SN-SMN_{CAP}$ observed in the “REST1 ” condition were only marginally anticorrelated with the negative affect scores following the emotional induction of in the negative context [Rho= -0.40; 95% CI(-0.68lower, -0.06upper); ROPE: 5%; Figure 4A], and not related to affect. The same pattern was observed in the neutral context with much less magnitude [Rho= -0.11; 95% CI(-0.45lower, 0.25upper); ROPE: 35%; Figure 4A]. No significant associations were observed between VIS_{CAP} , FPN_{CAP} and subjective affective scores respectively (**Figure 4A**).

Hypothesis 1. Task-related activity is determined by the impact of emotion on intrinsic brain network dynamics at rest.

Based on previous work indicating that intrinsic functional activity within resting-state brain networks may shape their engagement during cognitive tasks⁹², we performed two consecutive analyses. A first BSEM assessed whether the occurrences of a given CAP during “REST1” contribute to the modulation of CAPs expressed during the subsequent cognitive control “TASK” condition. Context-dependent variations (i.e., neutral and negative) of these relationships were also considered. The validity of this model was verified by inspecting the measurement invariance across conditions⁹³ for the latent factor RT TASK. Results (**Table S5**) showed no evidence of violations of weak invariance in our sample. Model integrity was also confirmed by trace plots and densities of individual parameters, which showed rapid mixing of the Markov chains, with satisfactory model convergence to our target distribution. This model convergence was validated by the Gelman and Rubin

diagnostics and effective effect size assessments, (see results in **Tables S6 and S7** respectively). Additionally, results from different prior distributions (i.e., sensitivity analyses) yielded low impact of the priors on the results (**Table S8**). Likewise, the serial autocorrelation assessment of each model parameters yielded low dependency between estimates. These data suggest a satisfactory performance of our estimation settings. The full results on model convergence assessments are illustrated in **Figures S5 and S6**.

Next, we determined whether parameter estimates in the model were meaningfully different from a defined null-result value, using the Bayesian concept of a region of practical equivalence (ROPE)⁹⁴. The latter postulates a decision criterion to define whether a parameter estimate density is indeed different from the null value. We chose a threshold of two-sided 5% or less density in the ROPE to consider a parameter meaningfully different from 0. This threshold goes in parallel with the standard two-sided 5% alpha in the frequentist framework, and follows the recommendations from simulation studies evaluating different Bayesian indices of existence and significance⁹⁵.

Results from this first BSEM analysis revealed several associations between CAPs expressed in the “negative REST1” and CAPs expressed in the subsequent “negative TASK”, which were not present between the same conditions of the neutral context [i.e., “neutral REST1” and “neutral TASK” (**Figure 4B**)]. More specifically, the occurrences of DMN_{CAP} during the “negative REST” were linked to occurrences of both DMN_{CAP} [*Median*= 0.52; 95% *CI*(0.16_{lower}, 0.88_{upper}); ROPE: 0.0%; **Figure 4B**] and FPN_{CAP} during the “negative TASK” condition [*Median*= 0.45; 95% *CI*(0.15_{lower}, 0.74_{upper}); ROPE: 0.0%; **Figure 4B**]. Only the relationship between DMN_{CAP} (REST) and FPN_{CAP} (TASK) was observed in the neutral context [*Median*= 0.89; 95% *CI* (0.40_{lower}, 1.43_{upper}); ROPE: 0.0%; **Figure 4B**]. Additionally, a bidirectional relationship between $SMN-SN_{CAP}$ and FPN_{CAP} was found exclusively in the negative

context: SMN-SN_{CAP} during “negative REST1” predicted the occurrences of FPN_{CAP} during the “negative TASK” condition [*Median*= -0.39; 95% *CI*(-0.67_{lower}, -0.08_{upper}); ROPE:1.0%; **Figure 4B**]; whereas conversely, occurrences of FPN_{CAP} during “negative REST1” predicted SMN-SN_{CAP} occurrences during the “negative TASK” condition [*Median*= -0.75; 95% *CI*(-1.03_{lower}, -0.21_{upper}); ROPE: 0.0%]. Finally, occurrences of VIS_{CAP} in “negative REST1” predicted the same CAP during the “negative TASK” condition [*Median*= 0.43; 95% *CI*(0.10_{lower}, 0.76_{upper}); ROPE: 0.0%; **Figure 4B**]. Again, the latter relationships were not seen in the neutral context. These data suggest that emotional context markedly modified the functional relationship between intrinsic brain networks that govern the recruitment of cognitive control processes (particularly FPN).

In the second step of this analysis, we probed for any mediation effects of behavioral performance [indexed by reaction time (RT TASK)] on the relationship of functional networks described above. In this analysis, the BSEM regressions revealed a partial mediation by the “RT TASK” index affecting the relationship between DMN_{CAP} “REST1” and FPN_{CAP} “TASK” in the negative context [*Median*=0.32; 95% *CI*(0.10_{lower}, 0.61_{upper}); ROPE: 2.0%; **Figure 5A**]. A further partial mediation by RT TASK was found in the negative context for the relationship between SMN-SN_{CAP} in “REST1” and FPN_{CAP} in “TASK” condition [*Median*=-0.33; 95% *CI*(-0.61_{lower}, -0.09_{upper}); ROPE: 1.0%; **Figure 5A**]. In this negative context, RT TASK was positively linked to the occurrences of DMN_{CAP} at “REST1” and negatively related to the occurrences of SMN-SN_{CAP} at the same “REST1” condition. No mediation of RT TASK was found for associations between networks in the neutral context. Consequently, the model decision rule on these effects yielded important differences between the neutral and negative emotional context. See **Figure 5C**. Full details on statistical results (i.e., direct, indirect and total effects) for all CAPs are reported in **Tables S9, S10, S11, S12**

