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Cardiovascular Disease Prevalence and its Relation to Risk Factors in Alaska Eskimos

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Abstract

Background and Aims—Although Eskimos were thought to be protected from cardiovascular disease (CVD), state health data show a large proportion of deaths from CVD, despite traditional lifestyles and high omega-3 fatty acid intake. This article explores CVD prevalence and its relation to risk factors in Alaska Eskimos.

Methods and Results—A population-based cohort of 499 Alaska Eskimos > age 45 from the Norton Sound region was examined in 2000-2004 for CVD and associated risk factors as part of the Genetics of Coronary Artery Disease in Alaska Natives study. CVD and atherosclerosis were evaluated and adjudicated using standardized methods. Average age was 58y; diabetes prevalence was low and high-density lipoprotein cholesterol (HDL-C) concentrations were high, but a large proportion smoked and had high pathogen burden. CVD was higher in men (12.6%) than in women (5.3%) (prevalence ratio 2.4, CI 1.3-4.4). Rates of stroke (6.1% in men, 1.8% in women) were similar to those for coronary heart disease (CHD) (6.1% men, 2.5% women). MI prevalence was low in both genders (1.9% and 0.7%). CVD was higher in men and in those >60 yrs. Hypertension, diabetes, high LDL-C, high apoB, and low HDL-C were all strong correlates (<.002) and albuminuria and CRP were also correlated with CVD (p<.05) after adjustment for age and gender. Carotid atherosclerosis was correlated with CVD (p=.0079) independent of other risk factors.

Conclusion—These data show high CHD and stroke prevalence in Alaska Eskimos, despite low average LDL-C and high HDL-C. Hypertension and high LDL-C were independent correlates; identifying these risk factors early and treating to target is recommended.

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Keywords

cardiovascular disease; risk factors; epidemiology; omega-3 fatty acid

Introduction

Alaska Eskimos, like many other populations, have undergone rapid changes in lifestyle during the past century. In 1975, Dyerberg et al. surveyed Greenland Eskimos,[1] found low rates of reported cardiovascular disease (CVD), and attributed the low rates to this population's regular consumption of marine mammals containing omega-3 fatty acids; this report led to the assumption that Alaska Eskimos were protected from CVD. As state health data[2,3] accumulated, however, it was observed that a high proportion of Eskimo deaths were from CVD, including fatal cardiac events and stroke. Indian Health Service (IHS) data confirm coronary heart disease (CHD) rates in Eskimos that are at least as high as other Alaska residents and stroke rates that appear to be higher than that of other groups.[4] Studies in other Inuit groups also suggest high rates of CVD.[5,6,7 8,9,10,11] This CVD is occurring despite the substantial proportion of the population maintaining traditional lifestyles and high intakes of omega-3 fatty acids. None of these findings, however, have been based on systematic population-based data.

The Genetics of Coronary Artery Disease in Alaska Natives (GOCADAN) study[12] was established to systematically quantify CVD and its risk factors in Eskimos residing in the Norton Sound region, as well as to investigate the genetic determinants of CVD and its risk factors. The cohort is composed of a population-based sample of Alaska Eskimos, in which it is possible to determine whether novel or traditional risk factors may be contributing to the rapidly changing rates. This manuscript presents the prevalence of CVD and examines its relation to traditional and novel risk factors in this population. We hypothesized that despite the unique lifestyle, traditional risk factors are of major importance.

Methods

Study Population

A total of 1,214 predominantly Inupiat Eskimos (537 men and 677 women) ≥ age 18 from eight villages and the town of Nome in the Norton Sound region of Alaska were examined in 2000-2004 for CVD and associated risk factors as part of the GOCADAN study.[12] Recruitment was conducted by family.[13] In seven of the eight villages and the town of Nome, an average of 82.6% of residents ≥ age 18 participated. Because no CVD events were observed in participants < age 45, the analysis was limited to men (n = 214) and women (n = 285) > age 45. Informed consent was obtained. The study was approved by the institutional review boards of the community and the GOCADAN study.

