Almond Consumption and Dietary Compensation
in Overweight and Obese Adults

by
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A Thesis Presented in Partial Fulfillment
of the Requirements for the Degree
Master of Science

Approved April 2011 by
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May 2011
ABSTRACT

Epidemiological studies have suggested a link between nut consumption and weight. The possible effects of regular nut consumption as a method of weight loss has shown minimal results with 2-3 servings of nut products per day. This 8 week study sought to investigate the effect of more modest nut consumption (1 oz./day, 5 days/week) on dietary compensation in healthy overweight individuals. Overweight and obese participants (n = 28) were recruited from the local community and were randomly assigned to either almond (NUT) or control (CON) group in this randomized, parallel-arm study. Subjects were instructed to eat their respective foods 30 minutes before the dinner meal. 24 hour diet recalls were completed pre-trial and at study weeks 1, 4 and 8. Self-reported satiety data were completed at study weeks 1, 4, and 8. Attrition was unexpectedly high, with 13 participants completing 24 dietary recall data through study week 8. High attrition limited statistical analyses. Results suggested a lack of effect for time or interaction for satiety data (within groups p = 0.997, between groups p = 0.367). Homogeneity of of inter-correlations could not be tested for 24-hour recall data as there were fewer than 2 nonsingular cell covariance matrices. In conclusion, this study was unable to prove or disprove the effectiveness of almonds to induce dietary compensation.
ACKNOWLEDGEMENTS

I would like to thank Lucky Westeer for his support and encouragement while completing this Master's of Science. Thank you to my committee members. You were all a great help and I appreciated your combined input. Thank you to my family, whose humor continues to inspire.
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Chapter I

Introduction

The United States is in the grip of an obesity crisis. Obesity, defined as a body mass index (BMI) of 30 kg/m\(^2\) or greater, is an independent risk factor for many diseases including cardiovascular disease, type 2 diabetes, and some cancers (1). The obesity rate has been increasing in prevalence for 25 years (2) despite attempts to intervene by health professionals. This is perhaps unsurprising considering that Americans at every level of socioeconomic status have unprecedented access to calorie-dense, convenient, cheap and palatable food options (3). The scope of the problem is further defined by data from the National Health and Nutrition Examination Survey (NHANES), which reports that in 2007, 24.1% of U.S. adults reported no leisure time physical activity (4). A long-term energy imbalance, skewed towards weight gain, has lead to the current prevalence of obesity in the U.S., estimated at greater than 30% of adults (2) and between 12.4% and 17.6% of children depending on age range (2). The current need for effective treatment measures cannot be overstated.

Current treatments for obesity range from dietary and physical activity interventions to prescription medications to surgery. Research suggests that people tend not to adhere to diet and physical activity interventions over the long-term (6-8). Drugs have side-effects and do not lead to large amounts of weight loss (9, 10). Surgery, while effective for weight loss (11, 12), is invasive and succeeds at the cost of concessions to quality of life. Currently, surgery for weight loss is considered only when several criteria have been met, one being a documented failure at nonsurgical approaches to long-term weight loss.
Pharmacotherapy and surgery for weight loss generally fail to address obesity for most of the individuals affected. Clearly, there is a need for an effective weight loss approach that is easy to enact and that does not require people to make quality-of-life concessions.

Given that adherence to diet and physical activity interventions is a bottleneck in their effectiveness (6-8), it may be that consideration of the sustainability of behavioral changes has not been adequately addressed. Some evidence has suggested the efficacy of a small-change approach to weight loss (13-15). The idea is that several small lifestyle changes can be sustainable for people and simultaneously be effective for weight loss and weight management. This approach may then have relevance both for weight loss in the obese as well as for weight management in those of normal weight.

One component of a small-change approach could be related to peanut and tree nut consumption. Consumption of peanuts and tree nuts is inversely associated with risk of several diseases and with weight (16-18). This is interesting given the high kilocalorie density of peanuts and tree nuts. Peanuts and tree nuts are generally good sources of protein and fiber, factors shown to increase satiety or feelings of fullness (19).

A wealth of data links peanut and tree nut consumption to lowered rates of several diseases including obesity. However, very few studies have assessed the ability of peanuts and tree nuts to reduce weight in the obese or to prevent weight gain in the non-obese. In light of these facts, this study aimed to assess the effects of a one ounce supplement of almonds eaten before the dinner meal on dietary compensation throughout the day. We hypothesized that one ounce of almonds
preloaded before the dinner meal would induce dietary compensation in a sample of overweight and obese healthy individuals from the Phoenix area.

**Purpose of Study**

The purpose of this randomized, parallel-arm, placebo-controlled study was to assess the effectiveness of a one ounce supplement of almonds, preloaded before the evening meal, to induce dietary compensation throughout the day in a population of healthy overweight and obese subjects from the Phoenix area.

**Aims and Hypotheses**

- **Primary Aim:**
  To determine the effect of one ounce of almonds preloaded before the dinner meal on dietary compensation throughout the day in a sample of healthy overweight and obese subjects from the Phoenix area.

- **Primary Hypothesis:**
  One ounce of almonds preloaded before the dinner meal will decrease daily kilocalories consumed compared to a kilocalorie-matched control group in healthy overweight and obese individuals from the Phoenix area.

**Definitions**

**Body Mass Index:** A ratio of body weight to height squared. Used as a way to measure obesity which corresponds to a BMI of at least 30 kg/m².

**Satiety:** Feeling of fullness. Lack of desire to eat. In the present study satiety is based on a self-reported number between 0 and 100 (0 equals greatest imaginable hunger, 100 equals greatest imaginable fullness).

**Delimitations and Limitations**

Participants in this study represented a convenience sample from the
Phoenix area. Subjects were healthy, overweight (25-29.9 kg/m$^2$) or obese ($\geq$ 30 kg/m$^2$), aged 20-75 years, did not exercise vigorously greater than twice per week, had reliable access to the internet, and had no known nut or dairy allergies or intolerances. The automated self-administered 24-hour recall used to gather dietary data has not been validated as a dietary assessment tool. Additionally, adherence to the study protocol was based on self-report, therefore cannot be certain. In light of these delimitations, generalization to dissimilar populations is inappropriate.
Chapter 2

Review of Literature

Obesity

Prevalence

Obesity is defined as a body mass index (BMI) of 30 kg/m$^2$ or greater in adults. In children, a BMI at or greater than the 95th percentile by sex and age defines obesity. Currently in the United States the obese account for 35% of the adult population, according to data published from 2005-06 (5). Among children (19 years or less), obesity rates range from 12.4% to 17.6% depending on age group. Obesity rates have been increasing since the 1980s and have now reached levels such that approximately two-thirds of adults in the U.S. are overweight or obese (4).

Etiology

Obesity is excessive body fat accumulation and results from a long-term imbalance between energy intake and energy expenditure. In efforts to identify the root causes of obesity, researchers have considered a wide range of possibilities. These efforts generally fall into one of several categories: food environment factors, behavioral factors, genetic factors, and psychological factors. Obesity is a complex problem. While the mechanism of how obesity came to be as prevalent as it is today is still hotly debated, this section will present findings from several studies showing possible contributing factors to current obesity rates.

Smickiklas-Wright et al., in an effort to show trends towards increasing portion sizes, compared the differences in quantities consumed during eating
occasions between 1989-91 and 1994-96. This study considered data from the Continuing Survey of Food Intakes by Individuals (CSFII) between the two time periods. Results suggested that even in this relatively small time frame, portion sizes of several foods, including soft drinks, coffee, tea, and ready-to-eat cereal increased (20).

Harris et al. studied the effects of television food advertising on eating behavior. The researchers suggested that unhealthy food advertising was responsible for unhealthy food choices. To test the theory that advertising would increase consumption of unhealthy foods, Harris et al. had children watch a cartoon either with or without food commercials. Children were given a bowl of crackers (containing a known weight) to snack on during the cartoon, which they watched alone. Results of this study showed that children who saw a cartoon with food commercials ate significantly more crackers than children who saw the same cartoon with non-food commercials. The researchers postulated that if children continued this snacking behavior for only 30 minutes a day it would result in an additional 94 kilocalories per day. It is important to note that amount eaten by children was not related to hunger. Thus, all children, regardless of appetite, responded to food advertising (21).

In an expansion of the first study, Harris et al. sought to show in a second study that adults would respond to food advertising similarly to children. In addition, this second study examined whether subjects were eating in response to images of palatable foods, or if the product message also influenced eating behavior and food choice. Researchers had adults watch a television clip with one of three different commercial sets: a fun and exciting food set (fast food,
candy, soda drink), a nutrition message set (granola bar, orange juice, instant oatmeal), or a non-food control. Subjects were then moved to another room where they were presented with multiple food options in amounts known to the researchers. Subjects were instructed to taste each food item, which ranged from healthy (carrots) to unhealthy (chocolate chip cookies) and to rate each food on a variety of measures. Subjects were also told they could eat as much as they liked. Results showed that participants who saw unhealthy food advertising ate significantly more snacks than did those who saw healthy food advertising or controls. In addition, participants who saw unhealthy food advertising spent a longer amount of time eating than did participants in the other two groups (21). These combined studies suggest the powerful effects of television food advertising on eating behavior and suggest one possible contributing factor to the obesity epidemic.

