

THE EFFECTS OF SMOKING CESSATION ON CONTROL OF FOOD INTAKE IN
POSTMENOPAUSAL AFRICAN-AMERICAN AND CAUCASIAN WOMEN

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Amanda K. Manning
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Abstract

Smoking cessation leads to greater weight gain in women than men, and older and postmenopausal women are at greater risk for weight gain than younger, premenopausal women. African-American postmenopausal women may be at the greatest risk. Weight gain after smoking cessation is primarily due to increased caloric intake. Currently, the literature regarding measurement of macronutrient intake after smoking cessation is plagued with methodological problems. The Geiselman Macronutrient Self-Selection Paradigm (MSSP) significantly and systematically varies fat across other macronutrients and the Geiselman Food Preference Questionnaire (FPQ) measures the negative feedback of satiation via pre- and postprandial hedonic ratings of foods. Fifty-five Caucasian and 32 African-American postmenopausal women were recruited for the present study. We measured changes in total caloric intake, and specific macronutrient intake with the use of the MSSP, and we measured hedonic ratings with the use of the FPQ before and after smoking cessation. We hypothesized that total caloric intake and intake of high-fat foods would increase postcessation. Also, we hypothesized that women would be able to reach satiation more readily while smoking than they would postcessation. We found that Caucasian females increased total caloric intake and intake of high-fat foods after smoking cessation; however, their level of satiation did not change from pre- to postcessation. Thus, the Caucasian women had to ingest significantly more total kcals, especially from high-fat foods, postcessation to achieve the same level of satiation that they reached with much smaller amount of food while still smoking. Total caloric intake, including intake of high-fat foods, did not differ from pre- to postcessation in African-American females. African-American women ingested significantly more total kcals and intake of high-fat foods than did Caucasian females, regardless of smoking status. African-American women also showed significantly smaller decreases in

hedonic ratings of high-fat foods from pre- to postprandial than did Caucasian women, indicating less satiating effect of high-fat foods in the African-American females.

Chapter 1: Review of Literature

Studies on smoking cessation, while numerous, have yet to address all of the factors regarding cessation's effect on weight gain among different sexes and races. In this section, the influences contributing to changes in weight gain between males and females and African-Americans and Caucasians are discussed. Further, discussion regarding the reason behind increased postcessation weight gain, increased caloric intake, as well as macronutrient-specific effects are presented. Finally, methodological problems in the literature are reviewed. They support a need for a more systematic measure of macronutrient intake – the Macronutrient Self-Selection Paradigm.

Smoking Cessation, Sex Differences, Race Differences, Aging and Weight Gain

Sex and Race Differences in Smoking Cessation and Weight Gain

Smoking cessation causes weight gain in both males and females, but sex differences exist regarding the extent of the gain. Typical postcessation weight gain for men is approximately 3.5 kg as seen from a 16-year follow-up (Froom et al., 1999); however women gain more than 4.5 kg after one year of having ceased smoking (Caan, Coates, Schaefer, Finkler, Sternfeld, and Corbett, 1996). Females gain from 3-4.2% more than men one year postcessation (O'Hara et al., 1998). Also, women are at a greater risk for major weight gain than men. Women were almost 4% more likely to gain more than 13 kg after cessation than men and 12.5% more likely to gain more than 20% of their body weight (Williamson et al., 1991). Thus it is well-documented that females gain more weight, including major weight gains, than males.

In addition to sex differences, certain races may be more susceptible to weight gain after smoking cessation. Regardless of smoking status, African-Americans weigh significantly more than Caucasians (Klesges et al., 1998). However, smoking cessation increases this disparity; weight gain after cessation is a more severe problem for African-Americans than for Caucasians

(Williamson et al., 1991). African-American quitters had a higher probability of postcessation weight gain than other races, including Caucasians. Average weight gain attributable to smoking cessation was 6.6 kg in African-Americans; however, Caucasians gained only 3.8 kg. Furthermore, they were more than twice as likely to gain $>8 - \leq 13$ kg, and more than three times as likely to gain >13 kg. Thus, African-Americans are at high risk of weight gain after smoking cessation.

Given the aforementioned studies, African-American females seem to be at the greatest risk of postcessation weight gain due to the influence of both sex and race. However, there is a dearth of research regarding this population. Vander Weg, et al. (2001) found a small but non-significant increase in weight in African-American women versus Caucasian women in a two-week abstinence study (1.21 kg vs. 0.81kg, respectively, non-significant). Both race and sex may influence weight gain after smoking cessation, and more research is necessary to investigate this potentially higher-risk group.

Aging/Menopause, Race and Weight Gain

Aging contributes to weight gain to a greater extent in women than in men. Over the course of 30 years, nationally representative surveys, including the National Health Examination and the National Health and Nutrition Examination Surveys, were taken in order to assess the prevalence of obesity in the United States using the measurement of body mass index (BMI) (Flegal, Carroll, Kuczmarski, & Johnson, 1998). A key finding is the trend of overweight (BMI of 25.0-29.9) across the life cycle of age-matched men versus women. For men, prevalence of overweight increased until reaching a plateau in the 30-39 year age group where their weight leveled off with only slight increases in later years. For women, however, the prevalence of overweight increased steadily with each increasing age group up through the 60-69 year range. In addition, prevalence of class II obesity (BMI 35.0-39.9) and class III obesity (≥ 40.0) was

higher for women than men. In general, women have a greater age-related increase in BMI than do men.

A critical component of aging in women that puts them at a higher risk for weight gain is menopause. The average age of menopause is 51 years (McKinlay, Brambilla, & Posner, 1992). Weight (Macdonald, New, Campbell, & Reid, 2003) and BMI (Pasquali et al., 1994) change during the transition from premenopause to perimenopause (the last seven years prior to menopause) to menopause. Perimenopausal and early postmenopausal women gained weight 5-7 years after baseline measurements ($m = 3.3 \text{ kg} \pm 5.1 \text{ kg}$) (Macdonald et al., 2003). From pre- to peri-menopause, BMI increased, and this increase remained after menopause up through 58 years of age. Average weight gain in a cohort of middle aged women (42-50 years) over three years was 2.25 kg with twenty percent of the women gaining 4.5 kg or more (Wing, Matthews, Kuller, Meilahn, & Plantinga, 1991). Postmenopausal women weigh 5.3 kg more than their premenopausal, age-matched counterparts (Matthews et al., 1989). Thus, menopause is characterized by weight gain and an increase in BMI.

In addition to changes in weight, fat distribution also changes during menopause. Abdominal obesity increases with postmenopausal weight gain (Schlienger & Pradignac, 1993; Aloia, Vaswani, Russo, Sheehan, & Flaster, 1995). Premenopausal women typically deposit fat in the femoral region, and in postmenopausal women, fat deposition increases in the abdominal region. Thus, the effects of aging and menopause put women at a greater risk for weight gain, and the changes in abdominal fat distribution poses further health risks.

Furthermore, racial disparities exist in weight gain occurring with aging and menopause. Wing, Matthews, Kuller, Meilhan, and Plantinga (1991) found that in a middle-aged cohort, African-American women gained more weight than Caucasian women over three years (4.05 kg vs. 2.07 kg, respectively). In addition, for women in the 50-59 year age group, overweight and

obesity, defined by BMI \geq 25.0, was almost 6% higher for African-American women than for Caucasian women (Flegal et al., 1998). Hence, African-American women may be at a higher risk for age-related weight gain due to the combined effects of race and aging/menopause.

Smoking Cessation and Aging/Menopause

Aging and menopausal status contribute to weight gain after smoking cessation. Female quitters older than 35 years gained significantly more weight than female quitters younger than 35 years (4.1 kg vs. 1.2 kg) (Becona & Vazquez, 1998). Caan et al. (1996) found that older age was the strongest predictor of greater postcessation weight gain among females. For every 10-year increment from 20-65 years old, postcessation weight increased by about 1 kg. In another study, from baseline before menopause to a two-year follow-up after menopause, women who had ceased smoking for 1-2 years experienced significantly more weight gain than non-smokers (Burnette, Meilahn, Wing, & Kuller, 1998). Women who began the study as premenopausal and who at follow-up were in the first year of menopause (amenorrheic for at least 12 months) gained weight in general. However, of that group, women who stopped smoking (quitters) gained the most weight, 2.74 kg, versus smokers and those who had never smoked. At 2 years after menopause, there was a significant increase in weight gain between those who had quit and those who had never smoked, just over 3 kg. Thus, not only do aging and menopause influence weight gain in general, they also affect weight gain after smoking cessation.

To augment this problem, smoking leads to earlier menopause. Smoking habits significantly predicted earlier menopause in a longitudinal study examining the transition from before to after menopause (Nilsson, Moller, Koster, & Hollnagel, 1997). The number of years of smoking was the strongest predictor; women who started smoking earlier were more likely to have an earlier menopause. Cramer and Xu (1996) concur with this result; in addition, they discovered that a higher number of pack-years also predicted early menopause. Willett et al.

(1983) reported that never-smokers experienced menopause 2 years later than those who smoked the most (≥ 35 cigarettes per day). Chiechi et al. (1997) found that female smokers went through menopause 1.5 years earlier than nonsmokers, while McKinlay, Brambilla, and Posner (1992) found the difference to be 1.8 years. They also found that smokers have an earlier and shorter perimenopause than non-smokers. Age-matched female smokers (40-50 years old) were about 30% more likely to be menopausal than nonsmokers and the risk of early menopause was also substantially greater (odds ratio 1.84, 95% C.L. 1.66-2.04) for women who had smoked for 30 or more years (Cramer, Harlow, Xu, Fraer, & Barbieri, 1995).

The effects of smoking are not limited to an earlier menopause, but they may also influence fat distribution. Smoking is also associated with higher abdominal fat deposition regardless of weight (Emery, Schmid, Kahn, & Filozof, 1993). Women who smoke and therefore tend to weigh less than nonsmoking counterparts nevertheless deposit more fat intra-abdominally. Thus, the effects of menopause at producing an increase in abdominal fat deposition can put women who smoke at an even greater risk for further increases in abdominal fat as well as greater weight gain.

Smoking Cessation, Caloric Intake, Macronutrient Effects and Research Methods

Increased Caloric Intake Following Smoking Cessation

Much of the literature regarding smoking cessation focuses on abstinence studies. Abstainers typically stop smoking for a short period of time during the study, anywhere from one week to one month as determined by the experimenter, attempting to mimic the effects of long-term smoking cessation. Abstinence studies use different populations than cessation studies; however, previous research shows similar effects on energy expenditure and caloric intake, leading abstinence research to be viewed as an important component of cessation research.

