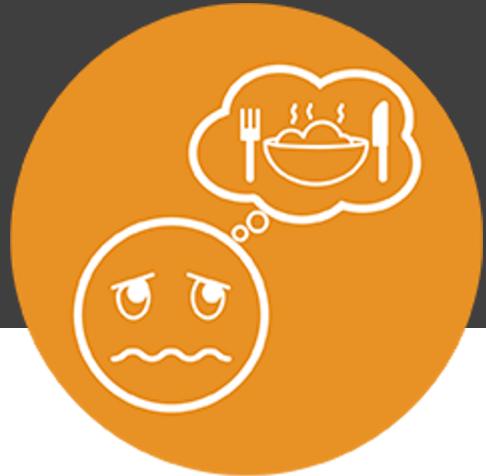


Stress-induced alterations in HPA-axis reactivity and mesolimbic reward activation in individuals with emotional eating

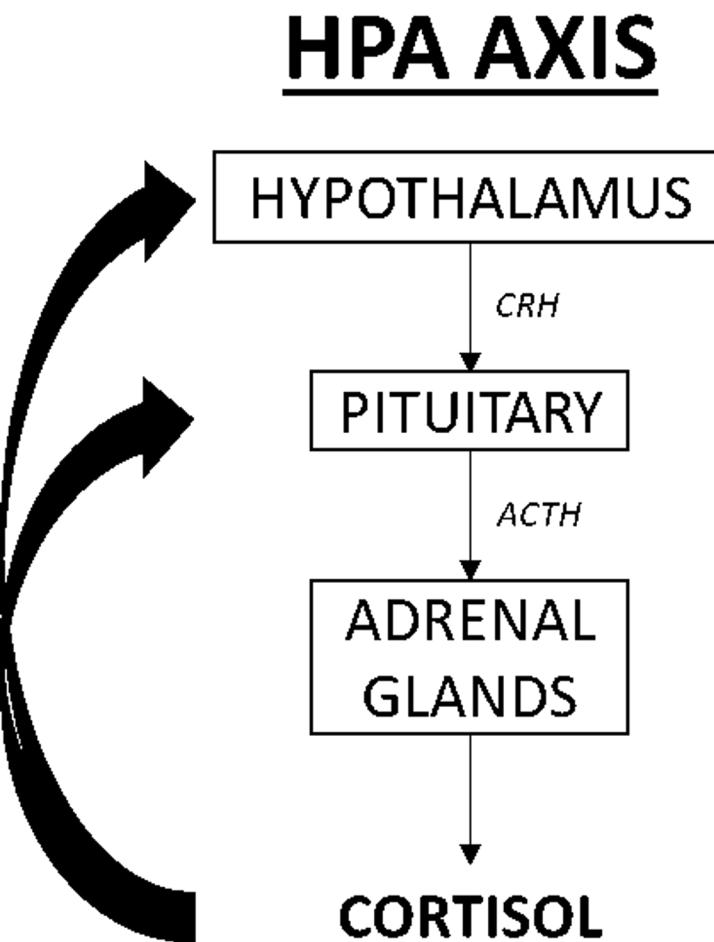
Rose Chang
09/10/2020

BACKGROUND (Emotional eating)



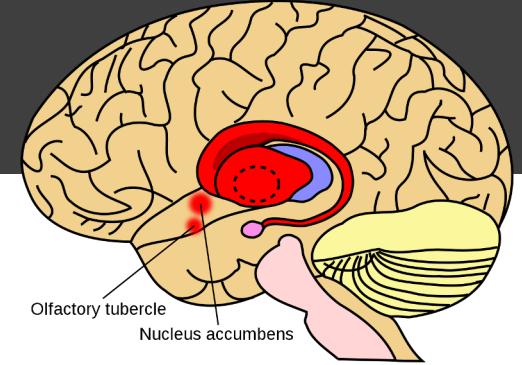
- **Emotional eating:** a tendency to eat in response to negative emotions or emotional distress
- Emotional eating has been linked with obesity (Geliebter & Aversa, 2003) & various subtypes of eating disorders
 - ANBP & BN (Ricca et al., 2012); BED (Pinaquy et al., 2003)
- suggested as an important psychopathological dimension that contributes to overeating (Cornelis et al., 2014)

BACKGROUND (EE & HPA axis)