Rolls et al. showed that increased portion sizes could contribute to obesity. Testing the effect of portion sizes on total amount eaten, the researchers fed subjects either 100%, 150%, or 200% of estimated energy needs for two consecutive days. Results showed that both men and women ate more kilocalories when given more food. This is significant in that the subjects did not compensate by eating less the second day. Thus, this study showed that increased portion size resulted in increased food and kilocalorie intake, which could, over time, contribute to obesity (22).

Rundle et al. examined the relation between neighborhood food environment and BMI when controlling for walkability. Researchers defined walkability as a half-mile radius around participants' home address.
Neighborhood food stores were classified into one of three categories: BMI healthy, BMI intermediate, or BMI unhealthy. Subjects were measured objectively for weight and height from which BMI was calculated. Results showed a significant association between walkability to BMI healthy food stores and BMI, such that access to BMI healthy food stores predicted lower BMI (3).

In an editorial from the New England Journal of Medicine, Jerome Kassirer suggested that there is an American ideal to be "slim, fit, and forever young." Also, the writer suggested that dieting behaviors are common in the U.S. (23) Researchers have examined whether there is a relationship between dieting behaviors and unhealthy eating habits. Gilhooly et al. studied the characteristics of foods craved while on energy-restricted diets. Participants were placed on energy-restricted diets for six months. Food cravings were assessed at baseline and again at six months. No difference in cravings was found, however, foods commonly craved by participants in this study were high in energy density and fat, and low in fiber and protein (24).

In a study by Swinburn et al., doubly-labeled water was used to estimate the relative contributions of energy intake to energy expenditure to the current obesity epidemic. As energy expenditure was positively correlated with weight, the researchers were able to conclude that energy intake drove the increase in obesity prevalence. Furthermore, the researchers suggested that reductions in weight would come only with large decreases in energy intake, large increases in energy expenditure, or a combination of both (25).

Many researchers have investigated to what extent genetics contribute to development of obesity. Obesity directly caused by a genetic mutation is rare and
so does not significantly contribute to current obesity rates. Genetics do play an important role, however, as heritability rates for obesity have been estimated between 40-70% (26). In a recent study by Cummings et al., researchers studied single nucleotide polymorphisms in a genetically homogenous sample of individuals living on the island of Mauritius. Researchers measured markers of the metabolic syndrome, including diabetes, as well as obesity-related phenotypes including fasting plasma glucose, waist-to-hip ratio, BMI, and fat mass. Results suggested a correlation between specific genetic variants and fat mass, which could indicate a genetic predisposition toward higher fat mass (27).

Jiao et al. examined the relationship between obesity and single gene polymorphisms (SNP) of the fibroblast growth factor receptor 1 (FGFR1) gene. To do this, researchers genotyped obese and lean adults and children and in so doing were able to relate SNPs in FGFR1 to obesity. Results showed a positive correlation between obesity and FGFR1 SNPs, with an odds ratio equal to 1.17 (28).

Many other SNPs are related to increased obesity risk. In fact, over 100 genes are suspected to influence obesity (29). Other specific examples include the fat mass and obesity (FTO) gene and the lipoprotein lipase (LPL) gene (29, 30). A detailed discussion of the genetic factors involved in the development of obesity is beyond the scope of this summary. It is notable that an increased predisposition toward obesity is associated with, but not caused by, SNPs, as the current obesity rates are likely a result of interactions between genetic and environmental factors (31).
Comorbidities of Obesity

Obesity often coexists with several other medical conditions. Brown et al. looked at the association between BMI and hypertension and dyslipidemia. Data were drawn from NHANES III, corresponding to the time frame from 1988-94. Results showed that higher BMIs were associated with higher diastolic and systolic blood pressures. Also, it was found that a BMI greater than 25 kg/m$^2$ was associated with higher blood cholesterol and lower high-density lipoprotein levels compared with BMIs less than 25 kg/m$^2$ (32). Obesity is also linked to higher rates of cardiovascular disease, diabetes, cancer, osteoarthritis, liver and kidney disease, sleep apnea, and depression (33).

In addition to increasing morbidity, excess adiposity increases risk of mortality. In an analysis of data from the Nurses Health Study, Manson et al. related BMI to mortality from different causes. In doing so, the researchers found that women with BMI values of 32 kg/m$^2$ or above had a relative risk of death from cardiovascular disease equal to 4.1 in comparison to those with a BMI below 19 kg/m$^2$. The relative risk of death from cancer was 2.1. Finally, these researchers found that the lowest mortality rate was observed in women that weighed at least 15% less than the national average for women of the same age (34).

In a similar study, Folsom et al. examined the health risks of obesity, but sought to distinguish between differing assessment methods. Researchers conducted a prospective cohort study of 31,702 women aged 55-69. Surveys were completed and sent by mail with information regarding participants BMI, waist-circumference, and waist-to-hip ratio. Results showed that all assessment
methods were predictive of diabetes and hypertension. When comparing the highest quintile to the lowest quintile for each assessment method, the relative risk of death was 1.2 for waist-to-hip ratio, 1.1 for waist circumference, and 0.91 for BMI. BMI was a better predictor of cancer incidence than waist-to-hip ratio. Finally, when multiple assessment techniques were used simultaneously, researchers found higher predictive potential (35).

Bigaard et al. examined the relation between waist circumference and body composition to all cause mortality in a study involving 27,178 men and 29,875 women aged 50-64 years. Results showed that for men the mortality relative risk was 1.36 times higher for every 10% increase in waist circumference. For women, the mortality relative risk was 1.3 times higher for every 10% increase in waist circumference. As the researchers were able to control for total adiposity, and as they found that waist circumference remained predictive of mortality, researchers concluded that increased mortality risk with excess adiposity is primarily due to abdominal adiposity (36).

**Treatments for Obesity**

**Lifestyle/Behavioral Interventions**

Stroebele et al. studied the impact of a small-change approach to weight reduction. To do this, 116 overweight and obese individuals were recruited to a one-week long program. Subjects were given the message to make small specific changes to their routines, such as cutting out 100 kilocalories a day, or increasing walking by 2000 steps per day. Subjects kept diet diaries and wore pedometers the week prior and week of the intervention. After comparison of the outcome variables of steps per day, total daily kilocalorie intake, macronutrient contents
and others pre and post intervention, participants showed statistically significant improvement in all areas (14).

Corbalan et al. looked at the effects of cognitive-behavioral therapy based on the Mediterranean diet for the treatment of obesity. In this study, 1406 obese participants underwent behavior modification, nutrition education, as well as increases in physical activity. Treatment lasted 34 weeks after which the average weight loss was 7.7 kilograms. Attrition (dropout rate) in this trial ranged from 4-9%. The researchers concluded that their program of cognitive-behavioral therapy was effective and clinically relevant (37).

In a landmark study, the Diabetes Prevention Program Research group sought to test the role of exercise and lifestyle interventions on prevention of diabetes compared with common drug therapy. The researchers recruited 3,234 non-diabetic persons with elevated fasting glucose levels to one of three groups: placebo, drug therapy (metformin), or a lifestyle modification program. The lifestyle modification program had the goal of reaching and maintaining a 7% reduction in weight through eating a reduced kilocalorie low-fat diet and moderate physical activity equaling 150 minutes per week. In addition, the lifestyle modification group attended a 16-lesson curriculum course covering topics related to behavior change, physical activity, and diet. Outcome variables of interest were weight loss, development of diabetes, and adherence. The average follow-up period for this study was 2.8 years (38).

Results of this study showed that after a four year follow-up the average weight loss maintained was 0.1, 2.1, and 5.6 kilograms for the placebo, drug therapy, and lifestyle modification groups, respectively. At four years, only 38%
of the lifestyle modification group had succeeded in maintaining a 7% reduction in body weight. Despite this, the incidence of diabetes was lowest in the lifestyle modification group, proving the efficacy of the program over drug therapy and placebo (38).

Jakicic et al. examined the effects of exercise of differing duration and intensity on weight loss. In this trial, 201 sedentary overweight and obese women were randomly assigned to one of four exercise groups. These were a vigorous intensity with high duration, a vigorous intensity with moderate duration, a moderate intensity with high duration, and a moderate intensity with moderate duration. All women were instructed to reduce daily kilocalorie intake to between 1200 and 1500 with kilocalories from fat equaling between 20% and 30%. This trial ran for 12 months, and by this time all groups had lost significant weight, with group means ranging from 8.9 to 6.3 kilograms. It is of note that there was a large variability in the amount of weight lost among participants. There were no significant differences in weight loss between groups based on exercise intensity or exercise duration (39).

Where Jakicic et al. found no differences in weight loss by duration of exercise, Jeffery et al. asked a similar question: does prescribing higher physical activity goals improve weight loss outcome? Researchers recruited 202 overweight men and women and randomly assigned them to a standard behavior therapy group with an energy expenditure component equaling 1000 kilocalories per week, or an energy expenditure only group equaling 2500 kilocalories per week. Results showed that higher physical activity goals resulted in significantly greater weight loss at 12 and 18 months. Mean weight losses maintained at 12
and 18 months in the high physical activity group were 8.5 and 6.7 kilograms, respectively. In the behavioral and lower physical activity group mean weight loss maintained at 12 and 18 months was 6.1 and 4.1 kilograms, respectively. Both groups showed a trend of weight regain and, as in other studies, there was a large variability within groups in how much weight was lost (40).

