CLINICAL RESEARCH STUDY

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Total Antioxidant Capacity from Diet and Risk of Myocardial Infarction: A Prospective Cohort of Women

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ABSTRACT

BACKGROUND: There are no previous studies investigating the effect of all dietary antioxidants in relation to myocardial infarction. The total antioxidant capacity of diet takes into account all antioxidants and synergistic effects between them. The aim of this study was to examine how total antioxidant capacity of diet and antioxidant-containing foods were associated with incident myocardial infarction among middle-aged and elderly women.

METHODS: In the population-based prospective Swedish Mammography Cohort of 49-83-year-old women, 32,561 were cardiovascular disease-free at baseline. Women completed a food-frequency questionnaire, and dietary total antioxidant capacity was calculated using oxygen radical absorbance capacity values. Information on myocardial infarction was identified from the Swedish Hospital Discharge and the Cause of Death registries. Hazard ratios (HR) and 95% confidence intervals (CI) were calculated using Cox proportional hazard models.

RESULTS: During the follow-up (September 1997-December 2007), we identified 1114 incident cases of myocardial infarction (321,434 person-years). In multivariable-adjusted analysis, the HR for women comparing the highest quintile of dietary total antioxidant capacity to the lowest was 0.80 (95% CI, 0.67-0.97; *P* for trend = 0.02). Servings of fruit and vegetables and whole grains were nonsignificantly inversely associated with myocardial infarction.

CONCLUSIONS: These data suggest that dietary total antioxidant capacity, based on fruits, vegetables, coffee, and whole grains, is of importance in the prevention of myocardial infarction.

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KEYWORDS: Antioxidants; Cohort; Myocardial infarction

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Coronary heart disease is a major cause of death in women.[1] High levels of reactive oxygen species and reactive nitrogen species have been implicated in the initiation and progression of atherosclerosis,[1-3] the underlying cause of coronary heart disease. Well-established coronary heart disease risk factors such as smoking[4] and aging[5] are associated with increased reactive oxygen species and reactive nitrogen species production, as are obesity,[6] hypertension,[7] and excessive alcohol consumption.[8] Antioxidants are proposed to play a key role in mitigating the atherosclerotic process by scavenging reactive oxygen species and reactive nitrogen species.[1] Accordingly, consumption of fruits and vegetables, major sources of antioxidants, have been inversely related to coronary heart disease.[9] By contrast, the use of high-dose single-antioxidant supplements does not protect against

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coronary heart disease and may even increase mortality, as shown by randomized controlled trials.[10-15] Thus, high-dose single-antioxidant supplements are not a good substitute for the very complex antioxidant network of thousands of compounds in foods, present at concentrations far below those used in most randomized controlled trials.

To our knowledge, no study has investigated the overall effect of the complex antioxidant network in diet in relation to coronary heart disease. The total antioxidant capacity measures, in one single value, the free-radical-reducing capacity of all antioxidants present in foods and the synergistic effects between these substances. The aim of the present study was to examine how total antioxidant capacity of diet and antioxidant-containing foods were associated with incident myocardial infarction among women from the population-based, prospective Swedish Mammography Cohort.

CLINICAL SIGNIFICANCE

- Dietary antioxidants are hypothesized to protect against coronary heart disease; however, no previous study has investigated the association between all antioxidants present in the diet and myocardial infarction.
- Total antioxidant capacity measures in a single value all antioxidants present in diet and the synergistic effects between them.
- A diet high in total antioxidant capacity, based on fruits, vegetables, coffee, and whole grains, was associated with lower myocardial infarction incidence.

METHODS

Ethics Statement

The Regional Ethical Review Board at Karolinska Institutet (Stockholm, Sweden) approved this investigation, and return of the self-administrated questionnaire was considered to imply informed consent to participate in the study.

Study Population

The Swedish Mammography Cohort was established between 1987 and 1990 among women residing in the Uppsala and Västmanland counties in central Sweden. All women born 1914-1948 were sent a questionnaire concerning diet, educational level, weight and height, and reproductive factors; 74% completed the questionnaire. To expand exposure data, a new questionnaire was sent in 1997 to all 56,030 eligible cohort members. The expanded 1997 questionnaire included questions on diet and questions on all major lifestyle factors, history of diseases, and use of some medications. Because more information on potential risk factors for myocardial infarction was collected in the 1997 questionnaire, completed by 38,984 women (70%), this questionnaire served as the baseline in the present study.

