

CONTROL OF FOOD INTAKE AND BODY WEIGHT FOLLOWING
SMOKING CESSATION IN PREMENOPAUSAL WOMEN

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ABSTRACT

Women experience more weight gain than men postcessation and are more aware of nicotine's weight suppressing effects than men. Postcessation weight gain in women can be largely accounted for by significant increases in high fat foods from pre- to postcessation. Overeating found in the luteal phase, further compounds the increased caloric intake found postcessation. Few studies have evaluated the long-term effects of smoking cessation on macronutrient content and weight gain; and most have relied on self-report data. This study used the Macronutrient Self-Selection Paradigm (MSSP) and Food Preference Questionnaire (FPQ) to assess food intake in 17 women in the luteal phase from baseline to 2-4 weeks postcessation (17 B2/PC1 subjects) and a subset of 10 women in the luteal phase from baseline to 2-4 weeks to 24 weeks (10 B2/PC1/PC2 subjects) smoking cessation. The 17 B2/PC1 subjects consumed significantly more total kilocalories intake, fat kilocalories intake, kilocalories intake of high fat foods, kilocalories intake of high sugar foods and kilocalories intake of High Fat/ High Sugar foods from baseline to Postcessation 1. The 10 B2/PC1/PC2 subjects yielded marginally nonsignificant results for the variables of total fat kilocalories intake (as compared to other macronutrients/ carbohydrates), total fat kilocalories intake across visits, and fat X carbohydrate across visits. The original sample size consisted of 37 women, however nearly half of the original sample experienced relapse (defined as one or more puffs of a cigarette during the time of the MSSP). These results suggest that an increase in foods high in fat and high in sugar 2-4 weeks postcessation are predominantly responsible for postcessation weight gain. Therefore, smoking cessation programs that are trying to help women maintain their weight should target nutritional advice especially to foods high in fat and sugar and recommend low fat alternatives to minimize weight gain postcessation.

INTRODUCTION

Cigarette use is a causative factor in 30 percent of all cancer deaths and in 90 percent of all lung cancer deaths in the United States (Subar, Harlan, & Mattson, 1990; U.S. Department of Health and Human Services, 2001). Women who smoke are significantly and appreciably at greater risk for the development of lung cancer than men who smoke comparable amounts. Since 1987 lung cancer has been the leading cause of cancer death among women (Harris, Zang, Anderson, & Wynder, 1993; U.S. Department of Health and Human Services, 2001); and further, since 1989 approximately three million U.S. women have died from a smoking-related disease (U.S. Department of Health and Human Services, 2001). Nevertheless, 22% percent of women in the United States are still smoking (U.S. Department of Health and Human Services, 2001); and women have historically been less successful in smoking cessation attempts than have men (CDC, 1991). Therefore it is of particular importance to identify the variables that promote smoking in females and, further, to identify variables that act as barriers to smoking cessation in order to create more effective intervention programs.

Females are more likely to diet and engage in both appropriate and inappropriate dieting strategies as compared to males (Klesges & Klesges, 1988). Smoking as a means to control appetite and lower body weight is one way in which women, more so than men, attempt to minimize body weight. Furthermore, women are more concerned about gaining weight postcessation than men and are less confident in their ability to control postcessation weight gain (Jeffery, Henrikus, Lando, Murray & Lui, 2000). Pomerleau & Kurth (1996) found that women who were contemplating smoking cessation would only allow themselves to gain 5.0 +/- 5.8 pounds (2.3 +/- 2.6 kg) compared to men who would allow themselves to gain 10.7 +/- 7.6 pounds (4.9 +/- 3.5 kg). In fact, 21% of females endorsed that they “smoked cigarettes and/ or drank caffeinated beverages” to lose weight (Klesges, Mizes, & Klesges, 1987). The attempt to maintain a lower body weight facilitates chronic female smoking. A survey of nearly 7,000 female students found that the factor that best discriminated experimental versus regular smoking was the use of smoking as a weight control strategy (Robinson, Klesges, Zbikowski, & Glaser, 1997). The use of smoking as a means to control body weight starts at an early age in females, and young female smokers are especially aware of nicotine’s weight suppressing effects (Hall, Tunstall, Vila & Duffy, 1992; Strauss & Mir, 2001). High school girls who report having tried to lose weight in the past year, having two or more eating disorder symptoms, or constantly thinking about weight and shape, are more than twice as likely to initiate smoking as those who do not report these dieting concerns (French, Perry, Leon, & Fulkerson, 1994).

Smoking cessation among women is typically associated with a weight gain of about 6 to 12 pounds (2.72-5.44 kg) in the year after they quit smoking (U.S. Department of Health and Human Services, 2001). The use of smoking as a weight control strategy by women is not unfounded; women do get more weight-control benefits from smoking and suffer more postcessation weight gain compared to men (Klesges & Klesges, 1988; Debon & Klesges, 1995). Peterson & Helton (2000) found that at eight weeks postcessation women gained as much as 9.8 pounds (4.45 kg) as compared to 5.5 pounds (2.50 kg) gained by men. The manifestation of this sex specific relationship between smoking cessation and weight gain is apparent. Indeed, women who quit smoking do have the largest increases in mean BMI each year (Owen-Smith & Hannaford, 1999; Williamson et al., 1991). Nicotine’s sex-related effects is consistent in the animal literature, in that nicotine produces greater weight-control effects in females than in male rats (Grunberg, Winders, & Popp, 1987); and female rats tend to gain more weight with

nicotine cessation as compared to male rats (Grunberg, 1986). Grunberg, Winders, & Popp (1987) found that in female rats the weight gain was substantial and rapid, but on the other hand the weight gain of the male rats remained below rats with no nicotine up to four months after nicotine cessation.

Increased caloric intake, especially within the first month after cessation, has been implicated as the primary contributing factor in long-term postcessation weight gain (Perkins, 1992). Humans experience as much as a 200-400 kcal increase in food consumption per day postcessation, thus favoring weight gain (Perkins, 1992; Klesges, Eck, Isbell, Fulliton, & Hanson, 1990). Likewise, Grunberg (1982) and Grunberg, Bowen, Maycock and Nespor (1985) found that rats treated with nicotine consume less food than saline treated rats, and that after cessation of nicotine administration the rats increased food consumption. Of particular interest is the fact that serotonin has been found to suppress carbohydrate and fat intake; and nicotine administration in rats and humans increases serotonin in the brain, thus amplifying the suppression of carbohydrate and fat intake (Blundell, Lawton, & Halford, 1995; Lebowitz, Weiss, & Shor-Posner, 1988). Increased carbohydrate and sugar intake following smoking cessation may be an attempt to readjust brain serotonin levels and to improve postcessation reduction in mood (Fernstrom & Wurtman, 1971). This was tested by Spring et al. (1991) and it was found that by increasing serotonin levels in abstaining smokers mood reduction and weight gain was prevented.

One of the primary nicotine withdrawal symptoms differentiating men and women is increased appetite in women (Pirie et al., 1992). Further, for women (but not men) the initial postcessation increased caloric intake predicts future weight gain, thus suggesting that this initial postcessation period is more critical to women's long-term food intake and weight control as compared to men's (Hall, McGee, Tunstall, Duffy, & Benowitz, 1989). Hall et al. (1989) found that postcessation increases in caloric intake are indeed chronic for women, but not men; in that men after 12 and 26 weeks postcessation showed a marked decrease in food intake (as compared to baseline), but women at the same intervals showed increases in food intake. Dietary restraint measures the intent to control weight through restrictive eating, this is measured in the Eating Inventory. Moreover, it has been suggested that the characteristic of dietary restraint may identify a particular group of women smokers whose eating is most influenced by smoking (Ogden & Fox, 1994; Perkins, Mitchell, Epstein & 1995). Female smokers high in restraint, more so than those low in restraint, are more likely to start and continue to smoke to control body weight and are less interested in quitting (Ogden & Fox, 1994; Perkins, Mitchell & Epstein, 1995; Perkins, Epstein, Fonte, Mitchell & Grobe, 1995).

Studies have found that from pre- to postcessation a significant increase in the consumption of foods high in fat is evident and that a parallel increase in consumption of all types of foods is not found (Eck et al., 1997; Allen, Hatsukami, Christianson & Brown, 2000). There is general agreement that postcessation weight gain is largely due to an increase in caloric intake. However postcessation changes in specific macronutrient intake are not clear. Several studies have indicated that significant increases in high sugar foods and other high carbohydrate foods with high fat content, account for the major difference in food intake and weight gain postcessation (Hall, McGee, Tunstall, Duffy, & Benowitz, 1989; Eck, Klesges, Meyers, Slawson, & Winders, 1997). It is noteworthy, that foods high in both fat and sugar are most consistently associated with hyperphagia and weight gain in females (Hall et al., 1989). Likewise, in animal studies, nicotine administration yields less fat consumption and lower body weight as compared to controls, and cessation from nicotine results in an increase in caloric intake, due to a

specific increase in fat consumption (Grunberg, 1982; Grunberg, Bowen & Morse, 1985). In fact, when only nonsweet low fat foods are available, there are no effects of nicotine cessation on food consumption or body weight in rats (Grunberg, et al., 1984).

Increases in food intake in the luteal phase of the menstrual cycle compound the fear of postcessation weight gain (Allen et al., 2000). The menstrual cycle can be divided into four phases: menses, late follicular phase, periovulatory phase and the luteal phase. Menses can be defined as beginning at the time of the menstrual blood flow and ending at the time of cessation of blood flow. The late follicular phase can be defined as the time period from the offset of menses until the periovulatory phase. The luteal phase can be defined as the time period following the periovulatory phase until the next menstrual bleeding. Estrogen concentrations are low during menses, higher during the late follicular phase, peak at ovulation and are relatively high (but lower than in the follicular phase) during the luteal phase. Progesterone concentrations are low during menses, the late follicular and periovulatory phases, and high during the luteal phase only. Studies have shown that an influx of estrogen suppresses food intake and that high progesterone levels in the luteal phase may block this suppression (Hall et al., 1989).

Females of a large number of mammalian species (including humans) show variation in food intake and body weight across phases of the estrous cycle/menstrual cycle, tending to eat less and lose weight when endogenous levels of estrogen are high and progesterone is low and to eat more and gain weight when progesterone levels are elevated and estrogen levels are lower (Brobeck, Wheatland, & Strominger, 1947; Gilbert & Gillman, 1956; Ota & Yokoyama, 1967; Czaja, & Goy, 1975; Morin & Fleming, 1978; Kemnitz, 1984; Allen et al., 2000). In normal weight women increases in food intake by as much as 500 kcal have been found during the luteal phase, but most studies report a 10-14% increase as compared to the rest of the menstrual cycle (Dalvit, 1981; Buffenstein et al., 1995; Li, Tsang & Lui, 1999). This increase in food intake in the luteal phase further compounds the increase of 200-400 kcals found postcessation. (Buffenstein et al., 1995; Dalvit, 1981). There are reports suggesting that hyperphagia occurs most in the late luteal phase (Buffenstein et al., 1995). Furthermore, postcessation women in the luteal phase of the menstrual cycle do gain more weight as compared to those in the follicular phase; thus suggesting that the luteal phase is indeed a complicating factor to be considered when women quit smoking (Pomerleau, Pomerleau, Namemek & Mehringer, 2000).

The luteal phase is associated with dramatic increases in fat consumption in women, which further compounds the increase in caloric intake postcessation (Bowen & Grunberg, 1990; Tarasuk & Beaton, 1991; Li et al., 1999), with as much as a 21% increase in fat intake during the luteal phase (Li et al., 1999). Animal studies show consistent results, in that when levels of progesterone are elevated and estrogen levels are lower, not only are more calories consumed, but significantly more calories as fat are consumed (Geiselman, Martin, Vanderweeke & Novin, 1981).

Few studies have looked at weight gain or changes in macronutrient intake further than one month after cessation. Further, most studies have relied on self-reports of food intake, without a valid laboratory assessment. No studies in the smoking cessation literature have tested macronutrient selection in a validated and reliable paradigm. The Macronutrient Self-Selection Paradigm (MSSP) and Food Preference Questionnaire (FPQ) are validated assessment instruments with strong within-subjects test-retest reliability that vary fat content of foods significantly and systematically (Geiselman, et al., 1998). Sugar, complex carbohydrate and protein are varied in a battery of test foods that are representative of those in which fat is consumed in the average diet (Geiselman, et al., 1998). The MSSP uses foods that can practicably be used and are available on the

supermarket shelves and are easy to prepare (Geiselman et al., 1998). Further the FPQ allows for the testing of a larger battery of foods in which time of preparation and other factors will not allow for inclusion in the MSSP. The MSSP was used to assess changes in fat and other specific macronutrient and total caloric intake at baseline (when subjects were still smoking) and at 1-2 weeks postcessation and at 24 weeks postcessation. The FPQ was used to assess changes in fat preference at baseline, 1-2 weeks postcessation and 24 weeks postcessation. Subjects were tested during the late luteal phase of their menstrual cycle, a phase prone to female hyperphagia. Also, female subjects taking oral contraceptives were tested using the time in the pill pack that most closely pharmacologically mimicked the late luteal phase. This test allowed for long term evaluation of macronutrient intake changes in women and determination of how these changes influence weight gain postcessation.