**Pharmacotherapies**

Several drug therapies are approved for weight loss. One drug called orlistat works to inhibit dietary fat absorption. In a study by Davidson et al. the effectiveness of a reduced kilocalorie diet with orlistat on weight control was assessed in obese adults. The study length was two years, which allowed researchers to assess orlistat's potential both to lead to weight loss and prevent weight regain. After a four week lead-in period, 892 subjects were randomly assigned to a placebo group or the orlistat group. After one year, those left in the study were re-randomized to a placebo group or one of two orlistat groups that differed in dosage. Weight change was the main outcome variable (41).

The results indicated that the orlistat group lost significantly more weight at the one year mark than did the placebo group. Also, at the two year mark, subjects receiving the higher dose of orlistat had the least amount of weight regain. All groups tended toward weight regain from year one to year two. It is of note that of all the subjects randomized for this trial, less than 50% completed the trial (41).

Sibutramine is another common weight loss drug. Unlike orlistat, sibutramine was developed as an anti-depressant until it was found to have weight-reducing effects. James et al. sought to test the efficacy of sibutramine on
weight maintenance after weight loss. This trial had 605 obese individuals first complete a weight loss program that included sibutramine and a reduced kilocalorie diet for six months. At six months, remaining participants were randomized to a sibutramine group or a placebo group and were followed for an additional 18 months. Participants taking sibutramine regained less weight than the control group from month six to month 24, but a pattern of weight regain was noted for both groups. Of the 605 participants originally enrolled, 261 completed the full 24 months (42).

Surgical Treatments

Surgical treatments for obesity are generally divided into cosmetic surgery and non-cosmetic surgery. Cosmetic surgery for weight loss includes liposuction, while non-cosmetic surgery includes bariatric surgeries of differing procedures. Klein et al. researched the effect of liposuction on indicators of metabolic disease often associated with obesity. Specifically this study looked at waist circumference, blood pressure, plasma glucose, plasma insulin, plasma lipoproteins and triglycerides in 15 obese women before and 10-12 weeks after liposuction surgery. From pre- to post-surgery, women lost a significant amount of weight and fat mass. Interestingly, not a single disease risk blood parameter changed significantly during the trial, suggesting that it is not overt fat loss that reduces disease risk (43).

Bariatric surgery has gained popularity in recent times as a treatment for weight loss in the morbidly obese. The safety of bariatric surgery has been shown for the perioperative period (30 days after surgery) (44). Christou et al. sought to investigate the long-term morbidity and mortality in morbidly obese individuals
that opted for bariatric surgery. To do this the researchers employed a cohort study design in which each bariatric surgery patient was matched with several controls that did not have bariatric surgery. 1,035 patients formed the treatment group while 5,746 patients formed the control group. Cohorts were followed for a maximum of five years from inception. At the end of the trial, bariatric surgery resulted in an average 67% reduction in excess weight compared with the control cohort. Also, the benefits of surgery were shown by an 87% relative risk reduction in death with surgery compared to non-surgical controls. Unfortunately, bariatric surgery did result in higher incidences of digestive disorders compared with controls (45).

**Peanuts/Tree Nuts**

*Disease Risk*

Peanut and tree nut consumption is associated with reduced risk of several diseases. Evidence of reduced disease risk with nut and legume consumption is supported by epidemiological studies as well as by experimental studies. Peanut and tree nut consumption has been linked to reduced incidence of cardiovascular disease (46), type 2 diabetes (47, 48), obesity (49), cholecystectomy (50), gallstone disease (51), and colorectal cancer (52). This section will briefly review relevant evidence of the benefits of peanut and tree nut consumption.

Li et al. studied the association between nut consumption and cardiovascular disease risk in type 2 diabetic women. In this prospective cohort study 6309 type 2 diabetic women were given food frequency questionnaires every 2-4 years between 1980 and 2002. With 54,656 person years of observation the researchers were able to conclude that nut consumption on the order of five
servings per week was associated with a relative risk of cardiovascular disease equal to 0.56 (46).

Jiang et al. showed an association between frequent nut and peanut butter consumers and reduced incidence of type 2 diabetes in women. Data from 83,818 women that took part in the Nurses' Health Study were analyzed for frequency of nut and peanut butter consumption. Findings indicated that the highest category of nut consumption (≥ 5 servings/week) was associated with a relative risk for type 2 diabetes of 0.73 (47).

Similarly, Villegas et al. looked at type 2 diabetes incidence of subjects enrolled in the Shanghai Women's Health Study. This prospective cohort study followed 64,227 women for an average of 4.6 years. Women had no history of cardiovascular disease, type 2 diabetes, or cancer at the time of recruitment. Researchers gave participants validated food frequency questionnaires, allowing researchers to assess different levels of legume intake. Results suggested that the relative risk of type 2 diabetes in the highest quintile of legume consumption was 0.51 compared with the lowest quintile of consumption (48).

Nut consumption has been linked to reduced incidence of cholecystectomy in a prospective cohort study by Tsai et al. Looking at data from the Nurses' Health Study the researchers followed 80,718 women from 1980 through 2000. Questionnaires assessing nut consumption were mailed to participants every two years. 1,393,256 person years of follow-up suggested that ≥ 5 ounces of nuts consumed per week was associated with a relative risk of cholecystectomy of 0.75 compared with women reporting rarely consuming nuts. Both peanuts and other nuts considered separately were associated with reduced risk of
cholecystectomy (50).

Tsai et al. evaluated the association between nut consumption and gallbladder disease using data from the Health Professional Follow-up Study. Nut consumption was evaluated using food frequency questionnaires beginning in 1986. 42,823 men completed the study, yielding 457,305 person years of observation. Results showed a relative risk of gallbladder disease equal to 0.70 in men reporting ≥ 5 ounces of nuts consumed weekly compared with men who consumed nuts rarely (51).

Epidemiological evidence suggests an association between peanut consumption and colorectal cancer. Yeh et al. studied 23,943 Taiwanese men and women in a prospective cohort study. Food frequency questionnaires were completed on a weekly basis. Interestingly, peanut consumption was associated with reduced risk of colorectal cancer in women but not men. The relative risk of colorectal cancer for men and women consuming peanuts were 0.73 and 0.42, respectively. These relative risks were highly variable due to the small number of cancer diagnoses in this cohort. Unfortunately, the food frequency assessment techniques used in this study did not allow researchers to determine an amount of peanuts associated with reduced risk (52).

Given the benefits of nut consumption suggested by epidemiological studies, several intervention studies have been done to assess the specific effects of nut consumption on several markers of disease risk. Nut consumption seems to have a positive effect on biomarkers of cardiovascular disease risk (53-58), risk markers related to type 2 diabetes (59, 60), and anti-oxidant status (61).

Sheridan et al. studied the health benefits of pistachios in a randomized
crossover trial. 15 subjects with hypercholesterolemia participated in this four week trial where 15% of the participants estimated energy needs were taken as pistachios. Results suggested improvements in several indices of risk including TC/HDL-C, LDL-C/HDL-C, and HDL-C. The researchers concluded that consuming 15% of calories (2-3 ounces in this study) as pistachios may reduce risk of coronary disease (58).

Studying macadamia nuts, Garg et al. used very similar procedures to that of Sheridan et al. Macadamia nuts replaced 15% of estimated energy requirements in the 17 hypercholesterolemic male subjects that participated in this trial. Researchers noted significant reductions in plasma markers of inflammation (leukotriene, LTB4) and markers of oxidative stress (8-isoprostane). As high levels of these factors are associated with increased incidence of coronary artery disease, the researchers concluded that although macadamia nut consumption led to increased total fat intake, the risk of coronary artery disease was lessened (55).

In an attempt to functionalize the lipid-lowering effects reported with nut consumption, Olmedilla-Alonso et al. studied the ability of walnuts to reduce serum lipids when eaten with a standardized meat product. This randomized crossover design had 25 subjects consume a meat product, fortified with walnut powder or not, five times per week for five weeks. Blood was measured periodically for indicators of disease risk. The meat product fortified with walnut powder had twice the kilocalorie density of the regular meat product. Despite this the walnut group saw significant decreases in total cholesterol, LDL cholesterol, and body weight (57).
Griel et al. looked at the effectiveness of macadamia nuts to change serum lipid levels. In this randomized crossover controlled-feeding study, 25 subjects consumed what the researchers termed an average American diet or a macadamia nut rich diet. The two diets were matched in their macronutrient composition, which had the macadamia nut group consuming 1.5 ounces of nuts daily. After a five week diet period, researchers noted a significant decrease in both total cholesterol and LDL cholesterol in the macadamia nut group (56).

A study by Almario et al. investigated the effects of walnuts on plasma fatty acids and lipoproteins in combined hyperlipidemia. There were four sequential diet groups in this study: a habitual diet, a habitual diet plus walnuts, a low fat diet, and a low fat diet plus walnuts. The habitual diet was followed for four weeks whereas other interventions were followed for six weeks. Each walnut intervention had subjects consuming about two ounces per day. Results of this study showed that despite increasing energy intake, walnut consumption did not lead to weight gain. A significant decrease in total cholesterol and LDL cholesterol was seen only in the low fat diet plus walnuts group (53).

Alper et al. sought to characterize the effects of peanut consumption on biomarkers associated with cardiovascular disease risk. This study employed a crossover design with three groups: a free-feeding group, a peanuts added group, and a peanuts substituted group. Each group was provided 500 kilocalories of peanuts to consume daily. The free-feeding group was told simply to eat the peanuts in any manner they wished. The peanuts added group were prescribed an isocaloric diet to which 50% of dietary fat energy was added. The peanuts substituted group reduced their fat kilocalories by 50% and had that energy
replaced by an equivalent amount in peanuts. Results showed a decrease in serum triglycerides in all treatment groups, however the free-feeding group triglycerides rebounded to baseline values after eight weeks. The researchers concluded that peanut consumption was helpful in reducing risk of cardiovascular disease (54).

