visited on 6/14/2019

Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults

The Evidence Report

NATIONAL INSTITUTES OF HEALTH
NATIONAL HEART, LUNG, AND BLOOD INSTITUTE

visited on 6/14/2019

visited on 6/14/2019

Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults

The Evidence Report

NIH Publication
No. 98-4083
September 1998
National Institutes of Health

National Heart, Lung, and Blood Institute in cooperation with The National Institute of Diabetes and Digestive and Kidney Diseases

visited on 6/14/2019

NHLBI Obesity Education Initiative Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults

F. Xavier Pi-Sunyer, M.D., M.P.H.
Chair of the Panel
Chief, Endocrinology, Diabetes, and Nutrition
Director, Obesity Research Center
St. Luke's/Roosevelt Hospital Center
Professor of Medicine
Columbia University College of Physicians and Surgeons
New York, NY

Diane M. Becker, Sc.D., M.P.H.
Director
Center for Health Promotion
Associate Professor
Department of Medicine
The Johns Hopkins University
Baltimore, MD

Claude Bouchard, Ph.D.
Professor of Exercise Physiology
Physical Activity Sciences Laboratory
Laval University
Sainte Foy, Quebec
CANADA

Richard A. Carleton, M.D.
Professor of Medicine
Brown University School of Medicine
Pawtucket, RI

Graham A. Colditz, M.D., Dr.P.H.
Associate Professor of Medicine
Harvard Medical School
Channing Laboratory
Boston, MA

William H. Dietz, M.D., Ph.D.
Director
Division of Nutrition and Physical Activity
National Center for Chronic Disease Prevention and Health Promotion
Centers for Disease Control and Prevention
Atlanta, GA

John P. Foreyt, Ph.D.
Professor of Medicine and Director
Nutrition Research Clinic
Baylor College of Medicine
Houston, TX

Robert J. Garrison, Ph.D.
Associate Professor
Department of Preventive Medicine
University of Tennessee, Memphis
Memphis, TN

Scott M. Grundy, M.D., Ph.D.
Director
Center for Human Nutrition
University of Texas
Southwestern Medical Center at Dallas
Dallas, TX

Barbara C. Hansen, Ph.D.
Professor of Physiology
Director of Obesity and Diabetes Research Center
University of Maryland School of Medicine
Baltimore, MD

Millicent Higgins, M.D.
Department of Epidemiology
School of Public Health
University of Michigan
Ann Arbor, MI

visited on 6/14/2019

James O. Hill, Ph.D.
Associate Director of Research
Center for Human Nutrition
University of Colorado Health Sciences Center
Denver, CO

Barbara V. Howard, Ph.D.
President
Medlantic Research Institute
Washington, DC

Robert C. Klesges, Ph.D.
Professor
University of Memphis Prevention Center
University of Memphis
Memphis, TN

Robert J. Kuczmarski, Dr.P.H., R.D.
Nutrition Analyst
National Center for Health Statistics
Centers for Disease Control and Prevention
Hyattsville, MD

Shiriki Kumanyika, Ph.D., R.D., M.P.H.
Professor and Head
Department of Human Nutrition and Dietetics
The University of Illinois at Chicago
Chicago, IL

R. Dee Legako, M.D.
Prime Care Canyon Park Family Physicians, Inc.
Edmond, OK

T. Elaine Prewitt, Dr.P.H., R.D.
Assistant Professor
Department of Preventive Medicine and Epidemiology
Loyola University Medical Center
Maywood, IL

Albert P. Rocchini, M.D.
Chief of Cardiology
University of Michigan Medical Center
Ann Arbor, MI

Philip L. Smith, M.D.
Professor of Medicine
Division of Pulmonary and Critical Care Medicine
The Johns Hopkins Asthma and Allergy Center
Baltimore, MD

Linda G. Snetselaar, Ph.D., R.D.
Associate Professor
Head of Preventive Nutrition Education
Department of Preventive Medicine
University of Iowa
Iowa City, IA

James R. Sowers, M.D.
Professor of Medicine and Physiology
Director
Division of Endocrinology, Metabolism, and Hypertension
Wayne State University School of Medicine
University Health Center
Detroit, MI

Michael Weintraub, M.D.
Director
Office of Drug Evaluation V
Food and Drug Administration
Rockville, MD

David F. Williamson, Ph.D., M.S.
Epidemiologist
Division of Diabetes Translation
Centers for Disease Control and Prevention
Chamblee, GA

visited on 6/14/2019

G. Terence Wilson, Ph.D.
Oscar K. Buros Professor of Psychology
Director, Rutgers Eating Disorders Clinic
Piscataway, NJ

Ex-Officio Members

Clarice D. Brown, M.S.
Project Manager
CODA Research, Inc.
Silver Spring, MD

Karen A. Donato, M.S., R.D.*
Executive Director of the Panel Coordinator
NHLBI Obesity Education Initiative
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD

Nancy Ernst, Ph.D., R.D.*
Nutrition Coordinator
Office of the Director
Division of Epidemiology and Clinical Applications
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD

D. Robin Hill, Ph.D.*
Social Science Analyst
Behavioral Medicine Branch
Division of Epidemiology and Clinical Applications
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD

Michael J. Horan, M.D., Sc.M.*
Director
Division of Heart and Vascular Diseases
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD

Van S. Hubbard, M.D., Ph.D.
Director, NIH Division of Nutrition Research Coordination
Chief, Nutritional Sciences Branch
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, MD

James P. Kiley, Ph.D.*
Director
Airway Biology and Disease Program
Division of Lung Diseases
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD

Eva Obarzanek, Ph.D., R.D., M.P.H.*
Research Nutritionist
Prevention Scientific Research Group
Division of Epidemiology and Clinical Applications
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD

Consultant

David Schriger, M.D., M.P.H., F.A.C.E.P.
Associate Professor
UCLA Emergency Medicine Center
University of California at Los Angeles
School of Medicine
Los Angeles, CA

San Antonio Cochrane Center

Elaine Chiquette, Pharm.D.
V.A. Cochrane Center at San Antonio
Audie L. Murphy Memorial Veterans Hospital
San Antonio, TX

* NHLBI Obesity Education Initiative Task Force Members

visited on 6/14/2019

Cynthia Mulrow, M.D., M.Sc.
V.A. Cochrane Center at San Antonio
Audie L. Murphy Memorial Veterans Hospital
San Antonio, TX

Staff

Adrienne Blount, B.S.
R.O.W. Sciences, Inc.
Rockville, MD

Maureen Harris, M.S., R.D.
R.O.W. Sciences, Inc.
Rockville, MD

Anna Hodgson, M.A.
R.O.W. Sciences, Inc.
Rockville, MD

Pat Moriarty, M.Ed., R.D.
R.O.W. Sciences, Inc.
Rockville, MD

The panel acknowledges the assistance of Dr. Rashid Chotani, Johns Hopkins University; Dr. Robert Klesges, University of Memphis; Dr. Walter Pories, East Carolina University; Dr. Ivan Baines, NHLBI; Dr. Christine Kelly, NHLBI; Glen Bennett, NHLBI; Dr. Fred Heydrick, BioReview; Debbie Lurie, Prospect Associates; Estelle Schwalb, Prospect Associates; Lori McCray, R.O.W. Sciences, Inc.; and Niyati Pandya, R.O.W. Sciences, Inc.

visited on 6/14/2019

Contents

Foreword vii
Executive Summary xi

1. Introduction 1
A. Rationale for Guidelines Development 1
B. Objectives of the Guidelines 2
C. Guideline Development Methodology 2
D. Statement of Assumptions 5
E. Intended Users of These Guidelines 5

2. Overweight and Obesity: Background 6
A. Health and Economic Costs 6
1. Prevalence and Time Trends 6
2. Demographic Variations in Overweight and Obesity Prevalence 9
3. Economic Costs of Overweight and Obesity 9
B. Prevention of Overweight and Obesity 11
C. Health Risks of Overweight and Obesity 12
1. Morbidity 12
2. Overweight/Obesity and Morbidity in Minority Populations 23
3. Obesity and Mortality 23
D. Weight Loss and Mortality 25
E. Environment 26
F. Genetic Influence in the Development of Overweight and Obesity 27

3 Examination of Randomized Controlled Trial Evidence 29
A. Why Treat Overweight and Obesity? 29
1. Blood Pressure 29
2. Serum/Plasma Lipids and Lipoproteins 33
3. Impaired Glucose Tolerance and Diabetes 39
4. Decreases in Abdominal Fat with Weight Loss 41
B. What Treatments are Effective? 42
1. Dietary Therapy 42
2. Physical Activity 44
3. Combined Therapy 47
4. Behavior Therapy 48
5. Pharmacotherapy 53
6. Surgery 54
7. Other Interventions for Overweight and Obesity Treatment 55

4. Treatment Guidelines 56
A. Overview 56
B. Assessment and Classification of Overweight and Obesity 56
1. Assessment of Overweight and Obesity 56
2. Classification of Overweight and Obesity 58
C. Assessment of Risk Status 62
D. Evaluation and Treatment Strategy 65
E. Exclusion from Weight Loss Therapy 70
F. Patient Motivation 70
G. Goals of Weight Loss and Management 71
1. Weight Loss 71
2. Weight Maintenance at Lower Weight 72
3. Prevention of Further Weight Gain 73
H. Strategies for Weight Loss and Weight Maintenance 73
1. Dietary Therapy 73
2. Physical Activity 77
3. Behavior Therapy 81
4. Combined Therapy 83
5. Pharmacotherapy 83
6. Surgery 86
I. Smoking Cessation in the Overweight or Obese Patient 91
J. Role of Health Professionals in Weight Loss Therapy 92

5. Summary of Recommendations 95

6. Future Research 98
A. Intervention Approaches 98
B. Causes and Mechanisms of Overweight and Obesity 99
C. Abdominal Fat, Body Weight and Disease Risk 99
D. Assessment Methods 99

7. Appendices 100
Appendix I.A.1 Guidelines Development Methodology 100
Appendix I.A.2 Literature Review 109
Appendix II Description of Evidence 112
Appendix III Special Populations 117
Appendix IV Obesity and Sleep Apnea 137
Appendix V Body Mass Index Chart—How to Measure Obesity 139
Appendix VI Practical Dietary Therapy Information 141
Appendix VII Resource List 165
Appendix VIII Glossary of Terms 168

List of Abbreviations 179

Reference List 181

Publication List 226

visited on 6/14/2019

Foreword

In 1995, the National Obesity Education Initiative of the National Heart, Lung, and Blood Institute (NHLBI), in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), convened the first Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults to develop clinical practice guidelines for primary care practitioners.

The impetus for these guidelines was the recognition that the prevalence of overweight and obesity in the United States is increasing, and that practitioners need to be alerted to the accompanying health risks. According to the latest statistics from the third National Health and Nutrition Examination Survey, 97 million Americans are overweight or obese. Excess weight is often accompanied by high blood pressure, high blood cholesterol, type 2 diabetes, coronary heart disease, and other health problems. The total costs attributable to obesity-related disease approach $100 billion annually in the United States.

The panel used the principles of evidence-based medicine, including an evidence model and evidence categories. It was the first time a panel thoroughly examined the scientific evidence for risks associated with overweight and obesity, and their treatments, and developed clinical practice recommendations based on their conclusions.

The panel was headed by Dr. F. Xavier Pi-Sunyer, of St. Luke's/Roosevelt Hospital Center in New York City. He and the other 23 panel members methodically and critically examined a vast amount of published scientific evidence. The panel also obtained scientific input from approximately 115 outside reviewers. The result was *The Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report.* Dr. David Schriger of the University of California at Los Angeles, who is a methodologist consultant in the area of evidence-based practice guidelines, and Dr. Elaine Chiquette of the San Antonio Cochrane Center in Texas, who developed the evidence tables, served as key advisors to the panel.

Evidence examined by the panel included: research on the relationship of overweight and obesity to high blood pressure, high blood cholesterol, type 2 diabetes, stroke, congestive heart failure, coronary heart disease, various cancers, osteoarthritis, and sleep apnea; risks associated with the distribution and amount of body fat; and various treatment strategies, including diet, physical activity, behavior therapy, pharmacotherapy, and surgery. The resulting guidelines document how different treatment strategies affect weight loss and how weight control affects the major risk factors for heart disease and stroke.

The guidelines have been reviewed and endorsed by many professional organizations. In fact, because of the associated risks between high blood pressure and high blood cholesterol and overweight and obesity, the document represents

visited on 6/14/2019

the first clinical practice guidelines to be reviewed and endorsed by members of the coordinating committees of both the National Cholesterol Education Program and the National High Blood Pressure Education Program which comprise approximately 52 professional societies, government agencies, and consumer organizations. Two additional groups endorsing the guidelines are the North American Association for the Study of Obesity and the NIDDK National Task force on the Prevention and Treatment of Obesity.

The report, the evidence model and its accompanying evidence tables, and a body mass index calculator are available on the NHLBI website at the following location: http://www.nhlbi.nih.gov./nhlbi/cardio/obes/prof/guidelns/ob_home.htm

An abbreviated version of the evidence report is being prepared and will be distributed to primary care physicians, nurses, registered dietitians and nutritionists, and other health care practitioners.

It is our hope that these clinical guidelines will not only help the health care practitioner understand the importance of weight management but also provide them with the tools to assess and treat their patients more effectively. Equally important, we hope that the guidelines lead to a greater public understanding of obesity and a greater appreciation for the persistent efforts of millions of people to lose weight.

Claude Lenfant, M.D.
Director
National Heart, Lung,
and Blood Institute

visited on 6/14/2019

Evidence Report Endorsements

NATIONAL CHOLESTEROL EDUCATION PROGRAM (NCEP)

Coordinating Committee Member Organizations

Theodore G. Ganiats, M.D., *American Academy of Family Physicians,* Gary Graham, M.D., *American Academy of Insurance Medicine,* Ronald Kleinman, M.D., *American Academy of Pediatrics,* Ann Smith, R.N., C.O.H.N.-S., *American Association of Occupational Health Nurses,* Richard C. Pasternak, M.D., F.A.C.C., *American College of Cardiology,* Gerald T. Gau, M.D., *American College of Chest Physicians,* Ruth Ann Jordan, M.D., *American College of Occupational and Environmental Medicine,* Thomas E. Nolan, M.D., *American College of Obstetricians and Gynecologists,* Alan J. Garber, M.D., Ph.D., *American Diabetes Association, Inc.,* Linda Van Horn, Ph.D., R.D., *American Dietetic Association,* Scott Grundy, M.D., Ph.D., *American Heart Association,* Sandra Cornett, R.N., Ph.D., *American Hospital Association,* Yank D. Coble, Jr., M.D., *American Medical Association,* Joan Watson, R.N., Ph.D., F.A.A.N., *American Nurses' Association,* Michael Clearfield, D.O., *American Osteopathic Association,* Joanne Mitten, M.H.E., *Association of State and Territorial Health Officials,* Gerald J. Wilson, M.B.A., *Citizens for Public Action on Blood Pressure and Cholesterol, Inc.,* Linda Burnes-Bolton, Dr.P.H., R.N., *National Black Nurses' Association, Inc.,* Luther T. Clark, M.D., *National Medical Association,* Darlene Lansing, M.P.H., R.D., *Society for Nutrition Education*

Associate Member Coordinating Committee Organizations

Stanley Wallach, M.D., *American College of Nutrition,* Donald O. Fedder, Dr.P.H., M.P.H., *Society for Public Health Education*

Federal Agency Liaison Coordinating Committee Representatives

Yvonne L. Bronner, Sc.D., R.D., L.D., *NHLBI Ad Hoc Committee on Minority Populations,* Francis D. Chesley, M.D., *Agency for Health Care Policy and Research,* William H. Dietz, M.D., Ph.D., *Centers for Disease Control and Prevention,* Thomas M. Lasater, Ph.D., *Coordinating Committee for the Community Demonstration Studies,* Alanna Moshfegh, M.S., R.D., *Department of Agriculture,* Col. Michael Parkinson, M.D., M.P.H., *Department of Defense,* Pamela Steele, M.D., *Department of Veterans Affairs,* Celia Hayes, M.P.H., R.D., *Health Resources and Services Administration,* Clifford Johnson, M.P.H., *National Center for Health Statistics,* Linda Meyers, Ph.D., *Office of Disease Prevention and Health Promotion*

NATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAM (NHBPEP)

Coordinating Committee Member Organizations

Lee A. Green, M.D., M.P.H., *American Academy of Family Physicians,* Jack P. Whisnant, M.D., *American Academy of Neurology,* Barry N. Hyman, M.D., F.A.C.P., *American Academy of Ophthalmology,* Lisa Mustone-Alexander, M.P.H., P.A., *American Academy of Physician Assistants,* Henry Guevara, B.S.N., R.N., C.O.H.N.-S., *American Association of Occupational Health Nurses,* Edward D. Frohlich, M.D., *American College of Cardiology,* Sheldon G. Sheps, M.D., *American College of Chest Physicians,* Ron Stout, M.D., *American College of Occupational and Environmental Medicine,* Jerome D. Cohen, M.D., *American College of Physicians,* Carlos Vallbona, M.D., *American College of Preventive Medicine,* James R. Sowers, M.D., *American Diabetes Association, Inc.,* Mary C. Winston, Ed.D., R.D., *American Dietetic Association,* Daniel W. Jones, M.D., *American Heart Association,* Roxane Spitzer, Ph.D., F.A.A.N., *American Hospital Association,* Nancy Houston Miller, B.S.N., *American Nurses' Association,* Linda Casser, O.D., *American Optometric Association,* William A. Nickey, D.O., *American Osteopathic Association,* Raymond W. Roberts, Pharm.D., *American Pharmaceutical Association,* Pamela J. Colman, D.P.M., *American Podiatric Medical Association,* Nancy McKelvey, M.S.N., R.N., *American Red Cross,* Barry L. Carter, Pharm.D., F.C.C.P., *American Society of Health-System Pharmacists,* Norman M. Kaplan, M.D., *American Society of Hypertension,* Jackson T. Wright, M.D., Ph.D., *Association of Black Cardiologists,* Gerald J. Wilson, M.B.A., *Citizens for Public Action on Blood Pressure and Cholesterol, Inc.,* Joseph L. Izzo, Jr., M.D., *Council on Geriatric Cardiology,* James W. Reed, M.D., F.A.C.P., F.A.C.E., *International Society on Hypertension in Blacks,* Rita Strickland, Ed.D., R.N., *National Black Nurses' Association, Inc.,* William Manger, M.D., Ph.D., *National Hypertension Association, Inc.,* Murray Epstein, M.D., *National Kidney Foundation, Inc.,* Otelio S. Randall, M.D., F.A.C.C., *National Medical Association,* Edwin Marshall, O.D., M.P.H., *National Optometric Association,* Harold W. "Pete" Todd, *National Stroke Association,* Kathryn M. Kolasa, Ph.D., R.D., L.D.N., *Society for Nutrition Education*

Federal Agency Liaison Coordinating Committee Representatives

Keith Ferdinand, M.D., F.A.C.C., *NHLBI Ad Hoc Committee on Minority Populations,* Francis D. Chesley, M.D., *Agency for Health Care Policy and Research,* H. Mitchell Perry, Jr., M.D., *Department of Veterans Affairs,* Jay Merchant, M.H.A., *Health Care Financing Administration,* Vicki Burt, R.N., Sc.M., *National Center for Health Statistics,* Elizabeth H. Singer, M.S., *National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK)*

NIDDK NATIONAL TASK FORCE ON THE PREVENTION AND TREATMENT OF OBESITY

NORTH AMERICAN ASSOCIATION FOR THE STUDY OF OBESITY (NAASO)

visited on 6/14/2019

visited on 6/14/2019

Executive Summary

Introduction

An estimated 97 million adults in the United States are overweight or obese, a condition that substantially raises their risk of morbidity from hypertension, dyslipidemia, type 2 diabetes, coronary heart disease, stroke, gallbladder disease, osteoarthritis, sleep apnea and respiratory problems, and endometrial, breast, prostate, and colon cancers. Higher body weights are also associated with increases in all-cause mortality. Obese individuals may also suffer from social stigmatization and discrimination. As a major contributor to preventive death in the United States today, overweight and obesity pose a major public health challenge.

Overweight is here defined as a body mass index (BMI) of 25 to 29.9 kg/m^2 and obesity as a BMI of ≥ 30 kg/m^2. However, overweight and obesity are not mutually exclusive, since obese persons are also overweight. A BMI of 30 is about 30 lb overweight and equivalent to 221 lb in a 6'0" person and to 186 lb in one 5'6". The number of overweight and obese men and women has risen since 1960; in the last decade the percentage of people in these categories has increased to 54.9 percent of adults age 20 years or older. Overweight and obesity are especially evident in some minority groups, as well as in those with lower incomes and less education.

Obesity is a complex multifactorial chronic disease that develops from an interaction of genotype and the environment. Our understanding of how and why obesity develops is incomplete, but involves the integration of social, behavioral, cultural, physiological, metabolic and genetic factors.

While there is agreement about the health risks of overweight and obesity, there is less agreement about their management. Some have argued against treating obesity because of the difficulty in maintaining long-term weight loss and of potentially negative consequences of the frequently seen pattern of weight cycling in obese subjects. Others argue that the potential hazards of treatment do not outweigh the known hazards of being obese. The intent of these guidelines is to provide evidence for the effects of treatment on overweight and obesity. The guidelines focus on the role of the primary care practitioner in treating overweight and obesity.

Evidence-Based Guidelines

To evaluate published information and to determine the most appropriate treatment strategies that would constitute evidence-based clinical guidelines on overweight and obesity for physicians and associated health professionals in clinical practice, health care policy makers, and clinical investigators, the National Heart, Lung, and Blood Institute's Obesity Education Initiative in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases convened the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults in May 1995. The guidelines are based on a systematic review of the published scientific literature found in MEDLINE from January 1980 to September 1997 of topics identified by the panel as key to extrapolating the data related to the obesity evidence model.

visited on 6/14/2019

Evidence from approximately 394 randomized controlled trials (RCTs) was considered by the panel.

The panel is comprised of 24 members, 8 ex-officio members, and a methodologist consultant. Areas of expertise contributed to by panel members included primary care, epidemiology, clinical nutrition, exercise physiology, psychology, physiology, and pulmonary disease. There were five meetings of the full panel and two additional meetings of the executive committee comprised of the panel chair and four panel members.

The San Antonio Cochrane Center assisted the panel in the literature abstraction and in organizing the data into appropriate evidence tables. The center pretested and used a standardized 25-page form or "Critical Review Status Sheet" for the literature abstraction. Ultimately, 236 RCT articles were abstracted and the data were then compiled into individual evidence tables developed for each RCT. The data from these RCTs served as the basis for many of the recommendations contained in the guidelines.

The panel determined the criteria for deciding on the appropriateness of an article. At a minimum, studies had to have a time frame from start to finish of at least 4 months. The only exceptions were a few 3-month studies related to dietary therapy and pharmacotherapy. To consider the question of long-term maintenance, studies with outcome data provided at approximately 1 year or longer were examined. Excluded were studies in which self-reported weights by subjects were the only indicators used to measure weight loss. No exclusions of studies were made by study size. The panel weighed the evidence based on a thorough examination of the threshold or magnitude of the treatment effect. Each evidence statement (other than those with no available evidence) and each recommendation is categorized by a level of evidence which ranges from A to D. Table ES-1 summarizes the categories of evidence by their source and provides a definition for each category.

- **Who is at Risk?** All overweight and obese adults (age 18 years of age or older) with a BMI of ≥25 are considered at risk for developing associated morbidities or diseases such as hypertension, high blood cholesteral, type 2 diabetes, coronary heart disease, and other diseases. Individuals with a BMI of 25 to 29.9 are considered overweight, while individuals with a BMI ≥30 are considered obese. Treatment of overweight is recommended only when patients have two or more risk factors or a high waist circumference. It should focus on altering dietary and physical activity patterns to prevent development of obesity and to produce moderate weight loss. Treatment of obesity should focus on producing substantial weight loss over a prolonged period. The presence of comorbidities in overweight and obese patients should be considered when deciding on treatment options.
- **Why Treat Overweight and Obesity?** Obesity is clearly associated with increased morbidity and mortality. There is strong evidence that weight loss in overweight and obese individuals reduces risk factors for diabetes and cardiovascular disease (CVD). Strong evidence exists that weight loss reduces blood pressure in both overweight hypertensive and nonhypertensive individuals; reduces serum triglycerides and increases high-density lipoprotein (HDL)-cholesterol; and generally produces some reduction in total serum cholesterol and low-density lipoprotein (LDL)-cholesterol. Weight loss reduces blood glucose levels in overweight and obese persons without diabetes; and weight loss also reduces blood glucose levels and HbA_{1c} in some patients with type 2 diabetes. Although there have been no prospective trials to show changes in mortality with weight loss in obese patients, reductions in risk factors would suggest that develop-

visited on 6/14/2019

Table ES-1:

Evidence Categories

Evidence Category	Sources of Evidence	Definition
A	Randomized controlled trials (rich body of data)	Evidence is from endpoints of well-designed RCTs (or trials that depart only minimally from randomization) that provide a consistent pattern of findings in the population for which the recommendation is made. Category A therefore requires substantial numbers of studies involving substantial numbers of participants.
B	Randomized controlled trials (limited body of data)	Evidence is from endpoints of intervention studies that include only a limited number of RCTs, post hoc or subgroup analysis of RCTs, or meta-analysis of RCTs. In general, Category B pertains when few randomized trials exist, they are small in size, and the trial results are somewhat inconsistent, or the trials were undertaken in a population that differs from the target population of the recommendation.
C	Nonrandomized trials Observational studies	Evidence is from outcomes of uncontrolled or nonrandomized trials or from observational studies.
D	Panel Consensus Judgment	Expert judgment is based on the panel's synthesis of evidence from experimental research described in the literature and/or derived from the consensus of panel members based on clinical experience or knowledge that does not meet the above-listed criteria. This category is used only in cases where the provision of some guidance was deemed valuable but an adequately compelling clinical literature addressing the subject of the recommendation was deemed insufficient to justify placement in one of the other categories (A through C).

visited on 6/14/2019

ment of type 2 diabetes and CVD would be reduced with weight loss.

- **What Treatments Are Effective?** A variety of effective options exist for the management of overweight and obese patients, including dietary therapy approaches such as low-calorie diets and lower-fat diets; altering physical activity patterns; behavior therapy techniques; pharmacotherapy*; surgery; and combinations of these techniques.

Clinical Guidelines

Treatment of the overweight or obese patient is a two-step process: assessment and treatment management. Assessment requires determination of the degree of overweight and overall risk status. Management includes both reducing excess body weight and instituting other measures to control accompanying risk factors.

Assessment: When assessing a patient for risk status and as a candidate for weight loss therapy, consider the patient's BMI, waist circumference, and overall risk status. Consideration also needs to be given to the patient's motivation to lose weight.

- **Body Mass Index.** The BMI, which describes relative weight for height, is significantly correlated with total body fat content. The BMI should be used to assess overweight and obesity and to monitor changes in body weight. In addition, measurements of body weight alone can be used to determine efficacy of weight loss therapy. BMI is calculated as weight (kg)/height squared (m^2). To estimate BMI using pounds and inches, use: [weight (pounds)/height (inches)^2] x 703. Weight classifications by BMI, selected for use in this report, are shown in Table ES-2. A conversion table of heights and weights resulting in selected BMI units is provided in Table ES-3.

- **Waist Circumference.** The presence of excess fat in the abdomen out of proportion to total body fat is an independent predictor of risk factors and morbidity. Waist circumference is positively correlated with abdominal fat content. It provides a clinically acceptable measurement for assessing a patient's abdominal fat content before and during weight loss treatment. The sex-specific cutoffs noted on the next page can be used to identify

TABLE ES-2:

CLASSIFICATION OF OVERWEIGHT AND OBESITY BY BMI

	Obesity Class	BMI (kg/m^2)
Underweight		< 18.5
Normal		18.5 – 24.9
Overweight		25.0 – 29.9
Obesity	I	30.0 – 34.9
	II	35.0 – 39.9
Extreme Obesity	III	≥ 40

* As of September 1997, the Food and Drug Administration (FDA) requested the voluntary withdrawal from the market of dexfenfluramine and fenfluramine due to a reported association between valvular heart disease and the use of dexfenfluramine or fenfluramine alone or combined with phentermine. The use of these drugs for weight reduction, therefore, is not recommended in this report. Sibutramine is approved by FDA for long-term use. It has limited but definite effects on weight loss and can facilitate weight loss maintenance (Note: FDA approval for orlistat is pending a resolution of labeling issues and results of Phase III trials.)

visited on 6/14/2019

HIGH RISK

Men > 102 cm (> 40 in)

Women > 88 cm (> 35 in)

increased relative risk for the development of obesity-associated risk factors in most adults with a BMI of 25 to 34.9 kg/m^2:

These waist circumference cutpoints lose their incremental predictive power in patients with a BMI ≥ 35 kg/m^2 because these patients will exceed the cutpoints noted above. Table ES-4 adds the disease risk of increased abdominal fat to the disease risk of BMI. These categories denote *relative* risk, not *absolute* risk; that is, relative to risk at normal weight. They should not be equated with absolute risk, which is determined by a summation of risk factors. They relate to the need to institute weight loss therapy and do not directly define the required intensity of modification of risk factors associated with obesity.

- **Risk Status.** Assessment of a patient's absolute risk status requires examination for the presence of:

  Disease conditions: established coronary heart disease (CHD), other atherosclerotic diseases, type 2 diabetes, and sleep apnea; patients with these conditions are classified as being at very high risk for disease complications and mortality.

  Other obesity-associated diseases: gynecological abnormalities, osteoarthritis, gallstones and their complications, and stress incontinence.

  Cardiovascular risk factors: cigarette smoking, hypertension (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg, or the patient is taking antihypertensive agents), high-risk LDL-cholesterol (≥ 160 mg/dL), low HDL-cholesterol (< 35 mg/dL), impaired fasting glucose (fasting plasma glucose of 110 to 125 mg/dL), family history of premature CHD (definite myocardial infarction or sudden death at or before 55 years of age in father or other male first-degree relative, or at or before 65 years of age in mother or other female first-degree relative), and age (men ≥ 45 years and women ≥ 55 years or postmenopausal). Patients can be classified as being at high absolute risk if they have three of the aforementioned risk factors. Patients at high absolute risk usually require clinical management of risk factors to reduce risk.

  Patients who are overweight or obese often have other cardiovascular risk factors. Methods for estimating *absolute risk* status for developing cardiovascular disease based on these risk factors are described in detail in the National Cholesterol Education Program's *Second Report of the Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults* (NCEP's ATP II) and the *Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* (JNC VI). The intensity of intervention for cholesterol disorders or hypertension is adjusted according to the absolute risk status estimated from multiple risk correlates. These include both the risk factors listed above and evidence of end-organ damage present in hypertensive patients. Approaches to therapy for cholesterol disorders and hypertension are described in ATP II and JNC VI, respectively. In overweight patients, control of cardiovascular risk factors deserves equal emphasis as weight reduction therapy. Reduction of risk factors will reduce the risk for cardiovascular disease whether or not efforts at weight loss are successful.

  Other risk factors: physical inactivity and high serum triglycerides (> 200 mg/dL). When these factors are present, patients can

visited on 6/14/2019

Table ES-3:

Selected BMI Units Categorized by Inches (cm) and pounds (kg)

	Body weight in pounds (kg)		
Height in inches (cm)	**BMI 25 kg/m²**	**BMI 27 kg/m²**	**BMI 30 kg/m²**
58 (147.32)	119 (53.98)	129 (58.51)	143 (64.86)
59 (149.86)	124 (56.25)	133 (60.33)	148 (67.13)
60 (152.40)	128 (58.06)	138 (62.60)	153 (69.40)
61 (154.94)	132 (59.87)	143 (64.86)	158 (71.67)
62 (157.48)	136 (61.69)	147 (66.68)	164 (74.39)
63(160.02)	141 (63.96)	152 (68.95)	169 (76.66)
64 (162.56)	145 (65.77)	157 (71.21)	174 (78.93)
65 (165.10)	150 (68.04)	162 (73.48)	180 (81.65)
66 (167.64)	155 (70.31)	167 (75.75)	186 (84.37)
67 (170.18)	159 (72.12)	172 (78.02)	191 (86.64)
68 (172.72)	164 (74.39)	177 (80.29)	197 (89.36)
69 (175.26)	169 (76.66)	182 (82.56)	203 (92.08)
70 (177.80)	174 (78.93)	188 (85.28)	207 (93.89)
71 (180.34)	179 (81.19)	193 (87.54)	215 (97.52)
72 (182.88)	184 (83.46)	199 (90.27)	221 (100.25)
73 (185.42)	189 (85.73)	204 (92.53)	227 (102.97)
74 (187.96)	194 (88.00)	210 (95.26)	233 (105.69)
75 (190.50)	200 (90.72)	216 (97.98)	240 (108.86)
76 (193.04)	205 (92.99)	221(100.25)	246 (111.58)

Metric conversion formula =
weight (kg)/height (m)²

Example of BMI calculation:
A person who weighs 78.93 kilograms and is 177 centimeters tall has a BMI of 25:

weight (78.93 kg)/height (1.77 m)² = 25

Non-metric conversion formula =
[weight (pounds)/height (inches)²] x 703

Example of BMI calculation:
A person who weighs 164 pounds and is 68 inches (or 5' 8") tall has a BMI of 25:

[weight (164 pounds)/height (68 inches)²] x 703 = 25

visited on 6/14/2019

be considered to have incremental absolute risk above that estimated from the preceding risk factors. Quantitative risk contribution is not available for these risk factors, but their presence heightens the need for weight reduction in obese persons.

- **Patient Motivation.** When assessing the patient's motivation to enter weight loss therapy, the following factors should be evaluated: reasons and motivation for weight reduction; previous history of successful and unsuccessful weight loss attempts; family, friends, and work-site support; the patient's understanding of the causes of obesity and how obesity contributes to several diseases; attitude toward physical activity; capacity to engage in physical activity; time availability for weight loss intervention; and financial considerations. In addition to considering these issues, the health care practitioner needs to heighten a patient's motivation for weight loss and prepare the patient for treatment. This can be done by enumerating the dangers accompanying persistent obesity and by describing the strategy for clinically assisted weight reduction. Reviewing the patients' past attempts at weight loss and explaining how the new treatment plan will be different can encourage patients and provide hope for successful weight loss.

Evaluation and Treatment: The general goals of weight loss and management are: (1) at a minimum, to prevent further weight gain; (2) to reduce body weight; and (3) to maintain a lower body weight over the long term. The overall strategy for the evaluation and treatment of overweight and obese patients is presented in the Treatment Algorithm on the next page. This algorithm applies only to the assessment for overweight and obesity and subsequent decisions

Table ES-4:

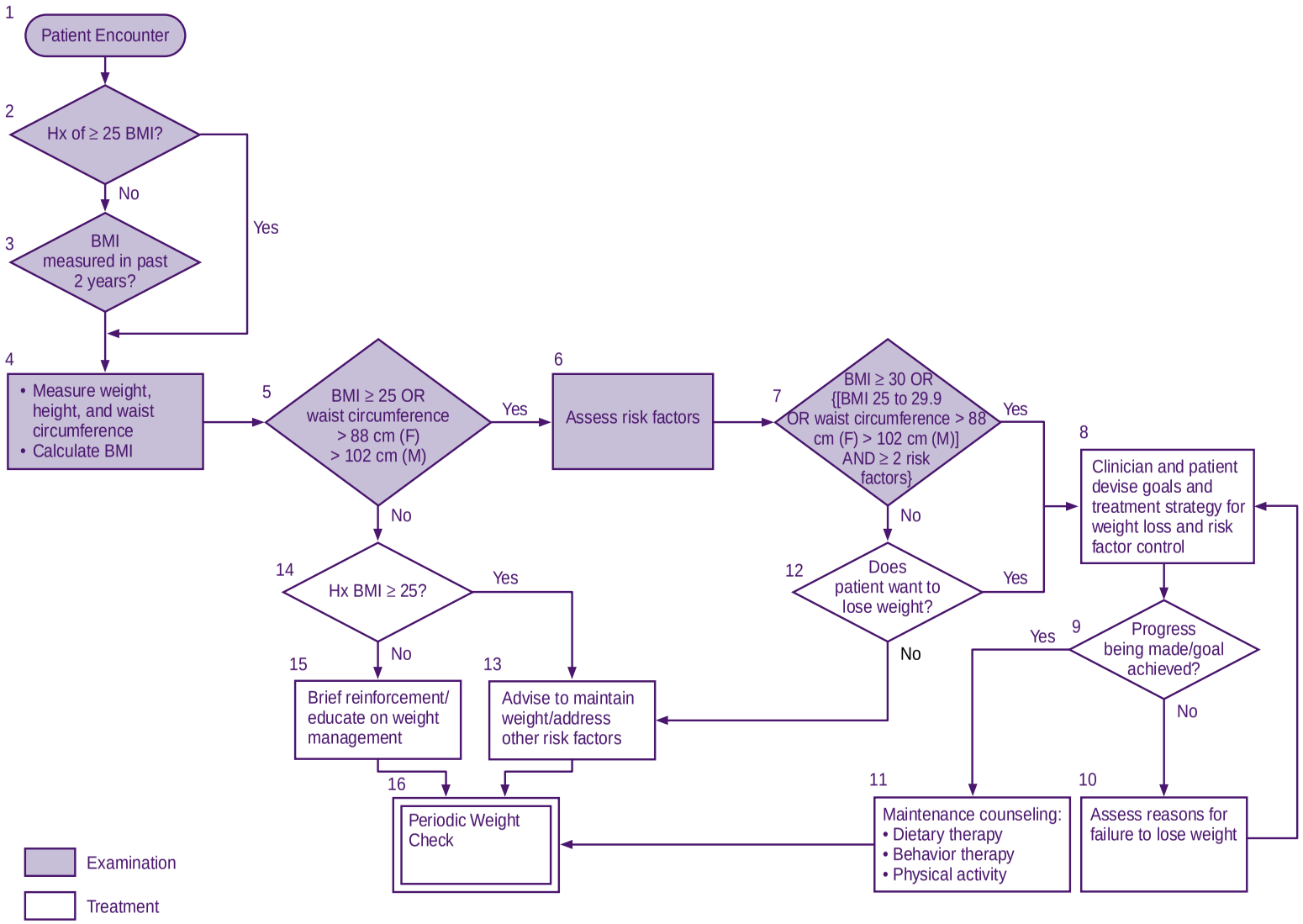
Classification of Overweight and Obesity by BMI, Waist Circumference and Associated Disease Risk*

			Disease Risk* Relative to Normal Weight and Waist Circumference	
	BMI (kg/m²)	Obesity Class	Men ≤ 102 cm (≤ 40 in) Women ≤ 88 cm (≤ 35 in)	> 102 cm (> 40 in) > 88 cm (> 35 in)
Underweight	<18.5		—	—
Normal+	18.5 –24.9		—	—
Overweight	25.0 – 29.9		Increased	High
Obesity	30.0 – 34.9	I	High	Very High
	35.0 – 39.9	II	Very High	Very High
Extreme Obesity	≥40	III	Extremely High	Extremely High

* Disease risk for type 2 diabetes, hypertension, and CVD.
\+ Increased waist circumference can also be a marker for increased risk even in persons of normal weight.

visited on 6/14/2019

Treatment Algorithm*



* This algorithm applies only to the assessment for overweight and obesity and subsequent decisions based on that assessment. It does not include any initial overall assessment for cardiovascular risk factors or diseases that are indicated.

visited on 6/14/2019

based on that assessment. It does not include any initial overall assessment for cardiovascular risk factors or diseases that are indicated.

- **Goals of Weight Loss and Management.**

  The *initial goal* of weight loss therapy is to reduce body weight by approximately 10 percent from baseline. If this goal is achieved, further weight loss can be attempted, if indicated through further evaluation.

  A *reasonable time line* for a 10 percent reduction in body weight is 6 months of therapy. For overweight patients with BMIs in the typical range of 27 to 35, a decrease of 300 to 500 kcal/day will result in weight losses of about ½ to 1 lb/week and a 10 percent loss in 6 months. For more severely obese patients with BMIs > 35, deficits of up to 500 to 1,000 kcal/day will lead to weight losses of about 1 to 2 lb/week and a 10 percent weight loss in 6 months. Weight loss at the rate of 1 to 2 lb/week (calorie deficit of 500 to 1,000 kcal/day) commonly occurs for up to 6 months. After 6 months, the rate of weight loss usually declines and weight plateaus because of a lesser energy expenditure at the lower weight.

  Experience reveals that lost weight usually will be regained unless a weight maintenance program consisting of dietary therapy, physical activity, and behavior therapy is continued indefinitely.

  After 6 months of weight loss treatment, efforts to maintain weight loss should be put in place. If more weight loss is needed, another attempt at weight reduction can be made. This will require further adjustment of the diet and physical activity prescriptions.

  For patients unable to achieve significant weight reduction, prevention of further weight gain is an important goal; such patients may also need to participate in a weight management program.

- **Strategies for Weight Loss and Weight Maintenance.**

  Dietary Therapy: A diet that is individually planned and takes into account the patient's overweight status in order to help create a deficit of 500 to 1,000 kcal/day should be an integral part of any weight loss program. A patient may choose a diet of 1,000 to 1,200 kcal/day for women and 1,200 to 1,500 kcal/day for men. Depending on the patient's risk status, the low-calorie diet (LCD) recommended should be consistent with the NCEP's Step I or Step II Diet (see page 74 of the guidelines). Besides decreasing saturated fat, total fats should be 30 percent or less of total calories. Reducing the percentage of dietary fat alone will not produce weight loss unless total calories are also reduced. Isocaloric replacement of fat with carbohydrates will reduce the percentage of calories from fat but will not cause weight loss. Reducing dietary fat, along with reducing dietary carbohydrates, usually will be needed to produce the caloric deficit needed for an acceptable weight loss. When fat intake is reduced, priority should be given to reducing saturated fat to enhance lowering of LDL-cholesterol levels. Frequent contacts with the practitioner during dietary therapy help to promote weight loss and weight maintenance at a lower weight.

  Physical Activity: An increase in physical activity is an important component of weight loss therapy, although it will not lead to substantially greater weight loss over 6 months. Most weight loss occurs because of decreased caloric intake. Sustained physical activity is most helpful in the prevention of weight regain. In addition, it has a benefit in reducing cardiovascular and diabetes risks beyond that produced by weight reduction alone. For most obese patients, exercise should be initiated slowly, and the intensity should be

visited on 6/14/2019

increased gradually. The exercise can be done all at one time or intermittently over the day. Initial activities may be walking or swimming at a slow pace. The patient can start by walking 30 minutes for 3 days a week and can build to 45 minutes of more intense walking at least 5 days a week. With this regimen, an additional expenditure of 100 to 200 calories per day can be achieved. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. This regimen can be adapted to other forms of physical activity, but walking is particularly attractive because of its safety and accessibility. Patients should be encouraged to increase "every day" activities such as taking the stairs instead of the elevator. With time, depending on progress and functional capacity, the patient may engage in more strenuous activities. Competitive sports, such as tennis and volleyball, can provide an enjoyable form of exercise for many, but care must be taken to avoid injury. Reducing sedentary time is another strategy to increase activity by undertaking frequent, less strenuous activities.

Behavior Therapy: Strategies, based on learning principles such as reinforcement, that provide tools for overcoming barriers to compliance with dietary therapy and/or increased physical activity are helpful in achieving weight loss and weight maintenance. Specific strategies include self-monitoring of both eating habits and physical activity, stress management, stimulus control, problem solving, contingency management, cognitive restructuring, and social support.

Combined Therapy: A combined intervention of behavior therapy, an LCD, and increased physical activity provides the most successful therapy for weight loss and weight maintenance. This type of intervention should be maintained for at least 6 months before considering pharmacotherapy.

Pharmacotherapy: In carefully selected patients, appropriate drugs can augment LCDs, physical activity, and behavior therapy in weight loss. Weight loss drugs that have been approved by the FDA for long-term use can be useful adjuncts to dietary therapy and physical activity for some patients with a BMI of ≥ 30 with no concomitant risk factors or diseases, and for patients with a BMI of ≥ 27 with concomitant risk factors or diseases. The risk factors and diseases considered important enough to warrant pharmacotherapy at a BMI of 27 to 29.9 are hypertension, dyslipidemia, CHD, type 2 diabetes, and sleep apnea. Continual assessment by the physician of drug therapy for efficacy and safety is necessary.

At the present time, sibutramine is available for long-term use. (Note: FDA approval of orlistat is pending a resolution of labeling issues and results of Phase III trials.) It enhances weight loss modestly and can help facilitate weight loss maintenance. Potential side effects with drugs, nonetheless, must be kept in mind. With sibutramine, increases in blood pressure and heart rate may occur. Sibutramine should not be used in patients with a history of hypertension, CHD, congestive heart failure, arrhythmias, or history of stroke. With orlistat, fat soluble vitamins may require replacement because of partial malabsorption. All patients should be carefully monitored for these side effects.

Weight Loss Surgery: Weight loss surgery is one option for weight reduction in a limited number of patients with clinically severe obesity, i.e., BMIs ≥ 40 or ≥ 35 with comorbid conditions. Weight loss surgery should be reserved for patients in whom efforts at medical therapy have failed and who are suffering from the complications of extreme obesity.

visited on 6/14/2019

Gastrointestinal surgery (gastric restriction [vertical gastric banding] or gastric bypass [Roux-en Y]) is an intervention weight loss option for motivated subjects with acceptable operative risks. An integrated program must be in place to provide guidance on diet, physical activity, and behavioral and social support both prior to and after the surgery.

- **Adapt Weight Loss Programs To Meet the Needs of Diverse Patients.** Standard treatment approaches for overweight and obesity must be tailored to the needs of various patients or patient groups. Large individual variation exists within any social or cultural group; furthermore, substantial overlap among subcultures occurs within the larger society. There is, therefore, no "cookbook" or standardized set of rules to optimize weight reduction with a given type of patient. However, to be more culturally sensitive and to incorporate patient characteristics in obesity treatment programs: consider and adapt the setting and staffing for the program; consider how the obesity treatment program integrates into other aspects of patient health care and self care; and expect and allow for program modifications based on patient responses and preferences.

The issues of weight reduction after age 65 involve such questions as: does weight loss reduce risk factors in older adults; are there risks associated with obesity treatment that are unique to older adults; and does weight reduction prolong the lives of older adults? Although there is less certainty about the importance of treating overweight at older ages than at younger ages, a clinical decision to forgo obesity treatment in older adults should be guided by an evaluation of the potential benefit of weight reduction and the reduction of risk for future cardiovascular events.

In the obese patient who smokes, smoking cessation is a major goal of risk factor management. Many well-documented health benefits accompany smoking cessation, but a major obstacle to cessation has been the attendant weight gain observed in about 80 percent of quitters. This weight gain averages 4.5 to 7 lb, but in 13 percent of women and 10 percent of men, weight gain exceeds 28 lb. Weight gain that accompanies smoking cessation has been quite resistant to most dietary, behavioral, or physical activity interventions.

The weight gained with smoking cessation is less likely to produce negative health consequences than would continued smoking. For this reason, smoking cessation should be strongly advocated regardless of baseline weight. Prevention of weight gain through diet and physical activity should be stressed. For practical reasons, it may be prudent to avoid initiating smoking cessation and weight loss therapy simultaneously. If weight gain ensues after smoking cessation, it should be managed vigorously according to the guidelines outlined in this report. Although short-term weight gain is a common side effect of smoking cessation, this gain does not rule out the possibility of long-term weight control.

SUMMARY OF EVIDENCE-BASED RECOMMENDATIONS

A Advantages of Weight Loss

The recommendation to treat overweight and obesity is based not only on evidence that relates obesity to increased mortality but also on RCT evidence that weight loss reduces risk factors for disease. Thus, weight loss may not only help control diseases worsened by obesity, it may also help decrease the likelihood of developing these diseases. The panel reviewed RCT evidence to determine the effect of weight loss on blood pressure and hypertension, serum/plasma lipid concentrations, and fasting blood glucose and fasting insulin. Recommendations focusing on

visited on 6/14/2019

these conditions underscore the advantages of weight loss.

1. Blood Pressure

To evaluate the effect of weight loss on blood pressure and hypertension, 76 articles reporting RCTs were considered for inclusion in these guidelines. Of the 45 accepted articles, 35 were lifestyle trials and 10 were pharmacotherapy trials. There is strong and consistent evidence from these lifestyle trials in both overweight hypertensive and nonhypertensive patients that weight loss produced by lifestyle modifications reduces blood pressure levels. Limited evidence exists that decreases in abdominal fat will reduce blood pressure in overweight nonhypertensive individuals, although not independent of weight loss, and there is considerable evidence that increased aerobic activity to increase cardiorespiratory fitness reduces blood pressure (independent of weight loss). There is also suggestive evidence from randomized trials that weight loss produced by most weight loss medications, except for sibutramine, in combination with adjuvant lifestyle modifications will be accompanied by reductions in blood pressure. Based on a review of the evidence from the 45 RCT blood pressure articles, the panel makes the following recommendation:

Weight loss is recommended to lower elevated blood pressure in overweight and obese persons with high blood pressure. Evidence Category A.

---

2. Serum/Plasma Lipids

Sixty-five RCT articles were evaluated for the effect of weight loss on serum/plasma concentrations of total cholesterol, LDL-cholesterol, very low-density lipoprotein (VLDL)-cholesterol, triglycerides, and HDL-cholesterol. Studies were conducted on individuals over a range of obesity and lipid levels. Of the 22 articles accepted for inclusion in these guidelines, 14 RCT articles examined lifestyle trials while the remaining 8 articles reviewed pharmacotherapy trials. There is strong evidence from the 14 lifestyle trials that weight loss produced by lifestyle modifications in overweight individuals is accompanied by reductions in serum triglycerides and by increases in HDL-cholesterol. Weight loss generally produces some reductions in serum total cholesterol and LDL-cholesterol. Limited evidence exists that a decrease in abdominal fat correlates with improvements in lipids, although the effect may not be independent of weight loss, and there is strong evidence that increased aerobic activity to increase cardiorespiratory fitness favorably affects blood lipids, particularly if accompanied by weight loss. There is suggestive evidence from the eight randomized pharmacotherapy trials that weight loss produced by weight loss medications and adjuvant lifestyle modifications, including caloric restriction and physical activity, does not result in consistent effects on blood lipids. The following recommendation is based on the review of the data in these 22 RCT articles:

Weight loss is recommended to lower elevated levels of total cholesterol, LDL-cholesterol, and triglycerides, and to raise low levels of HDL-cholesterol in overweight and obese persons with dyslipidemia. Evidence Category A.

---

3. Blood Glucose

To evaluate the effect of weight loss on fasting blood glucose and fasting insulin levels, 49 RCT articles were reviewed for inclusion in these guidelines. Of the 17 RCT articles accepted, 9 RCT articles examined lifestyle therapy trials and 8 RCT articles considered the effects of pharmacotherapy on weight loss and subsequent changes in blood glucose. There is strong evidence from the nine lifestyle therapy trials that

visited on 6/14/2019

weight loss produced by lifestyle modification reduces blood glucose levels in overweight and obese persons without diabetes, and weight loss reduces blood glucose levels and HbA_{1c} in some patients with type 2 diabetes. There is suggestive evidence that decreases in abdominal fat will improve glucose tolerance in overweight individuals with impaired glucose tolerance, although not independent of weight loss; and there is limited evidence that increased cardiorespiratory fitness improves glucose tolerance in overweight individuals with impaired glucose tolerance or diabetes, although not independent of weight loss. In addition, there is suggestive evidence from randomized trials that weight loss induced by weight loss medications does not appear to improve blood glucose levels any better than weight loss through lifestyle therapy in overweight persons both with and without type 2 diabetes. Based on a full review of the data in these 17 RCT articles, the panel makes the following recommendation:

Weight loss is recommended to lower elevated blood glucose levels in overweight and obese persons with type 2 diabetes. Evidence Category A.

B Measurement of Degree of Overweight and Obesity

Patients should have their BMI and levels of abdominal fat measured not only for the initial assessment of the degree of overweight and obesity, but also as a guide to the efficacy of weight loss treatment. Although there are no RCTs that review measurements of overweight and obesity, the panel determined that this aspect of patient care warranted further consideration and that this guidance was deemed valuable. Therefore, the following four recommendations that are included in the Treatment Guidelines were based on nonrandomized studies as well as clinical experience.

1. BMI To Assess Overweight and Obesity

There are a number of accurate methods to assess body fat (e.g., total body water, total body potassium, bioelectrical impedance, and dual-energy X-ray absorptiometry), but no trial data exist to indicate that one measure of fatness is better than any other for following overweight and obese patients during treatment. Since measuring body fat by these techniques is often expensive and is not readily available, a more practical approach for the clinical setting is the measurement of BMI; epidemiological and observational studies have shown that BMI provides an acceptable approximation of total body fat for the majority of patients. Because there are no published studies that compare the effectiveness of different measures for evaluating changes in body fat during weight reduction, the panel bases its recommendation on expert judgment from clinical experience:

Practitioners should use the BMI to assess overweight and obesity. Body weight alone can be used to follow weight loss, and to determine efficacy of therapy. Evidence Category C.

---

2. BMI To Estimate Relative Risk

In epidemiological studies, BMI is the favored measure of excess weight to estimate relative risk of disease. BMI correlates both with morbidity and mortality; the relative risk for CVD risk factors and CVD incidence increases in a graded fashion with increasing BMI in all population groups. Moreover, calculating BMI is simple, rapid, and inexpensive, and can be applied generally to adults. The panel, therefore, makes this recommendation:

visited on 6/14/2019

The BMI should be used to classify overweight and obesity and to estimate relative risk of disease compared to normal weight. Evidence Category C.

3. Assessing Abdominal Fat

For the most effective technique for assessing abdominal fat content, the panel considered measures of waist circumference, waist-to-hip ratio (WHR), magnetic resonance imaging (MRI), and computed tomography. Evidence from epidemiological studies shows waist circumference to be a better marker of abdominal fat content than WHR, and that it is the most practical anthropometric measurement for assessing a patient's abdominal fat content before and during weight loss treatment. Computed tomography and MRI are both more accurate but impractical for routine clinical use. Based on evidence that waist circumference is a better marker than WHR—and taking into account that the MRI and computed tomography techniques are expensive and not readily available for clinical practice—the panel makes the following recommendation:

The waist circumference should be used to assess abdominal fat content. Evidence Category C.

4. Sex-Specific Measurements

Evidence from epidemiological studies indicates that a high waist circumference is associated with an increased risk for type 2 diabetes, dyslipidemia, hypertension, and CVD. Therefore, the panel judged that sex-specific cutoffs for waist circumference can be used to identify increased risk associated with abdominal fat in adults with a BMI in the range of 25 to 34.9. These cutpoints can be applied to all adult ethnic or racial groups. On the other hand, if a patient is very short, or has a BMI above the 25 to 34.9 range, waist cutpoints used for the general population may not be applicable. Based on the evidence from nonrandomized studies, the panel makes this recommendation:

For adult patients with a BMI of 25 to 34.9 kg/m^2, sex-specific waist circumference cutoffs should be used in conjunction with BMI to identify increased disease risks. Evidence Category C.

C Goals for Weight Loss

The general goals of weight loss and management are to reduce body weight, to maintain a lower body weight over the long term, and to prevent further weight gain. Evidence indicates that a moderate weight loss can be maintained over time if some form of therapy continues. It is better to maintain a moderate weight loss over a prolonged period than to regain from a marked weight loss.

1. Initial Goal of Weight Loss from Baseline

There is strong and consistent evidence from randomized trials that overweight and obese patients in well-designed programs can achieve a weight loss of as much as 10 percent of baseline weight. In the diet trials, an average of 8 percent of baseline weight was lost. Since this average includes persons who did not lose weight, an individualized goal of 10 percent is reasonable. The panel, therefore, recommends that:

The initial goal of weight loss therapy should be to reduce body weight by approximately 10 percent from baseline. With success, further weight loss can be attempted if indicated through further assessment. Evidence Category A.

visited on 6/14/2019

2. Amount of Weight Loss

Randomized trials suggest that weight loss at the rate of 1 to 2 lb/week (calorie deficit of 500 to 1,000 kcal/day) commonly occurs for up to 6 months.

Weight loss should be about 1 to 2 lb/week for a period of 6 months, with the subsequent strategy based on the amount of weight lost. Evidence Category B.

D How To Achieve Weight Loss

The panel reviewed relevant treatment strategies designed for weight loss that can also be used to foster long-term weight control and prevention of weight gain. The consequent recommendations emphasize the potential effectiveness of weight control using multiple interventions and strategies, including dietary therapy, physical activity, behavior therapy, pharmacotherapy, and surgery, as well as combinations of these strategies.

1. Dietary Therapy

The panel reviewed 86 RCT articles to determine the effectiveness of diets on weight loss (including LCDs, very low-calorie diets (VLCDs), vegetarian diets, American Heart Association dietary guidelines, the NCEP's Step I diet with caloric restriction, and other low-fat regimens with varying combinations of macronutrients). Of the 86 articles reviewed, 48 were accepted for inclusion in these guidelines. These RCTs indicate strong and consistent evidence that an average weight loss of 8 percent of initial body weight can be obtained over 3 to 12 months with an LCD and that this weight loss effects a decrease in abdominal fat; and, although lower-fat diets without targeted caloric reduction help promote weight loss by producing a reduced caloric intake, lower-fat diets with targeted caloric reduction promote greater weight loss than lower-fat diets alone. Further, VLCDs produce greater initial weight losses than LCDs (over the long term of >1 year, weight loss is not different than that of the LCDs). In addition, randomized trials suggest that no improvement in cardiorespiratory fitness as measured by VO_2 max appears to occur in obese adults who lose weight on LCDs alone without physical activity. The following recommendations are based on the evidence extracted from the 48 accepted articles:

LCDs are recommended for weight loss in overweight and obese persons. Evidence Category A. Reducing fat as part of an LCD is a practical way to reduce calories. Evidence Category A.

Reducing dietary fat alone without reducing calories is not sufficient for weight loss. However, reducing dietary fat, along with reducing dietary carbohydrates, can facilitate caloric reduction. Evidence Category A.

A diet that is individually planned to help create a deficit of 500 to 1,000 kcal/day should be an intregal part of any program aimed at achieving a weight loss of 1 to 2 lb/week. Evidence Category A.

---

2. Physical Activity

Effects of Physical Activity on Weight Loss

Twenty-three RCT articles were reviewed to determine the effect of physical activity on weight loss, abdominal fat (measured by waist circumference), and changes in cardiorespiratory fitness (VO_2 max). Thirteen of these articles were accepted for inclusion in these guidelines. A review of these articles reveals strong evidence that physical activity alone, i.e., aerobic exercise, in obese adults results in modest weight loss and that physical activity in overweight and obese

visited on 6/14/2019

adults increases cardiorespiratory fitness, independent of weight loss. Randomized trials suggest that increased physical activity in overweight and obese adults reduces abdominal fat only modestly or not at all, and that regular physical activity independently reduces the risk for CVD. The panel's recommendation on physical activity is based on the evidence from these 13 articles:

Physical activity is recommended as part of a comprehensive weight loss therapy and weight control program because it: (1) modestly contributes to weight loss in overweight and obese adults (Evidence Category A), (2) may decrease abdominal fat (Evidence Category B), (3) increases cardiorespiratory fitness (Evidence Category A), and (4) may help with maintenance of weight loss (Evidence Category C).

Physical activity should be an integral part of weight loss therapy and weight maintenance. Initially, moderate levels of physical activity for 30 to 45 minutes, 3 to 5 days a week, should be encouraged. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. Evidence Category B.

Effects of Physical Activity and Diet on Weight Loss (Combined Therapy)

Twenty-three RCT articles were reviewed to determine the effects on body weight of a combination of a reduced-calorie diet with increased physical activity. Fifteen of these articles were accepted for inclusion in the guidelines. These articles contain strong evidence that the combination of a reduced-calorie diet and increased physical activity produces greater weight loss than diet alone or physical activity alone, and that the combination of diet and physical activity improves cardiorespiratory fitness as measured by VO_2 max in overweight and obese adults when compared to diet alone. The combined effect of a reduced calorie diet and increased-physical activity seemingly produced modestly greater reductions in abdominal fat than either diet alone or physical activity alone, although it has not been shown to be independent of weight loss. The panel's following recommendations are based on the evidence from these articles:

The combination of a reduced calorie diet and increased physical activity is recommended since it produces weight loss that may also result in decreases in abdominal fat and increases in cardiorespiratory fitness. Evidence Category A.

---

3. Behavior Therapy

Thirty-six RCTs were reviewed to evaluate whether behavior therapy provides additional benefit beyond other weight loss approaches, as well as to compare various behavioral techniques. Of the 36 RCTs reviewed, 22 were accepted. These RCTs strongly indicate that behavioral strategies to reinforce changes in diet and physical activity in obese adults produce weight loss in the range of 10 percent over 4 months to 1 year. In addition, no one behavior therapy appeared superior to any other in its effect on weight loss; multimodal strategies appear to work best and those interventions with the greatest intensity appear to be associated with the greatest weight loss. Long-term follow-up of patients undergoing behavior therapy shows a return to baseline weight for the great majority of subjects in the absence of continued behavioral intervention. Randomized trials suggest that behavior therapy, when used in combi-

visited on 6/14/2019

nation with other weight loss approaches, provides additional benefits in assisting patients to lose weight short-term, i.e., 1 year (no additional benefits are found at 3 to 5 years). The panel found little evidence on the effect of behavior therapy on cardiorespiratory fitness. Evidence from these articles provided the basis for the following recommendation:

Behavior therapy is a useful adjunct when incorporated into treatment for weight loss and weight maintenance. Evidence Category B.

There is also suggestive evidence that patient motivation is a key component for success in a weight loss program. The panel, therefore, makes the following recommendation:

Practitioners need to assess the patient's motivation to enter weight loss therapy; assess the readiness of the patient to implement the plan and then take appropriate steps to motivate the patient for treatment. Evidence Category D.

---

4. Summary of Lifestyle Therapy

There is strong evidence that combined interventions of an LCD, increased physical activity, and behavior therapy provide the most successful therapy for weight loss and weight maintenance. The panel makes the following recommendation:

Weight loss and weight maintenance therapy should employ the combination of LCDs, increased physical activity, and behavior therapy. Evidence Category A.

---

5. Pharmacotherapy

A review of 44 pharmacotherapy RCT articles provides strong evidence that pharmacological therapy (which has generally been studied along with lifestyle modification, including diet and physical activity) using dexfenfluramine, sibutramine, orlistat, or phentermine/fenfluramine results in weight loss in obese adults when used for 6 months to 1 year. Strong evidence also indicates that appropriate weight loss drugs can augment diet, physical activity, and behavior therapy in weight loss. Adverse side effects from the use of weight loss drugs have been observed in patients. As a result of the observed association of valvular heart disease in patients taking fenfluramine and dexfenfluramine alone or in combination, these drugs have been withdrawn from the market. Weight loss drugs approved by the FDA for long-term use may be useful as an adjunct to diet and physical activity for patients with a BMI of ≥ 30 with no concomitant obesity-related risk factors or diseases, as well as for patients with a BMI of ≥ 27 with concomitant risk factors or diseases; moreover, using weight loss drugs singly (not in combination) and starting with the lowest effective doses can decrease the likelihood of adverse effects. Based on this evidence, the panel makes the following recommendation:

Weight loss drugs approved by the FDA may be used as part of a comprehensive weight loss program, including dietary therapy and physical activity for patients with a BMI of ≥ 30 with no concomitant obesity-related risk factors or diseases, and for patients with a BMI of ≥ 27 with concomitant obesity-related risk factors or diseases. Weight loss drugs should never be used without concomitant lifestyle modifications. Continual assessment of drug therapy for efficacy and safety is necessary. If the drug is efficacious in helping the patient to lose and/or maintain weight loss

visited on 6/14/2019

and there are no serious adverse effects, it can be continued. If not, it should be discontinued. Evidence Category B.

---

6. Weight Loss Surgery

The panel reviewed 14 RCTs that examined the effect of surgical procedures on weight loss; 8 were deemed appropriate. All of the studies included individuals who had a BMI of 40 kg/m^2 or above, or a BMI of 35 to 40 kg/m^2 with comorbidity. These trials provide strong evidence that surgical interventions in adults with clinically severe obesity, i.e., BMIs ≥ 40 or ≥ 35 with comorbid conditions, result in substantial weight loss, and suggestive evidence that lifelong medical surveillance after surgery is necessary. Therefore, the panel makes the following recommendation:

Weight loss surgery is an option for carefully selected patients with clinically severe *obesity (BMI ≥ 40 or ≥ 35 with comorbid conditions) when less invasive methods of weight loss have failed and the patient is at high risk for obesity-associated morbidity or mortality. Evidence Category B.*

E Goals for Weight Loss Maintenance

Once the goals of weight loss have been successfully achieved, maintenance of a lower body weight becomes the challenge. Whereas studies have shown that weight loss is achievable, it is difficult to maintain over a long period of time (3 to 5 years). In fact, the majority of persons who lose weight, once dismissed from clinical therapy, frequently regain it—so the challenge to the patient and the practitioner is to maintain the weight loss. Successful weight reduction thus depends on continuing a maintenance program on a long-term basis. In the past, obtaining the goal of weight loss has been considered the end of weight loss therapy. Observation, monitoring, and encouragement of patients who have successfully lost weight should be continued long term. The panel's recommendations on weight loss maintenance are derived from RCT evidence as well as nonrandomized and observational studies.

1. Weight Maintenance Phase

RCTs from the Behavior Therapy section above suggest that lost weight usually will be regained unless a weight maintenance program consisting of dietary therapy, physical activity, and behavior therapy is continued indefinitely. Drug therapy in addition may be helpful during the weight maintenance phase. The panel also reviewed RCT evidence that considered the rate of weight loss and the role of weight maintenance. These RCTs suggest that after 6 months of weight loss treatment, efforts to maintain weight loss are important. Therefore, the panel recommends the following:

After successful weight loss, the likelihood of weight loss maintenance is enhanced by a program consisting of dietary therapy, physical activity, and behavior therapy which should be continued indefinitely. Drug therapy can also be used. However, drug safety and efficacy beyond 1 year of total treatment have not been established. Evidence Category B.

A weight maintenance program should be a priority after the initial 6 months of weight loss therapy. Evidence Category B.

Strong evidence indicates that better weight loss results are achieved with dietary therapy when the duration of the intervention is at least 6 months. Suggestive evidence also indicates that during dietary therapy, frequent contacts

visited on 6/14/2019

between professional counselors and patients promote weight loss and maintenance. Therefore, the panel recommends the following:

The literature suggests that weight loss and weight maintenance therapies that provide a greater frequency of contacts between the patient and the practitioner and are provided over the long term should be utilized whenever possible. This can lead to more successful weight loss and weight maintenance. Evidence Category C.

F Special Treatment Groups

The needs of special patient groups must be addressed when considering treatment options for overweight and obesity. The guidelines focus on three such groups including smokers, older adults, and diverse patient populations.

1. Smokers

Cigarette smoking is a major risk factor for cardiopulmonary disease. Because of its attendant high risk, smoking cessation is a major goal of risk-factor management. This aim is especially important in the overweight or obese patient, who usually carries excess risk from obesity-associated risk factors. Thus, smoking cessation in these patients becomes a high priority for risk reduction. Smoking and obesity together apparently compound cardiovascular risk, but fear of weight gain upon smoking cessation is an obstacle for many patients. Therefore, the panel recommends that:

All smokers, regardless of their weight status, should quit smoking. Evidence Category A. Prevention of weight gain should be encouraged and if weight gain does occur, it should be treated through dietary therapy, physical activity, and behavior therapy, maintaining the primary emphasis on the importance of abstinence from smoking. Evidence Category C.

2. Older Adults

The general nutritional safety of weight reduction at older ages is of concern because restrictions on overall food intake due to dieting could result in inadequate intake of protein or essential vitamins or minerals. In addition, involuntary weight loss indicative of occult disease might be mistaken for success in voluntary weight reduction. These concerns can be alleviated by providing proper nutritional counseling and regular body weight monitoring in older persons for whom weight reduction is prescribed. A review of several studies indicates that age alone should not preclude treatment for obesity in adult men and women. In fact, there is evidence from RCTs that weight reduction has similar effects in improving cardiovascular disease risk factors in older and younger adults. Therefore, in the panel's judgment:

A clinical decision to forego obesity treatment in older adults should be guided by an evaluation of the potential benefits of weight reduction for day-to-day functioning and reduction of the risk of future cardiovascular events, as well as the patient's motivation for weight reduction. Care must be taken to ensure that any weight reduction program minimizes the likelihood of adverse effects on bone health or other aspects of nutritional status. Evidence Category D.

visited on 6/14/2019

3. Diverse Patient Populations

Standard obesity treatment approaches should be tailored to the needs of various patients or patient groups. It is, however, difficult to determine from the literature how often this occurs, how specific programs and outcomes are influenced by tailoring, and whether it makes weight loss programs more effective. After reviewing two RCTs, four cross-sectional studies, and four intervention studies, as well as additional published literature on treatment approaches with diverse patient populations, the panel recommends the following:

The possibility that a standard approach to weight loss will work differently in diverse patient populations must be considered when setting expectations about treatment outcomes. Evidence Category B.

---

visited on 6/14/2019

Introduction

I

A Rationale for Guidelines Development

An estimated 97 million adults in the United States are overweight or obese, [1] a condition that substantially raises their risk of morbidity from hypertension, [2-6] type 2 diabetes, [7-10] stroke, [11-13] gallbladder disease, [14, 15] osteoarthritis, [16-18] sleep apnea and respiratory problems, [19-21] and endometrial, breast, prostate, and colon cancers. [22-24] As a major contributor to preventive death in the United States today, [25] overweight and obesity pose a major public health challenge. Not only is the prevalence of this serious medical condition soaring among adults (between 1960 and 1994, overweight increased from 30.5 to 32 percent among adults ages 20 to 74 and obesity increased from 12.8 percent to 22.5 percent), but it is also affecting ever greater numbers of American youth and exacting a particularly harsh toll from low income women and minorities. The Third National Health and Nutrition Examination Survey (NHANES III) estimated that 13.7 percent of children and 11.5 percent of adolescents are overweight, while a number of smaller, ethnic-specific studies suggest that overweight and obesity may afflict up to 30 to 40 percent of children and youth from minority populations. [26, 27]

The prevalence of overweight and obesity in adults in the United States increased markedly during the last decade. According to NHANES III data, 54.9 percent of U.S. adults aged 20 years and older are either overweight or obese; 32.6 percent are overweight, defined as having a body mass index (BMI)* of 25.0 to 29.9 kg/m^2; and 22.3 percent are obese with a BMI of ≥ 30 kg/m^2. [1] The panel acknowledges that overweight and obesity are not mutually exclusive; obese persons are also overweight. Since overweight and obesity lead to increased morbidity and mortality, these figures demonstrate the enormity of the public health problem, as well as the clinical problem, of overweight and obesity in this country.

In this report, overweight is defined as a BMI of 25.0 to 29.9 kg/m^2 and obesity as a BMI of ≥ 30 kg/m^2. The rationale behind these definitions is based on epidemiological data that show increases in mortality with BMIs above 25 kg/m^2. [28-32] The increase in mortality, however, tends to be modest until a BMI of 30 kg/m^2 is reached. [28, 31, 32] For persons with a BMI of ≥ 30 kg/m^2, mortality rates from all causes, and especially from cardiovascular disease, are generally increased by 50 to 100 percent above that of persons with BMIs in the range of 20 to 25 kg/m^2. [28, 31, 32]

Overweight and obesity result from a complex interaction between genes and the environment characterized by long-term energy imbalance due to a sedentary lifestyle, excessive caloric consumption, or both. [33] They develop in a sociocultural environment characterized by mechanization, sedentary lifestyle, and ready access to abundant food. Attempts to prevent overweight and obesity are difficult to both study and achieve. Indeed, few research efforts have investigated either individual or community-based prevention strategies. [34]

* The BMI is calculated as follows: BMI = weight (kg)/ height squared (m^2). Conversion: [weight (pounds)/height (inches) ^{2}] x 703 (1 lb = 0.45 kg) (1.in = 2.54 cm = 0.0254 m). A BMI of 25 is equivalent to 184 lb in a 6'0" person and to 155 lb in one5'6". A BMI of 30 is equivalent to 221 lb in a 6'0" person and to 186 lb in one 5'6". (The conversion of BMI according to weight for height is provided in Appendix V.)

visited on 6/14/2019

A substantial body of research, however, does exist on the health risks of overweight and obesity, and on methods for treatment. This report, which bases its recommendations primarily on published evidence, emphasizes the important role of primary care practitioners in evaluating all overweight and obese adults and promoting weight control through the use of multiple interventions and strategies tailored to particular patient needs. Although the recommendations and guidelines included in this report focus on the clinical assessment and treatment of overweight and obese patients, a second important goal is to encourage primary care practitioners to take an active role in preventing inappropriate weight gain among all their patients.

B Objectives of the Guidelines

- To identify, evaluate, and summarize published information about the assessment and treatment of overweight and obesity;
- To provide evidence-based guidelines for physicians, other health care practitioners, and health care organizations for the evaluation and treatment of overweight and obesity in adults; and
- To identify areas for future research.

C Guideline Development Methodology

The National Heart, Lung, and Blood Institute's (NHLBI) Obesity Education Initiative, in cooperation with the National Institute of Diabetes and Digestive and Kidney Diseases, convened the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. These guidelines address the treatment of overweight and obesity only in adults, but it is the judgment of the panel that guidelines for treating obesity in children are equally important and should be drafted as soon as possible (see Appendix III for information on overweight and obesity in children). The panel's charge was to develop evidence-based clinical guidelines for primary care practitioners; however, the guidelines should also be useful for certain specialists. The decision to develop "evidence-based" guidelines was based on the increased attention being paid to clinical practice guidelines from methodologists, professional associations, third-party payers and policy makers, and the NHLBI's mission to analyze research results with the goal of providing information that may enable health care practitioners to enhance their ability to detect, treat, and prevent disease. [35] In keeping with this approach, the panel

- defined topics to be included in the guidelines;
- developed an evidence model depicting the strategy of inquiry for each area of scientific interest;
- established criteria for searching and abstracting the literature;
- constructed and reviewed evidence tables of individual studies and summary tables of studies falling within a specific category of evidence; and
- identified the level or strength of the evidence that served as the basis for the recommendations.

A complete description of the methodology used to develop the guidelines is included in the report as Appendix I.A.1.

The guidelines are based primarily on a systematic review of the published scientific literature in English found in MEDLINE from January 1980 through September 1997. This was done in the interest of time and economy. This information was supplemented by material provided by the panel and an ancestral search of appropriate references in eligible articles. The literature was searched and systematically reviewed by

- establishing *a priori* eligibility criteria for inclusion of studies;

visited on 6/14/2019

- reviewing titles and abstracts to select promising articles;
- reviewing these full articles; and
- compiling evidence tables summarizing those articles that met the inclusion criteria.

As a priority, the panel identified randomized controlled trials (RCTs) as the strongest level of evidence for the evaluation of treatment efficacy. Only RCTs lasting 4 months or more were considered. The only exceptions were a few 3-month trials in the diet and pharmacotherapy sections. With the assistance of the San Antonio Cochrane Center*, 394 publications of RCTs were reviewed for data abstraction. RCT evidence serves as the basis for the many recommendations contained in these guidelines related to treatment efficacy. Instances when the panel had to make recommendations where RCTs were insufficient or absent are clearly indicated in the text. These instances most often pertain to issues of obesity assessment, classification, and measurement where RCT evidence would not be appropriate to answer the question. These issues are best addressed by epidemiological/observational studies of large population groups. In those few cases where the literature could not credibly support a recommendation, panel members relied on clinical experience and knowledge.

The panel recognizes that by relying primarily on only published literature, a publication bias (a positive result is more likely to be published than is a negative result) may exist, so that treatment efficacy may be overstated. [36-39] However, no other reliable sources of information were available.

The targeted population for the guidelines is all overweight and obese adults (18 years of age and older) with a BMI ≥ 25, with particular emphasis on those with cardiovascular risk factors. While the guidelines are appropriate for patients with a BMI ≥ 40, their care is often complicated and may require surgery. These guidelines are not intended for pregnant women. Excluded from the analysis were adults with pharmacologically induced obesity and those with specific genetic syndromes associated with overweight and obesity.

The selection of weight loss interventions to be considered was determined by the literature review. Namely, the panel considered any topic for which articles meeting inclusion criteria were found, including diet, physical activity, behavior therapy, pharmacological therapy, surgery, and combinations of these modalities. No clinical interventions were excluded at the outset. However, the panel did not consider other interventions such as acupuncture or hypnosis, for which no randomized trial articles were available. Clinical interventions to prevent further weight gain in individuals already overweight were also considered relevant.

The panel also evaluated population factors and clinical situations that might potentially influence the physiological, medical, behavioral, or sociocultural context for obesity identification and treatment. Evidence on special populations and situations was provided from RCTs and non-RCTs when available, but in many cases such evidence was meager. Population factors and clinical situations selected for special consideration for obesity classification and treatment were age, gender, race/ethnicity, socioeconomic status, pregnancy, eating disorders, sleep apnea, extreme obesity (BMI ≥ 40), concurrent treatment of other major conditions (such as heart disease or diabetes), and treatment of obesity in conjunction with smoking cessation. When evidence on these special populations or clinical conditions was insufficient to meet standards for inclusion in the main text of the guidelines, relevant issues are identified for the user, and in some cases are cross-referenced to an appendix (see Appendix III) or discussed in sidebar text as a commentary.

* The San Antonio Cochrane Center is one of 12 centers around the world that comprise the Cochrane Collaboration. The Cochrane Collaboration is an international organization established in 1993 whose mission is to prepare, maintain, and disseminate systematic reviews and meta-analyses of health care interventions.

visited on 6/14/2019

The panel recognized the possibility of an advocacy "bias" due to the large number of panel members drawn from organizations with an advocacy role in the treatment of obesity. As a result, it was agreed to obtain formal external reviews of the document from 59 professional societies, consumer groups and goverment agencies representing a wide spectrum of expertise and concern about obesity.

The format for those sections of the report based on the RCT evidence begins with an evidence statement followed by the rationale for that statement. At the end of a series of related evidence statements, a recommendation is given.

Each evidence statement (other than those with no available evidence) and each recommendation is categorized by a level of evidence (A through D) as described below. Statements for which there is no available evidence are so indicated.

Category A: Evidence is from endpoints of well-designed RCTs (or trials that depart only minimally from randomization) that provide a consistent pattern of findings in the population for which the recommendation is made. Category A therefore requires substantial numbers of studies involving substantial numbers of participants.

Category B: Evidence is from endpoints of intervention studies that include only a limited number of RCTs, post-hoc or subgroup analysis of RCTs, or meta-analysis of RCTs. In general, Category B pertains when few randomized trials exist, they are small in size, and the trial results are somewhat inconsistent, or the trials were undertaken in a population that differs from the target population of the recommendation.

Category C: Evidence is from outcomes of uncontrolled or nonrandomized trials or from observation studies.

Category D: Expert judgment is based on the panel's synthesis of evidence from experimental research described in the literature and/or derived from the consensus of panel members based on clinical experience or knowledge that does not meet the above-listed criteria. This category is used only in cases where the provisions of some guidance was deemed valuable but an adequately compelling clinical literature addressing the subject of the recommendation was deemed insufficient to justify placement in one of the other categories (A through C).

In applying these guidelines, the reader should note some caveats:

- The emphasis of these guidelines was to identify effective interventions, not to rank-order them in terms of their efficacy or effectiveness. The panel chose not to emphasize comparisons among interventions because there were few studies that compared long-term outcomes. Also, since individual preferences and circumstances often dictate choice of therapy, the panel wished to present a menu of options rather than a ranked list of choices.
- When no evidence was available on the efficacy of a treatment, the panel usually rendered no opinion. An absence of studies should not be confused with an absence of effect. While clinicians may prefer to use proven therapies rather than untested ones, the lack of testing does not denote that the untested therapy does not work.
- The limitations of RCTs must be kept in mind. The RCT is the primary method for demonstrating efficacy. Often, participants enrolled in RCTs differ from the individuals in a primary care practice, and effectiveness in the community may differ from efficacy as measured in an RCT.
- The potential exists for misinterpretation of clinical trial results. Analysis of endpoints not specified at the outset, or post hoc or subgroup analysis, should be viewed as hypothesis-generating rather than hypothesis-testing.

visited on 6/14/2019

D Statement of Assumptions

The panel has made every attempt to base its recommendations on published evidence, with particular attention to RCTs. Data from RCTs provide the strongest evidence regarding the impact of an intervention. The RCT literature predominantly describes short-term outcomes (< 1 year), although there are a small number of published RCTs of intermediate and long-term treatment and maintenance of weight loss. The panel chose to examine RCTs lasting 4 months or longer as their first priority. However, there are some 3-month studies included in the diet and pharmacotherapy sections.

Evidence of beneficial effects of weight reduction on risk factors and on diseases with which obesity is associated, and evidence of the association of obesity and mortality, are also available in the non-RCT epidemiological literature. Therefore, although the treatment recommendations in these guidelines are derived primarily from RCT evidence, they also come in part from the considered judgment of the expert panel members who weighed the non-RCT epidemiological evidence.

In setting forth its recommendations, the panel assumes that, for most individuals, the benefits of weight loss on overall health outweigh the harmful effects, and that weight loss can be maintained in many individuals with resulting long-term health benefits. The recommendations apply to all segments of the adult population, although apparent differences in applicability have been considered and some guidance is provided with respect to certain sectors of the population such as the elderly and in clinical situations. This additional guidance is in the form of notations within the text and is sometimes supplemented by an appendix (Appendix III).

E Intended Users of These Guidelines

These guidelines were developed primarily for use by physicians and associated health professionals in clinical practice. They should also be useful to managed care organizations or other groups that define benefit plans for patients or handle health care resources. Users of these guidelines are encouraged to note text and appendix references to situations in which weight reduction treatment may be contraindicated or may involve special treatment techniques or safety considerations (e.g., in older adults or in certain sociocultural contexts).

