THE EFFECT OF SLEEP DEPRIVATION ON HEDONIC EATING

by

Danielle DePorter

A Thesis Submitted to the Faculty of the

DEPARTMENT OF NUTRITIONAL SCIENCES

In Partial Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

In the Graduate College

THE UNIVERSITY OF ARIZONA

2017

STATEMENT BY AUTHOR

The thesis titled *The Effect of Sleep Deprivation on Hedonic Eating* prepared by *Danielle DePorter* has been submitted in partial fulfillment of requirements for a master's degree at the University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this thesis are allowable without special permission, provided that an accurate acknowledgement of the source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the copyright holder.

SIGNED: Danielle DePorter

APPROVAL BY THESIS DIRECTOR

This thesis has been approved on the date shown below:

_______________________________ *05/10/2017*

Jennifer Teske, Ph.D. Date

Assistant Professor in Nutritional Sciences

ACKNOWLEDGEMENTS

Funding for this research was supported by a Career Development Award-level 2 (F7212W to JAT) from the United States Department of Veterans Affairs Rehabilitation Research and Development Service, the United States Department of Agriculture (ARZT-1360220-H23-150 and ARZT-1372540-R23-131 to JAT). The author's declare no conflicts of interest.

TABLE OF CONTENTS

ABSTRACT

Background: The high availability of palatable, calorie dense and nutrient poor foods promote hedonic eating, defined as the drive to eat to obtain pleasure in the absence of an energy deficit. Poor sleep and hedonic eating, independently, drive obesity at the societal level. However, it is unknown whether the combination of sleep loss and access to palatable food synergistically increases weight gain. Objective: The purpose was to test whether chronic partial sleep deprivation by a method that increases weight gain also increases hedonic eating and exacerbates weight gain in rodents. We hypothesized that 1) type of diet, 2) preference for the diets and 3) sex would moderate the effect of sleep loss on calorie intake and weight gain in sleep deprived rodents.

Subjects/Methods: Three-month old male and female Sprague-Dawley rats (n = 58 males and n = 10 females) were acclimated to hedonic diets for 7-d and then exposed to pre-recorded environmental noise (8h/d for 9-d). Body weight was measured every other day, unless mentioned otherwise. Food intake, corrected for uneaten food, was measured daily. Results: Noise exposure did not affect body weight gain and total calorie intake among male rats who had access to both a high and a low-fat diet. The effect of noise exposure on chocolate intake differed between male rats who were classified as high or low preference for chocolate. Initial preference for chocolate was greater in females compared to male rats. Conclusions: These results highlight the importance of preference and sex when investigating the effects of partial sleep deprivation on hedonic eating and obesity.

INTRODUCTION

The rise in the incidence of obesity parallels the increase in the incidence of sleep disorders (e.g. poor sleep quality, disrupted circadian rhythms and/or sleeping <7 hours per night continuously[1]). Epidemiological studies and meta-analyses indicate a greater risk for obesity with either short (<7 hours per night) or long (>9 hours per night) sleep duration[2-5]. Secondary to its impact on obesity, inadequate sleep also increases risk for chronic diseases and all-cause mortality[6-11]. This highlights the overall negative effect of poor sleep on health. While it is clear that demographic factors (e.g. sex, race, environmental, socio-economics)[12-13] and behaviors (e.g. eating, physical activity) influence the relationship between lack of sleep and obesity, it is unclear if these factors, when paired in concert, potentiate obesity induced by poor sleep.

The increased availability and convenience of highly palatable, calorie dense and nutrient poor foods promote hedonic eating defined as the drive to eat to obtain pleasure in the absence of an energy deficit[14]. Hedonic eating has increased overall calorie intake and the incidence of obesity at the societal level[15] and may potentiate weight gain due to poor sleep[16]. Clinical sleep studies showed that sleep restriction (i.e. 4-5.5-hours of time in bed) or total sleep deprivation increased calorie intake by ~182-559 kcal/d[17-23] or had no effect on food intake[24-25]. Moreover, sleep curtailment (i.e. 5.5-hour time in bed) increased calorie intake due to additional snacks, rather than meals relative to those that sleep normally[23]. It is still unclear if the combination of lack of sleep and hedonic food intake will further potentiate obesity due to greater calorie intake.

In parallel to clinical studies, data from rodent studies show a relationship between palatable diets and weight gain. When given the choice to consume purified high or low fat pelleted diets, rodents prefer to eat the high fat diet[26,27-29-31,49-51,54] and gain more weight[27-28,31,49-51] compared to rats that ate low fat diets. Like pelleted diets, rodents that readily consumed a cafeteria style diet gained significantly more weight when fed this hedonic diet compared to rats fed normal rodent chow[31-43], yet differences exist across species and age[32,38,39,41,48]. There is an inconsistent effect of gender on feeding and weight gain. Male and female rodents are hyperphagic when fed a hedonic diet[40,42,43] with an exception of one study[38]. Likewise, with weight gain, rats gain more weight when fed hedonic diets[38,40,42,43] with one exception[33]. Gender differences in response to the diet depend upon how weight gain is expressed (e.g. percentage vs. grams)[42-43]. Few have determined the relationship between sleep loss, hedonic feeding and weight gain[31,44,48,52,53], and it is unclear if the combination of sleep loss and access to a hedonic cafeteria diet promotes obesity more so than sleep loss alone. Previous studies have fed a pelleted high fat diet and deprived sleep with the multiple platform method. Deprivation of 96-h[45] or 21-d[44] caused weight loss while 73-d[44] of deprivation caused weight gain.

We have shown that chronic exposure to pre-recorded environmental noise reduces sleep, increases calorie intake and weight gain in male rats[46-47]. Acute exposure to noise reduces physical activity and energy expenditure immediately after exposure in male rats[47]. Thus, the response to sleep disruption in these rats mirrors the weight gain and hyperphagic response shown in humans. Therefore, the purpose here was to test whether sleep deprivation by a method that increases weight gain also increases hedonic food intake and exacerbates weight gain in rodents. We hypothesized that access to a hedonic diet during sleep deprivation would synergistically increase energy intake and weight gain and there would be a sex effect. To test this hypothesis, we manipulated sleep through exposure to environmental noise to determine whether 1) type of diet, 2) preference for the diet and 3) sex influenced the effects on body weight and food intake in Sprague-Dawley rats.

MATERIALS AND METHODS

Animals:

Three-month old male and female Sprague-Dawley rats (Charles River, Kingston, NY) (n = 58 males and n = 10 females) were housed individually in solid-bottom cages with a perforated floor with a 12h light/dark photocycle (lights on at 0700h for study 1, 0300h for study 2 and 0600h for study 3) in a temperature-controlled environment (21-22°C). Rodent chow (Harlan Teklad 8604, Harlan, Madison, WI, 3 kcal/g, 14% calories from fat, 53% calories from carbohydrate and 33% calories from protein), high fat diet (D12451 Research Diets, New Brunswick, NJ, 4.73 kcal/g, 45% calories from fat, 35% calories from carbohydrate and 20% calories from protein), and/or low fat diet (D12450B

Research Diets, New Brunswick, NJ, 3.85 kcal/g, 10% calories from fat, 70% calories from carbohydrate and 20% calories from protein) and water were allowed *ad libitum* except where noted. A separate set of rats was used for each study. Studies were approved by the Institutional Animal Care and Use Committee at the Minneapolis VA Health Care System, the University of Minnesota and the University of Arizona.

Sleep deprivation:

Rats were subjected to pre-recorded environmental noise (random street noises, vehicle horn, ambulance siren, hammering, sudden braking vehicle, bell, alarm, air plane sound etc) during the light cycle (8 h/day for 9 or 17 consecutive days) that has been validated to partially deprive sleep (i.e. reduce sleep and increase wake)[46]. The 15-minute recording was repeated for 8 hours daily during the light cycle. To prevent acclimation to the environmental noise, the noise events, duration of these events, frequency of sound (800 to 20000 Hz), amplitude (85 to 100 dB, average global intensity was 85 db) and inter-noise interval were randomly distributed. The events also consisted of periods of silence followed by sharp attack rate noises randomly distributed in the sound sequence. This amplitude and frequency has been shown previously to produce no damage to the rodent cochlea[56-57]. Since the rodent audiogram is different from the human audiogram, we used high-frequency sounds to match the rat audiogram. This adaptation was done using the audio software, Audacity (open source software), to translate frequencies between the human and rodent audiogram. Sound samples were selected from the Best Service Studio

Box DVD3-Technical sample library (Best Service GmbH, Munchen, Germany) and were randomly arranged using Audacity (audio editor for recording, slicing and mixing audio clips) into a single 15-minute track.

Experimental Design:

Study 1. Effect of sleep deprivation on the consumption of pelleted high fat (HF) and low fat (LF) diets.

Twenty-four 8-week old male rats had *ad libitum* access to a high and low fat diet simultaneously for a 7-d acclimation period. During this time period, food intake and food spillage (i.e uneaten food particles) were measured daily and body weight was measured on days 1, 2, 3, 5 and 7. Body composition was measured (EchoMRI-900TM, Houston, TX) and then rats were randomized based on percent fat mass relative to body mass into one of two groups: control (sleep/wake *ad libitum*) or treatment (environmental noise exposure for 8h/d during the light cycle). During this 9-d treatment period, rats continued to have *ad libitum* access to both the high and low fat diets while body weight, food intake and food spillage were measured daily. Body composition was measured after the 9-d treatment period. Rats were euthanized by decapitation. Transcardial blood was obtained and centrifuged at 2000rpm for 20 minutes at 4°C to obtain plasma. The following tissues were collected after euthanasia: perirenal white adipose tissue, interscapular brown adipose tissue, gastrocnemius muscle, liver, rostral and caudal lateral hypothalamus, locus coeruleus, dorsal raphe, ventrolateral preoptic area and the hypothalamic paraventricular nucleus. Tissues were frozen immediately in liquid nitrogen following excision and were stored at -80°C for future analysis. Six of the 24 animals were excluded due to incomplete measurements and thus data analysis was completed on 18 rats.

Study 2. Effect of sleep deprivation on the consumption of chocolate.

Twenty-four 8-week old male rats were given a pre-determined amount of chocolate daily for 2-hours[76] (5g, 4.88 kcal/g, Hershey's Chocolate Kisses, Hershey, PA, 1200-1400h) in addition to rodent chow for 7-d (i.e. acclimation period). Rats had chow *ad libitum*. Following a body composition measurement (EchoMRI-900TM, Houston, TX), rats were randomized based on percent fat mass relative to body mass into one of two groups: control (sleep/wake *ad libitum*) or treatment (environmental noise exposure for 8h/d during the light cycle). During the 9-d treatment period, access to rodent chow *ad libitum* and chocolate for 2hours daily was identical to the acclimation period. Food intake, food spillage and body weight were measured daily. Calorie intake (i.e. 2-hour chow and chocolate and 22-hour chow) was determined daily. Calorie intake was corrected for spillage (i.e. uneaten food particles) and dehydration. After the treatment period, body composition was measured prior to euthanasia by decapitation. Transcardial blood and tissues were obtained and processed as described in study 1.

Study 3. Effect of sex on the consumption of chocolate during sleep deprivation.

