A Food Pattern Predicting Prospective Weight Change Is Associated with Risk of Fatal but Not with Nonfatal Cardiovascular Disease\textsuperscript{1,2}

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Abstract

Recently, a food pattern predictive for prospective weight change was identified within the European Prospective Investigation into Cancer and Nutrition-Potsdam cohort. Given the possible impact of weight change on cardiovascular disease (CVD) risk, we examined the association between the above mentioned food pattern and risk of CVD. The analyzed food pattern was defined by a high consumption of whole-grain bread, fruits, fruit juices, grain flaks and/or cereals, and raw vegetables, and a low consumption of processed meat, butter, high-fat cheese, margarine, and meat other than poultry. The associations between quartiles of the food pattern score and CVD morbidity and mortality were examined in 26,238 subjects of the European Prospective Investigation into Cancer and Nutrition-Potsdam cohort using a Cox's Proportional Hazards model for competing risks. During 6.4 y of follow-up, 379 incident cases of CVD were identified, of which 68 were fatal events. The food pattern was not associated with risk of nonfatal CVD. After adjusting for cardiovascular risk factors, the hazard ratios for fatal CVD across increasing quartiles of the score were 1.00, 0.85, 0.31, and 0.47, respectively ($P$ for trend = 0.016). The association of the food pattern with CVD risk differed between fatal and nonfatal events ($P$ for difference = 0.05). These findings from a large German cohort indicate that a food pattern predicting prospective weight change may be associated with the risk of fatal CVD. J. Nutr. 137: 1961–1967, 2007.

Introduction

Over the past decades, the prevalence of obesity has been increasing, and according to recent estimates, there are >300 million clinically obese people worldwide (1). Obesity is associated with a number of medical conditions including type 2 diabetes, cardiovascular disease (CVD),\textsuperscript{3} hypertension, dyslipidemia, and premature mortality (2). Given the impact of obesity on public health, efforts for weight reduction continue to be proposed as an effective measure of intervention. However, there is still no agreement about the impact of weight change compared with weight stability on CVD risk. Weight loss has at least temporary beneficial health effects, including improvements in blood pressure, blood lipids, fasting glucose, and insulin resistance (2). In contrast, weight gain is accompanied by an increase in several risk factors such as diabetes and hypertension (3–6), which contribute to the increased risk of coronary heart disease (CHD) (7–9) and stroke (10). Nevertheless, weight stability and even moderate weight gain have been associated with lowest mortality rates in several prospective studies, however, the strength of this relation is influenced by a number of factors such as health status, baseline weight, onset of weight change, and age (11–18). Especially in older people, weight loss strongly interacts with frailness, morbidity, and impaired functional status (19). Diet is one of the major determinants of body weight. Therefore it is conceivable that a diet of adequate nutrient density that supports a stable weight may be advantageous for good health and longevity.

Recently, a food pattern was identified that correlates positively with carbohydrate density and fiber and negatively with fat density. A high pattern score, characterized by a high consumption of whole-grain bread, fruits, fruit juices, grain flaks and/or cereals, and raw vegetables and a low consumption of processed meat, butter, high-fat cheese, margarine, and meat other than poultry, was predictive for low prospective weight change within the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam cohort (20). We studied the relationship between this food pattern and the risk of CVD within middle-aged and elderly subjects of the EPIC-Potsdam study.

Subjects and Methods

Study population. The EPIC-Potsdam Study is part of the large-scale European-wide prospective cohort study, EPIC. All study procedures...
were approved by the Ethical Committee of the state of Brandenburg, Germany, and the participants gave their informed consent. In total, 27,548 individuals, (16,644 women and 10,904 men), aged 35 to 65 y at baseline, were recruited from the general population between 1994 and 1998 (21). Participants underwent a baseline examination, including standardized blood pressure measurements, measurements of weight and height, self-administered questionnaires on diet and lifestyle, computer-guided interviews, and blood sampling. Information about changes in lifestyle and incident diseases is biennially assessed by self-administered questionnaires (22). In the current analysis, we excluded participants with missing follow-up (n = 293), self-reported myocardial infarction (MI) or stroke at baseline (n = 785), and missing dietary (n = 17), anthropometrical (n = 195), or lifestyle (n = 18) data, which left a total of 16,108 women and 10,130 men for analysis.