These guidelines also provide a state-of-the-art review of the scientific basis of the relation between obesity and major disease endpoints and of the scientific rationale for the management of the overweight and obese patients. The systematic assessment of the literature contained in this document should be a valuable resource to health care policy makers and clinical investigators.

visited on 6/14/2019

2

Overweight and Obesity: Background

A Health and Economic Costs

In 1973, and again in 1977, the John E. Fogarty International Center at the National Institutes of Health (NIH), as part of its preventive medicine series, sponsored two conferences that dealt with obesity as a public health problem; controversy was apparent regarding the cause-and-effect relationship between obesity and ill health. [40, 41] In 1985, an NIH Consensus Development Conference was held on the health implications of obesity. This conference provided important national recognition that obesity is a serious health condition that leads to increased morbidity and mortality. The Consensus Development Conference concluded that both prevention and treatment of obesity were medical priorities in the United States [42]. In that conference, the terms 'overweight' and 'obesity' were defined as part of a continuum of increasing health risk.

In 1990, the Nation's health goals for the year 2000 were set forth with the release of Healthy People 2000 [43], in which a national goal to reduce the prevalence of overweight was articulated. In 1993, the Deputy Assistant Secretary for Health (J. Michael McGinnis) and the former Director of the Centers for Disease Control and Prevention (CDC) (William Foege) co-authored a journal article, "Actual Causes of Death in the U.S." It concluded that a combination of dietary factors and sedentary activity patterns accounts for at least 300,000 deaths each year, and, obesity was a key contributor. [25] In 1995, the Institute of Medicine issued a report that expressed concern about the growing prevalence of overweight and obesity in this country, and suggested ways to evaluate various weight loss and weight maintenance programs available to U.S. consumers. [44]

1. Prevalence and Time Trends

Nationally representative U.S. health examination surveys, in which weight and height were measured in samples of the population, date back to 1960. Beginning with the Second National Health and Nutrition Examination Survey (NHANES II) (1976-1980), the definition of overweight that has been used to compare these epidemiologic surveys has been a statistical one that corresponded to the 85th percentile of body mass index (BMI) for men and women aged 20 through 29 years in NHANES II with no particular relation to a specific increase in disease risk. [45] Adults in these surveys have been categorized as overweight with a BMI ≥ 27.8 kg/m^2 for men and ≥ 27.3 kg/m^2 for women. [45] The rationale for using persons aged 20 to 29 years as the reference population is supported largely by the observation that the increases in body weight after age 29 that commonly occur with aging are attributable primarily to fat accumulation. [46,47] However, the BMI levels used for the definition of overweight and obesity are somewhat arbitrary, since the relationship between body weight and disease risk is continuous with the exception of the extremely underweight: disease risk increases as weight increases.

Figure 1 depicts data from several NHANES surveys using the panel's definition of over-

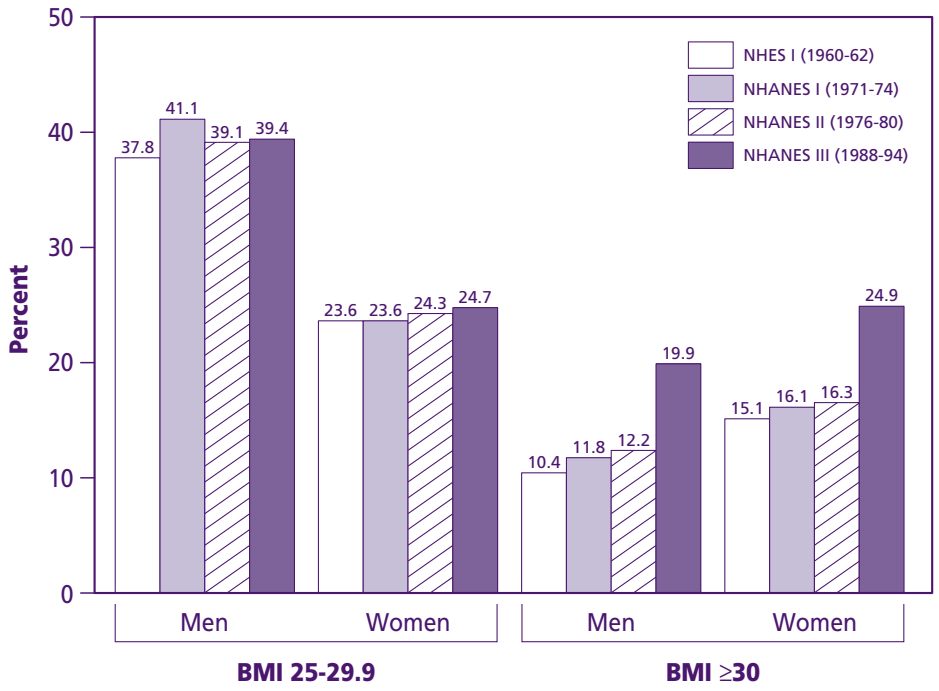
visited on 6/14/2019

weight as a BMI of 25 to 29.9 kg/m^2 and of obesity as a BMI of ≥ 30 kg/m^2. From 1960 to 1994, the prevalence of overweight increased slightly from 37.8 to 39.4 percent in men and from 23.6 to 24.7 percent in women (National Center for Health Statistics/CDC) [48]. In men and women together, overweight increased from 30.5 to 32.0 percent. [48] During the same time period, however, the prevalence of obesity increased from 10.4 to 19.9 percent in men and from 15.1 to 24.9 percent in women. In men and women together, obesity increased from 12.8 to 22.5 percent. Most of the increase occurred in the past decade. In addition to adults, obesity in U.S. children increased markedly as well [49] (see Appendix III) and, if unchecked, portends an even greater increase in adult obesity in the future.

Table II-1 shows the combined prevalence of overweight and obesity, defined as a BMI of ≥ 25.0 kg/m^2, among persons aged 20 to 80 plus years, by age, race/ethnicity, and gender in the United States, 1960 to 1994. [48] The increase in overweight and obesity appears to have occurred among U.S. adults across all ages, genders, and racial/ethnic groups. The most recent NHANES III surveys, conducted from 1988-1994, reported that 59.4 percent of men and 50.7 percent of women in the United States are overweight or obese. The prevalence is much higher in non-Hispanic Black women (66.0 percent), in Mexican-American women (65.9 percent), and in Mexican-American men (63.9 percent).

Using the definition of obesity as a BMI of ≥ 30 kg/m^2, Table II-2 shows that in the United States, 19.5 percent of men and 25.0 percent of

Figure 1. Age-Adjusted Prevalence of Overweight (BMI 25-29.9) and Obesity (BMI ≥30)



Source: CDC/NCHS, United States, 1960-94 (ages 20-74 years)

visited on 6/14/2019

TABLE II-1:

COMBINED PREVALENCE OF OVERWEIGHT AND OBESITY (BMI≥25.0 KG/M²) AMONG ADULTS AGE 20 TO 80+ YEARS, BY GENDER, RACE/ETHNICITY, AND AGE: UNITED STATES, 1960-1994.[48]

Gender, race/ethnicity, age 20 years and older, age adjusted:	NHES I 1960-62 (age 20-74)	NHANES I 1971-74 (age 20-74)	NHANES II 1976-80 (age 20-74)	HHANES 1982-84 (age 20-74)	NHANES III 1988-94 (age ≥20)
Both Sexes	43.3	46.1	46.0		54.9
Men	48.2	52.9	51.4		59.4
Women	38.7	39.7	40.8		50.7
White men	48.8	53.7	52.3		61.0
White women	36.1	37.6	38.4		49.2
Black men	43.1	48.9	49.0		56.5
Black women	57.0	57.6	61.0		65.8
White, non-Hispanic men			52.0		60.6
White, non-Hispanic women			37.6		47.4
Black, non-Hispanic men			48.9		56.7
Black, non-Hispanic women			60.6		66.0
Mexican-American men				59.7	63.9
Mexican-American women				60.1	65.9
Age and gender-specific categories:					
Men					
20-29	39.9	38.6	37.0		43.1
30-39	49.6	58.1	52.6		58.1
40-49	53.6	63.6	60.3		65.5
50-59	54.1	58.4	60.8		73.0
60-69	52.9	55.6	57.4		70.3
70-79	36.0	52.7*	53.3*		63.1
80+	N/A**	N/A**	N/A**		50.6
Women					
20-29	17.0	23.2	25.0		33.1
30-39	32.8	35.0	36.8		47.0
40-49	42.3	44.6	44.4		52.7
50-59	55.0	52.2	52.8		64.4
60-69	63.1	56.2	56.5		64.0
70-79	57.4	55.9*	58.2*		57.9
80+	N/A**	N/A**	N/A**		50.1

* Prevalence for age 70 to 74 years ** Not available

visited on 6/14/2019

women are obese. [48] The prevalence of obesity is much higher in minority women, being 36.7 percent in non-Hispanic Black women and 33.3 percent in Mexican-American women.

2. Demographic Variations in Overweight and Obesity Prevalence

Although NHANES III data show that the prevalence of overweight and obesity is much higher in African-American and Mexican-American women than in white women or in men, these data provide ethnicity-specific estimates of overweight and obesity prevalence for only three racial-ethnic groups: non-Hispanic whites, non-Hispanic blacks, and Mexican-Americans. Examination survey data indicating a high overweight and obesity prevalence in other ethnic groups (e.g., for Puerto Ricans and Cuban-Americans) are available from the Hispanic HANES (HHANES) (1982-1984) [27] and for American Indians [26] and Pacific-Islander Americans, [50] from smaller population-specific studies (see Appendix III). The prevalence of overweight and obesity is generally higher for men and women in racial-ethnic minority populations than in U.S. whites, with the exception of Asian-Americans, for whom overweight and obesity prevalence is lower than in the general population. [51] In the 1982-1984 HHANES, the age-adjusted prevalence of a BMI of ≥ 27.3 in Puerto Rican women was 40 percent.[27] The Strong Heart Study reported the average prevalence of overweight using BMI ≥ 27.8 or ≥ 27.3 for men and women, respectively, in three groups of American Indians studied during 1988-1989 as follows: in Arizona, 67 percent of the men and 80 percent of the women; in Oklahoma, 67 percent of the men and 71 percent of the women; and in South Dakota and North Dakota, 54 percent of the men and 66 percent of the women. [52]

Women in the United States with low incomes or low education are more likely to be obese than those of higher socioeconomic status; the association of socioeconomic status with obesity is less consistent in men [53] (Appendix III). Obesity is less common after the age of 70 among both men and women, possibly due to a progressive decrease in BMI with increasing age past the fifth decade or to an excess in mortality associated with increasing BMI in the presence of increasing age. [1]

3. Economic Costs of Overweight and Obesity

Alarm about the increasing prevalence of overweight and obesity in the United States in recent years [54, 55] centers on the link between obesity and increased health risks, [42, 56] which translates into increased medical care and disability costs. [46,57] The total cost attributable to obesity amounted to $99.2 billion in 1995. Approximately $51.6 billion of these dollars were direct medical costs associated with diseases attributable to obesity. The direct costs also associated with obesity represent 5.7 percent of the national health expenditure within the United States. [58] The indirect costs attributable to obesity are $47.6 billion and are comparable to the economic costs of cigarette smoking. [58, 59] Indirect costs represent the value of lost output caused by morbidity and mortality, and may have a greater impact than direct costs at the personal and societal levels. [58]

Although a comprehensive cost analysis of obesity is beyond the scope of this panel, a systematic review of the literature identified studies estimating the current economic burden of obesity in several Western countries. [57, 60-63] Published estimates of the economic costs of obesity such as those noted above use the prevalence-based approach, assuming that obesity is causally related to a range of chronic illnesses. Estimating the economic benefits of weight loss requires details of long-term weight maintenance and the time course of risk reduction following weight loss, and ultimately must also consider the costs of treatment to reduce weight.

visited on 6/14/2019

TABLE II-2:

Prevalence of Obesity (BMI≥30.0 kg/m²) Among Adults Age 20 to 80+ years, by Gender, Race/Ethnicity, and Age: United States, 1960-1994.[48]

Gender, race/ethnicity, age 20 years and older, age adjusted:	NHES I 1960-62 (age 20-74)	NHANES I 1971-74 (age 20-74)	NHANES II 1976-80 (age 20-74)	HHANES 1982-84 (age 20-74)	NHANES III 1988-94 (age ≥20)
Both Sexes	12.8	14.1	14.4		22.3
Men	10.4	11.8	12.2		19.5
Women	15.1	16.1	16.3		25.0
White men	10.1	11.4	12.0		20.0
White women	13.7	14.7	14.9		23.5
Black men	13.9	15.9	15.2		20.6
Black women	25.0	28.6	30.2		36.5
White, non-Hispanic men			12.0		19.9
White, non-Hispanic women			14.8		22.7
Black, non-Hispanic men			15.0		20.7
Black, non-Hispanic women			30.0		36.7
Mexican-American men				15.4	20.6
Mexican-American women				25.4	33.3
Age and gender-specific categories:					
Men					
20-29	9.0	8.0	8.1		12.5
30-39	10.4	13.3	12.1		17.2
40-49	11.9	14.2	16.4		23.1
50-59	13.4	15.3	14.3		28.9
60-69	7.7	10.3*	13.5*		24.8
70-79	8.6	11.1**	13.6**		20.0
80+	N/A**	N/A	N/A		8.0
Women					
20-29	6.1	8.2	9.0		14.6
30-39	12.1	15.1	16.8		25.8
40-49	17.1	17.6	18.1		26.9
50-59	20.4	22.0	22.6		35.6
60-69	27.2	24.0*	22.0*		29.8
70-79	21.9	21.0	19.4**		25.0
80+	N/A**	N/A**	N/A		15.1

* Prevalence for age 70 to 74 years ** Not available

visited on 6/14/2019

B Prevention of Overweight and Obesity

Prevention of overweight and obesity is as important as treatment. Prevention includes primary prevention of overweight or obesity itself, secondary prevention or avoidance of weight regain following weight loss, and prevention of further weight increases in obese individuals unable to lose weight. [44, 64]

National and international observational data suggest that environmental and behavioral factors are likely to be important in the tendency of individuals within and between populations to be obese during childhood or to gain weight progressively with age during adulthood. [65] These factors are also influenced by the genetic makeup of individuals. There has been a paucity of intervention research to demonstrate how these factors can be manipulated to prevent obesity. [64] In two community studies, namely the Minnesota Heart Health Program and the Stanford Five City Study, multifaceted weight loss and weight control programs within the community were not associated with prevention of weight gain in longitudinally followed cohorts. [66] In another community study, the Pawtucket Heart Health Program, BMI levels did not change in the intervention cities while they increased in the comparison cities. [67] One obesity prevention study of American Indian children who are at high risk of becoming obese is under way. [68] Otherwise, the only long-term report suggesting an effective approach to obesity prevention is from follow-up of obese children in an experimental study in which they had been treated with or without a family-oriented treatment program. Long-term follow-up (10 years) of these children supported the importance of family involvement in reducing the progression of obesity. [69] One population-based randomized controlled pilot study of obesity prevention suggests that programs for weight gain prevention are feasible and effective in adults. [34] Another study in China has shown that the prevention of weight gain through diet, physical activity, and their combination can help prevent diabetes. [70]

It has been suggested that primary prevention of obesity should include environmentally based strategies that address major societal contributors to over-consumption of calories and inadequate physical activity such as food marketing practices, transportation patterns, and lack of opportunities for physical activity during the workday. [71,72] People at lower socioeconomic levels living in urban areas also lack access to physical activity sites. Such strategies will be essential for effective initial and long-term prevention of obesity for large numbers of individuals and for the community at large. Research is needed to clarify the role of societal policies, procedures, laws, and other factors that serve as disincentives to lifelong caloric balance. The importance of obesity prevention needs to be brought to the attention of health care payors and practitioners, employers, educators, and public officials as an important priority to be addressed in policies, programs, and direct services to individuals and families. The development and implementation of appropriate policies and programs will require outcomes research that identifies effective weight gain prevention approaches. These programs must be useful for multiple settings, including health care facilities, schools, worksites, community and religious institutions, and be applicable to a broad population. In the end, efforts should be made to make the general public more aware of the need to prevent overweight and obesity.

Efforts to understand the genetic, developmental, environmental, and behavioral underpinnings of obesity and to mount successful prevention strategies are particularly critical for populations in which overweight and obesity and related health problems such as diabetes are disproportionately prevalent; for example, women in lower socioeconomic groups and women and sometimes men in many racial/ethnic minority

visited on 6/14/2019

populations as described in Chapter 2.A.2 of this report. Public health approaches for preventing obesity, that is, approaches designed to reduce the difficulty for any given individual of adopting healthful eating and activity patterns, will particularly benefit the socially disadvantaged, who—compared to the more advantaged— may have less access to preventive health services and fewer feasible options for making changes in their daily routines and lifestyles. [73-75]

Primary care practitioners are an important element in preventing and managing obesity in the United States. Prevention of overweight and obesity in primary care settings is compatible with efforts to prevent their health consequences, through control of dyslipidemia, high blood pressure, and type 2 diabetes. Thus, both the quality and quantity of life may be enhanced through preventive strategies. As detailed elsewhere in this report, high blood pressure, high blood cholesterol, and type 2 diabetes should be aggressively treated in overweight patients and may be treated prior to and in conjunction with weight loss.

C Health Risks of Overweight and Obesity

1. Morbidity

Above a BMI of 20 kg/m^2 , morbidity for a number of health conditions increases as BMI increases. Higher morbidity in association with overweight and obesity has been observed for hypertension, [2-6, 76-80] type 2 diabetes, [7, 8, 10, 81, 82, 84-89] coronary heart disease (CHD), [11,42,86,88,90] stroke, [11-13] gallbladder disease, [14,15] osteoarthritis, [16-18, 91-95] sleep apnea and respiratory problems [21, 96-98] and some types of cancer (endometrial, breast, prostate, and colon). [107-115] Obesity is also associated with complications of pregnancy, menstrual irregularities, hirsutism, stress incontinence, and psychological disorders (depression). [112, 116-128]

The nature of obesity-related health risks is similar in all populations, although the specific level of risk associated with a given level of overweight or obesity may vary with race/ethnicity, and also with age, gender, and societal conditions. For example, the absolute risk of morbidity in chronic conditions such as CHD is highest in the aged population, while the relative risk of having CHD in obese versus nonobese individuals is highest in the middle adult years. [129-131] A high prevalence of diabetes mellitus in association with obesity is observed consistently across races/ethnicities, while the relative prevalence of hypertension and CHD in obese versus nonobese populations varies between groups.

The health risks of overweight and obesity are briefly described below:

1.a. Hypertension

Data from NHANES III show that the age-adjusted prevalence of high blood pressure increases progressively with higher levels of BMI in men and women (Figure 2). [2] High blood pressure is defined as mean systolic blood pressure ≥ 140 mm Hg, or mean diastolic blood pressure ≥ 90 mm Hg, or currently taking antihypertensive medication. The prevalence of high blood pressure in adults with BMI ≥ 30 is 38.4 percent for men and 32.2 percent for women, respectively, compared with 18.2 percent for men and 16.5 percent for women with BMI < 25, a relative risk of 2.1 and 1.9 for men and women, respectively. The direct and independent association between blood pressure and BMI or weight has been shown in numerous cross-sectional studies [3-5], including the large international study of salt (INTERSALT) carried out in more than 10,000 men and women. [6] INTERSALT reported that a 10 kg (22 lb) higher body weight is associated with 3.0 mm Hg higher systolic and 2.3 mm Hg higher diastolic blood pressure. [6] These differences in blood pressure translate into an estimated 12 percent increased risk for CHD and 24 percent

visited on 6/14/2019

increased risk for stroke. [132] Positive associations have also been shown in prospective studies. [76-80]

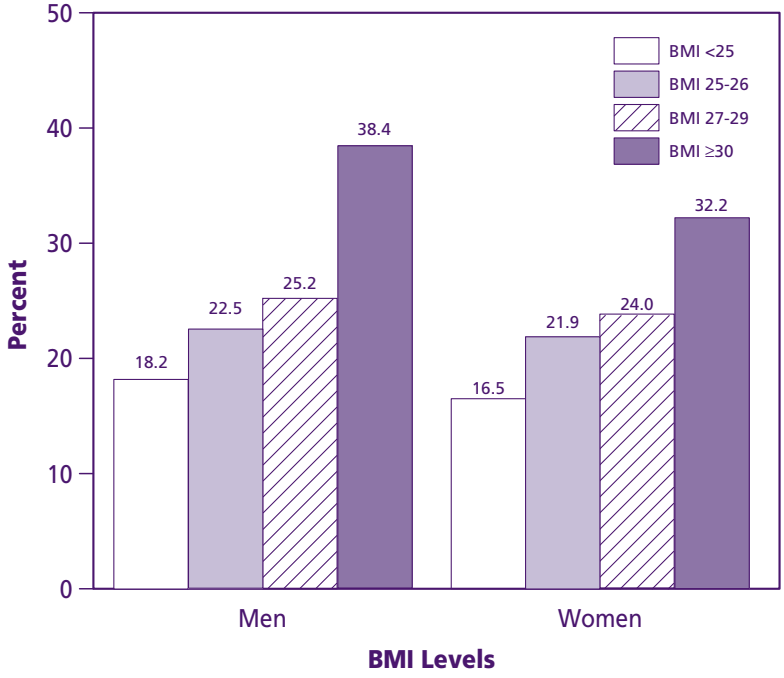
Obesity and hypertension are co-morbid risk factors for the development of cardiovascular disease. The pathophysiology underlying the development of hypertension associated with obesity includes sodium retention and associated increases in vascular resistance, blood volume, and cardiac output. These cardiovascular abnormalties associated with obesity are believed to be related to a combination of increased sodium retention, increased sympathetic nervous system activity, alterations of the renin-angiotensin system and insulin resistance. The precise mechanism whereby weight loss results in a decrease in blood pressure is unknown. However, it is known that weight loss is associated with a reduction in vascular resistance, total blood volume and cardiac output, an improvement in insulin resistance, a reduction in sympathetic nervous system activity, and suppression of the activity of the renin angiotensin aldosterone system. [764-769]

1.b. Dyslipidemia, manifested by:

High total cholesterol

The relationship of the age-adjusted prevalence of high total cholesterol, defined as ≥ 240 mg/dL (6.21 mmol/L), to BMI from NHANES III is shown in Figure 3. [2] At each BMI level, the prevalence of high blood cholesterol is greater in women than in men. In a smaller sample, higher body weight is associated with higher levels of total serum cholesterol in both men [133] and women [134] at levels of BMI > 25. Several large longitudinal studies also provide evidence that overweight, obesity and weight gain are associat-

Figure 2. NHANES III Age-Adjusted Prevalence of Hypertension* According to Body Mass Index



* Defined as mean systolic blood pressure ≥140 mm Hg, mean diastolic as ≥90 mm Hg, or currently taking antihypertensive medication.

Source: Brown C. et al. Body Mass Index and the Prevalence of Risk Factors for Cardiovascular Disease (submitted for publication).

visited on 6/14/2019

ed with increased cholesterol levels. [135-137] In women, the incidence of hypercholesterolemia also increases with increasing BMI. [138] In addition, the pattern of fat distribution appears to affect cholesterol levels independently of total weight. Total cholesterol levels are usually higher in persons with predominant abdominal obesity, defined as a waist-to-hip circumference ratio of ≥ 0.8 for women and ≥ 1.0 for men. [139]

High triglycerides

The strong association of triglyceride levels with BMI has been shown in both cross-sectional and longitudinal studies, for both sexes and all age groups. [133, 134, 140, 141] In three adult age groups, namely 20 to 44 years, 45 to 59 years, and 60 to 74 years, higher levels of BMI, ranging from 21 or less to more than 30, have been associated with increasing triglyceride levels; the difference in triglycerides ranged from 61 to 65 mg/dL (0.68 to 0.74 mmol/L) in women [134] and 62 to 118 mg/dL (0.70 to 1.33 mmol/L) in men. [133]

Low high-density lipoprotein cholesterol

The age-adjusted prevalence of low high-density lipoprotein (HDL)-cholesterol in relation to BMI levels, based on NHANES III data, is shown in Figure 4. [2] HDL-cholesterol levels at all ages and weights are lower in men than in women. Although low HDL-cholesterol in this study was defined as < 35 mg/dL (0.91 mmol/L) in men and < 45 mg/dL (1.16 mmol/L) in women [2], the panel accepts the definition of low HDL-cholesterol as < 35 mg/dL for men and women used by the National Cholesterol Education Program's *Second Report of the Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults* (Adult Treatment Panel II Report). [142] Cross-sectional studies have reported that HDL-cholesterol levels are lower in men and women with higher BMI. [143,144] Longitudinal studies have found that changes in BMI are associated with changes in HDL-cholesterol. A BMI change of 1 unit is associated with an HDL-cholesterol change of 1.1 mg/dL for young adult men and an HDL-cholesterol change of 0.69 mg/dL for young adult women. [145]

Normal to elevated low-density lipoprotein cholesterol

The link between total serum cholesterol and CHD is largely due to low-density lipoprotein (LDL). A high-risk LDL-cholesterol is defined as a serum concentration of ≥ 160 mg/dL. This lipoprotein is the predominant atherogenic lipoprotein and is therefore the primary target of cholesterol-lowering therapy. Cross-sectional data suggest that LDL-cholesterol levels are higher by 10 to 20 mg/dL in relation to a 10 unit difference in BMI, from levels of 20 to 30 kg/m^2. [133,134] According to extensive epidemiological data, a 10 mg/dL rise in LDL-cholesterol corresponds to approximately a 10 percent increase in CHD risk over a period of 5 to 10 years. [146]

Small, dense low-density lipoprotein particles

Few large-scale epidemiological data are available on small, dense LDL particles. [147-149] Clinical studies have shown that small, dense LDL particles are particularly atherogenic and tend to be present in greater proportion in hypertriglyceridemic patients with insulin resistance syndrome associated with abdominal obesity. [148-152]

1.c. Diabetes Mellitus

The increased risk of diabetes as weight increases has been shown by prospective studies in Norway [7], the United States [8], Sweden [9], and Israel. [10] More recently, the Nurses' Health Study, using data based on self-reported weights, found that the risk of developing type 2 diabetes increases as BMI increases from a BMI as low as 22. [81] Since women in particular tend to under-report weight, the actual BMI values associated with these risks are likely to be higher than the Nurses' Health Study data would suggest. An association between type 2 diabetes and increasing relative weight is also observed in popula-

visited on 6/14/2019

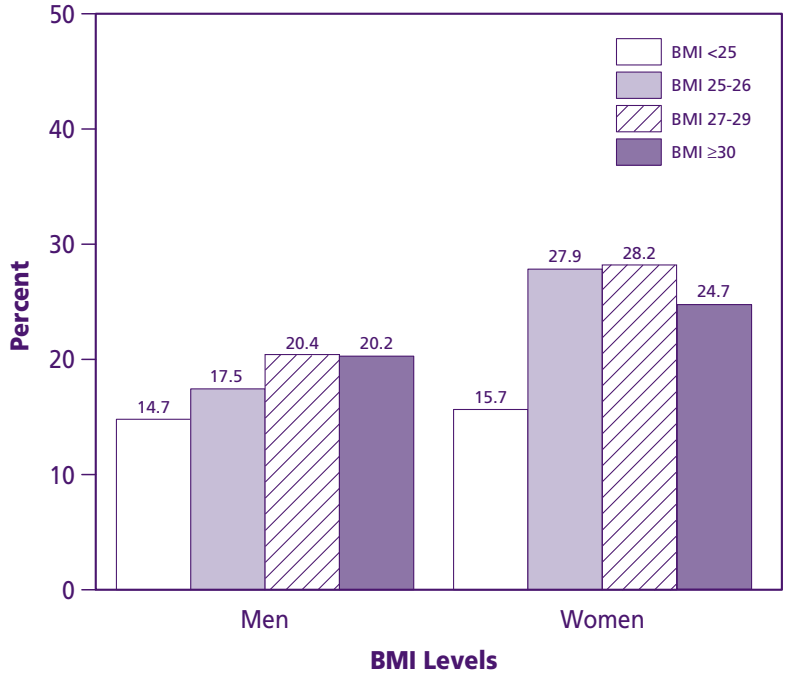
tions at high risk for obesity and diabetes, such as in American Indians. [153,154] In recent studies, the development of type 2 diabetes has been found to be associated with weight gain after age 18 in both men [82] and women. [81] The relative risk of diabetes increases by approximately 25 percent for each additional unit of BMI over 22 kg/m^2. [83] In addition, in a prospective study representative of the U.S. population, it was recently estimated that 27 percent of new cases of diabetes was attributable to weight gain in adulthood of 5 kg (11 lb) or more. [84] Both cross-sectional [85-87] and longitudinal studies [82,88,89] show that abdominal obesity is a major risk factor for type 2 diabetes. [82,87]

1.d. Coronary Heart Disease

Observational studies have shown that overweight, obesity, and excess abdominal fat are directly related to cardiovascular risk factors, including high levels of total cholesterol, LDL-cholesterol, triglycerides, blood pressure, fibrinogen and insulin, [86] and low levels of HDL-cholesterol. [42] Plasminogen activator inhibitor-1 causing impaired fibrinolytic activity is elevated in persons with abdominal obesity. [763] Overweight, obesity, and abdominal fat are also associated with increased morbidity and mortality from CHD. [11,42,155-161]

Recent studies have shown that the risks of nonfatal myocardial infarction and CHD death increase with increasing levels of BMI. Risks are lowest in men and women with BMIs of 22 or less and increase with even modest elevations of BMI. In the Nurses' Health Study, which controlled for age, smoking, parental history of CHD, menopausal status, and hormone use, rel-

Figure 3. NHANES III Age-Adjusted Prevalence of High Blood Cholesterol* According to Body Mass Index



*Defined as ≥240 mg/dL

Source: Brown C. et al. Body Mass Index and the Prevalence of Risk Factors for Cardiovascular Disease (submitted for publication).

visited on 6/14/2019

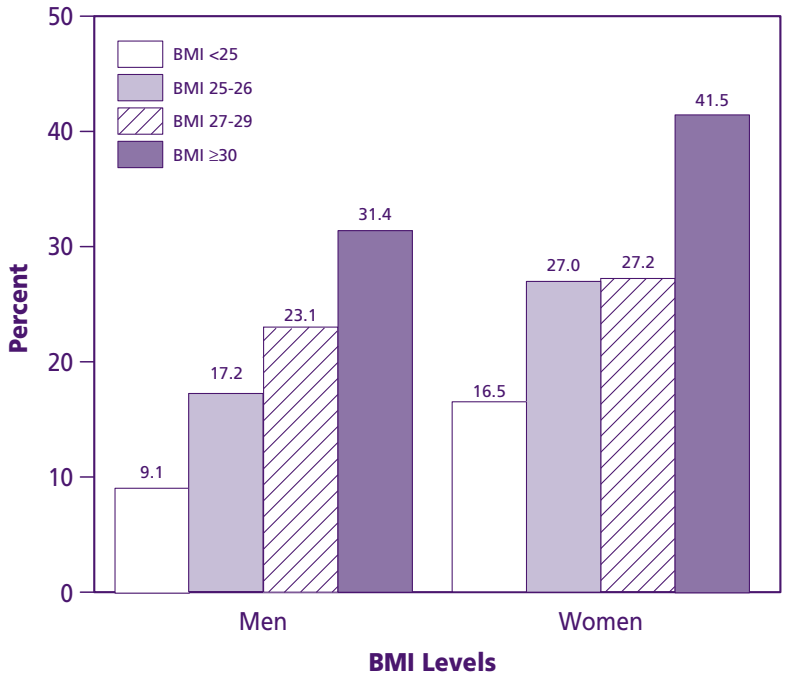
ative risks for CHD were twice as high at BMIs of 25 to 28.9, and more than three times as high at BMIs of 29 or greater, compared with BMIs of less than 21. [90] Weight gains of 5 to 8 kg (11 to 17.6 lb) increased CHD risk (nonfatal myocardial infarction and CHD death) by 25 percent, and weight gains of 20 kg (44 lb) or more increased risk more than 2.5 times in comparison with women whose weight was stable within a range of 5 kg (11 lb). [90] In British men, CHD incidence increased at BMIs above 22 and an increase of 1 BMI unit was associated with a 10 percent increase in the rate of coronary events. [162] Similar relationships between increasing BMI and CHD risk have been shown in Finnish, Swedish, Japanese, and U.S. populations. [90, 163, 164]

A relationship between obesity and CHD has not always been found. Two reasons may account for this: the first is an inappropriate controlling for cholesterol, blood pressure, diabetes, and other risk factors in statistical analysis; and the second is that there was not an adequate control for the confounding effect of cigarette smoking on weight. [88] People who smoke often have a lower body weight but more CHD.

1.e. Congestive Heart Failure

Overweight and obesity have been identified as important and independent risk factors for congestive heart failure (CHF) in a number of studies, including the Framingham Heart Study. [11, 165-169] CHF is a frequent complication of severe obesity and a major cause of death; duration of the obesity is a strong predictor of CHF. [170] Since

Figure 4. NHANES III Age-Adjusted Prevalence of Low HDL-Cholesterol* According to Body Mass Index



*Defined as <35 mg/dL in men and <45 mg/dL in women.

Source: Brown C. et al. Body Mass Index and the Prevalence of Risk Factors for Cardiovascular Disease (submitted for publication).

visited on 6/14/2019

hypertension and type 2 diabetes are positively associated with increasing weight, the coexistence of these conditions facilitates the development of CHF. [171] Data from the Bogalusa Heart Study demonstrate that excess weight may lead to acquisition of left ventricular mass beyond that expected from normal growth. [171] Obesity can result in alterations in cardiac structure and function even in the absence of systemic hypertension or underlying heart disease. Ventricular dilatation and eccentric hypertrophy may result from elevated total blood volume and high cardiac output. Diastolic dysfunction from eccentric hypertrophy and systolic dysfunction from excessive wall stress result in so-called "obesity cardiomyopathy". [172, 173] The sleep/apnea obesity hyperventilation syndrome occurs in 5 percent of severely obese individuals, and is potentially life-threatening. Extreme hypoxemia induced by obstructive sleep apnea syndrome may result in heart failure in the absence of cardiac dysfunction. [174]

1.f. Stroke

The relationship of cerebrovascular disease to obesity and overweight has not been as well studied as the relationship to CHD. A report from the Framingham Heart Study suggested that overweight might contribute to the risk of stroke, independent of the known association of hypertension and diabetes with stroke. [11] More recently published reports [12, 13] are based on larger samples and delineate the importance of stroke subtypes in assessing these relationships. They also attempt to capture all stroke events, whether fatal or nonfatal. These studies suggest distinct risk factors for ischemic stroke as compared to hemorrhagic stroke, and found overweight to be associated with the former, but not the latter. This may explain why studies that use only fatal stroke outcomes (and thus overrepresent hemorrhagic strokes) show only weak relationships between overweight and stroke. These recent prospective studies demonstrate that the risk of stroke shows a graded increase as BMI rises. For example, ischemic stroke risk is 75 percent higher in women with BMI > 27, and 137 percent higher in women with a BMI > 32, compared with women having a BMI < 21. [12]

1.g. Gallstones

The risk of gallstones increases with adult weight. Risk of either gallstones or cholecystectomy is as high as 20 per 1,000 women per year when BMI is above 40, compared with 3 per 1,000 among women with BMI < 24. [14] According to NHANES III data, the prevalence of gallstone disease among women increased from 9.4 percent in the first quartile of BMI to 25.5 percent in the fourth quartile of BMI. Among men, the prevalence of gallstone disease increased from 4.6 percent in the first quartile of BMI to 10.8 percent in the fourth quartile of BMI. [15]

1.h. Osteoarthritis

Individuals who are overweight or obese increase their risk for the development of osteoarthritis. [16-18,91,92] The association between increased weight and the risk for development of knee osteoarthritis is stronger in women than in men. [92] In a study of twin middle-aged women, it was estimated that for every kilogram increase of weight, the risk of developing osteoarthritis increases by 9 to 13 percent. The twins with knee osteoarthritis were generally 3 to 5 kg (6.6 to 11 lb) heavier than the co-twin with no disease. [16] An increase in weight is significantly associated with increased pain in weight-bearing joints. [175] There is no evidence that the development of osteoarthritis leads to the subsequent onset of obesity. [91] A decrease in BMI of 2 units or more during a 10-year period decreased the odds for developing knee osteoarthritis by more than 50 percent; weight gain was associated with a slight increase in risk. [93]

A randomized controlled trial of 6 months' duration examined the effect of weight loss on

visited on 6/14/2019

clinical improvement in patients with osteoarthritis. [176] Patients taking phentermine had an average weight loss of 12.6 percent after 6 months while the control group had an average weight loss of 9.2 percent. There was improvement in pain-free range of motion and a decrease in analgesic use in association with weight loss; patients with knee disease showed a stronger association than those with hip disease. Similarly, improvement of joint pain was observed in individuals who had undergone gastric stapling, resulting in an average weight loss of 45 kg (99 lb). [94, 95]

1.i. Sleep Apnea

Obesity, particularly upper body obesity, is a risk factor for sleep apnea and has been shown to be related to its severity. [19,20] The major pathophysiologic consequences of severe sleep apnea include arterial hypoxemia, recurrent arousals from sleep, increased sympathetic tone, pulmonary and systemic hypertension, and cardiac arrhythmias. [21] Most people with sleep apnea have a BMI > 30. [96,97] Large neck girth in both men and women who snore is highly predictive of sleep apnea. In general, men whose neck circumference is 17 inches or greater and women whose neck circumference is 16 inches or greater are at higher risk for sleep apnea. [98] Additional information on sleep apnea is included as Appendix IV.

1.j. Cancer

Colon Cancer

Many studies have found a positive relation between obesity and colon cancer in men but a weaker association in women. [8,22-24,99-106] More recent data from the Nurses' Health Study suggest that the relationship between obesity and colon cancer in women may be similar to that seen in men. Twice as many women with a BMI of > 29 kg/m^2 had distal colon cancer as women with a BMI < 21 kg/m^2. [107] In men, the relationship between obesity and total colon cancer was weaker than that for distal colon cancer. Other data from the Nurses' Health Study show a substantially stronger relationship between waist-to-hip ratio and the prevalence of colon polyps on sigmoidoscopy, than with BMI alone. [108] Even among leaner women, a high waist-to-hip ratio is also associated with significantly increased risk of colon polyps. [107]

Breast Cancer

Epidemiologic studies consistently show that obesity is directly related to mortality from breast cancer, predominantly in postmenopausal women, [8] but inversely related to the incidence of premenopausal breast cancer. [109-112] Ten or more years after menopause, the premenopausal "benefit" of obesity has dissipated. [113] Among postmenopausal women, peripheral fat is the primary source of estrogens, the major modifiable risk factor for postmenopausal breast cancer. This crossover in the relationship of obesity with breast cancer, pre- and postmenopausally, complicates prevention messages for this common female cancer. Recent data from the Nurses' Health Study, however, show that adult weight gain is positively related to risk of postmenopausal breast cancer. This relation is seen most clearly among women who do not use postmenopausal hormones. A gain of more than 20 lb from age 18 to midlife doubles a woman's risk of breast cancer. Even modest weight gains are positively related to risk of postmenopausal cancer. [114]

Endometrial Cancer

Obesity increases the risk of endometrial cancer. The risk is three times higher among obese women (BMI ≥ 30 kg/m^2) compared to normal-weight women. [115] However, the absolute risk of this condition is low when compared to breast cancer, heart disease, and diabetes. Adult weight gain is also related to increased risk. [115]

visited on 6/14/2019

Gallbladder Cancer

Obesity is related to the risk of gallbladder cancer, particularly among women. [177] Using a weight index of 100 as the average weight with a corresponding mortality ratio of 1.0 for the cohort, mortality ratios were 1.16 at a weight index of 120 to 129, 1.22 at 130 to 139, and 1.53 at ≥140.

1.k. Obesity and Women's Reproductive Health

Menstrual Function and Fertility

Obesity in premenopausal women is associated with menstrual irregularity and amenorrhea. [112,116] As part of the Nurses' Health Study, a case control study suggested that the greater the BMI at age 18 years, even at levels lower than those considered obese, the greater the risk of subsequent ovulatory infertility. [117] The most prominent condition associated with abdominal obesity is polycystic ovarian syndrome, [118] a combination of infertility, menstrual disturbances, hirsutism, abdominal hyperandrogenism, and anovulation. This syndrome is strongly associated with hyperinsulinemia and insulin resistance. [119]

Pregnancy

Pregnancy can result in excessive weight gain and retention. The 1988 National Maternal and Infant Survey observed that 41.6 percent of women reported retaining ≥ 9 lb of their gained weight during pregnancy, with 33.8 percent reporting ≥14 lb of retained weight gain. [120] The retained weight gain associated with pregnancy was corroborated by the study of Coronary Artery Risk Development in Young Adults (CARDIA). As a result of their first pregnancy, both black and white young women had a sustained weight gain of 2 to 3 kg (4.4 to 6.6 lb) of body weight. [121] Another study on a national cohort of women followed for 10 years reported that weight gain associated with childbearing ranged from 1.7 kg (3.7 lb) for those having one live birth during the study to 2.2 kg (4.9 lb) for those having three. [178] In addition, higher prepregnancy weights have been shown to increase the risk of late fetal deaths. [179]

Obesity during pregnancy is associated with increased morbidity for both the mother and the child. A tenfold increase in the prevalence of hypertension and a 10 percent incidence of gestational diabetes have been reported in obese pregnant women. [122] Obesity also is associated with difficulties in managing labor and delivery, leading to a higher rate of induction and primary Caesarean section. Risks associated with anesthesia are higher in obese women, as there is greater tendency toward hypoxemia and greater technical difficulty in administering local or general anesthesia. [123] Finally, obesity during pregnancy is associated with an increased risk of congenital malformations, particularly of neural tube defects. [123]

A certain amount of weight gain during pregnancy is desirable. The fetus itself, expanded blood volume, uterine enlargement, breast tissue growth, and other products of conception generate an estimated 13 to 17 lb of extra weight. Weight gain beyond this, however, is predominantly maternal adipose tissue. It is this fat tissue that, in large measure, accounts for the postpartum retention of weight gained during pregnancy. In turn, this retention reflects a postpartum energy balance that does not lead to catabolism of the gained adipose tissue. In part, this may reflect reduced energy expenditure through decreased physical activity, even while caring for young children, but it may also reflect retention of the pattern of increased caloric intake acquired during pregnancy. [180]

One difficulty in developing recommendations of optimal weight gain during pregnancy relates to the health of the infants. A balance must be achieved between high-birth-weight infants who may pose problems during delivery and who may face a higher rate of Caesarean sections and low-birth-weight infants who face a higher

visited on 6/14/2019

WEIGHT GAIN DURING PREGNANCY

BMI	Kilograms	Pounds
<19.8	12.5 to 18	28 to 40
19.8 to 26	11.5 to 16	25 to 35
>26 to 29	7 to 11.5	15 to 25
>29	≤6	≤13

infant mortality rate. [181] However, data from the Pregnancy Nutrition Surveillance System from the CDC showed that very overweight women would benefit from a reduced weight gain during pregnancy to help reduce the risk for high-birth-weight infants. [181]

The 1990 Institute of Medicine report made recommendations concerning maternal weight gain. [182] It recommended that each woman have her BMI measured and recorded at the time of entry into prenatal care. For women with a BMI of less than 20, the target weight gain should be 0.5 kg (1.1 lb) of weight gain per week during the second and third trimester. For a woman whose BMI is greater than 26, the weight gain target is 0.3 kg (0.7 lb) per week during the last two trimesters.

Women who are overweight or obese at the onset of pregnancy are advised to gain less total weight during the pregnancy (see box above). [182]

1.I. Psychosocial Aspects of Overweight and Obesity

A number of reviews have been published on the psychosocial aspects of obesity. [124-128,183] The specific topics that will be reviewed here include social stigmatization, psychopathology, binge eating, and body image perceptions.

Social stigmatization

In American and other Westernized societies there are powerful messages that people, especially women, should be thin, and that to be fat is a sign of poor self-control. [125, 126, 128, 184, 185] Negative attitudes about the obese have been reported in children and adults, [186-191] in health care professionals, [192-194] and in the overweight themselves. [195, 196]

People's negative attitudes toward the obese often translate into discrimination in employment opportunities, [197-199] college acceptance, [200] less financial aid from their parents in paying for college, [195, 201] job earnings, [202] rental availabilities, [203] and opportunities for marriage. [204]

Much of the research on the social stigma of obesity has suffered from methodological limitations. For example, a number of the early studies relied on line drawings rather than more lifelike representations of obese people and on checklists that forced one to make YES or NO choices. More importantly, there has been a lack of research that has looked at the impact of obesity in the context of other variables, such as physical attractiveness, the situational context, and the degree of obesity. [184,185] In addition, social stigma toward the obese has primarily been assessed among white individuals. There is some evidence that members of other racial and ethnic groups are less harsh in their evaluation of obese persons. One study assessed 213 Puerto Rican immigrants to the United States, and found a wide range of acceptable weights among them. [205] Crandall found that Mexican students were sig-

visited on 6/14/2019

nificantly less concerned about their own weight and were more accepting of other obese people than were U.S. students. [206] In addition, the degree of acceptance of obesity among people of lower education and income has not been well studied. Thus, these data are very incomplete with respect to racial and ethnic groups other than whites.

Psychopathology and Obesity

Research relating obesity to psychological disorders and emotional distress is based on community studies and clinical studies of patients seeking treatment. In general, community-based studies in the United States have not found significant differences in psychological status between the obese and nonobese. [126,183,207,208] However, several recent European studies in general populations do suggest a relationship between obesity and emotional problems. [209-211] Thus, it may be premature to state that there is no association between obesity and psychopathology or emotional distress in the general population. More focused, hypothesis-driven, and long-term studies are needed.[127, 212]

Overweight people seeking weight loss treatment may, in clinic settings, show emotional disturbances. [213-215] In a review of dieting and depression, there was a high incidence of emotional illness symptoms in outpatients treated for obesity. [213] However, several factors influenced these emotional responses, including childhood onset versus adult onset of obesity (those with childhood onset obesity appear more vulnerable). Another study that compared different eating disorder groups found that obese patients seeking treatment showed considerable psychopathology, most prominently mild to severe depression. [214] Sixty-two percent of the obese group seeking treatment showed clinically significant elevations on the depression subscale of the Minnesota Multiphasic Personality Inventory, and 37 percent of this same group showed a score of 20 or higher (indicating clinical depression) on the Beck Depression Inventory. Focusing on depression was considered an important component of the weight loss program. Another study compared obese people who had not sought treatment to an obese group that had sought treatment in a professional, hospital-based program, and to normal weight controls. [215] Again, obese individuals seeking treatment reported more psychopathology and binge eating compared to the other groups. Both obese groups reported more symptoms of distress than did normal weight controls. The authors suggest that the obese population is not a homogenous group, and thus, may not respond in the same way to standardized treatment programs. In particular, obese individuals seeking treatment in clinic settings are more likely than obese individuals not seeking treatment and normal controls to report more psychopathology and binge eating.

Binge Eating Disorder

Binge eating disorder (BED) is characterized by eating larger amounts of food than most people would eat in a discrete time period (e.g., 2 hours) with a sense of lack of control during these episodes.[762] It is estimated to occur in 20 to 50 percent of individuals who seek specialized obesity treatment. [216-218] In community-based samples, the prevalence is estimated to be approximately 2 percent. [219] Comparisons have been made between BED and bulimia nervosa (BN), an eating disorder characterized by recurrent and persistent binge eating, accompanied by the regular use of behaviors such as vomiting, fasting, or using laxatives. Studies comparing normal weight individuals who have BN with obese BED individuals have found that obese binge eaters are less likely to demonstrate dietary restraint and show few if any adverse reactions to moderate or severe dieting. Most obese binge eaters do not engage in inappropriate compensatory behaviors such as purging. [220] Compared with BN, the demographic distribution of BED

visited on 6/14/2019

is broader with respect to age, gender, and race [218, 219, 221-225] while data suggest that BED is as common in African-American women as in white women. [226] The difference between BED and BN is dramatic regarding gender. Very few men have BN, [227] whereas the distribution is close to equal in BED. [225, 228, 229]

Compared to obese nonbingers, obese individuals with BED tend to be heavier, [230] report greater psychological distress, and are more likely to have experienced a psychiatric illness (especially affective disorders). [225,231-236] They also report an earlier onset of obesity and a greater percentage of their lifetime on a diet. [237, 238] Some studies have shown histories of greater weight fluctuation or weight cycling in obese binge eaters compared with nonbingers, [219, 237, 238] but others have not. [239] These individuals are also more likely than nonbinging obese people to drop out of behavioral weight loss programs, [233] and to regain weight more quickly. [220,233,240]

Critics of behavioral treatment of obesity have argued that caloric restriction may cause or contribute to the episodes of binge eating and BN. [241] Three studies have tested this hypothesis. [218, 242,243] Neither moderate nor severe caloric restriction exacerbated binge eating. All three studies showed that weight control treatment featuring caloric restriction significantly reduced the frequency of binge eating in these patients.

Body Image

Body image is defined as the perception of one's own body size and appearance and the emotional response to this perception. [183,244] Inaccurate perception of body size or proportion and negative emotional reactions to size perceptions contribute to poor body image. Obese individuals, especially women, tend to overestimate their body size. [245-249]

People at greater risk for a poor body image are binge eaters, women, those who were obese during adolescence or with early onset of obesity, and those with emotional disturbances. [127, 235, 244, 250-253] It is no surprise, then, that in some groups of obese persons, these individuals are more dissatisfied and preoccupied with their physical appearance, and avoid more social situations due to their appearance. [254, 255] Body image dissatisfaction and the desire to improve physical appearance often drives individuals to seek weight loss. However, obese persons seeking weight reduction must come to terms with real limits in their biological and behavioral capacities to lose weight. Otherwise, weight loss attempts may only intensify the sense of failure and struggle that is already present among many obese individuals. For this reason, psychosocial interventions which incorporate strategies to improve body image may be helpful for those who want to lose weight and are very concerned about their physical appearance. A review of body image interventions in obese persons can be found in Rosen (1996). [256]

Body image perceptions of individuals in various ethnic and racial groups may be different, on average, from those of the mainstream culture. There may be a similar range of attitudes but on a different scale; for example, it may take a much greater degree of overweight to elicit negative reactions. [257] Differences in body image and weight-related concerns between black and white girls and women have been observed. [257] In general, black girls and women report: less social pressure to be slim, [258-260] fewer incidences of weight-related discrimination, [261] less weight and body dissatisfaction, and greater acceptance of overweight than their white counterparts. [259,262-266] College-age black women report less concern and fear about fatness, less drive to be thin, and less concern about dieting than do college-age white women. [267] In addition, black women may ascribe some positive qualities to being large, such as having stamina, strength, and solidity, and are less likely to link body size to health

visited on 6/14/2019

than white women. Black elementary school and high school girls were more likely to be trying to gain weight [268, 269] and less likely to be trying to lose weight as compared to white girls. [269] Because of the above, it is possible that weight control initiatives may elicit different reactions from black and white women. Less is known about the relationship between obesity and body image disturbance in other racial and ethnic groups. [270]

2. Overweight/Obesity and Morbidity in Minority Populations

The data on overweight and obesity in minority populations include men and women across a wide age range and geographic area. Relevant studies increasingly consist of well-designed, population-based surveys and longitudinal studies. These studies have standardized, objective measurements of overweight and obesity and risk factors or disease outcomes. There is now a wealth of evidence to demonstrate that overweight and obesity incidence (both generalized and abdominal) predisposes to chronic diseases in racial/ethnic minority populations as it does in whites, though the absolute risk may differ. [51-52, 271-284] Indications for treatment of overweight and obesity in minority populations are, therefore, the same as those for non-Hispanic whites. Apparent differences in the strength of association between obesity and disease in various populations are not necessarily relevant to individuals in clinical settings, and obesity should be treated in any situation in which excess weight is associated with an observable or probable risk of morbidity. In addition, from a public health perspective, the need for obesity prevention and treatment is particularly pressing in racial/ethnic minority populations because of the high proportion of overweight and obese persons in many such populations.

3. Obesity and Mortality

As stated in the introduction to the guidelines, in the majority of epidemiologic studies, mortality begins to increase with BMIs above 25 kg/m^2. [28-32] The increase in mortality generally tends to be modest until a BMI of 30 kg/m^2 is reached. [28, 29, 31, 32] For persons with a BMI of 30 kg/m^2 or above, mortality rates from all causes, and especially from cardiovascular disease, are generally increased by 50 to 100 percent above that of persons with BMIs in the range of 20 to 25 kg/m^2. [28,31,32] Three aspects of the association between obesity and mortality remain unresolved:

3.a. Association of Body Mass Index With Mortality

Many of the observational epidemiologic studies of BMI and mortality have reported a 'U-' or 'J-shaped' relationship between BMI and mortality. [28] Mortality rates are elevated in persons with low BMI (usually below 20) as well as in persons with high BMI. [28, 31, 32] In some studies, adjustment for factors that potentially confound the relationship between BMI and mortality, such as smoking status and pre-existing illness, tends to reduce the upturn in mortality rate at low BMI,[31] but in a meta-analysis the higher mortality at low BMIs was not eliminated after adjustment for confounding factors. [32] It is unclear whether the elevated mortality observed at low BMI is due to an artifact of incomplete control for confounding factors, [285] inadequate body fat and/or inadequate body protein stores that result from unintentional weight loss, [286] or individual genetic factors. Currently, there is no evidence that intentional weight gain in persons with low BMIs will lead to a reduction in mortality.

3.b. Association of Body Mass Index With Mortality in Older Adults

Many of the observational epidemiologic studies suggest that the relationship between BMI and mortality weakens with increasing age, especially among persons aged 75 and above. [287- 290] Several

visited on 6/14/2019

factors have been proposed to explain this observation. Older adults are more likely than younger adults to have diseases that both increase mortality and cause weight loss leading to lower body weight. [291-293] In addition, as people age, they tend to have larger waist circumferences that increase their risk of mortality even at lower BMIs. [294] Also, weight in middle age is positively related to risk of mortality in old age.[292] The impact of smoking on body weight and mortality is likely to be much stronger in older adults because of the cumulative health effects of smoking. [295]

BMI, which is an indirect estimate of adiposity, may underestimate adiposity in older adults whose BMI is similar to younger adults. [296] It is also possible that persons most sensitive to the adverse health effects of obesity are more likely to have died before reaching older ages, resulting in older cohorts that are more "resistant" to the health effects of obesity. Recently, a 20-year prospective study of a nationally representative sample of U.S. adults aged 55 to 74 years suggested that lowest mortality occurs in the BMI range of 25 to 30. [297 298] After adjusting for smoking status and pre-existing illness, lowest mortality occurred at a BMI of 24.5 in white men, 26.5 in white women, 27.0 in black men, and 29.8 in black women (see commentary on older adults on pages 90-91).

3.c. Association of Body Mass Index With Mortality in Ethnic Minorities

The levels of BMI associated with increased mortality are based on epidemiological studies of primarily white populations. The interest in confirming the association between BMI and mortality in other racial/ethnic groups stems partly from observations that lower-than-average total mortality has been observed among some populations with a high BMI level, [299] and partly from observations that within certain populations there appears to be no effect of obesity at all or at the BMI levels that are associated with higher mortality in whites.

African-Americans:
Three small cohort studies of narrowly defined populations of African-Americans failed to show the expected association of BMI and mortality based on data from white populations. [300-303] Although the shape of the association of BMI and mortality in two large, representative U.S. data sets (the National Health and Nutrition Examination Follow-up Study and the National Health Interview Survey) is similar for black and white males and females, [304] the BMI-related increase in risk begins at a 1 to 3 kg/m^2 higher BMI level for blacks than for whites. For example, in the National Health and Nutrition Examination Follow-up Survey, the estimated BMI associated with minimum mortality was 27.1 for black men and 26.8 for black women, compared with 24.8 and 24.3, respectively, for white men and women. On the basis of these data, the use of the cutpoint of BMI $\geq$ 30 kg/m^2 for defining obesity is clearly applicable to African-Americans as well as to whites.

Other Ethnic Minority Populations:
Limited data relating obesity to mortality in American Indians were identified, but no data were found relating obesity to mortality in Hispanic-Americans, Asian-Americans, or Pacific Islanders. [305] The lowest mortality rate among Pima men is observed at a BMI range of 35 to 40 kg/m^2 for men, and no relationship between BMI and mortality is observed among Pima women. [306, 307] Based on mortality data alone, it would be hard to justify using the same standard for defining obesity in populations, such as American Indians, among whom the mean BMI is much higher than in the general U.S. population. However, diabetes-related morbidity among obese American Indians is extremely high, [154] and the overall age-specific mortality among American Indians is generally higher

visited on 6/14/2019

than in the U.S. general population. [306,307] Thus, obesity in American Indians is associated with a compromised overall survival of the population.

Although the data on mortality are still fragmentary for many minority populations, there are no studies that would support the exclusion of any racial/ethnic group from the current definitions of obesity. Secular trends in many populations in the United States and throughout the world have demonstrated that longstanding overweight and obesity eventually leads to the emergence of chronic diseases. Therefore, prevalent overweight and obesity cannot be ignored even where the associated health problems have not reached the level that would be expected on the basis of data for white populations.

D Weight Loss and Mortality

A number of studies of "generic weight loss" (cause of weight loss unknown), "weight cycling" (cycles of weight loss followed by weight regain), and mortality have been published. [308-311] In most, [308,309,311] but not all, [310] of these studies, generic weight loss and weight cycling are associated with increases in mortality. None of these studies, however, differentiated between intentional and unintentional weight loss. [311] With the exception of the studies below, very little is currently known about factors related to intentional and unintentional weight loss in the general population or about the relationship between weight loss intention and mortality. [312]

Two studies of factors related to weight loss intention have been carried out in the general population: French and colleagues assessed correlates of intentional and unintentional weight loss of > 20 lb in the Iowa Women's Health Study, a cohort study of approximately 29,000 women with a mean age of about 65 years; [313-315] and Meltzer and Everhart analyzed data on 1-year self-reported weight change from approximately 9,000 participants in the nationally representative U.S. National Health Interview Survey, aged 45 years and above. [316] The results of these two studies suggest the following:

- In cross-sectional studies of weight loss recall, heavier persons are more likely to report intentional weight loss than unintentional weight loss, while the reverse is true for leaner persons;
- Intentional and unintentional weight losses occur with similar frequency in the U.S. population and contribute similarly to long-term weight fluctuation;
- The frequency of intentional weight loss is lower at older ages, while the frequency of unintentional weight loss is higher at older ages; and
- Unintentional weight loss occurs more often in persons who report that their health status is poor, who use medications for chronic health conditions, and who smoke. [312]

To date, only three studies have examined the relationship between intentional weight loss and mortality. Singh and colleagues [317] published results from a 1-year randomized controlled trial of a "cardioprotective diet" in East Indian patients hospitalized with recent myocardial infarction (mean age 50 years, mean BMI about 24 kg/m^2). Although this study was not designed to specifically test the efficacy of intentional weight loss on lower mortality, the authors found that those who lost at least 0.5 kg (1.1 lb) had a 50 percent lower incidence of cardiac events and a 54 percent lower risk of overall mortality compared with counterparts who lost < 0.5 kg (1.1 lb). [317]

Williamson and colleagues [318] published a 12-year prospective observational study of weight loss and mortality that directly assessed weight loss intention. They analyzed data from 43,457 overweight (BMI > 27), never-smoking, white women ages 40 to 64 years. Mortality ratios were compared for women who intentionally

visited on 6/14/2019

lost weight with those for women who had no change in weight. In women with obesity-related comorbidities, intentional weight loss of any amount was associated with a statistically significant 20 percent reduction in all-cause mortality, primarily due to a significant 40 to 50 percent reduction in mortality from obesity-related cancers; diabetes-related mortality was also significantly reduced by 30 to 40 percent in those who intentionally lost weight. In women with no comorbidities, intentional weight loss was generally unrelated to mortality; however, after subdividing intentional weight loss by time interval, it was found that a loss of at least 20 lb that occurred within the previous year was associated with small to modest increases in mortality. The authors concluded that the association between intentional weight loss and longevity in middle-age overweight women depends on health status. In addition, preliminary evidence suggests that intentional weight loss in middle-age overweight men may be associated with a similar reduction of diabetes-related mortality as was observed in the overweight women. [319]

The ongoing Swedish Obesity Study is a controlled trial of surgically induced weight loss and subsequent morbidity and mortality over a 10-year follow-up period (1,006 participants aged 37 to 57 years; initial BMI of 34 in men and 38 in women [320, 321]). Although the study is not randomized (participants self-select for surgery), the controls (who receive a behavioral weight loss program) are computer-matched to surgical participants on a large number of potential confounders including weight. [320] In a preliminary abstract, [322] the study reported the 2-year incidence rates shown in Table II-3.

These results for disease and risk factor incidences suggest that 10-year mortality will ultimately be lower in the surgical intervention group. Definitive mortality results have not been reported to date.

E Environment

The environment is a major determinant of overweight and obesity. Environmental influences on overweight and obesity are primarily related to food intake and physical activity behaviors [71]. In countries like the United States, there is an overall abundance of palatable, calorie-dense food. In addition, aggressive and sophisticated food marketing in the mass media, supermarkets, and restaurants, and the large portions of food served outside the home, promote high calorie consumption. Many of our sociocultural traditions promote overeating and the preferential consumption of high calorie foods. For many people, even when caloric intake is

TABLE II-3:

Two-Year Incidence Rates

	Control	Surgery
Diabetes	16 percent	0.5 percent
Hypertriglyceridemia	23 percent	6.0 percent
Low High-Density Lipoprotein Levels	16 percent	5.0 percent

visited on 6/14/2019

not above the recommended level, the number of calories expended in physical activity is insufficient to offset consumption. Mechanization limits the necessity of physical activity required to function in society. Many people are entrenched in sedentary daily routines consisting of sitting at work, sitting in traffic, and sitting in front of a television or a computer monitor for most of their waking hours.

In this obesity-promoting environment, individual attitudes and behaviors are critical in weight management. Many individuals may need extended treatment in clinical or community settings to enable them to cope with the complexities of long-term weight management, especially if there is a history of unsuccessful attempts at self-treatment. [44] When the typical daily routine is so strongly biased towards promoting and perpetuating overweight and obesity, very high levels of knowledge, motivation, personal behavioral management skill, and lifestyle flexibility are required for an overweight or obesity-prone individual to avoid becoming overweight, or progressing to moderate or severe obesity.

Although there are undoubtedly some inter- and intrapopulation variations in the genetic predisposition to become overweight or obese, several lines of evidence suggest that genetic factors alone cannot explain the demographic and ethnic variations in overweight and obesity prevalence. For example, there is a difference in obesity prevalence among low- and high-income white women in industrialized societies. [323,324] Other studies of populations, including migration studies, have shown an increase in average body weight in those who move from a traditional to a Westernized environment. [325-328] Culturally determined attitudes about food, physical activity, and factors that vary with income, education, and occupation may increase the level of difficulty in weight management. Body image concerns and other motivations for avoiding obesity or controlling weight within given limits also vary with ethnic background, age, socioeconomic status, and gender. Thus, the competence of practitioners in working with diverse sociocultural perspectives can be a critical factor in the success of obesity treatment. [257] For a discussion of cultural issues in obesity treatment and related references, see Appendix III.

F Genetic Influence in the Development of Overweight and Obesity

Obesity is a complex multifactorial chronic disease developing from interactive influences of numerous factors—social, behavioral, physiological, metabolic, cellular, and molecular. Genetic influences are difficult to elucidate and identification of the genes is not easily achieved in familial or pedigree studies. Furthermore, whatever the influence the genotype has on the etiology of obesity, it is generally attenuated or exacerbated by nongenetic factors.

A large number of twin, adoption, and family studies have explored the level of heritability of obesity; that is, the fraction of the population variation in a trait (e.g., BMI) that can be explained by genetic transmission. Recent studies of individuals with a wide range of BMIs, together with information obtained on their parents, siblings, and spouses, suggest that about 25 to 40 percent of the individual differences in body mass or body fat may depend on genetic factors. [329-331] However, studies with identical twins reared apart suggest that the genetic contribution to BMI may be higher, i.e., about 70 percent. [332] There are several other studies of monozygotic twins reared apart that yielded remarkably consistent results. [333] Some of the reasons behind the different results obtained from twin versus family studies have been reported. [334-336] The relative risk of obesity for first-degree relatives of overweight, moderately obese, or severely obese persons in comparison to the population prevalence of the condition

visited on 6/14/2019

reaches about 2 for overweight, 3 to 4 for moderate obesity, and 5 and more for more severe obesity. [337,338]

Support for a role of specific genes in human obesity or body fat content has been obtained from studies of Mendelian disorders with obesity as one of the clinical features, single-gene rodent models, quantitative trait loci from crossbreeding experiments, association studies, and linkage studies. From the research currently available, several genes seem to have the capacity to cause obesity or to increase the likelihood of becoming obese. [339] The rodent obesity gene for leptin, a natural appetite-suppressant hormone, has been cloned [340] as has been its receptor. [341] In addition, other single gene mutants have been cloned. [341, 342] However, their relationship to human disease has not been established, except for one study describing two subjects with a leptin mutation. [342] This suggests that for most cases of human obesity, susceptibility genotypes may result from variations of several genes.

Severely or morbidly obese persons are, on the average, about 10 to 12 BMI units heavier than their parents and siblings. Several studies have reported that a single major gene for high body mass was transmitted from the parents to their children. The trend implies that a major recessive gene, accounting for about 20 to 25 percent of the variance, is influenced by age and has a frequency of about 0.2 to 0.3. [343] However, no gene(s) has (have) yet been identified. Evidence from several studies has shown that some persons are more susceptible to either weight gain or weight loss than others. [344,345] It is important for the practitioner to recognize that the phenomenon of weight gain cannot always be attributed to lack of adherence to prescribed treatment regimens.

visited on 6/14/2019

Examination of Randomized Controlled Trial Evidence

A Why treat overweight and obesity?

The recommendation to treat overweight and obesity is based not only on the previously presented evidence that shows overweight is associated with increased morbidity and mortality, but also on randomized controlled trial (RCT) evidence that weight loss reduces risk factors for disease. Thus, weight loss may help control diseases worsened by overweight and obesity and may also decrease the likelihood of developing these diseases. The evidence meeting the panel's inclusion criteria is presented in the following section in the form of evidence statements with a corresponding rationale for each statement. The details of the studies reviewed but not discussed here are provided in Appendix II.

Many of the RCTs examined by the panel included the use of pharmacotherapy for weight loss. When the panel began its deliberations in 1995, two weight loss drugs, fenfluramine and dexfenfluramine, were being used widely for long-term weight loss, i.e., 14 million prescriptions were given out over 1½ years. However, as of September 1997, the Food and Drug Administration (FDA) requested the voluntary withdrawal of these drugs from the market due to a reported association between valvular heart disease and the use of dexfenfluramine and fenfluramine alone or combined with phentermine. [761] In November 1997, the FDA approved the drug sibutramine hydrochloride monohydrate for the management of obesity, including weight loss and maintenance of weight loss when used in conjunction with a reduced-calorie diet. Thus, at the present time only one weight loss drug is available for long-term weight loss. (Note: FDA approval for orlistat is pending a resolution of labeling issues and results of Phase III trials.) However, due to rapidly evolving information regarding the use of pharmacotherapy for weight loss, the panel decided to present (below) their critique of those pharmacotherapy trials meeting their criteria for consideration.

1. Blood Pressure

To evaluate the effect of weight loss on blood pressure and hypertension, 76 articles reporting the results of RCTs were potentially eligible for inclusion. Sixty articles included lifestyle trials that studied diet and/or physical activity, and 16 articles were of pharmacotherapy trials. Dietary interventions included low-calorie diets and diets that promoted macronutrient composition changes, such as amount and type of dietary fat. Physical activity, when included, was used to help promote increased energy expenditure. These trials did not always control for other dietary factors that lower blood pressure, such as dietary salt, and the degree to which those taking the blood pressure measurements were blinded to the patient's change in weight.

Of the 35 lifestyle RCT articles deemed acceptable, 16 included hypertensive patients, [346-361] and 19 were conducted in individuals with normal or high-normal blood pressure. [362-380]

visited on 6/14/2019

1.a.(1). Lifestyle Trials in Hypertensive Patients

Evidence Statement: *Weight loss produced by lifestyle modifications reduces blood pressure in overweight hypertensive patients. Evidence Category A.*

Rationale: A 1987 meta-analysis [381] covering five of the acceptable studies [351, 352, 356-358] in hypertensive patients concluded that weight loss accomplished by dietary interventions significantly lowered blood pressure. In hypertensive patients, 10 kg (22 lb) of weight loss was associated with an average reduction of 7 mm Hg systolic and 3 mm Hg diastolic blood pressure compared with controls. [381]

Since publication of this meta-analysis in 1987, almost all relevant studies have reported that weight loss reduces blood pressure or the need for medication in hypertensive patients: [346- 348, 350, 352-361]

- The Trial of Antihypertensive Interventions and Management (TAIM), conducted in hypertensive individuals not taking medication for 6 months, reported that, compared with controls, a mean net weight reduction of 4.7 kg (10.4 lb) reduced systolic and diastolic blood pressure by 2.8 mm Hg and 2.5 mm Hg, respectively; [355] the effects on blood pressure were equivalent to drug therapy among those participants who lost 4.5 kg (9.9 lb) or more. [360]
- One study in older (age 60 to 80 years) hypertensive adults whose blood pressure medication was withdrawn (Trial of Nonpharmacologic Interventions in the Elderly, TONE) showed that after 2 years, mean net weight loss of nearly 4 kg (9 lb) resulted in more participants free of trial endpoints (occurrence of high blood pressure, resumption of blood pressure medication, or occurrence of a cardiovascular-clinical complication [39.2 percent versus 26.2 percent]). [382] Furthermore, blood pressure control was similar for men and women and for African-Americans and whites. [353]
- In other studies of primarily middle-aged hypertensive adults, compared with controls, weight loss significantly reduced a return to hypertension medication at 1 year [354] and at 4 to 5 years. [348, 359]
- The Multiple Risk Factor Intervention Trial, which recruited 12,866 high-risk men, 30 percent of whom had hypertension, delivered an integrated intervention addressing several lifestyle behaviors and included weight loss as an important component. Significant reductions in systolic and diastolic blood pressure were found over 6 years compared with the usual care group and were directly related to weight loss: 1 kg (2.2 lb) of weight loss was associated with a reduction of 0.4 mm Hg systolic and 0.3 mm Hg diastolic blood pressure in men not taking antihypertensive medications. The effect was slightly lower for men taking antihypertensive medications.
- Only one RCT conducted in hypertensive patients reported no significant change in blood pressure despite weight loss of 3.3 kg (7.3 lb). [351]
- Another study conducted in hypertensive individuals suggested that weight loss reduced blood pressure only when sodium intake was also reduced. [349] This finding was not consistent with a prior study of hypertensive patients [358] and has not been corroborated in subsequent larger trials conducted in nonhypertensive individuals. [378, 379]

visited on 6/14/2019

1.a.(2). Lifestyle Trials in Nonhypertensive Individuals

Evidence Statement: *Weight loss produced by lifestyle modifications reduces blood pressure in overweight nonhypertensive individuals. Evidence Category A.*

Rationale: A semiquantitative review [383] covering four of the acceptable studies [363, 366, 372, 374] in nonhypertensive individuals concluded that weight loss through dietary interventions significantly lowered blood pressure. In nonhypertensives, excluding one outlier trial that showed very large reductions in blood pressure, [372] 1 kg (2.2 lb) of weight loss was associated with a reduction of 0.45 mm Hg in both systolic and diastolic blood pressure. Since this review, published in 1991, almost all relevant studies have reported that weight loss reduces blood pressure in nonhypertensive individuals. [364, 365, 367-371, 373, 375-380, 384]

The Trials of Hypertension Prevention Phase 1 (TOHP I) and Phase 2 (TOHP II) are among the larger, well-designed randomized trials, having follow-up rates exceeding 90 percent. They followed the participants for 18 months [378] and 3 to 4 years. [379] The sample size of the weight loss intervention arms ranged from approximately 300 (TOHP I) to 600 (TOHP II). The populations were diverse, consisting of 30 to 35 percent women and 15 to 18 percent African-Americans, and were recruited from 10 centers in TOHP I and 9 centers in TOHP II. Results from both TOHP I and TOHP II demonstrated that weight loss reduced blood pressure and the incidence of hypertension. Compared with controls, in both trials 10 kg (22 lb) of weight loss was associated with a reduction of 7 mm Hg systolic and 5 to 6 mm Hg diastolic blood pressure at 18 months. At 36 months, systolic and diastolic blood pressure was reduced by 6 and 4 mm Hg, respectively, for every 10 kg of weight loss.[379] In addition, weight loss reduced the incidence of hypertension at 18 months by 20 to 50 percent [378, 379] and at 3 years by 19 percent. [379] Secondary analyses from TOHP I [376] and analyses under way from TOHP II [385] demonstrated that the greater the weight loss the greater the blood pressure reduction, and as long as weight loss was maintained, blood pressure remained reduced. One study that focused on older African-American diabetics showed that weight loss of 2.4 kg (5.3 lb) at 6 months resulted in a reduction of 3.9 and 4.0 mm Hg systolic and diastolic blood pressure. [362] Only one study in nonhypertensive individuals [368] showed inconsistent results, where weight reduction decreased systolic (significant) and diastolic (nonsignificant) blood pressure at 12 months in women but not in men. Another study [377] showed short-term (3 months) blood pressure reduction but no longer-term (9 months) blood pressure reduction despite maintenance of weight loss of 9 kg (19.8 lb). [384]

1.b. Pharmacotherapy Trials

Evidence Statement: *Weight loss produced by most weight loss medications (except for sibutramine) and adjuvant lifestyle modifications is accompanied by reductions in blood pressure. Evidence Category B.*

To determine the effects of pharmacological therapy on weight loss and subsequent changes in blood pressure levels, 10 RCT articles were examined. [386-395] Weight loss studies using pharmacotherapy were conducted predominantly in white women and assumed that dietary changes were uniform in the active drug and placebo groups. All of these studies had a placebo-control group for which dietary recommendations for weight loss were provided; weight loss also occurred in the placebo group. No studies exam-

visited on 6/14/2019

ined whether weight loss using pharmacotherapy results in blood pressure reductions similar to weight loss produced by diet therapy, nor were the results compared with a pure control group with no weight loss. In addition, none of the studies that examined the effect of pharmacotherapy on blood pressure controlled for weight loss. In general, the use of phentermine, fenfluramine, and dexfenfluramine resulted in similar or better weight loss than that seen in the control or placebo group, and reductions in systolic and diastolic blood pressure were concomitantly similar to or better than those observed in the diet plus placebo group. [386, 390-393, 395] However, one study on dexfenfluramine [394] showed increased blood pressure compared to controls, even though weight loss was greater with dexfenfluramine than with placebo. Another study showed blood pressure lowering consistent with weight reduction produced by dexfenfluramine in women with upper-body obesity, but not in women with lower-body obesity. [387] In a combined summary of data on orlistat presented at the FDA Endocrinologic and Metabolic Drugs Advisory Committee Meeting (held in Bethesda, Maryland, in May 1997), only a small decrease in systolic (approximately 2 mm Hg) and diastolic (approximately 1 mm Hg) blood pressure was observed compared to the placebo. [388] In a study of sibutramine, a net weight loss at 3 months of approximately 2 kg (4.4 lb) did not result in lower blood pressure compared with placebo controls. [389] Eleven double-blind placebo-controlled trials lasting from 12 to 52 weeks showed that sibutramine is associated with mean increases in systolic blood pressure of 1 to 3 mm Hg, in diastolic pressure of 1 to 2 mm Hg, and mean increases in pulse rate of 3 to 5 beats per minute relative to placebo. In hypertensive obese patients, the mean change in blood pressure was the same in the sibutramine group and the placebo group; both dropped. Systolic pressure decreased 5.4 mm Hg in the sibutramine group and 5.8 mm Hg in the placebo group, while the diastolic drop was 5.9 mm Hg and 3.7 mm Hg, respectively. At the FDA recommended doses, 45 percent of patients had an increase, 35 percent had a decrease, and 20 percent had no change in blood pressure. [389]

1.c. Abdominal Fat

Evidence Statement: *Limited evidence suggests that decreases in abdominal fat reduce blood pressure in overweight nonhypertensive individuals, although this has not been shown to be independent of weight loss. Evidence Category C.*

Rationale: Five RCTs [365,369,373, 375,377] testing the effects of weight loss on blood pressure also measured abdominal fat by waist circumference or by dual-energy X-ray absorptiometry. In all five studies, waist circumference was reduced along with weight, and blood pressure was decreased. These studies were not designed to test whether reduction in abdominal fat reduces blood pressure independent of weight loss.

1.d. Fitness

Evidence Statement: *Increased aerobic activity to increase cardiorespiratory fitness reduces blood pressure independent of weight loss. Evidence Category A.*

Rationale: Seven RCTs testing the effects of weight loss on blood pressure in overweight and obese individuals also had measures of cardiorespiratory fitness, as measured by maximal oxygen uptake [346, 363, 369, 375, 377, 380]; or submaximal heart rate tests. [365] Weight loss was accompanied by increased fitness, primarily if the intervention included increased physical activity or physical activity combined with diet. [346, 363, 375, 377, 380] These

visited on 6/14/2019

studies were not designed to test whether increased cardiorespiratory fitness reduces blood pressure independent of weight loss in overweight and obese adults. By contrast, in 3 meta-analyses of 68 controlled studies of physical activity conducted in normotensive and hypertensive individuals, obesity was not an outcome measure nor was it included in the eligibility criteria. These meta-analyses showed that aerobic exercise to increase cardiorespiratory fitness significantly reduces blood pressure independent of or in the absence of weight loss. [396-398]

RECOMMENDATION: *Weight loss is recommended to lower elevated blood pressure in overweight and obese persons with high blood pressure. Evidence Category A.*

2. Serum/Plasma Lipids and Lipoproteins

Sixty-five RCT articles were evaluated for the effect of weight loss on serum/plasma concentrations of total cholesterol, low-density lipoprotein (LDL)-cholesterol, very low-density lipoprotein cholesterol, triglycerides, and high-density lipoprotein (HDL)-cholesterol. Studies were conducted on individuals over a range of overweight and lipid levels. Weight loss in 52 of these trials was induced by various lifestyle modifications, including diet modification (reducing calories, or saturated fat and cholesterol, or both) and increased physical activity.

2.a. Lifestyle Trials

Evidence Statement: *Weight loss produced by lifestyle modifications reduces serum triglycerides and increases HDL-cholesterol, and generally produces some reductions in serum total cholesterol, and LDL-cholesterol. Evidence Category A.*

Rationale: Fourteen RCT articles that studied the effects of diet and/or physical activity on lipids were reviewed. [365, 368, 370, 373, 380, 384, 399-406] Table III-1 summarizes RCTs in which diet alone was used to produce weight loss. Entries under percentage change were calculated as follows: the percentage change (value at the end of study minus the value at baseline, divided by the value at baseline) was determined for the intervention group and the control group; the percentage change for the control group was then subtracted from the percentage change for the intervention group, giving the numerical value presented. When a run-in period was used, the "baseline" subtracted was the value at the end of the run-in, which was when the intervention began.

Reductions in body weight varied from 5 to 13 percent compared to the control group. The following changes in plasma lipids and lipoproteins were observed: total cholesterol, 0 to -18 percent; triglycerides, -2 to -44 percent; LDL-cholesterol, -3 to -22 percent; and HDL-cholesterol, -7 to +27 percent. Most of the trials were of sufficient duration to ensure that these changes were not the result of acute caloric deficit.