Nut consumption has also been linked to positive changes in risk factors associated with type 2 diabetes. Johnston and Buller studied the independent effects of vinegar and peanuts on post-prandial glycemia. With a randomized crossover design, 11 healthy subjects consumed a high or low glycemic meal with one of three complementary conditions: control, vinegar, or peanuts. The researchers found that both the vinegar and peanut conditions reduced post-prandial glycemia after the high glycemic load meal. Interestingly, vinegar and peanut conditions were also found to result in moderately decreased energy consumption in the remainder of the day after the high glycemic load meal. This decreased energy consumption averaged 200 to 275 kilocalories per day (60).

In a study by Jenkins et al. the effect of almond consumption on insulin secretion and action was tested. In this randomized crossover trial 27 hyperlipidemic men and women consumed an isocaloric meal consisting of one of three conditions: almonds, almonds and a low fat muffin, or a low fat muffin. Each condition provided 423 kilocalories and was followed for one month. The only difference found in this trial was a significant decrease in C-peptide urinary excretion in the almond and almond plus muffin groups compared with the muffin group. As C-peptide is a marker of 24-hour insulin secretion, the researchers concluded that almonds have positive effects on insulin secretion that could
explain reduced coronary heart disease risk (59).

Almonds have also been shown to raise antioxidant status in a study by Jenkins et al. This study showed a decrease in two markers of lipid peroxidation with 423 kilocalories of almond consumption daily for one month compared with no almond consumption. Decreases in malondialdehyde and isoprostane demonstrated the antioxidant benefits of almond consumption and suggested another mechanism by which almonds may reduce coronary heart disease risk (61).

Kris-Etherton et al. studied the cardiovascular disease risk profile of an average American diet compared with that of four different cholesterol lowering diets: an American Heart Association/National Cholesterol Education Program Step II diet, a diet high in monounsaturated fatty acids (MUFA) from olive oil, a high MUFA diet from peanut oil, and a high MUFA diet from peanuts and peanut butter. This randomized crossover study found that MUFA diet groups led to an 11% decrease in total cholesterol and a 14% decrease in LDL cholesterol compared with the American diet group. These decreases were similar to the change in the Step II diet group. Interestingly, triglyceride levels decreased by 13% in the MUFA diet groups, but increased 11% in the Step II diet group compared with the American diet group. The MUFA diet groups led to larger decreases in cardiovascular disease risk than did the Step II diet. Specifically, the peanut group had a 21% reduced risk of cardiovascular disease (62).

**BMI**

Many epidemiological studies have linked nut consumption with reduced rates of obesity. Fraser et al. found a statistically significant negative association
between consumption of nuts and BMI, with higher nut consumers being less obese (63). This finding has been replicated with the work of Hu et al. studying data from the Nurses' Health Study and Sabate et al. and Griel et al. using data from the Continuing Survey of Food Intakes by Individuals (64-66).

Bes-Rastrollo et al. looked at the association of nut consumption to weight change and obesity risk with data from the Nurses' Health Study II. 51,188 women were followed from 1991 through 1998. Participants had no cardiovascular disease, type 2 diabetes or cancer. Dietary data was assessed with food frequency questionnaires, while weight and height were given by self-report. Results showed that women reporting nut consumption ≥ 2 times per week gained significantly less weight than women reporting consuming nuts rarely. Further, the association was significant when nut consumption was subdivided into peanuts or tree nuts. Also, results were similar for all BMI weight groups (normal, overweight, obese). Finally, nut consumption ≥ 2 times per week was associated with a relative risk of obesity equal to 0.71 (49).

*Body Weight*

While there is much evidence that peanuts and tree nuts are associated with lower rates of obesity, experimental evidence is lacking. Controlled trials of nut consumption typically do not have body weight as a primary end point. The following is a summary of several trials that have studied the effects of nut consumption on energy balance.

In one trial, Alper and Mattes had 15 healthy, normal weight individuals ingest on average approximately 500 kilocalories of peanuts daily for a total of eight weeks. Without any compensation effects, researchers calculated that this
additional kilocalorie load would result in a weight gain of 3.6 kilograms. The average measured weight change was only one kilogram, corresponding to 28% of predicted weight change (67).

In a similar study, Sabate et al. provided an additional 12% of estimated energy needs as walnuts to 90 healthy subjects for a six month duration. Weight gain was predicted to equal 5.3 kilograms without compensation. However, a weight gain of only 0.4 kilograms was observed. This corresponds to 7% of predicted weight increase (68).

Two studies of weight change have used almonds as the test food. Fraser et al. tested the effect of 15% of energy from almonds on weight change in a free-feeding study. Here the researchers recruited 81 male and female participants to a randomized crossover study lasting one year. For one six-month period, subjects were simply observed, while the second six-month period had subjects incorporating almonds into their diet however they wished. The researchers found no significant weight change in the group when almonds were consumed. The predicted weight gain without compensation was 6.4 kilograms. Actual weight gain was 0.65 kilograms for men and 0.14 kilograms for women, meaning weight change was 2-10% of predicted (69).

Hollis and Mattes performed the most recent trial of weight change with almond supplementation. Here, 20 overweight, healthy, adult women participated in a randomized crossover design. Experimental periods were ten weeks in length with a three week washout between periods. Subjects were supplemented with about 350 kilocalories of almonds daily. The expected weight gain without compensation was calculated to equal 3.4 kilograms, however no weight change
was observed (70).

Only one clinical trial has shown an increase in weight with nut supplementation. Lovejoy et al. had 20 normal weight adults consume 100 grams of almonds daily for 4 weeks. This corresponds to about 575 kilocalories daily. Almonds were provided whole or in a variety of pastries. While subjects were instructed to reduce kilocalories to maintain a steady state, it was found that men gained 0.9 kilograms and women gained 0.3 kilograms (71).

Weight loss

As epidemiological studies show an inverse association between weight and nut consumption, and as most experimental studies of nut supplementation do not result in weight gain, the usefulness of nuts as part of a weight loss regimen has been investigated. Wien et al. randomized 65 participants into two groups. One group consumed a formula-based diet enriched with almonds (~50% total of kilocalories), while the other group was able to self-select complex carbohydrates with supplemental safflower oil. Both diets provided about 1000 kilocalories daily and were matched for protein (29%), cholesterol, and saturated fat (3%). The diet intervention was 22 weeks in length. At week five participants were advised to begin a walking program consisting of 20-30 minute sessions, 3-5 sessions per week. Results showed that the group consuming the formula-based almond supplemented diet had more favorable reductions in weight (-18% versus -11%) and fat mass (-30% versus -20%) than did the complex carbohydrate group (72).

Another trial by Pelkman et al. randomized participants into either a moderate-fat diet (fat providing 33% of energy) that purposefully included
peanuts, or a low-fat diet (fat providing 16% of energy). Both diets were designed to produce one kilogram per week weight loss. After six weeks, diets were adjusted to maintain body weight for another four weeks. Both diets produced weight loss at the six week mark. The moderate-fat diet group lost an average of 7.2 kilograms while the low-fat diet group lost an average of 6.5 kilograms. At the end of the weight maintenance phase, both diet groups had lost about eight kilograms. Of note, the moderate-fat diet condition led to more favorable changes in lipid cardiovascular disease risk markers, including lower triglycerides and higher HDL than did the low-fat diet (73).

In a study by McManus et al. subjects were placed in either a moderate-fat diet group (35% of energy from fat) including nuts or a low-fat diet group (20% of energy from fat). This trial lasted for 18 months. Results showed that the moderate fat diet group lost 4.1 kilograms versus 2.9 kilograms in the low-fat diet group. Furthermore, attrition was 46% in the moderate-fat group versus 80% in the low-fat group (74).

Taken together, these studies show that addition of nuts to a diet regimen do not compromise the effects of the diet. In fact, in some cases, diets including nut supplementation have shown superior effects for weight loss, lipid cardiovascular disease risk markers, and attrition.

Possible mechanisms of action for weight change

As previously mentioned, nut consumption has been associated with reduced rates of obesity in epidemiological studies. This phenomenon has been attributed to three main factors; nuts are highly satiating, nut consumption increases resting energy expenditure, and fats in nuts have a limited
Bioaccessibility (75).

Consumption of fixed energy loads as nuts leads to reductions in self-reported hunger on questionnaires. In a study by Kirkmeyer et al., the primary determinant of satiety was found to be total food energy rather than volume, weight, macronutrient composition or sensory attributes (76). Also, similar satiety scores among peanut, olive, and safflower oils did not show significant differences in a study by Iyer et al., suggesting that fatty acid saturation is not a primary satiety factor (77).

Satiety can also be assessed by measuring dietary compensation, which is the spontaneous reduction of energy intake subsequent to nut consumption. By this measure, studies have reported compensation effects ranging from 54-100% of the kilocalories provided by the nuts (53, 67-71, 76). The lack of an independent effect for fatty acid saturation is further supported by the fact that walnuts, peanuts, and almonds exert comparable dietary compensation effects, while they differ markedly in proportions of poly and monounsaturated fats (78). Food form is a factor in dietary compensation where nut oils and butters have less of an effect than do equal kilocalorie loads of whole nuts (77).

