

Does Body Fatness Modify the Association Between Dietary Cholesterol and Risk of Coronary Death?

Results From The Chicago Western Electric Study

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The hypothesis that body fatness modifies the relation between dietary cholesterol and 25-year coronary mortality was examined in a cohort of 1,792 middle-aged men employed by the Western Electric Company in Chicago. Relative risks of coronary death (and 95% confidence intervals) associated with a 225 mg/day greater intake of dietary cholesterol for men with a subscapular skinfold thickness ≤ 14 , 15–20, and ≥ 21 mm were 1.44 (1.10–1.90), 1.07 (0.84–1.36), and 0.95 (0.76–1.20), respectively, after adjustment for age; serum total cholesterol level; systolic blood pressure; cigarette smoking; family history of cardiovascular disease; evidence of major organ system disease at baseline; and intake of saturated fatty acids, polyunsaturated fatty acids, energy, and ethanol. Adjusted relative risks associated with a 15-mm greater subscapular skinfold thickness for men with a dietary cholesterol intake ≤ 649 , 650–799, and ≥ 800 mg/day were 1.76 (1.04–2.98), 1.64 (1.04–2.57), and 1.00 (0.69–1.55), respectively. Fatter men apparently did not benefit from a diet lower in cholesterol, while men who ate a diet high in cholesterol apparently did not benefit from leanness. These results support the hypothesis that body fatness modifies the relation between dietary cholesterol and coronary mortality, perhaps because leaner men are more responsive than fatter men to the effects of dietary cholesterol on the concentration of low density lipoprotein cholesterol. (*Arteriosclerosis and Thrombosis* 1992;12:755–761)

KEY WORDS • body fatness • dietary cholesterol • coronary heart disease • mortality

Dietary cholesterol has been associated with increased risk of coronary death among middle-aged employed men in the prospective Chicago Western Electric Study.^{1,2} A 184 mg/1,000 kcal greater intake of dietary cholesterol was associated with a 46% greater risk of 25-year coronary death after adjustment for age, other dietary lipids, and other coronary risk factors, including serum total cholesterol.² The inference that dietary cholesterol is associated with the risk of coronary disease independently of serum total cholesterol is supported by results from the Honolulu Heart Program, the Zutphen Study, and the Ireland-Boston Diet-Heart Study.^{3–7} The results of these four within-population

prospective epidemiological studies are concordant with results from cross-population ecological investigations and animal experimentation.⁷

The biological basis for an association between dietary cholesterol and coronary disease that is independent of serum total cholesterol has not been established, but at least two possible mechanisms exist. Dietary cholesterol might affect atherogenesis independently of serum total cholesterol through 1) an effect on postprandial chylomicrons and remnant particles derived from them or 2) an effect on the relative concentrations of lipoprotein cholesterol, such as the ratio of low density lipoprotein cholesterol (LDL-C) to high density lipoprotein cholesterol (HDL-C).

Dietary cholesterol is initially transported in blood by chylomicrons. After ingestion of a meal that contains cholesterol, chylomicrons interact with lipoprotein lipase to produce cholesterol-rich chylomicron remnants, which may be atherogenic.^{8–10} As evidence, elevated serum levels of chylomicron remnants after a test meal have been associated with coronary disease in case-control studies.^{11–14}

The relation between dietary cholesterol and coronary heart disease might also be mediated by effects of dietary cholesterol on plasma lipoprotein cholesterol concentrations that are not reflected in measurements of plasma total cholesterol. Feeding experiments have shown that the primary effect of increasing dietary

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cholesterol is an increase in LDL-C.¹⁵ Increases in HDL-C have also been observed, but at least part of that effect may have been due to the fat in egg yolk rather than the cholesterol itself. In any event, the evidence indicates that dietary cholesterol increases the ratio of LDL-C to HDL-C.¹⁶⁻²⁴

Consistent differences among persons in sensitivity to the hypercholesterolemic effect of dietary cholesterol have been well documented and may be due to differences in absorption of dietary cholesterol, inhibition of cholesterol synthesis, or secretion of bile acids and biliary cholesterol.²¹⁻²⁷ In relation to leaner persons, fatter persons tend to have decreased absorption of cholesterol, increased secretion of bile acids and biliary cholesterol, and hyporesponsiveness to dietary cholesterol.²⁷⁻²⁹

