Original Article

Dietary intake and the risk of coronary heart disease among the coconut-consuming Minangkabau in West Sumatra, Indonesia

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Several nutrition and non-nutritional pathways are recognised in the development and occurrence of cardiovascular disease. In many populations, high intakes of saturated fat are associated with elevated serum cholesterol concentrations and increased coronary heart disease (CHD) mortality. However, several studies report that hyperlipidaemia and heart diseases are not common among populations who consume coconut, a source of saturated fat. A case-control study was conducted among the Minangkabau known to be high coconut consumers to examine the difference in food patterns and risk of coronary heart disease (CHD) between the coronary cases and their gender- and age-matched apparently healthy counterparts serving as controls. Eligible subjects with CHD were identified through the co-operation of five participating hospitals located in Padang and Bukittinggi in West Sumatra, Indonesia. A total of 93 eligible cases (62 men and 31 women) in the Case group and 189 subjects (113 men and 76 women) in the Control group were recruited. Information on the intakes of individual foods and dishes over the preceding 12 months was obtained using a semi-quantitative food frequency questionnaire. The Case group had significantly higher intakes of meats, eggs, sugar, tea, coffee and fruits, but lower intakes of soy products, rice and cereals compared to the controls. Coconut consumption as flesh or milk was not different between cases and controls. The cases had significantly higher intakes of protein and cholesterol, but lower intake of carbohydrate. Similar intakes of saturated and unsaturated fatty acids between the cases and controls indicated that the consumption of total fat or saturated fat, including that from coconut, was not a predictor for CHD in this food culture. However, the intakes of animal foods, total protein, dietary cholesterol and less plant derived carbohydrates were predictors of CHD.

Key Words: dietary intake, coconut consumption, coronary heart disease, case-control study, Minangkabau, saturated fat, Padang, Bukittinggi, West Sumatra, Indonesia.

Introduction

Epidemiological studies suggest a strong association between coronary heart disease (CHD) and several dietary factors.[1-4] High intakes of saturated fat in different populations are associated with the elevation of serum cholesterol concentrations and the mortality in CHD.[5-7] Experimental and metabolic studies suggest that coconut consumption can cause hyperlipidaemia and atherosclerosis. However, several studies report that hyperlipidaemia and heart diseases are uncommon among high coconut consuming populations.[8-9] Furthermore, most studies to date have assessed Caucasian populations consuming high intakes of total fat and low intakes of fish with the risk of developing CHD.[10] Only few studies are available exploring associations between coronary disease events in Asian populations with low total dietary fat and high fish and coconut intakes.[11] Minangkabau food culture therefore provides a unique opportunity to investigate whether the CHD risk could be predicted by the food patterns. This case-control study was conducted to examine the difference in food patterns and CHD risk between the CHD cases and their gender- and age-matched healthy individuals serving as the controls.

Subjects and methods

The study was conducted from February to August 1999. This research project was approved by the Monash University Standing Committee on Ethics in Research Involving Humans in November 1998 (Project Number 98/458) and acquired a written permission from the local government in West Sumatra, Indonesia. All subjects gave

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written informed consent prior to their participation in the study. All patients diagnosed with CHD less than 6 months were eligible for this study. All patients enrolled in the study had to have received the diagnosis from cardio-logists. The diagnosis followed the World Health Organi-zation criteria which was based on information on typical symptoms, typical changes in electrocardiograph (ECG) or enzymes.[12] Eligible patients were identified through the co-operation of five participating hospitals located in two cities in West Sumatra. The two cities were Padang, which is the capital of West Sumatra province, and Bukittinggi, a more rural county situated on the mountainous area 88 km north of Padang. Subjects in the case groups were recruited from the outpatient clinic of the Cardiovascular Unit in the five hospitals in Padang and Bukittinggi. Subjects in the Control group were recruited from the outpatient Ear, Nose and Throat and the Eyes Clinics from the same hospitals and came from the same areas as the cases. The controls were randomly selected from people matched to the Case subjects on the basis of age and gender. Subjects in the Control group who had health problems related to cardiovascular diseases, such as hypertension and diabetes mellitus, were not included. Pregnant women were also excluded. A total of 93 eligible cases (62 men and 31 women) and 189 subjects (113 men and 76 women) in the Control group were recruited.