As previously discussed, smoking cessation often leads to weight gain; the increase in body weight after smoking cessation is attributable primarily to an increase in daily caloric intake. In a review of four studies done in their lab, Hatsukami, Hughes, and Pickens (1985) found that increased body weight and increased caloric intake were typical tobacco withdrawal symptoms. Abstainers showed greater total caloric intake as well as greater weight gain than continuing-smokers (Hatsukami, Hughes, & Pickens, 1985; Allen, Brintnell, Hatsukami, & Reich, 2004). Intake may increase from approximately 250-300 kcals per day within the first few weeks of smoking cessation, up to as much as 383 kcal per day (Perkins, 1993; Perkins, 1992). After a 48-day period of cessation, females gained a mean of 2.2 kg, 96% of which was fat (Stamford, Matter, Fell, & Papanek, 1986). Further analyses showed that 69% of this gain was attributable to increased caloric intake. Change in total caloric intake was found to be a predictor of weight gain after a two-week abstinence period, and greater increases in caloric intake were connected with greater weight gain (Vander Weg, Klesges, Eck Clemens, Meyers, & Pascale, 2001). Hence, the primary contributor to weight gain after smoking cessation is an increase in caloric intake.

As well as influencing postcessation weight gain, sex differences play a role in postcessation caloric intake as well. Female abstainers increased caloric intake significantly more than male abstainers over a three week period of smoking abstinence (Klesges, Eck, Clark, Meyers, & Hanson, 1990). Ogden (1994) compared intakes of snack foods during short-term abstinence. Female abstainers consumed significantly more calories than males who abstained. Gilbert and Pope (1982) also investigated snacking behavior. They found that women who abstained consumed 44% more calories than male abstainers. Consequently, females increase caloric intake after cessation to a greater extent than do males.

Macronutrient-Specific Effects

Different macronutrients affect total caloric intake in diverse ways. High protein and high complex carbohydrate (CCHO) foods increase feelings of fullness and satiation, and high sugar and high fat foods stimulate appetite. Foods high in both sugar and fat are the most provocative of appetite.

Rolls, Hetherington, & Burley (1988) investigated the effects of specific macronutrients on hunger and intake. Subjects were provided with equicaloric preloads that were high in either protein, complex carbohydrates, fat, sugar, or high in both fat and sugar. Consumption of high protein and high complex carbohydrates preloads decreased hunger ratings significantly more than consumption of high sugar, high fat, and high sugar/high fat preloads. High protein and high complex carbohydrates preloads also increased feelings of fullness more than the high sugar, high fat, and high sugar/high fat preloads, indicating that foods high in protein and complex carbohydrates decrease hunger and increase fullness more than foods high in other macronutrients. Two hours after eating the preload, subjects were presented with an *ad libitum* test lunch. In comparison with other preloads, consumption of the high protein and high complex carbohydrates preloads both led to significantly less caloric intake during the test lunch. The investigators concluded that high complex carbohydrates and high protein preloads produced greater changes in hunger and fullness as well as being the most satiating.

Measure of Macronutrient Intake in Smoking Cessation Literature

Many studies have attempted to measure macronutrient changes after smoking cessation. However, the results of these studies are inconsistent. They show increases in intake of fat, sugar, complex carbohydrates, or no change in intake, demonstrating irregularities in design and results. A simple but problematic method of measuring intake is self-reports of daily intake. However, methodological problems in using self-reports prevent findings regarding macronutrient changes

after smoking cessation from being an accurate description of intake. Several studies rely on subjects' dietary records to document daily macronutrient intake (Hall et al., 1989; Allen et al., 2004; Rodin, 1987; Vander Weg, Klesges, Clemens, Meyers, and Pascale, 2001; Klesges, Eck, Clark, Meyers, and Hanson, 1990). These methods depend upon the reporting by the participants, which studies show is not always veridical (Hetherington, 2002). Although participants are trained in weighing food and recording intake, subjects may underreport intake of specific macronutrients, especially fat. Obese subjects underreported food intake by 37% during the week that they recorded intake (Goris, Westerterp-Plantenga, & Westerterp, 2000). Non-obese subjects also report lower intake; both obese and nonobese subjects underreported daily energy intake even though they were properly trained to use measuring devices (Bandini, Schoeller, Cyr, & Dietz, 1990). A study on women aged 23-53 years demonstrated that 49% of subjects underreported energy intake by 21%, especially reporting of high fat and high sugar foods (Scagliusi, Polacow, Artioli, Benatti, & Lancha, 2003). In a cohort of middle-aged women, those who underreported recorded a diet lower in fat and higher in protein than those who adequately reported, demonstrating that fat is especially underreported in dietary records (Samaras, Kelly, & Campbell, 1999). A review of studies conducted using doubly-labeled water and intake measurement concludes that "it must be remembered that self-reported intakes are only estimates of true habitual intake and should be viewed as such" (Schoeller, 1990). Therefore, macronutrient intake must be directly measured in order to eliminate errors and underreporting high fat and high sugar foods.

Nevertheless, direct measures of intake in smoking cessation studies are also flawed. Some investigators fail to distinguish between sugars and complex carbohydrates in choosing foods to provide to participants as well in analyzing macronutrient intake. For example, in a study by Spring et al. (1991), the researchers did not separately analyze intake of complex

carbohydrates and sugar, but instead they used a total carbohydrate variable. Because of the difference in control of food intake between these two macronutrients as demonstrated by Rolls, Hetherington, & Burley (1988), distinguishing between them is essential. Another study failed to discriminate between complex carbohydrates and sugars, and it did not give information about what foods were available to participants (Hatsukami, LaBounty, Hughes, & Laine, 1993). If a disproportionate number of high CHO or high sugar foods were available, intake might have been skewed and would have biased the results.

Variation in levels of fat in test foods is also lacking in these studies using direct measures. Ogden (1994) classified snacks as “sweet” or “savory.” All were high in fat, so participants necessarily increased fat intake with greater caloric intake. In the test lunches that Spring et al. (1991) presented to subjects, foods were purposefully manipulated to have similar levels of fat. The majority of the foods had a moderate level of fat (24%-45%). Instead, both high and low fat foods should have been presented to subjects in order to measure intake of fat in relation to other macronutrients.

Moreover, the experimenters’ selection of test foods regarding given levels of fat is further problematic. Typically, researchers only give a limited number of foods, and the lack of variety leads to inconsistent results. This failure to find a consistent level of fat preference suggests that fat preference is food-specific (Mela & Marshall, 1992; Mela & Sachetti, 1991). Mela and Sachetti presented subjects with different foods, each varying in two to five levels of fat. Subjects rated the pleasantness of each food. Results showed that there was no consistent relationship regarding preferred levels of fat across foods, leading to the conclusion that fat preference is food-specific. Therefore, assessment of overall fat preference must include a wide variety of foods. Using foods representative of the typical American diet in combination with personal food preferences of the participant will display a more accurate picture of normal fat

preference and macronutrient intake. In addition, a paradigm that varies fat content with other macronutrients, i.e. sugar, complex carbohydrates, and protein, allows for detection of changes in intake of different macronutrients in relation to fat intake.

Furthermore, using this novel test meal model in conjunction with a measure of hedonic ratings of foods enables the researcher to get a clear picture of intake and satiation. Hedonic ratings are participants' subjective ratings of the pleasantness of the food. Before meal initiation, both hunger and hedonic ratings are high, and at the end of the meal the negative feedback from satiation causes hedonic ratings to decrease and the meal to end (Rolls et al., 1988). In addition, a questionnaire presenting numerous food choices following the same criteria as the meal paradigm allows the researcher to test a wider variety of foods than the test meal alone.

Specific Aims

The Geiselman Macronutrient Self-Selection Paradigm (MSSP) presents a reliable and valid measure of intake by systematically and significantly varying macronutrients across foods (Geiselman et al., 1998). The design varies fat across protein, sugar, and complex carbohydrates using foods common in the American diet. The Geiselman Food Preference Questionnaire (FPQ) complements the MSSP by using an identical paradigm that includes foods mutually exclusive to those served in the MSSP. Decreases in hedonic ratings pre- to postprandially demonstrate the negative feedback of satiation. In addition, testing participants before and after smoking cessation allows for assessment of the influence of smoking on satiation. These tests show strong test-retest reliability for macronutrient and total caloric intake in the MSSP and hedonic responses to the FPQ.

Specific Aim 1: To assess specific macronutrient intake and total caloric intake with the use of the Macronutrient Self-Selection Paradigm (MSSP) in postmenopausal women at baseline

(while still smoking) and following smoking cessation. We hypothesized that women would increase caloric intake as well as intake of high fat foods postcessation.

Specific Aim 2: To assess hedonic responses to foods listed on our Food Preference Questionnaire (FPQ) measured both pre- and postprandially to serve as an indication of negative feedback in postmenopausal women prior to and following smoking cessation. We hypothesized that women would be able to reach satiation more readily while smoking than they would postcessation.

Specific Aim 3: To assess whether or not there are differences in responses on the above measures in postmenopausal African-American versus Caucasian women.

Chapter 2: Method

Participants

For this study, we recruited 87 postmenopausal female smokers, 55 Caucasian women and 32 African-American women aged 45-59 years. In order to be included in this study, participants had to be postmenopausal for at least one year. Postmenopausal status was defined as having been amenorrheic for at least 12 months and having follicle stimulating hormone (FSH) levels greater than 30 mIU/ml if not taking hormone replacement therapy (Matthews et al., 1989; Kuller, Gutai, Meilahn, & Plantinga, 1990) or having surgical menopause. Smoking was defined as a self report of more than 10 cigarettes per day for one year or more, an expired CO level greater than 10 ppm, and a serum cotinine level greater than 25 ng/ml. All subjects were required to have written consent from a physician to participate in this study.

Exclusion Criteria

Participants could not have displayed a history or presence of significant psychiatric illness (e.g. eating disorders, psychosis, psychoactive substance abuse, major depression) or physical illness (e.g. renal failure, hepatic failure, cancer, immunological disease). Also, they could not have been enrolled in a standardized weight-reduction program or have been taking medications for weight loss.

Instruments

Macronutrient Self-Selection Paradigm

The Macronutrient Self-Selection Paradigm (MSSP) significantly and systematically varies fat and macronutrient content in foods presented to the participants as a meal. Many of the foods offered in the MSSP are from the top 10 sources of dietary fat in the United States, such as luncheon meats, baked goods, and bread products (Block, Dresser, Hartman, & Carroll, 1985).

The MSSP presents the participant with large portions of foods that vary in macronutrient content. These foods are typically snacks and other easily prepared items. The food choices are prepared as a 2 (Fat factor: High Fat and Low Fat) X 3 (Carbohydrate [CHO] factor: High Simple Sugar, High Complex CHO, and Low CHO/High Protein) X 3 (specific foods within each cell) design. The six cells are High Fat/High Simple Sugar (HF/HS), High Fat/High Complex Carbohydrate (HF/HCCHO), High Fat/Low Carbohydrate/High Protein (HF/HP), Low Fat/High Simple Sugar (LF/HS), Low Fat/High Complex Carbohydrate (LF/HCCHO), and Low Fat/Low Carbohydrate/High Protein (LF/HP).