- **HPA axis** = our central stress response system
- Hyperactivity within the hypothalamic-pituitary-adrenal (HPA) axis has been found in eating disorders & obesity (Gluck et al., 2004; Koo-Loeb et al., 1998)
- Contrasting results in emotional eating
 - heightened cortisol level (Raspopow et al., 2010) vs. no differences (Van Strien et al., 2013)

BACKGROUND (EE & reward system)



- **Hyperactivation theory:** overactivation of food reward circuitry leads to increased risk for overeating and binge eating (Wood et al., 2016; Loxton & Tipman, 2017)
- **Hypoactivation theory:** hypoactivation of food reward circuitry leads to overeating as a compensatory behavior (Bohon et al., 2014)

GOAL/PURPOSE

To examine the relationship between emotional eating status and its associated neuroendocrine and neural alterations under stress



Hypothesis: Emotional eaters (EE) would exhibit hyperactive cortisol reactivity, display aberrant brain activation in response to anticipation and receipt of food reward, and engage in overeating of snacks.

METHOD (participants)

- **28** healthy men and women (14F; 14M) (**13 EE; 15 NE**)
 - tertile split from 40 participants (DEBQ emotional eating subscale)
- exclusion criteria: psychiatric disorders, substance abuse, psychotropic medications, endocrine disorders, diabetes, weight loss medications, MRI contraindications, suicidal ideations, traumatic brain injury, pregnancy, etc.
- mean age = 28.29 yrs, mean BDI = 25.8
- 57.1% Caucasian, 21.4% African American, 17.9% Asian, 3.6% Other
- paid up to \$425 upon completion

METHOD (procedures)

Screening (1.5 hr)

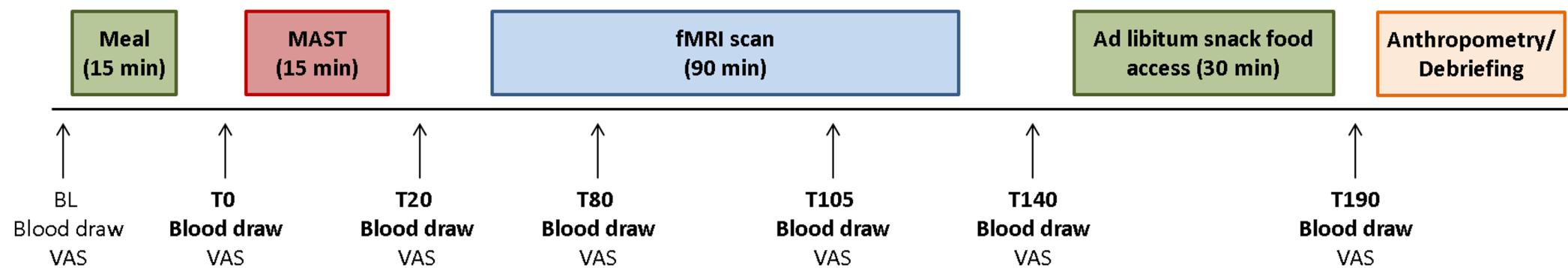
- SCID
- height, weight
- blood draw (for hematocrit level)

Visit 1 (5 hr)

MAST (stress/no stress)

Visit 2 (5 hr)

MAST (stress/no stress)