Specific Aims

Specific Aim 1: To assess fat and other specific macronutrient intake, (fat, sugar, complex carbohydrates and protein) total caloric intake, and fat preference in weight concerned women in the late luteal phase while still smoking, at 1-2 weeks postcessation, and at 24 weeks postcessation.

It is hypothesized that intake of high fat foods, especially those high in sugar, and fat preference will significantly increase postcessation.

Specific Aim 2: To determine the extent to which fat and other macronutrient intake and fat preference can predict weight gain postcessation.

It is hypothesized that an increase in foods high in fat, especially those high in sugar, and fat preference will be associated with an increase in postcessation weight gain.

Specific Aim 3: To determine whether dietary restraint, disinhibition, BMI, waist/hip ratio and weight concern predict changes in fat and other macronutrient intake and the increase in body weight postcessation.

MATERIALS AND METHODS

Subjects

Subject Criterion

Weight-concerned female smokers between the ages of 18-46 were recruited. Weight concern was defined as use of smoking as a weight control strategy and fear of weight gain postcessation. Women recruited had a regular menstrual cycle (defined as being between 25-35 days). The subjects could either be using no oral contraceptives or use monophasic, biphasic, or triphasic oral contraceptives. Smoking was defined as 1) self report of > 10 cigarettes per day for 1 year or more, 2) expired CO > 10 ppm.

Subject Demographics

Subjects were 37 Caucasian weight-concerned female smokers, recruited through Informational Sessions hosted at the Pennington Biomedical Research Center, paper and radio advertisement, and health fairs. Thirteen subjects were excluded from the analysis because they experienced smoking relapse and/or refused treatment. Relapse was defined as one or more puffs of a cigarette with no attempt at smoking cessation during the time period of the study. Two subjects were excluded from the analysis because they both were menstruating the day of the MSSP, therefore no longer being in the late luteal phase. Three subjects were excluded from the analysis because they did not fast the morning of the MSSP. Two subjects were excluded from the analysis because they had only completed Baseline 2. Therefore a total of 20 subjects were eventually excluded from the study.

Seventeen subjects were used for the analysis of food intake for the Baseline 2 and Postcessation 1 within-subject analysis (17 B2/PC1 subjects). Twelve of the 17 B2/PC1 subjects had physiologically-controlled menstrual cycles and 5 of the subjects had pharmacologically-controlled menstrual cycles. Of these 17, 10 subjects provided data for all 3 assessments that were used in a within-subjects analysis of food intake from Baseline 2 to Postcessation 1 to Postcessation 2 (10 B2/PC1/PC2 subjects). This group consisted of 10 subjects that completed all three assessments. Seven of the 10 B2/PC1/PC2 subjects had physiologically-controlled menstrual cycles and three of the subjects had pharmacologically-controlled menstrual cycles (all oral contraceptives were the monophasic type).

Thesis Study Design

The MSSP (Macronutrient Self-Selection Paradigm) and FPQ (Food Preference Questionnaire) were completed at baseline (approximately one month after starting the program, while the subject is still smoking), Postcessation 1 (approximately 7-14 days after initial smoking cessation) and Postcessation 2 (24 weeks after smoking cessation). Each of the three assessments was scheduled according to the late luteal phase of the menstrual cycle or for pharmacologically controlled subjects, the phase of the oral contraceptives that most closely mimics the luteal phase in hormone dosage.

Parent Study

STOP (Stop Treatment/ Obesity Prevention) is the parent study that is designed to help women quit smoking, while maintaining their weight. After enrollment into the program all subjects underwent baseline assessments, which included Screening 1, Baseline 1, and Baseline 2. At Screening 1 the subject completed a series of questionnaires including: weight assessment, dietary assessment, menstrual cycle assessment, smoking assessment, and psychosocial assessment. Baseline 1 included: dietary assessment, smoking assessment, psychosocial assessment and physical activity assessment. Baseline 2 included the MSSP/test lunch, dietary assessment and menstrual cycle assessment. After completion of the baseline assessments the subject waited until enough subjects had also completed the baseline (usually 5-10 subjects) and then participated in a two-week intensive smoking cessation program. During this same two weeks the subject completed Postcessation 1, which is the second MSSP. Subjects in the parent study were enrolled into a 36 week follow-up/intervention program, where they were randomly assigned to the group or tailored condition. The group condition participated in group smoking cessation sessions, which also included food pyramid guidelines and general exercise information. On the other hand, the tailored condition received individualized nutrition information and specific exercise guidelines (based on their preferences and answers to preliminary questionnaires). The 17 subjects for this analysis were members of the group condition. Twenty four weeks after quitting smoking the subjects completed Postcessation 2, which was the third and final MSSP (see Table 1).

Menstrual Cycle Assessment

The menstrual cycle can be divided into four distinct phases: menses, late follicular phase, periovulatory phase and the luteal phase. Menses can be defined as beginning at the time of the menstrual blood flow and ending at the time of cessation of blood flow. The late follicular phase can be defined as the time period from the offset of menses until the periovulatory phase. And the luteal phase can be defined as the time period following the periovulatory phase until the next menstrual bleeding.

At the initial screening all subjects, who have a physiologically controlled menstrual cycle, were given the choice of using a basal thermometer or ovulation kits to monitor ovulation. If the subject elected to use the basal thermometer, the subject was provided with a basal thermometer and a temperature log. The subject was required to measure her temperature each morning as soon as she waked up, before getting out of bed and record it on the temperature log. If the subject elected to use ovulation kits, a time to use the kits was estimated from previous duration of each individual's menstrual cycle. The subject was advised to use the ovulation kits approximately three days prior to the estimated time of ovulation to ensure accurate results.

All subjects were required to telephone the STOP study to report the onset and offset of menses; and also to telephone in when ovulation was detected. In women not using oral contraceptives the late luteal phase was estimated by using the time of ovulation

Table 1. Data Collection Schedule

Measures	Baseline	First two weeks of smoking cessation	24 weeks postcessation
Dietary Assessment			
1. MSSP	X	X	X
2. FPQ	X	X	X
3. 3 day food diaries	X	X	X
4. Three Factor Eating Questionnaire	X		
Weight Assessment			
1. Body Weight	X	X	X
2. Body Height	X	X	X
3. BMI	X	X	X
4. Waist/Hip Ratio	X	X	X
5. Weight Concern Measure	X	X	X
Menstrual Cycle Assessment	X	X	X
Smoking Assessment	X	X	X
Psychosocial Assessment	X	X	X

detection (either by thermometer or ovulation kits) to determine cycle length. For example, a woman who ovulated on day 14 of her cycle would have a cycle that is 28 days in length and therefore the late luteal was be from day 21-28. For women on oral contraceptives the time to schedule the MSSP depended on the type of oral contraceptive used. In the case of a monophasic pill (in which estrodial and progesterone are held constant for 21 days) the MSSP was scheduled between day 8 and day 20. In the case of diphasic and triphasic pills (in which the estradiol and progesterone are varied) the MSSP was scheduled at the week where progesterone was at its highest in relation to estrodial.

Subjects were required to monitor their menstrual cycle as detailed above, beginning at the screening visit until after the second Macronutrient Self-Selection Paradigm (MSSP) (immediately postcessation, called “Postcessation 1”). After Postcessation 1 until week 20 women were only asked to record menses onset and offset. Beginning at week 20 the women were required to start taking their temperature or using ovulation kits to detect ovulation again, until they had completed the 24 week MSSP. Detection of ovulation was defined as a spike in temperature (if using the basal thermometer) or a positive reading (if using the ovulation kits). Day 1 of a physiologically controlled cycle was considered the first day of menstrual bleeding and day 1 of a pharmacologically controlled cycle was considered the first day of a new pack of pills.

Macronutrient Self-Selection Paradigm and Food Preference Questionnaire

Dr. Paula Geiselman has developed two instruments—the Macronutrient Self-Selection Paradigm (MSSP) and Food Preference Questionnaire (FPQ) that vary macronutrient content significantly and systematically in a battery of foods that are commonly consumed in the American diet (Geiselman et al., 1998). Specifically, the MSSP and FPQ measure total caloric intake and intake of fat, sugar, high complex carbohydrates and proteins separately. The MSSP has strong test retest reliability for overall fat and other macronutrient intake and total caloric intake (Geiselman et al., 1998). It has further proven to be valid for measuring long term fat intake (Geiselman et al., 1998). The MSSP and FPQ work together as tools to assess food intake and overall fat preference; the MSSP, as the laboratory exercise directly assessing food intake and the FPQ, as a paper and pencil exercise examining overall fat preference.

The MSSP is a 2 (Fat Factor: High Fat and Low Fat) x 3 (Carbohydrate factor: High Simple Sugar, High Complex Carbohydrate and Low Carbohydrate/High Protein) x 3 (specific foods within each cell) design. The six cells in the design are High Fat/ High Sugar (HF/HS), High Fat/ High Complex Carbohydrate (HF/HCCCHO), High Fat/ Low Carbohydrate/ High Protein (HF/LCHO/HP), Low Fat/ High Sugar (LF/HS), Low Fat/High Complex Carbohydrate (LF/HCCCHO), and Low Fat/ Low Carbohydrate/High Protein (LF/LCHO/HP) (see Table 2).

Table 2. Macronutrient Self-Selection Paradigm

	High Simple Sugar	High Complex CHO	Low CHO/High Protein
High Fat	High Fat/ High Simple Sugar Three Foods	High fat/ High Complex CHO Three Foods	High Fat/ Low CHO/ High Protein Three Foods
Low Fat	Low Fat/ High Simple Sugar Three Foods	Low Fat/High Complex CHO Three Foods	Low Fat/ Low CHO/High Protein Three Foods

Ninety-two foods are available to be used in the MSSP. However, at the time of the MSSP 18 foods (3 from each of the 6 cells, e.g. 3 high fat/ high sugar, etc.) are chosen based on the subject's responses to three questions: How much do you like each of the following foods? How often do you eat each of the following foods? If cost, availability, and convenience were not factors, how often would you like to eat each of the following foods? Each of the three scales uses a Likert scale to rate each food. The scales are given to the subject at the initial screening visit, obtained from the subject at Baseline 1 and are analyzed after the Baseline 1 visit. Foods are chosen in such a way that there is no competition among foods within each cell, but instead foods are chosen to compete between cells. For example a high fat and low fat meat are both presented at the time of assessment so that the subject must chose between the low and high fat food. The foods that make up the MSSP were chosen in such a way that the majority of the fat in the

foods can be detected visually instead of having to be tasted (eg. High Fat/ High Sugar cell are chocolate bars and Low Fat/ High Sugar cell are grapes).

The foods that are included in the design of the MSSP are divided as follows: any food considered “high fat” is > 45% fat (>30% sugar is considered “high sugar”, > 30% complex carbohydrate is considered “high complex carbohydrate”, and >13% protein is considered “high protein”, though most foods were between 25%-35% protein) and any food considered “low fat” is < 20% fat.

The 18 foods that were selected for each subject were prepared according to a protocol that details the presentation, type of apparatus the food is to be presented on (e.g. bowl versus dinner plate) and amount. For example, a food such as cheese was presented in slices and also cubed, as to ensure that presentation did not affect choice. At the time of food preparation each food was weighed to determine its “pre weight.” The foods were randomly (using a random numbers table) placed on a table in a testing room where the subject ate alone, with no distractions. The subject’s personal belongings and watch were retained by the experimenter. After the subject completed the MSSP the foods were weighed again to determine their “post weight.”

The Food Preference Questionnaire was developed as a 2 (Fat: High Fat and Low Fat) x 3 (Carbohydrate Factor: High Simple Sugar, High Complex Carbohydrate, and Low Carbohydrate/High Protein) design (see Table 3).