We excluded women having history of cancer (except nonmelanoma skin cancer) and women reporting extreme total energy intake (± 3 SD from the mean value for $\log_e$-transformed energy). We also excluded women with cardiovascular diseases (identified through the Swedish Hospital Discharge Registry, *International Statistical Classification of Disease*, 10th Revision (ICD-10); code I11.0, I20-25, I50, and I60-69)[16] and diabetes (self-reported or recorded in the Swedish Hospital Discharge Registry) because these diagnoses may lead to change in dietary habits. The remaining cohort of 32,561 women was followed from September 1997 through December 2007.

Assessment of Food-frequency Questionnaire-based Total Antioxidant Capacity Estimates and Other Exposures

Women completed a 96-item food-frequency questionnaire on which they were asked how often, on average, they consumed each type of food or beverage during the last year. There were 8 predefined response categories, ranging from "never/seldom" to "3 or more times per day." Open-ended questions were used for foods and beverages commonly consumed (eg, bread, coffee and tea). The calculation of total antioxidant capacity estimates is described in detail elsewhere.[17] Briefly, we calculated estimates of total antioxidant capacity from diet using a database of the most common foods in the United States analyzed with the oxygen radical absorbance capacity (ORAC) assay.[18-20] ORAC measures the antioxidant capacity of diet to reduce free radicals, taking into account the synergism between compounds. In the 96-item food-frequency questionnaire, there were 31 items (including 17 fruit and vegetable items) with available ORAC values. Total antioxidant capacity of diet and nutrient intakes (saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids) were calculated by multiplying the average frequency of consumption of each food by ORAC (μmol Trolox [Hoffman-LaRoche, Basel, Switzerland] equivalents (TE)/100 g) or nutrient content of age-specific portion sizes. Because antioxidants in coffee and tea have been shown to be poorly absorbed, we took into account absorption (6% for coffee and 4% for tea)[21] when calculating total antioxidant capacity of diet. The correlation between total antioxidant capacity of diet and plasma ORAC was 0.31. Food items contributing to total antioxidant capacity of diet, as compared with food records, showed reasonable good validity; for example, for fruit and vegetable consumption, r ranged from 0.4 to 0.7 for individual fruit and vegetable items; for tea consumption, r was 0.8; and for dietary fiber, r was 0.5 (Wolk A, unpublished data). The total antioxidant capacity from diet was adjusted for total energy intake using the residual method.[22]

Body mass index was calculated by dividing reported weight (kg) by reported height (m^2). Self-reported weight

and height is highly correlated with measured values in Swedish women ($r = 0.9$ and $r = 1.0$, respectively).[23] Physical activity levels were estimated by multiplying the reported duration of 5 predefined activities (occupation, housework, walking or cycling, leisure-time exercise, and inactive leisure time) by the intensity of these activities and expressed as multiples of the metabolic equivalent per day (kcal $kg^{-1} \times h^{-1}$) of sitting quietly for 1 hour. The validity of the reported total physical activity against activity records in these women were satisfactory ($r = 0.6$).[24]

Identification of Myocardial Infarction and Follow-up of the Cohort

All women were followed from September 1997 (baseline) through December 2007. Cases of nonfatal and fatal myocardial infarction (I21) were ascertained through linkage via the national registration number to the Swedish Hospital Discharge Registry and the Cause of Death Registry, which are considered nearly complete. The registries for 1987 and 1995 were thoroughly validated and revealed high sensitivity (94%) and positive predictive value (86%) for myocardial infarction.[25]

Statistical Analysis

All women were followed until the date of myocardial infarction, death, or the end of follow-up (December 31, 2007), whichever came first. Women were categorized into 4 categories of fruit and vegetable consumption, whole grain consumption, and coffee consumption, as well as quintiles of total antioxidant capacity of diet. Cox proportional hazards models with age as the time-scale were used to estimate hazard ratios (HR), with 95% confidence intervals (CI)[26] using the PHREG command in SAS (version 9.2; SAS Institute, Inc., Cary, NC). All HRs were adjusted for potential risk factors, including body mass index (<18.5, 18.5-24.9, 25-29.9, 30+ kg/m^2), smoking (never, past, current [≤10, >10 cigarettes/day]), alcohol consumption (gram/day, continuous), energy intake (calories/day, continuous), physical activity (metabolic equivalent-hours in quartiles), educational level (<10, 10-12, >12 years), hormone replacement therapy use (yes/no), aspirin use (yes/no), hypertension (yes/no), hypercholesterolemia (yes/no), family history of myocardial infarction (yes/no), and dietary supplement use (multivitamin use, use of other supplements than multivitamin, no supplements use). In additional analyses, we further adjusted for fruit and vegetable consumption (gram/day, continuous) as well as intakes of saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids (all continuous, gram/day). Missing data on a covariate was treated as a separate category. To assess trends across quintiles, we used the median value of each category to create a single continuous variable. A *P*-value for the linearity assumption between total antioxidant capacity and myocardial infarction was obtained by testing whether the quadratic term was equal to zero. The proportional hazards assumption was assessed by calculating scaled Schoenfeld residuals, and we did not find evidence of violation of this assumption.

