

Dietary and plasma lipid, lipoprotein, and apolipoprotein profiles among elderly Hispanics and non-Hispanics and their association with diabetes¹⁻³

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ABSTRACT

Background: There are limited data about dietary intakes and plasma lipids of elderly US Hispanics.

Objective: The disparity in prevalence of type 2 diabetes among population groups underscored our need to assess dietary and plasma risk factors for cardiovascular disease.

Design: Plasma lipids and apolipoproteins and dietary intakes of macronutrients were measured in elderly subjects (60–98 y): 490 Hispanics of Caribbean origin (Puerto Ricans and Dominicans) and 163 non-Hispanic whites. Plasma values were related to ethnicity and to macronutrient intake. Differences in plasma lipids due to diabetes were assessed among the Hispanics.

Results: Intakes of carbohydrate and polyunsaturated fatty acids were higher and intakes of cholesterol and saturated and monounsaturated fatty acids were lower in Hispanics than in non-Hispanic whites. Concentrations of total cholesterol, HDL cholesterol, and apolipoprotein A-I were significantly lower among Hispanic women than among non-Hispanic white women; a similar trend was seen in men. Dyslipidemia (high triacylglycerols and low HDL cholesterol) was more prevalent among Hispanics with than without diabetes.

Conclusions: Ethnic differences in serum lipids exist and appear to be associated with differences in dietary intakes. However, both Hispanics and non-Hispanic whites had lipid profiles indicating a high risk of cardiovascular disease. Hispanics with diabetes were at higher risk of dyslipidemia than were those without diabetes. Our data suggest that lifestyle changes, including diet modification and exercise, could be of significant benefit to both ethnic groups. *Am J Clin Nutr* 2002;76:1214–21.

KEY WORDS Plasma lipids, lipoproteins, apolipoproteins, lipoprotein(a) cholesterol, macronutrients, elderly, Hispanics, Puerto Ricans, Dominicans, type 2 diabetes, Massachusetts Hispanic Elders Study

INTRODUCTION

Although Hispanic Americans will be the largest minority group in the United States by the year 2050 (1), there have been relatively few studies addressing the magnitude and severity of cardiovascular disease (CVD) and the effects of risk factors on the cardiovascular health of this population. Limited data suggest that elderly Hispanics have a lower overall mortality (2, 3) and lower prevalence of coronary heart disease (CHD) than do elderly non-Hispanic whites (4). The lipid profile of Mexican Americans,

compared with non-Hispanic whites, has been characterized by lower plasma concentrations of total cholesterol (TC) and higher concentrations of plasma triacylglycerols (5–9). However, little is known about the dietary and plasma lipid profiles of Caribbean Hispanics residing in the United States, where the largest groups are of Puerto Rican and Dominican origin.

The role of lipoproteins and apolipoproteins in atherogenesis has been clearly documented (10). An inverse relation between HDL cholesterol and the incidence of CHD has been shown (11). Concentrations of plasma LDL cholesterol are highly correlated with the prevalence of CHD (12). Lower plasma apolipoprotein A-I (apo A-I) concentrations in men and higher apolipoprotein B (apo B) concentrations in women have also been identified as independent risk factors for CHD in the non-Hispanic white population (13). However, for minority groups such as elderly Puerto Ricans, information on CVD risk factors is limited (14). Elevated plasma lipoprotein(a) [Lp(a)] cholesterol has been shown to be an independent CHD risk factor in men, with a relative risk of > 2 (15). However, we know of no published information on plasma concentrations of Lp(a) cholesterol among Hispanic elderly of Caribbean origin.

The prevalence of diabetes has rapidly increased over the past decade (16), due in part to the aging of the population, obesity, and unsound dietary habits. The higher prevalence of type 2 diabetes in Hispanic groups than in the general population has been widely documented (16–20). Among Puerto Ricans aged > 60 y, we found a prevalence of diabetes of > 40% for women and ≈ 36% for men (21). Conditions associated with type 2 diabetes mellitus such as insulin resistance, hyperinsulinemia, and hyperglycemia play an important role in the occurrence of CVD (22). However, there is a lack of information about the role of dietary and plasma lipids as CVD risk factors in elderly Hispanics of Caribbean origin with type 2 diabetes.

