

# Dietary Patterns are Linked to Cardiovascular Risk Factors but Not to Inflammatory Markers in Alaska Eskimos<sup>1–3</sup>

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## Abstract

Despite the tradition of a diet high in fish oils and abundant physical activity, coronary artery disease is increasing among Alaska Eskimos. Explanations for this observation include lifestyle changes. In this cross-sectional analysis, we evaluated dietary patterns of Alaska Eskimos and investigated the relations between these dietary patterns and known cardiovascular risk factors, including inflammatory markers. We used a principal component analysis with data from FFQ collected in 2000–2004 to determine dietary patterns of Alaska Eskimos. Four dietary patterns were identified: a traditional pattern, plus 3 patterns based on purchased food, one of which reflected healthy food choices. The traditional dietary pattern was associated with lower triglycerides ( $P < 0.001$ ) and blood pressure ( $P = 0.04$ ) and slightly higher LDL cholesterol (LDL-C) ( $P = 0.05$ ). Whereas the healthy purchased diet was associated with a trend toward lower LDL-C ( $P = 0.09$ ), the beverages and sweets diet was positively associated with LDL-C ( $P = 0.02$ ). Diet pattern was not associated with inflammatory markers or pathogen burden. Our data show that the traditional diet is related to a better profile of cardiovascular disease risk factors and should be encouraged. Programs are needed to encourage the availability of healthy food choices for those not able to obtain traditional foods. *J. Nutr.* 139: 2322–2328, 2009.

## Introduction

Population studies and intervention trials have demonstrated that a diet rich in (n-3) fatty acids from fish or (n-3) fatty acid supplements reduces cardiovascular disease (CVD).<sup>11</sup> Eskimos and Inuit peoples were thought to be protected from CVD because of their high intakes of marine mammals that contain (n-3) fatty acids (1). Dyerberg et al. (2) originally described marked increases in mean bleeding time and decreased platelet adherence among Greenland Inuit who consumed a diet rich in fish and marine mammal oils and early studies from Alaska reported a lower prevalence of coronary and aortic atherosclerosis in Alaska Natives compared with non-Natives (3,4).

Recent reviews of state health data, however, show an increase in CVD among Eskimos, with rates in the Norton Sound region of Alaska that are twice those of surrounding areas (3,5) and a prevalence of atherosclerotic plaque that is higher in Eskimos compared with other U.S. populations (6). Although ischemic heart disease mortality in Alaska Natives has not increased, rates have declined among U.S. whites in general. Ischemic heart disease mortality rates among Alaska Native men are now similar to those of other U.S. white men and stroke rates are higher in Alaska Natives compared with the U.S. population in general (7).

Two epidemiologic studies of Alaska Eskimos now show that dietary intake of (n-3) fatty acids in a moderate-to-high range is not associated with reduced atherosclerotic plaque (8) but that increasing amounts of saturated fat in the Eskimo diet are related to CVD risk (9–11). One explanation for these observations may be a lifestyle change among Alaska Eskimos that includes a shift from traditional foods to energy-dense convenience foods with a concurrent reduction in physical activity (12).

This lifestyle change suggests that it may be beneficial to examine overall dietary patterns rather than investigating associations between specific nutrients, because multiple dietary components (i.e. dietary patterns) act together to influence CVD risk. Factor analysis, specifically principal component analysis, has been used to derive eating patterns and to relate these

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<sup>3</sup> Supplemental Tables 1 and 2 are available with the online posting of this paper at [jn.nutrition.org](http://jn.nutrition.org).

<sup>11</sup> Abbreviations used: CRP, C-reactive protein; CVD, cardiovascular disease; GOCADAN, Genetics of Coronary Artery Disease in Alaska Natives; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; MET, metabolic equivalent; MUFA, mono-unsaturated fatty acid; TFA, *trans* fatty acid; TG, triglyceride.

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patterns to symptoms or diseases (13). CVD has been shown to be negatively related to traditional dietary patterns and positively related to Western diets in some (14–17) but not all (18) studies. In addition to the relationships among dietary patterns and conventional CVD risk factors, it has been hypothesized that some dietary patterns may influence CVD risk by potential effects on vascular inflammation (19), because low-grade systemic inflammation is a predictor of cardiovascular events (20–22).

In this article, we provide a description of dietary patterns derived using principal component analysis for Alaska Eskimos based on data from the participants of the Genetics of Coronary Artery Disease in Alaska Natives (GOCADAN) study, a population-based study of CVD and its risk factors in Alaska Eskimos. Additionally, we explored the relation between these dietary patterns and known cardiovascular risk factors, including inflammatory markers.

