Contributors

EDITORS

Nathan D Wong PhD MPH FACC FAHA FNLA
Professor and Director
Heart Disease Prevention Program
Division of Cardiology, Department of Medicine
Adjunct Professor, Departments of Epidemiology and Radiology
University of California
Irvine, California, USA

Shaista Malik MD PhD FACC
Associate Professor of Medicine
Medical Director, Preventive Cardiology,
Cardiac Rehabilitation, and Women's Heart Programs
Division of Cardiology, University of California
Irvine, California, USA

CONTRIBUTING AUTHORS

Nima Alipour DO MBA
Research Specialist
Department of Cardiology
University of California
Irvine, California, USA

Maciej Banach MD PhD FNLA FAHA FESC FASA FRSPH
Professor
Department of Hypertension
Chair of Nephrology and Hypertension
Medical University of Lodz
Zeromskiego, Lodz, Poland

Stanley Bassin EdD
Clinical Professor of Medicine
Heart Disease Prevention Program
Division of Cardiology
University of California Irvine School of Medicine
Irvine, California, USA

Nancy Boulos BS MPH
Heart Disease Prevention Program
Division of Cardiology
Department of Medicine
University of California
Irvine, California, USA

Richard J Glassock MD
Emeritus Professor
David Geffen School of Medicine at UCLA
Laguna Niguel, California, USA

Elliott M Groves MD MEng
Fellow of Cardiovascular Diseases
Department of Medicine, Division of Cardiology
University of California Irvine
Post-Doctoral Research Fellow
Edwards Life Sciences Center for Advanced Cardiovascular Technology
University of California-Irvine
Irvine, California, USA

Marta Michalska-Kasiczak MS PhD
Assistant Professor
Department of Hypertension
Chair of Nephrology and Hypertension, Medical University of Lodz
Zeromskiego, Lodz, Poland

Khurram Nasir MD MPH
Research Director
Center for Prevention and Wellness
Director, High-risk Cardiovascular Disease Clinic Baptist Health South Florida, Miami, Florida, USA
Assistant Professor, Johns Hopkins Ciccarone Center for Preventive Cardiology
Baltimore, Maryland, USA

Rohini J Patel MPH
Medical Student
Heart Disease Prevention Program
Division of Cardiology
University of California Irvine School of Medicine
Irvine, California, USA

Mohammad U Qazi MD
Cardiology Fellow
Department of Cardiovascular Medicine
University of California-Irvine
Irvine, California, USA

Paul D Rosenblit MD PhD FACE FNLA
Director
Diabetes/Lipid Management and Research Center
Orange County, California, USA
Clinical Professor
Division of Endocrinology, Diabetes, and Metabolism
Department of Medicine
Irvine, California, USA
Co-Director, Diabetes Outpatient Clinic, UCI Medical Center
Orange County, California, USA

Jacek Rysz MD PhD
Professor
Department of Nephrology Hypertension, and Family Medicine
Chair of Nephrology and Hypertension
Medical University of Lodz
Zeromskiego, Lodz, Poland

Sameer Shaharyar MD
Clinical Research Coordinator
Aventura Hospital and Medical Center
Aventura , Florida, USA

Geeta Sikand MA RDN
Associate Clinical Professor and Director of Nutrition
Heart Disease Prevention Program
Division of Cardiology
University of California Irvine School of Medicine
Irvine, California, USA

Haider J Warraich MD
Instructor in Medicine
Department of Medicine
Beth Israel Deaconess Medical Center
Harvard Medical School
Boston, Massachusetts, USA

Katherine Yu BS MD
Resident Physician
Department of Internal Medicine
University of California-Irvine
Irvine, California, USA

3
CHAPTER

Lifestyle Management in Diabetes

Rohini J Patel, Geeta Sikand, Stanley Bassin

ABSTRACT

This chapter will address the lifestyle recommendations for physical activity, obesity, and nutrition as a means of reducing incident type 2 diabetes as well as incident cardiovascular disease (CVD). Prospective cohort studies have indicated that both moderate-intensity level and high-intensity level physical activity provide beneficial risk reduction for both type 2 diabetes and CVD. Additionally, prospective cohort studies have indicated healthy diets through proper nutritional intake and lowered obesity decreases the risk for type 2 diabetes and CVD. Similarly, clinical trials have shown a similar result for modification of diet as a preferred method for disease risk reduction. Dietary modifications range from increasing specific nutrient intakes, changing dietary patterns, recommended dietary plans, to even an overall caloric limit. Overall, public health and medical professionals should promote the newly released guidelines by the American Heart Association and American College of Cardiology on physical activity levels as well as dietary recommendations to prevent type 2 diabetes and the development of CVD in diabetic individuals.

INTRODUCTION

Lifestyle modifications including a healthy eating pattern and physical activity are the key lifestyle components of diabetes management. They have been highly regarded as one of the first steps toward reducing an individual's risk for type 2 diabetes, which alone will increase the risk of developing many chronic diseases, including cardiovascular disease (CVD), chronic kidney disease, osteoporosis, depression, and certain cancers.[1,2] The lifestyle modifications of greatest importance toward reducing the global

burden of disease of diabetes, 19.6 million disability adjusted life years, are obesity, physical activity, and nutrition.[3,4] Similarly, these same lifestyle modifications can alleviate the global burden of CVD, 62.6 million disability adjusted life years.[3] Furthermore, a sedentary lifestyle has been attributed to many diseases and represents a significant portion of public health and clinical concerns. However, adherence to the suggested American Heart Association (AHA) physical activity guidelines has been met with little public success. For example, the suggested guideline by both the Centers for Disease Control and Prevention (CDC) and American College of Sports Medicine claims individuals should participate in 30 minutes of moderate level physical activity for a majority of the days in a week, of which only 39% of Americans follow.[1,5] Similarly, according to the National Health and Nutrition Examination Survey (NHANES) 1999–2000, the prevalence of overweight individuals was 64.5% and obese individuals was 30.5%, representing a body mass index (BMI) greater than or equal to 25 and greater than or equal to 30, respectively.[6] The prevalence of obesity continues to increase globally across gender, age, and race.[6]

