Life-Style Factors and Risk for Abdominal Aortic Aneurysm in a Cohort of Finnish Male Smokers

Markareetta E. Törnwall,[1] *Jarmo Virtamo,*[1] *Jari K. Haukka,*[1] *Demetrius Albanes,*[2] *and Jussi K. Huttunen*[1]

Prospective studies evaluating risk factors for abdominal aortic aneurysm are few. We studied the association of life-style factors with risk for abdominal aortic aneurysm among 29,133 male smokers 50–69 years of age, participants in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. During a mean follow-up of 5.8 years, 181 were diagnosed with ruptured abdominal aortic aneurysm or nonruptured abdominal aortic aneurysm plus aneurysmectomy. Risk for abdominal aortic aneurysm was positively associated with age [relative risk (RR) = 4.56, 95% confidence interval (CI) = 2.42–8.61 for >65 *vs* ≤55 years], smoking years (RR = 2.25, 95% CI = 1.33–3.81 for >40 *vs* ≤32 years), systolic blood pressure (RR = 1.92, 95% CI = 1.13–3.25 for >160 *vs* ≤130 mmHg), diastolic blood pressure (RR = 1.80, 95% CI = 1.05–3.08 for >100 *vs* ≤85 mmHg), and serum total cholesterol (RR = 1.85, 95% CI = 1.09–3.12 for >6.5 *vs* ≤5.0 mmol/liter). High-density lipoprotein cholesterol showed a strong inverse association with risk for aortic aneurysm (RR = 0.16, 95% CI = 0.08–0.32 for >1.5 *vs* ≤ 0.9 mmol/liter). High energy intake was associated with lower risk for aortic aneurysm (RR = 0.59, 95% CI = 0.38–0.94 for the highest quartile *vs* the lowest), whereas no associations with nutrients were evident. We conclude that classical risk factors for atherosclerotic diseases seem to be important in pathogenesis of large abdominal aortic aneurysms. (Epidemiology 2001;12:94–100)

Keywords: abdominal aortic aneurysm, cohort study, smoking, blood pressure, serum cholesterol, HDL cholesterol, diet.

Abdominal aortic aneurysm is an atherosclerotic disease that seldom shows any symptoms before a life-threatening event of rupture. Screening studies of Western populations have revealed a prevalence of abdominal aortic aneurysm between 4.1% and 8.8% among men over 55 years of age.[1–5] Since the 1950s–1980s, the incidence of abdominal aortic aneurysm has been reported to have increased two- to sevenfold in Western populations.[6–8] On the other hand, a report from Australia indicated a 12% decline in annual incidence for ruptured abdominal aortic aneurysm and for elective surgery in the 1990s.[9] Observations of mortality have been somewhat contradictory, indicating an increase in age-standardized mortality for ruptured abdominal aortic aneurysm in the United Kingdom, Australia, and Sweden,[10–12] and an unchanged mortality rate in Canada and the United States.[13,14]

Risk factors associated with abdominal aortic aneurysm are age, smoking, hypertension, hypercholesterolemia, and male sex, with a male-to-female ratio of 6 to 1.[1,2,9,15–18] The atherosclerotic etiology of abdominal aortic aneurysm has been challenged lately. Genetic and environmental factors affecting production and breakdown of elastin and collagen seem to be important.[19,20] The destruction of elastin unavoidably exposes collagen to a greater amount of biomechanical stress and thus increases risk for rupture.[19] The chronic inflammation present in aneurysms[19,20] is also considered an important contributor to atherosclerosis.[21]

Only a few cohort studies of risk factors have appeared, and evidence linking diet and abdominal aortic aneurysm is practically absent. The aim of this study was to explore the effect of life-style factors on risk for large abdominal aortic aneurysm in a cohort of Finnish male smokers, participants in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention (ATBC) Study.

Subjects and Methods

The ATBC Study was a randomized, double-blind, placebo-controlled trial with a two-by-two factorial design primarily aimed at examining the effect of antioxidant supplementation on cancer.[22] The participants were recruited by postal questionnaire from among the total population of men 50–69 years of age living in

From the [1]Kansanterveyslaitos (National Public Health Institute), Helsinki, Finland, and [2]The National Cancer Institute, Bethesda, MD.

