Primary prevention of atherosclerosis and obesity in young adults using dietary and educational interventions

Oksana Alexandrovna Matvienko
Iowa State University
INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

ProQuest Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600

UMI®
Primary prevention of atherosclerosis and obesity in young adults using dietary and educational interventions

by

Oksana Alexandrovna Matvienko

A dissertation submitted to the graduate faculty in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

Major: Nutrition

Program of Study Committee:
Douglas S. Lewis, Co-major Professor
Elisabeth Schafer, Co-major Professor
D. Lee Alekel
Patricia Murphy
Donald Beitz

Iowa State University
Ames, Iowa
2002

Copyright © Oksana Alexandrovna Matvienko, 2002. All rights reserved.
Graduate College
Iowa State University

This is to certify that the doctoral dissertation of

Oksana Alexandrovna Matvienko

has met dissertation requirements of Iowa State University

Signature was redacted for privacy.

Committee Member

Signature was redacted for privacy.

Committee Member

Signature was redacted for privacy.

Committee Member

Signature was redacted for privacy.

Co-Major Professor

Signature was redacted for privacy.

Co-Major Professor

Signature was redacted for privacy.

For the Major Program
# TABLE OF CONTENTS

## LIST OF FIGURES

vi

## LIST OF TABLES

vii

## ABSTRACT

viii

## CHAPTER 1. GENERAL INTRODUCTION

1

Dissertation organization

2

References

3

## CHAPTER 2. LITERATURE REVIEW

4

Part I. Primary prevention of atherosclerosis in young adults using dietary interventions

4

Prevalence of atherosclerosis and associated diseases in the United States

4

Importance of early prevention of atherosclerosis

4

Onset of atherosclerosis in adolescence

5

Atherosclerosis is difficult to reverse

6

Hypercholesterolemia – a major risk factor of atherosclerosis

7

Role of hypercholesterolemia in the pathogenesis of atherosclerosis

8

Dietary approaches to lower cholesterol in the general population

11

Phytosterols – effective cholesterol-lowering agents

12

Evidence of phytosterol effectiveness in lowering serum cholesterol

12

Plant sterols and stanols: structure, dietary intakes, and absorption

13

Hypocholesterolemic mechanism of phytosterols

16

Potential adverse effects of phytosterol consumption

18

Research directions

19

References

19

Part II. Primary prevention of obesity in young adults using educational interventions

26

Prevalence of overweight and obesity in the United States

26

Weight change patterns in adulthood

28

Health and economic consequences of obesity

30

Obesity treatment: success and limitations

33

Dietary interventions

34

Pharmacotherapy

35

Obesity prevention: success and limitations

37

Evidence of the nutrition knowledge-behavior association

39

Nutrition education is a cornerstone of prevention interventions

40

Nutrition education: definition, goals, and research directions

41

Fundamentals of nutrition education

41

Determinants of the information quality

42

Selecting a target audience for primary prevention of obesity

43

Receiver input

43
<table>
<thead>
<tr>
<th>CHAPTER 3. A SINGLE DAILY DOSE OF SOYBEAN PLANT STEROLS IN GROUND BEEF DECREASES SERUM TOTAL AND LOW-DENSITY LIPOPROTEIN CHOLESTEROL IN YOUNG, MILDLY HYPERCHOLESTEROLEMIC MEN</th>
<th>52</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abstract</td>
<td>52</td>
</tr>
<tr>
<td>Introduction</td>
<td>53</td>
</tr>
<tr>
<td>Subjects and methods</td>
<td>54</td>
</tr>
<tr>
<td>Subjects</td>
<td>54</td>
</tr>
<tr>
<td>Study design</td>
<td>55</td>
</tr>
<tr>
<td>Nutrient intake, health history, and body weight and height</td>
<td>56</td>
</tr>
<tr>
<td>Plasma lipid measurements</td>
<td>56</td>
</tr>
<tr>
<td>Neutral sterol quantitation</td>
<td>57</td>
</tr>
<tr>
<td>Statistical analyses</td>
<td>58</td>
</tr>
<tr>
<td>Results</td>
<td>58</td>
</tr>
<tr>
<td>Subject baseline characteristics</td>
<td>58</td>
</tr>
<tr>
<td>Plasma lipid concentrations</td>
<td>59</td>
</tr>
<tr>
<td>Lipoprotein subclass patterns</td>
<td>59</td>
</tr>
<tr>
<td>Plasma neutral sterol concentrations</td>
<td>60</td>
</tr>
<tr>
<td>Family history of CVD and plasma LDL cholesterol</td>
<td>60</td>
</tr>
<tr>
<td>Body weight and plasma metabolic variables</td>
<td>60</td>
</tr>
<tr>
<td>Discussion</td>
<td>61</td>
</tr>
<tr>
<td>Acknowledgement</td>
<td>64</td>
</tr>
<tr>
<td>References</td>
<td>64</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHAPTER 4. A COLLEGE NUTRITION SCIENCE COURSE AS AN INTERVENTION TO PREVENT WEIGHT GAIN IN FEMALE COLLEGE FRESHMEN</th>
<th>74</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abstract</td>
<td>74</td>
</tr>
<tr>
<td>Introduction</td>
<td>75</td>
</tr>
<tr>
<td>Methods</td>
<td>75</td>
</tr>
<tr>
<td>Subjects</td>
<td>75</td>
</tr>
<tr>
<td>Study design</td>
<td>76</td>
</tr>
<tr>
<td>Intervention</td>
<td>77</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>78</td>
</tr>
<tr>
<td>Results</td>
<td>78</td>
</tr>
<tr>
<td>Subject characteristics</td>
<td>78</td>
</tr>
<tr>
<td>Knowledge</td>
<td>78</td>
</tr>
<tr>
<td>Dietary intake</td>
<td>79</td>
</tr>
<tr>
<td>Body weight and BMI</td>
<td>79</td>
</tr>
<tr>
<td>Interaction of baseline BMI status with the intervention</td>
<td>80</td>
</tr>
<tr>
<td>Discussion</td>
<td>80</td>
</tr>
</tbody>
</table>
CHAPTER 5. COLLEGE STUDENTS' PERCEPTION OF OWN WEIGHT, ATTITUDES TOWARD DIETING, AND KNOWLEDGE AND BELIEFS ABOUT BODY WEIGHT

Abstract

Introduction

Methods

Subjects and study design

Statistical analysis

Results

Subjects

Distribution of body mass index

Energy expenditure

Weight concerns

Perception of own weight and desire to change weight

Perceived confidence in the ability to lose weight

Attempts and strategies to lose weight

Attitude toward weight loss supplements

Knowledge

Discussion

Implications for research and practice

References

CHAPTER 6. GENERAL CONCLUSIONS

APPENDIX A. CONSENT FORM AND DATA COLLECTION INSTRUMENTS FOR STUDY 1

Consent form

Nutrition and health questionnaire

Family history of heart disease and diabetes

APPENDIX B. CONSENT FORM AND DATA COLLECTION INSTRUMENTS FOR STUDY 3

Consent form

Survey

Knowledge test

Three-day physical activity record

ACKNOWLEDGEMENT
**LIST OF FIGURES**

| Figure 2.1. | Factors and pathophysiological processes involved in the onset and progression of atherosclerosis and associated diseases | 9 |
| Figure 2.2. | Structures of cholesterol and the two most common dietary plant sterols and their respective 5-dihydro derivatives | 15 |
| Figure 2.3. | Age-adjusted prevalence of overweight and obesity among U.S. adults, age 20-74 years, by sex and survey | 26 |
| Figure 2.4. | Prevalence of weight control status by sex: results from the Behavioral Risk Factor Surveillance System Surveys (BRFSS) | 27 |
| Figure 2.5. | Average weight gain in 18-30 year-old adults between baseline and 2, 5, and 7 years of the follow-up | 29 |
| Figure 2.6. | Relative risk of developing coronary heart disease by the amount of weight gained after age 18 | 31 |
| Figure 2.7. | Relative risk of heart attack, high blood pressure, and diabetes in weight gainers compared with weight maintainers | 32 |
| Figure 3.1. | Correlation between the percentage change in LDL-cholesterol from baseline and soybean phytosterol consumption | 73 |
| Figure 4.1. | Effect of the college course on the acquisition and retention of knowledge domains | 91 |
| Figure 5.1. | Body mass index distribution in men and women | 110 |
| Figure 5.2. | Correlation between the amount of time spent on both structured exercise and non-structured physical activities of moderate-high intensities and body mass index | 111 |
| Figure 5.3. | Women and men with a BMI less than 25 grouped by perceptions of their own weight | 112 |
| Figure 5.4. | Desire to change weight by gender | 113 |
LIST OF TABLES

Table 2.1. Effects of plant sterol or stanol treatment on plasma total and LDL-cholesterol concentrations in humans: results of selected randomized, double-blind studies of healthy normocholesterolemic and hypercholesterolemic males and females

Table 2.2. Weight maintenance after a weight-loss program

Table 2.3. Weight changes in response to low-intensity education: results of community-based interventions

Table 3.1. Lipid and sterol composition of control and phytosterol-supplemented cooked ground beef samples

Table 3.2. Baseline characteristics of subjects in the control and phytosterol-supplemented groups and of subjects who did not meet selection criteria

Table 3.3. Total plasma cholesterol, LDL cholesterol, HDL cholesterol, and triacylglycerol concentrations and total:HDL cholesterol in subjects at initial screening, baseline and after 2 and 4 weeks of consuming control or phytosterol-supplemented (treatment) ground beef

Table 3.4. Effect of phytosterol-supplemented ground beef on LDL and HDL subclass traits

Table 4.1. Course outcomes

Table 4.2. Characteristics of the intervention and control groups at baseline

Table 4.3. Body composition and energy and nutrient intakes at baseline, 4 months, and 16 months

Table 4.4. Characteristics of the higher BMI intervention and higher BMI control groups

Table 5.1. Perception by college women and men of own weight, weight concerns, and confidence in the ability to lose weight

Table 5.2. Students' responses to the knowledge test
ABSTRACT

Atherosclerosis is the underlying mechanism of coronary heart disease and stroke, the leading causes of disability and mortality in the United States. Obesity is a health condition that exacerbates atherosclerosis. Primary prevention of both atherosclerosis and obesity should begin before the age of 25. The purpose of the studies presented in this dissertation was to examine specific dietary and educational strategies to prevent atherosclerosis and obesity in young adults.

The main objective of the first study was to determine the effect of a single daily dose of soybean phytosterols added to ground beef on plasma total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) concentrations in mildly hypercholesterolemic young men. In a triple-blind 4-week study, 34 male college students with elevated plasma TC (5.85 ± 0.70 mmol/L), LDL-C (4.02 ± 0.60 mmol/L), and TC to high density lipoprotein cholesterol (HDL-C) ratio (5.5 ± 1.2) were randomly assigned to the control (ground beef alone) or treatment (ground beef with 2.7 g of phytosterols) groups. Consumption of phytosterol-fortified ground beef lowered plasma TC, LDL-C, and the TC:HDL-C by 9%, 15% and 10%, respectively, compared with the control group (p < 0.001). The LDL particle size did not change, suggesting that the decrease was primarily of particle number. The decreases were similar in subjects with (n = 8) and without (n = 9) a family history of premature cardiovascular disease (CVD) in the treatment group. Thus, phytosterol-fortified ground beef effectively lowers plasma TC and LDL-C and has the potential to become a functional food to help reduce the risk of CVD.

The objective of the second study was to test the hypothesis that a nutrition course that stresses fundamental principles of human physiology, energy metabolism, and genetics helps prevent weight gain during the first 16 months of college life. Forty female college freshmen who volunteered to participate in the study were randomly assigned to the intervention (college course, n = 21) or control (no course, n = 19) groups. The intervention was a one-semester nutrition science college course. Body weight, nutrient intakes, and knowledge were assessed at baseline, the end of the intervention (4 months from baseline), and one year later (16 months from baseline). The results showed that at 4 months higher
body mass index (BMI > 24) students in the intervention group (n = 11) consumed less fat (p = 0.04), protein (p = 0.03), and carbohydrate (p = 0.008) than did the higher BMI students in the control group (n = 6). Dietary changes reported by the higher BMI intervention students were associated with the maintenance of baseline body weight for one year in contrast with the higher BMI control students who gained 8.4 ± 6.8 kg (p = 0.012). The findings suggest that nutrition education emphasizing human physiology and energy metabolism is an effective strategy to prevent weight gain in at-risk college students.

The main objectives of the third study were to examine college students' concerns, attitudes, beliefs, and knowledge regarding body weight. A written questionnaire was administered to 220 undergraduate students. The results showed that 75% of students had a BMI < 25, yet 77% wanted to lose weight and 74% reported a history of weight loss attempts. Weight-loss strategies used most frequently by students included energy and fat restrictions and exercise followed by meal skipping and weight-loss supplements. Sixty-seven percent of students perceived themselves at low risk for future weight gain. Male and female students differed significantly in their perceived ability to control body weight and their reasons for weight concern. Students had misconceptions about the effects of macronutrients and certain foods on body fat deposition. Students generally were aware of biologic and lifestyle factors influencing body weight and fatness but had limited understanding of the mechanisms by which these factors exert their effects. BMI, academic major, and year of college attendance had fewer significant influences on students' weight-related attitudes and beliefs than did gender. Our findings suggest that education-based interventions to prevent obesity in college students should take into account gender-specific attitudes, beliefs, knowledge, and practices regarding weight.
CHAPTER 1: GENERAL INTRODUCTION

Coronary atherosclerosis and obesity are major health problems. Atherosclerosis is the underlying mechanism of coronary heart disease and stroke, the leading causes of disability and mortality in the United States. Obesity or excess body fatness is present in 60 percent of Americans (National Center for Health Statistics, 2002). Obesity exacerbates atherosclerosis and increases risk of developing other serious health conditions (Must et al., 1999).

Once established, obesity and atherosclerosis are difficult if not impossible to reverse. Fortunately, these conditions are preventable in a substantial proportion of the population. A healthful diet and a physically active lifestyle are well-known prevention strategies to lower the risk of major weight gain and atherosclerosis. There are three levels of prevention: primary, secondary, and tertiary. Primary prevention is aimed at decreasing the number of new cases of a disease, whereas the goals of secondary and tertiary prevention are to lower the rate of established cases and to stop the advancement of an existing disease, respectively (Thomas, 1995). Primary prevention is an ideal approach to deal with the issue of obesity and atherosclerosis at the population level; however, effective prevention interventions have yet to be developed. Prevention of chronic diseases is a relatively new area of research that has numerous knowledge gaps and lacks a strong theoretical foundation and empirical data. On a brighter side, health and medical experts unanimously agree that interventions to prevent obesity and atherosclerosis should be the leading priority, and the only way to achieve substantial reductions in morbidity and mortality due to these diseases (American Dietetics Association, 1996; Cooper et al., 2000). Recognition of the importance of prevention by health professionals will hopefully lead to the emergence of quality interventions in the near future.

Nutrition and health scientists are faced with a major challenge of finding preventive strategies that would be potent enough to significantly reduce the incidence of atherosclerosis and obesity in the United States and throughout the world. Such strategies could be directed toward the whole population or high-risk groups. One high-risk group is comprised of adults in their late teens and early twenties. Although these individuals are generally healthy, a few
years of consuming a high-fat diet and having a sedentary lifestyle considerably increase their risk of gaining excess body weight and/or developing atherosclerosis during the first two decades of their adult lives. Therefore, young adulthood offers a window of opportunity for interventions aimed at preventing the onset of nutrition-related health problems.

Disease prevention can be approached in a number of ways. Nutrition education is one strategy. The intention of educating individuals about the relationship between diet and health is to encourage these individuals to adopt sound dietary practices associated with a low risk of developing a health problem. Designing functional foods is another example of a prevention strategy. Functional foods contain biologically active compounds in amounts sufficient to produce favorable changes in the consumers' risk factors associated with a particular disease. It is, however, a combination of various methods rather than a single strategy that would ultimately make a difference in individual health.

In light of the importance of early prevention of chronic diseases, the studies presented in this dissertation were conducted with the intention to make a contribution to the field of primary prevention of obesity and coronary atherosclerosis in young adults. Simultaneous prevention of the two health problems is important because obesity is a major risk factor of atherosclerosis. The purpose of the first study was to determine the effects of a functional food containing phytosterols on serum cholesterol concentrations in young men. The second study examined the effectiveness of a science-based nutrition course in lowering the risk of weight gain in college women. The objective of the third study was to identify weight-related attitudes, knowledge, beliefs, and practices of college students that might be used for designing quality obesity prevention interventions tailored to the specific needs of young adults.

**Dissertation Organization**

This dissertation is organized in five chapters: a general introduction, a review of literature, three manuscripts, general conclusions, and appendices. The references cited in the general introduction, conclusion, and review of literature are listed after the appendices; references cited in each manuscript are included at the end of that manuscript.
References


CHAPTER 2: LITERATURE REVIEW
PART I. PRIMARY PREVENTION OF ATHEROSCLEROSIS IN YOUNG ADULTS USING DIETARY INTERVENTIONS

Prevalence of Atherosclerosis and Associated Diseases in the United States

Atherosclerosis is a slowly progressing inflammatory disease that is characterized by irregularly distributed lipid deposits in the intima (i.e., the innermost layer) of large and medium-sized arteries. Such deposits progress over a lifetime from fatty streaks to complicated lesions. Advanced lesions or plaques cause narrowing (stenosis) of the arterial lumen. Rupture of a plaque leads to the formation of a thrombus, occlusion of the involved artery, and ischemia (i.e., mechanical obstruction of the blood supply) of the heart, brain, or legs (McGill & McMahan, 1998).

Atherosclerosis is the underlying mechanism of coronary heart disease and stroke, the two most common forms of cardiovascular disease and the leading causes of disability and mortality in the United States. Combined, coronary heart disease and stroke account for 618,289 deaths or 65 percent of all deaths attributed to cardiovascular disease each year (American Heart Association, 2000). Advancements in the understanding of the pathogenesis of atherosclerosis in the 20th century resulted in the emergence of new treatments and medications allowing the prolongation of lives of patients with chronic forms of cardiovascular disease (Hunink et al., 1997). As a result, rates of mortality due to heart disease and stroke fell 60 percent between the 1960s and 1990s (Martin et al., 1999; National Heart, Lung, and Blood Institute, 1998). In contrast, the number of Americans developing atherosclerotic diseases changed only slightly. For example, between 1988 and 1998, the rate of death from coronary heart disease decreased 20 percent, whereas the actual number of deaths decreased only three percent (American Heart Association, 2000).

Importance of Early Prevention of Atherosclerosis

While treatment helps control progression of an established disease, primary prevention deals with a health problem before it develops. Effective prevention strategies could potentially decrease the incidence of advanced atherosclerosis or considerably delay its
onset, improve quality of life at older ages, and ease the financial burden associated with atherosclerotic diseases, which is approximately $146.2 billion a year for coronary heart disease and stroke combined (American Heart Association, 2000).

Scientists and medical professionals recognize the importance of early prevention of atherosclerotic diseases (Labarthe, 1999). Atherosclerosis begins in childhood (Strong et al., 1999). Its progression is accelerated during young adulthood under the influence of multiple risk factors, many of which are controllable with a healthy lifestyle (Berenson et al., 1998a; Willett et al., 1995). In addition, advanced atherosclerosis is only partially reversible even with the most rigorous treatments and newest medications.

**Onset of atherosclerosis in adolescence**

The evidence of early development of atherosclerosis comes primarily from necropsy studies of children, adolescents, and young adults who died of external causes. These studies provided important insights into the prevalence and severity of atherosclerotic lesions in coronary arteries of young individuals. Strong and McGill (1969) examined the aortic and coronary arteries of 4,737 males and females, aged 10-39 years. Early signs of atherosclerosis, fatty streaks, were found in aortas of all subjects. Advanced lesions (i.e., fibrous plaques) were present in a few coronary arteries before age 20 but increased in number and extent in older subjects. Similar findings have been reported by others (Stary, 1989; Berenson et al., 1992; Berenson et al., 1998a). More recently, Strong et al. (1999) summarized the autopsy results for 2,876 black and white men and women, 15-34 years of age. In agreement with previous studies, they reported that in all sex and race groups, the mean percentage of area involved with lesions and degree of lesion severity were greater in the oldest subjects aged 30-34 years, compared with the youngest subjects aged 15-19 years. The prevalence of total lesions in the right coronary artery increased from about 60 percent in the 15-19 year-olds to greater than 80 percent in men and 70 percent in women aged 30-34 years. Tuzcu and colleagues (2001) were able to determine the extent of atherosclerosis in 262 living young adults who had undergone a heart transplant procedure. Before transplantation, all donors were screened using coronary angiography, a technique that allows visualization of coronary arteries after the injection of a radioactive material, to identify those who were free of cardiovascular disease. Within 30 days after the...
transplantation, the patients were examined using intravascular ultrasound. This imaging technique permits direct examination of vessel walls for intimal thickness and detects early atherosclerotic lesions that are not visible with angiography. Of all cardiac transplant patients, 51.9 percent had clinically significant coronary atherosclerotic lesions (intimal thickness >0.5 mm) and coronary stenosis averaging 32.7 percent. The intimal thickness and percentage area stenosis were significantly correlated with donor age. The prevalence of coronary lesions with intimal thickness greater than 0.5 mm was 17, 37, 60, 71, and 85 percent in donors aged 13-19, 20-29, 30-39, 40-49, and older than 50 years, respectively.

Atherosclerosis is difficult to reverse

Cardiovascular disease is the result of an atherosclerotic process of several decades and becomes clinically diagnosable usually after the age of 40. Advanced lesions may not be amenable to treatment, but some regression in their size and severity may be possible with intensive lifestyle modifications and treatment. A number of secondary prevention clinical trials have examined the effects of dietary approaches on changes in risk factors of patients with established heart disease. Impressive changes were observed in participants of the Lifestyle Heart Trial (Ornish et al., 1990). In this trial 48 patients with coronary atherosclerosis were assigned to either a usual-care control or an experimental group. The experimental treatment included a very low-fat (<10% of energy) vegetarian diet, smoking cessation, stress management, and regular physical activity of moderate intensity. One year from baseline the experimental group showed a significant 37 percent reduction in cholesterol concentrations and a regression in percentage diameter stenosis from 40 to 37.8 percent. In contrast, the control group showed an increase in diameter stenosis from 42.7 to 46.1 percent. Overall, 82 percent of patients in the experimental group showed regression, and 53 percent of patients in the control group showed progression in percentage diameter stenosis. In the experimental group the greatest improvements were observed in the patients who had the highest adherence to the treatment. In the Stanford Coronary Risk Intervention Project 300 men and women with heart disease were assigned to an intensive treatment or a control (usual-care) group and followed for four years (Haskell et al., 1994; Quinn et al., 1994). The intensive treatment patients consumed a low-fat diet (<20% of energy) and took lipid-lowering medications. In addition, the patients were instructed to lose weight, stop
smoking, and increase their level of physical activity. At the end of the four-year period the intensive treatment group showed a significant 22 percent reduction in low-density lipoprotein (LDL) cholesterol concentrations compared with the control group. Stenosis of coronary arteries was on average 47 percent less in the treatment than in the control patients. Also, the intensive treatment patients had significantly fewer hospitalizations due to cardiac events than did the control patients. Several other dietary interventions have reported reductions in LDL-cholesterol concentrations and improvements in disease end points of variable magnitude (Turpeinen et al., 1968; Miettinen et al., 1972; Turpeinen, 1979; de Lorgeril et al., 1994; de Lorgeril et al., 1999; Frantz et al., 1989; Watts et al., 1992; Blankenhorn et al., 1990). Drugs such as HMG CoA reductase inhibitors (statins), bile acid sequestants, nicotinic acid, and fibric acids, effectively lower LDL-cholesterol concentrations five to 55 percent (National Cholesterol Education Program, 2001). Benefits of drug therapy include 30 and 26 percent reductions in cardiovascular events and mortality, respectively (Pignone et al., 2000). However, long-term safety of drug therapy remains to be determined (Pignone et al., 2001).

**Hypercholesterolemia – a Major Risk Factor of Atherosclerosis**

Clinical and epidemiologic studies have identified family history, older age, male gender, African and Hispanic races, dyslipidemia, hypertension, obesity, diabetes, cigarette smoking, high-fat low fiber diets, and a sedentary lifestyle as risk factors of atherosclerosis (American Heart Association, 2000; National Cholesterol Education Program, 2001). High-fat diets have been linked to atherosclerosis primarily through their effects on serum lipid concentrations (Schaefer, 2002; Pignone et al., 2001; Menotti, 1999). Dietary cholesterol and saturated fat are thought to act synergistically in raising serum total cholesterol and LDL-cholesterol above the desirable levels: less than 200 mg/dl (5.17 mmol/L) for total cholesterol and less than 100 mg/dl (2.58 mmol/L) for LDL-cholesterol (Schaefer, 2002; Gylling & Miettinen, 2001; National Cholesterol Education Program, 2001). Saturated fatty acids increase serum LDL-cholesterol concentrations by interfering with LDL receptor synthesis and activity, thereby decreasing the receptor-mediated removal of cholesterol from the circulation (Nicolosi et al., 1990; Spady et al., 1983; Bucci et al., 1998). Compared to
saturated fatty acids, dietary cholesterol appears to have a much less potent effect on serum cholesterol concentrations. However, there is evidence that high dietary intakes of cholesterol increase hepatic cholesterol content subsequently leading to downregulation of LDL receptor synthesis (Grundy, 1998). Also, trans fatty acids, the products of hydrogenation in which liquid vegetable oils are converted to solid fats used to make margarine, raise serum LDL-cholesterol and lower high-density lipoprotein (HDL) cholesterol concentrations by mechanisms not completely understood (Ascherio & Willett, 1997). In addition to diet, elevated serum cholesterol can be caused by genetic defects, such as mutations of the LDL-receptor gene found in familial hypercholesterolemia (Pisciotta et al., 2002). Genetically-induced hypercholesterolemia is present in a small proportion (~5%) of the population (Grundy, 1998; Dammerman & Breslow, 1995).

Hypercholesterolemia is a major independent risk factor of atherosclerosis (Klag et al., 1993; Anderson et al., 1987). There is a strong, graded relationship between serum cholesterol concentration and risk of cardiovascular morbidity and mortality, which begins rising at serum total cholesterol levels above 160-180 mg/dl (4.14-4.65 mmol/L) (Stamler et al., 1986; Neaton et al., 1992). In the study of 11,017 men aged 18-39 years who were followed for over three decades, an estimated risk of death from heart disease was 2.2-3.6 times greater, and life expectancy was 3.8-8.7 years shorter for men having plasma total cholesterol levels above 240 mg/dl (6.21 mmol/L) compared with those having total cholesterol levels below 200 mg/dl (5.17 mmol/L) (Stamler et al., 2000). Elevated serum cholesterol in young adulthood significantly increases risk of heart disease in middle age. Among 1,017 men, the risk of developing heart disease during a 42-year-follow-up was two-fold higher for men who were in the 75th percentile of cholesterol level compared with those who were in the 25th percentile at baseline (Klag et al., 1993). In the Framingham study of 1,959 men and 2,415 women under 50 years of age, serum cholesterol concentrations were significantly associated with cardiovascular mortality: for each 10 mg/dl increase in cholesterol there was a nine percent increase in death from cardiovascular disease (Anderson et al., 1987).

