

Dietary Fiber and Risk of Coronary Heart Disease

A Pooled Analysis of Cohort Studies

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Background: Few epidemiologic studies of dietary fiber intake and risk of coronary heart disease have compared fiber types (cereal, fruit, and vegetable) or included sex-specific results. The purpose of this study was to conduct a pooled analysis of dietary fiber and its subtypes and risk of coronary heart disease.

Methods: We analyzed the original data from 10 prospective cohort studies from the United States and Europe to estimate the association between dietary fiber intake and the risk of coronary heart disease.

Results: Over 6 to 10 years of follow-up, 5249 incident total coronary cases and 2011 coronary deaths occurred among 91 058 men and 245 186 women. After adjustment for demographics, body mass index, and lifestyle factors, each 10-g/d increment of energy-adjusted and measure-

ment error-corrected total dietary fiber was associated with a 14% (relative risk [RR], 0.86; 95% confidence interval [CI], 0.78-0.96) decrease in risk of all coronary events and a 27% (RR, 0.73; 95% CI, 0.61-0.87) decrease in risk of coronary death. For cereal, fruit, and vegetable fiber intake (not error corrected), RRs corresponding to 10-g/d increments were 0.90 (95% CI, 0.77-1.07), 0.84 (95% CI, 0.70-0.99), and 1.00 (95% CI, 0.88-1.13), respectively, for all coronary events and 0.75 (95% CI, 0.63-0.91), 0.70 (95% CI, 0.55-0.89), and 1.00 (95% CI, 0.82-1.23), respectively, for deaths. Results were similar for men and women.

Conclusion: Consumption of dietary fiber from cereals and fruits is inversely associated with risk of coronary heart disease.

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DIETARY FIBER MAY REDUCE the risk of coronary heart disease (CHD) through a variety of mechanisms, such as improving blood lipid profiles,¹⁻³ lowering blood pressure,^{4,5} and improving insulin sensitivity^{6,7} and fibrinolytic activity.⁸ Dietary fiber has been found to be inversely associated with risk factors for CHD in observational studies.⁹⁻¹²

The association between dietary fiber and CHD incidence has been examined in at least 10 prospective cohort studies.^{9,13-21} All but one¹⁸ of these studies reported an inverse association. Due to differences in methods and analytical techniques, the magnitude of this association for total fiber intake and for specific types of fiber (cereal, fruit, vegetable, soluble and insoluble) remains unclear. Furthermore, only 4 studies^{13,14,19,20} have reported findings for women separately from men. Negative publication bias and residual confounding by other lifestyle factors remain possibilities. We have therefore conducted a systematic analysis of 10

cohort studies from the United States and Europe included in the Pooling Project of Cohort Studies on Diet and Coronary Disease.

METHODS

The following criteria for the inclusion of studies in this pooled analysis were applied: a published prospective study with at least 150 incident coronary cases, assessment of usual dietary intake, and a validation study of the diet assessment method or a closely related instrument. Through literature searches and inquiring with experts in the field, 14 studies were identified that met these criteria, and investigators of 11 agreed to include their data in the project. One study was excluded from this analysis because it did not have data on dietary fiber intake. Investigators of 3 eligible studies, all from the United States, did not agree to participate. The remaining studies are described in **Table 1**. The follow-up experience of the Nurses' Health Study (NHS)¹⁴ was divided into 2 periods for analysis to take advantage of the repeated assessments of dietary intake and the long follow-up. The 1980-1986 follow-up period is referred to as Nurses' Health Study A (NHSa) and the 1986-1996 follow-up period of women who remained free of

Author affiliations are listed at the end of this article. The authors have no relevant financial interest in this article.