Address correspondence to: Markareetta E. Törnwall, ATBC Study, Department of Nutrition, KTL (National Public Health Institute), Mannerheimintie 166, 00300 Helsinki, Finland.

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southwestern Finland (N = 290,406). The enrollment took place from 1985 through 1988, and the study lasted until spring 1993. Men who were current smokers (at least five cigarettes per day) and willing to participate in the study (N = 42,957) were invited to undergo baseline examinations. Subjects with previous cancer; any serious disease limiting long-term participation; use of anticoagulants; or use of vitamin E (more than 20 mg per day), beta-carotene (more than 6 mg per day), or vitamin A (more than 20,000 international units per day) supplements were excluded. The final number of participants in the ATBC Study was 29,133. The men were randomly assigned into four intervention groups: 50 mg of alpha-tocopherol (DL-alpha-tocopheryl acetate) or 20 mg of beta-carotene or both or placebo, in one capsule per day. All subjects provided written informed consent before randomization. The institutional review boards of the National Public Health Institute (Finland) and the National Cancer Institute (United States) approved the study. A data and safety monitoring committee was convened twice annually throughout the study to evaluate unblinded data relevant to safety and efficacy.

At baseline, background information about medical history, physical activity, education, and smoking habits was collected through questionnaires. Blood pressure, height, and weight were measured, and a blood sample was drawn and serum stored at −70°C. Serum total and high-density lipoprotein (HDL) cholesterol concentrations were determined enzymatically—cholesterol oxidase-4-aminophenazone (CHOD-PAP) method, Boehringer Mannheim, Mannheim, Germany.[23] HDL cholesterol was measured after precipitation with dextran sulfate and magnesium chloride.[24] Serum alpha-tocopherol and beta-carotene concentrations were measured by high-performance liquid chromatography.[25]

The usual diet, including alcohol consumption over the previous 12 months, was assessed with a self-administered diet-history questionnaire at study entry. This questionnaire was satisfactorily completed by 93% of study subjects (N = 27,111). The questionnaire covered the consumption of about 200 food items and 70 mixed dishes and included a picture booklet with color photographs of different portion sizes. Nutrient intakes were computed by use of the database of the National Public Health Institute.

The endpoint of this study was abdominal aortic aneurysm, either nonruptured, which was electively or urgently operated on, or ruptured. Information on aortic aneurysm came from the National Register of Causes of Death and also from the National Hospital Discharge Register, in which operation codes are recorded. Both registers use the codes of the *International Classification of Diseases* (ICD). We searched for ICD-8 codes 44100–44199 (used until 1986), ICD-9 codes 4410A–4419X (used after 1986), and operation codes for aneurysmectomy with graft placement. Altogether, we found 202 cases. We collected hospital and autopsy records to identify those with ruptured infrarenal abdominal aortic aneurysm or nonruptured infrarenal abdominal aortic aneurysm with prosthetic graft placement. Of the 202 cases, 181 fulfilled these criteria. The use of registers made it possible to obtain information both from active participants and from those who had dropped out.

Risk factors were divided into clinically relevant categories: age in 5-year age groups, systolic (≤130, 131–160, and >160 mmHg) and diastolic (≤ 85, 86–100, >100 mmHg) blood pressure, serum total (≤5.0, >5.0–6.5, >6.5 mmol/liter) and HDL (≤0.9, >0.9–1.5, >1.5 mmol/liter) cholesterol, body mass index (≤25, >25–27, >27 kg/m^2), years of smoking (≤32, >32–40, >40), and number of cigarettes smoked daily (≤14, 15–24, >24). Three risk factors were dichotomized: history of diabetes mellitus, education (elementary school *vs* junior high school or more), and leisure-time exercise (no exercise *vs* slight to moderate exercise). We calculated crude rates per 10,000 person-years for all categories of each factor. We used the Cox proportional-hazards model to estimate relative risks. First, we adjusted each risk factor by age and smoking years, and secondly, a multivariate model was created including all of the above-mentioned factors and alpha-tocopherol and beta-carotene supplementation as main effects (alpha-tocopherol supplementation *vs* no alpha-tocopherol supplementation; beta-carotene supplementation *vs* no beta-carotene supplementation). Relative risks from only the multivariate model are reported, as the results were similar for age- and smoking-adjusted models and the multivariate model. This analysis also accounts for the effects of nutrients. Trends of association of the major findings are presented in graphs produced by locally weighted regression (loess), a nonparametric smoothing technique.[26]