Each food item in the three high-fat cells is $\geq 45\%$ fat (percentages are based on total kilocalories of a given food). Foods in the HF/HS cell are $\geq 45\%$ fat and $\geq 30\%$ sugar, foods in the HF/HCCHO cell are $\geq 45\%$ fat and $\geq 30\%$ complex carbohydrates, and foods in the HF/LCHO/HP cell are $\geq 45\%$ fat and $\geq 13\%$ protein, although most are 20-35% protein. Foods in the low fat cells are $< 20\%$ fat. Each subject is presented with three foods from each cell in the 2 X 3 X 3 design for a total of 18 foods for their meal.

Prior to the MSSP, the subjects completed a Food Selection Questionnaire (FSQ) to rate on a Likert scale the hedonic responses to 92 foods that conformed to the design of the MSSP. Each food fit into one of the six cells of the MSSP, and from this questionnaire the MSSP foods were chosen. The anchors of the scale were 1 = dislike extremely, 5 = neutral, neither like nor dislike, and 9 = like extremely. Foods given an intermediate score of 5-8 were presented to the participant; scores below a 5 indicated dislike and would probably not be eaten, and a score of 9 meant that the particular food might have been favored to the exclusion of others in the same cell. High- and low-fat varieties of the same foods were presented; for example, a subject was presented with both a high- and low-fat cheese, meat, bread, etc. Within each cell, each food was mutually exclusive of other foods presented in reference to the type of food; for example, only

one high-fat meat was presented, one high-fat cheese, etc. Foods were prepared in a variety of ways so as to acknowledge personal preferences in preparation; for example, cheese was cut into slices and cubes. Finally, mayonnaise and mustard were presented to each subject for the meal.

Macronutrient information as obtained from product labels if available or from Pennington's revision of *Bowes and Church's Food Values of Portions Commonly Used* (Pennington 1994). Intake was recorded in grams and converted to total kilocalories, then further divided into kilocalories of fat, sugar, complex carbohydrates, and protein. Summary data for each of the six cells and overall intake were recorded for each participant.

Food Preference Questionnaire

The Food Preference Questionnaire (FPQ) accompanies the MSSP and was developed according to the same design. The foods in the MSSP and the FPQ are mutually exclusive, allowing assessment of a wide variety of foods. Foods listed on the FPQ require substantial preparation and are therefore impractical to use on the MSSP test; examples are steaks, burgers, and ice cream. It is a 2 (Fat: High Fat and Low Fat) X 3 (CHO: High Simple Sugar, High Complex CHO, and Low CHO/High Protein) design employing the same guidelines for kilocalories of macronutrients per food as the MSSP. There are 72 foods in the questionnaire, with 12 foods listed from each of the cells. Each food is rated on the 9-point Likert scale.

Testing Procedures

Subjects were told not to eat or drink anything besides water after 10 pm the night before the MSSP test, to refrain from alcohol for 24 hours prior to the test, and not to exercise the morning of the test. This helped to ensure that all participants arrived in the same nutritional status for each test session. When the participant arrived at 10:30 am, she completed a questionnaire to determine if any condition, such as a cold or sinus infection, might have been interfering with her ability to smell or taste food. The questionnaire also asked if the subject had

complied with the instructions not to eat or drink past 10 pm the previous evening. The FPQ was then completed preprandially. Next, the subject was presented with 18 pre-weighed foods according to her responses on the FSQ and the guidelines of the MSSP. The order of food placement was randomized in order to limit researcher bias when placing the foods on the table. Water was also provided with the meal. The subject was told to eat as much or as little as she wanted until comfortably full, and to alert the researcher when she was finished. After finishing the meal the participant completed the FPQ once again, and the food was re-weighed to determine how much of each food was eaten. The participants were tested twice, once prior to smoking cessation, and again within one month of cessation.

Chapter 3: Results

Macronutrient Self-Selection Paradigm

A 2 X 2 ANOVA was conducted to investigate the relationship between race (African-American and Caucasian) and smoking status (baseline while still smoking and postcessation). Race was the between-subjects factor; smoking status was the within-subjects factor; and total kilocalories (kcal) intake was the dependent variable. Analyses yielded a significant race main effect ($F(1,85) = 9.4, p = 0.003$), indicating that African-American females ingested significantly more total kcal than Caucasian females across smoking status. As shown in Figure 1, African-American females consumed 968.8 total kcal, and Caucasian females consumed 795.8 total kcal. The smoking X race interaction was also significant ($F(1,85) = 6.882, p = 0.01$, see Figure 2). Post-hoc t-tests indicated that Caucasian females significantly increased total caloric intake from pre- to postcessation ($t(54) = 3.570, p = 0.001$). Caucasian females' total kcal intake at baseline was 743.5 kcal, whereas total kcal intake postcessation had increased to 848.1 kcal. However, African-American females total kcal intake did not differ between baseline ($m = 999.3$ kcal) and postcessation ($m = 938.2$ kcal; $t(31) = 0.927, p = 0.361$).

Difference in kcal intake of high fat foods and kcal intake of low fat foods across race and smoking status were examined with a 2 X 2 X 2 ANOVA. Race was the between-subjects factor; smoking status and fat content (high fat foods and low fat foods) were the within-subjects factors; and kcal intake was the dependent variable. This yielded a significant main effect for high fat foods versus low fat foods ($F(1,85) = 151.917, p = 0.001$), revealing that across the two levels of smoking status and the two levels of race, mean caloric intake of high fat foods ($m = 585.2$ kcal) was greater than mean caloric intake of low fat foods ($m = 225.3$ kcal, see Figure 3). Analyses also revealed a smoking X race X fat content interaction ($F(1,85) = 6.936, p = 0.01$). Post-hoc t-tests showed that Caucasian females increased kcal intake of high fat foods

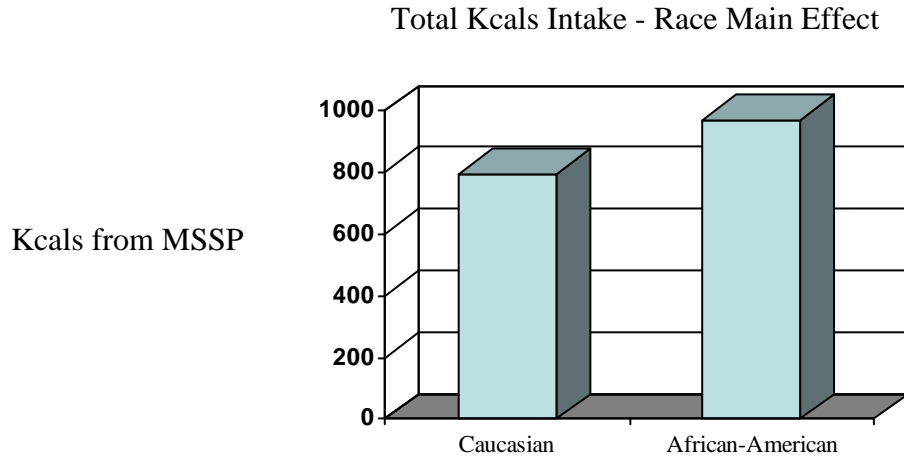


Figure 1. A race main effect shows that African-American females consumed significantly more total kcals than Caucasian females. African-American females consumed 968.8 total kcals (SE = ± 44.8 kcals), and Caucasian females consumed 795.8 total kcals (SE = ± 34.2 kcals).

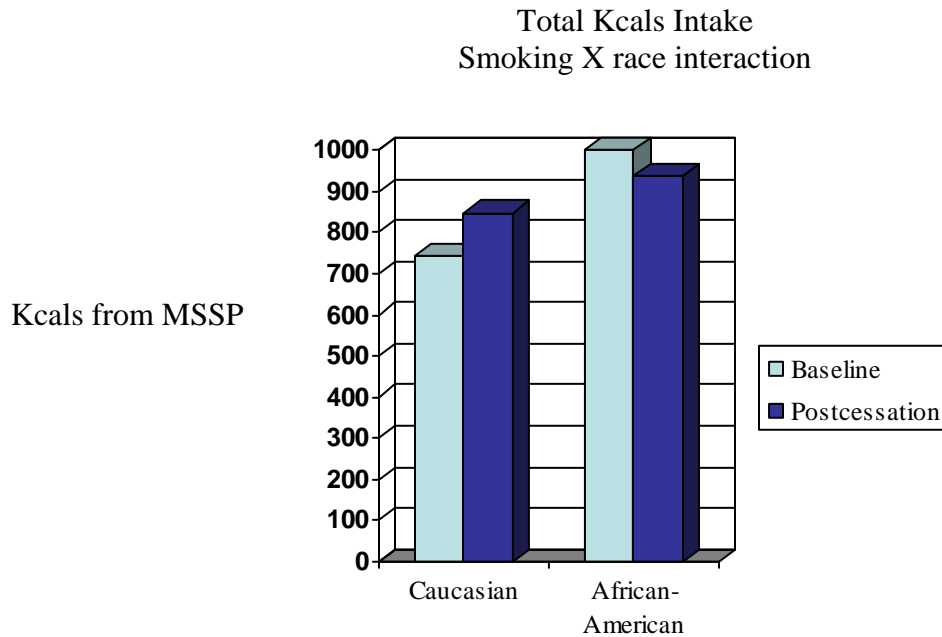


Figure 2. The smoking X race interaction demonstrates that Caucasian females' kcals intake increased from baseline (m = 743.5 kcals, SE = ± 42.0 kcals) to postcessation (m = 848.1 kcals, SE = ± 36.2 kcals), but African-American females' intake did not differ between baseline (m = 999.3 kcals, SE = ± 55.1 kcals) and postcessation (m = 938.2, SE = ± 47.4 kcals).

from baseline ($m = 485.8$ kcals) to postcessation ($m = 587.6$ kcals; $t(54) = 4.114$, $p = 0.001$) as seen in Figure 4. However, kcals intake of high fat foods did not differ between baseline ($m = 670.5$ kcals) and postcessation ($m = 597.0$ kcals) for African-American females ($t(31) = 1.141$, $p = 0.263$). Neither race showed a significant difference in kcals intake of low fat foods between baseline and postcessation (Caucasian females: $t(54) = 0.178$, $p = 0.859$, kcals intake low fat foods at baseline = 196.2, kcals intake low fat foods at postcessation = 198.4; African-American females: $t(31) = 0.019$, $p = 0.985$, kcals intake low fat foods at baseline = 253.1, kcals intake low fat foods at postcessation = 253.5).

A 2 X 2 X 2 X 3 ANOVA was conducted to examine the relationship between smoking, race, fat content and other macronutrients (sugar, complex carbohydrates (CCHO), and protein). Race was the between-subjects factor; smoking status, fat content, and other macronutrients were the within-subjects factors. The dependent variable was kcals intake. The ANOVA yielded a significant race X fat content X other macronutrients interaction ($F(2,170) = 3.527$, $p = 0.032$). Caucasian females' and African-American females' intake of each of the other macronutrients was assessed across fat levels in post-hoc tests. Analyses revealed that for each of the other macronutrients, Caucasian females ate significantly more high fat foods than low fat foods. As seen in Figure 5, Caucasian females' kcals intake of HF/HS foods ($m = 169.3$ kcals) was significantly higher than kcals intake of LF/HS foods ($m = 86.3$ kcals; $t(54) = 3.784$, $p = 0.001$). Their kcals intake of HF/HCCHO foods ($m = 175.1$ kcals) was significantly higher than kcals intake of LF/HCCHO foods ($m = 57.0$ kcals; $t(54) = 5.875$, $p = 0.001$). Finally, Caucasian females' kcals intake of HF/HP foods ($m = 191.5$ kcals) was significantly higher than kcals intake of LF/HP foods ($m = 54.9$ kcals; $t(54) = 9.475$, $p = 0.001$).

