Dietary intake and the risk of coronary heart disease in Japanese men living in Hawaii[1]

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ABSTRACT The relationship of dietary intakes to subsequent development of coronary heart disease (CHD) during a 6-year follow-up period was investigated in 7705 men of Japanese ancestry, ages 45 to 68 and living in Hawaii. Data on the intakes of calories and nutrients were obtained by 24-hr diet recall interviews at the base-line examination. An index for ingestion of traditional Japanese diet (Japanese diet score) was also calculated for each man. The men who subsequently developed myocardial infarction or died of CHD generally ate less than those who remained free of CHD, with statistically significant differences for total calories, total carbohydrate, complex carbohydrate or starch, simple carbohydrate other than sucrose, vegetable protein, alcohol, and Japanese diet score. However, when other major risk factors for CHD were taken into account, the negative association with CHD remained statistically significant only for alcohol and, to a lesser extent, total carbohydrate intakes. The lower total caloric intakes in CHD cases, largely due to decreased alcohol and carbohydrate intakes, could not be accounted for by either under-reporting of food consumption among obese men or diminished physical activity in CHD cases. *Am. J. Clin. Nutr.* 31: 1270–1279, 1978.

During the past two decades the relationship of diet to coronary heart disease (CHD) has been the subject of intensive epidemiological investigation, and abundant data indicating both positive and negative associations of certain dietary factors with the mortality and morbidity of CHD have been reviewed in recent articles (1–5). However, most of the evidence for such associations is based on comparisons of different countries or population groups with substantial diversity in the frequency of CHD and the way of life. Epidemiological studies within homogeneous Caucasian populations so far have consistently failed to show statistically significant associations between individual nutrient intakes and either the level of blood lipids or the risk of CHD within populations (6–12).

Japan has been ranked lowest in both CHD mortality and per capita consumption of saturated fat among the industrialized countries in the world (1), although a substantial increase has been noted in both figures for Japan in recent years (13). In 1957 Gordon (14, 15) reported that the mortality from CHD among Japanese migrants and their descendants in Hawaii and the United States mainland was intermediate between the low level in Japan and the high level among Caucasians in the United States. In 1958 Keys et al. (16) compared CHD prevalence, frequency of severe coronary atherosclerosis, blood lipid levels, and dietary fat among small samples of Japanese men in Japan, Hawaii, and Los Angeles, as well as Caucasians in Hawaii and Minnesota. They found a consistent relationship between the average intake of fat, especially saturated fat, the mean level of blood lipids, and the CHD frequency in these population samples, with an increasing gradient from indigenous Japanese to migrant Japanese to Caucasians. At that time, Larsen (17) also reported on the basis of clinical and ne-

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cropsy studies that the frequency of CHD and the amount of fat in diet were greatest for the Caucasians in Hawaii, intermediate for the Japanese in Hawaii, and lowest for the Japanese in Japan. More recently, Bassett et al. (18) studied hospitalized patients with acute myocardial infarction and sex-age-race matched controls among Hawaiian and Japanese men on the island of Oahu. They found that a strikingly high CHD mortality and morbidity in Polynesian Hawaiians could be attributed to overweight, carbohydrate intolerance, and hypertension, which were more common in Hawaiian men than in Japanese men, whereas the levels of serum cholesterol and triglyceride, cigarette smoking, and physical activity could not account for the interracial difference in CHD frequency. In the same study a detailed analysis of dietary intake suggested that the Hawaiian-Japanese difference in the frequency of CHD might be accounted for by unusual patterns of energy balance (tendency to a sporadically heavy caloric intake and a greater day-to-day variation in caloric intake) in Hawaiians rather than differences in specific nutrient intake (19). The Hawaiians consumed significantly greater amounts of both total and saturated fats than did their Japanese counterparts; yet the average level of serum cholesterol was higher in Japanese men.