Table 1. Characteristics of the Cohort Studies Included in the Pooling Project of Diet and Coronary Disease

Study	Baseline Cohort*	Date of Inception	Person-years of Follow-up	Age at Baseline, Median (Range), y	No. of CHD Cases		Dietary Questionnaire	Median Energy-Adjusted Fiber Intake, g/d				
					Total Events	Deaths		Total	Cereal	Fruit	Vegetable	
AHS ²²												
Men	9212	1977	52 835	54 (35-98)	148	49	FFQ	29.7
Women	13 430	1977	78 014	56 (35-99)	75	41	FFQ	19.9
ARIC ²⁶												
Men	5240	1987	45 861	54 (44-66)	269	51	FFQ	17.1	4.2	3.6	4.5	
Women	6481	1987	58 199	53 (44-66)	123	17	FFQ	15.2	3.2	4.2	4.2	
ATBC ¹⁶ (men only)	21 141	1985	121 813	56 (49-70)	1339	534	Diet history	18.9	12.0	1.8	4.0	
FMC ²⁴												
Men	2712	1966	24 599	47 (35-85)	322	147	Diet history	21.9	15.0	1.3	4.6	
Women	2481	1966	23 643	49 (35-86)	162	48	Diet history	17.7	10.8	2.1	3.8	
GPS ²³												
Men	1658	1974	14 365	50 (35-80)	102	38	Diet history	17.1	
Women	1666	1974	14 605	50 (35-80)	34	14	Diet history	14.3	
HPFS ¹⁵ (men only)	41 574	1986	383 206	52 (39-77)	1273	421	FFQ	20.6	5.2	3.8	6.5	
IWHS ²⁷ (women only)	30 180	1986	294 620	61 (52-71)	...	294	FFQ	17.8	4.3	4.1	7.0	
NHSa ¹⁴ (women only)	81 415	1980	513 915	46 (35-66)	397	97	FFQ	13.0	2.2	3.6	4.3	
NHSb ¹⁴ (women only)	61 706	1986	607 049	52 (39-66)	696	208	FFQ	16.9	3.7	3.6	5.8	
VIP ²⁵												
Men	9521	1992	39 230	50 (39-70)	134	38	FFQ	20.3	13.6	2.3	3.6	
Women	10 555	1992	43 872	50 (39-70)	23	4	FFQ	19.2	10.8	3.8	3.8	
WHS ¹³ (women only)	37 272	1993	190 755	52 (38-89)	152	10	FFQ	16.9	3.8	3.3	5.7	
Total	336 244	...	2 506 581	...	5249	2011

Abbreviations: AHS, Adventists Health Study; ARIC, Atherosclerosis Risk in Communities Study; ATBC, Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study; FFQ, food frequency questionnaire; FMC, Finnish Mobile Clinic Health Examination Survey; GPS, Glostrup Population Study; HPFS, Health Professionals Follow-up Study; IWHS, Iowa Women's Health Study; NHSa, Nurses' Health Study 1980-1986; NHSb, Nurses' Health Study 1986-1996; VIP, Vasterbotten Intervention Program; WHS, Women's Health Study.

*Sample size after excluding baseline cardiovascular disease, cancer, and very high or low reported caloric intake.

CHD until 1986 is referred to as Nurses' Health Study B (NHSb). Following the underlying theory of survival data, blocks of person-time in different periods are statistically independent, even if derived from the same people.²⁸ Therefore, pooling the estimates from these 2 periods is equivalent to using a single period but takes advantage of the enhanced exposure assessment in 1986 compared with 1980.

DIETARY ASSESSMENT

Diet was measured at baseline in each study using a food frequency questionnaire or diet history instrument. For the Adventist Health Study (AHS),²² only crude fiber was available for analysis. Therefore, to approximate the distribution of total dietary fiber in this cohort, we multiplied crude fiber by 3.5—the ratio of total to crude fiber from other studies. In addition to total dietary fiber, we examined fiber intake from 3 different food group sources, including cereals (grains), fruits, and vegetables, as well as insoluble (hemicellulose, cellulose, and lignin) and soluble (pectins, gums, and mucilages) fiber. Fiber from cereals, fruits, and vegetables was available for all studies with the exception of the AHS²² and the Glostrup Population Study (GPS).²³ A wide variety of foods contributed to each fiber type, with the relative contribution from certain foods varying among studies. Starchy vegetables, such as corn and peas, contributed substantially to vegetable fiber in all studies. Only the Finnish Mobile Clinic Health Examination Survey (FMC)²⁴ and the Vasterbotten Intervention Program (VIP)²⁵ included potato fiber with their vegetable fiber, and potato fiber was the most common form of vegetable fiber in these 2 studies. Only 6 studies (Atherosclerosis Risk in Communities Study [ARIC],²⁶ Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study [ATBC],¹⁶ Health Professionals Follow-up Study [HPFS],¹⁵ Iowa Women's Health Study