Table 3. Food Preference Questionnaire

	High Simple Sugar	High Complex CHO	Low CHO/ High Protein
High Fat	High Fat/High Simple Sugar Twelve Foods	High Fat./ High Complex CHO Twelve Foods	High Fat/ Low CHO/High Protein Twelve Foods
Low Fat	Low Fat/ High Simple Sugar Twelve Foods	Low Fat/ High Complex CHO Twelve Foods	Low Fat/ Low CHO/High Protein Twelve Foods

The FPQ contains an additional 72 foods (12 for each of the 6 cells) presented in random order that could not be included in the MSSP due to varying degrees of preparation difficulty. This too was prepared in a Likert scale using the same numeric labels as the MSSP.

Procedure for MSSP and FPQ

In preparation for the MSSP and FPQ the subjects were instructed to 1) fast from 10:00 pm the night before until the lunch (having only water to drink), 2) abstain from alcohol for 24 hours and 3) abstain from exercise the morning of the MSSP

At the time of arrival (at either 11:00 a.m. of 12:00 p.m.), the subject was escorted to the testing area. At this time the subject was given a pre-test questionnaire (which assesses any special circumstances, e.g. having a cold). Next the subject was given VAS #1 to complete. This was a series of visual analog scales that assessed the hunger of the subject at that moment in time. Next the subject was given FPQ #1 to complete and upon completion VAS # 2 was done. At this time the subject completed the MSSP (the actual test lunch) in a test room alone. Upon completion of the MSSP, which usually took 15-20 minutes the subject completed VAS # 3. Next the subject completed FPQ # 2 and

then VAS # 4. Finally, the subject was interviewed (using the post-MSSP interview) by the experimenter as to the quality of their experience.

Data Collection

Description of Assessments

Dietary Assessment: Macronutrient Self-Selection Paradigm, Food Preference Questionnaire, 3-day food diaries from the late luteal phase; The Dietary Restraint Scale (which measures intent to control weight through restrictive eating) and Disinhibition Scale (loss of control of food intake) on the Three-Factor Eating Questionnaire.

Weight Assessment: Body weight, Body height, BMI, waist/hip ratio; The Weight Concern Measure.

Data Assessment Schedule

The Weight Concern Measure was administered at screening and 24 weeks postcessation. Body weight, height, BMI, and waist/hip ratio were administered at screening, immediate postcessation (first 1-2 weeks numerous times) and 24 weeks postcessation. The MSSP and FPQ were conducted at baseline (approximately one month after starting the program, while the subject was still smoking), Postcessation 1 (approximately 7-14 days after initial smoking cessation) and Postcessation 2 (24 weeks after smoking cessation). The MSSP and FPQ were scheduled according to the late luteal phase of the menstrual cycle (see Table 7).

Outcome Variables

Primary Outcome Variable: The change in fat intake from precessation to 24 weeks postcessation.

Secondary Outcome Variable: The change in body weight from precessation to 24 weeks postcessation.

Statistical Power Analysis

A preliminary power analysis was completed prior to this study on a group of non smoking women. In that analysis a sample of 35 women was sufficient to detect any within-subjects changes of 10g fat intake with greater than 89 percent power. However, in the present study a sample of 17 subjects was sufficient to detect any within-subjects changes of 10g of fat intake with greater than 70 percent power. In the preliminary sample, the significance was less robust and therefore a more conservative approximation of sample size was necessary. The present study represents a different population in which the effect was more robust and an N of 17 is sufficient to satisfy power requirements.

Table 4. Data Assessment Schedule

Measures	Baseline	First two weeks of smoking cessation	24 weeks
Dietary Assessment			
1. MSSP	X	X	X
2. FPQ	X	X	X
3. 3 day food diaries	X	X	X
4. Three Factor Eating Questionnaire	X		
Weight Assessment			
1. Body Weight	X	X	X
2. Body Height	X	X	X
3. BMI	X	X	X
4. Waist/Hip Ratio	X	X	X
5. Weight Concern Measure	X		X
Menstrual Cycle Assessment	X	X	X
Smoking Assessment	X	X	X
Psychosocial Assessment	X	X	X

Statistical Analysis

Within-subjects analyses of variance (ANOVAs) were conducted to evaluate within-subjects variation in fat and macronutrient intake from Baseline 2 to Postcessation 1 (for the 17 B2/PC1 subjects) and from Baseline 2 to Postcessation1 to Postcessation 2 (for the 10 B2/PC1/PC2 subjects). In addition, multiple regression analyses were conducted to identify which factors were predictive of changes in fat and other macronutrient intake and body weight following smoking cessation.

RESULTS

Subject Characteristics

Subject characteristics are shown in Table 5 and Table 6. The mean age, body weight, BMI, waist to hip ratio, years as a smoker, cigarettes per day during the last year, disinhibition, and dietary restraint were assessed at the Screening visit (pre-smoking cessation).

Table 5. Subject Characteristics for 17 B2/PC1 Subjects

Variable	Mean	Range
Age (years)	37	28-46
Body weight (in pounds)	140	115-186
BMI	23	19-28
Waist to hip ratio	.77	.66-.89
Years as a smoker	19	10-30
Cigarettes per day during the last year	11-20	11-20
Dietary Restraint	8	1-18
Disinhibition	7	2-12

Table 6. Subject Characteristics for 10 B2/PC1/PC2 Subjects

Variable	Mean	Minimum
Age (years)	38	28-46
Body weight (in pounds and kilograms)	133	127-143
BMI	22	19-25
Waist to hip ratio	.76	.66-.84
Years as a smoker	19	10-30
Cigarettes per day during the last year	11-20	11-20
Dietary Restraint	8	1-18
Disinhibition	6	2-12

Total Kilocalories (Kcals) Intake

Within-subjects, analyses of variance (ANOVA) were conducted on the dependant variable of total kilocalories intake. The 17 B2/PC1 subjects consumed significantly more kilocalories at Postcessation 1 than at Baseline 2 ($F(1,16)=7.45$, $p=.02$) (see Table 7 and 8 and Figure 1). However, statistical analyses for the 10 B2/PC1/PC2 subjects yielded statistically nonsignificant results for total kilocalories intake across visits ($F(2,18)=1.64$, $p=.22$) (see Table 7 and 8 and Figure 1).

Kilocalories Intake from Specific Macronutrient Sources

17 B2/PC1 Subjects

A 2 (Visit: Baseline 2 and Postcessation 1) X 4 (Macronutrient: Fat, Sugar, Complex Carbohydrate, and Protein) within-subjects ANOVA was conducted for the 17 B2/PC1 subjects.

A significant main effect for Visit ($F(1,16) = 7.45, p = .02$) was obtained, showing that subjects consumed significantly more total kilocalories across macronutrient sources at Postcessation 1 than at Baseline 2 (see Table 7 and 8; Figure 1). A significant main effect for Macronutrient ($F(3,48) = 95.62, p = .00$) was also found (See Table 7 and 8; Figure 2). Therefore post hoc, Bonferroni t-tests were conducted and statistically significant results are reported in Table 8, showing that subjects ingested significantly more kilocalories from fat sources than from sugar, complex carbohydrate and protein sources; and significantly more kilocalories from complex carbohydrate sources than from protein sources (see Table 8; Figure 2). The Visit X Macronutrient interaction was statistically significant ($F(3,48) = 6.38, p = .01$) (see Table 7 and 8; Figure 2). Post hoc, Bonferroni t-tests revealed that subjects consumed significantly more kilocalories from fat sources at Postcessation 1 than at Baseline 2 ($t(16) = -2.73, p = .01$) (see Table 8), and significantly more kilocalories from sugar sources at Postcessation 1 than at Baseline 2 ($t(16) = -2.96, p = .01$) (see Table 8; Figure 2).

10 B2/PC1/PC2 Subjects

A 3 (Visit: Baseline 2, Postcessation 1, and Postcessation 2) X 4 (Macronutrient: Fat, Sugar, Complex Carbohydrate, and Protein) within-subjects, ANOVA was conducted for the 10 B2/PC1/PC2 subjects. A significant main effect for Macronutrient ($F(2,19) = 46.43, p = .00$) was obtained (see Table 7 and 8), therefore post hoc, Bonferroni t-tests were conducted and statistically significant differences are listed in Table 8, showing that subjects ingested significantly more kilocalories from fat sources than from sugar, complex carbohydrate and protein; and significantly more kilocalories from complex carbohydrate sources than from protein sources (see Table 8; Figure 2). The Visit main effect and the Visit X Macronutrient interaction yielded statistically nonsignificant results ($F(2, 18) = 1.64, p = .22$; $F(3,28) = 2.37, p = .09$; see Table 7 and 8).

Food Selection (Kcals Intake) From Specific Macronutrient Sources

17 B2/PC1 Subjects

A 2 (Fat: High Fat & Low Fat) X 3 (Carbohydrate: High Sugar, High Complex Carbohydrate, and Low Carbohydrate/ High Protein) X 2 (Visit: Baseline 2 and Postcessation 1) within-subjects ANOVA was conducted for the 17 B2/PC1 subjects. A main effect for Fat ($F(1,16) = 67.32, p = .00$) was obtained (see Table 7 and 8; Figure 3), indicating that significantly more kilocalories of high fat foods were consumed than kilocalories of low fat foods across visits.

The Carbohydrate main effect yielded statistically nonsignificant results ($F(2,32) = 1.24, p = .30$; see Table 7 and 8). A main effect of Visit ($F(1,16) = 8.07, p = .01$) was

found, reaffirming that subjects consumed significantly more kilocalories at the Postcessation 1 visit as compared to the Baseline 2 visit (see Table 7 and 8; Figure 1).

The Fat X Carbohydrate interaction was statistically, nonsignificant ($F(2,32) = .96$, $p = .40$; see Table 7 and 8; Figures 5-9).

The Fat X Visit interaction was significant ($F(1,16) = 8.29$, $p = .01$), therefore Bonferroni t-tests were conducted (see Table 7 and 8). Subjects consumed significantly more kilocalories of high fat foods at Postcessation 1 than at Baseline 2 ($t(16) = -2.91$, $p = .01$) (see Table 8; Figure 3).

The Carbohydrate X Visit interaction was statistically significant ($F(2,32) = 5.81$, $p = .02$; see Table 7 and 8). Post hoc, Bonferroni t-tests were conducted and subjects consumed significantly more kilocalories of high sugar foods at Postcessation 1 than at Baseline 2 ($t(16) = -3.67$, $p = .00$; see Table 8 and 9).

The Fat X Carbohydrate X Visit interaction was statistically significant ($F(2,32) = 4.93$, $p = .01$; see Table 7 and 8). Post hoc, Bonferroni t-tests were conducted and subjects consumed significantly more kilocalories of High Fat/High Sugar foods at Postcessation 1 than at Baseline 2 ($t(16) = -3.91$, $p = .00$; see Table 8 and 9; Figures 4-9).

10 B2/PC1/PC2 Subjects

A 2 (Fat: High Fat and Low Fat) X 3 (Carbohydrate: High Sugar, High Complex Carbohydrate, and Low Carbohydrate/ High Protein) X 3 (Visit: Baseline 2, Postcessation 1 and Postcessation 2) within-subjects ANOVA was conducted for the 10 B2/PC1/PC2 subjects. A main effect for Fat ($F(1,9) = 36.85$, $p = .00$) was found, indicating that subjects consumed more kilocalories of high fat foods than kilocalories of low fat foods across visits (see Table 7).

The main effect for Carbohydrate yielded statistically nonsignificant results ($F(2,18) = 1.37$, $p = .28$; see Table 7).

The main effect for Visit was statistically nonsignificant ($F(2,18) = 2.15$, $p = .15$) (see Table 7), indicating that the means of kilocalories intake at each visit were not significantly different.

The Fat X Carbohydrate interaction was statistically nonsignificant ($F(2,18) = .90$, $p = .43$; see Table 7 and 8).

The Fat X Visit interaction was statistically nonsignificant ($F(2,18) = 3.42$, $p = .06$) (see Table 7 and 8).

The Carbohydrate X Visit interaction was statistically nonsignificant ($F(2,15) = .90$, $p = .41$; see Table 7 and 8).

The Fat by Carbohydrate by Visit interaction was statistically nonsignificant ($F(4,36) = 2.26$, $p = .08$) (see Table 7 and 8; Figures 4-9).

