Nutrition, Physical Activity, Weight Management, and Health

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Abstract

Non-communicable chronic diseases, such as metabolic syndrome, cardiovascular dysfunction, type 2 diabetes, and obesity, make up a large portion of total world-wide mortality, and are becoming more prevalent in developing countries. These diseases have taken on a larger importance, as fertility rates in many developing countries are still rising and contributing to the overall planetary population. Chronic diseases are typically due to poor dietary habits, physical inactivity, and subsequent unhealthy body composition. However, achieving a healthy weight and then sustaining weight loss can reduce obesity-related disorders and improve the risk profile for chronic disease. Weight control through an improved diet style and higher levels of caloric expenditure can also improve the quality of life for people with diabetes, HIV/AIDS, and mental health, although the specific recommendations for the disease may vary slightly. These benefits will last for a long time if the modifications are sustained. Understanding the effects that various diets and physical activity modes and patterns have on healthy weight maintenance will ultimately prove beneficial for people who are otherwise at risk for a variety of chronic diseases.

Key words: Obesity, chronic disease, diet, exercise.

Título: Nutrición, actividad física, control de peso y salud.

Resumen

Enfermedades crónicas no transmisibles, tales como síndrome metabólico, disfunción cardiovascular, diabetes tipo 2 y obesidad, componen una gran proporción de la mortalidad mundial y se están volviendo más prevalentes en países en vía de desarrollo. Estas enfermedades han cobrado importancia debido a que la rata de fertilidad en muchos países subdesarrollados continúa aumentando, contribuyendo a la población mundial. Las enfermedades crónicas se deben generalmente a la presencia de hábitos alimenticios deficientes e inactividad física, con una composición corporal poco saludable como consecuencia. Sin embargo, alcanzar un peso saludable y después

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sostener la pérdida de peso puede reducir los trastornos relacionados con la obesidad y mejorar el perfil de riesgo para enfermedad crónica. El control de peso a través de un estilo dietario mejorado y mayores niveles de gasto calórico también puede mejorar la calidad de vida de personas con diabetes, VIH/sida y enfermedad mental, aunque las recomendaciones específicas para cada trastorno pueden variar levemente. Estos beneficios serán duraderos si las modificaciones se mantienen. Comprender los efectos que tienen diversas dietas y patrones de actividad física sobre la manutención de un peso saludable beneficiará a personas que de otro modo estarían en riesgo de sufrir una variedad de enfermedades crónicas.

Palabras clave: obesidad, enfermedad crónica, dieta, ejercicio.

Introduction

At the beginning of this century, non-communicable chronic diseases contributed approximately 60% of the total deaths in the world and about 46% of the global burden of disease (1). About half of these chronic disease deaths are attributable to cardiovascular disease (CVD), with many being related to type 2 diabetes or obesity. The problem of non-infectious chronic disease is not limited to the developed regions of the world, but is becoming a major problem in developing countries as well (2). Thus, the labeling of non-communicable diseases by previous generations as "diseases of affluence" no longer appears applicable, as these diseases continue to emerge both in poorer countries and in the poorer population groups of wealthier nations. This shift in disease pattern is taking place at an accelerating rate and is occurring faster in developing countries than it did in industrialized regions a century ago (3). Because these chronic diseases for the most part are related to inadequate physical inactivity and poor weight management, we shall focus on these issues in the present article. It should be kept in mind, however, that cigarette smoking and alcohol abuse also contribute to excess mortality from non-communicable chronic diseases (4).

Body weight, diet, and level of physical activity are important determinants of chronic illness, morbidity, and quality of life. The risks associated with many co-morbid conditions may be reduced with modest weight loss. Clinical studies suggest that minimal, sustained weight loss of 5% to 10% can reduce or eliminate obesity-related disorders (5). Weight control methods often produce short-term success, but sustained weight maintenance is difficult to reach (6-7). Weight cycling and relapse of body weight are common features after a weight loss intervention. The maintenance of treatment-induced weight loss thus remains a significant challenge in the management of obesity (8).

Nutritional therapy emphasizing mostly low-fat, whole plant foods

has been successfully utilized to achieve and maintain weight control, and can also be beneficial for normal blood glucose levels, hypertension, hyperlipidemia, dyslipidemia, cardiovascular disease risk, and mental status (9-12). These benefits have been found to last for years if the diet style is maintained (13-14).

Nutrition

A large amount of research finds that nutrition is one of the most critical factors for health. The accumulation of all research to this point, including animal, laboratory, clinical, and epidemiological findings, demonstrates compelling evidence for the link between nutritional deficiencies with chronic disease (15-16). The debate rages regarding what is considered the optimal diet style for prevention and/or reversal of various chronic diseases and conditions. Particularly for weight loss, high-protein, low-carbohydrate, Atkins-Style diets have been enormously popular in the mass media. Understanding beneficial long-term eating patterns is critical to minimizing the risk of unhealthy weight gain, given that the findings for the benefit on CVD death of weight reduction alone are unclear (17-18). Recent findings examining the benefits of a very low carbohydrate diet (19) do not support the belief that this style of diet is any better than standard diets for weight loss and CVD risk, despite other evidence to the contrary (20-21). The enormous interest in diets promoting high protein and/or low carbohydrate intake is occurring, while some evidence suggests that eating a diet high in processed sugars has followed the similar increasing curves of obesity and type 2 diabetes (22). Several studies have found better results in weight loss when comparing the effect of a free-living, very low carbohydrate diet to a low-fat, high-carbohydrate diets over a period of 6 months (20-21,23-27).