Data collection. Dietary habits during the previous year were assessed at baseline using a validated self-administered FFQ (23–26) that included questions on frequency and portion size of 148 single food items. On the basis of culinary usage or nutrient profiles, food items were grouped into 49 food groups (27). We constructed a food pattern score variable that was obtained by the unweighted sum of standardized intake of whole-grain bread, fresh fruit, fruit juice, grain and/or cereals, and raw vegetables minus the sum of the standardized intake of processed meat, butter, high-fat cheese, margarine, and meat other than poultry. This food pattern score was identified using reduced rank regression (RRR), and a high value of the score predicted a low prospective weight change within the EPIC-Potsdam cohort (20).

Anthropometric measures were obtained by trained personnel with participants dressed in light clothes and without shoes (28). BMI was calculated as body weight divided by height squared (kg/m²). Data about smoking status, physical activity, educational attainment, and medical history were assessed during the computer-guided interview conducted in the study center at baseline. Information about smoking was collapsed into 3 groups defined as “present smoking,” “never smoking,” and “past smoking.” Physical activity level (PAL) was calculated from the self-reported duration and intensity of physical activity (including, e.g., walking, bicycling, sports, and gardening) taking into account the metabolic equivalents (MET) (29). Three levels of educational attainment were defined: “less than high-school education,” “high-school education,” and “university degree.” Prevalent hypertension was defined as a systolic blood pressure ≥ 140 mm Hg or a diastolic blood pressure of ≥ 90 mm Hg, taking antihypertensive medication, or a self-report of a diagnosis of hypertension. A history of diabetes was based on self-reports of a diagnosis or taking antidiabetic medication. A history of hyperlipidemia was based on self-reports of a diagnosis or of taking cholesterol-lowering medication.

Assessment of incident cardiovascular events. Based on follow-up questionnaires that were returned and completed by 93 to 96% of the study participants at each wave, potential cases of MI or stroke were identified by self-reports or death certificates. Additional potential cases were identified through postbaseline dietary changes that were reported to be due to CVD. All potential incident cases were verified by medical records from the hospital, by contacting the patient’s attending physician, or by a review of death certificates according to WHO Monitoring Trends and Determinants in Cardiovascular Disease Study (MONICA) criteria (30) and those who developed a MI [International Classification of Diseases, tenth revision (ICD-10) I21] or stroke (ICD-10 I60, I61, I63, and I64) were used in the analyses. Death within 28 d after diagnosis and a death certificate in which the underlying cause of death was recorded with the above codes was considered as a fatal event resulting from CVD.

Statistical methods. Major characteristics of the study population are given as means ± SD or frequencies. The unpaired Student’s t test (continuous variables) and chi-square test or Fisher’s exact test (categorical variables) were used to compare characteristics of subjects with and without incident CVD, as appropriate. We categorized the study population into 4 groups based on quartiles of the above-mentioned food pattern score derived from Schulz et al. (20). Linear trend across quartiles was tested by introducing the quartile value as a continuous variable in a linear regression model for (continuous variables); for categorical variables, a test of independency (chi-square test) was applied.

Cox’s Proportional Hazards regression model was performed to investigate the relation between categories of the food pattern score and risk of CVD. Follow-up time was expressed as the time interval between baseline examination and first event of CVD (cases) or between baseline examination and date of death from any other cause, drop out, or most recent follow-up, whichever came first (remaining cohort). Hazard ratios (HR), presented as point estimates and corresponding 95% CI, were adjusted for age and gender in model 1. Model 2 additionally included total energy intake, BMI, smoking history, alcohol consumption at baseline, PAL, and educational attainment as covariates. The final model (model 3) was further adjusted for history of diabetes, history of hypertension, and history of hyperlipidemia. Additionally, we studied the impact of the potential effect modifiers on the association between the dietary pattern score and CVD risk by including interaction terms between quartiles of the pattern score variable with gender, age (≥ 60 y), obesity at baseline (BMI < 30 g/m², BMI ≥ 30 g/m²).