Table 7. MSSP Intake

Dependant Variables	Group/ Subgroup	Baseline 2 (mean)	Postcessation 1 (mean)	Postcessation 2 (mean)
Total Kcals	B2/ PC1	824.9	1013.5	--
	B2 / PC1/ PC2	790.8	1018.0	941.3
Fat Kcals	B2 / PC1	391.1	496.2	--
	B2 / PC1/ PC2	376.0	500.0	420.5
Sugar Kcals	B2/ PC1	133.11	177.71	--
	B2 / PC1/ PC2	132.78	185.57	184.75
CCHO Kcals	B2 / PC1	171.24	193.99	--
	B2 / PC1/ PC2	159.00	190.44	197.45
Protein Kcals	B2/ PC1	129.45	145.69	--
	B2 / PC1/ PC2	123.00	142.00	138.61
High Fat/ High Sugar Foods	B2 to PC1	154.4	290.4	--
	B2 to PC1 to PC2	136.1	287.0	206.8
High Fat/ High CCHO Foods	B2 to PC1	234.0	228.6	--
	B2 to PC1 to PC2	214.6	201.8	243.8
High Fat/ Low Carbohydrate, High Protein Foods	B2 to PC1	167.1	205.6	--
	B2 to PC1 to PC2	141.4	217.2	173.4
Low Fat/ High Sugar Foods	B2 to PC1	87.2	85.9	--
	B2 to PC1 to PC2	88.7	93.1	137.5
Low Fat/ High CCHO Foods	B2 to PC1	38.0	55.9	--
	B2 to PC1 to PC2	38.8	63.7	47.9
Low Fat/ Low Carbohydrate, High Protein Foods	B2 to PC1	62.6	62.4	--
	B2 to PC1 to PC2	58.5	53.2	61.4

Table 8. ANOVA Results

ANOVA	Group	P value
Total Kcals Analyses	B2/PC1	.02
	B2/ PC1/ PC2	.22
2 (Visit) X 4 (Macroutrient) Analyses		
a. Visit Effect	B2/PC1	.02
b. Macronutrient Main Effect	B2/PC1	.00
c. Visit X Macronutrient	B2/PC1	.01
3 (Visit) X 4 (Macronutrient) Analyses		
a. Visit Main Effect	B2/ PC1/ PC2	.22
b. Macronutrient Main Effect	B2/ PC1/ PC2	.00
c.Visit X Macronutrient	B2/ PC1/ PC2	.09
2 (Fat)X 3 (Carbohydrate) X 2 (Visit) Analyses		
a. Fat Main Effect	B2/PC1	.00
b. Carbohydrate Main Effect	B2/PC1	.30
c. Visit Main Effect	B2/PC1	.01
d. Fat X Carbohydrate	B2/PC1	.40
e. Fat X Visit	B2/PC1	.01
f. Carbohydrate X Visit	B2/PC1	.02
g. Fat X Carbohydrate X Visit	B2/PC1	.01
2 (Fat)X 3 (Carbohydrate) X 3 (Visit) Analyses		
a. Fat Main Effect	B2/ PC1/ PC2	.00
b. Carbohydrate Main Effect	B2/ PC1/ PC2	.28
c. Visit Main Effect	B2/ PC1/ PC2	.15
d. Fat X Carbohydrate	B2/ PC1/ PC2	.43
e. Fat X Visit	B2/ PC1/ PC2	.06
f. Carbohydrate X Visit	B2/ PC1/ PC2	.41
g. Fat X Carbohydrate X Visit	B2/ PC1/ PC2	.08

Table 9. Post hoc, Bonferroni T- Test Results

	Group	P value
2 (Visit) X 4 (Macronutrient)		
Macronutrient		
a. Fat Kcals versus Sugar Kcals	B2/PC1	.00
b. Fat Kcals versus CCHO Kcals	B2/PC1	.00
c. Fat Kcals versus Protein Kcals	B2/PC1	.00
d. CCHO Kcals versus Protein Kcals	B2/PC1	.00
Visit X Macronutrient		
a. B2 Fat Kcals versus PC1 Fat Kcals	B2/PC1	.01
b. B2 Sugar Kcals versus PC1 Sugar Kcals	B2/PC1	.01
3 (Visit) X 4 (Macronutrient)		
Macronutrient		
a. Fat Kcals versus Sugar Kcals	B2/PC1/PC2	.00
b. Fat Kcals versus Protein Kcals	B2/PC1/PC2	.00
c. Fat Kcals versus CCHO Kcals	B2/PC1/PC2	.00
d. CCHO Kcals versus Protein Kcals	B2/PC1/PC2	.01
2 (Fat)X 3 (Carbohydrate) X 2 (Visit)		
Fat X Visit		
a. B2 High Fat Kcals versus PC1 High Fat Kcals	B2/PC1	.01
Carbohydrate X Visit		
a. B2 High Sugar Foods versus PC1 High Sugar Foods	B2/PC1	.00
Fat X Carbohydrate X Visit Interaction		
a. B2 High Fat/ High Sugar versus PC1 High Fat/ High Sugar	B2/PC1	.00

Fat Preference

Within-subjects ANOVAs were done to assess Fat Preference at Baseline 2 and Postcessation 1 for 17 B2/PC1 subjects and at Baseline 2, Postcessation 1 and Postcessation 2 for the 10 B2/PC1/PC2 subjects. Statistically nonsignificant differences were found for the 17 B2/PC1 subjects ($F(1,16)=.80, p=.39$) and for 10 B2/PC1/PC2 ($F(2,18) = 1.52, p = .25$).

Multiple Regression Analyses

It was previously proposed to conduct regression analyses to determine whether dietary restraint, BMI, waist/ hip ratio, disinhibition and weight concern variables could predict postcessation changes in fat and other specific macronutrient intake and body weight.

Dietary restraint, disinhibition, BMI, waist to hip ratio, and weight concern were investigated to identify if any of these variables predicted the change in total kilocalories, fat kilocalories intake, sugar kilocalories intake, the intake of High Fat/High Sugar foods or body weight from Baseline 2 to Postcessation 1. Both simultaneous and stepwise methods of multiple regression analyses were attempted, however the independent

variables/ predictors did not meet the SPSS program's minimal criteria for predictive value; and therefore, this software excluded all independent variables from the analysis.

Dietary restraint, disinhibition, BMI, waist to hip ratio, and weight concern were investigated to identify if any predicted the change in total kilocalories intake from Baseline 2 to Postcessation 2. Both simultaneous and stepwise methods of multiple regression analyses were attempted, however the independent variables/ predictors did not meet the SPSS program's minimal criteria for predictive value; and therefore, this software excluded all independent variables from the analysis.

B2 High Fat/ High Sugar, B2 High Fat/ High Complex Carbohydrate, B2 High Fat/ High Protein, B2 Low Fat/ High Sugar, B2 Low Fat/ High Complex Carbohydrate, B2 Low Fat/ High Protein, B2 Total Fat kilocalories intake, and fat preference were investigated to identify if any predicted the change in body weight from Baseline 2 to Postcessation 1. Both simultaneous and stepwise methods of multiple regression analyses were attempted, however the independent variables/ predictors did not meet the SPSS program's minimal criteria for predictive value; and therefore, this software excluded all independent variables from the analysis

B2 High Fat/ High Sugar, B2 High Fat/ High Complex Carbohydrate, B2 High Fat/ High Protein, B2 Low Fat/ High Sugar, B2 Low Fat/ High Complex Carbohydrate, B2 Low Fat/ High Protein, B2 Total Fat kilocalories intake, PC1 High Fat/ High Complex Carbohydrate, PC1 High Fat/ High Protein, PC1 Low Fat/ High Sugar, PC1 Low Fat/ High Complex Carbohydrate, PC1 Low Fat/ High Protein, PC1 Total Fat kilocalories intake, and fat preference were investigated to identify if any predicted the change in body weight from Baseline 2 to Postcessation 2. Both simultaneous and stepwise methods of multiple regression analyses were attempted, however the independent variables/ predictors did not meet the SPSS program's minimal criteria for predictive value; and therefore, this software excluded all independent variables from the analysis

DISCUSSION

Total Kilocalories (Kcals) Intake

Women gain six to twelve pounds in the year following smoking cessation, and significant increases in caloric intake are responsible for this weight gain (Perkins et. al., 1990; U.S. Department of Health and Human Services, 2001). However, researchers have not been able to delineate if specific increases in certain macronutrients (fat, sugar, protein, or complex carbohydrates) are primarily responsible for the increase in caloric intake found postcessation. Further, previous studies have been limited in their findings, because only increases in high or low fat foods were studied, without any consideration for other macronutrient content of foods. This study confirmed previous findings, in that significant increases in food intake were found postcessation. The 17 B2/PC1 subjects and 10 B2/PC1/PC2 subjects consumed more kilocalories following smoking cessation. And although only the 17 B2/PC1 subjects yielded a significant difference, the 10 B2/PC1/PC2 subjects did also yield a marginally nonsignificant increase in total kilocalories consumed. Thus this research suggests that an increase in the total kilocalories consumed is likely to be a concern at approximately one-month and six-months smoking cessation.

Total Fat (Kcals) Intake

There has also been considerable controversy as to the extent to which fat intake increases following smoking cessation. Most research has found considerable increases in fat intake following short-term abstinence, however some studies have found no change in fat intake following smoking cessation (Rodin, 1987; Ogden et al., 1994). This study found an increase in kilocalories of fat sources consumed for the 17 B2/PC1 subjects and the 10 B2/PC1/PC2 subjects. However, significant findings were only found for the 17 B2/PC1 subjects and not for the 10 B2/PC1/PC2 subjects. These results suggest that there is not only an increase in total kilocalories consumed, but also an increase in kilocalories consumed as fat postcessation.

Food Selection (Kcals Intake) From Specific Macronutrient Sources

Past research has indicated that increases in foods high in fat are primarily responsible for the increase in food intake postcessation (Eck et al., 1997; Allen, Hatsukami, Christianson & Brown, 2000). However, these studies have neglected to significantly and systematically vary macronutrient content further, thereby missing any other influencing variables. This study confirms these findings, as a significant increase in high fat foods was found for the 17 B2/PC1 subjects. Comparable increases in low fat foods were not observed from baseline to smoking cessation, further suggesting that the increase in kilocalories consumed following smoking cessation is primarily a result of an increase in the consumption of high fat foods. Therefore, it can be inferred from these results that an increase in foods high in fat is primarily responsible for the increase in caloric intake following smoking cessation. This conclusion is in agreement with much of the literature on smoking cessation, and could be misconstrued as the “complete” picture in postcessation caloric intake, if the experimenter ceased further manipulations of the independent variables. This is the method in which most past research has

followed, in fact, it is only in recent years that researchers have considered specific macronutrients.

This study further significantly and systematically varied both high and low fat foods into the categories of high sugar, high complex carbohydrate and low Carbohydrate/ high Protein, to investigate whether possible changes in these macronutrients were contributing to the increase in intake of high fat foods postcessation. Previous research has not yielded consistent findings. Eck et al. (1997), found significant increases in all macronutrient groups (fat, sugar, complex carbohydrate, and protein) postcessation; while other research has indicated that significant increases in fat and sugar are chiefly responsible for the postcessation caloric increase (Grunberg, 1982; Grunberg et al, 1985; Hall et al., 1989); and still others assert that increases in carbohydrate intake are accountable for the increase in caloric intake (Klesges et al., 1990); and then some even assert that increases in fat, sugar and carbohydrate are the primarily accountable for the differential intake postcessation (Eck et al., 1997). Significant increases in the consumption of foods high in sugar from Baseline 2 to Postcessation 1 were found for the 17 B2/PC1 subjects. These results affirm that the postcessation increase in fat is driven specifically by foods high in sugar and not by changes in the intake of high complex carbohydrates or low carbohydrate/ high protein foods.

No studies have significantly and systematically varied foods by fat (high and low) and carbohydrate (High Sugar, High Complex Carbohydrate and Low Carbohydrate/ High Protein) to investigate increases in food intake postcessation. One study that did vary fat and carbohydrate found that in rats, nicotine cessation and in humans, smoking cessation is accompanied by a significant increase in the consumption of sweet-tasting, high-fat foods (Grunberg et al., 1985).

This study further affirmed these results in that a significant increase in consumption of foods high in fat and high in sugar was found for the 17 B2/PC1/PC2 subjects. These results suggest that the increase in high fat foods found previously was actually qualified by the increase in high fat/ high sugar foods following smoking cessation, however methodologic issues masked the effect. The post hoc Bonferroni t-test for the levels of High Fat/High Complex Carbohydrate, High Fat/ Low Carbohydrate, High Protein, Low Fat/ High Sugar, Low Fat/ High Complex Carbohydrate and Low Fat/ Low Carbohydrate, High Protein all yielded nonsignificant results; thus, further suggesting that foods high in fat and high in sugar are primarily responsible for previous findings that foods high in fat were the increase postcessation. By significantly and systematically varying fat and carbohydrate content, results suggest that significant increases in no other foods except those high in fat and sugar can be primarily responsible for weight gain postcessation.

