AN ABSTRACT OF THE DISSERTATION OF

Sue B. Davidson for the degree of Doctor of Philosophy in Nutrition and Food Management presented on August 23, 1999. Title: Effect on Eating Behavior, Lipids, Lipoproteins and Lipid Peroxidation of a High Monounsaturated Diet in Postmenopausal Women with Type 2 Diabetes.

Abstract approved:

Rosemary C. Wander

The objective of this study was to compare the effects on eating behavior, lipids, lipoproteins, lipid peroxidation, and glycemic control in women with type 2 diabetes of a high-monounsaturated fat diet (HM) compared to a high-carbohydrate diet (HC).

In an outpatient feeding study, ten hypertriglyceridemic postmenopausal type 2 diabetic women alternately for six weeks consumed the HM and HC diets. On the HM diet, 45% of total calories were consumed as carbohydrate and 40% as fat (27% monounsaturated) compared to 55% carbohydrate and 30% fat (10% monounsaturated) in the HC diet. At the beginning and end of each diet phase, total lipids, lipoproteins, lipid peroxidation, and glycemic variables were measured. For 8 days in each diet phase eating pattern frequency, palatability of foods, hunger and

fullness were assessed. At the end of each diet phase, taste testing to determine preference for fat was conducted.

Total cholesterol was significantly decreased on the HC diet. Serum triglyceride, very low density lipoprotein (VLDL) triglyceride and cholesterol, and apolipoproteins A-1 and B were not significantly different on the two diets. When comparing initial to final values, both diets lowered LDL-C; however, the change was greater on the HM diet. Lipid peroxidation variables improved when the HM diet was consumed. Glycemic variables improved on both diets.

No significant differences between total number of eating episodes on the HM and HC diet phases were found. Both diets were rated as highly palatable. Hunger and fullness ratings varied within and between subjects. However, fullness was more commonly experienced than hunger on both HM and the HC diet. Preference for fat was not found at the end of HM or HC diets. However, subjects differed significantly in ratings for liking of foods that were salty, sour, and bitter when compared to nondiabetic women.

Consumption of the HM and HC diets did not result in deterioration of lipid status. The HM diet by virtue of less oxidation of the LDL particle and improvement of glycemic control provides an important advantage over the HC diet. A description of eating behavior of women with type 2 diabetes emerged.

Effect on Eating Behavior, Lipids, Lipoproteins and Lipid Peroxidation of a High Monounsaturated Diet in Postmenopausal Women with Type 2 Diabetes

by

Sue B. Davidson

A DISSERTATION

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APPROVED:

Major Professor, representing Nutrition and Food Management

Head of Department of Nutrition and Food Management

Dean of Graduate School

I understand that my dissertation will become part of the permanent collection of the Oregon State University libraries. My signature below authorizes release of my dissertation to any reader upon request.

Sue B. Davidson, author

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Staff of the Oregon Health Sciences University Clinical Research Center were involved in recipe testing, preparation of study foods, ongoing adjustments of the diet to meet the needs of the patients, and drawing and processing of blood samples. Jonathan Fields provided assistance with data analysis. Dr. Richard Mattes has provided consultation in taste testing methodology and data analysis. Special thanks to Shi-Hua Du for teaching me about assays. Dr. Doug Derryberry, committee member for my minor, provided good advice regarding this project; many thanks are given to Dr. Maret Traber for being willing to serve on my committee under unusual circumstances.

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CONTRIBUTIONS OF AUTHORS

Dr. Rosemary Wander has been involved in the design, the majority of the assays, data analysis, writing of each manuscript, and in guiding my development as a researcher in this field. She exemplifies all of the best qualities of a scientest, mentor, and teacher. Dr. Matt Riddle provided clinical supervision of patients in the study, did the insulin and glucose assays, provided valued review of the manuscripts, and has guided my thinking about diabetes.

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DEDICATION

The patients who participated in this study are a very special group of women with whom I have formed a most unusual bond. I owe the greatest thanks to them. From their bodies, their blood, and their spirits as the study unfolded, I have learned that living with diabetes could be made better if more research studies were conducted specifically for the problems they face in living with food. To A.R., peace and love.

Effect on Eating Behavior, Lipids, Lipoproteins and Lipid Peroxidation of a High Monounsaturated Diet in Postmenopausal Women with Type 2 Diabetes

INTRODUCTION

Nearly 160,000 people with diabetes live in Oregon. Of this number, approximately 140,000 have type 2 diabetes (Center for Population Research and Census, 1991). The Oregon Behavioral Risk Factor Surveillance (BRFS) data suggest that diabetes occurs somewhat more frequently among women (5%) than men (4%) (Oregon Health Division, 1990), a pattern that is also seen nationally with type 2 diabetes occurring in 58.4% of women compared with 47.6% in men (National Diabetes Data Group, 1995). The BRFS data also revealed that between 1982 and 1988, the mortality rates among female Oregonians with diabetes rose from 13.5% to 17.5% (Oregon Health Division, 1990). In Oregon, the most common diagnosis for hospitalizations, beside diabetes, and the most common underlying cause of death in diabetics were diseases of the circulatory system (Oregon Health Division Diabetes Project, 1997).

Scope of the Problem

Diabetes, in these age groups and for women, is not benign. The morbidity and mortality in type 2 diabetes is linked to numerous forms of macrovascular disease such as stroke, hypertension, coronary and peripheral vascular disease. The risk for macrovascular disease is especially significant for women with type 2

diabetes (Gordon, Castelli, Hjortland, Kanel, and Dawber, 1977; National Diabetes Data Group, 1995, p. 251).

Diet therapy is viewed as one of the cornerstones of diabetes treatment, but the diabetic diet is difficult for patients to follow. People with diabetes view the diet as the most challenging and difficult part of their therapy (Lockwood, Frey, Gladish, and Hiss, 1986; Shimikawa, Herrera-Acena, Colditz, Manson, Stamper, and Willett, 1993; Sullivan and Joseph, 1998). There may be factors yet to be explored which account for the difficulty in following the diet in type 2 diabetes. For example, a number of research studies with obese nondiabetic women, a population similar to women with type 2 diabetes, have examined eating behavior such as preferences for particular foods, sensory and hedonic factors such as palatability, awareness of hunger and satiety, temporal patterns related to eating, attitudes about intake of food, or the interactions between these element of eating behavior (Blundell, 1993; Blundell and Macdiarmid, 1997; Caputo and Mattes, 1993; Cotton, Burley, Weststrate and Blundell, 1994; Drewnowski, Kiurth, Holden-Wiltse, Saari, 1992) and have examined these issues using different research methodologies. Such research is also possible with persons with type 2 diabetes but it has not been conducted.

Type 2 diabetes is a heterogeneous state, characterized by varying degrees of impairment of beta cell function and insulin secretion, varying degrees of insulin resistance, and variations in terms of tissue sensitivity to insulin (DeFronzo, 1988;

DeFronzo and Ferrannini, 1991). Metabolic processes that occur because of these defects vary across the natural history of a diabetic's disease, so that, in addition to dietary adjustments for obesity, it seems plausible that diet treatment in type 2 diabetes needs to better match the particular phase of type 2 diabetes. However, such recommendations do not exist and thus, prescribing the most appropriate diet for type 2 diabetes has been difficult.

Finally, people with type 2 diabetes have increased risk, earlier onset, and an accelerated progression of heart disease, the underlying process of which is atherosclerosis. This risk is associated with elevated plasma triglyceride concentrations, abnormal composition of very low density (VLDL) lipoprotein, lowered high density (HDL) lipoprotein concentrations, abnormal apolipoprotein concentrations (apolipoprotein A1 and/or B), oxidative modification of lipoproteins, and/or some combination of these factors (Ginsberg, 1996; Giugliano, Ceriello, Paolisso, 1996; Schwartz, Cayatte, Kelley, Rozek, Sprague, Valente, 1992; Steiner and Lewis, 1996). The therapeutic goals of the diet in type 2 diabetes must also encompass modulation of these factors to reduce risk of cardiovascular morbidity and mortality.

From 1979 till 1993, the American Diabetes Association (ADA) recommended that the diabetic diet should distribute 55-60% of the total calories as carbohydrate, 12-20% of the calories as protein, and 30% or less of the total calories as fat with <10% as saturated fats (SF), <10% as polyunsaturated fatty

acids (PUFA), leaving the remainder for monounsaturated fatty acids (MUFA). These recommendations improved lipid and lipoprotein abnormalities, e.g., elevated low density lipoprotein (LDL) cholesterol (LDL-C) which occurred in type 1 diabetes, but their effect on lipid and lipoprotein abnormalities in type 2 diabetes e.g., increased triglyceride and reduced HDL-C, was questioned. Controversy arose over this recommendation for increased carbohydrate in the diabetic diet because this has been shown to raise triglyceride levels in type 2 diabetics (Coulston, Hollenbeck, Swislocki, Chen and Reaven, 1987; Coulston, Hollenbeck, Swislocki and Reaven 1989; Blades and Garg, 1995). In 1994 (Franz, Horton, Bantle, Beebe, Brunzell, Coulston, Henry, Hoogwerf, and Stacpoole), the ADA recommendations for the type 2 diabetes diet were revised to state that

> "If dietary protein contributes 10-20% of the total caloric content of the diet, then 80-90% of calories remain to be distributed between dietary fat and carbohydrate....The distribution of calories from fat and carbohydrate can vary and can be individualized based on the nutrition assessment and treatment goals." (ADA, 1999, p. S43).

This most recent recommendation leaves decision making over the percent of calories that will come from carbohydrate and fat to the health care provider and/or dietitian. There is evidence, however, that in the current health care system, contact between health care providers and/or dietitians does not occur until insulin treatment is initiated (Arnold, Stephen, Hess and Hiss, 1993).

These three factors in type 2 diabetes - difficulty with the diet, controversy over the most efficacious dietary composition for type 2 diabetes, and concern over a diet composition which can minimize atherosclerotic factors most characteristic of type 2 diabetes - provide continued impetus for research studies which explore a diet for type 2 diabetes. Such studies need to define a diet that will reduce factors which contribute to risk for or progression of atherosclerosis, one that corrects lipid, lipoprotein and glycemic abnormalities, and most importantly, one that will be consumed by persons with type 2 diabetes over the long-term.

Study Objectives.

The objectives of this feeding study were twofold.

1) Describe variations in selected eating behaviors of subjects when ingesting a high carbohydrate (HC) diet compared to a diet enriched with monounsaturated fatty acids (HM). Specific objectives were to

    a) Determine if subjects had a temporal pattern of eating which varied with the two diet phases. The three elements of temporal pattern which were described were time of meals and snacks across 24 hour periods, variations in meal and snack macronutrient composition across 24 hour periods, and ratings of hunger and fullness in relationship to time of day;

b) Determine if there were differences in food preferences for high carbohydrate and high fat calorie foods in the two diet phases;

c) Determine if palatability, e.g., pleasantness, texture, smell, appearance, and richness of meals (composite of all foods), and individual foods, varied in the two diet phases.

2) Test the effect of increased levels of monounsaturated fatty acids in the diabetic diet on lipids, lipoproteins, and apolipoprotein levels, and on measures of lipid peroxidation.

Study Hypotheses.

There are two study hypotheses for this research.

Hypotheses related to study objective 1:

Perceptions of hunger will decrease and palatability and perceptions of fullness will increase while on the HM diet.

Hypotheses related to study objective 2:

Lipid levels (total cholesterol, triglyceride), lipoproteins (VLDL-C, LDL-C, and HDL-C) levels and composition, apolipoproteins A-1 and B, and measures of lipid peroxidation, e.g., lowered levels of plasma and urinary thiobarbituric acid reactive substances (TBARS), lengthened LDL-C lag time, and lower rates of production and final concentrations of conjugated dienes in LDL-cholesterol, will improve while on the HM diet.

REVIEW OF THE LITERATURE

This review of the literature will address the incidence and sequelae of type 2 diabetes, eating behavior, and macronutrient components such as carbohydrate and fat in the diet for type 2 diabetes.

Type 2 Diabetes

Type 2 diabetes, formerly called noninsulin-dependent diabetes (NIDDM), is a heterogeneous state with multiple metabolic and clinical phases as part of its natural history. The person who develops type 2 diabetes usually has one or more of the five independent risk factors present: smoking, hypertension, hyperlipidemia, obesity, and genetic predisposition (Davidson, 1998). It is estimated that between 80-90% of persons who develop type 2 diabetes are obese at the time of diagnosis. As a consequence of increased levels of food intake, insulin secretion is increased to deal with increased food intake. Insulin levels may be normal if beta cells in the pancreas are functioning adequately. Eventually, due to reduced numbers of insulin receptors or other factors, insulin levels rise. Hyperinsulinemia causes down regulation of insulin receptors, but if the beta cells can secrete insulin normally, hyperglycemia may not occur. As time passes, however, the beta cells of the pancreas may not be able to produce as much insulin as is needed to maintain normoglycemia, and moderate hyperglycemia will occur. At the time of diagnosis, it is estimated that hyperglycemia may have been present for as long as 4 to 7 years, although at levels not high enough to produce symptoms (Harris, Klein, Welborn,

Knuiman, 1992). Thus, type 2 diabetes is characterized by a relative rather than an absolute lack of secreted insulin.

Many of the risk factors for diabetes are also risk factors for insulin resistance syndrome, e.g., central obesity, hypertension, dyslipidemias (increased fasting triglycerides, large postprandial triglyceride-rich lipoprotein particles, decreased high-density lipoprotein (HDL) cholesterol concentration), and coronary artery disease (Davidson, 1998). Because this metabolic situation can last for some time, it is not uncommon for persons who are diagnosed with type 2 diabetes to also have one or more macrovascular, microvascular or neuropathic complications. There is a significant association between type 2 diabetes and macrovascular complications such as coronary artery disease; this association is increasingly attributed to insulin resistance (Haffner, 1996). At the present time, the mechanisms by which dyslipidemia such as high concentrations of triglyceride, or low HDL-cholesterol concentrations occur, are unclear (Davidson, 1998). Whatever the mechanism(s) or the relationship between type 2 diabetes and coronary heart disease, a therapeutic diet to improve the metabolic consequences of type 2 diabetes is very important.

A chronic illness such as type 2 diabetes is experienced by an adult as an involuntarily acquired requirement for change in many areas of daily living. It is not uncommon to respond to having type 2 diabetes with denial, anger, frustration, grief and depression (Rubin, Bierman and Toohey, 1993). These responses affect

diabetes self care management. Several surveys confirm that the diabetic diet is the most difficult aspect of living with diabetes (Lockwood et al., 1986; Shimikawa et al., 1993; Sullivan and Joseph, 1998). One way to increase the likelihood of a woman with type 2 following dietary recommendations is to assess, in addition to the impact on family and work, her eating behavior (Glasgow and Eakin, 1996; Schlundt, Rea, Line, and Pichert, 1996; Travis, 1997).

Eating Behavior.

Eating behavior is based on physiological, metabolic, endocrine, neural, and cognitive processes (Friedman, Tordoff, and Ramirez, 1986; Friedman and Mattes, 1993; Meguid, Yang and Gleason, 1996; Read, French and Cunningham, 1994). The interrelationship of these processes provides the basis for motivation to engage in eating behavior (Stricker and Verbalis, 1990; Morley, Gosnell, Kahn, Mitchell, and Levine, 1985). Eating behavior is characterized by eating pattern frequency, preference for particular foods, palatability of particular foods, motivations to eat, ideas or knowledge about eating and food, and awareness and interpretation of sensations of hunger and fullness (Gorman and Allison, 1995; Schlundt, 1995).

Eating pattern frequency.

Eating pattern frequency, sometimes called the "periodicity of eating" or temporal eating pattern, refers to the number of eating episodes that occur within a time period such as 24 hours (Gibney and Wolever, 1997). For example, it is

estimated that British adults eat 6.5 times per day, but as age increases, frequency of eating drops (elderly = 6.02; very elderly = 5.6 times per day) (Gatenby, 1997). There is variability in reported eating pattern frequency due, in part, to lack of a uniform definition within a culture and between cultures regarding what constitutes a "meal" and a "snack". Eating pattern frequency reported in men and women ages 25 - 74 in the NHANES I Epidemiologic Follow up Study ranged from 2 - 7 episodes per day (Kant, 1995). In obese women and men who were on a weight loss regimen, eating episodes ranged from three to seven per day (Debry, Azouaou, Vassilitch, and Mottaz, 1973). One of the most prolific researchers on eating pattern in the United States, John de Castro, never reports the actual number of eating episodes per day in the many studies he has conducted; rather, he reports only the composition (calories, carbohydrate, protein, fat) of all meals and snacks in 24 hour periods, so that it is not possible to determine the eating pattern frequency in any of his published studies. No data has been published regarding the eating pattern frequency of women with type 2 diabetes (de Castro, 1987; de Castro and Elmore, 1988; de Castro and de Castro, 1989; de Castro, 1993; de Castro, 1997).

Preference for and Palatability of Fats.

Preference for a particular food is based on cognitive processes related to knowing, liking and choosing one food over another. Palatability, defined as the degree of pleasantness or liking of sensory characteristics associated with the ingestion of certain foods, is based on taste, smell, color, odor, richness and texture.

Nondiabetic obese women have preference for high levels of fats in foods (Drewnowski, 1992), particularly foods which combine the sensory dimensions of sweetness and creaminess (Drewnowski et al, 1985). It seems intuitive that women with type 2 diabetes who are overweight or obese would also have these same preferences, although there is no research has been carried out to confirm this hypothesis.

Only one study has been identified which explores changes in preferences and ratings of palatability in type 2 diabetics when sweet and fat in the diabetic diet is reduced (Laitenen, Tuorila and Uusitupa, 1991). This study was conducted with recently diagnosed type 2 diabetics, 40-64 years of age (12 women, 19 men), whose body mass indexes ranged from 30.6 - 33.4 kg/m^2 and whose fasting blood glucoses were elevated. Three times (when therapy was initiated, after 6 and after 12 weeks), blood work related to diabetes was obtained and then tasting and rating of samples on a 9-point scale (1 = extremely unpleasant; 9 = extremely pleasant) of juices with varying sucrose levels, sweetened cookies, cheeses, and milk with various fat contents, was completed. Diet instruction was also provided at the time of the visit. The sucrose content of the juices ranged from 0 to 12%. One cookie was made with sorbitol, the other, with sucrose. The fat content within the milk samples ranged from 0% to 3.9%, and of the cheeses, 20% and 40%. The cheeses were presented first, then the juices, milk, and the cookies. However, the various samples within one of these categories were randomized each time the testing was done.

When the patient went home, a questionnaire was completed which assessed frequency of consumption and hedonic responses to 37 frequently eaten fat and sweet Finnish foods. The hedonic response options in the questionnaire were on a 5-point scale (1 = do not like at all; 5 = "like very much"; and an option for "I cannot say" for a food that was unfamiliar). Frequency of consumption was also rated on a 5-point scale (1 = once a month of less" to "daily"). When the questionnaire was completed, it was mailed in. The women in the study lost an average of 4 kg of weight in the ensuing 12 weeks. The hedonic ratings of cheeses remained between 7 and 8 on the 9-point scale (near the extremely pleasant end of the scale) throughout the study. Although the ratings for non-fat milk initially were between 6 and 7, they dropped significantly over the remainder of the study. Liking for the sweetest juices (9%, 12% sucrose) also dropped significantly.

The responses on the questionnaire showed that liking for high-fat foods and pastry decreased significantly whereas liking for low-fat foods and high-fibre foods increase significantly over the 12 week period. Significant decreases in consumption of high-fat foods and pastries and a significant increase in consumption of high-fibre foods were found. No significant correlations were found between changes in hedonic responses to foods and metabolic control data e.g., blood glucose, glycosylated hemoglobin.

This study is important because it is the only one that has attempted to measure preferences and palatability as persons with type 2 diabetes have eaten the

diabetic diet. However, the rating in the study was restricted to pleasantness. The purpose of palatability testing is to measure ratings of pleasantness for a variety of sensory characteristics of the food being eaten, e.g., pleasantness of its smell, taste, texture, creaminess, richness, sweetness, saltiness, sourness, and/or bitterness. For example, a high rating of pleasantness of saltiness or sweetness of a food is taken to mean that this particular food is highly palatable to the person. Had these other sensory aspects of food been assessed, a more robust picture of what made particular foods being consumed palatable to the subjects with type 2 diabetes would have emerged. Probably the most important point in this study is that preference for fat remains high even though the patient is not ingesting the food they find particularly palatable. This suggests that preference and palatability are dimensions of eating behavior which are integrated with other processes e.g., cognitive, neural, affective, cultural, and the expectation that change will be detected in physiological processes, such as blood sugar level, is misplaced.

One report of a dietary study comparing the HM to the HC diet (Campbell, Borkman, Marmot, Storlien, and Dyer, 1994) in free-living persons with type 2 diabetes provides some information regarding preference. In this study, the 10 male subjects with NIDDM were asked to rate the diet, ease of preparation, expense, taste, satiety, and variety of the HM compared to the HC diet, using visual analogue scaling. Campbell reports that the HM and HC diets were rated similarly in terms of palatability, although the various dimensions reported are not considered to be

dimensions of palatability but rather more related to the ease of preparing a particular diet.

Another study with free-living healthy adults has been conducted by Mattes (1993) in which subjects were randomized to three groups: a) reduced-fat group whose fat-restricted diet (American Dietetic Association) also prohibited any discretionary fat sources; b) a fat-modified ADA diet but with discretionary fat allowed; and c) no dietary modification. The diet phase was 12 weeks with 12 additional weeks of follow up. The design of this study included measures of adherence to the diet(s) such as 24-hour urinalysis matching the electrolytes excreted with those consumed in the diet, and testing for urinary lithium carbonate that was added to margarine used by two of the three diet groups. Subjects were provided with recipes, educational materials, follow up, praise and reinforcement. Taste testing to identify preferences for fat was conducted at baseline, 12 and 24 weeks.

The taste testing procedure used by Mattes involved presenting two groups of taste samples to the subjects. The first group of samples consisted of milk, chocolate milk, vanilla pudding, and tomato soup, each containing five levels of fat. The milk samples contained 0.18% (skim), 3.3% (whole), 11.5% (half and half), 37.5% (whipping cream), and 49.8% (whipping cream plus oil). Chocolate milk was made by combining the five levels of milk/fat with chocolate milk powder; vanilla pudding was made by combining pudding powder with the 5 levels of

milk/fat; tomato soup was made by combining the five levels of milk/fat with tomato soup base. The second set of taste samples consisted of samples of 10 common foods in the low and high fat version e.g., margarine, American cheese, cottage cheese, cream cheese, Monterey Jack cheese, pound cake, soda crackers, Italian salad dressing, French dressing, and mayonnaise). Each sample was tasted and savored twice, swallowed, and then the rating was completed. Deionized water was used to rinse and spit between each sample. Each twice-tasted sample was rated using a 9-point scale as to how much the patient liked the creaminess, oiliness, sweetness, saltiness, sourness, bitterness, and pleasantness of the sample. A high rating of liking and pleasantness was taken to mean that the sample was preferred and highly palatable.

Mattes found that palatability ratings for high-fat foods e.g., milk, chocolate milk, vanilla pudding, tomato soup made with whipping cream and whipping cream plus oil, and the high fat version of common foods, as measured by the taste testing procedure, were significantly reduced only in the group that had limited exposure to fats, e.g., the reduced-fat group with no discretionary fat allowed.

It is inferred that the other patient groups, e.g., those with a low fat diet which included discretionary fat and those on the control diet, did not have these same responses because they were still receiving foods which contained some fat. Like the subjects in Laitenen's study (1991), it could be inferred that the subjects who had the lowest fat diet still preferred and found palatable foods that were high

in fat, despite being without these foods for a period of 24 weeks. Neither of these studies measured the palatability of all the foods consumed for one day in the prescribed diet. Whether these findings constitute a preference for fat is not clear, nor is it clear that the ratings of palatability and preference for fat meant that the person would selectively seek to eat these foods, even if it meant breaking the research study diet or a therapeutic diet.

Mattes (1996) believes that there is a mechanism in the oropharyngeal area which monitors whether fat is in the food that enters the mouth. If fat is detected, a cascade of "cephalic" responses are initiated (Mattes, 1997). The connection to preference is less clear, since in each of these studies the foods that were evaluated were mixtures of carbohydrate, fat and protein and it could be that what was preferred was the mixture, rather than any one particular macronutrient in the food. Finally, correlations between measures of palatability and fat preferences and other physiological measures such as blood glucose level or body mass index are low to insignificant, indicating that the role of preference and palatability has yet to be linked to metabolic and other events going on at the same time.

Motivation to Eat.

A model has been developed (Figure 1) by which motivational aspects of eating behavior can be monitored prior to, during, and following a meal (adapted from Blundell, Rogers and Hill, 1988). Prior to a meal, the variables that are assessed include perceived level of hunger and fullness, ratings

of preference for a group of common foods 8 of which have high protein composition, 8 have high fat composition, and 8 are low calorie foods; forced-choice test between high carbohydrate and high protein items; and a checklist of bodily sensations related to general feelings and moods.

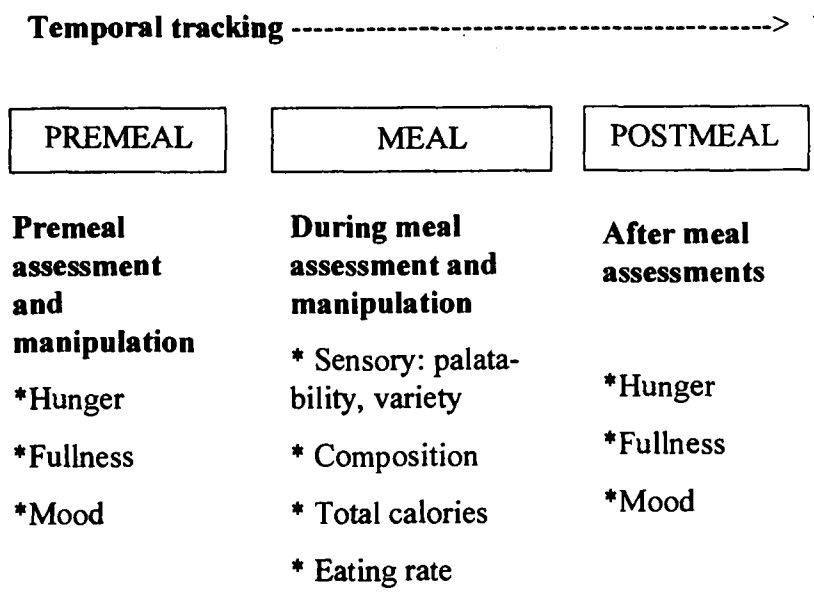

Figure 1. Experimental strategies for tracking changes in motivational variables and eating behavior.

During the meal, the time it takes to eat the meal, ratings of the palatability (sensory characteristics of the food that are pleasant), and ratings of hunger, fullness, and mood are completed. Throughout the whole episode, subjects maintain a diary so that any other aspects of the experience that could have been missed previously can be documented. A profile of predicted responses was developed

(Figure 2) as well as a schema of mediating variables (Figure 3) (adapted from Blundell, Rogers and Hill, 1988).

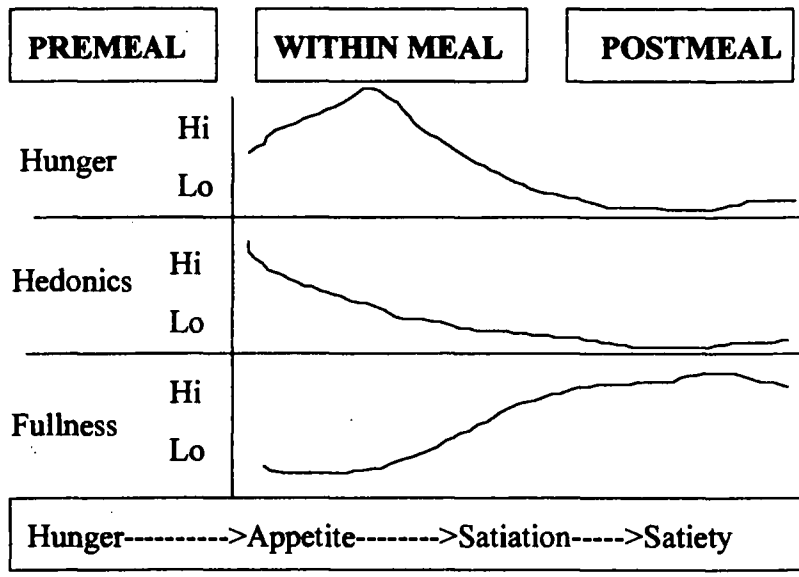

Figure 2. Predicted changes in motivational parameters which accompany eating during the course of a meal.

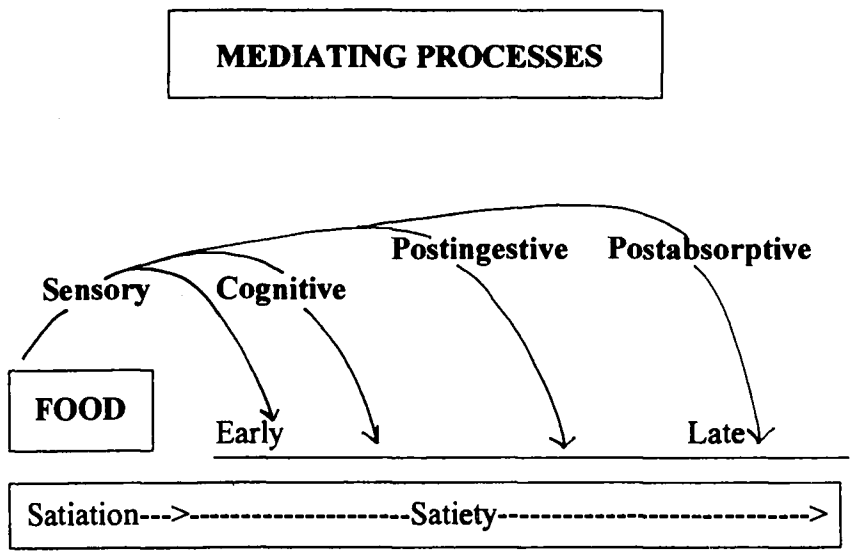

Figure 3. Contributions of sensory, cognitive, post-ingestive and post-absorptive stimuli to the time course of satiety.

This model has been tested to determine the satiating effects of meals differing in carbohydrate and protein content with normals (Barkeling, Rössner, and Björvell, 1990, Blundell and Hill, 1987) and with obese women (Hill and Blundell, 1990; Lawton, Burley, Wales and Blundell, 1993).

The motivation theory base related to eating that are incorporated in Blundell's work are homeostatic, incentive, and systems theory models (Toates, 1986). The homeostatic model, often called a "depletion-repletion" model, assumes that motivated behavior is synonymous with depletion and satiety with repletion. Thus, measurements of hunger and satiety over time reflect depletion of body nutrients and repletion of body nutrients, respectively. There are limitations to this view when it is applied to eating behavior. First, even when there is no depletion of nutrients, human beings are known to eat; and secondly, even when deficient, there is no adequate explanation in the homeostatic model for why humans seek food rather than, say, water.

Bindra (Bindra and Palfai, 1967) then expanded this theoretical base by proposing that there is interaction between the underlying physiological state and stimuli related to the goal (Toates, 1986). Stimuli associated with food, because of their positive or negative hedonic characteristics, "pull toward" or "push away" the person from the goal toward which the motivation is directed. Bindra includes viscerosomatic regulatory reactions to the stimuli such as hunger, fullness,

salivation, and transactional reactions to food stimuli such as smelling, biting, chewing, swallowing, which provide information about the goal of motivated behavior.

Systems motivational theorists focus on the goal part of motivated behavior, e.g., the food itself, and suggest that the goal possesses sensory stimuli which are detected and evaluated by the motivated person. At the central nervous system level, comparisons are made between the goal and other goals, memories are revived regarding the goal, the physiological state is reviewed and "alerted", and motivated behavior is demonstrated toward (or away from) the goal. Variables which are measured in the work of Blundell, Rogers and Hill builds on these three blended views of motivated behavior.

Measurements of hunger and satiety are based partially on homeostatic theory and on the hedonic capabilities of stimuli related from the goal. For example, the smell and sight of freshly made popcorn may emit positive hedonic stimuli which "pull toward" the food, especially if the motivated person has pleasant memories and experiences associated with eating popcorn and the knowledge that popcorn without butter on it is an acceptable alternative to a high fat snack. Measurement of variables prior to eating, and measurement of palatability of the food exemplify Blundell and Hill's effort to identify positive as well as negative hedonic aspects of food stimuli for the patient. Measurement of prospective consumption (how much do you think you can eat ?), of forced choices, and of favored foods where

comparisons are made and memories evoked, illustrates application of principles of the systems theory approach to motivated behavior.

Interactions between various elements of a motivational system are also known to occur. For example, it has been found that palatability of food is positively related to hunger during and following meals (Hill, Magson, and Blundell, 1984); that ingesting highly palatable food increases the rate of eating and decreases time of the meal (Bellisle, Lucas, Amrani, and LeMagnen, 1984), and evokes release of opoid-like substances in the brain associated with reward and satisfaction (Drewnowski, Krahn, Demotrack, Nairn, and Gosnell, 1992), which in turn may be a mechanism for the memory base and the formation of preferences for eating particular food or group of foods. There are no known studies with persons who have type 2 diabetes which have attempted to include these as well as other more common variables in feeding studies with this population of patients.

Thinking and Ideas about Food.

A variety of valid and reliable instruments exist which measure thinking and ideas about food. One instrument that is commonly used in obesity research is the Three-Factor Eating Questionnaire (Stunkard and Messick, 1985), subsequently renamed as the Eating Inventory (Stunkard and Messick, 1988). It is used to measure three related aspects of eating behavior. The first subscale in this instrument measures cognitive restraint of eating; by this is meant, the degree to which restraint in eating food is exercised to control caloric intake. A high score in this subscale

indicates highly restrained eating with strict control of caloric intake. The second subscale measures susceptibility to situational challenges to eat a particular prescribed diet. A high score in this subscale would indicate a feeling of vulnerability with easy loss of control of eating restraint. The third subscale is related to hunger and indicates responses to perceived hunger. A high score here would indicate that feeling hungry leads to a loss of eating restraint.

The Three Factor Eating Questionnaire has been used in a study of persons with type 1 (n=31) and type 2 diabetes (n=41) (25 women, 16 men) (Straub, Lamparter-Lang, Palitzsch and Schölmerich, 1996). The purpose of this study was to assess eating behavior and to see if correlations existed between the scores in subscales of the Three Factor Eating Questionnaire and measures of cardiac autonomic function including blood pressure, serum lipids, measures of glycemic control, and BMI. The subjects with type 2 diabetes had an average score of 14.9 ± 0.7 on the cognitive control subscale, a score which indicates a high degree of cognitive control, e. g. high restraint, with respect to eating foods consistent with the diet. On the susceptibility to social challenges subscale, the subjects with type 2 diabetes scored 3.4, indicating a fairly low degree of susceptibility to situations that would disinhibit and lead to loss of restraint. These two scores represent the best possible profile that could be obtained by the person in type 2 diabetes, e.g., restrained eating and low vulnerability to social situations. The investigators multiplied the cognitive control score by the susceptibility score to get a

disinhibition score; subjects with diabetes had an average disinhibition score of 52.7 ± 6.6 (range: 0 - 152). The only significant correlation (r = 0.40, p 0.01) that was found was between the susceptibility and the combined disinhibition score (0.43, p 0.01) and BMI. This r value is modest, and the correlation means that as the scores for loss of control and susceptibility to disinhibition rise and restraint over the diet is lost, BMI also increases. This makes sense. However, these type 2 diabetics rated themselves as able to control their eating and defend themselves from susceptibility to lose restraint in maintaining the diet, and if they were actually this successful, BMIs should have been lower. It may be that those type 2 diabetics in this study were responding to the questionnaire in ways that they felt they should respond, e.g., socially desirable responses, a type of response set bias. However, Allison, Kalinsky, and Gorman (1992) found that it was unlikely subjects endorse items in this instrument based on perceived desirability. It may also be that the factors chosen by Straub et al. are not the best to use with type 2 diabetics. The investigators did not report data related to the third subscale (hunger) because they found that it correlated very highly to the second subscale. Therefore, no information is available about the relationship between feelings of hunger and restraint; such information would have filled a gap in knowledge about eating behavior in persons with diabetes.

Other instruments with reliability and validity include the Dieter's Inventory of Eating Temptations (DIET), the Situation-based Dieting Self Efficacy (SDS),

Situational Appetite Measure (SAM), and Eating Self-Efficacy Scale (ESES), Forbidden Food Survey, Eating Related Characteristics Questionnaire (ECQ), and the Yale Eating Pattern Questionnaire (Schlundt, 1995, pp. 262 - 302). There are no reports of these instruments being used with women with type 2 diabetes.

Hunger and Fullness.

