Relationship Between Low Cardiorespiratory Fitness and Mortality in Normal-Weight, Overweight, and Obese Men

Ming Wei, MD, MPH
James B. Kampert, PhD
Carolyn E. Barlow, MS
Milton Z. Nichaman, MD, ScD
Larry W. Gibbons, MD, MPH
Ralph S. Paffenbarger, Jr, MD, DrPH
Steven N. Blair, PED

Between 1976 and 1980 to 1988 and 1994, the prevalence of obesity in the United States increased substantially, from 14.5% to 22.5%.1 In 1998, the US National Institutes of Health (NIH) and the World Health Organization published guidelines for the treatment of obesity.2,3 In these reports, overweight and obesity are defined, and treatment guidelines are provided for physicians. It is recommended that clinicians first classify patients by body mass index (BMI), calculated as weight in kilograms divided by the square of height in meters, with overweight defined as a BMI of 25.0 to 29.9 kg/m² and obesity as a BMI of at least 30.0 kg/m². Further stratification of risk is recommended by considering the presence of coexisting disease and cardiovascular disease (CVD) risk factors.

The NIH guidelines specify that obese persons with established CVD or type 2 diabetes mellitus (DM) are at “very high risk” for death and that patients with 3 or more CVD risk factors are “at high absolute risk.”4 Physical inactivity and serum triglyceride levels of more than 2.3 mmol/L (>200 mg/dL) are mentioned as “other risk factors” that indicate “incremental absolute risk” above that estimated from the preced-

©1999 American Medical Association. All rights reserved.
CARDIORESPIRATORY FITNESS AND MORTALITY IN MEN

METHODS

Patient Data

This study is based on data from the Aerobics Center Longitudinal Study (ACLS), an observational study of patients examined at a preventive medicine clinic in Dallas, Tex, from 1970 to 1993. The study has been reviewed and approved annually by the Cooper Institute Institutional Review Board. Study participants come to the clinic for periodic health examinations and counseling about diet, exercise, and other lifestyle factors associated with increased risk of chronic disease. Many participants are sent by their employers for the examination, some are referred by their personal physicians, and others are self-referred. We excluded patients with a history of cancer at baseline, those with a BMI of less than 18.5 kg/m² at the baseline examination, those younger than age 20 at baseline, and those with less than 1 year of follow-up.

Patients came for the examination after an overnight fast of at least 12 hours and gave their informed consent to participate in the examination and the follow-up study. Patients completed an extensive self-report of demographic characteristics, personal and family health history, and health habits, including a history of smoking and physical activity questionnaire. Patients underwent a physical examination by a physician. Trained technicians using procedures described in a detailed manual of operations conducted all examinations, which included measuring height, weight, and blood pressure; determining cardiorespiratory fitness by administering a maximal exercise test on a treadmill; and drawing blood for blood chemistry analysis. Lipid and fasting plasma glucose levels were determined by automated techniques in the Cooper Clinic laboratory, which participates in and meets quality control standards of the Centers for Disease Control and Prevention Lipid Standardization Program.

Statistical Analyses

This study uses all-cause and CVD mortality (International Classifications of Diseases, Ninth Revision, codes 390-449) as the outcome variables. The principal exposure variable for this report was body habitus. We assigned the men to 1 of 3 BMI categories using criteria from guidelines for the evaluation and treatment of obesity: normal weight (BMI, 18.5-24.9 kg/m²), overweight (BMI, 25.0-29.9 kg/m²), or obese (BMI ≥30.0 kg/m²). We calculated mortality rates for BMI strata by the presence or absence of 6 mortality predictors.

Two of the mortality predictors were disease conditions. Baseline CVD was ascertained by the medical history, physical examination, and exercise test. The definition of baseline CVD was previous myocardial infarction, stroke, myocardial revascularization, abnormal electrocardiogram at rest or during the exercise test, or failure to achieve at least 85% of a patient’s age-predicted maximal heart rate during the exercise test. Some patients were unable to continue the exercise test due to untoward signs or symptoms. Individuals with early test termination for any of these reasons would have their cardiorespiratory fitness underestimated and would be more likely than other patients to be classified as having low fitness. The reasons for early test termination also are likely to be associated with baseline chronic disease, which could lead to early mortality. Therefore, our conservative approach was to include these patients in the baseline CVD group. The second disease condition used as an exposure variable was type 2 DM, defined as a history of physician-diagnosed type 2 DM or having fasting plasma glucose levels of at least 7.0 mmol/L (≥126 mg/dL).