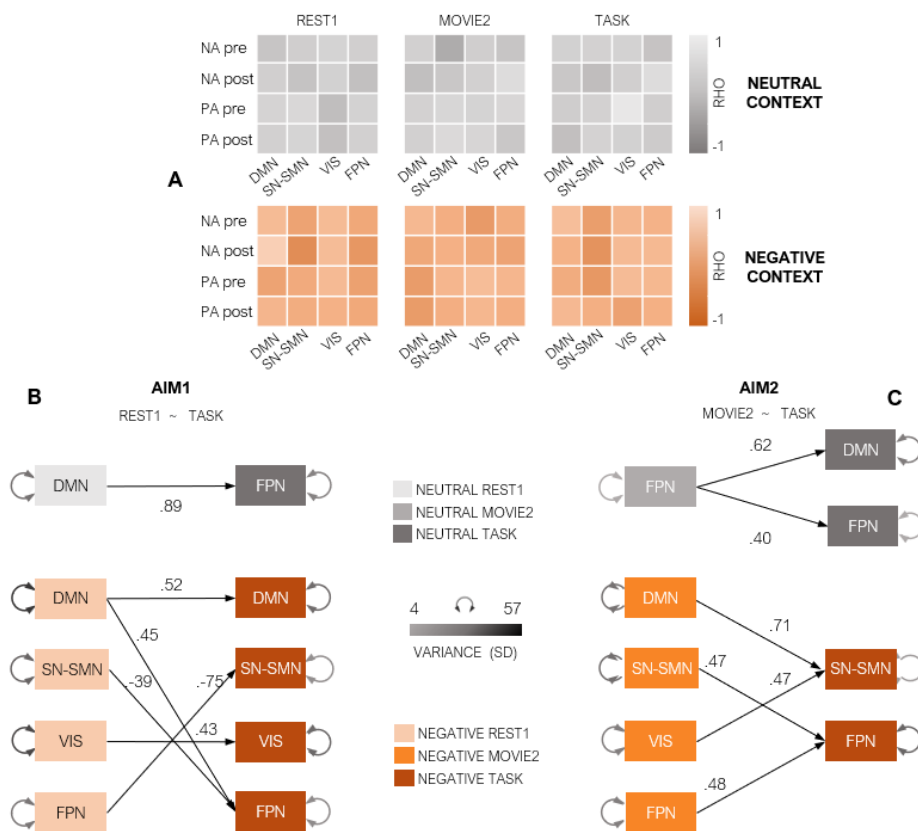


Figure 4. **A.** Bayesian correlation matrices of the brain CAPs (y-axis) and positive (PA) and negative (NA) subjective scores (x-axis) assessed before (pre) and following (post) the affective elicitation in the negative and neutral context respectively. **B** Associations between functional brain CAPs across the experimental conditions (color code) as estimated by BSEM. Significant relationships (represented by arrows) between CAPs during “REST1” and “TASK” conditions were greater in the negative context (bottom), compared to the neutral one (up). **C.** Likewise, the CAPs captured during “MOVIE2” condition reported more significant associations to those expressed at “TASK” condition of the negative context. Double-headed loop arrows depict the variance [standard deviation (SD)] of the CAPs residuals. Statistical significance of the posterior distribution values was based on the HDI+ROPE decision rule, where parameters with <2,5% in ROPE are considered meaningfully different from 0.

Hypothesis 2. Task-related activity is determined by the impact of emotion on brain networks engaged during movie watching.

Our second main question was whether brain activity patterns elicited during negative emotional events themselves could predict changes in brain networks recruited by the subsequent cognitive control task. As above, two successive multivariate BSEM regression analyses were conducted to test whether differences in temporal occurrences of CAPs observed during the just preceding emotional induction ("MOVIE2" condition) contribute to the modulation of CAPs expressed during cognitive control ("TASK" condition). When considering only direct regressions (i.e., preliminary BSEM without mediators), we found significant relationships in the negative context, specifically between occurrences of DMN_{CAP} and VIS_{CAP} during the "MOVIE2" condition and subsequent expression of $SN-SMN_{CAP}$ in the "TASK" [DMN_{CAP} : *Median*= 0.71; 95% *CI*(0.4_{lower}, 1.0_{upper}); ROPE: 0.0%. VIS_{CAP} : *Median*= 0.47; 95% *CI*(0.1_{lower}, 0.82_{upper}); ROPE: 0.0%; **Figure 4C**], as well as between occurrences of $SN-SMN_{CAP}$ and FPN_{CAP} during the "MOVIE2" condition and subsequent presence of FPN_{CAP} in the "TASK" [$SN-SMN_{CAP}$: *Median*= 0.47; 95% *CI*(0.14_{lower}, 0.89_{upper}); ROPE: 0.0%. FPN_{CAP} : *Median*= 0.48; 95% *CI*(0.25_{lower}, 0.69_{upper}); ROPE: 0.0%. **Figure 4C**]. In the neutral context, we found only significant effects of the FPN_{CAP} during "MOVIE2" on subsequent DMN_{CAP} and FPN_{CAP} occurrences in the "TASK" [DMN_{CAP} : *Median*=0.62; 95% *CI*(0.03_{lower}, 1.20_{upper}); ROPE: 1.0% . FPN_{CAP} : *Median*=0.40; 95% *CI*(0.20_{lower}, 0.62_{upper}); ROPE: 0.0% ; **Figure 4C**].

The subsequent BSEM, including RT TASK as a mediator (**Figure S4B**), showed partial mediation of these behavioral metrics, specifically related to the association between $SMN-SN_{CAP}$ in "MOVIE2" and FPN_{CAP} in "TASK" in the negative context [Indirect effect: *Median*= 0.32, 95% *CI*(0.10 lower, 0.64 upper); ROPE: 2.0%. **Figure 5B**]. RT TASK was positively linked to those brain CAPs. No such mediation was observed in the neutral context. Results from this model also revealed significant

differences in the direct and total effects between the two affective contexts (**Figure 5C**). See full details in **Tables S10, S11, S13, S14, S15, S16**

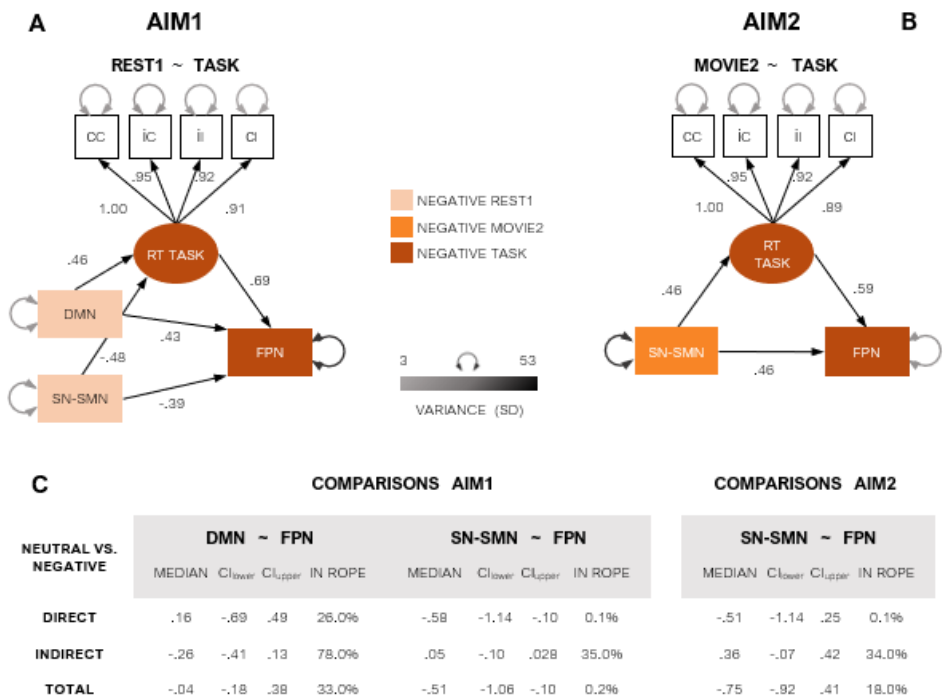


Figure 5. Functional associations between CAP occurrences during the cognitive task and the preceding rest (A) or movie (B) periods and their modulation by concomitant changes in reaction time indices during the cognitive control task. **A.** The relationship between DMN_{CAP} and SN-SMN_{CAP} expressions in "negative REST1" with subsequent FPN_{CAP} expression in the "negative TASK" condition was partially mediated by cognitive control performance (mean reaction times for "iC", "iI", "cI" trials). **B.** Likewise, the relationship between SN-SMN_{CAP} expression during the negative MOVIE2 and FPN_{CAP} expression during the cognitive control task was partially mediated by the same behavioral measures ("iC", "iI", "cI" trials) again specifically in the negative affect context. No such relationship was found for any of the CAPs in neutral context. See full results of indirect (i.e., mediation) effects in **Tables S9** and **S12**. Double-headed loop arrows depict the variance [standard deviation (SD)] of the CAPs residuals. **C.** Practical equivalence assessment of the difference in terms of magnitude effects between neutral and negative affective contexts. The statistical significance of the posterior distribution values was based on the HDI+ROPE decision rule (<2,5% in ROPE).