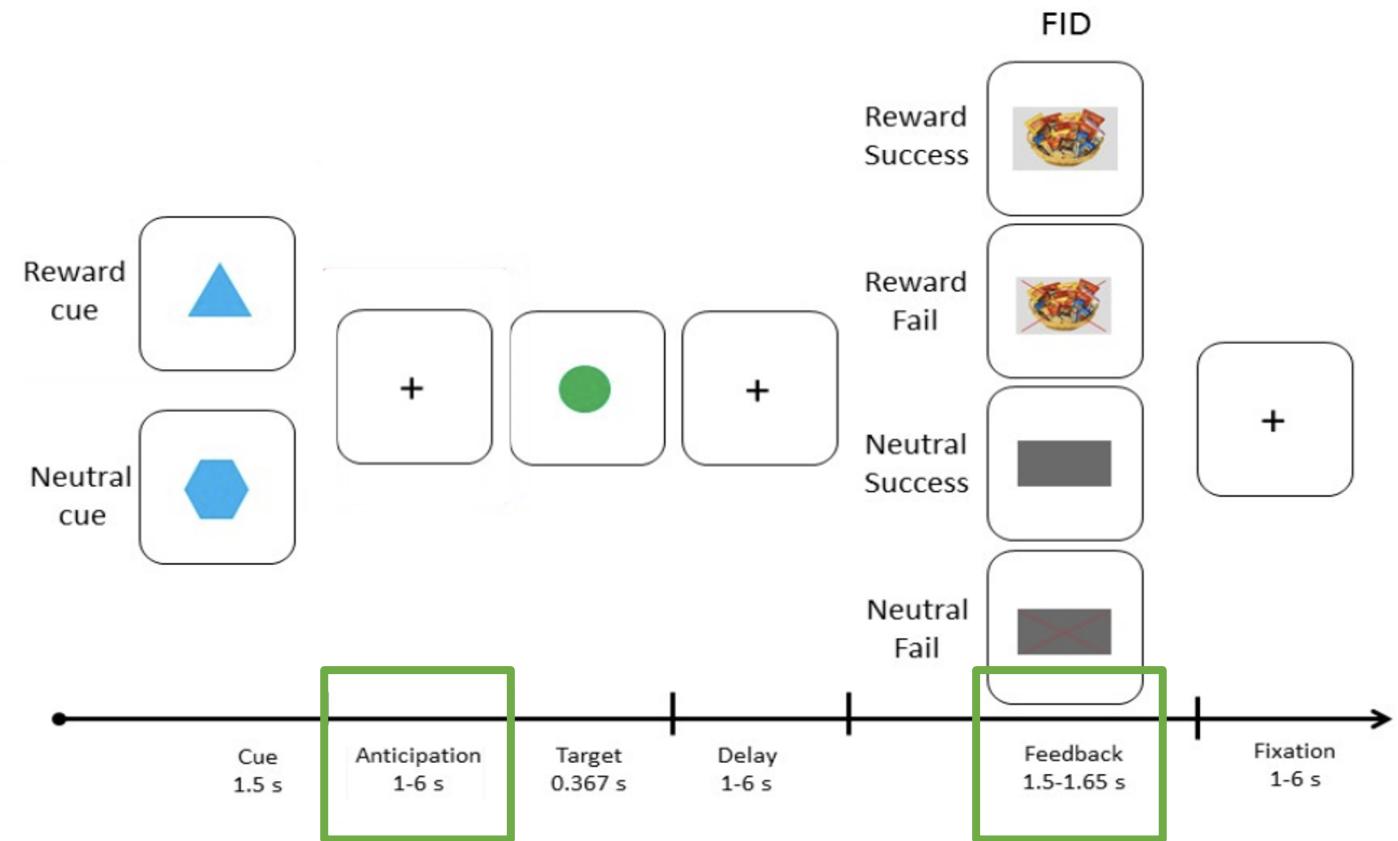


METHOD (measures)

- Emotional eating
 - Dutch Eating Behavior Questionnaire; emotional eating subscale
 - e.g. “Do you have a desire to eat when you are emotionally upset?”
- Snack Intake (ad libitum)
 - total calories; (Fig Newtons, M&M’s, Almonds, muffins, Hershey, etc.)
- Cortisol Level
 - baseline (fasting); Time 0 & Time 20 (prior to and following the MAST)
- Anxiety Level
 - “How nervous do you feel right now?” 0 (not at all) ~ 100 (never been more)

METHOD (fMRI paradigm)

- 3T scanner; 32-channel head coil
- Food Incentive Delay (**FID**) task
 - 3 runs (100 trials; 60 reward cues & 40 neutral cues) each



STATISTICAL ANALYSIS (fMRI data)

- standard preprocessing procedures using SPM12
 - realignment & geometric unwrapping using magnetic fields, motion correction, slice timing correction, EPI coregistration to the T1 image, normalization to the MNI space, smoothing with a 6 mm full-width-half-maximum (FWHM) Gaussian kernel
- focus on the BOLD responses during the anticipation & receipt phases of the FID task
 - anticipation of reward vs. anticipation of neutral
 - receipt of reward success vs. receipt of neutral success
- a priori ROIs (using the REX toolbox): caudate, nucleus accumbens (NAcc), putamen, amygdala, hypothalamus
 - defined using a manually segmented MNI-152 brain & implemented as overlays on the SPM8 canonical brain using PickAtlas toolbox

STATISTICAL ANALYSIS (all further)