The intake of nutrients (except alcohol intake) were log transformed and energy corrected by the residual method[27] and divided into quartiles, with the lowest quartile as the reference group. Alcohol intake was divided into five categories: no intake and ≤15, 16–30, 31–60, >60 gm per day. Crude rates per 10,000 person-years are presented for all categories of each nutrient and for alcohol. We used the Cox proportional-hazards model to estimate relative risks for abdominal aortic aneurysm associated with energy-adjusted intakes of the nutrients with simultaneous adjustment for age, years of smoking, and intake of energy, and secondly for blood pressure, serum total and HDL cholesterol, and supplementation group. Each nutrient was added to the model separately. In the analyses of nutrients, 169 incident cases of abdominal aortic aneurysm had completed diet-history questionnaire at baseline.

In addition, we assessed association between baseline serum alpha-tocopherol and beta-carotene concentrations and risk for abdominal aortic aneurysm by the Cox model. Serum levels were first adjusted for age, smoking habits, and total and HDL cholesterol, and secondly, a multivariate model was created including the other risk factors and alpha-tocopherol and beta-carotene supplementation groups.

TABLE 1. Medians and Interquartile Range of Baseline Variables: the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study Cohort. (N = 29,133)

Baseline Variable	Median	Interquartile Range
Age, years	57	53–61
Cigarettes/day	20	15–25
Years of smoking	36	31–42
Body mass index, kg/m^2	26.0	23.7–28.5
Systolic blood pressure, mmHg	140	128–154
Diastolic blood pressure, mmHg	88	80–94
Serum total cholesterol, mmol/liter	6.15	5.44–6.94
Serum HDL cholesterol, mmol/liter	1.12	0.95–1.33
Serum alpha-tocopherol, mg/liter	11.5	9.8–13.6
Serum beta-carotene, μg/liter	170	109–261
Total energy intake, kcal/day	2,720	2,254–3,262
Alcohol consumption, gm/day	11	3–26
History of diabetes mellitus, %	4	
Education beyond elementary school, %	15	
Exercise performed in leisure time, %	48	

HDL = high-density lipoprotein.

Results

The median age of men in this cohort was 57 years. On average, they smoked 20 cigarettes per day and had smoked 36 years at study entry (Table 1). During a mean follow-up of 5.8 years, 181 men experienced rupture of an abdominal aortic aneurysm (N = 77) or underwent elective aneurysmectomy and graft placement (N = 104). The median width of ruptured aneurysms was 7 cm, and that of nonruptured aneurysms was 6 cm at the time of surgery.

Relative risk for abdominal aortic aneurysm increased with increasing age, showing a clear positive association [multiadjusted relative risk = 0.56, 95% confidence interval (CI) = 2.42–8.61, among those >65 years of age compared with those ≤55 years of age] (Table 2). Years of smoking were positively associated with risk for aortic aneurysm. The relative risk was 2.25 (95% CI = 1.33–3.81) among those who had smoked >40 years compared with those who had smoked ≤32 years. The number of cigarettes smoked daily showed no clear association with risk for aortic aneurysm. High blood pressure was associated with increased risk for aortic aneurysm. Relative risk was 1.92 (95% CI = 1.13–3.25) among those with systolic blood pressure >160 mmHg compared with those with ≤130 mmHg. Accordingly, relative risk was 1.80 (95% CI = 1.05–3.08) among those with diastolic blood pressure >100 mmHg compared with ≤85 mmHg. Serum total cholesterol showed a moderate positive association with risk for aortic aneurysm. The relative risk was 1.85 (95% CI = 1.09–3.12) among those with serum total cholesterol >6.5 mmol/liter compared with those with ≤5.0 mmol/liter. HDL cholesterol showed a strong inverse association;