Table III-2 summarizes RCTs in which diet plus physical activity or physical activity alone was used to produce weight loss. Entries under percentage change were calculated as for Table III-1. Similar changes were observed as for diet alone, although there was perhaps a more consistent trend for all the lipid parameters with the combination of diet and physical activity and with physical activity alone.

visited on 6/14/2019

TABLE III-1:

Weight Reduction by Diet Alone

Study	N, Sex, and Duration	Weight Loss	Percentage Change TC	TG	LDL-C	HDL-C
Dengel [399]	28M experimental 14M control 9 months	-12	-9	-36	-9	-3
Hellenius[365]	40M experimental 39M control 6 months	BMI -2	-1	-2	-3	+2
Jalkanen[400]	24 experimental (wt) 22 experimental (lipid) 25 control (wt) 22 control (lipid) 12 months	-5	-7	-28	—	+8
Karvetti[368]	71F experimental 76F control 12 months	-6	-2	—	—	+12
	22M experimental 20M control 12 months	-11	0	—	—	+27
Marniemi[402]	37 (27F/10M) mixed diet 42 (32F/10M) control 12 months	-13	-4	-41	—	+18
	31 (23F/8M) lactovegetarian 42 (32F/10M) control 12 months	-11	-4	-21	—	+10
Puddey[370]	22M experimental 20M control 18 weeks	-8	-7	-27	—	+9
Simkin-Silverman[373]	253F experimental 267F control 6 months	-7	-8	-11	-9	+4
Svendsen[384]	50F experimental 20F control 12 weeks	-13	-18	-44	-22	+3
Wood[380]	40M experimental 40M control 12 months	-7	-5	-21	-5	+6
	31F experimental 39F control 12 months	-7	-7	-5	-8	-7
Wood[406]	42M experimental 42M control 12 months	-8	-2	-22	-3	+13

TC= Total Cholesterol TG= Triglycerides BMI= Body Mass Index
LDL-C= Low-Density Lipoprotein Cholesterol
HDL-C= High-Density Lipoprotein Cholesterol

visited on 6/14/2019

TABLE III-2:

Weight Reduction by Diet Plus Physical Activity or Physical Activity Alone

Study	N, Sex, and Duration	Weight Loss	Percentage Change TC	TG	LDL-C	HDL-C
Hellenius[365]	Diet+physical activity 39M experimental 39M control 6 month	BMI -4	-5	-12	-4	-1
	Physical activity only 39M experimental 39M control 6 months	BMI -2	0	-11	0	+2
King[401]	Physical activity only 40M high intensity group 41M control 12 months	BMI -1	—	-6	+2	-1
	Physical activity only 42M high intensity home 41M control 12 months	BMI -1	—	0	-1	+1
	Physical activity only 45M low intensity 41M control 12 months	BMI -4	—	-14	0	+3
	Physical activity only 34F high intensity group 34F control 12 months	BMI +2	—	+1	-4	+1
	Physical activity only 35F high intensity home 34F control 12 months	BMI 0	—	-2	+3	0
	Physical activity only 29F low intensity 34F control 12 months	BMI -2	—	+4	+3	0

TC= Total Cholesterol
TG= Triglycerides
BMI= Body Mass Index
LDL-C= Low-Density Lipoprotein Cholesterol
HDL-C= High-Density Lipoprotein Cholesterol

visited on 6/14/2019

Weight Reduction by Diet Plus Physical Activity or Physical Activity Alone (continued)

Study	N, Sex, and Duration	Weight Loss	Percentage Change TC	TG	LDL-C	HDL-C
Nilsson[403]	Diet+physical activity 30 (24M/6F) experimental 29 (22M/7F) control 12 months	BMI -2	-3	-4	-3	-1
Ronnemaa[404]	Physical activity only 13 (8M/5F) experimental 12 (7M/5F) control 4 months	-3	-6	-3	-5	+2
Schuler[405]	Diet+physical activity 56M experimental 57M control 12 months	-5	-10	-7	-10	+3
Svendsen[384]	Diet+physical activity 48F experimental 20F control 12 weeks	-14	-17	-35	-26	0
Wood[380]	Diet+physical activity 39M experimental 40M control 12 months	-11	-4	-46	-2	+17
	Diet+physical activity 42F experimental 39F control 12 months	-9	-5	-18	-8	+5

TC= Total Cholesterol
TG= Triglycerides
BMI= Body Mass Index
LDL-C= Low-Density Lipoprotein Cholesterol
HDL-C= High-Density Lipoprotein Cholesterol

visited on 6/14/2019

2.b. Pharmacotherapy Trials

Evidence Statement: *Weight loss produced by weight loss medications and adjuvant lifestyle modifications produces no consistent change in blood lipids. Evidence Category B.*

Rationale: The effects of pharmacological therapy on weight loss and subsequent changes in total serum cholesterol levels were evaluated by examining eight RCTs [386, 390-393, 395, 407,408] (Table III-3). The four trials of dexfenfluramine showed no consistent effects on total cholesterol; three trials showed decreases in triglycerides ranging from 14 to 40 percent and two trials showed increases in HDL-cholesterol of 8 percent in women to 27 percent in men. One trial of phentermine plus fenfluramine showed a 24 percent decrease in triglycerides and a slight change in HDL-cholesterol. [395] The trial of orlistat showed a decrease in total cholesterol and an increase in triglycerides, while the trial of fluoxetine showed the opposite. [407, 408] Other RCTs using orlistat have shown a modest improvement in lipids with weight loss. [388]

In a combined summary of data presented at the FDA Endocrinologic and Metabolic Drugs Advisory Committee Meeting (held in Bethesda, Maryland, in September 1996), use of sibutramine, which resulted in a greater net weight loss of 2.4 kg (5.3 lb) at 4 months, also resulted in modestly lower levels of total cholesterol, LDL-cholesterol, and triglycerides, along with slightly higher HDL-cholesterol levels, compared to placebos. [389]

2.c. Abdominal Fat

Evidence Statement: *Limited evidence suggests that decreases in abdominal fat correlate with improvements in the lipid profile of overweight individuals, although these improvements have not been shown to be independent of weight loss. Evidence Category C.*

Rationale: Four RCTs testing the effects of weight loss on blood lipids also included measures of abdominal fat, as measured by waist circumference. [365,373,375, 399] In each study, waist circumference was reduced along with weight, and blood lipids were improved. These studies were not designed to test whether reductions in abdominal fat reduce blood lipids independent of weight loss. However, evidence exists from epidemiologic observational studies that abdominal fat is related to an adverse lipid profile, including higher levels of total cholesterol, LDL-cholesterol, and triglycerides, and lower levels of HDL-cholesterol. [409,410]

2.d. Fitness

Evidence Statement: *Increased aerobic activity to increase cardiorespiratory fitness favorably affects blood lipids, particularly if accompanied by weight loss. Evidence Category A.*

Rationale: Nine RCTs testing the effects of weight loss on lipoproteins measured cardiorespiratory fitness, as measured by maximal oxygen uptake. [369, 375, 380, 384, 399, 401, 404-406] Weight loss was accompanied by increased fitness if the intervention included increased physical activity or physical activity combined with diet. [375, 380, 401, 404-406] These studies were not designed to test whether increased cardiorespiratory fitness reduces blood lipids independent of weight loss in overweight and obese adults. However, considerable evidence exists that aerobic exercise decreases triglycerides and total cholesterol and may increase HDL-cholesterol in men and women, particularly if accompanied by weight loss. [411]

visited on 6/14/2019

TABLE III-3:

WEIGHT REDUCTION BY DRUG THERAPY

Study	N, Sex, and Duration	Percentage Change				
		Weight Loss	TC	TG	LDL-C	HDL-C
Bremer[386]	Dexfenfluramine 12 (8M/4F) experimental 14 (8M/6F) control 12 weeks	-6	-14	-40	-9	+15
Mathus-Vliegen[391]	Dexfenfluramine 17 experimental 18 control 12 months	-4	+7	-14	—	—
O'Connor[392]	Dexfenfluramine 27 (6M/21F) experimental 24 (14M/10F) control 6 months	-6	+5	-26	—	+27M +8F
Pfohl[393]	Dexfenfluramine 19 experimental 15 control 12 months	-2	-3	+26	—	—
Weintraub[395]	Fen/Phen 53 Fen/Phen 51 placebo 34 weeks	-13	-6	-24	-2	-1
Drent[407]	Orlistat 20 (3M/17F) Orlistat 19 (3M/16F) placebo 12 weeks	-2	-2	+8	—	—
O'Kane[408]	Fluoxetine 7 Fluoxetine 9 placebo	-6	+9	-25	—	—

TC= Total Cholesterol
TG= Triglycerides
BMI= Body Mass Index
LDL-C= Low-Density Lipoprotein Cholesterol
HDL-C= High-Density Lipoprotein Cholesterol

visited on 6/14/2019

RECOMMENDATION: *Weight loss is recommended to lower elevated levels of total cholesterol, LDL-cholesterol and triglycerides and to raise low levels of HDL-cholesterol in overweight and obese persons with dyslipidemia. Evidence Category A.*

3. Impaired Glucose Tolerance and Diabetes

Forty-nine articles of RCTs were reviewed to evaluate the effect of weight loss on fasting blood glucose and fasting insulin. Studies were conducted in individuals with normal blood glucose levels (fasting plasma glucose < 115 mg/dL [< 6.4 mmol/L]), in individuals with impaired glucose tolerance (fasting plasma glucose of < 140 mg/dL [7.8 mmol/L] or 2 hours postprandial plasma glucose of ≥ 140 to < 200 mg/dL [7.8-11.1 mmol/L]), or in individuals with diabetes (fasting plasma glucose of > 140 mg/dL or 2 hours postprandial plasma blood glucose ≥ 200 mg/dL).*

The methods of weight loss included diet, physical activity, or both; behavior therapy; and pharmacotherapy. The dietary interventions included low calorie and very low calorie, and those that promoted changes in diet composition, such as amount and type of dietary fat. Physical activity varied from controlled individualized exercise programs to informal group sessions coupled with behavior therapy and dietary changes. The degree of blinding of outcome measures was not always well described, nor was there a systematic consideration of concurrent effects of changes in medication or adherence to medication among diabetic patients enrolled in these weight reduction studies.

Of the 17 RCT articles considered acceptable, 10 included normoglycemic individuals, [367, 373, 386, 387, 390-393, 403, 412] 2 included patients with impaired glucose tolerance, [70, 369] and 5 included patients with type 2 diabetes. [362, 404, 408, 413, 414]

3.a.Lifestyle Trials

Evidence Statement: *Weight loss produced by lifestyle modifications, reduces blood glucose levels in overweight and obese persons without type 2 diabetes, and reduces blood glucose levels and HbA_{1c} in some patients with type 2 diabetes. Evidence Category A.*

Rationale: Nine RCTs examined lifestyle therapy. [70, 362, 367, 369, 373, 403, 404, 412, 413] Five RCTs among normoglycemic overweight individuals examined the effects of behavior therapy or lifestyle change on weight loss. Changes in fasting glucose or fasting insulin are used as secondary outcome measures. Improvements in the levels of fasting insulin with weight loss ranged from 18 to 30 percent. While weight loss was maintained, improvements were observed for fasting glucose, fasting insulin, and glucose and insulin levels during oral glucose tolerance testing compared to controls. [367, 373, 403, 412]

Three RCTs of lifestyle modifications were conducted among overweight diabetic patients. [362,404,413] One RCT of diet and physical activity in African-Americans aged 55 to 79 years with type 2 diabetes showed a 2.4 kg (5.3 lb) weight loss at 6 months compared to the usual-care control group. The intervention group also decreased HbA_{1c} by 2.4 percentage units at 6 months. [362] In another study, weight loss of 5 kg (11 lb) through diet resulted in a reduction of HbA_{1c} by 2 percentage units at 6 months compared with the controls. [413] At 1 year, the difference in weight loss between the two groups was reduced, and diabetic control was similar in each group. In the third study, weight loss, through physical activity in 25 type 2 diabetes patients randomly

* The reviewed articles used the "old" definitions of the American Diabetes Association (ADA) for impaired glucose tolerance and diabetes. As of November 1997, the new ADA definitions define "impaired fasting glucose" as those individuals having a fasting plasma glucose of 110 to 125 mg/dL, and "diabetes" as those individuals having a fasting plasma glucose of ≥ 126 mg/dL or 2 hours postprandial plasma glucose of ≥ 200 mg/dL.

visited on 6/14/2019

assigned to physical activity (13 patients) or control (12 patients) groups, was associated with improved HbA_{1c}. [404]

One RCT of 530 individuals in China with impaired glucose tolerance compared the effects of diet, physical activity, and diet plus physical activity on the incidence of diabetes. The data were analyzed for the group as a whole and for the lean and overweight groups separately. The overweight individuals in the diet and diet plus physical activity groups lowered their BMIs, while the lean individuals did not. After 6 years, the cumulative incidence rates of diabetes were significantly lower in all three intervention groups than they were in the controls (43.8 percent in the diet group, 41.1 percent in the physical activity group, 46 percent in the diet plus physical activity group, and 67.7 percent in the controls). This was true also if only the overweight persons were included in the analysis. [70]

3.b. Pharmacotherapy Trials

Evidence Statement: *Weight loss produced by weight loss medications has not been shown to be any better than weight loss through lifestyle modification for improving blood glucose levels in overweight or obese persons both with and without type 2 diabetes. Evidence Category B.*

Rationale: Eight RCT articles considered the effects of pharmacotherapy on weight loss and subsequent changes in blood glucose. [386, 387, 390-393 408, 414]

Six trials in nondiabetic individuals reported weight loss with anorexiant drugs. [386, 387 390-393] One study of 75 subjects with refractory obesity showed that those taking dexfenfluramine had a significantly better improvement in their blood glucose levels as compared to placebo controls. [390] Another trial showed that after 6 months on dexfenfluramine, fasting serum insulin levels fell from 105 at baseline to 89 at 6 months, but glucose levels did not decrease. [392] This study was difficult to interpret. Four other RCTs of dexfenfluramine failed to show any significant differences between the active drug and placebo groups for glucose or insulin, despite significant differences in weight reduction. [386, 387, 391, 393]

Among diabetic patients, there were two trials of anorexiant drugs. One trial of dexfenfluramine compared with standard clinic practice showed significantly greater weight loss at 3 months but not at 6 or 12 months. [414] Weight loss at 3 months was 3.4 kg (7.5 lb) in the dexfenfluramine group and 1.59 kg (3.5 lb) in the clinic group. At 6 months, the weight loss was 3.13 kg (6.9 lb) in the dexfenfluramine group and 1.70 kg (3.7 lb) in the clinic group. By 1 year, both groups had regained some weight. Despite more than a 1 kg (2.2 lb) difference of weight between the dexfenfluramine group and the control group, there were no significant differences between groups in changes from baseline for HbA_{1c} at any of the follow-up times (3, 6, or 12 months). In the trial of fluoxetine, those patients on 60 mg of fluoxetine daily compared with the placebo group had significant weight loss at 6 months, a finding that was associated with improvements in glucose and HbA_{1c}. At 12 months, these differences were no longer significant. [408] The RCTs of orlistat showed a drop in both fasting glucose and HBA_{1c} in diabetic patients on a combination therapy of lifestyle change and orlistat for weight loss. [388]

3.c. Abdominal Fat

Evidence Statement: *Decreases in abdominal fat improve glucose tolerance in overweight individuals with impaired glucose tolerance, although this has not been shown to be independent of weight loss. Evidence Category C.*

---

visited on 6/14/2019

Rationale: Two RCTs testing the effects of weight loss on changes in glucose tolerance and insulin also had measures of abdominal fat, as measured by waist circumference. [369,373] In each study, waist circumference was reduced along with weight loss and glucose tolerance improved. Neither study was designed to test whether reductions in waist circumference improved glucose tolerance or diabetic control independent of weight loss.

Evidence exists from observational studies that abdominal fat is related to impaired glucose tolerance or type 2 diabetes independent of BMI. For instance, a few studies have found a significant relationship between abdominal fat loss and improvement in glucose tolerance and insulin action. [415-418]

3.d. Fitness

Evidence Statement: *Increased cardiorespiratory fitness improves glucose tolerance in overweight individuals, but no evidence shows this relationship to be independent of weight loss. Evidence Category C.*

Rationale: Two RCTs that examined the effects of weight loss on glucose tolerance in older men [369] and in persons with diabetes [404] also had measures of VO_2 max (oxygen consumption) as a measure of fitness. In both studies, physical activity resulted in a net weight loss of approximately 2.3 to 2.5 kg (2 to 3 percent) and increased VO_2 max by 9 to 18 percent. In one study [419] most measures of glucose tolerance improved, whereas in the other study [369] only 2-hour insulin improved. Neither study was designed to test whether increased fitness improves glucose tolerance independent of weight loss.

RECOMMENDATION: *Weight loss is recommended to lower elevated blood glucose levels in overweight and obese persons with type 2 diabetes. Evidence Category A.*

4. Decreases in Abdominal Fat With Weight Loss

Evidence Statement: *Weight loss is associated with decreases in abdominal fat, as measured by waist circumference. Evidence Category A.*

Rationale: Six acceptable RCTs on weight loss that measured waist circumferences all show that weight loss is associated with reductions in waist circumference. [365, 369, 373, 375, 384, 399] Waist circumference is commonly used as a surrogate measure for abdominal visceral fat. [420, 421] Although no RCTs examined changes in visceral fat per se, one observational study [422] showed significant decreases in visceral fat with a mean weight loss of 12.9 kg (28.4 lb).

In addition, many observational and non-RCT interventional studies have reported that weight loss caused by a variety of treatments reduces abdominal visceral fat levels. [417, 423-428] Fat located in the abdominal region is associated with greater health risks than that in peripheral regions, e.g., the gluteal-femoral area. [155-159, 429, 430] Many studies used the waist-to-hip ratio as the measure of abdominal fat; however, the panel considered waist circumference to be a better marker of abdominal fat content than the waist-to-hip ratio. For more information on abdominal fat, see Section 4.B.

visited on 6/14/2019

B What Treatments are Effective?

A variety of modalities may be used to treat overweight and obesity. To assess their effectiveness on weight loss, loss of abdominal fat, and cardiorespiratory fitness, evidence from RCTs was examined. These trials considered dietary therapy, physical activity, combined therapy, behavior therapy, pharmacotherapy, and surgery. The details of the studies reviewed and subsequently not included are provided in Appendix II.

1. Dietary Therapy

Eighty-six RCT articles evaluated the effectiveness of many different diets on weight loss: low-calorie diets (LCDs), very low-calorie diets (VLCDs), vegetarian, American Heart Association guidelines and the National Cholesterol Education Program's Step I diet with caloric restriction, and other low-fat regimens with varying combinations of macronutrients. The trials that included participants who were not overweight were rejected. Some trials were done in patient populations with diabetes, hypertension, or hyperlipidemia. Many of the trials do not mention whether concomitant physical activity programs were promoted, and many do not give information about the behavioral approaches that were used to deliver the diet or physical activity intervention. Approximately 60 percent of the trials are equal to or greater than 6 months in duration.

1.a Low-Calorie Diets

Evidence Statement: *LCDs can reduce total body weight by an average of 8 percent over 3 to 12 months. Evidence Category A.*

Rationale: Thirty-four RCT articles examined the impact on weight loss of an LCD consisting of approximately 1,000 to 1,200 kilocalories/day. Many of these LCDs also promoted low fat intake as a practical way to reduce calories. Twenty-five RCT articles covered interventions lasting 6 or more months. [346-348, 350, 354, 355, 359, 363-366, 368, 369, 373, 375, 378-380, 399, 400, 406, 413, 431-433]

Another nine RCTs lasted 12 to 21 weeks. [349, 351, 356, 370, 371, 377, 384, 434, 435]

All of the studies, regardless of the length of the intervention, showed that LCDs result in weight loss. From the 25 RCT articles with a duration of ≥ 6 months, compared to controls, LCDs brought about a mean weight loss of approximately 8 percent of body weight over a period of 6 months and up to 1 year. The nine studies with an intervention lasting from 3 months up to 6 months also averaged approximately 8 percent weight loss compared to controls. Four studies that included a long-term weight loss and maintenance intervention lasting 3 to 4.5 years reported an average weight loss of 4 percent over the long term. [348, 359, 366, 379]

Evidence Statement: *LCDs resulting in weight loss effect a decrease in abdominal fat. Evidence Category A.*

Four RCTs testing the effects of LCDs alone on weight loss also had measures of abdominal fat, as measured by waist circumference. [365, 369, 375, 399] The studies showed consistently that waist circumference decreases along with weight loss produced by LCDs. After 6 to 12 months, LCDs resulted in a mean weight loss of as little as 0.3 BMI unit to as much as 11 kg (24.3 lb) body weight, and a concomitant reduction in waist circumference of 1.5 to 9.5 cm compared to controls.

visited on 6/14/2019

Evidence Statement: *No improvement in cardiorespiratory fitness as measured by VO_2 max appears to occur in overweight or obese adults who lose weight on LCDs without increasing physical activity. Evidence Category B.*

Rationale: Ten RCTs examined whether weight loss through diet alone without increased physical activity had an effect on cardiorespiratory fitness as measured by VO_2 max when compared to controls with no weight loss. Four RCTs reported no improvement in VO_2 max, [363,375,399,435] while six RCTs reported an improvement. [346, 369, 380, 384, 406, 432] In three of the six studies, improvement in those on the LCD was shown because the control group had a large decrease in VO_2 max. [346,406,432] In all six studies that showed improvement, VO_2 max was expressed as ml/kg/min rather than liters/min. Because the ratio of ml O_2/kg/min increases with weight loss alone and not necessarily from an improvement in oxygen uptake, results from studies reporting VO_2 max in terms of ml O_2/kg/min are equivocal. The three studies that reported VO_2 max as liters/min consistently showed no improvement from diet alone. [375,399,435]

1.b. Very Low-Calorie Diets

Evidence Statement: *VLCDs produce greater initial weight loss than LCDs. However, the long-term (> 1 year) weight loss is not different from that of the LCD. Evidence Category A.*

Four RCTs compared VLCDs to LCDs. [436-439] The VLCDs provided about 400 to 500 kcal/day, whereas the LCDs provided approximately 1,000 to 1,500 kcal/day. During the active phase, VLCDs were given exclusively for 12 to 16 weeks and then were followed by LCDs for a total duration ranging from 24 weeks to 5 years. The participants in these trials were primarily women who were extremely obese.

VLCDs either alone or, usually, combined with behavioral therapy, promoted weight loss of approximately 13 to 23 kg (28.7 to 50.7 lb) during the active phase of the VLCD intervention, compared to 9 to 13 kg (19.8 to 28.7 lb) with LCDs. In three of the four studies, VLCDs resulted in 4 to 12 kg (8.8 to 26.5 lb) greater weight loss than the LCDs at the end of the active phase. [436-438] Over the medium term of 6 to 12 months, weight loss on the VLCDs ranged from 1.1 kg (2.4 lb) to as much as 10.4 kg (22.9 lb) greater than weight loss on LCDs. One study did not show a particular advantage of VLCD over LCD either during the active phase or at 24 weeks. [439] After 1 year, there was no long-term advantage of VLCDs over LCDs. [436, 437]

1.c. Lower-Fat Diets

Evidence Statement: *Although lower-fat diets without targeted caloric reduction help promote weight loss by producing a reduced caloric intake, lower-fat diets coupled with total caloric reduction produce greater weight loss than lower-fat diets alone. Evidence Category A. Lower-fat diets produce weight loss primarily by decreasing caloric intake. Evidence Category B.*

Rationale: Nine RCTs testing the effects of diet on weight loss promoted diets that varied in fat and caloric content. [317, 364, 369, 434, 440-444] Lower-fat diets varied from 20 to 30 percent of calories from fat, and calories ranged from 1,200 to 2,300 calories.

visited on 6/14/2019

Three RCTs, all 6 months or greater in duration, promoted lower-fat diets with ad libitum caloric intake. Two of the three RCTs reported that lower-fat diets with ad libitum energy intake resulted in a reduction in caloric intake of 85 kcal and 300 kcal, [317, 444] and all three RCTs produced greater weight loss by 1 to 3.9 kg (2.2 to 8.6 lb), compared to a higher-fat diet. [317, 441, 444] The third RCT had a large disparity in baseline caloric intake between the two diet groups, making the reported caloric intakes at the end of the study difficult to compare. [441]

Three RCTs compared lower-fat diets with targeted caloric reduction to lower-fat diets alone, and all three found that weight loss is greater in the lower-fat diet with caloric reduction than with the lower-fat diet alone. [369, 434, 442]

When similar caloric levels occur between lower-fat and higher-fat diets, similar amounts of weight loss were reported in two studies, [364, 443] whereas one study showed 1.8 kg (4 lb) greater weight loss on the lower-fat compared with the higher-fat diet, despite similar reported calorie levels. [440]

Taken together, these studies show that lower-fat diets ranging from 20 to 30 percent of calories can contribute to lower caloric intake even when caloric reduction is not the focus of the intervention, [317, 444] but when LCDs are targeted with lower-fat diets, better weight loss is achieved. [369, 434, 442] However, there is little evidence that lower-fat diets (per se) cause weight loss independent of caloric reduction.

RECOMMENDATION: *LCDs are recommended for weight loss in overweight and obese persons. Evidence Category A. Reducing fat as part of an LCD is a practical way to reduce calories. Evidence Category A.*

2. Physical Activity

Twenty-four RCT articles were reviewed for the effect of physical activity on weight loss, abdominal fat (measured by waist circumference), and changes in cardiorespiratory fitness (VO_2 max). Thirteen articles were deemed acceptable. [346, 363, 365, 369, 375, 401, 404, 406, 432, 434, 445-447] Only one of these RCTs compared different intensities and format with a control group, although the goal was to increase physical activity and not specifically to produce weight loss. [401] Results from this trial were subsequently reported after 2 years, but these no longer included the control group. [447] One additional study did not have a no-treatment control group, but compared three active treatment groups with each other: diet only, exercise only, and combination exercise plus diet. [448]

Most RCTs described the type of physical activity as cardiovascular endurance activities in the form of aerobic exercise such as aerobic dancing, brisk walking, jogging, running, riding a stationary bicycle, swimming, and skiing, preceded and followed by a short session of warmup and cool down exercises. Some physical activity programs also included unspecified dynamic calisthenic exercises. [363, 369, 406, 446]

The intensity of the physical activity was adapted to each individual and varied from 60 to 85 percent of the individual's estimated maximum heart rate, or was adjusted to correspond to approximately 70 percent of maximum aerobic capacity (VO_2 max). The measure of physical fitness included VO_2 max. The frequency of physical activity varied from three to seven sessions a week and the length of the physical activity session ranged from 30 to 60 minutes. Some physical activity programs were supervised, and some were home-based. Adherence to the prescribed physical activity program was recorded and reported in some studies and not mentioned in others. Most studies did not estimate the caloric expenditure from the physical activity or report calorie intake. The duration of the intervention

visited on 6/14/2019

varied from 16 weeks to 1 year; six articles reported on trials that lasted at least 1 year. [346, 363, 375, 401, 406, 432]

Evidence Statement: *Physical activity, i.e., aerobic exercise, in overweight and obese adults results in modest weight loss independent of the effect of caloric reduction through diet. Evidence Category A.*

Rationale: Twelve RCT articles examined the effects of physical activity, consisting primarily of aerobic exercise, on weight loss compared to controls. [346, 363, 365, 369, 375, 401, 404, 406, 432, 434, 445, 446] Ten of the 12 RCT articles reported a mean weight loss of 2.4 kg (5.3 lb) (or 2.4 percent of weight) [363, 369, 375, 406, 419, 432, 434] or a mean reduction in BMI of 0.7 kg/m^2 (2.7 percent reduction) [346, 365, 401] in the exercise group compared to the control group. In three of these ten studies, the weight loss was < 2 percent of body weight (< 2 kg) (4.4 lb). [369, 375, 434] In contrast, two RCTs showed no benefit on weight from exercise, reporting weight gain in the exercise group compared to the control group. [445, 446] In one of these studies, the control group received only diet advice but nevertheless lost 9 kg (19.8 lb), whereas the exercise group lost only 7 kg (15.4 lb). [445] In the second study, there was a total of only 10 participants, all having noninsulin-dependent diabetes mellitus, and the control group lost 3 kg (6.6 lb) whereas the exercise group lost only 2 kg (4.4 lb). [446] A meta-analysis of 28 publications of the effect on weight loss of exercise compared to diet or control groups showed that aerobic exercise alone produces a modest weight loss of 3 kg (6.6 lb) in men and 1.4 kg (3.1 lb) in women compared to controls. [449]

Ten articles reported on RCTs that had a diet-only group in addition to an exercise-only group. [346, 363, 365, 369, 375, 406, 432, 434, 445, 448] In every case except one, [365] the exercise-only group did not experience as much weight loss as the diet-only group. The diet-only group produced approximately 3 percent, or 3 kg (6.6 lb), greater weight loss than the exercise-only group.

No single study examined the length of the intervention in relation to the weight loss outcome. Only one study compared the effect on maximum oxygen uptake of different intensities and formats of physical activity over a 1-year follow-up [401] and 2-year follow-up period. [447] Better adherence over 1 year was found if the exercise was performed at home rather than in a group setting, regardless of the intensity level. Subsequently, the different exercise groups were compared with each other over the longer term (2 years), and better long-term adherence was found in the higher intensity home-based exercise group compared to the lower intensity home-based or higher intensity group-based exercise groups. [447]

The question of whether physical activity enhances long-term maintenance of weight loss has not been formally examined in RCTs. Examination of long-term weight loss maintenance produced by physical activity interventions compared with diet-only interventions cannot easily be compared between RCTs because of numerous differences between studies with respect to design, sample size, intervention content and delivery, and characteristics of the study population samples. However, a number of analyses of observational and post hoc analyses of intervention studies have examined whether physical activity has a beneficial effect on weight. Cross-sectional studies have generally shown that physical activity is inversely related to body weight [450-454] and rate of weight gain with age. [455] Longitudinal studies with 2 to 10 years of follow-up results have observed that physical activity is related to less weight gain over time [456-459], less weight gain after smoking cessation in women, [460] and weight loss over 2 years. [461] In

visited on 6/14/2019

addition, post hoc analyses of several weight loss intervention studies reported that physical activity was a predictor of successful weight loss. [454, 462, 463]

The results of these RCTs showed that physical activity produces only modest weight loss and observational analyses from other studies suggest that physical activity may play a role in long-term weight control and/or maintenance of weight loss.

Evidence Statement: *Physical activity in overweight and obese adults modestly reduces abdominal fat. Evidence Category B.*

Rationale: Only three RCTs testing the effect of physical activity on weight loss also had measures of abdominal fat as assessed by waist circumference. [365, 369, 375] One study demonstrated that physical activity reduced waist circumference compared with the control group, [365] and another study showed a small effect on waist circumference (0.9 cm) in men but not women. [375] One study in men showed a small increase in waist circumference. [369] Weight loss was modest in all of these studies. These studies were not designed to test the effects of physical activity on abdominal fat independent of weight loss.

However, several large studies in Europe, [464] Canada, [453] and the United States [465-468] reported that physical activity has a favorable effect on body fat distribution. These studies showed an inverse association between energy expenditure through physical activity and several indicators of abdominal obesity, such as waist circumference and waist-to-hip and waist-to-thigh circumference ratios.

Evidence Statement: *Physical activity in overweight and obese adults increases cardiorespiratory fitness independent of weight loss. Evidence Category A.*

Rationale: Eleven RCT articles testing the effect of physical activity alone on weight loss in men and women also included measures of cardiorespiratory fitness, as measured by maximal oxygen uptake (VO_2 max). [346, 363, 369, 375, 401, 404, 406, 432, 434, 445, 446] All 11 showed that physical activity increased maximum oxygen uptake in men and women in the exercise groups by an average of 14 percent (ml/kg body weight) to 18 percent (L/min). Even in studies with modest weight loss (< 2 percent), physical activity increased VO_2 max by an average of 12 percent (L/min) to 16 percent (ml/kg). [369, 375, 434]

One study that compared different formats and intensities of physical activity on VO_2 max reported that improvement in VO_2 max was related to adherence to the physical activity regime. In that study, the lower-intensity program was equally effective on VO_2 max as a higher-intensity program, largely as a result of different levels of adherence. [401]

The results of the RCTs strongly demonstrate that physical activity increases cardiorespiratory fitness in overweight and obese individuals.

RECOMMENDATION: *Physical activity is recommended as part of a comprehensive weight loss therapy and weight maintenance program because it: (1) modestly contributes to weight loss in overweight and obese adults (Evidence Category A), (2) may decrease abdominal fat (Evidence Category B), (3) increases cardiorespiratory fitness (Evidence Category A), and (4) may help with maintenance of weight loss (Evidence Category C).*

visited on 6/14/2019

3. Combined Therapy (Diet and Physical Activity)

Twenty-three RCT articles investigated the effects on body weight of a combination of a reduced calorie diet with increased physical activity. The control groups used diet alone or physical activity alone.

Of the 15 studies deemed acceptable, each of them compared the combined intervention with diet alone, [346, 365, 375, 377, 380, 384, 434, 435, 445, 448, 469-473] and 6 of them also compared the combined intervention to physical activity alone. [346, 365, 375, 434, 448, 474] The studies varied in terms of the length of the active intervention period as well as the length of the follow-up. Many of the programs lasted for more than 6 months, and five studies had intervention or follow-up data for at least 1 year. [346, 375, 380, 445, 473] Two studies included patients with type 2 diabetes. [470, 473] Participants in the diet and physical activity group most often participated in supervised group-based physical activity consisting of 30- to 60-minute sessions 3 times a week, and they were encouraged to maintain a maximum heart rate between 60 and 80 percent. There were three diet components: general dietary advice, caloric reduction of 500 to 1,000 kcal per day, or a diet containing 1,200 kcal per day.

Evidence Statement: *The combination of a reduced calorie diet and increased physical activity produces greater weight loss than diet alone or physical activity alone. Evidence Category A.*

Rationale: Of the 15 RCT articles, 12 observed that the combined diet and physical activity group had a mean greater weight loss of 1.9 kg (4.2 lb) and a mean greater BMI reduction of 0.4 (range of 0.3 to 0.5) than the diet-alone group. [346, 365, 375, 377, 380, 384, 434, 435, 448, 469, 470, 473]

One study compared different forms of physical activity in combination with diet and found that, compared to diet alone, the greatest weight loss occurred when the combination intervention included both aerobic and resistance training: 0.9 kg (2 lb) greater weight loss than aerobic exercise plus diet. [435] Another study reported that participants receiving resistance training in combination with diet had 2.2 and 5.0 kg (4.9 and 11 lb) greater weight loss than the diet-alone group after 24 and 48 weeks, respectively. [469]

Five of the six studies that compared combined intervention with physical activity alone observed that the combined intervention group had a 5.3 kg (11.7 lb) (range of 3.6 to 6.2 kg) (7.9 to 13.7 lb) greater weight loss and 0.9 change in BMI unit than the physical activity-alone group. [346, 365, 375, 434, 448] This greater weight loss was significant in three studies. [365, 375, 448]

Three RCTs compared the longer-term versus the shorter-term effects of the combination of physical activity and diet versus diet alone. [384, 469, 473] All three studies found that the combination resulted in approximately 1.5 to 3 kg (3.3 to 6.6 lb) greater weight loss than diet alone over the longer term of 9 months to 2 years.

Evidence Statement: *The combination of a reduced calorie diet and increased physical activity produces greater reductions in abdominal fat than either diet alone or physical activity alone, although it has not been shown to be independent of weight loss. Evidence Category B.*

Rationale: Three RCTs [365, 375, 471] that examined the combined effect of diet and physical activity on weight loss also had measures of abdominal fat, as measured by waist circumference. Two of the three RCTs showed that the combination intervention resulted in greater reduction in waist cir-

visited on 6/14/2019

cumference than diet alone in men [365, 375] and (only one) in women. [375] In the other RCT, no differences in reduced waist circumference were observed. [471] Of the three RCTs comparing combination therapy to physical activity alone, one study reported a greater waist reduction of 2.7 to 3.8 cm in women and men, respectively, [375] and by less than 1 cm in another study. [365] The effects of combination therapy on abdominal fat in these studies are not separable from the effects on weight loss.

Evidence Statement: *A combination of a reduced calorie diet and increased physical activity improves cardiorespiratory fitness as measured by VO_2 max when compared to diet alone. Evidence Category A.*

Rationale: Nine RCTs examined the effect of a combination of diet and physical activity on fitness compared to diet alone. [346, 375, 377, 380, 434, 435, 445, 448, 475] In most studies, the physical activity was in the range of 60 to 80 percent VO_2 max. All nine RCTs showed that a combination of diet and physical activity improved cardiorespiratory fitness, as measured by VO_2 max, more than diet alone, whether reported relative to body mass in ml O_2/kg/min or in absolute values by ml O_2/min. [346, 375, 377, 380, 434, 435, 445, 448, 475] Studies of longer duration tended to show the greatest improvement. This improvement was significant in six of the studies. [346, 375, 377, 380, 434, 448]

RECOMMENDATION: *The combination of a reduced calorie diet and increased physical activity is recommended, since it produces weight loss, decreases abdominal fat, and increases cardiorespiratory fitness. Evidence Category A.*

4. Behavior Therapy

Behavior therapy constitutes those strategies, based on learning principles such as reinforcement, that provide tools for overcoming barriers to compliance with dietary therapy and/or increased physical activity. Two questions are important in assessing the effect of behavior therapy in the treatment of overweight and obesity. First, does behavior therapy provide additional benefits above and beyond the other weight loss approaches—diet, physical activity, or drug therapy? Second, are there behavioral strategies that are more effective than others?

4.a. Additional Benefits Beyond Other Weight Loss Approaches

Thirty-six RCT articles were examined to evaluate whether behavior therapy provides additional benefits beyond other weight loss approaches. The acceptable RCTs compared behavior therapy plus another weight loss strategy to that weight loss strategy without behavior therapy.

Evidence Statement: *Behavior therapy, when used in combination with other weight loss approaches, provides additional benefits in assisting patients to lose weight short term (1 year). Evidence Category B. No additional benefits are found at 3 to 5 years in the absence of continued intervention. Evidence Category B.*

Rationale: Four RCTs addressed the question of whether behavior therapy, in order to reinforce a healthy diet, provides additional benefits above and beyond other weight loss approaches. Three studies compared behavior therapy plus dietary intervention to a dietary intervention alone. [436, 476, 477] No studies were found that examined the added benefit of behavior therapy when combined with either physical activity or diet and physical activity. One study was found that compared behavior therapy to drug therapy. [478]

visited on 6/14/2019

One study compared 36 obese women in one of three groups of 12 participating in a 16-week intervention: (1) individual counseling; (2) group counseling; and (3) group counseling plus behavior therapy. All groups lost weight at the end of treatment, and there were no significant differences among the groups. By the 1-year follow-up, participants in groups 1 and 3 were equally successful in maintaining weight loss, while most participants in group 2 regained most of their weight. [476]

Other studies examined the effect of VLCD alone and in combination with behavior therapy. [436, 477] Subjects were randomly assigned to either: (1) VLCD alone; (2) behavior therapy plus LCD (1,000 to 1,200 kcal), denoted as the behavior therapy-alone group; or (3) VLCD plus behavior therapy (combined treatment). They were assessed at the end of treatment and at 1- and 5-year follow-ups. At the end of treatment, and at the 1-year follow-up, weight loss was significantly greater for the combination group as compared to the other two groups. About one-third of those receiving behavior therapy (either in combination with an LCD or VLCD) were able to maintain their full end-of-treatment weight loss at 1 year, compared to only 5 percent of those in the diet-alone group. By the 5-year follow-up, the mean weight loss was not significantly different across groups.

Thus, although the weight loss results of behavioral treatment in combination with either an LCD or VLCD were quite good at the end of treatment and at the 1-year follow-up, they were not maintained by 5 years. These results may well have been due to the infrequent practice of the behavioral strategies by the subjects. Wadden and colleagues recommended that patients participate in a 6- to 12-month weight maintenance program after weight loss (which subjects did not do in the study described above), and that if they experience a gain of ≥5 kg (11 lb) which they cannot reverse on their own, that they should reenter a weight loss therapy program.

One study assessed the short- and long-term weight loss of behavior therapy, fenfluramine, or a combination of the two in 120 obese women. [478] Patients in the drug group and those in the combined drug plus behavior therapy group lost significantly more weight than did the group that received behavior therapy only and the control group; the drug-only group and combination group did not differ from each other at the end of the 6-month treatment. However, at the 1-year follow-up, significantly less weight was regained by the patients in the behavior-therapy-only group compared to the combined treatment group and the pharmacotherapy group, resulting in significantly better overall net weight loss. Results of this study suggest that the various interventions had different effects at the end of treatment and at follow-up.

To summarize, three of the four studies demonstrated that behavior therapy, when used in conjunction with other weight loss approaches, was more effective in reducing weight or delaying weight regain, either at the end of treatment or at the 1-year follow-up or both. [476-478] Behavior therapy was not better in its effect on weight loss at the 5-year follow-up.

No RCTs examined the additional effect of behavior therapy combined with diet and physical activity on cardiorespiratory fitness or abdominal fat.

4.b. Comparison of Behavior Therapy Strategies

Another way to examine the efficacy of behavior therapy in the treatment of overweight and obesity is to evaluate studies that compare various behavioral techniques with one another. Of 31 RCTs that compared one or more behavioral interventions, 19 were deemed acceptable.

visited on 6/14/2019

Evidence Statement: *No one behavior therapy appeared superior to any other in its effect on weight loss; rather, multimodal strategies appeared to work best and those interventions with the greatest intensity appeared to be associated with the greatest weight loss. Evidence Category A.*

Of the 19 RCTs, 17 produced weight loss. [367, 476, 479-493] Behavioral strategies to reinforce changes in diet and physical activity in obese adults produce weight loss in the range of 10 percent of baseline over 4 months to 1 year. The greatest amount of weight loss was usually observed in less than 12 months of the intervention; however, weight loss was observed for up to 2½ years in some studies. Multimodal strategies worked best, and the intensity (number of contacts and duration) and degree (albeit difficult to discern in some of these studies) of a particular reinforcement were most related to weight loss. Although initial therapy proved successful, it also appeared that when behavioral therapy ceased, weight was regained on average, but not all subjects relapsed back to baseline levels. Ten of the 19 studies were based on at least 1-year results. [367, 476, 479, 480, 484, 486-488, 492, 493]

Fourteen trials compared different behavior therapy strategies. Two trials compared cognitive behavior therapy techniques, specifically cognitive rehearsal, social pressure, and cue avoidance (see Glossary) with other behavior therapies. [479, 480] With regard to weight loss, cognitive rehearsal was no more effective than the group support or "social pressure" condition and was less effective than cue avoidance. [480] Another study indicated no clear benefits for cognitive rehearsal above and beyond other behavioral strategies. [479] One [483] of two studies that assessed the effect of monetary incentives reported greater weight loss. [367, 483]

The effect of extending treatment in various ways was evaluated in several studies. [486, 487, 489, 494] One study evaluated 20 weeks versus 40 weeks of standard behavior therapy and found greater weight loss with the extended therapy that was maintained at 72 weeks. [486] Another study evaluated behavior therapy and the effect of a post-treatment therapist on weight loss. [487] The study evaluated the effect of extended treatment using therapist contact in general, therapist contact emphasizing social influence, and extended treatment emphasizing physical activity. The emphasis of physical activity plus social influence produced greater weight loss than not having contact emphasizing these components. Extended contact with a therapist produced about 10 kg (22 lb) greater weight loss than no extended contact, even over the long term. At 18 months, all strategies that combined behavior therapy with a post-treatment maintenance program yielded a significantly greater long-term weight loss than behavior therapy alone.

Another study examined the effect of extending cognitive behavior therapy for 3 months in addition to the initial 3 months of therapy for obese patients with binge eating disorder, and found that extended cognitive behavior therapy improved binge eating behavior, although not weight. [494] A pilot study of women with type 2 diabetes compared a 16-week group-behavioral weight loss program to the same group, but with three individualized motivational interviewing sessions added. Both groups demonstrated significant weight loss with the motivational group losing 5.5 kg (12.1 lb) on average and the standard behavioral group losing 4.5 kg (9.9 lb); there were no differences between the two. However, those in the motivational interviewing group demonstrated better attendance at meetings (attending on average 13.3 meetings versus 8.9 meetings), had significantly better glucose control, and recorded blood glucose and food diaries more frequently [489] than did members of the standard behavioral group.

visited on 6/14/2019

A study that involved self-instruction plus incentives versus group instruction plus incentives demonstrated no effect of the group treatment on weight loss. [481] Weight loss was positively associated with attendance; however, mandatory attendance did not seem to increase program effectiveness and seemed to discourage attendance among men. The program was equally effective when implemented through either self-help materials or by professionals. Another study compared stimulus-narrowing during the reintroduction of food (achieved by providing prepackaged food) for those on VLCDs to providing regular food during the reintroduction. [495] They also compared reintroduction of foods based on progress in losing weight or maintaining weight loss versus a time-dependent basis. Neither strategy was helpful in maximizing weight loss or weight maintenance.

Two trials evaluated the impact of spousal involvement versus no spousal involvement in behavior therapy. [488, 492] One study found no overall differences in weight loss between those treated with spouses and those treated without spouses; however, women lost more weight when treated with a spouse, and men did better when treated alone ($P < .01$ at end of treatment; $P < .06$ at 1 year). [492] Another study found a positive effect of spousal involvement on weight loss soon after treatment was completed; however, this effect had largely dissipated by the 3-year follow-up. [488]

In summary, a variety of behavioral therapy strategies promote weight loss, with no one particular behavioral strategy having the greatest efficacy. Multimodal strategies with greater intensity appear to be most effective.

Evidence Statement: *Long-term follow-up of patients undergoing behavior therapy shows a return to baseline weight in the great majority of subjects in the absence of continued behavioral intervention. Evidence Category B.*

Rationale: Studies have shown that while weight loss is achieved, it is very difficult to maintain over a long period of time (1 to 5 years) in the absence of continued intervention. [436, 477, 486, 491] This emphasizes the great importance of continuing a maintenance program on a long-term basis.

Evidence Statement: *Little evidence exists on the effect of behavior therapy in combination with diet and physical activity on cardiorespiratory fitness.*

Rationale: One RCT examined the impact of different levels of behavior therapy in combination with diet and aerobic exercise on cardiorespiratory fitness. [484] Subjects were randomly assigned to one of three groups which varied in their degree of training in, and detailed application of, behavioral change principles (described as the basic, extended, and maximal behavioral therapy groups). All groups incorporated frequent intensive therapist contact, self-monitoring of behavior, regular objective assessment, and feedback of change in status. The three groups differed in the degree of emphasis given to behavioral self-management training, amount of detail provided on individual risk factors and overall coronary heart disease risk, and establishment of realistic short-term goals for coronary risk factor change. In terms of cardiorespiratory fitness, the group receiving the extended behavioral therapy and the group receiving the maximal behavior therapy showed improvements in VO_2 max of 2.90 and 3.40 mL/kg/min, respectively, when compared with controls who

visited on 6/14/2019

received the basic behavior therapy. However, it is unlikely that behavior therapy caused these improvements independent of weight loss.

No RCT evidence exists on the effect of behavior therapy combined with diet and physical activity on changes in abdominal fat as measured by waist circumference.

RECOMMENDATION: *Behavior therapy is a useful adjunct when incorporated into treatment for weight loss and weight maintenance. Evidence Category B.*

Commentary: Do the same treatment approaches for overweight and obesity fit diverse patient populations?

Whether or not the same weight reduction program will accomplish equivalent weight change in ethnically diverse population groups can be questioned on theoretical grounds. Weight reduction programs address motivations and behaviors strongly influenced by sociocultural factors that vary with ethnicity. [257, 496, 497] *Sociocultural factors may also influence the relative efficacy of behavioral programs in young, middle, and older adulthood; men or women; or any other patient groups where the motivations and lifestyle factors targeted may vary from those commonly assumed to be in operation. Innate or acquired physiological factors that affect energy metabolism may also influence the relative efficacy of behavioral weight reduction programs. For example, some cross-sectional comparisons have suggested that the relative weight loss achieved for a given degree of calorie restriction will be lower in African-Americans than whites because of a lower resting metabolic rate in African-Americans.* [498-501] *Another study suggests metabolic influences on weight loss in premenopausal versus postmenopausal women.* [502]

The diversity of populations in obesity treatment studies is limited, and there is not a coherent body of studies evaluating systematic differences in the outcomes of overweight and obesity treatment by age, socioeconomic status, or other participant characteristics that might be expected to influence outcome. However, the possibility of differences in the effectiveness of behavioral weight reduction approaches between African-Americans and whites has been suggested by reports from several intervention studies. [360, 503-505] *In some of these trials, weight reduction was the only intervention; in other instances it was combined with sodium reduction or other dietary or lifestyle changes. Taken together, these studies suggest that, on average, within the same program, weight losses of African-Americans are less than those of their white counterparts, particularly among women. A formal analysis of this issue in two RCTs—the Hypertension Prevention Trial and Phase 1 of the Trials of Hypertension Prevention—suggested that the weight gain in control participants was also higher among African-Americans than whites, but, in women, the net weight loss (i.e., active intervention minus control) was still less in African-Americans. There is ample evidence to support explanations of this differential weight loss on the basis of cultural or behavioral factors related to program adherence.* [257] *However, data to evaluate the alternative or additional contribution of metabolic factors to this differential are lacking. To date, no published studies permit separation of metabolic and behavioral influences on the observed ethnic differences in weight loss. (Also see commentary on pages 89 to 91 under treatment guidelines.)*

visited on 6/14/2019

RECOMMENDATION: *The possibility that a standard approach to weight loss will work differently in diverse patient populations must be considered when setting expectations about treatment outcomes. Evidence Category B.*

5. Pharmacotherapy

Since 1995, the use of the prescription drugs fenfluramine or dexfenfluramine for weight loss had increased greatly to 14 million prescriptions in 1½ years. The increased interest in drug treatment of obesity derives from the poor long-term results often obtained with behavior therapy, including diet and physical activity, as noted earlier in this report. The rationale for the addition of drugs to these regimens is that a more successful weight loss and maintenance may ensue. However, as of September 1997, the FDA requested the voluntary withdrawal of fenfluramine and dexfenfluramine from the market, due to a reported association between valvular heart disease with the drugs dexfenfluramine and fenfluramine, alone or combined with phentermine. [761] In November 1997, the FDA provided clearance for marketing the drug sibutramine hydrochloride monohydrate for the management of obesity, including weight loss and maintenance of weight loss when used in conjunction with a reduced-calorie diet. Due to the rapidly evolving information regarding the use of pharmacotherapy for weight loss, the panel decided to present their critique of those pharmacotherapy trials meeting their criteria for consideration.

Forty RCT articles evaluated the effect of pharmacotherapy on weight loss. In most studies, advice or behavioral therapy promoting reduced energy intake and increased physical activity was included in all treatment arms, including the placebo group.

Drugs that have been evaluated alone or in combination for at least 1 year by RCTs are phentermine, d,l-fenfluramine, dexfenfluramine, sibutramine, and orlistat. Two reports addressing dexfenfluramine [390, 506] represent a subset of a larger multicenter trial, the International Dexfenfluramine Study (INDEX trial). [507] Of the nine trials with dexfenfluramine, four had interventions of approximately 1 year, three of approximately 6 months, and two of 3 months. Most of the trials included a majority of white women, and some included persons with diabetes. Most of the trials have weight regain data. One RCT [508] evaluated the effect of dexfenfluramine versus placebo on patients who had been on an 8-week VLCD for an additional 24 weeks. One RCT [509] evaluated the effect of phentermine versus placebo on patients with osteoarthritis. One RCT [510] evaluated the effect of various doses of sibutramine on weight loss over a 24-week period. Two RCTs of 12 and 8 weeks in duration studied phenylpropanolamine versus placebo. [511, 512] One used phenylpropanolamine with diet, [512] and the other with diet, exercise, and behavior therapy. [511] Phenylpropanolamine is available as an over-the-counter drug. The average weight loss after 8 weeks of treatment was 2.59 kg (5.7 lb) for the drug versus 1.07 kg (2.4 lb) for the placebo, a difference of 1.5 kg (3.3 lb). [511] Short-term, but not long-term (over 1 year), effectiveness has been documented.

Evidence Statement: *Pharmacotherapy, which has generally been studied along with lifestyle modification including diet and physical activity, using dexfenfluramine, sibutramine, orlistat, or phentermine/fenfluramine, results in weight loss in obese adults when used for 6 months to 1 year. Evidence Category B.*

---

visited on 6/14/2019

Rationale: Data reporting the results of 52 weeks of drug treatment are available only for dexfenfluramine, orlistat, phentermine, d,l-fenfluramine, and sibutramine. The effect on weight loss from these drugs after 1 year is modest: 2.6 kg (5.7 lb) difference on average between dexfenfluramine and placebo (9.8 kg [21.6 lb] versus 7.2 kg [15.9 lb], baseline weights of 98.0 kg [216 lb] and 96.6 kg [213 lb]). [507] For sibutramine, a difference of 2.8 kg (6.2 lb) was seen at 1 year (4.4 kg [9.7 lb] versus 1.6 kg [3.5 lb]). [513] In another study of sibutramine, the difference was a net loss of 5.5 kg (12.1 lb) at 24 weeks with a 20 mg dose. [510] In all of the weight loss studies that were up to 1 year in duration, the majority of the weight loss occurred in the first 6 months with a plateauing or actual increase in weight in the following 6 months. [388, 389, 507, 514] For orlistat, a difference of 2.2 kg (4.9 lb) (4.3 kg [9.5 lb] versus 2.1 kg [4.6 lb]) was found in a 16-week study. [407] After a 210-week study on phentermine and d,l-fenfluramine, participants averaged a weight loss of 1.4 kg (3.1 lb) from baseline weight at week 210. [395, 514]

There are no known pharmacologic agents whose specific effect is to reduce abdominal fat or to improve cardiorespiratory fitness. Any improvements in abdominal fat or fitness would be secondary to weight loss or other interventions.

Sibutramine is approved for long-term use. (Note: FDA approval of orlistat is pending a resolution of labeling issues and results of Phase III trials.) Such drugs can then be used as an adjunct to modifications in behavior, including diet and physical activity. Drugs should be discontinued if significant weight loss is not achieved, i.e., a loss of < 4 pounds in the first 4 weeks, or if serious adverse effects occur.

RECOMMENDATION: *Weight loss drugs may only be used as part of a comprehensive weight loss program including diet and physical activity for patients with a BMI of ≥30 with no concomitant obesity-related risk factors or diseases, or for patients with a BMI of ≥27 with concomitant obesity-related risk factors or diseases. Evidence Category B.*

6. Surgery

Fourteen RCTs compared the weight-reducing effect of different surgical interventions. They did not compare surgery versus no intervention. Because of the nature of the intervention, there is a reluctance to do such a randomized trial.

Evidence Statement: *Surgical interventions in adults with a BMI ≥ 40 or a BMI ≥ 35 with comorbid conditions result in substantial weight loss. Evidence Category B.*

Rationale: Of the 14 RCTs that examined the effect of surgical procedures on weight loss, 8 were deemed appropriate. All the studies included individuals who had a BMI of 40 kg/m^2 or above, or a BMI of 35 to 40 kg/m^2 with comorbidity; most of the study participants were women. Weight loss due to surgical intervention such as the gastric bypass ranged from 50 kg (110 lb) to as much as 100 kg (220 lb) over a period of 6 months to 1 year. Gastroplasty with diet had a favorable net outcome on weight loss after 2 years compared to diet alone. [515] Vertical-banded gastroplasty was more effective than horizontal-banded gastroplasty. [516] Gastric resection with a modest biliopancreatic diversion without intestinal exclusion resulted in significantly greater weight loss than conventional Roux-en Y

visited on 6/14/2019

gastric bypass; this long-limb modification of Roux-en Y gastric bypass was shown to be safe and effective in patients who were 200 lb or more overweight and did not cause additional metabolic sequelae or diarrhea. [517]

The Swedish Obesity Study (SOS), [518] a non-RCT, found that gastric bypass produced greater weight loss than gastroplasty, 42.3 kg (93.3 lb) versus 29.9 kg (67 lb), at 1 year. The gastric bypass was deemed superior to gastroplasty or gastric partitioning in other RCTs as well. [518-522] The SOS also evaluated the correlation between stoma size and weight loss in patients assigned to gastric bypass or gastroplasty, and found no significant correlation in the gastric bypass group.

Comorbidity factors associated with weight loss showed improvement after surgery. One study showed that medical illnesses either improved (47 percent) or resolved (43 percent) in all but four patients (9 percent), and these four had unsatisfactory weight loss. [517] The Adelaide Study showed that 60 percent of the patients who initially had obesity-related comorbidity were free of medication for these comorbidities 3 years after surgery. [519]

Extremely obese persons often do not benefit from the more conservative treatments for weight loss and weight maintenance. Obesity severely impairs quality of life, and these individuals are at higher risk for premature death. [523] The National Institutes of Health Consensus Development Conference consensus statement, "Gastrointestinal Surgery for Severe Obesity, [523]" concluded that the benefits outweigh the risks and that this more aggressive approach is reasonable in individuals who strongly desire substantial weight loss and have life-threatening comorbid conditions.

The effects of surgery on abdominal fat or cardiorespiratory fitness independent of weight loss are unknown. One small study demonstrated that surgery (adjustable silicone gastric banding) reduced abdominal visceral fat. No analyses were performed to examine whether visceral fat reduction was independent of weight loss. [524] There are no data on the effects of surgery on changes in cardiorespiratory fitness.

RECOMMENDATION: *Surgical intervention is an option for carefully selected patients with clinically severe obesity (a BMI ≥40 or ≥35 with comorbid conditions) when less invasive methods of weight loss have failed and the patient is at high risk for obesity-associated morbidity and mortality. Evidence Category B.*

7. Other Interventions for Overweight and Obesity Treatment

Other types of interventions considered for overweight and obesity treatment included acupuncture, herbal remedies, supplements, and hypnosis. However, these treatments did not fulfill the *a priori* inclusion criteria, and for that reason were not included in this review.

visited on 6/14/2019

4

Treatment Guidelines

A Overview

The presence of overweight and obesity in a patient is of medical concern because it increases the risk for several diseases, particularly cardiovascular diseases (CVDs) and diabetes mellitus (see Chapter 2.C.) and it increases all-cause mortality. Treatment of the overweight and obese patient is a two-step process: assessment and management. Assessment requires determination of the degree of obesity and absolute risk status. Management includes both weight control or reducing excess body weight and maintaining that weight loss as well as instituting other measures to control associated risk factors. The aim of this guideline is to provide useful advice on how to achieve weight reduction and maintenance of a lower body weight. Obesity is a chronic disease, and both the patient and the practitioner need to understand that successful treatment requires a life-long effort.

B Assessment and Classification of Overweight and Obesity

1. Assessment of Overweight and Obesity

- *Determination of total body fat.* Overweight is defined as a body mass index (BMI) of 25 to 29.9 kg/m^2. Obesity is defined as an excess of total body fat that is documented by a BMI of ≥30 kg/m^2. Several methods are available for determining or calculating total body fat: total body water, total body potassium, bioelectrical impedance, and dual-energy X-ray absorptiometry.

Evidence Statement: *Measures of body fat give reasonably equivalent values for following overweight or obese patients during treatment. Evidence Category D.*

Rationale: Even though accurate methods to assess body fat exist, measuring body fat content by these techniques is often expensive and is not readily available clinically. Although bioelectrical impedance devices are becoming more readily available, they lose accuracy in severely obese persons and are of limited usefulness for tracking changes in total body fat in persons losing weight. Thus, bioelectrical impedance offers no significant advantage over BMI in the clinical management of patients. No trial data exist to indicate that one measure of fatness is better than any other for following overweight and obese patients during treatment. No studies have been published to compare the effectiveness of different measures for evaluating changes in body fat during weight reduction. BMI provides a more accurate measure of total body fat than relying on weight alone. It has an advantage over percent above ideal weight (e.g., based on the Metropolitan Life Insurance Tables). Ideal body weight tables were developed primarily from white, higher socioeconomic status populations and have not been documented to accurately reflect body fat content in the public at large. In addition, separate tables are required for men and women. The weight tables also are based on

visited on 6/14/2019

mortality outcomes and do not necessarily predict morbidity. BMI is recommended as a practical approach for the clinical setting. BMI provides an acceptable approximation for assessment of total body fat for the majority of patients. [525-527] However, simply measuring body weight is a practical approach to follow weight changes.

RECOMMENDATION: *Practitioners should use the BMI to assess overweight and obesity. Body weight alone can be used to follow weight loss and to determine efficacy of therapy. Evidence Category C.*

Rationale: The panel concentrated on tools available in the office, i.e., weight, height and the BMI. BMI is a practical indicator of the severity of obesity, and it can be calculated from existing tables. BMI is a direct calculation based on height and weight, regardless of gender. The limitations of BMI as a measure of total body fat, nonetheless, must be recognized. For example, BMI overestimates body fat in persons who are very muscular and can underestimate body fat in persons who have lost muscle mass (e.g., the elderly).

The BMI is calculated as follows:

BMI = weight (kg)/height squared (m^2)

To estimate BMI from pounds and inches use:

[weight (pounds)/ height (inches)^2] x 703 [528]

(1 lb = 0.4536 kg)
(1 in = 2.54 cm = 0.0254 m)

A simple BMI chart is provided in Appendix V. A patient should be weighed with shoes off and clad only in a light robe or undergarments.

- *Determination of degree of abdominal obesity.* The presence of excess fat in the abdomen, out of proportion to total body fat, is an independent predictor of risk factors and morbidity. Relatively accurate measures of total abdominal fat can be made by magnetic resonance imaging [529] or computed tomography. [500, 530] These methods, however, are expensive and not readily available for clinical practice. Research with these techniques, nonetheless, has shown that the waist circumference correlates with the amount of fat in the abdomen, and thus is an indicator of the severity of abdominal obesity. In this document the term "waist" circumference is used instead of "abdominal" circumference because it more accurately describes the anatomical site of measurement.

Evidence Statement: *Waist circumference is the most practical anthropometric measurement for assessing a patient's abdominal fat content before and during weight loss treatment. Computed tomography and magnetic resonance imaging are both more accurate but are impractical for routine clinical use. Evidence Category C.*

Rationale: Fat located in the abdominal region is associated with greater health risks than that in peripheral regions, e.g., the gluteal-femoral area. [155-159, 429, 430] Abdominal fat is described as having three compartments: visceral, retroperitoneal, and subcutaneous. [529, 531, 532] Several studies suggest that the visceral fat component of abdominal fat is the most strongly correlated with risk factors; [160, 161, 533, 534] other studies, however, indicate that the subcutaneous component is the most highly correlated with insulin resistance. [529, 535, 536] Therefore, the relative contributions of different components of abdominal fat to overall risk remain to be determined with certainty. Nonetheless, the presence of increased total abdominal fat appears to be an independent risk

visited on 6/14/2019

predictor when the BMI is not markedly increased. [537] Therefore, waist or abdominal circumference, as well as BMI, should be measured not only for the initial assessment of obesity, but also as a guide to the efficacy of weight loss treatment.

The waist-to-hip ratio (WHR) also has been used in a number of epidemiologic studies to show increased risk for diabetes, coronary artery disease, and hypertension. [500] However, waist circumference has been found to be a better marker of abdominal fat content than is WHR. [85] Whether WHR imparts any independent information about disease risk beyond waist circumference is uncertain, but between the two, the waist circumference appears to carry greater prognostic significance. Therefore, in clinical practice, abdominal fat content should be assessed and followed by measuring waist circumference.

RECOMMENDATION: *The waist circumference should be used to assess abdominal fat content. Evidence Category C.*

2. Classification of Overweight and Obesity

- *According to BMI.* The primary classification of obesity is based on the measurement of BMI. This classification is designed to relate BMI to risk of disease. It should be noted that the relation between BMI and disease risk varies among individuals and among different populations. Therefore, the classification must be viewed as a broad generalization. Individuals who are very muscular may have a BMI placing them in an overweight category when they are not overly fat. Also, very short persons (under 5 feet) may have high BMIs that may not reflect overweight or fatness. In addition, susceptibility to risk factors at a given weight varies among individuals. Some individuals may have multiple risk factors

Instructions for Measuring Waist Circumference, According to NHANES III Protocol

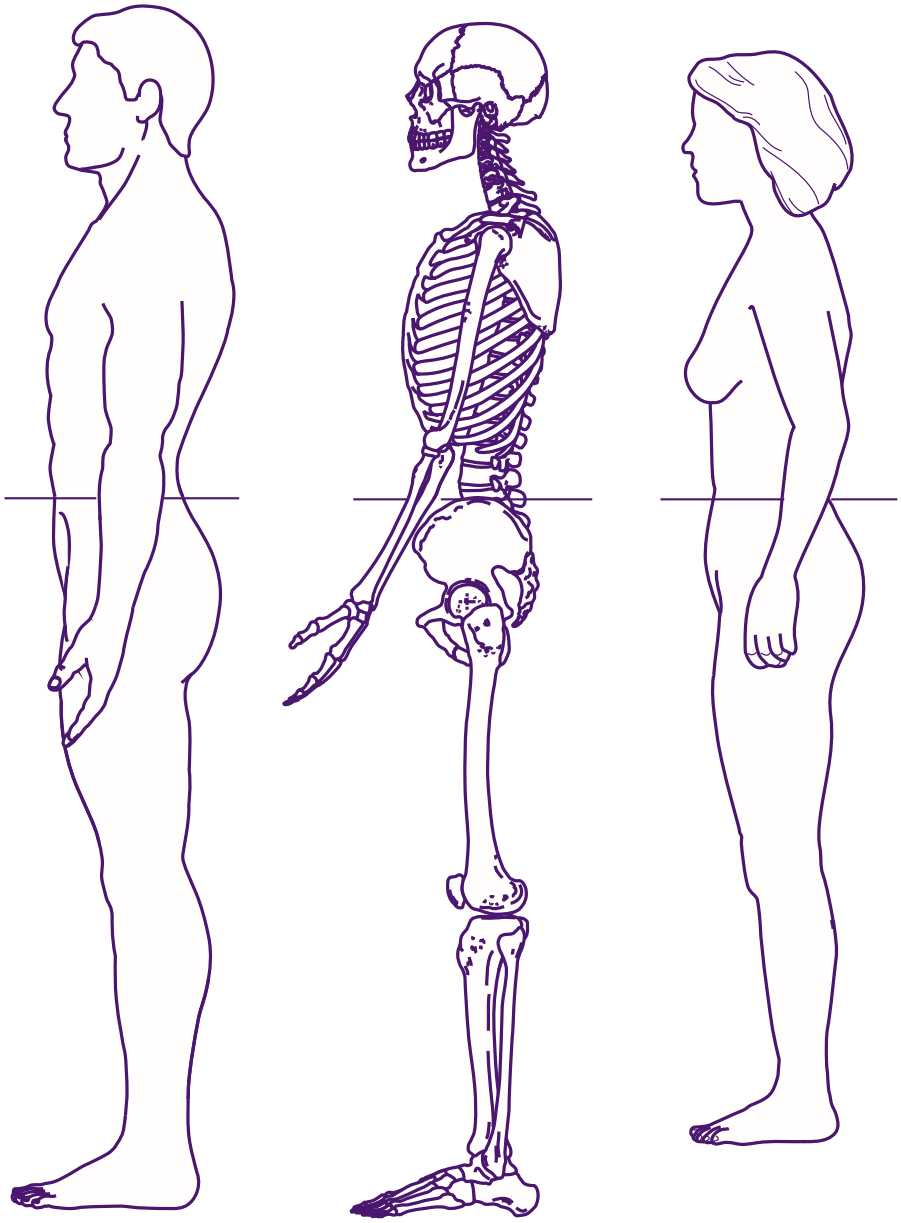
To define the level at which waist circumference is measured, a bony landmark is first located and marked. The subject stands and the examiner, positioned at the right of the subject, palpates the upper hip bone to locate the right iliac crest. Just above the uppermost lateral border of the right iliac crest, a horizontal mark is drawn, then crossed with a vertical mark on the midaxillary line. The measuring tape is placed in a horizontal plane around the abdomen at the level of this marked point on the right side of the trunk. The plane of the tape is parallel to the floor and the tape is snug, but does not compress the skin. The measurement is made at a normal minimal respiration (see Figure 5).

REF: U.S. Department of Health and Human Services, PHS. NHANES III Anthropometric Procedures Video. U.S. Government Printing Office Stock Number 017-022-01335-5. Washington, D.C.: U.S. GPO, Public Health Service; 1996. [538]

visited on 6/14/2019

with mild obesity, whereas others may have fewer risk factors with more severe obesity. It should also be noted that the risk levels shown for each increment in risk are relative risks; that is, relative to risk at normal weight. They should not be equated with absolute risk which is determined by a summation of risk factors. No randomized controlled trial studies exist that support a specific system for classification that establishes the degree of disease risk for patients during weight loss or weight maintenance. The classification is based on observational and prospective epidemiological studies.

Figure 5. Measuring tape position for waist (abdominal) circumference



visited on 6/14/2019

RECOMMENDATION: *The BMI * should be used to classify overweight and obesity and to estimate relative risk for disease compared to normal weight. Evidence Category C.*

Rationale: Overweight and obesity are classified by BMI (see Table IV-1).

Calculating BMI is simple, rapid, and inexpensive. The classification can be applied generally to adults. See Appendix III for comments about obesity and its classification in children. The basis for this BMI classification scheme stems from observational and epidemiologic studies which relate BMI to risk of morbidity and mortality. [11, 81-83, 131, 155, 278, 539-541] For example, relative risk for CVD increases in a graded fashion with increasing BMI in all population groups, although absolute risk in obese versus nonobese persons depends on the summed contribution of all risk factors.

Evidence Statement: *The same BMI cutpoints (see Table IV-1) can be used to classify the level of overweight and obesity for adult men and adult nonpregnant women, and generally for all racial/ethnic groups. Evidence Category C.*

Rationale: Clinical judgment must be used in interpreting BMI in situations in which it may not be an accurate indicator of total body fat. Examples are the presence of edema, high muscularity, muscle wasting, or for very short people. The relationship between BMI and body fat content varies somewhat with age, sex, and possibly ethnicity because of differences in factors such as composition of lean tissue, sitting height, and hydration state. [526, 542] For example, older persons often have lost muscle mass and have more fat for a given BMI than younger persons, women may have more fat for a given BMI than men, and persons with clinical edema

TABLE IV-1:

CLASSIFICATION OF OVERWEIGHT AND OBESITY BY BMI*

	Obesity Class	BMI (kg/m²)
Underweight		< 18.5
Normal		18.5 – 24.9
Overweight		25.0 – 29.9
Obesity	I	30.0 – 34.9
	II	35.0 – 39.9
Extreme Obesity	III	≥ 40

Source (adapted from): Preventing and Managing the Global Epidemic of Obesity. Report of the World Health Organization Consultation of Obesity. WHO, Geneva, June 1997.[65]

* Pregnant women who, on the basis of their prepregnant weight, would be classified as obese may encounter certain obstetrical risks. However, the inappropriateness of weight reduction during pregnancy is well recognized. [44] Hence, this guideline specifically excludes pregnant women.

visited on 6/14/2019

may have less fat for a given BMI than persons without edema. Nevertheless, these differences generally do not markedly influence the validity of BMI cutpoints either for classifying individuals into broad categories of overweight and obesity or for monitoring weight status of individuals in clinical settings. [526]

- *According to waist circumference.* Although waist circumference and BMI are interrelated, waist circumference provides an independent prediction of risk over and above that of BMI. Waist circumference measurement is particularly useful in patients who are categorized as normal or overweight on the BMI scale. At BMIs ≥ 35, waist circumference has little added predictive power of disease risk beyond that of BMI. It is therefore not necessary to measure waist circumference in individuals with BMIs ≥ 35.

Evidence Statement: *Sex-specific cutoffs for waist circumference can be used to identify increased risk associated with abdominal fat in adults with a BMI in the range of 25 to 34.9 kg/m^2. An increase in waist circumference may also be associated with increased risk in persons of normal weight. Evidence Category C.*

Waist circumference cutpoints can generally be applied to all adult ethnic or racial groups. On the other hand, if a patient is very short (under 5 feet) or has a BMI above the 25 to 34.9 kg/m^2 range, waist cutpoints used for the general population may not be applicable. Evidence Category D.

Rationale: A high waist circumference is associated with an increased risk for type 2 diabetes, dyslipidemia, hypertension, and CVD in patients with a BMI in a range between 25 and 34.9 kg/m.^{2} [82] Monitoring changes in waist circumference over time may be helpful, in addition to measuring BMI, since it can provide an estimate of increased abdominal fat even in the absence of a change in BMI. Furthermore, in obese patients with metabolic complications, changes in waist circumference are useful predictors of changes in CVD risk factors. [537] The waist circumference at which there is an increased relative risk is defined as follows:

HIGH RISK
Men > 102 cm (> 40 in)
Women > 88 cm (> 35 in)

To evaluate health risks, it is not necessary to measure waist circumference in patients with a BMI ≥ 35 kg/m^2; the measurement usually will be greater than the cutpoints given above, and waist measurements lose their predictive power at very high BMIs.

There are ethnic and age-related differences in body fat distribution that modify the predictive validity of waist circumference as a surrogate for abdominal fat. [526] These variations may partly explain differences between ethnic or age groups in the power of waist circumference or WHR to predict disease risks. [429, 543]

In some populations, waist circumference is a better indicator of relative disease risk than is BMI; examples include Asian-Americans or persons of Asian descent living elsewhere. [51, 273, 544] Waist circumference also assumes greater value for estimating risk for obesity-related disease at older ages. Table IV-2 incorporates both BMI and waist circumference in the classification of overweight and obesity, and provides an indication of disease risk.

visited on 6/14/2019

RECOMMENDATION: *For adult patients with a BMI of 25 to 34.9 kg/m², sex-specific waist circumference cutoffs should be used in conjunction with BMI to identify increased disease risk. Evidence Category C.*

C Assessment of Risk Status

The patient's risk status should be assessed by determining the degree of overweight or obesity based on BMI, the presence of abdominal obesity based on waist circumference, and the presence of concomitant CVD risk factors or comorbidities. Some obesity-associated diseases and risk factors place patients in a very high risk category for subsequent mortality. These diseases will require aggressive modification of risk factors in addition to their own clinical management. Other obesity-associated diseases are less lethal, but still require appropriate clinical therapy. Obesity also has an aggravating influence on several cardiovascular risk factors. Identifying these risk factors is required as a guide to the intensity of clinical intervention.

1. Determination of relative risk status based on overweight and obesity parameters.
Table IV-2 defines relative risk categories according to BMI and waist circumference. It is important to note that these categories denote *relative* risk, not *absolute* risk. They relate to the need to institute weight loss therapy, and do not directly define the required intensity of risk factor modification. The latter is determined by estimation

TABLE IV-2:

Classification of Overweight and Obesity by BMI, Waist Circumference and Associated Disease Risk*

			Disease Risk* Relative to Normal Weight and Waist Circumference	
	BMI (kg/m²)	Obesity Class	Men ≤102 cm (≤40 in) Women ≤88 cm (≤35 in)	>102 cm (>40 in) >88 cm (>35 in)
Underweight	<18.5		—	—
Normal+	18.5 – 24.9		—	—
Overweight	25.0 – 29.9		Increased	High
Obesity	30.0 – 34.9	I	High	Very High
	35.0 – 39.9	II	Very High	Very High
Extreme Obesity	≥40	III	Extremely High	Extremely High

* Disease risk for type 2 diabetes, hypertension, and CVD.
\+ Increased waist circumference can also be a marker for increased risk even in persons of normal weight.

Source (adapted from): Preventing and Managing the Global Epidemic of Obesity. Report of the World Health Organization Consultation of Obesity. WHO, Geneva, June 1997.[65]

visited on 6/14/2019

of absolute risk based on the presence of associated disease or risk factors.

2. Identification of patients at very high absolute risk.
The following disease conditions or target organ damage in hypertensive patients denote the presence of very high absolute risk that triggers the need for intense risk factor modification as well as disease management. For example, the presence of very high absolute risk indicates the need for aggressive cholesterol-lowering therapy. [142]

2.a. Established coronary heart disease (CHD)

2.a.1. History of myocardial infarction

2.a.2. History of angina pectoris (stable or unstable)

2.a.3. History of coronary artery surgery

2.a.4. History of coronary artery procedures (angioplasty)

2.b. Presence of other atherosclerotic diseases

2.b.1. Peripheral arterial disease

2.b.2. Abdominal aortic aneurysm

2.b.3. Symptomatic carotid artery disease

2.c. Type 2 diabetes

2.d. Sleep apnea (For more information, see Appendix IV.)

3. Identification of other obesity-associated diseases.
Obese patients are at increased risk for several conditions that require detection and appropriate management, but that generally do not lead to widespread or life-threatening consequences. These include:

3.a. Gynecological abnormalities

3.b. Osteoarthritis

3.c. Gallstones and their complications

3.d. Stress incontinence

Management options of risk factors for preventing CVD, diabetes mellitus, and other chronic diseases are described in detail in other reports. For details on the management of serum cholesterol and other lipoprotein disorders, refer to the National Cholesterol Education Program's Second Report of the Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II/ATP II) *(1993).* [142] *For the treatment of hypertension, the National High Blood Pressure Education Program recently issued the* Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) *(1997).* [545] *For the most recent recommendations about type 2 diabetes from the American Diabetes Association, see the ADA Clinical Practice Recommendations (1998).* [546] *Finally, smoking cessation is strongly recommended in obese smokers, with particular attention to methods that diminish the weight gain associated with cessation (see pages 91-92). For strategies and recommendations for supporting and delivering effective smoking cessation intervention, see the Agency for Health Care Policy and Research's* Clinical Practice Guidelines on Smoking Cessation. [547]

visited on 6/14/2019

Although obese patients are at increased risk for gallstones, this risk increases even more during periods of rapid weight reduction.

4. Identification of cardiovascular risk factors that impart a high absolute risk.
Patients can be classified as being at high absolute risk for obesity-related disorders if they have three or more of the multiple risk factors listed below. The presence of high absolute risk increases the intensity of cholesterol-lowering therapy [142] and blood pressure management. [545]

4.a. Cigarette smoking

4.b. Hypertension—A patient is classified as having hypertension if systolic blood pressure is ≥ 140 mm Hg or diastolic blood pressure is ≥ 90 mm Hg, or if the patient is taking antihypertensive agents.

4.c. High-risk low-density lipoprotein cholesterol—A high-risk LDL-cholesterol is defined as a serum concentration of ≥160 mg/dL. A borderline high-risk LDL-cholesterol (130 to 159 mg/dL) together with two or more other risk factors also confers high risk.

4.d. Low high-density lipoprotein cholesterol—A low HDL-cholesterol is defined as a serum concentration of < 35 mg/dL.

4.e. Impaired fasting glucose (IFG)—The presence of clinical type 2 diabetes (fasting plasma glucose of ≥ 126 mg/dL or 2 hours postprandial plasma glucose of ≥ 200 mg/dL) is a major risk factor for CVD, and its presence alone places a patient in the category of very high absolute risk (see above). IFG (fasting plasma glucose 110 to 125 mg/dL) is considered by many authorities to be an independent risk factor for cardiovascular (macrovascular) disease, justifying its inclusion among risk factors contributing to high absolute risk. Although including IFG as a separate risk factor for CVD departs from the ATP II and JNC VI reports, its inclusion in this list may be appropriate. IFG is well established as a risk factor for type 2 diabetes.

4.f. Family history of premature CHD—A positive family history of premature CHD is defined as definite myocardial infarction or sudden death at or before 55 years of age in the father or other male first-degree relative, or at or before 65 years of age in the mother or other female first-degree relative.

4.g. Age

4.g.1. Male ≥ 45 years

4.g.2. Female ≥ 55 years (or postmenopausal)

Methods for estimating absolute risk status for developing CVD based on these risk factors are described in detail in the ATP II and JNC VI reports. The intensity of intervention for high blood cholesterol or hypertension is adjusted depending on the absolute risk estimated by these factors. Approaches to therapy for cholesterol disorders and hypertension are described in the ATP II and JNC VI, respectively.

5. Other risk factors deserve special consideration for their relation to obesity. When these factors are present, patients can be considered to have incremental absolute risk above that estimated from the preceding risk factors. Quantitative risk contributions are not available for these risk factors, but their presence heightens the need for weight reduction in obese persons.

5.a. Physical inactivity—A lack of physical activity imparts an increased risk for both CVD and type 2 diabetes. Physical inactivity enhances the severity of other risk factors, but it also has been shown to be an "independent" risk factor for all-cause mortality or CVD mortality. [411, 548] Although physical inactivity is not listed as a risk factor that modifies the intensity of therapy required for elevated cholesterol or blood pressure, increased physical activity is indicated for management of these conditions (see the ATP II and JNC VI). Increased physical activity is espe-

visited on 6/14/2019

cially needed in obese patients because it promotes weight reduction and favorably modifies obesity-associated risk factors. Conversely, the presence of physical inactivity in an obese person warrants intensified efforts to remove excess body weight, because physical inactivity and obesity both heighten disease risks.

5.b. High triglycerides—Obesity is commonly accompanied by elevated serum triglycerides. The relationship between high triglycerides and CHD is complex. Triglyceride-rich lipoproteins may be directly atherogenic. In addition, elevated serum triglycerides are the most common manifestation of the atherogenic lipoprotein phenotype (high triglycerides, small LDL particles, and low HDL-cholesterol levels). [142, 549] Moreover, in the presence of obesity, high serum triglycerides are commonly associated with a clustering of metabolic risk factors known as the metabolic syndrome (atherogenic lipoprotein phenotype, hypertension, insulin resistance and glucose intolerance, and prothrombotic states). Thus, in obese patients, elevated serum triglycerides are a marker for increased cardiovascular risk. According to current guidelines (ATP II and JNC VI), the presence of high triglycerides does not modify the intensity of cholesterol or blood pressure-lowering therapy. Their presence in obese patients, however, calls for an intensified effort to achieve weight reduction and increase physical activity. Both will reduce the various risk factors characteristic of the metabolic syndrome, and thus should reduce overall cardiovascular risk as well as decrease the risk for type 2 diabetes.

According to the ATP II guidelines, [142] triglyceride levels are classified as follows:

Category	Serum Triglyceride Levels
Normal triglycerides	Less than 200 mg/dL
Borderline-high triglycerides	200 to 400 mg/dL
High triglycerides	400 to 1,000 mg/dL
Very high triglycerides	Greater than 1,000 mg/dL

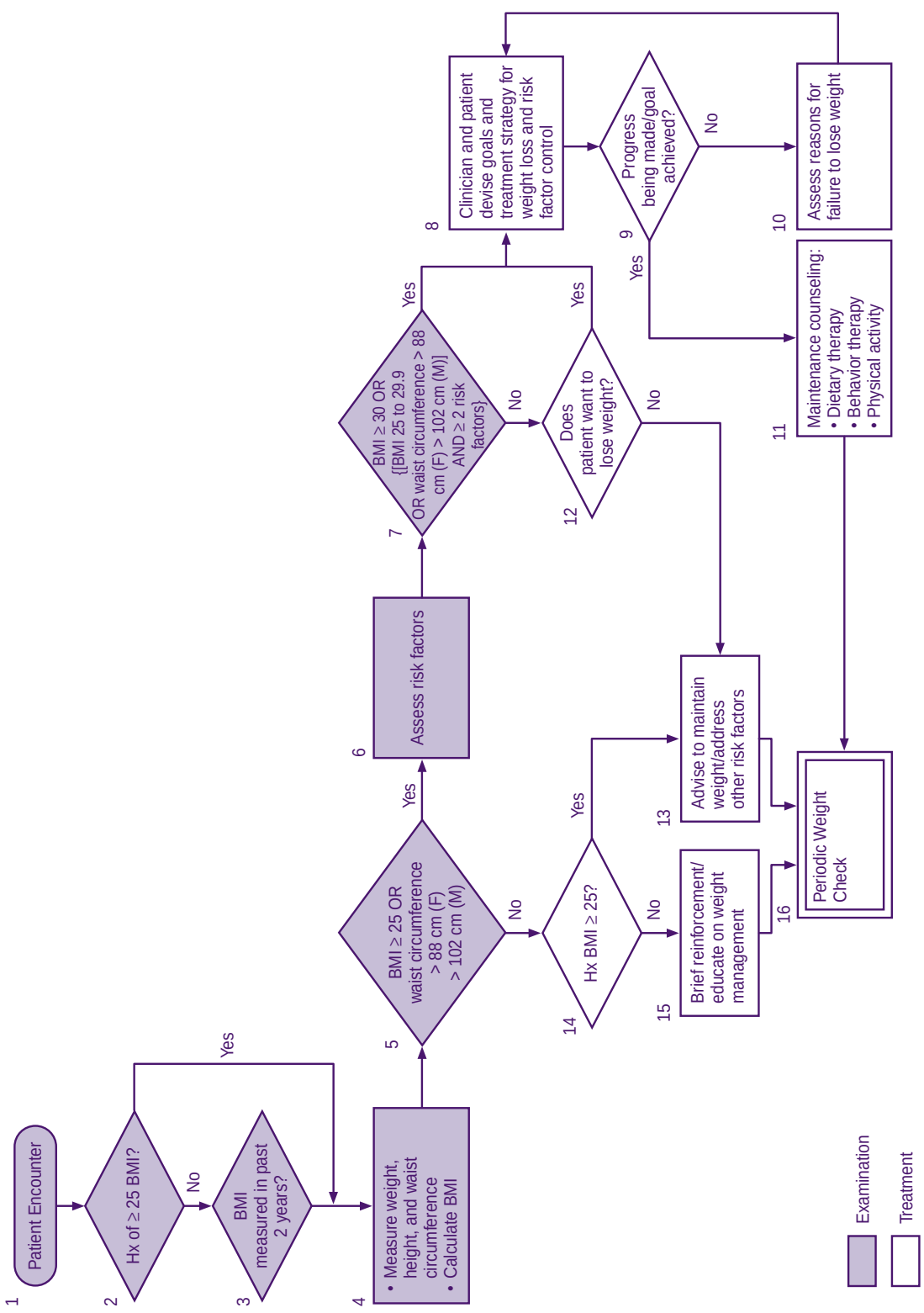
Patients with very high triglycerides are at increased risk for acute pancreatitis and must undergo immediate triglyceride-lowering therapy.

