Behavioural Brain Research 317 (2017) 163–178

Contents lists available at ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Research report

The impact of a junk-food diet during development on 'wanting' and 'liking'

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HIGHLIGHTS

- Junk-food (JF) exposure during development reduces weight gain in two strains of rats.
- JF-induced weight gain has opposite effects on cue attraction in males and females.
- JF gainers work harder for cues and are more attracted to a JF context.
- JF exposure in Long-Evans, but not Sprague-Dawley rats, blunts sucrose 'liking'.
- JF exposure reduces anxiety-like behavior in males, but not females.

ARTICLE INFO

Article history:
Received 5 August 2016
Received in revised form
15 September 2016
Accepted 16 September 2016
Available online 17 September 2016

Keywords:
'Wanting'
'Liking'
Incentive salience
Junk-food
Obesity
Motivation

ABSTRACT

The global increase in obesity rates has been tied to the rise in junk-food availability and consumption. Increasingly, children are exposed to a junk-food diet during gestation and early development. Excessive consumption of junk-food during this period may negatively impact the development of brain motivation and reward pathways. In this study we investigated the effects of a chronic junk-food diet throughout development on cue-motivated behavior ('wanting'), hedonic 'liking' for sweet tastes, as well as anxiety and weight gain in male and female Long-Evans (LE) and Sprague-Dawley (SD) rats. Here we found that chronic exposure to a junk-food diet resulted in large individual differences in weight gain (gainers and non-gainers) despite resulting in stunted growth as compared to chow-fed controls. Behaviorally, junk-food exposure attenuated conditioned approach (autoshaping) in females, particularly in non-gainers. In contrast, junk-food exposed rats that gained the most weight were willing to work harder for access to a food cue (conditioned reinforcement), and were more attracted to a junk-food context (conditioned place preference) than non-gainers. Hedonic 'liking' reactions (taste reactivity) were severely blunted in LE, but not SD rats, and 'liking' for sucrose negatively correlated with greater weight gain. Finally, junk-food exposure reduced anxiety-like behavior (elevated plus maze) in males but not females. These results suggest that junk-food exposure during development may give rise to dissociable differences in 'liking' and 'wanting' neural systems that do not depend on weight gain and may not be detected through Body Mass Index monitoring alone.

1. Introduction

Obesity is a global health risk, and the rapid escalation of its prevalence suggests shifting environmental factors may have a role in its growth. As of 2012, over 15% of children and over 30% of adults in the United States are obese, while another 30% of the population is overweight [1]. These numbers are representative of a growing obesity epidemic [1–3]. The growing accessibility of inexpensive processed foods and their increasingly pervasive advertising may play a role in this alarming trend [4,5]. Many of these processed foods are saturated with sugar, salt, and fat. Yet they lack adequate protein and other nutrients that are important for day-to-day health and normal growth and development, categorizing them as "junk-food". In countries with high and rising obesity rates, daily

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http://dx.doi.org/10.1016/j.bbr.2016.09.041
0166-4328/

food intake is not exclusively driven by hunger or energy demand. It is suggested that for some individuals, the increased palatability and accessibility of junk-food has seized neural reward and motivation mechanisms and turned food-seeking into errant food craving, which may lead to diet-induced obesity [6,7].

When a new food is first ingested, its sensory qualities may trigger sensations of hedonic pleasure and 'liking', which in turn promote 'wanting' to consume that food again [6,8]. With repeated exposure, however, the environmental cues associated with the junk-food may gain more motivating power and incentive value. The salience of cues associated with food is facilitated through activity in mesocorticolimbic systems, which makes rewards and their cues desired and 'wanted' [9–11]. The neural systems for 'liking' and 'wanting' typically function in close synchrony, but data show that they can be changed independently. For example, with repeated consumption of a reward, such as palatable junk-food, 'wanting' becomes sensitized [11,12]. Sensitization of 'wanting' was first described in the 'incentive sensitization' theory of addiction, and can result in a dissociation of 'wanting' and 'liking' that leads to strong feelings of desire for particular rewards and their cues, despite no increase and sometimes a reduction in 'liking' [13]. Although it was initially applied to drugs and their cues, recent evidence suggests this theory also applies to food cues. Food cues can play a similar role by triggering visual attention and enhancing the desire to eat [14–17], particularly in obese individuals who might be hyper-responsive to the motivational properties of these cues [18–20].

However, susceptibility to (incentive) sensitization appears to show a large degree of individual variation, with marked sex differences [21]. For example, there is evidence for individual variation in the level of attraction and motivation to junk-food cues [12,22,23]. In particular, we recently demonstrated that animals that gained excessive weight on a junk-food diet (gainers) displayed greater cue-induced approach to food cues even before gaining access to the diet [12], and were also more willing to work for the presentation of those cues (conditioned reinforcement) after obesity onset. However, many of these studies were carried out in adults. The current ease of access and high palatability of these foods means that exposure to a junk-food diet may begin as early as childhood or even prior to birth through the mother's diet.