TABLE 2. Crude Rates per 10,000 Person-Years, Relative Risks (RR), and 95% Confidence Intervals (CI) of Life-Style Factors for Abdominal Aortic Aneurysm

Risk Factor	Cutoff Points	Cases	Person-Years	Crude Rate*	RR†	95% CI
Age, years	≤55‡	21	61,543	3.4	1.00	
	>55–60	46	55,038	8.4	1.81	1.05–3.14
	>60–65	67	36,483	18.4	3.15	1.76–5.65
	>65	47	16,344	28.8	4.60	2.44–8.69
Cigarettes/day	≤14‡	43	34,271	12.5	1.00	
	15–24	94	80,742	11.6	1.01	0.70–1.46
	>24	44	54,395	8.1	0.81	0.52–1.27
Years of smoking	≤32‡	26	58,359	4.5	1.00	
	>32–40	53	64,998	8.2	1.45	0.88–2.39
	>40	100	45,691	21.9	2.25	1.33–3.81
Body mass index, kg/m^2	≤25‡	56	56,227	10.0	1.00	
	>25–27	60	56,272	10.7	0.84	0.57–1.26
	>27	65	56,793	11.4	0.78	0.55–1.11
Systolic BP, mmHg	≤130‡	33	53,700	6.1	1.00	
	131–160	96	90,186	10.6	1.34	0.87–2.07
	>160	52	25,494	20.4	1.95	1.15–3.30
Diastolic BP, mmHg	≤85‡	56	70,592	7.9	1.00	
	86–100	97	82,123	11.8	1.35	0.93–1.94
	>100	28	16,664	16.8	1.78	1.03–3.05
Total cholesterol, mmol/liter	≤5.0‡	18	22,068	8.2	1.00	
	>5.0–6.5	78	82,095	9.5	1.30	0.77–2.20
	>6.5	84	65,052	12.9	1.81	1.07–3.05
HDL cholesterol, mmol/liter	≤0.9‡	62	29,483	21.0	1.00	
	>0.9–1.5	109	116,182	9.4	0.40	0.29–0.56
	>1.5	9	23,524	3.8	0.15	0.07–0.31
History of diabetes mellitus	No‡	177	162,541	10.9	1.00	
	Yes	4	6,867	5.8	0.43	0.16–1.15
Basic education	Elementary school‡	155	142,900	10.8	1.00	
	Additional education	26	26,508	9.8	1.01	0.67–1.54
Exercise in leisure time	Yes‡	84	81,648	10.3	1.00	
	No	97	87,527	11.1	1.29	0.95–1.73

* Per 10,000 person-years.
† Multivariate model with simultaneous adjustment for all the risk factors and, additionally, alpha-tocopherol and beta-carotene supplementation group by Cox proportional-hazards model.
‡ Reference category.
BP = blood pressure.
HDL = high-density lipoprotein.

