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DIET AMONG SIBERIAN YUP'IKS OF ALASKA AND THE IMPLICATIONS FOR CARDIOVASCULAR DISEASE

A

THESIS

Presented to the Faculty
of the University of Alaska Fairbanks
in Partial Fulfillment of the Requirements
for the Degree of

DOCTOR OF PHILOSOPHY

By

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Fairbanks, Alaska

May 1996

DIET AMONG SIBERIAN YUP'IKS OF ALASKA

AND THE IMPLICATIONS FOR CARDIOVASCULAR DISEASE

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ABSTRACT

I investigated dietary factors associated with cardiovascular disease and their relation to blood lipids among Siberian Yup'iks. This study was prompted by reports of increasing mortality from cardiovascular disease in Alaska Natives and the need to know to what extent their unique diet may influence cardiovascular disease. Information on dietary intakes was collected in 1992 using two recall methods, from over one-half of the Siberian Yup'ik Eskimos (n = 65) ≥ 40 years-of-age in Gambell, Alaska, as part of a comprehensive screening for diabetes and cardiovascular disease. Based on analysis of 29 nutrients, mean daily intakes of monounsaturated fats and antioxidants -- vitamin E, selenium, and vitamin C (among men)-- exceeded US recommendations. Mean intake of n-3 fatty acids (7.0 g/d) exceeded the level associated with favorable physiologic responses (> 3 g/d) and was comparable to that of Greenlandic Eskimos (8.6 g/d). Although fat intake was high (44% of total energy for men, 42% for women), saturated (11%) and polyunsaturated fats (8%) were not different (P > 0.05), but energy from monounsaturated fat was greater (18% vs. 13%, P < 0.001) than the general US population (NHANES III). Native foods, including walrus, seal and whale, contributed 25% of the energy, > 50% of the protein, n-3 fatty acids, arachidonic acid, cholesterol, iron and vitamin B-12, and all of the eicosapentaenoic acid. Nonnative foods were frequent sources of saturated fats. Mean intakes of saturated fat and cholesterol exceeded recommendations. Correlations between foods and blood cholesterol, LDL, HDL, triglycerides and LDL-HDL ratio produced differences by age and sex. Multicollinearity occurred among several nutrients,

including α-tocopherol and n-6 fatty acids (r = 0.888). When multiple regression was applied among all participants, α-tocopherol and fresh bird were associated with reduced LDL-HDL ratio; body-mass index, pizza and syrup were positively associated. Adding obesity to this equation increased the percent of variation explained (42% to 59%). Dietary advice includes maintaining desirable weight, consuming a diet moderate in fat, rich in monounsaturated fats and low in saturated fats, and foods rich in n-3 fatty acids - - e.g. Native foods - - and vitamin E.

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ABBREVIATIONS USED

AA	Arachidonic acid
aa	Ascorbic acid
AHA	American Heart Association
AI/AN	American Indian/Alaska Native
AN	Alaska Native
apoB	Apolipoprotein B
apoE2	Apolipoprotein E2
CD	Cardiovascular disease
CHD	Coronary heart disease
CHO	Carbohydrate
CVD	Cerebrovascular disease
d	Day
DGLA	Di-homogammalinolenic acid
EFA	Essential fatty acids
EPA	Eicosapentaenoic acid (C20:5)
GLA	Gamma linolenic acid
HDL	High density lipoprotein cholesterol
HDLc	High density lipoprotein cholesterol
IA	Index of atherogenicity
ICD-9	International Classification of Diseases, 9th Revision
IGT	Impaired glucose tolerance
IHD	Ischemic heart disease
IHS	Indian Health Service
IT	Index of thrombogenicity
LA	Linoleic acid
LDL	Low density lipoprotein cholesterol
LDL-HDL	Ratio of low density lipoprotein cholesterol to high density lipoprotein cholesterol
MI	Myocardial infarction

ABBREVIATIONS USED (continued)

mtDNA	Deoxyribonucleic acid of the mitochondria
MUFA	Monounsaturated fatty acids
NCEP	National Cholesterol Education Program
NHANES II	Second National Health and Nutrition Examination Survey (1976-1980)
NHANES III	Third National Health and Nutrition Examination Survey (1988-1994)
NIDDM	Noninsulin-dependent diabetes mellitus
n-3	Omega-3 form of polyunsaturated fatty acids
n-6	Omega-6 form of polyunsaturated fatty acids
PS ratio, or P/S	Polyunsaturated fatty acid to saturated fatty acid ratio
PUFA	Polyunsaturated fatty acids
RDA	Recommended Dietary Allowances
SFA	Saturated fatty acids
SMFI	Sea mammal fish index
TFA	*Trans*-fatty acids
y	Year

CHAPTER 1
OVERVIEW

PURPOSE OF THESIS

The purpose of this study is to describe the intakes of dietary factors related to heart disease, especially fats, among a homogeneous group of Alaska Natives and correlate the dietary factors with physiological-risk factors for heart disease. Specific risk factors include hypercholesterolemia, hypertriglyceridemia, elevated low density lipoprotein (LDL, See Abbreviations Used, pp xiv-xv), reduced high density lipoprotein (HDL) and elevated LDL-HDL ratio. The unique diet of Alaska Natives and altered prevalence of heart disease from that of the United States (US) population as a whole and from other Native populations make this investigation worthwhile for two reasons: 1) potential risks of heart disease may be avoided by Alaska Natives if those risks are better understood; and 2) information learned about dietary practices of Alaska Natives and their relation to heart disease may provide information for other populations.

BACKGROUND AND LITERATURE REVIEW

History of dietary risk factors in cardiovascular disease

This chapter summarizes literature on diet and cardiovascular disease for 35 nutrients and four foods, as it pertains to Siberian Yup'iks. There are 93 nutrients of importance in human nutrition; at least 35 of these have been investigated in relationship to cardiovascular disease (Willett, 1990). Alcohol, β-carotene, calcium, carbohydrates, cholesterol, fats, fiber, folic acid, iron, magnesium, potassium, animal protein, sodium, selenium,

vitamins A, C, E, B-6 and B-12 have been investigated (TABLE 1-1). Total-energy balance, which involves dietary intake with other interdependent factors, also has been studied. Specific foods (e.g., eggs, fish, fish oils, and meat) have been investigated, but data relating foods to risk of cardiovascular disease are limited (TABLE 1-2).

Of the nutrients reviewed (TABLE 1-1), the evidence is most consistent for a beneficial effect on cardiovascular health from consumption of alcohol, linoleic acid, n-3 fatty acids, polyunsaturated and monounsaturated fatty acids, whereas there is a detrimental effect associated with consuming dietary cholesterol, myristic acid, and saturated fatty acid. There is some evidence for varying effects on cardiovascular health by soluble fiber, iron, β-carotene, animal and vegetable protein, selenium, vitamins B-6, B-12, folic acid, and *trans*-fatty acids. Reports of associations for ascorbic acid, sucrose, dietary fiber, and stearic acid have been conflicting or correlations were absent. Nutrients associated with hypertension (e.g. calcium, magnesium, potassium, and sodium) are mentioned but not discussed in detail here.

Diet has been associated with the risk of heart disease for the last 80 years since it was demonstrated that cholesterol was the dietary component responsible for experimentally induced hypercholesterolemia and atherosclerosis in rabbits (Anitschkow, 1967, National Research Council, 1989*a*). Few relationships are as well established as the association between levels of total-blood cholesterol and risk of coronary heart disease(CHD)(Willett, 1990). Associations also have been shown between diet and blood cholesterol in both individuals and populations, leading to the conclusion that abundant indirect evidence

supports the hypothesis that dietary lipids are a causal determinant of CHD. The classic "diet-heart" hypothesis is: high intake of saturated fats and cholesterol and low intake of polyunsaturated fats increase the level of blood cholesterol, which leads to development of atheromatous plaques. Accumulation of plaques narrows coronary arteries, reduces blood flow to the heart and myocardial infarction occurs. Serum cholesterol is clearly causally related to risk of CHD and a reduction of serum cholesterol, which can be achieved by alterations in dietary lipids, generally reduces the incidence of CHD. But a direct link between diet alterations and reduced heart disease has not been made. The hypothesis remains untested by the most rigorous scientific standards (i.e., replicated randomized experiment; Willett, 1990, McNamara, 1994). Yet a massive body of indirect evidence provides general support for the hypothesis.

Several observations, however, do not fit the diet-heart hypothesis (Willett, 1990). While CHD increased dramatically in the US during the first one-half of this century, consumption of total and saturated fats increased only slightly and polyunsaturated fat intake rose two to three fold. Since the late 1960s, mortality due to cardiovascular disease in the US has decreased steadily while dairy fat and lard have been partially replaced by vegetable fat, thus increasing intake of polyunsaturated fat and only slightly decreasing intake of saturated fat.

Other dietary components, such as alcohol (Hegsted and Ausman, 1988) or fiber (Pilch, 1987), may be related to cardiovascular disease, in addition to the fats described in the diet-heart hypothesis. Dose-response relationships between specific fatty acids and cholesterol intakes and

rates of CHD also are not clear. Willett (1990) concluded that further epidemiologic data were needed to provide sound dietary guidance for persons wishing to reduce their risk of CHD. Nonetheless, several groups have formulated dietary recommendations based on the available data (National Research Council, 1989*a*, National Cholesterol Education Program, 1994).

Much attention has been given to fat in the diet as a cause of atherosclerosis and cardiovascular disease. A brief synopsis of dietary fats and their relation to cardiovascular disease follows.

Fats

Associations between total fat and cardiovascular disease were noted prior to World War II and a large body of literature has been published and reviewed (National Research Council, 1989*a*). Epidemiological studies among countries using data on food disappearance revealed total fat intake was positively related to atheroma (Keys et al. In Wood and Oliver, 1992). Scrimshaw and Guzman (1968) reported a correlation (r = 0.67) between the percentage of energy from fat with atherosclerotic lesions based on data from 12 countries. High intake of fat, especially saturated fat, combined with high cholesterol intake, is atherogenic for many species of animals (Feldman, 1994). Kuller (1992) even proposes that atherosclerosis, the basic underlying pathology of CHD, be considered an example of a common source epidemic, the common source or agent being the amount and type of fat and cholesterol in the diet.

McNamara (1994), however, maintained that a common misconception is that any reduction in total fat has beneficial effects by lowering plasma cholesterol. He stated

that available data indicate that the key to reducing total cholesterol and LDL cholesterol levels was to lower intake of saturated fat, primarily myristic and palmitic acids. Little benefit in lowering LDL cholesterol is achieved by reducing unsaturated fatty acid intake as a means to reduce total fat intake, as unsaturated fat lowers plasma cholesterol and LDL (See Keys et al., 1965, Hegsted et al., 1965, National Research Council, 1989a). Hegsted et al. (1965) examined the effects of different types and amounts of fat and cholesterol on serum cholesterol. Amounts of dietary fat (within the limits of 22-40% of energy) did not influence concentration of serum cholesterol, whereas the proportion of saturated and unsaturated fatty acids was a major determinant.

Polyunsaturated fats

Experimental studies have shown that total cholesterol levels in plasma fall as the percentage of calories from polyunsaturated fat increases (Keys et al., 1965, Hegsted et al., 1965, McNamara, 1994, National Research Council, 1989*a*). Epidemiologic studies comparing populations from 18 countries further suggested that as the percentage of calories from polyunsaturated fats increases, mortality from CHD decreases, but the relationship was not statistically significant (Hegsted and Ausman, 1988; $r = -0.342$, $P > 0.05$). Based on findings of studies conducted among subjects on metabolic wards, it has been estimated that for every 1% increase in calories from polyunsaturated fats, levels of plasma cholesterol levels are lowered an average of 0.04 mmol/l (1.5 mg/dL)(Hegsted 1965, National Research Council, 1989*a*).

Sinclair (1980) concluded that atheroma and coronary

thrombosis were related to a relative deficiency of essential fatty acids and not to the total fat in the diet as other investigators had stated in the 1950's. He based this interpretation on the physiologic mechanisms of n-6 (linoleic) and n-3 (linolenic) fatty acids in phospholipids of cellular membranes.

According to Sinclair (1980), there are two classes of essential fatty acids (EFA), linoleic or n-6, and linolenic or n-3. These fatty acids are essential in glycerophosphatides (phospholipids) of cellular membranes, for transport and oxidation of cholesterol, and the formation of prostaglandins. In deficiency, cellular membranes are imperfectly formed, thus increasing the susceptibility of the cell to insults and permeability.

Nikitin et al. (1991) compared the diets of two native populations in Chukotka and reported a lower prevalence of ischemic heart disease among 30-59 year-old males in coastal villages than in a comparable population residing on the tundra. They attributed this finding partially to diet. Although coastal natives consumed more calories and fats than tundra inhabitants, their intakes were rich in polyunsaturated fatty acids, mainly from the meat of sea mammals.

Saturated fats

Saturated fats are twice as effective in raising serum cholesterol as polyunsaturated fats are in lowering it, based on human studies (Keys et al., 1965, Hegsted et al., 1965, McNamara, 1994, National Research Council, 1989a). Keys (1980) described a positive association of intake of saturated fats with coronary death in seven countries. Increases in saturated fat as the percentage of calories

paralleled increases in mean relative weight, serum cholesterol and incidence of CHD among Japanese living in Japan, Hawaii and California (Kato et al. 1973, Willett, 1990).

Monounsaturated fats

Evidence and opinion conflict regarding the role of monounsaturated fatty acids (TABLE 1-1). Intake of monounsaturated fatty acids in olive oil for example, has been associated with a remarkably low rate of coronary mortality among residents of Crete (Katan et al., 1995). In experiments where body weight was kept constant and olive oil was substituted for hard fats rich in saturated or *trans*-fatty acids, a more favorable profile of lipoproteins was produced over that achieved by replacing hard fats with carbohydrate. The monounsaturated diet produced lower LDL, cholesterol, and triglycerides, and higher HDL. At least one group (Mensink et al., 1989) observed reduced serum cholesterol and very low density lipoproteins (VLDL) on a diet rich in monounsaturated fatty acids. HDL, type 3 (HDL_3) rose as did apolipoprotein A-1 when compared with diets of high complex carbohydrate and high saturated fat.

N-3 fatty acids

N-3 and n-6 fatty acids also have been investigated, based on findings among Greenlandic Eskimos. These Eskimos consumed high levels of fish and sea mammals, which are rich sources of n-3 fatty acids, and experienced a low prevalence of cardiovascular disease (Bang et al., 1971). These investigators attributed the association to the particularly active antithrombotic activity of n-3 fatty acids both through anti-aggregating prostacyclin I_3 (PGI_3) and

thromboxane A_3(TXA_3), which does not have platelet aggregating properties (Dyerberg et al., 1978).

Bang et al. (1980) concluded that the rarity of ischemic heart disease in Greenlandic Eskimos might be partially explained by the antithrombotic effect of the long-chained polyunsaturated fatty acids, especially eicosapentaenoic acid (EPA) prevalent in diets rich in marine oils. These authors described a lower intake of saturated fats, and higher intakes of polyunsaturated and monounsaturated fatty acids among Greenlandic Eskimos, in comparison to intakes of Danes, and related their diet to blood lipids collected in previous examinations. Among 130 Greenlandic Eskimos measured in 1970, the investigators noted low blood cholesterol, triglycerides, low-density and very low-density lipoproteins, and for males, increased high-density lipoproteins. Bang et al. described the content of 20 fatty acids in the diets of 25 Greenlandic Eskimos, who hunted and fished and 25 wives of hunter-fishermen, and reported high levels of n-3 fatty acids and monounsaturated fatty acids (except for palmitoleic and oleic acids). Compared with Eskimos in Denmark and non-Eskimos, Greenlandic Eskimos had high levels of n-3 fatty acids, especially eicosapentaenoic fatty acid (EPA) and low levels of n-6 fatty acids, especially arachidonic acid (AA).

Parkinson et al.(1994) documented significantly ($P < 0.05$) lower levels of n-6 fatty acids and significantly ($P < 0.001$) higher levels of n-3 fatty acids in the plasma of residents of coastal villages in contrast to residents on a river. They also reported high levels of n-3 fatty acids in both groups of Alaskan Yup'ik Eskimos. Residents of coastal villages consumed marine mammals and seal oil more frequently than did residents of river villages.

Although the concept of a diet high in fish (presumably high in n-3 fatty acids) being protective against heart disease has gained in popularity, not all investigators agree. Kromhout et al. (1985) reported that Dutch men who consumed > 30 g of fish/d halved the risk of death from coronary heart disease. According to Ascherio et al. (1995), however, more is not necessarily better. Based on follow-up of 44,895 male health professionals in the US, they concluded that increasing fish intake from one to two servings to five to six servings per week did not substantially reduce the risk of coronary heart disease among men who were initially free of cardiovascular disease.

Multiple dietary factors

There is growing acknowledgment that multiple dietary factors influence the development of cardiovascular disease. Hegsted and Ausman (1988) correlated consumption of fat and alcohol with CHD from 18 countries. They noted simple correlations with CHD for saturated fat, $r = 0.71$, polyunsaturated fat, $r = -0.34$ and total alcohol, $r = -0.58$; but incorporating all three factors in multiple regression improved the correlation ($R^2 = 0.846$), demonstrating the importance of considering more than one factor simultaneously in explaining the variation in CHD among populations.

Similarly, Ulbricht and Southgate (1991) described seven dietary factors linked to coronary heart disease and concluded that an interrelationship of factors was needed, rather than single answers, to solve the question of risk factors. Two dietary factors are positively related to CHD, i.e. cholesterol-raising saturated fatty acids which are atherogenic, and saturated fatty acids which are

thrombogenic. Five dietary factors are negatively related; polyunsaturated fatty acids (PUFA) of the n-6 series, PUFA of the n-3 series, monounsaturated fatty acids(MUFA), dietary fiber, and antioxidants.

To consider several dietary factors that contribute to cardiovascular disease, Ulbricht and Southgate (1991) developed indices of atherogenicity[1] (IA) and thrombogenicity[2] (IT), which they applied to both foods and diet intakes. They advocated discarding the ratio of polyunsaturated:saturated fatty acid (PS) in favor of these new indices. The Eskimo diet (Bang et al., 1980) had an IA of 0.39, lower than the Danish (1.29) and British (0.93) diets. The IT for the Eskimo diet was 0.28 compared with Danish (1.51) and British (1.21) diets. They concluded that, without considering the multiple factors contributing to cardiovascular disease, future research would continue to produce conflicting results.

Hansen et al. (1994) proposed investigations be conducted in Greenland on n-3 PUFAs, MUFAs and antioxidants (specifically selenium) to elucidate the role of individual dietary components and their interactions in relation to IHD. Serum cholesterol, especially LDL-cholesterol, is regarded as an index of risk for ischemic heart disease

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[1] *Index of Atherogenicity (aS'+bS" + cS"'/ dP +eM +fM') where S' = C12:0, S" = C14:0 and S"' = C16:0; P = sum of n-6 and n-3 PUFA; M = oleic acid (C18:1); and M' = sum of other MUFA; a-f are empirical constants; b = 4 because myristic acid has about 4 times the cholesterol-raising potential of palmitic acid; others are unity lacking evidence for any other value.*

[2] *Index of thrombogenicity (mS^{iv}/nM + oM' +p(n-6) +q(n-3) + n-3/n-6) where S^{iv} = sum of C14:0, C16:0 and C18:0; n-6 = n-6 PUFA; n-3 = n-3PUFA. M and M' as before, m,n,o,p, and q are unknown empirical constants. m = unity; n,o,p = 0.5 because MUFA and n-6 PUFA are less antiatherogenic than n-3 PUFA; q = 3).*

(IHD). Although epidemiological studies have connected a high dietary intake of n-3 fatty acids with a low occurrence of IHD (Bang and Dyerberg, 1972, Bang et al.,1980), intervention studies have not demonstrated that n-3 acids can lower LDL-cholesterol (Hansen et al., 1994), whereas monounsaturated fatty acids (MUFAs), previously believed to be neutral, have been shown to lower LDL (Mensink et al., 1989). The epidemiological observations probably reflect the combined effect of both n-3 polyunsaturated fatty acids (PUFAs) and MUFAs (Hansen et al., 1994). Based on a recent hypothesis that oxidized LDL is involved in the earliest lesion of atherogenesis, the antioxidant balance also may be an important factor for IHD (Hansen et al., 1994). Hansen et al. (1994) suggested that epidemiological studies in Greenland could shed important light on the role of individual dietary components and their interactions because the traditional Greenlandic diet is rich in n-3 PUFAs, MUFAs and antioxidants (selenium). Diets of Alaska Natives also meet these criteria and epidemiological investigations in Alaska could shed light on the role of diet and cardiovascular disease.

The diet-heart relationship is much more complex than previously recognized and several new directions for dietary studies of coronary heart disease have been recommended (Ascherio and Willett, 1995). These include defining the magnitude of effects of dietary saturated fat and cholesterol, the protective effects of antioxidants, particularly vitamin E, the adverse effects of *trans*-fatty acids, the ideal amount of n-6 and n-3 polyunsaturated fats in the diet, the comparative benefits of high monounsaturated fat diets vs. low total fat diets, and identifying the active factors in diets high in plant-based

foods. Dietary recommendations for preventing CHD will need to recognize the potential associations of these aspects of diet with diseases other than CHD.

Despite numerous publications and hypotheses, the elucidation of the exact relationship of diet and cardiovascular heart disease remains to be done (Willett, 1990). Since 1990, studies have been conducted on different nutrients, including *trans*-fatty acids, antioxidants, and folate (TABLE 1-1), and on several foods (TABLE 1-2). Despite this research, a definitive explanation of the relationship has yet to be discovered.

Dietary practices of Alaska Natives

At least seven papers have described the dietary practices of Alaska Natives over the last 55 years. Any comparisons among these studies should be undertaken with caution. A study in one region may not be comparable with those in other regions because of differences in local foods, survey methodology, age of subjects, and data bases of food composition which vary in levels of completeness, descriptions of foods and nutrients. These variables make comparisons of studies over time or between regions only rough estimates at best.

Most authors described the intakes of energy and its sources, particularly fat, and the reliance on traditional foods. Rodahl (1954*a*, 1954*b*) studied 45 Alaskan Eskimo men and women from four northern communities (Anaktuvuk Pass, Barter Island, Kotzebue, and Gambell) in 1950-52, and reported they consumed 105 g fat/d and 37% of their calories from fat, comparable to "normal white men living in Alaska" who consumed 37.5%. A "larger," unreferenced study of Alaskan Eskimos averaged 139 g (40% of calories) in their

daily consumption of fat (Rodahl, 1954*a*).

In 1956-61, Heller and Scott (1967) conducted the extensive Alaska Dietary Survey, to: determine the food habits of Alaskan Eskimos and Indians; estimate the degree of their dependence on local food; estimate the adequacy of the diet; and predict medical or public health problems, which might arise from inadequate diets. They collected 4,840 diet records, most of them of 7-day duration on a seasonal basis, for both sexes and for all ages. Eleven villages, nine Eskimo and two Athapascan Indian, were included (Point Hope, Noatak, Shishmaref, Shungnak, Hooper Bay, Newtok, Kasigluk, Akiak, Napaskiak; Huslia and Allakaket).

Among their 16 conclusions were: 1) a wide range of mean daily intakes of nutrients was observed indicating enormous fluctuation in family and village food supplies throughout the year; 2) intakes of protein and niacin were generally high; calories, calcium, and ascorbic acid were low in ≥ 75% of the diets; 33% were low in vitamin A and thiamin, 25% were low in riboflavin; 33% of adolescents, pregnant and lactating women and women > 60 years-of-age and preschool children in southwest Alaska were deficient in iron; 3) extremely high mean intakes of iron were observed among all age levels except infants, for those living in coastal areas where meat of sea mammals was a dietary staple; 4) there was significant seasonal variation in vitamins C and A - - the highest levels observed in the summer and autumn; 5) the fat content of the diets was not considered excessive, slightly more than 33% total energy for adults; 6) diets of Eskimos and Indians were composed of imported and locally available foods, the proportions varying with geographic location. Local foods supplied one-

half or more of the protein, iron, vitamin A, riboflavin, niacin and ascorbic acid, whereas imported foods supplied most of the carbohydrate and calcium plus one-half or more of the thiamin, calories and fat; 7) regional differences in distribution of local and imported foods occurred. Sea mammal fats were almost equally as important as imported fats in diets of coastal Eskimos; in general, meat was the important protein source in northern Alaska, fish in the southwestern Alaska. Indians obtained most of their vitamin A from imported foods, whereas Eskimos depended on local foods for this nutrient. Mean daily intakes of vitamin A were highest in villages where sea mammal oils and local green plants ("greens") were eaten in abundance. Most of the ascorbic acid in Indian diets came from imported sources while at certain Eskimo villages - - northern and southwestern - - local greens and berries were the major source; 8) traditional seasonal food quest activities of former years have been greatly modified in recent years by intercultural contacts, more permanent village living and increased seasonal (mostly summer) employment of many men; 9) the marginal money economy limited use of imported foods primarily to milk, grains, sugars and fats; 10) the major changes in diets since aboriginal times included a) increase in carbohydrate from 10-33% of total calories; b)increased use of saturated fats, largely imported, particularly by the coastal and tundra Eskimos; c) increased calcium primarily because of use of milk; and d) more limited use of local foods.

According to Bang and Kristoffersen (1972), marked change in diet was noted between 1955-7 and 1965 in the diets of 90 inland Eskimos at Anaktuvuk Pass. The first report based on weighed portions and the second report based

on "the interview method," indicated that energy from protein decreased by about 50% and that from carbohydrate increased by nearly 50% (accompanied by an almost 90% increase in the DMFT [the average rate of decayed, missing and filled permanent teeth per person]). The percent of energy from fat was 41% in the first investigation, 40% in the second.

Knapp and Panruk (1978) conducted 24-h diet recalls among 269 residents (≥2 years-of-age) in five of the villages surveyed by Heller and Scott (1967). Compared with Heller and Scott's results, they reported a lower consumption of protein (17% of energy vs. 29%) and fat (35% vs. 40%), and a greater consumption of carbohydrate (50% vs. 31%). Mean energy intake was 7% less than the earlier investigators reported.

A more recent study described the nutrient and food intakes of 351 Native adults in eleven Alaskan communities (Bethel, Mountain Village, Kwigillingok, Dillingham, Pilot Point, Pedro Bay, Anchorage, Sitka, Kake, Kotzebue and Selawik) (Nobmann, 1989, Nobmann et al.,1992). Participants completed up to five 24-h recalls throughout the seasons. The participants consumed 37% of their energy from fat, which was similar to the general US population represented by participants in the National Health and Nutrition Examination Survey (NHANES II). Alaska Natives consumed six times as much fish as the general US population (Nobmann et al., 1992).

Other recent investigations compared dietary intakes among Alaska Natives with diabetes and obesity, two known risk factors for cardiovascular disease. Murphy et al. (1995) compared data from a food frequency questionnaire which was self-administered (15 foods) with levels of blood

glucose from 53% of residents ≥ 20 years-of-age in 15 southwestern Alaskan villages. Murphy et al. reported significantly higher intake of beef and pork (46.6% vs. 29.4% of intake frequencies each day, $P = 0.005$) among 42 Yup'ik Eskimos, who were glucose intolerant (either noninsulin-dependent diabetes mellitus [NIDDM] or impaired glucose tolerance) compared with 853 euglycemic Eskimos. The Eskimos with glucose intolerance also reported eating significantly less salmon and other fish (6.6% vs. 19.1% $P = 0.01$) and significantly more white bread (51% vs. 31.6% $P = 0.029$) than those who were euglycemic. Significantly more people were overweight than 25 years ago. Persons with glucose intolerance were significantly more overweight. Athapascan Indians had twice the Yup'ik Eskimo rate of NIDDM, with significantly more frequent non-indigenous food intake, and less frequent indigenous carbohydrate and fat intake.

In summary, total fat intake among Alaska Natives during the last 50 years has been comparable or slightly higher than their non-Native contemporaries. There appears to be a general trend towards an increase in imported foods and in the proportion of carbohydrate in the diet and perhaps a decrease in the total energy intake. The apparent increase in the prevalence of overweight individuals, however, may indicate that energy intake has not declined enough to maintain desirable body weight. Traditional foods are still a significant part of the diet indicating differences in sources of nutrients such as fats from those of non-Natives.

Cardiovascular disease in Alaska Natives

The early references to a relationship of diet to cardiovascular disease in the North are generally limited to observations of clinicians, serving Eskimos in Canada, Greenland and Alaska (Rodahl, 1954*a*). Although these references point to cardiovascular disease present in the 1940's among northern indigenous people, others noted lower cardiovascular disease among Greenlandic Eskimos when compared with populations in western Europe (Kromann and Green, 1980). Kromann and Green (1980) described three acute myocardial infarctions where 40 were expected by Danish standards, in a population of 1,800 Upernavik District Greenlandic Eskimos, whose activities included whaling, sealing, fowling and fishing. Sinclair (1953) urged further investigation based on summarizing several early reports that Eskimos consumed an unusually high quantity of fat, but had normal levels of serum total cholesterol.

Historically, cardiovascular disease was thought to be low among Alaska Natives (Rabinowitch, 1936), but it was considered common by Gottman (1960) based on autopsies from three Alaska Native Service hospitals. Arthaud (1970) also documented cardiovascular disease based on 339 autopsies from different Alaskan regions. Maynard et al. (1967) reported the Native rate of 2.0 to 2.2 deaths/1000 population considerably lower than the 1960 rate of 3.8/1000 for the US population in the age group 40-64 years. In a 1950-1952 study, Rodahl (1954*a*) concluded on the basis of roentgenological evidence of arteriosclerosis (chest and extremity X-rays examined for arteriosclerosis) in 84 Alaskan Eskimos, that arteriosclerosis was "neither more nor less than what one would expect to find in Whites of similar age groups."

Levels of serum cholesterol were comparable and were in some instances higher than in the non-Native counterparts. This was interpreted differently by different investigators. Wilber and Levine (1950) reported mean cholesterol levels in 70 Eskimos in Point Barrow, Alaska, in 1948 to be 203 mg/100 cc for males and 234 mg/100 cc for females, values that were higher than in white adults (177 mg, sex not reported) and in Canadian Eskimos (141 mg/100 cc, sex not reported). Values were collected in summer when there was an abundance of seal, walrus and polar bear meat. They stated that repeated clinical surveys in the population (not described) indicated an almost total absence of cardiovascular-renal diseases. Wilber and Levine also cited Keys' finding that there was no relation between blood cholesterol and habitual intake of cholesterol among younger and older men. Wilber and Levine (1950) concluded that higher cholesterol levels and supposedly low cardiovascular disease among Eskimos, plus a lack of relationship between blood cholesterol and dietary cholesterol showed "the causative role of serum cholesterol in development of atherosclerosis to be somewhat dubious." The role of high serum cholesterol as a cause of heart disease in this population was questioned as early as 1950. Nonetheless, Sinclair (1953), in summarizing Wilber and Levine's work, pointed out that although they did not give the ages of their subjects, cholesterol levels would hardly appear high compared with Keys' data. In studying 16 healthy Eskimos in 1950-52, Rodahl (1954*b*) reported a mean concentration of serum cholesterol of 203 mg/100 ml (mean age 35 y, sex unstated, some possibly from Gambell), which he indicated was about the same as in normal whites (207 mg/100 ml).

Middaugh (1990) characterized these studies as "fragile

evidence" to document the low incidence of atherosclerosis and cardiovascular disease in Arctic peoples. More specific definitions of heart disease and methodology in recent reports provide a clearer picture of cardiovascular disease.

Using data from 1981-83, Welty and Coulehan (1993) reported a lower rate of mortality from all cardiovascular diseases (including cerebrovascular disease) for Alaska Natives than the general US population (age-adjusted rates of 206.3 vs. 238.3/100,000). When comparing only ischemic heart disease rates (International Classification of Diseases, 9th Revision [ICD-9] codes 410-414), he reported rates for Alaska Natives also were lower (91.1 compared with general US rates of 139.3). But the rate of cerebrovascular disease was higher for Alaska Natives than for the general US population (44.2 vs. 35.8).

Currently, there are two general observations regarding cardiovascular disease among Alaska Natives, 1) mortality rates are lower than among non-Natives, yet 2) mortality rates are not decreasing and may be increasing. Lower relative risk and mortality from cardiovascular disease mortality have been documented among Alaska Natives when compared with other Alaskans (Middaugh, 1990) and other US residents (Davidson et al., 1993; Welty and Coulehan, 1993). Another encouraging study has documented lower prevalence of atherosclerotic lesions in the arteries of Alaska Natives compared with Alaskan non-Natives (Newman et al., 1993).

Middaugh (1990) concluded that Alaska Natives had a lower age-adjusted rate of mortality from atherosclerosis and cardiovascular disease compared with all other Alaskans (162.0 vs. 242.1; Relative Risk (RR) = 0.67). He presented causes of death from death certificates of 3,526 Alaska

Natives, and 9,938 other Alaskans between 1980-86, using ICD-9 definitions and age-adjusting (direct method using the 1980 Census, ICD-9 codes for cardiovascular diseases and atherosclerosis 401-404, 410-414, 429.2, and 440). Mortality rates for Alaska Natives for atherosclerosis and cardiovascular disease were consistently 60% of the death rate of others in all comparisons, with the exception of a slightly higher rate of acute myocardial infarction for female Natives. The death rate from cerebrovascular disease (CVD) rose strikingly with age for both Natives and others.

Davidson et al. (1993) have provided detailed data on mortality from cardiac diseases for indigenous residents of Alaska by major ethnic group based on death certificates. Their results were consistent with those of Middaugh (1990). When rates of different types of cardiovascular disease are age-adjusted and separated, there is a clearer picture of the problem. Mortality rates from all cardiac, ischemic, and rheumatic heart diseases were 80%, 61% and 202% of those corresponding to levels in Alaskan whites, whose cardiac mortality closely profiles US whites. The relative risk of all cardiac disease for Alaska Natives (AN) compared with Alaskan whites was 0.80 ($P < 0.01$), whereas the relative risk for ischemic heart disease was 0.61 ($P < 0.001$). Eight-hundred and twelve Alaska Natives died from all diseases of the heart (ICD-9, 390-429), 430 died from ischemic heart disease, and 30 died from rheumatic heart disease during 1979-1988. Davidson et al. demonstrated a marked variation of mortality rates for IHD among ethnically and geographically distinct populations of Alaska Natives (AN) suggesting influence from both genetic and environmental risks.

The lowest levels of mortality from ischemic heart

disease, < 1/3 that of US whites, occurred in Alaskan Eskimos in southwestern Alaska, an area where salmon consumption was high (Nobmann, 1989) and individuals exhibited high blood concentrations of n-3 fatty acids (Parkinson et al., 1994). Alaska Native men and women, who are Eskimo, had an IHD rate that was markedly lower than that of Alaskan whites (RR = 0.50, $P < 0.001$) and less than one-half the rate of US whites.

There has been a 48% decline in coronary heart disease among the general US population between 1970 and 1985 (Marmot, 1992). Deaths from all cardiac diseases combined in Alaska Natives have actually been increasing over the past 30 years, based on a comparison of 1979-1988 with 1955-65 rates using uniform coding (Davidson et al., 1993). This held in all regions of Alaska but the Southcentral region.

Others describe a steady rate of cardiovascular disease (all causes) among Alaska Natives during the last several years (IHS, 1992, 1994, personal communications A.J. D'Angelo, B. Boedecker), and from 1968 to 1980 (Blackwood, 1982). From 1981 through 1992 the rate of mortality from ischemic heart disease (ICD 410-414) among Alaska Natives was steady ranging from 156/100,000 in 1981 to 145/100,000 in 1992 (age-adjusted). Although these rates are below the US rates, they are not decreasing in contrast with rates for the US population as a whole, which dropped from 242/100,000 in 1981 to 197/100,000 in 1990 (S.W. Butler, Alaska Area Native Health Service, personal communication, 1996). Available data on cardiovascular disease from all causes among Alaska Natives show that the mortality rate is not much different from that of the general US population (148.0 Alaska Natives, 152.0 for US All Races) and almost 100 Alaska Natives/year dying from these diseases (Indian Health

Service [IHS], 1994).

Davidson et al.(1993) further noted that mortality rates from all diseases of the heart were greater among young adult Alaska Native men (age 30-44) than among their white counterparts ($P < 0.001$), indicating a potentially worsening problem, or possibly a different etiology. Welty and Coulehan (1993) also reported the 25-44 yr age group of American Indians and Alaska Natives had a higher death rate from cardiovascular disease than the US population as a whole. These differences in mortality among age groups could be because of differences in current and past eating practices among older and younger men.

Cardiovascular disease elsewhere

Marmot (1992) described the global picture of coronary heart disease as follows: CHD is emerging as a major cause of death in developing countries; rates are rising in many countries of Central and Eastern Europe while rates are falling in the wealthy European countries, North America, Australasia and Japan; within countries, CHD is more common among the less wealthy; and within the UK and USA, the decline in CHD has occurred faster among the higher socio-economic groups. Exploration of the underlying links between social position and CHD should be pursued, Marmot (1992) argued, with special attention to material conditions, social environment, nutrition, early environment and life-style including smoking and exercise.

Between 1970 and 1985 there was a 38% decrease in the already low CHD rates among Japanese men, despite that two-thirds of Japanese men smoke (Marmot, 1992). Marmot proposed that the decline in rates of CHD in Japan was attributable to the background level of risk determined by diet and

plasma lipids. Dietary fat was < 25% of the Japanese diet compared with 42% in the United Kingdom. The polyunsaturated to saturated fatty acid ratio was 1.1 compared with 0.34 in the UK. A reduction in salt intake, and no doubt other factors, may explain this decrease. Also, men in the Ni-Hon-San study of Japanese migrants, who were more traditionally Japanese, had a lower prevalence of CHD than men who were more Western in their culture and social relations, independent of their levels of serum cholesterol. Traditionality was defined as retaining ties to a close-knit community.

Donnan et al.(1994) described mortality from coronary artery disease in Hong Kong as only 1/4 that of northern Europe and the US, based on a case-control study of approximately 1,000 men and women. But it is and probably will remain a major cause of death in Hong Kong. There has been no decline of IHD mortality among the Chinese of Hong Kong, Taiwan and Singapore, unlike the declines in the US, Canada, Australia, Finland, and Britain. Donnan et al.(1994) confirmed the importance of the following risk factors: cigarette smoking, history of hypertension, history of diabetes, body fatness, lack of physical activity, which were all previously described in western populations, plus "more adverse conditions in childhood." This intriguing influence of early life experience was defined as inadequate food intake and less frequent meat in their diets as children.

This work is notable because it was done in a rapidly changing population, possibly similar to Alaska Natives. Donnan et al.(1994) reported higher-income people were at higher risk. Younger people ate more meat and dairy products, which may be contributing to the development of

coronary heart disease, and less fruits and vegetables. Mean percent of energy intake from fat for this population varied between 20% and 30%, which was the same as among case patients. Although a risk factor in other countries, high fat consumption may not play an important role among subjects consuming a traditional Chinese diet.

The Tarahumara Indians of Mexico in the mid 1970's represent the extreme in low intake of fat (12% of energy), low cholesterol (71 mg/d) and saturated fat (2% of energy)(Connor et al., 1978). Their intakes of carbohydrate and fiber were high (75% of energy and 19 g/d, respectively). Although cardiovascular disease was not described, the mean plasma cholesterol was 125 mg/dL, triglyceride 120 mg/dL, lipoprotein cholesterol LDL 87 mg/dL, VLDL 21 mg/dL and HDL 25 mg/dL. Total-plasma cholesterol correlated positively with dietary cholesterol (r = 0.874), which the authors reported was the first time in man such a correlation had been observed. These authors attributed this outcome to dietary cholesterol intake being below the so-called threshold level (which was suggested to be between 100 and 300 mg/d) above which differences in intake do not affect plasma cholesterol concentrations. Cholesterol intakes above 475 mg/d have usually not increased plasma cholesterol concentrations further.

Because 95% of the dietary fat was from vegetable sources, the PS ratio was > 2.0 compared with the US ratio of 0.35 in the 1970s. It is also worth noting that the traditional Tarahumara Indians were described as renowned for their competitive running. There was a virtual absence of hypertension, obesity, and the usual rise with age of the serum cholesterol in adults. Their diet was considered adequate, hypolipidemic and presumably antiatherogenic.

In the US in general, coronary heart disease is the leading cause of death and accounts for 80% of all cardiac deaths (Mahan and Arlin, 1992). The rates of cardiovascular disease in the US population as a whole increased from 1920 through the 1960's to a peak of more than 300 deaths per 100,000 population. From about 1967 through 1983, there has been a decline to an age-adjusted rate of 236.1 per 100,000 population (National Research Council, 1989*a*).

Welty and Coulehan (1993) described the mortality rate from cardiovascular disease for American Indians and Alaska Natives (AI/AN) as 19% lower than the general US rate. Changing patterns of nutrition and exercise and increasing prevalence of diabetes may have adverse effects, possibly leading to increased prevalence of heart disease, they proposed. Differences among AI/AN from different geographic regions of the US in the four major risk factors (smoking, hypertension, hypercholesterolemia and diabetes) probably explain differences in rates of cardiovascular morbidity and mortality.

The Strong Heart Study is now describing rates of risk factors among Indians of North and South Dakota, Oklahoma and Arizona, as well as their dietary intakes (Welty, 1994, personal communication). A preliminary comparison of risk factors among these groups with rates from Gambell Siberian Yup'iks and the general US population has been made (B. Howard, personal communication, 1994). She reported more obesity and smoking were observed among AI/AN compared with the US in general. American Indians also experienced more diabetes, although study participants at Gambell experienced the least. Hypertension was less common in AI/AN than in the general US population. The cholesterol, LDL, and HDL levels of AI were lower than the US values, but Siberian

Yup'iks had comparable cholesterol and LDL values and considerably higher HDL values than the general US population. Triglyceride values varied with Arizona Indians having the highest, Alaska Natives the lowest, and the other tribes closer to the US general population.

There were similar patterns of cardiovascular disease among Natives and non-Natives in Canada as in the US (Young et al., 1993). Among men, the rates for both Inuit and Indians were approximately 60% of the rate for all Canada. Among women, the rate for Indians was the highest among the three ethnic groups in the NWT and approached parity with the rate for all Canadians. Inuits smoked more than the predominantly non-Native Manitobans to the south. Obesity was prevalent in older Inuit women; hypertension among young Inuit men. Total cholesterol and triglyceride levels among Inuit were lower than or not different from Manitoba residents, except for Inuit women 25-44 year old. Relatively high HDL levels occurred in older Inuits of both sexes. Unlike the Alaska Natives, rates of ischemic heart disease among males and females in NWT appear to have gone down since 1975, similar to the decline among the total Canadian population. This could be due to the population including 50% non-Natives.

Among 704 Canadian Cree and Objiwa Indians evaluated in 1987, past and current rates of ischemic heart disease were low (Young, 1990). Their risk was lower than the risk of Canadians in general, but this was not uniform among all Native groups across Canada. Obesity, hypertension and diabetes, all known risk factors for IHD, were particularly prevalent. Risk factors that had an independent and statistically significant effect on measures of glucose included age, body mass index (BMI), triglycerides, family

history of diabetes, low educational level and high waist:hip ratio. When diabetic status was a dichotomous variable, age, family history, triglycerides and BMI were identified as risk factors. Sex was an important determinant of hypertensive status along with age, BMI, family history, single marital status, unemployment and serum cholesterol. Central (abdominal) obesity was the primary type observed. Physical inactivity was a determinant of BMI or skinfold thicknesses but no specific dietary factors (measured as daily caloric intake, major nutrients, and fiber from a food-frequency recall for 1 month) were identified from multivariate analysis. Young concluded there was probably enough data to support a concerted effort to reduce obesity, hypertension and diabetes, despite gaps in existing data. Available data should be used to plan health-promotion programs.

Nondietary cardiovascular disease risk factors

There are also nondietary risk factors for CHD that must be considered. Linscheer and Vergroesen (1994) concluded available data supported the concept that dietary composition, including fatty acid type, cholesterol concentration, and sodium content, in combination with a genetic predisposition to develop hypercholesterolemia, arterial hypertension, obesity and diabetes mellitus, were the major risk factors for developing atherosclerosis and its complications. The proportions of coronary heart disease attributable to modifiable risk factors included: high cholesterol (43%); inactivity (35%); high blood pressure (25%); smoking (22%); obesity (17%); and diabetes (8%) (Brownson et al., 1993).

Smoking

Smoking is prevalent among Alaska Natives. According to a 1985 survey, 58% and 33% of Alaska Native men and women, respectively, smoked, compared with 32% and 27% of US men and women of all races (Lanier et al., 1990). The relative risks of mortality from smoking-related coronary-heart disease (current smokers vs. never smoked) are 1.97 among men and 1.78 among women (Lanier, et al., 1990).

Genetic Interactions

The role of genetics in relation to CHD cannot be ignored. But CHD is generally associated with environmental influences or life style in the epidemiological literature. Life style determines the levels of risk factors, against the background of genetic variation (Epstein, 1992). Marmot (1992) argued that the occurrence of CHD has much more to do with the social and cultural features of a society than it does with the genetic composition of the people. First, rapid changes that occurred in the rates of CHD are not compatible with the rate of change in gene frequencies. Second, migrants tend to take on the CHD rate of their new country.