D Evaluation and Treatment Strategy

When physicians encounter patients in the clinical setting, the opportunity exists for identifying overweight and obesity and accompanying risk factors and for initiating treatment for both the weight and the risk factors, as well as chronic diseases such as CVD and type 2 diabetes. When assessing a patient for treatment of overweight and obesity, consider the patient's weight, waist circumference, and the presence of risk factors. The strategy for the evaluation and treatment of overweight patients is presented in Figure 6 (Treatment Algorithm). This algorithm applies only to the assessment for overweight and obesity and subsequent decisions based on that assessment. It does not reflect any initial overall testing for other conditions and diseases that the physician may wish to do. Approaches to therapy for cholesterol disorders and hypertension are described in ATP II and JNC VI, respectively. In overweight patients, control of cardiovascular risk factors deserves equal emphasis as weight loss therapy. Reductions of risk factors will reduce the risk for cardiovascular disease whether or not efforts at weight loss are successful. The panel recognizes that the assessment for overweight and obesity may take place as part of an overall health assessment; however, for clarity, the algorithm focuses only on the evaluation and treatment of overweight and obesity.

visited on 6/14/2019

Figure 6. Treatment Algorithm*



* This algorithm applies only to the assessment for overweight and obesity and subsequent decisions based on that assessment. It does not include any initial overall assessment for cardiovascular risk factors or diseases that are indicated.

visited on 6/14/2019

Each step (designated by a box) in this process is reviewed in this section and expanded upon in subsequent sections.

Box 1: "Patient Encounter"

A patient encounter is defined as any interaction between a health care practitioner (generally a physician, nurse practitioner or physician's assistant) that provides the opportunity to assess a patient's weight status and provide advice, counseling, or treatment.

Box 2: "History of Overweight or Recorded BMI ≥ 25"

The practitioner must seek to determine whether the patient has ever been overweight. While a technical definition is provided, a simple question such as "Have you ever been overweight?" will accomplish the same goal. Questions directed towards weight history, dietary habits, physical activities, and medications may provide useful information about the origins of obesity in particular patients.

Box 3: "BMI Measured in Past 2 Years"

For those who have not been overweight, a 2-year interval is appropriate for the reassessment of BMI. While this time span is not evidence-based, it is believed to be a reasonable compromise between the need to identify weight gain at an early stage and the need to limit the time, effort, and cost of repeated measurements.

Box 4: "Measure Weight, Height, Waist Circumference; Calculate BMI"

Weight must be measured so that the BMI can be calculated. Most charts are based on weights obtained with the patient wearing undergarments and no shoes. BMI can be manually calculated (kg/[height in meters]^2), but is more easily obtained from a nomogram (see Appendix V). Waist circumference is important because evidence suggests that abdominal fat is a particularly strong determinant of cardiovascular risk in those with a BMI of 25 to 34.9 kg/m^2. Increased waist circumference can also be a marker of increased risk even in persons of normal weight. The technique for measuring waist circumference is described on page 58. A nutrition assessment will also help to assess the diet and physical activity habits of overweight patients.

Box 5: "BMI ≥ 25, OR Waist Circumference > 88 cm(F) or > 102 cm(M)"

These cutpoints divide overweight from normal weight and are consistent with other national and international guidelines. The relation between weight and mortality is J-shaped, and evidence suggests that the right side of the "J" begins to rise at a BMI of 25. Waist circumference is incorporated as an "or" factor because some patients with BMI lower than 25 will have disproportionate abdominal fat, and this increases their cardiovascular risk despite their low BMI (see page 61). These abdominal circumference values are not necessary for patients with a BMI ≥ 35 kg/m^2.

Box 6: "Assess Risk Factors"

Risk assessment for CVD and diabetes in a person with evident obesity will include special considerations for the history, physical examination, and laboratory examination. Of greatest urgency is the need to detect existing CVD or end-organ damage. Since the major risk of obesity is indirect (obesity elicits or aggravates hypertension, dyslipidemias, and diabetes, which cause cardiovascular complications), the management of obesity should be implemented in the context of these other risk factors. While there is no direct evidence demonstrating that addressing risk factors increases weight loss, treating the risk factors through weight loss is a recommended strategy. The risk factors that should be considered are provided on pages 63-65. With regard to age, please see commentary on pages 90-91.

visited on 6/14/2019

Box 7: "BMI ≥ 30, OR ([BMI 25 to 29.9 OR Waist Circumference > 88 cm(F) or > 102 cm(M)] AND ≥ 2 risk factors)"

The panel recommends that all patients meeting these criteria attempt to lose weight. However, it is important to ask the patient whether or not they want to lose weight. Those with BMIs between 25 and 29.9 kg/m^2 who have one or no risk factors should work on maintaining their current weight rather than embark on a weight reduction program. The panel recognizes that the decision to lose weight must be made in the context of other risk factors (e.g., quitting smoking is more important than losing weight) and patient preferences.

Box 8: "Clinician and Patient Devise Goals"

The decision to lose weight must be made jointly between the clinician and patient. Patient involvement and investment is crucial to success. The patient may choose not to lose weight but rather to prevent further weight gain as a goal. The panel recommends as an initial goal the loss of 10 percent of baseline weight, to be lost at a rate of 1 to 2 lb/week, establishing an energy deficit of 500 to 1,000 kcal/day. (See pages 71-72.) For individuals who are overweight, a deficit or 300 to 500 kcal/day may be more appropriate, providing a weight loss of about 1/2 lb/week. Also, there is evidence that an average of 8 percent of weight can be lost in a 6-month period. Since the observed average 8 percent weight loss includes people who do not lose weight, an individual goal of 10 percent is reasonable. After 6 months, most patients will equilibrate (caloric intake balancing energy expenditure) and will require adjustment of energy balance if they are to lose more weight. (See pages 72-73.)

The three major components of weight loss therapy are dietary therapy, increased physical activity, and behavior therapy. (See pages 73-82.) Lifestyle therapy should be tried for at least six months before considering pharmacotherapy. In addition, pharmacotherapy should be considered as an adjunct to lifestyle therapy in patients with a BMI ≥ 30 with no concomitant obesity-related risk factors or diseases, or for patients with a BMI ≥ 27 with concomitant obesity-related risk factors or diseases. The risk factors or diseases considered important enough to warrant pharmacotherapy at a BMI of 27 to 29.9 are hypertension, dyslipidemia, CHD, type 2 diabetes, and sleep apnea. However, sibutramine, the only FDA approved drug for long-term use, should not be used in patients with a history of hypertension, CHD, congestive heart failure, arrhythmias, or history of stroke. Certain patients may be candidates for weight loss surgery. Each component of weight loss therapy can be introduced briefly. The selection of weight loss methods should be made in the context of patient preferences, analysis of past failed attempts, and consideration of the available resources.

Box 9: "Progress Being Made/Goal Achieved"

During the acute weight loss period and at 6-month and 1-year follow-up visits, the patients should be weighed, BMI calculated, and progress assessed. If at any time it appears that the program is failing, a reassessment should take place to determine the reasons (see Box 10). If pharmacotherapy is being used, appropriate monitoring for side effects is recommended (See page 86). If a patient can achieve the recommended 10 percent reduction in body weight in 6 months to 1 year, this change in weight can be considered good progress. The patient can then enter the phase of weight maintenance and long-term monitoring. It is important for the practitioner to recognize that some persons are more apt to lose or gain weight on a given regimen and that this phenomenon cannot always be attributed to degree of compliance. However, if significant obesity remains and absolute risk

visited on 6/14/2019

from obesity-associated risk factors remains high, at some point an effort should be made to reinstitute weight loss therapy to achieve further weight reduction. Once a limit of weight loss has been obtained, the practitioner is responsible for long-term monitoring of risk factors and for encouraging the patient to maintain a reduced weight level.

Box 10: "Assess Reasons for Failure to Lose Weight"

If a patient fails to achieve the recommended 10 percent reduction in body weight in 6 months or 1 year, a reevaluation is required. A critical question is whether the level of motivation is high enough to continue clinical therapy. If motivation is high, revise the goals and strategies (see Box 8). If motivation is not high, clinical therapy should be discontinued, but the patient should be encouraged to embark on efforts to lose weight or to at least avoid further weight gain. Even if weight loss therapy is stopped, risk factor management must be continued.

Failure to achieve weight loss should prompt the practitioner to investigate energy intake (dietary recall including alcohol intake, daily intake logs), energy expenditure (physical activity diary), attendance at behavior therapy group meetings, recent negative life events, family and societal pressures, or evidence of detrimental psychiatric problems (depression, binge eating disorder). If attempts to lose weight have failed, and the BMI is ≥ 40, surgical therapy should be considered.

Box 11: "Maintenance Counseling"

Evidence suggests that over 80 percent of persons who lose weight will gradually regain it (see page 72). Patients who continue on weight maintenance programs have a greater chance of keeping weight off. Maintenance consists of continued contact with the health care practitioner for continued education, support, and medical monitoring (see page 72 and 75).

Box 12: "Does the Patient Want to Lose Weight?"

All patients who are overweight (BMI 25 to 29.9), or do not have a high waist circumference, and have few (0 to 1) cardiovascular risk factors and do not want to lose weight, should be counseled regarding the need to keep their weight at or below its present level. Patients who wish to lose weight should be guided per Boxes 8 and 9. The justification for offering these overweight patients the option of maintaining (rather than losing) weight is that their health risk, while higher than that of persons with a BMI < 25, is only moderately increased (page 62).

Box 13: "Advise to Maintain Weight/Address Other Risk Factors"

Those who have a history of overweight and are now at appropriate weight, and those who are overweight and not obese but wish to focus on maintenance of their current weight, should be provided with counseling and advice so that their weight does not increase. An increase in weight increases their health risk and should be prevented. The physician should actively promote prevention strategies including enhanced attention by the patient to diet, physical activity, and behavior therapy. For addressing other risk factors, see Box 6, because even if weight loss cannot be addressed, other risk factors should be covered.

Box 14: "History of BMI ≥ 25"

This box differentiates those who are not overweight now and never have been from those with a history of overweight; see Box 2.

Box 15: "Brief Reinforcement"

Those who are not overweight and never have been should be advised of the importance of staying in this category.

Box 16: "Periodic Weight, BMI, and Waist Circumference Check"

Patients should receive periodic monitoring of their weight, BMI, and waist circumference.

visited on 6/14/2019

Patients who are not overweight or have no history of overweight should be screened for weight gain every 2 years. This time span is a reasonable compromise between the need to identify weight gain at an early stage and the need to limit the time, effort, and the cost of repeated measurements.

E Exclusion from Weight Loss Therapy

Patients in whom weight loss therapy is not appropriate include most pregnant or lactating women, those with serious psychiatric illness, and patients who have a variety of serious illnesses in whom caloric restriction might exacerbate the illness.

F Patient Motivation

Assessment of patient motivation is a prerequisite for weight loss therapy. Weight reduction in the clinical setting represents a major investment of time and effort on the part of the health care team and expense to the patient. For these reasons, motivation for weight loss should be relatively high before initiating clinical therapy. At the same time, it is the duty of the primary care practitioner to heighten a patient's motivation for weight loss when such is perceived to be of significant benefit for risk reduction. This can be done by enumerating the dangers associated with persistent obesity and by describing the strategy for clinically assisted weight reduction. For patients who are not motivated (or able) to enter clinical weight loss therapy, appropriate management of risk factors (e.g., high serum cholesterol, hypertension, smoking, and type 2 diabetes) is still necessary. Sustained weight reduction may facilitate control of cardiovascular risk factors and delay the onset of type 2 diabetes.

Evidence Statement: *Patient motivation is a key component for success in a weight loss program. Evidence Category D.*

Rationale: Assessment of the patient's motivation for treatment by the practitioner is important in the decision to initiate a weight loss program. The following factors must be evaluated:

- Reasons and motivation for weight loss—What is the extent of the patient's seriousness and readiness to undergo a sustained period of weight loss at this time? What is the patient's current attitude about making a lifelong commitment to behavior change?
- Previous history of successful and unsuccessful weight loss attempts—What factors were responsible for previous failures and successes at weight loss or maintenance of normal body weight?
- Family, friends, and work-site support—What is the social framework in which the patient will attempt to lose weight, and who are the possible helpers and antagonists to such an attempt?
- The patient's understanding of overweight and obesity and how it contributes to obesity-associated diseases—Does the patient have an appreciation of the dangers of obesity, and are these dangers of significant concern to the patient?
- Attitude toward physical activity—Is the patient motivated to enter a program of increased physical activity to assist in weight reduction?
- Time availability—Is the patient willing to commit the time required to interact with health professionals in long-term weight loss therapy?
- Barriers—What are the obstacles that will interfere with the patient's ability to implement the suggestions for change?
- Financial considerations—Is the patient willing to pay for obesity therapy? This may include having to pay for travel to the med-

visited on 6/14/2019

ical facility, time lost from work, and paying for professional counseling that is not covered by insurance.

One of the most important aspects of the initial evaluation is to prepare patients for treatment. Reviewing patients' past attempts at weight loss and explaining how the new treatment plan will be different can encourage patients and provide hope for successful weight loss. It is helpful to discuss the proposed course of treatment and describe necessary behaviors, such as keeping diaries of food intake and physical activity.

Finally, given the social stigmatization that obese patients often feel, even from health care professionals, the initial evaluation is an opportunity to show the patient respect, concern, and hope.[550] A patient who has shared feelings about being overweight and previous attempts to lose weight with a sympathetic listener may be more willing to consider new ideas and information. A partnership in which the patient feels supported and understood can help to sustain the necessary motivation for the difficult task of weight loss and maintenance. [551,552]

RECOMMENDATION: *Practitioners need to assess the patient's motivation to enter weight loss therapy; assess the readiness of the patient to implement the plan; and then take appropriate steps to motivate the patient for treatment. Evidence Category D.*

G Goals of Weight Loss and Management

The general goals of weight loss and management are: (1) to reduce body weight; and (2) to maintain a lower body weight over the long term; or (3) at a minimum, to prevent further weight gain. Specific targets for each of these goals can be considered.

1. Weight Loss

1.a. Target levels for weight loss—The initial target goal of weight loss therapy for overweight patients is to decrease body weight by about 10 percent. If this target can be achieved, consideration can be given to the next step of further weight loss.

Evidence Statement: *Overweight and obese patients in well-designed programs can achieve a weight loss of as much as 10 percent of baseline weight, a weight loss that can be maintained for a sustained period of time (1 year or longer). Evidence Category A.*

Rationale: The rationale for this initial goal is that even moderate weight loss, i.e., 10 percent of initial body weight, can significantly decrease the severity of obesity-associated risk factors. It can also set the stage for further weight loss, if indicated. Available evidence indicates that an average weight loss of 8 percent can be achieved in 6 months; however, since the observed average 8 percent includes people who do not lose weight, an individual goal of 10 percent is reasonable. This degree of weight loss can be achieved and is realistic, and moderate weight loss can be maintained over time. It is better to maintain a moderate weight loss over a prolonged period than to regain from a marked weight loss. The latter is counterproductive in terms of time, costs, and self-esteem. Patients generally will wish to lose more weight than 10 percent, and will need to be counseled and persuaded of the appropriateness of this initial goal. [553, 554] Further weight loss can be considered after this initial goal is achieved and maintained for 6 months.

RECOMMENDATION: *The initial goal of weight loss therapy should be to reduce body weight by approximately 10 percent*

visited on 6/14/2019

from baseline. With success, further weight loss can be attempted, if indicated through further assessment. Evidence Category A.

1.b. Rate of weight loss—A reasonable time line for weight loss is to achieve a 10 percent reduction in body weight over 6 months of therapy. For overweight patients with BMIs in the typical range of 27 to 35, a decrease of 300 to 500 kcal/day will result in weight losses of about ½ to 1 lb/week and a 10 percent weight loss in 6 months. For more severely obese patients with BMIs ≥ 35, deficits of up to 500 to 1,000 kcal/day will lead to weight losses of about 1 to 2 lb/week and a 10 percent weight loss in 6 months.

Evidence Statement: *Weight loss at the rate of 1 to 2 lb/week (calorie deficit of 500 to 1,000 kcal/day) commonly occurs for up to 6 months, at which point weight loss begins to plateau unless a more restrictive regimen is implemented. Evidence Category B.*

Rationale: To achieve significant weight loss, an energy deficit must be created and maintained. Weight can be lost at a rate of 1 to 2 lb/week with a calorie deficit of 500 to 1,000 kcal/day. After 6 months, this caloric deficit theoretically should result in a loss of 26 to 52 lb. However, the average amount of weight lost actually observed over this time period usually is in the range of 20 to 25 lb. A greater rate of weight loss does not yield a better result at the end of 1 year. [437] It is difficult for most patients to continue to lose weight after a period of 6 months due to changes in resting metabolic rates and difficulty in adhering to treatment strategies, although some can do so. To continue to lose weight, diet and physical activity goals need to be revised to create an energy deficit at the lower weight, since energy requirements decrease as weight is decreased. To achieve additional weight loss, the patient must further decrease calories and/or increase physical activity. Many studies show that rapid weight reduction almost always is followed by regaining of weight. Moreover, with rapid weight reduction, there is an increased risk for gallstones and, possibly, electrolyte abnormalities.

RECOMMENDATION: *Weight loss should be about 1 to 2 lb/week for a period of 6 months with the subsequent strategy based on the amount of weight lost. Evidence Category B.*

2. Weight Maintenance at Lower Weight

Once the goals of weight loss have been successfully achieved, maintenance of a lower body weight becomes a major challenge. In the past, obtaining the goal of weight loss has been considered the end of weight loss therapy. Unfortunately, once patients are dismissed from clinical therapy, they frequently regain the lost weight. This report recommends that observation, monitoring, and encouragement of patients who have successfully lost weight be continued on a long-term basis.

Evidence Statement: *After 6 months of weight loss treatment, efforts to maintain weight loss through diet, physical activity, and behavior therapy are important. Evidence Category B.*

Rationale: After 6 months of weight loss, the rate of weight loss usually declines and plateaus. [395, 507, 555] The primary care practitioner and patient should recognize that at this point, weight

visited on 6/14/2019

maintenance, the second phase of the weight loss effort, should take priority. Successful weight maintenance is defined as a weight regain of < 3 kg (6.6 lb) in 2 years and a sustained reduction in waist circumference of at least 4 cm. If a patient wishes to lose more weight after a period of weight maintenance, the procedure for weight loss outlined above can be repeated.

RECOMMENDATION: *A weight maintenance program should be a priority after the initial 6 months of weight loss therapy. Evidence Category B.*

Evidence Statement: *Lost weight usually will be regained unless a weight maintenance program consisting of dietary therapy, physical activity, and behavior therapy is continued indefinitely. Drug therapy can also be used; however, drug safety and efficacy beyond 1 year of total treatment have not been established. Evidence Category B.*

Rationale: After a patient has achieved the goals of weight loss, the combined modalities of therapy (dietary therapy, physical activity, and behavior therapy) must be continued indefinitely; otherwise, excess weight likely will be regained. Numerous strategies are available for motivating the patient; all of these require that the practitioner continue to communicate frequently with the patient. Long-term monitoring and encouragement can be accomplished in several ways: by regular clinic visits, at group meetings, or via telephone or E-mail. The longer the weight maintenance phase can be sustained, the better the prospects for long-term success in weight reduction. Drug therapy may also be helpful during the weight maintenance phase.

RECOMMENDATION: *After successful weight loss, the likelihood of weight loss maintenance is enhanced by a program consisting of dietary therapy, physical activity, and behavior therapy, which should be continued indefinitely. Drug therapy can also be used. However, drug safety and efficacy beyond 1 year of total treatment have not been established. Evidence Category B.*

3. Prevention of Further Weight Gain

Some patients may not be able to achieve significant weight reduction. In such patients, an important goal is to prevent further weight gain that would exacerbate disease risk. Thus, prevention of further weight gain may justify entering a patient into weight loss therapy. Prevention of further weight gain can be considered a partial therapeutic success for many patients. Moreover, if further weight gain can be prevented, this achievement may be an important first step toward beginning the weight loss process. Primary care practitioners ought to recognize the importance of this goal for those patients who are not able to immediately lose weight. The need to prevent weight gain may warrant maintaining patients in a weight management program for an extended period.

H Strategies for Weight Loss and Weight Maintenance

1. Dietary Therapy

In the majority of overweight and obese patients, adjustment of the diet to reduce caloric intake will be required. Dietary therapy consists, in large part, of instructing patients on how to modify their diets to achieve a decrease in caloric intake. A key element of the current recommendation is the use of a moderate reduction in caloric intake to achieve a slow but progressive weight loss. Ideally, caloric intake should be

visited on 6/14/2019

reduced only to the level required to maintain weight at the desired level. If this level of caloric intake is achieved, excess weight will gradually disappear. In practice, somewhat greater caloric deficits are used in the period of active weight loss, but diets with very low calories are to be avoided. Finally the composition of the diet should be modified to minimize other cardiovascular risk factors. [142]

The centerpiece of dietary therapy for weight loss in overweight patients is a low-calorie diet (LCD) (800 to 1,500 kcal/day). This diet is to be distinguished from a very low-calorie diet (VLCD) (250 to 800 kcal/day), which has been unsuccessful in achieving weight loss over the long term. The LCD recommended in this report also contains nutrient compositions that will decrease other risk factors, notably, high serum cholesterol and hypertension.

Evidence Statement: *LCDs can reduce total body weight by an average of 8 percent and help reduce abdominal fat content over a period of approximately 6 months. Evidence Category A.*

Rationale: A decrease in calorie intake is the most important dietary component of weight loss and maintenance. LCDs have been shown to reduce total body weight by an average of 8 percent over a period of 6 months, accompanied by significant reductions in waist circumference. Since this represents an average that includes individuals who did not lose weight, an individual average goal of 10 percent is feasible. When weight loss occurs, the loss consists of about 75 percent fat and 25 percent lean tissue. [556, 557] A deficit of 500 to 1,000 kcal/day will produce a weight loss of 70 to 140 grams/day, or 490 to 980 grams/week (1 to 2 lb/week). A deficit of 300 to 500 kcal/day will produce a weight loss of 40 to 70 grams/day, or 280 to 490 grams/week (1/2 to 1 lb/week) A patient may choose a diet of 1,000 to 1,200 kcal for women and 1,200 to 1,500 kcal for men.

VLCDs (less than 800 kcal/day) are not recommended for weight loss therapy because the deficits are too great, and nutritional inadequacies will occur unless VLCDs are supplemented with vitamins and minerals. [558] Moreover, clinical trials show that LCDs are just as effective as VLCDs in producing weight loss after 1 year. [437] Although more weight is initially lost with VLCDs, more is usually regained. Further, rapid weight reduction does not allow for gradual acquisition of changes in eating behavior. Successful behavior therapy is the key to long-term maintenance of weight at a reduced level. Finally, patients using VLCDs are at increased risk for developing gallstones.

Successful weight reduction by LCDs is more likely to occur when consideration is given to a patient's food preferences in tailoring a particular diet. Care should be taken to be sure that all of the recommended dietary allowances are met; this may require use of a dietary supplement. Dietary education is a necessary ingredient in achieving adjustment to an LCD. Educational efforts should pay particular attention to the following topics:

- energy value of different foods;
- food composition—fats, carbohydrates (including dietary fiber), and proteins;
- reading nutrition labels to determine caloric content and food composition;
- new habits of purchasing—preference to low-calorie foods;
- food preparation—avoiding adding high-calorie ingredients during cooking (e.g., fats and oils);

visited on 6/14/2019

- avoiding overconsumption of high-calorie foods (both high-fat and high-carbohydrate foods);
- maintain adequate water intake
- reducing portion sizes; and
- limiting alcohol consumption.

The Step I Diet in ATP II provides an appropriate nutrient composition for an LCD diet. The composition of the diet is presented in Table IV-3. Additional practical dietary advice for patients is included in Appendix VI.

RECOMMENDATION: *A diet that is individually planned to help create a deficit of 500 to 1,000 kcal/day should be an integral part of any program aimed at achieving a weight loss of 1 to 2 lb/week. Evidence Category A.*

RECOMMENDATION: *Reducing dietary fat alone without reducing calories is not sufficient for weight loss. However, reducing dietary fat, along with reducing dietary carbohydrates, can facilitate caloric reduction. Evidence Category A.*

Evidence Statement: *Optimally, dietary therapy should last at least 6 months. Evidence Category A.*

Rationale: Many studies suggest that the rate of weight loss diminishes after about 6 months. Shorter periods of dietary therapy usually result in lesser weight reductions. Therapeutic efforts should be directed toward behavior therapy as well as maintaining LCDs. [486, 570, 571]

Evidence Statement: *During dietary therapy, frequent contacts between professional counselors and patients promote weight loss and maintenance. Evidence Category C.*

Rationale: Frequent clinical encounters during the initial 6 months of weight reduction appear to facilitate reaching the goals of therapy. During the period of active weight loss, regular visits of at least once per month and preferably more often with a health professional for the purposes of reinforcement, encouragement, and monitoring will facilitate weight reduction. Weekly group meetings can be conducted at a low cost, and can contribute to favorable behavior changes. However, no clinical trials have been specifically designed to test the relative efficacy of different frequencies of encounters with physicians, dietitians, or others in the weight loss team. [487]

Evidence Statement: *The amount of time spent with the patient favorably affects weight change in overweight or obese adults given dietary therapy. Evidence Category D.*

Rationale: Training of a health professional in techniques of weight reduction, especially in behavior therapy and dietary principles, is expected to facilitate weight reduction. Further, adequate time must be made available to the patient to convey the information necessary, to reinforce behavioral and dietary messages, and to monitor the patient's response. Despite these judgments, none of the studies reviewed were designed to specifically address the type or qualifications of the health professional who implemented the various weight loss approaches. Many of the studies differed in the types of

visited on 6/14/2019

TABLE IV-3:

Low-Calorie Step I Diet

Nutrient	Recommended Intake
Calories[1]	Approximately 500 to 1,000 kcal/day reduction from usual intake
Total Fat[2]	30 percent or less of total calories
Saturated Fatty Acids[3]	8 to 10 percent of total calories
Monounsaturated Fatty Acids	Up to 15 percent of total calories
Polyunsaturated Fatty Acids	Up to 10 percent of total calories
Cholesterol[3]	< 300 mg/day
Protein[4]	Approximately 15 percent of total calories
Carbohydrate[5]	55 percent or more of total calories
Sodium Chloride	No more than 100 mmol per day (approximately 2.4 g of sodium or approximately 6 g of sodium chloride)
Calcium[6]	1,000 to 1,500 mg
Fiber[5]	20 to 30 g

1. A reduction in calories of 500 to1,000 kcal/day will help achieve a weight loss of 1 to 2 lbs/week. Alcohol provides unneeded calories and displaces more nutritious foods. Alcohol consumption not only increases the number of calories in a diet but has been associated with obesity in epidemiological studies [559-562] as well as in experimental studies. [563-566] The impact of alcohol calories on a person's overall caloric intake needs to be assessed and appropriately controlled.

2. Fat-modified foods may provide a helpful strategy for lowering total fat intake but will only be effective if they are also low in calories and if there is no compensation of calories from other foods.

3. Patients with high blood cholesterol levels may need to use the Step II diet to achieve further reductions in LDL-cholesterol levels; in the Step II diet, saturated fats are reduced to less than 7 percent of total calories, and cholesterol levels to less than 200 mg/day. All of the other nutrients are the same as in Step I.

4. Protein should be derived from plant sources and lean sources of animal protein.

5. Complex carbohydrates from different vegetables, fruits, and whole grains are good sources of vitamins, minerals, and fiber. A diet rich in soluble fiber, including oat bran, legumes, barley, and most fruits and vegetables may be effective in reducing blood cholesterol levels. A diet high in all types of fiber may also aid in weight management by promoting satiety at lower levels of calorie and fat intake. Some authorities recommend 20 to 30 grams of fiber daily, with an upper limit of 35 grams. [546, 567, 568]

6. During weight loss, attention should be given to maintaining an adequate intake of vitamins and minerals. Maintenance of the recommended calcium intakes of 1,000 to 1,500 mg/day is especially important for women who may be at risk of osteoporosis. [569]

visited on 6/14/2019

dietary intervention provided. Most programs involved dietitians and nutritionists as primary therapists and used group therapy rather than individual sessions. The length of time spent during each session and the nature of the practitioner/patient interaction tended not to be provided.

RECOMMENDATION: *The literature suggests that weight loss and weight maintenance therapies that provide a greater frequency of contacts between the patient and the practitioner and are provided over the long term should be put in place. This can lead to more successful weight loss and weight maintenance. Evidence Category C.*

2. Physical Activity

An increase in physical activity is an important component of weight loss therapy since it leads to increased expenditure of energy. Increased physical activity may also inhibit food intake in overweight patients. Physical activity can also be helpful in maintaining a desirable weight. In addition, sustained physical activity has the benefit of reducing overall CHD risk beyond that produced by weight reduction alone.

Several experts believe that a progressive decrease in the amount of energy expended for work, transportation, and personal chores is a major cause of obesity in the United States. These authorities note that total caloric intake has not increased over the last few decades; instead, the caloric imbalance leading to overweight and obesity is the result of a substantial decrease in physical activity and, consequently, a decrease in daily energy expenditure. However, this hypothesis is difficult to prove because appropriate data are lacking. Successful restoration of normal weight in many overweight and obese persons requires a higher level of energy expenditure. Increased regular physical activity is the way to achieve this goal of augmenting daily energy expenditure.

Evidence Statement: *Physical activity contributes to weight loss, both alone and when it is combined with dietary therapy. Evidence Category A.*

Rationale: Increased physical activity alone can create a caloric deficit and can contribute to weight loss. However, efforts to achieve weight loss through physical activity alone generally produce an average of a 2 to 3 percent decrease in body weight or BMI. Even so, increased physical activity is a useful adjunct to LCDs in promoting weight reduction.

Evidence Statement: *Physical activity in overweight and obese adults increases cardiorespiratory fitness independent of weight loss. Evidence Category A.*

Rationale: There is strong evidence that increased physical activity increases cardiorespiratory fitness, with or without weight loss. [346, 363, 369, 375, 401, 404, 406, 432, 434, 445, 447, 448] Improved cardiovascular fitness also improves the quality of life in overweight patients by improving mood, self-esteem, and physical function in daily activities. [572]

Evidence Statement: *Physical activity independently reduces CVD risk factors (Evidence Category A), and reduces risk for cardiovascular disease. Evidence Category C.*

---

visited on 6/14/2019

Rationale: There is considerable evidence that physical inactivity is an independent risk factor for CVD and diabetes. [572] Furthermore, the more active an individual is, the lower the risk. By the same token, increased physical activity appears to independently reduce risk for CVD morbidity and mortality, and diabetes. [411, 548, 573, 574] Physical activity reduces elevated levels of CVD risk factors, including blood pressure and triglycerides, increases HDL-cholesterol, and improves glucose tolerance with or without weight loss. [572]

Evidence Statement: *Physical activity contributes to a decrease in body fat, including a modest effect on abdominal fat. Evidence Category B.*

Rationale: Physical activity appears to have a favorable effect on distribution of body fat. [572] Several large cross-sectional studies in Europe, [464] Canada, [575] and the United States [466-468] showed an inverse association between energy expenditure through physical activity and several indicators of body fat distribution, such as WHR and waist-to-thigh circumference ratio. Fewer data are available on the effects of physical activity on waist circumference, although the ratio changes noted above suggest a decrease in abdominal obesity. Only a few randomized controlled trials that have tested the effect of physical activity on weight loss measured waist circumference. In some (but not all) studies, physical activity was found to produce only modest weight loss and decreased waist circumference. [365, 369, 375] However, it is not known whether the effects of physical activity on abdominal fat are independent of weight loss.

2.a. Strategies to increase physical activity

Many people live sedentary lives, have little training or skills in physical activity, and are difficult to motivate toward increasing their activity. For these reasons, starting a physical activity regimen may require supervision for some people. The need to avoid injury during physical activity is high. Extremely obese persons may need to start with simple exercises that can gradually be intensified. The practitioner must decide whether exercise testing for cardiopulmonary disease is needed before embarking on a new physical activity regimen. This decision should be based on a patient's age, symptoms, and concomitant risk factors.

For most obese patients, physical activity should be initiated slowly, and the intensity should be increased gradually. Initial activities may be walking or swimming at a slow pace. With time, depending on progress, the amount of weight lost, and functional capacity, the patient may engage in more strenuous activities. Some of these include fitness walking, cycling, rowing, cross-country skiing, aerobic dancing, and rope jumping. Jogging provides a high-intensity aerobic exercise, but can lead to orthopedic injury. If jogging is desired, the patient's ability to do this must first be assessed. The availability of a safe environment for the jogger is also a necessity. Competitive sports, such as tennis and volleyball, can provide an enjoyable form of physical activity for many, but again, care must be taken to avoid injury, especially in older people. As the examples listed in Table IV-4 show, a moderate amount of physical activity can be achieved in a variety of ways. People can select activities that they enjoy and that fit into their daily lives. Because amounts of activity are functions of duration, intensity, and frequency, the same amounts of activity can be obtained in longer sessions of moderately intense activities (such as brisk walking) as in shorter sessions of more strenuous activities (such as running).

A regimen of daily walking is an attractive form of physical activity for many people, particularly those who are overweight or obese. The patient

visited on 6/14/2019

TABLE IV-4:

Examples of Moderate Amounts of Activity*

Washing and waxing a car for 45-60 minutes	**Less Vigorous, More Time ****
Washing windows or floors for 45-60 minutes	
Playing volleyball for 45 minutes	
Playing touch football for 30-45 minutes	
Gardening for 30-45 minutes	
Wheeling self in wheelchair for 30-40 minutes	
Walking 1¾ miles in 35 minutes (20 min/mile)	
Basketball (shooting baskets) for 30 minutes	
Bicycling 5 miles in 30 minutes	
Dancing fast (social) for 30 minutes	
Pushing a stroller 1½ miles in 30 minutes	
Raking leaves for 30 minutes	
Walking 2 miles in 30 minutes (15 min/mile)	
Water aerobics for 30 minutes	
Swimming laps for 20 minutes	
Wheelchair basketball for 20 minutes	
Basketball (playing a game) for 15-20 minutes	
Bicycling 4 miles in 15 minutes	
Jumping rope for 15 minutes	
Running 1½ miles in 15 minutes (10 min/mile)	**More Vigorous, Less Time**
Shoveling snow for 15 minutes	
Stairwalking for 15 minutes	

* A moderate amount of physical activity is roughly equivalent to physical activity that uses approximately 150 calories of energy per day, or 1,000 calories per week.

** Some activities can be performed at various intensities; the suggested durations correspond to expected intensity of effort.

can start by walking 10 minutes, 3 days a week, and can build to 30 to 45 minutes of more intense walking at least 5 days a week and preferably most, if not all, days. [577, 578] With this regimen, an additional 100 to 200 calories per day of physical activity can be expended. Caloric expenditure will vary depending on the individual's body weight and intensity of the activity (see Table IV-5).

This regimen can be adapted to other forms of physical activity, but walking is particularly attractive because of its safety and accessibility.

visited on 6/14/2019

With time, a larger weekly volume of physical activity can be performed that would normally cause a greater weight loss if it were not compensated by a higher caloric intake.

Reducing sedentary time is another approach to increasing activity. Patients should be encouraged to build physical activities into each day. Examples include leaving public transportation one stop before the usual one, parking further than usual from work or shopping, and walking up stairs instead of taking elevators or escalators. New forms of physical activity should be suggested, e.g., gardening, walking a dog daily, or new athletic activities. Engaging in physical activity can be facilitated by identifying a safe area to perform the activity, e.g., community parks, gyms, pools, and health clubs. However, when these sites are not available, an area of the home can be identified and perhaps outfitted with equipment such as a stationary bicycle or a treadmill.

Health professionals should encourage patients to plan and schedule physical activity 1 week in advance, budget the time necessary to do it, and document their physical activity by keeping a diary and recording the duration and intensity of exercise.

RECOMMENDATION: *Physical activity should be an integral part of weight loss therapy and weight maintenance. Evidence Category A. Initially, moderate levels of physical activity for 30 to 45 minutes, 3 to 5 days per week should be encouraged. All adults should set a long-term goal to accu-*

TABLE IV-5:

Duration of Various Activities to Expend 150 Kilocalories for an Average 70 kg (154 lb) Adult

Intensity	Activity	Approximate duration in minutes
Moderate	Volleyball, noncompetitive	43
Moderate	Walking, moderate pace (3mph, 20 min/mile)	37
Moderate	Walking, brisk pace (4mph, 15 min/mile)	32
Moderate	Table tennis	32
Moderate	Raking leaves	32
Moderate	Social dancing	29
Moderate	Lawn mowing (powered push mower)	29
Hard	Jogging (5 mph, 12 min/mile)	18
Hard	Field hockey	16
Very Hard	Running (6 mph, 10 min/mile)	13

Source: Surgeon General's Report on Physical Activity and Health

visited on 6/14/2019

mulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. Evidence Category B.

3. Behavior Therapy

Behavioral strategies to reinforce changes in diet and physical activity can produce a weight loss in obese adults in the range of 10 percent of baseline weight over 4 months to 1 year. Unless a patient acquires a new set of eating and physical activity habits, long-term weight reduction is unlikely to succeed. The acquisition of new habits is particularly important for long-term weight maintenance at a lower weight. Most patients return to baseline weights in the absence of continued intervention. Thus, the physician or staff members must become familiar with techniques for modifying life habits of overweight or obese patients.

The goal of behavior therapy is to alter the eating and activity habits of an obese patient. Techniques for behavior therapy have been developed to assist patients in modifying their life habits.

Evidence Statement: *Behavior therapy, in combination with an energy deficit, provides additional benefits in assisting patients to lose weight short-term (1 year). Its effectiveness for long-term weight maintenance has not been shown in the absence of continued behavioral intervention. Evidence Category B.*

Rationale: The primary assumptions of behavior therapy are that:

- by changing eating and physical activity habits, it is possible to change body weight;
- patterns of eating and physical activity are learned behaviors and can be modified; and
- to change these patterns over the long term, the environment must be changed.

Behavior therapies provide methods for overcoming barriers to compliance with dietary therapy and/or increased physical activity, and are thus important components of weight loss therapy. Most weight loss programs incorporating behavioral strategies do so as a package that includes education about nutrition and physical activity. However, this standard "package" of management should not ignore the need for individualizing behavioral strategies. [579]

3.a. Behavior Therapy Strategies Used in Weight Loss and Weight Maintenance Programs

Studies reviewed for this report examined a range of modalities of behavioral therapy. No single method or combination of behavioral methods proved to be clearly superior. Thus, various strategies can be used by the practitioner to modify patient behavior. The aim is to change eating and physical activities behaviors over the long term. Such change can be achieved either on an individual basis or in group settings. Group therapy has the advantage of lower cost. Specific behavioral strategies include the following:

- Self-monitoring of both eating habits and physical activity—Objectifying one's own behavior through observation and recording is a key step in behavior therapy. Patients should be taught to record the amount and types of food they eat, the caloric values, and nutrient composition. Keeping a record of the frequency, intensity, and type of physical activity likewise will add insight to personal behavior. Extending records to time, place, and feelings related to eating and physical activity will help to bring previously unrecognized behavior to light. [580]

visited on 6/14/2019

- Stress management—Stress can trigger dysfunctional eating patterns, and stress management can defuse situations leading to overeating. Coping strategies, meditation, and relaxation techniques all have been successfully employed to reduce stress.
- Stimulus control—Identifying stimuli that may encourage incidental eating enables individuals to limit their exposure to high-risk situations. Examples of stimulus control strategies include learning to shop carefully for healthy foods, keeping high-calorie foods out of the house, limiting the times and places of eating, and consciously avoiding situations in which overeating occurs. [580]
- Problem solving—This term refers to the self-correction of problem areas related to eating and physical activity. Approaches to problem solving include identifying weight-related problems, generating or brainstorming possible solutions and choosing one, planning and implementing the healthier alternative, and evaluating the outcome of possible changes in behavior. [580] Patients should be encouraged to reevaluate setbacks in behavior and to ask "What did I learn from this attempt?" rather than punishing themselves.
- Contingency management—Behavior can be changed by use of rewards for specific actions, such as increasing time spent walking or reducing consumption of specific foods. [44] Verbal as well as tangible rewards can be useful, particularly for adults. Rewards can come from either the professional team or from the patients themselves. For example, self-rewards can be monetary or social and should be encouraged.
- Cognitive restructuring—Unrealistic goals and inaccurate beliefs about weight loss and body image need to be modified to help change self-defeating thoughts and feelings that undermine weight loss efforts. Rational responses designed to replace negative thoughts are encouraged. [580] For example, the thought, "I blew my diet this morning by eating that doughnut; I may as well eat what I like for the rest of the day," could be replaced by a more adaptive thought, such as, "Well, I ate the doughnut this morning, but I can still eat in a healthy manner at lunch and dinner."
- Social support—A strong system of social support can facilitate weight reduction. Family members, friends, or colleagues can assist an individual in maintaining motivation and providing positive reinforcement. Some patients may benefit by entering a weight reduction support group. Overweight patients should be asked about (possibly) overweight children and family weight control strategies. Parents and children should work together to engage in and maintain healthy dietary and physical activity habits.

3.b. Treatment of Obese Individuals with Binge Eating Disorder

If a patient suffers from binge eating disorder (BED), consideration can be given to referring the patient to a health professional who specializes in BED treatment. Behavioral approaches to BED associated with obesity have been derived from cognitive behavior therapy (CBT) used to treat bulimia nervosa. [227] Among the techniques are self-monitoring of eating patterns, encouraging regular patterns of eating (three meals a day plus planned snacks), cognitive restructuring, and relapse prevention strategies. [581]

RECOMMENDATION: *Behavior therapy strategies to promote diet and physical activity should be used routinely, as they are helpful in achieving weight loss and weight maintenance. Evidence Category B.*

visited on 6/14/2019

4. Combined Therapy

To achieve the greatest likelihood of success from weight loss therapy, the combination of dietary therapy with an LCD, increased physical activity, and behavior therapy will be required. Inclusion of behavior therapy and increased physical activity in a weight loss regimen will provide the best opportunity for weight loss, and hopefully for long-term weight control. In order to achieve weight loss, such a regimen should be maintained for at least 6 months before considering pharmacotherapy.

Evidence Statement: *Combined intervention of an LCD, increased physical activity, and behavior therapy provides the most successful therapy for weight loss and weight maintenance. Evidence Category A.*

Rationale: Clinical trials have demonstrated that combining behavior therapy, LCDs, and increased physical activity provides better outcomes for long-term weight reduction than programs that use only one or two of these modalities. A lower-fat diet markedly improves the potential of physical activity to achieve a negative energy balance. [317, 369, 434, 444, 582] In addition, lower-fat diets that are also low in saturated fats reduce serum cholesterol levels, which would reduce CVD risk. It is difficult to achieve a negative energy balance and weight loss with physical activity of moderate duration and intensity in individuals who consume a high-fat diet and alcohol. [583, 584]

RECOMMENDATION: *Weight loss and weight maintenance therapy should employ the combination of LCDs, increased physical activity, and behavior therapy. Evidence Category A.*

5. Pharmacotherapy

Drug therapy has undergone radical changes in the last 2 years. With the publication of the trials with phentermine and fenfluramine by Weintraub in 1992 (210 weeks), drug therapy began to change from short-term to long-term use. Both dexfenfluramine and fenfluramine alone, as well as the combination of phentermine/fenfluramine, were used long term. However, concerns about recently reported unacceptable side effects, such as valvular lesions of the heart causing significant insufficiency of the valves, [658] have led to the withdrawal of the drugs dexfenfluramine and fenfluramine from the market in September 1997. [761] No drugs remained that were approved by the Food and Drug Administration (FDA) for use longer than 3 months. In November 1997, the FDA approved a new drug, sibutramine, for use in obesity and is in the process of evaluating orlistat for long-term use.

Evidence Statement: *Appropriate weight loss drugs can augment diet, physical activity, and behavior therapy in weight loss. Evidence Category B.*

Rationale: The purpose of weight loss and weight maintenance is to reduce health risks. If weight is regained, health risks increase once more. The majority of persons who lose weight regain it, [585] so the challenge to the patient and the practitioner is to maintain the weight loss. Because of the tendency to regain weight after weight loss, the use of long-term medication to aid in the treatment of obesity may be indicated in carefully selected patients.

The drugs used to promote weight loss have been anorexiants or appetite suppressants. Three classes of anorexiant drugs have been developed, all of which affect neurotransmitters in the

visited on 6/14/2019

brain: those that affect catecholamines, those that affect serotonin, and those that affect both. They work by increasing the secretion of dopamine, norepinephrine, or serotonin into the synaptic neural cleft, or by inhibiting the reuptake of these neurotransmitters back into the neuron, or by both mechanisms. The new agent sibutramine has norepinephrine and serotonin effects. Another new agent, orlistat, has a different mechanism of action, the blockage of fat absorption. Very few trials longer than 6 months have been done with any drug. The ones tested for at least 1 year that received FDA approval for long-term use are shown in Table IV-6.

These drugs are effective but modest in their ability to produce weight loss. Net weight loss attributable to drugs generally has been reported to be in the range of 2 to 10 kg (4.4 to 22 lb), although some patients lose significantly more weight. It is not possible to predict how much weight an individual may lose. Most of the weight loss usually occurs in the first 6 months of therapy.

Adverse effects include primary pulmonary hypertension with fenfluramine and dexfenfluramine, [395, 586, 587] valvular heart disease with dexfenfluramine and fenfluramine, [658] and increases in blood pressure and pulse with sibutramine. [510] With orlistat, there is a possible decrease in the absorption of fat-soluble vitamins; overcoming this may require vitamin supplementation. People with a history of high blood pressure, CHD, congestive heart failure, arrhythmias, or history of stroke should not take sibutramine, and all patients taking the medication should have their blood pressure monitored on a regular basis. Depression has been described with the serotonergic drugs but it is generally not clinically significant. Neurotoxic effects with neuronal atrophy have been described with high doses of dexfenfluramine in rats and primates, but not in humans. [588] The risks for using

TABLE IV-6:

WEIGHT LOSS DRUGS+

Drug	Action	Adverse Effects
dexfenfluramine* fenfluramine*	serotonin reuptake inhibitor serotonin releaser	valvular heart disease primary pulmonary hypertension neurotoxicity
sibutramine	norepinephrine, dopamine, and serotonin reuptake inhibitor	increase in heart rate and blood pressure
orlistat±	inhibits pancreatic lipase, decreases fat absorption	decrease in absorption of fat-soluble vitamins soft stools and anal leakage possible link to breast cancer

\+ Ephedrine and caffeine, and, fluoxetine have also been tested for weight loss, but are not approved for use in the treatment of obesity. Mazindol, phentermine, benzphetamine, and phendimetrazine are approved for only short-term use for the treatment of obesity.

* FDA approval withdrawn

± FDA approval pending

visited on 6/14/2019

appetite suppressant drugs during pregnancy is unknown.

Evidence Statement: *Weight loss drugs approved by the FDA for long-term use may be useful as an adjunct to diet and physical activity for patients with a BMI of ≥ 30 with no concomitant obesity-related risk factors or diseases, and for patients with a BMI of ≥ 27 with concomitant obesity-related risk factors or diseases. Evidence Category B.*

Rationale: If after at least 6 months on a weight loss regimen of an LCD, increased physical activity, and behavior therapy, the patient has not lost the recommended 1 lb/week, careful consideration may be given to pharmacotherapy. There are few long-term studies evaluating the safety or effectiveness of many currently approved weight loss medications. At present, sibutramine is available for long-term use. (Note: FDA approval for orlistat is pending a resolution of labeling issues and results of Phase III trials.) Their risk/benefit ratio is such that they can be recommended for use with the degree of obesity outlined above. [589] Weight loss medications should be used only by patients who are at increased medical risk because of their weight and should not be used for "cosmetic" weight loss. The risk factors and diseases considered serious enough to warrant pharmacotherapy at BMI of 27 to 29.9 are hypertension, dyslipidemia, CHD, type 2 diabetes, and sleep apnea.

Not every patient responds to drug therapy. Tests with weight loss drugs have shown that initial responders tend to continue to respond, while initial nonresponders are less likely to respond even with an increase in dosage. [395, 507] If a patient does not lose 2 kg (4.4 lb) in the first 4 weeks after initiating therapy, the likelihood of long-term response is very low. [507] This finding may be used as a guide to treatment, either continuing medication in the responders or stopping it in the nonresponders. If weight is lost in the initial 6 months of therapy or is maintained after the initial weight loss phase, this should be considered a success, and the drug may be continued. It is important to remember that the major role of medications should be to help patients stay on a diet and physical activity plan while losing weight. Medication cannot be expected to continue to be effective in weight loss or weight maintenance once it has been stopped. [590, 591] The use of the drug may be continued as long as it is effective and the adverse effects are manageable and not serious. There are no indications for specifying how long a weight loss drug should be continued. Therefore, an initial trial period of several weeks with a given drug or combination of drugs may help determine their efficacy in a given patient. If a patient does not respond to a drug with reasonable weight loss, the physician should reassess the patient to determine adherence to the medication regimen and adjunctive therapies, or consider the need for dosage adjustment. If the patient continues to be unresponsive to the medication, or serious adverse effects occur, the physician should consider its discontinuation. [592]

Evidence Statement: *Adverse side effects from the use of weight loss drugs have been observed in patients. Evidence Category A.*

Rationale: The potential for side effects from the use of weight loss drugs is of great concern. Adverse effects with sibutramine include increased blood pressure and tachycardia. [590] With orlistat there are oily and loose stools and some fat-soluble vitamin malabsorption. [389] Thus, a multivitamin supplement is recom-

visited on 6/14/2019

mended. Side effects are generally mild and may improve with continued use, although their persistence may result in discontinuation of the drug.

There is a great interest in weight loss drugs among consumers. Because of the possibility of serious adverse effects, it is incumbent on the practitioner to use drug therapy with caution. Practitioners should be sure that patients are not taking drugs that have been withdrawn from the market for safety reasons, and herbal medications are not recommended as part of a weight loss program. These preparations have unpredictable amounts of active ingredients and unpredictable and potentially harmful effects. *In those patients with a lower level of obesity risk, nonpharmacological therapy is the treatment of choice.* It is important for the physician to monitor both the effectiveness and the side effects of the drug.

Evidence Statement: *Using weight loss drugs singly (not in combination) and starting with the lowest effective doses can decrease the likelihood of adverse effects. Evidence Category C.*

Rationale: Given the fact that adverse events may increase in association with combination drug therapy, it seems wise that, until further safety data are available, using weight loss drugs singly would be more prudent. Some patients will respond to lower doses, so that full dosage is not always necessary. The short-term use of drugs (< 3 months) has not generally been found to be effective.

Drugs should be used only as part of a comprehensive program that includes behavior therapy, diet, and physical activity. Appropriate monitoring for side effects must be continued while drugs are part of the regimen. Patients will need to return for followup in 2 to 4 weeks, then monthly for 3 months, and then every 3 months for the first year after starting the medication. After the first year, the doctor will advise the patient on appropriate return visits. The purposes of these visits are to monitor weight, blood pressure, and pulse, discuss side effects, conduct laboratory tests, and answer questions.

Since obesity is a chronic disorder, the short-term use of drugs is not helpful. The health professional should include drugs only in the context of a long-term treatment strategy. [593] The risk/benefit ratio cannot be predicted at this time, since not enough long-term data (> 1 year) are available on any of the available drugs.

RECOMMENDATION: *Weight loss drugs approved by the FDA may only be used as part of a comprehensive weight loss program, including dietary therapy and physical activity, for patients with a BMI of ≥ 30 with no concomitant obesity-related risk factors or diseases, and for patients with a BMI of ≥ 27 with concomitant obesity-related risk factors or diseases. Weight loss drugs should never be used without concomitant lifestyle modifications. Continual assessment of drug therapy for efficacy and safety is necessary. If the drug is efficacious in helping the patient lose and/or maintain weight loss and there are no serious adverse effects, it can be continued. If not, it should be discontinued. Evidence Category B.*

6. Surgery for Weight Loss

Surgery is one option for weight reduction for some patients with severe and resistant obesity. The aim of surgery is to modify the gastrointestinal tract to reduce net food intake. Most authorities agree that weight loss surgery should be reserved for patients with severe obesity, in whom efforts at other therapy have failed, and

visited on 6/14/2019

who are suffering from the complications of obesity.

Considerable progress has been made in developing safer and more effective surgical procedures for promoting weight loss. Surgical interventions commonly used include gastroplasty, gastric partitioning, and gastric bypass. These procedures are designed primarily to reduce food consumption. They have replaced previous procedures that were designed to promote malabsorption of nutrients. The latter procedures were fraught with side effects that made their use impractical or dangerous.

Evidence Statement: *Gastrointestinal surgery (gastric restriction [vertical gastric banding] or gastric bypass [Roux-en Y]) can result in substantial weight loss, and therefore is an available weight loss option for well-informed and motivated patients with a BMI ≥ 40 or ≥ 35, who have comorbid conditions and acceptable operative risks. Evidence Category B.*

Rationale: According to the National Institutes of Health's Consensus Development Conference on Gastrointestinal Surgery for Severe Obesity, [523] the risk for morbidity and mortality accompanying obesity increases with the degree of overweight. Thus, treatment of clinically severe obesity involves an effort to create a caloric deficit sufficient to result in weight loss and reduction of weight-associated risk factors or comorbidities. Surgical approaches can result in substantial weight loss, i.e., from 50 kg (110 lb) to as much as 100 kg (220 lb) over a period of 6 months to 1 year. A major limitation of nonsurgical approaches is the failure to maintain reduced body weight in many individuals.

Surgical procedures in current use (gastric restriction [vertical gastric banding] and gastric bypass [Roux-en Y]) can induce substantial weight loss, and serve to reduce weight-associated risk factors and comorbidities. Compared to other interventions available, surgery has produced the longest period of sustained weight loss. Assessing both perioperative risk and long-term complications is important and requires assessing the risk/benefit ratio in each case. Patients whose BMI equals or exceeds 40 kg/m^2 are potential candidates for surgery if they strongly desire substantial weight loss, because obesity severely impairs the quality of their lives. Less severe obese patients (BMIs between 35 and 39.9 kg/m^2) also may be considered for surgery. This group primarily includes those patients with high-risk comorbid conditions (cardiovascular, sleep apnea, uncontrolled type 2 diabetes) or weight-induced physical problems interfering with performance of daily life activities.

A recent retrospective study of severely overweight patients with noninsulin-dependent diabetes, who were referred for consideration of a gastric bypass procedure, allowed for a comparison of those who opted for the surgical procedure versus those who did not undergo the procedure because of personal preference or refusal of insurance payment. Patients undergoing the surgical procedure had a decrease in mortality rate for each year of follow up. [594] This latter observation provides initial documentation of the significant impact that reduction in weight may have on mortality.

Most of the surgery studies primarily included women of childbearing age. Caution should be exercised in selecting candidates for surgery to treat obesity, as pregnancy demands increase nutritional needs and a normal need for weight gain. Women with reproductive potential should be advised to avoid pregnancy until their weight has stabilized postoperatively and potential micronutrient deficiencies have been identified and treated.

visited on 6/14/2019

Of special note is that many of the studies reported to date have not had population samples representative of the general severely overweight population with respect to race, ethnicity, cultural or socioeconomic background, or gender.

Evidence Statement: *Patients opting for surgical intervention should be followed by a multidisciplinary team (medical, behavioral, and nutritional). Evidence Category D.*

Rationale: As for all other interventions for obesity, an integrated program should be in place that will provide guidance concerning the necessary dietary regimen, appropriate physical activity, and behavioral and social support both prior to and after the surgical procedure.

Evidence Statement: *Lifelong medical surveillance after surgical therapy is a necessity. Evidence Category C.*

Rationale: Since surgical procedures result in some loss of absorptive function, the long-term consequences of potential nutrient deficiencies must be recognized and adequate monitoring must be performed, particularly with regard to vitamin B_{12}, folate, and iron. Some patients may develop other gastrointestinal symptoms such as "dumping syndrome" or gallstones. Occasionally, patients may have postoperative mood changes or their presurgical depression symptoms may not be improved by the achieved weight loss. Thus, surveillance should include monitoring of indices of inadequate nutrition and modification of any preoperative disorders. Table IV-7 illustrates some of the complications that can occur following gastric bypass surgery.

TABLE IV-7:

Gastric Bypass Surgery Complications: 14-Year Follow Up

Vitamin B_{12} deficiency	239	39.9 percent
Readmit for various reasons	229	38.2 percent
Incisional hernia	143	23.9 percent
Depression	142	23.7 percent
Staple line failure	90	15.0 percent
Gastritis	79	13.2 percent
Cholecystitis	68	11.4 percent
Anastomotic problems	59	9.8 percent
Dehydration, malnutrition	35	5.8 percent
Dilated pouch	19	3.2 percent

Data derived from source (Pories WJ[595]) and modified based on personal communication.

visited on 6/14/2019

RECOMMENDATION: *Weight loss surgery is an option for carefully selected patients with* clinically severe *obesity (BMI ≥ 40 or ≥ 35 with comorbid conditions) when less invasive methods of weight loss have failed and the patient is at high risk for obesity-associated morbidity or mortality. Evidence Category B.*

COMMENTARY: Adapting Weight Loss Programs To Meet the Needs of Diverse Patient Populations.

Standard obesity treatment approaches should be tailored to the needs of patients. It is, however, difficult to determine from the literature how often this occurs and whether it makes weight loss programs more effective. Very few published reports of such adapted programs can be identified, particularly when a distinction is made from reports that include or focus on special populations but do not report any particular steps taken to modify the intervention for these populations. In addition, it is impossible to compare directly the amount of weight lost using specially adapted programs with that achieved when more standard approaches are used. Studies reporting these programs are sometimes pilot studies or descriptive reports that do not meet the standards of evidence set forth elsewhere in these guidelines. Where randomized controlled trials or quasi-experiments are available, they usually do not include an internal comparison with a program involving no adaptations. Appendix III illustrates the types of adaptations that have been reported.

Large individual variation exists within any social or cultural group; furthermore, there is substantial overlap among subcultures within the larger society. There is, therefore, no "cookbook" or standardized set of rules to optimize weight reduction with a given type of patient. A theoretical and qualitative analysis of cultural appropriateness in obesity treatment programs has been conducted, and it provides some guidance for incorporating patient characteristics and perspectives when designing and delivering weight loss programs. [257] *Some examples follow:*

- *Adapt the setting and staffing to the patient population. The setting should:*
  - *be physically accessible to the patient;*
  - *have features likely to be familiar to the patient;*
  - *be free of negative psychosocial connotations;*
  - *be devoid of aspects that create a large social distance among patients or between patient and practitioner; and*
  - *promote active patient participation and high patient self-esteem and self-efficacy.*

  The staff should be culturally self-aware and culturally competent in working with persons of diverse cultural backgrounds and income or educational levels. For example, cultural adaptation of programs has been approached with the assumption that community centers may be preferable to hospitals or medical offices as venues for conducting lifestyle weight reduction programs. Or, some programs have included peer educators as a possible way of helping to overcome background and social class differences by providing a bridge of knowledge, experience, and perspective between patients and practitioners (see Appendix III).

  - *List assumptions about the type of patient for whom the program will be best suited and evaluate the extent to which these assumptions are appropriate for prospective patients. Where appropriate, modify the program to avoid the need for certain assumptions. Consider patients'—*
    - *preexisting knowledge base;*
    - *day-to-day routine;*
    - *discretionary time;*

visited on 6/14/2019

- *financial resources and living situation; and*
- *cultural preferences for types of food and activity.*

  For example, redesign printed materials to be suitable for patients with low literacy skills or poor vision. Offer dietary and physical activity recommendations that will be feasible for low-income patients living in inner-city areas with limited access to supermarkets or with high crime rates.

- *Consider how the obesity treatment program fits in other aspects of the health care and self-care of the patient(s), and integrate other aspects where appropriate. For example, for those patients with diabetes, information about weight reduction should be aligned with other diabetes management advice.*
- *Expect and allow for program modifications based on patient feedback and preferences. Program appropriateness can be increased when patients can express their needs and preferences, and the program is then adapted to those needs and preferences. This is especially applicable when practitioners have limited common experience with patients.*

In recent years, a fat acceptance, nondieting advocacy group has developed. This has emerged from concerns about weight cycling and its possible adverse effects on morbidity and mortality. However, recent evidence suggests that intentional weight loss is not associated with increased morbidity and mortality. For this reason, the guidelines have been made explicit on the importance of intervention for weight loss and maintenance in the appropriate patient groups.

COMMENTARY: Weight Reduction After Age 65—What Are the Issues?

- *Are the indications for treating obesity in older adults the same as those for younger adults?*
- *Does weight loss reduce risk factors in older adults?*
- *Does weight reduction prolong the lives of older adults?*
- *Are there risks associated with obesity treatment that are unique to older adults?*

The higher prevalence of cardiovascular risk factors in overweight versus nonoverweight persons is clearly observed at older ages. [596, 597] *In addition, obesity is a major predictor of functional limitations and mobility impairments in older adults.* [596, 598-600] *Both observational data* [596] *and applicable randomized controlled trials cited in these guidelines suggest that weight loss reduces risk factors and improves functional status in older persons in the same manner as in younger adults.*

The question of whether weight reduction leads to increased survival among older adults has been raised because of observations that weight reduction after age 60 or 65 years may be unable to reverse the deleterious effects of longstanding obesity. [288, 596, 601, 602] *Also, weight loss at older ages has been associated with increased mortality.* [318, 603-607] *Some of the association of weight loss with higher mortality may be due to the increased frequency at older ages of involuntary weight loss due to identified or occult illness. In any case, the association of weight loss with higher mortality applies most clearly to individuals who enter old age with a BMI in the lower part of the range. It is not clear that it applies to overweight older persons with CVD risk factors. This issue has been difficult to clarify in observational data, but there are no randomized trials in which the effects of obesity treatment on mortality can be directly assessed, at any age.*

The wisdom or importance of treating obesity at older ages has also been questioned because of epidemiological observations suggesting a decreased significance of obesity-related relative risks at older ages (see chapter 2.3.b.). However, these relative-risk data are not a useful reference point for considering whether obese older persons will benefit.

visited on 6/14/2019

Relative risks are influenced by the characteristics of the comparison group. In this case, the comparison group is lower-weight older persons who have also had high morbidity and mortality.

Concerns about potential adverse effects of obesity treatment in older adults have been raised with respect to bone health and to dietary adequacy. Weight reduction may accelerate aging-related bone loss and thereby increase the risk of osteoporotic fractures in high-risk groups such as older white women. [608-610] *This concern is more relevant to weight loss in thin persons than in obese persons. Some evidence suggests that including resistance training and moderate weight-bearing exercise as a part of a weight reduction program may help maintain bone integrity.* [611, 612]

The general nutritional safety of weight reduction at older ages is of interest because restrictions on overall food intake due to dieting could result in inadequate intakes of protein or essential vitamins and minerals. In addition, involuntary weight loss indicative of occult disease might be mistaken for success in voluntary weight reduction. These concerns can be alleviated by providing proper nutritional counseling and regular body weight monitoring for older persons for whom weight reduction is prescribed.

Evidence Statement: *Age alone should not preclude treatment for obesity in adult men and women. Evidence Category D.*

Rationale: There is little evidence at present to indicate that obesity treatment should be withheld from adult men and women on the basis of age alone up to 80 years of age.

RECOMMENDATION: *A clinical decision to forego obesity treatment in an older adult should be guided by an evaluation of the potential benefits of weight reduction for day-to-day functioning and reduction of the risk of future cardiovascular events, as well as the patient's motivation for weight reduction. Care must be taken to ensure that any weight reduction program minimizes the likelihood of adverse effects on bone health or other aspects of nutritional status. Evidence Category D.*

I Smoking Cessation in the Overweight or Obese Patient

Cigarette smoking is a major risk factor for cardiopulmonary disease. Because of its attendant high risk, smoking cessation is a major goal of risk-factor management. This aim is especially important in the overweight or obese patient, who usually carries excess risk from obesity-associated risk factors. Thus, smoking cessation in such patients becomes a high priority for risk reduction.

Evidence Statement: *Smoking and obesity together increase cardiovascular risk, but fear of weight gain upon smoking cessation is an obstacle for many patients who smoke. Evidence Category C.*

Rationale: Both smoking and obesity are accompanied by increased risks for cardiovascular disease. Many well-documented health benefits are associated with smoking cessation, but a major obstacle to successful smoking cessation has been the attendant weight gain observed in about 80 percent of quitters. This weight gain averages 4.5 to 7 lb, but in 13 percent of women and 10 percent of men, weight gains in excess of 28 lb have been noted among quitters. [613-615] Weight gain is an important barrier to smoking cessation, particularly in women. [616, 617]

visited on 6/14/2019

Weight gain that accompanies smoking cessation so far has been relatively resistant to most dietary, behavioral, or physical activity interventions. [618, 619] Postcessation weight gain has been associated with a reduction in energy expenditure of up to 100 kcal/day, accounting for approximately one-third of the weight gain after smoking cessation. [620, 621] The reduction in energy expenditure appears to be the result of a decrease in the resting metabolic rate. [614] In general, no differences in the level of physical activity have been observed after smoking cessation. About two-thirds of the weight gain after smoking cessation appears to result from increased caloric intake. [621-623] However, dietary counseling programs combined with smoking cessation programs have not been very successful. There are several products that reduce postcessation weight gain during drug administration, including nicotine replacement therapy, [624] phenylpropanolamine, [625] and bupropion. [626] No matter what the drug, however, it appears that these drugs merely delay rather than prevent postcessation weight gain. That is, while providing weight gain suppression during drug administration, subjects on these drugs experience a rebound of weight gain once they go off the products and long-term weight gain is equal to that in those not receiving these drugs. [624, 626]

The weight gained with smoking cessation is less likely to produce negative health consequences than would continued smoking. For this reason, smoking cessation should be strongly reinforced in persons regardless of their baseline weight. Smoking is not an acceptable weight control therapy, although it seems to be used for this purpose by a great many people. Overweight patients, as well as all others, should be counseled to quit smoking. For practical reasons, it may be prudent to avoid initiating smoking cessation and weight loss therapy simutaneously. Prevention of weight gain through diet and physical activity should be stressed. If weight gain ensues after smoking cessation, it should be managed vigorously according to the guidelines outlined in this report. Although short-term weight gain is a common side effect of smoking cessation, this gain does not rule out the possibility of long-term weight control. There are no clinical trials to test whether ex-smokers are less likely to successfully achieve long-term weight reduction than those who never smoked.

RECOMMENDATION: *All smokers, regardless of their weight status, should quit smoking. Evidence Category A. Prevention of weight gain should be encouraged and if weight gain does occur, it should be treated through dietary therapy, physical activity, and behavior therapy, maintaining the primary emphasis on the abstinence from smoking. Evidence Category C.*

J Role of Health Professionals in Weight Loss Therapy

Evidence Statement: *A number of health professionals can play an important role in a weight loss and management program. Evidence Category B.*

Rationale: Various randomized controlled trials were reviewed that highlighted the role of the nutritionists, [413, 476] exercise physiologists and physical education instructors, [401, 447] nurses, [413] and psychologists [476, 487] in addition to the physician in providing assessment, treatment, and follow up during weight loss. As a result, it is suggested that the health professionals avail themselves of the various disciplines that offer expertise in dietary counseling, physical activity, and behavior change. The relationship between the physician and these disciplines can be a direct,

visited on 6/14/2019

formal one or a more indirect referral. It is important to emphasize that a positive attitude of support and encouragement from all professionals is crucial to continuing success. [627]

RECOMMENDATION: *A weight loss and maintenance program can be conducted by a practitioner without specialization in weight loss so long as that person has the requisite interest and knowledge. However, various health professionals with different expertise are available and helpful to a practitioner who would like assistance. Evidence Category B.*

visited on 6/14/2019

visited on 6/14/2019

Summary of Recommendations

A Advantages of Weight Loss

- Weight loss is recommended to lower elevated blood pressure in overweight and obese persons with high blood pressure. Evidence Category A.
- Weight loss is recommended to lower elevated levels of total cholesterol, low-density lipoprotein cholesterol, and triglycerides and to raise low levels of high-density lipoprotein cholesterol in overweight and obese persons with dyslipidemia. Evidence Category A.
- Weight loss is recommended to lower elevated blood glucose levels in overweight and obese persons with type 2 diabetes. Evidence Category A.

B Measurement of Degree of Overweight and Obesity

- Practitioners should use the body mass index (BMI) to assess overweight and obesity. Body weight alone can be used to follow weight loss, and to determine efficacy of therapy. Evidence Category C.
- The BMI should be used to classify overweight and obesity and to estimate relative risk for disease compared to normal weight. Evidence Category C.
- The waist circumference should be used to assess abdominal fat content. Evidence Category C.
- For adult patients with a BMI of 25 to 34.9 kg/m^2, sex-specific waist circumference cutoffs should be used in conjunction with BMI to identify increased disease risks. Evidence Category C.

C Goals for Weight Loss

- The initial goal of weight loss therapy should be to reduce body weight by approximately 10 percent from baseline. With success, further weight loss can be attempted, if indicated, through further assessment. Evidence Category A.
- Weight loss should be about 1 to 2 lb/week for a period of 6 months, with the subsequent strategy based on the amount of weight lost. Evidence Category B.

D How To Achieve Weight Loss

1. Dietary Therapy

- Low-calorie diets are recommended for weight loss in overweight and obese persons. Evidence Category A. Reducing fat as part of a low-calorie diet is a practical way to reduce calories. Evidence Category A.
- Reducing dietary fat alone without reducing calories is not sufficient for weight loss. However, reducing dietary fat, along with reducing dietary carbohydrates, can facilitate caloric reduction. Evidence Category A.
- A diet that is individually planned to help create a deficit of 500 to 1,000 kcal/day should be an integral part of any program aimed at achieving a weight loss of 1 to 2 lb/week. Evidence Category A.

2. Physical Activity

- Physical activity is recommended as part of a comprehensive weight loss therapy and weight maintenance program because it: (1) modestly contributes to weight loss in overweight and

visited on 6/14/2019

obese adults (Evidence Category A), (2) may decrease abdominal fat (Evidence Category B), (3) increases cardiorespiratory fitness (Evidence Category A), and (4) may help with maintenance of weight loss (Evidence Category C).

- Physical activity should be an integral part of weight loss therapy and weight maintenance. Evidence Category A. Initially, moderate levels of physical activity for 30 to 45 minutes, 3 to 5 days per week should be encouraged. All adults should set a long-term goal to accumulate at least 30 minutes or more of moderate-intensity physical activity on most, and preferably all, days of the week. Evidence Category B.
- The combination of a reduced calorie diet and increased physical activity is recommended since it produces weight loss, decreases abdominal fat, and increases cardiorespiratory fitness. Evidence Category A.

3. Behavior Therapy

- Behavior therapy is a useful adjunct when incorporated into treatment for weight loss and weight maintenance. Evidence Category B.
- Practitioners need to assess the patient's motivation to enter weight loss therapy; assess the readiness of the patient to implement the plan and then take appropriate steps to motivate the patient for treatment. Evidence Category D.
- Behavior therapy strategies to promote diet and physical activity should be used routinely, as they are helpful in achieving weight loss and weight maintenance. Evidence Category B.

4. Combined Therapy

- Weight loss and weight maintenance therapy should employ the combination of low-calorie diets, increased physical activity, and behavior therapy. Evidence Category A.

5. Pharmacotherapy

- Weight loss drugs approved by the FDA may be used as part of a comprehensive weight loss program including diet and physical activity for patients with a BMI of ≥ 30 with no concomitant obesity- related risk factors or diseases, and for patients with a BMI of ≥ 27 with concomitant obesity-related risk factors or diseases. Drugs should never be used without concomitant lifestyle modification. Continual assessment of drug therapy for efficacy and safety is necessary. If the drug is efficacious in helping the patient lose and/or maintain weight loss and there are no serious adverse effects, it can be continued. If not, it should be discontinued. Evidence Category B.

6. Weight Loss Surgery

- Weight loss surgery is an option in carefully selected patients with *clinically severe* obesity (BMI ≥ 40 or ≥ 35 with comorbid conditions) when less invasive methods of weight loss have failed and the patient is at high risk for obesity-associated morbidity or mortality. Evidence Category B.

E Goals for Weight Loss Maintenance

- After successful weight loss, the likelihood of weight loss maintenance is enhanced by a program consisting of dietary therapy, physical activity, and behavior therapy, which should be continued indefinitely. Drug therapy can also be used. However, drug safety and efficacy beyond 1 year of total treatment have not been established. Evidence Category B.
- A weight maintenance program should be a priority after the initial 6 months of weight loss therapy. Evidence Category B.

visited on 6/14/2019

F How To Maintain Weight Loss

- The literature suggests that weight loss and weight maintenance therapies that provide a greater frequency of contacts between the patient and the practitioner and are provided over the long term should be put in place. This can lead to more successful weight loss and weight maintenance. Evidence Category C.
- A weight loss and maintenance program can be conducted by a practitioner without specialization in weight loss so long as that person has the requisite interest and knowledge. However, various health professionals with different expertise are available and helpful to a practitioner who would like assistance. Evidence Category B.

G Special Treatment Groups

- All smokers, regardless of their weight status, should quit smoking. Evidence Category A. Prevention of weight gain should be encouraged and if weight gain does occur, it should be treated through diet, physical activity, and behavior therapy, maintaining the primary emphasis on the abstinence from smoking. Evidence Category C.
- A clinical decision to forego obesity treatment in older adults should be guided by an evaluation of the potential benefits of weight reduction for day-to-day functioning and reduction of the risk of future cardiovascular events, as well as the patient's motivation for weight reduction. Care must be taken to ensure that any weight reduction program minimizes the likelihood of adverse effects on bone health or other aspects of nutritional status. Evidence Category D.
- The possibility that a standard approach to weight loss will work differently in diverse patient populations must be considered when setting expectations about treatment outcomes. Evidence Category B.

visited on 6/14/2019

6

Future Research

Obesity is a heterogeneous chronic disorder that has many causes, although the fundamental basis is an imbalance between energy intake and energy expenditure. Future research needs to examine the most effective ways to treat and prevent obesity, the causes of obesity and their mechanisms, the influence of fat distribution on health risk, and the development of better methods for assessing energy intake and energy expenditure.

A Intervention Approaches

Considerable research is needed on intervention approaches to treat and prevent obesity. Increased research on behavioral theory specifically addressing obesity treatment and prevention for all individuals, including children and adolescents, needs to be conducted. Intervention methods to prevent weight gain with smoking cessation are of particularly high priority in helping achieve smoking cessation.

More research is needed on behavioral intervention methods conducted in various settings, particularly the primary care setting. Effective programs to treat or prevent obesity in culturally, ethnically, and socioeconomically diverse populations need to be developed and tested. Simple screening tools should be tested for their predictive value in achieving lifestyle modifications that lead to weight loss or weight control practices. Research is needed on identifying appropriate and successful intervention content; for example, magnitude of weight loss goals (smaller changes versus larger changes), and goals for the rate of weight loss (1 lb versus 2 lb per week; initial weight loss goal of 5 percent of body weight and, subsequently, an additional 5 percent versus a single initial goal of 10 percent at the outset). Of particular importance is research on the optimal amount of physical activity to promote weight loss, the maintenance of weight loss, and the prevention of obesity. Also important are strategies which preserve muscle and bone in the face of weight loss. More research is needed on identifying the characteristics of individuals who have successfully maintained their weight loss over the long term.

Research on surgical interventions for weight loss should include evaluating surgical risk, including not only complications, morbidity, and mortality, but also long-term postoperative surveillance to monitor vitamin and mineral nutritional adequacy. Evaluation of the health benefits of weight loss from surgery should include changes in fat distribution; cardiorespiratory fitness; obesity-related comorbidities, including blood pressure, blood lipids, and glucose tolerance; and degree of success in long-term weight loss maintenance. Finally, research is needed on techniques for integrating behavioral methods to promote long-term maintenance of weight loss after surgical treatment. Likewise, research on pharmacologic interventions for weight loss should include evaluating changes in fat distribution, cardiorespiratory fitness, obesity-related comorbidities, and the degree of success of long-term weight loss maintenance. Better methods for integrating behavioral methods, along with pharmacologic treatment, should also be investigated.

visited on 6/14/2019

Finally, research is needed on environmental and population-based intervention methods, including community- and school-based interventions, to augment public health approaches toward promoting weight maintenance and preventing obesity in the general population.

B Causes and Mechanisms of Overweight and Obesity

The regulation of energy balance needs to be explored, including the neuroendocrine factors that control energy intake, energy expenditure, and the differentiation of adipose tissue resulting from excess calories. The genes that are important in human obesity need to be identified. These include those that alter eating and physical activity behaviors, those that affect thermogenesis, and those associated with the comorbidities of obesity. The roles of environmental and behavioral influences on metabolic factors important in obesity, as well as gene-environment interactions, need to be studied. Predictive factors should be examined to identify who is most at risk of developing obesity, and whether there are critical periods of life when these factors are most operative. In addition, the influence of the intrauterine environment on the development of obesity needs to be investigated, particularly to determine whether early deprivation leads to a later propensity for overweight and associated comorbidities, such as insulin resistance, or if high maternal weight gain and high birth weight are related to the risk of obesity and its comorbidities.

C Abdominal Fat, Body Weight and Disease Risk

The influence of abdominal fat independent of total body fat on health risk needs to be further defined. More information is needed on the relationship between differential body fat compartments and increased risk, the distribution of body fat compartments among various racial group populations, and the relationship between abdominal fat and disease risk in racial groups. Weight loss studies should include measurements of abdominal fat, as well as cardiorespiratory fitness, to better assess health improvement. Intentional weight loss treatments need to be examined in terms of their acute and chronic effect on the development and progression of diabetes, heart disease, and overall mortality. Large prospective studies are needed to examine the relationship of body mass index and body fat distribution to overall mortality.

D Assessment Methods

Much of the current research is hampered by the lack of good methods to accurately, objectively, and economically assess energy intake and expenditure, including physical activity, body composition and fat distribution, and behavioral and psychological variables. More research is therefore needed to focus on measures to assess intake of fat and other dietary components, levels of physical activity, energy metabolism, and body fat and visceral obesity. In addition, better methods for assessment of psychological, behavioral, and psychosocial variables that may be related to behavioral risk factors for obesity (such as poor diet and inactive lifestyle) are needed, and particularly so for special population segments based on race, ethnicity, and socioeconomic status. Methods for assessing culture, social integration, and psychological stress should also be developed.

visited on 6/14/2019

Appendices

1 Appendix I.A.1. Guidelines Development Methodology

The panel has attempted to provide primary care practitioners with recommendations regarding effective strategies to evaluate and treat overweight and obesity in adults. The panel anticipates that the recommendations also will be followed by the large proprietary weight loss industry in the United States. The guideline is based on a systematic review of the scientific literature published in MEDLINE from January 1980 through September 1997. This was done in the interest of time and economy. This information was supplemented by material provided by the panel and a search of appropriate references in eligible articles. The panel identified randomized controlled trials as the strongest level of evidence for the evaluation of treatment efficacy. Thus, evidence from randomized controlled trials (RCTs) serves as the basis for many of the recommendations contained in this guideline. However, in some instances, the panel had to make recommendations in the absence of RCTs. Each evidence statement (other than those with no available evidence) and each recommendation is categorized by level of evidence (A through D) as described on page 108. Statements for which there is no available evidence are so indicated.

1.a. Panel Selection

The National Heart, Lung, and Blood Institute (NHLBI) Obesity Education Initiative Task Force recommended panel members representing the various expertise required to deal with the issues of cardiovascular disease and body weight. The executive director of the American Academy of Family Physicians (AAFP) also provided recommendations for the primary care practitioner who would represent AAFP. The panel members include primary care practitioners, clinicians from academic medical centers, nutritionists, exercise physiologists, pulmonologists, cardiologists, psychologists, behaviorists, epidemiologists, and experts on cost issues. Some of the panel members were also members of the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) National Task Force on the Prevention and Treatment of Obesity. The funding source for the guidelines was primarily the NHLBI, with the cooperation of the NIDDK of the National Institutes of Health.

1.b. Topics Included in the Guidelines

The panel decided to focus on *all* adult (18 years of age and older) overweight and obese patients with a body mass index (BMI) ≥ 25, and particularly those with cardiovascular risk factors. Excluded from the analysis were patients with known genetic or hormonal syndromes and pharmacologically induced obesity.

In addition, since pediatric obesity contributes to about one-third of adult obesity, the panel felt that some attention needed to be focused on the issue of overweight in children and adolescents (see Appendix III). Treatment issues surrounding overweight children and adolescents are quite different from the treatment of adults. The panel recommended that this issue warrants its own project as soon as possible.

visited on 6/14/2019

The therapeutic interventions requiring examination included diet, physical activity, behavior therapy, pharmacological therapy, surgery, and combinations of these. No clinical interventions were excluded at the outset. Because of the importance and complexity of the primary prevention of obesity, the panel decided not to deal with these concerns in detail and that the issue be addressed in a separate document. However, clinical interventions to prevent further weight gain in patients already overweight, or those patients not currently overweight at high risk for becoming overweight, were considered relevant therapeutic interventions. The guideline also contains some information on the cost of obesity.