Rats (n = 10 males and n = 8 females) were given access to a pre-determined amount of chocolate for 2h as described in study 2 for a 7-d acclimation period in addition to rodent chow *ad libitum*. Following the acclimation period, physical activity (TSE Systems, Chesterfield, MO) was measured which started at 1500h. Rats were then randomized based on cumulative distance during the baseline 23.5h physical activity test within sex into one of two conditions: control (sleep/wake *ad libitum,* n = 5 males and n = 4 females) or treatment (environmental noise exposure for 8h/d during the light cycle for 17-d, n = 5 males and n = 4 females), which created four groups. During the 17-d treatment period, rats continued to have chocolate and chow as described above. On day 18, rats were euthanized by decapitation, transcardial blood was collected and processed as described above. The following tissues were dissected: whole brain, liver, perirenal white adipose tissue and bicep femoris muscle. Tissues were placed in liquid nitrogen after dissection and stored at -80°C until analysis. Two females (1 control and 1 noise) were euthanized prior to the 7-d acclimation period due to surgical complications. Body weight was measured every other day during the study. Calorie intake (i.e. 2-hour chow and chocolate and 22-hour chow) was determined daily. Calorie intake was corrected for spillage (i.e. uneaten food particles) and dehydration.

<u>Statistical Analysis:</u>

Data were analyzed with unpaired t-test, two factor ANOVA or two factor repeated-measures ANOVA as appropriate followed by Fisher's multiple comparisons test where appropriate, to determine differences between individual means (GraphPad Prism version 7.02, La Jolla, CA). A separate analysis was completed for each time period (i.e. acclimation and treatment), each diet (high fat vs. low fat; chow vs. chocolate) and sex (male vs. female). An α level of 0.05 was used for all statistical tests. Data are expressed as mean ± SEM.

RESULTS

Study 1. Effect of sleep deprivation on the consumption of pelleted high fat (HF) and low fat (LF) diets

Weight gain: There was a main effect of time ($F_{(4, 64)} = 112.9$, $P < 0.0001$) during the 7-d acclimation period only (Table 1, Fig. 1A). Rats gained significantly more weight on day seven and day 17 compared to day one for both treatment groups (Fig. 1A, $P < 0.0001$ for both days). Although rats gained weight on each measurement day during the study, total weight gain during the acclimation and treatment periods was similar between treatment groups (Table 2).

Calorie intake: For 24-h calorie intake, there was a main effect of time during the 7-d acclimation period only ($F_{(6, 96)} = 9.11$, $P < 0.0001$, Table 1 and Fig. 1B). Daily calorie intake declined steadily during the acclimation period but was maintained during the treatment period at a level normal for rats at this age. Thus, relative to day one, calorie intake was significantly lower on day seven during the acclimation period (control: $P < 0.0001$ and noise: $P = 0.0002$) and day 17 during the treatment period ($P < 0.0001$ for both groups, Fig. 1B). Overall, total calorie intake was similar between treatment groups for both acclimation and treatment periods (Table 2). Finally, rats that were later randomized to the noise exposure group ate significantly more calories on days two and five compared to the control group ($P = 0.0073$ and $P = 0.0119$, respectively, Fig. 1B).

Rats in both groups consumed significantly more calories from the HF diet compared to the LF diet during the acclimation and treatment periods on each day during the study ($P < 0.0001$ for all groups, Fig. 1C, 1D and 1E). There was a main effect of time during the acclimation and treatment periods ($F_{(1, 32)} = 96.16$, $P < 0.0001$ and $F_{(1, 32)} = 163.30$, $P < 0.0001$, respectively, Table 1). However, when diets were analyzed individually to determine the effect of treatment across time within each diet, there was no effect of treatment and the interaction between treatment and time was not significant for both diets and time periods. There was a main effect of time for the HF diet during the acclimation period only ($F_{(6, 96)} = 6.65$, $P < 0.0001$, Table 1). Calorie intake from the HF diet for both groups was significantly lower on days seven and 17 compared to day one (day 7: control: $P < 0.0001$ and noise: $P = 0.0205$, day 17: $P < 0.0001$ for both groups, Fig. 1C). Calorie intake from the LF diet was significantly lower on day 17 compared to day one in the noise treatment group only ($P = 0.0353$, Fig. 1C). Overall, calorie intake from the HF and LF diets during the study periods were not significantly different across treatments (Table 2).

<u>Study 2. Effect of sleep deprivation on the consumption of chocolate.</u>

<u>Weight gain</u>: There was a main effect of time ($F_{(6, 132)} = 2.43$, $P = 0.0290$) during the 7-d acclimation period only and a significant interaction between treatment and time ($F_{(8,$

$_{176)}$ = 2.15, P = 0.0331) during the treatment period only (Supplementary Table 1 and 2 and Supplementary Fig. 1). Cumulative weight gain during the acclimation and 9-d treatment periods was similar between groups (Supplementary Table 2 and Supplementary Fig. 1A). Rats in the control group had gained significantly more weight by days nine and 12 compared to those that were exposed to noise (P = 0.0079 and P = 0.0076, respectively, Supplementary Fig. 1A).

Calorie intake: During the acclimation period, there was a main effect of time for each calorie intake endpoint and there was a significant interaction between treatment and time on 2-h chow intake for the acclimation period only (Supplementary Table 1). During the treatment period, there was a significant interaction between time and treatment on each calorie intake endpoint and a main effect of time on each calorie intake endpoint except for chocolate intake (Supplementary Table 1). Overall, 24-h calorie intake, 24-h chow, 22-h chow and chocolate intake during the acclimation and treatment period was similar between treatment groups with one exception. During the treatment period, noise-exposed rats ate significantly fewer calories from the 2-h chow compared to the control group (P = 0.0010, Supplementary Table 2).

We considered preference for chocolate intake, which was defined as the percentage of calories from chocolate relative to the total calories consumed during the 2-h exposure, to test the effect of noise exposure on calorie intake (Table 3). There was a main effect of treatment on 2-h chow and the percentage of calories from chocolate for both high and low preference rats only. There was a main effect of time on the percentage of calories from chocolate for both high and low preference rats, 22-h chow in low preference rats and total calorie intake in high preference rats. The interaction was significant for chocolate for high preference rats, the percentage of calories from chocolate in low preference rats and total calorie intake for low preference rats. Finally, the interaction was significant for both high and low preference rats for chow intake (24-h and 22-h, Table 3).

Rats in the treatment group maintained their daily calorie intake during the treatment period but rats in the control group ate significantly fewer calories on the final day of the treatment period compared to the first day independent of preference (Fig. 2A and 2B). By definition, rats that had a high preference for chocolate ate more chocolate than those with low preference (Fig. 2C and 2D). Regardless of preference, there was no effect of treatment and both groups maintained a relatively high or low intake of chocolate based on their defined preference. At the end of the treatment period, among the rats with a low preference for chocolate, those in the control group ate significantly more chocolate than the noise-exposed group. In contrast, among rats with a low preference for chocolate, those who were noise-exposed decreased their 2-h chow intake, while the control group maintained their intake (Fig. 2D). Independent of preference, the treatment group ate a significantly greater percentage of calories from chocolate at the beginning of the treatment period compared to the control group and maintained their high intake during the treatment period (Fig. 2E and 2F). However, the control group steadily increased chocolate intake independent of preference, such that the control group ate a significantly greater percentage of calories from chocolate on the final day of the treatment period relative to the first day (Fig. 2E and 2F). For rats with a high preference for chocolate, the treatment group maintained chow intake (24-h and 22-h) but the control group ate significantly fewer calories on the final day of the treatment period compared to the first day (Fig. 2G and 2I). In contrast, for those rats that had a low preference for chocolate, the treatment group ate significantly more chow (24-h and 22-h) while the control group ate significantly less chow on the final day of the treatment period compared to the first day (Fig. 2H and 2J).

Study 3. Effect of sex on the consumption of chocolate during sleep deprivation.

Weight gain and calorie intake data for males and females were analyzed separately and then the effect of the treatment across time was determined (Supplementary Tables 3 and 4).

Weight gain: There was a main effect of treatment on weight gain for females during the treatment period only ($F_{(1, 6)}$ = 10.05, P = 0.0193, Supplementary Table 3) and a main effect of time on weight gain for males during the acclimation period only ($F_{(3, 24)}$ = 3.90, P = 0.0212, Supplementary Table 4).

Calorie intake: During the acclimation and treatment periods, there was a main effect of time for each calorie intake endpoint for the males except for 2-h chow. There was also a main effect of time in females for the chocolate during the acclimation period and the treatment period for 2-h chow, 22-h chow and 24-h chow calorie intake

(Supplementary Table 3 and 4). There was a significant interaction between time and treatment for 2-h chow during the acclimation period for females. During the treatment period, there was a significant interaction for males for daily calorie intake as well as chow intake (24-h, 22-h, and 2-h) and for females for 24-h chow intake (Supplementary Table 3 and 4). Finally, there was a main effect of treatment during the treatment period for 2-h chow intake for males (Supplementary 4).

During the treatment period, females had similar 24-h calorie intake across treatments while males, independent of treatment, significantly increased calorie intake (control: $P = 0.0274$; and noise: $P < 0.0001$, Fig. 3I and 3J). Female noise-exposed rats significantly increased total calorie intake on the final day of treatment compared to the control group ($P = 0.0065$). For chow intake (24-h and 22-h) during the treatment period, only male noise-exposed rats significantly decreased their 24-h chow intake ($P = 0.0004$), but in contrast, significantly increased their 22-h chow intake ($P < 0.0001$) while female noise-exposed rats ate significantly more 22-h chow on the last day of treatment compared to the control group ($P = 0.0065$, Fig. 3E, 3F, 3G and 3H). During the acclimation period and regardless of sex, both control and noise-exposed rats significantly increased their chocolate intake. Moreover, for the remainder of the treatment period, females maintained chocolate intake while males continued to increase their intake of chocolate (Fig. 3C and 3D). Independent of treatment or sex, rats ate significantly more chocolate on days seven (male: control: $P = 0.0139$ and noise: $P = 0.0002$; and females: control: $P = 0.0432$ and noise: $P = 0.0003$) and 17 compared to day one (male: $P < 0.0001$ for both groups and females: control: $P = 0.0104$ and noise: $P = 0.0027$, Fig. 3C and 3D). The calories from 2-h chow remained low relative to the chocolate regardless of sex; however, both treatment groups within the females only significantly increased their calories from 2-h chow during the treatment period ($P = 0.0057$ and $P = 0.0019$, respectively, Fig. 3C and 3D). Although male rats did not increase 2-h chow intake during the treatment period, there were several days where males in the control group ate significantly more calories than the noise-exposed group (Fig. 3D).

DISCUSSION

We have previously reported shown that chronic partial sleep deprivation due to pre-recorded environmental noise exposure causes weight gain and hyperphagia in male rats[46]. This was the first rodent model of sleep loss that mirrored the relationship between sleep loss and obesity reported in epidemiological[2-5,58] and clinical studies[17,20,21,23]. Because other rodent methods of sleep deprivation (e.g. disk-over-water, "flower-pot" or "platform" method, gentle handling and forced physical activity) cause weight loss, skin lesions, hypothermia, stress and can result in death[60-65], here we chose to use noise exposure to reduce sleep[46-47]. We hypothesized that access to high energy dense foods would exacerbate calorie intake and weight gain in sleep deprived rodents. We also tested whether the types of diet and sex influenced weight gain and feeding due to noise exposure. In these results, we show that environmental noise exposure did not affect body weight gain and food intake in male or female rats despite access to energy dense foods and that preference and sex moderate the effects of sleep loss on hedonic eating.

We tested whether type of diet influenced feeding since intake of a hedonic diet differs based on the constituents within the diet. A "hedonic diet" may contain a variety of human snacks presented in a random order, a single palatable human food (e.g. chocolate) presented with rodent chow or a diet high in a single macronutrient with homogenous texture (e.g. pelleted-high fat diet)[14-15]. Rodents that sleep *ad libitum* are typically hyperphagic on high fat diets (e.g. pelleted high fat, lard, olive oil, coconut fat, fish oil)[26,29,30,66,67]. Likewise, during the acclimation period in our study, rats were hyperphagic. Hyperphagia was due to greater consumption of a high fat diet compared to the low-fat diet given daily (Fig. 1B, Table 1). Limited access to chocolate caused hyperphagia in male rats because intake from chocolate steadily increased over time and chow intake increased when the rats did not have access to chocolate (Supplementary Fig. 1B and 1C).