Since 1965, collaborative epidemiological studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California have been under way (20). These three study populations were considered to have essentially the same genetic background, yet to differ sufficiently in environmental and behavioral characteristics to allow meaningful inferences about the relation of these characteristics to differences in cardiovascular mortality and morbidity. Dietary habits were among the variables included in these studies, and estimates of nutrient intake based on 24-hr diet recall, as well as the relation of nutrients to the level of serum lipids, have previously been reported (21, 22). The intakes of total and animal protein, total and saturated fat, cholesterol, and simple carbohydrate were greater in the United States Japanese, whereas the intakes of total and complex carbohydrate and alcohol were greater in the residents of Japan (21). The observation was also made that the serum cholesterol level correlated positively (although weakly) with dietary intake of saturated fat, animal protein and cholesterol, and negatively with alcohol and complex carbohydrate intakes within homogeneous cohorts in Japan and Hawaii (22). Similar weak but significant relationships between these nutrients and serum cholesterol levels have recently been reported in an epidemiological study of rural and urban Puerto Rican men (23), although such observations have not been found in other epidemiologic studies. Further, higher rates of CHD mortality (24), prevalence (25), and incidence (26) for Japanese men in Hawaii and California than in Japan have been established; and positive associations of serum cholesterol with the incidence of CHD in the cohorts in Japan and Hawaii have been reported (27). However, the direct relationship between nutrient intakes of individual men and the subsequent risk of developing CHD has not heretofore been explored in any of these Japanese populations. The present report deals with this particular aspect in the cohort of the Honolulu Heart Study.

Methods

The Honolulu Heart Study is a prospective epidemiological investigation of CHD and stroke in a large cohort of men of Japanese ancestry born in the years 1900 to 1919 and living on the island of Oahu in 1965. Of the 11,148 eligible men who were located by updating World War II Selective Service files, 8,006 (72%) participated in the initial examination which was carried out during the years 1965 to 1968 (28). Of these 8006 men, 301 were found to have CHD at the initial examination. The remaining 7705 men free of CHD have been followed for the development of CHD. In this report analyses were made on the basis of 294 new CHD cases ascertained during a 6-year follow-up period. The ascertainment was done by both repeat examinations which were carried out 2 and 6 years after the initial examination, and community surveillance of morbidity and mortality based on periodic review of hospital discharge rosters, death certificates and obituary columns (29). The principal items of the initial examination were social and medical history; inquiries on dietary intake, smoking and drinking habits, as well as on physical activity; questionnaire on chest pain; anthropometric measurements; physical examination of cardiovascular system; blood chemistry including serum cholesterol, uric acid, triglyceride, and glucose which were determined on casual specimens one hour following ingestion of 50 g of glucose; hematocrit; urinalysis; lung function tests; and resting 12-

lead electrocardiogram. The manifestations of CHD in the incidence cases were classified as follows: death attributable to CHD including sudden death; nonfatal myocardial infarction (MI) ascertained by acute episode of chest pain with diagnostic ECG and/or enzyme evidence as obtained from hospital records, or by temporal change of ECG considered diagnostic of interval MI between examinations; acute coronary insufficiency (CI) ascertained by severe chest pain lasting more than 30 minutes, with documented transient ST-T wave changes on ECG and without elevation of enzyme levels; angina pectoris (AP) ascertained by episodic substernal pain brought on by exertion and relieved by rest. More detailed descriptions of the examination methods and the criteria for CHD have been given elsewhere (30).