We also corrected the HR of myocardial infarction per 4000 μmol/TE/day increment of ORAC (corresponding to approximately 1 standard deviation [SD] in the cohort) for bias due to dietary measurement error with the regression calibration method correcting for both random and systematic error.[27] Based on a validation study of 108 women from the Swedish Mammography Cohort,[17] we used the validity coefficient between total antioxidant capacity from diet and plasma ($r = 0.3$). All *P* values shown are 2-sided. *P*-values <.05 were considered statistically significant.

To evaluate whether the effect of total antioxidant capacity from diet varied by risk factors for myocardial infarction, we performed subgroup analyses by age (<65 years/≥65 years), body mass index (≤25/>25), smoking (nonsmokers/current smokers), and multivitamin supplement use (non-supplement users/multivitamin users). The likelihood ratio test was used to perform interaction tests. All *P* values shown are 2-sided. *P*-values <.05 were considered statistically significant.

RESULTS

During the average 9.9 years of follow-up (321,434 person-years), we identified 1114 cases of incident myocardial infarction (the average age of first myocardial infarction was 75.7 years). Baseline characteristics of the women are presented in **Table 1**. Women with higher total antioxidant capacity of diet were more likely to be nonsmokers, have ≥12 years of education, and to have hypercholesterolemia. Regarding dietary characteristics, women in the highest quintile of total antioxidant capacity of diet, as compared with the lowest quintile, had higher consumption of fruit and vegetables (3-fold), whole grains (15%), coffee (34%), and chocolate (38%), as well as 27% lower intake of saturated fatty acids and 19% lower intake of monounsaturated fatty acids. The major contributors to dietary total antioxidant capacity were fruit and vegetables (44%). The Pearson correlation coefficient between dietary total antioxidant capacity and fruit and vegetable consumption was 0.55. Other contributors were whole grains (18%), coffee (14%), and chocolate (4%).

The association between total antioxidant capacity of diet and incident myocardial infarction is presented in **Table 2**. In the multivariable-adjusted model, women in the highest quintile of total antioxidant capacity of diet, compared with the lowest quintile, had a 20% (95% CI, 3%-33%, *P* for trend = .02) lower risk of myocardial infarction. In sensitivity analyses, we evaluated whether the apparent inverse association with total antioxidant capacity of diet can be explained by consumption of fruit and vegetables by adding this variable (continuous, servings/day) to the model, and results did not substantially change (HR for women in the top quintile was 0.81; 95% CI, 0.64-1.02). When adjusting for intakes of saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids (all

Table 1 Age-standardized Background Characteristics of Women in the Swedish Mammography Cohort (n = 32,561)*