During the treatment, feeding declined and then was sustained at a level that would be expected to maintain normal body weight (Fig. 1B). Lack of hyperphagia while feeding these palatable diets contrasts some who used sleep loss methods that cause weight loss[44,45,65,68-72] with few exceptions[66,68]. For example, high fat-fed rats subjected to circadian desynchronization were hyperphagic but did not lose weight[66]. In addition, sleep deprived rodents fed a liquid high fat diet for 10-d had delayed hyperphagia (days

6-10) and maintained body weight[74]. Thus, based on our prior work[46-47] and the former studies we expected noise to stimulate total calorie intake. It is unclear why feeding declined or why feeding was similar between treatments but may be related to differences in methodology between studies. These may include the method used to deprive sleep, type of diet, duration of sleep deprivation, species, housing conditions and age[32]. It is plausible that we needed to test more animals based on the high variability in the weight gain response to other hedonic diets. Also, despite the palatability of the pelleted high fat diet due to high fat and sugar content, this diet does not model the heterogenous texture and variety of foods in a human diet since rats fed a cafeteria diet with 10 food options maintained hyperphagia for 10-20-d[40]. Clinical sleep disruption studies report increased food intake from a hedonic diet[18-24,75,77-78] with a few exceptions[24,25,79,80] where access to hedonic diet was limited[79]. Finally, preference for hedonic food and sex (male vs. female) may have contributed to the results, which will be discussed below.

Type of diet also influences weight gain during sleep manipulation. Rodents lose weight when sleep deprived by highly stressful methods regardless if they have access to a low or high energy diet[44,45,65,68-72]. Sleep deprivation caused a 18-22% reduction in weight from baseline[81] but the magnitude of weight loss varies based on the method used to deprive sleep[63]. We expected greater weight gain in noise-exposed rodents regardless of diet since others report weight gain after exposure to high energy dense foods[32-40,42,43,82] and our sleep deprivation method increases weight gain[46]. Here, weight gain was similar between treatments, which likely reflects similar total calorie intake (Fig. 1A-B and Supplementary Fig. 1A-B). To address the possibility that limited access to a palatable food would modify the effect of sleep loss on hedonic food intake, we provided chocolate for 2-h daily based on its high palatability and use in rodent binge eating studies[76,83]. We hypothesized that noise would cause weight gain since rats should binge eat the chocolate and increase chow intake in the absence of the chocolate. Despite that weight gain was unaffected by noise exposure (Supplementary Fig. 1A), overall chocolate consumption plateaued when male rats were exposed to noise but did not cause hyperphagia (Supplementary Fig. 1B-C). It is plausible that access to chocolate for only 2-h daily may have caused a “ceiling effect” such that rats were unable to physically consume more chocolate. Providing access to chocolate for a longer time or at multiple random times to prevent anticipation may have been required. Like high fat pelleted diets, rats may have become less interested in the chocolate over time, which suggests that a variety of randomly presented palatable food items (e.g. cafeteria diet) may have been more efficacious.

We determined whether preference for chocolate moderated the effect of noise on weight gain and feeding since preference influences these endpoints in rats that sleep undisturbed[84-86]. After rats were classified as high or low preference for chocolate, food intake was different between control and noise-expsosed groups but was dependent on preference. Non-noise exposed rats, regardless of high or low preference, reduced overall calorie intake despite that they maintained intake of chocolate. This overall reduction was due to the reduction in chow intake when rats did not have access to chocolate despite that the high preference rats maintained chow intake and the low preference rats reduced chow intake during the 2-h period. The latter explains the increase in the percent of calories from chocolate and is likely why chocolate is used in binge eating studies. For noise-exposed rats, high preference rats maintained chocolate and chow intake (2h and 22h, Fig. 2C,G,I). Low preference noise exposed rats non-significantly reduced chocolate intake and increased chow intake regardless of the presence of chocolate, which resulted in a maintenance of total calories (Fig. 2D,H,J). Thus, if we had the statistical power to include preference in the data analysis or stratify rats by preference, we may have observed an interaction between preference and treatment or a significant effect of noise with an extended treatment period.

We tested whether sex modified the effect of noise exposure on chocolate intake since rates of obesity[87-88] and sleep disorders are sex-dependent[20,38] with some exceptions[21]. Our data suggests that females prefer chocolate more than males since females initially consumed more chocolate than males during acclimation. In contrast, during noise exposure, chocolate intake among females plateaued while intake among males steadily increased such that intake was the same between sexes at the end of the treatment (Fig. 3C-D). The apparent sex-dependent preference for chocolate agrees with others[89-91] that showed females had greater preference for saccharin compared to the males[90]. While sex differences exist for food intake and weight gain in response to a hedonic diet among animals that slept undisturbed[40,42,38], no rodent study has tested whether sex influences the effect of sleep loss on hedonic eating. A prior study showed that despite that female rats ate more calories from cafeteria diet than males, female cafeteria diet fed rats gained more weight than female controls while cafeteria diet fed males lost weight compared to male controls[38]. Here regardless of treatment, females maintained while males had a variable increase in total calorie intake, which was related to husbandry procedures (Fig. 3I-J). Finally, the lack of a treatment effect on weight gain is likely due to the similar total calorie intake during the treatment phase within males and females (Fig. 3B).

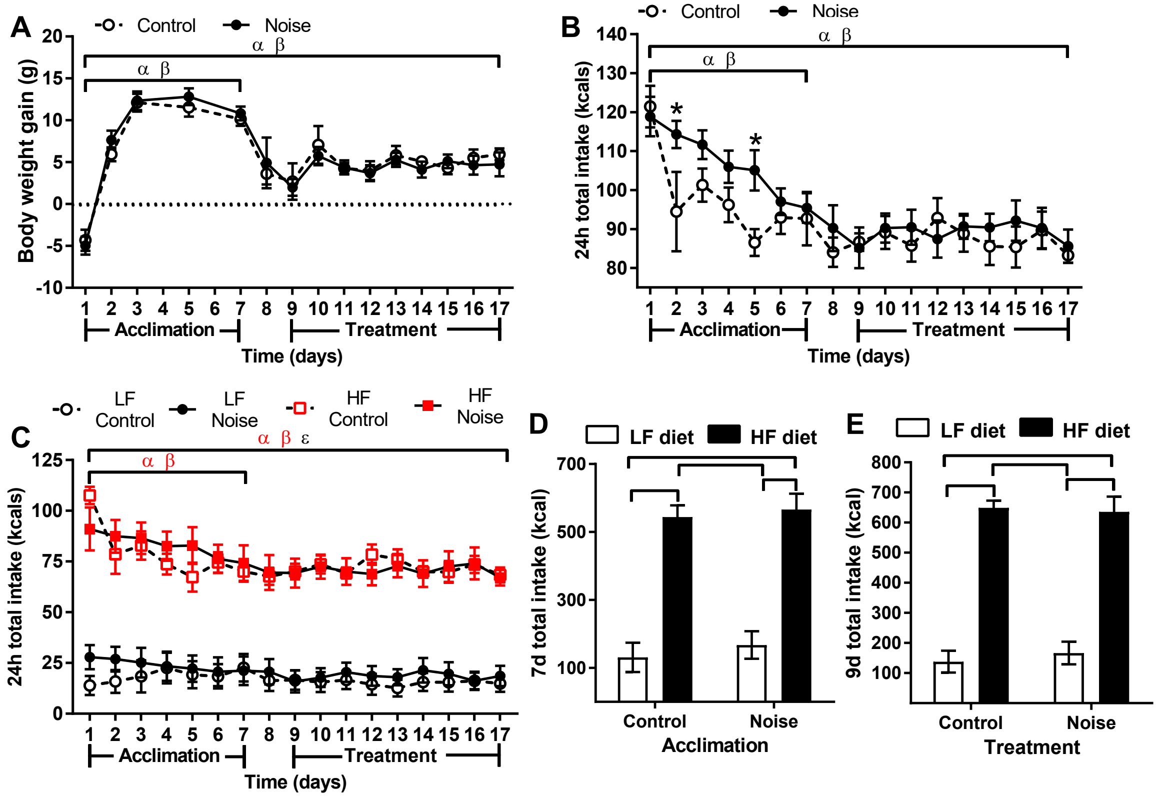
Effects of sleep loss in prior studies have been attributed to stress indicated by elevated plasma norepinephrine, adrenocorticotropic hormone[92] and corticosterone and cortisol[25] as well as reduced body temperature[20,25,60-62,92]. The combined effect of sleep loss and stress decreases food intake during acute exposure but increases food intake with chronic exposure[93]. Thus, it is unclear if sleep loss or the stress inherent to the method of sleep deprivation caused the change in food intake. Our non-published data shows that chronic noise exposure non-significantly increases corticosterone, thus it is unclear why feeding did not parallel this increase in corticosterone since we report hyperphagia in our published work[46]. Our noise exposure may be less stressful since it minimizes thermal regulation, handling and general stress inherent to the methods that deprive sleep with water, forced locomotion or gentle handling, respectively[46,93].

We acknowledge several limitations. First, in the study with males and females, rats had noise-induced sleep loss immediately after surgery and a wash out period prior to chocolate exposure. The stress inherent to sleep loss during post-surgical recovery likely affected feeding and weight gain for both sexes. Second, despite our prior reports showing noise-induced sleep loss with low physical activity or energy expenditure, we did not measure sleep and thus we did not validate that noise reduced sleep nor measure physical activity or energy expenditure[93]. Thus, similar weight gain between noise and non-exposed rats may be due to reductions in physical activity or energy expenditure. Third, we did not consider preference for pelleted high fat diet or the effect of low animal numbers on sex in the final study. It is plausible that rats did not consume more high fat diet in response to noise because they had a low preference for this diet. Finally, we did not account for the stimulatory effect of caffeine in the chocolate on locomotor activity or food intake. Caffeine (i.e. 5-20mg/kg, i.p. injection) increased locomotion but caffeine in drinking water for 40-d did not affect body weight or chow intake in rats[94]. The latter may be due to sex-specific effects of caffeine on the consumption of palatable foods[95].

In conclusion, we show that the type of hedonic diet and preference for chocolate influences the effect of environmental noise exposure on body weight gain and total food intake and that sex may mediate consumption of chocolate in response to noise exposure. Identifying preference for hedonic food items before noise-induced sleep deprivation and addressing potential sex-specific differences in hedonic food intake would be important for future studies seeking to delineate whether sleep loss exacerbates hedonic eating and weight gain.

FIGURES

Figure 1: Daily body weight gain (A) and daily food intake (B) during the 7-day acclimation and 9-day treatment period for male rats that slept ad libitum (open symbols) or were noise-exposed (filled symbols, 8-h/d for nine days) when fed a low fat (LF) and high fat (HF) diet ad libitum (C). Cumulative calorie intake for each diet is shown during the acclimation (D) and treatment (E) period. Data were analyzed with 2 factor ANOVA or 2 factor repeated measures ANOVA followed by Fisher's multiple comparisons test for each time period. Data expressed as mean ± SEM. N = 18 (control: n = 9 and treatment: n = 9). α and β $P < 0.05$ for the control and noise treatment group, respectively, for days one versus seven and one versus 17. ε $P < 0.05$ for LF-noise treatment group for days one versus 17. * $P < 0.05$ for control versus noise on specific days. Brackets in panels D and E, $P < 0.0001$, for comparisons between diets and treatment groups.