Body Weight

Women traditionally gain 6-12 pounds in the first year of smoking cessation, and the majority of that weight gain happens in the first 24 weeks of smoking cessation (U.S. Department of Health and Human Services, 2001). In this study the 17 B2/PC1 subjects gained < 1 pound at 3 weeks cessation and the 10 B2/PC1/PC2 subjects gained approximately 3 pounds at 24 weeks smoking cessation. All of the subjects for this analysis were part of the group condition of the STOP Study. Although this condition is used as the control for the study, minimal intervention is also given to this condition. This condition is given food pyramid, nutritional advice and general exercise guidelines in order to maintain their weight after smoking cessation. The minimal weight gain that

the 17 B2/PC1 and 10 B2/PC1/PC2 subjects experienced implies that the food pyramid, nutritional information and general exercise guidelines are helping the subjects maintain their body weight. Thus it can be suggested that the food pyramid, nutritional information and general exercise guidelines should be used in smoking cessation programs for women so that weight gain can be minimized.

Multiple Regression

Multiple regression analyses (both simultaneous and stepwise methods) were attempted to assess what factors predicted weight gain postcessation. However, the predictive value of the independent variables/ predictors was so minimal that SPSS excluded the predictors from the analysis. The minimal weight gain of the 17 B2/PC1 (< 1 pound at 3 weeks cessation) and the 10 B2/PC1/PC2 subjects (approximately 3 pounds at 24 weeks smoking cessation) also contributes to the lack of predictive value of the independent variables. The small range in the body weight change makes it even more difficult to find any significant predictors in the regression model.

Limitations

The MSSP results for the 17 B2/PC1 subjects yields a view of the changes in food intake from baseline to approximately one to four weeks cessation and the MSSP results for the 10 B2/PC1/PC2 subjects assesses the changes in food intake from baseline to one to four weeks to 24 weeks smoking cessation. The smoking cessation literature lacks many studies that assess food intake past a few weeks smoking cessation, and it is therefore unfortunate that the findings of the 10 B2/PC1/PC2 subjects are limited because of the small sample size. This lack of long-term follow-up is partially due to the fact research investigating the change in food intake following smoking cessation is traditionally plagued by relapsing subjects, in fact only approximately 25% of all subjects that start smoking cessation programs are successful (defined as maintained smoking cessation) (Department of Health and Hospitals, 2001). And although this analysis had better than average relapse issues, half of the original sample size had relapsed at the time of their MSSPs and had to be excluded from this analysis. However, in spite of this problem, the results of this study suggest that the specific changes in food intake can be inferred for long-term smoking cessation because nonsignificant trends for the 10 B2/PC1/PC2 were similar to significant results obtained for the 17 B2/PC1 subjects. And this was especially true for the changes in the consumption of high fat foods and high fat/high sugar foods. The Bonferroni t-tests for the 10 B2/PC1/PC2 subjects did approach significance for High Fat foods and for High Fat/ High Sugar foods, thus suggesting that the initial increase in High Fat/ High Sugar foods is maintained six months smoking cessation. The change in food intake from Baseline 2 to Postcessation 1 to Postcessation 2 would have allowed for a more long-term view of the pattern of food intake following smoking cessation.

Ethical concerns that require the researcher to protect human subjects can result in small effect sizes. In assisting women to quit smoking the subjects were divided into the “group” and “individualized” condition; and the group condition serves as the control. The group condition was given food pyramid, nutritional information and general exercise guidelines by a psychologist, dietician and exercise physiologist throughout the study. The minimal gain in body weight found in both the 17 B2/PC1 subjects and the 10 B2/PC1/PC2 subjects is a testament to the usefulness of this information. Subjects did

use the food pyramid, nutritional information, and exercise guidelines to successfully maintain their body weight. Therefore the results of the MSSP and FPQ could be diminished due to the fact that the subjects were consciously trying to maintain body weight by avoiding high fat, high sugar foods. And although significant differences in total kilocalories intake and in total fat kilocalories intake were found, other effects of smoking cessation on food intake could be masked because the subjects were informed about the dangers of weight gain postcessation.

CONCLUSIONS

Regardless of statistical power issues, suggestions about food intake following smoking cessation can be made—food intake does significantly increase following smoking cessation and this increase primarily consists of high fat/ high sugar foods. In this study it was found that women significantly increase their intake in foods high in fat and sugar following one month cessation and that comparable increases in no other fat by carbohydrate combination were found. Therefore smoking cessation programs that are trying to help women maintain their weight can give informed advice on how food intake will change following smoking cessation and possibly give low fat alternatives to high fat, high sugar foods to reduce weight gain.

REFERENCES

- Allen, S.S., Hatsukami, D., Christianson, D., & Brown, S. (2000). Energy intake and energy expenditure during the menstrual cycle in short term smoking cessation. Addictive Behaviors, 25 (4), 559-572.
- Blundell, J.E., Lawton, C. L., & Halford, J.C.G. (1995). Serotonin, Eating Behavior, and Fat Intake. Obesity Research, 3, (4), 471-476.
- Bowen, D.H., Grunberg, N.E. (1990). Variations in food preference and consumption across the menstrual cycle. Physiology & Behavior, 47, 287-291.
- Brobeck, J.R., Wheatland, M., & Strominger, J.L. (1947) Variations in regulation of energy exchange associated with estrus, diestrus and pseudopregnancy in rats. Endocrinology, 40 (2), 65-72.
- Buffenstein, R., Poppitt, S.D., McDevitt, R.M., & Prentice, A.M. (1995). Food intake and the menstrual cycle: a retrospective analysis, with implications for appetite research. Physiology and Behavior, 58 (6), 1067-1077.
- CDC. (1991). Cigarette smoking among adults—United States. MMWR, 42, 230-233.
- CDC. (1993). Mortality trends for selected smoking-related cancers and breast cancer—United States, 1950-1990. MMWR 42 (857), 863-868.
- Czaja, J.A., Goy, R.W. (1975). Ovarian hormones and food intake in female guinea pigs and rhesus monkeys. Hormonal Behavior, 6, 329-349.
- Dalvit, S.P. (1981). The effect of the menstrual cycle on patterns of food intake. American Journal of Clinical Nutrition, 34, 1811-1815.
- Debon, M., & Klesges, R.C. (1995). Smoking and smoking cessation: current conceptualizations and directions for future research. In A.J. Goreczny (Ed.), *Handbook of Health and Rehabilitation Psychology* (pp.135-156). New York: Plenum Press.
- Eck, L.H., Klesges, R.C., Meyers, A.W., Slawson, D.L. & Winders, S.A. (1997). Changes in food consumption and body weight associated with smoking cessation across menstrual cycle phase. Addictive Behaviors, 22 (6), 775-782.
- Fernstrom, J.D., & Wurtman, R.J. (1971). Brain serotonin content: Increase following ingestion of carbohydrate diet. Science, 174, 1023-1025.
- French, S.A., Perry, C.L., Leon, G.R., & Fulkerson, J.A. (1994). Weight concerns, dieting behavior and smoking initiation in adolescents: A prospective epidemiologic study. American Journal of Public Health, 84, 1818-1820.

Geiselman, P.J., Anderson, A.M., Dowdy, M.L. West, D.B., Redman, 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.

Geiselman, P.J., Martin, P.D., VanderWeele, D.A., Novin, D. (1981). Dietary self-selection in cycling and neonatally ovariectomized rats. Appetite, 2, 87-101.

Gilbert, C., & Gillman, J. (1956). The changing patterns of food intake and appetite during the menstrual cycle of the baboon (*papio ursinus*) with a consideration of some of the controlling endocrine factors. South African Journal of Medical Sciences, 21, 75-88.

Grunberg, N.E. (1982). The effects of nicotine and cigarette smoking on food consumption and taste preferences. Addictive Behaviors, 7, 317-331.

Grunberg, N.E. (1986). Nicotine as a psychoactive drug: Appetite regulation. Psychopharmacology Bulletin, 22 (3), 875-881.

Grunberg, N.E., Bowen, D. J., Maycock, V.A., & Nespor, S.M. (1985) The importance of sweet taste and caloric content in the effects of nicotine on specific food consumption. Psychopharmacology, 87, 198-203.

Grunberg, N.E., Bowen, D.J., Morse, D.E., (1984). Effects of nicotine on body weight and food consumption in rats. Psychopharmacology, 83, 93-98.

Grunberg, N.E., Popp, K.A., & Winders, S.E. (1988). Effects of nicotine on body weight in rats with access to “junk” foods. Psychopharmacology, 94, 536-539.

Grunberg, N.E., Winders, S.E., & Popp, K.A. (1987). Sex differences in nicotine’s effects on the consummatory behavior and body weight in rats. Psychopharmacology, 91, 221-225.

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, 81-86.

Hall, S.M., Tunstall, C.D., Vila, K.L., & Duffy, J. (1992) Weight gain prevention and smoking cessation: cautionary findings. American Journal of Public Health, 82, 799-803.

Harris, R.E., Zang, E.A., Anderson, J.I., and Wynder, E.L. (1993). Race and sex differences in lung cancer risk associated with cigarette smoking. International Journal of Epidemiology, 22,(4): 592-599.

Jeffery, R.W., Henrikus, D.J., Lando, H.A., Murray, D.M. & Liu, J.W. (2000) Reconciling conflicting findings regarding postcessation weight concerns and success in smoking cessation. Health Psychology, 19 (3), 242-246.

Kemnitz J.W., Eisele, S.G., Lindsay, K.A., Perelman, R.H., & Farrell, P.M. (1984). Changes in food intake during menstrual cycles and pregnancy of normal and diabetic rhesus monkeys. Diabetologia, *26*, 60-64.

Klesges, R.C., Eck, L.H., Isbell, T.R., Fulliton, E. & Hanson, C.L. (1990). The effects of smoking status on the dietary intake, physical activity and body fat of adult men. American Journal of Clinical Nutrition, *51*, 784-789.

Klesges, R.C., & Klesges, L.M. (1988). Cigarette smoking as a dieting strategy in a university population. International Journal of Eating Disorders, *7* (3), 413-419.

Klesges, R.C., Mizes, J.S., & Klesges, L.M. (1987). Dieting strategies in a college population. International Journal of Eating Disorders, *6*, 409-417.

Leibowitz, S.F., Weiss, G.F., & Shor-Posner, G. (1988). Hypothalamic Serotonin: Pharmacological, Biochemical, and Behavioral Analyses of Its Feeding-Suppressive Action. Neuropharmacology, *11*, (1), S51-S71.

Li, E.T.S, Tsang, L.B.Y., Lui, S.S.H. (1999). Menstrual cycle and voluntary food intake in young Chinese women. Appetite, *33*, 109-118.

Morin, L.P., & Fleming, A.S. (1978). Variation of food intake and body weight with estrous cycle, ovariectomy, and estradiol benzoate treatment in hamsters. Journal of Comparative and Physiological Psychology, *92* (1), 1-6.

Ogden, J. & Fox, P. (1994). Examination of the use of smoking for weight control in restrained and unrestrained eaters. International Journal of Eating Disorders, *16* (2), 177-185.

Ota, K., Yokoyama, A. (1967). Body weight and food consumption of lactating rats: effects of ovariectomy and of arrest and resumption of suckling. Journal of Endocrinology, *38*, 231-264.

Owen-Smith, V., Hannaford, P.C. (1999) Stopping smoking and body weight in women living in the United Kingdom. British Journal of General Practice, *49*, 989-990.

Perkins, K.A. (1992). Effects of tobacco smoking on caloric intake. British Journal of Addiction, *87*, 193-205.

Perkins, K.A., Epstein, L.H., Fonte, C., Mitchell, S., & Grobe, J.E. (1995) Gender, dietary restraint, and smoking's influence on hunger and the reinforcing value of food. Physiology & Behavior, *57*, 675-680.

Perkins, K.A., Mitchell, S.L. & Epstein, L.H. (1995). Physiological and subjective responses to food cues as a function of smoking abstinence and dietary restraints. Physiology & Behavior, *58* (2), 373-378.

Peterson, A.L. & Helton, J. (2000). Smoking cessation and weight gain in the military. Military Medicine, *165*, 536-538.

Pirie, P.L., McBride, C.M., Hellerstedt, W., Jeffrey, R.W., Hatsukami, D., Allen, S., & Lando, H. (1992). Smoking cessation in women concerned about weight. American Journal of Public Health, 82, 1238-1243.

Pomerleau, C.S., & Kurth, C.L. (1996). Willingness of women smokers to tolerate postcessation weight gain. Journal of Substance Abuse, 8, 3, 371-378.