Several studies have shown a limited fat bioaccessibility with nut consumption. One trial had ten participants consuming 95% of energy from fat as whole peanuts, peanut butter, or peanut oil with a vegetarian diet for six days. Results showed that 17.8% of the lipids were lost in feces with the whole peanut load compared with 7.0% and 4.5% with the peanut butter and peanut oil, respectively (79). Another study compared the fecal fat excretion of 63 adults consuming a customary diet containing 70 grams per day of whole peanuts,
peanut butter, peanut flour, or peanut oil. Results showed that fecal fat excretion was significantly higher with whole peanuts than with other groups. The energy lost as fecal fat in the whole peanut group was found to equal 12% of the peanut lipid (80).

Nuts also show a dose response in fecal fat excretion. One study by Haddad et al. had six individuals increase dietary fat from 30% to 43% of energy with the addition of pecans for four weeks. Results showed an increase in fecal fat excretion from 2.9% to 8.3% (81). Similarly, a dose response in fecal fat excretion was shown with almonds by Ellis et al. In this study, participants were progressively provided with 100, 150, and 200 grams per day of almonds. During this time, an increase in fecal fat content was noted from 3.5% to 9.9% (82).

Another study provided a 420 kilocalorie supplement to the diet of 27 hyperlipidemic subjects for three one-month periods. The supplement contained 50-100 grams of almonds, one-half the amount, or no almonds. Results showed fecal energy excretion equal to 60 kilocalories in the highest level of almond supplementation compared with baseline. The middle level of almond supplementation energy excretion was measured to be 27 kilocalories above baseline (83).

Mandalari et al. sought to understand the role of almond cell walls in the bioavailability of almond nutrients during the digestive process. To do this, the researchers put four different almond types through a simulated digestive process in vitro. The four different almond types were natural almonds, blanched almonds, finely ground almonds, and defatted finely ground almonds. Results showed that finely ground almonds had the highest bioavailability of fat (39%),
whereas the natural almonds yielded a more moderate (10%) fat bioavailability. This indicates that the degree to which the almond cell is broken down is a large determinant of lipid bioavailability (84). On the same topic, Cassady et al. had participants chew almonds for 10, 25, or 40 chews in a three arm randomized crossover trial. Fecal fat was measured as an outcome variable. The researchers found, as one might expect, that fewer chews were associated with higher fecal fat measures, likely indicating a lower energy bioaccessibility with less chewing (75).

Chronic consumption of peanuts has been associated with increased resting energy expenditure. One study by Alper and Mattes found that over 19 weeks of 500 kilocalorie per day peanut supplementation, resting energy expenditure was increased 11% (67). One study has been done examining the effects of almond supplementation on resting energy expenditure. Hollis et al. found an approximate 50 kilocalorie per day increase in resting energy expenditure with almond supplementation, however, this finding did not reach statistical significance due to lack of statistical power (70).

Though not studying nuts in particular, van Marken et al. looked at the effect of dietary fat composition on energy metabolism. Researchers noted that polyunsaturated fatty acids are oxidized more rapidly in the body than are saturated fatty acids. Knowing this, the researchers designed a study to test changes in resting metabolic rate (RMR) and diet-induced thermogenesis (DIT) for a diet with a high polyunsaturated-to-saturated fat ratio versus a diet with a low polyunsaturated-to-saturated fat ratio. As expected, when consuming a provided diet with a high ratio of polyunsaturated-to-saturated fats, both RMR
and DIT were higher than when consuming a low-ratioed polyunsaturated-to-saturated fat diet. The researchers concluded that dietary fat composition could have implications in the treatment of obesity (85).

**Conclusion**

The United States population currently is experiencing high rates of obesity. Obesity is not a benign condition. Rather, it is a health condition associated with increased risks of several metabolic diseases and death. While there are currently several treatments for obesity, all have flaws and drawbacks that limit accessibility and success of treatment.

Nut consumption on a regular basis is associated with reduced rates of obesity as well as reduced rates of comorbidities of obesity. Researchers have suggested that the inverse relationship between nut consumption and weight is due to three factors: nut consumption lends to feelings of fullness; nut consumption increases resting metabolic rate; fats in nuts have a limited bioavailability due to incomplete breakdown of cell walls with nut consumption. While there is much evidence that regular consumption of nuts does not increase weight, there is little experimental evidence to suggest the use of nuts in the treatment of obesity.
Chapter 3

Materials and Methods

Subjects

28 overweight (25-29.9 kg/m²) and obese (≥ 30 kg/m²) participants (age 20-75 years) were recruited from the Phoenix area for the study. Those who exercise vigorously more than twice per week were excluded from participation. Further exclusion criteria included a known dairy/nut allergy or intolerance, a recent history of dieting or weight change (±5 kg), an unresolved medical condition, lack of a reliable internet connection, and/or the use of prescription drugs known to influence eating behavior or weight. Pregnant women, recently pregnant women (six months post-postpartum or less), and women planning to become pregnant were also excluded.

Sample Size

To detect a three kilogram difference, assuming the standard deviation for body weight is 5.5 kilograms, power analysis calculations indicated that 108 subjects were needed to detect a treatment difference between groups. An additional 18 subjects were added to account for a presumed attrition of 20%. Therefore, the recruitment goal was 126 participants to be divided equally into two groups. A small but statistically significant difference in weight loss for individuals consuming one ounce of almonds five days a week compared with the control group was anticipated. The alpha level was set at 0.05. Beta error level was set at 0.2, giving a power of 80%. This study was approved by the Institutional Review Board of Arizona State University.
Study Design

This was a randomized, parallel arm intervention lasting eight weeks. Participants met with investigators prior to the start of the intervention and provided written consent. In addition, subjects completed a health history questionnaire, filled out demographic and validated physical activity questionnaires (86), and received instructions regarding completion of an automated self-administered 24-hour dietary recall. At this point subjects were randomly assigned to either the almond (NUT) or control (CON) group. Randomization was based on age, gender, body weight, and body mass index. Body composition was assessed via bio-electrical impedance. Participants received a four-week supply of their respective food product (1 oz. almonds or 2 oz. cheese sticks) and were instructed to eat one serving daily on weekdays 30 minutes before their evening meal. A comparison of the nutrient composition of the intervention foods is shown in Table 1.

Dietary intake was assessed with automated self-administered 24-hour dietary recalls (ASA24, National Cancer Institute) on two consecutive days at pre-trial and weeks one, four, and eight. Once enrolled, participants received a personalized calendar on which researchers had marked important study dates. At weeks one, four, and eight participants came to the test site to pick up their respective food product, to complete validated physical activity questionnaires (86), and to undergo anthropometric measurements. Self-reported satiety scores were recorded via email at study weeks one, four, and eight. Researchers prompted participants to answer the following question: "In general, how hungry/full were you yesterday evening?" Participants were encouraged to use a
validated, standardized satiety scale found on their personalized calendars as a reference when reporting satiety scores (Figure 1) (87). At study weeks four and eight subjects were given a $10 gift card (2 x $10 = $20).

Data Compilation

Data analyses for the present study were based on the automated self-administered 24-hour recalls and the self-reported satiety scores. These data were collected on two consecutive days and then summed and averaged for each study week (0, 4, and 8). In instances where participants reported one of two consecutive days, said value was taken to represent both data collection days. Failure to report satiety or 24-hour recall data on both of two consecutive data collection days for pre-trial or any study week eliminated said participant from data analysis.

Statistical Analysis

Data analysis was completed using the Statistical Package for Social Science (SPSS) version 18 for Windows (2009, Chicago, IL). Values are reported as mean ± standard error. Variables were tested for normality using the Kolmogorov Smirnov test. Data not normally distributed were log transformed and retested for normality. Nonparametric tests were used to analyze data not normally distributed. A repeated-measures ANOVA was used to test for significant time and time by treatment effects. Significance was set at p ≤ 0.05.
**Table 1.** Nutrient Composition for Daily Intervention Portion of Almonds and Cheese Sticks

<table>
<thead>
<tr>
<th>Food Item</th>
<th>Amount (oz)</th>
<th>Kcal</th>
<th>CHO (g)</th>
<th>Total Fat (g)</th>
<th>Saturated Fat (g)</th>
<th>Protein (g)</th>
<th>Fiber (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almonds</td>
<td>1</td>
<td>163</td>
<td>6.1</td>
<td>14</td>
<td>1</td>
<td>6</td>
<td>3.5</td>
</tr>
<tr>
<td>Cheese Sticks</td>
<td>2</td>
<td>178</td>
<td>14.1</td>
<td>10.2</td>
<td>3.9</td>
<td>7.5</td>
<td>0.9</td>
</tr>
</tbody>
</table>
Figure 1

Sample Satiety Scale

When you are asked by an investigator, select a number that indicates, in general, how you felt just before you ate dinner yesterday. [You may chose a number such as 65, etc..]
Chapter 4

Results

Descriptive Characteristics

Participants were recruited for this study via paper advertisements posted on the Arizona State University Polytechnic Campus, from emails sent out on university email lists and from advertisements in campus publications. A total of 28 participants were enrolled. 13 participants completed satiety scores through study week 8 and 13 participants completed automated 24-hour recalls through study week 8. Of note, participant distribution between treatment groups was different, therefore satiety and 24-hour recall data are not directly comparable. Rates of attrition in the study were 53.6% for both satiety data and 24-hour recall data.

