

**Julian Gaviria Lopez**

Department of Psychiatry, Amsterdam UMC  
Oldenaller 1, 1081 HJ Amsterdam, the Netherlands  
Email: jualgalo@gmail.com

**Major revisions of manuscript NCOMMS-22-35936, " FMRI network dynamics underpinning the impact of affective carry-over on cognitive control"**

Special thanks to the reviewers for their very constructive comments. We were glad they found interest in our work. Below, we provide detailed answers to the comments raised during the revision in a point-by-point fashion. Comments and questions from the reviewers are written in bold font, followed by our reply in regular font. When required, corresponding modifications in the new version of the manuscript are also marked with the green highlight tool in the respective files (main text and/or SI supplementary information).

**Commented [PV1]:** We need to have a very short letter to editor as well – to apologize for delay due to xxxx. And if there is a place for comments to the editor separate from reviewers, maybe you can briefly underscore the very length and redundancy of some comments of rev 1 and his / lack of familiarity with concept of dFC despite now standard method?!

**Commented [JG2R1]:** Good idea. Please find it in the attachments

**REVIEWER N°1.****1. As this paper stands it's impossible to understand or evaluate the fMRI methods as there is far too little detail given. Why did the authors choose this methodological approach to examining dynamic network activity? What methodological steps are involved?**

We are sorry if this methodology aspect was not sufficiently backed up, as CAP analysis has been described in details in previous publications by different teams<sup>1-6</sup> and already used in our own work<sup>7,8</sup>. Based on this literature, we chose the CAPs approach for the current study because, unlike other dynamic functional connectivity methods (dFC), it provides single-volume temporal resolution, with a minimal set of assumptions, constraints, and transformations of fMRI data<sup>2</sup>. Moreover, the single-volume resolution enables the exclusion of time points corrupted by noise without consequences for neighbor volumes<sup>9</sup>. This practical cleaning strategy improves outcomes compared to more classical analytical dFC strategies, characterized by a strong dependency on the time series of the fMRI volumes, such as slide window-based methods. We thank the reviewer for prompting us to clarify the rationale for using this approach. These arguments and additional details (with references to corresponding papers) have been included in the manuscript. Cf. "Materials and Methods. Stage 1: Dynamic functional connectivity analysis (CAPs identification)".

**Commented [PV3]:** Check it is done

**2. The Discussion section is mostly focused on reverse inference of cognitive processes evoked by CAP networks – a kind of network reverse-inference blobology, which doesn't do the study justice.**

We apologize if the discussion created this impression and have revised our wording in several places to prevent it. Describing the present study as "network reverse-inference blobology" is unfairly severe in our view due to the following reasons. (I) The CAP methodology specifically enabled us to define specific networks without any a priori on their role, and compare them with well-established functional systems that have been extensively reported and characterized in the literature<sup>10</sup>. Hence, the only valid "reverse inference" we make is to link our CAPs to these classic networks (e.g., DMN, DAN)

based on their spatial similarity, while the cognitive or affective functions associated with them are entirely based on conventional knowledge about these networks. (II) Unlike reverse inference, our study evaluated the predictive power of *quantifiable* brain patterns on *measurable* outcomes such as task-evoked brain activity. Namely, we predicted (rather than post-hoc infer) how prior dFC features of brain networks influence later network states and behavior. Furthermore, we tested that prediction with formal mediation. That aligns with a brain-as-predictor framework<sup>11-15</sup>, not with the reverse-inference fallacy<sup>16,17</sup>. (III) The implementation of a data-driven approach prevented any form of circularity in the analyses, which is a common mistake in reverse inference<sup>18,19</sup>. As we now more carefully emphasize, our discussion highlights this fact and warns readers against reverse inferences. In the new version, the section “Discussion. The impact of negative emotions on cognitive control is linked to their aftermath on intrinsic brain network dynamics” includes the following statement: “*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, in which observed dFC patterns forecast measurable brain or behavioral outcomes without implying that the activations alone confirm specific cognitive or emotional processes.*”.

**Commented [PV4]:** Ad a brief sentence somewhere to make this stement

**The contribution of this study would be much clearer if the authors discussed what the findings tell us about how or why measuring recurrence of fluctuating brain network patterns over time provides insight into carryover effects of emotional events.** And, given that, what are the implications for our understanding of such patterns of activity for real world applications related to mood disorders but also potentially performance of demanding task in emotionally fraught contexts?

We recognize the reviewer's concern that this point might have been insufficiently addressed. Therefore, we revisited the discussion to highlight the importance of considering affective processes as dynamic fluctuations evolving over time across multiple brain networks. In the new version of the manuscript, the introductory paragraph of the section “4.5. Discussion” initiates as follows: “*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.* Indeed, ongoing coherent activity is present both before and after emotion-eliciting events, rather than occurring in an on-off manner, and the recruited networks are partly shared with those involved in non-emotional processes or tasks. This aspect is entirely consistent with the theoretical perspective of component process models of emotion<sup>20</sup>, and accords with further neuroscientific evidence that describes a close interaction/overlap of cognitive/affective functions in the brain<sup>21</sup>. As we emphasize in our paper (Cf., “Introduction” section Page 1), such continuity in successive brain states during emotional episodes provide a valuable framework to account for the multiple influences of emotion on behavior and cognition, including memory,

**Commented [PV5]:** Add this point in intro or disc?

perception, action, thought, or mood<sup>22-25</sup>. Further details are also described in our response to question 25, in which the reviewer discusses this issue again.

Commented [pv6]: Make sure we have a couple of sentence on this in discussion

## Methods

### 3. What was the justification of the final sample size? Was a power analysis performed?

We acknowledge we did not address this point in our paper. However, there is no unique straightforward approach to calculate power for a new experimental design. Power is essentially a frequentist concept. In the Bayesian approach, by contrast, effects are a weighted result between the data and the prior. Namely, the validity of one effect lies in its level of dominance on the priors. Therefore, a major concern in Bayesian analysis regards to the impact of prior distributions on the estimation efficiency<sup>26</sup>, parameter estimation<sup>27</sup>, and model fit and selection<sup>28</sup>, rather than power values. For this reason, we performed a new sensitivity analysis<sup>29</sup> (cf. Table S8) that confirmed no impact of the selected priors on the posterior distributions. The new version of our manuscript includes five different assessments of model convergence and priors performance which converge to back up the reliability of results in our sample. They are found in the supplementary information, "Model convergence and priors assessment" section. Thank you for prompting us to clarify and justify this point.

### 4. I just want to note that is a great strength that the study took menstrual phase into account. As hormone levels have been found to influence many cognitive processes as well as emotional responses, more cognitive neuroscience studies should time scans by cycle phase.

We thank the reviewer for this appreciation. This was indeed a concern we shared with them.

### 5. Figure 1 is challenging to interpret. In Fig 1 b., the X axis just says subjective ratings, with no explanation of the scales (e.g., that valence ratings are on a bipolar scale and are higher for positive, whereas arousal and presumably emotional experience are unipolar scales where higher levels simply indicate more). The same issue goes for the PANAS scores in Fig 1D. The readers shouldn't need to have to refer to the supplemental materials to interpret the plots.

We apologize for these omissions. The new version of Figure 1 includes Y-axis for each plot. Based upon this comment, we specified in the legend the unipolar sense in both the Likert scales of the subjective assessments of the films (i.e., emotional experience, valence, arousal) and PANAs scores.