## Materials and Methods

**Study population.** A total of 1214 predominantly Inupiat Eskimos (537 men and 677 women)  $\geq 18$  y from the Norton Sound Region of Alaska were examined in 2000–2004 for CVD and associated risk factors as part of the GOCADAN study (23). Recruitment was conducted by family in 7 villages and the town of Nome (24). Of eligible residents, 82.6% participated. Informed consent was obtained. The study was approved by the institutional review boards of the Norton Sound Community and the participating institutions.

The analysis was based on data from participants who provided full nutritional data ( $n = 1173$ ). Extreme cases of energy consumption, i.e.  $< 500$  or  $> 8000$  kcal/d (1 kcal = 4.187 kJ), were excluded ( $n = 68$ ). After excluding participants with diabetes ( $n = 39$ ), which can alter dietary patterns and CVD risk factors, analyses were performed using data from 1066 participants ages 18–92 y. C-reactive protein (CRP)  $> 10$  is a marker of an active acute infection; therefore, participants with such elevated CRP levels ( $n = 66$ ) were excluded from the analyses relating dietary patterns to inflammatory markers.

**Measurements.** Dietary information was collected via FFQ, which evaluated consumption during the previous year (24). The questionnaire inquired about 97 food items, including major traditional foods and key foods available in small village stores. Foods from the FFQ were classified into 28 food groups on the basis of nutrient profiles or culinary usage (Supplemental Table 1). Foods that did not fit into any of the groups or that seemed to represent distinctive dietary behaviors were entered as separate food groups for the principal component analysis.

Blood samples were obtained following a 12-h overnight fast and measurements were obtained for a lipid panel [total serum cholesterol, total triglyceride (TG), HDL cholesterol (HDL-C), and LDL cholesterol (LDL-C)] analyzed via conventional enzymatic chemistry (25). Participants were considered hypertensive if they had systolic blood pressure  $\geq 140$  mm Hg, diastolic blood pressure  $\geq 90$  mm Hg (25), and/or were taking blood pressure medication at the time of the interview. Metabolic syndrome was defined via ATPIII criteria (26). Measures of inflammation consisted of serum CRP, homocysteine, and fibrinogen, as described previously (26), plus pathogen burden. The sum of the pathogen burden was calculated after measuring levels of IgG, IgA, and IgM antibodies to *Chlamydia pneumoniae* and serum levels of IgG antibodies to other pathogens using commercially available ELISA kits from Wampol (cytomegalovirus and *Helicobacter pylori*) and Focus (herpes simplex virus types 1 and 2). Serology values identified as positive were determined according to the manufacturer's instructions (27).

Data on known CVD risk factors, current drinking, smoking, and physical activity levels were collected. Current drinking was defined as having had an alcoholic drink within the last year. Self-reported smoking status was categorized as current or former smoker or as never smoked. Physical activity was calculated in metabolic equivalents (MET) from self-reported leisure time activities (28). MET was calculated from an

activity questionnaire developed for Native populations. MET times per week were computed by multiplying MET, for each recorded activity, by the weekly frequency and the fraction of the year during which it was performed. An overall mean weekly score was calculated as MET-times per week.

**Statistical analysis.** Baseline demographic characteristics were presented as means and SD for continuous variables or as numbers and percentages for categorical variables. Principal component analysis (29) was applied to foods or food groups derived from the FFQ data, using the Nutrition Data System for Research Database version 4.06\_34 (Minneapolis, MN), which contains nutrient information on Alaskan foods (30). Food groups were also defined by their origin (harvested or purchased). The statistical software SAS version 9.1 (SAS Institute) and Intercooled Stata 9.2 (Stata Corp) were used to conduct all analyses. Twenty-eight variables were created for the factor analysis, calculated as percentage of total daily energy consumed from a given food group. A factor score for each diet pattern was constructed by summing intakes from the 28 food groups weighted by factor loadings. Factors were rotated by an orthogonal transformation method (Orthogonal Varimax) to achieve a simpler structure with greater interpretability. The number of factors to retain was calculated using the Kaiser-Guttman rule (eigenvalue  $\geq 1$ ) and the scree plot. Twelve factors had eigenvalues  $> 1$ . Using the scree plot, the first 4 factors were retained and total variance explained by them was 25%. The factor loading matrix for the 4 dietary patterns is provided as an online supplement (Supplemental Table 2).

Relationships among the dietary patterns, lifestyle, and demographic variables, as well as percentage of energy from nutrients, were examined across quintiles of each factor using means  $\pm$  SE for continuous variables or numbers (percentages) for categorical variables. The first quintile represents the participants who had the lowest adherence to the pattern and the 5th quintile represents the participants who had the highest adherence. Quintiles 1, 3, and 5 are presented in the tables. These bivariate relationships were evaluated using a nonparametric trend test by quintiles of dietary patterns (nptrend in Stata). Adjusted means for CVD risk factors and inflammatory markers were obtained using ordinary least squares models, including dummy variables representing the levels of dietary patterns as covariates. Adjusted means represent predicted values of the dependent variables across quintiles of the dietary patterns. *P*-values for trends across quintiles were separately computed by treating the quintile variables as linear terms in the multivariate models ( $\alpha = 0.05$ ).