Hunger or fullness can be described as a motivational states which are linked to a set of behaviors such as eating or cessation of eating. Another common definition of hunger is as a set of bodily sensations and/or symptoms which are associated with the need for food (Mattes & Friedman, 1993). A person may detect these common or familiar sensations, interpret them as representing the need for food, and subsequently, seek food. Similarly, fullness is a term used to describe sensations or symptoms related to the desire to terminate a meal, and when detected, leads to behavior which discontinues intake of food. Hunger and fullness have been included as dependent variables in many research studies involving healthy men and women (Warwick, Hall, Pappas and Schiffman, 1993; Blundell, Burley, Cotton and Lawton, 1992), obese persons (Lawton, Burley, Wales and Blundell, 1993) and to a much lesser extent, individuals with type 2 diabetes (Campbell et al., 1994). Visual analogue scales with various types of anchors, e.g., not hungry at all, hungriest I have ever been; very full, not at all full, have been utilized with these scales. While hunger and fullness can be measured as subjective

experiences, the sensitivity of measurement, especially with hunger, has been poor (Mattes and Friedman, 1993).

In many of the studies which have measured hunger and fullness in normal and obese individuals, the researcher has administered a preload meal to subjects which contains a known or fixed amount of fat. The outcome on subsequent intake is taken to reflect the influence of the components of the preload on hunger or fullness/satiety. If a high fat preload is used, then subsequent reduction in hunger supposedly reflects the satiating ability of the fat in the preload. Blundell thinks this approach to eating behavior is limited. He proposes instead, when evaluating the effect of fat content of foods, to provide subjects with a wide range of foods and allow them to eat ad lib to comfortable fullness. When Blundell has done this with obese subjects, he has found what he calls passive over consumption of fats, suggesting that consumption of high fat foods does not result in satiation. Rather, ingestion of high fat foods seems to stimulate intake of more food (Lawton, Burley, Wales and Blundell, 1993).

Recently, Blundell et al., 1996 has found, based on analysis of a large data set in the Leeds High Fat Study, a positive correlation between persons with high fat intakes (g per day) and BMI, a relationship that has previously not been supported in other studies.

In summary, the measurement of hunger and fullness has been used by at least one research group for some period of time. Although they originally expected

to see a major reduction in hunger and increase in fullness at the end of meals that contained fat, and expected to see some kind of caloric adjustment in the selection of foods in the next meal(s), these responses did not appear in obese patients who were studied. Instead, rather than a significant reduction in hunger and rise in fullness after the meal, only weak ratings of fullness were obtained, these ratings lasted only a short time, and in the next meal, subjects who were in testing situations ate at the level of the previous meal, or overate. For these researchers, the measurement of hunger and fullness is a meaningful variable, although none of their studies have extended for more than an 12 - 18 hour period. Whether this same pattern would be true with person with type 2 diabetes is not known.

Common conceptions about human eating behavior continue to exist. For example, in the discussion of a large outpatient feeding study comparing HM with HC diets in men and women who had type 2 diabetes, the following analysis was offered:

"....it has been known since the 19th century that fat is more satiating and inhibits gastric emptying. Increased palatability of high-monounsaturated-fat diets may in fact mean better patient compliance to the prescribed diets." (Garg, Bantle, Henry, Coulston, Griver, Raatz, Brinkley, Chen, Grundy, Huet and Reaven, 1994, p. 1427).

It remains to be seen whether hunger is lower on a day to day basis, whether satiation is greater on a day to day basis, and whether high-monounsaturated-fat diets have increased palatability in women with type 2 diabetes.

Dietary Monounsaturated Fatty Acids and Type 2 Diabetes

The diet for the person with type 2 diabetes has long been the subject of debate. The effective diet should provide improvement of plasma lipids, lipiproteins, and glucose values over a period of time while reducing complications associated with the disease. For healthy individuals, replacing saturated fatty acids with monounsaturated fatty acids or equivalent caloric amounts of carbohydrate tends to decrease total cholesterol and LDL-cholesterol while raising HDL-cholesterol, changes which are associated with decreased risk of cardiovascular disease. However, consumption of a HC diet is also associated with an increase of serum triglycerides (Coulston, Hollenbeck, Swislocki, Chen and Reaven, 1987; Coulston, et al., 1989; Blades and Garg, 1995). Since a very common lipid abnormality in type 2 diabetes is elevated concentrations of serum triglycerides, and HC diets are also associated with elevations of triglycerides, the appropriateness of the HC diet for the person with type 2 diabetes has been questioned. This has led to exploration of the effect of diets with increased concentrations of monounsaturated fatty acids in persons with type 2 diabetes.

The feeding studies related to increased concentrations of monounsaturated fatty acids can be categorized into four groups: a) studies which compare the HM diet to a baseline/habitual diet (n=2); b) studies which compare the HM to the HC diet (n=16); c) studies which compare HM (*cis*) to HM (*trans*) diet (n=1); and d) studies which compare the HM diet to one that is enriched with polyunsaturated fatty acids (HP) (n=2).

Feeding Studies Comparing the HM Diet to a Baseline/Habitual Diet. Investigators in Ireland published two studies in 1996 which compared the effect of the HM diet to a habitual diet in free-living persons with type 2 diabetes and a control group of nondiabetics (Dimitriadis, Griffin, Collins, Johnson, Owens and Tomkin, 1996; Griffin, Dimitriadis, Lenehan, Owens, Collins, Johnson and Tomkin, 1996)(Appendix 1). The diet study period was four weeks in length; saturated and polyunsaturated fats in the diet were replaced with monounsaturate-rich spreads and extra virgin olive oil. The outcome variables in these two studies included lipids, lipoproteins, lipoprotein composition, LDL fatty acid profiles, cholesterol synthesis, insulin and blood glucose concentrations. When comparing initial to final values, a significant increase in HDL-cholesterol and predicted changes in the LDL fatty acid profile occurred with oleic acid rising and linoleic acid falling significantly.

Feeding Studies Comparing the HM to the HC Diet.

The first study on the effect of monounsaturated fatty acids in the diabetic diet with persons who have type 2 diabetes was reported by Garg, Bonanome,

Grundy, Zhang and Unger in 1989 (Appendix 2). Ten men with type 2 diabetes were fed 60% of their total calories as carbohydrate and 25% as fat (9% monounsaturated fatty acids) in one arm of the study; this was compared with 35% carbohydrate and 50% fat (35% monounsaturated fatty acids) in the other arm of the study. These subjects had slightly elevated plasma cholesterol and triglycerides at baseline when these values are compared to standards for norms. Whereas there were no significant changes in total cholesterol or LDL-cholesterol level on either diet, plasma triglyceride and apolipoprotein A-1 significantly increased on the HC diet but decreased on the HM diet. Glycemic variables were lower on both diets when compared to baseline.

This study was vigorously criticized on several points: a) the foods provided on either diet were not published, so it was not possible to assess the influence of refined or complex carbohydrate in the diet; b) the metabolic unit setting of the study was artificial and not applicable to free-living patients; c) the levels of fat in the diet were higher than the typical consumption of adult Americans, and d) the levels of monounsaturated fat used in the study would, under free-living conditions, most likely result in increased weight.

Despite these criticisms, 14 other studies have been conducted which compare the HM to HC diet from the period of 1990 to 1999 (Rivellese, Giacco, Genovese, Patti, Marotta, Pacioni, Annuzzi and Riccardi, 1990; Bonanome, Visonà, Lusiani, Beltramello, Confortin, Biffanti, Sorgato, Costa and Pagnan, 1991; Parillo,

Rivellese, Ciardullo, Capaldo, Giacco, Genovese and Riccardi, 1992; Garg, Grundy and Koffler, 1992; Rasmussen, Thomsen, Hansen, Vesterlund, Winther and Hermansen, 1993; Lerman Garber, Ichazo-Cerro, Zamora-Gonzàlez, Cardoso-Saldaña and Posadas-Romero, 1994; Campbell, et al., 1994; Garg, et al., 1994; Blades and Garg, 1995; Walker, et al., 1995; Rivellese, 1997; Low, Grossman and Gumbiner, 1996; Heilbronn, Noakes and Clifton, 1999).

The results of these studies with respect to the impact of monounsaturated fatty acids in persons with type 2 diabetes need to be understood within the context of the design of these studies. There is a great deal of variability in the composition of the independent variables, e.g., the HM and the HC diet. For example, the range of total percent of calories on the HM diet as fat ranges from 36 - 70%, (mean of 43.3 ± 8.64) with the monounsaturated fatty acids in the HM diet ranging from a low of 20 to a high of 33% (mean of 27.3 ± 7.35) of the fat in the diet. There is also variability in the strength of the independent variables because only half of the studies have been conducted within a metabolic unit or as an outpatient study, settings which are better able to ensure only the HM or HC diet is eaten, and that actual composition is equivalent or very close to what is planned. While all but two studies were randomized crossover designs, the length of time in the HM or HC arms of the study ranged from 2 weeks to as long as 12 weeks (mean of 4.88 weeks ± 2.80). Roughly half of the studies which should have had a washout phase, did not have one. Subjects in these studies were often not homogeneous, e.g., subjects on

either diet or diet plus oral agents, subjects on different types of oral agents. It is against this backdrop that some generalizations about the lipid, lipoprotein, and glycemic outcomes of HM as compared to HC diets are summarized.

The findings from these studies suggest that the HM diet, when compared to the HC diet, may increase or very modestly lower total cholesterol; significantly lower triglycerides and VLDL-triglyceride, if measured; have a variable effect on LDL-cholesterol; usually increase HDL-cholesterol, although modestly; and will not cause deterioration of glycemic control. The lack of a predictable or powerful impact of the HM diet when compared to the HC diet may also be due to design and sample issues.

Other outcome variables in these studies have shown that the HM diet significantly lowers daytime and 24 hour systolic blood pressure, daytime and diurnal diastolic blood pressure (Rasmussen, Thomsen, et al., 1993), and lowers von Willebrand factor in the blood (Rasmussen, Thomsen, Ingerslev and Hermansen, 1994).

During this period of time, however, the rationale for HM diets continued to be questioned and confirmation of hypertriglyceridemia seen with HC diets as a transient or a persistent phenomenon was sought. To answer this question, some feeding studies comparing HM to HC diets began to obtain postprandial as well as fasting sampling of outcome variables. For example, Rivellese (1997), in a study comparing HM and HC diets, noted that while there were statistically significant

differences between triglyceride and VLDL-triglyceride obtained at baseline in a 6 hour postprandial test at the end of the HM or HC period, these differences disappeared by the sixth postprandial hour. This comment suggests that the HM or HC diet effect may need to be obtained from postprandial tests as well as fasting tests of outcome variables at the end of a diet period..

Another explanation for the hypertriglyceridemia associated with HC diets was that the carbohydrates in the diet may have had a high glycemic index, thus raising insulin production, resulting in increased triglycerides and decreased HDL-cholesterol (Wolever, 1999). Careful attention to the glycemic index of carbohydrates without changing the amount of carbohydrate in the diet held as much promise to correct hypertriglyceridemia as did HM diets. This line of reasoning was exemplified in the study conducted by Rasmussen, Lauszus, Christiansen, Thomsen and Hermansen (1996) who fed 12 subjects with type 2 diabetes five differing test meals comprised of mashed potatoes, a food with a high glycemic index (Brand-Miller and Foster-Powell, 1999). The mashed potatoes were ingested alone, or in combination with 40g and 80g of olive oil, 50g and 100g of butter. Blood samples for lipids, blood glucose and insulin were collected at 15 minutes intervals for 4 hours postprandially. The findings were that triglycerides increased after each meal regardless of which fat was ingested. Of the two fats used, one high in saturated fatty acids, the other high in monounsaturated fatty acids, butter suppressed glucose response area and stimulated the release of insulin, whereas olive oil did not elicit

these responses. This study could be criticized because the actual fatty acid composition of the olive oil and butter was not ascertained, eating mashed potatoes does not replicate actual eating patterns of multiple foods within a meal, and the four hour period of data collection was probably too short. In summary, whatever monounsaturated fatty acids do metabolically and physiologically, is probably not adequately captured using the test meal approach alone.

Another approach to hypertriglyceridemia associated with HC diets has been to add fiber at levels of ~ 50 g/day to the complex carbohydrate (starch) already in the diet (Rivellese, Auletta, Marotta, Saldalamacchia, Giacco, Mastrilli, Vaccaro and Riccardi, 1994). Lipid outcomes in the HM, HC, and HC plus added fiber diets were equivalent on both the HC and the HM diet. However, there were problems with patient attrition in two of the three diet interventions. Some patients dropped because they could not reach fiber intakes of 50 g/day; other patients dropped the HM arm of the study because there weren't enough food options, the diet was not satisfying, and the amounts of oil that had to be added to food were unpleasant.

There are two studies which compare energy-restricted/hypocaloric HM with energy-restricted/hypocaloric HC diets (Low et al., 1998; Heilbronn, et al., 1999). The Heilbronn study was conducted with 35 free living type 2 diabetics randomized to energy-restricted HM, HC, and high saturated fat (SF) diets. The study period was 12 weeks; key foods were provided appropriate to the particular diet. The surprising finding in this study was that weight loss was not significantly

different between the three diet groups, and averaged about 7 kgs. Plasma triglycerides did not change significantly, nor did any one diet affect it. The reduction of total cholesterol was greater on the HM than the HC diet and increased in the SF diet. LDL-cholesterol was reduced on both HM and HC diets, but the reduction was significant only on HM. HDL-cholesterol levels were maintained on the HM and SF diets, and significantly reduced on the HC diet.

The other HM/HC hypocaloric study was with a group of 17 men and women in an outpatient feeding study (Low et al, 1996). Subjects were taken off diabetes medications prior to the study and unless blood glucose became greater than 16.5 mmol/L, no diabetes medication was given throughout the study. The subjects were then divided into a HM group (n=9) and a HC group (n=8). The caloric level was set at 50% of Harris Benedict estimations for each patient. The two concurrent diet phases were 6 weeks in length, followed by a 6-week refeeding phase. Weight loss outcomes of this study were similar between the HM and HC groups (7.3 vs 8.3 kg).

Feeding Studies Comparing the HM *cis* Versus the HM *trans* Diet.

The Christiansen, Schnider, Palmvig, Tauber-Lassen and Pedersen (1997) study was comprised of 16 type 2 subjects who were provided a diet with 30% of the total calories as fat; *cis* and *trans* forms of monounsaturated acids accounted for 20% of the total fat calories (Appendix 3). The HM-*cis* and HM-*trans* diets were compared to a diet high in saturated fat (SF). No significant differences were found

in the effect of the *cis* or *trans* form of the HM diet on lipids, lipoproteins, or glycemic variables.

Feeding Studies Comparing the HM to the HP Diet.

Two diet studies comparing the HM diet to a diet rich in polyunsaturated fatty acids (HP) in type 2 diabetics are reported (Thomsen, et al., 1994; Parfitt, Desomeaux, Bolton and Hartog, 1993) (Appendix 4). No significant differences were found in either study in terms of lipid or glycemic parameters when comparing the HM to the HP diet. However, Thomsen's study did find that 24 hour systolic and diastolic blood pressures were significantly lower on the HM diet. This finding is similar to that of Rasmussen et al.'s work (1993) comparing HM to HC diets.

Within this period of multiple HM dietary feeding studies, it was hypothesized that HP may more readily promote LDL oxidation; therefore, this variable was added to HM versus HP feeding studies. Oleate-rich plasma LDL of rabbits fed monounsaturated fatty acid enriched chow showed the most resistence to oxidative change, and there was limited uptake of this oleate-rich LDL into cultured endothelial cells (Parthasarathy, Khoo, Miller, Barnett, Witzum and Steinberg, 1990). Although the mechanisms were not fully known, evidence for a relationship between hyperglycemia, oxidation of LDL, and the early (macrophages and foam cells) and later changes (placque) associated with micro- and macrovascular changes in blood vessels is emerging (Giugliano et al., 1996). It is hypothesized that when the LDL particle is oxidized in the intima of a blood vessel, LDL is taken up in an

unregulated fashion causing cholesterol to accumulate and transform macrophages into foam cells.

Persons with type 2 diabetes have been shown to have higher levels of plasma lipid peroxides than normals or persons with type 1 diabetes (Griesmacher, Kindhauser, Andert, Schreiner, Toma, Knoebl, Pietschmann, Prager, Schnack, Schernthaner and Mueller, 1995). In addition, plasma levels of thiobarbituric acid reactive substances (TBARS) are elevated in persons with type 2 diabetes as compared to persons with normal and impaired glucose tolerance and when subjects who smoke or have vascular diseases are removed from the sample (Sundaram, Bhaskar, Vijayalingam, Viswanathan, Mohan, Shanmugasundaram, 1996; Haffner, Stern, Agil, Jialal and Mykkanen, 1995). Although the evidence is limited, HM diets seem to have an impact on measures of lipid peroxidation, suggesting that this diet may offer an advantages over others in reducing lipid peroxide formation through alteration in lipoprotein fatty acid content.

Of the reports of feeding studies involving type 2 diabetics and HM diets, only three have included in vitro measures of lipid peroxidation as an outcome variable. Parfitt et al. (1994), when comparing a baseline to a HM or HP diet, found that lipid peroxides were reduced on both HM and the HP diet, and conjugated dienes were significantly reduced as well as being the lowest on HM. In 1996, Dimitriadis et al. found that the HM diet increased LDL lag time from 140 to 170 minutes although this finding was not significant; however, maximum rate of

LDL oxidation was significantly reduced, and the concentration of conjugated dienes in LDL-cholesterol was also significantly reduced. Increased lag time in the LDL particle was also found in subjects on the hypocaloric HM diet (Gumbiner, Low and Reaven, 1998) whereas LDL lag time dropped when subjects were on the HC diet. While these findings related to lipid peroxidation are encouraging, they do not fully explain the complex in vivo phenomenon of lipid peroxidation which also involves levels of antioxidants such as vitamin E, vitamin C, and interactions of various enzymes which scavenge free radicals that are created. It appears, in view of the small number of reports of findings related to lipid peroxidation, that there is need for additional exploration of lipid peroxidation associated with diets high in monounsaturated fatty acids.

This review of the literature in the areas of eating behavior and HM diets suggests that little is known about eating behavior of postmenopausal women with type 2 diabetes. In addition, although nearly three hundred persons with type 2 diabetes have been subjects in studies comparing HM diets to other diet conditions, less than half of them have been women, very few have had coexistent hypertriglyceridemia or obesity, and the control of the independent variable of the HM diet has been lacking in most of the studies (Appendix 5). Designing and implementing well controlled dietary feeding studies to explore the effect of monounsaturated fatty acids in the context of diabetes and coexistent cardiovascular disease is of great importance because the findings from such studies can be

generalized and can contribute to formation of the evidence base for HM diets (Kris-Etherton and Dietschy, 1997).

Two articles for publication in the scientific literature describe the results of a study to describe eating behavior and the effect on lipids, lipoproteins, and lipid peroxidation in postmenopausal women with type 2 diabetes of a HM compared to a HC diabetic diet.

EATING BEHAVIOR OF POSTMENOPAUSAL WOMEN WITH TYPE 2 DIABETES

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Abstract

Eating behavior of ten women with type 2 diabetes was observed during an outpatient feeding study in which the effects of a diet enriched with monounsaturated fatty acids (HM) was compared with a diet high in carbohydrate (HC). Time of starting and stopping eating was positively correlated with temporal preference, e.g., morningness-eveningness preference type on HM and HC. No significant differences between number of meals, snacks, or total eating episodes in the HM and HC diet phases were found. HM and HC diets were both rated as highly palatable. Hunger and fullness ratings varied within and between subjects, with only two subjects exhibited a similar pattern in both diet phases. Taste testing to determine fat preference revealed no significant differences at the end of HM and HC. Subject's taste preferences when compared with weight- and age-matched nondiabetic women, showed similar ratings of liking for some high-fat and low-fat foods, although subjects and controls differed significantly in ratings of liking of salty, sour, and bitter foods. The findings indicate that temporal preferences are related to eating pattern and do not vary even if composition of the diet does. In addition, differences between diabetic and nondiabetic women when rating liking of some (salty, sour, bitter) tastes suggest that type 2 diabetes per se may alter some aspects of taste.

Introduction

People with diabetes view the diet as the most challenging and difficult part of their therapy and a gap between what is prescribed and what is actually eaten continues to be found (Lockwood *et al.*, 1986; Shimikawa *et al.*, 1993; Sullivan & Joseph, 1998). Closing the gap between established eating patterns and development of new eating patterns related to the diabetic diet is necessary, because the benefits of the diabetic diet are dependent on long-term ingestion. Women of all ages exceed the number of men with diagnosed diabetes in the United States, and except for 55-64 year olds, more new cases of diagnosed type 2 diabetes occur in women than men (National Diabetes Data Group, 1995). Thus, the challenges of changing eating behavior are particularly applicable to women with type 2 diabetes.

A taxonomy of situational challenges to eating the diabetic diet has been identified, and includes overeating foods at social events which are not a sanctioned part of the diet, having unsupportive family and friends, having to eat away from home where there is uncertainty over access and composition of food, and the need to plan ahead to have food consistent with therapeutic recommendations readily accessible. Other internally-anchored challenges include cravings and feeling deprived of palatable and preferred foods, discriminating between hunger related to low blood glucose from normal hunger and treating it effectively, being tempted to give up, and negative emotions (Schlundt *et al.*,1994). Boredom, confusion, guilt,

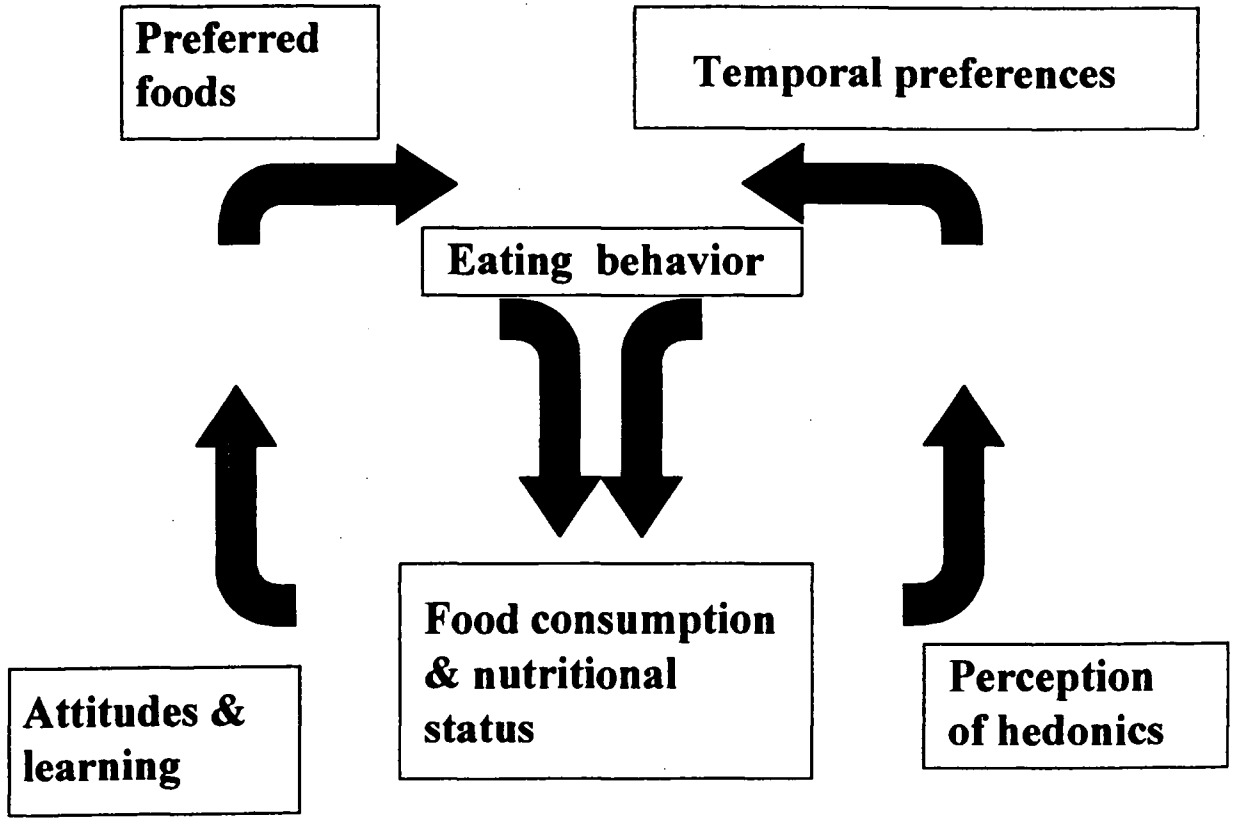
and anxiety about food, food preparation, and meal timing have also been reported (Rubin, 1993; Travis, 1997). These challenges also reflect factors which affect eating behavior in the person with diabetes.

Some of these variables related to eating behavior are depicted in Figure 4. Preferred foods are those that correspond to a person's liking for the texture, smell, and odor (hedonic characteristics) of the food; such foods are viewed as palatable and pleasant to eat. Other variables that may affect food selection and consumption are temporal preferences, and attitudes and learning (adapted from Drewnowski, 1997). Whereas preferred foods would guide number and frequency of meals, food selection, food consumption, and ultimately, nutritional status, preferences for time of day for eating most likely would guide the eating period of the day. Research studies of eating behavior in individuals who are obese have begun to build a basis for understanding the role of preferences in eating behavior.

One characteristic of eating behavior in women who are obese is a preference for fat (Drewnowski,1992). A potential way in which preference for fat leads to specific consumption patterns has been described by Blundell, *et al.*, (1993). In the laboratory setting, 12 obese women (average BMI =39 kg/m^2) were fed a low (527 kcal) or high (985 kcal) meal at midday. At the following dinner meal, subjects were free to select and eat high fat or high CHO foods. The total calories and components were taken as being representative of the satiating capacity of the components of the lunch meal previously ingested (either high fat or high CHO), and

FIGURE 4

Factors which affect eating behavior (adapted from Drewnowski, 1992)



food consumed after the free-choice meal were taken to represent the impact of composition of the free-choice meal on subsequent energy intake. These investigators concluded that high-fat foods have limited effect on satiation, because when subjects were free to chose foods after the high fat meal, they were hungry and chose high fat foods. Intake following the free choice meal was between 310-390 calories, not statistically significant. Blundell extends Drewnowski's observation of the preference for fat and says that due to the limited ability of fat to produce enduring satiation, hunger returns and in the next meals, leads to what he calls "passive over consumption." He also contends that obese subjects are unaware of these patterns of consumption. If obese women prefer fat prior to diagnosis of type 2 diabetes, it is likely that this preference will continue after diagnosis is established, and may explain the craving and feeling deprived of palatable and preferred foods described by women with diabetes. However, changing preference for high fat foods may be difficult.

Mattes (1993) conducted a study to see if preference for fat could be changed in healthy men and women who were ingesting a no-fat (NF) or a modified-fat (MF) diet, compared to subjects ingesting a control diet (C) for 12 weeks. During the study, taste testing which involved repeated measures of liking for sensory (creaminess, oiliness, sweetness, sourness, saltiness, bitterness, pleasantness) aspects of four common foods (milk, chocolate milk, vanilla pudding and tomato soup) containing five levels of fat and of 10 foods commonly available in

grocery stores in a regular fat and low-fat form was done. Mattes found that only in the NF group, compared to the MF and C groups, were pleasantness ratings reduced for the ten low/regular fat foods at the end of the study, and these ratings persisted for 12 weeks after the study ended. No estimates of hunger or satiation are reported. The study, in addition to suggesting a different methodology for taste testing and measuring shifts in fat preference, also suggests that to re-educate a person from a preference for fat requires having very low amounts of fat in the diet for a fairly long period of time, a situation which is very difficult for people to achieve on a free-living, long-term basis. An alternative would be to design a diet for overweight and obese women with type 2 diabetes with more rather than less fat which also achieves goals for glycemic control, making a better match between preference for fat and therapeutic requirements.

One of the approaches to test preferences for a particular food has been taste testing. Taste testing has been used to demonstrate alterations in taste perception such as increased thresholds for recognition of a tastant, or lack of recognition of a tastant. The implications of finding a high threshold for a particular taste, for example, sucrose, is that a person might eat more foods containing sucrose in an effort to taste it, or add more/seek foods higher in sucrose. There are two studies which explore taste testing in persons with type 2 diabetes.

Perlmuter *et al.*, 1986 studied 110 women and 107 men with type 2 diabetes on insulin (43%), oral agent (24%), and diet alone (24%) and carried out taste

testing with six concentrations of a sucrose solution (0.031, 0.062, 0.125, 0.25, 0.50, and 1.00 M). They found that the diabetic women made significantly higher mean magnitude ratings of the six stimulus concentrations of sucrose than diabetic men did. Perlmuter also found that as the concentration of the sucrose solution increased, the hedonic rating of the diabetic women decreased, a pattern that was not seen in the diabetic men. Finally, depression ratings correlated with altered taste evaluations in the diabetic women. The findings of this study could be used to support the idea that overeating in type 2 diabetes is based on being deprived of preferred tastes such as sweetness. However, if the women in this study were obese, it is more likely that the preferred taste would have been a particular mixture of sweetness and richness obtained from fat, rather than sweetness alone. This phenomenon has been described by Drewnowski (1985) with obese nondiabetic women as the "sweet-fat tooth". There were no test solutions in this study which included fat.

More recently, taste testing at the time of diagnosis and 3-5 months later has been reported with twenty patients with type 2 diabetes. Detection and recognition thresholds for serial dilutions of glucose, fructose, sodium chloride and urea, as well as visual analogue scaling of taste perception was measured (Perros *et al.*, 1996). The electrical taste threshold was higher in the diabetic patients as compared to controls, as were detection thresholds for glucose, and recognition thresholds for glucose, and salt. At the time of the second taste testing several months later when

hyperglycemia was markedly improved, abnormalities in the detection and recognition thresholds for glucose, although improved in the diabetic patients, were still present. These studies suggest that women with type 2 diabetes might have abnormalities in detection and recognition of various tastes within the diabetic diet when blood glucose control is poor. If the diabetic diet does not contain enough preferred foods, it could be viewed as unpleasant, rather than pleasant. The approach to taste testing exemplified in these studies has some limitations. The most obvious one is that tasting various solutions does not mimic tasting actual foods. Actual foods, when chewed, initiate chemosensory interactions within the mouth, tongue, palate which trigger brain and gut responses, e.g., cephalic responses to ingestion of food (Mattes, 1997). In addition, detection and recognition of a particular taste does not necessarily mean that it is a preference. These studies suggest that diabetes has an effect on taste, that the effect is greater when blood glucose levels are not optimal, and even when blood glucose control is improved, the alteration persists.

Another approach has been used to identify pleasantness of food being ingested in a particular diet through paper and pencil measures such as visual analogue scaling on various aspects of the diet, e.g., texture, smell, taste. Two studies have been reported in the diabetes literature which use these measures to describe ratings of palatability and preference for foods in the HC diet (Campbell *et al.*, 1994; Laitenen *et al.*, 1991). Campbell found that men rated a high

carbohydrate (HC) diet as being more palatable than a diet enriched with high monounsaturated fatty acids (HM), although it is not clear from the report if this rating was significantly different from ratings of the HM diet or if subjects were blind to the diets. The men in this study also rated fullness (satiety) higher on HC than the HM diet, although significance, as before, is not indicated. The Laitenen study found that although behavior for the diabetic women and men in the study was congruent with the high carbohydrate (HC) diet prescription, preference and palatability for high fat foods persisted as long as three months, even though subjects were not ingesting these foods. Preference for particular foods is inferred through responses to taste testing, or through paper and pencil measures. No studies have been found which test whether women with type 2 diabetes prefer a diet high in monounsaturated fats more than one high in CHO.

Eating frequency and timing, e.g., eating pattern frequency, could also be related to an person's preference for time of day for activity. Two Swedish investigators who studied night and shift workers (Horne and Östberg, 1976; Östberg, 1973;) found that in a given population, some people will have distinct preferences for mornings or for evenings as judged by mean 24-hour maximum oral body temperatures and majority of food intake. Individuals with distinct preferences for mornings start food intake early, and have a maximum body temperature that peaks around 1300. Individuals with a distinct preference for evenings start food intake approximately 2 hours later, and have maximum body temperatures that are

slightly higher and occur around 1715. Some individuals have a mixed preference, falling somewhere between these two patterns. Horne and Östberg (1976) then developed a self-assessment questionnaire to determine morningness-eveningness (M-E) preference. This questionnaire has been tested with healthy English and non-English speaking populations and in persons with a chronic disease (Beal, D. 1994; Breithaupt *et al.*, 1978; Foret, *et al.*, 1982; Horne & Östberg, 1976;Mecacci & Zani, 1983). No studies have reported the M-E preferences nor have relationships between M-E preferences and eating frequency patterns as a component of eating behavior been explored in women with type 2 diabetes.

de Castro and colleagues have examined meal frequency and eating patterns, macronutrient intake, and hunger and satiety ratings in a variety of populations and differing circumstances (de Castro, 1987; de Castro & Elmore, 1988; de Castro, J. M. & de Castro, E. S., 1989; de Castro, J. M., 1993; de Castro, J. M., 1997). Food intake diaries are the major data collection tool; time periods vary from 4 - 9 days in length. In college aged students, significant differences between men and women were found in terms of total calories of intake, meal size and meal composition across the day. Males ate more than females each day, e.g., more CHO and fat in the morning, but their intake declined over the day, and it was more common for males to miss the evening meal. On the other hand, females ate less in the morning, but what they ate contained more CHO; females ate approximately equivalent amounts of CHO and fat in increasing intake (in terms of calories) in the later part of the day.

In studies which followed this one, de Castro found that when meals are eaten with others, the CHO, fat, protein and total calories were greater than when meals were eaten in solitude, and that genetics strongly influence total calories ingested and meal size (de Castro, 1987).

Eating frequency patterns have been examined in individuals with type 2 diabetes (Beebe *et al.*, 1990; Bertelsen, 1993; Jenkins *et al.*, 1992). The purpose of these studies was not, however, to determine the eating pattern of persons with type 2 diabetes. Rather, these studies tested whether varied patterns of eating frequency were deleterious to blood glucose and insulin levels. These studies, while responsible for liberalizing recommendations about meal frequency to patients with type 2 diabetes, have been conducted in testing situations which may have little relationship to the day-to-day actual eating frequency patterns of women with type 2 diabetes.

Other variables such as hunger and fullness (satiation, satiety) have been linked to eating frequency. Hunger ratings have been significantly correlated with meal size ($r = 0.15 - 0.27$), although this r value seems low (de Castro & Ellmore, 1988). To replicate this finding, hunger ratings, eating frequency, and energy intake were assessed in 12 lean and 12 normal weight, healthy men and women (Mattes, 1990). After instruction, subjects maintained diet records for 7 weekday and weekend days on standardized forms, indicating all food and beverage consumed during waking hours. Hunger was rated on a 9-point scale with the

anchors "not at all hungry" and "as hungry as I have ever been". The correlation between hunger ratings and intake during the same hour of each day for week days was $r = 0.50$, $p < 0.02$, but was weaker and not significant for the preceding hour ($r = 0.36$); significant correlations were not found for weekend days. There was no significant association between hunger ratings and energy content or number of the eating occurrences. Whether hunger and fullness ratings and eating frequency are interrelated with each other, remains to be confirmed.

Attitudes about food intake have been reported in persons with diabetes (Straub *et al.*, 1996). The Three-factor Eating Questionnaire (TFEQ)(Stunkard & Messick, 1985) was administered to men and women with type I and type II diabetes (25 female, 16 male). The three subscales in this instrument measure cognitive control of eating behavior, susceptibility to eating problems, and hunger. High scores in the cognitive control subscale indicate highly restrained eating with strict control of caloric intake; high scores in the susceptibility subcale indicate vulnerability to situational challenges to eating a prescribed diet; the greater the vulnerability, the more easily control of eating restraint is lost. High scores in the hunger subscale indicate responses to perceived hunger. The authors combined the scores for the cognitive control and susceptibility to eating to obtain an overall score reflective of loss of control; the scores on the hunger subscale were not reported. The type 2 patients cognitive control average score was 14.9 ± 0.7 (very high cognitive control) and the average score for susceptibility to eating problems was

3.4 ± 0.0.4 (very low susceptibility). In type 2 patients, significant correlations were found between the susceptibility to eating problems ($r = 0.40$, $p < 0.01$) and disinhibition of eating control ($r = 0.43$, <0.01) and BMI (27.6 ± 0.86, range: 20.2 - 48). These findings suggest that the higher the BMI, the higher the scores for susceptibility to eating problems and disinhibition. However, the scores of the type 2 diabetics in these subscales reflect high cognitive control and alertness to challenging situations. This could mean that social desirability influenced or biased the response to these subscales. The findings would have seemed more plausible if high scores for control in these type 2 diabetics had negatively correlated with lower BMIs, but this was not the case.