Adequate intake of fruits, vegetables, and fiber-rich foods is known to be beneficial for health and appears to have protective effects for such diseases as some cancers (15,28-29). However, in some parts of Latin America, poor access to clean water may influence the appropriateness of food choices recommended on the basis of research in developed countries. In addition, some controversies persist regarding the definitive nature of causal links among nutrition, health, and disease, the degree of benefits that can be expected with change, and optimal quantitative advice (30-31). The exact role of dietary factors and nutritional risk in disease is still being researched, but the evidence is currently sufficient to warrant a concerted research effort in promoting healthy eating behavior. The

potential public health benefit from improved eating patterns, coupled with the low risk of adopting guidelines for healthy eating, provides a stronger foundation than ever before for efforts to understand and encourage good nutrition among the general population, patients, and persons at high risk for disease (28,32).

Physical Activity

Running parallel to the rising epidemic of overweight and obesity is the increasing rate of physical inactivity. Physical inactivity is widely recognized as a major threat to public health (2). Data from São Paulo, Brazil indicate that 70-80% of the population are remarkably inactive (33). A sedentary lifestyle combined with poor nutrition accounts for an estimated 16% of the actual causes of death and approximately 24.4 billion dollars per year in health care expenditures in the United States alone (4,34). In contrast, a physically active lifestyle has been associated with health benefits that include improved control of hypertension, diabetes, obesity, hyperlipidemia, and reduced overall morbidity and mortality (35-38). An objective of Healthy People 2010 is that adults exercise for at least 30 minutes of moderate physical activity for most, if not all, days of the week (39), but more than 60% of people do not achieve this amount and are inactive by definition (38).

Several studies have shown that people who are more physically active are less likely to gain weight over time than those who are not (40-43). Others found that most people are gaining weight due to consuming less than 100 excess calories/day (44). Therefore, increasing physical activity by 100 calories/day could theoretically prevent weight gain in most people, which averages out to 2,000 additional steps each day (44). Although this is a possible approach to preventing weight gain, the amount of physical activity that would be required for substantial weight loss is not feasible for many people. Furthermore, the National Weight Control Registry (NWCR), a database of almost 5,000 successful weight loss maintainers, shows that 90% of their participants report losing weight with both food restriction and physical activity (45).

Subjects in the NWCR who have succeeded in long-term weight loss maintenance report expending 2,800 calories/week in physical activity (45). More than 90% are maintaining their weight loss with high levels of regular physical activity. The amount of physical activity reported by the NWCR participants is positively correlated with the amount of weight they are maintaining. A decrease in physical activity in this group has been shown

to be a predictor of weight gain over time (46). Another study found that obese subjects who had previously lost weight and engaged in at least 200 minutes/week of physical activity were less likely to regain the lost weight than those participants who engaged in not as much physical activity (47). Some argue that using inexpensive, electronic pedometers and providing physical activity goals in steps per day is effective in increasing physical activity over the short run (48). However, combining dietary modification with increased physical activity will produce the best results.

Obesity

During the past twenty years, the developed world and cities within developing countries have fallen to a pervasive health epidemic: obesity. Approximately 1.7 billion people are now obese, its prevalence is rising in most countries, and dramatically increasing among children and adolescents (49). The incidence of overweight (defined as a body mass index [BMI] between 25 and 30 kg/m^2) and obesity (defined as a BMI > 30 kg/m^2) among adults is at an all time high and continuing to rise (50,51). More people are now overweight or obese than people who smoke, live in poverty, or drink heavily. Overweight/obesity has become such an epidemic that it is now listed as one of the leading health indicators in Healthy People 2010 (39) and obesity in the United States is second only to tobacco use as a public health threat. Obesity as a single causative factor now stands to actually negatively impact life expectancy rates, which have risen for the last 200 years (52). Obese individuals and even persons with mild to moderate overweight have increased risk for multiple conditions, many of which are associated with a relatively high rate of morbidity and mortality, such as type 2 diabetes, cardiovascular disease, endometrial, postmenopausal breast, kidney, and colon cancer, musculoskeletal disorders, sleep apnea, and gallbladder disease (5,7,51,53-56). For example, type 2 diabetes is directly related to body weight (57).

The number of overweight and obese persons is also emerging as a major financial drain, resulting in more than $70 billion a year in public health expenditures in the United States (58). Obesity also contributes to higher health care expenditures than either smoking or alcohol use (59). Overweight and obesity cost taxpayers $117 billion per year in direct health care costs and indirect costs, such as lost wages (60). One study found that obese adults (18 to 65 years of age) have 36% higher than average annual medical expenditures compared to those of normal weight (59).

Malnutrition and Obesity: A Double Burden of Disease in Developing Countries

Almost 30% of the people on our planet suffer from malnutrition (61). Among children under 5 years of age in the developing world, some 60% of all deaths are related to this condition (1). Concurrently, an epidemic of obesity, with its attendant co-morbidities of diabetes, stroke, and CVD, has also affected developing as well as industrialized countries (2). This is associated with a high prevalence of obesity beginning in youth in developing countries and regions as diverse as India, Nigeria, Latin America, and the Caribbean (62). Thus, beginning with malnutrition in early childhood, nutritional transitions may occur leading to relatively cheap high energy density diets that are basically inadequate.

The good news about economic development is that it leads to an increased food supply and a decrease in dietary deficiencies as has occurred in much of Latin America. The bad news is that some of the shifts that have occurred in food availability have led to higher energy density diets with increases in saturated fat and sugar as well as reduced fresh fruit and vegetable intake (63). Unfortunately, these dietary changes have occurred in conjunction with other unhealthy lifestyle changes including reduced physical activity at work and leisure (64). Thus, people within particular developing countries may concomitantly suffer from food shortages, nutrient inadequacies, and obesity, all leading to an increase in chronic diseases (2).

Metabolic Syndrome, Type 2 Diabetes, and Cardiovascular Risk

The worldwide increase in the prevalence of obesity in the past two decades has been accompanied by two major medical developments that have important consequences for the future prevalence of CVD. One of these has been the current worldwide epidemic of type 2 diabetes (65). The second development has been the recognition that obesity is associated with the clustering of a group of CVD risk factors that has been termed the metabolic syndrome (66).