The other 4 exposure variables were CVD risk factors: high serum cholesterol levels, defined as serum cholesterol higher than 6.2 mmol/L (≥240 mg/dL); hypertension, defined as a history of physician-diagnosed hypertension or blood pressure of at least 140/90 mm Hg; current cigarette smoking; and low cardiorespiratory fitness (maximal MET cut points for low fitness in...
We used cut points for the other quantitative exposure variables that have been recommended previously.2,3,16-18

We used Cox partial likelihood methods to provide point estimates and 95% confidence interval (CI) estimates19 adjusted for the covariables (age, examination year, and parental history of CVD) and other mortality predictors. All reported \( P \) values are 2-sided. We first calculated crude and net survival curves for the 3 BMI categories. We calculated age- and examination year-adjusted CVD and all-cause mortality rates for the 3 BMI categories. We then performed cross-tabulation analyses of age- and examination year-adjusted mortality rates using the BMI categories and the presence or absence of the primary exposure variables. We repeated these cross-tabulations with additional adjustment for parental history of CVD and each of the other exposures. We also calculated multivariate-adjusted population-attributable risks (PAR) as \( \text{PAR} = (1 - 1/RR) \), where \( p \) is the proportion of exposed decedents and relative risk (RR) is the adjusted RR for the exposure.20 Note that adjusted PARs for separate factors do not sum to the adjusted PAR for the combined factors,21 unless these factors are mutually exclusive.22

**RESULTS**

The study population included 25 714 men followed up for approximately 10 years, for a minimum of 1 year. Baseline characteristics of study participants by BMI categories are shown in Table 1. The population is homogeneous, with more than 95% white and about 80% college graduates. Most of the subjects were executives and professionals. Prevalence rates for normal weight, overweight, and obesity were 41%, 46%, and 13%, respectively. Men who were overweight or obese were more likely than the normal-weight men to have baseline disease, smoke cigarettes, be sedentary, and

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Normal Height (18.5-24.9)</th>
<th>Overweight (25.0-29.9)</th>
<th>Obese (≥30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>10 623</td>
<td>11 798</td>
<td>3293</td>
</tr>
<tr>
<td>Man-years of observation</td>
<td>112 114</td>
<td>117 639</td>
<td>29 028</td>
</tr>
<tr>
<td>No. of deaths</td>
<td>367</td>
<td>500</td>
<td>158</td>
</tr>
<tr>
<td>No. of cardiovascular disease deaths</td>
<td>126</td>
<td>237</td>
<td>76</td>
</tr>
<tr>
<td>Age, y‡</td>
<td>42.5 (10.3)</td>
<td>44.7 (9.8)</td>
<td>44.5 (8.5)</td>
</tr>
<tr>
<td>Exercise tolerance, METs‡</td>
<td>12.5 (2.2)</td>
<td>11.2 (1.9)</td>
<td>9.7 (1.7)</td>
</tr>
<tr>
<td>Body mass index, kg/m²‡</td>
<td>23.2 (1.3)</td>
<td>27.0 (1.4)</td>
<td>33.2 (3.7)</td>
</tr>
<tr>
<td>Total cholesterol level, mmol/L [mg/dL]‡</td>
<td>5.3 (1.2) [205.0 (46.4)]</td>
<td>5.6 (1.1) [217.0 (43.0)]</td>
<td>5.8 (1.1) [224.2 (43.0)]</td>
</tr>
<tr>
<td>Triglyceride level, mmol/L [mg/dL]‡</td>
<td>1.2 (1.3) [106.2 (115.0)]</td>
<td>1.7 (1.6) [150.4 (142.0)]</td>
<td>2.3 (1.7) [204.0 (150.4)]</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg‡</td>
<td>78.6 (9.1)</td>
<td>81.5 (9.4)</td>
<td>85.5 (9.8)</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg‡</td>
<td>119.3 (13.5)</td>
<td>122.1 (13.6)</td>
<td>126.5 (13.8)</td>
</tr>
<tr>
<td>Alcohol use, g/wk‡</td>
<td>152.6 (275.2)</td>
<td>186.8 (312.3)</td>
<td>182.8 (335.0)</td>
</tr>
<tr>
<td>Current cigarette smoking, %</td>
<td>15.8</td>
<td>20.0</td>
<td>20.3</td>
</tr>
<tr>
<td>Physically inactive, %‡</td>
<td>28</td>
<td>38</td>
<td>50</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>17.1</td>
<td>26.2</td>
<td>41.6</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>3.0</td>
<td>4.7</td>
<td>10.0</td>
</tr>
<tr>
<td>Parental cardiovascular disease, %</td>
<td>24.7</td>
<td>28.0</td>
<td>28.8</td>
</tr>
<tr>
<td>Prevalent cardiovascular disease, %§</td>
<td>9.1</td>
<td>12.7</td>
<td>16.5</td>
</tr>
</tbody>
</table>