Knowledge about diet can also influence eating behavior. In a three-phase study, 17 healthy free-living, normal weight subjects were told that in one phase, they would receive a lunch that contained the same amount of fat they usually consumed; in the other two periods, they would receive a lunch containing more, or less than their typical fat intake (Caputo & Mattes, 1993). Each diet period of the study consisted of the 24 hours before, the day of the lunch, and 24 hours after the lunch was eaten. In each period, subjects received the same low calorie, low fat lunch (kcals, 310; CHO: 42 g, protein 21.3 g, and fat 6.3 g). Subjects kept food records for all meals eaten 24 hours before and 24 hours after each test meal day. After baseline, participants significantly reduced intake after the high fat period and there was a significant effect of the information on fat intake. Clearly, knowledge

influenced future intake, even when the knowledge was erroneous. In addition, it was clear that these subjects could not distinguish an actual high fat or low fat meal from the meal that was given to them, a situation where the sensory cues might have been dissonant with the information.

There are formidable barriers to studying eating behavior in persons with type 2 diabetes. The studies that have been reviewed have used artificial eating/feeding situations, short time periods such as hours before and after a meal or one 24 hour period, measurement tools that are vulnerable to subject under- or over-reporting, limited to no control over food consumed, and calculated versus actual knowledge of food composition of the meals. If eating behavior was studied where free-living subjects were in daily contact with and observation by study personnel, and where there is a moderately high degree of control of the food ingested with proximate analysis of the diet to confirm actual food composition, some of the barriers to studying eating behavior could be reduced. This report describes the eating behavior of women with type 2 diabetes while participating in an outpatient feeding study where a diet rich in monounsaturated fatty acids (HM) was compared to a diet high in carbohydrates (HC). We, as well as others, have shown such diets influence plasma lipids, lipoproteins, and glycemic control in a similar manner. (Diabetes Care in review).

Methods

The study was approved by the Oregon Health Sciences University (OHSU) and Oregon State University Institutional Review Boards and the Advisory Committee of the Center for Clinical Research at OHSU. Each subject gave informed consent.

Subjects

Subjects for the feeding study were recruited from attendees of primary care, internal medicine and diabetes clinics at Oregon Health Sciences University in Portland, Oregon. From an initial pool of 410 women, 255 (62%) were dropped in preliminary screening through chart audit. Of the 155 remaining, nearly half (42%) were screened in a two-visit procedure which included a lipid profile (total cholesterol, triglyceride, LDL-cholesterol, HDL-cholesterol concentrations), apolipoprotein E phenotype, random blood sugar, glycosylated hemoglobin (GHb), and BMI. It was established that potential subjects were able to read English, their diabetes regimen was combination therapy (oral agent plus insulin), and they had NIDDM based on case review by diabetes specialists using criteria described by Bingham and Riddle (1989).

Other inclusion criteria were 1) a mean of two fasting triglyceride values >250 but <600 mg/dL; 2) a mean of two LDL-cholesterol values <160 mg/dL; 3) apolipoprotein E phenotype other than 2/2 or 4/2; 4) BMI >25 but <55 kg/m^2; and

5) postmenopausal status (no menstrual period for at least 12 months). From the resulting pool of thirty-eight women, ten were selected and assigned to two groups of five persons each. There were no significant differences between these two groups at entry into the study in terms of BMI, mean entry triglycerides, total cholesterol, LDL-cholesterol, or glycosylated hemoglobin (GHb).

Cognitive function was assessed using the Neurobehavioral Cognitive Status Examination (NCSE) (Kiernan, *et al.*, 1987), and Trailmaking A and B tests (Reitan, 1955). The NCSE assesses components of cognition such as level of consciousness, attention, orientation, language, constructions, memory, calculation and reasoning. The Trail Making test (Adult version) assesses recognition of numbers and letters, ability to scan a page and identify the number or letter in a sequence, integration of a numerical and alphabetical series, and completion of a task under time pressure These tests represent function of the right and left cerebral hemispheres as well as reflecting general brain function (Reitan and Wolfson, 1993). Trails A normal values range from 0 - 39 seconds; Trails B normal values range from 0 - 85 seconds. Affective status was assessed using the Zung Self-Rated Depression Scale (Zung, W., 1965). Morningness-eveningness preference was assessed by the Morningness-Eveningness Questionnaire, a paper and pencil test which identifies an individual's preference for times of rising, wakefulness, and bedtime (Horne & Östberg, 1976). The 19 questions on the Morningness-Eveningness Questionaire ask the respondent about "feeling best" times to get up,

level of alertness when arising and preferred times for going to bed, performing physical exercise, handling hard physical work, and working. There is one question related to appetite which asks the respondent to indicate "...appetite during the first half-hour after wakening in the morning." The tool has external validity with peak times of oral body temperature. A high score (70-86) indicates a definite morning preference type (I); a score of 59-69 indicates a moderate morning preference (II); a score of 42-58 indicates neither a morning nor an evening preference type (III); a score of 31-41 indicates a moderate evening preference type (IV), and a score of 16-30 indicates a definite evening preference type (V). The NCSE, Trailmaking, Zung Self-rated Depression scale, and Morningness-Eveningness Questionnaire tests were administered to subjects once at baseline.

A control group of ten nondiabetic overweight women matched to the study subjects were recruited for taste testing from Oregon Health Science Center staff, visitors, and from the community via announcements in a campus weekly newsletter. There were no significant differences between the control group and study subjects in terms of age or BMI.

Study Procedures

This outpatient feeding study consisted of four, consecutive 6-week periods. The first group of five patients entered and completed the HM diet phase, the washout phase, and the HC diet phase. The second group of subjects entered the

HC diet phase, the washout phase, and finally, the HM diet phase. Initial caloric levels for each patient were determined through analysis of patients' prestudy 3-day food records (Food Processor, ESHA Research, Salem, Oregon), semiquantitative food frequency (DIETSYS, National Cancer Institute, Bethesda, MD), and physical activity recall (Mayer *et al.*, 1991). Patients were instructed in protocols for research diets, study procedures, capillary blood glucose testing, treatment of hypoglycemia, and daily and weekly record keeping related to the study. Patients were also instructed to maintain their usual eating pattern within each 24-hour period, and were free to chose to eat any food provided by the study at any time of the day. Patients were blind to the diet phases. They were paid at the end of the study for each day of the diet phase that was completed.

Each day, foods for the next 24 hours were prepared and packaged by the dietitians and staff of the Clinical Research Center, and delivered by study personnel to the subject's home in the evening. At the time of food delivery, the subject was weighed on a portable, electronic scale (Detecto, Webb City, MO); capillary blood glucose, record keeping, and other aspects of diabetes control were reviewed. Deviations from the study protocol such as missing or being late with diabetes medications, diet breaks such as eating something not on the research diet or eating something not considered "free" food, or uneaten food left in the patient's sack, were recorded by study personnel.

Diet

The HM diet was calculated to contain 45% of the total calories as carbohydrate (35% complex, 10% refined), and 40% as fat (10% saturated, 27% monounsaturated, 3% polyunsaturated fatty acids). The HC diet was calculated to contribute 55% of the total calories as carbohydrate (45% complex, 10% refined), and 30% as fat (10% saturated, 10% monounsaturated, 10% polyunsaturated fatty acids). Protein (15% of total calories), cholesterol (< 300 mg/day), and fiber intake (15 grams/day) were held constant in both diet periods. Core foods of the HM and HC diets were developed and taste-tested by Clinical Research Center staff, study personnel, and subjects; acceptability of food items was ascertained by asking subjects to indicate foods they did not like and would not eat. The core foods were adapted to deal with these responses. All but one food had similar appearance in both dietary periods. These core foods were incorporated into a four-day cycle of foods for each diet. An oil high in oleic acid concentration (TriSun©, Wycliffe, OH) was incorporated into the foods on the HM diet; a soybean and sunflower oil high in linoleic acid was incorporated into the HC diet. Subjects were allowed to drink coffee and tea ad libitum; non-calorie sodas and seltzers were provided on request. Two hundred (200) calorie increments and decrements of the HM and HC diet were created to correct for energy intake and weight changes that might occur during the study. Physical activity was kept constant. None of the subjects drank alcohol or smoked.

Adherence to the research diet was ascertained through information recorded on the subject's daily journal, through observation and record keeping of study personnel at the time of the daily visit, and through subject's weekly responses to questions on a 5-item paper and pencil instrument (adapted from Padilla, *et al.*, 1992). Subject's daily journals were reviewed by study personnel every day at the time of the visit. Blood samples for the fatty acid profile in low density lipoprotein (LDL) were also taken in the HM and HC dietary phases to evaluate a profile consistent with the particular fatty acids being consumed.

Eating Behavior

Eating frequency data was obtained at baseline and in the washout phase through food intake records which were kept for one weekend and two week days. Eating frequency patterns while in the HM and HC diet phases were assessed for two, four-day diet cycles, e.g., a total of 8 days, from the beginning of the fourth week through the middle of the fifth week of each diet phase. The time of each eating episode and the specific food(s) which were eaten in the episode for these 24-hour periods were recorded by the subject on a paper and pencil instrument (adapted from Reiff & Reiff, 1992). An eating episode was defined as any time a subject ingested food or drink which contained one or more calories. A meal was defined as an eating episode which occurred 30 minutes or more from any preceding eating episode and consisted of 50 calories or more; a snack was defined as an

eating episode which occurred 30 minutes or more from any preceding eating episode and consisted of <50 calories. All eating episodes within each 24-hour period were recorded, as was the time of day when eating began, and the time of day when eating ceased.

Palatability was measured three ways: 1) rating the global (overall) pleasantness of the day's food; 2) rating liking of each specific food they were ingested for each day of the four-day diet cycles during the HM and during the HC diet phase, and 3) tasting and rating liking of foods containing varying levels of fat at the end of each diet phase.

The global palatability 6-item paper and pencil test required subjects to reflect on all the foods eaten for the day, and, using a 10 cm visual analogue scale (VAS) scale with the anchors "very pleasant"(1) and "very unpleasant"(10), rate the day's foods for tastiness, texture, smell, appearance, richness and overall pleasantness. The specific food palatability instrument required the subject to indicate, on a 9-point Likert scale (1=like extremely; 5=neither/nor; 9=dislike extremely), how much they liked each major food provided for that day of the four-day diet cycle. Completed paper and pencil ratings for global and specific palatability were collected each day at the time of the study personnel visit. These assessments occurred at midpoint in each dietary phase for two, four-day diet cycles so that a total of eight consecutive days were evaluated. This particular time in the diet period was chosen because it was believed that the novelty of the research diet

would have disappeared, and routine-ness of the diet, if any, would not yet have appeared.

Palatability of a variety of foods containing varying levels of fat were evaluated using the methodology described by Mattes (1993) with the ten diabetic postmenopausal women in the feeding study and with a control group of ten nondiabetic postmenopausal women matched for age and BMI to the study subjects. For study subjects, two taste testing episodes occurred after the end of the HM and after the end of the HC dietary phases. For control subjects, the two taste testing sessions were approximately one month apart.

The first part of the taste testing involved tasting, savoring and rating samples of milk, chocolate milk, vanilla pudding and tomato soup, each containing five levels of fat. The milk was obtained from local grocery stores. Skim milk contained 0.18% fat by weight; whole, 3.3%; half and half, 11.5%; and whipping cream, 37.1% fat, respectively. The highest level of fat in the milk was 49.8%; this was made by adding and mixing 15.5 g vegetable oil with 61g of the whipping cream. The chocolate milk samples were created by adding 7.5 g sweetened chocolate milk powder (Hershey, Hershey, PA) to 61 g of each of the milk samples, resulting in the following fat levels: chocolate skim milk, 0.50% fat by weight; chocolate whole milk, 3.3%; chocolate half and half, 10.6%; chocolate whipping cream, 33.3%; chocolate whipping cream plus, 45.6% fat. The vanilla pudding samples were created by mixing 13 g pudding powder (Jello Pudding, General

Foods, White Plains, NY) with 61 g of the various milk samples, achieving the following fat levels: vanilla pudding skim, 0.26% fat by weight; vanilla pudding whole, 2.86%; vanilla pudding half and half, 9.58%; vanilla pudding whipping cream, 30.7%, and vanilla pudding whipping cream plus, 42.7% fat. Two drops of yellow food coloring were added to each sample to achieve uniformity of color amongst the samples. The tomato soup samples contained 60 g soup base (Campbell's Tomato Soup, Campbell Soup, Camden, NJ), 60 g of the various milk samples, and achieved the following fat levels: tomato soup skim milk, 0.9% fat by weight; tomato soup whole 2.47%; tomato soup half and half, 6.61%; tomato soup whipping cream, 19.6%; tomato soup whipping cream plus, 28.8% fat. One drop of yellow and one drop of red food coloring was added to the samples to achieve uniformity of color. All samples were rated for creaminess, oiliness, sweetness, saltiness, sourness, bitterness, and pleasantness on a 9-point scale (1 =like extremely, 5 - neither like nor dislike; 9 = dislike extremely). All samples had a volume of 10 ml and had been kept at room temperature for one hour prior to the testing.

At the beginning of each tasting session, subjects and the controls were weighed, and current medications and health status verified. At the first tasting session, instruction in taste testing and a trial with two ice cream samples (one, frozen yogurt and one, regular ice cream) were conducted to orient the subject to tasting procedures, the rating form, and to answer questions. The milk, chocolate,

vanilla pudding and tomato soup samples were presented in the first portion of the testing session in random order. After tasting and savoring, the subjects and controls wrote a single rating on a paper and pencil instrument for that sample only, and then rinsed and expectorated using deionized water. The next sample and rating form were then presented.

The second part of the testing included tasting 10 items obtained from local grocery stores in regular and reduced fat form (margarine, American cheese, cottage cheese, cream cheese, Monterey Jack cheese, pound cake, soda crackers, Italian salad dressing, French salad dressing, and mayonnaise). The reduced fat foods contained an average of 3.5% fat (range: 0 - 9%); the regular fat foods contained an average of 15% fat (range: 4 - 25%). Regular and reduced-sugar jam, and regular and reduced-salt tomato juice were tasted and contained 2% and 5% sugar, and 95 mg and 430 mgs sodium, respectively. These foods indicated whether aspects of taste such as sourness, bitterness, and saltiness were functional. These last food items were tasted and rated in the same manner as all the others, using the 9-point Likert rating scale.

Motivation to eat was assessed through a 6-item instrument and adapted from Blundell, *et al.*, 1988). Using 10 cm horizontal visual analogue scales, the subject indicated the time of day, the desire to eat (very strong, very weak); how much food could be eaten (a large amount, nothing at all); and considering being on a research diet, the amount of food that could be eaten (more than is available, just

what is available, very little of what is available). This assessment was conducted at midpoint of the HM or HC dietary phase for a total of 8 days.

To explore whether ideas about the diet were changed while in the HM and the HC phases, subjects were asked on a weekly basis to indicate the degree to which they thought the study diet was helping their diabetes, using a 10 cm VAS with anchors of "a great deal" and "very little". This question was included in a 5-item weekly compliance instrument that was collected at the end of each week in the HM and HC dietary phase (adapted from Padilla, *et al.*, 1983).

Hunger and fullness was rated at midpoint of HM and HC dietary phases for two, four-day cycles. At every eating episode, subjects rated hunger (1+=very little hunger;+2=some hunger, +3 = great hunger; +4=very great hunger; +5 = completely hungry; 0 = neither hungry nor full;) and fullness (-1 = very little fullness; -2 = some fullness; -3 = great fullness; -4 = very great fullness; -5 = complete fullness) on a paper and pencil instrument (adapted from Reiff & Reiff, 1992). There are no known psychometric properties of this instrument.

Statistical Analyses

Descriptive statistics for all variables were obtained; results are expressed as means ± SD or SE as indicated. Three statistical evaluations were made: the relationship between Morningness-Eveningness preferences and when eating started and ceased, palatability of the HM diet compared to the HC diet; and the

identification of similarities in taste testing in study subjects and a weight-matched control group of nondiabetic women. Pearson's correlations were used to determine if there was a relationship between the Morningness-Eveningness preference score and start and stop times for eating. Effect of the HM or HC diet on taste palatability of the study subjects was obtained by comparing means in paired t-tests to identify if there were significant differences.

When comparing the diabetic versus control subjects, the palatability rating scores for the seven sensory characteristics (creaminess, oiliness, sweetness, saltiness, sourness, bitterness, pleasantness) of skim and white milk, chocolate milk made with skim and whole milk, vanilla pudding made with skim and whole milk, and tomato soup made skim and whole milk were combined to form a low fat variable. Then, the palatability rating score for the seven sensory characteristics (creaminess, oiliness, sweetness, saltiness, sourness, bitterness, pleasantness) for milk, chocolate milk, vanilla pudding, and tomato soup made with whipping cream and whipping cream plus oil were combined to form a high fat variable. The low- and high-fat variables were used in independent t-tests to determine if the ratings of the seven sensory characteristics were different between the type 2 diabetic subjects in the study and the control group of nondiabetic women. Ratings of the seven sensory characteristics for low- and high-fat foods, e.g., various cheeses, crackers, cake, salad dressings, strawberry jam, and tomato juice, were used in independent t-tests to see if there were differences in ratings between the type 2 diabetic subjects

in the study and the control group of nondiabetic women. Frequency and intensity of hunger and fullness was analyzed using procedures described by Kazdin (1992), and by descriptive statistics. In all analyses, where assumptions of normality were not met, nonparametric statistics such as Wilcoxon signed ranks test and Mann Whitney U were used. The $p \leq 0.05$ level of significance was used to assess statistical significance. Analyses were conducted using the CRUNCH statistical package (CRUNCH Software Corporation, Oakland, California).

Results

Baseline Characteristics of Subjects

Clinical characteristics of the subjects at baseline are given in Table 1. These women had had diabetes for at least a decade, and, given their glycosylated hemoglobin levels, had been in poor glycemic control. Two were depressed, as measured by the Zung Self-rated Depression scale. Six of the 10 subjects had cognitive function test results which indicated mild to moderate impairments. This characteristic was most likely related to poor glycemic control at entry into the study and may have also influenced some of the ratings of hunger, neither-hunger-nor-fullness, and fullness. Three subjects had morningness preference; one had eveningness preference, and six had neither morning nor evening preference.

TABLE 1

Baseline characteristics of subjects (n=10)
(Means ± SE)

Characteristic		Values
Age		56.9 ± 2.8
Years since diagnosis of diabetes		11.5 ± 1.8
Weight (kg)		101 ± 6.0
Body mass index (kg/m^2)		40.1 ± 2.0
Waist:hip ratio		0.92 ± 0.0
Total cholesterol (mmol/L)		5.53 ± 0.3
Triglycerides (mmol/L)		3.59 ± 0.4
Glycosylated hemoglobin (%)		12.3 ± 0.7
Neurobehavioral Cognitive Status Exam		
*Consciousness, orientation, attention, language, comprehension, repetition, naming, similarities, judgment:	Normal	n=10
*Construction	Normal	n=8
	Moderate impairment	n=2

TABLE 1 (Continued)

*Memory	Normal	n=7
	Mild impairment	n=2
	Moderate impairment	n=1
*Calculation	Normal	n=9
	Mild impairment	n=1
Trails Test		
A (average seconds ± SE)	40.90 ±5.59	
	Normal	n=6
	Mild/moderate impairment	n=1
	Moderate/severe impairment	n=3
B (average seconds ± SE)	115.20 ±14.88	
	Normal	n=4
	Mild/moderate impairment	n=3
	Moderate/severe impairment	n=3
Zung Self Rated Depression		
	Normal	n=8
	Mild depression	n=2
Morningness-Evening Preference		
	Definite morning	n=2
	Moderate morning	n=1
	Neither morning/evening	n=6
	Moderate evening	n=1
	Definite evening	n=0

Weight and Calories

Body weight of the subjects at the start and by the end of the two dietary periods was not significantly different (HM: 102.2 ±5.5 and 101.1 ±5.6 kgs; HC: 103.9 ±5.8 and 101.5 ±5.5 kgs). There was no significant difference in the average kcals consumed on the two diets (HM: 2700 ±166; HC: 2620 ±125 kcals, p=0.14).

Adherence

All ten subjects completed the study. Subjects' daily journals indicated that one-half (50%) experienced minor health-related events while consuming the HM and 40% while consuming the HC diet. None of these events compromised study participation. Daily journals also revealed an average of 3.7 and 3.9 deviations from the diet, e.g., leaving uneaten food or eating something not on the research diet while consuming the HM and HC diets, respectively. In both dietary periods, responses to the question "How often have you been able to eat just the food provided by the study?" and "A person has to be pretty creative to make study food taste better" clustered below 3.0 where 1=always and 10=never. This indicates a high degree of self-reported adherence with the research study diets and protocols. Daily records maintained by personnel conducting the study regarding deviations from study protocol concurred with subject journals. The anticipated increase in the mean area percent oleic acid concentration of LDL occurred when the HM diet was

consumed (HM: initial 15.7 ± 0.50 versus final 23.9 ± 0.80, p 0.000; HC: initial 16.6 ± 0.73 versus final 15.5 ± 0.32, p 0.07).

Eating Frequency Pattern

No significant differences were found when comparing average number of meals, number of snacks, or total number of eating episodes during waking hours in the baseline, HM, HC, or washout phases (Table 2). In addition, eating start and eating stop time of day did not change, nor did eating episode length when comparing baseline with washout, HM with HC. Eating pattern frequency was stable even when there were changes in diet composition.

To see if any relationships existed between temporal preference, expressed as Morningness-Eveningness (M-E) type, and the time when eating started and stopped in the various study phases, Pearson's correlations were performed (Table 3). The only statistically significant correlation between M-E type, diet phase and eating start time was in the HC diet phase where $r = 0.71$, $p = 0.05$. A trend toward significance was also seen between M-E type and eating start time in the washout phase ($r = 0.63$, $p = 0.09$). This finding means that when on the HC diet, the higher the number of the M-E category type, the more likely eating started later in the day, a finding consonent with the definitions of the various M-E preference types. Pearson's correlations with r's ranging from 0.72 to 0.82 were found between M-E

TABLE 2

Average number of meals, snacks, and total eating episodes in various study diet phases (n=10; means ± SD)

Eating episodes	Baseline	Washout	p	HM	HC	p
Number of meals	4.47 ± 0.29	4.86 ± 0.59	0.36	4.94 ± 0.4	4.60 ± 0.28	0.95
Number of snacks	1.20 ± 0.40	1.15 ±0.42	0.69	0.71±0.30	1.00 ± 0.43	0.40
Total eating episodes	5.78 ± 0.60	5.63 ± 0.76	0.18	5.64 ± 0.55	5.42 ± 0.54	0.16
Eating period start*	0747±178	0742±193	0.83	0642±277	0646±150	0.33
Eating period stop*	2002±471	1932±538	0.16	2141±154	2209±223	0.08
Eating period length (hrs/mins)						
	14:23 ±45	13:20 ±43	0.18	14:05 ±2:25	15:15 ±1:11	0.16

* = military time

TABLE 3

Relationship between morningness-eveningness preference category and times of starting and stopping eating, all study phases (n=8)

	Baseline		HM		Washout		HC	
	Start eating	**Stop eating**	**Start eating**	**Stop eating**	**Start eating**	**Stop eating**	**Start eating**	**Stop eating**
Morningness-Eveningness Preference Type								
r	0.57	0.72	0.59	0.82	0.63	0.59	0.71	0.82
p	0.14	0.04*	0.12	0.01*	0.09	0.13	0.05*	0.01*

* $p \pm 0.05$

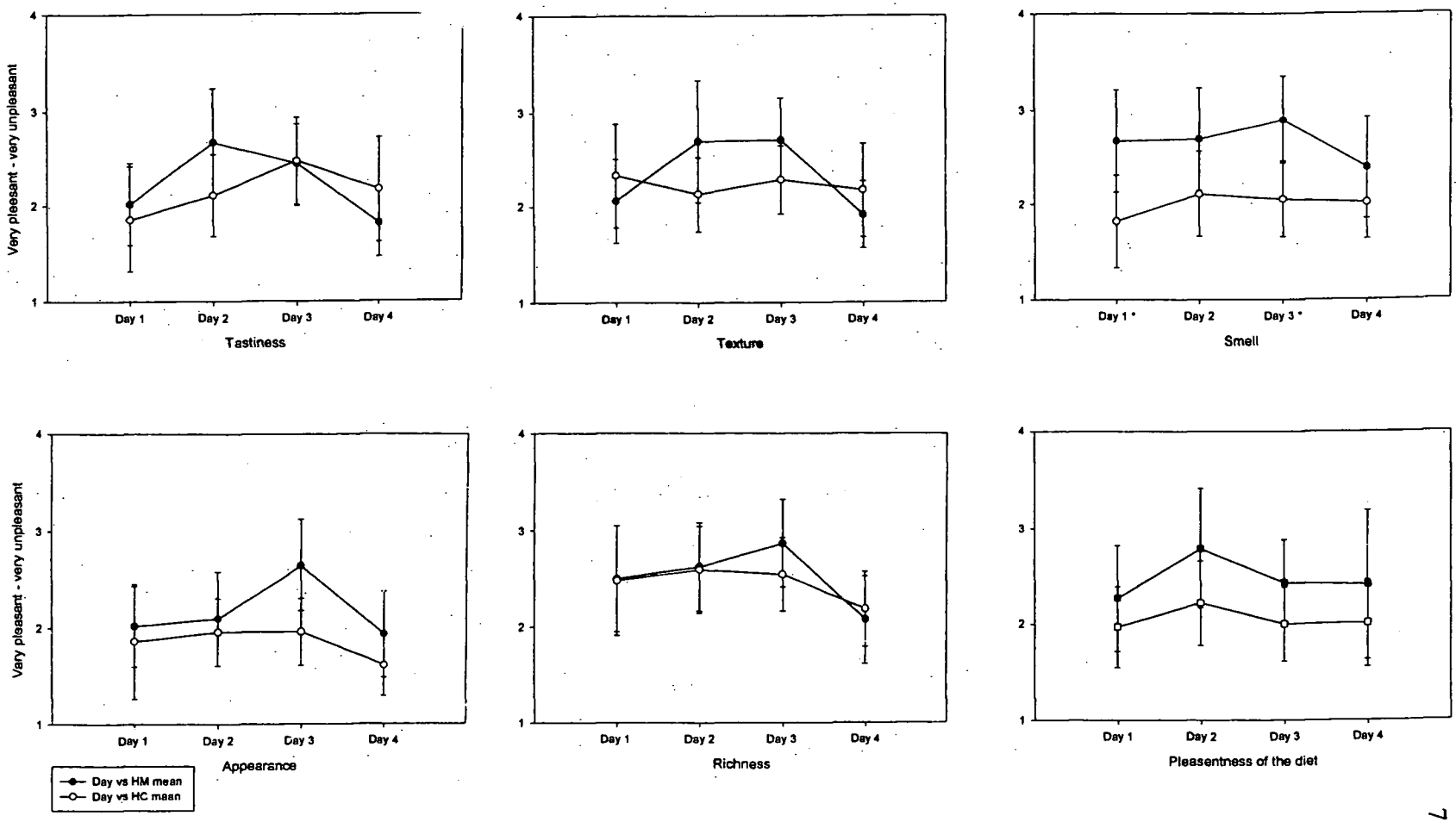
type and the time when eating stopped in the HM, washout, and HC phase. These correlations also suggest that the higher the number of the M-E category type, the later in the day eating will stop. It could be that time of day when eating starts and stops may be an eating frequency trait related to temporal preference.

Palatability.

The global rating of palatability revealed some significant differences between the HM and HC diet (Figure 5). Ratings differed significantly for smell on Day 1 of the four-day diet cycle (HM 2.67±0.54 vs HC 1.82±0.49; $p = 0.04$) and Day 3 of the four-day diet cycle (HM 2.89±0.46 vs HC 2.05±0.40; $p = 0.008$), with the rating being higher (less pleasant) for HM on both days. The foods that were offered on day 1 of the diet cycle included sausage with gravy over a biscuit or English muffin, juice, a tuna salad sandwich, fresh vegetables, marinated pasta salad, fruit, green salad with dressing, spaghetti with marinara sauce, bread and a pumpkin bar. Foods included on day 3 of the diet cycle included hashbrown quiche, juice, broccoli soup, saltines, canned vegetable, Mexican corn muffin, slice of cheese, fruit, green salad with dressing, calzone with marinara sauce, bread, and an oatmeal cookie. One or more of these foods such as the tuna salad or, potentially, the broccoli soup may have been the cause of these ratings related to smell; it seems unlikely that it was due to the Trisun oil in the food because the product literature indicates that this oil is odorless and tasteless. All other ratings of palatability such

FIGURE 5

Ratings of global palatability of the HM and HC diets (n=10)(means ± SE)



as tastiness, texture, appearance and richness for both diets varied from a low of about 1.80 (HM 1.80-tastiness, Day 4; HC 1.61-appearance, Day 4) to a high of 2.90 (HM 2.89-smell, Day 4; HC 2.59, richness, Day 3). Nearly all of these global ratings, however, are located at the "very pleasant" end of the VAS. It is possible that the diets were actually very pleasant to the subjects, response set bias is occurring, or the paper and pencil instrument is not sufficiently refined to be able to detect small changes.

Palatability ratings of specific foods showed that the foods provided in the HC diet scored less well than those provided on the HM diet, although all the ratings for palatability of specific foods were less than 4 on a 9-point scale (1=like extremely; 9=dislike extremely). On Day 2, the average palatability rating of canned or fresh fruit was significantly different for HM (1.39±0.16) vs HC (1.65±0.13), $p=0.04$. On Day 4, two specific foods, the scalloped potato dish and the vanilla pudding pie, were rated significantly different. The palatability rating of the potato dish on the HM diet was 1.00±0.0 vs the HC rating 1.45±0.20, $p=0.04$; the pudding pie was rated on HM 1.05±0.50 vs HC 1.90±0.29, $p=0.03$. The HM version of the potato dish was crispier whereas the HC version of the potato dish did not exhibit this characteristic. The pudding pie differed in its appearance between HM and HC diets, the only specific food for which this was the case. On the HM diet, this specific food was presented as a small tart with crumb crust and pudding filling. On the HC diet, it was presented as a pudding with a whipped topping. However, these

differences in palatability of specific foods were minor and the ratings, as with global palatability, were clustered at the "like" end of the scale. In this study, both HM and HC diets were rated the same and were rated as being highly palatable. This finding was unexpected because we hypothesized that subjects would rate the HC diet as less palatable than the HM diet because it contained less fat as a total percent of calories.

Palatability of Foods Containing Varying Levels of Fat and Differing Hedonic Characteristics by Subjects at the end of HM and HC Diets

There were significant differences when comparing taste testing responses for the period after HM with the period after HC (Table 4). While significant, these differences include various foods, different levels of fat, and different sensory characteristics for the foods, and no pattern linking the findings is found.

Palatability of Foods Containing Varying Levels of Fat and Hedonic Characteristics in Diabetics and Nondiabetics

Significant differences were found when comparing taste testing ratings of diabetic subjects to those of a control group of nondiabetic, weight and age-matched subjects. This difference is illustrated by the ratings of American cheese. Diabetic women significantly liked American cheese in the high fat form for the hedonic characteristics of creaminess, oiliness, sweetness, and pleasantness whereas nondiabetic women liked it less on these same hedonic characteristics (Figure 6).

TABLE 4

Palatability and liking for sensory characteristics of low and high-fat foods at the end of HM and HC diet phases (n=10; means ± SD) (1=like extremely; 5 = neither like nor dislike; 9 = dislike extremely)

	Fat level	HM	HC	p value
		Creaminess		
Whole milk	3.34%	3.40 ± 2.17	4.40 ± 2.17	0.009
Italian dressing	16%	4.90 ± 2.13	3.60 ± 2.12	0.02
		Oiliness		
Chocolate milk	49.8%	4.50 ± 1.08	3.60 ± 1.08	0.04
Pound cake	25%	4.60 ± 1.35	3.20 ± 1.48	0.03
		Sweetness		
Skim milk	0.18%	4.80 ± 0.42	4.00 ± 0.82	0.01
Margarine	17%	3.80 ± 1.48	4.80 ± 0.63	0.04
		Pleasantness		
Vanilla pudding	42.7%	2.70 ± 1.70	3.80± 1.99	0.05
Pound cake	25%	3.00 ± 1.05	2.10 ± 0.99	0.04

TABLE 4 (Continued)

		Saltiness		
Cottage cheese	2%	4.60 ± 1.17	3.90 ± 1.45	0.04
Soda crackers	14%	2.60 ± 0.70	4.40 ± 2.50	0.05
French dressing	0%	3.80 ± 1.62	3.10 ± 1.85	0.04
French dressing	18%	4.00 ± 2.11	2.90 ± 1.37	0.04
V8 Vegetable juice*		3.40 ± 1.17	5.00 ± 1.63	0.02
		Sourness		
Italian dressing	16%	4.00 ± 2.26	3.10 ± 1.60	0.05
French dressing	0%	3.80 ± 1.55	3.10 ± 1.45	0.04

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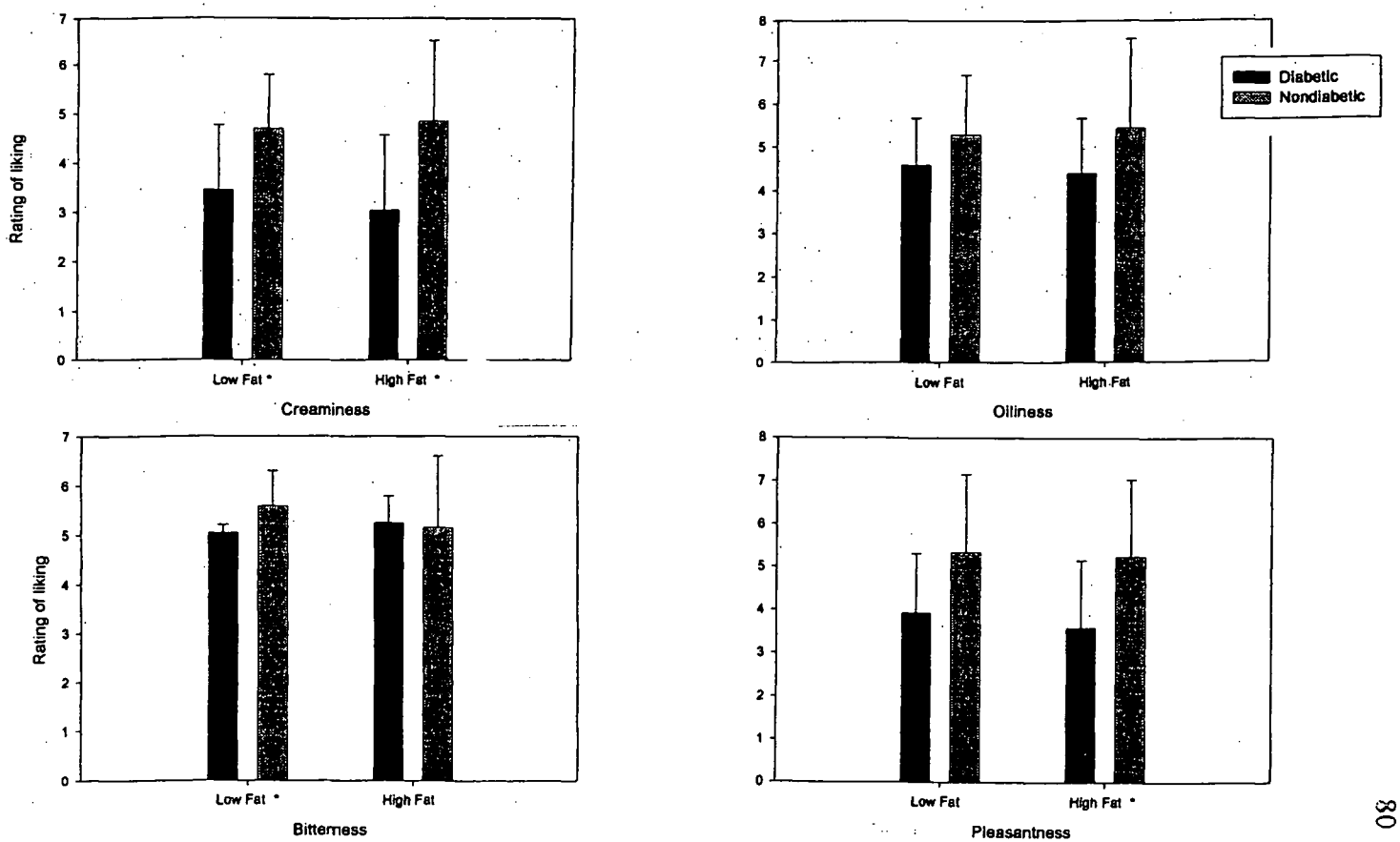
* = regular sodium level (430 mg)

FIGURE 6

Ratings of liking of selected sensory characteristics of American cheese by diabetic and nondiabetic women (n=10) (means ± SE) (* p = ≤ 0.05)



A different pattern emerged where diabetic women liked foods with the hedonic characteristics of saltiness, sourness, and bitterness significantly less than nondiabetic women did, with these significant differences about equally divided between low- and high-fat food samples (Table 5). In tasting these foods, diabetic women rated the saltiness and the sourness of selected foods as closer to the "neither like nor dislike" category than nondiabetic women whose liking for these characteristics was closer to "like very much" or "like moderately".