### 6. Fig 1C is particularly confusing to read/interpret. In the caption for the middle panel please explain the initials for task conditions on the x axis. In the right panel the x congruency axis reads I > C which makes me expect a difference score, but it doesn't look like a difference score.

Again, we apologize if this was not clear. All abbreviations and data followed the terms and conditions used in the text but indeed were not explained in an explicit way in the figure. In line with the reviewer's suggestion, the new legend of Figure 1C describes the different types of trials in the cognitive tasks on the x axis. In addition, the legends of Y-axis for each panel are more explicit. The legend of the

congruency effect ( $C < I$ ) indicates greater reaction time in incongruent trials when they are compared with congruent trials. As pointed out by the reviewer, the difference between the incongruent and the congruent trials ( $I - C$ ) would require a different illustration with different values. We thank the reviewer for prompting these changes which indeed will help make Figure 1 clearer to all readers.

**7. Cognitive tasks: I'm curious about why a face /name stroop was used when it's the colour word stroop that produces the classic interference effect and is easier to interpret in terms of direct conflict between the word and the colour the word is depicting? Was it because it lent itself to binary answers? None of the supporting literature cited to support the choice of stroop task used this version of a stroop task. The Westerhausen paper used a dichotic auditory task. The Verbruggen study used a spatial stroop with a Simon task. And the Rey-Mermet used the classic colour word stroop, which differs in that the conflict is between two forms of identical features rather than two things that are typically associated with each other -- a name and the gender associated with it. While all these versions of the task induce conflict of some sort and require cognitive control, the interpretation of processes evoked can differ and none of the papers support the choice of specific task made here.**

We recognize we did not give a detailed explanation for our choice of these tasks, which were however carefully compared and validated in preparatory phases of our study. The decision about these two versions of the task is a critical point in our design, and was based on multiple criteria and systematic testing. This was already briefly mentioned in the "Cognitive control tasks" section: "our selection and design of the 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". Our first criteria was to choose different tasks to avoid learning or habituation effects across sessions that could have modified our measure of conflict monitoring and confounded the effect of emotion on control with any interaction effects with learning and time. We fully agree with the reviewer that the color word stroop produces the most classic interference effect due to the direct correspondence between stimuli meaning. However, Stroop-like paradigms have also been consistently reported and studied, particularly in relation to cognitive conflict processes<sup>30,31</sup>, providing a useful variant to minimize repetition and learning effects. Moreover, the paradigms implemented here ensured good reliability in terms of the affective modulation of conflict monitoring with a reasonable number of trials. Please also note that other classic interference paradigms such as the arrow version of the Eriksen flanker task or the Simon task increase the possible contribution of episodic binding<sup>17</sup> and negative priming<sup>18</sup>, given the increased number of repeated trials. Given this state of results in the field, we decided to use these two different tasks in a counterbalanced manner, and incidentally confirmed a generalization of attentional control as previously reported across different conflict monitoring tasks at both behavioral and brain function levels. Further, by showing highly similar and reproducible emotional effects across the two tasks, our study also allowed us to directly verify the generalization of such effects.

**Commented [pv7]:** Need to flip I and C along X axis to match the data points  
And remove ">"

**Commented [PV8]:** Just wondering if for coherence the data for reach trial type in the middle graph (cc, ic, ci, ii) could be plotted in bars (emo vs neut) like mean RT in right side graph? (instead of lines)

**Commented [JG9R8]:** The effects in the CSE (cc, ic, ci, ii) are subtle. The 2D-line graph captures better the difference, compared to bar plots.

In any case, we thank the reviewer for pointing our lack of clarity in this part of the manuscript concerning our choice. We rephrased the text and added the rationale about using two tasks (Cf., main manuscript “Materials and Methods. Cognitive control tasks” section. Page 8). In addition, Westerhausen’s work was removed from the reference since it did not support our argument as precisely as in other work by Verbruggen et al<sup>32</sup>, Izard et al<sup>33</sup>, and Rey-Mermet et al<sup>34</sup>. (Cf., main manuscript “Materials and Methods. Cognitive control tasks” section. Page 8).

#### **fMRI Methods**

**8. As I read through Methods and results, I’m struggling to find information about what the rationale for using this particular approach to measuring brain dynamics? What theoretically does number of times CAPs occur each condition tell you about what’s going on in each condition? Given the research questions, why did the authors choose an approach where you look at recurring snapshots of BOLD spatial patterns rather than time series between pairs of voxels or seeds? This needs to be spelled out explicitly from the beginning – not offloaded onto a reference to another paper.**

We chose the CAPs approach for this study because it provides single-volume temporal resolution, with a minimal set of assumptions, constraints, and transformations of fMRI data<sup>2</sup>. This methodology has been valuably used and supported by several previous studies<sup>1-3,5,6</sup>, including our own<sup>4,7,8,35</sup>. By providing “snapshots” of network activity over time and quantifying them with different indices (such as duration, entry rates, etc.), CAP analysis provides a more accurate, richer and truly dynamic measure of brain connectivity. This is unlike sliding window-based methods that are characterized by arbitrary selections of the time intervals (i.e., time windows from milliseconds to minutes) and reflect more global/coarser fluctuations averaged over many timepoints. Also, the single-volume temporal resolution of our CAP analysis enables the exclusion of time points corrupted by noise without consequences for neighbor volumes<sup>9</sup>. This practical cleaning strategy improves outcomes compared to more classical analytical strategies, characterized by a strong dependency on the time series of the fMRI volumes. We thank the reviewer for prompting us to clarify our rationale for using this approach and added more information to explain and backup the methodology. The new version of the section “Stage 1: Dynamic functional connectivity analysis (CAPs identification)” now includes this information.

**9. Also I don’t get an understanding of from the methods, as currently written, is HOW the CAP analysis captures the temporal dynamics of identified networks as they fluctuate over time. After reading the three additional papers referenced for the methods I now understand that CAPs are identified by using clustering to identify spatial patterns across the brain at single time points and identifying how often these spatial patterns occur. But I could not extract even this basic outline of information from the paper.**

Again the reviewer fairly asks for a better description of the method in the paper itself, beyond referencing to previous work. This information was partially reported in the legend of Figure 2 of our initial manuscript, but we are sorry if this was not sufficient to the Reviewer’s eye. We now provide a dedicated section in the revised manuscript (Cf., main manuscript “CAPs generation” section. Page

10) to more completely describe the CAPs pipeline. Again, we thank the reviewer for helping us to improve the manuscript readability.

**10. The authors write (p. 12): “Further description of our methodological procedure is provided elsewhere<sup>55,80</sup>” This is not adequate. Please include a description of how CAPs were identified in this manuscript, so the reader doesn’t need to dig up and read two other papers to understand what was done! Even having read the referenced papers I am still missing the rationale for a number of specific choices made in this paper:**

This point follows from the previous questions. Inspired by these valid comments, we now provide a better description of the implemented methods through the new section on “CAPs generation”. Cf., main manuscript “CAPs generation” section. Page 10

#### TbCAP toolbox choices

##### Choice of Frames

**11. Given the use of the TbCAP seed free analysis option, exactly how are coactivation patterns calculated at a particular point in time? Going to the referenced Bolton TbCAPs paper, I read that CAPS approaches typically identify frames/timepoints at which a seed region reaches a sufficiently high level of activation (or sometimes de-activation). But there is a seed-free option which the authors of the current paper selected. So, how were the relevant frames for CAP identification chosen here? It’s clear from Fig 2 that motion-corrupted frames were rejected but what frames were retained? Was it every TR? If not, what were the criteria for frame selection prior to clustering?**

Again, following on the previous points, we added the new “CAPs generation” section which now states: “Rather than selecting time points depicting extreme coactivations of a seed region to the rest of the brain, the seed-free method implemented here classifies all the time points (except the corrupted ones) with similar spatial distributions of activity according to the k-means clustering algorithm”. This seed-free has been advocated by several other papers in recent years<sup>36–38</sup>.