Procedure

Each participant underwent a physical examination, personal interview, collection of biological specimens, and other diagnostic tests conducted by trained and certified field personnel. Anthropometry (height, weight, body mass index [BMI] and waist circumference) were performed with participants fasting, according to standard procedures.[12] Obesity was defined using National Heart, Lung, and Blood Institute criteria.[14] Seated blood pressure was measured three times under standard conditions, with the mean of the second and third value used as the final measure, and hypertension was defined per Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) criteria.[15] A 12-lead electrocardiogram was taken using a Marquette system. Clinical diagnosis of the

ECG was determined by cardiologists at the reading center, and Minnesota codes were applied using an automated system.[16] Smoking and alcohol intake habits were evaluated via questionnaire; participants were categorized as current, former, and never-smokers.[12] Dietary information was collected via a food frequency questionnaire (FFQ) that was developed and validated for Alaska Natives.[17] Physical activity was calculated in Metabolic Equivalents (METs) from self-reported leisure time activities.[18] Questionnaires also ascertained medical history and family history of CVD. Samples of whole blood, plasma, serum, and urine were collected from each participant and stored at −80° C. All laboratory methods have been published.[12] Diabetes was defined as fasting glucose ≥ 126 mg/dL or taking antidiabetic medications, per American Diabetes Association criteria;[19] for those without a fasting glucose measure (n = 30), a medical record review was performed to confirm the diagnosis. Low-density lipoprotein cholesterol (LDL-C) was calculated by the Friedewald formula.[20]

Because of previous reports of high prevalence of subclinical infections in this population,[21] levels of IgG, IgA, and IgM antibodies to *C. pneumoniae* were determined. Serum levels of IgG antibodies to other pathogens also were determined using commercially available enzyme-linked immunosorbent assay (ELISA) kits from Wampol (cytomegalovirus [CMV] and *Helicobacter pylori*), Focus (herpes simplex virus types 1 [HSV1] and 2 [HSV2]), DiaSorin Inc. (hepatitis A virus [HAV]), and Ortho (hepatitis C virus [HCV]). Serology values identified as positive were determined according to the manufacturer's instructions.[21,22]

For carotid ultrasound studies, B-mode imaging from multiple angles was performed to determine the presence and location of plaque (focal protrusion of the vessel ≥ 50% greater than the surrounding wall), as well as arterial wall dimensions. Plaque score (0-8) was determined as the number of arterial segments (left and right common carotid, bulb, internal and external carotid arteries) containing plaque; a participant with plaque was anyone with a score ≥ 1.[23]

Determination of prevalent CVD

The medical history included questions designed to identify inpatient or outpatient events that may have been cardiovascular. Medical records of all persons who responded affirmatively to any of the CVD questions plus records of all persons whose electrocardiogram was diagnosed as definite or possible for CHD were reviewed and abstracted according to a standardized procedure. All extracted records were then reviewed by an adjudication committee who determined prevalent disease using the following criteria: 1) definite myocardial infarction (MI) determined by coded electrocardiogram or history of MI, verified by enzymatic or ECG evidence in the medical record, and confirmed by the study adjudication committee; 2) cardiac intervention confirmed by evidence in the medical record of coronary angioplasty, atherectomy, stent, or bypass surgery; 3) other CHD determined as >75% stenosis confirmed by angiography; 4) heart failure defined as having one major or two minor Framingham criteria; and 5) stroke defined as progressive neurologic symptoms lasting more than 24 hours and confirmed by physician diagnosis; etiology was defined as ischemic, intracerebral hemorrhage, or subarachnoid hemorrhage confirmed by MRI/CT or unknown in the absence of MRI/CT data.