A questionnaire on demography, health status, lifestyle and general food habits and practices was developed and administered. Information on the intakes of individual foods and dishes over the past 12 months was obtained using a semi-quantitative food frequency questionnaire. Where an ingredient in a mixed dish or recipe was used in small amounts to add flavours, it was difficult to quantify. For example, coconut milk and grated coconut intakes were assessed in dishes where they were major ingredients. Where small amounts of either coconut milk or grated coconut were used, such as chicken satay or beef rendang, the dishes were included in the estimation of chicken or beef intake, but not in the estimation of coconut milk intake, even though they contained coconut milk (Table 1). When food dishes were categorised as animal or plant foods, this was also judged from the major ingredient(s) of those dishes (Table 2). Nutrient intakes included carbohydrates, proteins, total fat, monounsaturated fat, polyunsaturated fat (n-6 and n-3) and cholesterol. Information on the nutrient content of food items was obtained from the Nutrient Composition of Indonesian Foods and the Nutrient Composition of Malaysian foods.[13] Data on the fatty acid composition of various foods was taken from a USDA Nutrient Database for Standard Reference, www.nal.usda.gov/fnic/cgi-bin/ list_ nut.pl., retrieved on the 27th of February 2001 and used to calculate individual fatty acid intakes (United State Department of Agriculture, 2001).

Subjects whose daily energy intake was implausibly low or high, for example, less than 2,094 kJ (500 kcal) or more than 14,650 kJ (3,500 kcal), were not included in the further data analyses of food or nutrient intakes.[14] Nutrient intakes were presented in actual grams/day and as a percentage of total energy intake.

Statistical analysis

The Statistical Analysis System (SAS software version 6.12 for Windows, SAS Institute Inc., NC, USA, 1996) was used for all data analysis. All data analysis procedures were performed under the SAS/ASSIST. Descriptive statistics were used to report sample distributions and attributes for confounders and antecedent factors. Mean, standard deviation and percentiles were used for continuous variables, whereas for discrete variables, frequency and percentage were derived.

To examine the associations of CHD events with food and nutrient variables, all subjects were divided into four equal groups according to the quartile values of food and nutrient intakes. The odds ratio (OR) was computed as the rate in a specific quartile divided by the value in the group with lowest intake. In multivariate analysis, energy intake, the percentage of energy derived from fat intake, and other potentially confounding variables were simultaneously included into the models.[14]

Results

Food intake

Table 1 shows the mean intakes of each food group for Cases and Controls The cases had significantly higher intakes of meats, eggs, sugar, tea, coffee and fruits, but lower intakes of soy products, rice and cereals compared

Table 1. Descriptive statistics of average daily food consumption (g/day) by food groups for cases and controls

Food groups	*Cases*			*Controls*		
	Mean	±	SD	Mean	±	SD
Fish, seafood and products	67.5	±	33.8	58.4	±	30.8
Meats and meat products	47.1	±	40.2**	34.5	±	28.5
Eggs	37.7	±	31.7**	26.8	±	19.3
Milk and milk products	20.0	±	39.0	17.0	±	30.4
Soy products	90.1	±	82.5	100.7	±	79.5
Legumes and nuts	81.1	±	43.1	77.0	±	54.1
Coconut milk and grated coconut	42.0	±	21.7	38.2	±	18.2
Rice and cereals	382.3	±	108.9***	407.4	±	116.0
Vegetables	338.0	±	146.6	303.4	±	108.4
Fruits	129.9	±	73.7**	114.9	±	69.8
Non alcoholic beverages	131.1	±	105.6**	126.4	±	78.6
Sugar	34.6	±	28.8*	31.6	±	21.0
Palm oil	25.3	±	14.2	23.3	±	10.5

Significantly different from the Control group: *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$.

to the controls. The use of animal fat spreads was not common amongst the Minangkabau. For margarine, only 22% and 7% of the cases and controls respectively used margarine (1.5 teaspoons/d consumed by the cases compared to 0.7 by the controls) *(P*<0.001*)*.

Animal foods

The animal food group included fish, eggs, beef, chicken, and dairy foods. The Case group had significantly higher intakes of total animal foods, compared to the Control group (247 g/d vs 187 g/d, *P<0.0001*). This was mainly due to the difference in the intakes of meat and eggs. Table 2 shows distribution of the Cases and Controls according to animal foods consumed. The odds ratio for subjects who consumed animal foods in the highest quartile (above 210 g) to those in the lowest quartile (below 108 grams) was 4.8 (95% CI 2.25-10.30, *P<0.0001*).

