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Disconnect Between Hypothalamic-Pituitary-Adrenal Activity and Amygdala Activation Using Novel Neuroimaging Fear Paradigm in Youth With Avoidant/Restrictive Food Intake Disorder

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Background: Avoidant/restrictive food intake disorder (ARFID) is characterized by restrictive eating patterns without shape or weight concerns, leading to nutritional deficits and psychosocial impairment. Three distinct phenotypes have been described that can co-occur: 1) sensory sensitivity, 2) lack of interest in eating or food, and 3) food avoidance due to fear of aversive consequences. Hyperactivation of a "defensive motive system", characterized by a neurocircuit activation of the amygdala and the hypothalamic-pituitary-adrenal (HPA) axis, could mediate fear/threat pathways in ARFID. We hypothesized that youth with ARFID would show amygdala hyperactivation in response to an ARFID-specific fear paradigm, and greater amygdala activation would be associated with higher levels of cortisol. Method76 youth with ARFID assessed using the Pica, ARFID and Rumination Disorder Interview and 27 healthy controls (HC) underwent functional MRI scanning (3T Siemens) during a novel visual fear task. Subjects viewed a series of developmentally appropriate images: ARFID-specific fear (e. g., a person vomiting or choking) and neutral (e. g., household items, geometric shapes). Serum cortisol was measured prior to the MRI in the late morning after a 2-hour fast. Amygdala activation was compared between ARFID and HC for the contrast ARFID-specific fear vs. neutral images using SPM12. For this contrast, beta values were extracted and correlated with fasting cortisol levels using Pearson's correlation coefficient. **Results:** Groups did not differ for age, sex, or BMI z-score (ARFID mean (SD) age 16.5 (3.9) years, HC 17.8 (4. 0) years (p=0.14); ARFID 50% male, HC 42% male (p=0.66); ARFID BMI z-score -0.5 (1.7), HC 0. 0 (0.8) (p=0.15). In response to ARFID-specific fear vs. neutral images, both HC and ARFID exhibited increased activation in the bilateral amygdala (p<0. 05), however there were no significant differences in amygdala activation between ARFID vs. HC. Cortisol levels did not differ between groups (p=0.83). In the HC group, there was a positive correlation between amygdala activation in response to the contrast ARFID-specific fear vs. neutral images and cortisol levels (r=0.54, p=0. 02), which was absent in ARFID (r=-0. 02, p=0.88) (between-group difference p=0. 01). **Conclusion:** Using a novel fMRI paradigm, our results show that images related to ARFID-specific fears elicit robust increases in amygdala activation across ARFID and HC subjects, but no between-group differences. While higher amygdala activation was associated with higher cortisol levels in HC, this relationship was absent in ARFID, suggesting a disconnect of amygdala response and basal HPA activation to fear inducing stimuli. Future research will be important to improve our understanding of endocrine modulation of the fear response and relationship to restrictive eating in ARFID.

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