Risk factors that comprise the metabolic syndrome include, but are not limited to, central obesity, high blood pressure, glucose intolerance, elevated triglycerides, low levels of high density lipoprotein (HDL) cholesterol, fibrinolysis, and insulin resistance (67-68). Diagnostic guidelines for metabolic syndrome have been provided by the World Health Organization (68) and the National Cholesterol Education Program (NCEP) in the United States (67) among others. Recently,

using a hierarchical structure analysis, Shen et al. showed that four factors, obesity, insulin resistance, dyslipidemia, and hypertension, were all significantly associated with a common metabolic syndrome factor across gender and ethnic groups (69). To the extent that the insulin resistance factor was made up of fasting insulin and fasting glucose, this finding of an insulin resistance factor, helping to define metabolic syndrome, lends support to the WHO working definition, which specifies either glucose intolerance or insulin resistance as a prerequisite condition. Both type 2 diabetes and impaired glucose tolerance have previously been closely associated with the syndrome. Clustering of the syndrome components predicts both the development of manifest diabetes and CVD (70-71).

The relationship between psychosocial factors and metabolic syndrome is not well understood, but the Third National Health and Nutrition Examination Study in the United States found that women with a history of a major depressive episode were twice as likely to have the metabolic syndrome compared with those without a history of depression (72).

Type 2 diabetes accounts for the vast majority of diabetes cases worldwide and for more than 90% of cases in the United States (73). It develops when the production of insulin by the pancreas is insufficient to overcome the underlying abnormality of increased resistance to its action. In its early stage, type 2 diabetes is characterized by an overproduction of insulin (hyperinsulinemia). As the disease progresses, the insulin level falls, as the insulin producing cells of the pancreas begin to fail. Complications of untreated type 2 diabetes include: blindness, kidney failure, foot ulcerations that may lead to amputation, and increased risk of infections, stroke, and CVD. According to WHO, the criterion for diabetes is a fasting plasma glucose concentration = 126mg/dL (68).

In the year 2000, approximately 150 million people worldwide had type 2 diabetes and this figure is expected to double by 2025 (74). While type 2 diabetes is the fourth or fifth leading cause of mortality in most developed countries, it is also reaching epidemic proportions in many developing countries (75). It is expected that in the near future the majority of cases of type 2 diabetes will occur in these developing countries with India and China having more cases than any other country in the world (76). People with diabetes are more likely to die from a heart attack and are more likely than those without diabetes to have a second event (77). Patients with diabetes, who have never had a myocardial infarction, have as high a risk of heart attack

as non-diabetics who have already had a myocardial infarction (78).

Lifestyle Intervention for Prevention of Type 2 Diabetes and Cardiovascular Disease

Current evidence suggests that moderate weight reduction (5-10%) may reduce major risk factors for type 2 diabetes and CVD including obesity, elevated blood glucose, insulin resistance, dyslipidemia, fibrinolysis, inflammation, and high blood pressure (79-82). Both the NCEP (67) and the WHO Expert Panel (83) have stressed the importance of lifestyle modification (including caloric restriction, improved nutrition, and physical activity) in the prevention of type 2 diabetes and CVD.

The largest and most comprehensive study of the effect of lifestyle intervention in subjects at risk for type 2 diabetes was reported by the Diabetes Prevention Program (84). This trial randomly assigned 3234 non-diabetic persons with elevated fasting and post-load plasma glucose concentrations to placebo, metformin, or a lifestyle modification program. The goals were for participants to have 7% weight loss and 150 minutes of physical activity per week. Average follow-up was 2.8 years. The lifestyle intervention significantly reduced the incidence of type 2 diabetes by 58% and metformin by 31% as compared with placebo. The lifestyle intervention was significantly more effective than metformin. In a similar trial carried out in Finland, Tuomilehto et al. randomly assigned 522 middle-aged, overweight men and women with impaired glucose tolerance to either an intervention or a control group (85). Each subject in the intervention group received individualized counseling aimed at reducing weight and intake of total fat and saturated fat, and increasing intake of fiber and physical activity. Mean duration of follow-up was 3.2 years. During the trial the risk of diabetes was significantly reduced by 58%. The reduction in the incidence of diabetes was directly associated with changes in lifestyle. Both the DPP (84) and the Finish diabetes prevention trial (85) showed lifestyle changes that lasted several years and are thus an excellent model for such interventions. The Look AHEAD NIH trial now is assessing the long-term effects of such a program in terms of morbidity and mortality in type 2 diabetics (86).

HIV and AIDS

With the increased survival rate of people with HIV infection, primarily due to the use of antiretroviral medications, the co-morbid prevalence and impact of disability in this population has also risen. As in the general population, exer-

cise is a primary management strategy used to ameliorate impairments (problems with body function as a significant loss, such as pain or weakness), activity limitations (difficulties an individual may have, such as inability to engage in moderate exertion), and participation restrictions (problems, such as inability to work) in victims of HIV/AIDS (87). Exercise can be used to address unwanted increases in weight and body fat related to metabolic syndrome as a consequence of the use of highly active antiretroviral treatment (HAART) and from HIV infection itself (88).

Regular exercise has been found to slow down the progression of HIV and increase the CD4 cell count. The results of one study showed that HIV patients exercising 3-4 times per week were less likely to develop AIDS than those only carrying out daily exercise (89), revealing a need to slightly limit the amount of physical activity for people with HIV compared to other populations, where the WHO recommends one hour of moderate exercise per day for the prevention of chronic disease (90). Other improvements due to exercise include muscle strength and flexibility, cardiopulmonary fitness, and decreases in depression, anxiety, and anger (91-94). Progressive resistance exercise or a combination of progressive resistance exercise and aerobic exercise at least three times a week for at least four weeks appears to be safe and may lead to clinically important changes in body weight and composition for adults living with HIV/AIDS who are medically stable in immunological and virological status (95-96). These studies indicate that moderate levels of physical activity are safe and beneficial in the short term for individuals infected with HIV.

