Homeostatic and mesolimbic network functional connectivity during food reward processing in response to psychosocial stress

Hyeonmin Ahn[1,2*], Julia Hall[1], Jill M. Goldstein[1,2,6,7], Daniel G. Dillon[2,4], Diego A. Pizzagalli[2,4,5], and Laura M. Holsen[1,2,3]

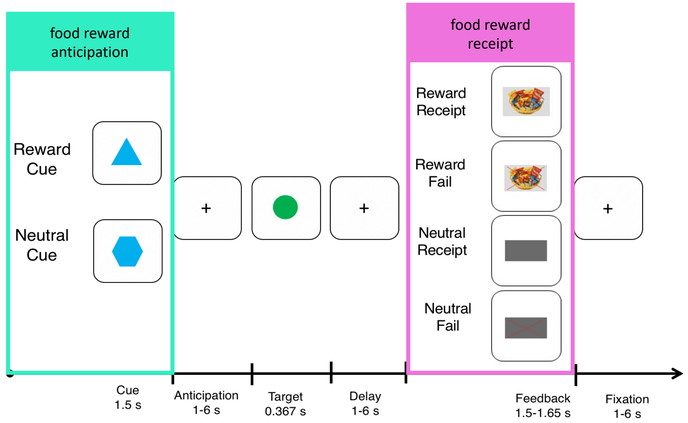
[1]Division of Women's Health, Department of Medicine, Brigham and Women's Hospital [2]Harvard Medical School [3]Department of Psychiatry, Brigham and Women's Hospital
[4]Center for Depression, Anxiety and Stress Research, McLean Hospital [5]McLean Imaging Center, McLean Hospital
[6]Departments of Psychiatry, Massachusetts General Hospital [7]Innovation Center on Sex Differences in Medicine, Massachusetts General Hospital

BACKGROUND

- Stress-related overeating has been suggested as a mechanism potentiating weight gain[1].
- Psychosocial stress impacts behavioral aspects of food reward[2,3], yet the coordinated activity of neural circuits which orchestrate these behaviors is not well understood.
- We investigated functional connectivity (FC) of homeostatic and mesolimbic reward pathways during food reward processing under psychosocial stress.

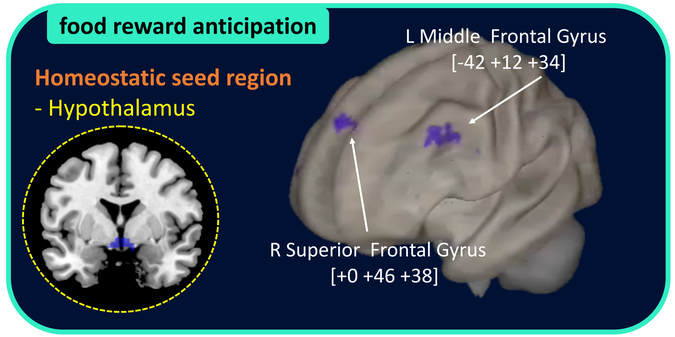
Methods

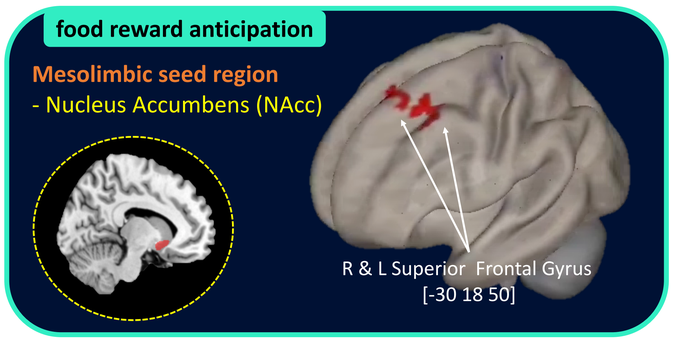
- 39 healthy adults completed a study involving exposure to acute psychosocial stress, followed by functional MRI scanning during a food incentive delay task.
- The Maastricht Acute Stress Test (MAST) combines hand immersion trials (involving ice cold water immersion) and mental arithmetic trials to induce stress physically and psychologically.
- fMRI data were analyzed using a generalized psychophysiological interaction (gPPI) analysis to investigate FC between homeostatic (hypothalamus) and mesolimbic regions [amygdala, nucleus accumbens (NAcc)] and whole brain.

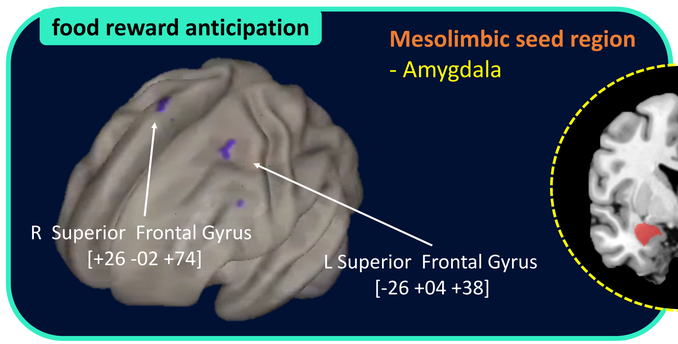


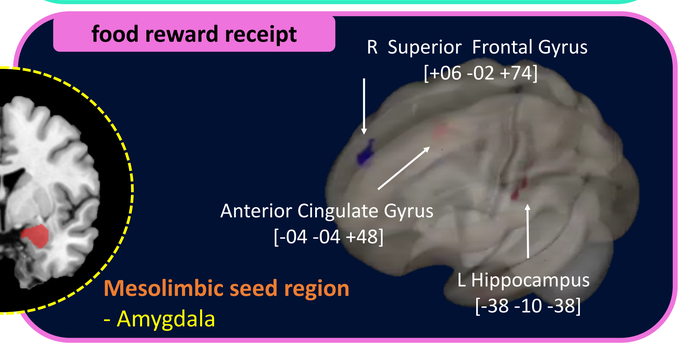
Results

- During food reward anticipation, we observed stress-induced decreases in hypothalamus FC to superior/middle frontal gyrus (p=0.01), in amygdala FC to superior frontal gyrus (p=0.04), and in NAcc FC to precentral gyrus (p=0.02), with increases in amygdala FC to precuneus (p<0.01) and NAcc FC to superior/middle frontal gyrus (p<0.01).
- During food reward receipt, amygdala showed increased FC to orbitofrontal cortex (p=0.02) and hippocampus (p=0.02), and decreased FC to superior frontal gyrus (p=0.05).









Conclusion

- We found that anticipation of food reward was associated with alterations in the coordinated activity between different networks (homeostatic, emotion, reward) and executive control regions under psychosocial stress.
- Our results contribute to the understanding of neural circuit synchrony involved in stress-induced food reward and may inform mechanisms underlying changes in eating behavior under stress. These findings may also contribute to the development of novel treatments for stress-related eating disorders.

Reference

1. Sinha, R., & Jastreboff, A. M. (2013). Stress as a common risk factor for obesity and addiction. *Biological psychiatry*, *73*(9), 827-835.
2. Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. Physiology & behavior, 91(4), 449-458.
3. Morris, M. J., Beilharz, J. E., Maniam, J., Reichelt, A. C., & Westbrook, R. F. (2015). Why is obesity such a problem in the 21st century? The intersection of palatable food, cues and reward pathways, stress, and cognition. Neuroscience & Biobehavioral Reviews, 58, 36-45.

This work was funded by the NIH (R01-DK104772; L.M. Holsen, PI)