Discussion

We conducted a data-driven analysis of dynamic functional connectivity (dFC) and BSEM to illustrate how negative emotions triggered by naturalistic stimuli evoked transient hyperconnectivity among multiple brain networks over time. These spatiotemporal features of brain activity led us to provide a dFC account of how cognitive control processes are influenced by prior emotional events and their lingering effects on brain networks.

Importantly, our behavioral measures of cognitive control, subjective affective ratings, and standard GLM analysis of task-related brain activation all converged to show strong effects of our experimental manipulations, confirming both the effectiveness and reliability of our paradigm.

We found that four distinct dFC patterns were differentially engaged across our experimental conditions (i.e., resting state, movie watching, and cognitive attentional task in either neutral or negative affective contexts). Some of these CAPs partly overlapped with classic IFNs reported in previous studies (e.g., DMN_{CAP1} and VIS_{CAP3})⁸⁶, while others shared spatial configurations with multiple “canonical” networks. Our CAP2 for instance, overlapped with both sensorimotor (SMN)⁹⁶ as well as salience-related (SN) areas⁸⁹. CAP4, on the other hand, overlapped with frontoparietal nodes associated with central executive control (CEN)⁹⁷, dorsal attention (DAN)^{53,98}, and more general task-positive networks⁹⁹. Furthermore, our CAP analysis quantitatively described how the temporal dynamics of these networks (i.e., their occurrence rates) fluctuated over time as function of current demands, such as resting state, movie watching, or cognitive control challenges. Although, as expected, these three condition were associated with a relative predominance of CAPs overlapping with the DMN, VIS, and CEN/SN fronto-parietal network (FPN), respectively, we could quantify the occurrences of each CAP in each condition and each affective context.

Commented [pv8]: Maybe a few places where we could shorten if we have to add our responses to the reviewers...

Secondly, and most crucially, our BSEM analysis demonstrated how the expression of distinct dFC patterns either during movie watching or during post-movie rest influenced the subsequent recruitment of brain systems mediating cognitive control, and how the latter were impacted by negative emotions. Critically, the dynamic relationships between brain networks involved in affective events and those responsible for cognitive control differed notably in negative affective conditions compared to neutral ones. In a complementary mediation analysis, these relationships were found to be significantly influenced by concurrent changes in behavioral measures of cognitive control, such as reaction time, supporting a direct connection between brain network associations and the behavioral effects of emotional experiences.

The impact of negative emotions on cognitive control is linked to their aftermath on intrinsic brain network dynamics.

Our first aim was to determine the relationships between the neural carry-over effects of negative emotion, emerging after eliciting events (i.e., CAPs in “REST1” condition)^{41,44}, and the activation pattern of brain networks recruited by cognitive control demands (i.e., CAPs in “TASK” condition)^{19,20,22}. Our results revealed **anticorrelated** associations between occurrences of DMN_{CAP} and $SN-SMN_{CAP}$ during the post-emotion resting state, and occurrences of FPN_{CAP} observed later during the TASK condition. In other words, the affective “aftermath” (or emotional “inertia”) underpinned by those spontaneous brain dFC following the first negative emotion induction^{41,100} predicted an increase in brain activation patterns expressed during a cognitive control task performed after another (similar) negative emotion induction. Accordingly, the number of occurrences of FPN during the “TASK” block significantly increased in the negative compared to neutral context (**Figure 3**). In addition, **higher** DMN_{CAP} **occurrences** at rest after emotion induction also predicted increases in its own occurrences during the task and correlated with more negative affect in subjective scores,

consistent with affective carry-over effects (or inertia) in this network⁴⁴. Most critically, these relationships among networks in the negative context were paralleled by concomitant effects on behavioral performance (as assessed by increased interference on RTs after negative emotions). The latter result supports the functional significance of neural changes observed in dFC and their behavioral significance for cognitive control.

Commented [JGL9]: (it can be supported by linking DMN and negative score)

Beyond their impact on subsequent activity of FPN during cognitive control, higher DMN occurrences at rest after negative events accords with a broader role of this network in affective states. DMN activity is generally related to self-reflective processing, most active during rest conditions¹⁰¹, but increasing evidence from recent fMRI studies points to an important role in affective experience and emotion regulation¹⁰². Previous work uncovered consistent carry-over effects in DMN at rest following exposure to various emotion conditions, including positive or negative movies^{41,47}, odors, or rewards^{45,59}, which may reflect adaptive processes restoring affective homeostasis after emotional challenges^{47,103}. Persistent DMN activation following emotion elicitation has also been related to the intensity of subjective affective experience¹⁰⁴. Our data therefore accord with the notion that more intense emotional events might require greater self-regulation, possibly through spontaneous homeostatic processes mediated by DMN that allow the brain to return to “normal” neutral states following transient situational challenges and acute stress responses.

Commented [JGL10]: supported by linking DMN and negative score)

Likewise, SN-SMN activity has also been linked to various aspects of affective processing. First, the SN subsystem is consistently implicated in the detection of salient events, with or without affective meaning^{105,106}. Second, different nodes within both SN and SMN (including insular and somatosensory cortices, respectively) play a key role in awareness of internal bodily states (i.e., interoception) and subjective emotional feelings^{107,108}. Based on the association between DMN, SN-SMN systems and their association with negative affect scores, We speculate that, in the post-

Commented [pv11]: Seems a bit redundant and could be cut

Commented [JG12R11]: It was edited

emotional resting state, increases in SN-SMNCAP might support the processing of interoceptive signals and bodily feelings¹¹⁴, possibly through a connection with self-referential representations elaborated in DMN areas that together contribute to subjective emotional states experience¹¹⁵. This interpretation accords with other effects of negative emotions and stress on selective attention and rumination, characterized by repetitive retrieval of aversive and threat-information about the self or past events, typically exacerbated by negative contextual cues¹⁰⁹ and associated with increased DMN activity⁴.

In sum, our results go beyond previous observations of SN-SMN-DMN responses to emotions.^{87,89,110}, and are the first to provide a direct quantitative characterization of long-lasting emotion-induced changes in functional brain dynamics and their impact on cognitive control networks and behavioral performance.