Kcals Intake of High and Low Fat Foods
Fat Main Effect

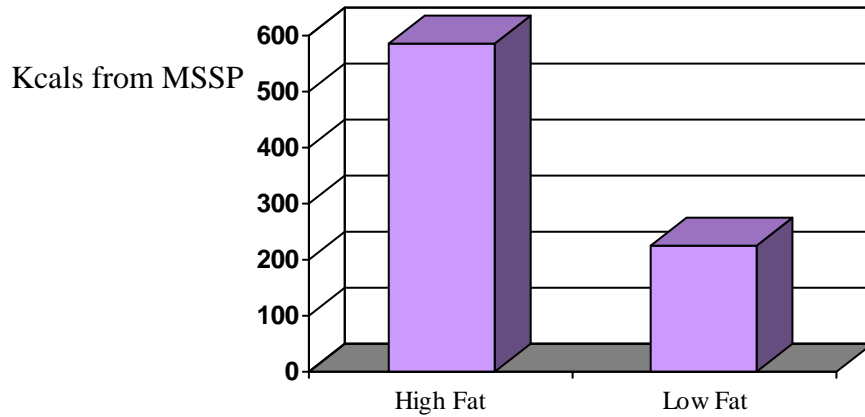


Figure 3. A main effect for high fat foods versus low fat foods demonstrates that mean caloric intake of high fat foods ($m = 585.2$ kcals, $SE = \pm 25.8$ kcals) was significantly greater than mean caloric intake of low fat foods ($m = 225.3$ kcals, $SE = \pm 11.0$ kcals)

Kcals Intake of High and Low Fat Foods– African-American vs. Caucasian
Smoking x Race x Fat

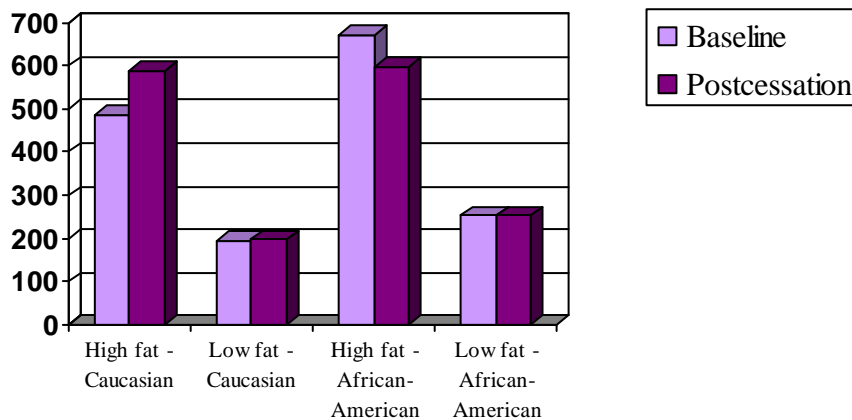


Figure 4. A smoking X race X fat content interaction reveals that Caucasian females increased kcals intake of high fat foods from baseline ($m = 485.8$ kcals, $SE = \pm 37.5$ kcals) to postcessation ($m = 587.6$ kcals, $SE = \pm 34.4$ kcals). However, African-American females kcals intake of high fat foods did not differ between baseline ($m = 670.5$ kcals, $SE = \pm 49.2$ kcals) and postcessation ($m = 597.0$ kcals, $SE = \pm 45.1$ kcals). Neither race significantly increased consumption of low fat foods.

Kcals Intake of HF/HS, LF/HS, HF/HCCHO, LF/HCCHO, HF/HP, LF/HP
Race x Fat x Other Macronutrients – Caucasian

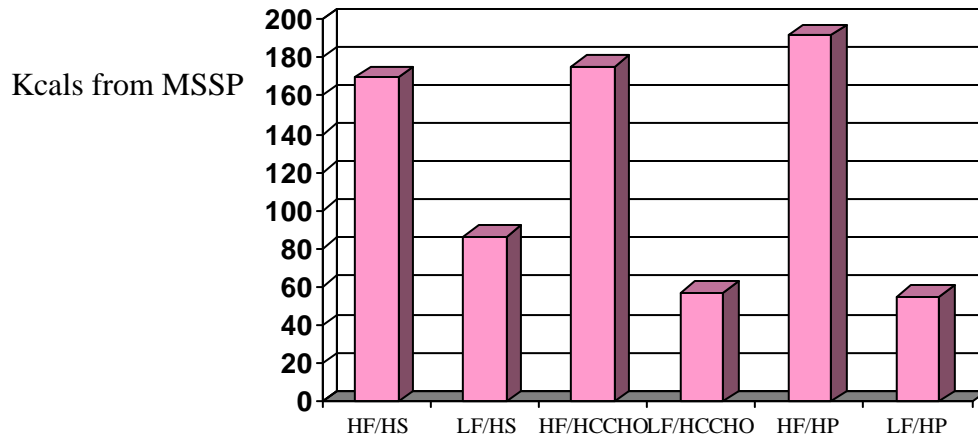


Figure 5. Caucasian females consumed significantly more kcals of HF/HS foods, HF/HCCHO foods, and HF/HP foods than low fat foods of each of the other macronutrients. Kcals intake of HF/HS ($m = 169.3$ kcals, $SE = \pm 20.0$ kcals) was significantly higher than kcals intake of LF/HS ($m = 86.3$ kcals, $SE = \pm 9.4$ kcals; $t(54) = 3.784$, $p = 0.001$). Kcals intake of HF/HCCHO foods ($m = 175.1$ kcals, $SE = \pm 17.5$ kcals) was significantly higher than kcals intake of LF/HCCHO foods ($m = 57.0$ kcals, $SE = \pm 8.2$ kcals; $t(54) = 5.875$, $p = 0.001$). Kcals intake of HF/HP foods ($m = 191.5$ kcals, $SE = \pm 17.0$ kcals) was significantly higher than kcals intake of LF/HP foods ($m = 54.9$, $SE = \pm 4.8$ kcals; $t(54) = 9.475$, $p = 0.001$).

African-American females showed a different pattern, as depicted in Figure 6. They consumed more kcals of HF/HCCHO foods ($m = 234.4$ kcals) than of LF/HCCHO foods ($m = 60.6$ kcals; $t(31) = 5.353$, $p = 0.001$) and more kcals of HF/HP foods ($m = 249.5$ kcals) than of LF/HP foods ($m = 64.9$ kcals; $t(31) = 6.403$, $p = 0.001$). They did not, however, consume significantly more kcals of HF/HS foods ($m = 149.9$ kcals) than LF/HS foods ($m = 127.8$ kcals; $t(31) = 0.695$, $p = 0.492$). African-American females consumed more HF/HCCHO foods and more HF/HP foods than LF/HCCHO foods and LF/HP foods, respectively. Unlike Caucasian females, they did not consume more HF/HS foods than LF/HS foods.

Food Preference Questionnaire

Hedonic ratings of macronutrients were assessed with 2X2X2 ANOVAs. The factors for each ANOVA were race, smoking status, and prandial (preprandial (before the MSSP lunch) and postprandial (after the MSSP lunch)). The dependent variable was hedonic ratings on the FPQ.

Kcals Intake of HF/HS, LF/HS, HF/HCCHO, LF/HCCHO, HF/HP, LF/HP
Race x Fat x Other Macronutrients – African-American

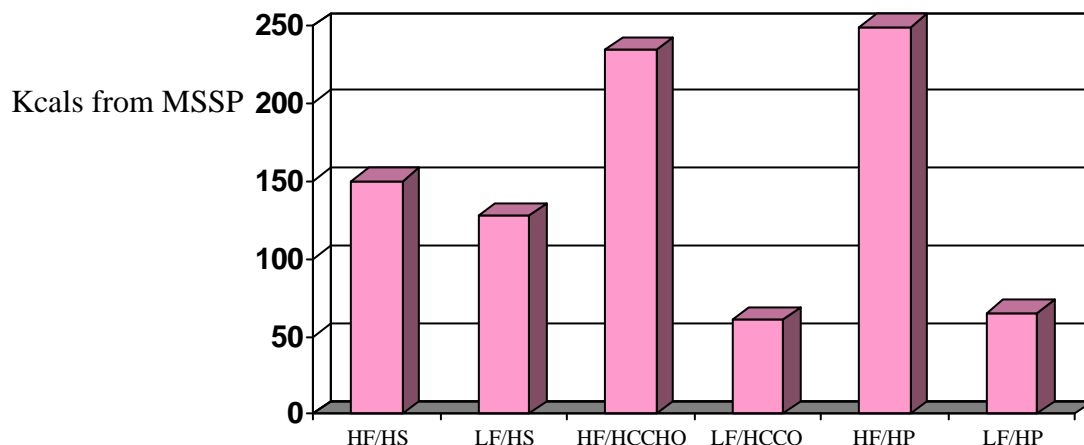


Figure 6. African-American females consumed more HF/HCCHO foods ($m = 234.4$ kcals, $SE = \pm 27.3$ kcals) than LF/HCCHO foods ($m = 60.6$ kcals, $SE = \pm 11.3$ kcals; $t(31) = 5.353$, $p = 0.001$) and more HF/HP foods ($m = 249.5$ kcals, $SE = \pm 27.3$ kcals) than LF/HP foods ($m = 64.9$, $SE = \pm 6.25$ kcals; $t(31) = 6.403$, $p = 0.001$), but they did not consume more HF/HS foods ($m = 149.9$, $SE = \pm 26.2$ kcals) than LF/HS foods ($m = 127.8$, $SE = \pm 14.4$ kcals; $t(31) = 0.695$, $p = 0.492$).

Responses to foods on the FPQ are on a scale from 1-9; the anchors of the scale are 1 = dislike extremely; 5 = neutral, neither like nor dislike; and 9 = like extremely. There was an additional column that the participant could mark if she had never tasted the given food. Responses in this column were scored as zero and were not included in the analyses. Analyses were conducted for hedonic ratings of HF foods, LF foods, HS foods, HCCHO foods, HP foods, HF/HS foods, HF/HCCHO foods, HF/HP foods, LF/HS foods, LF/HCCHO foods, and LF/HP foods.