We thought that, if any differences were found, it would show that diabetic and nondiabetic women would like and rate as very pleasant samples that had hedonic characteristics of creaminess, oiliness, and sweetness because these characteristics are known to be pleasant to obese women. This hypothesis was only partially support by the liking and pleasantness ratings of both diabetic and nondiabetic subjects for high fat samples that are creamy, oily, and sweet. However, the significant differences we found for liking of saltiness, sourness, and bitterness between diabetics and nondiabetic women were unexpected.

Motivation to Eat at a Meal.

Responses to questions regarding strength of desire to eat, degree of food that could be eaten, and degree of food that could be eaten under the conditions of a feeding study, showed no significant differences in the HM and HC phases.

TABLE 5

Palatability and liking for sensory characteristics of low and high fat foods in diabetic and nondiabetic women (n=20) (1=like extremely; 5 = neither like nor dislike; 9 = dislike extremely)

Food	Low fat sample			High fat sample		
	Diabetic	Nondiabetic	p value	Diabetic	Nondiabetic	p value
			Creaminess			
Chocolate milk	3.65±1.33	4.05±1.26	0.50	1.65±0.68	2.68±1.45	0.07
Vanilla pudding	2.25±0.09	1.35±	0.22	2.78±1.41	4.65±2.34	0.04
Saltines	3.60±1.71	4.55±0.76	0.13	3.35±1.63	4.45±0.96	0.08
			Oiliness			
Chocolate milk	4.80±0.82	5.03±0.66	0.31	4.98±1.48	6.27±1.57	0.07
Pound cake	4.35±0.78	4.30±1.49	0.09	4.25±1.03	4.05±1.19	0.69
			Sweetness			
Italian dressing	4.20±1.81	3.85±1.56	0.65	5.15±1.49	3.80±1.57	0.06
Mayonnaise	3.45±1.36	2.50±0.82	0.08	3.50±1.78	2.55±0.96	0.15
Strawberry jam	3.45±1.36	2.50±0.82	0.08	3.50±1.78	2.55±0.96	0.15

TABLE 5 (Continued)

Food	Low fat sample			High fat sample		
	Diabetic	Nondiabetic	p value	Diabetic	Nondiabetic	p value
			Pleasantness			
Chocolate milk	3.45±1.28	4.13±1.37	0.27	1.85±0.74	2.85±1.42	0.06
Vanilla pudding	2.73±0.95	3.20±0.80	0.80	3.13±1.27	4.60±2.80	0.07
Margarine	4.10±2.26	4.65±1.08	0.50	4.25±1.85	5.85±2.14	0.09
			Saltiness			
Cream cheese	4.10±1.31	3.15±1.18	0.11	4.50±1.45	3.05±1.42	0.04
Cottage cheese	4.15±1.49	4.25±1.16	0.87	4.60±1.24	3.40±1.13	0.04
Italian dressing	4.45±1.85	3.15±1.47	0.10	4.70±1.49	2.90±1.39	0.01
French dressing	4.85±1.08	3.10±1.24	0.003	4.55±1.61	3.50±1.33	0.13
			Sourness			
Cream cheese	4.35±1.29	3.60±1.71	0.28	5.00±1.65	3.05±1.50	0.01
Italian dressing	5.20±1.65	3.30±1.89	0.03	5.25±1.65	2.80±1.59	0.003
French dressing	4.70±0.59	3.25±1.28	0.004	5.05±1.94	3.35±2.07	0.07
Mayonnaise	5.05±0.16	4.40±1.08	0.19	5.00±0.24	4.25±1.01	0.03
Strawberry jam*	5.05±0.16	4.40±1.08	0.19	5.00±0.24	4.25±1.01	0.03

TABLE 5 (Continued)

	Low fat sample			High fat sample		
Food	Diabetic	Nondiabetic	p value	Diabetic	Nondiabetic	p value
			Bitterness			
V8 juice**	6.55±1.38	5.20±1.34	0.05	5.30±0.72	4.90±0.74	0.53

(* = low versus high sugar content; ** = low versus high sodium content)

Change in Ideas about the Diet.

Each week, subjects responded to the statement "The diet and foods in it are helping my diabetes" using a VAS with the anchors "a great deal" (1) and "very little" (10). There were no significant differences in the ratings to this question across the 6-weeks of the HM and the HC phases. Ratings in week 1 on the HM diet began at 2.24 ±0.84 and reached their lowest level, 1.22 ±0.41 in week 6. The ratings in the HC diet began at 1.87 ±0.60 in week 1, reached their lowest in week 4, 1.03 ±0.34, and ended at 1.15 ±0.37 in week 6. Overall, ratings are located at the "a great deal" end of the VAS scale. This question did not appear to detect shifts in beliefs regarding the diet.

Hunger and Fullness Sensations.

Frequency of hunger sensations, fullness sensations, and neither hunger nor fullness sensation ratings were identified in each subject for the HM and HC diet phases (Table 6). On HM during the recording period, subjects experienced an average of 26.3 ± 7.32 hunger sensations, 39.5 ± 12.43 fullness sensations, and 15 ± 3.54 neither-hunger-nor-full-sensations. On HC, subjects experienced an average of 3.5 ± 8.58 hunger sensations, 55.7 ± 23.8 fullness sensations, and 13.3 ± 3.04 neither-hunger-nor-fullness sensations. There were no statistically significant differences in the frequency of these sensations when comparing the recording period in HM to the recording period in HC.

TABLE 6

Summary of episodes of hunger, fullness, and neither-hunger-nor-fullness in HM and HC diet phases (n = 10; frequency and % for each recording period)

Subject	All hunger	All fullness	All neither hun- ger nor fullness	All hunger	All fullness	All neither hun- ger nor fullness
S 010	13 (38%)	21 (62%)	0 (0%)	16 (21%)	54 (72%)	5 (6%)
S 226	6 (7%)	76 (82%)	11 (12%)	3 (4%)	87 (96%)	1 (1%)
S 201	59 (47%)	64 (50%)	4 (3%)	70 (52%)	59 (43%)	7 (5%)
S 151	28 (51%)	22 (40%)	5 (9%)	77 (73%)	16 (16%)	13 (12%)
S 259	3 (7%)	25 (54%)	18 (39%)	15 (14%)	61 (57%)	31 (29%)
S 356	13 (28%)	15 (31%)	20 (42%)	77 (75%)	16 (16%)	13 (13%)
S 209	20 (21%)	38 (40%)	38 (40%)	25 (32%)	28 (36%)	25 (32%)
S 413	12 (35%)	0 (0%)	22 (65%)	69 (82%)	7 (8%)	8 (10%)
S 324	37 (12%)	128 (73%)	11 (6%)	2 (1%)	251 (97%)	7 (3%)
S 013	72 (73%)	6 (7%)	21 (21%)	37 (51%)	16 (22%)	20 (27%)
Sum	263	395	150	350	557	133
Mean ± SD	26.3 ±7.32	39.5 ± 12.4	15 ± 3.54	3.5 ±8.58	55.7 ± 23.8	13.3 ± 3.04

The intensity of hunger and fullness as experienced in HM and HC is depicted in Figure 7. The most common intensity of hunger was +2 (some hunger) in both HM and HC; the most common intensity of fullness was -4 (very great fullness) on HM and -5 (complete fullness) on HC. There were no statistically significant differences in the intensity of these sensations when comparing HM and HC.

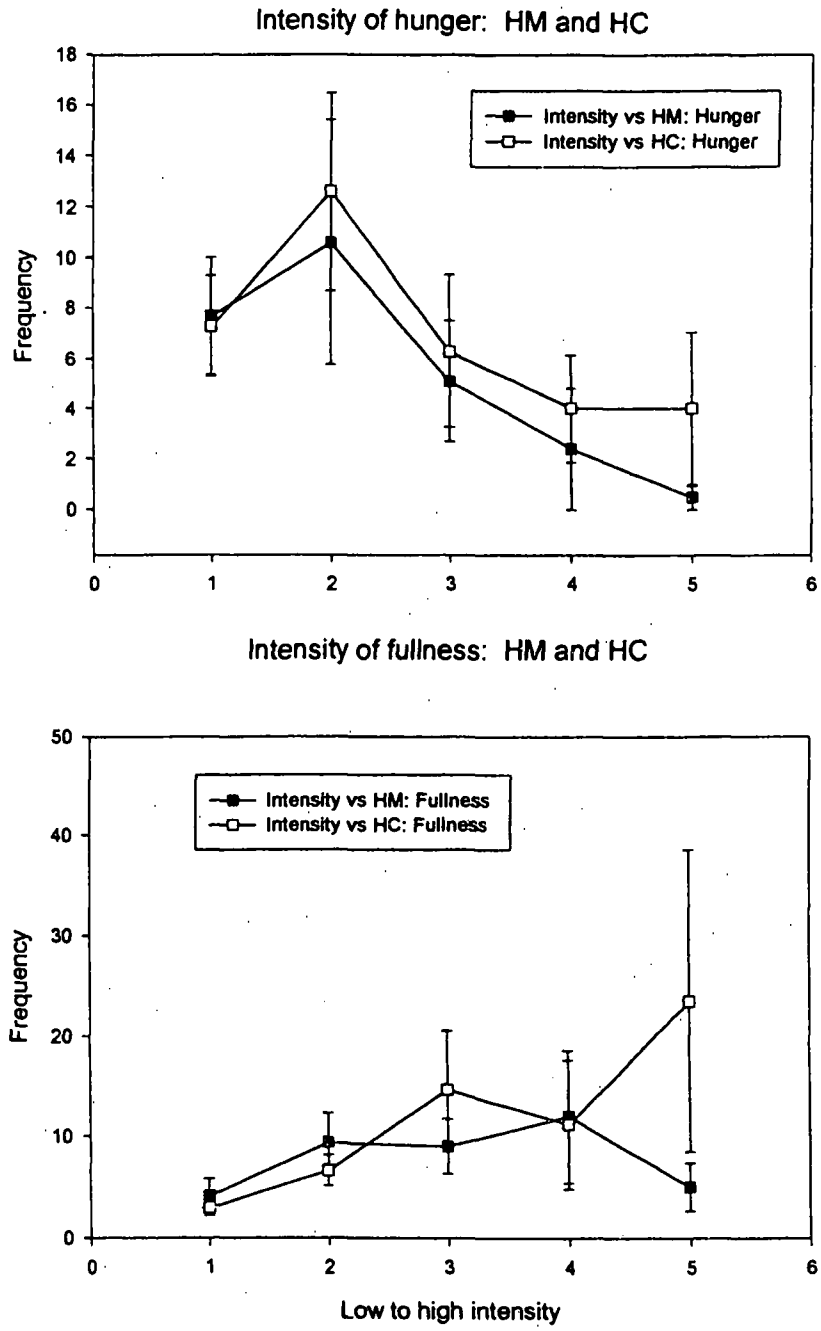
Pearsons correlations were performed to see if frequency of hunger, fullness, neither-hunger-nor fullness, intensity (magnitude) of hunger and fullness and meals, snacks, and total eating episodes in HM and HC were related. Several significant relationships in HC were found. The first relationship showed that +2 hunger and +3 hunger positively correlated with the number of snacks ($r = 0.71$ and 0.85; $p = 0.02$ and $p=0.002$, respectively) and total number of eating episodes in HC ($r = 0.67 - 0.77$, $p=0.01$ and $p=0.03$), suggesting that the condition of +2 or +3 hunger influenced snacks and total number of eating episodes in the HC diet phase.

Discussion

This feeding study provided opportunities to observe eating behavior of women with type 2 diabetes over a period of time and under the long-term impact of an diet enriched with monounsaturated fatty acids and a diet high in carbohydrate. The literature suggests that eating behavior variables which might have varied as a result of one or the other diet include ratings of palatability and pleasantness of the food, motivation to eat, and ratings of hunger and fullness. These variables were

FIGURE 7

Intensity of hunger and fullness: HM and HC diet phases (n = 10) (means ± SE)



measured through paper and pencil instruments for an 8-day period, midcycle in each diet phase.

In this study, both diets were rated as very palatable based on ratings of global and specific food palatability. Our findings differ from the men in Campbell's study (1994) who rated the HC diet as being more palatable than a HM diet, and who found that satiety was greater on the HC diet. It could be that the differences in rating the HM diet between
Campbell's subjects and ours are related to gender, or potentially, the particular foods included in the diets. In our work, subjects tasted and rated most of the study foods before the study began, in an effort to eliminate foods that were unacceptable, and adapt those which were found to be unpleasant; it is unclear if this happened in Campbell's study.

Another explanation for the equivalent rating of the HM and HC diets even though they differed in terms of composition is that these subjects had some undetected alteration in taste function for various foods, or possibly, one or more of the medications being taken on a daily basis in addition to insulin and oral agent, affected taste. These subjects were taking an average of six medications, in addition to insulin and an oral agent; several of these drugs alter taste, e.g., drugs in the categories of ACE inhibitors, antidepressants, beta-adrenergic blockers, nonsteroidal analgesics (United States Pharmacopeial Convention, 1995). Additionally, if our subjects had been sensitive to bitter tastes, a heritable trait which is more common in

women, they might have found the HC diet less palatable due to the inclusion of more fruits and vegetables which contain compounds that are tasted as bitter (Drewnowski, 1997; Bartoshuk, *et al.*, 1994). Testing for sensitivity to bitter taste may be an important assessment to include in evaluating taste function and palatability related to the diet of women with type 2 diabetes.

The taste testing in this study was conducted to answer whether these subjects preferred fat and if they did, whether they would find the HM diet more palatable. This line of inquiry did not reveal significant differences in the subjects at the end of HM and HC, even though there was a difference of 10% percent fat of total calories between HM and the HC diets. Having received one or the other diet for a six-week period did not alter the taste testing responses. It could be that the amount of time was not long enough to change fat preferences, although the HM and HC diet phases in this study contained fat levels that were well above those of the Mattes (1993) study.

The women with diabetes and the nondiabetic women liked the creaminess, oiliness and pleasantness of the high-fat form of chocolate milk more than they liked the low fat form of this food. However, both diabetic and nondiabetic women liked the creaminess, oiliness and pleasantness of vanilla pudding in the low-fat form more than the high-fat form. This finding suggests that the fat level was not the

only contributing factor to the ratings of liking; rather, some combination of other variables within these foods or within the mouth, tongue, and throat lead to the rating of liking.

A pattern can be seen in the ratings of foods in the taste testing for liking of the various hedonic characteristics. For creaminess and pleasantness, diabetics liked these characteristics in the low- or high-fat foods more than the nondiabetic women did. For oiliness, no pattern can be seen. For sweetness, nondiabetics had greater liking of both low- and high-fat foods than diabetics did, a finding that seems similar to the response of women in the Perlmuter study (1986). It could be that sensitivity to 6-N-propylthiouracil, cognitive levels, or some other aspect of ingestive behavior in type 2 diabetes accounts for these differences.

Taste testing in women with diabetes does have some consequences that need to be considered. Each woman with type 2 diabetes experienced a period of elevated capillary blood glucose 24 hours after testing; a plan for increased intake of fluids and other appropriate measures should be included if testing is done. Finally, there is a linkage that has to be made between a particular food rated as being liked on taste testing, and the consistent and frequent inclusion of this food in the day-to-day diet as representing a preference. This linkage was not established in this study.

Each subject's hunger and fullness responses to HM or HC were evaluated for patterns. On the individual level, only 4 (40%) of the subjects responded

similarly in the HM and the HC diet phases. Of these four, two subjects had the main sensation of fullness in HM and HC; one subject's main sensations were fullness and neither-hunger-nor-fullness, and the fourth subject's main sensations were hunger and fullness. No clear picture emerges in these women with type 2 diabetes with respect to their hunger and fullness experience, either within one or the other diet phase, or from one diet phase to another. As measured in this study, we are unable to say that the HM or HC diets has any direct effect on hunger or fullness. Indirectly, both diets lowered blood glucoses and lipid levels, as reported elsewhere.

Mattes (1990) has discussed the problems of analysis of hunger and fullness data in healthy adults. The women in this study were obese, and had established meal patterns that may have had little to do with actual sensations of hunger or fullness. That is, whether they did or did not have hunger had little relationship to whether they ate or not, something that Mattes also found in his subjects. Anecdotally, some subjects indicated that they didn't feel any sensations of hunger or fullness, thus accounting for diaries in which the ratings of neither-hungry-nor-full were the main recording. It could be that type 2 diabetes is accompanied by alterations in the normal cephalic, ingestive or postingestive signals and neurotransmitters that accompany eating. It could be that consumption patterns were oriented to environmental and other external prompts, or it could be that because of the length of time these women had been diagnosed with type 2 diabetes

(mean of 11.5 years), undetected changes in autonomic nervous system function had blunted hunger or fullness sensations. It could also be that these women were unable to feel the sensations of hunger and fullness due to subtly altered cognitive function, as indicated in their scores on the NCSE and Trails B tests. Finally, it could be that these hunger and fullness ratings represent what is normal eating, an issue which has yet to be resolved (Polivy & Herman, 1987). This view is supported to some degree by the correlations between temporal preference for time of the day and time that eating started and stopped in baseline, the two diet phases, and washout. These findings should be confirmed in other studies, but our findings suggests that assessment of M-E preference would provide valuable information when working with a patient on issues related to the diabetic diet. If hunger and fullness are considered to be needed cues for eating behavior within the context of living with type 2 diabetes, some type of awareness training of these body physiological signals may be necessary.

Attitudes and knowledge did not change in HM or HC. This was partially due to the fact that subject's were blind to the diet phases and instruction was limited to information needed to successfully participate and complete each diet phase of the study, rather than a full self care management program that would have included selecting diet composition. Had additional diabetes self care management information been provided, attitude and/or knowledge might have changed. The measurement tools used to detect these changes were not sufficiently developed,

detailed or sensitive to measure changes in thinking. Anecdotally, subject's blood sugars were normalized within the second week of the first diet phase, leading to statements such as "I guess food really does make a difference!" These comments suggest that self management skill development still needed to occur with these subjects even though they have been living with their diabetes an average of 11 years.

From the perspective of these subjects, this study reduced the pressure they experienced on a daily basis to consume the diabetic diet. All of the patients in this study had attempted to control eating and/or lose weight and had failed. All of the women worried about "eating the right thing", and none had developed long-term strategies to reduce that worry. All expressed relief to be in this diet study because they did not have to make daily decisions about food intake. In effect, some of the food-related challenges of living with diabetes were reduced. Several investigators have utilized food provision and delivery such as was used in this study for persons living with diabetes (Jeffrey, *et al.*, 1993) and/or cardiovascular disease (Pi-Sunyer, *et al.*, 1999), even though not all lipid abnormalities were corrected with this methodology. Even with this limitation, this study suggests several possibilities for diet therapy in type 2 diabetes for the future. It may be that for the woman with type 2 who is obese at the time of diagnosis, provision of food for a period of time while other aspects of self care management are mastered, is a more effective way of supporting self care management development. During the period of receiving food,

the woman would learn not that, but how food makes a difference to her diabetes through an individualized approach such as personal coaching in food preparation, grocery shopping, and dealing with challenging situations. When these aspects of living with this chronic disease are mastered, the provision of food can be tapered and discontinued. There may be other women with type 2 diabetes who would benefit from subscribing to a service where diet composition and foods appropriate to the composition are provided on a long-term basis because targeted therapeutic goals need to be met such as reduction of blood sugar, improvement of lipid profile, reduction of lipid peroxidation, or loss of weight. When these goals have been met, the decision for provision of food can be revisited.

It has been known for a long time that ingestion of and adherence to the diabetic diet is difficult to achieve; some of the reasons for this are unknown. The observation of eating behavior within this study provides some initial understanding of this phenomenon.

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EFFECT OF A HIGH MONUNSATURATED DIET ON SERUM LIPIDS, LIPID PEROXIDATION, AND GLYCEMIC CONTROL IN POSTMENOPAUSAL WOMEN WITH TYPE 2 DIABETES

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Abstract

Objective

To compare the effects on eating behavior, lipids, lipoproteins, lipid peroxidation,and glycemic control in women with type 2 diabetes of a high-monounsaturated fat (HM) diet compared to a high-carbohydrate diet (HC).

Research Design and Methods

In an outpatient feeding study, ten hypertriglyceridemic postmenopausal type 2 diabetic women alternately for six weeks consumed the HM and HC diets. On the HM diet, 45% of total calories were consumed as carbohydrate and 40% as fat (27% monounsaturated) compared with 55% and 30% fat (10% monounsaturated) in the HC diet. At the beginning and end of each diet phase, total lipids, lipoproteins, lipid peroxidation, and glycemic variables were measured.

Results

Total cholesterol was significantly decreased on the HC diet. Serum triglyceride, VLDL-triglyceride and cholesterol, and apolipoproteins A-1 and B were not significantly different on the two diets. When comparing initial to final values, both diets lowered LDL-C; however, the change was greater on the HM diet. Lipid peroxidation variables improved when the HM diet was consumed. Glycemic variables improved on both diets.

Conclusions

Consumption of the HM and HC diets did not result in deterioration of serum lipids. The HM diet by virtue of less oxidation of the LDL particle and improvement of glycemic control offers an important advantage over the HC diet.

Introduction

The appropriate diet for the person with type 2 diabetes has long been the subject of debate. The effective diet will provide improvement of plasma lipids, lipoproteins and glucose values over a period of time while reducing complications associated with the disease. For healthy individuals, replacing saturated fatty acids with monounsaturated fatty acids or equivalent caloric amounts of carbohydrate tends to decrease total cholesterol and LDL-cholesterol while raising HDL-cholesterol, changes which are associated with decreased risk for cardiovascular disease. However, consumption of a high carbohydrate diet is also associated with an increase of serum triglycerides. Since a very common lipid abnormality in type 2 diabetes is elevated serum triglycerides concentrations (1), a high carbohydrate diet for the person with type 2 diabetes seems inappropriate. This has led to further exploration of the effect of diets with increased concentrations of monounsaturated fatty acids in persons with type 2 diabetes.

The first study to explore the effect of monounsaturated fatty acids in the diabetic diet of persons with type 2 diabetes was reported by Garg et al (2). Ten men with type 2 diabetes were fed 60% of their calories as carbohydrate and 25% as fat (9% monounsaturated fatty acids) in one arm of the study; this was compared with 35% carbohydrate and 50% fat (33% monounsaturated fatty acids) in the other arm of the study. Plasma triglycerides and apolipoprotein A-1 significantly

increased on the high carbohydrate but decreased on the high monounsaturated fat diet. There were no significant changes in total cholesterol or LDL-cholesterol on either diet.

Since the Garg study, several other dietary feeding studies to evaluate the effects of diets enriched with monounsaturated fatty acids compared to increased levels of carbohydrate in persons with type 2 diabetes have been reported (3-9). Together, these studies suggest that the high monounsaturated diet, when compared to the high carbohydrate diet, very modestly lowers total cholesterol, significantly lowers triglycerides and VLDL-triglyceride, if measured, has a variable effect on HDL, and does not cause deterioration of glycemic control.

In addition to the usual lipid and lipoprotein outcome variables, lipid peroxidation of LDL was examined in an outpatient diet study of free living persons with type 2 diabetes where a diet rich in monounsaturated fatty acids was compared with one rich in polyunsaturated fatty acids (10). Lipid peroxidation is an important outcome variable to include in studies of persons with type 2 diabetes. Elevated levels of thiobarbituric acid reactive substances (TBARS) as a measure of lipid peroxidation have been measured in persons with type 1 and type 2 diabetes (11,12). In addition, recent studies have shown that oxidation of LDL may play a causative role in the development of the atherosclerotic plaque. This process directs LDL away from the LDL receptor to scavenger receptors and enables it to deposit triglyceride and cholesterol into foam cells and macrophages within the arterial

intima (13). Parfitt and colleagues (10) found that the amount of lipid peroxidation in plasma was similar on the monounsaturated and polyunsaturated diets and lower than the values measured after consumption of the high saturated fat baseline diet, a finding which is surprising because diets enriched with polyunsaturated fatty acids are believed to increase lipid peroxidation when compared to diets enriched with monounsaturated fatty acids.

More recently, another study has been reported in which the effects on lipids, glycemic control, and lipid peroxidation of two hypocaloric diets, one high in carbohydrate (HC) and the other high monounsaturated fatty acids (HM), was tested with male and female obese patients with type 2 diabetes (14). This feeding study involved taking the patients off all diabetic medications and entering them into a prestudy phase where an isocaloric, 55% carbohydrate diabetic diet was given. Then patients were assigned to the HM hypocaloric or the HC hypocaloric diet; the two diets provided approximately 50% of estimated need. After various lipid, glycemic and lipid peroxidation parameters were measured, patients continued as outpatients on the hypocaloric diet to which they had been assigned for six weeks. At that time, testing was done. The patients continued taking the hypocaloric diet but calories were gradually restored over a 4 week period until they returned to isocaloric levels. Besides losing 7-8 kgs of weight during the dieting phases, total cholesterol, triglycerides, and VLDL-cholesterol decreased more on the HM diet; HDL-C decreased more on the HC diet. Lag time of LDL oxidation increased

significantly after dieting and refeeding in the HM group (208 ±10 minutes and 221 ±13 minutes, respectively), whereas there was no change in lag time in the HC group during dieting or refeeding (146 ±11, 152 ±9 minutes, respectively); the HC diet lag times were significantly shorter than those on the HM diet. The value of this study is that it continues to expand the evidence that monounsaturated fatty acids in the diet, ingested for a 6- to 10-week period, are more effective than HC diets in achieving beneficial changes in lipids with no loss of glycemic control. Additionally, these fatty acids have the capacity to improve measures of lipid peroxidation, a variable linked to the higher risk of cardiovascular disease in type 2 diabetes.

These studies have varied in length of time of the dietary interventions and washout phases, degree of control over foods eaten (inpatients on metabolic units, outpatient feeding studies, outpatients preparing their own food with dietitian guidance); gender representation (66% men vs 34% women), and outcome variables. The treatment regimen has varied with diet alone (4); diet and oral agent (3,5,7,8,10); diet and insulin (2); or has been unclear about how many patients received which treatment (9,16). None of the studies were done with patients on combination therapy (oral agent plus insulin). Apolipoprotein E phenotype has not been addressed although it influences LDL concentrations independent of diabetes. As a result, there is a need for dietary studies with patients diagnosed with type 2 diabetes to select subjects who are as homogeneous as possible in terms of

treatment, to provide randomization of subjects and crossover designs, to maintain investigator control of food intake, to provide long enough feeding periods (months vs days), to adequately represent vulnerable groups such as women with type 2 diabetes, and to expand outcome variables to include phenomena such as lipid peroxidation (15).

The outpatient feeding study reported here describes the effects on plasma lipids, lipoproteins, lipid peroxidation, glycemic control in postmenopausal women with type 2 diabetes mellitus of a high-monounsaturated fat (HM) diet compared with the traditional diabetic high-carbohydrate (HC) diet. Patients selected for the study were homogeneous with respect to diabetes treatment regimen, apolipoprotein E phenotype, entry triglyceride and LDL-C concentrations.

Research Design and Methods

The study was approved by the Oregon Health Sciences University (OHSU) and Oregon State University Institutional Review Boards and the Advisory Committee of the Center for Clinical Research at OHSU. Each patient gave informed consent.

Patients for the study were recruited from attendees of primary care, internal medicine and diabetes clinics at Oregon Health Sciences University in Portland, Oregon. From a initial pool of 410 women, 255 (62%) were dropped after preliminary screening through chart audit due to documented episodes of ketosis,

other medical diagnoses (cancer, alcohol abuse, liver disease, unstable cardiovascular disease), dementia, recent use of steroids, high doses of vitamin B6, or regular menstrual cycles. Of the 155 remaining, nearly half (42%) were screened in a two-visit procedure which included a lipid profile (total cholesterol, triglyceride, LDL-cholesterol, HDL-cholesterol concentrations), apolipoprotein E phenotype, random blood sugar, glycosylated hemoblogin [GHb], and body mass index [BMI] (weight [kg]/ht [m^2]). It was established that potential subjects were able to read English, their diabetes regimen was combination therapy (Glynase PresTab©, Upjohn Company, Kalamazoo, MI; Novolin 70/30©, Novo Nordisk Pharmaceuticals, Inc., Princeton, New Jersey), and they had type 2 diabetes based on case review by the diabetes specialists using criteria described by Bingham and Riddle (16). Cognitive function was determined by the Neurobehavioral Cognitive Status Examination (17) and Trailmaking A and B (18).

Other inclusion criteria were 1) a mean of two fasting triglyceride values >250 but <600 mg/dL; 2) a mean of two LDL-cholesterol values <160 mg/dL; 3) apolipoprotein E phenotype other than 2/2 or 4/2; and 4) postmenopausal status (no menstrual period for at least 12 months). From the resulting pool of thirty-eight women, ten were selected and assigned to two groups of five persons each. There were no significant differences between the two groups at entry into the study in terms of BMI, mean entry triglycerides, total cholesterol, LDL-cholesterol, or Ghb.

Clinical characteristics of the patients at baseline are given in Table 7. None of the patients had recent myocardial infarction, unstable angina, or acute infection. Apolipoprotein E phenotypes were 3/2 (n=3), 3/3 (n=6), and 4/3 (n=1).

The outpatient feeding study consisted of four consecutive, 6-week intervals. The first group of five patients (Group 1) entered and completed the high monounsaturated (HM) study diet phase, entered and completed the washout phase, and entered and completed the high carbohydrate (HC) diet phase. These blocks of time are referred to as the first, second, and third phases for Group 1. The second group of five patients (Group 2) entered and completed the HC diet phase while Group 1 was in their washout phase; entered and completed the washout phase, and then entered and completed the HM diet phase. These blocks of time are referred to as the first, second, and third phases for Group 2. The washout phases were an average of 60 (57-63) days in length. Estimations of energy intake for the initial caloric level were made through analysis of patients' 3-day food record (Food Processor, ESHA Research, Salem, Oregon), semiquantitative food frequency (DIETSYS, National Cancer Institute, Bethesda, Maryland), and physical activity recall (19). Patients were instructed in research diets, study procedures, capillary blood glucose testing, treatment of hypoglycemia, and daily and weekly record keeping related to the study. During the dietary intervention phases, patients were instructed to maintain their usual eating pattern each 24-hour period and were free to chose to eat any food provided by the study at any time of the day. Patients were

TABLE 7

Characteristics of patients at baseline (Mean ±SE)

Characteristic	Value
Age (years)	56.9±2.8
Average years since diagnosis of diabetes	11.5±1.8
Weight (kg)	104±6.0
Body mass index (kg/m^2)	40.1±2.0
Waist:hip ratio	0.92±0.0
Baseline diet intake*	
Average calories consumed/day	2203±193
Percent carbohydrate†	47±2
Percent total fat†	36±1
Percent saturated fat†	12±0.9
Percent monounsaturated fat†	14±0.8
Percent polyunsaturated fat†	7±0.4
Total cholesterol (mmol/L)	5.53±0.3
Triglyceride (mmol/L)	3.59±0.4
LDL-cholesterol (mmol/L)	3.10±0.2
GHb (%)	12.3±0.7

TABLE 7 (Continued)

Neurobehavioral cognitive status exam		
Level of consciousness, orientation, attention, language: comprehension, repetition, naming, similarities, judgment:	normal	n=10
Construction:	normal	n=8
	moderate impairment	n=2
Memory:	normal	n=7
	mild impairment	n=2
	moderate impairment	n=1
Calculation:	normal	n=9
	mild impairment	n=1
Trails Test		
A (average seconds ± SE)		40.90±5.59
	normal	n=6
	mild/moderate impairment	n=1
	moderate/severe impairment	n=3
B (average seconds ± SE)		115.20±14.88
	normal	n=4
	mild/moderate impairment	n=3
	moderate/severe impairment	n=3

* Based on three-day food record analysis

† Percent of total calories consumed per day

blind to the specific diet. They were paid at the end of the study for each day of the two diet phases that were completed.

On a daily basis, foods for the next 24 hours were prepared and packaged by the dietitians and staff of the Clinical Research Center at Oregon Health Sciences University, and delivered by study personnel to the patients' homes in the evening. At the time of food delivery, the patient was weighed on a portable, electronic scale (Detecto, Webb City, MO), capillary blood glucose, record keeping, other aspects of diabetes control were reviewed, and the paper sack containing the preceding day's empty food containers and any uneaten food was collected. Deviations from the study protocol such as missing or being late with diabetes medication, diet breaks such as eating something not on the research diet or eating something not considered "free" food, or uneaten food left in the patient's sack were recorded by study personnel. Once weekly, a venous blood glucose was collected for analysis, and the patient completed a paper and pencil assessment of adherence to the study protocol.

Diets

The HM diet was calculated to contribute 45% of the total calories as carbohydrate (35% complex, 10% refined), and 40% as fat (10% saturated, 27% monounsaturated, 3% polyunsaturated fatty acids). The HC diet was calculated to contribute 55% of the total calories as carbohydrate (45% complex, 10% refined),

and 30% as fat (10% saturated, 10% monounsaturated, 10% polyunsaturated fatty acids). Protein (15% of total calories), cholesterol (< 300 mg/day), and fiber intake (15 grams/day) were held constant in both diet periods. Core foods of the HM and HC diets were developed and taste-tested by Clinical Research Center staff, study personnel, and patients; these foods had to appear similar in both dietary periods, varying only in composition. These core foods were incorporated into a four-day cycle of foods for each diet. A list of foods on the four-day cycle are available upon request. An oil high in oleic acid concentration (TriSun©, Wycliffe, OH) was incorporated into the foods on the HM diet; a soybean and sunflower oil high in linoleic acid was incorporated into the HC diet. Patients were allowed to drink coffee and tea ad libitum; non-calorie sodas and seltzers were provided on request. Two hundred (200) calorie increments and decrements of the HM and HC diet were created to correct for energy intake and weight changes that might occur during the study. Physical activity was kept constant. None of the patients drank alcohol or smoked.

Procedures

Adherence to the research diet was ascertained through information recorded on the patient's daily journal, through patients' weekly visual analogue scale (VAS) ratings on selected questions of a 5-item paper and pencil instrument adapted from Padilla (20), through observation and record keeping of study

personnel, and through anticipated changes in patients' LDL fatty acid profiles. The patients' daily journal was a place in which they could document problems with the diet. The journal was reviewed every day when study personnel visited. Eaten and uneaten foods were checked daily at the time of the food delivery; the data were recorded by study personnel. The weekly rating of adherence was collected at the end of each week at the time the patient's journal was replenished with record keeping forms for the next week. The LDL fatty acid profile was evaluated to see that the HM diet produced an increase in oleic acid concentration of LDL compared to that of the HC diet.

Blood samples were collected in Vacutainer® tubes appropriate to the sample after a 12-hour fast during the first, second, third, fifth, and last weeks of a diet phase. The lipid profile (total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol) was determined on samples taken at the first, second, fifth and sixth weeks. The values obtained on the first and second weeks and fifth and sixth week were averaged as a measure of the initial and final values since a more accurate assessment is made by averaging repeated measures (21). Free insulin, fructosamine and hematocrit were measured on samples obtained the first, third, and last weeks of a diet phase. VLDL and LDL composition, plasma TBARS, LDL oxidation, and vitamin C were measured on samples collected the first and sixth weeks. The blood samples for lipid peroxidation were immediately wrapped in foil, placed on ice, and a nitrogen head added to the sample. For these samples, analysis

occurred in less than 3 hours and samples were kept darkened at -4°C. In the first and last week of each diet period, patients collected a 24-hour urine sample. The urine was kept at ~5°C by storing the collecting bottles in a cooler provided to the patients equipped with a reusable ice substitute (Freez-Pak, Lifoam Leisure Products, Baltimore, MD). BHT (butylated hydroxytoluene, 2g/L, 50μL BHT/500μL sample) was added to both urine and plasma samples in which lipid peroxidation was measured. Other blood samples not immediately analyzed were stored at -80° C.

Patients performed capillary blood glucose testing twice daily, before breakfast and prior to the evening meal during each dietary phase. Blood glucose meters (Accu-chek Easy, Boehringer Mannheim Diagnostics, Indianapolis, IN) were cleaned and standardized when the patient began use of a new bottle of Chemstrips (Boehringer Mannheim Diagnostics, Indianapolis, IN). On weeks 2, 3, and 4 of each diet period, a random venous blood sugar was drawn immediately after the evening capillary blood glucose test and analyzed to assess patient accuracy in capillary blood glucose testing.

During each diet phase of the study, food for one four-day cycle representing 2400 calories was blended and frozen for proximate analysis, fatty acid profile, and concentration of α-tocopherol.