Childhood obesity has been implicated as a cofactor in a number of lifetime diseases such as depression, anxiety, diabetes, elevated blood pressure, orthopedic problems, and pulmonary complications [24–26], and has been associated with early mortality [27,28]. Previous studies have shown that a mother's diet during pregnancy alters the protein make-up of the offspring's cerebral cortex despite cross-fostering [29], while also producing changes in dopaminergic activity [30]. Developmental perspectives on the obesity epidemic are necessary to understand the increasing prevalence of childhood obesity across generations [1,25], and dissociations between 'liking' and 'wanting' could have a lasting impact when occurring within the plastic neural networks of a maturing brain. However, it is currently unclear whether overconsumption of junk-food is related to distortions of either 'liking' or 'wanting', or both, when exposure begins prenatally.

Here we examined the effect of lifetime exposure to junk-food on 'wanting' by measuring the degree to which food cues 1) elicit approach (autoshaping), 2) reinforce operant responding (conditioned reinforcement), and 3) by determining the attraction of a junk-food paired context (conditioned place preference). We also measured the impact of lifetime exposure to a junk-food diet on hedonic orofacial 'liking' reactions, using taste reactivity measures [31] in response to sucrose. In addition, since anxiety is often associated with increased consumption of fatty-sugary foods [32], we also evaluated individual differences in the impact of junk-food on levels of anxiety-like behavior using the elevated plus maze. Finally there are marked sex differences in the motivation for food [33,34]. Recent findings also show strain and sex differences for spatial learning [35,36], behavior toward unfamiliar foods [37] and metabolic responses [38]. Therefore measures of 'wanting', 'liking' and anxiety were determined in males vs. females, across two strains of rats, Long-Evans and Sprague-Dawley.

2. Materials & methods

2.1. Subjects

Long-Evans (LE) and Sprague-Dawley (SD) rats were bred in-house from breeding pairs purchased from both Harlan and Charles River. Rats were housed on a 12:12 h reverse light/dark cycle and had ad-lib access to food and water unless stated otherwise. All procedures were approved by the Institutional Animal Care and Use Committee for Wesleyan University.

2.2. Diet

Adult male and female rats were placed on either a standard chow and junk-food (JF) or a control diet (C; Teklad Rodent Lab Diet 2018 in pellet form; Envigo: 18.6% protein, 6.2% fat, 44.2% carbohydrates; 3.1 kcal/g) for seven days prior to assigning C and JF breeding pairs (Fig. 1A). The junk-food diet was a mash composed of a blend of Chips Ahoy chocolate chip cookies (260 g), Ruffles potato chips (80 g), Jiff creamy peanut butter (260 g), Nesquik chocolate flavored powder (260 g), powdered Rodent Lab Diet 2018 (400 g; 14% protein, 19.6% fat, 58% carbohydrates; 4.5 kcal/g), and water (355 ml). These foods contain a rich mix of sugars, salt, and fats, and were chosen as palatable representatives of what are commonly called 'junk-foods' implicated in human obesity. Breeding pairs were maintained on their designated diet (C or JF) until weaning of offspring (postnatal day (PND) 21). Litters were culled to a total of ten (five female; five male) and maintained on the same diet as their parents throughout the experiment, thus creating two groups: a control group that received only standard chow (C: N = 30: LE M/F = 10/10, SD M/F = 7/3), and an experimental group that received junk-food in addition to standard chow throughout gestation, post weaning and throughout the remainder of the experiment (JF: N = 60: LE M/F = 23/17, SD M/F = 8/12). After weaning, rats were housed by sex in groups of two or three. All tests were carried out in red light conditions during the dark cycle unless otherwise stated. Testing began at 12–14 weeks of age (i.e., adulthood). Animals were weighed once per week before weaning and twice per week post-weaning.

2.3. Autoshaping/conditioned reinforcement/extinction

2.3.1. Apparatus

All procedures were conducted in standard Med-Associates chambers equipped with two retractable levers (one CS (illuminated), one control) located on the front wall on either side of a recessed food cup, which delivered 45 mg sucrose pellets (Test-Diet). A speaker located at the top of the chamber delivered a 2.9 kHz tone. For the conditioned reinforcement session, the back wall was outfitted with nose-poke holes (one active, one inactive, location counterbalanced) located on either side of a central retractable lever. During this time the food cup on the front wall was covered with a custom metal plate. Med-PC software automatically collected lever responses, nose pokes, and food cup entries for all sessions. Chambers were placed in sound attenuating cabinets to reduce ambient light and noise. Red LED house lights were mounted to the wall of the cabinet and were turned on during all sessions.