- **Demographic data, DEBQ, baseline characteristics** = chi square & independent samples t-tests
- **Cortisol levels & subjective anxiety levels** = 2 (Group: NE/EE) x 2 (Visit: No-stress/Stress) x 2 (Time: T0/T20) repeated measures ANOVA
- **FID beta estimates & snack consumption** = 2 (Group: NE/EE) x 2 (Visit: No-stress/Stress) repeated measures ANOVA
 - beta estimates (degree of functional responses) extracted from REX and exported to SPSS

<RESULTS>

Demographic & Baseline Variables

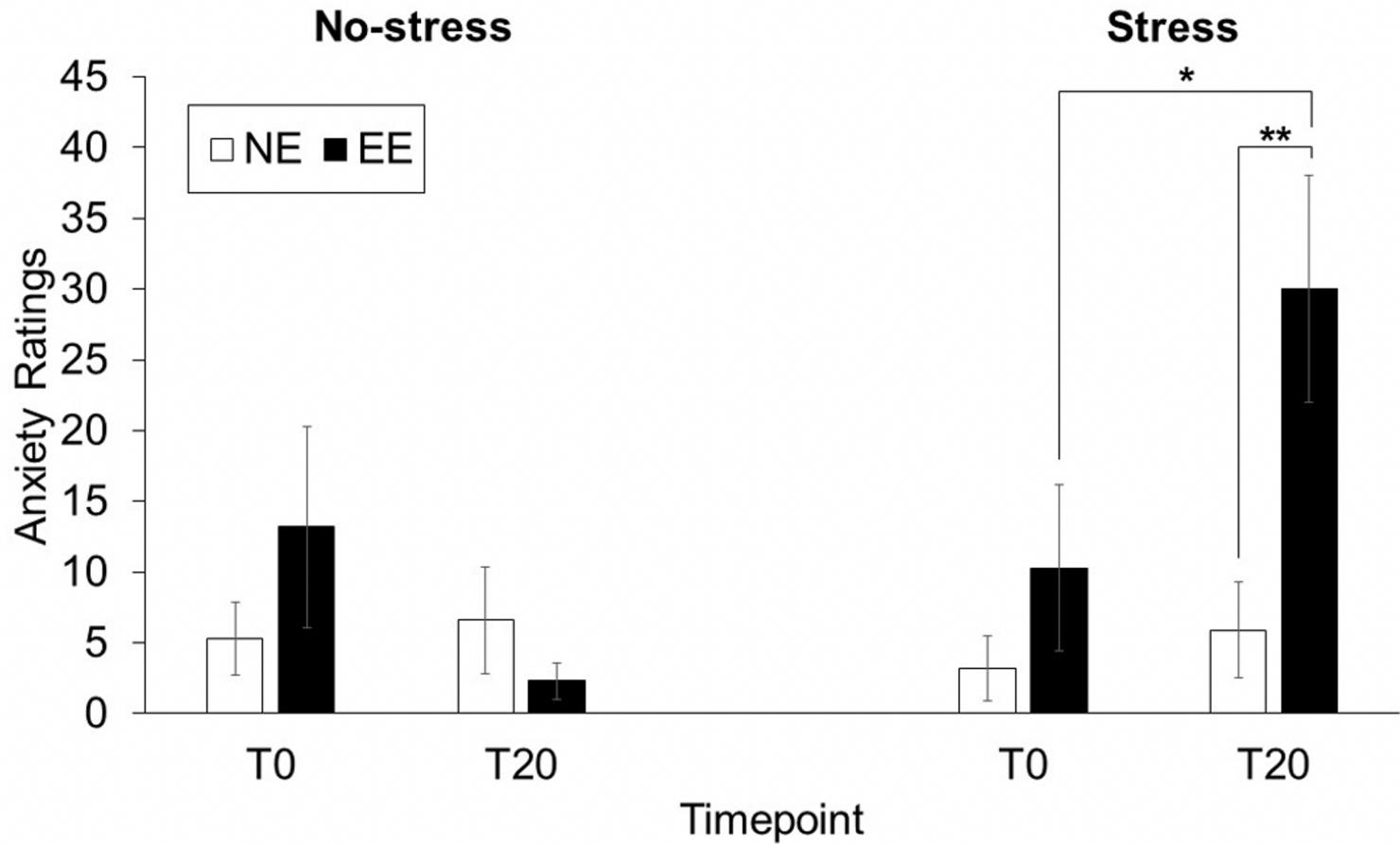
Variable	Group				Between-Group Comparisons
	Emotional Eaters (n=13)		Non-emotional Eaters (n=15)		
	Mean	SD	Mean	SD	
Age (years)	28.6	5.8	28.0	5.4	$t(26)=0.29, p=0.77$
BMI	26.2	3.4	25.5	5.8	$t(26)=0.40, p=0.70$
Emotional Eating (DEBQ)	3.0	0.7	1.3	0.2	$t(26)=9.37, p<0.001$
Baseline hunger (VAS)					
No-stress	55.5	26.1	60.9	26.3	$t(26)=-0.54, p=0.60$
Stress	61.0	26.7	52.3	30.9	$t(26)=0.78, p=0.44$
Breakfast consumed (%)					
No-stress	83.1	20.3	78.9	18.5	$t(26)=0.57, p=0.57$
Stress	83.0	22.0	80.6	21.1	$t(26)=0.29, p=0.77$
	n	%	n	%	
Sex					$\chi^2=1.29, p=0.26$
Female	8	28.6	6	21.4	
Male	5	17.9	9	32.1	
Physical activity (moderate)					$\chi^2=2.93, p=0.57$
>4 times per week	2	7.1	2	7.1	
2-4 times per week	8	28.6	9	32.0	
Once a week	2	7.1	2	7.1	
2-3 times per month	1	3.6	0	0.0	
Rarely or never	0	0.0	2	7.1	
Physical activity (very hard)					$\chi^2=2.99, p=0.56$
>4 times per week	3	10.7	1	3.6	
2-4 times per week	3	10.7	5	17.9	
Once a week	2	7.1	5	17.9	
2-3 times per month	3	10.7	3	10.7	
Rarely or never	2	7.1	1	3.6	

