Dietary fiber intake in relation to coronary heart disease and all-cause mortality over 40 y: the Zutphen Study

Martinette T Streppel, Marga C Ocké, Hendriek C Boshuizen, Frans J Kok, and Daan Kromhout

ABSTRACT
Background: Little is known about the effects of dietary fiber intake on long-term mortality.
Objective: We aimed to study recent and long-term dietary fiber intake in relation to coronary heart disease and all-cause mortality.
Design: The effects of recent and long-term dietary fiber intakes on mortality were investigated in the Zutphen Study, a cohort of 1373 men born between 1900 and 1920 and examined repeatedly between 1960 and 2000. During that period, 1130 men died, 348 as a result of coronary heart disease. Hazard ratios were obtained from time-dependent Cox regression models.
Results: Every additional 10 g of recent dietary fiber intake per day reduced coronary heart disease mortality by 17% (95% CI: 2%, 30%) and all-cause mortality by 9% (0%, 18%). The strength of the association between long-term dietary fiber intake and all-cause mortality decreased from age 50 y (hazard ratio: 0.71; 95% CI: 0.55, 0.93) until age 80 y (0.99; 0.87, 1.12). We observed no clear associations for different types of dietary fiber.
Conclusions: A higher recent dietary fiber intake was associated with a lower risk of both coronary heart disease and all-cause mortality. For long-term intake, the strength of the association between dietary fiber and all-cause mortality decreased with increasing age.

INTRODUCTION
Epidemiologic studies have shown that consumption of (whole-grain) cereals (1–4) and vegetables and fruit (5–8) may lower the risk of coronary heart disease (CHD) mortality. Dietary fiber is one of the components that may be responsible for the beneficial effects of these foods. Results from intervention trials have shown that (water-soluble) dietary fiber may lower blood cholesterol concentrations (9), reduce blood pressure (10, 11), promote body-weight loss (12), and improve insulin sensitivity (13)—thereby reducing the risk of CHD mortality (14–17). In a pooled analysis of cohort studies, total dietary fiber intake was inversely associated with the risk of CHD mortality. Furthermore, the intakes of dietary fiber from cereals or from fruit were inversely associated, independent of each other, with the risk of CHD mortality (14). However, little is known about the effects of long-term dietary fiber intake on all-cause mortality.
For prospective cohort studies in which dietary fiber intake is assessed only at baseline, consumption patterns and product composition are assumed to be constant over the entire study period. However, it is unlikely that exposure measurements in the past accurately reflected long-term dietary fiber intake, because consumption patterns change during the life-course. To obtain correct estimates of the long-term effects of dietary fiber intake, repeated measures and time-specific food-composition tables are needed (18, 19). Moreover, the use of repeated measures, especially when a cumulative average method is used, reduces within-subject variation over time and, thereby, reduces misclassification of dietary fiber intake (20).

The objective of the present study was to investigate recent and long-term total dietary fiber intake and dietary fiber intake from various food groups in relation to CHD and all-cause mortality. For this purpose, we used up to 7 repeated measures of dietary fiber intake.

SUBJECTS AND METHODS
Study population
The Zutphen Study started as the Dutch contribution to the Seven Countries Study, a longitudinal study of the relations of diet and other risk factors with chronic diseases (21). The Zutphen Study has been carried out since 1960 among middle-aged men in Zutphen, an old industrial town (=30 000 inhabitants) in the eastern part of the Netherlands. In 1960, a random sample was drawn of 1088 men born between 1900 and 1919 and residing for ≥5 y in Zutphen. Of those men, 878 participated in the Zutphen Study (response rate: 81%), and 872 took part in both dietary and physical examinations. The examinations were repeated in 1965 and 1970. In 1985, the group of 554 survivors was extended with a new random sample of men from the same birth cohort. Of the 1266 men who were invited, 939 men participated (response rate: 74%), and 825 men took part in both dietary and physical examinations. These examinations were repeated in 1990, 1995, and 2000.
Baseline data were collected in 1960—i.e., before the Helsinki Declaration was developed—and oral informed consent was obtained in view of follow-up data. In 1985 and 1990, the study was approved by the Medical Ethics Committee of the University of Leiden (Leiden, Netherlands); in 1995 and 2000, the study was approved by the Medical Ethics Committee of the Netherlands Organisation for Applied Scientific Research (TNO).