High Fat Foods

The prandial main effect was significant ($F(1,84) = 97.078$, $p = 0.001$), indicating that hedonic ratings of HF foods decreased significantly from preprandial ($m = 5.66$) to postprandial ($m = 3.943$) status (see Table 1). The race main effect was a marginally non-significant trend ($F(1,84) = 3.259$, $p = 0.075$) indicating that African-American females ($m = 5.079$) show a trend towards reporting higher hedonic ratings of HF foods than Caucasian ($m = 4.524$) females. As

Table 1: Means (SE's) for Prandial and Race Main Effects for FPQ Analysis

Analysis	Prandial		Race	
	Pre	Post	Caucasian	African-American
HF	5.660*	3.943*	4.524†	5.079†
	(±0.149)	(± 0.200)	(± 0.185)	(± 0.246)
LF	5.745*	4.073*	4.319*	5.500*
	(± 0.131)	(± 0.201)	(± 0.178)	(± 0.239)
HS	5.692*	4.036*	4.456*	5.217*
	(± 0.154)	(± 0.201)	(± 0.189)	(± 0.251)
HCCHO	5.480*	3.784*	4.334*	4.930*
	(± 0.134)	(± 0.195)	(± 0.174)	(± 0.231)
HP	6.115*	4.209*	4.619*	5.704*
	(± 0.135)	(± 0.209)	(± 0.180)	(± 0.241)
HF/HS	5.493*	3.825*	4.493	4.825
	(± 0.202)	(± 0.217)	(± 0.225)	(± 0.300)
HF/HCCHO	5.434*	3.714*	4.428	4.720
	(± 0.156)	(± 0.200)	(± 0.187)	(± 0.249)
HF/HP	6.359*	4.315*	4.871*	5.803*
	(± 0.141)	(± 0.217)	(± 0.183)	(± 0.246)
LF/HS	5.908*	4.268*	4.428*	5.748*
	(± 0.148)	(± 0.208)	(± 0.191)	(± 0.256)
LF/HCCHO	5.559*	3.871*	4.295*	5.135*
	(± 0.130)	(± 0.200)	(± 0.175)	(± 0.231)
LF/HP	5.871*	4.128*	4.395*	5.604*
	(± 0.149)	(± 0.210)	(± 0.192)	(± 0.255)

*Designates a significant main effect. See text for p-value

†Designates a marginally non-significant trend.

seen in Figure 7, the race X prandial interaction was also significant ($F(1,84) = 5.021, p = 0.028$). To determine if there were significant differences between races in the changes in hedonic ratings from pre- to postprandial, the differences between preprandial and postprandial ratings for each race were compared in post-hoc analyses. This analysis revealed that Caucasian ($m = 2.107$) females decreased hedonic ratings from pre- to postprandial significantly more than did African-American ($m = 1.326$) females ($t(84) = 2.241, p = 0.028$).

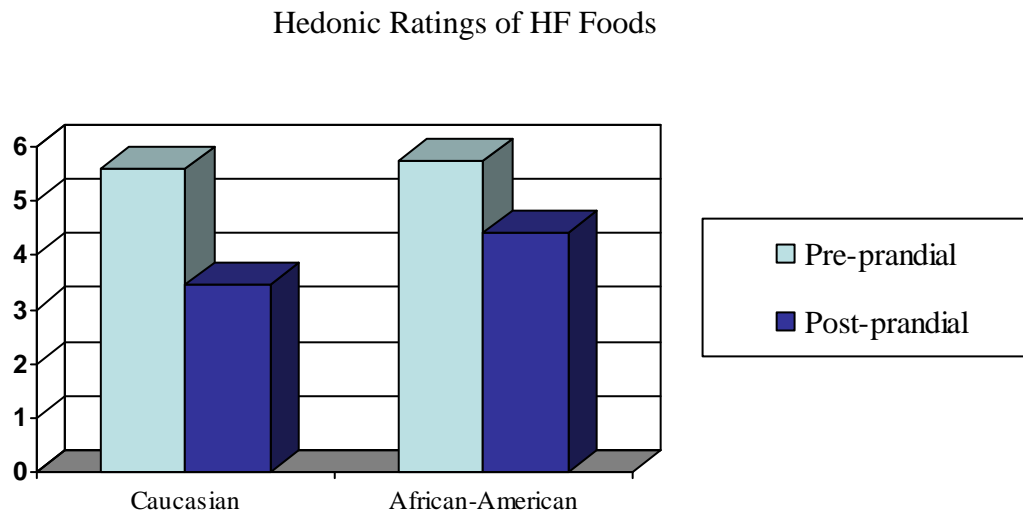


Figure 7. The race X prandial interaction was significant ($p = 0.028$). Post-hoc analyses revealed that Caucasian ($m = 2.107, SE = \pm 0.207$) females decreased hedonic ratings from pre- to post-prandial significantly more than did African-American ($m = 1.326, SE = \pm 0.284$) females.

Low Fat Foods

Hedonic ratings of LF foods revealed race and prandial main effects (Table 1). The race main effect ($F(1,82) = 15.747, p = 0.001$) indicated that African-American ($m = 5.5$) females reported higher hedonic ratings of LF foods than did Caucasian ($m = 4.319$) females. In addition, the prandial main effect ($F(1,82) = 104.786, p = 0.001$) suggested that hedonic ratings decreased significantly from preprandial ($m = 5.745$) to postprandial ($m = 4.073$) status.

High Sugar Foods

Ratings of HS foods indicated race and prandial main effects (Table 1). African-American ($m = 5.271$) females rated HS foods significantly higher than did Caucasian ($m =$

4.456) females ($F(1,84) = 6.723, p = 0.011$). Ratings of HS foods decreased significantly from preprandial ($m = 5.692$) to postprandial ($m = 4.036$) status ($F(1,84) = 94.29, p = 0.001$).

High Complex Carbohydrate Foods

Analyses of HCCHO foods revealed a significant race main effect ($F(1,81) = 4.255, p = 0.042$), indicating that African-American females ($m = 4.930$) rated HCCHO foods significantly higher than did Caucasian ($m = 4.334$) females (Table 1). Also, the prandial main effect was significant ($F(1,81) = 100.655, p = 0.001$; Table 1). Hedonic ratings decreased significantly from preprandial ($m = 5.480$) to postprandial ($m = 3.784$) status. Moreover, the race X prandial interaction was significant ($F(1,81) = 5.27, p = 0.024$) as seen in Figure 8. Post-hoc analyses revealed that Caucasian ($m = 2.083$) females had a significantly greater decrease from pre- to postprandial ratings than did African-American ($m = 1.307$) females ($t(81) = 2.296, p = 0.024$).

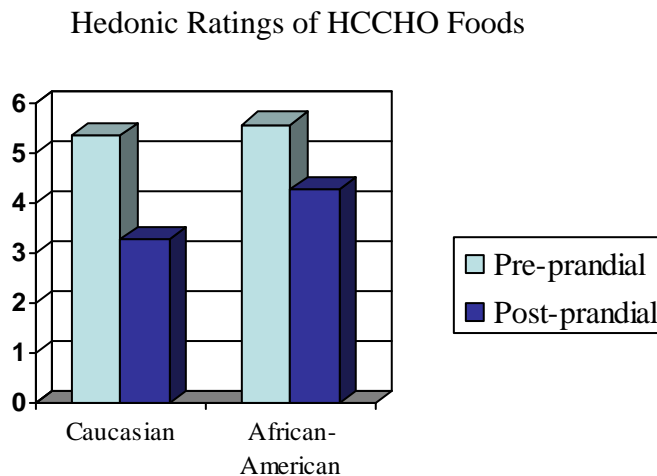


Figure 8. The race X prandial interaction was significant ($p = 0.024$). Post-hoc analyses revealed that Caucasian ($m = 2.083, SE = \pm 0.202$) females had a significantly greater decrease from pre- to post-prandial ratings than did African-American ($m = 1.307, SE = \pm 0.274$) females.

High Protein Foods

The significant race main effect for ratings of HP foods ($F(1,82) = 13.037, p = 0.001$) indicated that African-American ($m = 5.704$) females reported higher hedonic ratings of HP foods than did Caucasian ($m = 4.619$) females (Table 1). The significant prandial main effect (F

(1,82) = 109.386, $p = 0.001$) revealed that hedonic ratings of HP foods decreased significantly from preprandial ($m = 6.115$) to postprandial ($m = 4.209$) status (Table 1). Figure 9 shows that the race X prandial interaction was marginally non-significant ($F(1,82) = 3.145, p = 0.08$). Post-hoc analyses of this trend were also marginally non-significant ($t(82) = 1.773, p = 0.08$) suggesting that Caucasian ($m = 2.229$) females had a tendency to show a larger decrease from pre- to postprandial ratings than did African-American ($m = 1.583$) females.

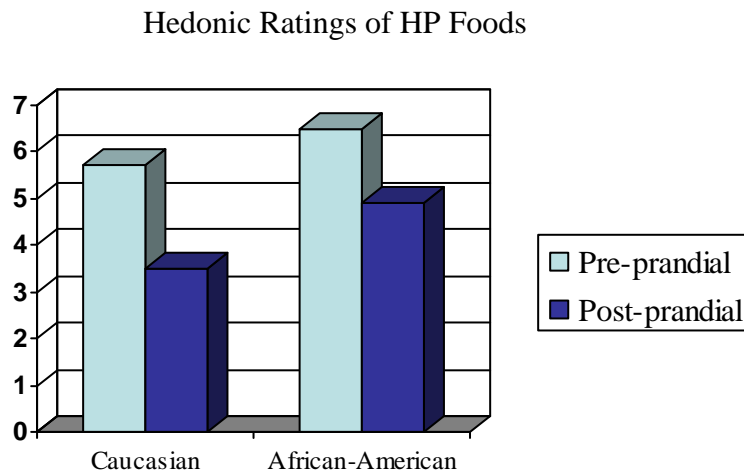


Figure 9. The race X prandial interaction was marginally non-significant ($p = 0.08$). Post-hoc analyses of this trend were also marginally non-significant ($p = 0.08$) suggesting that Caucasian ($m = 2.229, SE = \pm 0.203$) females had a tendency toward a larger decrease from pre- to post-prandial ratings than did African-American ($m = 1.583, SE = \pm 0.326$) females.

High Fat/High Sugar Foods

The significant prandial main effect ($F(1,84) = 80.045, p = 0.001$) indicated that hedonic ratings of HF/HS foods decreased significantly from preprandial ($m = 5.493$) to postprandial ($m = 3.825$) status (Table 1). The race X prandial interaction for hedonic ratings of HF/HS foods was marginally non-significant ($F(1,84) = 3.557, p = 0.063$, Figure 10). Post-hoc tests of this trend were also marginally non-significant ($t(84) = 1.886, p = 0.063$), suggesting that Caucasian ($m = 2.019$) females had a tendency to show a larger decrease from pre- to postprandial ratings than African-American ($m = 1.316$) females.