<RESULTS>

Subjective anxiety ratings

* $p<0.05$

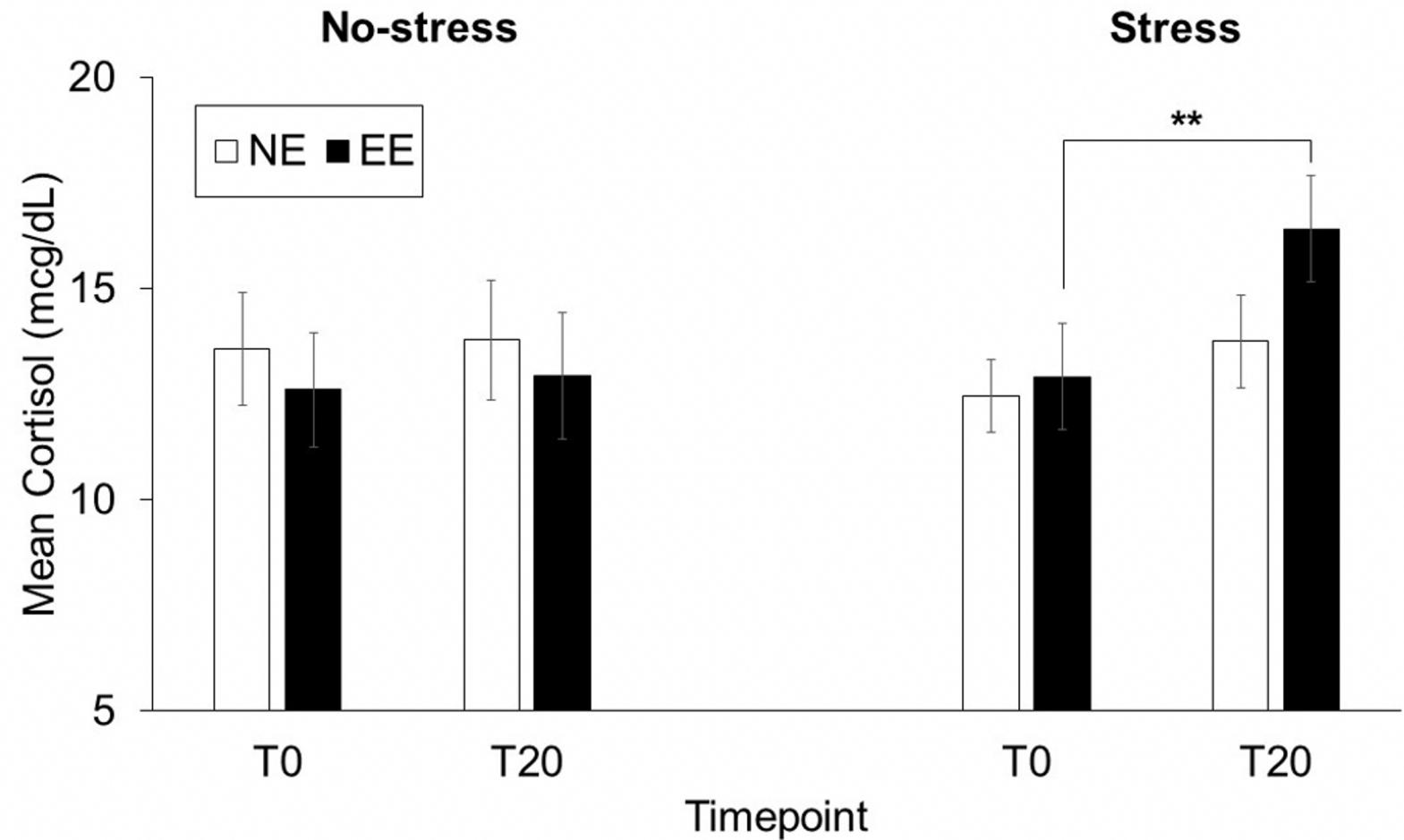
** $p<0.01$



<RESULTS>

Cortisol levels

** $p<0.01$

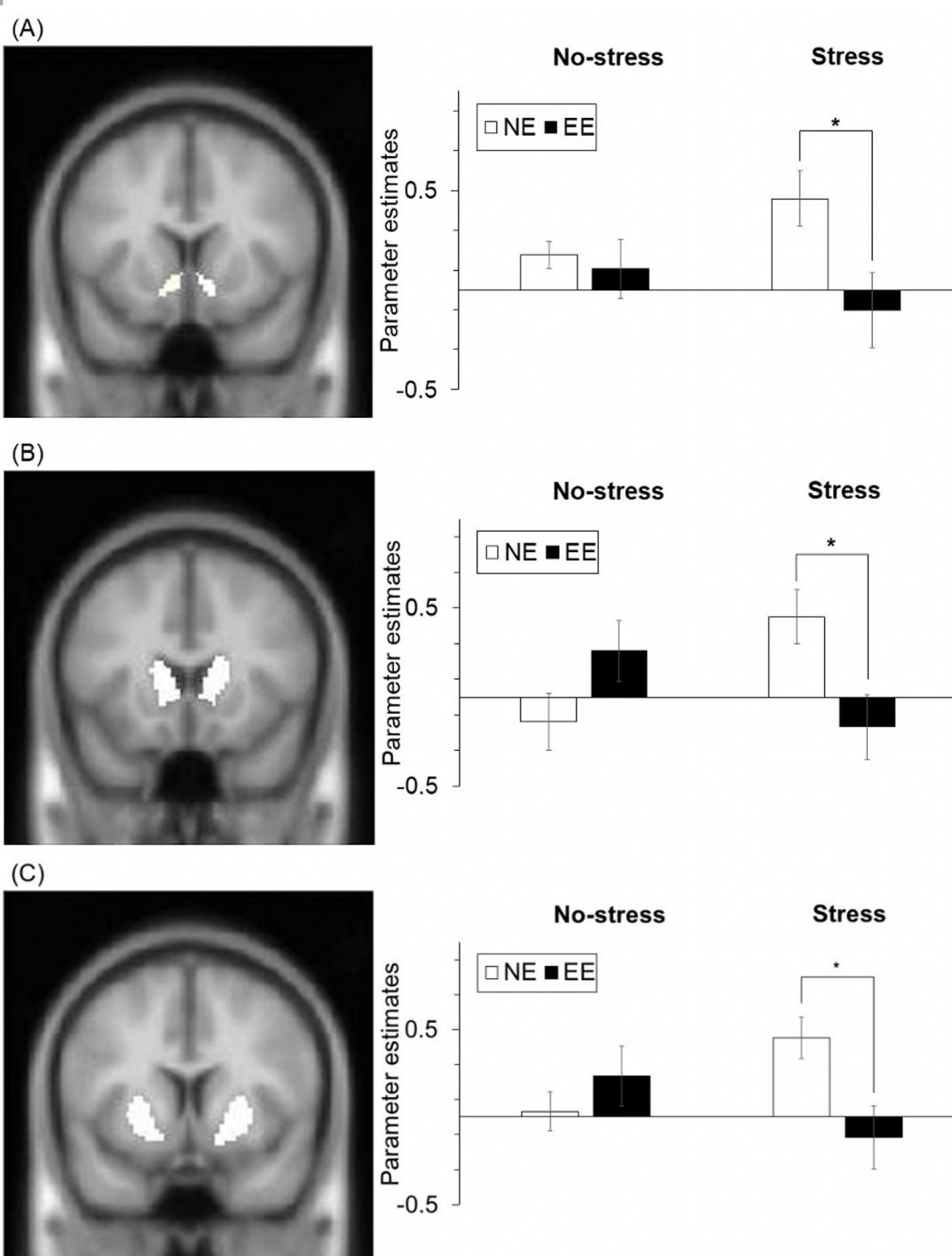


<RESULTS>

Reward-related anticipatory activation

- (A) NAcc
- (B) caudate
- (C) putamen

* $p < 0.05$



<RESULTS>

food reward
receipt & snack
intake

- Snack Intake
 - no significant main effects of Visit, Group, or Visit x Group interaction
- Brain activation during food reward receipt
 - no significant main effects of Group or Visit and no Visit x Group interaction effect

DISCUSSION

- Objective: to investigate the effect of stress on food intake, cortisol reactivity, and food reward circuitry among healthy individuals with varying emotional eating status
- Results (EE showed...)
 - (1) elevated cortisol & anxiety levels in response to a psychosocial stressor
 - (2) significantly reduced activation in caudate, NAcc, and putamen when anticipating food reward compared to NE (stress visit)
 - (3) no difference in snack consumption

DISCUSSION (1) elevated cortisol & anxiety levels

- Consistent with a prior report by Raspopow et al. (2010): EE with more pronounced changes in cortisol