While the benefits of exercise for the person with HIV are relatively straightforward, the nutritional recommendations have changed from before HAART until now. Malnutrition, low serum levels of micronutrients, chronic diarrhea, anorexia, malabsorption, impaired nutrient storage, increased energy demands, and altered metabolism were common in persons with AIDS prior to HAART and are still common in resource-limited countries (97). Studies conducted before the widespread use of HAART suggested that HIV infection is also associated with a proatherogenic lipid profile characterized by an increase in triglyceride levels, a decrease in HDL cholesterol levels, and the presence of small, dense LDL particles (98-100). While the use of multivitamin or single micronutrient supplementation has been modest at best (101), the use of HAART is leading to new questions about the importance of micronutrients for persons with HIV. Even though macronutrient

deficiencies are uncommon and less severe in developed countries today, HAART and HIV itself are having a profound affect on oxidative stress, lipodystrophy, and metabolic syndrome (97). In particular, the use of protease inhibitors (PI) has had further deleterious effects on metabolic risk factors. Specifically, the initiation of PI-based HAART is associated with the development of insulin resistance in 25% to 62% and the development of overt new-onset diabetes mellitus in 6% to 7% (88,102-103) in persons with HIV. Increases in LDL cholesterol and triglyceride levels following HAART have also been observed (104-105). Thus, in addition to recommending exercise to decrease visceral fat and improve lipid profiles, nutritional intervention for persons with HIV/AIDS on HAART should focus on high fiber, foods with a low glycemic index, low saturated fat and processed sugars, and high intake of fresh and natural fruits and vegetables.

Mental Health

Approximately one-quarter of all adults are suffering from a diagnosable mental disease (106), including depressive and other mood disorders. Nearly half (45%) of those with any mental disorder meet criteria for two or more disorders with severity strongly related to co-morbidity (106). Major Depressive Disorder (MDD) is the leading cause of disability for persons between the ages of 15 and 44 (107). Almost 15 million adults over 18 years of age are affected by MDD (106) and it is more prevalent in women than in men (108). Depressive disorders often co-occur with anxiety disorders and substance abuse (109). Almost half of lost employment productivity is due to MDD at $44 billion per year (110). The WHO's Global Burden of Disease Study looked at disability-adjusted life years, which measure lost years of healthy life regardless of whether the years were lost to premature death or disability for various diseases (111). Disability caused by MDD ranks second to CVD in the magnitude of disease burden in the developed world.

Several studies have investigated the effects of nutrients and/or specific components of the typical diet and their impact on depression. In a study of healthy college students, moderate and heavy coffee drinkers scored higher on a depression scale than did low users (112). In addition, the intake of caffeine has been linked with the degree of mental illness in psychiatric patients: the higher the intake, the more severe the depression (113). Excess intake of refined sugar from sweet foods can also aggravate depression. The combination of caffeine and refined sugar is likely even worse for depression than either substance consumed alone. In

one study, restricting sugar and caffeine in people with depression has been reported to elevate mood (114).

Low levels of folic acid have been noted in depressed patients (115). In studies of depressed patients, 15% to 38% have been shown to be deficient in serum or red blood cell folic acid (116-118). Depression is the most common symptom of a folic acid deficiency. Other symptoms of folic acid deficiency are: fatigue, apathy, and dementia. Inositol is a B vitamin required for the activity of several important neurotransmitters, including serotonin. Depressed people often have low levels of inositol. In one clinical study, subjects were given 12 grams of inositol per day and the results showed that they had therapeutic results similar to common antidepressant drugs, but with no unwanted side effects (119). Additional research has also confirmed the value of inositol for treating depression (120). The results of a number of clinical studies suggest that S-adenosyl-L-methionine may be a useful natural antidepressants (121).

While the use of these various nutrients may be beneficial, a more holistic approach to nutritional modification in depressed patients has not been investigated. Nutritional therapy emphasizing mostly low-fat, whole-plant foods, while avoiding simple sugars and chemical additives or preservatives, has been successfully utilized in other diseases and can also be beneficial for weight control, hypertension, hyperlipidemia, dyslipidemia, CVD risk, and mental status (9-12). Additionally, these benefits have been found to last for years if the diet style is maintained (13-14). However, randomized clinical trials are needed to establish efficacy and effectiveness.

Research has shown that regular exercise can improve mood in cases of mild to moderate depression (122). One study reported the relative risk of depression was 27 percent lower for people playing three or more hours of sport a week compared with those playing no sport at all (123). Another study compared the effects of exercise and drug therapy in treating depression in older people (124). The 156 depressed men and women were divided into three groups. Over 16 weeks, one group took antidepressants, the second group undertook an aerobic exercise program, and the third group used both medications and exercise. The results indicated that after 6 months those patients who continued to exercise were much less likely to experience a return of their depression than were the other patients. Only 8 percent of patients in the exercise group had their depression return, while 38 percent of the drug-only group and 31 percent of the exercise-plus-drug group relapsed. Other important

findings included that the more one exercised, the less likely one would see their depressive symptoms return and for each 50-minute increment of exercise, an accompanying 50 percent reduction in relapse risk was found.

Another study sought to examine the exercise-depression link by splitting a group of people suffering with MDD into two groups. One group exercised aerobically for one hour, three times a week for nine weeks while maintaining a course of psychotherapy and medication, and the other group continued with psychotherapy and medication only. The study showed significantly larger reduction scores in depression in the exercising group compared with the therapy and medication group (125). The effects of aerobic exercise have been contrasted against relaxation training on depressed individuals. Depression scores were reduced by both methods, although exercise provided greater reductions in depressive scores (126). Exercise benefits have been demonstrated in people who are not clinically depressed, but who present some depressive symptoms (127-128). Another study compared aerobic exercise, including jogging and cycling, to non-aerobic circuit exercise on a multi-gym. The study found both methods of exercise to produce significant reductions in depressive scores (129).