Assessment of food consumption and dietary fiber intake

In all dietary surveys, information on habitual food consumption was collected by using the cross-check dietary history method (22), adapted to the usual Dutch diet (23, 24). This method provides information about the participant’s usual food consumption pattern in the period 6–12 mo before the interview. From 1985 on, the information about the usual food consumption pattern was limited to the month preceding the interview, because consumption patterns in 1985 were much more complicated than those in the 1960s. The interviews were carried out by experienced dietitians in spring and early summer. Each participant was interviewed, in the presence of his wife if possible, about his usual food consumption on weekdays and weekends. On the basis of this daily pattern, average food consumption during a day or week (first check) and the quantity of foods bought per week (second check) were estimated, and those values were presented to the participants to calculate and verify their food consumption.

In the present study, dietary fiber is defined as constituents of plant cells that cannot be digested or absorbed in the human stomach and small intestine (25, 26), particularly pectin, cellulose, hemicellulose, and lignin (27, 28). The daily intakes of dietary fiber, total energy, and other nutrients were calculated by using time-specific food-composition tables to account for changes in product composition and for quantitative improvements in analytic methods (28–33). In addition, intakes of dietary fiber from bread and other cereal products, potatoes, legumes, fruit, and vegetables were calculated. Because the ratio of the interindividual and intraindividual variance in dietary fiber intake in the Zutphen Study was 2.1, the reproducibility of dietary fiber intake was relatively high (34).

Assessment of potential confounders

Detailed information on the type and amount of smoking was collected by using standardized questionnaires (35). Information on alcohol intake and the use of a prescribed diet was obtained from the cross-check dietary history (23, 24). During physical examinations, the men’s weight and height were measured, and body mass index (BMI; in kg/m²) was calculated. Information on the prevalence of myocardial infarction, stroke, diabetes mellitus, and cancer was collected throughout the study. The men were classified into 4 levels of socioeconomic status according to occupation at baseline.

Case ascertainment

Participants were followed until death or were censored on June 30, 2000. Three participants were lost to follow-up during the study and were censored after their last physical examination. For each participant who died, the final allocation of the cause of death was done by a clinical epidemiologist (A Menotti, Association for Cardiac Research, Rome, Italy) and coded according to the Eighth Revision of the International Classification of Diseases (36). Because the underlying cause of death in elderly persons often is difficult to ascertain, we included both primary and secondary causes of death in our analyses. CHD deaths, including cases of sudden death, were codes 410–414.

Statistical analysis

Before analyses, intakes of dietary fiber and other nutrients were adjusted for total energy intake by using the nutrient residual method (37). First, Cox proportional hazard analyses were performed with age as the time variable, and information on dietary fiber intake was updated at each measurement round (called the most recent intake). Second, the cumulative average dietary fiber intake was used to better represent long-term intake (20). For those men who were newly included in the study in 1985, information on food consumption and dietary fiber intake was missing for the period from 1960 through 1970. Because average dietary fiber intake was lower in 1985 than in 1960–1970, taking cumulative averages excluding earlier intakes in the men included in 1985 would underestimate their intakes compared with those of the men included in 1960. To account for this underestimation, multiple imputation (5 times) (38) of dietary fiber intake and other dietary covariates from 1960 through 1970 was carried out among those men who were newly included in 1985, with the use of an adapted version of predicted mean matching (LG Lazzeroni et al, unpublished observations, 1990). The SAS code that was used for the multiple imputation can be downloaded (Internet: www.rivm.nl/sasmacros).

For Cox proportional hazard models, the PHREG procedure of SAS/STAT software (version 9.1; SAS Institute, Inc, Cary, NC) was used. Hazard ratios (HRs) were calculated for every additional 10 g of energy-adjusted total dietary fiber intake and for energy-adjusted dietary fiber intake from bread and other cereal products, potatoes, legumes, vegetables, and fruit. The covariates in the model were total energy intake (kcal); trans unsaturated fatty acid, saturated fat and cis polyunsaturated fat intakes [nutrient residuals (37)]; alcohol intake (indicator variables for 0, >0–20, or >20 g/d); wine use (yes or no); fish intake (g/d); BMI (kg/m²); cigar or pipe smoking (never or long-term former, recent former, or current) (35); cigarette smoking duration (divided by 10 y); the daily number of cigarettes smoked (divided by 10 y); use of a prescribed diet (yes or no); and baseline socioeconomic status (indicator variables for manual workers, nonmanual workers, small-business owners, or professionals). A possible interaction between dietary fiber intake and age was tested, and the interaction term was included in the model if interaction < 0.10 was considered statistically significant.