Statistical methods

All data are expressed as mean ± SD. The natural log transformation was applied to variables that were highly skewed, and the transformed variables were used in all analyses. Prevalence proportions (per 100) were computed for various measures of CVD by gender. Prevalence ratios and 95% confidence intervals (CIs) were computed to compare prevalence of CVD between genders. Univariate assessment of associations of risk factors and CVD were performed by computing prevalence ratios and 95% CIs for variables stratified either by tertile

or by specific diagnostic criteria. Chi-square tests or Fisher's exact tests were applied where appropriate. In the case of LDL-C, apoB, and non-HDL-C, analyses were confounded by the use of lipid lowering medication; thus, a variable was created defining high LDL-C, apoB, or non-HDL-C as having a value above standard criteria (160 mg/dL for LDL-C, 190 for non-HDL-C, and 120 mg/dL for apoB) or taking lipid-lowering medication. Because of the small number of events, each potential risk factor was treated in a separate logistic regression model, adjusted for age and gender, to test the association between the potential risk factors and prevalence of CVD. Because the participants were members of extended families, the impact of relatedness was assessed according to the method of Wang et al.[24] Analysis of variance (ANOVA) was applied to examine the relationship of prevalent CVD events with the amount of plaque in a univariate model and a multivariate model adjusted for age, gender, hypertension, diabetes, and LDL-C.

Results

Table 1 shows data by gender for the cohort > age 45. Average age was 58 years in both genders; diabetes prevalence was low, and HDL-C concentrations were relatively high, but a high proportion smoked and had a high prevalence of subclinical pathogen burden. Men compared to women had lower BMI and less diabetes, but lower HDL-C, higher intake of saturated fat, and higher rates of smoking (all $p<0.01$).

Prevalence rates for categories of CVD by gender are shown (Table 2). Thirteen percent of men and 5% of women met the criteria for definite CVD, defined as MI, coronary artery bypass graft/percutaneous transluminal coronary angioplasty (CABG/PTCA), stroke or CHF, and rates were significantly higher in men (PR 2.4, [CI 1.3-4.4]). The most common type of CVD event was stroke, with 6% of men and 2% of women having definite documented stroke (PR 3.5 [CI 1.2-9.6]). MI was diagnosed in only 2% of men and <1% of women; however, when MI and coronary interventions were combined (composite CHD) rates were 6% in men and 2% in women (PR 2.5 [CI 1.0-6.1]). Only two men and four women met criteria for heart failure. Adjustment for relatedness did not appreciably change any of the prevalence rates.

Because of the relatively small number of events, relations among risk factors were evaluated for the composite CVD endpoint, stratified by potential risk factors (Table 3). Male gender ($p<.002$) and older age ($p<.0001$) were associated with higher prevalence of CVD. Hypertension, diabetes, macro-albuminuria, high LDL-C, high apoB, and low HDL-C were strong correlates of CVD (all $p<.001$) and physical activity was inversely related ($p<.05$). After adjustment for age and gender (Table 4), hypertension, high LDL-C, high apoB, low HDL-C, diabetes, albuminuria, and CRP showed significant associations. Adjustment for relatedness did not appreciably alter any of the observed associations.

We also examined the relationship of CVD events with the presence of carotid atherosclerosis as measured by ultrasound (Table 5). Total CVD and CHD events were correlated with the amount of plaque both in univariate analyses and after adjustment for other CVD risk factors ($p=.0086$ and .0079, respectively). No significant relation was observed with stroke.

Discussion

In this cross-sectional analysis of the GOCADAN cohort, we systematically assessed the prevalence of CVD in a population-based sample of Alaska Eskimos. We found rates of all forms of CVD, except for heart failure, to be much higher in men than in women. Despite the relatively young average age, rates of stroke were higher than those for MI and as high as a composite index of coronary disease that includes MI and revascularization. Because of the relatively low number of events, univariate assessments were stressed; these suggest that

several risk factors are having effects. In a model adjusted for age and gender, dyslipidemia, hypertension, diabetes, albuminuria, and CRP were significant correlates. The availability of data on carotid atherosclerosis in this population[23] confirmed that the cardiac events are indeed related to atherosclerosis, and carotid plaque was an independent correlate of CHD.