- High cortisol reactivity has been linked to increased food consumption in healthy adults (George et al., 2010)
- High cortisol reactivity & maladaptive emotional coping associated with increased food intake in obesity (Herhaus et al., 2020)
- **Individuals who engage in emotional eating may be physiologically sensitive to stress, which induces acute HPA-axis hyperactivity that may contribute to the maintenance of emotional eating behaviors.**

DISCUSSION (2) reduced activation during food anticipation

- Acute stress increases DA firing to monetary rewards, chronic stress attenuates activation in the reward-related regions (Valenti, Gill, Grace, 2012)
- Stress attenuates reward sensitivity to food cues in healthy individuals (Born et al., 2010), obesity (Wang et al., 2002), and BN (Jimerson et al., 1992)
- **Reduced striatal activation to food reward in response to stress may trigger compensatory behaviors such as emotional eating in an attempt to normalize striatal function.**

DISCUSSION (3) no difference in snack consumption

- Contrary to prior research indicating that stress increases food consumption among stress eaters (Zellner et al., 2006)
- Possible reasons
 - (1) food consumption within a lab setting vs. natural setting
 - (2) temporal delay (2 hours) between stress task and snack period
 - (3) some EE rely on compensatory behaviors other than eating to regulate their overconsumption (Frayn, Livshits, & Knauper, 2018)

STRENGTHS/LIMITATIONS

- The first to integrate fMRI, cortisol sampling, and a psychosocial stressor to examine differences in individuals with varying levels of emotional eating
- (1) constrained distribution of DEBQ scores due to small sample size (most scoring below 3.5 out of 5)
- (2) snack consumption quantified using total caloric intake without macro-/micronutrient variations and/or snack choice
- (3) fMRI paradigm does not include a gustatory stimulus (e.g. milkshake)

CONCLUSION

- These findings provide evidence of aberrant pathways (hyperactivity of the HPA-axis and hypoactivation during anticipation of food reward) underlying emotional eating and highlight stress reduction techniques as a potential therapeutic target for those at risk for developing clinically significant emotional eating behaviors.

RESEARCH INTERESTS

- Mood/anxiety disorders
- Eating behaviors; Eating disorders; Food addiction?
- Effect of stress in healthy individuals/psychiatric population
- fMRI
- Open to other new techniques/realms that I have not experienced as well

Thanks!