To compare the effects of risk factors for fatal with those of nonfatal CVD, competing risk analysis (31) was applied. Here, we used the data duplication method proposed by Lunn and McNeil (32) and applied Cox’s regression to the augmented data set stratified by the type of outcome. To account for doubling of the data, robust sandwich covariance estimates were employed using the COVSANDWICH (AGGREGATE) option in the PHREG procedure. Beside simultaneous risk estimation of a given exposure on more than one outcome, the model for the augmented data can be utilized to test for equality of exposure parameters. For that purpose, 2 different models were fitted. The 1st model included distinct exposure variables for each outcome, assuming that there were different effects of the exposure on risk of nonfatal CVD compared with risk of fatal CVD. The 2nd model was restricted to only 1 set of exposure variables, assuming that the food pattern score exhibited equal effects on either outcome. The model fit of the 2 models was then compared using the likelihood ratio test.

Statistical analyses were done using SAS software package, release 9.1 (SAS Institute). All tests were performed 2-sided with P < 0.05 considered significant.

Results

Among 26,238 participants with full covariate information (16,108 women, 10,130 men), a total of 379 incident cases of CVD (MI, n = 201; stroke, n = 178), including 68 fatal events (MI, n = 41; stroke, n = 27), were identified between baseline examination and June 2006. Overall, the ratio of MI to stroke did not differ between participants developing a nonfatal event and those developing a fatal event, as determined by the chi-square test (P = 0.18). Participants with incident CVD exhibited a similar risk profile at baseline when comparing nonfatal with fatal cases (Table 1); with the exception of relative usual intake of carbohydrates (g·MJ⁻¹·d⁻¹) and prevalent hypertension frequency, which was 84% in subjects developing a fatal CVD and 71% in those developing nonfatal CVD (P = 0.035). Both CVD groups tended to be older, to have a higher BMI, to have a higher proportion of men, to be current smokers, and to have baseline hypertension when compared with the remaining cohort. However, a significantly higher prevalence of hyperlipidemia and diabetes was restricted to subjects developing nonfatal CVD, whereas those with subsequent fatal CVD differed from participants without CVD in the fiber and carbohydrate content (g·MJ⁻¹·d⁻¹) of their diet.

Across the quartiles of the food pattern score there were striking differences in the distribution of men and women (Table 2), with men accounting for more than two-thirds of the participants within the 1st quartile of the pattern score and women dominating the 4th quartile (80%). The proportion of subjects who were current smokers at baseline recruitment decreased

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Discussion

This study showed that a food pattern of high consumption of whole-grain bread, fresh fruit, fruit juices, grain flakes and/or cereals, and raw vegetables, combined with a low consumption of processed meat, butter, high-fat cheese, margarine, and meat other than poultry significantly exhibited different associations on nonfatal vs. fatal CVD in middle-aged and elderly participants of the EPIC-Potsdam cohort. Although CVD morbidity was not related to the exposure, the risk of CVD mortality significantly decreased across quartiles of the food pattern score. The inverse association between the food pattern and CVD mortality was still observed after multivariable adjustment, suggesting that the effect of this pattern is independent of traditional CVD risk factors.

In 2005, Schulz et al. (20) identified this pattern on the basis of dietary information about macronutrient composition. The
pattern was inversely correlated with energy derived from fat and positively correlated with energy derived from carbohydrates and fiber, and a high pattern score was associated with low prospective weight change in the EPIC-Potsdam cohort. Weight stability was related to lower mortality rates or CVD risk than weight loss or weight gain in a number of prospective studies (11–18). This is in line with our finding that a diet favoring weight stability is associated with lower CVD mortality. However, there was no significant association with CVD morbidity.

A number of prospective studies suggest that dietary patterns may predict a risk of CVD (33). Because different techniques have been used to define summary variables, resulting dietary pattern are rather heterogeneous and can not be easily compared. Furthermore, dietary patterns may differ between populations according to sex, as well as ethnic and cultural groups. Yet, a high intake of fruits, vegetables, and whole-grain products is a common feature of dietary patterns associated with significantly reduced risk of CHD mortality (34), CVD mortality (35–37), total incidence of MI (38), or CHD (39,40), as well as the incidence of CVD (41). Fruits, vegetables, and whole-grain bread are major components of our food pattern score, too. Given this similarity, our finding of reduced CVD mortality associated with an increase of the food pattern score is thus in line with the above-mentioned studies, whereas the lack of association between the food pattern and CVD morbidity is difficult to explain. However, a “prudent” diet consisting of higher intake levels of fruits, vegetables, wholemeal bread, pasta, rice, oatmeal products, and fish and a lower intake of white bread was not associated with the risk of fatal CVD in men or with total CHD risk among men and women of the MONICA Denmark study (42,43).