##### Clustering.

**13. Is the number of 5 clusters used for comparison of different numbers of clustering solutions based on the default from the “cluster” function of the TbCAPS toolbox**

In line with state of the art methodology, the number of clusters was obtained by the consensus resampling-based algorithm<sup>24</sup>. To cross-validate the consensus results, we further ran spatial similarity analyses with multiple K-means solutions ( $k=3, k=4, k=5, k=6, k=8, k=12$ ), which yielded consistent co-activation maps across the different clustering scenarios. This information is described in the new “CAPs generation” section. And supplementary results of the spatial similarity analysis is reported in Figure S3.

##### Computation of metrics.

**14. I'm making the inference that the choice made at this decision point was number of entries. Or was it raw counts? What was the rationale behind deciding on this metric specifically?**

The present study reported the counts (also known as occurrences) as the main temporal metric for two reasons. First, it is the most basic metric from which further temporal indicators are derived (see<sup>4</sup>). Second, a study investigating the neuronal origin of the temporal dynamics of BOLD activity found coordinated temporal dynamics of calcium FC and hemodynamic FC in mice<sup>39</sup>, with the occurrences of the calcium CAPs as a temporal indicator of the signal fluctuation. Matsui et al. provides evidence that the fluctuations in BOLD signal observed in fMRI studies, particularly those using CAPs, are strongly influenced by underlying neuronal activity. These changes are found in the section "Dynamic functional connectivity (dFC)", page 10 of the revised manuscript.

Commented [PV10]: So you do mention this ref in the method? Good to justify! 😊

**BSEM (from the supplementary methods).**

**15. If it's not possible to use informed priors then what is the advantage of a Bayesian over a frequentist approach?**

As stated in the section "Stage 2: Bayesian structural equation modeling (BSEM)", BSEM outperforms frequentist approaches in handling uncertainty in data caused by high variability or small sample sizes. Therefore, BSEM produced more interpretable results, even when small sample sizes are modeled with relatively complex structural equation models. In contrast, initial modeling using the frequentist approach resulted in:

- Non-convergence: The optimizer fails to find a solution that meets one or more convergence criteria.
- Inadmissible solutions: Parameter values fall outside acceptable ranges.
- Low precision of parameter estimates, indicated by very large standard errors.

**Results**

The authors write (p. 15). "These (RT) 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."

**16. Then why use two different conflict tasks in the first place? Also just because RT and accuracy results didn't differ between tasks, it doesn't necessarily follow that they are evoking exactly the same underlying brain processes....**

This question echoes the previous question 7 above. The rationale for using two different tasks probing the same cognitive domain is explained in our response to question 7 (and now added in the revised manuscript). Inferring that both tasks evoke "exactly the same underlying brain processes" would be unfounded, as the reviewer remarks. For this reason, our brain activation (i.e., GLM) and dFC (i.e., CAPs) analyses only sought to identify "convergent" brain activity across tasks, in keeping with our results showing highly reproducible behavioral patterns across tasks (as noted by the reviewer) and underscoring a consistent congruence effect (CE) in both conditions. To recall, the FPN<sub>CAP</sub> accordingly showed a clear and strong brain-behavior relationship with performance on both tasks when the occurrences of all the CAPs were regressed out to determine their response to experimental conditions, further supporting a shared neural substrate. Overall, our data thus

demonstrate a remarkable consistency of both cognitive and affective effects in our paradigm. Nevertheless, our interpretation of results does not neglect the fact that further brain systems may underpin cognitive control and any specific components of either task. However, we kept our focus on the target variables of the study in line with our theoretical predictions: The identification of brain patterns underpinning affectively modulated cognitive control as quantified behaviorally by reaction times. We believe this further ensures the strength and generalizability of our results.

**Dynamic functional connectivity results.**

**17. More information and explanation are required. This paragraph jumps into a description of finding optimal k-means solutions without setting up a frame of reference that makes the description meaningful. The methods described in the caption to Figure 2 are also very hard to follow without a frame of reference.**

We are sorry if this was not clear to the reviewer and now add two important aspects explaining the pertinence of the dFC approach implemented in this study. We underscore 1) the CAPs' capability to capture more fine-grained features of the BOLD response in comparison to methods estimating brain functioning as "static" systems; 2) the potential identification of brain networks overlooked with more conventional dFC methods based on aprioristic functional brain parcellations.

**18. This is also true in the description of the SEM results -- there are a number of references to procedures or terms that are not described or explained anywhere in the manuscript.**

We are sorry if the reviewer was unfamiliar with some of the approaches or terms used in our study. Prompted by this important comment, we carefully inspected the references in the results section describing the SEM results to ensure they provide the most relevant information to interested readers. A few references were deleted to lighten the reading of the manuscript and others were underscored. We also carefully ensured to define terms used in the manuscript. Again, we thank the reviewer for his/her help to make the manuscript more legible.

**BSEM results.**

**19. It looks as though the RTs from each task condition were entered into the mediation model separately. Is this true? When RT is found to be a mediator is this RT across all the task conditions? So similar results to what would be observed if a mean RT across all task conditions were used? Or does the difference in RT between task conditions come into play here? If so, how does that work?**

The reviewer addresses here a critical aspect of the study. The RT scores were modelled in terms of the affective valence of the condition (i.e., "NEGATIVE TASK" and "NEUTRAL TASK" sessions respectively). Figure S4 provides an illustration of our modelling strategy. To recall, the type of task (i.e., Stroop or Flanker) was counterbalanced across subjects. Thus, half of the participants performed the Stroop task in the "NEGATIVE TASK" condition, and the remaining half performed the Flanker task in the same the "NEGATIVE TASK" condition. The same procedure was implemented in the "NEUTRAL TASK" condition. These conditions were fully counterbalanced across the whole sample.

Modelling the average RT would be inappropriate in our view for the following reasons: 1) If we introduced the average RT to the current models (i.e., the same value for "NEUTRAL TASK" and "NEGATIVE TASK" conditions), any potential significant mediation may be confounded by a stronger influence already identified in the "NEGATIVE TASK" condition. 2) Another scenario would be to examine the mediation of average RT in the CAPs interactions, but this would require averaging the CAPs indices as well. Consequently, the distinction in terms of the affective valence would be completely eliminated. This second analysis scenario would fall outside the scope of the present study. We therefore believe that our method is not only valid but fully appropriate for the purpose of this analysis.