It is not easy to compare CVD rates observed in the present study with rates reported in other studies of CVD in Eskimos because the latter were either based on self- or physician-report or death certificate data. Nevertheless, our data confirm the state health data[2,3] that show high rates of cardiac events and stroke and are consistent with the analysis of IHS data by Lanier et al.[4] that indicated high rates of stroke. The Alaska-Siberia Project also suggested high rates of CHD in a related group of Eskimos.[25] The low rate of MI is difficult to interpret from prevalence data; it could represent high initial mortality or rapid interventions upon presentation. The pattern of relatively high stroke rates also has been seen in other Inuit groups.[6,26,27] Possible explanations range from the unique environmental stressors to genetic susceptibility; prospective data are needed with larger numbers of events to explore all these endpoints in more detail.

A comparison of our data can be made with studies of other ethnic groups that used similar adjudication criteria. Prevalence rates for MI in American Indians in the Strong Heart Study, a population now established to have high incidence rates for CVD and stroke, were similar (2.8% for nondiabetic men and 0.4% for nondiabetic women).[28,29] On the other hand, the MI rates in whites and blacks in the ARIC population ages 45-64 were higher (7.9% and 6.1% in men and 1.9% and 3.4% in women, respectively).[30] American Heart Association data on stroke prevalence in the United States suggest that the rates observed in Eskimos of this age range are markedly higher than rates of stroke in other U.S. populations.[31]

Despite the low numbers of events, we were able to evaluate several key risk factors. Rates of CVD were 5-fold higher in persons with diabetes in this population. In contrast to other native populations, diabetes rates in Eskimos are lower than those for the United States in general[32] and thus do not appear to explain the current increases in CVD rates. Diabetes rates appear to be increasing, however, and if this trend continues the diabetes-associated CVD may exacerbate the rate of increase of CVD in this population; the association of CVD with albuminuria, a common complication of diabetes, supports this concern.

We focused our attention on other risk factors that might explain the current high rates of CVD in this population. Although hypertension rates are lower in Eskimos than in other U.S. populations,[33] hypertension was an important correlate of CVD in this population, and the association was also seen with carotid parameters;[23] this suggests that treating even mild hypertension to current standards could significantly reduce event rates. Another strong correlate was hyperlipidemia. We were able to evaluate this risk factor using both LDL-C and apoB as indices of atherogenic lipoproteins; as with hypertension, levels of these indicators are relatively low in Eskimos. Lipid lowering medication is currently being used in approximately 12% of the population; our results suggest treatment of LDL-C and non–HDL-C to current targets will be another effective strategy to reduce CVD. Concentrations of HDL-C are relatively high in this population; nevertheless, those with HDL-C <45 mg/dL (men) or 55 mg/dL (women) in univariate analyses had approximately 2.5-fold higher rates of CVD. The best strategies for raising HDL-C are weight loss and increased physical activity; these lifestyle interventions also can enhance cardiovascular function and reduce risk for diabetes and need to be the cornerstone of all CVD prevention strategies.

Smoking rates are high in this population; in the univariate analysis CVD rates were almost two-fold higher in current smokers; however the relation was not significant. It is difficult to assess independent associations of smoking because a high percentage of the population are

former smokers and almost all are exposed to high levels of passive smoke. Our previous analyses showed that smoking is significantly related to carotid atherosclerosis.[23, 34] Smoking cessation activities are currently proceeding in Eskimo communities and should be encouraged.

We have emphasized in our analyses the possibility of subclinical infection/inflammation as a potential cause of the increasing incidence of CVD in Eskimos. Our previous analyses showed that CRP is related to carotid IMT[23] and that pathogen burden is correlated with carotid plaque.[21] In the current analyses, CRP was a significant correlate of prevalent CVD. Because inflammatory markers increase after CVD events, these relations are difficult to evaluate in cross-sectional studies and await our longitudinal data.