Rose (1992) offered another perspective by distinguishing between the causes of disease in individuals and the causes of disease in populations. He differentiated between susceptibility, which cannot be changed, and exposure, which can be changed. An example of susceptibility is sex. Examples of exposure are smoking and diet. All are considered risk factors but are different. Differences between individuals are due more to genetic than to modifiable factors (although the lipid risk factors represent a complex outcome of both genetically determined

susceptibility and modifiable behavior). The reverse is true of risk differences between populations. The large differences in incidence observed between countries are due mainly to modifiable external factors rather than to differences in genetic susceptibility. Thus there is a greater potential for prevention at the population level, according to Rose.

Using somewhat different terminology, Kuller (1992) indicated that the distribution of cholesterol levels is a function of genetics or host susceptibility to the common source (dietary fat and cholesterol) and to other environmental factors, especially caloric balance, weight gain and loss, glucose-insulin metabolism and alcohol consumption. He maintained that the distribution of cholesterol levels in the population is a series of cholesterol distributions for each specific genetic polymorphism such as the apo-E polymorphism. (For example, the mean cholesterol for individuals with Apo E4*3 may be 15-20 mg higher than that for individuals with Apo E3*2.) Scheer et al. (1995), however, showed no statistically significant difference when total-serum cholesterol, HDL and LDL levels from 130 Alaska Natives, who underwent forensic necropsy were analyzed by genotype. They did show significant differences by apo E genotype in the extent of total surface lesions in the right and left coronary arteries. A decrease in lesions was seen for genotypes with the E2 allele and increase in lesions for the genotypes with the E4 allele, relative to the E3 homozygotes. Scheer et al. (1995) estimated that the apo E genotype explained approximately 3-6% of the variance in total-surface lesions in the coronary arteries after adjustment for age and sex.

Another approach to genetic interaction was presented

by Horrobin (1987). He theorized that while lower prevalence of coronary heart disease has been attributed to the high dietary intake of EPA from fish and marine mammals among Greenlandic Eskimos (Dyerberg et al., 1975, Horrobin, 1987), genetics are also operating. He described a potential genetic mechanism to explain biological differences. Even on a Western diet, Greenlandic Eskimos have levels of plasma arachidonic acid (AA) far below those seen in Europeans while levels of di-homogammalinolenic acid (DGLA, an intermediary in the production of AA from linoleic acid) are higher in Eskimos. These low AA and high DGLA levels seem to be due to a genetic abnormality of desaturation of essential fatty acid because they occur even when EPA intakes are low. AA is thought to be important in the pathogenesis of CHD. AA is considered prothrombotic because it generates thromboxane A2. DGLA has potent anti-thrombotic and vasodilator effects and can inhibit proliferation of smooth muscles. DGLA also seems to enhance greatly the conversion of EPA to prostaglandins. Horrobin proposed that the high EPA intake against the background of high DGLA and low AA is what is relevant. The genetically high DGLA and low AA are likely to be as important as dietary EPA in determining disease patterns in Eskimos.

Dietary differences can account for serum levels of EPA and linoleic acid (LA) in Greenlandic Eskimos and Danes, but DGLA and AA are probably genetic in origin, and likely due to the reduced activity of the enzyme delta-5-desaturase (D5D), which converts DGLA to AA and 20:4n-3 to 20:5n-3. The origins of docosahexaenoic acid (DHA, another n-3 fatty acid) also may be different in Greenlandic Eskimos and Danes, although DHA occurs at similar levels. Danes appear to make DHA from dietary α-linolenic acid, whereas Eskimos

may be unable to do so.

Genetic effects operate on two levels, the population level and the individual level. The former may indicate general attributes and susceptibility for a group, but the unique genetic composition of an individual will define probability of developing a condition with a genetic component such as cardiovascular disease. An understanding of both factors is useful in understanding the role of genetics in cardiovascular disease.

The genetic homogeneity of a population should make comparisons and explanations of phenomena, such as cardiovascular disease prevalence, easier. Scott and Wright (1983), however, reported that genetic diversity among 1,252 residents of 25 Central Yup'ik villages was greater than that of Eskimo-Aleut groups who speak different languages. They determined phenotypes of six polymorphic enzymes to reach this conclusion. Although these authors noted diversity within the Central Yup'ik population, comparisons of Central Yup'iks with other populations would probably show even greater diversity.

Shields et al. (1993) also addressed homogeneity among northern populations and reported limited diversity in sequencing of the control region of mitochondrial DNA (mtDNA) among subjects of circumarctic populations. These included Inupiaq Eskimos and Athapascans from Alaska, Chukchi from Siberia, Eskimos from West Greenland and Haida from Canada, referred to as circumarctic populations. Sequence diversity was analyzed in 33 mitochondrial lineages among 90 individuals of circumarctic groups. These authors reported the diversity within and among these Native groups to be considerably less than the sequence diversity observed within and among 145 individuals from three Amerind tribes.

A family tree inferred from differences between mtDNA sequences related populations in the following proximity: Inupiaq and Greenlandic, then Athapascan, then Chukchi, then Haida, then further removed are Bella Coola, Nuu-Chah-Nulth and Yakima, of the Pacific Northwest. They used sequence diversity in the mtDNA control region to define genetic affinities and mitochondrial lineages of populations.

Based on the absence of a genetic deletion among Alaskan Athapascans, Inupiaq Eskimos, Yup'ik Eskimos, Aleuts, Siberian (Yup'ik) Eskimos, and Chukchi, Shields et al. (1992) suggested the possibility of a common ancestry for these groups (e.g., that their common ancestor in Asia may have lacked the deletion). This deletion was Asian-specific 9-bp deletion between the genes for mitochondrial cytochrome oxidase II and lysine transfer RNA. These Alaskan and Siberian groups may also share ancestry with the Haidas of Canada, Inuits of Greenland and Dogrib Indians of NWT, who also lack the deletion marker.

These relatively close genetic backgrounds put cross-population comparisons among different Amerind populations on firmer ground by "reducing the noise" genetic variability could contribute to explaining variations in blood lipids. This lack of mtDNA variability also strengthens the argument for the role of factors other than genetics in the development of cardiovascular disease among northern populations. This interpretation would be consistent with Katz and Stamler's early conclusion that the differences that exist among peoples in incidence and severity of atherosclerosis tend to correlate with culturally conditioned variations in nutrition and diet, rather than with racial, climatic or other factors (Rodahl, 1954a).

Cardiovascular disease discussion and summary

Theoretical causes for lower cardiovascular disease

Several theories have been put forth concerning the causes of lower cardiovascular disease among Alaska Natives. Although genetics may be a contributing factor, each of the recent papers about Alaska Natives has suggested that diet is an important contributor (Welty and Coulehan, 1993; Middaugh, 1990; Davidson et al.,1993).

Feldman et al. (1972) measured concentrations of serum cholesterol of 168 Eskimos at Point Hope, Alaska, described the dietary habits of the community, and determined the cholesterol content of 17 indigenous foods. In spite of a diet high in cholesterol, which produces levels of higher serum cholesterol levels in both Eskimos and US whites, Feldman et al. (1972) attributed the low CHD reported by others (Wilber and Levin, 1950, Rodahl, 1954*a*, Sinclair, 1953, Gottman, 1960, Arthaud, 1970, Bang, et al. 1971) to the following: 1) a mean age of 26 in the Eskimo population studied; 2) < 3% of the Point Hope subjects ≥ 40 years-of-age had serum cholesterol > 250 mg/100 ml, whereas high cholesterol was more common in whites; 3) although there were some atherosclerotic changes, they did not usually produce clinical symptoms; 4) the variable dietary supply during the year may produce lower levels of serum cholesterol levels; and 5) during hypocholesterolemic periods when food was less plentiful the vascular-wall deposits may be slowly removed.

Middaugh (1990) observed that the comparatively low mortality rate for cardiovascular disease was remarkable in light of numerous changes experienced by Alaska Natives. Dietary changes occurred since the 1900's and western food was more available since the 1960s. Smoking and alcohol use

increased in the 1950's and use of snuff increased in the 1970's. These factors could obscure differences, but low rates of mortality from CHD persist. The latency period between individuals developing cardiovascular disease and observing changes in mortality rates, however, should be considered. He acknowledged that causality is not established by evidence of differences in rate of cardiovascular mortality and dietary intake of n-3 fatty acids from marine mammals and fish. Although low rates of cardiovascular disease could be related to genetic factors, other factors such as differences in diagnostic services, inaccuracies in death certificates or misclassification of race, he concluded that additional research was needed on the beneficial effects that may exist from consumption of n-3 fatty acids.

Welty and Coulehan (1993) pointed out the low rates of diabetes among Alaska Natives in general, which is a risk factor for cardiovascular disease. Although rates for diabetes vary among groups of Alaska Natives, the mortality rate for all Alaska Natives is still comparatively low (Schraer et al., 1988, Indian Health Service, 1994).

Parkinson et al. (1994) reported 6.8 and 13 times greater plasma levels of EPA in residents of two Yukon-Kuskokwim River villages where marine fish, salmon and seal oil were consumed, compared with non-Native participants where presumably these foods were not commonly consumed. These authors concluded that future investigations of IHD in this population are warranted because Alaska Natives have been exposed to most advances in curative medicine, consume what is considered by many to be a protective diet rich in fish oils, and have high rates of tobacco use.

Theoretical causes for increasing prevalence of cardiovascular disease

When IHS Areas are rank ordered for total cardiovascular disease (ICD-9 390-448) using rates for 1981-1983, the Alaska Area ranks seventh of eleven (Welty and Coulehan, 1993). Alaska's parity gap compared with the US as a whole is -32.0 (under parity), but the preventable gap[3] is 99.1 (the difference between the US theoretical best of 107.2 and the age-adjusted mortality rate among Alaska Natives from cardiovascular disease of 206.3)(Welty and Coulehan, 1993). Davidson et al.(1993) suggested possible dietary, genetic and behavioral associations with several specific categories of cardiac disease. They concluded that because improvements in diagnosis and treatment have paralleled those elsewhere in the US, increases in cardiovascular disease may be attributable to increased exposure to risk factors, such as diabetes (increasing) and obesity (increasing, Murphy et al., 1995). Nonetheless, both also increased in the US between 1965 and the early 1980's with a concurrent decline in mortality rates from cardiovascular disease.

Scott et al. (1958) described values of serum cholesterol among Eskimo men (17-53 years-of-age) as comparable with those of US men, whereas blood-pressure values were essentially in agreement although slightly higher. Torrey (1979) noted a prevalence of hypertension among Aleuts of St. Paul, Alaska, as high as any reported in the United States in 1966 and 1976.

There is a wide body of research applicable to the

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[3] *The difference between current mortality and US best of 107.2 projected as the rate attainable if all people stopped smoking, if all systolic blood pressures were < or = to 139 mm Hg, and if all serum cholesterol levels were < or = 219 mg / 100 ml.*

multiple issues of cardiovascular disease and diet among Alaska Natives. Based on this varied body of knowledge, I conclude that Alaska Natives have lower cardiovascular disease, specifically ischemic heart disease, rates than non-Natives in Alaska and the US at large. In contrast, their rates are not declining but have been increasing.

Although several exposure factors, such as smoking and inactivity, have been proposed as contributing to increased cardiovascular disease, the unique fatty-acid composition of the diet of Alaska Natives may be contributing to the maintenance of the lower rates. Although the susceptibility risk factors of gender, hypertension, and hypercholesterolemia may influence the likelihood of cardiovascular disease for the individual, other positive or negative dietary factors also may contribute to maintaining the death rates at their current level.

Dietary assessment methods

Assessing the dietary intake of individuals is a fundamental component of evaluating the effect of diet on any physiological outcome, including the development of cardiovascular disease. Increased attention has been directed to improving the methodology of dietary assessment in recent years. There are various methods employed to assess the dietary intakes of individuals, each with advantages and disadvantages. Thompson and Byers (1994) reviewed five types of dietary-assessment methods: dietary records; 24-h dietary recall, food frequency, brief dietary-assessment methods and diet history. The strengths and weaknesses of each are summarized.

Before discussing measures of dietary intake, which are physically noninvasive, it is worth noting that biomarkers

are being used to some extent. In addition to their use as disease predictors (e.g., blood cholesterol and cardiovascular disease), some can be used as surrogate measures of dietary intake. These measures also can be used to validate other forms of dietary assessment. The major problem with physiological measures is the numerous determinants of nutrient concentration in a well-fed population (Thompson and Byers, 1994). Willett (1990) noted that the reproduciblity correlation coefficients of many biological measures range from 0.5 to 0.7, which is comparable to less-invasive techniques of dietary surveys. Another problem with the use of biomarkers, which is important in this investigation, is the lack of a biochemical marker for total dietary fat. Similarly, the use of serum cholesterol to estimate the intake of dietary cholesterol for individuals results in severe misclassification (Willett, 1990). Each nutrient must be evaluated to determine if a biomarker is useful in measuring dietary intake. For an investigation that targets only a few nutrients with effective biochemical markers, the biochemical marker may be used. Nevertheless, in an investigation where many nutrients are of interest, the variation in effectiveness of biomarkers supports the use of other types of dietary assessment methods.

1) The dietary record is often considered the "gold standard" as it can provide quantitatively accurate information on food consumed during the recording period. Recording foods consumed at the time of consumption can reduce omissions, errors and inaccurate estimates of portion size. Nonetheless, this method requires literate subjects willing to take the time to record their intake, usually for 3-7 days. The

process of record keeping can also alter the intakes. Thus, even the gold-standard method has limitations on identifying true intake.

2) An interview using the 24-h dietary recall relies on an interviewer, thus reducing the need for literate respondents. Immediate recall of the previous day increases accuracy. One 24-h recall taking about 20 minutes is a relatively small burden on the interviewee. Weaknesses include inaccurate respondent recall, and dietary variation from day to day. The principal use of this technique is to describe the average dietary intake of a group.

3) The food-frequency method queries respondents about their intake of predetermined foods and is designed to estimate usual intake of foods and can be used to rank individuals according to their usual intake. This method requires less of the respondent, can be self-administered, and processed more rapidly than open-ended instruments. This method is used often in large-scale studies relying on mailed questionnaires. Details of dietary intake, however, may be lost and quantification is less accurate than recalls or records. Longer food lists tend to overestimate intake while shorter lists underestimate intake. Thus, results should be considered approximations of nutrient intakes.

4) Brief dietary assessments can be used when the total diet is not being studied, or when the process of measuring diet intake is used to stimulate interest and facilitate education about nutrition. Their brevity and correspondence with more extensive measures of dietary assessment make them attractive. These measures,

however, are not quantitatively meaningful and estimates of the dietary intake of a population cannot be made. The food behaviors correlated with dietary intake in one study may not correlate in another population.

5) The diet history as defined by Burke (1947) originally referred to collection of frequency of intake of foods plus the typical make-up of meals and a 3-day diet record. Some advocate using the term diet history for the process previously described as the food-frequency method (Hankin and Wilkens, 1994). Others advocate reserving the term diet history for ascertaining the usual intake in detail, plus the frequency and amount of food intake. The strength of this method is the detail provided. By reporting foods consumed by meal, recall may be more accurate. Weaknesses include the extensive resources needed for conducting the diet history interview. This method suffers from the same limitation as frequency data in respect to interpreting estimated intakes in relative rather than absolute terms. If respondents tend to snack rather than eat defined meals, the meal-based approach is less useful.

At this time there is no satisfactory gold standard for measuring dietary intakes among free-living populations. The earlier gold standard, detailed diet histories and multiple 24-h recalls, is considered less precise than it once was with the publication of two studies that independently determined that food intake was massively under-reported (Monsen, 1992). In one study, which compared energy expenditure using doubly labeled water with 2 weeks of intakes, obese and nonobese adolescents underreported

intakes by 20-43% (Bandini et al., 1990). The other study compared weighed and measured food intakes with the caloric intake required to maintain body weight (Mertz et al., 1991). These adults under-reported foods by 18%.

Hankin and Wilkens (1994) recommend that the dietary method for epidemiologic studies, such as cohort or case-control studies, should generally be a diet history (referred to as the quantitative-food frequency instrument by others and in this thesis). Selected food items consumed during a usual month should be determined, although data from the past year generally is recommended for large population studies.

When a food-frequency instrument is used, its effectiveness needs to be evaluated. Willett (1990) suggested the following methods: 1) comparison of means from relatively independent measures of the same intakes; 2) proportion of total intake accounted for by foods included on the questionnaire; 3) reproducibility; 4) validity; 5) comparison with biochemical markers; 6) correlation with a physiologic response; and 7) the ability to predict disease. The approaches are summarized below.

1) Comparison of means, such as with National Health and Nutrition Examination Survey (NHANES) is simple and inexpensive, but gives limited information on validity, and most seriously, provides no information on the ability of the questionnaire to discriminate among persons. Although similar mean values provide some reassurance that the questionnaire is reasonably comprehensive, it is possible to exclude important items and include erroneously large portion sizes and obtain comparable means.

2) Comparing foods for inclusion in a food-

frequency questionnaire with an open-ended method such as a 24-h recall has been used as support for the completeness of the questionnaire. Due to identification of ingredients for nutrient assessment in a 24-h recall, and due to collapsing of foods into general categories in the frequency tabulation or analysis, the ratio of foods listed on the frequency to foods listed on a 24-h recall will be underestimated, resulting in a conservative evaluation of the questionnaire. Although accounting for a low percentage of nutrient intake would raise concern about comprehensiveness of the questionnaire, foods might still be carefully selected to explain maximally the between-person variation in nutrient intake. Accounting for a high percentage of nutrients does not guarantee validity as respondents may misinterpret the questionnaire.

The highest priority in designing a dietary questionnaire is usually to discriminate among persons with respect to their intake, rather than to estimate their absolute intake. Selecting food items for inclusion in a frequency instrument may be facilitated by examining the proportion of between-person variance in nutrient intake that is accounted for by a food. Obtaining recalls or records from representative samples of the population also will facilitate food selection for inclusion in the frequency.

3) Reproducibility at two points in time (longer than a few weeks apart) may be useful as a first approximation of questionnaire performance. Variation due to both questionnaire and true change in diet can occur but the necessity in separating the causes is not

extremely serious when evaluating measurement error, according to Willett (1990). For most studies the levels of correlation on repeated dietary measurements (0.5-0.7) are comparable to many biological measurements among free-living subjects (e.g. serum cholesterol, 0.65, and blood pressure, 0.60 - 0.64).

4) Validity, or the questionnaire's ability to measure actual dietary intake, is assessed most directly by comparing individual estimates of nutrient intake from the frequency with those measured with a more accurate method. The problem of no perfect measure of dietary intake is dealt with by assuring that the errors of both methods be as independent (i.e. uncorrelated) as possible, given the subjects must be the same, to avoid spuriously high estimates of validity.

Among the available and feasible methods of comparison for validating a food-frequency questionnaire, diet records are likely to have the fewest correlated errors. The major sources of error with food-frequency questionnaires are restrictions imposed by a fixed list of foods, memory, perception of portion sizes and interpretation of questions. These sources of error are minimally shared by open-ended records of diet. Because errors in the two methods are largely independent, validity, if anything, tends to be understated. But diet records need to be kept for a sufficient number of days to represent average intake of the nutrients in question and cover the interval of time corresponding to the questionnaire, which is typically 1 year. The process of keeping a diet record may alter intake, however, and reduce correlation.

The use of multiple 24-h recalls is the primary alternative to diet records, but because errors are more likely to be correlated (both instruments rely on memory and perception of serving sizes), this method is probably suboptimal. Where subjects are illiterate or not highly motivated, 24-h recalls may be the only reasonable option. Data on food composition represent a source of error that is likely to remain correlated when comparing two methods.

The validation study Willett conducted with 173 nurses keeping four 1 week records of records, was expensive and took 2 years of full-time efforts by a research dietitian, a programmer and data-entry support. Based on cost, such a study may not be justifiable, but similar correlation coefficients (corrected for within person and between person variance) can be obtained with fewer days (2-5) of records or recalls per subject (Willett, 1990).

For culturally diverse populations, Hankin and Wilkens (1994) recommended validating the diet history (quantitative food frequency) by collecting multiple 24-h recalls or food records from representative samples of each ethnic group and comparing the mean dietary intakes with the values obtained from the diet history. Hankin and Wilkens recommended a quantitative diet history with multiple choices of serving sizes, in contrast to Willett, who used a single serving size. The unannounced multiple 24-h recalls collected over 1 year may be a satisfactory validation method in culturally diverse or less-motivated populations as opposed to records of weighed food. Eight to 12 recalls were used in studies Hankin and Wilkens cited. The

development and validation of dietary methods in epidemiologic studies among culturally diverse populations requires considerably more planning and pretesting than the methods used for a single homogeneous population.

5) Comparing biochemical indicators of dietary intake to assess the validity of a dietary questionnaire has great intuitive appeal as the gold standard, because measurement errors should be essentially uncorrelated. The capacity to demonstrate a correlation between these methods provides documentation of validity. The drawbacks include individual variability in absorption and metabolism of most nutrients, which causes variability in the biomarker unrelated to intake. Also day-to-day variation in dietary intake can cause fluctuation in biochemical markers, as can technical error associated with laboratory measurement. These factors can result in modest correlations even when the dietary measurements are highly accurate and precise. The use of biomarkers is further constrained due to lack of biochemical indicators for nutrients of interest such as total fat, carbohydrate, sucrose, or fiber. Other markers have strong homeostatic regulation and thus their association with intake is weak (e.g. plasma cholesterol, retinol and calcium). Although this makes cross-sectional validation of limited use, because individual metabolic differences may dominate between-person variation, changes in blood levels within subjects may be more informative.

6) Prediction of a physiologic response based on an established relationship between a nutrient intake

and the physiologic response may be used as qualitative evidence of validity. Examples include calcium intake and lower blood pressure, and previous milk intake and bone density. Unfortunately, relatively few such relationships are well established. Therefore, this cannot be a primary approach at this time.

7) The use of a questionnaire to demonstrate established relationships between a dietary factor and disease can be interpreted as qualitative support for questionnaire validity, but at this time this cannot be a primary approach, according to Willett (1990). Again this approach is limited by the small number of relationships between diet and disease that are reasonably well established. Examples could include saturated fat and coronary heart disease, and green and yellow vegetable consumption related to risk of squamous-cell lung cancer.

Considerations in analyzing and displaying dietary data

Evaluating validity requires several considerations (Willett, 1990). In analyzing data from a validation study, variables ultimately controlled for in an epidemiologic study, such as age and sex, should also be controlled for in the analysis of the validation study.

Correlation coefficients provide an attractive option for displaying results and should probably be done on log-transformed data because values are often skewed to higher values. One disadvantage of the correlation coefficient is that it is a function of the true between-person variation in the population as well of the accuracy of the questionnaire, but this also can be an advantage because it is capable of discriminating between subjects. In designing

a validation study, a realistic degree of desirable precision might be arrived at by considering that correlations for validity generally tend to be 0.5 to 0.7.

A simple regression equation is another alternative for displaying data. One other option is presentation of "actual values for surrogate categories." For example, subjects are first grouped into categories such as quartiles on the basis of the food-frequency questionnaire (the surrogate method), then the "true value" of the same subjects is assigned to the categories defined by the surrogate method. This method provides simple and direct presentation of the quantitative relationship between the dietary factor (food or nutrient) and risk of disease. This technique helps to interpret null findings, and it does not require a large number of days of intake per subject, because it involves computing means for subgroups, such as quartiles defined by the questionnaire. Even a single day of diet recording per subject provides unbiased estimates of the actual values for these categories.

Validation data can be presented in several ways. The minimum presentation would include means and standard deviations of the true and surrogate measures, plus their correlations. Regression coefficients with standard errors, and standard deviations of the residual for true exposure not accounted for by the surrogate measure, also may be useful.

Willett (1990) concluded that the interpretation of any study of diet and disease can be substantially enhanced by quantitative information on the validity of the method used to measure dietary intake. Any major study should include a validation component. If use of a method validated in another population is being considered, a validation study

should be repeated if the similarity of populations or circumstances is at all in doubt. In general, diet records provide the best available comparison; biochemical markers are potentially useful but do not exist for many dietary factors. Use of a small number of replicate measures per subject with statistical correction for within-person variation provides an alternative to costly multiple weeks of diet records, making a validation study feasible in most epidemiologic settings.

Willett and Stampfer (1986) proposed, and others concurred (Shekelle et al. 1987; Kushi et al., 1987), that epidemiologic studies of diet and disease should be directed at the effect of nutrient intakes independent of total caloric intake in most instances. They proposed that this is best done by employing nutrient intakes adjusted for caloric intake by regression analysis rather than by simply measuring nutrient densities. Until 1986, the importance of total-energy intake in epidemiologic studies often was overlooked (Willett and Stampfer, 1986). Intakes of most nutrients in free-living populations tend to be positively correlated with total caloric intake. In most situations, it is uncertain whether the absolute amount of a nutrient, the amount in relation to total-caloric intake, or the amount relative to another nutrient is most biologically relevant. Apart from methodological error, inter-individual differences in energy intake can be attributed to body size, physical activity, metabolic efficiency, and net-energy balance. When total caloric intake is associated with disease, the interpretation of individual nutrient intakes is complex. Because specific nutrients such as fat, are correlated with energy, they will also be correlated with disease simply on the basis of their correlation with

energy. For example, Willett and Stampfer (1986) pointed out that in nearly every study of diet and coronary heart disease, subjects who subsequently developed disease had lower total caloric intake on the average than those who remained free of disease. Lower caloric intake resulted in intakes of specific nutrients also tending to be lower.

Diet methodology discussion and summary

By applying appropriate techniques of dietary assessment, the contribution of various dietary factors to the level of blood lipids among Alaska Natives may be determined with greater accuracy. Proposed interventions can then be based on actual practices and on physiologic relationships of blood lipids and diet.

I chose both a 24-h recall and a food-frequency method to evaluate diet intake. The instruments are both acceptable for dietary analysis. The 24-h recall was selected because of its relative simplicity to administer, minimal respondent burden and ability to estimate mean intakes for a group (Pao and Cypel, 1990). This method is appropriately used to describe the average intake of a population (Dwyer, 1988, Pao and Cypel, 1990, Thompson and Byers, 1994). The results can be compared with other studies in which data for 24-h recall were collected. This technique also provides detail on food consumption, which the food frequency instrument cannot capture. In addition, recall for the preceding day may be more accurate than recall for the last year. On the other hand, a quantitative food-frequency (or diet history) can be used to describe "typical" intake of individuals. Using two instruments gave some assessment of the utility of the food frequency, because it had not been validated elsewhere. Assessing typical intake is important in

investigations of chronic disease, and the food frequency method is better at describing foods consumed throughout the year (Hankin and Wilkens, 1994). A food frequency instrument also may be more reliable in comparing ratios of nutrients to energy, subjects within a study, and in ranking them by intake categories (Thompson and Byers, 1994).

In this study I developed a new food-frequency instrument because many foods typically consumed by the people of Gambell are not included in more widely used instruments. Seasonality significantly effects nutrient consumption of Alaska Natives (Nobmann et al., 1992), and a single 24-h recall would not reflect seasonal variation. The importance of validating or calibrating an instrument in the population to be studied has been pointed out by several authors (Buzzard and Sievert, 1994, Thompson, and Byers, 1994, Nelson, 1991). Although one 24-h recall is insufficient to measure usual intake of an individual, it gives some frame of reference for the quality of the food-frequency data. I believe that a rough estimate of the validity of the food frequency could be ascertained from the data on the 24-h recalls.

The instruments provided complementary data. The 24-h recalls yielded more reliable estimates of absolute intakes for the group and these data were used to compare mean intakes with other populations (Chapter 2). Nonetheless, the food-frequency values provided better estimates of the individual's average intake, which was used for comparing with his or her blood lipids (Chapter 3) and for comparison of individuals within the study.

HYPOTHESES TO BE TESTED

In general, types of foods eaten by Siberian Yup'iks are hypothesized to effect biomarkers in their blood. These biomarkers are measures associated with risk of cardiovascular disease. Six specific hypotheses or predictions were generated based on literature about the associations of diet and blood lipids among Siberian Yup'iks. Although cardiovascular disease and diet relationships are of ultimate interest, blood lipids were chosen for comparison because the small population did not lend itself to comparisons with prevalence of cardiovascular disease, assuming it was low.

1) Intake of sea mammals and fish is negatively correlated with total cholesterol, LDL and triglyceride blood levels and positively correlated with HDL levels.

2) Saturated fatty acid intake is positively correlated with total cholesterol, LDL and triglyceride levels and negatively correlated with HDL levels.

3) Monounsaturated fatty acid intake is negatively correlated with total cholesterol, LDL and triglyceride levels.

4) High monounsaturated fatty acid intake is correlated with HDL in women and men.

5) High n-3 fatty acid intake is correlated with high HDL in women and men.

6) Seal oil or fish consumption or n-3 fatty acid intake is negatively correlated with impaired glucose tolerance (IGT) and diabetes. Adler et al. (1994) reported a negative correlation between IGT and diabetes and seal or salmon consumption among Southwest Alaska Natives.

Additional analyses will be conducted to explain the variation in blood lipids attributable to dietary variables.

TABLE 1-1 Literature review of nutrients and their relationship to cardiovascular disease.

Nutrient Association	Reference
Alcohol	
Most epidemiological studies show a U shaped association with total cardiac death rate with lowest incidence with 1-2 oz liquor/day.	Elwood et al., 1992
Alcoholic cardiomyopathy can result from consuming 30-50% energy from alcohol for 10-15 y. The relevance of alcohol-induced changes in serum HDL cholesterol to heart disease is not established.	Feinman and Lieber, 1994
Ascorbic Acid (aa)	
Overall, evidence suggests there is no significant role in the etiology or prevention of heart disease. No relation was seen between aa and blood lipids in some studies. Some experimental and epidemiologic evidence suggests possible roles for aa in the genesis or prevention of heart disease including effects on lipid metabolism, vascular tissue integrity and thrombotic episodes. Inverse associations between aa and heart disease have been related to the effects of aa on the integrity of vascular tissue and changes in fibrinolytic activity. The effects of aa intake have also been related to altered hepatic conversion of cholesterol to bile acids and tissue lipolytic activity.	Jacob, 1994

In the Kuopio Ischemic Heart Disease Risk Factor Survey (Salonen et al.,1988) of 175 men with CHD evidence and 449 controls, there was no difference in plasma ascorbic acid between those with CHD and those without.

Wood and Oliver, 1992

Calcium

Deficient intake is associated with hypertension.

Kotchen and Kotchen,1994

Carbohydrate

High carbohydrate diets decrease HDL cholesterol. Refined carbohydrates may exacerbate hypertriglyceridemia.

Feldman, 1994

High carbohydrate intake is positively correlated with VLDL and chylomicrons.

Linscheer and Vergroesen, 1994

Carbohydrate may have a role in ischemic heart disease. Carbohydrate is correlated with serum triglycerides in Type IV hyperlipidemia which is of etiologic significance for coronary heart disease in some people.

Sucrose

Several epidemiological studies and reviews have failed to find sufficient evidence that sucrose is associated with the development of coronary artery disease.

Macdonald, 1994

Fiber, Total dietary

Both positive and negative effects of dietary fiber on CHD have been reported. The Zutphen Study of 871 men found CHD mortality decreased with increasing total dietary fiber. Five prospective studies related lower fiber intake with increased incidence of CHD. But when controlled for energy

the predictive power of fiber disappeared in 2 studies and was reduced in others. Burr's and Fehily's randomized controlled trial of fiber and CHD showed no evidence of any benefit on any index of disease (1989). Men were told to increase whole meal bread and cereal ≥ than 18 g cereal fiber/day for 2 yrs. In fact subjects given advice to increase cereal fiber had higher mortality than those given no advice, although nonfatal MIs appeared to have been unchanged.

Elwood et al., 1992

High fiber and low CHD associations were not significant when controlled for energy or fat in most epidemiologic studies. Schneeman and Tietyen described evidence for a protective role of dietary fiber based on population studies as inconclusive. They reviewed a large number of clinical and animal studies and concluded that wheat bran and cellulose do not lower plasma cholesterol levels, while pectin, guar gum, oat bran, psyllium husk, beans (legumes), and fruits and vegetables do, specifically LDL-cholesterol levels. They cited a study of oat products where dietary fiber exerted an effect independent of fat (i.e. fat intake was already low). However the effect of fiber alone on plasma cholesterol is small and may not be evident in studies of free living populations with fewer than 40 participants.

Schneeman and Tietyen, 1994

Soluble Fiber

Foods containing soluble, viscous polysaccharides make a specific contribution to lowering plasma cholesterol.

Schneeman and Tietyen, 1994

In numerous small feeding trials, soluble fiber reduced serum cholesterol if eaten in sufficient quantities.

The fiber was in pectin from apples and citrus, guar gum from the cluster bean, widely distributed in fruits and pulses, baked beans and oats.

Elwood et al. (1992) concluded that the potential benefit of soluble fiber is probably worth further study. But it is yet to be tested, needing randomized controlled trials with death or survival outcomes.

Non cellulose pectin

Partially digestible non-cellulose pectin and gums (such as oat bran and some barleys but not most wheat bran varieties) may lower cholesterol. In patients with diabetes mellitus they may reduce cholesterol and hyperglycemia.

Feldman, 1994

Cholesterol

High intakes of cholesterol along with saturated fat are atherogenic and associated with reduced HDL and increased LDL. High cholesterol intake can induce β-VLDL which is important in atherosclerotic buildup. β-VLDL is present in plasma of patients with Type II hyperlipoproteinemia. Increased synthesis of LDL, induced by cholesterol, correlates with CHD occurrence and severity in rhesus monkeys.

Linscheer and Vergroesen, 1994

Cholesterol intake increases LDL synthesis and decreases LDL catabolism via the LDL receptor.

Feldman, 1992

Energy

In nearly every study of diet and coronary heart disease, subjects who subsequently develop disease have lower total caloric intake on the average than those who remain free of disease. This as most likely due to low physical activity related to low caloric intake although

variation in metabolic efficiency is usually impossible to eliminate as an explanation. Decreased physical activity has been associated with coronary heart disease by several investigators.

Willett, 1990

In obese individuals lower energy intake is associated with lower serum lipids up to age 70.

Feldman, 1992

Fat

A high fat intake is atherogenic especially with high cholesterol intake.

Feldman, 1994

Very low fat intake is associated with lower HDL. Keys hypothesized that total fat intake was related to atheroma but Sinclair rejected this, saying that relatively low linoleic and arachidonic and total saturated fat and unnatural fats caused atheroma and thrombosis (cholesterol gets esterified with abnormal or unusual fatty acids which aren't cleared and cause atheroma). Sinclair also proposed that phospholipids containing abnormal or unusually saturated fatty acids increase blood coagulability leading to thrombosis. Three randomized dietary trials of primary CHD prevention have been reported; Los Angeles Veterans Administration, Helsinki Mental Hospital, and The Oslo Study of high risk men. Diets were modified in fat composition or total fat. The Los Angeles and Finnish Mental Hospital Studies offered diets high in vegetable oils and linoleic acid and low saturated fat; the third study was low SFA and slightly increased PUFA. All three led to reduced dietary saturated and increased polyunsaturated fats, increased adipose linoleic

acid and lower coronary incidence in the experimental groups than in the controls but no difference in CHD mortality or total mortality.

Wood and Oliver, 1992

McNamara (1994) maintains a common misconception is any reduction in total fat has beneficial effects by lowering plasma cholesterol. He stated that the available data indicate the key to reducing total and LDL cholesterol levels is to lower saturated fat intake, primarily myristic and palmitic acids; little benefit in lowering LDL cholesterol is achieved by reducing unsaturated fatty acid intake as a means to reduce total fat intake to very low levels.

Monounsaturated fatty acids

There are two views regarding the effects of liquid oils rich in monounsaturated fats on lowering cholesterol. According to Feldman (1994), there are no special benefits of MUFA, nor are there special adverse effects of PUFA or low-fat-high-carbohydrate diets if extremes are avoided (i.e. very low fat/very high carbohydrate, very high absolute PUFA, or extreme reduction in dietary cholesterol). She cited conflicting reports of no effects of MUFA on HDL and triglycerides by Ginsberg et al. (1990) when MUFA was increased to 18% and total fat to 38% over the Step 1 AHA diet (10% MUFA and 30% of energy from fat and 250 mg cholesterol). Plasma total cholesterol and plasma LDL cholesterol were significantly reduced in both groups when compared with the group consuming the average American diet, i.e., 38% of energy as fat, 18% saturated fat and 10% MUFA and 500 mg cholesterol per day.

Ginsberg et al., 1990

Wardlaw and Snook (1990) found no significant changes in serum HDL cholesterol or apolipoprotein A-1 in 20 men consuming 37-43% energy as fat from corn oil, or as high-oleic acid sunflower oil, compared to baseline and a butter based diet. Both oil diets reduced serum total cholesterol (16-21%), LDL cholesterol (21-26%), triglycerides (10-21%) and apolipoprotein B-100 22-29% (all $P < 0.001$). Corn-oil based diet values fell further. Dreon et al. (1990) found LDL cholesterol and LDL total mass and HDL cholesterol and HDL total mass did not change significantly on exchanging fat type between polyunsaturated and monounsaturated fat, among 39 men and women. But HDL type 2 (HDL_2) was 50% higher and HDL type 3 (HDL_3) was 7% lower on the polyunsaturated fat diet compared with the monounsaturated fat intake. (HDL_2 levels have been shown to increase after weight loss. HDL_3 levels are positively correlated with alcohol consumption and both are inversely correlated with coronary heart disease risk.) They concluded there was no advantage with respect to plasma HDL concentrations in using predominantly monounsaturated rather than polyunsaturated fats in subjects who consumed reduced-fat, solid food diets.

Mensink et al. (1989) compared the effects of a monounsaturated fat diet (41% energy from total fat) with a complex carbohydrate diet (22% energy from fat) over a high saturated fat diet (38% energy from fat) in 48 men and women, and observed reduced serum cholesterol on both the MUFA and carbohydrate diets, increased VLDL cholesterol in the carbohydrate diet and decreased in the MUFA (olive oil) rich diet. HDL_2 and LDL levels fell on both diets. HDL_3 fell on the high carbohydrate diet and increased on the olive oil diet ($P < 0.05$) as did apolipoprotein (apo) A-1. The authors concluded that the lipoprotein risk profile for coronary

heart disease was affected more favorably by the olive-oil-rich diet than by the diet high in complex carbohydrates.

Mensink et al. (1989) also reported a marked and highly significant difference in the effect of diet on the ratio of apoA-1 to apoB, with the change in the olive oil group being 26% more favorable than in the carbohydrate group. It has been suggested that this ratio is superior to any other indicator for the risk of atherosclerotic disease. (apoB decreased on the olive oil diet.)

Polyunsaturated fatty acids

Polyunsaturated fats lower serum cholesterol, but only 1/2 as efficiently as saturated fats raise it.

Hegsted et al., 1965

Diets very high in PUFA decrease HDL levels.

Feldman, 1994

The level of PUFA intake has a small but significant effect in lowering HDL among Belgian subjects.

Kesteloot and Joosens, 1992

Keys found no association with polyunsaturated fat intake and CHD even at the 15 year mortality follow-up of the Seven Countries Study. However, Miettinen et al. found a significantly lower level of serum linoleic acid and lower polyunsaturated fatty acids among cases in a matched case control, prospective study of 33 men with myocardial infarction out of 1222 middle-aged men.

Wood and Oliver, 1992

Hornstra concluded that dietary PUFAs have an antithrombotic effect in man.

Linscheer and Vergroesen, 1994

Eicosapentaenoic acid (EPA) & other fatty acids in fish

Lower MI incidence among Eskimos was interpreted to be associated with reduced platelet aggregation, low LDL and VLDL, high physical activity, and high intake of C20:5n-3 and C22:6n-3.

Linscheer and Vergroesen, 1994

This was based on investigations of the blood lipids of 140 Greenlandic Eskimos (> 40 years-of-age), and the composition of 227 food specimens collected during subsequent expeditions conducted by Bang and Dyerberg.

Bang et al., 1980

EPA and the other n-3 fatty acids found in fish and fish oils may be hypocholesterolemic in normal subjects or raise LDL & HDL in hyperlipidemic subjects, and significantly lower triglycerides in both.

Feldman, 1994

20:5 n-3

EPA has particularly active antithrombotic activity both through production of PGI_3 and enhancement of TXA_3 a weak platelet agonist, over the stronger agonist, TXA_2.

Elwood, 1992

Dyerberg et al. (1978) showed that the vessel walls can use EPA to synthesize a potent anti-aggregating agent, probably a Δ^{17}-prostacyclin (PGI_3). A favorable lipid profile and low aggregability of platelets could lead to delayed atherosclerotic process in vessel walls of Eskimos. EPA and possibly linolenic acid may be more appropriate as beneficial agents than polyunsaturated fats in general. Dietary change or supplementation may reduce the development of thrombosis and atherosclerosis.

Cigarette smokers had lower EPA in platelets than non-smokers; but they consumed the same energy, fat,

carbohydrate and protein and twice as much salt, according to Wood and Oliver (1992). They theorize that smokers may reject foods high in PUFAS.

Among 6000 Edinburgh postal workers, platelet EPA was lower among those with angina, but it was not lower among those with acute MI when compared with those without.

Wood and Oliver, 1992

n-6 PUFAs

Polyunsaturated fats of the n-6 series, found in liquid vegetable oils such as corn, safflower and sunflower, decrease LDL cholesterol. They may decrease HDL if used in large amounts.

Feldman, 1994

Linoleic acid (18:2)

Generally, linoleic acid has proven beneficial in studies of abnormal lipoprotein metabolism, arterial thrombi formation, increased blood pressure and abnormal carbohydrate and insulin metabolism.

Linscheer and Vergroesen, 1994

Diets enriched in linoleic acid and reduced in saturated fatty acid significantly lower LDL, VLDL, serum cholesterol and triglycerides in man at both 30 and 40% of energy from fat levels.

Linscheer and Vergroesen, 1994

Epidemiologic, clinical and experimental evidence supported an inverse relationship between linoleic acid and CHD sufficiently strong to recommend that populations with high CHD mortality should supplement with more polyunsaturated oil, principally from cereals and vegetables aiming at a polyunsaturated fatty acid to saturated fatty acid ratio (PS) of 0.8, especially among those who smoke cigarettes. Cigarette smokers consumed less food containing

linoleic acid than non-smokers and have lower PS ratios and lower adipose linoleic acid levels and significantly less dietary intake of linoleic acid when compared with matched controls in a prospective study.

Among Edinburgh postal workers, the highest unadjusted relative risk for angina of 3.2 and for acute myocardial infarction of 3.0 occurred in the lowest quintile of adipose linoleic acid (which would not be made by the body).

Wood and Oliver, 1992

Trans-fatty acids (TFA)

Trans-fatty acids are produced when liquid vegetable oils are heated to form vegetable shortening and margarine. Relatively consistent evidence, based on both metabolic and epidemiologic studies, support an adverse effect of partially hydrogenated vegetable fat on CHD risk.

Mensink & Katan (1992) show a diet with 10% energy from *trans*-fatty acids increased LDL cholesterol and reduced HDL cholesterol compared with a similar diet with oleic acid. While a saturated fat diet increased LDL similarly, it did not reduce HDL, so the increase in the ratio of total cholesterol to HDL due to *trans*-isomers was about twice that seen with saturated fat. Similar adverse effects have been shown in three other metabolic studies at 3 and 6% of energy from *trans*-fatty acids. *Trans*-fatty acids also increase blood levels of Lp(a), another likely risk factor for CHD, according to two metabolic studies. In a large prospective study of US women, Willett et al., in 1993 reported those with the highest intake of *trans*-fatty acids from processed vegetable fats experienced the highest risk of myocardial infarction during the next eight years. Foods contributing most to intake of *trans*-fatty acids, such as

margarine, cookies, and white bread, also were associated with increased risk of CHD. Similar findings were observed in a case control study primarily among men (Ascherio et al., 1994). Margarine contributed the most TFA. A positive association of TFA with degree of atherosclerosis was observed in a cross-sectional study (Siguel and Lerman 1993). Consumption trends of *trans*-fatty acids were directly related to CHD in the US during this century.

Ascherio and Willett, 1995

Saturated Fat

Saturated fats are two times as potent in raising serum cholesterol as PUFAs are at lowering it.

Hegsted et al., 1965

There is a "highly significant relationship" between saturated fat and serum cholesterol according to three separate Belgium studies, and a positive association with all cause and cardiovascular mortality.

Kesteloot and Joossens, 1992

There was a strong correlation(r = 0.84) of SFA with CHD mortality in Key's Seven Countries Study of 11,579 men aged 40-59.

Keys, 1980

Diets high in saturated fat and cholesterol are atherogenic and reduce typical HDL (without apo-E), increase HDL with apo-E[HDLc] and increase LDL and β-VLDL, a cholesterol rich lipoprotein. High β-VLDL, which can be induced by high cholesterol feeding in man, likely plays a role in atherogenesis in addition to the well known atherogenic properties of LDL.

Linscheer and Vergroesen, 1994

ApoE2, a lipoprotein mutation and one of three isoforms now identified, is associated with accumulation of chylomicrons & VLDL remanents in the blood. High saturated fat intake leads to atherogenesis and reduced HDL without apoE. Dietary saturated long chain fatty acids raise plasma cholesterol levels and decrease LDL receptor activity.