[IWHS],²⁷ NHSb,¹⁴ and Women's Health Study [WHS]¹³) had estimates of insoluble and soluble fiber, but there is no standard method for estimating these fiber types based on food tables, so the results should be considered exploratory.

CASE ASCERTAINMENT

Standardized criteria were used to ascertain cases of fatal and nonfatal acute myocardial infarction in all studies.^{13-16,22-27} Because the IWHS²⁷ had only self-reported data on incident CHD, we used only fatal coronary cases from this study. We conducted separate analyses for all coronary events (fatal and nonfatal) and for coronary deaths.

STATISTICAL ANALYSIS

We excluded approximately 1% of participants from each study if they reported energy intakes greater or less than 3 SDs from the study-specific, log-transformed mean energy intake of the baseline population. Because the presence of clinical disease itself may cause dietary changes, we also excluded participants who reported a history of cardiovascular disease, diabetes, or cancer (except nonmelanoma skin cancer) at baseline. Four studies (ARIC, FMC, GPS, and IWHS) with follow-up periods longer than 10 years were truncated to reduce heterogeneity in study duration. Within each cohort, relative risks (RRs) (incidence rate ratios) per fiber increment were computed using proportional hazards regression models with the PROC PHREG program of SAS statistical software, version 8.²⁹ The RRs were adjusted for relevant baseline demographic, lifestyle, and dietary factors. Categories of covariates were standardized across studies with a few exceptions, as follows. For disease history, information across studies included any or all of the following: self-reported dis-

ease, medication use, or biologic measures (eg, blood pressure and serum cholesterol level). For physical activity, information across studies ranged from simple categories of low, moderate, and high leisure-time activity to a continuous metabolic index of total physical activity, which was grouped into quintiles. Physical activity was unavailable for one study. Alcohol intake was unavailable for 2 studies. Three regression models were computed, as follows. Model 1 included age (in years), energy intake (in kilocalories per day), smoking status (never, past, or current smoker and dose [1-4, 5-14, 15-24, and ≥ 25 cigarettes per day]), body mass index (a measure of weight in kilograms divided by the square of height in meters, < 23 , 23 - < 25 , 25 - < 27.5 , 27.5 - < 30 , or ≥ 30), physical activity (levels 1-5), education ($<$ high school, high school, $>$ high school), alcohol intake (0, < 5 , 5 - < 10 , 10 - < 15 , 15 - < 30 , 30 - < 50 , or ≥ 50 mL/d), multiple vitamin use (no, yes), hypercholesterolemia (no, yes), and hypertension (no, yes). Model 2 included covariates in model 1 and also energy-adjusted quintiles of dietary saturated fat, polyunsaturated fat, and cholesterol. Model 3 includes covariates in model 2 and also energy-adjusted quintiles of dietary and supplement sources of folic acid and vitamin E.