Characteristics	Quintiles of Total Antioxidant Capacity of Diet†				
	Q1 (n = 6512)	Q2 (n = 6512)	Q3 (n = 6512)	Q4 (n = 6512)	Q5 (n = 6513)
Median total antioxidant capacity of diet	8537	10,779	12,502	14,495	18,021
Non-dietary factors					
Age, mean (SD)	61.5 (9.5)	61.0 (9.0)	61.0 (8.9)	60.8 (8.8)	60.4 (8.7)
> 12 years of education, %	15.0	18.1	20.1	21.7	23.4
Current smokers, %	28.7	25.4	23.2	21.4	20.0
Body mass index, mean (SD)	25.1 (4.1)	24.9 (3.8)	24.9 (3.8)	25.7 (3.7)	25.6 (3.7)
Total physical activity score,‡ mean (SD)	42.0 (5.0)	42.4 (4.7)	42.6 (4.7)	42.6 (4.6)	43.0 (4.7)
Hypertension, %	19.0	18.4	18.1	17.7	18.9
Hypercholesterolemia, %	6.1	6.4	7.4	7.3	8.2
Family history of myocardial infarction, %	12.6	13.1	12.6	20.9	26.5
Aspirin use, %	43.1	44.9	43.5	41.3	41.9
Current and past users of hormone replacement therapy, %	61.4	58.1	58.3	61.4	59.4
Supplement users, %	56.7	57.9	60.0	60.1	62.3
Total energy, kcal/day (SD)	1770 (585)	1749 (510)	1746 (499)	1720 (490)	1738 (516)
Alcohol, gram/day (SD)	5.3 (12.1)	5.8 (10.2)	5.9 (7.7)	5.7 (7.8)	5.4 (9.1)
Foods, mean (SD)					
Fruits and vegetables, servings/day	2.4 (1.4)	3.4 (1.5)	4.0 (1.7)	4.9 (1.9)	6.8 (3.0)
Whole grains, servings/day	3.3 (2.0)	3.7 (2.0)	3.8 (2.0)	3.8 (2.0)	3.8 (2.0)
Coffee, servings/day	2.6 (1.4)	3.0 (1.6)	3.2 (1.7)	3.3 (1.8)	3.5 (2.0)
Tea, servings/week	5.1 (8.1)	5.0 (8.1)	4.6 (7.3)	4.5 (7.8)	4.5 (8.1)
Chocolate, servings/week	0.8 (0.9)	0.9 (1.1)	1.0 (1.2)	1.1 (1.5)	1.1 (1.8)
Nutrients, mean (SD)					
Saturated fatty acids, gram/day	31.4 (6.6)	29.2 (5.7)	27.5 (5.4)	25.8 (5.2)	23.0 (5.3)
Monounsaturated fatty acids, gram/day	21.8 (3.5)	20.0 (3.1)	20.2 (2.9)	19.3 (3.0)	17.6 (3.1)
Polyunsaturated fatty acids, gram/day	7.9 (2.1)	8.1 (1.8)	8.2 (1.9)	8.1 (1.8)	8.0 (1.8)

SD = standard deviation; ORAC = oxygen radical capacity absorbance.
*All variables except age are standardized to the age distribution of the cohort.
†μmol Trolox equivalents per day, as assessed with oxygen radical absorbance capacity assay.
‡Expressed as multiples of the metabolic equivalent per day (MET, kcal $kg^{-1} \times h^{-1}$) of sitting quietly for 1 hour.

continuous, gram/day), we observed a HR of 0.71 (95% CI, 0.58-0.87). The association was similar for nonfatal myocardial infarction (HR 0.78; 95% CI, 0.65-0.95) and fatal myocardial infarction (HR 0.76; 95% CI, 0.48-1.20).

Linear and quadratic relationships between total antioxidant capacity and myocardial infarction are presented in **Figure**. We found no significant evidence of departure from a constant change in the rate of myocardial infarction associated with every unit increase in total antioxidant capacity (P for nonlinearity = .25). The multivariable-adjusted HR of myocardial infarction for an increment of 4000 ORAC units/day (corresponding to approximately 1 SD in the study population) was 0.94 (95% CI, 0.88-1.00); the corrected HR (taking into account measurement error in the dietary total antioxidant capacity estimates) was 0.79. An increment of 4000 ORAC units is equivalent to approximately 1-2 apples or 2 peppers.

To investigate whether the observed association could be due to reversed causality, as cardiovascular risk factors may be associated with changes in dietary habits, we excluded cases that occurred in the first 3 years of follow-up. This sensitivity analysis did not indicate influence of reversed causality on the observed estimate; women in the highest as compared with the lowest quintile had HR = 0.81 (95% CI, 0.68-0.98).

In subgroup analysis, we examined whether the inverse association between dietary total antioxidant capacity and myocardial infarction varied by potential risk factors such as age, body mass index, smoking, saturated fatty acid intake, and multivitamin supplement use. The association was somewhat stronger among women aged >65 years (HR 0.74; 95% CI, 0.59-0.94), women with body mass index >25 (HR 0.76; 95% CI, 0.58-0.99), and current smokers (HR 0.66; 95% CI, 0.46-0.94), comparing the highest quintile with the lowest. However, there were no statistically significant interactions observed between dietary total antioxidant capacity and the above risk factors (all P for interaction >.16).