Feldman, 1994

Saturated fats, especially palmitic (16:0) and stearic (18:0) are prothrombotic according to Hornstra's studies and review of the literature.

Linscheer and Vergroesen, 1994

McNamara (1994) indicates that only three saturated fats appear to elevate plasma cholesterol: lauric acid (12:0), myristic acid (14:0), and palmitic acid (16:0).

McNamara (1994)

Myristic acid (14:0)

The main saturated fat in butter and other dairy fats most strongly increases LDL.

Ascherio and Willett, 1995

Palmitic acid (16:0)

The main fat in palm oil and in beef fat, modestly increases LDL. Ascherio and Willett, 1995

It is prothrombotic, according to Hornstra.

Linscheer and Vergroessen, 1994

Stearic acid (18:0)

The main fat in chocolate and beef has little effect on LDL.

Ascherio and Willett, 1995

But it is prothrombotic, according to Hornstra.

Linscheer and Vergroesen, 1994

Hoak (1994) concludes there is no evidence to support the concept that eating high stearic acid foods causes a

thrombogenic effect in humans.

Hoak, 1994

Folic acid (See Vitamin B-6, vitamin B-12 and folate)

Iron

Not enough studies, especially prospective ones have been done of body iron stores and CHD risk to reach any firm conclusions about Sullivan's 1981 hypothesis that CHD is positively related to body iron stores. In 1992 Salonen et al. showed among 1,931 Finnish men that those with serum ferritin levels ≥ 200 μg/l were 2.2 times more likely to have a heart attack than were men with lower serum ferritin levels; two other prospective studies did not show a relationship (Sempos and Looker, 1995). None of three prospective studies found a relationship between transferrin saturation and CHD. Of five case control studies of serum ferritin and CHD only one found positive associations among men and women. Another study of serum iron and heart disease found a positive relationship but two autopsy studies did not support the hypothesis. Iron may promote the production of tissue damaging free radicals, thus increasing CHD, but none of the studies have considered the effects of antioxidant status, i.e. vitamin E, β-carotene, and vitamin C.

Sempos and Looker, 1995

Ascherio et al. (1994) found in a prospective study that nonheme iron was not associated with risk of CHD. But heme iron, primarily from red meat was related to an increased risk of myocardial infarction. Absorption of heme iron is not down regulated in subjects with adequate tissue iron, so this may be important if tissue iron levels are important

with regard to CHD.

Ascherio and Willett, 1995

Magnesium

There is suggestive evidence for an association between lower dietary magnesium and higher blood pressures.

Kotchen and Kotchen, 1994

Magnesium plays a part in the synthesis of fatty acids.

Shils, 1994

Magnesium is part of hormone, energy and enzyme systems.

McLaren, 1994

Potassium

In societies with high potassium intakes both mean blood pressure and the prevalence of hypertension tend to be lower than in societies with low potassium intakes.

Kotchen and Kotchen, 1994

Protein

Meat or casein may be hypercholesterolemic.

Feldman, 1994

The correlation between animal protein and arteriosclerotic and degenerative heart disease among men 55-59 years in 22 countries was 0.756. This was similar to the correlation with energy from fat (0.659).

Yerushalmy and Hilleboe, 1957

Gordon et al. (1981) found no relationship, however, between age-adjusted mean intake of protein and CHD in three prospective cohorts of men.

Vegetable protein (e.g. soy) may lower cholesterol.

Feldman, 1994

Selenium

Phospholipid hydroperoxide glutathione peroxidase, which contains selenium, has been isolated from several systems and can reduce fatty acid hydroperoxides that are esterified in phospholipids. Selenium is part of glutathione peroxidase in red blood cells. The function of glutathione peroxidase is unknown but it may metabolize hydrogen peroxide and thus protect against injury.

Selenium deficiency appears to be the underlying condition which predisposes to Keshan disease, an endemic cardiomyopathy, characterized by insufficient heart function, or heart enlargement.

Analyzing selenium status from diet intake is risky as nutrient data bases may be incomplete.

Levander and Burk, 1994

Two Finnish prospective studies found that low selenium levels in blood were associated with increased subsequent risk of CHD. High fish intake may have confounded these studies (Ascherio and Willett, 1995). A US study found an inverse association between blood selenium and degree of atherosclerosis. In Holland selenium levels in toenail clippings were inversely associated with CHD risk, but no relationship was seen with serum selenium levels.

Ascherio and Willett, 1995

Salonen et al. (1991) investigated the effect of interactions between serum copper, selenium and LDL cholesterol concentrations on the progression of carotid atherosclerosis in 126 men. They found that high serum copper (1.76μmol/l), low serum selenium (1.4μmol)and high LDL (4.0 mmol/l) were associated with a progression of atherosclerosis.

Sodium

Approximately 30-50% of hypertensive patients and a smaller percentage of normotensive individuals are sensitive to sodium chloride. It is generally recommended that excessively high intakes be avoided (e.g. AHA recommends no more than 128 mmol [7.5 g]/d.)

Kotchen and Kotchen, 1994

Vitamin A

With one exception all retrospective population studies of antioxidant vitamins and CHD have found no associations. The exceptional study found that the plasma vitamin E/cholesterol ratio made a significant and independent contribution to the risk of angina (Riemersma et al., 1991). In prospective studies, vitamin A did not differ between cases dying from CHD and matched healthy controls in Eastern Finland; Stahelin found no association between serum vitamin A and death from heart disease and stroke among male pharmaceutical employees of Basel; nor was any association in death found in a later report. Epidemiological cross cultural studies of Finnish, Scottish and Italian men showed no consistent relationship of vitamin A with CHD. An inverse relationship was found between plasma vitamin A and CHD in 16 European study populations (r = 0.24). The vitamin A content of liver (the body's major store) did not differ between those dying from CHD and from accidental deaths in Singapore's ethnic groups.

Wood and Oliver, 1992

β-carotene

Carotenoids have been implicated in preventing the oxidation of low density lipoproteins and consequently reducing the formation of atherosclerotic lesions.

Olson, 1994

In a study of 16 European populations no relationship with CHD mortality was found. Plasma β-carotene levels were lowest in Scottish men, but there was no difference between Italian and Finnish men.

Wood and Oliver, 1992

In a case-control study of 110 Edinburgh men with angina and 394 controls, the inverse relation between angina and low plasma carotene disappeared after adjusting for smoking.

Riemersma et al., 1991

Vitamin B-6

The relationship between vitamin B-6 and cholesterol remains controversial lacking definitive studies, although supplemental intakes either decrease or prevent increase in serum cholesterol. In monkeys plasma pyridoxal 5'- phosphate is positively correlated with plasma HDL and negatively correlated with total cholesterol and LDL cholesterol.

Leklem, 1994

Roussow reported no differences in vitamin B-6 between possible and probable CHD cases compared with controls in a retrospective study.

Wood and Oliver, 1992

No causal relationship between lower vitamin B-6 levels and homocysteine levels (elevated levels have been shown to increase vascular disease risk) has been established.

Sigmon, 1995

Vitamin B-6, B-12, and folate

Inadequate intakes of vitamin B-6, folate or vitamin B-12 may cause increased blood levels of homocysteine, which in its severe form results in severe atherosclerosis in children. Elevated blood level of homocysteine was first

recognized in 1969 as an independent risk factor for coronary heart disease when elevated blood homocysteine levels and vascular disease were first related. More than 20 case-control and cross-sectional studies of over 2000 subjects indicate that patients with stroke and other cardiovascular diseases tend to have higher blood levels of homocysteine than subjects without disease.

Stampfer and Malinow, 1995

Whether increases in dietary folate or vitamin B-6 will reduce CHD has not been examined directly.

Ascherio and Willett, 1995

Selhub et al. (1995) showed dietary and plasma folate to be inversely related to carotid artery stenosis in 822 elderly men and women of the Framingham Study. The association was stronger than that of vitamin B-6 while the association with plasma vitamin B-12 was weak. Neither vitamin B-12 or B-6 intake was related to stenosis. Mean homocysteine levels reached a stable low level with folate intakes of approximately 400 μg/day in the elderly Framingham residents, suggesting that perhaps 40% of the population is not consuming enough folate to keep homocysteine levels low. Preliminary recommendations may be made based on secondary prevention. In the meantime, it will be prudent to ensure adequate dietary intake of folate.

Selhub et al., 1995

Vitamin E

Wood and Oliver (1992) describe vitamin E as the principal antioxidant but the evidence is not strong enough to support supplementation. Low dietary intake and low blood and hair levels are seen in regions endemic for Keshan disease, an endemic cardiomyopathy affecting children and

young women.

Linscheer and Vergroesen, 1994

Experimental evidence suggests Vitamin E can block the oxidative modification of LDL, an important step in atherogenesis.

Ascherio and Willett, 1995

The plasma vitamin E/cholesterol ratio was low in populations with high CHD r = 0.62. It is thought that when tissue antioxidants are low, peroxidation is potentiated, which probably favors incorporation of oxidized LDL into arterial walls. Increased tendency to peroxidation would favor thrombosis by allowing SFA to have an unbalanced influence on clotting mechanisms. Platelet adhesiveness also increases when vitamin E is low. The importance of oxidative modification of LDL and increased atherosclerosis, thrombosis and MI damage, may depend to some extent on the fatty acid composition and antioxidant vitamin E content of the diet. The inverse association of vitamin E levels with CHD is strong in cross-cultural comparisons. In one case control study (Riemersma et al., 1991) an inverse association of vitamin E with angina was demonstrated, but three retrospective and four prospective studies found no association between antioxidant vitamins and CHD. Wood et al. found among retrospective population studies of antioxidant vitamins A,C,E and carotene and CHD, that vitamin E was the only one inversely related to angina. This was found after controlling for CHD risks and season in the Edinburgh population case control study of 6000 postal workers. No difference in vitamin E was found in subjects with or without CHD in the Kuopio Risk Factor Study.

Wood and Oliver, 1992

Another mechanism for the role of anti-oxidants, e.g. vitamin E, is summarized by Hansen et al. (1994). The earliest recognized gross lesion in atherogenesis is the fatty streak, characterized by an accumulation of cells loaded with cholesteryl esters (foam cells) just beneath the endothelium. The cholesterol that accumulates in atherosclerotic lesions originates primarily in plasma lipoproteins, including LDL. Goldstein et al., were the first to discover a modified form of LDL that could be taken up readily enough by macrophages to convert the macrophages into cholesteryl ester-filled foam cells (National Research Council, 1989*a*). The modification of LDL depends on a common initiating step, the peroxidation of polyunsaturated fatty acids in the LDL lipids, a process that is inhibited by antioxidants e.g. vitamin E. The cytotoxicity of oxidized

LDL may influence the evolution of fatty streaks into more complex, advanced lesions.

Hansen et al., 1994

TABLE 1-2 Literature review of foods and their relationship to cardiovascular disease.

Food	Association	Reference
Coffee	Kawachi et al. (1994) conducted a meta-analysis of eight case-control studies and 15 cohort studies on the association between coffee drinking and risk of coronary heart disease and concluded that there was very little excess risk of coronary heart disease among habitual coffee drinkers using the cohort study data (relative risk of five c/day vs. none was 1.05). The case-control data (odds ratio was 1.63 comparing 5 c/day with none) but they do not rule out an increased risk of heart disease among a subgroup of people who acutely increase their coffee intake. Further epidemiological studies are needed to assess the risk of drinking boiled coffee (risks may be higher than from filtered coffee) and the risk of drinking decaffeinated coffee. In one randomized trial 3-6 c/day raised the concentrations of low density lipoprotein cholesterol.	Kawachi et al. 1994
	The most likely cholesterol-raising factors in unfiltered coffee are kahweol and cafestol (diterpenes).	Thelle, 1995
Fish, all kinds (Pelagic or ocean fish include herring, mackerel, sardines, pilchards, trout, and salmon)	Demersal or bottom feeding fish have lower levels of eicosapentaenoic acid [EPA]). There is greater interest in fatty fish based on the assumption that it is EPA which influences heart disease. In the Zutphen Study (Kromhaut et	

al., 1985) reduced cardiovascular disease incidence was observed although mostly lean white fish was consumed. CHD mortality was less than 1/2 among men who regularly ate a small amount of fish compared with those who ate none in the study of 852 men. Only 1/3 of the fish consumed was fatty fish, 2/3 was white fish with lower EPA. Its unclear from epidemiological evidence if white fish is protective.

Kromhaut et al., 1985

The Western Electric study of 1931 men found a weak protective effect of fish. Investigators who studied 11,000 Swedish twins found a significant beneficial effect of fish consumption. Those with the highest consumption of fish had 1/3 lower risk of MI than those consuming the lowest amount. The Multiple Risk Factor Intervention Trial found a 1/3 reduction in CHD mortality among the regular fish eaters. Other studies however have found results that are not consistent with protection from fish consumption. There was no evidence of benefit in a very large cohort in Norway of fish eating in a 13 year follow-up study. The Honolulu Heart Program, a cohort of 7272 Hawaiian men of Japanese ancestry, found no evidence of benefit from eating fish.

Elwood et al., 1992

Ascherio et al. (1995) found the risk of death due to coronary disease among 44,895 male health professionals who ate any amount of fish compared with those who ate no fish, was 0.74.

Ascherio et al. (1995)

Fish high in fat

According to Elwood et al. (1992), consumption of fatty fish should be encouraged, but cautiously as only one randomized controlled trial has been reported, by Burr et al. (1989). They evaluated fatty fish consumption in 1000

men who recently suffered an MI. There was a 29% reduction in all cause mortality among those advised to eat fatty fish. Japanese epidemiologic data showed lower CHD when residents in a fishing village consumed 250 g of fish/day compared with inland residents who consumed 90 g/day. Norwegian fish intake fell during the Second World War and treatment for MI rose in Oslo hospitals. The Caerphilly Prospective Study among South Wales men found that 1000 men eating fatty fish one time every 2-3 weeks had 1.04 CHD events/yr vs. 811 men who rarely or never ate fatty fish, experienced a 30% higher incidence of major CHD events.

Elwood et al. 1992

Fish or fish oils

There have been a variety of biochemical and metabolic studies along two investigative lines. The major findings are:

1) The greatest effect of fish and fish oils is in lowering triglycerides. There are inconsistent and smaller effects observed on other lipids.
2) The effect on thrombosis related mechanisms is mainly through reduced platelet aggregation which is likely due to n-3 fatty acids, and EPA in particular, displacing arachidonic acid in platelets and thereby reducing thromboxane synthesis.

Fish oils may reduce blood pressure but the findings are not consistent. They may also cause increased red cell deformability which may lead to reduced whole blood viscosity and possibly reduction of red cell adenosine diphosphate.

In three trials where fish oil was given after angioplasty there has been angiographic evidence of a

reduction in recurrence of stenosis, or narrowing of arteries. In two trials, no benefit of fish oil was detected.

Elwood et al., 1992

Tea, green

Green tea extract significantly lowered plasma triglyceride and cholesterol concentrations in rats. A cross-sectional study of 1,371 Japanese men over 40 years old found those drinking 10 or more cups of green tea/day had significantly decreased serum total cholesterol, triglyceride, low density lipoprotein, and very-low-density lipoprotein levels and increased high density lipoprotein levels, compared with those drinking less. Data on the 2254 women who participated in the study were not reported. The effects of green tea were not clearly observed among the women, whose serum lipid concentrations and hepatological markers remained low because so few of them were drinkers and smokers. This is the only reported study to evaluate the effects of green tea to date.

Imai and Nakachi, 1995

Tea, black

A Dutch study found decreased risk of coronary heart disease with black tea consumption, whereas a Scottish study did not. The Dutch researchers attributed their results to the high content of flavonoids in tea (also found in alcoholic beverages).

Anonymous, 1995

Flavonoids may act as antioxidants.

Thelle, 1995

CHAPTER 2

DIETARY INTAKES AMONG SIBERIAN YUP'IKS OF ALASKA AND IMPLICATIONS FOR CARDIOVASCULAR DISEASE

ABSTRACT

Cardiovascular disease, although lower among Alaska Natives than the general US population, appears to be increasing. I describe foods and 29 nutrients, associated with cardiovascular disease, consumed by 65 Siberian Yup'iks (51% of adults 40-87 y in Gambell, AK). In October, 1992, in-home interviews were used to collect data on 24-h recall and annual-food frequency. Dietary factors that were potentially protective out-numbered those associated with increased risk. Although fat intake was high (44% of energy for men, 42% women), intake of saturated fats (11%) was not significantly different from the general US population (National Health and Nutrition Examination Survey [NHANES] III). Energy from monounsaturated fats was significantly greater than the NHANES III sample (18% vs. 13% men, 19% vs. 12% women), whereas polyunsaturated fatty acids were similar (about 8%). Mean intake of n-3 fatty acids (7.0 g/d) was twice the 3 g/d level associated with favorable physiologic effects (reduced VLDL, blood viscosity, and cardiac arrhythmias, inhibited production of thromboxane, and increased synthesis of prostacyclin with reduced likelihood of thrombosis) and is comparable with intakes of Greenlandic Eskimos (8.6 g). Mean intakes of protein, iron, selenium, vitamin B-12, α-tocopherol, folacin (men), and vitamin C (men) compared favorably with the Recommended Dietary Allowances. Dietary risk factors included a high percent of energy from fat, low intake of fiber (14 g/d men, 9 g/d women), especially among older adults, low intakes of

vitamin B-6 and vitamin C (for women). Native foods, especially sea mammals, were significant sources of protein, n-3 fatty acids, and selenium. Their consumption is encouraged. Other dietary recommendations are proposed.

KEY WORDS: Alaska, Alaska Native, Siberian Yup'ik, diet, cardiovascular disease, survey, nutrients, Native foods

INTRODUCTION

This chapter characterizes the dietary intakes of Siberian Yup'ik adults as they relate to cardiovascular disease. Although some aspects of the diets of Alaska Natives have been described previously (Parkinson et al., 1994, Nobmann et al., 1992, Knapp and Panruk, 1978, Bell and Heller, 1978, Feldman et al., 1972, Heller and Scott, 1967, Mann et al., 1962), none relate dietary factors to cardiovascular disease. Studies of Eskimos in Greenland (Bang et al., 1980) and in Siberia (Nikitin et al., 1991) support a relationship between diet, particularly intake of n-3 fatty acid, and cardiovascular disease.

Several dietary factors are considered protective from cardiovascular disease. These include fish, n-3 fatty acids, polyunsaturated and monounsaturated fatty acids, folic acid, antioxidants, such as vitamin E, β-carotene and selenium (TABLES 1-1, 1-2). In 1980 the theory that diets high in n-3 fatty acids protected people from heart disease was supported by information on Greenlandic Eskimos (Bang et al., 1980). Potentially harmful factors include saturated fats, cholesterol and *trans*-fatty acids (TABLE 1-1).

The literature is not clear about whether cardiovascular disease is increasing in Alaska Natives. Historically, cardiovascular disease in Alaska Natives was

thought to be low (Rabinowitch, 1936, Rodahl, 1954*a*, Maynard et al., 1967), but Gottman (1960) stated it was common. In the 1980's mortality rates due to coronary heart disease among Alaska Natives were described as lower than those for non-Native Alaskans and the general US population (Davidson et al., 1993, Middaugh, 1990, Welty and Coulehan, 1993). Davidson et al. (1993), however, reported that the rate of mortality because of cardiovascular disease had increased over the last 30 years in all regions of Alaska except Southcentral. Mortality from cardiac disease was actually greater among 30-44 year old Native men than among their white counterparts in Alaska because of higher mortality rates of rheumatic heart disease and cardiomyopathy.

This paper investigates: 1) the dietary characteristics, which may influence cardiovascular disease, of a group of Siberian Yup'iks living in Alaska who still consume sea mammals; 2) whether the consumption of n-3 fatty acids seen in an Alaskan population of Siberian Yup'iks is comparable to that of Greenlandic Eskimos; and 3) how intakes of Siberian Yup'iks from Gambell compare with other populations. How these intakes might affect cardiovascular disease risk is also investigated.

My investigation is part of a collaborative project to explore relationships between diet and diabetes, hyperinsulinemia, and cardiovascular disease risk factors, among Siberian Yup'iks living on St. Lawrence Island, Alaska. The project investigators[1] obtained data on diet, physical activity, anthropometric measurements, occurrence of diabetes, cardiovascular disease risk factors, diabetes complications, and insulin levels. As part of the ongoing

[1] *Sven O.E. Ebbesson, Principal Investigator, University of Alaska.*

Alaska-Siberia Medical Research Program's larger study, "Diabetes Risk Factors in Alaskan and Siberian Eskimos", data also were gathered in the population of Siberian Yup'ik Eskimos across the Bering Straits on the Chukotka Peninsula, Russian Federation.

SUBJECTS AND METHODS

Subjects

The community of Gambell on St. Lawrence Island, in northwest Alaska, is composed of linguistically similar Eskimos (94% Siberian Yup'ik). Of the approximately 500 villagers, 127 were ≥ 40 years-of-age and were the focus of the study. One-hundred and seven individuals (84%) agreed to participate. Eighty-nine individuals (70%) completed the dietary-assessment protocol. Sixty-five individuals (51%) completed all assessments (dietary, blood lipid, and physical), and met the age and ethnic criteria for inclusion (Siberian Yup'ik based on subject-reported ethnicity of parents). Results are presented on those 65 individuals. Data were collected from 26 September to 3 October 1992.

Field techniques

Two standard methods of dietary assessment were used to measure intakes. These were a 24-h diet recall and a semiquantitative food-frequency questionnaire (Appendix A-1). The 24-h recall was used to measure mean intakes of the group. The food-frequency instrument included 91 foods to assess the usual diet of individuals during the previous year. The food list for frequency analysis was developed prior to the field visit from the following sources: foods reported as eaten by 351 Alaska Natives on 24-h recalls in all seasons (Nobmann et al., 1992, 1989), recommendations

from several Gambell residents, and food frequencies used in the Navajo Area Indian Health Service (IHS) (personal communication L. White, 1992) and in the Aberdeen Area IHS (personal communication E. Zephier, 1992). The draft frequency instrument was revised with additional foods after preliminary testing with Gambell residents, who were visiting Anchorage. These individuals were not part of the study.

Three individuals conducted the dietary interview. Two were registered dietitians (I was one), and one was a Siberian Yup'ik college student who resided in Gambell and was fluent in both Siberian Yup'ik and English. Interviewers were trained in standardized nutrition interviewing and data-collection techniques, and certified by the Nutrition Coordinating Center (NCC), University of Minnesota, Minneapolis.

Subjects were interviewed at home, and then reminded to fast overnight. The following morning they came to the Community Hall, where they received laboratory and physical evaluations and completed questionnaires on physical activity and medical history.

Each interviewer was assigned subjects based on the proximity of houses and on the need to communicate in Siberian Yup'ik. Respondents were first asked to recall what they ate the previous day. Standard measuring spoons, cups, bowls and glasses and a ruler were used to ascertain amounts. Intake of specific vitamin and mineral supplements was recorded and included in the 24-h recall analysis. During the food-frequency portion of the interview, respondents were shown standardized food models and asked if their usual portion was equal to one, one-half, two or more of the size shown. At the end of each day another

interviewer reviewed each 24-h recall to resolve omissions and errors. Following the field visit I reviewed each 24-h recall and resolved questions with the interviewers or the subjects. Records of the 24-h recalls were sent to the NCC, where a dietitian reviewed them, resolved additional questions, and supervised computer entry and nutrient calculations.

Nutrient analyses

To convert the food intakes to daily-nutrient intakes, an appropriate data base was identified. I used the Minnesota Nutrition Data System (NDS) software from NCC, Version 2.6, Food Database version 8A: Nutrient Database version 23. This system was selected based on its favorable comparison with other systems: it contained a large data base of foods, minimal missing values, extensive data on fatty acids and had been used in major investigations of diet and heart disease (See Nieman, 1992). Missing values were handled in the following manner; if an analytic value was not available for a nutrient in a food, NCC calculated the value based on the nutrient content of similar foods. A missing value was allowed only if: 1) the amount was believed to be negligible; 2) the food was usually eaten in small amounts; 3) it was unknown if the nutrient existed in the food; or 4) there was no way to estimate the value because the food was unlike any other (Schakel et al., 1988). I reviewed substitutions or interpretations made by NCC to ensure they were appropriate, and then these were further revised by NCC. Nutrient calculations for the food frequency data were performed by me using the same version of NDS software.

Nutrient data base expansions

To obtain the most complete calculations for nutrient intake, the food and nutrient data bases at NCC needed data on foods specific to Alaska. Information was provided on density of common portions, definitions of unusual foods, recipes of combination foods (Nobmann, 1992), and nutrient and fatty-acid composition of selected foods (Appendix A-2). Using these data and numerous published sources, NCC expanded their data bases on nutrients and foods for 23 foods consumed by Alaska Natives (Schakel et al., 1993). In 1994, nutrient data on seven additional foods of Alaska Natives listed in the food-frequency instrument were compiled (Nobmann, 1993 and Appendix A-2) and shared with NCC. Appendix A-3 lists energy and fat composition of foods added to the NDS Version 2.6/8A/23.

Statistics

Values from the food-frequency questionnaire were transformed into the number of times a food was eaten, or the frequency per year, using the following formula:
Frequency per year = (times reported per day, week, month or year) X (seasons food was eaten).

Spring, summer, and autumn were considered to have 10 weeks each, and winter 22 weeks, based on the perceptions of Gambell informants during pre-testing of the food frequency. Servings per year were computed as follows:
Servings per year = portion size X frequency per year, where portion size was specified by each respondent as either 1/2, 1, 2, or more of the standard portion size shown (Appendix A-1).

Data on food frequency were entered in a computer using Epi Info, Version 5.01 (Dean et al., 1990), and univariate

analyses of frequency of food consumption were performed. To translate foods consumed by each person according to the food frequency into nutrients, the nutrients in each standard portion size were multiplied by the number of servings of the food/year, divided by 365.2 and summed using Paradox software, Version 4.22. Nutrient data were transferred back to SPSS for Windows, Version 5.02, for further analysis by age and sex.

Nutrients were selected for analysis based on literature that indicated they were promotional or protective in the development of cardiovascular disease (TABLE 1-1). Nutrients were adjusted for energy by comparing nutrient/1000 kcal or by percent of energy. Logarithmic transformations were performed when this improved the normality of the distribution. The Lillefors test, which was based on a modification of the Kolmogorov-Smirnov test, was used to determine efficacy of transformation (SPSS, 1992). Two-way and one-way ANOVA were used to evaluate statistically significant differences at the 5% significance level for age and sex. Individuals 40-59 years-of-age (y) were compared with those ≥ 60 y. Pearson correlation coefficients between nutrient intakes from the 24-h recall and food-frequency questionnaires were computed. Statistical significance was defined as $P < 0.05$ unless otherwise stated.

Ethics

The project was approved by the Norton Sound Health Corporation, the Gambell Indian Reorganization Act (IRA) Board, and the Alaska Area and Indian Health Service Research and Publications Committees. Procedures and protocols were conducted by experienced professional staff

in compliance with Human Subjects Policy Guidelines of the National Institutes of Health, the University of Alaska, and the University of Washington. Prior to the arrival of the investigative team, the principal investigator (Sven O.E. Ebbesson), explained the study and consent forms to the village council and each resident individually and obtained the necessary signatures.

RESULTS

Twenty-nine men and 36 women participated. Their ages ranged from 40-87 y (TABLE 2-1).

Utility of the food frequency questionnaire

Effectiveness of the food-frequency questionnaire in evaluating dietary intake was assessed in four ways. First, I compared foods reported on the 24-h recall with foods on the food-frequency instrument; 74% of foods reported on the 24-h recall also were listed on the frequency in some form. Only a few foods were mentioned on the 24-h recalls that were not included; however, their omission was deemed inconsequential in the results from the food-frequency instrument. Foods reported on the 24-h recall more than six times, but omitted from the frequency instrument, included sugar substitutes (22 times), ascidians, known locally as sea foods (11 times), catsup (seven times), onions (six times), and other foods (less than six times).

When energy intakes from the two instruments were compared, the correlation was $r = 0.46$ (TABLE 2-2). When adjusted for age and sex, the correlation was still significant $r = 0.37$. Correlations of crude nutrients ranged from $r = -0.02$ for β-carotene to 0.52 for total fat and monounsaturated fatty acids.

I also assessed the ability of the food-frequency instrument to categorize participants by comparing the mean intake of fat from the 24-h recall for each quartile defined by food frequency (Willett, 1990; TABLE 2-3). The food-frequency instrument differentiated fat intakes of participants in each quartile when compared with mean intakes for each quartile from the 24-h recalls. The food-frequency instrument was not as discriminating when percent of energy from fat was assessed. Finally, mean intakes reported on the two instruments were compared for men and women separately, using log-transformed, paired data. The food-frequency questionnaire gave significantly higher values for 16 nutrients(43%) for both sexes than the 24-h recall ($P < 0.05$). These nutrients included carbohydrate (g and %), saturated fat, stearic acid, sum of n-6 fatty acids, cholesterol, polyunsaturated fatty acids, sucrose, iron, selenium, β-carotene, folacin, vitamin C, dietary fiber and water-soluble dietary fiber and energy. Results of the food-frequency instrument for men gave significantly higher intakes of monounsaturated fatty acids and cholesterol-saturated fatty acid-index[2] (CSI) than the 24-h recall. Women had significantly higher intakes of vitamin B-6 and α-tocopherol on the food frequency than on the 24-h recalls. Means of nutrients that did not differ significantly included both grams and percent of protein, fat, polyunsaturated fatty acids, percent of saturated and monounsaturated fatty acids, myristic acid, eicosapentaenoic

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[2] *CSI = (1.01 x g saturated fat) + (0.05 x mg cholesterol) A low value is desirable. A 30% fat, 10% saturated fat , 2000 kcal diet has a CSI = 37 / day. A "typical American diet" (40% fat, 14% saturated fat, 2000 kcal) has a CSI= 51 / day (S Connor et al., 1989).*

acid (EPA), sum of n-3 fatty acids, PS ratio[3], sodium, vitamin B-12, and animal protein.

Based on these evaluations, I conclude that the food-frequency instrument could yield useful estimates of usual intake, however, estimates for some nutrients may be too high. Therefore, comparisons of usual intakes by individuals with their blood lipids within the study population, were made using the food-frequency data (Chapter 3). Nonetheless, where absolute intakes are of interest, as in comparison with other studies where 24-h data were collected, I used 24-h recall data. Results in Chapter 2 are based on 24-h recall data unless otherwise stated.

Native foods

Of 997 foods reported in the 24-h recalls (TABLE 2-4, Appendix A-4), 145 (15%) were considered traditional Native foods (agutuk, berries, seal blubber, seal meat, birds, bird eggs, fish, moose, seafoods, walrus, seal oil, reindeer, stonecrop leaves, and whale). Native foods accounted for 54% of 147 times meat, fish and poultry were reported, and 28% of 125 times foods classified as fats were consumed.

Over 50% of the protein, n-3 fatty acids, arachidonic acid (20:4), EPA (20:5), cholesterol, iron, vitamin B-12, and animal protein came from Native foods based on the food-frequency analysis (TABLE 2-5). One quarter of each of the antioxidant nutrients, vitamin C, β-carotene, α-tocopherol, and selenium, came from Native foods. Only 25% of the energy, 25% of the saturated fat, 4% of the carbohydrate, 7% of the folacin, and 7% of the dietary fiber came from Native foods. When analyzed by age and sex, there were no

[3] *Polyunsaturated to saturated fatty acid ratio*

significant differences in percent of servings from Native foods (TABLE 2-6).

Both analyses of food frequency and 24-h recall revealed that traditional ocean foods were commonly consumed. Sea mammals ranked fourth among the most commonly mentioned foods on the 24-h recall (following coffee-tea, sugar, and bread-rolls-crackers, respectively). Walrus (*Odobenus rosmarus*), bearded seal (*Erignathus barbatus*), and bowhead whale (*Balaena mysticetus*) combined were consumed slightly more often than beef and chicken combined. Sea mammals, especially walrus, were dried and consumed about monthly. Eggs of murres and chickens were both consumed. Murre (genus *Uria*) eggs were gathered only in the spring although some people froze them for use year-round.

Fish ranked as the 14th most frequently consumed food on the 24-h recalls. Seal oil was most often consumed on a monthly basis, although seal oil or blubber ranked 12th on the 24-h recall. Seal blubber appeared more often than seal oil. Tunicates or ascidians (*Tunicata Ascidiacea*, Appendix A-2) also were eaten, but typically less than once per week by those who ate them.

Traditional mixed dishes were still consumed but with varying frequency. Berry agutuk (Eskimo ice cream made from local berries, shortening such as Crisco, and sugar) was generally eaten once per month or once per year by a third of the participants. People ate agutuk made with greens less often. "Deer" (reindeer, *Rangifer tarandus*) agutuk was eaten no more than once a month. Wild greens were eaten once a week by only a few people. Green juice soup (Appendix A-3) was a traditional food reported by 37 people, however, most ate it only once or twice a year.

Non-Native foods

Chicken was the most common non-Native meat, eaten with the skin 83% of the time and fried 75% of the time. Banquet Fried Chicken was the most popular brand (41%). The three brands of frozen, prepared fried chicken contained 54-59% of energy from fat and 14-17% total energy from saturated fat. The brands consumed are labeled "fried in vegetable oil" but the type of vegetable oil was not given. In comparison, chicken baked with Shake-n-Bake, no added fat, without skin, which was also consumed, had only 26% of energy from fat and 8% from saturated fat.

Other foods purchased from the stores included frankfurters, bacon or sausage, lunch meat and Spam. Noncarbonated soft drinks were more popular than soda pop. When asked specifically about water intake, more people reported drinking water on a daily basis than either soda pop or noncarbonated soft drinks. Sugar was used daily by many, often in coffee, which was consumed frequently. Pilot bread, a hard cracker, also was consumed daily by many.

Fruits (26 times) and vegetables (67 times) did not appear often on the 24-h recalls. Based on food frequency, vegetables were typically reported as consumed monthly. Potatoes other than french fries, and the food group including: green beans, corn, peas and other vegetables, were eaten weekly or more often. Wild greens most often were reported weekly, whereas tossed salad, lettuce, and canned or frozen greens were most often eaten monthly. Although orange juice was reported as consumed at least daily by nine people, no one reported it on the 24-h recall. Terminology may be a problem as Tang or orange-flavored Kool-aid may be considered orange juice by respondents but, not by the data-entry person. Fresh fruits were most commonly consumed

monthly. Frequently, respondents prefaced their quantitative response with the statement, "I eat them when I can get them."

Margarine, most often used daily, was reported more frequently than butter or traditional fats on both the food frequencies and 24-h recalls. Cereal and white bread were commonly consumed daily. Fry or fried bread was consumed weekly. Ice cream, chips, popcorn, doughnuts and cookies were consumed by some. Cake and pie were less commonly consumed.

Nutrient intakes

Comparison of nutrients consumed by men and women (TABLE 2-7) based on the 24-h recall, showed that men consumed significantly more energy, protein, animal protein, fat, sodium, fiber, vitamin C (all $P < 0.05$), vitamin B-6 and α-tocopherol (both $P < 0.01$) than did women. Nonetheless, there were no significant differences between sexes when the macronutrients were compared as percent of energy.

Based on 24-h recalls, there were significant differences in nutrient intake between younger (40-59 y) and older (60-87 y) adults within each sex (TABLE 2-8). Younger women consumed significantly more energy, total fat, saturated, polyunsaturated and monounsaturated fatty acids, vitamin C, and water-soluble dietary fiber, than did older women. Younger women also had a higher CSI. Older women obtained a significantly higher percentage of their energy from protein than did younger women. Intakes of younger men were not significantly different from those of older men with the exception that younger men consumed more water-soluble dietary fiber.

Analyses of food frequency revealed no significant differences in age groups, although there were significant differences between sexes (TABLE 2-9). Men consumed significantly ($P < 0.01$) more energy, protein, fat, carbohydrate, saturated fats, cholesterol and fatty acid 20:4 (arachidonic acid) than women (TABLE 2-9). Men also consumed significantly ($P < 0.05$) more monounsaturated and polyunsaturated fatty acids, total n-3 fatty acids and selenium than did women. The most significant difference in food-frequency data was that men consumed more energy than did women ($P = 0.003$). When the percent of energy from macronutrients and the nutrient levels/1000 kJ were evaluated, however, there were no significant differences. Men had significantly lower PS ratios than women (0.71 for men, 0.78 for women) from food frequency; but the ratios from the 24-h recalls, 0.96 and 0.84, respectively, did not differ significantly. With or without energy adjustments, there were no significant differences between the sexes in their intakes of vitamins A, C, percent of energy from EPA or total n-6 fatty acids. Women, however, consumed significantly more total α-tocopherol when adjusted for energy than did men.

Significant interaction effects were observed for percent of energy from fat, and saturated fatty acids using the food frequency with two-way ANOVA for age and sex. These were not observed when data from the 24-h recall were used. For men, the log-transformed values, for both percent of energy from fat and saturated fat by age, were positively correlated with age ($r = 0.393$, $P = 0.035$, and $r = 0.552$, $P = 0.002$, FIGURES 2-1, 2-3). For women, the correlations were not significant ($r = -0.020$, $P = 0.915$, and $r = -0.083$, $P = 0.635$, FIGURES 2-2, 2-4 respectively).

To test the null hypothesis that energy, nutrient, and fiber intakes of adult Siberian Yup'iks did not differ from the US population as a whole, intakes from the 24-h recall were compared with age-adjusted intakes of the NHANES III participants (McDowell et al., 1994; Alaimo et al., 1994). Siberian Yup'ik men and women consumed significantly ($P < 0.001$) less carbohydrate (g and percent of energy), fiber ($P < 0.01$ for men, $P < 0.001$ for women), and folacin ($P < 0.05$ for men, $P < 0.001$ for women) than did the national sample (TABLE 2-10). Yup'iks consumed more monounsaturated fat (g, $P < 0.05$) and percent of energy from protein ($P < 0.01$), fat ($P < 0.001$) and monounsaturated fat ($P < 0.001$) than did the national sample. Also, they consumed more vitamin A, β-carotene ($P < 0.01$ for men, $P < 0.05$ for women), and iron ($P < 0.01$). Siberian Yup'ik men consumed less ($P < 0.05$) saturated fat than did NHANES III men. Siberian Yup'ik women consumed more ($P < 0.05$) protein than did NHANES III women, but they consumed less ($P < 0.001$) vitamin B-6, vitamin C and potassium. Although these comparisons were significantly different, the null hypothesis could not be rejected for energy, fat, polyunsaturated fat, cholesterol, percent of saturated or polyunsaturated fat, vitamin B-12, or α-tocopherol among both sexes; for protein, vitamin B-6, vitamin C or potassium among men; and for saturated fat among women.

The major sources of selected nutrients among Siberian Yup'iks were compiled based on the food-frequency responses ($n = 64$). Monounsaturated fats came from muktuk (542 g/d for 64 people), margarine (222 g/d), chicken (222 g/d), fried bread (217 g/d), and pilot bread (201 g/d). Saturated fats came from butter (180 g/d), beef (164 g/d), fried bread (153 g/d), chicken (127 g/d) and muktuk (127 g/d). Total α-

tocopherol equivalents came from fried bread (118 mg/d), pilot bread (103 mg/d), berry agutuk (78 mg/d), margarine (71 mg/d), and chicken (49 mg/d). N-3 fatty acids came from muktuk (60 g/d), dried fish (22 mg/d) fresh walrus (16 mg/d) pilot bread (13 g/d) and seal oil (11 g/d).

Intakes of fatty acids of Greenlandic Eskimos and Siberian Yup'iks were compared, although different methods were used to estimate their intakes. The intakes of 50 Greenlandic Eskimos were analyzed from daily food samples collected by the double-portion technique and homogenized (Bang et al., 1980). Because data on the variance of intakes of Greenlandic Eskimo were not available, I could not formally test the null hypothesis that intakes of fatty acids of Siberian Yup'iks were similar to Greenlandic Eskimos. The proportions of saturated and polyunsaturated fatty acids were similar. The proportion of monounsaturated fatty acid, however, was lower for Siberian Yup'iks than for Greenlandic Eskimos (S:M:P::1:1.6:0.7 vs. S:M:P::1:2.5:0.8). The ratio of n-3 fatty acids to n-6 fatty acids was also lower for Siberian Yup'iks than for Greenlandic Eskimos (1:1.5 for Siberian Yup'iks vs. 1:0.4).

DISCUSSION

This study showed that the diet of Siberian Yup'iks in Gambell was characterized by a reliance on traditional foods, primarily sea mammals, despite that the variety of foods in stores has increased since the previously reported studies (Hughes and Hughes, 1960). Continued reliance on traditional foods could be due to both the availability of traditional foods and to the expense of shipping foods to the remote village. In addition, commercial foods are not always available. The diet of Gambell residents is different

from that of non-Native US residents. This is because long-standing practices define the traditional food supply as one derived from the sea that surrounds the island-based community. The collective diet of the participants has elements conducive to good cardiovascular health (lower saturated fat [among men] than NHANES III participants, higher monounsaturated fat, high n-3 fatty acids, adequate intakes of the antioxidants selenium, β-carotene and α-tocopherol), as well as elements associated with increased risk of cardiovascular disease (high proportion of energy from fat, low fiber, plus saturated fat and dietary cholesterol above some recommended levels).

Nutrient intakes conducive to cardiovascular health

Siberian Yup'ik men had intakes of several nutrients described in the literature as conducive to good cardiovascular health (TABLE 2-12). Mean intake of the potent hypercholesterolemic agent (Feldman, 1994), saturated fatty acids, was significantly lower among Siberian Yup'ik men than in the general US male population. Monounsaturated fatty acids, shown by some to reduce total cholesterol and LDL (Feldman, 1994) and increase HDL_3 (Mensink et al., 1989), were higher for both sexes. These conclusions are based on 24-h recall data, which is preferable to the food frequency-data to be consistent with the NHANES III methodology. Higher mean energy and saturated fat intakes, however, occurred with the food frequencies than with the 24-h recalls for both men and women.

Intakes by Siberian Yup'iks provide other advantages for good cardiovascular health (i.e., cholesterol intakes not significantly different than the NHANES III sample)

which was surprising considering the high intakes of meat of Siberian Yup'iks might result in higher intakes of cholesterol. Siberian Yup'iks also have a high level of n-3 fatty acids, and a more favorable PS ratio. Both mean and median intakes of cholesterol for men and women using data from the 24-h recall, were less than the American Heart Association's recommendation of no more than 300 mg/d (1993, TABLE 2-7), although intakes were higher than the National Cholesterol Education Program's recommendation (126 mg/1000 kcal among men, 142 mg/1000 kcal among women vs. <100/1000 kcal, 1994).

N-3 fatty acids have been shown to reduce levels of very-low-density lipoprotein cholesterol; inhibit thromboxane production and increase prostacyclin synthesis, resulting in reduced likelihood of thrombosis, reduced blood viscosity, and reduced risk of cardiac arrhythmias (Ascherio et al., 1995). Because Greenlandic Eskimos are known for their high intake of n-3 fatty acids (Bang et al., 1980), I compared the intake of Siberian Yup'iks from the 24-h recall (TABLE 2-7) with Greenlandic Eskimos. Siberian Yup'ik men consumed comparable amounts but women consumed less n-3 fatty acids than the Greenlandic Eskimos of the 1970's (8.6 g/d among Siberian Yup'ik men, 5.7 g/d, women, or 7.0 g/d ± 10.7 SD both sexes combined vs. an estimated 8.6 g/d for Greenlandic Eskimos of both sexes combined[4]). Yup'ik men and women consumed 11.5 and 9.2 g/d n-6 fatty acids, respectively, or 10.2 g/d ± 6.8 SD combined, compared with

[4] *To compare intakes, grams of n-3 fatty acids in Greenland Eskimo foods were estimated from data published by Bang et al. (1980), by summing the average percentage of total fatty acids for 18:3, 20:5, 22:5, and 22:6 = 13.7% x 69.9 g (the average daily fat consumption) x 0.9 (a best estimate factor to convert fatty acids from percent of total fatty acids to percent of total food, based on a conversion factor of 0.875 for separable lean beef) = 8.6 g.*

3.4 g/d for Greenlandic Eskimos[5]. Thus, it appears that Siberian Yup'iks are still consuming a diet rich in n-3 fatty acids. Although this level is somewhat lower than that of Greenlandic Eskimos of 20 years ago, it is within a comparable range. Greater n-6 fatty acid intake among Siberian Yup'iks than among Greenlandic Eskimos indicates consumption of more plant-based oils. Although these oils do not contain n-3 fatty acids, they also are preferable to saturated fat.

