Acute tryptophan depletion and sweet food consumption by overweight adults

Sherry L. Pagoto, Bonnie Spring, Dennis McChargue, Brian Hitsman, Malaina Smith, Bradley Appelhans, Donald Hedeker

1 Department of Medicine, Division of Preventive and Behavioral Medicine, University of Massachusetts Medical School, 55 Lake Avenue North, Worcester, MA 01655, USA
2 Department of Preventive Medicine, Northwestern University, Feinberg School of Medicine, 680 North Lake Shore Dr, Suite 1229, Chicago, IL 60611, USA
3 Department of Psychology, University of Nebraska-Lincoln, 238 Burnett Hall, Lincoln, NE 68588, USA
4 Centers for Behavioral and Preventive Medicine, The Miriam Hospital and Brown Medical School, Coro Building, Suite 500, One Hoppin Street, Providence, RI 02903, USA
5 School of Public Health (MC 923), University of Illinois at Chicago, 1603 W. Taylor St, #955, Chicago, IL 60612, USA

ARTICLE INFO

Article history:
Received 13 June 2008
Received in revised form 27 August 2008
Accepted 21 October 2008

Keywords:
Acute tryptophan depletion
Sweet food
Overweight
Serotonin
Obesity

ABSTRACT

Serotonergic involvement has been implicated in preferential consumption of treat foods. We tested the effect of acute tryptophan depletion (ATD) on food consumption by overweight and lean adults with and without a history of recurrent major depressive disorder (MDD). ATD and taste-matched placebo challenges were administered double-blind in counter-balanced order. Participants were classified as lean (n=36) or overweight (n=19) on the basis of body mass index (BMI). Total calorie, carbohydrate, protein, and sweet food consumption were assessed via a test meal 8-h following ATD. Four food items of comparable palatability were offered as a part of the test: two sweet (one carbohydrate-rich, and one protein-rich) and two non-sweet (one carbohydrate-rich, and one protein-rich). As compared to the placebo challenge, ATD significantly increased sweet calorie intake among overweight participants and increased their propensity to consume sweet food first before any other type of food. Lean participants’ sweet calorie intake and food preference were unaffected by ATD. Findings suggest serotonergic involvement in the sweet food consumption by overweight individuals.

© 2008 Elsevier Ltd. All rights reserved.

1. Introduction

Obesity, defined as a body mass index of 30 or greater, is a significant public health threat in the US, with 31% of the population affected (Flegal, Carroll, Ogden, & Johnson, 2002; Hedley et al., 2004). The growing obesity epidemic has been attributed to an environment where high-calorie, palatable foods are increasingly plentiful and easily accessible (Wadden, Brownell, & Foster, 2002). However, not all humans who are exposed to an abundance of highly palatable foods overeat or become overweight. Neurobiological variations may influence vulnerability towards overconsumption and weight dysregulation (Levitan et al., 2004).

Variation in brain serotonergic activity (5-hydroxytryptophan, 5-HT) has been implicated in appetite regulation and impulse control. Serotonin inhibits neuropeptide Y, resulting in suppression of hunger and food intake (Halford & Blundell, 2000). A chronic hypserotonergic state could result in a prolonged pattern of compulsive overeating, ultimately contributing to obesity. Supporting the role of 5-HT in weight regulation are data showing that stimulation of postsynaptic 5-HT receptors inhibits carbohydrate consumption in rats (Leibowitz, Weiss, & Shor-Posner, 1987; Shor-Posner, Grinker, Marinescu, Brown, & Leibowitz, 1986), with more vigorous stimulation reducing total calorie intake. Conversely, a low brain 5-HT level is associated with enhanced appetite (Leibowitz et al., 1987) and impaired impulse control in rats (Bizot, Le Bihan, Puech, Hamon, & Thiebot, 1999) and in humans at risk for alcoholism (Crean, Richards, & de Wit, 2002; LeMarquand, Benkelfat, Pihl, Palfour, & Young, 1999). Among obese humans, many medications that enhance serotonergic function induce weight loss (Hanotin, Thomas, Jones, Leutenegger, & Drouin, 1998; Pijl et al., 1991; Strain, Strain, & Zumoff, 1985; Toornvliet, Pijl, Hopman, Westendorp, & Meinders, 1997; Wadden et al., 2005), and reduce caloric intake (McTavish & Heel, 1992; Pijl et al., 1991; Wurtman et al., 1981) (although it should be noted that some serotonergic agents with prominent anticholinergic and antihistaminic action cause weight gain) (Ruetsch, Viala, Bardou, Martin, & Vacheron, 2005). Whether 5-HT plays a role in inhibitory control of eating in overweight individuals, specifically, has not been well studied.

