209. Abnormal Recruitment of Cognitive Control Processes in Intermittent Explosive Disorder During an Aggressive Interaction

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Background: Cognitive control processes support the flexible pursuit of goals through mechanisms such as performance monitoring. In previous research using high density EEG we have shown that psychiatrically healthy subjects engage cognitive control resources during a simulated aggressive interaction. We seek to expand on this research by investigating whether aggressive individuals diagnosed with intermittent explosive disorder (IED) show abnormal psychophysiological markers of cognitive control during this task.
Methods: 25 healthy men and women and 11 individuals with current IED completed a modification of a standardized laboratory paradigm for aggressive behavior (the Taylor Reaction-Time Task) while EEG was recorded on 128 scalp channels. Event-related potentials were averaged based on participants' responses to a (fictitious) opponent's provocation: escalation, matching, de-escalation, and a control condition. Error-related negativity (ERN), a psychophysiological index of performance monitoring, was measured at midline scalp electrodes (Afz, Fpz, Fz, Fcz, Cz, Cpz, and Pz). Neural generators of the ERN were examined separately in healthy and aggressive individuals using LORETA.
Results: A mixed model ANOVA showed a trend-level (p=.078) group x response interactive effect on the ERN amplitude. In healthy participants, ERN was modulated by response and was largest when participants escalated compared to other responses (ps<.05). Compared to healthy participants, IED participants showed increased ERN when de-escalating (p=.02).
Conclusions: Behavioral regulation in the face of provocation depends on cognitive control processes; however, preliminary data suggests these processes are disrupted in individuals with chronic, problematic aggression.

Keywords: Aggression, Cognitive Control, Electroencephalogram, Performance Monitoring, Intermittent Explosive Disorder

210. HPA-Axis Stress Reactivity Relates to the Degree of Prefrontal, Limbic, and Striatal Activation During Emotional Processing in Unipolar and Bipolar Depression

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Background: Depression is associated with dysregulated HPA-axis function and disrupted emotion processing. The neural networks involved in attenuation of HPA-axis reactivity share function with the circuitry involved in perception and modulation of emotion, however, direct links between these systems are understudied. The present study investigated whether cortisol activity prior to an fMRI scan was related to neural processing of emotional information in depressed subjects.
Methods: Forty-one adults (M = 40.33, SD = 15.57) with major depression (MDD; n = 29) or bipolar disorder (BP; n = 12) and 23 healthy control comparisons provided salivary cortisol samples prior to completing a facial emotion perception test during 3-tesla fMRI.
Results: Pre-scan cortisol was associated with engagement of the dorsal anterior cingulate (dACC), inferior parietal lobule, insula, putamen, precuneus, middle, frontal and postcentral gyri, posterior cingulate, and inferior temporal gyrus during emotion processing. Depression moderated this effect; in depressed subjects pre-scan cortisol was associated with attenuated activation of the insula, postcentral gyrus, precuneus, and putamen for fearful faces and the medial frontal gyrus for angry faces. Hypo-activity among MDD/BP participants was also observed for facial recognition in the dACC, putamen, middle temporal gyrus, precuneus, and caudate.
Conclusions: Across all subjects, cortisol increased activation in several regions involved in the perception and control of emotion. However, cortisol responsivity was associated with deactivation of several of these regions in depression, suggesting that HPA-axis activity in depression may interfere with the potentially adaptive recruitment of regions supporting emotion processing.

Keywords: cortisol, emotion processing, fMRI, depression, bipolar
Supported by: NIMH F31MH108258-01 (ATP); National Alliance for Research On Schizophrenia and Depression (NARSAD, SAL)

211. Neuropsychiatric Presentation of a Paraneoplastic Syndrome

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Background: We report a case of P/Q- Type Calcium channel antibody and N-Type Calcium channel antibody Paraneoplastic Encephalitis, leading to acute onset Fronto-Temporal Dementia in a 54-year-old man who had rapid response to plasmapheresis. Typically autoantibodies to the N and P/Q type calcium channel present as Lambert-Eaton Myasthenia Syndrome. Our case highlights a previously unreported manifestation of this paraneoplastic syndrome.
Methods: We used full neuropsychiatric testing, MRI, PET-CT scan, lumbar puncture, and autoantibody evaluation to diagnose his condition. We are treating his autoimmune process with plasma exchange.
Results: Due to the acute/sudden onset of the disease and the history of multiple autoimmune disorders we decided to further investigate possible autoimmune etiology this FTD. This patient meets criteria for a diagnosis of FTD, yet with the positive paraneoplastic panel, we hypothesized that the etiology of his disease is autoimmune and that his condition would improve with treatment for autoimmune neurologic disorders. He has no signs/symptoms of Lambert-Eaton. His behavior and cognition have improving by removing the autoantibodies with plasma exchange.