Cardiorespiratory fitness, body composition, and all-cause and cardiovascular disease mortality in men[1–3]

Chong Do Lee, Steven N Blair, and Andrew S Jackson

ABSTRACT

Background: Cardiorespiratory fitness and body fatness are both related to health, but their interrelation to all-cause and cardiovascular disease (CVD) mortality is unknown.

Objective: We examined the health benefits of leanness and the hazards of obesity while simultaneously considering cardiorespiratory fitness.

Design: This was an observational cohort study. We followed 21 925 men, aged 30–83 y, who had a body-composition assessment and a maximal treadmill exercise test. There were 428 deaths (144 from CVD, 143 from cancer, and 141 from other causes) in an average of 8 y of follow-up (176 742 man-years).

Results: After adjustment for age, examination year, cigarette smoking, alcohol intake, and parental history of ischemic heart disease, unfit (low cardiorespiratory fitness as determined by maximal exercise testing), lean men had double the risk of all-cause mortality of fit, lean men (relative risk: 2.07; 95% CI: 1.16, 3.69; $P = 0.01$). Unfit, lean men also had a higher risk of all-cause and CVD mortality than did men who were fit and obese. We observed similar results for fat and fat-free mass in relation to mortality. Unfit men had a higher risk of all-cause and CVD mortality than did fit men in all fat and fat-free mass categories. Similarly, unfit men with low waist girths (< 87 cm) had greater risk of all-cause mortality than did fit men with high waist girths (≥ 99 cm).

Conclusions: The health benefits of leanness are limited to fit men, and being fit may reduce the hazards of obesity. *Am J Clin Nutr* 1999;69:373–80.

KEY WORDS

Body composition, cardiorespiratory fitness, epidemiology, mortality, cardiovascular disease mortality, all-cause mortality, fat mass, fat-free mass, waist girth, men

INTRODUCTION

Obesity is a public health problem in the United States (1) and the prevalence of obesity has increased substantially over the past few decades (2). However, the health effects of body fatness in relation to longevity are unclear. Many studies show increased mortality in the leanest as well as the most obese individuals (3–6), but others do not observe this trend (7–9). Manson et al (9) suggest that findings of high mortality rates in individuals with low weight-for-height are associated with methodologic limitations such as failure to control for cigarette smoking, failure to eliminate early mortality due to preexisting disease, and inappropriate control for obesity-related biological factors. Nonetheless, a recent meta-analysis documented elevated mortality in association with leanness after accounting for smoking and preexisting disease (10).

Another unexplored methodologic limitation in obesity research is that body mass index (BMI; in kg/m^2) is commonly used to examine the obesity-mortality association even though BMI is not an accurate measure of obesity. Rather, it mainly indicates overweight for height but does not discriminate between fat mass and fat-free mass (FFM). Some studies show higher death rates in individuals with low BMIs and high waist-to-hip circumference ratios (WHRs), but not in those with high BMIs and low WHRs (11–13). The health effects of overweight on height and body composition in relation to cardiovascular disease (CVD) risk factors need further research (14, 15). There has been little research on the relation between measured body fatness and mortality (16).

We believe that cardiorespiratory fitness should also be considered in examining the relation between body composition and mortality. Cardiorespiratory fitness is a powerful predictor of all-cause and CVD mortality (17–19) and appeared to attenuate the relation between BMI and mortality in an earlier study (20). However, the health effects of body fatness and cardiorespiratory fitness in relation to longevity remain unexplored. Therefore, the purpose of this study was to examine the health consequences of body fatness and cardiorespiratory fitness in relation to all-cause and CVD mortality in men. We also assessed the associations of fat mass, FFM, and waist circumference to mortality after taking cardiorespiratory fitness into account.

SUBJECTS AND METHODS

Subjects and measurements

Subjects were 21 925 men aged 30–83 y who had complete preventive medical evaluations between 1971 and 1989 at the

[1] From the Division of Epidemiology & Clinical Applications, Cooper Institute for Aerobics Research, Dallas, and the Department of Health and Human Performance, University of Houston.

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[3] Address reprint requests to SN Blair, 12330 Preston Road, Dallas, TX 75230. E-mail: sblair@cooperinst.org.

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Cooper Clinic in Dallas. All subjects were residents of the United States and had no personal history of myocardial infarction, stroke, or cancer at baseline. All received body composition assessments and reached ≥85% of their age-predicted maximal heart rate [220 − age (in y)] during their treadmill tests.

The study protocol was reviewed and approved annually by the Institutional Review Board. All subjects gave their informed, written consent for the medical evaluation and subsequent registration in the follow-up study. The medical evaluation, performed after subjects had fasted overnight for ≥12 h, included a physical examination, anthropometric measurements, electrocardiogram, blood chemistry analyses, blood pressure assessment, maximal exercise treadmill test, self-report of health habits, and recording of demographic characteristics. Additional details of examination procedures are published elsewhere (17–19).

Serum samples were analyzed by automated techniques in a laboratory that participates in the Centers for Disease Control and Prevention Lipid Standardization Program, and blood pressure was measured by auscultatory methods with a mercury sphygmomanometer. Body weight and stature were measured with a standard physician's scale and stadiometer. In a subgroup of 14043 men, waist circumference was measured at the level of the umbilicus with a plastic tape measure. Body composition was assessed either by hydrostatic weighing, by skinfold-thickness measurements, or both following a standard procedure (21). We determined percentage body fat in men by hydrodensitometry using Siri's (22) two-component model. We also measured the sum of 7 (Σ7) skinfold thicknesses and estimated skinfold fat using a generalized body density equation (23).