Role of hypercholesterolemia in the pathogenesis of atherosclerosis

The pathogenesis of atherosclerosis is multifactorial and complex (Figure 2.1).
Individual factors
- Age
- Gender
- Race
- Family history
- Menopause

Lifestyle
- Cigarette smoking
- Obesity
- Psychological stress
- Dietary intakes

Wall shear stress
- Heart rate
- Blood velocity
- Vessel bifurcations

Health condition
- Hypercholesterolemia
- Atherogenic blood lipids
- Arterial hypertension
- Diabetes mellitus
- Metabolic syndrome
- Homocysteinemia
- Inflammation
- Infection

Hemostaseological factors
- Blood coagulation factors (fibrinogen, factor VII, tissue plasminogen activator, plasminogen-activator inhibitor type 1, D-dimer)
- C-reactive protein

Alteration of the endothelium and endothelial injury
Atherosclerotic process

- Coronary heart disease
- Stroke
- Gangrene of the legs

Figure 2.1. Factors and pathophysiological processes involved in the onset and progression of atherosclerosis and associated diseases. Figure adapted with modifications from Hanke et al. (2001).
At the tissue level, atherosclerosis begins with endothelial dysfunction (i.e., injury) caused by modified plasma lipoproteins, free radicals from cigarette smoking, hypertension, toxins, infectious microorganisms, and other factors (Hanke et al., 2001; Ross, 1999). The endothelium is a single layer of flat cells lining a blood vessel and serving as a barrier between the blood stream and the vessel itself. Injured endothelium has increased adhesiveness and permeability to circulating lipoproteins, monocytes, platelets, and other plasma constituents that are involved in an inflammatory response. Monocytes penetrate the arterial wall at the site of injury, undergo a number of complex phenotypic alterations, and transform into macrophages that express scavenger receptors for modified lipoproteins. In hypercholesterolemia, there is an abundance of circulating lipoproteins, such as LDL and very low-density lipoproteins, that are readily available for enzymatic and oxidative modifications by endothelial cells, the underlying smooth muscle cells, and macrophages themselves. Macrophages rapidly internalize and accumulate modified lipoproteins, which are chemotactic and capable of recruiting more monocytes and other cells to the affected area. In addition, macrophages secrete cytokines, chemokines, growth-regulating molecules, and hydrolytic enzymes that sustain the inflammatory state. Concurrently with the process of lipid accumulation in macrophages, also called foam cells, altered endothelium produces a variety of growth factors that stimulate migration and proliferation of subendothelial smooth muscle and connective tissue cells, which are involved in the formation of fibromuscular extracellular matrix. Together the matrix and lipid-filled macrophages form a plaque or a lesion, which increases in size in the presence of continuing atherogenic stimuli. Eventually the plaque will rupture and release its content -- lipid-rich, necrotic debris -- into the blood. Rupture of a plaque is accompanied by the formation of a thrombus, which causes occlusion of the blood vessel and ischemia of the affected organ (Castelli, 1998; Hanke et al., 2001; Ross 1999; McGill & McMahan, 1998; Fuster et al., 1992; Steinberg & Witztum, 1990).

In the broader view, although many factors are involved in the process of atherosclerosis, hypercholesterolemia remains an important target for prevention. It has been estimated that in 27 percent of men and 34 percent of women, chronically elevated serum cholesterol appears to be the principal contributor to disease progression (Wilson et al., 1998). In hypercholesterolemia, the cardiovascular system is saturated with lipoproteins that
are readily available for oxidative modification and uptake by macrophages. Although many aspects of the lipoprotein involvement in the process of atherosclerosis are not completely understood, emerging evidence indicates that modified lipoproteins may injure the endothelium, alter endothelial homeostatic properties, and promote a sustained inflammatory response, proliferation of macrophages and smooth muscle cells, and platelet aggregation (Ross, 1999; Chisolm & Chai, 2000).

**Dietary Approaches to Lower Cholesterol in the General Population**

Recognizing the importance of maintaining optimal plasma cholesterol concentrations for prevention of atherosclerotic diseases, the American Heart Association (AHA) has developed dietary guidelines for the general population. Because high-fat diets contribute to the onset and progression of atherosclerosis by raising serum cholesterol concentrations, the AHA advises individuals to limit total fat consumption to less than 30 percent of daily energy intake, saturated fat consumption to less than 10 percent of energy, and cholesterol to less than 300 mg/d (AHA Dietary Guidelines, 2000). For higher-risk individuals with elevated plasma cholesterol concentrations, established cardiovascular disease, and certain other health conditions, the guidelines are more stringent: limit total and saturated fat intakes to less than 30 and seven percent of energy, respectively, and reduce daily cholesterol intake to 200 mg. Metabolic ward studies, in which subjects’ food consumption is carefully controlled by researchers for the duration of a study showed that partial replacement of saturated fat with complex carbohydrates and unsaturated fats, and a 60 percent reduction in cholesterol intake can lower plasma total cholesterol concentration by 10-15 percent in individuals consuming weight-maintaining diets (Clarke et al., 1997). It has been estimated that a 10 percent reduction in plasma total cholesterol sustained for at least five years is associated with a 50 percent decreased risk of heart disease at age 40 (Law et al., 1994).

Americans meet the established guidelines only partially. Based on the results of the third National Health and Nutrition Examination Survey (1988-94), adult men and women, on average, consume 34 percent of total energy as fat, and 11.7 percent as saturated fat. Average daily intakes of cholesterol are estimated to be 349 mg for men and 230 mg for women (Ernst et al., 1997). Tang et al. (1998) reviewed 19 randomized controlled trials that
were conducted to determine the effect of dietary advice about the current recommendations for fat intake on serum total cholesterol concentrations of free-living individuals. The authors estimated that consumption of diets meeting the AHA guidelines for the general population for at least six months reduced total cholesterol, on average, only by three percent (95% confidence interval: 1.8% to 4.1%). Consumption of diets meeting the AHA guidelines for high-risk individuals reduced total cholesterol by 5.6 percent (95% confidence interval: 4.7% to 6.5%). The authors pointed out that the discrepancy between the results of the metabolic ward studies and the studies of free-living individuals was due to the limited compliance with dietary advice of free-living individuals. Clearly, effective interventions are needed to help the general public adopt the current recommendations for fat intake.

Reductions in fat and cholesterol intakes are not the only dietary approach to lower serum cholesterol concentrations. Certain nutritive and non-nutritive food components can also influence blood lipid levels. For example, a substitution of saturated fat with mono- and poly-unsaturated fatty acids has been shown to lower serum LDL-cholesterol concentrations by 7-15 percent (Lichtenstein et al., 1993; Schaefer, 2002). High intakes of soluble dietary fiber decrease LDL-cholesterol on average by 8-12 percent (Fernandez, 2001; Leinonen et al., 2000; Wolk et al., 1999; Van Horn, 1997). Soy proteins are currently under investigation for possible hypocholesterolemic properties (Sirtori & Lovati, 2001; Messina et al., 2002; Anderson et al., 1995). Finally, phytosterols, non-nutritive compounds found in plant tissues, have undergone intense scrutiny during the last quarter of the 20th century for their now well-established ability to lower serum cholesterol concentrations in humans. Phytosterols are the focus of the following discussion.

**Phytosterols - Effective Cholesterol-Lowering Agents**

**Evidence of phytosterol effectiveness in lowering serum cholesterol**

Many well-designed human studies showed that consumption of food products supplemented with 1.6-3.5 g/d of phytosterols on average decreases serum LDL- and total cholesterol concentrations by 15 and 10 percent, respectively, while having no effect on HDL-cholesterol and triacylglycerol concentrations (Miettinen & Gylling, 1999). Table 2.1 summarizes the results of several recent studies with normo- and hyper-cholesterolemic
subjects treated with phytosterol-supplemented foods. In these studies phytosterols were added to high- and reduced-fat margarines, table spread, butter, or low-fat yogurt. With one exception (Plat et al., 2000b), a daily phytosterol dose was divided between three meals. Reductions in total and LDL-cholesterol concentrations were statistically significant in all studies. Although most studies lasted over three weeks, significant decreases in cholesterol concentrations were reported after only one week of consuming a phytosterol-supplemented food (Mensink et al., 2002).

**Plant sterols and stanols: structure, dietary intake, and absorption**

Phytosterols are structurally very similar to cholesterol but have distinct side chain configurations at the C24 position. As cholesterol, phytosterols, such as sitosterol and campesterol, are 4-desmethylsterols. They have one unsaturated bond in the ring portion of their structures (Figure 2.2). Saturation of the bond with hydrogen results in the formation of the related compounds campestanol and sitostanol, collectively called stanols (Ling & Jones, 1995). Humans obtain phytosterols exclusively from dietary sources, including vegetable oils, seeds, nuts, cereals, beans, and some fruits and vegetables. Plant stanols are extracted from pine wood pulp or can be synthetically derived from plant sterols. Phytosterols most commonly occurring in nature are beta-sitosterol, campesterol, and stigmasterol (Moghadasian & Frohlich, 1999). In Western diets the intake of phytosterols is comparable with that of cholesterol and ranges from 160 mg/d to 360 mg/d (Patel et al., 1998). Vegetarians consume considerably larger amounts, 400-500 mg/d (Ling & Jones, 1995).

Phytosterols are partially absorbed in the small intestine. In humans absorption efficiency is estimated to be approximately 10 percent for campesterol and campestanol, four to five percent for sitosterol and stigmasterol, and negligible for sitostanol (Child & Kuksis, 1983; Heinemann et al., 1993; Gylling et al., 1999a), whereas cholesterol absorption ranges from 33 to 60 percent (Kesaniemi & Miettinen, 1987; Grundy & Mok, 1977). Structural features, such as length and configuration of the side chain, are thought to contribute to the differences in the absorption efficiency between phytosterols and cholesterol (Heinemann et al., 1993; Child & Kuksis, 1983).
Table 2.1. Effects of plant sterol or stanol treatment on plasma total (TC) and LDL-cholesterol concentrations in humans: results of selected randomized, double-blind studies of healthy normocholesterolemic (NC) and hypercholesterolemic (HC) males (M) and females (F).

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subjects</th>
<th>Study Design</th>
<th>Treatment</th>
<th>Decrease$^{1}$ in LDL (%)</th>
<th>Decrease$^{1}$ in TC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mensink et al., 2002</td>
<td>60 M.F NC</td>
<td>PC$^{2}$</td>
<td>3 g/d PSNE$^{3}$ in low-fat yogurt</td>
<td>13.7</td>
<td>8.6</td>
</tr>
<tr>
<td>Temme et al., 2002</td>
<td>42 M.F NC, HC</td>
<td>PC, CO$^{2}$</td>
<td>2 g/d PSE$^{3}$ in margarine</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>Plat et al., 2000b</td>
<td>39 M.F NC, HC</td>
<td>PC, CO</td>
<td>2.5 g 1/d 2.5 g 3/d PSNE in margarine</td>
<td>10.2</td>
<td>6.6</td>
</tr>
<tr>
<td>Hallikainen et al., 2000a</td>
<td>34 M.F HC</td>
<td>RM$^{2}$</td>
<td>2 g/d PSNE or PSE in margarine</td>
<td>7.3-9.2</td>
<td>10.4-12.7</td>
</tr>
<tr>
<td>Hallikainen et al., 2000b</td>
<td>22 M.F HC</td>
<td>SB$^{2}$</td>
<td>0.8, 1.6; 2.4; 3.2 g/d PSNE in margarine</td>
<td>1.7-10.4$^{4}$</td>
<td>2.8-11.3$^{4}$</td>
</tr>
<tr>
<td>Hallikainen et al., 1999</td>
<td>55 M.F HC</td>
<td>8 wk</td>
<td>2.16-2.3 g/d 2 types of PSNE in margarine</td>
<td>8.6-13.7</td>
<td>8.1-10.6</td>
</tr>
<tr>
<td>Hendriks et al., 1999</td>
<td>100 M.F NC, HC</td>
<td>PC</td>
<td>0.83, 1.61, 3.24 g/d PSE in spread</td>
<td>6.7-10</td>
<td>5-6.8</td>
</tr>
<tr>
<td>Gylling &amp; Meittinen, 1999</td>
<td>23 F NC, HC</td>
<td>5-6 wk</td>
<td>2.43, 3.16, 3.18 g/d 3 mixes of PSNE in margarine &amp; butter</td>
<td>9-11</td>
<td>5-7</td>
</tr>
<tr>
<td>Jones et al., 1999</td>
<td>32 M HC</td>
<td>30 d</td>
<td>1.7 g/d PSNE-PSE mix in margarine</td>
<td>15.5</td>
<td>9.1</td>
</tr>
<tr>
<td>Weststrate &amp; Meijer, 1998</td>
<td>100 M.W NC, HC</td>
<td>3.5 wk</td>
<td>1.5-3.3 g/d PSE or PSNE in margarine</td>
<td>14</td>
<td>8</td>
</tr>
</tbody>
</table>

1 All decreases were significant, P value < 0.05.
2 PC, placebo-controlled; CO, crossover; RM, repeated measures; SB, single-blind.
3 PSNE, plant stanol esters; PSE, plant sterol esters.
4 Significant, P value < 0.05, for sterol doses ≥ 1.6 g.
Figure 2.2. Structures of cholesterol and the two most common dietary plant sterols and their respective 5-dihydro derivatives. Campesterol has a methyl group at C-24, and sitosterol has an ethyl group at C-24 in the side chain.
Hypocholesterolemic mechanism of phytosterols

The hypocholesterolemic effect of phytosterols is attributed to the ability of these compounds to inhibit intestinal absorption of cholesterol (Plat & Mensink, 2001). Heinemann et al. (1991) observed 50 and 85 percent reductions in cholesterol absorption after intestinal perfusion of human subjects with a high dose (3.6 μmol/min) of sitosterol or sitostanol, respectively. In 11 colectomized patients who consumed a margarine containing 2 g of stanol esters for seven days, cholesterol absorption decreased by almost 40 percent and fecal output of neutral cholesterol increased by 36 percent (Miettinen et al., 2000). In other human feeding studies stanol ester consumption was reported to reduce cholesterol absorption up to 65 percent (Gylling & Miettinen, 1994; Gylling, et al., 1997).

Phytosterols are usually esterified with vegetable oil fatty acids to increase their solubility in fat (Wong, 2001). In the intestine phytosterol esters are cleaved by cholesterol esterase. This is an important step in the phytosterol processing because only free forms of plant sterols and stanols compete with cholesterol for incorporation into bile acid micelles (Plat & Mensink, 2001). Phytosterols, being more hydrophobic than cholesterol, displace both cholesterol and bile acids from the micelles, thus lowering micellar cholesterol content (Wong, 2001). Micelles transport phytosterols across the unstirred water layer to the brush border membrane where phytosterols diffuse out of the micelles into intracellular sites. The rate of sitosterol uptake by the intestinal epithelium is about 2-2.5 times slower than that of cholesterol, suggesting that there is some discrimination in sterol uptake at the level of the brush border (Compassi et al., 1997). However, a major difference in the processing of phytosterols and cholesterol is thought to occur during intracellular esterification and incorporation into chylomicrons (Compassi et al., 1997). In animal models, 70 to 80 percent of cholesterol is esterified by intracellular acyl-coenzyme A: cholesterol acyltransferase (ACAT) to prevent it from diffusion back into the intestinal lumen. In contrast, at most 10 to 20 percent of phytosterols undergo esterification (Ikeda et al., 1988). Small amounts of sterol esters are incorporated into chylomicrons and transported to the liver, where they are readily excreted via the bile (Ling & Jones, 1995). In humans, almost all (~ 99%) phytosterols are excreted in an unesterified form (Becker et al., 1993; Miettinen et al., 2000). After excretion, phytosterols may remain in the intestine for a few hours. In experiments
with rats, the fecal recovery of orally administered $^{14}$C labeled sitostanol was 70 percent after 24 hours and 97 percent after three days (Ikeda & Sugano, 1978).

In response to phytosterol-induced inhibition of cholesterol absorption, the endogenous synthesis of cholesterol increases (Vanhanen et al., 1994; Miettinen & Vanhanen, 1994; Gylling & Miettinen, 1996; Gylling, et al., 1997; Weststrate et al., 1999; Miettinen & Gylling, 1999). However, the compensatory increase in cholesterol synthesis varies among individuals. Gylling et al. (1999a) studied hypercholesterolemic subjects who did not show a reduction in serum cholesterol concentrations after a 12-month treatment with stanol esters. The authors reported that compared to the treatment responders, the non-responders had higher efficiency of cholesterol synthesis, which was determined by changes in the cholesterol precursor concentrations during the treatment. Also, higher baseline levels of cholesterol precursors were associated with smaller reductions in serum cholesterol concentrations following the treatment. It should be noted that at least one study did not find changes in endogenous cholesterol synthesis in subjects who consumed a phytosterol-supplemented food. However, the authors reported comparing the efficiency of cholesterol synthesis between the control and treatment groups only at follow-up and not before and after the treatment (Jones et al., 1999). It has also been proposed that variability in individual responses to phytosterol treatment could be partially attributed to apolipoprotein E (apoE) genotypes. Gylling & Miettinen (1992) reported that in healthy men fed a low-fat high cholesterol diet, cholesterol absorption was the highest in those having the apoE allele 4 and the lowest in those having the apoE allele 2. More recently, Hallikainen et al. (2000b) did not find that subjects with the apoE allele 4 were significantly different from the subjects with the apoE allele 3 in their LDL-cholesterol responses to treatment with various doses of stanol esters; however, the number of subjects in each apoE genotype group was small. In another study by the same team of researchers, the presence of the apoE allele 4 did have a significant effect on the subjects' cholesterol responses to a treatment with 2 g/d of stanol esters (Hallikainen et al., 2000a). In summary, emerging evidence suggests that individuals with low efficiency of endogenous cholesterol synthesis and high efficiency of cholesterol absorption may benefit most from consuming phytosterol-fortified foods. Alternatively, in individuals with lower rates of cholesterol absorption and higher rates of cholesterol
synthesis, the hypocholesterolemic effect of phytosterols may be neutralized by an enhanced endogenous cholesterol synthesis.

Not only oral but also intravenous administration of phytosterols reduces plasma cholesterol concentrations, suggesting that in addition to the intestinal inhibition of cholesterol absorption, there is yet another mechanism by which phytosterols may interfere with cholesterol metabolism (Ling & Jones, 1995). However, the intrinsic mechanism of phytosterols in lowering circulating cholesterol is unknown.

**Potential adverse effects of phytosterol consumption**

Phytosterols are safe when consumed in moderate amounts, such as those used in human studies. In humans, phytosterol intakes of up to 25 g/d for several months were not associated with adverse health effects (Pollak & Kritschevsky, 1981). In animal studies, very large doses (0.5-5 g/kg/d) of phytosterols were reported to increase membrane fragility of erythrocytes and liver microsomes and impair the function of the reproductive system; however, these side effects disappeared soon after phytosterol supplementation was discontinued (Ling & Jones, 1995). Phytosterol-fortified foods should be avoided by individuals with phytosterolemia, an extremely rare genetic disorder characterized by unusually high rates of intestinal absorption of phytosterols and an increased risk of premature atherosclerosis (Plat & Mensink, 2001).

A concern has been raised that phytosterol doses effective for cholesterol reduction may lower blood concentrations of fat-soluble vitamins and antioxidants. A number of studies showed that phytosterols had no effect on plasma concentrations of vitamin D, retinol, or plasma-lipid-standardized alpha-tocopherol (Plat et al., 2000a; Hallikainen et al., 2000a; Hallikainen & Uusitupa, 1999; Gylling et al., 1999b). Moreover, the reports of the effect of phytosterols on concentrations of blood carotenoids (lutein, lycopene, and alpha-carotene) are controversial (Mensink et al., 2002; Weststrate & Meijer, 1998; Hendriks et al., 1999; Hallikainen & Uusitupa, 1999; Gylling & Miettinen, 1999; Seirksma et al., 1999). There seems to be general agreement that phytosterol doses greater than one g/d significantly decrease LDL-cholesterol standardized beta-carotene concentrations; however, it remains to be determined whether a reported 15-20 percent reduction in beta-carotene due to phytosterol supplementation is associated with adverse health effects (Seirksma et al., 1999). Noakes et
al. (2002) found that consumption of one or more carotenoid-rich vegetable or fruit servings a day was sufficient to prevent lowering of plasma carotenoid concentrations in 46 hypercholesterolemic subjects treated with 2.3 g of either sterol or stanol esters.

**Research Directions**

In many individuals, serum cholesterol is a major modifiable risk factor for heart disease. Incorporating phytosterols into the diet is one safe and inexpensive approach to lowering cholesterol levels. In September of 2000, the Food and Drug Administration approved use of labeling health claims about the role of plant sterol and stanol esters in reducing the risk of heart disease (Food and Drug Administration, 2000). At the present time, margarines fortified with phytosterols, such as Benecol® and Take Control®, are available to American consumers. However, consumption of these products may add between 12 and 27 g of extra fat to the diet. The high prevalence of overweight and obesity in the United States makes limiting fat consumption a priority. Therefore, it is important to explore whether phytosterols incorporated into more nutritious and less high fat foods would lower cholesterol in humans as effectively as they do when added to margarine. Also, research is needed to determine to what extent phytosterol supplementation is effective in young adults, a major target group for primary prevention of atherosclerosis. Most published studies tested the effect of phytosterols in middle-aged and older individuals. Additionally, efforts should be made to understand how phytosterols affect newly emerging risk factors of coronary atherosclerosis, for example, atherogenic lipoprotein particles.

**References**


Hallikainen MA, Sarkkinen ES, Uusitupa MI. Plant stanol esters affect serum cholesterol concentrations of hypercholesterolemic men and women in a dose-dependent manner. J Nutr. 2000b;130:767-76.


Haskell WL, Alderman EL, Fair JM, Maron DJ, Mackey SF, Superko HR, Williams PT, Johnstone IM, Champagne MA, Krauss RM. Effects of intensive multiple risk factor...


PART II. PRIMARY PREVENTION OF OBESITY IN YOUNG ADULTS USING EDUCATIONAL INTERVENTIONS

Prevalence of Overweight and Obesity in the United States

Obesity is a health condition characterized by an excessively high amount of body fat in relation to lean body mass (Stunkard & Wadden, 1993). The National Heart, Lung, and Blood Institute's guidelines define overweight, or a pre-obese state, as a body mass index (BMI) of 25 to 29.9 kg/m² and obesity as a BMI of 30 kg/m² and higher (Must et al., 1999). Obesity is further categorized as class 1 (BMI 30-34.9), class 2 (BMI 35-39.9), and class 3 (BMI ≥40). According to the results of the U.S. National Health and Nutrition Examination Surveys (NHANES), the prevalence of overweight and obesity markedly increased in the American population between 1976-80 and 1988-94, reaching epidemic proportions (Figure 2.3). Obesity has spread across all socioeconomic and demographic groups (Mokdad et al., 2001; Flegal et al., 1998). The most recent estimates from the Centers for Disease Control and Prevention showed that in 1999, 26 and 35 percent of Americans between the ages of 20 and 74 years were obese and overweight, respectively (National Center for Health Statistics, 1999).

![Figure 2.3. Age-adjusted prevalence of overweight and obesity (BMI ≥25 kg/m²) among U.S. adults, age 20-74 years, by sex and survey. Source: American Heart Association (2000).]
Similar to the trends for the general population, the prevalences of overweight and particularly obesity have increased among young adults. The proportion of 18 to 29 year-olds having a BMI greater than 30 rose from 7.1 percent in 1991 to 12.1 percent in 1998 (Mokdad et al., 1999). In addition, the proportion of adults in the same age group having a BMI between 25 and 29.9 is estimated to be 24.8% (Flegal et al., 1998). Overall, approximately 37 percent of adults under the age of 30 have a BMI above normal (≥ 25).

The increase in the prevalence of obesity has been accompanied by a growing number of Americans struggling to lose excess body fat or to maintain their weight (Williamson et al., 1992; Serdula et al., 1999; Figure 2.4). It has been estimated that individuals wanting to lose weight, on average, make one weight loss attempt every one to three years for a duration ranging from four to six weeks to four to six months (Levy & Heaton, 1993; Williamson et al., 1992).

![Figure 2.4](image)

**Figure 2.4.** Prevalence of weight control status by sex: results from the Behavioral Risk Factor Surveillance System Surveys (BRFSS). Figure prepared from data reported by Williamson et al. (1992) and Serdula et al. (1999).
Weight Change Patterns in Adulthood

A number of studies have provided important insights about the patterns of weight change throughout adulthood. Williamson and colleagues (1990) determined the incidence of major weight gain (≥5 BMI units) among adults of different age and gender groups. BMI measurements were collected for 9,862 men and women 25-74 years of age in 1971-75 and 10 years later. Participants were grouped by baseline age. The cut-off points for overweight were set at a BMI of 27.8 for men, and a BMI of 27.3 for women. At baseline, the prevalence of overweight was the lowest (17.6-24.4%) in the youngest group, aged 25-34 years, and highest among 55-74 year-old women (38.3%) and 35-44 year-old men (28.8%). In contrast, the 10-year incidence of major weight gain was the highest in the youngest group, averaging 3.9 percent for men and 8.4 percent for women, second highest (3.4-7%) in the 35-44 age group, and the lowest (<2%) in the 55-74 age group. In all age groups, women were twice as likely as men to experience a major weight gain. Baseline weight status was a predictor of major weight gain during the follow-up. Men and women, 25-44 years of age, who were overweight at baseline had 1.9 to 2.3 times higher incidence of major weight gain than did men and women of the same age not overweight at baseline. Among participants having normal body weight at baseline, 16.3 percent of men and 13.5 percent of women gained more than five BMI units before the age of 35-44. Braddon et al. (1986) tracked changes in weight in 3,249 men and women from childhood to age 36. The authors reported that during childhood the prevalence of obesity reached its peak at age 11; however, many children outgrew their obesity during the adolescent years. The prevalence of overweight and obesity began rising again after the age of 20. By age 36, 43.3 percent of men and 32.6 percent of women had a BMI of 25 and higher, compared with 12.6 percent of men and 15.8 percent of women at age 20. The proportion of men and women who were obese both in childhood (before age 11) and in adult life was only 21.4 percent of the total population obese at age 36. The majority (78.6%) of obese 36 year-olds gained excess weight after the age of 20. Yong et al. (1993) followed 202 young adults from the age of 17 years for over three decades. They observed an average weight gain of 17.3 kg for men and 7.4 kg for women between ages 17 and 34, and a gain of 5.6 kg for both men and women between ages 34 and 47. Overall, by age 47, men were 34 percent and women were 23 percent heavier.
than they were at age 17. French et al. (1996) determined weight changes in 17,233 women, participants of the Iowa Women’s Health Study. The women reported gaining on average 6.1 kg between 18 and 30 years of age and 7.2 kg between 30 and 50 years of age. Lewis and colleagues (1997) followed 3,906 white and black adults 18-30 years of age whose weights were measured at baseline and 2, 5, and 7 years later. At each time of measurement the researchers observed weight gain in all gender and race groups (Figure 2.5). At year 5 weight gain was compared between the leanest and heaviest participants (Burke et al., 1996). An average weight gain was 3.9-4.8 kg and 4.6-7.8 kg for the leanest and heaviest men, and 3.2-4.8 kg and 4.7-7.0 kg for the leanest and heaviest women, respectively. In other words, initially heavier subjects on average gained more weight than did initially leaner subjects. There was a strong association between baseline weights and year-five weights in all race and gender groups, indicating a high degree of persistence of overweightness throughout young adulthood.