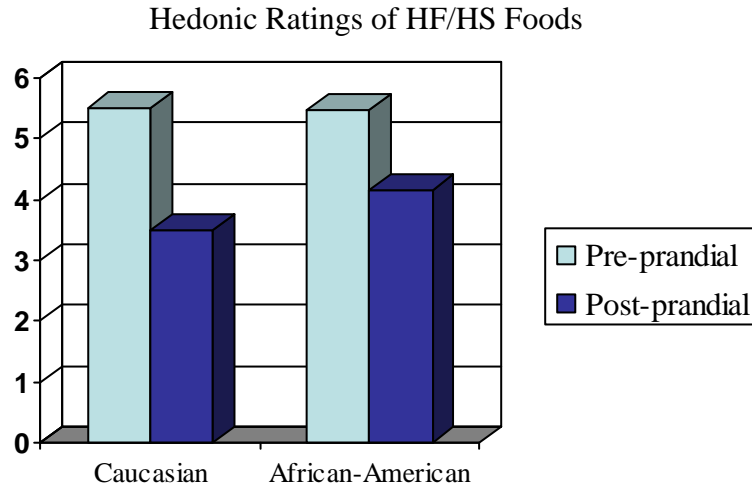


Figure 10. The race X prandial interaction for hedonic ratings of HF/HS foods was marginally non-significant ($p = 0.063$). Post-hoc tests of this trend were also marginally non-significant ($p = 0.063$), suggesting that Caucasian ($m = 2.019$, $SE = \pm 0.229$) females had a tendency to show a larger decrease from pre- to post-prandial ratings than African-American ($m = 1.316$, $SE = \pm 0.286$) females.

High Fat/High Complex Carbohydrate Foods

The significant prandial main effect ($F(1,81) = 93.065$, $p = 0.001$) indicated that hedonic ratings of HF/HCCCHO foods decreased significantly from preprandial ($m = 5.434$) to postprandial ($m = 3.714$) status (Table 1). As seen in Figure 11, the interaction of race X prandial was also significant ($F(1,81) = 7.258$, $p = 0.009$). Post-hoc tests ($t(81) = 2.694$, $p = 0.009$) revealed that Caucasian ($m = 2.202$) females had a significantly greater decrease in hedonic ratings from pre- to postprandial than did African-American ($m = 1.24$) females.

High Fat/High Protein Foods

The significant race main effect for HF/HP foods ($F(1,82) = 9.242$, $p = 0.003$) indicated that African-American females ($m = 5.803$) rated HF/HP foods significantly higher than Caucasian females ($m = 4.871$) (Table 1). Hedonic ratings of HF/HP foods decreased significantly from preprandial ($m = 6.359$) to postprandial ($m = 4.315$) status, as shown by the significant prandial main effect ($F(1,82) = 104.974$, $p = 0.001$) (Table 1). A marginally non-significant trend is shown in the race X prandial interaction ($F(1,82) = 3.514$, $p = 0.064$) as seen

in Figure 12. Post-hoc analyses revealed a marginally non-significant trend ($t(82) = 1.875, p = 0.064$), suggesting that Caucasian ($m = 2.419$) females had a tendency to decrease hedonic ratings of HF/HP foods from pre- to postprandial to a greater degree than did African-American ($m = 1.671$) females.

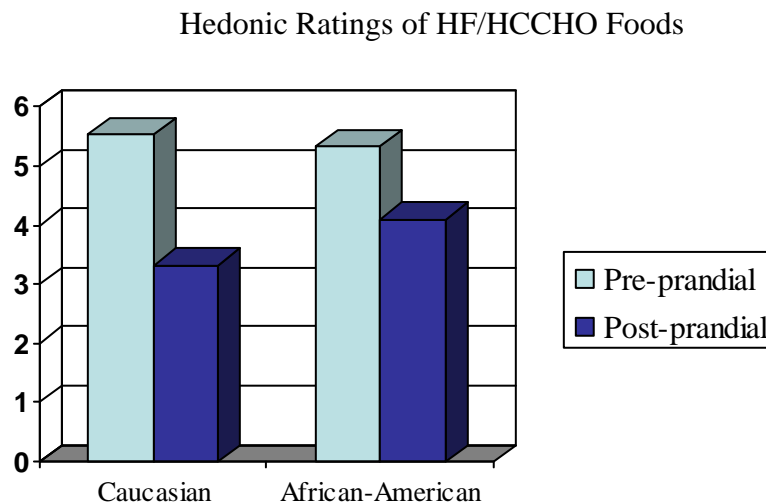


Figure 11. The race X prandial interaction for hedonic ratings of HF/HCCHO foods was significant ($p = 0.009$). Post-hoc tests ($p = 0.009$) revealed that Caucasian ($m = 2.202, SE = \pm 0.222$) females had a significantly greater decrease in hedonic ratings from pre- to post-prandial than did African-American ($m = 1.24, SE = \pm 0.266$) females.

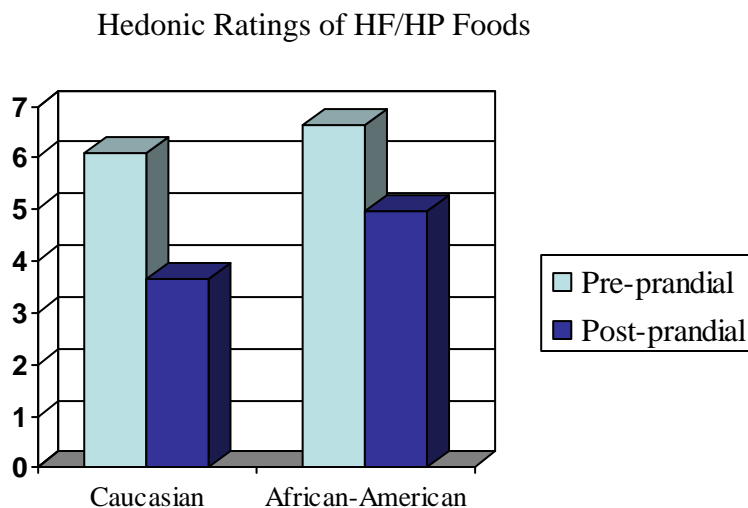


Figure 12. A marginally non-significant trend is shown in the race X prandial interaction ($p = 0.064$). Post-hoc analyses revealed a marginally non-significant trend ($p = 0.064$), suggesting that Caucasian ($m = 2.419, SE = \pm 0.229$) females had a tendency to decrease hedonic ratings of HF/HP foods from pre- to post-prandial to a greater degree than did African-American ($m = 1.671, SE = \pm 0.343$) females.

Low Fat/High Sugar Foods

Hedonic ratings of LF/HS foods revealed significant race and prandial main effects (Table 1). The significant race main effect ($F(1,82) = 17.138, p = 0.001$) revealed that African-American females ($m = 5.748$) rated LF/HS foods significantly higher than did Caucasian females ($m = 4.428$). The prandial main effect ($F(1,82) = 92.869, p = 0.001$) indicated that hedonic ratings of LF/HS foods decreased significantly from preprandial ($m = 5.908$) to postprandial ($m = 4.268$) status.

Low Fat/High Complex Carbohydrate Foods

The race main effect was significant for hedonic ratings of LF/HCCHO foods ($F(1,80) = 8.401, p = 0.005$), indicating that African-American ($m = 5.135$) females rated LF/HCCHO foods lower than Caucasian ($m = 4.295$) females (Table 1). The prandial main effect ($F(1,80) = 96.215, p = 0.001$) showed hedonic ratings of LF/HCCHO foods decreased significantly from preprandial ($m = 5.559$) to postprandial ($m = 3.871$) status (Table 1). The race X prandial interaction was a marginally non-significant trend ($F(1,80) = 3.194, p = 0.078$) as seen in Figure 13. Post-hoc tests revealed a marginally non-significant trend ($t(80) = 1.787, p = 0.078$); Caucasian ($m = 1.996$) females had a tendency to show larger decreases in hedonic ratings from pre- to postprandial than did African-American ($m = 1.381$) females.

Low Fat/High Protein Foods

The race main effect for LF/HP foods ($F(1,81) = 14.34, p = 0.001$) revealed that African-American females ($m = 5.604$) rated LF/HP foods significantly higher than Caucasian females ($m = 4.395$) (Table 1). The prandial main effect ($F(1,81) = 98.474, p = 0.001$) indicated that hedonic ratings of LF/HP foods decreased significantly from preprandial ($m = 5.871$) to postprandial ($m = 4.128$) status (Table 1). As seen in Figure 14, the race X prandial interaction was a marginally non-significant trend ($F(1,81) = 2.797, p = 0.098$). Post-hoc tests were also

marginally non-significant ($t(81) = 1.672, p = 0.098$); Caucasian ($m = 2.037$) females had tendency to show greater decreases in hedonic ratings from pre- to postprandial than did African-American ($m = 1.449$) females.

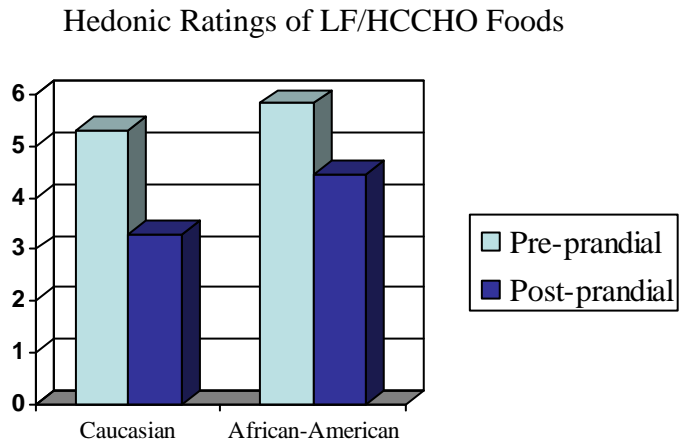


Figure 13. The race X prandial interaction was a marginally non-significant trend ($p = 0.078$). Post-hoc tests revealed a marginally non-significant trend ($p = 0.078$); Caucasian ($m = 1.996, SE = \pm 0.193$) females had a tendency toward larger decreases in hedonic ratings from pre- to post-prandial than did African-American ($m = 1.381, SE = \pm 0.306$) females.

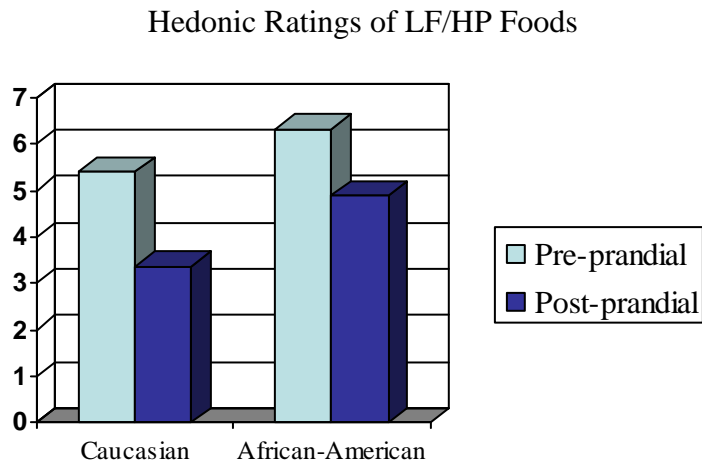


Figure 14. The race X prandial interaction was a marginally non-significant trend ($p = 0.098$). Post-hoc tests were also marginally non-significant ($p = 0.098$); Caucasian ($m = 2.037, SE = \pm 0.188$) females had a tendency to show greater decreases in hedonic ratings from pre- to post-prandial than did African-American ($m = 1.449, SE = \pm 0.329$) females.