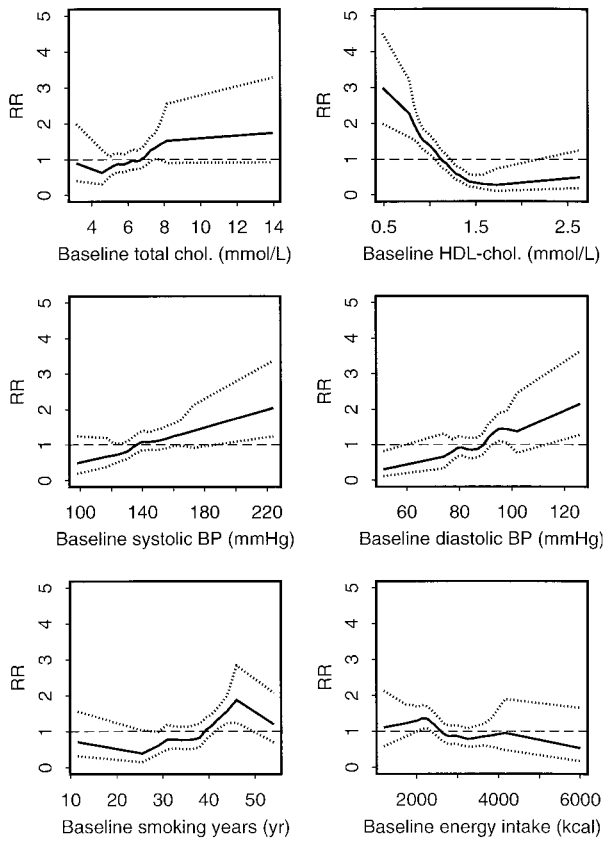

FIGURE 1. Age-adjusted smoothed slope estimates (solid line) and two standard errors (dotted line) for the relation of six risk factors and risk for abdominal aortic aneurysm.

relative risk was 0.16 (95% CI = 0.08–0.32) among those with serum HDL cholesterol >1.5 mmol/liter compared with those with ≤0.9 mmol/liter. The association was of similar magnitude whether HDL cholesterol was modeled alone or was modeled simultaneously with all other risk factors, including alcohol consumption and physical activity. For the major risk factors, age-adjusted smoothed slope estimates of relative risk with two standard errors are presented in Figure 1.

The association between major risk factors and risk for abdominal aortic aneurysm was also studied within age strata (Table 3). Risk increased with increasing years of smoking, systolic blood pressure, and serum total cholesterol only among those 60 years of age or less, whereas it increased with increasing diastolic blood pressure among those over 60 years of age. The decrease in risk for aneurysm with increasing serum HDL cholesterol was not dependent on age.

Intake of total energy was inversely associated with risk for abdominal aortic aneurysm. Multiadjusted relative risk was 0.59 (95% CI = 0.38–0.94) for the highest quartile of intake compared with the lowest. The association between energy intake and risk for aortic aneurysm remained similar when energy derived from alcohol was excluded (data not shown). Intake of macronutrients (fat, carbohydrates, protein, and fiber) was not associated with risk for aortic aneurysm (Table 4). With consumption of alcohol, no clear trend in relative risks was evident. Additionally, we evaluated the association for the different fatty acid groups (saturated fat, polyunsaturated fat, and *trans-* and *cis-*monounsaturated fat) and found no influence on risk. In the analysis of dietary

TABLE 3. Crude Rates and Relative Risks (RR) of Major Risk Factors for Abdominal Aortic Aneurysm by Age Category

	Age							
	<60 Years				>60 Years			
	Cases	Crude Rate*	RR†	95% CI	Cases	Crude rate*	RR†	95% CI
Years of smoking								
≤32‡	15	2.9	1.00		11	18.6	1.00	
33–40	33	6.3	2.05	1.11–3.80	20	16.1	0.94	0.44–2.01
>40	18	15.9	5.21	2.60–10.42	82	23.9	1.42	0.73–2.76
Systolic BP, mmHg								
≤130‡	13	3.2	1.00		20	15.4	1.00	
131–160	39	6.4	1.84	0.90–3.75	57	19.7	1.14	0.66–1.97
>160	15	10.3	2.91	1.17–7.22	37	33.8	1.72	0.90–3.27
Diastolic BP, mmHg								
≤85‡	17	3.6	1.00		39	16.4	1.00	
86–100	40	7.0	1.39	0.74–2.64	57	23.0	1.25	0.79–2.00
>100	10	8.1	1.24	0.49–3.19	18	42.2	2.11	1.09–4.08
Total cholesterol, mmol/liter								
≤5.0‡	3	2.1	1.00		15	20.0	1.00	
>5.0–6.5	21	3.7	1.82	0.54–6.09	57	22.0	1.18	0.66–2.13
>6.5	43	9.4	4.38	1.35–14.14	41	21.1	1.17	0.64–2.15
HDL cholesterol, mmol/liter								
≤0.9‡	24	11.9	1.00		38	41.1	1.00	
>0.9–1.5	40	5.0	0.39	0.23–0.66	69	19.0	0.41	0.27–0.62
>1.5	3	1.8	0.13	0.04–0.44	6	8.4	0.17	0.07–0.41

* Crude rate per 1,000 person-years.
† Multivariate model adjusted for all life-style factors (smoking years, number of cigarettes per day, systolic and diastolic blood pressure, serum total and high-density lipoprotein cholesterol, body mass index, education, exercise, and history of diabetes mellitus) and, additionally, alpha-tocopherol and beta-carotene supplementation group by Cox proportional hazards model.
‡ Reference category.
BP = blood pressure.
HDL = high-density lipoprotein.