Conclusions

Although medications are available to treat overweight, and surgery is available for obesity, complex medical regimens are costly to apply over a typical lifetime, have untoward side effects, and patients may fail to achieve the treatment goals required to lose and/or maintain excess fat and weight. Also, standard conventional medical treatment involving medications or surgery has not been proven to unequivocally cure or reverse the effects of obesity. Potentially modifiable environmental factors, including poor dietary choices and failing to engage in regular exercise, are known to be primary contributors to overweight and obesity and related problems including high blood pressure, CVD, stroke, diabetes, certain types of cancer, arthritis, and breathing problems. Given the enormous public health cost of obesity, focusing on healthy eating and a moderate and consistent exercise program should be the basis of any attempt to achieve weight loss and subsequent control (16). The true impact of the public health costs of obesity can only be understood within the context of both mental and physical health.

Reference

1. World Health Organization. Childhood nutrition and progress in implementing the

international code of marketing of breast-milk substitutes. Geneva; 2002. Report No.: A55/14.
2. World Health Organization. Diet, nutrition, and the prevention of chronic disease. Geneva: World Health Organization; 2003. Report No.: WHO Technical Report 916.
3. Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! Public Health Nutr. 2002 Feb;5(1A):205-14.
4. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. JAMA. 2004 Mar 10;291(10):1238-45.
5. Ditschuneit HH, Flechtner-Mors M. Value of structured meals for weight management: risk factors and long-term weight maintenance. Obes Res. 2001 Nov;9 Suppl 4:284S-9S.
6. Pasman WJ, Saris WH, Westerterp-Plantenga MS. Predictors of weight maintenance. Obes Res. 1999 Jan;7(1):43-50.
7. Westerterp-Plantenga MS, Kempen KP, Saris WH. Determinants of weight maintenance in women after diet-induced weight reduction. Int J Obes Relat Metab Disord. 1998 Jan;22(1):1-6.
8. Leermakers EA, Perri MG, Shigaki CL, Fuller PR. Effects of exercise-focused versus weight-focused maintenance programs on the management of obesity. Addict Behav 1999 Mar;24(2):219-27.
9. Anderson J. Dietary fiber in nutrition management of diabetes. In: Vahouny G, Kritchevsky D, editors. Dietary fiber: basic and clinical aspects. New York: Plenum Press; 1986. p. 343-60.
10. Anderson JW, Chen WJ, Sieling B. Hypolipidemic effects of high-carbohydrate, high-fiber diets. Metabolism. 1980 Jun;29(6):551-8.
11. Anderson JW, Gustafson NJ, Bryant CA, Tietyen-Clark J. Dietary fiber and diabetes: a comprehensive review and practical application. J Am Diet Assoc. 1987 Sep;87(9):1189-97.
12. Barnard RJ, Lattimore L, Holly RG, Cherny S, Pritikin N. Response of non-insulin-dependent diabetic patients to an intensive program of diet and exercise. Diabetes Care. 1982 Jul;5(4):370-4.
13. Barnard RJ, Massey MR, Cherny S, O'Brien LT, Pritikin N. Long-term use of a high-complex-carbohydrate, high-fiber, low-fat diet and exercise in the treatment of NIDDM patients. Diabetes Care. 1983 May;6(3):268-73.
14. Story L, Anderson JW, Chen WJ, Karounos D, Jefferson B. Adherence to high-carbohydrate, high-fiber diets: long-term studies of non-obese diabetic men. J Am Diet Assoc. 1985 Sep;85(9): 1105-10.
15. National Research Council. Diet and health: implications for reducing chronic disease risk. Washington: National Academy Press; 1989.
16. United States Department of Agriculture. Report of the dietary guidelines advisory committee on the dietary guidelines for Americans, 1995, to the Secretary of Health and Human Services and the Secretary of Agriculture. Washington: US Department of Agriculture; 1995.
17. Maru S, van der Schouw YT, Gimbrere CH, Grobbee DE, Peeters PH. Body mass index and short-term weight change in relation to mortality in Dutch women after age 50 y. Am J Clin Nutr. 2004 Jul;80(1):231-6.
18. 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 Jun 15;141(12):1128-41.
19. 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 Jan 5;293(1):43-53.
20. Brehm B, Seeley R, Daniels S, D'Alessio D. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. J Clin Endocrinol Metab. 2003;88: 1617-23.

21. Yancy WS, Jr., Olsen MK, Guyton JR, Bakst RP, Westman EC. A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: a randomized, controlled trial. Ann Intern Med. 2004 May 18;140(10):769-77.
22. Gross LS, Li L, Ford ES, Liu S. Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment. Am J Clin Nutr. 2004 May;79(5):774-9.
23. Brehm B, Spang S, Lattin B, Seeley R, Daniels S, D'Alessio D. The role of energy expenditure in the differential weight loss in obese women on low-fat and low-carbohydrate diets. J Clin Endocrinol Metab. 2005;90:1475-82.
24. Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, et al. A randomized trial of a low-carbohydrate diet for obesity. N Engl J Med. 2003 May 22;348(21):2082-90.
25. McAuley KA, Hopkins CM, Smith KJ, McLay RT, Williams SM, Taylor RW, et al. Comparison of high-fat and high-protein diets with a high-carbohydrate diet in insulin-resistant obese women. Diabetologia. 2005 Jan;48(1):8-16.
26. Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, et al. A low-carbohydrate as compared with a low-fat diet in severe obesity. N Engl J Med. 2003 May 22;348(21):2074-81.
27. Sondike SB, Copperman N, Jacobson MS. Effects of a low-carbohydrate diet on weight loss and cardiovascular risk factor in overweight adolescents. J Pediatr. 2003 Mar;142(3):253-8.
28. United States Department of Health and Human Services. The surgeon general's report on nutrition and health. Washington: United States Govt. Printing Office; 1988.
29. Block G, Patterson B, Subar A. Fruit, vegetables, and cancer prevention: a review of the epidemiological evidence. Nutr Cancer. 1992;18(1):1-29.
30. Bal DG, Foerster SB. Changing the American diet. Impact on cancer prevention policy recommendations and program implications for the American Cancer Society. Cancer. 1991 May 15;67(10):2671-80.
31. Willett WC. Diet and health: what should we eat? Science. 1994 Apr 22;264(5158): 532-7.
32. Thomas P. Improving America's diet and health: From recommendations to action. Washington: National Academy Press; 1991.
33. Matsudo V, Matsudo S, Andrade D, Araujo T, Andrade E, de Oliveira LC, et al. Promotion of physical activity in a developing country: the Agita Sao Paulo experience. Public Health Nutr. 2002 Feb;5(1A):253-61.
34. Colditz GA. Economic costs of obesity and inactivity. Med Sci Sports Exerc. 1999 Nov;31(11 Suppl):S663-S667.
35. Blair SN, Kampert JB, Kohl HW, III, Barlow CE, Macera CA, Paffenbarger RS, Jr., et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA. 1996 Jul 17;276(3):205-10.
36. Church T, Kamper J, Gibbons L, Barlow C, Blair S. Usefulness of cardiorespiratory fitness as a predictor of all-cause and cardiovascular disease mortality in men with systemic hypertension. Am J Cardiol. 2001;88:651-6.
37. 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 Jul 18;325(3):147-52.
38. United States Department of Health and Human Services. Physical activity and health: a report of the surgeon general. Atlanta (GA): United States Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996. Report No.: 017-023-00196-5.
39. United States Department of Health and Human Services. Healthy people 2010: understanding and improving health. Washington: United States Government Printing Office; 2000.