Not all subjects underwent both hydrostatic weighing and skinfold-thickness measurements; 9655 were measured for skinfold thickness only, 7180 for hydrostatic weight only, and 5090 for both measurements. To standardize these measurements, we developed a prediction model for hydrostatistically determining percentage body fat from percentage fat (%fat) estimated by Σ7 skinfold thicknesses from the 5090 men who provided both Σ7 skinfold thicknesses and hydrostatic weighing data. A regression analysis provided the following equation:

$$\text{Percentage body fat} = 1.511843 + 0.905469 \times \%\text{fat} \quad (1)$$

(SEE = 3.78; r = 0.82). We applied this prediction model to the skinfold data for the men who did not undergo hydrostatic weighing to estimate their percentage body fat. We further calculated fat mass and FFM (in kg) as follows:

$$\text{Fat mass (kg)} = \text{wt (kg)} \times (\%\text{fat}/100) \quad (2)$$
$$\text{FFM (kg)} = \text{wt (kg)} - \text{fat mass (kg)} \quad (3)$$

We assigned subjects to categories of lean, normal, or obese. These categories correspond to <25th, 25th to <75th, and ≥75th percentile scores. We also classified subjects as having low, moderate, and high categories of fat mass, FFM, and waist circumference by using these same percentile scores cutoff points.

Alcohol use, cigarette smoking habit, and parental history of ischemic heart disease (IHD; either parent died of IHD) were assessed by self-report on a medical history questionnaire. Alcohol consumption was classified as none, light (<15 units/wk), moderate (15–30 units/wk), and heavy (≥31 units/wk). One unit of alcohol intake was defined as a bottle or can of beer [355 mL (12 oz)], a glass of wine [148 mL (5 oz)], or 44 mL (1.5 oz) of hard liquor. Smoking status was classified as never smoked, former smoker, or current smoker. Current smokers were further classified as smoking <20, 20 to <40, and ≥40 cigarettes/d.

Cardiorespiratory fitness was measured by using a maximal treadmill exercise test as described previously (17). Total treadmill endurance time was used as an index of aerobic power; time on treadmill with this protocol correlated highly (r = 0.92) with

TABLE 1
Baseline characteristics of 21925 men across body fatness and cardiorespiratory fitness categories[1]

	Lean (<16.7% BF)		Normal (16.7 to <25.0% BF)		Obese (≥25.0% BF)		
	Fit (n = 5093)	Unfit (n = 327)	Fit (n = 9255)	Unfit (n = 1851)	Fit (n = 3217)	Unfit (n = 2182)	All men (n = 21925)
Age (y)	40.6 ± 8.0[2]	40.3 ± 8.3	44.5 ± 8.7	42.6 ± 8.2	48.0 ± 9.1	44.4 ± 8.6	43.8 ± 8.9
Height (cm)	178.9 ± 6.3	178.2 ± 6.9	178.9 ± 6.3	177.9 ± 6.4	178.9 ± 6.5	178.2 ± 6.6	178.7 ± 6.4
Weight (kg)	75.6 ± 8.5	77.3 ± 10.4	82.0 ± 9.1	83.9 ± 10.8	90.9 ± 12.0	96.8 ± 16.5	83.4 ± 12.4
Treadmill time (min)	22.0 ± 4.3	12.5 ± 2.5	18.5 ± 3.7	11.9 ± 2.4	15.8 ± 3.1	10.8 ± 2.6	17.5 ± 5.1
Systolic BP (mm Hg)	118.5 ± 13.1	119.8 ± 13.8	120.5 ± 13.2	121.7 ± 13.4	123.6 ± 13.9	126.6 ± 14.3	121.2 ± 13.6
Diastolic BP (mm Hg)	78.0 ± 9.0	80.3 ± 8.9	80.5 ± 9.2	81.9 ± 9.7	83.1 ± 9.3	85.1 ± 9.9	80.9 ± 9.5
Triacylglycerol (mmol/L)	1.1 ± 0.8	1.9 ± 3.0	1.5 ± 1.2	2.1 ± 1.7	1.7 ± 1.2	2.2 ± 1.7	1.6 ± 1.3
Serum glucose (mmol/L)	5.4 ± 0.7	5.7 ± 1.7	5.5 ± 0.7	5.7 ± 1.1	5.7 ± 0.9	5.9 ± 1.5	5.6 ± 0.9
Total cholesterol (mmol/L)	5.2 ± 0.9	5.5 ± 1.1	5.6 ± 1.3	5.7 ± 1.1	5.8 ± 1.0	5.8 ± 1.1	5.5 ± 1.2
BMI (kg/m^2)	23.6 ± 2.0	24.3 ± 2.6	25.6 ± 2.2	26.5 ± 2.7	28.3 ± 3.1	30.4 ± 4.6	26.1 ± 3.4
Percentage BF (%)	12.9 ± 3.0	13.5 ± 3.0	20.7 ± 2.3	21.5 ± 2.3	28.3 ± 3.4	30.1 ± 4.6	20.9 ± 6.4
Inactive (%)	20.9	77.4	28.7	70.5	39.3	70.0	36.8
Systolic BP ≥140 mm Hg (%)	7.4	10.1	9.3	11.4	13.2	20.1	10.7
Fasting glucose ≥6.7 mmol/L (%)	1.6	3.7	3.6	6.2	6.2	11.6	4.5
Abnormal electrocardiogram (%)	4.2	9.5	5.9	9.7	9.2	9.6	6.7
Family history of IHD (%)	23.5	22.9	29.6	27.9	32.2	31.6	28.5
History of hypertension (%)	9.4	14.5	14.9	20.2	20.6	29.6	16.4
History of diabetes (%)	1.8	4.9	1.8	5.5	2.8	5.1	2.6
Current smoker (%)	15.3	46.5	17.9	39.1	17.0	31.9	20.8
Past smoker (%)	26.1	13.2	30.3	19.3	32.7	24.2	27.9