TABLE 4. Crude Rates, Relative Risks (RR), and 95% Confidence Intervals (CI) for Abdominal Aortic Aneurysm by Quartiles of Daily Intake of Energy, Macronutrients, and Alcohol

Macronutrient	Quartile Medians	Cases	Person-Years	Crude Rate*	Multivariate RR†	95% CI
Total energy, kcal	1,984‡	61	39,135	15.6	1.00	
	2,495	48	39,291	12.2	0.92	0.63–1.35
	2,969	32	39,795	8.0	0.63	0.41–0.93
	3,695	28	39,846	7.0	0.59	0.38–0.94
Fat, gm	80‡	42	39,095	10.7	1.00	
	106	45	39,705	11.3	1.14	0.74–1.74
	130	38	39,658	9.6	0.92	0.59–1.44
	169	44	39,610	11.1	0.95	0.62–1.46
Protein, gm	72‡	44	39,563	11.1	1.00	
	91	37	39,741	9.3	0.82	0.53–1.27
	109	37	39,628	9.3	0.87	0.56–1.35
	136	51	39,135	13.0	1.13	0.75–1.70
Carbohydrates, gm	203‡	44	39,619	11.1	1.00	
	266	32	39,665	8.1	0.71	0.45–1.13
	322	38	39,686	9.6	0.78	0.50–1.21
	411	55	39,098	14.1	1.02	0.68–1.54
Fiber, gm	15‡	46	39,347	11.7	1.00	
	21	40	39,619	10.1	0.83	0.54–1.27
	27	36	39,816	9.0	0.74	0.48–1.15
	37	47	39,286	12.0	0.90	0.59–1.36
Alcohol, gm§	0‡	30	17,343	17.3	1.00	
	5	84	74,604	11.3	0.81	0.53–1.24
	23	27	35,155	7.7	0.70	0.41–1.19
	40	22	23,611	9.3	0.91	0.51–1.63
	78	6	7,354	8.2	0.93	0.38–2.29

* Crude rate per 10,000 person-years. Macronutrients except alcohol were log-transformed and energy-corrected by the residual method.
† Adjusted for age, years of smoking, total energy intake, serum total and high-density lipoprotein cholesterol, systolic and diastolic blood pressure, and alpha-tocopherol and beta-carotene supplementation group.
‡ Reference category.
§ Intake of alcohol was divided in five categories: nondrinkers and ≤15, 16–30, 31–60, and >60 gm per day.

antioxidants (vitamins C and E, carotenoids, and selenium), no association was evident (data on fatty acid groups and antioxidants not shown). Baseline serum levels of neither alpha-tocopherol nor beta-carotene were associated with risk for aortic aneurysm (data not shown).

Discussion

This large cohort study of male smokers revealed several risk factors for abdominal aortic aneurysm. Age showed a strong positive association. Risk for abdominal aortic aneurysm was 4.5 times as high among men who were over 65 years of age as it was among those 55 years of age or less. Results on smoking habits indicate that long history of smoking is an independent risk factor, but number of cigarettes smoked per day does not play an important role in risk for abdominal aortic aneurysm among such long-term smokers. Elevated blood pressure and high serum total cholesterol were independent risk factors. Serum HDL cholesterol showed a strong inverse association, with an 84% risk reduction associated with high HDL cholesterol.