In the analyses for most recent intake, all covariates were updated at each measurement round, and all available data were used for the analyses. In the analyses for long-term intake, the cumulative average intake of all dietary covariates was updated at each measurement round. Nondietary covariates were updated with the most recent value at each measurement round. Because multiple imputations yielded 5 versions of the dataset, analyses of long-term dietary fiber intake were performed 5 times, and the results were pooled by using MIANALYZE in SAS/STAT.

RESULTS

Population characteristics

During 40 y of follow-up (mean survival age: 77 y), 1130 of the 1373 men participating in the Zutphen Study died (Table 1). A
**Dietary Fiber Intake and Coronary and All-Cause Mortality**

**Table 1** Characteristics of men participating in the Zutphen Study by year of measurement†

<table>
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<tr>
<td>Participants (n)</td>
<td>872</td>
<td>721</td>
<td>615</td>
<td>349</td>
<td>231</td>
<td>114</td>
<td>51</td>
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<td>1960 cohort</td>
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<td>1985 cohort</td>
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<td>Cumulative deaths (n)</td>
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<td>All-cause</td>
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<tr>
<td>Coronary heart disease</td>
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<tr>
<td>Age (y)†</td>
<td>49 ± 6†</td>
<td>54 ± 5</td>
<td>59 ± 5</td>
<td>71 ± 5</td>
<td>75 ± 5</td>
<td>80 ± 4</td>
<td>83 ± 3</td>
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<tr>
<td>Total energy intake (kcal/d)</td>
<td>3107 ± 668</td>
<td>2965 ± 672</td>
<td>2599 ± 534</td>
<td>2240 ± 507</td>
<td>2102 ± 463</td>
<td>2104 ± 463</td>
<td>2073 ± 447</td>
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<td>Dietary fiber intake (g · 2500 kcal−1 · d−1)</td>
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<tr>
<td>1960 cohort</td>
<td>27 ± 2</td>
<td>24 ± 7</td>
<td>24 ± 6</td>
<td>28 ± 7</td>
<td>29 ± 8</td>
<td>28 ± 7</td>
<td>26 ± 8</td>
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<td>1985 cohort</td>
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<tr>
<td>Alcohol intake (g/d)</td>
<td>4 ± 10</td>
<td>6 ± 11</td>
<td>9 ± 12</td>
<td>13 ± 17</td>
<td>10 ± 14</td>
<td>11 ± 14</td>
<td>12 ± 14</td>
</tr>
<tr>
<td>Wine users (%)</td>
<td>2 ± 5</td>
<td>5 ± 6</td>
<td>23 ± 29</td>
<td>34 ± 44</td>
<td>25 ± 35</td>
<td>33 ± 36</td>
<td>36 ± 37</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.1 ± 2.7</td>
<td>24.9 ± 2.7</td>
<td>25.2 ± 2.8</td>
<td>25.5 ± 3.1</td>
<td>25.5 ± 3.2</td>
<td>25.3 ± 3.4</td>
<td>26.0 ± 3.3</td>
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<tr>
<td>Overall smoking (%)†</td>
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<tr>
<td>Never and long-term former smoker</td>
<td>6</td>
<td>6</td>
<td>9</td>
<td>26</td>
<td>50</td>
<td>60</td>
<td>72</td>
</tr>
<tr>
<td>Recent former smoker</td>
<td>6</td>
<td>11</td>
<td>15</td>
<td>31</td>
<td>17</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>Cigarette smoker</td>
<td>74</td>
<td>61</td>
<td>53</td>
<td>30</td>
<td>23</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Cigar or pipe (or both) smoker</td>
<td>14</td>
<td>21</td>
<td>23</td>
<td>13</td>
<td>10</td>
<td>6</td>
<td>8</td>
</tr>
</tbody>
</table>

† n = 1373.  
‡ Defined as the subject’s age on December 31st of the year preceding the year of examination.  
§ ± ± SD (all such values).  
¶ Never and long-term former smokers are defined as subjects who never smoked or who stopped smoking ≥10 y previously. Recent former smokers are defined as subjects who stopped smoking <10 y previously.