ATD is a procedure that acutely and transiently reduces 5-HT synthesis. By 5–7 h following depletion, brain 5HT synthesis is reduced by 85–90%, and depressive symptoms increase in those vulnerable to MDD (Hood, Bell, & Nutt, 2005). Most research using ATD methodology has examined affective changes among those...
2. Methods and materials

2.2. Measures

2.2.1. Body mass index (BMI)

Body mass index was measured by weight and height using self-report. BMI was calculated by (weight in pounds/height in inches)²×704.5. Participants with BMI of 19–24.9 or less were classified as lean, and those 25 to 24.9 were classified as overweight.

2.2.2. Palatability scale

The palatability scale assessed liking for 70 total food items. Food items consisted of sweet and non-sweet foods presented in random order. Items were drawn from a large pool of foods previously rated as palatable by a normative sample (Spring, Pagoto, McChargue, Hedeker, & Werth, 2003). Participants were asked to rate how much they liked each food on a 1–10 point scale. Higher responses indicated greater palatability.

2.3. Procedures

2.3.1. Experimental session

Participants were given the ATD and placebo challenge drinks on different test days separated by one week. Mixtures were administered double-blind and in counterbalanced order. Female participants completed the study between days 7 and 21 of their menstrual cycle. Smokers within each BMI group (58% of each) smoked at hourly intervals during the test sessions to prevent nicotine withdrawal. On each test day, participants arrived by 8:00 a.m. after a 12-hour fast. Ad libitum intake preceded the 12-hour fast because dietary restrictions for more than 12 h preceding ATD do not appear to be necessary to produce significant reduction in plasma tryptophan (Spring et al., 2007). Participants underwent baseline testing of mood (Hamilton Depression Rating Scale (Hamilton, 1960) and blood collection through venipuncture to assess plasma amino acid levels (see Table 1 for experimental session timeline). At 9:30 a.m., participants ingested one of two calorie-matched beverages. The ATD mixture consisted of the following 15 amino acids (102.5 total g): l-alanine (5.5 g), l-arginine (4.9 g), l-cystine (2.7 g), glycine (3.2 g), l-histidine (3.2 g), l-isoleucine (8.0 g), l-leucine (13.5 g), l-lysine monohydrochloride (110.0 g), l-methionine (3.0 g), l-phenylalanine (5.7 g), l-proline (12.2 g), l-serine (6.9 g), l-threonine (6.9 g), l-tyrosine (6.9 g), and l-valine (8.9 g). Three amino acids, methionine, cystine, and arginine, were encapsulated because their unpalatable taste could not be masked by chocolate and peppermint. The remaining amino acids were mixed with 3 oz of tonic water and blended with crushed ice, chocolate syrup, and peppermint extract. The capsules were ingested approximately 15 min prior to the drink. In the placebo condition, participants ingested capsules containing confectioner’s sugar. Followed 15 min later by a mixture consisting of 3 oz of tonic water blended with crushed ice, chocolate syrup, and peppermint extract. Baking soda and psyllium were also added to the placebo beverage in order to mimic the granular, salty taste of the amino acid mixture.

All breakfast, lunch and snack foods were provided to participants on test days. The foods served comprised the low-tryptophan diet used elsewhere (i.e., Delgado et al., 1990). Breakfast consisted of puffed rice, peaches, nondairy creamer, Knox gelatin and sugar. Lunch