The impact of negative emotions on cognitive control also reflects the brain response to emotion-eliciting events

Our second aim was to determine the functional neural associations between brain responses to negative emotional events themselves (CAPs in “negative MOVIE2” condition) and changes in dFC patterns associated with cognitive control directly after such events (CAPs in “negative TASK” condition). A key finding was that higher occurrences of FPN_{CAP} during the TASK was significantly influenced by increases in the preceding occurrences of SN-SMN_{CAP} during the “negative MOVIE2” block. In terms of emotion elicitation, increased SN-SMN activity induced by negative emotion and its influence on FPN might provide a neural substrate for the psychological construct of arousal¹¹¹, which constitutes a crucial but poorly understood dimension of emotional experience¹¹² underlying the regulation of attentional resources by affective signals¹¹³. In addition, this SN-SMN_{CAP}~FPN_{CAP} relationship involved not only a direct effect but also an indirect link with behavioral indices of cognitive performance (**Figure 5B**). This was also specific to the negative affective context and absent in the

Commented [JG13]: I don't see how to add Lane et al., 2021 doi: [10.3390/jintelligence9030042](https://doi.org/10.3390/jintelligence9030042) Unless you refer to another paper. If so, please add the respective doi. It saves me precious time

neutral context (**Figure 5C**). Such influence of SN-SMN occurrences might be consistent with sustained effects of arousal mechanisms increasing reactivity to behaviorally salient events and action readiness subsequent to negative events¹¹⁴, which could in turn modulate the expression of cognitive control processes mediated by FPN during the task^{89,115,116}. Thus, the recruitment of SN-SMN during negative emotion elicitation could play a causal role in regulating the FPN system and its engagement in subsequent tasks, thereby increasing arousal and influencing executive control functions for a prolonged period of time after emotional events.

In parallel, FPN activity during the task was also predicted by occurrences of this network during the movies. However, this effect was unrelated to changes in behavioral performance (i.e., no significant mediation linked to RTs). This might suggest a more general effect on attentional resources (i.e., control network activity) allocated to both emotionally relevant content in the movie and behaviorally relevant stimuli in the task, without affecting the inhibition of distractor stimuli during task performance. Accordingly, areas overlapping with FPN are implicated in the active maintenance and manipulation of task-relevant information during demanding cognitive tasks¹¹⁷, including goal-directed processing and top-down biasing of attention and perception based on endogenous information^{118,119}. In contrast, an upregulation of SN-SMN_{CAP} in response to negative emotional information in movies might influence subsequent attentional state and trigger affective biases in reactivity towards the environment through the modulation of FPN connectivity. This could act to enhance vigilance and facilitate detection of and response to potentially threatening stimuli at the cost of pursuing long-term endogenous goals^{120,121}, which could promote more diffuse attention and increase interference by distractors in our cognitive tasks (as suggested by behavioral data)^{122,123}. These findings would also provide a plausible neural substrate to account for a general slowdown of RTs frequently observed in negative affective conditions (as also found in our study), an effect often attributed to a modulation or suspension of ongoing goal-oriented activity triggered by threat

Commented [JG14]: Patrik: Interpreting SN-SMN occurrences in MOVIE2 and affective arousal makes more sense here than SN-SMN occurrences in REST1 and affective arousal. See response to question 25

Commented [pv15R14]: ok

Commented [pv16]: which ? FPN or SN-SMN? Please rephrase to clarify (itself or SN-SMN)

Commented [JG17R16]: Cf. Response to question 22.

signals¹²⁴. While our results reveal predictive links between emotion-driven network states and subsequent cognitive-control performance, we emphasize that these associations were derived from a Bayesian structural-equation model that was rigorously checked for convergence and prior sensitivity, rather than from post-hoc reverse inference. We therefore frame our findings within a brain-as-predictor framework^{92,125–128} in which observed dFC patterns forecast measurable brain or behavioral outcomes without implying that the activations alone confirm specific cognitive or emotional processes.

Limitations

Our study included only biological female participants, but the presence of non-binary genders was not controlled. This sex selection was based on pilot results in which women reported higher emotional ratings, and previous literature showing higher reactivity to negative emotions. Accordingly, emotional induction with social material was reported to be more reliable in females and motivated similar selection procedure in other studies on emotion regulation¹²⁹. However, it is possible that, in our paradigm, women adopted a more empathetic perspective in response to sad movie clips or may have experienced more negative emotions than males in similar contexts. Our results therefore need to be replicated in male participants.

Another possible limitation lies in the implementation of two different interference paradigms, even though they were fully counterbalanced across participants in terms of stimulus presentation and emotional valence (negative vs. neutral), and carefully validated in pilot testing to ensure similar behavioral performance. This was chosen by design to minimize the influence of trial- to-trial priming effects and habituation when tasks were repeated in different emotion

conetxts. Due to their specific characteristics, these two tasks might evoke partly different neural responses. However, we carefully counterbalanced them across different affective context and session order, and our results did confirm reliable conflict adaptation effects that were wer highly similar in both tasks and fully similar to previous research suggesting shared attentional-control processes⁵¹.

Lastly, we were not able to obtain reliable physiological measures in many participants (such as skin conductance or pupillary diameters), precluding us to investigate the relation of both affective and cognitive effects with autonomic differences between conditions. Peripheral physiological data would be useful in future follow-up studies to refine our understanding of emotional carry-over effects and better characterize their impact on affective and cognitive states.

Conclusions

To the best of our knowledge, this is the first study reporting a functional relationship based on whole-brain network dynamics to explain the prolonged influence of (negative) affective stimulation on cognitive control performance. By assessing spatiotemporal aspects of IFNs across different experimental conditions with a data-driven dFC approach, we uncovered how brain activity patterns underlying cognitive control are shaped by transient network fluctuations elicited by negative emotion-laden information and their impact on spontaneous brain connectivity expressed at rest. Specifically, our findings suggest that FPN expression during selective attention control demands is distinctively affected by prior occurrences of SN-SNM CAPs during emotional episodes, together with shifts in both SMN-SN and DMN expressions

after emotional episodes (i.e., at rest, during the affective aftermath presumably associated with emotion regulation and homeostatic recovery⁴⁴). Further, both associations were linked to concomitant behavioral indices of cognitive control, suggesting a direct role of these functional relationships between networks in the deployment of attentional processes during the cognitive control task. While SN-FPN connectivity may reflect the modulation of vigilance or arousal triggered by salience detection mechanisms, connectivity of DMN with SN-SNM and FPN could manifest information processing biases toward self-reflective processes associated with emotion regulation and introspection, which both could affect reactivity and selectivity in responding to external stimuli in the aftermath of negative emotions, as commonly reported in negative and stressful contexts.

