

Recalibration of ICN Resting-state Connectivity Following Multiple Sessions of PCC- or Amygdala-Targeted fMRI Neurofeedback for PTSD

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Introduction

Real-time functional magnetic resonance imaging (fMRI) neurofeedback (NFB) is a non-invasive technology that allows the self-regulation of brain states and is an emerging treatment therapy for post-traumatic stress disorder (PTSD). Intrinsic connectivity networks (ICNs) commonly show functional disruptions in PTSD. Namely, the amygdala—a region within the salience network (SN)—and the posterior cingulate cortex (PCC)—a critical hub in the default mode network (DMN)—are often hyperactive during trauma provocation and have demonstrated disrupted connectivity patterns at rest. Downregulation of these areas via NFB has been correlated with symptom reductions along with recalibration of altered brain networks. However, previous studies have mainly focused on examining changes in functional connectivity from task-based data during single-session fMRI NFB studies and have not investigated changes in resting-state connectivity longitudinally. Here, we aimed to examine changes in resting-state connectivity across multiple sessions of PCC- or amygdala-targeted fMRI NFB in individuals with PTSD.

Methods

We conducted an independent components analysis (ICA) to investigate changes in resting-state connectivity across three days between PTSD participants (N = 30) who were trained to downregulate activity in either the PCC (n = 15) or amygdala (n = 15) using an fMRI NFB trauma provocation paradigm. Whole-brain connectivity results were corrected for multiple comparisons using the false discovery rate (FDR), with significance established at $p < .05$ following FDR adjustment. We also examined participants' ability to regulate the NFB signal and evaluated PTSD symptom severity using the PTSD Checklist for DSM-5 (PCL-5) over the course of treatment. We plan to conduct analyses examining correlations between resting-state connectivity and PTSD symptom changes throughout the NFB intervention.

Results

Both groups were able to successfully regulate the NFB signal on all three days and showed significant improvements in PTSD symptom severity over the course of NFB. The PCC group displayed decreased resting-state connectivity between the SN and the left frontal pole, bilateral superior frontal gyrus, anterior cingulate gyrus, and precuneus from pre- to post-NFB intervention, however, there were no significant between-group differences in SN connectivity. The amygdala group as compared to the PCC group showed increases in resting-state connectivity between the DMN and the left medial temporal gyrus and left angular gyrus at post- compared to pre-intervention. Conversely, the PCC group as compared to the amygdala group showed increases in resting-state connectivity between the DMN and the right pre- and postcentral gyrus and the right superior parietal lobule at post- compared to pre-intervention.

Conclusions

These results indicate that multiple sessions of amygdala- or PCC-targeted NFB can recalibrate resting state DMN and SN connectivity with multiple brain areas implicated with PTSD. Here, resting-state connectivity changes in different ICNs suggest there are distinct neural mechanisms underlying PCC- versus amygdala-NFB, and provide evidence of broader network reorganization extending beyond the NFB training paradigm itself. The moderate sample size ($n = 15/\text{group}$) should be taken into account when interpreting results. Future research should evaluate resting-state connectivity at follow-up timepoints to assess whether these changes persist on a longer timescale. Collectively, these findings provide evidence that selectively targeting the PCC and amygdala during NFB is associated with unique neural connectivity patterns in individuals with PTSD.