**20. What is the direction of the relationship between RT and the effect of one CAP on another? For example, in Aim 2, is more frequent occurrence of the SN-SMN CAP during the movie on occurrence of the FPN CAP during the task associated with faster or slower RTs? Or more of an effect of the negative movie induction on RTs?**

We recognize that adding these details may give useful information on the functional nature of these effects. More frequent occurrences in SN-SMN CAP<sub>NEGATIVE MOVIE2</sub> interact with higher RT<sub>NEGATIVE TASK</sub> and FPN CAP<sub>NEGATIVE TASK</sub>. Conversely, less frequent occurrences in SN-SMN CAP<sub>NEGATIVE REST1</sub> interact with higher RT<sub>NEGATIVE TASK</sub> and FPN CAP<sub>NEGATIVE TASK</sub>. Certainly, reporting the direction of these relationships between RT and CAPs occurrences could be made more explicit in the manuscript. We thank the reviewer for this remark. To address this point, the new version of the "Results" section includes the directionality of the partial mediation in the aim 1 (Cf., "results. Hypothesis 1. Task-related activity is determined by the impact of emotion on intrinsic brain network dynamics at rest". Page 25), and the directionality of the partial mediation in the aim 2 respectively (Cf., "results. Hypothesis 2. Task-related activity is determined by the impact of emotion on brain networks engaged during movie watching". Page 27). Furthermore, the new supplementary tables report the direct effects of the associations between the CAPs and RT scores in both the neutral and negative contexts. Values related to the interaction of SN-SMN CAP<sub>NEGATIVE MOVIE2</sub> and FPN CAP<sub>NEGATIVE TASK</sub> are also reported. Cf., SI. Tables S9, S10, S13, S14).

**21. I didn't see where in the text the comparison of the magnitude effects between neutral and negative contexts in the BSEM models with RT mediators, illustrated in Fig 5c, was reported. If I understand the results illustrated in Figure 5C correctly, in the direct comparison of magnitude effects between neutral and negative contexts in the models with mediation, what really differed for both hypothesis one and two was the degree to which the SN\_SFN in either movie or rest predicted FPN CAP occurrence during the task as a direct effect. Is this correct? If so, is it not worth emphasizing?**

The reviewer rightly points upon the significant difference in terms of direct and total effects of SN-SMN CAP<sub>REST</sub> and SMN CAP<sub>MOVIE</sub> on subsequent FPN CAP<sub>TASK</sub>. This association was noted and discussed in the manuscript (cf., "Discussion. The impact of negative emotions on cognitive control also reflects the brain response to emotion-eliciting events". Page 35). However, we did not dwell on the specific effects of SN-SMN on FPN more than on other remarkable associations (e.g., DMN ~ FPN), focusing on the broader objectives of your study – namely, to describe brain networks whose dFC varied according to emotion and accounted for an impact on task performance. We feel this

**Commented [PV11]:** I am not sure to see which are the numbers the reviewer is focussing on in order to make this conclusion.. Can we re discuss together about the meaning of these values in the fig ?

**Commented [JG12R11]:** The reviewer is correct. According to Figure 5C, the only "significant" differences in terms of the affective context relate to SN-SMN~FPN in aims 1 and 2. "IN ROPE" values <1 are generally considered significant. However, I agree with your focus on the global results. Additionally, if the interaction between DMN and FPN was not significantly different between contexts, it does not mean that the interaction was not different at all! In this case, significance indicates the strength of the association, not its presence. I avoid stating this "magnitude/existence" issue to prevent the natural question: If this is the case, then why do you compare them?

broader description of neural functional substrates underlying the impact of negative emotions on cognitive control should be sufficient for the current manuscript, without further complexifying the results with additional details and risks for overinterpretation. Importantly, all these effects are reported in text and figures (as picked up by the reviewer). We therefore added only a brief summary statement in the text to underscore these effects.

**22. Also not mentioned is the finding (Tables S14, S16) is the direct and total effect of FPN CAP occurrence during movie watching on FPN occurrence during task. So it looks as though, if I'm interpreting the table correctly, that in the absence of strong emotional context, < in the main text as well.**

Here again, the reviewer correctly highlights an interesting result regarding the association between  $FPN_{MOVIE2}$  and  $FPN_{TASK}$  in both neutral and negative contexts. Similarly, associations between “ $DMN_{REST1}$ ” and “ $FPN_{TASK}$ ” conditions were also significant in both contexts. In the main manuscript, these effects are shown in Figure 4C and described in the corresponding results section. The Supplementary Information also reports this interaction in tables S13, S14, and S16, as the reviewer pointed out. Unlike  $FPN_{MOVIE2} \rightarrow FPN_{TASK}$  associations, changes in the relationship between “ $DMN_{REST1}$ ” and “ $FPN_{TASK}$ ” were explained (i.e., mediated) by behavioral performance indices, such as reaction time. We fully agree with the interpretation proposed by the reviewer, which also seems the most plausible to us, and we added a summary statement to clarify this in the results section. We avoided more elaborate speculation about these network relationships without more specific experimental manipulations to prevent the risk of reverse inferences, as rightly advised by the reviewer. However, we also rephrased part of our discussion of these results to make it more explicit that we share the same tentative interpretation with the reviewer (cf. “4.5. Discussion. The impact of negative emotions on cognitive control also reflects the brain response to emotion-eliciting events”. Page 33). . Thank you for helping us present and explain these results more clearly.

## DISCUSSION

**23. The authors write (P. 27): “We show here that enhanced DMN at rest after negative events predicts enhanced FPN recruitment by a subsequent cognitive task .” Is “enhanced” the appropriate description for an increase in the occurrence of DMN CAPs at rest? Given its conventional use, readers are likely to associate “enhanced” with “more” activation. And since readers are less used to thinking in terms of numbers of occurrence of network co-activation than in terms of magnitude of BOLD activation, it would help if the language consistently emphasizes that that is the measure being interpreted.**

The discussion has been edited in concordance with this observation. The new version of this section describes the results in terms of the number of occurrences. We thank the reviewer for this remark.

**24. There are a lot of interpretations that could be made about DMN activity carryover from rest to task in the negative condition. One important feature of DMN activity is it is consistently linked to internally-directed attention. An alternative interpretation to emotion regulation is the role of the DMN in episodic/autobiographical memory. It seems that an equally plausible interpretation of DMN carryover could be the more frequent and persistent re-occurrence of memories evoked by or associated with the negative movie**

**clips during rest and task. This arguably could also have an impact on cognitive control capacity. But this multiplicity of interpretations also speaks directly to the problems and limitations of reverse inference of activation patterns.**

We fully agree with the reviewer and noted this alternative hypothesis. To take into account this important remark and add support to potential interpretations, we assessed the relationship between DMN occurrences and subjective affective scores (measured on PANAS scale). Our results yielded a significant correlation between DMN CAP<sub>NEGATIVE REST1</sub> and the negative affect scores following the elicitation of negative emotions (negative context). Conversely the SN-SMN CAP<sub>NEGATIVE REST1</sub> was anticorrelated to the same negative affect scores. Notably, this correlation was not observed in the neutral context and was stronger in magnitude for DMN in the REST1 condition in comparison to "MOVIE2" and "TASK". These results have been included in the new version of the manuscript (cf. Main manuscript "Results. Association of affective scores with CAPs" section, Figure 4A, and the "discussion" section, respectively). This finding supports a link between DMN<sub>CAP</sub> functional dynamics and affect. As noted above, we refrained from proposing more elaborate interpretations precisely to avoid the risk of reverse inferences and always put forward hypotheses based on current standard knowledge of brain network functions, as suggested by the reviewer in his/her questions and comments.