*All tests for homogeneity across body mass index groups were significant at \( P \leq .05. \) MET indicates metabolic equivalent. †Values are presented as mean (SD). ‡Men reporting no leisure-time physical activity in the 3 months before the examination. §History of cardiovascular disease, abnormal electrocardiogram at rest or during the exercise test, or low maximal exercise heart rate.
have a family history of CVD. Overweight and obese men also had less favorable levels of clinical and health habit variables than normal-weight men. During the follow-up period, there were 1025 deaths (439 due to CVD) during the 258 781 man-years of follow-up. Survival curves for CVD and all-cause mortality by BMI category are presented in the **Figure**. Obese men had a 2.6 times higher risk for CVD (95% CI, 2.0-3.6) and a 1.9 times higher risk for all-cause mortality (95% CI, 1.5-2.3), after adjustments were made for age and examination year compared with normal-weight men. Overweight men had intermediate death rates between normal-weight and obese men. The age- and examination year-adjusted RR for CVD and all-cause mortality (calculated by cross-tabulating categories of BMI and presence or absence of other exposure variables and using the referent category of normal-weight men who did not have the specific mortality predictor) are shown in **Table 2**. Obese men with CVD at baseline had a higher risk for CVD mortality and all-cause mortality than did normal-weight men with no history of CVD. Results of the analyses for DM, hypertension, elevated cholesterol levels, current smoking, and low-cardiorespiratory fitness showed similar patterns of risk for each of these

**Table 2.** Age- and Examination Year-Adjusted Relative Risk (RR) of Cardiovascular Disease and All-Cause Mortality by Body Mass Index (BMI) and Other Mortality Predictors in 25 714 Men*