Altogether, our findings provide novel insights on brain mechanisms underlying the influence of emotions on cognition^{13,123}, and potentially related to resilience to stress¹³⁰, attention deficits in depression or anxiety¹³¹, and perseverative ruminative thinking in negative mood states^{41,132,133}. Further research is needed to investigate whether similar changes in brain dFC are also observed (and potentially exacerbated) in psychiatric diseases, populations with at-risk profiles, or healthy individuals with particular personality traits. In turn, it remains to be determined whether these neural measures may offer valuable biomarkers of (mal)adaptive emotion regulation abilities allowing better assessment and monitoring of psychopathology conditions.

Data availability

BIDS¹³⁴ of the raw data supporting the findings of this study will be available at <https://yareta.unige.ch/#/home> in full accordance to the FAIR principles for scientific data management¹³⁵.

Acknowledgments

This work was supported by the Sinergia Grant no. CRSII5_180319 from the Swiss National Science Foundation (SNF), the Swiss Center of Affective Sciences financed by UNIGE and SNF (Grant no. 51NF40_104897), and the Société Académique de Genève (SACAD). This study was conducted on the imaging platform at the Brain and Behavior Lab (BBL) and benefited from support of the BBL technical staff. Imaging analysis was carried out at University of Geneva on the high-performance computing (HPC) BAOBAB server.

References

1. van Steenbergen, H. Affective Modulation of Cognitive Control: A Biobehavioral Perspective. in *Handbook of Biobehavioral Approaches to Self-Regulation* (eds. Gendolla, G. H. E., Tops, M. & Koole, S. L.) 89–107 (Springer New York, New York, NY, 2015). doi:10.1007/978-1-4939-1236-0_7.
2. Tambini, A., Rimmele, U., Phelps, E. A. & Davachi, L. Emotional brain states carry over and enhance future memory formation. *Nat. Neurosci.* **20**, 271–278 (2017).
3. Leventhal, H. & Scherer, K. R. The Relationship of Emotion to Cognition: A Functional Approach to a Semantic Controversy. *Cogn. Emot.* **1**, 3–28 (1987).
4. Baez-Lugo, S. *et al.* Exposure to negative socio-emotional events induces sustained alteration of resting-state brain networks in older adults. *Nat. Aging* **3**, 105–120 (2023).
5. Zhou, H.-X. *et al.* Rumination and the default mode network: Meta-analysis of brain imaging studies and implications for depression. *NeuroImage* **206**, 116287 (2020).
6. Janiri, D. *et al.* Shared Neural Phenotypes for Mood and Anxiety Disorders: A Meta-analysis of 226 Task-Related Functional Imaging Studies. *JAMA Psychiatry* **77**, 172–179 (2020).
7. Perry, A., Roberts, G., Mitchell, P. B. & Breakspear, M. Connectomics of bipolar disorder: a critical review, and evidence for dynamic instabilities within interoceptive networks. *Mol. Psychiatry* **24**, 1296–1318 (2019).
8. Pessoa, L. On the relationship between emotion and cognition. *Nat. Rev. Neurosci.* **9**, 148–158 (2008).
9. Lewis, M. D. Bridging emotion theory and neurobiology through dynamic systems modeling. *Behav. Brain Sci.* **28**, 169–194 (2005).
10. Todd, R. M., Miskovic, V., Chikazoe, J. & Anderson, A. K. Emotional Objectivity: Neural Representations of Emotions and Their Interaction with Cognition. *Annu. Rev. Psychol.* **71**, (2020).
11. Sander, D. *et al.* Emotion and attention interactions in social cognition: Brain regions involved in processing anger prosody. *NeuroImage* **28**, 848–858 (2005).
12. Kragel, P. A. *et al.* Generalizable representations of pain, cognitive control, and negative emotion in medial frontal cortex. *Nat. Neurosci.* **21**, 283–289 (2018).

13. Okon-Singer, H., Hendler, T., Pessoa, L. & Shackman, A. J. The neurobiology of emotion cognition interactions: fundamental questions and strategies for future research. *Front. Hum. Neurosci.* **9**, 58 (2015).
14. Dolcos, F. *et al.* Neural correlates of emotion-attention interactions: From perception, learning, and memory to social cognition, individual differences, and training interventions. *Neurosci. Biobehav. Rev.* **108**, 559–601 (2020).
15. Pourtois, G., Schettino, A. & Vuilleumier, P. Brain mechanisms for emotional influences on perception and attention: What is magic and what is not. *Biol. Psychol.* **92**, 492–512 (2013).
16. Vuilleumier, P. Affective and motivational control of vision. *Curr. Opin. Neurol.* **28**, 29–35 (2015).
17. Schuch, S. & Koch, I. Mood states influence cognitive control: the case of conflict adaptation. *Psychol. Res.* **79**, 759–772 (2015).
18. van Steenbergen, H., Band, G. P. H. & Hommel, B. In the Mood for Adaptation: How Affect Regulates Conflict-Driven Control. *Psychol. Sci.* **21**, 1629–1634 (2010).
19. Kuhbandner, C. & Zehetleitner, M. Dissociable effects of valence and arousal in adaptive executive control. *PLoS ONE* **6**, (2011).
20. van Steenbergen, H., Band, G. P. H., Hommel, B., Rombouts, S. A. R. B. & Nieuwenhuis, S. Hedonic Hotspots Regulate Cingulate-driven Adaptation to Cognitive Demands. *Cereb. Cortex* **25**, 1746–1756 (2015).
21. Li, W. *et al.* Positive and negative emotions modulate attention allocation in color-flanker task processing: Evidence from event related potentials. *Motiv. Emot.* **38**, 451–461 (2014).
22. Melcher, T., Born, C. & Gruber, O. How negative affect influences neural control processes underlying the resolution of cognitive interference: An event-related fMRI study. *Neurosci. Res.* **70**, 415–427 (2011).
23. Hart, S. J., Green, S. R., Casp, M. & Belger, A. Emotional priming effects during Stroop task performance. *NeuroImage* (2010) doi:10.1016/j.neuroimage.2009.10.076.
24. Dennis, T. A., Chen, C.-C. & McCandliss, B. D. Threat-related attentional biases: an analysis of three attention systems. *Depress. Anxiety* **25**, E1–E10 (2008).