**25. In general there is a LOT of reverse inference and speculation the Discussion section that could be scaled back substantially (maybe making room for more concrete explanation of the methods earlier on). Moreover, the reverse inference approach in this section basically treats the CAP patterns as network level blobology. It would be much more informative if there were instead discussion of the importance and role that the frequency of fluctuating network configurations play, and of the importance of measuring them to understand emotional carryover effects -- rather than trying to reverse infer of the function of the key networks identified by the CAP analysis.**

We acknowledge the reviewer's concern regarding the global interpretations of our results and the criticism for reverse inferences. Please note, however, that this criticism better applies to many other studies than ours, where resting state fMRI is typically measured and interpreted without any control or manipulation of the (e.g., affective or cognitive) context preceding rest. In our view, a key contribution of the present study lies in the empirical demonstration of how transient features of the "intrinsic/spontaneous" functional brain networks (i.e., generated and sustained by the brain itself<sup>40</sup>) are driven by external stimuli or overt task demands. Thus, our findings add new knowledge regarding these brain networks. We believe this is a significant contribution to the fields of basic and translational neuroscience.

We acknowledge the reviewer's concern about the overall interpretation of our results and the criticism of reverse inferences. However, this criticism is more applicable to many other studies than to ours, where resting state fMRI is usually measured and interpreted without any control or manipulation of the (e.g., affective or cognitive) context before rest. In our view, a key contribution of this study is in the empirical demonstration of how transient features of the "intrinsic/spontaneous" functional brain networks (i.e., generated and maintained by the brain itself<sup>40</sup>) are influenced by

external stimuli or overt task demands. Therefore, our findings provide new insights into these brain networks. We believe this is a meaningful contribution to the fields of basic and translational neuroscience. We also believe that our results and interpretations go well beyond the “blobology” noted by the reviewer, as we address network dynamics, not activity level per se, analyzed across time points and task contexts, not in static contrasts between conditions.

Nevertheless, to mitigate this concern, different actions were taken in this revision along the lines proposed by the reviewer. First, the methods section provides a more detailed description of the implemented methodology (see previous responses to comments 10, 11, and 13) to highlight the dynamic nature of brain activity and its relevance to affective processing as emotions are dynamic in essence but this aspect is rarely studied in standard fMRI studies. Second, our interpretation on DMN<sub>CAP</sub> and SN-SMN<sub>CAP</sub> in the NEGATIVE REST1 block is now more solidly based on their link to negative affect, as reported now in the result sections. Importantly, all putative functions attributed to the CAP networks discussed in our paper are entirely grounded on extant literature and current knowledge concerning these networks. Our main interpretation is now reformulated as follows: “Based on the anticorrelation between DMN and SN-SMN systems and their link with negative affect scores, we speculate that, in post-emotional resting state, the SN-SMN system might encode interoceptive signals and bodily feelings through an interaction with self-referential representations elaborated in DMN areas that together contribute to subjective emotional experience” (cf., “4.5. Discussion. The impact of negative emotions on cognitive control is linked to their aftermath on intrinsic brain network dynamics”. Page 32). We thank the reviewer for their interest and very careful reading of our paper, as well as the many highly valuable comments that helped us to clarify and strengthen our work.

~~In addition the statement below has been removed, since we lack physiological data that links directly the observed BOLD activity with the psychological construct of arousal at resting state. “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 arousal<sup>41</sup>, which constitutes a crucial but poorly understood dimension of emotional experience<sup>42</sup> underlying the regulation of attentional resources by affective signals<sup>43</sup>.~~

**Commented [PV13]:** Not obvious here to which anticorrelation you refer too here? In general in literature ? In terms of main occurrences of our CAPS? In relation to emotional effects ??

**Commented [PV14]:** NO – we should keep – and perhaps rephrase and cite work of Valerio Zerbini on LC

**Commented [JG15R14]:** These lines were added again. Let me know if you think Zerbini's work was correctly added.

**REVIEWER#2:**

**26.** This is a well-written paper describing a thoughtful study examining the neural basis of how negative emotion influence cognitive control processes. They also use an interesting approach to examining dynamic patterns of functional connectivity (co-activation patterns; CAPs) in functionally defined neural networks that map reasonably well onto canonical resting state networks. They show that their emotion induction has the expected cognitive/behavioral effects during cognitive control, which lends confidence to the concomitant neural results.

We thank the reviewer for his/her encouraging comments about the originality and novelty of our study, and the helpful suggestions. In our revision, we made several changes in order to address all points raised by the reviewer.

**27.** That said, I am not sure the study is of the level of impact associated with a journal like Nature Communications. My largest concern here is that the sample size is quite small in relation to current high-impact fMRI studies (N=24). They use sophisticated Bayesian analyses to help address this, but it nonetheless leads to relatively low confidence in the replicability or generalizability of the results.

We agree that our sample size may appear modest, but we believe our experimental design and statistical modelling provide robust and innovative results aligning with the high standards of Nature Communications. Please note that our population sample was scanned on multiple conditions and different sessions, yielding a total of 2340 time-points per subject thanks to the high temporal resolution of our fMRI multiband sequence optimized for the purpose of dynamic functional connectivity measures used in our CAP analysis. We also used robust permutation statistics. Hence we consider that our work is solid and accords with state-of-the-art methodology. More generally, in line with previous works published in this journal<sup>44,45</sup>, we think that multiple factors determine the quality of scientific results. The suitability of the methodology for the obtained data and the sampling density have greater relevance than the sole sample size. Additionally, previous findings on the neural carryover effects of emotions<sup>46,47</sup> and emotion regulation<sup>48</sup> have been reported and replicated with smaller samples size.

Beyond this, I will list a number of major and minor issues throughout in the order they came up as I read the manuscript:

**28.** Starting in the abstract, but also in several other places, they use acronyms without first defining them. Two examples are FPN and IFNs. They also call the FPN (which I assume stands for frontoparietal network) by other names instead despite using the acronym, such as cognitive control network or executive control network. I'd keep network naming consistent and matching the acronyms.

Thanks to the reviewer for highlighting this inconsistency. The acronyms have been inspected and respectively explained along the manuscript. We also made several changes to stick with a single term for the same network throughout the paper.

Commented [PV16]: We add more included ? This is final dataset, right? Maybe just note it

Commented [JG17R16]: This is the very final dataset. And we used 6 blocks out of 10 for this study.

Commented [PV18]: Cite paper that compared data in n = small with dense sampling vs n= high with short /single session?

Commented [JG19R18]: It's unclear what you mention here. If you have a ref. in mind, please add DOI. 😊

**29. The introduction sometimes refers to effects without stating their directions or other details that would help with interpreting their reasoning. For example, they state: “Several studies reported behavioral changes in attentional processing induced by emotional stimuli, including effects on spatial orienting and resistance to distractor interference in conflict tasks”. In this case, it would be helpful to indicate which emotions (or what types of emotional cues) and specifically how they affect orienting and distractor resistance.**

The sentence mentioned by the reviewer aimed to illustrate the consistent impact of emotions on cognitive processing, but various and multidirectional effects have been reported in the literature. We therefore used a short (but less precise) summary statement to refer to these findings. However, following this pertinent suggestion, we now better describe the directionality of some of these effects (i.e., the impact of negative affect on cognitive control) in the first line of the 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 attention” (Cf., “introduction” section. Page 1).

