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Sensory-specific satiety is intact in rats made obese on a high-fat high-sugar choice diet

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ABSTRACT

Sensory-specific satiety (SSS) is the temporary decreased pleasantness of a recently eaten food, which inhibits further eating. Evidence is currently mixed whether SSS is weaker in obese people, and whether such difference precedes or follows from the obese state. Animal models allow testing whether diet-induced obesity causes SSS impairment. Female rats ($n = 24$) were randomly assigned to an obesogenic high-fat, high-sugar choice diet or chow-only control. Tests of SSS involved pre-feeding a single palatable, distinctively-flavored food (cheese- or cocoa-flavored) prior to free choice between both foods. Rats were tested for short-term SSS (2 h pre-feeding immediately followed by 2 h choice) and long-term SSS (3 day pre-feeding prior to choice on day 4). In both short- and long-term tests rats exhibited SSS by shifting preference towards the food not been recently eaten. SSS was not impaired in obese rats. On the contrary, in the long-term tests they showed stronger SSS than controls. This demonstrates that neither the obese state nor a history of excess energy consumption fundamentally causes impaired SSS in rats. The putative impaired SSS in obese people may instead reflect a specific predisposition, properties of the obesogenic diet, or history of restrictive dieting and bingeing.

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1. Introduction

Food choice and amount consumed are both strongly influenced by the sensory properties of available foods. Obviously, good-tasting foods are preferred and more likely to be overconsumed, but even the most palatable food can become progressively less pleasurable throughout a meal. *Sensory-specific-satiety* (SSS) refers to the declining pleasure and attraction to the sensory attributes of the specific foods eaten in the meal relative to other foods (Hetherington&Rolls, 1996, p. p267; Rolls, 1986; Rolls, Rowe, & Sweeney, 1981). Though eating to satiety generally suppresses appetite, the foods eaten in the meal become much less attractive than others. Hence, varied meals with multiple courses can often be quite large, and even after a large meal the pleasure of eating can rapidly return when dessert arrives. SSS is an immediate effect of the sensory attributes of the eaten food independent of its postingestive consequences. It contributes to meal termination and then gradually decays in the post-meal interval, and thus impaired SSS would promote overeating by permitting larger meals or more rapid resumption of eating.

Some evidence suggests humans who are obese may show a weaker or slower decline in hedonics during a meal. The clearest evidence comes from studies of habituation of automatic responses (e.g., salivation) or motivational impact (i.e., willingness to work for more) of a stimulus as a result of repetitive, monotonous exposure to it. Since eating inherently involves repetitive exposure, habituation of responses to initially-pleasurable sensations may explain loss of inter-

est and enjoyment of the food which promotes meal termination (Epstein, Temple, Roemmich, & Bouton, 2009). Indeed, reflexive salivation habituates over a series of small exposures to a palatable flavor and dishabituates with introduction of a new flavor (Epstein, Rodefer, Wisniewski, & Caggiola, 1992; Temple et al., 2006), but individuals with obesity show much less decline (Bond et al., 2009; Epstein, Paluch, & Coleman, 1996). Similarly, hedonic ratings of a sweet taste decrease more slowly over repeated tasting for individuals with obesity (Pepino & Mennella, 2012). Compared to lean children, overweight children exhibit slower decline in motivation to persist at a task to earn small tastes of a food (Temple, Giacomelli, Roemmich, & Epstein, 2007).

However, overall the evidence for SSS impairment in obesity is mixed. Studies relying on hedonic ratings of foods before and after a meal have generally not found lean-obese differences on these measures (Brondel et al., 2007; Snoek, Huntjens, Van Gemert, De Graaf, & Weenen, 2004). Nor do individuals with obesity show more rapid return of hedonic evaluation of a recently eaten food in the post-meal interval (Havermans, Roefs, Nederkoorn, & Jansen, 2012). Thus additional research on this topic is necessary to resolve these discrepancies and determine if and under what circumstances SSS impairments may occur, and further, the direction of causation. A difference in SSS could be a preexisting causal factor in overeating, or could emerge as a consequence of chronic overeating or positive energy balance. Thus controlled studies in an animal model of diet-induced obesity would be valuable for dissociating these possibilities.