---

Table 1

<table>
<thead>
<tr>
<th>Time</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00 a.m.</td>
<td>Baseline testing of depressive symptoms (M-HAMD) and plasma amino acid levels</td>
</tr>
<tr>
<td>8:30 a.m.</td>
<td>Standardized breakfast (puffed rice, peaches, nondairy creamer, gelatin, and sugar)</td>
</tr>
<tr>
<td>9:30 a.m.</td>
<td>Consumption of amino acid or placebo mixture</td>
</tr>
<tr>
<td>10:30 a.m.</td>
<td>Four low-protein cookies and Kool-aid</td>
</tr>
<tr>
<td>12:30 p.m.</td>
<td>Lunch (graham crackers, cream cheese, boiled potatoes and butter, green beans, gelatin)</td>
</tr>
<tr>
<td>1:30 p.m.</td>
<td>All reading materials removed from observation room</td>
</tr>
<tr>
<td>2:30 p.m.</td>
<td>Following the guided imagery procedure, the M-HAMD assessment and blood sampling for amino acid levels were repeated</td>
</tr>
<tr>
<td>3:00 p.m.</td>
<td>Snack (4 low-protein cookies and Kool-aid)</td>
</tr>
<tr>
<td>5:30 p.m.</td>
<td>Dinner test meal</td>
</tr>
</tbody>
</table>
was graham crackers, cream cheese, boiled potatoes and butter, green beans and Knox gelatin. The mid-day snack consisted of four low-protein cookies and Kool-Aid. When not participating in experimental procedures, each participant sat alone in a private room in which he or she could watch videos or read magazines of emotionally neutral content.

At 5-hour post-challenge (2:30 p.m.), which marked the estimated onset of maximal tryptophan depletion (range 5–7 h; Delgado et al., 1990), participants underwent a 3-minute guided imagery negative mood induction procedure. Seated in a reclining chair in a soundproof room, each participant listened to a tape of a negative event script. Scripts were equated between conditions on vividness and emotional valence (anxious, angry, sadness).

Participants were encouraged to sit back, close their eyes, and visualize the event as vividly as possible. Negative autobiographical imagery was used to establish a depression-related cognitive set that would allow the biological state produced by ATD to be experienced and interpreted as genuine dysphoria. Blood sampling was then repeated to allow an assessment of change in amino acid levels.

After the 8-hour assessment period, participants were served the dinner test meal. Based on each participant’s palatability ratings, four food items of comparable palatability were offered: two sweet (one carbohydrate-rich, and one protein-rich) and two non-sweet (one carbohydrate-rich, and one protein-rich). A carbohydrate-rich food was defined as having a carbohydrate to protein ratio of 6:1 or greater. A protein-rich food was defined as having a carbohydrate to protein ratio less than 6:1. To ensure that all food items were comparable on palatability, only those with palatability of 9 or 10 were selected (see Table 2 for sample foods and classifications). Most items were prepackaged foods (i.e., frozen pizza, French fries, string cheese, candy bars, fruit ice cups). Other items such as baked chicken, nuts, gummy bears, and chocolate pudding were pre-portioned by the study dietitian. Two servings of each food item were pre-weighed and served. The order in which participants consumed each item was recorded by a research assistant. Leftovers were weighed, from which total grams consumed was calculated. Consumption in grams was later converted to calories for the analyses. Twenty participants from the parent study sample of 75 were not included in this study because, due to a miscoded food item, they inadvertently received food from only 3 of the 4 food categories. At the end of the session, participants were given a high protein drink (Sustical) and instructed to consume it the following morning.

2.4. Statistical analysis

Preliminary analyses compared lean and overweight participants on demographic characteristics, nausea in response to amino acid and placebo challenges, and changes in plasma amino acids from baseline to 5 h. The dependent variable for the primary analysis of consumption was operationalized in two ways (calorie intake and first choice food).

2.4.1. Calorie intake model

The first model evaluated total calorie intake. Square root transformations were performed to normalize calorie data, which was positively skewed. A multivariate analysis of variance (MANOVA) for repeated measures was implemented with depression history (positive, negative) and BMI (lean, overweight) as between-subjects factors and condition (placebo, ATD), sweetness (sweet, non-sweet), and food type (carbohydrate-rich/protein-poor versus protein-rich/carbohydrate poor) as within-subjects factors. Gender, smoking status (non-smoker, smoker), depressive symptom (HAM-D) response to ATD challenge, and condition order (ATD challenge first, placebo first) were entered as covariates.

2.4.2. First choice

The sequence in which the participant chose to consume foods was also measured. The item that a participant elected to consume first was conceptualized as the most immediately desired food. Two mixed effects logistic regression models estimated the frequencies of first choices under placebo and ATD conditions as a function of BMI group (overweight, lean). The outcome variable was dichotomous. First choice was equal to 0 for protein and 1 for carbohydrate in the first model, and then first choice was equal to 0 for non-sweet and 1 for sweet in the second model.