DISCUSSION

In this large prospective population-based cohort study, we observed that higher total antioxidant capacity of diet was statistically significantly associated with lower risk of inci-

Table 2 Antioxidant-containing Foods* and Total Antioxidant Capacity of Diet† in Relation to Risk of Myocardial Infarction among 32,561 Women

	Categories					*P* for Trend
Fruit and vegetables, servings/day	≤2	3	4	≥5		
No. of cases	244	224	227	419		
Person-years	44,225	57,067	65,022	155,121		
Age-adjusted HR	1.00	0.90 (0.75-1.08)	0.88 (0.73-1.05)	0.75 (0.64-0.88)		<.001
Multivariable HR‡	1.00	0.95 (0.79-1.15)	0.95 (0.79-1.14)	0.86 (0.73-1.03)		.09
Whole grains, servings/day	≤2.3	2.3-3.4	3.4-4.7	≥4.7		
No. of cases	298	260	273	283		
Person-years	83,269	77,995	81,401	78,769		
Age-adjusted HR	1.00	0.90 (0.76-1.06)	0.82 (0.69-0.97)	0.81 (0.68-0.95)		.008
Multivariable HR‡	1.00	0.95 (0.81-1.13)	0.88 (0.74-1.04)	0.89 (0.74-1.07)		.18
Coffee, servings/day	0	<2	2-4	>4		
No. of cases	72	408	455	179		
Person-years	18,257	113,592	133,553	56,032		
Age-adjusted HR	1.00	0.87 (0.68-1.12)	0.93 (0.72-1.19)	1.13 (0.86-1.48)		.05
Multivariable HR‡	1.00	0.87 (0.68-1.12)	0.88 (0.69-1.13)	0.96 (0.72-1.26)		.83
Total antioxidant capacity of diet*, quintiles	1	2	3	4	5	
Median ORAC, μmol Trolox equivalents/day	8537	10,779	12,502	14,495	18,021	
No. of cases	288	231	203	201	191	
Person-years	63,259	64,366	64,385	64,534	64,890	
Age-adjusted HR	1.00	0.85 (0.71-1.01)	0.75 (0.63-0.90)	0.76 (0.63-0.91)	0.74 (0.62-0.89)	.001
Multivariable HR‡	1.00	0.88 (0.74-1.05)	0.80 (0.66-0.95)	0.83 (0.69-0.99)	0.80 (0.67-0.97)	.02

Hazard ratios (HR) with 95% confidence intervals were obtained from Cox proportional hazards models.
*Foods contributing ≥10% of total antioxidant capacity of diet.
†μmol Trolox equivalents per day.
‡Adjusted for age, education, smoking, body mass index, physical activity, hypertension, hypercholesterolemia, family history of myocardial infarction, aspirin use, hormone replacement therapy use, dietary supplement use, and intakes of total energy and alcohol.

dent myocardial infarction in a dose-response manner. Consumption of antioxidant-containing foods such as fruits and vegetables and whole grains were nonsignificantly inversely associated with the risk of myocardial infarction.

To our knowledge, this is the first study investigating dietary total antioxidant capacity in relation to myocardial infarction. The single total antioxidant capacity estimate is assumed to give a better measure of all antioxidants than sum of individual antioxidants, because it also reflects the synergism between the compounds. Moreover, we have previously observed in our study population that total antioxidant capacity of diet was inversely associated with incidence of total stroke.[28] Several foods that are major contributors to antioxidant intake in our study population have been linked to a decreased risk of coronary heart disease. In particular, high intake of fruit and vegetables, which contributed 44% of the dietary total antioxidant capacity in our study, have been inversely related to coronary heart disease in many studies.[9] Whole grains (18% of total antioxidant capacity) also are suggested to lower coronary heart disease risk.[29,30] Coffee consumption (14% of total antioxidant capacity) has been inversely related to coronary heart disease in some but not in all studies.[31] Chocolate consumption (4% of total antioxidant capacity) has been shown to have favorable effects on cardiovascular risk biomarkers such as flow-mediated dilation and diastolic blood pressure, as shown by a meta-analysis of randomized controlled trials.[32]

The pathogenesis of atherosclerosis involves oxidation of low-density lipoproteins caused by reactive oxygen species.[1] Antioxidants found in diet are thought to protect against coronary heart disease by scavenging reactive oxygen species.[1] In addition to antioxidant effects, flavonoids also may inhibit the atherosclerotic process through other pathways. Flavonoids have been shown to improve endothelial function, to decrease blood pressure, and to have antiplatelet and anti-inflammatory effects.[33]