Studies of these effects in animal models are few but do provide clear evidence for sensory-specific decreases in food motivation following from recent consumption. Work based on the habituation paradigm has shown in both infant and adult rats that a series of brief,

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small intra-oral infusions of a palatable flavored solution causes gradual decrease in the automatic mouthing and licking responses that indicate hedonic evaluation, and temporarily suppresses voluntary intake of that flavor (Swithers & Martinson, 1998; Swithers-Mulvey & Hall, 1992). The hedonic response immediately returns when the flavor is changed, demonstrating it is a sensory-specific effect. In monkeys, neural responses to the sight or taste of a food in several ventral forebrain areas are suppressed more for a recently-eaten food than for a non-eaten food (Rolls, Murzi, Yaxley, Thorpe, & Simpson, 1986). In experiments directly analogous to the SSS paradigm, rats fed a distinctive sweet or savory snack food in one meal consume substantially less in a second meal when offered the same food than if given the opposite food, and this effect is paralleled by shifting dopamine responses in prefrontal cortex and nucleus accumbens (Ahn & Phillips, 1999).

There is, however, little animal work specifically investigating diet-induced obesity (DIO) and SSS. The most relevant study does show that prior history with a palatable, varied cafeteria diet impairs SSS in rats (Reichelt, Morris, & Westbrook, 2014). But additional work would be necessary to determine if that impairment is attributable to weight gain itself, or specific macronutrients, or to the history of sensory variety, palatability, or some other aspect of the cafeteria diet. The purpose of the present experiment was to directly compare SSS in lean control rats versus rats with DIO induced by a high-fat, high sugar (HFHS) choice protocol. This protocol produces dramatic weight gain, and effectively models many physiological and behavioral aspects of human diet-induced obesity. Rats fed a HFHS choice diet persistently increase calorie intake and fat stores, adopt a pattern of "snacking" between meals, develop peripheral leptin resistance and impaired glucose metabolism, and show dysregulated food motivation (la Fleur, Luijendijk, van der Zwaal, Brans, & Adan, 2014; La Fleur, Luijendijk, Van Rozen, Kalsbeek, & Adan, 2011; La Fleur et al., 2007; Wald & Myers, 2015).

In this experiment SSS was measured by feeding rats a palatable snack food with distinctive sensory properties (either cheese-flavored corn snacks or chocolate-flavored breakfast cereal) for some time prior to offering a free choice between both of those foods. SSS would shift preference away from the food that had recently been eaten. In humans SSS acts in the short term to promote cessation of a meal, and also in the longer term when the same food is eaten repeatedly over days (Raynor & Wing, 2006; Rolls & De Waal, 1985; Weenen, Stafleu, & De Graaf, 2005). As these may represent separate processes, the present study included both a short-term and long-term tests.

2. Methods

2.1. Subjects

All procedures were approved by the Bucknell University IACUC. Subjects were 24 experimentally naive female Sprague Dawley rats from our breeding colony. At the start rats were 125–140 days old and weighed 303.6 ± 21.0 (Mean \pm SD). Rats were housed in $8 \times 16 \times 10.5$ " plastic tub cages with corncob bedding, in a colony room maintained at approximately 21 °C and 40% humidity, with a 12:12 h light:dark cycle (lights on at 0800).

2.2. Diet-induced obesity

Two groups matched for initial body weight were created by random assignment. The control group (CON, $n = 12$) was maintained on an ordinary, cereal-based lab chow (Mazuri 5663) ad libitum, whereas the diet-induced obesity (DIO) group ($n = 12$) was fed the same chow

plus ad libitum access to both lard (Armour Star, ConAgra Foods) and 30% sucrose solution. Lard was provided in a cup hung inside the cage and sucrose was in a 200 ml bottle on the cage lid. Both were provided in ample amounts and replenished daily. All CON and DIO rats also had ad libitum drinking water. Rats were maintained on these diets for 6 months prior to beginning the behavioral measurements.