The global problems associated with physical inactivity and obesity are in large part due to the advent of a sedentary lifestyle. As the idea of outdoor and physical activities is replaced with television viewing and video games, the overall health of the public is deteriorating, and thus attention has shifted from increasing physical activity alone to increasing activity and decreasing sedentary behavior.[7,8] Similarly, the introduction of highly processed foods, larger portions, and an overall lack of awareness on proper dietary health have contributed to the obesity epidemic.[9] As an interdependent disease, diabetes can be attributed to poor diet quality leading an individual to be either overweight or obese, which can be a result of poor physical activity and ultimately lead to CVD.[10]

Diabetes is a multifactorial chronic disease that itself increases one's risk for many diseases. However, the lifestyle components of diabetes can drastically reduce one's risk for further complications. Physical inactivity alone will increase an individual's risk for CVD, osteoporosis, obesity, depression, breast cancer, and colon cancer.[1] Obesity, in reference to CVD, will increase low-density lipoprotein (LDL) levels, cholesterol levels, triglyceride levels, blood pressure and blood glucose levels, and reduce high-density lipoprotein (HDL) levels.[1] According to a meta-analysis from 1977 to 1993, together, physical activity, dietary patterns, and obesity contribute to 300,000 deaths.[11]

The development of type 2 diabetes is a complicated process involving the interaction between an individual's genes and their environment or lifestyle choices.[12,13] Therefore, a targeted prevention should focus on lifestyle factors such as physical activity, obesity, and nutrition. In this chapter, we summarize

the current evidence on the benefits on physical activity, managing obesity, and the importance of nutrition on one's diet in hopes of preventing the onset of type 2 diabetes and ultimately the progression of diabetes to CVD.

PHYSICAL ACTIVITY AND THE DEVELOPMENT OF CARDIOVASCULAR DISEASE

Physical activity is a major component and a major determinant of total energy expenditure. Energy expenditure can be measured through the interplay between physical activity and diet.[14,15] By increasing an individual's energy expenditure through physical activity, it is believed that the obesity epidemic can be curbed.[14,15] However, energy expenditure is an uncommon unit of measure to the general public and is, therefore, represented as metabolic equivalents (METs), which correlate to body mass.[15] For example, in both weight-bearing and nonweight-bearing exercise, obese individuals have higher energy expenditure than their normal-weight counterparts.[16,17] The relationship between physical activity and energy expenditure is important for the overall determination of CVD risk in patients.

A similar study in Northern Ireland assessed the impact of discretionary activities on approximately 30 adults. Trends have indicated that the prevalence of sedentary lifestyles has increased both at home and at work.[18] Increased total energy expenditure from increased recreational activity is a lifestyle risk factor that can greatly decrease an individual's risk of coronary heart disease (CHD).[18] Discretionary activities are categorized as any activity that could be a household task, community activity, or cardiorespiratory activity.[18] Along with type of activity, the duration of discretionary activity and level of difficulty were also taken into account. Overall, the study concluded that the total energy expenditure values as a multiple of basal metabolic rate were 1.77 for females and 1.88 for males, thus indicating that increased discretionary activity can increase total energy expenditures, which will reduce an individual's risk for CHD and obesity.[18]

Physical activity is a primary lifestyle target because increased activity decreases the negative consequences of other CVD risk factors, such as hypertension, dyslipidemia, diabetes, and obesity.[19-21] The National Institutes of Health (NIH) Consensus Conference analyzed the importance of physical activity and cardiovascular health through a meta-analysis. The analysis concluded that more than one-third of United States (US) adults are not engaging in enough physical activity to provide them with health benefits.[21] There are several reasons for the gap between expected and observed physical activity patterns. These include poor programs to educate patients on the necessity of physical activity, a lack of community support through policy changes at schools and worksites, and a need for a national

campaign involving various health organizations.[21] Furthermore, patients do not perceive a benefit to physical activity and are, therefore, less likely to engage in this behavior. Overall, in order to empower individuals to increase their levels of physical activity, progress must be made on diet and nutrition, as well as the importance of reducing the prevalence of obesity.

OBESITY AND DIET IN RELATION TO CARDIOVASCULAR DISEASE

According to the World Health Organization's (WHO) definition of weight, an individual is considered overweight if their BMI is greater than or equal to 25 and obese if their BMI is greater than or equal to 30.[22] Furthermore, the Iowa Women's Health Study found that obesity as determined from BMI and waist-to-hip ratio (WHR) provides more information than obesity from BMI alone.[23] However, the most common definition of obesity is in reference to BMI, and a cross-sectional study using the NHANES data determined that the prevalence of obesity in both children and adults is increasing.[22] This is concerning because dietary factors and nutritional value in foods have contributed to a significant portion of the obesity epidemic. Furthermore, the dangers of obesity are often misunderstood, as it is a poorly recognized risk factor for diabetes and CVD.[9,24]

Additionally, the prevalence of obesity continues to greatly vary across gender, age, and race. For example, the prevalence of obesity in non-Hispanic Black women aged 40–59 years is 50.6% compared to only 22.9% of non-Hispanic White men aged 20–39 years.[22] Similarly, the prevalence of being overweight in non-Hispanic White girls was less than the prevalence in non-Hispanic Black and Mexican-American girls.[22]

The quality of an individual's diet is one of the leading indicators of overweight or obesity. Diet, however, can impact much more than BMI alone, and is, therefore, an important tool for cardiovascular health. Currently, the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7) attributes obesity, increased sodium consumption, and increased alcohol consumption to hypertension.[25] However, the Dietary Approaches to Stop Hypertension (DASH) clinical trial aims to determine which other dietary factors can influence blood pressure. Clinical trials with vegetarian diets have shown that high levels of fiber and potassium and magnesium minerals coupled with a reduced fat content can lower blood pressure.[26,27] Observational studies have shown a similar relationship with a decrease in blood pressure associated with increased intake of magnesium, potassium, calcium, and fiber.[28-32]