Baseline characteristics between treatment groups are shown in Table 2. There were no statistically significant differences between treatments groups at baseline in reference to their age, BMI, height, weight, body fat percentage, or waist circumference.
Table 2
Baseline Characteristics for Participants Completing 24-Hour Recall Data Through Study Week 8

<table>
<thead>
<tr>
<th></th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>44.6 ± 4.0</td>
<td>54.3 ± 3.9</td>
<td>0.239</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>34.6 ± 2.6</td>
<td>31.3 ± 1.2</td>
<td>0.519</td>
</tr>
<tr>
<td>Height (inches)</td>
<td>65.4 ± 0.9</td>
<td>67.1 ± 2.2</td>
<td>0.383</td>
</tr>
<tr>
<td>Weight (pounds)</td>
<td>210.4 ± 17.2</td>
<td>200.5 ± 8.6</td>
<td>0.768</td>
</tr>
<tr>
<td>Body Fat %</td>
<td>40.4 ± 3.0</td>
<td>39.5 ± 6.1</td>
<td>0.866</td>
</tr>
<tr>
<td>Waist Circumference (inches)</td>
<td>42.1 ± 2.1</td>
<td>41.6 ± 0.5</td>
<td>0.901</td>
</tr>
<tr>
<td>METS/week</td>
<td>28.3 ± 8.8</td>
<td>13.3 ± 6.7</td>
<td>0.072</td>
</tr>
</tbody>
</table>

1. Data are Mean ± Standard Error

Demographics of the study population are shown in Table 3. No Chi-square analysis could be performed on these data as cell frequencies were too small.

Table 3
Baseline Demographics for Participants Completing 24-Hour Recall Data Through Study Week 8

<table>
<thead>
<tr>
<th></th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Men/Women</td>
<td>2/8</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>Caucasian</td>
<td>10</td>
</tr>
<tr>
<td>Smoking</td>
<td>Yes/No</td>
<td>1/9</td>
</tr>
<tr>
<td>Activity Level</td>
<td>Not active</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Active</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Somewhat active</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Very active</td>
<td>0</td>
</tr>
</tbody>
</table>

Dietary Compensation

Mean kilocalories consumed at baseline, and weeks 1, 4, and 8 were
obtained and checked for statistical significance between and within treatment
groups. Values for mean kilocalories consumed are summarized in Table 4.

Table 4
Mean Energy Intake at Baseline and Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Kcals/Day</th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time x Interaction</td>
</tr>
<tr>
<td>Baseline</td>
<td>1780 ± 197</td>
<td>1659 ± 360</td>
<td>0.921</td>
</tr>
<tr>
<td>Week 1</td>
<td>1851 ± 184</td>
<td>1809 ± 335</td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>1732 ± 150</td>
<td>1876 ± 274</td>
<td></td>
</tr>
<tr>
<td>Week 8</td>
<td>1642 ± 148</td>
<td>1610 ± 270</td>
<td></td>
</tr>
</tbody>
</table>

1. Data are Mean ± Standard Error.
2. Unable to test homogeneity of inter-correlations as there are fewer than 2
   nonsingular cell covariance matrices.

Mean kilocalories per kilogram for trial days at baseline, and weeks 1, 4,
and 8 were obtained and checked for statistical significance between and within
treatment groups. Mean kilocalorie per kilogram values are summarized in Table
5.

Table 5
Mean Energy Intake per Kilogram Body Mass at Baseline and
Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Kcals/Kg</th>
<th>Nut (n = 9)</th>
<th>Cheese (n = 3)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time x Interaction</td>
</tr>
<tr>
<td>Baseline</td>
<td>20.3 ± 1.8</td>
<td>18.1 ± 3.1</td>
<td>0.834</td>
</tr>
<tr>
<td>Week 1</td>
<td>20.9 ± 2.3</td>
<td>19.7 ± 4.0</td>
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</tr>
<tr>
<td>Week 4</td>
<td>19.5 ± 1.8</td>
<td>20.9 ± 3.1</td>
<td></td>
</tr>
<tr>
<td>Week 8</td>
<td>18.6 ± 1.5</td>
<td>17.8 ± 2.6</td>
<td></td>
</tr>
</tbody>
</table>

1. Data are Mean ± Standard Error.
2. Unable to test homogeneity of inter-correlations as there are fewer than 2
   nonsingular cell covariance matrices.
Mean dietary fiber, carbohydrate, fat and protein consumed at baseline, and weeks 1, 4, and 8 were obtained and checked for statistical significance both between and within treatment groups. Mean dietary fiber, carbohydrate, fat, and protein values for baseline and study weeks 1, 4, and 8 are summarized in Table 6, 7, 8, and 9, respectively.

Table 6
Mean Fiber Intake at Baseline and Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Fiber/Day (grams)</th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time x Interaction</td>
</tr>
<tr>
<td>Baseline</td>
<td>13.3 ± 1.3</td>
<td>10.6 ± 2.4</td>
<td>0.486</td>
</tr>
<tr>
<td>Week 1</td>
<td>14.4 ± 1.2</td>
<td>14.7 ± 2.1</td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>14.8 ± 1.4</td>
<td>14.9 ± 2.6</td>
<td></td>
</tr>
<tr>
<td>Week 8</td>
<td>13.2 ± 1.1</td>
<td>14.4 ± 2.0</td>
<td></td>
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</table>

1. Data are Mean ± Standard Error.
2. Unable to test homogeneity of inter-correlations as there are fewer than 2 nonsingular cell covariance matrices.

Table 7
Mean Carbohydrate Intake at Baseline and Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Carbohydrate/Day (grams)</th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
<th>P value</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time x Interaction</td>
</tr>
<tr>
<td>Baseline</td>
<td>210.8 ± 27.5</td>
<td>167.1 ± 50.1</td>
<td>0.742</td>
</tr>
<tr>
<td>Week 1</td>
<td>206.0 ± 25.5</td>
<td>179.4 ± 46.5</td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>205.1 ± 22.8</td>
<td>183.8 ± 41.7</td>
<td></td>
</tr>
<tr>
<td>Week 8</td>
<td>184.2 ± 22.1</td>
<td>182.5 ± 40.4</td>
<td></td>
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</tbody>
</table>

1. Data are Mean ± Standard Error.
2. Unable to test homogeneity of inter-correlations as there are fewer than 2 nonsingular cell covariance matrices.
Table 8
Mean Fat Intake at Baseline and Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Fat/Day (grams)</th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time x Interaction</td>
</tr>
<tr>
<td>Baseline</td>
<td>76.0 ± 9.1</td>
<td>78.0 ± 16.5</td>
<td>0.665</td>
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<tr>
<td>Week 1</td>
<td>86.8 ± 11.2</td>
<td>82.5 ± 20.5</td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>74.2 ± 8.1</td>
<td>93.8 ± 14.8</td>
<td></td>
</tr>
<tr>
<td>Week 8</td>
<td>71.4 ± 6.7</td>
<td>64.1 ± 12.2</td>
<td></td>
</tr>
</tbody>
</table>

1. Data are Mean ± Standard Error.
2. Unable to test homogeneity of inter-correlations as there are fewer than 2 nonsingular cell covariance matrices.

Table 9
Mean Protein Intake at Baseline and Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Protein/Day (grams)</th>
<th>Nut (n = 10)</th>
<th>Cheese (n = 3)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Time x Interaction</td>
</tr>
<tr>
<td>Baseline</td>
<td>67.4 ± 9.1</td>
<td>76.3 ± 16.7</td>
<td>0.606</td>
</tr>
<tr>
<td>Week 1</td>
<td>67.8 ± 6.4</td>
<td>71.3 ± 11.7</td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>62.2 ± 3.4</td>
<td>84.7 ± 6.3</td>
<td></td>
</tr>
<tr>
<td>Week 8</td>
<td>69.4 ± 6.2</td>
<td>81.8 ± 11.3</td>
<td></td>
</tr>
</tbody>
</table>

1. Data are Mean ± Standard Error.
2. Unable to test homogeneity of inter-correlations as there are fewer than 2 nonsingular cell covariance matrices.

**Satiety**

Mean satiety scores at weeks 1, 4, and 8 were obtained and checked for statistical significance between and within treatment groups. Mean self-reported satiety scores are summarized in Table 10.
Table 10
Mean Satiety at Study Weeks 1, 4, and 8

<table>
<thead>
<tr>
<th>Satiety Score</th>
<th>Nut (n = 9)</th>
<th>Cheese (n = 4)</th>
<th>P value</th>
<th>Time x Interaction</th>
<th>Time Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Week 1</td>
<td>52.2 ± 5.2</td>
<td>66.6 ± 7.8</td>
<td>0.367</td>
<td>0.997</td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>52.0 ± 7.4</td>
<td>67.5 ± 11.2</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Week 8</td>
<td>59.6 ± 6.8</td>
<td>59.4 ± 10.2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Data are Mean ± Standard Error
2. 0 = Greatest imaginable hunger, 100 = Greatest imaginable fullness
Chapter 5

Discussion

We hypothesized that consumption of one ounce of almonds 30 minutes before the dinner meal on weekdays would result in a statistically significant reduction of daily kilocalories consumed in comparison with a kilocalorie-matched control food. In addition, it was presumed that this effect would be mediated by a greater level of satiety reported before the dinner meal in the NUT group compared with controls. No significant difference between groups regarding total kilocalories consumed was observed in the present study.