2.3. Test foods

Sensory-specific satiety tests involved two palatable snack foods: Cheese Balls (Utz brand, Hanover, PA) and Cocoa Puffs (General Mills, Golden Valley, MN). Both have corn flour as the chief ingredient but are distinct in taste and flavor. Cheese Balls are savory and high fat, and Cocoa Puffs are chocolate flavored, sweet, and lower in fat. Rats were familiarized with 5 g of each in the home cage prior to a preliminary preference measurement conducted approximately one week before the main experiment. In this initial preference test rats were given ad lib overnight (18 h) access to both foods in the absence of chow. All rats moderately preferred cocoa over cheese, but CON and DIO rats consumed similar amounts and had similar preference (Mean \pm SD intakes, CON: 15.1 ± 2.5 g cocoa and 7.9 ± 3.0 g cheese; DIO: 17.7 ± 3.8 g cocoa and 5.9 ± 3.0 g cheese. CON and DIO values are not significantly different.)

2.4. Long-term sensory-specific satiety

This test was conducted to determine if rats' relative preference for the two snack foods shifted after consuming one of them repeatedly over several days. During this testing HFHS diet was discontinued for DIO rats. Each rat was provided 20 g/day of only one of the snack foods for three consecutive days, plus ad libitum chow. Half the rats in CON and DIO received cheese and the other half cocoa. On the fourth day, all food was removed for 6 h prior to a free choice between cheese vs. Cocoa. Ample pre-weighed amounts of both foods were provided in adjacent feeders, and overnight (18 h) intake was measured by weighing the remainders the following day. Care was taken to collect any spilled food from the bedding for measurements. Rats were then given ad libitum chow for 6 days before the cycle was repeated, with each rat receiving the opposite initial food for three days prior to a second choice test. Thus all rats were tested for their preference for cheese vs. Cocoa after three days of eating cocoa, and after three days of eating cheese, but the order of those tests was counter-balanced. After this testing, DIO rats were returned to the HFHS diet for two weeks before proceeding.

2.5. Short-term sensory-specific satiety

This test was conducted to determine if consuming one of the two foods in a 2-hr period immediately impacts relative preference in the subsequent 2-hr period. All food was removed from the home cages 4 h prior to testing. Starting at dark onset, rats were provided with either cheese or cocoa (counterbalanced) in the absence of chow. Two hours later, remaining initial food was removed and each rat was provided pre-weighed amounts of both cheese and cocoa. Preference was determined by removing and weighing the remainders after 2 h. Rats were provided ad libitum chow for the next three days, and then the test was repeated with all rats receiving the opposite initial food.

2.6. Data analysis

Preference for cocoa over cheese was arbitrarily chosen to depict the results of the choice tests (%COC = intake of Cocoa \div [total Cocoa + Cheese intake] * 100). A %COC value of 50% would mean

equivalent intakes of cocoa and cheese in a choice test. SSS would be indicated if %COC after cheese pre-feeding was significantly greater than %COC following cocoa pre-feeding.

Short-term SSS and long-term SSS were analyzed separately but similarly. In each case rats' %COC after cocoa pre-feeding vs cheese pre-feeding was compared in a 2 (Pre-food) X 2 (Group) mixed ANOVA with pre-food as a within-subjects factor and group (CON or DIO) as a between-subjects factor.

For both short-term and long-term tests, the two groups' intakes during the pre-feeding were compared with independent *t*-tests. This was to determine if any between-group difference in apparent SSS revealed in the choice test was actually an artifact of the groups consuming different absolute amounts during the pre-feeding phase.

3. Results

3.1. Diet-induced obesity

As intended, the DIO group was substantially heavier than CON, $t(22) = 6.93, p < 0.001$, as depicted in Fig. 1.

3.2. Long-term SSS

Both groups showed a robust SSS effect on Day 4 after pre-feeding one of the foods on Days 1 through 3. Although all rats moderately preferred cocoa over cheese (mean %COC values > 50% in all tests), the strength of that preference varied significantly according to which food was recently eaten (Fig. 2A). %COC after cheese pre-feeding was significantly higher than after cocoa pre-feeding, $F(1,22) = 37.1, p < 0.001$, indicating SSS. Post-hoc tests of simple main effects (paired *t*) confirm that %COC was significantly greater after cheese than after cocoa for both groups (both $p < 0.01, df = 11$). Contrary to the hypothesis that SSS is impaired in obesity, the differential preference was larger in DIO than CON, Pre-food X Group interaction, $F(1,22) = 5.66, p < 0.05$, with no main effect of Group on %COC, $F(1,22) = 1.47, n.s.$