The DASH diet consisted of individuals in one of three groups: (i) control, (ii) fruits and vegetables and (iii) combination.[33] The control represented the

average consumption of Americans for macronutrients and fiber along with potassium, magnesium, and calcium levels closer to the 25th percentile.[33] The fruits and vegetables diet was similar to the control diet except that an emphasis was placed on fruit and vegetable consumption as well as potassium and magnesium levels closer to the 75th percentile of consumption.[33] Finally, the combination diet was also rich in fruits and vegetables, but contained low-fat dairy foods as well as decreased the amount of total fat, saturated fat, and cholesterol.[33] The results indicated that across all three diets, even after stratification by sex, minority status, and hypertension, the combination diet decreased systolic blood pressure by an average of 5.5 mmHg and diastolic by 3.0 mmHg compared to the control group. Smaller decreases were seen comparing the combination diet to the fruit and vegetable diet and fruit and vegetable to control (Figure 1).[33] Overall, the DASH trial proved the importance of dietary composition to reduce hypertension, a major risk factor for CVD and diabetes.

The Mediterranean diet has gained attention as a proven way to reduce weight gain, reduce the incidence of type 2 diabetes, and reduce CVD risk.[34] The Mediterranean diet is composed of a high consumption of fruits and vegetables and the use of olive oil as the primary source of fat. In a clinical trial, individuals who were randomized to the treatment group of Mediterranean diet, stress management training, exercise, group support, and smoking cessation found greater improvements in levels

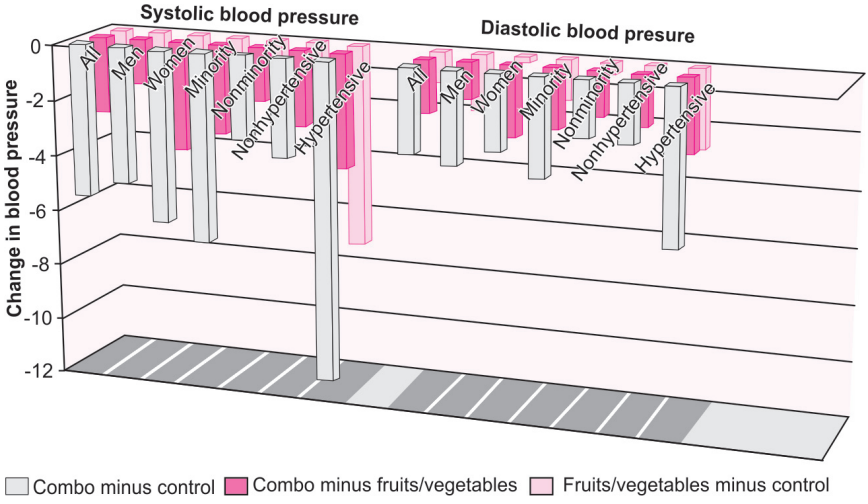

Figure 1 Change in systolic and diastolic blood pressure from the Dietary Approaches to Stop Hypertension subgroups subdivided into sex, minority status, and hypertensive status.

of glycosylated hemoglobin (HbA1c), LDL, and BMI compared to the control group.[34] The exact mechanism by which the Mediterranean diet works is unknown; however, a simplistic model has been created. The Mediterranean diet increases intake of foods that increase dietary fiber and increase unsaturated fat. Dietary fiber increases satiety, while unsaturated fat increases fat oxidation, and both work together to lower energy density leading to overall weight control.[34]

Finally, a randomized trial was conducted to ascertain the similarities and differences between several well-known diets and their effect on cardiovascular health. Four diets including the (i) Atkins, (ii) Ornish, (iii) Weight Watchers, and (iv) Zone were analyzed for their total weight loss and CVD risk factor reduction. All four indicated weight loss between 2.1 kg and 3.3 kg and an overall decrease in the total divided by HDL cholesterol (HDL-C) ratio, C-reactive protein and insulin levels.[35] Therefore, the importance of dietary interventions to reduce the burden of obesity and several CVD risk factors is becoming increasingly important.

PHYSICAL ACTIVITY AND THE DEVELOPMENT OF DIABETES

Physical activity is an important lifestyle risk factor to manage because it can lead to several chronic conditions including type 2 diabetes and CVD. In individuals with normal or impaired (prediabetes) glucose regulation, there is a potential joint association with sedentary lifestyles and obesity.[5,36,37] In a prospective cohort study conducted in Finland with over 4,000 participants, an inverse association between risk of type 2 diabetes and physical activity was found.[37] This relationship was observed across the board in the following groups: obese and impaired glucose regulation, either obese or impaired glucose regulation, or normal BMI and glucose regulated.[37] The results indicated hazard ratios for the risk of developing diabetes from light, moderate, and high physical activity as 1.00, 0.85, and 0.43, respectively.[37] Therefore, this proves that the more physical activity an individual engages in, the more protected he/she is from developing diabetes and CVD.

Another prospective cohort study, the Honolulu Heart Program followed 6,800 Japanese-Americans and measured their physical activity. Physical activity was grouped as basal, sedentary, moderate, and heavy, and the numbers of hours per group were also recorded. The cumulative incidence of diabetes per 1,000 was calculated per physical activity quintile and for all men, the highest quintile of physical activity had an incidence of diabetes of 34.3 per 1,000 while the lowest quintile had a diabetes incidence of 73.8 per 1,000 (Figure 2).[38]