**30. They underline words several times in the text. This seemed atypical, but is up to journal formatting rules.**

All the underlines have been removed. Thanks for this feedback. However, underlined words can be italicized in the final version if needed.

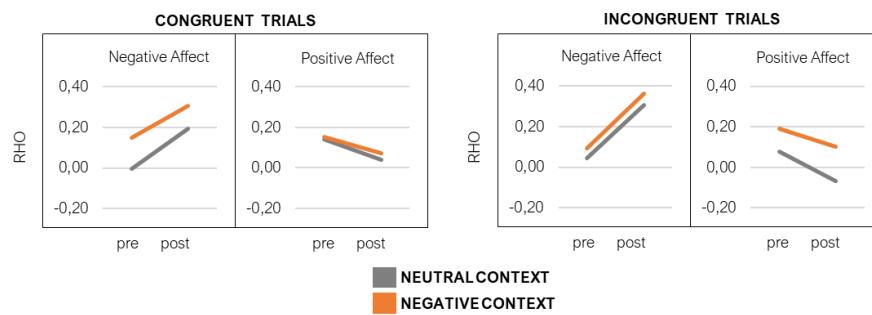
**31. Typo – 2nd to last line on page 5, “tracking” should be “track”. On the next line, “voxel” should maybe be “single-voxel” or “voxel-wise”?**

This typo was fixed in the new version of the manuscript (Cf., main manuscript “Introduction”. Page 3). Thanks for reporting it.

**32. How well was the content of the videos matched other than the affect-induction aspects. One might be concerned that relevant network effects involved priming from other content differences as opposed to the emotional differences. It would have been nice to see correlations between neural/behavior effects and individual differences in the magnitude of change in affective state (e.g., did those who felt worse after the negative movie also show stronger network or RT differences?). If so, it would help support the idea that it is the affect change and not other content differences that matter.**

This is a valid point that is often hard to address in a fully controlled way in naturalistic movie studies<sup>49</sup>. Our selection was guided by a broad matching of the variety of thematic contents (e.g. people alone, couples, mother-child interactions, indoor/outdoor scenes, etc.) but a precise identification and quantification of specific dimensions would be beyond the scope of this study. We do agree however that this would constitute an interesting issue warranting further investigation. To take this into account more formally, and better relate our findings to affective state proper, we ran further Bayesian assessments of the relationship between cognitive performance and affective scores and found positive correlations between the reaction time of congruent and incongruent trials and the negative

affect scores on PANAS scale before and after movie stimuli in both contexts (i.e., neutral and negative). The correlation between the negative affect scores measured after the movies with negative-valenced content and trials with the greatest interference effect (i.e., incongruent trials of the negative context) showed the largest magnitude ( $\rho = .36$ ; 95% CI(0.04<sub>lower</sub>, 0.79<sub>upper</sub>); ROPE: 7%; **Figure R1**. Third panel from left to right]. On the other side, the same congruent and incongruent trials were less associated with positive affect scores in both neutral and negative contexts, especially after exposure to the affective stimuli. Together, these results indicate that the affective valence of previous affective events is linked to changes in cognitive control. We also report additional analysis showing a correlation between subjective negative affect and network activity (cf., response to question 24). Although correlations do not provide causal evidence, these data allow linking emotion experience more directly to our relevant experimental measures of cognitive control, beyond more incidental difference in movie content or sensorial aspects.



**Figure R1.** Bayesian correlation coefficients (RHO) of the relationship between cognitive performance (i.e., reaction time of congruent and incongruent trials) and positive and negative subjective scores were examined before (pre) and following (post) the affective elicitation in the negative and neutral context, respectively.

**Commented [JG20]:** Patrik. I cannot explain: 1) why PANAS are linked to RT in both "neu" & "neg" contexts. 2) ~why "post" PANAS are more correlated than "pre" PANAS. However, these results address the question: Association between PANAS and RT.

### 33. I would have liked to see how emotions from the first movie affected subsequent resting state neural network activity. It would also help if they explained more what they found in their previous study analyzing the 2nd rest period, as it could be relevant to interpreting their findings here.

Thanks to the reviewer for this thoughtful suggestion. We agree that examining how emotions evoked by the first movie propagate into the subsequent resting-state period is an important question. We addressed this exact issue in our prior work<sup>8</sup> as pointed out by the reviewer. In the present study, we deliberately chose not to re-analyse brain associations depicted during the first movie and the subsequent rest segment, given that the new analyses (Cf. responses to questions 24 and 32), already provide direct evidence that CAPs occurrences are linked to fluctuations of the affective status. Additionally, the link between brain (i.e., CAPs) and emotions (i.e., behavior) has been included in multiple sections of the new version of the main manuscript.

**34. I found it a bit strange (and not theoretically motivated) that RTs were treated as mediators. Presumably, the RTs are a RESULT of neural activity during the cognitive control tasks, and their hypothesis is that the emotion induction affects this task-related neural activity. So the natural causal model to me would be that the emotion induction effects during rest/movie would have an influence on RTs as mediated by network activity during the cognitive control task.**

We thank the reviewer for raising this point; it is indeed a classic and mechanistic view that neural activity during the task precedes and drives reaction times. In the present work, however, we took a brain-to-brain perspective (see also response to question 2). Our goal was to quantify how the affective context (induced by naturalistic stimuli) modulates the brain activity (i.e., CAPs) observed during cognitive control challenges. Our rationale is consistent with accumulating evidence showing that brain-derived metrics often predict later brain states more powerfully than any single overt "behavioral" scores<sup>11,50,51</sup>. Because behavior is an abstract, multi-level construct—including RT, accuracy, autonomic, endocrine and neuroimaging indices—we sought to exploit the full neural signature rather than insist on a unidirectional neural → RT causal path. We now clarify this rationale in the revised Discussion (Cf., "Discussion" section. Page 34).

**35. They treated two cognitive control tasks as engaging identical processes and combined their data. It'd be helpful to know if this has ever been done in previously published studies and to include more consideration of limitations or potential concerns about doing this.**

Certainly, Schuch andcoalleagues<sup>31</sup> implemented stroop-face and flanker-number tasks in combination to assess the influence of mood states on cognitive control. In line with previous studies<sup>31,52</sup> our results suggest not only that negative-mood may trigger an increase in conflict adaptation but also that it represents a general phenomenon that occurs independently of the particular interference paradigm involved. In addition, there were several considerations that motivated our choice to use two different tasks, and our results actually confirmed that this was a further strength (e.g. allowing us to demonstrate the replicability and generalization of effects across tasks). See also our detailed response to Reviewer 1 on a similar point (Cf., question7). In any case, we now note this issue in the new "Limitations" section (Cf. main manuscript. Page 34).

**36. It could be clearer in the main text whether the CAPS were estimated by combining data across all periods (rest/movie/task) or only from rest.**

This is a strength of the CAPs approach: The CAPs estimation is agnostic regarding the experimental manipulation. Namely, the CAPs generation included the whole dataset, without any context (i.e., neutral, negative) nor condition (i.e., rest, movie, task) labeling. However, the expression of each CAP can then be quantified (with different indices) for each period separately. We clarified this point in the new "CAPs generation" section (Cf. "Materials and Methods. Stage 1: Dynamic functional connectivity analysis (CAPs identification)". Page 10). Thank you for allowing us to strengthen this part of our study.