This apparent group difference in SSS was not attributable to differential consumption of the pre-foods. CON and DIO rats consumed similar amounts of pre-food when it was cheese, $t(22) = 1.02, n.s.$, and when it was cocoa, $t(22) = 0.32, n.s.$

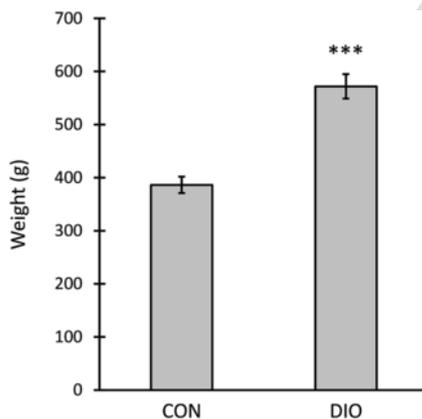


Fig. 1. Mean (\pm SEM) bodyweights of the CON and DIO groups at the outset of behavioral testing, following maintenance on chow only (CON) or chow and ad libitum 30% sucrose solution and lard (DIO). *** $p < 0.001$.

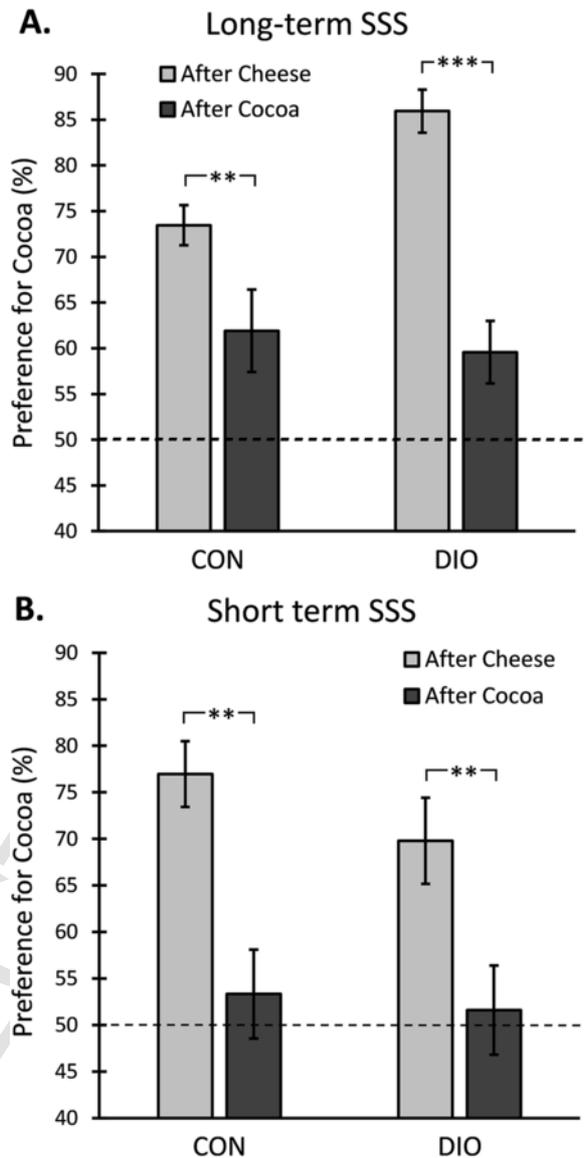


Fig. 2. Mean (\pm SEM) preference in the choice tests conducted after long-term (panel A) and short-term (panel B) pre-feeding. In the long-term SSS test, rats were fed either the cheese- or cocoa-flavored snack daily on Days 1–3, prior to a choice test on Day 4. In the short-term SSS test rats were fed either cheese- or cocoa-flavored snack for 2 h immediately prior to the 2 h choice. %COC is the relative intakes of cocoa and cheese, such that a value of 50% (dashed reference line) would indicate equal consumption of the two. ** $p < 0.01$, *** $p < 0.001$.