Pomerleau, C.S., Pomerleau, O.F., Namenek, R.J. & Mehringer, A.M. (2000) Short-term weight gain in abstaining smokers. Journal of Substance Abuse Treatment, 18, 339-342.

Robinson, L.A., Klesges, R.C., Zbikowski, S.M. & Glaser, R. (1997). Predictors of risk for different stages of adolescent smoking. Journal of Consulting and Clinical Psychology, 65, 653-662.

Rodin, J. (1987). Weight change following smoking cessation: The role of food intake and exercise. Addictive Behaviors, 12, 303-317.

Spring, B., Wurtman, J., Gleason, C., Wurtman, R., & Kessler, K. (1991). Weight gain and withdrawal symptoms after smoking cessation: A preventative intervention using d-Fenfluramine. Health Psychology, 10, 216-223.

Strauss, R.S., & Mir, H.M. (2001). Smoking and weight loss attempts in overweight and normal weight adolescents. International Journal of Obesity, 25, 1381-1385.

Subar, A.F., Harlan, L.C., Mattson, M.E. (1990). Food and nutrient intake differences between smokers and non-smokers in the US. American Journal of Public Health, 80 (11), 1323-1329.

Tarusuk, V., Beaton, G.B. (1991). Menstrual-cycle patterns in energy and macronutrient intake. American Journal of Clinical Nutrition, 53, 442-447.

U.S. Department of Health and Human Services. (2001). Women and Smoking: A Report of the Surgeon General. Washington DC: U.S. Government Printing.

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. New England Journal of Medicine, 324, 739-745.

APPENDIX A: FIGURES

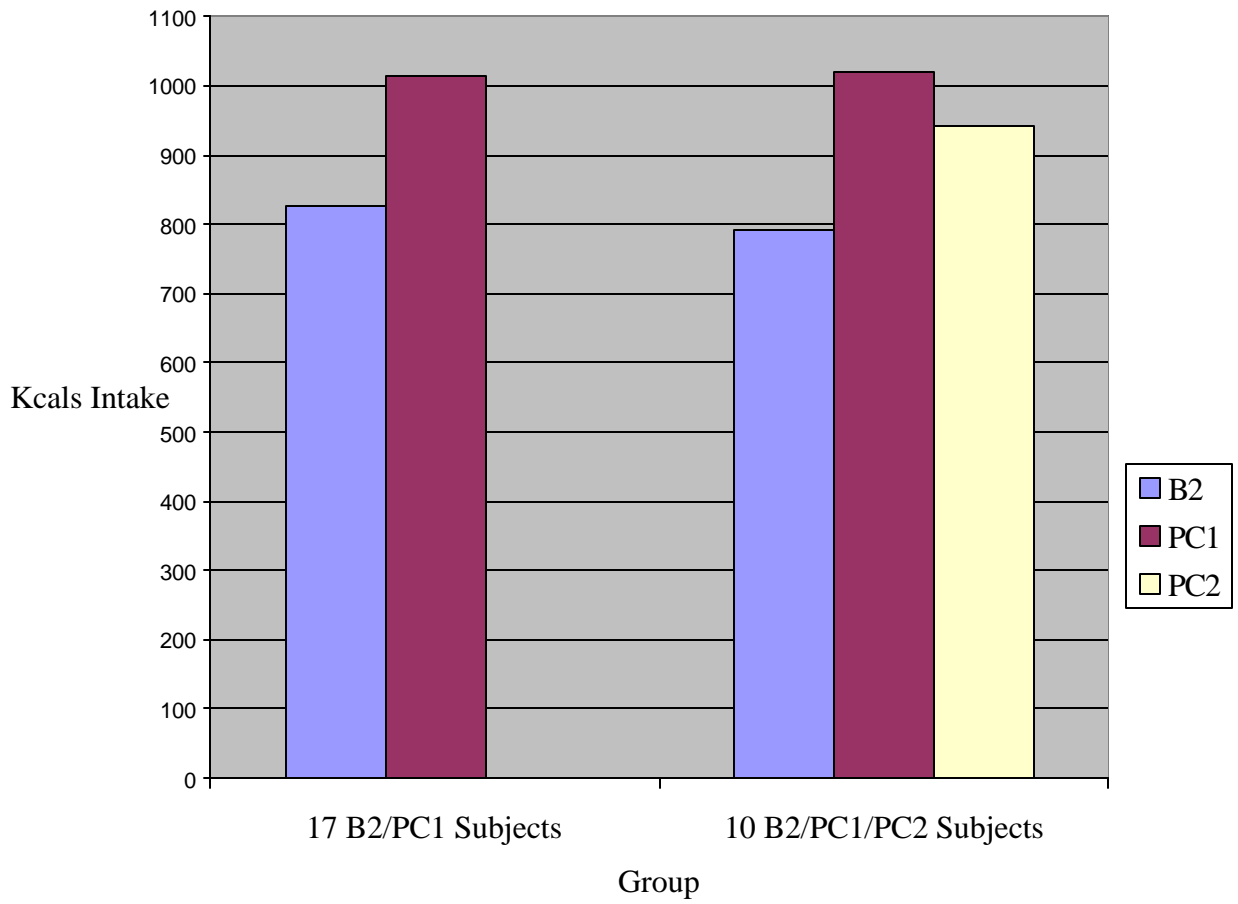


FIG. 1. Total Kilocalories Intake measured at the following times: B2 and PC1 for the 17 B2/ PC1 subjects and B2, PC1, and PC2 for the 10 B2/PC1/PC2 subjects.

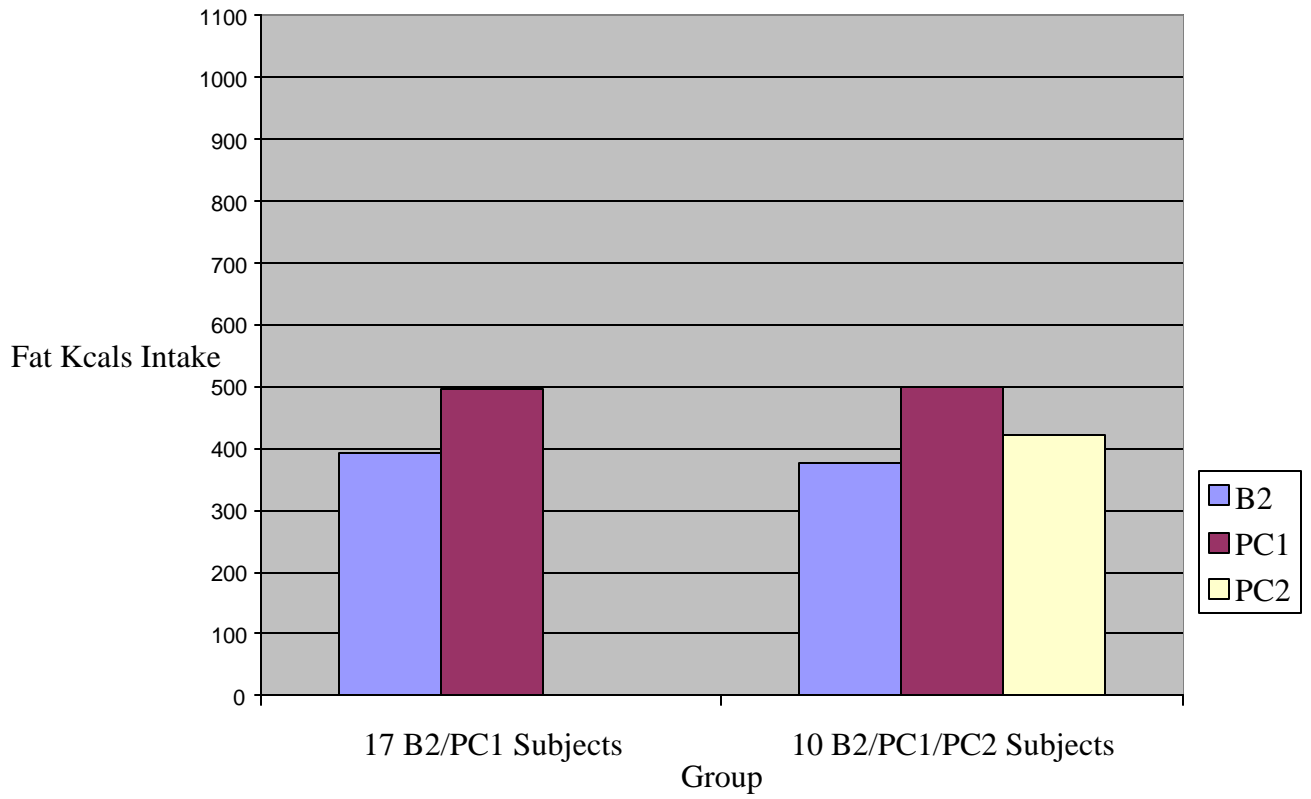


FIG. 2 Kilocalories Intake from Fat Sources measured at the following times B2 and PC1 for the 17 B2/PC1 subjects and B2, PC1, and PC2 for the 10 B2/PC1/PC2 subjects.

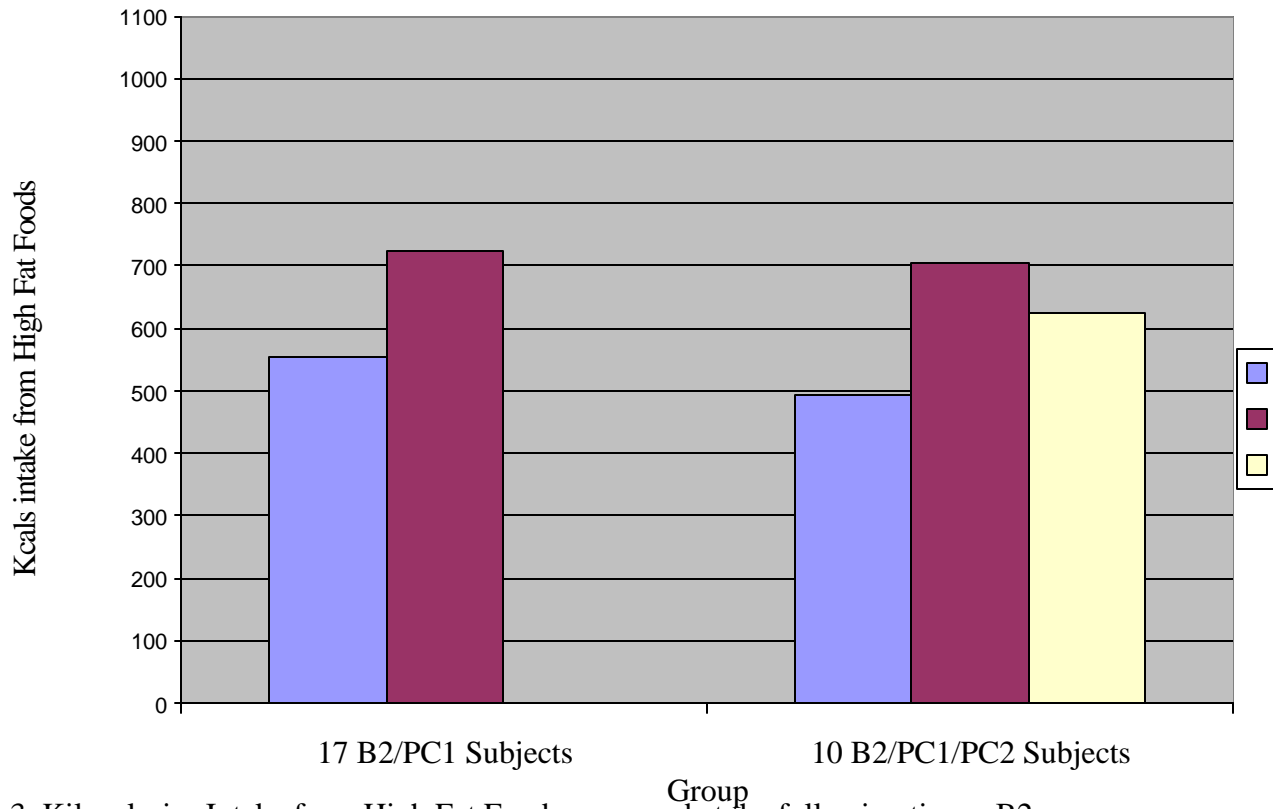


FIG. 3. Kilocalories Intake from High Fat Foods measured at the following times: B2 and PC1 for the 17 B2/PC1 subjects and B2, PC1, and PC2 for the 10 B2/PC1/PC2 subjects.