If dietary cholesterol were associated with the risk of coronary disease through an effect on postprandial chylomicrons, then the association should be stronger in fatter men than in leaner men. Although body fatness is positively associated with increased basal lipoprotein lipase activity, body fatness is also associated with insulin resistance, a diminished response of lipoprotein lipase activity to feeding or insulin infusion, delayed clearance of chylomicrons, and elevated concentrations of cholesterol-rich chylomicron remnants.^{10,30-33} Furthermore, maintenance of weight loss by initially fat persons has been associated with increases in both basal lipoprotein lipase activity and the response of lipoprotein lipase to insulin infusion.³⁴⁻³⁶

On the other hand, if dietary cholesterol were associated with the risk of coronary disease through an effect on the relative levels of lipoproteins, such as the ratio of LDL-C to HDL-C, then the association should be stronger in leaner men than in fatter men because fatter persons tend to be less responsive than leaner persons to the hypercholesterolemic effects of dietary cholesterol.²⁷ The purpose of the present study was to investigate whether the association between dietary cholesterol and risk of coronary death is modified by body fatness and if so, in which direction.

Methods

This analysis is based on a study of 1,792 middle-aged men employed by the Western Electric Company in Chicago in 1957. Information was obtained at the initial examination in 1958 about a large number of variables, including height, weight, subscapular skinfold thickness, blood pressure, serum total cholesterol, use of tobacco and alcohol, clinical and electrocardiographic (ECG) evidence of coronary disease, and food and beverages consumed during the preceding 4 weeks. Lipoprotein cholesterol levels were not measured. Family history of cardiovascular disease was coded positive when participants reported that a parent had died of such causes before age 60 or that a sibling had died of such causes at any age past childhood. Evidence of major organ system disease was considered present at the initial examination when any one or more of the following conditions were present: history of diabetes mellitus, urine glucose (2+), cardiomegaly or other clinically significant finding on chest x-ray films, hypertensive retinopathy, or a major ECG abnormality as defined by Minnesota codes 1.1, 1.2, 4.1, 4.2, 5.1, 5.2, 6.1, 6.2, 6.4, 7.1, 7.2, 7.4, 7.8, 8.1-8.6 (except 8.1.1 and 8.1.4), and 9.2.³⁷

Two nutritionists used standardized interviews and questionnaires based on Burke's procedure³⁸ to obtain a detailed account of the kinds and quantities of foods and beverages consumed during the preceding 28 days at both the initial examination and at the second examination 1 year later. These data were analyzed according to a food table derived from several sources³⁹⁻⁴³ to obtain each participant's usual daily intake of energy, cholesterol, saturated fatty acids, polyunsaturated fatty acids, and other nutrients.

Confounding by total energy intake can be a major problem in epidemiological studies of diet and coronary heart disease. Dietary cholesterol is strongly correlated with energy intake because it is associated with animal fat in the diet. Energy intake is associated inversely with the risk of coronary heart disease because it is related to physical activity. Thus, results that do not adjust for energy intake are largely uninterpretable. One approach to adjustment has been to express intake as milligrams of cholesterol per 1,000 kilocalories of energy. However, this approach does not necessarily avoid the problem of confounding entirely. In the present investigation, linear regression analysis was used to adjust intake of dietary cholesterol for intake of energy because this approach does eliminate the correlation with energy intake.⁴⁴

Men who continued to participate in the study were reexamined annually through 1970. Vital status was determined for all participants on the 25th anniversary of the initial examination, and cause of death was coded from the death certificate. Procedures by which participants were selected, examined, and followed up have been published.^{1,2,45} Excluded from analysis were men who, at the initial examination, had coronary disease, had missing data for key variables, reportedly consumed 50 ml or more of ethanol a day, or gave a history of physician-diagnosed diabetes mellitus.

Subscapular skinfold thickness and body mass index (body weight in kilograms divided by the square of height in meters) were both used as indexes of body fatness in the present investigation. The product-moment correlation between the two variables was 0.72. Results were similar for both, and only results based on subscapular skinfold have been presented here.

Men were classified into nine groups by dietary cholesterol and skinfold thickness, and coronary mortality rates were calculated for these nine groups. Proportional hazards regression analysis was used to calculate relative risks of coronary death in the groups after adjustment for age, percentage of calories from saturated and polyunsaturated fatty acids, energy intake, ethanol intake, serum total cholesterol, systolic blood pressure, cigarette smoking, family history of cardiovascular disease, and evidence of major organ system disease. Proportional hazards regression analysis was also employed to examine the association of dietary cholesterol and skinfold thickness, treated as continuous variables, to the risk of coronary death. The product of dietary cholesterol and skinfold thickness was included in the regression model to test whether body fatness modified the association of dietary cholesterol to the risk of coronary death.