Similar associations have been found in previous prospective studies. In the Whitehall study, among 18,000 male civil servants 41 fatal abdominal aortic aneurysms were detected,[16] and in the Honolulu Heart Program 151 aortic cases were diagnosed mostly by surgical and autopsy records from among more than 8,000 men.[15] The oldest cohort included both genders and reported 519 fatal aortic aneurysms among 800,000 subjects.[28] Smoking and hypertension were consistently associated with risk for aortic aneurysm.[15,16,28] In the Whitehall study, history of smoking showed a strong association with risk for abdominal aortic aneurysm, but increasing number of cigarettes smoked was not much associated with increasing risk, a finding similar to ours. Serum total cholesterol was related with risk for aortic aneurysm in the Honolulu Heart Program and in our data, whereas in the Whitehall study it was not related.[15,16] Serum HDL cholesterol was not assessed in previous prospective studies. Physical activity has shown an inverse or no association with risk for aortic aneurysm.[16,28] Height and relative weight have been positively associated with risk for aortic aneurysm.[16,28] In contrast, we found little association between body mass index and risk. Diabetes mellitus had little association with risk for aortic aneurysm in our study or in previous cohorts.[15,16]

Smoking and hypertension have also been associated with risk for abdominal aortic aneurysm in case-control studies.[17,18,29,30] The association between serum lipids and abdominal aortic aneurysm has been less consistent.[17,18,29,31]

None of the nutrients was associated with risk for abdominal aortic aneurysm in our study. This observation is not unique: in a cohort study from Hawaii of men with Japanese ancestry, nutrient intake (measured in a 24-hour recall) was not markedly associated with aortic aneurysm.[16] The study populations and measurement of nutrient intakes in these two studies were very different, however, and no details of dietary results appeared in the Honolulu Heart Program report. Intake of energy was inversely associated with risk for abdominal aortic aneurysm in our study, and some cohort studies of diet and

ischemic heart disease have reported a similar association.[32,33] This association might be explained by exercise, because a high amount of exercise is associated with greater need for energy, and thus high energy intake may be an indicator of better health and health awareness. Level of leisure-time exercise was only weakly associated with risk for abdominal aortic aneurysm in our data; nevertheless, our finding of the strong protective effect of a high level of HDL cholesterol gives some support for this explanation, because physical exercise is known to increase HDL cholesterol.[34]

In this study, 1.2% of men over 60 years of age at baseline were diagnosed with large abdominal aortic aneurysms. In previous screening studies, an abdominal aortic aneurysm was found in some 2% of men 60–75 years of age.[5,35] The sizes of the aneurysms were from 4 to 6 cm, whereas in our data the mean diameter was more than 6 cm, as our endpoint was a ruptured aneurysm or a large aneurysm that was prone to rupture and electively operated on. In the Rotterdam study, the prevalence of abdominal aortic aneurysms more than 5 cm in diameter was 0.8% in men over 55 years of age.[1] No screening for aortic aneurysm was performed in the ATBC Study. Case collection was based on registers, and we evaluated all hospital and autopsy records to exclude false-positive cases. In Finland all operations performed in hospitals are recorded in the National Hospital Discharge Register. The number of subjects with asymptomatic and uncomplicated abdominal aortic aneurysm in our data remains unknown. Nevertheless, we included in our analysis both ruptured and electively operated aneurysms. Thus most subjects with large and clinically relevant abdominal aortic aneurysms were probably included in the analysis. Sudden death cases that were not autopsied may, however, be a potential source of lost cases, although autopsies were performed on 64% of the ATBC Study participants who had cardiovascular disease (ICD-9 codes 390–459) as their underlying cause of death.

In assessment of nutrient intake, some degree of measurement error cannot be avoided. The reproducibility and validity of the questionnaire used in this study were both found to be satisfactory in a previous pilot study among 190 men.[36] The lack of association between nutrient intake and risk for abdominal aortic aneurysm is unlikely to be due to weaknesses of the dietary measurement. Thus, our results indicate that diet has little association with risk for abdominal aortic aneurysm. The lack of association between baseline serum levels of alpha-tocopherol and beta-carotene and risk for abdominal aortic aneurysm is in concordance with our finding of little association with antioxidant intake.

To conclude, aging, long history of smoking, hypertension, hypercholesterolemia, and especially low HDL cholesterol were associated with increased risk for abdominal aortic aneurysm among older male smokers. All of these factors are important risk factors for coronary heart disease and stroke, indicating that atherosclerosis contributes to the pathogenesis of abdominal aortic aneurysm. Nonsmoking and better control of blood pressure and serum cholesterol levels may thus be effective in long-term prevention of abdominal aortic aneurysm.

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