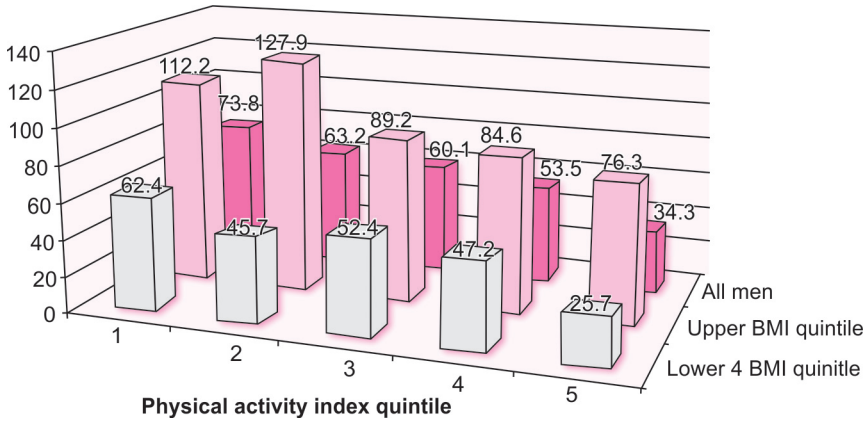

Figure 2 Age-adjusted 6-year cumulative incidence of diabetes by physical activity index quintile [quintile 1 (<29.1), quintile 2 (29.1–30.7), quintile 3 (30.8–33.2), quintile 4 (33.3–36.2), and quintile 5 (>36.2)]. The index is determined by multiplying the number of hours spent in each of five different activity levels by a weighting factor from oxygen consumption and then summing the values from each activity level

Similarly, the Nurses' Health Study, a prospective cohort study assessed the difference in moderate-intensity physical activity on diabetes incidence versus vigorous activity. Relative risks across quintiles of physical activity for developing diabetes were 1.0, 0.77, 0.75, 0.62, and 0.54 after adjusting for age, smoking, alcohol, history of hypertension, and high cholesterol.[37] Overall, the study proved that increased physical activity decreases an individual's risk of developing diabetes and even moderate level activity has a protective effect on diabetes and incident CVD.

Finally, the combined relationship between physical activity and BMI in relation to incident type 2 diabetes was studied in approximately 38,000 women from the Women's Health Study. This study proved that BMI is a greater predictor of diabetes than physical activity.[39] The hazard ratio for overweight was 3.22 compared to 9.09 for obese and lowest physical activity quartile 0.91.[39] This relationship between obesity and diabetes must be further explored, as weight is a major risk factor for diabetes and CVD.

OBESITY AND DIET IN RELATION TO DIABETES

Obesity and dietary patterns have long been recognized as risk factors for diabetes and contribute a significant burden to years of life lost.[40-42] Several clinical trials and cohort studies including the Finnish Diabetes Prevention Study, US Diabetes Prevention Program (DPP), Da Qing Study, Pima Indian Study, Women's Health Study, and the study of US Male Physicians have

proved that a decreased risk of diabetes is associated with a normal BMI and physical activity.[43] Additionally, in terms of dietary factors, it is believed that a high intake of saturated fats will increase one's risk of diabetes, likely through increasing abdominal adiposity, while nonstarch polysaccharides lower one's risk for diabetes and CVD.[43] Similarly, protective effects can be seen with increased intake of omega-3 fatty acids and low glycemic index (GI) foods and increased risk with a higher consumption of total fat intake and trans fatty acids.[43]

Dietary fat intake and its association with type 2 diabetes has been studied prospectively in several studies of women free at baseline from diabetes, cancer, and CVD.[44-46] Detailed dietary information from one study was collected at baseline and over the course of a 14-year follow-up, 2,507 incident cases of diabetes were recorded.[46] The study found that consumption of saturated, monounsaturated or polyunsaturated fatty acids did not increase the risk of diabetes, but a 2% increase in energy from *trans* fatty acids increased the relative risk of developing diabetes to 1.39.[46] Overall, the data suggests that diet is an important indicator of diabetes and that replacing 2% of energy from trans fatty acids with polyunsaturated fat would lower the risk of diabetes by 40%.[46]

Current data promotes the idea of a low-fat and high-carbohydrate diet in order to prevent diabetes and CVD.[47,48] However, nonhomogeneous molecules such as fats and carbohydrates must be further delineated in order to develop the best dietary patterns. For example, in order to prevent insulin sensitivity, higher intake of long-chain fatty acids is suggested.[49] Additionally, increased intake of whole grains and fiber decreases an individual's risk for diabetes.[49] Nonetheless, the association between diet and obesity as a major risk factor for diabetes must be further evaluated.

The Nurses' Health Study assessed the relationship between body fat distribution and risk of developing diabetes, specifically overall and central obesity.[50] The relative risk of developing diabetes for those in the 90th percentile of BMI (BMI = 29.9) compared to the 10th percentile (BMI = 20.1) was 11.2.[50] Similarly, comparing the 90th percentile of WHR (WHR = 0.86) to 10th percentile (WHR = 0.70), the relative risk was 3.1, and for 90th percentile of waist circumference (92 cm) to 10th percentile (67 cm), the relative risk was 5.1.[50] Therefore, BMI, WHR, and waist circumference are important tools for early diabetes risk assessment and that obesity in all forms, BMI or central adiposity is an indicator of diabetes risk and CVD risk.[51,52]

Finally, the Seattle Japanese-American Community Diabetes Study looked at the relationship between visceral adiposity and incident CHD. It was determined that intra-abdominal fat was responsible for the effects of low HDL-C and high triglycerides and their associated risk for CHD.[53] Intra-abdominal fat was significantly higher in those who developed CHD

TABLE 1: Baseline adipose, metabolic, and blood pressure variables by incident Coronary heart disease status

Baseline variable	Incident CHD status	
	CHD absent	CHD present
Number of patients	125	50
Body mass index (kg/m^2)	25.4 ± 0.3	25.8 ± 0.4
Abdomen subcutaneous fat (cm^2)	129.3 ± 5.7	146.0 ± 7.9
Intra-abdominal fat (cm^2)	108.9 ± 4.7	133.0 ± 7.6
Abdominal circumference (cm)	88.0 ± 0.7	90.2 ± 1.0
Fasting glucose (mmol/L)	6.41 ± 0.17	8.33 ± 0.54
Cholesterol (mmol/L)	6.05 ± 0.10	6.22 ± 0.17
LDL cholesterol (mmol/L)	3.94 ± 0.09	3.98 ± 0.15
HDL cholesterol (mmol/L)	1.36 ± 0.03	1.21 ± 0.04
Triglycerides (mmol/L)	1.60 ± 0.07	2.07 ± 0.19
Systolic blood pressure (mmHg)	135.1 ± 1.5	145.1 ± 2.8
Diastolic blood pressure (mmHg)	78.4 ± 0.8	84.8 ± 1.4

Note: Mean ± standard error of mean.