TABLE 1. Means, Standard Deviations, and Percentile Values for Variables Measured at the Initial Examination of 1,792 Middle-Aged Employed Men: The Chicago Western Electric Study, 1958

Variable	Mean	SD	Percentile values				
			First	10th	50th	90th	99th
Dietary cholesterol (mg/day)*	754	197	403	529	728	1,007	1,331
Saturated fatty acids (% energy)*	17	3	11	14	17	20	24
Polyunsaturated fatty acids (% energy)*	4	1	2	3	4	5	7
Energy intake (kcal/day)	3,161	899	1,494	2,138	3,051	4,279	6,137
Ethanol (ml/day)†	12	12	0	0	7	31	47
Subscapular skinfold thickness (mm)	18	7	5	10	18	26	36
Body mass index (kg/m ²)	25	3	19	21	25	29	34
Serum cholesterol (mmol/l)	6.4	1.4	3.6	4.8	6.3	8.2	10.2
Systolic blood pressure (mm Hg)	134	18	103	113	131	158	187
Cigarettes (No./day)	10	11	0	0	6	20	40

*Adjusted for total energy intake.

†Men consuming >50 ml ethanol per day were excluded.

Results

Cross-Sectional Results at the Initial Examination

Table 1 shows descriptive statistics for the distributions of variables measured at the initial examination of these 1,792 employed men who were 40–56 years of age, did not have coronary heart disease, and did not have a history of diabetes. Energy-adjusted intake of dietary cholesterol had a mean and a standard deviation of 754 and 197 mg/day, respectively. The first, 50th, and 99th percentile values were 403, 728, and 1,331 mg/day, respectively. No one consumed a diet that met current recommendations for dietary cholesterol, e.g., <300 mg/day. Mean intake of saturated fatty acids was also high, at 17% of calories, and mean intake of polyunsaturated fatty acids was low, at 4% of calories. Body fatness, as indicated by subscapular skinfold thickness and body mass index, varied widely.

Table 2 shows product-moment correlations of dietary cholesterol and subscapular skinfold thickness with each other and with other variables used in these analyses. The largest correlations were between skinfold thickness and systolic blood pressure ($r=0.22$) and between dietary cholesterol and percentage of calories from saturated fatty acids ($r=0.34$). The correlation between skinfold thickness and dietary cholesterol was essentially zero.

Dietary Cholesterol and 25-Year Risk of Coronary Death

Vital status of all participants was determined on the 25th anniversary of the initial examination, at which time 297 of 1,792 men had died of coronary heart disease during 39,111 person-years of follow-up. The overall coronary mortality rate was 7.6 deaths per 1,000 person-years.

TABLE 2. Product-Moment Correlation Coefficients of Subscapular Skinfold Thickness and Dietary Cholesterol With Each Other and With Other Selected Variables Measured at the Initial Examination of 1,792 Middle-Aged Employed Men: The Chicago Western Electric Study, 1958

Variable	Subscapular skinfold	Dietary cholesterol*
Dietary cholesterol (mg/day)*	0.01†	
Saturated fatty acids (% energy)	-0.06	0.34†
Polyunsaturated fatty acids (% energy)	-0.01	0.18
Energy intake (kcal/day)	-0.11	-0.01
Ethanol (ml/day)	0.00	-0.03
Serum cholesterol (mmol/l)	0.07	0.05
Systolic blood pressure (mm Hg)	0.22	0.01
Cigarettes (No./day)	-0.14	0.00
Family history of cardiovascular disease (0=absent, 1=present)	0.03	-0.01
Evidence of major organ system disease (0=absent, 1=present)	0.06	0.02

*Adjusted for total energy intake.

†Correlation coefficients ≥ 0.05 in absolute value are associated with $p < 0.05$.

TABLE 3. Unadjusted Coronary Mortality Rates per 1,000 Person-Years and Adjusted Relative Risks for Coronary Mortality Over 25 Years of Follow-up by Dietary Cholesterol Intake and Subscapular Skinfold Thickness at the Initial Examination for 1,792 Middle-Aged Men: The Chicago Western Electric Study, 1958-1983

Dietary cholesterol (mg/day)†	Subscapular skinfold thickness (mm)*		
	3-14	15-20	21-40
Unadjusted coronary mortality rates/1,000 person-years‡			
275-649	4.1	7.2	7.6
650-799	6.5	7.2	9.3
800-1,663	8.2	9.5	8.2
Adjusted relative risks of coronary death§			
275-649	1.0	1.7	1.6
650-799	1.6	1.7	2.2
800-1,663	1.8	2.1	1.6

*Mean values for subscapular skinfold thickness in the three groups were 10.8, 17.5, and 25.5 mm.