Our finding of differences in the relation between the food pattern score and fatal vs. nonfatal CVD incidence is hard to understand. Interestingly, we are not the first group reporting discrepancies between risk estimates for CVD morbidity and mortality. A recently published pooled analysis of 10 prospective cohort studies estimated that each 10-g increment in dietary fiber was associated with a rather modest decreased relative risk (RR) for nonfatal CHD by 14%, whereas risk reduction of fatal CHD would be nearly twice (RR = 0.73; 95% CI = 0.61–0.87) (44). The authors did not discuss this phenomenon, but it was hypothesized that the observed inverse association of fiber intake with risk of CVD might reflect a healthy lifestyle rather than a causal effect (45). Our food pattern score is positively correlated with fiber density and some of the components of the score (e.g., fruits, vegetables, whole-grain bread) are main sources of dietary fiber, which may explain similarities in findings. If there are unknown factors that predominantly effect CVD mortality, existing studies on the association between dietary pattern and CVD should be interpreted cautiously. There are a number of

### TABLE 2 Baseline characteristics of participants of the EPIC-Potsdam-cohort according to quartiles of the food pattern score

<table>
<thead>
<tr>
<th>General characteristics</th>
<th>Quartiles of food pattern score</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Age, y</td>
<td>50.0 ± 8.3</td>
<td>50.6 ± 9.0</td>
</tr>
<tr>
<td>Men, %</td>
<td>68.0</td>
<td>41.1</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.5 ± 4.1</td>
<td>26.3 ± 4.4</td>
</tr>
<tr>
<td>Physical activity level, MET/d</td>
<td>1.8 ± 0.4</td>
<td>1.8 ± 0.4</td>
</tr>
<tr>
<td>Smokers, %</td>
<td>29.0</td>
<td>21.7</td>
</tr>
<tr>
<td>University degree, %</td>
<td>39.2</td>
<td>36.1</td>
</tr>
<tr>
<td>Medical history, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalent hypertension</td>
<td>50.3</td>
<td>49.0</td>
</tr>
<tr>
<td>Prevalent hyperlipidemia</td>
<td>26.8</td>
<td>27.7</td>
</tr>
<tr>
<td>Prevalent diabetes</td>
<td>1.1</td>
<td>1.1</td>
</tr>
<tr>
<td>Food groups, g/d</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole-grain bread</td>
<td>22.4 ± 35.3</td>
<td>32.1 ± 40.4</td>
</tr>
<tr>
<td>Fresh fruit</td>
<td>98.1 ± 63.4</td>
<td>116.7 ± 70.0</td>
</tr>
<tr>
<td>Fruit juice</td>
<td>127.1 ± 146.2</td>
<td>161.5 ± 173.7</td>
</tr>
<tr>
<td>Grain flakes, grains, muesli</td>
<td>1.1 ± 4.2</td>
<td>2.1 ± 6.4</td>
</tr>
<tr>
<td>Raw vegetables</td>
<td>37.9 ± 27.0</td>
<td>45.4 ± 28.4</td>
</tr>
<tr>
<td>Processed meat</td>
<td>94.8 ± 62.1</td>
<td>60.1 ± 34.4</td>
</tr>
<tr>
<td>Butter</td>
<td>14.4 ± 17.7</td>
<td>8.7 ± 11.0</td>
</tr>
<tr>
<td>High-fat cheese</td>
<td>41.7 ± 32.5</td>
<td>28.3 ± 21.5</td>
</tr>
<tr>
<td>Margarine</td>
<td>21.6 ± 20.1</td>
<td>16.0 ± 13.7</td>
</tr>
<tr>
<td>Meat other than poultry</td>
<td>63.8 ± 39.1</td>
<td>43.4 ± 23.5</td>
</tr>
<tr>
<td>Further dietary characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy intake, MJ/d</td>
<td>10.5 ± 3.1</td>
<td>8.6 ± 2.6</td>
</tr>
<tr>
<td>Fiber density, g - MJ/d - g⁻¹</td>
<td>2.1 ± 0.4</td>
<td>2.4 ± 0.4</td>
</tr>
<tr>
<td>Carbohydrate density, g - MJ/d - g⁻¹</td>
<td>24.0 ± 3.2</td>
<td>26.3 ± 3.1</td>
</tr>
<tr>
<td>Fat density, g - MJ/d - g⁻¹</td>
<td>10.2 ± 1.5</td>
<td>9.5 ± 1.3</td>
</tr>
<tr>
<td>Alcohol consumption, g/d</td>
<td>21.0 ± 24.8</td>
<td>14.3 ± 17.8</td>
</tr>
</tbody>
</table>