CHD, coronary heart disease; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

(133.0 ± 7.6) compared to those who did not develop CHD (108.9 ± 4.7) (Table 1).[53] Additionally, the San Luis Valley Diabetes Study also determined that fat consumption can predict the progression of impaired glucose tolerance to noninsulin-dependent diabetes, even after controlling for BMI, obesity, and other glucose metabolism markers.[54,55] Overall, the importance of regulating obesity and dietary intake are important to prevent diabetes and CVD development.

NUTRITIONAL FACTORS IN RELATION TO DIABETES AND CARDIOVASCULAR DISEASE

Caffeine

Dietary nutritional factors are an important area of lifestyle modification as they directly relate to obesity, a major factor in diabetes and CVD risk. A systematic review of coffee consumption and risk of type 2 diabetes determined that habitual coffee intake lowers an individual's risk for diabetes, but that the exact mechanism is poorly understood.[56] The suggested mechanism involves components in coffee, other than caffeine, which could be beneficial for glucose metabolism, but have yet to be formalized.[57] Additionally, the negative consequences of coffee on other aspects of

health must be studied before increased coffee consumption can become a preventative recommendation.

Vitamin D and Calcium

Other nutritional factors that have been studied in reference to diabetes and cardiovascular risk are vitamin D and calcium. Over the course of a 20-year follow-up using the Nurses' Health Study, approximately 84,000 women were assessed at baseline and every 2–4 years on their vitamin D and calcium intake.[58] The results indicated that by increasing calcium consumption from less than 600 mg to greater than 1,200 mg and increasing vitamin D consumption from 400 IU to greater than 800 IU, the relative risk of diabetes decreases by 33%.[58] Therefore, a call for increased nutritional factors, such as dietary calcium and vitamin D, should be recommended to lower one's risk for both diabetes and CVD.[59]

Magnesium and Nuts

Magnesium intake in relation to diabetes risk has also been evaluated in a cohort study of approximately 120,000 men and women over the course of a 15-year follow-up. Data indicated that the relative risk of diabetes in men when comparing the highest quintile of magnesium intake to lowest quintile was 0.67 and 0.66 in women.[60] This confirms the dietary recommendation to increase magnesium intake through whole grains, nuts and green leafy vegetables.[59,60] Furthermore, increased consumption of nuts and peanut butter alone has been shown to improve glucose homeostasis and lower an individual's risk for diabetes.[61]

Glycemic Index

The GI is a tool used to assess an individual's glycemic content and is calculated by dividing the glycemic load by the carbohydrate intake in 1 day.[62] Glycemic load is computed by summing the carbohydrate content per servings of food multiplied by the average number of servings of that specific food item.[62] An analysis of data from the Atherosclerosis Risk in Communities (ARIC) study, however, indicated no relationship between GI and dietary fiber intake with diabetes risk.[63] Nonetheless, a prospective study analyzing potato intake, a high glycemic content carbohydrate, showed increased insulin resistance and risk for diabetes.[64] Furthermore, the relative risk when one serving of potatoes was in place of one serving of whole grains was 1.30 and overall the risk was greatest in obese women.[64] Therefore, it has been determined that diets with a high glycemic load increase an individual's risk for diabetes.[65,66]

Sugar-sweetened Beverages

Finally, one of the most hidden dietary factors leading to an increase in diabetes and cardiovascular risk is sugar-sweetened beverages. Sugar-sweetened beverages, often categorized as soft drinks, fruit punch, juice, lemonade, and energy drinks contain absorbable sugars, which cause weight gain and can lead to diabetes and CVD.[67,68] From the Nurses' Health Study II, it was determined that women who consume more than one sugar-sweetened beverage a day had a relative risk of 1.83 for diabetes compared to women who consumed less than one sugar-sweetened beverage a day.[68] Additionally, women whose intake rose over the course of the study gained an average of 4.69 kg of body weight and their BMI increased an average of 1.72.[68] Overall, sugar-sweetened beverage consumption is related to increased weight gain, obesity, diabetes, and CVD as the sugar content is readily absorbable in the body.

LIFESTYLE RECOMMENDATIONS AND FUTURE RESEARCH

Overall, the most important aspects of lifestyle modification include increasing physical activity levels and altering dietary patterns to decrease obesity.[69] The Diabetes Prevention Research Group conducted a randomized clinical trial on individuals at high risk for developing diabetes and whether a lifestyle intervention or administration of metformin produced lower risk. Overall, the lifestyle intervention, which consisted of following a low-fat diet and performing 150 minutes per week of moderate level physical activity, had a 58% lower incidence of diabetes compared to the placebo group and 31% lower incidence of diabetes compared to the metformin group.[69] Additionally, further research on the relationship between diet, physical activity, and obesity has indicated a need for information on environmental factors, maintenance of optimal activities, specific etiology and behavior change.[70] Environmental factors have been studied through an analysis of Pima Indians living in Mexico eating a traditional diet compared with Pima Indians living in Arizona consuming a Western diet. The results have indicated that regardless of a similar genetic background, the Pima Indians in Mexico had lower rates of obesity and diabetes.[71] Behavior change is also an important future area of interest because of complicated disease etiologies, comorbidities and the interaction between lifestyle changes and medication regiments.[70] Overall, the need for lifestyle recommendations remains critical and led to the Action for Health in Diabetes (Look AHEAD) trial.