<table>
<thead>
<tr>
<th>Mortality Predictor</th>
<th>Normal Weight†</th>
<th>Overweight†</th>
<th>Obese†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>54 (9653)</td>
<td>110 (10 306)</td>
<td>29 (2750)</td>
</tr>
<tr>
<td>Yes</td>
<td>7 (970)</td>
<td>127 (1492)</td>
<td>47 (543)</td>
</tr>
<tr>
<td>Type 2 diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>106 (10 301)</td>
<td>190 (11 242)</td>
<td>52 (2962)</td>
</tr>
<tr>
<td>Yes</td>
<td>20 (322)</td>
<td>47 (556)</td>
<td>24 (331)</td>
</tr>
<tr>
<td>Cholesterol levels ≤6.2 mmol (≤240 mg/dL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;6.2 mmol (&gt;240 mg/dL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>63 (8800)</td>
<td>126 (8706)</td>
<td>35 (1923)</td>
</tr>
<tr>
<td>Yes</td>
<td>63 (1823)</td>
<td>111 (3092)</td>
<td>41 (1370)</td>
</tr>
<tr>
<td>Current smoker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>93 (8942)</td>
<td>173 (9446)</td>
<td>56 (2623)</td>
</tr>
<tr>
<td>Yes</td>
<td>33 (1681)</td>
<td>64 (2352)</td>
<td>20 (670)</td>
</tr>
<tr>
<td>Low fitness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>81 (9636)</td>
<td>113 (9505)</td>
<td>16 (1619)</td>
</tr>
<tr>
<td>Yes</td>
<td>45 (987)</td>
<td>124 (2293)</td>
<td>60 (1674)</td>
</tr>
<tr>
<td>All-Cause Deaths (n = 1025)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>243 (9653)</td>
<td>307 (10 306)</td>
<td>86 (2750)</td>
</tr>
<tr>
<td>Yes</td>
<td>124 (970)</td>
<td>193 (1492)</td>
<td>72 (543)</td>
</tr>
<tr>
<td>Type 2 diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>328 (10 301)</td>
<td>415 (11 242)</td>
<td>114 (2962)</td>
</tr>
<tr>
<td>Yes</td>
<td>39 (322)</td>
<td>85 (556)</td>
<td>44 (331)</td>
</tr>
<tr>
<td>Cholesterol levels ≤6.2 mmol (≤240 mg/dL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;6.2 mmol (&gt;240 mg/dL)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>233 (8800)</td>
<td>277 (8706)</td>
<td>82 (1923)</td>
</tr>
<tr>
<td>Yes</td>
<td>134 (1823)</td>
<td>223 (3092)</td>
<td>76 (1370)</td>
</tr>
<tr>
<td>Current smoker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>283 (8942)</td>
<td>369 (9446)</td>
<td>113 (2623)</td>
</tr>
<tr>
<td>Yes</td>
<td>84 (1681)</td>
<td>131 (2352)</td>
<td>45 (670)</td>
</tr>
<tr>
<td>Low fitness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>265 (9636)</td>
<td>279 (9505)</td>
<td>35 (1619)</td>
</tr>
<tr>
<td>Yes</td>
<td>102 (987)</td>
<td>221 (2293)</td>
<td>123 (1674)</td>
</tr>
</tbody>
</table>

*Patients with a BMI of 18.5 to 24.9 kg/m² and without the mortality predictor served as the referent group for each analysis. Patients who were younger than 20 years, had a history of cancer, or had a BMI of less than 18.5 kg/m² were excluded from the analysis.

†Normal weight is defined as a BMI of 18.5 to 24.9; overweight, 25.0 to 29.9; and obese, at least 30.0 kg/m².
other risk predictors. When compared with the referent category, men
with the other mortality predictors had a steep direct gradient of risk across BMI
categories.

We repeated the analyses presented in Table 2 with additional adjustment
for parental history of CVD and each of the other exposure variables (data not
shown). The pattern of results was similar to those in Table 2, although RRs
were attenuated with the multivariate adjustment. However, each of the ex-
posure variables remained significantly associated with mortality in the
overweight and obese men. We also repeated the analyses in Table 2 for 2
groups of men, those followed up for less than 10 years and those followed up
for 10 or more years. The results from each of these analyses (data not
shown) were similar to those presented in Table 2.

There were substantial differences in the prevalence of the mortality predictors
in overweight and obese men. For example, for the 3293 obese men, low
fitness was the most common predictor with a prevalence rate about 5 times
higher than that of DM, which was the least common predictor. Hyperten-
sion had the highest prevalence in normal-weight and overweight men. The
multivariate-adjusted RRs and num-
ber of men with each of the mortality predictors for each BMI category, along
with the PAR for both CVD and all-
cause mortality, are shown in Table 3. We
performed a separate series of analy-
ses in each BMI stratum and calculated
multivariate-adjusted RRs for each mor-
tality predictor. The referent cat-

from the perspective of an individ-
ual patient, presence of CVD at base-
line is the strongest predictor of death
in all BMI strata, although low fitness is
similar to baseline CVD as a mortal-
ty predictor in obese men. From a
population perspective, baseline CVD
has the highest PAR in normal-weight
men, and CVD and low fitness have
comparable PARs in overweight and
obese men.

From the perspective of an individ-
ual patient, presence of CVD at base-
line is the strongest predictor of death
in all BMI strata, although low fitness is
similar to baseline CVD as a mortal-
ty predictor in obese men. From a
population perspective, baseline CVD
has the highest PAR in normal-weight
men, and CVD and low fitness have
comparable PARs in overweight and
obese men.