The univariate analyses showed an inverse association between CVD and physical activity. This inverse association has been seen in almost all studies[35,36,37,38,39] and is of particular interest in this study because it is probably a reflection of a change in lifestyle. No relation was observed between CVD and intake of either omega-3 fatty acids or saturated fat. Our previous analyses of the relations between individual dietary fatty acids and carotid atherosclerosis also showed no relation with omega-3 fatty acid intake, but did show a positive relation with saturated fatty acids.[40] Because this was a cross-sectional study, it is possible that diet changes were made by the people who had the CVD events; therefore, understanding the relations among diet and CVD awaits longitudinal data.

This study has several strengths. It is the first population-based assessment of CVD in Alaska Eskimos with events adjudicated using standardized criteria. Data for risk factors were collected using standardized techniques that are comparable with other longitudinal studies. This population has several unique features, including low rates of diabetes, low LDL-C and blood pressure, and high HDL-C. Lifestyle features include a diet high in omega-3 fatty acids and presence of substantial subclinical infection. On the other hand, this study is limited by the cross-sectional nature of the analyses which precludes determination of causative or predictive parameters. Also, because only 499 persons were >age 45, the numbers of events were low. We are conducting a continuous surveillance of this population and seeking to combine with other studies of Eskimos to obtain more reliable estimates of incidence rates and risk factor associations.

In summary, these data show high CHD and stroke prevalence in Alaska Eskimos. Hypertension and high LDL-C were strong independent correlates, indicating that efforts to identify these risk factors early and treat to target will be an effective prevention strategy.

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Table 1
Characteristics of participants > 45 years of age by gender

Variable	Men (n = 214)	Women (n = 285)	P value
Age (years)	57.9 ± 9.5	58.3 ± 10.9	0.696
BMI (kg/m^2)	26.5 ± 4.6	28.9 ± 6.2	<.0001
Waist circumference (cm)	89.5 ± 10.9	90.6 ± 13.5	0.301
LDL-C (mg/dL)	126.2 ± 36.2	124.0 ± 36.3	0.505
HDL-C (mg/dL)	57.1 ± 18.1	68.6 ± 18.9	<.0001
Triglyceride (mg/dL) †	111.7 (104.6-119.3)	124.3(117.5-131.6)	0.015
Non-HDL-C (mg/dL)	150.8 ± 38.4	151.4 ± 38.3	0.858
ApoB (mg/dL)	105.2 ± 24.1	106.5 ± 22.9	0.545
Hypertension	88 (41.1%)	117 (41.1%)	1.000
Diabetes mellitus	6 (2.8%)	23 (8.1%)	0.012
Smoking			0.010
--Never	29 (13.7%)	69 (24.3%)	
--Current	119 (56.1%)	133 (46.8%)	
--Past	64 (30.2%)	82 (28.9%)	
CRP (mg/L)†	1.4 (1.2-1.8)	1.3 (1.1-1.6)	0.616
CRP ≤ 10 (mg/L)†	1.1 (0.9-1.3)	1.0 (0.9-1.2)	0.744
Pathogen burden	3.6 ± 1.0	3.7 ± 0.9	0.153
Microalbuminuria	11 (5.1%)	31 (10.9%)	0.023
Macroalbuminuria	5 (2.3%)	1 (0.4%)	0.089
HOMA –IR	2.5 ± 4.1	3.0 ± 2.8	0.186
Fibrinogen (mg/dL)	358.3 ± 114.1	353.8 ± 107.0	0.663
Taking lipid-lowering medication	24 (11.2%)	39 (13.7%)	0.496
Hyperlipidemia[1]	73 (34.1%)	115 (40.4%)	0.162
Hyper LDL-C[2]	58 (27.1%)	83 (29.1%)	0.688
Hyper ApoB[3]	67 (31.3%)	105 (36.8%)	0.217
Physical activity (all MET)†	34.0 (27.8-41.6)	26.6 (22.6-31.4)	0.062
Percent energy from saturated fat	13.4 ± 3.9	13.2 ± 3.7	0.458

Notes. BMI = body mass index; CRP = C-reactive protein; HDL-C = high-density lipoprotein cholesterol; HOMA-IR = homeostasis model assessment of insulin resistance; LDL-C = low-density lipoprotein cholesterol.