The dietary information used for the present report was obtained at the initial examination from two sources: a dietary acculturation questionnaire and a 24-hr diet recall interview (21). The dietary acculturation questionnaire was a mailed, self-administered questionnaire designed to ascertain the extent to which a traditional Japanese dietary pattern is followed. The questionnaire listed eight foods considered as indicators of traditional Japanese dietary habits, nine foods as indicators of Americanization, and three foods as neutral or universal ones. Each subject was asked to check food items eaten at meals and snacks during the previous 24 hr. The ratio of the sum frequency of Japanese foods eaten to that of both Japanese and American foods was designated as the Japanese diet score for each subject. A striking difference was noted in the distribution of this score between indigenous Japanese men (68% had scores of 0.80 or greater and only 2% had scores less than 0.50) and Japanese men in the United States (only 4% had scores of 0.80 or greater and 48% had scores less than 0.50). The 24-hr diet recall interviews were conducted by dietitians on all study subjects at the initial examination to obtain quantitative assessment of food intake during the previous 24 hr. Food models and serving utensils were used to illustrate portion sizes, and food composition values were compiled from the best available sources to calculate the individual intakes of calories and nutrients (21). Dietary data were validated by 7-day dietary records in a subsample of the men examined 2 years later. Reproducibility was modest, though statistically significant, for most of the nutrients, with correlation coefficients ranging from 0.4 to 0.6 (21). Dietary methodology studies carried out in the past (31–33) have indicated that the shorter, more expedient 24-hr dietary recall method can be substituted for the more time-consuming 7-day dietary record collection procedures when an estimate of the mean intake of a group of roughly 50 persons or more is desired. It was the method of choice in the present study in which large numbers of men of varying literacy levels were to be interviewed quickly and comparably. This method has been used by most other present day large-scale dietary studies, such as those carried out by the United States Department of Agriculture in 1965 (34), the Ten State Nutrition Survey of 1968 to 1970 (35), and the First Health and Nutrition Examination Survey, 1971 to 1972 (36).

The relationship between the intake of individual nutrients and the incidence of CHD was first evaluated by comparing the age-adjusted means of dietary intake reported at the initial examination between men who developed CHD and those who remained free of CHD ("non-cases") during the 6-year follow-up period. Age adjustment was done by weighting the age-specific mean values according to the proportion of the total population at risk made up by that age group. The dietary variables with a substantial difference in mean value between CHD cases and non-cases were further subjected to a multivariate analysis using a logistic model for their contributions to the prediction of CHD risk, independent of other confounding risk factors. The logistic coefficients were estimated by the maximum likelihood method of Walker and Duncan (37). Significance of logistic coefficients was examined by the t statistic calculated by dividing each logistic coefficient by its standard error. Relative strengths of independent variables as risk factors for CHD were roughly compared by standardized coefficients, which were calculated by multiplying each coefficient by the standard deviation of that variable. A larger standardized coefficient represents a stronger risk factor.

Results

During the period of 6-year follow-up of 7705 men who had been free of CHD at the time of initial examination, a total of 294 new cases of definite CHD were ascertained by two repeat examinations and by comprehensive surveillance of morbidity and mortality. These CHD cases included 43 deaths due to CHD, 136 cases of MI, 27 cases of CI, and 88 cases of AP. In those men who experienced more than one type of CHD, the first manifestation was used for classification. In the present analysis, the CHD death and nonfatal MI groups were combined because both are generally regarded as "hard" cases of CHD, and nearly 80% of CHD deaths in this study population were due to acute MI or coronary occlusion.

Table 1 shows age-adjusted mean values of total calories and specific nutrients as determined by 24-hour diet recall at the initial examination, comparing CHD cases and non-cases. Men in the CHD death + MI group had significantly smaller intakes of total calories, total carbohydrate, starch, simple carbohydrate other than sucrose, and alcohol than did men remaining free of CHD. There was no significant difference in the average intake of any dietary variables between non-cases and either the CI or AP group.

In Table 2 age-adjusted mean values of the proportion of calories derived from specific nutrients are shown. There was a statis-

tically significant excess in the proportion of calories derived from protein, total fat, saturated fatty acids, and polyunsaturated fatty acids in the CHD death + MI group compared with those in non-cases. However, actual differences were very small (0.6 to 2.2% of total calories), and the absolute amount of intake of these nutrients (except polyunsaturated fatty acids) was greater in non-cases than in the CHD death + MI group (Table 1). Therefore, these findings ought to be interpreted with caution. On the other hand, alcohol provided a larger proportion of calories in non-cases than in the CHD death + MI group, with a highly significant difference. This is in accord with the difference in the absolute amount of alcohol intake. None of the differences in proportion of calories derived from specific nutrients between non-cases and either the CI or AP group reached the level of statistical significance, except for polyunsaturated fatty acids which showed a small excess in the AP group.