Two-sided 95% confidence intervals (CIs) were calculated. We used the random-effects model developed by DerSimonian and Laird³⁰ to combine the log_e RRs; the study-specific RRs were weighted by the inverse of the sum of their variances. We tested for heterogeneity among studies using the estimated between-studies variance component Q statistic.³⁰

Before performing the regression analysis, dietary fiber and all dietary covariates were adjusted within each study for energy intake.³¹ We analyzed the energy-adjusted dietary fiber as a continuous variable (increment of 10 g/d). We also examined quintiles and deciles, based on the cohort-specific distributions, to determine if associations were linear and consistent with the analyses of continuous fiber. Using absolute fiber intake cut points, we also examined the risk of CHD throughout the full range of fiber intake available for all of the studies. To calculate the *P* value for the test for trend across quintiles, participants were assigned the median value of their quintile of intake, and this variable was entered as a continuous term in the Cox regression models. Results of dietary fiber as a continuous variable were corrected for bias due to dietary measurement error, in fiber only, using the regression calibration method.^{32,33} This correction could not be performed for fiber intake from specific food sources, because few of the validation studies included these sources of fiber. Measurement error correction was not performed on other covariates and dietary factors in the models.

We evaluated whether the following variables modified the association between fiber intake and risk of CHD: sex, age (10-year categories), follow-up time, body mass index (< 25 , 25 - 30 , > 30), cigarette smoking (never smoker vs former or current smoker), saturated fat intake (percentage of energy intake quintiles), and history of hypertension and hypercholesterolemia (positive vs negative). For each factor of interest, a cross-product term of the score for the level of each factor and intake of fiber expressed as a continuous variable was included in separate multivariate models. The pooled *P* value for the test for effect modification was obtained using squared Wald statistics by pooling the study-specific interaction coefficients and dividing by the square of the SE of the pooled interaction term and referring the resulting statistics to a χ^2 distribution with 1 *df*. The lack of any statistically significant effect modification by age or follow-up time supports the assumption of proportional hazards.

RESULTS

A total of 91 058 men and 245 186 women, contributing 2 506 581 person-years of follow-up, were included in

these analyses. The total number of events was 5249, including 2011 fatal cases (Table 1). The median fiber intakes for each cohort are given in Table 1.

The RRs of all major coronary events (fatal and nonfatal) and coronary deaths for each 10-g/d increment of energy-adjusted total dietary fiber intake are given in **Table 2**. In analyses adjusted for all demographic and nondietary lifestyle factors, for each 10-g/d increment in dietary fiber, we observed pooled reductions in risk of 12% for all coronary events and 19% for coronary deaths. There was little attenuation of these pooled estimates with further adjustment for dietary intake of fatty acids, cholesterol (model 2), and dietary and supplemental folic acid and vitamin E (model 3). These associations were similar for men (RR of coronary death, 0.82; 95% CI, 0.72-0.94) and women (RR of coronary death, 0.80; 95% CI, 0.66-0.96). Further adjustment for alpha and beta carotene, n-3 marine fatty acids, and α -linolenic acid did not materially change the results (data not shown). Analysis of dietary fiber quintiles revealed similar findings (RR of top quintile compared with the bottom quintile, 0.90 for all events; *P* $<$.09, test for trend; RR for coronary deaths, 0.70; *P* $<$.001, test for trend). The results for model 3 were corrected or bias due to measurement error in fiber only; the RRs associated with a 10-g/d increment were 0.86 (95% CI, 0.78-0.96) for all coronary events and 0.73 (95% CI, 0.61-0.87) for coronary deaths.

The results for types of fiber are summarized in **Table 3**, with adjustment for all demographic, lifestyle, and dietary factors as we did for model 3. We observed pooled reductions in risk of all coronary events of 10% for each 10-g/d increment of cereal and 16% per 10-g/d increment of fruit fiber, although the finding for cereal fiber had a CI that included 1.00. Associations were stronger for coronary deaths than for all events, with reductions in risk of 25% for cereal fiber and 30% for fruit fiber for each 10-g/d increment. In contrast, vegetable fiber was not associated with CHD incidence or mortality. Heterogeneity (*P* = .025) was observed in RRs among the 8 studies included in the analysis of cereal fiber and all coronary events. This heterogeneity seemed to be explained by a sex difference due to positive associations in 3 cohorts of women—ARIC, NHSa, and VIP. No significant heterogeneity was observed in any of the other analyses.