## Results

More women than men participated in the study. At baseline, mean participant BMI was 27.6 (Table 1). Percentages of energy derived from total fat, SFA, and *trans* fatty acid (TFA) were higher than recommended by national guidelines (24). Daily intake of (n-3) fatty acids as a percentage of energy was high compared with national averages: mean 1.3% (4.6 g/d) and median 1.06% (3.3 g/d) (31). In contrast, the median intake in NHANES was lower than 0.5% and predominantly composed of  $\alpha$ -linolenic acid (32).

We derived 4 dietary patterns from the principal component analysis (Supplemental Table 2). Participants following factor 1, the "traditional" diet, consumed fish, native sea and land mammals and their fats and oils, wild greens, stew with mostly meat, stew with mostly rice or noodles, native birds, wild berries, and native berry *agutuk*. This diet scored low in candy bars, sugar, syrup, store-bought nonhydrogenated vegetable fats, peanut butter, milk, cheese, ice cream, and nondairy creamer.

Factor 2, the "Western" diet, scored high in store-bought meats, chicken, fatty meats, snack chips, pizza, fried potatoes, soda pop, milk, cheese, ice cream, nondairy creamer, stew with mostly meat, and stew with mostly rice or noodles. This diet was negatively correlated with native sea and land animal fats,

**TABLE 1** Baseline characteristics of the GOCADAN population, 2001–2004<sup>1</sup>

Variable	n	
Demographic and lifestyle		
Male	1066	460 (43.2)
Age, y	1066	41.7 ± 15.5
Women over age 50 y	1066	136 (13)
BMI, kg/m <sup>2</sup>	1060	27.6 ± 5.7
Waist circumference, cm	1044	86.3 ± 12.9
Education, y	1033	11.8 ± 2.3
Physical activity, <sup>2</sup> MET	957	40.3 (37.2–43.6)
Current alcohol drinker (yes)	1062	747 (70.3)
Current smoker (yes)	1066	639 (60.0)
Hypertension <sup>3</sup>	1060	206 (19.4)
Systolic blood pressure, mm Hg	1059	119.0 ± 14.4
All CVD	1066	28 (2.6)
Cancer	1066	45 (4.2)
Nutrient intake		
Fiber density, g/4187 kJ	1066	5.7 ± 2.4
Cholesterol density, mg/4187 kJ	1066	173.7 ± 77.5
Total intake, MJ/d	1066	1310 ± 640
Carbohydrate, % energy	1066	49.0 ± 10.8
Protein, % energy	1066	14.7 ± 4.2
Total fat, % energy	1066	37.3 ± 8.6
Monounsaturated fat, % energy	1066	14.2 ± 3.6
Saturated fat, % energy	1066	12.8 ± 3.6
Polyunsaturated fat, % energy	1066	7.1 ± 2.4
TFA, % energy	1066	2.0 ± 1.0
(n-3) Fatty acids, % energy	1066	1.3 ± 0.9
(n-3) Fatty acids, g/d	1066	4.6 ± 4.7
Biochemistry		
Plasma glucose, mmol/L	1022	5.1 ± 0.6
Serum HDL-C, mmol/L	1020	1.5 ± 0.5
Serum LDL-C, mmol/L	1020	2.3 ± 0.9
Serum TG, <sup>2</sup> mmol/L	1020	1.24 (1.20–1.27)
Serum insulin, pmol/L	1018	69.5 ± 50.7
Serum CRP, <sup>2</sup> mg/L	808	10 (9–11)
Plasma fibrinogen, mmol/L	1011	9.6 ± 2.9
Plasma homocysteine, μmol/L	1021	7.3 ± 3.0
Pathogen burden	795	3.1 ± 1.2

<sup>1</sup> Values are means ± SD or n (%) unless noted otherwise.

<sup>2</sup> Presented as geometric means with 95% CI.

<sup>3</sup> Hypertension was defined as systolic blood pressure ≥ 140 mm Hg, diastolic blood pressure ≥ 90 mm Hg, and/or participant taking blood pressure medication.

evaporated milk, store-bought hydrogenated vegetable fats, store-bought animal fats, pancakes, and hot cereal.