Biochemical Analyses

Cholesterol and triglycerides of plasma and lipoproteins were determined enzymatically (Sigma Diagnostics; St. Louis, Mo) as discussed previously (22). After precipitation of LDL and VLDL with phosphotungstic acid and $MgCl_2$, HDL-cholesterol was measured enzymatically (Sigma Diagnostics, St. Louis, MO). LDL-cholesterol was calculated using Friedewald's formula (23). Sequential ultracentrifugation was used to prepare VLDL and LDL (24) using a 50.3 Ti rotor (Beckman, Palo Alto, CA). VLDL was collected at density less than 1.006 g/mL; LDL was collected in the density range from 1.006 to 1.061 g/mL. VLDL-cholesterol and VLDL-triglyceride were calculated as the difference between total plasma cholesterol and triglyceride concentrations and those of the bottom fraction remaining after the first ultracentrifugation. LDL-cholesterol and LDL-triglyceride were calculated by subtracting the values measured in HDL from those measured in the bottom fraction of the second ultracentrifugation. Apolipoprotein A-1 and apolipoprotein B concentrations were measured by immunoturbidimetric methods (Raichem, San Diego, CA). Apolipoprotein E (baseline only) was measured through adaptation of the method of Assman (25).

Lipid peroxidation in plasma and urine was evaluated by measuring TBARS. Plasma and urinary TBARS were measured by the method described previously (22). In vitro Cu^{2+}-dependent oxidation of LDL was measured using the method of Esterbauer (26) as previously described (22) with the exception that the data were

determined relative to the concentration of cholesterol in the LDL particle rather than its protein content. The LDL fatty acid profile was measured by gas chromatography as previously described (27) using tricosanoic acid as an internal standard (Nu-Chek Prep, Elysian, MN). Both α- and γ-tocopherol were measured in the LDL fraction of plasma by high performance liquid chromatography (Shimadzu, Columbia, MO) with a fluorescence detector (22). Protein was determined by the method of Lowry (28). Vitamin C was measured in plasma spectrophotometrically with a 2, 4 dinitro-phenylhydrazine chromagen (29). Fasting serum insulin was measured by immunoassay (30) and the free insulin fraction separated by polyethylene glycol precipitation (31). GHb was determined by automated affinity chromatographic technique using boronic acid (32). Venous blood glucose samples were analyzed via the automated glucose oxidase/oxygen consumption method (Beckman) (33). Capillary blood sugar testing was done using Chemstrip BG strips in an Accuchek II blood glucose meter (Boehringer Mannheim Diagnostics).

Proximate analysis of the diet (dry matter, ash, fat and crude protein) was done by the Forage Analytical Laboratory, Oregon State University, Corvallis, OR. The fatty acid profile of food in the diet was measured by gas liquid chromatography (34). Alpha-tocopherol in the diet was measured by high performance liquid chromatography (22).

Statistical Methods

Three statistical evaluations were made: carryover between diets, the effect of the HM diet compared to the HC diet, and the effect of a diet (HM or HC) within a period. To determine if there were any carry-over effects related to sequence of the two diets for Group 1 and Group 2, the mean of the first and second week's primary response variables in the first and the mean of the first and second week's primary response variables in the third phase were compared by the two-tailed Student's t test (35). To compare the effect of the two dietary interventions, the mean final (fifth and sixth week) values measured when the women in Group 1 consumed the HM diet were combined with the mean final values measured when the women in Group 2 consumed the HM diet. These combined data are referred to as data obtained at the end of HM period. A similar combination was made with the data obtained when the HC diets were consumed. Effects of the two diets on major variables (lipid, lipoprotein, lipid peroxidation, and glycemic variables) were analyzed using paired t-tests. To determine if one of the dietary interventions significantly changed a variable, the initial values measured before a diet was consumed were compared to the final values after the diet was consumed, using a paired t-test. Wilcoxon's signed-rank test was used when data were not normally distributed. Where relevant, correlations were performed (35) to identify significant relationships between variables. Results are expressed as means ± as indicated. The $p \leq 0.05$ level of significance was used to assess statistical significance.

Results

Carryover

No significant differences were found when comparing the initial values for primary variables measured prior to consumption of the HC diet by Group 2 to those measured prior to the consumption of the HM diet by Group 2. However, significant differences were found for VLDL-triglycerides, LDL-cholesterol and LDL-triglycerides when comparing initial values on the HM diet for Group 1 to initial values prior to the consumption of the HC diet for Group 1. These findings were attributed to one patient in Group 1 having triglycerides at but not exceeding upper limits for participation in the study; these differences were not attributed to carry-over effect due to the length of the washout phases. Consequently, data from this patient were retained for all subsequent analyses.

Body weight of the patients at the start and by the end of the two dietary periods was not significantly different (HM: 102.2 ±5.5 and 101.1 ±5.7 kgs; HC: 103.9 ±5.8 and 101.5 ±5.5 kgs. There was no significant difference in the average kcals consumed on the two diets (HM: 2700 ±166; HC: 2620 ±125 kcals, $P = 0.14$).

Adherence

All ten patients completed the study. Patients' daily journals indicated that one-half of the patients experienced minor health-related events while consuming the HM diet, and 40% while consuming the HC diet. None of these events

compromised study participation. Patients' daily journals also revealed an average of 3.7 and 3.9 deviations from the diet, e.g., leaving uneaten food or eating something not on the research diet while consuming the HM and HC diets, respectively. In both dietary periods, patients' responded to two questions using a VAS scale with anchors of "always" and " never". Answers to the first question (How often have you been able to eat just the food provided by the study?) should have clustered at the "always" end of the scale. The mean rating in HM (1.27 ± 0.27) and HC (1.23 ± 0.32) indicated a high degree of self reported adherence with consumption of the research diet, although there were no significant differences between HM and HC. Answers to the second question "A person has to be pretty creative to make study food taste better", on the other hand, should have clustered toward the "never" end of the scale. Although varying the temperature of the food or other preparations which did not alter calories or composition, was accepted, the addition of condiments, spices, and other foods to make the diet taste better should have been limited. The mean rating on HM was 1.76 ± 0.67 and on HC was 1.70 ± 0.82, and while the ratings on HM and HC were not significantly different, the ratings indicate that some type of alteration was always used. It could also be that subjects misinterpreted the anchors on the rating scale and marked it incorrectly. Given other aspects of the study which confirm adherence, this interpretation seems probable. Daily records maintained by personnel conducting the study regarding deviations from study protocol concurred with patient journals. The anticipated

increase in the oleic acid concentration of LDL when the HM diet was consumed and linoleic acid concentration of LDL when the HC diet was consumed, occurred (Table 8).

Diet Composition

The analyzed composition of the HM and HC diets closely met the planned composition. The analysis of the dietary oils indicated that the major fatty acid in TriSun oil was oleic acid (18:1n9c), 84.8g/100g oil; the major fatty acid in soybean and safflower oil was linoleic acid (18:2n6), 76.5g/100g oil. Differences in the content of 18:1n9 and 18:2n6 were measured in the HM and the HC four-day diet cycle (Table 9). These differences are consistent with the intended design of the diets in the HM and HC dietary periods. On the HM diet, LDL composition reflected the increase in 18:1n9 and the reduction in 18:2n6 in the diet. On the HC diet, 18:1n9 concentrations in LDL were stable, and 18:2n6 concentrations in LDL rose (Table 8). Other aspects of the analyzed diet were within 1-2% of the planned diet (Table 10). There was slightly more α-tocopherol but less γ-tocopherol in the HM diet when compared to the HC diet. The average α-tocopherol content of the HM diet was 19.2 μg α-tocopherol/gm of diet and 13.7 μg α-tocopherol/gm of diet on the HC diet.

TABLE 8

LDL fatty acid profile in HM and HC diet periods.
Values are expressed as mean area percent ± SE for initial and final weeks of a diet period.

Fatty acid	HM diet			HC diet			
	Initial	Final	p*	Initial	Final	p*	p†
12:0	0.04 ± 0.03	0.14 ± 0.05	0.04	0.02 ± 2.04	0.05 ± 0.03	0.11	0.08
14:0	0.97 ± 0.06	0.74 ± 0.11	0.03	0.92 ± 0.08	0.99 ± 0.10	0.24	0.08
16:0	21.0 ± 0.05	19.5 ± 0.46	0.02	21.2 ± 0.90	21.5 ± 0.70	0.48	0.02
16:1	2.82 ± 0.24	2.18 ± 0.25	0.02	2.70 ± 0.19	2.71 ± 0.28	0.93	0.04
18:0	5.85 ± 0.17	5.78 ± 0.19	0.75	5.84 ± 0.21	6.06 ± 0.16	0.32	0.03
18:1n9	15.7 ± 0.50	23.9 ± 0.80	0.000	16.6 ± 0.73	15.5 ± 0.32	0.07	0.000
18:2n6	30.3 ± 0.99	23.6 ± 1.14	0.0002	27.7 ± 1.02	30.3 ± 1.25	0.02	0.000
18:3n3	0.53 ± 0.04	0.27 ± 0.03	0.005	0.53 ± 0.07	0.41 ± 0.04	0.20	0.005
20:2n6	0.22 ± 0.02	0.14 ± 0.03	0.04	0.26 ± 0.04	0.24 ± 0.01	0.77	0.02

TABLE 8 (Continued)

Fatty acid	HM diet			HC diet			
	Initial	Final	p*	Initial	Final	p*	p†
20:4n6	8.16 ± 0.52	8.00 ± 0.58	0.58	7.89 ± 0.46	8.20 ± 0.62	0.24	0.37
22:0	0.91 ± 0.05	1.28 ± 0.06	0.005	0.82 ± 0.03	0.82 ± 0.04	0.95	0.000
22:6n3	1.22 ± 0.10	1.36 ± 0.06	0.26	1.09 ± 0.09	1.12 ± 0.06	0.68	0.007
24:0	0.70 ± 0.05	1.02 ± 0.13	0.005	0.68 ± 0.06	0.65 ± 0.04	0.44	0.007

* p value when comparing initial to final within a period † p value when comparing mean final for HM to mean final for HC

TABLE 9

Fatty acid profile of the HM and HC diets
Values are expressed as mean area percent ± SD. The measured values are an average of each of the four-day cycles taken both times the diets were fed. (n=8 HM and 8 HC diet samples)

Fatty Acid	HM	HC
12:0	0.68 ± 0.25	1.10 ± 0.37
14:0	2.33 ± 0.31	3.67 ± 0.28
16:0	10.6 ± 2.56	18.3 ± 0.72
16:1n7	0.74 ± 0.10	0.98 ± 0.17
18:0	7.41 ± 0.48	8.59 ± 0.66
18:1n9	65.2 ± 1.48	29.3 ± 1.57
18:2n6	7.05 ± 0.76	31.5 ± 0.81
18:3n3	0.60 ± 0.15	1.49 ± 0.42
20:0	0.29 ± 0.06	0.16 ± 0.13
20:1n9	0.35 ± 0.05	0.32 ± 0.06
20:2n6	0.11 ± 0.04	*
20:4n6	0.02 ± 0.04	0.22 ± 0.43
22:0	0.77 ± 0.00	0.21 ± 0.09
22:1n9	*	0.02 ± 0.04
22:6n3	0.14 ± 0.14	*
24:0	0.20 ± 0.11	*

* Non-detectable

TABLE 10

Planned versus analyzed composition of the HC and HM diets
Values for analyzed diet expressed as mean %.
(n= 8 HM samples and 8 HC samples; total of 16)

	Planned	Analyzed
HM Diet		
Carbohydrate	45%	47%
Protein	15%	16%
Fat	40%	39%
Saturated	10%	---
Monounsaturated	27%	---
Polyunsaturated	3%	---
α-tocopherol (mg/day)	14.4	
γ-tocopherol (mg/day)	2.4	
HC Diet		
Carbohydrate	55%	57%
Protein	15%	16%
Fat	30%	27%
Saturated	10%	---
Monounsaturated	10%	---
Polyunsaturated	10%	---
α-tocopherol (mg/day)*	10.8	
γ-tocopherol (mg/day)*	6.1	

* Assuming intake of approximately 2400 calories/day

Lipids, Lipoproteins, Apolipoproteins and Glycemic Control

The LDL fatty acid profile of patients differed significantly when comparing initial to final values after either of the diets were consumed, and when comparing final values measured after the HM diet was consumed to the final values measured after the HC diet was consumed (Table 8).

The effect of the HM and HC diets on lipids, lipoproteins, apolipoproteins and glycemic control is summarized in Table 11. The only difference in the lipid and lipoprotein group of variables when comparing the final mean on HM with the final mean on HC is that total cholesterol concentration was lowered significantly on the HC diet ($P = 0.01$). However, when comparing initial to final mean concentrations of these variables for each diet, triglyceride concentration did not rise and showed a trend toward a decrease on the HC diet ($P = 0.07$), LDL-cholesterol concentrations were reduced significantly on HM ($P = 0.03$), and HDL-cholesterol showed a trend toward an increase on HM ($P = 0.07$).

The accuracy of patient-performed capillary blood sugar testing was determined by daily observation of patient technique during the evening capillary blood glucose test at the time of the visit, and by comparing a weekly random venous blood sugar tests paired with an evening capillary blood sugar test on weeks 2, 3, and 4 in both dietary periods. Pearson's correlations between the weekly venous and capillary blood sugar samples was high ($r = 0.67$) and improved as the study progressed ($r = 0.99$).

TABLE 11

Effect of HM and HC diet periods on serum lipids, lipoproteins, apolipoproteins, and glycemic variables. Values expressed as means ± SE.

	Initial	Final	p*	p†
Total cholesterol (mmol/L)				
HM diet period	5.79 ±0.24	5.61 ±0.23	0.20	0.0
HC diet period	5.87 ±0.43	5.24 ±0.22	0.04	
Triglyceride (mmol/L)				
HM diet period	2.66 ±0.26	2.85 ±0.53	0.50	0.39
HC diet period	3.39 ±0.79	2.42 ±0.41	0.07	
VLDL-cholesterol (mmol/L)				
HM diet period	0.67 ±0.15	0.86 ±0.21	0.28	0.44
HC diet period	1.06 ±0.39	0.97 ±0.31	0.20	
VLDL-triglyceride (mmol/L)				
HM diet period	1.43 ±0.23	1.69 ±0.47	0.87	0.14
HC diet period	2.28 ±0.81	2.18 ±0.65	0.72	
LDL-cholesterol (mmol/L)				
HM diet period	3.62 ±0.25	3.14 ±0.24	0.03	0.59
HC diet period	3.41 ±0.27	3.07 ±0.20	0.07	
HDL-cholesterol (mmol/L)				
HM diet period	0.93 ±0.05	1.01 ±0.07	0.07	0.20
HC diet period	0.91 ±0.05	0.94 ±0.06	0.28	

TABLE 11 (Continued)

	Initial	Final	p*	p†
Apolipoprotein A-1 (mg/dL)				
HM diet period	115 ±3.70	113 ±4.64	0.77	0.71
HC diet period	121 ±5.86	115 ±6.87	0.23	
Apolipoprotein B (mg/dL)				
HM diet period	94.9 ±5.63	93.5 ±9.24	0.80	0.65
HC diet period	90.1 ±9.86	88.8 ±8.25	0.70	
Morning capillary blood glucose (Average of week 1,2 compared with average of weeks 5, 6)(mmol/L)				
HM diet	8.61 ± 0.87	7.03 ± 0.56	0.07	0.22
HC diet	8.22 ± 0.63	7.12 ± 0.47	0.01	
Evening capillary blood glucose (Average of week 1,2 compared with average of weeks 5, 6)(mmol/L)				
HM diet	8.97 ± 0.81	7.12 ± 0.41	0.03	0.05
HC diet	9.62 ± 0.56	8.08 ± 0.47	0.04	
Fructosamine (week 1 compared to week 6)(mmol/L)				
HM diet	2.37 ± 0.07	2.15 ± 0.07	0.0008	0.004
HC	2.84 ± 0.36	2.39 ± 0.07	0.15	
GHb (%total)				
HM diet	-	9.56 ± 0.41	0.13	
HC diet	-	10.4 ± 0.41		

TABLE 11 (Continued)

Free insulin (week 1 compared to week 6)(μu/mL)

HM diet	15.9 ± 2.27	14.1 ± 2.38	0.49	0.8
HC diet	17.3 ± 3.60	17.2 ± 2.41	0.99	

* p value related to comparing initial to final within a diet period

† p value comparison of final mean in HM with final mean in HC

The average morning final capillary blood sugar concentration on HM was not significantly different from the average morning final capillary blood sugar concentration on HC. However, the average final evening capillary blood sugar concentration was significantly different with the HM concentration being lower ($P = 0.05$). Short-term, e.g., previous 2-3 weeks, and long-term, e.g., previous 6 weeks, measures of ambient blood sugar levels were evaluated. There was a significant difference between the final fructosamine concentrations on the HM diet compared with the HC diet ($P = 0.004$). These findings suggest that the HM diet period offered a slightly better improvement in blood sugar control, although both the HM and the HC diet improved blood sugar control from baseline in these patients.

The effect of the HM and HC diets on LDL composition is given in Table 12. The free cholesterol and phospholipid content of the LDL particle was significantly higher after the consumption of the HM diet.

Lipid Peroxidation

Plasma TBARS concentrations were lower ($P = 0.0000$) after consumption of the HM diet, and the greatest initial-to-final change in concentration of plasma TBARS occurred when patients were on the HM diet. No significant differences were observed in urinary TBARS between HM and HC diet phases. (Table 13).

TABLE 12

Effect of HM and HC diet periods on LDL composition.
Values expressed as mean percent ± SE.

	Initial	Final	p*	p†
LDL-triglyceride (%)				
HM diet	8.86 ± 0.58	8.92 ± 0.82	0.93	0.52
HC diet	10.2 ± 0.89	9.49 ± 0.97	0.14	
LDL-free cholesterol (%)				
HM diet	8.16 ± 0.28	8.18 ± 0.20	0.94	0.0006
HC diet	7.55 ± 0.23	6.87 ± 0.35	0.13	
LDL-cholesterol ester (%)				
HM diet	35.5 ± 1.41	37.2 ± 0.98	0.27	0.52
HC diet	37.1 ± 0.74	38.4 ± 1.60	0.28	
LDL-phospholipid (%)				
HM diet	21.2 ± 0.55	20.3 ± 0.40	0.03	0.02
HC diet	19.6 ± 0.40	19.1 ± 0.16	0.30	
LDL-protein (%)				
HM diet	26.3 ± 1.21	25.5 ± 0.99	0.95	0.67
HC diet	25.6 ± 0.51	26.1 ± 0.66	0.46	

* p value for tests for initial/final differences within a dietary period
† p value for tests comparing final values at the end of HM and HC period

TABLE 13

Initial and final values for lipid peroxidation and LDL oxidation on HM and HC diets. Values are expressed as means ± SE.

	Initial	Final	p*	p†
Plasma TBARS (μmol/L plasma)				
HM diet	1.51 ± 0.09	1.39 ± 0.08	0.001	0.000
HC diet	1.52 ± 0.07	1.51 ± 0.08	0.90	
Urinary TBARS (μmol/day/kg body weight)				
HM diet	202 ± 47.0	140 ± 37.4	0.09	0.11
HC diet	245 ± 69.7	281 ± 110	0.44	
LDL oxidation: Lag time (minutes)				
HM diet	65.0 ± 6.30	101 ± 8.15	0.01	0.01
HC diet	84.0 ± 8.15	63.5 ± 4.67	0.02	
LDL oxidation: Rate (nmols conjugated dienes/mg LDL-cholesterol/min)				
HM diet	7.94 ± 0.76	4.40 ± 0.24	0.003	0.003
HC diet	5.49 ± 0.39	7.55 ± 0.48	0.009	
LDL oxidation: Concentration (nmols conjugated dienes/mg LDL-cholesterol)				
HM diet	384 ± 16.4	310 ± 11.7	0.006	0.01
HC diet	351 ± 9.24	353 ± 8.37	0.87	

TABLE 13 (Continued)

	Initial	Final	p*	p†
α-tocopherol in LDL (nmols/mg LDL protein)				
HM diet	9.48 ± 6.10	13.3 ± 7.85	0.0004	0.008
HC diet	12.5 ± 2.51	10.4 ± 0.91	0.51	
γ-tocopherol in LDL (nmols/mg LDL protein)				
HM diet	3.49 ± 1.26	1.72 ± 0.77	0.0001	0.39
HC diet	3.44 ± 0.70	1.84 ± 0.30	0.005	
Plasma Vitamin C (mg/dL)				
HM diet	0.70 ± 0.10	0.96 ± 0.12	0.07	0.92
HC diet	0.68 ± 0.13	0.97 ± 0.13	0.02	

* p value for tests of initial/final differences within a dietary period
† p value for tests comparing final values at the end of HM and HC period

On the HM diet, mean LDL final lag time lengthened by 36 minutes whereas on HC the LDL final lag time was significantly shortened by 21 minutes ($P = 0.01$). On the HM diet, the rate of conjugated diene production was reduced compared to an increase on the HC diet ($P = 0.0001$). The final concentration of conjugated dienes produced from LDL oxidation also differed between the two diets. On the HM diet, final concentration of conjugated dienes was lower as compared to the HC diet ($P = 0.01$). Concentrations of vitamins which could act as antioxidants differed in the dietary periods. Final concentrations of α-tocopherol measured in the LDL particle were significantly greater on the HM diet compared to α-tocopherol concentrations on the HC diet ($P = 0.008$). The final concentration of γ-tocopherol in LDL did not differ between diets. The HM diet improved various measures of lipid peroxidation (plasma TBARS, lag, rate, concentration of LDL oxidation) while the HC diet did not.

Discussion

Diabetic diets enriched with monounsaturated fatty acids compared to diets high in carbohydrate have been reported to reduce total cholesterol, triglyceride, LDL-cholesterol and apolipoprotein B, and increase HDL-cholesterol and apolipoprotein A-1 levels. Monounsaturated fatty acid enriched diets are also reported to protect LDL against oxidation.

The results of this study, in terms of improvement of lipid status, are mixed. It was unexpected that the HC diet reduced total cholesterol the most, although others (6) have also reported this finding. We were expecting that HM would reduce triglyceride and VLDL-triglyceride, and would thus be a diet that would improve these patient's hypertriglyceridemia. However, it is difficult to draw conclusions about the impact of these diets on this variable because one patient's triglyceride concentration was so much greater than that of the others at the beginning of the HC diet. Other studies have seen greater changes than we did in triglycerides and, when measured, VLDL-triglyceride. The improvements of HDL-cholesterol with the HM diet are encouraging.

When these results related to lipid status are viewed in light of current standards for evaluation of dyslipidemia in diabetes (36), the findings are neither normal nor do they fall into the "high risk" category. In the study, final mean total cholesterol values fell into the normal category; final mean triglyceride fell into the "acceptable or borderline" and LDL-cholesterol values fell into the "normal or desirable" category; and mean HDL-cholesterol levels remained too low (37).

Our findings of improved glycemic control but continued abnormalities in some lipid parameters have been observed by others (38). In this study, the HM diet and combination therapy were sufficient to obtain improved glycemic status, but did not improve all parameters of lipid status. The concentration of LDL-cholesterol improved and that of HDL-cholesterol tended to improve. Other studies could be

designed with this population of postmenopausal women which, using combination therapy and the high monounsaturated diet as a base, explore the effect of exercise alone, and/or the effect of exercise and weight loss, and/or other nonpharmacological approaches which are less costly than drugs. Additionally, it is unclear what would happen if the HM diet were continued for 3-6 months; there are no longterm studies of this nature.

As far as we know, this is one of the first studies to demonstrate the impact of monounsaturated enriched diets on measures of lipid peroxidation in moderately hypertriglyceridemic postmenopausal women with type 2 diabetes. The results of this study confirm findings in other feeding studies with healthy males (39), with hypercholesterolemic patients (40), and patients with type 2 diabetes on hypocaloric diets (13) that monounsaturated fatty acid enriched diets decrease the susceptibility of LDL to oxidation through lengthened lag time, slower production and lower final concentrations of conjugated dienes.

The effect of the monounsaturated enriched diet may have been produced by differences in the α-tocopherol concentration of LDL. It rose significantly from beginning to the end of the HM period; this did not occur in the HC diet. The HM diet actually provided a greater intake of α-tocopherol. It has been argued by some (42) but not others (22,43) that supplements of 400 mg α-tocopherol per day are required to protect LDL from oxidation. This study suggests that small increases in

dietary α-tocopherol when coupled to intakes of monounsaturated fatty acids are sufficient to protect LDL from oxidation.

One of the criticisms of this monounsaturated enriched diabetic diet has been that it increases energy intake from fat. In the view of many, this increase would certainly lead to weight gain or, at the least, make it difficult to lose weight if it were utilized on a clinical basis with free living patients. However, in free-living patients with type 2 diabetes who are making food choices independently, the more relevant issue is whether the calories ingested from the diet exceed those that are needed for that individual. A recent review of energy balance and the HM and HC diet suggests that many of the common reasons offered for lower rather than higher (40%) fat intake levels in patients with diabetes are only weakly supported (41). Thus, we believe this diet is as viable an option as any other in the treatment of type 2 diabetes in postmenopausal women.

The HM diet holds promise as an alternative to the traditional HC diet for women with type 2 diabetes. However, to explicate fully the mechanisms by which this occurs in women with type 2 diabetes, further studies using refined techniques for measuring lipid peroxidation events, ingestion of HM for longer periods than six weeks, and use of the HM diet soon after diagnosis of type 2 diabetes may be what is needed.

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CONCLUSIONS

In this chapter, a general summary of the findings, a discussion of the findings in light of the study hypotheses, conclusions and suggestions for further research will be given.

General Summary of Findings

A. The ten subjects consumed an average of 2620 - 2700 calories each day and body weight at the start was not significantly different from body weight at the end of each dietary period (HC: 103.9 ± 5.8 and 101.5 ± 5.5 kgs; HM: 102.2 ± 5.5 and 101.1 ± 5.6 kgs).

B. All subjects completed the study. Half of the subjects experienced minor health-related events on either diet; none of these events compromised study participation. Subjects deviated from the research diet an average of 3.8 times. Self reported adherence with the research diet (HC, HM) was high.

C. There was no carryover effect, although subjects in Group 1 began the HC diet with significant differences from the beginning of the HM diet in initial VLDL-triglycerides, LDL-cholesterol, and LDL-triglyceride concentrations. These differences were not attributed to carryover because the average length of time between dietary phases was about 55 days.

D. Validation of the independent variable of the study (HM and HC diet) was achieved through measures of LDL fatty acid composition in the HM and HC dietary phases, and through proximate analysis of the diet in the HM and HC dietary phases. An increase in LDL oleic acid content was found during the HM dietary phases, confirming that the HM diet was being consumed; LDL linoleic acid content rose during the HC dietary phases, confirming that the HC diet was being consumed. Proximate analysis of the diet was within 1 - 2 % of the planned composition of the diet.

E. The average number of meals (range: 4.47 ± 0.29 to 4.94 ± 0.40), snacks (range: 0.71 ± 0.30 to 1.20 ± 0.40), and total eating episodes (range: 5.42 ± 0.54 - 5.78 ± 0.60) did not vary significantly between any of the dietary phases.

F. The start of the eating period did not vary significantly between dietary phases (range in military time: 0642 to 0747). However, there was a trend toward significance for the time when eating stopped (range in military time: 1932 [washout phase] to 2209 [HC phase], p. 0.08). Eating period length ranged from a low of 13 hours and 20 minutes ± 43 minutes (washout) to a high of 15 hours and 15 minutes ± 71 minutes (HC). Significant correlations were found between morningness-eveningness type and the time eating started in the HC dietary phase, and for the time eating stopped in the baseline, HM and HC dietary phases, e.g., morningness preference types started eating earlier and stopped earlier and eveningness preference types started and stopped eating later in the day.

G. The HM and the HC diets were rated as being highly palatable. Three specific foods on the HM diet were rated as being more palatable than the HC diet.

H. There were no significant differences for ratings of preference for foods containing fat at the end of the HM and HC diet phases. When ratings of preference for fat of diabetic subjects in the study were compared with ratings of a control group of nondiabetic obese women, some significant differences emerged in rating of liking of various low- and high-fat foods. However, a more noticeable pattern of significant differences was seen between diabetic subjects and the nondiabetic control group in terms of their ratings of liking for foods with the sensory characteristics of saltiness, sourness and bitterness. Diabetic women rated these foods as close to the "neither like nor dislike" category whereas nondiabetic women rated these foods in the "like very much" or "like moderately" category.

I. No significant differences were found in terms of motivation to eat or changes in beliefs and ideas about eating when comparing the HM to the HC diet.

J. Subjects experienced more episodes of fullness in the HM and the HC dietary phases, as compared to episodes of neither-hunger-nor-fullness or episodes of hunger.

K. The HC diet lowered total cholesterol significantly more when compared to the HM diet, although the final mean was still higher than it should be, according to recommended standards.

L. When comparing changes in the initial to the final values for lipids, triglyceride concentration decreased on HC, LDL-cholesterol concentrations were significantly reduced on HM, and HDL concentrations increased on HM.

M. Free cholesterol and phospholipid content of the LDL particle was significantly higher after consumption of the HM diet.

N. Several measures of lipid peroxidation were significantly improved on HM as compared to the HC diet. These measures included significantly lower plasma TBARS, a shortened LDL final lag time, a slower rate of formation, a lower final concentration of conjugated dienes, and a higher final concentration of α-tocopherol in LDL.

O. Final values of the short-term measure of ambient blood sugar, e.g., fructosamine, were significantly lower on HM when compared to the HC diet. There was a significant difference between the final average evening capillary blood glucose on HM as compared to HC.

Findings Related to Study Hypotheses

Hypotheses related to Study Objective 1

Hypothesis 1: Perceptions of hunger will decrease on the HM diet. The average frequency of hunger episodes reported during the HM and HC recording periods were 26.3 ± 7.32 versus 39.5 ± 12.43, respectively. There were no significant differences between the frequency of hunger episodes between HM and HC ($p = 0.26$). Average hunger ratings at the highest (5 = completely hungry)

and the lowest intensity levels (1 = very little hunger) were also not significantly different on HM compared to HC. Therefore, there is no evidence that perceptions of hunger decreased on the HM as compared to the HC diet.

Hypothesis 2: Perceptions of fullness will increase while on the HM diet. The average frequency of fullness episodes reported during the recording periods were 39.5 ± 12.4 (HM) versus 55.7 ± 23.8 (HC). Within the HM and HC dietary phases, fullness was reported significantly more frequently than hunger was reported, $p = 0.005$. Average fullness ratings at the highest (5 = complete fullness) and the lowest intensity (1 = very little fullness) were not significantly different when comparing HM and HC. Perceptions of fullness were reported more frequently than perceptions of hunger, but fullness occurred on the HM and the HC diet.

Hypothesis 3: Perceptions of palatability will increase while on the HM diet.

a. Global ratings of palatability. The ratings of global (thinking about the diet for a whole day) palatability were significantly different for smell on Day 1 and Day 3. However, all ratings of global palatability dimensions (pleasantness, tastiness, texture, smell, appearance and richness) were located at the "very pleasant" end of the VAS scale, e. g., no rating was greater than 2.89, indicating that both diets were pleasant and palatable to the subjects. The null hypothesis is accepted.

b. Specific food palatability. All palatability ratings of specific foods were ≤ 4 on a 9 point scale where 1 = like extremely and 9 = dislike extremely

(range: HM, 1.00 - 4.00; HC: 1.33 - 3.25). Significant differences in ratings of specific foods occurred on Day 2 for canned fruit (HM 1.39 ± 0.16 versus HC 1.65 ± 0.13, p 0.03) and on Day 4 for a potato dish (HM, 1.00 ± 0.00 versus HC 1.45 ± 0.20) and a pudding pie (HM 1.05 ± 0.50 versus HC 1.90 ± 0.45, p. 0.03). Therefore, there was increased palatability of three specific foods on the HM diet as compared to the HC diet.

Hypotheses related to Study Objective 2

Hypothesis 1: Total lipids (total cholesterol, triglyceride) will improve while on the HM diet. Final mean concentrations of total cholesterol were significantly lower on the HC diet as compared to the HM diet (p= 0.01) However, the final mean value of total cholesterol was at a level between normal/desirable and acceptable/borderline (Garber et al., 1992), so that although total cholesterol was lower on HC, it was still higher than it should have been from a clinical perspective. Final mean concentrations of total triglycerides were higher on HM as compared to HC (p = 0.07) and the final HM triglyceride value was in a range considered too high to be acceptable from a clinical perspective (Garber et al., 1992) . Thus, HM did not improve total cholesterol or triglycerides.

Hypothesis 2: Lipoproteins will improve while on the HM diet. There were no significant differences between the final mean concentrations of VLDL-cholesterol or VLDL-triglyceride on HM or HC. There were no significant differences between the final mean concentrations of LDL-cholesterol when

comparing HM to HC. However, the initial to final concentrations of LDL-cholesterol are significantly different on HM ($p= 0.03$) and on HC ($p= 0.07$), and these changes from initial to final are concentrations which exemplify shifting from the acceptable/borderline to the normal/desirable range (Garber, 1992; ADA, 1998). The HDL-cholesterol HM final mean concentrations approached significance ($p= 0.07$) when compared to HC. To summarize, initial to final LDL-cholesterol values improved (dropped) on the HM diet, and HDL-cholesterol values rose on the HM diet, changes which are in the right direction for improvement of lipoproteins. Other lipoproteins (VLDL-cholesterol, VLDL-triglyceride) demonstrated no significant changes on HM or HC.

Hypothesis 3: Lipoprotein composition will improve while on the HM diet. LDL-free cholesterol was significantly higher ($p = 0.0006$) as was LDL-phospholipid final mean percent ($p= 0.02$) on HM when comparing HM to HC. There are no clinical standards exist against which to compare these findings.

Hypothesis 4: Apolipoproteins will improve on the HM diet. There were no significant differences in the concentrations of apolipoprotein A-1 or apolipoprotein B when comparing initial to final, or final mean concentrations between HM and HC. These concentrations fall within normal ranges.

Hypothesis 5: Measures of lipid peroxidation will improve while on the HM diet. Plasma TBARS were significantly lower when comparing initial to final concentrations on HM ($p= 0.001$) and when comparing final means of HM to

HC (p =0.000). Urinary TBARS concentrations approached significantly lower levels on HM (p= 0.09) when initial to final values on HM and HC are compared, but there was no significant difference when comparing final mean values. LDL oxidation lag time was longer (p= 0.01), LDL oxidation rate was significantly lower (p=.0.003), and the concentration of LDL conjugated dienes was significantly lower when comparing final mean values in HM to HC(p = 0.01). The concentration of α-tocopherol in LDL was also significantly higher on HM as compared to the HC diet. Lipid peroxidation products as measured in plasma TBARS, and processes associated with LDL oxidation were improved on HM.

Conclusions Related to Study Objectives

A. The first objective of this research study was to describe variations in selected eating behaviors of subjects when ingesting a HC diet compared to a HM diet. To meet this objective, three eating behaviors were investigated: a) temporal patterns of eating, e.g., time of meals and snacks across the 24 hour period, meal and snack macronutrient composition across a 24 hour period, and ratings of hunger and fullness in relationship to time of day; b) differences in preferences for high carbohydrate and high fat foods in the dietary phases; and c) palatability (e.g., pleasantness, texture, smell, appearance, and richness of all foods (global palatability) and individual foods (specific palatability) in the HM and HC diet phases.

1. Temporal patterns of eating. Each patient had a temporal pattern related to eating times. For example, one patient intiated the first eating episode of the day in all dietary phases within a one hour period, e.g., between 0730 and 0830; her last eating episode of the day in all dietary phases occurred within a stable window of time at the other end of her day, e.g., between 2030 and 2200, an eating period between 12 hours and 15 minutes and 14 hours at the maximum. By contrast, another patient's initial eating episode of the day began between 0700 and 1000 and the last eating episode ended between 2245 and 2400, an eating period of 17 hours. These aspects of eating pattern appear to be related to patients' personally preferred temporal pattern for getting up, for going to bed, and for engaging in life's activities at the time of ones perceived best energy level. Relationships between the temporal pattern of eating and temporal preferences for morning or evening were found as exemplified by the correlations between times eating started and stopped and the Horne Ostberg Morningness-Eveningness preference category. The total number of eating episodes for waking hours found in this study is roughly comparable to the British elderly (Gibney, 1997), and in the middle of the range reported by Kant (1995). This study suggests that adding identification of temporal preference may be a way of understanding temporal patterns of eating in women who have type 2 diabetes. Having type 2 diabetes carries with it many constraints related to time, e.g., times for taking insulin, and eating at certain times related to the peak action of the insulin or running the risk of

having a low blood sugar reaction. These subjects maintained a temporal pattern that was stable across the dietary phases; whether this pattern emerged as a kind of adaptive concession to living with diabetes, or whether it was their preferred pattern prior to the diagnosis of type 2 diabetes and had not changed, was not explored. However, temporal preference needs to be the template against which recommendations for living with diabetes are made and there are valid and reliable instruments with which to assess it.