To determine if the associations observed for cereal and fruit fiber were independent, we included these fiber types in the same regression model. The results of these analyses were similar for all events (fruit fiber: RR, 0.81; 95% CI, 0.69-0.95; cereal fiber: RR, 0.89; 95% CI, 0.76-1.05) and deaths (fruit fiber: RR, 0.65; 95% CI, 0.49-0.86; cereal fiber: RR, 0.71; 95% CI, 0.59-0.87), suggesting that the effects of cereal and fruit fiber were independent of each other. We also examined the associations between soluble and insoluble fiber and CHD risk. Intake of both types of fiber was inversely associated with risk of all coronary events and of coronary deaths. No heterogeneity was observed among the RRs. The associations were stronger for soluble fiber (all events: RR per 10-g/d increment, 0.72; 95% CI, 0.55-0.93; deaths: RR, 0.46; 95% CI, 0.28-0.74) than for insoluble fiber (all events: RR, 0.90; 95% CI, 0.83-0.97; deaths: RR, 0.80; 95% CI,

Table 2. Study-Specific and Pooled Multivariate Relative Risks (95% Confidence Intervals) of Coronary Heart Disease per 10-g/d Increments of Total Dietary Fiber*

Variable	All Events			Deaths		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
AHS						
Men	0.84	0.89	1.04 (0.43-2.51)	0.56	0.22	0.32 (0.05-2.18)
Women	1.39	1.53	1.41 (0.46-4.36)	1.55	1.45	1.85 (0.28-12.1)
ARIC						
Men	0.94	1.00	1.02 (0.74-1.40)	0.70	0.71	0.54 (0.19-1.57)
Women	0.66	0.67	0.73 (0.39-1.34)	0.70
ATBC (men only)	0.96	0.95	0.96 (0.85-1.09)	0.84	0.81	0.84 (0.69-1.03)
FMC						
Men	0.89	0.89	0.97 (0.74-1.28)	0.83	0.73	0.74 (0.49-1.12)
Women	0.61	0.58	0.74 (0.42-1.31)	0.59	0.44	0.33 (0.09-1.22)
GPS						
Men	0.87	0.93	0.93 (0.54-1.59)	1.00	1.53	1.66 (0.66-4.20)
Women	1.02	1.44	1.00 (0.34-2.98)	0.94	0.81	0.37 (0.04-3.91)
HPFS (men only)	0.85	0.84	0.87 (0.78-0.97)	0.77	0.82	0.84 (0.69-1.03)
IWHS (women only)	0.78	0.78	0.80 (0.61-1.04)
NHSa (women only)	0.95	0.94	0.96 (0.72-1.27)	1.05	1.14	0.96 (0.55-1.67)
NHSb (women only)	0.84	0.82	0.87 (0.72-1.05)	0.79	0.81	0.75 (0.53-1.07)
VIP						
Men	0.95	0.92	0.91 (0.63-1.31)	0.69	0.55	0.53 (0.24-1.15)
Women	1.11	2.02	3.18 (0.98-10.37)	0.65
WHS (women only)	0.75	0.74	0.77 (0.50-1.17)	1.46	4.10	1.77 (0.24-12.90)
Pooled	0.88 (0.84-0.93)	0.88 (0.83-0.94)	0.91 (0.85-0.97)	0.81 (0.74-0.88)	0.81 (0.72-0.92)	0.81 (0.73-0.91)
<i>P</i> value for relative risk†	<.001	<.001	.005	<.001	.001	<.001
<i>P</i> value for heterogeneity‡	.42	.39	.82	.95	.29	.73
After measurement error correction			0.86 (0.78-0.96)			0.73 (0.61-0.87)
<i>P</i> value for relative risk			.005			<.001
<i>P</i> value for heterogeneity			.93			.80

Abbreviations: AHS, Adventists Health Study; ARIC, Atherosclerosis Risk in Communities Study; ATBC, Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study; FMC, Finnish Mobile Clinic Health Examination Survey; GPS, Glostrup Population Study; HPFS, Health Professionals Follow-up Study; IWHS, Iowa Women's Health Study; NHSa, Nurses' Health Study 1980-1986; NHSb, Nurses' Health Study 1986-1996; VIP, Vasterbotten Intervention Program; WHS, Women's Health Study; ellipses, data not applicable.