[1] BF, body fat; BP, blood pressure; IHD, ischemic heart disease.
[2] $\bar{x}$ ± SD.

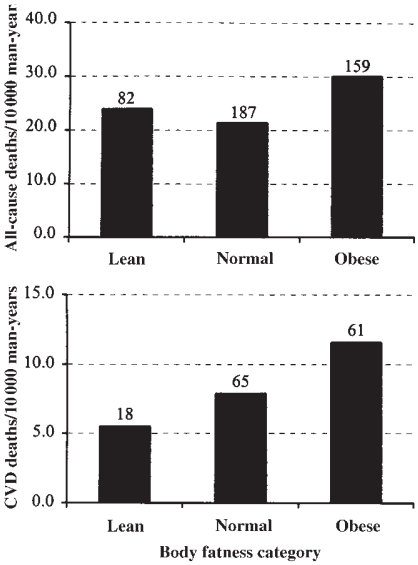

FIGURE 1. All-cause (top) and cardiovascular disease (CVD; bottom) death rates per 10 000 man-years of follow-up, adjusted for age (single year) and examination year, across body fatness categories. Body fatness categories were, in percentage body fat, lean (<16.7%), normal (16.7% to <25.0%), and obese (≥25.0%). Numbers atop the bars represent the number of deaths.

maximal oxygen uptake ($\dot{V}O_2$max) (24). Men in the least-fit 20% of each age group were classified as physically unfit, and all others as physically fit (18). We also calculated $\dot{V}O_2$max in $mL \cdot kg\ FFM^{-1} \cdot min^{-1}$ and classified men in the lowest quartile of oxygen uptake in each age group as physically unfit, and all others as physically fit. All subjects were cross-tabulated by cardiorespiratory fitness levels across body fatness categories as follows: *1*) fit and lean, *2*) unfit and lean, *3*) fit and normal, *4*) unfit and normal, (*5*) fit and obese, and *6*) unfit and obese. We also cross-tabulated by cardiorespiratory fitness levels across fat mass, FFM, and waist circumference categories.

All subjects were followed for mortality from the baseline examination to the date of death or to December 31, 1989. Deaths among study subjects were identified from the National Center for Health Statistics National Death Index and official death certificates from the departments of vital records of the various states. The underlying cause of death was determined by a nosologist according to the *International Classification of Diseases,* Ninth Edition, with CVD defined as codes 390 to 449.9.

Statistical analysis

All-cause and CVD death rates per 10 000 man-years (for which a man-year is 1 man followed for 1 y) of follow-up, adjusted for age and examination year, were calculated across body fatness and waist circumference categories. Proportional hazards regression was used to examine the associations among cardiorespiratory fitness, body fatness, and all-cause and CVD mortality (25). We also examined the associations among cardiorespiratory fitness, fat mass, FFM, and waist circumference to all-cause and CVD mortality. The relative risks (RRs) of all-cause and CVD mortality were estimated after adjustment for age and examination year and further adjustment for cigarette smoking, alcohol intake, and parental history of IHD. Physically fit men in the lowest quartile of each body composition variable were the reference category. The 95% CIs were calculated for each RR. All statistical procedures were performed with SAS software (26).

RESULTS

During an average of 8 y of follow-up (176 742 man-years), there were 428 deaths: 144 from CVD, 143 from cancer, and 141 from other causes. Baseline descriptive characteristics of the subjects across body fatness categories and cardiorespiratory fitness level are shown in **Table 1**. Unfit men had a slightly higher degree of body fatness than did their fit counterparts within lean, normal, and obese categories [0.6%, 0.8%, and 1.8% higher, respectively ($P < 0.001$)]; treadmill times were progressively lower in unfit men, indicating lower cardiorespiratory fitness, across lean, normal, and obese categories. Fit, lean men had the highest average estimated maximal aerobic power [13.4 metabolic equivalents (METs); 1 MET = $\dot{V}O_2$ ($3.5\ mL \cdot kg^{-1} \cdot min^{-1}$)], whereas unfit, obese men had the lowest average maximal aerobic power (8.7 METs). We tested differences between groups with a two-factor analysis of variance (continuous data) or log linear models (categorical data). The main effects for both fitness and fatness were highly significant ($P < 0.001$) for all variables except that height was not related to fatness. We also tested fitness and