1 Values are means ± SD or frequencies, n = 26,238.

2 The food pattern score is given by the unweighted sum of standardized intake of whole-grain bread, fresh fruit, fruit juices, grains and cereals, and raw vegetables minus the sum of standardized intake of processed meat, butter, high fat cheese, margarine, and meat other than poultry.

3 P for linear trend (continuous variables) or P of chi-square test (categorical variables).

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studies that rely only on mortality rates (34–37,42). In contrast, those studies addressing total incidence rates without discriminating between fatal and nonfatal events may be biased by a disproportionate influence of unknown factors on fatal risk estimates that also shift estimates for total incidence (38,40–43,46–49).

Supported by previous studies, our investigation raises the question as to whether the severity of a cardiovascular event may be determined by factors other than the food pattern score, which, to date, has not yet been considered. The WHO MONICA project demonstrated that about two-thirds of the decline in CHD mortality between the early 1980s and 1990s is explained by a decrease in CHD incidence, whereas the remaining one-third of the decline is explained by improvements in the medical management of the disease (30). Given the impact of medical management on the course of disease, group-specific differences in the access or usage of health care may at least partly explain our finding of an inverse association between the food pattern score and CHD mortality. Unfortunately, case numbers were rather low among those subgroups that could bias results by disproportionating frequency of fatal CVD events. However, participants in the highest quartile of the pattern score exhibited characteristics associated with a worse prediction of survival from CVD, insofar as they were more likely to have no partner, to be unemployed (data not shown), or to be female. Thus, major subgroups with a higher chance of dying from CVD do not dominate the reference group, which otherwise could have explained the inverse association of the pattern score with fatal CVD in the absence of an association with nonfatal CVD. Also, adjustment for partnership and employment status only marginally changed the relative risk estimates (data not shown). Furthermore, interaction terms between quartiles of the pattern score variable with gender or with age (<60 y, ≥60 y) were not significant in the multivariable adjusted model (model 3), suggesting that neither gender nor higher age, as a major determinant of case fatality (50), modifies the relation between exposure and severity of CVD.

When interpreting the results herein, methodological aspects related to the study should be considered. Due to the lower number of fatal compared with nonfatal cases, effects are less likely to reach significance in the first group. Despite this situation, there was a significant inverse association with the risk of CVD mortality across quartiles of the food pattern score, but not with the risk of CVD morbidity. To our knowledge, this is the first study analyzing the association of a food pattern with nonfatal vs. fatal CVD using a single statistical model and taking advantage of comparative risk estimation.

Our study is limited by the fact that exposure was defined on the basis of a single FFQ. Documented food group intake was found to be reproducible and of moderate validity. We cannot rule out that inaccuracies of the FFQ or misreporting may have resulted in misclassification of participants and thus a distortion of risk estimates. Obese people are more likely to underreport their energy intake as well as misreport their dietary composition (51) and, in the detailed analyses of Schulz et al. (20), the association between the food pattern score and prospective weight change was stronger in nonobese subjects. However, we found no evidence of effect modification by obesity (BMI <30 g/m², BMI ≥30 g/m²), suggesting that our findings are not distorted by obese subjects who tend to misreport their dietary habits.