*Model 1 includes age (in years), energy intake (in kilocalories per day), smoking status (never, past, current smoker and dose [1-4, 5-14, 15-24, and ≥25 cigarettes per day]), body mass index ([calculated as weight in kilograms divided by the square of height in meters] <23, 23-<25, 25-<27.5, 27.5-<30, ≥30), physical activity (levels 1-5), education (<high school, high school, >high school), alcohol intake (0, <5, 5-<10, 10-<15, 15-<30, 30-<50, ≥50 mL/d), multiple vitamin use (no, yes), hypercholesterolemia, and hypertension. Model 2 includes covariates in model 1 and also energy-adjusted quintiles of dietary saturated fat, polyunsaturated fat, and cholesterol. Confidence intervals are not shown for models 1 and 2 for simplicity of presentation. Model 3 includes covariates in model 2 and also energy-adjusted quintiles of dietary and supplement sources of folic acid and vitamin E. Both ARIC women and VIP women had insufficient cases for analysis of deaths.

†*P* value, test for no association.

‡*P* value, test for between-studies heterogeneity.

0.69-0.92). The results were similar, although with wider CIs, when soluble and insoluble fiber were included in the same model.

The results for measurement error-corrected total dietary fiber were generally consistent when stratified by age, follow-up time (including exclusion of the first 2 years or stratification by first and second 5-year periods), overweight status, smoking, and intake of saturated fat (data not shown). There were no significant interactions between dietary fiber and any of these variables.

Finally, the results according to absolute cut points of energy-adjusted dietary fiber are presented in the **Figure** for coronary mortality. The reference category was 18 to 21 g/d and adjustment is the same as in Table 3.

COMMENT

The results of the present study suggest that dietary fiber is inversely associated with risk of CHD in both men and women. The associations were stronger for

coronary mortality (27% reduction in risk for each 10-g/d increment in total dietary fiber) than for all events (14% reduction in risk). Although cereal and fruit fiber had strong inverse associations with CHD risk, no such associations were observed for vegetable fiber. These associations seemed to be independent of other dietary factors, sex, age, baseline body mass index, smoking, history of hypertension, diabetes, and hypercholesterolemia.

The RRs were generally consistent across the studies. The only observation of heterogeneity in RRs was for the analysis of cereal fiber and total coronary events, in which 3 cohorts of women (AHS, NHSa, and VIP) had RRs greater than 1.00. In NHSa, an older version of the food frequency questionnaire was used, with limited information available for quantifying total fiber and especially cereal fiber. The relative contribution of refined grains to cereal fiber in NHSa seems to have been exaggerated, whereas the opposite seems to have occurred for whole grains. Because whole grains, but not refined grains,

Table 3. Study-Specific and Pooled Multivariate Relative Risks (95% Confidence Intervals) of Coronary Heart Disease per 10-g/d Increments of Cereal Fiber, Fruit Fiber, and Vegetable Fiber*