Table 3 shows age-adjusted mean values of the Japanese diet score and of nutrient intakes by food group. Intakes of vegetable protein, complex carbohydrate and alcohol, as well as the Japanese diet score, were significantly larger in non-cases than in the CHD death + MI group. Again, no significant difference was demonstrated between non-cases and either the CI or AP group except for the intake of vegetable protein, which was significantly larger in non-cases than in the CI group.

From the foregoing it can be stated that differences in dietary pattern, if any, between non-cases and CHD were demonstrable only in hard cases of CHD, and that the men who subsequently developed MI or died of CHD had reported smaller intakes of total calories, carbohydrates other than sucrose, and alcohol than those who remained free of CHD. The lower Japanese diet score suggests a choice of more American type of food items in the CHD death + MI group than in non-cases, though this was not substantiated by the average intakes of saturated fatty acids, sucrose, and cholesterol in the 24-hr diet recall data.

The role of these dietary variables in predicting CHD death and MI independent of the known major CHD risk factors was

TABLE 1
Age-adjusted[a] mean 24-hr intake of individual nutrients for CHD cases and non-cases[b]

Dietary variable	Non-cases (7411 Men)		CHD death + MI (179 Men)			Acute CI (27 Men)			Angina (88 Men)		
	Mean	SD	Mean	SD		Mean	SD		Mean	SD	
Total calories	2290	738	2125	666	***[d]	2297	773	NS	2270	733	NS
Total protein (g)	94	36	92	33	NS[c]	92	36	NS	98	37	NS
Total fat (g)[e]	86	39	85	37	NS	80	39	NS	87	37	NS
SFA (g)[e]	32	16	31	15	NS	30	17	NS	31	14	NS
MFA (g)[e]	33	16	32	15	NS	30	15	NS	32	14	NS
PFA (g)[e]	15	10	16	9	NS	14	8	NS	17	10	NS
Total carbohydrate (g)	262	97	240	91	**	260	104	NS	258	91	NS
Starch (g)	165	73	150	69	**	150	77	NS	167	69	NS
Sucrose, refined (g)	45	37	45	37	NS	46	38	NS	41	32	NS
Other simple carbohydrate (g)	52	36	45	28	**	64	39	NS	50	37	NS
Alcohol (g)	14	31	6	16	***	23	33	NS	9	23	NS
Cholesterol (mg)	549	318	521	293	NS	557	330	NS	587	312	NS
Caffeine (mg)	436	387	460	385	NS	535	300	NS	508	419	NS
P/S ratio[e]	0.54	0.32	0.56	0.32	NS	0.57	0.28	NS	0.61	0.36	NS

[a] Adjusted to the entire population at risk in 5-year age groups by the direct method. [b] Men who remained free of CHD during the 6-year follow-up period. [c] NS = not significant; SFA = saturated fatty acids; MFA = mono-unsaturated fatty acids; PFA = polyunsaturated fatty acids. [d] *, **, *** indicate significant differences by *t* test from the mean for non-cases at $P < 0.05$, 0.01, and 0.001, respectively. [e] Ratio of PFA/SFA.

TABLE 2
Age-adjusted[a] means of proportions of calories derived from specific nutrients for CHD cases and non-cases[b]

Dietary variable	Non-cases (7411 Men) Mean	SD	CHD death + MI (179 Men) Mean	SD		Acute CI (27 Men) Mean	SD		Angina (88 Men) Mean	SD	
% Cal as protein	17	4	17	4	*[c,d]	16	4	NS	17	4	NS
% Cal as fat	33	9	35	9	**	31	11	NS	34	9	NS
% Cal as SFA[e]	12	4	13	4	*	12	5	NS	12	4	NS
% Cal as PFA[e]	6	3	7	3	*	6	2	NS	7	3	*
% Cal as carbohydrate	46	11	46	10	NS[c]	46	10	NS	46	10	NS
% Cal as starch	29	12	28	10	NS	27	11	NS	30	9	NS
% Cal as sucrose	8	6	8	6	NS	8	5	NS	7	5	NS
% Cal as alcohol	4	8	2	5	***	7	10	NS	3	6	NS

[a] Adjusted to the entire population at risk in 5-year age groups by the direct method. [b] Men who remained free of CHD during the 6-year follow-up period. [c] Without rounding, the means for non-cases and CHD death + MI group were 16.6 and 17.4, respectively. [d] *, **,*** indicate significant differences by t test from the mean for non-cases at $P < 0.05$, 0.01, and 0.001, respectively. [e] NS = not significant; SFA = saturated fatty acids; PFA = polyunsaturated fatty acids.