Previous randomized controlled trials testing high doses of antioxidant supplements containing 1 to 3 compounds have failed to see any benefit on coronary heart disease.[10-15] One randomized controlled trial that studied the effect of a low-dose mixture of 5 antioxidant supplements (including 120 mg ascorbic acid, 30 mg vitamin E, 6 mg beta carotene, 100 μg selenium, and 20 mg zinc) did not observe any association with ischemic cardiovascular diseases.[34] Notably, in a meta-analysis of high doses and very high doses of single supplements of vitamin A, β-carotene, or vitamin E tested in several randomized controlled trials, higher all-cause mortality was reported.[35] In contrast to supplements

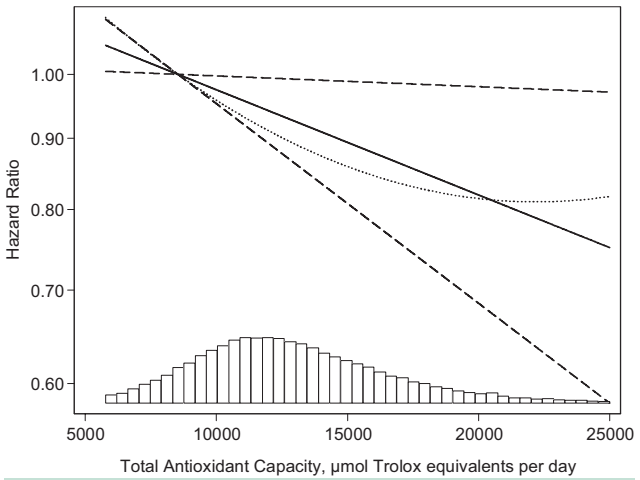

Figure Multivariable-adjusted hazard ratios for incident myocardial infarction according to dietary total antioxidant capacity, based on the oxygen radical absorbance capacity assay (μmol Trolox equivalents). Hazard ratios were adjusted for age, education, smoking, body mass index, physical activity, hypertension, hypercholesterolemia, family history of myocardial infarction, aspirin use, hormone replacement therapy use, dietary supplement use, and intakes of total energy and alcohol. Solid curve represents point estimates of the linear trend. Dotted line represents point estimates of the quadratic trend. Dashed lines represent 95% confidence intervals for the linear trend. The distribution of total antioxidant capacity is presented at the bottom of the figure as a histogram.

of single antioxidants, the dietary total antioxidant capacity reflects all present antioxidants, including thousands of compounds, all of them in doses present in our usual diet, and even takes into account their synergistic effects. Moreover, this study included women who were healthy at study entry, while the majority of the randomized controlled trials were performed among participants with established atherosclerosis who may not benefit from antioxidant supplementation.

Our study has several strengths, including the prospective population-based design, detailed data on diet and potential risk factors, and the practically complete follow-up of the study cohort.

There are, however, some potential limitations with our study. The total antioxidant capacity has not been measured for Swedish foods; we used American ORAC values. Antioxidant content may vary with geographic location and growing conditions. The observed association could be explained by consumption of fruit and vegetables; however, after this adjustment the association between total antioxidant capacity of diet and myocardial infarction remained statistically significant. We did not have ORAC values for dietary supplements, thus, our results can only be translated to food items and not to antioxidant supplements. Measurement error in self-reported dietary intake may lead to misclassification of exposure. Because of the prospective design, the misclassification is most likely to be nondifferential and therefore lead to attenuation of the observed association. Moreover, when we corrected for measurement error, an even stronger association was observed. We only estimated total antioxidant capacity of diet, which does not include antioxidant capacity of endogenous antioxidants. In our validation study, we observed a somewhat weak correlation between total antioxidant capacity from diet and plasma,[17] which can be partly explained by the fact that plasma total antioxidant capacity values are influenced by many factors, such as endogenous antioxidants, homeostatic control mechanisms of plasma antioxidants, absorption, and the extent of the metabolism of dietary antioxidants.[36] The study was performed among women and cannot, therefore, be generalized to men. Furthermore, the observed inverse association could be due to a phenomenon in which women who eat an antioxidant-rich diet are more health conscious and have other healthy behaviors. When adjusting for several potential confounders such as smoking, physical activity, and educational level, the results remained statistically significant; however, we cannot rule out residual confounding by healthy lifestyle factors. Intervention studies would be needed to definitively determine the health effects of total antioxidant capacity.

In conclusion, our data suggest that a diet high in total antioxidant capacity is associated with lower risk of incident myocardial infarction. The major contributors to dietary total antioxidant capacity were fruits, vegetables, coffee, and whole grains.

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