40. Dipietro L, Dziura J, Blair S. Estimated change in physical activity levels (PAL) and prediction of 5-yr weight change in middle-aged men: the aerobics center longitudinal study. International Int J Obes Relat Metab Disord. 2004;28(12):1541-7.
41. Haapanen N, Miilunpalo S, Pasanen M, Oja P, Vuori I. Association between leisure time physical activity and 10-year body mass change among working-aged men and women. Int J Obes Relat Metab Disord. 1997 Apr;21(4):288-96.
42. Schmitz KH, Jacobs DR, Jr., Leon AS, Schreiner PJ, Sternfeld B. Physical activity and body weight: associations over ten years in the CARDIA study. Coronary Artery Risk Development in Young Adults. Int J Obes Relat Metab Disord. 2000 Nov;24(11):1475-87.
43. 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 May;17(5):279-86.
44. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? Science. 2003 Feb 7;299(5608):853-5.
45. Klem ML, Wing RR, McGuire MT, Seagle HM, Hill JO. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. Am J Clin Nutr. 1997 Aug;66(2):239-46.
46. Wing RR, Hill JO. Successful weight loss maintenance. Annu Rev Nutr. 2001;21:323-41.
47. Jakicic JM, Winters C, Lang W, Wing RR. Effects of intermittent exercise and use of home exercise equipment on adherence, weight loss, and fitness in overweight women: a randomized trial. JAMA. 1999 Oct 27;282(16):1554-60.
48. Wyatt H, Peters J, Reed G, Grunwald G, Barry M, Thompson H, et al. Using electronic step counters to increase lifestyle physical activity: Colorado on the move. Journal of Physical Activity and Health. 2004;1:181-91.
49. Deitel M. Overweight and obesity worldwide now estimated to involve 1.7 billion people. Obes Surg. 2003 Jun;13(3):329-30.
50. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA. 2002 Oct 9;288(14):1723-7.
51. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. 2000. Report No.: 894.
52. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, et al. A potential decline in life expectancy in the United States in the 21st century. N Engl J Med. 2005 Mar 17;352(11):1138-45.
53. Burton BT, Foster WR. Health implications of obesity: an NIH Consensus Development Conference. J Am Diet Assoc. 1985 Sep;85(9):1117-21.
54. Pasman WJ, Saris WH, Muls E, Vansant G, Westerterp-Plantenga MS. Effect of exercise training on long-term weight maintenance in weight-reduced men. Metabolism. 1999 Jan;48(1):15-21.
55. Pi-Sunyer F, Becker D, Bouchard C, Carlton R. NHLBI obesity education initiative expert panel on the identification, evaluation, and treatment of overweight and obesity in adults. Obesity Research. 1998;6:51S-209S.
56. Visscher TL, Seidell JC. The public health impact of obesity. Annu Rev Public Health. 2001;22:355-75.
57. American Diabetes Association. National Diabetes Fact Sheet. 2002. Ref Type: Generic.
58. Wickelgren I. Obesity: how big a problem? Science. 2001;280(5368):1364-7.
59. Sturm R. Datawatch: the effects of obesity, smoking, and drinking on medical problems and costs. Health Aff. 2002;21(2):245-53.
60. United States Office of the Surgeon General. The surgeon general's call to action to prevent and decrease overweight and obesity. Washington: United States Office of the Surgeon General; 2001.
61. World Health Organization. A global agenda for combating malnutrition: Progress report. Geneva: World Health