A comparison of Yup'ik intakes for other nutrients with those of other groups (TABLE 2-11), indicates higher dietary risks for cardiovascular disease among Yup'iks than among Greenlandic Eskimos in intakes of total fat, saturated and monounsaturated fats, but lower than that of Danes (Bang et al., 1980). Although closer to Greenlanders than to Danes, mean intakes of Siberian Yup'iks were greater than the mean intakes of Greenlanders for energy (1541 kcal), fat (69.9 g), and percent of energy from fat (39%). Percent of energy from carbohydrate was comparable in the two Eskimo groups (36% and 38%) and below that of the Danish diet (47%). Percent of energy from protein (23%) was higher for Greenlandic Eskimos than for Siberian Yup'iks. The PS ratio was highest among Siberian Yup'ik men (0.96) and women (0.84) using the 24-hr recall (0.71 and 0.78, respectively, using food frequency). The PS ratio of the Greenlandic Eskimo diet was 0.84 and Danish diets was 0.24 according to Bang et al. (1980). Comparing percent of energy from fats in

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[5] *For comparison, grams of n-6 fatty acids were estimated from data published by Bang et al. (1980) by summing the average percent fatty acids from total fatty acids for 18:2 and 20:4 = 5.4% x 69.9 g (average fat intake / d) x 0.9 (a best estimate factor to convert fatty acids from percent of total fatty acids to percent of total food, based on a conversion factor of 0.875 for separable lean beef) = 3.4 g.*

each group revealed that Siberian Yup'iks consumed more saturated fat and less monounsaturated fat than did Greenlandic Eskimos of the 1970's. Siberian Yup'iks consumed less saturated fat and more monounsaturated fat than did Danes, and also more monounsaturated fat than did the NHANES III sample studied in 1988-91.

In addressing the hypothesis proposed by Bang et al.(1980) that diets high in n-3 fatty acids protect people from heart disease, I asked if this theory held in this population of Eskimos. As early as 1927 Greenlandic Eskimos were reported to exhibit little coronary heart disease (Elwood, 1992). Rates of mortality from ischemic heart disease among Alaska Natives, from 1979 through 1988, were lower than Alaskan and US whites (151.8, 248.4 and 253.5/100,000 per y, respectively). Although mortality rates due to ischemic heart disease in the Norton Sound Region where Gambell is located are higher (219/100,000) than other Alaskan regions with Alaska Native populations, they are still lower than rates for whites (Davidson, 1993). Rates of mortality due to ischemic heart disease for this village, however, are not known and may vary from those of the region or from other Alaska Natives.

Whereas Siberian Yup'iks may be consuming a protective diet, more recent reports on Greenlandic Eskimos indicate different results (de Knijff et al.,1992, Hart Hansen, 1990). Based on a 1989 survey among the Nanortalik Inuit, de Knijff proposed that more westernized lifestyles including altered diets, and smoking and alcohol consumption were contributing to plasma lipids characteristic of non-Natives among Greenlandic Eskimos. They reported markedly lower EPA (3.2% vs. 15.8% of total fatty acids) and higher linoleic acid (49.5% vs. 20.4%) levels in the plasma than Bang et al.

(1971) observed among the Uummannaq Inuit of Greenland. Although dietary intakes were not measured objectively, only 13% of the 133 Nanortalik Inuit surveyed claimed to consume an exclusively Inuit diet. By 1990 the same degree of atherosclerosis was noted in Native Greenlanders as in healthy Danish subjects who served as controls (Hart Hansen, 1990). Yet the rate of IHD is lower than in most western societies, leading de Knijff et al.(1992) to suggest that changes toward a potentially atherogenic lipoprotein profile among Greenlandic Inuit started rather recently and that it is too early to observe the consequence, as measured by increased IHD mortality.

Both the mean intakes of n-3 fatty acids of Siberian Yup'iks (8.6 g/d for men, 5.7 g/d for women) and the median intakes (3.2 g/d for men, 2.5 g/d for women) are high. These levels exceed the amount considered therapeutic in clinical trials of fish oils (i.e. 3 g/d, Ascherio et al., 1995). Median intakes are usually lower than the means in studies of nutrient intakes because of skewed patterns of consumption. In contrast, the 1986 investigation of 44,895 male health professionals in the US reported a median n-3 fatty acid intake of only 0.58 g/d for men in the top fifth of the group (Ascherio et al., 1995). Although n-3 fatty acids are considered protective against heart disease, Ascherio et al. concluded that increasing fish intake from one to two servings per week to five to six servings did not substantially reduce the risk of coronary heart disease. Nonetheless, even six servings per week may be too low, as protective cardiovascular effects occur at doses of 3 g or more of n-3 fatty acids per day, amounts several times higher than the median 0.58 g/d consumed by men in their highest category of intake. In contrast, the median intakes

of n-3 fatty acids among Siberian Yup'ik men exceed the minimal therapeutic value of 3 g/d and are almost as high among women.

Antioxidants

Antioxidants constitute another protective factor which may influence cardiovascular disease. The oxidation hypothesis states that the oxidative modification of LDL (or other lipoproteins) is important and possibly obligatory in the pathogenesis of the atherosclerotic lesion (Witztum, 1994). These results, however, conflicted with investigations into specific antioxidants, such as vitamin A and vitamin C, and their protective effects (Wood, 1992). Antioxidants measured in this study included vitamin E (total α-tocopherol), β-carotene, vitamin C and selenium. I noted high intakes of selenium, which occurs in fish and whale. Selenium may inactivate hydrogen peroxide and protect against injury (Levander, 1994). Mean intakes were greater than the Recommended Dietary Allowances (RDA; National Research Council, 1989*b*; 129 μg vs. 70 μg RDA for men, and 95 μg vs. 55 μg RDA for women). B-carotene intakes were significantly greater than NHANES III participants (TABLE 2-10) and sufficient to prevent vitamin A deficiency symptoms without even considering other forms of vitamin A (Mahan and Arlin, 1992). Mean intakes of vitamin C exceeded the RDA (60 mg/d) for men but not for women. Mean intakes of vitamin E, which were inversely associated with CHD (Wood, 1992), exceeded the RDAs (10 mg α-tocopherol equivalents for men, 8 mg for women). Given the conflicting results of other published studies, I propose that more complete investigations are needed to evaluate the effects of antioxidants on cardiovascular disease and to ascertain

levels of risk associated with high intakes including taking supplemental antioxidants. Using current standards, antioxidant intakes of Siberian Yup'iks are generally good.

Folacin intakes, which have been inversely associated with heart attacks and carotid-artery stenosis (Selhub, 1995), were equal to levels associated with low risk of heart attack among one third of the participants. Recent attention has focused on low intake of folacin (<400 μg/d) associated with elevated concentrations of homocysteine and risk of heart attack (Stampfer, 1995) and carotid-artery stenosis (Selhub, 1995). Thirty-three percent of my sample consumed > 400 μg/day folacin according to the food-frequency questionnaires. Nevertheless, 36% consumed < 230 μg/day, which has been associated with increased carotid-artery stenosis. The mean intakes (566 ± 576 SD for men and 381 ± 269 SD μg/d for women from food frequency) for Siberian Yup'iks were comparable with the elderly subjects from the Framingham Heart Study reported by Selhub et al. (1995)(363 ± 195 SD for men, and 395 ± 246 SD for women). Based on the 24-h recall, mean values were lower, 231 μg for men, 152 μg for women, which exceeded the recommended dietary allowances for men (200 μg), but not for women (180 μg, National Research Council, 1989*b*). Because one 24-h recall may not accurately reflect folacin intake when foods like vegetables and fruits are not eaten commonly, the food-frequency values may be preferable. The correlation between the two instruments for estimating folacin intake was poor, indicating inaccurate estimation with 24-h recall data or food frequency. Missing values for folacin in the data base also contributed to underestimated intakes. Evaluating homocysteine levels would be a useful adjunct in further investigations.

Nutrients associated with risk

The intakes of several dietary components were not conducive to lowering the risk of heart disease and may have implications for the cardiovascular health of Siberian Yup'iks. Potential negative influences include low intakes of total fiber and vitamin C and high intakes of total fat. Mean saturated fat and cholesterol intakes were higher than the NCEP (National Cholesterol Education Program, 1994) recommendations (11% vs. <10% of energy from saturated fats and 126 mg/1000 kcal among men, 142 mg/1000 kcal among women compared with the recommendation of <100 mg cholesterol/1000 kcal).

Fiber intakes of 9-13 g (from 24-h recall) are lower than the 20-35 g/d recommended for adults (Pilch, 1987), and the 50 g/d intake reported to significantly lower plasma cholesterol (Anderson, 1988). The low intakes of vegetables and fruits (TABLE 2-4) reflect a traditional diet low in fiber. Intakes of fruits and vegetables were less than the recommended (National Research Council, 1989*a*) five servings a day and, thus, intakes of vitamin C were low. Levels of vitamin C may be underestimated slightly due to coding interpretations of fruit-flavored drinks, which vary in content of vitamin C. I do not place as much emphasis on these dietary-promoting effects, i.e., low intakes of fiber and vitamin C, as I do on the protective components, because evidence for the role of fiber and vitamin C in the development of cardiovascular disease is not clear (Chapter 1, Wood and Oliver, 1992, Elwood, 1992).

Avoiding high intake of total fat is recommended (National Research Council, 1989*a*). Although mean intake of fat (in grams) was greater for Siberian Yup'iks than NHANES III participants, the difference was not significant (*P* >

0.05 using 24-h recall data, TABLE 2-10). Nonetheless, fat intake reported on the frequency analysis was significantly greater for Siberian Yup'iks than NHANES III participants ($P < 0.05$ for men, $P < 0.01$ for women). Compared with all other groups but Danes, the proportion of energy from fat was higher for Siberian Yup'iks (TABLE 2-11). Compared with the National Cholesterol Education Program guideline to eat < 30% of total energy from fat, men consumed 44% and women 42% of their energy from fat.

Conclusions on the importance of total intake of fat by Siberian Yup'iks could be reached with information on prevalence of heart disease morbidity or mortality; however, obtaining such rates may not be possible given the small size of the population. Therefore, mortality rates and disease relationships from elsewhere can be considered. The rationale for current national recommendations to reduce total fat, appears to be intended to reduce saturated fat and control weight more than to limit total fat (American Heart Association, 1993). Fat intakes from the 24-h recalls of 45% of energy among Siberian Yup'ik men and 42% among women are higher than the recommended 30% of energy, and higher than the national sample (34%). Food-frequency intakes, while lower (37% for men and 38% for women) also exceed the recommended and national intakes. If the cardiovascular health of Siberian Yup'iks is good, our findings support the idea that total intake of fat may be less important to cardiovascular risk than current recommendations would indicate, because the proportion of fat in the diet of Siberian Yup'iks was probably even higher in the past than today. Alternatively, if the cardiovascular health of Siberian Yup'iks is not good, our findings support the concept that high intake of total fat may be

contributing to increasing rates of cardiovascular disease. Changes in many other risk factors such as use of tobacco also would need to be considered, however.

In contrast to intakes of total fat, the proportions of saturated and polyunsaturated fat, based on the 24-h recall, among Siberian Yup'iks are closer than NHANES III and other populations to the national recommendations for heart health[6] (TABLE 2-11). Evidence is stronger for a positive association with cardiovascular mortality for saturated fat than for total fat (TABLE 1-1).

Although specific data on the incidence and mortality due to cardiovascular disease for Gambell would be desirable, they are not available. Rates in the larger region, the Norton Sound Health Care Unit of which Gambell is a part, may give some indication, but variations in genetics and diets within the region, the small population, and the rates being available for only 1979-1988, generalizations should be undertaken with caution. The region includes Inupiaq, Siberian and Central Yup'iks that are each distinct populations (Shields et al., 1993). Nevertheless, the age-adjusted mortality rate for all diseases of the heart among Alaska Natives, both sexes combined, in this region was higher than among their white counterparts (376/100,000 vs. 346 US white, 358 Alaskan white, Davidson et al., 1993). Of greater interest in investigating diet and cardiovascular disease, are the

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[6] *The National Cholesterol Education Program (NCEP) recommends the following cholesterol lowering diet: achieve desirable weight; fat ≤ 30% energy; saturated fat <10% energy; PUFA (including about 2% n-3 fatty acids) up to 10%; remainder monounsaturated fats; < 100 mg cholesterol / 1000 calories (Feldman, 1994). The American Heart Association (1993) recommends < 30% of calories be consumed as total fat; < 10% from saturated fat,; up to 10% from polyunsaturated fats, cholesterol < 300 mg / d; no more than 7.5 g / d sodium chloride (3 g / d sodium) and no more that 2 drinks or 1 oz ethanol / d; achieve and maintain desirable weight.*

specific rates of ischemic heart disease that are generally lower for Alaska Natives than for Alaskan Whites and US Whites (152/100,000 vs. 248 and 254, respectively). The rate for both sexes combined for Norton Sound (219) was the highest of the eight regions compared by Davidson et al.(1993). The rate for IHD for Norton Sound men (273) was the highest among regions; women (116) ranked fourth highest. Neither, however, exceeded the corresponding Alaskan white rates (284 men, 192 women) or US white rates (281 men, 228 women).

If I predicted the mortality rate due to coronary heart disease based on saturated and polyunsaturated fat consumption using Hegsted and Ausman's (1988) equation[7] for men from 18 countries, a mortality rate of 369/100,000 would be expected for Siberian Yup'ik men, using the data from 24-h recall. This is significantly lower than the predicted 433/100,000 for US men based on NHANES III data ($P < 0.001$). Again, lacking data for Gambell, I compared the mortality rate for Norton Sound Region for all diseases of the heart for men (Davidson et al.,1993), and observed actual values were comparable to those predicted (402 actual vs. 369/100,000 Hegsted's equation, chi-square $P > 0.05$). If the rate for Gambell men is similar to that for Norton Sound men, the equation predicting a similar rate may indicate that comparatively low saturated fat and higher polyunsaturated fat intakes plays a significant role in decreased mortality from cardiovascular disease among Siberian Yup'iks. Although Hegsted and Ausman improved the correlation of the CHD prediction from $r = 0.787$ to 0.918 by

[7] *Coronary heart disease = 43.9S - 26.1P + 111.5 $r = 0.787$ where S = percent of saturated fat intake, P = percent of polyunsaturated fat intake (Hegsted and Ausman, 1988).*

including alcohol consumption, I did not include this variable because it was consumed infrequently. Alcohol cannot be legally sold in Gambell. Beer was the alcoholic beverage reported most often, which was consumed two times per month, by one person. This equation does not address the effects of other risk factors associated with cardiovascular disease including tobacco and blood pressure.

Concern has been expressed that *trans*-fatty acids, formed in the partial hydrogenation of vegetable oils, may increase the risk of coronary disease (Ascherio et al., 1994). Ascherio et al. reported a positive, significant association between margarine intake of 2.5 pats per day compared with less than one pat per day and myocardial infarction, which could not be explained by other risk factors for coronary heart disease. Margarine was mentioned 34 times on the 24-h recalls by our subjects. According to the food frequency, mean intake was < 2 teaspoons per day and almost one-half of the subjects ate none or < 1 teaspoon per day. Evaluation of other sources of *trans*-fatty acids, such as shortening (e.g. Crisco), and their relationship to levels of LDL and HDL may be warranted (Chapter 3).

Measurement error probably influenced carbohydrate intake in the food-frequency analysis. According to the food-frequency analysis, intake estimates were higher than NHANES III estimates. Whereas the 24-h recall data showed a significantly lower carbohydrate intake among Siberian Yup'ik men and women than NHANES III estimates. The latter may reflect a large number of high carbohydrate foods listed but not necessarily consumed in abundance, on the food frequency. This constitutes a potential weakness of the food-frequency instrument. Of the total carbohydrates consumed, many were simple sugars and few were complex

carbohydrates (Appendix A-4). Based on the comparative ranking of foods consumed by other Alaska Natives in 1987-1988, and participants in the previous national study (NHANES II, 1976-1980), Alaska Natives used table sugar more frequently than the general US population (Nobmann, 1992). While a lower carbohydrate intake may be appropriate, complex carbohydrates with high fiber would presumably be better for good cardiovascular health (National Research Council, 1989*a*), which includes low plasma cholesterol levels (McNamara, 1993).

Foods

While nutrients and their ratios are of interest, people choose and consume foods, which may have other attributes not completely defined in terms of known nutrients. Foods also may be markers for other behaviors important to cardiovascular health. Use of traditional versus nontraditional food is an example. The physical process of obtaining and preserving traditional foods may have health benefits that are independent of consuming those foods. For example, high levels of physical activity associated with hunting and harvesting sea mammals may prevent obesity and may alter endorphin levels related to stress, another possible risk factor for cardiovascular disease. Marmot (1992) alludes to lower prevalence of CHD independent of the level of serum cholesterol, among men who were more traditionally Japanese than men who were Westernized in their culture and social relations. Because intervention efforts will involve advice about foods, it is important to understand the role foods play.

Murphy et al. (1995) quantified food intake and related it to a negative health outcome, impaired glucose tolerance

(IGT), among Southwest Alaska Yup'ik Eskimos. IGT is associated with diabetes, another risk factor for heart disease. Food-frequency data, using a 15 item self-administered questionnaire and blood-glucose levels, collected from 53% of the residents ≥ 20 years in 15 villages, were compared. Murphy et al. (1995) reported significantly higher beef-pork intake (46.6% vs. 29.4% P = 0.005) among 42 Yup'iks, who were glucose intolerant (either non-insulin dependent diabetes mellitus or impaired glucose tolerance) compared with 853 who were euglycemic. The glucose-intolerant Eskimos also reported eating significantly less salmon-fish (6.6% vs. 19.1% P = 0.01) and significantly more white bread (51% vs. 31.6% P = 0.029).

When the intakes of traditional Native foods from the food frequency are compared with an earlier Alaskan study (Heller and Scott, 1967), the percentages of energy and protein from Native foods (25% and 53%) are shown to be less than those observed in the late 1950's (44% and 85%, TABLE 2-5). Both time and geographic differences may, however, contribute to these differences. Non-Native foods are more available in rural Alaska than they were 40 years ago. Geographic differences are illustrated by the less-frequent consumption of fish among Siberian Yup'iks compared with other Alaska Natives. Fish ranked only 14th in frequency of consumption in the 24-h recall analysis, whereas among Natives elsewhere in Alaska, it ranked 4th (Nobmann et al, 1992).

When Siberian Yup'iks were compared with contemporary Native groups in Yukon, Canada (Wein, 1994), they consumed more energy (25% vs. 17%) but less protein (53% vs. 58%) from Native foods. Wein (1994) noted that traditional foods provided over one-half the protein, zinc, iron, riboflavin,

niacin and vitamin B-12. My data also reflected that traditional foods provided over one-half the protein, iron, and vitamin B-12 plus n-3 fatty acids, arachidonic acid, EPA, animal protein and cholesterol. Of the 91 foods listed on the food frequency, 10 with the greatest amount of saturated fat per average serving included: butter, beef, fry bread, chicken, muktuk, stew with mostly meat, margarine, pilot bread, berry agutuk, and cheese. Thus, traditional foods make a significant contribution for several nutrients, whereas nontraditional foods were frequent sources of harmful saturated fats.

I suggest that a combination of dietary factors operate in protecting and promoting cardiovascular risk Siberian Yup'iks. These data support the concept that the factors affecting cardiovascular health are many and complex (Ulbricht and Southgate, 1991, Hegsted and Ausman, 1988, Willett, 1990). Although my data are limited by small numbers, they constitute more than one half of the Gambell Siberian Yup'ik population ≥ 40 years-of-age. Estimates from the 24-h recall represent the mean intakes in autumn 1992.

Intakes of saturated fats approach recommendations, while polyunsaturated fat intake is within recommendations. Intakes of monounsaturated fats are high, which may have additional advantages if the antioxidant theory is supported. Indeed, substitution of polyunsaturated fats with oleic acid in the diet has been suggested as a strategy to protect LDL from oxidation (Witztum, 1994). Mean intakes of protein, iron, selenium, α-tocopherol, vitamin B-12, folacin (for men), and vitamin C (for men) meet or exceed current Recommended Dietary Allowances (National Research Council, 1989*b*). The RDAs reflect the concurrence of scientific opinion concerning the maintenance of good nutrition for

practically all healthy people in the United States. In contrast, the percent of energy from fat is excessive using current national recommendations, but parallels traditional diets of Alaska Natives which are high in fat and protein and low in carbohydrate.

Appropriateness of the Methodology

I attempted to minimize acknowledged sources of error in estimating dietary intake. These included potential differences between participants and nonparticipants, respondent accuracy, interviewer and coder interpretations, data-base accuracy and completeness, error in nutrient analysis, biological variability of species, location of sample from the animal or plant, season, sex, and methods of food preparation. Respondent accuracy was increased through home-based interviews over what might be expected in a clinical setting because subjects could show the interviewer a food or a portion size, instead of relying on a verbal description.

The sources of energy consumed by women participants varied from those of women nonparticipants (36 women who completed all parts of the investigation were compared with eight women who completed only the dietary component). Women who participated in all phases were significantly ($P < 0.05$) different. They were older, consumed more polyunsaturated fatty acids, and less carbohydrate than women who did not participate fully. Nevertheless, it is more likely that the eight women are less representative of Gambell women than are the 36 based on their smaller number. There were no significant differences in energy sources observed between men participants ($n = 29$) and nonparticipants ($n = 10$).

The two instruments I used to measure dietary intake

are both acceptable for dietary analysis, and provided complementary data. A new food-frequency instrument was developed because many foods typically consumed by the people of Gambell are not included in more widely used food-frequency instruments. Also, seasonality has significant effects on nutrient consumption of Alaska Natives (Nobmann et al., 1992), which a single 24-h recall would not reveal. Because it is important to validate or calibrate an instrument in the population to be studied (Buzzard and Sievert, 1994, Thompson and Byers, 1994, Nelson, 1991),I believed that a rough estimate of the validity of the food frequency could be ascertained from the 24-h recall data, because small-village populations of several hundreds of people in Alaska, do not lend themselves to identifying nonstudy subjects for more formal validation studies. The most common method of assessing the validity of questionnaires, compares the agreement in ranking of subjects between questionnaire and standard (Nelson, 1991). The food frequency and the 24-h recall were consistent in ranking crude fat intake, but inconsistent in ranking percent of energy from fat (TABLE 2-3). There may be insufficient variation in percent of energy from fat intake, however, to rank individuals, as noted in another study (Willett, 1990).

Correlations between the two instruments for fats (0.4-0.5 adjusted for age and sex and 0.3-0.4 when also adjusted for energy, TABLE 2-2) compare favorably with other studies that range from 0.29-0.75 (Willett, 1990, Nelson, 1991, Thompson and Byers, 1994). Nelson (1991) pointed out that neither a questionnaire nor the standard (in this instance the 24-h recall) assesses true intake and the correlation of the two measures is inevitably an underestimate of the

correlation of the questionnaire with the truth. Only a few nutrients in our study were not significantly correlated (TABLE 2-2) including β-carotene, folacin, dietary and water-soluble dietary fiber. This may reflect day to day variations in intake rather than faults with food frequency, because intakes of fruits and vegetables were infrequent. The lack of significant correlations for EPA may be attributed to inconsistent use from day to day of sea mammals or fish. The poor correlation of sodium may be attributed to omitting questions about added salt in the food frequency but asking about it in the 24-h recall.

The higher values of food frequency compared with the 24-h recall can probably be attributed to weaknesses in the food frequency component including inaccurate portion sizes and differences in cognitive processes needed to recall intake in the distant past. Respondents tend to overestimate when given more items on a food frequency (Thompson and Byers, 1994, Krebs-Smith, 1995). There is also a strong tendency to describe portions as "medium" regardless of the quantitative definition ascribed to "medium." Also, individuals give larger estimates of frequency for items eaten more often than they do for items eaten less often (See Smith, 1991). Thus, frequency estimates of bread or pilot bread may be overstated. Despite these exceptions, these data on food frequency can estimate usual intakes of individuals for within-study comparisons.

CONCLUSIONS AND IMPLICATIONS

The diets of Siberian Yup'ik adults in Gambell, Alaska who participated in this study may have several benefits with respect to cardiovascular disease. The percent of energy from monounsaturated fats is significantly greater

than their counterparts in the general US population, and men consume significantly less saturated fats than their general US counterparts. Siberian Yup'iks tend to be closer than the NHANES III participants, to the dietary recommendations of the National Cholesterol Education Program (NCEP), with the exception of the recommendation to limit fat to 30% of energy. Siberian Yup'iks have high intakes of n-3 fatty acids and a favorable PS ratio. Intakes of n-3 fatty acids of men are comparable to those of Greenlandic Eskimos. Although the intakes of n-3 fatty acids appear to be less among women than those of Greenlandic Eskimos in the 1970's (sexes not reported separately), they are still very high and the PS ratios for both sexes are comparable to Greenlandic Eskimos. When compared with the Recommended Dietary Allowances (RDA)(National Research Council, 1989*b*), mean intakes from the 24-h recalls met or exceeded recommendations for protein, iron, selenium, vitamin B-12, total α-tocopherol, folacin (for men), and vitamin C (for men), despite the potential under-estimation due to missing nutrient data.

There are potential problems in the diet when compared with the NCEP recommendations. Intakes of total fat by Siberian Yup'iks are further from the recommended 30% of energy than the national sample. Intakes of fiber are lower than 20-35 g/d, the range recommended by at least one national US group (Life Sciences Research Office, Pilch, 1987). Compared with the RDA, mean intakes were less than the recommended levels for vitamin B-6 for both sexes and for vitamin C for women. Values for vitamin B-6, however, may be artificially low due to missing nutrient data and values for vitamin C may be low due to coding errors.

The positive aspects of the diet outweigh the

negatives, in my opinion, based on the literature and my results. There is reason for concern, however, based on the significant use of non-Native foods, which contribute more saturated fats and less n-3 fatty acids. There is potential for increasing heart disease if nontraditional foods rich in saturated fats make up a larger portion of the diet in the future.

This study describes the diet of > 50% of the population, who are ≥ 40 years in Gambell. Because of the small number of subjects and that additional nondietary variables influence mortality due to cardiovascular disease, predictions about cardiovascular health of this population are limited. More definitive explanations will require analyzing other risk factors concurrently.

Despite the limitations of these estimates, intervention efforts are appropriate to maintain the current desirable dietary practices and reduce undesirable practices for the population as a whole. Current recommendations are consistent with the following recommendations made by Nobmann et al.(1992) for Alaska Natives in general. They were based on the Surgeon General's recommendations, the RDAs and the dietary intakes observed among 351 Alaska Natives at that time. They are: continue to eat traditional fish and lean meat instead of fatty meat; substitute water frequently in place of sugar-containing or caffeine-containing beverages; eat more calcium-rich foods, fruits and vegetables; substitute whole-grain breads for white bread. Additional recommendations are to reduce: energy-dense sweets; cured meats; canned soups; other salty foods; and salt in cooking or while eating.

Recommendations based on the current data, that include more detailed information on fat intakes, are further

customized for Siberian Yup'iks. Dietary recommendations for the prevention of cardiovascular disease among Siberian Yup'iks, also include promote continued consumption of traditional sea mammals, fish and sea mammal fats, such as seal blubber -- which are sources of n-3 fatty acids, monounsaturated fatty acids, and total α-tocopherol. Continue consumption of whale, which is also a source of selenium.

Minimize the intake of foods high in saturated fat. This includes less fatty beef, butter, milk fat, margarine, and processed meats such as frankfurters. Promote the use of chicken without the skin. Encourage discarding the skin and cooking without frying, or encourage store managers to stock alternatives to higher fat, fried chicken, such as skinless fried chicken or plain chicken for home preparation without added fat.

Encourage intake of unsaturated fats over saturated fats. This reduces total and LDL cholesterol. Because monounsaturated fats may not reduce HDL levels as much as n-6 polyunsaturated fatty acids, consumption of monounsaturated fat should be promoted. Use of oils from sea mammals and vegetable oils high in monounsaturated fatty acids, such as canola and olive oil, are recommended. Although high monounsaturated vegetable oils are more consistent with the current proportions of fatty acid intakes, these oils should be used in limited amounts to replace margarine, butter and hydrogenated vegetable fats.

When carbohydrate is used, sources of complex carbohydrates, which contain dietary fiber, such as fruits, vegetables, and whole grains are preferable to the simple carbohydrates of sugar. Promotion of high carbohydrate intake seems inappropriate in this population, given

historically low carbohydrate intakes.

Finally, it is recommended that energy intake and output are balanced to maintain a healthy body weight.

These guidelines are most appropriate for the Siberian Yup'ik population from which the data were gathered. To the extent that people younger than 40 years-of-age in Gambell and in other northern communities share these dietary practices, they also may benefit from these dietary guidelines. Dietary interventions might be geared to the younger people of the community based on the assumption they will continue their current dietary practices as they grow older.

TABLE 2-1
Siberian Yup'ik adults (≥ 40 y) completing dietary and physical assessments by age, October 1992, Gambell, Alaska.

Age, years	Men	Women	Total
40-49	8	12	20
50-59	11	7	18
60-69	6	8	14
70-79	3	5	8
80-89	1	4	5
TOTAL	29	36	65

TABLE 2-2

Correlation coefficients of nutrient intakes from the food frequency questionnaire with the 24-h recall, Siberian Yup'iks (n = 64) October 1992, Gambell, Alaska.

Nutrient[1]	Crude[2] Correlation		Adjusted[3] Correlations			
			Age & Sex		Age, Sex & Energy	
	r	*P*	*r*	*P*	*r*	*P*
Energy	0.456	0.000	0.374	0.003		
Protein	0.337	0.006	0.269	0.034	0.049	0.709
Fat	0.520	0.000	0.491	0.000	0.307	0.016
Carbohydrate	0.227	0.071	0.129	0.319	0.109	0.404
Saturated fat	0.452	0.000	0.430	0.000	0.443	0.000
Monounsaturated fat	0.521	0.000	0.507	0.000	0.337	0.008
Polyunsaturated fat	0.507	0.000	0.484	0.000	0.362	0.004
Percent energy from						
Protein	0.222	0.079	0.210	0.101	0.218	0.091
Carbohydrate	-0.221	0.079	0.177	0.169	0.190	0.143
Fats	0.267	0.033	0.312	0.014	0.311	0.015
Saturated	0.486	0.000	0.512	0.000	0.522	0.000
Monounsaturated	0.329	0.008	0.362	0.004	0.367	0.004

TABLE 2-2 (continued)

Polyunsaturated	0.347	0.005
Myristic acid (14:0)	0.404	0.001
Stearic acid (18:0)	0.336	0.007
Linoleic (18:2)	0.356	0.004
EPA (20:5)	0.233	0.103
n-3 fatty acids	0.320	0.010
n-6 fatty acids	0.338	0.006
Cholesterol	0.223	0.077
Sucrose	0.244	0.052
Sodium	0.170	0.179
Iron	0.275	0.028
Selenium	0.263	0.036
β-carotene	-0.021	0.873
Vitamin B-6	0.121	0.345
Vitamin B-12	0.368	0.003
Folacin	0.050	0.697
Vitamin C	0.261	0.037
α-tocopherol	0.441	0.000
Animal protein	0.313	0.012

0.371	0.003	0.388	0.002
0.380	0.002	0.170	0.191
0.317	0.012	0.420	0.001
0.330	0.009	0.318	0.012
0.027	0.838	-0.135	0.299
0.296	0.019	-0.055	0.676
0.318	0.012	0.444	0.000
0.177	0.170	-0.017	0.899
0.192	0.145	0.158	0.235
0.084	0.526	-0.120	0.370
0.169	0.200	0.031	0.820
0.281	0.031	0.291	0.027
0.000	1.000	0.127	0.343
0.043	0.744	-0.020	0.865
0.312	0.014	0.160	0.222
0.140	0.284	0.167	0.202
0.254	0.048	0.238	0.067
0.410	0.001	0.270	0.037
0.254	0.048	0.075	0.570

TABLE 2-2 (continued)

Dietary fiber	0.125	0.324	0.063	0.629	0.184	0.155
Water soluble fiber	-0.025	0.846	-0.109	0.400	-0.067	0.608

[1]All variables were log transformed to improve normality except linoleic acid, dietary fiber and water soluble dietary fiber.

[2]Pearson correlation coefficients.

[3]Partial correlation coefficients.

TABLE 2-3
Comparison* of results based on food frequency ranking with means from the 24-h diet recall, using fat intakes of 64 Siberian Yup'ik men and women, October 1992, Gambell, Alaska.

Food Frequency Rank (by quartiles)	Mean Intakes from 24-h recall	
	Men	Women
	Total fat (g)	
1	63	49
2	116	58
3	129	101
4	144	151
	% of energy from fat	
1	40	37
2	39	44
3	52	43
4	42	47

*Based on method of Willett (1990).

TABLE 2-4
Summary of 997 foods consumed during one 24-h recall by Siberian Yup'ik adults (n = 65) October 1992, Gambell, Alaska.

Food Group	Number of Times	Percent of foods reported
Beverages	233	22.0
Coffee	95	9.0
Tea	95	9.0
Fruit-flavored beverages	27	2.6
Carbonated beverages	14	1.3
Snacks and Sweets	173	16.4
Sugar	127	12.0
Sugar substitute	21	2.0
Frosting, syrups, jelly, candy	19	1.8
Peanut butter	9	0.9
Popcorn, Potato chips	6	0.6
Meat, poultry and fish	147	13.9
Sea Mammals	49	4.6
Seal meat	22	2.1
Walrus	21	2.0
Whale meat (mallu)	6	0.1
Birds or Poultry	29	2.7
Pork products	19	1.8
All fish	17	1.6

TABLE 2-4 (continued)

All beef	17	1.6
> 22.5% fat	8	0.8
12-22% fat	6	0.6
9%leanest fat	3	0.3
Seafoods(ascidians)	12	1.1
Land animals	4	0.4
Moose	2	0.2
Reindeer	2	0.2
Breads, cereals and baked products	133	12.6
Fats	125	11.8
Margarines	34	3.2
Creamer, Imitation	22	2.1
Seal Blubber, oil	20	1.9
Mayonnaise, oils, shortening	17	1.6
Whale, skin and fat	15	1.4
Butter	13	1.2
Vegetables	67	6.3
Milk products	34	3.2
Fruits	26	2.5

TABLE 2-5
Proportion of energy and nutrients from native foods from three studies.

Nutrient	Present study[1]	Heller & Scott[2]	Wein[3]
Energy	25	47,44	17
Protein	53	85,84	58
Carbohydrate	4	2, 2	1
Fat	31	44,42	16
Saturated fats	23		12
Monounsaturated fats	34		11
Polyunsaturated fats	30		9
N-3 fatty acids	65		
N-6 fatty acids	16		
fatty acid 12:0	0		
fatty acid 14:0	38		
fatty acid 16:0	24		
fatty acid 18:0	18		
fatty acid 18:2	15		
fatty acid 20:4	66		
fatty acid 20:5	100		
Cholesterol	58		44
Sodium	13		
Iron	68	76,75	50
Selenium	26		
ß-carotene	29		
Vitamin B-6	19		43
Vitamin B-12	74		126†
Folacin	7		11
Vitamin C	23	55,50	8
Total α-tocopherol	24		

TABLE 2-5 (continued)

Animal protein	68	
Dietary fiber	7	1
Water soluble fiber	3	

[1]Nutrients from native foods are expressed as percent of all foods consumed by Siberian Yup'iks, based on food frequency analysis.

[2]Heller and Scott, 1967. Values are percents for males and females. Based on mean daily intakes from 4840 diet records from a total population of 2252 in 11 villages.

[3]Wein, 1994. Based on 122 household heads of four Yukon First Nation communities who completed 381 24-h recalls during four seasons. Mean percentage of daily nutrients from traditional foods was based on the number of persons who consumed at least one traditional food on any recall day/total participants, or n/N = 103/122. While the method varies from my method, the values are presented to compare current practices among another Northern Native group.

†Mean daily intake of vitamin B-12 by traditional food consumers exceeded the mean daily intake of all participants.

TABLE 2-6

Proportion of servings from native foods[1] (% of servings, Mean ± SEM) consumed by Siberian Yup'ik men and women by age, October 1992, Gambell, Alaska.

Age Group (yrs)	Men	Women	Both	P^2
40-59	11.5 ± 2.36	7.8 ± 0.98	9.7 ± 1.30	0.162
60-87	7.0 ± 1.00	9.2 ± 0.93	8.3 ± 0.71	0.149
Total	10.0 ± 1.62	8.4 ± 0.68	9.1 ± 0.82	0.386
P^2	0.093	0.343	0.367	

[1] Based on food frequency data.

[2] t tests for independent means. Differences were not significant ($P > 0.05$) with or without log transformation.

TABLE 2-7

Mean ± SEM daily nutrient intakes of Siberian Yup'ik adults based on one 24-h recall. Median is given in parentheses. Data are for October 1992, Gambell, Alaska.

Nutrient	Men, *n* = 29	Women, *n* = 36	Significance of difference
Energy,kJ	9288 ± 758 (8350)	7078 ± 774 (6199)	*[1,2]
[kcal	2219 ± 181 (1995)	1691 ± 185 (1481)	*]
Protein, g	128 ± 19 (85)	86 ± 11 (63)	*
Carbohydrate,g	176 ± 13 (176)	140 ± 12 (138)	ns
Fat, g	113 ± 13 (100)	86 ± 14 (68)	*
Fatty Acids			
Saturated, g	25 ± 2.3 (26)	20 ± 2.2 (18)	ns
Monounsaturated,g	45 ± 4.9 (39)	39 ± 6.1 (29)	ns
Polyunsaturated,g	21 ± 2.3 (17)	17 ± 2.5 (13)	ns
14:0 (myristic),g	2.7 ± 0.4 (2.2)	2.3 ± 0.4 (1.4)	ns
18:0 (stearic), g	6.3 ± 0.8 (4.3)	4.7 ± 0.5 (3.9)	ns

TABLE 2-7 (continued)

18:2 (linoleic),g	11.1 ± 1.3	(9.9)
20:5 (EPA), g	2.7 ± 0.8	(0.8)
n-3 fatty acids[3]	8.6 ± 2.2	(3.2)
n-6 fatty acids[4]	11.5 ± 1.3	(10.8)
Cholesterol, mg	280 ± 36	(199)
PS Ratio	0.96 ± 0.11	(0.79)
CSI Ratio	39 ± 3.7	(37)
Sucrose, g	48 ± 6.6	(42)
Percent of Energy from		
Protein	21.6 ± 1.8	(19.8)
Carbohydrate	35.6 ± 2.7	(35.9)
Fat	43.5 ± 2.3	(42.5)
Saturated	10.5 ± 0.8	(10.7)
Monounsaturated	17.6 ± 1.0	(17.8)
Polyunsaturated	7.8 ± 0.4	(7.8)
Sodium, mg	3105 ± 336	(2809)
Iron, mg	45 ± 9.2	(31)

8.9 ± 1.1	(8.1)	ns
1.9 ± 0.6	(0.7)	ns
5.7 ± 1.6	(2.5)	ns
9.2 ± 1.1	(8.4)	ns
241 ± 29	(231)	ns
0.84 ± 0.06	(0.83)	ns
32 ± 3.2	(29)	ns
40 ± 6.4	(30)	ns
21.6 ± 1.7	(19.7)	ns
36.4 ± 2.1	(36.0)	ns
41.8 ± 2.2	(41.9)	ns
10.7 ± 0.7	(9.5)	ns
18.5 ± 1.2	(17.5)	ns
8.1 ± 0.5	(7.8)	ns
2250 ± 211	(2253)	*
33 ± 7.0	(14)	ns

TABLE 2-7 (continued)

Selenium, μg	129 ± 27 (101)	95 ± 11 (79)	ns
β-carotene, μg	2798 ± 804 (640)	1434 ± 387 (399)	ns
Vitamin B-6, mg	1.91 ± 0.30 (1.37)	1.01 ± 0.13 (0.88)	**
Vitamin B-12, μg	15.6 ± 3.51 (8.7)	10.0 ± 2.14 (4.1)	ns
Folacin, μg	231 ± 36.6 (134)	152 ± 16.7 (119)	ns
Vitamin C, mg	88 ± 20.4 (61)	41 ± 7.3 (28)	*
α-tocopherol eq, mg	13.3 ± 1.94 (9.5)	8.0 ± 1.28 (5.7)	**
Animal protein, g	110 ± 19 (57)	73 ± 10 (52)	*
Dietary fiber, g	13.6 ± 1.52 (12.0)	9.2 ± 0.96 (10.4)	*
Water soluble fiber, g	3.9 ± 0.45 (3.6)	2.6 ± 0.26 (2.8)	*

[1] Two way ANOVA tests were used to evaluate significant differences for sex and age.

[2] All variables were log transformed to improve normality, with the exception of 18:2 (linoleic acid), dietary fiber, and water soluble dietary fiber.

[3] n-3 fatty acids = 18:3 + 20:5 + 22:5 + 22:6

[4] n-6 fatty acids = 18:2 + 20:4

* $P < 0.05$. ** $P < 0.01$. ns = not significant.

TABLE 2-8

Comparison of younger (40-59 y) with older Siberian Yup'iks' mean (± SEM) daily nutrient intakes[1]. Data are based on one 24-h recall, by men and women, October 1992, Gambell, Alaska. Median estimates are shown in parentheses.

Nutrient[2]	Men, *n* = 29	Women, *n* = 36
Energy, kJ		
40-59 y[3]	9196 ± 854 (7835)	7877 ± 883 (7471)
≥ 60 y[4]	9459 ±1540 (10656)	6182 ± 1306 (4432)
F[5]	0.763	0.049*
[kcal		
40-59 y	2197 ± 204 (1872)	1882 ± 211 (1785)
≥ 60 y	2260 ± 368 (2546)	1477 ± 312 (1059)
F	0.763	0.049*]
Carbohydrate, g		
40-59 y	188 ± 18 (179)	153 ± 13 (148)
≥ 60 y	155 ± 19 (155)	125 ± 19 (106)
F	0.309	0.106
Fat, g		
40-59 y	113 ± 16 (93)	100 ± 15 (75)
≥ 60 y	113 ± 25 (111)	71 ± 23 (34)
F	0.052	0.027*

TABLE 2-8 (continued)

Saturated fats, g			
40-59 y	25.5±	3.0	(26.2)
≥ 60 y	23.6±	3.8	(22.6)
F	0.65		
Monounsaturated fats, g			
40-59 y	45.3±	5.7	(34.6)
≥ 60 y	44.6±	9.7	(44.5)
F	0.457		
Polyunsaturated fats, g			
40-59 y	20.1±	2.6	(16.6)
≥ 60 y	21.4±	4.8	(21.2)
F	0.653		
CSI Ratio			
40-59 y	39.3±	4.5	(43.9)
≥ 60 y	38.5±	6.6	(37.0)
F	0.870		
Protein, % of energy from			
40-59 y	19.6±	1.9	(15.5)
≥ 60 y	25.5±	3.8	(20.7)
F	0.149		

23.3±	2.6	(21.9)
15.9±	3.3	(10.3)
0.022*		
45.3±	7.4	(32.8)
31.0±	9.9	(15.1)
0.032*		
19.1±	2.7	(14.7)
13.6±	4.3	(7.6)
0.022*		
38.0±	4.2	(38.6)
25.4±	4.6	(17.8)
0.010*		
18.4±	2.1	(15.3)
25.2±	2.5	(24.2)
0.049*		

TABLE 2-8 (continued)

Fat ,% of energy from			
40-59 y	44.3±	2.2	(43.2)
≥ 60 y	42.1±	5.3	(40.2)
F	0.371		
Saturated fat, % of energy from			
40-59 y	10.7±	1.1	(10.7)
≥ 60 y	10.1±	1.4	(10.2)
F	0.751		
Monounsaturated fat, % of energy from			
40-59 y	18.0±	1.0	(18.9)
≥ 60 y	16.8±	2.2	(16.1)
F	0.317		
Polyunsaturated fat, % of energy from			
40-59 y	7.9±	0.5	(7.3)
≥ 60 y	7.6±	0.8	(7.9)
F	0.577		
Vitamin B-12, μg			
40-59 y	11.1±	2.4	(8.7)
≥ 60 y	24.1±	8.8	(15.3)
F	0.350		

45.8±	2.9	(45.2)
37.4±	3.2	(38.3)
0.081		
11.4±	1.0	(10.8)
9.9±	1.1	(8.5)
0.215		
20.5±	1.6	(19.2)
16.2±	1.6	(14.6)
0.101		
8.8±	0.6	(8.4)
7.3±	0.8	(6.8)
0.071		
8.8±	2.5	(3.8)
11.3±	3.6	(4.5)
0.595		

TABLE 2-8 (continued)

Vitamin C, mg						
40-59 y	100	± 29	(61)	54	± 11	(38)
≥ 60 y	64	± 23	(38)	28	± 8	(15)
F	0.145			0.018*		
Dietary fiber, g						
40-59 y	15.5	± 2.0	(18.0)	10.7	± 1.3	(11.6)
≥ 60 y	9.9	± 2.0	(8.5)	7.5	± 1.3	(5.2)
F	0.081			0.095		
Water soluble fiber, g						
40-59 y	4.6	± 0.6	(3.7)	3.1	± 0.4	(3.0)
≥ 60 y	2.7	± 0.5	(2.1)	2.0	± 0.3	(1.9)
F	0.047*			0.034*		

[1] Nutrients displayed if two way ANOVA tests revealed significant differences for sex or age.

[2] All variables were log transformed to improve normality, except dietary fiber and water soluble dietary fiber.

[3] n = 19 men, 19 women; [4] n = 10 men, 17 women.

[5] P value from one way ANOVA used to test for differences between age groups for each sex. * $P < 0.05$, ** $P < 0.01$ between age groups.

TABLE 2-9

Comparison of mean (± SEM) nutrient intakes of Siberian Yup'ik men (*n* = 29) and women (*n* = 35) based on food frequency responses. Data are for autumn 1992 in Gambell, Alaska. Median values are shown in parentheses.