However, due to several factors the null hypothesis can neither be accepted nor rejected. This study suffered from an unexpectedly high rate of attrition, defined here as failure to submit either of two consecutive 24-hour dietary recalls at any point during the trial. This left so little data for parameters being studied with the automated self-administered 24-hour recall that it limited the validity of planned statistical analyses. Specifically, in reference to the mixed between-within groups ANOVA, Box's M statistic is used to test the assumption of homogeneity of intercorrelations. For the 24-hour recall dataset, Box's M could not be calculated as there were fewer than two nonsingular cell covariance matrices. This same difficulty applies to all variables collected from the automated self-administered 24-hour recall, including kilocalories per kilogram body mass, fiber, carbohydrate, fat, and protein consumed. Thus, there was simply too little data to prove or disprove the null hypothesis.

As mentioned, it was presumed that any dietary compensation effect seen in the NUT group would be mediated by higher reported satiety. Interpretation of
the satiety results then depended on the dietary compensation results. No statistically significant difference was found between treatment groups regarding self-reported satiety scores. It is unclear if this is due to a lack of dietary compensation effect or a mis-presumption that satiety would mediate dietary compensation. Kirkmeyer et al found energy content to be the primary determinant of a foods impact on hunger (76). It may be that any additional satiety effect provided by the almonds over the cheese sticks was overshadowed by the fact that they were kilocalorie-matched. Alternatively, the lack of statistical significance could simply be a manifestation of type II error, as this study was underpowered.

Satiety as a research concept is a participant self-reported, subjective parameter, most often gathered in person at the time the participant is feeling hungry or full. The present study deviated from this norm by asking participants to recall their satiety level from the day before. Recall of satiety may be influenced by different factors than satiety reported at the time it is felt and thus could potentially lead to misinterpretations. Additionally, this satiety question was posed via email, which could result in less accountability to provide accurate responses on the part of participants.

Initial power calculations were based on detecting a three kilogram difference between treatment groups. To do this, 108 subjects would have needed to complete the trial. However, despite enrollment efforts, 28 participants started the trial and 13 finished. Limitations in recruitment may have been related to limited financial incentives that were offered. Additionally, recruitment through campus flyers, campus email lists, and advertisements in campus publications
may have drawn on too few people.

While the present trial was underpowered from the start, attrition was also much higher than expected. As dietary data were gathered via a relatively new computer interface (i.e. ASA24) it may be that participants found this interface off-putting, though this is somewhat counter to a recent feasibility validation study of a similar, but not identical, web-based 24-hour dietary recall program. Arab et al showed high participant retainment with 92% of subjects completing eight assigned dietary recalls over a two-month period (88). It may be relevant that while ASA24 is based on validated dietary recall methods, it has not been validated itself. Also notable is the higher dropout rate in the control group versus the NUT group. This may have been due, in part, to the choice of control food, as cheese sticks were less shelf stable than the almonds.

The present study was unable to prove or disprove the effectiveness of almond supplementation on dietary compensation. Thus, the effectiveness of almonds pre-loaded before the dinner meal for treating overweight and obesity or preventing weight gain is not known. Researchers can benefit from the deficiencies of the present study in designing future trials investigating the weight loss potential of almond supplementation.
Chapter 6

Conclusion

Obesity reflects a serious individual health condition associated with increased risks of several metabolic diseases and death. The problem is made much worse as the prevalence of obesity is greater than 30% of adults in the U.S. While there are currently several treatments for obesity, all have flaws and drawbacks that limit accessibility and success of treatment.

Nut consumption on a regular basis is associated with reduced rates of obesity as well as reduced rates of comorbidities of obesity. Researchers have suggested that the inverse relationship between nut consumption and weight is due to three factors: nut consumption lends to feelings of fullness; nuts consumption increases resting metabolic rate; fats in nuts have a limited bioavailability due to incomplete breakdown of cell walls with chewing. While there is much evidence that regular consumption of nuts does not increase weight, there is little experimental evidence to suggest the use of nuts in the treatment of obesity.

The present study was not able to prove or disprove the hypothesis that consumption of one ounce of almonds, 30 minutes before the dinner meal, five days a week for eight weeks would result in dietary compensation when compared with controls. Unexpectedly high rates of attrition, together with an underpowered study limited statistical analyses. Researchers can use the deficiencies of the present study to design future trials to address questions as to the effectiveness of almonds for the specific treatment of obesity.
References


22. Rolls BJ, Roe LS, Meengs JS. Larger portion sizes lead to a sustained increase in energy intake over 2 days. J Am Diet Assoc. 2006;106(4):543-549.


71. Lovejoy JC, Most MM, Lefevre M, Greenway FL, Rood JC. Effect of diets enriched in almonds on insulin action and serum lipids in adults with normal


83. Kendall CWC, Jenkins DJA, Marchie A, Ren Y, Ellis PR, Lapsley KG. Energy


Appendix A

Power Calculation
Appendix A

Power Calculation

Power analyses indicate that 108 subjects are needed to detect a difference between treatment groups. We anticipate a small but statistically significant decrease in weight for individuals consuming 1 oz. of almonds (163 kcals) 5 days a week for 8 weeks compared with the control group. To detect a 3 kg difference, assuming that the standard deviation for body weight is 5.5 kg and accounting for a 20% rate of attrition, goal enrollment will be 126 participants or 63 subjects per group. The alpha level will be set at 0.05 and the beta error level is 0.2 resulting in a power of 80%.
Appendix B

Informed Consent Form
ASU NUTRITION: ALMOND/DAIRY TRIAL

INTRODUCTION
The purposes of this form are (1) to provide you with information that may affect your decision as to whether or not to participate in this research study, and (2) to record your consent if you choose to be involved in this study.

RESEARCHERS
Dr. Carol Johnston, Professor and Director of the ASU Nutrition Program, and Marshall Jahns, nutrition masters student, have requested your participation in a research study.

STUDY PURPOSE
The purpose of the research is to examine the effects of daily almond or dairy product consumption on body composition in overweight individuals.

DESCRIPTION OF RESEARCH STUDY
You have indicated to us that you are healthy and not allergic to nuts or dairy products. You have also indicated that you are willing to consume almonds or cheese sticks as required in this study; to adhere to the study diet and activity restrictions; and, to use an online system to enter five random 24-hr dietary recalls. Initially you will come to the test site to complete a brief health history questionnaire to demonstrate the absence of medical conditions or situations that may impact the study. At this visit you will be trained on a computer to enter 24-hr diet information. Your weight and height will be measured and we will measure your waist circumference. The scale that determines your body weight will also provide information regarding your body composition by sending a weak electrical current through your body that cannot be felt. This first meeting will take 1-2 hours. At this visit you will be scheduled for three (3) more appointments at the test site which will take about 30 minutes each. At these visits we will repeat measurements of your weight, waist circumference, and body composition. You will be receiving follow-up phone calls, or emails if preferred, by researchers so any questions can be answered. This study will last about 2 months.

At the start of the study you will be randomly assigned to the almond group or to the cheese group; that is, you will not be able to choose which group you are in. You need to eat the specified amount of almonds (1 oz) or cheese (2 oz) at about ½ hour prior to the evening meal five days per week (Mondays through Fridays). You will be provided with a calendar to keep a record of your consumption of the test foods. All test foods will be provided to you at the start of the study and at week 4. You will be asked to provide automated 24-hr diet data via emails from the National Cancer Institute. The NCI offers this diet analysis program to researchers across the country. We will register you at the
NCI site by subject number and email address. You will receive these emails on 5 occasions during the study. During the 8-week trial, you need to consume your normal diet and maintain your current physical activity schedule. We do not want you to start a new diet or exercise program while you are in the study. If you begin taking new medications during the study, you are to notify the study investigators. About 130 people will participate in this study. This study is funded by the ASU Foundation.

**RISKS**
The experimental food items are commonly consumed foods; yet some participants may be allergic or intolerant to nut and dairy, or to other things that are often manufactured with these items. Individuals will be carefully screened to exclude individuals with these conditions/situations.

**BENEFITS**
This study will provide information regarding the effect of moderate almond consumption and/or dairy products on body composition in overweight individuals. There are no direct benefits to you if you participate in this study.

**NEW INFORMATION**
If the researchers find new information during the study that would reasonably change your decision about participating, then they will provide this information to you.

**CONFIDENTIALITY**
All information obtained in this study is strictly confidential unless law requires the disclosure. The results of this research study may be used in reports, presentations, and publications, but your name or identity will not be revealed. In order to maintain confidentiality of your records, Dr. Johnston will use subject codes on all data collected, maintain a master list separate and secure from all data collected, and limit access to all confidential information to the study investigators.

**WITHDRAWAL PRIVILEGE**
You may withdraw from the study at any time for any reason without penalty or prejudice toward you. Your decision to withdraw would not affect you in any manner.

**COSTS AND PAYMENTS**
You will receive two $10.00 gift certificates to Target for full participation in this study. The first gift card will be received at week 4 and the second will be given at the time of trial completion.

**COMPENSATION FOR ILLNESS AND INJURY**
If you agree to participate in the study, then your consent does not waive any of your legal rights. However, in the event of harm, injury, or illness arising from this study, neither Arizona State University nor the researchers are able to give you any money, insurance coverage, free medical care, or any compensation for such injury. Major injury is not likely but if necessary, a call to 911 will be placed.