![Figure 2.5](image)

**Figure 2.5.** Average weight gain (kg) in 18-30 year-old adults between baseline and 2, 5, and 7 years of the follow-up. Figure prepared from data reported by Lewis et al. (1997).

In summary, the likelihood of major weight gain between ages 18-20 and 30-34 years is high. On average, men seem to gain most of their adult weight by the mid-forties, whereas women appear to gain weight more gradually and continuously throughout adulthood.
Both overweight and lean young adults are at risk of gaining a major portion of excess weight during the first half of their adult life, although the risk of a major weight gain is higher for individuals who are overweight in their late teens and early twenties. Based on the data reported by Yong et al. (1993), Lewis et al. (1997), and French et al. (1996), average yearly rates of weight gain in young adulthood are 0.8-1.2 kg for men and 0.4-0.8 kg for women. The rates of weight gain slow down somewhat after young individuals reach their mid-thirties.

Health and Economic Consequences of Obesity

Obesity is a risk factor for many health conditions including hypertension, type two diabetes mellitus, dyslipidemia, cardiovascular disease, stroke, gallbladder disease, sleep apnea, respiratory dysfunction, osteoarthritis, deep vein thrombosis, some types of cancer, and depression (Visscher & Seidell, 2001; National Task Force, 2000). In general, the risk of developing a major health problem rises with increasing BMI and is the highest at a BMI of greater than 30 (Must et al., 1999).

Among all obesity-related health problems, cardiovascular disease, diabetes, and hypertension are health conditions most exacerbated by excess body weight. In the Framingham Heart Study, 2,252 men and 2,818 women were assessed for developing cardiovascular disease during a 26-year follow-up (Hubert et al., 1983). Over the course of the study, 31 percent of participants had developed the disease. At year 26 the incidences of cardiovascular events were highest among the heaviest men and women in the over 50 years of age group; however, the effect of overweight was most pronounced among individuals in the under 50 years age group. In the later group the incidence of coronary heart disease was 2-2.4 times higher for the heaviest men and women compared with the leanest men and women. In contrast, in the over 50 group, the incidence of heart disease was increased only 1.3-fold for both the heaviest men and women compared with their leanest counterparts. Rimm et al. (1995) reported that in middle-aged men having a BMI between 25 and 29, the risk of developing heart disease was 72 percent greater compared with similarly-aged men having a BMI less than 23. In the Nurses' Health Study of 115,818 women, both overweight status in the second half of adult life and weight changes after age 18 were associated with
the risk of developing nonfatal myocardial infarction and fatal heart disease (Willett et al., 1995). Relative risk of developing heart disease was 1.5, 2.1, and 3.6 times greater for women having a BMI of 23-24.9, 25-28.9, and above 29, respectively, compared with women having a BMI of 21 and lower. In women with a BMI of 29 and higher, 72 percent of risk was attributed to excess body weight. The amount of weight gained after age 18 was a strong predictor of future cardiovascular events. A weight gain of more than 5 kg was associated with a significantly increased risk of heart disease later in life (Figure 2.6).

![Relative Risk of Developing Coronary Heart Disease by Weight Gain](image)

**Figure 2.6.** Relative risk of developing coronary heart disease by the amount of weight gained after age 18. For the relative risk calculation, women whose weight remained stable (±5 kg) throughout adulthood were used as a referent group. Data were adjusted for age, smoking, menopausal status, parental history of heart disease, and postmenopausal hormonal therapy. Figure prepared from data reported by Willett et al. (1995).

Excess body weight and diabetes have a graded relationship: the greater the degree of overweight, the higher the risk of diabetes. In one study, the prevalence of diabetes was 2.4 percent for normal weight women, 7.2 percent for women having a BMI between 25 and 34.9, and 13.2-19.9 percent for women with a BMI of 35 and above (Must et al., 1999). Among the NHANES III participants diagnosed with diabetes, 21 percent had a BMI between 27 and 29.9 and 46 percent had a BMI greater than 30 (National Task Force, 2000). Ford et al. (1997) and Mokdad et al. (2000) estimated that in adults the risk of developing diabetes increases 4.5 to 9 percent for every kilogram gained.
As in diabetes, risk of developing hypertension rises with the degree of adiposity. In a study of 14,676 adults, the prevalence of hypertension was 23.5, 34.2, 49, and 65 percent for normal weight, overweight, obese (BMI 30-34.9), and very obese (BMI ≥ 35) men, respectively (Must et al., 1999). In the same study a similar pattern was observed for women. In the Iowa Women's Health Study, the risk of having diabetes, hypertension, or a heart attack was significantly associated with the amount of weight gained after 18 years of age and was the highest for women who gained 20 percent or more (French et al., 1996; Figure 2.7).

![Figure 2.7](image_url)

**Figure 2.7.** Relative risk of heart attack, high blood pressure (HBP), and diabetes in weight gainers. Women who gained ≥20% since age 18 were compared to a referent group of women whose weight remained within ±5% of weight at age 18. All women were within a normal range for body weight at age 18. Data were adjusted for baseline weight, smoking, and education level. Figure prepared from data reported by French et al. (1996).

Obesity increases risk of premature death from all causes and related co-morbidities (Allison et al., 1999; Solomon & Manson, 1997). In the Nurses' Health Study, non-smoking women with a BMI of 32 and higher had a 2-fold increased risk of all-cause mortality compared with women having a BMI of 22 and lower (Manson et al., 1995). With respect to specific diseases, women with a BMI greater than 29 were 4.6-5.8 times more likely to die of coronary heart disease and twice as likely to die of colon, breast, and endometrial cancers than were women with a BMI of 22 and lower. In addition, the relative risk of dying from heart disease was 2.6 times greater for women who gained 10-19 kg and 7.4 times greater for
women who gained more than 20 kg after age 18 than it was for women whose weight remained stable (±4 kg) or whose weight gain did not exceed 10 kg. Thus, even a relatively modest weight gain of 10 kg in young adulthood significantly increases the risk of cardiovascular mortality in middle age. Similar associations between weight status and mortality have been reported for men. The lowest rate of cardiovascular mortality was observed in men with a BMI less than 27.5, and whose weight remained within four percent of that at age 20 (Rosengren et al., 1999). Weight gains of 4-10 percent and greater than 35 percent after age 20 were associated with 1.57-fold and 2.76-fold increases in risk of fatal coronary disease, respectively. A recent study of 1,046,154 adults reinforced the relationship between obesity and increased mortality by reporting the lowest rates of death from all causes for non-smoking men and women with respective BMI of 23.5-24.9 and 22-23.4 (Calle et al., 1999). In summary, the lowest morbidity and mortality rates for both men and women are associated with a BMI below 25 and either weight stability or a weight gain not exceeding 4.5 kg during adult life (Solomon & Manson, 1997).

In 1995 the estimated direct cost of obesity was 5.7 percent, or $52 billion of the annual national health care expenditure (Wolf & Colditz, 1998). The economic burden of obesity is estimated to be 9.4 percent when indirect expenditures, such as costs of lost productivity and work disability attributable to obesity, are taken into account (Colditz, 1999). Sturm (2002) reported that treatment of obesity costs more than that of smoking and problem drinking, contributing to a 36 percent increase in inpatient and outpatient spending and a 77 percent increase in medication use compared to those of normal weight.

**Obesity Treatment: Success and Limitations**

Although genetic factors account for 20-50 percent of the variance in obesity among individuals (Bouchard & Perusse, 1993; Bouchard & Perusse, 1996), experts generally agree that the marked increase in the prevalence of obesity in all demographic, geographic, and socio-economic groups during the last two decades was not caused by changes in the population’s genetic profile (Kumanyika, 2001; Willett, 1998; Mokdad et al., 1999). On the other hand, a considerable amount of evidence has accumulated that overeating and a sedentary lifestyle are leading modifiable factors contributing to excess weight gain in many
individuals (Lewis et al., 1997; Romieu et al., 1988). Therefore, obesity interventions commonly use various combinations of energy restriction, exercise, and other behavior modifications. Treatment options also include pharmacological therapy and, for severely obese (BMI > 40) persons, surgery (Glazer, 2001).

**Dietary interventions**

Scientific literature provides abundant examples of well-designed treatment programs that have resulted in large weight losses in overweight patients; however, even the most effective weight loss interventions rarely produced long-lasting effects. Wadden (1993) summarized findings of several randomized trials that used low-calorie diets and behavior modifications to treat obesity. He reported that, on average, patients consuming 1,200 kcal/d were able to lose 8.5 kg in four to five months and those consuming 400 to 800 kcal/d were able to lose 20 kg or more in three months. The majority of the patients regained 30 to 50 percent of the lost weight within one year and almost all the weight within five years. The findings reported by Wadden have been confirmed by other studies (Table 2.2).

### Table 2.2. Weight maintenance after a weight-loss program.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subjects, mean age</th>
<th>Entry mean weight, kg (BMI)</th>
<th>Mean weight loss, kg</th>
<th>Follow-up duration, years</th>
<th>Weight loss regained, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anderson et al., 1999</td>
<td>112 F,M¹</td>
<td>108 (37.3)</td>
<td>29.7</td>
<td>3</td>
<td>73.4</td>
</tr>
<tr>
<td></td>
<td>46 yr.</td>
<td></td>
<td></td>
<td>5</td>
<td>77.2</td>
</tr>
<tr>
<td>Pasman et al., 1999</td>
<td>67 F</td>
<td>87 (32.1)</td>
<td>9.7</td>
<td>1.2</td>
<td>72.2</td>
</tr>
<tr>
<td></td>
<td>37.9 yr.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grodstein et al., 1996</td>
<td>192 F.M</td>
<td>105 (37.6)</td>
<td>22.0</td>
<td>3</td>
<td>85.0</td>
</tr>
<tr>
<td></td>
<td>47.6 yr.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walsh &amp; Flynn, 1995</td>
<td>143 F.M</td>
<td>107.7 (37.7)</td>
<td>21.4</td>
<td>4.5</td>
<td>87.6</td>
</tr>
<tr>
<td></td>
<td>46.5 yr.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hensrud et al., 1994</td>
<td>24 F</td>
<td>73.7 (27.0)</td>
<td>12.5</td>
<td>1</td>
<td>42.0</td>
</tr>
<tr>
<td></td>
<td>59 yr.</td>
<td></td>
<td></td>
<td>4</td>
<td>87.0</td>
</tr>
</tbody>
</table>

¹F, female; M, male.
Typically 75 to 85 percent of lost weight is regained within three to five years following participation in a treatment program. About 25 percent of patients are able to maintain a weight loss of at least 10 percent, which is associated with significant health benefits, and, among this 25 percent, only 12-14 percent are able to maintain a weight loss of greater than 75 percent for more than three years (Grodstein et al., 1996; Walsh & Flynn, 1995). Latner et al. (2000) proposed that the problem of poor weight maintenance might be solved through a life-long participation in a weight-loss intervention as offered, for instance, by the ongoing Trevose Behavior Modification Program. This program uses common dietary and behavioral treatment methods. In contrast to other interventions, participants are allowed to remain in the program as long as they wish providing that they comply with the program’s somewhat strict participation rules (Latner et al., 2000). The program success is remarkable. The follow-up assessment showed that individuals who remained in the program for two years were on average 19.3 percent (17.9 kg) below their initial body weight, and those who remained in the program for five years were 17.3 percent (15.7 kg) below their initial weight. Unfortunately, a major drawback of this program and obesity treatment interventions in general, is a high dropout rate. In the case of the Trevose program, out of 202 initially enrolled participants, 85 percent completed the five-week core intervention, 52 percent remained in the program for one year, 23 percent remained for three years, and only 18 percent completed five years of treatment. Even short-lasting interventions have low retention rates. In one 12-week program that used a very-low calorie diet and education about weight management, less than 50 percent of patients completed the entire course of treatment (Anderson et al., 1999).

Pharmacotherapy

At the present time obesity drugs are prescribed to individuals having a BMI greater than 27 with obesity-related co-morbidities, or a BMI greater than 30 without co-morbidities in accordance with the guidelines established by the Food and Drug Administration (FDA). It has been estimated that between 1996 and 1998 approximately 2.5 percent, or 4.6 million of U.S. adults used prescription drugs for weight control (Khan et al., 2001). Presently available drugs induce weight loss by different mechanisms. Anorectic drugs (phentermine, mazindol, diethylpropion hydrochloride, fenflurmine, dexfenfluramine, and fluoxetine
hydrochloride) reduce energy intake by suppressing hunger, appetite, and cravings for fat and carbohydrate. These drugs act on neurotransmitters -- serotonin and norepinephrine -- that are involved in the regulation of energy balance. Another drug, Orlistat\textsuperscript{\textregistered}, decreases absorption of dietary fat by acting on intestinal lipoprotein lipase, an enzyme that is involved in fat digestion (Glazer, 2001). Short-term treatments (up to one year) with obesity drugs approved by the FDA appear to be safe; however, data are insufficient to determine whether obesity drugs are effective and safe for long-term use that might be necessary for individuals with chronic obesity (Atkinson, 1997).

Drug therapy is usually complementary to dietary and behavior modifications (Goldstein & Potvin, 1994; Atkinson, 1997). An average weight loss achievable with obesity drugs in excess of placebo ranges from 1.5 percent (1.5 kg) to 8.1 percent (7.9 kg) over a period of eight to 12 months (Goldstein & Potvin, 1994; Glazer, 2001). The overall effect of obesity drugs is characterized as modest but is associated with physiologically significant health benefits, particularly when pharmacotherapy is combined with other treatments (Glazer, 2001; Atkinson, 1997). Limitations of drug therapy include diminishing responsiveness to a drug over time (i.e., reaching a weight plateau), abuse potential, and side effects ranging from intestinal discomfort to decreased mental acuity and pulmonary hypertension (Goldstein & Potvin, 1994; Atkinson, 1997; Glazer, 2001). Moreover, obesity drugs are only effective while taken. Discontinuation of drug treatment causes patients to regain the lost weight (Bray, 1996).

Drug therapy is unlikely to solve the obesity problem at the population level in the near future. The use of obesity drugs requires medical supervision because long-term benefits and adverse effects of obesity drugs are not well established (NIH Technology Assessment Conference Panel, 1993). Furthermore, drugs are expensive. It has been estimated that the cost of one-kg weight loss ranges between $91 and $433, depending on the type and dose of the drug prescribed (Glazer, 2001). It is unlikely that certain population groups, for instance, young and low-income individuals, would be able to afford drug therapy. In recent publications, scientists expressed optimism about new experimental drugs that promise to be effective and safe. However, it will take some time before these drugs are
properly tested for their long-term efficacy and safety and become readily available and affordable to the general public (Atkinson, 1997; Bray & Greenway, 1999).

In summary, common obesity treatment options, including dietary and behavior modifications and drugs, can produce statistically and physiologically significant weight losses. Unfortunately, these losses are sustained only by a small percentage of individuals. Many patients have difficulty adhering to intensive dietary regimens for a long period of time and, upon completion of a treatment, gradually revert to old habits or follow less strict guidelines that eventually lead to weight regain. Pharmacotherapy is currently restricted to patients with clinically significant obesity for whom behavioral changes alone are insufficient to produce a weight loss associated with health benefits. The effect of obesity drugs is generally modest. High costs may limit the availability of obesity treatments to the general population.

**Obesity Prevention: Success and Limitations**

In view of obesity treatment limitations, the importance of obesity prevention has been increasingly emphasized in the scientific and medical literature during the last quarter of the 20th century (Crawford et al., 2000; Bouchard, 1996; National Task Force on Prevention and Treatment of Obesity, 1994). Many overweight individuals could avoid the stress and cost associated with obesity treatment if they knew how to prevent excess weight gain. Even more importantly, obesity is a chronic disease and, as some experts believe, is incurable at the present time (Bray & Greenway, 1999). Long-term obesity may cause adverse metabolic and physiological changes that cannot be reversed even with weight loss. These changes could be avoided, however, through prevention of weight gain (Kumanyika, 2001; Ruderman et al., 1998; Bouchard, 1996; Jeffery, 1993).

Although emphasis on obesity prevention has increased, a surprisingly small number of studies have attempted weight gain prevention in the adult population (Table 2.3). These community-based studies used low-intensity education to improve nutrition knowledge of the target audience. It was assumed that improved knowledge would influence nutrition-related attitudes and behaviors. It was also expected that changes in dietary behaviors would have a favorable effect on body weight. Information about obesity-associated health risks and
weight-control strategies was disseminated through mass media channels, printed materials, classes, seminars, and workshops. Self-reported or measured changes in weight following the intervention were used to assess intervention effectiveness. Collectively, these studies demonstrated that low-intensity education increased obesity awareness and produced improvements in health habits, but had small and inconsistent effects on weight.

Table 2.3. Weight changes in response to low-intensity education: results of community-based interventions.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Subjects. age</th>
<th>Intervention groups</th>
<th>Follow-up, years</th>
<th>Weight change, kg</th>
<th>Effect, P≤</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miles et al., 2001</td>
<td>2121 F.M† all ages</td>
<td>One group</td>
<td>0.5</td>
<td>-4.2</td>
<td>.001b</td>
</tr>
<tr>
<td>Jeffery &amp; French, 1999</td>
<td>1226 F.M 20-45 yr.</td>
<td>Education Education &amp; incentive No contact control</td>
<td>3</td>
<td>+1.6 +1.5 +1.8</td>
<td>N/S⁵, d</td>
</tr>
<tr>
<td>Taylor et al., 1991</td>
<td>743 F.M 25-74 yr.</td>
<td>Education No contact control</td>
<td>6</td>
<td>+0.57 +1.25</td>
<td>N/S⁵</td>
</tr>
<tr>
<td>Forster et al., 1988</td>
<td>219 F.M 45.9 yr.</td>
<td>Education No contact control</td>
<td>1</td>
<td>-1.0 -0.2</td>
<td>.03c</td>
</tr>
</tbody>
</table>

†F, female; M, male.
 Different from baseline.
 Treatment vs. control.
 N/S, no significant difference.
 Mean age, range was not reported.

Two interventions – “The Pound of Prevention” program (Forster et al., 1988) and the British Broadcasting Corporation’s (BBC) “Fighting Fat, Fighting Fit” campaign – reported a significant effect of the intervention on weight (Miles et al., 2001). However, both had design limitations that might have influenced their outcomes. Forster et al. (1988) reported that their participants expressed a greater than average interest in losing weight, which could have contributed to the significant difference in weight between the intervention and control groups at the end of the program. Indeed, in a later trial of the “Pound of Prevention” program, no difference in weight change was found between two treatment groups (Jeffery et
al., 1997; Jeffery & French, 1999). Similarly, in the Stanford Five-City Project the educational approach did not prevent weight gain in the intervention group (Taylor et al., 1991). The BBC’s mass-media campaign appeared to be effective; however, it was limited by self-reported changes in weight, absence of a control group, and a short follow-up (Miles et al., 2001).

The disappointing results of the “Pound of Prevention” and Stanford Five-City programs prompted some researchers to conclude that education as a strategy to prevent obesity may be inherently ineffective or insufficient when used alone (Jeffery, 1995). Interestingly enough, this conclusion has been widely accepted by health professionals despite the fact that the community education interventions were pioneering studies in the field of obesity prevention and, as such, had a number of serious weaknesses (Wardle et al., 2000; Hardeman et al., 2000). First, none of these studies reported evaluating the nutrition information for quality. In other words, it was not determined whether the study participants found information presented to them understandable and applicable in their circumstances. Second, participants’ knowledge before and after intervention was not assessed. Thus, there were no data to indicate whether a change in knowledge occurred. Third, the intervention designs did not include theoretical frameworks to facilitate behavioral changes even though the science of health education has long emphasized the importance of using communication, behavioral, and learning models in interventions that are aimed at influencing behavior (Gillespie, 1984; Gillbert & Sawyer, 1995; American Dietetic Association, 1990).

Evidence of the Nutrition Knowledge-Behavior Association

Although community education interventions were weak and inconsistent in their effect on weight, they produced significant improvements in specific dietary behaviors, such as an increase in fruit and vegetable consumption and a decrease in the intakes of fried foods, fatty meats, sweets, butter, and whole milk. The improvements in food choices suggest that nutrition education does have an effect on dietary behavior. Recent observational studies reinforce the role of nutrition knowledge in adapting healthful dietary practices. Wardle et al. (2000) administered a knowledge test and a food frequency questionnaire to 1,040 adult men and women. The authors reported that knowledge scores were significantly correlated
with intakes of fruits, vegetables, and fat. Subjects in the highest knowledge quintile were almost 25 times more likely to consume a healthful diet (i.e., high in fruits and vegetables and moderate in fat) than were subjects in the lowest quintile. The proportion of participants consuming at least five servings of fruit and vegetables a day was 52 percent in the highest quintile and only 11 percent in the lowest quintile. The associations between nutrition knowledge and dietary patterns were independent of socioeconomic factors, such as level of education and occupation. The authors did not report whether there was a difference in body weight between the two knowledge groups. In a similarly designed study, Dallongeville et al. (2001) examined associations between nutrition knowledge and intakes of specific foods, alcoholic beverages, and dietary fat in 361 middle-aged French men. The results showed that men in the highest knowledge tertile were significantly more likely to consume foods such as olive oil, cheese, and cereals and less likely to consume sunflower oil, fatty meat, dry vegetables (e.g., tomato), and beer than were men in the lowest knowledge tertile. Also, intakes of total and saturated fat as a proportion of daily energy intake were significantly lower, although still above recommended, among men in the highest knowledge tertile compared with men in the lowest tertile. Despite the differences in food choices and fat intake, the two knowledge groups were consuming similar amounts of total daily calories. Thus, it was not surprising that both groups had a mean BMI of 27. Marietta et al. (1999) examined the knowledge-behavior association in 208 undergraduate students. The knowledge of food labels was a positive and significant predictor of attitudes toward labels and use of labels when purchasing food. Overall, available evidence suggests that nutrition knowledge is associated with healthier food choices; however, the adoption of healthier dietary habits does not always translate into measurable outcomes, such as changes in weight. A major challenge of future interventions to prevent obesity is to identify educational strategies that are potent enough to influence nutrition behaviors to the extent that a desirable effect on weight is achieved.

Nutrition Education is a Cornerstone of Prevention Interventions

Obesity prevention is a relatively new area of research for which a strong theoretical foundation has yet to be developed (Shannon, 1990). However, education is a fundamental
strategy of any health promotion and disease prevention intervention: therefore, some concepts and models of general nutrition education apply to obesity prevention.

Nutrition education: definition, goals, and research directions

Nutrition education is defined as any set of learning experiences that facilitate the voluntary adoption of eating and other nutrition-related behaviors conducive to health and well-being (American Dietetic Association, 1996). Thus, the ultimate goal of nutrition education is to influence behavior by enabling individuals to make informed decisions about their own diets (Gillespie, 1987; Parham 1993). Nutrition education is concerned with many issues including learning domains (knowledge, attitude, and behavior) (Johnson & Johnson, 1985), communication and learning theories explaining the process of information delivery and individuals’ responses to nutrition intervention programs (Gillespie & Yarbrough, 1984), determinants of dietary behavior (Shannon, 1990), and behavioral models for understanding the dietary change process (American Dietetic Association, 1996).

Fundamentals of nutrition education

Because nutrition education is aimed at behavior change, it is much more complex than a mere transmission of information from an educator to an individual (i.e., receiver). Gillespie (1984) developed the nutrition communication model that includes the following components: a) inputs of an educator and a receiver, b) an intervening process that involves both the receiver’s attention and comprehension of information and an interaction between an educator and a receiver, and c) outcomes or responses associated with acceptance or rejection of information by a receiver, which could occur at the cognitive, affective, or behavioral level. Furthermore, each component of the model has a number of subcomponents. For example, the educator input consists of channels of communication, sources of information, and message content and presentation, whereas the receiver input consists of one’s previous skills, beliefs, attitudes, experiences, and habits that could influence one’s response to new information. Collectively, the model components determine how well a nutrition message is understood, accepted, and implemented by an individual (Gillespie, 1984; Gillespie & Yarbrough, 1984). The relationship between nutrition education and behavior is further complicated by multiple behavior determinants that influence individual responses to nutrition information. These determinants can be internal (physiological and psychological),
such as: age, taste preferences, personality characteristics, and emotional state; and external (environmental), such as: cultural expectations, advertising appeals, food availability and cost, peer influence, and many others (Shannon, 1990; Guthrie, 1994). Finally, the effect of nutrition education on behavior can only be established when appropriate instruments to measure nutrition knowledge pertinent to the behavior of interest are used. In other words, knowledge tests unrelated or insufficiently sensitive to a behavior in question will fail to detect the education-behavior association even when such association exists (Wardle et al., 2000; Parmenter & Wardle, 2000). It is beyond the scope of this review to address all aspects of nutrition education. The primary focus of the following discussion is on the quality of nutrition information for interventions aimed at prevention of obesity. In Gillespie’s communication model, the information quality concept is defined as the educator and receiver inputs.

**Determinants of the information quality**

In spite of their limitations, community education studies were a step toward more effective interventions to combat obesity. These studies identified a number of factors influencing intervention effectiveness. Jeffery and French (1999) pointed out that participants of the “Pound of Prevention” program seemed to have a good general awareness of obesity as a health problem and of basic weight control techniques. They suggested that future interventions may need to include more specific and complex weight-related messages. Miles et al. (2001) and Jeffery and French (1997) noted that different approaches might be required for groups that exhibit low interest and poor participation in the program including men, young adults, persons not overweight but at risk of weight gain, and less educated and low-income individuals. Foster et al. (1998) reported that their intervention worked better for certain subgroups of participants including men, nonsmokers, persons without previous program experience, and those closer to their desirable adult weight. In the Stanford Five-City Project, the intervention had the strongest effect on women between 25 and 34 years of age (Taylor et al., 1991). Collectively, findings of the community education studies suggest that various population groups can be affected differently by the same intervention. Therefore, future prevention programs should target groups of individuals with similar demographic, socioeconomic, and psychological characteristics rather than the whole
population (Parham, 1993). The rationale for using the group approach is that individuals within each group are likely to have similar experience, opinions, and habits that determine specific information needs of these individuals (Parham, 1993; Gillespie, 1987). Mullen et al. (1992) and Kok et al. (1997) pointed out that one of the most important factors explaining the effectiveness of education-based interventions is the relevance of health messages to the needs of a target audience. They defined relevance as "the tailoring of the program to knowledge, beliefs, circumstances, and prior experience of the learner, as assessed by pretesting or other means" (Kok et al., 1997, p. 24).