**37. They often talk about differences in network relationships across tasks that were only present in negative or neutral contexts. But were those formally detected as interaction**

**Commented [pv21]:** Yes, let's also talk about this  
We might argue that this point is valid from a logical mechanistic perspective of the brain producing behavior – but this is not necessarily relevant to our analysis. Here the idea was to capture changes in how the brain activity is differently engaged by cog conflict depending on (affective) state in relation to the change observed in the behavior, which is composite product of multiple processes and networks, so we use RT as an index of the impact on performance -- best estimated by the interference in RTs... in other word, we focus ton brain to brain effects, not just brain to behavior.  
This could be easier than new indirect analysis ??

**effects? This isn't explicit in the main text. In general, what their statements mean in terms of main effects and interactions in the models could be clearer, since it's stated somewhat informally in the text, and associated results tables are only in supplemental materials.**

We agree with the reviewer and have accordingly revised the main manuscript, changing the term "interaction" in the sections related to the regression and mediation analyses to avoid confusion between the "associations" between brain networks and behavioral scores and the statistical "interaction" computed in the preliminary ANOVA analyses (Cf., "Results. Behavioral performance and affective modulation of cognitive control tasks. Page 15). We thank him/her for pointing out this issue.

**38. Typo – one of their listed “ROPE” effects says “0.” (presumably missing a 0).**

This typo was fixed in the new version of the manuscript (Cf. main manuscript . Page 24). Thanks for pointing it out.

**39. Their descriptions of network relationships were almost always stated non-directionally. It would be helpful for interpretation if they were clearer in their descriptions whether relationships between networks were positive or negative.**

Cf. Response to question 20.

**40. Since MOVIE2 comes between REST1 and the task, it seems reasonable to expect that MOVIE2 responses would moderate or mediate the effects of REST1 on the task neural/behavioral measures. Was this examined at all?**

We thank the reviewer for this insightful question. We did not examine how brain CAPs at "MOVIE2" mediate the effects of "REST1" on "TASK" neural/behavioral measures. This analytical approach was not pursued because the primary aim of this paper was to inspect the differential effects of emotion aftermaths (REST1) versus emotion elicitation (MOVIE2) on cognitive control performance.

The differential patterns observed between these two aims—showing distinct connectivity profiles and network dynamics—suggest that emotion aftermaths and emotion elicitation operate through separate mechanisms rather than sequential mediation pathways. However, we acknowledge that future investigations examining the potential mediating role of MOVIE2 in the REST1-TASK relationship could provide valuable insights into the temporal cascade of emotional influences on cognitive control.

**41. In their discussion, they refer to constructs such as self-regulation and cognitive control (which is often associated with voluntary emotion regulation), which sound overlapping. They also refer to salience vs. "affective meaning". It would be helpful if they better clarified what they mean by these abstract constructs and how they're distinguished.**

In the present work, "self-regulation" refers to spontaneous homeostatic regulation processes that allow the brain to return to "normal" neutral states following transient situational challenges and acute stress responses. These self-regulatory processes may not be entirely under voluntary control (in fact, we did not give any regulation instruction to participants), and their efficiency may fluctuate over the lifespan<sup>53</sup> and with pathological

alterations of mood. In this sense, previous works have shown more frequent occurrences of default-mode co-activation patterns (i.e., CAPs) in borderline patients during depression, compared to other mood states and compared to controls<sup>7</sup>. On the other hand, "cognitive control" refers to situations where people prioritize specific thoughts and actions by enhancing the processing of relevant information and suppressing the processing of irrelevant information<sup>54</sup>. This is usually associated with more voluntary / goal strategies. In response to this pertinent suggestion, these terms have been defined more explicitly in the manuscript, and we now report additional data concerning these conditions. Cf. "Introduction". Page 3. Also, Cf. "Discussion. The impact of negative emotions on cognitive control is linked to their aftermath on intrinsic brain network dynamics". Page 31.

**42. They state: "We surmise that, in post-emotional resting state, the SN-SMN system might encode interoceptive signals and bodily feelings through an interaction with self-referential representations elaborated in DMN areas that together contribute to subjective emotional experience." This suggests interaction between networks during their 1st resting state scan that they could very easily test in their data.**

This is a rightful suggestion. Accordingly, we assessed the association between DMN<sub>CAP</sub> and SN-SMN<sub>CAP</sub>. Our analysis revealed stronger associations in the "REST1" conditions, compared to the "MOVE2" and "TASK" (see results below). Furthermore, this DMN<sub>CAP</sub> - SN-SMN<sub>CAP</sub> association was negative in the "REST1<sub>NEGATIVE</sub>" condition, and positive in the "REST1<sub>NEUTRAL</sub>" condition. The results concerning the "REST1" conditions are now reported in the manuscript.

Parameters		rho	CI_low	CI_high	PD (%)	in ROPE (%)
DMN <sub>NEUTRAL REST1</sub>	SN-SMN <sub>NEUTRAL REST1</sub>	0,75	0,52	0,89	100	0,00
DMN <sub>NEUTRAL MOVIE2</sub>	SN-SMN <sub>NEUTRAL MOVIE2</sub>	0,09	-0,28	0,42	70	0,36
DMN <sub>NEUTRAL TASK</sub>	SN-SMN <sub>NEUTRAL TASK</sub>	0,57	0,28	0,78	100	0,00
DMN <sub>NEGATIVE REST1</sub>	SN-SMN <sub>NEGATIVE REST1</sub>	-0,25	-0,57	0,10	90	0,19
DMN <sub>NEGATIVE MOVIE2</sub>	SN-SMN <sub>NEGATIVE MOVIE2</sub>	-0,16	-0,50	0,19	81	0,29
DMN <sub>NEGATIVE TASK</sub>	SN-SMN <sub>NEGATIVE TASK</sub>	0,26	-0,05	0,60	92	0,15

**43. They hypothesize mechanisms associated with sustained arousal. Do they have any peripheral physiological data during scanning to test this suggestion?**

Our paradigm design initially included pupil size and respiration recordings as proxies of affective arousal. However, multiple technical issues prevent us from using physiological data from more than half of the participants, so we could not include these data. To acknowledge this issue, we included this limitation in the "limitations" section.

**REVIEWER #3:**

This study investigated neural carry-over from an emotion induction task to a cognitive control task. It used innovative dynamic connectivity methods to identify relationships between neural patterns elicited by negative films which were related to FPN engagement during conflict resolution. These relationships were not observed during/after a neutral film. The strengths of the investigation include a well-piloted task, a commitment to making the data available after publication, and the consideration of dynamic networks (vs a traditional voxelwise linear model). However, there were several limitations as well, which diminished the potential impact of the paper on the field. Ultimately, I found the behavioral effects of the task to be better specified than the purported neural mechanisms, and the dynamic connectivity measure did not allow for precise investigation of specific behavioral effects. Furthermore, the mediation analysis using behavior as a mediator were puzzling to me.

Thank you for your careful evaluation of our paper. We were pleased that the reviewer found interest in our paradigm, despite a few concerns. Below, we describe changes in our manuscript that should help clarify these concerns.