TABLE 3
Age-adjusted[a] means of nutrients by food group and of Japanese diet score for CHD cases and non-cases[b]

Dietary variable	Non-cases (7411 Men) Mean	SD	CHD death + MI (179 Men) Mean	SD		Acute CI (27 Men) Mean	SD		Angina (88 Men) Mean	SD	
g											
Animal protein	71	33	70	30	NS[c]	72	29	NS	73	34	NS
Vegetable protein	24	10	22	10	*[d]	20	10	*	24	10	NS
Saturated fat	60	33	56	31	NS	58	34	NS	56	27	NS
Unsaturated fat	26	20	28	19	NS	21	18	NS	30	20	NS
Simple carbohydrate	92	55	86	51	NS	106	57	NS	86	44	NS
Complex carbohydrate	170	74	154	70	**	154	77	NS	172	70	NS
Alcohol	14	31	6	16	***	23	33	NS	9	23	NS
Japanese diet score[e]	49	18	45	19	*	44	20	NS	48	19	NS

[a] Adjusted to the entire population at risk in 5-year age groups by the direct method. [b] Men who remained free of CHD during the 6-year follow-up period. [c] Not significant. [d] *, **, *** indicate significant differences by t test from the mean for non-cases at $P < 0.05$, 0.01, and 0.001, respectively. [e] Ratio of (Japanese foods/sum of Japanese foods and American foods eaten during 24 hours) × 100.

evaluated by multivariate analysis using a logistic model. Several combinations of diet variables were tried. In all of these, alcohol intake was found to be an important and significant (negative) predictor of CHD. When age, systolic blood pressure, serum cholesterol, relative weight, cigarette smoking, and alcohol intake were taken into account, total carbohydrate intake was the only additional diet variable that was significantly related (negatively) to CHD death + MI. These results are shown in Table 4. The magnitude of the standardized coefficients indicates that while alcohol consumption was as important a predictor as any of the major CHD risk factors in this cohort, total carbohydrate intake makes a weaker contribution. The smaller caloric intake of the CHD death + MI group was largely due to decreased amounts of alcohol and carbohydrate, so that total calories per se did not contribute to the risk function when these other variables were included.

Discussion

Based on international comparisons involving seven countries, Keys (38) reported that remarkably high correlations were found between average saturated fat intakes as percentage of total calories and either average serum cholesterol levels or CHD incidence among the populations studied. On the other hand, no significant association was demonstrated between dietary variables and either serum cholesterol levels or

CHD incidence within cohorts of the Framingham Study (7), the Israel Ischemic Heart Disease Study (8–10), and the Tecumseh Study (11, 12). Keys (39) discussed in detail possible factors which could account for this discrepancy, and concluded, "Within a culturally homogeneous population it is fruitless to attempt to characterize with any reliablity the individuals in respect to the nutrient variables relevant to serum lipids, atherosclerosis, and coronary heart disease. This conclusion refers to methods used so far, but the methods are not responsible; the unescapable limitation is the spontaneous variability of the individuals themselves."

Migrant Japanese men in Hawaii and California surpassed indigenous Japanese men in average intakes of saturated fat, animal protein, and cholesterol, as well as in average serum cholesterol levels and in CHD morbidity and mortality. However, a previous analysis (22) indicated that dietary variables could account for only a small part of the variance in serum cholesterol levels. Furthermore, no positive association was demonstrated between the risk of CHD and these dietary variables in the present study, although the level of serum cholesterol was identified as an important risk factor for CHD in the same population (30). These inconsistent findings with respect to diet-serum cholesterol-CHD relationships may be attributed, as Keys suggested, mainly to intrinsic variation among individuals in serum cholesterol levels, as well as large intra-individual variability in nutrient intake and, additionally, to the limited method (single interview of 24-hr diet recall) used to estimate year-round habitual dietary intakes (40).