A
Control
Noise
α β
Body weight gain (g)
Acclimation
Treatment
Time (days)
B
24h total intake (kcals)
C
LF Control
LF Noise
HF Control
HF Noise
α β ε
D
LF diet
HF diet
7d total intake (kcal)
Acclimation
E
9d total intake (kcal)

Figure 2: Male rats, classified as high and low preference for chocolate slept ad libitum (open symbols) or were noise-exposed (filled symbols, 8-h/d for nine days) during the 9-day treatment period. Total calorie intake (A and B) and calorie intake from chow and chocolate during 2-hour exposure to chocolate (red squares) and chow (black circles) (C and D), percentage of calories from chocolate during that 2-h exposure (E and F), 22-h chow (G and H) and 24-h chow (I and J). Data were analyzed with 2 factor repeated measures ANOVA followed by Fisher's multiple comparisons test for each time period. Data expressed as mean ± SEM. For high preference animals: N = 17 (control: n = 8 and treatment: n = 9) and for low preference animals: N = 7 (control: n = 4 and treatment: n = 3). α and β $P < 0.05$ for the control and treatment groups, respectively for days nine versus 17. * $P < 0.05$ for control versus noise on specific days.

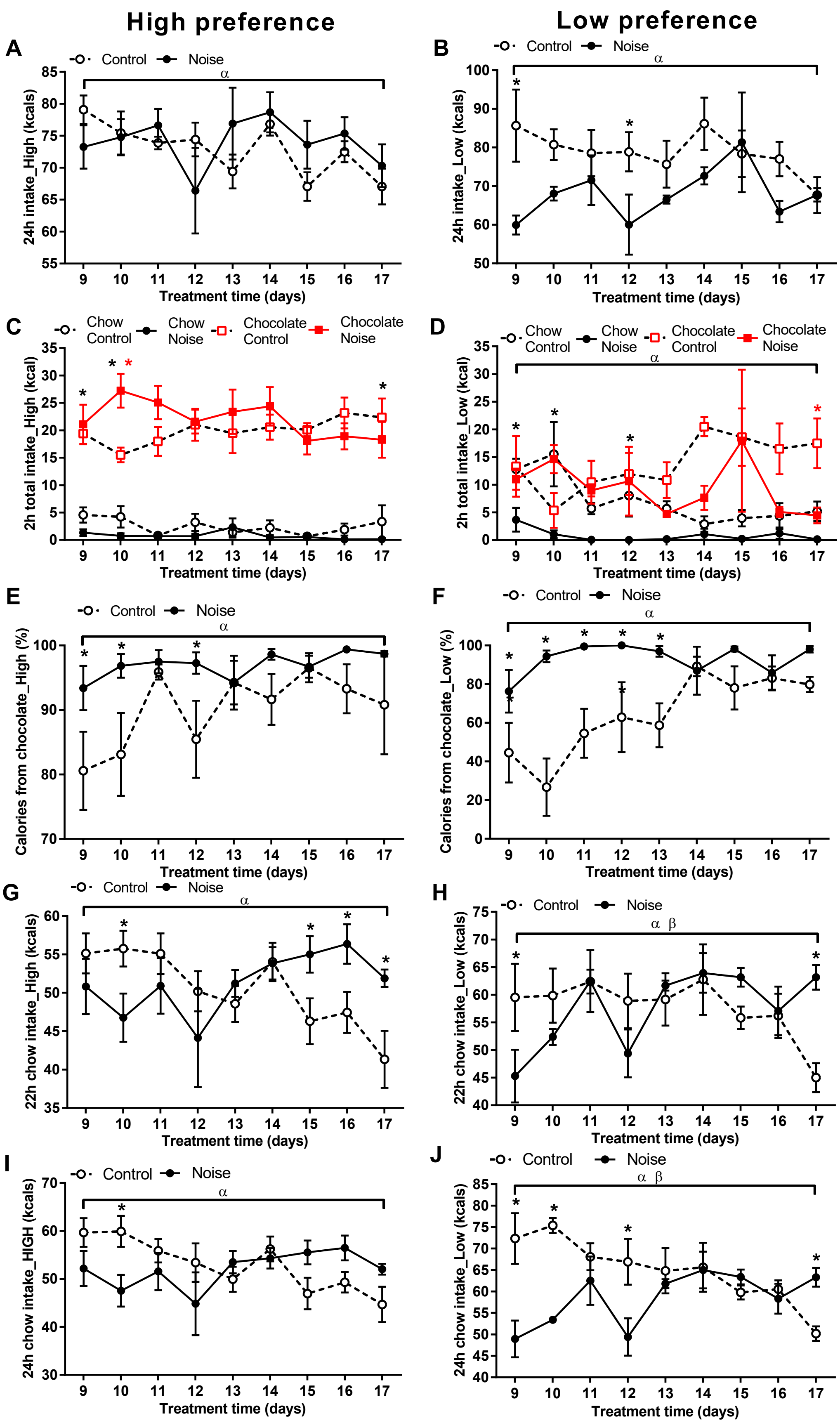
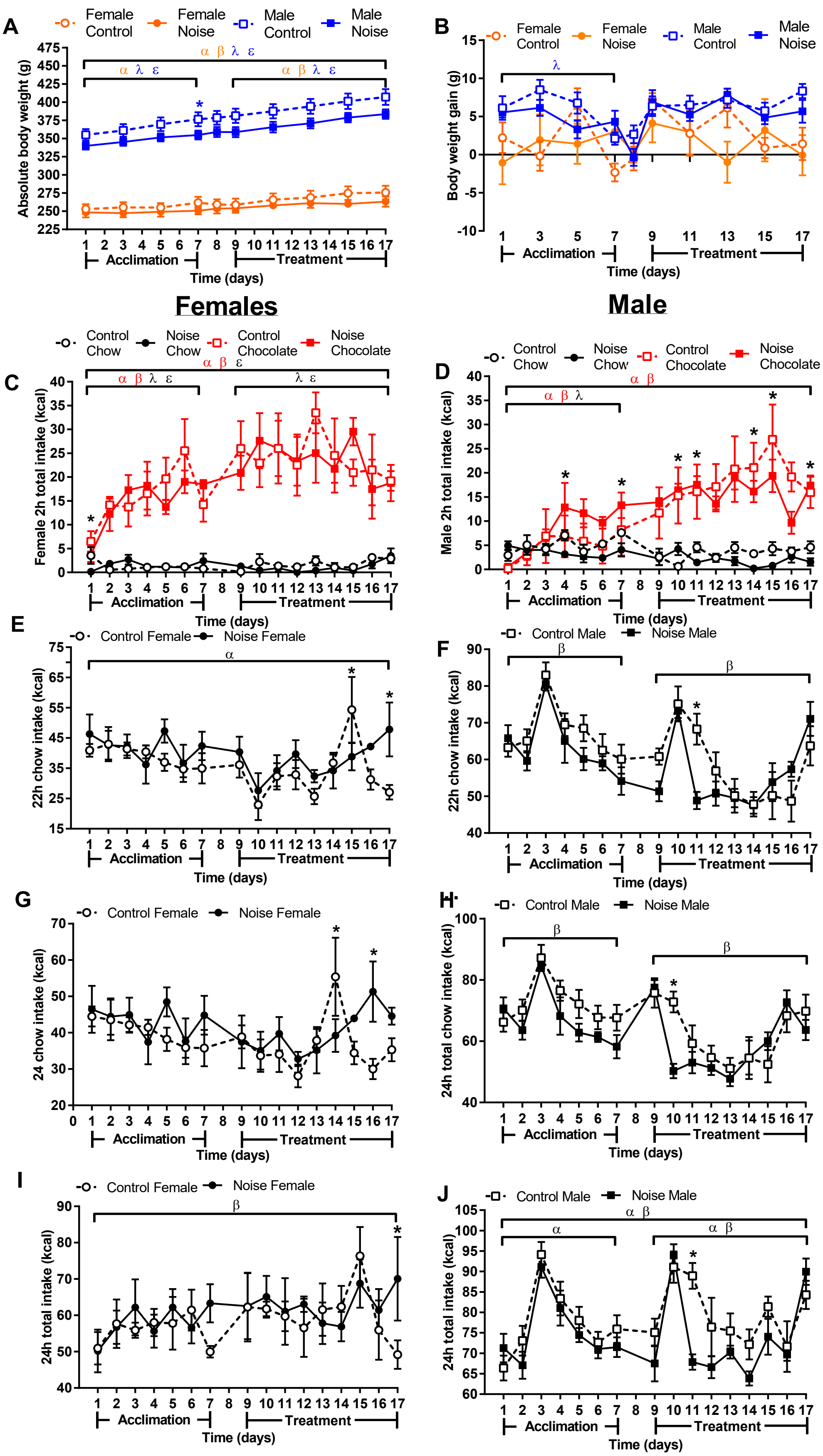

High preference
Low preference
A
Control
Noise
α
24h intake_High (kcals)
Treatment time (days)
B
Control
Noise
α
24h intake_Low (kcals)
Treatment time (days)
C
Chow Control
Chow Noise
Chocolate Control
Chocolate Noise
2h total intake_High (kcal)
Treatment time (days)
D
Chow Control
Chow Noise
Chocolate Control
Chocolate Noise
α
2h total intake_Low (kcal)
Treatment time (days)
E
Control
Noise
α
Calories from chocolate_High (%)
Treatment time (days)
F
Control
Noise
α
Calories from chocolate_Low (%)
Treatment time (days)
G
Control
Noise
α
22h chow intake_High (kcals)
Treatment time (days)
H
Control
Noise
α β
22h chow intake_Low (kcals)
Treatment time (days)
I
Control
Noise
α
24h chow intake_HIGH (kcals)
Treatment time (days)
J
Control
Noise
α β
24h chow intake_Low (kcals)
Treatment time (days)

Figure 3: Absolute body weight (A) and body weight gain (B) for males (squares) and females (circles) during the 7-day acclimation and 9-day treatment period for rats that slept ad libitum (open symbols) or were noise-exposed (filled symbols, 8-h/d for nine days) and had rodent chow ad libitum and a daily 2-hour exposure to chocolate (C and D). Calorie intake from 22-h chow (E and F), 24-h chow (G and H) and total calories (I and J). Data were analyzed with 2 factor repeated measures ANOVA followed by Fisher's multiple comparisons test for each time period. Data expressed as mean ± SEM. N = 18 (control: n = 5 males and 4 females and treatment: n = 5 males and 4 females). α and β P < 0.05 for the control and noise treatment group, respectively, for days one versus seven, one versus 17 and nine versus 17. ε and ƛ P < 0.05 for the control and noise chocolate treatment group, respectively, for days seven versus one, nine versus 17 and 17 versus one. * P < 0.05 for control versus noise on specific days.


A
Female Control
Female Noise
Male Control
Male Noise
Absolute body weight (g)
α β λ ε
α λ ε
α β λ ε
Acclimation
Treatment
Time (days)
B
Female Control
Female Noise
Male Control
Male Noise
Body weight gain (g)
λ
Acclimation
Treatment
Time (days)
Females
Male
Control Chow
Noise Chow
Control Chocolate
Noise Chocolate
C
Female 2h total intake (kcal)
α β ε
α β λ ε
λ ε
Acclimation
Treatment
Time (days)
D
Male 2h total intake (kcal)
α β
α β λ
Acclimation
Treatment
Time (days)
E
Control Female
Noise Female
22h chow intake (kcal)
α
Acclimation
Treatment
Time (days)
F
Control Male
Noise Male
22h chow intake (kcal)
β
β
Acclimation
Treatment
Time (days)
G
Control Female
Noise Female
24 chow intake (kcal)
Acclimation
Treatment
Time (days)
H
Control Male
Noise Male
24h total chow intake (kcal)
β
β
Acclimation
Treatment
Time (days)
I
Control Female
Noise Female
24h total intake (kcal)
β
Acclimation
Treatment
Time (days)
J
Control Male
Noise Male
24h total intake (kcal)
α β
α
α β
Acclimation
Treatment
Time (days)

TABLES

Table 1. Two factor repeated measures ANOVA or two factor ANOVA for study 1.