The Look AHEAD was a clinical trial in 16 locations around the United States that assessed the impact of an intense lifestyle intervention in diabetic

TABLE 2: Primary outcome (death from cardiovascular cases, nonfatal myocardial infarction, nonfatal stroke or hospitalization for angina) from Look AHEAD in subgroups

Subgroup	Control	Intervention	Hazard ratio
Overall	418 (1.92)	403 (1.83)	0.95 (0.83–1.09)
No cardiovascular disease at baseline	274 (1.42)	240 (1.23)	0.86 (0.72–1.02)
Yes cardiovascular disease at baseline	144 (5.92)	163 (6.56)	1.13 (0.90–1.42)
Male	245 (2.94)	232 (2.72)	0.93 (0.78–1.11)
Female	173 (1.29)	171 (1.26)	0.97 (0.79–1.20)
White	303 (2.19)	286 (2.06)	0.94 (0.80–1.11)
Black	46 (1.32)	63 (1.82)	1.34 (0.91–1.96)
Hispanic	43 (1.54)	31 (1.06)	0.66 (0.41–1.05)
Native American	13 (1.18)	10 (0.86)	0.74 (0.31–1.76)
Asian or Pacific Islander	3 (1.67)	1 (0.38)	0.71 (0.06–8.28)
Other	10 (2.35)	12 (2.96)	1.15 (0.45–2.89)

AHEAD, Action for Health in Diabetes.

Note: Control and intervention show number of events (rate/100 person-year). Hazard ratios (HR) present as HR (95% confidence interval).

patients at risk for cardiovascular events.[72] The lifestyle intervention group consisted of a caloric goal between 1,200 kcal and 1,800 kcal per day with less than 30% from fat and greater than 15% from protein, meal replacement products, and a minimum of 175 minutes of moderate level physical activity per week.[72] This was compared to the control group, which received education and support. However, the Look AHEAD trial was suspended early due to the finding that weight loss did not reduce the rate of CVD in diabetic patients.[72] This was likely due to the fact that over time the differences between control and intervention groups for weight, physical fitness, waist circumference and glycated hemoglobin all became closer together (Table 2). Therefore, more research must be done on health behavior maintenance programs, specifically related to lifestyle interventions and dose-response models in order to decrease the risk of CVD in individuals with type 2 diabetes.[73]

Additionally, the Executive Summary for the Standards of Medical Care in Diabetes has listed several recommendations, both to prevent type 2 diabetes and manage diabetes. To prevent type 2 diabetes, the American Diabetes Association (ADA) from the DPP recommends 7% weight loss in body weight, physical activity for 150 minutes per week including resistance training, reduced dietary fat intake, increased consumption of whole grains, 14 g of fiber per 1,000 kcal, and to overall limit their intake of sugar-sweetened

beverages.[12,13] Furthermore, to manage diabetes, the ADA and DPP recommends mixing macronutrients such as carbohydrates, protein and fat, carbohydrate counting to achieve glycemic control, saturated fat intake less than 7% of total calories, and lowering intake of trans fat.[12,13]

Finally, the most recent lifestyle guidelines have come from the AHA and American College of Cardiology (ACC). Lifestyle management guidelines were broken up into LDL cholesterol (LDL-C) lowering benefits, blood pressure lowering benefits and physical activity. To lower an individual's LDL, dietary recommendations include increased intake of vegetables, fruits, whole grains, low-fat dairy, poultry, fish, legumes, nontropical vegetable oils and nuts, while decreasing consumption of sweets, sugar-sweetened beverages, and red meat.[74] Additionally, it is recommended to follow a dietary pattern such as the DASH diet, US Department of Agriculture (USDA) Food Pattern, or AHA diet in order to obtain 5–6% of calories from saturated fat and reduce the percent of calories from trans fats.[74] To lower blood pressure, the above dietary guidelines are suggested as well as lowered sodium intake of less than 2,400 mg of sodium per day or less than 1,500 mg/day or even less than 1,000 mg/day for greatest effect on blood pressure.[74] In terms of physical activity, guidelines of three to four times a week for an average of 40 minutes each time at a level of moderate- to vigorous-intensity physical activity are reccomended.[74] Finally, specific dietary guidelines were created for individuals who are overweight or obese due to their increased risk for diabetes and CVD. These guidelines include a total 1,200–1,500 kcal/day diet for women or 1,500–1,800 kcal/day diet for men, or a reduction of 500 kcal/day for women or 750 kcal/day for men, or an evidence-based diet that restricts specific foods such as high-carbohydrate foods, low-fiber foods or high-fat foods.[75] They also recommend comprehensive high-intensity lifestyle intervention consisting of at least 14 sessions in 6 months provided in individual or group sessions by a trained interventionist. Overall, these new guidelines for dietary and physical activity modifications must be adopted by public health officials and physicians so that individuals in the community are aware of the changes and can modify their individual lives to match the guidelines and reduce their risk for both diabetes and CVD.

CONCLUSION

Cardiovascular disease and diabetes are major contributors to the global burden of diseases and are the fastest growing public health and medical concerns. This chapter addressed the lifestyle modifications, both dietary and physical activity, to decrease both the incidence of diabetes as well as decrease the incidence of CVD. The AHA and ACC have recently released modified lifestyle guidelines based on critical reviews of the currently

available literature that specifically address dietary recommendations and physical activity levels for all individuals as well as separate guidelines for obese and overweight indivudals.[74,75] Increased physical activity, decreased obesity and increased nutrition have been attributed to decreasing the global burden of disease for both diabetes and CVD. Lifestyle modification techniques are important because they can prevent the onset of disease and prevent relying on pharmacological therapies for risk reduction. Therefore, public health and medical professionals must continue to advocate for lifestyle modifications in their patients at risk for developing type 2 diabetes and CVD by following the newly established AHA and ACC guidelines.