In summary, three major factors that are associated with the information quality have been identified. First is a target audience: the more specific the target audience, the easier it is to orient educational messages to it (Achterberg, 1994; Gillespie, 1987). Second is the receiver input or specific attitudes, beliefs, knowledge, and practices of the target population. Third is the educator input or decisions that an educator makes about message content, complexity, and treatment (Gillespie & Yarbrough, 1984). The application of these factors to designing a nutrition education intervention is illustrated in the following discussion.

**Selecting a target audience for primary prevention of obesity**

While the prevalence of overweight peaks after the age of 35, many adults experience major weight gain between the ages 25 and 34 (Jeffery & French, 1997; Burke et al., 1996; Williamson et al., 1990). Thus, individuals in their late teens and early twenties would benefit from education-based interventions to prevent excessive weight gain in adulthood.

**Receiver input**

The first step in designing interventions on life-long weight management for young adults is to learn about weight-related knowledge, attitudes, and practices of this population group (Gillespie, 1987). Some of these attitudes and practices have already been determined. A general consensus is that men and particularly women in their early twenties are excessively concerned with body weight and shape, often for appearance reasons (Bailey & Goldberg, 1989; Drewnowski & Yee, 1987; Haberman & Luffey, 1998; Ritchie, 1998; Sciacca et al., 1991; McArthur & Haward, 2001; Rodin, 1993). The results of several published studies showed that the vast majority of college-age women wish to lose weight. This includes women who are not overweight based on their BMIs (Bailey & Goldberg,
Young men are not as preoccupied with thinness as women of the same age group; however, they are not indifferent about their physical appearance either. Drewnowski and Yee (1987) surveyed 226 18 year-old women and men. The researchers reported that 85 percent of women and 40 percent of men wished to lose weight and 45 percent of men wished to gain weight. Both men and women who wished to lose weight had negative body perceptions and viewed themselves as overweight.

Dissatisfaction with physical appearance contributes to a high prevalence of weight loss attempts among young individuals who take various approaches to attain a desirable physique. Weight control methods most frequently used by this population group can be considered reasonable and include exercise and energy restriction (Serdula et al. 1999; Page & Fox, 1998). However, at any one time only 20 percent of 18-29 year-old men and women use both exercise and energy restriction simultaneously, whereas the majority use one method or the other (Serdula et al., 1999). Young adults also resort to questionable weight control practices. Blanck et al. (2001) found that the prevalence of using nonprescription weight loss products among 18-34 year-old adults was 17.3 percent for women and 3.5 percent for men.

Young adults' attitudes toward weight and weight control practices have been studied extensively; however, specific knowledge of this population group regarding weight management has not been described. Research findings for adolescents discussed below generally show poor understanding of concepts pertinent to weight control. It is important to keep in mind, however, that findings for adolescents may have limited application to young adults. Although it has not been shown, it could be expected that 20-25 year-olds would know more about nutrition and weight control than would 13-16 year-olds.

Kilpatrick et al. (1999) reported that learning about problems of being overweight or underweight had a greater impact on weight change efforts among adolescents in grades 7th through 12th than did learning about the importance of exercise and diet. Mitchell (1990) found that college students had misconceptions about energy density of dietary fat and carbohydrate even after completing a basic nutrition course. Likewise, Searles et al. (1986) reported that 57 percent of surveyed high-school students believed that dietary carbohydrate
and protein were more energy dense than fat. In the same study, only 27 percent of students knew that body fat is essential for health, and less than 22 percent knew the number of calories needed to be expended to lose one pound of fat. Skinner and Woodburn (1984) surveyed 1,193 adolescents on the basics of weight control. Eighty-three percent of the adolescents recognized that reducing daily energy intake is an effective weight loss strategy; however, when asked to choose from a list of foods and meals those that were low in calories, they did poorly.

**Educator input**

Once knowledge, attitudes, and practices of the intended audience have been determined, an educator makes decisions about the intervention itself. In other words, an educator determines the content, complexity, and treatment of information to be presented to the audience (Gillespie & Yarbrough, 1984). Because the long-term objective of nutrition education is to influence behavior, the information has to be pertinent to the behavior of interest (Achterberg, 1994). Gillespie (1987) emphasized the importance of distinguishing between information that the intended audience perceives as important for making desired changes, and information that is important to an educator in his/her work. Another factor to consider is adequate exposure of the intended audience to the information (Kilpatrick et al., 1999). Presentation of the same messages several times in various contents is required for an individual to make a transition from awareness of a certain practice to adoption of the practice (Gillespie & Yarbrough, 1984). Finally, an educator should realize that changes in behavior do not occur immediately. Initially, individuals are very resistant to change because it may contradict their present behavior. Thus, an educator should provide individuals with ample time for information processing and making a desired change (Gillespie & Yarbrough, 1984).

Nutrition messages should contain enough information for making sound decisions and be adequate in complexity (Gillespie & Yarbrough, 1984). Prior education of an individual must be taken into account (Johnson & Johnson, 1985). Although simple concepts are easy to communicate, Gillespie (1987) cautioned against underestimating the amount and depth of information the audience may want. Messages that are too simple may not be helpful in making changes. On the other hand, if an educator believes that complex concepts
would be appropriate for the intended audience, she/he is responsible for finding a way to present these concepts in an understandable manner. One advantage of presenting complex concepts rather than a number of simple facts is that complex information stimulates development of critical thinking skills that have applicability beyond a single message (Tsui, 1999).

The final component of the educator input is message treatment, which includes the message format and channel of delivery. Message treatment largely depends on the educator’s objectives and creativity, available resources, and characteristics of the intended audience. An educator can use various channels for message delivery including printed materials, lectures, or the mass media (Gillespie & Yarbrough, 1984).

Research Directions
Adults between the ages of 18 and 30 are a primary target audience for education-based interventions to prevent obesity. There is a need for objective data on young adults’ knowledge, attitudes, and behavior regarding weight management. Research is also needed to examine the effectiveness of different types of nutrition education messages and instructional techniques for weight gain prevention in this population group.

References


CHAPTER 3: A SINGLE DAILY DOSE OF SOYBEAN PLANT STEROLS IN GROUND BEEF DECREASES SERUM TOTAL AND LOW DENSITY LIPOPROTEIN CHOLESTEROL IN YOUNG, MILDLY HYPERCHOLESTEROLEMIC MEN

A paper published in the American Journal of Clinical Nutrition

Oksana A. Matvienko, Douglas S. Lewis, Mike Swanson, Beth Arndt, David L. Rainwater, Jeanne Stewart, D. Lee Alekel

Abstract

Consumption of phytosterol-supplemented margarine lowers plasma total cholesterol (TC) and LDL cholesterol (LDL-C) concentrations in older middle-aged hypercholesterolemic individuals. The effects of incorporating phytosterols into lower fat foods on plasma lipids of young men at increased risk of developing cardiovascular disease (CVD) have not been studied. We tested the hypothesis that a single daily dose of soybean phytosterols added to ground beef will lower plasma TC and LDL-C concentrations in mildly hypercholesterolemic young men. In a triple-blind 4-wk study, 34 male college students with elevated plasma TC (5.85 ± 0.70 mmol/L), LDL-C (4.02 ± 0.60 mmol/L), and TC:HDL-C ratio (5.5 ± 1.2) were randomly assigned to the control (ground beef alone) or treatment (ground beef with 2.7 g of phytosterols) groups. The phytosterol mixture was two-thirds esterified and one-third non-esterified and consisted of β-sitosterol (48%), campesterol (27%), and stigmasterol (21%). Consumption of phytosterol-supplemented ground beef lowered plasma TC, LDL-C, and the TC:HDL-C by 9%, 15% and 10%, respectively, compared with the control group (P < 0.001). The LDL particle size did not change, suggesting that the decrease was primarily of particle number. The decreases were similar in

2 Graduate student and primary author. Department of Food Science and Human Nutrition, Iowa State University, Ames.
3 Associate Professor and author for correspondence. Department of Food Science and Human Nutrition, Iowa State University, Ames.
4 ConAgra Foods, Inc., Omaha NE.
5 Department of Genetics, Southwest Foundation for Biomedical Research, San Antonio TX.
6 Department of Food Science and Human Nutrition, Iowa State University, Ames.
subjects with \((n = 8)\) and without \((n = 9)\) a family history of premature CVD in the treatment group. Phytosterol-supplemented ground beef effectively lowers plasma TC and LDL-C and has the potential to become a functional food to help reduce the risk of CVD.

**Introduction**

Atherosclerosis begins in adolescence \((1, 2)\) as fatty streak lipid deposits in the arterial wall. High concentrations of plasma total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) accelerate atherogenesis in the teenage years, with their effects amplified in young adulthood, 20 to 30 years before coronary artery disease (CAD) becomes clinically manifest \((3)\). By 30 to 34 years of age, approximately 19% of men have advanced lesions in the left anterior descending coronary arteries \((4)\). The long-term significance of the early etiology of atherosclerosis is apparent from the observations of Klug et al. \((5)\). They showed that 22 year-old men with plasma TC >5.40 mmol/L were 5.6 times more likely to develop CAD, 6.0 times more likely to have a heart attack, and 9.6 times more likely to die during the next 40 years than those with plasma TC <4.45 mmol/L. More recently, Stamler et al. \((6)\) reported that young men with plasma TC concentrations >5.15 mmol/L had greater relative mortality risk during 16 to 25 years of follow-up than did men with plasma TC <5.15 mmol/L. The National Cholesterol Education Program Adult Treatment Panel recommended measuring blood cholesterol levels in all adults 20 years of age and older \((7)\).

Although there is controversy about using hypocholesterolemic drugs for primary prevention of atherosclerosis in young adults \((7, 8)\), dietary intervention to lower plasma TC and LDL-C concentrations remains a sensible and well-accepted preventive approach to decreasing cardiovascular disease (CVD) morbidity and mortality. Despite the well-documented rationale for medical nutrition therapy, many eligible patients with borderline \((>3.35 \text{ mmol/L})\) or high \((>4.10 \text{ mmol/L})\) LDL-C concentrations are not routinely referred for dietary treatment, and among those who are, compliance may be limited \((9)\). Indeed, cholesterol-lowering diets are usually low in total fat, saturated fat, and cholesterol and thus may not be sufficiently palatable to induce long-term adherence necessary for health benefits \((10, 11)\).
Phytosterols are in the forefront of nutraceutical research on the development of food products lowering plasma cholesterol concentrations. The discovery of the hypocholesterolemic effect of phytosterols led to the marketing of foods such as margarine and cooking or salad oils that are high in fat and of low nutritional value. Consumption of high fat versions of these food products can contribute 22-45% of the recommend daily fat intake (67 g) for an individual consuming a 8360 kJ (2000 kcal) diet. Clearly, adding phytosterols to a variety of foods that are lower in fat and higher in nutritional value would provide a healthier diet with cardiovascular benefits. We tested the hypothesis that soybean phytosterols added to lean ground beef as part of a daily lunch would significantly lower plasma TC and LDL-C concentrations in young, mildly hypercholesterolemic men.

Subjects and Methods

Subjects
The Iowa State University Human Subjects Review Committee approved the study protocol. The subjects were recruited from the undergraduate and graduate student body at Iowa State University with newspaper advertisements and posted flyers. One hundred eighty-one men volunteered to participate in the study and were screened for blood lipids and health-related behavioral characteristics. Thirty-six white men met the selection criteria, which included plasma TC concentration >5.10 mmol/L and LDL-C concentration >3.35 mmol/L. Each subject agreed to consume lunch Monday through Friday in the Human Metabolic Unit for 28 days. The exclusion criteria were medical history of diabetes mellitus or CAD, smoking and use of alcohol or drugs, or taking any type of medication affecting blood lipids.

The subjects were randomly assigned to either a treatment (phytosterol-supplemented ground beef; n = 18) or a control (ground beef only; n = 18) group. Thirty-four subjects completed the study. One subject dropped out due to personal reasons, and one subject was excluded for failing to show up for lunch. The subjects gave written informed consent before the study (Appendix A). Each subject received a $250 stipend upon completion of the study. Six subjects who were enrolled in a university meal plan were also reimbursed for missed meals.
Study design

We used a randomized triple-blind design to evaluate the subjects' plasma lipid and lipoprotein responses to the consumption of control and treatment ground beef lunches. ConAgra Foods, Inc. provided the control and treatment ground beef packaged in 2 differently colored wrappers. Only scientists at ConAgra Foods, Inc. knew the color code: neither the investigators nor the subjects knew which color corresponded to which type of ground beef. The color code was changed to a number code (group 1 and group 2) by the investigators; the scientists at ConAgra Foods, Inc. did not know which data belonged to which group. The codes were broken simultaneously after the plasma lipoprotein data were analyzed and summarized. Sensory tests conducted in January 2000 with 31 men of a similar age showed that the subjects could not tell the difference between the types of ground beef.

The soybean extracted phytosterol mix was obtained from Henkel Corporation (Unilever Research Laboratories, Vlaardingen, The Netherlands). The phytosterol mix contained 67% sterol esters and 33% free sterols. The sterol composition was 48% β-sitosterol, 27% campesterol, 21% stigmasterol, and 5.9% other sterols, according to a report from the Unilever Research Laboratories. The mixture also contained 4.1% free fatty acids including 61% linoleic, 23% oleic, 3.5% stearic, and 7.7% palmitic acids. The frozen phytosterols were thoroughly mixed in a blender with a small amount of boneless minced beef that was 85% lean and 15% fat by weight. This mixture was added to a large amount of boneless minced beef that was 90% lean and 10% fat by weight. After grinding, 2-5 kg of the beef blend was stuffed into color-coded plastic tubes and clipped on both ends. The raw ground beef was cooked at 177°C until the internal temperature reached 74°C. There was no visible loss due to drippings during cooking. The lipid composition of the cooked treatment and control ground beef is presented in Table 3.1.

The subjects were served lunch in the Human Metabolic Unit each weekday for 4 weeks (March 28-April 26, 2000). Each lunch included a fixed portion of cooked ground beef (mean ± SD: 112 ± 2 g). On average, each portion for the treatment group contained 2.7 g phytosterols. The ground beef was served as a hamburger (Monday), a sloppy joe (Tuesday), spaghetti with meat sauce (Wednesday), 2 tacos (Thursday), and a cheeseburger (Friday). For weekends, the subjects received precooked frozen chili and a ground beef-rice
casserole, along with heating instructions. The subjects were given color-coded cards corresponding to the colored wrapping of the hamburger, which they presented to the kitchen personnel at lunch. The subjects consumed the entire ground beef portion of each meal. Compliance was assured by the supervision of the subjects during the lunches. The subjects had ad libitum access to fresh and cooked vegetables, a variety of fruit, chips, salads, pickles, condiments, beverages, and deserts. Compliance for weekend meal consumption was assessed on the last day of the study by the subjects’ anonymous responses to the question “How many weekend meals did you not eat?” Each subject was asked to provide only their group identification, not their individual identification. Overall weekend compliance was 94% with no difference between treatment and control groups. All subjects were instructed to maintain their habitual dietary and physical activity patterns; however, they were requested not to consume red meat other than that provided during the experimental lunch and to limit egg consumption to 2-3 eggs/wk.

**Nutrient intake, health history, and body weight and height**

A trained graduate research assistant using interviewer-administered questionnaires obtained health information at screening and during the first week of the study (Appendix A). This information included personal medical history, current medications, family history of CVD, presence of high blood lipid concentrations or diabetes mellitus, weekly alcohol consumption, and previous tobacco use. Baseline nutrient and energy intakes were estimated by a semi-quantitative food frequency questionnaire (13). Physical activity during the previous 7 days was assessed using the Five-City Project physical activity recall, which allowed calculation of daily energy expenditure (total and per kg body weight; 14).

Height was measured twice and averaged. Weight was measured at baseline and on the days of the blood draws at weeks 2 and 4. The subjects wore light clothing and removed shoes for the weight and height measurements. Body mass index (in kg/m²) was calculated for each subject at baseline and at weeks 2 and 4.

**Plasma lipid measurements**

Blood samples were obtained at baseline and weeks 2 and 4 after the subjects had fasted overnight. Blood was drawn into a red-gray mottled-top evacuated tube containing SST Gel and clot activator (yellow separator) and into 2 7-mL purple-top EDTA evacuated
tubes (all obtained from Quest Diagnostics, Ceterboro, NJ). The samples were centrifuged at 1380 X g at room temperature for 15 min within 30-60 min after phlebotomy. The plasma samples were stored at 4°C before cholesterol and lipoprotein cholesterol measurements and at -80°C before lipoprotein subclass and plasma sterol measurements. Sample analyses included lipid and comprehensive metabolic panels performed at the regional facility of Quest Diagnostics (Des Moines, IA), a certified clinical laboratory. Plasma HDL-C was measured after precipitation of apolipoprotein B with dextran sulfate and magnesium chloride. The intraassay coefficient of variation (CV) was <5%. The concentration of LDL-C was calculated according to the Friedewald equation (15).

Separation and quantification of lipoprotein subclasses were performed using nondenaturing polyacrylamide gradient gel electrophoresis as described elsewhere (16). Use of a composite gradient gel allowed simultaneous separation of LDL and HDL particles (17). Cholesterol in gels was stained with Sudan Black B (Sigma Chemical Co, St Louis) and scanned by an LKB-Ultrascan XL laser densitometer with GelScan XL software (version 2.1). Absorbance profiles, representing the size distributions of lipoprotein constituents, were converted to ASCII files with the Gelcon program (Pharmacia-LKB Biotechnology) and analyzed with software developed at the Department of Genetics, Southwest Foundation for Biomedical Research, San Antonio, TX (17). We measured 4 traits representing size distributions of lipoproteins: large and small LDL and large and small HDL. Large LDL was defined as the proportion of LDL absorbance (21-29 nm) on particles ≥25.5 nm and small LDL as the proportion on particles <25.5 nm. Similarly, large and small HDL were defined as the proportion of HDL absorbance (7.2-20 nm) on particles in the size intervals 8.8-12.9 nm and 7.2-8.8 nm, respectively (i.e., separation into HDL2 and HDL3). In addition, we measured LDL peak diameter. All samples were run in duplicate on different gels and the values analyzed represent the average values. For a subset of the samples, the number of LDL particles was determined by nuclear magnetic resonance (NMR, LIPOMED, Raleigh, NC) as described elsewhere (18).

Neutral sterol quantitation

Neutral sterols were extracted from the human plasma samples using the Folch extraction method (19) and subjected to gas chromatography analysis in the Hewlett Packard
6890 gas chromatograph (Palo Alto, CA). Samples were analyzed as trimethylsilyl ether derivatives of the neutral sterols. Peaks were identified by comparison with known standards (Steraloids, Newport, RI; Sigma, St. Louis, MO; USA) and quantified with the use of an internal standard, 5-α-cholestane.

**Statistical analyses**

The effects of the consumption of the treatment ground beef on concentrations of plasma lipids, neutral sterols and metabolic values, lipoprotein particle sizes and distributions, body weight, and BMI were analyzed by repeated-measures analysis of variance (ANOVA) with SPSS/PC + 9.0 (SPSS Inc., Chicago, IL). The treatment (phytosterol-supplemented and control ground beef) was the between-subject factor and the time of measurement (baseline, week 2, and week 4) was the within-subject factor. A significant treatment-by-time interaction indicated that a significant change in a variable of interest (e.g., LDL-C) during the study was caused by the phytosterol supplementation. In some analyses, elevated blood lipids or family history of CVD was used as another between-subject factor. The independent sample *t* test was used for comparison of baseline values between the 2 groups. Whenever there was a significant interaction as determined by the repeated-measures analysis of variance, we used the independent *t* test to examine between-group differences and Bonferroni-adjusted pairwise comparisons to evaluate changes within each group. Significance was set at *α* = 0.05 level, but *P* ≤ 0.1 are also reported to better balance Type I and Type II statistical errors. Data are presented as means ± SDs.

**Results**

**Subject baseline characteristics**

The baseline characteristics of subjects in the treatment and control groups are presented in Table 3.2. There were no significant differences in any measured variable between the 2 groups. The apparently higher mean body weight of the treatment group was largely due to the heaviest subject (128 kg) and the 2 lightest subjects (57 and 63 kg) being randomly assigned to the sterol-supplemented and control groups, respectively. The mean weight for all subjects was in the 75th percentile reported by the third National Health and Nutrition Examination Survey for weight among non-Hispanic white males 20-29 y (20). The
subjects' baseline mean plasma TC and HDL-C concentrations were in the 90th-95th and the 25th-50th percentiles, respectively, for non-Hispanic white males 20-29 years of age (21, 22). Eleven (32%) of the 34 who completed the study knew they had high blood cholesterol before the study, 17 (50%) reported that they had a family history of premature CVD and high blood lipids, and 5 (15%) reported a family history of diabetes.

The subjects' mean energy intake was 13249 ± 2564 kJ. Protein, carbohydrate, and fat intakes were 15.6 ± 2.5%, 49.1 ± 6.4%, and 35.6 ± 5.8% of total energy intake, respectively. These values were similar to those reported for non-Hispanic white males 20-29 y: 13062 kJ, 14%, 47.3%, and 34.4% for energy, protein, carbohydrate, and fat, respectively (23). Mean dietary cholesterol intake was 328 ± 95 mg for our subjects and 364 mg for non-Hispanic white males aged 20-29 y.

### Plasma lipid concentrations

At week 4, TC and LDL-C concentrations and TC:HDL-C had decreased 9.3%, 14.6%, and 9.1%, respectively (P < 0.001), from baseline in subjects consuming 2.7 g soybean phytosterols/d, whereas subjects in the control group had no significant changes (Table 3.3). In the treatment group, 82% of the decrease in TC, 75% of the decrease in LDL-C, and 80% of the decrease in the TC/HDL-C ratio occurred after only 2 weeks of feeding. Consumption of the treatment ground beef did not influence plasma HDL-C or triacylglycerol concentrations. The correlation between phytosterol intake expressed per kilogram of body weight and the magnitude of the decline in plasma LDL-C was not significant (Figure 3.1).

### Lipoprotein subclass patterns

Neither the proportion of absorbance on large and small LDL nor the LDL particle diameter was significantly influenced by treatment (Table 3.4). Moreover, no subject changed from LDL pattern A (mean peak diameter ≥ 25.5 nm) to pattern B (mean peak diameter < 25.5 nm) or vice versa in the treatment group. NMR analysis of the week 4 plasma samples showed that the mean LDL particle number tended to be lower (P = 0.058) in the treatment than in the control group (1467 ± 212 compared with 1696 ± 424 nmol/L). The LDL particle diameter measured by NMR (n = 34) was strongly correlated (r = 0.784, P ≤ 0.0001) with the LDL particle diameter determined by gradient gel electrophoresis.
Correlations between changes in small and large LDL particle distribution and changes in plasma concentrations of phytosterols were not significant.

The proportion of large HDL\(_{2a+2b}\) increased after 4 wk in both the treatment and control groups combined (\(P = 0.007\), time effect) (Table 3.4). Pairwise comparisons showed that the increase was significant for the treatment (\(P = 0.042\)) but not for the control group. However, there was no significant treatment-by-time interaction on large HDL subclasses. The modest increase in the proportion of large HDL was primarily due to an increase in HDL\(_{2a}\) in the treatment (\(P = 0.004\)) and control (\(P = 0.084\)) groups. The increase in large HDL in both groups corresponded with a trend (\(P = 0.068\), time effect) for a decrease in the proportion of small HDL\(_{3a-3b-3c}\) particles.

**Plasma neutral sterol concentrations**

In the treatment group, the plasma campesterol concentration increased from 8.0 ± 4.0 \(\mu\)mol/L at baseline to 13.1 ± 4.1 \(\mu\)mol/L after 4 wk (\(P \leq 0.001\)). Plasma \(\beta\)-sitosterol concentrations tended to increase (3.9 ± 2.4 to 5.9 ± 1.8 \(\mu\)mol/L, \(P = 0.1\)). Plasma stigmasterol concentrations did not change significantly (0.50 ± 0.70 to 0.78 ± 0.62 \(\mu\)mol/L).

**Family history of CVD and plasma LDL cholesterol**

Approximately one-half of the subjects in each group reported a family history of premature CVD and elevated blood lipids. In the treatment group, 8 subjects reported a family history and 9 subjects reported no family history of CVD. At baseline, subjects in the treatment group who had a family history had higher TC (6.3 ± 0.9 mmol/L) and LDL-C (5.55 ± 0.5 mmol/L) concentrations (\(P < 0.01\)) than did subjects without a history (4.35 ± 0.8 and 3.85 ± 0.5 mmol/L for TC and LDL-C, respectively). However, at week 4, the magnitude of decreases in TC and LDL-C was not significantly different between these subgroups of the treatment group. The mean reductions in TC and LDL-C were 8.7% and 12.6% for the subjects with a family history and 8.9% and 15.6% for the subjects without a family history of CVD.

**Body weight and plasma metabolic variables**

Subjects in the treatment and control groups tended (\(P = 0.09\), time effect) to gain a small amount of weight during the 4-wk experimental period (0.34 ± 1.1 kg, range: -2.2 to +2.6 kg). Treatment had an effect on blood urea nitrogen:creatinine, which increased from
baseline 11.9 ± 3.0 to 13.8 ± 2.8 in the treatment group and decreased from 14 ± 3.0 to 13.8 ± 2.6 in the control group ($P = 0.045$, treatment-by-time interaction). Phytosterol intake tended to influence total blood urea nitrogen, which increased from 5.0 ± 1.0 to 6.0 ± 1.5 mmol/L in the treatment group but did not change in the control group (6.0 ± 1.0 to 6.0 ± 1.0) ($P = 0.061$, treatment-by-time interaction). There was no effect of treatment on plasma total protein, albumin, or creatinine. However, in both groups, there were small but significant ($P < 0.001$, time effect) changes in plasma total protein from 75 ± 4.0 to 76 ± 4.0 g/L, albumin from 47 ± 2 to 48 ± 2 g/L, and creatinine from 109 ± 10 to 110 ± 10 $\mu$mol/L. Plasma concentrations of protein, creatinine, nitrogen, nitrogen:creatinine, and all other metabolic variables were nonetheless within the reported normal range (24). The serum liver enzymes alkaline phosphatase and aspartate aminotransferase were not influenced by treatment.

**Discussion**

The present study differs from previous studies (1, 25, 26) on the effects of phytosterol consumption on plasma cholesterol concentrations in several respects. First, the phytosterols were mixed into a ground beef product that was low in fat and consumed only once daily. Previous experiments examined the effects of phytosterols in high-fat products such as margarine or spreads that were usually consumed 3 times/d (12, 25, 26). Second, to the best of our knowledge, the current study is the first to report the effects of phytosterol consumption on LDL and HDL subclass patterns. Lipoprotein subclasses are influenced by dietary and pharmacological interventions that affect LDL-C concentrations (27–33). Finally, the present study tested the effects of phytosterols in young adult men with at least 3 risk factors for developing CVD. Most other studies tested the effect of phytosterol-supplemented food products primarily in older normo- and hypercholesterolemic adults (12, 25, 26).