Chapter 4: Discussion

We found that Caucasian females significantly increased intake of total kcals following smoking cessation. This concurs with prior studies that have found that participants increased total caloric intake after smoking cessation (Hatsukami et al., 1985; Ogden, 1994; Allen et al., 2004; Perkins, 1993; Perkins, 1992). In the current study, this increase in total kcals observed in Caucasian women was due specifically to an increase in consumption of kcals of HF foods. Caucasian females' intake of kcals of LF foods did not change from pre- to postcessation. Prior studies assessing changes in macronutrient intake following smoking cessation have been plagued with methodological errors that make it difficult to interpret the effect of smoking cessation on macronutrient intake. Our laboratory is the first to assess postcessation changes in macronutrient intake using a measure that significantly and systematically varies macronutrients across foods in order to evaluate macronutrient intake. This finding establishes the importance of increased intake of HF foods in postcessation hyperphagia in Caucasian females.

Increased consumption of high-fat foods in Caucasian females after smoking cessation would be expected to put these women at a greater risk for weight gain. Dietary fat does, in fact, play a role in overweight and obesity, and this effect is mediated by hyperphagia (Bray & Popkin, 1998; Lissner & Heitman, 1995). Fat contributes to overeating due to its high level of palatability, its weak effects on satiation and satiety, and its high caloric density. Humans prefer higher levels of fat. As seen in a study by Drewnowski (1983), participants rated the pleasantness of different levels of fat and sugar in milk/cream mixtures. Preference ratings (palatability) for the mixtures increased with increasing levels of fat, but no effect was seen for sugar.

Also, many studies found that subjects will overeat when presented with high-fat foods. These studies indicate the weak effect of high-fat foods on satiation. For example, Green and Blundell (1996b) found that participants presented with *ad libitum* access to high-fat snacks

consumed significantly more kcals than participants presented with high-carbohydrate snacks. Lawton et al. (1993) found similar effects in obese females. When presented with *ad libitum* access to either high-fat or high-carbohydrate foods, subjects ingested significantly more kcals when high-fat foods were offered than they did when high-carbohydrate foods were offered. Also, subjects presented with either a high-density or a low-density preload increased caloric intake significantly more when offered high-fat snacks than when offered high-carbohydrate snacks (Green & Blundell, 1996a). Furthermore, subjects given high-fat snacks did not decrease energy intake the rest of the day or have significantly different hunger ratings than subjects who ingested the high-carbohydrate snacks despite the increase in energy consumed from the high-fat snack. These postprandial results following the consumption of the high-fat snacks demonstrate the weak effect of fat on satiety.

In addition, high-fat foods are more calorically dense than low-fat foods. Fat contains 9 kcals/gram and carbohydrates and proteins each contain 4 kcals/gram. Consumption of high-fat foods promotes positive energy balance and weight gain (Lissner & Heitmann, 1995). Thus, because of its high level of palatability, its weak effect on satiation and satiety, and its higher caloric density, increased consumption of high-fat foods after smoking cessation may put Caucasian females at a greater risk for weight gain.

African-American females in the present study did not change the amount of total kcals consumed after smoking cessation. This is contrary to the majority of the literature showing an increase in food intake following smoking cessation (Hatsukami, Hughes, & Pickens 1985; Allen, Brintnell, Hatsukami, & Reich, 2004; Perkins 1993; Perkins 1992). However, most studies have been conducted using primarily Caucasians and therefore the effect of smoking cessation on food intake in African-Americans is not clear. In addition to the lack of changes in consumption of total kcals seen in the current study, no changes were seen in intake of kcals of HF foods

following smoking cessation in African-American females. However, it is noteworthy that the race main effect showed that, regardless of smoking status, African-American females ingested significantly more total kcals and significantly more kcals of HF foods than Caucasian females. Caucasian females increased total kcals consumed from 743.5 kcals to 848.1 kcals from pre- to postcessation, whereas at baseline, African-American females ingested 999.3 kcals and postcessation they consumed 938.2 kcals. Caucasian females specifically increased their consumption of HF foods from 485.8 kcals of at baseline to 587.6 kcals postcessation. African-American females ingested 670.5 kcals of HF foods before cessation and 597.0 kcals after cessation. As African-American females were already consuming significantly more kcals at baseline than was observed in the Caucasian females' increased consumption postcessation, the present results may represent a ceiling effect for the African-American women.

During a meal, "the pleasantness of a food does not remain constant but instead decreases as the food is consumed" (Rolls et al., 1988). In both humans and animals (Berridge, 1991) the pleasantness of food decreases after ingestion. In humans, reports of pleasure ratings, or hedonic ratings, are measured to determine the effect of this change that is produced by the negative feedback from the meal. At the termination of a meal, hedonic ratings of food are significantly lower than before the initiation of the meal, indicating that the pleasure received from the food has decreased. This loss of pleasure is thought to be a significant contributor to meal termination. As the subject reaches meal termination, or satiation, the food loses pleasantness, and the hedonic ratings decrease (Cabanac & Lafrance, 1990). In accordance with these findings, participants in this study rated foods as less pleasant at the termination of the meal than they did immediately prior to the meal. This effect was found, regardless of race and smoking status, in all analyses conducted. In other words, for each analysis (HF, LF, HS, HCCHO, HP, HF/HS,

HF/HCCHO, HF/HP, LF/HS, LF/HCCHO, LF/HP), the hedonic ratings of the foods presented on the FPQ decreased significantly from pre- to postprandial.

As expected, both African-American and Caucasian females decreased hedonic ratings of HF foods from pre- to postprandial. However, Caucasian females demonstrated a significantly greater decrease in hedonic responses to foods, especially HF foods. These FPQ results indicate that foods, especially HF foods, were less satiating in African-American females than they were in Caucasian females. The lesser satiating effects observed in African-American females was associated with significantly greater kcals intake and greater intake of HF foods in African-American females than in Caucasian females.

The pre- to postprandial decrease in hedonic responses on the FPQ did not change for either race from pre- to postcessation. These results suggest that both races achieved the same degree of satiation while they were still smoking as they did postcessation. The African-American women showed no change in food intake in the MSSP from pre- to postcessation. Thus, for the African-American women, neither their total caloric intake, including the intake of HF foods, nor their satiation changed from pre- to postcessation. However, this was not the effect that we observed in the Caucasian women. The Caucasian women showed a significant increase in total caloric intake from pre- to postcessation, and this was due specifically to an increase in consumption of HF foods. Thus, the Caucasian women had to consume significantly more total caloric intake from HF foods postcessation to reach the same level of satiation that they achieved with the consumption of much smaller amounts of food while they were still smoking. Hence, these results suggest that a decreased capability of achieving satiation may be responsible for the increase in total caloric intake, specifically due to increased consumption of HF foods, following smoking cessation in Caucasian, postmenopausal women.

As previously noted, nicotine leads to hypophagia and reduced weight; whereas, smoking cessation leads to weight gain, and this effect is mediated by hyperphagia (Miyata, Meguid, Fetissof, Torelli, & Kim, 1999), Levin et al., 1993, Zhang et al., 2001). Nicotine's anorectic effects are mediated by the central nervous system, primarily stemming from its effects on hypothalamic monoamines, specifically dopamine (DA) and serotonin (5-HT) (Miyata et al., 1999; Zhang et al., 2001). DA modulates feeding, specifically meal initiation, food intake, and body weight maintenance; whereas, 5-HT controls satiation, food intake, and body weight maintenance (Meguid et al., 2000). Using animal models, Yang et al. (1999) found that eating led to increases in hypothalamic DA and 5-HT. Moreover, nicotine administration caused an increase in hypothalamic DA and 5-HT and significantly decreased food intake.

The present study did not address meal initiation (which is influenced by DA) but, rather, addressed meal termination; that is, satiation. As noted above, serotonin has been reported to be associated with satiation and a decrease in food intake. Moreover, 5-HT has been reported to specifically decrease intake of high-fat foods (Smith, York, & Bray, 1999). These serotonergic effects in producing satiation and decreasing food intake, especially intake of high-fat foods, are the inverse of the ingestive results obtained in Caucasian women postcessation in the present study. One would expect a postcessation decrease in serotonergic activity, as it has been shown in rodents that nicotine administration into the lateral hypothalamus produces increases release of serotonin and that discontinuation of chronic nicotine administration produces a decrease in hypothalamic serotonin (Miyata et al., 1999; Yang et al., 1999). Thus, it is suggested that a decrease in 5-HT activity, which would be expected following smoking cessation, may be a potential cause of the decreased capability of achieving satiation and the resultant increase in total caloric intake, specifically due to increased consumption of high-fat foods, observed in the Caucasian women postcessation in the present study.

References

- Allen, S. S., Brintnell, D. M., Hatsukami, D., & Reich, B. (2004). Energy intake and physical activity during short-term smoking cessation in postmenopausal women. *Addictive Behaviors, 29*(5), 947-951.
- Aloia, J. F., Vaswani, A., Russo, L., Sheehan, M., & Flaster, E. (1995). The influence of menopause and hormonal replacement therapy on body cell mass and body fat mass. *American Journal of Obstetrics and Gynecology, 172*(3), 896-900.
- Bandini, L. G., Schoeller, D. A., Cyr, H. N., & Dietz, W. H. (1990). Validity of reported energy intake in obese and nonobese adolescents. *The American Journal Of Clinical Nutrition, 52*(3), 421-425.
- Becona, E., & Vazquez, F. L. (1998). Smoking cessation and weight gain in smokers participating in a behavioral treatment at 3-year follow-up. *Psychological Reports, 82*(3 Pt 1), 999-1005.
- Berridge, K. C. (1991). Modulation of taste affect by hunger, caloric satiety, and sensory-specific satiety in the rat. *Appetite, 16*(2), 103-120.
- Block, G., Dresser, C. M., Hartman, A. M., & Carroll, M. D. (1985). Nutrient sources in the American diet: quantitative data from the NHANES II survey. II. Macronutrients and fats. *American Journal of Epidemiology, 122*(1), 27-40.
- Bray, G. A., & Popkin, B. M. (1998). Dietary fat intake does affect obesity! *Am J Clin Nutr, 68*(6), 1157-1173.
- Burnette, M. M., Meilahn, E., Wing, R. R., & Kuller, L. H. (1998). Smoking cessation, weight gain, and changes in cardiovascular risk factors during menopause: the Healthy Women Study. *American Journal of Public Health, 88*(1), 93-96.
- Caan, B., Coates, A., Schaefer, C., Finkler, L., Sternfeld, B., & Corbett, K. (1996). Women gain weight 1 year after smoking cessation while dietary intake temporarily increases. *Journal of the American Dietetic Association, 96*(11), 1150-1155.
- Cabanac, M., & Lafrance, L. (1990). Postingestive alliesthesia: the rat tells the same story. *Physiol Behav, 47*(3), 539-543.
- Chiechi, L. M., Ferreri, R., Granieri, M., Lobascio, A., Bianco, G., Berardesca, C., et al. (1997). Smoking and menopause. *Clinical and Experimental Obstetrics & Gynecology, 24*(1), 26-27.
- Cramer, D. W., Harlow, B. L., Xu, H., Fraer, C., & Barbieri, R. (1995). Cross-sectional and case-controlled analyses of the association between smoking and early menopause. *Maturitas, 22*(2), 79-87.
- Cramer, D. W., & Xu, H. (1996). Predicting age at menopause. *Maturitas, 23*(3), 319-326.