**COMMENT**

Overweight and obesity are prevalent
in the United States and in many other
countries. In the cohort of well-
educated men examined in this study,
46% were overweight and 13% were
obese, which is similar to percentage
rates for a representative sample of US
men. When compared with normal-
weight men in our study, obese men
had an almost 3-fold higher risk of CVD
mortality and a 2-fold higher risk of all-
cause mortality. These rates are com-
parable to other studies. Although car-
diorespiratory fitness has a genetic
component, which explains 25% to 40%
of the variation in fitness, it is clear that
habitual physical activity is the most
important determinant of fitness, and
fitness is improved in most individu-
als with appropriate exercise participa-
tion.

Data presented in this article sup-
port the hypothesis that low cardio-
respiratory fitness adds to overweight
and obesity in influencing mortality
adversely. The strongest predictor of
mortality in our data was baseline CVD,
which was expected. All other charac-
teristics that we evaluated (DM, el-

cated cholesterol levels, hyperten-
sion, current cigarette smoking, and low
fitness) were comparable predictors of
mortality in both overweight and obese
men. Overweight men with any of the
mortality predictors other than CVD

**Table 3. Multivariate Adjusted Relative and Population Attributable Risks (PAR) of Cardiovascular Disease and All-Cause Mortality by Body Mass Index (BMI) Categories and Other Characteristics in 25,714 Men**

<table>
<thead>
<tr>
<th>Mortality Predictor</th>
<th>Normal Weight†</th>
<th>Overweight†</th>
<th>Obese†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of Men</td>
<td>Cardiovascular Disease Death</td>
<td>All-Cause Mortality</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>322</td>
<td>1.5 (0.9-2.4) [5]</td>
<td>1.3 (0.9-1.8) [2]</td>
</tr>
<tr>
<td>High cholesterol levels</td>
<td>1621</td>
<td>1.2 (0.8-1.7) [6]</td>
<td>1.0 (0.8-1.3) [0]</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1823</td>
<td>2.1 (1.4-3.0) [26]</td>
<td>1.5 (1.2-1.9) [12]</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1681</td>
<td>1.7 (1.2-2.6) [11]</td>
<td>1.4 (1.1-1.8) [7]</td>
</tr>
<tr>
<td>Low fitness</td>
<td>987</td>
<td>1.7 (1.1-2.5) [15]</td>
<td>1.6 (1.3-2.1) [10]</td>
</tr>
</tbody>
</table>

©1999 American Medical Association. All rights reserved.
had about a 3-fold higher CVD death rate and a 2-fold higher all-cause death rate when compared with normal-weight men without the condition. Obese men with any one of the other characteristics other than baseline CVD had CVD death rates about 5-fold higher and all-cause death rates about 3-fold higher than in normal-weight men without the characteristic.

Low cardiorepiratory fitness was a strong predictor of mortality in our cohort, with RRs comparable with, if not greater than, the RRs for DM, high cholesterol levels, hypertension, and current cigarette smoking (Table 2 and Table 3). Our findings suggest that it is as important for a clinician to assess an obese patient’s fitness status as it is to measure fasting plasma glucose and cholesterol levels, evaluate blood pressure, and inquire about smoking habits. We recognize that many, if not most, primary care physicians may not have an exercise testing laboratory and that the cost of such measurements exceed those needed for obtaining blood lipid and glucose levels and measuring blood pressure. However, there is an extensive network of community facilities such as health clubs or YMCAs and YWCAs that offer fitness testing services performed by well-trained exercise clinicians for a modest cost.

If testing is not feasible, we encourage clinicians to evaluate their patients’ physical activity habits. This is probably important for all patients, but in view of our results, it is essential for overweight and obese patients. For example, the Physician Assisted Counseling for Exercise program includes simple scales to assess patients’ activity patterns and their motivational readiness to become more active, and the program’s efficacy has been confirmed. A behaviorally based, lifestyle, physical activity, counseling approach, in which sedentary individuals are encouraged to integrate more activity into their daily routines, by climbing stairs, taking short walks, and generally increasing daily activity, has been shown to be effective over a 2-year period.