	Time period	
	7d - Acclimation	9d - Treatment
Body weight gain (g)		
Treatment	$F_{(1, 16)}$ = 0.62, P = 0.4416	$F_{(1, 16)}$ = 1.49, P = 0.2396
Time	$F_{(4, 64)}$ = 112.9, P < 0.0001	$F_{(8, 128)}$ = 1.89, P = 0.0668
Interaction	$F_{(4, 64)}$ = 0.47, P = 0.7533	$F_{(8, 128)}$ = 0.16, P = 0.9953
24-h calorie intake (kcal)		
Treatment	$F_{(1, 16)}$ = 3.85, P = 0.0675	$F_{(1, 16)}$ = 0.14, P = 0.7091
Time	$F_{(6, 96)}$ = 9.11, P < 0.0001	$F_{(8, 128)}$ = 0.78, P = 0.6201
Interaction	$F_{(6, 96)}$ = 1.84, P = 0.0997	$F_{(8, 128)}$ = 0.67, P = 0.7158
24-h HF diet (kcal)		
Treatment	$F_{(1, 16)}$ = 0.20, P = 0.6561	$F_{(1, 16)}$ = 0.06, P = 0.8157
Time	$F_{(6, 96)}$ = 6.65, P < 0.0001	$F_{(8, 128)}$ = 1.07, P = 0.3858
Interaction	$F_{(6, 96)}$ = 2.00, P = 0.0728	$F_{(8, 128)}$ = 0.56, P = 0.8090
24-h LF diet (kcal)		
Treatment	$F_{(1, 16)}$ = 0.38, P = 0.5475	$F_{(1, 16)}$ = 0.30, P = 0.5922
Time	$F_{(6, 96)}$ = 0.23, P = 0.9651	$F_{(8, 128)}$ = 0.35, P = 0.9425
Interaction	$F_{(6, 96)}$ = 1.60, P = 0.1564	$F_{(8, 128)}$ = 0.37, P = 0.9327
Total HF and LF (kcal)		
Treatment	$F_{(1, 32)}$ = 0.50, P = 0.4862	$F_{(1, 32)}$ = 0.04, P = 0.8426
Time	$F_{(1, 32)}$ = 96.16, P < 0.0001	$F_{(1, 32)}$ = 163.30, P < 0.0001
Interaction	$F_{(1, 32)}$ = 0.03, P = 0.8619	$F_{(1, 32)}$ = 0.30, P = 0.5886

LF: low fat diet. HF: high fat diet

Table 2. Cumulative weight gain and calorie intake for each diet and time period for study 1.

	Time period			
	7-d Acclimation		9-d Treatment	
Treatment:	Control	Noise	Control	Noise
Body weight gain (g)	35.4 ± 3.1	38.6 ± 2.7	44.6 ± 2.8	39.6 ± 3.0
24-h total intake (kcal)	685.5 ± 26.1	748.3 ± 18.6	787.1 ± 29.5	802.4 ± 27.7
HF diet (kcal)	544.2 ± 33.9	566.1 ± 46.8	649.1 ± 24.2	635.8 ± 50.5
LF diet (kcal)	131 ± 43.4	167.4 ± 40.4	138 ± 36.5	166.6 ± 37.6

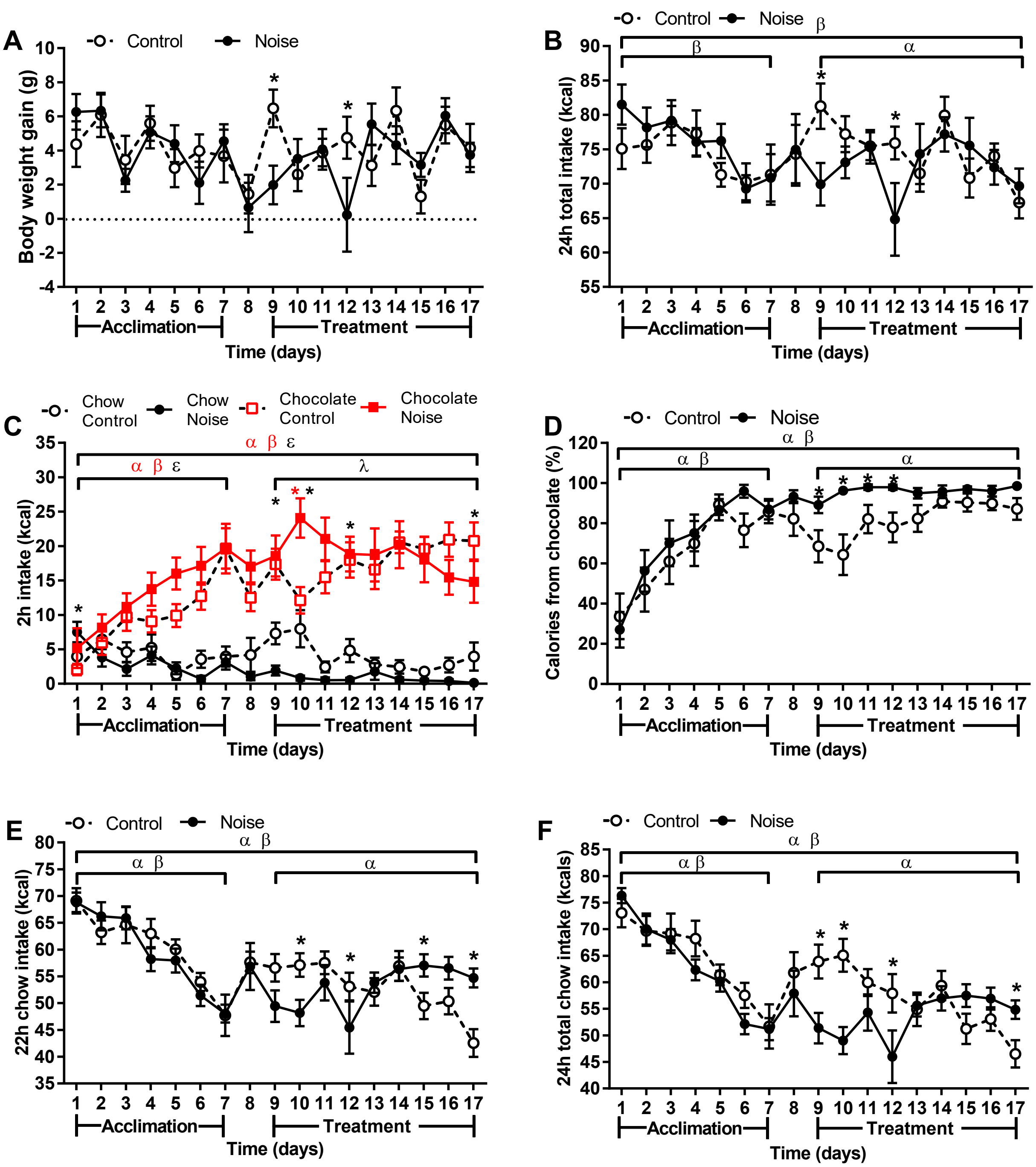
Data analyzed by unpaired t-test followed by Fisher's multiple comparisons test. Data expressed as mean ± SEM. N = 18 (control: n = 9 and treatment: n = 9). LF: low fat diet. HF: high fat diet

Table 3. Two factor repeated measures ANOVA or two factor ANOVA for rats that were classified as having a high or low preference for chocolate during the 9-d treatment period during study 2.

	9-d treatment time period		
	Treatment	Time	Interaction
2-h chow intake (kcal)			
High	$F_{(1, 15)} = 5.44$, $P = 0.0341$	$F_{(8, 120)} = 1.09$, $P = 0.3742$	$F_{(8, 120)} = 1.11$, $P = 0.3608$
Low	$F_{(1, 5)} = 91.25$, $P = 0.0002$	$F_{(8, 40)} = 2.09$, $P = 0.0596$	$F_{(8, 40)} = 1.24$, $P = 0.3032$
2-h chocolate intake (kcal)			
High	$F_{(1, 15)} = 0.74$, $P = 0.4025$	$F_{(8, 120)} = 0.30$, $P = 0.9655$	$F_{(8, 120)} = 2.19$, $P = 0.0325$
Low	$F_{(1, 5)} = 1.01$, $P = 0.3617$	$F_{(8, 40)} = 1.52$, $P = 0.1806$	$F_{(8, 40)} = 2.20$, $P = 0.0480$
2-h chocolate/2-h total intake (kcal)			
High	$F_{(1, 15)} = 6.41$, $P = 0.0230$	$F_{(8, 120)} = 2.20$, $P = 0.0323$	$F_{(8, 120)} = 1.50$, $P = 0.1655$
Low	$F_{(1, 5)} = 12.59$, $P = 0.0164$	$F_{(8, 40)} = 2.68$, $P = 0.0186$	$F_{(8, 40)} = 2.44$, $P = 0.0299$
22-h chow intake (kcal)			
High	$F_{(1, 15)} = 0.09$, $P = 0.7619$	$F_{(8, 120)} = 1.87$, $P = 0.0706$	$F_{(8, 120)} = 3.77$, $P = 0.0006$
Low	$F_{(1, 5)} = 0.00$, $P = 0.9782$	$F_{(8, 40)} = 2.80$, $P = 0.0147$	$F_{(8, 40)} = 4.23$, $P = 0.0010$
24-h chow intake (kcal)			
High	$F_{(1, 15)} = 0.10$, $P = 0.7594$	$F_{(8, 120)} = 1.79$, $P = 0.0844$	$F_{(8, 120)} = 4.12$, $P = 0.0002$
Low	$F_{(1, 5)} = 2.79$, $P = 0.1555$	$F_{(8, 40)} = 2.14$, $P = 0.0544$	$F_{(8, 40)} = 8.07$, $P < 0.0001$
24-h intake (kcal)			
High	$F_{(1, 15)} = 0.18$, $P = 0.6739$	$F_{(8, 120)} = 2.05$, $P = 0.0455$	$F_{(8, 120)} = 1.50$, $P = 0.1646$
Low	$F_{(1, 5)} = 2.84$, $P = 0.1528$	$F_{(8, 40)} = 2.01$, $P = 0.0705$	$F_{(8, 40)} = 2.18$, $P = 0.0499$

SUPPLEMENTARY FIGURES

Supplementary Figure 1: Daily body weight gain (A) and daily food intake (B) during the 7-day acclimation and 9-day treatment period for male rats that slept ad libitum (open symbols) or were noise-exposed (filled symbols, 8-h/d for nine days) that had rodent chow ad libitum and a daily 2-hour exposure to chocolate (C). Daily percentage of calories from chocolate during the 2-h exposure (D) and chow intake during the 22-h (E) and 24-h periods (F). Data were analyzed with 2 factor repeated measures ANOVA followed by Fisher's multiple comparisons test for each time period. Data expressed as mean ± SEM. N = 24 (control: n = 12 and treatment: n = 12). α and β $P < 0.05$ for the control and noise treatment group, respectively, for days one versus seven, one versus 17 and nine versus 17. ε and λ $P < 0.05$ for the control and noise treatment group, respectively, for days nine versus 17. * $P < 0.05$ for control versus noise on that specific day.


A
Control
Noise
Body weight gain (g)
Time (days)
Acclimation
Treatment
B
24h total intake (kcal)
C
Chow Control
Chow Noise
Chocolate Control
Chocolate Noise
2h intake (kcal)
D
Calories from chocolate (%)
E
22h chow intake (kcal)
F
24h total chow intake (kcals)

SUPPLEMENTARY TABLES

Supplementary Table 1. Two factor repeated measures ANOVA or two factor ANOVA for study 2.