Factor 3, the “purchased healthy” diet pattern, was based on purchased food that reflected an attempt to follow the current consensus of scientific dietary advice. Participants following this diet consumed dry beans, store-bought fruits, vegetables, lettuce salad, dark bread, hot cereal, store-bought nonhydrogenated vegetable fats, peanut butter, milk, cheese, ice cream, nondairy creamer, and lower fat milks and did not regularly consume flavored drinks, soda pop, snack chips, or pizza.

Factor 4, the “beverages and sweets” diet, correlated positively with coffee, tea, candy bars, sugar, syrup, eggs, store-bought animal fats, and pancakes. Participants following this pattern did not regularly consume white bread, pasta, rice, cold cereal, pilot bread, store-bought fruits, vegetables, lettuce salad, or lower fat milk.

The traditional diet tended to be associated with older age and less smoking and alcohol consumption ( $P = 0.07$ ). The

Western diet was associated with younger age, more education, and more physical activity, but also more alcohol consumption. The purchased healthy diet was associated with older age, greater BMI and waist circumference, more education, less smoking, lower alcohol consumption, and less physical activity. The beverages and sweets diet was associated with older age, male gender, and greater alcohol consumption and smoking (Table 2).

Participants who scored higher on the traditional dietary pattern had lower percentages of energy intake from carbohydrates and sucrose and significantly higher percentages from protein and fat, especially PUFA, (n-3) fatty acids, and mono-unsaturated fatty acid (MUFA) (Table 3). Fiber density was markedly higher in the 5th quintile of this pattern.

The Western diet was associated with a higher percentage of energy from protein in increasing quintiles, but a lower percentage of fat of all types, especially MUFA, PUFA, and TFA, with increasing quintiles. Intake of (n-3) fatty acids was significantly lower with each increasing quintile (>50% difference between the 5th and 1st quintiles).

Participants following the purchased healthy diet had a lower mean carbohydrate intake (as percentage of total energy intake), mostly attributed to the lower sucrose intake (a difference of almost 50%) and higher fiber density (almost a doubling in mean fiber density) in increasing quintiles of this diet. However, fat of all types and cholesterol also increased with increasing quintiles.

Participants in the 5th quintile of the beverages and sweets diet had a significantly higher sucrose intake compared with participants in the lower quintiles of this pattern. This pattern was positively related to SFA and cholesterol intake, but not to PUFA, and was negatively related to fiber density.

Participants in the higher quintiles of the traditional diet had lower serum TG concentrations ( $P < 0.001$ ) and tended to have higher serum HDL-C concentrations ( $P = 0.13$ ) (Table 4). Followers of the Western diet had higher homocysteine concentrations and systolic blood pressure (Table 4). Followers of the purchased healthy diet had lower mean homocysteine and fibrinogen and tended to have lower LDL-C ( $P = 0.09$ ). The beverages and sweets diet was significantly associated with higher LDL-C but lower CRP concentrations. All the trends in inflammatory markers involved differences that were not clinically meaningful.

## Discussion

The current analysis is the first systematic examination of dietary patterns in a population-based sample of Alaska Eskimos and reveals a set of patterns that reflect the changing lifestyles and limited availability of foods for purchase. Historically, Eskimo diets depended on fish and sea mammals, wild game, berries, roots, and wild greens. The traditional diet appears to reflect this eating style, with large amounts of sea mammals and wild game. The lower carbohydrate and sucrose intake in increasing quintiles of this diet reflects the low intake of breads and sweets, and the higher fiber density in increasing quintiles may reflect intake of greens and wild berries. This dietary pattern appears to reflect the traditional lifestyle and is encouraged by health professionals serving Alaska Eskimos. As can be expected, individuals following this pattern were older but also exhibited a trend toward more physical activity.

The 3 other dietary patterns reflect either less accessibility or less use of traditional foods. The Western diet included a lot of store-bought meat; this diet was characterized by higher