†Mean values for energy-adjusted intake of dietary cholesterol in the three groups were 558, 723, and 967 mg/day.

‡The number of men in each group varied from 180 to 231.

§Adjusted by proportional hazards regression analysis for age, percentage of calories from saturated and polyunsaturated fatty acids, energy intake, ethanol intake, serum total cholesterol level, systolic blood pressure, cigarette smoking, family history of cardiovascular disease, and evidence of major organ system disease at baseline.

As shown in Table 3, the 1,792 men were classified into nine groups according to intake of dietary cholesterol and subscapular skinfold thickness. Inspection of the coronary mortality rates in the upper section of Table 3 indicates that the lowest coronary mortality rate occurred in the group with the lowest skinfold thickness and the lowest dietary cholesterol. A clear stepwise association between dietary cholesterol and coronary death was apparent for men with a skinfold thickness ≤ 14 mm. The unadjusted mortality rates were 4.1, 6.5, and 8.2 deaths per 1,000 person-years, respectively, for men who consumed ≤ 649 , 650-799, and ≥ 800 mg/day of dietary cholesterol. After adjustment by proportional hazards regression analysis for age, percentage of calories from saturated and polyunsaturated fatty acids, intake of energy and ethanol, serum total cholesterol level, systolic blood pressure, cigarette smoking, family history of cardiovascular disease, and evidence of major organ system disease at baseline, the corresponding relative risks shown in the lower section of Table 3 were 1.0, 1.6, and 1.8.

The association between dietary cholesterol and coronary death was less clear in the group with a subscapular skinfold thickness of 15-20 mm and was not apparent at all in the group with a subscapular skinfold thickness ≥ 21 mm.

To test the null hypothesis that the association between dietary cholesterol and coronary death was not modified by body fatness, a proportional hazards regression analysis was done in which the risk of coronary death was regressed on dietary cholesterol, subscapular skinfold thickness, and the cross-product of dietary cholesterol times subscapular skinfold thickness for all 1,792 men. Other variables in this analysis were those listed previously. The regression coefficient for the cross-product term was negative and statistically significant ($p=0.01$). The regression coefficients and SEMs for the main effects and the cross-product term were as follows: dietary cholesterol, 0.002549 ± 0.0008796 ; subscapular skinfold thickness, 0.10736 ± 0.03533 ; and the cross-product term, -0.000112 ± 0.0000441 . These results support the idea that dietary cholesterol is associated with the risk of coronary death but that the strength of the association is greater for leaner men than for fatter men.

Three other proportional hazards regression analyses were done to quantify the association between dietary cholesterol and risk of coronary death at different levels of body fatness. Other variables in these analyses were those listed above. The relative risks of coronary death associated with a 225 mg/day greater intake of dietary cholesterol were 1.44, 1.07, and 0.95 for men with a subscapular skinfold thickness ≤ 14 , 15-20, and ≥ 21 mm, respectively (see Table 4). Mean body mass index, by subscapular skinfold thickness, was 23, 25, and 28 kg/m^2 , respectively.

Subscapular Skinfold Thickness and 25-Year Risk of Coronary Death

As shown in Table 3, subscapular skinfold thickness tended to be positively associated with coronary death in men with intakes of dietary cholesterol < 800 mg/day but apparently was not associated with coronary death in men with greater intakes. To quantify this association more precisely, three more proportional hazards regression analyses were done as described above. These analyses showed that the adjusted relative risks of coronary death (and 95% confidence intervals) associated with a 15-mm greater subscapular skinfold thickness for men whose intake of dietary cholesterol was ≤ 649 ,

TABLE 4. Adjusted Relative Risks for Coronary Mortality Associated With a 225 mg/Day Greater Intake of Dietary Cholesterol According to Subscapular Skinfold Thickness for 1,792 Middle-Aged Employed Men: The Chicago Western Electric Study, 1958-1983

Subscapular skinfold thickness (mm)	Mean body mass index (kg/m^2)	No. of men	No. of coronary deaths	Relative risk*	95% Confidence interval
3-14	23	554	77	1.44	1.10-1.90
15-20	25	655	114	1.07	0.84-1.36
21-40	28	583	106	0.95	0.76-1.20

*Coefficients (± 1 SEM) for the proportional hazards regression of coronary mortality on dietary cholesterol adjusted for age, percentage of calories from saturated and polyunsaturated fatty acids, energy intake, ethanol intake, serum total cholesterol level, systolic blood pressure, cigarette smoking, family history of cardiovascular disease, and evidence of major organ system disease at baseline were 0.001629 ± 0.000619 , 0.000305 ± 0.000548 , and -0.000205 ± 0.000521 , respectively, for the groups shown above.

