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American College of Sports Medicine Roundtable: Physical Activity in the Prevention and Treatment of Obesity and Its Comorbidities

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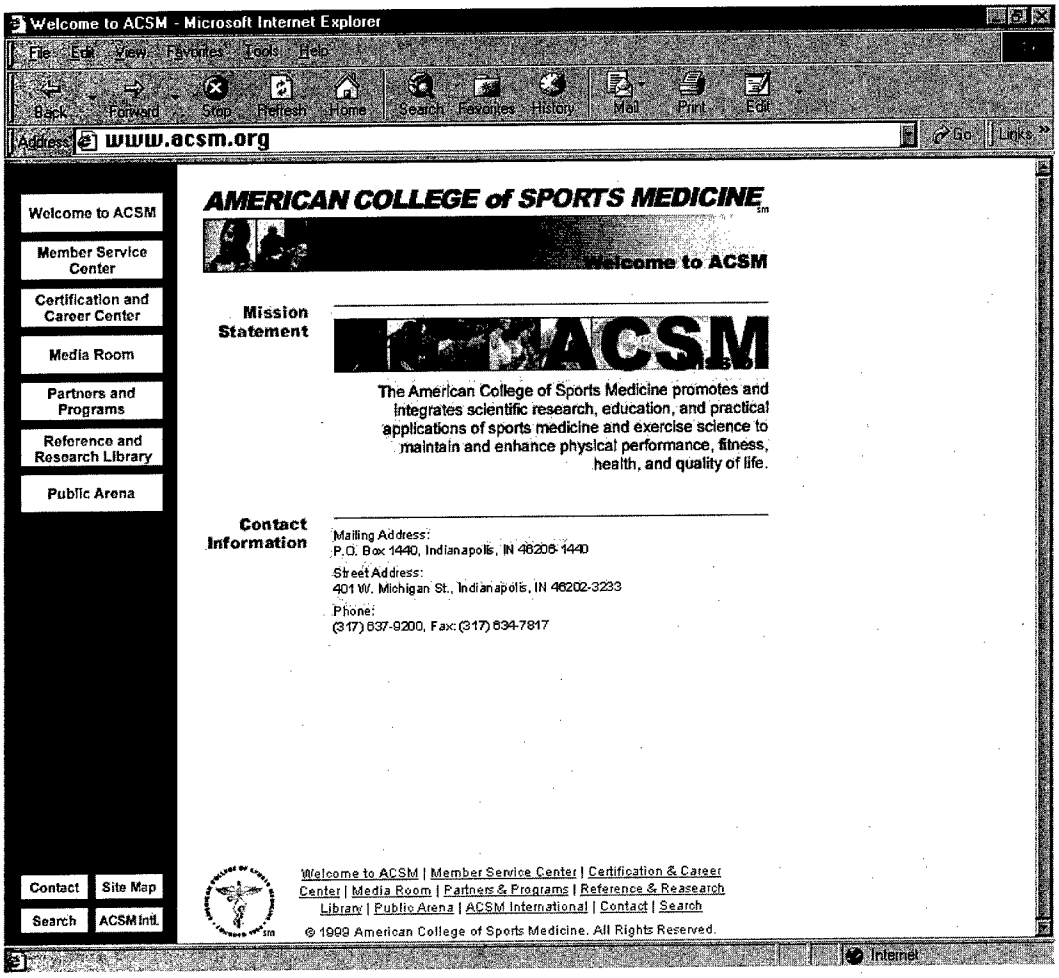

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AMERICAN COLLEGE of SPORTS MEDICINE$_{SM}$

ROUNDTABLE

PREFACE

Physical activity in the prevention and treatment of obesity and its comorbidities

We consider the consensus conference an outstanding success, the result of the contributions and hard work of many groups and individuals. The conference would not have been possible without the financial support of several public and private groups. M&M Mars provided a major grant to ACSM, which allowed us to commit to the conference and initiate the planning process. We are grateful for their substantial support and encouragement. The Robert Wood Johnson Foundation provided funds for publication of the review papers and consensus statement in this supplement of *Medicine and Science in Sports and Exercise.* Colleagues from the North American Society for the Study of Obesity provided financial support and scientific expertise in planning the program. Knoll Pharmaceutical Company and the International Food Information Council provided unrestricted educational grants to support the program. Several units of the U.S. Public Health Service provided additional financial resources without which the meeting would not have been possible. We are grateful for the support of the U.S. Centers for Disease Control and Prevention; the National Institute of Diabetes and Digestive and Kidney Diseases; the National Heart, Lung, and Blood Institute; and the National Institute of Child Health and Human Development. The National Coalition for Promoting Physical Activity also was a co-sponsor of the conference.

We thank the Program Committee for their hard work and creative suggestions in developing the program. Outstanding scientists from the physical activity and obesity research community who served on the Program Committee were Drs. William Dietz, John Foreyt, James Hill, Gay Israel, and Rena Wing, with Drs. Steven Rizk and Barbara Campaigne as *ex officio* members.

The Consensus Committee worked long hours before, during, and after the meeting, reviewing the evidence on physical activity and obesity, asking insightful and provocative questions, deliberating the issues, and writing the consensus report. We thank Dr. Scott Grundy for his leadership as Chair of the Consensus Committee and Drs. George Blackburn, Millicent Higgins, Ronald Lauer, Michael Perri, and Donna Ryan for their important contributions.

Twenty-four outstanding and dedicated scientists from around the world accepted the charge of the Program Committee to thoroughly review the evidence of a specific aspect of physical activity and obesity. They met the deadlines of preparing their papers before the conference so that the Consensus Committee could review them before coming to the meeting, participated vigorously in the discussions and debates at the conference, and cheerfully (we think) revised their reports after the exchange of ideas in Indianapolis. Their collected works, along with the Consensus Statement, comprise the material assembled in this special issue of *Medicine and Science in Sports and Exercise.* We hope, and expect, that this collection of papers, along with the Consensus Statement, will represent a prime source of information for all those interested in the topic of physical activity and obesity and will provide direction for research and policy in physical activity and obesity for the next several years.

Planning, organizing, and presenting this conference required enormous effort by the dedicated staff of the ACSM. We thank Jim Whitehead, ACSM Executive Vice-President, for his support and leadership throughout the planning process and during the meeting. Dr. Barbara Campaigne, formerly Director of Research at ACSM, was intimately involved in planning the program and provided excellent staff support to the Co-Chairs and the Program Committee. She was instrumental in keeping the process on schedule and in attending to countless details. Jane Gleason joined ACSM shortly before the meeting was held. She came into a challenging situation, in that Dr. Campaigne left ACSM about the time Jane joined our staff. Without missing a beat, Jane worked closely with the Co-Chairs in obtaining the review papers from participants, distributing them to the Consensus Committee, and coordinating many last minute details. Jane worked long hours with the Consensus Committee during and after the meeting as they developed their report. Amy Katzenberger and her staff did their usual outstanding job of handling logistical details for the meeting and in making the meeting a pleasure for the attendees. Other ACSM staff who provided the essential behind the scenes work without which no meeting is successful included Dave Brewer, Bev Brown, Claire Heister, Gail Hunt, Sandy Kuiper, Mark Robertson, and Amy Trobec. We extend our grateful thanks to them and applaud their high level of professionalism.

Steven N. Blair, P.E.D., FACSM
Claude Bouchard, Ph.D., FACSM
Co-Chairs of the Program Committee

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MEDICINE & SCIENCE IN SPORTS & EXERCISE®

Roundtable held February 4–7, 1999, Indianapolis, IN.

AMERICAN COLLEGE of SPORTS MEDICINE

ROUNDTABLE INTRODUCTION

Introductory comments for the consensus on physical activity and obesity

CLAUDE BOUCHARD and STEVEN N. BLAIR

Pennington Biomedical Research Center, Baton Rouge, LA; and Cooper Institute, Dallas, TX

A document developed by a group of experts from many disciplines over a period of about 2 years at the initiative of the National Heart, Lung and Blood Institute with the collaboration of several other NIH components was published in 1998 (10). Its purpose was to propose guidelines for the prevention and treatment of obesity and its comorbidities. One of the lessons learned in the course of the development of these guidelines was that the body of knowledge on physical activity and relevant obesity outcomes is extremely limited. There are few randomized clinical trials that have lasted 1 year or more, with reasonable statistical power, adequate monitoring of intervention protocols, high levels of compliance, and proper measurement of the outcome variables. The net result is a general lack of a solid research database regarding the role of physical activity in the prevention and treatment of overweight and obesity as well as their comorbidities. The evidence-based approach taken in the NIH Report is one that will have a growing influence on health-related issues, and all exercise specialists concerned with these topics, including physical activity and obesity, would do well to become familiar with it.

Another important publication published in 1998 was that of the World Health Organization consultation report on the worldwide obesity epidemic (20). It proposed a classification of body weight based on the body mass index (BMI) defined as weight in kilograms divided by height in meters squared (kg/m^2). The main merits of the classification are that it is simple, it is based on a large body of epidemiological and clinical data, and it provides a useful tool for international comparisons, for monitoring changes over time in a given country as well as changes associated with major lifestyle alterations, implementation of new public health policies, or other relevant interventions. The BMI classification scheme is reproduced in Table 1. We would like to emphasize that *overweight* is defined as a BMI ranging from 25 to 29.9 and *obesity* is set at a BMI of 30 and above. We have asked the participants at the Consensus Conference to use these cutoff points and the related terminology in their papers.

ASSESSMENT OF THE QUALITY OF THE EVIDENCE

The organizers of the Consensus Conference proposed early on to build on the foundations that proved to be so useful for the NIH Report (10). First, invited experts were asked to undertake the review of the topic assigned to them by drawing from the studies that had been retained by the NHLBI Panel and then expand their literature search to other sources when applicable. Second, participants were instructed to use the four evidence categories as defined in the NHLBI Report to assess the level and quality of evidence for each particular issue they were addressing and in developing a series of summary statements on the levels of evidence.

Table 2 reproduces the NHLBI report table on evidence categories. *Evidence Category A* is attained when there is a rich body of data from randomized controlled trials (RCT). The evidence is from endpoints of well-designed RCT that provide a consistent pattern of findings. Category A therefore requires substantial number of studies involving substantial number of participants (10).

Evidence Category B is reached when there is a limited body of data from RCT. It is applicable if few randomized trials exist, they are small in size, trial results are somewhat inconsistent, or trials were undertaken in populations that differ from the target population. Category B may also be attained based on the results of meta-analysis of RCT (10).

Evidence Category C is granted when the data supporting the conclusion are from uncontrolled or nonrandomized trials, or from cross-sectional or prospective observational studies (10).

Finally, *Evidence Category* D can be given when the provision of some guidance is deemed valuable but there is

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TABLE 1. Classification of overweight and obesity by BMI.

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

Adapted from WHO Report (20) Obesity: preventing and managing the global epidemic. Report of a WHO consultation on obesity. Geneva: World Health Organization, 1998.

no compelling scientific or clinical data to justify the use of categories A to C. Category D results from the expert judgment of participants and panel members (10).

PHYSICAL ACTIVITY AND THE OBESITY EPIDEMIC

Body weight is a function of energy balance over an extended period of time. Positive energy balance over weeks and months results in weight gain, whereas negative energy balance has the opposite effect. The increase in the prevalence of overweight and obesity cases worldwide is occurring against a background of a progressive reduction in the energy expended for work and occupational activities as well as for the accomplishment of personal chores and daily necessities (8,12,17). The reduction in energy expenditure associated with physical activity brought about by automation and changing job and professional environmental circumstances has been nothing but dramatic in the second half of this century. In contrast, the energy expenditure of leisure time physical activity may have increased slightly but not enough to keep pace with the changes brought about by urbanization and automation.

The availability of relatively inexpensive and highly palatable foods in almost unlimited abundance is undoubtedly contributing to the epidemic as some of the affected individuals eat many times a day and consume large portions (6). The proportion of calories derived from fats is also potentially involved (1,5), particularly in those who consume a high-fat diet while living a sedentary life (16) although the exact contribution of a high-fat diet to the current obesity epidemic remains controversial (15,18).

The increase of the last decades in the prevalence of overweight and obesity is thought to result from the following circumstances: a) a large proportion of the population is consuming more calories than individuals of past generations with no change in habitual daily energy expenditure; b) for a large number of people, there is an abnormally low daily energy expenditure for a normal caloric intake; or c) for others, caloric intake per capita is actually lower than expected in comparison with previous generations but daily energy expenditure is, on the average, even lower (3). In all three scenarios, energy expenditure of physical activity is a major determinant.

There are several prospective studies that have demonstrated the presence of a significant and inverse relationship between the level of habitual physical activity and weight gain over a number of year. It is not infrequent in these studies to observe that the level of physical activity is a better predictor of weight gain than estimates of caloric or fat intake. One can hypothesize that the contribution of a diminished energy expenditure to the current overweight and obesity epidemic is determined by the decrease in the level of habitual physical activity associated with work and chores of daily living and by the growing amount of time spent in a very sedentary mode, such as watching TV, working on the computer, playing video games, etc. It is not associated with decreases in resting metabolic rate or in dietary-induced thermogenesis. Indeed, there is absolutely no indication that there is a downward secular trend for these two components of daily energy expenditure.

Can Overweight and Obesity Be Prevented?

It would seem judicious to consider the prevention of obesity as an important agenda item (11). The rationale supporting the view that a good fraction of the obesity cases could have been prevented is based on the following considerations (2). First, the level of heritability of obesity or body fat content is only moderate. Second, most intermediate phenotypes that can be defined as determinants of body fat content are also characterized by low to moderate levels of heritability. Third, the prevalence of overweight and obesity has been steadily increasing over the last 50

TABLE 2. Evidence categories.

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

From NHLBI Report (10) Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the Evidence Report. *Obes. Res.* 6(Suppl. 2), 1998.

years or so, and population studies in the Western countries seem to indicate that the prevalence is still rising. This increase has occurred over a period of time that is too short to be caused by changes in the frequency of obesity genes or susceptibility alleles.

A more physically active lifestyle is likely to be the cornerstone of a prevention strategy centered on the concept of the promotion of healthy weights (4). Moreover, there are reasons to believe that energy balance will be easier to achieve in the long term if the physically active lifestyle is associated with a moderate level of dietary fat (about 30% of calories). Energy balance, and particularly balance between lipid intake and lipid oxidation, is quite difficult and perhaps impossible to sustain for most people when dietary fat intake is high (16).

Two lines of evidence support the concept that overweight or obesity can be prevented. The first results from the comparison of the Arizona Pima Indians and Mexican Pima Indians living in Maycoba, a poor and remote area of Mexico (7,13). Pima Indians living in Arizona exhibit one of the highest prevalence rates of obesity and Type II diabetes in the world. In contrast, the 208 Mexican Pima Indians measured, among a total population of about 600 according to a recent census, had mean BMI of 25.9 for women (mean age of 34.1 yr) and 23.6 for men (mean age of 39.8 yr) (7), values that are markedly lower than their Arizonian kin. These data are admittedly preliminary, but they strongly suggest that environment and lifestyle have a strong impact on body mass for height.

The second observation comes from the survey of monozygotic twins discordant for BMI. Rönnemaa and collaborators surveyed monozygotic twin pairs of the Finnish Twin Cohort registry (14). A total of 1453 such pairs born between 1932 and 1957 responded to a mail questionnaire in 1990. Among them, 50 pairs were identified as being discordant for BMI. The latter was defined as a BMI difference between the two identical brothers or sisters of at least 4 kg/m^2, with one twin having a BMI of at least 27. Hence, about 3% of this sample of identical twins were markedly discordant for an indicator of overweight or obesity. In a subgroup of 23 of these pairs who were extensively studied under controlled laboratory conditions, the mean body weight difference between the overweight or obese co-twin and the lean brother or sister reached 16 kg in men and 19 kg in women (14). Such data indicate that for the same genetic characteristics, it is possible to remain normal weight or become obese. There is no doubt that dietary and physical activity habits can have a major contribution to body weight regulation over and above those imposed by the genotype.

The tools available to reverse this unhealthy trend are remarkably simple in appearance as they center on the promotion of eating regular and healthy meals, avoiding high caloric density snacks, drinking water instead of energy-containing beverages, keeping dietary fat at about 30% of calories, cutting down on TV viewing time, walking more, participating more in sports and other energy-consuming leisure activities, and other similar measures (2). However, it will be a daunting task to change the course of nations that have progressively become quite comfortable with an effortless lifestyle in which individual consumption is almost unlimited. It will require massive resources and an unprecedented level of cohesiveness among all public health agencies and private organizations to begin reversing the trends that have emerged over the last decades (20). Foreyt and Goodrick (6) have argued that the increase in the prevalence of overweight and obesity appears to be unstoppable as a side effect of modernization.

Physical Activity and the Comorbidities

Because the health consequences of excess body fat do not become immediately manifest, the current epidemic of obesity in children, adolescents, and young adults will translate later in unprecedented prevalence of Type II diabetes, hypertension, cardiovascular disease, gallbladder disease, postmenopausal breast cancers, osteoarthritis at the knees, back pain, and physical and mental disabilities. It has been estimated that Americans alone are currently spending about $100 billion annually as a result of the direct and indirect costs of obesity (19). The health consequences of a situation in which obesity is ever becoming more prevalent will be catastrophic.

It is well established that regular physical activity has favorable effects on several of the comorbidities of obesity, particularly those pertaining to cardiovascular diseases and Type II diabetes. Some data also indicate that mortality rates are lower in the overweight and moderately obese men and women who are physically fit compared with the unfit (9). Thus, regular physical activity plays an important role not only because of its contribution to the regulation of energy balance but also because it reduces the risk of being affected by the comorbidities of obesity and results in lower all-cause and cardiovascular death rates.

CONTENT OF THE SUPPLEMENT

The focus of the February 1999 Conference was on the development of a consensus document concerning the role of physical activity in the prevention and treatment of obesity and its comorbidities and to identify the most important research issues to be addressed. The specific goals were defined as follows: a) to summarize the relevant scientific literature, b) to develop a consensus statement, c) to prepare a series of recommendations for research, and d) to publish a document summarizing the proceedings of the conference.

The various texts published in this supplement of *Medicine and Science in Sports and Exercise* pertain to the realization of these goals. After this introductory chapter, the Evidence Report of the Independent Panel is presented. This consensus statement covers all the topics that were discussed during the conference and identifies a series of research questions to be pursued. This is followed by a series of papers dealing with the magnitude of the overweight and obesity epidemic (K. M. Flegal), the determinants of overweight and obesity (J. O. Hill and E. L. Melanson), the assessment of physical activity level and energy expenditure (K. R. Westerterp), and the current levels of physical activity and of inactivity in children and adults (M.

Pratt, C. A. Macera, and C. Blanton). Subsequently, the papers on physical activity in the prevention and treatment of overweight and obesity are presented: contribution of a sedentary lifestyle (S. A. Jebb and M. S. Moore), prevention (L. DiPietro), treatment in adults (R. R. Wing), treatment in children (L. H. Epstein and G. S. Goldfield), pregnancy (S. Rössner), and menopause (A. Astrup). These papers are followed by others dealing with mobilization of abdominal fat (R. Ross and I. Janssen), regulation of food intake (J. E. Blundell and N. A. King), selection of macronutrients (A. Tremblay and V. Drapeau), and aging (M. J. Toth, T. Beckett, and E. T. Poehlman). Finally, a whole series of papers deal with the role of physical activity in the prevention and treatment of health outcomes in those who are overweight or obese: epidemiological background (J. C. Seidell, T. L. S. Visscher, and R. T. Hoogeveen), comorbidities (F. X. Pi-Sunyer), dislipoproteinemia (M. L. Stefanick), impaired glucose tolerance and diabetes (D. E. Kelley and B. H. Goodpaster), hypertension (R. H. Fagard), thrombogenic factors (R. Rauramaa and S. Väisänen), other morbid conditions and impairments (A. Rissanen and M. Fogelholm), mortality rates (S. N. Blair and S. Brodney), and economic costs (G. A. Colditz).

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AMERICAN COLLEGE of SPORTS MEDICINE$_{SM}$

ROUNDTABLE CONSENSUS STATEMENT

Physical activity in the prevention and treatment of obesity and its comorbidities

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On February 4–6, 1999, the American College of Sports Medicine sponsored a scientific roundtable on the role of physical activity in the prevention and treatment of obesity and its comorbidities. The purpose of the conference was to provide an evidence-based review of the current state of knowledge on physical activity as a modality for coping with the "epidemic" of obesity occurring in the United States and other nations (1,5). Participants of the conference were requested to review existing literature and to classify available data according to accepted evidence-based categories. The categories employed are those outlined in the recent Clinical Guidelines on the Identification, Evaluation, and Treatment of Obesity in Adults reported by a task force of the Obesity Education Initiative (OEI) of the National Heart Lung and Blood Institute (NHLBI) (2) (Table 1).

A particular goal of this roundtable was to review randomized clinical trials (RCT) that provide evidence in Categories A and B. Unfortunately, there are a limited number of RCT of physical activity in obese populations. On the other hand, the field is rich in observational data and studies in human physiology and behavior (Category C). A sizable portion of the Category C evidence derives from large and replicated studies and provides a considerable base upon which recommendations can be made. Such recommendations indeed are set forth in the OEI report (2), the NIH Consensus Conference Statement on Physical Activity and Cardiovascular Health (December 18–20, 1995) (3), and the Report of the Surgeon General on Physical Activity and Health (4). The current roundtable aimed to extend these previous reports by examining prior and new publications in more depth and by categorizing evidence more precisely as to type and strength. The manuscripts upon which presentations and the current panel report are based are being published concurrently in *Medicine and Science in Sports and Exercise*. The glossary of key terms used in the current report are those derived from the Surgeon General's report on physical activity and health (4) (Table 2).

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BACKGROUND

Prevalence and trends in overweight and obesity. The most recent National Health and Nutritional Examination Survey (1988–1994) (NHANES III) (1) revealed that 54.9% of American adults are overweight or obese (see Table 3 for OEI classification of overweight and obesity). Since 1960, overweight and obesity have increased across all ages, genders, and racial/ethnic groups. Prevalence in the obese category has increased by about 10%. Not only has the overall distribution of body mass index (BMI) shifted to higher levels, but the distribution has become even more skewed toward the high end. In the recent survey (1), the highest prevalence of obesity was found among non-Hispanic black women, Mexican-American women, Mexican-American men, and among less well-educated and low-income people.

The recent OEI report (2) emphasized that waist circumference is a good indicator of abdominal obesity, which at BMI levels $\leq 35\ kg \cdot m^{-2}$ is correlated more closely with the comorbidities of overweight than is BMI (see Table 4 for OEI classification of disease risk according to waist circumference). Cross-sectional epidemiological data are not available to define the prevalence of abdominal obesity in the population; neither are data available to correlate waist circumference with comorbidities in the whole population. Such data would add significantly to our understanding of the metabolic consequences of overweight.

Prevalence and trends in physical activity. The 1996 Behavioral Risk Factor Survey (BRFSS) reveals a high prevalence of physical inactivity among American adults and high school students (see Table 5 for the classification of physical activity used in the Surgeon General's report on physical activity). Adolescents are more active

TABLE 1. Categories of evidence.

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

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

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

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

than adults; 64% of high school students report participating in vigorous (hard or very hard) activity for at least 20 min on three or more days per week. Boys engage in physical activity more than girls, and whites in general more than in blacks or Hispanics. Physical activity declines at higher grades in school, especially among girls. Among adults, only 28% of men and women achieve moderate or vigorous levels of physical activity. Further, 27% of men and 31% of women report no regular physical activity outside of work. Educational level is a factor in exercise pattern. Less than 20% of college graduates report being inactive, whereas nearly half the population with a high school education are inactive. Black and Hispanic men and women are less active than white men and women. In spite of long-term societal trends in activity patterns, these have not been assessed quantitatively; moreover, leisure-time activity apparently has not changed much over the recent decades. Future surveys might well examine the impact of changes in activity patterns at work and at home, and in active and passive leisure time activities (i.e., less active recreation during and after school and more television viewing, computer work, and playing video games).

The economic costs of obesity and inactivity. The medical costs of obesity and physical inactivity can be estimated from the strengths of their associations with various diseases—coronary heart disease, diabetes, gallstone disease—and comorbid risk factors. The direct costs of a lack of physical activity, defined conservatively as absence of leisure-time physical activity, are approximately 24 billion dollars, or 2.4% of the U.S. health care expenditures. Direct costs for obesity defined as a BMI greater than 30 $kg \cdot m^{-2}$, in 1995 dollars, totals 70 billion dollars. The costs of inactivity are independent of obesity, and the costs of obesity are independent of those due to a lack of physical activity. Overall, the direct costs of inactivity and obesity are estimated to consume some 9.4% of national health care expenditures in the United States.

Determinants of overweight and obesity. The high and increasing prevalence of overweight and obesity must be due in large part to environmental factors. Development of obesity requires that energy intake exceed energy expenditure; maintenance of obesity demands a higher energy input or a lower energy expenditure, or both, than needed for a healthier weight. Factors affecting both intake and expenditure of energy probably play a role in the causation and maintenance of obesity. Societal trends go against reducing energy intake *as well as* against increasing energy expenditure. A relatively high intake of energy is driven by a food supply that contains readily available, energy-dense foods, served in large portions. Energy expenditure is lowered by progressively lesser amounts of physical activity required at work and at leisure. Most energy expenditure is obligatory, determined by resting metabolic rate and the thermic response to food intake, but physical activity must not be discounted in the equation for total energy expenditure. Even in the absence of occupations or recreations that consume large amounts of energy, regular physical activity offers a means to lessen the severity of overweight and obesity in the population. A sustained increment in energy expenditure of 200 $kcal \cdot d^{-1}$ through increased physical activity would reduce body weight by about 5 kg over a period of 6 months to 1 yr, assuming no increase in food consumption. Unfortunately, any weight loss achieved by moderate physical activity can be easily reversed by small compensatory increases in food intake.

Measurements of body habitus and energy parameters. If the role of physical activity in the causation or treatment of obesity is to be placed on a quantitative basis, accurate measurements must be made of body composition, energy intake and expenditure, and levels of physical activity. Methods for measurements in each of these areas are improving but still have significant limitations. Significant strides have been made in measurements of body composition—total body fat, body fat distribution, lean body mass, and muscle mass. Several of these techniques are increasingly being used in epidemiological studies, but the most sophisticated measurements can be costly. Despite the advantage of being safe enough for use in children and pregnancy, the latter are too laborious and costly for use in

TABLE 2. Glossary of terms.

Aerobic training—Training that improves the efficiency of the aerobic energy-producing systems and that can improve cardiorespiratory endurance.

Cardiorespiratory endurance (cardiorespiratory fitness)—A health-related component of physical fitness that relates to the ability of the circulatory and respiratory systems to supply oxygen during sustained physical activity.

Exercise (exercise training)—Planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness.

Maximal heart rate reserve—The difference between maximum heart rate and resting heart rate.

Maximal oxygen uptake ($\dot{V}O_{2max}$)—The maximal capacity for oxygen consumption by the body during maximal exertion. It is also known as aerobic power, maximal oxygen consumption, and cardiorespiratory endurance capacity.

Maximal heart rate (HR_{max})—The highest heart rate value attainable during an all-out effort to the point of exhaustion.

Metabolic equivalent (MET)—A unit used to estimate the metabolic cost (oxygen consumption) of physical activity. One MET equals the resting metabolic rate of approximately 3.5 mL $O_2 \cdot kg^{-1} \cdot min^{-1}$.

Physical activity—Bodily movement that is produced by the contraction of skeletal muscle and that substantially increases energy expenditure.

Physical fitness—A set of attributes that people have or achieve that relates to the ability to perform physical activity.

Resistance training—Training designed to increase strength, power, and muscle endurance.

TABLE 3. Classification of overweight and obesity by BMI.

	BMI ($kg \cdot m^{-2}$)	Obesity Class
Underweight	<18.5	
Normal	18.5–24.9	
Overweight	25.0–29.9	
Obesity	30.0–34.9	I
	35.0–39.9	II
Extreme obesity	≥40	III

large populations. Methods for measurement of nutrient and energy intake leave much to be desired. Questionnaires and diaries of food intake are plagued by under reporting. Precise methods for estimating energy intake, as would be required to define differences in intake responsible for weight gain, are not available.

The most objective and accurate method for assessing the level of physical activity and energy expenditure of activity is average daily metabolic rate, determined by the doubly labeled water, minus the basal metabolic rate. This method, however, is limited to studies in small numbers of subjects. More applicable to population studies are motion sensors, specifically, accelerometers. These instruments reliably assess patterns of physical activity. Accelerometers offer the advantage of measuring motion from nonexercise (i.e., lifestyle) activity as well as exercise activity.

Another way to estimate "integrated" physical activity is to measure cardiorespiratory fitness. This measure is represented by maximal oxygen consumption determined under exercise conditions. This measurement carries the advantage of being quantitative and available for epidemiological studies. It is not, however, a direct measure of physical activity but only a reflection of it. Moreover, cardiorespiratory fitness is largely an indication of recent intensive exercise; it may not be a useful tool to assess the effects of moderate exercise, and it is also influenced by inherited characteristics.

PHYSICAL ACTIVITY IN THE ETIOLOGY AND TREATMENT OF OBESITY

Sedentary life habits and inactivity in the etiology of overweight and obesity. No RCT are available that address whether sedentary life habits and inactivity contribute to the development of obesity in populations. Available data therefore are restricted to observational studies (Evidence Category C). Studies of ecological trends in populations provide suggestive evidence that declining amounts of physical activity have contributed importantly to a rising prevalence of overweight and obesity. Some studies have

TABLE 4. Disease risk* relative to normal weight and waist circumference.[a]

BMI ($kg \cdot m^{-2}$)	Men ≤102 cm (≤40 in) Women ≤88 cm (≤35 in)	>102 cm (>40 in) >88 cm (>35 in)
25–29.9[b]	Increased	High
30–34.9	High	Very high
35–39.9	Very high	Very high
≥40	Extremely high	Extremely high

[a] Disease risk for Type II diabetes, hypertension, and cardiovascular disease.
[b] Increased waist circumference can denote increased disease risk even in persons with normal weight.

TABLE 5. Classification of physical activity intensity, based on physical activity lasting up to 60 min.

	Relative Intensity		
Intensity	$\dot{V}O_{2max}$ Heart Rate Reserve (%)	Maximal Heart Rate (%)	RPE[a]
Very light	<25	<30	<9
Light	25–44	30–49	9–10
Moderate	45–59	50–69	11–12
Hard	60–84	70–89	13–16
Very hard	≥85	≥90	>16
Maximal	100	100	20

[a] Borg rating of Relative Perceived Exertion 6–20 scale (Borg, G. A. Psychophysical bases of perceived exertion. *Med. Sci. Sports Exerc.* 14:377–381, 1982.)
This table was modified from one in the Surgeon General's Report on Physical Activity and Health (4) Physical activity and health: a report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, 1996.

reported secular decreases in energy intake concurrently with increases in body weight, both in children and adults; these decreases infer a corresponding even greater decrease in energy expenditure, which would be a requirement for weight gain.

Two cross-sectional studies, each on about 700 children, noted lower levels of activity being correlated with higher levels of body fatness. In these studies, confounding variables could not be fully excluded. Children of parents with less formal education and lesser incomes had greater body fatness that was associated with more watching of television and other sedentary activities. In another cross-sectional study, Minnesota researchers used questionnaires to obtain information about food intake and exercise frequency on large samples of women and men. Body weight correlated inversely with high intensity exercise in men and with both high intensity exercise and walking in women. At the same time, high consumption of certain foods and drinks—alcohol, dairy products, and meat—were positively associated with body weight. This study supported a dual etiology for increased body weight.

A review of eight prospective studies revealed some link between low levels of physical activity and risk of developing obesity. In one study in U.S. male health professionals, those reporting vigorous exercise had lower body weights at follow-up. There was a direct association between television viewing habits and body weight. Relationships were more pronounced in middle-aged men than elderly men. In the Women's Gothenburg study, low levels of leisure-time physical activity appeared to be a risk factor for weight gain in women consuming high-fat diets. Taken together, the various observational studies support an inverse relationship between the level of physical activity and body fatness. This support resides in Evidence Category C, and it can be called only moderately strong—the data suffer from a lack of precision in measurements of energy intake and total physical activity.

Prevention of weight gain through physical activity. RCT are not available to address whether physical activity can prevent weight gain in the general population. Data are restricted to observational studies (Evidence Cat-

egory C). The age-related pattern of body weight demonstrates an increasing prevalence of weight gain through the sixth decade. Several large-scale longitudinal studies have assessed the role of habitual physical activity in preventing the progression of weight gain from acceptable weight through overweight to obesity, along with its attendant comorbidities. These studies suggest that physical activity and fitness attenuate weight gain but do not necessarily prevent weight gain or promote weight loss. Fitness (and maintaining fitness) further appears to protect against sizable weight gain. Although body weight increases with advancing age, even among the most fit and highly active, this increase may be attenuated through physical activity. Avoiding a sizable weight gain with aging through physical activity nevertheless may mitigate comorbidities of obesity. A considerable body of data exists that physical activity can attenuate weight gain with aging with attendant benefits in reduction of comorbidities (Evidence Category C).

Treatment of overweight and obesity in adults. Ten RCT have addressed whether exercise interventions alone will produce weight loss. The majority of these studies show that physical activity alone in the form of aerobic exercise produces some weight loss. The effect of exercise alone on weight loss nonetheless appears to be modest—generally about 1–2 kg over the duration of study. Evidence from this group of RCT was not considered robust enough for Evidence Category A. Several studies were relatively small, and on the whole, observed weight changes were modest, although consistent. The panel therefore assigned the summed results to Evidence Category B.

Another 13 RCT examined whether the combination of reduced-energy diets and increased physical activity produce a greater weight loss than does a low-energy diet alone. Most studies favored diet + exercise regimens over diet alone, but the difference was statistically significant in only a minority of studies. In one of the latter, diet + exercise proved better in men, but not in women. The panel concluded that the overall lack of statistical significance was probably due to the short time-frame of the exercise programs, small sample sizes with inadequate statistical power, and difficulty with adherence to exercise. These limitations in design placed the summed results of this group of RCT in Evidence Category B.

A few of the prior RCT were extended to examine whether a regimen of diet + exercise produces better maintenance of weight loss than does diet alone. Efficacy data from RCT in free-living adults are limited by small sample sizes and by poor long-term adherence to the recommended exercise and diets. Of six RCT that bear on the question, two showed significant long-term effects favoring diet + exercise over diet alone; the others were inconclusive. The relatively strong trends of the two positive studies lead the panel to conclude that diet + exercise provides benefit over diet alone in maintaining weight loss (Evidence Category B). This conclusion is bolstered by correlational analyses as well as studies of successful weight losers, which consistently show that physical activity is strongly associated with better long-term maintenance of weight loss (Evidence Category C).

Treatment of obesity in children and adolescents. Obesity acquired in childhood appears to be predictive of adult obesity; therefore, prevention of obesity in childhood and adolescence is needed. Six RCT have tested whether low energy diets + exercise produce better weight loss than diet alone in children. The RCT were relatively small and were not powered to provide a definitive result. Taken together the RCT demonstrated that exercise incrementally adds to low-energy diets for short-term changes in percent overweight or percent body fat (Evidence Category B). The evidence further suggested to the panel that whereas exercise in overweight children and adolescents has limited effects on percentage overweight, it still improves body composition. The resulting fitness favorably modifies cardiovascular risk factors (Evidence Category D).

Physical activity for prevention and treatment of weight gain in pregnancy. RCT were not uncovered that tested whether increased physical activity prevents or attenuates excessive weight gain during pregnancy. Few observational studies of exercise in pregnancy have examined maternal weight gain as an outcome. One prospective study compared women who chose to remain physically active during their pregnancies with women who chose to terminate their exercise habits. During the third trimester, exercising women showed reduced rates of weight gain and significantly smaller increases in fat accumulation (as measured by the sum of five skinfolds) compared with nonexercising women. Similarly, according to an observational study, women who had lower levels of leisure-time physical activity retained more of their pregnancy-associated weight gain than those who exercised. These observational studies were considered to be suggestive of benefit of exercise for weight control both during pregnancy and afterward and were assigned an Evidence Category C. The panel concluded that the development of physical activity interventions in pregnant women represents a potentially fruitful avenue for the prevention of overweight and obesity in women (Evidence Category D).

Physical activity and changes in body weight and fat distribution at menopause. Weight gain during and after menopause contributes to overweight and obesity in older women. Three RCT have compared an exercise regimen with no intervention and another three RCT have compared diet + exercise with diet alone on body weight and fat distribution in postmenopausal women. The results of these RCT do not allow a firm conclusion as to whether physical activity will prevent or limit a gain in total body fat or abdominal fat after the menopause, or whether exercise provides incremental benefit over diet alone in an obesity treatment program for postmenopausal women. The available RCT were deemed to be insufficiently powered to adequately address this question. On the other hand, observational studies, both cross-sectional and longitudinal, suggest that postmenopausal women with high levels of physical activity have lower body fat and less abdominal fat during the menopause than do those with low levels of physical activity; exercisers moreover are less likely to gain in total body fat and abdominal fat (Evidence Category C).

Older populations. Several interventional studies have examined whether exercise in the older population can slow down (or reverse) progressive depletion of muscle and accumulation of fat that occurs with advancing age. The panel considered intervention studies of at least 2 months' duration and that included exercise, but not diet. Patients ranged in age from 55 to 86 yr and underwent either resistance training or aerobic training. Randomization occurred in about one-fourth of studies with aerobic training and in about half with resistance training. Aerobic training reportedly reduced fat mass without changing fat-free mass; the magnitude of loss of fat mass depended on duration of the trial. Resistance training reduced fat mass and increased fat-free mass, unrelated to the duration of the exercise program. The panel concluded that resistance training reduces fat mass and increases muscle mass in persons over age 55, whereas aerobic training reduces fat mass but does not increase muscle mass (Evidence Category B).

Physical activity and abdominal obesity. Five RCT examined whether increased physical activity alone, without diet-induced weight loss, will reduce body weight and abdominal girth. In four of five RCT, addition of exercise without dietary change failed to reduce body weight, body fat, or abdominal obesity. In only one RCT did exercise alone produce significant weight loss and decrease in abdominal circumference. In nine nonrandomized trials, the same question produced similar trends: exercise alone produced little or no change in body weight, body fat, or abdominal circumference. However, these 14 studies were not designed to determine whether there was a preferential reduction in abdominal fat. When visceral and subcutaneous abdominal fat are measured outcomes, exercise has been demonstrated in one randomized trial and four nonrandomized trials to result in reductions in both compartments. The panel concluded that increased physical activity alone without weight loss is not associated with reductions in abdominal girth. Physical activity with or without weight loss is associated with reductions in visceral and abdominal subcutaneous tissue (Evidence Category C).

OVERWEIGHT, OBESITY, AND COMORBID CONDITIONS

Obesity-associated mortality rates. Prospective studies show the relationship between BMI and total mortality to be J-shaped, with minimal mortality occurring in the BMI range of 18.5–24 $kg \cdot m^{-2}$. Smoking along with overt and subclinical illnesses partially confounds the increased mortality at the lowest BMI. By excluding deaths in the first 5 yr and by confining the analysis to healthy nonsmokers, the studies reveal a linear relationship with mortality increasing progressively from lowest to highest levels of BMI. Multiple factors influence mortality rates and may confound the relationship between physical activity and reductions in premature mortality (Evidence Category C). This category of evidence is strengthened by a study in twins in which leisure-time activity correlated positively with lower mortality, after controlling for genetic and familial factors. These various studies still may not adequately control for life habits, risk factors, and socioeconomic status, all of which are correlated with BMI and mortality rates. Causes of death and life expectancy moreover have changed over time; birth cohorts have been exposed to different environmental factors that further complicate the interpretation of data. These confounding factors however almost certainly do not negate the causal relationship between BMI and mortality, which is linear and strong (Evidence Category C).

Obesity-associated comorbidities. Strong epidemiological and metabolic data demonstrate that obesity contributes to a number of medical conditions: insulin resistance, glucose intolerance, diabetes mellitus, hypertension, dyslipidemia, sleep apnea, arthritis, hyperuricemia, gallbladder disease, and certain types of cancer. This evidence, which belongs to Category C but is overwhelmingly strong, has been reviewed in detailed in the recent NHLBI OEI report (2). The contribution of obesity to several cardiovascular endpoints—coronary heart disease, heart failure, cardiac arrhythmia, and stroke—appears to be *independent* of the influence of obesity on known risk factors. Many epidemiological and metabolic studies confirm an influence of obesity on risk factors related to insulin resistance and upon the development of Type II diabetes. This influence is augmented when the excess body fat is distributed predominantly to the abdomen and trunk (Evidence Category C). In spite of the strong associations between obesity and its comorbidities, much remains to be learned about underlying mechanisms and quantitative aspects of these associations.

Exercise and insulin resistance, impaired glucose tolerance, and diabetes. Intervention-based RCT consistently show that increased physical activity improves insulin action, and thus reduces insulin resistance, in obese subjects (Evidence Category A). Epidemiological data suggest that exercise alone and exercise combined with weight reduction retard the transition from impaired glucose tolerance to Type II diabetes (Evidence Category C). For treatment of hyperglycemia in patients with diabetes, intervention studies on the value of exercise are mixed. Although exercise enhances insulin sensitivity, many patients with diabetes cannot engage in high-intensity exercise. Further, when defects in insulin secretion are severe, the treatment of hyperglycemia shifts toward providing more insulin to overcome insulin deficiency. Of note, any benefits from exercise bouts are short lived; therefore, if exercise is to be an effective adjunct in the long-term control of hyperglycemia, it must be regular and sustained.

Dyslipoproteinemias. Observational studies reveal that the major lipoprotein abnormality accompanying obesity is atherogenic dyslipidemia (elevated triglycerides, increased small low density lipoprotein (LDL), and low high density lipoprotein (HDL) levels). An elevated serum LDL cholesterol, when it occurs in overweight persons, appears to be related more to high intakes of cholesterol and cholesterol-raising fatty acids (saturated and *trans* fatty acids) than to overweight *per se*. Beyond observational studies, 11 RCT of 5–12 months' duration and of large enough sample

size to detect significant changes in HDL-C (and possibly other lipoproteins) have been carried out. These studies indicated that in the absence of weight loss exceeding at least 2.5 kg, an increased physical activity rarely raises HDL cholesterol or lowers triglycerides in overweight women (pre- or post-menopausal) or men. However, if physical activity is of a volume that results in at least 4.5-kg weight loss, HDL cholesterol will be raised, and triglycerides lowered, in men and postmenopausal women. Further, the addition of physical activity to a low-energy, low-fat diet will reverse the HDL-lowering effect of the low-fat diet in overweight men and women. Finally, in men and postmenopausal women with atherogenic dyslipidemia, the addition of physical activity to a modestly weight reducing, low-fat diet significantly enhances the LDL-lowering effect of the diet. The panel considered the RCT evidence for these conclusions to be strong (Evidence Category A).

Hypertension. A review of 44 RCT, involving 68 study groups and including 2674 participants, revealed that dynamic aerobic training reduces blood pressure by −3.4/−2.4 mm Hg ($P < 0.001$), after controlling for weight loss and dietary change and after weighting for the number of participants in each group. Blood pressure was reduced significantly more in groups of hypertension patients (−7.4/−5.8 mm Hg) than in normotensive study groups (−2.6/−1.8 mm Hg). Decreases in blood pressure were independent of weight loss and were not related to initial body mass index. Finally, eight RCT indicated that exercise alone is less effective for reducing blood pressure than diet alone (−3.5/−2.9 vs −5.4/3.7 mm Hg, respectively, $P < 0.02$); moreover, adding exercise to a low-energy diet seemingly does not reduce blood pressure significantly more than does diet alone. The panel concluded that RCT provide strong evidence that physical activity can reduce blood pressure in both lean and obese subjects. Furthermore, blood pressure reduction is independent of weight loss or changes in body composition, although the response does not go beyond the blood pressure reduction obtained by diet alone (Evidence Category A).

Cross-sectional surveys further suggest an inverse relationship between habitual physical activity or measured physical fitness and blood pressure. Three prospective studies agree that physically active subjects have both lower blood pressure and a reduced incidence of hypertension; these beneficial effects appears to be independent of a number of confounding factors including BMI or body fat. In one study, Harvard alumni who did not engage in vigorous play at sports in postcollege years displayed a greater risk for developing hypertension than did those who did play; this difference was observed only in those who were overweight at baseline. The panel agreed that observational studies support the concept that physical activity reduces the risk for developing hypertension, independent on its effects on body size or body composition (Evidence Category C).

Thrombogenesis. A prothrombic state is one component of the metabolic syndrome that has been reported to occur in obese patients. The effects of physical activity on the coagulation system are complex and not well understood. Physical activity has an acute effect of promoting thrombosis and activating fibrinolysis. This effect might explain the acute coronary events that occasionally occur during intense exercise. Limited data further suggest an effect of physical activity on coagulation factors over a longer term. For example, cross-sectional studies suggest an inverse relationship between levels of physical activity and plasma fibrinogen levels. Still, intervention RCT and several noncontrolled studies remain contradictory, whether the endpoint is fibrinogen, plasminogen activator inhibitor, tissue plasminogen activator, or other coagulation factors. Taken as a whole, observational studies are modestly suggestive that regular physical activity is antithrombogenic (Evidence Category C). Limited RCT data further suggest that moderate-intensity physical activity carried out on a regular basis reduces platelet aggregation (Evidence Category B).

Cancer. Many observational studies have found an association between obesity and some forms of cancer, notably, cancers of the colon, breast, endometrium, and possibly the prostate gland. Whether obesity plays a causative role in the development of these cancers is uncertain. Epidemiological studies also find a strong inverse relationship between physical activity and colon cancer. The strength and consistency of this association is so strong that a causal connection seems likely (Evidence Category C).

Prospective studies of physical activity and morbidity/mortality in overweight or obese individuals. Among over 700 articles related to this topic, 25 were selected as providing data on the relation of physical activity to morbidity and mortality in strata of overweight and obese individuals. One question addressed was whether higher levels of physical activity or cardiovascular fitness reduces morbidity, mortality, and disability in overweight or obese persons. The panel discerned that active and fit individuals appear to have lower rates of disease and death than inactive persons who are not physically fit. This worsening gradient was present in various strata of body habitus. A second question was whether overweight and obese persons who are physically active have a lower risk of morbidity, mortality, and disability than normal weight individuals who are sedentary. The data indicated that being physically active and fit reduces obesity-related chronic diseases and decreases risk for early death and that active and fit persons who are overweight or obese actually have lower morbidity and mortality risk than normal weight persons who are sedentary. The evidence related to these questions was considered to be moderately strong and was classified under Evidence Category C.

CONCLUSIONS

The prevalence of overweight and obesity is alarmingly high and is increasing in the United States and many other nations (5). These conditions impose a heavy burden of morbidity and premature mortality as well as financial costs. Overweight and obesity are due to a relatively high intake of energy that is not matched with appropriate energy expen-

diture. One component of energy expenditure comes from exercise and other forms of physical activity. That most people in our society live sedentary lives is obvious to the casual observer and is a conclusion supported by observational studies. On the other hand, the solution to the problems of overweight and obesity cannot come exclusively from the institution of regimens of moderate exercise in the general population. Increments in energy expenditure brought about by moderate exercise are insufficient to completely forestall weight gain with advancing age or to reverse higher levels of overweight and obesity. Further, current social structures are not conducive to marked increments in energy expenditure through increased physical activity. Thus, curtailment of energy intake and efforts to increase physical activity will both be necessary to cope with the "epidemic" of overweight and obesity.

Nonetheless, a large body of evidence has been acquired to support the need to include increased physical activity in a program of weight control. Some of this evidence comes from RCT, although there have been too few such studies in the exercise field. Many observational studies, however, lend strong support to a place for regular physical activity in weight control. They suggest that a decline in physical activities in both occupational and leisure-time settings contributes to increasing overweight and obesity and that a restructuring of social conditions to allow and encourage physical activity would help to reverse current trends. Several RCT further indicate that combining a physical activity regimen with low-energy diets facilitates weight loss and maintenance of lower body weights. Finally, increased physical activity appears to have an independent beneficial effect on several comorbidities of obesity, notably insulin resistance, hyperglycemia, dyslipidemia, and, possibly, a prothrombic state. In addition, an active and fit way of life appears to attenuate morbidity and mortality risk in overweight and obese individuals.

Research needs. Continued monitoring of patterns and trends in physical activity is needed for representative samples of the population. Improved methods of measurement appropriate for population-based observational studies are also required. Increasingly evidence-based recommendations require RCT to bolster the results of observational studies. Various epidemiological studies—cross-sectional, case-control, and prospective—may uncover new relationships and correlations. These can be extended by in-depth investigations of human metabolism and physiology. Nonetheless, in some crucial areas, causal relationships and clinical benefit of therapeutic modalities can be established only through RCT. The fields of physical activity and exercise physiology are rich in observational studies and research in human metabolism and physiology. On the other hand, they are lacking in RCT, although in recent years, an impressive number of RCT are being reported. It nonetheless can be said that one of the major needs in the field of physical activity is for more and better-designed RCT of physical activity in overweight and obese groups.

If results of RCT on the benefits of physical activity are to be convincing to the medical community, they must be based on sound methodology. They must have sufficient sample size, duration, and statistical power to yield a definitive result. The volume of exercise in the "treatment arm" of the study must be high enough to significantly affect metabolism and physiology. Adherence to the exercise intervention must be documented. The endpoints of the study should be clearly defined and be expressed in quantitative terms. Among endpoints, effects of intervention on magnitude of weight loss versus maintenance of weight loss should be differentiated. The experimental groups should contain both men and women; many previous trials appear to have been lacking in women as subjects. New RCT should be designed to test both efficacy (dose-response) and effectiveness (based on intention to treat) of the intervention. Development of appropriate control groups is mandatory; for example, consideration might be given to comparing different intensities or types of physical activity, rather than a simple comparison of exercise versus sedentary groups.

If RCT designed to evaluate the impact of physical activity on body weight are to be meaningful, quantitative methodology will be required. A great need is for the ability to measure total, 24-h physical activity. Current methodology is inadequate, although accelerometer technology is promising. The use of "physical fitness" as a surrogate for integrated physical activity needs more validation; of course, physical fitness may be an independent parameter related to health outcomes. The field of physical activity shares with the nutrition field the need for accurate measurements of energy consumption. The doubly labeled water technique is the "gold standard" for estimating energy (and intake in a weight-stable condition) but is impractical for use in epidemiological studies. Newer quantitative methodology is needed. Thus, if RCT are to take their rightful place in the physical-activity field, sound methodology for quantitative measurement is a necessary underpinning. Improvements in methodology will allow a more accurate assessment of the potential benefits of physical activity in the treatment of obesity and its comorbidities.

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The obesity epidemic in children and adults: current evidence and research issues

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ABSTRACT

FLEGAL. K. M. The obesity epidemic in children and adults: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S509–S514, 1999. **Purpose:** The term "epidemic" of obesity implies that obesity is a characteristic of populations, not only of individuals. The purpose of this paper is to review evidence on obesity in populations and to identify future research issues. **Methods:** To examine recent increases in the population prevalence of overweight or obesity, a literature search was undertaken. **Results:** Trends in overweight or obesity among adults showed considerable variability internationally. Some countries, including Canada, Finland (men), New Zealand, the United Kingdom, the United States, and Western Samoa showed large increases in prevalence (>5 percentage points), whereas several other countries showed smaller or no increases. Overweight is also increasing among children and adolescents, at least in some countries. It is not clear what the expected prevalence of overweight or obesity might be in the current environment, and these findings may be most usefully viewed as shifts in the distribution of a population characteristic. The reasons for these shifts are not clear. The health implications of these shifts are also not clear, in part because trends in cardiovascular risk factors do not always parallel trends in obesity. Of the classic epidemiologic triad of host, agent, and environment, the environment has often received the least attention. **Conclusions:** The economic, social, and cultural factors that influence the distribution of body mass index in a population are not well understood. Future research needs include continued monitoring of trends in obesity and in related health conditions and observational studies to examine the causes of these trends. Public health research should aim at defining realistic goals and strategies to improve health in an environment conducive to high levels of overweight and obesity. **Key Words:** BODY WEIGHT, BODY MASS INDEX, OVERWEIGHT, OBESITY, TRENDS, HEALTH SURVEYS

Obesity is often studied as a characteristic of an individual and discussed in relation to individual dietary patterns, physical activity levels, or genetic constitution. However, obesity can also be considered as a characteristic of populations, as well as of individuals. The term "epidemic" of obesity implies that we need to examine obesity as a population level characteristic and to develop an understanding of obesity from a population perspective.

What is an epidemic and how can we judge whether there is an epidemic of obesity? The word epidemic can be used generally to mean simply a large number of cases or more precisely to mean an increase in the number of cases over past experience or normal expectancy for a given population, time, and place (20). The term comes from the study of infectious disease but has been applied to describe issues as diverse as tobacco smoking and violence, as well as obesity (11,28).

The distribution of body mass index (BMI), like the distribution of other biological characteristics, shows a considerable degree of natural variability. Terms such as "overweight" or "obese" are applied to certain specified levels of BMI. Although many definitions have been used, the most practical current definitions of overweight and obesity for international use in adults are those of the World Health Organization (WHO), with overweight defined as a BMI of 25 or greater and obesity defined as a BMI of 30 or greater (47).

In the United States, a series of national surveys dating from 1960 includes measured height and weight for adults and children (8,43). For both children and adults, data from these surveys showed that the prevalence of overweight and obesity had increased markedly in the United States by the most recent (1988–94) survey. The age-adjusted prevalence of overweight, defined as a BMI of 25 or greater, has been high among adults at least since 1960–62, when 48% of adult men and 39% of adult women were overweight (BMI $\geq$ 25). By the 1988–94 survey, the age-adjusted prevalence of overweight had increased to 59% for men and 50% for women. The prevalence of obesity, defined as a BMI of 30 or greater, was lower than the prevalence of overweight but also showed an increase. The age-adjusted prevalence of obesity (BMI $\geq$ 30) in 1960–62 was 10% for men and 15% for women, increasing to 20% for men and 25% for women by the 1988–94 survey.

Definitions for the prevalence of overweight or obesity in children are less standardized than those for adults. Esti-

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TABLE 1. Trends in the prevalence of overweight or obesity for children and adults.

Country	Reference	Ages (yr)	Study years	Findings
Australia	Bennett and Magnus (1)	25–64	1980, 1983, 1989	BMI > 30 increased from 9.3 to 11.5% for men, from 8.0 to 13.2% for women
Brazil	Monteiro et al. (25)	1–4	1974–75, 1989	Overweight (2 *Z*-score units above NCHS ref) decreased from 4.7 to 3.8% for boys and increased from 4.6 to 5.3% for girls
Brazil	Sichieri et al. (35)	18 +	1974–75, 1985	BMI ≥ 30 increased from 2.5 to 4.8% for men and 6.9 to 11.7% for women
Canada	Millar and Stephens (24)	25–64	1985, 1991	Self-reported BMI > 27 increased from 20 to 30% for men, from 14 to 20% for women
China	Popkin et al. (30)	20–45	1989, 1991	BMI ≥ 27 increased from 1.7 to 2.7% for men and remained at 4.3% for women
Denmark	Sorensen et al. (40)	18–26	1955, 1965, 1973–75	BMI ≥ 30 increased from 1.5 to 4.6% for young men eligible for military service
Finland	Pietinen et al. (29)	30–59	1972, 1977, 1982, 1987, 1992	BMI > 30 for men increased from 11 to 21% (N. Karelia) and from 11 to 20% (Kuopio); for women, declined from 22 to 18% (N. Karelia) or remained at 22% (Kuopio)
Germany	Hoffmeister et al. (17)	25–69	1985, 1988, 1990	BMI ≥ 30 increased from 15.1 to 17.2% for men and from 16.5 to 19.3% for women
Israel	Gofin et al. (12)	50 +	1970, 1986	BMI ≥ 30 increased from 11.9 to 15.8% for men and from 28.6 to 32.6% for women
Mauritius	Hodge et al. (15)	25–74	1987, 1992	BMI > 30 increased from 3.4 to 5.3% for men and from 10.4 to 15.1% for women
Netherlands	Seidell et al. (33)	20–59	1987–1991	BMI ≥ 30 increased by 1.7 for men and 1.9% for women (adjusted for age and education)
New Zealand	Simmons et al. (36)	35–64	1982, 1987, 1994	BMI > 25 increased from 52.8 to 64.2% (men) and 36.5 to 44.9% (women)
Singapore	Lim et al. (23)	18	1967–91	BMI ≥ 25 increased from 1.4 to 12.1% for male inductees
Sweden	Kuskowska-Wolk and Bergstrom (21, 22)	16–84	1980–81, 1988–89	Self-reported BMI > 30 increased from 4.9 to 5.3% for men; self-reported BMI > 28.6 increased from 8.7 to 9.1% for women
United Kingdom	Offer (26)	16–64	1980, 1995	BMI ≥ 30 increased from 6 to 15% for men and from 8 to 17% for women
United States	Troiano and Flegal (43)	6–17	1963–65, 1966–70, 1971–74, 1976–80, 1988–94	Overweight (BMI > 95th percentile) increased from 3.9 to 11.4% for boys and from 4.3 to 9.9% for girls, ages 6–11 yr; increased for ages 12–17 yr from 4.6 to 11.4% for boys and from 4.5 to 9.9% for girls
United States	Flegal et al. (8)	20–74	1960–62, 1971–74, 1976–80, 1988–94	BMI ≥ 30 increased from 10.4 to 19.9% for men and from 15.1 to 24.9% for women
Western Samoa	Hodge et al. (16)	25–74	1978, 1991	Prevalence of BMI > 30 increased from 37.7 to 56.9% for men and from 58.5 to 74.3% for women in Apia; similar increases in other areas

mates of the prevalence of overweight for children from the most recent national survey in the United States, all based on the same data set, range widely, from 11 to 24%, depending on the definitions used (43,44). Using the provisional 95th percentile of BMI from the revised United States growth charts as a definition of overweight, the age-adjusted prevalence of overweight was 4% for both boys and girls ages 6–11 yr in 1963–65 and 5% for both boys and girls ages 12–17 yr in 1966–70. By 1988–94, the prevalence had increased to 11% among boys and 10% among girls in both age ranges.

To examine the current trends in overweight and obesity in more detail, a MEDLINE search for the years 1989–98 was undertaken to identify English language publications that might provide appropriate data for comparison. Additional references were gathered based on further reading. Studies were selected in which data were gathered through a systematic survey process of a defined area on a national or regional scale, weight and height were measured, and data were published using cutpoints of body mass index at least roughly similar to those used for the WHO classification. Studies from Sweden and Canada were included that used self-reported rather than measured weight or height but otherwise met these criteria. A study from Israel was included because it provided estimates of trends from repeated cross-sectional surveys of a defined area. These studies are tabulated in Table 1. Studies of smaller areas, such as those in the MONICA project, were not included.

To summarize the available information from the studies listed in Table 1 and from data published by the World Health Organization (47), the prevalence of obesity is highest in Western Samoa and other Pacific island populations, intermediate in European countries, and populations such as those in the United States with considerable European admixture and lowest in less developed countries such as Brazil and in Asian countries such as China and Japan. Large recent increases in overweight or obesity for adults (>5 percentage points) have been noted in Canada (based on self-reported data), Finland (men), New Zealand, the United Kingdom, the United States, and Western Samoa (8,24,26,29,36). Smaller increases, less than 5 percentage points, appear to have occurred in several other countries, including Australia, Brazil, China (men), Germany, Israel, Mauritius, the Netherlands, and Sweden (self-reported data) (1,12,15,17,21,22,30,33,35). For women in China and Finland, the prevalence showed no increase and even decreased for women in one area of Finland (29,30). There are fewer studies on trends among children and adolescents. Data on young men of military age (late adolescence and early adulthood) from Denmark and Singapore show considerable

TABLE 2. Effect of changes in smoking on prevalence of overweight or obesity.

Country	Reference	Conclusion
Australia	Boyle et al. (3)	The increase in body weight could not be explained by the decreases in smoking rate
New Zealand	Simmons et al. (36)	Recent trends in smoking cessation explain only a small percentage of the increase in body mass
Sweden	Wolk and Rossner (46)	The reported decrease in smoking only partially explains the BMI increase in the adult population
United States	Flegal et al. (10)	Smoking cessation accounted for only a small part of the recent increase in the prevalence of overweight

recent increases (23,40). Data from the United States suggest large increases among United States children (43), and a news report has suggested similar findings recently for children from the Netherlands (34).

Trend data provide the most convincing suggestion of an epidemic. For example, in the United States, the prevalences of overweight and obesity stayed fairly constant over the 20-yr period from 1960 to 1980, varying by only a few percentage points (8). Between the 1976–80 survey and the 1988–94 survey, however, the prevalence of overweight increased from 46 to 54% and the prevalence of obesity from 14.5 to 22.5%. This sudden increase after 1980 was unexpected.

Although there has been considerable speculation about the reasons for the increases observed in the United States and in some other countries, solid data are lacking. Two possibilities often mentioned are changes in dietary intake and changes in physical activity, both difficult to measure on either an individual or a population level. Prentice and Jebb examined this issue for the United Kingdom and concluded that sedentary lifestyles were at least as important as dietary changes and might represent the dominant factor (31). A study by Simmons and associates identified increased energy consumption as the dominant factor associated with increases in overweight in New Zealand (36). Greksa concluded that both increased energy intake and lower activity levels were factors leading to the increase in obesity in Western Samoa (13). Researchers in several countries have examined the question of the effect of smoking cessation, as tabulated in Table 2 (3,10,36,46) and reached the general conclusion that although smoking affects weight at the individual level, the impact of changes in smoking is insufficient to explain the observed changes in weight in the population.

The use of the term "epidemic" suggests a disease model, which may be considered one of several competing explanatory models for obesity (38). The term epidemic in this and other contexts not related to infectious disease (for example, tobacco or violence) might also be considered more as a rhetorical device, used to express concern and to mobilize for action, than as an exact scientific term. In the United States, the prevalence of overweight (BMI $\geq$ 25) was high in 1988–94 (59% for men and 49% for women) but was also quite high in 1960–62 (48% for men and 39% for women), so this is not a new phenomenon. The prevalence of obesity (BMI $\geq$ 30) was 20% for men and 25% for women in 1988–94 as compared with 10% for men and 15% for women in 1960–62. It is not clear whether these should be considered epidemic levels. To address the question of whether we are experiencing an epidemic of obesity also requires some consideration of what the expected numbers might be. Was there a golden age in which there was the "right" prevalence of obesity? Can obesity be prevented completely? Or is it likely that in any population where there is an adequate food supply there will be some cases of obesity and many cases of overweight? These changes in BMI perhaps should be considered to represent the expected population response to a cheap, abundant, and easily obtained food supply under current social conditions. The increasing prevalence of obesity can usefully be viewed as a shift in the distribution of a population characteristic rather than as an increase in the number of cases (9).

The net health implications of the increases are not completely clear. Three of the surveys in Table 1 included some investigations of trends in cardiovascular disease and risk factors. These surveys generally do not show increases in risk factors such as hypertension or hyperlipidemia that parallel the trends in overweight. In the United States, the prevalence of hypertension and elevated cholesterol appear to have declined over the same period as the increase in obesity (4,14,18). In Australia, cardiovascular mortality fell, average blood pressure levels declined, and the prevalence of hypertension decreased (1). There was no overall favorable trend in lipid results, but total cholesterol levels decreased significantly in younger men and older women. In Germany, cardiovascular mortality also continued a downward trend (17). There was a favorable increase in the high density lipoprotein (HDL)/total cholesterol ratio. Blood pressure showed no significant increases except a rise of 2 mm Hg in systolic blood pressure for women. Smaller studies, such as the Minnesota Heart study in the United States and several local WHO MONICA Project sites, also show that increases in BMI or overweight are only sometimes accompanied by parallel increases in obesity-related risk factors such as hypertension or hyperlipidemia and are sometimes accompanied by declines in these risk factors (6,27,41,45).

In societies such as the United States, the prevalence of such cardiovascular risk factors as hypertension or hyperlipidemia is high even among those who are not overweight or obese, particularly at older ages. For example in the United States in 1988–94, the estimated prevalence of elevated cholesterol (defined as serum cholesterol $>$ 240 $mg \cdot dL^{-1}$ or taking cholesterol-lowering medications) for women with a BMI of 22.5–24.9 was 6% at ages 20–39 yr, 23% at ages 40–59 yr, and 49% at ages 60–79 yr. The corresponding estimates for a BMI of 30.0–32.4 were 11%, 33%, and 45%. These data show that although hypercholesterolemia generally increases with increasing BMI, particularly at younger ages, it is also at high levels at older ages even among those who are not classified as overweight or obese.

Whether or not we regard overweight as epidemic, we can use the epidemiological triad of host, agent, and environment to consider causal factors (20). The agent by definition is energy imbalance, where energy intake exceeds energy expenditure. Although energy balance can be influenced by changes in such factors as resting metabolic rate or lean body mass, the main modifiable factors affecting energy balance are dietary energy intake and energy expended through physical activity. Features of the host also affect obesity, in ways that are not well understood. Experimental studies clearly show differences between individuals in their ability to gain weight under conditions of overfeeding or to lose weight under conditions of underfeeding (2). Some individuals appear to be more susceptible than others.

In classic epidemiological thinking, there is a third entity: the environment that facilitates the epidemic by bringing the susceptible host and the agent together. The environment has to be considered to encompass not just the physical environment, such as the layout of cities, but the environment of economic and social organization and cultural values. Developing a cheap, stable, and adequate food supply has been an important goal of human efforts for millennia. Present-day society is characterized by a highly organized system of food production and distribution. The environment in the United States, Western Europe, and other areas, in which food is widely available and which has reduced malnutrition, hunger, and the fear of starvation, apparently also results in high levels of overweight and obesity in many countries.

It is likely that research could benefit from going beyond a narrowly mechanistic focus on energy intake and physical activity and examining the economic, social, and cultural context more broadly. For example, one of the most consistently documented findings is that obesity varies by socioeconomic status among women (37,39). Many other health conditions, as well as access to health care, have also been observed to vary by socioeconomic, education or income levels (19). Research on variations in obesity by socioeconomic status may benefit from the insights and methodologies used by researchers studying other aspects of the relation of socioeconomic status to health. For another example, both in Europe and the United States, differences in the prevalence of overweight or obesity between countries or between race-ethnic groups are often more pronounced for women than for men (8,32). The reasons for this difference are unclear but are likely to be complex. The work of social scientists, including economic and social historians, sociologists and anthropologists, can help clarify the economic, social and cultural factors that may directly or indirectly affect the distribution of body mass index in a population (5,7,26,42).

RESEARCH NEEDS

Both continued monitoring of trends in overweight and obesity and additional analyses of existing data are important. At present, we do not know what further changes may be in store and whether the observed trends will continue or level off. The United States experience suggests that obesity may increase suddenly even in countries in which the prevalence has been stable over time. Existing and future survey and trend data should be exploited to the fullest extent possible. Many characteristics of the current situation are not well described. Shifts in the whole distribution of BMI should be examined, not just changes in means and prevalences. Systematic detailed comparisons between countries are infrequent but would be useful. More detailed analyses of trends by age, sex, socioeconomic status, and other variables would also be useful.

Monitoring of concurrent trends in mortality, in diseases such as diabetes, in risk factors such as blood cholesterol levels, and in other outcomes such as disability associated with obesity is also important. Clearly, obesity is linked with increased health hazards. However, the relationship of obesity to health may vary over time and between subgroups. The trends in obesity are not clearly associated with parallel increases in cardiovascular risk factors, for example. Research should also be directed at better definitions of obesity-associated risks and a better understanding of ways to identify the population characteristics associated with increased health hazards.

Better understanding of the causes of the observed increases in overweight or obesity in many countries is also an important goal. We know remarkably little about the causes of these increases. By necessity, research is limited to observational studies. However, it may be possible to take advantage of the many natural experiments being conducted all around us, as societies change and modernize. From a population perspective, it may be useful to focus less on the "agents" of diet and physical activity and more on the "environment" of social and economic organization and cultural values. The work of economic and social historians, sociologists, and anthropologists may lead to better understanding of the social forces at work.

Public health research aimed at determining what is realistically achievable on a population basis and at what economic and social cost is essential. Can we specify and defend reasonable goals? What distribution of BMI could be expected under the current circumstances? In the United States, the prevalence of overweight in middle age is greater than 60% in all sex-race-ethnic groups. For example, an estimated 73% of white men ages 50–59 yr are overweight (BMI $\geq$ 25), and 33% are obese (BMI $\geq$ 30). Even 50% of men and women aged 80 yr or above are overweight. The resources and efforts required to reduce these high prevalences appreciably in the future would be considerable, and success is by no means certain. A viable public health strategy should aim at improving health, not only at reducing the prevalence of obesity. Public health research should aim at defining realistic goals and strategies to improve health in an environment conducive to high levels of overweight and obesity.

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Overview of the determinants of overweight and obesity: current evidence and research issues

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ABSTRACT

HILL. J. O., and E. L. MELANSON. Overview of the determinants of overweight and obesity: current evidence and research issues. *Med. Sci. Sports Exerc.,* Vol. 31, No. 11, Suppl., pp. S515–S521, 1999. **Purpose:** The prevalence of obesity has reached epidemic proportions in many countries around the world. However, the genetic and environmental factors contributing to obesity are incompletely understood. **Methods:** We reviewed studies relating to the regulation of energy balance and how these factors may contribute to the development of obesity. **Results:** Although it is widely believed that genetics contribute significantly to the variability in body fatness, the available data do not support a role for defects in resting metabolic rate, substrate metabolism, dietary induced thermogenesis, or the energy cost of physical activity as significant causes of obesity. Furthermore, it is safe to say that the human genotype has not changed substantially over the past two to three decades. Data from several national surveys indicate that over the past few decades, there has been either a slight increase or a very modest decline in total energy and fat intake. This suggests that decreases in physical activity are a major contributing factor. Participation in leisure time physical activity is low but has remained relatively constant. However, an increased reliance on technology has substantially reduced work-related physical activity and the energy expenditure required for daily living. **Conclusion:** The most likely environmental factor contributing to the current obesity epidemic is a continued decline in daily energy expenditure that has not been matched by an equivalent reduction in energy intake. Because daily energy expenditure is decreasing, it is difficult for most people to restrict intake to meet energy requirements, and more and more people are becoming obese. Thus, increasing physical activity may be the strategy of choice for public health efforts to prevent obesity. **Key Words:** BODY WEIGHT REGULATION, DIETARY FAT, PHYSICAL ACTIVITY, ENERGY INTAKE, ENERGY EXPENDITURE

Obesity occurs when energy intake exceeds energy expenditure, and an understanding of how obesity occurs must begin with an understanding of how energy balance is regulated. Individuals reach energy balance and achieve stability of body weight when there is a balance between energy intake and energy expenditure and between the intake and oxidation of each macronutrient (27). The body weight and body composition that is maintained at the point of reaching energy balance is determined by many genetic and environmental factors that are incompletely understood.

Within any given environment, there will be a variation in body fatness among people who are in energy balance. Although genetic factors contribute to some of this variation (6,10,57,58), we cannot state the contribution of genetic factors with certainty. The available data indicate that the genetic contribution to variability in body fatness lies somewhere between 25 and 70%; studies in monozygous twins suggest this may be on the order of 50–70%, but family studies suggest it may be closer to 25–50%. Understanding the way in which genes contribute to this variation in body fatness is important and is the subject of much investigation.

It is clear that substantial changes in the environment can affect body fatness (14,45). As the environment becomes more obesity-conducive (as has the environment in the United States), the average body weight and fatness of the population will increase. Genes may protect some individuals from becoming obese and contribute to differences in the extent to which obesity occurs, but environmental factors may be overwhelming our genetic defenses against obesity.

Body weight is remarkably constant in many people over long periods of time, suggesting that the body has some capacity to adjust energy intake and/or energy expenditure to maintain its current state of energy balance (31,62). However, such studies suggest that the capacity to maintain a constant body weight and composition is limited, and can be overcome with a sufficient challenge (e.g., extended periods of over- or underfeeding). It is only when environmental pressure toward positive energy balance produces a sufficient challenge to exceed the capacity of the body to adapt that weight and fat gain occurs. When the organism is faced with a sufficient challenge, such as chronic overfeeding, the increases that occur in body fat mass seem to help restore energy and macronutrient balance (3,19). This re-

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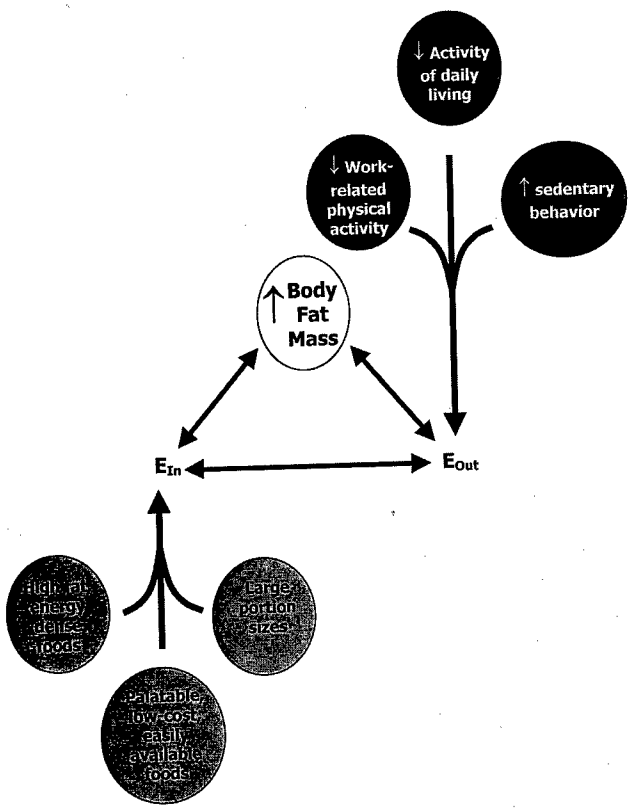

Figure 1—The effect of environmental factors on energy balance. When energy intake (E_{in}) equals energy expenditure (E_{out}), the system is in energy balance and body fat mass stable. In the current environment, factors (in circles) on the left are driving E_{in} up, whereas factors on the right are driving E_{out} down, creating a state of positive energy balance, leading to an increase in the body fat mass.

sults in a new steady-state of body weight but at a higher body weight.