	Time period	
	7d - Acclimation	9d - Treatment
Body weight gain (g)		
Treatment	$F_{(1, 22)}$ = 0.05, P = 0.8213	$F_{(1, 22)}$ = 3.16, P = 0.0894
Time	$F_{(6, 132)}$ = 2.43, P = 0.0290	$F_{(8, 176)}$ = 1.87, P = 0.0673
Interaction	$F_{(6, 132)}$ = 0.72, P = 0.6364	$F_{(8, 176)}$ = 2.15, P = 0.0331
24-h intake (kcal)		
Treatment	$F_{(1, 22)}$ = 0.33, P = 0.5718	$F_{(1, 22)}$ = 0.74, P = 0.3997
Time	$F_{(6, 132)}$ = 5.47, P < 0.0001	$F_{(8, 176)}$ = 3.03, P = 0.0033
Interaction	$F_{(6, 132)}$ = 0.98, P = 0.4423	$F_{(8, 176)}$ = 2.80, P = 0.0061
2-h chocolate intake (kcal)		
Treatment	$F_{(1, 22)}$ = 1.79, P = 0.1945	$F_{(1, 22)}$ = 0.12, P = 0.7353
Time	$F_{(6, 132)}$ = 18.89, P < 0.0001	$F_{(8, 176)}$ = 0.32, P = 0.9582
Interaction	$F_{(6, 132)}$ = 0.75, P = 0.6075	$F_{(8, 176)}$ = 3.59, P = 0.0007
2-h chow intake (kcal)		
Treatment	$F_{(1, 22)}$ = 0.50, P = 0.4867	$F_{(1, 22)}$ = 14.51, P = 0.0010
Time	$F_{(6, 132)}$ = 3.97, P = 0.0011	$F_{(8, 176)}$ = 3.10, P = 0.0027
Interaction	$F_{(6, 132)}$ = 2.44, P = 0.0288	$F_{(8, 176)}$ = 2.04, P = 0.0441
2-h chocolate intake/2-h total intake (kcal)		
Treatment	$F_{(1, 22)}$ = 0.29, P = 0.5935	$F_{(1, 22)}$ = 9.76, P = 0.0049
Time	$F_{(6, 132)}$ = 23.19, P < 0.0001	$F_{(8, 176)}$ = 4.31, P < 0.0001
Interaction	$F_{(6, 132)}$ = 0.9335, P = 0.4733	$F_{(8, 176)}$ = 2.7, P = 0.0079
22-h chow intake (kcal)		
Treatment	$F_{(1, 22)}$ = 0.12, P = 0.7330	$F_{(1, 22)}$ = 0.00 P = 0.9847
Time	$F_{(6, 132)}$ = 25.78, P < 0.0001	$F_{(8, 176)}$ = 3.03, P = 0.0033
Interaction	$F_{(6, 132)}$ = 0.78, P = 0.5831	$F_{(8, 176)}$ = 6.36, P < 0.0001
24-h chow intake (kcal)		
Treatment	$F_{(1, 22)}$ = 0.36, P = 0.5553	$F_{(1, 22)}$ = 1.25, P = 0.2757
Time	$F_{(6, 132)}$ = 32.01, P < 0.0001	$F_{(8, 176)}$ = 3.00, P = 0.0035
Interaction	$F_{(6, 132)}$ = 1.17, P = 0.3237	$F_{(8, 176)}$ = 8.72, P < 0.0001

N = 24 (control: n = 12 and treatment: n = 12).

Supplementary Table 2. Cumulative weight gain and calorie intake for each diet and time period for study 2.

	Time period			
	7d – Acclimation		9d - Treatment	
Treatment:	control	noise	control	noise
Body weight gain (g)	30.1 ± 2.9	31.0 ± 2.3	38.1 ± 2.1	32.6 ± 2.3
2-h chocolate intake (kcal)	69.0 ± 8.3	91.0 ± 14.2	161.4 ± 14.8	169.7 ± 19.5
2-h chow intake (kcal)	23.6 ± 3.7	29.1 ± 6.8	36.2 ± 7.5	7.2 ± 1.4*
22-h chow intake (kcal)	423.9 ± 11.9	416.6 ± 9.7	475.8 ± 15.2	475.4 ± 16.0
24-h chow intake (kcal)	453 ± 15.3	432.7 ± 9.7	512 ± 20.5	482.5 ± 16.5
24-h intake (kcal)	522 ± 15.4	531.2 ± 13.6	673.3 ± 17.4	652.3 ± 17.2

Data analyzed by unpaired t-test followed by Fisher's multiple comparisons test. Data expressed as mean ± SEM. N = 24 (control: n = 12 and treatment: n = 12). * $P < 0.05$ as compared to control group for treatment.

Supplementary Table 3. Two factor repeated measures ANOVA or two factor ANOVA for females only in study 3.

	Time period	
	7d - Acclimation	9d - Treatment
Absolute body weight (g)		
Treatment	$F_{(1, 6)} = 0.64, P = 0.4543$	$F_{(1, 6)} = 0.82, P = 0.4002$
Time	$F_{(3, 18)} = 5.0, P = 0.0109$	$F_{(4, 24)} = 18.49, P < 0.0001$
Interaction	$F_{(3, 18)} = 1.58, P = 0.2281$	$F_{(4, 24)} = 2.84, P = 0.0463$
Body weight gain (g)		
Treatment	$F_{(1, 6)} = 0.25, P = 0.6369$	$F_{(1, 6)} = 10.05, P = 0.0193$
Time	$F_{(3, 18)} = 0.83, P = 0.4958$	$F_{(4, 24)} = 0.80, P = 0.5362$
Interaction	$F_{(3, 18)} = 1.67, P = 0.2091$	$F_{(4, 24)} = 0.77, P = 0.5560$
24-h intake (kcal)		
Treatment	$F_{(1, 6)} = 0.36, P = 0.5710$	$F_{(1, 6)} = 0.31, P = 0.5985$
Time	$F_{(6, 36)} = 0.82, P = 0.5635$	$F_{(8, 48)} = 0.83, P = 0.5774$
Interaction	$F_{(6, 36)} = 0.81, P = 0.5674$	$F_{(8, 48)} = 0.81, P = 0.6005$
2-h chocolate intake (kcal)		
Treatment	$F_{(1, 6)} = 0.08, P = 0.7836$	$F_{(1, 6)} = 0.02, P = 0.9031$
Time	$F_{(6, 36)} = 7.98, P < 0.0001$	$F_{(8, 48)} = 1.57, P = 0.1576$
Interaction	$F_{(6, 36)} = 1.38, P = 0.2479$	$F_{(8, 48)} = 1.02, P = 0.4343$
2-h chow intake (kcal)		
Treatment	$F_{(1, 6)} = 0.18, P = 0.6844$	$F_{(1, 6)} = 1.05, P = 0.3442$
Time	$F_{(6, 36)} = 0.37, P = 0.8900$	$F_{(8, 48)} = 3.40, P = 0.0036$
Interaction	$F_{(6, 36)} = 3.13, P = 0.0143$	$F_{(8, 48)} = 1.25, P = 0.2922$
22-h chow intake (kcal)		
Treatment	$F_{(1, 6)} = 2.04, P = 0.2031$	$F_{(1, 6)} = 3.79, P = 0.0996$
Time	$F_{(6, 36)} = 0.82, P = 0.5640$	$F_{(8, 48)} = 2.61, P = 0.0188$
Interaction	$F_{(6, 36)} = 0.5894, P = 0.7365$	$F_{(8, 48)} = 1.75, P = 0.1115$
24-h chow intake (kcal)		
Treatment	$F_{(1, 6)} = 2.3, P = 0.1802$	$F_{(1, 6)} = 0.79, P = 0.4073$
Time	$F_{(6, 36)} = 0.84, P = 0.5488$	$F_{(8, 48)} = 2.17, P = 0.0472$
Interaction	$F_{(6, 36)} = 0.54, P = 0.7748$	$F_{(8, 48)} = 2.65, P = 0.0170$

Supplementary Table 4. Two factor repeated measures ANOVA or two factor ANOVA for males only in study 3.

	Time period	
	7d - Acclimation	9d - Treatment
Absolute body weight (g)		
Treatment	$F_{(1, 8)} = 2.89$, $P = 0.1277$	$F_{(1, 8)} = 3.40$, $P = 0.1023$
Time	$F_{(3, 24)} = 110.0$, $P < 0.0001$	$F_{(4, 32)} = 179.7$, $P < 0.0001$
Interaction	$F_{(3, 24)} = 3.61$, $P = 0.0278$	$F_{(4, 32)} = 0.17$, $P = 0.9538$
Body weight gain (g)		
Treatment	$F_{(1, 8)} = 1.55$, $P = 0.2482$	$F_{(1, 8)} = 0.61$, $P = 0.4555$
Time	$F_{(3, 24)} = 3.90$, $P = 0.0212$	$F_{(4, 32)} = 1.40$, $P = 0.2566$
Interaction	$F_{(3, 24)} = 2.03$, $P = 0.1369$	$F_{(4, 32)} = 0.84$, $P = 0.5113$
24-h intake (kcal)		
Treatment	$F_{(1, 8)} = 0.43$, $P = 0.5295$	$F_{(1, 8)} = 2.58$, $P = 0.1466$
Time	$F_{(6, 48)} = 30.52$, $P < 0.0001$	$F_{(8, 64)} = 16.55$, $P < 0.0001$
Interaction	$F_{(6, 48)} = 1.31$, $P = 0.2702$	$F_{(8, 64)} = 3.67$, $P = 0.0014$
2-h chocolate intake (kcal)		
Treatment	$F_{(1, 8)} = 0.71$, $P = 0.4232$	$F_{(1, 8)} = 0.22$, $P = 0.6522$
Time	$F_{(6, 48)} = 5.59$, $P = 0.0002$	$F_{(8, 64)} = 3.98$, $P = 0.0007$
Interaction	$F_{(6, 48)} = 0.82$, $P = 0.5566$	$F_{(8, 64)} = 1.91$, $P = 0.0737$
2-h chow intake (kcal)		
Treatment	$F_{(1, 8)} = 3.42$, $P = 0.1016$	$F_{(1, 8)} = 7.47$, $P = 0.0257$
Time	$F_{(6, 48)} = 1.37$, $P = 0.2441$	$F_{(8, 64)} = 0.51$, $P = 0.8414$
Interaction	$F_{(6, 48)} = 1.92$, $P = 0.0972$	$F_{(8, 64)} = 2.86$, $P = 0.0088$
22-h chow intake (kcal)		
Treatment	$F_{(1, 8)} = 1.10$, $P = 0.3257$	$F_{(1, 8)} = 0.19$, $P = 0.6768$
Time	$F_{(6, 48)} = 20.6$, $P < 0.0001$	$F_{(8, 64)} = 24.1$, $P < 0.0001$
Interaction	$F_{(6, 48)} = 0.98$, $P = 0.4509$	$F_{(8, 64)} = 6.20$, $P < 0.0001$
24-h chow intake (kcal)		
Treatment	$F_{(1, 8)} = 1.73$, $P = 0.2248$	$F_{(1, 8)} = 0.38$, $P = 0.5554$
Time	$F_{(6, 48)} = 19.64$, $P < 0.0001$	$F_{(8, 64)} = 23.69$, $P < 0.0001$
Interaction	$F_{(6, 48)} = 2.06$, $P = 0.0760$	$F_{(8, 64)} = 5.45$, $P < 0.0001$

REFERENCES

1. Watson, N. F., M. S. Badr, G. Belenky, D. L. Bliwise, O. M. Buxton, D. Buysse, D. F. Dinges, J. Gangwisch, M. A. Grandner, C. Kushida, R. K. Malhotra, J. L. Martin, S. R. Patel, S. F. Quan, E. Tasali, and C. C. Panel, 2015, Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society on the Recommended Amount of Sleep for a Healthy Adult: Methodology and Discussion: Sleep, v. 38, p. 1161-83.
2. Wu, Y., Q. Gong, Z. Zou, H. Li, and X. Zhang, 2016, Short sleep duration and obesity among children: A systematic review and meta-analysis of prospective studies: Obes Res Clin Pract.
3. Cappuccio, F. P., F. M. Taggart, N. B. Kandala, A. Currie, E. Peile, S. Stranges, and M. A. Miller, 2008, Meta-analysis of short sleep duration and obesity in children and adults: Sleep, v. 31, p. 619-26.
4. Logue, E. E., E. D. Scott, P. A. Palmieri, and P. Dudley, 2014, Sleep duration, quality, or stability and obesity in an urban family medicine center: J Clin Sleep Med, v. 10, p. 177-82.
5. Wu, J., H. Wu, J. Wang, L. Guo, X. Deng, and C. Lu, 2015, Associations between Sleep Duration and Overweight/Obesity: Results from 66,817 Chinese Adolescents: Sci Rep, v. 5, p. 16686.