2. Temporal pattern of eating: times of meals and snacks. The pattern of meals and snacks across the 24 hour period was also to be identified. This aspect of eating behavior has been a major variable in most other studies of eating patterns (de Castro, 1987, 1988, 1989, 1993; Gibney and Wolever, 1997). de Castro's definition of meal and snack was used in this study, e.g., a meal is any eating episode in which over 50 calories are consumed and which occurs at least 30 minutes since the termination of the last eating episode; a snack is any eating episode consisting of less than 50 calories. However, the definition of meals and snacks differs among studies; de Castro has several other less restrictive definitions to use when analyzing data in his studies, so use of this definition was somewhat arbitrary. Although data related to meals and snacks and time of day was collected, it became clear that every meal and snack consumed during the HM and HC dietary phases was going to reflect the caloric level and particular composition of the diet

for that particular dietary phase for that particular patient. Therefore, further exploration of the data related to meals and snacks was dropped.

3. Temporal pattern of eating: hunger and fullness. Data related to hunger and fullness were collected, as with other aspects of temporal pattern, for an 8-day period at the midpoint of the HM and HC diet phases. Both hunger and fullness were marked on a paper and pencil record, using a 5-point scale for each sensation adapted from the work of Reiff and Reiff (1992) (see Appendix forms). A category called "neither-hungry-nor-full" was located in the center of the page; hunger ratings (1 to 5) were marked in an area downward from the center of the page; fullness ratings (1 - 5) were marked in an area upward from the center of the page. An optimal recording would have required the patient to connect markings for these three conditions e.g., hunger, fullness, neither-hunger-nor-fullness, to each other across the hours of recording from left to right on the form. An additional space on the form provided a place for the subject to write down the time and the foods that were eaten. Unanticipated problems with recording on this form were encountered. Some patients could not identify what the feeling of hunger was, and consequently made few of their markings in the hunger section, and most of their markings in the "neither hunger nor full" or fullness section of the paper and pencil form. Secondly, four of the subjects were working in jobs which made hourly recording difficult, so that lapses in the recording distorted what was actually experienced. There was missing data related to foods that, due to the design of the

study and the method of checking food intake each day, were known to have been eaten. The form turned out to be complicated and although data was obtained, not all of it was usable. In the end, data related to episodes of hunger, fullness and neither-hunger-nor-fullness were identified from the data within the HM and HC dietary phases, but relationships between these sensations, meals and snacks, and time of day were not explored due to poor quality of some of the data.

4. Preferences for high carbohydrate and high fat foods in dietary phases. This study objective was met through a taste testing procedure (Mattes, 1993) that was administered at the end of the HC and the end of the HM dietary phases to diabetic subjects, and to a comparison group of 10 nondiabetic obese women matched in terms of age and BMI.

There were significant differences in ratings of liking at the end of the HM and HC dietary phases; however, these significant ratings could have occurred by chance alone. There were two reasons for comparing ratings of liking after HM to those after HC: a) obese women have a preference for fat, and b) liking for fat persists even though it is not being ingested. It was believed that high fat palatable foods would be rated higher (closer to 1.00) at the end of the dietary phase least likely to provide such foods, e.g., HC diet. Chocolate milk made with whipping cream or whipping cream with oil, is a high-fat, high carbohydrate food and can be used to illustrate what happened. After HM, the mean rating for the combined

sensory characteristics of chocolate milk made with whipping cream (creaminess, oiliness, sweetness and pleasantness) was 2.4 ± 1.35 versus 2.43 ± 1.07 after HC.

On the other hand, the mean ratings for the combined sensory characteristics of chocolate milk made with whipping cream/oil were 2.63 ± 1.26 after HM and 2.30 ± 0.90 after HC, meaning that the chocolate milk made with the highest level of fat was more preferred after HC. This finding would seem to support the idea that the higher the fat in the particular sample after HC, the greater the rating of liking for the sample, but none of the results were supported at the level of significance. If subjects cannot actually detect differing fat levels within food, the rating of palatability in taste testing is really a response to a combination of the sensory characteristics of the food (creaminess, sweetness, pleasantness), the food itself, and other factors interacting together. The rating scheme used here provides data about only a small part of this integrated response. Finally, the methodology used to collect data for this study objective did not encompass exploration of the relationship between the palatability of a single food and preference for the food.

Significant differences emerged from the ratings of liking and palatability in the taste testing when comparing the diabetic women to those who were nondiabetics. The differences did not occur in foods that were high-fat or high in carbohydrate content; rather, they occurred in foods that had the sensory characteristics of saltiness, sourness, and bitterness. It could be that there were deficits in taste function in the diabetic women which blocked their ability to detect

these sensory characteristics. Taste is an integrated function of taste and smell; traditional testing of taste and additional testing of olfaction should be done prior to taste testing in the manner of Mattes (1993). Finally, there is evidence that there are distinct groups of tasters who are sensitive to bitter. This dimension of taste function was not explored in the study.

5. Global palatability of the HM and HC diet and specific palatability of foods in the HM and HC diets. Paper and pencil instruments were used to identify palatability of the diet as a whole and of specific foods in the diet. Few significant differences were found in these two approaches to estimates of palatability for either diet, and both were rated as palatable.

B. The second objective of the study was to test the effect of increased levels of monounsaturated fatty acids in the diabetic diet on lipids, lipoproteins, apolipoproteins, and lipid peroxidation. This objective of the study was completed congruent with the study design; the findings have already been discussed in a previous section. Some conclusions will be offered about the design of the study, patient criteria, and measurement of dependent variables such as lipid peroxidation.

1. Study design. This study met all but one of the desirable design criteria for a feeding study examining fatty acid effects in humans (Kris-Etherton and Dietzchy, 1997). Where this study is weak is in having sufficient subjects to achieve statistical power, e.g., the sample needed to be 20 rather than 10 to provide 80% power of analysis. The decision to study 10 patients was made on the basis of

available funding, personnel, and work load for the Clinical Research Center. However, the study sample can be extended in the future, given that the sufficient elements of the design are retained.

2. Study criteria. Two study criteria were adapted to enter the 10 women who completed the study: BMI and cognitive function testing results.. The pool of 10 available women had an average BMI that exceeded study parameters, but all other study parameters meet study criteria. A decision was made to hold to all other parameters but not the BMI parameters. This meant that markedly obese women were entered into the study. The factor of their obesity may well have affected findings; whatever the effect was, it was not due to weight loss because the HM and HC dietary phases were isocaloric.

The cognitive function testing at the outset of the study showed that 6 of the 10 subjects had mild to moderate deficits in some areas of cognitive function. It was difficult to envision how these findings would affect an individual subject's participation in the study, but it is very likely that participation was affected by it. Subjects had small breaks from the study protocol and other responses such as lack of judgment in decision making that affected one or both phases of the study. However, daily contact between subjects, study personnel, and research center dietitians made it possible to find a variety of solutions to these problems, and all the subjects completed the study. Cognitive testing should have been repeated at the end of each study phase. This would have made it clear whether or not

improvements in glycemic control that were achieved in each dietary phase had also improved cognitive function.

Finally, subjects in this study were on combination therapy (oral agent plus insulin). The criteria for and efficacy of combination therapy has been a matter of controversy. However, it is being used with type 2 diabetics who are further along in the natural history of type 2 diabetes than any of the subjects in HM diet studies reviewed in Chapter 2 (Riddle, Hart, Bouma, Phillipson and Youker, 1989; Riddle, 1990). Therefore, this subjects in this study are a subpopulation of type 2 diabetics who are unique and greater characterization of their stage of type 2 diabetes is needed.

3. Measurement of lipid peroxidation. At the time of the study, the approaches to measure lipid peroxidation were to measure byproducts of lipid peroxides as malondialdehyde (TBARS) in plasma and urine, and to measure lipid peroxidation in a system where LDL oxidation is stimulated by an oxidant such as copper. However, other methods of measuring lipid peroxidation are emerging, e.g., the TRAP method in plasma; headspace gas chromatography analysis for aldehydes generated by lipid peroxides (Ceriello, Lizzo, Bortolotti, Russo, Motz, Tonutti, Crescentini, and Tabora, 1998; Frankel, 1997). It has also been found that in diabetics with poor glycemic control, oxidized lipids in the diet can be detected postpandially in chylomicrons, thus increasing the likelihood that the oxidized lipid might be passed along to the LDL particle via this route. A system of measuring

oxidized lipids ingested in the diet, oxidation processes generated within the body affecting lipoproteins such as LDL, and byproducts of lipid peroxidation such as can be found in urine or other body fluids may need to be developed to determine the net amount of lipid peroxidation that is occurring (Staprāns, Xian-Mang, Hardman and Feingold, 1999).

Recommendations for Future Research

Additional outpatient feeding studies exploring the effects of diets enriched with monounsaturated fatty acids need to be conducted with subpopulations of persons with type 2, e.g. postmenopausal women, those who are obese, those with lipid abnormalities associated with type 2 diabetes, those on combination therapy, to replicate the findings of this study. The HM diet needs to be tested to determine if, in free living subjects, weight gain is an inevitability. This could also be tested in a study design where women with type 2 are provided the HM diet for a period, followed by a period of time during which they learn to prepare the diet themselves, and finally, a period of HM diet where subjects are fully independent in preparing and eating the diet (Pi-Sunyer, Resnick, Maggio, McCarron, Reusser, Morris, Hatton, Stern, Metz, Haynes, Snyder, Oparil, Clark, Kris-Etherton, McMahon, 1999; Wing, 1997).

The lipid-related dependent variables in HM studies could be expanded to include newer methods of measuring lipid peroxidation, or measures of the effect of monounsaturated fatty acids on immune function, markers of atherosclerotic

processes, other lipoproteins e.g., dense LDL and VLDL particles, postprandial lipid responses to the HM diet, and/or changes in insulin resistence through glycemic clamp techniques.

Further studies related to eating behavior in type 2 diabetes present opportunities and dilemmas, but need to be conducted. On the one hand, it is desirable to know the composition of the diet, a factor that is best accomplished in the context of a feeding study. On the other hand, the free-living eating behavior of women with type 2 diabetes may be studied by techniques adapted from those suggested by Booth, 1999 in which focus groups are conducted with successful and unsuccessful dieters of women with type 2 diabetes for the purpose of identifying what works best in the context of free living to achieve the aims of therapeutic diets. Development of instruments which discriminate between palatability, liking, pleasantness, preference, and various sensory characteristics which characterize foods that are preferred and/or pleasant, are needed.

A different approach to research on eating behavior in type 2 diabetes would focus on better characterization of the intrameal and postprandial physiological dynamics of gastric distention, hormonal and neuropeptide responses associated with ingestion of the HM diet such as have been recently demonstrated by Jones, Kwiatek, Berry, Samson, Kong and Horowitz (1999). Characterization of the incidence and consequence of 6-N-propylthiouracil (PROP) tasters and nontasters among women with type 2 diabetes could also be explored. Development of better

paper and pencil tools for measurement of hunger and fullness is needed, and use of designs and/or instruments utilized in eating disorders that better characterize the impact of knowledge, attitudes, and beliefs on eating behavior could be incorporated in studies of eating behavior of women who have type 2 diabetes.

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APPENDICES

APPENDIX TABLES

APPENDIX TABLE 1

Effect of high monounsaturated (HM) diets compared to baseline/habitual diets on lipid and glycemic variables.

Investigator	HM diet (%)				Baseline diet (%)				Total cholesterol (mmol/L)	Triglyceride (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Hemoglobin A1C (%)
	Fat	S	M	P	Fat	S	M	P					
Dimiatradis [1996]	40	13	20	7	40	18	12	10	-0.50[1]	+0.10[1]	-0.30[1]	-0.10*[1]	-0.30[1]
Griffin [1996]	40	13	20	7	40	18	12	10	+0.30[1]	-0.20[1]	-0.10[1]	+0.20*[1]	-

Legend:

HM = high monounsaturated diet; S = saturated fat; M = monounsaturated fat; P = polyunsaturated fat
1= + value denotes that value is greater/higher after HM than after baseline; - denotes that value is lower/less after HM than after baseline
* = $p \leq 0.05$

APPENDIX TABLE 2

Effect of high monounsaturated (HM) compared to high carbohydrate (HC) diets on lipid and glycemic variables.

Investigator	HM diet (%)				HC diet (%)				Total cholesterol (mmol/L)	Triglyceride (mmol/L)	VLDL-cholesterol (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Fasting glucose	Fasting insulin (pmol/L)	Hemoglobin A1C (%)
	Fat	S	M	P	Fat	S	M	P								
Garg [1989]	50	10	33	7	25	9	9	6	- 0.23 [1]	- 0.62* [1]	-0.38 * [1]	+ 0.06 [1]	+ 0.10* [1]	- 0.89* [1]	-	+ 0.30 [1]
Rivellese [1990]	43	8	31	4	21	5	2	14	+ 0.12 [1]	- 0.28* [1]	- 0.13 * [1]	+ 0.33 [1]	+ 0.01 [1]	-	-	-
Bonanome [1991]	40	10	25	5	25	10	10	4	+ 0.30 [1]	+ 0.10 [1]	-	+ 0.10 [1]	+ 0.10 [1]	+ 0.40 [1]	-	- 0.20 [1]
Parillo [1992]	40	7	29	4	20	5	13	2	+ 0.16* [1]	- 0.16* [1]	- 0.21* [1]	-	+ 0.04 [1]	+0.36 [1]	-	- 0.20 [1]
Garg [1992]	45	5	31	10	20	3	11	6	- 0.65* [1]	-1.69* [1]	- 0.64* [1]	-0.06 [1]	+ 0.07 [1]	+0.01 [1]	-	-
Rasmussen [1993]	50	10	30	7	32	11	11	7	+ 0.70 [1]	HM=HC	-	+0.30 [1]	HM=HC	- 0.70* [1]	- 0.32* [1]	-
Lerman [1994]	40	11	24	5	20	6.6	6.6	6.6	- 0.24* [1]	-0.36* [1]	-	- 0. 10 [1]	HM=HC	- 0.73* [1]	-	-
Campbell [1994]	37	7	22	8	22	7	8	7	- 0.40 [1]	- 0.30* [1]	-	- 0.20 [1]	HM=HC	+ 0.20 [1]	- 3.0 [1]	-
Garg [1994]	45	10	25	3	30	10	10	10	- 0.10 [1]	- 0.44* [1]	- 0.15* [1]	HM=HC	+ 0.40 [1]	+ 0.20 [1]	HM=HC	+ 0.30 [1]
Blades [1995]	40	10	25	10	30	10	10	10	- 0.03 [1]	- 0.44* [1]	- 0.11* [1]	+ 0.90 [1]	+ 0.10 [1]	-	-	-
Walker [1995]	36	5	20	11	21	4	10	9	+ 0.07 [1]	- 0.16 [1]	- 0.10 [1]	+ 0.18 [1]	+ 0.02 [1]	- 0.10* [1]	+ 0.40 [1]	+ 0.10 [1]
Rasmussen [1994]	50	10	30	7	32	11	11	7	-	-	-	-	-	-	. - 0.70 [1]	+ 0.20 [1]
Parillo [1996]	40	7	29	4	20	5	13	2	HM=HC	-0.24-26* [2]	-	-	HM=HC [2]	-0.20 to 0.30 [2]	[2]0.60-1.8	-
Low [1996] Gumbiner [1998]	70	8	50	13	10	1	1	8	-0.1* [1]	-0.80* [2]	-	+0.10 [2]	HM=HC	-0.80* [2]	-	-
Heilbronn [1999]	32	7	15	9	10	4	3	2	-0.30	+0.03	-	-0.04	-0.07	-0.46*	-	-0.25*

Legend: HM = high monounsaturated diet; HC = high carbohydrate diet; S = saturated fat; M = monounsaturated fat; P = polyunsaturated fat * = $p \leq 0.05$

1= + value is greater/higher after HM than after HC; - denotes that value is lower/less after HM than after HC

2 = + value is greater/higher after HM than after HC; - denotes that value is lower/less after HM than after HC; first no. denotes mild diabetes; second no. denotes moderate diabetes

APPENDIX TABLE 3

Effect of HM-cis and HM-trans diets on lipid and glycemic variables

Investigator	HM *cis* diet (%)				HM *trans* diet (%)				Total cholesterol (mmol/L)	Triglyceride (mmol/L)	VLDL-cholesterol (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Fasting glucose	Fasting insulin (pmol/L)	Hemoglobin A1C (%)
	Fat	S	M	P	Fat	S	M	P								
Christiansen [1997]	30	5	20	5	30	5	20	5	- 0.14 [1]	- 0.03 [1]	-	- 0.10 [1]	- 0.02 [1]	- 0.60 [1]	-3.0 [1]	- 0.02 [1]

Legend: HM = high monounsaturated diet; S = saturated fat; M = monounsaturated fat; P = polyunsaturated fat
1= + value denotes that value is greater/higher after HM cis than after HM trans ; - denotes that value is lower/less after HM cis than after HM trans
* = $p \leq 0.05$

APPENDIX TABLE 4

Effect of HM diets compared to high polyunsaturated (HP) diets on lipid and glycemic variables.

Investigator	HM diet (%)				HP diet (%)				Total cholesterol (mmol/L)	Triglyceride (mmol/L)	VLDL-cholesterol (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Fasting glucose	Fasting insulin (pmol/L)	Hemoglobin A1C (%)
	Fat	S	M	P	Fat	S	M	P								
Thomsen [1995]	49	10	30	7	49	9	10	27	+ 0.20 1	+ 0.10 [1]	-	+ 0.20 [1]	HM=HP	-	-	-
Parfitt [1994]	50	15	28	7	47	15	15	17	HM=HP	- 0.30 [1]	+ 0.03 [1]	- 0. 30 [1]	. . + 0.09 [1]	HM=HP	-	-

Legend: HM = high monounsaturated diet; S = saturated fat; M = monounsaturated fat; P = polyunsaturated fat
1= + value denotes that value is greater/higher after HM than after HP; - denotes that value is lower/less after HM than after HP
* = $p \leq 0.05$

APPENDIX TABLE 5

Comparison of HM feeding study designs and subject groups

Investigators	Design	Length of diet phases (wks)	Washout period	Sample size and sex	Study setting	Diabetes medicationss	Weight (Kgs)	BMI	Age
Garg [1989]	Random cross over	4	None	10 males	Inpatient	Regular insulin ac bk; ac dinner	88	29	56
Rivellese [1990]	Random crossover	2	None	8 3 female; 5 male	Free living; no food provided	50% on oral agents	_	22	45
Bonanome [1991]	HC, HM, HC	8	NA	19 9 female 10male	Free living, no food provided	All on oral agents	77	28.8	55
Parillo [1992]	Random crossover	2	None	10 3 female,7 male	Inpatient	50% on oral agents	-	26.7	53
Garg [1992]	Crossover	4	None	10 males	Inpatient	None	86.7	27.7	61.5
Rasmussen [1993]	Crossover	3	Yes	15 5 female 10male	Outpatient some food provided	50% on oral agents	80.5	27.0	57
Lerman Gerber [1994]	Random crossover	4	Yes	12 females	Free living	Most on oral agents	66	28.0	56
Campbell [1994]	Random crossover	2	Yes	10 males	Free living	10% on oral agent	-	26.5	55
Parfitt [1994]	Random crossover	6	Yes	13 males	Free living	77% on oral agents	-	28.4	58
Garg [1994]	Random crossover	6	Yes	42 9 female33 male	Outpatient feeding study	100% on oral agent	-	28.1	58
Blades [1995]	Random crossover	6	Yes	10 males	Outpatient feeding study	100% on oral agents	-	28.6	61.3
Walker [1995]	Random crossover	12	Yes	24 15female 9 male	Free living	Some on oral agents	-	29.2	58.3
Rasmussen [1994]	Crossover	3	Yes	15 5female 10male	Outpatient some food provided	77% on oral agent	80.5	27.0	57
Parillo [1996]	Random crossover	2	None	18 Sex not given	Inpatient	50% on oral agent	-		

APPENDIX TABLE 5 (Continued)

Investigators	Design	Length of diet phases (wks)	Washout period	Sample size and sex	Study setting	Diabetes medicationss	Weight (Kgs)	BMI	Age
Dimitriadis [1996]	Baseline–>HM	4	NA	9 4female 5 male	Outpatient some food provided	78% on oral agents	-	-	63
Griffen [1996]	Baseline–>HM	4	NA	8 6 female 2 male	Outpatient some food provided	88% on oral agent	-	27.9	46.8
Gumbiner [1998]	3 phases 2 parallel groups	6	NA	17 9 female 8 male	Outpatient feeding study	None	CHO gp: 110.4 HM gp: 101.8	CHO: 37.2 HM: 36.3	CHO: 51 HM: 55
Christiansen [1997]	Random crossover	6	No	16 7female 9 male	Outpatient some food provided	None	98	33.5	
Heilbronn [1999]	Randomized to 3 groups	12	NA	35 27female 8 male	Outpatient some food provided	49% on oral agents	-	HC: 33.3 HM: 33.6	HC: 58.9 HM: 58.9
Totals	14/18: crossover 5/18: other design		Yes: 8/14 No: 6/14	301 Females: 114 Males: 156	Inpatient: 4 Outpatient:: 8 all food: 3 some food: 6 Free living: 6	No meds: 3 Oral: 31 Insulin: 1			
Average ± SD		4.55 ± 2.54					86.4 kg ± 14.1	28.7 ± 3.58	55.7 ± 4.77
Range		2 - 12		8 - 42			66 - 110.4	22 - 37.2	45 - 63

APPENDIX TABLE 6

Final means for every variable in HM and HM diet phases

Variable	HM		HC		P/NP	t/Wilcoxon	p
Lipid profile	x̄	SE	x̄	SE			
Total cholesterol (mg/dL)	217.01	8.77	202.46	8.38	P	3.06	0.01
Triglycerides (mg/dL)	252.24	46.66	241.69	36.65	NP	-0.87	0.39
LDL-C (calculated, mg/dL)	121.48	9.37	118.56	7.78	P	0.553	0.6
HDL-C (mg/dL)	39.09	8.87	36.20	7.43	P	1.39	0.20
Lipoproteins							
VLDL-C (mg/dL)	33.25	8.22	37.32	12.08	NP	-0.76	0.44
VLDL-triglyceride (mg/dL)	149.87	41.56	192.78	57.38	NP	-1.48	0.14
LDL-C (calculated, mg/dL)	96.65	11.49	98.52	9.42	P	-0.25	0.81
LDL-triglyceride (%)	8.92	2.58	9.49	3.06	NP	-1.27	0.52
LDL-free cholesterol (%)	8.18	0.62	6.87	1.10	P	3.59	0.006
LDL-cholesterol ester (%)	37.22	0.98	38.43	1.6	P	2.93	0.02
LDL-phospholipid (%)	20.30	1.26	19.09	0.52	P	2.93	0.02
LDL-Protein (%)	25.60	0.99	26.12	2.10	P	-0.44	0.67
LDL-triglyceride (nmol/mg LDLPro)	406.00	129.96[1]	410.50	112.86[1]	P	-0.111	0.91
LDL-free cholesterol (nmol/mg LDLPro)	845.70	133.50[1]	678.90	73.89[1]	P	5.15	0.0006
LDL-cholesterol ester (nmol/mg LDLPro)	2298.10	452.40[1]	2303.40	477.67[1]	P	-0.02	0.98
LDL-phospholipid (nmol/mg LDLPro)	1045.50	190.96[1]	944.70	79.80[1]	NP	1.07	0.57
LDL-Protein (mg/dL)	81.80	33.01[1]	84.63	21.30[1]	P	-0.33	0.75

APPENDIX TABLE 6 (Continued)

Variable	HM		HC				
Apolipoproteins	x̄	SE	x̄	SE	P/NP	t/Wilcoxon	P
A-1 (mg/dL)	113.35	4.64	114.80	6.87	P	-0.39	0.71
B (mg/dL)	93.45	9.23	88.8	8.25	NP	0.46	0.65
TBARS							
Plasma (nmol/mL)	1.39	0.08	1.51	0.08	P	-7.56	0.000
Urine (nmol/μmol creatinine)	1.00	0.18	2.10	0.73	NP	-1.89	0.06
Urine (nmol/day)	13642	3798	28463	11093	NP	-1.50	0.11
Urine (nmol/day/Kg body weight)	139.67	37.44	281.27	109.86	NP	-1.58	0.11
Fatty acids in LDL (weight percent)							
12:0	0.1	0.14	0.1	0.1	NP		0.08
14:0	0.7	0.4	1.0	0.3	P		0.08
14:1	0.2	0.1	0.0	—	NP		0.32
15:0	0.2	0.1	0.2	0.1	NP		0.11
16:0	19.5	1.5	21.5	2.2	P		0.02
16:1n7	2.2	0.8	2.7	0.9	P		0.04
18:0	5.8	0.6	6.1	0.5	P		0.03
18:1n9c	23.89	2.5	15.5	1	P		0.0
18:1n7	1.4	0.2	1.3	0.2	P		1.4

APPENDIX TABLE 6 (Continued)

Variable	HM		HC				
Fatty acids in LDL continued	x̄	SE	x̄	SE	P/NP	t/Wilcoxon	p
19:0	0.0	0.3	0.0	0.0	—		—
18:2n6c	23.6	3.6	30.3	4.0	P		0.00
20:0	0.4	0.2	0.3	0.1	NP		0.8
18:3n3c	0.3	0.1	0.4	0.1	NP		0.005
20:1n9	0.2	0.2	0.1	0.1	NP		0.1
21:0	0.1	0.1	0.1	0.1	NP		0.1
20:2n6	0.1	0.1	0.2	0.0	NP		0.00
20:3n6	0.7	0.3	1.7	0.2	NP		0.24
22:0	1.3	0.2	0.8	0.1	P		0.0001
20:4n6	8.0	1.8	8.2	2.0	P		0.37
23:0	0.5	0.1	0.4	0.1	NP		0.05
20:5n3	0.6	0.1	0.4	0.1	P		0.0001
24:0	1.0	0.4	0.7	0.1	NP		0.007
24:1	1.0	0.2	1.1	0.3	P		0.27

APPENDIX TABLE 6 (Continued)

Variable	HM		HC				
Fatty acids in LDL continued	x̄	SE	x̄	SE	P/NP	t/Wilcoxon	p
22:5n3	0.3	0.1	0.3	0.1	NP		0.57
22:6n3	1.4	0.2	1.1	0.2	NP		0.007
Saturated	29.6	1.5	31.1	2.3	P		0.04
MUFA	29.3	2.0	21.2	1.1	P		0.000
PUFA	36.0	3.5	42.9	3.0	P		0.00
LDL oxidation							
Lag time (mins)	100.67	8.15	63.22	5.21	NP	2.55	0.01
Rate (nmol CD/mg LDL-C/min)	4.40	0.24	7.66	0.48	P	-7.48	0.0001
Concentration (nmol CD/mg LDL-C)	310.11	11.66	352.11	9.32	NP	-2.55	0.01
Vitamins in LDL							
Vitamin C (mg/dL)	0.96	0.12	0.97	0.13	P	-0.10	0.92
α tocopherol (nmol/mg LDLPro)	13.27	3.22	10.38	2.87	P	3.37	0.008
γ tocopherol (nmol/mg LDLPro)	1.72	0.81	1.84	0.96	NP	-0.87	0.39
Glycemic variables	x̄	SE	x̄	SE	P/NP	t/Wilcoxon	p
Glycosylated Hb (% total)	9.56	0.41	10.35	0.41	P	-1.67	0.13
Fructosamine (mmol/L)	2.15	0.07	2.39	0.07	P	-3.79	0.004
Week 2/week 4 fasting blood sugar	146.40	10.63	191.70	17.1	P	-2.54	0.03
Final weekly AM capillary blood sugar	127.46	11.12	136.28	8.75	P	-1.08	0.31

APPENDIX TABLE 6 (Continued)

Variable	HM		HC				
Glycemic variables continued	x̄	SE	x̄	SE	P/NP	t/Wilcoxon	p
Mean weeks 5,6 AM capillary blood sugar	126.65		134.90		P	-8.25	0.22
Final weekly PM capillary blood sugar	130.63	10.07	142.67	10.86	P	-1.87	0.10
Mean weeks 5,6 PM capillary blood sugar	128.23		145.06		P	-16.82	0.05
Final free insulin (μU/ml)	16.98	2.38	17.23	2.41	P	-0.14	0.89
Final free insulin (pmol/ml)	121.83	17.07	123.63	17.3	P	-0.14	0.89

Legend: HM=high monounsaturated diet; HC = high carbohydrate diet; SE = standard error; P/NP=parametric, nonparametric distribution

APPENDIX TABLE 7

Initial and final means, change scores and p values: HM and HC diet phases

	HM Diet				HC Diet			
Variable	**Initial mean ± SE**	**Final mean ± SE**	**▲**	**p**	**Initial mean ± SE**	**Final mean ± SE**	**▲**	**p**
Lipids Total cholesterol (mg/dL)	224 ± 9.59	217 ± 8.77	6.83	0.20	227 ± 16.8	203 ± 8.38	24.6	0.04
Triglycerides (mg/dL)	236 ± 22.8	252 ± 46.7	16.2	0.50	300 ± 69.0	242 ± 36.7	58.5	0.07
LDL (calculated, mg/dL)	140 ± 9.60	121 ± 9.37	18.7	0.03	132 ± 10.1	119 ± 7.78	13.1	0.07
HDL-C (mg/dL)	35.9 ± 1.87	39.1 ± 2.80	3.17	0.01	35.1 ± 2.00	36.2 ± 2.32	1.15	0.28
Lipoproteins VLDL-C (mg/dL	25.8 ± 5.98	33.3 ± 8.22	7.44	0.30	41.1 ± 15.1	37.3 ± 12.1	3.76	0.20
VLDL-triglyceride (mg/dL)	127 ± 20.0	150 ± 41.6	23.2	0.90	202 ± 71.9	193 ± 57.4	9.16	0.70
LDL-C (mg/dL)	105 ± 11.1	96.6 ± 11.5	8.73	0.21	103 ± 7.98	98.5 ± 9.42	4.57	0.28
LDL-triglyceride (%)	8.86 ± 0.58	8.92 ± 0.82	0.07	0.93	10.2 ± 0.89	9.49 ± 0.97	0.70	0.14
LDL-free cholesterol (%)	8.16 ± 0.28	8.18 ± 0.20	0.02	0.94	7.55 ± 0.23	6.87 ± 0.35	0.68	0.13
LDL-cholesterol ester (%)	35.5 ± 1.41	37.2 ± 0.98	1.68	0.27	37.1 ± 0.74	38.4 ± 1.60	1.35	0.28
LDL-phospholipid (%)	21.2 ± 0.55	20.3 ± 0.40	0.85	0.03	19.6 ± 0.40	19.1 ± 0.16	0.49	0.33
LDL-protein (%)	26.3 ± 1.21	25.5 ± 0.99	0.83	0.95	25.6 ± 0.51	26.1 ± 0.66	0.52	0.46

Legend: SE: standard error of the mean; HM= high monounsaturated diet; HC = high carbohydrate diet; ▲ = difference between initial and final mean

APPENDIX TABLE 7 (Continued)

Variable	HM Diet				HC Diet			
	Initial mean ± SE	Final mean ± SE	Δ	p	Initial mean ± SE	Final mean ± SE	Δ	p
LDL-triglyceride (nmol/mg LDL protein)	388 ± 29.7	406 ± 41.1	18.4	0.67	451 ± 35.1	411 ± 35.7	40.1	0.09
LDL-free cholesterol (nmol/mg LDL protein)	824 ± 56.1	846 ± 42.2	21.9	0.64	769 ± 33.7	679 ± 26.3	90.4	0.03
LDL-cholesterol ester (nmol/mg LDL protein)	2135 ± 146	2298 ± 143	163	0.37	2249 ± 80.1	2303 ± 151	54.3	0.66
LDL-phospholipid (nmol/mg LDL protein)	1054 ± 61.2	1046 ± 60.4	8.70	0.88	990 ± 41.5	945 ± 25.2	45.7	0.11
LDL-protein (mg/dL)	93.8 ± 9.28	81.8 ± 10.4	12.0	0.08	88.2 ± 6.36	84.6 ± 6.74	3.55	0.54
Apolipoproteins A-1 (mg/dL)	115 ± 3.70	113 ± 4.64	1.40	0.80	121 ± 5.86	115 ± 6.87	5.80	0.20
B (mg/dL)	94.9 ± 5.63	93.5 ± 9.24	1.40	0.80	90.1 ± 9.86	88.8 ± 8.25	1.25	0.70
TBARS Plasma (nmol/mL)	1.51 ± 0.09	1.39 ± 0.08	0.12	0.001	1.52 ± 0.07	1.51 ± 0.08	0.01	0.90
Urine (nmol/μmol creatinine)	1.91 ± 0.55	0.98 ± 0.18	0.93	0.13	1.86 ± 0.56	2.10 ± 0.73	0.24	0.80
Urine (nmol/day)	23901 ± 7149	13641 ± 3798	10260	0.10	25800 ± 8785	28463 ± 11092	2663	0.40
Urine (nmol/day/Kg body wt)	202 ± 47.0	140 ± 37.4	62.5	0.09	245 ± 69.7	110 ± 36	0.36	0.40

APPENDIX TABLE 7 (Continued)

Variable	HM Diet				HC Diet			
	Initial mean ± SE	Final mean ± SE	▲	p	Initial mean ± SE	Final mean ± SE	▲	p
LDL Oxidation Lag time (minutes)	65 ± 6.30	101 ± 8.15	35.7	0.01	84.0 ± 8.15	63.5 ± 4.67	20.5	0.02
Rate (nmol conjugated dienes/mg LDL-C/min)	7.94 ± 0.76	4.40 ± 0.24	3.54	0.003	5.49 ± 0.39	7.55 ± 0.48	2.06	0.009
Concentration (nmol conjugated dienes/ mg LDL-C)	384 ± 16.4	310 ± 11.7	74.3	0.006	351 ± 9.24	353 ± 8.37	1.50	0.87
α tocopherol (nmol/mg LDL protein	9.48 ± 6.10	13.3 ± 7.85	3.79	0.0004	12.5 ± 2.51	10.4 ± 0.91	2.07	0.51
γ tocopherol (nmol/mg LDL protein	3.49 ± 1.26	1.72 ± 0.71	1.77	0.001	3.44 ± 0.70	1.84 ± 0.30	1.60	0.005
Vitamin C (mg/dL)	0.70 ± 0.10	0.96 ± 0.12	0.26	0.07	0.68 ± 0.13	0.97 ± 0.13	0.29	0.02
Fatty acids in LDL 16:0	21.0 ± 0.05	19.5 ± 0.46	1.48	0.02	21.2 ± 0.90	21.5 ± 0.70	0.29	0.48
18:0	5.85 ± 0.17	5.78 ± 0.19	0.07	0.75	5.84 ± 0.21	6.06 ± 0.16	0.22	0.32
18:1n9c	15.7 ± 0.50	23.9 ± 0.80	8.20	0.00	16.6 ± 0.73	15.5 ± 0.32	1.05	0.07
18:2n6c	30.3 ± 0.99	23.6 ± 1.14	6.70	0.0002	27.7 ± 1.02	30.4 ± 1.25	2.62	0.02
18:3n3c	0.53 ± 0.04	0.27 ± 0.03	0.26	0.005	0.53 ± 0.07	0.41 ± 0.04	0.12	0.20

APPENDIX TABLE 7 (Continued)

	HM Diet				HC Diet			
Variable	**Initial mean ± SE**	**Final mean ± SE**	**▲**	**p**	**Initial mean ± SE**	**Final mean ± SE**	**▲**	**p**
20:2n6	0.22 ± 0.02	0.14 ± 0.03	0.08	0.04	0.26 ± 0.04	0.24 ± 0.01	0.02	0.71
20:3n6	1.55 ± 0.07	1.71 ± 0.09	0.16	0.13	1.52 ± 0.08	1.71 ± 0.07	0.19	0.02
20:4n6	8.16 ± 0.52	8.00 ± 0.58	0.16	0.58	7.89 ± 0.46	8.20 ± 0.62	0.31	0.24
20:5n3	0.74 ± 0.07	0.55 ± 0.04	0.18	0.002	0.59 ± 0.05	0.44 ± 0.04	0.12	0.02
22:5n3	0.46 ± 0.04	0.32 ± 0.04	0.14	0.007	0.30 ± 0.04	0.28 ± 0.04	0.02	0.47
22:6n3	1.22 ± 0.10	1.36 ± 0.06	0.14	0.26	1.09 ± 0.09	1.12 ± 0.06	0.03	0.68
Saturated	30.4 ± 0.41	29.62 ± 0.48	0.81	0.09			1.96	0.51
Monounsaturated (MUFA)	21.9 ± 0.68	29.3 ± 0.62	7.34	0.000	22.6 ± 0.85	21.3 ± 0.34	1.32	0.08
Polyunsaturated (PUFA)	43.4 ± 0.99	36.0 ± 1.11	7.32	0.0001	40.1 ± 1.04	42.9 ± 0.93	2.78	0.02
Glycemic variables Fructosamine (mmol/L)	2.37 ± 0.07				2.84 ± 0.36			
Fructosamine (wk3)(mmol/L)	2.40 ± 0. 09		0.25[1]	0.005	2.36 ± 0.07		0.03[1]	0.84
Fructosamine (wk6)(mmol/L)		2.15 ± 0.07	0.22[2]	0.0008		2.39 ± 0.07	0.45[2]	0.15