**TABLE 2** Demographic and lifestyle variables by quintile of dietary pattern (GOCADAN, 2000–2004)<sup>1,2</sup>

Factors	Male, <i>n</i> (%)	Age, <i>y</i>	BMI, <i>kg/m</i> <sup>2</sup>	Waist circumference, <i>cm</i>	Education, <i>y</i>	MET <sup>3</sup>	Current alcohol drinker, <i>n</i> (%)	Current smoker, <i>n</i> (%)
Factor 1, Traditional foods								
Q1	96 (45)	39 ± 1.0	28.2 ± 0.4	87.6 ± 0.9	12 ± 0.1	35 ± 28–43	166 (78)	135 (63)
Q3	100 (47)	42 ± 1.1	27.4 ± 0.4	86.6 ± 0.9	12 ± 0.2	41 ± 34–49	157 (74)	135 (63)
Q5	90 (42)	46 ± 1.1	27.3 ± 0.3	85.6 ± 0.8	12 ± 0.2	49 ± 42–55	133 (62)	110 (52)
<i>P</i> -trend	0.06	<0.01	0.19	0.24	0.06	0.33	<0.01	0.07
Factor 2, Western foods								
Q1	82 (38)	53 ± 1.1	27.3 ± 0.4	86.6 ± 0.9	11 ± 0.2	33 ± 28–40	136 (64)	120 (56)
Q3	93 (44)	42 ± 1.0	27.5 ± 0.4	86.2 ± 0.9	12 ± 0.2	41 ± 31–44	144 (68)	132 (62)
Q5	96 (45)	33 ± 0.8	27.7 ± 0.4	85.7 ± 1.0	12 ± 0.1	49 ± 40–55	166 (78)	126 (59)
<i>P</i> -trend	0.37	<0.01	0.64	0.24	<0.01	<0.01	<0.01	0.52
Factor 3, Purchased healthy foods								
Q1	86 (40)	34 ± 0.9	27.3 ± 0.4	84.9 ± 0.9	12 ± 0.1	45 ± 37–54	171 (81)	152 (71)
Q3	97 (46)	43 ± 1.0	27.7 ± 0.4	86.9 ± 0.9	12 ± 0.1	40 ± 34–47	150 (70)	117 (55)
Q5	75 (35)	48 ± 1.0	28.3 ± 0.4	87.7 ± 0.9	12 ± 0.2	35 ± 30–42	130 (61)	96 (45)
<i>P</i> -trend	0.04	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Factor 4, Beverages and sweets								
Q1	68 (32)	37 ± 1.1	28.0 ± 0.4	85.9 ± 1.0	12 ± 0.2	42 ± 35–50	136 (64)	93 (44)
Q3	102 (48)	41 ± 1.0	27.6 ± 0.4	87.2 ± 0.8	12 ± 0.2	41 ± 35–49	160 (76)	133 (62)
Q5	107 (50)	46 ± 0.9	27.1 ± 0.4	85.6 ± 0.9	12 ± 0.2	38 ± 31–46	162 (76)	161 (76)
<i>P</i> -trend	<0.01	<0.01	0.14	0.86	<0.03	0.57	0.01	<0.01

<sup>1</sup> Values are means ± SD or *n* (%) unless noted otherwise.

<sup>2</sup> *P*-value for the trend test is based on a nonparametric trend test (Stata's *nptrend* command) for continuous variables and chi-square test for categorical variables by food factor quintiles.

<sup>3</sup> MET are presented as geometric means and 95% CI.

percentage of energy from protein intake and lower (n-3) fatty acid intake in increasing quintiles. This diet was consumed by younger, more active, slightly more educated participants in increasing quintiles.

The purchased healthy diet was consumed more by older, more educated, less active individuals, perhaps because as individuals age or become overweight they suffer from risk factors and disease that prompt changes to a more healthful diet.