Our results show that lean ground beef is a viable food matrix to deliver an effective dose of phytosterols to lower plasma TC and LDL-C in young men. The 9% and 15% declines in TC and LDL-C, respectively, after the consumption of a single daily dose of 2.7 g of soybean phytosterols in ground beef are similar to the 8–13 % decline in plasma TC and LDL-C after consumption of multiple daily doses of a similar amount and type of soybean phytosterols in margarine (25). Furthermore, the magnitude of the decline in TC and LDL-C
in our study is comparable with the 8–12% decline observed in studies with stanol ester-fortified margarine (12). Thus, the phytosterol mixture in beef, like phytosterol esters in margarine (26), can lower plasma TC and LDL-C concentrations as effectively as margarine containing stanol esters.

Phytosterols are generally thought to lower plasma cholesterol by interfering with absorption of dietary and biliary cholesterol (34–38). It has been suggested that phytosterols act most effectively when consumed along with cholesterol-containing foods (35, 39, 40). Thus, many published studies report feeding phytosterol-fortified foods at most if not all meals. A recent study by Plat et al. (41) showed that consumption of 2.5 g of phytostanol esters at one meal was as effective in lowering LDL-C as consumption of the same phytostanol dose divided over 3 meals. Our findings show that soybean phytosterols consumed in a single daily dose also have a significant hypocholesterolemic effect. The efficacy of a single relatively large dose may result from saturating the enterocyte and prolonging the presence of the phytosterol within the cell, thereby inhibiting cholesterol absorption during subsequent meals. There is experimental evidence suggesting that such a hypothesis is tenable. Approximately 5% of β-sitosterol is absorbed and substantial amounts are incorporated into intestinal mucosal cell membranes (42–44). In enterocytes, phytosterols may inhibit cholesterol esterification and cause non-esterified cholesterol to diffuse or to be transported back into the intestinal lumen (45, 46).

Neither the most effective dose nor the optimal cholesterol-lowering ratio of esterified to free soybean phytosterols was determined in this study. There was no significant association between the percentage of change in plasma LDL-C concentration and total phytosterol intake in a range of doses from 0.025 to 0.0425 g/kg body weight (1.75–3.0 g/d for a 70 kg individual). Decreases in LDL-C, similar to those in the current study, were reported in subjects consuming sterol esters in margarine at either 0.02 or 0.045 g/kg body weight (1.6–3.4 g/d; 26).

To our knowledge no studies have examined the effects of phytosterol consumption on changes in lipoprotein subclass patterns. Dietary interventions can reduce LDL-C by decreasing LDL particle number, particle size, or both in some individuals (28). A reduction in particle size is undesirable because smaller LDL is associated with an atherogenic lipid
profile (47), although some researchers do not think this evidence is compelling (48). In our study, subjects fed phytosterol-supplemented beef did not have a significant shift from larger LDL to smaller LDL. This suggests that the decrease in LDL-C resulted primarily from a decrease in the number of LDL particles. Indeed, the NMR analysis performed at week 4 showed that the subjects in the treatment group tended to have a lower mean LDL particle number than did the control subjects. There is evidence that LDL particle size is influenced by metabolic and dietary factors affecting plasma triacylglycerol (49–51) and is strongly and inversely correlated with plasma triacylglycerol concentrations (52–54). Perhaps phytosterol supplementation had no effect on LDL particle size in our study because plasma triacylglycerol was not affected.

Phytosterol treatment did not significantly affect HDL-C concentrations or HDL subclasses. However, the percentage of HDL as large particles (HDL_{2a+2b}) increased and as small particles (HDL_{3a+3b+3c}) tended to decrease in both groups during the experiment. The changes were more apparent in the treatment than the control group. Larger HDL particles are thought to be more protective against atherosclerosis than smaller particles (55–57). A shift from larger to smaller HDL particles is observed in some subjects receiving dietary and drug treatments to lower LDL-C (27, 29–33). Studies with a greater number of subjects are necessary to determine if phytosterols significantly affect HDL subclasses.

Ground beef is a suitable food for phytosterol supplementation for several reasons. First, ground beef is the major single source of protein for young adult white men (58, 59). Second, consumption of moderate amounts of meat as part of a healthy diet is important in meeting recommended dietary allowances for vitamin B_{12}, niacin, and zinc (60). Third, lean ground beef (≤ 15% fat by weight) as part of 170 g (6 oz) cooked meat, poultry, or fish/d can be included in a healthy diet according to the American Heart Association (61,62).

Phytosterol-supplemented foods available to consumers today are spreads such as Benecol (McNeil Consumer Products, Fort Washington, PA) and Take Control (Lipton, Englewood Cliffs, NJ). When used as directed (3 times/d) these foods add 12–27 g extra fat but provide no other nutrients (63, 64). The high prevalence of overweight and obesity in the United States makes limiting fat consumption a priority. The phytosterol-supplemented lean ground beef used in this study has less total fat and is considerably more nutritious than the currently
marketed phytosterol spreads. One disadvantage of fortifying foods with phytosterols is that phytosterols lower the absorption of β-carotene (65, 66). Although we did not measure plasma concentrations of β-carotene, Plat et al. (41) reported that a decrease in plasma β-carotene concentrations was similar when stanol ester supplemented food was consumed either 1 or 3 times/d.

The results of our study show that lean ground beef is an excellent vehicle for delivering a dose of plant sterols to effectively lower plasma TC and LDL-C concentrations and improve the TC:HDL-C, while providing approximately 29 g of protein and only about 13 g of fat. Clearly, ground beef supplemented with phytosterols could become a hypocholesterolemic functional food consumed as part of a healthy diet to lower the risk of heart disease in mildly hypercholesterolemic young adults.

Acknowledgement

This work was supported by ConAgra, Inc, Omaha; the Center for Designing Foods to Improve Nutrition, Iowa State University; NIH grants HL-28972 and HL-45522; the Hatch Act; and the State of Iowa.

References


Table 3.1. Lipid and sterol composition of cooked control and phytosterol-supplemented (treatment) ground beef samples.\(^1\)

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Control beef ((n = 8))</th>
<th>Treatment beef ((n = 8))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triacylglycerol (g/100g)</td>
<td>10.9 ± 0.91</td>
<td>11.1 ± 1.91</td>
</tr>
<tr>
<td>Fatty acids (% of total fat)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C8:0</td>
<td>0.01 ± 0.002</td>
<td>0.02 ± 0.003</td>
</tr>
<tr>
<td>C10:0</td>
<td>0.05 ± 0.001</td>
<td>0.06 ± 0.003</td>
</tr>
<tr>
<td>C12:0</td>
<td>0.07 ± 0.001</td>
<td>0.08 ± 0.004</td>
</tr>
<tr>
<td>C14:0</td>
<td>2.86 ± 0.022</td>
<td>2.59 ± 0.167</td>
</tr>
<tr>
<td>C14:1</td>
<td>0.70 ± 0.013</td>
<td>0.53 ± 0.028</td>
</tr>
<tr>
<td>C15:0</td>
<td>0.53 ± 0.009</td>
<td>0.46 ± 0.023</td>
</tr>
<tr>
<td>C16:0</td>
<td>25.8 ± 0.16</td>
<td>24.0 ± 0.365</td>
</tr>
<tr>
<td>C16:1</td>
<td>3.57 ± 0.119</td>
<td>1.09 ± 0.082</td>
</tr>
<tr>
<td>C17:0</td>
<td>1.34 ± 0.02</td>
<td>1.09 ± 0.019</td>
</tr>
<tr>
<td>C17:1</td>
<td>0.80 ± 0.009</td>
<td>0.60 ± 0.011</td>
</tr>
<tr>
<td>C18:0</td>
<td>18.4 ± 0.265</td>
<td>16.5 ± 0.35</td>
</tr>
<tr>
<td>C18:1</td>
<td>18.1 ± 0.181</td>
<td>16.5 ± 0.614</td>
</tr>
<tr>
<td>C18:2</td>
<td>2.4 ± 0.144</td>
<td>7.8 ± 0.362</td>
</tr>
<tr>
<td>C18:3</td>
<td>0.45 ± 0.028</td>
<td>0.61 ± 0.065</td>
</tr>
<tr>
<td>C20:0</td>
<td>0.54 ± 0.033</td>
<td>ND(^2)</td>
</tr>
<tr>
<td>C20:4</td>
<td>0.46 ± 0.09</td>
<td>0.70 ± 0.194</td>
</tr>
<tr>
<td>Total sterols, g/100g</td>
<td>ND</td>
<td>2.7 ± 0.41</td>
</tr>
<tr>
<td>Cholesterol, mg/100g</td>
<td>85.7 ± 5.7</td>
<td>81.5 ± 11.1</td>
</tr>
<tr>
<td>(\beta)-sitosterol(^3), g/100g</td>
<td>ND</td>
<td>1.35</td>
</tr>
<tr>
<td>Campesterol(^3), g/100g</td>
<td>ND</td>
<td>0.75</td>
</tr>
<tr>
<td>Stigmasterol(^3), g/100g</td>
<td>ND</td>
<td>0.58</td>
</tr>
</tbody>
</table>

\(^1\) Mean ± SD.
\(^2\) ND, not detectable.
\(^3\) Calculated from information provided by Henkel Corporation (Unilever Research Laboratories, Vlaardingen, Netherlands; see Methods).
Table 3.2. Baseline characteristics of subjects in the control and phytosterol-supplemented (treatment) groups and of subjects who did not meet selection criteria.¹

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control group (n = 17)</th>
<th>Treatment group (n = 17)</th>
<th>Excluded subjects (n = 133)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>22.2 ± 3.9</td>
<td>23.6 ± 3.9</td>
<td>21.4 ± 2.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>81.2 ± 14.3</td>
<td>87.4 ± 15.1</td>
<td>81.4 ± 11.1</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.7 ± 3.5</td>
<td>27.0 ± 4.5</td>
<td>24.1 ± 2.4</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>5.80 ± 0.7</td>
<td>5.90 ± 0.8</td>
<td>4.10 ± 0.6</td>
</tr>
<tr>
<td>HDL</td>
<td>1.05 ± 0.2</td>
<td>1.10 ± 0.25</td>
<td>1.56 ± 0.15</td>
</tr>
<tr>
<td>LDL</td>
<td>3.95 ± 0.7</td>
<td>4.10 ± 0.65</td>
<td>2.40 ± 0.55</td>
</tr>
<tr>
<td>Total:HDL cholesterol</td>
<td>5.60 ± 1.2</td>
<td>5.50 ± 1.3</td>
<td>4.00 ± 1.0</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/L)</td>
<td>1.72 ± 0.72</td>
<td>1.52 ± 0.52</td>
<td>1.28 ± 0.76</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.1 ± 0.4</td>
<td>5.00 ± 0.4</td>
<td>−²</td>
</tr>
</tbody>
</table>

¹Mean ± SD. The data of subjects excluded from the study were not used for any analysis and are provided for comparison. There were no significant differences in any variable measured at baseline between the treatment and control groups.

²No data.
Table 3.3. Total plasma cholesterol, LDL cholesterol, HDL cholesterol, and triacylglycerol concentrations and total:HDL cholesterol in subjects at initial screening, baseline and after 2 and 4 weeks of consuming control or phytosterol-supplemented (treatment) ground beef.\(^1\)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Screening(^2)</th>
<th>Baseline</th>
<th>Week 2</th>
<th>Week 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>5.70 ± 0.05</td>
<td>5.80 ± 0.70</td>
<td>5.60 ± 0.60</td>
<td>5.75 ± 0.80</td>
</tr>
<tr>
<td>Treatment group</td>
<td>5.95 ± 0.70</td>
<td>5.90 ± 0.80</td>
<td>5.45 ± 0.75</td>
<td>5.35 ± 0.70(^3,4)</td>
</tr>
<tr>
<td>LDL Cholesterol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>4.00 ± 0.45</td>
<td>3.95 ± 0.60</td>
<td>3.85 ± 0.60</td>
<td>3.90 ± 0.70</td>
</tr>
<tr>
<td>Treatment group</td>
<td>4.05 ± 0.70</td>
<td>4.10 ± 0.65</td>
<td>3.65 ± 0.65</td>
<td>3.50 ± 0.70(^3,4)</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1.04 ± 0.15</td>
<td>1.05 ± 0.20</td>
<td>1.00 ± 0.20</td>
<td>1.05 ± 0.20</td>
</tr>
<tr>
<td>Treatment group</td>
<td>1.15 ± 0.30</td>
<td>1.10 ± 0.25</td>
<td>1.10 ± 0.25</td>
<td>1.15 ± 0.25</td>
</tr>
<tr>
<td>Total:HDL cholesterol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>5.6 ± 1.0</td>
<td>5.6 ± 1.2</td>
<td>5.8 ± 1.4</td>
<td>5.6 ± 1.1</td>
</tr>
<tr>
<td>Treatment group</td>
<td>5.5 ± 1.1</td>
<td>5.5 ± 1.3</td>
<td>5.1 ± 1.2</td>
<td>5.0 ± 1.2(^3,4)</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>1.58 ± 0.64</td>
<td>1.70 ± 0.75</td>
<td>1.70 ± 0.65</td>
<td>1.70 ± 0.65</td>
</tr>
<tr>
<td>Treatment group</td>
<td>1.66 ± 0.58</td>
<td>1.50 ± 0.50</td>
<td>1.55 ± 0.50</td>
<td>1.60 ± 0.65</td>
</tr>
</tbody>
</table>

\(^1\)Mean ± SD, \(n = 17\) in the control group and \(n = 17\) in the treatment group.

\(^2\)Initial screening was done 6 wk before the study; the data were not included in any analysis but are provided as additional information.

\(^3\)Significant treatment by time interaction, \(P \leq 0.001\) (repeated-measures ANOVA).

\(^4\)Significantly different from baseline, \(P < 0.001\) (Bonferroni-adjusted pairwise comparisons).
Table 3.4. Effect of phytosterol-supplemented ground beef on LDL and HDL subclass traits.¹

<table>
<thead>
<tr>
<th>Lipoprotein subclass and group</th>
<th>Baseline</th>
<th>Week 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL peak particle diameter (nm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>26.1 ± 0.7</td>
<td>26.1 ± 0.8</td>
</tr>
<tr>
<td>Treatment group</td>
<td>26.4 ± 0.5</td>
<td>26.3 ± 0.6</td>
</tr>
<tr>
<td>Large LDL, 25.5-29.0 nm (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>79.0 ± 14.6</td>
<td>79.4 ± 19.1</td>
</tr>
<tr>
<td>Treatment group</td>
<td>88.0 ± 10.1</td>
<td>84.4 ± 12.8</td>
</tr>
<tr>
<td>Small LDL, 21.0-25.5 nm (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>21.0 ± 14.6</td>
<td>20.6 ± 19.1</td>
</tr>
<tr>
<td>Treatment group</td>
<td>12.0 ± 10.1</td>
<td>15.6 ± 12.8</td>
</tr>
<tr>
<td>Large HDL₂₃₁₂b (%)³</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>45.6 ± 6.8</td>
<td>47.6 ± 6.5</td>
</tr>
<tr>
<td>Treatment group</td>
<td>40.5 ± 9.0</td>
<td>43.6 ± 9.0</td>
</tr>
<tr>
<td>Small HDL₃₃₀₃c (%)³</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control group</td>
<td>50.4 ± 8.1</td>
<td>49.0 ± 8.3</td>
</tr>
<tr>
<td>Treatment group</td>
<td>55.3 ± 9.5</td>
<td>51.6 ± 13.3</td>
</tr>
</tbody>
</table>

¹Means ± SD; n = 17 in the control group and 17 in the treatment group.
²Significant time effect, P = 0.007 (repeated-measures ANOVA).
³Time effect, P = 0.068 (repeated-measures ANOVA).
Figure 3.1. The correlation between the percentage change in LDL-cholesterol (LDL-C) from baseline and soybean phytosterol consumption was not significant \( (r = 0.32, n = 17) \). The percentage change in LDL-C was calculated by subtracting LDL-C plasma concentrations at baseline from LDL-C at week 4. Phytosterol consumption was calculated by dividing the phytosterol intake by the mean weight at baseline, week 2, and week 4.
CHAPTER 4: A COLLEGE NUTRITION SCIENCE COURSE AS AN INTERVENTION TO PREVENT WEIGHT GAIN IN FEMALE COLLEGE FRESHMEN

A paper published in the Journal of Nutrition Education\(^1\)
Oksana Matvienko\(^2\), Douglas S. Lewis\(^3\), Elisabeth Schaefer\(^4\)

Abstract

The objective of this study was to test the hypothesis that a nutrition course that stresses fundamental principles of human physiology, energy metabolism, and genetics helps prevent weight gain during the first 16 months of college life. A randomized control trial was conducted from January 1997 to May 1998 using volunteers. Forty female college freshmen participated in the intervention (college course, \(n = 21\)) and control (no course, \(n = 19\)) groups. The intervention was a one-semester nutrition science college course. Body weight, nutrient intakes, and knowledge were measured at baseline, the end of the intervention (4 months from baseline), and 1 year later (16 months from baseline). Statistical analysis was conducted using a repeated measures analysis of variance. Higher body mass index (BMI) students (BMI > 24) in the intervention group (\(n = 11\)) reported lower fat (\(p = 0.04\)), protein (\(p = 0.03\)), and carbohydrate (\(p = 0.008\)) intakes compared with the higher BMI students in the control group (\(n = 6\)). Dietary changes reported by the higher BMI intervention students were associated with the maintenance of baseline body weight for 1 year in contrast with the higher BMI control students who gained 8.4 ± 6.8 kg (\(p = 0.012\)). The findings suggest that nutrition education emphasizing human physiology and energy metabolism is an effective strategy to prevent weight gain in at-risk college students.

\(^2\) Graduate student and primary author. Department of Food Science and Human Nutrition, Iowa State University, Ames.
\(^3\) Associate Professor and author for correspondence. Department of Food Science and Human Nutrition, Iowa State University, Ames.
\(^4\) Professor, Department of Food Science and Human Nutrition, Iowa State University, Ames.
Introduction

The incidence of overweight and obesity is increasing in the United States despite high intakes of non-caloric, sugar-free, and low fat foods.\textsuperscript{1,2} Obesity and even body weight at the high end of the normal range are associated with the increased prevalence of chronic diseases and mortality.\textsuperscript{3-5} Considering that current treatments for obesity and overweight are largely ineffective, the prevention of obesity is an important health priority.\textsuperscript{6}

Although unwanted weight gain can occur at any age, it commonly occurs between 18 and 34 years of age.\textsuperscript{7-9} The transition from high school to college may be an important period when individuals may experience weight gain (the legendary "freshman 15").\textsuperscript{10,11}

Many high school students lack sufficient knowledge of nutrition and physiology to adequately deal with the pending weight gain that may occur with age. Although 83\% of high school students knew that excess energy is stored as body fat, 25\% could not recognize from where energy originates.\textsuperscript{12} Only 43\% recognized that dietary fat is more energy dense than carbohydrate and protein. Lack of basic physiologic knowledge is also evident. Only 27\% of students realized that body fat is essential for health, and less than 22\% knew the number of calories needed to be expended to lose one pound of fat.\textsuperscript{12}

Most students learn nutrition from home economics classes, television, parents, labels of food packages, radio, books, and newspapers and magazines.\textsuperscript{13} These sources often provide easy-to-understand factual information on foods, nutrients in food, and eating behavior. The higher critical thinking skills of synthesis and problem solving -- necessary to understand how biology and nutrition interact to determine body weight -- are seldom addressed. Therefore, most teenagers learn nutrition in a "black box" with little clue as to how the body uses energy. The purpose of this study was to test the hypothesis that a college level science-based course that stresses principles of human physiology, energy metabolism, genetics, and nutrient utilization will help prevent weight gain in first year college women.

Method

Subjects

Posted flyers and student newspaper advertising were used to recruit subjects for the 16-month study. The recruitment materials noted the commonality of unwanted weight gain
among young adults and invited students to enroll for a two-credit class in the scientific principles of weight regulation. Students were told they could earn $200. Qualifications for participation were listed – being a freshman or sophomore, aged 18 to 26, and without any previous nutrition class. Forty college women were randomly assigned to the intervention (n = 21) or control (n = 19) groups. Only female students were studied because an insufficient number of male students responded to the recruitment effort. Three subjects, originally assigned to the intervention group, were transferred to the control group because of academic schedule conflicts. Replacements were semi-randomly chosen (based on academic schedule) from the control group.

Thirty-five subjects were Caucasian, two were African-American, one was Asian, and two did not identify their ethnicity. Forty subjects completed the four-month intervention and thirty-three subjects completed the one-year follow-up. The dropouts did not differ significantly from the subjects who completed the study in baseline age, body weight, BMI, parents’ BMI, energy, and macronutrient intakes.

Study design

Body weight, nutrient intakes, and knowledge were measured at baseline, at the end of the intervention (four months from baseline), and one year following the end of the intervention (16 months from baseline) using the same instruments at each time.

Height and weight were measured, and body mass index was calculated \[\text{BMI} = \frac{\text{wt (kg)}}{\text{ht (m}^2)}\] for all subjects. Height was measured on a platform with a rigid vertical upright board to which two wooden measuring sticks (calibrated in tenths of cm) were attached. Barefooted, the subject stood erect with heels, buttocks, and shoulders in contact with the upright and the head held so that the line of sight was horizontal. Height was recorded to the nearest 0.1 cm. Weight was measured to the nearest 0.1 kg by using a “Health O Meter” scale (ABCO Health O Meter Inc., Bridgeview, IL). Subjects wore light street clothes and removed their shoes, pocket contents, and watches prior to weighing.

Dietary intake was estimated with a 116-item semi-quantitative food frequency questionnaire designed by Block and associates\(^14\) and used by the National Cancer Institute.\(^15\) The questionnaire has been validated with biochemical indicators and food records.\(^16,17\)
**Intervention**

The intervention was not a weight loss program, and directions on how to lose weight by dieting and exercise were not provided. The intervention was a college course for credit, composed of both lectures and laboratory exercises. During the lectures students were taught science principles related to dietary and body sources of biological energy, conversion of potential energy to active energy, and the interaction between genetic factors, food composition, diet, and physical activity and energy utilization. Basic information on how cells and body tissues utilize fat was provided. The effects of dieting, eating disorders, fasting, and pharmacological and drug interventions on energy balance were also discussed. Students were exposed to the scientific literature through discussions of relevant published human studies on energy balance and its control by genetic, dietary, and physical activity factors. Classroom exercises included estimation of basal metabolic rate, body composition, fat distribution, energy expenditure, and fat utilization for various physical activities. Laboratory exercises included body composition measurements, serving sizes, food sensory exercises with low and high fat foods, and food preparation methods. The intended course outcomes are presented in Table 4.1.

All subjects were given the same knowledge test at baseline, four months, and 16 months. The test was a typical college final exam composed of multiple choice and short answer questions and took approximately two hours to complete. Test results were categorized as 1) total knowledge score; 2) nutrition knowledge score (food composition and energy content, Food Guide Pyramid and labels, dietary recommendations, and serving sizes); 3) physiologic knowledge score (adipose and muscle physiology, neural and systemic control of hunger and appetite, body composition); and 4) metabolic knowledge score (nutrient oxidation during fasting, postprandial, postabsorptive, and exercise; lipolysis and lipogenesis). Maximal possible scores were 13 points for nutrition knowledge, 50 points for physiologic knowledge, and 26 points for metabolic knowledge. The total maximal score was 70 points. During the data analysis, some questions were counted in more than one knowledge domain. Reliability coefficients for the measure of knowledge, using a test-retest estimate of reliability, were: \( r (9) = .881, p < 0.05 \) for overall knowledge score, \( r (9) = .907, p < 0.05 \) for nutrition knowledge score, \( r (9) = .761, p < 0.05 \) for knowledge of energy
metabolism, and \( r (9) = .744, p < 0.05 \) for physiologic knowledge. Validity was determined by a panel of three nutrition professors.

**Statistical analysis**

Analysis of the intervention effects was conducted using a repeated measures analysis of variance (ANOVA) (SPSS/PC+9.0, SPSS Inc, Chicago, IL), with groups as a between-subject factor and time of measurement (baseline, 4 and 16 months) as a within-subjects factor. BMI status at baseline was used as an independent variable to determine whether the intervention had effects on subjects with different BMIs. We divided students into upper \((n = 17)\) and lower \((n = 23)\) BMI groups using a natural break point in the data. Desirable BMI was defined as BMI \(\leq 24\) and the higher BMI was defined as BMI \(> 24\).

Data were checked for normality and logarithmic transformation was performed when appropriate. Significance was set at \( \alpha < 0.05 \), but we also report differences when \( \alpha \) was less than 0.1 to better balance statistical Type I and Type II errors. The effects tested in the analyses were the treatment-by-time and treatment-by-BMI status-by-time interactions during two periods: baseline to 4 months (short-term effect) and 4 to 16 months (long-term effect). When there was a significant interaction, the differences between groups were further examined by using an independent-samples t-test.

**Results**

**Subject characteristics**

The intervention and control groups did not differ at baseline, with the exception of knowledge: the intervention group had significantly lower scores on physiology and energy metabolism knowledge than did the control group (Table 4.2).

**Knowledge**

The mean ratio of knowledge scores was calculated to determine the improvement of subjects' knowledge between baseline and the end of the intervention (mean ratio = score at the end of the intervention/score at baseline). As expected, the total knowledge score of the intervention subjects improved \(2.2 \pm 1.5\) fold (mean ratio \(\pm\) SD), whereas the total knowledge score of the control subjects did not change (ratio, \(1.0 \pm 0.2\); between-groups difference, \(p = 0.001\)). The significant improvement in total knowledge in the intervention group was
attributed to the increase in all knowledge domains with a greater improvement in
physiologic and metabolic knowledge than in nutrition knowledge (Figure). At 16 months,
the total knowledge score of the intervention group declined somewhat but remained
significantly higher (ratio, 1.8 ± 1.3) than the score of the control group (1.1 ± 1.3; between-
groups difference, p = 0.05). Retention of knowledge by the intervention group varied by
knowledge domain: physiologic and metabolic knowledge was retained better than nutrition
knowledge (Figure 4.1). Baseline BMI status had no effect on knowledge acquisition or
retention.

Dietary intake

At the end of the intervention, the intervention subjects reported consuming fewer
kcal/day than at baseline, whereas the control subjects reported consuming more kcal/day
(time-by-treatment interaction, p = 0.013). Energy intake per kilogram of body weight was
lower in the intervention group than in the control group (time-by-treatment interaction, p =
0.014). The changes in energy consumption reported by the intervention and control groups
corresponded to changes in carbohydrate and protein intakes (time-by-treatment interaction
for carbohydrate, p = 0.004; for protein, p = 0.066). Although total fat intake decreased by
18% in the intervention group, the difference in total fat intake between the groups was not
statistically significant.

The significant differences in self-reported energy, carbohydrate, and protein intakes
observed between the groups at four months disappeared at one year after the end of the
intervention. However, the intervention subjects were consuming a mean of 23 g of fat per
day less than the control subjects, although this was not statistically significant due to large
variability in fat intake.

