**BACKGROUND**

Fig. 1: The hypothalamic-pituitary-adrenal (HPA) axis drives the stress response by releasing cortisol; if chronic, it affects amygdala and hippocampal volume. 

**METHODS**

**Participants:** N = 59, Ages 25-65 (mean = 40.4, S.D = 13.2), 66% Female, 86% White.

1. **Stress and Adversity Inventory:** The lifetime stressor severity index was used, combining severity and frequency of all stressors from across the lifespan.

2. **Structural MRI scan:** Amygdala, hippocampal, and intracranial volumes (ICV; total space within the skull) extracted with Freesurfer Software.

3. **Trier Social Stress Test (TSST):** Participants completed a public speaking and mental math task with salivary cortisol collected at 10-min intervals throughout reactivity and recovery periods.

**RESULTS**

**CONCLUSIONS**

- Our results supported hypotheses (excluding reactivity predictions), but the associations between brain regions of interest with lifetime and acute stress appear to be due to overall ICV differences.

- Interestingly, the ICV results suggest that a larger brain capacity within the skull is beneficial and may have a protective effect in response to stress (or alternatively that more adaptive stress responses may contribute to growth of a larger brain capacity).

- Future adequately powered studies are needed to determine the relationships between lifetime stress, amygdala and hippocampal volume, and the acute stress response.

Fortunately, we have now completed data on a larger sample of participants to do just that!

**REFERENCES**


**FUNDING**

This study was supported by funding from the National Institute of Mental Health (R01 MH084545 to RJD and SMS) in part by a core grant to the Waisman Center from the National Institute of Child Health and Human Development (P50 HD043453).