**45. The behavioral effects reported are quite interesting – the fact that the emotion manipulation exaggerated both the main effect of conflict and the conflict sequence effect is a great foundation. However, the neural investigation is not able to identify which regions or dynamic functional networks represent the impact of negative emotion on conflict or conflict adaptation, merely performance of the entire tasks (for example, the main effect of RT for the negative vs neutral conditions).**

We are grateful for this positive appreciation of our experimental manipulation and the findings. Regarding the neural correlates of affective priming on cognitive control, the results reported here provided two different brain accounts of the relationship between negative emotions and conflict adaptation. First, our GLM-based brain activation analysis revealed greater BOLD activation in response to attentional interference ( $I > C$  trials) preceded by negative emotions, compared to brain activity underlying  $I > C$  trials preceded by neutral emotions. The areas implicated included the posterior parietal gyri, the dorsolateral prefrontal cortex, and extrastriate visual areas bilaterally (Cf. "Standard GLM analysis of fMRI during cognitive control tasks" section. Page 17). This is fully consistent with a modulation of the recruitment of attentional control networks subsequent to negative emotions.

Second, we specifically investigated which brain networks among those influenced by emotion during movie watching influenced changes in both brain and behavioral performance via our mediation analysis. Remarkably, our main results highlight greater temporal variability in the  $|FPN_{CAP}|$  observed during cognitive control in the negative context than during cognitive control in the neutral context. Furthermore, there was a consistent association between the brain  $FPN_{CAP}$  and the behavioral reaction time index (RT). The magnitude of this brain-behavior association increased in the negative

Commented [pv22]: And DMN ?

Commented [JG23R22]: DMN is key at REST1

context, where the participants exhibited lower cognitive performance (Cf. Direct effects between  $RT_{TASK} \sim FPN_{TASK}$  in the negative context [ $\beta=59$ , Table S14, Figure 5] vs direct effect effects  $RT_{TASK} \sim FPN_{TASK}$  in the neutral context [ $\beta=30$ , Table S13]). Moreover, we show that these changes in brain activity during task performance are predicted by changes in specific brain networks during movie watching period (FPN and SN).

Altogether, these data provide a detailed account of how brain systems engaged during cognitive control are dynamically influenced by emotional state. In our view, this is a major contribution of the present study: By capturing the temporal fluctuations of the functional brain (i.e., CAPs), we were able to provide novel insights into the neural substrates of the interaction between emotions and subsequent cognitive control. [We have rephrased several sentences in our manuscript to further underscore these implications.]

Commented [pv24]: Hope we did ☺

**46. Furthermore, I was unclear on how the authors were conceptualizing behavioral performance as it relates to the model. At several points in the paper, they indicate that they are interested in how negative emotion impacts cognitive control. However, when they model behavior, they include it as a mediator, indicating that they see negative emotion as causing changes in behavioral performance, which then recruit the FPN network changes. Furthermore, the discussion does not directly address the FPN is engaged more during the negative condition, but that's associated with LOWER performance, not better control.**

The reviewer rightfully highlights these points that may have been insufficiently discussed in our initial manuscript and are now better explained. First, as noted in the previous point, FPN expression was enhanced during cognitive control after negative emotions in parallel to lower performance and longer RTs, which we interpret as greater recruitment due to higher task difficulty. In general, there is no general rule relating fMRI activation levels with task performance, and such relationships might differ between regions (e.g., cognitive vs sensory or motor) and/or tasks (e.g., perception vs memory). Hence, there is no reason to assume that greater FPN would be a marker of better control – if anything, it may instead reflect longer processing and prolonged response selection due to difficulty/conflict ambiguity. In contrast, easier and fluent processing in easy tasks tends to produce lower BOLD activity. In fact, our data support this interpretation.

Second, we understand that using RT as a mediator in our BSEM analysis might appear counterintuitive and we apologize if this was not clearly justified in the method. Please recall that our main goal in this study was to understand the functional dynamic interaction between brain networks over time as a function of emotional context, by probing network dynamics at different stages during and after the response to emotional events. Hence, we primarily investigated brain-to-brain effects and tested to what extend they paralleled concomitant differences in behavior (rather than focussing only on a brain-to-behavior relationship). In other words, here we aimed at capturing how the changes in brain systems engaged by cognitive conflict after negative emotions depended on the impact of emotion on brain (and affective) state prior to the task, and how this relation reflected the change observed in the behavior by using RT as a composite marker of this impact (rather than the outcome

measure of emotional effects). We have added a more explicit statement in our method section to clarify this point.

**47. No power analysis was provided, which is unusual. It is unclear whether the authors were well powered enough with a final sample of 24 to conduct the analyses they report.**

Please see our response to Question 3 above.

**48. I was curious whether the magnitude of self-reported negative affect in response to the film moderated the behavioral performance on the tasks. This would be a behavioral carry-over effect beyond the group-level manipulation.**

Two of our results provide evidence on the influence of negative affect on the performance of the behavioral systems. First, the reaction time of the same task shows significant differences between the neutral and negative contexts. To recall, the repeated measurements design enabled us to measure this variable at the subject level. Second, correlation analyses (rho) yielded significant associations between cognitive performance (i.e., reaction time) and scores of positive and negative affect, specially after the affective induction. Cf., Response to question 32. Figure R1.

**49. The interpretation of the DMN at rest is that it is reflecting more intense subjective experience, but this is not tested with the subjective data available, nor is the subjective data used rather than the brain data to test the same hypothesis.**

The link between DMN<sub>CAP</sub> and subjective experiences (e.g., emotions) was previously addressed by Reviewer 1 (Cf. question 24). To recall, new results demonstrating correlations between DMN occurrences and subjective affective scores (measured on PANAS scale) are now included in the latest version of the manuscript (cf. "results" section, Figure 4A, and the "discussion" section, respectively). This finding supports a connection between DMNCAP functional dynamics and affect.

Smaller comments:

**50. The authors should include that their sample was limited to only those identifying as women in their limitations section. Furthermore, the authors should clarify whether they measured sex or gender and be consistent throughout.**

This limitation has been mentioned in the new "Limitations" section. In addition, the expression "gender" has not been used purposefully to avoid uncertainty regarding our biological definition of "women" and we only refer to sex categories here. We thank the reviewer for this cautionary notice.

**51. The authors indicate that the CSE was driven by the iC condition- usually I think of it as more driven by cl vs il. Is this a limitation?**

We defined congruence sequence effect (CSE) as the "influence of the preceding trial on the current one" (Cf., Main manuscript. Page 17). In this way, and critically related to the objectives of our study, we reported significant congruence sequence effects (CSE) in both emotional contexts, which was amplified in the negative context for the two most difficult trials: namely, "iC" and "cl" trials because

Commented [pv25]: Is it true?

Commented [JG26R25]: Partially true. Your are more accurate when mention the shift in the stimulus response.

both require a shift in the stimulus-response link (cf., main manuscript. Figure 1C). We underscore this in the method section.

**53. I recommend moving the regions identified in the CAPs to the main manuscript. In the supplemental material, please specify which table reports congruency effects on page 6.**

Thank you for this suggestion. The new version of the main manuscript includes the tables listing the obtained brain systems (i.e., the four key co-activation patterns). Additionally, table S1 reporting the congruency sequence effects (i.e., CSE) is mentioned in “Results, Behavioral performance and affective modulation of cognitive control tasks” section. Results from further effects (e.g., congruency effects (CE)) are directly described in the same section.

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