depression symptoms are associated with motor network abnormalities for 8,940 individuals.
Results: Developmental motor delays were associated with current depression diagnoses, current symptoms, and a familial risk loading for depression. Current motor abnormality symptoms were also each associated with all depression metrics, including current diagnoses, current symptoms, and familial risk loading. Motor network corticostriatal connectivity was related to current depression symptom levels.
Conclusions: Motor development and symptoms are critically related to depression symptoms, diagnoses, and familial risk loading. Motor function may reflect core biological vulnerability to depression as evidenced by familial risk and motor network connectivity. Finally, individuals with familial risk for psychosis and depression showed the greatest motor abnormalities.
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Keywords: Depression, Motor Symptoms, Resting State Functional Connectivity, Adolescent Brain Cognitive Development (ABCD) Study, Development

Development of a Novel Multimodal Social Reward Task for Neurobiological Adult and Late-life Depression Studies

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Background: Exposure to social rewards protects against depression. Positive social interactions elicit activation in reward regions, which are hypoactive in depression especially in late life. Psychotherapy can increase engagement in social interactions and in turn increase activity in the reward system. We developed a paradigm to assess changes in social reward processing using EEG and MRI during psychotherapy for late life depression.
Methods: This personalized task, modeled after the Monetary Incentive Delay task, measures social reward anticipation and receipt. In Study 1, we administered the task three times over 9 weeks to 25 older adults (75% females; mean age = 72.23): 13 healthy controls and 12 depressed older adults receiving 9 weeks of psychotherapy. In Study 2, 16 participants (10 depressed; 6 healthy control) completed the task during an MRI scan.
Results: Regression model showed that depressed older adults performed slower than controls across task conditions and time points ($F(1,200)=15.33$, $p < 0.001$). Mixed-effects model showed significant improvement in task performance in depressed individuals across treatment ($F(3,77) = 6.32$, $p < .001$). Additionally, improvement in reaction time predicted subsequent reduction in depression severity over the course of psychotherapy ($F(3,66)=13.38$; $p<.001$). Preliminary fMRI results showed increased activation in reward regions in response to social rewards, compared to the non-reward condition.
Conclusions: Our pilot data suggest that task performance changes over time and predicts improvement in depression severity with psychotherapy. If validated, our task can be used as an objective measure of social reward processing in clinical trials.
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Enhancing Trauma-Memories Reconsolidation With One-Time Ketamine Infusion

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Background: NMDA receptor antagonists were found to have a vital role in extinction and reconsolidation processes. During reconsolidation, memories are activated into a labile state and can be stored in an altered form. This idea might have clinical implications in the treatment of PTSD, with current psychotherapies' success rate rounding 50%. Here we used a single subanesthetic intravenous infusion of ketamine (NMDA receptor antagonist; 0.5mg/kg over 40min) to enhance post-retrieval extinction of real traumatic memories.
Methods: 24 PTSD patients were randomized to ketamine or midazolam groups. The procedure included a one-time 40min infusion following reactivation of the traumatic memory, while in the MR scanner. This was followed by 5 prolonged exposure sessions. Brain activation during memory retrieval was assessed before and after treatment, as well as 30 and 90-days follow-up.
Results: Ketamine caused a significant decline in the amygdala and hippocampus reactivation to the traumatic event at the end of treatment, compared with the midazolam group (mean group difference amygdala=-31.78, SD=10.54, 95%CI = [-52.57, -11.48]; hippocampus = -20.12, SD=10.71, 95%CI = [-41.73, -0.07]). The ketamine group presented a decline in connectivity between amygdala and hippocampus, and hippocampus and vmPFC, compared to the midazolam group. Skin conductance response to the traumatic event was lower in the ketamine group.
Conclusions: These results suggest that one-time ketamine infusion, received after retrieval of the traumatic memory, enhances the reconsolidation process, and promotes post-retrieval extinction in humans. These findings are also presenting a proof of concept for the ability to use ketamine to augment short-term prolonged exposure for PTSD.
Supported By: NARSAD
Keywords: PTSD, Ketamine, Reconsolidation

Inflammatory Signaling and Corticostriatal Functional Connectivity to Anticipated Valence and Salience of Reward and Threat Stimuli: An Investigation in Depressed vs. Non-Depressed Young Adults

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