3. Results

3.1. Participant characteristics

Participants (N=55) were 55% female and had a mean age of 33.19 years (SD=10.85). The sample was 62% Caucasian, 24% African-American, 12% other, and 2% unknown. BMI ranged from 18.64 to 32.39. Thirty-six participants were lean (BMI between 18.5 and 24.9) and 19 were overweight (BMI between 30 and 34.9). Mean BMI was 22.82 (SD=1.67) for participants in the lean group (BMI<25) and 27.75 (SD=1.95) for participants in the overweight group. A total of 30 participants were smokers (21.8±10.4 cigarettes per day, 18.4±11.2 years). Almost half of participants (n=25; 45%) had a history of MDD, because individuals with a history of MDD were selectively recruited for the parent study where history of MDD was an independent variable.

Table 3 displays demographic characteristics of lean and overweight participants. No differences were observed between lean and overweight participants on demographic variables, MDD history, or smoking status. The ATD mixture did not appear to cause significant nausea, as evidenced by the lack of difference in Visual Analogue Scale scores of nausea between baseline and 5 h (p=.50). Scores were 6.9±13.4 at baseline and 10.2±20.0 at 5-h. The possible range of values was 0–100. No differences were observed between low and high BMI groups in either condition in Visual Analogue Scale scores of hunger between baseline and 5 h [PBO: t(48)=−.108, p=.914; AA: t(49)=−.323, p=.748]. Further, no differences were observed in Visual Analogue Scale scores of nausea between baseline and 5 h [PBO: t(48)=−.108, p=.914; AA: t(49)=−.323, p=.748].
between low and high BMI groups in either condition on change in tryptophan [PBO: t(32)=−1.148, p=.260; AA: t(32)=.344, p=.733] or the ratio of tryptophan to other LNAAs [PBO: t(32)=1.492, p=.145; AA: t(32)=.055, p=.956].

3.2. Caloric intake

The repeated measures MANOVA for total caloric intake revealed no significant interaction of condition by BMI, F(1,46)=.40, p=.52, indicating that the effect of ATD on caloric consumption did not differ between lean and overweight participants. The carbohydrate hypothesis, which suggested that ATD would affect carbohydrate consumption, was not supported, F(1,46)=.32, p=.86. The three-way interaction of condition by food type by BMI was also not significant, F(1,46)=.41, p=.52.

The sweetness hypothesis, which suggested that ATD would affect sweet calorie consumption was supported by a significant condition by sweetness interaction, F(1,46)=6.90, p=.01. Participants consumed on average 10.78 calories more of sweets during ATD compared to placebo. They also consumed 28.71 calories more of non-sweets during ATD compared to placebo. BMI moderated this effect, as revealed by a significant interaction between BMI and condition and sweetness, F(1,46)=7.23, p=.01. This significant interaction was followed by post-hoc paired comparisons, which were not significant for lean participants, t(33)=.95, p=.32, but significant for overweight participants, t(18)=−2.37, p=.02. Overweight participants increased their sweet calorie intake in ATD compared to placebo by an average of 91 calories (see Fig. 1). Although statistically non-significant, lean participants decreased their sweet intake in ATD compared to placebo by an average of 16.97 and 36.82 calories, respectively, in overweight participants by an average of 10.78 calories more of sweets during ATD compared to placebo. Lean individuals tended to decrease their sweet calorie intake in response to ATD. On average, overweight participants consumed 123 more calories of sweet-tasting food than their lean counterparts. We also found that overweight participants responded to ATD by choosing to eat sweet foods first, before consuming non-sweet foods. In contrast, lean participants responded to ATD in the opposite manner, that is, they more often chose to eat non-sweet foods first. These effects on sweet food calorie intake and preference persisted after adjusted for gender, MDD history, and any ATD associated depressive symptoms.

Results of the present study suggest that acutely lowering 5-HT has a selective influence on sweet food consumption. The present findings are consistent with previous research that links sweet, palatable food intake to 5-HT deficiency (Asin et al., 1992; Wogar et al., 1991). At first glance, results appear to be at variance with prior studies showing that indirect serotonergic agonists selectively reduce elevated intake of carbohydrates, independent of sweetness (Wurtman et al., 1993, 1981). However, since the high-carbohydrate snack foods often overconsumed by overweight dysphoric adults also tend to be sweet (Drewnowski, Kurth, Holden-Wiltse, & Saari, 1992), the discrepancy may be more apparent than real.