All of these factors add to the error of nutrient estimates and make it more difficult to demonstrate underlying relationships which may be present. In other words, random error in estimates of the nutrient intake, as might be expected to occur when a 24-hr recall is obtained, would ordinarily lead to underestimates of any true relation between the nutrients and CHD (41–43). Given the nature of our data, negative findings cannot be considered definitive. The finding of weak relationships may suggest stronger true associations.

Another factor possibly accounting for the weak relation between diet and serum cholesterol or CHD is that recent dietary habits might not represent the lifetime dietary pattern. Considering the long course of the atherosclerotic process, it would be misleading to relate the recent dietary practice to the risk of CHD if the diet was substantially different in the past. In the case of the Honolulu Heart Study, a specific inquiry was made at the initial examination about individuals' general dietary patterns (Oriental, Western, or mixture) at that time and around 1940. It was found that the proportion of men who considered their dietary pattern as Oriental diminished strikingly between 1940 (48%) and 1965 (14%), whereas the proportion of men with Western diet was essentially unchanged (13% in 1940 and 14% in 1965), and those with a mixed diet increased from 39% to 72% during the same period.

Thus, the dietary pattern among Japanese men living in Hawaii has been conspicuously changing toward less traditional and more Westernized patterns although their recent diet is still predominantly a mixture of Oriental and Western foods. These findings and other observations (44) suggest the need for cautious interpretation of data relating only the recent diet to CHD in this population.

TABLE 4
Standardized coefficients[a] for selected independent variables in multivariate logistic functions[b] in prediction of risk for CHD death and myocardial infarction[c]

Independent variable	Standardized Coefficient	Significance level[d]
Age (yr)	0.292	***
Systolic blood pressure (mmHg)	0.434	***
Serum cholesterol (mg/dl)	0.202	**
Relative weight (%)	0.248	**
Cigarettes per day	0.461	***
Alcohol intake (g/24 hr)	−0.621	***
Carbohydrate intake (g/24 hr)	−0.191	*

[a] Logistic coefficient times standard deviation of each variable. [b] Logistic coefficients were estimated by the maximum likelihood method of Walker and Duncan (29). [c] The data were based on 175 cases of CHD death + MI and 7290 men without CHD for whom all necessary information was available. [d] *, **, *** indicate logistic coefficients significantly different from zero at $P < 0.05$, 0.01, and 0.001, respectively.

A rather unexpected finding in the present analysis was that men going on to CHD death or MI reported eating less than the non-cases, with a substantial difference in intakes of total calories, carbohydrates except sucrose, and alcohol. Also, in a pathological study based on 226 autopsied men of the Honolulu Heart Study, dietary calories and starch intake were significantly and inversely related to the degree of coronary atherosclerosis (44a). Very similar findings have been observed by Bassett et al. (19) in a case-control study of Hawaiian and Japanese men hospitalized for myocardial infarction. The intakes of total calories and of almost all nutrients were substantially lower in cases than in controls in both ethnic groups. The authors emphasized the relatively low caloric intake in the Hawaiians, who were much more overweight than the Japanese, but did not refer to the difference between cases and controls. Similar findings have also been reported by Little et al. in a case-control study of Canadian men surviving myocardial infarction (45). They found that total calories and total carbohydrate intakes were lower in cases than in controls consistently from the fourth to the eighth decades of age, with statistically significant differences in the fourth and seventh decades.

In case-control studies the lower caloric intake in the cases might result from the influence of CHD or its treatment on the reported diet. This bias is largely avoided in a prospective study like the present one. However, lower intakes of nutrients in CHD cases might result from selective under-reporting, according to the following hypothesis: obese persons tend to underreport their dietary intakes consciously or unconsciously; CHD cases include a larger proporation of obese persons; so, average nutrient intakes in CHD cases appear lower than actual values. In order to test the validity of this hypothesis, age-adjusted mean values of total calories were compared between the CHD death + MI group and non-cases within the same strata of relative weight. The relative weight (%) was calculated as the subject's weight/ideal weight × 100, where the ideal weights were defined at each height in a manner comparable to Metropolitan Life Insurance Company ideal weights (46). As shown in Table 5, age-adjusted mean values of total calories were consistently lower in CHD cases than in non-cases in all classes of relative weight with a statistically significant difference in the largest relative weight class. There was no trend of decreased reported caloric intake with increased relative weight except in the heaviest group of CHD cases. Therefore, this hypothesis cannot account for the lower total calories in the CHD death + MI group.