25. Dreisbach, G. & Fischer, R. Conflicts as aversive signals. *Brain Cogn.* **78**, 94–98 (2012).
26. Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S. & Cohen, J. D. Conflict Monitoring and Cognitive Control. *Psychol. Rev.* **108**, 624–652 (2001).
27. Gehring, W. J. & Willoughby, A. R. The medial frontal cortex and the rapid processing of monetary gains and losses. *Science* (2002) doi:10.1126/science.1066893.
28. Bush, G., Luu, P. & Posner, M. I. Cognitive and emotional influences in anterior cingulate cortex. *Trends Cogn. Sci.* **4**, 215–222 (2000).
29. Etkin, A., Egner, T. & Kalisch, R. Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends Cogn. Sci.* **15**, 85–93 (2011).
30. Cole, M. W. & Schneider, W. The cognitive control network: Integrated cortical regions with dissociable functions. *NeuroImage* **37**, 343–360 (2007).
31. Chen, T. *et al.* A domain-general brain network underlying emotional and cognitive interference processing: evidence from coordinate-based and functional connectivity meta-analyses. *Brain Struct. Funct.* **223**, 3813–3840 (2018).
32. Vuilleumier, P. The role of the human amygdala in perception and attention. in *The human amygdala*. 220–249 (The Guilford Press, New York, NY, US, 2009).
33. Padmala, S., Bauer, A. & Pessoa, L. Negative emotion impairs conflict-driven executive control. *Front. Psychol.* **2**, 1–5 (2011).
34. Kalanthroff, E., Cohen, N. & Henik, A. Stop feeling: inhibition of emotional interference following stop-signal trials. *Front. Hum. Neurosci.* **7**, 78 (2013).
35. Shackman, A. J. *et al.* The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nat. Rev. Neurosci.* **12**, 154–167 (2011).
36. Vuilleumier, P. Affective and motivational control of vision. *Curr. Opin. Neurol.* **28**, 29–35 (2015).
37. Meaux, E. & Vuilleumier, P. *Emotion Perception and Elicitation. Brain Mapping: An Encyclopedic Reference* vol. 3 (Elsevier Inc., 2015).

38. Pichon, S., Miendlarzewska, E. A., Eryilmaz, H. & Vuilleumier, P. Cumulative activation during positive and negative events and state anxiety predicts subsequent inertia of amygdala reactivity. *Soc. Cogn. Affect. Neurosci.* **10**, 180–190 (2015).
39. Provenzano, J., Verduyn, P., Daniels, N., Fossati, P. & Kuppens, P. Mood Congruency Effects are Mediated by Shifts in Salience and Central Executive Network Efficiency. *Soc. Cogn. Affect. Neurosci.* 987–995 (2019) doi:10.1093/scan/nsz065.
40. Qiao-Tasserit, E., Corradi-Dell'Acqua, C. & Vuilleumier, P. The good, the bad, and the suffering. Transient emotional episodes modulate the neural circuits of pain and empathy. *Neuropsychologia* **116**, 99–116 (2018).
41. Eryilmaz, H., Van De Ville, D., Schwartz, S. & Vuilleumier, P. Impact of transient emotions on functional connectivity during subsequent resting state: A wavelet correlation approach. *NeuroImage* **54**, 2481–2491 (2011).
42. Lamke, J. P. *et al.* The impact of stimulus valence and emotion regulation on sustained brain activation: Task-rest switching in emotion. *PLoS ONE* **9**, (2014).
43. Borchardt, V. *et al.* Echoes of Affective Stimulation in Brain connectivity Networks. *Cereb. Cortex* **28**, 4365–4378 (2018).
44. Gaviria, J., Rey, G., Bolton, T. A. W., Ville, D. V. D. & Vuilleumier, P. Dynamic functional brain networks underlying the temporal inertia of negative emotions. *NeuroImage* **240**, 118377 (2021).
45. Carlson, H. *et al.* Sustained effects of pleasant and unpleasant smells on resting state brain activity. *Cortex* **132**, 386–403 (2020).
46. Rey, G. *et al.* Dynamics of amygdala connectivity in bipolar disorders: a longitudinal study across mood states. *Neuropsychopharmacology* **46**, 1693–1701 (2021).
47. Gaviria, J. *et al.* Brain functional connectivity dynamics at rest in the aftermath of affective and cognitive challenges. *Hum. Brain Mapp.* **42**, 1054–1069 (2021).
48. Bolton, T. A. W. *et al.* TbCAPs: A toolbox for co-activation pattern analysis. *NeuroImage* **211**, 116621 (2020).

49. Liu, X., Zhang, N., Chang, C. & Duyn, J. H. Co-activation patterns in resting-state fMRI signals. *NeuroImage* 1–10 (2018) doi:10.1016/j.neuroimage.2018.01.041.
50. Andrews-Hanna, J. R. The brain's default network and its adaptive role in internal mentation. *Neuroscientist* **18**, 251–270 (2012).
51. Dixon, M. L. *et al.* Heterogeneity within the frontoparietal control network and its relationship to the default and dorsal attention networks. *Proc. Natl. Acad. Sci.* **115**, E1598–E1607 (2018).
52. Clare Kelly, A. M., Uddin, L. Q., Biswal, B., Castellanos, F. X. & Milham, M. P. Competition between functional brain networks mediates behavioral variability. *NeuroImage* **39**, 527–537 (2008).
53. Wu, T. *et al.* Hick-hyman law is mediated by the cognitive control network in the brain. *Cereb. Cortex* **28**, 2267–2282 (2018).
54. Protopopescu, X. *et al.* Orbitofrontal cortex activity related to emotional processing changes across the menstrual cycle. *Proc. Natl. Acad. Sci.* **102**, 16060–16065 (2005).
55. Petersen, N., Kilpatrick, L. A., Goharзад, A. & Cahill, L. Oral contraceptive pill use and menstrual cycle phase are associated with altered resting state functional connectivity. *NeuroImage* **90**, 24–32 (2014).
56. Pletzer, B., Crone, J. S., Kronbichler, M. & Kerschbaum, H. Menstrual Cycle and Hormonal Contraceptive-Dependent Changes in Intrinsic Connectivity of Resting-State Brain Networks Correspond to Behavioral Changes Due to Hormonal Status. *Brain Connect.* **6**, 572–585 (2016).
57. Andreano, J. M., Touroutoglou, A., Dickerson, B. & Feldman B., L. Hormonal Cycles, Brain Network Connectivity, and Windows of Vulnerability to Affective Disorder. *Trends Neurosci.* **41**, 660–676 (2018).
58. Endicott, J. The menstrual cycle and mood disorders. *J. Affect. Disord.* **29**, 193–200 (1993).
59. Eryilmaz, H., Van De Ville, D., Schwartz, S. & Vuilleumier, P. Lasting Impact of Regret and Gratification on Resting Brain Activity and Its Relation to Depressive Traits. *J Neurosci* **34**, 7825–7835 (2014).
60. Ullsperger, M., Planck, M., Cognitive, H. & Sciences, B. The conflict adaptation effect : It ' s not just priming. **5**, 467–472 (2005).
61. Rey-Mermet, A. *Finding an Interaction between Stroop Congruency and Flanker Congruency Requires a Large Congruency Effect: A within-Trial Combination of Conflict Tasks. Attention, Perception, and Psychophysics* vol. 82 (Attention, Perception, & Psychophysics, 2020).