TABLE 5. Mean Height and Weight at the Initial Examination (1958), Mean Weight at the Fifth and 10th Examinations (1963 and 1968), and Product-Moment Correlations Between 1958 and 1968 Weights by Subscapular Skinfold Thickness and Intake of Dietary Cholesterol at the Initial Examination for 1,241 Men With Data at All Three Examinations: The Chicago Western Electric Study

Dietary cholesterol (mg/day)	Subscapular skinfold (mm)	No. of men	Mean height (cm)	Mean body weight (kg)			Correlation between 1958, 1968 weights
				1958	1963	1968	
<649	≤14	129	172	69.5	71.3	74.7	0.83
	15-20	145	172	77.6	78.4	81.1	0.78
	≥21	132	172	85.9	85.9	88.5	0.89
650-799	≤14	131	173	71.6	73.3	77.0	0.83
	15-20	151	172	77.5	78.7	82.0	0.85
	≥21	143	172	83.9	84.1	86.7	0.86
≥800	≤14	125	173	70.4	71.5	75.2	0.86
	15-20	162	172	77.9	78.8	81.7	0.86
	≥21	123	172	86.3	86.1	87.9	0.82
All	≤14	385	172	70.5	72.0	75.6	0.84
	15-20	458	172	77.7	78.6	81.6	0.83
	≥21	398	172	85.3	85.3	87.6	0.86

650-799, and ≥800 mg/day were 1.76 (1.04-2.98), 1.64 (1.04-2.57), and 1.00 (0.64-1.55), respectively.

Change Over Time in Dietary Cholesterol and Body Weight

Within-person change in the intake of dietary cholesterol after the initial examination would result in misclassification of participants according to usual intake. If such misclassification were unrelated to body fatness, it would tend to decrease the apparent strength of the association between intake of dietary cholesterol and risk of coronary death equally for leaner and fatter men⁴⁶ and would not produce the patterns seen in Tables 3 and 4. However, if misclassification were greater for fatter men than leaner men, the association would be attenuated more for fatter men than for leaner men, thereby giving the appearance that body fatness was modifying the association between dietary cholesterol and the risk of coronary death. This might have occurred if fatter men were more likely than leaner men to decrease their intake of dietary cholesterol after the initial examination, for instance, as a result of a weight-reduction diet.

This possibility was investigated by analyzing data on the intake of dietary cholesterol that were obtained at both the first and second examinations for 1,744 of these 1,792 men. Overall, the mean intake of dietary cholesterol declined by 24 mg/day from 1958 to 1959. When stratified by subscapular skinfold thickness, mean differences were slightly larger for leaner men than for fatter men: -28, -24, and -20 mg/day for men with skinfolds ≤14, 15-20, and ≥21 mm, respectively. The product-moment correlations between intake of dietary cholesterol in 1958 and 1959 for these three groups were 0.63, 0.50, and 0.63, respectively. The overall correlation was 0.58. These results do not support the hypothesis that fatter men were more likely than leaner men to change their intake of dietary cholesterol between 1958 and 1959.

Data on changes in diet over a longer period were not obtained for all participants. However, body weight was measured at each annual examination, and changes in body weight were used as a surrogate for dieting to lose weight. Table 5 shows mean height and body weight according to subscapular skinfold thickness and intake of dietary cholesterol for 1,241 men who had these data for the first, fifth, and 10th examinations (1958, 1963, and 1968, respectively). Mean height was virtually identical in all nine groups. Mean body weight at the initial examination was 70.5, 77.7, and 85.3 kg for men with a subscapular skinfold thickness ≤14, 15-20, and ≥21 mm, respectively.

This pattern of association between body weight and subscapular skinfold thickness was repeated within each stratum of dietary cholesterol intake at the initial examination and at subsequent examinations in 1963 and 1968. Mean body weight was greater in 1968 than in 1958 for each group. Increases were greater for men with initially thinner than thicker skinfolds. Product-moment correlations between body weight in 1958 and 1968 varied among the nine groups from 0.78 to 0.89. The coefficients for men with skinfolds ≤14, 15-20, and ≥21 mm were 0.84, 0.83, and 0.86, respectively. These results indicate that body weight was at least as stable over time for men with initially thicker skinfolds as for men with initially thinner skinfolds.