**TABLE 3** Mean dietary intake by quintile of dietary pattern (GOCADAN, 2000–2004)

	Carbohydrate	Protein	Total fat	MUFA <sup>1</sup>	SFA <sup>2</sup>	PUFA	TFA	(n-3)	Fiber density	Cholesterol density	Sucrose	Total
								Fatty acids				energy intake
	% energy							<i>g/d</i>	<i>g/kJ</i>	<i>mg/kJ</i>	% energy	<i>MJ/d</i>
Factor 1, Traditional foods												
Q1	52.6 ± 0.8	12.7 ± 0.3	35.8 ± 0.7	13.1 ± 0.3	13.3 ± 0.3	6.7 ± 0.2	6.8 ± 0.4	2.7 ± 0.1	5.1 ± 0.2	142 ± 4.5	17.4 ± 0.7	1352 ± 44
Q3	49.0 ± 0.7	14.5 ± 0.2	37.4 ± 0.5	14.2 ± 0.2	13.0 ± 0.2	7.1 ± 0.2	7.5 ± 0.3	4.4 ± 0.3	5.5 ± 0.1	178 ± 4.8	12.6 ± 0.5	1328 ± 44
Q5	43.3 ± 0.7	17.5 ± 0.3	39.7 ± 0.6	15.9 ± 0.3	12.3 ± 0.2	7.7 ± 0.2	7.3 ± 0.4	7.2 ± 0.5	6.3 ± 0.2	204 ± 5.5	10.1 ± 0.4	1313 ± 47
<i>P</i> -trend	<0.01	<0.01	<0.01	<0.01	0.33	<0.01	0.05	<0.01	<0.01	<0.01	<0.01	0.81
Factor 2, Western foods												
Q1	48.0 ± 0.7	13.0 ± 0.2	40.1 ± 0.6	15.3 ± 0.3	13.6 ± 0.3	7.8 ± 0.2	9.1 ± 0.5	6.6 ± 0.5	5.7 ± 0.2	166 ± 4.3	13.1 ± 0.6	1367 ± 43
Q3	50.4 ± 0.7	14.5 ± 0.3	36.1 ± 0.6	13.7 ± 0.2	12.5 ± 0.2	6.8 ± 0.2	6.5 ± 0.3	4.2 ± 0.2	5.8 ± 0.2	175 ± 5.9	15.4 ± 0.4	1307 ± 46
Q5	46.9 ± 0.8	16.5 ± 0.3	37.2 ± 0.5	14.3 ± 0.2	12.8 ± 0.2	6.8 ± 0.2	5.6 ± 0.3	3.1 ± 0.2	5.8 ± 0.2	186 ± 6.4	10.7 ± 0.3	1181 ± 41
<i>P</i> -trend	0.39	<0.01	<0.01	0.02	0.02	<0.01	<0.01	<0.01	0.05	0.07	0.02	> 0.01
Factor 3, Purchased healthy foods												
Q1	58.8 ± 0.8	12.1 ± 0.3	29.8 ± 0.5	11.5 ± 0.2	10.0 ± 0.2	5.8 ± 0.1	6.8 ± 0.3	3.8 ± 0.2	4.1 ± 0.1	146 ± 5.1	19.5 ± 0.6	1486 ± 45
Q3	47.1 ± 0.6	15.0 ± 0.2	38.8 ± 0.5	14.6 ± 0.2	13.6 ± 0.2	7.2 ± 0.2	7.3 ± 0.3	5.3 ± 0.3	5.4 ± 0.1	180 ± 4.8	12.7 ± 0.4	1347 ± 43
Q5	44.6 ± 0.6	16.0 ± 0.2	40.7 ± 0.6	15.3 ± 0.2	14.1 ± 0.3	7.8 ± 0.2	6.1 ± 0.3	3.7 ± 0.2	8.1 ± 0.2	177 ± 5.8	9.4 ± 0.4	1059 ± 38
<i>P</i> -trend	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.04	0.31	<0.01	<0.01	<0.01	<0.01
Factor 4, Beverages and sweets												
Q1	53.4 ± 0.7	14.0 ± 0.3	33.8 ± 0.6	12.8 ± 0.2	11.3 ± 0.2	6.8 ± 0.2	6.1 ± 0.3	3.6 ± 0.2	7.2 ± 0.2	129 ± 3.5	13.0 ± 0.5	1216 ± 42
Q3	48.0 ± 0.7	15.0 ± 0.3	37.7 ± 0.5	14.6 ± 0.2	12.7 ± 0.2	7.2 ± 0.2	7.8 ± 0.4	5.2 ± 0.3	5.5 ± 0.1	169 ± 3.8	12.6 ± 0.5	1389 ± 46
Q5	48.1 ± 0.9	14.8 ± 0.3	38.0 ± 0.7	14.3 ± 0.3	13.6 ± 0.3	6.8 ± 0.2	6.6 ± 0.3	3.9 ± 0.2	4.4 ± 0.1	222 ± 7.5	16.2 ± 0.7	1263 ± 41
<i>P</i> -trend	<0.01	<0.01	<0.01	<0.01	<0.01	0.88	0.07	0.02	<0.01	<0.01	<0.01	0.12

<sup>1</sup> Data are unadjusted means ± SE.