In terms of reliable diagnostic measures, the panel decided to concentrate on useful tools readily available in a physician's office, i.e., weight, height and BMI. The panel did not search for evidence to prove that measures of body composition or metabolic rate are useful tools; however, they did include studies using these diagnostic measures.

1.c. Development of the Evidence Model

The panel determined the evidence model used to develop the guidelines for the evaluation and treatment of obesity (Figure 7, and Figures 7a and 7b). The model contains two broad categories, the association of weight to cardiovascular and noncardiovascular mortality and morbidity in the population and the clinical issues related to the treatment of the overweight individual. The population portion of the model addresses such questions as, "Is there an association between weight and cardiovascular risk factors?", and "Is there an association between weight and cardiovascular and noncardiovascular morbidity and mortality?" The patient portion of the model focuses on the clinical setting and addresses such questions as, "Does a reduction in weight result in a decrease in cardiovascular morbidity and mortality?"

The model illustrates the areas requiring a thorough examination of the relevant scientific literature through data abstraction and evaluation (the hollow shaded arrows), the areas where the literature has been firmly established (the hollow arrows), and the areas requiring a consideration of the preexisting evidence for minority populations (the crosshatched arrows). The arrows in the model represent the questions the panel considered when looking at the evidence (see the Evidence Model questions on pages 110 to 111).

1.d. Search and Review of the Literature

The literature was searched and systematically reviewed by

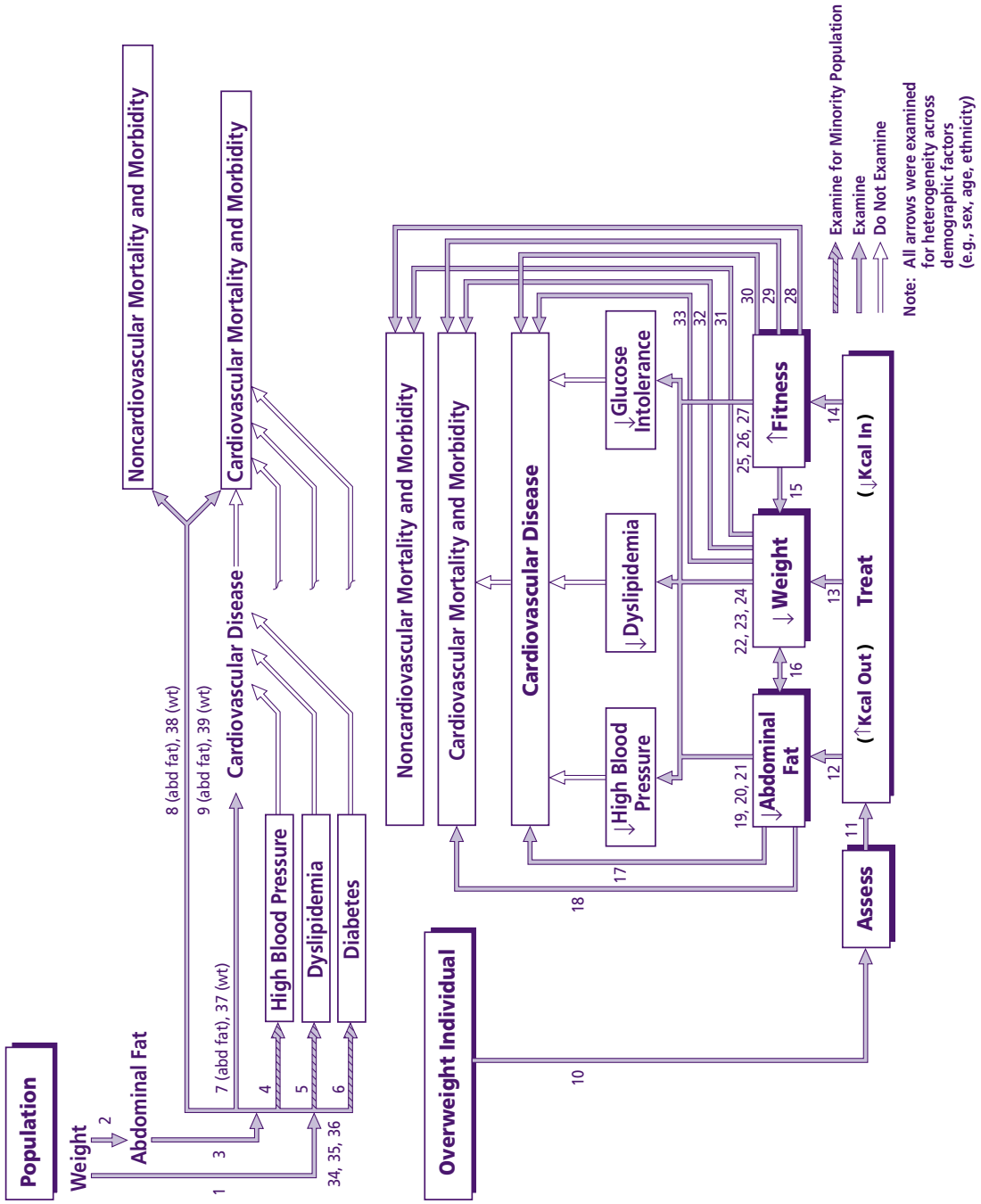
- establishing *a priori* eligibility criteria for inclusion of studies;
- reviewing titles and abstracts to select promising articles;
- reviewing these full articles; and
- compiling evidence tables summarizing those articles that met the inclusion criteria.

The panel decided on the parameters for the literature search, particularly for the search dealing with the model linking treatment to weight loss. The parameters of the search included year of publication, country, language, study design, length of the study, outcome measures (which, what, and when), and patient characteristics (age and weight).

The panel decided to limit its search to English-language studies, but included foreign studies that provide an English abstract and are randomized controlled clinical trials. The search included human studies only published in MEDLINE from January 1980 to September 1997. No editorials, letters, or case reports were accepted. The panel was aware that two books published in the early 1980s coded the bulk of the obesity literature written to that point.

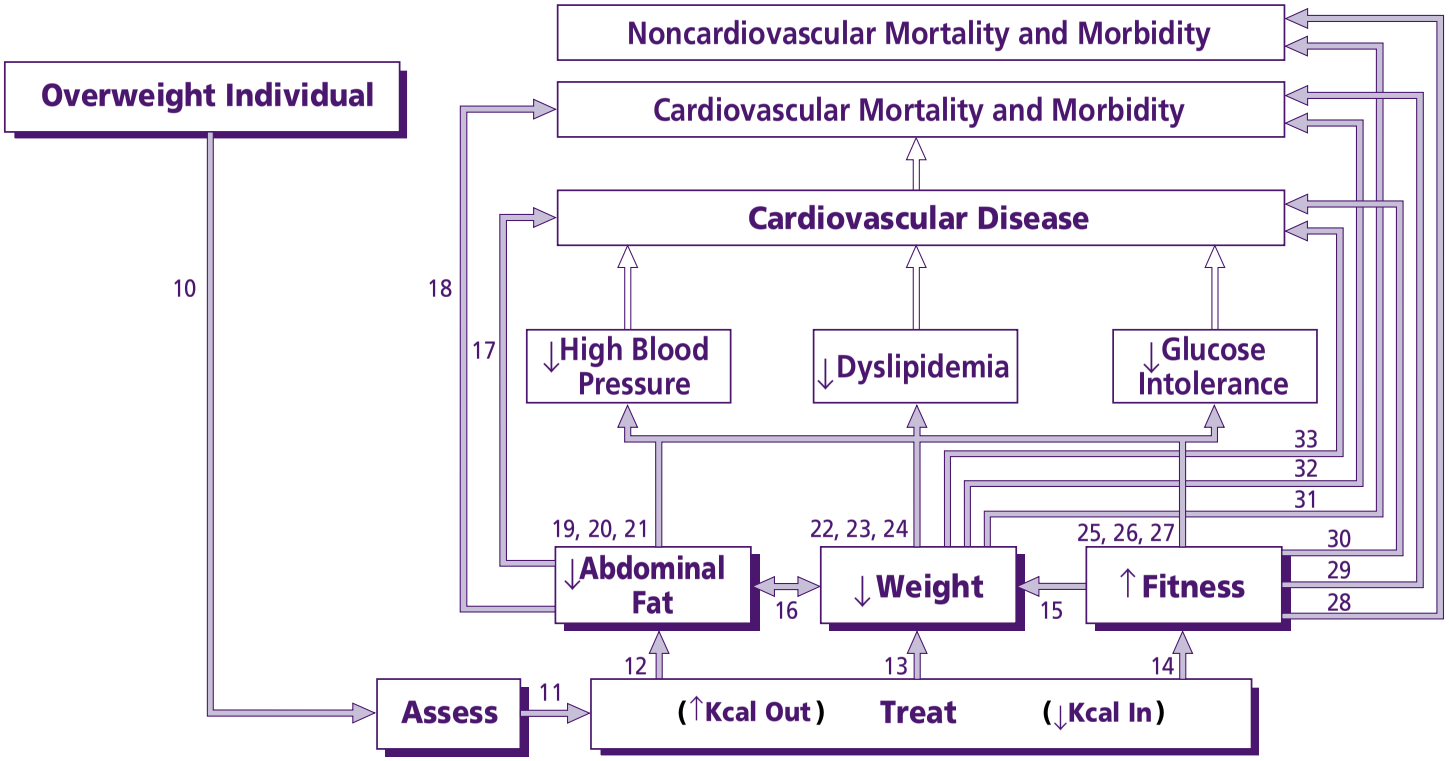
visited on 6/14/2019

Figure 7
Evidence Model



visited on 6/14/2019

Figure 7a
Treatment Evidence Model

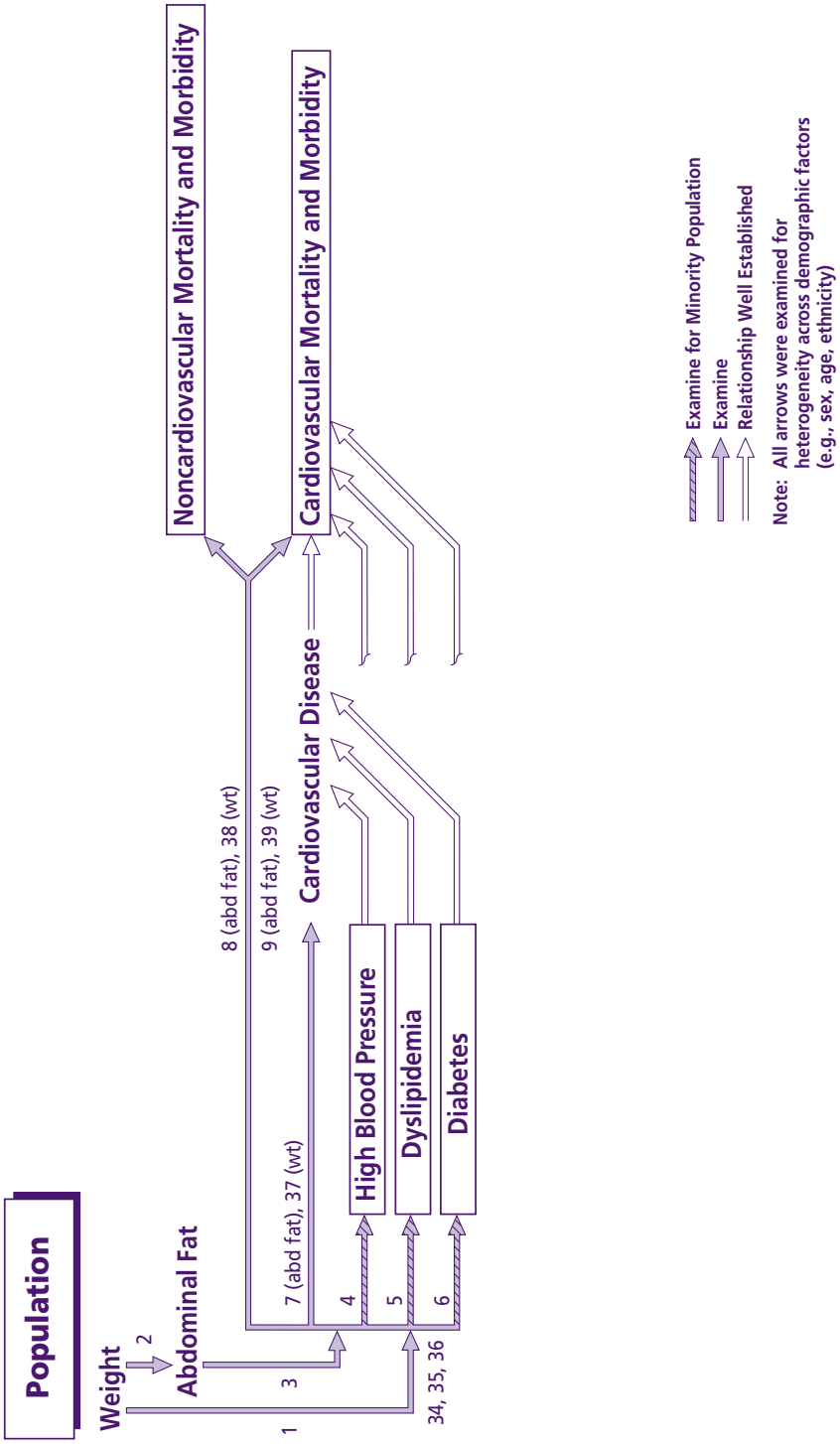


Examine
Relationship Well Established

Note: All arrows were examined for heterogeneity across demographic factors (e.g., sex, age, ethnicity)

visited on 6/14/2019

Figure 7b
Evidence Model
Association of Weight to Cardiovascular and Noncardiovascular Mortality and Morbidity



visited on 6/14/2019

Panel staff and librarians at the National Library of Medicine's National Information Center for Health Services Research determined the medical subject heading terms used in the literature review. The number of citations found by topic area is provided in Appendix I.A.2.

Figure 8 illustrates the various steps used to collect the evidence. Of the 43,627 titles acquired from the MEDLINE search, 18,217 were duplicates, leaving 25,410 titles to be considered. Using the ProCite reference database, the 25,410 titles were screened by two reviewers and ultimately marked for their appropriateness. Abstracts for the appropriate 8,040 remaining titles were then screened for relevance in two rounds. Two individuals independently reviewed each abstract using the inclusion and exclusion criteria developed by the panel. A third reviewer would sporadically check the quality of the screening. This review led to 2,440 possibly relevant abstracts. Due to the greater quality of the evidence, the decision was made that randomized controlled trials pertinent to the treatment portion of the model be the top priority for data abstraction. Ultimately, 394 articles of randomized controlled trials were reviewed for data abstraction.

1.e. Literature Abstraction and Preparation of Evidence Tables

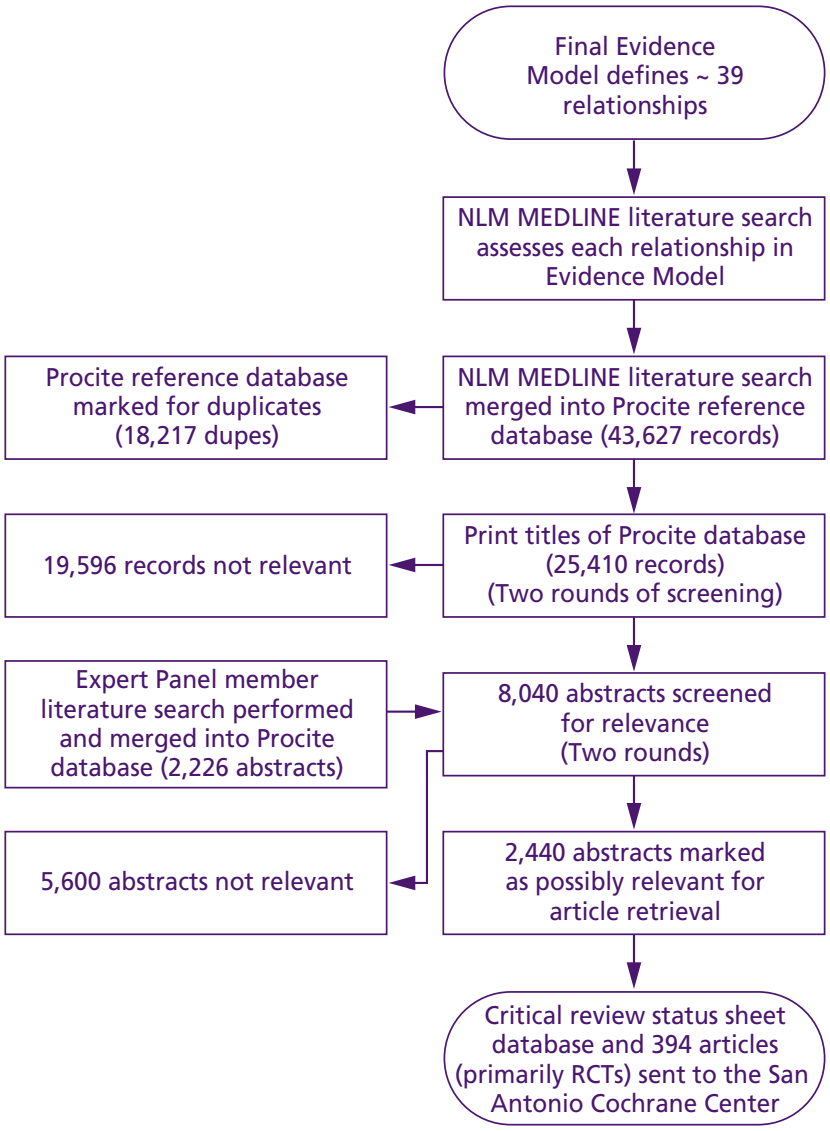
In order to abstract the correct data related to the treatment portion of the model in a consistent manner, a 25-page form called the "Critical Review Status Sheet (CRSS)" was developed. The CRSS was reviewed and approved by the panel. It took into account the inclusion and exclusion criteria described below and allowed for (1) the collection of data on the study's purpose and design; (2) the intervention parameters, including the format, setting, practitioners, population demographics, and detailed descriptions of the intervention per se, be it dietary, physical activity, behavioral, pharmacotherapy, or other types of interventions (surgery, acupuncture, hypnosis, etc.); and (3) outcome measures, including the method of determining the outcome, and adverse events. The CRSS was pilot-tested by staff as well as by staff of the San Antonio Cochrane Center.

The San Antonio Cochrane Center is one of 12 centers around the world that comprise the Cochrane Collaboration. The Cochrane Collaboration is an international organization established in 1993 whose mission is to prepare, maintain, and disseminate systematic reviews and meta-analysis of health care interventions. The San Antonio Center provides advice on multiple aspects of systematic review, including searching and selecting materials, abstracting materials, organizing materials into evidence tables, and performing meta-analysis.

Inclusion and exclusion criteria—The appropriateness of an article was determined by applying criteria determined by the panel. These criteria included the time frame for the study, i.e., the minimum amount of time that must pass before the outcome measure is made; how body weight was reported; and the type and size of the study. In defining the time frame, the panel needed to clarify if the important outcome was weight loss or sustained weight loss. Due to the importance of both outcomes, the panel decided to include studies that considered the effects during the acute phase of weight loss, and those that examined effects during the maintenance phase. Both types of studies were deemed important, and two cutpoints were considered appropriate for follow-up. At a minimum, studies had to have a time frame from start to finish of at least 4 months. However, in order to consider the question of long-term maintenance, studies with outcome data provided at approximately 1 year or longer were examined. The panel decided to exclude studies that used only self-reported weights for their measurement. No exclusion of studies was made by study size.

visited on 6/14/2019

Figure 8
Obesity Guideline Development Process
Evidence Collection Schema



visited on 6/14/2019

Preparation of evidence tables—Of the 394 articles of randomized controlled trials considered for data abstraction by the San Antonio Cochrane Center, 158 were rejected for a variety of reasons; i.e., randomization was not adequate or subjects were not all overweight or obese. Ultimately, 236 articles were abstracted by two separate reviewers who independently read and abstracted each article according to the CRSS. The reviewers met, compared coding, and resolved discrepancies. The data were then compiled into individual evidence tables developed for each RCT. In addition, summary tables were developed to compile the evidence necessary to address the 23 questions relevant to the treatment portion of the model. All of the evidence tables will be available for online retrieval (see information regarding availability of the full obesity evidence report and other resources related to weight control on pages 165-167, and 226-228.

1.f. The Formulation of the Evidence into the Guidelines

In order to consider the evidence for each of the 23 questions, the panel met in groups of 6 to 10 members. During the first round of considering the evidence, 12 small groups of panel members were randomly assigned to consider the evidence for 2 or 3 questions. They developed evidence statements and determined the strength of the evidence using the criteria noted on page 108. Their recommendations were presented to the full panel, which then made additional recommendations regarding their conclusions.

After considering additional studies for questions where adequate RCTs were not available, another iteration of evidence statements and recommendations was developed. The full panel met again to consider this iteration, which was considered by small groups assigned to focus on either a specific treatment modality or outcome measure. The small groups brought their recommendations back to the full panel for their final consideration. The panel weighed the evidence based on a thorough examination of the threshold or magnitude of the treatment effect.

Criteria:

Each evidence statement (other than those with no available evidence) and each recommendation is categorized by a level of certainty (A through D) as described on the next page. The consensus process used for drawing conclusions and writing the recommendations was a group process that took into account all opinions. Conclusions reflect the widest possible agreement of the panel.

1.g. Consideration of Special Populations and Situations

The panel also evaluated population factors and clinical situations that might potentially influence the physiological, medical, behavioral, or sociocultural context for obesity identification and treatment. Evidence on special populations and situations was captured from non-RCT evidence when available, but in many cases such evidence was meager. Population factors selected for special consideration with respect to obesity classification and treatment were age, gender, race/ethnicity, socioeconomic status, pregnancy, eating disorders, sleep apnea, extreme obesity, concurrent treatment of other major conditions (such as heart disease or diabetes), and treatment of obesity in conjunction with smoking cessation.

These considerations were addressed in all stages of guideline development. Persons with relevant special expertise were invited to serve on the panel. The search for evidence was formulated to ascertain studies that include special populations. Studies evaluated were coded to permit analysis of the extent to which the special populations or situations of interest had been included and whether findings for these populations and situations deviated from those for the majority populations in these studies. In the numerous cases where evidence on these special

visited on 6/14/2019

Evidence Categories

Evidence Category	Sources of Evidence	Definition
A	Randomized controlled trials (rich body of data)	Evidence is from endpoints of well-designed RCTs (or trials that depart only minimally from randomization) that provide a consistent pattern of findings in the population for which the recommendation is made. Category A therefore requires substantial numbers of studies involving substantial numbers of participants.
B	Randomized controlled trials (limited body of data)	Evidence is from endpoints of intervention studies that include only a limited number of RCTs, post hoc or subgroup analysis of RCTs, or meta-analysis of RCTs. In general, Category B pertains when few randomized trials exist, they are small in size, and the trial results are somewhat inconsistent, or the trials were undertaken in a population that differs from the target population of the recommendation.
C	Nonrandomized trials Observational studies	Evidence is from outcomes of uncontrolled or nonrandomized trials or from observational studies.
D	Panel Consensus Judgment	Expert judgment is based on the panel's synthesis of evidence from experimental research described in the literature and/or derived from the consensus of panel members based on clinical experience or knowledge that does not meet the above-listed criteria. This category is used only in cases where the provision of some guidance was deemed valuable but an adequately compelling clinical literature addressing the subject of the recommendation was deemed insufficient to justify placement in one of the other categories (A through C).

visited on 6/14/2019

issues was insufficient to meet standards for inclusion in the main text of the guideline, potentially important issues were identified for the user and cross-referenced to an appendix or discussed in sidebar text.

1.h. External Review of the Guidelines

The external review of the guidelines included 115 reviewers from 59 government agencies, professional societies, and consumer groups represented on the Coordinating Committees of the National Cholesterol Education Program and the National High Blood Pressure Education Program, as well as the NIDDK's National Task Force on the Prevention and Treatment of Obesity, and selected members of the North American Association for the Study of Obesity.

Reviewers were asked to evaluate the guideline based on five criteria: validity, clarity, flexibility, completeness, and clinical applicability. Reviewers were also encouraged to provide additional comments. Comments of the peer reviewers were evaluated by the panel and panel staff, and were incorporated into the guideline where appropriate.

1.i. Caveats to Recommendation Use

In applying these guideline recommendations, the reader should note some caveats:

- The emphasis of these guidelines was to identify effective interventions, not to rank-order interventions in terms of relative efficacy or effectiveness. The panel chose not to emphasize comparisons among interventions, because there were few studies that compared long-term outcomes, and, since patient preference often dictates choice of therapy, we wished to present a menu of options rather than a ranked list of choices.
- When no evidence was available on the efficacy of various treatments, the panel usually rendered no opinion. An absence of studies should not be confused with an absence of effect. While clinicians may wish to use proven therapies in preference to untested therapies, the lack of testing does not prove that the untested therapy does not work.
- The limitations of RCTs must be kept in mind. The RCT is the primary method for demonstrating efficacy. Often, patients enrolled in RCTs differ from the patients in a primary care practice, and effectiveness in the community may differ from efficacy as measured in an RCT. The potential exists for misinterpretation of clinical trial results. Analysis of endpoints not specified at the outset, post hoc, or subgroup analyses should be viewed as hypothesis-generating rather than hypothesis-testing.

Appendix I.A.2. Literature Review

The results of the literature search are provided below in terms of the population (top) and clinical (bottom) portions of the evidence model. A total of 43,627 records were obtained from the search; the following notes the number of records per topic.

Population model search citations by topic area (22,017 total):

cardiovascular disease	3,675 citations
hypertension	93 citations
diabetes	65 citations
dyslipidemia	73 citations
cardiovascular morbidity and mortality	225 citations
noncardiovascular morbidity and mortality	4,272 citations

Clinical model search citations by topic area (11,268 total):

cardiovascular disease	1,866 citations
hypertension	616 citations
diabetes	380 citations
dyslipidemia	917 citations

visited on 6/14/2019

Evidence Model Questions
Population Model (Top)

Arrow Number	Question
1	Not considered
2	What is the evidence that relates weight to abdominal fat?
3	Not considered
4	What is the evidence that relates abdominal fat to high blood pressure in minority populations?
5	What is the evidence that relates abdominal fat to dyslipidemia in minority populations?
6	What is the evidence that relates abdominal fat to diabetes in minority populations?
7	What is the evidence that relates abdominal fat to cardiovascular disease?
8	What is the evidence that relates abdominal fat to noncardiovascular mortality and morbidity?
9	What is the evidence that relates abdominal fat to cardiovascular mortality and morbidity?
34	What is the evidence that relates weight to high blood pressure in minority populations?
35	What is the evidence that relates weight to dyslipidemia in minority populations?
36	What is the evidence that relates weight to diabetes in minority populations?
37	What is the evidence that relates weight to cardiovascular disease?
38	What is the evidence that relates weight to noncardiovascular mortality and morbidity?
39	What is the evidence that relates weight to cardiovascular mortality and morbidity?

visited on 6/14/2019

Evidence Model Questions
Clinical Model (Bottom)

Arrow Number	Question
10	Not considered
11	Not considered
12	What is the evidence that treatment directly affects abdominal fat?
13	What is the evidence that treatment directly affects weight loss?
14	What is the evidence directly relating treatment and fitness?
15	What is the evidence that fitness is directly related to weight loss?
16	What is the evidence that weight loss directly affects abdominal fat?
17	What is the evidence that a reduction of abdominal fat is directly related to cardiovascular disease?
18	What is the evidence that a reduction of abdominal fat is directly related to cardiovascular mortality and morbidity?
19	What is the evidence that a reduction in abdominal fat directly affects high blood pressure?
20	What is the evidence that a reduction in abdominal fat directly affects dyslipidemia (cholesterol)?
21	What is the evidence that a reduction in abdominal fat directly affects glucose tolerance?
22	What is the evidence that weight loss directly affects high blood pressure?
23	What is the evidence that weight loss directly affects dyslipidemia (cholesterol)?
24	What is the evidence that weight loss directly affects glucose tolerance?
25	What is the evidence that fitness directly affects high blood pressure?
26	What is the evidence that fitness directly affects dyslipidemia (cholesterol)?
27	What is the evidence that fitness directly affects glucose tolerance?
28	What is the evidence that fitness is directly related to noncardiovascular mortality?
29	What is the evidence that fitness is directly related to cardiovascular mortality and morbidity?
30	What is the evidence that fitness is directly related to cardiovascular disease?
31	What is the evidence directly relating weight loss and noncardiovascular mortality and morbidity?
32	What is the evidence directly relating weight loss and cardiovascular mortality and morbidity?
33	What is the evidence directly relating weight loss and cardiovascular disease?

visited on 6/14/2019

cardiovascular morbidity and mortality	71 citations
noncardiovascular morbidity and mortality	6,507 citations

Note: The number of citations per topic do not add up to the total citations per top or bottom model or the overall NLM search. The total number includes all of those listed above plus the citations for fitness, weight loss, and abdominal fat.

2 Appendix II. Description of Evidence

A. Why Treat Overweight and Obesity?

Blood Pressure

To evaluate the effect of weight loss on blood pressure and hypertension, 76 articles reporting randomized controlled trials (RCTs) were reviewed. Of these, 60 were lifestyle trials that studied diet and/or physical activity and 16 were pharmacotherapy trials of anorexiants. The 60 lifestyle trials were: [346-352, 354-380, 405, 470, 473, 484, 490, 674-694] The 16 articles on pharmacotherapy trials of anorexiants were: [386-395, 512, 695-699]

Lifestyle Trials

Of the 60 lifestyle trials reviewed, 35 were accepted and 25 were not included for these reasons:

- the no-treatment control group also lost weight; [470, 473, 484, 490, 675-680, 683, 684, 688, 690, 692, 693]
- there was no appropriate control group; [687]
- the dropout rate was more than 35 percent; [685, 686]
- the results were not compared according to randomized treatment assignments; [681]
- the population at baseline was not overweight; [689-691]
- blood pressure was not reported as a primary or secondary outcome; [405] or
- the intervention period was less than 4 weeks. [674]

Pharmacotherapy Trials

Of the 16 pharmacotherapy trials reviewed, 10 were accepted and 6 were not included because:

- the drug that was tested is not recommended for weight loss [695] [ephedrine alone] [696] [a beta-adrenoceptor agonist] [697] [ephedrine combined with caffeine] [698] [testosterone] [699] [cimetidine], or
- only very short-term data are available. [512]

Serum/Plasma Lipids and Lipoproteins

Sixty-five RCT articles were evaluated for the effect of weight loss on serum/plasma concentrations of total cholesterol, low-density lipoprotein cholesterol, very low-density lipoprotein cholesterol, triglycerides, and high-density lipoprotein cholesterol. In 52 of these trials weight loss was induced by various lifestyle modifications, including diet modification and increased physical activity. [356, 365, 367-370, 373, 380, 384, 399-406, 412, 431, 433, 434, 440, 446, 447, 469, 470, 473, 475, 484, 490, 677-679, 681, 683-685, 688-690, 693, 694, 700-709]

Thirteen RCT articles were reviewed that considered the effects of pharmacological therapy on weight loss and subsequent changes in total serum cholesterol level. [386, 390-393, 395, 407, 408, 696-698, 710, 711]

Lifestyle Trials

Of the 52 lifestyle trials:

- 27 RCT articles examined the effect on plasma lipids of weight loss induced by diet alone, i.e., these studies simply included a diet intervention without a formal physical activity program or medications. [356, 368-370, 373, 399, 400, 402, 412, 431, 433, 440, 484, 490, 677, 679, 683, 688-690, 693, 701, 702, 704, 706, 708, 709]
- 11 RCT articles examined weight loss induced by increased physical activity alone; [365, 401, 404, 406, 447, 678, 685, 700, 703, 705, 707] and
- 14 RCT articles considered the combination of caloric restriction and physical activity. [367, 380, 384, 403, 405, 434, 446, 469, 470, 473, 475, 681, 684, 694]

Of the 52 lifestyle trials reviewed, 14 met the criteria for inclusion. [365, 368, 370, 373, 380, 384, 399-406] The

visited on 6/14/2019

remaining 38 were not considered because:

- The no-treatment control group also lost weight. [434, 446, 469, 470, 473, 475, 484, 490, 677-679, 683, 684, 688, 690, 693, 694, 701, 705, 706, 708, 709]
- There was no appropriate control group; [431, 700]
- There was no difference in weight loss between the treatment and control group; [447, 702, 703]
- The dropout rate was more than 35 percent; [685]
- The population was not overweight; [689, 690]
- The results were not reported according to randomized treatment; [681]
- The outcome measures were not reported separately for the overweight and not overweight groups; [440]
- There was no measure of at least one lipoprotein fraction or measure of change in lipids between the beginning and end of treatment; [356, 367, 412, 433, 704] or
- Lipoprotein concentrations could not be determined from the figures in the paper, [707] or it was difficult to get precise measures. [369]

Pharmacotherapy Trials

The effects of pharmacological therapy on weight loss and subsequent changes in total serum cholesterol levels were evaluated by examining 13 RCT articles: 5 on the effect of dexfenfluramine; [386, 390- 393] 2 on fluoxetine; [408, 711] 1 on the combination of phentermine and fenfluramine (phen/fen); [395] 2 on orlistat; [407, 710] 1 on testosterone decanoate; [698] 1 on ephedrine alone or in combination with caffeine; [697] and 1 on a beta adrenoceptor agonist. [696] Of the 13 pharmacotherapy trials reviewed, 8 were accepted and 5 were not considered for these reasons:

- The main outcome of the treatment was not weight loss; [698]
- The drug in question is not currently being recommended for weight loss; [696-698]
- The dropout rate was more than 35 percent; [711] or
- It was a marginal 12-week study. [710]

Impaired Glucose Tolerance and Diabetes

Forty-nine articles on RCTs were reviewed to evaluate the effect of weight loss on fasting blood glucose and fasting insulin. [70, 362, 367, 369, 373, 386, 387, 390-393, 403, 404, 408, 412-415, 439, 440, 446, 470, 473, 474, 482, 485, 490, 492, 493, 576, 676-678, 681, 683, 684, 689, 690, 693, 694, 696-698, 700-702, 708, 709, 712]

Studies were conducted in individuals with normal blood glucose levels (fasting plasma glucose < 115 mg/dL [< 6.4mmol/L]), [367, 373, 386, 387, 390-393, 403, 412, 415, 677, 678, 681, 683, 684, 689, 690, 694, 696-698, 700] in individuals with impaired glucose tolerance (fasting plasma glucose of < 140 mg/dL [7.8 mmol/L] or 2 hours postprandial plasma glucose of ≥ 140 to < 200 mg/dL [7.8 to 11.1 mmol/L]), [70, 369] or in individuals with diabetes (fasting plasma glucose of > 140 mg/dL or 2 hours postprandial plasma blood glucose ≥ 200mg/dL). [362, 404, 408, 413, 414, 439, 440, 446, 470, 473, 474, 482, 485, 490, 492, 493, 576, 676, 693, 701, 702, 708, 709, 712*]

Lifestyle Trials

Of the 38 trials reviewed on the role of lifestyle in weight loss and subsequent changes in blood glucose levels, 16 were on diet alone, [70, 373, 412, 413, 415, 439, 440, 576, 677, 683, 689, 693, 701, 702, 708, 709] 8 dealt with physical activity alone [70, 369, 404. 474, 676, 678, 690, 700], 8 focused on diet plus physical activity, [70, 362, 403, 446, 470, 473, 681, 684] and 5 examined the role of behavior therapy. [367, 482, 485, 490, 493] One study included diet, behavior therapy and physical activity. [694] One study included diet, behavior therapy and physical activity. [694] One study [414] included diet, behavioral therapy and drug therapy. Of the 38 trials, 9 were accepted, and 29 were not considered for these reasons:

- There was a control group that had a weight loss intervention or there was no difference in weight loss between the treatment and control

* The reviewed articles used the "old" definitions of the American Diabetes Association (ADA) for impaired glucose tolerance and diabetes. As of November 1997, the new ADA definitions define "impaired fasting glucose" as those individuals having a fasting plasma glucose of 110 to 125 mg/dL, and "diabetes" as those individuals having a fasting plasma glucose of ≥ 126 mg/dL or 2 hours postprandial plasma glucose of ≥ 200 mg/dL.

visited on 6/14/2019

groups; [439, 446, 470, 473, 474, 482, 485, 490, 492, 493, 576, 676-678, 683, 684, 690, 693, 694, 701, 702, 708, 709]

- The dropout rate was more than 35 percent; [415]
- The results were not reported according to randomized treatment assignments; [681]
- Outcome measures were not reported separately for the obese and nonobese people who lost weight; [440]
- There was no appropriate control group; [700] or
- The population was not overweight. [689, 690]

Pharmacotherapy Trials

Of the 12 pharmacotherapy trials reviewed, 8 were accepted and 4 were excluded for these reasons:

- The main outcome of the treatment was not weight loss; [698] or
- The drug used is not currently being recommended for weight loss. [696-698, 712]

B. What Treatments Are Effective?

Dietary Therapy

Of the 86 RCT articles evaluated for the effectiveness of dietary therapy on weight loss, 49 were accepted; 37 were not considered for the following reasons:

- Diet alone was not evaluated; [376, 405]
- There was no control group; [442, 446, 675, 676, 680, 683, 686, 688, 693, 702, 709, 713-720]
- There was no appropriate control group; [677, 704]
- The dropout rate was more than 35 percent; [415]
- The weight loss intervention was less than 12 weeks; [402, 474, 477, 576, 674, 708, 714, 721-723]
- The study did not provide specific information to assess the diet; [724] or
- The patients were not overweight. [689-691]

Physical Activity

Twenty-three RCT articles were reviewed for the effect of physical activity on weight loss, body fat distribution (abdominal fat), and fitness level; of these, 13 were accepted and 10 were not considered. The 23 RCTs were: [346, 363, 365, 369, 375, 401, 404, 406, 432, 434, 445-447,474, 475,678, 685, 700, 705, 707, 725-727] Of the 23 RCTs, 11 were conducted on overweight or obese populations with an average body mass index > 25 kg/m^2. [346, 365, 369, 401, 434, 445-447, 685, 700, 725] Four RCTs were conducted on an overweight or obese population with an average weight of 120 percent to 160 percent of ideal body weight. [363, 406, 432, 475] One RCT was conducted on obese or overweight populations with a percent of body fat > 25 for men or > 30 for women. [678]

Nine RCT articles presented results on men, [363, 365, 369, 406, 432, 700, 705, 707, 726] six RCTs were conducted on women, [434, 445, 446, 678, 685, 725] and seven RCTs covered both men and women. [346, 401, 404, 447, 474, 475, 727]

One RCT article included hypertensive individuals, [346] three RCTs included people with type 2 diabetes, [404, 446, 474] and one RCT included individuals with hyperlipidemia. [685]

Ten RCT articles were not included because:

- The population at baseline was not overweight, [705, 727] or it was not clear if the population was overweight; [707, 726]
- The intervention period was less than 12 weeks; [474]
- There was no appropriate control group; [475, 678, 700, 725] and
- The dropout rate was more than 35 percent. [685]

Combined Therapy

Twenty-three RCT articles investigated the effects on body weight of a combination of a reduced-calorie diet with increased physical activity. The control groups used diet alone or exercise alone. [346, 365, 375-377, 380, 384, 405, 434, 435, 445, 448, 469-475,

visited on 6/14/2019

[681, 684, 728, 729] Of the 23, 15 were accepted and 8 were not considered for the following reasons:

- There was no exercise-alone or diet-alone comparison group; [376, 405, 475, 681, 684, 728, 729] or
- The intervention was less than 12 weeks. [474]

Behavior Therapy

Additional Benefits Beyond Other Weight Loss Approaches

Thirty-six RCT articles were reviewed to evaluate whether behavior therapy provides additional benefit beyond other weight loss approaches. Of the 36, 4 were accepted and 32 were not considered. The 36 trials were: [350, 373, 376, 379, 395, 399, 400, 412, 414, 415, 436-438, 443,444, 469, 470, 473, 476,-478, 490, 510, 677, 679, 688, 700,708, 709, 730-736]

Four RCT articles that compared behavior therapy plus another weight loss strategy to that weight loss strategy without behavior therapy, and that met the inclusion criteria, were accepted. Three of the accepted studies compared behavior therapy to a dietary intervention. [436, 476,477] No studies were found that compared behavior therapy to either exercise or to a combination of diet and exercise. One study was found that compared behavior therapy to drug therapy. [478]

Thirty-two of the articles were not considered because:

- Either behavior therapy was not used or it was not possible to ascertain whether it was used; [412, 490, 677, 730]
- Behavior therapy was used either alone or as part of an intervention, but the comparison groups used did not allow for determining the unique effect of behavior therapy; [350, 373, 376, 399, 400, 414, 415, 444, 470, 700, 732, 735] or
- Behavior therapy was integrated into all of the intervention groups, making it impossible to detect the unique effect of behavior therapy. [379, 395, 437, 438, 443, 469, 473, 510, 679, 688, 708, 709, 731, 733, 734, 736]

Comparison of Behavior Therapy Strategies

Another way to examine the efficacy of behavior therapy in the treatment of obesity is to evaluate studies that compare various behavioral techniques to one another. These studies are primarily from the behavioral psychology literature.

Thirty-one RCT articles were reviewed that compared one or more behavioral interventions. [367, 445, 476, 479-495, 632, 680, 694, 737-744] Most of the trials used a group format and followed subjects over time; three included booster sessions. [484, 487, 491] Twelve of the studies provided 1 year or more of follow-up weights. Most studies lacked a pure, no-treatment, control group. There were limited intervention data on special populations, including various ethnic groups and populations with low income and education. Men and women were well represented. The majority of patients were young and middle-aged adults.

Nine of the trials studied special patient populations: subjects with type 2 diabetes; [482, 485, 489, 490, 492 493, 739] subjects at high risk for coronary artery disease; [484] and subjects with binge eating disorder. [494] Trials with subjects with type 2 diabetes used behavioral approaches to increase adherence to the American Diabetes Association diet and improve glycemic control, rather than to lose weight as the primary objective (although all reported an effect on weight).

Of the 31 articles reviewed, 19 were accepted and the following 12 were not included because:

- The results were not presented by treatment group; [694]
- The dropout rate was more than 35 percent; [445, 632, 742, 744]
- Self-reported body weight was used; [741]
- There was no control group; [680]
- The treatment duration was less than 12 weeks; [737-739, 743] or
- The population was not overweight. [740]

visited on 6/14/2019

Pharmacotherapy

Forty RCTs evaluated the effect of pharmacotherapy on weight loss. In most studies, advice or behavioral therapy that promoted reduced energy intake and increased physical activity was included in all treatment arms, including the placebo group. The RCTs addressed the following pharmacotherapies: BRL 26830A, [696] cimetidine, [699] dexfenfluramine, [386, 387, 390-393, 414, 478, 506-508, 711, 745, 746] ephedrine, [695, 697, 747] fenfluramine, [478] fenfluramine and phentermine, [395, 514, 748, 749] femoxetine, [750] fluoxetine, [408, 712, 730, 734, 751, 752] mianserine, [753] orlistat, [407, 710] phenylpropanolamine, [511, 512] phentermine, [176, 754] testosterone, [698] yohimbine, [755] and sibutramine. [510] Most of the patients in these trials weighed more than 120 percent of their ideal body weight or had a BMI of 25 to 30.

RCTs that examined cimetidine, mianserine, yohimbine, testosterone, femoxetine, and BRL 26830A were not considered either because they are not now being studied for obesity treatment or because they are not approved by the Food and Drug Administration (FDA) for obesity treatment. Fluoxetine, and other selective serotonin reuptake inhibitors commonly used for the treatment of depression, have not been approved for use as antiobesity agents. Ephedrine has been tested alone and in combination with caffeine. Although a significant 3.4 kg difference was found at 6 months in patients treated with this combination compared with those on placebo [695], the ephedrine/caffeine combination is not approved by the FDA for use in the United States. In addition, studies with a dropout rate greater than 35 percent were not considered [711]. As of September 1997, two FDA-approved drugs, dexfenfluramine and fenfluramine, were withdrawn from the market by the manufacturer due to an observed association with valvular heart disease in patients taking the combination of phentermine/fenfluramine, or "Redux." The panel's deliberations on the evidence regarding these drugs are included in the report for informational purposes only.

Weight Loss Surgery

Fourteen RCTs compared the weight-reducing effect of different surgical interventions. [515-522, 524, 692, 706, 733, 756, 757] One study compared the effectiveness of a very low-calorie diet to surgery. [515] Another study compared the effectiveness of horizontal-banded to vertical-banded gastroplasty in a pretreated, very low-calorie formula diet group. [516] Six studies compared two or more of the following procedures: gastroplasty (vertical or horizontal), gastric bypass, and gastric partitioning. [517-522] Seven studies reported long-term (1 year or more) follow up. [515, 517-522] Two studies looked at comorbidity factors associated with weight loss. [517, 519]

Of these 14 RCT studies, the following five were not included because:

- Two studies looked at the effect on weight loss of jejunoileostomy, a procedure that is no longer recommended; they were not included because of the complications associated with this procedure; [692, 706] and
- Three studies compared the use of the gastric balloon to sham procedures; [733, 756, 757] however, this procedure is no longer utilized except in research studies.

visited on 6/14/2019

3 Appendix III. Special Populations

A.1. Prevalence of Overweight/Obesity in U.S. Children

a. Number and percentage of children (ages 6 to 11 years) and adolescents (ages 12 to 17 years) who were overweight, by gender and race/ethnicity—United States, Third National Health and Nutrition Examination Survey (NHANES III), 1988-1994.

b. Prevalence of overweight (BMI ≥ 85th percentile) in Native American school-age children by gender and age, 1990-1991.

c. Selected studies describing the prevalence of overweight in Native Americans, 1981-1993.

A.2.Overweight or BMI Data for Puerto Rican-Americans, Cuban-Americans, and Selected Samples of American Indians, Asians, and Pacific Islanders.

a. Percentage of overweight Cuban and Puerto Rican males and females, age 20 to 74, 1982-1984.

b. Selected studies describing the prevalence of overweight in Native Americans, 1981-1993.

c. Prevalence of overweight (BMI ≥ 85th percentile) and obesity (BMI ≥ 95th percentile) in American Indians and Alaska Natives and United States all races, by gender and age, 1987.

d. Overweight prevalence and BMI data for Asian-Americans (n = 13,031) examined in a northern California HMO, 1978-1985.

e. Body mass index in four groups of adult Samoans, by age and gender.

f. Prevalence of obesity (BMI ≥ 30 kg/m^2) in 1991, by age, sex, and western Samoa location.

A.3. Socioeconomic Status Differences in the Prevalence of Overweight/Obesity

a. Prevalence of overweight in U.S. adults by race/ethnicity, gender, and education, 1988-1991.

b. Prevalence of overweight in U.S. adults by race/ethnicity, gender, and annual family income, 1988-1991.

c. Prevalence of overweight in U.S. adults by age, race/ethnicity, gender, and occupation, 1988-1991.

d. Age-adjusted percentage of people 20 years of age and older who are overweight (high BMI) by gender, race/ethnicity, and income level, 1988-1991.

e. Percentage of medicare beneficiaries who are overweight by race, gender, and education, 1991.

f. Percentage of medicare beneficiaries who are overweight by race, gender, and annual family income, 1991.

Note: Due to the fact that the prevalence estimates in this appendix are based on available published data, the definitions used to define overweight and obesity are not the same as those used by the panel.

visited on 6/14/2019

TABLE III.A.1.a:

Number and Percentage of Children (aged 6-11 years) and Adolescents (aged 12-17 years) who were Overweight[1], by gender and Race/Ethnicity[2]

United States, Third National Health and Nutrition Examination Survey (NHANES III), 1988-1994

	Children			Adolescents[4]		
Characteristic	**No.**	**(%)**	**(95% CI)[3]**	**No.**	**(%)**	**(95% CI)**
Males						
White, non-Hispanic	446	(13.2)	(8.7%-17.6%)	281	(11.6)	(7.6%-15.6%)
Black, non-Hispanic	584	(14.7)	(11.2%-18.3%)	412	(12.5)	(9.2%-15.8%)
Mexican-American	565	(18.8)	(14.6%-23.0%)	406	(15.0)	(10.8%-19.1%)
Total	1,673	(14.7)	(11.5%-17.9%)	1,154	(12.3)	(9.3%-15.3%)
Females						
White, non-Hispanic	428	(11.9)	(7.2%-16.5%)	342	(9.6)	(5.5%-13.6%)
Black, non-Hispanic	538	(17.9)	(14.5%-21.2%)	447	(16.3)	(11.9%-20.8%)
Mexican-American	581	(15.8)	(10.3%-21.3%)	412	(14.0)	(6.8%-21.2%)
Total	1,606	(12.5)	(9.4%-15.7%)	1,274	(10.7)	(7.7%-13.7%)
Total[5]	3,279	(13.7)	(11.4%-15.9%)	2,428	(11.5)	(9.0%-14.0%)

1 Overweight is defined as a BMI (kg/m^2) at or above gender- and age-specific 95th-percentile BMI cutoff points calculated at 6-month age intervals, derived respectively from National Health Examination Survey, cycles 2 and 3.

2 Numbers for other racial/ethnic groups were too small for meaningful analysis.

3 Confidence interval

4 Excludes pregnant females and one person with an outlier sample weight.

5 Total estimates include racial/ethnic groups not shown.

Source: *MMWR*, 3-7-97[759].

visited on 6/14/2019

TABLE III.A.1.b:

Prevalence of Overweight (BMI≥85th percentile) in Native American School-age Children by Gender and Age, 1990-91*

	Boys		Girls	
Age	Number	%	Number	%
5	364	36.0	404	30.3
6	539	30.6	494	37.0
7	482	46.5	538	43.1
8	524	42.6	495	42.2
9	511	45.2	469	38.8
10	493	40.5	404	37.6
11	419	36.0	384	35.4
12	369	47.4	311	41.8
13	281	43.8	231	42.4
14	246	35.4	228	36.4
15	249	39.0	209	55.0
16	204	26.5	181	39.8
17	165	35.8	152	40.4
18	75	32.0	43	39.5

* Adapted from Jackson MY and Strauss KF.

Source: Broussard et al. Toward comprehensive obesity prevention programs in Native American communities. *Obesity Res* 1995;3:289-97S. [26]

visited on 6/14/2019

TABLE III.A.1.c:

Selected Studies Describing the Prevalence of Overweight in Native Americans, 1981-1993

Population	Source	Age	Year	Definition of Overweight *	Percentage Total	Percentage Males	Percentage Females
Preschool children							
Native Americans, national	Measured	<5	1988	a	11.2		
Mescalero Apache, NM	Measured	1-5	1988	a	19.5		
School-age children							
Native Americans, national	Measured	5-18	1990-1991	b	39.3		
Devils Lake Sioux, ND	Measured	9-13	1989	b		32.1	30.6
Winnebago and Omaha, NE	Measured	7-17	1990	b		32.7	34.4
Pueblo Indians, NM	Measured	9-13	1988-1991	b	40.4		
Navajo Indians, NM	Measured	9-13	1988-1991	b	29.1		
Navajo Indians, NM & AZ	Measured	5-17	1989	c		12.5	11.2
Navajo Indians, NM & AZ	Measured	14-18	1988-1990	b		25.0	33.0

* Definition of Overweight:
a=Weight-for height > 95th percentile NCHS reference population
b=BMI ≥ 85th% NHANES II reference population
c=Weight-for-age > 95th NCHS reference population

Source: Broussard et al. Toward comprehensive obesity prevention programs in Native American communities. *Obesity Res* 1995;3:289-97S. [26]

visited on 6/14/2019

TABLE III.A.2.a:

Percentage of Overweight Cuban and Puerto Rican Males and Females, Ages 20-74, 1982-1984

Gender/Age	Cubans	Puerto Ricans
Males		
20-74 yrs	29.4	25.2
20-74 yrs (age adjusted*)	28.5	25.7
20-24 yrs	21.2	15.8
25-34 yrs	27.9	18.9
35-44 yrs	25.8	33.4
45-54 yrs	34.6	32.9
55-64 yrs	31.7	26.4
65-74 yrs	31.1	31.6
20-29 yrs**	23.5	15.6
Females		
20-74 yrs	34.1	37.3
20-74 yrs (age adjusted*)	31.9	39.8
20-24 yrs	13.6	23.6
25-34 yrs	23.8	26.5
35-44 yrs	32.7	42.7
45-54 yrs	37.2	50.2
55-64 yrs	51.4	49.0
65-74 yrs	39.6	61.0
20-29 yrs**	16.2	22.5

* Age adjusted by the direct method to the 1980 census population 20 to 74 years of age using 6 age groups.

** Overweight is defined as a sex-specific body mass index (kilograms divided by height in meters squared) equal to or greater than the 95th percentile for examinees 20 to 29 years of age examined in the second National Health and Nutrition Examination Survey (NHANES II).

Note: Excludes pregnant women.

Source: Najjar MF, Kuczmarski RJ. 1989. Anthropometric data and prevalence of overweight for Hispanics: 1982-1984. National Center for Health Statistics. *Vital Health Stat* 11(239). [27]

visited on 6/14/2019

TABLE III.A.2.b:

Selected Studies Describing the Prevalence of Overweight in Native Americans, 1981-1993

Population	Source	Age	Year	Definition of Overweight *	Percentage Total	Percentage Males	Percentage Females
Adults							
Native Americans, national	Self-report	18+	1987	a		33.7	40.3
Native Americans by region	Self-report	18+	1985-1988	a			
Southwest						30.0	28.5
Plains						27.8	35.7
West						22.7	26.8
Other regions						24.0	23.9
Alaskan Yup'ik Eskimos	Measured	18+	1987-1988	a		34.0	56.0
Alaskan Athabascan Indians	Measured	18+	1987-1988	a		29.0	55.0
Cherokee Indians, NC	Measured	18-27	1982	c			59.1
Navajo Indians, NM & AZ	Measured	20+	1988	a	43.7	33.1	50.7
Navajo Indians, NM & AZ	Measured	20-74	1986-1987	d		42.1	54.7
Penobscot Indians, ME	Self-report	18+	1981	c	35.0		
Pima Indians, AZ	Measured	20-54	1981-1988	a		61-78	81-87
		55+	1981-1988	a		31-53	44-74
Zuni Indians, NM	Measured	20-39	1987	e	40.0	33.8	56.2
		40-59	1987	e	70.7	55.6	79.2

* Definition of overweight and obesity used by NHANES/NCHS:
a=BMI≥85th % NHANES II reference population
b=Weight-for-age>95th NCHS reference population
c=>120% desirable weight for height, NHANES I
d=BMI > 85th% NHANES I reference population
e=BMI > 27 kg/m^2 for males, BMI > 26 kg/m^2 for females

Source: Broussard et al. Toward comprehensive obesity prevention programs in Native American communities. *Obesity Res* 1995;3:289-97S. [26]

visited on 6/14/2019

TABLE III.A.2.c:

Prevalence of Overweight (BMI≥85th percentile) and Obesity (BMI≥95th percentile) in American Indians and Alaska Natives and U.S. all races, by Gender and Age, 1987*

	Overweight		Obese	
Age	American Indians and Alaska Natives	U.S. All Races	American Indians and Alaska Natives	U.S. All Races
Males				
Total (18+)	33.7	24.1**	13.8	9.1**
18-24	21.5	13.1**	11.0	5.5***
25-34	31.8	19.5**	11.2	7.6
35-44	37.8	27.0**	11.2	10.4
45-54	49.1	33.8**	28.2	14.1**
55-64	45.5	33.1**	16.5	13.0***
65+	25.2	23.0**	11.1	5.4***
Females				
Total (18+)	40.3	25.0**	16.6	8.2**
18-24	25.2	11.5**	11.7	3.9**
25-34	45.1	17.4**	13.8	6.0**
35-44	48.5	28.1**	19.7	10.8**
45-54	54.0	32.0**	18.7	10.9
55-64	45.6	36.2**	18.8	11.5
65+	45.6	30.1**	20.7	7.7**

* Agency for Health Care Policy and Research, 1987 National Medical Expenditure Survey (NMES). American Indians and Alaska Natives who are eligible for care through the Indian Health Service (IHS) and who live on or near reservations. BMI, body mass index in kg/m^2.

** Statistically significant at $\alpha=0.05$.

*** Relative SE greater ≥ 30%

Source: Broussard et al. Prevalence of obesity in American Indians and Alaska Natives. *Am J Clin Nutr* 1991;53:1535-42S. [628]

visited on 6/14/2019

TABLE III.A.2.d:

Overweight Prevalence and BMI Data for Asian-Americans (n=13,031) Examined in a Northern California HMO, 1978-1985

Trait	Chinese	Filipino	Japanese	Other Asian
Age (mean ± SD)				
Males	40.2 ± 14.2	39.2 ± 13.4	42.1 ± 14.1	35.9 ± 10.4
Females	38.0 ± 13.6	36.8 ± 11.6	40.9 ± 13.4	34.1 ± 10.5
BMI ≥ 24.4 kg/m^2				
Males	26.9	41.8	38.0	28.9
Females	12.8	25.5	18.0	14.6
Mean BMI*				
Males	22.9	23.9	23.7	23.6
Females	21.2	22.8	21.6	22.2

* Adjusted for age, marital status, education, and alcohol intake.

Source: Klatsky AL, Armstrong MA. Cardiovascular risk factors among Asian-Americans living in Northern California. *Am J Public Health* 1991;81:1423-1428. [51]

visited on 6/14/2019

TABLE III.A.2.e:

BMI in Four Groups of Adult Samoans, by Age and Gender*

Gender/Age	Western Samoa	Manu'a	Tutuila	Hawaii
Males				
20-24	24.6 ± 2.8	26.4 ± 2.6	26.5 ± 3.6	30.4 ± 4.6
25-34	25.1 ± 3.2	30.7 ± 4.4	30.2 ± 5.0	31.3 ± 6.0
35-44	26.5 ± 4.3	29.2 ± 4.7	30.4 ± 5.5	32.6 ± 5.4
45-54	27.1 ± 4.9	27.2 ± 4.7	32.0 ± 6.4	31.3 ± 5.7
55-64	28.8 ± 5.4	28.0 ± 5.9	28.8 ± 5.5	32.1 ± 5.0
65-74	26.5 ± 3.9	28.7 ± 4.5	28.6 ± 6.2	31.6 ± 8.9
Total	26.2 ± 4.2	28.6 ± 5.0	29.8 ± 5.7	31.6 ± 5.7
Females				
20-24	25.3 ± 3.9	29.5 ± 7.9	27.9 ± 4.9	29.9 ± 5.8
25-34	26.7 ± 4.1	32.3 ± 6.0	32.4 ± 6.6	34.3 ± 7.8
35-44	29.0 ± 4.7	35.1 ± 5.8	34.4 ± 6.6	35.8 ± 7.7
45-54	29.6 ± 4.8	33.8 ± 7.4	35.3 ± 6.7	34.7 ± 6.5
55-64	31.0 ± 8.4	30.7 ± 6.1	33.3 ± 7.5	34.1 ± 5.5
65-74	28.6 ± 4.1	29.5 ± 5.8	32.3 ± 5.9	33.4 ± 6.9
Total	27.7 ± 4.8	32.7 ± 6.9	33.0 ± 6.9	33.6 ± 7.2

* SD. BMI in kg/m^2.

Source: McGarvey ST. Obesity in Samoans and a perspective on its etiology in Polynesians. *Am J Clin Nutr* 1991;53:1586S-94S. [50]

visited on 6/14/2019

TABLE III.A.2.f:

Prevalence of Obesity (BMI≥ 30kg/m²) in 1991, by Age, Gender, and Western Samoa Location

	Apia		Poutasi		Tuasivi	
Gender/Age	**No.**	**(%)**	**No.**	**(%)**	**No.**	**(%)**
Males						
25-34	71	45.1	58	36.2	51	21.6
35-44	77	70.1	58	43.1	58	46.6
45-54	64	67.2	48	52.1	51	45.1
55-64	78	51.3	40	57.5	44	40.9
65+	40	60.0	28	32.1	30	43.3
Overall	330	58.5	232	44.4	234	39.3
Age-std*		56.9		43.5		35.9
95% CI		51.2 - 62.6		36.9 - 50.1		29.7 - 42.1
Females						
25-34	97	57.7	74	52.7	61	52.5
35-44	83	89.2	48	66.7	77	57.9
45-54	120	84.2	40	70.0	63	66.7
55-64	96	86.5	34	64.7	40	52.5
65+	43	62.8	27	63.0	30	46.7
Overall	439	77.7	223	61.9	271	57.2
Age-std*		74.3		61.5		56.5
95% CI		69.9 - 78.7		55.1 - 68.0		50.2 - 62.7

* Age-std=age standardized by direct method to 1986 census population.

Source: Hodge AM, Dowse GK, Toelupe P, Collins VR, Imo T, Zimmet PZ. Dramatic increase in the prevalence of obesity in Western Samoa over the 13-year period 1978-1991. *Int J Obesity* 1994;18:419-428. [629]

visited on 6/14/2019

TABLE III.A.3.a:

Prevalence of Overweight* in U.S. Adults, by Race/Ethnicity, Gender, and Education, 1988-1991

	Black				Mexican American				White			
Gender	<12y	12y	>12y	Total	<12y	12y	>12y	Total	<12y	12y	>12y	Total
Males	29	29	36	31	39	43	40	40	39	36	30	34
Females	55	51	49	52	53	40	47	49	39	38	30	35

* Overweight is defined as ≥ 27.8 kg/m^2 for men and ≥ 27.3 kg/m^2 for women.
Source: NCHS/CDC NHANES III Phase 1, 1988-1991. [760]

TABLE III.A.3.b:

Prevalence of Overweight* in U.S. Adults, by Race/Ethnicity, Gender, and Annual Family Income, 1988-1991

	Black					Mexican American					White				
Gender	<$10K	$10-29,999	$30-49,999	$50,001+	Total	<$10K	$10-29,999	$30-49,999	$50,001+	Total	<$10K	$10-29,999	$30-49,999	$50,001+	Total
Males	23	31	34	42	31	33	39	45	48	40	33	35	36	31	34
Females	53	50	54	49	52	52	51	43	42	49	43	35	37	27	35

* Overweight is defined as ≥ 27.8 kg/m^2 for men and ≥ 27.3 kg/m^2 for women.
Source: NCHS/CDC NHANES III Phase 1, 1988-1991. [760]

visited on 6/14/2019

TABLE III.A.3.c:

Prevalence of Overweight* in U.S. Adults, by Age, Race/Ethnicity, Gender, and Occupation, 1988-1991

	Black					Mexican American					White				
Gender/Age	White Collar	Blue Collar	Farm	Service	Total	White Collar	Blue Collar	Farm	Service	Total	White Collar	Blue Collar	Farm	Service	Total
Males															
25-44	28	35	15	32	31	46	32	30	36±	35	30	31	24	30±	30
45-64	44	35	15	38±	37	41	58	38	54±	51	38	38±	47	21±	37
Females															
25-44	47	48		55	48	48	40		41	46	27	38		31	29
45-64	56	55		66	60±	55	55		58	57	41	45±		43	42

* Overweight is defined as ≥ 27.8 kg/m^2 for men and ≥ 27.3 kg/m^2 for women.
± Estimate not reliable because of small sample size.
Source: NCHS/CDC NHANES III Phase 1, 1988-1991. [760]

visited on 6/14/2019

TABLE III.A.3.d:

Age-Adjusted Percentage of People 20 Years of Age and Older who are Overweight (High BMI), by Gender, Race/Ethnicity, and Income Level, 1988-1991

Sex and Poverty	Total Population*			Non-Hispanic White			Non-Hispanic Black			Mexican American		
	Sample	Percent	SE	Sample	Percent	SE	Sample	Percent	SE	Sample	Percent	SE
Males												
Below poverty	712	30.4	2.24	142	29.8	3.02	211	29.6	2.53	338	36.9	4.22
At or above poverty	3,069	31.9	1.00	1,625	32.3	1.02	724	32.8	1.65	621	40.6	1.02
Under 131% poverty	1,034	32.8	2.49	238	33.3	3.18	313	28.4	2.20	453	37.2	3.44
131-350% poverty	1,793	32.7	1.38	865	33.6	1.64	460	33.2	2.26	401	41.4	1.75
Over 350% poverty	954	29.0	1.34	664	28.7	1.39	162	36.4	3.31	105	46.1	5.72
Females												
Below poverty	850	47.3	2.70	189	46.9	5.12	290	50.0	3.96	347	50.5	3.02
At or above poverty	2,710	32.2	1.20	1,442	30.1	1.34	637	49.0	1.61	540	44.4	2.89
Under 131% poverty	1,185	46.1	2.11	321	44.7	3.88	390	51.1	3.42	441	50.3	2.79
131-350% poverty	1,643	33.8	1.49	806	31.9	1.54	421	49.0	2.12	349	44.4	4.21
Over 350% poverty	732	28.0	2.30	504	26.4	2.39	116	46.1	3.11	97	40.0	6.81

* Includes data for racial/ethnic groups not shown separately.

Note: BMI is an index used to relate weight to stature. For this analysis, overweight in men is defined as a BMI ≥ 27.8 kg/m^2; for women, it is defined as a BMI ≥ 27.3 kg/m^2.

Source: HHS, NHANES III, 1988-1991. [760]

visited on 6/14/2019

TABLE III.A.3.e:

Percentage of Medicare Beneficiaries who are Overweight*, by Race, Gender, and Education, 1991

Gender/ Age	Black <HS	Black HS	Black >HS	Black Total	White <HS	White HS	White >HS	White Total
Males								
65-74	32.1	33.9**	47.5**	34.0	34.0	30.1	25.5	29.8
75+	20.1	14.9**	16.6**	21.8	18.1	11.1	11.1	15.0
All ages	27.4	26.5	35.5**	28.9	27.8	22.7	19.9	23.8
Females								
65-74	61.8	45.7	45.0**	54.1	40.6	34.5	27.9	34.6
75+	44.7	12.3	43.9	40.2	29.2	21.9	19.6	24.7
All ages	55.2	32.7	44.6	48.7	36.2	29.6	24.6	30.9

* Overweight is defined as a BMI ≥ 27.8 kg/m^2 for males and ≥ 27.3 kg/m^2 for females.
** Based on samples of fewer than 30 persons.
Source: Medicare Current Beneficiary Survey: 1991, Non-Institutionalized Persons only (N=9,721). [630]

visited on 6/14/2019

TABLE III.A.3.f:

Percentage of Medicare Beneficiaries who are Overweight*, by Race, Gender, and Annual Family Income, 1991

Gender/ Age	Black ≤$10,000	$10,001-30,000	$30,001-50,000	$50,001+	Unknown/ not rep	Total	White ≤$10,000	$10,001 30,000	$30,001-50,000	$50,001+	Unknown/ not rep	Total
Males												
65-74	24.0	41.7	27.2	59.02**	0.0**	34.0	26.2	32.3	30.7**	23.9	25.6	29.8
75+	17.8	27.9	—	—	0.0	21.8	16.1	17.3	9.9	9.3	11.5	15.0
All ages	20.6	35.2	11.3	0.0**	0.0**	28.9**	22.0	26.3	22.7	18.0	19.8	23.8
Females												
65-74	59.1	46.1	27.2	34.0**	0.0	54.1	43.2	36.3	25.7**	20.8	25.3	34.6
75+	43.4	28.1	23.0	100.0**	0.0	40.2	27.7	24.4**	20.1**	18.2	20.0	24.7
All ages	53.0	38.2	29.3	30.5**	0.0	48.7**	37.9	31.4	23.1	20.6	23.8	30.9

* Overweight is defined as ≥ 27.8 kg/m^2 for men and ≥ 27.3 kg/m^2 for women.
** Based on samples of fewer than 30 persons.
Source: Medicare Current Beneficiary Survey: 1991, Non-Institutionalized Persons only (N=9,721). [630]

visited on 6/14/2019

A.4. Annotated Bibliography of Studies Specifically Designed To Achieve Weight Reduction in Special Populations.

The following annotations describe weight reduction studies in special populations or situations. From the total number of studies identified in which minorities or other special populations were included, the studies listed below are those for which the treatment or intervention program included design features specifically geared to the needs of a minority population or other special population of interest. Thus, these studies provide examples of ways in which researchers or service practitioners have attempted to adapt conventional weight reduction approaches to the needs of diverse client groups. The annotations highlight the published reports with respect to the setting and the nature of the program. Study results are not included because relatively few studies are randomized controlled trials, several are pilot studies or program descriptions, and many do not lend themselves to a clear evaluation of success rates. Boldface material indicates the particular population or situation studied.

Agurs-Collins TD, Kumanyika SK, TenHave TR, Adams-Campbell LL. A randomized controlled trial of weight reduction and exercise for diabetes management in ***older African-American subjects.*** Diabetes Care *1997;20:1503-1511.* [362]

A randomized controlled trial conducted at an urban hospital in Washington, DC. The intervention group was offered 12 weekly group sessions, 1 individual session, and 6 biweekly group sessions; the usual care group was offered one class and two informational mailings. Participants were recruited through various clinics and through community outreach, including announcements in newsletters and church bulletins, flyers distributed at clinics and churches, and presentations at community gatherings. Program materials depicted African-American individuals, families, and community settings and reflected language, social values, and situations relevant to older Americans or African-Americans. All guidance about foods and recipes was based on types of foods and flavorings characteristic in African-American communities. Recipes were provided by participants and used in direct instruction. Ample time was allowed for the participants to discuss and work through dietary adherence issues unique to their social contexts (e.g., church meals).

Avila P, Hovell MF. Physical activity training for weight loss in ***Latinas****: A controlled trial.* Int J Obes *1994;18:476-482.* [631]

A 10-week physical activity program designed for obese Latino women in San Diego, California. Volunteers were recruited through flyers that were distributed in a low-income community medical clinic, the local church, English as a Second Language classes, and the local grocery store. Sessions focused on self-change behavior, nutrition education, gaining support from a "buddy," and an exercise component. The sessions were led by a bicultural, bilingual physician.

Cousins JH, Rubovits DS, Dunn JK, Reeves RS, Ramirez AG, Foreyt JP. Family versus individually-oriented intervention for weight loss in ***Mexican American women.*** Public Health Rep *1992;107(5):549-555.* [632]

A study that compared three groups of obese Mexican American women in Texas, ages 18 to 45 years. Families were recruited through media promotion and local contacts within the community, primarily through churches and health agencies. The comparison group received a bilingual manual with information on nutrition, exercise, and behavioral principles for weight loss. The manual was designed to reflect the cultural values of the population. The comparison group also received a cookbook with recipes for fat-modified traditional Mexican American foods. The *individual* group received the same printed materials and attended weekly classes led by bilingual registered dietitians. Classes provid-

visited on 6/14/2019

ed individual and group activities. The *family* group received the materials and attended classes that emphasized a family-oriented approach to making changes in eating habits and exercise behavior; spouses and children were invited. Family group members received a modified version of the manual with additional information on partner support and parenting skills to encourage family changes in eating and exercise behaviors. Spouses were encouraged to attend classes with the participants, and separate classes were held for preschool-age children.

Daniel EL. A multi-intervention weight management program for ***low-income rural women.*** J Am Diet Assoc *1989;89:1310-1311.* [633]

A pilot weight management program involving low-income rural women who were enrolled in the Special Supplemental Food Program for Women, Infants and Children (WIC) and/or food stamp programs in rural western New York State. The program included five biweekly sessions that focused on nutrition, behavior modification, aerobic exercise, and stress management. Handouts were designed for low-literacy or non-English-speaking adults, and plastic food models were used to discuss serving sizes and portion control. Suggestions for healthful eating were based on WIC foods whenever possible. Exercise focused on aerobic dance because it can done at home with no special equipment or child care needed and can be adapted to almost any kind of music.

Domel SB, Alford BB, Cattlett HN, Gench BE. Weight control for ***black women.*** J Am Diet Assoc *1992;92(3):346-348.* [634]

A program for black women with low educational levels at four literacy program sites in economically disadvantaged areas of Dallas, Texas. The program consisted of 11 weekly sessions taught by a registered dietitian and included nutrition education, behavior modification, active learner participation, and self-responsibility. It stressed practical, lifelong methods for weight control. Materials were geared toward low-literacy participants; for example, a green traffic light was used for low-calorie foods, and a yellow traffic light was used for high-calorie foods. Shopping tips included ways to save money and decrease calories; audiocassettes included a radio show format with disk jockey, guest speaker, and "phoned-in" questions from listeners.

Domel SB, Alford BB, Cattlett HN, Rodriguez ML, Gench BE. A pilot weight control program for ***Hispanic women.*** J Am Diet Assoc *1992 Oct;92(10):1270-1.* [635]

A 10-session pilot weight control program for Hispanic women in Dallas, Texas. Modifications to fit the Hispanic culture included adding appropriate ethnic foods and recipes, stressing the importance of health for the entire family, and reformatting some of the materials. To this end, some of the pamphlets were changed to a comic book format, and audiocassetes were changed from a radio show format to a dialog between two *comadres.* The program was taught in Spanish by a bilingual Hispanic dietitian at a local church. Handouts were available in English and Spanish. Participants were recruited through articles in Spanish and English newspapers, public service announcements on Spanish radio stations, church bulletins, and various flyers. A promotional event was also held at each site 1 week before the program began.

Fox RA, Haniotes H, Rotatori A. A streamlined weight loss program for ***moderately retarded adults*** *in a sheltered workshop setting.* Appl Res Ment Retard *1984;5(1):69-79.* [636]

A 15-week behavioral weight loss program for obese, moderately retarded adults that included 10 weeks of treatment and 5 weeks of maintenance. The program was conducted in the sheltered workshop where all of the participants worked, and participants were given time off during the day to attend sessions. Picture materials were developed to facilitate the learning and

visited on 6/14/2019

practice of the behavioral weight loss strategies; no reading skills were required. All participants in the study lived with their parents, and all parents were instructed in the behavioral weight reduction techniques and the forms so that they could provide guidance and support to their child at home during the course of the program. Daily homework assignments were given so that participants could practice behavioral strategies outside of the training setting. Bimonthly phone calls to the parents were made to discuss their child's progress and difficulties in implementing behavior techniques. To encourage attendance, at each session, participants were entered into a drawing to win small prizes, and awards were also given for each pound of weight lost. Follow-up was conducted for 1 year.

Glasgow RE, Toobert DJ, Hampson SE, Brown JE, Lewinsohn PM, Donnely J. Improving self-care among ***older patients with type 2 diabetes.*** *The "Sixty-Something..." Study. Patient Educ Couns 1992;19:61-74.* [482]

A 10-session, self-management training program designed for persons older than 60 years of age. Recruitment was conducted through local diabetes care professionals, presentations, mailings, newsletters of local and State diabetes associations, presentations at health fairs, flyers distributed at grocery stores and pharmacies, and local television and radio advertising. The program was offered at no cost, and participants were provided glucose testing materials and a coupon for a pair of walking shoes. Sessions were held during daylight hours in well-known, accessible, and pleasant facilities that earlier focus group participants thought were most convenient. In response to focus group feedback, the program focused on dietary and exercise self-care behaviors. The group was led by an interdisciplinary team of psychologists, a registered dietitian, certified exercise leaders, and other educators. Group meetings were kept small and focused on developing individualized plans to overcome barriers to adherence. Participants chose the specific self-care area on which they wished to focus. Follow-up was conducted for 6 months.

Heath GW, Wilson RH, Smith J, Leonard BE. Community-based exercise and weight control. Diabetes risk reduction and glycemic control in ***Zuni Indians.*** Am J Clin Nutr *1991;53:1642S-1646S.* [637]

A 2-year exercise intervention in the Zuni Indian community in western New Mexico. Participants were recruited through personal invitation, recommendation from the medical staff, and a general community advertising campaign. Community events such as foot races were offered throughout the year, supported by community agencies and local businesses. The program was coordinated by a health educator, two health education assistants, and 48 Zuni Indians who were trained in exercise and group leadership.

Kanders BS, Ullmann-Joy P, Foreyt JP, Heymsfield SB, Heber D, Elashoff RM, Ashley JM, Reeves RS, Blackburn GL. The black American lifestyle intervention (BALI): The design of a weight loss program for ***working-class African-American women.*** J Am Diet Assoc *1994;94(3):310-312.* [638]

A 10-week weight loss program for obese, low-income African-American women ages 40 to 64 years in Boston, New York, Houston, and Los Angeles. The program focused on diet, exercise, and behavior modification using information obtained from interviews of obese African-American women concerning these issues. Participants were placed on a culturally appropriate, low-fat diet, including two free meal replacement shakes. Lactaid was provided to those participants who were lactose intolerant. Group sessions involving goal-setting, problem-solving, and role-playing were led by a female African-American nutritionist. All educational materials, recipes, and menu plans were reviewed by minority health professionals to ensure cultural appropriateness.

visited on 6/14/2019

Kumanyika SK, Brancato J, Brewer A, Carnaghi M, Doroshenko LH, Rosen R, Rosofsky W, Self MS. Interventions in the Trials of Nonpharmacologic Intervention in the Elderly. Effective approaches to weight and sodium reduction among ***older adults.*** Circulation *1996;94(8):I-690.* [639]

The Trials of Nonpharmacologic Intervention in the Elderly (TONE) was a randomized comparison of weight reduction, sodium reduction, and combined weight/sodium reduction programs versus a no-treatment control group conducted in four university research centers located in Maryland, North Carolina, New Jersey, and Tennessee. Education and counseling addressed relevant nutrition knowledge and behavior change skills. Contacts were weekly for 4 months, biweekly for 4 months, and then monthly. The study addressed challenges that an older population faces, such as literacy limitations, and used oral and visual examples to explain new concepts; concrete, step-by-step instruction; limited use of complex sentences; and minimal need for abstract reasoning. Only a small amount of information was given at one time to prevent overload. Easy-to-read typeface was used, as well as generous use of graphics. Physical changes of aging (e.g., impaired hearing, vision, or memory and speed of absorbing new information) were addressed by using simultaneous oral and visual presentation and seating and room arrangements to accommodate those who wished to be close to the speaker. Large typeface was used in all visual materials, and the room was arranged to avoid glare. Graphics and text references were chosen to depict older persons, and counseling was geared to build on previously learned nutrition practices, preferences, and food-related attitudes.

Kumanyika SK, Charleston JB. Lose weight and win: A church-based weight loss program for blood pressure control among ***black women.*** Patient Educ Couns *1992;19(1):19-32.* [640]

A behaviorally oriented weight control program offering eight weekly diet counseling/exercise sessions. The program was a component of the Baltimore Church High Blood Pressure Program (CHBPP), which encompassed black churches in the Baltimore, Maryland, black community. Recruitment was through CHBPP networks, including lay volunteers from each congregation who had also been trained as blood pressure measurement specialists. Advertisement of the program was by word of mouth as well as by presentations at meetings of the ministerial alliance. Announcements were also placed in church bulletins. The meetings were held on church property, often following other scheduled church activities such as choir practice. The group format emphasized an unrushed, supportive, relaxed environment and included a diet component led by a registered dietitian and an exercise component consisting of low-impact aerobics.

Lasco RA, Curry RH, Dickson VJ, Powers J, Menes S, Merritt RK. Participation rates, weight loss, and blood pressure changes among ***obese women*** *in a nutrition-exercise program.* Public Health Rep *1989;104(6):640-646.* [641]

A 10-week exercise and nutrition intervention targeted to obese residents of a **black urban community** in Atlanta between the ages of 18 and 59 years. Free transportation and child care were provided to encourage participation. Classes on nutrition, community resources, and exercise were offered, including low-impact aerobic dance, water exercises, and walking. Home visits by a public health educator were planned to build family support and alleviate spousal concerns. Participants were asked to comment on the program, and their suggestions were incorporated into the special interest sessions. Participation was monitored, and absentees were called and encouraged to attend the next session.

visited on 6/14/2019

McNabb W, Quinn M, Kerver J, Cook S, Karrison T. The Pathways church-based weight loss program for urban ***African-American women at risk for diabetes.*** Diabetes Care *1997;20:1518-1523.* [642]

A randomized, 14-week, church-based weight loss program targeting urban, obese African-American women. The program was developed using locally conducted focus groups with the target population and administered by trained lay facilitators recruited from urban churches. Facilitators assisted participants in identifying and providing solutions for their dietary problems. Aspects of the program included emphasizing weight loss for general well-being and health rather than improved physical appearance, achieving culturally appropriate body size, and using ethnic foods and food combinations.

McNabb WL, Quinn MT, Rosing L. Weight loss program for ***inner-city black women*** *with non-insulin-dependent diabetes mellitus: PATHWAYS.* J Am Diet Assoc *1993;93(1):75-77.* [643]

An 18-week weight loss program, including 12 core weekly sessions that focused on nutrition information and behavior modification and 6 follow-up or "reinforcement" sessions that provided support and strategies for overcoming obstacles the women faced along the way. The program was designed to deal with the obstacles obese, inner-city black women face in trying to exercise and lose weight and was partly based on feedback from a sample of women who were interviewed before the study was designed. An expert panel composed of local minority health care professionals reviewed program materials, which were written at a low-literacy level. Participants were provided with information about foods, including ethnic and regional foods such as greens, salt pork, and ham hocks.

Mount MA, Kendrick OW, Draughon M, Stitt KR, Head D, Mount R. Group participation as a method to achieve weight loss and blood glucose control. J Nutr Educ *1991;23:25-29.* [644]

A biweekly, 10-session nutrition, diabetes, and weight loss education program with 30 **black adults in rural west Alabama**. The program used self-help groups to assist participants with diabetes management by providing peer interaction and support. Groups were led by a public health nutritionist, nutrition graduate student, and a lay facilitator chosen from each group. Topics included definition of diabetes, meal planning, diet modification to reduce complications, and weight control.