	Men				Women				[1,2]
Food energy, kJ	14482	±	1833	(12368)	8651	±	732	(7300)	**
[Food energy, kcal	3460	±	438	(2955)	2067	±	175	(1744)	**]
Protein, g	168	±	32	(132)	95	±	7.3	(88)	**
Carbohydrate, g	372	±	44	(337)	231	±	24	(180)	**
Fat, g	147	±	22	(104)	87	±	7.9	(76)	**
Fatty acids									
Saturated, g	42	±	6.1	(33)	28	±	2.3	(20)	**
Monounsaturated, g	65	±	10.1	(45)	39	±	3.6	(31)	*
Polyunsaturated, g	27	±	3.8	(21)	18	±	1.5	(15)	*
PS ratio	0.71	±	0.04	(0.70)	0.78	±	0.03	(0.79)	*
N-3 fatty acids, g	4.5	±	0.9	(3.4)	2.6	±	0.3	(2.4)	*

TABLE 2-9 (continued)

20:5 (EPA), g	1.4	±
N-6 fatty acids, g	20.3	±
20:4 (Arachidonic), g	0.5	±
Cholesterol, mg	583	±
% of energy from		
Protein	19	±
Carbohydrate	44	±
Fat	37	±
Saturated fats	11	±
Monounsaturated fats	16	±
Polyunsaturated fats	7	±
N-3 fatty acids	1	±
20:5(EPA)	0.3	±
N-6 fatty acids	5	±
20:4 (Arachidonic)	0.1	±

0.4	(0.9)	0.7	±	0.1	(0.6)	ns
2.8	(15.3)	13.7	±	1.2	(11.5)	ns
0.1	(0.3)	0.3	±	0.03	(0.2)	**
114	(463)	330	±	38	(304)	**
1.2	(18)	20	±	1.0	(19)	ns
2.2	(44)	44	±	1.8	(44)	ns
1.6	(38)	38	±	1.3	(39)	ns
0.7	(10)	10	±	0.4	(10)	ns
0.8	(17)	17	±	0.6	(17)	ns
0.4	(7)	8	±	0.3	(8)	ns
0.1	(1)	1	±	0.1	(1)	ns
0.04	(0.3)	0.3	±	0.03	(0.3)	ns
0.4	(5)	6	±	0.3	(6)	ns
0.01	(0.1)	0.1	±	0.01	(0.1)	ns

TABLE 2-9 (continued)

Minerals								
Selenium, μg	186	±	28	(136)	110	±	10	(95) *
Sodium, mg	3739	±	530	(2924)	2338	±	220	(1927) ns
Vitamins								
Vitamin A,total μg RE	5594	±	1502	(3083)	3511	±	500	(2952) ns
Vitamin C, mg	304	±	77	(203)	184	±	31	(143) ns
Total α-tocopherol eq	17	±	2.6	(14)	12	±	1.0	(10) ns
Folacin, μg	566	±	107	(429)	381	±	46	(332) ns

[1] Two way ANOVA tests were used to evaluate significant differences for sex and age.

[2]All variables were log transformed to effect normality, with the exception of % energy from polyunsaturated fat, % energy from 20:4, sodium/1000 kcal and total α-tocopherol/1000 kcal.

*Significantly different at $P < 0.05$.
**Significantly different at $P < 0.01$.

TABLE 2-10

Comparison of mean nutrient intakes of Siberian Yup'ik adults with the general US population represented by NHANES III participants[1,2], based on one 24-h recall[3]. Participants are listed by sex. For SEM see Table 2-7.

Nutrient	Men			Women		
	Siberian Yup'iks (n=29)	General US (n=2385)	Signif. diff.	Siberian Yup'iks (n=36)	General US (n=2338)	Signif. diff.
Energy, kJ	9288	9555[5]	ns	7078	6709	ns
[kcal	2219	2283	ns	1691	1603	ns]
Protein, g	128	89	ns	86	63	*
Carbohydrate, g	176	267	***	140	200	***
Fat, g	113	89	ns	86	61	ns
Saturated fat, g	25	30	*	20	21	ns
Monounsaturated fat, g	45	34	*	39	23	*

TABLE 2-10 (continued)

Polyunsaturated fat, g	21	19
Cholesterol, mg	280	316
% of energy from		
Protein	21.6	16.0
Carbohydrate	35.6	47.5
Fat	43.5	34.4
Saturated fat	10.5	11.6
Monounsaturated fat	17.6	12.9
Polyunsaturated fat	7.8	7.2
Vitamin A, μg RE	11734	1200
β-carotene, μg	2798	572
Vitamin B-6, mg	1.91	2.09
Vitamin B-12, μg	15.6	5.0
Folacin, μg	231	318

ns	17	13	ns
ns	241	214	ns
**	21.6	16.2	**
***	36.4	50.7	***
***	41.8	33.5	***
ns	10.7	11.3	ns
***	18.5	12.3	***
ns	8.1	7.3	ns
*	5312	983	*
**	1434	518	*
ns	1.01	1.52	***
ns	10.0	6.2	ns
*	152	248	***

TABLE 2-10 (continued)

Vitamin C, mg	88	111	ns	41	97	***
α-tocopherol eq, mg	13.3	10.3	ns	8.0	7.7	ns
Iron, mg	45.0	17.2	**	32.6	12.3	**
Potassium, mg	2719	2782	ns	1714	2415	***
Sodium, mg	3105	3602	ns	2250	2632	ns
Total fiber, g	13.6	17.9	**	9.2	13.7	***

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[1] McDowell et al, 1994.

[2] Alaimo et al, 1994.

[3] Caution should be exercised in interpreting the means and significance test results which assume normality, as both NHANES III and Siberian Yup'ik data are very skewed, due to day to day variations in food and nutrient intake.

[4] All NHANES data weighted to proportions in Alaska sample by age.

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

TABLE 2-11

Comparisons between Siberian Yup'iks with other native populations and sub-populations of the general US population and Denmark in the intakes of protein, carbohydrate and fats. Mean percents of energy were based on one 24-h recall (Table 2-5) and data from Alaska Natives, Chukotka Natives, Greenland Eskimos, Tarahumara Indians, US NHANES III and Danes. Number of participants is shown in parentheses.

Study (*n*)	Protein	CHO	Fats				
			Total	SFA	MUFA	PUFA	PS Ratio
Siberian Yup'iks[1]							
men,(29)	21.6	35.6	43.5	10.5	17.6	7.8	0.96
women, (36)	21.6	36.4	41.8	10.7	18.5	8.1	0.84
Total, (65)	21.6	36.0	42.6	10.6	18.1	8.0	0.89
Alaska Natives[2]							
men, (102)	19	41	38				
women, (97)	18	44	37				
Greenlandic Eskimos[3]							
all, (50)	23	38	39	8.9[4]	22.3	7.5	0.84
Danes[3] (unstated)	11	47	42	22.1[4]	14.5	5.3	0.28

TABLE 2-11 (continued)

Chukotka, Russia[5]							
Tundra men, (104)	23.9	34.5	30.5	14.9	12.3	2.6	0.24
Coastal men, (165)	31.0	23.8	38.2	11.4	15.4	6.6	0.52
US NHANES III[6]							
men, (2385)	16.0	47.5	34.4	11.6	12.9	7.2	
women, (2338)	16.2	50.7	33.5	11.3	12.3	7.3	
Tarahumara Indians, Mexico[7]							
men, (108)			12	3	4	5	2.1
women, (66)			11	3	4	5	2.4

[1] Based on one 24-h recall.

[2] Up to five 24-recalls per person averaged for Alaska Natives, Nobmann et al., 1989.

[3] The double portion technique was used to sample data for Greenlandic Eskimos, Bang et al., 1980.

[4] I estimated the % of energy from saturated, monounsaturated, and polyunsaturated fats from the published % of total fatty acids and % of total fat (Bang et al., 1980).

[5] Based on one 24-h recall, Nikitin et al., 1991.

[6] Based on one 24-h recall, McDowell et al., 1994.

[7] Diet histories were collected for Tarahumara Indians, Connor et al., 1978.

TABLE 2-12

Dietary factors associated with cardiovascular disease risk.

Risk factors	Protective factors	Debated factors[1]
Saturated fatty acids	Moderate alcohol	Ascorbic acid
Cholesterol	C18:2, Linoleic acid	Sucrose
C14:0, Myristic acid	n-3 fatty acids	Iron
Animal protein[2]	Polyunsaturated fats	Dietary fiber
Trans-fatty acids[2]	Monounsaturated fats	C18:0, Stearic acid
Carbohydrate[2]	Water soluble fiber[2]	Vitamin A
Fat[2]	ß-carotene[2]	
C16:0, Palmitic	Vegetable protein[2]	
12:0, Lauric acid	Selenium	
	Vitamin B-6[2]	
	Vitamin B-12[2]	
	Folic acid	
	Energy (physical activity surrogate[2])	
	C20:5n-3, EPA	
	C22:6n-3, DHA	

TABLE 2-12 (continued) 141

n-6 fatty acids

Vitamin E

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[1] Evidence in the scientific literature is limited, conflicting, or negative for effect.

[2] Most, but not all, evidence supports effect.

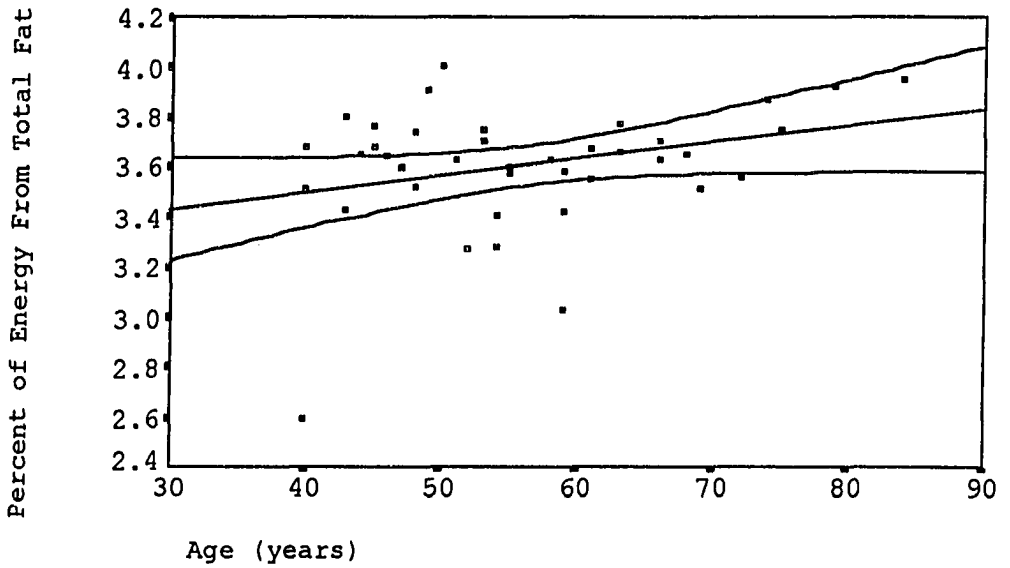

FIGURE 2-1 Relationship of percent of energy from total fat with age among 29 Siberian Yup'ik men with 95% confidence region, 1992 (R^2 = 0.154, P < 0.05). Fat intake from food frequency questionnaire (log transformed).

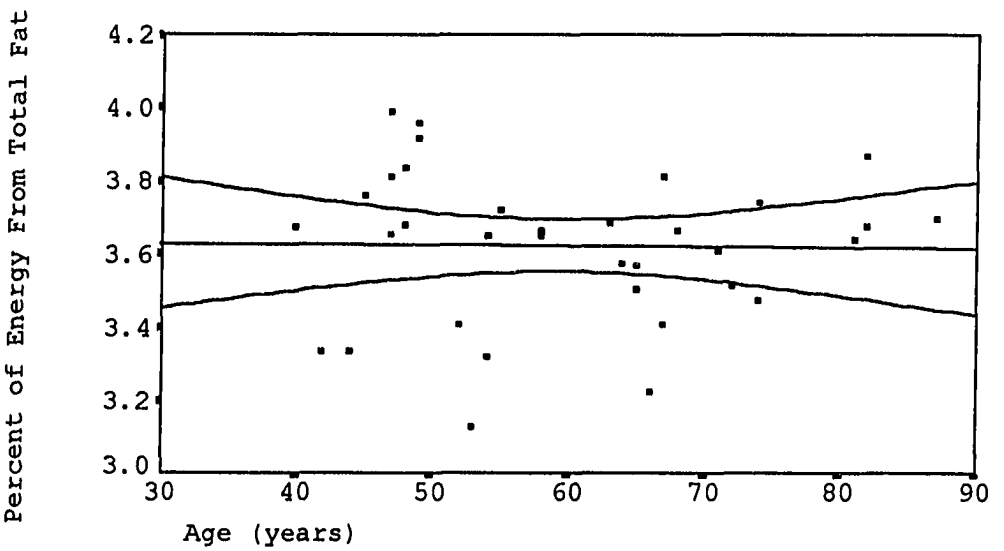

FIGURE 2-2 Relationship of percent of energy from total fat with age among 36 Siberian Yup'ik women with 95% confidence region, 1992 (R^2 = 0.000, P > 0.05). Fat intake from food frequency questionnaire (log transformed).

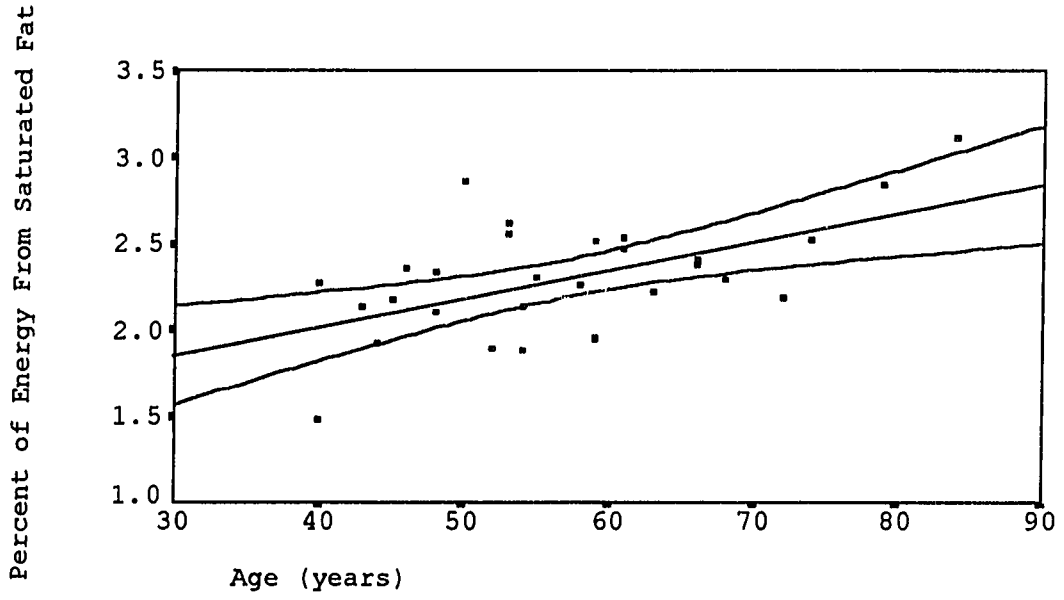

FIGURE 2-3 Relationship of percent of energy from saturated fat with age among 29 Siberian Yup'ik men with 95% confidence region, 1992 (R^2 = 0.305, P < 0.01). Saturated fat intake from food frequency questionnaire (log transformed).

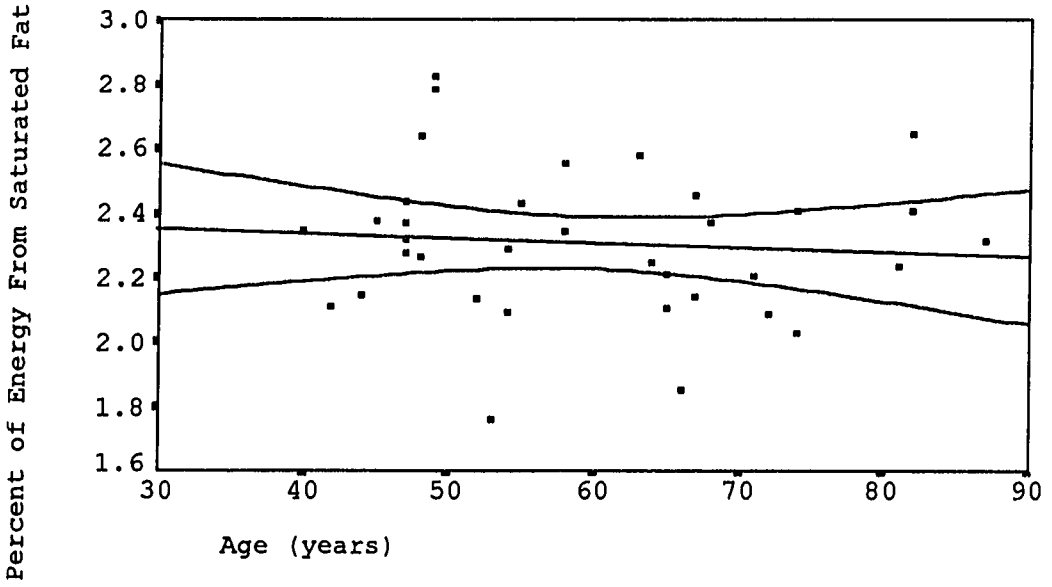

FIGURE 2-4 Relationship of percent of energy from saturated fat with age among 36 Siberian Yup'ik women with 95% confidence region, 1992 (R^2 = 0.007, P > 0.05). Saturated fat intake from food frequency questionnaire (log transformed).

CHAPTER 3

ASSOCIATIONS BETWEEN DIETARY FACTORS AND SERUM LIPIDS RELATED TO CARDIOVASCULAR DISEASE AMONG SIBERIAN YUP'IKS OF ALASKA

ABSTRACT

The diet of northern Native people is reported to protect against cardiovascular disease. In 1992, frequency of consumption of 91 foods among 64 Siberian Yup'ik adults (> 40 years-of-age) was measured during home-based interviews. Nutrient and food intakes were correlated with measurements of blood cholesterol, LDL, HDL, triglycerides and LDL-HDL ratio. Foods and nutrients that were significantly correlated with the LDL-HDL ratio, explained 42% of the variation of the LDL-HDL ratio among all subjects in regression analysis. When body mass index (BMI) was included 59% of the variation was explained. Coefficients were negative for α-tocopherol, fresh bird, evaporated milk, and cheese, and positive for BMI, syrup and pizza. BMI had a positive effect on the LDL-HDL ratio among younger adults, women and all participants, but it did not contribute to explaining the variation in the LDL-HDL ratio among older adults or men. This supports emphasizing weight control among younger Siberian Yup'ik women. Generally, traditional foods were negatively correlated with the LDL-HDL ratio whereas non-traditional or western foods were positively correlated, supporting consumption of native foods for cardiovascular health among Siberian Yup'iks.

INTRODUCTION

The diet of Siberian Yup'ik Natives of Alaska is unusually high in sea mammals and limited in variety of Western foods (Chapter 2). Studies in Greenland (Bang et al., 1980) and Siberia (Nikitin et al., 1991) showed similarly high intakes of sea foods. Consumption of fish, which is high in n-3 fatty acids, has been related to reduced coronary heart disease among men in both a prospective study (Kromhout et al., 1985) and a randomized-controlled trial of the effect of different diet interventions on death and myocardial reinfarction (Burr et al., 1989). Cardiovascular disease has been lower among Alaska Natives than in the general US population. Because: 1) a vast body of knowledge has linked intakes of fat with serum lipid levels; and 2) serum lipids can characterize the degree of risk from cardiovascular disease, I investigated, among Siberian Yup'ik adults, whether various dietary factors correlated with serum lipids. In the absence of data on the prevalence of cardiovascular disease, serum lipids were used as the most proximal surrogate of risk from cardiovascular disease. Serum lipids were selected as the outcome measures because, among the US population in general, 43% of coronary heart disease is attributable to high-blood cholesterol. It is the major modifiable risk factor for coronary-heart disease when compared with proportions attributable to inactivity (35%), high blood pressure (25%), smoking (22%), obesity (17%) and diabetes (8%) (Brownson et al., 1993).

This paper reports on the relationships between serum cholesterol, high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglycerides, and LDL-HDL ratio levels among Siberian Yup'ik adults with

their consumption of energy, 35 nutrients, 91 foods, body mass index (BMI)[1] and smoking practice. Included dietary nutrients were protein, carbohydrate, fat, saturated, polyunsaturated and monounsaturated fats, n-3, n-6 and selected fatty acids, cholesterol, vitamins (ß-carotene, B-6, B-12, C, E, folacin), minerals (iron, selenium, sodium) and energy. Foods included sea mammals, fish, seal oil, beef, sources of *trans*-fatty acids, and a combination of seafoods (sea mammal fish index [SMFI]). The rationale for this investigation was to shed light on the relation of diet to the following risk factors associated with cardiovascular disease: hypercholesterolemia; hypertriglyceridemia; elevated low density lipoprotein; low levels of high-density lipoprotein; and elevated LDL-HDL ratios. My general hypothesis states types of foods eaten by Siberian Yup'iks affect these biomarkers. Six specific hypotheses were tested. McNamara (1990) states that the LDL-HDL ratio is one of the strongest determinants of CHD risk based on the importance of low HDL in cardiovascular disease risk documented by Gordon et al. (1989). I used the LDL-HDL ratio to test my general hypothesis using a multiple regression approach.

METHODS

Field techniques

Usual dietary intakes during the previous 1 year were determined by home-based surveys in September and October 1992, using a 91-item food-frequency questionnaire. A detailed description of the collection and nutrient analysis appears in Chapter 2.

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[1] *Body mass index = (body weight [kg] / height ^{2} [m]) (Keys et al., 1972)*

Venous blood samples were obtained from participants after they fasted for 12 h. The samples were frozen and stored for up to 6 days in the field, then stored at -70 F° in Anchorage until they were shipped to Medlantic Research Institute, Washington, DC for analysis. Samples were analyzed using methods established for the Strong Heart Study (Lee, et al., 1990, See also Ebbesson et al. in preparation, for more detailed analysis of lipids). Total cholesterol, total triglyceride, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol levels are summarized and compared with levels generally associated with increased risk (TABLE 3-1). Participants were weighed dressed in their underwear on a standard balance beam scale to the nearest 0.1 kg and their heights were measured without shoes to the nearest 1 cm.

Statistical analyses

Nutrients and foods were log transformed when it improved the normality of the distribution. Pearson bivariate correlations were generated using the blood lipids and the LDL-HDL ratio with nutrients. Correlations were generated for the entire sample, by sex and by age group (40-59 years old [y] and 60-87 y). When nutrients were significantly correlated for any age or sex group, the nutrients were further analyzed by adjusting for energy and correlating them with blood lipids.

Nutrient intakes were controlled for energy using the residual method of Willett[2] (1990) in the following manner.

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[2] *(Calorie adjusted intake = a + b, where a = residual for subject from regression model with nutrient intake as the dependent variable and total calorie intake as the independent variable and b = the expected nutrient intake for a person with mean caloric intake (Willett and Stampfer, 1986).*

Nutrients were log transformed prior to computations to improve the normality of the distribution and the inequality of variance when residuals were plotted (Shekelle et al., 1987, and Willett, 1990). Scatterplots of log-transformed energy (independent) and nutrient intakes (dependent) were used to locate the expected nutrient intake at the mean caloric intake. The expected nutrient intake was added to each residual to create the energy adjusted intake.

Because the LDL-HDL ratio is considered one of the strongest determinants of risk from coronary heart disease (McNamara, 1990), and because both LDL and HDL levels can be altered by diet (Sacks et al., 1986), the LDL-HDL ratio was selected as the outcome measure for further analysis.

An index representing sea mammal and fish intake was generated by summing the number of servings per year of selected foods, as follows:

Daily Sea Mammal Fish Index (SMFI) = Sum of servings per year (Fresh Fish, Walrus, Muktuk (outer skin and attached blubber of whale), Mukluk (Bearded seal), Dry Fish, Dry Seal, Seal Oil) / 365.2.

The SMFI was correlated with total serum cholesterol, HDL, LDL, triglycerides and LDL-HDL ratio.

Statistical significance was defined as $P < 0.05$ unless otherwise noted. Although numerous correlations were generated, no adjustments were made to account for 1 of 20 correlations may be occurring by chance. This permitted all possible significant ($P < 0.05$) correlations to be identified. Generating simple correlations was essential to understanding this diet and cardiovascular disease system, by modeling variables suggested in the literature. The *P* values are presented for comparative purposes. It is recognized that *P* values would need to be smaller to infer

significance.

To explain the variance in the LDL-HDL ratio, multiple regression was performed after log transformation of all variables. Regression analyses were conducted on the entire sample, by gender and by age group. Separate regression analyses were conducted on foods and nutrients that were selected if they were significantly correlated with the LDL-HDL ratio. When multicollinearity occurred among the nutrients, the nutrient with the greatest correlation with the LDL-HDL ratio was selected for the regression. The same nutrients were entered in the regressions with the LDL-HDL ratio and nutrients, for men, women, younger participants and all participants. Nutrients were selected for entry if they were significantly correlated among any group. Among older participants n-3 fatty acid was also entered while α-tocopherol was not. Nutrients remaining in the regression equations were combined with foods that were significantly correlated to determine relative effects of both nutrients and foods. Two other variables that could confound results were entered in the regression equations, body-mass index and smoking (presence or absence of smoking).

RESULTS

The age and sex of the Siberian Yup'ik residents of Gambell, Alaska, $\geq$ 40 years-of-age, who participated, are described in TABLE 2-1 (Chapter 2). One woman over 59 years-of-age did not answer the food frequency questions, resulting in 64 participants described in Chapter 3. Mean blood lipids are compared with generally accepted thresholds for increased risk in TABLE 3-1.

Initial hypotheses

Several hypotheses were formulated prior to data analyses and results are summarized in the text and in TABLES 3-2 through 3-4.

Hypothesis 1: Sea mammal and fish intake is negatively correlated with total cholesterol, LDL and triglyceride blood levels and positively correlated with HDL levels.

While no significant Pearson bivariate correlations were observed for the SMFI and blood lipids among the entire sample, there were several notable findings among age and sex groups. The only negative correlations which were obvious were with LDL and LDL-HDL ratio among younger men (r = -0.483, P = 0.036 and r = -0.510, P = 0.026 respectively, TABLE 3-2).

Among people over 60, there were two strong associations, a positive association with the LDL-HDL ratio (r = 0.583, P = 0.002) and a negative association with HDL (-0.507, P = 0.008). Among older women positive associations were observed with LDL (r = 0.595, P = 0.015) and the LDL-HDL ratio (r = 0.625, P = 0.010). Among older men, there was a negative association with HDL (r = -0.877, P = 0.001) and a positive correlation with triglycerides (r = 0.653, P = 0.041). Overall positive correlations (6) outweighed hypothesized negative correlations (4). Mostly there were no consistent correlations, giving strong support to reject the hypothesis, except for levels of LDL and SMFI among young men.

Hypothesis 2: Saturated fatty acid intake is positively correlated with total cholesterol, LDL and triglycerides and negatively correlated with HDL levels.

No strong positive correlations were observed. There was a negative correlation between serum cholesterol and

dietary saturated fat among all participants (r = -0.290, P = 0.020, nonenergy adjusted, TABLE 3-3). When energy adjusted, the correlation was less obvious (r = -0.241, P = 0.055, TABLE 3-4). The pattern of negative correlations seen among younger and older men before energy adjusting were maintained after energy adjustment among older men but not among younger men. No significant correlation was noted for any age group of women.

Similar negative correlations were seen among all participants between LDL and dietary saturated fat (non-energy adjusted r = -0.281, P = 0.025), but the energy adjusted, saturated fat correlation was less apparent (r = -0.240, P = 0.056). The obvious correlation for the entire sample is attributed only to the male segments (TABLE 3-3).

There were no significant correlations between HDL and saturated fat or between triglycerides and saturated fat (TABLES 3-3, 3-4).

Hypothesis 3: Monounsaturated fatty acid intake is negatively correlated with total cholesterol, LDL and triglycerides.

Monounsaturated fatty acid intake among all participants (unadjusted for energy, TABLE 3-3) was negatively correlated with LDL (r = -0.252, P = 0.045), not strongly correlated with serum cholesterol (r = -0.241, P = 0.055), and not correlated with triglycerides (r = -0.001, P = 0.992). Among all men monounsaturated fat was negatively correlated with LDL (r = -0.592, r = 0.001) and cholesterol (r = -0.539, P = 0.003). Among younger men negative correlations were observed (r = -0.601, P = 0.006 with LDL and r = -0.510, P = 0.026 with cholesterol). They were also observed among older men (r = -0.739, P = 0.015

with LDL and r = -0.747, P = 0.013 with cholesterol). After energy adjusting (TABLE 3-4), only LDL and cholesterol levels of all men remained notably negatively correlated with monounsaturated fatty acids (r = -0.437, P = 0.018 and r = -0.428, P = 0.021, respectively). Among older men cholesterol remained strongly negatively correlated (r = -0.678, P = 0.031).

Hypothesis 4: High monounsaturated fatty acid intake is positively correlated with HDL in men and women.

No significant correlations were observed in unadjusted values (TABLE 3-3) among all subjects (r = -0.016, P = 0.899) or when the sexes were separated (men, r = 0.164, P = 0.395, women, r = -0.008, p = 0.962), or when sexes were divided by sex and age (40-59 y vs. 60-87 y). No significant correlations were observed when adjusted for energy (TABLE 3-4).

Hypothesis 5: High n-3 fatty acid intake is correlated with high HDL in men and women.

There were no significant correlations using unadjusted n-3 fatty acid intakes for the group(r = -0.059, P = 0.642 TABLE 3-3), for men (r = 0.124, P = 0.523) or for women (r = -0.068, P = 0.696). No correlations were significant after energy adjustment.

Hypothesis 6: Seal oil or fish consumption or n-3 fatty acid intake is negatively correlated with impaired glucose tolerance and diabetes.

When intakes of seal oil and fresh fish were correlated with blood glucose levels (two hours after a 75 g glucose load), there were no significant findings for the entire group or among each sex. When glucose levels were correlated with seal oil among the seven women with the highest levels (>140 mg/dL), there was a positive correlation (r = 0.785,

P = 0.021) but when one woman who consumed the most (almost 40 T/y) was excluded, the correlation was no longer significant (r = -0.209, P = 0.653). An association of glucose level with intake of fresh fish then became obvious but the correlation was positive (r = 0.833 P = 0.039).

Further analyses

Because only two hypotheses were partially supported (#1 a negative correlation was observed between the SMFI and LDL among young men only, and #3 negative correlations between monounsaturated fat and LDL for men, and cholesterol for all men and older men, using energy adjusted values), further analyses were conducted. To better understand the correlations of blood lipids with the SMFI, the seven foods used to define the index were correlated separately.

Correlations of foods with blood lipids

Of the seven foods (not log transformed), none were correlated with the four serum levels until the data were analyzed by sex (data not shown). Among women, fresh fish (values for whitefish were used) and muktuk were notably correlated with cholesterol (r = 0.459 P = 0.006, r = 0.361 P = 0.033, respectively). Fish and muktuk were also correlated with LDL levels (r = 0.429, P = 0.010, r = 0.392 P = 0.020, respectively). Among older women the association between fresh fish and cholesterol was similar but less notable (r = 0.483 P = 0.058). Among younger women, the correlations with cholesterol were also similar for fresh fish (r = 0.496 P = 0.031) and for muktuk (r = 0.584 P = 0.009); with LDL and fresh fish (r = 0.488 P = 0.034), and LDL and muktuk (r = 0.553 P = 0.014). Among younger and older men there were no significant associations between

fresh fish and cholesterol. Among older men, however, there was a positive association for fresh fish with triglycerides (r = 0.717 P = 0.019) and a negative association with HDL (r = -0.655 P = 0.040).

When log-transformed values for foods were correlated among women, fresh fish was still significantly correlated with cholesterol (r = 0.381, P = 0.024) and LDL (r = 0.402, P = 0.017), but muktuk was not. Among older women, dried fish and LDL were correlated (r = 0.525, P = 0.037). Among younger women, correlations were observed for cholesterol and muktuk (r = 0.503, P = 0.028), cholesterol and fresh fish (r = 0.461, P = 0.047), and LDL and muktuk (r = 0.485, P = 0.035). Among younger men, HDL and muktuk were correlated (r = 0.460, P = 0.047). Among older men fresh fish was negatively correlated with HDL (r = -0.082, P = 0.003) and positively correlated with triglycerides (r = 0.843, P = 0.002). None of the seven foods were correlated for all men.

Correlations of nutrients with blood lipids

Blood cholesterol: When blood cholesterol was correlated with fourteen energy adjusted nutrients, no significant correlations were seen among all participants (TABLE 3-4). Among all men generally strong negative correlations were seen for cholesterol with intakes of saturated, monounsaturated, polyunsaturated, total fat, and stearic acid (18:0). Among younger men protein and animal protein were negatively correlated. Among older men saturated, monounsaturated, total fat and stearic acid were negatively correlated. Among all men and younger men, notable positive correlations were observed for carbohydrate, and among older men for protein and iron.

Among women, the only notable correlation was between blood cholesterol and eicosapentaenoic acid (EPA, 20:5, r = 0.346, P = 0.042).

Blood LDL: Correlations of nutrients with LDL were generally similar to those with blood cholesterol with the exception that among all participants stearic acid was negatively correlated with LDL (r = -0.227, P = 0.027) but was not correlated with cholesterol. Among older men correlations of LDL with saturated, monounsaturated fats, protein, and iron were not significant. Among younger men n-3 fatty acids were notably negatively correlated with LDL. Among all men and older men n-6 fatty acids, linoleic acid (18:2) and stearic acid were negatively correlated with LDL. Among older women animal protein was positively correlated with LDL.

Blood HDL: Significant correlations of nutrients with HDL were less common. A positive correlation existed between HDL and total α-tocopherol for the entire sample. Other correlations occurred mainly among women. They included, among older women, positive correlations of HDL with polyunsaturated fats, n-6 fatty acids, linoleic acid and total α-tocopherol. Among all women positive correlations were observed with stearic acid (18:0) and total α-tocopherol. The only negative correlation with HDL was observed among older men with EPA.

Blood triglycerides: The only significant correlation with triglycerides was a positive correlation with carbohydrate among younger women. A similar correlation was seen for carbohydrate using another energy adjustment method (percent of energy from carbohydrate, TABLE 3-3).

Correlations with LDL-HDL ratio

Blood LDL-HDL ratio: All HDL levels exceeded 35 mg/dL (FIGURE 3-1), below which is considered a risk factor for cardiovascular disease (National Cholesterol Education Program, 1994, Feldman, 1994). To consider the high HDL levels among participants (TABLE 3-1), the LDL-HDL ratio was generated and correlated with 23 nutrients (TABLE 3-5) and 91 foods (TABLE 3-6).

Among the entire sample all notable correlations were negative between the LDL-HDL ratio and 18:2, n-6 fatty acids, α-tocopherol, 18:0 and polyunsaturated fats (TABLE 3-5). Among older adults strong positive correlations were observed (with animal protein, EPA, protein, iron and n-3 fatty acids) as well as negative correlations for the same nutrients observed among all participants. Among younger participants there were no significant correlations. Among men, energy, the index of atherogenicity and the index of thrombogenicity were negatively correlated. Among women there were no significant correlations with the LDL-HDL ratio.

Results of correlating the LDL-HDL ratio with 91 foods (TABLE 3-6) from the food frequency revealed correlations of similar magnitude. Most notable were a negative correlation with evaporated milk among young subjects, and a positive correlation with fresh fish among older subjects.

Multiple regression analyses

Because blood lipids are affected by various factors, a combination of factors may best describe cardiovascular risk. Multiple-regression analyses were performed, incorporating the sixteen foods and five nutrients, which were significantly correlated with the LDL-HDL ratio in

bivariate analysis, to identify which dietary factors contribute the most in explaining the variation in blood lipids. Nutrients and foods were evaluated for multicollinearity. Where two or more nutrients were correlated with each other one was selected for multiple regression (TABLE 3-7).

When backward multiple regression analysis was conducted among all subjects using selected foods, age and sex, 36% (adjusted R^2) of the variation in the LDL-HDL ratio was explained by six foods (TABLE 3-8, see footnotes 2 through 6 for foods entered). Foods were removed from the equation when significance of the F test was > 0.10. Variables explaining a significant ($P < 0.10$) proportion of the variation include negative coefficients for fresh bird, tomatoes or tomato juice, evaporated milk, and cheese, and positive coefficients for syrup and pizza. Among younger participants negative coefficients were observed for mukluk, evaporated milk and chicken. Among older participants a positive coefficient was observed for fresh fish. Among men a negative coefficient was observed for chips. Among women positive coefficients were observed for meat stew and pizza.

When multiple regression was applied among all subjects with the LDL-HDL ratio and nutrients, age and sex (TABLE 3-9, footnotes 2 through 6 list nutrients entered), 9% of the variation was explained. Only one variable was significant, α-tocopherol, which had a negative effect. Among younger participants none of the nutrients were significant. Among older participants animal protein had a significant positive coefficient and n-6 fatty acids a negative coefficient ($P = 0.065$). Among men the significant correlation of the index

of atherogenicity (IA)[3] was negative. Among women α-tocopherol had a negative coefficient ($P = 0.058$).

When the nutrients remaining in the regression equation ($P \leq 0.10$) were combined with the initially significant foods and regression analysis performed again (TABLE 3-10), the percent of variation in LDL-HDL ratio among all subjects explained increased to 42%. Seven variables remained in the equation, α-tocopherol, fresh bird, fresh whale, evaporated milk, cheese, syrup, and pizza (age, sex and tomatoes or tomato juice were removed from the equation). Among younger adults mukluk, evaporated milk and chicken explained 39% of the variation (sex was removed). Among older adults 59% of variation was explained by animal protein, n-6 fatty acids and fresh fish (sex, IA, α-tocopherol and dried fish were removed). Among men, 42% of the variation could be explained by the IA and chips (meat stew, age, cheese, and tomatoes or tomato juice were removed). Among women the regression explained 42% of the variation in the blood LDL-HDL ratio with three variables, α-tocopherol, meat stew and hot dogs (pizza, fresh bird, non-dairy creamer were removed).

When body-mass index and smoking were included in the regression analyses of LDL-HDL ratio with foods and nutrients among all participants, body-mass index increased the proportion of the variation in the LDL-HDL ratio explained from 42% to 59% (TABLE 3-11). By itself BMI explained 16% of the variation. It had a positive influence on LDL-HDL ratio. Smoking explained no significant variation

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[3] *Index of Atherogenicity (aS'+bS" + cS"'/ dP +eM +fM') where S' = C12:0, S" = C14:0 and S"' = C16:0; P = sum of n-6 and n-3 PUFA; M = oleic acid (C18:1); and M' = sum of other MUFA; a-f are empirical constants; b = 4 because myristic acid has about 4 times the cholesterol-raising potential of palmitic acid; others are unity lacking evidence for any other value (Ulbricht and Southgate, 1991).*

in the equation or by itself. Among younger participants the BMI was most significant in explaining the variation in LDL-HDL ratio, but among older participants it was not a factor. Among men BMI was not a factor, however among women it was highly significant ($P = 0.001$) and altered which foods remained in the equation. Pizza and fresh bird remained while meat stew and hot dogs were no longer significant ($P < 0.10$).

DISCUSSION

The intent of this analysis was to determine what dietary factors best explain the variation in serum lipids. Such correlations may be low as factors other than diet also play a role. Specifically, LDL can be elevated by obesity, diabetes, hypothyroidism, and familial hyperlipidemia, in addition to dietary saturated fat and cholesterol (Mahan and Arlin, 1992). Estrogen decreases LDL. Hypertension and smoking also are associated with high LDL (McNamara, 1990).

Blood lipids

HDL is higher in females than in males beginning at puberty. Racial differences have been described with blacks having slightly higher HDL levels than whites, a factor that may be involved in the lesser incidence of CHD among this group (See Levy 1986, Mahan and Arlin, 1992). HDL levels also increase with exercise, loss of excess weight and modest consumption of alcohol (Wood et al., 1988, Levy, 1986). HDL is lowered by obesity, lack of exercise, heavy smoking, androgenic steroids, β-adrenergic blocking agents, hypertriglyceridemia, and genetic factors (McNamara, 1990). Such factors need to be examined in conjunction with dietary variables to define their relative contributions to blood

lipid levels and ultimately to cardiovascular disease. The discussion in this paper however, primarily addresses dietary variables as they relate to blood lipids, although the potential influence of body weight (as measured by the body-mass index) and smoking were also considered.

The blood lipid levels of Siberian Yup'iks were unusual when compared with reference values derived from white North American populations (Rifkind and Segal, 1983). None of the participants fell below 35 mg/dL HDL, the threshold level for this risk factor. The Siberian Yup'ik man with the lowest value was 36 mg/dL which is above the 10th percentile for white males (30 mg/dL). The lowest HDL value for a Yup'ik woman was 40 mg/dL, the same as the 10th percentile for white females. When the blood lipids of Siberian Yup'ik men and women (divided by ten year age increments) were compared with NHANES III participants (Johnson et al., 1993), the HDL levels of the Siberian Yup'iks were generally higher and the triglycerides generally lower. While total cholesterol and LDL tended to be higher among Siberian Yup'ik men than among NHANES III men, large standard deviations and small numbers among Siberian Yup'iks make significant differences unlikely. Total cholesterol and LDL levels of Siberian Yup'ik women compared with NHANES III women were also similar. Since total cholesterol is a function of both HDL and LDL, potentially higher total cholesterol levels may be explained by the higher levels of HDL. HDL normally accounts for <25% of the total plasma cholesterol (Levy, 1986), but among Siberian Yup'ik men HDL is 26% and among women 28%. It is important to assess the components of total cholesterol as well as their relationship to each other in defining an outcome against which to measure diet variables. It is also worth noting

that if blood levels are already high, the narrow distribution may make correlations difficult.

Hypotheses

Based on research findings in the literature and national recommendations on the role of diet and heart disease, I postulated six hypotheses to be tested by this data set. In brief, four of the six hypotheses were rejected (numbers 2,4,5 and 6). There were no significant positive correlations between saturated fat intake with cholesterol, LDL or triglycerides and no significant negative correlations between saturated fat and HDL, as hypothesized. There were no significant correlations between monounsaturated fatty acid intakes and HDL where a positive correlation was expected. N-3 fatty acid intakes were not correlated with high HDL, as expected, and the expected negative correlation between seal oil or fish consumption and blood glucose level was not found.

Support was found for hypothesis three; a negative correlation between monounsaturated fatty acid intake and LDL was observed for all and for men as well as a negative correlation with cholesterol among men. Partial support was found for hypothesis 1) negative correlations between the SMFI and LDL and SMFI and LDL-HDL ratio were seen only among young men. However, I found no significant negative correlations between SMFI and total cholesterol, or SMFI and triglyceride level in any of the groups.

Some of the discrepancies between the six hypotheses and the results may be explained by hypotheses being based on differences reported between populations while the differences observed here are between individuals in the same population. Cross-sectional studies of dietary lipids

and blood cholesterol are not optimal to define the effects of dietary factors on blood lipids. These factors are best studied by controlled, preferably blinded, feeding trials in 10-50 human subjects (See Willett, 1990). Further, correlation of dietary fat intake with a single blood measurement of total cholesterol within a general US population is low because controlled metabolic studies indicate that most of the between-person variation in cholesterol is unrelated to dietary fats. In addition, a very large sample size would be needed to detect a significant positive correlation between diet and blood cholesterol.

The most interesting findings are the lack of correlations expected as shown by tests of the six hypotheses. High intakes of saturated fat and dietary cholesterol are reported to result in high levels of LDL (Mahan and Arlin 1992). Nonetheless, in this study saturated fat was not significantly correlated with LDL except for the negative correlation (r = -0.443, P = 0.016, TABLE 3-4) with all men. In addition, the significant associations between LDL and dietary cholesterol (r = 0.252 P = 0.045 for all, r = -0.414, P = 0.026 for men, r = -0.473,, P = 0.041 for young men) disappeared after cholesterol intake was adjusted for energy. One explanation may be that men who are active have a high saturated fat and cholesterol intake. But their activity or some unidentified variable may play a more important role in blood cholesterol and LDL levels than their intake of saturated fat. The disappearance of significance after energy adjustment indicates a contribution from energy to the LDL-HDL ratio.
Another explanation is that the high LDL mean levels for men (149 mg/dL) and women (161 mg/dL) narrow the range over

which a positive correlation could occur.

Finding substantial negative correlations between intakes of total, saturated, monounsaturated and polyunsaturated fat with blood cholesterol for men in my data was also surprising. The investigations of fat intake among populations of countries positively correlates fat intake with increased cardiovascular mortality, presumably resulting from increased blood cholesterol as described in the diet heart hypothesis. My results showed energy was also negatively correlated with blood cholesterol, again indicating other factors may be interacting to explain the negative correlations, such as increased physical activity. High levels of exercise could be associated with increased dietary energy intake, increased HDL, decreased triglycerides and have no affect on total cholesterol and LDL (Mahan and Arlin, 1992).