Another possible explanation is that the men who will develop CHD eat fewer calories because they are less physically active. An index of physical activity, representing the weighted sum of the hours spent at various levels of physical activity in a usual day, after the method used in the Framingham Study (47), was obtained at the initial examination. It was lower in CHD cases than in non-cases ($P<0.05$), although the difference became statistically nonsignificant in multivariate analysis. There was a

TABLE 5
Age-adjusted[a] mean values of total calories according to relative weight for CHD death + MI cases and non-cases[b]

Relative weight	Non-cases			CHD death + MI		
	No. of Men[c]	Total calories		No. of Men	Total calories	
		Mean	SE		Mean	SE
%						
Less than 110	3206	2287	12	45	2185	106
110–119	1965	2321	16	64	2261	83
120–129	1418	2264	19	40	2173	120
130 or greater	816	2281	28	30	1796[d]	140

[a] Adjusted to the entire population at risk in 5-year age groups by the direct method. [b] Men who remained free of CHD during the 6-year follow-up period. [c] Six men were excluded because of missing data. [d] Indicates significant difference by t test from the mean for non-cases at $P < 0.001$.

consistent trend of diminishing caloric intake with decreasing physical activity index in both CHD cases and non-cases. However, the age-adjusted mean caloric intake was significantly smaller in CHD cases than in non-cases, even after controlling for physical activity index.

As noted above, in multivariate analysis alcohol and total carbohydrate intakes were significant (negative) predictors of CHD death + MI, and after they and other variables were taken into account, caloric intake did not appear to be important. The strong inverse relationship between alcohol intake and subsequent CHD in this population was also found in a separate analysis based on the usual alcohol consumption (ounces per month) which was recorded independently of 24-hr diet recalls during the initial examination (48). The apparent protective effect of moderate alcohol consumption on risk of MI and CHD death may be mediated through its relation to lipoprotein fractions in plasma. Alcohol use has been shown to be positively correlated with high density lipoprotein cholesterol levels in plasma, and inversely correlated with low density lipoprotein cholesterol levels (49). Increased high density lipoproteins and decreased low density lipoproteins levels would be expected to have a protective effect against CHD (50).

There is moderate intercorrelation among the other nutrients, so that it is not surprising that only one or two should remain significant in multivariate analysis. However, it is not certain why, among the variables considered, total carbohydrate was the one which persisted. Total carbohydrate intake may be an indication of how Japanese the overall diet is. Sucrose intake did not differ in the CHD cases and non-cases, and the largest part of the difference in carbohydrate intake was due to starch. The rice and bread eaten in Hawaii, as main sources of starch, are mostly highly refined grain products and are presumably of low fiber content. We are not aware of any other hypotheses linking carbohydrate intake per se to lower CHD rates, although, in general, societies with high carbohydrate intakes (and low fat intakes) have low rates for CHD.

In summary, there do appear to be dietary differences between men remaining free of CHD and men going on to CHD death or MI during a 6-year period of follow-up in this population of Japanese ancestry. Besides the unquestionably low intake of alcohol, there was a decreased intake of total carbohydrate, mainly from starch, in the CHD group. Considering the amount of error which is inherent in 24-hr diet recall data, the true difference may be somewhat greater than that measured. However, this association does not mean that carbohydrate intake per se is protective against CHD. Some unmeasured characteristics may be associated with both CHD risk and carbohydrate intake. Also, the results of this study do not rule out a possible role of saturated fat and other nutrients characteristic of Western diet as risk factors for CHD, since there had been a substantial change in dietary pattern in this Japanese population during the 25 years preceding the beginning of this study.

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