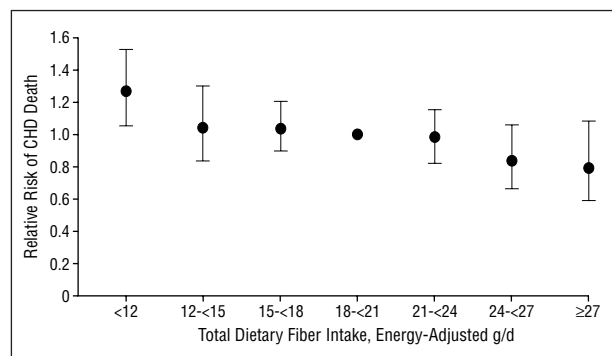
	All Events			Deaths		
	Cereal	Fruit	Vegetable	Cereal	Fruit	Vegetable
ARIC						
Men	0.68	0.90	1.17	0.90	0.20	1.79
Women	1.95	0.54	1.62
ATBC (men only)	1.02	0.72	0.64	0.94	0.41	0.57
FMC						
Men	0.95	1.27	1.00	0.82	1.10	0.34
Women	0.75	1.49	0.47	0.44	0.25	1.97
HPFS (men only)	0.77	0.76	0.98	0.68	0.74	1.13
IWHS (women only)	0.49	0.70	0.98
NHSa (women only)	1.69	0.74	1.07	1.34	0.95	1.10
NHSb (women only)	0.66	1.02	1.16	0.61	0.92	0.98
VIP						
Men	0.83	1.30	1.24	0.54	0.12	3.11
Women	3.52	8.23	0.09
WHS (women only)	0.93	0.45	0.90	0.03	3.93	0.44
Pooled	0.90 (0.77-1.07)	0.84 (0.70-0.99)	1.00 (0.88-1.13)	0.75 (0.63-0.91)	0.70 (0.55-0.89)	1.00 (0.82-1.23)
<i>P</i> value for relative risk†	.23	.04	.97	.003	.004	.97
<i>P</i> value for heterogeneity‡	.02	.24	.46	.30	.39	.46

Abbreviations: ARIC, Atherosclerosis Risk in Communities Study; ATBC, Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study; FMC, Finnish Mobile Clinic Health Examination Survey; HPFS, Health Professionals Follow-up Study; IWHS, Iowa Women's Health Study; NHSa, Nurses' Health Study 1980-1986; NHSb, Nurses Health Study 1986-1996; VIP, Vasterbotten Intervention Program; WHS, Women's Health Study; ellipses, data not applicable.

*Model includes age (5-year age groups), energy intake (in kilocalories per day), smoking status (never, past, or current smoker and dose [1-4, 5-14, 15-24, and ≥ 25 cigarettes per day]), body mass index ([calculated as weight in kilograms divided by the square of height in meters] <23, 23-<25, 25-<27.5, 27.5-<30, ≥ 30), physical activity (levels 1-5), education (<high school, high school, >high school), alcohol intake (0, <5, 5-<10, 10-<15, 15-<30, 30-<50, ≥ 50 mL/d), multiple vitamin use (no, yes), history of hypercholesterolemia and hypertension, and energy-adjusted quintiles of dietary saturated fat, polyunsaturated fat, and cholesterol, and quintiles of dietary and supplement sources of folic acid and vitamin E. Both ARIC women and VIP women had insufficient cases for analysis of deaths.

†*P* value, test for no association.

‡*P* value, test for between-studies heterogeneity.



Relative risk of death from coronary heart disease (CHD) by category of total dietary fiber intake. The relative risks are adjusted for the same variables as in Table 3. Error bars indicate 95% confidence intervals.

have been shown to reduce risk of CHD,^{34,35} such measurement error in cereal fiber intake could explain the unexpected NHSa findings. Indeed, the previously published findings of NHS included analysis of the dietary fiber intake average over the repeated food frequency questionnaires (1984, 1986, and 1990) and the results revealed a strong inverse association—an RR of 0.63 for each 5-g/d increment in cereal fiber.¹⁴ The findings for the women of the AHS and VIP were not consistent with those for the men in those studies. Furthermore, because the CIs were very wide for these estimates, we are unable to draw any meaningful inferences from them.