TABLE 2
Body fatness and relative risks (RR) of all-cause and cardiovascular disease mortality by cardiorespiratory fitness level in men[1]

Body fatness category and cardiorespiratory fitness level	Man-years of follow-up	All-cause mortality			Cardiovascular disease mortality		
		Deaths	RR of death (95% CI)[2]	Multivariate RR of death (95% CI)[3]	Deaths	RR of death (95% CI)[2]	Multivariate RR of death (95% CI)[3]
	man-y (%)	*n*			*n*		
Lean (<16.7% body fat)							
Fit (*n* = 5093)	41 854 (23.7)	68	1.00	1.00	13	1.00	1.00
Unfit (*n* = 327)	3883 (2.2)	14	2.06 (1.15, 3.66)	2.07 (1.16, 3.69)	5	3.18 (1.13, 8.96)	3.16 (1.12, 8.92)
Normal (16.7 to <25.0% body fat)							
Fit (*n* = 9255)	68 546 (38.8)	127	0.80 (0.59, 1.08)	0.80 (0.59, 1.08)	43	1.43 (0.76, 2.66)	1.43 (0.77, 2.67)
Unfit (*n* = 1851)	19 669 (11.1)	60	1.61 (1.14, 2.28)	1.62 (1.15, 2.30)	22	2.91 (1.47, 5.79)	2.94 (1.48, 5.83)
Obese (≥25.0% body fat)							
Fit (*n* = 3217)	21 874 (12.4)	65	0.93 (0.65, 1.31)	0.92 (0.65, 1.31)	19	1.35 (0.66, 2.77)	1.35 (0.66, 2.76)
Unfit (*n* = 2182)	20 916 (11.8)	94	1.92 (1.40, 2.62)	1.90 (1.39, 2.60)	42	4.08 (2.18, 7.61)	4.11 (2.20, 7.68)

[1] Cardiorespiratory fitness level (fit or unfit) from reference 18.
[2] Adjusted for age (single year) and examination year.
[3] Adjusted for age (single year), examination year, smoking habit, alcohol intake, and parental history of ischemic heart disease.

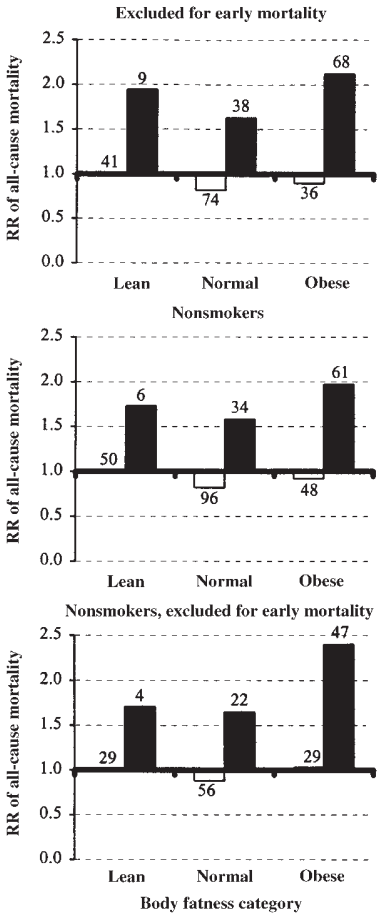

FIGURE 2. Body fatness and relative risks (RRs) of all-cause mortality by cardiorespiratory fitness categories in men: □, fit; ■, unfit. Fit, lean men comprised the reference category, represented by the heavy line at 1.0. Unfit men were the least-fit 20% of each age group, and fit refers to all other men (18). RRs were adjusted for age (single year), examination year, smoking habit, alcohol intake, and parental history of ischemic heart disease. Early mortality indicates the first 5 y of follow-up. Body fatness categories were, in percentage body fat, lean (<16.7%), normal (16.7% to <25.0%), and obese (≥25.0%). Numbers above or below the bars represent the number of deaths.

fatness interactions, and all were significant ($P \leq 0.001$) except height, diastolic blood pressure, and serum glucose.

All-cause and CVD death rates per 10 000 man-years of follow-up, adjusted for age and examination year across body fatness categories, are shown in **Figure 1**. There was a direct relation between body fatness and all-cause ($P = 0.01$ for linear trend) and CVD ($P = 0.004$ for linear trend) death rates.

The associations among cardiorespiratory fitness, body fatness, and all-cause and CVD mortality are shown in **Table 2**. Cox proportional hazards regression analyses, adjusted for age and examination year, showed that fit men had lower death rates than did their unfit counterparts within lean, normal, and obese categories. Unfit, lean men had twice the risk of all-cause mortality as did fit, lean men ($P = 0.02$) and also had higher risk (2.2 times) of all-cause mortality when compared with fit, obese men ($P = 0.008$). The all-cause mortality rate of fit, obese men was not significantly different from that of fit, lean men. These results were similar after additional adjustment for cigarette smoking, alcohol intake, and parental history of IHD. To evaluate further the possible bias of subclinical disease at baseline, we constructed another multivariate model by adding baseline electrocardiographic status (normal or abnormal). This analysis led to adjusted RRs that were nearly identical to those reported in Table 2. Exclusion for early mortality also made little difference in these results (**Figure 2)**.

