

Physical Activity in the Prevention and Management of Coronary Heart Disease

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HIGHLIGHT

“Along with stopping smoking, maintaining a physically active lifestyle is one of the least expensive and most productive health behaviors available to the public.”

INTRODUCTION

The concept that a sedentary lifestyle leads to an increase in the clinical manifestations of coronary heart disease (CHD), especially myocardial infarction and sudden death, has become generally accepted by the public and many health professionals. Most often, the idea has been expressed that regular exercise, in conjunction with other risk-reducing behaviors, will help protect against an initial cardiac episode (primary prevention); will aid in the recovery of patients following myocardial infarction, coronary artery bypass surgery, or coronary angioplasty (cardiac rehabilitation); and will reduce the risk of recurrent cardiac events (secondary prevention).

Evidence relating level of habitual exercise to risk of CHD has been derived from a variety of sources including animal studies, clinical impressions, observational surveys of the general population or special groups, and experimental studies in which the exercise of subjects assigned to “treatment” was increased in relation to sedentary control subjects. No one of these studies provides irrefutable evidence of a causal relationship between exercise status and CHD pathology, even though many sources of information do generally support such a contention. This situation is not unique to our understanding of the preventive role of exercise as it relates to CHD since a similar situation exists for all other “lifestyle” risk factors.

The presentation of information here is designed to provide the scientific basis for making decisions regarding the potential value of exercise in the primary and secondary prevention of CHD. Data on the relationship of exercise to CHD are reviewed, the possible biologic mechanisms by which beneficial effects may occur are summarized, the risks of developing cardiac complications during exercise are briefly discussed, and physical activity guidelines for promoting cardiovascular health are provided.

PHYSICAL ACTIVITY AND THE PRIMARY PREVENTION OF CORONARY HEART DISEASE

During the past half century more than 50 studies have been published reporting on the association between habitual level of physical activity and the prevalence or incidence of initial clinical manifestations of CHD, especially myocardial infarction and sudden cardiac death (Berlin & Colditz, 1990; Powell et al., 1987). These studies have included the determination of on-the-job or leisure-time activity in free-living populations of many men and relatively few women with activity classifications based on job category, self-report questionnaires, or interviewer determinations. Manifestations of CHD were established by examination of death certificates, hospital or physician records, questionnaires completed by the subjects or physicians, and medical evaluations conducted by the investigators. Reported activity levels range from daily caloric expenditures exceeding 6,000 kilocalories (kcal) per day in Finnish lumberjacks at one extreme to very sedentary civil servant managers and postal clerks at the other. Studies have been conducted in major industrial environments as well as rural and primate living areas.

As a result of the diverse protocols used in the various studies, including sample-selection procedures, physical activity classification methods, clinical event determination criteria, and statistical treatment of the data, it is not possible to collate the results into a single summary statement or interpretation. However, certain findings, although not universally obtained, occur sufficiently frequently to warrant the formulation of preliminary conclusions to use as a basis for program recommendations and planning future research.

More Active Persons Appear to Be at Lower Risk

The general impression obtained as the result of a comprehensive review of the scientific reports containing data on the primary preventive effect of physical activity is that more active people develop less CHD than their inactive counterparts, and when they do develop CHD, it occurs at a later age and tends to be less severe (Berlin & Colditz, 1990; Powell et al., 1987). The results of the numerous reports are quite variable, with some studies demonstrating a highly significant beneficial effect of exercise (Lakka et al., 1994; Morris et al., 1980; Paffenbarger, Wing, & Hyde, 1978; Shapiro et al., 1969; Shaper & Wannamethee, 1991), others showing a favorable but nonsignificant trend in favor of the more active (Costas et al., 1978; Salonen, Puska, & Tuomilehto, 1982), and a few early studies showing no difference in CHD rates (Chapman & Massey, 1964; Paul, 1969). Of major importance is the consistent finding that being physically active *does not increase* an individual's overall risk of CHD.

No specific study characteristics can be identified that explain the differences in results among the various studies, but in some cases the physical activity measure is not very accurate or reliable and the activity gradient among the population is quite small (Shapiro et al., 1969). Also, with populations in whom CHD mortality is exceptionally high and in whom major risk factors such as hypercholesterolemia, hypertension, and cigarette smoking are prevalent, even very high levels of physical activity do not appear to exert a major protective effect. Finnish lumberjacks are an example of very physically active individuals in whom CHD risk remains high (Karvonen et al., 1961). It appears that in observational studies that are well designed, the inverse association between activity and CHD mortality is stronger than reported in scientifically less rigorous studies (Powell et al., 1987).