**TABLE 4** Inflammatory markers and CVD risk factors by quintile of dietary pattern<sup>1-3</sup>

Food factors	CRP <sup>4</sup>	Fibrinogen	Homocysteine	Pathogen burden	Systolic blood pressure	LDL-C	HDL-C	TG <sup>4</sup>
	nmol/L	mmol/L	μmol/L		mm Hg	mmol/L		
Factor 1, Traditional foods								
Q1	8.09 (6.57–10.00)	9.0 (8.6–9.4)	7.3 (6.9–7.6)	3.0 (2.9–3.1)	120 (118–122)	2.87 (2.74–3.00)	1.50 (1.45–1.58)	1.37 (1.28–1.46)
Q3	8.09 (6.57–10.00)	9.4 (9.0–9.8)	7.5 (7.1–7.9)	3.0 (2.9–3.2)	119 (117–121)	3.03 (2.90–3.16)	1.55 (1.50–1.63)	1.25 (1.17–1.34)
Q5	7.62 (6.10–9.52)	9.6 (9.2–10.0)	6.8 (6.4–7.2)	3.0 (2.9–3.2)	118 (116–120)	2.98 (2.85–3.11)	1.60 (1.53–1.66)	1.27 (1.19–1.36)
<i>P</i> -trend	0.47	0.15	0.12	0.25	0.04	0.05	0.13	<0.01
Factor 2, Western foods								
Q1	8.29 (6.57–10.48)	9.5 (9.0–9.9)	6.9 (6.4–7.3)	3.0 (2.9–3.2)	119 (117–121)	2.98 (2.82–3.12)	1.55 (1.50–1.63)	1.25 (1.16–1.34)
Q3	8.67 (7.05–11.43)	9.5 (9.1–9.9)	7.2 (6.8–7.6)	3.0 (2.9–3.2)	117 (115–119)	3.00 (2.98–3.13)	1.55 (1.48–1.61)	1.27 (1.19–1.36)
Q5	8.57 (6.95–11.43)	9.5 (9.1–9.9)	7.6 (7.2–8.0)	3.1 (2.9–3.3)	121 (119–122)	2.93 (2.79–3.06)	1.50 (1.45–1.58)	1.28 (1.29–1.37)
<i>P</i> -trend	0.72	0.75	0.02	0.74	0.05	0.26	0.36	0.50
Factor 3, Purchased healthy foods								
Q1	7.71 (6.19–9.52)	9.8 (9.4–10.3)	7.6 (7.2–8.0)	3.1 (2.9–3.3)	119 (117–121)	2.98 (2.82–3.11)	1.48 (1.42–1.55)	1.30 (1.21–1.39)
Q3	7.43 (6.10–9.24)	9.4 (9.0–9.8)	7.4 (7.0–7.8)	3.0 (2.8–3.2)	118 (116–120)	3.00 (2.87–3.11)	1.58 (1.50–1.63)	1.23 (1.15–1.32)
Q5	9.43 (7.52–11.71)	9.2 (8.4–9.6)	6.9 (6.5–7.3)	3.0 (2.8–3.2)	118 (117–120)	2.87 (2.75–3.00)	1.53 (1.48–1.61)	1.27 (1.19–1.36)
<i>P</i> -trend	0.39	0.01	0.01	0.52	0.97	0.09	0.23	0.65
Factor 4, Beverages and sweets								
Q1	9.62 (7.71–12.00)	9.9 (10.3–9.4)	7.3 (6.9–7.7)	3.9 (2.8–3.2)	119 (117–121)	2.90 (2.77–3.03)	1.55 (1.50–1.63)	1.29 (1.21–1.38)
Q3	7.71 (6.19–9.52)	9.2 (8.8–9.6)	7.2 (6.8–7.6)	3.0 (2.9–3.2)	119 (116–120)	3.08 (2.95–3.21)	1.55 (1.48–1.61)	1.24 (1.16–1.33)
Q5	7.14 (5.71–8.86)	9.6 (9.2–10.1)	7.3 (6.8–7.6)	3.0 (2.8–3.2)	118 (116–120)	3.11 (1.98–3.26)	1.48 (1.42–1.53)	1.27 (1.19–1.34)
<i>P</i> -trend	0.04	0.48	0.87	0.19	0.18	0.02	0.28	0.52

<sup>1</sup> Values are adjusted means (95% CI). Adjusted means are computed based on the multiple regression models of the dependent variables on categorized food factor quintiles (dummy variables for each quintile and reference group = Q1), adjusting for age, gender, BMI, MET, smoking and drinking status, education level, total energy intake, cancer status, baseline CVD, hypertension, and cholesterol medication use.

<sup>2</sup> The *P*-value in *P*-trend rows represents the significance level for the trend in each risk factor and marker (adjusted for the same covariates) tested, using the quintile variable as a linear term

<sup>3</sup> The models for all inflammatory markers exclude participants with CRP > 10.

<sup>4</sup> The CRP and TG variables were log-transformed to compute the adjusted means. The means presented were then back-transformed by  $e^{\text{mean}(\log \text{CRP})}$ .

This explanation is supported by the lower drinking and smoking rates across increasing quintiles of this pattern. The lower carbohydrate, especially sucrose, with increasing quintiles may reflect the consumption of fewer sweets, flavored drinks, and soda pop, and the increasing fiber density may reflect a higher intake of fruits and vegetables, dark bread, and hot cereal. However, although *trans*-fat intake was lower and PUFA was higher with increasing quintiles, total and saturated fat intake was also higher with increasing quintiles, consistent with the increasing protein intake. The limited food supply available for purchase in Alaska may make following a healthy purchased diet difficult. In particular, saturated fat is the cooking fat most commonly available.

The high percentage of energy from sucrose in the beverages and sweets dietary pattern, despite a low percentage of energy from carbohydrates, reflects the high intake of candy bars, sugar, and syrup and the low intake of complex carbohydrates. The high percentages of energy from SFA and cholesterol density across increasing quintiles of this pattern reflect the positive association with store-bought animal fats. The low-fiber density across increasing quintiles of this diet is consistent with the negative association with fruits and vegetables.