We cannot, at present, determine the degree or pattern of positive energy balance required to overcome the body's energy balance regulatory capacity and produce weight gain. Obesity is not a disease that develops quickly; rather, the typical pattern is gradual weight gain over a period of several years. This could be attributed, for example, to a chronic small positive energy balance or to episodic periods of more pronounced positive energy balance. In either case, we are dealing with very small degrees of energy imbalance that may not be detectable even with our most sophisticated techniques for assessing energy intake and energy expenditure.

GENES VERSUS ENVIRONMENT

Given that the American genotype has not changed substantially over the past two to three decades, we must look to the environment as the primary cause of the obesity epidemic. The most likely explanation for the high prevalence of obesity in the United States is an environment that produces constant pressure toward positive energy balance by promoting energy intake and discouraging physical activity. This is illustrated in Figure 1. A recent report from the World Health Organization concluded that ". . . behavioral factors tend to overwhelm an individual's normal subconscious adjustments in food intake and metabolism that occur as part of a biological capacity to maintain energy balance" (66). The report identifies a fall in spontaneous, work-related physical activity and the availability of high fat, energy dense foods as two principal environmental factors that promote behaviors that lead to positive energy balance.

A Low Level of Energy Expenditure as a Cause of Obesity

Daily energy expenditure consists of three components—resting metabolic rate (RMR), the thermic effect of food (TEF), and the energy expended in physical activity (EE_{act}). The major determinant of EE_{act} is the amount of physical activity performed, but there are also variations between subjects in the energy cost of physical activity.

The available data do not support a role for a metabolic defect in resting energy expenditure as a significant cause of obesity. There is substantial variation in RMR, some of which appears due to genetic factors (8,46), but based on results of several prospective studies, it appears that a low RMR is not a major factor involved in the etiology of human obesity (24,52,65). RMR is primarily determined by body composition (68), and there is no indication that RMR has declined over the past few decades.

TEF varies within and between individuals, and the question of whether TEF is lower in obesity is controversial (15). Differences in TEF between lean and obese subjects, where found, have been small and there is no evidence that differences in TEF play a role in the development of obesity. Further, there is no indication that TEF has declined over the past few decades.

Finally, there is little evidence to suggest that differences in the energy cost of physical activity play a major causal role in obesity (29). It is worth noting that as obesity develops, the energy cost of weight bearing physical activity increases (41,42).

Some investigators have suggested that a relatively high respiratory quotient (RQ), reflective of reduced fat oxidation, may be a cause of obesity (69). In individuals eating similar diets, those with lower rates of fat oxidation could require larger fat mass in order to achieve energy and fat balance. However, the extent to which "defects" in substrate utilization cause obesity versus contribute to normal variation in body fatness is unclear and deserves further research.

In contrast, there are substantial data to suggest that differences in amount of physical activity contribute to differences in body weight and body fatness, and play an important role in whether obesity develops. Most evidence comes from cross-sectional and population studies that consistently show a negative relationship between level of physical activity and indices of obesity, such as body mass index (BMI) (23,53,67). Additionally, cohort studies, where subjects were studied periodically over years, consistently show that high levels of physical activity are protective against obesity (47,67). Although we lack a definitive prospective study to show that a low level of physical activity is a risk for obesity development and that a high level of

physical activity is protective against obesity, an overwhelming amount of indirect evidence suggests this is the case. The amount of physical activity that protects against obesity is not known, but some have suggested that a physical activity level of 1.75 (daily energy expenditure of 1.75 times the basal metabolic rate) should be our target (66). We need more information in order to provide guidance to the public on this issue.

Environmental Influences on Physical Activity

If a low level of physical activity is a major determinant of low energy expenditure, it is useful to understand more about this component of energy expenditure. Genetics may affect amount of physical activity (7), but it is generally accepted that genetic influences on this component of energy expenditure are less than on other components. Thus, we must consider how changes in the environment over the past few decades may have influenced the levels of daily physical activity.

First, how have changes in our environment affected the amount of physical activity required in daily living? Although it is intuitively obvious that improvements in technology over the past few decades have substantially reduced the energy expenditure required for daily living, this has not been definitively documented. All indications are that work-related physical activity has declined. The only data available come from Finland, where work-related physical activity reportedly declined by 225 $kJ \cdot d^{-1}$ between 1982 and 1992 (21). Similarly, there is reason to believe that household related physical activity has declined rapidly over the past two to three decades. One can, for example, estimate the energy savings due to proliferation of energy-saving devices such as washing machines, dishwashers, computers, remote control devices, and microwave ovens. Daily energy expenditure has also likely declined due to an increased use of prepared foods. Although each may reduce physical activity only slightly, together these energy savings accumulate and can have a significant impact upon total energy expenditure. There is a great need for developing methods for assessing lifestyle physical activity to determine the extent of this decrease over time.

Second, what are the secular trends of participation in leisure time physical activity (LTPA)? Most data suggest that participation in LTPA has remained relatively constant over the past few decades (54,63). Analysis of the NHANES III data suggests that about 22% of United States adults do not participate in LTPA, with higher rates in Hispanic (46%) and African-American (40%) women, groups with a particularly high prevalence of obesity (13). Although the overall prevalence of individuals reporting no LTPA is low, most indications are that the rate has remained relatively constant over the past two to three decades and, thus, has not likely contributed to a decline in total energy expenditure. The available data suggest that there has not been a systematic increase in this component of physical activity. It is therefore very likely that on a secular basis, participation in LTPA has not increased enough to offset significant declines in other aspects of physical activity, leading to a situation that favors positive energy balance in a large percentage of the population. This is not limited to adults, as it is also likely that significant declines have occurred in the amount of physical activity that children receive in schools. It is not possible to quantify the extent of this decline over the past two to three decades, but the requirement for physical education has declined in most schools as has the number of school children participating in physical education classes (50). A decline in mandatory physical education from 200 $min \cdot wk^{-1}$ (40 $min \cdot d^{-1}$, 5 $d \cdot wk^{-1}$) to 60 $min \cdot wk^{-1}$ (30 min, 2 $d \cdot wk^{-1}$) could result in a decline in energy expenditure of approximately 100 $kcal \cdot d^{-1}$.

The amount of energy expenditure required for daily living also appears to be declining due to an increase in attractive sedentary activities such as television watching, video games, and computer interactions. Again, we do not have good measures of sedentary activity that would allow us to examine changes over time. Although it is likely that increases in sedentary activities are not reducing time spent in LTPA, increases in time spent in sedentary activities likely represents a lower energy expenditure than would occur otherwise.

A High Energy Intake as a Cause of Obesity

Energy intake can only be evaluated in relation to energy expenditure. If we accept that total energy expenditure has declined over the past two to three decades, obesity would have occurred unless there has been an equivalent decline in energy intake. The available data are problematic in that is has been obtained from self-reports, which have been shown to misrepresent the actual amount of energy consumed (33). If, however, we assume that measures taken over time accurately reflect change in energy intake, the available data suggest there has been either an increase or a very modest decline in total energy intake over the past two to three decades (17,18). This suggests the increase in obesity may have resulted from a decline in energy expenditure that was not matched by an equivalent reduction in energy intake.

Environmental Influences on Energy Intake

We must therefore consider which factors in the environment are promoting energy intake and thus making it difficult for most individuals to accurately match intake to a low level of energy expenditure. Environmental factors that have been implicated include excess dietary fat, the energy density of consumed food, sugar intake, large portion sizes, meal patterns (i.e., frequency of eating), and availability and cost of food.

Diets high in fat have been suggested to increase the risk of overeating and obesity (40,55,56,59). It is important to realize that most of these studies have measured voluntary food intake in subjects eating very low (e.g., <20%) and very high (>40%) fat diets. There is a need for information regarding the effect of dietary composition on voluntary energy intake for diets varying in %fat over the range of usual consumption (e.g., 20–40% fat).

Several studies suggest that reducing dietary fat intake may help reduce total energy intake (9,34,36). Additionally, when dietary fat is covertly reduced with noncaloric fat substitutes, most subjects do not compensate by increasing voluntary fat intake or by increasing intake of other macronutrients (30). Dietary obesity can be reversed by switching rodents from a high fat to a low fat diet (26,49). However, the extent to which this occurs depends on the extent and duration of the dietary obesity (28).

It is misleading to conclude that high fat diets are a sole cause of obesity. It would be more accurate to consider a high fat diet as a factor increasing the probability of overeating, or alternatively, to consider a low fat diet as decreasing the probability of overeating. Again, whether overeating occurs with a high fat diet may depend on genetic factors as well as on nongenetic factors such as the level of physical activity. The available data would suggest that as the food supply increases in fat, the percentage of people experiencing sustained or episodic overeating is also increasing. Reducing the fat content of the food supply should reduce the likelihood of overeating and could be a useful means of reducing the prevalence of obesity.

There are substantial data to suggest that energy intake is influenced by the energy density (energy per weight of food) of the diet (48). Because diets high in fat are also high in energy density, some have suggested that it is energy density and not the fat in the diet *per se* that produces overeating. If people tend to eat a constant volume of food, more total energy will be consumed when the diet is high in energy density than when it is low in energy density. Even if energy density is an important determinant of total energy intake, are variations in dietary fat still the major determinant of energy density? This question is currently being studied in a number of laboratories. An important research question is whether fat-and calorie-modified foods, which may be low in fat but high in energy density, contribute to increased energy intake.

Several authors have pointed out an apparent paradox whereby obesity in the United States has increased over the past few decades while dietary fat as a percentage of total calories has slightly declined (25). In fact, self-reported food intake from NHANES surveys suggests that dietary fat intake in absolute terms has remained relatively constant over this period, but since total energy intake has increased, the proportion of intake from fat has declined (17). It is very misleading to conclude from this that dietary fat intake is not likely to play a role in the recent increase in the prevalence of obesity. It may be *because* the diets are high in fat that the additional increase in energy intake has occurred.

Despite the common perception that sugar contributes to overeating and obesity, the available data suggest this is not the case (2). The portions in which food is presented to Americans, however, has increased over the past few decades and could be contributing to overeating. This can be seen in "supersizing" in fast-food restaurants, in the larger portions served in other restaurants, and in the increase in the size of products such as candy bars and soft drinks. Although there is insufficient research to definitely implicate portion size as a contributor to overeating, it is reasonable to think this might be the case. The likelihood of overeating at any given meal will likely be related to the portions of food served at that meal. This is especially likely if the food served is high in fat and high in energy density.

Some researchers have suggested that the pattern of food consumption may relate to obesity. However, a recent review concluded that based on available data, there does not appear to be a relationship between meal patterning and obesity (5). Finally, our current environment is one is which food is abundant and relatively inexpensive. Very little systematic research has been conducted relating energy intake to the cost and availability of food. The extent to which this promotes energy intake and overeating deserves further study.

COMBINED ENVIRONMENTAL EFFECTS ON ENERGY BALANCE

We have specified the influence of the environment on increasing energy intake and on decreasing physical activity. However, to understand the development of obesity, we must consider the two together. A low daily energy expenditure would not necessarily be a cause of obesity unless there was an inability to adjust energy intake appropriately. This indeed seems to be the case. If total energy expenditure has declined over the past two to three decades, avoiding obesity would have necessitated a comparable decline in energy intake. Unfortunately, our measurement devices are not sufficiently accurate to determine whether this has been the case; in fact, the rise in obesity argues that it has not.

Physiologically, we may not have a good ability to restrict energy intake to match a low level of energy expenditure. In fact, there has previously been survival value in the opposite ability—to secure sufficient energy intake to avoid depletion of body energy stores. The fact that we are not all obese within our current environment suggests either that many people are maintaining a relatively high level of energy expenditure through regular physical activity or that these individuals have a good ability to restrict energy intake to meet a low rate of energy expenditure.

The most likely explanation for the high prevalence of obesity in the United States today is that our environment requires low levels of energy expenditure. Our species has not evolved with the ability to restrict energy intake to match a low level of energy expenditure. This creates degrees of positive energy balance (either sustained or episodic) that exceed our ability to adjust energy intake or energy expenditure and require an increase in the body fat mass. Whether and to what extent an increase in body fat mass is required may be influenced by genetic factors.

Individuals who engage in high levels of physical activity and who consume habitual diets low in fat and low in energy density may be protected against developing obesity. In fact, it may be the case that either behavior can protect against obesity. There is evidence, for example, that animals or humans who engage in regular physical activity may avoid weight gain on a high fat diet (4,39,61) and that sedentary

individuals can avoid obesity by consuming a low fat diet (39,43).

There is a need for substantial research in this area. If regular physical activity and a low fat, low energy density diet are protective against obesity, how much of each is required to prevent obesity? It makes sense to work toward obesity prevention guidelines that combine diet and physical activity. The optimum diet to prevent obesity in a sedentary person is likely to be different than in an active person, and the physical activity needed to prevent obesity is likely different in a person whose habitual diet is high in fat versus low in fat.

CAUSES OF OBESITY IN SPECIAL POPULATIONS

Obesity presents a particular problem for some minority groups. Obesity is higher, for example, in Hispanic and African-American women than in Caucasian women (20). This increased prevalence has been associated with metabolic and behavioral factors. RMR is lower in African-Americans than Caucasians (1,11,22,32), and a low RMR has been suggested to be a cause of the high prevalence of obesity in African-American women. However, RMR is also lower in African-American men than in Caucasian men, but the prevalence of obesity is not different (20). The high prevalence of obesity in minorities may be due to greater environmental pressures that promote greater excess energy intake and reductions in physical activity (38,44). Black women, for example, are less active and eat a diet higher in fat than Caucasian women (13,44). It could be that the combination of low RMR and low physical activity interact to affect the high prevalence of obesity in African-American women. We do not have enough data on behavioral and metabolic factors that may contribute to the higher prevalence of obesity in Hispanic women and men.

The reduced-obese have been studied as a population at high risk for obesity development. It has been suggested that reduced-obese individuals may suffer from a low relative RMR and an impaired capacity to oxidize fat (35,37). However, this is highly controversial, with many reports that RMR and fat oxidation are not impaired in the reduced-obese (16,65). This is a valuable population that needs further study.

Aging is also associated with an increase in obesity. Several investigators have shown that a reduction in physical activity may be a key component in initiating the positive energy balance that leads to obesity with advancing age (12,51). Individuals who maintain high levels of physical activity as they age are able to avoid storing excess body fat (60,64).

SUMMARY

The cause of the worldwide obesity epidemic is an environment that encourages excessive energy intake and discourages physical activity. These pressures are sufficient to create a state of positive energy balance that requires an increase in the body fat mass in order to reestablish energy balance. The data available suggest that declining levels of physical activity may be a key factor in the development of obesity. In our current environment, energy expenditure is low in most people, due to a low level of physical activity. Obesity can only be avoided if energy intake is restricted to meet low energy requirements. It is difficult for most people to do this consistently, and more and more people are becoming obese. Becoming obese is a natural response to our current environment and without changes in the environment, obesity will likely become a characteristic of our species.

There are two major environmental strategies to preventing obesity—increasing physical activity or decreasing food intake. Either strategy will be difficult to implement. However, given that human physiology is aimed at ensuring sufficient energy intake through multiple redundant pathways, we suggest that increasing physical activity may be the strategy of choice for public health efforts to prevent obesity.

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Assessment of physical activity level in relation to obesity: current evidence and research issues

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ABSTRACT

WESTERTERP, K. R. Assessment of physical activity level in relation to obesity: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S522–S525, 1999. **Purpose:** Validations of methods for the assessment of physical activity and studies on the relation between energy expenditure of activity and obesity were reviewed, with suggestions for further research. **Design:** Validation studies of field methods for the assessment of physical activity against doubly labeled water were evaluated, studies on the relation between doubly labeled water assessed energy expenditure of activity and obesity are discussed. **Methods:** Three field methods for the assessment of physical activity, validated with doubly labeled water as a criterion method, were included: activity questionnaires, heart rate monitoring, and motion sensors. **Results:** The triaxial accelerometer came out as the best field method for the assessment of physical activity, followed by the Baecke activity questionnaire. The majority of obese subjects are moderately active, and an increase in the activity level of obese subjects is limited by the ability to perform exercise of higher intensity. **Conclusions:** Accelerometers are an objective tool for the assessment of physical activity in large populations over periods long enough to be representative for normal daily life and with minimal discomfort to the subjects. The accelerometer can be used to distinguish differences in activity levels between individuals and to assess the effect of interventions on physical activity within individuals. **Key Words:** DOUBLY LABELED WATER, ACTIVITY QUESTIONNAIRES, HEART RATE MONITORING, MOTION SENSORS, ACCELEROMETERS

To get more insight into the interaction between daily physical activity and health, an objective and reliable method for the assessment of physical activity in free-living subjects is required. The method should be suitable to measure physical activity in large populations over periods long enough to be representative for normal daily life and with minimal discomfort to the subjects. Presently there are a large number of techniques for the assessment of physical activity, which can be grouped into five general categories: behavioral observation; questionnaires (including diaries, recall questionnaires, and interviews); physiological markers like heart rate; calorimetry; and motion sensors. Validated techniques of estimating habitual physical activity are needed to study the relationship between physical activity and health. The greatest obstacle to validating field methods of assessing physical activity in humans has been the lack of an adequate criterion with which techniques may be compared. The interrelation of various field methods may be of some value, but because there are errors in all methods, it is impossible to determine the true validity of any one of them in doing so (17). However, calorimetry, more specifically the doubly labeled water method, is becoming a gold standard for the validation of field methods of assessing physical activity (16).

The doubly labeled water method allows accurate measurement of average daily metabolic rate (ADMR) under unrestricted conditions over 1- to 3-wk intervals (23). In combination with a measurement of basal metabolic rate (BMR), the activity level of a subject can be calculated. The physical activity level is calculated by expressing ADMR as a multiple of BMR (PAL = ADMR/BMR (8)) or by adjusting ADMR for BMR as suggested by Carpenter et al. (6). Doubly labeled water for the assessment of ADMR in man was first applied by Schoeller and Van Santen (25), and the technique has since then been evaluated in meetings of its users (19,24,28).

The current review comprises validations of methods for the assessment of physical activity against doubly labeled water, and studies on the relation between doubly labeled water assessed energy expenditure of activity and obesity. Finally, suggestions are made for further research.

METHODS

So far, three field methods for the assessment of physical activity have been validated with the doubly labeled water technique as a criterion measure: questionnaires, heart rate monitoring, and motion sensors. A literature search yielded 14 papers including doubly labeled water in combination

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with one of the three methods mentioned above, four on questionnaires, eight on heart rate monitoring, and two on motion sensors.

Studies on activity questionnaires comprised a 1-wk (5) or 2-wk activity diary (27), two 7-d activity recall questionnaires (21), the physical activity scale for the elderly (26), the Baecke questionnaire, the Five-City questionnaire, and an adapted version of the Tecumseh Community Health study questionnaire (18). The activity diary method was as described by Bouchard et al. (3). Subjects recorded every 15 min of the waking interval a number corresponding to one of a grouping of nine activity categories (5) or 12 activity categories (27), according to their average physical activity during that time period. Numbers were converted to ADMR by multiplying the integrated mean 24-h activity score with the measured BMR. The activity recall method included a standard questionnaire that categorizes activities by their intensity, using the compendium of Ainsworth et al. (1). Energy expenditure was then calculated by multiplying the amount of time spent in each activity by the corresponding number of MET. The physical activity scale for the elderly (PASE) was a brief questionnaire as described by Washburn et al. (30). It comprises activities commonly engaged in by elderly persons, and the reference period is 1 wk. The PASE-score is the sum of the time spent in each activity, multiplied by an item weight factor. The Baecke questionnaire is a brief questionnaire with three categories, work, sport and leisure time, adding up to a total activity index (2). The Five-City questionnaire asks the time spent in vigorous activities, moderate activities, light activities, and sleeping. Each category is multiplied by the reported hours and a weight factor to calculate an activity index. The Tecumseh questionnaire was an adapted version of the questionnaire of Reiff et al. (224). Subjects were interviewed on the estimated hours per week of sports participation, home repair and maintenance activities, sleeping and eating, quiet leisure time, and remaining activities. The hours per week were multiplied by the PAL values as listed by Ainsworth et al. (1) to get a figure for total activity.

Studies on heart rate monitoring generally included 2–4 d of continuous heart rate monitoring during the 7- to 14-d observation intervals with doubly labeled water. Observation days were weekdays as well as weekend days and results were weighted in a ratio according the doubly labeled water interval. All studies applied individually assessed heart-rate energy-expenditure calibration equations for the estimation of daily energy expenditure. The calibration equations comprised two linear regression lines, one for heart rates below and one for heart rates above the average value for sedentary activities; the so-called flex heart rate.

Studies on motion sensors included a single-axial accelerometer and an triaxial accelerometer for movement registration. Johnson et al. (10) monitored body movement with the single-axial Caltrac over 3 d of a 13- to 16-d observation interval with doubly labeled water. Bouten et al. (4) monitored body movement with a triaxial accelerometer over the first 7 d of a 14-d observation interval with doubly labeled water. Westerterp and Bouten (34) reported results of the same study with a different data analysis.

TABLE 1. Correlation between the activity score of questionnaires and the doubly labeled water assessed physical activity level.

Questionnaire	Subject No.	Correlation	Reference
Activity diary	6	0.72**	27
Activity diary	50	Not presented	5
Activity recall	13	0.67*	21
Physical activity scale for the elderly	21	0.68**	26
Baecke questionnaire	19	0.69***	18
Five-City questionnaire	19	0.42	18
Tecumseh questionnaire	19	0.64**	18

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

RESULTS

Most studies on activity questionnaires showed an association between the derived activity score and the doubly labeled water assessed physical activity level (Table 1). The Baecke questionnaire tended to show the highest correlation, and next best was the physical activity scale for the elderly and the Tecumseh questionnaire. The index of the Five-City questionnaire was not related to the doubly labeled water assessed physical activity level. As expected, an activity diary was superior to activity recall.

Total energy expenditure assessed with heart rate monitoring (TEE_{HR}) was not different from total energy expenditure assessed with doubly labeled water (TEE_{DLW}) at group level in all studies. However, individual differences were large as shown by the SD of the mean. The reported extremes ranged between −17% (13) and +52% (14).

Of the motion sensors, the single-axial Caltrac was not a meaningful predictor of physical activity (Table 3). The triaxial accelerometer for movement registration showed a close correlation with the doubly labeled water assessed PAL (4) as well as with ADMR adjusted for resting metabolic rate (34). The standard error of estimate of ADMR in the latter study was 0.9 $MJ \cdot d^{-1}$.

DISCUSSION

Doubly labeled water is an accepted criterion measure for other methods for the assessment of physical activity level and energy expenditure. Unfortunately, the doubly labeled water method itself cannot be applied at a population level. Oxygen-18 water is expensive and not readily available. Of the three reviewed methods for the assessment of physical

TABLE 2. Total energy expenditure as assessed with heart rate monitoring (TEE_{HR}) and with doubly labeled water (TEE_{DLW}); data are mean ± SD.

Subject No.	TEE_{HR} ($MJ \cdot d^{-1}$)	TEE_{DLW} ($MJ \cdot d^{-1}$)	Difference (%)	Reference
6	15.5 ± 3	13.3 ± 2.3	+17 ± 13	27
14	13.0 ± 3.8	12.9 ± 3.8	+2 ± 18	14
10	9.8 ± 1.6	8.5 ± 1.4	+16 ± 17	7
36	8.9 ± 2.0	9.2 ± 1.8	−3 ± 10	13
6 (obese)	9.5 ± 0.8	9.0 ± 0.6	+6 ± 5	15
7 (nonobese)	8.4 ± 2.0	8.4 ± 2.3	0 ± 8	
13 (obese)	10.3 ± 1.3	10.9 ± 1.8	−5 ± 11	21
8	16.1 ± 2.5	16.2 ± 4.2	+3 ± 23	9
9	7.4 ± 2.1	7.4 ± 2.1	−1 ± 12	29

TABLE 3. Correlation between the activity score of motion sensors and the doubly labeled water assessed physical activity level.

Motion sensor	Subject number	Correlation	Reference
Single-axial accelerometer	31	−0.09	10
Triaxial accelerometer	30	0.73**	4
Triaxial accelerometer	30	0.80***	34

** $P < 0.01$; *** $P < 0.001$.

activity level, the triaxial accelerometer for movement registration showed the best result. A simple method like the Baecke questionnaire also came out surprisingly well. Heart rate monitoring showed large discrepancies for individual data. Unfortunately, studies on the comparison of total energy expenditure as assessed with heart rate monitoring and with doubly labeled water (Table 2) did not present a correlation between the derived activity score of both methods as presented for the questionnaires (Table 1) and motion sensors (Table 3). Some studies presented the correlation between total energy expenditure, as assessed with heart rate monitoring and with doubly labeled water. This correlation, however, is not primarily a function of physical activity but of resting metabolic rate, the largest component of total energy expenditure.

Each of the three methods for the assessment of physical activity reviewed above has a number of positive and negative aspects. Positive aspects of questionnaires like the Baecke questionnaire are the short time needed for a subject to fill out the 21 questions, the simple scoring system for the calculation of an activity index, and the coverage of the normal daily life activity pattern of the subject. A disadvantage of questionnaires is the fact that subjects can easily overestimate or underestimate the time spent in activities, and most questionnaires are not applicable for all subject categories from children, people with and without jobs, to the elderly. Heart rate monitoring is an objective method. However, heart rate is affected by more factors than physical activity, data conversion needs individual measurements of heart rate in combination with oxygen consumption, and heart rate monitors are not tolerated by subjects for time intervals representative of daily life like 1 wk or more. Heart rate monitoring remains a proxy measure for physical activity (12). Motion sensors give objective information but the optimal instrument is not yet on the market. The triaxial accelerometer used in the studies of Bouten at al. (4) and of Westerterp and Bouten (34) was a self-made instrument, not yet commercially available. The commercial available triaxial accelerometer named Tritrac (Reining International, Ltd., Madison, WI) has not yet been validated with doubly labeled water. Additionally, the size of the validated triaxial accelerometer and of the Tritrac, respectively, 170 g, 238 cm^3 and 275 g, 270 cm^3, limits wearing comfort. We now use a miniaturized version of the validated triaxial accelerometer measuring 30 g, 11 cm^3 (33).

A research issue for physical activity level and energy expenditure of activity in relation to obesity is the definition of risk groups. Successful intervention should start before obesity is manifest. Doubly labeled water studies show an increase of activity associated energy expenditure with body mass index, and the average PAL of lean and obese subjects is quite similar (20,32). The majority of obese subjects is moderately active and an increase in the activity level of obese subjects is limited by the ability to perform exercise of higher intensity.

Exercise training potentially increases energy expenditure and decreases body fat, a beneficial aspect for somebody with too much body fat. However, women tend to compensate for an exercise-induced increase in expenditure with an increased intake, resulting in a smaller effect on body mass and fat mass compared with men. Cross-sectional data confirm the evidence mentioned; a higher activity energy expenditure is related to a lower percent body fat in men, whereas no such relationship is apparent in women (35).

The combination of energy restriction with exercise training does not result in additional fat loss. Doubly labeled water studies have shown that training induced energy expenditure during dieting is compensated by a reduction of physical activity outside the training interval (32). However, there are indications that exercise training helps subjects to comply with energy restricted diet. Successful maintenance of body mass and body composition, after weight reduction, will be facilitated at a higher level of energy turnover (31).

Finally, there is an indication that the effect of overfeeding, i.e., a positive energy balance, on weight gain is a function of habitual physical activity. Levine at al. (11) recently showed, by measuring changes in ADMR with doubly labeled water after overfeeding, that resistance to fat gain in nonobese men and women was a function of what they called nonexercise activity thermogenesis (NEAT). An objective activity monitor like an triaxial accelerometer would allow more insight in the components of NEAT as an important potential determinant of obesity.

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Levels of physical activity and inactivity in children and adults in the United States: current evidence and research issues

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ABSTRACT

PRATT, M., C. A. MACERA, and C. BLANTON. Levels of physical activity and inactivity in children and adults in the United States: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S526–S533, 1999. **Purpose:** The purpose was to describe current levels of physical activity and inactivity among adults and young people in the United States. **Methods:** Estimates of participation in regular physical activity were derived from three national surveys for adults (National Health Interview Survey, National Health and Nutrition Examination Survey, and the Behavioral Risk Factor Surveillance System) and from the Youth Risk Behavior Survey for high school students. **Results:** Overall, 63.8% of high school students surveyed on the 1997 YRBS reported participating in vigorous physical activity for at least 20 min on 3 or more days per week. Participation in vigorous activity was higher for boys (72.3%) than girls (53.5%), whites (66.8%) compared with blacks (53.9%) and Hispanics (60.4%), and decreased with advancing grade. Among adults, 27.7% meet recommended levels of either moderate or vigorous physical activity, whereas 29.2% report no regular physical activity outside of their work. Gender differences in participation in physical activity are less pronounced than in youth, and age-related patterns were complex. Whites are more active than blacks and Hispanics, and persons with higher family incomes and more education report being more physically active. There have been only minor changes in reported participation in leisure time physical activity over the past 15 yr. **Conclusion:** National estimates of physical activity appear to be reliable and valid for adults but may be less so for adolescents and are poor measures for children. Research is needed to determine the role that objective monitoring with accelerometers may play in surveillance. Reliable and valid measures of occupational, household, and transportation-related physical activity and sedentary behaviors are needed to better characterize the range of activity that is associated with health.
Key Words: OBESITY, EXERCISE, SEDENTARY BEHAVIOR, PUBLIC HEALTH

Regular physical activity is an important contributor to a healthy lifestyle and preventing chronic disease (27,28,47). Physical activity may be especially important in preventing obesity (47,49). Assessment of current levels of physical activity and inactivity is critical for defining the extent of the problem, guiding public health efforts, and evaluating progress toward national health objectives (14,30,45,46,48).

Measures of physical activity have been included in the three major national surveys used to assess the health and health behaviors of the U.S. population: National Health and Nutrition Examination Survey (NHANES), National Health Interview Survey (NHIS), and the Behavioral Risk Factor Surveillance System (BRFSS). A national survey of physical activity and fitness of children and adolescents aged 6–18 yr was carried out between 1984 and 1986: National Children and Youth Fitness study I and II (NCYFS I and II) (33,34). This survey has not been repeated. Current estimates of youth physical activity are derived from the Youth Risk Behavior Survey (YRBS). Each of the current national surveys is described briefly below.

NHANES. Different questions on physical activity have been included in NHANES I, II, and III. These questions have not assessed the duration of participation in physical activity and thus cannot be used to assess the overall quantity of physical activity or progress toward national objectives for vigorous or moderate physical activity (16).

NHIS. The Health Promotion/Disease Prevention supplements of NHIS in 1990, 1991, and 1995 included detailed assessment of participation in as many as 24 specific activities. Results for several activity categories and many demographic groups are published in *Physical Activity and Health: A Report of the Surgeon General* (23,47).

BRFSS. The BRFSS is a random-digit-dialed telephone survey of health behaviors of the noninstitutionalized U.S. population 18 yr of age and older. The BRFSS is conducted year round in all 50 states and the District of Columbia, and it is the only system that provides data at the state level (10,32). The 1996 survey results presented in this report included data from approximately 120,000 respondents. The median response rate for eligible persons contacted in the state-based surveys was 77.9% in 1996.

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YRBS. The YRBS was developed by the Centers for Disease Control and Prevention (CDC) in 1990 to monitor health behaviors, including physical activity, in young people. It is a school-based survey of high school students in grades 9–12 carried out in the spring of odd numbered years. In 1997, 16,262 questionnaires were completed in 151 schools with an overall response rate of 69% (24). In 1992, the YRBS was administered as part of the NHIS to assess health behaviors of young people aged 12–21, including those not attending school. Results from the 1992 survey were reported in 1996 in *Physical Activity and Health: A Report of the Surgeon General* (47).

For this report we have relied on weighted estimates from the most recent national data sources: the 1996 BRFSS for adults and the 1997 YRBS for high school students (12,24,44). The reliability of both surveys has been demonstrated to be very good (4,38,40,41). Validity has not been established for any of the national surveys of physical activity, although similar physical activity questionnaires for adults appear to be valid (25). Prevalence estimates for physical activity and inactivity are influenced not only by survey design and administration but by the analytic approaches and definitions used in presenting the data (5,6). The data presented here will use the following definitions of physical activity.

Vigorous physical activity. Persons are categorized as vigorously physically active if they report 3 or more sessions per week of 20 min or greater duration of activity at an intensity equal to 50% or more of their age and gender specific maximum cardiorespiratory capacity (46,47).

For high school students, vigorous physical activity is defined as responding positively to a question on the YRBS assessing participation in activities that made them sweat and breathe hard for at least 20 min on 3 or more days during the past 7 days (24).

Moderate physical activity. Persons are defined to be moderately physically active if they report 5 or more sessions per week of any activity included in the BRFSS, and the total activity time for the week equals 150 min or more. All activity sessions must be at least 10 min long (5,9).

TABLE 1. Participation in physical activity, U.S. adults, BRFSS 1996.

Demographic Group	Inactive	Vigorous	Moderate	Recommended
Overall	29.2	12.6	23.4	27.7
Men	27.2	11.6	24.3	28.2
Women	30.9	13.8	22.7	27.6
Age (yr)				
Men				
18–29	19.7	6.7	27.8	29.6
30–44	25.6	9.5	21.0	24.9
45–64	32.5	14.4	21.8	27.0
65–74	32.1	18.2	30.6	35.2
75+	36.9	19.5	27.1	32.1
Women				
18–29	26.3	10.8	23.8	27.4
30–44	28.3	14.9	22.2	28.3
45–64	32.3	15.8	23.0	28.6
65–74	36.3	15.4	24.1	28.4
75+	47.3	11.0	17.6	20.5
Race/Ethnicity				
White	26.8	13.5	24.3	29.0
Black	38.9	9.3	19.0	22.0
Hispanic	38.9	9.2	19.1	22.3
Asian/Pacific Islander	27.9	10.5	22.9	27.3
American Indian	28.6	8.7	27.0	30.3

TABLE 2. Participation in physical activity, U.S. adults, BRFSS 1996.

Demographic Group	Inactive	Vigorous	Moderate	Recommended
Education				
<High school	48.9	5.5	16.0	17.3
High school graduate	34.3	9.8	20.8	24.1
Some college	24.8	13.6	25.1	29.7
College graduate	17.2	19.1	29.2	36.1
Income ($)				
<$10,000	42.6	6.4	19.4	21.1
$10,000–14,999	41.5	7.5	19.1	21.5
$15,000–19,999	38.4	8.6	20.2	22.9
$20,000–24,999	33.7	10.2	21.0	24.6
$25,000–34,999	30.5	11.8	22.9	27.0
$35,000–49,000	24.6	14.1	25.7	30.6
$50,000–74,999	20.0	16.8	27.7	33.1
≥$75,000	15.1	19.6	31.5	38.7
Region				
Northeast	26.9	13.9	25.2	29.8
Midwest	29.2	12.8	22.3	26.8
South	34.1	12.5	20.6	25.2
West	23.1	11.4	27.4	31.0
Urban-rural				
Metro > 1 million	27.4	12.6	23.9	28.4
Metro 50,000–1 million	28.8	13.5	23.5	28.1
Urban 20,000–19,999	30.8	13.2	23.6	27.9
Urban 2,500–19,999	34.3	10.7	21.5	24.7
Rural	35.7	9.8	21.4	24.8
BMI				
<25	27.3	14.6	25.1	30.1
25–29.9	28.0	12.1	23.6	27.8
≥30	37.0	7.6	18.2	21.0

Recommended physical activity. Recommended physical activity is defined as meeting either the requirements for moderate or vigorous physical activity. This group includes all adults participating in physical activity at or above levels recommended for health benefits (5,9,28,29,47).

Inactivity. Inactivity or no physical activity refers to no reported leisure-time physical activity during the past month (5,6,47). This definition does not address the many sedentary behaviors that contribute to total time spent at low levels of energy expenditure (1). These behaviors may be important to health and obesity prevention but are not assessed well in current national surveys (18).

In the following section, levels of physical activity and inactivity from the most current national data sources are presented for adults and young people by a variety of demographic categories. These results are summarized in Tables 1 and 2 for adults and in Tables 3, 4, and 5 for high school students.

Levels of Physical Activity among Adults

The most recent national data from the 1996 BRFSS indicate that 29.2% of adults are inactive in their leisure time, 43.1% participate in some activity but not enough to ensure health benefits, and 27.7% are physically active at recommended levels (Fig. 1). Of the 27.7% who are regularly active, 23.4% report 5 or more sessions of activity totaling at least 150 min and are estimated to meet the recommendation for accumulating 30 min of moderate intensity activity 5 or more days per week. Among adults, 12.6% meet the vigorous activity recommendation (20 min or more of vigorous intensity activity 3 or more times per

TABLE 3. Percentage of high school students who participated in vigorous physical activity, by sex, race/ethnicity, and grade—United States, Youth Risk Behavior Survey, 1997.[a]

Category	Female	Male	Total
Race/ethnicity			
White[b]	58.4 (±5.7)[c]	73.4 (±2.6)	66.8 (±3.2)
Black[b]	41.3 (±4.1)	67.1 (±3.3)	53.9 (±3.0)
Hispanic	49.9 (±4.9)	69.2 (±3.7)	60.4 (±3.1)
Grade			
9	66.1 (±4.8)	78.7 (±4.1)	72.7 (±3.0)
10	55.7 (±3.2)	74.3 (±3.4)	65.9 (±2.7)
11	49.4 (±5.0)	68.9 (±3.8)	60.0 (±3.7)
12	43.6 (±7.7)	68.4 (±2.6)	57.5 (±3.8)
Total	53.5 (±3.8)	72.3 (±2.0)	63.8 (+2.1)

[a] Activities that caused sweating and hard breathing for at least 20 min on ≥3 of the 7 d preceding the survey.
[b] Non-Hispanic.
[c] Ninety-five percent confidence interval.
Adapted with permission from: Kann, L., S. A. Kinchen, B. I. Williams, et al. Youth Risk Behavior Surveillance—United States, 1997. In: *CDC Surveillance Summaries*, August 14, 1998. *MMWR* 47(No. SS-3):1–32, 1998.

week). Although it is difficult to make comparisons between countries because of differences in survey questions and methodology, similar patterns of activity and inactivity are seen in all developed countries that have national health surveys that assess physical activity (43).

Sex. Gender differences in participation in physical activity in the United States are relatively minor as can be seen in Table 1. More women (30.9%) than men (27.2%) report no physical activity, but levels of vigorous, moderate, and total (recommended) activity are quite similar. Increased participation in physical activity by women over the past decade has closed what was once a noticeable gender gap (7,8,23).

Age. Patterns of participation in physical activity by age vary between men and women and are complex (Table 1). Inactivity increases progressively with age for both men and women. Above 75 yr of age 36.9% of men and nearly half (47.3%) of women report no activity. Vigorous physical activity also increases progressively with age for men, whereas it peaks in middle-aged for women. Vigorous activity rates may be overestimated for older men and women, due to over adjustment for age-related declines in maximal cardiorespiratory capacity (47). The same absolute level of activity (i.e., walking) may be classified as moderate for a younger person and vigorous for an older person with a reduced cardiorespiratory capacity. There is little variation in total and moderate activity for women until after age 75 when participation declines. For men, levels are highest in the 18–29 and 65–74 age groups with a marked dip during middle age, perhaps associated with demands of employment. There is a corresponding increase in physical activity when men reach retirement age (65–74). The absence of this increase for women may reflect a cohort effect. Social acceptance of physical activity in 1996 was likely to be less for women in the 65 and over age groups than men of that age.

Race-ethnicity. National surveys of physical activity consistently show large differences in participation in physical activity by race-ethnicity (7,23,46,47). The 1996 BRFSS is no exception to this pattern. Whites report less inactivity (26.8%) and more total activity (29.0%) than either blacks (38.9% and 22.0%) or Hispanics (38.9% and 22.3%). Physical activity levels among Asian and Pacific Islanders and Native Americans are similar to those for whites (Table 1). Adjusting for age and socioeconomic factors diminishes, many, but not all, of the observed differences in physical activity by race (51).

Education and income. These closely linked variables both reflect socioeconomic status and a cluster of

TABLE 4. Percentage of high school students who were enrolled in a physical education (PE) class and attended PE class daily, by sex, race/ethnicity, and grade—United States, Youth Risk Behavior Survey, 1997.

	Enrolled in PE Class			Attended PE Class Daily		
Category	Female	Male	Total	Female	Male	Total
Race/ethnicity						
White[a]	46.7 (±16.2)[b]	51.8 (±14.8)	49.5 (±15.0)	21.3 (±6.9)	25.8 (±7.6)	23.8 (±6.8)
Black[a]	39.4 (±6.0)	53.7 (±6.6)	46.3 (±5.7)	28.2 (±5.4)	37.1 (±6.7)	32.5 (±5.8)
Hispanic	50.3 (±6.2)	52.6 (±6.0)	51.6 (±5.3)	37.3 (±5.9)	39.3 (±4.8)	38.4 (±3.9)
Grade						
9	68.7 (±8.5)	69.6 (±6.9)	69.2 (±6.9)	42.1 (±11.2)	43.0 (±10.7)	42.6 (±10.5)
10	50.1 (±14.2)	56.0 (±12.0)	53.3 (±12.7)	28.1 (±7.2)	32.8 (±6.9)	30.6 (±6.4)
11	34.2 (±12.2)	43.5 (±12.8)	39.3 (±11.8)	15.5 (±4.0)	22.5 (±5.8)	19.3 (±3.8)
12	28.4 (±12.4)	42.3 (±15.2)	36.1 (±13.6)	13.9 (±5.5)	23.2 (±7.3)	19.1 (±5.7)
Total	44.9 (±11.2)	52.0 (±11.2)	48.8 (±10.9)	24.6 (±5.4)	29.8 (±6.5)	27.4 (±5.6)

[a] Non-Hispanic.
[b] Ninety-five percent confidence interval.
Adapted with permission from: Kann, L., S. A. Kinchen, B. I. Williams, et al. Youth Risk Behavior Surveillance—United States, 1997. In: *CDC Surveillance Summaries*, August 14, 1998. *MMWR* 47(No. SS-3):1–32, 1998.

TABLE 5. Percentage of high school students who played on sports teams sponsored by the school, and played on sports teams unaffiliated with the school,[a] by sex, race/ethnicity, and grade—United States, Youth Risk Behavior Survey, 1997.

Category	Played on Sports Teams Run by the School			Played on Sports Teams Unaffiliated with the Schools		
	Female	Male	Total	Female	Male	Total
Race/ethnicity						
White[b]	49.4 (±6.6)	58.7 (±4.1)	54.6 (±4.6)	34.7 (±4.9)	46.6 (±3.9)	41.4 (±3.7)
Black[b]	32.9 (±3.5)	56.4 (±3.2)	44.3 (±2.5)	25.1 (±3.4)	51.8 (±3.8)	38.0 (±2.9)
Hispanic	32.3 (±3.9)	46.9 (±3.4)	40.2 (±2.6)	24.0 (±3.7)	40.5 (±3.4)	32.9 (±2.8)
Grade						
9	48.5 (±4.8)	57.2 (±6.1)	53.1 (±4.5)	36.8 (±3.7)	51.3 (±4.4)	44.4 (±3.1)
10	45.0 (±5.6)	58.0 (±6.4)	52.2 (±5.5)	34.7 (±5.3)	47.3 (±4.3)	41.7 (±3.9)
11	40.7 (±5.8)	54.0 (±4.2)	48.0 (±3.3)	26.4 (±4.7)	41.6 (±5.5)	34.7 (±4.0)
12	35.7 (±8.2)	53.4 (±5.5)	45.5 (±6.1)	21.9 (±5.9)	42.6 (±4.8)	33.5 (±4.9)
Total	42.3 (±4.4)	55.5 (±3.4)	49.5 (±3.5)	29.8 (±3.9)	45.4 (±3.2)	38.3 (±3.0)

[a] During the 12 months preceding the survey.
[b] Non-Hispanic.
[c] Ninety-five percent confidence interval.
Adapted with permission from: Kann, L., S. A. Kinchen, B. I. Williams, et al. Youth Risk Behavior Surveillance—United States, 1997. In: *CDC Surveillance Summaries*, August 14, 1998. *MMWR* 47(No. SS-3):1–32, 1998.

factors (knowledge, time, social support, access to facilities, and neighborhood safety) that appear to influence participation in physical activity (35,36,47). Of the demographic factors assessed in the BRFSS, education is most closely associated with participation in physical activity. Nearly half (48.9%) of persons with less than a high school education report no physical activity, whereas only 17.3% of college graduates are inactive. The gradient is almost as steep for family income: inactivity falls from 42.6% to 15.1% from the lowest to highest income categories (Table 1). Because the BRFSS and other national surveys of physical activity focus on assessing leisure time physical activity and do not adequately address occupational activity, there is likely to be some misclassification of lower income or education persons with physically demanding jobs as inactive (12,23).

Body mass index (BMI). Persons with higher levels of self-reported physically activity usually have lower indices of body weight or fat (13,47,50). In the 1996 BRFSS, persons with BMI of less than 25 reported less inactivity and more regular physical activity than overweight (BMI 25–29.9) or obese (BMI ≥ 30) persons (Table 1). Participation in physical activity differed only slightly between normal weight and overweight persons but was substantially lower among the obese. Increases in inactivity with increasing BMI (normal, overweight, obese) were more marked for women (27.8%, 31.7%, and 40.9%) than for men (26.8%, 25.6%, and 32.7%). Patterns of declining participation in recommended physical activity by BMI category also differed between genders: men (normal weight 29.3%, overweight 29.2%, obese 23.5%), women (normal weight 30.7%, overweight 26.0%, obese 18.9%).

Geographic analyses. Regional differences in participation in physical activity are consistent across different surveys and years (12,47). In the 1996 BRFSS, the usual pattern of the greatest reported participation in physical activity in the west, lowest in the southeast, and intermediate levels in the midwest and northeast regions of the United States was observed (Table 2). A gradient in participation in regular physical activity has also been seen between urban and rural counties (12). Inactivity levels rise from 27.4% in metropolitan counties with populations greater than one million to 35.7% in rural counties (Table 2). This pattern is most marked in the south, present in the midwest and northeast, and absent in the west (12). The trend toward declining participation in regular physical activity as county population decreases is less clear. Regional and urban-rural differences in physical activity persist after adjustment for age, sex, education, and income (12).

Seasonality. There are clear and consistent seasonal differences in participation in physical activity (11,47). Vigorous, moderate, and recommended activity peaks in the summer months, whereas inactivity peaks in the winter. The range between the highest and lowest months is 10.4% for inactivity (January vs August) and 7.6% for recommended physical activity (August vs December). The seasonal patterns for inactivity and recommended physical activity are displayed in Figure 2.

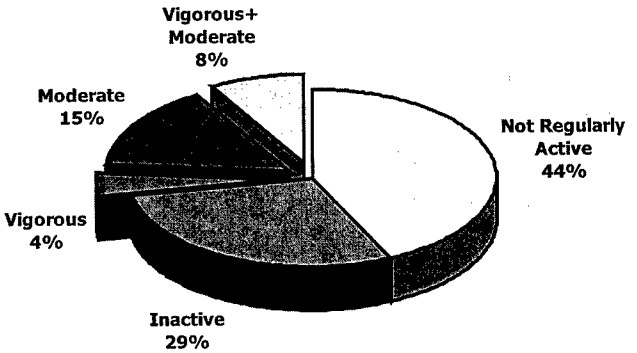

Figure 1—Physical activity, U.S. adults, BRFSS, 1996.

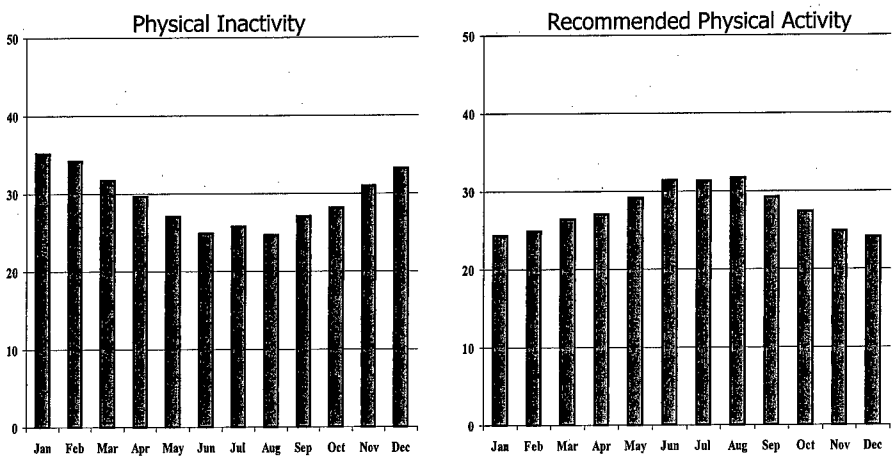

Figure 2—Physical activity patterns by month, U.S. adults, BRFSS, 1996.

Trends. It is widely believed that participation in physical activity is declining among adults in the United States. However, national survey data are not able to support this contention. Levels of physical activity and inactivity as measured by the BRFSS (Fig. 3) and NHIS have been remarkably stable during the past decade (8,39,47). Data sources consistent enough to adequately assess trends before 1985 do not exist. A synthesis of noncomparable surveys suggests that leisure-time physical activity probably increased between the 1960s and 1980s (42). The stability of leisure time physical activity over the past decade or more may mask an overall decline in physical activity. Several authors have postulated that there have been substantial decreases in overall energy expenditure driven by large decreases in the amount of physical activity required for work, transportation, and routine daily tasks (22,31).

Levels of Physical Activity Among Children and Adolescents

The 1997 YRBS provides the most recent estimates of participation in physical activity by young people. However, this survey is limited to high school students in grades 9–12. The 1992 NHIS included a YRBS supplement that was administered to a nationally representative sample of young people aged 12–21 yr. This paper primarily reports data from the 1997 YRBS with some additions from the 1992 YRBS and the 1995 National College Health Risk Behavior Survey (NCHRBS) (19). Variation in prevalence estimates between these surveys reflect not only the differences in populations but differences in season and mode of administration and question wording (47). Vigorous physical activity is assessed on these surveys with questions that allow a general comparison with adult levels of vigorous physical activity. There are additional questions on walking and bicycling, stretching, and strengthening exercises that are not included in this paper (24,47).

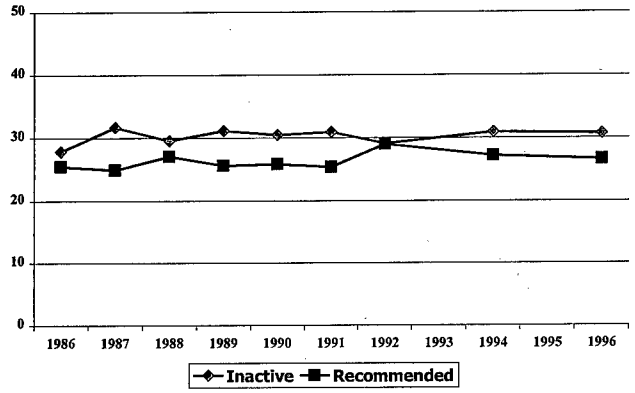

Figure 3—Participation in physical activity: adults aged 18+, BRFSS, 1986–1996.

Overall, 63.8% of high school students responding to the 1997 YRBS reported participating in vigorous physical activity. On the 1992 NHIS-YRBS, 53.7% of young people aged 12–21 reported participating in vigorous physical activity, and on the 1995 NCHRBS, 41.8% of students 18–24 and 30.6% of students 25 and older were vigorously active. Vigorous activity levels among young people are clearly much higher than those for adults, although direct comparisons are hampered by different questions and survey methodology.

Sex. All surveys of children and adolescents in the United States show higher levels of vigorous physical activity among boys than girls (19,24,47). In the 1997 YRBS 72.3% of boys and 53.5% of girls report vigorous physical activity (Table 3). Male students are also more vigorously active than female students in the NHIS-YRBS (60.2% vs 47.2%) and the NCHRBS (43.7% vs 33.0%). This difference is present in all grade, age, and race-ethnicity groups. The gender difference in participation in regular physical activity appears to be much greater among high school and college-age youth than among adults.

Age-grade. There is a progressive decrease in the prevalence of reported participation in vigorous physical activity with both grade and age. Between grades 9 and 12 reported vigorous physical activity falls from 72.7% to 57.5% (Table 3). The decline is more marked among girls (66.1% to 43.6%) than among boys (78.7% to 57.5%). Similarly large decrease between ages 12 and 21 are observed in the NHIS-YRBS for both boys (70.8% to 42.2%) and girls (66.2% to 30.2%) (46). Data from the NCHRBS is consistent with a continued decline in participation with age during the college years. Vigorous physical activity is reported by 41.8%

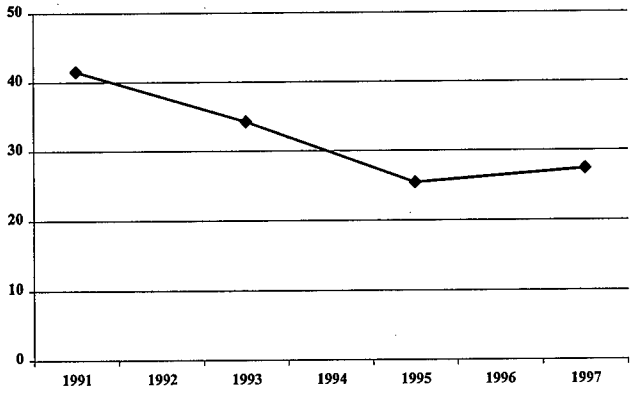

Figure 4—Attend physical education daily, grades 9–12, YRBS, 1991–1997.

of students ages 18–24, and 30.2% of students age 25 and older (19). There appears to be a steady decline in participation in vigorous physical activity beginning at age 12–14 and extending into early adulthood. Preliminary results from a regional study suggests that this decline may begin as early as first grade (37). However, current data to assess physical activity or fitness at the national level simply do not exist for children less than 12.

Race-ethnicity. White respondents to the 1997 YRBS reported greater participation in vigorous physical activity (66.8%) than either Hispanic (60.4%) or black (53.9%) students (Table 3) (24). These differences were greater for girls than boys.

Education and income. Parental education and income are not collected on the YRBS. In the NHIS-YRBS increased participation in vigorous physical activity was associated with higher family income and education level of the responsible adult (26,47). This relationship persists after standardization for age, sex, race-ethnicity, and school status (26). The relative difference in participation in physical activity between low and high education and income groups is less for adolescents than for adults.

Geographic analyses. Regional differences in participation in physical activity based on national surveys have not been reported for young people. Based on data from 33 states participating in the state-based component of the 1997 YRBS, it appears that regional patterns of participation in vigorous physical activity among high school students may be similar to that for adults, with higher levels in the west (24). This is an area in which much additional work is needed.

Trends. The public perception of an increasingly sedentary way of life among children is even more widespread than for adults (3,15). However, there is even less good information available on national trends in youth physical activity or fitness than for adults. No fitness data exist since the completion of NCYFS II in 1986 (34). The YRBS physical activity questions have been standardized only since 1993 and provide information only on young people attending school in grades 9–12. There has been no significant change in reported vigorous physical activity between 1993 and 1997: 1993, 65.8%; 1995, 63.7%; and 1997, 63.8%.

Special Issues for Youth

Television watching. Physical inactivity appears to be a distinct behavior from physical activity, and may be an important health promotion target (21). TV watching is a major contributor to sedentary activity, especially among children, and may be a useful marker for overall levels of sedentary behavior (18). It may also be important to track because of the positive association between hours of TV watched and indices of body fat and weight in children (2,17,20). Among children aged 8–16 yr examined in NHANES III, 61.0% reported watching 2 or more hours of television per day.

Physical education (PE). PE is a unique setting that provides instruction and education about physical activity and the opportunity to participate in regular physical activity. There has been a clear decline in both enrollment in PE and daily participation in PE since 1991 (Fig. 4) (24,47). In 1991, 41.6% of high school students participated in daily PE, but by 1997 this had fallen to 27.4%. PE attendance also declines markedly by grade, especially for girls (Table 4).

Organized sports. In contrast with the very small proportion of adults who participate in organized sports, a large number of high school students play on organized sports team during the course of a year (24,47) (Table 5). Nearly half (49.5%) of students play on school teams and 38.3% play on teams outside of school. Unlike for PE, there is minimal decline in participation on sports teams between grades 9 and 12 (53.2% to 45.5%). Organized sports teams appear to be an unrealized public health opportunity for maintaining and promoting physical activity for young people.

MAJOR CONCLUSIONS

All of the conclusions which follow are based on large national surveys that have been adjusted to be representative of the overall U.S. population. They are observational studies and fall into Evidence Category C.

Adults

- 29% report no physical activity during leisure time.
- 28% engage regularly in recommended levels of physical activity.
- Physical inactivity increases with age for both men and women.
- Gender differences in physical activity are small in adults.
- Higher levels of education and income and white race are associated with greater participation in physical activity.
- Obese persons (BMI $\geq$ 30) report more inactivity and less regular physical activity than overweight (BMI 25–29.9) and normal weight (BMI $<$25) persons.
- Participation in regular leisure-time physical activity has been stable during the past decade. Trends in overall physical activity cannot be assessed with currently available data.

Children and Adolescents

- Approximately 60% of adolescents participate in regular vigorous physical activity. This level is much higher than for adults.
- Boys report participating in vigorous physical activity substantially more than girls. Other differences by race-ethnicity and socioeconomic factors are similar to those seen in adults.
- Vigorous physical activity declines progressively and significantly with advancing age and grade.
- Enrollment and daily participation in physical education have declined since 1991
- National survey data is inadequate to assess trends in physical activity and fitness among children and adolescents.

Research Issues

- Improve the assessment of moderate intensity and lifestyle physical activity.
- Improve the assessment of occupational, housework, and transportation related physical activity in population-based surveys.
- Develop reliable and valid measures of sedentary behaviors that can be used on population-based surveys.
- Investigate the role of activity monitoring as a component of national population-based surveys of physical activity.
- Routinely monitor physical activity among children less than 12 yr of age and periodically assess fitness among children and adolescents.
- Improve physical activity survey methodology, reliability, and validity for children
- Standardize questions, sampling, definitions, and analysis to facilitate comparisons of physical activity behaviors between countries, surveys, and over time.
- Carry out longitudinal studies to examine changes in physical activity and sedentary behaviors during the critical transitions from childhood to adolescence and from adolescence to young adulthood.
- Improve our understanding of age and gender related differences in participating in physical activity throughout life.

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Contribution of a sedentary lifestyle and inactivity to the etiology of overweight and obesity: current evidence and research issues

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ABSTRACT

JEBB, S. A., and M. S. MOORE. Contribution of a sedentary lifestyle and inactivity to the etiology of overweight and obesity: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S534–S541, 1999. **Purpose:** The etiology of overweight and obesity is clearly multifactorial, but ultimately it is determined by the long-term balance between energy intake and expenditure. This review will consider the effects on body weight and the risk of obesity of sedentary lifestyles, within the context of dietary habits. **Methods:** The data from ecological, cross-sectional, and prospective studies that have assessed physical activity and dietary intake and their relationship to body weight were reviewed. **Results:** Ecological analyses imply that the increase in the prevalence of obesity is more strongly related to lower levels of physical activity than higher energy intakes. However, there is a paucity of pertinent data from cross-sectional or prospective studies. There is some evidence that both a high proportion of dietary fat and low levels of physical activity may increase the likelihood of weight gain. However, even the most comprehensive studies are unable to account for more than a small proportion of the interindividual variance in weight gain, so it is difficult to usefully assess their relative importance. Furthermore, there are insufficient data that pertain to "sedentary lifestyles" to segregate any putative effect from a protective effect of exercise. All the data in this review is NHLBI Evidence category C. **Conclusions:** This review provides clear evidence that low levels of physical activity are associated with an increased risk of weight gain and obesity. On balance, the evidence is suggestive of a causal link, but the experimental designs are too weak is provide conclusive evidence. The potential effect of interactions between diet and activity have largely been ignored. To make progress in this area, a number of key issues need to be resolved with regard to the methodology, study design, and statistical analysis of prospective epidemiological studies. In the meantime, data need to be drawn from other sources, particularly those studies designed to elucidate the mechanism of action of diet and physical activity in the etiology of obesity, to establish rational interventions to guide public health policies. **Key Words:** OBESITY, SEDENTARY LIFESTYLES

In recent years many countries have issued recommendations in relation to nutrition and health. Many of these encourage the maintenance of a healthy weight, but the specific advice to achieve this goal varies considerably. Most countries recommend changes in both diet and physical activity. Dietary guidelines usually include a reduction in fat intake, but the precise recommendations regarding physical activity range from a reduction in sedentary activities or an increase in habitual activities, to vigorous exercise of a precise duration or frequency. These recommendations are largely based on the results of experimental research studies, and there has been relatively little analysis of the role of each of these factors in free-living subjects.

This review will consider ecological, cross-sectional, and prospective observational studies, which include measurements of weight (or body mass index (BMI)), dietary intake, *and* physical activity. It has not been possible to include those studies that have collected all these data but where it has been published in independent publications. Furthermore, only those studies in which weight, BMI, or weight change is the outcome variable have been included. In spite of these stringent criteria, there is enormous heterogeneity between studies that limits the interpretation of the data. There are also a number of methodological issues, particularly in relation to the methods used to measure the key exposures—diet and physical activity. Carefully controlled experimental investigations that include independent measurements of dietary intake and physical activity have illustrated the tendency to underreport dietary intake and overreport physical activity (14,20). Gross misreporting of dietary data can be identified from basic physiological principles, and many of the studies in this review are suspect (10). At a population level, larger and heavier subjects would be expected to have higher habitual energy needs than their smaller counterparts, but many studies show no such association. More detailed interpretations of the macronutrient composition of the diet are therefore vulnerable

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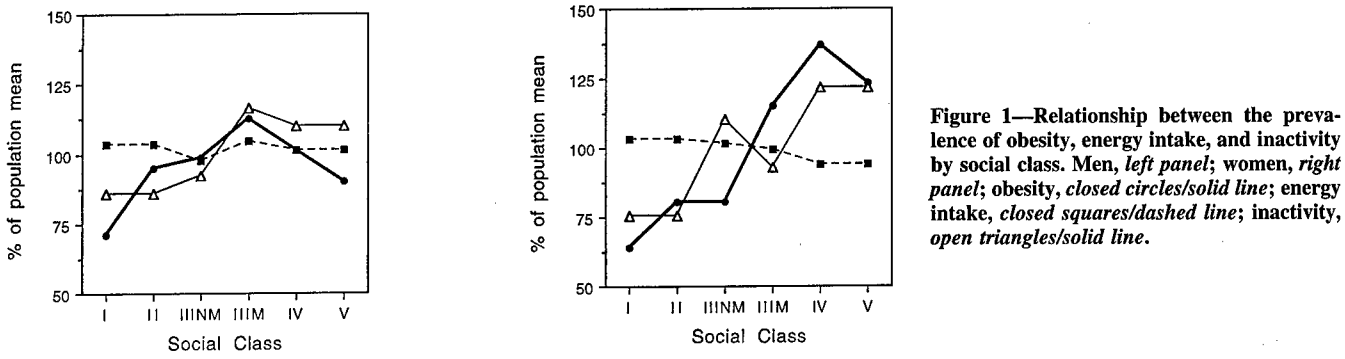

Figure 1—Relationship between the prevalence of obesity, energy intake, and inactivity by social class. Men, *left panel*; women, *right panel*; obesity, *closed circles/solid line*; energy intake, *closed squares/dashed line*; inactivity, *open triangles/solid line*.

to errors caused by biased reporting of dietary intake. There is no reliable method to validate information on physical activity, but it would be naive to imagine this data is entirely accurate. In view of the small short-term energy imbalance, which in the long-term leads to obesity, these methodological limitations thwart any robust conclusions from the following studies.

It is particularly difficult to precisely identify the contribution of a sedentary lifestyle *per se* to the etiology of obesity because few studies have quantified any sedentary activities and there has been no attempt to describe any form of global "sedentary lifestyle index." Instead, sedentariness is often inferred from the absence of active pursuits. However, this indirect approach may limit the validity of the data. Indeed, it is possible that the quantification of specific sedentary activities, e.g., TV viewing, may prove to be more robust than the measurement of physical activity. The potential for important interactions between inactivity and diet has also largely been ignored, yet it is plausible that sedentary lifestyles are associated with specific dietary habits, with regard to the macronutrient composition of the diet or eating frequency.

ECOLOGICAL STUDIES

Understanding the differences in the prevalence of obesity between countries, particularly in the developing versus developed world, is beset by methodological difficulties. No single study has attempted such an analysis, although the ecological data relating the prevalence of obesity to dietary intake; specifically, fat (2,15), sugar (12), and physical activity (7) have recently been independently reviewed.

Some studies have reported secular decreases in energy intake concurrent with increases in weight and or fatness, in both children and adults, and it has been inferred that this corresponds to a decrease in physical activity (e.g., 6,26,30,31). Only one study has attempted to simultaneously compare data on energy intake and physical activity with trends in obesity (22). In the United Kingdom, there has been an increase in the prevalence of obesity from 6 and 8% in men and women in 1980 to 17 and 20% in 1997. However, the National Food Survey (NFS) shows a decrease in household food intake, corrected for changes in confectionery, soft drinks, and alcohol consumption, of over 20%. It is probable that there have been increases in energy consumed outside the home over this period, but because this represents such a small proportion of total energy intake (11% at present), it is unlikely to account for the entire decrease in energy intake over this period. Moreover, the NFS data is supported by the accumulated results of cross-sectional dietary surveys which together also show a fall in reported energy intake (21). This implies that there have been even greater decreases in physical activity. Unfortunately, there are no direct measurements of secular trends in physical activity, but good data exist to show significant increases in the time spent watching TV (a proxy measure of leisure-time sedentary activities) and increased car ownership (a proxy measure for the decline in the personal energy cost of transport).

Secular trends in obesity can also be observed in the comparison of two cross-sectional studies in Finland (8). In a subsample of participants in the Finnmonica survey, detailed measurements of energy intake are available from 3-d food diaries and questionnaire-derived estimates of occupational activity, transport to and from work, leisure activity, and time spent sleeping. Over a 10-yr period the prevalence of overweight (BMI > 27 kg·m^{-2}) increased from 39 to 43% in men but was almost unchanged in women, increasing from 33 to only 34%. Energy intake declined by approximately 10% in both sexes, although when the data were edited to remove those clearly underreporting their habitual energy intake, the decrease was only 4%. There was a decrease in the energy cost of transport to work and other work-related activity. This was almost counterbalanced by an increase in the energy cost of leisure activity in women but less adequately in men. Thus, it could be argued either that the greater increase in leisure-time activity in women, or greater decrease in energy cost of work in men, has led to the disparity in the rates of increase in the prevalence of obesity, rather than differences in energy intake.

The analysis of secular trends in obesity is complicated by a multitude of other concurrent changes in lifestyle. However, an analysis of cross-sectional trends from large nationally representative surveys in the United Kingdom provides further supporting evidence of the importance of physical activity over and above energy intake (Figs. 1 and 2). There is a marked social class gradient in the prevalence of overweight and obesity, especially in women where it ranges from 11.1% in professional women to 23.6% in those who are partly skilled or unskilled (23). Despite differences

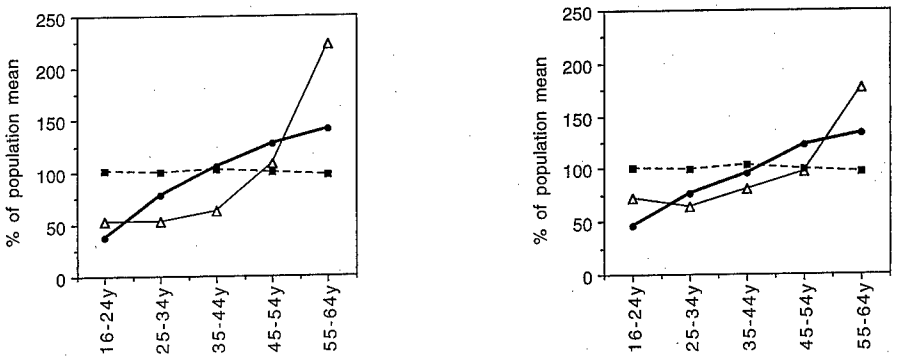

Figure 2—Relationship between the prevalence of obesity, energy intake, and inactivity by age. Men, *left panel*; women, *right panel*; obesity, *closed circles/solid line*; energy intake, *closed squares/dashed line*; inactivity, *open triangles/solid line*.

in the types of food consumed there are remarkably small differences in energy intake between groups (11). However, there are significant differences in reported physical activity, with 18% men and 21% women in social classes IV and V reporting no activity in the preceding 4 wk, compared with only 14% and 13% in social classes I and II (27). A similar analysis across the age range shows that the prevalence of obesity in men and women increases from 5.8% and 8%, respectively, in 16- to 24-yr-olds to 21.5% and 23.2% in 55- to 64-yr-olds (23). Between 16 and 44 yr, there is little or no relationship between the increasing prevalence of obesity and differences in either energy intake or levels of inactivity, but from 44 to 64 yr, there is a strong association between rising levels of inactivity and obesity in both men and women.

These ecological analyses, albeit using data from diverse sources and collected at different points in time, provide indirect evidence that sedentary lifestyles play an important and possibly dominant role in the etiology of overweight and obesity. However, these association studies, using data predominately collected in the United Kingdom, have not yet been replicated elsewhere. These data fall into Evidence Category C, being from observational studies only.

CROSS-SECTIONAL STUDIES

There are a number of relevant cross-sectional studies that include data on weight, BMI, or fatness and measurements of diet and physical activity. Studies that have considered the difference in diet and physical activity between groups of lean and obese subjects have been excluded from this analysis because of the high probability of *post hoc* changes in lifestyle as a consequence of obesity. However, this problem cannot be entirely eliminated from studies in which relative weight is used as a continuous variable. Cross-sectional studies identify associations rather than etiological agents.

Only two cross-sectional studies have been identified in children that report measurements of diet, physical activity, and weight (or fatness) (25,26). Each included approximately 700 children, but although there was a trend toward an association between low activity (or increased sedentary activities) and fatness, these relationships were not significant after adjustment for confounding variables. Sunnegardh et al. (26) reported that children, especially girls, whose parents had low levels of education had more body fat than those of better educated parents, whereas Shannon et al. (25) observed that the weak relationship between TV viewing and obesity was only apparent in the less affluent school districts.

Other cross-sectional analyses can be drawn from the baseline data collection for two of the prospective studies described below (9,13). In a small study of United States Caucasian subjects (142 men and 152 women) Klesges et al. (13) measured dietary intake using a food frequency questionnaire and physical activity by using the Baecke scale comprising 16 items covering work, sport, and nonsport leisure activities. They observed a strong positive effect of the proportion of dietary fat on BMI but no relationship with physical activity. For women, parental obesity was the strongest predictor of BMI, although this was not significant for men.

French et al. (9) studied 1913 women and 1639 men as part of the Health Worker Project conducted in 32 companies in Minnesota. Energy intake was measured using an 18-item food frequency questionnaire, based on food groups and physical activity using a 13-item exercise frequency questionnaire that included high intensity activities, moderate intensity activities, group and racquet sports, and occupational activity. For women, high intensity activities and walking were both significantly inversely related to body weight, as was alcohol intake. Consumption of soft drinks was positively associated with weight. For men, only high intensity activities were significantly inversely related to body weight. The consumption of sweets was also inversely correlated with weight, whereas dairy products, alcohol, and meat were positively related to body weight. In addition, previous dieting, participation in a weight loss program, or currently dieting were all associated with higher body weight. Thus, although offering some support to the hypotheses that the absence of aerobic activities and consumption of high-fat foods may contribute to weight gain, this study shows the importance of previous dieting history as a determinant of subsequent weight change. This is consistent with other studies (3,4) and suggests that the mechanism of this weight rebound (increased intake versus decreased expenditure) has not been adequately described by the available measures of diet and physical activity.

Thus, low levels of physical activity are frequently associated with an increased prevalence of obesity and vigorous

activity with a decreased prevalence of obesity. However, it is unclear whether this is a causal association or a *post hoc* effect. These data fall into Evidence Category C, being from observational studies only.

PROSPECTIVE STUDIES

There are eight prospective studies that include data on changes in weight, or fatness, along with measurements of energy intake and physical activity (3,9,13,16–18,24,28). However, drawing a consensus across studies is limited by their heterogeneity (Table 1). Most studies include adults only, although one refers to children (17) and one has studied the transitional phase from adolescence to adulthood, beginning with age 13, with a 15-yr follow-up (28). This study also has the longest follow-up period, whereas all others range from 2 to 7 yr. Most studies include 100–400 subjects (13,16,17,28), two include 3000–4000 subjects (9,18), and two have over 12,000 participants (3,24). The measurement of the key exposure variables, diet and physical activity, varies considerably. There are also important differences in the adjustment for potential confounding variables. Only two studies have specifically measured any sedentary pursuits, in each case TV viewing (3,17).

The statistical analysis also differs. Half the studies have used measurements made at a single time point to predict subsequent weight changes (13,16,17,24). The remaining studies have used both baseline and follow-up measurements in an analysis that effectively considers the change in weight in relation to the change in diet or physical activity (3,9,18,28). Both these approaches have their own limitations. By using only baseline measurements of exposure, there is a risk that changes in diet and/or physical activity will mask the associations with subsequent weight changes. Yet studies in which changes in diet and/or physical activity are used to explain changes in weight may be confounded by *post hoc* effects and do not allow any conclusions about cause or consequence. Nonetheless, prospective studies provide the most robust data to assess the relationship between physical activity and weight change.

The overall relationships between diet, physical activity, and other factors with respect to the risk of overweight or obesity are shown in Table 2. Most studies show some evidence of a link between low levels of physical activity and the risk of obesity. Twisk et al. (28) found an inverse relationship between physical activity and fatness (but not BMI). French et al. (9) and Rissanen et al. (24) found an inverse relationship between physical activity and weight change across all subjects, although in other studies the association is limited to subgroups of the population. In the study of United States male health professionals, there was evidence of both an inverse association with vigorous activity and a positive association with TV/VCR viewing, in men aged 45–64 yr but not in those $\geq$ 65 yr (3). In the studies of Klesges et al. (13) and Paeratakul et al. (18), the association was only observed for women and in the Women's Gothenburg study low levels of leisure-time physical activity were a risk factor for weight gain only in those women also consuming a high-fat diet (16). Maffeis et al. (17) found no association between physical activity and or TV viewing and the change in relative BMI in children.