Mulrow C, Bailey S, Sönksen PH, Slavin B. Evaluation of an ***audiovisual diabetes education*** *program. Negative results of a randomized trial of patients with non-insulin dependent diabetes mellitus.* J Gen Intern Med *1987;2:215-219.* [645]

A randomized, controlled trial conducted at a hospital in central London. The intervention focused on improving glucose and weight control in **low-income, low-literacy obese patients** with non-insulin dependent diabetes. The trial excluded those patients who were taking insulin and patients who were older than 70 years of age. Of the 120 patients recruited, 55 percent were female, and 49 percent were **West Indian black**. Patients within the intervention group participated in monthly group sessions that included materials specifically designed for diabetic patients with low literacy or monthly group sessions without low-literacy materials. Low-literacy sessions used standardized audiovisual lessons and written materials and included common West Indian foods.

Pleas J. Long-term effects of a lifestyle-change obesity treatment program with ***minorities.*** J Natl Med Assoc *1988;80(7):747-752.* [646]

A 12-week weight management program with lifestyle change as its central focus (n=12) located at a neighborhood YMCA. Recruiting was conducted through articles and announcements

visited on 6/14/2019

in local newspapers and church bulletins. The weekly sessions lasted 2 hours, with the first hour devoted to lectures on nutrition, behavioral change, and weight loss; the second hour involved exercise, which included endurance exercises and walking.

Shintani TT, Hughes CK, Beckham S, O'Connor HK. Obesity and cardiovascular risk intervention through the ad libitum feeding of ***traditional Hawaiian diet****.* Am J Clin Nutr *1991;53:1647S-1651S.* [647]

Under the premise that, historically, **Native Hawaiians** have been thin due to their traditional diet, a diet intervention for Native Hawaiians was conducted with traditional Hawaiian foods on the western coast of the island of Oahu. Recruitment was conducted through newspaper advertisements, articles, flyers, and a public presentation about the program. The selection of food for the program consisted of foods available in Hawaii before Western contact, such as taro, poi, sweet potatoes, yams, breadfruit, greens, fruit, seaweed, fish, and chicken. Foods were prepared in the traditional manner. Breakfast was eaten together, and cultural or health education sessions were conducted during the group dinner session.

Sullivan J, Carter JP. A nutrition-physical fitness intervention program for ***low-income black parents.*** J Natl Med Assoc *1985;77(1):39-43.* [648]

An 8-week intervention for obese, low-income black mothers (n=10) of children younger than 3 years was conducted at the Parent Child Development Center of the Urban League in New Orleans, Louisiana. Aerobic exercise with soul music was incorporated into the sessions because it was thought to appeal to the population and because dancing can be done safely in the home with no special equipment. Child care was provided for the mothers during the sessions.

4 Appendix IV. Obesity and Sleep Apnea

Obstructive sleep apnea is defined as an absence of breathing during sleep. Currently, it is recognized that sleep apnea is part of a continuum from health to disease. [649, 650] Apnea is currently defined as cessation of airflow for at least 10 seconds and is characterized as either central (if no respiratory effort occurs), obstructive (if continued effort is noted), or mixed (if both central and obstructive components are present). [651-653] Apnea is associated with either a fall in oxyhemoglobin desaturation or an arousal from sleep. Hypopneas, which are defined as partial reductions in airflow associated with falls in oxygen saturation or arousals from sleep, are also recorded.

The sleep apnea syndrome has been clinically defined as recurrent apnea or hypopnea associated with clinical impairment usually manifested as increased daytime sleepiness or altered cardiopulmonary function. In general, the average number of episodes of apnea and hypopnea per hour are reported as an index (AHI) or as a respiratory disturbance index (RDI). Classically, an AHI of greater than five episodes per hour has been the definition of the presence of the sleep apnea syndrome. [651, 652] Other commonly used cutoff points are an AHI of 10 or 15 episodes per hour or an overnight total of 30 apneic-hypopneic episodes. [653]

There are several correlates of sleep apnea. It is well recognized that it is more prevalent in males, although the difference is less pronounced in population-based studies than in laboratory-based studies. [20] Some studies have suggested that the prevalence of sleep apnea in women increases after menopause. Snoring also correlates with sleep apnea, increasing up to late middle age and decreasing thereafter. [20, 282, 654, 655] The other major correlate of sleep apnea is obesity [20, 653, 654, 656] in both men and women. In general, women have to be significantly more obese than men for the clinical syndrome to be apparent. [657] At present, no published epidemiologic studies

visited on 6/14/2019

have examined the relationship between race and sleep apnea. Given the high prevalence of obesity among specific populations and minorities, sleep apnea may be highly prevalent in these groups.

The major pathophysiologic consequences of severe sleep apnea include severe arterial hypoxemia, recurrent arousals from sleep, increased sympathetic tone, pulmonary and systemic hypertension, and cardiac arrhythmias. [658] These phenomena may result in acute hemodynamic and chronic structural change in the coronary arteries, possibly associated with relative myocardial ischemia, rupture of atheromatous plaques, and increased risk for thrombosis at the site of any unstable plaque. [659] Similar mechanisms acting in the cerebrovascular system may be involved in an increased risk of stroke. Finally, the sympathetic tone may be associated with hypertension as well as increased platelet aggregability. [660]

The primary goal for treatment of individuals with sleep apnea is to reduce the severity of the respiratory events that are associated with oxyhemoglobin desaturation and arousal from sleep. There are only two cohort studies in clinic-based populations that demonstrate a significant reduction in cardiovascular mortality among sleep apnea patients who are treated progressively compared to those who are treated conservatively. [661, 662] However, both of these studies were retrospective and suffered from a significant bias of ascertainment.

The evidence that treatment of obesity ameliorates obstructive sleep apnea is reasonably well established. Although the studies are small, both surgical and medical approaches to weight loss have been associated with a consistent but variable reduction in the number of respiratory events, as well as improvement in oxygenation. In general, surgical interventions, which have included a gastric bypass or jejunoileostomy, have been reserved for people who are severely obese. [663-666] While this approach to weight reduction was commonly used in patients with severe obstructive sleep apnea, they recently have been abandoned because of complications and side effects. However, the use of surgical gastric procedures has been successful in improving sleep apnea in a number of studies.

On the other hand, weight reduction has been associated with comparable reduction in the severity of sleep apnea, as well as improved evidence of renal function and hypertension [667-671] Both medical and surgical studies have demonstrated that as little as 10-percent weight reduction is associated with a more than 50-percent reduction in the severity of sleep apnea. Moreover, more recent data suggest a possible "threshold" effect that is directly related to the collapsibility of the upper airway. Those individuals who demonstrate a minimally collapsible upper airway apparently achieve a greater effect for the same percentage of weight reduction. [672]

Finally, there is evidence that standard treatments for sleep apnea do reduce specific cardiovascular risk factors. Specifically, the most commonly employed treatment, continuous positive airway pressure, has been shown to reduce waking arresting carbon dioxide and reduced heart rate, and pulmonary artery pressure decreased hematocrit and improved ventricular ejection fraction. [673] Other standard surgical approaches that have been employed to widen the upper airway have been shown to reduce the severity of the apnea, but there are no data examining the associated cardiovascular risk factors. No studies have specifically examined the effects of treatment of sleep apnea on obesity, but it generally has been noted that all nonweight loss treatments of sleep apnea have not been associated with any significant weight loss other than might be accrued from surgical interventions that temporarily reduce the ability to eat in the immediate postoperative period.

visited on 6/14/2019

5 Appendix V. Body Mass Index—How to Measure Obesity

Body mass index (BMI) is usually measured with the Quetelet index as follows: weight divided by height squared (W/H^2 [kg/m^2]).

To use the table, find the appropriate height in the left-hand column. Move across to a given weight. The number at the top of the column is the BMI at that height and weight. Pounds have been rounded off. [758]

Body Mass Index

Height (inches)	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35
	Body Weight (pounds)																
58	91	96	100	105	110	115	119	124	129	134	138	143	148	153	158	162	167
59	94	99	104	109	114	119	124	128	133	138	143	148	153	158	163	168	173
60	97	102	107	112	118	123	128	133	138	143	148	153	158	163	168	174	179
61	100	106	111	116	122	127	132	137	143	148	153	158	164	169	174	180	185
62	104	109	115	120	126	131	136	142	147	153	158	164	169	175	180	186	191
63	107	113	118	124	130	135	141	146	152	158	163	169	175	180	186	191	197
64	110	116	122	128	134	140	145	151	157	163	169	174	180	186	192	197	204
65	114	120	126	132	138	144	150	156	162	168	174	180	186	192	198	204	210
66	118	124	130	136	142	148	155	161	167	173	179	186	192	198	204	210	216
67	121	127	134	140	146	153	159	166	172	178	185	191	198	204	211	217	223
68	125	131	138	144	151	158	164	171	177	184	190	197	203	210	216	223	230
69	128	135	142	149	155	162	169	176	182	189	196	203	209	216	223	230	236
70	132	139	146	153	160	167	174	181	188	195	202	209	216	222	229	236	243
71	136	143	150	157	165	172	179	186	193	200	208	215	222	229	236	243	250
72	140	147	154	162	169	177	184	191	199	206	213	221	228	235	242	250	258
73	144	151	159	166	174	182	189	197	204	212	219	227	235	242	250	257	265
74	148	155	163	171	179	186	194	202	210	218	225	233	241	249	256	264	272
75	152	160	168	176	184	192	200	208	216	224	232	240	248	256	264	272	279
76	156	164	172	180	189	197	205	213	221	230	238	246	254	263	271	279	287

visited on 6/14/2019

Body Mass Index

Height (inches)	Body Weight (pounds)																		
	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54
58	172	177	181	186	191	196	201	205	210	215	220	224	229	234	239	244	248	253	258
59	178	183	188	193	198	203	208	212	217	222	227	232	237	242	247	252	257	262	267
60	184	189	194	199	204	209	215	220	225	230	235	240	245	250	255	261	266	271	276
61	190	195	201	206	211	217	222	227	232	238	243	248	254	259	264	269	275	280	285
62	196	202	207	213	218	224	229	235	240	246	251	256	262	267	273	278	284	289	295
63	203	208	214	220	225	231	237	242	248	254	259	265	270	278	282	287	293	299	304
64	209	215	221	227	232	238	244	250	256	262	267	273	279	285	291	296	302	308	314
65	216	222	228	234	240	246	252	258	264	270	276	282	288	294	300	306	312	318	324
66	223	229	235	241	247	253	260	266	272	278	284	291	297	303	309	315	322	328	334
67	230	236	242	249	255	261	268	274	280	287	293	299	306	312	319	325	331	338	344
68	236	243	249	256	262	269	276	282	289	295	302	308	315	322	328	335	341	348	354
69	243	250	257	263	270	277	284	291	297	304	311	318	324	331	338	345	351	358	365
70	250	257	264	271	278	285	292	299	306	313	320	327	334	341	348	355	362	369	376
71	257	265	272	279	286	293	301	308	315	322	329	338	343	351	358	365	372	379	386
72	265	272	279	287	294	302	309	316	324	331	338	346	353	361	368	375	383	390	397
73	272	280	288	295	302	310	318	325	333	340	348	355	363	371	378	386	393	401	408
74	280	287	295	303	311	319	326	334	342	350	358	365	373	381	389	396	404	412	420
75	287	295	303	311	319	327	335	343	351	359	367	375	383	391	399	407	415	423	431
76	295	304	312	320	328	336	344	353	361	369	377	385	394	402	410	418	426	435	443

To use the table, find the appropriate height in the left-hand column. Move across to a given weight. The number at the top of the column is the BMI at that height and weight. Pounds have been rounded off.[758]

visited on 6/14/2019

6 Appendix VI. Practical Dietary Therapy Information

A.1. Shopping—What To Look For
a. Low-Calorie Shopping List
b. Low-Calorie, Lower-Fat Alternatives
c. Fat-Free Versus Regular—Calorie Comparisons
d. Good Sources of Calcium

A.2. Food Preparation—What To Do
■ Low-Calorie, Low-Fat Cooking/Serving Methods

A.3. Dining Out—How To Choose
a. General Tips for Healthy Dining Out
b. Tips for Healthy Multicultural Dining Out

A.4. Sample Reduced-Calorie Menus
a. Traditional American Cuisine—Reduced Calorie
b. Asian-American Cuisine—Reduced Calorie
c. Southern Cuisine—Reduced Calorie
d. Mexican-American Cuisine—Reduced Calorie
e. Lacto-Ovo Vegetarian Cuisine—Reduced Calorie

VI. A.1. Shopping—What To Look For

A.1.a. Low-Calorie Shopping List

Make a shopping list. Include the items you need for your menus and any low-calorie basics you need to restock in your kitchen.

Dairy case

- ❏ Low-fat (1%) or fat-free (skim) milk
- ❏ Low-fat or reduced-fat cottage cheese
- ❏ Fat-free cottage cheese
- ❏ Low-fat cheeses
- ❏ Low-fat or nonfat yogurt
- ❏ Light or diet margarine (tub, squeeze or spray)
- ❏ Reduced-fat or fat-free sour cream
- ❏ Fat-free cream cheese
- ❏ Eggs/Egg substitutes
- ❏ _____
- ❏ _____
- ❏ _____

Breads, muffins, rolls

- ❏ Bread, bagels, pita bread
- ❏ English muffins
- ❏ Yeast breads (whole wheat, rye, pumpernickel, multi-grain, raisin)
- ❏ Corn tortillas (not fried)
- ❏ Low-fat flour tortillas
- ❏ Fat-free biscuit mix
- ❏ Rice crackers
- ❏ Challah
- ❏ _____
- ❏ _____

Cereals, crackers, rice, noodles, and pasta

- ❏ Plain cereal, dry or cooked
- ❏ Saltines, soda crackers (low sodium or unsalted tops)
- ❏ Graham crackers
- ❏ Other low-fat crackers
- ❏ _____
- ❏ _____
- ❏ _____
- ❏ Rice (brown, white, etc.)

visited on 6/14/2019

- ❏ Pasta (noodles, spaghetti)
- ❏ Bulgur, couscous, kasha
- ❏ Potato mixes (made without fat)
- ❏ Rice mixes (made without fat)
- ❏ Other
- ❏ Wheat mixes
- ❏ Tabouli grain salad
- ❏ Hominy
- ❏ Polenta
- ❏ Polvillo
- ❏ Hominy grits
- ❏ Quinoa
- ❏ Millet
- ❏ Aramanth
- ❏ Oatmeal
- ❏ _____
- ❏ _____

Meat case

- ❏ White meat chicken and turkey (skin-off)
- ❏ Fish (not battered)
- ❏ Beef, round or sirloin
- ❏ Extra lean ground beef such as ground round
- ❏ Pork tenderloin
- ❏ 95% fat-free lunch meats or low-fat deli meats
- ❏ Meat equivalents:
  - ❏ Tofu (or bean curd)
  - ❏ Beans (see bean list)
  - ❏ Eggs/egg substitutes (see dairy list)
  - ❏ _____
  - ❏ _____
  - ❏ _____

Fruit (fresh, canned, and frozen)

Fresh Fruit:

- ❏ Apples
- ❏ Bananas
- ❏ Peaches
- ❏ Oranges
- ❏ Pears
- ❏ Grapes
- ❏ Grapefruit
- ❏ Apricot
- ❏ Dried Fruits
- ❏ Cherries
- ❏ Plums
- ❏ Melons
- ❏ Lemons
- ❏ Limes
- ❏ Plantains
- ❏ Mango
- ❏ Papaya
- ❏ _____
- ❏ _____

Exotic Fresh Fruit:

- ❏ Kiwi
- ❏ Olives
- ❏ Figs
- ❏ Quinces
- ❏ Currants
- ❏ Persimmons
- ❏ Pomegranates
- ❏ Anon
- ❏ Caimito
- ❏ Chirimoya

visited on 6/14/2019

- ❏ Guanabana
- ❏ Mamey
- ❏ Papayas
- ❏ Zapote
- ❏ Guava
- ❏ Starfruit
- ❏ Ugli fruit
- ❏ Dried pickled plums
- ❏ Litchee nuts
- ❏ Winter melons
- ❏ _____
- ❏ _____

Canned Fruit (in juice or water):

- ❏ Canned pineapple
- ❏ Applesauce
- ❏ Other canned fruits (mixed or plain)
- ❏ _____

Frozen Fruits (without added sugar):

- ❏ Frozen blueberries
- ❏ Frozen raspberries
- ❏ Frozen 100% fruit juice
- ❏ _____

Dried Fruit:

- ❏ Raisins/dried fruit (these tend to be higher in calories than fresh fruit)

Vegetables (fresh, canned, frozen)

Fresh Vegetables:

- ❏ Broccoli
- ❏ Peas
- ❏ Corn
- ❏ Cauliflower
- ❏ Squash
- ❏ Green Beans
- ❏ Green Leafy Vegetables
- ❏ Spinach
- ❏ Lettuce
- ❏ Cabbage
- ❏ Artichokes
- ❏ Cucumber
- ❏ Asparagus
- ❏ Mushrooms
- ❏ Carrots or celery
- ❏ Onions
- ❏ Potatoes
- ❏ Tomatoes
- ❏ Green peppers
- ❏ Chilies
- ❏ Tomatillos
- ❏ _____

Canned Vegetables: (low sodium or no salt added)

- ❏ Canned tomatoes
- ❏ Tomato sauce or pasta
- ❏ Other canned vegetables
- ❏ Canned vegetable soup, reduced sodium

Frozen Vegetables: (without added fats)

- ❏ Frozen broccoli
- ❏ Frozen spinach
- ❏ Frozen mixed medley, etc.
- ❏ Frozen yucca
- ❏ _____

Exotic Fresh Vegetables

- ❏ Okra

visited on 6/14/2019

- ❏ Dandelions
- ❏ Eggplant
- ❏ Grape leaves
- ❏ Mustard greens
- ❏ Kale
- ❏ Leeks
- ❏ Boniato
- ❏ Chayote
- ❏ Borenjena
- ❏ Plaintain
- ❏ Cassava
- ❏ Prickly pear cactus
- ❏ Bamboo shoots
- ❏ Chinese celery
- ❏ Water chestnuts
- ❏ Bok choy
- ❏ Burdock root
- ❏ Napa cabbage
- ❏ Taro
- ❏ Seaweed
- ❏ Bean sprouts
- ❏ Amaranth
- ❏ Choy sum
- ❏ Calabacita
- ❏ Sea vegetables
- ❏ Rhubarb
- ❏ _____

Beans and legumes (if canned, no salt added)

- ❏ Lentils
- ❏ Black beans
- ❏ Red beans (kidney beans)
- ❏ Navy beans
- ❏ Black beans
- ❏ Pinto beans
- ❏ Blackeyed peas
- ❏ Fava beans
- ❏ Mung beans
- ❏ Italian white beans
- ❏ Great white northern beans
- ❏ Chickpeas (garbanzo beans)
- ❏ Dried beans, peas, and lentils (without flavoring packets)
- ❏ Canned bean soup

Baking items

- ❏ Flour
- ❏ Sugar
- ❏ Imitation butter (flakes or buds)
- ❏ Nonstick cooking spray
- ❏ Canned evaporated milk—fat free (skim) or reduced-fat (2%)
- ❏ Nonfat dry milk powder
- ❏ Cocoa powder, unsweetened
- ❏ Baking powder
- ❏ Baking soda
- ❏ Cornstarch
- ❏ Unflavored gelatin
- ❏ Gelatin, any flavor (reduced calorie)
- ❏ Pudding mixes (reduced calorie)
- ❏ Angel food cake mix
- ❏ Other low-fat mixes
- ❏ Other
- ❏ _____
- ❏ _____

visited on 6/14/2019

Frozen foods

- ❏ Frozen fish fillets— unbreaded
- ❏ Egg substitute
- ❏ Frozen 100 percent fruit juices (no sugar added)
- ❏ Frozen fruits (no sugar added)
- ❏ Frozen vegetables (plain)
- ❏ Other frozen foods
- ❏ _____

Condiments, sauces, seasonings, and spreads

- ❏ Low-fat or nonfat salad dressings
- ❏ Mustard (Dijon, etc.)
- ❏ Catsup
- ❏ Barbecue sauce
- ❏ Other low-fat sauces
- ❏ _____
- ❏ _____
- ❏ Jam, jelly, or honey
- ❏ Spices _____
- ❏ _____
- ❏ Flavored vinegars
- ❏ Hoisin sauce, plum sauce
- ❏ Salsa or picante sauce
- ❏ Canned green chilies
- ❏ Soy sauce (low sodium)
- ❏ Bouillon cubes/granules (low sodium)
- ❏ Other
- ❏ _____

Beverages

- ❏ No-calorie drink mixes
- ❏ Reduced-calorie juices
- ❏ Unsweetened iced tea
- ❏ Carbonated water
- ❏ Water

visited on 6/14/2019

Low-Calorie Shopping List

We live in a fast-moving world. To reduce the time you spend in the kitchen you can improve your organization by using a shopping list and keeping a well-stocked kitchen. Shop for quick low-fat food items, and fill your kitchen cupboards with a supply of low-calorie basics.

Read labels as you shop. Pay attention to the serving size and the servings per container. All labels list total calories in a serving size of the product. **Compare the total calories in the product you choose with others like it; choose the one that is lowest in calories.** Below is a label that identifies important information.

Product:

Nutrition Facts

Serving Size 1 cup (228g)
Servings Per Container 2

Amount Per Serving
Calories 250 Calories from Fat 110

	% Daily Value*
Total Fat 12g	18%
Saturated Fat 3g	15%
Cholesterol 30mg	10%
Sodium 470mg	20%
Total Carbohydrate 31g	10%
Dietary Fiber 0g	0%
Sugars 5g	
Protein 5g	

Vitamin A 4% • Vitamin C 2%
Calcium 20% • Iron 4%

* Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs:

	Calories:	2,000	2,500
Total Fat	Less than	65g	80g
Sat Fat	Less than	20g	25g
Cholesterol	Less than	300mg	300mg
Sodium	Less than	2,400mg	2,400mg
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

Calories per gram:
Fat 9 • Carbohydrates 4 • Protein 4

Check for:

- Serving size
- Number of servings

- Calories
- Total fat in grams
- Saturated fat in grams
- Cholesterol in milligrams
- Sodium in milligrams

Here, the label gives the amounts for the different nutrients in one serving. Use it to help you keep track of how many calories, fat, saturated fat, cholesterol, and sodium you are getting from different foods.

- The "% Daily Value" shows you how much of the recommended amounts the food provides in one serving, if you eat 2,000 calories a day. For example, one serving of this food gives you 18 percent of your total fat recommendation.

- Here you can see the recommended daily amount for each nutrient for two calorie levels. If you eat a 2,000 calorie diet, you should be eating less than 65 grams of fat and less than 20 grams of saturated fat. If you eat 2,500 calories a day, you should eat less than 80 grams of fat and 25 grams of saturated fat. Your daily amounts may vary higher or lower depending on the calories you eat.

visited on 6/14/2019

VI. A.1.b. Low-Calorie, Lower-Fat Alternatives

These low-calorie alternatives provide new ideas for old favorites. When making a food choice, remember to consider vitamins and minerals. Some foods provide most of their calories from sugar and fat but give you few if any vitamins and minerals.

This guide is not meant to be an exhaustive list. We stress reading labels to find out just how many calories are in the specific products you decide to buy.

HIGHER-FAT FOODS	LOWER-FAT FOODS
Dairy Products	
❑ Evaporated whole milk	❑ Evaporated fat-free (skim) or reduced-fat (2%) milk
❑ Whole milk	❑ Low-fat (1%), reduced-fat (2%), or fat-free (skim) milk
❑ Ice cream	❑ Sorbet, sherbet, low-fat or fat-free frozen yogurt, or ice milk (check label for calorie content)
❑ Whipping cream	❑ Imitation whipped cream (made with fat-free [skim] milk) or low-fat vanilla yogurt
❑ Sour cream	❑ Plain low-fat yogurt
❑ Cream cheese	❑ Neufchatel or "light" cream cheese or fat-free cream cheese
❑ Cheese (Cheddar, Swiss, Jack)	❑ Reduced-calorie cheese, low-calorie processed cheeses, etc.
❑ American cheese	❑ Fat-free cheese
❑ Regular (4%) cottage cheese	❑ Fat-free American cheese or other types of fat-free cheeses ❑ Low-fat (1%) or reduced-fat (2%) cottage cheese
❑ Whole milk mozzarella cheese	❑ Part-skim milk, low-moisture mozzarella cheese
❑ Whole milk ricotta cheese	❑ Part-skim milk ricotta cheese
❑ Coffee cream (½ and ½) or nondairy creamer (liquid, powder)	❑ Low-fat (1%) or reduced-fat (2%) milk or non-fat dry milk powder
Cereals, Grains, and Pasta	
❑ Ramen noodles	❑ Rice or noodles (spaghetti, macaroni, etc.)
❑ Pasta with white sauce (alfredo)	❑ Pasta with red sauce (marinara)
❑ Pasta with cheese sauce	❑ Pasta with vegetables (primavera)
❑ Granola	❑ Bran flakes, crispy rice, etc.
	❑ Cooked grits or oatmeal
	❑ Reduced-fat granola

visited on 6/14/2019

HIGHER-FAT FOODS	LOWER-FAT FOODS
	Meat, Fish, and Poultry
❑ Coldcuts or lunch meats (bologna, salami, liverwurst, etc.)	❑ Low-fat coldcuts (95 to 97% fat-free lunch meats, low-fat pressed meats)
❑ Hot dogs (regular)	❑ Lower-fat hot dogs
❑ Bacon or sausage	❑ Canadian bacon or lean ham
❑ Regular ground beef	❑ Extra lean ground beef such as ground round or ground turkey (read labels)
❑ Chicken or turkey with skin; duck, or goose	❑ Chicken or turkey without skin (white meat)
❑ Oil-packed tuna	❑ Water-packed tuna (rinse to reduce sodium content)
❑ Beef (chuck, rib, brisket)	❑ Beef (round, loin) (trimmed of external fat) (choose select grades)
❑ Pork (spareribs, untrimmed loin)	❑ Pork tenderloin or trimmed, lean smoked ham
❑ Frozen breaded fish or fried fish (homemade or commercial)	❑ Fish or shellfish, unbreaded (fresh, frozen, canned in water)
❑ Whole eggs	❑ Egg whites or egg substitutes
❑ Frozen TV dinners (containing more than 13 grams of fat per serving)	❑ Frozen TV dinners (containing less than 13 grams of fat per serving and lower in sodium)
❑ Chorizo sausage	❑ Turkey sausage, drained well (read label)
	❑ Vegetarian sausage (made with tofu)
	Baked Goods
❑ Croissants, brioches, etc.	❑ Hard french rolls or soft brown 'n serve rolls
❑ Donuts, sweet rolls, muffins, scones, or pastries	❑ English muffins, bagels, reduced-fat or fat-free muffins or scones
❑ Party crackers	❑ Low-fat crackers (choose lower in sodium)
	❑ Saltine or soda crackers (choose lower in sodium)
❑ Cake (pound, chocolate, yellow)	❑ Cake (angel food, white, gingerbread)
❑ Cookies	❑ Reduced-fat or fat-free cookies (graham crackers, ginger snaps, fig bars) (compare calorie level)

visited on 6/14/2019

HIGHER-FAT FOODS	LOWER-FAT FOODS
Snacks and Sweets	
❑ Nuts	❑ Popcorn (air-popped or light microwave), fruits, vegetables
❑ Ice cream, e.g., cones or bars	❑ Frozen yogurt, frozen fruit or chocolate pudding bars
❑ Custards or puddings (made with whole milk)	❑ Puddings (made with skim milk)
Fats, Oils, and Salad Dressings	
❑ Regular margarine or butter	❑ Light spread margarines, diet margarine, or whipped butter, tub or squeeze bottle
❑ Regular mayonnaise	❑ Light or diet mayonnaise or mustard
❑ Regular salad dressings	❑ Reduced-calorie or fat-free salad dressings, lemon juice, or plain or herb flavored or wine vinegar
❑ Butter or margarine on toast or bread	❑ Jelly, jam, or honey on bread or toast
❑ Oils, shortening, or lard	❑ Nonstick cooking spray for stir-frying or sautéing
	❑ As a substitute for oil or butter, use applesauce or prune puree in baked goods
Miscellaneous	
❑ Canned cream soups	❑ Canned broth-based soups
❑ Canned beans and franks	❑ Canned baked beans in tomato sauce
❑ Gravy (homemade with fat and/or milk)	❑ Gravy mixes made with water or homemade with the fat skimmed off and fat-free milk
❑ Fudge sauce	❑ Chocolate syrup
❑ Avocado on sandwiches	❑ Cucumber slices or lettuce leaves
❑ Guacamole dip or refried beans with lard	❑ Salsa

visited on 6/14/2019

VI. A.1.c. Fat-Free Versus Regular—Calorie Comparison

A calorie is a calorie is a calorie…whether it comes from fat or carbohydrate. Anything eaten in excess can lead to weight gain. You can lose weight by eating less calories and by increasing your physical activity. Reducing the amount of fat and saturated fat that you eat is one easy way to limit your overall calorie intake. However, eating fat-free or reduced-fat foods isn't always the answer to weight loss. For example, if you eat twice as many fat-free cookies as regular cookies you may not have reduced your overall calorie intake. The following list of foods and their fat-free varieties will show you that just because a product is fat-free, it doesn't mean that it is "calorie-free." And, calories do count!

Fat-Free or Reduced Fat	Calories	Regular	Calories
Reduced-Fat Peanut Butter, 2 tablespoons	190	Regular Peanut Butter, 2 tablespoons	190
Cookies:		Cookies:	
Reduced-Fat Chocolate Chip Cookie, 1 cookie	128	Regular Chocolate Chip Cookie, 1 cookie	136
Fat-Free Fig Cookie, 1 cookie	70	Fig Cookie, 1 cookie	50
Ice Cream:		Ice Cream:	
Premium Nonfat Frozen Yogurt ½ cup	190	Regular Ice Cream ½ cup	180
Premium Reduced-Fat Ice Cream ½ cup	190	Regular Ice Cream ½ cup	180
Fat-Free Caramel Topping, 2 tablespoons	130	Butterscotch Caramel Topping, 2 tablespoons	130
Reduced-Fat Granola Cereal, ¼ cup	110	Granola Cereal, ¼ cup	130
Reduced-Fat Croissant Roll, 1 roll	110	Regular Croissant Roll, 1 roll	130
Baked Tortilla Chips, 1 ounce	110	Regular Tortilla Chips, 1 ounce	130
Reduced-Fat Breakfast Bar, 1 bar	140	Breakfast Bar, 1 bar	130

visited on 6/14/2019

VI. A.1.d. Good Sources of Calcium

Calcium is not just for growing children. It is an important mineral that adults also need to keep their bones and teeth strong and their muscles functioning. Many people do not eat enough calcium everyday. The following is a list of good sources of calcium and tips on how to include more calcium in your diet everyday.

Source	Calcium (milligrams)
Milk (1 cup)	
Whole	300
2% reduced-fat	300
1%* low-fat	300
Fat free*	300
Yogurt* (1 cup)	
Plain, low-fat	415
Flavored, low-fat	315
Plain, fat free	315
Cheese (1 ounce)	
Reduced-fat Cheddar*	120
American	175
Swiss Cheese	270
Mozzarella, part-skim	185
Cottage Cheese (½ cup)	
2% reduced-fat	75
Calcium fortified cottage cheese	300
Ice Cream	
Regular, ½ cup	90
Low-fat, ½ cup	100
Frozen Yogurt	
Low-fat, ½ cup	100
Beans, dried cooked, 1 cup	90
Salmon, with bones, 3 ounces	205
Tofu, processed with calcium sulfate, ½ cup	435
Spinach, fresh cooked	244
Turnip Greens, fresh cooked, 1 cup	100
Kale, fresh cooked	94
Broccoli, fresh cooked	75
Waffle, 7” diameter	180
Pancakes, (2) 4” diameter	115
Pizza, with vegetables, ¼ 12” pie	180

* Low-fat and nonfat varieties of foods are still good sources of calcium.

visited on 6/14/2019

Calcium Requirements

Age	Women	Men
19-24	1,200 mg	1,200 mg
25-50	1,000 mg	800 mg

Tips for Fitting in Calcium

- Eat cereal with fat-free milk. Try adding fresh fruit.
- Drink an extra glass of milk every day; try calcium-fortified milk.
- Spread calcium-fortified cottage cheese* on crackers or bagel. Add fresh fruit.
- Drink calcium-fortified orange juice.*
- Blend a yogurt smoothie with low-fat or fat-free yogurt and milk, and fresh or frozen fruit.
- Make instant pudding with low-fat or fat-free milk.
- Choose frozen yogurt for dessert instead of cake or cookies.
- Add a slice of low-fat or fat-free cheese to sandwiches.
- Substitute calcium fortified tofu in stir-fries for chicken, shrimp, or beef.
- Sauté greens (kale, bok choy, collard greens) in cooking spray and lemon juice and herbs.

*Read food labels for products with added calcium

visited on 6/14/2019

VI. A.2. Food Preparation—What to Do

Low-Calorie, Low-Fat Cooking/ Serving Methods

Cooking low-calorie, low-fat dishes may not take a long time, but best intentions can be lost with the addition of butter or other added fats at the table. It is important to learn how certain ingredients can add unwanted calories and fat to low-fat dishes—making them no longer lower in calories and lower in fat! The following list provides examples of lower fat-cooking methods and tips on how to serve your low-fat dishes.

Low-Fat Cooking Methods

These cooking methods tend to be lower in fat:

- Bake
- Broil
- Microwave
- Roast—for vegetables and/or chicken without skin
- Steam
- Lightly stir-fry or sauté in cooking spray, small amounts of vegetable oil, or reduced sodium broth
- Grill seafood, chicken or vegetables

How To Save Calories and Fat

Look at the following examples for how to save calories and fat when preparing and serving foods. You might be surprised at how easy it is!

- Two tablespoons of butter on a baked potato can add an *extra* 200 calories and 22 grams of fat! However, ¼ cup salsa only adds 18 calories and no fat!
- Two tablespoons of regular clear Italian salad dressing will add an *extra* 136 calories and 14 grams of fat. Reduced fat Italian dressing only adds 30 calories and 2 grams of fat!

Try These Low-Fat Flavorings—added during preparation or at the table

- Herbs—oregano, basil, cilantro, thyme, parsley, sage, or rosemary
- Spices—cinnamon, nutmeg, pepper, or paprika
- Reduced-fat or fat-free salad dressing
- Mustard
- Catsup
- Fat-free or reduced-fat mayonnaise
- Fat-free or reduced-fat sour cream
- Fat-free or reduced-fat yogurt
- Reduced sodium soy sauce
- Salsa
- Lemon or lime juice
- Vinegar
- Horseradish
- Fresh ginger
- Sprinkle of butter flavor (not made with real butter)
- Red pepper flakes
- Sprinkle of parmesan cheese (stronger flavor than most cheese)
- Sodium-free salt substitute
- Jelly or fruit preserves on toast or bagels

visited on 6/14/2019

VI. A.3. Dining Out—How to Choose

VI. A.3.a. General Tips for Healthy Dining Out

Whether or not you're trying to lose weight, you can eat healthy when dining out or bringing in food, if you know how. The following tips will help you move toward healthier eating as you limit your calories, as well as fat, saturated fat, cholesterol, and sodium when eating out.

You Are the Customer -

- Ask for what you want! Most restaurants will honor your requests.
- Ask questions! Don't be intimidated by the menu - your server will be able to tell you how foods are prepared or suggest substitutions on the menu.
- If you wish to reduce portion sizes - try ordering appetizers as your main meal.

General Tips: Limiting your calories and fat can be easy as long as you know what to order. Try asking these questions when you call ahead or before you order. Ask the restaurant "Do you or would you on request...":

- Serve margarine (rather than butter) with the meal?
- Serve fat-free (skim) milk rather than whole milk or cream?
- Use less oil when cooking?
- Trim visible fat off poultry or meat?
- Leave all butter, gravy, or sauces off a side dish or entree?
- Serve salad dressing on the side?
- Accommodate special requests if made in advance by telephone or in person?
- Above all else, don't get discouraged. There are usually several healthy choices to choose from at most restaurants.

Reading the Menu -

- Choose lower-calorie, low-fat cooking methods. Look for terms like steamed, in its own juice (au jus), garden fresh, broiled, baked, roasted, poached, tomato juice, dry boiled (in wine or lemon juice), and lightly sautéed or stir-fried.
- Be aware of foods high in calories, fat, and saturated fat. Watch out for terms like butter sauce, fried, crispy, creamed, in cream or cheese sauce, au gratin, au fromage, escalloped, parmesan, hollandaise, bernaise, marinated (in oil), stewed, basted, sautéed, stir-fried, casserole, hash, prime, pot pie and pastry crust.

Specific Tips for Healthy Choices

Breakfast

- Fresh fruit or small glass of citrus juice
- Whole grain bread, bagel or English muffin with jelly or honey
- Whole grain cereal with low-fat (1%) or fat-free milk
- Oatmeal with fat-free milk topped with fruit
- Omelet made with egg whites or egg substitute
- Multigrain pancakes without butter on top
- Nonfat yogurt (try adding cereal or fresh fruit)

Beverages

- Water with lemon
- Flavored sparkling water (noncaloric)
- Juice spritzer (half fruit juice and half sparkling water)
- Iced tea
- Tomato juice (reduced sodium)

visited on 6/14/2019

Bread

Most bread and breadsticks are low in calories and low in fat. The calories add up when you add butter, margarine, or olive oil to the bread. Also, eating a lot of bread in addition to your meal will fill you up with extra unwanted calories and not leave enough room for fruits and vegetables.

Appetizers

- Steamed seafood
- Shrimp* cocktail (limit cocktail sauce - it's high in sodium)
- Melons or fresh fruit
- Bean soups
- Salad with reduced fat dressing (or add lemon juice or vinegar)

*If you are on a cholesterol-lowering diet, eat shrimp and other shellfish in moderation.

Entree

- Poultry, fish, shellfish and vegetable dishes are healthy choices
- Pasta with red sauce or with vegetables (primavera)
- Look for terms like baked, broiled, steamed, poached, lightly sautéed or stir-fried
- Ask for sauces and dressings on the side
- Limit the amount of butter, margarine, and salt you use at the table

Salads/Salad Bars

- Fresh greens, lettuce and spinach
- Fresh vegetables - tomato, mushroom, carrots, cucumber, peppers, onion, radishes, and broccoli
- Beans, chick peas and kidney beans
- Skip the nonvegetable choices: deli meats, bacon, egg, cheese, croutons
- Choose lower-calorie, reduced-fat or fat-free dressing, lemon juice, or vinegar

Side Dish

- Plain vegetables and starches (rice, potato, noodles) make good additions to meals and can also be combined for a lower-calorie alternative to higher-calorie entrees
- Ask for side dishes without butter or margarine
- Ask for mustard, salsa or low-fat yogurt instead of sour cream or butter

Dessert/Coffee

- Fresh fruit
- Nonfat frozen yogurt
- Sherbet or fruit sorbet (these are usually fat free, but check the calorie content)
- Try sharing a dessert
- Ask for low-fat milk for your coffee (instead of cream or half-'n-half)

VI. A.3.b. Tips for Healthy Multicultural Dining Out

If you're dining out or bringing in, it is easy to find healthy foods. Knowing about American food terms, as well as other ethnic cuisines can help make your dining experience healthy and enjoyable! The following list includes healthy food choices (lower in calories and fat) and terms to look for when making your selection.

Chinese

Choose More Often...

- Steamed
- Jum (poached)
- Chu (boiled)
- Kow (roasted)
- Shu (barbecued)
- Hoison sauce with assorted Chinese vegetables: broccoli, mushroom, onion, cabbage, snow peas, scallions, bamboo shoots, water chestnuts, asparagus

visited on 6/14/2019

- Oyster sauce (made from seafood)
- Lightly stir-fried in mild sauce
- Cooked in light wine sauce
- Hot and spicy tomato sauce
- Sweet and sour sauce
- Hot mustard sauce
- Reduced sodium soy sauce
- Dishes without MSG added
- Garnished with spinach or broccoli
- Fresh fish filets, shrimp, scallops
- Chicken, without skin
- Lean beef
- Bean curd (tofu)
- Moo Shu vegetable, chicken or shrimp
- Steamed rice
- Lychee fruit

French

Choose More Often...

- Dinner salad with vinegar or lemon juice dressing (or other reduced fat dressing)
- Crusty bread without butter
- Fresh fish, shrimp, scallops, steamed mussels (without sauces)
- Chicken breast, without skin
- Rice and noodles without cream or added butter or other fat
- Fresh fruit for dessert

Italian

Choose More Often...

- Lightly sautéed with onions
- Shallots
- Peppers and mushrooms
- Artichoke hearts
- Sun-dried tomatoes
- Red sauces - spicy marinara sauce (arrabiata), marinara sauce or cacciatore
- Light red sauce or light red or white wine sauce
- Light mushroom sauce
- Red clam sauce
- Primavera (no cream sauce)
- Lemon sauce
- Capers
- Herbs and spices - garlic and oregano
- Crushed tomatoes and spices
- Florentine (spinach)
- Grilled (often fish or vegetables)
- Piccata (lemon)
- Manzanne (eggplant)

Middle Eastern

Choose More Often ...

- Lemon dressing, lemon juice
- Blended or seasoned with Middle Eastern spices
- Herbs and spices
- Mashed chickpeas
- Fava beans
- Smoked eggplant
- With tomatoes, onions, green peppers and cucumbers
- Spiced ground meat
- Special garlic sauce
- Basted with tomato sauce
- Garlic

visited on 6/14/2019

- Chopped parsley and/or onion
- Couscous (grain)
- Rice or bulgur (cracked wheat)
- Stuffed with rice and imported spices
- Grilled on a skewer
- Marinated and barbecued
- Baked
- Charbroiled or charcoal broiled
- Fresh fruit

Japanese

Choose More Often...

- House salad with fresh ginger and cellophane (clear rice) noodles
- Rice
- Nabemono
- Chicken, fish or shrimp teriyaki, broiled in sauce
- Menrui or soba noodles, often used in soups
- Yakimono (broiled)
- Tofu or bean curd
- Grilled vegetables

Indian

Choose More Often...

- Tikka (pan roasted)
- Cooked with or marinated in yogurt
- Cooked with green vegetables, onions, tomatoes, peppers, and mushrooms
- With spinach (saag)
- Baked leavened bread
- Masala
- Tandoori
- Paneer
- Cooked with curry, marinated in spices
- Lentils, chick beans, garbanzo beans, beans
- Garnished with dried fruits
- Chickpeas (garbanzo) and potatoes
- Basmati rice (pullao)
- Matta (peas)
- Chicken or shrimp kebab

Mexican

Choose More Often...

- Shredded spicy chicken
- Rice and black beans
- Rice
- Ceviche (fish marinated in lime juice and mixed with spices)
- Served with salsa (hot red tomato sauce)
- Served with salsa verde (green chili sauce)
- Covered with enchilada sauce
- Topped with shredded lettuce, diced tomatoes and onions
- Served with or wrapped in a corn or wheat flour (soft) tortilla
- Grilled
- Marinated
- Picante sauce
- Simmered with chili vegetarian tomato sauce

Thai

Choose More Often...

- Barbecued, sautéed, broiled, boiled, or steamed, braised, marinated
- Charbroiled
- Basil sauce, basil or sweet basil leaves
- Lime sauce or lime juice
- Chili sauce or crushed dried chili flakes

visited on 6/14/2019

- Thai spices
- Served in hollowed-out pineapple
- Fish sauce
- Hot sauce
- Napa, bamboo shoots, black mushrooms, ginger, garlic
- Bed of mixed vegetables
- Scallions, onions

Steakhouses

Choose More Often...

- Lean broiled beef (no more than 6 ounces) - London broil, filet mignon, round and flank steaks
- Baked potato without added butter, margarine or sour cream. Try low-fat yogurt or mustard.
- Green salad with reduced fat dressing
- Steamed vegetables without added butter or margarine. Try lemon juice and herbs.
- Seafood dishes (usually indicated as "surf" on menus)

Fast Food

- Grilled chicken breast sandwich without mayonnaise
- Single hamburger without cheese
- Grilled chicken salad with reduced-fat dressing
- Garden salad with reduced-fat dressing
- Low-fat or nonfat yogurt
- Fat-free muffin
- Cereal with low-fat milk

Deli/Sandwich Shop

Choose More Often...

- Fresh sliced vegetables in pita bread with low-fat dressing, yogurt or mustard
- Cup of bean soup (lentil, minestrone)
- Turkey breast sandwich with mustard, lettuce, tomato
- Fresh fruit

visited on 6/14/2019

Sample Reduced-Calorie Menus

	Calories	Total CHO % kcal	Total Fat % kcal	Sodium (mg)	SFA % kcal	Cholesterol (mg)	Protein % kcal
Traditional Cuisine							
1,600	1,613	55	29	1,341	8	142	19
1,200	1,247	58	26	1,043	7	96	19
Asian-American Cuisine							
1,600	1,609	56	27	1,296	8	148	20
1,200	1,220	55	27	1,043	8	117	21
Southern Cuisine							
1,600	1,653	53	28	1,231	8	172	20
1,200	1,225	50	31	867	9	142	21
Mexican-American Cuisine							
1,600	1,638	56	27	1,616	9	143	20
1,200	1,239	58	26	1,364	8	91	19
Lacta-Ovo Vegetarian Cuisine							
1,600	1,650	56	27	1,829	8	82	19
1,200	1,205	60	25	1,335	7	44	18

visited on 6/14/2019

TABLE VI.A.4.a:

Sample Menu: Traditional American Cuisine, Reduced Calorie

	1,600 Calories	1,200 Calories
Breakfast		
Whole Wheat Bread	1 slice	1 slice
Jelly, regular	2 tsp	2 tsp
Cereal, Shredded Wheat	1 cup	½ cup
Milk, 1% low-fat	1 cup	1 cup
Orange Juice	¾ cup	¾ cup
Coffee, Regular	1 cup	1 cup
Milk, 1% low-fat	1 oz	—
Lunch		
Roast Beef Sandwich		
Whole Wheat Bread	2 slices	2 slices
Lean Roast Beef, unseasoned	2 oz	2 oz
American Cheese, low-fat, low-sodium	1 slice (¾ oz)	—
Lettuce	1 leaf	1 leaf
Tomato	3 slices	3 slices
Mayonnaise, low-calorie	2 tsp	1 tsp
Apple	1 medium	1 medium
Water	1 cup	1 cup
Dinner		
Salmon	3 oz	2 oz
Vegetable Oil	1½ tsp	1½ tsp
Baked Potato	¾ medium	¾ medium
Margarine	1 tsp	1 tsp
Carrots seasoned with	½ cup	½ cup
margarine	½ tsp	—
Green Beans seasoned with	½ cup	½ cup
margarine	½ tsp	½ tsp
White Dinner Roll	1 medium	1 small
Ice Milk	½ cup	—
Iced Tea, unsweetened	1 cup	1 cup
Water	2 cups	2 cups
Snack		
Popcorn, air popped	2½ cups	2½ cups
Margarine	1½ tsp	¾ tsp

Calories:	1,613	Calories:	1,247
Total Carb, % kcals:	55	Total Carb, % kcals:	58
Total Fat, % kcals:	29	Total Fat, % kcals:	26
*Sodium, mg:	1,341	*Sodium, mg:	1,043
SFA, % kcals:	8	SFA, % kcals:	7
Cholesterol, mg:	142	Cholesterol, mg:	96
Protein, % kcals:	19	Protein, % kcals:	19

1,600: 100% RDA met for all nutrients except: Vit E 99%, Iron 73%, Zinc 91%
1,200: 100% RDA met for all nutrients except: Vit E 80%, Vit B_2 96%, Vit B_6 94%, Calcium 68%, Iron 63%, Zinc 73%
* No salt added in recipe preparation or as seasoning. Consume at least 32 oz. water.

visited on 6/14/2019

TABLE VI.A.4.b:

Sample Menu: Asian-American Cuisine, Reduced Calorie

	1,600 Calories	1,200 Calories
Breakfast		
Banana	1 small	1 small
Whole Wheat Bread	2 slices	1 slice
Margarine	1 tsp	1 tsp
Orange Juice	¾ cup	¾ cup
Milk, 1% low-fat	¾ cup	¾ cup
Lunch		
Beef Noodle Soup, canned, low-sodium	½ cup	½ cup
Chinese Noodle and Beef Salad		
Beef Roast	3 oz	2 oz
Peanut Oil	1½ tsp	1 tsp
Soy Sauce, low-sodium	1 tsp	1 tsp
Carrots	½ cup	½ cup
Zucchini	½ cup	½ cup
Onion	¼ cup	¼ cup
Chinese Noodles, soft-type	¼ cup	¼ cup
Apple	1 medium	1 medium
Tea, unsweetened	1 cup	1 cup
Dinner		
Pork Stir-fry with Vegetables		
Pork Cutlet	2 oz	2 oz
Peanut Oil	1 tsp	1 tsp
Soy Sauce, low-sodium	1 tsp	1 tsp
Broccoli	½ cup	½ cup
Carrots	1 cup	½ cup
Mushrooms	¼ cup	½ cup
Steamed White Rice	1 cup	½ cup
Tea, unsweetened	1 cup	1 cup
Snack		
Almond Cookies	2 cookies	—
Milk, 1% low-fat	¾ cup	¾ cup

Calories:	1,609	Calories:	1,220
Total Carb, % kcals:	56	Total Carb, % kcals:	55
Total Fat, % kcals:	27	Total Fat, % kcals:	27
*Sodium, mg:	1,296	*Sodium, mg:	1,043
SFA, % kcals:	8	SFA, % kcals:	8
Cholesterol, mg:	148	Cholesterol, mg:	117
Protein, % kcals:	20	Protein, % kcals:	21

1,600: 100% RDA met for all nutrients except: Zinc 95%, Iron 87%, Calcium 93%
1,200: 100% RDA met for all nutrients except: Vit E 75%, Calcium 84%, Magnesium 98%, Iron 66%, Zinc 77%
* No salt added in recipe preparation or as seasoning. Consume at least 32 oz. water.

visited on 6/14/2019

TABLE VI.A.4.c:

Sample Menu: Southern Cuisine, Reduced Calorie

	1,600 Calories	1,200 Calories
Breakfast		
Oatmeal, prepared with 1% low-fat milk	½ cup	½ cup
Milk, 1% low-fat	½ cup	½ cup
English Muffin	1 medium	—
Cream Cheese, light, 18% fat	1 T	—
Orange Juice	¾ cup	½ cup
Coffee	1 cup	1 cup
Milk, 1% low-fat	1 oz	1 oz
Lunch		
Baked Chicken, without skin	2 oz	2 oz
Vegetable Oil	1 tsp	½ tsp
Salad		
Lettuce	½ cup	½ cup
Tomato	½ cup	½ cup
Cucumber	½ cup	½ cup
Oil and Vinegar Dressing	2 tsp	1 tsp
White Rice, seasoned with	½ cup	¼ cup
margarine, diet	½ tsp	½ tsp
Baking Powder Biscuit, prepared with vegetable oil	1 small	½ small
Margarine	1 tsp	1 tsp
Water	1 cup	1 cup
Dinner		
Lean Roast Beef	3 oz	2 oz
Onion	¼ cup	¼ cup
Beef Gravy, water-based	1 T	1 T
Turnip Greens, seasoned with	½ cup	½ cup
margarine, diet	½ tsp	½ tsp
Sweet Potato, baked	1 small	1 small
Margarine, diet	½ tsp	¼ tsp
Ground Cinnamon	1 tsp	1 tsp
Brown Sugar	1 tsp	1 tsp
Cornbread prepared with margarine, diet	½ medium slice	½ medium slice
Honeydew Melon	¼ medium	⅛ medium
Iced Tea, sweetened with sugar	1 cup	1 cup
Snack		
Saltine Crackers, unsalted tops	4 crackers	4 crackers
Mozzarella Cheese, part-skim, low-sodium	1 oz	1 oz

Calories:	1,653	Calories:	1,225
Total Carb, % kcals:	53	Total Carb, % kcals:	50
Total Fat, % kcals:	28	Total Fat, % kcals:	31
*Sodium, mg:	1,231	*Sodium, mg:	867
SFA, % kcals:	8	SFA, % kcals:	9
Cholesterol, mg:	172	Cholesterol, mg:	142
Protein, % kcals:	20	Protein, % kcals:	21

1,600: 100% RDA met for all nutrients except: Vit E 97%, Magnesium 98%, Iron 78%, Zinc 90%

1,200: 100% RDA met for all nutrients except: Vit E 82%, Vit B_1 & B_2 95%, Vit B_3 99%, Vit B_6 88%, Magnesium 83%, Iron 56%, Zinc 70%

* No salt added in recipe preparation or as seasoning. Consume at least 32 oz. water.

visited on 6/14/2019

TABLE VI.A.4.d:

Sample Menu: Mexican-American Cuisine, Reduced Calorie

	1,600 Calories	1,200 Calories
Breakfast		
Cantaloupe	1 cup	½ cup
Farina, prepared with 1% low-fat milk	½ cup	½ cup
White Bread	1 slice	1 slice
Margarine	1 tsp	1 tsp
Jelly	1 tsp	1 tsp
Orange Juice	1½ cup	¾ cup
Milk, 1% low-fat	½ cup	½ cup
Lunch		
Beef Enchilada		
Tortilla, corn	2 tortillas	2 tortillas
Lean Roast Beef	2 ½ oz	2 oz
Vegetable Oil	⅔ tsp	⅔ tsp
Onion	1 T	1 T
Tomato	4 T	4 T
Lettuce	½ cup	½ cup
Chili Peppers	2 tsp	2 tsp
Refried Beans, prepared with vegetable oil	¼ cup	¼ cup
Carrots	5 sticks	5 sticks
Celery	6 sticks	6 sticks
Milk, 1% low-fat	½ cup	—
Dinner		
Chicken Taco		
Tortilla, corn	1 tortilla	1 tortilla
Chicken Breast, without skin	2 oz	1 oz
Vegetable Oil	⅔ tsp	⅔ tsp
Cheddar Cheese, low-fat, low-sodium	1 oz	½ oz
Guacamole	2 T	1 T
Salsa	1 T	1 T
Corn, seasoned with	½ cup	½ cup
margarine	½ tsp	—
Spanish Rice without meat, seasoned without margarine	½ cup	½ cup
Banana	1 large	½ large
Coffee	1 cup	1 cup
Milk 1%	1 oz	1oz

Calories:	1,638	Calories:	1,239
Total Carb, % kcals:	56	Total Carb, % kcals:	58
Total Fat, % kcals:	27	Total Fat, % kcals:	26
*Sodium, mg:	1,616	*Sodium, mg:	1,364
SFA, % kcals:	9	SFA, % kcals:	8
Cholesterol, mg:	143	Cholesterol, mg:	91
Protein, % kcals:	20	Protein, % kcals:	19

1,600: 100% RDA met for all nutrients except: Vit E 97%, Zinc 84%

1,200: 100% RDA met for all nutrients except: Vit E 71%, Vit B_1 & B_3 91%, Vit B_2 & Iron 90%, Calcium 92%, Magnesium 95%, Zinc 64%

* No salt added in recipe preparation or as seasoning. Consume at least 32 oz. water.

visited on 6/14/2019

TABLE VI.A.4.e:

Sample Menu: Lacto-Ovo Vegetarian Cuisine, Reduced Calorie

	1,600 Calories	1,200 Calories
Breakfast		
Orange	1 medium	1 medium
Pancakes, made with 1% low-fat milk, egg whites	3 4" circles	2 4" circles
Pancake Syrup	2 T	1 T
Margarine, diet	1½ tsp	1½ tsp
Milk, 1% low-fat	1 cup	½ cup
Coffee	1 cup	1 cup
Milk, 1% low-fat	1 oz	1 oz
Lunch		
Vegetable Soup, canned, low-sodium	1 cup	½ cup
Bagel	1 medium	½ medium
Processed American Cheese, low-fat and low sodium	¾ oz	—
Spinach Salad		
Spinach	1 cup	1 cup
Mushrooms	⅛ cup	⅛ cup
Salad Dressing, regular calorie	2 tsp	2 tsp
Apple	1 medium	1 medium
Iced Tea, unsweetened	1 cup	1 cup
Dinner		
Omelette		
Egg Whites	4 large eggs	4 large eggs
Green Pepper	2 T	2 T
Onion	2 T	2T
Mozzarella Cheese, made from part-skim milk, low-sodium	1½ oz	1 oz
Vegetable Oil	1 T	½ T
Brown Rice, seasoned with	½ cup	½ cup
margarine, diet	½ tsp	½ tsp
Carrots, seasoned with	½ cup	½ cup
margarine, diet	½ tsp	½ tsp
Whole Wheat Bread	1 slice	1 slice
Margarine, diet	1 tsp	1 tsp
Fig Bar Cookie	1 bar	1 bar
Tea	1 cup	1 cup
Honey	1 tsp	1 tsp
Snack		
Milk, 1% low-fat	¾ cup	¾ cup

Calories:	1,650	Calories:	1,205
Total Carb, % kcals:	56	Total Carb, % kcals:	60
Total Fat, % kcals:	27	Total Fat, % kcals:	25
*Sodium, mg:	1,829	*Sodium, mg:	1,335
SFA, % kcals:	8	SFA, % kcals:	7
Cholesterol, mg:	82	Cholesterol, mg:	44
Protein, % kcals:	19	Protein, % kcals:	18

1,600: 100% RDA met for all nutrients except: Vit E 92%, Vit B_3 97%, Vit B_6 67%, Magnesium 98%, Iron 73%, Zinc 68%
1,200: 100% RDA met for all nutrients except: Vit E 75%, Vit B_1 92%, Vit B_3 69%, Vit B_6 59%, Iron 54%, Zinc 46%
* No salt added in recipe preparation or as seasoning. Consume at least 32 oz. water.

visited on 6/14/2019

7 Appendix VII. Resource List

For additional information on overweight and obesity, and related conditions or diseases, you may wish to contact the professional organizations listed below.

The Federal consumer health information gateway, healthfinder, (www.healthfinder.gov) offers convenient access to these and many other resources.

American Dietetic Association
216 West Jackson Boulevard
Chicago, IL 60606-6995
(800) 877-1600
FAX: (312) 899-1979
http://www.eatright.org

- Eat Right America program
- List of nutrition resources
- Find a dietitian, 1-800-366-1655

American College of Sports Medicine
P.O. Box 1440
Indianapolis, IN 46206-1440
(317) 637-9200
FAX: (317) 634-7817
http://www.acsm.org

- Health and Fitness Summit and Exposition
- National Coalition for Promoting Physical Activity
- Public information

National Heart, Lung, and Blood Institute
Education Programs Information Center
P.O. Box 30105
Bethesda, MD 20824-0105
(301) 251-1222
FAX: (301) 251-1223
http://www.nhlbi.nih.gov/nhlbi/nhlbi.htm (general NHLBI site)

- Online publications on blood pressure, overweight, cholesterol, heart disease, sleep disorders, and asthma

National Institute of Diabetes and Digestive and Kidney Diseases
31 Center Drive, MSC-2560
Building 31, Room 9A-04
Bethesda, MD 20892-2560
(301) 496-3583
FAX: (301) 496-7422
http://www.niddk.nih.gov

- Many online patient information publications on diabetes
- Many online publications on nutrition and obesity
- WIN — the Weight-Control Information Network 1-800-WIN-8098

The Weight-Control Information Network
National Institute of Diabetes and Digestive and Kidney Diseases
1 WIN WAY
Bethesda, MD 20892-3665
(301) 570-2177
FAX: (301) 570-2186
1-800-WIN-8098

National Digestive Diseases Information Clearinghouse (NIDDK)
2 Information Way
Bethesda, MD 20892-3570
(301) 654-3810
FAX: (301) 907-8906

National Kidney and Urologic Diseases Information Clearinghouse (NIDDK)
3 Information Way
Bethesda, MD 20892-3580
(301) 654-4415
FAX: (301) 907-8906

National Diabetes Information Clearinghouse (NIDDK)
1 Information Way
Bethesda, MD 20892-3560
(301) 654-3327
FAX: (301) 907-8906

visited on 6/14/2019

American Diabetes Association
1660 Duke Street
Alexandria, VA 22314
1-800 DIABETES
http://www.diabetes.org
- List of publications

American Cancer Society
Atlanta, GA
1-800-ACS-2345
http://www.cancer.org
- Dietary guidelines online
- List of publications

National Cancer Institute
Office of Cancer Communications
9000 Rockville Pike
Building 31, Room 10A-24
Bethesda, MD 20892
(800) 4-CANCER (800-422-6237)
http://www.nci.nih.gov
- Many publications online, including those on nutrition and cancer

National Eating Disorders Organization
6655 South Yale Avenue
Tulsa, OK 74136
(918) 481-4044
FAX: (918) 481-4076
http://www.laureate.com/aboutned.html
- Publications list
- Online descriptive information

Eating Disorders Awareness and Prevention, Inc.
603 Stewart Street, Suite 803
Seattle, WA 98101
(206) 382-3587
http://members.aol.com/edapinc/home.html
- Online descriptive information
- Publications list

American Anorexia/Bulimia Association, Inc.
165 West 46th Street #1108
New York, NY 10036
(212) 575-6200
http://members.aol.com/amanbu/index.html
- Online descriptive information

National Association of Anorexia Nervosa and Associated Disorders
P.O.Box 7
Highland Park, IL 60035
(847) 831-3438
FAX: (847) 433-4632
http://www.medpatients.com/Health%20Resources/NAANAD.htm

American Heart Association
7272 Greenville Avenue
Dallas, TX 75231-4596
(214) 706-1220
FAX: (214) 706-1341
1-800-AHA-USA1 (800-242-8721)
http://www.americanheart.org
- Publications list
- Online information
- Stroke Connection 1-800-553-6321

Hypertension Network, Inc.
http://www.bloodpressure.com
- Online information
- Weekly research updates

National Institute of Neurological Disorders and Stroke
P.O. Box 5801
Bethesda, MD 20824
(301) 496-5751
http://www.ninds.nih.gov
- Online publications on stroke
- Publications list

National Center on Sleep Disorders Research
National Heart, Lung, and Blood Institute
Two Rockledge Centre, Suite 7024
6701 Rockledge Drive, MSC 7920
Bethesda, MD 20892-7920
(301) 435-0199
FAX: (301) 480-3451

visited on 6/14/2019

American Sleep Disorders Association
1610 14th Street NW, Suite 300
Rochester, MN 55901
(507) 287-6006
Fax: (507) 287-6008
http://www.asda.org

- List of member centers
- Online descriptive information

The Sleep Medicine Home Page
http://www.users.cloud9.net/~thorpy

- List of internet resources
- List of centers
- Online descriptive information

National Mental Health Association
1201 Prince Street
Alexandria, VA 22314-2971
(703) 684-7722
Fax: (703) 684-5968
800/969-NMHA Information Center
http://www.nmha.org

North American Association for the Study of Obesity (NAASO)
8630 Fenton Street
Suite 412
Silver Spring, MD 20910
(301) 563-6526
Fax: (301) 587-2365
http://www.naaso.org

visited on 6/14/2019

8 Appendix VIII. Glossary of Terms

Abdominal fat: Fat (adipose tissue) that is centrally distributed between the thorax and pelvis and that induces greater health risk.

Absolute risk: The observed or calculated probability of an event in a population under study, as contrasted with the relative risk.

Aerobic exercise: A type of physical activity that includes walking, jogging, running, and dancing. Aerobic training improves the efficiency of the aerobic energy-producing systems that can improve cardiorespiratory endurance.

Age-adjusted: Summary measures of rates of morbidity or mortality in a population using statistical procedures to remove the effect of age differences in populations that are being compared. Age is probably the most important and the most common variable in determining the risk of morbidity and mortality.

Anorexiant: A drug, process, or event that leads to anorexia.

Anthropometric measurements: Measurements of human body height, weight, and size of component parts, including skinfold measurement. Used to study and compare the relative proportions under normal and abnormal conditions.

Atherogenic: Causing the formation of plaque in the lining of the arteries.

Behavior therapy: Behavior therapy constitutes those strategies, based on learning principles such as reinforcement, that provide tools for overcoming barriers to compliance with dietary therapy and/or increased physical activity.

Biliopancreatic diversion: A surgical procedure for weight loss that combines a modest amount of gastric restriction with intestinal malabsorption.

BMI: Body mass index; the body weight in kilograms divided by the height in meters squared (wt/ht^2) used as a practical marker to assess obesity; often referred to as the Quetelet Index. An indicator of optimal weight for health and different from lean mass or percent body fat calculations because it only considers height and weight.

Body composition: The ratio of lean body mass (structural and functional elements in cells, body water, muscle, bone, heart, liver, kidneys, etc.) to body fat (essential and storage) mass. Essential fat is necessary for normal physiological functioning (e.g., nerve conduction). Storage fat constitutes the body's fat reserves, the part that people try to lose.

BRL 26830A: An atypical B adrenoreceptor agonist drug that in obese rodents showed an increased metabolic rate and caused a reduction in weight by decreasing body lipid content. It is not approved as a weight loss drug by FDA.

Carbohydrates: A nutrient that supplies 4 calories/gram. They may be simple or complex. Simple carbohydrates are called sugars, and complex carbohydrates are called starch and fiber (cellulose). An organic compound—containing

visited on 6/14/2019

carbon, hydrogen, and oxygen—that is formed by photosynthesis in plants. Carbohydrates are heat producing and are classified as monosaccharides, disaccharides, or polysaccharides.

Cardiovascular disease (CVD): Any abnormal condition characterized by dysfunction of the heart and blood vessels. CVD includes atherosclerosis (especially coronary heart disease, which can lead to heart attacks), cerebrovascular disease (e.g., stroke), and hypertension (high blood pressure).

Central fat distribution: The waist circumference is an index of body fat distribution. Increasing waist circumference is accompanied by increasing frequencies of overt type 2 diabetes, dyslipidemia, hypertension, coronary heart disease, stroke, and early mortality. In the body fat patterns called android type (apple shaped) fat is deposited around the waist and upper abdominal area and appears most often in men. Abdominal body fat is thought to be associated with a rapid mobilization of fatty acids rather than resulting from other fat depots, although it remains a point of contention. If abdominal fat is indeed more active than other fat depots, it would then provide a mechanism by which we could explain (in part) the increase in blood lipid and glucose levels. The latter have been clearly associated with an increased risk for cardiovascular disease hypertension and type 2 diabetes mellitus. The gynoid type (pear-shaped) of body fat is usually seen in women. The fat is deposited around the hips, thighs, and buttocks, and presumably is used as energy reserve during pregnancy and lactation.

Cholecystectomy: Surgical removal of the gallbladder and gallstones, if present.

Cholecystitis: Inflammation of the gallbladder, caused primarily by gallstones. Gallbladder disease occurs most often in obese women older than 40 years of age.

Cholesterol: A soft, waxy substance manufactured by the body and used in the production of hormones, bile acid, and vitamin D and present in all parts of the body, including the nervous system, muscle, skin, liver, intestines, and heart. Blood cholesterol circulates in the bloodstream. Dietary cholesterol is found in foods of animal origin.

Cimetidine: A weight loss drug that is thought to work by suppression of gastric acid or suppression of hunger by blocking histamine H_2 receptors. It is not approved by the FDA.

Cognitive behavior therapy: A system of psychotherapy based on the premise that distorted or dysfunctional thinking, which influences a person's mood or behavior, is common to all psychosocial problems. The focus of therapy is to identify the distorted thinking and to replace it with more rational, adaptive thoughts and beliefs.

Cognitive rehearsal: A technique used in cognitive behavior therapy. In a weight loss program, for example, individuals first imagine the situation that is causing temptation (such as eating a high fat food), describe the thoughts and feelings that accompany the imagined situation, and make positive self-statements about the situation (e.g., "I am feeling good about choosing a low calorie drink rather than the high fat cheese."). Then the next step is to follow the positive self-statement with an adaptive behavior (such as walking away from the buffet line to chat with a friend). Finally, individuals are encouraged to reward themselves for doing well in a difficult situation, with either positive statements or material rewards, or both. The idea is to rehearse one's thoughts and behaviors prior to experiencing the potentially difficult situation, and to be armed with healthy adaptive responses.

Cognitive restructuring: A method of identifying and replacing fear-promoting, irrational beliefs with more realistic and functional ones.

visited on 6/14/2019

Comorbidity: Two or more diseases or conditions existing together in an individual.

Computed tomography (CT) scans: A radiographic technique for direct visualization and quantification of fat that offers high image contrast and clear separation of fat from other soft tissues. CT can estimate total body adipose tissue volume and identify regional, subcutaneous, visceral, and other adipose tissue depots. Radiation exposure, expense, and unavailability restrict the epidemiologic use of CT.

Confounding: Extraneous variables resulting in outcome effects that obscure or exaggerate the "true" effect of an intervention.

Coronary heart disease (CHD): A type of heart disease caused by narrowing of the coronary arteries that feed the heart, which needs a constant supply of oxygen and nutrients carried by the blood in the coronary arteries. When the coronary arteries become narrowed or clogged by fat and cholesterol deposits and cannot supply enough blood to the heart, CHD results.

Cue avoidance: A stimulus control technique often used in weight loss programs in which individuals are asked to reduce their exposure to certain food cues by making a variety of changes in their habits. The rationale is to make it easier on oneself and reduce temptation by reducing contact with food cues. For example, coming home from work and feeling tired is a time when many people reach for the high fat foods if they are available. By not having the high fat foods within reach, one can avoid eating them.

Dexfenfluramine: A serotonin agonist drug used to treat obesity. FDA approval has been withdrawn.

Diabetes: A complex disorder of carbohydrate, fat, and protein metabolism that is primarily a result of relative or complete lack of insulin secretion by the beta cells of the pancreas or a result of defects of the insulin receptors.

Diastolic blood pressure: The minimum pressure that remains within the artery when the heart is at rest.

Diethylproprion: An appetite suppressant prescribed in the treatment of obesity.

Dopamine: A catecholamine neurotransmitter that is found primarily in the basal ganglia of the central nervous system. Major functions include the peripheral inhibition and excitation of certain muscles; cardiac excitation; and metabolic, endocrine and central nervous system actions.

Dual energy X-ray absortiometry (DEXA): A method used to estimate total body fat and percent of body fat. Potential disadvantages include whole body radiation and the long time required for scanning while the subject lies on a hard table.

Dyslipidemia: Disorders in the lipoprotein metabolism; classified as hypercholesterolemia, hypertriglyceridemia, combined hyperlipidemia, and low levels of high-density lipoprotein (HDL) cholesterol. All of the dyslipidemias can be primary or secondary. Both elevated levels of low-density lipoprotein (LDL) cholesterol and low levels of HDL cholesterol predispose to premature atherosclerosis.

Efficacy: The extent to which a specific intervention, procedure, regimen, or service produces a beneficial result under ideal conditions. Ideally, the determination of efficacy is based on the results of a randomized control trial.

Energy balance: Energy is the capacity of a body or a physical system for doing work. Energy balance is the state in which the total energy intake equals total energy needs.

Energy deficit: A state in which total energy intake is less than total energy need.

Ephedrine: A sympathomimetic drug that stimulates thermogenesis in laboratory animals and

visited on 6/14/2019

humans. Animal studies show that it may reduce fat content and, therefore, body weight by mechanisms that probably involve increased expenditure and reduced food intake.

Extreme obesity: A body mass index ≥ 40.

Femoxetine: A selective serotonin reuptake inhibitor drug used in obese patients for weight loss.

Fenfluramine: A serotonin agonist drug used in the treatment of obesity. FDA approval has been withdrawn.

Fibrinogen: A plasma protein that is converted into fibrin by thrombin in the presence of calcium ions. Fibrin is responsible for the semisolid character of a blood clot.

Fluoxetine: An antidepressant drug used to promote weight loss whose action is mediated by highly specific inhibition of serotonin reuptake into presynaptic neurons. Serotonin acts in the brain to alter feeding and satiety by decreasing carbohydrate intake, resulting in weight reduction.

Framingham Heart Study: Study begun in 1948 to identify constitutional, environmental, and behavioral influences on the development of cardiovascular disease. Framingham data show that increased relative weight and central obesity are associated with elevated levels of risk factors (e.g., cholesterol, blood pressure, blood glucose, uric acid), increased incidence of cardiovascular disease, and increased death rates for all causes combined.

Gallstones: Constituents in the gallbladder that are not reabsorbed, including bile salts and lipid substances such as cholesterol that become highly concentrated. They can cause severe pain (obstruction and cramps) as they move into the common bile duct. Risk factors for cholesterol gallstone formation include female gender, weight gain, overweight, high energy intake, ethnic factors (Pima Indians and Scandinavians), use of certain drugs (clofibrate, estrogens, and bile acid sequestrants), and presence of gastrointestinal disease. Gallstones sometimes develop during dieting for weight reduction. There is an increased risk for gallstones and acute gallbladder disease during severe caloric restriction.

Gastric banding: Surgery to limit the amount of food the stomach can hold by closing part of it off. A band made of special material is placed around the stomach near its upper end, creating a small pouch and a narrow passage into the larger remainder of the stomach. The small outlet delays the emptying of food from the pouch and causes a feeling of fullness.

Gastric bubble/balloon: A free-floating intragastric balloon used in the treatment of obesity.

Gastric bypass: A surgical procedure that combines the creation of small stomach pouches to restrict food intake and the construction of bypasses of the duodenum and other segments of the small intestine to cause food malabsorption. Patients generally lose two-thirds of their excess weight after 2 years.

Gastric exclusion: Same as gastric partitioning and Roux-en Y bypass. A small stomach pouch is created by stapling or by vertical banding to restrict food intake. A Y-shaped section of the small intestine is attached to the pouch to allow food to bypass the duodenum as well as the first portion of the jejunum.

Gastric partitioning: See gastric exclusion.

Gastroplasty: See also jejuno-ileostomy. A surgical procedure that limits the amount of food the stomach can hold by closing off part of the stomach. Food intake is restricted by creating a small pouch at the top of the stomach where the food enters from the esophagus. The pouch initially holds about 1 ounce of food and expands to 2-3 ounces with time. The pouch's lower outlet usually has a diameter of about 1/4 inch. The

visited on 6/14/2019

small outlet delays the emptying of food from the pouch and causes a feeling of fullness.

Genotype: The entire genetic makeup of an individual. The fundamental constitution of an organism in terms of its hereditary factors. A group of organisms in which each has the same hereditary characteristics.

Glucose tolerance: The power of the normal liver to absorb and store large quantities of glucose and the effectiveness of intestinal absorption of glucose. The glucose tolerance test is a metabolic test of carbohydrate tolerance that measures active insulin, a hepatic function based on the ability of the liver to absorb glucose. The test consists of ingesting 100 grams of glucose into a fasting stomach; blood sugar should return to normal in 2 to 2½ hours after ingestion.

Hemoglobin A_{1c}: One of the fractions of glycosylated hemoglobin A. Glycosylated hemoglobin is formed when linkages of glucose and related monosaccharides bind to hemoglobin A and its concentration represents the average blood glucose level over the previous several weeks. HbA_{1C} levels are used as a measure of long-term control of plasma glucose (normal, 4 to 6 percent). In controlled diabetes mellitus, the concentration of glycosylated hemoglobin A is within the normal range, but in uncontrolled cases the level may be 3 to 4 times the normal concentration. Generally, complications are substantially lower among patients with HbA_{1C} levels of 7 percent or less than in patients with HbA_{1C} levels of 9 percent or more.

Hemorrhagic stroke: A disorder involving bleeding within ischemic brain tissue. Hemorrhagic stroke occurs when blood vessels that are damaged or dead from lack of blood supply (infarcted), located within an area of infarcted brain tissue, rupture and transform an "ischemic" stroke into a hemorrhagic stroke. Ischemia is inadequate tissue oxygenation caused by reduced blood flow; infarction is tissue death resulting from ischemia. Bleeding irritates the brain tissues, causing swelling (cerebral edema). Blood collects into a mass (hematoma). Both swelling and hematoma will compress and displace brain tissue.

Heritability: The proportion of observed variation in a particular trait that can be attributed to inherited genetic factors in contrast to environmental ones.

High-density lipoproteins (HDL): Lipoproteins that contain a small amount of cholesterol and carry cholesterol away from body cells and tissues to the liver for excretion from the body. Low-level HDL increases the risk of heart disease, so the higher the HDL level, the better. The HDL component normally contains 20 to 30 percent of total cholesterol, and HDL levels are inversely correlated with coronary heart disease risk.

Hirsutism: Presence of excessive body and facial hair, especially in women; may be present in normal adults as an expression of an ethnic characteristic or may develop in children or adults as the result of an endocrine disorder. Apert's hirsutism is caused by a virilizing disorder of adrenocortical origin. Constitutional hirsutism is mild-to-moderate hirsutism present in individuals exhibiting otherwise normal endocrine and reproductive functions; it appears to be an inheritable form of hirsutism and commonly is an expression of an ethnic characteristic. Idiopathic hirsutism is of uncertain origin in women, who may exhibit menstrual abnormalities and sterility. Some authorities believe the hirsutism reflects hypersecretion of adrenocortical androgens.

Hypercholesterolemia (high blood cholesterol): Cholesterol is the most abundant steroid in animal tissues, especially in bile and gallstones. The relationship between the intake of cholesterol and its manufacture by the body to its utiliza-

visited on 6/14/2019

tion, sequestration, or excretion from the body is called the cholesterol balance. When cholesterol accumulates, the balance is positive; when it declines, the balance is negative. In 1993, the NHLBI National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults issued an updated set of recommendations for monitoring and treatment of blood cholesterol levels. The NCEP guidelines recommended that total cholesterol levels and subfractions of high-density lipoprotein (HDL) cholesterol be measured beginning at age 20 in all adults, with subsequent periodic screenings as needed. Even in the group of patients at lowest risk for coronary heart disease (total cholesterol <200 mg/dL and HDL >35 mg/dL), the NCEP recommended that rescreening take place at least once every 5 years or upon physical examination.

Hypertension: High blood pressure (i.e., abnormally high blood pressure tension involving systolic and/or diastolic levels). The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure defines hypertension as a systolic blood pressure of 140 mm Hg or greater, a diastolic blood pressure of 90 mm Hg or greater, or taking hypertensive medication. The cause may be adrenal, benign, essential, Goldblatt's, idiopathic, malignant PATE, portal, postpartum, primary, pulmonary, renal or renovascular.

Hypertriglyceridemia: An excess of triglycerides in the blood that is an autosomal dominant disorder with the phenotype of hyperlipoproteinemia, type IV. The National Cholesterol Education Program defines a high level of triglycerides as being between 400 and 1,000 mg/dL.

Incidence: The rate at which a certain event occurs (i.e., the number of new cases of a specific disease occurring during a certain period).

Insulin-dependent diabetes mellitus (type I diabetes): A disease characterized by high levels of blood glucose resulting from defects in insulin secretion, insulin action, or both. Autoimmune, genetic, and environmental factors are involved in the development of type I diabetes.

Ischemic stroke: A condition in which the blood supply to part of the brain is cut off. Also called "plug-type" strokes. Blocked arteries starve areas of the brain controlling sight, speech, sensation, and movement so that these functions are partially or completely lost. Ischemic stroke is the most common type of stroke, accounting for 80 percent of all strokes. Most ischemic strokes are caused by a blood clot called a thrombus, which blocks blood flow in the arteries feeding the brain, usually the carotid artery in the neck, the major vessel bringing blood to the brain. When it becomes blocked, the risk of stroke is very high.

Jejuno-ileostomy: See gastroplasty.

J-shaped relationship: The relationship between body weight and mortality.

Lipoprotein: Protein-coated packages that carry fat and cholesterol throughout the bloodstream. There are four general classes: high-density, low-density, very low-density, and chylomicrons.

Locus/loci: A general anatomical term for a site in the body or the position of a gene on a chromosome.

Longitudinal study: Also referred to as a "cohort study" or "prospective study"; the analytic method of epidemiologic study in which subsets of a defined population can be identified who are, have been, or in the future may be exposed or not exposed, or exposed in different degrees, to a factor or factors hypothesized to influence the probability of occurrence of a given disease or other outcome. The main feature of this type of study is to observe large numbers of subjects over an extended time, with comparisons of

visited on 6/14/2019

incidence rates in groups that differ in exposure levels.

Low-calorie diet (LCD): Caloric restriction of about 800 to 1,500 calories (approximately 12 to 15 kcal/kg of body weight) per day.

Low-density lipoprotein (LDL): Lipoprotein that contains most of the cholesterol in the blood. LDL carries cholesterol to the tissues of the body, including the arteries. A high level of LDL increases the risk of heart disease. LDL typically contains 60 to 70 percent of the total serum cholesterol and both are directly correlated with CHD risk.

Lower-fat diet: An eating plan in which 30 percent or less of the day's total calories are from fat.

Macronutrients: Nutrients in the diet that are the key sources of energy, namely protein, fat, and carbohydrates.

Magnetic resonance imaging (MRI): Magnetic resonance imaging uses radio frequency waves to provide direct visualization and quantification of fat. The sharp image contrast of MRI allows clear separation of adipose tissue from surrounding nonlipid structures. Essentially the same information provided by CT is available from MRI, including total body and regional adipose tissue, subcutaneous adipose, and estimates of various visceral adipose tissue components. The advantage of MRI is its lack of ionizing radiation and hence its presumed safety in children, younger adults, and pregnant women. The minimal present use of MRI can be attributed to the expense, limited access to instrumentation, and long scanning time.

Menopause: The cessation of menstruation in the human female, which begins at about the age of 50.

Meta-analysis: Process of using statistical methods to combine the results of different studies. A frequent application is pooling the results from a set of randomized controlled trials, none of which alone is powerful enough to demonstrate statistical significance.

Mianserine: An antidepressant sometimes used in the pharmacotherapy of bulimia nervosa.

Midaxillary line: An imaginary vertical line that passes midway between the anterior and posterior axillary (armpit) folds.

Monounsaturated fat: An unsaturated fat that is found primarily in plant foods, including olive and canola oils.

Myocardial infarction (MI): Gross necrosis of the myocardium as a result of interruption of the blood supply to the area; it is almost always caused by atherosclerosis of the coronary arteries, upon which coronary thrombosis is usually superimposed.

NHANES: National Health and Nutrition Examination Survey; conducted every 10 years by the National Center for Health Statistics to survey the dietary habits and health of U.S. residents.

Neural tube defects: These defects include problems stemming from fetal development of the spinal cord, spine, brain, and skull, and include birth defects such as spina bifida, anencephaly, and encephalocele. Neural tube defects occur early in pregnancy at about 4 to 6 weeks, usually before a woman knows she is pregnant. Many babies with neural tube defects have difficulty walking and with bladder and bowel control.