The behavioral pattern of reacting to 5-HT deficiency and emotional distress by increasing sweets intake resembles the clinical profile in atypical depression, which is often accompanied by overweight (Paykel, Parker, Rowan, Rao, & Taylor, 1983). Binge eating is at the extreme end on a continuum of emotionally-triggered eating that can vary considerably in frequency and energy content. In some

Table 4

<table>
<thead>
<tr>
<th></th>
<th>Lean (n=36)</th>
<th>Overweight (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>882.38 (492.24)</td>
<td>1041.43 (681.57)</td>
</tr>
<tr>
<td>ATD</td>
<td>886.26 (328.83)</td>
<td>1148.99 (738.54)</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>290.68 (214.18)</td>
<td>353.94 (245.92)</td>
</tr>
<tr>
<td>ATD</td>
<td>307.98 (203.16)</td>
<td>371.28 (277.20)</td>
</tr>
<tr>
<td>Protein</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>591.41 (448.35)</td>
<td>687.49 (611.02)</td>
</tr>
<tr>
<td>ATD</td>
<td>578.27 (305.13)</td>
<td>777.71 (773.82)</td>
</tr>
<tr>
<td>Sweet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>266.76 (202.85)</td>
<td>254.74 (256.19)</td>
</tr>
<tr>
<td>ATD</td>
<td>234.10 (133.97)</td>
<td>345.33 (286.95)</td>
</tr>
<tr>
<td>Non-sweet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>615.33 (395.77)</td>
<td>786.69 (563.64)</td>
</tr>
<tr>
<td>ATD</td>
<td>652.15 (301.87)</td>
<td>803.06 (561.62)</td>
</tr>
</tbody>
</table>

3.2.1. First choice

In the carbohydrate model, condition did not significantly interact with body mass index (z=.60, p=.54). In the sweet model, BMI and condition significantly interacted (z=2.15, p=.01). Among lean participants, ATD diminished the odds of sweet being selected as first choice. Among overweight participants, ATD increased the odds of a sweet being selected as first choice (see Fig. 2).

4. Discussion

Our study is the first to reveal differential intake among overweight and lean individuals in response to ATD. Overweight participants increased their sweet calorie intake in response to ATD relative to placebo. Lean individuals tended to decrease their sweet calorie intake in response to ATD. On average, overweight participants consumed 123 more calories of sweet-tasting food than their lean counterparts. We also found that overweight participants responded to ATD by choosing to eat sweet foods first, before consuming non-sweet foods. In contrast, lean participants responded to ATD in the opposite manner, that is, they more often chose to eat non-sweet foods first. These effects on sweet food calorie intake and preference persisted after adjusted for gender, MDD history, and any ATD associated depressive symptoms.

Fig. 1. Difference in sweet and non-sweet calorie intake between placebo and ATD.

Fig. 2. Change in number of participants making sweet and non-sweet first choices from placebo to ATD.
overweight and obese individuals the 5-HT neuronal system may be
dysregulated, similar to what is observed in bulimics (Kaye et al.,
2000). Jimerson and colleagues propose that binge eating increases
the plasma tryptophan to large neutral amino acid ratio, increasing
brain tryptophan availability and enhancing 5-HT synthesis (Jimerson,
Lesem, Kaye, & Brewerton, 1992). The current findings raise the
prospect that serotonin plays a role in the overeating patterns of
overweight and obese individuals similar to its involvement in bulimic
binge eating.

Intake of sweets has also been shown to release mesolimbic
dopamine (DA) in nonhumans (Avena & Hoebel, 2003; Salamone,
Cousins, McCullough, Carriero, & Berkowitz, 1994), not unlike drugs
of abuse. Serotonin neurons, in turn, inhibit mesolimbic DA neurons
(Alex & Pehek, 2007; Rothman & Baumann, 2006). Reduced 5-HT
responses. Nonhuman data show increased intake of sweet and fatty
foods after various stressors (Dallman et al., 2004; Dess, Choe, &
Minor, 1998), as well as an association with abdominal obesity
(Dallman, Pecoraro, & La Fleur, 2005). Our finding that ATD increased
overweight participants’ consumption of calories from sweets
suggests a need to elucidate common neurobiological mechanisms
whereby ATD and other stressors promote increased consumption of
sweets leading to overweight and obesity.