Moderate Amounts of Exercise May Be Protective

A striking feature of many studies demonstrating a reduced CHD risk for more active individuals is that the greatest difference in risk is achieved between those people who do almost nothing and those who perform a moderate amount of exercise on a regular basis. Much smaller differentials in risk are observed when moderately active individuals are compared with the most active persons (Leon et al., 1987; Lakka et al., 1994; Paffenbarger et al., 1993).

The amount of activity, in both intensity and duration, that is associated with a decrease in CHD clinical manifestations varies substantially among the different reports. Several studies have observed significant differences in CHD indicators with quite small differences in habitual activity level at a relatively low intensity (Leon et al., 1987; Kahn, 1963; Shapiro et al., 1969), whereas other authors interpret their data to indicate that a “threshold” of higher intensity or amount of activity is needed in order to obtain a benefit (Cassel et al., 1971; Morris et al., 1980; Paffenbarger, Wing, & Hyde, 1978). The types of activity performed by the more active groups include brisk walking on level or hilly ground, climbing stairs, lifting and carrying light objects, lifting heavy objects, operating machinery or appliances, light and heavy gardening, performing home maintenance or repairs, and participating in active games and sports. The results of several studies, however, indicate that an intensity threshold of approximately 7 kcal/min (e.g., brisk walking, heavy gardening) may exist, with exercise more vigorous than this providing greater protection than a similar amount of less vigorous activity (Cassel et al., 1971; Morris et al., 1990). Of greatest benefit seems to be large muscle dynamic or “aerobic” activity that substantially increases cardiac output with rather small increases in mean arterial blood pressure. Such activity is in contrast to heavy resistance or isometric exercise that substantially increases arterial blood pressure with a relatively small increase in cardiac output. Participation in “physical fitness” or “athletic conditioning” programs contributes little to the more active classification in most observational studies so far reported.

On - the - Job Act iv it y

Most of the initial observations establishing an association between physical activity status and CHD manifestations used on-the-job activity. It is much easier (but likely less accurate) to classify individuals as inactive or active according to their job title or description than it is to obtain self-assessments by interview, questionnaire, or direct observation of job-related or leisure-time activity. The early studies that obtained a difference in CHD rates between inactive and active classifications, such as the reports by Morris on London busmen (Morris et al., 1953), Kahn’s observation on Washington, D.C., postal workers (Kahn, 1963) or Taylor’s studies on U.S. railroad workers (Taylor et al., 1970) included job situations in which the major exercise of the more active groups was walking on flat ground, up stairs, or up and down hills. If the more physically active status is responsible for the lower CHD rates in these populations, the intensity threshold for an activity-related benefit is not high. Other on-the-job studies, however, have not found any protective association with activity level until a classification of “heavy work” was obtained (Cassel et al., 1971; Morris et al., 1953; Morris et al., 1990).

The estimated net energy expenditure (above the energy expenditure of the inactive group) associated with a decrease in CHD mortality in the various occupational studies ranges from 300 to 800 kcal/day. The intensity of the activity contributing to this increased energy expenditure includes walking, lifting and carrying objects, farming, and laboring-type jobs. In most cases, the higher-intensity activities (i.e., heavy lifting or carrying) are performed in relatively short bursts throughout the workday with the lower intensities (i.e., walking) being carried out for longer durations.

Leisure-Time Activity

Accurate quantitative assessments of leisure time or non-job activity status are difficult to obtain on samples of the size needed to evaluate the relationship of habitual activity status and CHD clinical events. Various diary and recall techniques have been used, and they all present significant administration and scoring difficulties. However, in the studies that have attempted to quantitate various aspects of non-job activities, several have identified an inverse association between activity status and CHD similar to the relationship reported in the earlier occupational-based studies.

Evidence of some protection possibly being provided by non-job, low-intensity activity on a regular basis was first reported by Rose (1969). He observed that the prevalence of "ischemic-type" resting electrocardiographic (ECG) abnormalities was inversely associated with duration of walking to work among 8,948 executive grade civil service workers in London. Those employees who walked 20 or more minutes to work on a regular basis had one third fewer ECG abnormalities than their counterparts who rode to work. This association could not be accounted for as a result of differences in age, grade of employment, smoking habits, serum cholesterol, or glucose tolerance. Those who walked regularly, however, tended to be a little less overweight.