3.3. Short-term SSS

Both groups demonstrated SSS when tested immediately after pre-feeding one of the foods for 2 h. Again all rats moderately preferred cocoa over cheese, but preference for cocoa was affected by the pre-feeding (Fig. 2B). %COC after cheese pre-feeding was significantly higher than after cocoa pre-feeding, $F(1,32) = 41.3, p < 0.001$, indicating SSS. Post-hoc tests of simple main effects (paired *t*) confirm that %COC was significantly greater after cheese than after cocoa for both groups (both $p < 0.01, df = 11$). Preferences were similar in CON and DIO rats, no main effect of Group, $F(1,22) = 0.66, n.s.$, and no Pre-food X Group interaction, $F(1,22) = 0.70, n.s.$

CON and DIO rats consumed similar amounts of pre-food during the cheese pre-feeding, $t(22) = 1.22$, *n.s.*, and during the cocoa pre-feeding, $t(22) = 1.69$, *n.s.*, confirming that the similar preference shift in the choice test followed similar pre-feeding amounts.

4. Discussion

This experiment measured SSS by pre-feeding rats one food for a period of time prior to a free choice between it and another food. These tests demonstrated SSS acting on both short-term (hours) and longer-term (days) time scales. In the short-term, 2-h access to only one food decreased relative preference for that food in the subsequent 2-h. In the longer term, eating one of the foods repeatedly over three days affected preference on the fourth day. In neither case did rats made obese on a high-fat, high-sugar diet show impaired SSS. On the contrary, their long-term SSS was somewhat stronger than controls. Short-term SSS was equivalent in the two groups.

The apparent stronger long-term SSS in obese rats was unexpected. While no prior work has indicated stronger SSS in obese individuals, this could be viewed as consistent with an increased reactivity to orosensory properties in obesity (i.e., 'finickiness'). For instance, diet-induced obese rats treat high sugar concentrations as more palatable but low sugar concentrations as less palatable than do lean control rats (Shin, Townsend, Patterson, & Berthoud, 2011), suggesting they are over-reactive to sensory manipulations that nudge preference.

Another possibility is the apparently stronger SSS is secondary to enhanced sweet craving in particular. *Post hoc* inspection of the data reveals this effect was asymmetrical: obese rats showed stronger SSS than controls only when the pre-food was cheese. Obese and lean rats were similar when the pre-food was cocoa. The three-day prefeeding with cheese involved extended abstinence from any sweet taste after prior chronic access to ad libitum sugar. Such abstinence after habitual sugar consumption can powerfully stimulate motivation for sugar (Avena, Long, & Hoebel, 2005; Grimm, Fyall, & Osincup, 2005), which escalates with the duration of abstinence (the 'incubation of craving' (Grimm et al., 2005)). That would explain why it would be seen in the long-term but not short-term SSS tests. Pre-feeding with cocoa (which was sweet) did cause SSS but not any moreso than for control rats, further suggesting that the increased long term SSS among obese rats likely reflects enhanced sweet motivation.

The present finding that SSS is not impaired in rats made extremely obese on a HFHS diet should not be taken to suggest that no impairments in SSS should be expected in humans of different weight status. Rather the goal of an animal model like this is to help elucidate how any such differences may come about. The present results cast doubt on the view that the physiological consequences of the obese state *per se* or of chronic positive energy balance, or even the experiential history with excessive sweet and fat stimuli, fundamentally disrupt the core features of SSS – a memory of recently consumed food and a consequent decrease in the attraction to or evaluation of that food.

While SSS is a memory-mediated phenomenon, and a variety of work links obesity to short-term memory impairment (Gunstad, Paul, Cohen, Tate, & Gordon, 2006; Kanoski & Davidson, 2011; Winocur & Greenwood, 2005), profoundly amnesic humans still exhibit normal SSS even when lacking declarative memory of recent eating (Higgs, Williamson, Rotshtein, & Humphreys, 2008), setting SSS apart from some other memory systems involved in meal patterns. The present finding provides evidence that the memory systems mediating SSS are spared from the impairments caused by diet-induced obesity.