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In the present study we compared the effects of ethnicity and diet on plasma concentrations of lipids, lipoproteins, and apolipoproteins between a representative sample of elderly Hispanics of Caribbean origin and of elderly non-Hispanic whites from the same neighborhood. In addition, we examined dietary intakes, patterns of dyslipidemia, and other lipid risk factors in elderly Hispanics with type 2 diabetes.

SUBJECTS AND METHODS

Study sample

A 2-stage random sampling method was used to interview a representative sample of elderly (≥ 60 y) Hispanics ($n = 779$) living in Massachusetts and a neighborhood comparison group of elderly non-Hispanic whites ($n = 251$) for the Massachusetts Hispanic Elders Study. In the present study, we selected 490 Hispanics of Caribbean origin (Puerto Ricans and Dominicans) and 163 non-Hispanic whites. Plasma lipid and dietary data were available for all subjects; $\approx 80\%$ of the subjects (372 Hispanics and 132 non-Hispanic whites) had fasting plasma samples. The Human Investigation Review Board of Tufts University, New England Medical Center, approved the protocol, and subjects gave informed consent before participating.

Field data collection

Sociodemographic, health, and nutrition-related data were collected between 1993 and 1997 at the subjects' homes. Dietary data were collected with use of a semiquantitative food-frequency questionnaire, specially adapted for this population, with open-ended questions for portion sizes (23). Blood samples were drawn during a subsequent visit.

Fasting (12 h) blood samples were collected in tubes containing 0.15% EDTA and centrifuged at $1000 \times g$ for 20 min at 40°C to separate plasma. HDL cholesterol was measured in plasma after precipitation of apo B-containing lipoproteins with dextran sulfate-MgCl₂ (24). Plasma TC, triacylglycerol, and HDL cholesterol were measured by enzymatic methods with an automated analyzer (CCX analyzer; Abbott Diagnostics Spectrum, Irving, TX) and Abbott enzymatic reagents (25). All lipid assays were performed in duplicate, and the CVs within and between runs were 2–5%. LDL-cholesterol concentrations were calculated with use of the Friedewald formula: LDL cholesterol = TC – (triacylglycerol/5 + HDL cholesterol) (26). Plasma apo A-I and apo B were measured with a commercially available immunoturbidimetric assay (Spectrum CCX analyzer; Abbott Diagnostics Spectrum) with reagents and calibrators from Incstar Corporation (Stillwater, MN) (27, 28). The CVs between and within runs were $< 5\%$ for apo A-I and $< 10\%$ for apo B. Lp(a)-cholesterol concentrations were measured with the Lipopro Lp(a) kit (Genzyme Diagnostics, Cambridge, MA) according to the directions in the package insert (29, 30). Nonfasting blood samples were collected from subjects that were unable to fast, and the same procedures were followed for measuring plasma values.

A diagnosis of type 2 diabetes was based on current recommendations (31), which include a fasting glucose concentration ≥ 7.0 mmol/L (≥ 126 mg/dL) or a random glucose concentration ≥ 11.1 mmol/L (≥ 200 mg/dL) or the use of medications for diabetes (insulin or other drugs). We excluded one subject with type 1 diabetes with an age of onset of 13 y. All selected subjects had an age of onset of type 2 diabetes of > 25 y of age.

Anthropometric measurements of stature and knee height were taken with a Harpenden pocket stadiometer (Holtain Ltd, Crosswell, United Kingdom). Weight was taken with a Seca balance scale (Seca Corporation, Columbia, MD) with a capacity of 150 kg. Waist circumference was measured with a nonstretchable measuring tape, at the level of the smallest area of the waist, to the nearest 0.1 cm.

Information on age, years of education, income, physical activity, and alcohol and tobacco use was collected by questionnaire. Medication use was verified by observing actual medications at the subjects' homes. We measured overall physical activity with a modified version of the Harvard Alumni Physical Activity Questionnaire (32), which included questions about the number of hours per day (mean of a regular weekday and a regular weekend day) spent sleeping and in sedentary, light, moderate, and vigorous activities; the interviewer ensured that the total time added up to 24 h.

Data analysis

Nutrient analyses were performed by using values from the Minnesota Nutrient Data System (program 2.8, version 25; University of Minnesota, Minneapolis). We excluded 17 subjects (10 Hispanic women, 5 Hispanic men, and 2 non-Hispanic white men) identified as outliers on the basis of daily total energy intakes < 2510 kJ (600 kcal) or $> 16\,736$ kJ (4000 kcal). Data are presented as the contribution to total energy intake from total carbohydrates, total fat [monounsaturated (MUFA), saturated (SFA), and polyunsaturated (PUFA) fatty acids], and alcohol. Cholesterol (mg) intake was also evaluated.