The relationship of leisure-time activity to CHD mortality among middle-aged male civil servant workers in Britain has been studied using a two-day activity recall procedure (Morris et al., 1980; Morris et al., 1990). Morris and coworkers reported that non-job activity, as assessed by a self-administered 48-hour recall questionnaire completed on a Monday for the preceding Friday and Saturday, was significantly associated with CHD mortality only when activities requiring a peak energy expenditure of 7.5 kcal/min for 30 minutes or longer each day were performed ("vigorous activity"). Lesser amounts of activity appeared to carry no protective benefit. This apparent protective action of "vigorous activity" was not related to plasma total cholesterol, blood pressure, cigarette smoking, or adiposity, and it occurred at all ages from 40 to 69 years.

Paffenbarger and colleagues (1986, 1993) have continued to evaluate the relationship between past and recent physical activity habits and cardiovascular health in Harvard University alumni. Analyses of 572 first heart attacks among 16,936 men between 1962 and 1972 and 1,413 total deaths between 1962 and 1978 showed that it was habitual post-college exercise, not student sports play, that predicted low coronary heart disease risk. They have shown that sedentary alumni who were ex-varsity athletes had high risk while sedentary students who became active in later life seemed to acquire a low risk. These results are similar to several reports of job-related activity and heart disease risk where more physically active jobs early in a career followed by years of sedentary work resulted in higher risk than when the active job was continued throughout a person's career.

The relationship of self-selected leisure-time physical activity (LTPA) to first major CHD events and overall mortality was studied in 12,138 middle-aged men participating in the Multiple Risk Factor Intervention Trial (Leon et al., 1987). Total LTPA over the preceding year was quantitated in mean minutes per day at baseline by questionnaire, with subjects classified into tertiles (low, moderate, and high) based on LTPA distribution. During seven years of follow-up, moderate LTPA was associated with 64% as many fatal CHD events and sudden deaths, and 73% as many total deaths as low LTPA ($p < .01$). Mortality rates with high LTPA were similar to those in moderate LTPA; however, combined fatal and nonfatal major CHD events were 20% lower with high as compared with low LTPA ($p < .05$). These risk differentials persisted after statistical adjustments for possible confounding variables, including other baseline risk factors.

Physical activity at work and in leisure time was studied by questionnaire in a random sample of residents living in Eastern Finland (Salonen, Puska, & Tuomilehto, 1982). The study population consisted of 3,978 men aged 30–59 years and 3,688 women aged 35–59 years. During a seven-year follow-up, low physical activity at work was associated with an increased risk of myocardial infarction, cerebral stroke, and death due to any disease in both men and women, even after controlling for age, cholesterol, diastolic blood pressure, weight and smoking status using a multiple logistic model. The relative risk for myocardial infarction was 1.5 (95% confidence interval 1.2–2.0) for men and 2.4 (95% confidence interval 1.5–3.7) for women. Men and women at highest risk were those who reported no vigorous exercise during either work or leisure time while those at lowest risk reported vigorous exercise during both times.

Recently Lakka and colleagues (1994) reported the results of following 1,453 men aged 42 to 60 years for about five years. In the more active third of the men (>2.2 hours per week of activity) the relative risk of a myocardial infarction was 0.31 (95% confidence interval = 0.12 to 0.85; $p = 0.02$) compared to the least active third. Similar results were obtained when aerobic capacity as determined by maximal oxygen uptake was related to risk of myocardial infarction.

A major criticism of the observational studies that demonstrate a protective effect of exercise is that the differences in CHD rates between active and inactive individuals may be due to less healthy people selecting a less active lifestyle, not that increased activity prevents disease. Such self-selection may account for some of the differences reported; but in several reports, the investigators considered this problem in their data analyses and still found that being physically active was of significant benefit (Kahn, 1963; Paffenbarger, Wing, & Hyde, 1978). Also, Paffenbarger and colleagues (1993) recently reported that an increase in activity from one examination to the next was associated with a lower CHD mortality rate. These results strengthen the argument that it is an increase in activity that causes the reduction in mortality.

PHYSICAL FITNESS AND PRIMARY PREVENTION

Only in the past decade have studies been published that have adequately measured cardiovascular functional capacity or physical fitness on a sample of sufficient size and then followed their clinical status long enough to be able effectively to evaluate the relationship of physical fitness to future CHD or total mortality. If a higher level of habitual activity causes a reduction in cardiovascular morbidity and mortality, then a similar association should be observed with an accurate and reliable measure of fitness.