It is possible there may be differences for obese and lean humans in aspects of SSS that are not captured by this animal model. For in-

stance, in humans, distraction by external stimuli prevents the gradual decrease in food evaluation that ordinarily follows repeated exposure (Epstein et al., 1992, Epstein, Paluch, Smith & Sayette 1997) potentially explaining how distractors like TV promote overeating. Such features typical of human eating contexts are not modelled in this experiment. Short term tests were conducted in the first several hours after lights out, when rats eat vigorously, with presumably minimal environmental distractors. Thus it remains possible that differential susceptibility to some intervening factor like distraction could produce differences in SSS. Thus research on SSS may benefit from pursuing situational moderators of the effect, as the present results show neither the obese state *per se* nor chronic overconsumption directly impact the core mechanism of SSS.

Work that supports lean-obese differences in this realm mainly comes from the habituation model, which is conceptually linked to SSS. There are procedural differences in experiments stemming from the two perspectives (Epstein et al., 2009), but the habituation model predicts many of the features of SSS and habituation may be a core neuropsychological mechanism underlying SSS. If that is the case, the present findings support the view that lean-obese differences that have been shown in this domain (5–10) do not result from obesity but may instead be a pre-existing risk factor that predisposes some individuals towards overeating. For instance, individual differences in habituation rate among normal weight pre-teens prospectively predicted subsequent BMI gains in the following year (Epstein, Robinson, Roemmich, & Marusewski, 2011), consistent with the idea that a tendency to slowly habituate to food stimuli may be an early phenotypic marker of potential weight problems.

While the present study shows that neither the obese state *per se* nor a history of excess energy consumption directly causes SSS impairment in rats, other research with animal models suggest some alternative causal links between obesity and SSS impairment in humans. The first is a history of periodic binge-like consumption interspersed with restriction. Rats maintained on a cyclic schedule of food restriction which promotes binge-like overeating subsequently showed impaired SSS (Ahn & Phillips, 2012). That effect was not seen in rats who were merely restricted to limited rations for an extended period, nor was body weight itself a factor, demonstrating that the alternation between restriction and excess consumption was the relevant variable. Since humans with obesity often have a history of repeated, self-imposed but short-lasting restrictive diets, they may experience a similar result. In support of this notion, individuals with bulimia – which is defined by binge eating but not overweight – also show impaired SSS (Hetherington & Rolls, 1989).

The second factor revealed by animal models that could impact SSS in humans is the sensory variety in a processed 'junk food' diet. In the present experiment DIO was induced with consumption of sucrose solution and lard, giving the rats experience with palatability but only limited variety. Other experiments using a cafeteria diet model which induces hyperphagia with a wider range of differently flavored processed 'supermarket' foods (e.g., pastries, chips, meat snacks, etc) has found subsequent impairment of SSS in rats (Reichelt et al., 2014). Since those rats, like the DIO rats in the currently study, were hyperphagic and obese, the different outcome suggests that the sensory properties of the obesogenic diet may determine whether SSS is affected. It has been suggested that sensory-nutrient variability in the diet – that is, the extent to which varied flavors do or do not reliably predict differential post-ingestive outcomes – might impair normal satiety processes, especially learned responses to foods' sensory attributes (Davidson & Swithers, 2004; Hardman, Ferriday, Kyle, Rogers, & Brunstrom, 2015; Martin, 2016).

For these reasons, along with the 'incubation of sweet craving' phenomenon mentioned previously, future work on SSS in humans

and in animal models should pay close attention to the specific properties of the test foods relative to the background diet. In the present study, for instance, maintenance on a *sweet* and high-fat diet to induce DIO presumably had some effects on rats' choices between sweet and non-sweet test foods. Clear effects of SSS were evident nonetheless, demonstrating how robust SSS can be.

Finally, because SSS appears to be an important influence on food selection and appetite, both within individual meals and in the longer term, it can be considered as a target of behavioral/lifestyle interventions to discourage overeating. Such an approach would have slim chance of success were it the case that individuals with obesity simply did not experience a significant degree of SSS. The present finding that SSS is fully intact in this an animal model of obesity leaves SSS as a potentially viable option for intervention, at least in some individuals. That option would benefit from further work on the pre-existing individual differences that may promote the onset of obesity, and the specific features diet and prior history that may modulate the effect.

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