As a whole, the patterns observed in this analysis are consistent with earlier analysis of nutrient intake in this sample of Alaska Eskimos (24). Although native foods accounted for only 15% of the energy consumed, they contributed disproportionately more protein, fat, MUFA, and PUFA, (n-3) fatty acids, vitamin B-12, and iron than did store-bought foods. Generational differences were observed in the proportion of nutrients consumed from native foods. These results are consistent with

studies of other Inuit groups. Studies of Yukon First Nations, Dene/Métis, and Inuit cultural groups have demonstrated extensive knowledge of diverse food sources, unique foods with exceptional nutrient quality, and unique patterns of food use incorporating varying levels of local cultural food with purchased food (33,34). Previous studies have described the association between fish intake as part of a traditional diet and a favorable coronary risk factor profile. The Japan Public Health Center-Based Study Cohort I, a population-based sample of participants born between 1930 and 1949 (ages 40–59 y), had lower hazard ratios in the highest (8 times/wk or median intake = 180 g/d) compared with the lowest (once a week or median intake = 23 g/d) quintiles of fish intake (35).

Analyses of the associations of the dietary patterns with risk factors confirm previous suggestions from analyses of individual nutrients that consumption of traditional Alaskan foods is associated with a desirable cardiovascular risk factor profile (9–12). Our observation of decreasing blood pressure and a trend toward decreasing HDL-C with higher quintiles of the traditional diet is also consistent with our previous report showing an association of (n-3) fatty acid intake with metabolic syndrome (11). We observed associations of the traditional dietary pattern with slightly increasing LDL-C; trials of (n-3) fatty acids also have been shown to increase LDL-C (1). We are investigating the latter association further through an examination of relations among dietary patterns and lipoprotein subfractions.

The beverages and sweets diet was positively associated with LDL-C levels across quintiles, consistent with the higher intakes of SFA, TFA (*P* = 0.07), and cholesterol. The purchased healthy diet was associated with a trend toward lower LDL-C. Although

this diet was associated with lower TFA intake, intakes of both SFA and cholesterol were higher across quintiles. LDL-C levels may be determined also by variations in other components, such as fiber, or by differences in physical activity.

It must be emphasized that along with changes in dietary intake there have been dramatic changes in activity in this population that could also explain the associations with risk factors. This change in activity is associated in some individuals with transition to employment. In those maintaining subsistence lifestyles, tools and automated equipment have resulted in energy excess. In all cases, there has been increased access to heated environments, thus again resulting in changes in energy balance (36).

Our exploration of relations with inflammatory markers showed few clinically meaningful associations. The purchased healthy diet was associated with lower homocysteine and fibrinogen and the Western diet was associated with higher homocysteine levels. This association may have resulted from the low fruit and vegetable intake in the Western diet and, therefore, the lower folic acid content. However, differences in homocysteine levels between quintiles were small. The U.S. FDA authorized the addition of folic acid to enriched grain products in March 1996, with compliance mandatory by January 1998. The effects over time of folic acid fortification on cardiovascular health parameters may be elucidated in future analyses from this cohort. Data for CRP showed no meaningful relations; CRP was lower in increasing quintiles of the beverages and sweets diet, but the differences were small. Some studies have shown a significant association between diet and CRP, as well as other inflammatory markers (15,37), but in another study, the associations were not significant (38). To the best of our knowledge, no previous study has explored the association between diet and pathogen burden; there were no associations with any of the diet patterns. Thus, the data suggest that diet has only a small role in affecting inflammatory markers; in this population, the high pathogen burden and inflammation must be attributable to other factors.

This study's strengths include the large, population-based sample, a dietary instrument validated for use in this population, and standardized methods for collection of risk factor data. This study is limited by its cross-sectional design. Associations between macronutrient intakes, CVD risk factors, and inflammatory markers must be interpreted with caution, given the possibility of reverse causation. A potential flaw in the analysis of food frequency data is that a single recipe or food was used for all consumers. We used one recipe for *agutuk* and applied it to all. There may be some inaccuracy in grouping *agutuk* made with store-bought hydrogenated vegetable fat with the *agutuk* made with a traditional fat.

In summary, the identification of distinct dietary patterns reflecting the changing lifestyle of Alaska Eskimos afforded a tool to elucidate dietary changes. This analysis has shown beneficial associations of the traditional diet with CVD risk factors and should be useful in future longitudinal analyses of determinants of chronic diseases. Our data also can be useful to practitioners in targeting educational messages for individuals' specific eating patterns and to community planners in encouraging the availability of healthy foods for those unable to obtain traditional foods.

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