This issue was examined in a study of 4,276 men, 30 to 69 years of age, who were screened as part of the Lipid Research Clinic's prevalence survey and followed for an average of 8.5 years (Ekelund et al., 1988). Examination at baseline included assessment of conventional coronary risk factors and treadmill exercise testing. The heart rate during submaximal exercise (stage 2 of the Bruce exercise test) and the duration of exercise were used as measures of physical fitness. After adjustment for age and cardiovascular risk factors, a lower level of physical fitness was associated with a higher risk of death from CVD and CHD. The relative risk for death due to CVD for the least fit healthy men versus the most fit healthy men was 3.6 (95% confidence interval = 1.6 to 5.6; $p = 0.0004$) and for death due to CHD it was 2.8 (95% confidence interval = 1.3 to 6.1; $p = 0.007$). Highly significant associations were also seen for men who had CVD at their initial evaluation and for all-cause mortality. Thus, a low level of physical fitness is associated with a higher risk of death, especially from CVD and CHD, in men independent of conventional risk factors.

The relationship of physical fitness, as measured by maximal treadmill performance, to all-cause and cause-specific mortality was evaluated in 10,224 men and 3,120 women who had completed comprehensive medical examinations at the Cooper Clinic (Blair et al., 1989). Average follow-up was slightly more than eight years, for a total of 110,482 person-years of observation. There were 240 deaths in men and 43 deaths in women. Age adjusted all-cause mortality rates declined across physical fitness quintiles from 64.0 per 10,000 person-years in the least-fit men to 18.6 per 10,000 person-years in the most-fit men. Corresponding values for women were 39.5 per 10,000 person-years to 8.5 per 10,000 person-years. These trends remained after statistical adjustments for age, smoking habit, cholesterol level, systolic blood pressure, fasting blood glucose level, parental history of coronary heart disease, and follow-up interval. Higher levels of physical fitness appeared to delay all-cause mortality primarily due to lowered rates of cardiovascular disease and cancer.

The relationship of maximal oxygen uptake measured during a maximal test on a cycle ergometer to CVD mortality during a 16-year follow-up was reported for 2,014 Norwegian men initially aged 40 to 59 years (Sandvick et al., 1993). The relative risk of death from any cause in fitness quartile 4 (highest) as compared with quartile 1 (lowest) was 0.54 after adjustment for age, smoking status, serum lipids, blood pressure, resting heart rate, vital capacity, body mass index, level of physical activity, and glucose tolerance. The adjusted relative risk of death from CVD in fitness quartile 4 as compared with quartile 1 was 0.41 ($p = 0.013$). The corresponding relative risks for quartile 3 and 2 (as compared to quartile 1) were 0.45 ($p = 0.026$) and 0.59 ($p = 0.15$), respectively.

The results of these three physical fitness studies and the data reported by Sobolski, Kornitzer, and De Backer (1987) and Lakka and colleagues (1994) are highly consistent with the observational data on physical activity and cardiovascular mortality: the least fit and active have the highest rate of disease with only moderate increases in fitness and activity associated with a significant reduction in risk. There is a continued dose-response relationship at higher levels of fitness, but the magnitude of the benefit tends to decline as fitness levels increase, with the most fit generally having the lowest CHD mortality rate.

SECONDARY PREVENTION OF CORONARY HEART DISEASE

As with primary prevention, there is no definitive study demonstrating a significant reduction in new cardiac events as a result of exercise training in patients with established CHD. But in addition to studies that have simply compared morbidity or mortality rates in active with inactive cardiac patients, controlled experimental trials have been conducted in which myocardial infarction patients have been randomly assigned to exercise and control groups.

Here again, the trend in mortality favors the more physically active patients, with benefits apparently derived from an increase in caloric expenditure of no more than 300 to 400 kcal per session three to four times per week at a moderate intensity (60 to 75% of maximal exertion or aerobic capacity). All of the studies published, which show either no differences or lower mortality rates in the active population, have either design or implementation flaws that prohibit definitive conclusions regarding the hypothesis, "Does an increase in exercise reduce the future likelihood of recurrent myocardial infarction, cardiac arrest, or sudden cardiac death?"