We observed similar associations among cardiorespiratory fitness, body fatness, and all-cause mortality in nonsmokers and in nonsmokers with exclusion for early mortality and adjustment for age, examination year, alcohol intake, and parental history of IHD (Figure 2). Of nonsmokers, unfit, lean men had 1.7 times the risk of all-cause mortality of fit, lean men, with the highest all-cause mortality in unfit, obese men (RR: 1.97; 95% CI: 1.35, 2.88). Unfit nonsmokers in all body-composition groups had higher mortality risks than did fit nonsmokers after further exclusion for early mortality.

Unfit, lean men also had a high risk of CVD mortality when compared with their fit counterparts in all body fatness categories (Table 2). After multivariate adjustment for age, examination year, cigarette smoking, alcohol intake, and parental history of IHD, we observed that fit, lean men had the lowest CVD mortality, and that unfit, obese men had the highest. Unfit, lean men had 3.2 times the risk of CVD mortality of fit, lean men (95% CI: 1.12, 8.92; $P = 0.03$). However, fit, obese men had a lower risk of CVD mortality than did unfit, lean men.

When we further examined the relation of estimated $\dot{V}O_2$max (in $mL \cdot kg\ FFM^{-1} \cdot min^{-1}$) and body fatness with all-cause and

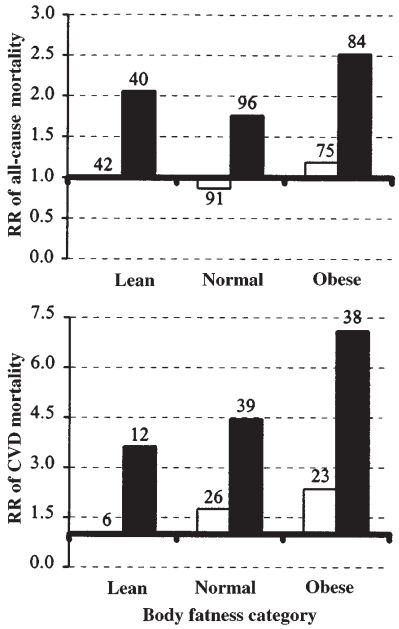

FIGURE 3. Body fatness and relative risks (RRs) of all-cause and cardiovascular disease (CVD) mortality by cardiorespiratory fitness categories in men: □, fit; ■, unfit. Fit, lean men comprised the reference category, represented by the heavy line at 1.0. Unfit men were the lowest quartile of oxygen uptake ($mL \cdot kg\ FFM^{-1} \cdot min^{-1}$) in each age group, and fit refers to all other men. RRs were adjusted for age (single year), examination year, smoking habit, alcohol intake, and parental history of ischemic heart disease. Body fatness categories were, in percentage body fat, lean (<16.7%), normal (16.7% to <25.0%), and obese (≥25.0%). Numbers above or below the bars represent the number of deaths.

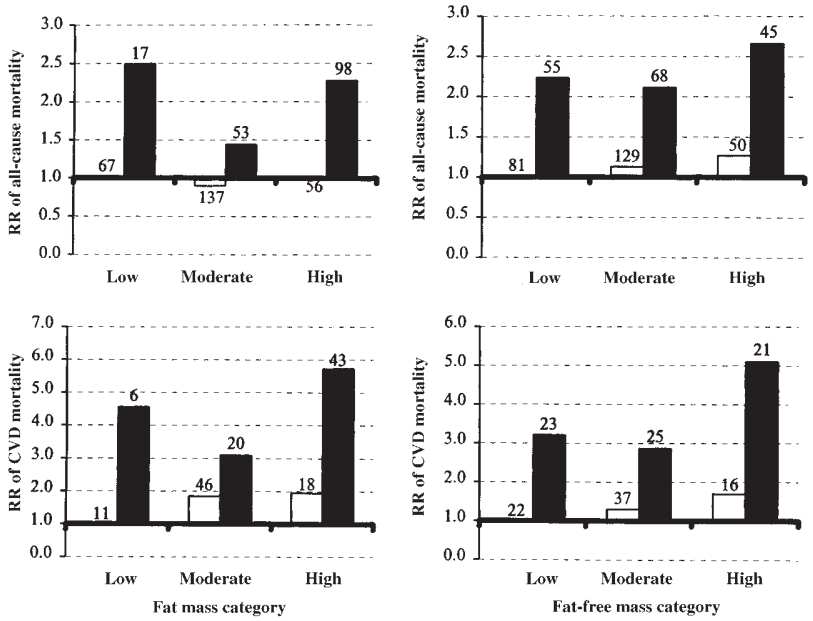

FIGURE 4. Fat mass, fat-free mass, and relative risks (RR) of all-cause and cardiovascular disease (CVD) mortality by cardiorespiratory fitness categories in men; □, fit; ■, unfit. Fit, lean men comprised the reference category, represented by the heavy line at 1.0. Unfit men were the least-fit 20% of each age group, and fit refers to all other men (18). RRs were adjusted for age (single year), examination year, smoking habit, alcohol intake, and parental history of ischemic heart disease. Fat mass measurements across low, moderate, and high categories were <12.8, 12.8 to <21.7, and ≥21.7 kg; fat-free mass measurements across these categories were <60, 60 to <70, and ≥70 kg. Numbers above or below the bars represent the number of deaths.