Four of the studies' findings on fiber and CHD included in the pooling project had been previously published.¹³⁻¹⁶ In the NHS and the HPFS, the strongest inverse associations were observed for cereal fiber, with weaker associations for fruit and vegetable fiber. In the ATBC, inverse associations were generally observed for all types of fiber. In the WHS, Liu et al¹³ observed the strongest associations for fruit fiber intake and risk of total cardiovascular disease, whereas no associations were observed with incidence of myocardial infarction. Six published studies on fiber and CHD were not included in the pooling project because they did not meet the requirements of at least 150 incident cases^{17,18,20,21} or use of a validated dietary assessment⁹ or we were previously unaware of their existence.¹⁹ Of these, 3 reported statistically significant inverse associations between dietary fiber intake and CHD,^{17,19,21} 2 reported inverse associations that were not statistically significant,^{9,20} and 1 study reported a nonsignificant positive association.¹⁸ Although Mann et al¹⁸ observed a nonsignificant increased risk of CHD with increasing total fiber consumption, this finding may have been spurious due to the small number of events (38 deaths).

There has been little support for an inverse association between vegetable fiber intake and risk of CHD. One possible explanation for this finding is the nutrient-poor high glycemic load nature of common starchy and heavily processed vegetables, such as corn and peas. Two

studies (VIP and FMC) also included potato in their vegetable fiber analysis. Dietary glycemic load has been shown to substantially increase risk of CHD and type 2 diabetes mellitus.³⁶⁻³⁸ Therefore, any beneficial effects of vegetable fiber may be countered by some adverse effects of starchy vegetables. More attention needs to be given, both in research and public health recommendations, to the types of foods being studied and recommended. As such, one limitation of the present study is the absence of food data to complement these analyses on fiber. Although such pooled analyses of foods and dietary patterns in relation to CHD are beyond the scope of the current investigation, they should be included in future efforts.

Of additional interest is whether protection from CHD may come from both soluble and insoluble fiber. Previous studies¹³⁻¹⁶ have supported this possibility, with no consistent advantage of either class of fiber. Although we observed inverse associations for both types of fiber in the present analyses, the RRs were stronger for soluble fiber, reaching 0.46 for risk of coronary mortality per each 10-g/d increment. These results must be interpreted with caution, because only 6 studies estimated insoluble and soluble fiber, and there is no standard method used to derive these estimates. However, a characteristic of soluble fiber that may explain these findings is its propensity to increase intraluminal viscosity of the small intestine, thereby slowing the absorption of nutrients and potentially binding bile acids.³⁹ Such effects have been shown to decrease insulin secretion and improve glucose control,^{5,40,41} lower serum cholesterol levels,⁴² and possibly lower blood pressure.^{4,5} Nevertheless, the finding of inverse associations between both soluble and insoluble fiber and CHD risk in the present analysis supports recommendations to increase consumption of all types of fiber-rich foods.

An advantage of the pooling project is the inclusion of previously unpublished results that may have been susceptible to negative publication bias in the past. Thus, the pooled results may be closer to the true association than individually published findings. Other advantages include the systematic conduct of the analytic strategy across all studies, modeling exposures and important covariates uniformly. Such efforts decrease the likelihood of heterogeneity among RR estimates, thus enhancing generalizability of the pooled estimates. Therefore, the pooling project makes the best use of the available observational data to address hypotheses about diet and chronic disease. Although the ability to use the data from validation studies to correct the dietary fiber for measurement error was a strength of this analysis, the measurement error correction must be interpreted with caution because we were unable to adjust all of the covariates and other dietary factors for measurement error. Other limitations include the heterogeneity of dietary assessment and food table methods. For the soluble and insoluble fiber analyses, in particular, there is no accepted method of measurement, and only 6 of the studies had quantified these fiber types. However, we found only one instance of statistically significant heterogeneity in the RR estimates among studies, suggesting that the limitations of our methods did not undermine the validity of the findings.

In conclusion, our results suggest that dietary fiber intake during adulthood is inversely associated with CHD risk. Coronary risk was 10% to 30% lower for each 10-g/d increment of total, cereal, or fruit fiber. These results provide strong confirmation of the results of previously published cohort studies, and they are supported by numerous experimental studies that demonstrate a wide range of possible biological mechanisms through which fiber may reduce the risk of CHD. Therefore, the recommendations to consume a diet that includes an abundance of fiber-rich foods to prevent CHD are based on a wealth of consistent scientific evidence.

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