PAR estimates for any characteristic are based on several assumptions and must be interpreted carefully. However, overweight or obese patients with baseline CVD have substantially increased risk for death, although the RRs for low fitness presented in Table 3 are nearly as high as they are for CVD. From a public health perspective, low fitness, with its high prevalence, also should receive attention. About 50% of the obese men in our study were unfit, whereas 16% had baseline CVD and 10% had DM. The prevalence of these conditions was 19%, 11%, and 5%, respectively, in overweight men. The PAR for all-cause mortality in obese men underscores the importance of low fitness. If the association between fitness and mortality is causal and if all obese unfit men in our cohort had been fit, there might have been as many as 44% fewer deaths among obese men in our study. If none of these men had CVD at baseline, there might have been as many as 27% fewer deaths. In overweight men, the PARs for all-cause mortality were comparable for low fitness and prevalent CVD.

Our study has several strengths. Our data on cardiorespiratory fitness are determined by a maximal exercise test on a treadmill, and the fitness data provide quantitative risk estimates. We also have laboratory measurements of CVD risk factors, which provide objective data on the other mortality predictors included in this report, and an extensive physical examination, which allows for thorough evaluation of the presence or absence of baseline disease. Our large sample size allowed us to perform cross-tabulation analyses to evaluate the various risk predictors by BMI strata and to analyze data in 2 follow-up intervals.

A limitation of our study is that it included only men, because we do not yet have enough deaths in the women in our cohort to perform analyses similar to those reported herein. However, in our previous reports on fitness in which we have been able to perform parallel analyses in men and women, results are generally similar. We also have few members of minority groups in our cohort, and the men in our study are primarily from mid- to upper-socioeconomic strata, so generalization to other groups should be done with caution. We only have baseline data on fitness, other exposures, and weight, so we do not know if changes in any of these variables occurred during follow-up or from the influence of possible changes on the results.

In conclusion, low cardiorepiratory fitness is as important as type 2 DM and other CVD risk factors as a predictor of CVD mortality and all-cause mortality in overweight or obese men. Clinicians should evaluate fitness in their patients just as they now obtain a medical history and measure blood pressure and cholesterol and plasma glucose levels. Evaluating fitness, or at least physical activity, allows for more complete risk stratification in overweight and obese patients and can enhance clinical decision making.

Author Affiliations: The Cooper Institute for Aerobics Research (Drs Wei, Kampert, and Blair and Ms Barlow), and Cooper Clinic (Dr Gibbons), Dallas, Tex; Westat Inc, Rockville, Md (Dr Nichaman); and Department of Health Research and Policy, Stanford University School of Medicine, Stanford, Calif (Dr Paffenbarger).

Financial Disclosures: Dr Blair has served as a consultant for Knoll Pharmaceutical Co, Mount Olive, NJ; Roche Laboratories, Nutley, NJ; and General Mills, Minneapolis, Minn; has received research funding from Polar Electro Oy, Oulu, Finland; McNeil Consumer Products, Fort Washington, Pa; and Knoll Pharmaceutical Co; has served on the scientific advisory boards of Life Fitness International, Franklin Park, Ill; E-Med, St Paul, Minn; Jenny Craig, La Jolla, Calif; and Bally Total Fitness Sports Medicine, Chicago, Ill; holds stock in LEARN Center, Dallas, Tex; and is on the board of directors for E-Med.

Funding/Support: This work was supported in part by US Public Health Service research grant AG06945 from the National Institute on Aging, Bethesda, Md, and by several grants from private contributors.

Acknowledgment: We thank the physicians and technicians of the Cooper Clinic for collecting the data for this study, Kenneth H. Cooper, MD, for initiating the Aerobics Center Longitudinal Study, Melba S. Morrow, MA, for editorial assistance, and Stephanie Parker for secretarial support. We are grateful for the guidance of the Scientific Advisory Board of the Cooper Institute.

REFERENCES


©1999 American Medical Association. All rights reserved.

The scientist takes off from the manifold observa-tions of predecessors, and shows his intelligence, if any, by his ability to discriminate between the impor-tant and the negligible, by selecting here and there the significant steppingstones that will lead across the difficulties to new understanding. The one who places the last stone and steps across to the terra firma of accomplished discovery gets all the credit. Only the initiated know and honor those whose patient integrity and devotion to exact observation have made the last step possible.

—Hans Zinsser (1878-1940)