Differential fMRI responses to visual food cues between lean and obese subjects

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TARGET AUDIENCE Neuroscientists and physiologists involved in obesity research.

PURPOSE Obesity is at an epidemic proportion, affecting one-third of American adults and 17% of American children. Obesity increases the risk of cardiovascular diseases, such as stroke and heart diseases, cancer and diabetes regardless of race and gender[1]. To better understand how the brain responds to food intake, we used fMRI to probe the neural responses to visual food cues in obese and lean subjects during "fasted" and "satiated" state.

METHODS BOLD fMRI at 3T was performed on ten lean subjects (5M and 5F, 20-41yo, BMI=22.1±2.1kg/m2, fasting glucose level=82±8mg/dL) and the obese subjects (5M and 5F, 22-47yo, BMI=31.3±1.6kg/m^2, fasting glucose level= 91.4±11.8mg/dL). Each fMRI trial consisted of participants viewing high-caloric content food versus non-food pictures over 15 min[2]. fMRI was performed in the fasted state. The subjects were then asked to drink a standard glucola solution (75g glucose in 296 ml) via a peroral tube over 5 min while in the scanner. The second fMRI trial was repeated starting after glucola ingestion.

RESULTS Obese relative to lean displayed increased reactivity in orbitofrontal cortex (BA 47), precuneus (BA 18), fusiform gyrus (BA 19) and inferior frontal gyrus (BA 46), but reduced activity in caudate, precuneus (BA 7), lentiform nucleus, thalamus, parahippocampus and precentral gyrus (BA 2 and 6) in the fasted condition (**Figure 1**). Obese showed increased reactivity in precuneus (BA 31), superior temporal gyrus (BA 22), inferior parietal lobe (BA 40) and orbitofrontal cortex (BA 47) but reduced activity in the precuneus (BA 7), lentiform nucleus, parahippocampus and cerebellum in the satiated condition (**Figure 2**).

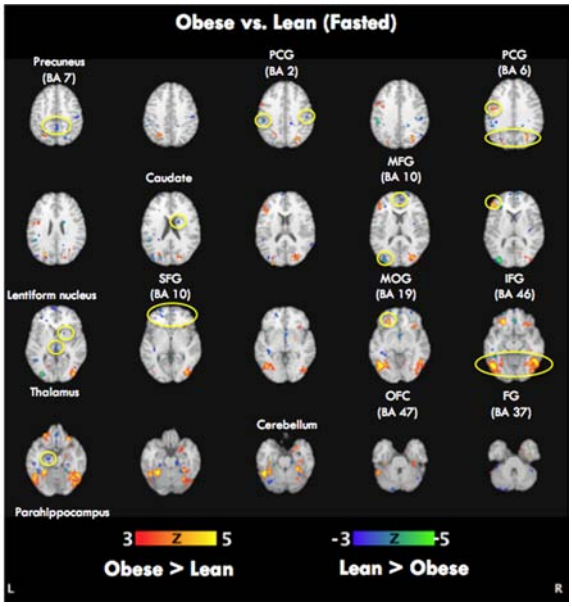

Figure 1. Fasted state: differences in brain response to visual food cues between obese and lean.

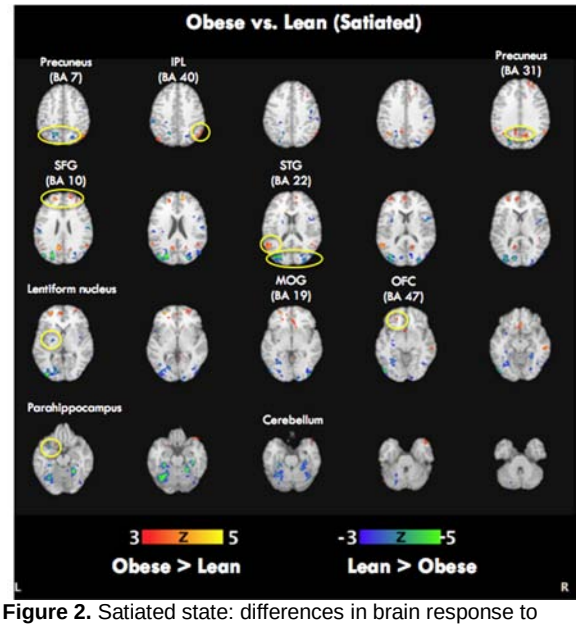

Figure 2. Satiated state: differences in brain response to visual food cues between obese and lean.

DISCUSSION

Obese vs. lean in fasted condition: Obese subjects exhibited increased reactivity in orbitofrontal cortex, fusiform gyrus and inferior frontal gyrus compared to lean. Except for the inferior frontal gyrus, these structures belong to the motivation component of food intake regulation[1]. Fusiform gyrus is part of the secondary visual cortex and it has been implicated in attention tasks. This finding suggests that obese are more attentive to food cues in comparison with lean subjects. Orbitofrontal cortex is responsible for reward value assessment and reward error prediction[3]. Activation of this area has been reported to correlate positively with individual's subjective ratings of pleasantness of food, suggesting that highly palatable foods cause a stronger response in obese compared to lean[4]. Activation of inferior frontal gyrus has been reported in memory retrieval tasks and it falls into the decision-making category. In order to make a decision regarding food selection, it is necessary to evaluate the reward value of the food by accessing to memories of previous experiences.

Lean subjects displayed increased activations compared to obese in regions corresponding to the motivation circuitry, such as caudate, lentiform nucleus, thalamus and parahippocampus. However, cognitive control/decision-making regions were engaged (medial frontal gyrus), suggesting that even though food cues provoke a response, the prefrontal cortex inhibits an inappropriate behavior.

Obese vs. lean in satiated condition: Obese subjects exhibited increased reactivity in orbitofrontal cortex, superior temporal gyrus and inferior parietal lobe. Our findings showed that the orbitofrontal cortex activation did not decrease after eating. However, the prefrontal cortex was engaged. Prefrontal cortex involvement has been reported previously[5] to counterbalance the orbitofrontal cortex. Increased activation of the inferior parietal lobe in obese has been associated with an increase in hunger ratings[6] and it has been reported that a lesion in superior temporal gyrus caused an increment in hunger feelings[7].

Obese subjects displayed decreased activations compared to lean in regions corresponding to the motivation circuitry such as the precuneus, lentiform nucleus and parahippocampus, suggesting that food cues still elicit a response after eating. This may be because only glucose was used. It has been shown that the pleasantness and the motivation to eat more of the same food decrease gradually. However, motivation to consume foods of different taste characteristics remains[8].

CONCLUSION We identified stronger activities in structures, such as orbitofrontal cortex and fusiform gyrus, known to engage in reward processing and attention in obese compared with lean. We also found that the orbitofrontal cortex activation did not decrease after eating in obese compared to lean subjects, suggesting ……. These findings indicate there are strong neural correlates of eating disorders, which may involve the reward/craving circuitry.

REFERENCES (1) Grundy et al. 2004. (2) Labar et al. 2001. (3) Drehet et al. 2012. (4) Kringelbach et al. 2003. (5) Martin et al. 2010. (6) Karhumen et al. 1997. (7) Fisher et al. 2006. (8) Smeets et al. 2006.