Body weight and BMI

The mean weight and BMI were unchanged in both groups during the 4-month
intervention and 12 months following the intervention (Table 4.3). The majority of subjects
(70%) showed little or no change in total body weight although 30% of subjects (15% in the
intervention and 15% in the control group) showed a greater than 5% weight gain or loss.
Interaction of baseline BMI status with the intervention

Dividing the intervention and control subjects into two BMI groups -- desirable and higher -- highlighted the effects of the intervention. The higher BMI group included 11 of the original 21 intervention subjects and 6 of the original 19 control subjects. At baseline, the higher BMI subjects had significantly higher weight (77 vs. 60 kg), BMI (28 vs. 22), average parental BMI (30 vs. 26), and reported consuming more energy as fat (30% vs. 25%, p < 0.1) compared with the desirable BMI group. Overall, there were no significant differences between the intervention and control students with desirable BMI throughout the study. However, there were significant differences between the higher BMI intervention and control groups. At 4 months, the higher BMI intervention subjects reported consuming 548 fewer kcal/day, whereas the higher BMI control subjects reported consuming 319 more kcal/day (treatment-by-BMI interaction, p = 0.018). The difference between the groups in energy intake was attributed to the changes in the intakes of fat (p = 0.04), protein (p = 0.03), and carbohydrate (p = 0.008). The higher BMI intervention subjects significantly decreased their percent of energy from fat from baseline to 4 months while there was no difference for the higher BMI control group during this period (Table 4.4).

At 16 months, there was no significant difference in self-reported energy intake between the higher BMI intervention and control groups. However, the percent of energy from fat remained lower in the higher BMI intervention group compared with the higher BMI control group (p = 0.002). At 16 months, the higher BMI intervention subjects had lost on average 1.4 kg of body weight, whereas the higher BMI control subjects had gained on average 9.2 kg (p = 0.025) (Table 4.4).

Discussion

Despite study design limitations, including a small sample size and self-selection of subjects, the study results provide evidence that a college nutrition science course focused on energy metabolism is a viable intervention to help freshmen college students maintain body weight during the first 16 months of college life. Students who took the course demonstrated improved knowledge of nutrition, energy metabolism, and physiologic mechanisms of energy balance. Perhaps more importantly, they retained the more complex concepts of metabolic
and physiologic mechanisms of energy balance for 1 year after the end of the course
intervention. It was not clear that such acquisition and retention of course knowledge was
effectively translated into maintaining body weight largely because 70% of students,
including those in the control group, did not change weight during the first 16 months of
college life. Nevertheless, when only subjects with higher BMIs were considered, the
intervention clearly exerted a positive influence. The students who took the course
maintained their body weight whereas the control students gained a considerable amount of
weight. However, the low number of subjects in the higher BMI control group necessitates a
larger study to confirm these preliminary results.

Only a few interventions have been directed toward weight gain in college students
and those focused on weight reduction in overweight individuals. Sloan et al.\textsuperscript{19} reported
weight loss with a weight control program that used diet and behavior modification, but not
education. There was a 40\% dropout rate. Hudiburgh\textsuperscript{20} combined behavior modification, a
nutrition course, and exercise, and reported that the overweight students lost 10 pounds by
the end of a semester. However, the follow-up of the students was incomplete, and a small
number reported that they were able to maintain the weight loss 1 year later.

Numerous studies have investigated the effects of nutrition education on weight loss
in obese subjects, but few studies have used educational interventions to prevent weight gain
in non-obese individuals. The goal of preventing weight gain in college freshmen is
comparable to the goals of the Pound of Prevention Study targeted toward older adults.\textsuperscript{21-24}
The Pound of Prevention study examined the efficacy of low-intensity education to prevent
weight gain in diverse groups of adults. These studies demonstrated that the low intensity
approach to weight gain prevention sustained interest over a lengthy period of time but had
little effect in preventing weight gain relative to a control group.\textsuperscript{23} Earlier studies from the
Pound of Prevention group reported that the intervention used was more successful in those
with heavier weights at baseline,\textsuperscript{21} similar to our results; however, we used an intensive
educational approach.

Consistent with a previous report for high school students,\textsuperscript{25} the female freshmen in
our study demonstrated a low baseline knowledge of nutrients, nutrient energy contents, food
labels, and the groups of the U.S. Department of Agriculture (USDA) Food Guide Pyramid.
They also knew virtually nothing about energy metabolism and expenditure. High school nutrition education is predominantly taught within a family and consumer sciences class and emphasizes broad areas such as types of nutrients, food labels, and food groups. Gillespie\textsuperscript{26} suggested that complex concepts presented as narrowly focused messages may be more effective in enhancing analytical and decision-making skills necessary for better knowledge retention. Our course included several challenging concepts, for instance, how genetics, diet, and physical activity influence the partitioning of energy between storage and expenditure. Consistent with studies by Contento et al.\textsuperscript{27} and Chapman et al.,\textsuperscript{28} our subjects demonstrated high scores on all knowledge domains upon the completion of the course. Since the major focus of the course was on physiology and energy metabolism, it was not surprising that the increase in metabolic and physiologic knowledge was considerably higher than that observed for nutrition knowledge.

The intervention students retained physiologic and metabolic knowledge for up to 1 year. In contrast, knowledge of the food label, nutrients, energy density, and the USDA Food Guide Pyramid was not retained 1 year after the course concluded. Why the more complex concepts of energy metabolism were retained is not known, although this may be at least partially explained by the course design and emphases and the pedagogical techniques used for teaching this course. These techniques included classroom activities, demonstrations with three-dimensional models, and multi-modal sensory lectures.\textsuperscript{29} There were no detailed handouts accompanying the lectures, which required that notes be taken during class.\textsuperscript{30} Laboratory exercises, lectures focused on problem solving,\textsuperscript{31,32} and frequent quizzes may have also strengthened retention of knowledge.\textsuperscript{33}

A major finding of the current experiment was that the intervention had a profound effect on a subgroup of students who were at greater risk for weight gain, specifically students with a higher baseline BMI, higher parental BMI, and higher fat intakes. These students might have been more likely to make dietary changes because they might feel themselves to be at higher risk. The effectiveness of the intervention to prevent weight gain in the higher BMI students is consistent with others\textsuperscript{21,34} who found that weight gain prevention programs had better success among heavier individuals. In our study, the higher BMI intervention students significantly reduced total energy and, more importantly, daily fat
intake compared with higher BMI control students. Indeed, the percent of energy from fat and total fat intake of the higher BMI intervention students declined such that they were lower than those reported by higher BMI control and desirable BMI students. Many of the differences between the higher BMI intervention and control groups decreased in magnitude or disappeared over time. However, one year after the end of the intervention, the percent of energy from fat was still significantly lower for the higher BMI students who completed the nutrition course. Decreased fat consumption could be one of the major contributors to the ability of these students to maintain their baseline weight in contrast with the control students who gained weight.24

The results of this study should be carefully interpreted for several reasons. First, the subjects were self-selected, and as such they are likely to be more weight conscious and be more receptive to information relating to health than the general student population. There were, however, no significant differences in the answers to the food frequencies and psychosocial questionnaires between the students in the experiment and 37 female college students starting a general introductory nutrition class.35 Second, the number of subjects in our study is small. The lack of significant effects on nutrient intake is likely explained by the low power of the study as a result of the small sample size. Third, our study did not compare a variety of modalities of education which limits the interpretation that learning nutrition biochemistry and physiology is more effective at weight control than learning general nutrition. The application of a “college” course to the general public is probably not feasible, given the potential cost and that a high level of academic commitment, preparation, and effort is expected. Finally, the long-term effects of a college course were not assessed.

Implications for Research and Practice

The results of this study support targeted education that translates research results from the literature on human obesity and weight control as a strategy for preventing unwanted weight gain in young adults. In particular, this study showed that a specific educational intervention was most effective for members of a population at-risk for weight gain. Nutrition educators can use these results to tailor a science-based, problem-solving educational intervention approach, targeted towards the higher BMI late adolescent or young
adult. In a climate of increasing overweight and obesity at younger and younger ages, the National Task Force on Prevention and Treatment of Obesity proposed that prevention strategies such as increased physical activity and low-fat diets be targeted to susceptible individuals and groups. Our results suggest that a nutrition science course works and is likely to enhance the nutrition educator's effectiveness in addressing an important public health problem.

Acknowledgment

This work was supported by a grant from the Center for Designing Foods to Improve Nutrition, Iowa State University.

References

Table 4.1. Course outcomes.

**Students are expected to know:**
1. The current trends for the prevalence of overweight for the US population.
2. The ages that overweight is most likely to occur.
3. That body weight in a normal adult reflects the extent of energy stored and that changes in weight are reflected by changes in the amount of stored energy.

**Students are expected to be able to:**
1. Determine how much energy they need.
2. Calculate the amount of energy in food.
3. Determine how much energy they eat.
4. Estimate their body composition and relative risk for chronic disease and early morbidity.
5. Calculate how much energy they use daily.

**Students are expected to understand:**
1. How potential energy in food and the body is converted to active energy.
2. How potential energy is stored in fat tissue and how energy in fat tissue is mobilized.
3. How exercise uses the potential energy in fat.
4. How overeating results in weight gain.

**Students are expected to discover:**
1. Whether one can spontaneously lose weight after short-term overfeeding.
2. Five reasons why eating too much effectively increases body fat.
3. Whether eating carbohydrates really increases body fat.
4. How much fat can be actually lost in one week.
Table 4.2. Characteristics of the intervention and control groups at baseline (mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>Intervention (n = 21)</th>
<th>Control (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometric measurements</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>19.3 ± 0.8</td>
<td>19.5 ± 1.1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165.8 ± 7.6</td>
<td>166.6 ± 7.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>67.7 ± 12.9</td>
<td>65.7 ± 12.7</td>
</tr>
<tr>
<td>BMI [Wt (kg)/Ht (m²)]</td>
<td>24.6 ± 4.7</td>
<td>23.7 ± 4.6</td>
</tr>
<tr>
<td>Mothers' BMI</td>
<td>27.0 ± 7.4</td>
<td>27.2 ± 6.4</td>
</tr>
<tr>
<td>Fathers' BMI</td>
<td>28.2 ± 5.7</td>
<td>27.5 ± 4.8</td>
</tr>
<tr>
<td>Average Parents' BMI</td>
<td>27.8 ± 5.6</td>
<td>27.4 ± 5.2</td>
</tr>
<tr>
<td><strong>Knowledge (test scores)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>23.5 ± 7.7</td>
<td>29.4 ± 7.7</td>
</tr>
<tr>
<td>Nutrition</td>
<td>5.2 ± 2.5</td>
<td>6.1 ± 2.2</td>
</tr>
<tr>
<td>Physiology</td>
<td>15.3 ± 6.2&lt;sup&gt;1&lt;/sup&gt;</td>
<td>20.1 ± 6.1</td>
</tr>
<tr>
<td>Metabolism</td>
<td>6.8 ± 3.9&lt;sup&gt;1&lt;/sup&gt;</td>
<td>10.2 ± 3.2</td>
</tr>
</tbody>
</table>

<sup>1</sup> Difference from the control group, p < .05.
Table 4.3. Body composition and energy and nutrient intakes at baseline, 4 months, and 16-months (mean ± SD).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>n</th>
<th>Baseline</th>
<th>4 months</th>
<th>n</th>
<th>16 month</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometric measurements</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight, kg</td>
<td>Intervention</td>
<td>21</td>
<td>67.7 ± 12.9</td>
<td>67.5 ± 12.4</td>
<td>18</td>
<td>67.7 ± 13.6</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>65.7 ± 12.7</td>
<td>67.5 ± 12.7</td>
<td>15</td>
<td>68.9 ± 18.4</td>
</tr>
<tr>
<td>BMI [kg/m²]</td>
<td>Intervention</td>
<td>21</td>
<td>24.6 ± 4.7</td>
<td>24.6 ± 4.6</td>
<td>18</td>
<td>24.5 ± 4.8</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>23.7 ± 4.6</td>
<td>24.1 ± 4.8</td>
<td>15</td>
<td>25.2 ± 6.5</td>
</tr>
<tr>
<td><strong>Self-reported energy and macronutrient intakes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Energy (kcal/day)</td>
<td>Intervention</td>
<td>21</td>
<td>2056 ± 893</td>
<td>1730 ± 691²</td>
<td>18</td>
<td>1841 ± 802</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>2172 ± 1206</td>
<td>2244 ± 1000</td>
<td>15</td>
<td>2105 ± 808</td>
</tr>
<tr>
<td>Energy/Wt (kcal/kg)</td>
<td>Intervention</td>
<td>21</td>
<td>30.6 ± 11.7</td>
<td>26.0 ± 9.8²</td>
<td>18</td>
<td>27.7 ± 11.6</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>34.2 ± 19.2</td>
<td>35.3 ± 17.0</td>
<td>15</td>
<td>32.0 ± 14.4</td>
</tr>
<tr>
<td>Energy from Fat (%)</td>
<td>Intervention</td>
<td>21</td>
<td>29.3 ± 10.3</td>
<td>25.7 ± 10.9</td>
<td>18</td>
<td>28.8 ± 10.0⁴</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>30.5 ± 7.9</td>
<td>29.7 ± 10.8</td>
<td>15</td>
<td>32.9 ± 9.7</td>
</tr>
<tr>
<td>Total Fat (g/day)</td>
<td>Intervention</td>
<td>21</td>
<td>72.2 ± 58.2</td>
<td>59.2 ± 39.2</td>
<td>18</td>
<td>58.1 ± 33.9</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>73.9 ± 46.6</td>
<td>69.6 ± 45.8</td>
<td>15</td>
<td>81.5 ± 50.3</td>
</tr>
<tr>
<td>Total Protein (g/day)</td>
<td>Intervention</td>
<td>21</td>
<td>80.6 ± 24.3</td>
<td>68.2 ± 21.9⁴</td>
<td>18</td>
<td>71.7 ± 32.7</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>84.4 ± 40.9</td>
<td>89.2 ± 37.6</td>
<td>15</td>
<td>81.1 ± 28.3</td>
</tr>
<tr>
<td>Total CHO (g/day)</td>
<td>Intervention</td>
<td>21</td>
<td>266.2 ± 96.8</td>
<td>227.9 ± 104.5³</td>
<td>18</td>
<td>256.9 ± 127.7</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>19</td>
<td>286.3 ± 163.1</td>
<td>312.4 ± 138.8</td>
<td>15</td>
<td>260.5 ± 83.7</td>
</tr>
</tbody>
</table>

¹,² Difference from the control group at the end of the intervention. ³p < 0.1, ²p < 0.05, ⁴p < .01.

*Difference from the control group 1 year after the intervention, p < .1.
Table 4.4. Characteristics of the higher BMI intervention and higher BMI control groups (mean ± SD).

<table>
<thead>
<tr>
<th></th>
<th>n Baseline</th>
<th>4 months</th>
<th>16 months</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometric measurements</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>75.4 ± 12.1</td>
<td>74.6 ± 12.2</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>80.5 ± 15.3</td>
<td>81.3 ± 16.2</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>27.5 ± 5.3</td>
<td>27.2 ± 5.2</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>29.3 ± 5.4</td>
<td>29.5 ± 5.9</td>
</tr>
<tr>
<td><strong>Self-reported energy and macronutrient intakes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Energy (kcal)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>2270 ± 1012</td>
<td>1722 ± 858</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>1789 ± 771</td>
<td>2108 ± 1416</td>
</tr>
<tr>
<td>Energy/Wt (kcal/kg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>30.4 ± 12.3</td>
<td>23.1 ± 10.5</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>24.1 ± 15.1</td>
<td>30.6 ± 26.0</td>
</tr>
<tr>
<td>Energy as Fat (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>29.9 ± 11.1</td>
<td>21.6 ± 9.7</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>36.8 ± 7.1</td>
<td>37.3 ± 13.4</td>
</tr>
<tr>
<td>Total Fat (g/day)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>83.5 ± 74.1</td>
<td>57.4 ± 46.7</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>72.9 ± 34.6</td>
<td>81.2 ± 69.6</td>
</tr>
<tr>
<td>Total Protein (g/day)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>87.9 ± 19.6</td>
<td>66.9 ± 27.9</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>71.8 ± 28.7</td>
<td>70.8 ± 44.7</td>
</tr>
<tr>
<td>Total CHO (g/day)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Higher BMI Intervention</td>
<td>11</td>
<td>283.5 ± 91.6</td>
<td>232.4 ± 115.9</td>
</tr>
<tr>
<td>Higher BMI Control</td>
<td>6</td>
<td>205.0 ± 99.6</td>
<td>267.5 ± 167.6</td>
</tr>
</tbody>
</table>

$^a,b$ Difference from the higher BMI control group. $^a p < .05$. $^b p < .01$. 
Figure 4.1. Effect of the college course on the acquisition and retention of knowledge domains. Data presented are mean ratios of test scores to baseline scores. Error bars represent standard deviations. The asterisks indicate significant differences between the mean ratios of the intervention and control groups.
CHAPTER 5: COLLEGE STUDENTS’ PERCEPTION OF OWN WEIGHT, ATTITUDES TOWARD DIETING, AND KNOWLEDGE AND BELIEFS ABOUT BODY WEIGHT

A paper to be submitted to the Journal of Nutrition Education and Behavior

Abstract

The objectives of this study were to examine college students’ perception of their own weight, attitudes toward weight and dieting, beliefs and concerns about weight, knowledge of basic fat metabolism and factors influencing the amount of body fat, and the level of habitual physical activity. A written questionnaire was administered to 220 undergraduate students. The results showed that 75% of students had a BMI < 25, yet only 60% perceived themselves as having the “right weight”, 77% wanted to lose weight, and 74% reported a history of weight loss attempts. Weight-loss strategies used most frequently by students included energy and fat restrictions, exercise, meal skipping, and weight-loss supplements. Sixty-seven percent of students perceived themselves at low risk for future weight gain. Male and female students differed significantly in their perceived ability to control body weight and their reasons for weight concern. Students had misconceptions about the effects of macronutrients and certain foods on body fat deposition. Students generally were aware of biologic and lifestyle factors influencing body weight and fatness but had limited understanding of the mechanisms by which these factors exert their effects. Sixty percent of students reported engaging in at least 30 min/d of moderate-vigorous physical activity. BMI and academic major and year had fewer significant influences on students’ weight-related attitudes and beliefs than did gender.

Introduction

Overweight and obesity in the United States have reached epidemic proportions. While the prevalence of overweight peaks after the age of 35, many people experience major weight gain between the ages of 25 and 34 (Jeffery & French, 1997; Burke et al., 1996;
Williamson et al., 1990). Thus, individuals in their late teens and early twenties would benefit from education-based interventions to prevent excessive weight gain in adulthood.

Many factors contribute to the effectiveness of nutrition education interventions. One of these factors is the tailoring of educational messages to perceived needs of the intended audience (Kok et al., 1997). Thus, the first step in designing interventions to prevent weight gain in young adults is to learn about weight-related knowledge, attitudes, and practices of this population group (Gillespie, 1987). Some of these attitudes and practices have already been determined. A general consensus is that men and particularly women in their early twenties are excessively concerned with body weight and shape, often for appearance reasons (Bailey & Goldberg, 1989; Haberman & Luffey, 1998; Ritchie, 1998; Sciacca et al., 1991; McArthur & Howard, 2001; Drewnowski & Yee, 1987; Rodin, 1993). Studies showed that between 40% and 85% of college women perceived themselves overweight, although the majority of these women were not overweight based on the established standards (Sciaccia et al., 1991; Haberman & Luffey, 1998; Bailey & Goldberg, 1989). Drewnowski and Yee (1987) reported that 85% of female college freshmen wanted to lose weight.

Young individuals take various approaches to attain a desirable physique. Weight control methods most frequently used by this population group can be considered reasonable and include exercise and energy restriction (Serdula et al., 1999; Page & Fox, 1998). However, only 20% of 18-29 year-old men and women use both exercise and energy restriction simultaneously, whereas the majority use one method or the other (Serdula et al., 1999). A smaller proportion of young adults also resort to questionable weight control practices. Blanck et al. (2001) found that the prevalence of using nonprescription weight loss products among 18-34 year-old adults was 17.3% for women and 3.5% for men.

Young adults' perceptions of their own weight and attitudes toward weight-control practices have been studied; however, specific knowledge of this population group regarding weight management has not been described. Research findings for adolescents generally show poor understanding of concepts pertinent to weight control. Searles et al. (1986) reported that the majority of high school students had misconceptions about energy density of dietary fat, carbohydrate, and protein. Skinner & Woodburn (1984) found that 83% of adolescents recognized that reducing daily energy intake is an effective weight loss strategy;
however, when asked to choose from a list of foods and meals those that were low in calories, they did poorly. Findings for adolescents may have a limited application to young adults. Although it has not been shown, it could be expected that 20-25 year-olds would know more about nutrition and weight control than would 13-16 year-olds. At the present time there are not enough data to determine whether weight-related knowledge that young adults may have is sufficient to prevent them from excessive weight gain in adulthood.

The objectives of the present study were to a) examine college students’ perception of their own weight and attitudes toward weight and dieting, b) identify students’ concerns and beliefs about weight, c) determine their knowledge of basic fat metabolism and factors influencing body fatness, and d) assess students’ level of habitual physical activity. In addition, we compared weight-related beliefs, attitudes, knowledge, and practices between students of different genders, majors, and weight categories, and by year of college attendance.

Method

Subjects and study design

The Iowa State University Human Subjects Review Committee approved the study protocol. Any undergraduate college student between the ages of 18 and 30 years was eligible for participation. Two hundred and twenty students 20.7 ± 1.7 (mean ± SD) years of age from Iowa State University and the University of Northern Iowa volunteered to participate in the study.

Each student was asked to complete a questionnaire and a 3-day physical activity record (Appendix B). The questionnaire was administered in class with the instructor’s permission and consisted of 3 parts. The first part was designed to obtain demographic information, weight and height, information on students’ perceptions of their own weight, confidence in the ability to lose weight, risk of future weight gain, reasons for weight concern, attitudes toward dieting and weight-loss supplements, and a history of weight control practices. Self-reported weights and heights were used to calculate body mass index (BMI, kg/m²) for each student.
The second part of the questionnaire consisted of 15 multiple choice questions to test students' knowledge of three concepts: 1) the influence of genetics and age on body weight, 2) the ways in which the body uses fat, and 3) how diet can change body fat mass. Four knowledge scores were calculated for each student: 3 concept scores and the total score. Each concept was tested using 5 questions; thus, a concept score was a sum of points for the 5 corresponding answers. The total score was the sum of all points. A possible range of scores was from zero to 17 (2 questions had a 2-point answer). A reliability coefficient for the measure of knowledge (i.e., total score), using a test-retest estimate of reliability, was \( r(7) = .809, \ p < 0.05 \). Validity was determined by an expert panel of four nutrition professors.

The third part of the questionnaire was presented in the form of a newspaper advertisement of a weight loss supplement that included 6 true and false claims, such as “you can lose up to 7 pounds of fat during the first 2 weeks of using this product without dieting and strenuous exercise” and “carefully formulated supplements that have a right combination of proteins and amino acids can replace body fat with lean muscle tissue.” Students were asked to evaluate each statement by choosing one of the possible answers - “agree,” “disagree,” or “don’t know.” Students also were asked to indicate whether or not they would try this product if they needed to lose weight for any reason by choosing one of five answers ranging from “strongly disagree” to “strongly agree.”

The Bouchard 3-day physical activity records (Bouchard et al., 1983) designed to obtain self-recorded physical activity over 2 weekdays and 1 weekend day were distributed in class for 1 week later. The records were used to calculate the 3-day mean energy expenditure for each student. We also calculated the amount of time spent on physical activities of moderate-high intensity that included time spent on both structured exercise and non-structured physical activity (e.g., brisk walking between classes from one building to another, biking to campus, dancing in a night club, etc.) performed at moderate and high intensities.

**Statistical analysis**

The results were summarized using descriptive statistics including means ± standard deviations (SD) and frequencies. Data were checked for normality. Comparisons between groups were performed using an independent t-test, chi-square analysis, and one-way
analysis of variance with the Tukey test of multiple comparisons. Pearson correlations were calculated to determine associations between knowledge scores and BMI and between physical activity time and BMI. Significance was set at $\alpha < .05$. All analyses were performed with SPSS/PC statistical software (SPSS Inc, Chicago).

Results

Subjects

The sample was composed of 6.8% ($n = 15$) freshmen, 24.5% ($n = 54$) sophomores, 37.7% ($n = 83$) juniors, and 30.9% ($n = 68$) seniors. Fifteen percent ($n = 33$) of students were majoring in dietetics, 35.5% ($n = 78$) were exercise, physical education, or related majors, and 49.5% ($n = 109$) had majors not related to nutrition, exercise, or health ("other"). Ninety-eight percent of students were Caucasian and 72% were women.

Distribution of body mass index

Men had a higher mean BMI (25.5 ± 4) compared with women (22.6 ± 3; $p < 0.0001$; Figure 5.1). Seventy-five percent of all students had a BMI < 25, 19.5% had a BMI between 25.0 and 29.9, and 5.9% had a BMI ≥ 30. Overall, 51% of students had a BMI of 20 to 24. There were no differences in BMI when students were compared by major or year in college. Among students with a BMI between 25 and 29.9, 40% ($n = 6$) were women, and among those with a BMI ≥ 30, 54% ($n = 7$) were women.

Energy expenditure

Fifty-two percent of students (98 women and 17 men) completed and returned the 3-day activity records. Mean daily energy expenditure was higher in men 3,908 ± 844 kcal (16,335 ± 3,528 kJ) than women 2,918 ± 557 kcal (12,197 ± 2,328 kJ; $p < 0.001$). The amount of time spent on both moderate and vigorous structured exercise and non-structured physical activity averaged 54 min/d for women and 67 min/d for men and did not differ significantly between genders, majors, students with a BMI < 25 and ≥ 25, or by year in college. Among students who completed the records, 58% reported at least 30 min/d and 26% reported between 10 and 29 min/d of moderate to vigorous physical activity. Time spent on these activities was not correlated with BMI (Figure 5.2).
Weight concerns

More women than men were very concerned about their own weight. The primary reasons for concern were "weight affects how I feel about myself" for women and "weight is important for good health" for men ($\chi^2 = 3.1, df = 2, p = .001$; Table 5.1). The percentage of students being very concerned about weight did not differ between students with a BMI $< 25$ and $\geq 25$ and was 24 – 30%. The majority of students (67.5%) indicated that their chances of becoming overweight as they grow older were very or moderately low (Table 5.1). Seventy percent of students with a BMI $< 25$ and 58.3% of students with a BMI $\geq 25$ perceived their chance of becoming overweight to be low or moderately low. The perceived risk of becoming overweight did not differ between majors or by year in college.

Perception of own weight and desire to change weight

Students' perceptions of own weight and desire to change weight are presented in Table 5.1. Among students with a BMI $< 25$, 32% ($n = 43$) of women and 6.7% ($n = 2$) of men perceived themselves as being somewhat or a lot overweight (Figure 5.3). Overall, 88.6% of women and 48.4% of men wanted to lose weight, 29% of men wanted to gain weight, and only 8.9% of women and 22.6% of men did not want to change their body weight (Figure 5.4). Seventy-six percent of students with a BMI $< 25$ and 80% of students with a BMI $\geq 25$ wanted to lose weight. Thirty-nine percent of students with a BMI $< 25$ and 61% of students with a BMI $\geq 25$ wanted to lose more than five pounds. Among students with a BMI $\geq 25$, 53% of men and 90% of women perceived themselves as being somewhat or a lot overweight. Major or year in college did not have an effect on weight perception or desire to change weight.