...total of 348 men died of CHD. Among the participants who were included in the study in 1960, average daily dietary fiber intake decreased remarkably—from 33 g/d in 1960 to 21 g/d among the survivors in 2000. In addition, average total energy intake decreased from 3107 kcal/d in 1960 to 2073 kcal/d in 2000. Consequently, average daily dietary fiber intake per 2500 kcal remained relatively constant during 40 y of follow-up (Table 1). Among those men who were newly included in the study in 1985, the average daily intake of dietary fiber was comparable to that in men who started the study in 1960. Bread and other cereal products were the major source of total dietary fiber intake (between 29% and 34% of total dietary fiber intake) in all survey rounds (Figure 1). Total dietary fiber intake was most strongly correlated with dietary fiber intake from bread and other cereal products; correlation coefficients varied between 0.65 in 1995 and 0.78 in 1970.

**Dietary Fiber Intake and Mortality**

After adjustment for potential confounders, every additional 10 g of recent dietary fiber intake was inversely associated with CHD (HR: 0.83; 95% CI: 0.70, 0.98; Table 2) and all-cause mortality (0.91; 0.82, 1.00; Table 3). In crude analyses, every additional 10 g of long-term dietary fiber intake was inversely associated with all-cause mortality. However, adjustment for potential confounders attenuated this association, and the HR increased from 0.88 (0.79, 0.98) to 0.93 (0.83, 1.04) (Table 3).

For the association between long-term dietary fiber intake and all-cause mortality, we observed a significant and positive interaction with age (P = 0.03), which indicated that these associations weaken at a greater age. For all-cause death, the HR for every additional 10 g of long-term dietary fiber intake increased from 0.71 (0.55, 0.93) at age 50 y to 0.99 (0.87, 1.12) at age 80 y (Figure 2). For CHD mortality, the increase in HRs was comparable, but the associations were not statistically significant (Figure 2). We did not observe clear differences in the effects of dietary fiber from various sources on either CHD (Table 2) or all-cause mortality (Table 3).

**Discussion**

In the present study, recent dietary fiber intake was inversely associated with both CHD and all-cause mortality. For long-term intake, the strength of the associations between dietary fiber and all-cause mortality decreased with increasing age. We observed no clear differences in the effect of dietary fiber intake from various food groups on mortality.

The major strength of the present study was the collection of detailed information on usual dietary intake at each of 7 examination rounds and on coronary death during 40 y of follow-up. This enabled us to study recent and long-term—ie, cumulative average—dietary fiber intakes in relation to coronary and all-cause mortality and to study a possible interaction with age. The detailed information on potential confounders made it possible to study the independent relations of (various sources of) dietary fiber intake with mortality.

The present study also has some weaknesses. First, for those men who were newly included in the study in 1985, information about dietary fiber intake was missing for the period of 1960 to 1970. By multiple imputation (38) of dietary fiber intake and other dietary covariates in 1960–1970, we were able to counter an underestimation of cumulative average intake from 1985 for those men who were newly included in the study. However, the assumptions that were made in the multiple imputation method may have led to less precise effect estimates. Without imputation...
of dietary fiber intake and other dietary covariates, the associations between long-term dietary fiber intake and mortality were slightly attenuated, but the overall conclusions remained the same. Therefore, it is unlikely that bias occurred. Second, information on physical activity was not collected continuously during the whole follow-up period and therefore was not available for the present study. Adjustment for total energy intake per kg body weight as a proxy for physical activity (39) did not change our results.

