Study shows that food may be addictive: research indicates food craving is ‘hard-wired’ in the brain

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A international group of researchers have found that food craving activates different brain networks between obese and normal weight patients. This indicates that the tendency to want food may be ‘hard-wired’ into the brain of overweight patients, becoming a functional brain biomarker.

Obesity is one of the most difficult problems facing modern society. Treating obesity is a health priority, but most efforts (aside from bariatric surgery) have met with little success. In part, this is because the mechanisms associated with the desire to eat are poorly understood. Recently, studies are beginning to suggest that the brain mechanisms underlying obesity may be similar to those in substance addiction, and that treatment methodologies may be approached in the same way as other substance addictions, such as alcohol or drug addiction.

To test this, a group of researchers in the University of Granada, Spain, and Monash University in Australia, have looked for the functional connectivity differences in brain reward systems of normal-weight and obese individuals.

The researchers gave buffet-style food to 39 obese and 42 normal-weight individuals. Later, they were put into functional MRI brain scanners and shown photographs of the food to stimulate food craving. The functional MRI scans showed that food craving was associated with different brain connectivity, depending on whether the subject was normal-weight or overweight.

They found that in obese individuals, the stimulus from food craving was associated with a greater connectivity between the dorsal caudate and the somatosensory cortex, implicated in reward-based habits and the coding of the energetic value of foods, respectively. However, with normal weight individuals, food craving was associated with a greater connectivity between different parts of the brain – e.g. between the ventral putamen and the orbitofrontal cortex.

The researchers then measured Body Mass Index (BMI) three months afterwards and found that 11% of the weight gain in the obese individuals could be predicted by the presence of the increased connectivity between the dorsal caudate and the somatosensory cortex areas of the brain.

According to lead researcher, Oren Contreras-Rodríguez:

“There is an ongoing controversy over whether obesity can be called a “food addiction”, but in fact there is very little research which shows whether or not this might be true. The findings in our study support the idea that the reward processing following food stimuli in obesity is associated with neural changes similar to those found in substance addiction.

This still needs to be viewed as an association between food craving behaviour and brain changes, rather than one necessarily causing the other. However, these findings provide potential brain
Biomarkers which we can use to help manage obesity, for example through pharmacotherapies and brain stimulation techniques that might help control food intake in clinical situations.”

Notes for Editors

Please mention the ECNP conference in any story from this press release

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Background

The ECNP is an independent scientific association dedicated to the science and treatment of disorders of the brain. It is the largest non-institutional supporter of applied and translational neuroscience research and education in Europe.

ECNP organises a wide range of scientific and educational activities, programmes and events across Europe, promoting the exchange of high-quality experimental and clinical research and fostering young scientists and clinicians.

The 28th ECNP Congress takes place in Amsterdam from 29 August-1 September. It is Europe’s premier scientific meeting for disease-oriented brain research, annually attracting between 5,000 and 7,000 neuroscientists, psychiatrists, neurologists and psychologists from around the world.

Website: www.ecnp.eu

Abstract

P.1.i.033 Brain correlates of the desire to food predict body mass index change in excess weight adults O. Contreras-Rodríguez1 # C. Martín-Pérez2, R. Vilar-López2, J. Verdejo-Román3, A. Verdejo-García4 1 Bellvitge Hospital, Department of Psychiatry, Bellvitge, Spain; 2 University of Granada, Department of Psychology, Granada, Spain; 3 University of Granada, Department of Psychology, Granada, Spain; 4 University of Monash, Department of Psychology, Melbourne, Australia

Purpose of the study: Some theories and studies now support that greater anticipated reward from food increases the risk for overeating and obesity [1]. However, although the accumulating evidence of enhanced brain activation during food anticipation [2], no study has comprehensively investigated the association between the functional connectivity of the reward-based striatal brain networks during rest and the subjective desire to appetizing food and subsequent weight increase. This study aims to investigate the association between the functional connectivity of the reward-based striatal brain networks during rest and the desire to high-calorie food in excess relative to normal weight individuals, and whether such brain correlates predict weight increases in the excess weight individuals.

Methods: Eighty-one participants (participants with overweight/ obesity = 39; normal-weight participants = 42) aged 33.34 years (46.9% women) were included in the present study. Ventral and dorsal striatal connectivity were investigated using a validated seed-based multiple regression method [3]. The resulting connectivity maps were compared between the study groups using two-sample models. Moreover, connectivity maps were explored in relation with the desire to consume high-calorie food-items, previously tasted in a previous buffet, and BMI change after a 3-months diet using correlation and regression analyses, respectively.
Results: Excess weight individuals showed significant functional connectivity changes relative to normal weight individuals in both ventral and dorsal reward-based striatal networks. Specifically, excess weight participants mainly showed increased connectivity between the ventral caudate/nucleus accumbens seed and the orbitofrontal cortex, between the ventral striatum (both caudate and putamen seeds) with parietal cortices, including the posterior cingulate cortex, the left angular gyrus and a cluster comprising somatosensory cortices and tempo-insula regions, as well as increased anticorrelation between the ventral caudate/nucleus accumbens seed and the dorsal anterior cingulate cortex extending to the SMA and the posterior insula, and between the ventral putamen seed and the lateral orbitofrontal cortex. Additionally, excess weight participants showed increased connectivity between the dorsal caudate and the somatosensory cortex bilaterally. The functional connectivity between the dorsal caudate and the somatosensory cortex was positively associated with the desire to consume high-calorie food-items and such brain signature predicted respective weight changes after 3-months diet in excess weight individuals. Normal weight individuals, in turn, showed the functional connectivity between the ventral putamen and the orbitofrontal cortex positively associated with the desire to consume high-calorie food items. This latest association was only significant in the excess weight individuals with BMI levels as mediators.

Conclusions: Findings support previous evidence of changes in brain functioning in excess weight individuals. Specifically, results herein show that, in excess weight individuals, these are present even in the intrinsic functional connectivity of reward-based networks, associate food preference and that some gain relevance at increased BMI levels. Importantly, such neural correlates may be useful biomarkers to monitor prediction success.