The associations between dietary variables and the risk of obesity are more sporadic. Rissanen et al. (24) and Klesges et al. (13) both report a positive association between greater energy intake and weight gain in women, whereas Paeratakul et al. (18) and Coakley et al. (3) each found associations between the proportion of fat in the diet and weight gain in men. French et al. (9) found a relationship between some food groups and the risk of obesity, specifically dairy products, sweets, meat and French fries in women and sweets and eggs in men, but did not calculate specific energy or macronutrient associations. Maffeis et al. (17) found no association between diet and the change in relative BMI in children. Twisk et al. (28) observed that higher energy and macronutrient intakes were associated with greater lean body mass, but not fatness or BMI. This presumably relates to the increased dietary needs for growth in this group, who were followed from adolescence through to adulthood.

Many studies observe that smoking cessation is strongly associated with weight gain (3,13,24), although this was not seen in the study of Paeratukul et al. (18). Previous voluntary weight loss is also a strong predictor of subsequent weight gain in studies in which this has been recorded (3,9). This is consistent with the commonly observed phenomenon of weight rebound. These factors do not explain the primary mechanism of weight change, but they may be important within the context of preventative strategies to allow the development of interventions that are more precisely targeted at high risk groups.

Many prospective studies offer some support to that hypothesis that physical activity can attenuate the rate of weight gain. However, it is difficult to precisely quantify the contribution of physical activity within the context of other potentially important factors, especially diet. Again these data fall into Evidence Category C.

THE BALANCE OF EVIDENCE

The evidence reviewed in this paper shows clearly that low levels of activity are associated with overweight and obesity. This is reinforced by data from other studies (reviewed by DiPietro (5)), which have measured physical activity, but not diet, in relation to weight change. There is good reason to believe that this may be a causal relationship, but definitive statements are limited by the methodological flaws of these studies, particularly with respect to the quantification of physical activity and dietary factors. However, a recent analysis of the errors generated by imprecise measurements of physical activity in the study of weight change have shown that there is a substantial underestimate of the relative importance of physical activity levels (32). Thus, the evidence presented here is likely to represent a conservative assessment of the importance of inactivity as a contributor to weight gain.

TABLE 1. Design of prospective studies of weight changes in relation to diet and physical activity.

Author:	Rissanen et al., 1991 (24)	Klesges et al., 1992 (13)	French et al., 1994 (9)	Lissner et al., 1997 (16)	Coakley et al., 1998 (3)	Paeratukul et al., 1998 (18)	Maffies et al., 1998 (17)	Twisk et al., 1998 (28)
Population	12 Finnish communities	U.S., middle-class, mostly Caucasian	32 companies in Minnesota, U.S.	Women in Gothenburg, Sweden	U.S. male health professionals	Representative sample from 8 provinces of China	Italian school-children	Amsterdam Growth and Health Study
Number	6504 male, 6105 female	142 male, 152 female	1639 male, 1913 female	361 female	19,478 male	1636 male, 1848 female	58 male, 54 female	83 male, 98 female
Age (yr)	25–64	Mean: 34.8 male 33.1 female	Mean: 38	38–60	40–75	20–45	Mean: 8.6	13
Follow-up (yr)	4–7 (median = 5.7)	2	2	6	4	2	4	1, 2, 3, 4, 8, 15
Obesity Index	Weight: in light clothing; height: no shoes	Weight and height: light clothing, no shoes	Weight and height: clothed, with shoes	Measured weight and height	Self-reported weight and height	Weight and height: light clothes, no shoes	Measured weight and height: underwear only, no shoes	Measured weight and height, skinfold thickness at 4 sites
Dietary Assessment	In one-sixth of sample, dietary history over preceding year: energy intake, macronutrients, coffee, and alcohol	Food frequency questionnaire over preceding year: energy intake and % fat.	18-item food frequency questionnaire: food groups	Diet history including frequency of 69 items: energy and % fat	Food frequency questionnaire mid-term: energy-adjusted fat intake.	3 × 24 h dietary recall: energy and % fat	Diet history with parents and child: energy and macronutrients	Cross-check diet history: energy and macronutrient intake
Physical Activity Assessment	Leisure-time activity (at follow-up only); frequent, occasional, rare.	Baecke scale; 16 items including work, sport, and nonsport leisure	13 item exercise frequency questionnaire: high intensity, group and racket sports, and occupational	Occupational activity scored 1–4; leisure activity scored as sedentary, somewhat active or most active	Questionnaire: Hours/week in vigorous activity, hours/week watching TV or VCR	Occupational activity only: scored as sedentary, moderate, or strenuous	Questionnaire: sleep, sport, planned exercise, TV viewing, time at school	Structured interview: total activity expressed as METS·wk^{-1}
Confounders	Age, education, health status, marital status, parity, smoking, alcohol, coffee	Age, smoking, alcohol, parental obesity, pregnancy	Age, education, marital status, occupation, smoking, treatment group, previous or current dieting	Age, smoking	Age, smoking, dieting history, eating frequency	Urban/rural residence, age, smoking, education, occupation, income	Age, pubertal status, parental BMI	Smoking, alcohol
Statistics	Logistic regression and analysis of covariance	Stepwise regression adjusted for baseline weight	Linear regression adjusted for baseline measurements	Multivariate regression and linear regression	Multiple regression adjusted for baseline measurements	Fixed effects model adjusted for baseline measurements	Various multivariate regression models	Auto-regression model adjusted for prior measurements

TABLE 2. Results of prospective studies described in Table 1.

Author:	Rissanen et al., 1991 (24)	Klesges et al., 1992 (13)	French et al., 1994 (9)	Lissner et al., 1997 (16)	Coakley et al., 1998 (3)	Paeratukul et al., 1998 (18)	Maffies et al., 1998 (17)	Twisk et al., 1998 (28)
Population	12 Finnish communities	U.S. middle-class, mostly Caucasian	32 companies in Minnesota, U.S.	Women in Gothenburg, Sweden	U.S. male health professionals	Representative sample from 8 provinces of China	Italian school-children	Amsterdam Growth and Health Study
Outcome variable	1. Increase in weight; 2. relative risk of gaining ≥5 kg	Increase in weight	Increase in weight	Increase in weight	Increase in weight	Increase in BMI	Increase in relative BMI	Increase in BMI; 2. change in sum of skinfold thicknesses (SSF); 3. change in LBM calculated from SSF
Relationships between outcome variable and dietary factors	Men: not significant; women: higher baseline energy and macronutrient intake	Men: increase in % fat; women: higher baseline energy intake; higher baseline % fat; increase in energy intake	Men: increase in consumption of eggs or sweets; women: increase in consumption of French fries, sweets, dairy products, or meat	High baseline energy and % fat intake in sedentary leisure group only	45–64 yr: eating between meals and energy-adjusted fat intake	All subjects: Increases in % fat, but only significant for men, not women, when analyzed separately	None	Absolute energy, fat an carbohydrate intake in men associated with greater LBM, but not SFF of BMI.
Relationships between outcome variables and physical activity	Men and women: infrequent physical activity at follow-up	Men: high baseline sports activity; women: low levels of work or leisure activity	Men: decrease in high intensity activity; women: decrease in walking or high intensity activity	Low activity in high % fat group only	45–54 yr: TV/VCR viewing; 45–64 yr: low levels of vigorous activity	All subjects: decrease in occupational physical activity, but only for women, not men, when analyzed separately	None	Low levels of physical activity associated with greater SSF and lower LBM, but not BMI
Other factors significantly associated with outcome variable	Smoking cessation, low education, marriage, high coffee consumption	Women: smoking cessation, pregnancy	Men: previous participation in weight loss program; women: previous participation in weight loss program or baseline dieting		Smoking cessation; previous weight loss			
Other comments		Men: higher baseline energy intake and increases from baseline associated with weight *loss*	Final model accounted for 10% of variance in change in body weight, excluding baseline body weight in men and 9% in women	Significant interaction between dietary fat and inactivity	Smoking and dieting associated with weight loss	Changes in smoking habits *not* related to weight change; no effect of socioeconomic variables (place of residence, education, occupation, income)	Parental BMI accounted for 13.5% of the variance in children's relative BMI	Change in BMI is poor outcome variable during growth; No statistical adjustment for time-intervals between repeated measurements.

RESEARCH ISSUES

This analysis of the evidence relating to the role of sedentary lifestyles in the etiology of overweight and obesity has highlighted the inadequacies of data currently available and the limitations of existing studies. This leads onto a number of key research issues.

Sampling. The studies to date differ in the demographics of the population sample. Of the prospective studies, only that of Paeratakul et al. (18) is truly randomly selected, and it is rare that the sample accurately reflects the composition of the wider population with respect to gender, age, and ethnicity. The recruitment procedures for studies need to be carefully documented and the characteristics of subjects who drop out identified. There may also be a need for studies that specifically address certain groups of the population who may be at a critical period for the development of obesity. By selecting homogeneous samples at high risk of weight change, e.g., ex-smokers, postpartum women, successful weight-losers, it may be possible to identify important etiological factors using a smaller population base.

Standardization. The diversity of measurements of exposure and outcome variables in different studies limits their comparability and precludes any overall analysis that may have greater statistical power than individual studies. Much greater use could be made of studies working to common measurement and design protocols.

Outcome variables. Most studies use weight and height, or the BMI, as a measure of obesity. However, this fails to distinguish between individuals in whom the excess weight is fat and those who are particularly muscular. Studies in which fatness has been measured have sometimes revealed different associations between BMI or fatness and the exposures under study, especially in children, where the relationship between weight and fatness is more variable (28). Most of the reference methods to measure body composition are unsuitable for large epidemiological studies, whereas prediction methods tend to be less accurate. Other measurements such as waist circumference (fatness) or mid-arm muscle mass (leanness) need to be evaluated to potentially discriminate between individuals of similar BMI but very different body composition.

Exposure variables. It is necessary to more precisely define the exposure variables of interest. In dietary studies, the emphasis has been upon total energy, or more recently, macronutrient intake. Issues such as the energy density of the diet have not been included in any of the studies in this review, but this may be an important dietary variable (19). Aspects of eating behavior may also be relevant, e.g., eating frequency, eating alone or in company, and eating out of the home. With regard to physical activity, it is still unclear which dimensions of physical activity are the most relevant to the prevention of weight gain, e.g., total energy expenditure, bouts of vigorous activity, frequency of activity, or the proportion of time spent in sedentary activities. Understanding the mechanism by which dietary factors and physical activity, or their interaction, may influence the development of obesity is integral to the future design of methods to test the relationship between physical activity and obesity.

The quantification of diet and physical activity is beset by methodological errors that tend to be biased in favor of underreporting of intake and overreporting of physical activity (14). Dietary records of energy intake must be evaluated against physiologically derived estimates of energy requirements (10), but in considering their contribution to body weight or weight change, there is the added difficulty that the reported errors appear to increase with the level of obesity (1). However, this procedure only considers total energy and biomarkers for other specific components of dietary intake are also required. Estimates of physical activity by many of the questionnaires used in earlier studies have been shown to relate more closely to physical fitness than habitual activity (29). More sensitive questionnaires are required, validated against reference methods to measure physical activity. Alternatively, there is scope for the development of direct measurements of physical activity which remove the element of subjective reporting. Technological developments in heart-rate monitoring and/or accelerometers may shortly offer a viable solution to large-scale measurements of physical activity and combined with appropriate software may yield data on both energy expenditure and patterns of physical activity. In this respect, equal attention should be paid to the quantification of sedentary pursuits, which are not necessarily the reciprocal of measured activities and perhaps require independent measurement procedures.

Analysis of confounders. There are many factors that may confound simplistic associations between diet or physical activity and obesity. This is a particular problem in prospective studies, where the relatively precise documentation of factors such as previous weight loss or smoking status may appear to be more strongly associated with weight change than the imprecise estimates of the direct modulators (energy intake and energy expenditure/physical activity). However, the proportion of the variance in weight change that can be accounted for is modest, even in the most comprehensive analyses, suggesting that these studies are either failing to account for important variables or the level of measurement imprecision is too great to reliably assess the true effect of these factors.

Repeated measures. Obesity is a chronic condition that develops over many years. During its incubation, there may be many different etiological agents acting sequentially or in concert. Long-term prospective studies that make repeated measurements of both exposure and outcome variables will be particularly powerful. However, careful attention needs to be paid to the design and statistical analysis of such studies to generate the most meaningful data. Additional problems arise in studies that bridge childhood and adulthood because the nature of the outcome variable may need to change. Growth is associated with physiologically orchestrated changes in both fatness and leanness, which lead to variable relationships between BMI and fatness, whereas in adulthood the majority of weight change is due to changes in body fat and thus changes in BMI are a

reasonable proxy for changes in fatness at a population level.

Statistical considerations. Given the measurement imprecision in the assessment of both exposure and outcome variables, the strength of the observed relationship will tend to be diminished. Appropriate statistical procedures to account for this attenuation need to be developed. This will help to refine the association between physical activity and obesity and also to predict the likely outcome of specific intervention targets.

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Physical activity in the prevention of obesity: current evidence and research issues

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ABSTRACT

DiPIETRO, L. Physical activity in the prevention of obesity: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S542–S546, 1999. **Purpose:** The relation between habitual physical activity and the prevention of overweight and obesity in adults based on the evidence from the epidemiologic literature is described. **Methods:** Literature was reviewed of current findings from large population-based studies of forward directionality in which physical activity was considered as a primary study factor. **Results:** The longitudinal evidence suggests that habitual physical activity plays more of a role in attenuating age-related weight gain, rather than in promoting weight loss. Moreover, recent data suggest that increasing amounts of physical activity may be necessary to effectively maintain a constant body weight with increasing age. **Conclusion:** Over decades, small savings in excess weight gain accumulate into net savings that may be quite meaningful with regard to minimizing the risk associated with obesity-related disorders. The question remains as to how important maintaining a constant body weight through middle age and into older age is to healthy, already-active people of normal body weight. **Key Words:** EPIDEMIOLOGY, EXERCISE, LONGITUDINAL, OVERWEIGHT, RISK

Recent survey data from the third National Health and Nutrition Examination Survey (NHANES III) suggest that overweight [i.e., "preobesity," defined as a body mass index (BMI = $kg \cdot m^{-2}$) = 25.0–29.9] is present in approximately 32% of the adults living in the United States (7). The overall prevalence of overweight currently is highest in men and women age 60–69 yr (45% and 34%, respectively) and then is progressively lower at older ages (7,11). Trend data further suggest that the population as a group, and particularly African-American women of lower socioeconomic status, is becoming heavier with time and that the increase in prevalence of overweight may be most accelerated among those individuals who are already overweight or obese (11,12). Among respondents to the NHANES-I (1971–1975) Epidemiologic Follow-up Study (1981–1984) who were not overweight (i.e., BMI $\geq$ 27.8 for men and 27.3 for women) at baseline, the risk of becoming overweight during the 10-yr follow-up was similar in men and women and was highest among adults aged 35–44 yr (16.3% among men and 13.5% among women) (21). The cumulative incidence of major weight gain ($\geq$5 kg), however, was higher in women compared with men and highest among adults aged 25–34 yr (3.9% among men and 8.4% among women). Recently, Flegal and colleagues (7) report a marked increase in the prevalence of "obesity" (i.e., BMI $\geq$ 30 $kg \cdot m^{-2}$) between NHANES II, 1976–80 (14.5%), and NHANES III, 1988–94 (22.5%), which corroborates trends observed internationally.

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CURRENT STATUS OF KNOWLEDGE

Question 1. How Effective Is Physical Activity in Reducing Overweight and Obesity?

Evidence statement. Numerous intervention studies have explored the impact of exercise training of various intensities on the reduction of weight and body fat (see 5 for review). We can conclude from these studies that: 1) physical activity affects body composition and weight favorably, by promoting fat loss, while preserving lean mass; 2) the rate of weight loss is positively related to the frequency and duration of the exercise session, as well as the duration of the exercise program, thereby suggesting a dose-response relationship; and 3) although, the rate of weight loss resulting from increased physical activity is relatively slow, physical activity may nonetheless be a more effective strategy for long-term weight regulation than dieting alone (2) (evidence category B or C).

Less is known about how physical activity patterns affect *attained weight* and *weight gain* among the general population—data are especially scarce among younger (<18 yr) and older (>55 yr) age groups, as well as for minority populations.

Observational studies. The inverse association between physical activity or exercise and body weight has been reported in many cross-sectional epidemiologic studies (see 5 for review). Indeed, these cross-sectional, population-based studies consistently report lower weight or body mass with higher categorical levels of self-reported physical activity—especially for activities of higher intensity, presumably due to the better reporting of these activities. Interpreting cross-sectional data, however, is difficult, because the directionality of the association between physical activity and weight cannot be determined. Naturally, physical activity patterns and choices may affect weight, just as weight may influence physical activity, and both may be heavily influenced by genetic factors (1).

Longitudinal or cohort studies (i.e., those with forward directionality) may give better information on the etiologic impact of physical activity on the *risk* or *development*, of excess weight gain over time because, by definition, the exposure (physical activity), measured either prospectively or retrospectively, must precede the outcome. Given the increasing prevalence of overweight in the population and the health consequences associated with this condition, its primary prevention therefore assumes considerable public health significance. Unfortunately, there is a paucity of longitudinal data that examine the role of physical activity on weight gain among the general population. Therefore, the purpose of this paper is to present the current evidence of the epidemiologic relation between habitual physical activity and the prevention of overweight and obesity. Accordingly, we present findings from large population-based studies of forward directionality in which physical activity is considered as the primary study factor on the risk and development of overweight. These findings are all evidence category C, in that they are based on observational data.

Question 2. Does Regular Physical Activity Prevent Weight Gain in the Population?

Evidence statement. The hypothesis that physical activity affects body weight inversely is logical, but observational population-based data on the longitudinal relationship between these two variables are somewhat confusing (4,6,8–10,12–15,18,21). Cross-sectional associations between physical activity or fitness and body weight are stronger than those seen longitudinally (3,5), and although there is evidence of a relation to *attenuated weight gain*, it is not clear that increased physical activity actually prevents or reverses age-related weight gain at the population level (Table 1) (evidence category C).

Several large-scale observational studies have determined a relationship between higher baseline levels of physical activity or improvements in physical activity and either attenuated weight gain (4,6,8–10,12–14) or a lower odds of a significant weight gain (3,6,15,21). Recently, Ching and colleagues (3) studied the relationship between both reported physical activity and a direct indicator of sedentary behavior—TV/VCR viewing—and overweight (i.e., BMI $\geq$ 27.3 kg·m^{-2}) in a large cohort of male health professionals. Cross-sectional analyses of the baseline data yielded results similar to other cross-sectional studies of physical activity and overweight; that is, a strong inverse graded association between quintile of physical activity (MET h·wk^{-1}) and a strong positive gradient between TV/VCR viewing (h·wk^{-1}) and the prevalence of overweight. After 2 yr of follow-up, higher baseline levels of physical activity and lower levels of TV/VCR viewing remained independently related to a lower risk of becoming overweight. The magnitude of the longitudinal relation was much smaller, however, than what was seen cross-sectionally with these same data, presumably due to the relatively lower cumulative incidence of overweight (~5%) over the 2-yr follow-up in that particularly select cohort of male health professionals.

Four-year follow-up data from the Male Health Professionals Study (4) and 2-yr follow-up data from the China Health and Nutrition Survey, 1989–1991 (14) support the notion of an inverse longitudinal relation between increased physical activity and weight gain. Indeed, middle-aged men in the Male Health Professionals study who increased vigorous activity, decreased TV viewing, and stopped eating between meals actually lost 1.4 kg over 4 yr, compared with a 1.4-kg weight gain among the entire study cohort. Further, recent findings from DiPietro et al. (6) in the Aerobics Center Longitudinal Study (ACLS) cohort and Lewis et al. (12) in the biracial CARDIA cohort show that improved cardiorespiratory fitness relates inversely to weight change. Indeed, improvements in treadmill time between the first and second exam significantly minimized weight gain over the respective 7.5-yr and 7-yr follow-up periods. These results were observed in both sexes, although the magnitude of the relationships were stronger and more stable in men than in women in the ACLS cohort. The findings in all four studies (4,6,12,14) were not affected by age, height, baseline weight, baseline fitness or activity level, smoking, number of clinic visits, and length of follow-up.

A recent cross-sectional analysis by Williams (19) on data from the National Runners' Health Study, suggest that substantial increases in activity level are necessary to maintain body weight with age. The ACLS (6) and The Male Health Professional Follow-up cohort data (4) corroborate these findings in that even among the fitter (6) or most vigorously active (4) men, *improvements* in treadmill time, fitness class, or vigorous activity were necessary for weight loss over time; indeed, men who were fit and *remained* fit, or were vigorously active and *remained* so, actually gained small amounts of weight over the follow-up, even after adjustment for age and smoking. Further, *improvements* in fitness class among the ACLS cohort demonstrated a stronger protective effect on the odds of a $\geq$ 5 or $\geq$ 10 kg weight gain in men than did maintaining the same level of fitness.

Williamson et al. (21) determined the association between the 10-yr change in physical activity level and the magnitude of *concurrent* weight change in more than 9,000 adults responding to the NHANES I Epidemiologic Follow-up Study (1971–1975 to 1982–1984). Their results suggest that even relative to people who stay very active over time, sedentary people who increase their activity can *minimize*

TABLE 1. Summary of longitudinal epidemiologic studies of physical activity and weight change.

Study	Reference	Population	Design/Methods	Results	Effect Size
DiPietro et al. (6)	*Int. J. Obes.* 22:55–62, 1998	Middle-age cohort of 4599 men and 724 women	Prospective (7.5-yr follow-up); weight measured at 3 time points; fitness determined by GXT	Improvements in fitness related to attenuated weight gain and lower odds of ≥5 or ≥10 kg gain in both ♂ and ♀	For each 1-min improvement in TMT, $b = -0.60$ kg ($P < 0.001$) for both ♂ and ♀; $OR_{5kg} = 0.86$ for ♂ and 0.91 for ♀ ($P < 0.01$); $OR_{10kg} = 0.79$ for both ♂ and ♀ ($P < 0.01$)
Coakley et al. (4)	*Int. J. Obes.* 22:89–96, 1998	Cohort of 19,478 male health professionals (40–75 yr)	Prospective (4-yr follow-up); self-reported weight, activity, and TV viewing	Increased vigorous activity related to attenuated weight gain and even weight loss	Each 1.5 $h \cdot wk^{-1}$ increase over 4 yr related to a 0.2-kg weight loss ($P < 0.05$) in ♂ 45–54 yr
Paeratakul et al. (14)	*Int. J. Obes.* 22:424–431, 1998	Cohort of 3,484 working adults (20–45 yr) in China	Prospective (2-yr-follow-up); reported level of occupational activity and measured weight	Increased activity related to attenuated weight gain in ♀ only	For each categorical increase in occupational activity, $b = -0.12$ ($P < 0.02$)
Lewis et al. (12)	*Am. J. Public Health* 87:635–642, 1997	Biracial younger cohort of 1823 men & 2083 women (18–30 yr)	Prospective (7-yr follow-up); weight measured at 4 time points; fitness determined by GXT	Decreases in fitness related to increased weight gain in both ♂ and ♀	For each 1-min decline in TMT, $b = 1.5$ kg for ♂ and $b = 2.1$ kg for ♀ ($P < 0.05$)
Ching et al. (3)	*Am. J. Public Health* 86:25–30, 1996	Cohort of 22,076 male health professionals (40–75 yr)	Cross-sectional and prospective (2-yr follow-up); self-reported weight, activity, and TV viewing	Higher baseline levels of activity related to lower risk of becoming overweight[a]	RR (>14.5 vs ≤6.9 $METS \cdot wk^{-1}$) = 0.80 ($P < 0.05$); each 10-MET increase in weekly activity predicted a 0.30-U decrease in 2-yr BMI
Kawachi et al. (9)	*Am. J. Public Health* 86:999–1004, 1996	Cohort of 121,700 female nurses (40–75 yr)	Prospective (2-yr follow-up); self-reported weight and activity	♀ who quit smoking *and* increased activity gained less weight than those who simply quit smoking over 2 yr	Compared with smokers, ♀ who simply quit gained 2.3 kg more; those who quit and increased activity 8–16 $METS \cdot wk^{-1}$ gained only 1.8 kg more ($P < 0.05$) over 2 yr
French et al. (8)	*Int. J. Obes.* 18:145–154, 1994	Cohort of 1639 male and 1913 female employees (27–50 yr)	Prospective (2-yr follow-up); measured weight, self-reported activity	Higher levels of activity related to attenuated weight gain in both ♂ and ♀	For each added session of walking or high-intensity activity $b = -0.86$ and -3.54 kg, respectively for ♂ and $b = -1.76$ and -1.39 kg for ♀
Williamson et al. (21)	*Int. J. Obes.* 17:279–286, 1993	Representative cohort of 3515 men and 5810 women (18–74 yr) in U.S.	Prospective (10-yr follow-up); reported activity Δ and measured weight Δ studied concurrently	Low activity at follow-up related to major weight gain (>13 kg) occurring over previous 10 yr	OR (low vs high activity) for major weight gain = 3.1 for ♂ and 3.8 in ♀ ($P < 0.05$)
Klesges et al. (10)	*Am. J. Clin. Nutr.* 55:818–822, 1992	Selected sample of 142 men and 152 women (24–52 yr)	Prospective (2-yr follow-up); reported activity Δ and measured weight Δ studied concurrently	Decrease in work activity over 2 yr related to greater weight gain in ♀ only	For each unit decrease in work activity, $b = 5.87$ kg ($P < 0.02$) over 2 yr
Owens et al. (13)	*Circulation* 85:1265–1270, 1992	Cohort of 507 women (42–50 yr)	Prospective (3-yr follow-up); reported activity and measured weight	Higher baseline activity related to attenuated weight gain	For each $_{log}$ $kcal \cdot wk^{-1}$ increase in activity, $b = -5.84 \cdot 10^{-4}$ ($P < 0.01$)
Voorrips et al. (18)	*Int. J. Obes.* 16:199–205, 1991	Selected sample of 45 older women (71 yr)	Retrospective cohort; recall of activity and weight at ages 12, 25, 40, and 55 yr and currently	♀ currently active had a lower weight index[b] at age 25, 40, 55 yr and currently	Mean difference in the weight index in currently active vs sedentary was 2.7 U at 25 yr, 2.6 at 40 yr and 3.5 at 55 yr ($P < 0.01$)
Rissanen et al. (15)	*Eur. J. Clin. Nutr.* 45:419–430, 1991	Large cohort of Finnish men and women (25–64 yr)	Prospective (5-yr follow-up); measured weight; self-reported activity	Inverse graded relation between activity category (frequent; occasional; rare) and risk of 5-kg gain	RR = 1.0, 1.5, 1.9 for ♂; and RR = 1.0, 1.5, 1.8 for ♀; ($P < 0.05$)

GXT, graded exercise challenge; TMT, treadmill time; BMI, body mass index ($kg \cdot m^{-2}$).

[a] Overweight defined as BMI ≥ 27.8 $kg \cdot m^{-2}$.

[b] Weight index calculated from the sum of perception scores (1 = low BMI to 5 = high BMI) from front- and side-view silhouettes and comparison to peers (1 = thinner than; 3 = same as; 5 = fatter than) at ages 12, 25, 40, and 55 yr.

♂, men; ♀, women.

the excess risk of major weight gain compared to people who decrease their activity, thus supporting the contention of lower risk of becoming overweight with an increase in level of activity over time.

Thus, the longitudinal evidence linking habitual physical activity to the prevention of excess weight gain among the general population or more selective populations seems clear. Most of these findings suggest that physical activity or fitness plays more of a role in attenuating age-related weight gain and preventing significant weight gain, rather than in promoting weight loss. Whereas the magnitude of the relation of improvements in physical activity or fitness and attenuated weight gain appears small in several studies (resulting in small effect sizes), over decades these small savings in excess weight gain accumulate into net savings that are quite meaningful with regard to minimizing the risk associated with obesity-related disorders (9). Moreover, results from the ACLS (6) and the Male Health Professional Follow-up (4) cohorts, as well as cross-sectional trend data from Williams (19) suggest that increasing amounts of physical activity may be necessary to effectively maintain a constant body weight with increasing age. The question remains as to how important maintaining a constant weight through middle and into older age is to healthy, already-active people of normal body weight.

RESEARCH PRIORITIES

Clearly, there is a need for additional longitudinal research on the relation between physical activity and weight gain at the population level. Suggestions for research priorities comprise several areas ranging from descriptive epidemiology, to the testing of more complex etiologic hypotheses in the population, to the linking of population-based research to that which is laboratory- or clinical-based, in order to test more mechanistic hypotheses.

1. Tracking or Surveillance Studies

One of the primary limitations of the longitudinal, epidemiologic research done thus far is the lack of multiple measures of both physical activity and body weight. Typically, physical activity is determined at baseline, with body weight measured at baseline and then again at follow-up. Surveillance studies that track physical activity patterns and body weight over 10–20 yr with measurements of both taken at 2- to 5-yr intervals are extremely important to describe accurately the contribution of physical activity to long-term weight regulation. Naturally, more sophisticated statistical techniques are needed to analyze these multiply repeated data appropriately. Random-effects regression modeling (16) is a technique that models on the trajectory of change in weight over several consecutive years for each individual and then computes the average effect of physical activity level on those trajectories, while simultaneously controlling for changes in other influential variables.

2. Powerful Longitudinal Studies of Women

To date, the majority of studies of physical activity and body weight have involved larger populations of men than women (3,4,6,8,10,17,19). Several investigators have noted that women (particularly premenopausal women) are more resistant to weight change with physical activity due to a greater proportion of less lipolytically responsive gluteofemoral adipose tissue. Because women seem to have the highest risk of substantial weight gain through middle age, public health interest would render it logical to study this group more intensely than men.

3. Longitudinal Studies through Transitions

Currently we have no longitudinal data on infants, adolescents, and young and older adults with regard to physical activity and weight change. These recommended studies would be further enhanced if the particular cohort were followed through a period of natural change in their physiology—for example: 1) infancy through adolescence; 2) adolescence to age 30 yr; 3) middle age to older age (65 yr); and 4) pre- through peri-, through post-menopause. In addition, few population-based studies have assessed the association between these variables in older (i.e., $\geq$65 yr) men and women. Thus, little is known about the benefits of physical activity to weight *maintenance* (i.e., the prevention of weight loss) and subsequent health outcomes in older age.

3. Improved Epidemiologic or Statistical Methods

Inconsistent or confusing longitudinal relationships may be a consequence of several methodological issues, which make interpreting the data quite difficult. These issues include: a) a low prevalence of higher-intensity physical activity in the general population; b) measurement error with regard to self-reported activity, especially that of lower-intensity; c) inappropriate time-frame of the physical activity assessment; d) effect modification by age, sex, and weight status; and e) failure to adjust for important statistical confounders, such as diet and smoking. Thus methods of assessing lower-intensity activity with accuracy (that is, validity *and* precision) over several time points, as well as sample sizes that are large enough to allow ample power for stratification by age, sex, and/or weight status and for the statistical control of important confounding variables are requisite to well-designed longitudinal studies of the population.

4. Linking Field-Based with Laboratory- or Clinical-Based Studies

Epidemiologic studies have been extremely important to our understanding of the etiologic relationships among numerous risk factors and disease or functional outcomes as they are observed in the general population. What these studies lack, however, is the ability to identify the specific physiologic mechanisms that explain these observed statistical associations. By drawing representative subsamples from large cohort studies for more intensive laboratory or

clinical study, we can begin to: a) map genetic determinants of weight regulation in order to differentiate their impact from behavioral determinants; b) identify signaling pathways and mechanisms of energy balance from the cellular- to the applied, whole-body-level in children and adults; and c) determine the magnitude of impact of structural (anatomical) changes on functional changes at the whole-body level. This information can then be translated back to the population level in terms of specific interventions designed to prevent substantial weight gain and obesity throughout the lifespan.

In memory of my mentor Ethan R. Nadel, Ph.D., FACSM.

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Physical activity in the treatment of the adulthood overweight and obesity: current evidence and research issues

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ABSTRACT

WING, R. R. Physical activity in the treatment of the adulthood overweight and obesity: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S547–S552, 1999. **Purpose:** The purpose of this paper is to review the evidence on the role of physical activity in the treatment of adult overweight and obesity. Three specific questions are addressed: (1) Does exercise alone produce weight loss? (2) Does exercise in combination with diet produce greater weight loss than diet only? and (3) Does exercise in combination with diet produce better maintenance of weight loss than diet alone? **Methods:** The literature initially identified by the Expert Panel on Clinical Guidelines for the Treatment of Obesity, three key meta analyses, and additional literature searches were used to identify randomized trials related to the three aforementioned topics. These articles were reviewed and tabulated. **Results:** Six of 10 randomized studies found significantly greater weight loss in exercise alone versus no treatment controls. The magnitude of the effect averaged 1–2 kg. Only 2 of 13 studies found significant differences in initial weight loss for diet plus exercise versus diet only, although almost all studies pointed in this direction. Six studies were identified with maintenance periods of at least 1 yr. In two of the six there were significant long term differences favoring diet plus exercise, but in every study considered the direction of the difference favored diet plus exercise. Other strong evidence showing benefits of exercise for long-term weight loss comes from correlational analyses which consistently find that those individuals who report the greatest exercise have the best maintenance of weight loss. **Conclusions:** Randomized trials consistently show benefits of exercise for weight loss, but the effects are often modest. This may result from small sample sizes, short study duration, and poor adherence to the exercise prescriptions. To better define the doses and types of exercise that will promote long-term weight loss, it is necessary to develop better ways to measure exercise and promote adherence to exercise.
Key Words: WEIGHT LOSS MAINTENANCE, EXERCISE, PHYSICAL ACTIVITY, OBESITY

The combination of diet plus exercise is recommended for obese patients desiring to lose weight. The purpose of this paper is to review the empirical literature supporting this recommendation. Specifically, this literature review will address three important questions:

- Does exercise alone produce weight loss?
- Does exercise in combination with diet produce greater weight loss than diet alone?
- Does exercise in combination with diet produce better maintenance of weight loss than diet alone?

The National Heart, Lung, and Blood Institute, in combination with the National Institute of Diabetes and Digestive and Kidney Disease, recently convened an Expert Panel on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults (22). Since this panel addressed issues similar to the ones being addressed in this paper, their literature search was used extensively in preparing this report. The Expert Panel selected randomized controlled trials which involved 4 months or more of treatment regardless of the sample size involved. Thirteen articles were used for the comparison of exercise versus no treatment (2,4,8,9,12,13,16–18,27,33,37,42) and 15 for the comparison of diet plus exercise to diet alone (1,2,4,5,11–13,20,23,33–36,39,43). The Expert Panel based their conclusions on (a) the number of studies that favored exercise versus no exercise and (b) the mean differences between the two conditions. These articles were re-reviewed for the present paper, and a few other studies meeting the Expert Panel's criteria were identified. The present review used a different criterion for evaluating the effect of exercise on weight loss, namely, the number of studies that showed significant differences favoring exercise. In addition three meta-analyses related to this topic were used in developing this paper (3,10,21).

DOES EXERCISE ALONE PRODUCE WEIGHT LOSS?

The Expert Panel concluded that in 10 of the 12 studies that met their review criteria the exercise condition had larger weight losses than the control, with a mean difference in weight loss of 2.4 kg (or a BMI difference of 0.7 $kg \cdot m^{-2}$).

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TABLE 1. Weight loss in exercise alone vs no treatment control group.

	Duration	Exercise Alone		Control		Signif.[a]
		N	Weight Loss	*N*	Weight Loss	
Anderssen 1995 (2)	1 yr	49 M/F	−0.9 kg	43 M/F	+1.1 kg	*
Hammer 1989 (12)	4 months	8 F	−6.7 kg	4 F	−5.8 kg	*
Hellienus 1993 (13)	6 months	39 M	−0.3 BMI	39 M	+0.3 BMI	*
Katzel[b] 1995 (16)	9 months	49 M	−1%	18 M	+0.5%	NS
King[c] 1991 (17)	1 yr	29–35 F	−0.6 to +0.4 BMI	34 F	0 BMI	NS
		40–45 M	−0.9 to −0.2 BMI	41 M	+0.1 BMI	NS
Rönnemaa 1988 (27)	4 months	13 M/F	−2.0 kg	12 M/F	+0.5 kg	*
Stefanik 1998 (33)	1 yr	43 F	−0.4 kg	45 F	+0.8 kg	NS
		47 M	−0.6 kg	46 M	+0.5 kg	NS
Verity 1989 (37)	4 months	5 F	−2.1 kg	5 F	−2.9 kg	NS
Wood 1983 (41)	1 yr	48 M	−1.9 kg	33 M	+0.6 kg	*
Wood 1988 (42)	1 yr	47 M	−4.0 kg	42 M	+0.6 kg	*

[a] Significance of difference in weight loss for exercise vs control.
[b] Data interpreted from graph.
[c] This study included 3 exercise conditions: high intensity group based/high intensity home based/low intensity home based. None differed in weight loss from controls.

In the one meta-analysis that addressed this issue, Garrow and Sommerbell (10) reached a similar conclusion: that exercise produces a slight weight loss in men (net difference = 3.0 kg) and in women (net difference of 1.4 kg).

Table 1 shows the results of 10 randomized controlled trials comparing exercise interventions to no-treatment controls that were considered in the current review. The study by Bertram et al. (4) used a diet condition as the "control" group and thus this study has been excluded. Another study used by the Panel provided follow-up data from the King study (18); at the point of follow-up, the control group was no longer available. This study has also been excluded from the present review. The references by Wood (42), Frey-Hewitt (9), and Fortman (8) apparently all present results of the same study, and thus only the Wood study is used here. Finally, an additional study by Wood (41) was identified that met inclusion criteria. The exercise programs used in these studies stressed aerobic exercises such as brisk walking, but a few also included some resistance training. In general, subjects in these studies were men who were only slightly overweight.

While the Expert Panel considered all these articles equally, it should be noted that the sample sizes in the studies by Verity (37) (N = 5 per condition), Rönnemaa (27) (N = 12–13 per condition), and Hammer (12) (N = 8 in exercise and 4 in control) seriously limit conclusions from these articles. The large weight losses in the Hammer study (12), even in the control group, suggest that these subjects were changing their diet. The Katzel study (16) specifically asked patients not to lose weight and thus interpretation of the small weight loss that occurred is difficult.

Despite these concerns, the studies reviewed in Table 1 support the conclusion that exercise produces larger weight loss than the no treatment control. In 6 of the 10 studies reviewed here, the differences were significant. However, in all cases except the Wood study (42), the effect of exercise was very modest.

Thus, the conclusion from these studies is that exercise alone produces modest weight losses. (Evidence Category A). A key question raised by these data is whether it is really the exercise that produces the weight loss, or whether participants in such studies also change their intake, despite instructions to the contrary. Although not included in the Expert Panel's review, a study by Bouchard et al. (6) on the long term impact of significant amounts of exercise provides the best data on the effect of exercise independent of changes in diet. In this 100-d long study conducted at a residential facility, the five male participants exercised twice a day, 6 d·wk^{-1}, at 55% of $\dot{V}O_{2max}$ and their intake was held constant. Subjects in this study lost 8 kg over the 100 d. This study is the clearest proof that exercise alone can produce weight loss.

DOES EXERCISE IN COMBINATION WITH DIET PRODUCE GREATER WEIGHT LOSS THAN DIET ALONE?

The Expert Panel used 15 randomized trials to compare diet alone versus diet plus exercise. They concluded that 12 of the 15 articles showed a greater weight loss in the combined diet and exercise group (1.9 kg) and greater reduction in BMI (0.3 to 0.5) than in the diet only group. Ten of the 15 were reconsidered here; one of the studies used in the Expert Panel reported follow-up data (35) rather than being a separate study and was thus excluded; one could not be located (23); and three had only 3-month duration for the intervention (11,34,37). Three other studies not originally reviewed by the Expert Panel were identified (7,28,29). Thus, in the present review, the answer to this question is based on 13 studies.

The characteristics of the studies in Table 2 differed from those in Table 1 in that now the samples were predominately female and the intervention phase in the majority of studies was shorter, lasting 4–6 months. Moreover, although not shown in the table, subjects in these studies were more overweight than in Table 1.

Only two of the 13 studies showed a statistically significant difference in the weight loss obtained in the diet plus exercise condition compared with the diet alone condition (39,43), with the Wood et al. (43) study finding the significant difference in men only. While the direction of results very consistently favors diet plus exercise, as noted by the Expert Panel, the magnitude of the difference is small and rarely reaches statistical significance. This may result in part

TABLE 2. Weight loss in diet only vs. diet plus exercise.

		Diet Only		Diet + Aerobic Exercise			Diet + Resistance Exercise		
	Duration	N	Weight Loss	N	Weight Loss	Signif.[a]	N	Weight Loss	Signif.[a]
Andersen 1995 (1)	1 yr	16 F	−12.9 kg	16 F	−13.4 kg	NS	18 F	−17.9 kg	NS
Anderssen[b] 1995 (2)	1 yr	52 M/F	−4.0 kg	65 M/F	−5.6 kg	NS			
Bertram 1990 (4)	4 months	8 F	−9.3 kg	13 F	−7.0 kg	NS			
		15 F	−8.1 kg						
Blonk 1994 (5)	6 months	26 M/F	−1.2 kg	27 M/F	−2.9 kg	*			
Dengel 1994 (7)	10 months	28 M	−9.3 kg	23 M	−8.1 kg	NS			
Hammer 1989 (12)	4 months	8 F	−11.5 kg	6 F	−12.9 kg	NS			
Hellenius 1993 (13)	6 months	40 M	−0.3 BMI	39 M	−0.6 BMI	NS			
Marks 1995 (20)	5 months	10 F	−3.7 kg	8 F	−4.5 kg	NS	11 F	−3.5 kg	NS
Ross 1995 (28)	4 months	12 F	−10.0 kg	12 F	−11.7 kg	NS			
Ross 1996 (29)	4 months	11 M	−11.9 kg	11 M	−11.6 kg	NS	11 M	−13.2 kg	NS
Stefanik 1998 (33)	1 yr	46 F	−2.7 kg	43 F	−3.1 kg	NS			
		49 M	−2.8 kg	48 M	−4.2 kg	NS			
Sweeney[c] 1993 (36)	6 months	10 F	−13 kg	10 F	−13 kg	NS	10 F	−12 kg	NS
Wood 1991 (43)	1 yr	40 M	−5.1 kg	39 M	−8.7 kg	*			
		31 F	−4.1 kg	42 F	−5.1 kg	NS			

[a] Significance of difference between diet plus exercise vs diet only.
[b] This study compared diet only to diet plus exercise plus behavior modification.
[c] Data interpreted from graph.

from the small number of subjects and short treatment period used in many of these studies (however, the Stefanik study (33) which had the largest sample and lasted a full year failed to show a statistically significant difference between conditions).

Interestingly, four studies also included a diet plus resistance training condition (1,20,36). Again, these studies showed no significant differences in weight loss between the diet only group and the diet plus resistance exercise condition.

A larger number of studies were used in the meta-analyses, but the conclusions appear similar to those reported here. Miller (21) reported that average weight losses in diet alone and diet plus exercise were similar (10.7 kg and 11.0 kg, respectively). Ballor and Poehlman (3) reported no difference in weight loss (mean = 10 kg).

Thus, the conclusion to this question is that in most studies exercise does not significantly increase initial weight loss over and above that obtained with diet only. However, in almost all studies the diet plus exercise group lost somewhat more weight than the diet alone condition (Evidence Category A).

These studies raise several key questions for discussion. Most important is the question of why the effect of exercise on weight loss is so modest. Perhaps this is caused in part by the short duration of many of the studies. If participants are asked to increase their exercise by 1000 kcal·wk^{-1}, and the study lasts only 4–6 months, then differences in weight loss of only 2–3 kg would be expected. In many of the studies cited, this is close to the magnitude of the difference that is observed. It is also possible that individuals in diet plus exercise programs compensate for the energy expended in exercise by reducing physical activity at other times in the day or by eating somewhat more.

A second question relates to the interaction between the severity of the dietary restriction and the effect of exercise. A review of the studies in Table 2 suggests that programs with more severe dietary restriction (and hence larger weight losses) are less likely to observe an effect of exercise. For example, Andersen (1) used a 925-kcal diet for 16 wk with four servings of liquid formula per day and a frozen entree for dinner. This study produced the largest weight losses in Table 2, but no effect of exercise was seen over and above the effect of the diet.

Issues related to possible gender differences and ethnic differences cannot be resolved based on these studies.

DOES EXERCISE IN COMBINATION WITH DIET PRODUCE BETTER MAINTENANCE OF WEIGHT LOSS?

The Expert Panel discussed three studies of diet plus exercise that included follow-up periods (1,35,39). They noted that at follow-up all three found 1.5–3 kg greater weight losses in the combined diet plus exercise condition.

The meta-analysis by Miller (21) noted that at 1-yr follow-up patients in the diet only group maintained a weight loss of 6.6 kg, whereas those in the diet plus exercise group maintained a weight loss of 8.6 kg. Neither this overall weight loss nor the percent of weight loss retained differed significantly between conditions, although the abstract notes that at 1-yr follow-up the diet plus exercise programs tended to be superior.

In the current review a criterion of 1 yr or more for follow-up was used; since the study by Svendsen (35) had only a 6-month follow-up, it was omitted, but several other appropriate studies were identified (Table 3). Again, using statistical significance as the criterion, it was found that two of the six studies (24,39) had a significantly greater weight loss at follow-up in the diet plus exercise group than in the diet group; in the other studies (31,32,38,40) there were no significant differences at follow-up. However, it should be noted that in every study the direction of the difference favored diet plus exercise.

It is important to point out that in these studies with longer follow-up, participants who were initially randomized to diet only may have begun to exercise whereas those in diet plus exercise frequently fail to continue to exercise.

TABLE 3. Weight loss (kg) at follow-up in studies of diet vs diet plus exercise.

	Short Term						Long Term					
		Diet Only		Diet + Exer.				Diet Only		Diet + Exer.		
	Duration	*N*	Wt. Loss	*N*	Wt. Loss	Signif.[a]	Duration	*N*	Wt. Loss	*N*	Wt. Loss	Signif.[a]
Pavlou[b] 1989 (24)	8 wk	56	−10	54	−12	NS	18 months	56	−3	54	−11	*
Sikand 1988 (31)	16 wk	10	−17.5	11	−21.8	NS	2 yr	8	−0.8	7	−9.2	NS
Skendner 1996 (32)	12 wk	29	−6.9	27	−6.0	NS	1 yr	29	−6.8	27	−8.9	NS
							2 yr	15	+0.9	21	−2.2	NS
Wadden 1997 (38)	24 wk	21	−17.7	21	−15.8	NS	48 wk	21	−15.3	21	−13.5	NS
							100 wk	21	−6.9	21	−8.5	NS
Wing 1988 (39)	10 wk	15	−5.6	13	−9.3	*	1 yr	15	−3.8	13	−7.9	*
Wing 1998 (40)	24 wk	35	−9.1	31	−10.3	NS	1 yr	33	−5.5	30	−7.4	NS
							2 yr	35	−2.1	32	−2.5	NS

[a] Significance of difference between diet only and diet plus exercise.
[b] Data were approximated from graph of results.

Thus, while failing to find a significant difference with the intent-to-treat analysis, some studies find that those individuals who continue to exercise have the best weight losses. For example, although Wadden et al. (38) found no differences in long-term weight loss for subjects randomized to diet only versus diet and exercise, there was a strong association between self-reported level of exercise at follow-up and long-term weight loss. Subjects who reported regular exercise at follow-up maintained a weight loss of 12.1 kg versus 6.1 kg in the nonexercisers.

There are also many correlational studies that show a strong association between exercise at follow-up and maintenance of weight loss (26). From such data, one cannot determine whether it is the exercise *per se* that promotes weight loss maintenance or whether exercise is just part of a constellation of weight controlling behaviors (or a marker for this constellation). However, the correlational analyses support the possibility that the failure to see a significant effect of exercise in the randomized trials described in Table 3 may be related to poor long-term adherence to exercise in those assigned to the diet plus exercise condition or to adoption of exercise in the diet only condition.

Thus correlational data clearly show that continued exercise is associated with long-term maintenance of weight loss. Randomized trials support this finding. In all of the long-term randomized trials reviewed, weight losses at follow-up were greater in diet plus exercise than in diet only. However, the difference was statistically significant in only two of the six randomized controlled trials. (Evidence Category B)

RESEARCH ISSUES

The goal of weight loss intervention is to produce long-term sustained weight loss. The most important question to be addressed is thus whether there is a type or amount of exercise that will really improve maintenance of weight loss. To answer this question, we must be able to ensure that subjects who are randomly assigned to exercise complete the prescribed exercise. To accomplish this, we must first tackle several preliminary issues.

1. Better Assessment of Physical Activity

It is important that we first find a way to more accurately quantify physical activity. At present, doubly labeled water is the "gold standard," but this approach is too expensive for most intervention studies (with large sample sizes and multiple measurement intervals) and does not provide information about the type or pattern of physical activity. The alternative assessment approaches that are currently available, including mechanical devices and self-report, have problems related to validity and reliability. Thus a major goal for the field should be to develop a better way to measure physical activity. Similarly, if we are going to really understand the role of exercise in promoting long-term weight loss, it is important to be able to more accurately determine how much and what type of food the participants are consuming.

2. How Can We Improve Adherence to Exercise in Overweight Participants?

Secondly, if we are going to evaluate the effects of exercise on long-term weight loss maintenance, we need to be able to get overweight individuals to adhere to exercise long term. Overweight individuals typically have low levels of adherence to exercise, and exercise can improve weight loss only if the exercise is actually performed. Thus, we need to develop new approaches to promoting exercise adherence in obese individuals. For example, there have been several studies comparing home-based and supervised exercise. King et al. (17) found that men and women ages 50–65, adhered better to exercise programs they could complete at home rather than in supervised group settings. Likewise, Perri et al. (25) compared the effects of a supervised group exercise program versus a home-based program on exercise adherence and weight loss. At 12 months, the subjects who had been randomly assigned to the home-based programs had better exercise adherence and weight loss. Thus, home-based exercise appears to improve long-term exercise adherence, but makes documentation of the prescribed exercise more difficult.

Since lack of time is the primary barrier cited for exercise, Jakicic (14) has examined the effect of prescribing exercise in multiple short bouts (four 10-min bouts) rather than

longer bouts (one 40-min bout). Adherence to exercise and initial weight loss were both improved by the multiple bout regimens. Improvements in fitness were comparable in the two conditions. Currently, Jakicic is replicating and extending this study and investigating whether providing home exercise equipment promotes adherence and weight loss.

These are provided as examples of the type of research that is needed on adherence. Other variables deserving attention include the effect of social support on exercise adherence, reinforcement for exercise, and ways to prompt exercise by phone or mail. Research is needed to determine whether these adherence strategies are effective for individuals of different races, genders, or age groups.

3. How Should Exercise be Prescribed to Improve Long-Term Weight Loss Maintenance?

If we could measure physical activity and produce better adherence to our exercise prescriptions, we could then begin to determine how exercise should be prescribed to promote long-term weight loss. Typically in behavioral weight loss programs, individuals are taught to gradually increase their physical activity to a level of 1000 kcal·wk^{-1} with the emphasis on aerobic activities (primarily brisk walking). This goal is similar to the recommendations made by the CDC to "accumulate" 30 min of exercise on most days in the week. However, there has been little empirical investigation of this exercise prescription. There are several questions that need to be answered regarding this topic:

How much total exercise is needed?. Recent data suggest that 1000 kcal·wk^{-1} may not be sufficient to produce maintenance of weight loss, and that levels of approximately 2500 kcal·wk^{-1} are associated with weight loss maintenance. Schoeller et al. (30) concluded that the threshold of physical activity for weight maintenance was 47 kJ·kg^{-1} body weight·d^{-1}. This translated to an average of 80 min·d^{-1} of moderate activity or 35 min·d^{-1} of vigorous activity. Similarly in the National Weight Control Registry successful weight losers report expending approximately 2800 kcal·wk^{-1} in physical activity (19).

Does the intensity of exercise matter or is it the total calorie expenditure? The premise of behavioral weight loss programs has been that it is the total calorie expenditure that affects weight loss, not the intensity. However, there do not appear to be any long-term randomized trials that have specifically tested this hypothesis. In the National Weight Control Registry, individuals who have lost weight and maintained their weight loss report expending over 800 calories·wk^{-1} in high intensity exercises such as running or aerobic dance. Similarly, Jeffery et al. (15) found that the best maintainers of weight loss were those in the top quartile of physical activity level at study end (month 18). These individuals had a median level of activity of 2550 kcal·wk^{-1}, with again approximately 800 calories·wk^{-1} in high intensity activity. Such high intensity exercise may help individuals attain the large overall energy expenditure (without such exercise requiring inordinate amounts of time) or alternatively such high intensity exercise may differentially affect appetite, mood, or other factors associated with weight loss maintenance.

Does the type of exercise matter?. To date, few studies have examined the effect of resistance training versus aerobic exercise on weight loss and maintenance. Because resistance training increases lean body mass, this form of exercise may be particularly beneficial for weight loss. Although initial studies have not shown such benefits, further research on this question is needed.

4. Why Does Exercise Improve Maintenance?

Finally, if we better understood the way in which physical activity might improve maintenance of weight loss, we would be better able to prescribe exercise for this purpose. Specifically, it is unclear whether in those studies where exercise has positive effects on weight loss maintenance, it is because exercise burns calories or because of other physiological, psychological, or behavioral effects of exercise. A better understanding of the mechanism would help us to determine the type of exercise to prescribe.

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Physical activity in the treatment of childhood overweight and obesity: current evidence and research issues

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ABSTRACT

EPSTEIN, L. H. and G. S. GOLDFIELD. Physical activity in the treatment of childhood overweight and obesity: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S553–S559, 1999. **Purpose:** This paper reviews the utility of exercise as a treatment for overweight and obese children and adolescents. **Methods:** Computer database searches identified 13 studies that met the following criteria for inclusion: 1) obese children or adolescents were provided either different types of exercise programs or an exercise program compared with a no-exercise control, 2) subjects were randomly assigned to groups or assigned by matching on demographic and anthropometric variables, and 3) the exercise program was at least 2 months in duration. **Results:** The only area in which there were a sufficient number of studies to make a quantitative analysis was the comparison of diet versus diet plus exercise programs, which suggested that exercise adds to the effect of diet in the short-term treatment of pediatric obesity. There was not enough research to evaluate the effects of exercise alone. The majority of findings indicate fitness changes are greater for subjects provided exercise alone or exercise combined with diet in comparison with subjects provided no exercise (control) or diet alone. **Conclusions:** Research on effects of exercise or physical activity in pediatric obesity treatments are encouraging and may be important for improving treatment outcome for obesity and comorbid conditions. Recommendations for future research are presented. **Key Words:** ACTIVITY, EXERCISE, OBESITY, CHILDREN, ADOLESCENTS, WEIGHT LOSS

Exercise or increasing physical activity is one of the cornerstones of pediatric obesity treatment, along with dietary and behavior change (11). Exercise can increase energy expenditure and create a negative energy balance, facilitating weight loss. In addition, exercise increases fitness, and exercise may have independent effects on many of the diseases often associated with obesity. This paper provides an evidence-based review (37) of controlled clinical research examining the utility of exercise and physical activity in the treatment of childhood and adolescent obesity with body composition, fitness, and coronary risk factors as outcomes. Recommendations for future research are presented.

METHODS

Articles in which overweight or obese children and adolescents were placed on exercise programs for the purpose of weight loss were identified using computerized literature searches in several data bases, including *Medline* and *PsychInfo*, from January 1966 through November 1998. Thirteen studies met the following criteria for inclusion: 1) obese children or adolescents were provided either different types of exercise programs or an exercise program that was compared with a no-exercise control condition, 2) subjects were either randomly assigned to groups or matched on demographic and anthropometric variables, and 3) the exercise program was at least 2 months in duration. Table 1 presents subjects' age, sample size, group assignment, and dietary and exercise components. Results for the most common body composition changes during treatment and follow-up are shown in Table 2, with changes in measures of fitness shown in Table 3. Treatment was operationalized as intervention continuing as long as subjects were seen at least once per month. Where available, baseline values, end of treatment changes, and end of follow-up changes have been provided. The preferred test of between group differences is the significant differences in rate of change between groups over time, but if this information was not available, within group differences from baseline to treatment and follow-up are presented.

Methodological Characteristics of the Studies

Experimental design. Random assignment or stratified random assignment was used in 12 of the 13 studies. The remaining study matched subjects to groups based on age, weight, height, and percent overweight (40). The studies were divided into three groups. The first group compared exercise versus no exercise controls, without a diet. The second group compared diet plus exercise versus diet alone

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TABLE 1. Characteristics of controlled studies examining the effects of exercise in pediatric obesity treatment.

Study	Age	N	Groups	Exercise	Diet
Becque et al. 1988 (3)	12–13	11	1. Diet + Ex	3 ×/wk: 50-min supervised aerobic	ADA exchange diet
		11	2. Diet		
		14	3. Control		
Blomquist et al. 1965 (4)	8–9	22	1. Ex	2 extra gym classes/wk, 45-min. intense	None
		21	2. Control		
Epstein et al. 1995 (16)	8–12	55	1. Diet + Ex	Reinforced for ↑ activity	900–1200 kcal
			2. Diet + Sed	Reinforced for ↓ Sed	
			3. Diet + Com	Combined ↑ Ex + ↓ Sed	
Epstein et al. 1994 (17); Epstein et al. 1985 (20)	8–12	13	1. Diet + Pro	Programmed aerobic exercise	900–1200 kcal
		12	2. Diet + LS	Lifestyle activity	
		10	3. Diet + Cal	Calisthenics	
Epstein et al. 1985 (21)	8–12	23	1. Diet + Ex	3-mile walk, stationary ex, 10 min/3×/wk	900–1200 kcal
			2. Diet		
Epstein et al. 1994 (17); Epstein et al. 1984 (19)	8–12	18	1. Diet + Ex	Lifestyle activity	900–1200 kcal
		18	2. Diet		
		17	3. Control		
Epstein et al. 1982 (18)	8–12	8	1. Diet + Pro	Programmed aerobic exercise	900–1200 kcal
		10	2. Diet + LS	Lifestyle activity	
		11	3. Pro		
		8	4. LS		
Ewart et al. 1998 (22)	9th grade	44	1. Aerobic Ex	50 min aerobic classes	None
		44	2. Physical Ed	50 min physical education classes	
Gutin et al. 1997 (30)	7–11	17	1. Aerobic Ex	40 min 5 times a week	None
		18	2. Control		
Hills and Parker 1988 (32)	Prepub	10	1. Diet + Ex	1/wk gymnastics, 20 min, 3–4/wk home ex	Nutrition class
		10	2. Diet		
Owens et al. 1999 (38)	7–11	35	1. Aerobic Ex	40 min 5 times a week	None
		39	2. Control		
Reybrouck et al. 1990 (40)	4–16	14	1. Diet + Ex	15–40 min daily aerobic exercise	800–1000 kcal
		11	2. Diet		
Rocchini et al. 1988 (41)	10–17	23	1. Diet + Ex	40 min 3/wk aerobic exercise	ADA exchange diet
		22	2. Diet		
		18	3. Control		

Ex, exercise; x/wk, number of times activity was performed per week; Sed, reinforced for decreasing sedentary behavior; Com, reinforced for decreasing sedentary behavior and increasing active behaviors; Pro, programmed aerobic exercise; LS, lifestyle exercise; Cal, calisthenics; Prepub, prepubertal; ADA, American Dietetic Association.

controls. The third group compared different types of exercise programs.

Sample characteristics. There is considerable variability between studies in the range of subject ages, including subjects 7–13 yr of age (3,16,18–21,30,38), "prepubertal" participants (32), adolescents (22), and children and adolescents combined in the same samples (40,41). One study used males only (4), another female subjects (22), with the remaining studies using male and female subjects.

Definitions of obesity included: 20% above ideal weight for height (4,16,18–21,40), two standard deviations above weight for height standards (4), a body mass index (BMI) greater than the 85th percentile for sex and age (22), body weight that exceeded the 95th percentile for age (32), tricep skinfold greater than the 85th percentile for gender, age, and ethnicity (38), and the combination of elevated weight for height and skinfold measures (3,41).

Characteristics of exercise prescription. Nine studies used aerobic exercise, with frequency varying from three to four times per week (3,20,41), five times per week (22,30,38), or daily exercise (40). The school-based studies provided two (4) and five (22) extra physical education classes per week emphasizing intensive exercise. Two studies contrasted aerobic versus lifestyle programs (18,20), one aerobic versus physical education classes (22), and one study targeting reductions in sedentary behavior (16). Seven studies incorporated caloric restrictions of various types (3,18–21,40,41). The content of the nutrition education classes used by Hills and Parker (32) was not described.

Evaluation of studies. Because of the small number of randomized, controlled studies evaluating exercise interventions for obesity in pediatric samples, the effects of exercise were generally established by qualitative comparison of results. Quantitative meta-analytic methods were used to evaluate the six studies that compared the short-term effects of diet plus exercise versus diet alone. Effect sizes were established using the formula $\text{Mean}_{\text{diet + exercise}} - \text{Mean}_{\text{diet}} / \text{Standard Deviation}_{\text{diet}}$.

RESULTS

The results will be discussed according to the type of experimental design.

Exercise Versus No Exercise Controls

In two studies Gutin et al. (30,38) found significant reductions in body fat and increases in fitness compared with controls, whereas no significant effects of exercise on body composition or fitness were found by Blomquist et al. (4).

Diet Versus Diet and Exercise Versus No Intervention

Three studies demonstrated significantly greater changes in body composition for the diet plus exercise group com-

TABLE 2. Results of controlled studies examining the effects of exercise in pediatric obesity treatment.

	Baseline				Treatment					Follow-up				
Study Group	%OW*	BMI*	Kg*	%BF*	Months	%OW†	BMI†	Kg†	%BF†	Months	%OW†	BMI†	Kg†	%BF†
Becque et al. 1988 (3)														
1			67.9	38.3	5			−1.6	−3.0					
2			77.2	44.0				−0.4	−3.5					
3			68.7	39.8				+3.2	+0.7					
Blomquist et al. 1965 (4)														
1			41.4		4			+0.8						
2			42.7					+0.8						
Epstein et al. 1995 (16)														
1					4	-13.2_a			−3.5	12	-8.7_a			-1.2_a
2						-19.9_b			−5.0		-18.7_b			-4.8_b
3						−17.0			−4.5		-10.3_a			-2.2_a
Epstein et al. 1994 (17); Epstein et al. 1995 (20)														
1	47.8		56.3		6	−17.4‡		+1.1		120	-10.9_a			
2	48.3		56.4			−19.6‡		−1.1			-19.7_a			
3	48.0		56.3			−20.7‡		−1.1			$+12.2_b$			
Epstein et al. 1985 (21)														
1	48.0		53.8		6	-27.5_a		-6.8_a		12	−25.4‡		−3.9‡	
2	48.1		54.0			-18.8_b		-3.8_b			−18.7‡		−1.4	
Epstein et al. 1994 (17); Epstein et al. 1984 (19)														
1	44.0				6	-16.0_a				120	−8.4			
2	44.0					-21.0_a					−10.0			
3	44.0					$+3.0_b$					NR			
Epstein et al. 1982 (18)														
1	37.2	24.1	50.1		6	−10.3‡	−1.3‡			17	$+0.1_a$	$+1.2_a$		
2	40.5	24.9	53.0			−19.0‡	−3.0‡				-13.8_b	-1.5_b		
3	38.7	25.3	57.5			−13.9‡	−2.1‡				-9.7_a	-0.7_a		
4	34.0	24.0	52.3			−13.2‡	−2.0‡				-11.2_b	-1.0_b		
Ewart et al. 1998 (22)														
1		24.8			4.5		+0.3							
2		24.1					0.0							
Gutin et al. 1997 (30)														
1		31.4	63.2	47.2	4				-4.1_a					
2		28.8	59.7	43.8					-0.6_b					
Hills and Parker 1988 (32)														
1			51.6		4			−5.5						
2			50.2					+2.6						
Owens et al. 1999 (38)														
1			57.5	44.5	4			+1.1‡	-2.2_a					
2			56.9	44.1				+2.0‡	0.0_b					
Reybrouck et al. 1990 (40)														
1	57.3		58.1		4	-25.5_a				8	−29.6			
2	50.0		50.4			-15.8_b					−19.3			
Rocchini et al. 1988 (41)														
1			72.0	41.0	5			-2.4_a	-6.0_a					
2			73.0	43.0				-2.5_a	-4.0_a					
3			73.0	41.0				$+4.0_b$	$+1.0_b$					

* Baseline.
† Change from baseline.
‡ Value significantly different from baseline value (not reported when between-group differences are reported).
%OW, Percent overweight; BMI, Body Mass Index; Kg, weight in kilograms; %BF, Percent body fat; NR, value not reported.
Values with different subscript letters differ significantly from each other.

pared with the diet only group at post-treatment (21,32,40), while the other three studies did not find significant differences between groups (3,19,41). The mean effect size for the six studies, based on one study using weight as the dependent variable (32), three using percent overweight (19,21,40), and two using percent body fat (3,41), was 0.45. Two studies included follow-up assessments, with one showing significant additive effects of exercise at 12 months (21), a second showing no significant differences between groups at 10 yr (17,19). Two studies showed diet and exercise to be more effective than diet alone in increasing fitness (Table 3) as measured by physical work capacity (21) and submaximal oxygen consumption (41), with one study finding no differences between groups (3). In addition to fitness effects, two randomized, controlled studies examined the effects of exercise on coronary risk factors. In these studies diet plus exercise provided greater beneficial effects on high density lipoprotein (HDL) (3) and systolic blood pressure (41) compared with diet alone and control.

Types of Exercise Programs

Epstein et al. (18,20) conducted two studies that compared the effects of lifestyle activity versus traditional programmed aerobic exercise. In the first comparison (18) lifestyle activity showed larger reductions in percent overweight at 6 and 17 months than aerobic exercise. No differences in fitness were observed at 6 months, as the aerobic group showed larger initial changes, but the lifestyle program showed better maintenance. Epstein et al. (20)

TABLE 3. Results of controlled studies examining the effects of exercise on fitness in pediatric obesity treatment.

Study Groups	Fitness Measure	Units	Pre-Rx	Treatment		Follow-up	
				Months	%RxΔ	Months	%FUΔ
Becque et al. 1988 (3)							
1	$\dot{V}O_{2max}$	mL·kg^{-1}	32.7	5	+2.1		
2			28.5		+0.7		
3			30.6		−2.9		
Blomquist et al. 1965 (4)							
1	PWC_{170}	kpm·min^{-1}	489	4	+8.8		
2			512		+7.2		
Epstein et al. 1985 (20)							
1	PWC_{150}	kpm·min^{-1}	394.3	6	NR	12	+31.1‡
2			407.3		+26.4‡		+8.2
3			NR		NR		NR
Epstein et al. 1985 (21)							
1	PWC_{150}	kpm·kg^{-1}	6.6	6	+19.7‡	12	$+37.9_a$
2			6.5		+6.2		$+16.9_b$
Epstein et al. 1982 (18)							
1&3	Step test	HR	122.8	2	-16.8_a	6	−10.0‡
2&4			118.7		-8.4_b		−7.8‡
Ewart et al. 1988 (22)							
1	Step test	s	515.5	4.5	$+11.1_a$		
2			486.8		-9.2_b		
Gutin et al. 1997 (30)							
1	Submax	HR	119	4	-2.8_a		
2			123		$+4.3_b$		
Owens et al. 1999 (38)							
1	Submax	HR	120	4	-3.2_a		
2			124		$+0.2_b$		
Rocchini et al. 1988 (41)							
1	$\dot{V}O_{2submax}$	mL·min^{-1}	1349	5	-17.8_a		
2			1226		-2.1_b		
3			1225		$+4.2_b$		

‡, value significantly different from baseline value (not reported when between group differences are reported). Pre-Rx, Pretreatment; %Rx Δ, percent change from baseline to post-treatment; %FU Δ, percent change from baseline to follow-up; $\dot{V}O_{2max}$, oxygen consumption measured during exercise at maximum capacity; PWC_{170}, Physical work capacity with heart rate at 170 beats·min^{-1}; PWC_{150}, Physical work capacity with heart rate at 150 beats·min^{-1}; Submax, exercising at a submaximum work load of 49 W; $\dot{V}O_{2submax}$, oxygen consumption measured during exercise at a submaximum work load of 15 W initially followed by increases of 30-W intervals; Step test, submaximum exercise involving stepping up and down on a bench at a specified rate and duration; kpm·kg, kiloponds per kilogram of body weight; mL·min^{-1}, millilitres per minute of exercise; NR, Not reported.
Values with different subscript letters differ significantly from each other.

compared lifestyle and aerobic exercise programs with a low intensity calisthenic group to control for the nonspecific effects of exercise. At 6- and 12-month assessments all groups showed similar reductions in percent overweight and weight. The lifestyle group maintained the decreases in percent overweight at 2 yr, while the aerobic and calisthenic exercise groups showed increases in percent overweight. At 5 yr the lifestyle exercise group had lower percent overweight than the calisthenic group, while at 10 yr both lifestyle and aerobic groups had lower percent overweight and weight than the calisthenic group (17). Programmed exercise demonstrated larger improvements in fitness than lifestyle activity at 12 months.

Based on the observation that sedentary behaviors are related to the development of childhood obesity (9,28), Epstein et al. (16) compared groups that were differentially reinforced for increased physical activity and/or reduction in sedentary behaviors. Reducing sedentary behaviors proved more successful than increasing physical activity at both the end of treatment and 1-yr follow-up. All groups showed equivalent increases in fitness.

Finally, Ewart et al. (22) compared the effects of programmed aerobic exercise or physical education classes on BMI of adolescent girls at high risk for hypertension. No differences between exercise groups were found at the end of the 18-wk intervention. The aerobic exercise intervention showed significant improvements in fitness and systolic blood pressure compared with the physical education group, while both groups showed significant reductions in diastolic blood pressure.

DISCUSSION

This review highlights the limited number of randomized controlled studies investigating the efficacy of exercise in the treatment of pediatric obesity. The small number of controlled outcome studies in combination with the use of different dependent variables across studies limits the strength of conclusions that can be drawn. The only area in which there are sufficient studies to make quantitative data-based conclusions for body composition changes is in the comparison of diet versus diet plus exercise. These results indicate diet plus exercise improve short-term obesity treatment by almost one-half standard deviation above and beyond the contribution made by diet alone (Evidence Category B). Exercise reliably increases fitness effects whether compared with diet alone or no exercise controls (Evidence Category B). There are too few studies comparing exercise versus no exercise, or comparing different exercise programs to make firm conclusions. In addition, there are very

limited data on long-term effects of exercise, and any conclusions would be premature.

Future Research Directions

Exercise adherence. A very important aspect of exercise intervention research is adherence. Failure to adhere to the exercise program limits short- and long-term effectiveness of the intervention and makes accurate evaluation impossible. Adherence may be affected by characteristics of the exercise program, such as intensity and duration, but also may relate to the structure of the exercise program. It is tempting to use on-site exercise interventions to facilitate measurement of adherence and better control intensity and duration of the exercise. However, research with adults has shown superior long-term effects for home-based, rather than exercise site-based, programs (33,39). If this effect generalizes to pediatric programs, it becomes extremely important to balance experimental control over the characteristics of the exercise program versus superior long-term effectiveness of programs that maximize choice.

Improving adherence to physical activity in obese pediatric samples may be very challenging, as obesity predicts poor adherence to exercise programs (10) and environmental contingencies need to be stronger for obese than nonobese children to choose active behaviors over sedentary ones (13,14). One way to improve adherence to physical activity programs may be to build activity into the child or adolescent's lifestyle, which has shown to maximize short- and long-term adherence in obese children (18,20). Compared with aerobic exercise, lifestyle activity programs maximize choice and perceived control over exercise behavior, both of which have been shown to be important determinants of exercise adherence (48).

Another way to increase adherence would be to increase the reinforcing value of being active or reduce access to sedentary behaviors that are powerful reinforcers for obese children and compete with being active. Identifying methods of enhancing the reinforcing value of exercise would be of great importance as it would provide putative benefits to adherence, weight control, and fitness, thus enhancing the overall effect of exercise as a means of treating and preventing obesity. Children with sedentary lifestyles commonly choose sedentary behaviors when given the choice between being sedentary or active, and research suggests limiting access to sedentary behaviors can result in increased physical activity (12–14). In addition, sedentary behaviors can be used to reinforce obese children for being more physically active (43).

Diet. One effect of combining diet and exercise is to increase negative energy balance and theoretically improve weight loss over diet alone. In addition, diet may influence adherence or performance to physical activity and thus weight loss. For example, basic animal research suggests that caloric reductions lead to spontaneous increases in activity, although these increases may only be limited to nonobese animals (46). There are suggestive data that controlling for calories, a greater carbohydrate versus fat intake will lead to significant differences in energy expenditure (2). In addition, intake of foods with a low glycemic index may improve performance and lower perception of workloads, making exercise more pleasant (8). It would be potentially very important to determine whether variations in total calories or the macro- or micro-nutrient composition of the diet influenced activity levels in children, and then diets could be used to enhance physical activity in obese children.

Individual differences. There may be individual differences that influence the effects of exercise on pediatric obesity. Research suggests that age is related to activity level, the types of activities engaged in, and equally important, types of activities enjoyed (44). Interventions may need to be different across developmental stages. An example of one type of exercise that may depend on age is resistance training. Recent data suggests that resistance training can prevent the gain of additional adiposity in adolescents (50), but safety can be an issue when using resistance training in prepubescent children (1).