Discussion

Prior investigations have shown that dietary cholesterol was associated with risk of coronary death in the Chicago Western Electric cohort and that this association persisted after adjustment for serum total cholesterol as well as other coronary risk factors.^{1,2} Since then, another study found that leaner men tended to be more responsive than fatter men to the hypercholesterolemic effects of dietary cholesterol.^{26,27} That finding led to the question of whether body fatness modifies the association between dietary cholesterol and coronary death. Results of the present investigation indicate that the

association between dietary cholesterol and coronary death was stronger for men with thinner than thicker subscapular skinfold thickness. These results support the inference that the association between dietary cholesterol and coronary death is mediated through effects on serum lipoprotein cholesterol, which are inadequately reflected in serum total cholesterol, e.g., increases in the ratio of LDL-C to HDL-C.¹⁵⁻²⁴ This hypothesis could be examined more directly by adjustment for concentrations of LDL-C and HDL-C, but these data are not available for the Western Electric Study initiated in 1957.

The present results do not support the idea that the association of dietary cholesterol to coronary death is mediated by effects on postprandial chylomicron remnants. If that were the mechanism, the association between dietary cholesterol and coronary death should have been stronger in fatter men than leaner men. However, these results do not rule out such an effect. In this observational study, effects of dietary cholesterol on atherogenesis via plasma lipoprotein cholesterol might have obscured effects on atherosclerosis via chylomicron remnants.

The observed patterns of associations might have occurred if fatter men were more likely than leaner men to decrease their intake of dietary cholesterol during the follow-up period. This hypothesis has a certain plausibility because reduction in dietary cholesterol would usually occur in weight-reduction diets. However, no evidence of this occurrence was seen when changes in dietary cholesterol intake from 1958 to 1959 and changes in body weight from 1958 to 1968 were examined.

This result should be viewed in the light of at least two caveats. First, intake of dietary cholesterol in 1958 and 1959 by men in the Western Electric Study was very high by today's standards. The mean was 753 mg/day. This may not have been unusual for middle-aged US men at that time. Mean intake of dietary cholesterol for 437 men in the Framingham Study, determined by a similar method at about the same time, was 704 mg/day.⁴⁷ Because no participant in the present study was habitually consuming a low-cholesterol diet, the results bear directly only on intakes in the range of 400-500 mg/day and higher. Second, subscapular skinfold thickness is an imperfect measurement of body fatness and does not measure the distribution of body fat. Body fat distribution, rather than body fatness, may have modified the association between dietary cholesterol and coronary death. Further research is needed with more precise and detailed measurements of body fatness, body fat distribution, plasma lipids and lipoproteins, and diet.

The small negative association between body fatness and energy intake seen in this cohort ($r = -0.11$) persisted after adjustment for cigarette smoking, has been seen in other population studies, and probably represents a negative association between body fatness and physical activity.^{48,49} If this interpretation is correct, then fatter men in this cohort may have had less efficient postprandial lipolysis than leaner men due in part to lower physical activity.⁵⁰

A large body of evidence links body fatness, particularly abdominal fatness, to coronary disease and other adverse effects on health.⁵¹ In the present study, sub-

scapular skinfold thickness was positively associated with the risk of coronary death, and the association persisted after adjustment for potentially confounding factors. Similar results (not shown here) were obtained when body mass index was used in place of the skinfold thickness. This association has been observed previously in the Western Electric Study, but it was not statistically significant until a larger number of deaths had accrued with 25 years of follow-up.

An association between skinfold thickness and coronary death was not observed among men who consumed ≥ 800 mg/day of dietary cholesterol. Both dietary cholesterol and body fatness adversely affect the ratio of LDL-C to HDL-C, and it seems possible that this common effect may be involved in their apparent interaction.

In summary, these results support the idea that dietary cholesterol and body fatness are associated with increased risk of coronary heart disease. Persons with lower intakes of dietary cholesterol and lower body fatness had the lowest 25-year risk of coronary death. The results indicate that these two factors are not independent of one another. Fatter men apparently did not benefit from a diet lower in cholesterol, while men who consumed a diet very high in cholesterol apparently did not benefit from leanness. These results support public health recommendations to maintain ideal body weight and to avoid a high intake of dietary cholesterol to decrease the risk of coronary heart disease.⁴⁸

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