- Emery, E. M., Schmid, T. L., Kahn, H. S., & Filozof, P. P. (1993). A review of the association between abdominal fat distribution, health outcome measures, and modifiable risk factors. *American Journal of Health Promotion, 7*(5), 342-353.
- Flegal, K. M., Carroll, M. D., Kuczmarski, R. J., & Johnson, C. L. (1998). Overweight and obesity in the United States: prevalence and trends, 1960-1994. *International Journal of Obesity and Related Metabolic Disorders, 22*(1), 39-47.
- Froom, P., Kristal-Boneh, E., Melamed, S., Gofer, D., Benbassat, J., & Ribak, J. (1999). Smoking cessation and body mass index of occupationally active men: the Israeli CORDIS Study. *American Journal of Public Health, 89*(5), 718-722.
- Geiselman, P. J., Anderson, A. M., Dowdy, M. L., West, D. B., Redmann, S. M., & Smith, S. R. (1998). Reliability and validity of a macronutrient self-selection paradigm and a food preference questionnaire. *Physiology & Behavior, 63*(5), 919-928.
- Gilbert, R. M., & Pope, M. A. (1982). Early effects of quitting smoking. *Psychopharmacology, 78*(2 (Print)), 121-127.
- Goris, A. H., Westerterp-Plantenga, M. S., & Westerterp, K. R. (2000). Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. *The American Journal Of Clinical Nutrition, 71*(1 (Print)), 130-134.
- Green, S. M., & Blundell, J. E. (1996a). Effect of fat- and sucrose-containing foods on the size of eating episodes and energy intake in lean dietary restrained and unrestrained females: potential for causing overconsumption. *Eur J Clin Nutr, 50*(9), 625-635.
- Green, S. M., & Blundell, J. E. (1996b). Subjective and objective indices of the satiating effect of foods. Can people predict how filling a food will be? *European Journal of Clinical Nutrition, 50*(12), 798-806.
- Hall, S. M., McGee, R., Tunstall, C., Duffy, J., & Benowitz, N. (1989). Changes in food intake and activity after quitting smoking. *Journal of Consulting and Clinical Psychology, 57*(1), 81-86.
- Hatsukami, D., Hughes, J. R., & Pickens, R. (1985). Characterization of tobacco withdrawal: physiological and subjective effects. *NIDA Research Monograph, 53*, 56-67.
- Hatsukami, D., LaBounty, L., Hughes, J., & Laine, D. (1993). Effects of tobacco abstinence on food intake among cigarette smokers. *Health Psychology, 12*(6), 499-502.
- Hetherington, M. M. (2002). The physiological-psychological dichotomy in the study of food intake. *Proc Nutr Soc, 61*(4), 497-507.
- Klesges, R. C., Eck, L. H., Clark, E. M., Meyers, A. W., & Hanson, C. L. (1990). The effects of smoking cessation and gender on dietary intake, physical activity, and weight gain. *International Journal of Eating Disorders, 9*(4), 435-446.

- Klesges, R. C., Ward, K. D., Ray, J. W., Cutter, G., Jacobs, D. R., Jr., & Wagenknecht, L. E. (1998). The prospective relationships between smoking and weight in a young, biracial cohort: the Coronary Artery Risk Development in Young Adults Study. *Journal of Consulting and Clinical Psychology, 66*(6), 987-993.
- Kuller, L. H., Gutai, J. P., Meilahn, E., Matthews, K. A., & Plantinga, P. (1990). Relationship of endogenous sex steroid hormones to lipids and apoproteins in postmenopausal women. *Arteriosclerosis, 10*(6), 1058-1066.
- Levin, E. D., Briggs, S. J., Christopher, N. C., & Rose, J. E. (1993). Sertraline attenuates hyperphagia in rats following nicotine withdrawal. *Pharmacol Biochem Behav, 44*(1), 51-61.
- Lissner, L., & Heitmann, B. L. (1995). Dietary fat and obesity: evidence from epidemiology. *Eur J Clin Nutr, 49*(2), 79-90.
- Macdonald, H. M., New, S. A., Campbell, M. K., & Reid, D. M. (2003). Longitudinal changes in weight in perimenopausal and early postmenopausal women: effects of dietary energy intake, energy expenditure, dietary calcium intake and hormone replacement therapy. *International Journal of Obesity and Related Metabolic Disorders, 27*(6), 669-676.
- Matthews, K. A., Meilahn, E., Kuller, L. H., Kelsey, S. F., Caggiula, A. W., & Wing, R. R. (1989). Menopause and risk factors for coronary heart disease. *The New England Journal of Medicine, 321*(10), 641-646.
- McKinlay, S. M., Brambilla, D. J., & Posner, J. G. (1992). The normal menopause transition. *Maturitas, 14*(2), 103-115.
- Mela, D. J., & Marshall, R. J. (1992). Sensory properties and perception of fats. In D. J. Mela (Ed.), *Dietary fats: Determinants of preference, selection and consumption* (pp. 43-57). London: Elsevier Applied Science.
- Mela, D. J., & Sacchetti, D. A. (1991). Sensory preferences for fats: relationships with diet and body composition. *The American Journal of Clinical Nutrition, 53*(4), 908-915.
- Miyata, G., Meguid, M. M., Fetissov, S. O., Torelli, G. F., & Kim, H. J. (1999). Nicotine's effect on hypothalamic neurotransmitters and appetite regulation. *Surgery, 126*(2), 255-263.
- Nilsson, P., Moller, L., Koster, A., & Hollnagel, H. (1997). Social and biological predictors of early menopause: a model for premature aging. *Journal of Internal Medicine, 242*(4), 299-305.
- O'Hara, P., Connett, J. E., Lee, W. W., Nides, M., Murray, R., & Wise, R. (1998). Early and late weight gain following smoking cessation in the Lung Health Study. *American Journal of Epidemiology, 148*(9), 821-830.
- Ogden, J. (1994). Effects of smoking cessation, restrained eating, and motivational states on food intake in the laboratory. *Health Psychology, 13*(2), 114-121.

- Pasquali, R., Casimirri, F., Labate, A. M., Tortelli, O., Pascal, G., Anconetani, B., et al. (1994). Body weight, fat distribution and the menopausal status in women. The VMH Collaborative Group. *International Journal of Obesity and Related Metabolic Disorders*, 18(9), 614-621.
- Perkins, K. A. (1992). Effects of tobacco smoking on caloric intake. *British Journal of Addiction*, 87(2), 193-205.
- Perkins, K. A. (1993). Weight gain following smoking cessation. *Journal of Consulting and Clinical Psychology*, 61(5), 768-777.
- Rodin, J. (1987). Weight change following smoking cessation: the role of food intake and exercise. *Addictive Behaviors*, 12(4), 303-317.
- Rolls, B. J., Hetherington, M., & Burley, V. J. (1988). The specificity of satiety: the influence of foods of different macronutrient content on the development of satiety. *Physiology & Behavior*, 43(2), 145-153.
- Samaras, K., Kelly, P. J., & Campbell, L. V. (1999). Dietary underreporting is prevalent in middle-aged British women and is not related to adiposity (percentage body fat). *International Journal of Obesity and Related Metabolic Disorders*, 23(8), 881-888.
- Scagliusi, F. B., Polacow, V. O., Artioli, G. G., Benatti, F. B., & Lancha, A. H., Jr. (2003). Selective underreporting of energy intake in women: magnitude, determinants, and effect of training. *Journal of the American Dietetic Association*, 103(10), 1306-1313.
- Schlienger, J. L., & Pradignac, A. (1993). Relationships between weight and menopause. *Revue Francaise de Gynecologie et d'Obstetrique*, 88(5), 291-295.
- Schoeller, D. A. (1990). How accurate is self-reported dietary energy intake? *Nutrition Reviews*, 48(10), 373-379.
- Smith, B. K., York, D. A., & Bray, G. A. (1999). Activation of hypothalamic serotonin receptors reduced intake of dietary fat and protein but not carbohydrate. *Am J Physiol*, 277(3 Pt 2), R802-811.
- Spring, B., Wurtman, J., Gleason, R., Wurtman, R., & Kessler, K. (1991). Weight gain and withdrawal symptoms after smoking cessation: a preventive intervention using d-fenfluramine. *Health Psychology*, 10(3), 216-223.
- Stamford, B. A., Matter, S., Fell, R. D., & Papanek, P. (1986). Effects of smoking cessation on weight gain, metabolic rate, caloric consumption, and blood lipids. *The American Journal of Clinical Nutrition*, 43(4), 486-494.
- Vander Weg, M. W., Klesges, R. C., Eck Clemens, L. H., Meyers, A. W., & Pascale, R. W. (2001). The relationships between ethnicity, gender, and short-term changes in energy balance following smoking cessation. *International Journal of Behavioral Medicine*, 8(2), 163-177.

- Willett, W., Stampfer, M. J., Bain, C., Lipnick, R., Speizer, F. E., Rosner, B., et al. (1983). Cigarette smoking, relative weight, and menopause. *American Journal of Epidemiology*, *117*(6), 651-658.
- Williamson, D. F., Madans, J., Anda, R. F., Kleinman, J. C., Giovino, G. A., & Byers, T. (1991). Smoking cessation and severity of weight gain in a national cohort. *The New England Journal of Medicine*, *324*(11), 739-745.
- Wing, R. R., Matthews, K. A., Kuller, L. H., Meilahn, E. N., & Plantinga, P. L. (1991). Weight gain at the time of menopause. *Archives of Internal Medicine*, *151*(1), 97-102.
- Yang, Z. J., Blaha, V., Meguid, M. M., Oler, A., & Miyata, G. (1999). Infusion of nicotine into the LHA enhances dopamine and 5-HT release and suppresses food intake. *Pharmacol Biochem Behav*, *64*(1), 155-159.
- Zhang, L., Meguid, M. M., Miyata, G., Varma, M., & Fetissov, S. O. (2001). Role of hypothalamic monoamines in nicotine-induced anorexia in menopausal rats. *Surgery*, *130*(2), 133-142.

Vita

Amanda Manning graduated with a Bachelor of Arts from DePauw University in May of 2004. She then studied for a year in Spain, after which she began her graduate career at Louisiana State University. She studied biological psychology and worked in a lab at Pennington Biomedical Research Center. Amanda finished her master's degree in psychology in December of 2007.