A variety of other individual difference variables may influence physical activity in the treatment of obesity, including gender, degree of obesity, and comorbidities. Boys and girls are socialized differently regarding exercise, have different exercise skills, may prefer different exercise activities (44), and girls show greater increases in adiposity during puberty than boys (36). Programs may need to be tailored to gender differences. In addition, obesity and low initial levels of fitness may attenuate the positive consequences associated with being active, reducing the motivation to maintain an exercise program. Likewise, a variety of comorbid conditions may influence exercise programs. For example, there is an increased prevalence of obesity in children and adolescents with asthma (26,34), and the asthma may interfere with participation in physical activity, which would make it more difficult for obese children with asthma to lose and maintain weight loss.

Ethnic and minority subjects. Few studies have included children or adolescents from ethnic or minority backgrounds in exercise intervention studies, despite data indicating these persons tend to be less physically active (24,52) and are more likely to be obese than nonminority children (27,42). There may be important characteristics of ethnic or minority groups that can influence exercise patterns. For example, the environments in which minority children live may be more dangerous than environments in which nonminority families live, reducing access of minority children to outdoor activities. In addition, exercise may be less valued and socially accepted in certain minority environments compared with people with nonminority status. Research with children from ethnic and minority backgrounds is needed to determine whether the effects of exercise on pediatric obesity generalize to these groups, and the potential barriers to implementing exercise programs need to be identified.

Physical and psychological comorbidity. Exercise may be particularly useful for treating the comorbid conditions often associated with obesity. Obesity is often associated with cardiovascular risk factors, and exercise improves

the treatment effect for obese children with elevations in lipids (3) and blood pressure (41). These improvements in comorbid conditions may be mediated by exercise influenced weight loss, improvements in fitness produced by exercise, or a combination of weight loss and fitness change.

Obese children and adolescents seeking treatment often experience psycho-social comorbidity (6). Exercise can reduce psychopathology in adults (7,47), as well as enhance mood in normal weight adults (23) and normal weight children and adolescents (5), but there is no controlled research examining these factors in obese children and adolescents. Improvements in weight as well as fitness changes may be related to improvement in psychological comorbidity in obese children and adolescents.

Characteristics of exercise programs. The majority of exercise programs have focused on aerobic exercise. However, there has been very little research on the best way to implement aerobic exercise programs to maximize weight loss and adherence. The best schedule for increasing intensity or duration of aerobic activity needs to be identified. Research in young, normal-weight men indicates that isocaloric low intensity, long duration exercise results in greater total fat oxidation than moderate intensity exercise (49). These results underscore the importance of investigating the effects of intensity of exercise on weight loss in obese children. Several other important issues need to be empirically studied. For example, can resistance training be used safely in a pediatric population to maximize the development of lean body mass and increase total energy expenditure? Is it better to focus on one activity or is cross training better? Can the combination of aerobic and resistance training enhance the effects of aerobic exercise?

The structure of the program may be important for developing active lifestyles in treating obesity. Data from several trials incorporating moderate to intense aerobic exercise with children in the United States (29,30) and Japan (31,45) suggest that school-based exercise interventions may be a promising approach to treating childhood and adolescent obesity. It is important to investigate further the efficacy of school-based exercise programs since they provide an opportunity to develop healthy, active lifestyles in large numbers of children and adolescents (51). In addition, use of the family to structure and support activity programs may be useful for long-term change, as parent activity level is a strong predictor of child activity (25,35), and inclusion of parents in the treatment may improve long-term outcomes of obesity treatment (15).

SUMMARY

There are limited controlled data on physical activity in pediatric obesity treatment. The initial research is encouraging, but more research is needed to maximize the effects of physical activity on obese children. Investigators need to take into account subject characteristics, characteristics of the exercise programs, and the outcomes to be achieved to develop the most effective exercise programs. The development of an active lifestyle in obese children has the potential for multiple benefits on obesity, comorbid physical and psychological problems, and acquisition of an active lifestyle that may accrue lifelong health benefits.

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Physical activity and prevention and treatment of weight gain associated with pregnancy: current evidence and research issues

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ABSTRACT

RÖSSNER, S. Physical activity and prevention and treatment of weight gain associated with pregnancy: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S560–S563, 1999. **Purpose:** The purpose of this study was to examine the evidence in the literature for a relationship between physical activity and weight development during and after pregnancy. **Methods:** A retrospective analysis of the literature, mainly based on an extended MEDLINE search and the Pregnancy and Childbirth Database (Cochrane), was conducted. **Results:** Weight development during pregnancy is the result of numerous interacting factors, with physical activity being one important determinant of weight outcome and eventually also overweight and obesity. Several methodological matters complicate the interpretation of the interrelationships: generally body weight and not fat has been measured, sociobehavioral confounders have rarely been accounted for, and the time frame to determine the effect of pregnancy on later weight development has been highly variable. Most studies concentrate on the role of physical activity, such as recreational activity and sports, on the safety of the pregnant mother and the fetus. The few studies that address the question of exercise and fat deposition found slightly a smaller increase in skinfold measures in pregnant women who exercised. Factors such as the self-selection of well-educated women under study and of normal body weight, as well as the lack of controls, limit the possibilities to which these results can be extrapolated. **Conclusion:** Little information is available on these issues and the quality of information is at most at the evidence type D level, according to the NHLBI classification system. Future research priorities include proper prospective control trials of this important aspect of an obesity preventing life style tool, as well as studies concerning the preventive effects of physical activity on weight retention after pregnancy, an issue not as yet addressed in the literature. **Key Words:** BODY WEIGHT, EXERCISE, OBESITY, PHYSICAL ACTIVITY, PREGNANCY

The assessment of the interaction between energy intake, physical activity, and pregnancy-associated body weight increase is complex for a number of reasons. An examination of the literature makes it clear that a sequence of events makes the analysis of the relationship between physical activity and obesity in pregnancy difficult.

1. The study of the weight development itself during and after pregnancy is full of inherent complications in analyzing these relationships.
2. The study of physical activity during pregnancy has generally focused on the potential risk of such activity on the well-being of mother and child rather than on the possible relationship with a later accumulation of adipose tissue during and immediately after the pregnancy.

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WEIGHT DEVELOPMENT DURING AND AFTER PREGNANCY: METHODOLOGICAL ISSUES

Whereas the objective of this review is to focus on obesity related problems, almost all studies in this area have measured body weight rather than body fat. For obvious ethical reasons, some of the more complex but precise techniques to measure adipose tissue are not available in studies of this type.

Numerous sociobehavioral confounders responsible for the apparent effect of parity on body weight often have not been accounted for or are sometimes impossible to account for. Longitudinal studies partly overcome these problems by using each mother as her own control.

Three main methodological constraints have to be overcome: 1) accurate measurements of body weight from conception and onwards must be obtained; 2) sufficient time for weight lost after delivery must be allowed; and 3) the parallel effect of aging during pregnancy and follow-up must be accounted for.

In the extensive review by Harris (8) the general outcome can be summarized as follows: Ninety percent of all studies

reviewed found a body weight greater after pregnancy than before (by 0.2 to 10.6 kg). Only 3 of 71 studies (11,15,20) complied with all three aforementioned methodological criteria.

In this subgroup (11,15,20) the average "cost" of a pregnancy was 0.9 to 3.3 kg. These differences in weight gain became slightly smaller (0.4 to 3.0 kg) after control for several sociobehavioral confounders. Thus in the literature it is generally rare that factors obviously of importance in evaluating the role of physical activity, such as age, height, education, parity, giving up work, smoking habits, prepregnant weight and activity, alcohol consumption, marital status, morbidity, and dieting behavior, are accounted for(8). This evaluation is further complicated by the fact that changes in these confounders may take place in different ways and during various phases of the pregnancy (24).

MAIN FOCUS ON MATERNAL FITNESS AND FETAL WELL-BEING

In the discussion of physical activity, a great deal of attention in the literature is addressed to one particular form of physical activity, namely, the question of exercise. In many cases this trend seems to have developed in response to the fitness interest of the 1970s and onwards. The safety of aerobic exercise, the duration of a running program, etc., have been more the focus than the potential benefit of adipose tissue mass control by increasing energy expenditure through physical activity (16). Furthermore, most studies on physical activity in pregnancy have analyzed its role from an obstetrical point of view. Examples include importance of exercise during pregnancy on labor and labor associated variables (such as length of labor (10), Apgar scores of newborns (7,22), and postpartum recovery (18,19,23)).

FACTORS AFFECTING PHYSICAL ACTIVITY DURING PREGNANCY

Numerous psychological factors may indirectly affect eating behavior, food selection, and thus body weight as a consequence of physical activity during pregnancy.

In the study by Sibley et al. (14) women exercised by swimming during the second trimester; they found, surprisingly enough, no improvement in several fitness parameters but an increased sense of well-being, improved appetite, and a more restful sleep pattern.

Wallace et al. (18) also reported higher self-esteem in exercising pregnant women. Indirectly it has been suggested that physical activity during pregnancy may reduce depression and mood swings (6).

In the descriptive, nonexperimental, and uncontrolled study by Horns et al. (9) the only characteristic that differed between active and inactive groups was educational level, which was higher in active women, whereas race, marital status, and cesarean section rates or age did not differ significantly.

REVIEWS OF PHYSICAL ACTIVITY/EXERCISE AND PREGNANCY

Searches in MEDLINE and the Pregnancy and Childbirth Database (Cochrane Collaboration) reviewed by Bell et al. (1) in 1994 illustrate that physical activity to control adipose tissue development receives little attention. This review with 136 references mainly deals with the effects of exercise on the maternal cardiorespiratory system (49 references), the effects of exercise induced trauma on mother and fetus (8 references), the effects of exercise on maternal heat production and fetal consequences (17 references), and the fetus and uterus response to exercise (42 references). This review concludes by identifying the need for randomized trials in which sedentary women and recreational athletics are studied separately.

The review by Sternfeld (16) contains a table of observational studies of exercise and pregnancy outcome. Of the 14 studies reviewed only three mention maternal weight development as an outcome in the findings: the studies of Clapp et al. (2,3) and Dale et al. (5). The other studies mainly report effects on pregnancy complications, risk of prematurity, labor time and complications, and birth weight. No studies ranks higher than evidence level D on the NHLBI classification system.

Clapp and Little (4)

One of the few studies addressing the question of recreational exercise on pregnancy weight gain and subcutaneous fat deposition is that by Clapp and Little (4). The study is prospective, but not randomized. Women planning a child were studied; 44 chose to remain physically active during pregnancy, and 35 stopped their exercise habits when they learned of their pregnancy. No differences in any parameters studied were observed during the first and second trimester, but in the third trimester exercising women had a reduced rate of weight gain and significantly less increase in five skinfold measures. This study thus demonstrates that well-educated nonsmoking highly motivated women who maintained a balanced diet and were not obese before pregnancy accumulated slightly less fat in the last trimester. Although this is one of the few studies addressing the issue of physical activity and control of obesity, several limitations remain. Self-selection, nonquantification of the amount exercised, and a very selective group of women, whose weight throughout the pregnancy remained well within the normal range, limit the possibilities for extrapolation of these results to the population at risk, for whom a pregnancy is a major trigger for weight retention and subsequent obesity (12).

Recommendations to the family physician by Wang and Apgar (17)

A 1998 review in American Family Physician (17) is interesting for what it does not tell about exercise during pregnancy as a method to improve weight control. The review concludes that "a firm basis for exercise recommendations is lacking." Weight control is only mentioned in that

"maternal weight should be routinely followed," which probably does not come as a major surprise to either physician or pregnant woman. Nor is it surprising to note that boxing and wrestling are not recommended during pregnancy, whereas walking, stationary cycling, low impact aerobics, and swimming are encouraged. The latter activities obviously agree with recommended program to control inappropriate gain of adipose tissue. Wang and Apgar underscore that there remain major deficits in our knowledge: The outcome of several sports have not been analyzed, and mainly middle-to-upper class women have been studied.

As part of an extensive analysis of NHANES I data, the relationship between parity-associated weight gain with sociodemographic and behavioral factors was analyzed in white and Afro-American U.S. women (21). Data from 2952 women were examined at baseline and 10 yr later, and as part of the analysis, factors increasing the probability of gaining more than 11.4 kg during that time period were assessed. Data show that weight increase is more likely in women with several indicators of lower socioeconomic status, but surprisingly nonrecreational physical activity was associated with substantial weight gain, even after some adjustment. However, when follow-up rather than baseline measurements (simple three-level questions on activity) were used, the association disappeared. Because of sample size limitations, no data on inactivity and weight gain were available in the Afro-American group. These findings underscore the problem in interpreting data on physical activity because half the sample changed their reported activities between baseline and follow-up. As Wolfe et al. (21) point out, few studies on postpartum weight retention and physical activity have been published. In Öhlin and Rössner (24) factors associated with weight retention were studied and related to trends in behavioral patterns which were constructed *a priori*. Major determinants of weight retention 1 yr after delivery were changes in eating habits relating to higher intake, more snacking, and irregularity. The subgroup of women who had retained more than 5 kg 1 yr after delivery were less often physically active in their leisure time, 54% versus 46%, $P < 0.05$. In our extreme group with a weight development corresponding to the NHANES figures, 23% of women retaining more than $\geq$ 10 kg 1 yr after delivery were quite inactive in their leisure time versus 4% of women with less weight retention ($P < 0.001$). More women with BMI < 24 kg$\cdot^{-2}$ returned to a higher level of physical activity after pregnancy than did women with higher prepregnancy body weight (23% vs 15%, $P < 0.005$) (25).

RESEARCH PRIORITIES

This review demonstrates that data, primarily focusing on the role of physical activity to control inappropriate weight gain during pregnancy and after delivery is scarce and unsystematic. Data on prevention—as is outlined in the title of this chapter—is completely lacking. MEDLINE searches and an analysis of review articles underscore that the main interest has been stimulated by the needs of obstetricians to identify behavioral traits during pregnancy that improve the chances of a successful pregnancy outcome. Focus has been more on the importance of maternal weight development for the well-being of the fetus and the newborn than on the fact that inappropriate weight gain during pregnancy, as a recognized high risk period, may in the long run expose the woman to several serious consequences associated with obesity. The fact that a pregnancy may be an important trigger for future accelerated weight increase and obesity is documented in the literature (13) but rarely addressed.

Weight data, although often of moderate quality, is often available in previous studies, and it is possible that further retrospective analyses of already existing material could be useful in elucidating the role of physical activity for prevention and treatment of obesity. This review clearly indicates that to study the potential of physical activity on future weight development, prospective, randomized, controlled studies are essential. On the other hand, the limited amount of data available illustrate the scope of the problem. Complete randomization to activity/nonactivity may be impossible to obtain, blinding may be difficult, and the long time span makes interpretations more difficult. We still do not know, for example, whether the role of physical activity is most important for the overall weight outcome before, during, or after pregnancy. The few data available (24) suggest that effects on weight can only be observed in the third trimester and that postpartum overall weight retention correlated in particular with the physical activity 6–12 months after delivery.

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Physical activity and weight gain and fat distribution changes with menopause: current evidence and research issues

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ABSTRACT

ASTRUP, A. Physical activity and weight gain and fat distribution changes with menopause: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S564–S567, 1999. **Purpose:** At the onset of menopause a woman's body weight reaches its maximum. For any given subsequent body weight there is an increase in relative body fat and abdominal fatness with advancing age. The increased body fatness and abdominal fat distribution are associated with a high incidence of cardiovascular disease and certain cancers, particularly breast cancer. The aim of this analysis was to assess the role of physical activity for weight gain and changes in fat distribution occurring with menopause. **Methods:** A systematic review based on a *Medline* search was conducted. **Results:** According to cross-sectional observational studies postmenopausal women with high levels of physical activity have lower body fat and abdominal fat. Longitudinal studies show that physically active women are less likely to gain body fat and abdominal fat after menopause than sedentary women (Evidence category C). There are very few randomized controlled trials (RCT) comparing exercise with no intervention, and diet with diet plus exercise, and the results do not allow a firm conclusion as to whether physical activity may prevent or limit the gain of total fat and abdominal fat after menopause, or whether it may be effective as part of an obesity treatment program. **Conclusions:** There is a need for RCT to evaluate the effect of increased physical activity and fitness as a tool for prevention of the changes in body composition associated with menopause and aging in normal weight women. The efficiency of different programs of exercise training as a treatment option in postmenopausal women with existing overweight and obesity should be investigated. RCT should have the appropriate design and size and use accurate methods to assess exercise compliance, body composition, and intra-abdominal adipose tissue. **Key Words:** EXERCISE, OBESITY, BODY COMPOSITION, AGING

There are only a few studies on the changes in body fat and fat distribution occurring with menopause in women. This is an important issue because of the relationship between obesity and cardiovascular disease and between obesity and certain cancers, particularly breast cancer, in postmenopausal women. Body weight reaches its maximum in women very close to the time of menopause and for any given body weight there is a subsequent increase in relative body fat and abdominal fatness with advancing age (22).

Some studies have found that the increase in body weight accompanying menopause is more related to age than to menopause *per se* (32,34), whereas others have found more specific menopause-related increases in body weight (16), total body fat (14), central fat accumulation (14,28), and intra-abdominal fat accumulation (12). Nevertheless, even in weight-stable postmenopausal women in their early sixties there is a progressive change in body composition, the most notable net change being loss of lean tissue in the legs and gain in fat tissue in the trunk (2). It is possible that the redistribution of body fat is related to estrogen decrease (3,21), but only one RCT has shown that early postmenopausal women treated with hormonal replacement therapy did not experience the accumulation of abdominal fat observed in the placebo group, while hormones had no effect on fat-free mass (7).

CAUSES OF THE INCREASED CARDIOVASCULAR RISK IN POSTMENOPAUSAL WOMEN

Premenopausal women have a lower overall incidence of atherosclerosis and coronary heart disease (CHD) compared with men of similar age. However, atherosclerosis and CHD rise sharply in women during the postmenopausal years, and the biological and behavioral changes that increase CHD risk have been studied during the natural transition from pre- to postmenopausal status. These risk factor changes include increased serum total cholesterol and low-density lipoprotein cholesterol (LDL-C), decreased high-density lipoprotein cholesterol (HDL-C), weight gain, increased abdominal fat distribution, and decreased physical activity. One hypothesis is that the changes may result from decreased ovarian function and estrogen production after menopause. The decrease in estrogen production is presumed to increase

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CHD by affecting, in concert, several risk factors adversely, such as the lowering of hepatic LDL receptor activity affecting lipid profile unfavorably, and increases in coronary atherosclerosis, endothelial function, and arterial thrombosis (8).

Several observational studies comparing users of estrogen replacement therapy (ERT) with nonusers have consistently shown that ERT use is associated with a lower incidence and mortality of CHD (1,6), and in women with established coronary disease a lower risk of reinfarction, CHD-related death, and coronary artery restenosis (20). Three different meta-analyses have concluded that ERT decreases the risk of CHD by 35–50% (17). The seemingly overwhelming nature of the observational data showing a lower risk of CHD in hormone users raised concerns about the ethics of randomized trials in which some women would receive not ERT but a placebo. The first large RCT on hormone replacement therapy (estrogen plus progestin) versus placebo in secondary prevention of CHD emphasized the shortcomings of observational studies by showing the opposite of the expected outcome, i.e., that hormones did not reduce the overall rate of CHD events but increased the rate of thromboembolic events and gallbladder disease (9). The role of ERT in prevention of CHD in postmenopausal women without established CHD awaits the outcome of ongoing randomized trials. The conflicting conclusions reached from observational studies and the RCT may be result from the "healthy cohort effect." Women with healthy behaviors, such as those who follow a low-fat diet and exercise regularly, may use hormones selectively. These differences in behavior may not be taken into account in the analysis of observational studies because they are not measured, are poorly measured, or are unmeasurable (9,17). Without refuting the role of ERT, it is suggested that lifestyle factors that may contribute to explain the lower CHD and mortality of the postmenopausal hormone users in the observational studies should be considered in more detail. Notably, lean women who exercise at least 4 $h \cdot wk^{-1}$ have a 72% lower risk of postmenopausal breast cancer than sedentary women (29).

OBSERVATIONAL STUDIES ON PHYSICAL ACTIVITY AND BODY FAT GAIN

Older subjects are more inactive than their younger counterparts (22), and this difference has been suggested to contribute to obesity. It is supported by 10-yr follow-up data of females with a mean age of 46 from the NHANES-I study where, at baseline and at follow-up, physical activity was inversely related to body weight (33). Subjects with a low physical activity level at follow-up were 3.8 times more likely to have gained > 13 kg during the preceding 10 yr (33). Another prospective study has found that low levels of physical activity are associated with weight gain and larger increases in waist circumference in postmenopausal women (4).

Cross-sectional studies have demonstrated that postmenopausal women who regularly perform endurance exercise have less body fat and trunk fat than sedentary controls (31). In these studies, both exercise volume and fitness were inversely related to total fat mass (31). Other studies have failed to confirm this finding. In 95 postmenopausal women physical activity was positively correlated to muscle strength, which was also correlated to lean tissue mass, but no correlations were found to body fat or fat distribution (2). However, in larger cross-sectional studies where more confounders were controlled for, intra-abdominal fat measured with computed tomography was inversely related to physical activity estimated by the Baecke questionnaire (10). When the quantification of physical activity is improved by measurement of total free living energy expenditure by doubly labeled water technique, reduced physical activity levels are associated with increased body fat (19).

INTERVENTION STUDIES ON WEIGHT GAIN AND FAT DISTRIBUTION

Uncontrolled studies suggest that endurance training reduces both body fat and abdominal fat in postmenopausal women (11) and that strength training may cause a 10% reduction in intra-abdominal adipose tissue without any notable decrease in body weight or total body fat (30). Studies combining diet and exercise do not allow a separation of the effects of diet and exercise (24). In The Evidence Report on the treatment of overweight and obesity, the following conclusions were reached (15): 1) Physical activity contributes to weight loss, both alone and when it is combined with dietary therapy (Evidence Category A);. 2) Physical activity contributes to a decrease in body fat, including a modest effect on abdominal fat (Evidence Category B); and 3) Physical activity in overweight and obese adults increases cardiovascular fitness independent of

TABLE 1. Randomized controlled trials on effect of physical activity on body weight and fat distribution in overweight and obese postmenopausal women.

Author	N	Mean age	Mean BMI	Duration	Activity	Results
Kriska et al. 1986 (13)	114 experimental 115 control	58 57	25.2 25.1	2 yr	Walking	Not reported
Ready et al. 1995 (18)	15 experimental 10 control	61	29.4 32.1	6 months	Walking	BW: −1.9 vs + 0.6 kg* BF: −1.4 vs + 0.3 kg* WHR: No difference
Seals et al. 1997 (23)	10 experimental 5 control	55	27.1	12 wk	Aerobic exercise	No difference in change in BW or waist circumference
Stefanick et al. 1998 (25)	43 experimental 45 control	57	26.3	1 yr	Aerobic exercise	No difference in change in BW or WHR

BW, body weight; BF, body fat, WHR, waist-to-hip circumference ratio.
* Statistically significant difference between changes in the two groups.

TABLE 2. Randomized controlled trials on effect of diet versus diet plus physical activity on body weight and fat distribution in overweight and obese postmenopausal women.

Author	N	Mean age	Mean BMI	Duration	Activity	Results
Svendsen et al. 1993 (26)	50 diet 48 diet + exercise	54	29.7	12 wk	Combined aerobic/anaerobic	BW: −10.3 vs −9.5 kg BF: −9.6 vs −7.8 kg* WHR: −0.03 vs −0.03 kg
Fox et al. 1996 (5)	25 diet	66	30.1	24 wk	Aerobic exercise	Decrease in BW and BF in both groups with no group difference
	16 diet + exercise		30.6			
Stefanick et al. 1998 (25)	46 diet 43 diet + exercise	57	26.3	1 yr	Aerobic exercise	BW: −3.1 vs −2.7 kg WHR: −0.01 vs 0.01 kg

BW, body weight; BF, body fat, WHR, waist-to-hip circumference ratio.
* Statistically significant difference between changes in the two groups.

weight loss (Evidence Category A) and 4) reduces cardiovascular disease risk factors and reduces risk for cardiovascular disease (Evidence Category C). Very little of the background literature pertains to menopausal women.

Only a very few randomized controlled trials have tested the effect of exercise on body fat and abdominal fat in a design allowing a firm conclusion on the effect of exercise *per se*, either comparing exercise versus nonexercise (13,18) or comparing diet versus diet plus exercise (5,25,26). They are summarized in Tables 1 and 2. The study populations were generally overweight or obese, but in one study women with low levels of HDL cholesterol and high levels of LDL cholesterol only were studied (25), and in the study by Seals et al. (23) postmenopausal women with hypertension were studied. Only the 12-wk study by Svendsen et al. (26) found significant additional weight and fat loss when a combination of aerobic and anaerobic exercise training was added to an energy restricted diet. Marked improvements in cardiovascular risk factors were observed in both intervention groups, but no additional benefit was detected in the exercise group (26). At 6 months' follow-up little weight was regained and a weight loss of about 8 kg was maintained in both intervention groups (27). The women from the former exercise-plus-diet group, who continued to exercise at follow-up, had greater reductions in body weight, body fat, and abdominal fat than those who were not exercising (27).

CONCLUSIONS ON CURRENT EVIDENCE

Observational cross-sectional and longitudinal studies show that postmenopausal women with high levels of physical activity have lower body fat and abdominal fat and are less likely to have gained total and abdominal fat during menopause than those with low levels of physical activity (Evidence Category C).

RCT comparing exercise with no intervention and diet with diet plus exercise in postmenopausal women are very few, and the results do not allow a firm conclusion as to whether physical activity may prevent or limit the gain of total fat and abdominal fat during menopause or whether it may be effective as part of an obesity treatment program.

CONCLUSIONS ON RESEARCH ISSUES

There is a need for RCT to evaluate the effect of increased physical activity and fitness as a tool for prevention of the changes in body composition associated with menopause and aging in normal women. The influence of the abdominal fat gain independent of total fat gain associated with menopause on health risk needs further clarification (cardiovascular disease, diabetes, osteoporosis, breast cancer). Further, the efficiency of different programs of exercise training as a treatment option in subjects with existing overweight and obesity should be investigated both alone and in combination with dietary therapy. RCT should have the appropriate design and size and use accurate methods to assess exercise compliance, cardiovascular fitness, body composition, and intra-abdominal adipose tissue. Concomitant HRT should be assessed as a possible effect modifier. Studies should also assess the importance of ethnic origin, race, and socioeconomic factors.

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Is abdominal fat preferentially reduced in response to exercise-induced weight loss?

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ABSTRACT

ROSS, R. and I. JANSSEN. Is abdominal fat preferentially reduced in response to exercise-induced weight loss? *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S568–S572, 1999. **Purpose:** It is known that a preferential deposition of fat in the abdominal region is the obesity phenotype that conveys the greatest health risk. Although physical activity is commonly prescribed to reduce obesity, the influence of exercise-induced weight loss on abdominal fat is unclear. This review was undertaken to clarify whether abdominal fat is preferentially reduced consequent to weight loss induced by regular exercise. **Methods:** A literature search (*Medline*, 1966–1998) was performed using appropriate keywords to identify studies reporting changes in both whole body and abdominal fat in response to exercise. **Results:** At present there are no randomized controlled trails (RCT) wherein it was clear that exercise alone induced weight loss. For the four RCT within which regular exercise was not associated with weight loss, abdominal fat measured by waist circumference was unchanged. A similar trend is observed for the nonrandomized studies. Abdominal obesity as measured by waist circumference is unchanged for those studies reporting no loss in weight or fat; however, a modest reduction (~3 cm) is observed in response to exercise-induced weight loss of about 3 kg. Without exception, these studies were not designed to determine whether abdominal obesity was preferentially reduced. Absent from the literature are RCT that employ imaging techniques (e.g., computerized tomography or magnetic resonance imaging) to determine whether exercise-induced weight loss is associated with reductions in either visceral or abdominal subcutaneous fat. However, the findings from four nonrandomized or controlled studies report that exercise with or without weight loss is associated with reductions in both visceral and subcutaneous fat. **Conclusions:** There is insufficient evidence to determine whether exercise-induced weight loss is associated with reductions in abdominal fat. Clearly there is a need for carefully controlled studies wherein the primary aim is to determine the influence of regular exercise on total and abdominal adiposity. **Key Words:** PHYSICAL ACTIVITY, ABDOMINAL OBESITY, VISCERAL FAT, SUBCUTANEOUS FAT

Abdominal obesity, in particular abdominal subcutaneous and visceral fat, are known to be independent predictors of the metabolic risk factors that are antecedents for cardiovascular disease and type 2 diabetes (7,8,14,18). This suggests that treatments designed to reduce obesity and related co-morbidities would ideally be associated with substantial reductions in abdominal fat. As exercise is a common feature of obesity reduction programs, it is important to clarify whether exercise-induced weight loss is associated with corresponding reductions in abdominal obesity. Indeed, the intent of this review was to determine whether exercise-induced weight loss is associated with corresponding reductions in abdominal obesity measured by external anthropometry (e.g., waist circumference) and/or abdominal subcutaneous and visceral fat measured using imaging techniques.

The format of this review follows the guidelines set forth in the recent National Institutes of Health, National Heart, Lung, and Blood Institute (NHLBI) document (17). As such the *Current Knowledge* section is comprised of a series of evidence statements followed by a brief rationale for each. Following each evidence statement is an evidence category that, for the most part, is consistent with the criteria established by the Expert Panel (17). The single exception is that in this review, randomized, controlled trials (RCT) less than 4 months in duration were weighted similarly to longer term trials. The rationale for this position is provided within the review.

To consider whether exercise-induced weight loss is associated with concomitant reductions in abdominal obesity a *Medline* search (1966–1998) was performed using appropriate keywords. The reference lists of those studies identified were then reviewed for additional studies. Appropriate studies were identified using the following inclusion criteria:

1. The subjects participating in the exercise group had to consume an isocaloric diet for the duration of the study, thereby ensuring that the negative energy balance observed (e.g., significant weight loss and/or body composition change) was induced by the increase in physical activity.
2. Measurements of whole-body and regional fat distribution were obtained, and the authors reported whether abdominal fat (measured either by waist circumference or imaging techniques) was preferentially reduced.

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TABLE 1. Effect of physical activity without weight loss on abdominal obesity measured by anthropometry: evidence from randomized, controlled trials.

References	Subjects		Treatment	Study Duration	Reduction in Weight (kg)	Reduction in Body Fat	Reduction in WHR	Reduction in WC (cm)
	Sex	BMI (kg)						
Stefanick et al., 1998 (24)	Hyperlipidemic premenopausal women	(~70)	Control group	1 yr	+0.8	Not reported	0.01	Not reported
			Aerobic exercise		0.4		0.01	
	Hyperlipidemic men	(~84)	Control group	1 yr	+0.5	Not reported	0.01	Not reported
			Aerobic exercise		0.6		0.01	
Mourier et al., 1997 (16)	NIDDM men and women	30 ± 2	Control group	2 months	0.2	1.3%	0.01	0.0
		30 ± 1	Aerobic exercise		1.5	0.6%	0.03	1.0
Treuth et al., 1994 (26)	Older men	(75 ± 9)	Control group	4 months	NS	+0.6 kg	NS	NS
		(85 ± 12)	Resistance exercise		NS	2.0 kg*	0.03	+2.0
Ballor et al., 1988 (3)	Obese women	(73 ± 2)	Control group	2 months	0.4	0.1 kg	Not reported	+0.4
		(74 ± 3)	Aerobic exercise		0.5	0.6 kg		+0.6
Hellénius et al., 1993 (11)	Hyperlipidemic men	25 ± 3	Control group	6 months	+0.3 (BMI)	Not reported	0.05	0.3
		25 ± 3	Aerobic exercise		0.3 (BMI)*		0.06	2.2*
Dengel et al., 1996 (6)	Obese older men	(90 ± 4)	Control group	10 months	0.2	0.4 kg	0.00	Not reported
		(95 ± 5)	Aerobic exercise		0.5	0.5 kg	0.01	
Katzel et al., 1997 (12)	Obese older men	30 ± 2	Control group	9 months	NS	NS	NS	NS
		30 ± 2	Aerobic exercise		NS	1.0%	NS	NS

* Significant within group change, Exercise > Control ($P < 0.05$).
BMI, body mass index; WHR, waist-to-hip ratio; WC, waist circumference; NIDDM, non-insulin-dependent diabetes mellitus; NS, nonsignificant change ($P > 0.05$).

3. The subjects had a body mass index (BMI) greater than 30 $kg \cdot m^{-2}$.

The initial literature search could not identify any study that met all criteria. Thus, it is not possible to consider whether abdominal fat is preferentially reduced when the energy deficit is induced by regular exercise.

A second review was performed using the following selection criteria:

1. All subjects in the exercise group were instructed not to change their diet (eating) habits, and thus in theory a negative energy balance would be induced by an increase in exercise.
2. Measurements of whole-body and regional fat distribution were obtained, and the authors reported whether abdominal fat was preferentially reduced.

Five RCT (3,11,16,24,26) and nine nonrandomized studies (2,4,5,9,10,13,15,23,25) met the first of these criteria. However, without exception these studies were not designed to determine whether there was a preferential reduction in abdominal fat. Because of the paucity of data we included two RCT wherein the subjects in the exercise group were instructed to maintain weight (6,12).

CURRENT STATUS OF KNOWLEDGE

Evidence Statement: Physical activity, in the absence of weight loss, is not associated with reductions in abdominal obesity as measured by waist circumference or waist-hip-ratio. (Evidence Category B). Seven RCT consider whether exercise has an influence on abdominal obesity as measured by waist circumference and/or waist-hip-ratio (Table 1). For five studies the subjects in the exercise group were instructed not to change their dietary (energy consumption) habits (3,11,16,24,26). Despite this instruction, in all but one study the addition of exercise did not result in body weight or fat loss, nor was abdominal obesity reduced. In contrast to these observations, waist circumference decreased by 2 cm within the single study that reported a significant exercise-induced weight loss (11). In summary, the evidence from these studies suggest that physical activity without weight or fat loss is not associated with a reduction in abdominal obesity (Table 1).

A similar trend is observed for the nonrandomized studies (Table 2). Whereas waist circumference is unchanged for those studies reporting no loss in weight or fat (5,10,13), a modest reduction is observed in response to weight loss of about 2 kg (2,15,23). A caveat in interpretation of those studies reporting an exercise-induced decrease in abdominal obesity is that waist circumference was measured at the level of the umbilicus. Waist circumference measured at this level is not recommended for use in intervention studies since the umbilical level may change with weight loss (19,21,22).

From the studies in Tables 1 and 2 come the following summary observations:

1. There are no RCT wherein it is clear that physical activity alone has induced a negative energy balance.
2. Based on limited RCT evidence, exercise without weight loss is not associated with a reduction in abdominal obesity measured by waist circumference.
3. Limited evidence from nonrandomized or controlled studies suggests that a modest reduction in waist circumference is observed in response to weight loss.
4. Without exception, these studies were not designed to determine whether abdominal obesity was preferentially reduced.

Evidence Statement: Physical activity with or without weight loss is associated with reductions in visceral and abdominal subcutaneous adipose tissue. (Evidence Category C). Absent from the literature are RCT that consider whether visceral or abdominal subcutaneous fat is preferentially mobilized when the energy deficit is induced by regular physical activity. However, we are aware of a single RCT wherein the effects of

TABLE 2. Effect of physical activity on abdominal obesity measured by anthropometry: evidence from nonrandomized trials.

References	Subjects		Treatment	Study Duration	Reduction in Weight (kg)	Reduction in Body Fat	Reduction in WHR	Reduction in WC (cm)
	Sex	BMI						
Katzel et al., 1997 (13)	Obese men	29	Aerobic exercise	9 months	0.1	0.9%	0	0.5
Fonong et al., 1996 (10)	Older men and women	~25	Aerobic exercise	2 months	0.2	0	NR	2
Lehmann et al., 1995 (15)	NIDDM men and women	31	Control group	3 months	0.0	0.5%	0.00	0.0
		31	Aerobic exercise		0.7	2.3%	0.03†	3.3†
Andersson et al., 1991 (2)	9 men	~25	Aerobic exercise	3 months	2.0*	2.9 kg*	0.01	2.9*
	22 women		Aerobic exercise		0.7	2.6 kg*	0.01	3.6*
Coon et al., 1989 (5)	Obese older men	31	Weight loss group	9 months	11.0*	6.0%*	0.02	NR
		30	Aerobic exercise		0.2	0.0%	+0.02	NR
Schwartz et al., 1991 (23)	Young men	26	Aerobic exercise	~7 months	0.5	1.6 kg	0.00	1.8*
	Older men	26	Aerobic exercise		2.5*	2.4 kg	0.02*	3.2*

* Significant within group change ($P < 0.05$).
† Significantly different than change in control group ($P < 0.05$).
BMI, body mass index; WHR, waist-to-hip ratio; WC, waist circumference; NR, not reported; NIDDM, non-insulin-dependent diabetes mellitus.

exercise alone on visceral and subcutaneous fat *per se* were considered (Table 3). Mourier et al. (16) observed large reductions in both visceral (~48%) and abdominal subcutaneous adipose tissue (~18%) in response to moderate exercise performed three times per week for 8 wk in men and women with type 2 diabetes. In that study no change in body weight was observed (Table 3).

Four nonrandomized studies considered the effects of exercise alone on visceral and subcutaneous fat (Table 3). Table 3 reveals that exercise with (4,9,23) or without weight loss (23,25) is associated with reductions in both visceral and abdominal subcutaneous fat. These studies were not designed to determine whether visceral fat was preferentially reduced in comparison with total fat.

The majority of the studies reviewed did not monitor energy intake carefully, nor did they prescribe an exercise program that resulted in a meaningful energy expenditure. For example, in the study by Mourier et al. (16) the regimen prescribed included stationary cycling three times per week for approximately 30 min. This represents a very modest energy expenditure and no doubt at least partially explains the absence of weight loss. In contrast, Bouchard et al. (4) rigorously controlled energy intake through the prescription of an isocaloric diet for the duration of a 3-month study (Table 3). In that study the seven twin pairs exercised on a stationary bicycle 6 $d \cdot wk^{-1}$ such that 1000 kcal were expended each day. In response to this program the authors observed that an exercise-induced 5 kg reduction in body weight was associated with a 36% reduction in visceral adiposity. Although this study did not include a control group, to our knowledge it is the only study wherein the effects of exercise-induced weight loss on visceral adiposity are reported.

Consistent with evidence from RCT, several studies in Table 3 report exercise-induced reductions in visceral and subcutaneous fat without a corresponding change in waist circumference. In fact this is true for the study that reported the greatest reduction in visceral fat (16). As the magnitude of the reduction in visceral and subcutaneous fat is greater than the error generally attributed to repeated measurements of abdominal fat by either computerized tomography or magnetic resonance imaging (1,20), it is assumed that the reductions observed in visceral fat are real and not the consequence of technical error. Thus it is unclear why a substantial reduction in abdominal adipose tissue is observed without a corresponding reduction in waist circumference. These observations are troubling, however, as they suggest that the influence of exercise on abdominal obesity measured by external anthropometry may provide misleading results.

RESEARCH PRIORITIES

Issues and limitations. Absent from the literature are RCT in which the primary objective is to determine the

TABLE 3. Effect of physical activity on visceral and abdominal subcutaneous adipose tissue.

References	Subjects		Treatment	Study Duration	Reduction in Weight (kg)	Reduction in Body Fat	Reduction in VAT cm^2 (%)	Reduction in SAT cm^2 (%)	VSR	Reduction in WC (cm)
	Sex	BMI (kg)								
Randomized Control Trials										
Mourier et al., 1997 (16)	NIDDM men and women	30 ± 1	Control group	2 months	0.2	1.3%	5 (3)	9 (3)	NR	0.0
		30 ± 1	Aerobic exercise		1.5	0.6%	76 (48)†	41 (18)†		1.0
Nonrandomized Control Trials										
Treuth et al., 1995 (25)	Older women	25 ± 1	Resistance exercise	4 months	0.1	0.4 kg	14 (10)*	17 (6)	−0.04*	+2.0
Bouchard et al., 1994 (4)	14 young men (twins)	(82 ± 5)	Aerobic exercise	~3 months	5.0*	5.0 kg*	29 (36)*	67 (27)*	NR	NR
Schwartz et al., 1991 (23)	Young and older men	26 ± 3	Young men (28 yr)	~7 months	0.5	1.6 kg	11 (17)*	21 (10)*	NR	1.8*
		26 ± 3	Older men (67 yr) (Aerobic exercise)		2.5*	2.4 kg*	35 (25)*	35 (20)*		3.2*
Després et al., 1991 (9)	Obese women	34 ± 4	Aerobic exercise	14 months	3.7*	4.6 kg*	3 (3)	60 (11)*	NR	NR

* Significant within group change ($P < 0.05$).
† Significantly different than change in control group ($P < 0.05$).
BMI, body mass index; VAT, visceral adipose tissue; SAT, subcutaneous adipose tissue; VSR, viseral-to-subcutaneous ratio; WC, waist circumference; RCT, randomized controlled trial; NIDDM, non-insulin-dependent diabetes mellitus; NR, not reported.

influence of exercise-induced weight loss on total and regional fat distribution. Accordingly, the potential influence that age, race, gender, and exercise modality or intensity may have on exercise-induced changes in fat distribution is unknown. Moreover, for those studies that have attempted to define the exercise-induced effects on regional adiposity, energy intake and expenditure are poorly controlled. In addition, we are unaware of any study that reports whether abdominal fat is preferentially reduced in comparison with total adiposity. Thus the following questions remain unanswered:

1. Is exercise-induced weight loss associated with a reduction in abdominal obesity (visceral and/or subcutaneous) that is greater than exercise alone (e.g., no weight loss)?
2. Is there a preferential reduction in abdominal obesity?
3. What is the impact of varying degrees of exercise-induced weight loss on abdominal adiposity? Are reductions in abdominal obesity linearly related to the magnitude of weight loss (i.e., 7 vs 15%)?
4. Is equivalent diet- or exercise-induced weight loss associated with a reduction in abdominal obesity (visceral and/or subcutaneous), and if so, are there treatment differences?

RECOMMENDATIONS

Shorter trials be used to determine the influence of exercise-induced weight loss on abdominal fat. Assurance that exercise is uniquely responsible for inducing a negative energy balance requires tight control of energy intake. This implies that attempts must be made to ensure the consumption of an isocaloric diet throughout the course of the study. This is of particular concern when the magnitude of the energy expenditure induced by exercise is relatively small. Indeed, for most of the studies reviewed, in particular the RCT, it is likely that small variations in energy intake compensated for the increased energy expended during exercise. As a consequence little change is observed in either body weight or total and regional fat. Tight control of energy intake is not easily accomplished for long-term trials (e.g., 1 yr). It is suggested, therefore, that shorter term trials (e.g., 4 months) may be more feasible with respect to controlling both energy intake and expenditure. In other words, shorter trials permit more rigorous control of energy intake and would also facilitate the prescription of more extensive daily exercise regimens.

Increase the energy expenditure induced by the exercise treatment. For the most part researchers have employed exercise regimens that are characteristically associated with improvements in cardiovascular fitness (e.g., peak $\dot{V}O_2$) but not necessarily associated with significant energy expenditure and thus meaningful weight loss. If the objective is to determine the influence of regular exercise on obesity and its co-morbidities, it is suggested that the exercise program be designed with the priority to increase energy expenditure. In this way low intensity exercise (e.g., 50–60% of maximum heart rate (brisk walking)) should be prescribed on an almost daily basis for longer durations (e.g., ~60 min).

Use a bony landmark (e.g., floating rib) to locate waist circumference measurement. That waist circumference did not change in two of the three studies wherein substantial reductions in visceral and subcutaneous fat was observed is a concern. The use of a bony landmark to guide the measurement of waist circumference in intervention will help assure that technical issues do not explain this phenomenon. It has been reported that reductions in waist circumference measured at the level of the last rib (floating rib) follow corresponding reductions in visceral fat (22).

Acquire total and regional body composition measurements. Determination of whether abdominal fat is preferentially reduced in response to exercise requires obtaining both whole body and regional measurements of fat distribution. Although not generally feasible, these measurements ideally would be obtained using either imaging or duel energy x-ray absorptiometry techniques. Acquisition of whole body and regional measurements of fat distribution would permit the researcher to identify the separate contribution of abdominal fat verses, for example, gluteal femoral or lower body fat. Traditionally, determination of differences in regional obesity with weight loss have entailed comparisons of abdominal versus whole body adiposity. This analysis is confounded by the inclusion of abdominal obesity in the whole body measurement.

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Physical activity and regulation of food intake: current evidence

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ABSTRACT

BLUNDELL, J. E., and N. A. KING. Physical activity and regulation of food intake: current evidence. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S573–S583, 1999. **Objective:** The evidence was reviewed on how physical activity could influence the regulation of food intake by either adjusting the sensitivity of appetite control mechanisms or by generating an energy deficit that could adjust the drive to eat. **Design:** Interventionist and correlational studies that had a significant influence on the relationship between physical activity and food intake were reviewed. Interventionist studies involve a deliberate imposition of physical activity with subsequent monitoring of the eating response. Correlational studies make use of naturally occurring differences in the levels of physical activity (between and within subjects) with simultaneous assessment of energy expenditure and intake. **Subjects:** Studies using lean, overweight, and obese men and women were included. **Results:** Only 19% of interventionist studies report an increase in energy intake after exercise; 65% show no change and 16% show a decrease in appetite. Of the correlational studies, approximately half show no relationship between energy expenditure and intake. These data indicate a rather loose coupling between energy expenditure and intake. A common sense view is that exercise is futile as a form of weight control because the energy deficit drives a compensatory increase in food intake. However, evidence shows that this is not generally true. One positive aspect of this is that raising energy expenditure through physical activity (or maintaining an active life style) can cause weight loss or prevent weight gain. A negative feature is that when people become sedentary after a period of high activity, food intake is not "down-regulated" to balance a reduced energy expenditure. **Conclusion:** Evidence suggests that a high level of physical activity can aid weight control either by improving the matching of food intake to energy expenditure (regulation) or by raising expenditure so that it is difficult for people to eat themselves into a positive energy balance. **Key Words:** EXERCISE, APPETITE CONTROL, ENERGY INTAKE, EATING, ENERGY EXPENDITURE

Understanding the interaction between physical activity and energy intake (EI) leads to both theoretical and practical implications. More than 40 years ago, the common sense view implied that "the regulation of food intake functions with such flexibility that an increase in energy output due to exercise is automatically followed by an equivalent increase in caloric intake" (60, p. 169). Mayer went on to point out the fallacies inherent in such an attitude. Mayer's own findings demonstrated that physical activity was not invariably coupled to EI and even produced evidence that very low physical activity (sedentariness) was associated with high EI. Today, there is considerably more evidence that EI (food consumption) is only weakly coupled to exercise-induced metabolic activity. Indeed, this may be one example of a more general phenomenon indicating a weak relationship between metabolic variables and food intake or eating patterns.

One possible reason for this is that EI cannot simply be considered as the self-administration of fuel. Energy intake signifies eating behavior, and behavior is shaped and driven by both biological (under the skin) and environmental (beyond the skin) variables as well as by mental events. It is widely accepted that behavioral patterns can be held in place by environmental contingencies (relationships between cues and responses) by associations between physiological signals and salient environmental stimuli (including food stimuli), as well as by more obvious social and cultural influences. The major importance of this perspective is that, once established, a pattern of eating and food choice can be maintained independently of many physiological events. In other words, there appears to be a lack of tight coupling between the behavior which forms the basis of EI (i.e., eating), the behavioral vehicle for energy expenditure (i.e., physical activity) and the metabolism associated with energy expenditure (EE).

One practical consequence of this loose coupling concerns the role of physical activity in the control of body weight. Again, as stated by Mayer (60), the idea that an activity-induced increase in energy output is automatically followed by an increase in EI often leads "to the disparagement of physical activity as a factor in weight control" (p. 169).

POSSIBLE RELATIONSHIPS BETWEEN PHYSICAL ACTIVITY AND FOOD INTAKE

In examining the relationship between exercise and EI, one major issue is what happens to EI when physical activity is increased? Four major possibilities can be envisaged:

1) A compensatory effect occurs such that as EE increases, there is a corresponding rise in EI. If there is a compensatory increase in EI, will it be sufficient to fully compensate for the increase in EE?

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2) An exercise-induced suppression of EI takes place in which the metabolic or psychological effects of physical activity suppress EI.

3) No change in EI in response to change in activity-induced EE.

4) An exercise-induced alteration in food choice or nutrient selection occurs.

The strongest logical argument would favor possibility number 1. However, if a compensatory increase in EI did occur, this could only come about by some combination of the following circumstances:

1. Increase size of individual eating episodes
2. Increase frequency of eating episodes
3. Increase energy density ($kJ \cdot g^{-1}$) of foods consumed
4. Increase intake of energy-rich fluids.

In considering why food intake may fail to change in response to alterations in physical activity, it should be kept in mind that eating (meal patterns, food choice, etc.) is a set of behaviors that have been built up over many years and that are heavily influenced by physiological limits (e.g., preabsorptive factors) and social influences (6). Therefore, any change in metabolic activity has to overcome these two factors. The ways in which EI can be increased require a mandatory change in some form of behavior.

CLASSIFICATION OF STUDIES

Various types of studies can be called on to yield information pertaining to the effect of physical activity on the regulation of food intake. The studies can be classified into three main approaches and are summarized in Tables 3–5 (at the end of the text).

1. Intervention studies. "What is the EI response to an activity-induced change in EE?"

2. Correlational studies (between subjects). "Is EI positively associated with differing levels of activity-induced EE in different groups of subjects?"

3. Correlational studies (within subjects). "Does EI change systematically with differing levels of physical activity in the same individual?"

In intervention studies, there is a deliberate imposition of physical activity EE (increase or decrease) with subsequent measurement of food (energy) intake. Within this category, the studies can be further categorized according to the length of the imposed change in EE and the associated measurement of EI (e.g. short-term, 1–10 d; medium-term, 2–8 wk; and long-term, 2–12 months). The short-term studies are typically laboratory based in which both the imposition of EE and measurement of EI (*ad libitum* test meal) are accurately carried out within the laboratory environment. The medium- and long-term studies typically include a less accurate method of measuring the subsequent effect on food intake, e.g., self-record food diaries. The remainder of the studies can be classified as correlational. This means that there is no experimenter imposed change in activity (EE) but self-selected levels of physical activity are monitored together with the ongoing level of EI. There are two types of correlational designs: within and between subjects. In between-subjects studies, the EE and EI are measured in *groups* of individuals with differing chosen levels of physical activity (e.g., sedentary vs moderately active vs extremely active). This type of study indicates whether EE is related to the EI shown by the different groups. The within-subjects correlational study involves measurement of EE and EI in the same individuals performing different amounts of physical activity at different times during their normal lifestyle (e.g., coal miners and soldiers) or when undertaking specified amounts of physical activity. This type of study should indicate how EI responds to changing levels of EE (activity-induced) in the same individuals on different occasions. Therefore, in both types of correlational studies, no deliberate attempt is made to alter an individual's habitual activity. It can be seen that the different experimental designs will possess differing intrinsic strengths and weaknesses.

Apart from the structural differences in the designs of intervention and correlational studies, there is also a major substantive issue. The intervention studies seek to record processes that may accompany a change from one state to another (usually from sedentariness to activity). In other words, these studies should reflect a *dynamic process*. However, correlational studies generally compare differences between *stable states* (rather than the process of change from one state to another). This difference should reflect the consequences of adjustment itself, and the "*settling down*" after adjustment has occurred.

DOES AN ENERGY DEFICIT, INDUCED BY EXERCISE, CREATE AN AUTOMATIC INCREASE IN HUNGER AND FOOD INTAKE?

Many people believe that exercise generates an increase in hunger and a drive to eat. It is also a truism that refraining from eating prevents energy entering the biological system, whereas doing physical activity causes energy to leave the system. Therefore, at a given time, failing to eat (when a meal is usually taken) or doing exercise (when it normally would not have been done) should each create a net negative energy balance. There are many examples in the literature of food restriction (skipping a meal) or an energy differential (between two meals of different sizes) giving rise to hunger and increased food intake (14,31,35,51). The response to this type of energy deficit appears logical and is well accepted. The relationship between exercise-induced energy deficits and food consumption has been investigated, and most of the evidence shows that there is no increase in hunger or EI as a result of an exercise-induced energy deficit (36,43–45,47,49,56,66,77,89).

Overall, the body of evidence points to a rather weak coupling between EI and physical activity-induced EE (7,46,48). Tables 1 and 2 show a summary of the results of all the studies included in Tables 3, 4, and 5. For those studies that imposed a deliberate increase in physical activity, 31 indicated no corresponding change in EI, 9 reported an increase, and 8 reported a decrease. For the correlational studies, 15 indicated a positive relationship between EE and EI, and 12 showed no relationship.

A criticism of some of the studies (especially the short-term interventions) is that EI may not have been tracked for

TABLE 1. Summary of intervention studies indicating whether energy intake decreases, increases, or shows no change after the introduction of physical activity.

Duration	Decrease	Increase	No change
Short-term			
Obese and OW	1	0	4
Lean	2	6	14
Medium-term			
Obese and OW	1	0	3
Lean	0	1	1
Long-term			
Obese and OW	2	1	4
Lean	2	1	5
Total	8	9	31

a sufficiently long enough period of time after the increased physical activity. It was claimed by Edholm et al. (22) that although there was no relationship between EE and EI on the same day, there was a positive relationship between EE on one day and EI 2 d later. This led Edholm et al. to remark that "We do not eat for today but for the day before yesterday" (21). However, despite subsequent attempts, the same authors failed to replicate this finding (23). This phenomenon of a 2-d delayed compensation has been subject to criticism (17), and more recently, to further scrutiny (13,67). De Castro (13) failed to detect any relationship between physical activity and food intake on several days of self-monitoring. Only with the energy balance data from Tour de France cyclists (67) was there a confirmation of the 2-d lag between EE and intake. However, with these particular elite athletes, there is also a very clear association between EI and EE on any particular day of the race.

In general, the outcomes of the intervention studies challenge the commonly held view that exercise induces a physiological need that automatically generates a drive to eat to restore the energy used. Instead, the evidence points to a rather weak coupling between activity-induced EE and EI. One reason for this is that the behavioral acts of food intake are held in place by environmental contingencies and short-term postingestive physiological responses arising from eating itself. Exercise-induced changes in postabsorptive physiology (energy metabolism) appear to have only a weak influence on eating behavior. This weak coupling between EE and EI has positive implications for the use of physical activity in weight loss or weight maintenance.

DOES PHYSICAL ACTIVITY SUPPRESS APPETITE? (EXERCISE-INDUCED ANOREXIA)

The idea that physical activity can suppress appetite is commonly encountered. Two phenomena are apparent. The first is a very short-term transient suppression of hunger that can occur during and shortly after a period of vigorous or intense exercise (43–45,49,77,89). This suppression is not seen with moderate exercise. The second phenomena is more dramatic and indicates how under extreme circumstances (endurance exploring or long-distance swimming) appetite is suppressed so that individuals cannot take in sufficient energy to match the very high levels of EE. Here individuals can sustain a negative energy balance (on average between 2 and 7 $MJ \cdot d^{-1}$) for up to 21 d in a high-altitude climbing expedition (88), 48 d in Arctic trekking (73), or 55 d in the case of trans-Atlantic swim (62). In this latter case, the swimmer, Guy Delage, had increased his body weight by 17 kg before the swim in the expectation of a weight loss. Indeed, despite the provisions being especially designed to include his most preferred foods, for the first 2 wk Delage suffered from a severe loss of appetite and, even later, his daily EI never matched his daily expenditure. Consequently, at the end of the swim he had lost 14 kg body weight.

These extreme environmental conditions are clearly not typical of day-to-day living, but they do provide evidence that the body can tolerate large energy deficits for considerable periods of time when EE due to physical activity is high. However, in certain elite athletes, large daily EE can be matched by equally large daily EI (68). It has recently been suggested that "highly experienced" cyclists, such as those who participate in the Tour de France, have learned how to eat an enormous amount of food during hard physical work (86). In addition, these circumstances are different from endurance explorers. For example, the Tour de France cyclists are able to retire after the day's cycling to a luxury hotel and a soft bed with a conducive environment for eating and recuperation. This is obviously not available to individuals carrying their own provisions and isolated in the middle of an Arctic icefield or the Atlantic Ocean. It is also worth noting that these elite cyclists are aware that if EI is insufficient, performance will be impaired and they may not survive the next stage of the Tour. Therefore, individuals engaged in regular heavy physical activity probably eat both in anticipation of future energy requirements, as well as in response to energy expended. Consequently, the intensity and duration of physical activity, together with the nature of the environment in which the activity is carried out, will influence the time taken to reach a new equilibrium of energy balance at a new body weight.

IS FOOD INTAKE DOWN REGULATED WHEN PHYSICAL ACTIVITY IS REDUCED?

Why is it that when people change from an active to a sedentary lifestyle, food intake is not drawn downward to a new lower level in equilibrium with the reduced EE? This issue was addressed more than 40 years ago by Mayer and colleagues, who demonstrated in both rats (59) and humans (60) that substantial decreases in activity (into the sedentary range) could be associated with increases in food intake and obesity. Recent findings have confirmed that EI remains at some stable preferred level even when EE is manipulated. In two separate studies (using two separate sets of individuals), in which identical *ad libitum* diets were fed, EE and EI were measured either in a free-living environment (75) or in a whole body calorimeter (74). Subjects ate virtually identical amounts in both situations, despite the EE being substantially different (difference

TABLE 2. Summary of correlational studies indicating the existence of an association between measured (estimated) energy intake and energy expenditure under various levels of physical activity.

	No Relationship between EE and EI	Positive Relationship between EE and EI
Between subjects	4	7
Within subjects	8	8
Total	12	15

TABLE 3a. Short-term intervention studies that have examined the effect of physical activity on appetite.

Author(s) and Year	Gender, Sample Size, and Age (yr)	Weight	Protocol	Results
Almeras et al., 1995 (2)	Male N = 11 age^R = 24–36	Lean	Two treatments: exercise + rest; 1 d of exercise (or rest) and 2 d of *ad libitum* food intake	No effect of exercise on food or macronutrient intake; relationship between exercise respiratory quotient and energy intake
Durrant et al., 1982 (20)	Male and female N = 2 and 14 age^M (lean = 22.0; obese = 27.0)	Lean and obese	3 d of rest and 3 d of exercise (cycling) to increase expenditure by 100 kcal·d^{-1}; food measured *ad libitum*	Increase in energy intake in lean but not in obese
Gilsenan et al., 1998 (30)	Male N = 7 age^M = 37.0	Lean	Two separate 1-d periods in a whole body calorimeter; 1 d of no exercise (low EE) and 1 d of exercise (normal EE); Food measured *ad libitum*	No difference in energy intake between two periods despite differing levels of energy expenditure
Horton et al., 1994 (34)	Female N = 10 age^M = 32.0)	Lean	Energy expenditure and energy intake measured for 2 separate days in a whole body calorimeter in a group of cyclists and noncyclists; 1 d of increased activity and 1 d of reduced activity; food measured using *ad libitum* test meals	Cyclists in energy balance on day of cycling and noncyclists in energy balance on day of noncycling; no reduction in energy intake in cyclists on day of reduced activity
Imbeault et al., 1997 (36)	Male N = 11 age^M = 24.4	Lean	Two intensities (30 and 70% $\dot{V}O_{2max}$) of exercise and one control condition; energy intake measured using *ad libitum* test meal	No effect of exercise on energy intake and hunger; intense exercise induced a larger relative energy intake than low-intensity exercise
Jankowski and Foss, 1972 (38)	Male N = 14 age^M = 26.3	Overweight	Three treatment periods: running for 1 mile at 6.2 mph, running for 440 yards at 6.2 mph and resting; foods in house preweighed before study and 24 h later	No difference in energy intake
King et al., 1994 (43)	Male N = 11 age^R = 21–27	Lean	Study 1: three treatment conditions; rest period, low-intensity exercise, and high-intensity exercise (cycling); food intake measured using *ad libitum* test meal	Transitory suppression of subjective feeling of hunger with intense exercise only; no effect on energy intake
	Male N = 12 age^R = 22–31	Lean	Study 2: three treatment conditions; rest period, high-intensity (short-duration), and high-intensity (long-duration) exercise (cycling)	Transitory suppression of subjective feeling of hunger after exercise; no effect on energy intake
King and Blundell, 1995 (44)	Male N = 12 age^M = 24.4	Lean	Study 1: four treatment conditions; 2 periods of rest, one followed by high-fat meal, other by low-fat meal; 2 periods of exercise (cycling), one followed by high-fat meal, other by low-fat meal	Transitory suppression of hunger after exercise; increase in energy intake during high-fat meal; no effect on energy intake
King and Blundell, 1995 (44)	Male N = 12 age^M = 24.2	Lean	Study 2: As study 1, except cycling session replaced with treadmill running	As above; no difference between the effects of cycling and running
King et al., 1996 (45)	Female N = 13 age^M = 22.6	Lean	Repeat of King and Blundell (1995; study 1) except dietary unrestrained female subjects	No effect of exercise on food intake hunger; exercise induced an increase in perceived pleasantness of both low-fat and high-fat meals
King et al., 1997 (47)	Male N = 8 age^M = 26.0	Lean	2 d of an exercise treatment (1 d exercise + 1 d rest) compared with 2 d of a rest treatment (2 d rest); food intake monitored using self-record food diaries	No increase in hunger or food intake on day of exercise or day after; no difference in daily energy intake between exercise and rest treatments
Kissileff et al., 1990 (49)	Female N = 18 age^M (lean = 22.7; obese = 24.3)	Lean and obese	Two intensities of cycling; high (40 min at 90 W) and low (40 min at 30 W); control period of no exercise; test meal 15 min postexercise	Reduction of food intake after intense exercise in lean but not in obese individuals
Lavin et al., 1998 (50)	Male N = 16 age^R = 22–30	Lean	Two intensities (30 and 70% $\dot{V}O_{2max}$) of exercise and a control (rest) condition followed by an *ad libitum* test meal 1 h later	Energy intake higher after both exercise sessions compared with rest
Lluch et al., 1998 (56)	Female N = 12 age^M = 21.7	Lean	Repeat of King and Blundell (1995) study 2 except using dietary restrained female subjects	No effect of exercise on energy intake; exercise induced an increase in pleasantness of the low-fat meal only
McGowan et al., 1986 (61)	Male N = 7 age^M = 24.7	Lean	21 d of three different phases of training loads: 7 d normal pace and mileage, 7 d of normal pace and double mileage, and 7 d of rest (no training); food intake measured using self-record food diaries	No change in energy intake between the three phases
O'Hara et al., 1977 (63)	Male N = 6 age^M = 38.0	Obese	10 d of 3.5 h of exercise in a cold (−34°C) chamber; food intake measured using self record food diaries	No effect of exercise on energy intake
Passmore et al., 1952 (65)	Male N = 5 age^R = 19–25	Lean	13 consecutive d of 3 d of sedentary, 5 d of hard physical work (outdoor walking), and 5 d of sedentary phase; weighed intakes of food eaten in lab	Energy expenditure matched energy intake during all phases
Reger et al., 1984 (66)	Female N = 11 age^M = 27.0	Lean	Three treadmill running treatments; long-duration (60 min at 50% $\dot{V}O_{2max}$), short-duration (30 min at 50% $\dot{V}O_{2max}$), and mixed intensity (1 min at 70% $\dot{V}O_{2max}$) alternating with 3 min at 40% $\dot{V}O_{2max}$ for total of 30 min) and control treatment (no exercise); test meal approx 15 min postexercise	Brief suppression of subjective feeling of hunger; no effect on energy intake
Staten, 1991 (72)	Male and female N = 10 and N = 10	Lean	10 d of two treatments: 5 d of treadmill running at 70% $\dot{V}O_{2max}$ for 1 h·d^{-1} and 5-d rest period; preweighed food eaten in metabolic ward	Male subjects increased intake, female subjects intake unchanged; both in negative energy balance
Thompson et al., 1988 (77)	Male N = 16 age^M = 23.8	Lean	Two intensities of cycling: high (68% $\dot{V}O_{2max}$ for 29 min) and low (35% $\dot{V}O_{2max}$ for 58 min); control period of rest; *ad libitum* test meal 1 h postexercise	Brief suppression of hunger; no reduction in energy intake
Tremblay et al., 1994 (79)	Male N = 9 age^M = 28.3	Lean	Four treatments; 60 min of treadmill (55–60% $\dot{V}O_{2max}$) followed by low-fat diet, high-fat diet, and a mixed diet; one rest period followed by mixed diet	Negative energy balance with exercise treatments followed by low-fat and mixed diets; positive energy balance with exercise followed by high-fat diet; no effect of exercise on energy intake
Verger et al., 1992 (81)	Male N = 15 age^R = 19–25	Lean	Two treatments; 75 min of continuous swimming and 75 min of rest; *ad libitum* test meal 0, 30, 60, and 90 min postexercise	Increased hunger and energy intake 1 h after exercise compared with rest
Verger et al., 1992 (80)	Male and female N = 8 and N = 5 age^R = 20–25	Lean	Two treatments; 2 h of nonstop athletic activity and 2 h of rest; *ad libitum* test meal 0, 30, 60, and 120 min postexercise	Increased hunger and energy intake 1 h after exercise compared with rest
Verger et al., 1994 (82)	Male N = 28	Lean	Two treatments; 2 h of variety of continuous athletic activities and 2-h rest period; *ad libitum* test meal 2 h postexercise	Energy intake increased after exercise
Westerterp et al., 1997 (89)	Male N = 20 age^M = 25.0	Lean and obese	*Ad libitum* food intake measured immediately before and after 2 h of cycling or 2 h of sauna	Short-term suppression of hunger and energy intake; increase in carbohydrate intake after exercise and sauna

age^R, age range; age^M, mean age.

TABLE 3b. Medium-term intervention studies that have examined the effect of physical activity on appetite.

Author(s) and Year	Gender, Sample Size, and Age (yr)	Weight	Protocol	Results
Dickson-Parnell and Zeichner, 1985 (16)	Female $N = 33$ $age^M = 23.6$	Overweight	7 wk of monitoring 3 groups; control group (no exercise), high-intensity exercise group (cycling at 80% max heart rate, 3 times/wk), and low-intensity exercise group (cycling at 55% max heart rate, 3 times/wk); food intake measured using food diaries pre-, at wk 2, 4, and 6 of training	Reduced intake on exercise days compared with rest days for exercise groups
Epstein and Wing, 1978 (25)	Female $N = 17$	Lean	5 wk of aerobic exercise (jogging 2 miles·d^{-1} and 1 miles·d^{-1} for the last week); food intake measured using food diaries	No effect of exercise on energy intake
Keim et al., 1990 (42)	Female $N = 12$ $age^M = 30.0$	Obese	Three 18-d treatments of varying energy expenditures; sedentary, moderate exercise (increased daily expenditure by 12.5%), and long-duration exercise (increased daily expenditure by 25%); food intake measured by preweighing	No effect of exercise on energy intake
Woo et al., 1982 (90)	Female $N = 6$ $age^M = 42.6$	Obese	Three 19-d treatments of varying energy expenditures; sedentary, mild exercise (increase daily expenditure by 10%), and moderate exercise (increase daily expenditure by 25%); food intake measured by preweighing in metabolic ward	No effect of exercise on energy intake
Woo et al., 1982 (91)	Female $N = 3$ $age^M = 30.0$	Obese	57 d of treadmill walking at approx 3 mph for 110 $min·d^{-1}$ (to increase daily energy expenditure by approx 25%); food intake measured by preweighing in metabolic ward	No effect of exercise on energy intake
Woo and Pi-Sunyer, 1985 (92)	Female $N = 5$ $age^M = 37.0$	Lean	As Woo et al. (1990), except using lean female subjects	Compensatory increase in energy intake

age^R, age range; age^M, mean age.

between free-living active lifestyle and enforced sedation in the calorimeter). It should be kept in mind that these comparisons were not based on a within-subjects single experiment but were made on the basis of separate outcomes from two independent studies. Nevertheless, it seems safe to conclude that hunger and the pattern of eating are not closely related to perturbations in EE induced by large adjustments in physical activity. When subjects were obliged to become sedentary (in the calorimetric chamber), their eating habits apparently continued in their habitual form.

DOES PHYSICAL ACTIVITY ADJUST THE TUNING OF EI TO EE?

For individuals whose body weight is stable, EI must be matched to EE. Years ago, Mayer proposed that the mechanisms regulating energy balance would operate more accurately if EE was high (59,60). The idea arose from studies in rats and on Indian factory employees and indicated that the lower the activity level (greater the sedentariness), the greater was body weight or body mass index (BMI). This gave rise to the idea of a "threshold" level of physical activity, above which EI to EE matching was good, and below which it was poor. Recently, a retrospective analysis of doubly labeled water studies (69) provided partial support for this view (the threshold seemed to operate in men but not women). However, another explanation may be possible for these data. The greater an individual's level of EE, the more difficult it will be for EI to rise above this value (i.e., individuals will find it difficult to eat themselves into a positive energy balance). With a low EE, it will be relatively easy for EI to overtake this value (particularly with high fat, high energy dense foods), resulting in a positive energy balance and high BMI. This leaves open a more intriguing idea that physical activity may sensitize the physiological mechanisms involved in appetite control, thereby improving the precision with which EI could be tuned to EE.

These arguments, however, depend on the philosophy of energy regulation. One perspective is that organisms function so as to maintain energy balance and a stable body weight (sometimes called a set-point). Alternatively, the biological objective may operate so as to ensure that EI is maintained "at least" to the level of EE (so as to prevent a negative EB and life-threatening weight loss) and not to ensure a matching of EI and EE. Therefore, there may be no biological pressure to prevent a positive energy balance.

WHAT PROCESSES MEDIATE IN THE "ADJUSTMENT" TO A STEADY STATE?

Most of the intervention studies showed that an increase in food intake did not automatically follow an increase in physical activity. This means that the process of "adjustment" is not immediate and may take several months. More than half of the correlational studies (Table 2) indicate a positive relationship between physical activity and EI. From this, it can be deduced that at some time point a balance is achieved between physical activity and EI that represents a new steady state (see Fig. 1). This is clearly necessary because the body could not tolerate a permanent energy deficit or loss in body weight. Therefore, at

TABLE 3c. Long-term intervention studies that have examined the effect of physical activity on appetite.

Author(s) and Year	Gender, Sample Size, and Age (yr)	Weight	Protocol	Results
Andersson et al., 1991 (3)	Female and male N = 22 and N = 9 age^{M} = 36.0 and 37.0	Obese	3-months of supervised, mixed-physical training, three sessions/wk (60 min per session); food intake measured using pre-, mid-, and post-training diet history	No effect of exercise on energy intake
Broeder et al., 1992 (10)	Male N = 47 age^{R} = 18–35	Lean	12 wk of high-intensity endurance or resistance training (4 d·wk^{-1}); food intake measured using food diaries at wk 0, weeks 6–7, and posttreatment	Small decrease in energy intake
Dempsey, 1964 (15)	Male N = 7 age^{R} = 18–28	Obese and lean	18 consecutive weeks of training (8 wk) for 1 h·d^{-1}, no training (5 wk) and training (8 wk) for 1 h·d^{-1}; food intake measured using food diaries 3 wk before and every day during training weeks	No effect of exercise on energy intake
Holloszy et al., 1964 (33)	Male N = 15 age^{M} = 41.7	Overweight	6 months of supervised exercise (calisthenics and distance running approx 3 times/wk); food intake measured using pre- and post-training food diary records	No effect of exercise on energy intake
Janssen et al., 1989 (37)	Male and female N = 18 and N = 9 age^{M} = 35.8 and 34.6	Lean	Energy intake measured using food diary before and after 18 months of endurance training	Energy intake increased in male but not in female subjects
Johnson et al., 1972 (39)	Female N = 20 age^{M} = 21.4	Lean	10 wk of physical activity of 5 sessions/wk (30 min/session @ heart rate of approx 160 bpm); food intake measured using 3-d dietary records at pre-, mid-, and post-training	Significant decrease in energy intake between pre- and post-training
Leon et al., 1979 (55)	Male N = 10 age^{M} = 25.0	Overweight	16 wk of treadmill walking at 3.2 mph @ 10% grade for 5 sessions/wk (sessions varied from 15–90 min); food intake measured using 3-d food diary records pre-, 4, 8, 12, and 16 wk of training	Decrease in energy intake
Tagliaferro et al., 1986 (76)	Female N = 10 age^{M} = 25.6	Lean	10 wk of running at 70–80% max heart rate for 2 h·wk^{-1} divided into 3–5 sessions/wk; food intake measured using food diaries pre- and post-training	Slight decrease in energy intake
Ludo et al., 1997 (57)	Male N = 18 exercisers age^{M} = 33.0 N = 12 controls age^{M} = 35.0	Lean	18 wk of weight-training program; food intake measured using food diaries at weeks 0, 8, and 18	No effect of exercise on energy intake
Watt et al., 1976 (83)	Male N = 30 age^{M} = 49.2	No info	12 wk of supervised variety of exercises, for 45 min/session, 3 sessions/wk; food intake measured using 3-d food diaries pre- and post-training	Slight decrease in energy intake
Westerterp et al., 1992 (87)	Male and female N = 16 and N = 16 age^{R} = 28–41	Lean and overweight	44 wk of marathon training (4 sessions/wk); food intake measured using food diaries at weeks 0, 8, 20, and 40	Slight decrease in energy intake in male and slight increase in energy intake in female subjects
Wood et al., 1983 (93)	Male N = 48 exercisers age^{M} = 45.3 N = 33 controls age^{M} = 46.2	Lean	12 months of training (3–5 times/wk @ 70–85% of max); food intake measured using food diaries	No effect of exercise on energy intake
Wood et al., 1988 (94)	Male N = 47 exercisers age^{M} = 44.1 N = 42 controls age^{M} = 45.2	Overweight	12 months of endurance training; food intake measured using food diaries	Energy intake decreased in months 7 and 12

age^{R}, age range; age^{M}, mean age.

TABLE 4. Correlational studies in which measures have been made of physical activity and energy intake between different groups of subjects.