Data are means ± STD, or N (%).

† Geometric mean and 95% confidence interval.

[1] LDL-C > 160, non-HDL-C > 190, ApoB > 120, or taking lipid-lowering medication.

[2] LDL-C > 160 or taking lipid-lowering medication.

[3] ApoB > 120 or taking lipid-lowering medication.

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Table 2

Prevalence of CVD by gender in Alaska Eskimos > age 45 years

Category	Men		Women		PR* M/F	CI†
	N	PP‡	N	PP		
Definite MI	4	1.87 *(1.88)***	2	0.70 *(0.68)*	2.66	0.49-14.41
CABG/PTCA	12	5.61 *(5.63)*	6	2.11 *(2.20)*	2.66	1.02- 6.98
Composite CHD	13	6.07 *(6.06)*	7	2.46 *(2.52)*	2.47	1.00- 6.09
Definite stroke	13	6.07 *(6.46)*	5	1.75 *(1.71)*	3.46	1.25- 9.56
CHF	2	0.93	4	1.40	0.67	0.12- 3.60
All CVD	27	12.62 *(12.56)*	15	5.26 *(5.26)*	2.40	1.31- 4.39
All CVD (including possible MI)	28	13.08 *(12.93)*	15	5.26 *(5.27)*	2.49	1.36- 4.54

Notes. CAGB/PTCA = coronary artery bypass graft/percutaneous transluminal coronary angioplasty; CHF = congestive heart failure; CVD = cardiovascular disease; MI = myocardial infarction

** Second value in italics is prevalence after adjustment for relatedness.

‡ PP, prevalence proportion

* PR, prevalence ratio.

† CI, confidence interval.

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Table 3

Prevalence proportion (per 100) of CVD according to risk factors in Alaska Eskimos

Variable	PP[§]	PR[*]	95% CI	P value	P value[†]
Gender				0.0021	
Men	13.1	---	---		Referent
Women	5.3	0.40	0.22- 0.73		0.0030
Age (years)				<.0001	
<60	4.4	---	---		Referent
≥60	15.8	3.55	1.92- 6.54		<0.0001
BMI (kg/m^2)				0.2268	
<25	5.3	---	---		Referent
[25-30)	10.1	1.92	0.88- 4.19		0.2869
≥30	9.1	1.73	0.77- 3.88		0.4027
Waist circumference (cm)					
<102, 88 (men, women)	7.5	---	---	0.4208	Referent
≥ 102, 88 (men, women)	9.6	1.28	0.70- 2.33		0.5849
Hyper LDL-C				<.0001	
No	5	---	---		Referent
Yes	17.7	3.53	1.99- 6.26		<0.0001
Hyper ApoB				0.0006	
No	5.5	---	---		Referent
Yes	14.5	2.64	1.48- 4.70		0.0012
HDL-C (mg/dL)				<.0001	
<45 or 55 (men, women)	18	---	---		Referent
≥45 or 55 (men, women)	6	0.33	0.19- 0.58		0.0002
Hypertension				<.0001	
No	2	---	---		Referent
Yes	18	8.84	3.80-20.57		<0.0001
Never smoking				0.3896	
No	8.8	---	---		Referent