Neuronal atrophy: Nerve cell death and functional loss.

Obesity: The condition of having an abnormally high proportion of body fat. Defined as a body mass index (BMI) of greater than or equal to 30. Subjects are generally classified as obese when body fat content exceeds 30 percent in women and 25 percent in men. The operational definition of obesity in this document is a BMI ≥ 30.

visited on 6/14/2019

Observational study: An epidemiologic study that does not involve any intervention, experimental or otherwise. Such a study may be one in which nature is allowed to take its course, with changes in one characteristic being studied in relation to changes in other characteristics. Analytical epidemiologic methods, such as case-control and cohort study designs, are properly called observational epidemiology because the investigator is observing without intervention other than to record, classify, count, and statistically analyze results.

Orlistat: A lipase inhibitor used for weight loss. Lipase is an enzyme found in the bowel that assists in lipid absorption by the body. Orlistat blocks this enzyme, reducing the amount of fat the body absorbs by about 30 percent. It is known colloquially as a "fat blocker." Because more oily fat is left in the bowel to be excreted, Orlistat can cause an oily anal leakage and fecal incontinence. Orlistat may not be suitable for people with bowel conditions such as irritable bowel syndrome or Crohn's disease.

Osteoarthritis: Noninflammatory degenerative joint disease occurring chiefly in older persons, characterized by degeneration of the articular cartilage, hypertrophy of bone at the margins, and changes in the synovial membrane. It is accompanied by pain and stiffness.

Overweight: An excess of body weight but not necessarily body fat; a body mass index of 25 to 29.9 kg/m^2.

Peripheral regions: Other regions of the body besides the abdominal region (i.e., the gluteal-femoral area).

Pharmacotherapy: A regimen of using appetite suppressant medications to manage obesity by decreasing appetite or increasing the feeling of satiety. These medications decrease appetite by increasing serotonin or catecholamine—two brain chemicals that affect mood and appetite.

Phenotype: The entire physical, biochemical, and physiological makeup of an individual as determined by his or her genes and by the environment in the broad sense.

Phentermine: An adrenergic isomeric with amphetamine, used as an anorexic; administered orally as a complex with an ion-exchange resin to produce a sustained action.

Polyunsaturated fat: An unsaturated fat found in greatest amounts in foods derived from plants, including safflower, sunflower, corn, and soybean oils.

Postprandial plasma blood glucose: Glucose tolerance test performed after ingesting food.

Prevalence: The number of events, e.g., instances of a given disease or other condition, in a given population at a designated time. When used without qualification, the term usually refers to the situation at specific point in time (point prevalence). Prevalence is a number not a rate.

Prospective study: An epidemiologic study in which a group of individuals (a cohort), all free of a particular disease and varying in their exposure to a possible risk factor, is followed over a specific amount of time to determine the incidence rates of the disease in the exposed and unexposed groups.

Protein: A class of compounds composed of linked amino acids that contain carbon, hydrogen, nitrogen, oxygen, and sometimes other atoms in specific configurations.

Randomization: Also called random allocation. Is allocation of individuals to groups, e.g., for experimental and control regimens, by chance. Within the limits of chance variation, random allocation should make the control and experimental groups similar at the start of an investigation and ensure that personal judgment and prejudices of the investigator do not influence allocation.

visited on 6/14/2019

Randomized clinical trial (RCT): An epidemiologic experiment in which subjects in a population are randomly allocated into groups, usually called study and control groups, to receive or not to receive an experimental prevention or therapeutic product, maneuver, or intervention. The results are assessed by rigorous comparison of rates of disease, death recovery, or other appropriate outcome in the study and control groups, respectively. RCTs are generally regarded as the most scientifically rigorous method of hypothesis testing available in epidemiology.

Recessive gene: A gene that is phenotypically expressed only when homozygous.

Refractory obesity: Obesity that is resistant to treatment.

Relative risk: The ratio of the incidence rate of a disease among individuals exposed to a specific risk factor to the incidence rate among unexposed individuals; synonymous with risk ratio. Alternatively, the ratio of the cumulative incidence rate in the exposed to the cumulative incidence rate in the unexposed (cumulative incidence ratio). The term relative risk has also been used synonymously with odds ratio. This is because the odds ratio and relative risk approach each other if the disease is rare (<5 percent of population) and the number of subjects is large.

Resting metabolic rate (RMR): RMR accounts for 65 to 75 percent of daily energy expenditure and represents the minimum energy needed to maintain all physiological cell functions in the resting state. The principal determinant of RMR is lean body mass (LBM). Obese subjects have a higher RMR in absolute terms than lean individuals, an equivalent RMR when corrected for LBM and per unit surface area, and a lower RMR when expressed per kilogram of body weight. Obese persons require more energy for any given activity because of a larger mass, but they tend to be more sedentary than lean subjects.

Risk: The probability that an event will occur. Also, a nontechnical term encompassing a variety of measures of the probability of a generally unfavorable outcome.

Roux-en Y bypass: See gastric exclusion; the most common gastric bypass procedure.

Saturated fat: A type of fat found in greatest amounts in foods from animals, such as fatty cuts of meat, poultry with the skin, whole-milk dairy products, lard, and in some vegetable oils, including coconut, palm kernel, and palm oils. Saturated fat raises blood cholesterol more than anything else eaten. On a Step I Diet, no more than 8 to 10 percent of total calories should come from saturated fat, and in the Step II Diet, less than 7 percent of the day's total calories should come from saturated fat.

Secular trends: A relatively long-term trend in a community or country.

Serotonin: A monoamine vasoconstrictor, found in various animals from coelenterates to vertebrates, in bacteria, and in many plants. In humans, it is synthesized in the intestinal chromaffin cells or in the central or peripheral neurons and is found in high concentrations in many body tissues, including the intestinal mucosa, pineal body, and central nervous system. Produced enzymatically from tryptophan by hydroxylation and decarboxylation, serotonin has many physiologic properties (e.g., inhibits gastric secretion, stimulates smooth muscle, serves as central neurotransmitter, and is a precursor of melatonin).

Sibutramine: A drug used for the management of obesity that helps reduce food intake and is indicated for weight loss and maintenance of weight loss when used in conjunction with a reduced-calorie diet. It works to suppress the appetite primarily by inhibiting the reuptake of the neurotransmitters norepinephrine and serotonin. Side effects include dry mouth, headache,

visited on 6/14/2019

constipation, insomnia, and a slight increase in average blood pressure. In some patients it causes a higher blood pressure increase.

Sleep apnea: A serious, potentially life-threatening breathing disorder characterized by repeated cessation of breathing due to either collapse of the upper airway during sleep or absence of respiratory effort.

Social pressure: A strategy used in behavior therapy in which individuals are told that they possess the basic self-control ability to lose weight, but that coming to group meetings will strengthen their abilities. The group is asked to listen and give advice, similar to the way many self-help groups, based on social support, operate.

Stoma size: The size of a new opening created surgically between two body structures.

Stress incontinence: An involuntary loss of urine that occurs at the same time that internal abdominal pressure is increased, such as with laughing, sneezing, coughing, or physical activity.

Stress management: A set of techniques used to help an individual cope more effectively with difficult situations in order to feel better emotionally, improve behavioral skills, and often to enhance feelings of control. Stress management may include relaxation exercises, assertiveness training, cognitive restructuring, time management, and social support. It can be delivered either on a one-to-one basis or in a group format.

Stroke: Sudden loss of function of part of the brain because of loss of blood flow. Stroke may be caused by a clot (thrombosis) or rupture (hemorrhage) of a blood vessel to the brain.

Submaximal heart rate test: Used to determine the systematic use of physical activity. The submaximal work levels allow work to be increased in small increments until cardiac manifestations such as angina pain appear. This provides a more precise manipulation of workload and gives a reliable and quantitative index of a person's functional impairment if heart disease is detected.

Surgical procedures: See jejuno-ileostomy, gastroplasty, gastric bypass, gastric partitioning, gastric exclusion, Roux-en Y bypass and gastric bubble.

Systolic blood pressure: The maximum pressure in the artery produced as the heart contracts and blood begins to flow.

Triglyceride: A lipid carried through the bloodstream to tissues. Most of the body's fat tissue is in the form of triglycerides, stored for use as energy. Triglycerides are obtained primarily from fat in foods.

Type 2 diabetes mellitus: Usually characterized by a gradual onset with minimal or no symptoms of metabolic disturbance and no requirement for exogenous insulin. The peak age of onset is 50 to 60 years. Obesity and possibly a genetic factor are usually present.

Validity: The degree to which the inferences drawn from study results, especially generalization extending beyond the study sample, are warranted when account is taken of the study methods, the representativeness of the study sample, and the nature of the population from which it is drawn.

Vertical banded gastroplasty: A surgical treatment for extreme obesity; an operation on the stomach that involves constructing a small pouch in the stomach that empties through a narrow opening into the distal stomach and duodenum.

Very low-calorie diet (VLCD): The VLCD of 800 (approximately 6-10 kcal/kg body weight) or fewer calories per day is conducted under physician supervision and monitoring and is restricted to severely obese persons.

visited on 6/14/2019

Very low-density lipoprotein (VLDL): The lipoprotein particles that initially leave the liver, carrying cholesterol and lipid. VLDLs contain 10 to 15 percent of the total serum cholesterol along with most of the triglycerides in the fasting serum; VLDLs are precursors of LDL, and some forms of VLDL, particularly VLDL remnants, appear to be atherogenic.

Visceral fat: One of the three compartments of abdominal fat. Retroperitoneal and subcutaneous are the other two compartments.

VO_2 max: Maximal oxygen uptake is known as VO_2 max and is the maximal capacity for oxygen consumption by the body during maximal exertion. It is used as an indicator of cardiorespiratory fitness.

Waist circumference: To define the level at which the waist circumference is measured, a bony landmark is first located and marked. The subject stands, and the technician, positioned to the right of the subject, palpates the upper hip bone to locate the right ileum. Just above the uppermost lateral border of the right ileum, a horizontal mark is drawn and then crossed with a vertical mark on the midaxillary line. The measuring tape is then placed around the trunk, at the level of the mark on the right side, making sure that it is on a level horizontal plane on all sides. The tape is then tightened slightly without compressing the skin and underlying subcutaneous tissues. The measure is recorded in centimeters to the nearest millimeter.

Waist-hip-ratio (WHR): The ratio of a person's waist circumference to hip circumference. WHR looks at the relationship between the differences in the measurements of waist and hips. Most people store body fat in two distinct ways, often called the “apple” and “pear” shapes, either the middle (apple) or the hips (pear). For most people, carrying extra weight around their middle increases health risks more than carrying extra weight around their hips or thighs. Overall obesity, however, is still of greater risk than body fat storage locations or WHR. A WHR ≥ 1.0 is in the danger zone, with risks of heart disease and other ailments connected with being overweight. For men, a ratio of .90 or less is considered safe, and for women .80 or less.

Yohimbine: An alkaloid that possesses adrenergic-blocking properties and is used in arteriosclerosis and angina pectoris, formerly used as a local anesthetic and mydriatic and for its purported aphrodisiac properties.

visited on 6/14/2019

List of Abbreviations

AAFP	American Academy of Family Physicians
ADA	American Diabetes Association
AHCPR	Agency for Health Care Policy and Research
AHI	Apnea and Hypopnea Index
ATP II	Second Report of the Expert Panel on the Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II)
BED	Binge Eating Disorder
BMI*	Body Mass Index
BN	Bulimia Nervosa
CAD	Coronary Artery Disease
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CHD*	Coronary Heart Disease
CHF	Congestive Heart Failure
CPAP	Continuous Positive Airway Pressure
CRSS	Critical Review Status Sheet
CT*	Computed Tomography
CVD	Cardiovascular Disease
DEXA*	Dual-Energy X-ray Absorpiometry
DNA	Deoxyribonucleic Acid
FDA	Food and Drug Administration
HDL*	High-Density Lipoprotein
HHANES	Hispanic Health and Nutrition Examination Survey
HMO	Health Maintenance Organization
IGT	Impaired Glucose Tolerance
INDEX trial	International Dexfenfluramine Study
INTERSALT	International study of SALT
IOM	Institute of Medicine
JNC VI	Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
LCD*	Low-Calorie Diets
LDL*	Low-Density Lipoprotein
MEDLINE	MEDLARS (Medical Literature Analysis and Retrieval System) On-Line
MeSH	Medical Subject Headings
MMPI	Minnesota Multiphasic Personality Inventory
MRI*	Magnetic Resonance Imaging
NAASO	North American Association for the Study of Obesity

visited on 6/14/2019

NCEP	National Cholesterol Education Program
NCHS	National Center for Health Statistics (CDC)
NHANES*	National Health and Nutrition Examination Survey
NHBPEP	National High Blood Pressure Education Program
NHES	National Health Examination Survey
NHIS	National Health Interview Survey
NHLBI	National Heart, Lung, and Blood Institute
NIDDK	National Institute of Diabetes and Digestive and Kidney Diseases
NIH	National Institutes of Health
NLM	National Library of Medicine
RCTs*	Randomized Controlled Trials
RDI	Respiratory Disturbance Index
SOS	Swedish Obesity Study
SSRIs	Selective Serotonin Reuptake Inhibitors
TAIM	Trial of Antihypertensive Interventions and Management
TOHP I	Trials of Hypertension Prevention (Phase 1)
TOHP II	Trials of Hypertension Prevention (Phase 2)
TONE	Trial of Nonpharmacologic Interventions in the Elderly
VLCD*	Very Low-Calorie Diets
VLDL*	Very Low-Density Lipoprotein
VO_2 max*	Oxygen Consumption
WHR*	Waist-to-Hip Ratio

*Terms defined in the glossary.

visited on 6/14/2019

Reference List

1. Kuczmarski RJ, Carrol MD, Flegal KM, Troiano RP. Varying body mass index cut-off points to describe overweight prevalence among U.S. adults: NHANES III (1988 to 1994). Obes Res. 1997;5:542-548.
2. Brown CD, Donato KA, Obarzanek, E, et al. Body mass index and prevalence of risk factors for cardiovascular disease. Obes Res. 1998; submitted for publication.
3. Association of Life Insurance Medical Directors of America. Society of Actuaries. Blood Pressure Study, 1979. Chicago: The Society; 1980.
4. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Weight and blood pressure. Findings in hypertension screening of 1 million Americans. JAMA. 1978;240:1607-1610.
5. Criqui MH, Mebane I, Wallace RB, Heiss G, Holdbrook MJ. Multivariate correlates of adult blood pressures in nine North American populations: The Lipid Research Clinics Prevalence Study. Prev Med. 1982;11:391-402.
6. Dyer AR, Elliott P. The INTERSALT study: relations of body mass index to blood pressure. INTERSALT Co-operative Research Group. J Hum Hypertens. 1989;3:299-308.
7. Westlund K, Nicolaysen R. Ten-year mortality and morbidity related to serum cholesterol. A follow-up of 3,751 men aged 40-49. Scand J Clin Lab Invest Suppl. 1972;127:1-24.
8. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. J Chronic Dis. 1979;32:563-576.
9. Larsson B, Bjorntorp P, Tibblin G. The health consequences of moderate obesity. Int J Obes. 1981;5:97-116.
10. Medalie JH, Papier C, Herman JB, et al. Diabetes mellitus among 10,000 adult men. I. 5-year incidence and associated variables. Isr J Med Sci. 1974;10:681-697.
11 Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation. 1983;67:968-977.
12. Rexrode KM, Hennekens CH, Willett WC, et al. A prospective study of body mass index, weight change, and risk of stroke in women. JAMA. 1997;277:1539-1545.
13. Walker SP, Rimm EB, Ascherio A, Kawachi I, Stampfer MJ, Willett WC. Body size and fat distribution as predictors of stroke among US men. Am J Epidemiol. 1996;144:1143-1150.
14. Stampfer MJ, Maclure KM, Colditz GA, Manson JE, Willett WC. Risk of sympto-

visited on 6/14/2019

matic gallstones in women with severe obesity. Am J Clin Nutr. 1992;55:652-658.

15. Khare M, Everhart JE, Maurer KR, Hill MC. Association of ethnicity and body mass index (BMI) with gallstone disease in the United States. Am J Epidemiol. 1995;141:S69.

16. Cicuttini FM, Baker JR, Spector TD. The association of obesity with osteoarthritis of the hand and knee in women: a twin study. J Rheumatol. 1996;23:1221-1226.

17. Hart DJ, Spector TD. The relationship of obesity, fat distribution, and osteoarthritis in women in the general population: the Chingford Study. J Rheumatol. 1993;20:331-335.

18. Hochberg MC, Lethbridge-Cejku M, Scott WW Jr, Reichle R, Plato CC, Tobin JD. The association of body weight, body fatness and body fat distribution with osteoarthritis of the knee: data from the Baltimore Longitudinal Study of Aging. J Rheumatol. 1995;22:488-493.

19. Millman RP, Carlisle CC, McGarvey ST, Eveloff SE, Levinson PD. Body fat distribution and sleep apnea severity in women. Chest. 1995;107:362-366.

20. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med. 1993;328:1230-1235.

21. Shepard JW Jr. Hypertension, cardiac arrhythmias, myocardial infarction, and stroke in relation to obstructive sleep apnea. Clin Chest Med. 1992;13:437-458.

22. Bostick RM, Potter JD, Kushi LH, et al. Sugar, meat, and fat intake, and nondietary risk factors for colon cancer incidence in Iowa women (United States). Cancer Causes Control. 1994;5:38-52.

23. Chute CG, Willett WC, Colditz GA, et al. A prospective study of body mass, height, and smoking on the risk of colorectal cancer in women. Cancer Causes Control. 1991;2:117-124.

24. Garland C, Shekelle RB, Barrett-Connor E, Criqui MH, Rossof AH, Paul O. Dietary vitamin D and calcium and risk of colorectal cancer: a 19-year prospective study in men. Lancet. 1985;1:307-309.

25. McGinnis JM, Foege WH. Actual causes of death in the United States. JAMA. 1993;270:2207-2212.

26. Broussard BA, Sugarman JR, Bachman-Carter K, et al. Toward comprehensive obesity prevention programs in Native American communities. Obes Res. 1995;3:2895-2975.

27. Najjar MF, Kuczmarski RJ. Anthropometric data and prevalence of overweight for Hispanics: 1982-1984. National Center for Health Statistics. Vital Health Stat [11]. 1989;239:1-106.

28. World Health Organization. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser. 1995;854:1- 452.

29. VanItallie TB, Lew EA. Overweight and Underweight. In: Lew EA, Gajewski J, eds. Medical Risks: Trends in Mortality by Age and Timed Elapsed. Vol 1. New York: Praeger; 1990: Chapter 13.

30. VanItallie TB. Health implications of overweight and obesity in the United States. Ann Intern Med. 1985;103:983-988.

31. Manson JE, Stampfer MJ, Hennekens CH, Willett WC. Body weight and longevity. A reassessment. JAMA. 1987;257:353-358.

visited on 6/14/2019

32. Troiano RP, Frongillo EA Jr, Sobal J, Levitsky DA. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. Int J Obes Relat Metab Disord. 1996;20:63-75.

33. National Research Council. Committee on Diet and Health. Implications for Reducing Chronic Disease Risk. Washington, DC: National Academy Press; 1989.

34. Forster JL, Jeffery RW, Schmid TL, Kramer FM. Preventing weight gain in adults: a pound of prevention. Health Psychol. 1988;7:515-525.

35. Lenfant C. NHLBI Clinical Guidelines: another look. Circulation. 1995;91:617-618.

36. Dickersin K, Min YI, Meinert CL. Factors influencing publication of research results. Follow-up of applications submitted to two institutional review boards. JAMA. 1992;267:374-378.

37. Dickersin K. The existence of publication bias and risk factors for its occurrence. JAMA. 1990;263:1385-1389.

38. Dickersin K, Min YI. Publication bias: the problem that won't go away. Ann N Y Acad Sci. 1993;703:135-146.

39. Allison DB, Faith MS, Gorman BS. Publication bias in obesity treatment trials? Int J Obes Relat Metab Disord. 1996;20:931-937.

40. Bray GA. Obesity in Perspective: a Conference. John E. Fogarty International Center for Advanced Study in the Health Sciences. Washington, DC: Govt Print Office; 1975. DHEW publication no. (NIH) 75-708.

41. Bray GA. Obesity in America. An overview of the Second Fogarty International Center conference on obesity. Int J Obes. 1979;3:363-375.

42. Health implications of obesity. National Institutes of Health Consensus Development Conference Statement. Ann Intern Med. 1985;103:1073-1077.

43. U.S. Department of Health and Human Services. Healthy People 2000: National health promotion and disease prevention objectives: Washington, DC: Public Health Service; 1991. DHHS publication no. (PHS) 91-50213.

44. Thomas PR, ed. Weighing the Options. Criteria for Evaluating Weight-Management Programs. Washington, DC: National Academy Press; 1995.

45. Najjar MF, Rowland M. Anthropometric reference data and prevalence of overweight, United States, 1976-80. Vital Health Stat [11]. 1987;238:1-73.

46. Wolf AM, Colditz GA. Social and economic effects of body weight in the United States. Am J Clin Nutr. 1996;63:466S-469S.

47. Flynn MA, Nolph GB, Baker AS, Krause G. Aging in humans: a continuous 20-year study of physiologic and dietary parameters. J Am Coll Nutr. 1992;11:660-672.

48. Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960-1994. Int J Obes. 1998;22:39-47.

49. Centers for Disease Control. Prevalence of overweight among adolescents—United States, 1988-91. MMWR Morb Mortal Wkly Rep. 1994;43:818-821.

50. McGarvey ST. Obesity in Samoans and a perspective on its etiology in Polynesians. Am J Clin Nutr. 1991;53:1586S-1594S.

visited on 6/14/2019

51. Klatsky AL, Armstrong MA. Cardiovascular risk factors among Asian Americans living in northern California. Am J Public Health. 1991;81:1423-1428.

52. Welty TK, Lee ET, Yeh J, et al. Cardiovascular disease risk factors among American Indians. The Strong Heart Study. Am J Epidemiol. 1995;142:269-287.

53. Leigh JP, Fries JF, Hubert HB. Gender and race differences in the correlation between body mass and education in the 1971-1975 NHANES I. J Epidemiol Community Health. 1992;46:191-196.

54. Kuczmarski RJ. Prevalence of overweight and weight gain in the United States. Am J Clin Nutr. 1992;55:495S-502S.

55. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults. The National Health and Nutrition Examination Surveys, 1960 to 1991. JAMA. 1994;272:205-211.

56. Pi-Sunyer FX. Medical hazards of obesity. Ann Intern Med. 1993;119:655-660.

57. Seidell JC. The impact of obesity on health status: some implications for health care costs. Int J Obes Relat Metab Disord. 1995;19 (Suppl 6):S13-S16.

58. Wolf AM, Colditz GA. Current estimates of the economic costs of obesity in the United States. Obes Res. 1998;6:97-106.

59. Wolf AM, Colditz GA. The cost of obesity: the U.S. perspective. Pharmacoeconomics. 1994;5:34-37.

60. Colditz GA. Economic costs of obesity. Am J Clin Nutr. 1992;55:503S-507S.

61. Levy E, Levy P, Le Pen C, Basdevant A. The economic cost of obesity: the French situation. Int J Obes Relat Metab Disord. 1995;19:788-792.

62. Segal L, Carter R, Zimmet P. The cost of obesity - the Australian perspective. Pharmacoeconomics. 1994;5:45-52.

63. Seidell JC. Obesity in Europe. Obes Res. 1995;3:89S-93S.

64. National Task Force on Prevention and Treatment of Obesity. Towards Prevention of Obesity: Research Directions. Obes Res. 1994;2:571-584.

65. World Health Organization. Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation of Obesity. Geneva, 3-5 June 1997.

66. Winkleby MA, Feldman HA, Murray DM. Joint analysis of three U.S. community intervention trials for reduction of cardiovascular disease risk. J Clin Epidemiol. 1997;50:645-658.

67. Carleton RA, Lasater TM, Assaf AR, Feldman HA, McKinlay S. The Pawtucket Heart Health Program: community changes in cardiovascular risk factors and projected disease risk. Am J Public Health. 1995;85:777-785.

68. Davis SM. Cardiovascular Health Promotion Project. American Indian Program Recaptures Past to Improve Children's Future (Pathways). Heartmemo (Special Edition). 1996:25-27.

69. Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year followup of behavioral, family-based treatment for obese children. JAMA. 1990;264:2519-2523.

70. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. Diabetes Care. 1997;20:537-544.

71. James WP. A public health approach to the problem of obesity. Int J Obes Relat Metab Disord. 1995;19 (Suppl 3):S37-S45.

visited on 6/14/2019

72. Jeffery RW. Population perspectives on the prevention and treatment of obesity in minority populations. Am J Clin Nutr. 1991;53 (6 Suppl):1621S-1624S.

73. Lewin-Epstein N. Determinants of regular source of health care in Black, Mexican, Puerto Rican, and non-Hispanic White population. Med Care. 1991;29:543-557.

74. O'Hare WP. America's Minorities—The demographics of diversity. Population Bulletin. Washington, DC: Population Reference Bureau, Inc.; 1992;47:4.

75. Link BG, Phelan JC. Understanding sociodemographic differences in health—the role of fundamental social causes. Am J Public Health. 1996;86:471-473.

76. Ballantyne D, Devine BL, Fife R. Interrelation of age, obesity, cigarette smoking, and blood pressure in hypertensive patients. BMJ. 1978;1:880-881.

77. Brennan PJ, Simpson JM, Blacket RB, McGilchrist CA. The effects of body weight on serum cholesterol, serum triglycerides, serum urate, and systolic blood pressure. Aust N Z J Med. 1980;10:15-20.

78. Havlik RJ, Hubert HB, Fabsitz RR, Feinleib M. Weight and hypertension. Ann Intern Med. 1983;98:855-859.

79. Loggie JM, Horan MJ, Hohn AR, Gruskin AB, Dunbar JB, Havlik RJ. Juvenile hypertension: highlights of a workshop. J Pediatr. 1984;104:657-663.

80. MacMahon SW, Blacket RB, Macdonald GJ, Hall W. Obesity, alcohol consumption and blood pressure in Australian men and women. The National Heart Foundation of Australia Risk Factor Prevalence Study. J Hypertens. 1984;2:85-91.

81. Colditz GA, Willett WC, Stampfer MJ, et al. Weight as a risk factor for clinical diabetes in women. Am J Epidemiol. 1990;132:501-513.

82. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care. 1994;17:961-969.

83. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med. 1995;122:481-486.

84. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of US adults. Am J Epidemiol. 1997;146:214-222.

85. Despres JP, Nadeau A, Tremblay A, et al. Role of deep abdominal fat in the association between regional adipose tissue distribution and glucose tolerance in obese women. Diabetes. 1989;38:304-309.

86. Haffner SM, Mitchell BD, Hazuda HP, Stern MP. Greater influence of central distribution of adipose tissue on incidence of non-insulin-dependent diabetes in women than men. Am J Clin Nutr. 1991;53:1312-1317.

87. Sparrow D, Borkan GA, Gerzof SG, Wisniewski C, Silbert CK. Relationship of fat distribution to glucose tolerance. Results of computed tomography in male participants of the Normative Aging Study. Diabetes. 1986;35:411-415.

88. Lundgren H, Bengtsson C, Blohme G, Lapidus L, Sjostrom L. Adiposity and adipose tissue distribution in relation to incidence of diabetes in women: results from a prospective population study in Gothenburg, Sweden. Int J Obes. 1989;13:413-423.

89. Ohlson LO, Larsson B, Svardsudd K, et al.

visited on 6/14/2019

The influence of body fat distribution on the incidence of diabetes mellitus. 13.5 years of follow-up of the participants in the study of men born in 1913. Diabetes. 1985;34:1055-1058.

90. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women. Risk within the 'normal' weight range. JAMA. 1995;273:461-465.

91. Carman WJ, Sowers M, Hawthorne VM, Weissfeld LA. Obesity as a risk factor for osteoarthritis of the hand and wrist: a prospective study. Am J Epidemiol. 1994;139:119-129.

92. Felson DT, Anderson JJ, Naimark A, Walker AM, Meenan RF. Obesity and knee osteoarthritis. The Framingham Study. Ann Intern Med. 1988;109:18-24.

93. Felson DT, Zhang Y, Anthony JM, Naimark A, Anderson JJ. Weight loss reduces the risk for symptomatic knee osteoarthritis in women. The Framingham Study. Ann Intern Med. 1992;116:535-539.

94. Felson DT. Weight and osteoarthritis. J Rheumatol Suppl. 1995;43:7-9.

95. McGoey BV, Deitel M, Saplys RJ, Kliman ME. Effect of weight loss on musculoskeletal pain in the morbidly obese. J Bone Joint Surg [Br]. 1990;72-B:322-323.

96. Chua W, Chediak AD. Obstructive sleep apnea. Treatment improves quality of life—and may prevent death. Postgrad Med. 1994;95:123-126, 131, 135-138.

97. Loube DI, Loube AA, Mitler MM. Weight loss for obstructive sleep apnea: the optimal therapy for obese patients. J Am Diet Assoc. 1994;94:1291-1295.

98. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnoea syndrome. Eur Respir J. 1990;3:509-514.

99. Giovannucci E, Ascherio A, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Physical activity, obesity, and risk for colon cancer and adenoma in men. Ann Intern Med. 1995;122:327-334.

100. Graham S, Marshall J, Haughey B, et al. Dietary epidemiology of cancer of the colon in western New York. Am J Epidemiol. 1988;128:490-503.

101. Klatsky AL, Armstrong MA, Friedman GD, Hiatt RA. The relations of alcoholic beverage use to colon and rectal cancer. Am J Epidemiol. 1988;128:1007-1015.

102. Lee IM, Paffenbarger RS Jr. Quetelet's index and risk of colon cancer in college alumni. J Natl Cancer Inst. 1992;84:1326-1331.

103. Le Marchand L, Wilkens LR, Mi MP. Obesity in youth and middle age and risk of colorectal cancer in men. Cancer Causes Control. 1992;3:349-354.

104. Martinez ME, Giovannucci E, Spiegelman D, et al. Physical activity, body size, and colorectal cancer in women. Am J Epidemiol. 1996:146-S73.

105. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med. 1992;327:1350-1355.

106. Phillips RL, Snowdon DA. Dietary relationships with fatal colorectal cancer among Seventh-Day Adventists. J Natl Cancer Inst. 1985;74:307-317.

107. Giovannucci E, Colditz GA, Stampfer MJ,

visited on 6/14/2019

Willett WC. Physical activity, obesity, and risk of colorectal adenoma in women (United States). Cancer Causes Control. 1996;7:253- 263.

108. Giovannucci E. Insulin and colon cancer. Cancer Causes Control. 1995;6:164-179.

109. Chu SY, Lee NC, Wingo PA, Senie RT, Greenberg RS, Peterson HB. The relationship between body mass and breast cancer among women enrolled in the Cancer and Steroid Hormone Study. J Clin Epidemiol. 1991;44:1197-1206.

110. Helmrich SP, Shapiro S, Rosenberg L, et al. Risk factors for breast cancer. Am J Epidemiol. 1983;117:35-45.

111. Rosenberg L, Palmer JR, Miller DR, Clarke EA, Shapiro S. A case-control study of alcoholic beverage consumption and breast cancer. Am J Epidemiol. 1990;131:6-14.

112. Willett WC, Browne ML, Bain C, et al. Relative weight and risk of breast cancer among premenopausal women. Am J Epidemiol. 1985;122:731-740.

113. Hunter DJ, Willett WC. Diet, body size, and breast cancer. Epidemiol Rev. 1993;15:110-132.

114. Huang Z, Hankinson SE, Colditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. JAMA. 1997;278:1407-1411.

115. Schottenfeld D, Fraumeni JF. Cancer Epidemiology and Prevention. New York: Oxford University Press, 1996.

116. Hartz AJ, Barboriak PN, Wong A, Katayaa KP, Rimm AA. The association of obesity with infertility and related menstrual abnormalities in women. Int J Obes. 1979;3:57-73.

117. Rich-Edwards JW, Goldman MB, Willett WC, et al. Adolescent body mass index and infertility caused by ovulatory disorder. Am J Obstet Gynecol. 1994;171:171-177.

118. Dunaif A. Polycystic Ovary Syndrome. Boston: Blackwell Scientific Publications; 1992.

119. Garbaciak JA Jr., Richter M, Miller S, Barton JJ. Maternal weight and pregnancy complications. Am J Obstet Gynecol. 1985;152:238-245.

120. Keppel KG, Taffel SM. Pregnancy-related weight gain and retention: implications of the 1990 Institute of Medicine guidelines. Am J Public Health. 1993;83:1100-1103.

121. Smith DE, Lewis CE, Caveny JL, Perkins LL, Burke GL, Bild DE. Longitudinal changes in adiposity associated with pregnancy. The CARDIA Study. Coronary Artery Risk Development in Young Adults Study. JAMA. 1994;271:1747-1751.

122. Johnson SR, Kolberg BH, Varner MW, Railsback LD. Maternal obesity and pregnancy. Surg Gynecol Obstet. 1987;164:431-437.

123. Prentice A, Goldberg G. Maternal obesity increases congenital malformations. Nutr Rev. 1996;54:146-152.

124. Stunkard AJ, Wadden TA. Psychological aspects of human obesity. In: Bjorntorp P, Brodoff BN, eds. Obesity. Philadelphia: Lippincott; 1992:352-360.

125. Brownell KD, Fairburn C. Psychosocial consequences of obesity. In: Stunkard AJ, Sobal JG, eds. Eating Disorders and Obesity: A Comprehensive Handbook. New York: Guilford Press; 1995:417-421.

126. Wadden TA, Stunkard AJ. Psychosocial consequences of obesity and dieting-research and clinical findings. In: Stunkard

visited on 6/14/2019

AJ, Wadden TA, eds. Obesity Theory and Therapy. New York: Raven Press; 1993:163-177 .

127. Faith MS, Allison DB. Assessment of psychological status among obese persons. In: Thompson JK, ed. Body Image, Eating Disorders, and Obesity: An Integrative Guide For Assessment and Treatment. Washington, DC: American Psychological Association; 1996:365-387.

128. Allon N. The stigma of overweight in everyday life. In: Wolman BB, DeBerry S, eds. Psychological Aspects of Obesity: A Handbook. New York: Van Nostrand Reinhold; 1982:130-174.

129. Feinleib M. Epidemiology of obesity in relation to health hazards. Ann Intern Med. 1985;103:1019-1024.

130. Garrison RJ, Castelli WP. Weight and 30-year mortality of men in the Framingham Study. Ann Intern Med. 1985;103:1006-1009.

131. Rabkin SW, Mathewson FA, Hsu PH. Relation of body weight to development of ischemic heart disease in a cohort of young North American men after a 26 year observation period: the Manitoba Study. Am J Cardiol. 1977;39:452-458.

132. Cutler JA, Psaty BM, MacMahon S, Furberg CD. Public health issues in hypertension control: what has been learned from clinical trials. In: Laragh JH, Brenner BM, eds. Hypertension: Pathophysiology, Diagnosis, and Management. New York: Raven Press; 1995:253-270.

133. Denke MA, Sempos CT, Grundy SM. Excess body weight. An underrecognized contributor to high blood cholesterol levels in white American men. Arch Intern Med. 1993;153:1093-1103.

134. Denke MA, Sempos CT, Grundy SM. Excess body weight. An underrecognized contributor to dyslipidemia in white American women. Arch Intern Med. 1994;154:401- 410.

135. Ashley FW Jr, Kannel WB. Relation of weight change to changes in atherogenic traits: the Framingham Study. J Chronic Dis. 1974;27:103-114.

136. Hershcopf RJ, Elahi D, Andres R, et al. Longitudinal changes in serum cholesterol in man: an epidemiologic search for an etiology. J Chronic Dis. 1982;35:101-114.

137. Shekelle RB, Shryock AM, Paul O, et al. Diet, serum cholesterol, and death from coronary heart disease. The Western Electric study. N Engl J Med. 1981;304:65-70.

138. Manson JE, Colditz GA, Stampfer MJ, et al. A prospective study of obesity and risk of coronary heart disease in women. N Engl J Med. 1990;322:882-889.

139. Reeder BA, Angel A, Ledoux M, Rabkin SW, Young TK, Sweet LE. Obesity and its relation to cardiovascular disease risk factors in Canadian adults. Canadian Heart Health Surveys Research Group. Can Med Assoc J. 1992;146:2009-2019.

140. Carlson LA, Lindstedt S. The Stockholm prospective study. 1. The initial values for plasma lipids. Acta Med Scand Suppl. 1968;493:1-135.

141. Mann JI, Lewis B, Shepherd J, et al. Blood lipid concentrations and other cardiovascular risk factors: distribution, prevalence, and detection in Britain. BMJ. 1988;296:1702-1706.

142. National Cholesterol Education Program. Second Report of the Expert Panel on Detection, Evaluation, and Treatment of

visited on 6/14/2019

High Blood Cholesterol in Adults (Adult Treatment Panel II). Circulation. 1994;89:1333-1445.

143. Glueck CJ, Taylor HL, Jacobs D, Morrison JA, Beaglehole R, Williams OD. Plasma high-density lipoprotein cholesterol: association with measurements of body mass. The Lipid Research Clinics Program Prevalence Study. Circulation. 1980;62:IV-62-69.

144. Garrison RJ, Wilson PW, Castelli WP, Feinleib M, Kannel WB, McNamara PM. Obesity and lipoprotein cholesterol in the Framingham offspring study. Metabolism. 1980;29:1053-1060.

145. Anderson KM, Wilson PWF, Garrison RJ, Castelli WP. Longitudinal and secular trends in lipoprotein cholesterol measurements in a general population sample: The Framingham offspring study. Atherosclerosis. 1987;68:59-66.

146. Law MR, Wald NJ, Thompson SG. By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease? BMJ. 1994;308:367-773.

147. Rydker PM, Hennekens CH, Stampfer MJ. A prospective study of lipoprotein (a) and the risk for myocardial infarction. JAMA. 1993;270:2195-2199.

148. Miller BD, Alderman EL, Haskell WL, Fair JM, Krauss RM. Predominance of dense low-density lipoprotein particles predicts angiograpic benefit of therapy in the Stanford coronary risk intervention study. Circulation. 1996;94:2146-2153.

149. Lamarche B, Tchernof A, Moorjani S, et al. Small, dense low-density lipoprotein particles as a predictor of the risk of ischemic heart disease in men. Prospective results from the Quebec Cardiovascular Study. Circulation. 1997;95:69-75.

150. Reaven GM, Chen YD, Jeppesen J, Maheux P, Krauss RM. Insulin resistance and hyperinsulinemia in individuals with small, dense low density lipoprotein particles. J Clin Invest. 1993;92:141-146.

151. Selby JV, Austin MA, Newman B, et al. LDL subclass phenotypes and the insulin resistance syndrome in women. Circulation. 1993;88:381-387.

152. Tchernof A, Lamarche B, Prud'Homme D, et al. The dense LDL phenotype: association with plasma lipoprotein levels, visceral obesity, and hyperinsulinemia in men. Diabetes Care. 1996;19: 629-637.

153. Knowler WC, Pettitt DJ, Savage PJ, Bennett PH. Diabetes incidence in Pima Indians: contributions of obesity and parental diabetes. Am J Epidemiol. 1981;113:144-156.

154. Lee ET, Howard BV, Savage PJ, et al. Diabetes and impaired glucose tolerance in three American Indian populations aged 45-74 years. The Strong Heart Study. Diabetes Care. 1995;18:599-610.

155. Higgins M, Kannel W, Garrison R, Pinsky J, Stokes J 3rd. Hazards of obesity—the Framingham experience. Acta Med Scand Suppl. 1988;723:23-36.

156. Donahue RP, Abbott RD. Central obesity and coronary heart disease in men. Lancet. 1987;2:1215.

157. Ducimetiere P, Richard JL. The relationship between subsets of anthropometric upper versus lower body measurements and coronary heart disease risk in middle-aged men. The Paris Prospective Study I. Int J Obes. 1989;13:111-121.

158. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjostrom L. Distribution of adipose tissue and risk of

visited on 6/14/2019

cardiovascular disease and death: a 12 year follow up of participants in the population study of women in Gothenburg, Sweden. BMJ. 1984;289:1257-1261.

159. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. BMJ. 1984;288:1401-1404.

160. Bjorntorp P. The associations between obesity, adipose tissue distribution, and disease. Acta Med Scand Suppl. 1988;723:121-134.

161. Fujioka S, Matsuzawa Y, Tokunaga K, Tarui S. Contribution of intra-abdominal fat accumulation to the impairment of glucose and lipid metabolism in human obesity. Metabolism. 1987;36:54-59.

162. Shaper AG, Wannamethee SG, Walker M. Body weight: implications for the prevention of coronary heart disease, stroke, and diabetes mellitus in a cohort study of middle aged men. BMJ. 1997;314:1311-1317.

163. Tokunaga K, Matsuzawa Y, Kotani K, et al. Ideal body weight estimated from the body mass index with the lowest morbidity. Int J Obes. 1991;15:1-5.

164. Jousilahti P, Tuomilehto J, Vartiainen E, Pekkanen J, Puska P. Body weight, cardiovascular risk factors, and coronary mortality. 15-year follow-up of middle-aged men and women in eastern Finland. Circulation. 1996;93:1372-1379.

165. DiBianco R. The changing syndrome of heart failure: an annotated review as we approach the 21st century. J Hypertens Suppl. 1994;12:S73-S87.

166. Kannel WB, Cupples A. Epidemiology and risk profile of cardiac failure. Cardiovasc Drugs Ther. 1988;2 (Suppl 1):387-395.

167. Savage MP, Krolewski AS, Kenien GG, Lebeis MP, Christlieb AR, Lewis SM. Acute myocardial infarction in diabetes mellitus and significance of congestive heart failure as a prognostic factor. Am J Cardiol. 1988;62:665-669.

168. Eriksson H, Svardsudd K, Larsson B, et al. Risk factors for heart failure in the general population: the study of men born in 1913. Eur Heart J. 1989;10:647-656.

169. Eriksson H, Wilhelmsen L, Caidahl K, Svardsudd K. Epidemiology and prognosis of heart failure. Z Kardiol. 1991;80 (Suppl 8):1-6.

170. Shimizu M, Isogai Y. Heart failure due to metabolic heart disorders. Nippon Rinsho. 1993;51:1362-1366.

171. Urbina EM, Gidding SS, Bao W, Pickoff AS, Berdusis K, Berenson GS. Effect of body size, ponderosity, and blood pressure on left ventricular growth in children and young adults in the Bogalusa Heart Study. Circulation. 1995;91:2400-2406.

172. Alpert MA, Hashimi MW. Obesity and the heart. Am J Med Sci. 1993;306:117-123.

173. Garavaglia GE, Messerli FH, Nunez BD, Schmieder RE, Grossman E. Myocardial contractility and left ventricular function in obese patients with essential hypertension. Am J Cardiol. 1988;62:594-597.

174. Ritter MM, Schraudolph M, Richter WO, Herbert M, Wiebecke B, Schwandt P. Obesity, heart failure and pulmonary insufficiency in a 26-year-old female. Med Klin.1990;85:371- 375.

175. Huang C, Ross PD, Lydick E, Wasnich RD. Factors associated with joint pain among postmenopausal women. Int J Obes Relat Metab Disord. 1997;21:349-354.

visited on 6/14/2019

176. Williams RA, Foulsham BM. Weight reduction in osteoarthritis using phentermine. Practitioner. 1981;225:231-232.

177. Garfinkel L. Overweight and mortality. Cancer. 1986;58:1826-1829.

178. Williamson DF, Madans J, Pamuk E, Flegal KM, Kendrick JS, Serdula MK. A prospective study of childbearing and 10-year weight gain in US white women 25 to 45 years of age. Int J Obes Relat Metab Disord. 1994;18:561-569.

179. Cnattingius S, Bergstrom R, Lipworth L, Kramer MS. Prepregnancy weight and the risk of adverse pregnancy outcomes. N Eng J Med. 1998;338:147-152.

180. Shils ME, Olson JA, Shike M, eds. Modern Nutrition in Health and Disease. Philadelphia: Lea & Febiger; 1994.

181. Cogswell ME, Serdula MK, Hungerford DW, Yip R. Gestational weight gain among average-weight and overweight women—what is excessive? Am J Obstet Gynecol. 1995;172:705-712.

182. Institute of Medicine. Committee on Nutritional Status During Pregnancy and Lactation. Nutrition During Pregnancy: Part I, Weight Gain: Part II, Nutrient Supplements. Washington, DC: National Academy Press, 1990.

183. O'Neil PM, Jarrell MP. Psychological aspects of obesity and dieting. In: Wadden TA, Van Itallie TB, eds. Treatment Of The Seriously Obese Patient. New York: Guilford Press; 1992:252-270.

184. Jarvie GJ, Lahey BB, Graziano W, Framer E. Childhood obesity and social stigma: what we know and what we don't know. Dev Rev. 1983;3:237-273.

185. DeJong W, Kleck RE. The Social Psychological Effects of Overweight. In: Herman CP, Zanna MP, Higgins ET, eds. Physical Appearance, Stigma, and Social Behavior. Hillsdale, NJ: L. Erlbaum; 1986:65-88.

186. Goodman N, Dornbusch SM, Richardson SA, Hastof AH. Variant reactions to physical disabilities. Amer Sociolo Rev. 1963;28:429-435.

187. Lerner RM, Gellert E. Body build identification, preference, and aversion in children. Dev Psychol. 1969;5:456-462.

188. Richardson SA, Hastorf AH, Goodman N, Dornbusch SM. Cultural uniformity in reaction to physical disabilities. Am Soc Rev. 1961;26:241-247.

189. Staffieri JR. Body build and behavioral expectancies in young females. Dev Psychol. 1972;6:125-127.

190. Staffieri JR. A study of social stereotype of body image in children. J Pers Soc Psychol. 1967;7:101-104.

191. Lerner RM, Korn SJ. The development of body build stereotypes in males. Child Dev. 1972;43:908-920.

192. Blumberg P, Mellis LP. Medical students' attitudes toward the obese and the morbidly obese. Int J Eat Disord. 1985;4:169-175.

193. Maddox GL, Liederman V. Overweight as a social disability with medical implications. J Med Educ. 1969;44:214-220.

194. Rand CS, Macgregor AM. Morbidly obese patients' perceptions of social discrimination before and after surgery for obesity. South Med J. 1990;83:1390-1395.

195. Crandall CS, Biernat M. The ideology of anti-fat attitudes. J Appl Soc Psychol. 1990;20:227-243.

196. Maddox GL, Back KW, Liederman WR.

visited on 6/14/2019

Overweight as social deviance and disability. J Health Soc Behav. 1968;9:287-298.

197. Larkin JC, Pines HA. No fat persons need apply: experimental studies of the overweight stereotype and hiring preference. Sociology of Work and Occupations. 1979;6:312-327.

198. Pingitore R, Dugoni BL, Tindale RS, Spring B. Bias against overweight job applicants in a simulated employment interview. J Appl Psychol. 1994;79:909-917.

199. Roe DA, Eickwort KR. Relationships between obesity and associated health factors with unemployment among low income women. J Am Med Womens Assoc. 1976;31:193-194, 198-199, 203-204.

200. Canning H, Mayer J. Obesity: its possible effect on college acceptance. N Eng J Med. 1966;275:1172-1174.

201. Crandall CS. Do heavy-weight students have more difficulty paying for college? Personality Soc Psychol Bull. 1991;17:606-611.

202. Sargent JD, Blanchflower DG. Obesity and stature in adolescence and earnings in young adulthood. Analysis of a British birth cohort. Arch Pediatr Adolesc Med. 1994;148:681- 687.

203. Karris L. Prejudice against obese renters. J Soc Psych. 1977;101:159-160.

204. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med. 1993;329:1008-1012.

205. Massara EB, Stunkard AJ. A method of quantifying cultural ideals of beauty and the obese. Int J Obes. 1979;3:149-152.

206. Crandall CS, Martinez R. Culture, ideology, and antifat attitudes. Personality Soc Psychol Bull. 1996;22:1165-1176.

207. Striegel-Moore R, Rodin J. The influences of psychological variables in obesity. In: Brownell KD, Foreyt JP, eds. Handbook of Eating Disorders: Physiology, Psychology, and Treatment of Obesity, Anorexia, and Bulimia. New York: Basic Books; 1986:99-121.

208. Wadden TA, Stunkard AJ. Psychopathology and obesity. Ann N Y Acad Sci. 1987;499:55-65.

209. Lapidus L, Bengtsson C, Hallstrom T, Bjorntorp P. Obesity, adipose tissue distribution and health in women—results from a population study in Gothenburg, Sweden. Appetite. 1989;12:25-35.

210. Lissau I, Sorensen TI. Parental neglect during childhood and increased risk of obesity in young adulthood. Lancet. 1994;343:324-327.

211. Sullivan M, Karlsson J, Sjostrom L, et al. Swedish obese subjects (SOS)—an intervention study of obesity. Baseline evaluation of health and psychosocial functioning in the first 1743 subjects examined. Int J Obes Relat Metab Disord. 1993;17:503-512.

212. Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. Psychol Bull. 1995;117:3-20.

213. Stunkard AJ, Rush J. Dieting and depression reexamined: a critical review of reports of untoward responses during weight reduction for obesity. Annals Intern Med. 1974;81:526- 533.

214. Prather RC, Williamson DA. Psychopathology associated with bulimia,

visited on 6/14/2019

binge eating, and obesity. Int J Eat Disord. 1988;7:177-184.

215. Fitzgibbon ML, Stolley MR, Kirschenbaum DS. Obese people who seek treatment have different characteristics than those who do not seek treatment. Health Psychol. 1993;12:342-345.

216. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. Addict Behav. 1982;7:47-55.

217. Loro AD Jr, Orleans CS. Binge eating in obesity: preliminary findings and guidelines for behavioral analysis and treatment. Addict Behav. 1981;6:155-166.

218. Marcus MD, Wing RR, Fairburn CG. Cognitive treatment of binge eating versus behavioral weight control in the treatment of binge eating disorder. Annals of Behavioral Medicine. 1995;17:S090.

219. Spitzer RL, Devlin M, Walsh BT, et al. Binge eating disorder: a multisite field trial of the diagnostic criteria. Int J Eat Disord. 1992;11:191-203.

220. Yanovski SZ, Gormally JF, Leser MS, Gwirtsman HE, Yanovski JA. Binge eating disorder affects outcome of comprehensive very-low-calorie diet treatment. Obes Res. 1994;2:205-212.

221. Spitzer RL, Yanovski S, Wadden T, et al. Binge eating disorder: its further validation in a multisite study. Int J Eat Disord. 1993;13:137-153.

222. Wilfley DE, Agras WS, Telch CF, et al. Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: a controlled comparison. J Consult Clin Psychol. 1993;61:296-305.

223. Wilfley DE, Cohen LR. Psychological treatment of bulimia nervosa and binge eating disorder. Psychopharmacol Bull. 1997;33:437-453.

224. Yanovski SZ. Binge eating disorder: Current knowledge and future directions. Obes Res. 1993;1:306-324.

225. Yanovski SZ, Nelson JE, Dubbert BK, Spitzer RL. Association of binge eating disorder and psychiatric comorbidity in obese subjects. Am J Psychiatry. 1993;150:1472-1479.

226. Wilfley DE, Pike KM, Striegel-Moore R. Toward an integrated model of risk for binge eating disorder. J Gender Cult Health. 1997;2:1-32.

227. Fairburn CG, Wilson GT. Binge Eating: Nature, Assessment, and Treatment. New York: Guilford Press; 1993.

228. Castonguay LG, Eldredge KL, Agras WS. Binge eating disorder: current state and future directions. Clini Psychol Rev. 1995;15:865-890.

229. Striegel-Moore RH, Wilson GT, Wilfley DE, Elder KA, Brownell KD. Binge eating in an obese community sample. Int J Eat Disord. 1998;23:27-37.

230. Telch CF, Agras WS, Rossiter EM. Binge eating increases with increasing adiposity. Int J Eat Disord. 1988;7:115-119.

231. Kenardy J, Arnow B, Agras WS. The aversiveness of specific emotional states associated with binge-eating in obese subjects. Aust N Z J Psychiatry. 1996;30:839-844.

232. Marcus MD, Wing RR, Ewing L, Kern E, Gooding W, McDermott M. Psychiatric disorders among obese binge eaters. Int J Eat Disord. 1990;9:69-77.

233. Marcus MD, Wing RR, Hopkins J. Obese binge eaters: affect, cognitions, and

visited on 6/14/2019

response to behavioral weight control. J Consult Clin Psychol. 1988;56:433-439.

234. Molinari E, Ragazzoni P, Morosin A. Psychopathology in obese subjects with and without binge-eating disorder and in bulimic subjects. Psychol Rep. 1997;80:1327-1335.

235. Mussell MP, Mitchell JE, de Zwaan M, Crosby RD, Seim HC, Crow SJ. Clinical characteristics associated with binge eating in obese females: a descriptive study. Int J Obes Relat Metab Disord. 1996;20:324-331.

236. Specker S, de Zwaan M, Raymond N, Mitchell J. Psychopathology in subgroups of obese women with and without binge eating disorder. Compr Psychiatry. 1994;35:185-190.

237. Brody ML, Walsh BT, Devlin MJ. Binge eating disorder: reliability and validity of a new diagnostic category. J Consult Clin Psychol. 1994;62:381-386.

238. de Zwaan M, Nutzinger DO, Schoenbeck G. Binge eating in overweight women. Compr Psychiatry. 1992;33:256-261.

239. Kuehnel RH, Wadden TA. Binge eating disorder, weight cycling, and psychopathology. Int J Eat Disord. 1994;15:321-329.

240. Keefe PH, Wyshogrod D, Weinberger E, Agras WS. Binge eating and outcome of behavioral treatment of obesity: a preliminary report. Behav Res Ther. 1984;22:319-321.

241. Garner DM, Wooley SC. Confronting the failure of behavioral and dietary treatments for obesity. Clinl Psychol Rev. 1991;11:729-780.

242. Telch CF, Agras WS. The effects of a very low-calorie diet on binge eating. Behav Therapy. 1993;24:177-194.

243. Yanovski SZ, Sebring NG. Recorded food intake of obese women with binge eating disorder before and after weight loss. Int J Eat Disord. 1994;15:135-150.

244. Cash TF, Hicks KL. Being fat versus thinking fat: relationships with body image, eating behaviors, and well-being. Cogni Ther Res. 1990;14:327-341.

245. Collins JK. Methodology for the objective measurement of body image. Int J Eat Disord. 1987;6:393-399.

246. Collins JK, Beumont PJ, Touyz SW, Krass J, et al. Variability in body shape, perception in anorexic, bulimic, obese, and control subjects. Int J Eat Disord. 1987;6:633-638.

247. Gardner RM, Martinez R, Sandoval Y. Obesity and body image: an evaluation of sensory and non-sensory components. Psychol Med. 1987;17:927-932.

248. Glucksman ML, Hirsch J. The response of obese patients to weight reduction. 3. The perception of body size. Psychosom Med. 1969;31:1-7.

249. Garner DM, Garfinkel PE, Stancer HC, Moldofsky H. Body image disturbances in anorexia nervosa and obesity. Psychosom Med. 1976;38:327-336.

250. Grilo CM, Wilfley DE, Brownell KD, Rodin J. Teasing, body image, and self-esteem in a clinical sample of obese women. Addict Behav. 1994;19:443-450.

251. Mussell MP, Peterson CB, Weller CL, Crosby RD, de Zwaan M, Mitchell JE. Differences in body image and depression among obese women with and without binge eating disorder. Obes Res. 1996;4:431-439.

252. Stunkard A, Mendelson M. Obesity and the body image. I. Characteristics of distur-

visited on 6/14/2019

bances in the body image of some obese persons. Am J Psychiatry. 1967;123:1296-1300.

253. Stunkard A, Burt V. Obesity and the body image. II. Age at onset of disturbances in the body image. Am J Psychiatry. 1967;123:1443-1447.

254. Cash TF. The psychology of physical appearance: aesthetics, attributes, and images. In: Cash TF, Pruzinsky T, eds. Body Images: Development, Deviance, and Change. New York: Guilford Press, 1990:51-79.

255. Tiggemann M, Rothblum ED. Gender differences in social consequences of perceived overweight in the United States and Australia. Sex Roles. 1988;18:75-86.

256. Rosen JC. Improving body image in obesity. In: Thompson JK, ed. Body Image, Eating Disorders, and Obesity. Washington, DC: American Psychological Association; 1996:425-440.

257. Kumanyika SK, Morssink CB. Cultural appropriateness of weight management programs. In: Dalton S, ed. Overweight and Weight Management: The Health Professional's Guide To Understanding and Practice. Gaithersburg, MD: Aspen Publishers; 1997:69-103.

258. Childress AC, Brewerton TD, Hodges EL, Jarrell MP. The Kids' Eating Disorders Survey (KEDS): a study of middle school students. J Am Acad Child Adolesc Psychiatry. 1993;32:843-850.

259. Kumanyika S, Wilson JF, Guilford-Davenport M. Weight-related attitudes and behaviors of black women. J Am Diet Assoc. 1993;93:416-422.

260. Powell AD, Kahn AS. Racial differences in women's desires to be thin. Int J Eat Disord. 1995;17:191-195.

261. Striegel-Moore RH, Schreiber GB, Pike KM, Wilfley DE, Rodin J. Drive for thinness in black and white preadolescent girls. Int J Eat Disord. 1995;18:59-69.

262. Desmond SM, Price JH, Hallinan C, Smith D. Black and white adolescents' perceptions of their weight. J Sch Health. 1989;59:353-358.

263. Kemper KA, Sargent RG, Drane JW, Valois RF, Hussey JW. Black and white females' preceptions of ideal body size and social norms. Obes Res. 1994;2:117-126.

264. Rand CS, Kuldau JM. Morbid obesity: a comparison between a general population and obesity surgery patients. Int J Obes Relat Metab Disord. 1993;17:657-661.

265. Stevens J, Kumanyika SK, Keil JE. Attitudes toward body size and dieting: differences between elderly black and white women. Am J Public Health. 1994;84:1322-1325.

266. Wilfley DE, Schreiber GB, Pike KM, Striegel-Moore RH, Wright DJ, Rodin J. Eating disturbance and body image: a comparison of a community sample of adult black and white women. Int J Eat Disord. 1996;20:377-387.

267. Rucker CE, Cash TF. Body images, body-size perceptions, and eating behaviors among African-American and white college women. Int J Eating Disord. 1992;12:291-300.

268. Schreiber GG, Robins M, Striegel-Moore R, Obarzanek E, Morrison JA, Wright DJ. Weight modification efforts reported by black and white preadolescent girls: National Heart, Lung, and Blood Institute Growth and Health Study. Pediatrics. 1996;98:63-70.

269. Rosen JC, Gross J. Prevalence of weight reducing and weight gaining in adolescent

visited on 6/14/2019

girls and boys. Health Psychol. 1987;6:131-147.

270. Gittelsohn J, Harris SB, Thorne-Lyman AL, Hanley AJ, Barnie A, Zinman B. Body image concepts differ by age and sex in an Ojibway-Cree community in Canada. J Nutr. 1996;126:2990-3000.

271. Cooper RS, Ford E. Comparability of risk factors for coronary heart disease among blacks and whites in the NHANES-I Epidemiologic Follow-up Study. Ann Epidemiol. 1992;2:637-645.

272. Folsom AR, Burke GL, Byers CL, et al. Implications of obesity for cardiovascular disease in blacks: the CARDIA and ARIC studies. Am J Clin Nutr. 1991;53:1604S-1611S.

273. Fujimoto WY, Newell-Morris LL, Grote M, Bergstrom RW, Shuman WP. Visceral fat obesity and morbidity: NIDDM and atherogenic risk in Japanese-American men and women. Int J Obes. 1991;15 (Suppl2): 41-44.

274. Galanis DJ, McGarvey ST, Sobal J, Bausserman L, Levinson PD. Relations of body fat and fat distribution to the serum lipid, apolipoprotein and insulin concentrations of Samoan men and women. Int J Obes Relat Metab Disord. 1995;19:731-738.

275. Haffner SM, Diehl AK, Stern MP, Hazuda HP. Central adiposity and gallbladder disease in Mexican Americans. Am J Epidemiol. 1989;129:587-595.

276. Havas S, Fujimoto W, Close N, McCarter R, Keller J, Sherwin R. The NHLBI workshop on Hypertension in Hispanic Americans, Native Americans, and Asian/Pacific Islander Americans. Public Health Rep. 1996;111:451-458.

277. Howard BV, Lee ET, Cowan LD, et al. Coronary heart disease prevalence and its relation to risk factors in American Indians. The Strong Heart Study. Am J Epidemiol. 1995;142:254-268.

278. Lipton RB, Liao Y, Cao G, Cooper RS, McGee D. Determinants of incident non-insulin-dependent diabetes mellitus among blacks and whites in a national sample. The NHANES I Epidemiologic Follow-up Study. Am J Epidemiol. 1993;138:826-839.

279. Federation of American Societies for Experimental Biology. Life Sciences Research. Third Report on Nutrition Monitoring in the United States. Washington, DC: Interagency Board for Nutrition Monitoring and Related Research; 1995.

280. Ettinger WH, Wahl PW, Kuller LH, et al. Lipoprotein lipids in older people. Results from the Cardiovascular Health Study. The CHS Collaborative Research Group. Circulation. 1992;86:858-869.

281. Neser WB, Thomas J, Semenya K, Thomas DJ, Gillum RF. Obesity and hypertension in a longitudinal study of black physicians: the Meharry Cohort Study. J Chronic Dis. 1986;39:105-113.

282. Schmidt-Nowara WW, Coultas DB, Wiggins C, Skipper BE, Samet JM. Snoring in a Hispanic-American population. Risk factors and association with hypertension and other morbidity. Arch Intern Med. 1990;150:597-601.

283. Warne DK, Charles MA, Hanson RL, et al. Comparison of body size measurements as predictors of NIDDM in Pima Indians. Diabetes Care. 1995;18:435-439.

284. Wei M, Gaskill SP, Haffner SM, Stern MP. Waist circumference as the best predictor

visited on 6/14/2019

of noninsulin dependent diabetes mellitus (NIDDM) compared to body mass index, waist/hip ratio, and other anthropometric measurements in Mexican Americans—a 7-year prospective study. Obes Res. 1997;5:16-23.

285. Lee IM, Manson JE, Hennekens CH, Paffenbarger RS Jr. Body weight and mortality. A 27-year follow-up of middle-aged men. JAMA. 1993;270:2823-2828.

286. Allison DB, Faith MS, Heo M, Kotler DP. Hypothesis concerning the U-shaped relation between body mass index and mortality. Am J Epidemiol. 1997;146:339-349.

287. Stevens J, Cai J, Pamuk ER, Williamson DF, Thun MJ, Wood JL. The effect of age on the association between body-mass index and mortality. N Engl J Med. 1998;338:1-7.

288. Diehr P, Bild DE, Harris TB, Duxbury A, Siscovick D, Rossi M. Body mass index and mortality in nonsmoking older adults: The cardiovascular health study. Am J Public Health. 1998;88:623-629.

289. Cornoni-Huntley JC, Harris TB, Everett DF, et al. An overview of body weight of older persons, including the impact on mortality. The National Health and Nutrition Examination Survey I—Epidemiologic Follow-up Study. J Clin Epidemiol. 1991;44:743-753.

290. Harris T, Cook EF, Garrison R, Higgins M, Kannel W, Goldman L. Body mass index and mortality among nonsmoking older persons. The Framingham Heart Study. JAMA. 1988;259:1520-1524.

291. Baumgartner RN, Heymsfield SB, Roche AF. Human body composition and the epidemiology of chronic disease. Obes Res. 1995;3:73-95.

292. Losonczy KG, Harris TB, Cornoni-Huntley J, et al. Does weight loss from middle age to old age explain the inverse weight mortality relation in old age? Am J Epidemiol. 1995;141:312-321.

293. Fried LP, Kronmal RA, Newman AB, et al. Risk factors for 5-year mortality in older adults (for the Cardiovascular Health Study Collaborative Research Group). JAMA. 1998;279:585-592.

294. Folsom AR, Kaye SA, Sellers TA, et al. Body fat distribution and 5-year risk of death in older women. JAMA. 1993;269:483-487.

295. Willett WC, Stampfer M, Manson J, VanItallie T. New weight guidelines for Americans: justified or injudicious? Am J Clin Nutr. 1991;53:1102-1103.

296. Roche AF. Sarcopenia: a critical review of its measurements and health-related significance in the middle-aged and elderly. Am J Hum Biol. 1994;6:33-42.

297. Durazo-Arvizu RA, McGee DL, Cooper RS, Liao Y, Luke A. Mortality and optimal body mass index in a sample of the U.S. population. Am J Epidemiol. 1998;147:739-749.

298. Sorkin JD, Zonderman AB, Costa PT Jr., Andres R. Twenty year follow-up of the NHANES I cohort: tests of methodologic hypotheses. Obes Res. 1996;4 (Suppl 1): M46.

299. Stern MP, Patterson JK, Mitchell BD, Haffner SM, Hazuda HP. Overweight and mortality in Mexican Americans. Int J Obes. 1990;14:623-629.

300. Johnson JL, Heineman EF, Heiss G, Hames CG, Tyroler HA. Cardiovascular disease risk factors and mortality among black women and white women aged 40-

visited on 6/14/2019

64 years in Evans County, Georgia. Am J Epidemiol. 1986;123:209-220.

301. Stevens J, Keil JE, Rust PF, Tyroler HA, Davis CE, Gazes PC. Body mass index and body girths as predictors of mortality in black and white women. Arch Intern Med. 1992;152:1257-1262.

302. Tyroler HA, Knowles MG, Wing SB, et al. Ischemic heart disease risk factors and 20-year mortality in middle-age Evans County black males. Am Heart J. 1984;108:738-746.

303. Wienpahl J, Ragland DR, Sidney S. Body mass index and 15-year mortality in a cohort of black men and women. J Clin Epidemiol. 1990;43:949-960.

304. Durazo-Arvizu R, Cooper RS, Luke A, Prewitt TE, Liao Y, McGee DL. Relative weight and mortality in U.S. blacks and whites: findings from representative national population samples. Ann Epidemiol. 1997;7:383-395.

305. Hodge AM, Dowse GK, Collins VR, Zimmet PZ. Mortality in Micronesian Nauruans and Melanesian and Indian Fijians is not associated with obesity. Am J Epidemiol. 1996;143:442-455.

306. Hanson RL, McCance DR, Jacobsson LT, et al. The U-shaped association between body mass index and mortality: relationship with weight gain in a Native-American population. J Clin Epidemiol. 1995;48:903-916.

307. Pettitt DJ, Lisse JR, Knowler WC, Bennett PH. Mortality as a function of obesity and diabetes mellitus. Am J Epidemiol. 1982;115:359-366.

308. Andres R, Muller DC, Sorkin JD. Long-term effects of change in body weight on all-cause mortality. A review. Ann Intern Med. 1993;119:737-743.

309. Weight cycling. National Task Force on the Prevention and Treatment of Obesity. JAMA. 1994;272:1196-1202.

310. Williamson DF, Pamuk ER. The association between weight loss and increased longevity. A review of the evidence. Ann Intern Med. 1993;119:731-736.

311. Williamson DF. "Weight cycling" and mortality: how do the epidemiologists explain the role of intentional weight loss? J Am Coll Nutr. 1996;15:6-13.

312. Williamson DF. Intentional weight loss: patterns in the general population and its association with morbidity and mortality. Int J Obes Relat Metab Disord. 1997;21 (Suppl 1):S14-S19.

313. French SA, Jeffery RW, Folsom AR, Williamson DF, Byers T. Weight variability in a population-based sample of older women: reliability and intercorrelation of measures. Int J Obes Relat Metab Disord. 1995;19:22-29.

314. French SA, Jeffery RW, Folsom AR, Williamson DF, Byers T. History of intentional and unintentional weight loss in a population-based sample of women aged 55 to 69 years. Obes Res. 1995;3:163-170.

315. French SA, Jeffery RW, Folsom AR, Williamson DF, Byers T. Relation of weight variability and intentionality of weight loss to disease history and health-related variables in a population-based sample of women aged 55-69 years. Am J Epidemiol. 1995;142:1306-1314.

316. Meltzer AA, Everhart JE. Unintentional weight loss in the United States. Am J Epidemiol. 1995;142:1039-1046.

317. Singh RB, Rastogi SS, Verma R, et al. Randomised controlled trial of cardioprotective diet in patients with recent acute

visited on 6/14/2019

myocardial infarction: results of 1-year follow up. BMJ. 1992;304:1015-1019.

318. Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40-64 years. Am J Epidemiol. 1995;141:1128-1141.

319. Williamson DF, Pamuk E, Thun M, et al. Prospective study of international weight loss and mortality in overweight men aged 40-64 years. Obes Res. 1997;5:94S. [abstract].

320. Sjostrom L, Larsson B, Backman L, et al. Swedish obese subjects (SOS). Recruitment for an intervention study and a selected description of the obese state. Int J Obes Relat Metab Disord. 1992;16:465-479.

321. Sjostrom CD, Lissner L, Sjostrom L. Relationships between changes in body composition and changes in cardiovascular risk factors: the SOS intervention study. Obes Res. 1997;5:519-530.

322. Sjostrom L. Natural history of massive obesity. Obes Res. 1995;3:317S. [abstract].

323. Sobal J, Stunkard AJ. Socioeconomic status and obesity: A review of the literature. Psychol Bull. 1989;105:260-275.

324. Kumanyika SK. Obesity in minority populations: An epidemiologic assessment. Obes Res. 1994;2:166-182.

325. Ravussin E, Valencia ME, Esparza J, Bennett PH, Schultz LO. Effects of a traditional lifestyle on obesity in Pima Indians. Diabetes Care. 1994;17:1067-1074.

326. Kawate R, Yamakido M, Nishimoto Y. Migrant studies among the Japanese in Hiroshima and Hawaii. Proc 10th Congr Int Diabetes Federation. 1980:526-531.

327. Kawate R, Yamakido M, Nishimoto Y. Diabetes mellitus and its vascular complications in Japanese migrants on the Island of Hawaii. Diabetes Care. 1979;29:161-170.

328. Taylor R, Bennett P, Uili R, Joffres N, Levey S, Zimmet P. Diabetes in Wallis Polynesians: a comparison of residents of Wallis Island and first generation migrants of Noumea. Diabetes Res Clin Pract. 1985;1:169-178.

329. Bouchard C, Perusse L, Leblanc C, Tremblay A, Theriault G. Inheritance of the amount and distribution of human body fat. Int J Obes. 1988;12:205-215.

330. Tambs K, Moum T, Eaves L, et al. Genetics and environmental contributions to the variance of the body mass index in a Norwegian sample of first-degree and second-degree relatives. Am J Human Biology. 1991;3:257-268.

331. Vogler GP, Sorensen TI, Stunkard AJ, Srinivasan MR, Rao DC. Influences of genes and shared family environment on adult body mass index assessed in an adoption study by a comprehensive path model. Int J Obes Relat Metab Disord. 1995;19:40-45.

332. Stunkard AJ, Harris JR, Pedersen NL, McClearn GE. The body-mass index of twins who have been reared apart. N Engl J Med. 1990;322:1483-1487.

333. Allison DB, Kaprio J, Korkeila M, Koskenvuo M, Neale MC, Hayakawa K. The heritability of body mass index among an international sample of monozygotic twins reared apart. Int J Obes Relat Metab Disord. 1996;20:501-506.

334. Maes HHM, Neale MC, Eaves LJ. Genetic and environmental factors in relative body weight and human adiposity. Behav Gene. 1997;27:325-351.

visited on 6/14/2019

335. Allison DB. Methodological issues in obesity research: examples from new directions in assessment and management. In: VanItallie TB, Simopoulos AP, eds. Obesity. Philadelphia: Charles Press; 1995:129-132.

336. Allison DB, Pi-Sunyer FX. Obesity Treatment: Establishing Goals, Improving Outcomes, and Reviewing the Research Agenda. New York: Plenum Press; 1995.

337. Allison DB, Faith MS, Nathan JS. Risch's lambda values for human obesity. Int J Obes. 1996;20:990-999.

338. Lee JH, Reed DR, Price RA. Familial risk ratios for extreme obesity: implications for mapping human obesity genes. Int J Obes Relat Metab Disord. 1997;21:935-940.

339. Perusse L, Chagnon YC, Dionne FT, Bouchard C. The human obesity gene map: the 1996 update. Obes Res. 1997;5:49-61.

340. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. Nature. 1994;372:425-432.

341. Tartaglia LA, Dembski M, Weng X, et al. Identification and expression cloning of leptin receptor, OB-R. Cell. 1995;83:1263-1271.

342. Montague CT, Farooqi IS, Whitehead JP, et al. Congenital leptin deficiency is associated with severe early-onset obesity in humans. Nature. 1997;387:903-908.

343. Bouchard C, Perusse L, Rice T, Rao DC. The genetics of human obesity. In: Bray GA, Bouchard C, James WPT, eds. Handbook of Obesity. New York: M. Dekker; 1998: Chapter 10.

344. Bouchard C, Tremblay A. Genetic effects in human energy expenditure components. Int J Obes. 1990;14 (Suppl 1):49-55.

345. Bouchard C, Tremblay A, Despres JP, et al. The response to long-term overfeeding in identical twins. N Engl J Med. 1990;322:1477-1482.

346. Anderssen S, Holme I, Urdal P, Hjermann I. Diet and exercise intervention have favourable effects on blood pressure in mild hypertensives: the Oslo Diet and Exercise Study (ODES). Blood Press. 1995;4:343-349.

347. Croft PR, Brigg D, Smith S, Harrison CB, Branthwaite A, Collins MF. How useful is weight reduction in the management of hypertension? J R Coll Gen Pract. 1986;36:445-448.

348. Davis BR, Blaufox MD, Oberman A, et al. Reduction in long-term antihypertensive medication requirements. Effects of weight reduction by dietary intervention in overweight persons with mild hypertension. Arch Intern Med. 1993;153:1773-1782.

349. Fagerberg B, Andersson OK, Isaksson B, Bjorntorp P. Blood pressure control during weight reduction in obese hypertensive men: separate effects of sodium and energy restriction. BMJ. 1984;288:11-14.

350. Grimm RH Jr, Cohen JD, Smith WM, Falvo-Gerard L, Neaton JD. Hypertension management in the Multiple Risk Factor Intervention Trial (MRFIT). 6-year intervention results for men in special intervention and usual care groups. Arch Intern Med. 1985;145:1191-1199.

351. Haynes RB, Harper AC, Costley SR, et al. Failure of weight reduction to reduce mildly elevated blood pressure: a randomized trial. J Hypertens. 1984;2:535-539.

352. Heyden S. The workingman's diet. II.

visited on 6/14/2019

Effect of weight reduction in obese patients with hypertension, diabetes, hyperuricemia and hyperlipidemia. Nutr Metab. 1978;22:141-159.

353. Kumanyika SK, Bahnson J, Bottom J, et al. Race and sex influences on the efficacy of non-pharmologic step-down therapy in hypertensive older adults. Can J Cardiol. 1997;13 (Suppl B):369B [abstract].

354. Langford HG, Blaufox MD, Oberman A, et al. Dietary therapy slows the return of hypertension after stopping prolonged medication. JAMA. 1985;253:657-664.

355. Langford HG, Davis BR, Blaufox D, et al. Effect of drug and diet treatment of mild hypertension on diastolic blood pressure. The TAIM Research Group. Hypertension. 1991;17:210-217.

356. MacMahon SW, Macdonald GJ, Bernstein L, Andrews G, Blacket RB. A randomized controlled trial of weight reduction and metoprolol in the treatment of hypertension in young overweight patients. Clin Exp Pharmacol Physiol. 1985;12:267-271.

357. Ramsay LE, Ramsay MH, Hettiarachchi J, Davies DL, Winchester J. Weight reduction in a blood pressure clinic. BMJ. 1978;2:244-245.

358. Reisin E, Abel R, Modan M, Silverberg DS, Eliahou HE, Modan B. Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. N Engl J Med. 1978;298:1-6.

359. Stamler R, Stamler J, Grimm R, et al. Nutritional therapy for high blood pressure. Final report of a 4-year randomized controlled trial—the Hypertension Control Program. JAMA. 1987;257:1484-1491.

360. Wassertheil-Smoller S, Langford HG, Blaufox MD, et al. Effective dietary intervention in hypertensives: sodium restriction and weight reduction. J Am Diet Assoc. 1985;85:423- 430.

361. Whelton PK, Applegate WB, Ettiger WH, et al. Efficacy of weight loss and reduced sodium intake in the Trial of Nonpharmacologic Interventions in the Elderly (TONE). Circulation. 1996;94 (Suppl I):I-178 [abstract].

362. Agurs-Collins TD, Kumanyika SK, Have TR, Adams-Campbell LL. A randomized controlled trial of weight reduction and exercise for diabetes management in older African-American subjects . Diabetes Care. 1997;20:1503-1511.

363. Fortmann SP, Haskell WL, Wood PD. Effects of weight loss on clinic and ambulatory blood pressure in normotensive men. Am J Cardiol. 1988;62:89-93.

364. Hakala P, Karvetti RL. Weight reduction on lactovegetarian and mixed diets. Changes in weight, nutrient intake, skinfold thicknesses and blood pressure. Eur J Clin Nutr. 1989;43:421-430.

365. Hellenius ML, de Faire U, Berglund B, Hamsten A, Krakau I. Diet and exercise are equally effective in reducing risk for cardiovascular disease. Results of a randomized controlled study in men with slightly to moderately raised cardiovascular risk factors. Atherosclerosis. 1993;103:81-91.

366. The Hypertension Prevention Trial: three-year effects of dietary changes on blood pressure. Hypertension Prevention Trial Research Group. Arch Intern Med. 1990;150:153-162.

367. Jeffery RW, Wing RR, Thorson C, et al. Strengthening behavioral interventions for weight loss: a randomized trial of food provision and monetary incentives. J Consult Clin Psychol. 1993;61:1038-1045.

visited on 6/14/2019

368. Karvetti RL, Hakala P. A 7-year follow-up of a weight reduction programme in Finnish primary health care. Eur J Clin Nutr. 1992;46:743-752.

369. Katzel LI, Bleecker ER, Colman EG, Rogus EM, Sorkin JD, Goldberg AP. Effects of weight loss vs aerobic exercise training on risk factors for coronary disease in healthy, obese, middle-aged and older men. A randomized controlled trial. JAMA. 1995;274:1915- 1921.

370. Puddey IB, Parker M, Beilin LJ, Vandongen R, Masarei JR. Effects of alcohol and caloric restrictions on blood pressure and serum lipids in overweight men. Hypertension. 1992;20:533-541.

371. Reid CM, Dart AM, Dewar EM, Jennings GL. Interactions between the effects of exercise and weight loss on risk factors, cardiovascular haemodynamics and left ventricular structure in overweight subjects. J Hypertens. 1994;12:291-301.

372. Rocchini AP, Katch V, Schork A, Kelch RP. Insulin and blood pressure during weight loss in obese adolescents. Hypertension. 1987;10:267-273.

373. Simkin-Silverman L, Wing RR, Hansen DH, et al. Prevention of cardiovascular risk factor elevations in healthy premenopausal women. Prev Med. 1995;24:509-517.

374. Stamler R, Stamler J, Gosch FC, et al. Primary prevention of hypertension by nutritional-hygienic means. Final report of a randomized, controlled trial. JAMA. 1989;262:1801-1807.

375. Stefanick ML, Mackey S, Sheehan M, Ellsworth N, Haskell WL, Wood PD. Effects of the NCEP Step 2 diet and exercise on lipoprotein in postmenopausal women and men with low HDL-cholesterol and high LDL-cholesterol. New Engl J Med 1998;339:12-20.

376. Stevens VJ, Corrigan SA, Obarzanek E, et al. Weight loss intervention in phase 1 of the Trials of Hypertension Prevention. The TOHP Collaborative Research Group. Arch Intern Med. 1993;153:849-858.

377. Svendsen OL, Hassager C, Christiansen C. Effect of an energy-restrictive diet, with or without exercise, on lean tissue mass, resting metabolic rate, cardiovascular risk factors, and bone in overweight postmenopausal women. Am J Med. 1993;95:131-140.

378. The effects of nonpharmacologic interventions on blood pressure of persons with high normal levels. Results of the Trials of Hypertension Prevention, Phase I. JAMA. 1992;267:1213-1220.

379. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The Trials of Hypertension Prevention, phase II. The Trials of Hypertension Prevention Collaborative Research Group. Arch Intern Med. 1997;157:657-667.

380. Wood PD, Stefanick ML, Williams PT, Haskell WL. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. N Engl J Med. 1991;325:461-466.

381. MacMahon S, Cutler J, Brittain E, Higgins M. Obesity and hypertension: epidemiological and clinical issues. Eur Heart J. 1987;8 (Suppl B):57-70.

382. Whelton PK, Appel LJ, Espeland MA, et al. Sodium reduction and weight loss in the treatment of hypertension in older persons: A randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). JAMA. 1998;279:839-846.

visited on 6/14/2019

383. Cutler JA. Randomized clinical trials of weight reduction in nonhypertensive persons. Ann Epidemiol. 1991;1:363-370.

384. Svendsen OL, Hassager C, Christiansen C. Six months' follow-up on exercise added to a short-term diet in overweight postmenopausal women—effects on body composition, resting metabolic rate, cardiovascular risk factors and bone. Int J Obes Relat Metab Disord. 1994;18:692-698.

385. Steven VJ. Weight loss and blood pressure changes over three years: results of the Trials of Hypertension Prevention (TOHP) Phase II. In: Cardiovascular Health: Coming Together for the 21st Century. A National Conference. 1998 Feb 19-21; San Francisco, CA: National Heart, Lung, and Blood Institute; 1998. Abstract R.6F.

386. Bremer JM, Scott RS, Lintott CJ. Dexfenfluramine reduces cardiovascular risk factors. Int J Obes Relat Metab Disord. 1994;18:199-205.

387. Ditschuneit HH, Flechtner-Mors M, Adler G. The effects of dexfenfluramine on weight loss and cardiovascular risk factors in female patients with upper and lower body obesity. J Cardiovasc Risk. 1996;3:397-403.