Author(s) and Year	Gender, Sample Size, and Age (yr)	Weight	Protocol	Results
Almeras et al., 1991 (1)	Male N = 7 skiers age^M = 20.9 N = 8 controls age^M = 22.3	Lean	Daily energy expenditure (HR-factorial method) and energy intake (food diaries) measured in a group of cross-country skiers and a sedentary control group	Energy intake greater in cross-country skiers compared with sedentary controls; energy intake similar to expenditure within both groups
Beidleman et al., 1995 (4)	Female N = 10 runners age^M = 21.5 N = 10 controls age^M = 24.1	Lean	Energy expenditure (HR factorial) and energy intake (food diaries) measured for 3 consecutive days in a group of runners and sedentary controls	Energy intake lower than energy expenditure in both runners and sedentary controls; no difference in energy intake between two groups despite differing levels of energy expenditure
Blair et al., 1981 (5)	Male and female N = 34 and 27 age^R = 35–59	Lean	Energy intake (3-d food diaries) in a group of runners compared with sedentary controls	Lean runners had higher energy intakes than heavier controls
Boulay et al., 1994 (8)	Male N = 7 skiers age^M = 21 N = 8 controls age^M = 22	Lean	Identical study to above; in this study, two methods of measuring energy expenditure (HR vs diary) were compared	Energy intake greater in cross-country skiers compared with sedentary controls; energy expenditure similar to energy intake in controls but depended on the method used in skiers
Broeder et al., 1992 (9)	Male N = 21 - low fit) age^M = 26.4 N = 38 - mod fit age^M = 22.8 N = 10 - high fit age^M = 25.4	Lean and overweight	Sample divided into 3 groups depending on fitness level as assessed by $\dot{V}O_{2max}$ (high, moderate, and low)	High fit group greater intake than moderate and low fit groups
Butterworth et al., 1994 (11)	Female N = 34 age^M = 29.0	Lean	Physical activity (Caltrac) and energy intake (food diaries) measured for 1 d·wk^{-1} for 10 wk in a group of individuals varying in activity level	Higher energy intakes in physically active
George et al., 1989 (29)	Male and female N = 109 and 118 age^R = 17–54	Lean and overweight	Physical activity (activity diaries) and energy intake (food diaries) measured for 3 consecutive days	Energy intake not matched with activity levels
Maughan et al., 1989 (58)	Male and female	Lean and overweight	Physical activity (weekly running distance) and energy intake (food diaries) measured in a group of individuals varying in activity level	Positive relationship between energy intake and weekly running distance
Mayer et al., 1956 (60)	Male N = 213	Lean and overweight	Individuals categorized into physical activity groups by job type; food intake measured by dietary interviews	U-shaped relationship between activity group and energy intake; sedentary individuals had greater energy intakes than those engaged in "light" work but similar to those engaged in medium to heavy work
Stubbs et al., 1995 (74, 75)	Male N = 6 - study a age^M = 41.8 N = 7 - study b age^M = 36.9	Lean	Two studies using different volunteers (74 and 75); *ad libitum* food intake was measured in a whole body calorimeter (74) in which energy expenditure was inherently lowered and in the free-living (75) in which energy expenditure was higher than in (74)	Energy intake matched energy expenditure in free living (75) study, but not in calorimeter (74) study; no downregulation of energy intake with lower energy expenditure
Tremblay et al., 1983 (78)	Male and female N = 821 and 772	Lean and overweight	Energy intake (3-d food diaries) and physical activity (activity diaries) measured in a group of individuals divided into low, medium, and high activity	Linear relationship between activity level and energy intake in male but not in female subjects

age^R, age range; age^M, mean age.

TABLE 5. Correlational studies in which measures have been made of physical activity and energy intake within the same group of subjects.

Author(s) and Year	Gender, Sample Size, and Age (yr)	Weight	Protocol	Results
Cole and Ogbe, 1987 (12)	Male $N = 20$ age$^M = 24.0$	Lean and overweight	Energy expenditure (activity diary) and energy intake food diaries) measured for 7 consecutive days	Energy expenditure was lower than energy intake
Durnin et al., 1957 (18)	Female $N = 12$	Lean and overweight	Energy intake (food diaries) and energy expenditure (indirect calorimeter) measured for 7 consecutive days	No coupling between energy expenditure and energy intake; only 8% of subjects showed a significant positive correlation between daily energy intake and energy expenditure
Durnin and Brockway, 1959 (19)	Male $N = 4$	Lean and overweight	Energy intake (food diaries) and energy expenditure measured for 7 consecutive days	No coupling between energy expenditure and energy intake; no subjects showed a significant positive correlation between daily energy intake and energy expenditure
Edholm et al., 1955 (22)	Male $N = 12$ age$^M = 19.5$	Lean	Energy intake and expenditure monitored during 2 wk of army training; cafeteria-style food provided, which was weighed before and after eating	Energy intake and energy expenditure matched over 1 wk but not daily; correlation between expenditure on one day and intake 2 d later
Edholm et al., 1970 (23)	Male $N = 34$ age$^M = 19.2$	Lean	Identical to Edholm et al., 1955 (21) except for 3 wk	Energy intake and energy expenditure matched over 7 d but not daily
Edwards et al., 1993 (24)	Female $N = 9$	Lean	Energy expenditure (doubly labelled water) and energy intake (food diaries) measured for 7 consecutive days	Negative relationship between energy expenditure and energy intake
Fariduddin et al., 1975 (26)	Male and female $N = 39$	Lean	Energy expenditure (indirect calorimetry) and energy intake (weighed intakes) measured for 3–4 d	Coupling between energy expenditure and energy intake
Garry et al., 1955 (28)	Male $N = 19$	Lean	Energy intake (food diaries) and energy expenditure (indirect calorimeter) measured for 7 consecutive days	No coupling between energy expenditure and energy intake; only 10% of subjects showed a positive correlation between daily energy intake and energy expenditure
Katch et al., 1969 (40)	Female $N = 15$	Lean	16 wk of swimming and tennis; food intake (food diaries) measured pre- and post-training	No change in energy intake
Milon et al., 1996 (62)	Male $N = 1$	Lean	Energy intake measured during 55 d of swimming the Atlantic	Energy intake significantly lower than energy expenditure
Parizkova and Poupa, 1963 (64)	Female $N = 7$ age$^M = 23.0$	Lean	Energy intake (food diary) measured during varying levels of training periods	Energy intake increased in response to higher levels of training and decreased during lower levels of training
Saris et al., 1989 (68)	Male $N = 5$	Lean	Energy intake (food diaries) and energy expenditure (estimated) were monitored for 3 wk	Energy intake matched energy expenditure
Sjodin et al., 1994 (70)	Male and female $N = 4$ and $N = 4$ age$^M = 26.0$ and 25.0	Lean	Energy expenditure (doubly labelled water) and energy intake (food diaries) were measured for 7 d of training in cross-country skiers	Close match between energy expenditure and energy intake in both male and female subjects
Smoak et al., 1988 (71)	Male $N = 16$	Lean	Energy expenditure (factorial method) and energy intake (observed) were monitored before and during 113 h of intense physical activity	Energy intake increased in response to the increase in energy expenditure
Westerterp et al., 1986 (85)	Male $N = 5$	Lean	Energy expenditure (doubly labeled water) and energy intake (food diaries) were measured for 3 separate 7-d (consecutive) intervals	Energy intake did not match energy expenditure during all 3 phases; energy expenditure was always higher than energy intake
Worme et al., 1991 (95)	Male $N = 8$ age$^M = 30.0$	Lean	Energy intake (food diaries) and energy expenditure (estimated using Harris-Benedict equation and physical activity multiple factors) measured before, during, and after 31 d of training at altitude	Energy intake increased in response to increased physical activity

age^R, age range; age^M, mean age.

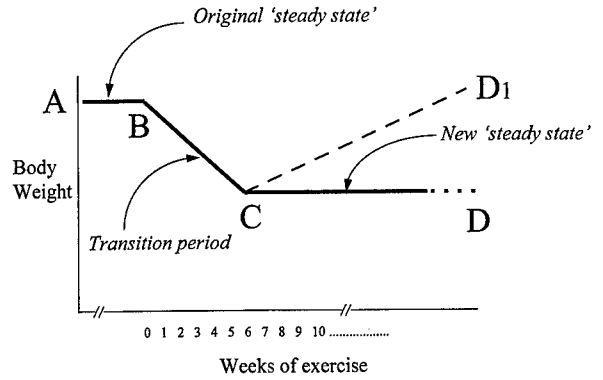

Figure 1—Schematic diagram to illustrate the proposed impact of exercise on body weight. A–B indicates an initial steady state; point B represents the commencement of exercise; B–C indicates the transition stage when a higher EE is not compensated by EI; C represents the time at which new forces (behavioral and metabolic) interact to induce a new steady state (C–D).

some stage during the period of "adjustment" some mechanism(s) must "kick in" in order to prevent a continuing energy deficit and further weight loss. What behavioral and/or physiological mechanisms could be responsible for such compensatory processes? At what stage (and under what conditions) do such compensatory mechanisms occur? During the imbalance between EE and EI, there are several physiological consequences that occur in response to a loss in body weight. This period of imbalance or adjustment has been labeled as the "transition period" (32). One of the major physiological consequences of weight loss is a compensatory reduction in resting metabolic rate (27,52,53). There is also an increase in $\dot{V}O_{2max}$ in response to exercise (41,54) that would have a subsequent effect of reducing the exercise-induced EE, especially if the volume (intensity/duration) of exercise remained unchanged. Another effect of the reduction in body weight would be to reduce the net exercise-induced EE (84). However, taking these physiological changes collectively, it is unlikely that they could be solely responsible for achieving a new coupling between EE and EI (steady state), i.e., a constant body weight. It is more likely that behavioral process are also involved (i.e., a combination of physiology and behavior). However, it has been suggested that some individuals may become "energy efficient" in response to increases in the volume (intensity/duration) of exercise (86), producing a discrepancy between the predicted and actual weight loss responses. Therefore, it is possible that the physiological processes (i.e., reduced resting metabolic rate and reduced exercise-induced EE) have the combined effect of gradually reducing the total EE (hence, the differential between EE and EI) until a steady state (balance between EE and EI) is reached.

Considering the profile in Figure 1, we can envisage a transition (adjustment) from an original steady state (A–B) to a new steady state (C–D). A number of changes may have occurred to allow C–D to develop. For example, there may have been a loss of 100% compliance with the exercise regime, physical activity in the nonexercise time may have been reduced (recovery periods), and EI may have been permitted to increase (allowance of food rewards and mistaken self-indulgence). These behavioral adaptations would be superimposed upon any metabolic adjustment (probably resulting in energy saving).

Therefore, because exercise cannot continue to reduce weight indefinitely—and the emergence of a new steady state must occur—one important feature in the use of exercise must be to delay the arrival of point C. It remains to be demonstrated which are the most potent factors that reduce the effectiveness of exercise. These may vary from individual to individual. It follows, of course, that if exercise is abandoned at point C the weight would be restored to point D1 (or possibly above this).

SUMMARY

The evidence reviewed indicates that engaging in physical activity does not automatically generate an increase in the drive to eat that would compensate for the energy expended. Indeed, after exercise, food intake most commonly remains unchanged and, in certain circumstances, physical activity can suppress appetite. However, when physical activity is reduced (when an individual becomes more sedentary), food intake is not down-regulated; this leads to a positive energy balance. All of these findings suggest a rather loose (not tight) coupling between activity-induced EE and EI. When activity level changes and an individual moves from one steady state of energy balance to another (at a different body weight), the mechanisms responsible for this adjustment have yet to be defined. There is some evidence that the processes mediating in the relationship between activity and food intake may operate differently in men and women. Raising EE through physical activity should make a significant contribution to the prevention of obesity, and to weight loss for those already overweight. There is no strong biological imperative to match EI to activity-induced EE. However, social, environmental, and cognitive forces may all intervene to alter the relationship between physical activity and food intake.

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Physical activity and preference for selected macronutrients

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ABSTRACT

TREMBLAY, A., and V. DRAPEAU. Physical activity and preference for selected macronutrients. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S584–S589, 1999. **Objective:** The impact of physical exercise on macronutrient preferences was examined with a perspective to improve preventive and therapeutic strategies of obesity. **Design:** The literature was reviewed pertaining to the acute effects of physical activity and the short-term and chronic effects of exercise training on macronutrient preferences. **Results:** The presently available literature does not permit to establish a consensus regarding the impact of physical activity, be it acute or long-term, on macronutrient selection. However, one observation stands out and that is the fact that dietary fat intake needs to be controlled in order for exercise to produce a negative energy and fat balance. **Conclusion:** Because active individuals do not systematically choose foods that are low in fat content, it is important to provide nutritional guidelines in a context where physical activity aims at reducing or better controlling body weight. **Key Words:** ENERGY, FAT, EXERCISE, OBESITY, LIPID

The lack of physical activity in an industrialized world is a factor that likely explains a significant portion of the increased risk to develop obesity among sedentary individuals. Theoretically, the corollary of this statement implies that weight-reducing programs based on the regular practice of aerobic physical activities should favor a substantial body weight and fat loss. However, clinical experience and experimental data reveal that the use of exercise alone to induce long-term negative energy balance does not systematically produce the expected outcome. This phenomenon may be explained by the fact that the energy cost of additional exercise is sometimes not sufficient to produce a significant change in daily energy balance. The failure of regular activity participation to induce weight loss in some obese individuals might be also partly attributable to a compensation in postexercise energy expenditure and intake. Specifically, this means that a decrease in nonexercise physical activity participation and an increase in postexercise energy intake can compensate substantially or even totally for the surplus of energy expended during physical activities.

In a recent series of studies, diet composition has been shown to exert a significant influence on postexercise energy intake. In the first study to document this issue, the intake of a diet conforming to dietary guidelines for fat intake prevented the compensation in postexercise energy intake to overcome the energy cost of exercise (23). Conversely, a high-fat diet was associated with a large overfeeding that was sufficient to totally compensate for the impact of exercise on energy expenditure. In subsequent studies, the physical activity-low fat diet combination was also found to permit a substantial acute negative energy balance (5,14). Taken together, these observations suggest that the activity-low fat diet combination can induce an acute daily energy deficit that can be as large as 1000 $kcal \cdot d^{-1}$ (4.2 $MJ \cdot d^{-1}$), but they also draw attention on the possibility that activity practice may result in an overall positive energy balance if diet has a high fat content.

The effect of physical activity and diet composition on energy balance emphasizes the importance to make appropriate food choices if an active individual wishes to lose or maintain body weight. This rationale also raises the question as to whether exercise *per se* can alter food choices and/or macronutrient selection. The main aim of this paper is to document this issue with a perspective to improve preventive and therapeutic strategies of obesity.

WHAT IS A MACRONUTRIENT PREFERENCE?

Evidence statement. There is a need toward the development of standardized measurements of macronutrient preferences (evidence category B).

The concept of macronutrient preference is not systematically considered by nutrition agencies. In practical terms, a macronutrient preference likely reflects a preferential taste for foods having a high content of a specific macronutrient. There is no well-accepted specific measurement for this phenotype, but it can be estimated with different indirect measurements. One of them is the habitual macronutrient content of the diet, be it assessed by dietary record, recall, or history. According to this procedure, a high percentage of

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ingested calories from a given macronutrient is assumed to reflect an increased preference for this macronutrient.

A second strategy to assess macronutrient preference consists of using a buffet test meal that offers a variety of foods of different macronutrient composition. Such a test is generally performed in the laboratory under conditions mimicking as closely as possible free living conditions. In this context, it is postulated that a preference for a specific macronutrient will be reflected by a preferential selection of foods having a high content of this macronutrient.

Macronutrient preference can also be evaluated by the search of a macronutrient mix providing the greatest appreciation by a subject. An example of this experimental strategy was used by Drewnowski and Greenwood (6), who tested different carbohydrate-lipid solutions to determine whether human obesity is associated with a specific macronutrient preference. Their results revealed a greater preference for lipid in obese and postobese subjects than in their lean counterparts.

Up to now, there is no consensus on how to specifically measure macronutrient preference, and this phenotype is thus measured with different strategies that do not necessarily reflect the same phenomenon across available studies. Therefore, if macronutrient preference is proven to be a key issue in the study of energy balance, it will be relevant to pursue investigations in this area to establish an operational definition of this concept.

ACUTE EFFECT OF PHYSICAL ACTIVITY ON MACRONUTRIENT PREFERENCE

Evidence statement. The acute effect of physical activity on macronutrient preference is not clearly established (evidence category B).

Prolonged vigorous physical activities are known to induce glycogen depletion, which may represent an effect promoting a specific preference for carbohydrate to facilitate glycogen store replenishment (14). This hypothesis is partly documented by studies in which the acute effect of exercise on macronutrient preference has been tested. As shown in Table 1, animal experimentation does not systematically support this idea.

This table also summarizes human studies that have specifically focused on postexercise macronutrient preference. In their first study pertaining to this issue, Verger et al. (24) found an increased preference for foods with high carbohydrate content after a prolonged exercise. However, this finding could not be reproduced in a subsequent study (25). Indeed, by using a comparable exercise test, they observed an increase in relative protein intake. In a more recent study, Westerterp-Platenga et al. (26) noted an increased preference for carbohydrate and a decreased preference for fat after exercise. In addition, these authors criticized the experimental approach of Verger et al. for the reduced availability of foods containing predominantly one macronutrient in their buffet-type meal. Accordingly, Horio and Kawamura also found a preference for sucrose after exercise in university students (9).

In our laboratory, we examined the impact of exercise sessions of different intensity but of similar energy cost on the composition of postexercise *ad libitum* intake from a buffet-type meal (10). The results showed no effect of exercise on postexercise food quotient (FQ). At best, we found a nonsignificant positive correlation between exercise RQ and postexercise FQ.

In summary, the available literature pertaining to the acute effect of physical activity on macronutrient preference does not permit to identify a clear effect of activity. In addition, available data also do not allow to establish a relationship between the composition of the substrate mix oxidized during exercise and spontaneous food selection in the postexercise state.

SHORT-TERM EXERCISE-TRAINING AND MACRONUTRIENT PREFERENCE

Evidence statement. There is no clear effect of short-term exercise-training on macronutrient preference (evidence category B).

A summary of the main studies documenting the impact of short-term exercise-training on macronutrient preference in animals and humans is presented in Table 2. A diversity of effects is reported in animals, ranging from an enhancing effect of training on spontaneous carbohydrate and protein intake or either the opposite or just a significant decrease in fat intake. Interestingly, *ad libitum* energy intake was found to fluctuate according to changes in macronutrient intake. An accentuation of the preference for carbohydrate and/or a decrease in spontaneous fat intake were associated with no compensation or a decrease in energy intake.

Table 2 also shows that data collected in humans are less conclusive. Indeed, the majority of available data tend to show that short-term training has no effect on both macronutrient selection and energy intake. This table also presents recent data demonstrating that short-term training can increase preference for fat in female subjects only (1). In summary, as for data related to the acute effect of aerobic activities, the currently available literature does not permit to identify a systematic effect of the regular practice of physical activity over several weeks on macronutrient selection.

CHRONIC TRAINING AND MACRONUTRIENT SELECTION

Evidence statement. There is no specific effect of chronic training on macronutrient preference. However, chronic training increases daily energy intake in trained individuals (evidence category B).

Theoretically, the long-term exposure to physical activity represents the condition that is expected to exert the greatest impact on macronutrient selection. Indeed, the feeding behavior of the regular exerciser is not only affected by the exercise stimulus but also by changes in body fatness that may influence macronutrient preference.

Table 3 summarizes human studies that have addressed this issue. Two of these studies were longitudinal interven-

TABLE 1. Acute effect of exercise on food selection and energy intake.

Study	*N*, Gender	Physical Activity	Impact of Physical Activity on	
			Food selection	Energy intake
In experimental animals				
Larue-Achagiotis et al. (15)	11 male adult Wistar rats	Treadmill exercises: 15m·min^{-1} for 120 min No exercise	↓ PROT (%) intake after exercise, disappears when food intake is delayed ↓ CHO (%) after 90 min delay ↓ FAT (%) after exercise and seems to persist over 24h	↓ 24-h intake with exercise
In human subjects				
Verger et al. (24)	13 subjects (5 female, 8 male)	Various athletic activities at 70–80% of maximal HR for 2 h	↑ CHO (%) 60 and 120 min after exercise ↓ PROT (%) with time delay	↑ 500 kcal 60 min after exercise
Verger et al. (25)	58 young men: experimental; N=28; control, *N*=30	Cross-country (running and jumping) 400 kcal·h^{-1} for 2h	↑ PROT (%)	↑ intake (≈25%) (< energy cost of exercise)
King et al. (13)	Study 1: 12 male adults	High-intensity exercise: cycling at 72% $\dot{V}O_{2max}$ for 27 min Low intensity exercise: cycling at 36% $\dot{V}O_{2max}$ for 63 min No exercise	No difference between the three conditions	No difference in absolute or relative energy intake between the three conditions at test meal or whole day
	Study 2: 12 male adults	Short duration exercise (26 min): cycling at 77% $\dot{V}O_{2max}$ Long duration exercise (52 min): cycling at 74% $\dot{V}O_{2max}$ No exercise	? (no indication)	No difference in absolute energy intake between the three conditions ↓ relative energy intake at test meal and whole day for long duration exercise compared with short duration and no exercise
Westerterp-Platenga et al. (26)	30 subjects: obese, *N*=10; nonobese, *N*=10 (cycling vs rest); nonobese, *N*=10 (sauna vs rest)	Cycling at 60% $\dot{V}O_{2max}$ for 2 h	No difference between obese and nonobese ↑ CHO (%), ↓ FAT (%), ↑ perception of sweet after exercise and sauna compared with rest condition	↓ intake after exercise only compared with rest condition
Imbeault et al. (10)	11 male adults	Low-intensity exercise (Liex): walking at 35% $\dot{V}O_{2max}$ for 2054 kJ High-intensity exercise (Hiex): running at 72% $\dot{V}O_{2max}$ for 2022 kJ No exercise	No difference between the three conditions	↓ in intake adjusted for energy cost of exercise (relative energy intake) after Hiex
Horio and Kawamura (9)	58 university students	Cycling at 50% $\dot{V}O_{2max}$ for 30 min	↑ preferences for sucrose and citric acid	—

TABLE 2. Effect of short-term exercise training on food selection and energy intake.

Study	*N*, Gender	Physical Activity	Impact of Physical Activity on	
			Food selection	Energy intake
In experimental animals				
Parizkova and Stankova (19)	22 male rats: experimental, *N*=11; control, *N*=11	Treadmill 8 m·min^{-1} 50 min·d^{-1} for 200d	↑ CHO (%)	No difference (kcal or g)
Gerado-Gettens et al. (8)	72 Sprague Dawley female rats: control, *N*=24; weight-cycled sedentary rats, *N*=24; weight-cycled exercise rats, *N*=24	Treadmill 20 m·min^{-1}min 60 min·d^{-1} 6 d·wk^{-1} for 16 wk	↑ PROT (%) after 1 cycle in weight-cycled exercise compared with weight-cyclef sedentary ↑ CHO (%) after second refeeding period in weight-cycled exercise compared with weight-cycled sedentary	No difference between weight-cycled sedentary and exercise
Miller et al. (17)	39 Sprague Dawley female rats: experimental, *N*=18; control, *N*=21	Treadmill 20 m·min^{-1} (mild to moderate intensity) 60 min·d^{-1} 6d·wk^{-1} for 15 wk	↓ FAT (%)	No difference between groups
Larue-Achagiotis et al. (16)	15 Wistar adult rats: control, *N*=5; sedentary, *N*=10; exercise, *N*=10	20 m·min^{-1} 2 h·d^{-1} for 20 d	↓ FAT (%) in exercise group compared with their baseline values	↓ in caloric intake in exercise group compared to their baseline values
Oudot et al. (18)	18 Female rats: control, *N*=6; sedentary, *N*=6; exercise, *N*=6	16 m·min^{-1} 3 h·d^{-1} for about 11 wk	↑ PROT (%) with forced activity in exercise group ↑ CHO (%) and ↓ FAT (%) in exercise group when compared to controls	No difference
Even et al. (7)	24 male Wistar rats: sedentary, *N*=15; exercise, *N*=9	20 m·min^{-1} 2 h·d^{-1} for 3 wk	↑ PROT (%) ↓ CHO (%)	No difference
In human subjects				
Keim et al. (12)	12 moderately overweight women (16–42% above desirable BW)	Treadmill walking session 65–80% $\dot{V}O_{2max}$ Moderate-duration exercise (M-ex: 31–49 min) Long-duration exercise (L-ex: 51–88 min) 7 d·wk^{-1} for 18 d No exercise	↓ PROT (%) slightly in L-ex and M-ex groups compared with no-ex No difference between M-ex or L-ex	No difference between exercise and no-exercise No difference between M-ex or L-ex
Butterworth et al. (3)	30 elderly women: experimental, *N*=14; Control, *N*=16	Walking 60% of HR reserve (moderate exercise program) 30–40 min/session 5 d·wk^{-1} for 12 wk	No difference	No difference
Ambler et al. (1)	32 females: control, *N* = 15; exercise; *N* = 17 39 males: control, *N* = 19; exercise, *N* = 20 adolescents	Training program with endurance activities 2h·d^{-1} for 4–5 wk	↑ FAT (%) and ↓ CHO (%) after exercise training in females only	No difference

TABLE 3. Effect of chronic training on food selection and energy intake in humans.

Study	*N*, Gender	Physical Activity	Impact of Physical Activity on		Additional Information
			Food selection	Energy intake	
Katch et al. (11)	Female swimmers During training Out of training	Swimming	No difference	—	Longitudinal study
de Wijn et al. (4)	Male rowers (*N*=8) During training Out of training	Rowing	No difference	↑ intake (+951 kcal·d^{-1})	Longitudinal study
Blair et al. (2)	61 long-distance runners (34 male, 27 female); 80 controls (38 male, 42 female)	Running at least 24 km·wk^{-1} for the last past year: male, 65 mk·wk^{-1}; female, 55 km·wk^{-1}	↓ PROT (%)	↑ intake (40–60% higher on weight adjusted basis)	Dietary intake: 3-d food records
Smith et al. (21)	34 females: competitive swim team (CS), *N*=9; synchronized swim team (SS), *N*=15; control, *N*=10	CS group: swam 6 d·wk^{-1}, 8000 yards·d^{-1} in 2 h SS group: swam 3d·wk^{-1}, 1500 yards·d^{-1} in 2 h	No difference between the 3 groups	↑ intake as the level of physical activity increase (+438 kcal/d)	Dietary intake: 4-d food records 4 times
Thompson et al. (22)	Male endurance runners, *N*=20; control, *N*=14	Running average 113 km·wk^{-1}	No difference	↑ intake (+585 kcal·d^{-1}) from CHO	Dietary intake: 10-d food records
Reggiani et al. (20)	Female athletes, *N*=21; control; *N*=21	Variety of athletic activities, 2 h·d^{-1}, 5 d·wk^{-1}	↑ FAT (%) ↓ CHO (%)	↑ intake (+413 kcal·d^{-1}) from FAT	Dietary intake: 7-d food records
Butterworth et al. (3)	Elderly women: highly conditioned group, *N*=12; sedentary group, *N*=30	Exercised aerobically for more than 1 h·d^{-1} for at least the past 5 yr, $\dot{V}O_{2max}$ > 26 mL·kg^{-1}·min^{-1}	No difference in diet composition	↑ intake/kg	Dietary intake: 7-d food records

tions. In these two studies, neither a swimming program (11) or a rowing training program (4) had significant effect on habitual diet composition.

Table 3 also presents data obtained in cross-sectional studies comparing habitual macronutrient intake in trained individuals and sedentary controls. Again, no systematic pattern of food selection can be detected from this set of data despite the fact that these individuals are frequently exposed to dietary information promoting an increase in carbohydrate intake. It is, however, of interest to note that in each case, reported daily energy intake was increased in trained individuals.

SUMMARY AND PERSPECTIVE FOR OBESITY PREVENTION AND TREATMENT

Evidence statement. The practice of physical activity is not associated with a preference for specific macronutrients in active individuals. Consequently, fat intake needs to be controlled in active individuals to favor a negative energy balance (evidence category B).

The analysis of studies documenting habitual macronutrient intake in relation to the practice of physical activities does not permit to clearly identify particularities in the feeding behavior of active individuals. Indeed, available data suggest that the exposure to an exercise stimulus may favor a preferential consumption of foods having a high content of either carbohydrate, fat, protein, or alcohol. In other words, physical activity seems to have the potential to induce about any change in macronutrient intake, depending on circumstances that are not well characterized and understood. In fact, the study of macronutrient preferences will remain a difficult issue of investigation as long as the following points will not be clarified: 1) the concept of macronutrient preference deserves a better characterization and should be measured using standardized methodology, and 2) physical activity-related factors and underlying biological mechanisms influencing macronutrient preferences should be characterized. In addition, the study of macronutrient preference under free living conditions will remain difficult because of factors such as the large availability of foods containing a high amount of more than one macronutrient, the supplementation of many foods with artificial compounds that mimic the taste or texture of some macronutrients, and health campaigns toward healthier food habits.

This analysis of relevant literature emphasizes at least one implication regarding the prevention and treatment of obesity that pertains to the relevance of reducing dietary fat intake in the context of a physical activity program to favor a spontaneous energy deficit. In that respect, the literature described above suggests that the ability of physical activity *per se* to spontaneously alter macronutrient selection in the obese is uncertain. This thus implies that dietary advice is likely necessary to guide obese individuals toward good food selection instead of only relying on the impact of physical activity to induce such changes.

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Physical activity and the progressive change in body composition with aging: current evidence and research issues

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ABSTRACT

TOTH, M. J., T. BECKETT, and E. T. POEHLMAN. Physical activity and the progressive change in body composition with aging: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S590–S596, 1999. **Purpose:** The purpose was to review studies that have examined the effect of aerobic (AEX) or resistance exercise (REX) on body composition in older individuals (>55 yr). Our goal was to examine the effect of these two exercise paradigms on fat mass and fat-free mass and to consider those factors that may explain variability in findings among studies. **Methods:** We conducted a literature search (*Medline,* 1984–1999) for intervention studies (at least 2 months in duration) that have examined the independent effect of either REX or AEX on body composition in older individuals. **Results:** AEX decreased fat mass (range: −0.4 to −3.2 kg) but had little effect on fat-free mass. The change in fat mass with AEX was related to the duration of the exercise program ($r = 0.51$; $P < 0.02$) but not to body composition methodology. In contrast, REX reduced fat mass (range: −0.9 to −2.7 kg) and increased fat-free mass (range: 1.1 to 2.1 kg). Changes in body composition with REX were not related to body composition methodology or the duration of the exercise program. **Conclusion:** Both AEX and REX appear to be beneficial in reducing body fat. REX appears to have the additional benefit of increasing fat-free mass.
Key Words: RESISTANCE EXERCISE, AEROBIC EXERCISE, BODY COMPOSITION, ELDERLY

In recent years, a significant number of studies have examined the effect of physical activity on body composition in older individuals. The high level of interest in this topic is partly mediated by the recognition of: 1) the reported age-related changes in physical activity, body composition, and their impact on health risk and 2) the potential role that increasing physical activity may have on improving cardiovascular and metabolic health in older individuals. This area of investigation, coupled with significant improvements in methods to measure energy expenditure (i.e., doubly labeled water) and body composition, have broadened our understanding of the relationship between physical activity and body composition.

Physical activity declines with age. The decline in physical activity and associated reduction in daily energy expenditure are thought to be important mediators of deleterious changes in body composition. Concomitant with the decline in physical activity, a loss of fat-free mass (specifically, skeletal muscle mass) and increase in fat mass is frequently observed. These age-related changes in body composition contribute to increased disease risk and reduced functional independence in the elderly. It is important to note, however, that these conclusions are derived primarily from cross-sectional observations. Thus, the "true" rate of age-related changes in body composition remains enigmatic. Moreover, given the paucity of longitudinal studies that have measured physical activity and body composition, it is currently unclear which of these events is primary and which is secondary.

Despite the absence of causal evidence that age-related changes in body composition are the result of a decline in physical activity, investigators have begun to examine the effects of physical activity on body composition using exercise training experiments. In this review we examine exercise intervention studies that have focused on older men and women. We considered the independent effects of aerobic (AEX) and resistance exercise (REX) training on body composition. The rationale for separating these two exercise paradigms is that their effects on body composition may differ: AEX primarily reduces body fat by promoting negative energy imbalance, whereas resistance exercise primarily increases fat-free mass by stimulating skeletal muscle growth. To review the current literature, a search was conducted using the *Medline* medical data base from 1984 to 1999 to locate studies (English language) that have examined the effect of either REX or AEX on total body composition. After locating articles through *Medline*, reference lists were searched for other studies not identified by the computer. Criteria for selection were: 1) articles published between 1984 and 1999; 2) subjects aged >55 yr; 3) an intervention design (i.e., pre- and postexercise measure-

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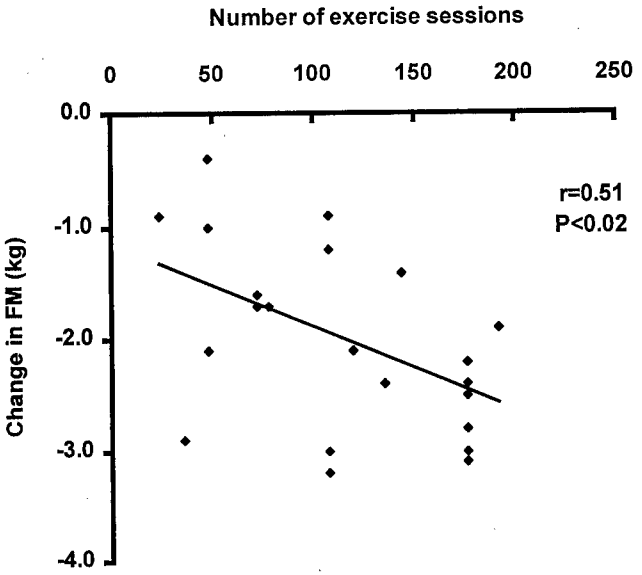

Figure 1—Relationship between changes in fat mass with AEX and total number of sessions in exercise program. Analysis was performed with those studies that observed a change in fat mass. Number of exercise sessions was calculated by multiplying the number of exercise sessions per week by the length of the program (weeks).

ments) of at least 2 months; 4) measurement of at least one component of body composition (i.e., either fat-free mass or fat mass) by either underwater weighing, dual energy x-ray absorptiometry, skinfolds, total body water, or *in vivo* neutron activation; and 5) no concomitant weight loss intervention (dietary restriction or pharmacological agent). Studies that examined the effect of combined AEX/REX on body composition were not included.

CURRENT STATUS OF KNOWLEDGE

The general conclusions drawn from this review of literature are that: 1) AEX is effective in reducing fat mass but has little effect on fat-free mass; 2) the magnitude of AEX-induced changes in fat mass are related to the total number of sessions in the exercise program; and 3) REX is effective in reducing fat mass and increasing fat-free mass.

Changes in Fat Mass with AEX

Evidence Statement: AEX is an effective intervention to decrease fat mass in older individuals. (Evidence Category C). AEX was effective in reducing body fat stores in 20 out of 22 studies that did not instruct patients to maintain their body weight. The effect of AEX to reduce body fat is not unexpected, although the mechanism by which this change occurs is unclear. AEX-induced changes in body fat were not related to the method used to assess body composition. However, the change in fat mass was related to the total number of sessions in the AEX program (Fig. 1). That is, as the number of exercise sessions increased, the quantity of fat lost increased. Thus, the length of exposure to the exercise stimulus or the absolute caloric expenditure of the exercise program may be an important determinant of the amount of weight lost during AEX in older individuals.

The long-standing theory is that AEX reduces body fat stores by promoting negative energy imbalance (i.e., energy expenditure exceeds energy intake). Specifically, AEX increases daily energy expenditure in older individuals through the direct energetic cost of the exercise and possibly by increasing resting energy expenditure. Recent findings, however, showed that 8 wk of high-intensity AEX did not increase daily energy expenditure in older men and women (15). Thus, the effect of AEX on body fat stores may be mediated by changes in energy intake rather than changes in energy expenditure. Further studies are needed to delineate the mechanism by which AEX promotes a reduction in adiposity. Understanding the mechanism of AEX-induced changes in body composition will allow the refinement of exercise prescriptions (i.e., intensity and duration) to optimize reductions in body fat.

Changes in Fat-Free Mass with AEX

Evidence Statement: AEX is not an effective intervention to increase fat-free mass in older individuals. (Evidence Category C). AEX does not appear to affect fat-free mass in older individuals. Only 8 out of 36 studies found an increase in fat-free mass, and this increase was less than 1 kg in most studies. Changes in the hydration of fat-free mass due to increased glycogen storage may account for these minimal changes in fat-free mass, although this has not been directly tested. The lack of effect of AEX on fat-free mass is not surprising, however, considering that this mode of exercise does not provide a significant anabolic stimulus to promote muscle growth. These results should not, however, be taken as evidence that AEX has no influence on fat-free mass. The possibility should be considered that AEX training may be effective in attenuating the age-related reduction in fat-free mass that results from inactivity and other hormonal and lifestyle factors. That is, AEX may provide an adequate stimulus to maintain fat-free mass with age. This hypothesis, however, has yet to be systematically tested.

Changes in Fat Mass with REX

Evidence Statement: REX is an effective intervention to decrease fat mass in older individuals. (Evidence Category C). REX reduced fat mass in 15 out of 28 studies. On average, in the studies that found a reduction in fat mass with REX, the loss of fat mass was similar to those changes induced by AEX (REX: -1.7 ± 0.4 kg vs AEX: -1.9 ± 0.8 kg). This is somewhat surprising considering that the direct energetic cost of resistance exercise is smaller compared with that of AEX. Although REX has been shown to increase resting energy expenditure (7,43), similar changes have been found with AEX (39). Because no study has examined the effect of REX on free-living, daily energy expenditure, the effect of REX on energy balance remains unclear. Studies that examine changes in daily energy expenditure and its components

TABLE 1. Changes in body composition with aerobic exercise in older individuals.

Study	Exercise Program		Subjects		Random/ Nonrandom	Method	Change FFM (kg)	Change FM (kg)	Change Wt (kg)
	Frequency (times/wk)	Duration (months)	Number/Sex	Age					
Binder et al. 1996 (2)	3	11	Ex: 23W Ct: 17W	65 ± 1 67 ± 1	N	DXA	1.0	−2.2	−1.2
Brown et al. 1997 (3)	4	11	Ex: 20W Ct: 16W	65 ± 1 67 ± 1	N	DXA	0.9	−2.8	−1.9
Butterworth et al. 1997 (4)	5	3	Ex: 14W Ct: 16W	73 ± 2 74 ± 1	R	SOS	NS	NS	NS
Coggan et al. 1992 (8)	4	11	Ex: 12M, 11W Ct: None	65 ± 1	N	UWW	0	−3.0	−3.0
Coon et al. 1989 (9)	3	11	Ex: 10M Ct: None	60 ± 3	N	UWW	NS	NS	NS
Cunningham et al. 1987 (11)	3	12	Ex: 113M Ct: 111M	63(Ex + Ct)	R	SOS	NS	NS	NS
Ehsani et al. 1991 (13)	4	12	Ex: 10M Ct: None	64 ± 1	N	SOS	NS	−1.9	NS
Goran and Poehlman 1992 (15)	3	2	Ex: 6M, 5W Ct: None	66 ± 2	N	UWW	0.9	−0.9	NS
Hagberg et al. 1989 (16)	3	6.5	Ex: 16M & W Ct: 12M & W	72(Ex + Ct)	R	SOS	NS	−1.7	NS
Hersey et al. 1994 (17)	3	6	Ex: 8M, 8W Ct: 3M, 6W	72(Ex + Ct)	R	SOS	NS	−1.7	NS
Kahn et al. 1990 (19)	5	6	Ex: 13M Ct: None	69	N	UWW	NS	−2.1	−2.4
Katzel et al. 1995 (20)	3	9	Ex: 71M Ct: 26M	61 ± 1 60 ± 1	R	UWW	1.2	−1.2	NS
Katzel et al. 1997 (21)	3	9	Ex: 21M Ct: None	59 ± 2	R	UWW	0.8	−0.9	NS
King et al. 1991 (22)	3	12	Ex: 127M, 98W Ct: 41M, 34W	50–65	R	UWW	NS	NS	NS
Kirwan et al. 1993 (23)	4	9	Ex: 5M, 7W Ct: None	65 ± 1	N	UWW	NS	−1.4	−1.8
Kohrt et al. 1998 (25)	3	9	Ex: 18W Ct: 10W	66 ± 3	N	DXA	0.7	−3.0	−2.3
Kohrt et al. 1992 (26)	4	11	Ex: 47M, 46W Ct: 16M, 13W	60–70(Ex + Ct)	N	UWW	NS	−2.4	−2.6
Kohrt et al. 1995 (27)	3	9	Ex: 5W Ct: 6W	65 ± 3 66 ± 3	N	DXA	NS	−1.2	−0.8
Kohrt et al. 1997 (28)	3	9	Ex: 14W Ct: 12W	66 ± 1 68 ± 1	N	DXA	NS	−3.2	−2.8
Lan et al. 1998 (29)	5	11	Ex: 9M, 11W Ct: 9M, 9W	65 ± 1 66 ± 1	N	SOS	NS	NS	NS
Meredith et al. 1989 (31)	3	12	Ex: 5M, 5W Ct: None	65 ± 1	N	UWW	NS	NS	NS
Nelson et al. 1991 (34)	4	12	Ex: 18W exp Ct: 18W	60 ± 1(Ex + Ct)	N	UWW	NS	NS	NS
Nieman et al. 1993 (37)	5	3	Ex: 14W Ct: 16W	73 ± 1(Ex + Ct)	R	SOS	NS	NS	NS
Pickering et al. 1997 (38)	3	4	Ex: 4M, 6W Ct: None	62 ± 1	N	SOS	NS	−2.1	NS
Poehlman and Danforth 1991 (39)	3	2	Ex: 13M, 6W Ct: None	64 ± 2	N	UWW	NS	NS	NS
Poehlman et al. 1992 (40)	3	2	Ex: 6M, 1W Ct: None	66 ± 1	N	UWW	NS	NS	NS
Poehlman et al. 1994 (41)	3	2	Ex: 10M, 8W Ct: None	66 ± 1	N	UWW	NS	NS	NS
Posner et al. 1992 (42)	3	4	Ex: 166M & W Ct: 81M & W	69	R	SOS	1.0	−0.4	NS
Schwartz 1988 (47)	3	3	Ex: 10M Ct: None	65 ± 2	N	UWW	NS	−2.9	−2.8
Schwartz et al. 1992 (48)	5	6.75	Ex: 15M Ct: None	68 ± 2	N	UWW	NS	−2.4	−2.5
Seals et al. 1984 (49)	3	6	Ex: 7M, 4W Ct: 6M, 4W	63 ± 1 63 ± 2	N	SOS	NS	−1.6	−1.2
Sial et al. 1998 (50)	3	4	Ex: 3M, 3W Ct: None	74 ± 2	N	DXA	1.1	−1.0	NS
Spina et al. 1993 (51)	4	11	Ex: 15M, 15W Ct: None	64 ± 3	N	UWW	NS	−2.5	−3.0
Spina et al. 1993 (52)	4	11	Ex: 10W Ct: None	63 ± 4	N	UWW	NS	−3.1	−3.2
Spina et al. 1997 (53)	5	9	Ex: 8M Ct: None	66 ± 2	N	SOS	NS	−31.3mm	−5.1
Thomas et al. 1985 (54)	3	12	Ex: 88M Ct: 100M	63(Ex + Ct)	R	SOS	NS	−2.4mm	NS

Values are mean ± SE.
FFM, fat free mass; FM, fat mass; Wt, body weight; Ex, exercise group; Ct, control; R, randomized trial; N, nonrandomized trial; DXA, dual energy X-ray absorptionmetry; SOS, sum of skinfold thickness; UWW, underwater weighing.

TABLE 2. Changes in body composition with resistance exercise in older individuals.

Study	Exercise Program: Frequency (times/wk)	Exercise Program: Duration (months)	Subjects: Number/Sex	Subjects: Age	Random/ Nonrandom	Method	Change FFM (kg)	Change FM (kg)	Change Wt (kg)
Ades et al. 1996 (1)	3	3	Ex: 6M, 6W Ct: 5M, 7W	70 ± 1 71 ± 1	R	UWW	NS	NS	NS
Campbell et al. 1999 (5)	2	12	Ex: 18M Ct: None	56–69	R	UWW	2.1	−2.1	NS
Campbell et al. 1994 (6)	3	3	Ex: 8M, 4W Ct: None	65 ± 2	R	UWW	1.4	−1.8	NS
Campbell et al. 1995 (7)	3	3	Ex: 8M, 6W Ct: None	56–80	R	UWW	1.4	−1.8	NS
Craig et al. 1989 (10)	3	3	Ex: 9M Ct: None	63 ± 1	N	SOS	NS	NS	NS
Dupler and Cortes 1993 (12)	3	3	Ex: 11M, 9W Ct: None	69 ± 1 63 ± 2	N	SOS	NS	NS	NS
Fiatarone et al. 1994 (14)	3	2.5	Ex: 9M, 16W Ct: 12M, 14W	86 ± 1 89 ± 1	R	IVNA	NS	NS	NS
Hagberg et al. 1989 (16)	3	6.5	Ex: 19M & W Ct: 12M & W	72 ± 1(Ex + Ct)	R	SOS	NS	−1.2	NS
Hersey et al. 1994 (17)	3	6	Ex: 9M, 8W Ct: 3M, 6W	72(Ex + Ct)	R	SOS	NS	NS	NS
Hurley et al. 1995 (18)	3	4	Ex: 23M Ct: 12M	60 ± 1 56 ± 1	N	UWW	1.3	−1.5	NS
Koffler et al. 1992 (24)	3	3.25	Ex: 7M Ct: None	60 ± 2	N	UWW	NS	−1.6	NS
Kohrt et al. 1992 (26)	2	9	Ex: 13W Ct: 12W	65 ± 1 68 ± 1	N	DXA	2.0	−2.7	NS
Menkes et al. 1993 (30)	3	4	Ex: 18M Ct: 7M	59 ± 2 55 ± 1	N	UWW	NS	−1.5	NS
Meredith et al. 1992 (32)	3	3	Ex: 6M Ct: 6M	65 ± 2 68 ± 1	R	UWW	NS	NS	NS
Miller et al. 1994 (33)	3	4	Ex: 11M Ct: None	58 ± 1	R	UWW	1.2	−1.4	NS
Nelson et al. 1996 (35)	2	12	Ex: 20W Ct: 19W	61 ± 1 57 ± 1	R	UWW DXA	1.3 NS	−0.9 NS	NS NS
Nichols et al. 1993 (36)	3	6	Ex: 15W Ct: 15W	68 ± 2 65 ± 1	R	DXA	1.5	NS	NS
Pratley et al. 1994 (43)	3	4	Ex: 13M Ct: None	58 ± 1	N	UWW	1.6	−1.9	NS
Rall et al. 1996 (44)	2	3	Ex: 3M, 5W Ct: 2M, 4W	70 ± 2 69 ± 1	R	DXA	NS	NS	NS
Ryan et al. 1995 (45)	3	4	Ex: 8W Ct: None	57 ± 2	N	DXA	1.1	NS	NS
Ryan et al. 1996 (46)	3	4	Ex: 8W Ct: None	58 ± 2	N	DXA	NS	NS	NS
Treuth et al. 1994 (55)	3	4	Ex: 13M Ct: 9M	60 ± 4 62 ± 3	N	UWW DXA	1.7 2.0	−1.7 −2.0	NS NS
Treuth et al. 1995 (56)	3	4	Ex: 14W Ct: None	67 ± 1	N	UWW	NS	NS	NS
Tsutsumi et al. 1997 (57)	3	3	Ex: 14M, 14W Ct: 11M, 3W	68(Ex + Ct)	R	SOS	2.0	−2.0	NS
Yarasheski et al. 1995 (58)	4	4	Ex: 11M Ct: None	66	R	UWW	2.1	−1.7	NS
Yarasheski et al. 1997 (59)	4	4	Ex: 15M Ct: None	66	R	UWW	2.1	−2.1	NS

Values are mean ± SE.
FFM, fat free mass; FM, fat mass; Wt, body weight; Ex, Exercise group; Ct, control group; R, randomized trial; N, nonrandomized trial; UWW, underwater weighing; SOS, sum of skinfold thickness; IVNA, *in vivo* neutron activation; DXA, dual energy X-ray absorptiometry.

with REX are needed to define the mechanism underlying REX-induced reductions in adiposity.

Changes in Fat-Free Mass with REX

Evidence Statement: REX is an effective intervention to increase fat-free mass in older individuals. (Evidence Category C). In contrast to AEX, REX increased fat-free mass in 15 out of 28 studies (range: 1.1 to 2.1 kg). This finding is not surprising considering that REX is a potent anabolic stimulus to skeletal muscle growth. However, the specific component of fat-free mass that is increased with REX training has not been defined. Fat-free mass is a heterogenous compartment of body mass that is composed on a chemical level of minerals, water, protein and glycogen. Because the absolute (i.e., kilograms) contribution of minerals, protein, and glycogen to fat-free mass is quite small, changes in fat-free mass resulting from REX are likely caused by changes in body water. In support of this notion, data from Campbell et al. (5,6) showed that the increase in fat-free mass observed in older men and women following a 12-wk REX program was almost completely accounted for by an increase in total body water. Similar changes in total body water with REX training were found

by Nelson et al. (35) in a 1-yr randomized study of older women. These changes in body water may be partially caused by increased skeletal muscle mass with REX. That is, REX-induced increases in skeletal muscle mass cause an increase in total body water because approximately 73% of skeletal muscle mass is water. Results from Nelson et al. (35) which showed that changes in total body water with REX primarily resulted from increased intracellular water support this conclusion. Thus, although REX appears to be effective in increasing fat-free mass in older men and women, a significant portion of this increase may be caused by changes in body water.

Body composition methodology and changes in fat mass and fat-free mass with exercise. Recent developments in body composition methodology permit investigators to reliably assess multiple components of body mass. Despite these advances, however, most studies examining the effects of AEX and REX on body composition have employed the classic two-compartment model of body composition which divides body mass into fat and fat-free compartments. Because two-compartment models have been used, the specific tissue component of fat-free mass that changes with exercise has yet to be clearly defined. For example, as mentioned above, a significant portion of the REX-induced change in fat-free mass may be caused by changes in body water. Moreover, exercise-induced changes in body composition may be incorrect because of propagation of error. That is, in two-compartment models of body composition that measure one component of body composition and calculate the second component from the difference with body weight, error in the measured component of body composition is propagated to the calculated component. This may affect estimated changes in fat and fat-free mass with either AEX or REX. From these caveats, we can see that changes in body composition observed with AEX and REX may be dependent on the type of body composition methodology used. Thus, we recommend that future studies of the effects of AEX and REX on body composition in the elderly employ several body composition techniques to measure the various tissue components of fat-free mass, in particular body water, so as to clearly define the exact tissue components of body mass that are affected by AEX and REX.

RESEARCH PRIORITIES

Although our review of literature allows several tentative conclusions, many questions remain unanswered. We suggest the following questions and areas for future investigation:

- Only 10 of 36 AEX studies and 15 of 26 REX studies were randomized, controlled studies. Future studies should randomize subjects to exercise and control groups.
- What is the effect of gender in changes in body composition with AEX and REX? Most studies have not specifically compared the effect of gender on changes in body composition with AEX and REX despite a growing body of evidence that suggests a sex dimorphism in the response of certain hormonal and physiological systems to exercise.
- What is the effect of the intensity and duration (i.e., total number of exercise sessions) of the exercise intervention on body composition variables? Future studies should examine temporal changes in body composition with AEX and REX training regimens by conducting serial measurements of body composition throughout the exercise program and should examine AEX and REX programs of various intensities.
- What component or components of body composition are affected by AEX and REX training? Future studies should measure multiple components of body composition, including body water, using various techniques to determine the components of body composition affected by exercise.
- What is the mechanism underlying reductions in fat mass with AEX and REX training? Future studies should focus on measuring changes in energy expenditure and energy intake with AEX and REX training with doubly labeled water and multiple body composition methodologies.

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Overweight and obesity in the mortality rate data: current evidence and research issues

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ABSTRACT

SEIDELL, J. C., T. L. S. VISSCHER, and R. T. HOOGEVEEN. Overweight and obesity in the mortality rate data: current evidence and research issues. *Med. Sci. Sports Exerc.,* Vol. 31, No. 11, Suppl., pp. S597–S601, 1999. **Purpose:** The relation between indicators of overweight (body mass index (BMI)) and all-cause mortality and factors that potentially affect such a relationship were reviewed. **Methods:** The literature was reviewed. **Results:** Although there are many reports on the relationship between indicators of overweight (such as BMI) and all-cause mortality, there are no two studies that have been analyzed identically. It is now usually assumed that there is a U- or J-shaped association between BMI and mortality, but there are many issues that remain unsolved until today. These issues include the effects of: adequate control for cigarette smoking; adequate control for (sub)clinical disease at baseline; adequate control for intermediate risk factors; adequate measures for exposure to obesity; age, period, and cohort effects; adequate control for underlying lifestyle factors; adequate control or stratification for ethnicity and socioeconomic status; effects of sample size and duration of follow-up; and reliance on self-reported body weight and height. **Conclusion:** The literature is dominated by studies in young adult and middle-aged white inhabitants of North America and Europe. In those populations, it seems well accepted that lowest mortality is in the range of BMI between 18.5 and 25 $kg \cdot m^{-2}$. When BMI reached values of 30 $kg \cdot m^{-2}$ or more, mortality is substantially elevated by about 50–150%. These results may not be generalizable to other populations, and more studies are needed. All evidence is of category C (observational studies). **Key Words:** OBESITY, MORTALITY, OVERWEIGHT, BODY MASS INDEX, ETHNICITY, LIFESTYLE

In this short review, we will limit our discussion to the nature of the relationship between body mass index (BMI) and all-cause mortality. All of the evidence cited here is from observational studies (evidence category C). It is unlikely that the issue of body weight and mortality will ever be resolved from randomized controlled trials. The relation between indicators of overweight and mortality has been the subject of many studies over the last 40 years. Many of the important studies have recently been reviewed by Stevens (27). It is often stated that the relationship between BMI and all-cause mortality is U- or J-shaped (15,30). It has been argued that the high mortality at low BMI is due to confounding of smoking and presence of disease and that, in fact, mortality rates are progressively increasing throughout the range of BMI (11,12,16,33). The relation between BMI and mortality is still subject to debate in the literature partly because most studies suffer from methodological drawbacks. Often cited are those proposed by Manson et al. in 1987 (15):

i. failure to control for cigarette smoking;

ii. failure to eliminate effects of clinical or subclinical illness present at baseline; and

iii. inappropriate adjustment for intermediate risk factors in the relation between BMI and mortality such as hypertension, hyperlipidemia, and diabetes.

Other problems are:

iv. the use of one single estimate of overweight by the BMI ($kg \cdot m^{-2}$) as an indicator of exposure of individual to excess fat mass;

v. age, period, and cohort effects that may modify the association between BMI and mortality;

vi. failure to control for underlying lifestyle factors contributing to variation in BMI in the population;

vii. failure to adequately control for effects of socioeconomic status and ethnicity;

viii. insufficient sample size and duration of follow-up both affecting the strength of the association between BMI and mortality; and

ix. reliance on self-reported height and weight.

We will briefly discuss some of these methodological issues below:

i. The potential role of smoking habits concerns the possibility that this is a confounder or an effect modifier in the relationship between overweight and death. The role of smoking as a confounder can be explained by the observation that smokers have lower BMI compared with nonsmok-

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ers as well as higher mortality rates. This could attenuate the relationship between BMI and mortality. The issue of smoking being an effect modifier of the relationship between BMI and mortality was first raised by Garrison et al. in 1983 who reported that there appeared to be a J- or U-shaped relationship between "Metropolitan Relative Weight" (MRW) and death in male smokers but a direct linear relationship in nonsmokers (7). These data have been recently reanalyzed this time with the conclusion that significant U- or J-shaped associations between MRW and mortality could be found in female and male smokers and nonsmokers (24). The authors advocate caution in interpreting relationships by visual inspection only especially when data are thin. In addition, it became clear that Garrison excluded nonsmokers at the lowest level of relative weight.

Also in other studies it has been proposed that the relationship between BMI and mortality in healthy nonsmokers is linear (11,16), but in many studies a curvilinear relationship remains.

Although the issue whether or not smoking should be considered to be a confounder or an effect modifier of the overweight-mortality relationship is not settled, it is safe to recommend to always stratify the analyses by smoking habits if only because this allows more direct estimates of absolute risk.

ii. The issue of preexisting clinical and subclinical disease is controversial. Although not many dispute the possibility that, especially at the lower ranges of BMI, some diseases may lead to dramatic weight loss as well as a high risk of dying, the methods to overcome these have not been clearly established. Because information about subclinical disease is usually unavailable, most researchers follow the advice by Manson et al. "to disregard mortality within the first few years of follow-up, based on the assumption that such deaths are largely due to disease present at entry" (15). Allison et al. recently completed a meta-analyses on this issue and concluded that "the effect of eliminating early deaths was statistically significant but minuscule in magnitude" and that "either preexisting disease does not confound the BMI-mortality association or that eliminating early deaths is inefficient for reducing that confounding" (1). This issue clearly needs further research.

iii. In early studies like the Framingham Heart Study, the association between metropolitan relative weight and mortality was evaluated from statistical models that incorporated other risk factors at the same time, such as blood pressure, serum cholesterol, and blood glucose (31) From such analyses, MRW did not appear to be independently related to mortality, which was interpreted that obesity *per se* was not a risk factor and that obesity is benign when it exists without other major risk factors for cardiovascular disease. Similar conclusions were also drawn by Keys from analyses of the Seven-Countries Study (8). Although these authors did acknowledge that other cardiovascular disease risk factors are likely to be intermediates in the causal chain linking obesity to mortality, the emphasis in their interpretation of the data was to focus on treatment of other risk factors than obesity. In the last 10 years or so, more authors tend to concentrate on age- and smoking-adjusted results, and emphasis has shifted toward treatment of obesity as a primary target. Another issue is that the coexistence of obesity with other risk factors for mortality such as diabetes mellitus, hypertension, and hyperlipidemia may potentiate the risk of dying in obese subjects (16,22). For some time, it was proposed that lean hypertensives have higher mortality compared with obese hypertensives, but this has not been confirmed in later studies (22,26). Stratification by risk factor status is not done often but should be considered when possible.

iv. It is well accepted that there are limitations in the use of BMI as a measure of fatness. Especially in the middle-range of BMI values, contributions of lean body mass and body fat mass are both relatively large. It is likely that body fat and lean body mass have different associations with mortality. One alternative interpretation for this curvilinear association is that it is the result of the combination of two linear functions, namely that increasing fat mass is directly associated with increasing risk and increasing lean mass is inversely associated with decreasing risk (2).

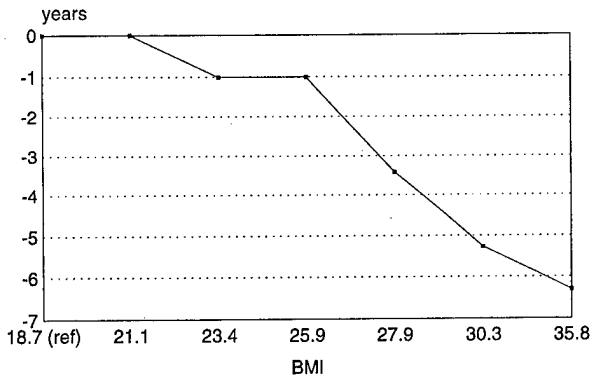
The issue of the limitation of BMI as a measure of fatness becomes an increasingly important one at older ages. For a given BMI, an older woman is likely to have more fat mass but less muscle and lean body mass than a younger woman.

Most epidemiological studies on relationships between exposures and outcomes have a limitation in common that they regard one measurement of exposure as indicative of exposure. This implies that investigators have to ignore effects of body weight changes before and after the measurement. Several reports suggest that weight change and weight fluctuations are independent determinants of mortality (14,17,34,35). Weight changes and fluctuations may be more common toward extremes of the distribution of BMI in the populations and may affect the risk estimates at these levels.

v. Age, period, and cohort effects are general issues that are not unique to the BMI-mortality relationship but may be relevant here. Age, period (calendar time), and cohort (year of birth). When analyses of the impact of age on the BMI-mortality relationship is evaluated (3,28), usually cohorts of different age-groups are followed for an identical period, and mortality rates and ratios are calculated in categories of BMI.

Not only do these groups have different ages, they also have different birth-years and may or may not share time periods that affect both mortality and body weight.

For example, one of the largest studies on BMI and mortality was performed in Norway where 1.7 million Norwegians aged 20–90 yr were recruited between 1963 and 1979 and followed for an average of 10 years (32). Ninety-year-olds recruited in 1963 were born in 1873 and experienced as youngsters the large tuberculosis epidemics at the turn of the century and also experienced World War I and II as adults. During most of their life up to 1963, infectious diseases were the major cause of death, cardiovascular diseases were rare, and life expectancy was low. Those recruited in their twenties were born in the period around



calculated from Manson et al N Engl J Med 1995;333:677-85

Figure 1—Estimated differences in life expectancy in 30-yr-old women across categories of BMI (BMI < 19 kg·m^{-2}) as the reference. Calculated from relative risks in the Nurses' Health study and applied to life expectancy in The Netherlands.

World War II when food restriction was common. Thus, besides an effect of age, we may also be looking at effects of different past and unshared exposures affecting body weight development and mortality and generizability to people of different ages living today may not be appropriate.

vi. The issue of control for underlying lifestyle factors is one that has not been dealt with extensively in the past. Variation in body weight is partly the result of variation in energy expenditure and energy intake. It is thought that obese subjects are less active but have higher metabolic rates compared with nonobese subjects. Cause and effect are difficult to ascertain in the activity-obesity relationship. Physical inactivity may lead to increased obesity (21). Alternatively, obese subjects have often physical limitations caused by impaired respiratory function and musculoskeletal problems (9), which may lead to reduced physical activity. Blair et al. have proposed that high levels of obesity may be relatively benign when accompanied by high levels of fitness (10). The coexistence of high levels of fitness and obesity, however, may be rather low in most sedentary societies.

Also with respect to dietary intake data there are problems in methodology and interpretation of findings. High fat intakes have been implicated in the development and sustaining of high body weights, although the evidence is not as strong as often proposed. With regard to food intake data, there is the additional problem of underreporting of intake and intermittent dieting (4).

The risk of obesity may, however, be very well dependent upon underlying behavioral characteristics. Obesity produced in Finnish male subjects by high intakes of beer, sedentary lifestyle, and high-fat diets (19) may be associated with very different health risks compared with obesity in traditional Mediterranean diets and heavy labor. In a study comparing measures of obesity and other risk factors among European women aged 38 yr, we observed that Dutch women had an average BMI of 23 kg·m^{-2} and women in a village outside Naples, Italy, had a mean BMI of 28 kg·m^{-2} (23). Nevertheless, cholesterol levels were higher in Dutch women (mean: 5.6 mmol·L^{-1} vs 4.9 mmol·L^{-1} in Italian women). This may reflect underlying differences in diet and/or genetic factors. Alcohol consumption may also be important in this respect. In one study, it was observed that moderate and high alcohol consumption were associated with increased mortality especially at low levels of BMI (5).

vii. Effects of socioeconomic status and ethnicity: low socioeconomic status is related to increased prevalence of obesity as well as increased mortality and this may therefore confound the relationship between obesity and mortality. Many of these confounding effects may be due to underlying differences in lifestyle, but it is also likely that high educational levels reflect other reasons for low mortality and low BMI (like better access to adequate medical interventions and prevention programs). Few studies have adequately dealt with the influence of components of socioeconomic status (education, income, and profession) on the BMI-mortality relationship. Tayback et al. (29), for instance, observed in elderly women that low BMI was associated with increased mortality only in women with poor socioeconomic status. Ethnicity is in many societies strongly associated with socioeconomic status. Without trying to be complete, it is clear that the relations between BMI and mortality vary across ethnic groups. This partly reflects limited appropriateness of BMI as an indicator of body fatness (6), but there may be other reasons. In Pima Indians, for instance, relative excess mortality is not observed until BMI reach values well over 40 kg·m^{-2} (18). At BMI levels below 40 kg·m^{-2}, there is no relationship between BMI and mortality in this particular subgroup.

viii. Sample size and duration of follow-up: Sjöström (25) showed in a review of 51 cohorts (40 studies) that studies in which no relationship between BMI and mortality was found were clustered among small and/or short-term studies. All studies with more than 20,000 subjects and 20 of 21 studies with more than 7000 subjects showed a positive association between BMI and mortality. He estimated that follow-up needs to be longer than about 5 years (but preferably much longer) to be able to demonstrate effects of obesity and mortality. It seems obvious that duration of follow-up is important (in young women, a short follow-up will not be sufficient to see the long-term impact of obesity on chronic diseases and mortality), but very little systematic work is done in this area. For instance, in Seventh-Day Adventist women followed for 26 yr, it was observed that in those aged 30–54 a weak linear association between BMI and mortality was observed during the first 8 yr of follow-up, a significant linear relation during years 9–14 of follow-up, and a U-shaped association during 15–16 yr of follow-up (13).

ix. Self-report of weight and height. Many of the largest studies use self-reported height and weight. Although the validity is reasonably adequate for ranking individuals, there is evidence that with increasing degrees of obesity subjects tend to underestimate their weight (20).

Measures of effect: Usually the relation between BMI and mortality is expressed in terms of relative risk in categories of BMI as compared with an arbitrary reference category. The choice of reference categories and cut-off points for

TABLE 1. Current issues in the study of the relationship between body mass index and all-cause mortality.

Issue	Problem	State of the Art	Recommendation
Smoking behavior	Confounder/effect modifier	Could be either, formal testing rarely done	Stratification, test for interaction if statistical power is sufficient
(Sub)clinical illness at baseline	May explain mortality at low BMI	Usually done by exclusion of early mortality but may not be sufficient	Data quality on (sub)clinical illness should be improved
Intermediate risk factors	Are sometimes controlled for in analyses	Is now usually not done but they may be effect modifiers	More formal tests for interaction needed
Single BMI value as exposure to obesity	BMI is not ideal measure of body fatness; weight fluctuations may affect outcome	BMI tends to be accepted although problems in aging and ethnic variations are usually acknowledged	Better data on body composition and fat distribution needed; more emphasis on voluntary and involuntary weight history and development needed
Age, period, and cohort effects	Effect of age studied ignoring cohort and period effects	A general problem in epidemiological research	Needs to be properly addressed
Underlying lifestyle determinants of BMI (other than smoking)	Activity, fitness, diet, and alcohol may all be confounders/effect modifiers	Fitness is now made an issue but remains problematic in terms of causality	Methodology for unbiased estimates of both sides of the energy balance needed
SES and ethnicity	May both be confounders/effect modifiers	Is rarely studied for SES; data on ethnicity are now becoming more common	Effects of components of SES need to be addressed in future
Self-reported data	May attenuate or cause flat-slope syndrome	Validation usually done by correlations with measured data	Real effects need to be studied in more detail

other categories will greatly affect the estimates of relative risk. Absolute risks or population attributable risks are alternatives less often used but these suffer from the same drawbacks with respect to choice of classification of BMI.

Differences in life expectancy are also very illustrative, but we know of no studies in which this was expressed with respect to overweight and obesity. From the data of the Nurses' Health study, we estimated differences in life expectancy by degree of overweight taking the leanest subgroup (average BMI 18.5 $kg \cdot m^{-2}$ as the reference). Only data of women who had never smoked were used. From our calculations, we estimate that women with BMI over 30 $kg \cdot m^{-2}$ live about 5–6 yr fewer than very lean women (Fig. 1).

This is a compelling illustration of the importance of preventing and treating obesity adequately and vigorously.

CONCLUSIONS AND RECOMMENDATIONS

Although many studies have been published on the relation between BMI and all-cause mortality, there are still many issues that need to be resolved before definite conclusions can be made (see Table 1). Most of the evidence points toward a U- or J-shaped association. The extent to which the increased mortality is attributable to smoking and (sub)clinical illness remains not totally clear, but both do not seem to play a major role at high BMI levels. Among the research priorities are the development of more appropriate measures of body fatness especially in the elderly and nonwhite populations. The effects of underlying lifestyles leading to obesity and leanness as well as to mortality may be among the most important research topics. This is not an easy task and requires the development of accurate methodology that allows quantification of energy intake and expenditure in population studies.

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Comorbidities of overweight and obesity: current evidence and research issues

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ABSTRACT

PI-SUNYER, F. X. Comorbidities of overweight and obesity; current evidence and research issues. *Med. Sci. Sports Exerc.,* Vol. 31, No. 11, Suppl., pp. S602–S608, 1999. **Purpose:** The evidence with regard to the relationship of obesity with medical comorbidities was assessed and priority research issues identified. **Methods:** The existing literature in English was surveyed. **Results:** The evidence is overwhelming on the association of obesity to a number of medical conditions. These include: insulin resistance, glucose intolerance, diabetes mellitus, hypertension, dyslipidemia, sleep apnea, arthritis, hyperuricemia, gall bladder disease, and certain types of cancer. The independent association of obesity seems also clearly established for coronary artery disease, heart failure, cardiac arrhythmia, stroke, and menstrual irregularities. The relationship between central (or upper body) obesity and the above conditions is positive for most of them but with a lesser number of studies. Most of the fat distribution studies have been done using anthropometric measurements rather than the more accurate magnetic resonance imaging or computer tomographic scans. Priority research issues include the following: more definitive data on the relation of central fat to comorbidities; the proportional importance of subcutaneous versus visceral fat in producing comorbidities; the relationship between obesity and psychiatric disease; the genetics of the relationship between obesity and each of the comorbidities; the independent contribution of diet and of sedentariness to the development of each of the comorbidities; the impact of gender, race, intensity, and duration on these associations. **Conclusions:** The evidence for the relationship of obesity to a number of comorbidities is strong, though the strength of the relationship varies with the condition. Much more research is necessary on causation and on what other factors may play an interactive role. **Key Words:** RISK FACTORS, DISEASE, FAT, FAT DISTRIBUTION

There is a very large bibliography on the association of overweight and obesity with other medical conditions. The data include evidence for overweight and obesity and also for the distribution of body fat. The evidence that is available in English with regard to the former will be reviewed first and with regard to the latter second.

CURRENT STATUS OF KNOWLEDGE

Overweight and Obesity

Type II diabetes mellitus. Although the relationship of obesity to Type II diabetes mellitus is not wholly clear, two facts are incontrovertible. Excess body fat leads to increasing insulin resistance (82), and insulin resistance predisposes to diabetes (82,116). Why, however, some individuals may be obese for years without developing diabetes, whereas others develop it readily, must depend on their genetic make-up. Until more is known about the genes responsible for insulin resistance and for Type II diabetes, the exact mechanisms involved will not be clarified.

Insulin resistance, impaired glucose tolerance, and diabetes mellitus have been strongly associated with overweight and obesity in many cross-sectional (68,100,115,120,149,150) (52,64,88,113,142,151,159) and longitudinal (17,20,57,81,92) studies.

Hypertension. The association between increased blood pressure and overweight and obesity has been shown repeatedly in both cross-sectional (2,3,7,14,34, 47,61,97,123,127,133,141,143,152,155) and longitudinal (66,69,103,140) studies. The independence of obesity as a risk factor for hypertension is underscored by observations of a much higher prevalence of hypertension in obese persons than in the general population. This has been reported both in populations with a high prevalence of hypertension, such as African-Americans (129), as well as in populations with a low prevalence of hypertension, such as Mexican-Americans (54). Weight gain leads to an increase in blood pressure, as has been reported in the Framingham study (72) among others. The mechanisms proposed for this relationship have been many (114) and until the genes responsible for this multi-etiological condition and the relationship of these genes to the genes that predispose to obesity have been identified, these mechanisms will not be clarified.

Dyslipidemia. Obesity has been associated with two particular abnormalities: increased levels of triglycerides and decreased high-density lipoprotein cholesterol (high density lipoprotein (HDL)-C). Both of these are now recognized as independent risk factors for cardiovascular disease (8,9,48). Cross-sectional (128,143) as well as longitudinal (8,9) studies have shown the relationship of obesity to

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higher levels of triglycerides. Also, low HDL-C levels have been found in both cross-sectional (45,143) and longitudinal (48) studies. An increase in triglycerides has also been described longitudinally with an increase in weight (4). Total and low-density lipoprotein cholesterol (low density lipoprotein (LDL)-C) have been found to be either normal or elevated (8,37,128,142). In more recent years, there have been descriptions of a qualitative change in the size of LDL particles, with an increase in the number of small, dense ones that are more atherogenic (10). The relationship has been clearly shown for insulin resistance (11,98,117) and has thereby been extrapolated to obesity, which is an insulin resistant state (98). More research on the presence of this LDL-C phenotype (phenotype B) in obese populations is required.

Coronary heart disease. Coronary heart disease (CHD) is usually described epidemiologically as angina pectoris, nonfatal myocardial infarction, and sudden death. There have been differing opinions on the relationship of obesity to CHD. This is because, since the obesity enhances the risk of hypertension, dyslipidemia, and diabetes mellitus, all of which are risk factors for CHD, the independent effect of the obesity itself has been difficult to verify. However, recent studies have left little doubt of the relationship in both men and women (18,21,38,49,70,78,79,99,121,131). There is also an association between obesity and left ventricular hypertrophy and congestive heart failure (66,70,71,73,74). Arrhythmia and sudden death are also associated with obesity (31,66,73,101). The causative mechanisms between obesity and sudden death is not clear. Whether it is related to alterations in sympathetic and/or parasympathetic nervous activity has been debated (42,63,158).

Gall bladder disease. Both cross-sectional (16,50,83,120) and longitudinal (41,75,96,109,130,138) studies have reported an association between gall bladder disease and overweight and obesity.

Respiratory disease. There is a gradually increasing impairment of respiratory function with increasing obesity. The sheer burden of the extra fat in the chest wall and the abdomen causes a decreased compliance of respiratory function and reduces lung volume (102,147). A ventilation-perfusion abnormality occurs (12) that initially causes hypoxia with normal pCO_2 (30,65). Eventually, however, as the severity of obesity increases, hypercarbia also develops. Sleep apnea occurs as the severity and duration of the obesity exacerbates (119,146,157). This may be obstructive sleep apnea (118) or central (13) or a combination of the two. Eventually, the obesity-hypoventilation syndrome, with depression of hypercapnic and hypoxic respiratory drives, irregular breathing, frequent apneic periods with resultant hypoxia (84) and daytime sleepiness supervenes. Finally, cor pulmonale can occur (124).