62. Izard, C. E., Frühholz, S., Godde, B., Finke, M. & Herrmann, M. Spatio-temporal brain dynamics in a combined stimulus-stimulus and stimulus-response conflict task. *NeuroImage* **54**, 622–634 (2011).
63. Verbruggen, F., Liefoghe, B., Notebaert, W. & Vandierendonck, A. Effects of stimulus-stimulus compatibility and stimulus-response compatibility on response inhibition. *Acta Psychol. (Amst.)* **120**, 307–326 (2005).
64. Egner, T., Etkin, A., Gale, S. & Hirsch, J. Dissociable Neural Systems Resolve Conflict from Emotional versus Nonemotional Distracters. *Cereb. Cortex* **18**, 1475–1484 (2008).
65. Macleod, C. M. Half a Century of Research on the Stroop Effect : An Integrative Review. **109**, 163–203 (1991).
66. Verguts, T., Notebaert, W., Kunde, W. & Wühr, P. Post-conflict slowing : cognitive adaptation after conflict processing. 76–82 (2011) doi:10.3758/s13423-010-0016-2.
67. Preti, M. G., Bolton, T. A. W. & Van De Ville, D. The dynamic functional connectome: State-of-the-art and perspectives. *NeuroImage* **160**, 41–54 (2017).
68. Chang, C. *et al.* Tracking brain arousal fluctuations with fMRI. *Proc. Natl. Acad. Sci. U. S. A.* **113**, 4518–23 (2016).
69. Liu, X. & Duyn, J. H. Time-varying functional network information extracted from brief instances of spontaneous brain activity. *Proc. Natl. Acad. Sci.* **110**, 4392–4397 (2013).
70. Li, W., Li, Y., Hu, C., Chen, X. & Dai, H. Point process analysis in brain networks of patients with diabetes. *Neurocomputing* **145**, 182–189 (2014).
71. Matsui, T., Murakami, T. & Ohki, K. Neuronal Origin of the Temporal Dynamics of Spontaneous BOLD Activity Correlation. *Cereb. Cortex* **29**, 1496–1508 (2019).
72. Liu, X., Chang, C. & Duyn, J. H. Decomposition of spontaneous brain activity into distinct fMRI co-activation patterns. *Front. Syst. Neurosci.* **7**, 101 (2013).
73. Monti, S., Tamayo, P., Mesirov, J. & Golub, T. Consensus clustering: A resampling-based method for class discovery and visualization of gene expression microarray data. *Mach. Learn.* **52**, 91–118 (2003).
74. van de Schoot, R. & Miočević, M. *Small Sample Size Solutions. Small Sample Size Solutions* (Routledge, 2020). doi:10.4324/9780429273872.

75. Smid, S. C., McNeish, D., Miočević, M. & van de Schoot, R. Bayesian Versus Frequentist Estimation for Structural Equation Models in Small Sample Contexts: A Systematic Review. *Struct. Equ. Model.* **27**, 131–161 (2020).
76. Rosseel, Y. Small Sample Solutions for Structural Equation Modeling. in *Small Sample Size Solutions* 226–238 (Routledge, 2020). doi:10.4324/9780429273872-19.
77. Merkle, E. C. & Rosseel, Y. Blavaan: Bayesian Structural Equation Models Via Parameter Expansion. *J. Stat. Softw.* **85**, (2018).
78. Makowski, D., Ben-Shachar, M. & Lüdtke, D. bayestestR: Describing Effects and their Uncertainty, Existence and Significance within the Bayesian Framework. *J. Open Source Softw.* **4**, 1541 (2019).
79. Chen, J. Y. & Johnson, M. K. The Stroop congruency effect is more observable under a speed strategy than an accuracy strategy. *Percept. Mot. Skills* (1991) doi:10.2466/pms.1991.73.1.67.
80. Egner, T. Congruency sequence effects and cognitive control. *Cogn. Affect. Behav. Neurosci.* **7**, 380–390 (2007).
81. Rey-Mermet, A., Gade, M. & Steinhauser, M. Sequential conflict resolution under multiple concurrent conflicts: An ERP study. *NeuroImage* **188**, 411–418 (2019).
82. Rey, G. *et al.* Modulation of brain response to emotional conflict as a function of current mood in bipolar disorder: Preliminary findings from a follow-up state-based fMRI study. *Psychiatry Res. - Neuroimaging* **223**, 84–93 (2014).
83. Kerns, J. G. Anterior Cingulate Conflict Monitoring and Adjustments in Control. *Science* **303**, 1023–1026 (2004).
84. Egner, T. & Hirsch, J. Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nat. Neurosci.* **8**, 1784–1790 (2005).
85. Ashburner, J. *et al.* SPM12 Manual. *Funct. Imaging Lab.* 475–1 (2013) doi:10.1111/j.1365-294X.2006.02813.x.
86. Uddin, L. Q., Yeo, B. T. T. & Spreng, R. N. Towards a Universal Taxonomy of Macro-scale Functional Human Brain Networks. *Brain Topogr.* **32**, 926–942 (2019).

87. Buckner, R. L. & DiNicola, L. M. The brain's default network: updated anatomy, physiology and evolving insights. *Nat. Rev. Neurosci.* (2019) doi:10.1038/s41583-019-0212-7.
88. Bassett, D. S., Yang, M., Wymbs, N. F. & Grafton, S. T. Learning-induced autonomy of sensorimotor systems. *Nat. Neurosci.* **18**, 744–751 (2015).
89. Seeley, W. W. The Salience Network: A Neural System for Perceiving and Responding to Homeostatic Demands. *J. Neurosci. Off. J. Soc. Neurosci.* **39**, 9878–9882 (2019).
90. Kragel, P. A., Reddan, M. C., LaBar, K. S. & Wager, T. D. Emotion schemas are embedded in the human visual system. *Sci. Adv.* **5**, (2019).
91. Niendam, T. A. *et al.* Meta-analytic evidence for a superordinate cognitive control network subserving diverse executive functions. *Cogn. Affect. Behav. Neurosci.* **12**, 241–268 (2012).
92. Cole, M. W., Ito, T., Bassett, D. S. & Schultz, D. H. Activity flow over resting-state networks shapes cognitive task activations. *Nat. Neurosci.* **19**, (2016).
93. Widaman, K. F., Ferrer, E. & Conger, R. D. Factorial Invariance Within Longitudinal Structural Equation Models: Measuring the Same Construct Across Time. *Child Dev. Perspect.* **4**, 10–18 (2010).
94. Kruschke, J. K. Rejecting or Accepting Parameter Values in Bayesian Estimation. *Adv. Methods Pract. Psychol. Sci.* **1**, 270–280 (2018).
95. Makowski, D., Ben-Shachar, M. S., Chen, S. H. A. & Lüdtke, D. Indices of Effect Existence and Significance in the Bayesian Framework. *Front. Psychol.* **10**, 1–14 (2019).
96. Bassett, D. S., Yang, M., Wymbs, N. F. & Grafton, S. T. Learning-induced autonomy of sensorimotor systems. *Nat. Neurosci.* **18**, (2015).
97. Cole, M. W., Repovš, G. & Anticevic, A. The frontoparietal control system: A central role in mental health. *Neuroscientist* **20**, 652–664 (2014).
98. Dixon, M. L. *et al.* Interactions between the default network and dorsal attention network vary across default subsystems, time, and cognitive states. *NeuroImage* **147**, 632–649 (2017).
99. Fox, M. D. *et al.* The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proc. Natl. Acad. Sci.* **102**, 9673–9678 (2005).