6. Shan, Z., H. Ma, M. Xie, P. Yan, Y. Guo, W. Bao, Y. Rong, C. L. Jackson, F. B. Hu, and L. Liu, 2015, Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies: Diabetes Care, v. 38, p. 529-37.

7. Broussard, J. L., D. A. Ehrmann, E. Van Cauter, E. Tasali, and M. J. Brady, 2012, Impaired insulin signaling in human adipocytes after experimental sleep restriction: a randomized, crossover study: Ann Intern Med, v. 157, p. 549-57.

8. Cappuccio, F. P., D. Cooper, L. D'Elia, P. Strazzullo, and M. A. Miller, 2011, Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies: Eur Heart J, v. 32, p. 1484-92.

9. Faraut, B., E. Touchette, H. Gamble, S. Royant-Parola, M. E. Safar, B. Varsat, and D. Léger, 2012, Short sleep duration and increased risk of hypertension: a primary care medicine investigation: J Hypertens, v. 30, p. 1354-63.

10. Hammond, E. C., 1964, SOME PRELIMINARY FINDINGS ON PHYSICAL COMPLAINTS FROM A PROSPECTIVE STUDY OF 1,064,004 MEN AND WOMEN: Am J Public Health Nations Health, v. 54, p. 11-23.

11. Grandner, M. A., L. Hale, M. Moore, and N. P. Patel, 2010, Mortality associated with short sleep duration: The evidence, the possible mechanisms, and the future: Sleep Med Rev, v. 14, p. 191-203.

12. Stamatakis, K. A., G. A. Kaplan, and R. E. Roberts, 2007, Short sleep duration across income, education, and race/ethnic groups: population prevalence and

growing disparities during 34 years of follow-up: Ann Epidemiol, v. 17, p. 948-55.

13. Paeratakul, S., M. A. White, D. A. Williamson, D. H. Ryan, and G. A. Bray, 2002, Sex, race/ethnicity, socioeconomic status, and BMI in relation to self-perception of overweight: Obes Res, v. 10, p. 345-50.

14. Ramirez, I., 1990, What do we mean when we say "palatable food"?: Appetite, v. 14, p. 159-61.

15. Mela, D. J., 2006, Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity: Appetite, v. 47, p. 10-7.

16. Mozaffarian, D., T. Hao, E. B. Rimm, W. C. Willett, and F. B. Hu, 2011, Changes in diet and lifestyle and long-term weight gain in women and men: N Engl J Med, v. 364, p. 2392-404.

17. Bosy-Westphal, A., S. Hinrichs, K. Jauch-Chara, B. Hitze, W. Later, B. Wilms, U. Settler, A. Peters, D. Kiosz, and M. J. Muller, 2008, Influence of partial sleep deprivation on energy balance and insulin sensitivity in healthy women: Obes Facts, v. 1, p. 266-73.

18. Brondel, L., M. A. Romer, P. M. Nougues, P. Touyarou, and D. Davenne, 2010, Acute partial sleep deprivation increases food intake in healthy men: Am J Clin Nutr, v. 91, p. 1550-9.

19. Calvin, A. D., R. E. Carter, T. Adachi, P. G. Macedo, F. N. Albuquerque, C. van der Walt, J. Bukartyk, D. E. Davison, J. A. Levine, and V. K. Somers, 2013, Effects of

experimental sleep restriction on caloric intake and activity energy expenditure: Chest, v. 144, p. 79-86.

20. Markwald, R. R., E. L. Melanson, M. R. Smith, J. Higgins, L. Perreault, R. H. Eckel, and K. P. Wright, 2013, Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain: Proc Natl Acad Sci U S A, v. 110, p. 5695-700.

21. Spaeth, A. M., D. F. Dinges, and N. Goel, 2013, Effects of Experimental Sleep Restriction on Weight Gain, Caloric Intake, and Meal Timing in Healthy Adults: Sleep, v. 36, p. 981-990.

22. St-Onge, M. P., A. L. Roberts, J. Chen, M. Kelleman, M. O'Keeffe, A. RoyChoudhury, and P. J. Jones, 2011, Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals: Am J Clin Nutr, v. 94, p. 410-6.

23. Nedeltcheva, A. V., J. M. Kilkus, J. Imperial, K. Kasza, D. A. Schoeller, and P. D. Penev, 2009, Sleep curtailment is accompanied by increased intake of calories from snacks: Am J Clin Nutr, v. 89, p. 126-33.

24. Schmid, S. M., M. Hallschmid, K. Jauch-Chara, B. Wilms, C. Benedict, H. Lehnert, J. Born, and B. Schultes, 2009, Short-term sleep loss decreases physical activity under free-living conditions but does not increase food intake under time-deprived laboratory conditions in healthy men: Am J Clin Nutr, v. 90, p. 1476-82.

25. Benedict, C., M. Hallschmid, A. Lassen, C. Mahnke, B. Schultes, H. B. Schiöth, J. Born, and T. Lange, 2011, Acute sleep deprivation reduces energy expenditure in healthy men: Am J Clin Nutr, v. 93, p. 1229-36.

26. Liang, N. C., N. T. Bello, and T. H. Moran, 2015, Wheel running reduces high-fat diet intake, preference and mu-opioid agonist stimulated intake: Behav Brain Res, v. 284, p. 1-10.

27. Woods, S. C., R. J. Seeley, P. A. Rushing, D. D'Alessio, and P. Tso, 2003, A controlled high-fat diet induces an obese syndrome in rats: J Nutr, v. 133, p. 1081-7.

28. Lemonnier, D., 1972, Effect of age, sex, and sites on the cellularity of the adipose tissue in mice and rats rendered obese by a high-fat diet: J Clin Invest, v. 51, p. 2907-15.

29. Buettner, R., K. G. Parhofer, M. Woenckhaus, C. E. Wrede, L. A. Kunz-Schughart, J. Schölmerich, and L. C. Bollheimer, 2006, Defining high-fat-diet rat models: metabolic and molecular effects of different fat types: J Mol Endocrinol, v. 36, p. 485-501.

30. Harris, R. B., J. Zhou, B. D. Youngblood, I. I. Rybkin, G. N. Smagin, and D. H. Ryan, 1998, Effect of repeated stress on body weight and body composition of rats fed low- and high-fat diets: Am J Physiol, v. 275, p. R1928-38.

31. Perron, I. J., A. I. Pack, and S. Veasey, 2015, Diet/Energy Balance Affect Sleep and Wakefulness Independent of Body Weight: Sleep, v. 38, p. 1893-903.

32. Rothwell, N. J., and M. J. Stock, 1982a, Effects of feeding a palatable "cafeteria' diet on energy balance in young and adult lean (+/?) Zucker rats: Br J Nutr, v. 47, p. 461-71.

33. Rothwell, N. J., and M. J. Stock, 1986, Body weight and brown fat activity in hyperphagic cafeteria-fed female rats and their offspring: Biol Neonate, v. 49, p. 284-91.

34. Rothwell, N. J., and M. J. Stock, 1982b, Energy expenditure of 'cafeteria'-fed rats determined from measurements of energy balance and indirect calorimetry: J Physiol, v. 328, p. 371-7.

35. Rothwell, N. J., M. J. Stock, and D. A. York, 1984, Effects of adrenalectomy on energy balance, diet-induced thermogenesis and brown adipose tissue in adult cafeteria-fed rats: Comp Biochem Physiol A Comp Physiol, v. 78, p. 565-9.

36. Rothwell, N. J., M. J. Stock, and B. P. Warwick, 1985, Energy balance and brown fat activity in rats fed cafeteria diets or high-fat, semisynthetic diets at several levels of intake: Metabolism, v. 34, p. 474-80.

37. Rothwell, N. J., M. J. Stock, and B. P. Warwick, 1983, The effect of high fat and high carbohydrate cafeteria diets on diet-induced thermogenesis in the rat: Int J Obes, v. 7, p. 263-70.

38. Rothwell, N. J., M. E. Saville, and M. J. Stock, 1982, Effects of feeding a "cafeteria" diet on energy balance and diet-induced thermogenesis in four strains of rat: J Nutr, v. 112, p. 1515-24.

39. Spaeth, A. M., D. F. Dinges, and N. Goel, 2015, Resting metabolic rate varies by race and by sleep duration: Obesity (Silver Spring), v. 23, p. 2349-56.

40. Prats, E., M. Monfar, J. Castellà, R. Iglesias, and M. Alemany, 1989, Energy intake of rats fed a cafeteria diet: Physiol Behav, v. 45, p. 263-72.

41. Gac, L., V. Kanaly, V. Ramirez, J. A. Teske, M. P. Pinto, and C. E. Perez-Leighton, 2015, Behavioral characterization of a model of differential susceptibility to obesity induced by standard and personalized cafeteria diet feeding: Physiol Behav, v. 152, p. 315-22.

42. Sclafani, A., and A. N. Gorman, 1977, Effects of age, sex, and prior body weight on the development of dietary obesity in adult rats: Physiol Behav, v. 18, p. 1021-6.

43. Warneke, W., S. Klaus, H. Fink, S. C. Langley-Evans, and J. P. Voigt, 2014, The impact of cafeteria diet feeding on physiology and anxiety-related behaviour in male and female Sprague-Dawley rats of different ages: Pharmacol Biochem Behav, v. 116, p. 45-54.

44. de Oliveira, E. M., B. Visniauskas, S. Sandri, S. Migliorini, M. L. Andersen, S. Tufik, R. A. Fock, J. R. Chagas, and A. Campa, 2015, Late effects of sleep restriction: Potentiating weight gain and insulin resistance arising from a high-fat diet in mice: Obesity (Silver Spring), v. 23, p. 391-8.

45. Martins, P. J., L. Fernandes, A. C. de Oliveira, S. Tufik, and V. D'Almeida, 2011, Type of diet modulates the metabolic response to sleep deprivation in rats: Nutr Metab (Lond), v. 8, p. 86.

46. Mavanji, V., J. A. Teske, C. J. Billington, and C. M. Kotz, 2013, Partial sleep deprivation by environmental noise increases food intake and body weight in obesity-resistant rats: Obesity (Silver Spring), v. 21, p. 1396-405.

47. Parrish, J. B., and J. A. Teske, 2017, Acute partial sleep deprivation due to environmental noise increases weight gain by reducing energy expenditure in rodents: Obesity (Silver Spring), v. 25, p. 141-146.