Plant foods

The plant food group included rice and cereal, tempeh and tofu, legumes and nuts, vegetables, fruits and coconut milk and grated coconut. There was no difference in plant food intake between the two groups. Average intake of plant food was 1,061 g/d for the Case group and 1,028 g/d in the Control group.

Macronutrients

Table 3 shows the descriptive statistics of the macronutrients for the Case and Control groups. In total, the Case group had significantly higher intakes of protein and cholesterol, but a lower intake of carbohydrate (*P*<0.0001 for all cases). Between the men, the cases had significantly higher intake of total energy, protein and cholesterol, but a lower intake of carbohydrate (*P*<0.05, *P*<0.01, *P*<0.0001 and *P*<0.001 respectively). Between the women, the cases had a significantly lower intake of carbohydrate (*P*<0.05). As expected, rice was the major food source of total energy intake in this population. Rice and cereals contributed as much as 32% and 36% of total energy for the Case and Control groups, respectively. Other major food sources of total energy were fish, vegetables and soy dishes for both groups.

Amongst the macronutrients included in the univariate logistic regression analysis, intakes of protein and cholesterol were found to be a risk factor for CHD. More subjects in the Case group were in the highest quartiles of protein and total cholesterol (Table 4). The odds ratio for those with intakes of protein and cholesterol in the highest quartile were 1.01 (95% CI 1.01–1.02) and 1.00 (95% CI

Table 2. Distribution of cases and controls according to their animal food consumption

Animal food intake (g/d)	*Cases*		*Controls*	
	n	%	*n*	%
<108.1	14	15.1	57	30.1
108.1 – 149.8	18	19.4	52	27.5
149.8 – 210.4	27	29.0	43	22.8
>210.4	34	36.6	37	19.6

Table 3. Macronutrient intakes of the case and control groups

	Total			Men			Women		
	Mean	±	SD	Mean	±	SD	Mean	±	SD
Cases	n	=	93	n	=	62	n	=	31
Total energy (kcal)	1765	±	534	1823	±	548 *	1647	±	491
Total energy (kJ)	7502	±	2436	7658	±	2303	6893	±	2054
Protein (g)	92.0	±	33.5 **	93.1	±	34.2 **	88.5	±	32.5 ****
% total energy	20.6	±	3.1 ****	20.3	±	3.3 **	21.1	±	2.7 ****
Nutrient density (g/MJ)	12.3	±	1.9	12.1	±	1.9	12.6	±	1.6
Carbohydrates (g)	204.1	±	55.9 ***	213.2	±	58.3 **	186.0	±	46.1 *
% total energy	55.8	±	7.5 ***	56.6	±	7.5 **	55.3	±	7.5 *
Nutrient density (g/MJ)	28.0	±	4.6	28.4	±	4.5	27.8	±	4.5
Total fats (g)	47.2	±	20.9	47.7	±	21.2	46.4	±	20.6
% total energy	23.6	±	5.6	23.1	±	5.5	24.6	±	5.8
Nutrient density (g/MJ)	6.4	±	1.4	6.1	±	1.5	6.5	±	1.5
Dietary cholesterol (mg)	296	±	205 ****	327	±	207 ****	235	±	192
Fibre (g)	10.1	±	4.0	10.1	±	4.1	10.0	±	3.6
Controls	n	=	189	n	=	113	n	=	76
Total energy (kcal)	1657	±	487	1666	±	475	1643	±	507
Total energy (kJ)	6934	±	2038	6971	±	1987	6877	±	2122
Protein (g)	79.3	±	28.2	80.4	±	28.9	77.7	±	27.1
% total energy	19.0	±	2.7	19.1	±	2.8	18.9	±	2.5
Nutrient density (g/MJ)	11.4	±	1.6	11.4	±	1.7	11.3	±	1.5
Carbohydrates (g)	204.7	±	52.9	205.7	±	50.0	203.1	±	57.4
% total energy	57.6	±	6.5	57.7	±	6.7	57.5	±	6.2
Nutrient density (g/MJ)	30.1	±	3.9	30.1	±	40.5	29.9	±	3.7
Total fats (g)	44.0	±	18.6	43.9	±	18.1	44.2	±	19.3
% total energy	23.4	±	4.9	23.2	±	4.8	23.6	±	5.1
Nutrient density (g/MJ)	6.2	±	1.3	6.2	±	1.3	6.3	±	1.4
Dietary cholesterol (mg)	187	±	109	190	±	113	182	±	103
Fibre (g)	9.3	±	3.6	9.2	±	3.4	9.4	±	3.9

Significantly different from the Control group: *, *P*<0.05; **, *P*<0.01; ***, *P*<0.001; ****, *P*<0.0001.