Similar to my findings, intake of fat-containing foods assessed by a simple frequency questionnaire in the Tecumseh Heart Study (Nichols et al., 1976) was not positively associated with serum cholesterol or triglycerides among men and women. They found correlations of dietary fat with serum cholesterol among 4,057 adults were -0.02 for women and -0.11 for men. Correlations of fat with triglycerides were 0.02 for women and -0.05 for men. Nichols et al.(1976) concluded based on their findings and those of others in four large epidemiological studies, that diet-lipid relationships that are evident in comparisons of different populations apparently do not apply to individuals within a given population thus providing evidence that factors other than fat (e.g. degree of adiposity) are greater determinants.

The Tecumseh Study (Nichols et al., 1976) is sometimes cited as evidence against the diet-heart hypothesis. The hypothesis states that high intake of saturated fats and cholesterol and low intake of polyunsaturated fats increase the level of serum cholesterol, which leads to development of atheromatous plaques, artery narrowing, reduced blood flow and finally myocardial infarction (Chapter 1). On the other hand, Shekelle et al.(1981) found a positive correlation between dietary fat intake and serum total cholesterol among nearly 2,000 men in the Western Electric Study, although it was low (r = 0.08, P = 0.006).

Considering the numerous variables that affect LDL and HDL levels the large amount of variation explained by regression of the food and nutrient variables in my investigation was surprising. The large variance attributable to overweight as measured by the BMI is more consistent with at least one previous study (Nichols et al., 1976) in which investigators attributed changes in blood cholesterol and triglycerides to obesity more than to the dietary components, fat, sugar, starch or alcohol. In my study, attributing changes of blood lipids to the foods I identified, is not readily explained by physiologic mechanisms.

Multiple regression analyses with LDL-HDL ratio as dependent variable

Multiple regression analysis showed that food types, dietary nutrient intake and BMI can help to explain the blood LDL-HDL ratio. Among women the negative contribution to the equation of α-tocopherol plus the positive contributions of hot dogs and meat stew increase the proportion of variability which was explained. These foods,

however, were replaced with pizza (positive coefficient) and fresh bird (negative association) when BMI was considered. Among men, while not expected, IA and chips both made negative contributions to the equation to explain LDL-HDL ratio. Among older subjects animal protein and fresh fish made positive contributions while n-6 fatty acids made a negative contribution to the equation. Among younger subjects mukluk, evaporated milk and chicken made negative contributions to the equation, but again when the BMI was added to the equation it made a positive contribution of comparable significance to the negative contributions of chicken and evaporated milk.

In the last regression analysis conducted in this study (TABLE 3-11), among all participants seven variables contributed to the blood LDL-HDL ratio of which four were negatively correlated. The negative association of α-tocopherol with the LDL-HDL ratio may be related to the fact that α-tocopherol is highly correlated with polyunsaturated fats which are known to lower LDL and were negatively correlated with LDL among men in this study (TABLE 3-4). In the correlation analyses those factors associated with lipids tend to be negatively correlated while those with protein have a positive correlation (TABLE 3-5).

Fresh bird, fresh whale and evaporated milk were negatively correlated with the LDL-HDL ratio. These data support the healthy aspects of a more traditional eating pattern, particularly when a more traditional lifestyle includes fewer purchased foods (potentially higher in saturated fat), and the physical activity of hunting, harvesting and preparing wild birds and whale. Conversely, if individuals with high consumption of pizza and syrup have a less active lifestyle, then the higher carbohydrate

consumption has the deleterious effect of decreasing HDL levels. Although my data showed no significant correlations with HDL, positive correlations between carbohydrate and LDL for younger men (r = 0.535, P = 0.018) and for all men (r = 0.419, P = 0.024) and between carbohydrate and blood cholesterol for younger men (r = 0.526, P = 0.021) and for all men (r = 0.448, P = 0.015) were found (TABLE 3-4). These correlations were not significant prior to energy adjustment (TABLE 3-3).

In any list of more than a few variables, one might expect a correlation to appear by chance when P = 0.05. This explanation may be applied to each of the variables, although some can be explained by other plausible mechanisms. The correlations for cheese and pizza (TABLE 3-6) are examples of possible chance occurrences as they have opposite signs, and explanations based on physiologic mechanisms are not readily apparent. Possible explanations for some of the other correlations follow.

Evaporated milk is used in coffee and tea which are consumed at least daily by all but a few participants. Six or more cups per day is not uncommon. Lower LDL-HDL levels were found for those who consumed evaporated milk (FIGURES 3-2, 3-3). In contrast a positive correlation with LDL-HDL was found among women who consumed non-dairy creamer, an alternate to evaporated milk in coffee and tea. My explanation for this contrast in coffee-tea additives is that the negative effect of non-dairy creamer is due to its tropical oil content (such as coconut, palm, and palm kernel) which are high in saturated fat. Coconut oil is high in palmitic acid (C 16:0) which is hypercholesterolemic (Mahan and Arlin, 1992).

Among younger adults a traditional food, mukluk, was negatively correlated with the LDL-HDL ratio as was evaporated milk and chicken (TABLE 3-8). Chicken is lower in saturated fat and higher in polyunsaturated fat than beef, another western food alternative. Thus the lipid composition of chicken should contribute to the negative correlation with the LDL-HDL ratio as would be expected for mukluk.

Among older adults, the positive correlations of LDL-HDL ratio with both fish and animal protein is surprising and at first glance disturbing as fish is usually thought to be protective from cardiovascular disease. However, the mechanism by which fish protects involves the antithrombotic attributes of n-3 fatty acids found in fish (Dyerberg et al., 1978) rather than the atherogenic mechanisms associated with blood lipids which are examined in this paper.

The negative regression of n-6 fatty acids with the LDL-HDL ratio (TABLE 3-10), is explained by the LDL lowering effect of n-6 polyunsaturated fatty acids found in vegetable seeds and their oils in western diets. The overall positive regression of LDL-HDL ratio with fish and animal protein intake among older men and women is similar to the hypercholesterolemic correlation of animal protein described by Yerushalmy and Hilleboe (1957).

In most reviews of dietary studies related to cardiovascular disease (Feldman 1994, McNamara, 1994, Marmot and Elliott, 1992) and in one study in Alaska (Feldman, 1972) little attention is given to protein intake because it is usually consumed with fat and the bulk of investigations have emphasized dietary fat. However, Yudkin (1957) and Yerushalmy and Hilleboe(1957) showed that animal protein was better correlated than the intake of fat with human coronary disease in multipopulation studies (Kritchevsky, 1983). When

comparing data from 15 countries on correlations of fat, protein, energy and sugar with coronary thrombosis, Yudkin (1957) concluded that coronary thrombosis is associated with higher living standards. Yet participants within my study population have living standards similar to each other and protein was still positively correlated. There is reason for concern about protein intake in this population as their intake is high. Siberian Yup'ik men and women consume more protein than NHANES III men and women respectively (TABLE 2-8). There exists the possibility that the positive regression coefficient for fresh fish more strongly reflects protein intake to the detriment of protective fats in fish. Resolving this question requires a dependent variable which measures both atherogenicity and thrombogenicity, i.e. cardiovascular disease itself.

A negative partial regression coefficient for the index of atherogenicity (IA) with LDL-HDL ratio among men (TABLE 3-10) was unexpected as the IA (Ulbricht and Southgate, 1991) combines several dietary components known to be associated with the development of atherosclerosis (TABLES 3-9, 3-10). The significant negative relationship of chips, such as potato chips, with the LDL-HDL ratio is also difficult to explain. The combination of the IA and chips explains 42% of the variance. I thought men who eat chips as they consume alcohol may be protected by an increase in HDL associated with moderate alcohol (Feinman and Lieber, 1994) but chips were not significantly correlated with beer consumption (r = 0.356 P = 0.096) among the 23 men who consumed both. Potato chips are neutral to positive with respect to cardiovascular disease as one ounce of potato chips contains 1.2 mg α-tocopherol which is protective (TABLES 3-9, 3-10) and they contain more polyunsaturated

than saturated fats (3.6 g vs. 1.8/20 g or 10 chips) as well as monounsaturated fats (1.2 g) (Mahan and Arlin, 1992), thus supporting the finding of protection from α-tocopherol and polyunsaturated fat.

Among women, the major foods that influenced the LDL-HDL ratio in the multiple regression were the positive associations with stew (made from mostly meat and vegetables) and hot dogs (TABLE 3-10), which suggests that eating western foods leads to higher LDL-HDL ratios. With the incorporation of BMI in the equation (TABLE 3-11), the foods remaining in the equation were replaced with pizza and fresh bird. Again the western food (pizza) was positively correlated but the Native food (fresh bird) was negatively correlated.

Although sex and age did not remain in any of the multiple regression analyses, there were differences in significant variables between the sexes and between older and younger adults. (Some notable examples are the negative effect of IA among men which explained 22% of the variation (TABLE 3-9) but was not a factor for women, while among women the negative effect of α-tocopherol explained 8% of the variation but was not a factor among men. The positive contribution of animal protein and the negative contribution of n-6 fatty acids which accounted for 52% of the variation among older adults, did not reach significance ($P < 0.10$) among younger adults. There are several possible explanations for differences. While most of the risk factors for coronary heart disease and the strategies for preventing disease among men are also important among women, the magnitude of their effects may differ (Rich-Edwards, 1995). A risk factor unique to women is menopause. Dietary intakes also differ based on age and sex. Younger adults and men

generally consume more than older adults and women (McDowell, 1994, Alaimo, 1994) as confirmed for this Yup'ik population where young men consumed more energy than women and older men (Chapter 2, TABLE 2-8).

There are several negative correlations of LDL-HDL ratio with Native foods (TABLE 3-6). They make a significant contribution to lower LDL-HDL ratios with other foods in the multiple regression analysis, particularly for young adults (TABLES 3-8, 3-9). More positive correlations were noted with nontraditional foods among all subjects and women (TABLE 3-6) and some (e.g. syrup and pizza) were significant influences in the multiple regression (TABLES 3-8, 3-10). However, among older adults and men, the opposite is true, traditional foods (fresh and dried fish) are positively correlated (TABLE 3-6). I suggest evaporated milk could be considered a traditional food, on the basis of its ready availability in Alaska for at least the last 50 years.

Correlations of foods high in *trans*-fatty acids

Trans-fatty acids have been implicated in promoting heart disease in a case control study (Ascherio et al., 1994) and other investigations, as they raise the LDL and lower the HDL levels. Foods identified as sources of *trans*-fatty acids include some natural sources, dairy products and beef (Willett, 1990), and sources associated with hydrogenated vegetable fats, margarines, white bread, cakes and cookies (Ascherio and Willett, 1995). None of these foods, except cheese and evaporated milk were individually correlated with the LDL-HDL ratio (TABLE 3-6). Both were negatively correlated, when a positive correlation might be expected. Even the combined intake of margarine, white bread, cake, cookies and beef (log transformed) was not

significantly correlated with the LDL-HDL ratio. One unknown is the *trans*-fatty acid content of pizza. If it is high then the positive correlation of pizza with the LDL-HDL ratio among all subjects and among women might be explained by the *trans*-fatty acid hypothesis.

Limitations

In general, most of the correlations between blood levels and LDL-HDL ratio and food types and nutrient intakes (TABLES 3-2, 3-3, 3-6) are considered to be weak ($r < 0.5$), although the probability of them occurring by chance was $P < 0.05$ and, in several cases $P < 0.01$. The Framingham Study (1970) concluded that the only discernible association between diet factors they investigated among 912 men and women, and their serum cholesterol was a weak negative association between caloric intake and serum cholesterol level in men (-0.20). Stronger relationships may be seen in my study because the population was more genetically homogeneous and the diet intakes were more variable due to consumption of both traditional and commercial foods.

A very important qualification of my interpretation of these data is inter-correlations between nutrients. Multicollinearity is best addressed in observational studies such as this by choosing regressors for analysis that have as little intercorrelation as possible (Wonnacott and Wonnacott, 1990). Although I controlled for energy intake, other nutrients were significantly correlated. For example, among all subjects n-6 fatty acids were inter-correlated with polyunsaturated fatty acids, 18:0, 18:2 and α-tocopherol ($r > 0.7$, < -0.7 TABLE 3-7). Therefore conclusions about which is the most important variable, using multiple regression, are difficult. Only one variable

from this list could be selected for inclusion. While α-tocopherol explained most variation in LDL-HDL ratios among all participants (TABLES 3-9, 3-10), it may be a surrogate for another correlated variable such as polyunsaturated fatty acid, n-6 fatty acids or linoleic (18:2) acid. Likewise, among older adults, n-6 fatty acids may be a surrogate for α-tocopherol, polyunsaturated fatty acids or linoleic acid.

I did not adjust for the probability that some correlations will be significant by chance alone, because the variables are not independent of each other. For example fat and fatty acids will move in the same direction when correlated with another measure. Establishing the significance level at 0.05 permitted identification of potential associations which a more strict significance level would not detect. Given the descriptive nature of the investigation and the small population the 0.05 level identified a greater number of potential associations. Because the critical value for α in the simple correlations was set at 0.05 rather than a lower value, the correlations lose some impact and outcomes with a high degree of significance cannot be inferred.

Fewer significant correlations were observed once nutrient intake was adjusted for variation in energy intake especially for young men. This supports the argument that in epidemiologic analyses a measure of nutrient intake should be used that is independent of total caloric intake (Willett, 1990). After adjusting for energy the negative correlation of cholesterol with iron intake among younger men disappeared, while that among older men became positive and significant. Among older women, adjusting for energy intake revealed a significant correlation between HDL and

linoleic acid intake.

From the above discussion, it is clear that drawing firm conclusions about the relationship between diet and blood lipids that affect cardiovascular disease from this sample of 64 adults, should be done with extreme caution. The sample size is small which limits the fractional treatment of factors other than sex and age. For example because women as a group had higher HDL levels than men in this study (TABLE 3-1) and elsewhere (Rifkind and Segal, 1983) the explanation of the differences within each sex is not possible due to my inability to subdivide the many nutrients which are too correlated with each other.

Dietary choice and food use were assumed to represent those of a healthy population. To verify to what extent people were modifying their diet, we asked participants if they altered their eating practices for any reason. Only four participants identified heart disease as a health problem that affected their diet. Of the three older women and one older man, one avoided salt and sweets, one ate mostly traditional foods when short on money, one avoided caffeine, and one avoided fat.

Strengths

The data in this study provide an estimate of diet intakes and their relationship to blood lipids among Gambell adults. Although the absolute sample size is small, it represented over 50% of the adult population of Gambell. Therefore, these findings, along with published information about diet and cardiovascular disease, are available to formulate dietary recommendations for Siberian Yup'iks. While I did not measure life style except by type of foods consumed, the life style associated with acquisition,

processing and consumption of traditional foods may be an important factor in cardiovascular health. Based on this investigation I cannot reject the hypothesis that the life style associated with traditional food consumption is desirable, since it is generally consistent with lower LDL-HDL ratios, a strong predictor for cardiovascular disease risk.

CONCLUSION

This investigation explored the dietary factors associated with blood lipids. Mean blood cholesterol and LDL levels of Siberian Yup'iks were above thresholds for treatment and low HDL levels were not observed. Triglyceride levels were also low. The unusual nature of the diet and the blood lipids provided the opportunity to explore their unique relationships. The findings indicate that a variety of foods and nutrients may be contributing to variations in the blood lipids of Siberian Yup'iks.

While 42% of the variation in the LDL-HDL ratio can be attributed to the selected dietary factors, the effects of specific nutrients cannot be neatly separated. While α-tocopherol, animal protein, n-6 fatty acids and the index of atherogenicity were identified as variables significantly correlated with LDL-HDL ratios, among at least a portion of the participants, the correlation of each with other nutrients makes definitive assignment of nutrient equivocal. Nevertheless, the analysis supports the recommendation to emphasize intake of foods rich in α-tocopherol, n-6 fatty acids and polyunsaturated fats.

In the final regression equations for older, younger, men, women and all participants, eleven foods remained, of which seven were significant ($P < 0.05$) in explaining LDL-

HDL ratio variations. The foods may be grouped as traditional (fresh bird, fresh fish and evaporated milk) and non-traditional foods (syrup, pizza, chicken, chips). Consumption of traditional foods, while not uniform for all groups in the analyses, was generally consistent with lower LDL-HDL ratios. Conversely, non-traditional foods tended to be positively correlated with the LDL-HDL ratio.

While the presence or absence of smoking was not a factor in explaining the LDL-HDL ratio variation, the inclusion of a measure of obesity was highly significant. This was true among all participants, among younger participants and among women. The positive influence of obesity on the LDL-HDL ratio make it an important variable in improving the LDL-HDL ratios.

To increase our understanding of the role of foods in altering blood lipids this investigation needs to be replicated. If subsequent results are consistent, and if dietary practices change towards a more western style, blood LDL values could increase and HDL lipid values decrease resulting in higher LDL-HDL ratios and potentially more cardiovascular disease.

TABLE 3-1 Comparison of mean blood lipid levels of Siberian Yup'iks (±SEM) with thresholds for increased cardiovascular disease risk†, October 1992, Gambell, Alaska.

Blood lipid Age	Men[1] Mean±SEM	Women[2] Mean±SEM	Threshold
LDL-HDL ratio			
40-59 y[3]	3.0± 0.3	2.7± 0.3	>3.5[4]
≥ 60 y[5]	2.3± 0.3	2.4± 0.2	
Both	2.8± 0.2	2.6± 0.2	
Cholesterol, mg/dL			
40-59 y	236 ± 9	251 ±14	>200[6]
≥ 60 y*	198 ±13	235 ± 8	
Both	223 ± 8	243 ± 8	
LDL			
40-59 y	161 ± 8.9	168 ±13.7	>130[7]
≥ 60 y	127 ± 13	154 ± 7.1	
Both	149 ± 7.9	161 ± 7.9	
HDL mg/dl			
40-59 y	58 ± 3.5	67 ± 3.7	<35[8]
≥ 60 y*	58 ± 4.1	68 ± 4.3	
Both	58 ± 2.6	67 ± 2.8	
Triglycerides mg/dl			
40-59 y	85 ± 8.3	79 ±12.6	>200[9]
≥ 60 y	63 ± 9.6	67 ± 6.4	
Both	77 ± 6.5	73 ± 7.3	

†Ebbesson et al.(in preparation) show more detailed comparisons of age and sex differences.

*$P < 0.05$ differences between sexes.

[1]n = 29 [2]n = 36 [3]n = 19 younger men, 19 younger women.

[4]McNamara 1990. Diet therapy to reduce CHD is indicated at > 3.5.

[5]*n* = 10 older men, 17 older women.

[6] National Cholesterol Education Program (1994) Treatment guidelines recommend: if blood cholesterol is 200-239 mg/dL and HDL is ≥ 35 mg/dL and there are < two other risk factors, instruction in diet modification, exercise and other risk reduction. If HDL is < 35 mg/dL, lipoprotein analysis is recommended. If cholesterol is ≥ 240 mg/dL, lipoprotein analysis is recommended.

[7] Ibid. If LDL is 130-159 mg/dL and ≥ two risks, diet and physical activity modification are recommended. If LDL ≥ 160 mg/dL, and < two risks, initiate appropriate diet therapy.

[8]Ibid. HDL < 35 mg/dl is considered a risk factor for coronary heart disease and patients should have a lipoprotein analysis.

[9] Ibid. Levels below 200 mg/dl are considered normal. Weight reduction, alcohol restriction and increased physical activity are recommended for all patients with elevated triglycerides.

TABLE 3-2

Pearson bivariate correlation coefficients between blood lipids and Sea Mammal Fish Index (SMFI)[1] of consumption among Siberian Yup'iks, 1992, Gambell, Alaska.

Age,Sex groups	*n*	Cholesterol		LDL		HDL		Triglycerides	
		r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
All	64	0.018	0.886	0.077	0.547	-0.062	0.627	-0.000	0.998
Men	29	-0.160	0.406	-0.133	0.491	0.056	0.773	0.123	0.527
Younger	19	-0.438	0.061	-0.483	0.036*	0.351	0.141	-0.170	0.486
Older	10	-0.047	0.898	0.152	0.676	-0.877	0.001**	0.653	0.041*

TABLE 3-2 (continued)

Women	35	0.322	0.059	0.400	0.017*	-0.063	0.721	-0.175	0.315
Younger	19	0.332	0.165	0.314	0.190	0.184	0.450	-0.201	0.410
Older	16	0.344	0.193	0.595	0.015*	-0.337	0.202	-0.138	0.611
All young	38	-0.079	0.637	-0.094	0.576	0.177	0.287	-0.127	0.447
All old	26	0.119	0.563	0.321	0.110	-0.507	0.008**	0.204	0.318

[1]All values were log transformed.

Sea Mammal Fish Index (SMFI) = Sum of servings per year (Fresh Fish, Walrus, Muktuk [outer skin and attached blubber of whale], Mukluk [Bearded seal], Dry Fish, Dry Seal, Seal Oil) / 365.2.

*$P < 0.05$.

**$P < 0.01$.

TABLE 3-2 (continued)

Age,Sex	*n*	LDL-HDL ratio	
		r	*P*
All	64	0.092	0.468
Men	29	-0.123	0.524
Younger	19	-0.510	0.026*
Older	10	0.556	0.095
Women	35	0.316	0.064
Younger	19	0.124	0.614
Older	16	0.625	0.010*
All young	38	-0.173	0.299
All old	26	0.583	0.002**

TABLE 3-3

Bivariate correlation coefficients between blood lipids and nutrient intakes[1,2] (unadjusted for energy) among Siberian Yup'iks, 1992, Gambell, Alaska.

Nutrients	*n*	Blood Lipids: Cholesterol *r*	Cholesterol *P*	LDL *r*	LDL *P*	HDL *r*	HDL *P*	Triglycerides *r*	Triglycerides *P*
Saturated Fats, g									
All	64	-0.290	0.020*	-0.281	0.025*	-0.058	0.650	-0.019	0.882
Men	29	-0.590	0.001*	-0.602	0.001*	0.074	0.702	-0.051	0.793
younger	19	-0.480	0.038*	-0.533	0.019*	0.146	0.551	0.045	0.857
older	10	-0.848	0.002*	-0.785	0.007*	-0.150	0.680	-0.129	0.722
Women	35	0.162	0.352	0.176	0.312	0.000	0.99	-0.048	0.783
younger	19	0.086	0.726	0.052	0.833	0.172	0.48	-0.120	0.626
older	16	0.265	0.321	0.381	0.145	-0.192	0.476	0.027	0.923
Monounsaturated fats, g									
All	64	-0.241	0.055	-0.252	0.045*	-0.016	0.899	-0.001	0.992
Men	29	-0.539	0.003*	-0.592	0.001*	0.164	0.395	0.031	0.872
younger	19	-0.510	0.026*	-0.601	0.006*	0.253	0.295	0.048	0.844
older	10	-0.747	0.013*	-0.739	0.015*	-0.129	0.723	-0.080	0.825
Women	35	0.208	0.232	0.224	0.196	-0.008	0.962	-0.098	0.574

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*
younger	19	0.198	0.417
older	16	0.200	0.457
Polyunsaturated fats, g			
All	64	-0.236	0.061
Men	29	-0.514	0.004*
younger	19	-0.508	0.026*
older	10	-0.704	0.023*
Women	35	0.193	0.266
younger	19	0.165	0.501
older	16	0.222	0.409
n-3 fatty acids, g			
All	64	-0.157	0.217
Men	29	-0.433	0.019*
younger	19	-0.574	0.010*
older	10	-0.468	0.172
Women	35	0.299	0.081
younger	19	0.357	0.133
older	16	0.215	0.425

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
0.169	0.489	0.152	0.535	-0.138	0.573
0.308	0.246	-0.180	0.504	-0.076	0.780
-0.266	0.034*	0.019	0.882	-0.013	0.922
-0.586	0.001*	0.194	0.314	0.019	0.923
-0.610	0.006*	0.260	0.283	0.020	0.935
-0.736	0.015*	0.005	0.989	0.040	0.912
0.204	0.241	-0.010	0.955	-0.102	0.561
0.144	0.557	0.047	0.849	-0.049	0.843
0.296	0.265	-0.067	0.804	-0.236	0.380
-0.133	0.296	-0.059	0.642	0.025	0.847
-0.453	0.014*	0.124	0.523	0.078	0.688
-0.648	0.003*	0.305	0.204	-0.049	0.842
-0.376	0.284	-0.482	0.158	0.362	0.305
0.355	0.036*	-0.068	0.696	-0.095	0.586
0.329	0.170	0.142	0.561	-0.091	0.712
0.424	0.102	-0.289	0.278	-0.121	0.656

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	Cholesterol *P*
n-6 fatty acids, g			
All	64	-0.246	0.050
Men	29	-0.499	0.006*
younger	19	-0.426	0.069
older	10	-0.736	0.015*
Women	35	0.145	0.406
younger	19	0.072	0.768
older	16	0.237	0.376
Energy			
All	64	-0.208	0.099
Men	29	-0.419	0.024*
younger	19	-0.431	0.065
older	10	-0.670	0.034*
Women	35	0.153	0.380
younger	19	0.149	0.542
older	16	0.125	0.644
Fat, g			
All	64	-0.262	0.037
Men	29	-0.564	0.001*

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
-0.294	0.018*	0.064	0.615	-0.057	0.653
-0.575	0.001*	0.196	0.309	-0.050	0.798
-0.518	0.023*	0.201	0.409	0.007	0.979
-0.798	0.006*	0.176	0.626	-0.121	0.740
0.133	0.447	0.038	0.830	-0.110	0.528
0.056	0.819	0.008	0.973	-0.030	0.903
0.238	0.374	0.081	0.764	-0.304	0.252
-0.199	0.11	-0.079	0.537	0.042	0.743
-0.477	0.009	0.179	0.352	0.009	0.965
-0.517	0.023	0.271	0.262	-0.069	0.778
-0.680	0.030*	-0.076	0.834	0.116	0.749
0.194	0.265	-0.108	0.538	0.012	0.945
0.131	0.594	0.022	0.928	0.085	0.730
0.305	0.250	-0.277	0.300	-0.167	0.536
-0.267	0.033	-0.030	0.815	-0.008	0.948
-0.606	0.000*	0.139	0.472	0.002	0.992

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol		
		r	*P*	*r*
younger	19	-0.514	0.024*	-0.594
older	10	-0.790	0.007*	-0.769
Women	35	0.195	0.262	0.211
younger	19	0.162	0.507	0.133
older	16	0.226	0.401	0.334
Protein, g				
All	64	-0.182	0.151	-0.160
Men	29	-0.380	0.042*	-0.428
younger	19	-0.537	0.018*	-0.602
older	10	-0.459	0.182	-0.480
Women	35	0.196	0.260	0.269
younger	19	0.205	0.401	0.174
older	16	0.194	0.473	0.486
Carbohydrate, g				
All	64	-0.141	0.267	-0.133
Men	29	-0.244	0.202	-0.316
younger	19	-0.208	0.393	-0.296
older	10	-0.561	0.092	-0.608
Women	35	0.086	0.623	0.120

LDL		HDL	Triglycerides	
P	*r*	*P*	*r*	*P*
0.007*	0.221	0.362	0.038	0.877
0.009*	-0.120	0.741	0.009	0.980
0.224	-0.008	0.964	-0.084	0.633
0.587	0.143	0.560	-0.118	0.630
0.206	-0.171	0.526	-0.071	0.795
0.208	-0.032	0.803	-0.026	0.839
0.021*	0.212	0.269	0.009	0.963
0.006*	0.299	0.213	-0.121	0.623
0.160	-0.159	0.662	0.358	0.310
0.118	-0.076	0.664	-0.142	0.415
0.477	0.209	0.390	-0.163	0.504
0.056	-0.441	0.087	-0.103	0.705
0.294	-0.110	0.385	0.108	0.397
0.095	0.165	0.392	0.325	0.867
0.219	0.226	0.353	-0.074	0.765
0.062	0.030	0.934	0.130	0.721
0.493	-0.148	0.395	0.126	0.471

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*	*r*
younger	19	0.110	0.653	0.106
older	16	-0.007	0.980	0.141
PS ratio[3]				
All	64	0.173	0.172	0.089
Men	29	0.213	0.266	0.090
younger	19	-0.024	0.921	-0.138
older	10	0.279	0.435	0.113
Women	35	0.030	0.865	0.019
younger	19	0.140	0.568	0.176
older	16	-0.164	0.545	-0.292
Percent energy from fat				
All	64	-0.184	0.145	-0.221
Men	29	-0.506	0.005*	-0.486
younger	19	-0.354	0.137	-0.371
older	10	-0.802	0.005*	-0.671
Women	35	0.123	0.483	0.065
younger	19	0.021	0.933	-0.001
older	16	0.372	0.156	0.215

LDL		HDL	Triglycerides	
P	*r*	*P*	*r*	*P*
0.665	-0.120	0.625	0.285	0.237
0.602	-0.205	0.445	-0.304	0.252
0.486	0.176	0.164	0.018	0.891
0.641	0.242	0.205	0.150	0.438
0.572	0.263	0.278	-0.064	0.795
0.757	0.269	0.453	0.294	0.410
0.915	-0.023	0.898	-0.107	0.540
0.471	-0.299	0.214	0.174	0.475
0.273	0.354	0.179	-0.666	0.005*
0.080	0.113	0.376	-0.123	0.331
0.008*	-0.039	0.840	-0.014	0.944
0.118	-0.026	0.917	0.239	0.325
0.034*	-0.217	0.547	-0.396	0.257
0.711	0.238	0.168	-0.239	0.167
0.996	0.259	0.283	-0.443	0.057
0.424	0.227	0.397	0.244	0.363

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol		
		r	*P*	*r*
Percent energy from saturated fats				
All	64	-0.266	0.034*	-0.264
Men	29	-0.565	0.001*	-0.488
younger	19	-0.323	0.177	-0.280
older	10	-0.711	0.021*	-0.543
Women	35	0.058	0.739	0.005
younger	19	-0.123	0.616	-0.159
older	16	0.414	0.111	0.321
Percent energy from monounsaturated fats				
All	64	-0.126	0.322	-0.172
Men	29	-0.422	0.023*	-0.430
younger	19	-0.329	0.169	-0.374
older	10	-0.661	0.037*	-0.570
Women	35	0.149	0.394	0.099
younger	19	0.093	0.704	0.074
older	16	0.281	0.292	0.147
Percent energy from polyunsaturated fats				
All	64	-0.086	0.502	-0.167
Men	29	-0.331	0.080	-0.379

LDL		HDL		Triglycerides
P	*r*	*P*	*r*	*P*
0.035*	0.024	0.851	-0.133	0.297
0.007*	-0.158	0.412	-0.130	0.502
0.245	-0.189	0.440	0.254	0.294
0.105	-0.203	0.573	-0.519	0.125
0.977	0.228	0.188	-0.139	0.426
0.514	0.333	0.164	-0.440	0.059
0.225	0.105	0.700	0.432	0.095
0.174	0.135	0.287	-0.096	0.451
0.020*	0.025	0.896	0.056	0.772
0.115	0.059	0.810	0.236	0.331
0.085	-0.296	0.407	-0.133	0.714
0.571	0.213	0.219	-0.253	0.142
0.764	0.255	0.292	-0.440	0.060
0.588	0.162	0.549	0.198	0.463
0.186	0.199	0.115	-0.111	0.383
0.043*	0.088	0.650	0.024	0.903

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*	*r*
younger	19	-0.283	0.240	-0.341
older	10	-0.375	0.286	-0.435
Women	35	0.093	0.594	0.025
younger	19	0.011	0.965	0.009
older	16	0.363	0.167	0.063
Percent energy from protein				
All	64	0.038	0.766	0.062
Men	29	0.002	0.994	0.012
younger	19	-0.365	0.124	-0.353
older	10	0.768	0.009*	0.757
Women	35	0.051	0.769	0.095
younger	19	0.061	0.805	0.046
older	16	0.131	0.628	0.346
Percent energy from carbohydrate				
All	64	0.126	0.321	0.124
Men	29	0.418	0.024	0.383
younger	19	0.504	0.028*	0.505
older	10	0.262	0.465	0.074
Women	35	-0.094	0.591	-0.093

LDL		HDL	Triglycerides	
P	*r*	*P*	*r*	*P*
0.153	0.060	0.808	0.155	0.525
0.209	0.172	0.634	-0.140	0.699
0.889	0.222	0.201	-0.259	0.133
0.970	0.043	0.861	-0.260	0.283
0.817	0.618	0.011*	-0.280	0.294
0.627	0.082	0.522	-0.122	0.339
0.951	0.099	0.610	0.003	0.989
0.138	0.151	0.537	-0.130	0.596
0.011*	-0.088	0.810	0.326	0.358
0.586	0.050	0.774	-0.218	0.208
0.851	0.233	0.336	-0.317	0.186
0.189	-0.316	0.233	0.127	0.638
0.329	-0.100	0.432	0.179	0.157
0.040*	-0.031	0.872	0.059	0.762
0.027*	-0.115	0.641	-0.005	0.984
0.840	0.497	0.144	0.116	0.750
0.596	-0.142	0.416	0.275	0.110

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*
younger	19	-0.004	0.986
older	16	-0.368	0.161
Stearic acid 18:0, g			
All	64	-0.293	0.019*
Men	29	-0.563	0.001*
younger	19	-0.428	0.068
older	10	-0.862	0.001*
Women	35	0.134	0.442
younger	19	0.031	0.899
older	16	0.281	0.293
Sodium, mg			
All	64	-0.218	0.083
Men	29	-0.470	0.010*
younger	19	-0.440	0.060
older	10	-0.664	0.036*
Women	35	0.196	0.259
younger	19	0.197	0.419
older	16	0.165	0.541

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
0.018	0.941	-0.308	0.199	0.499	0.030*
-0.447	0.083	0.184	0.496	-0.403	0.122
-0.307	0.014*	-0.006	0.962	-0.041	0.751
-0.589	0.001*	0.088	0.651	-0.050	0.797
-0.483	0.036*	0.127	0.604	0.052	0.831
-0.838	0.002*	-0.048	0.895	-0.141	0.697
0.124	0.480	0.064	0.715	-0.088	0.615
-0.010	0.968	0.191	0.432	-0.147	0.549
0.333	0.207	-0.070	0.797	-0.045	0.868
-0.236	0.061	0.003	0.982	-0.013	0.917
-0.536	0.003*	0.179	0.353	-0.032	0.868
-0.525	0.021*	0.208	0.393	-0.031	0.900
-0.706	0.023	0.099	0.785	-0.016	0.966
0.229	0.186	-0.040	0.821	-0.041	0.815
0.157	0.521	0.143	0.559	-0.005	0.985
0.347	0.188	-0.242	0.366	-0.145	0.593

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*	*r*
Selenium, μg				
All	64	-0.187	0.139	-0.227
Men	29	-0.349	0.063	-0.447
younger	19	-0.428	0.067	-0.550
older	10	-0.434	0.210	-0.522
Women	35	0.130	0.456	0.111
younger	19	0.171	0.485	0.130
older	16	0.061	0.822	0.083
Vitamin B-6, mg				
All	64	-0.126	0.323	-0.164
Men	29	-0.369	0.049*	-0.453
younger	19	-0.293	0.223	-0.386
older	10	-0.465	0.176	-0.566
Women	35	0.251	0.145	0.255
younger	19	0.347	0.145	0.279
older	16	0.092	0.735	0.246
Myristic Acid 14:0, g				
All	64	-0.204	0.105	-0.144
Men	29	-0.551	0.002*	-0.503

LDL		HDL	Triglycerides	
P	*r*	*P*	*r*	*P*
0.072	0.028	0.826	0.076	0.550
0.015*	0.219	0.254	0.095	0.625
0.015*	0.252	0.299	0.060	0.807
0.122	0.108	0.766	0.183	0.612
0.524	0.059	0.736	0.003	0.989
0.595	0.046	0.853	0.194	0.427
0.760	0.075	0.783	-0.343	0.194
0.195	0.073	0.569	-0.008	0.951
0.013*	0.226	0.239	0.011	0.955
0.103	0.243	0.316	0.010	0.969
0.088	0.170	0.640	0.149	0.680
0.139	0.054	0.759	-0.071	0.684
0.247	0.289	0.230	-0.079	0.748
0.358	-0.263	0.325	-0.033	0.904
0.256	-0.169	0.181	0.039	0.760
0.005*	-0.029	0.881	-0.045	0.816

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*
younger	19	-0.528	0.020*
older	10	-0.684	0.029*
Women	35	0.236	0.172
younger	19	0.218	0.369
older	16	0.245	0.361
Iron, mg			
All	64	-0.106	0.403
Men	29	-0.261	0.171
younger	19	-0.483	0.036*
older	10	-0.170	0.638
Women	35	0.183	0.292
younger	19	0.195	0.423
older	16	0.184	0.495
Animal Protein, g			
All	64	-0.174	0.169
Men	29	-0.375	0.045*
younger	19	-0.553	0.014*
older	10	-0.378	0.282
Women	35	0.187	0.282

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
-0.540	0.017	0.118	0.632	0.022	0.928
-0.524	0.120	-0.393	0.261	-0.116	0.749
0.286	0.096	-0.104	0.554	0.055	0.755
0.201	0.411	0.101	0.680	-0.023	0.927
0.427	0.099	-0.320	0.227	0.146	0.590
-0.080	0.528	-0.006	0.961	-0.075	0.558
-0.303	0.110	0.231	0.228	-0.007	0.971
-0.537	0.018*	0.317	0.187	-0.189	0.440
-0.205	0.570	-0.120	0.741	0.454	0.188
0.260	0.131	-0.078	0.654	-0.209	0.229
0.182	0.456	0.143	0.559	-0.183	0.454
0.463	0.071	-0.388	0.138	-0.256	0.338
-0.136	0.285	-0.037	0.770	-0.053	0.679
-0.400	0.032*	0.200	0.298	-0.022	0.909
-0.603	0.006*	0.316	0.188	-0.166	0.497
-0.337	0.342	-0.393	0.262	0.460	0.181
0.268	0.120	-0.068	0.697	-0.162	0.353

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*	*r*
younger	19	0.170	0.486	0.142
older	16	0.255	0.340	0.583
EPA 20:5, g				
All	64	0.016	0.898	0.065
Men	29	-0.177	0.357	-0.154
younger	19	-0.540	0.017*	-0.592
older	10	0.075	0.837	0.232
Women	35	0.388	0.021*	0.448
younger	19	0.505	0.027*	0.472
older	16	0.226	0.399	0.457
Linoleic acid 18:2, g				
All	64	-0.245	0.051	-0.295
Men	29	-0.497	0.006*	-0.573
younger	19	-0.419	0.074	-0.511
older	10	-0.738	0.015*	-0.800
Women	35	0.144	0.411	0.129
younger	19	0.070	0.775	0.053
older	16	0.238	0.376	0.233

LDL		HDL		Triglycerides
P	*r*	*P*	*r*	*P*
0.561	0.241	0.321	-0.241	0.320
0.018*	-0.508	0.044*	0.016	0.953
0.608	-0.066	0.604	0.058	0.650
0.424	0.031	0.872	0.189	0.327
0.008*	0.332	0.165	-0.091	0.710
0.519	-0.741	0.014*	0.614	0.059
0.007*	-0.025	0.886	-0.144	0.409
0.041*	0.226	0.353	-0.154	0.528
0.075	-0.305	0.250	-0.124	0.647
0.018*	0.068	0.593	-0.060	0.640
0.001*	0.196	0.308	-0.055	0.779
0.025*	0.198	0.416	0.006	0.982
0.005*	0.185	0.610	-0.131	0.717
0.460	0.041	0.818	-0.108	0.537
0.828	0.005	0.983	-0.025	0.918
0.385	0.091	0.739	-0.305	0.250

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol *r*	*P*	*r*
Cholesterol, mg				
All	64	-0.276	0.027*	0.252
Men	29	-0.416	0.025*	-0.414
younger	19	-0.425	0.069	-0.473
older	10	-0.476	0.164	-0.358
Women	35	-0.049	0.779	-0.042
younger	19	-0.116	0.637	-0.143
older	16	0.093	0.733	0.214
Total α-tocopherol, eq, mg				
All	64	-0.228	0.069	-0.272
Men	29	-0.475	0.009*	-0.541
younger	19	-0.407	0.083	-0.503
older	10	-0.677	0.032*	-0.705
Women	35	0.160	0.359	0.143
younger	19	0.106	0.667	0.066
older	16	0.253	0.344	0.274
CSI[4]				
All	64	-0.299	0.016*	-0.281
Men	29	-0.547	0.002*	-0.552

LDL		HDL	Triglycerides	
P	*r*	*P*	*r*	*P*
0.045*	-0.052	0.686	-0.052	0.683
0.026*	0.027	0.891	0.058	0.766
0.041*	0.163	0.506	0.045	0.856
0.310	-0.533	0.113	0.271	0.448
0.810	0.082	0.642	-0.211	0.224
0.559	0.253	0.296	-0.234	0.335
0.426	-0.219	0.415	-0.200	0.458
0.030*	0.068	0.596	-0.032	0.804
0.002	0.168	0.385	0.022	0.909
0.028*	0.240	0.322	0.012	0.963
0.023*	-0.034	0.925	0.114	0.753
0.413	0.090	0.609	-0.145	0.406
0.789	0.130	0.596	-0.053	0.830
0.304	0.051	0.852	-0.319	0.228
0.024*	-0.065	0.609	-0.031	0.807
0.002*	0.053	0.786	-0.009	0.963

TABLE 3-3 (continued)

Nutrients	*n*	Cholesterol		LDL		HDL		Triglycerides	
		r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
younger	19	-0.472	0.041*	-0.522	0.022*	0.150	0.540	0.047	0.850
older	10	-0.796	0.006*	-0.706	0.023*	-0.311	0.382	0.012	0.974
Women	35	-0.069	0.692	-0.083	0.637	0.030	0.865	-0.125	0.474
younger	19	-0.018	0.943	-0.046	0.851	0.211	0.386	-0.183	0.454
older	16	0.203	0.452	0.324	0.220	-0.212	0.431	-0.067	0.805

[1]Log transformed to improve normality of distribution.

[2] Of 35 nutrients tested, there were no significant correlations for: vitamin C , vitamin B-12, sucrose, β-carotene, dietary and soluble fiber, and folacin.

[3] Polyunsaturated to Saturated Fatty Acid Ratio

[4] Log of the Cholesterol Saturated Fat Index. CSI=(1.01 x g saturated fat) + (0.05 x mg cholesterol) (Connor et al., 1989)

* $P < 0.05$.

TABLE 3-4

Pearson bivariate correlation coefficients between blood lipids and energy adjusted nutrient intakes[1,2] for those found to be significant when analyzed using non-energy adjusted nutrient intakes, among Siberian Yup'iks, 1992, Gambell, Alaska.