CVD mortality (**Figure 3**), similar results were obtained as for analyses in which fitness was expressed in $mL \cdot kg^{-1} \cdot min^{-1}$. Mortality risk was elevated in unfit, lean men, with the highest all-cause and CVD mortality in unfit, obese men. Unfit men had substantially higher risk of CVD in all fatness categories, but there also was a direct association between body fatness and CVD mortality in fit men ($P = 0.05$ for linear trend).

We also examined the relations of cardiorespiratory fitness, fat mass, and FFM to all-cause and CVD mortality (**Figure 4**). Unfit men had a higher risk of all-cause and CVD mortality than did fit men in all fat mass and FFM categories. Unfit men in the lowest quartile of fat mass and FFM had a greater risk of all-cause and CVD mortality than did fit men in the highest quartile of fat mass and FFM.

We also observed results similar to the analyses presented above when the men were stratified by waist circumference. There were 162 deaths (40 from CVD, 54 from cancer, and 68 from other causes) during an average of 5.6 y of follow-up (78 008 man-years of observation) in the subgroup of 14 043 men who had waist girth assessed at baseline. We calculated all-cause and CVD death rates per 10 000 man-years of follow-up and adjusted for age and examination year across waist circumference categories. All-cause and CVD death rates directly increased with larger sizes of waist girth, although the trends were not significant (**Figure 5**).

The associations among cardiorespiratory fitness, waist circumference, and all-cause mortality are shown in **Table 3**. After multivariate adjustment for age, examination year, cigarette smoking, alcohol intake, and parental history of IHD, we observed that fit men had lower risk of all-cause mortality in all waist circumference categories than unfit men. Unfit men in the lowest quartile of waist girth had 4.9 times ($P < 0.001$) the risk of all-cause mortality of their peers who were fit. In contrast, fit men in the highest quartile of waist girth had no elevated risk of all-cause mortality and had much lower mortality risk than unfit men in the lowest quartile of waist girth.

DISCUSSION

Although there is a strong direct relation between BMI and mortality (27), there has been little research on the relation between measured body fatness and mortality (16). We examined

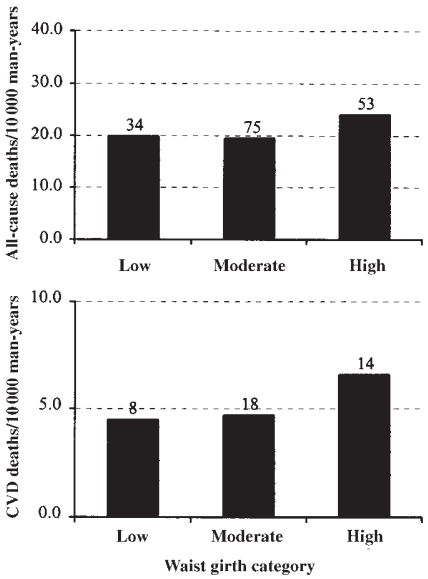

FIGURE 5. All-cause and cardiovascular disease (CVD) death rates per 10 000 man-years of follow-up, adjusted for age (single year) and examination year, across waist girth categories. Waist girth categories were low (<87 cm), moderate (87 to <99 cm), and high (≥99 cm). Numbers atop the bars represent the number of deaths.

TABLE 3
Waist circumference and relative risks (RRs) of all-cause mortality by cardiorespiratory fitness level in 14043 men[1]

Waist circumference category and cardiorespiratory fitness level	Deaths	Man years of follow-up	RR of death (95% CI)[2]	Multivariate RR of death (95% CI)[3]
	n	*man-y (%)*		
Low waist circumference (<87 cm)				
Fit (*n* = 3247)	26	18579 (23.8)	1.00	1.00
Unfit (*n* = 136)	8	1022 (1.3)	4.71 (2.13, 10.43)	4.88 (2.20, 10.83)
Moderate waist circumference (87 to <99 cm)				
Fit (*n* = 6237)	60	34189 (43.8)	1.08 (0.68, 1.71)	1.05 (0.66, 1.67)
Unfit (*n* = 616)	15	4211 (5.4)	2.08 (1.10, 3.93)	2.05 (1.08, 3.87)
High waist circumference (≥99 cm)				
Fit (*n* = 2645)	24	12994 (16.7)	0.98 (0.56, 1.72)	0.95 (0.54, 1.66)
Unfit (*n* = 1162)	29	7013 (9.0)	2.47 (1.45, 4.19)	2.40 (1.41, 4.07)

[1]Cardiorespiratory fitness level (fit or unfit) from reference 18.
[2]Adjusted for age (single year) and examination year.
[3]Adjusted for age (single year), examination year, smoking, alcohol intake, and parental history of ischemic heart disease.

the health effects of body composition among 21925 men after taking cardiorespiratory fitness into account. Our database is unique, with measures of body composition and maximal exercise test data on a large sample of men. We observed a direct relation between body fatness and all-cause and CVD mortality. However, being fit apparently decreased high mortality risk in obese men. This association was similar in nonsmokers and after exclusion for early mortality in both the entire population and in nonsmokers.

