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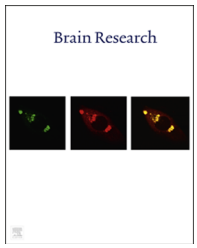

Research Report

Neural correlates of taste and pleasantness evaluation in the metabolic syndrome

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ABSTRACT

Metabolic syndrome (MetS) is a constellation of cardiometabolic abnormalities that commonly occur together and increase risk for cardiovascular disease and type II diabetes. Having MetS, especially during middle-age, increases the risk for dementia in later life. Abdominal obesity is a central feature of MetS; therefore, increased efforts to prevent obesity and identify predictors of weight gain are of extreme importance. Altered processing of food reward in the brain of obese individuals has been suggested to be a possible mechanism related to overeating. We scanned fifteen healthy middle-aged controls (aged 44–54) and sixteen middle-aged adults with MetS after a fast (hungry) and after a preload (sated), while they rated the pleasantness of sucrose (sweet) and caffeine (bitter) solutions. Data were analyzed using voxelwise linear mixed-effects modeling, and a region of interest analysis to examine associations between hypothalamic activation to sweet taste and BMI during hunger and satiety. The results indicate that middle-aged individuals with MetS respond with significantly less brain activation than controls without MetS during pleasantness evaluation of sweet and bitter tastes in regions involved in sensory and higher-level taste processing. Participants with higher BMI had greater hypothalamic response during pleasantness evaluation of sucrose in the sated condition. Importantly, this study is the first to document differential brain circuitry in middle-aged adults with MetS, a population at risk for poor physical and cognitive outcomes. Future research aimed at better understanding relationships among MetS, obesity, and brain function is warranted to better conceptualize and develop interventions for overeating in these disorders.

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Table 1 – Criteria for clinical diagnosis of the metabolic syndrome (Alberti et al., 2009).

Measure	Categorical cut points
Elevated waist circumference	Population and country-specific definitions
Elevated triglycerides (drug treatment for elevated triglycerides is an alternate indicator)[a]	≥150 mg/dL (1.7 mmol/L)
Reduced HDL-C (drug treatment for reduced HDL-C is an alternate indicator)[a]	<40 mg/dL (1.0 mmol/L) in males <50 mg/dL (1.3 mmol/L) in females
Elevated blood pressure (antihypertensive drug treatment in a patient with a history of hypertension is an alternate indicator)	Systolic ≥130 and/or diastolic ≥85 mmHg
Elevated fasting glucose[b] (drug treatment of elevated glucose is an alternate indicator)	≥100 mg/dL

Abbreviations: HDL-C=high-density lipoprotein cholesterol.
[a] The most commonly used drugs for elevated triglycerides and reduced HDL-C are fibrates and nicotinic acid. A patient taking 1 of these drugs can be presumed to have high triglycerides and low HDL-C. High-dose ω-3 fatty acids presume high triglycerides.
[b] Most patients with type 2 diabetes mellitus will have the metabolic syndrome by the proposed criteria.

1. Introduction

The metabolic syndrome (MetS) is a constellation of cardiometabolic abnormalities that tend to co-occur more often than would be expected by chance. The classification is used to identify individuals at increased risk for cardiovascular disease (CVD; Lakka et al., 2002) and type 2 diabetes mellitus (T2DM; Reaven, 1988), who are optimal candidates for behavioral, pharmaceutical and surgical interventions. The risk factors most commonly linked to MetS include insulin resistance, dyslipidemia (elevated triglyceride and low high-density lipoprotein [HDL] cholesterol levels), central obesity, elevated blood pressure, and impaired glucose tolerance or diabetes mellitus (see Table 1). MetS is highly prevalent in the United States, with approximately one-fifth of adults meeting criteria (Beltran-Sanchez et al., 2013). Additionally, not only is the syndrome associated with deleterious physical health outcomes, but having the MetS during middle age substantially increases the risk for future cognitive impairment and dementia (Kalmijn et al., 2000; Whitmer et al., 2005).

The underlying pathophysiological cause of MetS is still relatively unknown, despite a large body of literature on the topic. In debates among the World Health Organization (WHO), the National Cholesterol Education Program Adult Treatment Panel III (ATP, 2002), the International Diabetes Federation (IDF; Alberti et al., 2005), and the American Heart Association/National Heart, Lung and Blood Institute (AHA/NHLBI; Grundy et al., 2005) over how to diagnostically classify the syndrome, both abdominal obesity and insulin resistance have been proposed as primary mechanisms (Abbasi et al., 2002; Alberti et al., 2009; Despres, 2006). Given that abdominal obesity may be a central factor to development of the syndrome, and lifestyle factors including overconsumption of palatable food, play a significant role in the development of abdominal obesity, increased efforts to prevent obesity and to better understand why some individuals gain excessive weight are of extreme importance.

Food reward is a crucial factor involved in food consumption. Specifically, food reward is a complex integrative process that reflects a combination of flavor (taste, smell, texture, irritation), learned associations, and physiological state such as hunger and satiety (Berridge, 1996). The hedonic tone, or pleasantness of a stimulus is modulated by internal signals; thus, food is most pleasant to consume when hungry and decreases as an individual eats to satiety (Cabanac, 1971).

The mesolimbic DA system modulates the experience of food reward and thus, plays an important role in energy intake (Martel and Fantino, 1996). DA release in the dorsal striatum facilitates feeding (Szczypka et al., 2001) and correlates with pleasantness ratings (Small et al., 2003). DA receptor agonists suppress appetite and lead to weight loss (Leddy et al., 2004; Towell et al., 1988), while DA antagonists tend to increase appetite and lead to weight gain (Baptista, 1999).

There is evidence to suggest that abnormal functioning of the DA system may underlie disordered eating in obesity. D2/D3 agonist administration greatly reduces rats' preference for chocolate (Cooper and Al-Naser, 2006) and D2 receptor levels are decreased in the striatum of pathologically obese persons (Wang et al., 2001). Increased BMI is associated with a blunted striatal response to a chocolate milkshake (i.e., an olfactory-taste mixture) especially in individuals with the A1 allele of the Taq1A polymorphism of the DRD2 receptor gene (Stice et al., 2008a). Reduced activation during pleasantness evaluation of a sweet taste in the nucleus accumbens, caudate nucleus and amygdala is strongly related to higher BMI and waist circumference in older adults (Green et al., 2011).

The hypothalamus plays a crucial role in the maintenance of energy homeostasis through modulation of eating behavior, neuroendocrine function, and reward (Berthoud and Morrison, 2008). Hypothalamic nuclei integrate hormone and nutrient signals regarding an individual's current state of energy balance. The arcuate nucleus of the hypothalamus is sensitive to the body's state of energy balance (Berthoud and Morrison, 2008). The periventrical nucleus of the hypothalamus receives input from the arcuate nucleus, has widespread projections in the cortex, and is associated with neuroendocrine function and the hypothalamic–pituitary axis. The lateral hypothalamus receives inputs from the arcuate nucleus in addition to other regions involved in sensory processing, reward value, and learning and memory (OFC, nucleus accumbens, amygdala, ventral tegmental area, insula) and has widespread projections to the cortex (Berthoud, 2002). Activation of the lateral hypothalamus is strongly related to feeding behavior, and this region projects widely throughout the brain. Electrical stimulation of the

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