Nutrients	*n*	Blood Lipids: Cholesterol		LDL		HDL		Triglycerides	
		r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
Saturated Fats									
All	64	-0.241	0.055	-0.240	0.056	0.034	0.789	-0.139	0.274
Men	29	-0.528	0.003*	-0.443	0.016*	-0.181	0.347	-0.133	0.491
younger	19	-0.277	0.252	-0.222	0.362	-0.229	0.347	0.270	0.264
older	10	-0.647	0.043*	-0.474	0.166	-0.199	0.582	-0.543	0.105
Women	35	0.039	0.826	-0.020	0.910	0.240	0.165	-0.140	0.423
younger	19	-0.139	0.571	-0.172	0.482	0.322	0.179	-0.441	0.059
older	16	0.406	0.119	0.287	0.282	0.144	0.594	0.463	0.071
Monounsaturated fats									
All	64	-0.129	0.308	-0.176	0.165	0.134	0.292	-0.095	0.454
Men	29	-0.428	0.021*	-0.437	0.018*	0.028	0.885	0.056	0.772
younger	19	-0.335	0.161	-0.381	0.108	0.063	0.798	0.234	0.335
older	10	-0.678	0.031*	-0.588	0.074	-0.295	0.408	-0.127	0.726

TABLE 3-4 (continued)

Nutrients	*n*	Cholesterol *r*	*P*
Women	35	0.151	0.385
younger	19	0.096	0.696
older	16	0.282	0.290
Polyunsaturated fats			
All	64	-0.112	0.377
Men	29	-0.371	0.048*
younger	19	-0.325	0.174
older	10	-0.444	0.198
Women	35	0.117	0.503
younger	19	0.030	0.902
older	16	0.370	0.159
Protein			
All	64	-0.040	0.752
Men	29	-0.154	0.426
younger	19	-0.478	0.039*
older	10	0.632	0.050*
Women	35	0.107	0.542
younger	19	0.112	0.649

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
0.103	0.558	0.212	0.222	-0.254	0.142
0.076	0.756	0.256	0.289	-0.440	0.059
0.152	0.574	0.156	0.565	0.193	0.474
-0.194	0.125	0.191	0.132	-0.107	0.402
-0.424	0.022*	0.106	0.584	0.024	0.901
-0.391	0.098	0.087	0.723	0.147	0.548
-0.504	0.138	0.156	0.666	-0.121	0.740
0.053	0.764	0.211	0.225	-0.263	0.128
0.026	0.915	0.048	0.846	-0.259	0.284
0.115	0.671	0.541	0.031*	-0.298	0.263
-0.013	0.922	0.053	0.677	-0.108	0.397
-0.165	0.392	0.161	0.403	0.006	0.976
-0.495	0.031*	0.229	0.347	-0.142	0.562
0.609	0.062	-0.175	0.628	0.539	0.108
0.168	0.335	0.019	0.915	-0.232	0.180
0.090	0.713	0.262	0.278	-0.321	0.180

TABLE 3-4 (continued)

Nutrients	*n*	Cholesterol *r*	*P*
older	16	0.188	0.487
Carbohydrate			
All	64	0.145	0.253
Men	29	0.448	0.015*
younger	19	0.526	0.021*
older	10	0.401	0.251
Women	35	-0.107	0.539
younger	19	-0.015	0.952
older	16	-0.375	0.152
Fat			
All	64	-0.185	0.143
Men	29	-0.508	0.005*
younger	19	-0.356	0.135
older	10	-0.805	0.005*
Women	35	0.123	0.480
younger	19	0.021	0.931
older	16	0.373	0.155

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
0.485	0.057	-0.441	0.087	0.060	0.825
0.142	0.262	-0.092	0.468	0.175	0.167
0.419	0.024*	-0.047	0.807	0.057	0.770
0.535	0.018*	-0.134	0.583	0.001	0.996
0.206	0.569	0.534	0.112	0.099	0.785
-0.109	0.532	-0.136	0.437	0.278	0.106
0.010	0.969	-0.317	0.186	0.505	0.027
-0.472	0.065	0.211	0.434	-0.377	0.150
-0.222	0.079	0.112	0.378	-0.123	0.332
-0.488	0.007*	-0.038	0.844	-0.014	0.945
-0.373	0.116	-0.024	0.921	0.238	0.326
-0.674	0.032*	-0.217	0.547	-0.394	0.260
0.066	0.706	0.238	0.169	-0.239	0.167
-0.001	0.998	0.260	0.283	-0.443	0.057
0.216	0.421	0.225	0.402	0.242	0.366

TABLE 3-4 (continued)

Nutrients	*n*	Cholesterol *r*	*P*
n-3 fatty acids			
All	64	0.028	0.820
Men	29	-0.186	0.333
younger	19	-0.450	0.053
older	10	0.157	0.665
Women	35	0.293	0.087
younger	19	0.340	0.155
older	16	0.250	0.351
n-6 fatty acids			
All	64	-0.128	0.313
Men	29	-0.344	0.068
younger	19	-0.193	0.428
older	10	-0.536	0.110
Women	35	0.013	0.943
younger	19	-0.128	0.601
older	16	0.268	0.316
Stearic acid (18:0)			
All	64	-0.230	0.068

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
0.058	0.647	0.011	0.933	-0.018	0.888
-0.146	0.449	-0.023	0.904	0.119	0.537
-0.470	0.042*	0.185	0.448	0.004	0.987
0.297	0.405	-0.578	0.080	0.363	0.303
0.330	0.053	0.045	0.796	-0.188	0.280
0.322	0.178	0.192	0.431	-0.272	0.259
0.402	0.123	-0.164	0.544	0.014	0.959
-0.225	0.074	0.223	0.077	-0.158	0.211
-0.404	0.030*	0.118	0.544	-0.095	0.625
-0.244	0.313	0.010	0.968	0.094	0.701
-0.639	0.047*	0.428	0.218	-0.373	0.289
-0.079	0.652	0.252	0.144	-0.227	0.190
-0.125	0.611	-0.024	0.924	-0.198	0.418
-0.029	0.915	0.621	0.010*	-0.332	0.209
-0.277	0.027*	0.136	0.285	-0.165	0.193

TABLE 3-4 (continued)

Nutrients	*n*	Cholesterol	
		r	*P*
Men	29	-0.483	0.008*
younger	19	-0.194	0.427
older	10	-0.772	0.009*
Women	35	-0.029	0.867
younger	19	-0.234	0.335
older	16	0.407	0.118
Iron			
All	64	0.024	0.852
Men	29	-0.038	0.843
younger	19	-0.373	0.116
older	10	0.666	0.036*
Women	35	0.110	0.530
younger	19	0.122	0.619
older	16	0.147	0.586
Animal protein			
All	64	-0.052	0.681
Men	29	-0.168	0.384
younger	19	-0.481	0.037*

LDL		HDL		Triglycerides	
r	*P*	*r*	*P*	*r*	*P*
-0.443	0.016*	-0.110	0.569	-0.117	0.544
-0.166	0.496	-0.189	0.439	0.234	0.334
-0.691	0.027*	0.035	0.924	-0.575	0.082
-0.134	0.442	0.352	0.038*	-0.209	0.228
-0.275	0.255	0.318	0.185	-0.444	0.057
0.142	0.601	0.434	0.093	0.256	0.339
0.052	0.683	0.055	0.664	-0.136	0.283
-0.052	0.787	0.188	0.328	-0.017	0.929
-0.387	0.102	0.253	0.296	-0.219	0.369
0.626	0.053	-0.082	0.821	0.550	0.100
0.173	0.321	-0.017	0.921	-0.256	0.138
0.119	0.626	0.146	0.550	-0.261	0.281
0.379	0.148	-0.300	0.259	-0.211	0.432
-0.004	0.973	0.023	0.858	-0.120	0.346
-0.149	0.441	0.134	0.487	-0.044	0.822
-0.481	0.037*	0.252	0.299	-0.200	0.413

TABLE 3-4 (continued)

Nutrients	*n*	Cholesterol		LDL		HDL		Triglycerides	
		r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
older	10	0.553	0.097	0.630	0.051	-0.447	0.195	0.480	0.160
Women	35	0.102	0.562	0.167	0.338	0.007	0.970	-0.210	0.226
younger	19	0.081	0.741	0.064	0.796	0.258	0.286	-0.340	0.154
older	16	0.257	0.336	0.567	0.022*	-0.482	0.059	0.207	0.441
Linoleic acid (18:2)									
All	64	-0.126	0.321	-0.223	0.076	0.223	0.076	-0.158	0.212
Men	29	-0.341	0.070	-0.401	0.031*	0.118	0.541	-0.101	0.603
younger	19	-0.184	0.450	-0.235	0.332	0.009	0.970	0.090	0.716
older	10	-0.544	0.104	-0.646	0.044*	0.434	0.210	-0.384	0.273
Women	35	0.011	0.948	-0.082	0.640	0.251	0.146	-0.218	0.209
younger	19	-0.128	0.602	-0.126	0.608	-0.028	0.910	-0.185	0.449
older	16	0.263	0.326	-0.035	0.898	0.621	0.010*	-0.328	0.215
EPA (20:5)									
All	64	0.169	0.183	0.222	0.078	-0.024	0.849	0.041	0.750
Men	29	0.050	0.798	0.113	0.561	-0.074	0.703	0.217	0.257
younger	19	-0.406	0.085	-0.414	0.078	0.246	0.310	-0.071	0.772
older	10	0.389	0.267	0.543	0.10	-0.668	0.035*	0.528	0.116
Women	35	0.346	0.042*	0.386	0.022*	0.049	0.779	-0.179	0.304

TABLE 3-4 (continued)

Nutrients	*n*	Cholesterol		LDL		HDL		Triglycerides	
		r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
younger	19	0.438	0.061	0.415	0.077	0.227	0.350	-0.227	0.350
older	16	0.215	0.425	0.394	0.131	-0.208	0.440	-0.043	0.875
Total α-tocopherol									
All	64	-0.100	0.432	-0.206	0.103	0.283	0.024*	-0.141	0.265
Men	29	-0.349	0.064	-0.398	0.033*	0.073	0.707	0.034	0.861
younger	19	-0.164	0.503	-0.227	0.349	0.070	0.777	0.133	0.587
older	10	-0.568	0.087	-0.626	0.053	0.055	0.880	0.090	0.805
Women	35	0.030	0.864	-0.088	0.615	0.408	0.015*	-0.333	0.051
younger	19	-0.102	0.679	-0.143	0.559	0.217	0.373	-0.287	0.234
older	16	0.336	0.203	0.044	0.871	0.635	0.008*	-0.406	0.119

[1]Calorie-adjusted intake = a + b, where a = residual for subject from regression model with nutrient intake as the dependent variable and total caloric intake as the independent variable and B = the expected nutrient intake for a person with mean caloric intake (Willett, 1990; Willett and Stampfer, 1986).

[2] Nutrients where significance is $P > 0.05$, after adjusting for energy: myristic acid (14:0), sodium, selenium, vitamin B-6 and cholesterol.

* $P < 0.05$.

TABLE 3-5 Significant[1] Pearson correlation coefficients for blood LDL-HDL ratios with nutrient intake[2] among Siberian Yup'iks, 1992, Gambell, Alaska.

Food	Correlations			
	Positive		Negative	
	r	*P*	*r*	*P*
Men and Women,				
40-87 y, *n* = 64				
α-tocopherol			-0.322	0.009*
n-6 Fatty acids			-0.297	0.017
Fatty acid 18:2			-0.296	0.017
Fatty acid 18:0			-0.278	0.026
Polyunsaturated fat			-0.255	0.042
Men and Women,				
60-87 y[3], *n* = 26				
Animal protein	0.688	0.000*		
Fatty acid 20:5	0.601	0.001*		
Protein	0.589	0.002*		
Iron	0.471	0.015		
n-3 fatty acids	0.452	0.020		
Fatty acid 18:2			-0.602	0.001*

TABLE 3-5 (continued)

n-6 fatty acids	-0.597	0.001*
α-tocopherol	-0.456	0.019
Fatty acid 18:0	-0.407	0.039
Polyunsaturated fat	-0.400	0.043
Men, 40-84 y[4], *n* = 29		
Index of Atherogenicity[5]	-0.501	0.006*
Index of Thrombogenicity[5]	-0.450	0.014
Energy	-0.429	0.020

[1]Of 23 nutrients tested, 13 were significantly correlated ($P < 0.05$) with LDL-HDL ratio and are shown.

*$P < 0.01$.

[2]Log-transformed nutrients from food frequency. All were energy adjusted except energy.

[3]Among men and women, 40-59 y, none of the correlations tested was significant ($P < 0.05$).

[4]Among women, none of the correlations tested was significant ($P < 0.05$).

[5]Ulbricht and Southgate, 1991.

TABLE 3-6 Significant[1] Pearson correlation coefficients for LDL-HDL ratios with foods[2] used by Siberian Yup'iks, 1992, Gambell, Alaska.

Food	Correlations					
	Positive			Negative		
	r	*P*	*n*[3]	*r*	*P*	*n*
Subjects, 40-87 y						
Pizza	0.278	0.038	56			
Syrup	0.250	0.050	62			
Tomatoes or tomato juice				-0.295	0.027	56
Cheese				-0.281	0.026	63
Fresh whale meat				-0.278	0.026	64
Fresh wild birds				-0.258	0.040	64
Evaporated milk				-0.248	0.050	63
Subjects, 40-59 y						
Evaporated milk				-0.442	0.006*	37
Chicken				-0.377	0.020	38
Mukluk (Bearded seal)				-0.337	0.038	38
Subjects, 60-87 y						
Fresh fish	0.521	0.006*	26			
Dried fish	0.412	0.036	26			

TABLE 3-6 (continued)

Men, 40-84 y						
Chips				-0.492	0.015	24
Tomatoes						
or tomato juice				-0.469	0.021	24
Soup, with mostly						
rice, macaroni,						
or noodles				-0.408	0.043	25
Cheese				-0.397	0.037	28
Women, 40-87 y						
Stew, with						
mostly meat	0.450	0.016	28			
Non-dairy creamer	0.392	0.020	35			
Pizza	0.363	0.045	31			
Hot dogs	0.334	0.050	35			
Fresh wild birds				-0.334	0.050	35

[1]$P < 0.05$ for 17 foods. Seventy-four foods were not significantly correlated.

*$P < 0.01$.

[2]Log-transformed foods from food frequency.

[3]Number for each correlation varies due to differences in number of responses to each food. Foods at the end of the questionnaire were more likely to be omitted.

TABLE 3-7 Inter-correlated nutrients[1] (r > 0.7 or < -0.7) which were energy adjusted consumed by Siberian Yup'iks by age or sex groups, 1992, Gambell, Alaska.

Nutrient	Fat	MUFA	PUFA	SFA	N-6	18:0	18:2	AnPr	Na	Se	Fe	α-toc	N-3
All													
MUFA	0.973												
SFA	0.857	0.747											
PUFA	0.820	0.812			0.857								
18:0	0.768			0.895	0.704								
18:2			0.847		1.000	0.704							
α-toc			0.830		0.888		0.885						
Younger													
MUFA	0.980												
SFA	0.870	0.787				0.915							
PUFA	0.887	0.866											
18:0	0.766			0.915									
18:2	0.703		0.857			0.774							
Na					0.776								
α-toc					0.907				0.746				

TABLE 3-7 (continued)

Nutrient	Fat	MUFA	PUFA	SFA	N-6	18:0	18:2	AnPr	Na	Se	Fe	α-toc	N-3
CHO					-0.730		-0.713						
Fe							0.901						
Older													
MUFA	0.940												
SFA	0.854												
PUFA					0.822								
18:0	0.758			0.848									
18:2			0.815		1.000							0.855	
α-toc			0.750		0.856								
Protein							0.930				0.813		
20:5													
0.938													
14:0			0.750										
Men													
MUFA	0.967												
SFA	0.839	0.711											
PUFA	0.835	0.826											
18:0	0.727			0.904									
18:2			0.821			0.716							

TABLE 3-7 (continued)

Nutrient	Fat	MUFA	PUFA	SFA	N-6	18:0	18:2	AnPr	Na	Se	Fe	α-toc	N-3
Na					0.799								
α-toc					0.902				0.750	0.734			
Fe							0.818						
Se					0.781				0.722				
Women													
MUFA	0.983												
SFA	0.885	0.804											
PUFA	0.806	0.803											
18:0	0.843												
18:2		0.760	0.890		0.892								
α-toc					0.836								
Fe							0.904						

[1]Nutrients include fat, monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), saturated fatty acids (SFA), n-6 fatty acids (N-6), stearic acid (18:0), linoleic acid (18:2), animal protein (AnPr), sodium (Na), selenium (Se), iron (Fe), α-tocopherol (α-toc), N-3 fatty acids (N-3), myristic acid (14:0), carbohydrates (CHO) and eicosapentaenoic acid (20:5).

TABLE 3-8 Multiple regression[1] between blood LDL-HDL ratio and food constituents used by Siberian Yup'iks. Significance of F is in parentheses. 1992, Gambell, Alaska.

Variables remaining in equations	Partial Regression Coefficient	*P*	*F*	(Sig)	AdjR^2
Subjects 40-87 y, men and women[2]			5.926	(0.000)	0.358
Fresh bird	-0.041	0.013			
Tomatoes or tomato juice	-0.026	0.058			
Evaporated milk	-0.024	0.061			
Cheese	-0.024	0.054			
Syrup	0.032	0.005*			
Pizza	0.033	0.007*			
Constant	0.667	0.000*			
Subjects 40-59 y[3]			8.696	(0.002)	0.391
Mukluk (Bearded seal)	-0.097	0.026			
Evaporated milk	-0.050	0.001*			
Chicken	-0.044	0.024			
Constant	0.173	0.316			
Subjects 60-87 y[4]			8.923	(0.006)	0.241
Fresh fish	0.157	0.006			
Constant	1.254	0.000*			
Men[5]			7.015	(0.015)	0.207
Chips	-0.046	0.015			
Constant	0.648	0.000*			
Women[6]			7.040	(0.004)	0.309
Meat stew	0.140	0.13			
Pizza	0.040	0.020			
Constant	1.441	0.000*			

[1]Backward elimination.
Variables entered and initial significance:
[2]syrup, 0.013, cheese, 0.030 evaporated milk, 0.042, fresh bird, 0.076, pizza, 0.012, age, 0.265, fresh whale, 0.241, tomatoes or tomato juice, 0.229, constant, 0.326, sex, 0.61.
[3]evaporated milk, 0.002, mukluk, 0.011, chicken, 0.024, constant, 0.080, sex, 0.139.
[4] constant, 0.001, fresh fish, 0.093, dry fish, 0.456, sex, 0.768.
[5] tomatoes or tomato juice, 0.225, chips, 0.245, soup with rice, macaroni or noodles, 0.380, age, 0.524, constant, 0.539, cheese, 0.699.
[6]constant, 0.000, meat stew, 0.040, pizza, 0.148, fresh bird, 0.199, hot dogs, 0.251, non-dairy creamer,0.386.
All variables were log transformed, except age and sex.
*$P < 0.01$.

TABLE 3-9

Multiple regression[1] between blood LDL-HDL ratio and intake of nutrients, by age and sex among Siberian Yup'iks. Significance of F values is in parentheses. 1992, Gambell, Alaska.

Variables remaining in equations	Partial Regression Coefficient	*P*	*F*	(Sig)	Adj R^2
Subjects 40-87 y[2]			7.166	(0.010)	0.089
α-tocopherol	-0.429	0.010**			
Constant	1.973	0.000**			
Subjects 60-87 y[3,4]			11.780	(0.000)	0.564
Animal protein	0.629	0.006**			
n-3 fatty acids	0.295	0.084			
n-6 fatty acids	-0.363	0.065			
Constant	-1.132	0.304			
Men[5]			9.059	(0.006)	0.223
IA	-0.317	0.006**			
Constant	0.711	0.000**			
Women[6]			3.872	(0.058)	0.078
α-tocopherol	-0.565	0.058			
Constant	2.330	0.004			

[1]Backward elimination. All variables log transformed. Variables entered, F value (initial significance) and *P* value (significance) of each variable:

[2]All: F = 1.247 (0.296) for six variables, Adj R^2=0.023; Index of atherogenicity (IA), -0.068 (0.491), age, -3.217E-04 (0.944), animal protein, -0.047 (0.700), sex, -0.030 (0.804), n-6 fatty acids, -0.090

(0.772), α-tocopherol, -0.288 (0.478) and constant, 2.045 (0.009**).

[3]Among subjects 40-59 y, none of the following variables achieved significance ($P < 0.05$): F=1.193 (0.334), Adj R^2=0.157; α-tocopherol, -0.372 (0.458), animal protein, -0.167 (0.240), sex, -0.161 (0.303), IA, -0.118 (0.343), n-6 fatty acids, 0.183 (0.643).

[4]Older subjects: F=7.002 (0.001**) for five variables, Adj R^2 = 0.546: IA, 0.065 (0.541), animal protein, 0.641 (0.007**), sex, 0.124 (0.308), n-6 fatty acids, -0.408 (0.055), n-3 fatty acids, 0.263 (0.137), constant, -1.279 (0.304).

[5]Men: F = 2.391 (0.069) for five variables, Adj R^2=0.199: IA, -0.353 (0.021*), animal protein, -0.135 (0.447), age, -0.004 (0.591), n-6 fatty acids -0.683 (0.096), α-tocopherol, 0.852 (0.155), constant, 1.066 (0.322).

[6]Women: F=0.927 (0.478), Adj R^2=0.138; age, 7.972E-04 (0.894), animal protein, 0.020 (0.906), IA, 0.124 (0.392), α-tocopherol, -0.787 (0.208), n-6 fatty acids 0.218 (0.681).

*$P < 0.05$, **$P < 0.01$.

TABLE 3-10 Multiple regression[1] relating blood LDL-HDL ratio to foods and nutrients consumed by Siberian Yup'iks. Significance of F values is in parentheses. 1992, Gambell, Alaska.

Variables remaining in equations	Partial Regression Coefficient	*P*	*F*	(Sig)	Adj R^2
Subjects 40-87 y[2], *n* = 64			6.412	(0.000)	0.417
α-tocopherol	-0.375	0.013*			
Fresh bird	-0.035	0.051			
Fresh whale	-0.034	0.093			
Evaporated milk	-0.032	0.015*			
Cheese	-0.024	0.053			
Syrup	0.028	0.008**			
Pizza	0.024	0.049*			
Constant	1.497	0.001**			
Subjects 40-59 y[3], *n* = 38			8.669	(0.000)	0.391
Mukluk	-0.097	0.026*			
Evaporated milk	-0.050	0.001**			
Chicken	-0.044	0.024*			
Constant	0.173	0.316			
Subjects 60-87 y[4] , *n* = 26			12.879	(0.000)	0.588
Animal protein	0.543	0.017*			
n-6 fatty acids	-0.408	0.034*			
Fresh fish	0.091	0.041*			
Constant	-0.216	0.860			
Men[5], *n* = 29			9.345	(0.000)	0.421
IA[7]	-0.292	0.007**			
Chips	-0.035	0.032*			
Constant	0.508	0.000**			

Women[6] , n = 35			7.426	(0.000)	0.417
α-tocopherol	-0.619	0.023*			
Meat stew	0.124	0.018*			
Hot dogs	0.030	0.035*			
Constant	2.901	0.001**			

[1]Backward elimination.

Variables entered and initial significance for each group:

[2]evaporated milk, 0.013, syrup, 0.014, α-tocopherol, 0.019, constant, 0.019, pizza, 0.031, cheese, 0.070, fresh bird, 0.075, fresh whale, 0.123, age, 0.131, tomatoes or tomato juice, 0.680, sex, 0.843.

[3]evaporated milk, 0.002, mukluk, 0.011, chicken, 0.024, constant, 0.080, sex, 0.139.

[4] animal protein, 0.021, fresh fish, 0.081, sex, 0.545, n-6 fatty acids, 0.049, n-3 fatty acids, 0.169, constant, 0.947.

[5]index of atherogenicity, 0.034, chips, 0.212, meat stew, 0.385, age, 0.474, constant, 0.613, cheese, 0.909, tomatoes or tomato juice, 0.963.

[6]constant, 0.001, meat stew, 0.058, α-tocopherol, 0.079, hot dogs, 0.177, fresh bird, 0.235, non-dairy creamer, 0.403, pizza, 0.446.

All variables were log transformed, except for age and sex; nutrients were adjusted for energy.

[7] Index of atherogenicity (Ulbricht and Southgate, 1991).

*P < 0.05. **P < 0.01.

TABLE 3-11 Multiple regression[1] relating blood LDL-HDL ratio to foods and nutrients consumed by Siberian Yup'iks, body mass index[2] and smoking practice[3]. Significance of F values is in parentheses. 1992, Gambell, Alaska.

Variables remaining in equations	Partial Regression Coefficient	*P*	*F*	(Sig)	Adj R^2
Subjects 40-87 y[4], *n* = 64			12.001	(0.000)	0.592
BMI[2]	0.985	0.000**			
α-tocopherol	-0.360	0.005**			
Fresh bird	-0.050	0.000**			
Evaporated milk	-0.025	0.019*			
Cheese	-0.020	0.053			
Syrup	0.032	0.001**			
Pizza	0.029	0.005**			
Constant	-1.587	0.036*			
Subjects 40-59 y[5], *n* = 38			10.203	(0.000)	0.506
BMI	0.797	0.006**			
Mukluk	-0.077	0.051			
Chicken	-0.047	0.009**			
Evaporated milk	-0.044	0.002**			
Constant	-2.367	0.011*			
Subjects 60-87 y[6] , *n* = 26			12.879	(0.000)	0.588
Animal protein	0.543	0.017*			
n-6 fatty acids	-0.408	0.034*			
Fresh fish	0.091	0.041*			
Constant	-0.216	0.860			
Men[7], *n* = 29			9.345	(0.001)	0.421
IA[8]	-0.292	0.007**			
Chips	-0.035	0.032*			

Constant	0.508	0.000**			
Women[9], n = 35			9.457	(0.000)	0.484
BMI	1.010	0.001**			
Pizza	0.048	0.002**			
Fresh bird	-0.047	0.022*			
Constant	-2.380	0.015*			

[1]Backward elimination.

[2]Body mass index (BMI) = body weight(kg)/height(m)^2

[3]Currently smokes or doesn't smoke.

Variables entered, partial regression coefficient and initial significance for each group:

[4]evaporated milk, -0.031 (0.008), cheese -0.021 (0.048), syrup 0.031 (0.002), α-tocopherol -0.422 (0.004), constant -1.887 (0.037), pizza 0.028 (0.010), fresh bird -0.041 (0.013), fresh whale -0.022 (0.283), age 0.006 (0.105), tomatoes or tomato juice 0.002 (0.856), sex 0.040 (0.648), smokes 0.013 (0.900), BMI 0.957 (0.000).

[5]evaporated milk, -0.041 (0.004), mukluk -0.094 (0.024), chicken -0.046 (0.009), constant -2.107 (0.031), sex -0.153 (0.153), BMI 0.781 (0.008), smokes 0.021 (0.853).

[6] animal protein 0.532 (0.024), fresh fish 0.059 (0.299), sex 0.081 (0.448), n-6 fatty acids -0.341 (0.093), n-3 fatty acids 0.319 (0.129), constant -1.238 (0.536), smokes 0.143 (0.387), BMI 0.108 (0.782).

[7]index of atherogenicity -0.349 (0.018), chips -0.024 (0.311), meat stew -0.004 (0.876), age 0.011 (0.208), constant -3.091 (0.216), cheese -0.008 (0.754), tomatoes or tomato juice 0.007 (0.781), smokes 0.188 (0.330), BMI 0.877 (0.197).

[8] Index of atherogenicity (Ulbricht and Southgate, 1991).

[9]constant -0.173 (0.905), meat stew 0.081 (0.131), hot dogs 0.028 (0.086), fresh bird -0.036 (0.118), non-dairy creamer 0.009 (0.527), pizza 0.021 (0.332), α - tocopherol -0.281 (0.307), age -0.005 (0.434), BMI 0.743 (0.024), smokes -0.077 (0.672).

All variables were log transformed, except for age and sex; nutrients were adjusted for energy.

*$P < 0.05$, **$P < 0.01$.

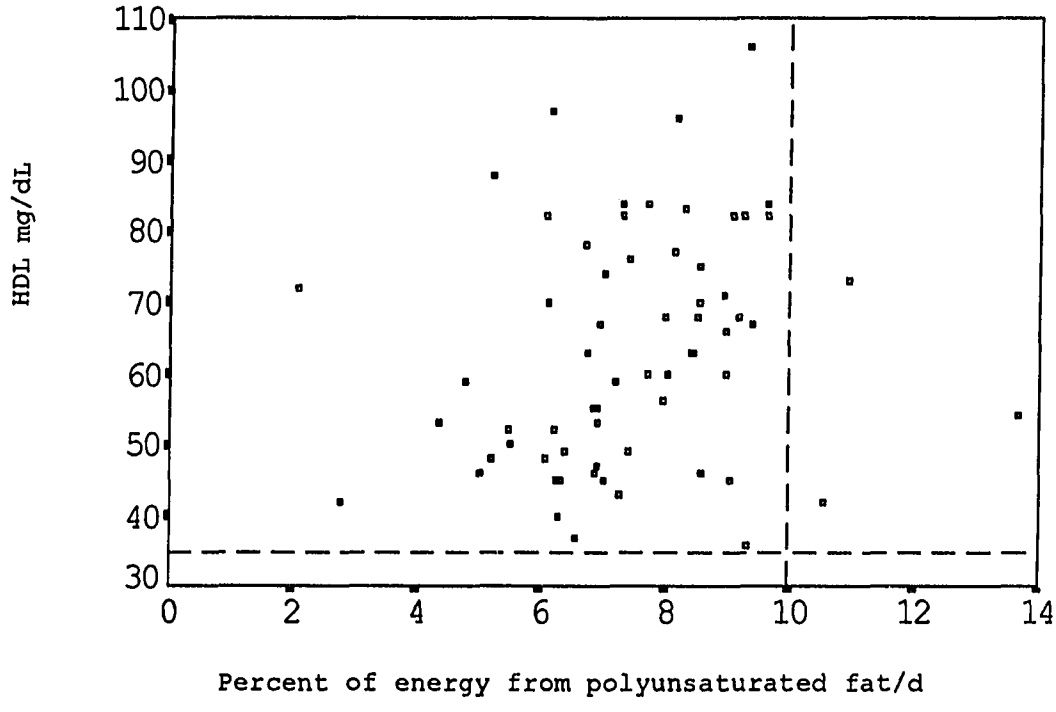

FIGURE 3-1 Blood HDL compared with percent of energy from polyunsaturated fat among 64 Siberian Yup'iks, 1992 (from food frequency questionnaire). Horizontal line represents 35 mg/dL HDL, below which is considered a risk factor for cardiovascular disease. Vertical line represents the NCEP recommendation: up to 10% of energy should be from polyunsaturated fatty acids.

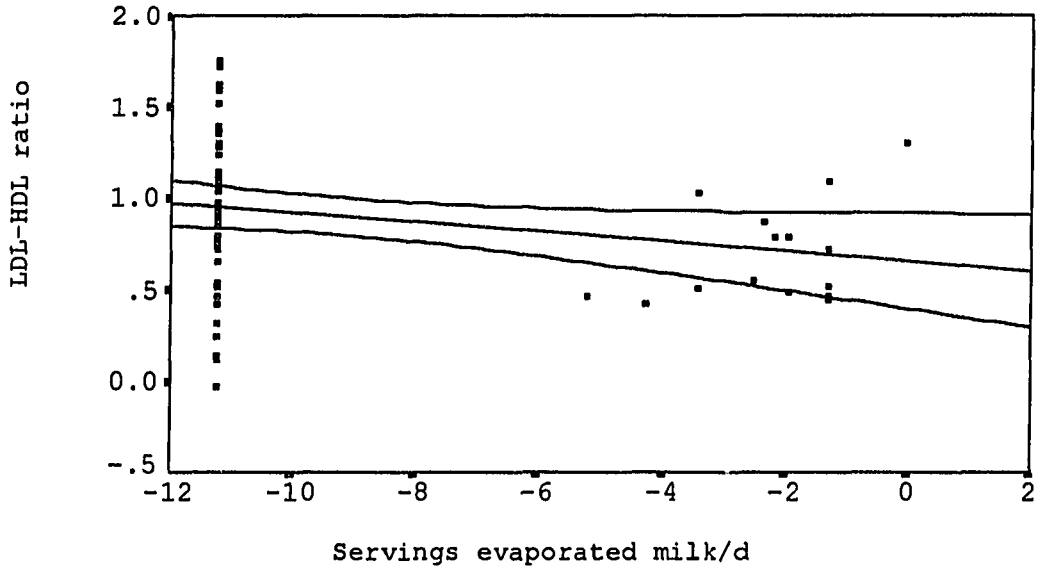

FIGURE 3-2 Blood LDL-HDL ratio compared with evaporated milk consumed by 64 Siberian Yup'iks, 40-87 years of age, 1992 (Log transformed). Values are from food frequency questionnaire. R^2 = 0.062, P = 0.05. Servings < -10 represent people who do not consume evaporated milk.

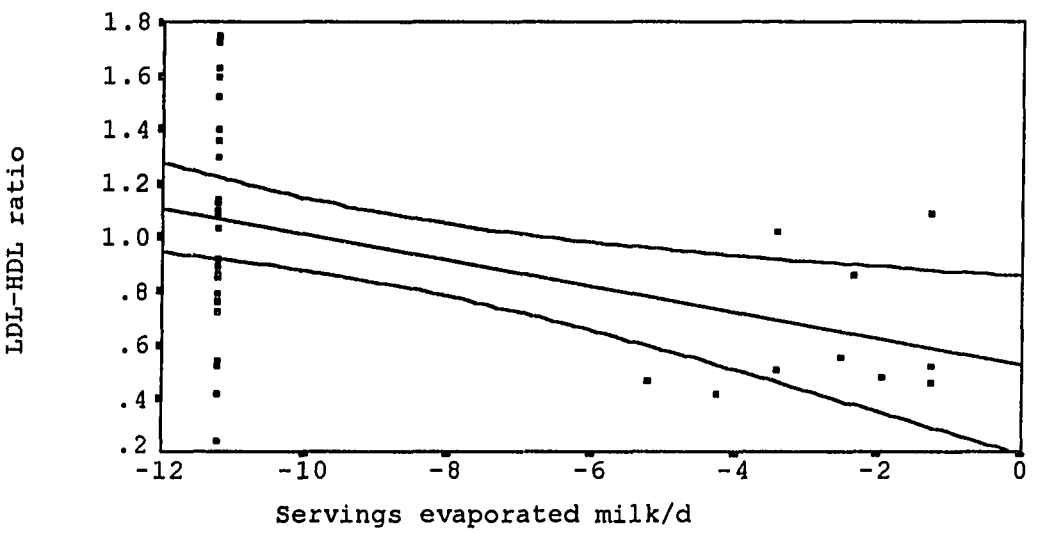

FIGURE 3-3 Blood LDL-HDL ratio compared with evaporated milk consumed by 38 Siberian Yup'iks, 40-59 years of age, 1992 (Log transformed). Values are from food frequency questionnaire. R^2 = 0.195, P = 0.006. Servings < -10 represent people who do not consume evaporated milk.

CHAPTER 4
SYNOPSIS

KEY FINDINGS

The analyses reported in Chapters 2 and 3 on the dietary intakes of Siberian Yup'iks revealed some findings that were expected, whereas others were surprising. It was anticipated that intakes of saturated fat would be positively correlated with blood cholesterol and with LDL among individuals, but they were not. In fact, they were notably negatively correlated for men. Both Rose (1992) and Willett (1990) observed that intakes of individuals do not correlate well with blood lipids although average intakes for populations do show a correlation. The difference in correlations when populations or individuals are compared may explain the lack of positive correlations in my data.

In this sample of Siberian Yup'iks, although absolute intakes of saturated fat by men were significantly lower than the general US intakes (NHANES III participants), the percents of energy from saturated fats and from polyunsaturated fats were not significantly different from the general US population. If saturated fats were twice as effective in raising serum cholesterol as polyunsaturated fats were in lowering it, then levels of cardiovascular disease would be comparable to US rates if all other factors were equal. Although there is no assessment of cardiovascular disease in the Gambell population, the trend in mortality rates from cardiovascular disease among Alaska Natives in general is one of convergence, with US rates declining and Alaska Native rates steady or increasing.

Factors other than saturated fats are involved in cardiovascular disease and in the lives of Siberian Yup'iks. In addition to non-dietary factors such as smoking and hypertension, n-3 fatty acids are consumed in levels high enough to theoretically elicit positive physiological changes. High intake of n-3 fatty acids can result in a reduction in VLDL levels, an inhibition of thromboxane production, an increase in prostacyclin synthesis with a resulting reduction in the likelihood of thrombosis, a reduced blood viscosity and a reduced risk of cardiac arrhythmias (Ascherio et al., 1995). Although these endpoints would measure the effects of n-3 fatty acid intake, they were not considered in this investigation. The effects of dietary n-3 fatty acids on Siberian Yup'iks should be considered.

Intakes of monounsaturated fats among both men and women were significantly greater than their NHANES III counterparts. Individual intakes were negatively correlated with LDL and cholesterol among all men, and the negative correlation with cholesterol was even stronger among older men. This was one of the few initial hypotheses that was supported, at least among men. A higher intake of monounsaturated fatty acids may be desirable as MUFAs lower total cholesterol and LDL but not HDL (type 3) as diets with higher amounts of polyunsaturated fat and carbohydrate do.

Carbohydrate intakes of men and women were significantly lower than NHANES III counterparts. Triglyceride levels, not surprisingly, also were low. Only younger women exhibited a notable positive correlation between carbohydrate and triglyceride levels.

Mean intakes of selected antioxidants are adequate when compared with the Recommended Dietary Allowances (RDA,

National Research Council, 1989*b*). Mean intakes of selenium, β-carotene, vitamin E, and iron exceeded the RDA among both men and women. The mean intake of vitamin C exceeded the RDA among men.

Among individuals, the nutrients α-tocopherol, n-6 fatty acids and the index of atherogenicity were markedly negatively correlated with the LDL-HDL ratio among at least one subgroup of the study population, whereas animal protein was positively correlated, but only among older adults. Total α-tocopherol and n-6 fatty acids also were correlated with each other, making it difficult to separate the effects of one from the other.

Traditional foods were generally negatively correlated with the LDL-HDL ratio, which is desirable. Nontraditional foods were positively correlated which is undesirable ($P < 0.05$).

Differences in dietary habits based on sex and age, were present among the residents of Gambell, as they were among other populations. Differences in both diet and cardiovascular disease risk factors must be taken into account in future studies as well as in intervention efforts. Men and women in general differ in their cardiovascular disease experience (Elliott, 1995).

The inclusion of the body-mass index in the regression equation among all participants increased the percent of variation explained in the LDL-HDL ratio from 42% to 59%. BMI had a positive effect on the LDL-HDL ratio among all participants, younger adults and women. Smoking, another possible confounding variable, did not explain any significant variation in the LDL-HDL ratio.

The assessment of dietary intake is a difficult procedure. In this study the use of two instruments provided

complementary data. The 24-h recalls yielded estimates of absolute intakes for the group, for 1 day in autumn, 1992, and these data were used to compare mean intakes with other populations where 24-h recalls were obtained (Chapter 2). The food-frequency values, however, provided better estimates of average intakes of individuals which were used for comparison with blood lipids of individuals (Chapter 3).

IMPLICATIONS

All of the correlations reported in Chapter 3, reflect linear associations between dietary components and blood lipids, but they do not prove cause and effect. Evidence beyond association is needed to prove the etiological relation of an agent(s) to a disease. One must show that the agent is the cause of the disease, and without the agent the disease does not occur. The task of identifying causative dietary agents for cardiovascular disease, is complicated by the influence of many variables and by the intercorrelation of nutrients. It should not be implied that one factor is the sole cause in a multifactorial disease such as cardiovascular disease (Steinberg, 1989).

This investigation addresses the presumed intermediary step in the association of diet and cardiovascular disease, that is, the blood lipids. By examining the significance and direction of the coefficients of foods, nutrients and body-mass index remaining in the regression equation to explain the LDL-HDL ratio variations among all participants, I conclude that α-tocopherol rich foods and fresh bird have significant protective effects. The relationships were least likely to occur by chance among the variables remaining in the equation. Body-mass index, syrup and pizza have detrimental effects. Based on the intercorrelation of α-

tocopherol with other nutrients foods rich in α-tocopherol also would be rich in polyunsaturated fats, n-6 fatty acids and linoleic acid. Food sources of α-tocopherol and linoleic acid in the diet of Siberian Yup'iks include pilot bread, fry bread, margarine and berry agutuk.

Where the coefficients for foods and nutrients were negative, there may be need for less concern if consumption of these foods continues. Foods and nutrients which are positively correlated deserve further attention based on the level of significance. The positive coefficient for animal protein and LDL-HDL ratio among older subjects suggests that they are consuming too much animal protein. The level of significance, however, was not as great as the significance for other variables among all participants ($P = 0.017$ for protein among older participants vs. $P < 0.001$ for BMI and bird among all participants). Animal protein, total protein and iron also were intercorrelated indicating any change in one nutrient will be reflected in the others.

When offering advice it is important to avoid creating harm by advocating a change which will have unintended effects. If a recommendation is made to limit a food, one must consider what is eaten in place of that food. The substitute could be worse. That could be the case if a recommendation to limit protein intake was made. The alternate choices may be foods with increased saturated fat which could raise LDL levels, or foods with increased carbohydrate which may lower HDL. Until better information is available active reduction of protein is not recommended.

The lack of significance in the regression, of nutrients expected to be significant, such as saturated fatty acid and dietary cholesterol, is noteworthy. In the absence of these dietary factors in the regression, more

attention may be placed on stressing protective factors such as monounsaturated fatty acids and n-3 fatty acids found in sea mammals and fish.

APPLICATION

Attempts to modify dietary patterns of an entire population must be based on evidence that the proposed intervention will have a significant impact on overall CHD mortality and be free of any potentially harmful side effects (McNamara, 1994). McNamara predicts that a population based approach to reducing plasma cholesterol levels will probably have a lowering affect on CHD incidence and mortality, although quantitatively it will be a relatively small effect overall. The objective among Alaska Natives is to improve health to the highest possible level by reducing the incidence of CHD morbidity and mortality. The role of modifying the diets of individuals is a complementary one to that of modifying the diet of the population. The focus of this investigation is the population through a detailed survey of 51% of the individuals.

Promulgating dietary advice cannot be done casually. It needs to be done with consideration for the entire picture (Marshall, 1995). Recognition of the total context of the role of food in the community is critical. This is especially true in Alaska Native communities where social structures and customs have evolved based on successfully obtaining sea mammals, for example. The positive aspects of sharing the work, dividing the harvest and celebrating the success of providing sea mammals in the community extend beyond the food's value as a source of nutrients.

Native foods still make a significant contribution to

the diet and social structure, although changes have occurred in eating patterns in Gambell since 1955 and even more changes since 1940 (Hughes and Hughes, 1960). Yet in 1984, per capita harvests of subsistence animals in Gambell were the second highest among 98 communities in Alaska surveyed (Wolfe and Walker, 1987). Hughes and Hughes (1960) described the people of Gambell as walrus eaters. In 1984 the harvest of marine mammals was > 3 times that of fish harvest (Wolfe and Walker, 1987). They still eat walrus, but other foods are eaten as well such as seal, poultry, pork, fish and beef.

Other diseases and dietary advice which may potentially conflict, need to be considered when formulating dietary advice. For example, nutrition recommendations were recently published for people with diabetes mellitus (American Dietetic Association, 1994). The Association recommended < 10% of energy from saturated fats, up to 10% from polyunsaturated fats, 10-20% from protein and the remainder from monounsaturated fats and carbohydrate based on individual nutrition assessment and treatment goals. These levels are generally consistent with current mean intakes for the population of Siberian Yup'iks (Chapter 2).

STRENGTHS AND LIMITATIONS

The strengths and limitations of data on which recommendations are made must be considered. Consistent data from more than one study, including cohort and case control studies, are desirable. Although consistency of findings is the objective, and defining specific variables to explain the findings is the goal, without crystal clear results, interpretation is subject to the vagaries of the complex topic and the small number of subjects. Multiple regression

is difficult to apply to dietary studies where intercorrelation occurs (Hegsted et al., 1965). The results don't prove that these variables affect the LDL-HDL ratio, only that they are associated and can explain the variance to some degree. When measured against these criteria my results need to be compared with other studies, but no cohort or case control investigations have been conducted among Siberian Yup'iks.

The strengths of this study include detailed information both on one day and on the past year. The results represent over one-half of the people ≥40 years-of-age from Gambell.

The weaknesses of the study include the small number of participants (n = 65) and the small number of Siberian Yup'iks in Gambell from which to recruit participants. Only adults ≥40 years-of-age were recruited to compare results with similar studies. Whether the participants represent the population ≥ 40 years may be questioned because of differences observed between women who participated in all phases of the investigation (n = 36) and 10 women who completed only the dietary component. Women participants compared with nonparticipants in the medical component were significantly older, consumed significantly more polyunsaturated fat and significantly less carbohydrate ($P < 0.05$). Whether these 10 are any more representative of all the Siberian Yup'ik women ≥ 40 y, than are the 36 participants in my study also is open to question. No significant differences were observed between men participants and nonparticipants (29 and 8 respectively).

Other factors were not considered in this investigation which also may influence blood lipids. These include hypertension, exercise, genetics and medical history.

Despite these limitations, dietary guidance can be formulated on the basis of what we currently know. I propose the following dietary advice as appropriate for residents of Gambell, Alaska, based on this study and other research (Chapter 1). As in all science, the recommendations are put forth with the understanding that future findings may require modification. When presenting dietary recommendations to the public, it is important to convey the concept that scientific findings are continually evolving.

Attaining and maintaining desirable weight is recommended. This is based on both reports of increasing health risks associated with higher BMI and on my results. The significant contribution of BMI in explaining the LDL-HDL ratio variation supports increased attention to achieving and maintaining desirable weight, especially among younger women.

Emphasis should be placed on limiting saturated fat rather than on limiting total fat. A moderate intake of fat with high intake of monounsaturated fatty acids is consistent with the comparatively low cardiovascular disease among Alaska Natives, and also is consistent with the traditional Alaska Native diet.

Foods high in monounsaturated fats and relatively low in saturated fats include walrus, seal oil, whale, olive oil, corn oil, peanut oil and margarine. A dietary pattern including these foods, with the exception of vegetable oils, is closer to traditional intakes, and therefore is more likely to be followed. I advocate a moderate fat intake if the intake of saturated fat is low and the intake of monounsaturated fat is high.

I encourage the use of foods containing monounsaturated fatty acids because excess polyunsaturated fatty acids tend

to suppress the immune system in laboratory animals and promote chemically induced cancers. In humans high intakes of polyunsaturated fatty acids lower HDL levels and when present in LDL particles, make them more susceptible to oxidation, thus enhancing atherogenesis. A growing reluctance to recommend high intakes of polyunsaturated fatty acids led Grundy and Friedman (1995) to conclude that intakes of polyunsaturated fatty acids probably should not exceed current levels. Although both monounsaturated fats and carbohydrates lower LDL concentrations, carbohydrates reduce HDL and raise VLDL levels. Monounsaturated fats generate more energy than carbohydrates, but if weight control is not a concern, monounsaturated fats are the appropriate substitute for saturated fats in the diet (See Grundy and Friedman, 1995). This further supports the recommendation of monounsaturated fat consumption among Siberian Yup'iks.

There seems to be little justification for promoting increased carbohydrates for the Siberian Yup'ik population. An exception may be considered for individuals if body weight control is an issue when energy dense foods need to be reduced and when energy expenditure cannot be increased. When carbohydrates are eaten complex carbohydrates should be advocated over simple carbohydrates, in order to increase fiber content of the diet.

A reduction in total fat intake to 30% of energy and a concomitant increased carbohydrate intake, as advocated for the general US population is inappropriate among Siberian Yup'iks. The recommendation for the US population does not acknowledge the uniqueness of Siberian Yup'ik diets. Recommending a higher fat intake is only appropriate, however, when the fat sources are high in monounsaturated

fats. It is not appropriate if the foods are high predominantly in saturated fat.