Cancer. The relationship of obesity to cancer has been a very difficult question to sort out and is still deeply mired in controversy. Part of the problem is that even though, in longitudinal studies, it is clear that obesity is associated with some cancers, it has been very difficult to separate the role of overweight and obesity *per se* from the effect of the macronutrient composition of the diet or of total calories (114). The American Cancer Society study, which was a longitudinal study that followed 750,000 men and women for 12 yr, found that the mortality ratio for cancer for men who were 40% or more overweight was 1.33 and for women it was 1.55 (43). These obese men had higher rates of colorectal and prostate cancers, whereas the women had higher rates of endometrial, gallbladder, cervical, ovarian, and breast cancers (43). With regard to breast cancer, other longitudinal studies have described a greater risk for obese postmenopausal women but not for obese premenopausal women (23,93,94,108,132,136,145,154). There have been a number of studies that have shown positive associations between weight and endometrial cancer (40,85,89,90,125,132,137). In men, some studies have shown increased colon cancer (91,112), but others have not (105,139,153,156).

Osteoarthritis. Cross-sectional studies have reported an association between obesity and osteoarthritis of the knees (1,6,22,39,46,59,77,120). A twin study in which one twin weighed more than the other showed a strong association between obesity and osteoarthritis (58). This report concluded that every 5 kg of gain in weight increased the risk of knee arthritis by 35%. The evidence for the relationship of obesity to osteoarthritis of the hip is much less strong (59,77,144).

Association between Fat Distribution and Medical Conditions

A considerable literature is now available with regard to the association of central or upper body fat distribution and health risk. Most of the epidemiological studies that have been published have been done using anthropometric measurements as surrogates for fat distribution.

Hypertension. There have been a number of cross-sectional (29,53,68,110) and longitudinal (66) epidemiological studies that have reported an association between central fat distribution and hypertension.

Type II diabetes mellitus. There are a number of studies, both cross-sectional (44,56,60,67) and longitudinal (55,57,76,95,106), that have shown an association between obesity and diabetes mellitus. The increased central fat distribution leads to insulin resistance and impaired glucose tolerance (28,35,36,80,111,126).

Dyslipidemia. There have been a number of studies that have described dyslipidemia in patients with central fat distribution. The syndrome is similar to that seen for obesity *per se*, with hypertriglyceridemia, low HDL-cholesterol, and an increased number of small dense LDL particles, which are very atherogenic (24–27,107,135). In addition, increased apoB lipoprotein levels have been described (89).

Cardiovascular disease. Cross-sectional studies have shown a relationship between central fatness and CHD (89). Longitudinal studies have reported similar findings (19,29,32,33,62,86,87,121,134).

There is also a report of enhanced risk for stroke with central obesity (148).

Other medical conditions. Gall bladder disease has been reported to be associated with central fat distribution (56).

RESEARCH PRIORITIES

There are many unanswered questions on the association of obesity and central fat distribution with health risks. Research priorities with regard to these associations are numerous. Most important is the fact that all of the above cited studies show an association or correlation between obesity, central fat distribution, and morbidity, but correlation does not necessarily indicate causality (5). Three studies have suggested that obesity and cardiovascular risk factors are not pleiotropic (51,104,122). In addition, within person changes in fatness lead to changes in morbidity (71,103,140). Also, there is a report of a negative relationship between baseline cardiovascular risk factors and future fatness, suggesting that these risk factors are not elevated before weight gain (15). Clear confounders are physical activity, composition of the diet, caloric intake, and smoking. The role of each of these confounders on comorbid conditions needs to be investigated.

More investigation is also needed on the following:

1. The relation of central fat to comorbidities, with better characterization of the central fat so as to be able to sort out the contribution of central versus subcutaneous fat tissue to each comorbidity.

2. The genetics of the relationship between obesity, fat distribution, and each of the comorbidities.

3. The impact of gender, race, intensity, and duration of obesity and fat distribution on each of the comorbidities.

4. The interaction between obesity and central fat distribution and other potential associated factors responsible for particular comorbidities.

5. The relationship between obesity and psychiatric disease.

6. The independent contribution of diet and of sedentariness to the development of each of the comorbidities.

CONCLUSION

The evidence for the relationship of obesity to a number of comorbidities is strong, but much more research is necessary on causation and on what other factors play a role.

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Physical activity for preventing and treating obesity-related dyslipoproteinemias

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ABSTRACT

STEFANICK, M. L. Physical activity for preventing and treating obesity-related dyslipoproteinemias. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S609–S618, 1999. **Purpose:** The clinical trial data were reviewed on effects of physical activity on obesity-related dyslipoproteinemias (specifically low HDL-cholesterol (HDL-C), elevated triglycerides (TG), and high total and LDL-cholesterol (TC and LDL-C)) in adult men and women. **Methods:** Effort was made to identify all randomized clinical trials (RCT), with exercise intervention programs of at least 4 months' duration, which had lipoprotein outcomes. Those that had both an exercise only intervention and control groups or both a diet plus exercise and identical diet only intervention groups were reviewed. Tables were developed of baseline characteristics and weight and lipoprotein changes for aerobic exercise trials by body mass index: 1) < 25.0 kg·m^{-2}, 2) 25.0–29.9 kg·m^{-2}, and 3) ≥ 30.0 kg·m^{-2} and for studies involving resistance exercise or increased energy expenditure from daily activities versus structured exercise programs. **Results:** Very few RCT were found that specifically addressed the role of physical activity in preventing or treating obesity-related adverse lipoprotein levels. There was essentially no evidence found in lean or overweight men or women to support a specific role for exercise in improving undesirable lipoprotein levels; however, trial data strongly suggest that the addition of exercise to a hypocaloric, reduced-fat diet improves HDL-C and TG in men and women with generally desirable initial levels and reduces LDL-C in men and women with initially elevated LDL-C levels. The evidence is also reasonably strong that weight loss, including that achieved solely by exercise, improves HDL-C and TG in obese men, without reducing LDL-C, whereas it remains weak for women. There are also virtually no trial data to support a role for resistance exercise or an increase in daily living activities for improving obesity-related lipoproteins. **Conclusions:** Current evidence from RCT is too limited to determine whether physical activity can raise low HDL-C or lower high TG or LDL-C levels in overweight and obese individuals. **Key Words:** EXERCISE, LIPOPROTEINS, HIGH DENSITY LIPOPROTEIN CHOLESTEROL, LOW DENSITY LIPOPROTEIN CHOLESTEROL, TRIGLYCERIDES, WEIGHT LOSS, OVERWEIGHT

The 1985 Panel for the National Institutes of Health (NIH) Consensus Development Conference on the Health Implications of Obesity recognized hypercholesterolemia as one of the adverse effects of obesity (18). In 1993, the second National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP-II) promoted weight loss to treat hyperlipidemia and low levels of high-density lipoprotein cholesterol (HDL-C) in overweight patients (7). Subsequently, the Panel for the 1995 NIH Consensus Development Conference on Physical Activity and Cardiovascular Health concluded that exercise training of at least 12 wk duration results in beneficial changes in HDL-C (19) but stopped short of linking this to the loss of body fat that may accompany increased aerobic exercise. More recently, the Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity recommended weight loss to lower elevated levels of total cholesterol (TC), LDL-cholesterol (LDL-C), and triglycerides (TG), as well as to raise low levels of HDL-C, in overweight and obese persons with dyslipidemia (8). In addition, the Expert Panel concluded that physical activity, particularly when combined with dietary therapy, contributes to weight loss. The Expert Panel did not, however, evaluate the relationship between exercise-induced weight loss and lipoprotein changes or discuss a specific role for physical activity in preventing or treating lipoprotein disorders associated with obesity. The literature pertaining to these issues will be reviewed here.

Because of space limits, this review will be confined to randomized, controlled trials (RCT) that were designed to determine effects of increased physical activity on both weight and lipoproteins. This follows the evidence-based approach of the Expert Panel which recognized RCT as the best source of information (8). Therefore, the large number of epidemiological studies that have reported higher HDL-C and lower TG levels in individuals who self-selected a more physically active lifestyle, and who are generally much leaner than their sedentary counterparts (23,24,27,28,30), will not be discussed, nor will this paper review longitudinal studies which have reported significant relationships between physical activity level and weight and lipoprotein changes over time (16,23,24,30). Finally, this paper will not review the many elegantly designed metabolic and clinical studies designed to focus on the physiological effects of

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TABLE 1. Randomized trials of lipoprotein effects of aerobic exercise in individuals with mean BMI < 25.0 kg·m^{-2}.

Study	Interventions: Treatment Groups	*N* post (baseline); Sex; Basic (key) Inclusion criteria	Training Duration	Mean[a] Baseline BMI [Wt] and Lipids	Weight Change (kg) [BMI]	HDL-C (mmol·L^{-1})	LDL-C (mmol·L^{-1})	TG (mmol·L^{-1})	TC (mmol·L^{-1})
Huttunen et al., 1979 (11)	1: Aerobic exercise 2: Control	90 (100); male; age 40–45 yr	4 months	Wt: 79.2 HDL: 1.3 LDL: 4.8 TG:1.5 TC: 6.8	1: −0.9 2: −0.6 NS	1: +0.14 2: +0.02 $P < 0.01$	1: −0.45[b] 2: −0.25[b] NS	1: −0.24[b] 2: +0.15[b] $P < 0.05$	1: −0.8[b] 2: −0.3[b] NS
Wood et al., 1983 (29)	1: Aerobic exercise 2: Control	78 (81); male; age 30–55 yr; <140% ideal wt; sedentary	9–12 months	BMI: 24.9 HDL: 1.3 LDL: 3.8 TG: 1.4 TC: 5.6	1: −1.9 2: +0.6 $P = 0.002$	1: +0.05 2: +0.01 NS	1: −0.13 2: +0.03 NS	1: −0.09 2: +0.04 NS	1: −0.13 2: +0.05 NS
Leighton et al., 1990 (15)	1: Aerobic exercise + diet (counseling on low-fat) 2: Diet only (same)	51 (17 + 49); male and female; age ≥ 20 yr; high TC (> age-spec. NIH, 1985)	26 wk	Wt: 72.5 HDL: 1.8 LDL: 4.7 TG: 1.6 TC: 7.0	1: −3.0 2: 0.0 $P < 0.001$	1: −0.23 2: −0.21 NS	1: −0.51 2: −0.21 NS	1: −0.10 2: −0.19 NS	1: −0.98 2: −0.49 NS
Duncan et al., 1991 (5)	1: Aerobic walking (8.0 km·h^{-1}) 2: Brisk walking (6.4 km·h^{-1}) 3: Strolling (4.8 km·h^{-1}) 4: Control	59 (102); female; premenopausal; age 20–40 yr; sedentary	24 wk	BMI: 23.7 HDL: 1.4 LDL: 2.9 TG: 1.0 TC: 4.8	1: +1.1 2: +0.1 3: +0.8 4: +3.7 NS	1: +0.08 2: +0.06 3: +0.08 4: +0.02 NS	1: +0.09 2: −0.25 3: −0.12 4: +0.13 NS	1: +0.11 2: 0.00 3: −0.09 4: −0.15 NS	1: +0.23 2: −0.18 3: −0.09 4: +0.11 NS
Singh et al., 1993 (21)	1: Aerobic exercise + low-fat diet plus vegetables, fruits, and grains 2: Low-fat diet only	457 (419 + 44); male and female; age 25–65 yr; with ≥1 CHD risk factor (or >110% wt, sedentary, CHD)	24 wk	BMI: 24.4 HDL: 1.2 LDL: 4.3 TG: 1.8 TC: 6.3	1: −6.5[c] 2: −2.3[c] $P < 0.01$	1: +0.20[c] 2: −0.05[c] NS	1: −0.61[c] 2: −0.23[c] $P < 0.01$	1: −0.33[c] 2: −0.13[c] $P < 0.01$	1: −0.82[c] 2: −0.28[c] $P < 0.01$

[a] Mean estimated from baseline means reported for individual treatment groups.
[b] Data interpreted from graphs.
[c] Values calculated from percent change from baseline.

exercise on body composition and lipoprotein metabolism in highly controlled settings, which generally involve a small number of participants (22,24). An attempt is made to focus on RCT that are more likely to be generalizable to the millions of overweight and/or sedentary people who may be prescribed weight loss and/or increased physical activity to improve an adverse lipoprotein profile.

Tables 1–3 present RCT reporting both weight and lipoprotein effects of 4 months or longer of aerobic exercise training, with or without dietary change, in normal weight (mean body mass index (BMI) < 25.0 kg·m^{-2} as the cut-point), overweight (BMI = 25.0–29.9 kg·m^{-2}), and obese (BMI > 30 kg·m^{-2}) individuals (8), respectively. Studies were included if they had an aerobic exercise and a control group or an aerobic exercise plus dietary change group and a group that made identical dietary changes, without the exercise component. The tables include the primary recruitment criteria, length of exercise training period, and mean baseline values for BMI (or weight, if neither BMI nor height were presented, using an estimated height of ≤ 178 cm, for men, and ≤ 166 cm, for women, to categorize such studies) and HDL-C, LDL-C, TG, and TC, generally estimated by averaging mean values for individual treatment groups, with an adjustment for number of subjects per group, and treatment group changes for HDL-C, LDL-C, TG, and TC, with statistical information on key between group comparisons. Despite an attempt to include all relevant trials, omissions are regrettably likely, and the reader should incorporate such trials into his/her perspective, as he/she becomes aware of them.

The tables are not segregated by lipoprotein profile; however, the reader should note that the NCEP-ATP II guidelines (7) for persons without coronary heart disease (CHD) specify the following levels for high-risk lipoproteins: TC ≥ 6.2 mmol·L^{-1} (≥ 240 mg·dL^{-1}); HDL-C < 0.9 mmol·L^{-1} (<35 mg·dL^{-1}); and LDL-C ≥ 4.1 mmol·L^{-1} (160 mg·dL^{-1}), for persons with fewer than two risk factors, and ≥ 3.4 mmol·L^{-1} (130 mg·dL^{-1}), in the presence of two or more risk factors. Risk factors included in the 1993 NCEP algorithms were: age ≥ 45 for men and ≥ 55 for women; current smoking; blood pressure = 140/90 mm Hg; low HDL-C; diabetes mellitus; and family history of premature CHD in a first degree relative. Neither sedentary status nor obesity were included as risk factors in NCEP ATP-II. HDL-C ≥ 1.6 mmol·L^{-1} (≥ 60 mg·dL^{-1}) is considered a negative risk factor. Triglycerides of 2.3–4.6 mmol (200–399 mg·dL^{-1}) are considered borderline high, with levels <2.3 being normal and >4.6 being high.

RANDOMIZED TRIALS OF LIPOPROTEIN EFFECTS OF EXERCISE IN NORMAL WEIGHT INDIVIDUALS

Table 1 presents five trials conducted in men (11,29), women (5) or both, combined (15,21), who spanned a range of body weights, from lean to obese, with a mean BMI falling in the normal weight range (<25 kg·m^{-2}). Three of the studies involved subjects with high-risk mean TC and LDL-C levels (11,15,21). Huttunen et al. (11) found no effect of 4 months of

individualized aerobic exercise (walking, jogging, swimming, skiing, or cycling), compared with controls, on weight or TC or LDL-C in 90 men with high baseline TC and LDL-C levels; however, HDL-C was significantly increased and TG levels were decreased in the exercisers.

In contrast, Wood et al. (29) reported a significant, albeit modest, weight loss (2.5 kg; $P < 0.01$) and reduction in percent body fat by hydrostatic weighing (3.8%; $P < 0.001$), in exercisers versus controls, in a study of 78 predominantly normolipidemic men who were assigned to 9–12 months of either control ($N = 32$) or 3 d·wk^{-1} of supervised walking and jogging ($N = 46$), with three choices of training distance and intensity on any given day, based on selection of group leader. There were, however, no differences between groups in HDL-C or TG changes, nor in TC, LDL-C, and apolipoprotein AI, AII, or B changes. Secondary analyses, which separated the exercisers into four "treatment-dose" groups (based on weekly mileage), found significant treatment effects for HDL-C and LDL-C. In exercisers who averaged at least 12.9 km (8 miles) per week, HDL-C increased by 0.11 mmol·L^{-1} (4.4 mg·dL^{-1}; $P < 0.05$) compared with controls. Further exploratory analyses showed that the weekly mileage correlated significantly with body fat changes, which were also significantly related to HDL-C changes.

Leighton et al. (15) reported data from 51 of 17 men and 49 women with elevated TC levels who were randomized to 26 wk of diet counseling aimed at reducing total and saturated fat and cholesterol intake, with or without a supervised exercise program, which consisted of a variety of aerobic exercises at 80% heart rate reserve (stationery bicycling, walking, jogging, stair climbing, or rowing). Although exercising dieters lost 3 kg ($P < 0.001$) and controls did not change weight, differences between groups were not reported; however, it was reported that there were no differences in HDL-C, LDL-C, TG, or TC between groups.

Duncan et al. (5) reported results from 59 of 102 premenopausal women with low-risk lipoprotein profiles, who were assigned to no exercise or to 5 d·wk^{-1} of 4.8 km·d^{-1}of walking at one of three speeds, 4.8 km·h^{-1} (strollers, S), 6.4 km·h^{-1} (brisk walkers, BW), or 8.0 km·h^{-1} (aerobic walkers, AW). Maximal oxygen uptake increased significantly ($P < 0.001$) in walkers versus controls, and in a dose-response manner (AW > BW > S), with differences between aerobic walkers and strollers reaching significance ($P < 0.001$). Neither weight changes (gain) or HDL-C increases from baseline differed between any of the exercise groups and control or other walking groups, nor were there any significant differences between groups for LDL-C, TG, or TC.

Singh et al. (21) found significant reductions in TC and LDL-C in a study involving over 450 South Asian men (90% of sample) and women who were assigned to a reduced saturated fat and cholesterol diet and moderate exercise (brisk walking and spot running) compared to diet only. This study was confounded, however, by having additional dietary goals of decreasing total energy intake and increasing the intake of vegetables, fruits, grains, and nuts in the exercising diet group. There was significant weight loss and TG reduction in the exercise plus complex diet group versus diet only; however, HDL-C changes did not differ between groups (21).

In summary, only one of the five trials in normal weight individuals showed a significant improvement in HDL-C and TG with exercise training and these differences were in men with high TC and LDL-C, but normal HDL-C and TG levels (11). The only trial that showed an improvement in LDL-C or TC with exercise involved an additional dietary component that differed from the diet only comparison group (21).

Evidence statement. The evidence is weak to support a role for aerobic exercise training in improving an adverse lipoprotein profile in normal weight men or women, largely due to an absence of clinical trials in lean individuals with low HDL-C, high TG, and/or elevated LDL-C or total cholesterol (Category 2).

RANDOMIZED TRIALS OF LIPOPROTEIN EFFECTS OF EXERCISE IN OVERWEIGHT INDIVIDUALS

Table 2 presents six large trials conducted in men (3,10,13,25) and premenopausal (32) and postmenopausal (13,25,26) women with a mean BMI of 25.0–29.9 kg·m^{-2}. The 132 overweight, premenopausal women randomized into the second Stanford Weight Control Project had relatively low-risk lipoproteins, even though their percent body fat was 35.7%, based on hydrostatic weighing and their abdomen-to-hip ratio was 0.87 (32). In these women, aerobic exercise (brisk walking), combined with a hypocaloric, low-fat diet, did not result in greater weight loss than the diet alone, although weight loss was significant ($P < 0.001$) in both dieters (5.4 kg) and dieting exercisers (6.4 kg) compared with controls, as was fat weight loss (4.5 kg and 6.0 kg, respectively), which was also not significantly different between intervention groups. HDL-C and HDL_2-C were, however, significantly increased in diet + exercise women versus diet only women (0.17 mmol·L^{-1} and 0.19 mmol·L^{-1}, respectively; $P < 0.01$), partially due to a decrease in HDL-C in women assigned to the low-fat diet without exercise, compared with controls (−0.10 mmol·L^{-1}; NS), whereas HDL-C increased in diet + exercise women versus controls (0.7 mmol·L^{-1}; NS). Diet only women also decreased apolipoprotein A-I versus control (−8.8 mg·dL^{-1}; $P < 0.05$), whereas this was increased in diet + exercise (1.9 mg·dL^{-1}; NS). Diet + exercise women also decreased TG versus control (−0.15 mmol·L^{-1}; $P < 0.05$), but there were no significant differences between dieters and dieting exercisers in reductions in TC or LDL-C, which were significantly reduced in both groups, versus controls ($P < 0.05$), as was apolipoprotein B (−5.8 and −6.0 mg·dL^{-1}; $P < 0.01$). Neither the LDL-C to HDL-C nor apolipoprotein B to AI ratios were improved in dieters compared with controls, whereas both ratios were reduced in dieting exercisers ($P < 0.05$). These results demonstrate that exercise can offset the HDL-C-lowering effect of a

TABLE 2. Randomized trials of lipoprotein effects of aerobic exercise in individuals with BMI 25.0–29.9 kg·m^{-2}.

Study	Interventions: Treatment Groups	*N* post (baseline); Sex; Basic (key) Inclusion criteria	Training Duration	Mean[a] Baseline BMI [Wt] and Lipids	Weight Change (kg) [BMI]	HDL-C (mmol·L^{-1})	LDL-C (mmol·L^{-1})	TG (mmol·L^{-1})	TC (mmol·L^{-1})
Wood et al., 1991 (32)	1: Aerobic exercise + hypocaloric low-fat diet 2: Hypocaloric low-fat diet 3: Control	112 (132); female premenopausal age 25–49 yr BMI = 24–30 kg·m^{-2} sedentary	9–12 months	BMI: 27.9 HDL: 1.5 LDL: 3.1 TG: 0.9 TC: 5.0	1: −5.1 2: −4.1 3: +1.3 1 vs 2 NS 1, 2 vs 3 $P < 0.001$	1: +0.02 2: −0.15 3: −0.05 1 vs 2 $P < 0.01$ 1 vs 3 NS	1: −0.29 2: −0.28 3: −0.03 1 vs 2 NS 1, 2 vs 3 $P < 0.001$	1: −0.02 2: +0.09 3: +0.13 1 vs 2 NS 1 vs 3 $P < 0.05$	1: −0.28 2: −0.39 3: −0.03 1 vs 2 NS 1, 2 vs 3 $P < 0.05$
King et al., 1991 (14)	1: Aerobic exercise-higher intensity, group-based 2: Aerobic exercise-higher intensity, home-based 3: Aerobic exercise-lower intensity, home-based 4: Control	167 (197); male age 50–65 yr; sedentary 131 (160); female; post-menopausal age 50–65 yr sedentary	10–12 months	Men BMI: 27.4 HDL: 1.2 LDL: 3.9 TG: 1.5 TC: Women BMI: 26.6 HDL: 1.5 LDL: 4.3 TG: 1.1 TC:	Men 1: [−0.2] 2: [−0.2] 3: [−0.9] 4: [+0.1] NS Women 1: [+0.4] 2: [+0.1] 3: [−0.6] 4: [+0.0] NS	Men 1: +0.01 2: +0.03 3: +0.05 4: +0.02 NS Women 1: +0.02 2: +0.01 3: +0.01 4: +0.01 NS	Men 1: −0.06 2: −0.19 3: −0.16 4: −0.13 NS Women 1: −0.41 2: −0.13 3: −0.15 4: −0.26 NS	Men 1: −0.06 2: −0.19 3: −0.16 4: −0.13 NS Women 1: +0.08 2: +0.05 3: +0.12 4: +0.06 NS	Not reported
Svendsen et al., 1993 (26)	1: Hypocaloric, low-fat (NUPO) diet + aerobic and anaerobic exercise 2: Hypocaloric NUPO diet 3: Control	118 (121); female post-menopausal age 45–54 yr BMI ≥ 25 kg·m^{-2}	12 wk	Wt: 77.6 HDL: 1.6 LDL: 4.6 TG: 1.3 TC: 6.9	1: −10.3 2: −9.5 3: +0.5 1 vs 2 NS 1, 2 vs 3 $P < 0.001$	1: −0.10 2: −0.05 3: −0.09 NS	1: −0.99 2: −1.08 3: −0.07 1 vs 2 NS 1, 2 vs 3 $P < 0.001$	1: −0.30 2: −0.50 3: +0.12 1 vs 2 NS 1, 2 vs 3 $P < 0.001$	1: −1.23 2: −1.36 3: −0.11 1 vs 2 NS 1, 2 vs 3 $P < 0.001$
Hellenius et al., 1993 (10)	1: Aerobic exercise 2: Low-fat diet 3: Aerobic exercise + low-fat diet 4: Control	157 (158); male age 35–60 yr; TC: 5.2–7.8 and TG ≤5.6 mmol·L^{-1}	6 months	BMI: 25.3 HDL: 1.4 LDL: 4.3 TG: 1.4 TC: 6.2	1: [−0.3] 2: [−0.3] 3: [−0.6] 4: [+0.3] 1–3 vs 4 $P < 0.01$ 1 vs 3, NS	1: +0.01 2: +0.01 3: −0.03 4: −0.02 NS	1: +0.01 2: +0.01 3: −0.03 4: −0.02 NS	1: −0.10 2: +0.03 3: −0.12 4: +0.06 NS	1: −0.12 2: −0.19 3: −0.45 4: −0.13 NS
Anderssen et al., 1995 (3)	1: Aerobic exercise 2: Hypocaloric diet 3: Aerobic exercise + diet 4: Control	209 (198 + 21); male and female; age 41–50 yr HDL-C < 1.2, LDL-C = 5.2–7.7 TG > 1.4 mmol·L^{-1} BMI ≥ 24 kg·m^{-1}, DBP = 86–99mm Hg sedentary	12 months	BMI: 28.8 HDL: 1.0 LDL: — TG: 2.3 TC: 6.3	Mean BP tertiles[b] 1: −0.03 2: −1.06 3: −1.80 4: +0.03 1 vs 4, NS 2, 3 vs 4 $P < 0.05$	Mean, BP tertiles vs control[c] 1: +0.03 2: +0.04 3: +0.12 1 vs 4, NS 2, 3 vs 4 $P < 0.05$	Not reported	Mean, BP tertiles vs control[c] 1: −0.43 2: −0.44 3: −0.79 1, 2, 3 vs 4 $P < 0.05$ 2 vs 3, NS	Mean, BP tertiles vs control[c] 1: −0.01 2: −0.08 3: −0.32 1, 2, 3 vs 4 NS 2 vs 3, NS
Stefanick et al., 1998 (25)	1: Aerobic exercise 2: Low-fat diet (NCEP Step II) 3: Aerobic exercise + low-fat (NCEP Step II) diet 4: Control	190 (197); male age 30–64 yr HDL-C ≤ 1.17, LDL-C = 3.2–4.9, TG < 4.5 mmol·L^{-1} BMI ≤34 kg·m^{-2} 177 (180); female post-menopausal age 45–64 yr HDL-C ≤ 1.55, LDL-C = 3.2–5.4, TG < 4.5 mmol·L^{-1} BMI ≤ 32 kg·m^{-2}	9–12 months	Men BMI: 27.0 HDL: 0.9 LDL: 4.0 TG: 1.9 TC: 5.8 Women BMI: 26.3 HDL: 1.2 LDL: 4.2 TG: 1.8 TC: 6.2	Men 1: −0.6 2: −2.8 3: −4.2 4: +0.5 1 vs 4 NS 2 vs 3 NS 3 vs 4, 1 $P < 0.001$ Women 1: +0.06 2: −2.7 3: −3.1 4: +0.8 1 vs 4 NS 2 vs 3 NS 3 vs 4, 1 $P < 0.001$	Men 1: + 0.03 2: −0.02 3: +0.01 4: −0.01 NS Women 1: +0.06 2: +0.01 3: −0.03 4: +0.03 NS	Men 1: −0.09 2: −0.28 3: −0.52 4: −0.12 1 vs 4 NS 2 vs 3 NS 3 vs 4, 1 $P < 0.001$ Women 1: −0.15 2: −0.19 3: −0.38 4: −0.07 1 vs 4 NS 2 vs 3 NS 3 vs 4 $P < 0.05$	Men 1: −0.15 2: −0.07 3: −0.08 4: +0.10 NS Women 1: −0.14 2: −0.05 3: −0.12 4: +0.02 NS	Men 1: −0.13 2: −0.34 3: −0.53 4: −0.10 1 vs 4 NS 2 vs 3 NS 3 vs 4, 1 $P < 0.01$ Women 1: −0.15 2: −0.20 3: −0.45 4: −0.03 1 vs 4 NS 2 vs 3 NS 3 vs 4 $P < 0.01$

[a] Mean estimated from baseline means reported for individual treatment groups.
[b] Overall mean calculated from means for treatment groups presented by baseline diastolic blood pressure tertiles (<84, 84–91, >91 mm Hg).
[c] Overall mean calculated from net-differences between treatment group and control for each diastolic blood pressure tertile.

low-fat diet in overweight, premenopausal women who have low-risk lipoproteins; however, there seems to be no further benefit to the LDL-C- or TC-lowering effect of the low-fat diet in such women.

The Stanford-Sunnyvale Health Improvement Program (SSHIP) trial (13) randomly assigned 197 men and 160 postmenopausal women, aged 50–65 yr, to control or one of three 1-yr exercise groups: high-intensity, group-based aerobic exercise, involving three 40-min endurance training sessions per week at 73–88% of peak treadmill heart rate; high-intensity, home-based aerobic exercise, the same prescription, but performed by individuals from their home;

and low-intensity, home-based aerobic exercise, five 30-min sessions per week at 60–73% maximum heart rate. The women had mean LDL-C levels of 4.3 mmol·L^{-1}, placing them at elevated CHD risk, whereas the men had LDL-C of 3.9 mmol·L^{-1}. Despite significant improvements in all three exercise groups in $\dot{V}O_{2max}$, averaging 5% increase compared with controls, there were no significant weight or body composition (by hydrostatic weighing) changes or changes in any of the lipid factors: HDL-C, LDL-C, TG or TC. At the end of the 1-yr, controlled trial, men and women in the three exercise groups were encouraged to continue their originally assigned exercise prescriptions for a 2nd yr; however, the controls were offered an exercise program and discontinued from the trial (14). At the end of this year, small, but significant HDL-C increases were reported within both home-based groups (sexes combined) compared with baseline values, particularly in the lower-intensity group, whose prescription required more frequent exercise sessions per week; however, there were no significant differences between groups. HDL-C increases were associated with decreases in waist-to-hip ratio, in both sexes ($P < 0.04$).

Svendsen et al. (26) randomized 121 postmenopausal women with a BMI $\geq$ 25 kg·m^{-2} and a high mean TC and LDL-C levels at baseline who were recruited to control or to a special low-fat, hypocaloric formula diet with or without a 12-wk aerobic exercise program (bicycling, stair walking, or treadmill running) combined with resistance weight training. There were no significant differences in weight or lipid changes between women assigned to diet only versus diet plus exercise, although both intervention groups lost significant weight and improved LDL-C, TG, and TC compared with control. Neither group changed HDL-C compared with control.

Hellenius at al (10) randomized 158 men with high initial TC levels to one of four groups: aerobic exercise (walking, jogging, etc.), a reduced-fat diet (NCEP Step 1, i.e., total fat < 30% of calories, saturated fat < 10%, monounsaturated fat 10–15%, complex carbohydrates, 50–60%, and cholesterol < 300 mg·d^{-1}; 7), aerobic exercise + the diet, or control for 6 months. All three intervention groups lost significant weight compared with control, but there were no differences among intervention groups; however, the dieting exercisers lost more fat weight (1.8 kg) than diet only, who, in turn, lost 1.2 kg lean weight, whereas dieting exercisers lost none ($P < 0.05$ vs dieters). Despite considerable weight loss in the diet and diet + exercise groups, HDL-C changes did not differ from controls; however, TG, TC, and LDL-C decreased in both dieters and dieting exercisers versus controls ($P < 0.001$) but did not differ between the weight loss groups. Therefore, the addition of aerobic and anaerobic exercise to a weight-reducing diet produced no greater lipoprotein changes than that seen with the diet alone.

The Oslo Diet and Exercise Study (ODES) was designed to investigate exercise and diet effects on blood pressure in normotensives and mild hypertensives (3). Weight changes are presented by diastolic blood pressure (DBP) tertiles (<84, 84–91, > 91 mm Hg) for each treatment group, rather than by treatment assignment, requiring some manipulation of the data to estimate an overall mean for each treatment group. Weight loss, versus control, was significant for diet only and diet + aerobic exercise, but not for exercise only; and weight loss did not appear to differ between diet only and diet + exercise. The lipid data are presented as net differences between treatment group and control within each tertile, and significant net differences are represented by equal signs of confidence limits. Values presented in Table 2 were estimated by adding reported means for each DBP tertile within a treatment group and dividing by 3. Although triglycerides, HDL-C, and TC are reported to be significantly and favorably improved in all treatment groups, presumably versus control, there appear to be no differences between intervention groups.

The Stanford Diet and Exercise for Elevated Risk (DEER) trial (25) investigated the effects of aerobic exercise (walking, jogging), a reduced-fat diet (NCEP Step 2, i.e., total fat < 30% of calories, saturated fat < 7%, and cholesterol < 200 mg·d^{-1}; 7), aerobic exercise + the diet, or control for 9–12 months on the lipoprotein profile of 197 men and 180 postmenopausal women who were recruited to have both low HDL-C and elevated LDL-C levels. Total and fat weight losses were significant in dieters and dieting exercisers in both sexes compared with controls but did not differ between diet only and diet + exercise or between exercise only and control in either sex. In DEER men, dieters lost a mean of 3.3 kg (2.1 kg fat weight), and dieting exercisers lost 4.7 kg (3.5 kg fat weight) compared with controls ($P < 0.001$), whereas weight loss in exercisers (1.2 kg, 1.0 kg fat weight) was not significant. Lean mass loss did not differ between dieters or dieting exercisers and control men but was significantly greater in both dieting groups compared with exercise only (1.3 kg for both). In DEER women, dieters lost a mean of 3.5 kg and dieting exercisers lost 3.9 kg compared with controls, whereas weight loss in exercisers (1.2 kg) was not significant. Lean mass losses were minimal and did not differ between groups. It is worth noting that although changes in caloric intake did not differ between exercise only and control groups, both exercise men and women tended to increase daily intake by about 100 kcal·d^{-1}, based on multiple unannounced diet recalls, which totaled 700 kcal·wk^{-1}, during which they were expending approximately 900-1000 kcal·wk^{-1} in walking and jogging.

HDL-C changes did not differ among DEER treatment groups in men or women (25); therefore, neither the modest weight loss achieved by the NCEP Step 2 diet, nor exercise, alone or combined with the diet, increased HDL-C in overweight men or women who would be encouraged to adopt these lifestyle changes to improve their lipoprotein profile. It is worth noting that HDL-C was more likely to be increased in the exercise only groups and that weight loss, particularly body fat loss, was strongly correlated with HDL-C increases in both exercise only men and women. In addition, there was a significant negative effect of assignment to the low-fat diet on HDL-C. Greater weight loss than

TABLE 3. Randomized trials of lipoprotein effects of aerobic exercise in individuals with BMI ≥30 kg·m^{-2}.

Study	Interventions: Treatment Groups	*N* post (baseline) Sex; Basic (key) Inclusion criteria	Training Duration	Mean[a] Initial BMI and Lipids (kg·m^{-2})	Weight Change (kg)	HDL-C (mmol·L^{-1})	LDL-C (mmol·L^{-1})	TG (mmol·L^{-1})	TC (mmol·L^{-1})
Wood et al., 1988 (31)	1: Aerobic exercise 2: Hypocaloric diet (no change in % fat calories) 3: Control	131 (155); male age 30–59 yr 120–160% "ideal" weight; sedentary	9–12 months	BMI: 30.7 HDL: 1.1 LDL: 3.8 TG: 1.5 TC: 5.7	1: −4.0 2: −7.2 3: +0.6 1, 2 vs 3 $P < 0.001$ 1 vs 2: $P < 0.01$	1: +0.11 2: +0.12 3: −0.02 1 vs 3 $P < 0.01$ 1 vs 2 NS 2 vs 3 .001	1: −0.25 2: −0.31 3: −0.21 NS	1: −0.16 2: −0.27 3: +0.08 1, 2 vs 3 $P < 0.05$ 1 vs 2: NS	1: −0.25 2: −0.36 3: −0.23 NS
Wood et al., 1991 (32)	1: Aerobic exercise + hypocaloric low-fat diet 2: Hypocaloric low-fat diet 3: Control	119 (132); male age 25–49 yr BMI = 28–34 kg·m^{-2}; sedentary	9–12 months	BMI: 30.7 HDL: 1.1 LDL: 3.6 TG: 1.4 TC: 5.4	1: −8.7 2: −5.1 3: +1.7 1 vs 2 $P < 0.01$ 1, 2 vs 3 $P < 0.001$	1: +0.14 2: +0.02 3: −0.05 1 vs 2, 3 $P < 0.01$ 2 vs 3 NS	1: −0.27 2: −0.39 3: −0.20 NS	1: −0.48 2: −0.12 3: +0.18 1 vs 2 $P < 0.05$ 1 vs 3 $P < 0.001$	1: −0.38 2: −0.42 3: −0.14 NS
Katzel et al., 1995 (12)	1: Aerobic exercise without weight change 2: Hypocaloric diet 3: Control	111 (170); male age 46–80 yr 120–160% ideal body weight; sedentary	9 months	BMI: 30.2 HDL: 0.9 LDL: 3.1 TG: 1.5 TC: 4.7	1: −0.5 2: −9.5 3: +0.2 1 vs 3: NS 1 vs 2 $P < 0.05$	1: +0.05[b] 2: +0.11[b] 3: +0.02[b] 1 vs 3: NS 1 vs 2: $P < 0.001$	1: −0.12[b] 2: −0.19[b] 3: +0.16[b] 1 vs 3: $P < 0.05$ 1 vs 2: NS	1: −0.13[b] 2: −0.27[b] 3: +0.13[b] 1 vs 3: NS 1 vs 2: NS	1: −0.09[b] 2: −0.19[b] 3: +0.29[b] 1 vs 3: $P < 0.05$ 1 vs 2 NS
Ready et al., 1995 (20)	1: Aerobic exercise (walking) 2: Control	25 (40); female post-menopausal TC: 5.9–8.0 and TG ≤4.2 mmol·L^{-1} not active	6 months	BMI: 30.6 HDL: 1.3 LDL: 4.7 TG: 1.8 TC: 6.7	1: −1.9 2: −0.6 $P < 0.05$	1: 0.00 2: −0.07 NS	1: −0.19 2: +0.08 NS	1: −0.12 2: +0.17 $P < 0.05$	1: −0.30 2: +0.01 $P < 0.05$
Fox et al., 1996 (9)	1: Aerobic + resistance exercise (200 kcal·d^{-1}) + hypocaloric diet (−500 kcal·d^{-1}) 2: Hypocaloric diet (−500 kcal·d^{-1}) 3: Hypocaloric diet (−700 kcal·d^{-1})	40 (46); female post-menopausal for ≥5 yr age ≥60 yr 120–140% ideal weight	24 wk	BMI: 30.3 HDL: 1.6 LDL: 3.8 TG: 1.6 TC: 5.8	1: −7.1 2: −6.6 3: −5.8 NS	1: −0.06 2: −0.15 3: −0.08 NS	1: −0.11 2: +0.13 3: +0.06 NS 1: −0.11 2: +0.13 3: +0.06 NS Triacyglyc.	1: −0.08 2: −0.12 3: −0.03 NS	

[a] Mean estimated form baseline means reported for individual treatment groups.
[b] Data interpreted from graphs.

that achieved in this study may be needed to overcome the HDL-C-lowering effect of the low-fat diet.

Compared with controls, LDL-C reductions were not significant in DEER men or women who adopted the NCEP Step 2 diet without increasing activity level or in men or women who increased their exercise level without altering their diet; however, significant LDL-C reductions were seen in both men and women assigned to the diet plus aerobic exercise (25). Whether slightly greater weight loss in the diet + exercise group played a role is unclear; however, this finding provides strong support for the addition of exercise, whatever its role on weight loss, for the management of lipoproteins.

In summary, none of these major trials of overweight men and women showed any benefit of aerobic exercise alone, compared with control, or of the addition of aerobic exercise to a low-fat diet, compared to the diet alone, on HDL-C, LDL-C, TG, or TC; however, the combination of diet and aerobic exercise resulted in significant lipoprotein improvements compared with controls in three of four trials that examined this question. Furthermore, a low-fat diet alone failed to reduce LDL-C in men (10,25) or postmenopausal women (25) who had high LDL-C levels; however, the addition of exercise to the diet was effective in lowering LDL-C in both sexes in a trial that involved men and women who also had low HDL-C (25). Finally, only one of these trials showed significant weight loss with aerobic exercise alone (10); none showed greater weight loss by adding exercise to diet versus diet only, whereas this combination resulted in significant weight loss versus control in all four trials which examined this question.

Evidence statement. Aerobic exercise training is unlikely to improve the lipoprotein profile in overweight men or women, unless it is combined with a hypocaloric diet; however, if the caloric reduction is achieved primarily by reducing saturated fat, benefits to HDL-C are likely to be reduced or eliminated, whereas improvements to LDL-C will be improved (Category 1). The addition of exercise to a weight-reducing and/or reduced-fat diet produces only a modest benefit to HDL-C, TG, and/or LDL-C compared with what is achieved by the diet alone (Category 1).

RANDOMIZED TRIALS OF LIPOPROTEIN EFFECTS OF EXERCISE IN OBESE (BMI ≥ 30 kg·m^{-2}) MEN AND WOMEN

Table 3 presents three relatively large exercise training trials of obese men (12,31,32), one of which involves men with low HDL-C, but who also have low-risk LDL-C and

TC levels (12), and two medium-sized trials of obese postmenopausal women (9,20), one of which has women with high TC levels (20). No other major trials were found; therefore, data on the effects of exercise in obese individuals with adverse lipoproteins are extremely limited, particularly in premenopausal women.

The first Stanford Weight Control Project, SWCP-I (31), involving men recruited to be 20–60% above ideal body weight, was designed to test whether weight loss by aerobic exercise (walking, jogging), with diet composition and caloric intake held constant, differed from weight loss by caloric restriction, with no change in diet composition or activity level, in its effects on HDL-C and other lipoproteins. Compared with controls, total and fat weight losses were significantly greater ($P < 0.001$) in both dieters (−7.8 kg and −5.6 kg, respectively) and exercisers (−4.6 kg and −3.8 kg); whereas, lean mass loss was greater only in dieters (−2.1 kg; $P < 0.001$), who lost more lean weight than exercisers (−0.7 kg; $P < 0.01$). Fat weight loss did not differ between dieters and exercisers, whereas dieters lost significantly more total weight than exercisers ($P < 0.05$). HDL-C was elevated in both dieters (0.11 mmol·L^{-1}) and exercisers (0.12 mmol·L^{-1}) compared with controls ($P < 0.01$), whereas TG was reduced (−0.27 and −0.16 mmol·L^{-1}; $P < 0.05$), but these changes did not differ between the two weight loss groups nor did LDL-C changes, which were no different from control for either weight loss group. Thus, weight loss achieved by caloric restriction alone, with no change in the percent of calories from fat, or by exercise with no dietary changes, improved HDL-C and TG levels but not LDL-C, and there was no difference with weight loss by exercise or caloric restriction. This study did demonstrate that exercise was effective in producing weight loss in obese men, provided attention was given to preventing dietary changes (which was generally a plea to prevent increasing calories, as opposed to decreasing food intake) and that exercise-induced weight loss was effective in raising HDL-C and reducing TG in such men.

The second Stanford Weight Control Project, SWCP-II (32), compared aerobic exercise (walking, jogging) combined with a low-fat diet (NCEP Step 1; 7) with the diet only and controls in 132 men, aged 25–49 yr, who were recruited to have a BMI of 28–34 kg·m^{-2}. [These men had a mean of 28.2% body fat, by hydrostatic weighing compared with 35.7% body fat in SWCP-II women, who were recruited to have BMI of 24–30 kg·m^{-2} and are described in the previous section. Both sexes had a mean fat mass of 27 kg.] In men, weight loss was significant in both diet only (−6.8 kg) and diet + exercise (−10.4 kg) compared with controls ($P < 0.001$), as was fat weight loss (−5.5 kg and −9.0 kg, respectively). Compared with men assigned to diet alone, dieting exercisers lost more total weight (−2.4 kg; $P < 0.01$) and fat weight (−3.5 kg: $P < 0.001$); in fact, the addition of exercise to the diet increased loss of body fat by 81%. Waist-to-hip girth was significantly reduced in both diet only and diet + exercise versus control ($P < 0.001$), but the decrease was significantly greater in dieting exercisers compared with diet only ($P < 0.001$).

Compared with controls, HDL-C was significantly increased in dieting exercisers (0.19 mmol·L^{-1}), as was apolipoprotein A-I (7.2 mg·dL^{-1}), whereas HDL-C was not significantly increased in dieters (0.7 mmol·L^{-1}); therefore, HDL-C increases in dieting exercisers were also significant compared with diet only ($P < 0.01$). Diet + exercise men decreased TG versus control (−0.66 mmol·L^{-1}; $P < 0.001$) and diet only men (-0.36 mmol·L^{-1}; $P < 0.05$). In contrast, LDL-C decreases were not significant in either dieters or dieting exercisers compared with controls; however, apolipoprotein B was reduced in both (−5.8 mg·dL^{-1} and −6.0 mg·dL^{-1}; $P < 0.01$). Reductions in the LDL-C to HDL-C ratio were significant in both diet only and diet + exercise men versus control ($P < 0.05$) but did not differ between weight loss groups. There was also a significant reduction in the ratio of apolipoprotein B to apolipoprotein AI in both dieting groups versus control ($P < 0.001$), and this reduction was greater in the diet plus exercise men versus diet only ($P < 0.05$).

Katzel and colleagues (12) specifically compared the effects of weight loss (achieved by diet, without a change in activity level) with aerobic exercise, without weight loss, in 111 of 170 obese (120–160% of ideal body weight) men, aged 46–80 yr, who were randomized to 9 months of control, weight loss by diet, or aerobic exercise, consisting of treadmill and cycle ergometer workouts. Before baseline testing, all three groups were instructed for 3 months on an isoenergetic reduced-fat (NCEP Step 1) diet, which may explain the low mean HDL-C, LDL-C, and TC levels in all men at baseline. Men in both the aerobic exercise and control groups were encouraged to continue a low-fat diet, without losing weight, throughout the trial; therefore, exercisers presumably increased their intake of low-fat foods. Men assigned to weight loss lost about 9.5 kg, 75% of which was fat mass, and did not change $\dot{V}O_{2max}$, whereas exercisers did not change average weight but reduced percent body fat by 0.8% ($P < 0.005$). Compared with controls, HDL-C was significantly increased in the weight loss group (0.12 mmol·L^{-1}; $P < 0.01$) but not in the exercise group, whereas TG, TC and LDL-C were decreased ($P < 0.05$) in both the weight loss and exercise groups, and these changes did not differ between intervention groups. It is unclear whether the reduction in LDL-C and TC in exercisers was due to the increased exercise or increased intake of low-fat foods to maintain weight.

A comparison of the amount of weight lost by men in the two Stanford weight loss studies (31,32) and the Katzel study (12) raises some interesting issues. In SWCP-I (31), weight loss by reducing portion sizes without changing diet composition resulted in total and fat body weight losses of 7.8 and 6.2 kg, respectively, whereas those who lost weight by exercise only lost 4.6 kg and 4.4 kg. In SWCP-II (32), reducing calories by targeting dietary fat resulted in total and fat weight losses of 6.8 and 5.5 kg, and the addition of aerobic exercise increased this to 10.4 kg and 9.0 kg. Men who completed 1-yr tests in the weight loss group in the Katzel study (12) lost 9.5 kg and about 7 kg. HDL-C was similarly raised in SWCP-I by diet- and exercise-induced

TABLE 4. Randomized trials of lipoprotein effects of resistance exercise.

Study	Interventions: Treatment Groups	*N* post (baseline) Sex; Basic (key) Inclusion criteria	Training Duration	Mean[a] Initial BMI and Lipids ($kg \cdot m^{-2}$)	Weight Change (kg)	HDL-C ($mmol \cdot L^{-1}$)	LDL-C ($mmol \cdot L^{-1}$)	TG ($mmol \cdot L^{-1}$)	TC ($mmol \cdot L^{-1}$)
Manning et al., 1991 (17)	1: Strength training 2: Control	22 (24); female age 22–57 yrs obese; sedentary	12 wk	BMI: 32.1 HDL: 1.6 LDL: 3.1 TG: 1.2 TC: 5.2	1: +1.4 2: +0.4 NS	1: −0.10 2: −0.03 NS	1: −0.08 2: +0.16 NS	1: +0.17 2: +0.21 NS	1: −0.05 2: +0.21 NS
Boyden et al., 1993 (4)	1: Resistance training 2: Control	103 (88); female premenopausal age 28–39 yrs	5 months	BMI: 22.4 HDL: 1.4 LDL: 3.5 TG: 0.9 TC: 4.7	1: +0.4 2: 0.0 NS	1: +0.02 2: +0.02 NS	1: −0.36 2: −0.07 $P < 0.04$	1: −0.05 2: −0.03 NS	1: −0.33 2: −0.09 $P < 0.02$
Andersen et al., 1995 (1)	Hypocaloric (liquid) diet + 1: Aerobic exercise (AE) 2: Strength training (ST) 3: AE + ST 4: No exercise	53 (66); female mean age 43.6 obese	48 wk	BMI: 36.2 HDL: 1.4 LDL: 3.5 TG: 1.6 TC: 5.7	1: −13.4 2: −17.9 3: −15.3 4: −12.9 NS	Data not shown NS	Data not shown NS	Data not shown NS	1: −0.43 2: −0.93 3: −0.62 4: −1.07 NS

[a] Mean estimated from baseline means reported for individual treatment groups.

weight loss, even though weight loss was greater with diet. Weight loss accompanying the SWCP-II low-fat diet was similar in magnitude to that from the SWCP-I diet and greater than that from the SWCP-I exercise, yet HDL-C was not elevated, unless exercise was added and/or greater weight loss was achieved, presumably because of a HDL-C-lowering effect of the low fat diet. Together, these studies suggest a HDL-C raising effect of weight loss and exercise, and a HDL-lowering effect of a low-fat diet, which interact to determine the HDL-C change. The greater weight loss achieved on the Katzel low-fat diet could explain the HDL-C increase in that study, or this may be due to the initiation of the low-fat diet 3 months before baseline measures and intervention. Similar issues are likely to exist for women; however, women may be more susceptible to the HDL-C-lowering effect of a low-fat and less responsive to the HDL-raising effect of weight loss or exercise, based on very limited data, such as from SWCP-II (32). Higher initial HDL-C levels may also contribute to sex differences in responses to exercise, as might sex differences in ability to lose weight with exercise (33).

Table 3 also presents two relatively small, 6-month exercise studies of obese postmenopausal women (9,20). [Studies with ≤ 10 women per group were not included in this review.] In women with high LDL-C and TC, Ready et al. (20) reported greater, albeit modest, weight loss (−1.3 kg) in those assigned to walking ($N = 15$) versus control ($N = 10$; $P < 0.05$) and significant ($P < 0.05$) decreases in TG and TC in walkers but no differences between groups in HDL-C or LDL-C changes. Fox et al. (9) found no weight loss or lipid change differences between women assigned to weight loss by either of two hypocaloric diets (−500 $kcal \cdot d^{-1}$ vs −700 $kcal \cdot d^{-1}$) or by the −500 $kcal \cdot d^{-1}$ diet combined with a 200 $kcal \cdot d^{-1}$ energy deficit arising from a combination of aerobic exercise (1 h of walking 3 $d \cdot wk^{-1}$) and resistance training (at 80% of one-repetition max capacity, 2 $d \cdot wk^{-1}$).

In summary, three relatively large RCT of obese men have provided evidence that weight loss improves HDL-C and TG; however, none have been restricted to men with low HDL-C or elevated TG. One of these studies involved weight loss achieved solely by exercise with no dietary change (31). The only trial that showed LDL-C improvements with aerobic exercise required an increase in low-fat foods to maintain weight (12), whereas neither trial that promoted exercise-induced weight loss (31,32) provided evidence that weight loss or exercise reduce LDL-C. Evidence that weight loss and/or aerobic exercise improves dyslipoproteinemias in obese women is even weaker).

Evidence statement. Weight loss achieved by exercise with no dietary change (or by caloric restriction with no exercise change) is likely to improve HDL-C and TG in obese men with normal lipoproteins, whereas the evidence that exercise or weight loss improve these lipids in men with dyslipidemias is weak (Category 1). Even less evidence exists to support a role for aerobic exercise in treating lipid disorders in obese women.

Table 4 presents three studies involving resistance exercise versus control, two in obese women (1,17) and one in normal weight women (4), none of which involved women with dyslipoproteinemias. [The author did not find RCT of lipoprotein effects of resistance training in lean or obese men, regardless of lipoprotein status.] Andersen et al. (1) randomized women who were consuming a hypocaloric (formula) diet to aerobic exercise, resistance training, the combination of these two modes of exercise, or no exercise. All four groups lost substantial weight, with mean total losses of 13–18 kg; however, there were no differences between groups in weight loss or HDL-C, LDL-C, TG, or TC changes. Manning et al. (17) also found no differences between obese women randomized to 12 wk of strength training versus control for weight gain (+1.4 vs 0.4) or lipid changes. On the other hand, Boyden et al. (4) reported significant reductions in LDL-C and TC in lean, premenopausal women with low-risk lipoproteins who were assigned to resistance training compared with controls; however, groups did not differ in weight changes or HDL-C or TG.

TABLE 5. Randomized trials of lipoprotein effects of lifestyle versus structured aerobic exercise.

Study	Interventions: Treatment Groups	*N* post (baseline) Sex; Basic (key) Inclusion criteria	Training Duration	Mean[a] Initial BMI and Lipids ($kg \cdot m^{-2}$)	Weight Change (kg)	HDL-C ($mmol \cdot L^{-1}$)	LDL-C ($mmol \cdot L^{-1}$)	TG ($mmol \cdot L^{-1}$)	TC ($mmol \cdot L^{-1}$)
Dunn et al., 1999 (6)	1: Aerobic exercise (structured program) 2: Lifestyle (accumulate 30 min exercise most days)	190 (116 + 119); male and female age 35–60 yr; living w/in 16 km of study center; sedentary	24 months	BMI: 28.2 HDL: 1.3 LDL: 3.5 TG: 1.9 TC: 5.6	1: +0.7 2: −0.1 NS	1: −0.05 2: −0.03 NS	1: −0.12 2: −0.04 NS	1: +0.07 2: −0.11 $P < 0.05$	1: −0.13 2: −0.11 NS
Andersen et al., 1999 (2)	Behavioral-diet wt loss+ 1: Aerobic exercise (struct) 2: Lifestyle (Daily Living)	38 (40); female mean age 42.9; ≥15 kg ideal wt	16 wk	BMI: 31.9 HDL: 1.3 LDL: 3.5 TG: 1.2 TC: 5.4	1: −8.3 2: −7.9 NS	1: +0.13 2: +0.14 NS	1: −0.38 2: −0.31 NS	1: −0.21 2: −0.28 $P < 0.05$	1: −0.60 2: −0.57 NS

[a] Mean estimated from baseline means reported for individual treatment groups.

Evidence statement. There is essentially no data, especially in men, on effects of resistance exercise (strength training) on obesity-related dyslipoproteinemias.

LIFESTYLE (DAILY ENERGY EXPENDITURE) VERSUS STRUCTURED EXERCISE

There has been considerable interest in recent years in the potential benefit of increasing energy expenditure through daily activities, rather than through a structured exercise program. Table 5 presents two randomized trials that compared these two exercise modes and reported weight and lipoprotein outcomes. Dunn et al. (6) combined results from 190 (of 116 plus 119) overweight men and women (mean = 28.2 $kg \cdot m^{-2}$) who were randomly assigned for 2 yr to "lifestyle" (accumulation of at least 30 min of moderate activity exercise, adapted to each individual, on most days of the week) or a structured aerobic exercise program at 50–85% of maximal aerobic power. The aerobic exercise group actually gained 0.7 kg over the 2-yr period, but this did not differ from controls (−0.1 kg). There were no differences between groups in HDL-C, LDL-C, or TC, which started at low-risk levels; however, TG increased in the structured exercise group (+0.07) and decreased in the lifestyle group (−0.11), resulting in a significant difference between groups ($P < 0.05$). Andersen et al. (2) randomized obese (≥ 15 plus kg ideal body weight) women to the same behavior + dietary weight loss program combined with either structured aerobic exercise or lifestyle for 4 months and found no differences between groups in weight or lipid changes.

Evidence statement. Effects of energy expenditure through daily activities on lipoproteins has not been shown to differ from effects of structured physical activity; however, the data are very limited at this time.

RESEARCH PRIORITIES

To determine whether physical activity can prevent or treat lipoprotein disorders associated with obesity, studies need to be conducted in overweight and obese individuals who have specific adverse lipoprotein profiles. There are very few well-designed randomized clinical trials of exercise effects on lipoproteins in obese individuals, particularly in women, and those that exist are limited to the lower end of the class I obesity range, mean BMI < 31 $kg \cdot m^{-2}$. Furthermore, despite a growing consensus that individuals with central obesity are more likely to have a higher prevalence of obesity-related CHD risk factors, no randomized, controlled trials were found which specifically evaluated the effect of physical activity on overweight or obese men or women with central obesity. This might be of particular importance in women, for whom most data are derived from women with the more common gynoid obesity pattern, who may differ in their ability to lose weight or improve lipoproteins versus those with android obesity.

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Effects of physical activity on insulin action and glucose tolerance in obesity

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ABSTRACT

KELLEY, D. E. and B. H. GOODPASTER. Effects of physical activity on insulin action and glucose tolerance in obesity. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S619–S623, 1999. **Purpose:** The purpose of this paper is to examine the effect of physical activity on glucose tolerance in relation to obesity. **Methods:** We reviewed current literature, with particular emphasis on randomized clinical trials, to prepare an evidence-based evaluation of the effects of physical activity on glucose intolerance in obesity. **Results:** This literature review indicates that physical activity has favorable effects on reducing insulin resistance in obesity and among patients with type 2 diabetes mellitus. Improvement in glucose tolerance is less consistently observed and is related to intensity of exercise, collateral changes in adiposity, the interval between exercise and testing of glucose tolerance, and the baseline severity of glucose intolerance. **Conclusion:** A review of currently published clinical trial data supports the conclusion that physical activity can reduce insulin resistance and improve glucose intolerance in obesity. **Key Words:** DIABETES MELLITUS, OBESITY, GLUCOSE TOLERANCE, EXERCISE

The purpose of this paper is to review the recent published literature concerning effects of physical activity on insulin sensitivity and or glucose tolerance in relation to obesity. Insulin resistance and glucose intolerance are associated with obesity (11,38). Physical activity is commonly prescribed in conjunction with nutritional recommendations to achieve and maintain weight loss and thereby improve glucose homeostasis. In the review of the literature, the emphasis is placed on data from randomized clinical trials (RCT), with particular emphasis upon studies in which exercise was a direct and monitored intervention. Moreover, studies that examined exercise alone rather than in combination with weight loss were given emphasis. Several important and recent studies have examined the combined effects of exercise and weight loss on insulin action and glucose tolerance (9,50). However, since this review is directed at the effects of physical activity on glucose homeostasis independent of weight loss, the combined intervention data are less helpful to address this issue. Consideration was also given to modality of physical activity (e.g., aerobic vs resistance), intensity of effort, frequency and duration of exercise sessions, and overall length of the period of intervention. In addition to the principal focus, as defined above, recent important epidemiological data on the effects of physical activity upon glucose intolerance are also reviewed along with data on the impact of physical activity in patients with type 2 diabetes mellitus (DM).

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In preparing this review, the guidelines set forth for preparation of the recent NIH consensus statement on prevention of obesity (38) have been followed, with the aim of providing a single evidence-based statement. The greatest emphasis has been given to the studies of long duration, but because of a relative lack of such data we have also included studies less than 4 months in duration, weighting these accordingly. Two recently published randomized controlled trials (RCT) of relatively long duration are summarized in Table 1, along with two additional RCT, although the latter examined the effect of physical activity in lean subjects. We have also summarized within Table 2 data from seven additional nonrandomized studies conducted without control groups but that nevertheless provide important information regarding various aspects of the effects of physical activity on glucose tolerance.

CURRENT STATUS OF KNOWLEDGE

Evidence Statement: An increase in physical activity improves insulin action in obesity with or without concomitant changes in weight and/or body composition. (Evidence Category B/C). The RCT by Dengel et al. (10) and by Katzel et al. (30) examined the effect of exercise training on insulin action in obese men. These research subjects, previously sedentary, performed moderate aerobic exercise over a 9- to 10-month period. Exercise was performed during three sessions weekly at a moderate intensity. At completion, the exercise intervention groups achieved an increase in aerobic capacity but had not changed weight (Table 1). Neither group of subjects were glucose intolerant before intervention, and

TABLE 1. Effects of exercise training on glucose tolerance in obesity: evidence from randomized controlled trials.

Citation	Subjects	Treatment	Training Duration	Exercise Intensity/Duration	Δ GT	Δ Body Composition
Obese subjects						
Dengel et al. 1996 (10)	Men, 60 ± 2 yr, 31 ± 1% fat	Aerobic exercise	10 months 3 ×/wk	50–60% HRR 15–30 min	Fasting Ins, −8% Ins AUC, −19%	NC
Katzel et al. 1995 (30)	Men, 61 ± 1 yr, BMI = 30 ± 1	Aerobic exercise	9 months 3 ×/wk	50–80% HRR 30–45 min	Fasting Ins, NC Gluc AUC, NC Ins AUC, −17%	NC
Lean subjects Eriksson et al. 1998 (14)	Men & women, 60 5 yr, BMI = 26 ± 0.6 w/IGT	Aerobic exercise	6 months 3 ×/wk	60% HR_{max} 40–70 min	Fasting Ins, NC Ins secretion during IVGTT, −	NC
Hellénius et al. 1995 (18)	Men, 46 ± 5 yr, BMI = 25.3 ± 2.9	Aerobic exercise	6 months 2–3 ×/wk	60–80% HR_{max} 39±50 min	Fasting Ins, −14% Ins AUC, −8.5% Gluc AUC, NC	BMI decreased

BMI, body mass index (kg·m^2), IGT, impaired glucose tolerance, NIDDM, non-insulin-dependent diabetes mellitus, A-A, African-American; HRR, heart rate reserve; Ins, insulin; Gluc, glucose; AUC, Area under the curve from OGTT; K_G, glucose effectiveness; NC, no change from exercise training.

glucose response during an oral glucose tolerance test (OGTT) at the completion of the intervention had not changed as a result of exercise training. However, in both trials, insulin levels during the OGTT were reduced, indicating improved insulin action as a result of exercise training. Similarly, the two RCT that examined lean subjects also found that exercise training improved insulin action regardless of the absence of weight loss (14,18). Of note, and to be addressed more fully later, postintervention glucose tolerance in these studies was assessed within 2–3 d following the last exercise session. Summarizing the results from these RCT, an increase in physical activity by aerobic exercise improves insulin action even without weight loss.

The majority of the eight nonrandomized controlled trials (Table 2) that were reviewed also yielded results consistent with an effect of physical activity to improve insulin action (23–37,39–44). In the majority of these studies, the outcome measures were glucose and insulin levels during an OGTT. The designs of these studies were diverse in regard to the overall duration of the exercise program and in regard to the intensity, frequency, modality, and length of individual exercise sessions. However, each study did attempt to prevent weight loss during exercise training. On first appraisal, the impact of exercise training on glucose tolerance was not consistent among these nonrandomized studies. As shown in Table 2, four studies reported reduced glucose levels during OGTT as a result of exercise training (4,12,23,44) while the remainder observed no change in glucose tolerance (6,29,47,48). Part of this disparity in results seemed to be a function of the degree of glucose intolerance of the subjects before intervention. Improvement was generally found in those individuals who had impaired glucose tolerance but not necessarily among those individuals with established type 2 DM, as will be discussed later in this paper in more detail.

One of the key issues brought out in several studies (4,6,29,44) summarized within Table 2 is that exercise training can elicit improvement in insulin action in obesity within 1 wk of intervention. This finding indicates that physical activity can elicit improvements in insulin action

TABLE 2. Effects of exercise training on glucose tolerance in obesity: evidence from nonrandomized trials.

Citation	Subjects	Treatment	Training Duration	Exercise Intensity/ Duration	Δ GT	Δ Body Composition
Hughes et al. 1995 (22)	Men & women, 66 ± 1.5 yr, BMI = 25.4, 36.9% fat	Aerobic exercise	3 months 4 ×/wk	50–75% HRR	Ins AUC, decreased	−1.4 kg NC in % body fat
Angelopoulos et al. 1998 (4)	Men, 37 ± 2 yr, BMI = 34.4 ± 3,	Aerobic exercise	10 d consecutively	75% HR_{max}/40 min	Ins AUC, −19% Gluc AUC, −14%	NC
Kang et al. 1996 (29)	Men, 43 ± 3 yr, BMI = 31.7 ± 1.8	Aerobic exercise	7 d consecutively 70% $\dot{V}O_{2peak}$/50 min	50% $\dot{V}O_{2peak}$/70 min Gluc AUC, NC	Gluc & Ins AUC, NC	NC
DiPietro et al. 1998 (12)	Men & women, 73 ± 1 yr	Aerobic exercise	4 months 4 ×/wk	65% HR_{max}/60 min	Ins AUC, −22% Gluc AUC, −25%	NC
Brown et al. 1997 (6)	A-A women, 51 ± 8 yr, 44 ± 2% fat	Aerobic exercise	7 d consecutively	65% HR 50 min	Fasting Ins, −32% K_G, NC	NC
Segal et al. 1991 (47)	Men, 31 ± 2 yr, 32.5 ± 1.5% fat	Aerobic exercise	3 months 4 ×/wk	Ventiliatory threshold 60 min	Fasting ins, NC Gluc AUC, NC Ins AUC, NC	NC
Smutok et al. 1994 (48)	Men w/IGT, NIDDM, 53 ± 6 yr, BMI = 28.7, 27 ± 5% fat	Aerobic exercise	5 months 3 ×/wk	50–80% HRR	Ins AUC, −21% 30 min	NC
Smutok et al. 1994 (48)	Men w/IGT, NIDDM, 52 ± 11 yr, BMI = 29.4, 26 ± 5% fat	Strength exercise	5 months 3 ×/wk	Moderate intensity	Ins AUC, −22%	NC
Rogers et al. 1988 (44)	Men with mild NIDDM, 53 ± 3 y, BMI = 30.3	Aerobic exercise	7 d consecutively	68% $\dot{V}O_{2max}$ 50–60 min	Gluc AUC, −36% Ins AUC, −32%	NC NC

BMI, body mass index (kg · m^{-2}); IGT, impaired glucose tolerance; NIDDM, non-indulin-dependent diabetes mellitus; A-A, African-American; HRR, heart rate reserve; Ins, insulin; Gluc, glucose; AUC, area under curve from OGTT; K_G, glucose effectiveness; NC, no change from exercise training.

without concomitant weight loss and without necessarily evoking a true training adaptation response within skeletal muscle morphology. The finding further raises the question of whether improvement in insulin action during longer duration of exercise intervention programs represents a "training adaptation" (45), the impact of the most recent single bout of exercise, or an interaction of these two processes. Several studies addressed, directly or indirectly, the issue concerning the hiatus from the last exercise session to the determination of insulin action, and in this manner they addressed the interaction between potential training adaptations and insulin action. In the study by Segal et al. (47), despite a fairly robust effect upon fitness, no change in insulin action, as determined during insulin infusion studies, was observed following 12 wk of aerobic exercise training. In that study changes of weight were prevented and post-training assessments of insulin action were performed 4–5 d following the last exercise session. A recent study by King et al. (32) illustrates that beneficial effects of exercise training on insulin action decline within 3–4 d following an exercise session. The findings of Segal et al. (47) seem to indicate that acute effects of exercise are as important, and perhaps more important, than chronic "training" effects on insulin action and also suggest that less effect on insulin action will be obtained in the absence of weight loss. From a clinical perspective, these data reinforce the importance of sustaining a regular frequency of exercise to maintain a beneficial effect on insulin action.

While the majority of studies in this review have focused on the effects of aerobic exercise, one study compared aerobic versus strength training on insulin action (48). In that study it was found that these two exercise modalities resulted in similar improvements in insulin action. This suggests that improvements in insulin action from exercise training can occur without concurrent improvements in cardiorespiratory fitness. This evidence is bolstered by results from shorter-term exercise training interventions in which no improvements in cardiorespiratory fitness were observed (4,6,29,44).

The exercise intensity and duration of the training bouts may also influence improvements in insulin action. In a study determining the effects of exercise intensity and duration on insulin action, Kang et al. (29) found that higher intensity exercise resulted in an improvement in insulin action following seven consecutive days of exercise, while isocaloric bouts of exercise at lesser intensity, despite the equivalent energy expenditure, did not improve insulin action. The higher intensity exercise was associated with a greater utilization of muscle glycogen (28), suggesting that the pattern of substrate utilization and, in particular, the depletion of muscle glycogen may regulate the short-term effects of exercise on insulin action, as has been suggested (26). Another factor that may alter insulin action is the macronutrient composition of the diet. Indeed, many of the studies we have presented in this review have altered the caloric intake and/or the diet composition to maintain body weight during the exercise intervention. However, in the study by Hughes et al. (23), improvements resulting from exercise training were shown to be independent of an increased carbohydrate intake.

There are strong epidemiological data that a program of regular exercise can reduce the risk for developing type 2 DM (19,33,34). These favorable effects appear to be most evident among those at highest risk, although recent weight gain will lessen the protective effect of exercise (19). In a large prospective randomized trial, 577 individuals with impaired glucose tolerance identified from screening a population of more than 110,000 men and women found that exercise lowered the risk of conversion to type 2 diabetes during the ensuing 6 yr (41). Positive effects were also observed for diet alone, or in combination with exercise. Within a trial of this size and design, it is difficult to ascertain clearly the amount of exercise actually performed or the extent of cross-over between arms of treatment. Nevertheless, these data do suggest the potential effectiveness of lifestyle interventions of exercise and diet to lessen the risk of type 2 DM among individuals with IGT. A similar pattern of findings was observed among middle-aged British men (42), among middle-aged Japanese men (49), and among African-Americans (27). Follow-up data from the Malmo preventive trial in men with IGT indicate favorable effects on mortality rates among those engaged in long-term interventions of diet and physical activity (15). Data from the Insulin Resistance Atherosclerosis Study (IRAS) substantiate a link between physical activity and insulin sensitivity, and between the latter and risk for progression of atherosclerosis (36). Several important studies concerning the mechanisms of these favorable effects of exercise upon insulin resistance in skeletal muscle have been published in recent years (21,22,26). Using an NMR method, Perseghian et al. (42) observed that following glycogen depleting exercise previous defects in insulin-stimulated glucose transport and phosphorylation were improved within skeletal muscle of insulin-resistant adult offspring of parents with type 2 DM.

While the epidemiological data indicate that physical activity can reduce the risk of type 2 DM (19,27,33,34,41,42,49), the capacity for physical activity to improve metabolic control in the setting of established Type 2 DM is a less consistent finding. There are several recent and excellent reviews on the effect of exercise on glucose control in patients with type 2 DM (1,2,16,26,37,51), that complement earlier important studies (20,46). In general, exercise intervention results in a modest improvement of glucose control, a reduction in need for medication, or some combination of effects. In many patients improvements in glucose tolerance may not occur following exercise, and this is likely a result of a reduced capacity for insulin secretion, although other factors may also contribute. Some of the earlier studies emphasized the impact of relatively high intensity exercise to reverse or improve glucose tolerance in patients with type 2 DM and relatively mild hyperglycemia (20). More recently, several studies indicate the efficacy of milder intensity exercise to ameliorate insulin resistance in type 2 DM (5,7,12,39,52). Also, several studies indicate a favorable effect of circuit resistance training on insulin sensitivity in type 2 DM (14,25). While the insulin resistance of skeletal muscle

in patients with type 2 DM is widely recognized, several recent studies find that during exercise rates of glucose utilization are moderately increased compared with nondiabetic subjects exercising at the same intensity (8,17,28,35).

RESEARCH PRIORITIES

This review has outlined recent research on the effects of physical activity upon glucose tolerance and insulin sensitivity in obesity. The evidence suggests that exercise training does have a beneficial effect on glucose and insulin homeostasis and, most particularly, on insulin resistance. However, several important questions remain. There are not enough data concerning exercise and glucose homeostasis in women and minorities. More information would be useful to examine the interaction of body composition changes and improvements in insulin action as a result of exercise training. Given the strong association between regional adipose tissue depots, fatty acids, and insulin resistance of obesity, this area deserves further investigation with respect to exercise. For example, King et al. (32) showed that insulin action immediately following exercise was impaired because of elevated levels of circulating fatty acids, which have been shown to induce insulin resistance (31). It has also been recently demonstrated that an accumulation of intramuscular triglyceride is associated with insulin resistance in obesity (40). In young lean individuals, exercise training improves the ability to oxidize intramuscular fatty acids (24); however, data regarding the effects of exercise training on substrate selection in obesity and how this may influence insulin action are scarce. Finally, and of greatest importance is how much, at what intensity, and how often should exercise be done to obtain favorable effects on health (3,13).

Therefore, several meaningful questions remain regarding exercise training, insulin action, and obesity:

1. What is the dose-response association between the length of the intervention, the exercise training intensity, and its frequency and duration with effects on insulin action and glucose homeostasis; in other words how much and what type of exercise is needed?
2. How do the improvements in glucose and insulin homeostasis resulting from exercise compare with those induced by diet alone or diet in combination with exercise?
3. What are the optimal yet safest means to reverse impaired glucose tolerance or improve type 2 DM using exercise interventions?
4. What is the interaction between changes in body composition and regional fat distribution and the effects of exercise on glucose homeostasis?
5. What are the effects of exercise on disordered fatty acid metabolism in the setting of insulin resistance and what are the interactive effects of glucose and fatty acid metabolism in response to exercise training?
6. Will a program of regular exercise reduce or delay mortality and morbidity from coronary heart disease (CHD) endpoints (MI, CABG, angioplasty, etc.) in the settings of obesity and glucose intolerance and diabetes and, if so, how much exercise is needed to achieve these effects?