388. Bone HG, Reedy KR, Sherwin RS. FDA Orlistat Transcript. Endocrinologic and Metabolic Drugs Advisory Committee Meeting # 67. 1997.

389. Bone HG, Reedy KR, Colley CA. FDA Sibutramine Transcript. Endocrinologic and Metabolic Drugs Advisory Committee Meeting # 64. 1996.

390. Mathus-Vliegen EM, van de Voorde K, Kok AM, Res AM. Dexfenfluramine in the treatment of severe obesity: a placebo-controlled investigation of the effects on weight loss, cardiovascular risk factors, food intake and eating behaviour. J Intern Med. 1992;232:119- 127.

391. Mathus-Vliegen EM. Prolonged surveillance of dexfenfluramine in severe obesity. Neth J Med. 1993;43:246-253.

392. O'Connor HT, Richman RM, Steinbeck KS, Caterson ID. Dexfenfluramine treatment of obesity: a double blind trial with posttrial followup. Int J Obes Relat Metab Disord. 1995;19:181-189.

393. Pfohl M, Luft D, Blomberg I, Schmulling RM. Long-term changes of body weight and cardiovascular risk factors after weight reduction with group therapy and dexfenfluramine. Int J Obes Relat Metab Disord. 1994;18:391-395.

394. Swinburn BA, Carmichael HE, Wilson MR. Dexfenfluramine as an adjunct to a reduced-fat, ad libitum diet: effects on body composition, nutrient intake and cardiovascular risk factors. Int J Obes Relat Metab Disord. 1996;20:1033-1040.

395. Weintraub M, Sundaresan PR, Madan M, et al. Long-term weight control study. I (weeks 0 to 34). The enhancement of behavior modification, caloric restriction, and exercise by fenfluramine plus phentermine versus placebo. Clin Pharmacol Ther. 1992;51:586-594.

396. Arroll B, Beaglehole R. Exercise for hypertension. Lancet. 1993;341:1248-1249.

397. Fagard RH. Physical fitness and blood pressure. J Hyper. 1993;11:S47-S52.

398. Kelley G, Tran ZV. Aerobic exercise and normotensive adults: a meta-analysis. Med Sci Sports Exerc. 1995;27:1371-1377.

399. Dengel JL, Katzel LI, Goldberg AP. Effect of an American Heart Association diet, with or without weight loss, on lipids in obese middle-aged and older men. Am J Clin Nutr. 1995;62:715-721.

visited on 6/14/2019

400. Jalkanen L. The effect of a weight reduction program on cardiovascular risk factors among overweight hypertensives in primary health care. Scand J Soc Med. 1991;19:66-71.

401. King AC, Haskell WL, Taylor CB, Kraemer HC, DeBusk RF. Group- vs home-based exercise training in healthy older men and women. A community-based clinical trial. JAMA. 1991;266:1535-1542.

402. Marniemi J, Seppanen A, Hakala P. Long-term effects on lipid metabolism of weight reduction on lactovegetarian and mixed diet. Int J Obes. 1990;14:113-125.

403. Nilsson PM, Lindholm LH, Schersten BF. Lifestyle changes improve insulin resistance in hyperinsulinaemic subjects: a 1-year intervention study of hypertensives and normotensives in Dalby. J Hypertens. 1992;10:1071-1078.

404. Ronnemaa T, Marniemi J, Puukka P, Kuusi T. Effects of long-term physical exercise on serum lipids, lipoproteins and lipid metabolizing enzymes in type 2 (non-insulin-dependent) diabetic patients. Diabetes Res. 1988;7:79-84.

405. Schuler G, Hambrecht R, Schlierf G, et al. Regular physical exercise and low-fat diet. Effects on progression of coronary artery disease. Circulation. 1992;86:1-11.

406. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. N Engl J Med. 1988;319:1173-1179.

407. Drent ML, van der Veen EA. Lipase inhibition: a novel concept in the treatment of obesity. Int J Obes Relat Metab Disord. 1993;17:241-244.

408. O'Kane M, Wiles PG, Wales JK. Fluoxetine in the treatment of obese type 2 diabetic patients. Diabet Med. 1994;11:105-110.

409. Emery EM, Schmid TL, Kahn HS, Filozof PP. A review of the association between abdominal fat distribution, health outcome measures, and modifiable risk factors. Am J Health Promot. 1993;7(5):342-353.

410. Bjorntorp P. Abdominal fat distribution and disease: an overview of epidemiological data. Ann Med. 1992;24:15-18.

411. Leon AS. Physical Activity and Cardiovascular Health: A National Consensus. IL: Human Kinetics; 1997.

412. Hjermann I, Leren P, Norman N, Helgeland A, Holme I. Serum insulin response to oral glucose load during a dietary intervention trial in healthy coronary high risk men: the Oslo study. Scand J Clin Lab Invest. 1980;40:89-94.

413. Heller SR, Clarke P, Daly H, et al. Group education for obese patients with type 2 diabetes: greater success at less cost. Diabet Med. 1988;5:552-556.

414. Manning RM, Jung RT, Leese GP, Newton RW. The comparison of four weight reduction strategies aimed at overweight diabetic patients. Diabet Med. 1995;12:409-415.

415. Colman E, Katzel LI, Rogus E, Coon P, Muller D, Goldberg AP. Weight loss reduces abdominal fat and improves insulin action in middle-aged and older men with impaired glucose tolerance. Metabolism. 1995;44:1502-1508.

416. Fujioka S, Matsuzawa Y, Tokunaga K, et al. Improvement of glucose and lipid metabolism associated with selective reduction of intra-abdominal visceral fat in premenopausal women with visceral fat obesity. Int J Obes. 1991;15:853-859.

visited on 6/14/2019

417. Marks SJ, Moore NR, Clark ML, Strauss BJ, Hockaday TD. Reduction of visceral adipose tissue and improvement of metabolic indices: effect of dexfenfluramine in NIDDM. Obes Res. 1996;4:1-7.

418. van der Merwe MT, Wing JR, Celgow LH, et al. Metabolic indices in relation to body composition changes during weight loss on dexfenfluramine in obese women from two South African ethnic groups. Int J Obes Relat Metab Disord. 1996;20:768-776.

419. Ronnemaa T, Mattila K, Lehtonen A, Kallio V. A controlled randomized study on the effect of long-term physical exercise on the metabolic control in type 2 diabetic patients. Acta Med Scand. 1986;220:219-224.

420. Han TS, van Leer EM, Seidell JC, Lean ME. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. BMJ. 1995;311:1401-1405.

421. Pouliot MC, Despres JP, Lemieux S, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol. 1994;73:460-468.

422. van der Kooy K, Leenen R, Seidell JC, Deurenberg P, Droop A, Bakker CJ. Waist-hip ratio is a poor predictor of changes in visceral fat. Am J Clin Nutr. 1993;57:327-333.

423. Bouchard C, Tremblay A, Despres JP, et al. The response to exercise with constant energy intake in identical twins. Obes Res. 1994;2:400-410.

424. Leenen R, van der Kooy K, Deurenberg P, et al. Visceral fat accumulation in obese subjects: relation to energy expenditure and response to weight loss. Am J Physiol. 1992;263:E913-E919.

425. Stallone DD, Stunkard AJ, Wadden TA, Foster GD, Boorstein J, Arger P. Weight loss and body fat distribution: a feasibility study using computed tomography. Int J Obes. 1991;15:775-780.

426. Ross R, Rissanen J, Pedwell H, Clifford J, Shragge P. Influence of diet and exercise on skeletal muscle and visceral adipose tissue in men. J Appl Physiol. 1996;81:2445-2455.

427. Ross R, Rissanen J. Mobilization of visceral and subcutaneous adipose tissue in response to energy restriction and exercise. Am J Clin Nutr. 1994;60:695-703.

428. van der Kooy K, Leenen R, Seidell JC, Deurenberg P, Hautvast JG. Effect of a weight cycle on visceral fat accumulation. Am J Clin Nutr. 1993;58:853-857.

429. Dowling HJ, Pi-Sunyer FX. Race-dependent health risks of upper body obesity. Diabetes. 1993;42:537-543.

430. Kissebah AH, Vydelingum N, Murray R, et al. Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metab. 1982;54:254-260.

431. Berglund A, Andersson OK, Berglund G, Fagerberg B. Antihypertensive effect of diet compared with drug treatment in obese men with mild hypertension. BMJ. 1989;299:480- 485.

432. Frey-Hewitt B, Vranizan KM, Dreon DM, Wood PD. The effect of weight loss by dieting or exercise on resting metabolic rate in overweight men. Int J Obes. 1990;14:327-334.

433. Oberman A, Wassertheil-Smoller S, Langford HG, et al. Pharmacologic and nutritional treatment of mild hypertension:

visited on 6/14/2019

changes in cardiovascular risk status. Ann Intern Med. 1990;112:89-95.

434. Hammer RL, Barrier CA, Roundy ES, Bradford JM, Fisher AG. Calorie-restricted low-fat diet and exercise in obese women. Am J Clin Nutr. 1989;49:77-85.

435. Marks BL, Ward A, Morris DH, Castellani J, Rippe JM. Fat-free mass is maintained in women following a moderate diet and exercise program. Med Sci Sports Exerc. 1995;27:1243-1251.

436. Wadden TA, Sternberg JA, Letizia KA, Stunkard AJ, Foster GD. Treatment of obesity by very low calorie diet, behavior therapy, and their combination: a five-year perspective. Int J Obes. 1989;13 (Suppl 2):39-46.

437. Wadden TA, Foster GD, Letizia KA. One-year behavioral treatment of obesity: comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. J Consult Clin Psychol. 1994;62:165-171.

438. Wadden TA, Foster GD, Letizia KA, Mullen JL. Long-term effects of dieting on resting metabolic rate in obese outpatients. JAMA. 1990;264:707-711.

439. Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN. Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. Diabetes Care. 1994;17:30-36.

440. de Bont AJ, Baker IA, St. Leger AS, et al. A randomised controlled trial of the effect of low-fat diet advice on dietary response in insulin-independent diabetic women. Diabetologia. 1981;21:529-533.

441. Jeffery RW, Hellerstedt WL, French SA, Baxter JE. A randomized trial of counseling for fat restriction versus calorie restriction in the treatment of obesity. Int J Obes Relat Metab Disord. 1995;19:132-137.

442. Schlundt DG, Hill JO, Pope-Cordle J, Arnold D, Virts KL, Katahn M. Randomized evaluation of a low fat ad libitum carbohydrate diet for weight reduction. Int J Obes Relat Metab Disord. 1993;17:623-629.

443. Shah M, McGovern P, French S, Baxter J. Comparison of a low-fat, ad libitum complex-carbohydrate diet with a low-energy diet in moderately obese women. Am J Clin Nutr. 1994;59:980-984.

444. Sheppard L, Kristal AR, Kushi LH. Weight loss in women participating in a randomized trial of low-fat diets. Am J Clin Nutr. 1991;54:821-828.

445. Bertram SR, Venter I, Stewart RI. Weight loss in obese women—exercise vs. dietary education. S Afr Med J. 1990;78:15-18.

446. Verity LS, Ismail AH. Effects of exercise on cardiovascular disease risk in women with NIDDM. Diabetes Res Clin Pract. 1989;6:27-35.

447. King AC, Haskell WL, Young DR, Oka RK, Stefanick ML. Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. Circulation. 1995;91:2596-2604.

448. Gordon NF, Scott CB, Levine BD. Comparison of single versus multiple lifestyle interventions: are the antihypertensive effects of exercise training and diet-induced weight loss additive? Am J Cardiol. 1997;79:763-767.

449. Garrow JS, Summerbell CD. Meta-analysis: effect of exercise, with or without dieting, on the body composition of overweight subjects. Eur J Clin Nutr. 1995;49:1-10.

visited on 6/14/2019

450. Miller WC, Lindeman AK, Wallace J, Niederpruem M. Diet composition, energy intake, and exercise in relation to body fat in men and women. Am J Clin Nutr. 1990;52:426-430.

451. Fitzgerald SJ, Kriska AM, Pereira MA, de Courten MP. Associations among physical activity, television watching, and obesity in adult Pima Indians. Med Sci Sports Exerc. 1997;29:910-915.

452. Klesges RC, Eck LH, Isbell TR, Fulliton W, Hanson CL. Physical activity, body composition, and blood pressure: a multi-method approach. Med Sci Sports Exerc. 1991;23:759-765.

453. Tremblay A, Despres JP, Leblanc C, et al. Effect of intensity of physical activity on body fatness and fat distribution. Am J Clin Nutr. 1990;51:153-157.

454. French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter JE. Predictors of weight change over two years among a population of working adults: the Healthy Worker Project. Int J Obes Relat Metab Disord. 1994;18:145-154.

455. Williams PT. Evidence for the incompatibility of age-neutral overweight and age-neutral physical activity standards from runners. Am J Clin Nutr. 1997;65:1391-1396.

456. Ewbank PP, Darga LL, Lucas CP. Physical activity as a predictor of weight maintenance in previously obese subjects. Obes Res. 1995;3:257-263.

457. Klesges RC, Klesges LM, Haddock CK, Eck LH. A longitudinal analysis of the impact of dietary intake and physical activity on weight change in adults. Am J Clin Nutr. 1992;55:818-822.

458. Ching PL, Willett WC, Rimm EB, Colditz GA, Gortmaker SL, Stampfer MJ. Activity level and risk of overweight in male health professionals. Am J Public Health. 1996;86:25-30.

459. Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. Int J Obes Relat Metab Disord. 1993;17:279-286.

460. Kawachi I, Troisi RJ, Rotnitzky AG, Coakley EH, Colditz GA. Can physical activity minimize weight gain in women after smoking cessation? Am J Public Health. 1996;86:999-1004.

461. Bild DE, Sholinsky P, Smith DE, Lewis CE, Hardin JM, Burke GL. Correlates and predictors of weight loss in young adults: the CARDIA study. Int J Obes Relat Metab Disord. 1996;20:47-55.

462. Kayman S, Bruvold W, Stern JS. Maintenance and relapse after weight loss in women: behavioral aspects. Am J Clin Nutr. 1990;52:800-807.

463. Grodstein F, Levine R, Troy L, Spencer T, Colditz GA, Stampfer MJ. Three-year follow-up of participants in a commercial weight loss program. Can you keep it off? Arch Intern Med. 1996;156:1302-1306.

464. Seidell JC. Environmental influences on regional fat distribution. Int J Obes. 1991;15:31-35.

465. Kaye SA, Folsom AR, Prineas RJ, Potter JD, Gapstur SM. The association of body fat distribution with lifestyle and reproductive factors in a population study of post-menopausal women. Int J Obes. 1990;14:583-591.

466. Slattery ML, McDonald A, Bild DE, et al. Associations of body fat and its distribu-

visited on 6/14/2019

tion with dietary intake, physical activity, alcohol, and smoking in blacks and whites. Am J Clin Nutr. 1992;55:943-949.

467. Troisi RJ, Heinold JW, Vokonas PS, Weiss ST. Cigarette smoking, dietary intake, and physical activity: effects on body fat distribution—the Normative Aging Study. Am J Clin Nutr. 1991;53:1104-1111.

468. Wing RR, Matthews KA, Kuller LH, Meilahn EN, Plantinga P. Waist-to-hip ratio in middle-aged women. Associations with behavioral and psychological factors and with changes in cardiovascular risk factors. Arterioscler Throm. 1991;11:1250-1257.

469. Andersen RE, Wadden TA, Bartlett SJ, Vogt RA, Weinstock RS. Relation of weight loss to changes in serum lipids and lipoproteins in obese women. Am J Clin Nutr. 1995;62:350-357.

470. Blonk MC, Jacobs MA, Biesheuvel EH, Weeda-Mannak WL, Heine RJ. Influences on weight loss in type 2 diabetic patients: little long-term benefit from group behaviour therapy and exercise training. Diabet Med. 1994;11:449-457.

471. Neumark-Sztainer D, Kaufmann NA, Berry EM. Physical activity within a community-based weight control program: program evaluation and predictors of success. Public Health Rev. 1995;23:237-251.

472. Sweeney ME, Hill JO, Heller PA, Baney R, DiGirolamo M. Severe vs moderate energy restriction with and without exercise in the treatment of obesity: efficiency of weight loss. Am J Clin Nutr. 1993;57:127-134.

473. Wing RR, Epstein LH, Paternostro-Bayles M, Kriska A, Nowalk MP, Gooding W. Exercise in a behavioural weight control programme for obese patients with Type 2 (non-insulin-dependent) diabetes. Diabetologia. 1988;31:902-909.

474. Kaplan RM, Hartwell SL, Wilson DK, Wallace JP. Effects of diet and exercise interventions on control and quality of life in non-insulin-dependent diabetes mellitus. J Gen Intern Med. 1987;2:220-228.

475. Leighton RF, Repka FJ, Birk TJ, et al. The Toledo Exercise and Diet Study. Results at 26 weeks. Arch Intern Med. 1990;150:1016-1020.

476. Long CG, Simpson CM, Allott EA. Psychological and dietetic counselling combined in the treatment of obesity: a comparative study in a hospital outpatient clinic. Hum Nutr Appl Nutr. 1983;37:94-102.

477. Wadden TA, Stunkard AJ. Controlled trial of very low calorie diet, behavior therapy, and their combination in the treatment of obesity. J Consult Clin Psychol. 1986;54:482-488.

478. Craighead LW, Stunkard AJ, O'Brien RM. Behavior therapy and pharmacotherapy for obesity. Arch Gen Psychiatry. 1981;38:763-768.

479. Bennett GA. An evaluation of self-instructional training in the treatment of obesity. Addict Behav. 1986;11:125-134.

480. Bennett GA. Cognitive rehearsal in the treatment of obesity: a comparison against cue avoidance and social pressure. Addict Behav. 1986;11:225-237.

481. Forster JL, Jeffery RW, Sullivan S, Snell MK. A work-site weight control program using financial incentives collected through payroll deduction. J Occup Med. 1985;27:804-808.

482. Glasgow RE, Toobert DJ, Hampson SE, Brown JE, Lewinsohn PM, Donnelly J. Improving self-care among older patients with type II diabetes: the Sixty Something... Study. Patient Educ Couns. 1992;19:61-74.

483. Jeffery RW, Hellerstedt WL, Schmid TL. Correspondence programs for smoking cessa-

visited on 6/14/2019

tion and weight control: a comparison of two strategies in the Minnesota Heart Health Program. Health Psychol. 1990;9:585-598.

484. Lovibond SH, Birrell PC, Langeluddecke P. Changing coronary heart disease risk-factor status: the effects of three behavioral programs. J Behav Med. 1986;9:415-437.

485. Muchmore DB, Springer J, Miller M. Self-monitoring of blood glucose in overweight type 2 diabetic patients. Acta Diabetol. 1994;31:215-219.

486. Perri MG, Nezu AM, Patti ET, McCann KL. Effect of length of treatment on weight loss. J Consult Clin Psychol. 1989;57:450-452.

487. Perri MG, McAllister DA, Gange JJ, Jordan RC, McAdoo G, Nezu AM. Effects of four maintenance programs on the long-term management of obesity. J Consult Clin Psychol. 1988;56:529-534.

488. Rosenthal B, Allen GJ, Winter C. Husband involvement in the behavioral treatment of overweight women: initial effects and long-term follow-up. Int J Obes. 1980;4:165-173.

489. Smith DE, Heckemeyer CM, Kratt PP, Mason DA. Motivational interviewing to improve adherence to a behavioral weight-control program for older obese women with NIDDM. A pilot study. Diabetes Care. 1997;20:52-54.

490. Uusitupa M, Laitinen J, Siitonen O, Vanninen E, Pyorala K. The maintenance of improved metabolic control after intensified diet therapy in recent type 2 diabetes. Diabetes Res Clin Pract. 1993;19:227-238.

491. Wadden TA, Berkowitz RI, Vogt RA, Steen SN, Stunkard AJ, Foster GD. Lifestyle modification in the pharmacologic treatment of obesity: a pilot investigation of a potential primary care approach. Obes Res. 1997;5:218-226.

492. Wing RR, Marcus MD, Epstein LH, Jawad A. A "family-based" approach to the treatment of obese Type II diabetic patients. J Consult Clin Psychol. 1991;59:156-162.

493. Wing RR, Epstein LH, Nowalk MP, Scott N, Koeske R, Hagg S. Does self-monitoring of blood glucose levels improve dietary compliance for obese patients with Type II diabetes? Am J Med. 1986;81:830-836.

494. Eldredge KL, Agras WS, Arnow B, et al. The effects of extending cognitive-behavioral therapy for binge eating disorder among initial treatment nonresponders. Int J Eat Disord. 1997;21:347-352.

495. Agras WS, Berkowitz RI, Arnow BA, et al. Maintenance following a very-low-calorie diet. J Consult Clin Psychol. 1996;64:610-613.

496. Sobal J, Devin C. Social aspects of obesity. In: Dalton S, ed. Overweight and Weight Management: The Health Professional's Guide To Understanding and Practice. Gaithersburg, MD: Aspen Publishers; 1997:312-331.

497. Brown PJ; Cultural perspectives on the etiology and treatment of obesity. In: Stunkard AJ, Wadden TA, eds. Obesity Theory and Therapy. New York: Raven Press; 1993:179-195.

498. Foster GD, Wadden TA, Vogt RA. Resting energy expenditure in obese African American and Caucasian women. Obes Res. 1997;5:1-8.

499. Yanovski SZ, Reynolds JC, Boyle AJ, Yanovski JA. Resting metabolic rate in African-American and Caucasian girls. Obes Res. 1997;5:321-325.

visited on 6/14/2019

500. Albu JB, Murphy L, Frager DH, Johnson JA, Pi-Sunyer FX. Visceral fat and race-dependent health risks in obese nondiabetic premenopausal women. Diabetes. 1997;46:456-462.

501. Allison DB, Edlen-Nezin L, Clay-Williams G. Obesity among African American women: prevalence, consequences, causes, and developing research. Women's Health. 1997;3:243-274.

502. Andersson B, Seidell J, Terning K, Bjorntorp P. Influence of menopause on dietary treatment of obesity. J Intern Med. 1990;227:173-181.

503. Kumanyika SK, Obarzanek E, Stevens VJ, Hebert PR, Whelton PK. Weight-loss experience of black and white participants in NHLBI- sponsored clinical trials. Am J Clin Nutr. 1991;53:1631S-1638S.

504. Wing RR, Anglin K. Effectiveness of a behavioral weight control program for blacks and whites with NIDDM. Diabetes Care. 1996;19:409-413.

505. Wylie-Rosett J, Wassertheil-Smoller S, Blaufox MD, et al. Trial of antihypertensive intervention and management: greater efficacy with weight reduction than with a sodium-potassium intervention. J Am Diet Assoc. 1993;93:408-415.

506. Andersen T, Astrup A, Quaade F. Dexfenfluramine as adjuvant to a low-calorie formula diet in the treatment of obesity: a randomized clinical trial. Int J Obes Relat Metab Disord. 1992;16:35-40.

507. Guy-Grand B, Apfelbaum M, Crepaldi G, Gries A, Lefebvre P, Turner P. International trial of long-term dexfenfluramine in obesity. Lancet. 1989;2:1142-1145.

508. Finer N. Body weight evolution during dexfenfluramine treatment after initial weight control. Int J Obes Relat Metab Disord. 1992;16 (Suppl 3):S25-S29.

509. Williams R.A., Foulsham BM; Weight reduction in osteoarthritis using phentermine. Practitioner. 1981; 225:231-232.

510. Bray GA, Ryan DH, Gordon D, Heidingsfelder S, Cerise F, Wilson K. A double-blind randomized placebo-controlled trial of sibutramine. Obes Res. 1996;4:263-270.

511. Weintraub M, Ginsberg G, Stein EC, et al. Phenylpropanolamine OROS (Acutrim) vs. placebo in combination with caloric restriction and physician-managed behavior modification. Clin Pharmacol Ther. 1986;39:501-509.

512. Schteingart DE. Effectiveness of phenylpropanolamine in the management of moderate obesity. Int J Obes Relat Metab Disord. 1992;16:487-493.

513. Jones SP, Smith IG, Kelly F, Gray JA. Long term weight loss with sibutramine. Int J Obes. 1995;19 (Suppl 2):41 [abstract 071].

514. Weintraub M, Sundaresan PR, Schuster B, et al. Long-term weight control study. IV (weeks 156 to 190). The second double-blind phase. Clin Pharmacol Ther. 1992;51:608-614.

515. Andersen T, Backer OG, Stokholm KH, Quaade F. Randomized trial of diet and gastroplasty compared with diet alone in morbid obesity. N Engl J Med. 1984;310:352-356.

516. Andersen T, Backer OG, Astrup A, Quaade F. Horizontal or vertical banded gastroplasty after pretreatment with very-low-calorie formula diet: a randomized trial. Int J Obes. 1987;11:295-304.

517. Brolin RE, Kenler HA, Gorman JH, Cody RP. Long-limb gastric bypass in the super-obese. a prospective randomized study. Ann Surg. 1992;215:387-395.

visited on 6/14/2019

518. Naslund I. The size of the gastric outlet and the outcome of surgery for obesity. Acta Chir Scand. 1986;152:205-210.

519. Hall JC, Watts JM, O'Brien PE, et al. Gastric surgery for morbid obesity. The Adelaide Study. Ann Surg. 1990;211:419-427.

520. Laws HL, Piantadosi S. Superior gastric reduction procedure for morbid obesity: a prospective, randomized trial. Ann Surg. 1981;193:334-340.

521. Lechner GW, Callender AK. Subtotal gastric exclusion and gastric partitioning: a randomized prospective comparison of one hundred patients. Surgery. 1981;90:637-644.

522. Pories WJ, Flickinger EG, Meelheim D, Van Rij AM, Thomas FT. The effectiveness of gastric bypass over gastric partition in morbid obesity: consequence of distal gastric and duodenal exclusion. Ann Surg. 1982;196:389-399.

523. Gastrointestinal surgery for severe obesity. National Institutes of Health Consensus Development Conference Statement. Am J Clin Nutr. 1992;55:615S-619S.

524. Busetto L, Perini P, Giantin V, et al. Relationship between energy expenditure and visceral fat accumulation in obese women submitted to adjustable silicone gastric banding (ASGB). Int J Obes Relat Metab Disord. 1995;19:227-233.

525. Zumoff B, Strain GW, Miller LK, et al. Plasma free and non-sex-hormone-binding-globulin-bound testosterone are decreased in obese men in proportion to their degree of obesity. J Clin Endocrinol Metab. 1990;71:929-931.

526. Gallagher D, Visser M, Sepulveda D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? Am J Epidemiol. 1996;143:228-239.

527. Heymsfield SB, Allison DB, Heshka S, Pierson RN Jr. Assessment of human body composition. In: Allison DB, ed. Handbook of Assessment Methods For Eating Behavior and Weight Related Problems: Measures, Theory, and Research. Thousand Oaks, CA: Sage Publications; 1995:515-560.

528. Matz R. Calculating body mass index. Ann Intern Med. 1993;118:232.

529. Abate N, Garg A, Peshock RM, Stray-Gundersen J, Adams-Huet B, Grundy SM. Relationship of generalized and regional adiposity to insulin sensitivity in men with NIDDM. Diabetes. 1996;45:1684-1693.

530. Lean ME, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management. BMJ. 1995;311:158-161.

531. Sjostrom L, Kvist H, Cederblad A, Tylen U. Determination of total adipose tissue and body fat in women by computed tomography, 40K, and tritium. Am J Physiol. 1986;250:E736-E745.

532. Jensen MD, Kanaley JA, Reed JE, Sheedy PF. Measurement of abdominal and visceral fat with computed tomography and dual-energy x-ray absorptiometry. Am J Clin Nutr. 1995;61:274-278.

533. Despres JP, Moorjani S, Ferland M, et al. Adipose tissue distribution and plasma lipoprotein levels in obese women. Importance of intra-abdominal fat. Arteriosclerosis. 1989;9:203-210.

534. Pouliot MC, Despres JP, Nadeau A, et al. Visceral obesity in men. Associations with glucose tolerance, plasma insulin, and

visited on 6/14/2019

lipoprotein levels. Diabetes. 1992;41:826-834.

535. Abate N, Garg A, Peshock RM, Stray-Gundersen J, Grundy SM. Relationships of generalized and regional adiposity to insulin sensitivity in men. J Clin Investigation. 1995;96:88-98.

536. Goodpaster BH, Thaete FL, Thaete JA, Simoneau, Kelley DE. Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. Diabetes. 1997;46:1579-1585.

537. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Despres J. A single threshold value of waist girth identifies normal-weight and overweight subjects with excess visceral adipose tissue. Am J Clin Nutr. 1996;64:685-693.

538. National Center for Health Statistics. NHANES III Anthropometric Procedures Video. Washington, DC: U.S. Government Printing Office; 1996. Stock Number 017-022-01335-5.

539. Gordon T, Doyle JT. Weight and mortality in men: the Albany Study. Int J Epidemiol. 1988;17:77-81.

540. Hamm P, Shekelle RB, Stamler J. Large fluctuations in body weight during young adulthood and 25-year risk of coronary death in men. Am J Epidemiol. 1989;129:312-318.

541. Lindsted K, Tonstad S, Kuzma JW. Body mass index and patterns of mortality among Seventh-day Adventist men. Int J Obes. 1991;15:397-406.

542. Norgan NG, Jones PR. The effect of standardising the body mass index for relative sitting height. Int J Obes Relat Metab Disord. 1995;19:206-208.

543. Conway JM, Yanovski SZ, Avila NA, Hubbard VS. Visceral adipose tissue differences in black and white women. Am J Clin Nutr. 1995;61:765-771.

544. Potts J, Simmons D. Sex and ethnic group differences in fat distribution in young United Kingdom South Asians and Europids. J Clin Epidemiol. 1994;47:837-841.

545. Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. The Sixth Report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (JNC VI). Arch Intern Med. 1997;157:2413-2446.

546. American Diabetes Association. ADA Clinical Practice Recommendations 1998. Diabetes Care. 1998;21(Suppl 1):S1-S93.

547. Fiore MC, Bailey WC, Cohen SJ. Smoking Cessation. U.S. Department of Health and Human Services, PHS, Agency for Health Care Policy and Research. Clinical Practice Guidelines No. 18. 1996.

548. Paffenbarger RS Jr, Hyde RT, Wing AL, Lee IM, Jung DL, Kampert JB. The association of changes in physical-activity level and other lifestyle characteristics with mortality among men. N Engl J Med. 1993;328:538-545.

549. NIH Consensus Conference. Triglyceride, high-density lipoprotein, and coronary heart disease. NIH Consensus Development Panel on Triglyceride, High-Density Lipoprotein, and Coronary Heart Disease. JAMA. 1993;269:505-510.

550. Wadden TA, Wingate BJ. Behavioral assessment and treatment of markedly obese patients. In: Wadden TA, VanItallie TB, eds. Treatment of the Seriously Obese Patient. New York: Guilford Press; 1992:290-330.

visited on 6/14/2019

551. Stunkard AJ. Talking with patients. In: Stunkard AJ, Wadden TA, eds. Obesity Theory and Therapy. New York: Raven Press; 1993:355-363.

552. Wadden TA, Wingate BJ. Compassionate treatment of the obese individual. In: Brownell KD, Fairburn CG, eds. Eating Disorders and Obesity. New York: Basic Books; 1995:564-571.

553. Foster GD, Wadden TA, Vogt RA, Brewer G. What is a reasonable weight loss? Patients' expectations and evaluations of obesity treatment outcome. J Consult Clinl Psychol. 1997;65:79-85.

554. Williamson DF, Serdula MK, Anada RF, Levy A, Byers T. Weight loss attempts in adults: goals, duration, and rate of weight loss. Am J Pub Health. 1992;82:1251-1257.

555. Wing RR, Blair E, Marcus M, Epstein LHH, Harvey J. Year-long weight loss treatment for obese patients with type II diabetes: does including an intermittent very-low-calorie diet improve outcome? Am J Med. 1994;97:354-362.

556. VanItallie TB, Yang M-U. Current concepts in nutrition: diets and weight loss. N Engl J Med. 1977;297:1158-1161.

557. Yang M, VanItallie TB. Effect of energy restriction on body composition and nitrogen balance in obese individuals. In: Wadden TA, VanItallie TB, eds. Treatment of the Seriously Obese Patient. New York: Guilford Press; 1992:83-106.

558. Very low-calorie diets. National Task Force on the Prevention and Treatment of Obesity, National Institutes of Health. JAMA. 1993;270:967-974.

559. Tremblay A, Buemann B, Theriault G, Bouchard C. Body fatness in active individuals reporting low lipid and alcohol intake. Eur J Clin Nutr. 1995;49:824-831.

560. Gruchow HW, Sobocinski KA, Barboriak JJ, Scheller JG. Alcohol consumption, nutrient intake and relative body weight among US adults. Am J Clin Nutr. 1985;42:289-295.

561. de Castro JM, Orozco S. Moderate alcohol intake and spontaneous eating patterns of humans: evidence of unregulated supplementation. Am J Clin Nutr. 1990;52:246-253.

562. Veenstra J, Schenkel JA, van Erp-Baart AM, et al. Alcohol consumption in relation to food intake and smoking habits in the Dutch National Food Consumption Survey. Eur J Clin Nutr. 1993;47:482-489.

563. Tremblay A, Wouters E, Wenker M, St-Pierre S, Bouchard C, Despres JP. Alcohol and a high-fat diet: a combination favoring overfeeding. Am J Clin Nutr. 1995;62:639-644.

564. Tremblay A, St-Pierre S. The hyperphagic effect of a high-fat diet and alcohol intake persists after control for energy density. Am J Clin Nutr. 1996;63:479-482.

565. Poppitt SD, Eckhardt JW, McGonagle J, Murgatroyd PR, Prentice AM. Short-term effects of alcohol consumption on appetite and energy intake. Physiol Behav. 1996;60:1063-1070.

566. Foltin RW, Kelly TH, Fischman MW. Ethanol as an energy source in humans: comparison with dextrose-containing beverages. Appetite. 1993;20:95-110.

567. Butrum RR, Clifford CK, Lanza E. NCI dietary guidelines: rationale. Am J Clin Nutr. 1988;48(3 Suppl):888-895.

568. United States. Public Health Service. Office of the Surgeon General. Nutrition and

visited on 6/14/2019

Health: A Report of the Surgeon General. Washington, DC: Government Printing Office; 1988:192.

569. NIH Consensus Conference. Optimal calcium intake. NIH Consensus Development Panel on Optimal Calcium Intake. JAMA. 1994;272:1942-1948.

570. Wadden TA, Letizia KA. Predictors of attrition and weight loss in patients treated by moderate and severe caloric restriction. In: Wadden TA, VanItallie TB, eds. Treatment of the Seriously Obese Patient. New York: Guilford Press; 1992:383-410.

571. Foreyt JP, Goodrick GK. Factors common to successful therapy for the obese patient. Med Sci Sports Exerc. 1991;23:292-297.

572. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Surgeon General's Report on Physical Activity and Health. Atlanta, GA: CDC; 1996.

573. Blair SN, Kampert JB, Kohl HW III, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA. 1996;276:205-210.

574. Kohl HW, Gordon NF, Villegas JA, Blair SN. Cardiorespiratory fitness, glycemic status, and mortality risk in men. Diabetes Care. 1992;15:184-192.

575. Tremblay A, Nadeau A, Despres JP, St-Jean L, Theriault G, Bouchard C. Long-term exercise training with constant energy intake. 2: effect on glucose metabolism and resting energy expenditure. Int J Obes. 1990;14:75-84.

576. Wing RR, Marcus MD, Blair EH, Burton LR. Psychological responses of obese Type II diabetic subjects to very low-calorie diet. Diabetes Care. 1991;14:596-599.

577. Pate RR, Pratt M, Blair SN, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA. 1995;273:402-407.

578. NIH Consensus Conference. Physical Activity and Cardiovascular Health. JAMA. 1996;276:241-246.

579. Wadden TA, Foster GD. Behavioral assessment and treatment of markedly obese patients. In: Wadden TA, VanItallie TB, eds. Treatment of the Seriously Obese Patient. New York: Guilford Press; 1992:290-330.

580. Wadden TA. The treatment of obesity. In: Stunkard AJ, Wadden TA, eds. Obesity: Theory and Therapy. New York: Raven Press; 1993:197-217.

581. Wilson GT, Fairborn CG. Treatments of eating disorders. In: Nathan PE, Gorman JM, eds. A Guide To Treatments That Work. New York: Oxford University Press; 1997:501-530.

582. Schlundt DG, Hill JO, Pope-Cordle J, Arnold D, Virts KL, Katahn M. Randomized evaluation of a low fat ad libitum carbohydrate diet for weight reduction. Int J Obes Relat Metab Disord. 1993;17:623-629.

583. Tremblay A, Almeras N, Boer J, Kranenbarg EK, Despres JP. Diet composition and postexercise energy balance. Am J Clin Nutr. 1994;59:975-979.

584. King NA, Blundell JE. High-fat foods overcome the energy expenditure induced by high-intensity cycling or running. Eur J Clin Nutr. 1995;49:114-123.

585. Methods for voluntary weight loss and control. NIH Technology Assessment

visited on 6/14/2019

Conference Panel. Ann Intern Med. 1992;116:942-949.

586. Atanassoff PG, Weiss BM, Schmid ER, Tornic M. Pulmonary hypertension and dexfenfluramine. Lancet. 1992;339:436.

587. Brenot F, Herve P, Petitpretz P, Parent F, Duroux P, Simonneau G. Primary pulmonary hypertension and fenfluramine use. Br Heart J. 1993;70:537-541.

588. Voelker R. Obesity drug renews toxicity debate. JAMA. 1994;272:1087-1088.

589. Food and Drug Administration. Draft Guidance Clinical Evaluation of Weight Control Drug. Rockville, MD: FDA; 1996.

590. Bray GA. Use and abuse of appetite-suppressant drugs in the treatment of obesity. Ann Intern Med. 1993;119:707-713.

591. Galloway SM, Farquhar DL, Munro JF. The current status of antiobesity drugs. Postgrad Med J. 1984;60 (Suppl 3):19-26.

592. Long-term pharmacotherapy in the management of obesity. National Task Force on the Prevention and Treatment of Obesity. JAMA. 1996;276:1907-1915.

593. Weintraub M, Bray GA. Drug treatment of obesity. Med Clin North Am. 1989;73:237-249.

594. MacDonald KG Jr, Long SD, Swanson MS, et al. The gastric bypass operation reduces the progression and mortality of non-insulin-dependent diabetes mellitus. J Gastrointest Surg. 1997;1:213-220.

595. Pories WJ, Swanson MS, MacDonald KG Jr, et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. Ann Surg. 1995;222:339-350; discussion 350-352.

596. Harris TB, Savage PJ, Tell GS, Haan M, Kumanyika S, Lynch JC. Carrying the burden of cardiovascular risk in old age: associations of weight and weight change with prevalent cardiovascular disease, risk factors, and health status in the Cardiovascular Health Study. Am J Clin Nutr. 1997;66:837-844.

597. Tayback M, Kumanyika S, Chee E. Body weight as a risk factor in the elderly. Arch Intern Med. 1990;150:1065-1072.

598. Galanos AN, Pieper CF, Cornoni-Huntley JC, Bales CW, Fillenbaum GG. Nutrition and function: is there a relationship between body mass index and the functional capabilities of community-dwelling elderly? J Am Geriatr Soc. 1994;42:368-373.

599. Keil JE, Gazes PC, Sutherland SE, Rust PF, Branch LG, Tyroler HA. Predictors of physical disability in elderly blacks and whites of the Charleston Heart Study. J Clin Epidemiol. 1989;42:521-529.

600. Launer LJ, Harris T, Rumpel C, Madans J. Body mass index, weight change, and risk of mobility disability in middle-aged and older women. The epidemiologic follow-up study of NHANES I. JAMA. 1994;271:1093-1098.

601. Willett WC. Weight loss in the elderly: cause or effect of poor health? Am J Clin Nutr. 1997;66:737-738.

602. Galanis DJ, Harris T, Sharp DS, Petrovitch H. Relative weight, weight change, and risk of coronary heart disease in the Honolulu Heart Program. Am J Epidemiol. 1998;147:379- 386.

603. Chaturvedi N, Fuller JH. Mortality risk by body weight and weight change in people with NIDDM. The WHO Multinational Study of Vascular Disease in Diabetes. Diabetes Care. 1995;18:766-774.

visited on 6/14/2019

604. Iribarren C, Sharp DS, Burchfiel CM, Petrovitch H. Association of weight loss and weight fluctuation with mortality among Japanese-American men. N Engl J Med. 1995;333:686-692.

605. Pamuk ER, Williamson DF, Madans J, Serdula MK, Kleinman JC, Byers T. Weight loss and mortality in a national cohort of adults, 1971-1987. Am J Epidemiol. 1992;136:686-697.

606. Rumpel C, Harris TB, Madans J. Modification of the relationship between the Quetelet index and mortality by weight-loss history among older women. Ann Epidemiol. 1993;3:343-350.

607. Wallace JI, Schwartz RS, LaCroix AZ, Uhlmann RF, Pearlman RA. Involuntary weight loss in older outpatients: incidence and clinical significance. J Am Geriatr Soc. 1995;43:329-337.

608. Ensrud KE, Cauley J, Lipschutz R, Cummings SR. Weight change and fractures in older women. Study of Osteoporotic Fractures Research Group. Arch Intern Med. 1997;157:857-863.

609. Langlois JA, Harris T, Looker AC, Madans J. Weight change between age 50 years and old age is associated with risk of hip fracture in white women aged 67 years and older. Arch Intern Med. 1996;156:989-994.

610. Shapses SA, Heymsfield SB, Ricci TA. Voluntary weight reduction increases bone turnover and loss. In: Burckhardt P, Heaney R, Dawson-Hughes B, eds. Nutritional Aspects of Osteoporosis. New York: Springer-Verlag; 1998.

611. Michel BA, Lane NE, Bloch DA, Jones HH, Fries JF. Effect of changes in weight-bearing exercise on lumbar bone mass after age fifty. Ann Med. 1991;23:397-401.

612. Marcus R, Drinkwater B, Dalsky G, et al. Osteoporosis and exercise in women. Med Sci Sports Exerc. 1992;24(Suppl 6):S301-S307.

613. Gerace TA, Hollis J, Ockene JK, Svendsen K. Smoking cessation and change in diastolic blood pressure, body weight, and plasma lipids. MRFIT Research Group. Prev Med. 1991;20:602-620.

614. Klesges RC, Meyers AW, Klesges LM, La Vasque ME. Smoking, body weight, and their effects on smoking behavior: a comprehensive review of the literature. Psychol Bull. 1989;106:204-230.

615. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in a national cohort. N Engl J Med. 1991;324:739-745.

616. Pirie PL, Murray DM, Luepker RV. Gender differences in cigarette smoking and quitting in a cohort of young adults. Am J Public Health. 1991;81:324-327.

617. Pomerleau CS, Kurth CL. Willingness of female smokers to tolerate postcessation weight gain. J Subst Abuse. 1996;8:371-378.

618. Hall SM, Tunstall CD, Vila KL, Duffy J. Weight gain prevention and smoking cessation: cautionary findings. Am J Public Health. 1992;82:799-803.

619. Spring B, Pingotore R, Kesler K. Cigarette smoking and body weight: strategies to minimize weight gain after smoking cessation. Int J Obes. 1992;16:S19-S23.

620. Perkins KA, Epstein LH, Marks BL, Stiller RL, Jacob RG. The effect of nicotine on energy expenditure during light physical activity. N Engl J Med. 1989;320:898-903.

621. Stamford BA, Matter S, Fell RD, Papanek

visited on 6/14/2019

P. Effects of smoking cessation on weight gain, metabolic rate, caloric consumption, and blood lipids. Am J Clin Nutr. 1986;43:486-494.

622. Gilbert RM, Pope MA. Early effects of quitting smoking. Psychopharmacology (Berl). 1982;78:121-127.

623. Spring B, Wurtman J, Gleason R, Wurtman R, Kessler K. Weight gain and withdrawal symptoms after smoking cessation: a preventive intervention using d-fenfluramine. Health Psychol. 1991;10:216-223.

624. Gross J, Stitzer ML, Maldonado J. Nicotine replacement: effects of postcessation weight gain. J Consult Clin Psychol. 1989;57:87-92.

625. Klesges RC, Klesges LM, Meyers AW, Klem ML, Isbell T. The effects of phenylpropanolamine on dietary intake, physical activity, and body weight after smoking cessation. Clin Pharmacol Ther. 1990;47:747-754.

626. Hurt RD, Sachs DPL, Clover ED, et al. A comparison of sustained-release bupropion and placebo for smoking cessation. N Engl J Med. 1997;337:1195-1202.

627. Perri MG, Lauer JB, Yancey DZ, et al. Effects of peer support and therapist contact on long-term weight loss. J Consult Clin Psych. 19;55:615-617.

628. Broussard BA, Johnson A, Himes JH, et al. Prevalence of obesity in American Indians and Alaska Natives. Am J Clin Nutr. 1991;53:1535S-1542S.

629. Hodge AM, Dowse GK, Toelupe P, Collins VR, Imo T, Zimmet PZ. Dramatic increase in the prevalence of obesity in western Samoa over the 13 year period 1978-1991. Int J Obes Relat Metab Disord. 1994;18:419-428.

630. Health Care Financing Administration. Medicare Current Beneficiary Survey: 1991. Percentage of medicare beneficiaries who are overweight, by race, gender, and education. Published Data. 1991.

631. Avila P, Hovell MF. Physical activity training for weight loss in Latinas: a controlled trial. Int J Obes Relat Metab Disord. 1994;18:476-482.

632. Cousins JH, Rubovits DS, Dunn JK, Reeves RS, Ramirez AG, Foreyt JP. Family versus individually oriented intervention for weight loss in Mexican-American women. Public Health Rep. 1992;107:549-555.

633. Daniel EL. A multi-intervention weight management program for low-income rural women. J Am Diet Assoc. 1989;89:1310-1311.

634. Domel SB, Alford BB, Cattlett HN, Gench BE. Weight control for black women. J Am Diet Assoc. 1992;92:346-348.

635. Domel SB, Alford BB, Cattlett HN, Rodriguez ML, Gench BE. A pilot weight control program for Hispanic women. J Am Diet Assoc. 1992;92:1270-1271.

636. Fox RA, Haniotes H, Rotatori A. A streamlined weight loss program for moderately retarded adults in a sheltered workshop setting. Appl Res Ment Retard. 1984;5:69-79.

637. Heath GW, Wilson RH, Smith J, Leonard BE. Community-based exercise and weight control: diabetes risk reduction and glycemic control in Zuni Indians. Am J Clin Nutr. 1991;53:1642S-1646S.

638. Kanders BS, Ullmann-Joy P, Foreyt JP, et al. The black American lifestyle intervention (BALI): the design of a weight loss program for working-class African-American women. J Am Diet Assoc. 1994;94:310-312.

visited on 6/14/2019

639. Kumanyika SK, Brancato J, Brewer A, et al. Interventions in the Trials of Nonpharmacologic Intervention in the Elderly. Effective approaches to weight and sodium reduction among older adults. Circulation. 1996;94:I-690.

640. Kumanyika SK, Charleston JB. Lose weight and win: a church-based weight loss program for blood pressure control among black women. Patient Educ Counseling. 1992;19:19-32.

641. Lasco RA, Curry RH, Dickson VJ, Powers J, Menes S, Merritt RK. Participation rates, weight loss, and blood pressure changes among obese women in a nutrition-exercise program. Public Health Rep. 1989;104:640-646.

642. McNabb W, Quinn M, Kerver J, Cook S, Karrison T. The PATHWAYS church-based weight loss program for urban African-American women at risk for diabetes. Diabetes Care. 1997;20:1518-1523.

643. McNabb WL, Quinn MT, Rosing L. Weight loss program for inner-city black women with non-insulin-dependent diabetes mellitus: Pathways. J Am Diet Assoc. 1993;93:75-77.

644. Mount MA, Kendrick OW, Draughon M, Stitt KR, Head D, Mount R. Group participation as a method to achieve weight loss and blood glucose control. J Nutr Educ. 1991;23:25-29.

645. Mulrow C, Bailey S, Sonksen PH, Slavin B. Evaluation of an audiovisual diabetes education program: Negative results of a randomized trial of patients with non-insulin-dependent diabetes mellitus. J Gen Intern Med. 1987;2:215-219.

646. Pleas J. Long-term effects of a lifestyle-change obesity treatment program with minorities. J Natl Med Assoc. 1988;80:747-752.

647. Shintani TT, Hughes CK, Beckham S, O'Connor HK. Obesity and cardiovascular risk intervention through an ad libitum feeding of traditional Hawaiian diet. Am J Clin Nutr. 1991;53:1647S-1651S.

648. Sullivan J, Carter JP. A nutrition-physical fitness intervention program for low-income black parents. J Natl Med Assoc. 1985;77:39-43.

649. Gleadhill IC, Schwartz AR, Schubert N, Wise RA, Permutt S, Smith PL. Upper airway collapsibility in snorers and in patients with obstructive hypopnea and apnea. Am Rev Respir Dis. 1991;143:1300-1303.

650. Gold AR, Schwartz AR, Wise RA, Smith PL. Pulmonary function and respiratory chemosensitivity in moderately obese patients with sleep apnea. Chest. 1993;103:1325-1329.

651. Block AJ, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea, and oxygen desaturation in normal subjects. A strong male predominance. N Engl J Med. 1979;300:513-517.

652. Guilleminault C, Dement WC. Sleep Apnea Syndromes. Conference on Sleep Apnea Syndromes. New York: Liss; 1978.

653. Schmidt-Nowara WW. Cardiovascular consequences of sleep apnea. Prog Clin Biol Res. 1990;345:377-385.

654. Norton PG, Dunn EV. Snoring as a risk factor for disease: an epidemiological survey. BMJ (Clin Res Ed). 1985;291:630-632.

655. Jennum P, Hein HO, Suadicani P, Gyntelberg F. Cardiovascular risk factors in snorers. A cross-sectional study of 3,323 men aged 54 to 74 years: the Copenhagen Male Study. Chest. 1992;102:1371-1376.

656. Stradling JR, Crosby JH. Predictors and

visited on 6/14/2019

prevalence of obstructive sleep apnoea and snoring in 1001 middle aged men. Thorax. 1991;46:85-90.

657. Guilleminault C, Quera-Salva MA, Partinen M, Jamieson A. Women and the obstructive sleep apnea syndrome. Chest. 1988;93:104-109.

658. Connolly HM, Crary JL, McGoon MD, et al. Valvular heart disease associated with Fenfluramine—Phentermine. N Engl J Med. 1997;337:581-588.

659. Hedner J. Vascular function in OSA. Sleep. 1996;19(10 Suppl):S213-S217.

660. Tofler GH, Brezinski D, Schafer AI, et al. Concurrent morning increase in platelet aggregability and the risk of myocardial infarction and sudden cardiac death. N Engl J Med. 1987;316:1514-1518.

661. He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. Chest. 1988;94:9-14.

662. Partinen M, Jamieson A, Guilleminault C. Long-term outcome for obstructive sleep apnea syndrome patients. Mortality. Chest. 1988;94:1200-1204.

663. Harman EM, Wynne JW, Block AJ. The effect of weight loss on sleep-disordered breathing and oxygen desaturation in morbidly obese men. Chest. 1982;82:291-294.

664. Charuzi I, Ovnat A, Peiser J, Saltz H, Weitzman S, Lavie P. The effect of surgical weight reduction on sleep quality in obesity-related sleep apnea syndrome. Surgery. 1985;97:535-538.

665. Peiser J, Lavie P, Ovnat A, Charuzi I. Sleep apnea syndrome in the morbidly obese as an indication for weight reduction surgery. Ann Surg. 1984;199:112-115.

666. Sugerman HJ, Fairman RP, Baron PL, Kwentus JA. Gastric surgery for respiratory insufficiency of obesity. Chest. 1986;90:81-86.

667. Rubinstein I, Colapinto N, Rotstein LE, Brown IG, Hoffstein V. Improvement in upper airway function after weight loss in patients with obstructive sleep apnea. Am Rev Respir Dis. 1988;138:1192-1195.

668. Smith PL, Gold AR, Meyers DA, Haponik EF, Bleecker ER. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med. 1985;103:850-855.

669. Aubert-Tulkens G, Culee C, Rodenstein DO. Cure of sleep apnea syndrome after long-term nasal continuous positive airway pressure therapy and weight loss. Sleep. 1989;12:216-222.

670. Pasquali R, Colella P, Cirignotta F, et al. Treatment of obese patients with obstructive sleep apnea syndrome (OSAS): effect of weight loss and interference of otorhinolaryngoiatric pathology. Int J Obes. 1990;14:207-217.

671. Suratt PM, McTier RF, Findley LJ, Pohl SL, Wilhoit SC. Effect of very low-calorie diets with weight loss on obstructive sleep apnea. Am J Clin Nutr. 1992;56:182S-184S.

672. Schwartz AR, Gold AR, Schubert N, et al. Effect of weight loss on upper airway collapsibility in obstructive sleep apnea. Am Rev Respir Dis. 1991;144:494-498.

673. American Thoracic Society. Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. Am J Resp Crit Care Med. 1994;150:1738-1745.

674. Andersson B, Elam M, Wallin BG, Bjorntorp P, Andersson OK. Effect of energy-restricted diet on sympathetic muscle

visited on 6/14/2019

nerve activity in obese women. Hypertension. 1991;18:783- 789.

675. Andersson OK, Fagerberg B, Hedner T. Haemodynamic adjustment to weight reduction—separate effects of energy versus salt restriction. J Hypertens Suppl. 1983;1:35-37.

676. Collins RW, Anderson JW. Medication cost savings associated with weight loss for obese non-insulin-dependent diabetic men and women. Prev Med. 1995;24:369-374.

677. Darne B, Nivarong M, Tugaye A, et al. Hypocaloric diet and antihypertensive drug treatment. A randomized controlled clinical trial. Blood Press. 1993;2:130-135.

678. Gillett PA, Eisenman PA. The effect of intensity controlled aerobic dance exercise on aerobic capacity of middle-aged, overweight women. Res Nurs Health. 1987;10:383-390.

679. The treatment of mild hypertension study. A randomized, placebo-controlled trial of a nutritional-hygienic regimen along with various drug monotherapies. Arch Intern Med. 1991;151:1413-1423.

680. Jeffery RW, Gillum R, Gerber WM, Jacobs D, Elmer PJ, Prineas RJ. Weight and sodium reduction for the prevention of hypertension: a comparison of group treatment and individual counseling. Am J Public Health. 1983;73:691-693.

681. Kanaley JA, Andresen-Reid ML, Oenning L, Kottke BA, Jensen MD. Differential health benefits of weight loss in upper-body and lower-body obese women. Am J Clin Nutr. 1993;57:20-26.

682. Kumanyika SK. The impact of obesity on hypertension management in African Americans. J Health Care Poor Underserved. 1997;8:352-364.

683. Mellies MJ, Vitale C, Jandacek RJ, Lamkin GE, Glueck CJ. The substitution of sucrose polyester for dietary fat in obese, hypercholesterolemic outpatients. Am J Clin Nutr. 1985;41:1-12.

684. Page RC, Harnden KE, Cook JT, Turner RC. Can lifestyles of subjects with impaired glucose tolerance be changed? A feasibility study. Diabet Med. 1992;9:562-566.

685. Ready AE, Drinkwater DT, Ducas J, Fitzpatrick DW, Brereton DG, Oades SC. Walking program reduces elevated cholesterol in women postmenopause. Can J Cardiol. 1995;11:905-912.

686. Rigaud D, Ryttig KR, Angel LA, Apfelbaum M. Overweight treated with energy restriction and a dietary fibre supplement: a 6-month randomized, double-blind, placebo-controlled trial. Int J Obes. 1990;14:763-769.

687. Rissanen A, Pietinen P, Siljamaki-Ojansuu U, Piirainen H, Reissel P. Treatment of hypertension in obese patients: efficacy and feasibility of weight and salt reduction programs. Acta Med Scand. 1985;218:149-156.

688. Ryttig KR, Rossner S. Weight maintenance after a very low calorie diet (VLCD) weight reduction period and the effects of VLCD supplementation. A prospective, randomized, comparative, controlled long-term trial. J Intern Med. 1995;238:299-306.

689. Singh RB, Niaz MA, Bishnoi I, Singh U, Begum R, Rastogi SS. Effect of low energy diet and weight loss on major risk factors, central obesity and associated disturbances in patients with essential hypertension. J Hum Hypertens. 1995;9:355-362.

690. Singh RB, Niaz MA, Ghosh S. Effect on

visited on 6/14/2019

central obesity and associated disturbances of low-energy, fruit- and vegetable-enriched prudent diet in north Indians. Postgrad Med J. 1994;70:895-900.

691. Singh RB, Rastogi SS, Sircar AR, Mehta PJ, Sharma KK. Dietary strategies for risk-factor modification to prevent cardiovascular diseases. Nutrition. 1991;7:210-214.

692. Stokholm KH, Nielsen PE, Quaade F. Correlation between initial blood pressure and blood pressure decrease after weight loss: a study in patients with jejunoileal bypass versus medical treatment for morbid obesity. Int J Obes. 1982;6:307-312.

693. Walker KZ, O'Dea K, Nicholson GC, Muir JG. Dietary composition, body weight, and NIDDM. Comparison of high-fiber, high-carbohydrate, and modified-fat diets. Diabetes Care. 1995;18:401-403.

694. Wing RR, Jeffery RW, Burton LR, Thorson C, Kuller LH, Folsom AR. Change in waist-hip ratio with weight loss and its association with change in cardiovascular risk factors. Am J Clin Nutr. 1992;55:1086-1092.

695. Astrup A, Breum L, Toubro S, Hein P, Quaade F. The effect and safety of an ephedrine/caffeine compound compared to ephedrine, caffeine and placebo in obese subjects on an energy-restricted diet. A double blind trial. Int J Obes Relat Metab Disord. 1992;16:269-277.

696. Connacher AA, Jung RT, Mitchell PE. Weight loss in obese subjects on a restricted diet given BRL 26830A, a new atypical beta adrenoceptor agonist. BMJ (Clin Res Ed). 1988;296:1217-1220.

697. Daly PA, Krieger DR, Dulloo AG, Young JB, Landsberg L. Ephedrine, caffeine and aspirin: safety and efficacy for treatment of human obesity. Int J Obes Relat Metab Disord. 1993;17 (Suppl 1):S73-S78.

698. Marin P, Holmang S, Jonsson L, et al. The effects of testosterone treatment on body composition and metabolism in middle-aged obese men. Int J Obes Relat Metab Disord. 1992;16:991-997.

699. Rasmussen MH, Andersen T, Breum L, Gotzsche PC, Hilsted J. Cimetidine suspension as adjuvant to energy restricted diet in treating obesity. BMJ. 1993;306:1093-1096.

700. Coon PJ, Bleecker ER, Drinkwater DT, Meyers DA, Goldberg AP. Effects of body composition and exercise capacity on glucose tolerance, insulin, and lipoprotein lipids in healthy older men: a cross-sectional and longitudinal intervention study. Metabolism. 1989;38:1201-1209.

701. Franz MJ, Monk A, Barry B, et al. Effectiveness of medical nutrition therapy provided by dietitians in the management of non-insulin-dependent diabetes mellitus: a randomized, controlled clinical trial. J Am Diet Assoc. 1995;95:1009-1017.

702. Milne RM, Mann JI, Chisholm AW, Williams SM. Long-term comparison of three dietary prescriptions in the treatment of NIDDM. Diabetes Care. 1994;17:74-80.

703. Ready AE, Fitzpatrick DW, Boreskie SL, Hrycaiko DW. The response of obese females to low impact exercise and diet counselling. J Sports Med Phys Fitness. 1991;31:587-595.

704. Singman HS, Berman SN, Cowell C, Maslansky E, Archer M. The Anti-Coronary Club: 1957 to 1972. Am J Clin Nutr. 1980;33:1183-1191.

705. Suter E, Marti B, Gutzwiller F. Jogging or

visited on 6/14/2019

walking—comparison of health effects. Ann Epidemiol. 1994;4:375-381.

706. Viddal KO. Intestinal bypass. A randomized, prospective clinical study of end-to-side and end-to-end jejunoileal bypass. Scand J Gastroenterol. 1983;18:627-634.

707. Williams PT, Wood PD, Haskell WL, Vranizan K. The effects of running mileage and duration on plasma lipoprotein levels. JAMA. 1982;247:2674-2679.

708. Wing RR, Marcus MD, Salata R, Epstein LH, Miaskiewicz S, Blair EH. Effects of a very low-calorie diet on long-term glycemic control in obese Type 2 diabetic subjects. Arch Intern Med. 1991;151:1334-1340.

709. Wing RR, Blair E, Marcus M, Epstein LH, Harvey J. Year-long weight loss treatment for obese patients with type II diabetes: does including an intermittent very low-calorie diet improve outcome? Am J Med. 1994;97:354-362.

710. Drent ML, Larsson I, William-Olsson T, et al. Orlistat (Ro 18-0647), a lipase inhibitor, in the treatment of human obesity: a multiple dose study. Int J Obes Relat Metab Disord. 1995;19:221-226.

711. Pedrinola F, Sztejnsznajd C, Lima N, Halpern A, Medeiros-Neto G. The addition of dexfenfluramine to fluoxetine in the treatment of obesity: a randomized clinical trial. Obes Res. 1996;4:549-554.

712. Connolly VM, Gallagher A, Kesson CM. A study of fluoxetine in obese elderly patients with type 2 diabetes. Diabet Med. 1995;12:416-418.

713. Alpert MA, Terry BE, Lambert CR, et al. Factors influencing left ventricular systolic function in nonhypertensive morbidly obese patients, and effect of weight loss induced by gastroplasty. Am J Cardiol. 1993;71:733-737.

714. Cairella G, Cairella M, Marchini G. Effect of dietary fiber on weight correction after modified fasting. Eur J Clin Nutr. 1995;49 (Suppl 3):S325-S327.

715. Foster GD, Wadden TA, Peterson FJ, Letizia KA, Bartlett SJ, Conill AM. A controlled comparison of three very low-calorie diets: effects on weight, body composition, and symptoms. Am J Clin Nutr. 1992;55:811-817.

716. Hakala P. Weight reduction programmes at a rehabilitation centre and a health centre based on group counselling and individual support: short- and long-term follow-up study. Int J Obes Relat Metab Disord. 1994;18:483-489.

717. Han TS, Richmond P, Avenell A, Lean ME. Waist circumference reduction and cardiovascular benefits during weight loss in women. Int J Obes Relat Metab Disord. 1997;21:127-134.

718. Lean ME, Han TS, Prvan T, Richmond PR, Avenell A. Weight loss with high and low carbohydrate 1200 kcal diets in free living women. Eur J Clin Nutr. 1997;51:243-248.

719. Miura J, Arai K, Tsukahara S, Ohno M, Ikeda Y. The long-term effectiveness of combined therapy by behavior modification and very low-calorie diet: 2 years followup. Int J Obes. 1989;13 (Suppl 2):73-77.

720. Walker KZ, O'Dea K, Johnson L, et al. Body fat distribution and non-insulin-dependent diabetes: comparison of a fiber-rich, high-carbohydrate, low-fat (23%) diet and a 35% fat diet high in monounsaturated fat. Am J Clin Nutr. 1996;63:254-260.

721. Foster GD, Wadden TA, Feurer ID, et al. Controlled trial of the metabolic effects of a very low-calorie diet: short- and long-

visited on 6/14/2019

term effects. Am J Clin Nutr. 1990;51:167-172.

722. Golay A, Allaz AF, Morel Y, de Tonnac N, Tankova S, Reaven G. Similar weight loss with low- or high-carbohydrate diets. Am J Clin Nutr. 1996;63:174-178.

723. Rossner S, Flaten H. VLCD versus LCD in long-term treatment of obesity. Int J Obes Relat Metab Disord. 1997;21:22-26.

724. Jarrett RJ, Keen H, Murrells T. Changes in blood pressure and body weight over ten years in men selected for glucose intolerance . J Epidemiol Community Health. 1987;41:145-151.

725. Gwinup G. Weight loss without dietary restriction: efficacy of different forms of aerobic exercise. Am J Sports Med. 1987;15:275-279.

726. Hespel P, Lijnen P, Van Hoof R, et al. Effects of physical endurance training on the plasma renin-angiotensin-aldosterone system in normal man. J Endocrinol. 1988;116:443-449.

727. King AC, Taylor CB, Haskell WL, DeBusk RF. Influence of regular aerobic exercise on psychological health: a randomized, controlled trial of healthy middle-aged adults. Health Psychol. 1989;8:305-324.

728. Karvetti RL, Knuts LR. Effects of comprehensive rehabilitation on weight reduction in myocardial infarction patients. Scand J Rehabil Med. 1983;15:11-16.

729. Liao Y, Emidy LA, Gosch FC, Stamler R, Stamler J. Cardiovascular responses to exercise of participants in a trial on the primary prevention of hypertension. J Hypertens. 1987;5:317-321.

730. Darga LL, Carroll-Michals L, Botsford SJ, Lucas CP. Fluoxetine's effect on weight loss in obese subjects. Am J Clin Nutr. 1991;54:321-325.

731. Elmer PJ, Grimm R Jr, Laing B, et al. Lifestyle intervention: results of the Treatment of Mild Hypertension Study (TOMHS). Prev Med. 1995;24:378-388.

732. Foreyt JP, Ramirez AG, Cousins JH. Cuidando El Corazon—a weight-reduction intervention for Mexican Americans. Am J Clin Nutr. 1991;53:1639S-1641S.

733. Hogan RB, Johnston JH, Long BW, et al. A double-blind, randomized, sham-controlled trial of the gastric bubble for obesity. Gastrointest Endosc. 1989;35:381-385.

734. Marcus MD, Wing RR, Ewing L, Kern E, McDermott M, Gooding W. A double-blind, placebo-controlled trial of fluoxetine plus behavior modification in the treatment of obese binge-eaters and non-binge-eaters. Am J Psychiatry. 1990;147:876-881.

735. Stamler J, Briefel RR, Milas C, Grandits GA, Caggiula AW. Relation of changes in dietary lipids and weight, trial years 1-6, to changes in blood lipids in the special intervention and usual care groups in the Multiple Risk Factor Intervention Trial. Am J Clin Nutr. 1997;65:272S-288S.

736. Wing RR, Shoemaker M, Marcus MD, McDermott M, Gooding W. Variables associated with weight loss and improvements in glycemic control in type II diabetic patients in behavioral weight control programs. Int J Obes. 1990;14:495-503.

737. DeLucia JL, Kalodner CR, Horan JJ. The effect of two nutritional software programs used as adjuncts to the behavioral treatment of obesity. J Subst Abuse. 1988-1989;1:203-208.

738. DeLucia JL, Kalodner CR. An individualized cognitive intervention: does it increase the efficacy of behavioral interventions for obesity? Addict Behav. 1990;15:473-479.

739. Heitzmann CA, Kaplan RM, Wilson DK,

visited on 6/14/2019

Sandler J. Sex differences in weight loss among adults with type II diabetes mellitus. J Behav Med. 1987;10:197-211.

740. Jeffery RW, Forster JL, French SA, et al. The Healthy Worker Project: a work-site intervention for weight control and smoking cessation. Am J Public Health. 1993;83:395- 401.

741. Jeffery RW, Forster JL, Snell MK. Promoting weight control at the worksite: a pilot program of self-motivation using payroll-based incentives. Prev Med. 1985;14:187-194.

742. Jones SE, Owens HM, Bennett GA. Does behaviour therapy work for dietitians? An experimental evaluation of the effects of three procedures in a weight reduction clinic. Hum Nutr Appl Nutr. 1986;40:272-281.

743. Kalodner CR, DeLucia JL. The individual and combined effects of cognitive therapy and nutrition education as additions to a behavior modification program for weight loss. Addict Behav. 1991;16:255-263.

744. Schwartz SH, Inbar-Saban N. Value self-confrontation as a method to aid in weight loss. J Pers Soc Psychol. 1988;54:396-404.

745. Breum L, Astrup A, Andersen T, et al. The effect of long-term dexfenfluramine treatment on 24-hour energy expenditure in man. A double-blind placebo controlled study. Int J Obes. 1990;14:613-621.

746. Finer N, Finer S, Naoumova RP. Drug therapy after very-low-calorie diets. Am J Clin Nutr. 1992;56:195S-198S.

747. Pasquali R, Cesari MP, Melchionda N, Stefanini C, Raitano A, Labo G. Does ephedrine promote weight loss in low-energy-adapted obese women? Int J Obes. 1987;11:163-168.

748. Weintraub M, Hasday JD, Mushlin AI, Lockwood DH. A double-blind clinical trial in weight control. Use of fenfluramine and phentermine alone and in combination. Arch Intern Med. 1984;144:1143-1148.

749. Weintraub M, Sundaresan PR, Schuster B, et al. Long-term weight control study. II (weeks 34 to 104). An open-label study of continuous fenfluramine plus phentermine versus targeted intermittent medication as adjuncts to behavior modification, caloric restriction, and exercise. Clin Pharmacol Ther. 1992;51:595-601.

750. Bitsch M, Skrumsager BK. Femoxetine in the treatment of obese patients in general practice. A randomized group comparative study with placebo. Int J Obes. 1987;11:183-190.

751. Fernandez-Soto ML, Gonzalez-Jimenez A, Barredo-Acedo F, Luna del Castillo JD, Escobar-Jimenez F. Comparison of fluoxetine and placebo in the treatment of obesity. Ann Nutr Metab. 1995;39:159-163.

752. Goldstein DJ, Rampey AH Jr, Enas GG, Potvin JH, Fludzinski LA, Levine LR. Fluoxetine: a randomized clinical trial in the treatment of obesity. Int J Obes Relat Metab Disord. 1994;18:129-135.

753. Cook RF, Howard AN, Mills IH. Low-dose mianserin as adjuvant therapy in obese patients treated by a very-low-calorie diet. Int J Obes. 1981;5:267-272.

754. Steel JM, Munro JF, Duncan LJ. A comparative trial of different regimens of fenfluramine and phentermine in obesity. Practitioner. 1973;211:232-236.

755. Sax L. Yohimbine does not affect fat distribution in men. Int J Obes. 1991;15:561-565.

756. Benjamin SB, Maher KA, Cattau EL Jr, et

visited on 6/14/2019

al. Double-blind controlled trial of the Garren-Edwards gastric bubble: an adjunctive treatment for exogenous obesity. Gastroenterology. 1988;95:581-588.

757. Mathus-Vliegen EM, Tytgat GN, Veldhuyzen-Offermans EA. Intragastric balloon in the treatment of super-morbid obesity. Double-blind, sham-controlled, crossover evaluation of 500-milliliter balloon. Gastroenterology. 1990;99:362-369.

758. Bray GA, Gray DS. Obesity. Part I—Pathogenesis. West J Med. 1988;149:429-441.

759. Centers for Disease Control and Prevention. Number and percentage of children and adolescents who were overweight by gender and race/ethnicity: United States NHANES III, 1988-1994. Morb Mortal Wkly Rep. 1997.

760. National Center of Health Statistics. Center of Disease Prevention and Control, National Health and Nutrition Examination Survey, Phase III. Prevalence of overweight in U.S. adults, by race/ethnicity, gender, and education, 1988-1991.

761. Centers for Disease Control. Cardiac valvulopathy associated with exposure to Fenfluramine or Dexfenfluramine: U.S. Department of Health and Human Services interim public health recommendation, November 1997. MMWR Morb Mortal Wkly Rep. 1997;46:1061-1065.

762. American Psychiatric Association. Task Force on DSM-IV, Diagnostic and Statistical Manual of Mental Disorders : DSM-IV. 4th ed. Washington, DC: The Association; 1994.

763. Landin K, Stigendal L, Eriksson E, et al. Abdominal obesity is associated with an impaired fibrinolytic activity and elevated plasminogen activator inhibitor-I. Metabolism. 1990;39:1044-1048.

764. Reisin E, Frohlich ED, Messerli FH, Dreslinski GR, et al. Cardiovascular changes after weight reduction in obesity hypertension. Ann Intern Med. 1983;98: 315-319.

765. Tuck MI, Sowers J, Dornfield L, et al. The effect of weight reduction on blood pressure plasma renin activity and plasma aldosterone level in obese patients. N Engl J Med. 1981;304:930-933.

766. Rocchini AP, Key J, Bondie D, et al. The effect of weight loss on the sensitivity of blood pressure to sodium in obese adolescents. N Engl J Med. 1989;321:580-585.

767. Landsberg L, Krieger DR, Obesity, metabolism and the sympathetic nervous system. Am J Hypertension. 1989;2:125S-132S.

768. Jacobs DB, Sowers JR, et al. Effects of weight reduction on cellular cation metabolism and vascular resistance. Hypertension. 1993;21:308-314.

769. Frohlich ED, Apstein C, Chobanian AV, et al. The heart in hypertension. N Engl J Med. 1992;327:998-1008.

visited on 6/14/2019

Publication List

Reports and Materials on Cardiovascular Health from the National Heart, Lung, and Blood Institute

Unless otherwise noted, contact the NHLBI Information Center for the price and availability of the publications listed below:
NHLBI Information Center
P.O. Box 30105
Bethesda, MD 20824-0105
e-mail:nhlbiic@dgsys.com

Obesity and Physical Activity

Strategy Development Workshop for Public Education on Weight and Obesity--Summary Report. Summary of a 1992 workshop held to identify issues in educating the public, identifying priority audiences, approaches, and communications channels for weight and physical activity. *Available at http: //www.nhlbi.nih.gov/nhlbi/cardio/obes/prof/obeshc.htm*

Methods for Voluntary Weight Loss and Control (National Institutes of Health Technology Assessment Conference). An edited summary of a 1992 NIH Technology Assessment Conference on American weight loss practices, success of methods of weight loss and control, short- and long-term benefits and adverse effects of weight loss, principles for selecting a weight loss and control strategy, and future directions for research. *Available at http://text.nlm.nih.gov/nih/ta/www/10.html*

[Full proceedings available in Danford, D., Fletcher, S.W. Methods for Voluntary Weight Loss and Control, National Institutes of Health Technology Assessment Conference. Annals of Internal Medicine Supplement, 119(7pt2)641-770(1993).]

NIH Consensus Development Conference on Physical Activity and Cardiovascular Health. An edited summary of a 1995 NIH Consensus Development Conference which examined the accumulating evidence on the role of physical activity in the prevention and treatment of cardiovascular disease and its risk factors. *Available at http: //odp.od.nih.gov/consensus/ statements/cdc/intros/physact_intro.html*

[Full proceedings available from Leon, A. (ed.) Physical Activity and Cardiovascular Health: A National Consensus. Human Kinetics:1995.]

High Blood Pressure

The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNCVI). Provides guidance for primary care clinicians on the prevention, detection, treatment, and control of high blood pressure. Lifestyle modification and pharmacotherapy are discussed. Treatment algorithms and patient handouts are included.

Working Group Report on Hypertension in Diabetes. Guides clinicians in the care of persons with hypertension and diabetes. Addresses epidemiological, diagnostic, clinical consideration, and special concerns, such as kidney disease, sexual dysfunction, obesity, and pregnancy.

Working Group Report on Hypertension in the Elderly. Assists clinicians in their care of elderly patients with high blood pressure and health care professionals in hypertension control programs that serve the elderly. Presents the prevalence and epidemiology, benefits of antihypertensive therapy, detection and evaluation, therapy, and management considerations for the fastest growing special population in the United States.

visited on 6/14/2019

Controlling High Blood Pressure in Older Women: Clinical Reference Manual. Assists clinicians in the care of older patients with high blood pressure, especially women. Presents the benefits of anithypertensive therapy, detection, evaluation, and treatment strategies. Includes charts to help clinicians improve patient adherence to therapy and select antihypertensive drug therapy.

High Blood Cholesterol

Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP II). Provides guidelines for detecting, evaluating, and treating high blood cholesterol in adult patients. Discusses classification of blood cholesterol, patient evaluation, and dietary and drug treatment. Includes helpful tools for the clinician including a dietary questionnaire, menus, and patient handouts.

Cholesterol Lowering in the Patient with Coronary Heart Disease: Physician Monograph. Reviews the scientific evidence that cholesterol lowering in patients with coronary heart disease produces dramatic benefits. Provides guidance on implementing dietary and drug treatment and improving patient adherence to lowering cholesterol in the patient with coronary heart disease.

Enhance your Patient Education with NHLBI Materials

Obesity and Physical Activity

Check Your Weight and Heart Disease I.Q. A true/false quiz that helps the public determine what they know about weight and heart disease. Correct answers are explained on the back of the quiz sheet.

Check Your Physical Activity and Heart Disease I.Q. Uncovers exercise myths and provides information on ways to improve heart health through physical activity.

Exercise and Your Heart: A Guide to Physical Activity. Provides your patients with information on the effects of physical activity on their heart, and practical guidelines for starting and staying on their own exercise program. The benefits, risks, and common myths about exercise are discussed, and tips and sample activity programs are included.

High Blood Pressure

High Blood Pressure: Treat It For Life. Helps patients take action against high blood pressure, by losing weight if overweight, increasing physical activity, choosing foods lower in salt and sodium, limiting alcohol, and taking medication as prescribed. Includes information for women taking birth control pills, older persons, African Americans, and people with diabetes or high blood cholesterol. A sample walking program, menu ideas, and recipes are included.

Eat Right to Help Lower Your High Blood Pressure. Presents practical information in an easy to read (5th grade level) conversational text brochure presented in large type with colorful illustrations. Helps patients reduce high blood pressure by losing weight, being more active, drinking less alcohol, and using less salt and sodium.

Facts About The DASH Diet. Describes the eating plan from the "Dietary Approaches to Stop Hypertension" (DASH) study, which was shown to lower blood pressure. The diet is rich in fruits, vegetables, and lowfat dairy products, and lower in saturated fat, total fat, and cholesterol. A full week's menu, recipes, and helpful tips are included.

Controlling High Blood Pressure: A Woman's Guide. Explains how high blood pressure affects women's health and tells the simple steps used to prevent and control high blood pressure, as well as the types of medication used to treat the condition. A table of generic names of blood pressure medications and a chart for recording blood pressure readings are given.

High Blood Cholesterol

So You Have High Blood Cholesterol. Gives patients the information on what they need to know about and what they need to do to lower their high blood cholesterol level. Diet, physical activity, weight loss strategies, and medications are discussed. Case studies highlight stategies for action.

visited on 6/14/2019

Step by Step: Eating To Lower Your High Blood Cholesterol. Advises patients on how to make diet and lifestyle changes to lower their high blood cholesterol levels. Explains the Step I and Step II diets and gives practical tips for buying and preparing foods, eating out, increasing physical activity and selecting a weight loss program. Sample menus at two calorie levels and nutrient content tables of a wide variety of foods are included.

Eat Right to Lower Your High Blood Cholesterol. Provides practical information on dietary changes to high blood cholesterol in an easy-to-read (5th grade) conversation style. Includes shopping and cooking tips, sample menu and snack suggestions, and a tear-off shopping guide.

Live Healthier, Live Longer: Lowering Cholesterol for the Person With Heart Disease. Gives the patient with coronary heart disease information on how to lower high blood cholesterol through diet, physical activity and weight control to prevent future heart attacks and improve the quality of life. Practical information included on choosing foods lower in saturated fat, total fat, and cholesterol.

Multiple Risk Factors

Healthy Heart Handbook for Women. Tells women (both with and without heart disease) how to take action to make their hearts healthier. Topics include how to talk to the doctor, blood pressure and blood cholesterol, physical activity, weight loss, hormone replacement therapy, heart attack symptoms, and heart-healthy eating.

Package of Eight Easy-To-Read Booklets in Spanish and English on Preventing Heart Disease. Written especially for Latino families, these booklets include: Watch Your Weight, Stay Active and Feel Better, Cut Down on Fat--Not Taste, Take Steps--Prevent High Blood Pressure, Cut Down on Salt and Sodium, Learn Your Cholesterol Number, Protect Your Heart--Lower Your Blood Cholesterol, and Kick the Smoking Habit.

Delicious Heart-Healthy Latino Recipes (bilingual). Provides 23 traditional Latino recipes lower in calories, fat, saturated fat, cholesterol, and sodium.

Improving Cardiovascular Health in African Americans--Package of Seven Easy-To-Read Booklets. Written especially for African Americans, these booklets include: Lose Weight If You Are Overweight--Embrace Good Health, Stay Physically Active--Energize Yourself, Prevent High Blood Pressure--Protect Your Heart, Eat Less Salt and Sodium--Spice Up Your Life, Learn Your Cholesterol Number--Empower Yourself, Eat Foods Lower in Saturated Fat and Cholesterol--Be Heart Smart, Stop Smoking--Refresh Yourself.

Heart-Healthy Home Cooking--African American Style. Provides 20 favorite African American recipes lower in calories, fat, saturated fat, cholesterol, and sodium.

Stay up-to-date on line—

- Visit the NHLBI website and find many of these publications on line at — www.nhlbi.nih.gov/nhlbi/nhlbi.htm.
- Learn of new publications as they become available by joining the NHLBI listserve.

It's easy as 1, 2, 3!

1. Address an e-mail message to LISTSERV@LIST.NIH.GOV. No subject is necessary.
2. In the body of the message type in SUBSCRIBE NHLBIINFO.
3. A confirmation will be sent to you. When the confirmation is received, send a reply message and type "OK" in the body of the reply.

visited on 6/14/2019

Discrimination Prohibited: Under provisions of applicable public laws enacted by Congress since 1964, no person in the United States shall, on the grounds of race, color, national origin, handicap, or age, be excluded from participation in, be denied the benefits of, or be subjected to discrimination under any program or activity (or, on the basis of sex, with respect to any education program or activity) receiving Federal financial assistance. In addition, Executive Order 11141 prohibits discrimination on the basis of age by contractors and subcontractors in the performance of Federal contracts, and Executive Order 11246 states that no federally funded contractor may discriminate against any employee or applicant for employment because of race, color, religion, sex, or national origin. Therefore, the Obesity Initiative Program must be operated in compliance with these laws and Executive Orders.

visited on 6/14/2019

U.S. DEPARTMENT OF HEALTH AND
HUMAN SERVICES

Public Health Service
National Institutes of Health
National Heart, Lung, and Blood Institute

NIH Publication No. 98-4083
September 1998