1 = fructosamine difference score and p value based on means for week 3 and week 6
2 = fructosamine difference score and p value based on means for week 1 and week 6

APPENDIX TABLE 7 (Continued)

Variable	HM Diet				HC Diet			
	Initial mean ± SE	Final mean ± SE	▲	p	Initial mean ± SE	Final mean ± SE	▲	p
Random BS wk1 (mg/dL)	152 ± 17.8				188 ± 20.6			
Random BS wk2 (mg/dL)	188 ± 21.6				176 ± 12.0			
Random BS wk3 (mg/dL)	167 ± 13.8				177 ± 15.3			
Random BS wk4 (mg/dL)	146 ± 10.6				192 ± 17.1			
Capillary BS, wk1 (mg/dL)	153 ± 17.7				177 ± 13.9			
Capillary BS, wk2 (mg/dL)	163 ± 11.7				186 ± 11.8			
Capillary BS, wk3 (mg/dL)	154 ± 8.29				148 ± 9.22			
Capillary BS wk4 (mg/dL)	128 ± 10.1				173 ± 17.1			
AM capillary BS, wk1 (mg/dL)	158 ± 16.7							
AM capillary BS, wk2 (mg/dL)	153 ± 15.2		29.3	0.007 [3]	141 ± 11.6		14.1	0.06 [3]
AM capillary BS, wk5 (mg/dL)		123 ± 8.54	34.9	0.07 [4]		127 ± 9.28	28.1	0.008 [4]
AM capillary BS, wk6 (mg/dL)		127 ± 11.1	24.8	0.01 [5]		129 ± 8.34	11.6	0.18 [5]
PM capillary BS, wk1 (mg/dL)	173 ± 18.0		42.8	0.03 [6]	180 ± 10.9		34.9	0.03 [6]
PM capillary BS, wk2 (mg/dL)	165 ± 14.7		17.2	0.14 [7]	167 ± 9.73		20.7	0.06 [7]
PM capillary BS, wk 5 (mg/dL)		148 ± 11.0	25.5	0.11 [8]		145 ± 9.05	21.5	0.09 [8]

APPENDIX TABLE 7 (Continued)

	HM Diet				HC Diet			
Variable	**Initial mean ± SE**	**Final mean ± SE**	**▲**	**p**	**Initial mean ± SE**	**Final mean ± SE**	**▲**	**p**
PM capillary BS, wk 6 (mg/dL)		131 ± 10.1	34.8	0.07 [9]		145 ± 9.05	21.5	0.09 [9]
Mean, wk1,2 AM capillary BS	155 ± 15.6	127 ± 10.1	28.5	0.07	148 ± 11.4	128 ± 8.43	33.4	0.01
Mean, wk 1,2 PM capillary BS	162 ± 14.5	128 ± 7.36	33.4	0.03	173 ± 10	146 ± 8.47	27.8	0.04
Free insulin % bound	51.6 ± 3.57	49.7 ± 2.67	1.89	—	50.31 ± 3.2	49.4 ± 2.81	0.89	—
Free insulin (μU/mL)	15.9 ± 2.27	14.1 ± 2.38	1.09	0.49	17.3 ± 3.60	17.2 ± 2.41	0.02	0.99
Free insulin (pmol/mL)	114 ± 16.3	122 ± 17.1	7.79	—	124 ± 22.6	124 ± 17.3	0.14	—

3 = AM capillary BS difference score and p value based on means for week 2 and week 5
4 = AM capillary BS difference score and p value based on means for week 1 and week 5
5 = AM capillary BS difference score and p value based on means for week 2 and week 6
6 = PM capillary BS difference score and p value based on means for week 1 and week 6
7 = PM capillary BS difference score and p value based on means for week 2 and week 5
8 = PM capillary BS difference score and p value based on means for week 1 and week 5
9 = PM capillary BS difference score and p value based on means for week 2 and week 6

APPENDIX TABLE 8

Four day menu cycle for HM and HC Diets

Day One HM	HC
Sausage and gravy* over biscuits* Choice of juice 2% milk Tuna salad* sandwich on choice of bread with lettuce, tomato, mustard (optional) Marinated pasta salad with Italian dressing* Fruit Marinara sauce with ground beef over pasta with Parmesan cheese Sourdough bread with butter-blend spread Green tossed salad Choice of dressing Pumpkin bar*	Sausage and gravy over English muffin Oatmeal Choice of juice 2% milk Tuna salad* sandwich with lettuce, tomato and mustard (optional) Marinated pasta with Italian dressing* Fruit Marinara sauce with ground beef over pasta with Parmesan cheese Sourdough bread Green tossed salad Choice of salad dressing* Pumpkin bar*

Day Two HM	HC
Hearty pancakes* with butter-blend spread* and syrup Choice of juice 2% milk Ham salad* and cheddar cheese sandwich on choice of bread with lettuce, tomato, and mustard (optional) Green salad with choice of dressing* Fruit Oriental chicken with stir-fried vegetables White or brown rice Cheesy bread stick* Brownie* with chocolate frosting	Hearty pancakes* with butter and lite syrup Choice of juice Cornflakes 2% milk Ham salad and cheddar cheese sandwich on choice of bread with lettuce, tomato, and mayonnaise Soft pretzel with mustard Green salad with dressing of choice Fruit Oriental chicken with stir-fried vegetables White rice Cheesy bread stick* Wheatberry roll Brownie with chocolate frosting

* = Trisun oil incorporated into recipe

APPENDIX TABLE 8 (Continued)

Day Three	HM	HC
	Hashbrown breakfast quiche*	Hashbrown breakfast quiche in pastry*
	Choice of juice	Rice Krispies
	2% milk	Choice of juice
	Cream of broccoli soup*	2% milk
	Mexican cornbread muffin	Cream of broccoli soup
	Cheddar cheese	Bagel
	Carrot sticks	Cheddar cheese
	Fruit	Carrot sticks
	Calzone filled with cheeses and vegetables, topped with marinara sauce	Saltines
	Green tossed salad with choice of salad dressing*	Fruit
	Oatmeal cookie*	Calzone filled with cheeses and vegetables, topped with marinara sauce
		Green beans and corn
		Branola bread
		Oatmeal cookie

Day Four	HM	HC
	Muffins* (banana or applesauce)	Muffin (banana or applesauce)
	Butter-blend spread*	Cream of wheat
	Choice of juice	Choice of juice
	2% milk	2% milk
	Turkey sandwich with mayonnaise*, choice of bread, lettuce, tomato, mustard (optional)	Turkey sandwich with mayonnaise*, choice of bread, lettuce and tomato
	Green tossed salad	Clinical Research Center vegetable soup
	Choice of salad dressing*	Fat-free Saltines
	Meatloaf* patty	Carrot sticks
	Cheesy scalloped potatoes*	Canned fruit
	Steamed vegetable	Meatloaf patty
	Zucchini bread*	Cheesy scalloped potatoes
	Vanilla pie*	Steamed vegetable
		Wheatberry roll
		Banana
		Zucchini bread*
		Vanilla pudding with lite Cool-Whip

APPENDIX TABLE 9

Weekly compliance ratings in HM and HC diet phases

	HM	HC			
Question	Mean ± SE	Mean ± SE	P/NP	t./Wilcoxon	p
WEEK ONE					
Q1. How often have you been able to eat just foods provided by the study?	1.27 ± 0.27	1.23 ±0.32	NP	1.12	0.26
Q2. Things have happened that have interfered with doing twice daily CBS.	1.48 ± 0.68	0.73 ± 0.18	NP	1.36	0.17
Q3. A person has to be pretty creative to make study food taste better.	1.76 ± 0.67	1.70 ± 0.82	NP	0.15	0.87
Q4. The diet and foods in the study are helping my diabetes.	2.24 ± 0.84	1.87 ± 0.60	NP	0.84	0.40
Q5. I have had some alcohol to drink last week.	0.97 ± 0.17	1.08 ± 0.19	P	-0.76	0.47

	HM	HC			
Question	Mean ± SE	Mean ± SE	P/NP	t./Wilcoxon	p
WEEK TWO					
Q1. How often have you been able to eat just foods provided by the study?	1.39 ±0.28	1.14 ± 0.22	NP	0.21	0.83
Q2. Things have happened that have interfered with doing twice daily CBS.	1.57 ± 0.77	1.02 ±0.32	NP	0.56	0.57
Q3. A person has to be pretty creative to make study food taste better.	1.98 ± 0.80	1.36 ± 0.39	NP	-0.28	0.78
Q4. The diet and foods in the study are helping my diabetes.	1.97 ± 0.64	1.46 ± 0.52	NP	0.68	0.50
Q5. I have had some alcohol to drink last week.	1.14 ± 0.16	1.11 ± 0.16	P	-0.14	0.89

Legend: 1-always; 10-never; HM = high monounsaturated diet; HC = high carbohydrate diet; CBS = capillary blood sugar; P/NP = parametric/nonparametric test of means)

APPENDIX TABLE 9 (Continued)

Question	HM Mean ± SE	HC Mean ± SE	P/NP	t./Wilcoxon	p
WEEK THREE					
Q1. How often have you been able to eat just foods provided by the study?	1.59 ± 0.55	2.03 ± 0.85	NP	-0.14	0.89
Q2. Things have happened that have interfered with doing twice daily CBS.	1.00 ± 0.22	0.91 ± 0.21	NP	0.21	0.83
Q3. A person has to be pretty creative to make study food taste better.	1.77 ± 0.86	1.09 ± 0.26	NP	0.54	0.59
Q4. The diet and foods in the study are helping my diabetes.	1.72 ± 0.51	1.42 ± 0.43	NP	0.35	0.73
Q5. I have had some alcohol to drink last week.	1.22 ± 0.26	1.16 ± 0.14	NP	0.63	0.53

Question	HM Mean ± SE	HC Mean ± SE	P/NP	t./Wilcoxon	p
WEEK FOUR					
Q1. How often have you been able to eat just foods provided by the study?	1.02 ±0.14	1.39 ± 0.29	p	-1.59	0.15
Q2. Things have happened that have interfered with doing twice daily CBS.	0.75 ± 0.16	1.48 ± 0.72	NP	-0.90	0.37
Q3. A person has to be pretty creative to make study food taste better.	1.83 ± 0.77	2.97 ± 0.98	NP	-1.84	0.07
Q4. The diet and foods in the study are helping my diabetes.	1.32 ± 0.44	1.03 ± 0.34	NP	1.33	0.18
Q5. I have had some alcohol to drink last week.	1.11 ± 0.14	1.07 ± 0.20	P	0.25	0.80

APPENDIX TABLE 9 (Continued)

Question	HM Mean ± SE	HC Mean ± SE	P/NP	t./Wilcoxon	p
WEEK FIVE					
Q1. How often have you been able to eat just foods provided by the study?	1.05 ± 0.17	2.01 ± 0.42	NP	-2.19	0.03
Q2. Things have happened that have interfered with doing twice daily CBS.	0.73 ± 0.16	1.18 ± 0.32	NP	-2.21	0.03
Q3. A person has to be pretty creative to make study food taste better.	1.80 ± 0.77	2.11 ± 0.81	NP	-0.51	0.61
Q4. The diet and foods in the study are helping my diabetes.	1.74 ± 0.49	1.46 ± 0.52	NP	0.63	0.53
Q5. I have had some alcohol to drink last week.	1.15 ± 0.14	1.15 ± 0.13	P	-0.00	1.00

Question	HM Mean ± SE	HC Mean ± SE	P/NP	t./Wilcoxon	p
WEEK SIX					
Q1. How often have you been able to eat just foods provided by the study?	2.32 ±1.56	1.55 ± 0.37	NP	-0.67	0.50
Q2. Things have happened that have interfered with doing twice daily CBS.	0.88 ± 0.28	0.91 ± 0.24	NP	-1.63	0.10
Q3. A person has to be pretty creative to make study food taste better.	1.02 ± 0.40	1.82 ± 0.78	NP	-1.29	0.20
Q4. The diet and foods in the study are helping my diabetes.	1.22 ± 0.41	1.15 ± 0.37	NP	-0.68	0.50
Q5. I have had some alcohol to drink last week.	0.96 ± 0.20	1.05 ± 0.14	NP	-0.27	0.79

APPENDIX TABLE 10

Global ratings of palatability of HM and HC diets

(1 = very positive; 10 = very negative; all tests of differences of means based on Wilcoxon signed rank test; SE = standard error)

DAY ONE **HM** **HC**

Palatability components	Mean ± SE	Mean ± SE	Wilcoxon	p
Pleasantness	2.27 ± 0.55	1.97 ± 0.42	0.97	0.33
Tastiness	2.02 ± 0.43	1.86 ± 0.55	0.05	0.96
Texture	2.06 ± 0.44	2.33 ± 0.55	-0.65	0.51
Smell	2.67 ± 0.54	1.82 ± 0.49	2.09	0.04
Appearance	2.02 ± 0.42	1.86 ± 0.60	0.66	0.51
Richness	2.50 ± 0.55	2.48 ± 0.57	0.36	0.72

DAY TWO **HM** **HC**

Palatability components	Mean ± SE	Mean ± SE	Wilcoxon	p
Pleasantness	2.79 ± 0.62	2.22 ± 0.44	1.28	0.20
Tastiness	2.67 ± 0.57	2.11 ± 0.43	1.22	0.22
Texture	2.69 ± 0.65	2.13 ± 0.39	0.87	0.39
Smell	2.69 ± 0.54	2.11 ± 0.45	0.87	0.39
Appearance	2.10 ± 0.49	1.96 ± 0.35	0.26	0.80
Richness	2.62 ± 0.46	2.59 ± 0.45	0.61	0.54

APPENDIX TABLE 10 (Continued)

DAY THREE **HM** **HC**

Palatability components	Mean ± SE	Mean ± SE	Wilcoxon	p
Pleasantness	2.43 ± 0.45	2.00 ± 0.38	0.77	0.44
Tastiness	2.44 ± 0.43	2.47 ± 0.47	-0.65	0.51
Texture	2.70 ± 0.45	2.28 ± 0.36	1.38	0.17
Smell	2.89 ± 0.46	2.05 ± 0.40	2.65	0.008
Appearance	2.65 ± 0.47	2.54 ± 0.38	0.82	0.11
Richness	2.86 ± 0.45	2.54 ± 0.38	0.82	0.41

DAY FOUR **HM** **HC**

Palatability components	Mean ± SE	Mean ± SE	Wilcoxon	p
Pleasantness	2.41 ± 0.77	2.01 ± 0.45	-0.18	0.86
Tastiness	1.82 ± 0.36	2.17 ± 0.55	-0.53	0.59
Texture	1.91 ± 0.35	2.17 ± 0.49	-1.72	0.09
Smell	2.38 ± 0.54	2.01 ± 0.38	0.53	0.59
Appearance	1.93 ± 0.45	1.61 ± 0.32	0.47	0.64
Richness	2.07 ± 0.30	2.18 ± 0.39	-0.65	0.51

APPENDIX TABLE 11

Specific food palatability ratings during the four-day diet cycle of HM and HC

(Rating scale: 1 = like extremely; 9 = dislike extremely; all tests of differences between means are Wilcoxon signed rank tests; 1 = flavor or type varied depending on patient choice; 2 = subject could omit).

DAY ONE FOODS **HM** **HC**

Foods	Mean ± SE	Mean ± SE	Wilcoxon	p
Sausage/gravy	1.40 ±0.22	1.50 ± 0.18	-0.55	0.58
Biscuit/English muffin	1.55 ±0.24	1.70 ± 0.21	-0.43	0.67
Juice [1]	1.72 ± 0.32	1.55 ± 0.22	0.68	0.50
Tuna salad	1.55 ± 0.24	1.80 ± 0.29	0.42	0.67
Sandwich bread	2.50 ± 0.64	1.80 ± 0.21	1.15	0.25
Lettuce/tomato	1.78 ± 0.28	1.55 ± 0.17	1.66	0.10
Fresh/canned fruit	1.75 ± 0.28	1.65 ± 0.18	0.27	0.79
Marinated pasta salad	2.83 ± 0.53	3.25 ± 0.52	-1.20	0.23
Mustard [2]	2.44 ± 0.51	2.50 ± 0.34	-0.34	0.73
Spaghetti with marinara	1.65 ± 0.24	2.15 ± 0.42	-1.16	0.25
Green salad	1.60 ± 0.22	1.70 ± 0.17	-0.63	0.53
Pumpkin bar	1.30 ± 0.15	1.45 ± 0.23	-0.74	0.46

APPENDIX TABLE 11 (Continued)

DAY TWO FOODS **HM** **HC**

Foods	Mean ± SE	Mean ± SE	Wilcoxon	p
Pancakes	2.00 ±0.37	1.70 ± 0.21	0.71	0.48
Syrup (lite)	2.00 ± 0.42	1.55 ± 0.26	1.02	0.31
Juice [1]	2.20 ±0.40	2.10 ± 0.32	0.43	0.67
Butter	2.95 ± 0.68	2.35 ± 0.54	1.49	0.16
Ham slice	1.80 ± 0.30	1.70 ± 0.23	0.00	1.00
Slice of cheese	1.85 ± 0.22	1.90 ±0.24	-0.14	0.89
Lettuce/tomato	2.00 ± 0.36	1.45 ± 0.14	1.81	0.07
Fresh/canned fruit	1.39 ± 0.16	1.65 ± 0.13	-2.12	0.04
Sandwich bread	2.30 ± 0.56	1.55 ± 0.19	1.19	0.23
Mustard [2]	2.40 ± 0.45	2.05 ± 0.33	0.57	0.57
Mayonnaise	4.00 ± 0.67	3.00 ± 0.45	1.85	0.06
Green salad	1.65 ± 0.21	1.50 ± 0.11	0.80	0.43
Oriental chicken	2.50 ± 0.48	3.10 ± 0.80	-0.48	0.63
Stir fry veggies [1]	1.85 ± 0.24	2.10 ± 0.24	-0.94	0.35
White rice	2.45 ± 0.56	2.10 ± 0.30	0.60	0.55
Cheesy bread stick	1.85 ± 0.28	2.15 ± 0.28	-1.07	0.29
Brownie with frosting	1.50 ± 0.27	1.35 ± 0.13	0.17	0.86

APPENDIX TABLE 11 (Continued)

DAY THREE **HM** **HC**

Foods	Mean ± SE	Mean ± SE	Wilcoxon	p
Hashbrown quiche	2.20 ±0.59	2.20 ± 0.37	0.06	0.95
Juice [1]	1.70 ± 0.29	1.65 ± 0.26	0.32	0.75
2% milk	1.83 ± 0.35	2.05 ± 0.44	-1.66	0.10
Broccoli soup	3.00 ± 0.55	2.45 ± 0.44	0.86	0.39
Mexican corn muffin or bagel	2.40 ± 0.49	1.33 ± 0.21	1.13	0.26
Slice of cheese	1.90 ± 0.32	2.10 ± 0.28	-0.69	0.49
Calzone with marinara	2.06 ± 0.36	2.00 ± 0.29	0.28	0.78
Oatmeal cookie	1.50 ± 0.27	2.35 ± 0.41	-1.58	0.11
Fresh/canned fruit	1.56 ± 0.24	1.45 ± 0.16	0.11	0.91

APPENDIX TABLE 11 (Continued)

DAY FOUR **HM** **HC**

Foods	Mean ± SE	Mean ± SE	Wilcoxon	p
Muffin				
Banana	1.00 ± 0.00	1.56 ± 0.18	-	-
Applesauce	2.00 ± 0.58	1.67 ± 0.33	-	-
Salt-free turkey	1.61 ± 0.20	2.05 ± 0.24	-1.20	0.23
Sandwich bread	2.00 ± 0.34	1.90 ± 0.30	-0.09	0.93
Lettuce/tomato	1.39 ± 0.20	1.55 ± 0.17	0.41	0.68
Mustard [2]	2.11 ± 0.42	2.05 ± 0.25	0.32	0.75
Fresh/canned fruit	1.30 ± 0.13	1.50 ± 0.18	-1.41	0.16
Saltines	2.80 1.11	3.05 ± 0.74	-0.96	0.34
Meatloaf patty	1.45 ± 0.30	1.80 ± 0.38	-1.60	0.11
Cheesy scalloped potatoes	1.00 ± 0.00	1.45 ± 0.20	-2.06	0.04
Canned/fresh vegetable	2.60 ± 0.88	1.95 ± 0.23	0.30	0.30
Zucchini bread	1.75 ± 0.33	1.60 ± 0.21	0.73	0.73
Vanilla pie/pudding	1.05 ± 0.05	1.90 ± 0.29	0.03	0.03

APPENDIX TABLE 12

Motivation to eat in HM and HC diet phases (mean ±SE) (n=10)

Question 2: How strong is your desire to eat right now? (1 - very strong; 10 = very weak)

	Day 1			Day 2			Day 3			Day 4		
	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner
HM	5.09±2.53	4.84±2.62	4.40±3.46	4.53±2.60	4.72±2.74	5.34±2.27	3.87±2.10	4.45±2.30	4.34±2.45	6.03±2.89	4.95±2.76	3.83±2.71
HC	6.08±1.53	6.93±2.26	6.05±1.86	6.83±1.54	5.64±2.41	5.97±2.15	7.00±0.90	6.84±2.72	5.21±2.51	6.19±3.30	5.61±2.50	5.45±3.64
p value	0.50	0.14	0.58	0.07	0.48	0.58	0.08	0.22	0.69	0.89	0.69	0.35

Question 3: How hunger do you feel? (1 = as hunger as I have ever felt; 10 - not at all hungry)

	Day 1			Day 2			Day 3			Day 4		
	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner
HM	5.27±2.57	4.98±2.70	5.10±3.45	5.11±2.85	5.09±2.58	5.86±2.50	3.89±2.38	4.70±1.96	4.34±2.67	5.94±2.74	4.86±2.80	3.56±2.51
HC	6.61±1.20	7.02±2.04	5.86±1.36	6.77±1.89	6.28±2.35	6.96±2.10	6.95±0.96	7.24±2.24	5.35±2.17	6.65±2.07	5.69±1.91	6.05±3.19
p value	0.50	0.14	0.72	0.39	0.21	0.31	0.14	0.22	0.50	0.50	0.50	0.22

APPENDIX TABLE 12 (Continued)

Question 4: How full do you feel? (1=very full; 10 = not at all full)

	Day 1			Day 2			Day 3			Day 4		
	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner
HM	4.75±2.41	4.53±1.72	5.05±1.96	5.50±2.36	6.13±2.32	6.04±2.25	4.90±2.43	5.78±2.54	5.41±3.05	5.23±2.73	5.26±2.51	5.78±2.98
HC	7.65±1.70	6.37±1.26	7.65±2.24	5.61±2.08	5.99±2.54	5.81±1.93	6.29±2.49	6.83±2.55	7.57±1.79	6.56±2.80	6.71±2.03	7.25±1.94
p value	0.08	0.35	0.14	0.80	0.91	0.50	0.14	0.50	0.14	0.35	0.34	0.25

Question 5: How much food do you think you could eat? (1 = a large amount; 10 = nothing at all)

	Day 1			Day 2			Day 3			Day 4		
	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner
HM	5.85±2.33	5.31±2.09	5.49±2.60	4.53±2.31	4.32±2.43	5.32±2.29	4.61±2.31	5.36±2.23	4.36±2.43	5.62±2.37	4.68±2.14	3.73±2.30
HC	5.35±2.08	5.56±1.21	4.91±1.29	5.78±1.27	5.11±1.53	5.90±1.70	6.01±1.71	6.05±1.29	4.55±2.48	6.41±1.73	5.69±1.44	5.19±2.49
p value	0.89	0.89	0.72	0.24	0.33	0.51	0.69	0.50	0.69	0.50	0.50	0.34

Question 6: Conidering you are on this research diet, how much do you think you could eat right now? (1 = more than what is available for the meal; 10 = very little of what is available for the meal?)

	Day 1			Day 2			Day 3			Day 4		
	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner	Bk	Lunch	Dinner
HM	5.50±0.92	5.34±0.88	4.96±0.17	4.46±1.60	5.31±1.84	4.81±0.93	5.22±1.03	5.04±1.36	5.26±0.81	5.49±1.00	5.45±0.87	5.36±0.91
HC	5.45±1.01	4.77±0.18	5.35±0.94	4.88±0.18	5.18±0.74	4.91±0.54	4.85±0.24	4.83±0.15	4.88±0.15	4.75±0.16	4.79±0.22	4.64±0.19
p value	0.72	0.07	0.72	0.51	0.55	0.83	0.69	0.89	0.59	0.35	0.22	0.07

APPENDIX TABLE 13

Palatability and liking for sensory characteristics of low and high fat foods after HM and HC diets

($\bar{x}$ ±SD; n = 10; Scale: 1 = like extremely; 5 = neither/nor; 9 = dislike extremely. HM=high mono; HC= high CHO diet; WH = whole milk; HH = half and half; WC = whipping cream; WC+ = whipping cream plus oil; VANPUD = vanilla pudding; TSUP=tomato soup; MARGN=margarine; AMRCZ= American cheese; CRMCZ: cream cheese; CTGCZ: cottage cheese; MJCZ: Monterey Jack cheese; LBCKE: pound cake; SODCX: soda crackers; ITDSG: Italian salad dressing; FRDSG: French salad dressing; MAYO: mayonnaise)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
MILK: Skim							
HM	4.40 ± 0.84	4.70 ± 1.49	4.80 ± 0.42	5.10 ± 0.32	5.30 ± 0.67	5.30 ± 0.67	4.56 ± 1.33
HC	4.20 ± 2.53	4.80 ± 0.63	4.00 ± 0.82	5.00 ± 0.00	5.00 ± 0.00	5.00 ± 0.00	3.78 ± 2.11
p	0.80	1.00	0.01*	0.32	0.18	0.18	0.22
MILK: Whole							
HM	3.40 ± 2.17	4.20 ± 2.04	4.10 ± 1.45	5.00 ± 0.00	5.30 ± 0.95	5.30 ± 0.95	3.80 ± 2.10
HC	4.40 ± 2.17	5.00 ± 0.47	4.50 ± 0.85	5.00 ± 0.00	5.40 ± 1.26	5.40 ± 1.26	4.50 ± 2.46
p	0.009*	0.23	0.50	-	0.65	0.65	0.33
MILK: HH							
HM	3.20 ± 1.23	3.90 ± 1.60	3.80 ± 1.48	4.90 ± 0.32	5.20 ± 0.63	5.20 ± 0.63	3.60 ± 1.71
HC	2.70 ± 1.42	4.30 ± 0.95	3.40 ± 1.58	5.00 ± 0.00	5.00 ± 0.00	5.00 ± 0.00	2.70 ± 1.25
p	0.19	0.44	0.44	0.32	0.32	0.32	0.10

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
MILK: WC							
HM	3.60 ± 2.07	4.90 ± 1.52	4.00 ± 1.33	5.00 ± 0.00	5.00 ± 0.00	5.10 ± 0.32	4.10 ± 2.02
HC	3.60 ± 3.10	5.60 ± 2.10	4.00 ± 1.41	5.00 ± 0.00	5.00 ± 0.00	5.00 ± 0.00	4.30 ± 2.54
p	1.00	0.23	1.00	-	-	0.32	0.74
MILK: WC+							
HM	4.30 ± 2.79	5.00 ± 2.36	3.70 ± 1.64	5.00 ± 0.00	5.20 ± 0.63	5.20 ± 0.63	4.20 ± 2.15
HC	3.80 ± 2.94	4.40 ± 2.12	3.50 ± 1.51	5.00 ± 0.00	5.20 ± 0.63	5.00 ± 0.00	3.90 ± 2.47
p	0.68	0.26	0.75	-	-	0.32	0.67
CHOCSKIM:							
HM	4.30 ± 1.57	4.80 ± 1.03	4.50 ± 2.12	5.10 ± 0.32	5.30 ± 0.67	5.10 ± 0.57	3.90 ± 2.38
HC	3.80 ± 1.69	4.80 ± 0.42	3.80 ± 1.48	5.00 ± 0.00	5.00 ± 0.00	5.10 ± 0.32	3.40 ± 1.51
p	0.24	1.00	0.39	0.32	0.18	1.00	0.50
CHOCWH:							
HM	3.40 ± 2.07	4.80 ± 0.78	3.20 ± 1.75	5.00 ± 0.00	5.00 ± 0.00	5.00 ± 0.00	3.00 ± 1.63
HC	3.10 ± 0.99	4.80 ± 0.42	3.70 ± 0.82	5.00 ± 0.00	5.00 ± 0.00	4.90 ± 0.32	3.50 ± 1.18
p	0.78	1.00	0.46	-	-	0.32	0.46
CHOCHALF:							
HM	3.00 ± 1.70	4.90 ± 1.10	3.40 ± 1.90	4.90 ± 0.32	5.10 ± 0.32	5.22 ± 0.67	2.90 ± 2.18
HC	2.90 ± 0.99	4.50 ± 1.27	3.10 ± 1.52	5.00 ± 0.00	5.00 ± 0.00	5.00 ± 0.00	2.80 ± 1.23
p	0.86	0.23	0.88	0.00	0.32	0.32	0.91

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
TSUPWC:							
HM	3.40 ± 1.90	4.67 ± 1.73	4.00 ± 1.25	4.22 ± 1.48	4.80 ± 1.75	5.40 ± 1.26	4.10 ± 2.08
HC	3.40 ± 2.41	4.78 ± 1.20	4.50 ± 1.84	4.22 ± 1.39	4.90 ± 1.91	5.40 ± 1.26	3.60 ± 2.32
p	1.00	0.71	0.38	1.00	0.56	-	0.24
TSUPWC+:							
HM	3.10 ± 2.42	4.60 ± 1.90	4.30 ± 1.06	4.60 ± 0.84	4.90 ± 0.88	5.40 ± 0.97	3.90 ± 2.13
HC	3.50 ± 1.78	4.60 ± 1.43	5.00 ± 0.94	4.40 ± 1.08	4.60 ± 1.51	5.10 ± 0.31	3.70 ± 1.70
p	0.59	0.92	0.11	0.48	0.32	0.18	0.79
MARGN LO:							
HM	3.50 ± 2.22	4.60 ± 2.76	4.20 ± 1.62	3.80 ± 1.81	5.10 ± 0.32	5.20 ± 0.63	4.70 ± 2.11
HC	3.00 ± 1.25	4.60 ± 2.07	4.60 ± 1.08	4.00 ± 1.63	5.00 ± 0.00	5.00 ± 0.00	3.80 ± 1.69
p	0.55	0.89	0.46	0.89	0.32	0.32	0.34
MARGN HI:							
HM	3.40 ± 2.07	5.50 ± 2.76	3.80 ± 1.48	3.70 ± 1.64	5.10 ± 0.32	5.00 ± 0.00	5.30 ± 2.21
HC	3.70 ± 2.45	6.10 ± 2.13	4.80 ± 0.63	4.30 ± 1.15	4.90 ± 0.32	5.30 ± 0.95	5.90 ± 2.69
p	0.61	0.78	0.04*	0.35	0.16	0.32	0.24
AMRCZ LO:							
HM	3.10 ± 1.52	4.80 ± 1.99	4.30 ± 1.57	4.70 ± 2.11	5.00 ± 0.47	5.40 ± 0.97	4.30 ± 2.71
HC	4.90 ± 2.33	5.00 ± 0.17	4.90 ± 1.73	4.70 ± 1.77	5.40 ± 1.08	5.50 ± 0.97	5.50 ± 2.32
p	0.09	0.76	0.22	1.00	0.34	0.85	0.21

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
AMRCZ HI:							
HM	4.00 ± 2.79	5.44 ± 2.65	3.80 ± 1.87	4.30 ± 2.45	4.60 ± 1.35	5.00 ± 1.56	4.60 ± 3.17
HC	3.70 ± 1.83	5.11 ± 1.61	5.00 ± 0.94	4.70 ± 1.59	4.50 ± 1.18	5.10 ± 1.29	4.60 ± 1.84
p	0.72	0.59	0.08	0.57	0.68	0.57	1.00
CRMCZ LO:							
HM	2.30 ± 1.42	3.70 ± 1.83	3.60 ± 1.71	3.10 ± 1.79	3.40 ± 1.84	5.10 ± 0.32	2.40 ± 1.17
HC	2.00 ± 1.15	3.50 ± 1.84	4.60 ± 1.17	2.90 ± 1.20	3.90 ± 1.97	4.60 ± 1.26	2.70 ± 1.83
p	0.47	0.67	0.07	0.76	0.34	0.18	0.63
CRMCZ HI:							
HM	1.70 ± 0.67	3.50 ± 2.17	3.50 ± 1.78	2.90 ± 1.66	3.30 ± 1.70	4.70 ± 1.34	2.00 ± 0.94
HC	2.00 ± 0.82	3.70 ± 1.49	4.10 ± 1.53	2.60 ± 1.17	3.20 ± 1.62	4.60 ± 1.26	2.10 ± 0.57
p	0.28	0.72	0.11	0.47	0.56	0.32	0.59
CTGCZ: LO							
HM	3.90 ± 2.42	4.80 ± 1.75	4.40 ± 1.08	4.60 ± 1.17	4.20 ± 0.92	5.10 ± 0.32	3.80 ± 1.99
HC	3.10 ± 2.33	5.00 ± 1.89	4.70 ± 1.06	3.90 ± 1.45	4.80 ± 0.92	5.00 ± 0.00	3.90 ± 2.13
p	0.17	0.76	0.45	0.04*	0.14	0.32	0.86
CTGCZ: HI							
HM	2.80 ± 1.62	4.40 ± 2.07	3.80 ± 1.62	3.50 ± 1.43	3.70 ± 1.42	5.10 ± 0.32	2.80 ± 1.62
HC	2.60 ± 0.84	4.80 ± 0.92	4.60 ± 0.70	3.50 ± 0.97	4.10 ± 1.10	5.00 ± 0.00	2.70 ± 0.95
p	0.73	0.56	0.15	1.00	0.37	0.32	0.89

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
MJKCZ LO:							
HM	3.60 ± 2.37	4.60 ± 1.96	4.30 ± 1.25	3.80 ± 1.87	4.20 ± 1.69	4.80 ± 1.69	3.70 ± 2.11
HC	3.10 ± 1.85	4.30 ± 2.11	4.00 ± 1.41	3.40 ± 1.26	4.00 ± 1.70	4.90 ± 1.60	4.00 ± 2.36
p	0.36	0.55	0.54	0.40	0.44	0.56	0.67
MJKCZ HI:							
HM	3.40 ± 2.50	4.50 ± 1.84	4.00 ± 1.89	4.00 ± 2.00	3.70 ± 1.83	4.60 ± 1.65	3.30 ± 2.21
HC	2.10 ± 0.88	4.30 ± 1.83	4.10 ± 1.73	2.90 ± 1.20	4.20 ± 1.75	4.90 ± 1.45	2.80 ± 1.39
p	0.14	0.86	0.86	0.09	0.36	0.41	0.49
LBCKE LO:							
HM	4.40 ± 1.17	4.40 ± 1.65	3.10 ± 1.85	4.40 ± 1.35	5.00 ± 0.00	5.10 ± 0.32	3.40 ± 1.71
HC	3.30 ± 2.11	4.10 ± 1.79	2.70 ± 1.83	3.90 ± 1.66	4.60 ± 1.26	4.60 ± 1.26	2.90 ± 2.08
p	0.14	0.60	0.50	0.18	0.32	0.18	0.42
LBCKE HI:							
HM	4.00 ± 1.41	4.60 ± 1.35	2.90 ± 1.20	4.40 ± 1.08	5.00 ± 0.00	5.00 ± 0.00	3.00 ± 1.05
HC	3.10 ± 1.66	3.20 ± 1.48	2.20 ± 0.63	3.90 ± 1.60	4.60 ± 1.26	4.60 ± 1.26	2.10 ± 0.99
p	0.23	0.03*	0.07	0.41	0.32	0.32	0.04*
SODCX LO:							
HM	4.10 ± 1.52	4.30 ± 1.16	4.22 ± 1.56	3.76 ± 2.17	5.00 ± 0.00	5.00 ± 0.00	3.60 ± 2.37
HC	3.90 ± 1.52	4.70 ± 1.34	4.00 ± 1.73	3.89 ± 2.26	5.00 ± 0.00	5.00 ± 0.00	3.20 ± 2.30
p	0.72	0.41	0.56	0.67	-	-	0.80