In summary, our assessment of the recent literature suggests that exercise improves glucose homeostasis and insulin action independent of body weight changes. Our perception, however, is that on the whole this body of data is sparse and requires further corroboration. We believe that further studies are required to examine the mechanisms of an altered substrate metabolism in obesity and how these may relate to improved health and prevention of metabolic disease.

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Physical activity in the prevention and treatment of hypertension in the obese

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ABSTRACT

FAGARD, R. H. Physical activity in the prevention and treatment of hypertension in the obese. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S624–S630, 1999. **Purpose:** The purpose of this paper was to assess the value of physical exercise in the prevention and treatment of hypertension with particular attention to possible interactions with relative weight. **Methods:** We describe epidemiological studies and report meta-analyses of randomized intervention trials, i.e., randomized controlled trials on dynamic physical training and randomized comparative trials of exercise and diet. **Results:** Epidemiological studies show an inverse relationship between physical activity or fitness and the incidence of hypertension, which was either independent of body size or more pronounced in the overweight. The weighted net reduction of blood pressure in response to dynamic physical training averages 3.4/2.4 mm Hg ($P < 0.001$), which appears to be unrelated to the initial body mass index (BMI) and to its training-induced changes. Exercise is less effective than diet in lowering blood pressure ($P < 0.02$), and adding exercise to diet does not appear to further reduce blood pressure. Future studies should observe scientific criteria more strictly, address the truly obese (BMI $\geq$ 30 $kg \cdot m^{-2}$) and attempt to resolve the blood pressure lowering mechanisms. **Conclusion:** Physical activity contributes to the control of blood pressure in overweight as well as in lean subjects. **Key Words:** AEROBIC POWER, BLOOD PRESSURE, BODY FAT, BMI, DIET, EXERCISE

Salt restriction, moderation of alcohol consumption, weight reduction, and increased physical activity are generally accepted lifestyle measures for the management of hypertension (25,60). Meta-analyses concluded that adequate dynamic physical training contributes to the control of blood pressure (15,21), but it has not been investigated whether the effect would be more pronounced in overweight and obese than in lean subjects. The present review therefore addresses the following questions: 1) Is body mass index (BMI) a significant determinant of the preventive effect of physical activity or fitness on the development of hypertension? 2) Is the effect of dynamic physical training on blood pressure different in obese and nonobese subjects? 3) Are training-induced blood pressure changes related to changes in weight? 4) How does the influence of physical training compare with that of dietary intervention? The first question will be addressed by description of the available material, the others by use of meta-analytical techniques (14).

METHODS

Selection of Papers

Articles published before August 1998 and relevant to the aims of this review were identified by a computer-assisted literature search and by checking the reference lists of papers on the topic. When meta-analytical techniques were applied, several criteria were used for acceptability of studies: random allocation to intervention groups and control groups or control phases in case of cross-over design; full publication in a peer-reviewed journal; absence of confounding by some other intervention during the intervention of interest. To assess the influence of dynamic physical training on blood pressure, only randomized controlled trials of at least 4 wk duration concerning normotensive or hypertensive subjects, or both, in whom cardiovascular diseases were reasonably well excluded, were considered. When the effects of exercise were compared with those of dietary intervention or with the combination of diet and exercise, random allocation to the intervention groups was required; however, a control group without intervention was not a prerequisite for inclusion. Finally, studies were accepted only when the actual blood pressures for the intervention and the control groups or phases, or the pressure changes during the intervention and control periods, were available.

Statistical Analysis

Database management and statistical analyses were performed with the SAS software (The SAS Institute Inc., Cary, NC). Meta-analyses consisted of analyses of pooled data with study groups as the units of analysis, with weighting for the number of participants in each group. The net effects of physical training were assessed by weighted

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TABLE 1. Baseline data and net changes in response to exercise.

		Baseline	Net change	
	N	Mean (95% CL)	Mean (95% CL)	*P*
Peak $\dot{V}O_2$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	59	31.4 (29.6; 33.2)	+3.7 (+3.2; +4.3)	<0.001
Heart rate ($beats \cdot min^{-1}$)	48	71.1 (69.3; 72.9)	−4.9 (−5.9; −3.9)	<0.001
BMI ($kg \cdot m^{-2}$)	64	25.6 (25.0; 26.1)	−0.34 (−0.46; −0.22)	<0.001
Body fat (%)	17	27.0 (25.1; 28.9)	−1.15 (−1.62; −0.69)	<0.001
Blood pressure (mm Hg)				
Systolic	68	126.2 (123.3; 129.0)	−3.4 (−4.5; −2.3)	<0.001
Diastolic	68	79.9 (77.9; 82.0)	−2.4 (−3.2; −1.6)	<0.001

Values are weighted means and 95% confidence limits (CL).
Abbreviations: N, number of groups; $\dot{V}O_2$, oxygen uptake.

pooled analyses of the changes in the intervention groups adjusted for control data. The effects of exercise versus diet or versus diet and exercise combined were assessed by weighted pooled analyses on the differences between the changes in the exercise groups and the changes in the intervention groups with which the effect of exercise was compared. Results are reported as weighted means and 95% confidence limits (95% CL). Finally, weighted metaregression analysis was applied to assess whether variations in the results were related to variations in study group characteristics (14).

RESULTS

Physical Activity, Fitness, and Future Blood Pressure

Several studies addressed the relationships between physical activity or fitness and future blood pressure or the incidence of hypertension. Gillum et al. (18) followed 106 subjects from youth to middle age during 32 yr. Physical fitness was derived from pulse rate during a single-stage treadmill exercise test. Individuals who were judged fit had lower systolic blood pressure at follow-up, independent of weight and BMI and of their changes during the follow-up period ($r = 0.26$; $P < 0.01$). Blair et al. (3) related physical fitness, assessed by maximal treadmill testing in healthy normotensive men ($N = 4,820$) and women ($N = 1,219$), aged 20 to 65 yr, to the incidence of hypertension. After the baseline examination the subjects participated in a follow-up mail survey; the follow-up interval ranged from 1 to 12 yr, with a median of 4 yr. Those with excellent and superior fitness (28% of the participants) comprised the reference high physical fitness category, whereas the remaining four physical fitness categories (very poor to good) comprised the comparison group (72%). After adjustment for age, sex, baseline BMI, blood pressure, and follow-up interval, persons with low levels of physical fitness had a relative risk of 1.52 for the development of hypertension when compared with the highly fit persons ($P = 0.02$). Sawada et al. (47) investigated the relationship between physical fitness and incidence of hypertension through a prospective study in 3,305 Japanese men whose blood pressure was normal when they received their first physical examination before the age of 50. The blood pressure of 425 subjects was diagnosed as hypertension in the fifth year. The relative risk of hypertension, after adjustment for age, initial blood pressure, body fat, and other confounders was 1.9 times higher in the least fit compared with the fittest group ($P < 0.01$).

Paffenbarger et al. (43) assessed the incidence of hypertension in 14,998 Harvard male alumni during a 6- to 10-yr follow-up beginning 16 to 50 yr after college entrance. Presence or absence of a background of collegiate sports did not influence the risk of hypertension in this study population, nor did stair-climbing, walking or light sports play by alumni, based on physical activity information obtained by mailed questionnaires in a postcollege health survey. Higher levels of BMI, weight gain since college, history of parental hypertension, and lack of strenuous exercise independently predicted increased risk of hypertension in alumni. Alumni who did not engage in vigorous sports play in postcollege years (59%) were at 35% greater risk of hypertension than the 41% who did ($P < 0.001$), and this relationship held at all ages, 35 to 74 yr. In addition, the inverse relationship between contemporary vigorous exercise and hypertension was most evident for alumni overweight-for-height. Alumni with a BMI less than 36 U.S. units (20% over ideal weight-for-height) but nonparticipants in vigorous sports were at only 15% increased risk of hypertension over comparably light participants, while alumni with a BMI greater than 36 U.S. units but nonparticipants were at 58% increased risk over similarly heavy participants. Among men with little gain in BMI since college, there was a 25% greater risk of hypertension among inactives than among actives, while the

TABLE 2. Baseline data and net changes in response to exercise in lean and overweight study groups, respectively.

		Lean		Overweight	
		N	Mean (95% CL)	*N*	Mean (95% CL)
Age (yr)	BL	27	47.8 (40.6; 55.0)	37	49.6 (46.6; 52.6)
Peak $\dot{V}O_2$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	BL	25	30.7 (27.3; 34.1)	33	31.5 (29.4; 33.6)
	CH		+3.5 (+2.6; +4.4)		+3.9 (+3.2; +4.6)
Heart rate ($beats \cdot min^{-1}$)	BL	20	67.4 (64.6; 70.2)	25	72.9 (70.7; 75.1)
	CH		−4.9 (−6.6; −3.2)		−4.4 (−5.7; −3.2)
BMI ($kg \cdot m^{-2}$)	BL	27	23.4 (23.1; 23.7)	37	27.0 (26.5; 27.5)
	CH		−0.15 (−0.33; +0.03)		−0.45 (−0.60; −0.31)
SBP (mm Hg)	BL	27	123.9 (119.6; 128.2)	37	127.8 (123.7; 131.9)
	CH		−3.3 (−5.1; −1.6)		−3.3 (−4.7; −1.9)
DBP (mm Hg)	BL	27	76.5 (74.1; 78.9)	37	82.4 (79.2; 85.5)
	CH		−2.5 (−3.5; −1.6)		−2.5 (−3.7; −1.2)

Values are weighted means and 95% confidence limits (CL).
N, number of groups; $\dot{V}O_2$, oxygen uptake; SBP, DBP, systolic, diastolic blood pressure; BL, baseline; CH, net change.

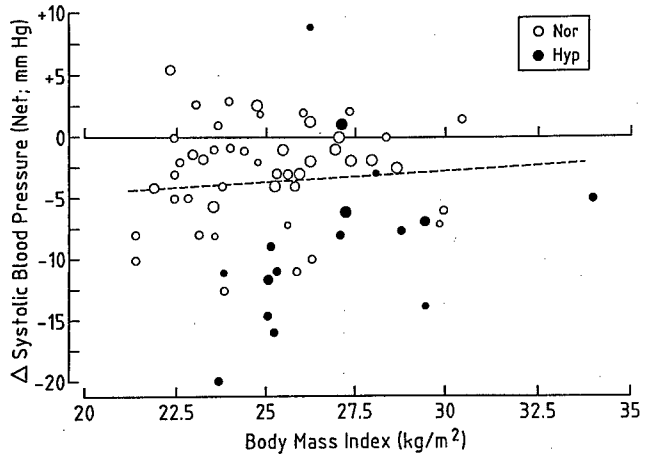

Figure 1—Changes in systolic blood pressure with training, adjusted for control data, vs baseline BMI in normotensive (○) and hypertensive (●) study groups. The four sizes of the circles represent the number of analyzable trained subjects in each group, i.e., < 10, 10–19, 20–29, and ≥ 30, respectively. The regression line is the result of weighted metaregression analysis (r = 0.08; *P* = 0.51).

corresponding figure among those whose gain in BMI was greater than five U.S. units (or > 11.5 kg for a constant height) was 43%.

Evidence Statement: Higher levels of physical activity or fitness are associated with a lower incidence of hypertension; the effect of overweight on this relationship is uncertain. (Evidence Category C)

Physical Training and Blood Pressure Control

Overall results. We identified 44 randomized controlled trials on the effect of dynamic aerobic or endurance exercise on blood pressure at rest in otherwise healthy normotensive or hypertensive individuals (1,2,4–11,16, 17,20,22–24,27–42,44–46,49,50,52–58). Of the 2,674 participants, 65% were men. Nineteen studies comprised only men, four only women, and the others included both sexes (or sex unknown in one). Some studies involved several groups of subjects or applied different training regimens in the same participants, so that a total of 68 training groups/programs are available for analysis. Average age of the groups ranged from 21 to 79 yr (median: 44). Duration of training ranged from 4 to 52 wk (median: 16) with a frequency of one to seven weekly sessions (median: three) of 15–70 min each, including warm-up and cool-down activities (median: 50 min). The exercises involved walking, jogging, running in 69% of the studies, cycling in 50%, swimming in 3%, and other exercises were included in 23% of the training regimens. Average training intensity in the various groups varied between 30 and 85% of maximal exercise performance (median: 65%).

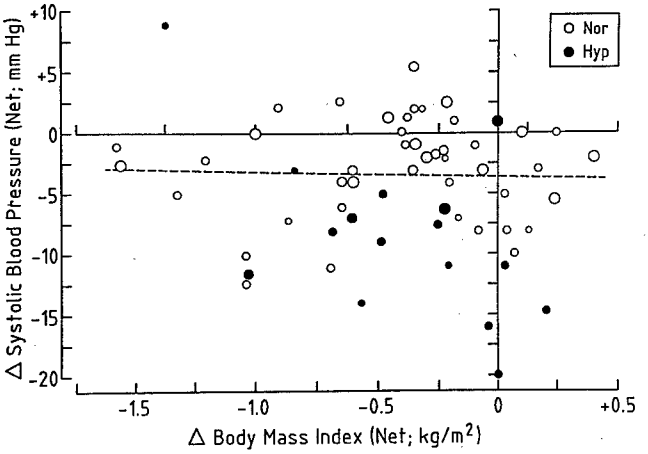

Figure 2—Changes in systolic blood pressure vs changes in BMI with training, adjusted for control data, in normotensive (○) and hypertensive (●) study groups. The four sizes of the circles represent the number of analyzable trained subjects in each group, i.e., < 10, 10–19, 20–29, and ≥ 30, respectively. The regression line is the result of weighted metaregression analysis (r = 0.09; *P* = 0.51).

Control data were collected only at the beginning and at the end of the control period in 23 studies; three control groups were subjected to light dynamic or recreational exercises, 10 were seen at least once in the research facilities, and another eight were contacted regularly by the investigators. Resting blood pressure was measured by an automatic device in 5 of the 44 studies; when pressure was measured by use of a random zero device (N = 15) or by conventional (or unspecified) methodology (N = 24), the investigator was blinded to the treatment in only five and three studies, respectively.

Table 1 summarizes the overall results. In the 68 study groups, the changes of blood pressure in response to training, after adjustment for the control observations, ranged from +9 to −20 mm Hg for systolic blood pressure and from +11 to −11 mm Hg for diastolic pressure. The overall net changes averaged −3.4/−2.4 mm Hg ($P < 0.001$), that is, after adjustment for control observations and after weighting for the number of trained participants that could be analyzed in each study group, whose total number amounted to 1,529. Peak oxygen uptake increased significantly by 11.8% (95% CL: 10.3; 13.4), whereas heart rate, BMI, and percent body fat decreased by, respectively 6.8% (5.5; 8.2), 1.2% (0.8; 1.7), and 4.3% (2.5; 6.1).

Influence of BMI. Table 2 summarizes the results according to whether average baseline BMI, available in 64 study groups, was below 25 $kg \cdot m^{-2}$ (lean groups) or ≥ 25 $kg \cdot m^{-2}$ (overweight and obese groups). Age and peak oxygen uptake were similar in the two groups. Overweight subjects had higher baseline values for heart rate and diastolic blood pressure. Training-induced changes in peak oxygen uptake and heart rate did not differ between the groups. Whereas BMI decreased significantly in the overweight ($P < 0.001$) but not in the lean subjects ($P = 0.12$), the weighted net changes of systolic and diastolic blood pressure were significant ($P < 0.001$) and similar in the two groups.

In addition, weighted metaregression analysis showed that there was no significant relationship between the changes in systolic (r = 0.08; P = 0.51) and diastolic (r = 0.06; P = 0.61) blood pressure, respectively, and baseline BMI (N = 64). Finally, changes in blood pressure were not related to changes in BMI for systolic (r = 0.09; P = 0.51) or for diastolic pressure (r = 0.07; P = 0.59) (N = 61).

TABLE 3. Baseline data and changes in response to exercise and to diet.

		N	Exercise (E) Mean (95% CL)	Diet (D) Mean (95% CL)	E vs D§ P
Age (yr)	BL	11	49.9 (45.9; 53.9)	49.3 (45.2; 53.4)	
Peak $\dot{V}O_2$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	BL	10	31.6 (28.5; 34.8)	31.4 (28.3; 34.5)	
	CH		+3.4 (+2.3; +4.4)	+1.0 (−0.2; +2.1)	=0.002
BMI ($kg \cdot m^{-2}$)	BL	11	28.3 (26.9; 29.6)	28.6 (27.1; 30.1)	
	CH		−0.42 (−0.67; −0.17)	−1.58 (−2.18; −0.97)	=0.002
SBP (mm Hg)	BL	11	125.6 (120.1; 131.1)	124.5 (118.8; 130.1)	
	CH		−3.6 (−5.2; −2.0)	−5.9 (−7.7; −4.1)	=0.004
DBP (mm Hg)	BL	11	81.8 (77.5; 86.1)	80.9 (77.0; 84.8)	
	CH		−2.7 (−3.8; −1.7)	−4.2 (−5.9; −2.4)	=0.02

Values are weighted means and 95% confidence limits (CL).
N, number of groups; $\dot{V}O_2$: oxygen uptake; SBP, DBP: systolic, diastolic blood pressure; BL: baseline; CH: change.
§ Paired comparison of training-induced changes with exercise and with diet.

Figures 1 and 2 illustrate these relationships for systolic blood pressure.

Influence of blood pressure. Average blood pressure was in the normotensive range, defined as systolic blood pressure lower than 140 mm Hg and diastolic pressure below 90 mm Hg, in 52 study groups. Sixteen groups were classified as hypertensive at baseline. The training-induced weighted net change of blood pressure averaged −7.4 (−10.5;−4.3)/−5.8 (−8.0;−3.5) mm Hg in the hypertensives and −2.6 (−3.7;−1.5)/−1.8 (−2.6;−1.1) mm Hg in the normotensives.

Influence of age. Weighted metaregression analysis revealed that baseline BMI ($P = 0.65$), systolic ($P = 0.29$), and diastolic ($P = 0.38$) blood pressure did not differ according to age. Whereas BMI (y) decreased more in younger than in older subjects in response to exercise training (Δ BMI = −0.82 + 0.01 × year; r = 0.28; $P = 0.03$), age was not a significant determinant of the blood pressure response ($P = 0.33$ for systolic, and $P = 0.68$ for diastolic pressure).

Evidence Statement: Dynamic aerobic training reduces blood pressure independent of changes in weight; the blood pressure lowering effect depends on the initial blood pressure, but not on BMI or age. Evidence Category A

Physical Training, Diet, and Blood Pressure Control

We identified 10 randomized trials in which the influence of diet was compared with that of exercise alone and/or with the combined effects of diet and exercise in mostly overweight subjects (2,7,16,19,23,26,45,49,51,59). Two of these studies did not include a nonexercise nondiet control group (19,59), so that the results have not been adjusted for control data in the meta-analysis. Study duration ranged from 4 to 52 wk (median = 38). Table 3 summarizes the results for the paired comparison of exercise and diet (11 study groups). Only physical training increased peak oxygen uptake. The reduction in BMI was significantly more pronounced in the diet groups than in the exercise groups. Finally the reduction of blood pressure with diet alone (−5.9/−4.2 mm Hg) was superior to that of exercise alone (−3.6/−2.7 mm Hg).

The results on the comparison of combined exercise and diet intervention with diet alone are shown in Table 4 ($N = 11$). Only the combined intervention increased peak oxygen uptake. Diet alone was less effective in reducing BMI. Nevertheless, there was no evidence that adding physical training to diet was more effective for blood pressure control than diet alone.

Evidence Statement: Dynamic aerobic training is less effective than diet in lowering blood pressure and exercise does not add to the blood pressure reduction by diet alone. Evidence Category A

DISCUSSION

Several epidemiological studies suggest that the incidence of hypertension is less in physically fit or active people than in unfit or sedentary subjects (3,18,43,47). This relationship was independent of baseline BMI or body fat (3,18,47). However, Paffenbarger et al. (43) reported that the inverse relationship between exercise and the incidence

TABLE 4. Baseline data and changes in response to exercise plus diet and to diet alone.

		N	Exercise + Diet (ED) Mean (95% CL)	Diet (D) Mean (95% CL)	E vs D§ P
Age (yr)	BL	11	48.5 (45.4; 51.6)	48.7 (45.6; 51.8)	
Peak $\dot{V}O_2$ ($mL \cdot min^{-1} \cdot kg^{-1}$)	BL	9	29.7 (25.2; 34.2)	29.1 (24.3; 33.8)	
	CH		+5.4 (+4.1; +6.6)	+0.8 (−0.03; +1.7)	<0.001
BMI ($kg \cdot m^{-2}$)	BL	11	28.6 (27.1; 30.1)	28.6 (27.0; 30.2)	
	CH		−2.04 (−2.73; −1.35)	−1.66 (−2.37; −0.95)	<0.001
SBP (mm Hg)	BL	11	129.4 (120.1; 138.7)	128.3 (120.1; 136.5)	
	CH		−7.1 (−9.9; −4.4)	−6.8 (−9.8; −3.9)	=0.84
DBP (mm Hg)	BL	11	83.1 (77.0; 89.2)	82.1 (76.6; 87.6)	
	CH		−5.5 (−7.5; −3.4)	−4.2 (−6.0; −2.4)	=0.14

Values are weighted means and 95% confidence limits (CL).
N, number of groups; $\dot{V}O_2$: oxygen uptake; SBP, DBP: systolic, diastolic blood pressure; BL: baseline; CH: change.
§ Paired comparison of training-induced changes with exercise and with diet.

of hypertension was more evident for overweight than for lean Harvard alumni.

The results from cross-sectional studies on the associations between physical activity, fitness, and blood pressure are not quite consistent (12,13). Whereas several studies did not observe significant independent relationships, others did find that blood pressure was lower in fitter or more active subjects. On the whole, the differences in blood pressure between the most and the least fit or active rarely exceeded 5 mm Hg after controlling for confounding factors such as age and body size (12,13). It remains difficult, however, to ascribe differences in blood pressure within a population to differences in levels of physical activity or fitness because of the possible confounding factors which cannot be accounted for. Therefore, longitudinal intervention studies are more appropriate to assess the effect of physical exercise on blood pressure. The present meta-analysis of 44 randomized controlled trials involving 68 study groups indicates that dynamic aerobic exercise reduces blood pressure at rest by an average of 3.4 mm Hg for systolic and 2.4 mm Hg for diastolic pressure above blood pressure changes in nonexercising control groups or periods. Baseline BMI did not affect the blood pressure response: the change in blood pressure was indeed similar in overweight and lean participants. However, the lowering of blood pressure was more pronounced in hypertensive subgroups than in the normotensives. The exercise programs led to decreases of BMI (61), particularly in the overweight and obese subjects, but these changes did not determine the blood pressure response.

Weight reduction has been shown to lower blood pressure in overweight subjects (48). The overall analysis of randomized trials in which physical training was compared with dietary intervention in mostly overweight subjects indicates that diet was more effective in reducing both BMI and blood pressure. When exercise was added to diet, BMI was reduced to a greater extent than with diet alone, but there was no additional effect on blood pressure.

RESEARCH PRIORITIES

Physical Training and Obesity

Figure 1 shows that a large number of data are available in overweight subjects (BMI: 25–29.9 $kg \cdot^{-2}$), but that few randomized controlled trials involved obese patients (BMI $\geq$ 30 $kg \cdot m^{-2}$). Therefore, future studies should concentrate on the truly obese, not only to assess the influence on blood pressure but also to define the ideal training program in terms of mode, frequency, time, and intensity.

Scientific Validity of Studies

Although a large number of trials were controlled and applied randomization techniques, other important scientific criteria have not always been observed. The following shortcomings were identified in a variable number of training studies: lack of regular follow-up of the control subjects; no advice to keep diet or lifestyle, or both, constant throughout the study periods; a high number of drop-outs; inadequate statistical analyses and lack of adjustment for confounding variables; failure to blind the person who measured the blood pressure to the treatment or to use stationary or ambulatory automated blood pressure devices; and lack of use of an appropriate cuff size in the obese. Future studies should address these issues. However, it should be realized that it is difficult to blind the participants to the treatment in training studies; the inclusion of low-level exercise as placebo treatment is controversial.

Mechanisms. The results on hemodynamic changes in response to dynamic training are conflicting; some authors claim that the lowering of blood pressure is based on a reduction of systemic vascular resistance whereas others observed a decrease of cardiac output. Most studies found a decrease of plasma noradrenaline concentrations suggesting a reduction in autonomic nervous activity. Other possible blood pressure lowering mechanisms have been addressed only rarely in randomized controlled exercise trials. Future studies, particularly in the obese, should not only focus on blood pressure but also on mechanisms involved in blood pressure regulation such as the renin-angiotensin-aldosterone system, prostaglandins, endothelial relaxing factor and endothelin, the sympathetic nervous system, insulin sensitivity and finally, genetic polymorphisms that might influence the blood pressure response to physical training.

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Physical activity in the prevention and treatment of a thrombogenic profile in the obese: current evidence and research issues

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ABSTRACT

RAURAMAA, R. and S. B. VÄISÄNEN. Physical activity in the prevention and treatment of a thrombogenic profile in the obese: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S631–S634, 1999. **Purpose:** To evaluate the impact of regular physical activity on thrombogenic profile in obese individuals. **Design:** *Medline*-based literature search with emphasis on controlled randomized clinical trials. The focus was on the impact of physical activity on platelet aggregation, fibrinogen, and plasminogen activator inhibitor-1 (PAI-1) in overweight and obese subjects. **Results:** Physical activity increases *acutely* 1) platelet number and activity, 2) activation of coagulation leading to a thrombin generation, and 3) simultaneous activation of fibrinolysis. On the other hand, hemostatic changes resulting from regular exercise training are limited to few data on platelets and blood coagulation and to some indications of increased fibrinolysis. Obesity is a risk factor for atherosclerotic cardiovascular diseases, and it is associated with hypertriglyceridemia, hyperinsulinemia, and non-insulin-dependent diabetes (NIDDM). These states interfere with a balance between blood coagulation and fibrinolysis leading to an increased thrombogenesis. Regular physical activity reduces platelet aggregability, while the effects on plasma fibrinogen and fibrinolytic activity remain unclear. **Conclusions:** Although obesity associates with several unfavorable derangements in the hemostatic system, data on the interactions of regular physical activity with blood coagulation in overweight or obese subjects are scarce. Therefore, controlled randomized clinical trials with adequate statistical power are needed for the evaluation of physical activity in the prevention and treatment of obesity-related atherothrombotic disorders. **Key Words:** EXERCISE, OBESITY, BLOOD COAGULATION, FIBRINOLYSIS, PLATELET AGGREGATION, FIBRINOGEN, PLASMINOGEN ACTIVATOR INHIBITOR-1

Vascular hemostasis results from a regulated interaction of a coagulation and fibrinolytic systems, which are in dynamic equilibrium in a normal situation. Any imbalance between these systems leads to a tendency to bleeding or to an increased thrombogenesis. Obesity is an independent risk factor for the development of atherosclerotic cardiovascular disease, and it is associated with hypertriglyceridemia, hyperinsulinemia, and non-insulin-dependent diabetes (NIDDM). These states increase blood coagulability, a complicated set of interactions among several clotting and fibrinolytic factors. In this paper, the emphasis will be on the impact of physical activity on platelet aggregation, fibrinogen, and plasminogen activator inhibitor-1 (PAI-1) in the obese. While exercise-induced acute effects on hemostatic factors have been reviewed recently (4), the present article focuses on the impact of exercise training on blood coagulation and fibrinolysis in controlled clinical trials.

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In general, physical exercise increases acutely 1) platelet number and activity, 2) activation of coagulation leading to a slight but significant thrombin generation, and 3) simultaneous activation of fibrinolysis. While the hemostatic balance is maintained during exercise, fibrinolysis diminishes rapidly during the recovery phase; this might constitute a key mechanism for the exercise-induced cardiovascular complications. Currently, hemostatic changes resulting from regular exercise training are limited to few data on platelets, blood coagulation, and to some indications of increased fibrinolysis. Although obesity associates with several unfavorable derangements in the hemostatic system, data on the interactions of regular physical activity with the hemostasis and coagulation in overweight or obese subjects are scarce. Several factors make it difficult to evaluate and combine the results from the exercise studies; a large variation in study populations and in exercise intensities and durations, as well as in measurements of physical activity, are probably the main reasons for the contradictory results. Moreover, the duration of the intervention may have been too short and the number of subjects too small to reveal the true exercise-induced effects on hemostatic factors.

TABLE 1. Summary table of controlled clinical trials (r = randomized) on the effects of physical activity on platelet aggregation, fibrinogen, and plasminogen activator inhibitor-1 (PAI-1).

References	Duration and Type Exercise Program	Subjects (age in yr)	Exercise-Induced Effects
Platelet aggregation			
Rauramaa et al., 1984 (26)	8 wk, mild intensity, r	60 men (32–44)	6-keto-prostaglandin levels increased
Rauramaa et al., 1986 (27)	12 wk, moderate intensity, r	53 overweight men (30–49)	Platelet aggregability decreased
Wang et al., 1995 (43)	8-wk training and 12-wk deconditioning, r	23 men (mean ± SD, 21 ± 1)	Platelet aggregability decreased; reversed back after deconditioning
Wang et al., 1997 (44)	Training (2 menstrual cycles (MC)) and a deconditioning (3 MCS) period, r	16 women (22 ± 1)	Platelet aggregability decreased; reversed back after deconditioning
Fibrinogen			
Schneider et al., 1988 (34)	6 wk, physical training 3 times per wk	16 sedentary NIDDM patients (52 ± 2) and 9 controls (48 ± 4)	No effect
Rankinen et al., 1994 (24)	6 months, aerobic exercise, r	119 normolipidemic men (51–53)	No effect
Svendsen et al., 1996 (37)	12-wk diet and exercise intervention, r	121 overweight women (54 ± 3)	No effect
Schuit et al., 1997 (35)	6 months, intensive training, r	73 men and 71 women (60–80)	Fibrinogen increased
PAI-1			
de Geus et al., 1992 (8)	8 months, different programs, r	50 men (25–40)	No effect
Rankinen et al., 1994 (24)	6 months, aerobic exercise, r	119 normolipidemic men (51–53)	No effect
Svendsen et al., 1996 (37)	12-week diet and exercise intervention, r	121 overweight women (54 ± 3)	Diet decreased PAI-1 with no additional effect of physical activity
van den Burg et al., 1997 (38)	12 wk, submaximal training	39 men (20–30)	PAI-1 decreased
Schuit et al., 1997 (35)	6 months, intensive training, r	73 men and 71 women (60–80)	PAI-1 decreased moderately
Väisänen et al., 1999 (41)	3 yr, low intensity, r	136 men (53–63)	PAI-1 decreased in the 4G4G subjects

PLATELET AGGREGATION

Platelets react to many foreign surfaces, especially to the damaged endothelium of vessel walls, by changing their shape to irregular spheres and putting out pseudopods. These activated platelets adhere to the vessel wall and to each other to form aggregates. The secondary aggregation, which leads to irreversible platelet aggregation and development of firm hemostatic plug, associates with prostaglandin formation (13). Strenuous exercise activates platelets acutely, while moderate exercise has a suppressing effect on platelet aggregation in young healthy men (45). Regular physical activity reduces platelet aggregability in overweight middle-aged men (27)and in young men (43), as well as in young women (44) (Table 1).

Physical activity can induce at least two different mechanisms that affect platelet function. Regular physical activity increases serum high density lipoprotein (HDL), which can stimulate prostacyclin production (26) and thereby decrease platelet aggregation. In addition, physical activity increases release of nitric oxide, a potent mediator of antiplatelet effects, for instance, by elevating cGMP contents in platelets, which in turn suppress platelet reactivity (43). However, the resting and exercise-induced reductions in platelet aggregation reverse back to the pretraining level after deconditioning (43,44), indicating the importance of engaging in physical activity in a regular manner.

Regular physical training inhibits platelet aggregability in overweight men. (Evidence Category B)

FIBRINOGEN

Fibrinogen plays a central role in the final phase of the blood coagulation cascade, and the binding of fibrinogen to platelet glycoprotein IIb/IIIa receptors is the principal mechanism for platelet aggregation (15). Fibrinogen is elevated in inflammatory states, smokers, obesity, diabetes, and hyperlipidemia, and epidemiological studies show that increased fibrinogen level is an independent risk factor for cardiovascular disease and mortality (6,7). Although an inverse relationship between fibrinogen and physical activity and/or cardiorespiratory fitness has been found in several cross-sectional studies (40), the fibrinogen lowering effect of regular physical activity has been reported only in few exercise intervention studies: in an uncontrolled study in old men (36), in sedentary subjects with newly diagnosed NIDDM (39), and after 10 wk of strenuous and intensive training in young men (21). On the contrary, an intensive exercise program increased plasma fibrinogen in elderly subjects (35) (Table 1). Thus far, only one study has been concentrated on overweight subjects, and they reported that combined diet and physical activity program could not decrease fibrinogen level (37). Fibrinogen is an acute phase protein, and it has a relatively high intraindividual variability (29). Therefore, repeated measurements are preferable to show the real exercise-induced effect on fibrinogen level (42).

Recently two studies have examined whether genetic polymorphisms in the fibrinogen genes can modulate the association between physical activity and plasma fibrinogen. We found an interaction between habitual physical activity and an α-fibrinogen polymorphism on fibrinogen level in postmenopausal women. The physically most active women, who were homozygous for the more frequent *Rsa* I allele, displayed a decreased plasma fibrinogen, while the association was not seen in other genotypes (28). An acute phase response in fibrinogen level was reported in young men after an exhausting 2-d military exercise period. The subjects carrying the A allele of the G-453-A polymorphism in the β-fibrinogen gene had higher increase in plasma fibrinogen than in men with the GG genotype (21). Controlled exercise intervention studies are needed to evaluate further the effect of genetic variation at the fibrinogen gene loci on the relationship between physical activity and fibrinogen level in both genders as well as in obese subjects.

Despite cross-sectional findings and nonrandomized trials suggesting that physical activity decreases plasma fibrinogen, this hypothesis has not yet been confirmed in the few randomized controlled trials in either obese or nonobese subjects. (Evidence Category C)

PLASMINOGEN ACTIVATOR INHIBITOR-1 (PAI-1)

Most data on thrombogenic profile in the obese relate to PAI-1, the primary physiological inhibitor of the fibrinolytic system. Elevated PAI-1 activity decreases fibrinolytic activity and increases the risk of coronary artery disease, venous thromboembolism, and acute myocardial infarction (10,12,30). Obese and NIDDM subjects have significantly higher plasma PAI-1 than control subjects (19), and waist/hip-ratio correlates strongly positively with coagulation factors and negatively with fibrinolytic factors in premenopausal women with abdominal obesity (1). Older women receiving postmenopausal hormone therapy have more favorable PAI-1 and fibrinogen levels than nonusers (20).

The active role of adipose tissue as an important contributor to thrombogenesis has been understood only very recently (31). Elevated plasma PAI-1 activity may result in PAI-1 release from an increased visceral adipose tissue (16). In addition to insulin (32), two cytokines, tumor necrosis factor alpha (TNF-α) and transforming growth factor beta (TGF-β), induce PAI-1 gene expression in the adipose tissue, the excess of which in obese subjects serves as an additional source for PAI-1 production (32,33). Strenuous physical activity in young athletes increases acutely serum TNF-α (46). However, it is not known how regular exercise training may modulate these two cytokines chronically or acutely in a response to a single bout of physical exercise.

Fibrinolytic response to acute physical activity is modified by exercise intensity (25), and increased plasma epinephrine during exercise is the primary stimulus for t-PA, thereby leading to reduction in PAI-1 activity (2). However, this exercise-induced increase in fibrinolytic activity is short lived (25). There is also suggestive evidence that regular exercise training decreases PAI-1 level at least in sedentary young men (9,38) and in elderly men (3,36) (Table 1). Currently, no corresponding data are available on fibrinolytic activity in obese subjects. Several preanalytical factors such as diurnal variation and difficulties in blood collection and handling have significant effects on fibrinolytic activity (11,14), and these confounding factors make it essential to include a reference group in a study design. The 4G allele of the 4G/5G polymorphism in the PAI-1 promoter gene associates with an increased PAI-1 level and higher risk of cardiovascular diseases (5,18,23). Moreover, at least in diabetic patients (17,23) and subjects who have suffered myocardial infarction (22), homozygotes for the 4G allele are particularly sensitive for the increasing effect of hypertriglyceridemia on PAI-1 level. These observations raise the question of whether the response to physical activity in fibrinolysis varies between different 4G/5G genotypes. We observed in a 3-yr controlled randomized exercise intervention in a population based sample of middle-aged men that the 4G4G subjects tend to have higher PAI-1 activity than other genotypes, but the 4G4G men were also more sensitive to a PAI-1 lowering effect of physical activity (41). A change in waist circumference, although not identical to measurement of visceral fat, did not explain this finding.

Regular physical training increases fibrinolysis, which may be modified by the genetic variability; however, data pertaining specifically to the obese are currently not available. (Evidence Category B)

RESEARCH ISSUES

To avoid false negative conclusions in clinical intervention trials on the effects of physical activity on thrombogenic profile, not only in obese subjects but also in other populations, it is necessary to make careful power calculations with a large enough sample size, preferably as a representative sample of the study population. Another central issue is a requirement for controlled randomized studies. In the future, studies on physical activity and thrombogenic factors should neither be funded nor published if they do not fulfill these basic criteria.

We suggest the following research topics for future clinical trials:

1. Effects of regular low-to-moderate intensity physical activity on thrombogenesis involved in atherosclerosis with special reference to body composition.
2. Effects of regular low-to-moderate intensity physical activity on thrombogenic profile in postmenopausal women with special reference to body composition.
3. Effects of regular physical activity of different intensities on hemostatic factors with special references to genetic variations in thrombogenic factors.
4. Exercise-induced antithrombotic mechanisms and their modification by regular physical activity in obese and nonobese subjects.

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Physical activity in the prevention and treatment of other morbid conditions and impairments associated with obesity: current evidence and research issues

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ABSTRACT

RISSANEN, A. and M. FOGELHOLM. Physical activity in the prevention and treatment of other morbid conditions and impairments associated with obesity: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S635–S645, 1999. **Purpose:** To evaluate the current status of knowledge concerning the effects of physical activity in the treatment and prevention of obesity- related problems, including cancers of the colon, breast, uterus, and prostate; gallstones; osteoarthritis; back pain; sleep apnea; reproductive abnormalities; and impaired health-related quality of life. **Design:** A *Medline* literature search on the effects of physical activity in the above conditions was conducted. Only studies with some measure of weight and a description of the type of physical activity were included. **Results:** No controlled randomized trials of exercise in the treatment of any of the studied conditions in obese patients were identified. Most of the epidemiologic studies reviewed were beset with severe methodological weaknesses. Most of the 18 studies on physical activity and colon cancer risk showed a protective effect that in some studies appeared to be greater than expected by weight loss alone. Some but not all studies of hormone-dependent cancers and gallstones showed a protective effect for physical activity. There were insufficient data on the role of exercise for the other morbid conditions studied. **Conclusion:** The scarce data available on the role of physical activity in the prevention of obesity-related chronic conditions listed above suggest a protective role that needs to be examined further in studies with improved methodologies. Well-designed intervention trials are needed to assess the role of physical activity in the treatment and long-term outcome of obese patients with these co-morbid conditions. **Key Words:** EXERCISE, LEISURE TIME ACTIVITY, OCCUPATIONAL ACTIVITY, COLON CANCER, BREAST CANCER, ENDOMETRIAL CANCER, PROSTATE CANCER, OSTEOARTHRITIS, BACK PAIN, GALLSTONES, SLEEP APNEA, HEALTH-RELATED QUALITY OF LIFE

Obesity and sedentary lifestyle are associated with a number of chronic disabling conditions. This review covers the knowledge on the role of physical activity in some of these conditions and discusses the problems inherent to studies addressing the complex interrelationships between lifestyle variables and their consequences to health.

BREAST CANCER

An excess risk of postmenopausal breast cancer has been documented for obese women, especially for those with increased abdominal fat (103). Adult weight gain predicts the risk of postmenopausal breast cancer (35). Clinical studies agree that obesity worsens the prognosis of breast cancer in both pre- and postmenopausal women (36). Few studies have been able to disentangle the effects of obesity from those of diet, nutrition, and physical exercise (60).

There is some evidence that physical activity may reduce breast cancer risk in both pre- and postmenopausal women. Most but not all of the several epidemiological studies, all beset with methodological problems, suggest a protective effect of physical activity (44). Although the effect of body mass index (BMI) has been taken into account in the analyses of several studies (Table 1), it is not clear to what extent the protective effect of exercise is independent of body weight. The possible biological mechanisms behind the protective effect include reduction in body weight and in endogenous steroid exposure, changes in growth factors, and enhancement of natural immune mechanisms (78).

Given the biological plausibility and the strength of the evidence from the literature, prevention of obesity and weight gain in adulthood and regular physical exercise could be expected to help reduce the risk of postmenopausal breast cancer in obese women. However, public health recommendations cannot be made on the basis of the existing evidence. Similarly, insufficient data are yet available about

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TABLE 1. Epidemiologic studies on breast cancer and physical activity.

Author, Year, Country	Study Design and Population	Breast Cancer Cases	Physical Activity Definition	Result	Comment
Frisch et al. 1985, USA (21)	Retrospective cohort, 2622 former college athletes and 2776 nonathletes aged 21–80 yr, follow-up 56 yr	Total = 69 Premenopausal = 13 Postmenopausal = 56	Member of a team over a year or nonteam athlete who trained regularly 5 times/wk running >2 miles	Inactive vs active: RR = 1.9 (CI = 1.0–3.5)	Adjusted for age, menarche, parity, family history of cancer, smoking, oral contraceptives, replacement therapy, leanness
Albanes et al. 1989, USA (1)	Prospective cohort, 7407 sample of US civilian population, aged 25–74 yr, follow-up 10 yr	Total = 122 Premenopausal = 46 Postmenopausal = 76	Two questions on recreational and nonrecreational activity	Inactive vs active: RR = 1.7 (0.8–2.9) Premenopausal RR = 0.4 (0.1–1.8) Postmenopausal RR = 1.5 (0.7–2.8)	Adjusted for age, smoking, economic status, reproductive history, family breast cancer history, diet fat or energy, BMI
Paffenbarger et al. 1992, USA (69)	Prospective cohort, 2370 alumnae aged 40–50 yr, follow-up 15 yr	Total = 73	Type, frequency and duration of activities: number of city blocks walked and stairs climbed daily	>1000 kcal vs <1000 kcal/week: RR = 0.9 (0.5–1.4)	Adjusted for age, BMI, history of maternal cancer
Bernstein et al. 1994, USA (4)	Population based case-control, 1090 women, aged 36–40 yr	Total = 545, all under 40 yr	Average number of hours per week subject engaged in physical exercise between menarche and reference date.	Active vs inactive: Premenopausal OR = 0.4 (0.3–0.6)	Adjusted for age, menarche, age at first birth, months of lactation, first degree family history of breast cancer, BMI, oral contraceptives
Friedenreich and Rohan 1995, Australia (19)	Population based case-control, 902 women, aged 20–74 yr	Total = 444 Premenopausal = 110 Postmenopausal = 258 Menopause not determined = 76	Number of hours/wk for light, moderate, or vigorous physical activity converted into kcal of energy expended per week.	Active vs inactive: OR = 0.7 (0.5–1.0) Premenopausal OR = 0.6 (0.3–1.2) Postmenopausal OR = 0.7 (0.4–1.2)	Adjusted for age, menarche, age at first birth, parity, family history of breast cancer, personal history of benign breast disease, smoking, menopause, education, oophorectomy, oral contraceptive use, replacement therapy, energy intake, BMI
Hirose et al. 1995, Japan (32)	Hospital-based case-control, 24349 women, aged 20–80 yr	Total = 1052 Premenopausal = 607 Postmenopausal = 445	Physical exercise for health: not at all vs at least 2 times/wk	Active vs inactive: Premenopausal OR = 0.7 (0.5–1.0) Postmenopausal OR = 0.7 (0.5–1.0)	Adjusted for BMI, menarche, delivery, smoking, diet
Taioli et al. 1995, USA (88)	Hospital-based case-control, 1148 women, over 25 yr	Total = 617 Premenopausal = 196 Postmenopausal = 421	Strenuous exercise 3 times a week, for 1 yr or more, calculated as average weekly energy expenditure	Energy expenditure 1750 kcal vs none: Premenopausal OR = 0.6 (0.2–1.6) Postmenopausal OR = 1.1 (0.5–2.6)	Adjusted for age, education, BMI, menarche, parity
Steenland et al. 1995, USA (83)	Prospective cohort, 7413 women, aged 25–74 yr	Total = 122 Premenopausal = 46	Self-reported nonrecreational physical activity (a lot, some, little)	Little vs lots: OR = 0.9 (0.5–1.5) Some vs lots 0.9 (0.6–1.2)	Adjusted for age, BMI, alcohol consumption, cholesterol, diabetes, income, menopause, race, recreational physical activity, pulse, smoking
McTieman et al. 1996, USA (59)	Population based case-control, 1029 women, aged 50–64 yr	Total = 537, all postmenopausal	Leisure time physical activity in two periods in life: 12–21 yr, and during the 2-yr period prior to reference date, at least 24 times/yr	Active vs inactive: OR = 0.7 (0.4–1.1)	Adjusted for age, BMI, menarche, menopause, age at first birth, parity, family history of breast cancer, previous benign breast disease, oral contraceptives, replacement therapy, alcohol, dietary fat, education, mammogram
Thune et al. 1997, Norway (93)	Prospective cohort, 25624 sample of Norveigan women aged 20–54 yr, follow-up 13, 7 yr	Total = 351 Premenopausal = 100 Postmenopausal = 251	Leisure time and occupational physical activity in the year preceding each survey	Active vs inactive: *Leisure time* Total: RR = 0.6 (0.4–0.9) Premenopausal RR = 0.5 (0.2–1.1) Postmenopausal RR = 0.7 (0.4–1.1) *Occupational* Total: RR = 0.5 (0.2–0.9) Premenopausal RR = 0.5 (0.24–0.95) Postmenopausal RR = 0.8 (0.5–1.2)	Adjusted for age at entry, county of residence, number of children, age at birth of first child, intake of total fat and energy, BMI
Chen et al. 1997, USA (6)	Population based case-control, 1708 women, aged 21–45 yr	Total = 747 Premenopausal = 643 Perimenopausal = 10 Postmenopausal = 92	Leisure-time physical activity in the 2-yr period prior to the reference date and at age 12–21, calculated as total MET of recreational activity/wk from average number of physical activity episodes/wk and average hours for each level of intensity	Active vs inactive: OR = 0.9 (0.7–1.2)	Adjusted for age, BMI, county of residence, age at menarche, age at first term pregnancy, parity, family history of breast cancer, education, family income, smoking, alcohol

TABLE 1.—*Continued*

Author, Year, Country	Study Design and Population	Breast Cancer Cases	Physical Activity Definition	Result	Comment
Fraser et al. 1997, USA (18)	Prospective cohort, 20341 women, aged 24–90 yr	Total = 218	Two questions on frequency of vigorous recreational and current occupational activity	Low vs high level of total physical activity: RR = 1.5 (1.1–1.9)	Adjusted for age, age at first birth, oral contraceptive use, hormone replacement therapy, family history of cancer, history of benign breast disease, energy and fat intake
Coogan et al. 1997, USA (8)	Population based case-control study, 11676 women, aged under 75 yr	Total = 4863 Premenopausal = 2104 Postmenopausal = 3557	Occupational physical activity	Active vs inactive: Premenopausal OR = 0.6 (0.3–1.3) Postmenopausal OR = 0.9 (0.6–1.2)	Adjusted for family history of breast cancer, alcohol consumption, BMI, menopausal status, education, history of benign breast disease, age at menarche, parity, age at first birth, level of physical activity at ages 14 to 22
Hu et al. 1997, Japan (34)	Hospital-based case control, 526 women aged 25–65 yr	Total = 157 Premenopausal = 87	Self-reported total h/wk in moderate and strenuous activities	Active vs inactive: *Strenuous activity* Premenopausal OR = 0.7 (0.4–1.4) Postmenopausal OR = 0.5 (0.2–1.5) *Moderate activity* Premenopausal OR = 1.0 (0.3–1.9) Postmenopausal OR = 1.4 (0.6–3.1)	Adjusted for age, age at menarche, age at first birth, BMI 4 yr before study, duration of breast feeding, parity, residential area
Mezzetti et al. 1998, Italy (60)	Hospital-based case-control, 5157 women, aged 23–74 yr	Total = 2528 Premenopausal = 956 Postmenopausal = 1572	Occupational and leisure time physical activity as a sum score.	Medium vs high score: Premenopausal OR = 1.0 (0.8–1.4) Postmenopausal OR = 1.2 (1.0–1.4) Low vs high score: Premenopausal OR = 1.4 (0.9–2.1) Postmenopausal OR = 1.6 (1.2–2.2)	Adjusted for age, center, beta-carotene, vitamin E, alcohol, BMI, energy intake, education, menopausal status
Gammon et al. 1998, USA (23)	Population-based case-control, 3173 women under 45 yr	Total = 1668 Premenopausal = 1475 Naturally postmenopausal = 34 Surgically postmenopausal = 136	Frequencies of vigorous and moderate physical activity for three different time periods: ages 12–13, age 20 yr and the year prior to the interview expressed in quartiles of physical activity in relative units/yr	Higher vs lowest quartile (ref.): *Ages 12–13 yr* OR (2nd Quart) = 1.1 (0.9–1.3) OR (3rd Quart) = 0.9 (0.7–1.1) OR (4th Quart) = 1.0 (0.8–1.2) *Age 20 yr* OR (2nd Quart) = 1.0 (0.8–1.2) OR (3rd Quart) = 1.1 (0.9–1.4) OR (4th Quart) = 1.1 (0.9–1.4) *Past year (year before interview)* OR (2nd Quart) = 1.1 (0.9–1.4) OR (3rd Quart) = 1.1 (0.9–1.4) OR (4th Quart) = 1.1 (0.9–1.3)	Adjusted for age, center, age at first birth, age at menarche, parity, lactation, number of abortions, number of miscarriages, menopausal status, marital status, education, family income, race, BMI at age 20 yr, BMI in adulthood, months of oral contraceptive use, hormone replacement therapy, alcohol, smoking, caloric intake in the past year, history of breast biopsy, family history of breast cancer
Sesso et al. 1998, USA (77)	Prospective cohort study, 1566 alumnae aged 37–69 yr, follow-up 31 yr	Total = 109 Premenopausal = 28 Postmenopausal = 81	An index of weekly energy expenditure estimated from daily number of flights of stairs climbed and city blocks walked and number of h/wk spent in various sport, expressed as kcal/wk	Active vs inactive (<500 kcal/wk, ref.) *Before age 55* RR (500–999 kcal) = 0.8 (0.3–2.5) PP (≥1000 kcal) = 1.8 (0.8–4.3) *After age 55* RR (500–999 kcal) = 1.0 (0.6–1.6) PP (≥1000) = 0.5 (0.3–0.9)	Adjusted for age, BMI

RR, relative risk; OR, odds ratio; CI, 95% confidence interval; MET, metabolic equivalent.

TABLE 2. Epidemiologic studies on colon cancer and physical activity.

Author, Year, Country	Study Design and Population	Physical Activity Definition	Result	Comment
Slattery et al. 1988, USA (80)	Case-control M: 110 cases, 180 controls F: 119 cases, 204 controls	Occupational and leisure time activity for 2 yr prior to diagnosis	Active vs inactive: M: OR = 0.7 (90% CI 0.4–1.3) F: OR = 0.5 (90% CI 0.3–0.9)	Result unchanged after adjustment for age, BMI, fiber and energy intake
Kune et al. 1990, Australia (42)	Case-control M: 388 cases, 398 controls F: 327 cases, 329 controls	Occupational and recreational activity combined	No relation between activity and colorectal cancer in either sex. Suggestive inverse relation among women	Adjusted for age, BMI, diet
Slattery et al. 1990, USA (81)	Case-control M: 290 cases F: 323 cases Population based controls	Self-reported occupational and leisure-time activity	High vs low activity: M: RR = 0.7 (90% CI 0.4–1.4) F: RR = 0.5 (90% CI 0.3–0.9)	Adjusted for age, BMI, fiber consumption
Gerhardsson et al. 1990, Sweden (24)	Case-control M: 163 cases F: 189 cases 512 controls	Questions on activity during work and recreational hours	Active vs sedentary: M: RR (fairly active) = 0.8 RR (very active) = 0.6 (0.2–1.7) F: RR (fairly active) = 0.8 RR (very active) = 0.4 (0.2–1.1)	Adjusted for age, BMI, total energy intake, protein, fat, fiber, and browned meat
Longnecker et al. 1995, USA (53)	Case-control 405 cases 703 community controls	Occupational activity 5 yr and 20 yr ago and representative for lifetime. Leisure-time spent in six activities	"More than light work" vs sedentary work: RR = 0.7 (0.3–1.5) More than 2 h leisure-time activity weekly vs none: RR = 0.6 (0.3–1.0) No relation for rectal cancer	Adjusted for BMI, race, smoking, socioeconomic status, family history, diet, alcohol intake
White et al. 1996, USA (94)	Case-control M: 251 cases F: 193 cases Registry controls	Lifetime occupational activity. Recreational and household activity over 10 yr prior to the 2 yr before diagnosis	Moderate or high intensity recreational activity at least 2 times/wk vs none: RR = 0.7 (0.5–1.0) Total occupational plus recreational activity ≥5 h/wk vs none: RR = 0.8 (0.6–1.1)	Adjusted for age, BMI, diet, education, smoking, frequency of constipation
Slattery et al. 1997, USA (82)	Case-control M: 1.099 cases, 1.290 controls F: 889 cases, 1.120 controls	Activity at home, leisure, and work for the referent year, 10, and 20 yr ago.	Least active vs most active: M: OR = 0.8 (0.5–0.8) F: OR = 1.6	Adjusted for age, family history, BMI, use of aspirin, energy intake, dietary fiber, and calcium intake
Le Marchand et al. 1997, USA (46)	Case-control M: 689 cases F: 494 cases 1192 controls Multiethnic population in Hawaii	Lifetime recreational activity Daily physical activity Occupational physical activity	Highest vs lowest quartile: Lifetime activity M: RR = 0.6 (0.4–0.8) ($P < 0.01$ for trend) F: RR = 0.7 (0.5–1.1) Daily activity M: RR = 1.0 (0.7–1.5) F: RR = 0.6 (0.4–1.0) ($P < 0.05$ for trend) Occupational activity M: RR = 1.3 (0.9–1.9) F: RR = 1.5 (0.9–2.3)	Adjusted for family history, alcohol intake, smoking, dietary constituents, calories, BMI 5 years ago
Severson et al. 1989, USA (78)	Prospective cohort of 8.006 Japanese men in Hawaii 128 colon cancers	Total physical activity index based on reported activity	Higher vs lowest tertile: RR (middle tertile) = 0.6 (0.4–0.8) RR (highest tertile) = 0.7 (0.5–1.0) No relation for rectal cancer.	Adjusted for age and BMI
Albanes et al. 1989, USA (1)	Prospective cohort (NHANES I follow-up) 5,138 men, 7,407 women, 128 colorectal cancers	Two questions on physical activity. In your usual day, aside from recreation, how active are you? Do you get much exercise in things you do for recreation?	Nonrecreational activity Active vs inactive: M: RR = 0.6 (0.3–1.4) F: RR = 1.4 (0.5–3.3) Recreational activity Much vs little: M: RR = 1.0 (0.5–1.9) F: RR = 0.8 (0.4–1.7)	Adjusted for age, BMI, smoking, socioeconomic status, diet
Ballard-Barbash et al. 1990, USA (2)	Prospective cohort (the Framingham study) 1,906 men and 2,308 women 73 male colon cancers 79 female colon cancers	Total measure index based on reported activity	Highest vs lowest tertile: M: RR = 0.6 (0.–1.0) F: RR = 0.9 (0.6–1.7)	Adjusted for BMI, education, cholesterol, alcohol intake, smoking, height

TABLE 2.—*Continued*

Author, Year, Country	Study Design and Population	Physical Activity Definition	Result	Comment
Thun et al. 1992, USA (90)	Prospective cohort (Cancer Prevention Study II) 5,746 controls sampled from a population of 764,343 adults. 611 male colon cancers 539 female colon cancers	Physical activity in work or play; none, slight, moderate, or heavy	Heavy exercise vs none: M: RR = 0.6 (0.3–1.3) F: RR = 0.9 (0.4–2.0) Inverse trends not statistically significant	Adjusted for BMI, diet, family history, and aspirin use
Giovannucci 1995, USA (27)	Prospective cohort (US Health Professionals Follow-up Study) 47,723 men 203 colon cancers	Average time/wk over the past year in walking, moderate, and vigorous activities (list). Stairs climbed at walking pace were recorded	Highest vs lowest activity: RR = 0.5 (0.3–0.9) $P < 0.05$ for inverse trend for colon cancer with increasing activity. No relation for rectal cancer	Adjusted for age, BMI, polyps, family history, diet, alcohol intake, aspirin smoking
Thune and Lund 1996, Norway (92)	Prospective cohort of 28,2874 Norwegian women and 53,242 Norwegian men 236 male colon cancers 170 male rectum cancers 99 female colon cancers 58 female rectum cancers	Physical activity during recreation and occupation during last year	Active vs sedentary: F: RR = 0.6 (0.8–1.5) $P = 0.04$ for trend M: RR = 1.0 (0.6–1.5) Among men over 45 at baseline lower risk with higher activity (active vs sedentary RR = 0.7). No relation for rectal cancer in women or men	Adjusted for age, BMI, cholesterol, geographic region
Martinez et al. 1997, USA (57)	Prospective cohort (Nurses' Health Study) 121700 women 396 female colon cancers	Average time/wk over the past year in walking, moderate, and vigorous activities: walking or hiking outdoors, jogging, running, lap swimming, bicycling, tennis squash or raquetball, calisthenics or rowing. Leisure time energy expenditure calculated as weekly MET hours	Energy expenditure >2 MET vs <2 MET h/wk: RR = 0.5 (0.3–0.9) $P = 0.01$ for trend for decreasing risk with increasing level of physical activity. Results stronger for distal than proximal colon. A tendency toward higher risk for increasing waist-to-hip ratio.	Adjusted for age, family history, smoking, aspirin use, red meat consumption, alcohol intake, hormonal replacement. Obesity an independent risk factor
Lee et al. 1997, USA (47)	Prospective cohort (Physicians' Health Study) 21807 men 217 colon cancer cases	Baseline question about the frequency of vigorous exercise. At 3-yr follow-up a question about regular vigorous exercise	Active vs inactive (<1 times/wk): RR (1×/wk) = 1.1 (0.7–1.7) RR (2–4×/wk) = 1.2 (0.8–1.6) RR (>5×/wk) = 1.1 (0.7–1.6) $P = 0.06$ for trend	Adjusted for age, obesity, alcohol and treatment (aspirin or placebo, β-carotene or placebo)
Hsing et al. 1998, USA (33)	Prospective cohort 17.663 white men 120 colon cancers 25 rectal cancers	Self-reported physical activity	Moderate to heavy physical activity reduced risk by 10–30%	Adjusted for age, smoking, alcohol intake, occupation
Will et al. 1998, USA (98)	Prospective cohort (Cancer Prevention Study) 349.631 persons 3218 colon cancer cases	Usual level of exercise at work or play	Activity vs no exercise or play: ICD (moderate) = 0.8 (0.8–0.9) ICD (heavy) = 0.7 (0.6–0.8)	

RR, relative risk; OR, odds ratio; MET, metabolic equivalent.

TABLE 3. Epidemiologic studies on endometrial cancer and physical activity.

Author, Year, Country	Study Design and Population	Physical Activity Definition	Result	Comment
Sturgeon et al. 1993, USA (85)	Case-control 405 cases 297 population controls	Recreational and nonrecreational physical activity by interview	Inactive vs active Recreational activity: RR (average) = 1.2; RR (inactive) = 1.9 Nonrecreational: RR (average) = 1.3; RR (inactive) = 2.2 (1.2–3.1)	Adjustment made for BMI, age, study area, education, parity, contraceptive, and postmenopausal estrogen use, smoking, and for recreational/ nonrecreational activity decreased the risk of inactivity, but they remained statistically significant
Goodman et al. 1997, USA (27)	332 cases 511 controls from multiethnic Hawaian population	Frequency and duration of recreational and nonrecreational physical activity Summary measure for hours spent in physical activity since age 15 yr.	Highest vs lowest quintile of activity: Recreational activity: OR = 0.9 Nonrecreational activity: OR = 0.8	The risk gradient became significant for nonrecreational activity when adjustment was made by body size
Levi et al. 1993, Italy and Switzerland (51)	Case-control 274 cases 572 noncancer hospital controls	Overall self-rated score for total physical activity and for selected types of physical activity	Less activity vs most activity (ref): Age 25 yr RR (low) = 1.8 (1.2–2.9) RR (lowest) = 2.6 (1.1–6.2) Age 35 yr RR (low) = 2.3 (1.4–3.6) RR (lowest) = 2.7 (1.1–6.2) Age 45 yr RR (low) = 1.7 (1.0–2.7) RR (lowest) = 3.6 (1.6–8.4) Age 55 yr RR (low) = 1.4 (0.8–2.4) 4 (lowest) = 11.9 (4.2–33.8) Age 65 yr RR (low) = 1.3 (0.5–3.0) RR (lowest) = 8.5 (2.4–29.8)	The trends became less evident but remained statistically significant when adjustment was made for BMI, age, education, parity, menopausal status, oral contraceptive and estrogen use, estimated caloric intake.
Moradi et al. 1998, Sweden (62)	Cohort study Census population of 1960 and 1970, follow-up 19 yr	Occupational physical activity	Inactive vs highly/very highly active (ref.) 1960 RR (light activity) = 1.10 (1.1–1.2) RR (sedentary) = 1.2 (1.1–1.3) *P* for trend < 0.001 1970 RR (light activity) = 1.2 (1.1–1.2) RR (sedentary) = 1.3 (1.2–1.4) 1960 and 1970 RR (light activity) = 1.2 RR (sedentary) = 1.3 *P* for trend < 0.001	The risks were unaltered by adjustment for place or residence, calendar year of follow-up, and socioeconomic status Data on BMI not available

RR, relative risk; OR, odds ratio.

the potential benefits of physical activity in weight control after breast cancer has been diagnosed and during treatment.

Increased physical activity may reduce the risk of postmenopausal breast cancer in overweight and obese women. (Evidence category C)

COLON CANCER

Several studies have documented a positive relationship between obesity and colon cancer and colon adenoma in both men (25) and women (57), but the findings are not fully consistent (79). The excess risk is most evident with abdominal adiposity (57) and with adolescent obesity (79).

Several case-control and cohort studies have consistently shown that physical activity decreases the risk of colon cancer (Table 2). Some of these studies suggest a dose-response relation between increasing level of activity and decreasing level of cancer risk. Overall, an about 50% reduction in cancer incidence has been observed among subjects with the highest level of physical activity across many studies with heterogeneous designs and variable measures of physical activity, including either occupational or leisure time activity or both. The observed relation is stronger for the distal colon and weak or nonexistent for rectum. Although confounding cannot be fully ruled out, the protective effect appears to persist after controlling for other lifestyle factors, including BMI, diet, and alcohol consumption. The proposed mechanisms include reduced bowel transit time and changes in insulin or prostaglandin metabolism resulting from increased physical activity (7).

The evidence of a strong inverse relationship between physical activity and risk of colon cancer seems unequivocal enough to justify the public health message that modest increases in physical activity of the population will help reduce the incidence of colon cancer.

Increased physical activity reduces the risk of colon cancer in overweight and obese men and women. (Evidence Category C)

ENDOMETRIAL CANCER

The risk of endometrial cancer is associated with increased weight and body size (27). There are several case-control studies showing that inactivity is associated with an increased risk of endometrial cancer even when BMI is taken into account (Table 3). However, confounding by diet and other factors hamper the interpretation of these data.

Physical inactivity increases the risk of endometrial cancer in obese women. (Evidence Category C)

PROSTATE CANCER

Overweight is a risk factor for prostate cancer. The findings concerning the role of physical activity are controversial. Some case-control (97) and cohort studies (26,78) in which allowance has been made for BMI show no relationship between physical activity and prostate cancer, whereas some cohort studies (1,48,67) show a protective effect. A protective effect of exercise against benign prostatic hyperplasia has been observed in the U.S. Health Professionals Study, but the results were not controlled for weight (70).

RESEARCH PRIORITIES CONCERNING THE ROLE OF PHYSICAL ACTIVITY IN OBESITY-RELATED CANCERS

Most epidemiological studies of cancer, obesity, and physical activity to date are beset with major methodological weaknesses, including problems in assessing physical activity and inadequate control of confounding factors. Because of the complex interrelationships between lifestyle variables, large long-term cohort studies are needed with careful designs, including proper definition and size of the study population, maintenance of high response rates, and improved and standardized methods of measuring factors linked to lifestyle and behavior.

It is not known whether the type, intensity, and timing in life cycle are important determinants of the protective effect of physical activity. There is a need for better assessment of the mode, intensity, and timing of physical activity in both observational and intervention studies.

There has been little research on the effects of physical activity and weight reduction on the progression of hormone-dependent cancers and colon cancer. Long-term RCT of exercise and dietary intervention in patients with these cancers are needed.

GALLBLADDER DISEASE

The risk of gallstones increases with adult weight (12) and weight gain and is also related to central location of fat (61). Rapid weight loss is a strong predictor of gallstone formation, especially in women (54). The early studies on the role of physical activity in gallbladder disease (3,20,37,38,74,89,96) yielded controversial results, whereas most later studies suggest a protective effect for physical activity (Table 4). A distinct protective effect of physical activity against the development of symptomatic gallstones was recently observed in the U.S. Health Professionals Study (50). The findings suggest that symptomatic gallstone disease could be prevented by physical exercise even beyond its benefit for control of body weight. The mechanisms by which exercise may influence gallstone pathogenesis are poorly understood (94).

Research priorities. The protective effect of exercise against gallstones has been documented in the U.S. male population. Large-scale, high-quality epidemiological studies on well-defined patterns of physical activity (see section on research priorities in cancers) are needed to evaluate the effects of exercise in women and in other populations.

Also, RCT are needed to assess whether physical activity can prevent the formation of gallstones in obese people during and after weight reduction.

OSTEOARTHRITIS

Obesity is a strong risk factor of osteoarthritis of the knee (14,16,56) and the hip (7,11). Excess weight contributes to disability in patients with osteoarthritis (95). Weight loss reduces the risk of developing knee osteoarthritis (15), but its effect on the progression of the disease is unknown.

Some studies suggest that vigorous physical activity may predispose to osteoarthritis by the means of mechanical insult to the joint (9,16). An increased risk has been associated with strenuous sports (41) and with competitive but not recreational running (43). A beneficial effect of physical exercise in the prevention of osteoarthritis has not been demonstrated in any study thus far. Both physical exercise therapy (11) and weight reduction (58) may alleviate the symptoms of arthritis, but few studies have addressed the simultaneous effects of these modalities. One small uncontrolled study combining exercise with weight reduction (10) suggested that physical exercise may help preserve lean body mass during weight loss in obese patients with rheumatoid arthritis, but no effect of symptoms was observed in this study.

BACK PAIN

Obesity and low physical activity are among the proposed risk factors of low back pain, but the findings are controversial (30,49,100). While many RCT show therapeutic benefits of exercise (17,65,66) and deleterious effect of bed rest (55) in the treatment of back pain, the evidence of benefits of physical activity in back pain is equivocal (13,55).

The evidence of benefits of physical activity, beyond the effect on body weight regulation, in the prevention and treatment of osteoarthritis and back pain is inconclusive. Given the public health importance of these conditions and the theoretical benefits of weight control and physical exercise in these conditions, more research in this area is needed.

RESEARCH PRIORITIES CONCERNING THE ROLE OF PHYSICAL ACTIVITY IN OBESITY-RELATED MUSCULOSKELETAL DISORDERS

The research priorities concerning the role of physical activity in obesity-related musculosceletal disorders are as follows:

1. High-quality epidemiologic studies are needed to assess the preventive potential of physical activity and its various dimensions, including types of activity,

TABLE 4. Epidemiologic studies on gallbladder disease and physical activity.

Author, Year, Country	Study Design and Population	Gallbladder Disease	Physical Activity Definition	Results	Comment
Williams and Johnston 1980, Canada (99)	Case-control: 97 women aged 15–50 yr	Radiography or history of cholecystectomy	Physical activity questionnaire	Light activities mainly: 78% cases 22% controls ($P = 0.01$)	
Jorgensen et al. 1989, Denmark (38)	Case-control: 4087 population of Copenhagen County	Ultrasonography	Recorded physical activity at work and leisure time	Active vs inactive: M: OR = 1.3	Adjusted for age, maximum BMI, familial gallstones, education
Kato et al. 1992, USA (39)	Prospective cohort: 7831 Japanese-American men	Radiography	Questionnaire on leisure time physical activity	Low vs higher activity: RR (Intermediate) = 0.9–0.7 RR (Highest) = 0.6 (0.5–0.8) $P < 0.01$ for trend	Results unchanged after adjustment for age, smoking, alcohol use, BMI, height, total calories, blood pressure, serum glucose, and triglycerides
Kono et al. 1995, Japan (40)	Case-control Military men aged 49–55 undergoing a health examination 31 ostcholecystectomy cases 41 cases with gallstones 2156 controls	Ultrasonography	Questionnaire about weekly frequency and time spent in exercise	Low vs higher activity: OR (Medium) = 1.6 OR (Highest) = 2.0 (0.8–4.9)	Adjusted for BMI, smoking, alcohol use, glucose tolerance, hospital and military rank
Ortega et al. 1997, Spain (68)	Case-control 54 cases 46 controls	Ultrasonography	Questionnaire about time spent in various daily activities, calculated as activity h/d	Cases spent 40% fewer hours than controls in physically active tasks ($P < 0.0001$)	
Sahi et al. 1998, USA (73)	Prospective cohort (Harvard alumni study) 16785 men aged 15–25 yr, with long follow-up	Self-reported physician-diagnosed gallbladder disease	Baseline: physical activity during college Follow-up: reported time used in specified physical activities, summed up to estimate total weekly energy expenditure in kcal	Active vs inactive: Baseline physical activity RR (<5 h/wk vs >5 h/wk) = 0.9 Follow-up physical activity (<500 kcal/wk = 1.0, ref.) RR (500–999) = 1.2 RR (up to 2000) = 1.1 RR (up to 3500) = 1.3 RR (>3500) = 0.7	Adjusted for age, BMI, BMI change, calendar year, smoking
Leizmann et al. 1998, USA (50)	Prospective cohort (US Health Professionals Study), 45813 U.S. men aged 40–75 yr 828 new cases	New symptomatic gallstones diagnosed by ultrasound or radiography	Baseline: Questionnaire reported nonoccupational physical activity during the previous year, calculated as weekly MET-units. Follow-up: average time spent in specified activities	Highest vs lowest quintile (ref.): *Total physical activity* RR = 0.7 (0.6–0.9) *P* for trend <0.001 *Vigorous activity* Age 40–64 yr RR = 0.5 (0.4–0.7) *P* for trend <0.001 Age ≥65 yr RR = 0.8 (0.5–1.3) *P* for trend >0.2 *Nonvigorous activity* Age 40–64 yr RR = 0.9 (0.7–1.2) Age ≥65 yr RR = 0.9 (0.7–1.3) Significant protection by specific type of exercise (jogging, racquet sports but not walking, stair climbing etc.)	Adjusted for BMI at age 21 and in 1986, diabetes, smoking, intake of cholesterol lowering drugs and NSAIDs, alcohol use, fiber, carbohydrates

NSAID, nonsteroidal anti-inflammatory drugs; RR, relative risk; OR, odds ratio.

intensity, duration, frequency, changes in activity levels over time for various chronic disease and functional end points, including arthritis and low back pain. These studies should be designed to take into account and explore the complex interrelationships between physical activity, body weight, lifestyle, diet, and health.

2. Intervention and observational studies of weight loss with various dimensions of physical activity as defined above in the progression of established osteoarthritis of the knee and hip are needed.
3. RCT to test the hypothesis that certain types of exercise would be beneficial during weight reduction in patients with osteoarthritis.

OTHER COMPLICATIONS

Obesity is a risk factor for obstructive sleep apnea (101), which improves by weight reduction (87). Exercise could improve sleep apnea by facilitating weight loss and also perhaps by stimulating the respiratory drive. In an uncontrolled trial of 11 patients, a 2-h supervised exercise session twice weekly for 6 months improved the symptoms of sleep apnea although the body weight remained unchanged (63).

Obesity is associated with reproductive disorders including polycystic ovary syndrome, menstrual disorders, infertility, miscarriage, and pregnancy complications. These problems are ameliorated by weight reduction (64). We identified no studies that have specifically examined the

relationship between these conditions and physical activity in obese patients.

Research priorities. RCT are needed to test whether exercise alone or combined with weight reduction by diet is effective in the treatment of sleep apnea in obese subjects and in normalizing reproductive abnormalities in obese women.

HEALTH-RELATED QUALITY OF LIFE

Obese persons often have limitations in psychological and physical well-being and in other aspects of the day-to-day life, often summarized under the multidimensional concept of health-related quality of life (HRQL) (29,75,86). Weight reduction appears to improve some dimensions of HRQL (76), but the few data are difficult to interpret, especially in less severe obesity (76).

Physical activity improves several aspects of HRQL (71), including psychological and physical functioning and perceived health (84). Some uncontrolled trials (22,28,45,72) have shown an improvement of mood and HRQL in obese patients in weight loss programs with exercise, but the possible independent contribution of exercise cannot be determined from these studies. Exercise has also been reported to be useful in the treatment of binge-eating disorder (52).