We observed similar results across strata of fat mass and FFM. Unfit men in the lowest quartile of fat mass and FFM had a greater risk of all-cause and CVD mortality than their fit counterparts. Fit men in the highest quartile of fat mass and FFM had a lower risk of all-cause and CVD mortality than did unfit, lean men. Our data indicate that cardiorespiratory fitness levels in men influence the health effects of obesity. We did not observe elevated mortality risk in men with high amounts of fat mass and FFM if they also were fit.

Several studies report that abdominal obesity is associated with elevated death rates (11–13). Although WHR has been commonly used to examine abdominal obesity, some studies suggest that waist girth rather than WHR is a better predictor of abdominal obesity (28–30). Measurement of waist girth instead of WHR for risk stratification is recommended in recent guidelines from the US National Institutes of Health and the World Health Organization (31, 32). Björntorp (33) reports that abdominal obesity, rather than peripheral obesity, also is associated with increased risk. Some studies show higher death rates in those with abdominal obesity who were underweight (a low BMI and high WHR) than in those without abdominal obesity who were overweight (a high BMI and low WHR) (11–13). No prior studies have reported the health effects of waist girth while also considering cardiorespiratory fitness. Our data show that fit men with low waist girth had lower risk of all-cause mortality than did unfit men in the same waist girth category. Unfit men with a high waist girth had a death rate 2.4 times greater than did the fit men with low waist girth, and fit men in the high waist girth category had a rate of all-cause mortality similar to fit men with low waist girth.

Our results support the hypothesis that moderate-to-high cardiorespiratory fitness reduces mortality risk across categories of body composition. Although most researchers agree that obesity is associated with health hazards and leanness is associated with health benefits, lean men in our study had increased longevity only if they were physically fit; furthermore, obese men who were fit did not have elevated mortality. In general, unfit, lean men were inactive and had low aerobic power despite their favorable IHD risk factor profiles at baseline, whereas fit, obese men were highly active and had high aerobic power at baseline.

A limitation of our study was that our subjects were white men in the middle and upper socioeconomic levels, although this homogeneity reduces the likelihood of confounding by socioeconomic characteristics. We hope that other investigators will examine these issues in other populations. The possibility of bias due to baseline health status is a consideration in all observational studies, including this one, but we think that serious bias is unlikely in this case because all study participants were given extensive medical examinations at baseline, which enabled us to exclude those with a history of myocardial infarction, stroke, or cancer. In addition, men who failed to achieve ≥85% of their age-predicted maximal heart rate on the maximal exercise test were excluded; this should have eliminated men who did not have a history of disease but were not feeling well as a result of an undiagnosed condition. We also adjusted the analyses for presence or absence of an abnormal electrocardiogram result. This exclusion eliminated men with angina, arrhythmia, or electrocardiographic abnormalities on the treadmill test, as well as resting electrocardiographic abnormalities. The effect of all these exclusion criteria was to minimize the possible bias of baseline subclinical disease. Another limitation of our study was that we estimated, rather than directly measured, residual lung volume during underwater weighing. Morrow et al (34) reported that the prediction accuracy of body fatness measured by densitometry when residual lung volume was estimated was only slightly better than anthropometric assessments. Nonetheless, the densitometry and skinfold-thickness estimates of body composition were likely to be more accurate measures of body fatness than BMI or height-weight indexes. Finally, we had only a one-time assessment of the exposure variables of cardiorespiratory fitness and body composition, and we do not know the extent to which these characteristics might have changed during follow-up. However, changes in the exposure variables during follow-up would cause misclassification and would be likely to lead to underestimates of RRs. Therefore, the true associations between fitness or body fatness and mortality may have actually been stronger than indicated by our results.

In summary, we found that obesity did not appear to increase mortality risk in fit men. For long-term health benefits we should focus on improving fitness by increasing physical activity rather than relying only on diet for weight control. Aerobic exercise improves IHD risk factors (35), and increases in physical activity or fitness extend longevity (18, 36). Although some studies show that there is no difference between diet and aerobic exercise in reducing IHD risk factors (37–39), or even report that diet is better than aerobic exercise for improving IHD risk factors in overweight men (40), our data show that fit men had greater longevity than unfit men regardless of their body composition or risk factor status. Obese men should be encouraged to increase their cardiorespiratory fitness by engaging in regular, moderate-intensity physical activity; this should benefit them even if they remain overweight.