I recommend the continued use of traditional foods, especially sea mammals. They contributed one third of the monounsaturated fat to the diet and less than one fourth of the saturated fats.

FUTURE RESEARCH

There are several directions for future research on diet and cardiovascular disease among Alaska Natives. First, a replication study would be useful to corroborate or refute these findings. *Trans*-fatty acid content of foods and intakes are deserving of further investigation. Ascertaining the kinds of fish consumed and expanding the nutrient data base with additional analyses of different food samples would improve the precision of subsequent investigations. Further minor modification of the food frequency questionnaire is recommended (e.g. refining portion sizes and adding missing foods based on 24-h recalls in different seasons). Any future study should address hypotheses to test the unexpected correlations with LDL-HDL ratios, for animal protein(+ correlation), syrup(+), pizza (+), cheese (-) and chips (-).

Correlating the nutrients which act on mechanisms other than blood lipids i.e. n-3 fatty acids on clotting time, and antioxidants on thrombogenesis would be worthwhile. A case control study also would be useful in exploring dietary differences between those with cardiovascular disease and those without, but the study population would have to be larger than Siberian Yup'iks only in order to obtain enough cases. Correlating nutrients and foods with diagnosed cardiovascular disease would be most desirable, but may be

problematic given the small size of the population. Diet of the distant past (10-20 years ago) could be assessed through historic records of food outlets and subsistence harvests, and through interviews with individuals to determine the extent that historical diet correlates with current coronary disease experience. Alternately, investigators could reassess the coronary heart disease status of current study participants in 10-20 years. Mortality is a latent marker for cardiovascular disease needing several years to manifest itself, so dietary practices measured now could explain events several years from now.

BENEFITS

The benefits of this survey of the diet and blood lipid status of the people of Gambell are important for documenting the unique nature of the diet. Such investigations benefit the people of Gambell by offering recommendations on dietary practices which should enhance the maintenance of cardiovascular health. Documentation of the unique nature of the native diet of Siberian Yup'iks enhances our general understanding of diet and its relation to cardiovascular disease.

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APPENDIX A-1

NATIVE DIETARY INTAKE QUESTIONNAIRE

Circle or fill in answers

STUDY NO ___ ___ ___ TIME DIET INTERVIEW STARTED _____AM or PM

Birth Date Age in years _____

Sex: (circle) Male Female Ethnic Group _____

Are there any health problems that affect your diet (what you eat)?

_____

_____

If you take vitamins or mineral pills, how long have you taken them?
List Name(s) _____
_____ months _____ years _____ _____
How many days per week? _____ _____ _____
How many pills per week? _____ _____ _____

Where do you get them? (circle) store bought

Clinic/Hospital (Specify which) _____ Other (Specify)_____

Where do you get your drinking water? (circle)

Municipal Water Lake Rain Spring Ice Bottled
Well Other River

Do you use mineral water? Yes _____ No _____ Sometimes _____

Do you add salt to cooked foods? Yes _____ No _____ Sometimes _____

How much sugar do you add to a cup of tea?

_____ level tsp(s)/ _____ rounded tsp(s) _____ cube(s) _____ none

How much sugar do you add to a cup of coffee?

_____ level tsp(s)/ _____ rounded tsp(s) _____ cube(s) _____ none

Did you drink alcohol last week? Yes _____ No _____

If yes, what kind? (Circle)

Beer Homebrew Wine Other Liquor

Name _____
P.O. Box _____
Mailing address _____

Send results? Yes No

If person refused, to be interviewed, give reason_____

Interviewer _____ Time interviewed ended _____
length of interview (minutes)

Language of Interview 1. English _____ 2. Siberian Yup'ik _____

Recipes, Miscellaneous notes or picture of food size

Please do no write below this line

Food Frequency Questionnaire

"Now I'm going to ask about what you usually eat over the year. For each food, tell me about how often you eat them, and how much you eat at one time. If you ate the food only in certain seasons, tell me which season. For example: In the last year, how many times on average did you eat this size serving of chicken?" (show model) Answer: three times a week, all year long, two times the size.

						Season				
	day	week	month	year	<1	F	W	Sp	Su	Yr
Example Answer: **chicken** 1 serving,1/2, 2, more		3								X
Fresh meat, fish, birds (Show chicken model) **fish** 1 serving, 1/2, 2, more										
walrus 1 serving, 1/2, 2, more										
seal 1 serving, 1/2, 2, more										
whale 1 serving, 1/2, 2, more										
wild birds 1 serving, 1/2, 2, more										
reindeer 1 serving, 1/2, 2, more										
mukluk (bearded seal) 1 serving, 1/2, 2, more										
liver 1 serving, 1/2, 2, more										
muktuk 1 serving, 1/2, 2, more										

beef (any kind) 1 serving, 1/2, 2, more										
chicken 1 serving, 1/2, 2, more										
pork or ham 1 serving, 1/2, 2, more										
Spam 1 serving, 1/2, 2, more										
Dried meats, fish and birds (show dried model) "For dried meats, fish and birds, do you usually eat this much, 1/2 this, twice this or more at a time? (circle)										
dried fish 1 serving, 1/2, 2, more										
dried walrus 1 serving, 1/2, 2, more										
dried seal 1 serving, 1/2, 2, more										
dried whale 1 serving, 1/2, 2, more										
dried birds 1 serving, 1/2, 2, more										
murre eggs How many at a time?_____										
chicken eggs How many at a time?_____										

hot dogs How many at a time?_____										
bacon or sausage How many pieces at a time?_____										
luncheon meats If yes kinds _____ How many slices?_____										
cheese If yes, kinds _____ How many slices?_____										
Mixed dishes (show 14 oz bowl) "For these mixed dishes do you usually eat 1 full bowl, 1/2 of this, 2 bowls or more at a time?" (circle)										
berry agutuk list kinds of fat usually used _____ 1 1/2 2 more										
deer meat agutuk list kinds of fat usually used _____ 1 1/2 2 more										
agutuk made with greens list kinds of fat usually used _____ 1 1/2 2 more										
french fries, or fried potatoes 1 1/2 2 more										

white rice 1 1/2 2 more										
Beverages (show 12 oz glass) This glass holds the same as a can of soda, a can of beer or a bottle of beer; tell me if you usually have 1, 1/2, 2, 6 or more. Hi-C, Tang, Hawaiian Punch, Kool-aid, Gatorade or other drinks with added vitamin C 1 1/2 2 6 more										
regular soda, not diet 1 1/2 2 6 more										
milk What type was it usually? evaporated, whole, 2%, 1% skim or non-fat or some other type __whole/regular __2% low fat __1% __skim/non-fat __evaporated __other __don't know 1 1/2 2 more										
beer 1 1/2 2 6 >6										
liquor or home brew 1 1/2 2 more										
wine 1 1/2 2 more										

water 1 1/2 2 more										
For the following foods do you usually eat 1, 1/2, 2 or more? (Show tablespoon) **sugar** 1 1/2 2 more										
jelly 1 1/2 2 more										
syrup 1 3 6 more										
peanut butter 1 1/2 2 more										
margarine 1 1/2 2 more										
butter 1 1/2 2 more										
crisco 1 1/2 2 more										
seal oil 1 1/2 2 more										
vegetable oil **kind?**_____ 1 1/2 2 more										
mayonnaise or salad dressing **kind?**_____ 1 1/2 2 more										
non-dairy creamer **kind?**_____ 1 1/2 2 more										
What kind of sweets do you like_____ _____ _____										
cake 1 1/2 2 more pieces										
pie 1 1/2 2 more pieces										

cookies 1 2 3 more										
doughnuts 1 2 3 more										
candy (gumdrop size) 1 5 10 more										
pilot bread 1 2 3 more										
fry bread 1 1/2 2 more										
dark bread or rolls 1 1/2 2 more										
white bread or rolls 1 1/2 2 more										
coffee (show 10 oz mug) 1 1/2 2 6 more										
tea (show 10 oz mug) 1 1/2 2 6 more										
For these foods tell me if you usually have 1, 1/2, 2, or more cups. (show 1 cup)										
carrots or mixtures containing carrots 1 1/2 2 more										
Brussels sprouts, cauliflower or broccoli 1 1/2 2 more										
sweet potatoes, orange squash, pumpkin 1 1/2 2 more										

tomatoes or tomato juice 1 1/2 2 more										
tossed salad or lettuce 1 1/2 2 more										
wild greens 1 1/2 2 more										
canned, frozen greens 1 1/2 2 more										
seaweed, opah 1 1/2 2 more										
green beans, corn, peas and other vegetables 1 1/2 2 more										
kidney beans, baked beans, or soup beans 1 1/2 2 more										
berries 1 1/2 2 more										
orange juice 1 1/2 2 more										
peaches or apricots 1 1/2 2 more										
orange, whole 1 1/2 2 more										
apple, whole 1 1/2 2 more										
banana, whole 1 1/2 2 more										
potatoes other than french fries, whole 1 1/2 2 more										

(Show 14 oz. bowl for next foods) **stew with mostly meat (with vegetables)** 1 1/2 2 more										
soup with mostly rice, noodles or macaroni 1 1/2 2 more										
green juice soup 1 1/2 2 more										
spaghetti or pasta with tomato sauce 1 1/2 2 more										
cereal -kind, brand- 1 1/2 2 more										
ice cream 1 1/2 2 more										
chips 1 1/2 2 more										
popcorn 1 1/2 2 more										
pancakes 1 1/2 2 more										
pizza no. slices ____ kind _____										

Are there any other foods or beverages you ate at least once a week in the past year?

____yes (specify) _____no

These are the final few questions!

About how many servings of fruit do you eat a day on average? _____

About how many servings of vegetables do you eat a day on average? _____

That's the last food question!
Your comments will help us improve our questions.
Do you have any comments about the ways we can improve?

May we contact you again to learn how you eat in different seasons?

Yes No If yes, phone no. _____ or no phone

"Thank you for your help! Please remember do not eat for 12 hours before coming to City Hall tomorrow."

Food Frequency Responses _____ reliable
_____ unreliable

APPENDIX A-2

DERIVATION OF NUTRIENT VALUES OF FOODS CONSUMED BY SIBERIAN YUP'IKS

The NCC Nutrient Data System 2.6/8A/23 was used to calculate the intakes. When nutrient values for Alaska Native Foods were lacking, NCC used values compiled by Nobmann (1993) from published sources and original analyses. Those values were weighted based on the number of samples reported.

According to Draper (personal communication), Wo (1973) reported fatty acid values as percent of total fatty acids, not percent of total food. USDA, in reporting grams of fatty acid per 100 grams of food, uses conversion factors[1] to account for not all fat is fatty acid. While it would have been preferable to compute the conversion factors for Wo's work before averaging, this was not done. It was felt that for this study, whose main purpose is to compare intakes among individuals, the omission of the conversion factors would not seriously affect the results. So few food samples have been tested and the biological variability among samples overshadows the variability introduced by the omission of the conversion factor for some of the samples.

To obtain information on foods where nutrient values did not exist, Alaskans were asked for samples which were shipped to one of two laboratories for analysis. The results for foods consumed by Siberian Yup'ik subjects are listed below, along

with laboratory quality control information[2,3]. Not all nutrients were analyzed for each food. The number of samples was dependent on what was obtained from contributors.

---

[1] 0.875 used for separable lean beef and 0.918 for separable beef fat(USDA, 1986) and 0.933-(0.143/total lipid expressed as g/100 g food) for finfish (USDA, 1987), are examples. Different conversion factors for numerous foods have been compiled from published sources and used in the Agriculture Handbook 8 (USDA, 1976, 1984, 1986, 1987) revisions.

[2]Laboratory 1) USDA Agricultural Research Service, Beltsville Human Nutrition Research Center, Diet and Human Performance Laboratory, Beltsville, MD. (James P. Church, Personal communication, 1995). Two types of errors were considered, extraction and instrumental. All samples were extracted by the Folch method, within a short time interval, which extracts lipids with percentages in the mid to high 90's for most food samples while a few, mostly collagenous samples containing gristle or skin, are in the low 90's or high 80's. Instrumental errors were less than 2% which was within acceptable limits. Analytical optimization of the gas liquid chromatography system was confirmed on each of the 7 days samples were analyzed, using a standard mixture, composed of 17 pure fatty acid methyl esters

in iso-octane.

[3]Laboratory 2) Columbia Laboratories, Inc., Corbett, OR. The Quality Assurance Program for Nutrition Chemistry (Columbia Laboratories, 1992) stated:
Comparability: Data collected and reported will be consistent with the National MSIS (Model State Information System) and FRDS (Federal Reporting Data System). AOAC Sections were referenced for each nutrient (13th and 15th editions). Proximate analyses were ± 0.5% accuracy with ± 1 SD precision. Other nutrient analyses were ± 10.0% accuracy and ± 2 SD precision.
Validation: Spiked samples were used to check for precision and accuracy and must meet the criterion of 85% or better for recovery and reference samples must be within two SD of the true value.
Analytical procedures: Calibration Standards were purchased through Aldrich Chemical Co. and Sigma Chemical Co.
Source Traceability: Standards Receiving and Instrumentation Standards Logs were used to document details of the standards and their use.
Specific procedures used to assess data precision accuracy and completeness included % recovery from spiked sample - 85%, and quality control samples and reference samples where values obtained must be > or < 2 SD from the mean.

APPENDIX A-2 (continued)

Nutrient values of Alaska Native foods analyzed by Nobmann, ED (1990-93) and referenced by Nutrition Coordinating Center.

Caribou fat (*Rangifer tarandus*)	
Nutrients per 100 g food	Sample(Lab 1) 1[a]
Moisture, g	13
Fat, g	73.5
Fatty acids[b],g:	
Saturated, total	32.5
12:0	0.0
14:0	0.86
16:0	11.0
18:0	19.1
Monounsaturated, total	28.2
16:1	1.32
18:1	25.4
20:1	0.46
Polyunsaturated, total	5.09

APPENDIX A-2 (continued)

N-3 fatty acids	2.38
18:3	2.32
20:5	0.07
22:5	0.0
22:6	0.0
N-6 fatty acids	2.71
18:2	2.12
20:4	0.0
Other fatty acids over 0.5% of wet weight	
17:0	0.99
18:1n7	0.60
Cholesterol, mg	37

---

[a]Fat from front thigh. Caribou taken from Nelchina Herd 75 miles north of Anchorage 6 miles south of the Glenn Highway, 28 August 1990.

[b]The grams of fatty acid per 100 g of food were calculated by the Laboratory from normalized percent of fatty acid, based on the assumption that 90.0% of the fat was composed of fatty acids.

APPENDIX A-2 (continued)

Fireweed, leaves, top, flower, raw (*Epilobium latifolium*)

Nutrients per 100 g food	Sample(Lab 2) 1[a]
Total dietary fiber, g	6.0
Sodium, mg	50
Phosphorus, mg	204
Potassium, mg	251

[a] Three samples (from Peters Creek, Gulkana, and Nanak Island in Prince William Sound, AK), collected 20, 29, 30 May 1990. Samples were combined prior to analysis.

APPENDIX A-2 (continued)

Fish, dried, salmon, king, smoked and brined (*Oncorhynchus*)

Nutrients per 100 g food	Sample(Lab 2 all samples) 1[a]	2[b]	3[c]	4[d]	$\bar{x}$
Moisture, g	18.7	15.9	41.1	18.8	23.6
Energy, kcal	446	466	349		420
Protein, g	44.9	47.6	27.2		39.9
Carbohydrate, g	4.9	0.7	3.2		2.9
Fat, g	27.9	32.1	25.7		28.6
Total dietary fiber, g	1.1	4.0	1.0		2.0
Ash, g	3.5	3.7	2.8		
Calcium, mg	25	17	28		23
Iron, mg	4.8	3.9	4.7		4.5
Sodium, mg	644	745	703	680	693
Phosphorus, mg	509	452	322		428
Potassium, mg	806	832	461		700
Vitamin A, Carotene, IU	220	240	50		170
RE	22	24	5		17

APPENDIX A-2 (continued)

Retinol, IU	440	340	290		357
RE	44	34	29		36
Vitamin C, mg	0.0	0.0	0.0		0.0
Thiamin, mg	0.09	0.07	0.03		0.06
Riboflavin, mg	0.27	0.36	0.22		0.28
Niacin, mg	12.6	13.7	9.1		11.8
Lipids:					
% calories from fat	56	62	66	34	55
Fatty acids, g :					
Saturated, total	6.88	7.10	5.71	8.18	6.97
12:0	0.0	0.0	0.00	0.00	0.00
14:0	1.04	1.39	1.07	1.57	1.02
16:0	4.73	4.60	3.71	5.45	1.26
18:0	1.10	1.11	0.93	1.16	1.08
Monounsaturated, total	15.57	19.3	13.13	19.68	16.9
14:1	0.08	0.08	0.07	0.09	0.08
16:1	2.06	2.44	2.01	2.96	2.37
18:1	8.79	9.16	6.33	10.03	8.58
20:1	2.76	4.21	2.82	3.89	3.42
22:1	1.88	3.41	1.90	2.71	2.48

APPENDIX A-2 (continued)

Polyunsaturated, total	4.05	4.05	3.33	4.81	4.06
N-3 fatty acids	3.83	3.68	2.88	4.46	3.71
18:3	0.00	0.00	0.00	0.00	0.00
20:5	2.04	2.15	1.68	2.54	2.10
22:6	1.79	1.51	1.20	1.92	1.61
N-6 fatty acids	0.22	0.37	0.45	0.35	0.35
18:2	0.22	0.37	0.45	0.35	0.35
Cholesterol, mg	137	151	59	80	107

[a] Yukon River, Fort Yukon, AK, 27 June 1990. Fish back strip with skin on was washed 3-4 times, put in salt solution (non-iodized salt) for 3-5 minutes, so not too salty. Strips were hung to dry in an open cache over a cottonwood smoke, for 2 1/2 weeks. Fire was kept going the entire time, but the fire did not heat the fish. Weather was sunny, about 80 degrees F.

[b] Kuskokwim River, Napamute, AK. Raw fish sliced and placed in brine for 15-20 minutes. Hung for 1 day outside, then put in smokehouse for > 1 month, then left in fish cache until 24 or 25 June 1990.

[c] Lower Yukon River (mouth of river), AK, 1990. A five gallon bucket was half filled with water; rock salt was added until a potato floated in the solution (about 2-3 cups). Fish were added until the bucket was full. Fish were hung to dry

APPENDIX A-2 (continued)

with a cold smoke using alder wood.

[d]Yukon River, six miles south of Kaltag, AK.

APPENDIX A-2 (continued)

Reindeer, meat; Raw (*Rangifer tarandus*)

Nutrients per 100 g food	Sample(Lab 1) 1[a]
Moisture, g	74
Fat, g	3.87
Lipids:	
Fatty acids, g:	
Saturated, total	1.29
12:0	0.0
14:0	0.04
16:0	0.61
18:0	0.60
Monounsaturated, total	1.16
16:1	0.06
18:1	1.05
20:1	0.01
Polyunsaturated, total	0.41
N-3 fatty acids	0.12
18:3	0.09

APPENDIX A-2 (continued)

20:5	0.03
22:5	0.0
22:6	0.0
N-6 fatty acids	0.29
18:2	0.21
20:4	0.07
Cholesterol, mg	16

[a] Shoulder meat. From Hagemeister Island off Togiak, AK.

APPENDIX A-2 (continued)

Salmon eggs, king, raw (*Oncorhynchus tschawytsch*)

Nutrients per 100 g food	Sample(all from Lab 1) 1[a]	2[a]	3[b]	4[c]	$\bar{X}$
Moisture, g	54	55	59	69	59
Fat, g	16.3	15.3	14.3	11.6	14.4
Lipids:					
Fatty acids, g:					
Saturated, total	2.35	2.03	2.17		2.18
12:0	0.0	0.0	0.00		0.00
14:0	0.39	0.35	0.33		0.36
16:0	1.42	1.23	1.35		1.33
18:0	0.42	0.36	0.44		0.41
Monounsaturated, total	5.59	4.72	2.89		4.4
16:1	0.87	0.75	0.56		0.73
18:1	3.83	2.74	1.62		2.73
20:1	0.68	0.56	0.22		0.49
Polyunsaturated, total	4.68	3.83	4.67		4.39
N-3 fatty acids	4.19	3.42	4.39		4.0
18:3	0.09	0.08	0.04		0.07

APPENDIX A-2 (continued)

20:5	1.37	1.11	1.87		1.45
22:5	0.47	0.39	0.59		0.48
22:6	1.96	1.60	1.70		1.75
N-6 fatty acids	0.49	0.41	0.28		0.39
18:2	0.19	0.16	0.07		0.14
20:4	0.24	0.19	0.15		0.19
Cholesterol, mg	237	179	171	109	174

[a] Upper Cook Inlet, West side, Kenai, AK, 9 June 1990.

[b]Sitka Sound, Sitka, AK, 2 July 1990.

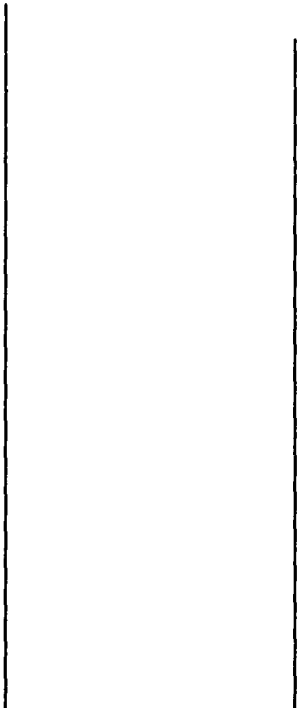
[c]Dillingham, AK, End of June, 1990.

APPENDIX A-2 (continued)

Salmonberries, (*Rubus spectabilis*)

Nutrients per 100 g food	Sample(Lab 2) 1[a]
Total dietary fiber, g	1.0
Phosphorus, mg	26
Potassium, mg	108
Vitamin C, mg	0.3

[a]Starrgauin State Park,Sitka, AK, 5 July 1990.



APPENDIX A-2 (continued)

Seal oil (*Pinnipedia*)

Nutrients per 100 g food	Sample 1[a] (Lab1)	Sample 2[b] (1)
Moisture, g	0.0	0.0
Energy, kcal		
Protein, g		
Carbohydrate, g		
Fat, g	100	100
Ash, g		
Calcium, mg		
Iron, mg		
Sodium, mg		
Phosphorus, mg		
Potassium, mg		
Vitamin A,		
Carotene, IU		
RE		
Retinol, IU		

3^c (2)	$\overline{X}$
0.0	0.0
854	
0.4	
8.6	
90.9	97.0
	0.0
1	
0.6	
7	
0	
8	
0	
0	
753	

APPENDIX A-2 (continued)

RE	
Vitamin C, mg	
Thiamin, mg	
Riboflavin,mg	
Niacin,mg	
Lipids:	
Fatty acids,g:	
Saturated, total	14.41
12:0	0.05
14:0	2.94
16:0	9.11
18:0	1.79
Monounsaturated, total	56.50
16:1	14.96
18:1	35.37
20:1	3.76
Polyunsaturated, total	21.84
N-3 fatty acids	18.38
18:3	0.53
20:5	4.64

75
0
0
0.00
0.4

12.41	15.20	14.01
0.0		0.03
2.87	3.61	3.14
7.89	10.24	9.08
1.02	1.35	1.39
49.4	38.00	47.97
17.32	13.55	15.28
18.48	24.45	26.10
5.46		4.61
31.74	33.15	28.91
9.64	31.63	26.55
0.43	8.02	2.99
10.45	12.63	9.24

APPENDIX A-2 (continued)

22:5	2.92	5.76		4.34
22:6	7.71	11.37	10.98	10.02
N-6 fatty acids	3.46	2.10	1.52	2.36
18:2	2.36	0.83	1.52	1.57
20:4	0.72	0.68		0.70
Other fatty acids over 0.5% of wet weight				
18:4 n-3	2.05			
18:1 n-7		5.36		
Cholesterol, mg	3	2	67.8	24.27

[a]Fur seal from Dahl herd near Ketchikan, AK, March, 1989. Fat parboiled, then fried until fat is crisp and oil poured off.

[b]Spotted or Larga seal from Bering Sea near Kipnuk, AK, 23 September 1990.

[c]Ringed seal from Dillingham, AK Region, 1990.

APPENDIX A-2 (continued)

Tunicates, Ascidians[a] (*Tunicata Ascidiacea*)

Nutrients per 100 g food	Single Samples(All Lab 2) 1[b]	2[c]
Moisture, g	90.8	83.1
Energy, kcal	23	34
Protein, g	2.7	5.6
Carbohydrate, g	1.7	1.9
Fat, g	0.6	0.4
Ash, g	4.2	9.0
Calcium, mg	43	348
Iron, mg	13.0	33.1
Sodium, mg	705	606
Potassium, mg	80	103
Vitamin A,		
Carotene,IU	0.0	0.0
Retinol, IU	0.0	0.0
Vitamin C, mg	0.0	0.0
Thiamin, mg	0.00	0.00
Riboflavin,mg	0.10	0.20

3[d]	4[e]
86.5	90.4
41	28
7.1	3.8
2.0	2.2
0.5	0.5
3.9	3.1
451	47
9.7	3.3
650	656
118	102
0.0	0.0
0.0	0.0
0.0	0.0
0.00	0.00
0.10	0.20

APPENDIX A-2 (continued)

Nutrients, units per 100 g food	Sample(Lab 2) 1[b]	2[c]
Niacin,mg	0.2	0.6
Lipids:		
Percent calories from fat	23	11
Saturated, total	0.09	0.09
14:0	0.03	0.03
16:0	0.06	0.06
18:0	0.00	0.00
Monounsaturated, total	0.08	0.17
16:1	0.04	0.05
18:1	0.04	0.10
20:1	0.00	0.02
Polyunsaturated, total	0.12	0.02
N-3 fatty acids	0.12	0.02
18:3	0.00	0.00
22:6	0.12	0.02
N-6 fatty acids	0.00	0.00

3[d]	4[e]
0.3	0.0
11	16
0.05	0.08
0.01	0.03
0.04	0.05
0.00	0.00
0.16	0.14
0.04	0.04
0.08	0.08
0.04	0.02
0.04	0.04
0.04	0.04
0.00	0.00
0.04	0.04
0.00	0.00

APPENDIX A-2 (continued)

18:2	0.00	0.00	0.00	0.00
Cholesterol, mg	5.2	10.3	3.1	7.1

[a] All 4 samples from Bering Sea beach, Gambell, AK, 1 October 1992. Ascidians are a class of animal in the phylum, chordata, sub-phylum, urochordata, commonly known as tunicates. The class, Ascidiacea, are often called seasquirts. Samples were contributed and identified in Siberian Yup'ik, by Gambell residents. They were collected from the beaches following autumn storms. They also are consumed in the Far East. Due to wide variations in appearance and nutritive value, nutrients in the samples were not averaged.

[b]Kemagluk are 3" diameter solid spheres with a wart-like outer, brown coat or tunic, hence the name "tunicate".

[c]Mamaghwaaq is a colonial ascidian, approximately 2" by 1/4", formed in fingerlike yellow-orange branches.

[d]Riighnak is a simple ascidian, about 2" in diameter with a hard crusted, brown sphere.

[e]Tukughnak, a colonial ascidian averages 2" in diameter, with an amorphous, folded, off-white exterior. Some are orange tinted.

APPENDIX A-2 (continued)

Walrus, skin (*Odobenus Rosmarus*)

Nutrients per 100 g food	Sample(Lab 1) 1[a]
Moisture, g	50
Fat, g	24.0
Lipids:	
Fatty acids, g :	
Saturated, total	2.97
12:0	0.04
14:0	0.70
16:0	1.82
18:0	0.23
Monounsaturated, total	13.62
16:1	6.37
18:1	3.69
20:1	0.44
Polyunsaturated, total	3.11
N-3 fatty acids	2.69

APPENDIX A-2 (continued)

18:3	0.03
20:5	1.42
22:5	0.60
22:6	0.42
N-6 fatty acids	0.42
18:2	0.15
20:4	0.20
Other fatty acids over 0.5% of wet weight	
18:1n7	2.06
Cholesterol, mg	39

[a]Outer 1 inch of skin with fur analyzed from hind quarter. Bering Sea near Kipnuk, AK, 13 April 1990.

APPENDIX A-2 (continued)

Whale, blubber, bowhead, (*Balaena mysticetus*)

Nutrients, units per 100 g food	Sample(Lab 1) 1[a]	2[b]
Moisture, g	55	30
Fat, g	24.7	60.7
Lipids:		
Fatty acids, g :		
Saturated, total	2.90	
12:0	0.02	
14:0	0.81	
16:0	1.67	
18:0	0.32	
Monounsaturated, total	15.12	
16:1	5.05	
18:1	3.32	
20:1	2.90	
Polyunsaturated, total	4.16	
N-3 fatty acids	3.66	
18:3	0.08	

$\bar{X}$
43
42.7

APPENDIX A-2 (continued)

20:5	1.98		
22:5	0.48		
22:6	0.78		
N-6 fatty acids	0.50		
18:2	0.29		
20:4	0.08		
Other fatty acids over 0.5% of wet weight			
22:1	1.73		
Cholesterol, mg	42	39	41

[a]Barrow, AK, late May, 1990.

[b]Skin and blubber exact site on animal not known, 6 x 6 x 3 inches deep. Taken on sea ice about 4 miles north of Point Barrow, AK, 10 May 1990.

APPENDIX A-2 (continued)

Whale, meat, bowhead (*Balaena mysticetus*)

Nutrients per 100 g food	Sample(All Lab 1) 1[a]	2[b]
Moisture, g	68	59
Fat, g	3.36	21.5
Lipids:		
Fatty acids, g :		
Saturated, total	0.87	3.75
12:0	0.0	0.02
14:0	0.17	0.95
16:0	0.56	2.10
18:0	0.12	0.53
Monounsaturated, total	1.61	11.92
16:1	0.52	3.55
18:1	0.63	3.55
20:1	0.19	2.23
Polyunsaturated, total	0.41	4.28
N-3 fatty acids	0.34	3.90
18:3	0.01	0.07

3^c	$\bar{x}$
75	67
1.1	8.7
0.17	1.60
0.0	0.01
0.03	0.38
0.10	0.92
0.04	0.23
0.47	4.67
0.10	1.39
0.17	1.45
0.09	0.84
0.10	1.60
0.08	1.44
0.0	0.03

APPENDIX A-2 (continued)

20:5	0.21	2.24	0.05	0.83
22:5	0.03	0.53	0.01	0.19
22:6	0.07	0.85	0.01	0.31
N-6 fatty acids	0.07	0.38	0.02	0.16
18:2	0.04	0.16	0.01	0.07
20:4	0.02	0.09	0.01	0.04
Other fatty acids over 0.5% of wet weight				
22:1		0.79		
Cholesterol, mg	23	21	9	17.7

[a]Bowhead meat, animal part unknown. Barrow Alaska, late May, 1990.

[b]Bowhead 90B1, skeletal muscle, exact location unknown. Taken on sea ice about 4 miles north of Point Barrow, AK, 9 May 1990.

[c]Bowhead 90B2, skeletal muscle, exact location unknown. Taken on sea ice about 4 miles north of Point Barrow, AK, 10 May 1990. Grams fatty acid/100 g muscle was calculated from normalized percent fatty acid, assuming 75% of fat is fatty acids.

APPENDIX A-3

ENERGY AND FAT VALUES FOR ALASKA NATIVE FOODS USED TO ANALYZE INTAKES OF SIBERIAN YUP'IKS

Food* (Reference number)	Per 100 g				
	Energy (kcal)	Fat (g)	SFA (g)	MUFA (g)	PUFA (g)
Agutuk, berry (1,2) F (3 qt blackberries, 3 c shortening, 1 c water, 1/2 c corn oil, 1 c sugar)	240	17.25	3.98	7.12	5.36
Agutuk, "deer" (reindeer)(3) F (8 c reindeer meat, 1 c beef broth, 1 c seal oil, 1 c caribou fat)	213	23.76	6.39	10.78	4.39
Agutuk, with greens (3,4,5) F (3 qt sourdock, 2 c shortening, 1 c caribou fat, 1 c sugar)	263	24.76	7.38	10.46	5.15

APPENDIX A-3(continued)
Food* (Reference number)

Dried birds (selected guinea hen, etc.) (5) F

Eggs, murre(substituted duck eggs) (6)

Eggs, king salmon roe (3)

Fireweed leaves, raw (*Epilobium latifolium*) (3)

Fish, dried (selected salmon, king, smoked, brined) (3) F

Fish stew
(4 oz salmon, cooked, pink, 1 c chicken broth, 1/4 c macaroni, 1/4 c rice, 2 T onions)

Fry bread (fried bread)

Energy (kcal)	Per 100 g Fat (g)	SFA (g)	MUFA (g)	PUFA (g)
337	14.69	4.87	7.19	1.79
185	13.77	3.68	6.53	1.22
250	14.00	2.04	4.13	4.12
37	0.08	0.00	0.00	0.00
420	30.00	6.97	16.90	4.06
87	2.06	0.58	0.68	0.60
416	24.19	6.04	10.56	6.31

APPENDIX A-3(continued) Food* (Reference number)	Energy (kcal)	Per
Green juice soup (3,5) F (2 c fermented *Sedum rosea* or stonecrop juice, 1/4 c flour, 1/2 c sugar)	108	
Mukluk or Oogruk (bearded seal) (Substituted seal meat, cooked)F	180	
Reindeer (*Rangifer tarandus*)	167	
Salmonberries (*Rubus spectabilis*) (3)	44	
Seal blubber (2)	823	
Seal, dried (3,1,2)	243	
Seaweed, kelp	43	
Stonecrop, leaves (Sedum rosea)(3)	33	
Tunicates or Sea squirts (*Tunicata Ascidiacea*) Siberian Yup'ik names (3):		
Kemagluk	23	
Mamaghwaaq	34	

100 g Fat (g)	SFA (g)	MUFA (g)	PUFA (g)
0.84	0.01	0.01	0.02
3.70	0.50	0.80	0.40
4.42	1.70	1.33	0.62
0.10	0.00	0.00	0.00
90.00	6.20	38.00	15.70
6.60	1.17	2.33	0.90
0.56	0.25	0.10	0.05
1.00	0.00	0.00	0.00
0.60	0.09	0.08	0.12
0.40	0.09	0.17	0.02

APPENDIX A-3(continued)
Food* (Reference number)

Opah

Riighnak

Tukughnak

Walrus blubber (*Odobenus rosmarus*)

Walrus, dried (3) F

Walrus, liver (3,1)

Walrus, skin (mattak) (2,3)

Whale blubber, (*Balaena mysticetus*) (3)

Whale meat, dried, (*Balaena mysticetus*), F

Willow leaves, raw (*Salix pulchra cham.*) (1,2,3)

Wild greens (substituted sourdock) (4) F

Energy (kcal)	Per 100 g Fat (g)	SFA (g)	MUFA (g)	PUFA (g)
82	2.20	**	**	**
41	0.50	0.05	0.16	0.04
28	0.50	0.08	0.14	0.04
746	80.00	6.50	24.60	10.90
267	2.6	0.63	1.17	0.63
130	3.00	0.24	0.92	0.42
171	3.00	0.35	1.64	0.47
870	96.50	9.90	44.25	7.72
329	17.53	3.31	9.16	3.04
102	1.60	0.16	0.08	0.56
20	0.64	0.05	0.04	0.08

APPENDIX A-3 (continued)

*Values calculated by Nutrition Coordinating Center (NCC), University of Minnesota. Nutrition Data System Version 2.6/8a/23 (1994). See Appendix A-2 for foods analyzed by E.D. Nobmann.

F = Food values used in analyzing data from the food frequency questionnaire.

** No data available or known to be comparable.

1) Kuhnlein et al., 1991.

2) Kuhnlein and Soueida, 1992.

3) Nobmann, 1993.

4) USDA Handbook 8-11, 1984.

5) Schakel et al., 1993.

6) USDA Handbook 8-1, 1976.

APPENDIX A-4

ALL FOODS AND SUPPLEMENTS CONSUMED BY SIBERIAN YUP'IKS BASED ON ONE 24 HOUR RECALL, OCTOBER 1992, GAMBELL, ALASKA.

FOOD GROUP	TIMES CONSUMED	PERCENT OF TIMES
Beverages	233	
Coffee, brewed hot	95	9.0
Tea, hot or iced	83	7.9
Fruit flavored beverages	27	2.6
Tea, herbal	12	1.1
Carbonated beverages	9	.9
Cola beverages	5	.5
Gatorade	1	.1
Tap water	1	.1
Breads, cereals and baked products	133	
Crackers, low fat, Pilot bread	56	5.3
Bread, white and whole wheat	18	1.7
Cereals, dry	10	0.9
Fry or fried bread	10	0.8
Cookies, high fat, homemade	10	0.9
Cookies, fig bar	3	0.3
Biscuit, baking powder	3	0.3
Roll, sweet, yeast, basic recipe	3	0.3
Roll, hamburger and hot dog	3	0.3

Fried rice	2	0.2
Rice, white, ckd w/o salt	2	0.2
Cream of wheat	2	0.2
White cake, pudding in mix	2	0.2
Chocolate cake, pudding in mix	1	0.1
Coffee cake, quick bread	1	0.1
Pastry, toaster poptart	1	0.1
Yellow cake, pudding in mix	1	0.1
Doughnut, yeast	1	0.1
Muffin, homemade, plain	1	0.1
Pancake, prepared with water	1	0.1
Rice noodles, ckd w salt	1	0.1
Tortilla, flour, plain	1	0.1
Condiments, herbs and spices	35	
Salt	11	1.0
Catsup, cocktail sauce	7	0.7
Sauce, barbecue, bottled	3	0.3
Garlic, fresh	2	0.2
Salt, seasoning	2	0.2
Basil, ground	2	0.2
Thyme, ground	2	0.2
Pepper, black	1	0.1
Pickle dill	1	0.1
Pickle relish, sweet	1	0.1
Mustard, prepared yellow	1	0.1
Sauce, Chinese gravy	1	0.1
Teriyaki sauce	1	0.1
Eggs	6	
Salmon eggs, king, raw	3	0.3

Duck, whole	1	0.1
Chicken, whole	1	0.1
Omelet, plain	1	0.1
Fats	125	
Margarines:	34	3.2
stick or tub, 80% fat, unknown source, 15.0-15.99 g SFA, 1.6-1.79 P/S ratio	16	1.5
stick or tub, 80% fat, soybean, 15.0-15.99 g SFA, 1.6-1.79 P/S	9	0.9
stick or tub, 80% fat, soybean, 21.0-21.99 g SFA/100 g, 0.2-0.39 P/S	4	0.4
stick or tub, 80% fat, soybean, 11.0-11.9 g SFA/100 g, 0.8-0.99 P/S	1	0.1
table spread, 72% fat, soybean,14.0-14.99 g SFA/100 g, 1.6-1.79 P/S	1	0.1
table spread, 52% fat, soybean, 8.0-8.99 g SFA/100 g, 2.42-2.59 P/S	1	0.1
stick or tub, 80% fat, unknown source, 13.0-13.99 g SFA/100 g, 2.4-2.59 P/S	1	0.1
table spread, 52% fat, unknown source, 8.0-8.99 g SFA/100 g, 2.4-2.59 P/S	1	0.1
Creamer, imitation	22	2.1
Blubber, seal	19	1.8

Whale, skin and fat	15	1.4
Butter, salted	13	1.2
Mayonnaise	9	0.9
Gravy, canned, meat w or w/o milk	4	0.4
Oil, vegetable, composition unknown	3	0.3
Shortening, household	3	0.3
Oil, seal	1	0.1
Oil, soybean, unhydrogenated	1	0.1
Oil, corn	1	0.1
Fruits	26	
Fruit cocktail, swt, cnd or frozen	3	0.3
Peaches, sweetened	3	0.3
Apple, fresh with skin	2	0.2
Apricots, swt, cnd	2	0.2
Blackberries, native, *Empetrum nigrum*	2	0.2
Blueberries, fresh	2	0.2
Pineapple juice, frozen	2	0.2
Pear, fresh and swt, canned	2	0.2
Grape juice, unswt	1	0.1
Lemon juice	1	0.1
Plums, swt, cnd	1	0.1
Raisins	1	0.1
Salmonberries	1	0.1
Meat, Poultry and fish	147	
Chicken, turkey, Cornish game hen (dark meat)	26	2.5

Seal meat, ckd	22	2.1
Walrus meat, ckd	17	1.6
Frankfurter, beef and pork	7	0.7
Fish, Smoked 19.0-22.9% fat (e.g. sable fish)	6	0.6
Whale meat (mallu)	6	0.6
Beef, 12.5-17.4% fat	5	0.5
Tukughnak seafood	5	0.5
Beef, 22.5-27.4% fat	4	0.4
Pork, fresh 27.5-32.4% fat	4	0.4
Pork, smoked 2.5-7.4% fat	3	0.3
Beef, 37.5-42.4% fat	3	0.3
Beef, 7.5-12.4% fat	3	0.3
Fish, 7.0-10.9% fat, ckd (1.0-1.99% fatty acids)	3	0.3
Liver, walrus	3	0.3
Riighnak seafood	3	0.3
Sardines, cnd, drained	3	0.3
Bacon, regular, ckd	2	0.2
Luncheon meat,cnd, ham, etc.	2	0.2
Moose, ckd	2	0.2
Wild duck, ckd	2	0.2
Mamaghwak seafood	2	0.2
Reindeer, ckd	2	0.2
Salmon cake	1	0.1
Beef, 17.5-22.4% fat	1	0.1
Bologna, beef or pork	1	0.1
Ground beef, regular	1	0.1
Fish, 0-2.9% fat ckd	1	0.1
Fish, 3.0-6.9% fat ckd	1	0.1
Fish, 11.0-14.9% fat,ckd	1	0.1

Kemagluk seafood	1	0.1
Oopah seafood	1	0.1
Poultry skin, ckd	1	0.1
Tuna, cnd, oil pack, drained	1	0.1
Walrus, skin (mattak)	1	0.1
Milk	34	
Milk, canned evaporated, whole	13	1.2
Milk, % fat unknown	7	0.7
Cheese, 29-33% fat	5	0.5
Milk, skim	4	0.4
Milk, 1/2% fat	2	0.2
Milk, powdered, nonfat	2	0.2
Ice cream, avg 11% fat	1	0.1
Miscellaneous	5	
Flour, all purpose	3	0.3
Bouillon cube, beef	2	0.2
Mixtures	32	
Macaroni, plain or with cheese	7	0.7
Beef vegetable stew	6	0.6
Corned beef hash	3	0.3
Chowder, clam, New England	3	0.3
Agutuk, fruit	2	0.2
Pizza, cheese and meat	2	0.2
Soup, cream of mushroom	2	0.2
Chop suey, without meat, fish or poultry	1	0.1
Egg roll w shrimp & pork	1	0.1
Fish stew	1	0.1

Soup, meat or vegetable	1	0.1
Spreadables, chicken	1	0.1
Taco, beef	1	0.1
Tomato soup, cnd, undiluted	1	0.1
Nuts	10	
Peanut butter w salt	9	0.9
Almonds	1	0.1
Snacks and Sweets	173	
Sugar	127	12.0
Sugar substitute	21	2.0
Frosting	7	0.7
Potato chips 33.5-37.04% fat	5	0.5
Candy bar, chocolate	3	0.3
Jam, jelly	3	0.3
Syrup, sugar and table	3	0.3
Candy, hard	2	0.2
Candy, chocolate Tootsie Roll	1	0.1
Popcorn, homemade	1	0.1
Vegetables	67	
Potatoes, boiled, pan fried, french fried, mashed	19	1.8
Corn, ear and whole kernel	7	0.7
Stonecrop leaves	7	0.7
Onions, ckd, fresh or raw	6	0.6
Peas, green ckd	5	0.5
Green beans, ckd, fresh or frozen	4	0.4

Fireweed leaves, raw	3	0.3
Broccoli	2	0.2
Carrots	2	0.2
Celery, raw	2	0.2
Vegetable juice cocktail	2	0.2
Cauliflower	1	0.1
Mixed vegetables	1	0.1
Mushrooms	1	0.1
Sauerkraut, ckd, cnd	1	0.1
Spinach, cnd drained	1	0.1
Sprouts, soybean, raw	1	0.1
Tomatoes	1	0.1
Willow leaves, raw	1	0.1
Supplements		
Vitamin A 1 dose = 5000 IU	2	0.2
Thiamin (Vitamin B_1)	2	0.2
Riboflavin (Vitamin B_2) 1 dose = 1.7 mg	2	0.2
Niacin 1 dose = 20 mg	2	0.2
Vitamin B-6 1 dose = 2 mg	2	0.2
Vitamin B-12 1 dose = 6.0 μg	2	0.2
Folate 1 dose = 400 μg	2	0.2
Vitamin C 1 dose = 60 mg	2	0.2
Vitamin D 1 dose = 400 IU	2	0.2
Vitamin E 1 dose = 30 IU	2	0.2
Analgesic, aspirin	1	0.1
B-carotene 1 dose = 2994 μg	1	0.1
Calcium 1 dose = 1000 mg	1	0.1
Pantothenic acid 1 dose =	1	0.1

10 mg Stress Tablet (Stress 1 dose = 1 tablet	1	0.1