Our results confirm those from large prospective cohort studies that found an inverse association between dietary fiber intake and CHD mortality (40–42). Pereira et al (14) conducted a pooled analysis of 10 cohort studies from the United States and Europe and estimated that every additional 10 g

TABLE 2
Recent and long-term energy-adjusted dietary fiber intake in relation to 40-y coronary heart disease (CHD) mortality (348 events) within the Zutphen Study\(^1\)

<table>
<thead>
<tr>
<th>Dietary fiber intake</th>
<th>Recent intake(^2)</th>
<th>Long-term intake(^2)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Crude</td>
<td>Adjusted</td>
</tr>
<tr>
<td></td>
<td>HR 95% CI</td>
<td>HR(^4,5) 95% CI</td>
</tr>
<tr>
<td>Total dietary fiber</td>
<td>0.87 0.74, 1.02</td>
<td>0.83 0.70, 0.98</td>
</tr>
<tr>
<td>Dietary fiber (per 10-g/d increment)</td>
<td></td>
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<tr>
<td>From bread and other cereal products</td>
<td>0.85 0.66, 1.10</td>
<td>0.84 0.64, 1.10</td>
</tr>
<tr>
<td>From potatoes</td>
<td>0.84 0.58, 1.22</td>
<td>0.71 0.48, 1.06</td>
</tr>
<tr>
<td>From legumes</td>
<td>0.64 0.34, 1.20</td>
<td>0.64 0.34, 1.20</td>
</tr>
<tr>
<td>From vegetables</td>
<td>1.02 0.55, 1.89</td>
<td>0.88 0.48, 1.65</td>
</tr>
<tr>
<td>From fruit</td>
<td>1.12 0.76, 1.65</td>
<td>1.13 0.75, 1.70</td>
</tr>
</tbody>
</table>

\(^1\) \(n = 1373\). HR, hazard ratio, obtained from time-dependent Cox proportional hazard models. Dietary fiber intake was adjusted for total energy intake by using the nutrient residual method (37).

\(^2\) Dietary fiber intake and all covariates were updated at each measurement round.

\(^3\) The updated cumulative average intake of all dietary covariates was used. Nondietary covariates were updated with most recent values at each measurement round.

\(^4\) HRs were adjusted for total energy, saturated fat, \textit{trans} unsaturated fatty acid, and \textit{cis} polyunsaturated fat acid intakes; alcohol intake; wine use; fish intake; prescribed diet; the number of cigarettes smoked; the duration of cigarette smoking; cigar or pipe smoking; BMI; and socioeconomic status.

\(^5\) Because of missing data in the covariates, the number of events was less than 348—ie, 336 CHD deaths.
Recent and long-term energy-adjusted dietary fiber intake in relation to 40-y all-cause mortality (1130 events) within the Zutphen Study

<table>
<thead>
<tr>
<th>Dietary fiber intake</th>
<th>Recent intake$^2$</th>
<th>Long-term intake$^2$</th>
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<tbody>
<tr>
<td></td>
<td>Crude HR 95% CI</td>
<td>Adjusted HR 95% CI</td>
</tr>
<tr>
<td>Total dietary fiber</td>
<td>0.89 0.81, 0.97</td>
<td>0.91 0.82, 1.00</td>
</tr>
<tr>
<td>Dietary fiber (per 10-g/d increment)</td>
<td></td>
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</tr>
<tr>
<td>From bread and other cereal products</td>
<td>0.88 0.76, 1.02</td>
<td>0.92 0.79, 1.07</td>
</tr>
<tr>
<td>From potatoes</td>
<td>1.05 0.86, 1.29</td>
<td>0.95 0.77, 1.19</td>
</tr>
<tr>
<td>From legumes</td>
<td>0.98 0.71, 1.35</td>
<td>0.83 0.60, 1.15</td>
</tr>
<tr>
<td>From vegetables</td>
<td>0.94 0.66, 1.32</td>
<td>0.91 0.64, 1.29</td>
</tr>
<tr>
<td>From potatoes</td>
<td>0.78 0.62, 0.99</td>
<td>0.94 0.74, 1.21</td>
</tr>
</tbody>
</table>

$^1$ n = 1373. HR, hazard ratio, obtained from time-dependent Cox proportional hazard models. Dietary fiber intake was adjusted for total energy intake by using the nutrient residual method (37).

$^2$ Dietary fiber intake and all covariates were updated at each measurement round.