Randomized clinical trials of cardiac rehabilitation following hospitalization for myocardial infarction usually have demonstrated a tendency for lower mortality in treated patients, but a statistically significant reduction occurred in only one trial (Wilhelmsen et al., 1975; Rechnitzer et al., 1983; Shaw, 1981). To overcome the problem of inadequate power of any one study to detect small but clinically important benefits on cardiovascular morbidity and mortality in randomized trials of rehabilitation, a meta-analysis was performed on the combined results of ten clinical trials (Oldridge et al., 1988). All of the trials had to have good documentation of myocardial infarction, randomization of patients, a rehabilitation program lasting at least six weeks, follow-up for 24 months or longer, and comprehensive documentation of outcome. Data on a total of 4,347 patients were analyzed. The pooled odds ratio of 0.76 (95% confidence interval, 0.63 to 0.92) for all-cause deaths and of 0.75 (95% confidence intervals, 0.62 to 0.93) for cardiovascular death were significantly lower for the rehabilitation group than the control group, with no significant difference for recurrent myocardial infarction. A similar review, but evaluating a total of 22 randomized trials of rehabilitation after myocardial infarction reached a very similar conclusion (O'Conner et al., 1989).

BIOLOGIC MECHANISMS PROTECTING AGAINST CORONARY HEART DISEASE

A variety of biologic changes or mechanisms have been proposed to explain how physical activity might decrease the development of CHD clinical manifestations or improve the clinical status of patients with CHD. Most of these changes either decrease myocardial oxygen demand or increase myocardial oxygen supply and thus decrease the likelihood of myocardial ischemia at rest or during exercise. These mechanisms can be classified as either those that contribute to the maintenance or increase of oxygen supply to the myocardium or those that contribute to a decrease in myocardial work and oxygen demands. Also, it is possible that exercise training enhances the intrinsic mechanical or metabolic functioning of the myocardium or increases its electrical stability. It is these very same mechanisms through which all preventive and therapeutic measures for reducing clinical manifestations of CHD work. The specific data supporting the possible existence of these mechanisms have been extensively reviewed recently (Bouchard, Shephard, & Stephens, 1994).

CARDIOVASCULAR RISKS DURING EXERCISE

When someone dies suddenly due to a “heart attack” during vigorous recreational or sporting activities the event receives much more publicity than if the same individual had died while at home or work. Because of this publicity, the percentage of all sudden cardiac deaths (SCD) that occur during sporting activities in the general population probably is lower than it would seem based on casual observation. However, if an individual has underlying cardiac disease that significantly reduces myocardial perfusion, the increased myocardial oxygen demand imposed by either vigorous static or dynamic exercise can precipitate sudden cardiac arrest or sudden death (Thompson & Mitchell, 1984).

Adults in the general population who participate in vigorous activities such as jogging, long-distance running, cross-country skiing, cycling, or vigorous sports may be at greater risk of SCD during exercise than when not exercising (Siscovick et al., 1984; Thompson et al., 1982; Vuori, Makarainen, & Jaaselainen, 1978). For example, in a study of 133 men who died suddenly of cardiac arrest without known prior heart disease, Siscovick et al. (1984) reported that while the overall risk of cardiac arrest was lower in men performing habitual physical activity, the risk of primary cardiac arrest was transiently increased during vigorous exercise compared to that at other times. Among men who performed vigorous activity very infrequently the risk of cardiac arrest during that activity was 56 times greater (95% confidence limits = 23 to 131) than at other times, but this risk decreased to a factor of 5 (95% confidence limits = 2 to 14) among men at the highest level of vigorous activity. Based on the circumstances surrounding 2,606 sudden deaths in Finland during one year, Vuori and colleagues (1978) concluded that SCD in connection with sporting activities in the general population are quite rare; instantaneous deaths were even rarer (<1% of all incidences of SCD) and occurred only with coexisting activity. Of the deaths associated with exercise, 73% were caused by acute or chronic ischemic heart disease and most of the subjects had serious cardiovascular risk factors that were known in advance or could have been identified easily.

In adults who have had a recent medical evaluation, the risk for SCD during exercise is extremely small. Gibbons and colleagues (1980) documented only two nonfatal cardiac arrests and no deaths in 374,798 person-hours of vigorous exercise. In a very large experience obtained by Vander and associates (Vander et al., 1982) from 40 exercise facilities over five years, the fatality rate associated with exercise in the general population was quite low. In 33,726,000 participant-hours of exercise, only 38 fatal cardiovascular complications occurred for a fatality rate of one death every 887,526 hours of participation. This means one could expect one death per year if 3,400 adults were exercising five hours per week each. Also, the mortality rate while exercising in this group is about one percent per year, nearly the same as the annual CHD mortality rate for middle-aged men in the United States.