REFERENCES

1. Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. Circulation. 2006;114:82-96.
2. Global Strategy on Diet, Physical Activity and Health. World Health Organization. 2004:1-14.
3. Global Burden of Disease 2004 Update. World Health Organization. 2008:40-51.
4. Ramlo-Halsted BA, Edelman SV. The natural history of type 2 diabetes: practical points to consider in developing prevention and treatment strategies. Clinical Diabetes. 2000;18:80-4.
5. Thompson PD, Buchner D, Piña IL, Balady GJ, Williams MA, Marcus BH, et al. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: a statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention) and the Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity). Circulation. 2003;107:3109-16.
6. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA. 2002;288:1723-7.
7. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. JAMA. 2003;289:1785-91.
8. Crespo CJ, Smit E, Troiano RP, Bartlett SJ, Macera CA, Andersen RE. Television watching, energy intake, and obesity in US children: results from the third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc Med. 2001;155:360-5.
9. Grundy SM. Multifactorial causation of obesity: implications for prevention. Am J Clin Nutr. 1998;67: 563S-72S.
10. Helmrich SP, Ragland DR, Leung RW, Paffenbarger RS Jr. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. N Engl J Med. 1991;325:147-52.
11. McGinnis JM, Foege WH. Actual causes of death in the United States. JAMA. 1993;270:2207-12.
12. Diabetes Prevention Program (DPP) Research Group. The Diabetes Prevention Program (DPP): description of lifestyle intervention. Diabetes Care. 2002;25:2165-71.
13. American Diabetes Association. Standards of medical care in diabetes—2013. Diabetes Care. 2013:36 Suppl 1:S11-66.
14. Ravussin E, Lillioja S, Knowler WC, Christin L, Freymond D, Abbott WG, et al. Reduced rate of energy expenditure as a risk factor for body-weight gain. N Engl J Med. 1988;318:467-72.
15. Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS, Di Pietro L, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obes Rev. 2003;4:101-14.
16. Hulens M, Vansant G, Lysens R, Claessens AL, Muls E. Exercise capacity in lean versus obese women. Scand J Med Sci Sports. 2001;11:305-9.

17. Foster GD, Wadden TA, Kendrick ZV, Letizia KA, Lander DP, Conill AM. The energy cost of walking before and after significant weight loss. Med Sci Sports Exerc. 1995;27:888-94.
18. Livingstone MB, Strain JJ, Prentice AM, Coward WA, Nevin GB, Barker ME, et al. Potential contribution of leisure activity to the energy expenditure patterns of sedentary populations. Br J Nutr. 1991;65:145-55.
19. Santa-Clara H, Fernhall B, Baptista F, Mendes M, Sardinha LB. Effect of a one-year combined exercise training program on body composition in men with coronary artery disease. Metabolism. 2003;52: 1413-7.
20. Nakanishi N, Takatorige T, Suzuki K. Daily life activity and risk of developing cardiovascular risk factors. Diabetes Care. 2005;28:1500-2.
21. Physical activity and cardiovascular health. NIH Consensus Development Panel on Physical Activity and Cardiovascular Health. JAMA. 1996;276:241-6.
22. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. JAMA. 2004;291:2847-50.
23. Folsom AR, Kushi LH, Anderson KE, Mink PJ, Olson JE, Hong CP, et al. Associations of general and abdominal obesity with multiple health outcomes in older women: the Iowa Women's Health Study. Arch Intern Med. 2000;160:2117-28.
24. Lakka HM, Lakka TA, Tuomilehto J, Salonen JT. Abdominal obesity is associated with increased risk of acute coronary events in men. Eur Heart J. 2002;23:706-13.
25. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42:1206-52.
26. Rouse IL, Armstrong BK, Beilin LJ, Vandongen R. Blood-pressure-lowering effect of a vegetarian diet: controlled trial in normotensive subjects. Lancet. 1983;1(8314-5):5-10.
27. Margetts BM, Beilin LJ, Vandongen R, Armstrong BK. Vegetarian diet in mild hypertension: a randomised controlled trial. Br Med J (Clin Res Ed). 1986;293(6560):1468-71.
28. Ascherio A, Rimm EB, Giovannucci EL, Colditz GA, Rosner B, Willett WC, et al. A prospective study of nutritional factors and hypertension among US men. Circulation. 1992;86:1475-84.
29. Harlan WR, Hull AL, Schmouder RL, Landis JR, Thompson FE, Larkin FA. Blood pressure and nutrition in adults. The National Health and Nutrition Examination Survey. Am J Epidemiol. 1984;120:17-28.
30. Stamler J, Caggiula A, Grandits GA, Kjelsberg M, Cutler JA. Relationship to blood pressure of combinations of dietary macronutrients. Findings of the Multiple Risk Factor Intervention Trial (MRFIT). Circulation. 1996;94:2417-23.
31. Stamler J, Elliott P, Kesteloot H, Nichols R, Claeys G, Dyer AR, et al. Inverse relation of dietary protein markers with blood pressure. Findings for 10,020 men and women in the INTERSALT Study. INTERSALT Cooperative Research Group. International study of salt and blood pressure. Circulation. 1996;94:1629-34.
32. Obarzanek E, Velletri PA, Cutler JA. Dietary protein and blood pressure. JAMA. 1996;275:1598-603.
33. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336:1117-24.
34. Schröder H. Protective mechanisms of the Mediterranean diet in obesity and type 2 diabetes. J Nutr Biochem. 2007;18:149-60.
35. Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. JAMA. 2005;293:43-53.
36. Marcus BH, Williams DM, Dubbert PM, Sallis JF, King AC, Yancey AK, et al. Physical activity intervention studies: what we know and what we need to know: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism (Subcommittee on Physical Activity); Council on Cardiovascular Disease in the Young; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research. Circulation. 2006;114:2739-52.