We tried to minimize the possibility of confounding using a sequential modeling approach to adjust for a wide range of well-described potential confounding factors, which resulted in only marginally altered risk estimates for either outcome. Furthermore, the use of a food pattern is an approach that infers tight interactions between nutrients and foods commonly consumed in combination. Although this method does not distinguish between particular nutrients possibly effecting disease risk, it is closer to reality than traditional single-nutrient or single-food approaches because it accounts for interactions and substitution effects between foods and macronutrients, respectively. The food pattern approach is easy to translate into dietary advice and may be particularly informative when a combination of several dietary components is relevant for a disease, however, this approach should be considered as a complementary method in the study of diet–disease relationships (52,53).

### Table 3: Risk of CVD within the EPIC-Potsdam cohort according to quartiles of the food pattern score

<table>
<thead>
<tr>
<th></th>
<th>CVD morbidity</th>
<th>CVD mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Quartiles of food pattern score²</td>
<td>P for trend³</td>
</tr>
<tr>
<td>CVD events, n</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Model 4</td>
<td>108</td>
<td>72</td>
</tr>
<tr>
<td>HR</td>
<td>1.00</td>
<td>0.75</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.56–1.02</td>
<td>0.53–1.03</td>
</tr>
<tr>
<td>Model 5</td>
<td>1.00</td>
<td>0.79</td>
</tr>
<tr>
<td>HR</td>
<td>0.58–1.07</td>
<td>0.57–1.12</td>
</tr>
<tr>
<td>95% CI</td>
<td>0.58–1.05</td>
<td>0.54–1.09</td>
</tr>
</tbody>
</table>

¹ Values are HR or 95% CI, n = 26,238.
² The food pattern score is given by the unweighted sum of standardized intake of whole-grain bread, fresh fruit, fruit juices, grains and cereals, and raw vegetables minus the sum of standardized intake of processed meat, butter, high fat cheese, margarine, and meat other than poultry.
³ Further adjusted for total energy intake, BMI, smoking status (present smoker, past smoker, never smoker), alcohol consumption, PAL, and educational attainment (less than high-school education, high-school education, university degree).
⁴ Additionally includes history of hypertension, history of hyperlipidemia, history of diabetes.
Several approaches have been used to define dietary patterns (33,54,55) and our food pattern was identified by use of reduced rank regression (20), a dimension reduction technique recently introduced to nutritional epidemiology (56). Essentially, this method extracts linear functions of predictors (food groups) such that variation in a set of disease-related response variables is maximized. Thus, it allows modeling of a food pattern associated with a predefined pathway between diet and disease. In the underlying study of Schulz et al. (20), nutrient densities of daily dietary fat, carbohydrates, and fiber (each in g/MJ) have been used as response variables for derivation of the pattern. When separately analyzing the relation of fat density, carbohydrate density, or fiber density to CVD risk within the EPIC-Potsdam cohort, we see similar differences between fatal and nonfatal incidence rates (data not shown). However, our study focuses on a food pattern that combines the information of these 3 highly correlated nutrient densities, which has the advantage that changes in the usual intake of several key food groups on CVD risk can be estimated. It is important to note that the choice of response variables in RRR depends on current knowledge about pathways contributing to disease risk. Just recently, RRR has successfully been applied to identify a food pattern associated with homocysteine metabolism within the Coronary Risk Factors for Atherosclerosis in Women (CORA) Study (38). This food pattern predicted CHD risk within the CORA study and the EPIC-Potsdam study and shares some similarities with the body weight-associated food pattern of Schulz et al. (20), because intake of fresh fruits and whole-grain bread is positively correlated with both pattern scores. Because diet is likely to effect the development of a given disease via several mechanisms and pathways, it is not surprising that certain food groups are components of more than one RRR-derived food pattern; even if the response sets differ (e.g., a plant-based diet may modulate body weight as well as homocysteine metabolism, blood lipids, or blood pressure).

In conclusion, we analyzed the relation between CVD risk and a food pattern recently shown to be associated with stable weight and demonstrated significantly different effects of the exposure on fatal vs. nonfatal CVD. The inverse association of the food pattern with fatal CVD but not nonfatal CVD requires further investigation. Together with findings from other studies, our results should be regarded as strong evidence for further investigation into this potentially important aspect of CVD occurrence and its prevention in populations.

Acknowledgments

We thank Ellen Kohlsdorf and Wolfgang Berningau for data management and Wolfgang Fleischhauer for case ascertainment.

Literature Cited


Food pattern and cardiovascular disease