Perceived confidence in the ability to lose weight

Students' perceived confidence in the ability to lose weight when needed is presented in Table 5.1. Men reported being more confident than women ($\chi^2 = 25, df = 2, p < 0.0001$). Confidence in the ability to lose weight differed between majors. Fifty percent of exercise, 42% of dietetics, and 28% of students in other majors perceived themselves as very confident, whereas 15% of exercise, 21% of dietetics, and 34% of other major students indicated being slightly or not confident ($\chi^2 = 12.9, df = 4, p = 0.012$). Although the confidence level did not differ between students with a BMI $< 25$ and a BMI $\geq 25$, it varied
by perceived weight status ($\chi^2 = 17.7$, df = 2, p < 0.0001). Among students who perceived themselves as thin or having normal body weight 45% and 16% were very and slightly confident, respectively. Among those who perceived themselves as overweight, 27% and 41% were very and slightly confident, respectively. Confidence was not associated with knowledge scores.

**Attempts and strategies to lose weight**

Fifty-five percent of women and 29% of men reported trying to lose weight at the time the questionnaire was administered. Overall, 74% of students (130 women and 32 men) attempted to lose weight at some point in life, on one or more occasions. More women than men reported ever attempting to lose weight ($\chi^2 = 23.3$, df = 2, p < 0.001; Table 5.1). Major had no statistically significant effect on weight-loss attempts: 88% of dietetics, 70.5% of exercise, and 71.5% of students in other majors reported ever trying to lose weight. Among female students, 81.3% with a BMI < 25 and 87.5% with a BMI ≥ 25 reported a history of weight loss attempts. Among male students, 33.4% with a BMI < 25 and 68.8% with a BMI ≥ 25 reported ever trying to lose weight. A history of weight-loss attempts did not vary by year of college attendance.

Men and women with a history of weight loss attempts (74% of the entire sample) reported the mean achieved weight loss of 4.4 ± 3.5 kg (range, 0.4 – 18.2 kg). Among these students the most frequently reported reasonable weight loss strategies were limiting the amount of food (reported by 79.8% of students), eating foods low in fat (74.8%), and exercising more than usual (74.8%). The most frequently reported questionable strategies were skipping meals (33%), taking over-the-counter weight loss supplements (20.2%), and fasting (9.8%). Limiting food consumption was more prevalent among women (84.6%) than men (60.6%; $\chi^2 = 9.4$, df = 1, p = 0.002) as was skipping meals (reported by 37% of women and 18.2% of men; $\chi^2 = 4.2$, df = 1, p = 0.041). The use of over-the-counter weight loss supplements was more prevalent among men (36.4%) than women (16.2%; $\chi^2 = 6.7$, df = 1, p = 0.01). The use of exercise to lose weight was common among both women (77%) and men (67%), as was the consumption of low-fat foods (reported by 78.5% of women and 66.7% of men). The total number of weight loss practices used was significantly higher for students with a BMI ≥ 30 compared with students having a BMI between 25 and 29.9 and students
having a BMI < 25 (p = 0.003). The use of dietary weight loss supplements varied by major and year in college. Among supplement users (n = 34), the majority (56%; 9 women and 10 men) were exercise majors followed by other majors (38%; 12 women and one man) and dietetics students (5.9%; 2 women). The use of supplements increased with the year in college: 17.6% of sophomores and 38 – 41% of juniors and seniors reported ever using supplements. Due to the small number of students who reported using supplements, we could not determine whether the differences between majors and by year of college attendance were statistically significant without violating the assumptions for a Chi-square test.

**Attitude toward weight loss supplements**

Forty percent of all students reported that they would try certain weight loss supplements. Among these students, the preferences were given to products that claim to control cravings for snacks and sweets (62% responded that they would try it), suppress appetite and hunger (48.8%), increase muscle mass (27.4%), burn body fat during sleep (22.6%), and burn body fat by increasing body temperature (15.5%). While only 18% of dietetics students indicated that they would try any of the products, 41 - 43% of other and exercise major students were willing to do so ($\chi^2 = 6.9, df = 2, p = 0.031$). In response to the advertisement of a weight-loss product, 74% of students indicated that they would not try the product, 19.6% were uncertain, and 6.4% responded that they would try it.

**Knowledge**

Proportions of the correct answers for all students are reported in Table 5.2. Since only one man was majoring in dietetics, men’s knowledge scores were compared to those of women of exercise and other majors. There were no differences between men and women in concept or total knowledge scores, although there were small significant differences in responses to a few individual questions. Mean total scores were 9.5 ± 2.2 points (or 56% of the maximum possible score of 17) for women and 8.9 ± 2.3 points (53%) for men. Overall, the level of knowledge was low for all major groups, including dietetics students whose mean score was only 60.5% of the maximum possible score. Knowledge scores were 10.27 ± 1.7 for dietetics students, 9.23 ± 2.2 for exercise students, and 9.17 ± 2.3 for students of other majors. Dietetics students’ knowledge score was higher than that of other majors (p = 0.03) and tended to be higher than that of exercise majors (p = 0.059). The difference between
dietetics and other majors was due to a higher score (p = 0.012) for the third concept (body fat can be changed through diet). The knowledge scores did not differ by year in college or BMI status. There was no association between the total knowledge scores and BMI.

Students’ responses to the weight loss advertisement were summarized separately from responses to the knowledge test. The majority of students (70% and higher) correctly identified true and false claims in the advertisement, such as “you can lose up to seven pounds of fat during the first two weeks without dieting and exercise,” “for most people, losing excess weight is difficult,” and “most people who use dietary supplements do not regain lost weight.” However, only 58.6% knew that “lean muscle mass is important for burning body fat,” 54% disagreed that “protein and amino acid supplements can replace body fat with muscle tissue,” and 26% disagreed that “the body transforms fat into energizing sugars, which are rapidly metabolized.”

**Discussion**

One objective of the present study was to examine students’ perceptions of their own weight and attitudes toward weight and dieting. We found that although two-thirds of students had a BMI < 25, only 60% rated themselves as having about the right weight, and 75% of all students wanted to lose weight regardless of their BMI status. Twice as many women than men wanted to lose weight. Women with a BMI in the upper half of the normal range were more likely to perceive themselves overweight than were women with a BMI in the lower half. Since BMI is not a sensitive indicator of body fatness, it is possible that some of the women correctly perceived themselves as overweight (Heymsfield et al., 2000). However, for the majority of these women, body weight perception was probably influenced by other factors. For instance, the mass media promoting extreme slimness as a “standard” of the ideal body is a well-known contributor to the formation of unrealistic expectations about one’s physical appearance and preoccupation with weight (Lindeman, 1999; Rodin, 1993). The normal process of physical maturation (i.e., widening of hips) that 18 year-old women undergo is confused with becoming fatter is another contributor to weight misperceptions (Page & Fox, 1998). Male students with a BMI < 25 seemed to have more accurate perceptions of their own weight compared with female students in the same BMI category.
Among men with a BMI ≥ 25, 50% perceived themselves as having normal weight and the other 50% perceived themselves as overweight. As in women, the insensitivity of BMI to body fatness could partially account for the discrepancy between reported and perceived weights in men with higher BMI. In other words, male students with larger lean mass might have been incorrectly categorized as overweight based on their BMI. Overall, our findings support previous reports that inaccurate perceptions of body weight are common among college students, although more so among women than men (Haberman & Luffey, 1998; Sciacca et al., 1991; Ritchie, 1988).

In the present study the prevalence of weight loss practices was high for both genders: 82% of female and 52% of male students reported trying to lose weight at least on one occasion. Consistent with previous findings for college students (Page & Fox, 1998; McArthur & Howard, 2001), the majority of students in this study chose reasonable weight loss strategies, such as limiting energy and fat intakes and increasing physical activity. Nonetheless, the reports of questionable practices including fasting, skipping meals, and diet pills were not uncommon. Men were twice as likely as women to use over-the-counter “fat-burning” and “muscle-building” supplements (e.g., Ripped Fuel, Xenadrine, Hydroxycut, Advocare, Metabolife) and women were twice as likely as men to skip meals. It should be noted that some of the supplements used by the students contained ephedra known to have adverse health effects ranging from increased blood pressure and heart rate to heart attack and death (Lagatus, 2000). About 40% of all students responded that they would try weight-loss supplements in the future if they needed to lose weight. The types of supplements that the students were willing to try included those claiming to suppress appetite, hunger, and cravings for snacks and sweets. The supplement choices, quite possibly, could reflect specific dietary struggles experienced by the students. The high proportion of students willing to try weight-loss supplements is a cause for concern. It suggests that these individuals could be persuaded by clever advertisement campaigns to purchase weight-loss products that are typically ineffective and perhaps dangerous.

Male and female students in this study differed significantly in their confidence to control body weight and their reasons for weight-related concerns. Less than 30% of female and over 60% of male students were very confident in their ability to lose body fat when
needed. Male students may be overestimating their ability to control the amount of body fat. Literature provides abundant examples of limited success of weight loss attempts and a high rate of recidivism following weight loss (National Task Force on Prevention and Treatment of Obesity, 1994). An unreasonably high level of confidence could potentially interfere with young men's participation in programs to prevent weight gain. Men were 1.5 times as likely as women to indicate that their primary reason for weight concern was health. Overall, only 43% of students attributed their weight concerns to health, perhaps due to a limited knowledge of the relationship between overweight and morbidity. Alternatively, the students might have been aware of the relationship, but felt that a desirable physique was more important to them at this stage of their lives.

The assessment of habitual physical activity showed that almost 60% of students who completed activity records met the current recommendation of at least 30 min/d of moderate to vigorous exercise on most days of the week (Pate et al., 1995). This finding suggests that students on a residential campus are considerably more active than the general population. According to the 1996 report of the Surgeon General, only 28% of American adults are adequately active (Physical Activity and Health, 1996). The students' high level of physical activity could be explained by a combination of factors. First of all, students may have more opportunities for routine physical activity, such as brisk walking between classes from one building to another and biking to and from campus, than other groups. Also, students have easy access to a variety of exercise facilities on campus. In addition, social interactions with peers provide students with opportunities and motivation to participate in team sports and recreational activities. We found no association between time spent on moderate-vigorous activities and BMI. Variability in type and intensity of physical activities could partially account for this finding. For example, weight training and an aerobic workout of moderate intensity would result in different energy expenditures per unit of time. We did not collect data on students' exercise preferences, which was a limitation of this study, as was a 52% return rate for physical activity records. Hence, the findings related to physical activity should be interpreted with caution.

College students' understanding of the effects of genetic, physiologic, and environmental factors on body fat metabolism has not been previously described. Our earlier
study that included a small self-selected sample of college female freshmen showed that a nutrition education course emphasizing human physiology and energy metabolism prevented weight gain for at least a year in students with a BMI > 25 (Matvienko et al., 2001). In light of that finding, one of the objectives of this study was to determine knowledge of basic fat metabolism in a larger and more diverse group of students. We found that the students were generally aware of the associations between biologic and lifestyle factors and body fatness; however, a lack of specific knowledge was evident. For instance, 85% of students agreed that most adults gain weight with age, but only about 35% knew that major weight gain is likely to occur between ages 20 and 35. Interestingly, while 85% of students were aware of age-related weight changes, 67% estimated their risk of future weight gain as very or moderately low. Research evidence shows that even the leanest individuals gain weight as they age. Lewis et al. (1997) reported that during a 5-year period, an average weight gain for 18-30 year-old adults was 3.9 – 4.8 kg and 4.6 – 7.8 kg for the leanest and heaviest men, and 3.2 – 4.8 kg and 4.7 – 7.0 kg for the leanest and heaviest women, respectively. Braddon et al. (1986) reported that by age 36, 43.3% of men and 32.6% of women had a BMI ≥ 25, compared to 12.6% of men and 15.8% of women at age 20. Thus, the majority of students in the present study did not perceive themselves at-risk for weight gain and, perhaps, underestimated their own risk of gaining excess weight.

Students demonstrated poor knowledge of body fat utilization and of the effects of diet and exercise on fat metabolism. About 50% of students did not know that muscle mass is important for losing body fat and that aerobic exercise of moderate intensity, such as brisk walking, utilizes more body fat per unit time than does anaerobic activity. Over 80% of students disagreed that an effective weight-loss diet has to limit energy intake. This finding indicates that the students’ interpretation of a weight-loss diet differs from that of health professionals and, perhaps, is influenced by infomercials and other advertisements on fad diets and products claiming to produce weight loss without energy restriction. Students had misconceptions about the effects of dietary fat and carbohydrate on body fat deposition. Almost 80% of students believed that dietary carbohydrate rather than fat is converted to body fat and at least 30% believed that overeating high fat foods made with “good” fats, such as vegetable oils and margarine, does not cause weight gain. Mitchell (1990) and Auld et al.
(1991) pointed out that misconceptions about diet are very common among the general public and require special attention in nutrition education programs.

We did not find a significant association between knowledge scores and BMI partly because of the small variations in both. About 85% of all students fell into the middle tertile of knowledge score. Similarly, 75% of students had a BMI < 25. In addition, the lack of association could be due to limitations of the knowledge test which was short (only 15 questions) and did not cover all areas pertinent to effective weight gain prevention. For instance, there were no questions about food composition, dietary guidelines, or the effects of overweight on health. In order to increase the number of participants in the study, the test was administered in class. The instructors were willing to spare only a small portion of class time for the study; thus, we had to limit the number of questions.

Although we observed a number of differences between male and female students, there were only a few significant differences between students of different majors, BMI categories, or years of college attendance. To our knowledge, this is the first study that compared students of different majors. Dietetics students were least likely to use weight-loss products compared with exercise and other students. Both dietetics and exercise students had a higher level of confidence in their ability to lose body fat when needed than did other students. Also, dietetics majors had better knowledge about the effects of diet on body weight than did other majors. Male and female students with a BMI ≥ 30 engaged in weight-loss practices more frequently compared with students with a BMI of 25 – 29.9 or < 25. Weight-related attitudes, beliefs, and knowledge did not differ by the number of years in college.

In addition to limitations of the knowledge test and the instrument used to collect physical activity data as previously discussed, this study was limited by the use of volunteers, lack of ethnic diversity in the study population, reliance on self-reported weights and heights, and small numbers of dietetics students and freshmen. Despite these limitations, the results of the present study demonstrated that the undergraduate students, particularly women, had inaccurate perceptions of their own weight and frequently engaged in both reasonable and questionable weight-control practices. Students appeared to underestimate their risk of future weight gain. Furthermore, male students generally expressed a high level of confidence in their ability to control the amount of body fat, although the reasons for such high confidence
are not clear. Most students had a general awareness of the connection between biologic and environmental factors and body fatness but did not know how these factors exert their effects. Male and female students differed in their weight-related attitudes, beliefs, and practices. Major, BMI category, and academic classification had smaller influences on students' attitudes and beliefs than did gender.

Implications for Research and Practice

College students, without doubt, would benefit from education-based interventions to prevent excess weight gain. Such interventions should be designed to help students form realistic perceptions of their own weight and their risk of weight gain, to improve students' understanding of the importance of maintaining healthy weight throughout adulthood for health rather than appearance reasons, and to decrease students' misconceptions about macronutrients and their role in body fat deposition. The intervention content could include specific information about the effects of genetics and age on body weight, macronutrient metabolism, limited effectiveness and potential danger of questionable weight control practices, and mechanisms by which biologic and lifestyle factors influence body weight. The overall purpose of education-based interventions to prevent obesity should be to provide students with tools for selecting appropriate dietary practices related to weight management. To increase success of interventions, health professionals need to tailor information to the needs of both men and women. In other words, interventions should be designed to address differences in attitudes, beliefs, and practices regarding weight between male and female students.

References


Table 5.1. Perception by college women and men of own weight, weight concerns, and confidence in the ability to lose weight.

<table>
<thead>
<tr>
<th>Perception of own weight and desire to change weight</th>
<th>Women (n = 158)</th>
<th>Men (n = 62)</th>
<th>Total (n = 220)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I think I am</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Too thin</td>
<td>1.3 (2)</td>
<td>11.3 (7)</td>
<td>4.1 (9)</td>
</tr>
<tr>
<td>About the right weight</td>
<td>58.2 (92)</td>
<td>58.1 (36)</td>
<td>58.2 (128)</td>
</tr>
<tr>
<td>Somewhat overweight</td>
<td>36.1 (57)</td>
<td>27.4 (17)</td>
<td>33.6 (74)</td>
</tr>
<tr>
<td>A lot overweight</td>
<td>4.4 (7)</td>
<td>3.2 (2)</td>
<td>4.1 (9)</td>
</tr>
<tr>
<td><strong>I want to improve my weight by</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Losing 1-5 lbs</td>
<td>37.3 (59)</td>
<td>21.0 (13)</td>
<td>32.7 (72)</td>
</tr>
<tr>
<td>Losing &gt; 5 lbs</td>
<td>51.3 (81)</td>
<td>27.4 (17)</td>
<td>44.5 (98)</td>
</tr>
<tr>
<td>Gaining 1-5 lbs</td>
<td>1.9 (3)</td>
<td>11.3 (7)</td>
<td>4.5 (10)</td>
</tr>
<tr>
<td>Gaining &gt; 5 lbs</td>
<td>0.6 (1)</td>
<td>17.7 (11)</td>
<td>5.5 (12)</td>
</tr>
<tr>
<td>Don't want to change</td>
<td>8.9 (14)</td>
<td>22.6 (14)</td>
<td>12.7 (28)</td>
</tr>
<tr>
<td><strong>Weight concerns</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I am concerned about my weight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very concerned</td>
<td>28.5 (45)</td>
<td>17.7 (11)</td>
<td>25.5 (56)</td>
</tr>
<tr>
<td>Somewhat concerned</td>
<td>39.2 (62)</td>
<td>30.6 (19)</td>
<td>36.8 (81)</td>
</tr>
<tr>
<td>Slightly concerned</td>
<td>30.4 (48)</td>
<td>33.9 (21)</td>
<td>31.4 (69)</td>
</tr>
<tr>
<td>Not concerned at all</td>
<td>1.9 (3)</td>
<td>17.7 (11)</td>
<td>6.4 (14)</td>
</tr>
<tr>
<td><strong>Why are you concerned about your weight?</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>It is important to look good</td>
<td>9.8 (14)</td>
<td>15.5 (9)</td>
<td>11.5 (23)</td>
</tr>
<tr>
<td>It is important for good health</td>
<td>37.1 (53)</td>
<td>55.2 (32)</td>
<td>42.5 (85)</td>
</tr>
<tr>
<td>It affects how I feel about myself</td>
<td>52.4 (75)</td>
<td>24.1 (14)</td>
<td>44.0 (88)</td>
</tr>
<tr>
<td>Other people tell me to lose/gain</td>
<td>0</td>
<td>3.4 (2)</td>
<td>1.0 (2)</td>
</tr>
<tr>
<td>It affects my athletic performance</td>
<td>0.7 (1)</td>
<td>1.7 (1)</td>
<td>1.0 (2)</td>
</tr>
<tr>
<td><strong>My chance of becoming overweight as I get older</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very low</td>
<td>31.7 (45)</td>
<td>42.6 (26)</td>
<td>35.0 (71)</td>
</tr>
<tr>
<td>Moderately low</td>
<td>31.7 (45)</td>
<td>34.4 (21)</td>
<td>32.5 (66)</td>
</tr>
<tr>
<td>About 50%</td>
<td>20.4 (29)</td>
<td>11.5 (7)</td>
<td>17.7 (36)</td>
</tr>
<tr>
<td>Moderately high</td>
<td>14.1 (20)</td>
<td>9.8 (6)</td>
<td>12.8 (26)</td>
</tr>
<tr>
<td>Very high</td>
<td>2.1 (3)</td>
<td>1.6 (1)</td>
<td>2.0 (4)</td>
</tr>
<tr>
<td><strong>Attempts to lose weight</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In the past, I have tried to lose weight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>17.7 (28)</td>
<td>48.4 (30)</td>
<td>26.4 (58)</td>
</tr>
<tr>
<td>Once</td>
<td>20.3 (32)</td>
<td>19.4 (12)</td>
<td>20.0 (44)</td>
</tr>
<tr>
<td>More than once</td>
<td>62.0 (98)</td>
<td>32.3 (20)</td>
<td>53.6 (118)</td>
</tr>
<tr>
<td><strong>Perceived confidence in the ability to lose weight</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>If I had to lose weight, I am confident I know how to do it successfully</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very confident</td>
<td>27.8 (44)</td>
<td>62.9 (39)</td>
<td>37.7 (83)</td>
</tr>
<tr>
<td>Somewhat confident</td>
<td>40.5 (64)</td>
<td>27.4 (17)</td>
<td>36.8 (81)</td>
</tr>
<tr>
<td>Slightly confident</td>
<td>29.7 (47)</td>
<td>9.7 (6)</td>
<td>24.1 (53)</td>
</tr>
<tr>
<td>Not confident at all</td>
<td>1.9 (3)</td>
<td>0</td>
<td>1.4 (3)</td>
</tr>
</tbody>
</table>

*a* Due to missing data, n = 143 for women and n = 57 for men.

*b* Due to missing data, n = 142 for women and n = 61 for men.
Table 5.2. Students' (n = 220) responses to the knowledge test.

<table>
<thead>
<tr>
<th>Concepts and questions</th>
<th>Correct answers (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Concept 1: Genetics and age influence body weight</strong></td>
<td></td>
</tr>
<tr>
<td>Q 1. Most adults gain weight as they get older</td>
<td>85.0</td>
</tr>
<tr>
<td>Q 2. Weight gained in teen years is likely to persist into adulthood</td>
<td>48.6</td>
</tr>
<tr>
<td>Q 3. A major weight gain occurs between 20 and 35 years</td>
<td>34.1</td>
</tr>
<tr>
<td>Q 4. Genes determine how the body works and looks</td>
<td>68.6</td>
</tr>
<tr>
<td>Q 5. Inherited weight can be maintained through diet and exercise</td>
<td>93.6</td>
</tr>
<tr>
<td><strong>Concept 2: The body has specific ways of utilizing fat on a daily basis</strong></td>
<td></td>
</tr>
<tr>
<td>Q 1. Dietary fat is deposited as body fat</td>
<td>21.4</td>
</tr>
<tr>
<td>Q 2. During sleep about 50% of energy that the body uses is from fat</td>
<td>13.6</td>
</tr>
<tr>
<td>Q 3. Body fat utilization during exercise depends on many factors</td>
<td>62.3</td>
</tr>
<tr>
<td>Q 4. In 20 minutes, walking burns more fat calories than some other activities</td>
<td>46.8</td>
</tr>
<tr>
<td>Q 5. Over 24 hours, the body uses fat primarily for low-intensity activities</td>
<td>51.6</td>
</tr>
<tr>
<td><strong>Concept 3: Body fat can be changed through diet</strong></td>
<td></td>
</tr>
<tr>
<td>Q 1. It takes weeks to months to lose body fat</td>
<td>85.5</td>
</tr>
<tr>
<td>Q 2. Once body fat is gained it is difficult to lose</td>
<td>66.4</td>
</tr>
<tr>
<td>Q 3. During a 7-day fasting, the body uses more than 70% of energy from fat</td>
<td>28.2</td>
</tr>
<tr>
<td>Q 4. An effective weight loss diet means that energy intake must be limited</td>
<td>28.2</td>
</tr>
<tr>
<td>Q 5. Weight gain is caused by overeating any kind of fatty foods</td>
<td>37.7</td>
</tr>
</tbody>
</table>

*For complete questions, refer to Appendix B.*
Figure 5.1. Body mass index distribution in men (n = 68; top graph) and women (n = 158; bottom graph).
Figure 5.2. Correlation between the amount of time spent on both structured exercise and non-structured physical activity of moderate-high intensity and BMI. After excluding an outlier (exercise time 335 min/d), $r = .135$, $p = 0.152$, $n = 114$. 
Figure 5.3. Women (n = 134) and men (n = 30) with a BMI < 25 grouped by perceptions of their own weight. A box is the middle 50%, line in box plot is the median, and vertical bars indicate the range.
"I want to improve my weight"

Figure 5.4. Desire to change weight by gender.
CHAPTER 6: GENERAL CONCLUSIONS

Although studies presented in this dissertation varied in their design and specific objectives, their common purpose was to identify and test prevention strategies to lower the risk of atherosclerosis and obesity in young adults. The first study examined the effect of consuming a phytosterol-supplemented food on serum cholesterol concentrations in young hypercholesterolemic men. This study was unique for a number of reasons. First of all, phytosterols were added to nutritious lean ground beef, whereas previous studies used margarine, spread, or butter — products that are energy-dense and poor sources of micronutrients — as a vehicle for delivering phytosterols. Second, the effect of phytosterol supplementation was examined in young rather than middle-aged and older individuals. Third, responsiveness to the phytosterol treatment was compared between individuals with and without family history of heart disease. Fourth, changes in lipoprotein subclasses following the phytosterol treatment were determined. The main conclusion that can be drawn from this study is that ground beef supplemented with phytosterols is an effective functional food for lowering cholesterol concentrations in young men who are at increased risk of developing advanced atherosclerosis. Further research is needed to identify other foods suitable for phytosterol supplementation so that consumers have a variety of products from which to choose. Also, long-term safety of phytosterol-supplemented products should be evaluated.

The second study examined the effect of a science-based college-level course on changes in knowledge and body weight of young women. A unique component of this study was the course content. Although the course was designed with the intention to prevent weight gain in students during their freshman year, it did not provide directions on how to lose weight. Instead, students learned about basic principles of human physiology, energy metabolism, nutrient utilization, and the interaction between genetic factors, food composition, diet and physical activity, and energy use. Students who completed the course were able to maintain their baseline weight for one year. The effect of the course was most profound on women with a BMI greater than 24 who were at increased risk of gaining excess weight. In the broader view, this study demonstrated that high-intensity education is an
effective approach to prevent unwanted weight gain short-term. Limitations of the study include the small number of subjects and a relatively short duration. Hence, further research is warranted to confirm the effectiveness of high-intensity education in preventing weight gain in a larger population of women.

The third study examined college students’ attitudes, beliefs, knowledge, and behavior regarding weight management. Theoretical models suggested that education-based interventions aimed at influencing behavior should be tailored to specific needs of the intended audience. Because young adults should be a primary target audience for interventions to prevent obesity, it is important to know their specific weight-related needs and concerns. Two major conclusions emerged from this study. First, male and female students were significantly different in their beliefs and attitudes toward weight control. Second, students had limited knowledge of mechanisms by which genetic and environmental factors influence body fatness. Thus, nutrition educators face many challenges, including designing appropriate education-based interventions to address the needs of both young women and men and determining whether the young individuals’ knowledge about weight is sufficient to prevent weight gain later in life.
APPENDIX A:
CONSENT FORM AND DATA COLLECTION INSTRUMENTS
FOR STUDY 1
A SINGLE DAILY DOSE OF SOYBEAN PLANT STEROLS IN GROUND BEEF
DECREASES SERUM TOTAL AND LOW DENSITY LIPOPROTEIN CHOLESTEROL
IN YOUNG, MILDLY HYPERCHOLESTEROLEMIC MEN
CONSENT FORM

Consent to participate in a research study

Study Title: A single daily dose of soybean plant sterols in ground beef decreases serum total and low-density lipoprotein cholesterol in young, mildly hypercholesterolemic men.