Table 4. Distribution of cases and controls in the highest and lowest quartiles, the odds ratio (95% confidence intervals) according to their protein and cholesterol intakes

	Cases	*Controls*	Odds ratio	95% CI
	n (%)	*n* (%)		
Protein (g/day)				
1st quartile (<61.7 g/day)	16 (17.2)	54 (28.6)	1.01	1.01 – 1.02
4th quartile (>100.2 g/d)	35 (37.6)	36 (19.0)		
Cholesterol (mg/day)				
1st quartile (<125.2 mg/d)	16 (17.2)	55 (29.1)	1.005	1.003 – 1.007
4th quartile (>271 mg/d)	40 (43.0)	30 (15.9)		

1.00–1.01), respectively, compared to those in the lowest quartile.

Dietary fat and fatty acids

Table 5 shows percent energy contribution of dietary fat for the Case and Control groups. The table shows that there were no significant differences in the intakes of saturated and unsaturated fatty acids between the cases and controls, except for arachidonic acid (C20:4). The intakes of individual saturated fatty acids (SFAs) were similar in both groups. Lauric (C12:0), palmitic (C16:0) and myristic (C14:0) acids accounted for 43%, 25% and 17% of total fat intake for both the Case and Control groups. The average intake of marine and plant n-3 fatty acids was 1.9 g/d for the Case group and 1.7 g/d for the Control group.

Table 6 shows the percentage of fat from food groups containing fat. Fish dishes were the major source of total fat in both groups followed by soy dishes, rice and cereal dishes. The amounts of coconut milk and grated coconut were estimated in various dishes with coconut as the major ingredient. Fish and soy dishes were the major food sources for monounsaturated fatty acids (MUFAs). The Case and Control groups were not different in terms of food sources of MUFAs, PUFAs and n-3/n-6 fatty acid ratio. As expected, fish was almost the only source of long chain n-3 in the present study - it accounted for more than 90% of n-3 fatty acid intake in both groups. The cases had a significantly higher intake of arachidonic acid (C20:4) due to the higher consumption of meat and eggs. The proportion of food sources for n-6 fatty acids was similar in both groups. Egg, fish and beef were the most important sources of food for arachidonic acids, while soy, eggs and beef were the most important food sources for linoleic acid.

Multivariate analysis

To determine the predictive power of food and nutrient variables for coronary events, regression of food and nutrient variables was performed. Results are presented in Table 7. Included in this model were food variables, such as animal food intake, and nutrient variables such as total energy, total protein, carbohydrate and cholesterol intake. In addition, other aspects of diet, especially saturated fat intake, and lifestyle factors such as physical activity and stress level, are known to be related to CHD events. These variables were included in the regression analysis. Presented in this table are the partial correlation coefficients for the variables included in the models. Table 7 shows the odds ratio and 95% CI of CHD by food and nutrient intakes. For the total population, higher intake of carbohydrate, higher physical activity, lower animal intake and stress level, were protective against CHD. For men, carbohydrate intake was no longer protective, but lower animal intake and stress, and higher physical activity, were protective. For women, total carbohydrate intake, animal intake and stress levels were predictors for CHD.

Discussion

One major concern in estimating food intake in a case control study is the strong influence of current diet on recall of previous diet. In a case control study by Willet et al.,[14] cases tended to over or underreport past dietary practices. However, the author suggested that diet may be recalled with acceptable levels up to approximately 10 years; beyond this period greater uncertainty exists.