Variable	PP[§]	PR[*]	95% CI	P value	P value[†]
Yes	6.1	0.70	0.30- 1.61		0.4343
Current smoking				0.1324	
No	10.5	---	---		Referent
Yes	6.7	0.64	0.36- 1.15		0.1436
Smoking status				0.1033	
Never	6.1	---	---		Referent
Current	6.7	1.10	0.45- 2.71		0.668
Past	12.3	2.01	0.83- 4.89		0.3012
CRP ≤ 10 (mg/L)				0.1345	
<1	6.3	---	---		Referent
[1,3)	8.3	1.31	0.60- 2.84		0.6009
[3,10]	13.6	2.16	0.99- 4.69		0.0565
Pathogen burden				0.1275	
0-3	6.3	---	---		Referent
≥4	10.3	1.64	0.86- 3.15		0.117
Triglyceride (mg/dL)				0.1884	
<100	8.5	---	---		Referent
[100,150)	6.1	0.72	0.33- 1.56		0.4466
≥150	12.1	1.41	0.73- 2.73		0.2559
Diabetes				<.0001	
No	7.2	---	---		Referent
Yes	31	4.29	2.28- 8.07		<0.0001
Macroalbuminuria				0.0099[**]	
No	8.1	---	---		Referent
Yes	50	6.16	2.62-14.47		0.0041
Percent energy from saturated fat[‡]				0.2940	
Quartile 1	9.7	---	---		Referent
Quartile 2	4.4	0.45	0.16- 1.26		0.0469
Quartile 3	7	0.72	0.30- 1.73		0.2396

Variable	PP[§]	PR[*]	95% CI	P value	P value[†]
Quartile 4	10.6	1.09	0.50- 2.37		0.7881
Physical activity (METS)[2]				0.0496	
Quartile 1	12.3	---	---		Referent
Quartile 2	5.7	0.46	0.18- 1.17		0.0234
Quartile 3	6.5	0.53	0.22- 1.28		0.0389
Quartile 4	2.8	0.23	0.07- 0.79		0.0040

Notes. CVD = cardiovascular disease; BMI = body mass index; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

[§]PP: prevalence proportion.

[*]PR: prevalence ratio.

[†]The p value is adjusted for relatedness.

[**]Fisher's exact test.

[1]Percent energy from saturated fat quartile 1: 2.5-10.7, n=113; quartile 2: 10.7-12.9, n=114; quartile 3: 12.9-15.7, n=114; quartile 4: 15.7-30.1, n=113.

[2]Physical activity quartile 1: 0.06-15.6 METS, n=106; quartile 2: 15.7-34.7 METS, n=106; quartile 3: 35.1-73.5 METS, n=107; quartile 4: 73.8-395.5 METS, n=106.

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Table 4

Logistic regression coefficients and prevalence odds ratios for CVD in Alaska Eskimos

Variable	Coefficient	Standard Error	OR†	95% CI†	P†
High LDL-C (Y/N)	1.4532	0.3490	4.28	2.16-8.48	<.0001
Hypertension (Y/N)	2.0706	0.4682	7.93	3.17-19.85	<.0001
High apoB (Y/N)	1.2435	0.3487	3.47	1.75-6.87	0.0004
Diabetes (Y/N)	1.6058	0.5089	4.98	1.84-13.51	0.0016
HDL-C (mg/dL)	-0.0330	0.0109	0.97	0.95-0.99	0.0025
Log CRP (mg/L)*	0.4279	0.1784	1.53	1.08-2.18	0.0165
Macroalbuminuria (Y/N)	1.8905	0.9187	6.62	1.09-40.09	0.0396

Notes. CRP = C-reactive protein; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol.

†Separate model for each risk factor, adjusted for age and gender.

*Participants with CRP>10mg/L were excluded for the CRP analysis.

†OR: odds ratio; 95% CI: 95% confidence interval.

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Table 5

Relations among CVD and carotid atherosclerosis

Carotid atherosclerosis	CVD event	N	Mean	SD	Median	P value[1]	P value[2]
Log (affected seg+1)*	**All CVD**					<.0001	0.0086
	--No	436	0.73267	0.63003	0.69315		
	--Yes	40	1.30368	0.59086	1.38629		
	CHD					<.0001	0.0079
	--No	456	0.74631	0.63443	0.69315		
	--Yes	20	1.56372	0.34750	1.60944		
	Definite stroke					0.1154	0.2492
	--No	460	0.77195	0.64119	0.69315		
	--Yes	16	1.03067	0.75245	1.09861		

*Log of the number of carotid segments with plaque.

[1]Univariate model.

[2]Multivariate model: adjusted for age, gender, hypertension, diabetes, and high LDL-C.