Research priorities. RCT are needed to determine whether physical activity improves the various aspects of health-related quality of life beyond its effect of weight loss. Valid assessment of HRQL should be included in all intervention studies of obese persons.

Also, RCT are needed to study the effects of physical activity in binge eating disorder.

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Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues

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ABSTRACT

BLAIR. S. N. and S. BRODNEY. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. *Med. Sci. Sports Exerc.*, Vol. 31, No. 11, Suppl., pp. S646–S662, 1999. **Purpose:** The purpose of this review was to address three specific questions. 1) Do higher levels of physical activity attenuate the increased health risk normally observed in overweight or obese individuals? 2) Do obese but active individuals actually have a lower morbidity and mortality risk than normal weight persons who are sedentary? 3) Which is a more important predictor of mortality, overweight or inactivity? **Methods:** We initially identified more than 700 articles that included information on the exposure variables of body habitus (body mass index, body composition, or body fat pattern) and physical activity habits, and on outcomes such as morbidity or mortality. To be included in the review, we required that an article include an analysis of one of our outcomes by strata of the two exposure variables. We excluded review articles and reports of cross-sectional analyses. We used an evidence-based approach to evaluate the quality of the published data. **Results:** We summarized results from 24 articles that met all inclusion criteria. Data were available for the outcomes of all-cause mortality, cardiovascular disease mortality, coronary heart disease (CHD), hypertension, type 2 diabetes mellitus, and cancer. Summary results for all outcomes except cancer were generally consistent in showing that active or fit women and men appeared to be protected against the hazards of overweight or obesity. This apparent protective effect was often stronger in obese individuals than in those of normal weight or who were overweight. There were too few data on cancer to permit any conclusions. **Conclusions:** There are no randomized clinical trials on the topics addressed in this review. All studies reviewed were prospective observational studies, so all conclusions are based on *Evidence Category C.* The conclusions for the three questions addressed in the review are: 1) regular physical activity clearly attenuates many of the health risks associated with overweight or obesity; 2) physical activity appears to not only attenuate the health risks of overweight and obesity, but active obese individuals actually have lower morbidity and mortality than normal weight individuals who are sedentary, and 3) inactivity and low cardiorespiratory fitness are as important as overweight and obesity as mortality predictors. Research needs include extending current observations to more diverse populations, including more studies in women, the elderly, and minority groups, assessment methods need to be improved, and randomized clinical trials addressing the questions discussed in this review should be undertaken. Owing to size, complexity, and cost, these trials will need to be designed with valid noninvasive measures of subclinical disease processes as outcomes. **Key Words:** PHYSICAL ACTIVITY, CARDIORESPIRATORY FITNESS, BODY MASS INDEX, OVERWEIGHT, OBESITY, BODY FAT DISTRIBUTION, EPIDEMIOLOGY

Overweight and obesity are directly related to increased risk of several chronic diseases and impaired physical function, whereas physical activity and cardiorespiratory fitness are most often inversely associated with similar increased risks (25,38). In observational studies both overweight and obesity are correlated with physical activity and cardiorespiratory fitness, with sedentary and unfit persons having greater weight for height, body mass index (BMI, $kg \cdot m^{-2}$), and percent body fat (2,5–8,11,14,15,17,32). Data from controlled clinical trials have shown that increases in physical activity result in weight loss and changes in body composition and fat distribution (4,9,16,35,36,40–42). Thus, it is possible that some, perhaps much, of the overweight and obesity seen in U.S. populations is caused by a sedentary, physically inactive lifestyle. This topic is reviewed in detail elsewhere in these proceedings.

It is difficult to know how much of the higher morbidity and mortality seen in overweight or obese individuals results from the elevated weight and how much results from inactivity. Whereas most observational studies of the relationship between physical activity or cardiorespiratory fitness and morbidity or mortality include adjustment for some aspect of body composition, many of the studies of overweight or obesity and morbidity and mortality have not included data on physical activity.

Barlow et al. (2) published the first report that included analyses specifically designed to evaluate the relation of cardiorespiratory fitness to mortality in individuals classified as normal, overweight, or obese based on their BMI. Based on their results these investigators suggested the

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hypothesis that moderate to high levels of cardiorespiratory fitness protect against much, if not most, of the increased mortality that accompanies overweight and obesity. This hypothesis is the basis for this review of the available evidence.

The material presented in this review addresses three specific questions:

1. Do higher levels of physical activity or cardiorespiratory fitness attenuate the increased risk of morbidity and mortality in overweight or obese persons?
2. If the evidence supports the hypothesis that higher levels of physical activity and cardiorespiratory fitness attenuate the increased risk associated with overweight and obesity, do overweight or obese individuals who are physically active or fit actually have a lower risk of morbidity and mortality than normal weight individuals who are sedentary (i.e., what is the magnitude of the protective effect of activity or fitness in overweight and obese individuals)?
3. Which is a more important predictor of mortality in individuals, overweight and obesity or inactivity and low fitness?

METHODS AND DEFINITIONS

We evaluated several different measures of health, overweight or obesity, and sedentary habits. Outcome variables considered for this report include all-cause mortality, the presence or development of cardiovascular disease, hypertension, stroke, CHD, type 2 diabetes mellitus, cancer, and functional health status. We found no articles on stroke or functional health status that met the inclusion criteria described below, and these variables are not mentioned further. Exposure variables included body habitus (BMI or body composition as estimated by hydrostatic weighing or skinfold thickness, and fat distribution as estimated by waist girth), and evidence of a sedentary life style (low levels of self-reported occupational or leisure-time physical activity or low levels of cardiorespiratory fitness as determined by objective laboratory assessment). These exposure variables have limitations. Current opinion holds that it is the amount and distribution of body fat that are the principal causes of high rates of chronic disease seen in overweight or obese individuals, but few studies have included direct measurement of these parameters. Physical activity is difficult to quantify, and self-report of activity patterns is the most common measurement approach used in studies of physical activity and health. Although many of the activity questionnaires and procedures have established validity, the amount of variance explained in criterion measures of energy expenditure by these methods is low to moderate (27). Cardiorespiratory fitness is assessed objectively, but it is relatively expensive and logistically complicated to perform in epidemiological studies. Limitations of cardiorespiratory fitness assessments are that these measurements only reflect activity over recent months before the assessment and are at least partly determined by heredity.

Identifying Source Material

Our objective was to identify all published reports that were from prospective investigations and included one of the outcome measures listed above, in which results were presented in strata of body habitus cross-tabulated by categories of physical activity or cardiorespiratory fitness. We excluded reports in which outcomes were given by strata of body habitus with adjustment for activity or fitness or by strata of activity or fitness with adjustment for body habitus. Such reports typically did not include sufficient information that would allow us to fully characterize the independent effect of one exposure variable across the full range of the other. We also excluded review articles and cross-sectional analyses.

Another problem that arises in considering the questions under review in this report is that many of the major studies on obesity and health did not include measurement of physical activity or fitness. In reports on obesity and health in which investigators indicated that they adjusted for physical activity, there is often no, or at best an incomplete, description of how the physical activity was measured. In other instances activity was assessed so crudely, perhaps with a single global question, that little confidence could be placed in the measurement. In these circumstances determination of the true independent effects of body habitus and energy expenditure on outcomes is impossible because of the great difference in the reliability of assessment of the two exposure variables. For these reasons, we believe that the most valid approach to evaluate the independent contributions of body habitus and energy expenditure to health is to use objective and valid measures of each. Measurement of body habitus and cardiorespiratory fitness in a laboratory or clinical setting meets this criterion, although we also include reports in which physical activity was measured with a standardized method that has been shown to have a strong and graded association with health outcomes.

We began with papers identified in the 1996 publication, *Physical Activity and Health: A Report of the Surgeon General* (38). Next, we performed computer searches with keywords related to our identified outcome measures in combination with keywords related to energy expenditure (physical fitness, cardiorespiratory fitness, exertion, exercise, or physical activity) and body habitus (BMI, overweight, obesity, or fat distribution). We also searched our personal files and reference lists in identified published articles. We initially identified more than 700 potentially relevant articles. After exclusions based on the above criteria, this process identified 24 articles that are included in this report.

Critical Analysis of Published Articles. Both authors reviewed each article on the final list. We summarized results from the articles in Tables 1 - 6, with a different table for each outcome measure. Each table includes the study reference; a brief description of the study population; the method of assessing physical activity or fitness; the method of assessing body habitus; a description of outcomes, the number of events, and the length of follow-up (where

TABLE 1. All-cause mortality by cross tabulation of body habitus and cardiorespiratory fitness or physical activity.

Study	Population	Physical Activity or Fitness	Body Habitus	Outcome	Adjusted For	Results
Cardiorespiratory Fitness Blair et al. 1989 (3)	*N* = 10,244 men and 3,120 women who completed a preventive medical exam between 1971–1981 Excluded men and women with history of chronic disease at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Low fit = least fit 20% Moderate fit = 21–60% High fit = 61–100%	Measured height and weight to calculate BMI <20, 20–25, >25	All-cause mortality *N* = 240 (men) *N* = 43 (women) 110,482 py Mean follow-up = 8 yr	Age	Adjusted death rate/10,000 py BMI <20-Men Low fitness = 155 Moderate fitness = 55 High fitness = 38 BMI 20–25-men Low fitness = 46 Moderate fitness = 26 High fitness = 15 BMI >25-men Low fitness = 48 Moderate fitness = 22 High fitness = 20 BMI <20-women Low fitness = 74 Moderate fitness = 23 High fitness = 10 BMI 20–25-women Low fitness = 28 Moderate fitness = 11 High fitness = 5 BMI >25-women Low fitness = 14 Moderate fitness = 20 High fitness = 6
Barlow et al. 1995 (2)	*N* = 25,389 men who completed a preventive medical exam between 1971–1989 No exclusions for chronic disease at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Low fit = least fit 20% Moderate fit = 21–60% High fit = 61–100%	Measured height and weight to calculate BMI <27.0, 27.0–30.0, >30.0	All-cause mortality *N* = 673 212,364 py Mean follow-up = 8.5 yr	Age	Adjusted RR (95% CI); [death rate/10,000 py] BMI <27.0 Low fitness = 1.0; [52.1] Moderate fitness = 0.49 (0.31–0.76); [28.6] High fitness = 0.34 (0.21–0.57); [20.0] BMI 27.0–30.0 Low fitness = 1.0; (49.1) Moderate fitness = 0.61 (0.38–0.96); (29.8) High fitness = 0.40 (0.24–0.68); (19.7) BMI >30.0 Low fitness = 1.0; [62.1] Moderate/high fitness = 0.29 (0.17–0.49); [18.0]
Lee 1998 (19)	*N* = 21,856 men aged 30–83 who completed a preventive medical exam between 1971–1989 Excluded men with history of myocardial infarction, stroke, or cancer at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Unfit = least fit 20% Fit = all others	Measured height and weight to calculate BMI 19.0–<25.0, 25.0–<27.8, ≥27.8	All-cause mortality *N* = 427 176,189 py Mean follow-up = 8.1 yr	Age, exam year, smoking habit, alcohol intake	Adjusted RR (95% CI) BMI 19.0–<25.0 Fit = 1.00 Unfit = 2.25 (1.59–3.17) BMI 25.0–<27.8 Fit = 0.96 (0.73–1.26) Unfit = 1.68 (1.19–2.37) BMI ≥27.8 Fit = 1.08 (0.77–1.50) Unfit = 2.24 (1.68–2.98) Similar results for nonsmokers and nonsmokers without early mortality

TABLE 1. continued.

Lee et al. 1999 (18)	N = 21,925 men aged 30–83 who completed a preventive medical exam between 1971–1989 Excluded men with history of myocardial infarction, stroke, or cancer at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Unfit—least fit 20% Fit—all others	Measured % body fat by hydrostatic weighing or sum of 7 skinfolds Body fatness (%): Lean <16.7, Normal 16.7–<25.0, Obese ≥25.0 Waist circumference (cm), subgroup of 14,043 men: Low <87, Moderate 87–<99, High ≥99	All-cause mortality N = 428 deaths 176,742 py Mean follow-up = 8 yr	Age, exam year, smoking habit, alcohol intake, parental history of ischemic heart disease	Adjusted RR (95% CI) Lean (<16.7% body fat) Fit = 1.0 Unfit = 2.07 (1.16–3.69) Normal (16.7–<25.0% body fat) Fit = 0.80 (0.59–1.08) Unfit = 1.62 (1.15–2.30) Obese (≥25.0% body fat) Fit = 0.92 (0.65–1.31) Unfit = 1.90 (1.39–2.60) Waist circumference Low (<87 cm) Fit = 1.0 Unfit = 4.88 (2.2–10.83) Moderate (87–<99 cm) Fit = 1.05 (0.66–1.67) Unfit = 2.05 (1.08–3.87) High (≥99 cm) Fit = 0.95 (0.54–1.66) Unfit = 2.40 (1.41–4.07)
Physical Activity Paffenbarger et al. 1986 (28)	N = 16,936 male Harvard alumni aged 35–74 Excluded men with history of coronary heart disease at baseline	Self-reported physical activity <500, 500–1999, ≥2000 kcal·wk^{-1}	Self-report of height and weight to calculate BMI <23.9, 23.9–25.2, ≥25.3	All-cause mortality N = 1413 Follow-up = 12–16 yr	Age	Adjusted death rates/10,000 py BMI <23.9 <500 = 113 500–1999 = 69 ≥2000 = 56 BMI 23.9–25.2 <500 = 81 500–1999 = 73 ≥2000 = 45 BMI ≥25.3 <500 = 85 500–1999 = 66 ≥2000 = 60
Rosengren, A. and L. Wilhelmsen 1997 (32)	N = 7,142 men aged 47–55 Excluded men with history of clinical coronary heart disease or a positive Rose questionnaire at baseline	Self-reported leisure time physical activity Sedentary, moderately active, regular exercise or athletic training	Measured height and weight to calculate BMI <24.1, 24.1–26.6, >26.6	All-cause mortality N = 2,640 Mean follow-up = 20 yr	Age	Adjusted death rate/1,000 py BMI <24.1 Sedentary = 20.9 Moderately active = 15.6 Regular exercise/athletic training = 10.2 BMI 24.1–26.6 Sedentary = 17.5 Moderately active = 13.6 Regular exercise/athletic training = 9.4 BMI >26.6 Sedentary = 20.6 Moderately active = 15.8 Regular exercise/athletic training = 14.1

BMI, body mass index (kg·m^{-2}); py, person-years; RR, relative risk; CI, confidence interval.

TABLE 2. Cardiovascular disease (CVD) mortality by cross tabulation of body habitus and cardiorespiratory fitness.

Study	Population	Physical Activity or Fitness	Body Habitus	Outcome	Adjusted For	Results
Lee et al. 1998 (19)	N = 21,856 men aged 30–83 who completed a preventive medical exam between 1971–1989 Excluded men with history of myocardial infarction, stroke, or cancer at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Unfit—least fit 20% Fit—all others	Measured height and weight to calculate BMI 19.0–<25.0, 25.0–27.8, ≥27.8	CVD mortality N = 144 (ICD codes: 390–449.9) 176,189 py Mean follow-up = 8.1 yr	Age exam year, smoking habit, alcohol intake	Adjusted RR (95% CI) BMI 19.0–<25.0 Fit = 1.00 Unfit = 2.8 (1.43–5.49, P = 0.003) BMI 25.0–<27.8 Fit = 1.8 (1.07–3.01, P = 0.03) Unfit = 3.7 (2.05–6.50, P < 0.001) BMI ≥27.8 Fit = 1.8 (not given) Unfit = 4.8 (2.85–8.05, P < 0.001)
Lee et al. 1999 (18)	N = 21,925 men aged 30–83 who completed a preventive medical exam between 1971–1989 Excluded men with history of myocardial infarction, stroke, or cancer at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Unfit—least fit 20% Fit—all others	Measured % body fat by hydrostatic weighing or sum of skinfolds Body fatness (%): Lean <16.7, Normal 16.7–<25.0, Obese ≥25.0	CVD mortality N = 144 (ICD codes: 390–449.9) 176,742 py Mean follow-up = 8 yr	Age, exam year, smoking habit, alcohol intake, parental history of ischemic heart disease	Adjusted RR (95% CI) Lean (<16.7% body fat) Fit = 1.0 Unfit = 3.16 (1.12–8.92) Normal (16.7–<25.0% body fat) Fit = 1.43 (0.77–2.67) Unfit = 2.94 (1.48–5.83) Obese (≥25.0% body fat) Fit = 1.35 (0.66–2.76) Unfit = 4.11 (2.20–7.68) Unfit men had a higher risk of CVD mortality than fit men in all fat and fat-free mass categories
Farrell et al. 1998 (10)	N = 25,341 men who completed a preventive medical exam between 1971–1989 No exclusions for chronic disease at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Low fit = least fit 20% Moderate fit = 21–60% High fit = 61–100%	Measured height and weight to calculate BMI <27.0, ≥27.0	CVD mortality N = 226 (ICD codes: 390–449.9) 211,996 py Mean follow-up = 8.4 yr	Age, year of baseline exam, health status, smoking status	Adjusted death rate/10,000 py BMI <27.0 Low = 14.5 Moderate = 9.6 High = 6.7 P < 0.001 BMI ≥27.0 Low = 17.9 Moderate = 10.6 High = 9.5 P = 0.24

BMI, body mass index (kg·m^{-2}); py, person-years; RR, relative risk; CI, confidence interval.

TABLE 3. Coronary heart disease (CHD) by cross tabulation of body habitus and physical activity.

Study	Population	Physical Activity or Fitness	Body Habitus	Outcome	Adjusted For	Results
Paffenbarger et al. 1978 (31)	N = 16,936 Harvard male alumni aged 35–74 Excluded men reporting a diagnosed heart attack at baseline	Self-reported physical activity Physical activity index <2000 kcal·wk^{-1} and ≥2000 kcal·wk^{-1}	Self-report of height and weight to calculate BMI <26.7, ≥26.7	CHD (fatal and nonfatal heart attacks) N = 572 117,680 py Follow-up = 6–10 yr	Age	Adjusted RR BMI <26.7 <2000 kcal·wk^{-1} compared to ≥2000 kcal·wk^{-1}. RR = 1.68, $P < 0.001$ BMI ≥26.7 <2000 kcal·wk^{-1} compared to ≥2000 kcal·wk^{-1} RR = 1.33, $P = 0.206$
Morris et al. 1980 (24)	N = 17,944 male executive grade civil servants Excluded men with CHD at baseline	Self-report of 5-min by 5-min record of time spent in physical activity on previous Friday and Saturday	Self-report of height and weight to calculate BMI <23, 23.1–25.0, 25.1–28.0, ≥28.1	CHD (1st clinical episode) N = 1,138 150,000 py Mean follow-up = 8.5 yr	Age	Rate % BMI <23.0 Vigorous exercise = 2.4 No vigorous exercise = 5.2 BMI 23.1–25.0 Vigorous exercise = 3.3 No vigorous exercise = 6.9 BMI 25.1–28.0 Vigorous exercise = 4.4 No vigorous exercise = 6.9 BMI ≥28.1 Vigorous exercise = 0.7 No vigorous exercise = 8.8
Paffenbarger et al. 1984 (29)	N = 16,936 Harvard male alumni aged 35–74 Excluded men with CHD at baseline	Self-reported physical activity Physical activity index <500, 500–1999, ≥2000	Self report of height and weight to calculate BMI <23.9, 23.9–25.2, ≥25.3	CHD (fatal and nonfatal heart attacks) N = 572 117,680 py Follow-up = 6–10 yr	Age	RR and rates not printed in paper RR of CHD lower by progressively higher levels of physical activity (<500, 500–1999, ≥2000 kcal·wk^{-1}) for each BMI stratum (<23.9, 23.9–25.2, ≥25.3). CHD inversely related to exercise ($P < 0.001$) and CHD positively related to BMI ($P < 0.01$)
Salonen et al. 1988 (33)	N = 15,088 Finnish men and women aged 30–59 Excluded men and women with cardiovascular disease or inability to participate in physical activity at baseline	Self-reported physical activity from 8 questions 4 questions on LTPA and 4 on occupational activity	Measured height and weight to calculate BMI <27.0, ≥27.0	Ischemic heart disease mortality N = 90 men N = 12 women Mean follow-up = 6 yr	None	Crude IHD rate/1,000 py BMI <27.0: men Active occupation/High LTPA = 8.1 Active occupation/LTPA = 14.1 Sedentary occupation/High LTPA = 19.9 Sedentary occupation/Low LTPA = 25.9 BMI ≥27.0: men Active occupation/High LTPA = 7.4 Active occupation/Low LTPA = 17.1 Sedentary occupation/High LTPA = 8.6 Sedentary occupation/Low LTPA = 41.7 BMI <27.0: women Active occupation/High LTPA = 0.5 Active occupation/Low LTPA = 3.2 Sedentary Occupation/High LTPA = 1.2 Sedentary Occupation/Low LTPA = 0.0 BMI ≥27.0: women Active occupation/High LTPA = 2.1 Active occupation/Low LTPA = 1.0 Sedentary Occupation/High LTPA = 6.2 Sedentary Occupation/Low LTPA = 0.0

TABLE 3. continued.

Morris et al. 1990 (23)	*N* = 9,376 male executive grade civil servants aged 45–64 Excluded men with clinical history of CHD at baseline	Self-reported physical activity in previous 4 wk 4 groups: 1) frequent/intense, 2) next lesser degree, 3) residual/ little, 4) none	Self-report of height and weight to calculate BMI <24.0, 24–26.9, ≥27.0	CHD (fatal and nonfatal heart attacks) *N* = 474 87,563 py Mean follow-up = 9.3 yr	Age, family history, stature, energy output, questions on health beliefs, cigarette smoking, health conscious diet, weight in past year, BMI, cardiovascular history	Adjusted attack rate/1,000 py BMI <24.0 Group 1 = 1.9 Group 2 = 3.4 Group 3 = 4.3 Group 4 = 5.5 BMI 24–26.9 Group 1 = 2.4 Group 2 = 6.0 Group 3 = 5.7 Group 4 = 6.2 BMI ≥27.0 Group 1 = 1.3 Group 2 = 4.8 Group 3 = 7.1 Group 4 = 7.3
Rosengren and Wilhelmsen, 1997 (32)	*N* = 7,142 men aged 47–55 Excluded men with history of clinical CHD or a positive Rose questionnaire at baseline	Self-reported LTPA Sedentary, moderately active, regular exercise or athletic sports	Measured height and weight to calculate BMI <24.1, 24.1–26.6, >26.6	CHD mortality *N* = 672 Mean follow-up = 20 yr	Age	Adjusted death rate/1000 BMI <24.1 Sedentary = 4.6 Moderately active = 5.0 Regular exercise/athletic sports = 3.0 BMI 24.1–26.6 Sedentary = 6.2 Moderately active = 3.8 Regular exercise/athletic sports = 3.1 BMI >26.6 Sedentary = 7.4 Moderately active = 5.3 Regular exercise/athletic sports = 4.3
Haapanen et al. 1997 (12)	*N* = 865 Finnish men aged 35–63 Excluded men with disease or inability to participate in physical activity at baseline	Self-reported physical activity by mailed questionnaire (23 questions) LTPA categorized as low, moderate, high	Self-report of height and weight to calculate BMI ≤27.0, >27.0	CHD (fatal and nonfatal) *N* = 108 Mean follow-up = 10 yr	Age	Adjusted incidence rate/1,000 py BMI ≤27.0 Low = 14.3 Moderate = 9.6 High = 7.3 BMI >27.0 Low = 20.5 Moderate = 11.9 High = 11.1

BMI, body mass index ($kg \cdot m^{-2}$); py, person-years; RR, relative risk; LTPA, leisure time physical activity; CHD, coronary heart disease.

TABLE 4. Hypertension (HTN) by cross tabulation of body habitus and physical activity.

Study	Population	Physical Activity or Fitness	Body Habitus	Outcome	Adjusted For	Results
Paffenbarger et al. 1983 (26)	N = 14,998 male Harvard alumni aged 35–74 Excluded men with physician-diagnosed HTN at baseline	Self-reported physical activity by mailed questionnaire Vigorous sports played (yes or no)	Self-report of height and weight to calculate BMI <22.5, 22.5–23.8, 23.9–25.2, 25.3–26.6, ≥26.7	Self-reported incidence of physician-diagnosed HTN N = 681 105,662 py Follow-up 6–10 yr	Age	Adjusted RR of not participating in vigorous sports as compared with participating in vigorous sports BMI <22.5 RR = 0.91 (P = 0.554) BMI 22.5–23.8 RR = 1.27 (P = 0.268) BMI 23.9–25.2 RR = 1.23 (P = 0.248) BMI 25.3–26.6 RR = 1.50 (P = 0.022) BMI ≥26.7 RR = 1.65 (P = 0.01)
Paffenbarger et al. 1991 (30)	N = 5,463 male Univ. of Pennsylvania alumni aged 40–54 Excluded men with physician-diagnosed HTN at baseline	Self-reported physical activity by mailed questionnaire Vigorous sports played (yes or no)	Self-report of height and weight to calculate BMI <22.5, 22.5–23.9, 24.0–25.9, 26.0–26.9, ≥27.0	Self-reported incidence of physician-diagnosed HTN N = 739 cases 83,731 py Follow-up = 15 yr	Age	Adjusted RR of not participating in vigorous sports as compared with participating in vigorous sports BMI <22.5 RR = 1.25 BMI 22.5–23.9 RR = 1.25 BMI 24.0–25.9 RR = 1.07 BMI 26.0–26.9 RR = 1.25 BMI ≥27.0 RR = 1.30

BMI, body mass index (kg·m^{-2}); py, person-years; RR, relative risk.

applicable); confounding variables considered in the analyses; and a summary of the study results. We used the evidence-based approach for rating the quality of evidence that was described in the report on obesity treatment guidelines from the National Institutes of Health (25). There are no randomized controlled clinical trials that provide data on the questions under consideration in this report (Evidence Category A or B), and we did not include any consensus judgment reports (Evidence Category D). Therefore, all reports included in our results are classified in Evidence Category C (prospective observational studies).

RESULTS

We present separate tables on the relationship of body habitus and physical activity or cardiorespiratory fitness to the outcomes of all-cause mortality, cardiovascular disease mortality, CHD, hypertension, type 2 diabetes mellitus, and cancer.

All-cause Mortality

A summary of the review of reports on all-cause mortality is presented in Table 1. There are four separate reports on cross-tabulations of cardiorespiratory fitness and BMI, body composition, or fat distribution. All these reports are from the Aerobics Center Longitudinal Study (ACLS). The first of these papers, published in 1989, had a short period of follow-up and the number of deaths was small, especially in women (3). Another limitation of this report is that the characterization of body habitus was limited to BMI categories of underweight, normal weight, and moderately overweight. The primary purpose of the study was to evaluate the relation of cardiorespiratory fitness to mortality, and detailed analyses are lacking to clarify the independent effects of the two exposures. Nonetheless, fit men and women tended to have lower all-cause mortality rates than unfit individuals in each BMI stratum. In this population, the highest mortality occurred in individuals with a BMI < 20. This is most likely a result of a lack of adjustment for smoking status and the presence of chronic disease at baseline.

The later reports from the ACLS focus specifically on the questions addressed in this review (2,18,19). These reports are from extended follow-up in the ACLS cohort, but because the number of events remains low in women, the reports include data for men only. Different selection criteria were used for the three studies, with the principal difference being whether individuals with chronic disease were excluded at baseline. The studies also differ in the classification and in the specific measure of body habitus used in analyses. Results, nevertheless, are consistent across the three studies. Men with higher levels of BMI, percent body fat, fat mass, fat-free mass, and waist girth who also were fit had lower death rates than unfit men with similar levels of body habitus measurements. In fact, in all analyses fit men with higher body habitus measurements, indicating obesity and/or overweight, had death rates not significantly different from fit men with low body habitus measurements. In these data men with high body habitus measurements had elevated mortality only if they also were unfit.

We also found two studies that used all-cause mortality as the outcome and physical activity and BMI as the exposure variables (28,32). The results were similar to those seen for cardiorespiratory fitness in the ACLS cohort. Active men in each BMI stratum had lower death rates than inactive men in the same stratum, and active men in the high BMI group

TABLE 5. Type 2 diabetes mellitus (DM) by cross tabulation of body habitus and physical activity or cardiorespiratory fitness.

Study	Population	Physical Activity or Fitness	Body Habitus	Outcome	Adjusted For	Results
Physical activity						
Helmrich et al. 1991 (13)	N = 5,990 male Univ. of Pennsylvania alumni aged 39–68 Excluded men with DM at baseline	Self-reported LTPA <500, 500–1999, ≥2000 kcal·wk^{-1}	Self-report of height and weight to calculate BMI <24.0, 24.0–25.99, ≥26.0	Self-report of physician diagnosis of Type 2 DM N = 202 cases 98,524 py Follow-up = 14 yr	Age	Adjusted incidence rate/10,000 py BMI <24.0 <500 = 12.1 500–1999 = 11.6 ≥2000 = 12.0 BMI 24.0–25.99 <500 = 25.9 500–1999 = 25.9 ≥2000 = 16.9 BMI ≥26.0 <500 = 49.5 500–1999 = 38.9 ≥2000 = 33.2
Manson et al. 1991 (22)	N = 87,253 US female nurses aged 34–59 Excluded women with DM, cardiovascular disease, and cancer in 1980	Self-reported physical activity Single question on frequency of vigorous activity per week (<1/week or ≥1/week)	Self-report of height and weight to calculate BMI <27.0, ≥27.0	Self-report of confirmed diagnosis of type 2 DM N = 1303 670,397 py Follow-up = 8 yr	Age	Adjusted RR (95% CI) BMI <27.0 <1/wk = 1.0 ≥1/wk = 0.73 (0.59–0.89), $P < 0.01$ BMI ≥27.0 <1/wk = 1.0 ≥1/wk = 0.79 (0.69–0.90), $P < 0.01$
Manson et al. 1992 (21)	N = 21,271 male physicians aged 40–84 Excluded men with DM, myocardial infarction, stroke, or cancer at baseline	Self-reported physical activity Single question on frequency of vigorous activity per week (<1/wk or ≥1/wk)	Self-report of height and weight to calculate BMI <23.0, 23.0–24.4, 24.5–26.4, >26.4	Self-report of physician-diagnosis of Type 2 DM N = 285 105,141 py Mean follow-up = 5 yr	Age	Adjusted incidence rate/100,000 py BMI <23.0 <1/wk = 101 ≥1/wk = 123 BMI 23.0–24.4 <1/wk = 187 ≥1/wk = 163 BMI 24.5–26.4 <1/wk = 353 ≥1/wk = 245 BMI >26.4 <1/wk = 733 ≥1/wk = 448

TABLE 5. continued.

Burchfiel et al. 1995 (6)	*N* = 8,006 Japanese-American men aged 45–68 Excluded men with DM at baseline	Self-reported physical activity in previous 24 h Basal, sedentary, slight, moderate, heavy	Measured height and weight to calculate BMI Mean lower 4 BMI quintiles = 22.7 Upper BMI quintile = 28.2	Type 2 DM Self-report use of DM medication at 1 or 2 follow-up exams *N* = 391 Follow-up = 6 yr	Age	Adjusted cumulative incidence/1,000 All men: lower BMI strata-22.7 Basal = 62.4 Sedentary = 45.7 Slight = 52.4 Moderate = 47.2 Heavy = 25.7 *P* = 0.0004 All men: upper BMI strata-28.2 Basal = 112.2 Sedentary = 127.9 Slight = 89.2 Moderate = 84.6 Heavy = 76.3 *P* = 0.0833 Men <225 mg·dL^{-1}: lower BMI strata-22.7 Basal = 52.8 Sedentary = 35.9 Slight = 39.2 Moderate = 30.9 Heavy = 13.0 *P* < 0.0001 Men <225 mg·dL^{-1}: upper BMI strata-28.2 Basal = 60.3 Sedentary = 85.0 Slight = 55.5 Moderate = 58.8 Heavy = 63.8 *P* = 0.9242
Cardiorespiratory fitness Wei et al. 1999 (39)	*N* = 8,633 men aged 30–79 who completed at least 2 medical evaluations between 1970–1995 Excluded men with history of DM or fasting plasma glucose ≥126 mg·dL^{-1} at baseline	Cardiorespiratory fitness—maximal exercise treadmill test Low fit = least fit 20% Moderate fit = 21–60% High fit = 61–100%	Measured height and weight to calculate BMI <27.0, ≥27.0	Type 2 DM *N* = 149 52,588 py Mean follow-up = 6.1 yr	None	Incidence/1,000 py BMI <27.0 Low = 3.5 Moderate = 1.9 High = 1.2 *P* < 0.001 BMI ≥27.0 Low = 7.9 Moderate = 4.1 High = 2.8 *P* = 0.006 (Incidence rates obtained from author)

LTPA, leisure time physical activity; BMI, body mass index (kg·m^{-2}); py, person-years; RR, relative risks; CI, confidence intervals.

TABLE 6. Cancer by cross tabulation of body habitus and physical activity.

Study	Population	Physical Activity or Fitness	Body Habitus	Outcome	Adjusted For	Results
Thune et al. 1997 (37)	*N* = 25,624 pre- and post-menopausal Norwegian women aged 20–69 who completed surveys in 1974–78 and 1977–83 Excluded women with cancer at baseline	Self-reported LTPA in past year Sedentary, moderate, regular exercise	Measured height and weight to calculate BMI <22.8, 22.8–25.7, >25.7	Breast cancer *N* = 351 cases 359,930 py Mean follow-up = 14 yr	Age at entry, height, country of residence, number of children	Adjusted RR (95% CI) for level of physical activity in 1974–78 and 1977–83 surveys: BMI <22.8 Sedentary = 1.0 Moderate = 0.76 (0.43–1.35) Regular = 0.23 (0.09–0.60) *P* for trend = 0.002 BMI 22.8–25.7 Sedentary = 1.0 Moderate = 0.87 (0.40–1.88) Regular = 0.83 (0.33–2.09) *P* for trend = 0.73 BMI >25.7 Sedentary = 1.0 Moderate = 1.14 (0.57–2.27) Regular = 1.38 (0.60–3.17) *P* for trend = 0.42 Similar results were reported for level of physical activity in the 1977–83 survey only
Albanes et al. 1989 (1)	*N* = 5,138 men and 7,407 women age 25–74 Natl. Health and Nutrition Exam. Survey I cohort examined in 1971–75 Excluded men and women with cancer at baseline	Self-reported physical activity in nonrecreational activities Very active, moderately active, quite inactive	Measured height and weight to calculate BMI <22.0, 22.0–26.0, >26.0	All-site cancer confirmed through hospital record/death review *N* = 460 men and 399 women	Age	Adjusted RR (95% CI) BMI <22.0: men Very active = 1.0 Moderately active = 1.4 (0.9–2.2) Quite inactive = 3.1 (1.9–5.2) *P* < 0.0001 BMI 22.0–26.0: men Very active = 1.0 Moderately active = 1.5 (1.1–2.2) Quite inactive = 1.9 (1.1–3.0) *P* = 0.005 BMI >26.0: men Very active = 1.0 Moderately active = 0.8 (0.6–1.1) Quite inactive = 1.2 (0.7–1.3) *P* = 0.83 BMI <22.0: women Very active = 1.0 Moderately active = 1.1 (0.7–1.7) Quite inactive = 1.8 (0.9–3.4) *P* = 0.19 BMI 22.0–26.0: women Very active = 1.0 Moderately active = 1.1 (0.8–1.7) Quite inactive = 2.0 (1.2–3.5) *P* = 0.03 BMI >26.0: women Very active = 1.0 Moderately active = 0.9 (0.7–1.3) Quite inactive = 0.9 (0.5–1.4) *P* = 0.57

TABLE 6. continued.

Slattery et al. 1997 (34)	N = 2,073 first primary cases of colon cancer and 2,466 age and sex-matched controls from Kaiser Permanente Medical Care Program	Interviewer-administered questionnaire on physical activity patterns for past 1, 10, and 20 yr Low, intermediate, high	Measured height and reported weight for referent year to calculate BMI BMI in men: (weight·ht^{-2}) BMI in women: (wt·wt$^{-1.5}$) BMI = low, intermediate, high (not defined)	Colon cancer	Age at diagnosis, family history of colorectal cancer, dietary fiber and calcium, use of aspirin or nonsteroidal anti-inflammatory drugs, dietary cholesterol, energy intake	Adjusted RR (95% CI) Low BMI: men Low = 1.51 (0.96–2.37) Intermediate = 1.30 (0.91–1.85) High = 1.00 Intermediate BMI: men Low = 1.62 (1.02–2.57) Intermediate = 1.63 (1.15–2.30) High = 1.13 (0.74–1.71) High BMI: men Low = 2.64 (1.75–3.98) Intermediate = 2.41 (1.72–3.37) High = 1.41 (0.94–2.11) Low BMI: women Low = 1.17 (0.74–1.85) Intermediate = 0.91 (0.61–1.38) High = 1.00 Intermediate BMI: women Low = 1.34 (0.86–2.09) Intermediate = 0.91 (0.61–1.37) High = 0.95 (0.57–1.59) High BMI: women Low = 1.74 (1.13–2.68) Intermediate = 1.22 (0.82–1.84) High = 0.71 (0.42–1.18)
Levi et al. 1993 (20)	N = 274 histologically confirmed cases and 572 controls admitted to hospital in Italy and Switzerland	Self-rated assessment of total physical activity; 1 (highest), 2, 3, 4 (lowest)	Self-reported height and weight to calculate BMI <25.0, ≥25.0	Endometrial cancer	Study center, age	Adjusted RR (95% CI) BMI <25.0 1 = 1.0 2 = 0.7 (0.4–1.2) 3 = 0.6 (0.3–1.1) 4 = 1.8 (0.6–5.2) P = 0.08 BMI ≥25.0 1 = 1.0 2 = 1.0 (0.6–1.8) 3 = 1.8 (0.9–3.4) 4 = 4.7 (1.9–11.3) P = 0.01

LTPA, leisure time physical activity; BMI, body mass index (kg·m^{-2}); py, person-years; RR, relative risks; CI, confidence intervals.

had lower death rates than sedentary men in the low BMI group. A limitation of these reports is that the upper BMI categories included individuals with only mild to moderate overweight.

Cardiovascular Disease Mortality

We only found three reports on cardiovascular disease mortality (defined as ICD-9 codes 390 to 449.9) that met our review criteria (see Table 2). These include two studies from extended follow-up in the ACLS cohort discussed in the section on all-cause mortality (18,19) and one additional report from the same study (10). Results of these analyses were similar to that for all-cause mortality. Unfit men had substantially higher cardiovascular disease death rates than fit men in the same body habitus group (BMI, percent body fat, fat mass, and fat-free mass). Although there was an inverse trend for cardiovascular disease deaths across fitness categories in individuals with a BMI $\geq$ 27.0 (rates 17.9, 10.6, and 9.5/10,000 man-years), the trend was not significant ($P = 0.24$) (10). Fit men in the highest body habitus groups had lower death rates than unfit men in the lowest body habitus groups. However, there was a tendency, even in the fit men, for cardiovascular disease death rates to be higher in higher body habitus groups than in the lower body habitus groups, which suggests that higher fitness does not eliminate excess cardiovascular disease mortality in individuals in the higher body habitus groups. This is in contrast to results from the studies on all-cause mortality where no excess risk was seen in the fit men who were overweight and/or obese (Table 1).

CHD

There were seven studies that included CHD as an outcome (Table 3), sometimes combined as fatal and nonfatal events and sometimes as fatal events, and BMI and physical activity as exposure variables. BMI was determined by self-report in five of the studies and measured in the other two studies. Physical activity was estimated from self-reports of leisure-time physical activity in all studies, and one study also included data on self-reported occupational activity (33). All reports included data for men, but only one study included data for women (33). The report on women includes only 12 events, and the results are inconsistent, both within exposure groups for the women and in relation to the results for men in this and other papers.

Results of the cross-tabulation of body habitus and physical activity in relation to CHD were similar to those described earlier for all-cause and cardiovascular disease mortality. Physically active men had lower rates of CHD than inactive men in all studies, and in all BMI strata. In general, active men in the high BMI group had lower CHD rates than inactive men in the low BMI group. In some studies the difference between these divergent groups was striking. For example, Morris et al. (23) reported that men with a BMI $\geq$ 27.0 who regularly participated in vigorous exercise had a heart attack rate of 1.3/1000 man-years, and men with a BMI $<$ 24.0 who were inactive had a rate of 5.5/1000 man-years. Salonen et al. (33) reported that men with a BMI $\geq$ 27.0 in an active occupation and with high leisure time physical activity had a CHD mortality rate of 7.4/1000. In contrast, men with a BMI $<$ 27.0 who were sedentary at work and in leisure time had a mortality rate of 25.9/1000. Regular physical activity appeared to provide substantial protection against CHD, especially in overweight men.

Hypertension

We found only two papers with hypertension as the outcome that met our inclusion criteria (Table 4). Paffenbarger et al. (26,30) followed two cohorts of college alumni for up to 15 yr for the development of incident physician-diagnosed hypertension. Validity of self-report of both exposure and outcome assessments was high in these well-educated men. In general, active men in both studies had a lower risk of developing hypertension than sedentary men in different BMI categories, although this difference was significant only in the two highest BMI categories in the Harvard men. Among the inactive University of Pennsylvania alumni, the incidence of hypertension was greater than among active men in all BMI categories. None of these differences were statistically significant.

In summary, regular physical activity appears to reduce the risk of developing hypertension in men with elevated BMI, but this association is less marked than was observed for the mortality outcomes reviewed earlier. Reduction in risk for active men was greatest in men in the high BMI categories.

Type 2 Diabetes Mellitus

Five studies with type 2 diabetes mellitus as the outcome variable are summarized in Table 5. One study was in women and four studies included only men. Four of the studies relied on self-reported physical activity, height and weight, and presence of type 2 diabetes mellitus from mail-back questionnaires. Active individuals in these studies had a lower incidence of type 2 diabetes than sedentary participants, and this association tended to be stronger among those in the highest BMI categories. For example, there was a 39% lower risk ($P = 0.005$) in the active men with a BMI $>$ 26.4 when compared with sedentary men in the Physicians' Health Study, and this association was absent or much weaker in the lighter men (21). Data from the Honolulu Heart study were similar to those reported in the Physicians' Health Study, with the additional observation that the trend across activity categories in men with a BMI $\geq$ 28.2 was not significant for all men combined or men with a glucose $<$ 225 mg·dL^{-1} (6). Results for men and women were similar in these studies.

Studies relating physical activity and type 2 diabetes mellitus that rely on self-reported physical activity and presence of diagnosed diabetes may underestimate the true risk of sedentary habits in relation to this disease. This is because of the limitations of self-reported exercise habits and also because approximately 50% of the prevalent cases of type 2 diabetes are undiagnosed, which leads to substan-

tial misclassification on both the exposure and outcome variables. Wei et al. (39) recently reported a greater than 3-fold increased risk for developing type 2 diabetes in men with low cardiorespiratory fitness when compared with high fit men. The odds ratio was 3.7 when adjusted for age, history of parental diabetes, and length of follow-up, and this was only reduced to 3.2 after additional adjustment for BMI, high blood pressure, high density lipoprotein (HDL)-cholesterol, total cholesterol, triglycerides, cigarette smoking status, alcohol intake, and change in fitness. In addition to an objective measurement of the activity exposure, diabetes in this study also was determined objectively by measuring fasting plasma glucose concentrations at baseline and at follow-up. These authors reported a steep inverse gradient of risk across fitness categories among individuals with BMI of < 27.0 ($P < 0.001$) and ≥ 27.0 ($P < 0.006$) (see Fig. 1). Fit men with a BMI of ≥ 27.0 had a slightly lower incidence of type 2 diabetes than unfit men in the BMI $<$ 27.0 group. Wei et al. (39) also reported a substantially increased risk of developing impaired fasting glucose (fasting plasma glucose ≥ 110 to < 126 mg·dL^{-1}). The odds ratio for impaired fasting glucose in men with low fitness when compared with men with high fitness was 2.1 in the fully adjusted model. The results were similar among men in high and low strata of BMI.

Being active and fit appears to reduce the risk of type 2 diabetes, at least in heavy individuals. This association was strongest in the study with objective measures of both exposures and outcome.

Cancer

We found four studies in which cancer was the outcome (Table 6). Physical activity was assessed in all studies by self-report. Self-reported height and weight were used to calculate BMI in one study. Two studies were in women only, and the other two studies included both women and men. In general, we consider the results for cancer inconclusive. In several of the analyses cancer risk was higher in sedentary individuals, but the results in the highest BMI groups were not consistent. For example, sedentary women with a BMI ≥ 25.0 had nearly five times higher endometrial cancer risk than highly active women in the same BMI category (20). However, there was a nonsignificant trend of higher risk of breast cancer across activity categories in women with a BMI > 25.7 (37). In the same study, a steep inverse gradient of breast cancer risk across activity groups was found in women with a BMI < 22.8. It is difficult to generalize the results presented in Table 6 because of the inconsistent results associated with different cancer outcomes. In some studies the outcome was a single type of cancer, and in one study cancer at all sites was the outcome of interest.

CONCLUSIONS

The review performed for this report addressed three questions:

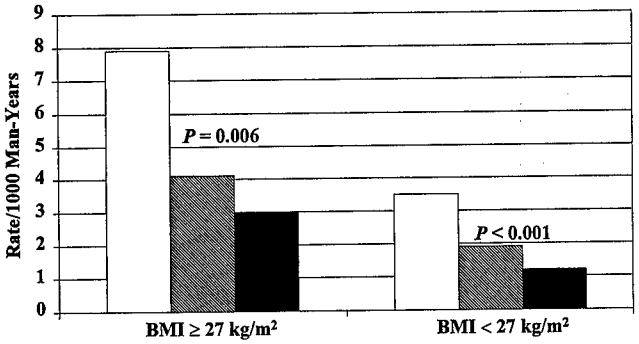

Figure 1—Incidence of type 2 diabetes (per 1000 man-years) among cardiorespiratory fitness levels according to BMI (kg·m^{-2}). White bars indicate the low fitness group, diagonal lines the moderate fitness group, and black bars the high fitness group. Adapted by permission from 39. Wei, M., L. W. Gibbons, T. L. Mitchell, J. B. Kampert, C. D. Lee, and S. N. Blair. The association between cardiorespiratory fitness and impaired fasting glucose and type 2 diabetes mellitus in men. *Ann. Intern. Med.* 130:89–96, 1999.

1. Do higher levels of physical activity or cardiorespiratory fitness attenuate the increased risk of morbidity and mortality in overweight or obese persons?

Evidence statement. Overweight and obese individuals who are active and fit have lower rates of disease and death than overweight and obese individuals who are inactive and unfit. This inverse gradient of risk across activity or fitness categories is present in various strata of body habitus and frequently is steeper in the higher categories of body habitus variables.

Rationale. Active and fit individuals in nearly all the studies summarized in Tables 1–6 had lower rates of morbidity and mortality than sedentary and unfit individuals in all strata of body habitus. These findings remained after adjustment for age and other potential confounding variables and were seen for the various specific outcomes included here. An active and fit way of life apparently provides protection against several chronic diseases, and this association typically is present in all strata of body habitus. The inverse gradient of risk across levels of activity or fitness often was steeper in the higher strata of body habitus than among leaner individuals. For example, Morris et al. (23) report heart attack rates in men with a BMI ≥ 27.0 of 7.3/1000 man-years in sedentary men and only 1.3/1000 man-years in regular vigorous exercisers. Corresponding rates in men with a BMI < 24.0 were 5.5/1000 man-years in sedentary men and 1.9/1000 man-years in regular vigorous exercisers.

2. Do overweight or obese individuals who are physically active or fit actually have a lower risk of morbidity and mortality than normal weight individuals who are sedentary (i.e., what is magnitude of the protective effect of activity or fitness in overweight and obese individuals)?

Evidence Statement: Overweight or obese individuals who are active and fit are less likely to develop obesity-related chronic diseases and have early death than normal weight persons who lead sedentary lives.

Rationale. Overweight or obese individuals who were active and fit had morbidity and mortality rates that were at least as low, and in many instances much lower, than normal weight individuals who were sedentary. Perhaps the strongest evidence supporting this conclusion comes from the reports by Lee et al. (18,19). These investigators followed a large cohort of men for an average of 8.5 yr. The strength of this study is that objective measurements were available for cardiorespiratory fitness and several measures of body habitus, including BMI, percent body fat, fat mass, fat-free mass, and waist circumference. Furthermore, the study was large enough to allow for analyses in smokers and nonsmokers, with exclusion for early mortality and with adjustment for several important potential confounders. In this report unfit men in the normal range of body habitus (BMI 19–25, percent body fat $< 16.7\%$, or waist circumference < 87 cm) had more than a two-fold higher mortality risk than fit men in the highest category of body habitus (BMI ≥ 27.8, percent body fat $\geq 25\%$, or waist circumference ≥ 99 cm). This apparent protection of moderate to high cardiorespiratory fitness extends to men who clearly are obese (BMI $>$ 30) (2).

3. Which is a more important predictor of mortality in individuals, overweight or obesity or inactivity and low fitness?

Evidence statement. Inactivity and low cardiorespiratory fitness are as important as overweight or obesity as predictors of mortality, at least in men.

Rationale. The question considered here relates to risk for individuals and not to the importance of either of these exposures as public health problems. It is clear that overweight and obesity are associated with increased risk of morbidity and mortality (25) and that an active way of life protects against morbidity and mortality (38). Because many studies on obesity and health have not adequately accounted for the possible confounding effects of physical activity, the independent effects of these two exposures are unclear. It is tempting, based on the reports reviewed here from the ACLS (2,18,19), to conclude that low fitness is a more important predictor of mortality in individuals than overweight or obesity, because excess mortality risk is largely eliminated in overweight or obese men who are also fit. In our judgment, such a conclusion would be premature. Other reports reviewed here did not have the specific purpose of addressing this question, and there are limitations on some of the analyses that did not focus specifically on this issue. In addition, data from the ACLS are available only for men, and this cohort is from middle to upper socioeconomic strata, with few members of minority groups. This homogeneity is an advantage in relation to internal validity of the study but presents limitations for generalization. Replication of the studies reported from the ACLS is needed.

Issues and limitations. All studies reviewed here are from Evidence Category C. It is unlikely that there will ever be randomized clinical trials to address specifically the questions posed in this report because of the required size, complexity, and cost. Thus, the best evidence we are ever likely to have on this issue will come from prospective observational studies.

Although the results reviewed here are remarkably consistent for various endpoints, because different measures of habitual physical activity or fitness, different strata of body habitus, and different populations were used in the various studies, conclusions must remain tentative. Few studies included women, and for some that did, the number of events was small. There are virtually no data available from minority groups. Health status or change in health status often was not considered or was inadequately addressed, although results are similar for studies that excluded unhealthy individuals at baseline and those that did not. Only reports from the ACLS had the specific purpose of addressing the questions considered in this review. While other reports had some data related to the issue, the lack of focus on the relevant question often led to incomplete analyses insofar as the present review is concerned.

Crude measurements of self-reported physical activity are an important limitation of many of the studies. In some studies, physical activity appears not to have been a very important variable under consideration during the planning phase of the study. When only one or two very simple questions on habitual physical activity are included as part of the study measurements, it seems reasonable to conclude that this was not a high priority item for the investigators. Under these conditions it is not realistic to expect that adequate and appropriate analyses can be performed with physical activity as an exposure. As an extreme example, suppose that an investigator wanted to address question 3 that is posed in this report. Further suppose that in that study the investigator measured physical activity by the question "Are you physically active or not?" and assessed body habitus by computed tomography scanning. One could hardly expect to find that inactivity is as important as body habitus as a predictor of mortality in such a study, even if inactivity actually were more important. Therefore, one of the limitations to keep in mind when evaluating results from the studies reviewed here is the relative validity of the exposure measures used.

Another of the major limitations of several of the current studies is that the upper body habitus stratum often began in the mild overweight range and may not have included many truly obese individuals. The conclusions presented here should not be extended to individuals with Class II or Class III obesity (BMI ≥ 35.0).

Research recommendations. Additional research is needed to address the questions posed for this review. We recommend that the following types of studies be conducted:

1. Replicate prospective observational studies of the predictive validity of physical activity or cardiorespiratory fitness and body habitus for morbidity and mortality in diverse population groups. It is especially important that studies be conducted in women, older persons, and minority groups.
2. Prospective observational studies with other outcome measures need to be conducted. Overweight and obe-

sity increase risk for functional limitations associated with aging and for knee osteoarthritis. It is not clear that regular physical activity might reduce these risks in overweight or obese persons.

3. Improve the assessment of physical activity in prospective observational studies of populations so that less misclassification occurs for this exposure. Ideally, objective measurements of activity by electronic monitoring should be considered.
4. Develop creative designs for randomized clinical trials to determine the independent and interactive effects of physical activity and body habitus on subclinical markers of chronic disease. Random assignment to activity interventions with blocking on strata of body habitus is one possible approach. Because controlled trials with morbidity and mortality as outcome measures are unlikely to be carried out in relation to the issues addressed in this review, investigators should incorporate more direct measures of disease processes in smaller, logistically-feasible clinical trials. Examples of such measures are ultrasound measures of carotid wall thickness, coronary artery calcification by electron beam computed tomography, near-infrared spectroscopy assessment of lipid content of atherosclerotic plaque, endothelial function of the coronary circulation by phase contrast, magnetic resonance imaging (MRI) with infusion of adenosine, endothelial function of the peripheral circulation by reactive hyperemia in response to ischemic exercise, left ventricular function by MRI, and mechanical function of vasculature by pulse wave velocity measurements.

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Economic costs of obesity and inactivity

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ABSTRACT

COLDITZ, G. A. Economic costs of obesity and inactivity. *Med. Sci. Sports Exerc.,* Vol. 31, No. 11, Suppl., pp. S663–S667, 1999. **Purpose:** The purpose of this paper is to assess the economic costs of inactivity (including those attributable to obesity). These costs represent one summary of the public health impact of increasingly sedentary populations in countries with established market economies. Components of the costs of illness include direct costs resulting from treatment of morbidity and indirect costs caused by lost productivity (work days lost) and forgone earnings caused by premature mortality. **Methods:** We searched the *Medline* database for studies reporting the economic costs of obesity or inactivity, or cost of illness. From the identified references those relating to obesity or conditions attributable to obesity were reviewed. Chronic conditions related to inactivity include coronary heart disease (CHD), hypertension, Type II diabetes, colon cancer, depression and anxiety, osteoporotic hip fractures, and also obesity. Increasing adiposity, or obesity, is itself a direct cause of Type II diabetes, hypertension, CHD, gallbladder disease, osteoarthritis and cancer of the breast, colon, and endometrium. The most up-to-date estimates were extracted. To estimate the proportion of disease that could be prevented by eliminating inactivity or obesity we calculated the population-attributable risk percent. Prevalence based cost of illness for the U.S. is in 1995 dollars. **Results:** The direct costs of lack of physical activity, defined conservatively as absence of leisure-time physical activity, are approximately 24 billion dollars or 2.4% of the U.S. health care expenditures. Direct costs for obesity defined as body mass index greater than 30, in 1995 dollars, total 70 billion dollars. These costs are independent of those resulting from lack of activity. **Conclusion:** Overall, the direct costs of inactivity and obesity account for some 9.4% of the national health care expenditures in the United States. Inactivity, with its wide range of health consequences, represents a major avoidable contribution to the costs of illness in the United States and other countries with modern lifestyles that have replaced physical labor with sedentary occupations and motorized transportation. **Key Words:** INACTIVITY, ECONOMIC COSTS, MORBIDITY, OBESITY, PREVALENCE.

Inactivity is a major cause of impaired glucose tolerance and contributes to poor energy balance (obesity guidelines). In cultures with abundant energy intake, lack of physical activity contributes to the development of obesity—or weight gain—as a consequence of excess energy intake beyond energy expenditure. Inactivity is related to increased risk of cardiovascular disease (CVD) (1). Cardiovascular disease is the leading cause of death in the United States. Approximately 58 million persons in the United States (20% of the total population) have one or more types of CVD (29). These types of CVD include: high blood pressure, CHD, stroke, rheumatic heart disease, and other forms of heart disease. Behavioral risk factors for CVD include physical inactivity and being overweight. In 1994 CVD accounted for 45.2% of all deaths in the United States.

Other major morbidity caused by inactivity includes colon cancer (7), osteoporosis and particularly hip fracture (18), and Type II diabetes (47). Higher levels of activity may reduce risk of breast cancer, although the magnitude of this relation remains uncertain (12). In each of these major medical conditions the adverse effect of inactivity is independent of body weight or adiposity. As a consequence of this independence, the burden of inactivity can be added to that attributable to obesity.

Overweight and obesity are major risk factors for many chronic diseases and also exacerbate many chronic conditions, including hypertension, high cholesterol, and osteoarthritis (34,46). Men and women at even modest levels of adiposity are at increased risk of morbidity. Further, adult weight gain is related to increased risk of coronary heart disease (CHD) (40,48), non-insulin-dependent or Type II diabetes mellitus (4,8), postmenopausal breast cancer (20), and mortality (26).

To address the full impact of behaviors such as inactivity and excess weight on health and their consequences, one can summarize the burden of premature mortality. McGinnis and Foege (28) estimate that lack of activity and diet contribute to 14% of mortality. Hahn et al. (17) estimate that sedentary lifestyle contributes to 23% of deaths from the leading chronic diseases. Data from the follow-up of the Iowa Women's Health Study (23) indicate a strong inverse relation between physical activity and total mortality among women, and the Harvard Alumni study shows a similar relationship among men (36).

Although mortality is commonly used as a measure of disease burden, it does not account for the morbidity associated with chronic conditions. Further, it omits any accounting of the impact of lifestyle on health-related quality

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of life. One can use an economic measure to summarize this broad range of health effects. Such a measure can account for both nonfatal and fatal conditions.

The direct costs of illness include the costs of diagnosis and treatment related to any disease (hospital stay, nursing home, medications, physician visits). The value of lost productivity is an indirect cost of illness. This includes wages lost by people unable to work because of disease and also the forgone wages resulting from premature mortality. These future earnings after death are translated into current monetary value using an inflation or discount factor, usually 3%. We do not quantify these costs in the current analysis.

Approximately 60 million U.S. adults are overweight, one-third of the population (10,32). Further, the prevalence of overweight and obesity has been increasing (10). The age-adjusted prevalence of obesity increased by 30% from 1980 to 1994 (22). The proportion of the U.S. adult population aged > 20-yr old exceeding the healthy weight ranges (that is the prevalence of BMI> 25.0) is high; 59.4% of men, 50.7% of women, or 54.9% of the total U.S. population. Overall 22.8% of the adult population, 24.8% of women and 20% of men, are obese (BMI >30).

Despite its benefits, many people do not engage in regular physical activity. The Behavioral Risk Factor Surveillance System (BRFSS) reported that in 1995 approximately 28.8% of the U.S. adult population reported no leisure-time physical activity (30). In some states up to 48% reported no leisure-time physical activity.

METHODS

We searched the MEDLINE database for studies reporting the economic costs of obesity or inactivity, or cost of illness. From the identified references, those relating to obesity or conditions attributable to obesity were reviewed. As estimates of the cost of obesity have been reported more than once for several countries, the most up-to-date estimates were extracted.

To estimate the proportion of disease that could be prevented by eliminating inactivity or obesity we calculate the population-attributable risk percent. This is the maximum proportion of disease attributable to the specific exposure (obesity or lack of physical activity). PAR% is based on the incidence of disease in the exposed (i.e., inactive group) as compared with the nonexposed, taking relative risks from analyses that control for confounders (e.g., age, smoking, dietary intake, etc.). PAR% is calculated using P(RR-1)/1 +P (rr-1), where P is the prevalence of exposure in the population and RR the relative risk for disease.

The procedures to estimate the costs of diabetes and gallstones are presented elsewhere (6,21). Annual direct costs of hypertension and CHD were extrapolated from Hodgson and Kopstein (19). All costs are inflated to 1995 dollars using the medical component of the consumer price index.

TABLE 1. Costs of inactivity (billion $), in the United States, 1995.

Condition	Relative Risk	PAR%	Direct Costs
Type 2 diabetes	1.5	12%	6.4
CHD	2	22%	8.9
Hypertension	1.5	12%	2.3
Gall bladder disease	2	22%	1.9
Cancer			
Breast	1.2	5%	0.38
Colon	2	22%	2.0
Osteoporotic fractures	2	18%	2.4
Total			24.3 billion

RESULTS

Cost attributable to inactivity. Using the prevalence of inactivity as 28.8% (the median prevalence for U.S. adults reporting no leisure-time physical activity in 1995), we conservatively estimate the PAR% for inactivity and multiply this by the annual costs of illness caused by lack of physical activity. We estimate that 22% of CHD, 22% of colon cancer, 22% of osteoporotic fractures, 12% of diabetes and hypertension, and perhaps 5% of breast cancer are attributable to lack of physical activity (see Table 1).

Overall, sedentary behavior and lack of physical activity costs the U.S. a conservative total of 24.3 billion dollars per year for direct health care delivery costs. That is, approximately 2.4% of all health care costs in 1995 are due to or are the result of lack of physical activity.

In a sensitivity analysis we estimate the costs resulting from inactivity using the upper bound of state level reports of no physical activity (48%). This may more closely approximate the proportion of the population with insufficient activity to avoid the health consequences of inactivity. Using this prevalence and retaining the relative risk for the association between activity and the major health outcomes we estimate the costs of inactivity as 37.2 billion dollars (3.7% of direct health care costs).

Costs of obesity. The direct costs of obesity have been estimated for several countries. Because the majority of identified studies follow the methods reported by Colditz in 1992, this approach is described here (6). The prevalence-based estimate of the burden of obesity is based on the disease prevalence and the estimated proportion of disease attributable to obesity. Diseases considered included non-insulin-dependent diabetes mellitus (NIDDM), gall bladder disease, cardiovascular disease, cancer (colon and prostate in men; breast, endometrium, cervix, and ovarian in women). This analysis used a definition of obesity as BMI greater than 27.8 for men and 27.3 for women. Subsequent analyses have approximated the current recommended definition of obesity at BMI of 30.

Using a prevalence-based approach to estimate the costs of obesity in the United States, Wolf and Colditz (49) estimate the proportion of disease attributable to obesity and the associated costs. Conditions included in their analysis were Type II diabetes, CHD, hypertension, gallbladder disease, postmenopausal breast cancer, endometrial cancer, colon cancer, and osteoarthritis. Using prevalences from the National Health and Nutrition Examination Survey III (NHANES III) (22.4% overall and 24.9% for breast and

TABLE 2. Costs ($ billions) of obesity (BMI > 30) in the United States, 1995.

Condition	Relative Risk	PAR%	Direct Costs
Type 2 diabetes	11	69%	36.6
CHD	4	40%	16.2
Hypertension	4	40%	7.6
Gall bladder disease	5.5	50%	4.3
Cancer			
Breast	1.3	7%	.53
Endometrium	2.5	27%	.23
Colon	1.5	10%	0.89
Osteoarthritis	2.1	20%	3.6
Total			70 billion

Uses prevalence of obesity = 22.5% as reported in NHANES III and for breast and endometrial cancer uses prevalence of 24.9% as reported by Flegal et al. 1998 (10).

endometrial cancers) we update the costs attributable to obesity. Overall the direct health care costs of obesity were approximately 70 billion dollars or 7% of total health care cost (see Table 2).

Costs of obesity in other countries. Using this same approach as Colditz, Levy (24) estimated that the costs of obesity in France was approximately 2% in 1992 (Table 3). In The Netherlands, Seidell estimates that the cost is 4% of the national health care costs (44), and Segal estimates that for Australia obesity is responsible for 2% of the costs of health care (43). The definition of obesity, the conditions included in cost estimates, and the findings from these studies are summarized in Table 3.

We have previously reported the economic costs of obesity for the United States using overweight defined as body mass index greater than 27 $kg \cdot m^{-2}$. Now, with the World Health Organization definition of obesity as moderate obesity for BMI 25–29.9, severe overweight as BMI 30–39.9, and massive obesity as BMI 40 or more, we conservatively estimate the economic impact of obesity for BMI greater than 29 (49). It is clear that this underestimates the total costs of obesity, in part because we omit the costs for illness among those who are overweight (BMI 25–29.9). In addition, we omit several conditions related to obesity. These include benign prostatic hypertrophy which is related to increased abdominal adiposity (14) and lack of activity (37) and infertility (J. W. Richards et al., personal communication). Asthma may also be increased by obesity.

Indirect costs. Early retirement and increased risk of disability pensions together add indirect costs that are not included in the estimates for individual countries summarized above. Narbro et al. (31) estimate that for Sweden, obese subjects are 1.5–1.9 times more likely to take sick leave and that 12% of obese women had disability pensions attributable to obesity, costing some 300 million U.S. dollars for 1 million in the female adult population. Overall approximately 10% of sick leave and disability pensions in women may be related to obesity and obesity related conditions (31). Discrimination against those who are obese may lead to poor quality of life and downward social mobility (42).

The indirect costs attributable to obesity amount to at least 48 billion dollars. The major contributor to these costs is CHD (48%), which accounts for the large portion of premature mortality. Other indirect costs were NIDDM (17.5%), and osteoarthritis (17.1%); the latter largely resulted from excess bed days, work days lost, and restricted activity days.

Total direct costs of inactivity and obesity. The sum of obesity (7% of health care costs) and of inactivity (2.4%) is here used to estimate the total direct costs of inactivity. Overall a minimum 9.4% of all direct costs incurred in delivering health care in the U.S. are attributable to insufficient energy expenditure which directly leads to medical conditions or alternatively the accumulation of adiposity which then contributes to excess morbidity and mortality. As there are clear health benefits with increasing levels of physical activity, the definition of inactivity is somewhat arbitrary and could be raised so that more of the population is considered inactive. Under a broader definition of inactivity, more of the chronic conditions considered here would be attributable to lack of activity. Such changes would substantially increase the economic burden of lack of activity and of obesity or excess adiposity. As noted for obesity in The Netherlands, the majority of health care costs arise from the overweight rather than the obese population. This reflects the distribution of adiposity in the population, and the principle set forth by Rose, that disease arises not from the tails of the distribution but from those at average risk (41).

Note that these are conservative estimates of the current U.S. costs. The costs of obesity are estimated for those with BMI of 30 or more kilograms per meter squared. Adverse health affects are present at levels of obesity below a BMI of 30 (34,46) and higher levels of activity are associated with even lower risk of many chronic conditions (47). There are substantial additional costs incurred among those who are overweight (BMI 25 to 29.9; 32% of the U.S. population). Likewise for estimates of inactivity, we use the prevalence of approximately 28% of the U.S. population being inactive.

DISCUSSION

The public health burden of inactivity and obesity is substantial. Estimates of the proportion of deaths caused by these factors range from 14 to 23% of total mortality in the United States. The public health burden of inactivity is thus substantial for mortality but also for chronic conditions that negatively impact quality of life as well as life expectancy. Health consequences of sedentary lifestyle are independent of those associated with obesity as indicated in several major studies which have simultaneously evaluated physical activity and obesity in relation to colon cancer, diabetes, and total mortality. For example, evaluating relationships between physical activity and colon cancer, Giovannucci (13) and Martinez (27) found that the inverse associations with level of activity were independent of BMI. Likewise Blair has shown independent effects of fitness and activity on mortality (2,3). These and other data support the assumption of independence and hence allow us to add the economic

TABLE 3. Cost of obesity from different countries.

Author	Country, Year ($)	Definition of Obesity (BMI)	Conditions Included	% Total	Comment
Wolf and Colditz 1998 (49)	United States, 1995	29	Type 2 DB, CHD, Hypertension; gallbladder; breast, endometrial, and colon cancer; osteoarthritis	5%	
Levy 1995 (24)	France 1992	27	Hypertension, MI, angina, stroke, venous thrombosis, NIDDM, hyperlipidemia, gout, osteoarthritis, gallbladder disease, colorectal cancer, breast cancer, genitourinary cancer, hip fracture	2%	
Seidell 1995 (44)	Netherlands	25		4%	Costs of overweight greater than that of obesity
Segal 1994 (43)	Australia 1989	30	NIDDM, gallstones, hypertension, CHD, breast cancer, colon cancer	2%	Comparable to cost of alcohol-related conditions in Australia

costs of obesity to the costs of sedentary lifestyle. We therefore here added the direct costs of obesity to those for sedentary lifestyle.

The costs of inactivity and obesity are similar to the total estimated impact of cigarette smoking in the United States ($47 billion) (25). These costs of inactivity and obesity reflect, in large part, the impact of weight gain in adult life. These costs could be avoided if individuals maintained a healthy weight throughout adulthood. This is now a priority recommendation for the U.S. Department of Health and Human Service dietary and weight guidelines (46).

Gorsky et al. (15) simulated three hypothetical cohorts to estimate the costs of health care according to level of obesity over a 25-yr period, discounting future costs at 3% per year. They estimate that 16 billion additional dollars will be spent over the next 25 years treating health outcomes associated with obesity among middle-aged women. Using an incidence-based approach to cost of illness, Oster et al. (35) estimated the excess costs of health services according to level of obesity. Using a conservative approach that does not include any future weight gain and starting with NHANES3 population estimates for BMI, cholesterol, hypertension, and diabetes, they estimate the lifetime future costs per person as comparable with those of smoking.

A social cost that is not considered in any of the economic summaries is that of reduced physical functioning that is associated with higher levels of obesity (5,11). Inactivity or low levels of physical fitness are important determinants of loss of function, and weight training studies show improvements in function in frail elderly (9). Research is needed to quantify these relations and incorporate the deterioration in quality of life into summaries of the burden of inactivity.

While benign prostatic hyperplasia has not been considered here, it is noteworthy that this common condition may be but one of many complications of obesity and inactivity that has been overlooked. Likewise there is a direct relation between increasing adiposity and the diagnosis of infertility among young women (39). Recent evidence also suggests that obesity is related to asthma. This common condition which is rising in prevalence around the world is related to adiposity among children and also adults.

The recent interest in and documentation of relations between weight-bearing exercise and decreased risk of osteoporotic fractures of the hip points to important work remaining to be incorporated into economic costs of illness studies. Growing evidence that activity is associated with higher bone density and also with better muscle tone, together contributing to lower risk of falls and fractures (16,33,38,45), now points to the need for better estimates of the social and economic burden of hip fracture.

In summary, growing levels of both inactivity and obesity pose major health problems in Western society. These preventable sources of morbidity and mortality require focused strategies to increase the level of energy expenditure. Substantial benefits will likely accrue through reduced health care costs but also through reduction in the indirect costs as well as gains in quality of life.

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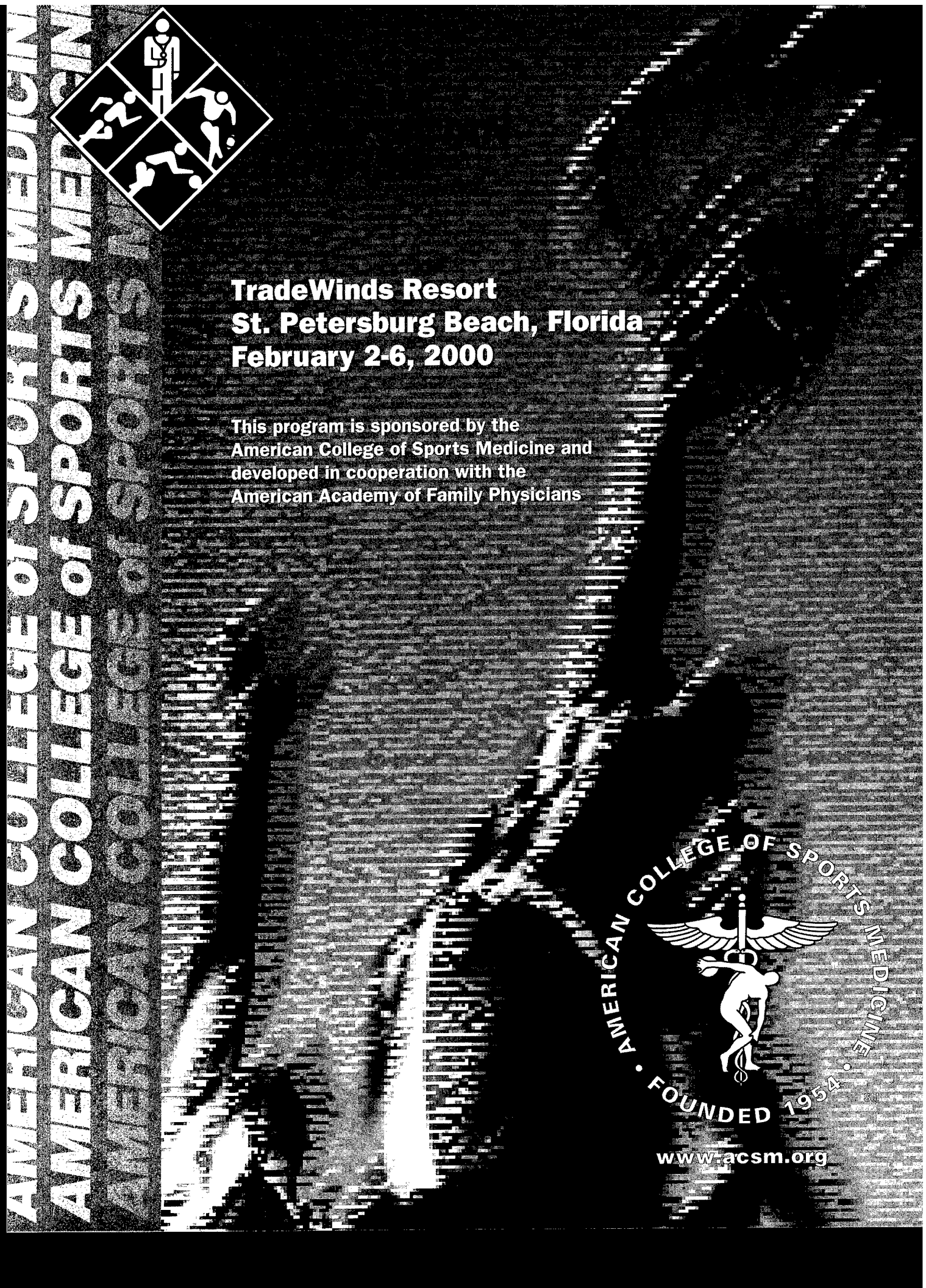
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