The incidence of CHD in this population could not be explained by the introduction of Western foods, because the influence of Western food in this study population was nearly nil. Although the cases had significantly higher intakes of margarine (1.5 teaspoon/d) compared to the controls (0.7 teaspoon/d), the intake of dairy foods (other than milk) was nil and fast food was 0.7 g/d. In contrast, a study in Japan suggested an association between the increase of Western style fat-rich foods such as butter and margarine, cheese, bread and ham & sausage with an increase of mortality from degenerative diseases.[15]

Total fat intake

The present study did not find any significant differences in dietary fat intake between the Case and Control groups, and there was no relationship with CHD. Although other studies have found positive correlation with CHD,1 total fat intake was not always associated with CHD risk or mortality. Reviewing a wide range of studies, there is little evidence that a high intake of dietary fat predisposes to CHD.[16-19] However, most of such studies were done in populations with high fat intakes. Populations with low fat intakes tended to be at low risk of CHD,[20] and have lower plasma lipid concentrations.[21]

The increased intake of saturated fat in different populations has been confirmed to be associated with the

elevation of serum cholesterol concentrations and CHD mortality.[22-23] It has been suggested that a high intake of coconut oil may contribute to this relationship.[24-25] In contrast, when studies were conducted within populations,[26] it was not always possible to demonstrate significant relationships between intake of saturated fat and the incidence of CHD, as it was shown in the present study. Likewise, it has been difficult to demonstrate significant relationships between saturated fat intake and serum lipid levels in an observational study.[27]

There are several possible explanations for this, such as insufficient precision of the methods used for dietary surveys, large intra-individual variations in food intake, genetic variation with low and high responders to changes in dietary fat, or a small and heterogenous sample with regard to age and gender. Another possibility is that relationships between dietary fat intake and serum lipid levels are more complex than that has been realised hitherto. For example, the source of saturated fatty acids e.g from plant or animal may be important and have

Table 5. % Energy contribution of dietary fat for the Case and Control groups

% Energy	Total			Men			Women		
	Mean	±	SD	Mean	±	SD	Mean	±	SD
Cases									
Total dietary fat	23.6	±	5.6	23.1	±	5.5	24.6	±	4.8
Saturated fatty acids	14.3	±	3.6	14.0	±	2.3	14.5	±	3.3
Short chain fatty acids	1.0	±	0.3	0.9	±	0.3	1.0	±	0.3
Capric acid (C10:0)	0.8	±	0.2	0.7	±	0.2	0.8	±	0.2
Lauric acid (C12:0)	6.1	±	1.7	5.8	±	1.5	6.3	±	1.6
Myristic acid (C14:0)	2.5	±	0.6	2.4	±	0.6	2.5	±	0.6
Palmitic acid (C16:0)	3.5	±	0.9	3.5	±	0.9	3.5	±	0.9
Stearic acid (C18:0)	1.5	±	0.4	1.5	±	0.4	1.4	±	0.4
Monounsaturated fatty acids	5.3	±	1.5	5.2	±	1.5	5.3	±	1.5
Palmitoleic (C 16:1)	0.3	±	0.1	0.3	±	0.1	0.3	±	0.1
Oleic acid (C 18:1)	5.1	±	1.5	5.1	±	1.5	5.2	±	1.6
Polyunsaturated fatty acids	4.4	±	1.7	4.0	±	1.4	4.8	±	1.5
n-6 fatty acids	3.4	±	1.5	3.1	±	1.3	3.8	±	1.4
Linoleic acid (C18:2)	3.3	±	1.5	3.0	±	1.3	3.7	±	1.4
Arachidonic acid (C20:4)	0.06	±	0.03****	0.08	±	0.03****	0.07	±	0.03*
n-3 fatty acids	0.7	±	0.3	1.0	±	0.3	1.1	±	0.3
α-linolenic (C18:3)	0.3	±	0.0	0.3	±	0.1	0.4	±	0.2
Eicosapentaenoic acid (C20:5, EPA)	0.2	±	0.1	0.2	±	0.1	0.2	±	0.1
Docosahexaenoic acid (C22:6, DHA)	0.5	±	0.2	0.5	±	0.2	0.5	±	0.2
n-3/n-6 fatty acid ratio	0.3	±	0.2	0.3	±	0.2	0.3	±	0.1
P:S ratio	0.3	±	0.1	0.3	±	0.1	0.3	±	0.1
Controls									
Total dietary fat	23.4.	±	4.9	23.2	±	4.8	23.6	±	5.1
Saturated fatty acids	13.9	±	3.0	13.8	±	2.9	14.1	±	3.1
Short chain fatty acids	1.0	±	0.3	1.0	±	0.2	1.0	±	0.3
Capric acid (C10:0)	0.8	±	0.2	0.8	±	0.2	0.8	±	0.2
Lauric acid (C12:0)	6.0	±	1.4	6.0	±	1.4	6.0	±	1.4
Myristic acid (C14:0)	2.4	±	0.5	2.4	±	0.5	2.5	±	0.6
Palmitic acid (C16:0)	3.5	±	0.8	3.4	±	0.7	3.6	±	0.8
Stearic acid (C18:0)	1.4	±	0.3	1.4	±	0.3	1.4	±	0.4
Monounsaturated fatty acids	5.1	±	1.1	5.0	±	1.1	5.1	±	1.1
Palmitoleic (C16:1)	0.3	±	0.1	0.3	±	0.1	0.3	±	0.1
Oleic (C18:1)	5.0	±	1.2	5.0	±	1.2	5.1	±	1.2
Polyunsaturated fatty acids	4.4	±	1.4	4.3	±	1.3	4.4	±	1.4
n-6 fatty acids	3.5	±	1.3	3.4	±	1.3	3.6	±	1.3
Linoleic acid (C18:2)	3.4	±	1.3	3.4	±	1.3	3.5	±	1.3
Arachidonic acid (C20:4)	0.06	±	0.02	0.06	±	0.02	0.06	±	0.02
n-3 fatty acids	0.9	±	0.2	0.9	±	0.3	0.9	±	0.3
α-linolenic (C18:3)	0.4	±	0.2	0.3	±	0.1	0.4	±	0.1
Eicosapentaenoic acid (C20:5, EPA)	0.1	±	0.1	0.1	±	0.1	0.1	±	0.0
Docosahexaenoic acid (C22:6, DHA)	0.4	±	0.2	0.4	±	0.2	0.4	±	0.1
n-3/n-6 fatty acid ratio	0.3	±	0.1	0.3	±	0.2	0.3	±	0.1
P:S ratio	0.3	±	0.1	0.3	±	0.1	0.3	±	0.1