37. Hu FB, Sigal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Willett WC, et al. Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. JAMA. 1999;282:1433-9.
38. Burchfiel CM, Sharp DS, Curb JD, Rodriguez BL, Hwang LJ, Marcus EB, et al. Physical activity and incidence of diabetes: the Honolulu Heart Program. Am J Epidemiol. 1995;141:360-8.
39. Weinstein AR, Sesso HD, Lee IM, Cook NR, Manson JE, Buring JE, et al. Relationship of physical activity vs body mass index with type 2 diabetes in women. JAMA. 2004;292:1188-94.
40. Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. Years of life lost due to obesity. JAMA. 2003; 289:187-93.
41. Narayan KM, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of BMI on lifetime risk for diabetes in the US. Diabetes Care. 2007;30:1562-6.
42. Montonen J, Knekt P, Härkänen T, Järvinen R, Heliövaara M, Aromaa A, et al. Dietary patterns and the incidence of type 2 diabetes. Am J Epidemiol. 2005;161:219-27.
43. Steyn NP, Mann J, Bennett PH, Temple N, Zimmet P, Tuomilehto J, et al. Diet, nutrition and the prevention of type 2 diabetes. Public Health Nutr. 2004;7(1A):147-65.
44. Harding AH, Day NE, Khaw KT, Bingham S, Luben R, Welsh A, et al. Dietary fat and the risk of clinical type 2 diabetes: the European prospective investigation of Cancer-Norfolk study. Am J Epidemiol. 2004;159:73-82.
45. Meyer KA, Kushi LH, Jacobs DR Jr, Folsom AR. Dietary fat and incidence of type 2 diabetes in older Iowa women. Diabetes Care. 2001;24:1528-35.
46. Salmerón J, Hu FB, Manson JE, Stampfer MJ, Colditz GA, Rimm EB, et al. Dietary fat intake and risk of type 2 diabetes in women. Am J Clin Nutr. 2001;73:1019-26.
47. National Research Council (US) Committee on Diet and Health. Diet and Health: Implications for Reducing Chronic Disease Risk. Washington, DC: National Academies Press; 1989.
48. US Department of Agriculture—US Department of Health and Human Services. Nutrition and Your Health: Dietary Guidelines for Americans. Homes and Garden Bulletin No. 232. Washington, DC: US Government Printing Office; 2000.
49. Hu FB, van Dam RM, Liu S. Diet and risk of type II diabetes: the role of types of fat and carbohydrate. Diabetologia. 2001;44:805-17.
50. Carey VJ, Walters EE, Colditz GA, Solomon CG, Willett WC, Rosner BA, et al. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. The Nurses' Health Study. Am J Epidemiol. 1997;145:614-9.
51. Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. Am J Clin Nutr. 2005;81:555-63.
52. Koh-Banerjee P, Wang Y, Hu FB, Spiegelman D, Willett WC, Rimm EB. Changes in body weight and body fat distribution as risk factors for clinical diabetes in US men. Am J Epidemiol. 2004;159:1150-9.
53. Fujimoto WY, Bergstrom RW, Boyko EJ, Chen KW, Leonetti DL, Newell-Morris L, et al. Visceral adiposity and incident coronary heart disease in Japanese-American men. The 10-year follow-up results of the Seattle Japanese-American Community Diabetes Study. Diabetes Care. 1999;22:1808-12.
54. Marshall JA, Bessesen DH, Hamman RF. High saturated fat and low starch and fibre are associated with hyperinsulinaemia in a non-diabetic population: the San Luis Valley Diabetes Study. Diabetologia. 1997;40:430-8.
55. Marshall JA, Hoag S, Shetterly S, Hamman RF. Dietary fat predicts conversion from impaired glucose tolerance to NIDDM. The San Luis Valley Diabetes Study. Diabetes Care. 1994;17:50-6.
56. van Dam RM, Hu FB. Coffee consumption and risk of type 2 diabetes: a systematic review. JAMA. 2005;294:97-104.
57. van Dam RM, Feskens EJ. Coffee consumption and risk of type 2 diabetes mellitus. Lancet. 2002;360:1477-8.
58. Pittas AG, Dawson-Hughes B, Li T, van Dam RM, Willett WC, Manson JE, et al. Vitamin D and calcium intake in relation to type 2 diabetes in women. Diabetes Care. 2006;29:650-6.

59. Colditz GA, Manson JE, Stampfer MJ, Rosner B, Willett WC, Speizer FE. Diet and risk of clinical diabetes in women. Am J Clin Nutr. 1992;55:1018-23.
60. Lopez-Ridaura R, Willett WC, Rimm EB, Liu S, Stampger MJ, Manson JE, et al. Magnesium intake and risk of type 2 diabetes in men and women. Diabetes Care. 2004;27:134-40.
61. Jiang R, Manson JE, Stampfer MJ, Liu S, Willett WC, Hu FB. Nut and peanut butter consumption and risk of type 2 diabetes in women. JAMA. 2002;288:2554-60.
62. Wolever TM, Nguyen PM, Chiasson JL, Hunt JA, Josse RG, Palmason C, et al. Determinants of diet glycemic index calculated retrospectively from diet records of 342 individuals with non-insulin-dependent diabetes mellitus. Am J Clin Nutr. 1994;59:1265-9.
63. Stevens J, Ahn K, Juhaeri, Houston D, Steffan L, Couper D. Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults: the ARIC study. Diabetes Care. 2002;25:1715-21.
64. Halton TL, Willett WC, Liu S, Manson JE, Stampfer MJ, Hu FB. Potato and french fry consumption and risk of type 2 diabetes in women. Am J Clin Nutr. 2006;83:284-90.
65. Schulze MB, Liu S, Rimm EB, Manson JE, Willett WC, Hu FB. Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger and middle-aged women. Am J Clin Nutr. 2004;80:348-56.
66. Salmerón J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, et al. Dietary fiber, glycemic load, and risk of NIDDM in men. Diabetes Care. 1997;20:545-50.
67. Popkin BM, Nielsen SJ. The sweetening of the world's diet. Obes Res. 2003;11:1325-32.
68. Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, et al. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA. 2004;292:927-34.
69. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002;346:393-403.
70. Wing RR, Goldstein MG, Acton KJ, Birch LL, Jakicic JM, Sallis JF, et al. Behavioral science research in diabetes: lifestyle changes related to obesity, eating behavior, and physical activity. Diabetes Care. 2001;24:117-23.
71. Ravussin E, Valencia ME, Esparza J, Bennett PH, Schulz LO. Effects of a traditional lifestyle on obesity in Pima Indians. Diabetes Care. 1994;17:1067-74.
72. Wing RR, Bolin P, Brancati FL, Bray GA, Clark JM, Coday M, et al. Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. N Engl J Med. 2013;369:145-54.
73. Ross R, Janssen I. Physical activity, total and regional obesity: dose-response considerations. Med Sci Sports Exerc. 2001;33:S521-7.
74. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Miller NH, Hubbard VS, et al. 2013 AHA/ACC guidelines on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63(25 Pt B): 2960-84.
75. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. Circulation. 2014;129(25 Suppl 2):S102-38.