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REFERENCES

1. Pi-Sunyner FX. Medical hazards of obesity. Ann Intern Med 1993;119:655–60.
2. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. JAMA 1994;272:205–11.
3. Rissanen A, Heliovaara M, Knekt P, Aromaa A, Reunanen A, Maatela J. Weight and mortality in Finnish men. J Clin Epidemiol 1989;42:781–9.
4. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. J Chronic Dis 1979;32:563–76.
5. Waaler HT. Height, weight and mortality: the Norwegian experience. Acta Med Scand Suppl 1984;679:1–56.
6. Seidell JC, Verschuren WM, van Leer EM, Kromhout D. Overweight, underweight, and mortality: a prospective study of 48,287 men and women. Arch Intern Med 1996;156:958–63.
7. Lee I-M, Manson JE, Hennekens CH, Paffenbarger RS Jr. Body weight and mortality: a 27-year follow-up of middle-aged men. JAMA 1993;270:2823–8.
8. Lindsted K, Tonstad S, Kuzma JW. Body mass index and patterns of mortality among Seventh-day Adventist men. Int J Obes 1991;15:397–406.
9. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. N Engl J Med 1995;333:677–85.
10. Troiano RP, Frongillo EA Jr, Sobal J, Levitsky DA. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. Int J Obes 1996;20:63–75.
11. Larsson B, Svärdsudd K, Welin L, Wilhelmsen L, Björntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. Br Med J 1984;288:1401–4.
12. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjostrom L. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of participants in the population study of women in Gothenburg, Sweden. Br Med J 1984;289:1257–61.
13. Folsom AR, Kaye SA, Sellers TA, et al. Body fat distribution and 5-year risk of death in older women. JAMA 1993;269:483–7.
14. Segal KR, Dunaif A, Gutin B, Albu J, Nyman A, Pi-sunyer X. Body composition, not body weight, is related to cardiovascular disease risk factors and sex hormone levels in men. J Clin Invest 1987;80:1050–5.
15. Van Itallie TB. Health implications of overweight and obesity in the United States. Ann Intern Med 1985;103:983–8.
16. Spataro JA, Dyer AR, Stamler J, Shekelle RB, Greenlund K, Garside D. Measures of adiposity and coronary heart disease mortality in the Chicago Western Electric Company Study. J Clin Epidemiol 1996;49:849–57.
17. Blair SN, Kohl HW, Paffenbarger RS Jr, Clarke DG, Cooper KH, Gibbons LW. Physical fitness and all-cause mortality: a prospective study of healthy men and women. JAMA 1989;262:2395–401.
18. Blair SN, Kohl HW, Barlow CE, Paffenbarger RS Jr, Gibbons LW, Macera CA. Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men. JAMA 1995;273:1093–8.
19. Blair SN, Kampert JB, Kohl HW, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. JAMA 1996;276:205–10.
20. Barlow CE, Kohl HW, Gibbons LW, Blair SN. Physical fitness, mortality and obesity. Int J Obes 1995;19:S41–4 (suppl).
21. Pollock ML, Wilmore JH, Fox SM III. Exercise in health and disease: evaluation and prescription for prevention and rehabilitation. Philadelphia: WB Saunders, 1984.
22. Siri WE. The gross composition of the body. In: Lawrence JH, Tobias CA, eds. Advances in biological and medical physics. New York: Academic Press, 1956.
23. Jackson AS, Pollock ML. Generalized equations for predicting body density of men. Br J Nutr 1978;40:497–504.
24. Pollock ML, Bohannon RL, Cooper KH, et al. A comparative analysis of four protocols for maximal treadmill stress testing. Am Heart J 1976;92:39–46.
25. Cox DR. Regression models and life tables. J R Stat Soc 1972;34:187–220.
26. SAS Institute. SAS/STAT software: the PHREG procedure, version 6. Cary, NC: SAS Institute Inc, 1991.
27. Baumgartner RN, Heymsfield SB, Roach AF. Human body composition and epidemiology of chronic disease. Obes Res 1995;3:73–95.
28. Pouliot MC, Després JP, Lemieux S, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol 1994;73:460–8.
29. Lemieux S, Prud'homme D, Bouchard C, Tremblay A, Despres JP. A single threshold value of waist girth identifies normal-weight and overweight subjects with excess visceral adipose tissue. Am J Clin Nutr 1996;64:685–93.
30. Seidell JC, Oosterlee A, Deurenberg P, Hautvast JGA, Ruijs JHJ. Abdominal fat deposits measured with computed tomography: effects of degree of obesity, sex, and age. Eur J Clin Nutr 1988;42:805–15.
31. National Institutes of Health, National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Rockville, MD: National Institutes of Health, National Heart, Lung, and Blood Institute, 1998.
32. World Health Organization. Obesity: preventing and managing the global epidemic. Geneva: World Health Organization, 1998.
33. Björntorp P. How should obesity be defined? J Intern Med 1990;227:147–9.
34. Morrow JR, Jackson AS, Bradley PW, Hartung GH. Accuracy of measured and predicted residual lung volume on body density measurement. Med Sci Sports Exerc 1986;18:647–52.
35. Tran ZV, Weltman A, Glass GV, Mood DP. The effects of exercise on blood lipids and lipoproteins: a meta-analysis of studies. Med Sci Sports Exerc 1983;15:393–402.

36. Paffenbarger RS Jr, Hyde RT, Wing AL, Lee I-M, Jung DL, Kampert JB. The association of changes in physical activity level and other lifestyle characteristics with mortality among men. N Engl J Med 1993;328:538–45.
37. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. N Engl J Med 1988;319:1173–9.
38. Hellénius ML, de Faire U, Berglund B, Hamstern A, Krakau I. Diet and exercise are equally effective in reducing risk for cardiovascular disease: results of a randomized controlled study in men with slightly to moderately raised cardiovascular risk factors. Atherosclerosis 1993;103:81–91.
39. Fortmann SP, Haskell WL, Wood PD. Effects of weight loss on clinic and ambulatory blood pressure in normotensive men. Am J Cardiol 1988;62:89–93.
40. Katzel LI, Bleecker ER, Colman EG, Rogus EM, Sorkin JD, Goldberg AP. Effects of weight loss vs aerobic exercise training on risk factors for coronary disease in healthy, obese, middle-aged and older men. JAMA 1995;274:1915–21.