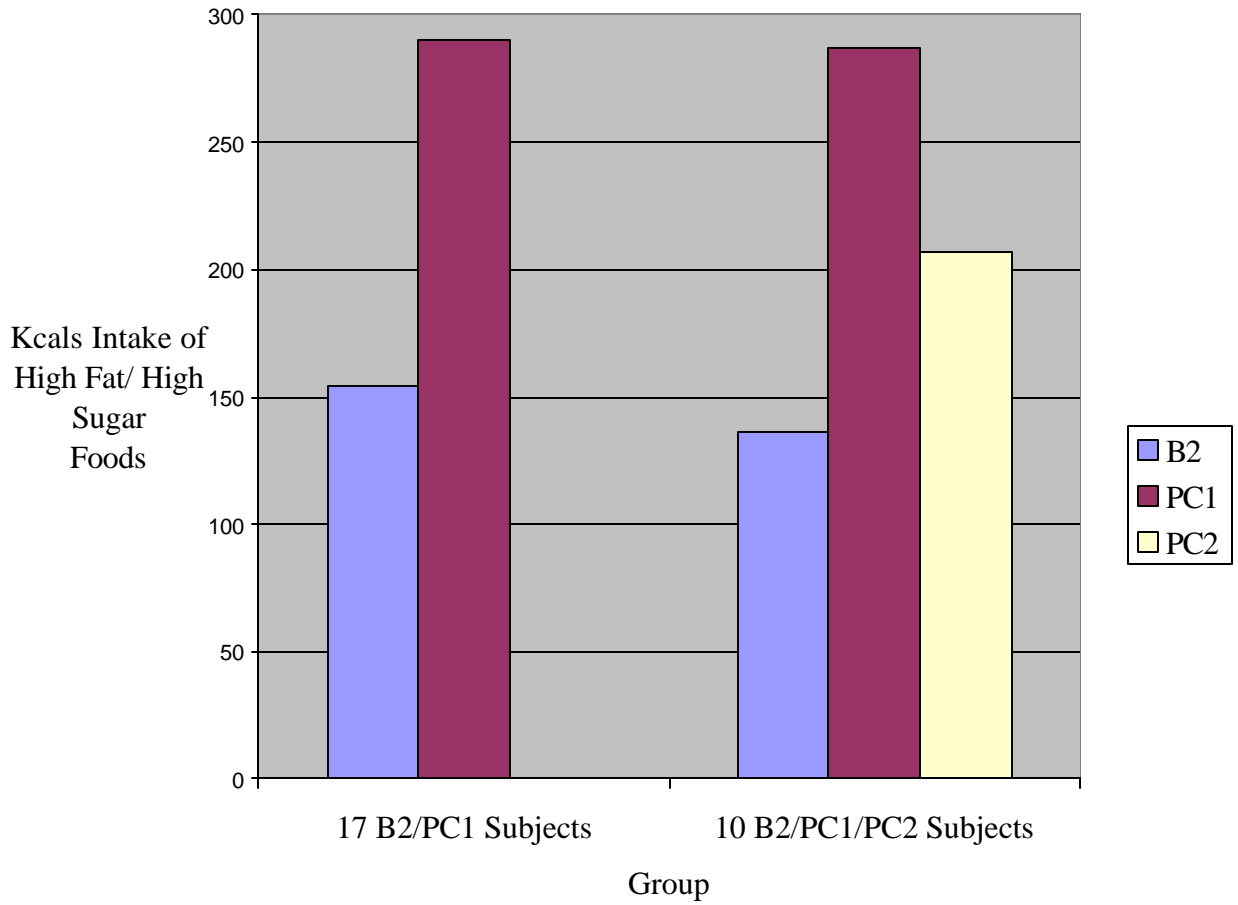


FIG. 4. Intake of High Fat/ High Sugar Foods measured in kilocalories at the following times: B2 and PC1 for the B2/PC1 subjects and B2, PC1, and PC2 for the B2/PC1/PC2 subjects.

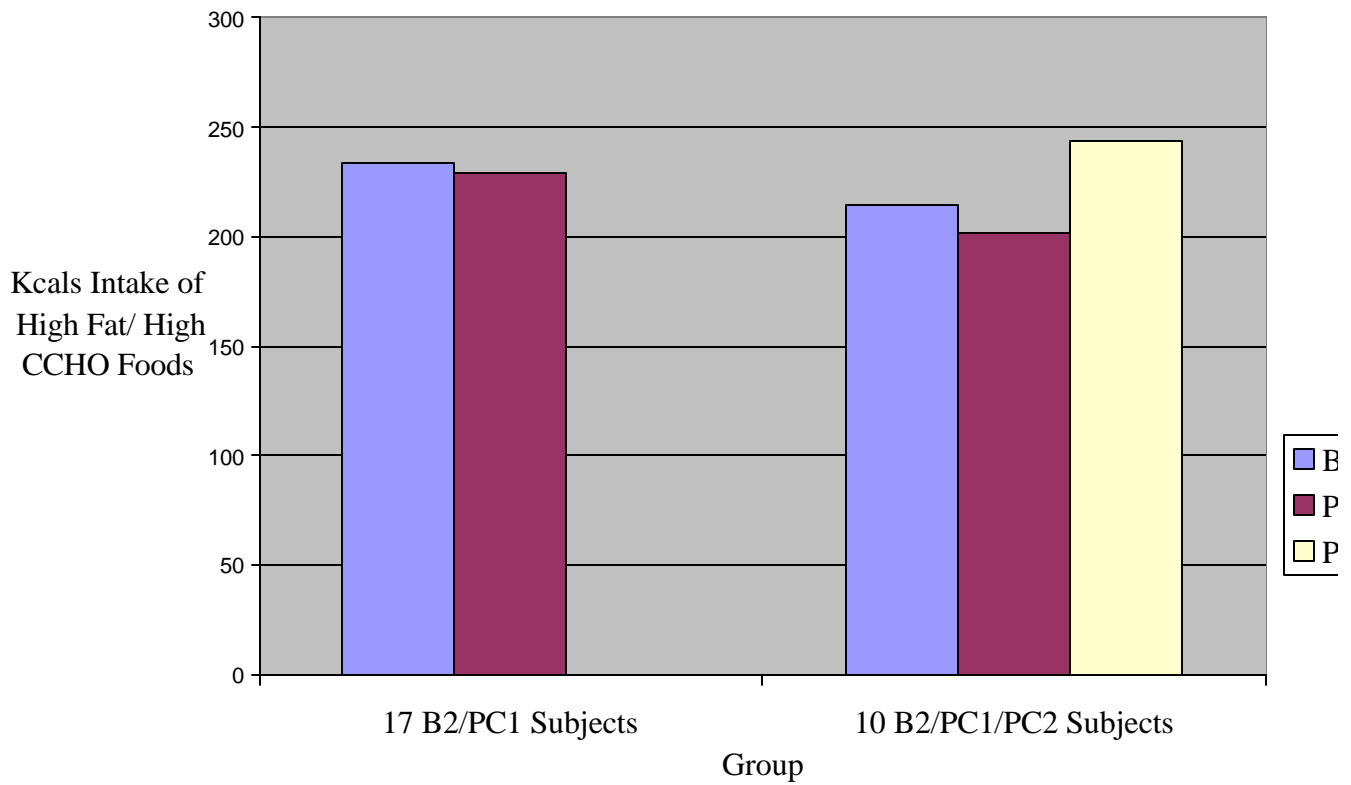


FIG. 5. Intake of High Fat/ High CCHO Foods measured in kilocalories at the following times: B2 and PC1 for the B2/PC1 subjects and B2, PC1 and PC2 for the B2/PC1/PC2 subjects.

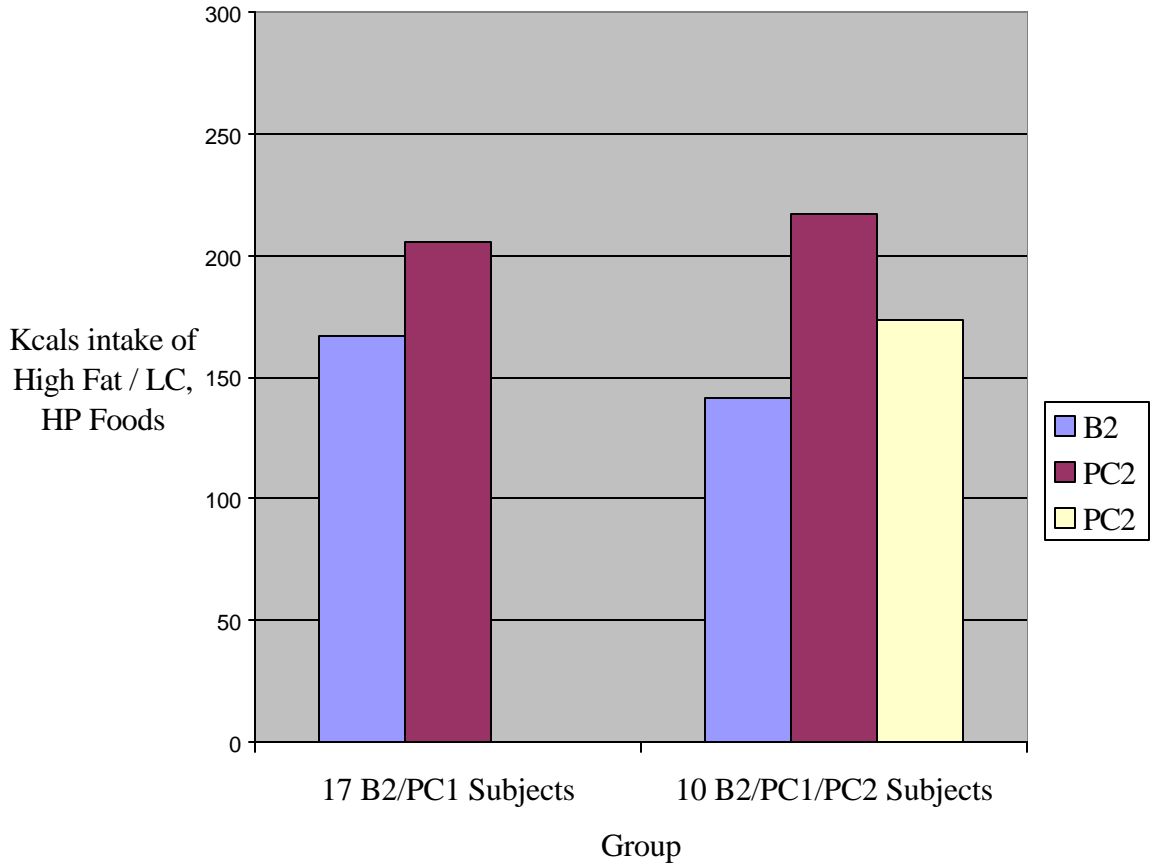


FIG. 6. Intake of High Fat/ Low Carbohydrate, High Protein Foods measured in kilocalories at the following times: B2 and PC1 for the B2/PC1 subjects and B2, PC1, and PC2 for the B2/PC1/PC2 subjects.