This study has several limitations. First, the range of BMI was
restricted by exclusion criteria. Differences between the lean and
overweight groups might have been greater if more obese individuals
(BMI≥35) had participated in the study. Those with class II or greater
obesity were excluded because it remains unclear whether the amino
d acid dosages used in the present ATD protocol are effective in more
obese individuals. In addition, self-reported measures of height and
weight were used instead of more objective measures. Second,
carbohydrate-rich foods were not purely carbohydrate in composition
and protein-rich foods were not purely protein, because single
nutrient foods rare in nature or in a typical diet. Carbohydrate-rich foods
were chosen to behave at least >6:1 carbohydrate to protein
because such foods have insufficient protein to block the insulin-
mediated rise in plasma tryptophan after carbohydrate intake
(Yokogoshi & Wurtman, 1986). A third limitation is that the first
food consumed might not have necessarily been the most immedi-
ately desired food. This assumption was based on findings that
palatable food cues trigger activation of brain regions that enhance
preferences for immediate over delayed rewards (Hariri et al., 2006;
Kelley, Schlitz, & Landry, 2005; McClure, Ericson, Laibson, Lowenstein,
& Cohen, 2007; McClure, Laibson, Loewenstein, & Cohen, 2004;
Tanaka et al., 2004), however other factors could have guided the first
food consumed. Finally, the sample size was estimated based on the
hypotheses of the parent study. The number of participants tested
may, therefore, may have been insufficient to detect differences in
food consumption across conditions, given that previously observed
effects of ATD on food intake have been modest (Oldman et al., 1995).
On the other hand, however, some studies using much smaller sample
sizes (N=20) did detect effects of ATD on food intake in recovered
bulimics (e.g., Weltzin et al., 1995). The present study was an initial
exploration into the effect of ATD on food intake in a non-eating
disordered population, larger studies are merited to further explore
this effect which will help elucidate the role of serotonin in “comfort
eating.”

In conclusion, the present study provides evidence of serotonergic
involvement in the food consumption of overweight individuals.
Acutely lowering serotonin synthesis by tryptophan depletion
heightened the intake of sweet-tasting foods by overweight indivi-
duals, regardless of gender, smoking status, history of MDD, or level of
depressive symptoms. Whether tryptophan depletion enhanced the
incentive salience of the rewarding food (Robinson & Berridge, 1993,
2000) or interfered with the ability to modulate food intake warrants
further investigation.

Acknowledgements

This study was supported by a K23 HL073381 to Sherry Pagoto,
PhD; an R01 HL59348, a VA Merit Review award to Bonnie Spring,
PhD; a K08 DA00467 to Dennis McGcharge; and a K08 DA017145 to
Brian Hitsman, PhD.

References

of dopamine neurotransmission. Pharmacology & Therapeutics, 11, 296–320.
Asin, K. E., Davis, J. D., & Bednark, L. (1992). Differential effects of serotonergic and
catecholaminergic drugs on ingestive behavior. Psychopharmacology (Berlin), 109,
415–421.
hyperactivity (cross-sensitization) and sugar hyperphagia. Pharmacology, Biochem-
y and Behavior, 74, 635–639.
to delay of reward in rats. Psychopharmacology, 146, 400–412.
behavior in men with or without a family history of alcoholism. Behavioral Brain
Research, 136, 349–357.
Dallman, M. F., La Fleur, S. E., Pecoraro, N. C., Gomez, F., Houshyar, H., & Akana, S. F.
(2004). Minireview: Glucocorticoids—Food intake, abdominal obesity, and
Dallman, M. F., Pecoraro, N. C., & La Fleur, S. E. (2005). Chronic stress and comfort foods:
(1990). Serotonin function and the mechanism of antidepressant action. Reversal of
antidepressant-induced remission by rapid depletion of plasma tryptophan.
Archives of General Psychiatry, 47, 411–418.
et al. (1994). Serotonin and the neurobiology of depression. Effects of tryptophan
depression in drug-free depressed patients. Archives of General Psychiatry, 51,
865–874.
energy food and sucrose treatment. Journal of Experimental Psychology: Animal
Behavior Processes, 24, 60–71.
human obesity: Carbohydrates versus fats. Appetite, 18, 207–221.
Flegal, K. M., Carroll, M. D., Ogden, C. L., & Johnson, C. L. (2002). Prevalence and trends in
288, 1723–1727.
appetite control. Annals of Medicine, 32, 222–232.
Psychiatry, 23, 56–62.
ramide and desfenfluramine in the treatment of obesity. Obesity Research, 6,
for immediate over delayed rewards is associated with magnitude of ventral striatal
Prevalence of overweight and obesity among US children, adolescents, and adults,