48. Mavanji, V., C. J. Billington, C. M. Kotz, and J. A. Teske, 2012, Sleep and obesity: a focus on animal models: Neurosci Biobehav Rev, v. 36, p. 1015-29.

49. Guan, Z., A. N. Vgontzas, E. O. Bixler, and J. Fang, 2008, Sleep is increased by weight gain and decreased by weight loss in mice: Sleep, v. 31, p. 627-33.

50. Jenkins, J. B., T. Omori, Z. Guan, A. N. Vgontzas, E. O. Bixler, and J. Fang, 2006, Sleep is increased in mice with obesity induced by high-fat food: Physiol Behav, v. 87, p. 255-62.

51. Luppi, M., M. Cerri, D. Martelli, D. Tupone, F. Del Vecchio, A. Di Cristoforo, E. Perez, G. Zamboni, and R. Amici, 2014, Waking and sleeping in the rat made obese through a high-fat hypercaloric diet: Behav Brain Res, v. 258, p. 145-52.

52. Laposky, A. D., M. A. Bradley, D. L. Williams, J. Bass, and F. W. Turek, 2008, Sleep-wake regulation is altered in leptin-resistant (db/db) genetically obese and diabetic mice: Am J Physiol Regul Integr Comp Physiol, v. 295, p. R2059-66.

53. Laposky, A. D., J. Shelton, J. Bass, C. Dugovic, N. Perrino, and F. W. Turek, 2006, Altered sleep regulation in leptin-deficient mice: Am J Physiol Regul Integr Comp Physiol, v. 290, p. R894-903.

54. Lindqvist, A., P. Mohapel, B. Bouter, H. Frielingsdorf, D. Pizzo, P. Brundin, and C. Erlanson-Albertsson, 2006, High-fat diet impairs hippocampal neurogenesis in male rats: Eur J Neurol, v. 13, p. 1385-8.

55. Mavanji, V., J. A. Teske, C. J. Billington, and C. M. Kotz, 2010, Elevated sleep quality and orexin receptor mRNA in obesity-resistant rats: Int J Obes (Lond), v. 34, p. 1576-88.

56. Rabat, A., J. J. Bouyer, J. M. Aran, M. Le Moal, and W. Mayo, 2005, Chronic exposure to an environmental noise permanently disturbs sleep in rats: inter-individual vulnerability: Brain Res, v. 1059, p. 72-82.

57. Ouis, D., 1999, Exposure to Nocturnal Road Traffic Noise: Sleep Disturbance and its After Effects: Noise Health, v. 1, p. 11-36.

58. Patel, S. R., A. Malhotra, D. P. White, D. J. Gottlieb, and F. B. Hu, 2006, Association between reduced sleep and weight gain in women: Am J Epidemiol, v. 164, p. 947-54.

59. Chaput, J. P., J. P. Després, C. Bouchard, and A. Tremblay, 2008, The association

between sleep duration and weight gain in adults: a 6-year prospective study from the Quebec Family Study: Sleep, v. 31, p. 517-23.

60. Ramanathan, L., S. Gulyani, R. Nienhuis, and J. M. Siegel, 2002, Sleep deprivation decreases superoxide dismutase activity in rat hippocampus and brainstem: Neuroreport, v. 13, p. 1387-90.

61. Ramanathan, L., S. Hu, S. A. Frautschy, and J. M. Siegel, 2010, Short-term total sleep deprivation in the rat increases antioxidant responses in multiple brain regions without impairing spontaneous alternation behavior: Behav Brain Res, v. 207, p. 305-9.

62. Rechtschaffen, A., and B. M. Bergmann, 1995, Sleep deprivation in the rat by the disk-over-water method: Behav Brain Res, v. 69, p. 55-63.

63. Laposky, A. D., J. Bass, A. Kohsaka, and F. W. Turek, 2008a, Sleep and circadian rhythms: key components in the regulation of energy metabolism: FEBS Lett, v. 582, p. 142-51.

64. Novati, A., V. Roman, T. Cetin, R. Hagewoud, J. A. den Boer, P. G. Luiten, and P. Meerlo, 2008, Chronically restricted sleep leads to depression-like changes in neurotransmitter receptor sensitivity and neuroendocrine stress reactivity in rats: Sleep, v. 31, p. 1579-85.

65. Koban, M., and C. V. Stewart, 2006, Effects of age on recovery of body weight following REM sleep deprivation of rats: Physiol Behav, v. 87, p. 1-6.

66. Bartol-Munier, I., S. Gourmelen, P. Pevet, and E. Challet, 2006, Combined effects of high-fat feeding and circadian desynchronization: Int J Obes (Lond), v. 30, p. 60-7.

67. Woods, S. C., D. A. D'Alessio, P. Tso, P. A. Rushing, D. J. Clegg, S. C. Benoit, K. Gotoh, M. Liu, and R. J. Seeley, 2004, Consumption of a high-fat diet alters the homeostatic regulation of energy balance: Physiol Behav, v. 83, p. 573-8.

68. Everson, C. A., C. B. Smith, and L. Sokoloff, 1994, Effects of prolonged sleep deprivation on local rates of cerebral energy metabolism in freely moving rats: J Neurosci, v. 14, p. 6769-78.

69. Everson, C. A., and W. R. Crowley, 2004, Reductions in circulating anabolic hormones induced by sustained sleep deprivation in rats: Am J Physiol Endocrinol Metab, v. 286, p. E1060-70.

70. Koban, M., and K. L. Swinson, 2005, Chronic REM-sleep deprivation of rats elevates metabolic rate and increases UCP1 gene expression in brown adipose tissue: Am J Physiol Endocrinol Metab, v. 289, p. E68-74.

71. Hanlon, E. C., M. E. Andrzejewski, B. K. Harder, A. E. Kelley, and R. M. Benca, 2005, The effect of REM sleep deprivation on motivation for food reward: Behav Brain Res, v. 163, p. 58-69.

72. Everson, C. A., and T. A. Wehr, 1993, Nutritional and metabolic adaptations to prolonged sleep deprivation in the rat: Am J Physiol, v. 264, p. R376-87.

73. Papakonstantinou, E., D. H. Ryan, and R. B. Harris, 2003, Dietary fish oil does

not protect rats exposed to restraint or sleep deprivation stress: Physiol Behav, v. 78, p. 759-65.

74. Koban, M., L. V. Sita, W. W. Le, and G. E. Hoffman, 2008, Sleep deprivation of rats: the hyperphagic response is real: Sleep, v. 31, p. 927-33.

75. Chaput, J. P., L. Klingenberg, A. Astrup, and A. M. Sjödin, 2011, Modern sedentary activities promote overconsumption of food in our current obesogenic environment: Obes Rev, v. 12, p. e12-20.

76. Corwin, R. L., 2004, Binge-type eating induced by limited access in rats does not require energy restriction on the previous day: Appetite, v. 42, p. 139-42.

77. Hogenkamp, P. S., E. Nilsson, V. C. Nilsson, C. D. Chapman, H. Vogel, L. S. Lundberg, S. Zarei, J. Cedernaes, F. H. Rångtell, J. E. Broman, S. L. Dickson, J. M. Brunstrom, C. Benedict, and H. B. Schiöth, 2013, Acute sleep deprivation increases portion size and affects food choice in young men: Psychoneuroendocrinology, v. 38, p. 1668-74.

78. Shechter, A., R. Rising, S. Wolfe, J. B. Albu, and M. P. St-Onge, 2014, Postprandial thermogenesis and substrate oxidation are unaffected by sleep restriction: Int J Obes (Lond), v. 38, p. 1153-8.

79. Klingenberg, L., J. P. Chaput, U. Holmbäck, P. Jennum, A. Astrup, and A. Sjödin, 2012, Sleep restriction is not associated with a positive energy balance in adolescent boys: Am J Clin Nutr, v. 96, p. 240-8.

80. Hursel, R., F. Rutters, H. K. Gonnissen, E. A. Martens, and M. S. Westerterp-

Plantenga, 2011, Effects of sleep fragmentation in healthy men on energy expenditure, substrate oxidation, physical activity, and exhaustion measured over 48 h in a respiratory chamber: Am J Clin Nutr, v. 94, p. 804-8.

81. Rechtschaffen, A., B. M. Bergmann, C. A. Everson, C. A. Kushida, and M. A. Gilliland, 2002, Sleep deprivation in the rat: X. Integration and discussion of the findings. 1989: Sleep, v. 25, p. 68-87.

82. Nascimento, A. F., M. M. Sugizaki, A. S. Leopoldo, A. P. Lima-Leopoldo, R. A. Luvizotto, C. R. Nogueira, and A. C. Cicogna, 2008, A hypercaloric pellet-diet cycle induces obesity and co-morbidities in Wistar rats: Arq Bras Endocrinol Metabol, v. 52, p. 968-74.

83. Oswald, K. D., D. L. Murdaugh, V. L. King, and M. M. Boggiano, 2011, Motivation for palatable food despite consequences in an animal model of binge eating: Int J Eat Disord, v. 44, p. 203-11.

84. Gugusheff, J. R., Z. Y. Ong, and B. S. Muhlhausler, 2015, The early origins of food preferences: targeting the critical windows of development: FASEB J, v. 29, p. 365-73.

85. Swithers, S. E., and T. L. Davidson, 2008, A role for sweet taste: calorie predictive relations in energy regulation by rats: Behav Neurosci, v. 122, p. 161-73.

86. CORBIT, J. D., and E. STELLAR, 1964, PALATABILITY, FOOD INTAKE, AND OBESITY IN NORMAL AND HYPERHAGIC RATS: J Comp Physiol Psychol, v. 58, p. 63-7.

87. Flegal, K. M., M. D. Carroll, C. L. Ogden, and C. L. Johnson, 2002, Prevalence and trends in obesity among US adults, 1999-2000: JAMA, v. 288, p. 1723-7.

88. Madrid-Valero, J. J., J. M. Martínez-Selva, B. Ribeiro do Couto, J. F. Sánchez-Romera, and J. R. Ordoñana, 2017, Age and gender effects on the prevalence of poor sleep quality in the adult population: Gac Sanit, v. 31, p. 18-22.

89. Valenstein, E. S., V. C. Cox, and J. W. Kakolewski, 1969, Sex differences in hyperphagia and body weight following hypothalamic damage: Ann N Y Acad Sci, v. 157, p. 1030-48.

90. Valenstein, E. S., J. W. Kakolewski, and V. C. Cox, 1967, Sex differences in taste preference for glucose and saccharin solutions: Science, v. 156, p. 942-3.

91. Fukushima, A., H. Hagiwara, H. Fujioka, F. Kimura, T. Akema, and T. Funabashi, 2015, Sex differences in feeding behavior in rats: the relationship with neuronal activation in the hypothalamus: Front Neurosci, v. 9, p. 88.

92. Suchecki, D., and S. Tufik, 2000, Social stability attenuates the stress in the modified multiple platform method for paradoxical sleep deprivation in the rat: Physiol Behav, v. 68, p. 309-16.

93. Dallman, M. F., S. E. la Fleur, N. C. Pecoraro, F. Gomez, H. Houshyar, and S. F. Akana, 2004, Minireview: glucocorticoids--food intake, abdominal obesity, and wealthy nations in 2004: Endocrinology, v. 145, p. 2633-8.

94. Bake, T., M. Murphy, D. G. Morgan, and J. G. Mercer, 2014, Large, binge-type meals of high fat diet change feeding behaviour and entrain food anticipatory

activity in mice: Appetite, v. 77, p. 60-71.

95. Landry, G. J., B. A. Kent, D. F. Patton, M. Jaholkowski, E. G. Marchant, and R. E. Mistlberger, 2011, Evidence for time-of-day dependent effect of neurotoxic dorsomedial hypothalamic lesions on food anticipatory circadian rhythms in rats: PLoS One, v. 6, p. e24187.