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
SODCX HI:							
HM	3.80 ± 1.62	3.90 ± 1.60	4.70 ± 0.95	2.60 ± 0.70	5.00 ± 0.00	5.00 ± 0.00	2.40 ± 0.70
HC	4.10 ± 1.52	4.40 ± 1.08	4.70 ± 0.95	4.40 ± 2.50	5.00 ± 0.00	5.00 ± 0.00	3.00 ± 2.05
p	0.58	0.10	-	0.05*	-	-	0.38
ITDSG LO:							
HM	3.40 ± 1.96	3.10 ± 1.85	3.50 ± 1.84	3.67 ± 2.45	3.60 ± 2.17	4.70 ± 1.70	2.90 ± 2.13
HC	3.30 ± 1.77	3.70 ± 1.94	3.80 ± 1.69	3.11 ± 1.36	3.70 ± 2.36	4.90 ± 1.66	3.20 ± 2.25
p	0.86	0.24	0.18	0.38	0.86	0.58	0.89
ITDSG HI:							
HM	4.90 ± 2.13	4.50 ± 1.43	3.70 ± 1.83	3.30 ± 2.06	4.00 ± 2.26	5.50 ± 1.72	2.20 ± 1.03
HC	3.60 ± 2.12	4.10 ± 2.69	3.80 ± 1.62	2.80 ± 1.62	3.10 ± 1.60	4.60 ± 1.26	3.50 ± 2.27
p	0.02*	0.57	0.86	0.39	0.05*	0.17	0.12
FRDSG LO:							
HM	2.80 ± 1.55	4.00 ± 2.00	3.90 ± 2.13	3.80 ± 1.62	3.80 ± 1.55	5.10 ± 0.74	3.60 ± 2.10
HC	2.80 ± 2.15	3.90 ± 1.60	3.90 ± 2.46	3.10 ± 1.85	3.10 ± 1.45	4.60 ± 1.35	3.30 ± 2.11
p	1.00	0.65	0.52	0.04*	0.04*	0.26	0.52
FRDSG HI:							
HM	2.60 ± 1.90	3.90 ± 2.13	3.70 ± 2.16	4.00 ± 2.11	3.60 ± 2.17	4.70 ± 1.42	4.00 ± 2.45
HC	2.70 ± 1.16	4.40 ± 2.22	3.70 ± 2.16	2.90 ± 1.37	3.70 ± 2.36	5.10 ± 1.79	3.60 ± 2.46
p	0.52	0.92	1.00	0.04*	0.56	0.10	0.55

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
MAYO LO:							
HM	2.50 ± 1.27	5.30 ± 2.21	3.89 ± 1.05	4.30 ± 1.95	5.00 ± 1.49	5.20 ± 0.79	4.20 ± 2.41
HC	2.50 ± 1.43	4.70 ± 2.41	3.89 ± 1.45	3.80 ± 1.40	4.20 ± 1.87	5.50 ± 0.85	3.80 ± 2.35
p	0.76	0.31	1.00	0.58	0.40	0.26	0.36
MAYO HI:							
HM	2.10 ± 1.20	3.90 ± 2.02	3.50 ± 1.35	3.60 ± 2.07	4.30 ± 1.42	5.10 ± 0.32	2.70 ± 1.06
HC	2.40 ± 1.08	3.30 ± 1.57	3.60 ± 1.78	3.40 ± 1.90	4.20 ± 2.49	4.70 ± 1.34	3.70 ± 2.54
p	0.41	0.75	0.86	0.67	0.86	0.41	0.19
JAM LO:							
HM	2.70 ± 1.70	3.90 ± 1.79	2.20 ± 1.32	3.30 ± 2.00	4.30 ± 1.49	4.56 ± 1.33	2.10 ± 1.60
HC	3.90 ± 2.47	4.40 ± 1.26	3.00 ± 1.41	4.30 ± 1.25	4.80 ± 1.13	5.00 ± 0.00	2.70 ± 2.00
p	0.09	0.28	0.20	0.10	0.41	0.32	0.10
JAM HI:							
HM	2.70 ± 1.49	3.80 ± 1.62	2.80 ± 2.39	3.76 ± 1.64	4.70 ± 0.67	5.00 ± 0.00	3.10 ± 2.23
HC	2.70 ± 1.34	4.50 ± 1.08	2.70 ± 1.42	3.76 ± 1.30	4.10 ± 1.45	5.10 ± 0.32	2.20 ± 0.79
p	1.00	0.20	0.95	1.00	0.11	0.32	0.48
V8 LOSALT:							
HM	3.70 ± 2.11	4.60 ± 1.65	4.80 ± 0.92	3.40 ± 1.17	4.90 ± 1.97	4.90 ± 1.53	4.30 ± 2.16
HC	4.20 ± 1.62	5.10 ± 1.10	4.90 ± 1.10	5.00 ± 1.63	5.20 ± 1.22	5.00 ± 0.82	4.90 ± 2.18
p	0.46	0.32	0.83	0.02*	0.47	0.79	0.19

APPENDIX TABLE 13 (Continued)

	Creaminess	Oiliness	Sweetness	Saltiness	Sourness	Bitterness	Pleasantness
V8HISALT:							
HM	3.70 ± 2.00	4.60 ± 1.78	4.33 ± 1.50	3.20 ± 1.55	3.80 ± 1.69	4.70 ± 1.34	3.20 ± 2.15
HC	4.70 ± 1.64	4.90 ± 1.29	4.11 ± 1.69	4.00 ± 1.76	4.40 ± 1.35	5.10 ± 0.32	3.70 ± 2.06
p	0.10	0.67	0.56	0.17	0.28	0.32	0.28

APPENDIX TABLE 14

Palatability and liking for sensory characteristics of low and high fat foods in diabetic and nondiabetic women
(n =20; 10 diabetic, 10 nondiabetic)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
MILK								
Creaminess	4.10 ± 1.71	4.23 ± 1.44	-0.18	0.86	3.83 ± 2.35	4.75 ± 1.91	-0.97	0.35
Oiliness	4.68 ± 0.82	5.03 ± 0.66	-1.05	0.31	4.98 ± 1.48	6.27 ± 1.57	-1.91	0.07
Sweetness	4.35 ± 0.60	4.38 ± 1.22	43.0	0.63	3.80 ± 1.16	4.53 ± 1.18	-1.39	0.18
Saltiness	5.03 ± 0.08	4.85 ± 0.43	37.0	0.35	4.97 ± 0.08	4.90 ± 0.32	49.5	0.97
Sourness	5.25 ± 0.47	5.10 ± 0.29	40.5	0.53	5.18 ± 0.55	5.13 ± 0.32	46.0	0.80
Bitterness	5.23 ± 0.49	5.15 ± 0.34	0.40	0.70	5.08 ± 0.17	5.05 ± 0.11	49.0	0.97
Pleasantness	4.20 ± 1.64	3.83 ± 1.25	42.0	0.58	4.13 ± 1.95	5.55 ± 1.84	-1.69	0.11
CHOCOLATE MILK								
Creaminess	3.65 ± 1.33	4.05 ± 1.26	-0.69	0.50	1.65 ± 0.68	2.68 ± 1.45	-2.02	0.07
Oiliness	4.80 ± 0.41	4.55 ± 1.11	44.0	0.68	4.13 ± 0.97	4.75 ± 1.85	-0.94	0.36
Sweetness	3.80 ± 1.04	3.73 ± 1.36	0.14	0.89	2.23 ± 0.83	2.88 ± 0.98	-1.60	0.13
Saltiness	5.03 ± 0.08	4.60 ± 0.96	36.5	0.35	4.98 ± 0.08	4.55 ± 0.76	38.5	0.44
Sourness	5.08 ± 0.17	5.05 ± 0.11	49.0	0.97	5.00 ± 0.00	5.00 ± 0.00	50.0	0.97

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
Bitterness	5.03 ± 0.25	4.90 ± 0.74	46.0	0.80	5.00 ± 0.00	4.83 ± 0.67	45.0	0.74
Pleasantness	3.45 ± 1.28	4.13 ± 1.37	-1.14	0.27	1.85 ± 0.74	2.85 ± 1.42	-1.98	0.06
VANILLA PUDDNG								
Creaminess	2.25 ± 0.99	2.93 ± 1.35	-1.27	0.22	2.78 ± 1.41	4.65 ± 2.34	-2.17	0.04*
Oiliness	4.08 ± 1.15	4.68 ± 1.65	-0.94	0.36	4.80 ± 1.60	5.83 ± 1.78	-1.35	0.19
Sweetness	2.95 ± 0.75	3.10 ± 1.59	-0.27	0.79	3.08 ± 1.16	3.70 ± 1.86	-0.90	0.38
Saltiness	4.98 ± 0.08	4.65 ± 0.50	32.5	0.22	5.00 ± 0.00	4.90 ± 0.50	45.0	0.74
Sourness	5.03 ± 0.08	4.90 ± 0.24	1.55	0.14	5.00 ± 0.00	4.95 ± 0.37	50.0	0.97
Bitterness	5.00 ± 0.00	5.03 ± 0.18	50.0	0.97	5.00 ± 0.00	5.05 ± 0.20	45.0	0.74
Pleasantness	2.73 ± 0.95	3.20 ± 1.80	46.0	0.80	3.13 ± 1.27	4.60 ± 2.08	-1.91	0.07
TOMATO SOUP								
Creaminess	4.88 ± 1.78	4.98 ± 1.86	-0.12	0.90	3.35 ± 1.66	3.53 ± 1.61	-0.24	0.81
Oiliness	5.33 ± 1.33	5.65 ± 1.79	-0.46	0.65	4.87 ± 1.56	5.43 ± 1.57	-0.79	0.44
Sweetness	4.83 ± 0.73	4.73 ± 1.66	0.17	0.86	4.45 ± 0.72	3.98 ± 1.22	1.06	0.30
Saltiness	5.00 ± 1.34	4.63 ± 1.66	0.56	0.59	4.45 ± 1.04	3.70 ± 1.34	1.40	0.18
Sourness	5.15 ± 1.11	4.83 ± 1.09	0.66	0.52	4.80 ± 1.40	4.35 ± 1.10	0.80	0.43
Bitterness	5.38 ± 0.78	5.20 ± 0.78	44.0	0.68	5.33 ± 0.94	4.83 ± 0.92	50.0	0.97

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristics	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
Pleasantness	5.00 ± 1.93	5.03 ± 1.97	-0.03	0.98	3.85 ± 2.11	4.40 ± 1.91	-0.61	0.55
MARGARINE								
Creaminess	2.95 ± 1.74	3.95 ± 1.91	-1.23	0.24	4.05 ± 2.63	3.85 ± 2.14	0.19	0.85
Oiliness	4.05 ± 1.94	5.20 ± 1.87	-1.35	0.19	4.95 ± 1.89	5.95 ± 1.99	-1.15	0.26
Sweetness	3.50 ± 1.84	4.60 ± 0.91	-1.70	0.11	3.90 ± 1.31	4.35 ± 0.91	-0.89	0.38
Saltiness	4.35 ± 1.51	4.05 ± 1.57	0.44	0.67	4.55 ± 1.14	4.00 ± 1.20	1.05	0.31
Sourness	4.35 ± 1.51	4.05 ± 1.51	0.44	0.67	5.00 ± 0.00	5.00 ± 0.24	50.0	0.97
Bitterness	4.95 ± 0.50	5.10 ± 0.32	41.0	0.53	5.15 ± 0.47	5.15 ± 0.47	50.0	0.97
Pleasantness	4.10 ± 2.26	4.65 ± 1.08	-0.70	0.50	4.25 ± 1.85	5.85 ± 2.14	-1.79	0.09
AMERICAN CHEESE								
Creaminess	3.45 ± 1.32	4.70 ± 1.09	-2.31	0.03*	3.05 ± 1.52	4.85 ± 1.68	-2.51	0.02*
Oiliness	4.60 ± 1.08	5.30 ± 1.38	-1.27	0.22	4.40 ± 1.27	5.45 ± 2.11	-1.35	0.19
Sweetness	4.40 ± 0.97	4.90 ± 1.29	-0.98	0.34	4.20 ± 1.27	4.85 ± 0.63	-1.45	0.17
Saltiness	4.80 ± 1.38	4.95 ± 1.48	-0.23	0.82	4.55 ± 1.14	5.15 ± 0.78	33.5	0.22
Sourness	5.10 ± 0.32	5.25 ± 0.54	40.5	0.53	4.40 ± 1.27	4.85 ± 1.70	-0.67	0.51
Bitterness	5.05 ± 0.16	5.60 ± 0.70	23.5	0.04*	5.25 ± 0.54	5.15 ± 1.44	41.0	0.53
Pleasantness	3.95 ± 1.36	5.35 ± 1.81	-1.95	0.06	3.60 ± 1.56	5.25 ± 1.80	-2.19	0.04*

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
CREAM CHEESE								
Creaminess	2.80 ± 1.95	2.85 ± 1.68	-0.06	0.95	2.95 ± 2.28	2.35 ± 1.31	43.5	0.63
Oiliness	4.20 ± 1.40	4.10 ± 1.81	0.14	0.89	4.05 ± 1.72	4.05 ± 1.79	0.00	1.00
Sweetness	4.30 ± 0.92	4.40 ± 1.15	-0.22	0.83	4.50 ± 1.39	4.20 ± 1.53	46.5	0.85
Saltiness	4.10 ± 1.31	3.15 ± 1.18	1.72	0.11	4.50 ± 1.45	3.05 ± 1.42	2.23	0.04*
Sourness	4.35 ± 1.29	3.60 ± 1.71	1.11	0.28	5.00 ± 1.65	3.05 ± 1.50	2.77	0.01*
Bitterness	5.15 ± 0.34	4.90 ± 0.70	45.0	0.74	5.40 ± 1.10	4.70 ± 1.32	45.0	0.74
Pleasantness	2.90 ± 1.43	3.10 ± 1.56	-0.30	0.77	3.30 ± 1.74	2.55 ± 1.28	35.5	0.28
COTTAGE CHEESE								
Creaminess	3.45 ± 2.01	3.70 ± 2.04	-0.28	0.79	3.45 ± 2.03	2.80 ± 0.89	0.93	0.37
Oiliness	4.40 ± 1.51	4.85 ± 1.53	-0.66	0.51	4.50 ± 1.53	4.55 ± 1.40	-0.08	0.94
Sweetness	4.05 ± 1.46	4.60 ± 0.81	-1.04	0.31	4.45 ± 1.38	4.20 ± 0.95	0.47	0.64
Saltiness	4.15 ± 1.49	4.25 ± 1.16	-0.17	0.87	4.60 ± 1.24	3.40 ± 1.13	2.26	0.04*
Sourness	4.70 ± 1.34	4.50 ± 0.71	0.47	0.64	4.75 ± 1.40	3.90 ± 1.08	1.52	0.15
Bitterness	5.20 ± 0.35	5.05 ± 0.16	39.5	0.44	5.30 ± 0.63	5.05 ± 0.16	39.5	0.44
Pleasantness	3.50 ± 1.68	4.10 ± 1.56	-0.83	0.42	3.70 ± 1.92	2.90 ± 0.97	1.18	0.25

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
MONTEREY JACK CHEESE								
Creaminess	3.55 ± 1.74	3.45 ± 1.69	0.13	0.90	3.20 ± 1.59	3.05 ± 1.34	0.23	0.82
Oiliness	4.60 ± 1.37	4.35 ± 1.92	0.34	0.74	4.00 ± 1.41	4.70 ± 1.55	-1.06	0.31
Sweetness	4.75 ± 1.14	4.25 ± 1.14	0.98	0.34	4.45 ± 0.76	4.05 ± 1.55	0.73	0.47
Saltiness	4.35 ± 1.18	3.40 ± 1.35	1.68	0.11	4.35 ± 0.94	3.45 ± 1.38	1.70	0.11
Sourness	4.90 ± 0.97	4.05 ± 1.62	1.42	0.17	4.70 ± 1.27	3.95 ± 1.61	1.16	0.26
Bitterness	5.35 ± 0.78	4.50 ± 1.72	1.43	0.17	5.15 ± 0.67	4.55 ± 1.40	35.0	0.28
Pleasantness	3.90 ± 2.16	3.90 ± 1.76	0.00	1.00	3.55 ± 1.86	3.70 ± 1.77	-0.19	0.86
POUND CAKE								
Creaminess	3.30 ± 1.27	3.90 ± 1.37	-1.01	0.32	3.25 ± 1.34	3.65 ± 1.06	-0.74	0.47
Oiliness	4.35 ± 0.78	4.30 ± 1.49	0.09	0.93	4.25 ± 1.03	4.05 ± 1.19	0.40	0.69
Sweetness	3.00 ± 0.88	2.70 ± 1.65	33.0	0.22	2.95 ± 0.96	2.90 ± 1.39	0.09	0.93
Saltiness	4.60 ± 0.84	4.30 ± 1.32	41.0	0.53	4.40 ± 0.88	4.30 ± 1.06	0.23	0.82
Sourness	5.00 ± 0.00	4.80 ± 0.62	45.0	0.74	5.00 ± 0.00	4.80 ± 0.63	45.0	0.74
Bitterness	5.00 ± 0.00	4.85 ± 0.67	50.0	0.97	5.00 ± 0.00	4.85 ± 0.67	50.0	0.97
Pleasantness	2.75 ± 0.92	3.15 ± 1.65	45.0	0.74	2.75 ± 1.16	3.00 ± 1.23	-0.47	0.65

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
SALTINES								
Creaminess	3.60 ± 1.71	4.55 ± 0.76	-1.60	0.13	3.35 ± 1.63	4.45 ± 0.96	-1.84	0.08
Oiliness	4.35 ± 1.38	4.80 ± 0.42	48.0	0.91	3.90 ± 1.39	4.45 ± 1.07	-0.99	0.33
Sweetness	4.30 ± 1.09	4.35 ± 1.31	-0.09	0.93	4.25 ± 1.23	4.80 ± 0.63	-1.26	0.22
Saltiness	3.50 ± 0.88	4.00 ± 1.56	-0.88	0.39	3.50 ± 1.13	3.65 ± 1.33	-0.27	0.79
Sourness	4.85 ± 0.47	5.00 ± 0.00	45.0	0.74	5.00 ± 0.00	5.00 ± 0.00	0.50	0.97
Bitterness	5.00 ± 0.00	5.00 ± 0.00	50.0	0.98	5.00 ± 0.00	5.00 ± 0.00	50.0	0.97
Pleasantness	2.70 ± 1.21	3.80 ± .72	-1.66	0.12	2.70 ± 1.48	3.10 ± 0.94	35.5	0.28
ITALIAN DRESSNG								
Creaminess	3.55 ± 1.12	3.80 ± 1.48	-0.43	0.67	4.70 ± 2.15	3.95 ± 1.61	0.88	0.39
Oiliness	4.05 ± 1.34	3.65 ± 1.62	0.60	0.56	5.15 ± 2.10	4.35 ± 2.14	0.85	0.41
Sweetness	4.20 ± 1.81	3.85 ± 1.56	0.46	0.65	5.15 ± 1.49	3.80 ± 1.57	1.97	0.06
Saltiness	4.45 ± 1.85	3.15 ± 1.47	1.74	0.10	4.70 ± 1.49	2.90 ± 1.39	2.79	0.01*
Sourness	5.20 ± 1.65	3.30 ± 1.89	2.39	0.03*	5.25 ± 1.65	2.80 ± 1.59	3.38	0.003*
Bitterness	5.15 ± 0.47	4.65 ± 1.56	37.0	0.35	5.55 ± 0.83	4.75 ± 0.98	1.97	0.07
Pleasantness	3.60 ± 1.35	3.35 ± 1.86	0.34	0.74	4.95 ± 2.14	3.85 ± 2.21	1.13	0.27

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
FRENCH DRESSING								
Creaminess	3.00 ± 1.33	2.75 ± 1.36	0.42	0.68	3.80 ± 1.72	2.85 ± 1.16	1.45	0.16
Oiliness	4.60 ± 0.74	3.80 ± 1.67	1.39	0.18	4.50 ± 2.10	4.15 ± 1.65	0.42	0.68
Sweetness	4.10 ± 1.49	3.55 ± 2.09	0.68	0.51	4.50 ± 1.51	3.69 ± 1.96	1.15	0.26
Saltiness	4.85 ± 1.08	3.10 ± 1.24	3.36	0.003*	4.55 ± 1.61	3.50 ± 1.33	1.59	0.13
Sourness	4.70 ± 0.59	3.25 ± 1.28	3.27	0.004*	5.05 ± 1.94	3.35 ± 2.07	1.90	0.07
Bitterness	5.00 ± 0.00	4.80 ± 0.86	35.0	0.28	5.40 ± 1.29	4.70 ± 1.59	1.08	0.30
Pleasantness	3.90 ± 1.70	3.30 ± 1.80	0.77	0.45	4.95 ± 1.72	3.65 ± 2.22	1.46	0.16
MAYONNAISE								
Creaminess	2.70 ± 1.80	3.60 ± 1.85	-1.10	0.28	2.55 ± 1.38	3.05 ± 1.46	-0.79	0.44
Oiliness	4.00 ± 1.39	4.45 ± 1.30	-0.75	0.47	4.15 ± 1.31	4.40 ± 0.99	-0.48	0.64
Sweetness	3.45 ± 1.36	2.50 ± 0.82	1.89	0.08	3.50 ± 1.78	2.55 ± 0.96	1.49	0.15
Saltiness	4.55 ± 0.86	4.25 ± 1.38	44.5	0.74	4.50 ± 0.94	4.10 ± 1.29	0.79	0.44
Sourness	5.05 ± 0.16	4.40 ± 1.08	32.0	0.19	5.00 ± 0.24	4.25 ± 1.01	2.29	0.03*
Bitterness	5.05 ± 0.16	4.80 ± 0.63	40.5	0.53	5.00 ± 0.00	5.10 ± 0.21	-1.50	0.15
Pleasantness	2.85 ± 1.80	2.65 ± 1.69	0.26	0.80	2.80 ± 1.32	2.60 ± 0.84	0.41	0.69

APPENDIX TABLE 14 (Continued)

	Low fat sample				High fat sample			
Food/sensory characteristic	Diabetic	Nondiabetic	t or U	p value	Diabetic	Nondiabetic	t or U	p value
STRAWBERRY JAM								
Creaminess	2.70 ± 1.80	3.60 ± 1.85	36.5	0.35	2.55 ± 1.38	3.05 ± 1.46	-0.79	0.44
Oiliness	4.00 ± 1.39	4.45 ± 1.30	-0.75	0.47	4.15 ± 1.31	4.40 ± 0.99	-0.48	0.64
Sweetness	3.45 ± 1.36	2.50 ± 0.82	1.89	0.08	3.50 ± 1.78	2.55 ± 0.96	1.49	0.15
Saltiness	4.55 ± 0.86	4.25 ± 1.38	44.5	0.74	4.50 ± 0.94	4.10 ± 1.29	0.79	0.44
Sourness	5.05 ± 0.16	4.40 ± 1.08	32.0	0.19	5.00 ± 0.24	4.25 ± 1.01	2.29	0.03*
Bitterness	5.05 ± 0.16	4.80 ± 0.63	40.5	0.53	5.00 ± 0.00	5.10 ± 0.21	-1.50	0.15
Pleasantness	2.85 ± 1.80	2.65 ± 1.68	0.26	0.80	2.80 ± 1.32	2.90 ± 1.24	-0.18	0.86
V8 TOMATO JUICE								
Creaminess	5.75 ± 2.13	4.50 ± 1.63	1.48	0.16	4.25 ± 1.42	4.40 ± 1.41	-0.24	0.82
Oiliness	5.95 ± 1.59	5.15 ± 1.11	1.31	0.21	4.85 ± 1.27	4.80 ± 1.23	0.09	0.93
Sweetness	5.55 ± 1.69	5.15 ± 0.71	0.69	0.50	4.45 ± 1.32	3.65 ± 1.83	1.12	0.28
Saltiness	5.45 ± 1.62	4.65 ± 1.33	1.20	0.24	4.35 ± 1.53	3.35 ± 1.51	1.46	0.16
Sourness	6.30 ± 1.78	5.05 ± 1.52	1.69	0.11	4.80 ± 1.34	3.95 ± 1.28	1.45	0.16
Bitterness	6.55 ± 1.38	5.20 ± 1.34	24.0	0.05*	5.30 ± 0.72	4.90 ± 0.74	40.5	0.53
Pleasantness	5.70 ± 1.96	5.30 ± 1.72	0.49	0.63	3.80 ± 1.70	3.60 ± 0.25	0.25	0.81

APPENDIX TABLE 15

Composition of foods used in taste testing

I. Four foods constructed with varying levels of fat

	Milk	Chocolate milk	Vanilla pudding	Tomato Soup	Range
Skim milk	0.18%	0.50%	0.26%	0.86%	0.18 - 0.86%
Whole milk	3.34%	3.30%	2.86%	2.47%	2.47 - 3.34%
Half and half	11.49%	10.57%	9.58%	6.61%	6.61 - 11.49%
Whipping cream	37.05%	33.33%	30.65%	19.60%	19.60 - 37.05%
Whipping cream/ oil	49.80%	45.63%	42.66%	28.80%	28.80 - 49.80%

II. Low and high fat foods commercially available (average serving 1 T., 14 g)

Food	Low fat (gms)	Low fat (%)	High fat (gms)	High fat (%)
Margarine	4.5	7%	11	17%
Cream cheese	6	9%	10	15%
Monterey Jack cheese	6	9%	9	14%
American cheese	0	0%	7	10%
Cottage Cheese	2.5	2%	5	4%
Pound cake	0	0%	16	25%
Soda crackers	0	0%	1.88 g	14%

APPENDIX TABLE 15 (Continued)

II. Low and high fat foods commercially available (continued)

Food	Low fat (gms)	Low fat (%)	High fat (gms)	High fat (%)
Italian dressing	0	0%	11	16%
French dressing	0	0%	12	18%
Mayonnaise	5	8%	11	17%
Mean ± SD	2.4 ± 2.71	3.5 ± 4.17	9.39 ± 3.95	15.0 ± 5.44

III. Low and high sugar foods commercially available

	Low sugar (gms)	Low sugar (%)	High sugar (gms)	High sugar (%)
Strawberry jam	5	2%	14	5%

IV. Low and high sodium foods commercially available

	Low sodium (mgs)	Low sodium (%)	High sodium (mgs)	High sodium (%)
V8 Vegetable juice	95		430	

APPENDIX TABLE 16

Intensity of hunger and fullness: HM and HC diet phases

(Hunger, fullness: +5=complete; 4=very great; 3=great; 2=some; 1=very little)
(n=10; frequency/percent)

	High monounsaturated fat diet										High carbohydrate diet									
	Hunger					Fullness					Hunger					Fullness				
	+5	+4	+3	+2	+1	-5	-4	-3	-2	-1	+5	+4	+3	+2	+1	-5	-4	-3	-2	-1
S010	0 0%	0 0%	0 0%	0 0%	13 100	0 0%	1 14%	5 71%	1 14%	0 0%	0 0%	0 0%	0 0%	0 0%	16 100	0 0%	0 0%	18 100	0 0%	0 0%
S226	0 0%	0 0%	0 0%	2 50%	2 50%	2 6%	3 9%	8 25%	11 34%	8 25%	0 0%	0 0%	1 100%	0 0%	0 0%	1 4%	7 29%	7 29%	6 25%	3 13%
S201	0 0%	6 30%	7 35%	7 35%	0 0%	0 0%	10 48%	5 24%	3 14%	3 14%	6 26%	3 13%	1 4%	12 52%	1 4%	0 0%	2 9%	13 59%	5 23%	2 9%
S151	0 0%	0 0%	3 23%	9 69%	1 8%	0 0%	0 0%	2 18%	7 64%	2 18%	0 0%	0 0%	10 26%	19 50%	9 24%	0 0%	0 0%	1 10%	4 40%	5 50%
S259	0 0%	0 0%	0 0%	0 0%	3 100	4 57%	0 0%	0 0%	2 29%	1 14%	0 0%	1 11%	0 0%	3 33%	5 56%	9 53%	1 6%	0 0%	5 29%	2 12%
S356	0 0%	0 0%	0 0%	2 18%	9 82%	0 0%	0 0%	3 45%	2 29%	2 29%	0 0%	0 0%	0 0%	10 53%	9 47%	0 0%	0 0%	0 0%	2 25%	6 75%
S209	1 8%	0 0%	2 17%	0 0%	9 75%	3 18%	1 6%	1 6%	4 24%	8 47%	0 0%	1 9%	3 27%	5 46%	2 18%	1 10%	2 20%	4 40%	0 0%	3 30%
S413	0 0%	0 0%	0 0%	2 20%	8 80%	0 0%	0 0%	0 0%	0 0%	0 0%	2 7%	5 17%	5 17%	5 21%	11 38%	0 0%	0 0%	0 0%	3 75%	1 25%

APPENDIX TABLE 16 (Continued)

	High monounsaturated fat diet										High carbohydrate diet									
	Hunger					Fullness					Hunger					Fullness				
	+5	+4	+3	+2	+1	-5	-4	-3	-2	-1	+5	+4	+3	+2	+1	-5	-4	-3	-2	-1
S324	0 0%	0 0%	0 0%	6 19%	25 81%	1 2%	15 28%	6 11%	14 26%	17 32%	0 0%	0 0%	0 0%	0 0%	2 100	30 48%	16 25%	6 10%	8 13%	3 5%
S013	0 0%	0 0%	5 14%	25 68%	7 19%	0 0%	0 0%	0 0%	3 100	0 0%	0 0%	0 0%	1 4%	8 30%	18 67%	0 0%	0 0%	1 9%	3 27%	7 64%
SUM	1	6	17	53	77	10	30	30	47	41	8	10	21	63	73	47	28	50	36	29
Mean	0.10	0.60	1.7	5.3	7.7	1.0	3.0	3.0	4.7	4.1	0.8	1.0	2.1	6.3	7.3	4.7	2.8	5.0	3.6	2.9
Mag	5	24	51	106	77	50	120	90	94	41	40	40	63	126	73	235	112	147	66	29
Mean	0.5	2.4	5.1	1.1	7.7	5.0	1.2	9.0	9.4	4.1	4.0	4.0	6.3	1.3	7.3	2.4	1.1	1.5	6.6	2.9

APPENDIX TABLE 17

Subjects with abnormal NCSE and/or Trails scores

(NCSE rating scale: 1 = average range; 2 = mild impairment; 3 = moderate impairment; 4 = severe impairment. Trails rating (A or B form): 1 = perferctly normal; 2 = normal; 3 = mild/moderate impairment; 4 = moderate/severe impairment)

Subject	NCSE: construction (n=2)	NCSE: memory (n=3)	NCSE: calculation (n=1)	Trails A (n=4) 40%	Trails B (n=6) 60%
S010				X (mild)	X(mild)
S226	X (mod)				X(mild)
S201		X (mild)		X (mod)	X(mod)
S151					
S259		X (mild		X (mod)	X(mod)
S356					
S209				X (mod)	X(mod)
S413	X (mod)	X(mod)	X (mild)		X(mild)
S324					
S013					

APPENDIX TABLE 18

Medications taken by patients during HM and HC diet phases

Medication Category	Specific drug (number of subjects taking); *=effect on taste
ACE inhibitors	Zestril (4)*; Vasotec (1)*
Anticoagulants	Coumadin (1)
Antidepressants	Zoloft (1)*; Prozac (3)*
Anti-gout	Allopurinol (1)
Antihypertensives	Prazocin (1)
Antispasmodics	Bentyl (1)
Beta-adrenergic blockers	Timoptic (ophthalmic) (1)*; Atenolol (1)*
Calcium channel blockers	Cardazim (1); Verapamil (1)
Decongestants	Actifed (1); Lorazepam (1)
Diuretics	K+sparing: Spironolactone (2); Loop: Lasix (3)
Electrolytes	K+: MicroK (1); K-Dur (1)
Gastric acid secretion inhibitors	Zantac (1); Omeprazole (3)
Hormone replacement	Premarin (oral, cream)(6); thyroid (4)
Nonsteroidal analgesics	ASA (4); Ectorin (1); Advil (1); Voltaren(1)*; Naprosyn (1)*; Tylenol (1); Relafen (1)*
Skeletal muscle relaxants	Flexeril (1)
Stool softeners	Colace (2)
Vasodilators	Dypridamole (1); Isorbide (1); Nitropatch (1)
Vitamins	Calcium (Tums) (2)

Source of information: US Pharmacopeial Convention (1995). Advice for the patient: Drug information in lay language, 16th Ed, Vol. II. Williston, VT: US Pharmacopeia.

APPENDIX FORMS

APPENDIX FORM 1

DAILY SELF CARE LOG

NAME: PATIENT ID:
DAY/DATE: WEIGHT:

BLOOD GLUCOSE TESTS	TODAY'S DIABETES MEDS
AM_____PM_____ OTHERS	ORAL AGENT:_____ INSULIN:_____ SITE OF INJECTION(S):

OTHER ASPECTS OF MY DIABETES

Low blood sugar reactions today: No Yes

If yes, time of reaction:_____Blood sugar level:_____

Severity of reaction: Mild Moderate Severe

ACTIVITY LEVEL FOR THE DAY	ALCOHOL INTAKE FOR THE DAY
My activity level for today is (circle one answer) 1 = very decreased from usual 2 = slightly decreased from usual 3 = same as usual 4 = slightly increased from usual 5 = very increased from usual	My alcohol intake for today is None Some---------> Time:_____ Ounces_____

LIVING WITH MY RESEARCH DIET

1.	I am having problems with constipation.	YES	NO
2.	I am having problems with diarrhea.	YES	NO
3.	This food is:_____		

OVERALL RATING FOR TODAY

Worst day of my life Best day of my life

APPENDIX FORM 2

THE FOOD YOU ATE TODAY (Global palatability)

Think back over all of the food you ate today in each meal and snack. If you need to, review the sheet that comes with your food to recall the various foods.

Place an "X" on the line to correspond with your feeling.

* How would you rate the overall pleasantness of all the foods that you ate today?

Very pleasant _____ Very unpleasant

* How would you rate the tastiness of all of the foods that you ate today?

Very pleasant _____ Very unpleasant

* How would you rate the overall texture of all of the foods that you ate today?

Very pleasant _____ Very unpleasant

* How would you rate the smell of all of the foods that you ate today?

Very pleasant _____ Very unpleasant

* How would you rate the appearance of all the foods that you ate today?

Very pleasant _____ Very unpleasant

* How would you rate the richness of all the foods that you ate today?

Very pleasant _____ Very unpleasant

APPENDIX FORM 3

Day One Foods: What's your rating? (Specific palatability)

Instructions: We have listed most of the foods that you received today. Now, we want to know how much you like each of them. Please give us your opinion about each food by circling your answer.

1. Sausage in gravy (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

2. Juice (kind you had_____)(circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

3. English muffin (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

APPENDIX FORM 3 (Continued)

4. Oatmeal (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

5. Fruit spread (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

6. Tuna salad (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

APPENDIX FORM 3 (Continued)

7. Bread (kind you had_____)(circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

8. Mustard (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

9. Mayonnaise (circle your answer)

   A. LIKE EXTREMELY
   B. LIKE VERY MUCH
   C. LIKE MODERATELY
   D. LIKE SLIGHTLY
   E. NEITHER/NOR
   F. DISLIKE SLIGHTLY
   G. DISLIKE MODERATELY
   H. DISLIKE VERY MUCH
   I. DISLIKE EXTREMELY

APPENDIX FORM 3 (Continued)

10. Lettuce and tomatoes (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

11. Marinated pasta salad (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

12. Italian salad dressing on pasta (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

APPENDIX FORM 3 (Continued)

13. Fresh/canned fruit (kind you had_____)(circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

14. Marinara beef sauce over spaghetti noodles (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

15. Parmesan cheese (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

APPENDIX FORM 3 (Continued)

16. Bread (kind you had_____)(circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

17. Green tossed salad (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

18. Salad dressing (kind you had_____)(circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

APPENDIX FORM 3 (Continued)

19. Pumpkin bar-cookie (circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

20. Fresh or canned fruit (kind you had_____)(circle your answer)

A. LIKE EXTREMELY
B. LIKE VERY MUCH
C. LIKE MODERATELY
D. LIKE SLIGHTLY
E. NEITHER/NOR
F. DISLIKE SLIGHTLY
G. DISLIKE MODERATELY
H. DISLIKE VERY MUCH
I. DISLIKE EXTREMELY

21. Are there any other things you want to tell us about these particular foods?

Thank you.

APPENDIX FORM 4

Before breakfast (Prospective consumption)

Instructions: Prior to eating the food you have chosen for this meal, please answer the questions below. Where it asks, place an "X" on the line to indicate what your feeling is.

1. What time of day is it?

2. How strong is your desire to eat right now?

Very strong — Very weak

3. How hungry do you feel?

As hungry as I have ever felt — Not at all hungry

4. How full do you feel?

Very full — Not at all full

5. How much food do you think you could eat?

A large amount — Nothing at all

6. Considering that you are on this research study diet, how much food do you think you could eat right now?

More than what is available for this meal | Just what is available for this meal | Very little of what is available for this meal

APPENDIX FORM 5

Usual hunger/fullness & food intake pattern