Organization; 2000. Report No.: WHO/NHD/00.6.
62. Peña M, Bacallao J. Obesity among the poor: An emerging problem in Latin America and the Caribbean. In: Peña M, Bacallao J, editors. Obesity and poverty: a new public health challenge. Washington: Pan American Health Organization; 2000. p. 3-10.
63. Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! Public Health Nutr. 2002 Feb;5(1A):205-14.
64. Matsudo V, Matsudo S, Andrade D, Araujo T, Andrade E, de Oliveira LC, et al. Promotion of physical activity in a developing country: the Agita Sao Paulo experience. Public Health Nutr. 2002 Feb;5(1A):253-61.
65. Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. Nature. 2001 Dec 13;414(6865):782-7.
66. Grundy SM. Obesity, metabolic syndrome, and cardiovascular disease. J Clin Endocrinol Metab. 2004 Jun;89(6):2595-600.
67. Executive Summary of the third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001 May 16;285(19):2486-97.
68. World Health Organization. Definitions, diagnosis, and classification of diabetes mellitus and its complications. Report of a WHO consultation. Part I: Diagnosis and classification of diabetes mellitus. Geneva: World Health Organization; 1999.
69. Shen BJ, Goldberg RB, Llabre MM, Schneiderman N. Is the factor structure of the metabolic syndrome comparable between men and women and across three ethnic groups: the Miami Community Health Study. Ann Epidemiol. 2006 Feb;16(2):131-7.
70. Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA. 2002 Dec 4;288(21):2709-16.
71. Lorenzo C, Okoloise M, Williams K, Stern MP, Haffner SM. The metabolic syndrome as predictor of type 2 diabetes: the San Antonio heart study. Diabetes Care. 2003 Nov;26(11):3153-9.
72. Kinder LS, Carnethon MR, Palaniappan LP, King AC, Fortmann SP. Depression and the metabolic syndrome in young adults: findings from the Third National Health and Nutrition Examination Survey. Psychosom Med. 2004 May;66(3):316-22.
73. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care. 1998 Sep;21(9):1414-31.
74. 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 Feb;7(1A):147-65.
75. Amos AF, McCarty DJ, Zimmet P. The rising global burden of diabetes and its complications: estimates and projections to the year 2010. Diabet Med. 1997;14 Suppl 5:S1-85.
76. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care. 1998 Sep;21(9):1414-31.
77. Miettinen H, Lehto S, Salomaa V, Mahonen M, Niemela M, Haffner SM, et al. Impact of diabetes on mortality after the first myocardial infarction. The FINMONICA Myocardial Infarction Register Study Group. Diabetes Care. 1998 Jan;21(1):69-75.
78. Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med. 1998 Jul 23;339(4):229-34.
79. Pritchett AM, Foreyt JP, Mann DL. Treatment of the metabolic syndrome: the im-

pact of lifestyle modification. Curr Atheroscler Rep. 2005 Mar;7(2):95-102.
80. Park HS, Lee K. Greater beneficial effects of visceral fat reduction compared with subcutaneous fat reduction on parameters of the metabolic syndrome: a study of weight reduction programmes in subjects with visceral and subcutaneous obesity. Diabet Med. 2005 Mar;22(3): 266-72.
81. Hong K, Li Z, Wang HJ, Elashoff R, Heber D. Analysis of weight loss outcomes using VLCD in black and white overweight and obese women with and without metabolic syndrome. Int J Obes (Lond). 2005 Apr;29(4):436-42.
82. Jellema A, Plat J, Mensink RP. Weight reduction, but not a moderate intake of fish oil, lowers concentrations of inflammatory markers and PAI-1 antigen in obese men during the fasting and postprandial state. Eur J Clin Invest. 2004 Nov;34(11): 766-73.
83. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med. 1998 Jul;15(7): 539-53.
84. 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 Feb 7;346(6):393-403.
85. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med. 2001 May 3;344(18):1343-50.
86. Ryan DH, Espeland MA, Foster GD, Haffner SM, Hubbard VS, Johnson KC, et al. Look AHEAD (Action for Health in Diabetes): design and methods for a clinical trial of weight loss for the prevention of cardiovascular disease in type 2 diabetes. Control Clin Trials. 2003 Oct;24(5): 610-28.
87. World Health Organization. International classification of functioning, disability, and health. Geneva: World Health Organization; 2001.
88. Dever LL, Oruwari PA, Figueroa WE, O'Donovan CA, Eng RH. Hyperglycemia associated with protease inhibitors in an urban HIV-infected minority patient population. Ann Pharmacother. 2000 May;34(5): 580-4.
89. Mustafa T, Sy FS, Macera CA, Thompson SJ, Jackson KL, Selassie A, et al. Association between exercise and HIV disease progression in a cohort of homosexual men. Ann Epidemiol. 1999 Feb;9(2): 127-31.
90. World Health Organization. The world health report 2002: Reducing risks, promoting healthy life. Geneva: World Health Organization; 2002.
91. LaPerriere A, Fletcher MA, Antoni MH, Klimas NG, Ironson G, Schneiderman N. Aerobic exercise training in an AIDS risk group. Int J Sports Med. 1991 Jun;12 Suppl 1:S53-S57.
92. MacArthur RD, Levine SD, Birk TJ. Supervised exercise training improves cardiopulmonary fitness in HIV-infected persons. Med Sci Sports Exerc. 1993 Jun;25(6):684-8.
93. Rigsby LW, Dishman RK, Jackson AW, Maclean GS, Raven PB. Effects of exercise training on men seropositive for the human immunodeficiency virus-1. Med Sci Sports Exerc. 1992 Jan;24(1):6-12.
94. Spence DW, Galantino ML, Mossberg KA, Zimmerman SO. Progressive resistance exercise: effect on muscle function and anthropometry of a select AIDS population. Arch Phys Med Rehabil. 1990 Aug;71(9):644-8.
95. Lox CL, McAuley E, Tucker RS. Aerobic and resistance exercise training effects on body composition, muscular strength, and cardiovascular fitness in an HIV-1 population. Int J Behav Med. 1996;3(1): 55-69.
96. Roubenoff R, McDermott A, Weiss L, Suri J, Wood M, Bloch R, et al. Short-term progressive resistance training increases