**VOLUNTARY CONSENT**
Any questions you have concerning the research study or your participation in the study, before or after your consent, will be answered by Dr. Carol Johnston; 7001 E. Williams
Field Rd., Mesa, AZ 85212; 480-727-1713.

If you have questions about your rights as a subject/participant in this research, or if you feel you have been placed at risk, you can contact the Chair of the Human Subjects Institutional Review Board, through the ASU Office of Research Integrity and Assurance, at 480-965 6788.

This form explains the nature, demands, benefits and any risk of the project. By signing this form you agree knowingly to assume any risks involved. Remember, your participation is voluntary. You may choose not to participate or to withdraw your consent and discontinue participation at any time without penalty or loss of benefit. In signing this consent form, you are not waiving any legal claims, rights, or remedies. A copy of this consent form will be given to you.

Your signature below indicates that you consent to participate in the above study.

__________________________
Subject's Signature

_________________________
Printed Name

__________________________
Date

__________________________
Contact phone number

_________________________
Email (print clearly)

**INVESTIGATOR’S STATEMENT**

"I certify that I have explained to the above individual the nature and purpose, the potential benefits, and possible risks associated with participation in this research study, have answered any questions that have been raised, and have witnessed the above signature. These elements of Informed Consent conform to the Assurance given by Arizona State University to the Office for Human Research Protections to protect the rights of human subjects. I have provided the subject/participant a copy of this signed consent document."

__________________________  ________________________
Signature of Investigator     Date
Appendix C

Telephone Screening Script
Appendix C

Telephone Screening Script

Standardized Phone Script

Date:________________________  Time:________________________

Interviewer:____________________

Thank you for your interest in our study. Before I can enroll you in this study, I will need to ask you a few simple questions that will allow me to determine if you qualify to participate.

Participant

Name:_____________________________________________________________

Contact information:_____________________________________________________________

Gender:  M    F

Weight________________ (weight stable over past 6 mo?)  Y      N
Age___________________

Height___________________  Calculated
BMI_____________________

1. How often do you usually exercise? Days__________________ Minutes

2. What would you consider the intensity of your exercise to be:

<table>
<thead>
<tr>
<th>Mild</th>
<th>Moderate</th>
<th>Intense</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. Are you currently Pregnant/Lactating/Anticipating being pregnant?   Y      N

4. Have you recently been pregnant in the last 6 months?   Y      N

5. Are you currently taking any medications?   Y      N

   If so, what medications do you currently take?
   Medication  Time on Medication


57
6. Are you currently being treated for any medical condition?  Y  N
7. Are you currently being treated for a chronic disease or physical condition?  Y  N
8. Do you have any known allergies or intolerances to any nuts/dairy products?  Y  N
9. This study is expected to last 8 weeks in length, is there any reason that may prevent you from completing this study which entails consuming one serving of nuts or a dairy product daily?

10. Are you willing to access the National Cancer Institute’s web page and complete diet entries on 5 days?  Y  N

Notes__________________________________________________________
Appendix D

Exclusion List of Medications that Influence Weight
Appendix D

Exclusion List of Medications that Influence Weight

<table>
<thead>
<tr>
<th>Used in the Treatment of:</th>
<th>Generic Name</th>
<th>Brand Names</th>
</tr>
</thead>
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<tr>
<td>Psychiatric Disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Olanzapine</td>
<td>Zyprexa</td>
<td></td>
</tr>
<tr>
<td>12. Clozapine</td>
<td>Clozaril</td>
<td></td>
</tr>
<tr>
<td>13. Lithium</td>
<td>Eskalith,</td>
<td></td>
</tr>
<tr>
<td>14. Ziprasidone</td>
<td>Lithobid, Geodon</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>Elavil</td>
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</tr>
<tr>
<td>Paroxetine</td>
<td>Paxil</td>
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</tr>
<tr>
<td>Phenelzine</td>
<td>Nardil</td>
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<td>Tranylcypromine</td>
<td>Parnate</td>
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<tr>
<td>Nefazadone</td>
<td>Serzone</td>
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<tr>
<td>Bupropion</td>
<td>Wellbutrin SR</td>
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<tr>
<td>Epilepsy</td>
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</tr>
<tr>
<td>Valproate</td>
<td>Depakene</td>
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<td>Gabapentin</td>
<td>Neurontin</td>
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<td>Carbamazepine</td>
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<td>Topiramate</td>
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<td>Corticosteroids</td>
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<td>Diabetes</td>
<td>Insulin</td>
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<td></td>
<td>Sulfonylureas</td>
<td>Diabeta</td>
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<td>Thiazolidinediones</td>
<td>Avandia, Actos</td>
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<td></td>
<td>Biguanide metformin</td>
<td>Glucophage</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Propranolol</td>
<td>Inderal</td>
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Appendix E

Sample Participant Calendar
## Appendix E

**Sample Participant Calendar**

### May 2010

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<th></th>
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<th>2</th>
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<th>5 24 h RECALL</th>
<th>6 24 h RECALL</th>
<th>7 24 h RECALL</th>
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<tr>
<td>9</td>
<td>10</td>
<td>test food consumed ½ h prior to dinner</td>
<td>test food consumed ½ h prior to dinner</td>
<td>test food consumed ½ h prior to dinner</td>
<td>test food consumed ½ h prior to dinner</td>
<td>test food consumed ½ h prior to dinner</td>
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<tr>
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</table>

### June 2010

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th>1</th>
<th>2 RETURN to ASU</th>
<th>3 24 h RECALL</th>
<th>4 24 h RECALL</th>
<th>5</th>
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<td>16</td>
<td>17</td>
<td>18</td>
<td>19</td>
<td></td>
</tr>
</tbody>
</table>
When you are asked by an investigator, select a number that indicates, in general, how you felt just before you ate dinner yesterday. [You may chose a number such as 65, etc..]

24-Hour Recall Instructions
ASA24 website accessed by going to https://asa24beta.westat.com

15. Click on the “Begin ASA24” button. It may take a minute or two for the program to download…be patient 😊

16. This message may come up…If a new window fails to open after clicking on ‘Begin ASA24’, please check to see that pop-ups are allowed for this site. To unblock pop-ups look under the tool bar for the pop-up blocked bar. Right click on the pop-up blocked bar and choose the option to allow pop-ups.

17. Username: Password:

18. You may wish to view the tutorial including the detailed tutorials.
You will need audio for this. It will take less than 10 minutes and is very helpful.
Appendix F

Health History/Questionnaire
Appendix F

Health History/Questionnaire

ID#___________________

1. Gender:  M    F

2. Age:  __________

3. Have you lost or gained more than 5 lbs in the last 12 months? 
   Yes    No
   If yes, how much lost or gained? ___________
   How long ago? ___________

4. Ethnicity: (please circle)  Native American     African-American
   Caucasian     Hispanic     Asian     Other

5. Do you smoke? No, never ___________
   Yes _______     # Cigarettes per day = ___________
   I used to, but I quit _______ months/years (circle) ago

6. Do you take any medications regularly?  Yes    No

   If yes, list type and date you started:
   Medication                        Date
   ___________________________________________________
   ___________________________________________________
   ___________________________________________________
   ___________________________________________________

7. Do you currently take supplements (vitamins, minerals, herbs, etc.) ?
   Yes    No

   If yes, list type and frequency:
   Supplement  Dosage  Frequency
   ___________________________________________________
   ___________________________________________________

8. Do you currently take supplements (vitamins, minerals, herbs, etc.) ?
   Yes    No

   If yes, list type and frequency:
   Supplement  Dosage  Frequency
   ___________________________________________________
9. Have you ever been hospitalized? ______
If yes, please explain?

10. Please ANSWER (YES/NO) if you currently have or if you have ever been diagnosed with any of the following diseases or symptoms:

<table>
<thead>
<tr>
<th>Disease</th>
<th>YES</th>
<th>NO</th>
<th>YES</th>
<th>NO</th>
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<tr>
<td>Leg or Ankle Swelling</td>
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<td>Any Heart Problems</td>
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<td>Coughing of Blood</td>
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<td>Feeling Faint or Dizzy</td>
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<td>Lung Disease</td>
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<td>Liver Disease</td>
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<td>Kidney Disease</td>
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<td>Thyroid Disease</td>
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<td>Anemia</td>
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<td>Hormone Imbalances</td>
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<td>Emotional Problems</td>
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Please elaborate on any condition listed above.

11. How would you rate your lifestyle?
Not active ___________ Active ____________
Somewhat active ___________ Very Active ____________

12. Please circle the total time you spend in each category for an average week.

**Light activities** such as:
Slow walking, golf, slow cycling, doubles tennis, easy swimming, gardening
Hours per week:  0  1  2  3  4  5  6  7  8  9  10+

**Moderate activities** such as:
Mod. Walking, mod. cycling, singles tennis, mod. swimming, mod. weight lifting
Hours per week:  0  1  2  3  4  5  6  7  8  9  10+

**Vigorous activities** such as:
Fast walking/jogging, fast cycling, court sports, fast swimming, heavy/intense weight lifting
Hours per week:  0  1  2  3  4  5  6  7  8  9  10+

13. How much alcohol do you drink? (average drinks per day)

14. Do you have any food allergies/intolerances?  Yes  No  If yes, explain:

15. Do you follow a special diet? (weight gain/loss, vegetarian, low-fat, etc.)  Yes  No

If yes, explain:

16. How often do you *usually* consume nuts or nut products including peanuts?