FIGURE 9.1

Biological mechanisms by which exercise may contribute to the primary or secondary prevention of coronary heart disease.*

Maintain or increase myocardial oxygen supply

- Delay progression of coronary atherosclerosis (possible)
 - Improve lipoprotein profile (increase HDL-C/LDL-C ratio) (probable)
 - Improve carbohydrate metabolism (increase insulin sensitivity) (probable)
 - Decrease platelet aggregation and increase fibrinolysis (probable)

Decrease adiposity (usually)

Increase coronary collateral vascularization (unlikely)

Increase epicardial artery diameter by dilatation or remodeling (possible)

Increase coronary blood flow (myocardial perfusion) or distribution (possible)

Decrease myocardial work and oxygen demand

Decrease heart rate at rest and during submaximal exercise (usually)

Decrease systolic and mean arterial pressure during submaximal exercise (usually) and at rest (possible)

Decrease cardiac output during submaximal exercise (possible)

Decrease circulating plasma catecholamine levels (decrease sympathetic tone) at rest (probable) and at submaximal exercise (usually)

Increase myocardial function

Increase stroke volume at rest and in submaximal and maximal exercise (likely)

Increase ejection fraction at rest and during exercise (likely)

Increase intrinsic myocardial contractility (possible)

Increase myocardial function resulting from decreased "afterload" (probable)

Increase myocardial hypertrophy (probable); but this may not reduce CHD risk

Increase electrical stability of myocardium

Decrease regional ischemia at rest or at submaximal exercise (possible)

Decrease catecholamines in myocardium at rest (possible) and at submaximal exercise (probable)

Increase ventricular fibrillation threshold due to reduction of cyclic AMP

(possible)

*Expression of likelihood that effect will occur for an individual participating in endurance-type training program for 16 weeks or longer at 65% to 80% of functional capacity for 25 minutes or longer per session (300 kilocalories) for three or more sessions per week ranges from unlikely, possible, likely, probable, to usually.

Abbreviations: HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; CHD = coronary heart disease; AMP = adenosine monophosphate.

PHYSICAL ACTIVITY TO PROMOTE CARDIOVASCULAR HEALTH

Recently a consensus was reached by an expert panel working under the auspices of the Centers for Disease Control and Prevention and the American College of Sports Medicine on a "public health recommendation" for promoting physical activity (Pate et al., 1995). While these recommendations were made on the basis that improvement in overall health of the person was the primary goal, much of the data supporting these recommendations were derived from the favorable relationship between physical activity and improved CHD risk factor status and lower CHD mortality. Thus, these guidelines provide a useful framework for recommending a program of physical activity to promote cardiovascular health. The essence of these recommendations is that all adults will benefit by performing at least 30 minutes of physical activity at a moderate intensity or higher on most days. For maximum cardiovascular benefits, this exercise should be of the endurance or aerobic type using the larger muscles of the legs or trunk including brisk walking, jogging, hiking, cycling, swimming, rowing, aerobic and active social dancing, selected calisthenics, and a variety of active games or sports.

A new twist in these recommendations as compared to those issued previously by various organizations is that the 30 minutes of activity can be achieved by performing short bouts of moderate intensity activity throughout the day in addition to performing a single bout of activity for 30 minutes or longer. These recommendations are not precise on the issue of how long these short bouts of activity need to be to warrant credit as time exercising. My suggestion, until more scientific data are available, is to assign credit to only those bouts of activity that last for five minutes or longer. The activity during these bouts needs to be of at least moderate intensity (defined as activity equivalent in intensity to that of brisk walking). These guidelines emphasize that prior guidelines by the President's Council on Physical Fitness and Sports, the American College of Sports Medicine, and the American Heart Association are still valid and that these new recommendations are an attempt to expand the opportunity for adults to exercise for health benefits. These guidelines do not change either the recommended intensity or amount of activity to be performed. Similar guidelines for children and youth were recently published (Sallis et al., 1994).

Existing scientific data strongly support the value of frequently performed activity of moderate intensity as part of a comprehensive program of heart disease prevention and cardiac rehabilitation. Physical activity and endurance fitness make contributions to decreased risk independent of other established heart disease risk factors and can provide substantial health benefits beyond cardiovascular health (Bouchard, Shephard, & Stephens, 1994). Along with stopping smoking, maintaining a physically active lifestyle is one of the least expensive and most productive health behaviors available to the public.

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