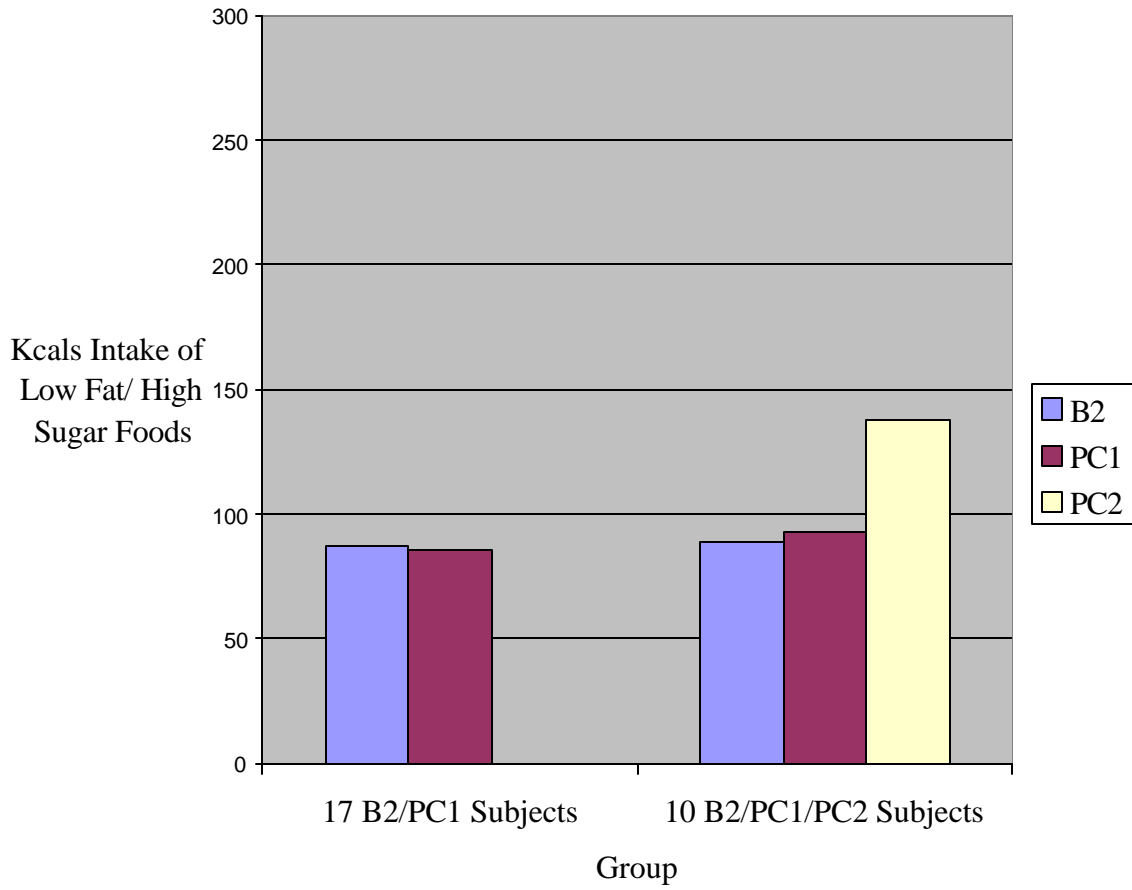


FIG. 7. Intake of Low Fat/ High Sugar Foods measured in kilocalories at the following times: B2 and PC1 for the 17 B2/PC1 subjects and B2, PC1, PC2 for the 10 B2/PC1/PC2 subjects.

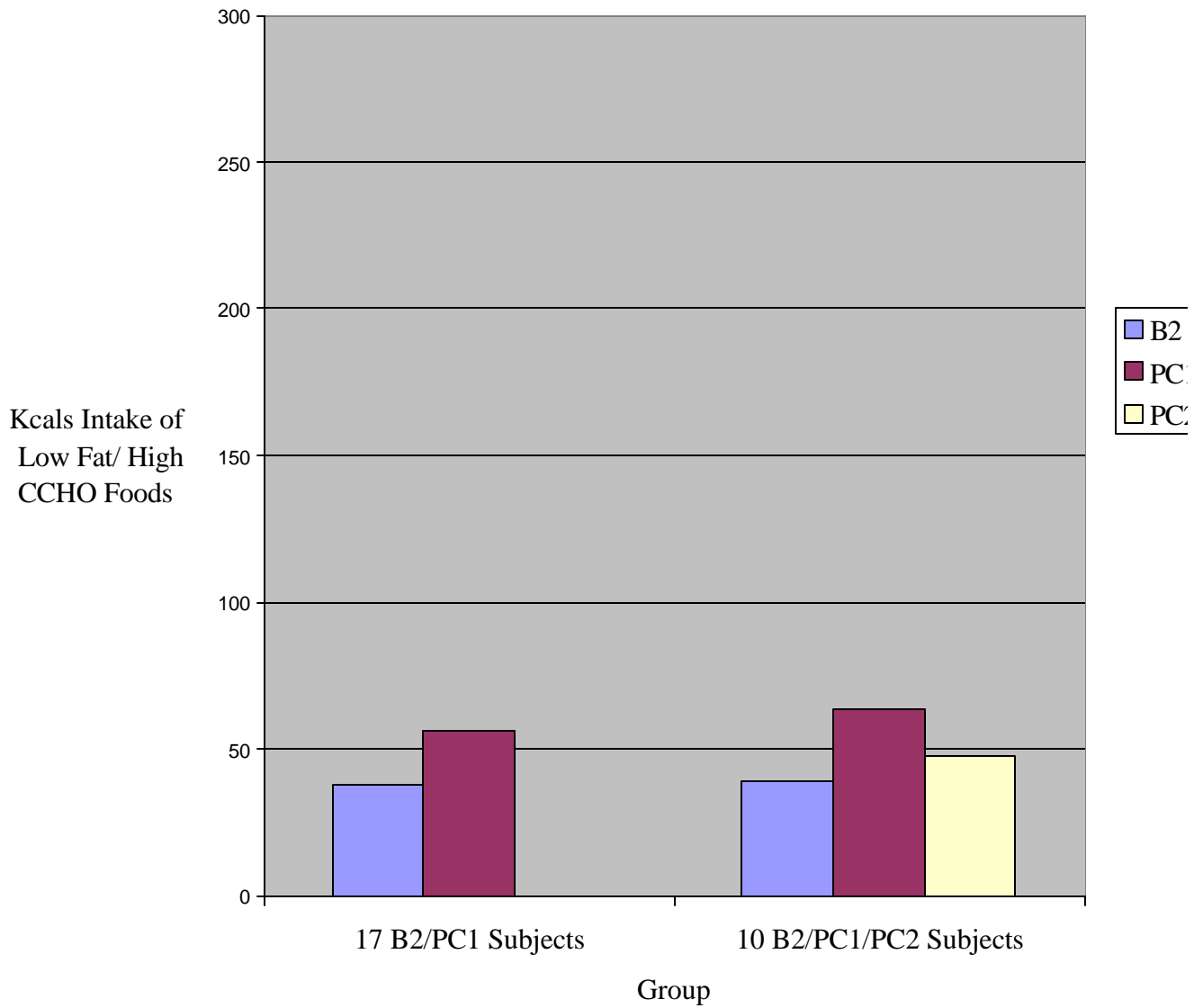


FIG. 8. Intake of Low Fat/ High CCHO Foods measured in kilocalories at the following times: B2 and PC1 for the B2/PC1 subjects and B2, PC1 and PC2 for the B2/ PC1/PC2 subjects.

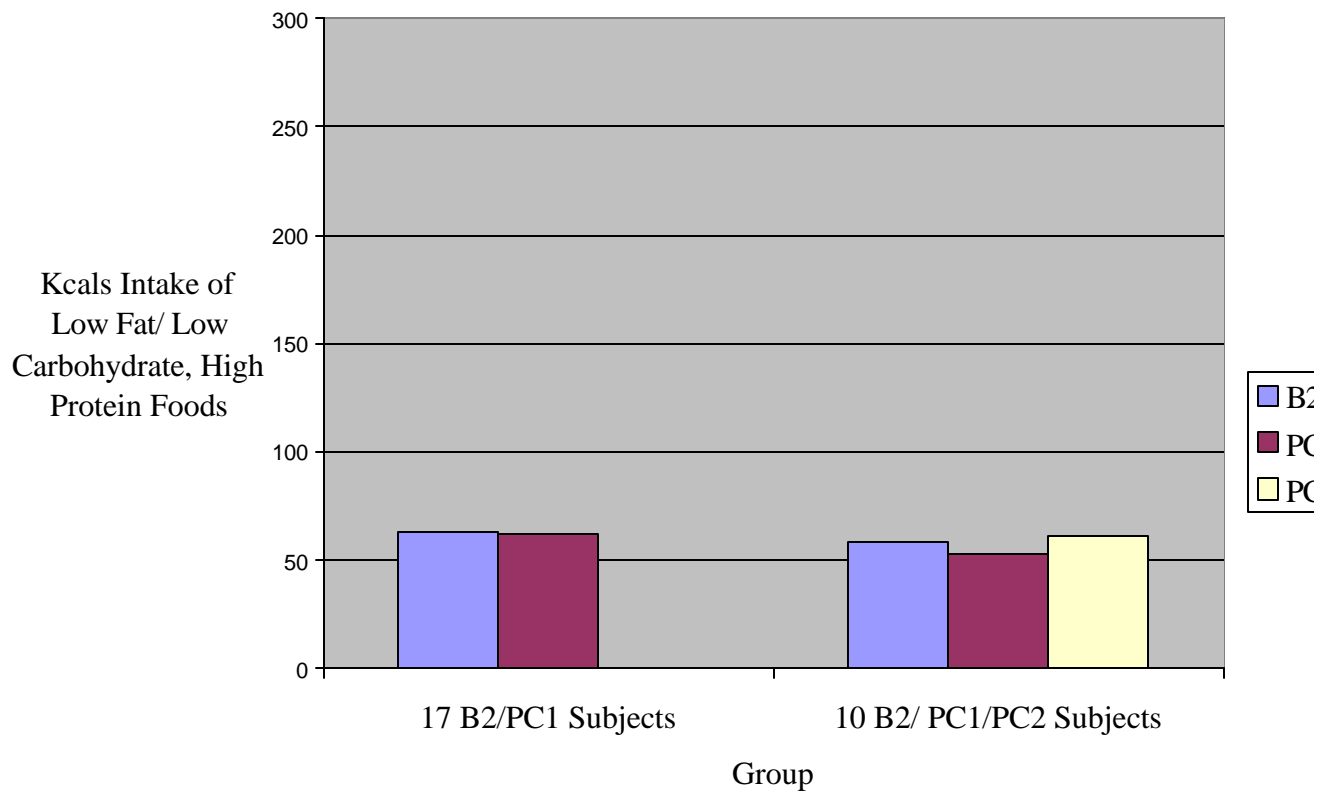


FIG. 9. Intake of Low Fat/ Low Carbohydrate, High Protein Foods measured in kilocalories at the following times: B2 and PC1 for the 17 B2/PC1 subjects and B2, PC1, and PC2 for the 10 B2/PC1/PC2 subjects.

APPENDIX B: RELEVANT QUESTIONNAIRE

STOP Study Food Preference Questionnaire

Please mark the box which indicates how much you like each of the following foods

1=Dislike Extremely

5=Neutral, Neither Like nor Dislike

9=Like Extremely

	Don't know/ Never tasted	1	2	3	4	5	6	7	8	9
chocolate layer cake										
pasta with alfredo sauce										
American cheese										
canned pears										
cream of wheat										
vanilla pudding										
roasted skinless chicken										
Snickers										
crescent rolls										
BBQ chicken wings										
canned apricots										
pita bread										
fat free string cheese										
pecan pie										
cream of celery soup										
mozzarella cheese										
banana, fresh										
long grain rice										
canned shrimp in water										
apple spice cake										
pizza rolls										
fried chicken leg										
dates, dried										
dill pickle										
stewed chicken breast										
vanilla ice cream										
onion rings										
pot roast										
bagel, plain										
ground turkey										
chocolate ice cream										
potato sticks										
hamburger patty										
prunes, dried										
white rice										
fat-free cheddar cheese										
Mounds coconut candy bar										
tortilla chips										

¹ Used with permission of Dr. Paula J. Geiselman

² Published in *Physiology & Behavior*, Vol. 63, No. 5, pp. 919-928, 1998.

	Don't know/Never tasted	1	2	3	4	5	6	7	8	9
prime rib										
popsicle, fruit flavored										
French bread										
roasted skinless turkey										
cheesecake, plain										
fast-food biscuit										
sirloin steak										
cantaloupe, fresh										
baked potato, plain										
turkey breast canned in water										
fudge brownie										
Stove- Top stuffing										
fried egg										
apple, raw										
sweet potato, baked, plain										
boiled crawfish										
chocolate cupcake with chocolate icing										
cheese straws										
peanut butter										
jelly, any flavor										
orange, fresh										
corn, whole kernel										
boiled shrimp										
M&M plain candies										
French fries										
fried catfish fillets										
watermelon, fresh										
Leeks										
broiled red snapper										
M&M peanut candies										
potato salad (mayonnaise type)										
scrambled eggs										
honeydew melon, fresh										
parsnips, cooked										
Spinach										
chocolate pudding										

¹ Used with permission of Dr. Paula J. Geiselman

² Published in Physiology & Behavior, Vol. 63, No. 5, pp. 919-928, 1998.

APPENDIX C: PERMISSION LETTER

January 8, 2002

Louisiana State University
Graduate School
David Boyd Hall, Room 114
Baton Rouge, LA 70808

To Whom It May Concern:

This letter is to formally give my permission to Jamie Neal to reprint the Food Preference Questionnaire in her thesis titled, Control of Food Intake and Body Weight Following Smoking Cessation in Premenopausal Women.

Sincerely,



Paula J. Geiselman Ph.D.
Pennington Biomedical Research Center
Louisiana State University

VITA

Jamie Neal was born and raised in New Orleans, Louisiana. Ms. Neal attended the University of Mississippi and received a Bachelor of Arts in Psychology in May, 1999. Ms. Neal now lives in LaPlace, Louisiana, and plans to marry in June 2002.