Significantly different from the Control group: *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$; ****, $P<0.0001$.

Table 6. Percentage of total fat from food groups

Food groups	*Cases*	*Controls*
	Mean ± SD	Mean ± SD
Fish, seafood and products	26.9 ± 10.7	24.0 ± 11.6
Soy products	18.1 ± 11.9 **	22.1 ± 12.5
Cereal and cereal products	11.8 ± 8.3	12.9 ± 7.5
Meats and meat products	11.5 ± 7.2 **	9.2 ± 6.4
Vegetables	9.8 ± 4.5	10.3 ± 4.3
Eggs	10.2 ± 7.3	9.0 ± 5.3
Nuts and seeds	4.4 ± 5.1	4.4 ± 4.4
Milk and milk products	4.1 ± 8.7	4.5 ± 9.3
Fruits	2.8 ± 2.4	2.8 ± 1.9
Confectionary	0.2 ± 0.7	0.2 ± 0.7
Non alcoholic beverages	0.4 ± 1.2	0.7 ± 1.7

Significantly different from the Control group: **, $P<0.01$.

differing effects on CHD risk.

Subjects in the Case group of the present study were recruited from a well diagnosed group of CHD patients from several hospitals in West Sumatra, and those in the Control group were recruited carefully to match the age and gender of the Cases. The use of validated FFQ may have helped identify similarities between the Case and Control groups in terms of total fat intake, contribution to total energy intake by total fat and individual fatty acids.

Fatty acid intakes

Only limited information is available regarding the association between individual SFAs and the risk of CHD. Experimental studies have found that different classes of SFAs have different effects on plasma lipid and lipoprotein concentrations.[28-29] The differential effects of specific saturated fats on plasma lipids and lipoproteins imply that these fats may have different effects on CHD risk.[3] In the present study, both the cases and the controls had a similar total SFA intake of about 27 g/d, equivalent to 129 g coconut milk or 31.5 g coconut oil. The results do not support an association between total SFA intake and CHD events. Moreover, this finding further suggests that the consumption of coconut products, especially coconut milk or coconut oil, which were the main source of SFAs in the Minangkabau cuisine, does not increase CHD risk, at least in this study population. Results from several studies indicate that diets high in monounsaturated fatty acids have a more favourable effect on serum lipoproteins,[30] and are cardio-protective.[31] However, in this study, no association was found between monounsaturated and polyunsaturated fat and CHD events, although the cases had a significantly higher intake of arachidonic acid than the controls. A similar study was reported with coronary angiographic findings.[32]