100. Waugh, C. E., Shing, E. Z. & Avery, B. M. Temporal Dynamics of Emotional Processing in the Brain. *Emot. Rev.* **7**, 323–329 (2015).
101. Andrews-Hanna, J. R., Smallwood, J. & Spreng, R. N. The default network and self-generated thought: Component processes, dynamic control, and clinical relevance. *Ann. N. Y. Acad. Sci.* **1316**, 29–52 (2014).
102. Fox, K. C. R. *et al.* Affective neuroscience of self-generated thought. *Ann. N. Y. Acad. Sci.* **1426**, 25–51 (2018).
103. Kuppens, P. Its about Time: A Special Section on Affect Dynamics. *Emot. Rev.* **7**, 297–300 (2015).
104. Waugh, C. E., Hamilton, J. P. & Gotlib, I. H. The neural temporal dynamics of the intensity of emotional experience. *NeuroImage* **49**, 1699–1707 (2010).
105. Uddin, L. Q. Salience processing and insular cortical function and dysfunction. *Nat. Rev. Neurosci.* **16**, 55–61 (2015).
106. Underwood, R., Tolmeijer, E., Wibroe, J., Peters, E. & Mason, L. Networks underpinning emotion: A systematic review and synthesis of functional and effective connectivity. *NeuroImage* **243**, 118486 (2021).
107. Critchley, H. D., Wiens, S., Rotshtein, P., Öhman, A. & Dolan, R. J. Neural systems supporting interoceptive awareness. *Nat. Neurosci.* **7**, 189–195 (2004).
108. Adolphs, R., Tranel, D. & Damasio, A. R. Dissociable neural systems for recognizing emotions. *Brain Cogn.* **52**, 61–69 (2003).
109. Piguet, C. *et al.* Neural substrates of rumination tendency in non-depressed individuals. *Biol. Psychol.* **103**, 195–202 (2014).
110. Critchley, H. D. & Garfinkel, S. N. Interoception and emotion. *Curr. Opin. Psychol.* **17**, 7–14 (2017).
111. Posner, J., Russell, J. A. & Peterson, B. S. The circumplex model of affect: An integrative approach to affective neuroscience, cognitive development, and psychopathology. *Dev. Psychopathol.* **17**, 715–734 (2005).
112. Satpute, A. B., Shu, J., Weber, J., Roy, M. & Ochsner, K. N. The Functional Neural Architecture of Self-Reports of Affective Experience. *Biol. Psychiatry* **73**, 631–638 (2013).

113. Grimm, C. *et al.* Tonic and burst-like locus coeruleus stimulation distinctly shift network activity across the cortical hierarchy. *Nat. Neurosci.* **27**, 2167–2177 (2024).
114. Blakemore, R. L. & Vuilleumier, P. An emotional call to action: Integrating affective neuroscience in models of motor control. *Emot. Rev.* **9**, 299–309 (2017).
115. Cai, W., Ryali, S., Chen, T., Li, C. S. R. & Menon, V. Dissociable roles of right inferior frontal cortex and anterior insula in inhibitory control: Evidence from intrinsic and task-related functional parcellation, Connectivity, And response profile analyses across multiple datasets. *J. Neurosci.* (2014) doi:10.1523/JNEUROSCI.3048-14.2014.
116. Cai, W. *et al.* Causal Interactions Within a Frontal-Cingulate-Parietal Network During Cognitive Control: Convergent Evidence from a Multisite–Multitask Investigation. *Cereb. Cortex* **26**, 2140–2153 (2016).
117. Pievsky, M. A. & McGrath, R. E. The Neurocognitive Profile of Attention-Deficit/Hyperactivity Disorder: A Review of Meta-Analyses. *Arch. Clin. Neuropsychol.* **33**, 143–157 (2018).
118. Dosenbach, N. U. F., Fair, D. A., Cohen, A. L., Schlaggar, B. L. & Petersen, S. E. A dual-networks architecture of top-down control. *Trends Cogn. Sci.* **12**, 99–105 (2008).
119. Corbetta, M. & Shulman, G. L. Control of goal-directed and stimulus-driven attention in the brain. *Nat. Rev. Neurosci.* **3**, 201–215 (2002).
120. Hermans, E. J., Henckens, M. J. a G., Joëls, M. & Fernández, G. Dynamic adaptation of large-scale brain networks in response to acute stressors. *Trends Neurosci.* **37**, 304–14 (2014).
121. van Oort, J. *et al.* How the brain connects in response to acute stress: A review at the human brain systems level. *Neurosci. Biobehav. Rev.* (2017) doi:10.1016/j.neubiorev.2017.10.015.
122. Chepenik, L. G., Cornew, L. A. & Farah, M. J. The influence of sad mood on cognition. *Emotion* **7**, 802–811 (2007).
123. Song, S. *et al.* The influence of emotional interference on cognitive control: A meta-analysis of neuroimaging studies using the emotional Stroop task. *Sci. Rep.* **7**, 1–9 (2017).
124. Öhman, A., Flykt, A. & Esteves, F. Emotion drives attention: Detecting the snake in the grass. *J. Exp. Psychol. Gen.* **130**, 466–478 (2001).

125. Bernstein-Eliav, M. & Tavor, I. The Prediction of Brain Activity from Connectivity: Advances and Applications. *Neuroscientist* (2022) doi:10.1177/10738584221130974.
126. Tavor, I. *et al.* Task-free MRI predicts individual differences in brain activity during task performance. *Science* **352**, 1773–1776 (2016).
127. Brown, J. A. *et al.* Patient-Tailored, Connectivity-Based Forecasts of Spreading Brain Atrophy. *Neuron* **104**, 856–868.e5 (2019).
128. Finn, E. S. *et al.* Functional connectome fingerprinting: identifying individuals using patterns of brain connectivity. *Nat. Neurosci.* **18**, 1–11 (2015).
129. Vrtička, P., Bondolfi, G., Sander, D. & Vuilleumier, P. The neural substrates of social emotion perception and regulation are modulated by adult attachment style. *Soc. Neurosci.* **7**, 473–493 (2012).
130. Young, C. B. *et al.* Dynamic Shifts in Large-Scale Brain Network Balance As a Function of Arousal. *J. Neurosci.* **37**, 281–290 (2017).
131. Aubry, J.-M. *et al.* Resting-state functional connectivity of emotion regulation networks in euthymic and non-euthymic bipolar disorder patients. *Eur. Psychiatry* **34**, 56–63 (2016).
132. Garfinkel, S. N., Critchley, H. D. & Pollatos, O. The interoceptive system: Implications for cognition, emotion, and health. *Handb. Psychophysiol. Fourth Ed.* 427–443 (2016)
doi:10.1017/9781107415782.019.
133. Harrison, B. J. *et al.* Modulation of Brain Resting-State Networks by Sad Mood Induction. *PLoS ONE* **3**, e1794 (2008).
134. Gorgolewski, K. J. *et al.* The brain imaging data structure, a format for organizing and describing outputs of neuroimaging experiments. *Sci. Data* **3**, 160044 (2016).
135. Wilkinson, M. D. *et al.* The FAIR Guiding Principles for scientific data management and stewardship. *Sci. Data* **3**, 160018 (2016).