Conducted by: [Investigators’ names and contact information]

Purpose
You are being asked to participate in a research study. The title of the study is “Effectiveness of a Novel Plant Sterol to Improve Serum Lipid Profiles in Young Adults.” To be eligible for participation, you must be a college male, 18 to 30 years of age, a non-smoker, with a body mass index between 20 and 26 and have mildly elevated blood cholesterol. The objective of this study is to determine if consumption of beef products containing plant sterol “X” reduces blood cholesterol, and maintains HDL cholesterol in young men with elevated blood cholesterol.

Procedures
If you decide to volunteer, we will assign you to either the experimental or control group. We will take blood samples to measure your total cholesterol, LDL cholesterol, HDL cholesterol, triacylglycerol, and bile acid concentrations. We will also estimate your dietary intake of nutrients. To estimate your normal dietary intake, you will be asked to sit down and discuss what you normally eat with trained nutrition students. This discussion will take approximately 45 minutes. You will be asked questions about what types of food you eat and how often you eat each food. You will be provided with lunches in the Human Metabolic Unit dining room Monday through Friday for a period of four weeks. Each lunch will include a beef entrée. The beef may contain vegetable material (i.e., filler). You will be required to eat all the food provided. We will also ask that you follow dietary instructions: minimize your meat intake during the week, maintain your normal meat intake on weekends and minimize your intake of alcoholic beverages.

Alternatives
Whether you participate in this study or not is completely up to you. If you decide not to participate this decision will not have any adverse consequences.

Risks
This study does not involve any risk of harm greater than you normally experience in everyday life. The plant sterol that may be added to your lunch is a natural component in vegetables and is not a threat to your health. Providing blood samplers will be similar to what is done at a physical’s exam at your physician office.

Benefits
You may or may not personally benefit from this study. It is likely that consumption of the plant sterol will be beneficial by lowering blood cholesterol. If desired, you will be given
results of your blood lipid profile and dietary analysis of nutrient intakes that reflect whether or not your intakes of these nutrients are higher than or lower than recommended.

Confidentiality
Research records of your participation in the study will be kept confidential and will not be released without your written permission. Information about you from this study will be identified by a code number known only to you and to the study personnel. The code will be stored in a secure location with access only by study personnel. By signing this document you allow us to make just the results (not your name or other identification) available to the sponsor of the study and publication in a peer-reviewed science journal. Any information obtained in connection with this study will be used in a manner that does not publicly disclose your identity and will be kept confidential. Absolute confidentiality cannot be guaranteed, since research documents are not protected from subpoena.

Costs / Compensation
You will be paid $250 for participation upon completion of the study. Additionally, the meals, blood lipid analysis, and dietary assessment will be free of charge.

Right to Refuse or Withdraw
You may refuse to participate or may change your mind about being in the study and quit after the study has started. Participation in this study is entirely voluntary and you may refuse to participate or withdraw from the study at any time. However, if you do not complete the study, you will not be paid for your participation.

Principal Investigator’s Disclosure of Personal and Financial Interests in the Research Study and Sponsor
The investigators have no financial interest in this research and will not benefit monetarily from this investigation.

Questions
If you have any questions, contact [Investigator’s’ names and contact information].
NUTRITION AND HEALTH QUESTIONNAIRE

Date: __________________________
Project ID: ______________________
Name: __________________________
Date of birth: _____________________
SS#: ____________________________

1. Academic classification:
   ___ 1. Freshman
   ___ 2. Sophomore
   ___ 3. Junior
   ___ 4. Senior
   ___ 5. Graduate student
   ___ 6. Other

2. College of major:
   ___ 1. Agriculture
   ___ 2. Business
   ___ 3. Design
   ___ 4. Education
   ___ 5. Engineering
   ___ 6. FCS
   ___ 7. Liberal arts and sciences
   ___ 8. Vet Med
   ___ 9. Undeclared

3. Ethnic background:
   ___ 1. Caucasian
   ___ 2. African American
   ___ 3. Hispanic
   ___ 4. Native American
   ___ 5. Asian
   ___ 6. Other (Specify _________________________)

4. Present residence:
   ___ 1. Residence hall
   ___ 2. Fraternity
   ___ 3. Married student housing
   ___ 4. Parent’s home
   ___ 5. Apartment (with spouse)
   ___ 6. Apartment (with roommates)
   ___ 7. Apartment (alone)
5. Have you ever been diagnosed with:
   ___1. Diabetes
   ___2. Consistently high blood pressure, > 140/90
   ___3. Heart Disease
   ___4. Ulcers
   ___5. Gastrointestinal disorder (Specify:__________________________)
   ___6. High blood lipids
   ___7. Thyroid disorders (Specify:__________________________)
   ___8. Hyperglycemia

6. Do you have a family history of:
   ___1. High blood lipids
   ___2. Diabetes
   ___3. Cardiovascular disease

7. Have you been diagnosed by a medical doctor with:
   ___1. Food allergy (Specify:__________________________)
   ___2. Other allergies (Specify:__________________________)
   ___3. Asthma

8. Are you presently taking any:
   ___1. Over the counter medications. (Specify:__________________________)
   ___2. Prescription medications (Specify:__________________________)

9. Do you currently use tobacco in any form?  No__ Yes__

10. Have you ever smoked?  No__ Yes__
    If yes specify  started:__________________________  stopped:__________________________

11. In the past 12 months, has your weight
    Increased  No__ Yes__ (If yes, how much__________________________)
    Decreased No__ Yes__ (If yes, how much__________________________)

12. Are you currently trying to lose weight?  No__ Yes__

13. Are you on a special diet?  No__ Yes__
    If yes specify: _______________________________

14. Are there any foods you normally avoid?  No__ Yes__
    If yes specify: _______________________________

15. Are there any foods you particularly like and eat frequently?  No__ Yes__
    If yes specify:
    What foods:__________________________
    How often you eat them:__________________________
    How much:__________________________
16. How often do you consume red meat in a typical week?

- 1-2 times/week
- 2-3 times/week
- 5-6 times/week
- >6 times/week

17. Do you take nutrition supplements?  No___ Yes___
   If yes specify:
   Vitamin & mineral (what kind?)________________________
   Vitamin (what kind?)________________________
   Mineral (what kind?)________________________
   Other (including protein powder)________________________

18. Are you taking any anabolic steroids?  No___ Yes___
   If yes specify________________________

19. Do you exercise?  No___ Yes___
   If yes, specify the amount (days/week and minutes/session):
   Aerobic________________________
   Weight lifting________________________
   Team sports (specify)________________________
   Other (specify)________________________

20. Would you describe your typical workout as:
   - 1. Light
   - 2. Light-moderate
   - 3. Moderate
   - 4. Moderate-heavy
   - 5. Heavy

21. In a typical week, how many do you consume of:
   Beer________ Hard liquor or mixed drinks_______ Wine________
FAMILY HISTORY OF HEART DISEASE AND DIABETES

Please, answer the following questions:

1. Have any of your blood-related relatives (not in-laws) been diagnosed with any form of cardiovascular (heart) disease before the age of 55?
   
   YES  NO

   If YES, which relative(s)? ___________________________

2. Have any of your blood-related relatives been diagnosed with high blood lipids or cholesterol levels before the age of 55?

   YES  NO

   If YES, which relative(s)? ___________________________

3. Have any of your blood-related relative(s) been diagnosed with
   
   a. Type I Diabetes (insulin-dependent)

   YES  NO

   If YES, which relative(s)? ___________________________

   At what age was he/she diagnosed? __________________

   b. Type II Diabetes (non-insulin dependent)

   YES  NO

   If YES, which relative(s)? ___________________________

   At what age was he/she diagnosed? __________________
APPENDIX B:
CONSENT FORM AND DATA COLLECTION INSTRUMENTS
FOR STUDY 3
COLLEGE STUDENTS' PERCEPTION OF OWN WEIGHT, ATTITUDES TOWARD DIETING, AND KNOWLEDGE AND BELIEFS ABOUT BODY WEIGHT
CONSENT FORM

Dear Student,

We would like you to be a part of our research study. The purpose of this study is to learn about students' beliefs related to lifelong weight management. All we ask you to do is to answer questions in the attached survey. There are no right or wrong answers. We are interested in your honest feelings and beliefs. The survey will take about 10 to 15 minutes of your time. You do not need to give us your name but if you do, your confidentiality and anonymity are absolutely assured. We will assign you a project identification number and your name will be removed from the survey as soon as we process it. Your survey will be stored in a secure location with accessed only by study personnel. By signing this consent form you will allow us to make just the results (not your name or other identification) available for publication in a science journal.

You participation in this study is entirely voluntarily and nonparticipation will not affect you in any way.

This research is under the direction of [Name of investigators].

[Contact information]

I have read the description above and agree to participate in this study, please assign me a project ID number.

Your name__________________________________________

Signature_________________________ Date__________
SURVEY

We would like you to answer questions about weight-related issues. There are no right or wrong answers. We are interested in your honest feelings about these issues. By filling out this questionnaire, you will give us your permission to use the information for research purposes. Your confidentiality and anonymity are absolutely assured. We will assign you a project identification number and your name will be removed from the survey as soon as we process it. Participation or nonparticipation will not affect your class evaluation and grades in any way.

Your academic classification:

___ Freshman
___ Sophomore
___ Junior
___ Senior
___ Grad student
___ Other, specify: ____________________

Your major (occupation): ____________________

Ethnic background:

___ Caucasian
___ African American
___ Hispanic
___ Native American
___ Asian
___ Other, specify: ____________________

Present residence:

___ Residence hall
___ Fraternity/sorority
___ Married student housing
___ Parents’ home
___ Apartment with spouse
___ Apartment with roommates
___ Apartment alone
___ Other, specify ____________________

Date of birth: _______________

Your current weight in pounds __________ or kilograms __________

Your current height in feet/inches __________ or meters/centimeters __________

How often do you weigh yourself each month? ______
How many of the following courses have you taken in college?

- Nutrition
- Exercise
- Health
- Physiology
- Biology
- Chemistry

Do you currently smoke?

- No
- Occasionally (one cigarette a month or less frequently)
- Regularly (more than two cigarettes a month)

Are your parents overweight or obese?

- No
- One of my parents is overweight/obese
- Both parents are overweight/obese

Next set of questions is about your body weight and dieting habits. Please notice that some questions require more than one answer. It is important to us that you answer all the questions.

1. (Circle ONE) I think that I am
   a. too thin
   b. about the right weight
   c. somewhat overweight
   d. a lot overweight

2. I am currently trying to lose weight
   a. yes
   b. no

3. (Circle ONE) In the past, I have tried to lose weight
   a. never → skip questions 4a and 4b, go to question 5
   b. once → go to questions 4a
   c. more than once → go to question 4a

4. (Circle ALL that apply) I have tried (or currently trying) to lose weight by
   a. limiting amount of food I eat
   b. eating foods low in fat
   c. skipping meals
   d. fasting for few days
   e. exercising more than I usually do
   f. taking weight loss supplements available over-the-counter or through Internet. Which one(s)?

__________________________________________
g. taking prescription diet pills. Which one(s)?

h. participating in a weight loss program. What program?

i. using popular diet plan(s). What diet plan?

j. other, specify:

5. What is a maximal weight loss you have ever achieved? ______ lbs or ______ kg

6. Overall, I am concerned about my body weight and shape

   very concerned          somewhat concerned          slightly concerned          not concerned

7. (Circle ONE) I am primarily concerned about my weight because:
   a. weight is important to look good
   b. weight is important for good health
   c. my weight affects the way I feel about myself
   d. other people tell me that I need to lose/gain weight
   e. other, please specify:

8. (Circle ONE) I would like to improve my body weight and shape by:
   a. losing 1-5 lb
   b. losing > 5 lb
   c. gaining 1-5 lb
   d. gaining > 5 lb
   e. I don't want to change my body weight.

9. (Circle ALL that apply) If I ever needed to lose weight, I would do it by:
   a. limiting amount of food I eat
   b. eating foods low in fat
   c. skipping meals
   d. fasting for few days
   e. exercising more than I usually do
   f. taking weight loss supplements available over-the-counter
   g. taking prescription diet pills
   h. participating in a weight loss program
   i. using popular diet plan(s) such as high-protein diet, or high carbohydrate diet
   j. other

10. If I had to lose weight, I am confident I know how to do it successfully.

   very confident          somewhat confident          slightly confident          not confident

11. (Circle ONE) I think my chance of becoming overweight as I get older is:

   none    small    small to moderate    about 50%    moderate to high    very high
   (0%)   (0-15%)  (20-40%)        (50-75%)    (>75%)
KNOWLEDGE TEST

The questions are grouped by concepts.

Concept 1: Genetics and age influence body weight

1. Most adults including lean individuals gain weight as they get older.
   a. strongly agree
   b. agree
   c. don't know
   d. disagree
   e. strongly disagree

2. A person who is overweight as a teenager will most likely be overweight for the rest of his/her life.
   a. strongly agree
   b. agree
   c. don't know
   d. disagree
   e. strongly disagree

3. If I get fatter as I get older, I will most likely gain this fat:
   a. between 20 and 35 years
   b. between 35 and 45 years
   c. between 45 and 60
   d. after 60
   e. don't know

4. A gene is DNA that I inherited from my parents that determines:
   a. only how I look
   b. how my body works and looks
   c. my lifestyle and emotions
   d. what I do, say, and think
   e. none of the above
   f. don't know

5. Despite my inherited body weight and shape, I can maintain my body weight by eating healthy and exercising.
   a. strongly agree
   b. agree
   c. don't know
   d. disagree
   e. strongly disagree
Concept 2: The body has specific ways of utilizing fat on a daily basis

1. After I eat meals my body will normally deposit fat within 24 hours. This fat mostly comes from:
   a. dietary fat
   b. carbohydrate
   c. protein
   d. don’t know

2. Overnight (during sleep), my body will get:
   a. more than 70% of needed energy from body fat
   b. about 50% of needed energy from body fat
   c. more than 70% of needed energy from carbohydrate and protein
   d. about 50% of needed energy from protein
   e. don’t know

3. The amount of body fat that is used during physical activity depends upon:
   a. how long and how hard I exercise
   b. how much oxygen I breathe during the activity
   c. the diet that I normally eat
   d. all of the above
   e. don’t know

4. I would lose more body fat during 20 minutes of:
   a. weight lifting
   b. playing a team sport
   c. brisk walking
   d. watching TV
   e. don’t know

5. Over a 24-hour period, fat in my body is used mostly to provide energy:
   a. for daily activities: standing, sitting, resting, and sleeping
   b. for food digestion one to two hours after each meal
   c. during exercise
   d. don’t know

Concept 3: Fat mass can be changed through diet

1. If I have to lose body fat, the fastest I can do it is:
   a. within 24 to 48 hours
   b. within weeks to months
   c. don’t know
2. It is okay if I gain extra body fat as I get older because I can readily lose body fat and return to my previous weight.
   a. strongly agree
   b. agree
   c. don't know
   d. disagree
   e. strongly disagree

3. When not eating for 7 days, my body will get:
   a. more than 70% of needed energy from body fat
   b. about 50% of needed energy from body fat
   c. more than 70% of needed energy from carbohydrate and protein
   d. about 50% of needed energy from protein
   e. don't know

4. An effective weight loss diet means that energy intake must be limited.
   a. strongly agree
   b. agree
   c. don't know
   d. disagree
   e. strongly disagree

4. (Circle ALL that apply) I am more likely to gain body fat if I overeat:
   a. high-fat foods such as fatty meat, butter, cheese, and pies and cakes
   b. high-carbohydrate foods such as pasta, bread, potatoes, and cereal
   c. high-carbohydrate foods such as candy, fruit juice, non-diet soda, cookies
   d. high-fat foods including cakes and pies that are made with "good" fats such as vegetable oils, and margarine
   e. don't know
Protein Nite Loss (PNL) is a nutritional supplement formulated to help the body to melt excessive body fat. PNL is a natural product that does not stimulate, starve or trick the body into unhealthy weight loss. The body has the ability to breakdown its reserves of stored fat by transforming fat into energizing sugars, which are rapidly metabolized (A D ?). PNL greatly enhances this process. In fact, you can lose up to 7 pounds of fat during the first two weeks of using PNL without dieting and strenuous exercise (A D ?).

PNL also supplies the body with important nutrients to increase lean muscle mass. Lean muscle mass is important in burning fat (A D ?). Carefully formulated supplements such as PNL that contain a right combination of proteins and amino acids can replace body fat with lean muscle tissue (A D ?).

For most people, losing excess body weight is difficult (A D ?). If you are one of those people, you will benefit from using PNL, which will improve your metabolism. Once you have lost unwanted pounds, you can discontinue taking PNL because your weight loss is permanent. In fact, most people who use dietary supplements and weight loss diets do not regain lost weight (A D ?).

Because PNL is safe and effective (not requiring dangerous caloric restrictions), many chiropractic, naturopathic and medical doctors are currently utilizing similar products personally and professionally as a part of their total health care program.

If you were to lose weight and/or build muscle mass, would you try this supplement?

- strongly disagree
- disagree
- don’t know
- agree
- strongly agree
BOUCHARD THREE-DAY PHYSICAL ACTIVITY RECORD


Please, read all instructions carefully before you start working on this assignment.

Part I: Recording physical activities

1. Record daily physical activities over 3 days: 2 weekdays and 1 weekend day.
2. Fill in the dates and your name where required.
3. How to choose days
   a. Choose days when you do your typical activities. Running a marathon is not a typical activity.
   b. It is better to choose, for example, Monday and Tuesday rather than Monday and Wednesday because your class schedule and daily activities are likely to be different on the two consecutive days.
   c. If you exercise every other day, choose one day when you exercise and one day when you don’t exercise.
   d. If you exercise four or more times a week, choose a weekend day when you exercise. If you exercise fewer than 4 times a week, choose a weekend day when you don’t exercise.
   e. If you do different types of exercise on different days of the week, choose two days when you do two different activities; for example, weight lifting and aerobic exercise or team games and swimming, etc.
4. The 3-day record is divided into 15-min intervals. Activities are quantified on a 1-to-9 scale of energy cost. Study the example below. It will help you understand how to enter your activities.
5. Each box should be filled with the number (code) corresponding to the activity which you have carried out during the 15 minute period. ALWAYS REFER TO THE ACTIVITY CODE LIST FOR THE PROPER CODING.
6. If an activity is carried out over a long period (e.g., sleeping) you can draw a continuous line in the rectangular boxes which follow until such a time when there is a change in activity (see the example).
7. There should be only one number per box: write in the activity which was carried out the most during the given period.
8. Balance certain activities. For example, for 30 minutes of volleyball, fill in 1 x 15 min “standing” and 1 x 15 min of “intense physical activities.”
9. If you don’t know how to encode a certain activity or have any other questions related to this assignment, ask your instructor.
Part II: Calculations
1. Median energy cost in kilocalories per kilogram per 15-min intervals is used to compute daily energy expenditure.
2. Following the example below, calculate your each day and 3-day mean energy expenditure. Fill in the "My Energy Expenditure" form (attached).
3. When calculating your energy expenditure per day, pay attention to the units.

Check list
Your completed assignment should include:
1. "3-day physical activity record" sheet (fill in dates in the appropriate places)
2. "My energy expenditure" sheet

Example: Part I. Recording physical activities

Day 1: 03/15/01 Jane Doe

<table>
<thead>
<tr>
<th>Hour</th>
<th>Min.</th>
<th>0-15</th>
<th>15-30</th>
<th>30-45</th>
<th>45-60</th>
</tr>
</thead>
<tbody>
<tr>
<td>12-1am</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>1-2 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-4 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-5 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-6 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-7 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-8 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8-9 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9-10 am</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>10-11 am</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-12 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12-1 pm</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2 pm</td>
<td></td>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-4 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-5 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-6 pm</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>6-7 pm</td>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>7-8 pm</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8-9 pm</td>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>9-10 pm</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-11 pm</td>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-12 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1</td>
</tr>
</tbody>
</table>
Example: Part II. Calculations

Jane Doe DAY 1: 03/15/01, Thursday

<table>
<thead>
<tr>
<th>Activity code</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34</td>
</tr>
<tr>
<td>2</td>
<td>30</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>9</td>
<td>0</td>
</tr>
</tbody>
</table>

Total: 96

**N = the number of 15-min. intervals that you carried out an activity**

Total must add up to 96:
96 boxes × 15 min = 1440 min
1440 / 60 min = 24 hours

0.26 kcal/kg/15 min × 34 15-min periods = 8.84 kcal/kg
0.38 kcal/kg/15 min × 30 15-min periods = 11.40 kcal/kg
0.57 kcal/kg/15 min × 2 15-min periods = 1.14 kcal/kg
0.70 kcal/kg/15 min × 8 15-min periods = 5.60 kcal/kg
0.83 kcal/kg/15 min × 19 15-min periods = 15.77 kcal/kg
1.5 kcal/kg/15 min × 3 15-min periods = 4.5 kcal/kg

Total = 47.25 kcal/kg – Day 1 Energy Expenditure

Jane’s weight (will be your weight in kg) = 70 kg
(to convert pounds to kilograms, divide pounds by 2.2)

45.75 kcal/kg × 70 kg = 3203 kcal – Day 1 Energy Expenditure
### Activity Codes

<table>
<thead>
<tr>
<th>Activity code</th>
<th>Activities</th>
<th>Approximate energy expenditure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td><strong>Lying down:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sleeping</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>Resting in bed</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td><strong>Sitting:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Listening in class</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Watching TV</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Riding a bus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Taking a bath</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reading</td>
<td>0.38</td>
</tr>
<tr>
<td></td>
<td>Eating</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Driving a car</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Writing by hand or typing</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td><strong>Standing:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Washing oneself</td>
<td>0.57</td>
</tr>
<tr>
<td></td>
<td>Cooking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Shaving</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Combing hair</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dusting</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td><strong>Light activities:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Getting dressed</td>
<td>0.70</td>
</tr>
<tr>
<td></td>
<td>Taking a walk (strolling)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Taking a shower</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td><strong>Light manual work:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Housework (washing windows, sweeping, etc.)</td>
<td>0.83</td>
</tr>
<tr>
<td></td>
<td>Moderately quick walking (going to school, shopping)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Baking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lab work</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Riding a moped</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td><strong>Light sport and leisure activities:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Volleyball</td>
<td>1.2</td>
</tr>
<tr>
<td></td>
<td>Table tennis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cycling (leisure)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Baseball (except the pitcher)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td><strong>Moderate manual work:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Repairing a fence</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td>Farm work</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Loading bags or boxes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Shoveling snow</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td><strong>Moderate sport and leisure activities:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Baseball (pitcher)</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>Badminton</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Canoeing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cycling</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dancing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tennis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Horseback riding</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Swimming</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gymnastics</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Brisk walking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Jogging (slow running)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cross-country skiing (leisure)</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td><strong>Intense manual work and exercise:</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sawing with a hand saw</td>
<td>1.95</td>
</tr>
<tr>
<td></td>
<td>Cutting tree branches</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mountain climbing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Football</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Running in a race</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Racquetball</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Basketball</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Boxing</td>
<td></td>
</tr>
</tbody>
</table>
Name: __________________________

**Activity Record**

<table>
<thead>
<tr>
<th>Hour</th>
<th>0-15</th>
<th>16-30</th>
<th>31-45</th>
<th>46-60</th>
<th>Hour</th>
<th>0-15</th>
<th>16-30</th>
<th>31-45</th>
<th>46-60</th>
<th>Hour</th>
<th>0-15</th>
<th>16-30</th>
<th>31-45</th>
<th>46-60</th>
</tr>
</thead>
<tbody>
<tr>
<td>12-1am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12-1am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12-1am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1-2 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1-2 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2-3 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2-3 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-4 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3-4 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3-4 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-5 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4-5 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4-5 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-6 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5-6 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5-6 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-7 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-7 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-7 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-8 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7-8 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7-8 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8-9 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8-9 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8-9 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9-10 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9-10 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9-10 am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-11am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10-11am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10-11am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-12pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11-12pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11-12pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12-1pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12-1pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12-1pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-2pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1-2 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1-2 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2-3 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2-3 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-4 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3-4 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3-4 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4-5 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4-5 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4-5 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-6 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5-6 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5-6 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-7 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-7 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-7 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-8 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7-8 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7-8 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8-9 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8-9 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>8-9 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9-10 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9-10 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>9-10 pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-11pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10-11pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10-11pm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-12am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11-12am</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11-12am</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In each box, write the number that corresponds to the activity which you have carried out during this 15-minute period. Please consult the Activity Codes to establish the proper coding. If an activity is carried out over a long period (e.g., sleeping) you can draw a continuous line in the rectangular boxes which follow until such a time when there is a change in activity.
Name: ____________________

My Energy Expenditure

<table>
<thead>
<tr>
<th>Activity Code</th>
<th>N</th>
<th>Activity Code</th>
<th>N</th>
<th>Activity Code</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>5</td>
<td>5</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>7</td>
<td>8</td>
<td>8</td>
<td>9</td>
<td>9</td>
</tr>
</tbody>
</table>

Total: ____________________

Calculations

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.26 × ___ = ___</td>
<td>0.26 × ___ = ___</td>
</tr>
<tr>
<td>2</td>
<td>0.38 × ___ = ___</td>
<td>0.38 × ___ = ___</td>
</tr>
<tr>
<td>3</td>
<td>0.57 × ___ = ___</td>
<td>0.57 × ___ = ___</td>
</tr>
<tr>
<td>4</td>
<td>0.70 × ___ = ___</td>
<td>0.70 × ___ = ___</td>
</tr>
<tr>
<td>5</td>
<td>0.83 × ___ = ___</td>
<td>0.83 × ___ = ___</td>
</tr>
<tr>
<td>6</td>
<td>1.20 × ___ = ___</td>
<td>1.20 × ___ = ___</td>
</tr>
<tr>
<td>7</td>
<td>1.40 × ___ = ___</td>
<td>1.40 × ___ = ___</td>
</tr>
<tr>
<td>8</td>
<td>1.50 × ___ = ___</td>
<td>1.50 × ___ = ___</td>
</tr>
<tr>
<td>9</td>
<td>1.95 × ___ = ___</td>
<td>1.95 × ___ = ___</td>
</tr>
</tbody>
</table>

My weight (in kilograms)

_____

My 3-day mean energy expenditure (in kcal):

\[
\frac{_____ + _____ + _____}{3} = _____
\] kcal
ACKNOWLEDGMENT

I am indebted to my co-major professors Douglas Lewis and Elisabeth Schafer for their guidance, encouragement, and financial support throughout my graduate studies. I am grateful to my committee members Drs. D. Lee Alekel, Patricia Murphy and Don Beitz for their support and guidance. I would also like to acknowledge scientists Kathy Hanson and Jeanne Stewart for their tremendous help with my phytosterol project. I express my heartiest thanks and appreciation to the Department of Food Science and Human Nutrition and Community Nutrition faculty and graduate students at Iowa State University for their emotional support. A special thank you to Dr. Iradje Ahrabi-Fard at University of Northern Iowa who was extremely helpful with subject recruitment for one of my education projects.