strength and lean body mass in adults infected with human immunodeficiency virus. AIDS. 1999 Feb 4;13(2):231-9.
97. Tang AM, Lanzillotti J, Hendricks K, Gerrior J, Ghosh M, Woods M, et al. Micronutrients: current issues for HIV care providers. AIDS. 2005 Jun 10;19(9): 847-61.
98. Feingold KR, Krauss RM, Pang M, Doerrler W, Jensen P, Grunfeld C. The hypertriglyceridemia of acquired immunodeficiency syndrome is associated with an increased prevalence of low density lipoprotein subclass pattern B. J Clin Endocrinol Metab. 1993 Jun;76(6): 1423-7.
99. Grunfeld C, Kotler DP, Hamadeh R, Tierney A, Wang J, Pierson RN. Hypertriglyceridemia in the acquired immunodeficiency syndrome. Am J Med. 1989 Jan;86(1): 27-31.
100. Shor-Posner G, Basit A, Lu Y, Cabrejos C, Chang J, Fletcher M, et al. Hypocholesterolemia is associated with immune dysfunction in early human immunodeficiency virus-1 infection. Am J Med. 1993 May;94(5):515-9.
101. Tang AM, Smit E. Selected vitamins in HIV infection: a review. AIDS Patient Care STDS. 1998 Apr;12(4):263-73.
102. Carr A, Samaras K, Thorisdottir A, Kaufmann GR, Chisholm DJ, Cooper DA. Diagnosis, prediction, and natural course of HIV-1 protease-inhibitor-associated lipodystrophy, hyperlipidaemia, and diabetes mellitus: a cohort study. Lancet. 1999 Jun 19;353(9170):2093-9.
103. Walli R, Goebel FD, Demant T. Impaired glucose tolerance and protease inhibitors. Ann Intern Med. 1998 Nov 15;129(10): 837-8.
104. Calza L, Manfredi R, Farneti B, Chiodo F. Incidence of hyperlipidaemia in a cohort of 212 HIV-infected patients receiving a protease inhibitor-based antiretroviral therapy. Int J Antimicrob Agents. 2003 Jul;22(1):54-9.
105. Nolan D. Metabolic complications associated with HIV protease inhibitor therapy. Drugs. 2003;63(23):2555-74.
106. Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005 Jun;62(6):617-27.
107. World Health Organization. The world health report 2004: changing history, annex table 3: Burden of disease in DALYs by cause, sex, and mortality stratum in WHO regions, estimates for 2002. Geneva: The World Health Organization; 2004.
108. Kessler RC, Berglund P, Demler O, Jin R, Koretz D, Merikangas KR, et al. The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). JAMA. 2003 Jun 18;289(23):3095-105.
109. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005 Jun;62(6):593-602.
110. Stewart WF, Ricci JA, Chee E, Hahn SR, Morganstein D. Cost of lost productive work time among US workers with depression. JAMA. 2003 Jun 18;289(23): 3135-44.
111. Murray C, Lopez A. The global burden of disease and injury series. Volume I. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020. Geneva, World Health Organization; 1996. Ref Type: Generic.
112. Gilliland K, Andress D. Ad lib caffeine consumption, symptoms of caffeinism, and academic performance. Am J Psychiatry. 1981 Apr;138(4):512-4.
113. Greden JF, Fontaine P, Lubetsky M, Chamberlin K. Anxiety and depression associated with caffeinism among psychiatric inpatients. Am J Psychiatry. 1978 Aug;135(8):963-6.
114. Christensen L. Psychological distress and diet-effects of sucrose and caffeine. J Appl Nutr. 1988;40:44-50.

115. Alpert JE, Fava M. Nutrition and depression: the role of folate. Nutr Rev. 1997 May;55(5):145-9.
116. Fava M, Borus JS, Alpert JE, Nierenberg AA, Rosenbaum JF, Bottiglieri T. Folate, vitamin B12, and homocysteine in major depressive disorder. Am J Psychiatry. 1997 Mar;154(3):426-8.
117. Godfrey PS, Toone BK, Carney MW, Flynn TG, Bottiglieri T, Laundy M, et al. Enhancement of recovery from psychiatric illness by methylfolate. Lancet. 1990 Aug 18;336(8712):392-5.
118. Reynolds EH, Preece JM, Bailey J, Coppen A. Folate deficiency in depressive illness. Br J Psychiatry. 1970 Sep; 117(538):287-92.
119. Levine J, Barak Y, Gonzalves M, Szor H, Elizur A, Kofman O, et al. Double-blind, controlled trial of inositol treatment of depression. Am J Psychiatry. 1995 May; 152(5):792-4.
120. Levine J, Gonzales M, Babur I, Stir S, Elizur A, Kofman O, et al. Inositol 6 gm daily may be effective in depression but not in schizophrenia. Hum Psychopharmacol. 1993;8:49-53.
121. Fava M, Giannelli A, Rapisarda V, Patralia A, Guaraldi GP. Rapidity of onset of the antidepressant effect of parenteral S-adenosyl-L-methionine. Psychiatry Res. 1995 Apr 28;56(3):295-7.
122. Bloch D. Healing from depression: 12 weeks to a better mood. A body, mind, and spirit recovery program. Berkeley, CA: Ten Speed Press; 2002.
123. Paffenbarger RS, Jr., Lee IM, Leung R. Physical activity and personal characteristics associated with depression and suicide in American college men. Acta Psychiatr Scand. Suppl 1994;377:16-22.
124. Babyak M, Blumenthal JA, Herman S, Khatri P, Doraiswamy M, Moore K, et al. Exercise treatment for major depression: maintenance of therapeutic benefit at 10 months. Psychosom Med. 2000 Sep;62(5):633-8.
125. Byrne A, Byrne DG. The effect of exercise on depression, anxiety and other mood states: a review. J Psychosom Res. 1993 Sep;37(6):565-74.
126. McCann IL, Holmes DS. Influence of aerobic exercise on depression. J Pers Soc Psychol. 1984 May;46(5):1142-7.
127. Berger B, Owen D, Man F. A brief review of literature and examination of acute mood benefits of exercise in Czechoslovakian and United States swimmers. Int J Sport Psychol. 1993;24(2): 130-150.
128. King AC, Taylor CB, Haskell WL. Effects of differing intensities and formats of 12 months of exercise training on psychological outcomes in older adults. Health Psychol. 1993 Jul;12(4):292-300.
129. Martinsen E, Medhus A. Adherence to exercise and patients' evaluation of physical exercise in a comprehensive treatment program for depression. Nordic Psychiatric Quarterly. 1989;43:411-415. Ref Type: Journal (Full).

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