Total energy, protein and dietary cholesterol intakes

The present study provides no evidence that total energy intake has implications for CHD, although other studies have found a positive relationship between total energy intake and CHD.[1] A few case-control studies examined the association of CHD with protein intake. Smit *et al.*, (1999) found a significant positive association between CHD with protein intake.[4] Analyses of the association between protein intake and CHD risk are difficult to interpret because they involved simple comparisons of means between cases and non-cases without adjustment for intakes of specific types of dietary fatty acids. In the present study, in univariate analysis, the odds ratio of subjects with total protein intake in the top 25% compared to the lowest 25% was 3.2 (95% CI 1.6–6.6). But after adjusting for other risk factors, total protein failed to enter the model, suggesting that it was not a risk factor for CHD.

Dietary cholesterol intake was associated with an increased risk of CHD in some studies but not others.[33-34] In the present study, the intake of cholesterol was positively associated with CHD in the total population, especially in men, but not in women. In univariate analysis, the odds ratio of subjects with cholesterol intake in the highest quartile, compared to the lowest quartile, was 4.7 (95% CI 2.3–9.7). Dietary cholesterol has been reported to increase liver cholesterol synthesis, resulting in down-regulation of LDL receptor concentrations.[35] The

Table 7. Odds Ratio (95% confidence interval) of coronary events by food and nutrient variables

	Total	Men	Women
Total Carbohydrate	0.7	NA	0.98 **
(highest vs lowest quartile)	*(0.36 – 1.47)*		*(0.97 – 0.99)*
Animal food intake	4.8 ****	5.6 ***	4.7 *
(highest vs lowest quartile)	*(2.25 – 10.30)*	*(1.99 – 16.89)*	*(1.28 – 16.98)*
Physical activity	0.4 **	0.3 *	NA
(highest vs lowest quartile)	*(0.2 – 0.8)*	*(0.1 – 0.7)*	
Stress level	2.9 **	2.8 *	3.6 **
(highest vs lowest quartile)	*(1.6–6.5)*	*(1.2 – 6.3)*	*(1.3 – 10.3)*
Smoke	NA	NA	0.2 **
(highest vs lowest quartile)			*(0.04 – 0.7)*

Variables entered into model include the intakes of animal food, carbohydrate, protein, cholesterol, saturated fat, physical activity, stress level and smoking status; NA: data not available. The variable was removed from the model (the significance level did not reach 0.15). Significantly different from the odds ratio 1.0: *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$; ****, $P<0.0001$.

background quality of dietary intake, bound in Minangkabau people, as with other population, effects serum cholesterol and lipoprotein status.

Food intakes

In general, the intakes of several food groups were different between the cases and the controls. Only total animal intake was found to be independently correlated with CHD events. A higher fish intake (which was also associated with higher n-3 fatty acids), has been recognised to reduce very-low-density lipoproteins, inhibit thromboxane production, increase prostacyclin synthesis, reduce the likelihood of thrombosis, risk of cardiac arrhythmias, and blood viscosity.[10,36] Other populations with higher fish intake, such as the Eskimos and the Japanese, have long been recognised to have a low rate of CHD.[37-38] However, in the present study, it was observed that both the Case and Control groups had a high intake of n-3 fatty acids. Some studies conducted in North America and European countries, where the fish consumption was lower than in the present study, had also found no correlation between fish intake and CHD incidence.[26,39] In this population, coconut (a saturated fat source) is used in the cooking of fish and vegetables. Hence, any potentially adverse effects of coconut-derived saturated fat may be offset by the cardio-protective role of the coconut-associated fish and vegetable intakes in the Minangkabau food culture.

Conclusions

Two conclusions can be drawn from this study. Firstly, intakes of Western foods in this study population were minimal, so that the incidence of CHD cannot possibly be explained by the introduction of Western foods. Secondly, the results from this study showed that the intakes of total fat and saturated fat were not associated with CHD events. Thus, CHD could not be predicted by saturated fat intake in this population. In contrast, intakes of animal food, total protein, dietary cholesterol and total carbohydrate were found to be predictors of CAD.

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