$^3$ The updated cumulative average intake of all dietary covariates was used. Nondietary covariates were updated with most recent values at each measurement round.

$^4$ HRs were adjusted for total energy, saturated fat, trans unsaturated fatty acid, and cis polyunsaturated fat intakes; alcohol intake; wine use; fish intake; prescribed diet; the number of cigarettes smoked; duration of cigarette smoking; cigarette or pipe smoking; BMI; and socioeconomic status.

$^5$ Because of missing data in the covariates, the number of events was less than 1130—ie, 1048 all-cause deaths.

dietary fiber/d reduced CHD mortality risk by 19%. We found a similar association of recent dietary fiber intake and CHD mortality. In the pooled analysis of Pereira et al (14), this association became stronger (HR: 0.73; 95% CI: 0.61, 0.87) after adjustment for measurement error (by using the regression calibration method) in dietary fiber intake. In the present study, the cumulative average dietary fiber intake was used not only to better represent long-term intake but also to reduce measurement error by reducing the within-subject variation in dietary fiber intake over time (20). Until age 70 y, the effects of cumulative average dietary fiber intake on CHD mortality were indeed stronger than those of recent intake; however, the observed associations were not statistically significant.

Moreover, Pereira et al (14) observed that dietary fiber intakes from cereals or from fruit were, independent of each other, inversely associated with the risk of CHD mortality. Every additional 10 g of dietary fiber intake from cereals lowered CHD mortality risk by 29%, and that of dietary fiber from fruit lowered CHD mortality risk by 35%. In the present study, we did not observe a clear difference between the effects of dietary fiber from various sources on CHD mortality. Because total dietary fiber intake was inversely associated with mortality, the intake of all types of fiber-rich foods should be encouraged.

Several mechanisms have been proposed by which dietary fiber may reduce CHD mortality risk (16). Soluble fibers increase the rate of bile acid excretion by binding bile acids in the small intestine, thereby reducing serum total and LDL cholesterol (43–45). In the large intestine, soluble fibers are fermented by bacteria. One of the by-products of this process is short-chain fatty acids. Hepatocytes, when exposed to these short-chain fatty acids, may increase glucose oxidation, decrease free fatty acid release and insulin clearance, and improve insulin sensitivity (46, 47). The increase in insulin sensitivity results in lower circulating insulin concentrations, an effect that has been shown to reduce blood pressure (48). Moreover, the short-chain fatty acids inhibit cholesterol synthesis by limiting the action of 3-hydroxy-3-methylglutaryl–coenzyme A reductase (46). The effects of dietary fiber on LDL cholesterol, insulin sensitivity, and blood pressure
may explain the inverse association between dietary fiber intake and CHD mortality. In the present study, the associations between dietary fiber intake and mortality were stronger for recent intake than for long-term intake. Moreover, the effects of long-term dietary fiber were stronger at younger ages—in other words, when dietary fiber intake was calculated over a shorter period of time. This finding suggests that the mechanisms by which dietary fiber may reduce mortality are primarily short-term effects.

Besides the inverse association between dietary fiber intake and CHD mortality, we observed a significant and inverse association with all-cause mortality, which confirmed the results of Todd et al. (49) and Kromhout et al. (50). However, for long-term dietary fiber intake, an inverse association with all-cause mortality was present only until age 70 y. Because the effects on all-cause mortality were less strong than those on CHD mortality, the inverse association between dietary fiber intake and all-cause mortality is mainly explained by the inverse association with CHD mortality.

In conclusion, recent dietary fiber intake was inversely associated with CHD and all-cause mortality risk. For long-term intake, the strength of the associations between dietary fiber and all-cause mortality decreased with increasing age. Differences between the effects of various sources of dietary fiber on mortality were not observed.

The authors thank Marieke Hoevenaar-Blom for performing the preliminary analyses in the present study.

The authors’ responsibilities were as follows—MTS: analyzed the data and wrote the manuscript; MCO: obtained funding, analyzed the data, and contributed to the writing of the manuscript; HCB: analyzed the data and revised the manuscript; FIK: assisted with statistical analyses and revised the manuscript; and DK: proposed and designed the study, obtained the data, contributed to the writing of the manuscript; and revised the manuscript. None of the authors had a personal or financial conflict of interest.

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