A physical activity score was calculated by multiplying the time spent in different activities by corresponding weighting factors that parallel the increased rate of oxygen consumption associated with increasingly more intense physical activity; the procedure used in the Framingham Offspring Study (33, 34) was followed. Smoking was dichotomized as current smokers and nonsmokers. For subjects unable to stand or with a stooped posture, stature was estimated from knee height with the use of equations developed for these Hispanic subjects (35) and existing published equations for non-Hispanic whites (36). Body mass index (BMI; in kg/m²) and waist circumference (cm) were used as linear variables.

We used the cutoffs recommended by the National Cholesterol Education Program (37) to define high triacylglycerol (≥ 2.3 mmol/L, or ≥ 200 mg/dL), high TC (≥ 6.2 mmol/L, or ≥ 240 mg/dL), low HDL cholesterol (≤ 1.0 mmol/L, or ≤ 40 mg/dL), and high LDL cholesterol (≥ 4.13 mmol/L, or ≥ 160 mg/dL). High Lp(a) cholesterol was defined as a concentration ≥ 0.26 mmol/L (≥ 10 mg/dL), as used previously (15). Dyslipidemia was defined as a high triacylglycerol concentration, a low HDL-cholesterol concentration, or both.

The Statistical Package for the Social Sciences (SPSS for WINDOWS, release 10.0; SPSS Inc, Chicago) was used for the statistical analyses. Comparative analyses were conducted by using the general linear models procedure (SPSS Inc). We used logistic regression to compare across ethnic or diabetes groups. The association of ethnicity with plasma concentrations of triacylglycerol, HDL cholesterol, and LDL cholesterol was tested with multiple regression models, with control for socioeconomic status. We also tested interactions between age, sex, ethnicity (Hispanic or non-Hispanic; Puerto Rican or Dominican), and diabetes, with TC and triacylglycerol as dependent variables. These interactions were not significant at $P < 0.05$.



TABLE 1

Descriptive characteristics, by sex, of elderly Hispanics of Caribbean origin and non-Hispanic whites from the Massachusetts Hispanic Elders Study

| | Hispanics | Non-Hispanic whites |
|---------------------------------|---------------------------|---------------------|
| Women | | |
| <i>n</i> | 289 | 96 |
| Age (y) | 69.0 ± 0.4 ^{1,2} | 72.1 ± 0.7 |
| Duration of education (y) | 4.1 ± 0.2 ² | 11.8 ± 0.4 |
| Poverty (% below poverty level) | 67.6 ² | 25.5 |
| Current smoker (%) | 12.5 ² | 30.2 |
| Physical activity score | 28.9 ± 0.2 | 29.5 ± 0.3 |
| BMI (kg/m ²) | 28.4 ± 0.4 | 28.2 ± 0.6 |
| Waist circumference (cm) | 96.1 ± 0.8 | 95.2 ± 1.4 |
| Diabetes prevalence (%) | 39.8 ³ | 25.0 |
| Men | | |
| <i>n</i> | 201 | 67 |
| Age (y) | 68.8 ± 0.5 | 70.0 ± 0.9 |
| Duration of education (y) | 5.3 ± 0.3 ² | 12.0 ± 0.9 |
| Poverty (% below poverty level) | 49.7 ⁴ | 26.9 |
| Current smoker (%) | 21.4 ³ | 34.3 |
| Physical activity score | 29.3 ± 0.2 | 30.0 ± 0.4 |
| BMI (kg/m ²) | 27.3 ± 0.3 | 27.9 ± 0.6 |
| Waist circumference (cm) | 100.0 ± 0.9 | 103.3 ± 1.6 |
| Diabetes prevalence (%) | 32.8 ³ | 19.4 |

¹Age-adjusted $\bar{x} \pm SE$ from general linear models comparing ethnic groups. Logistic regression models were used to assess age-adjusted differences in proportions.

²⁻⁴Significantly different from non-Hispanic whites: ² $P < 0.001$, ³ $P < 0.05$, ⁴ $P < 0.01$.

Associations of plasma values with dietary variables were tested with regression models. Dependent variables were used in their original scales, except for triacylglycerol, which was log transformed because of a lack of normality. We also evaluated similar models for apo A-I and apo B. Because the results closely paralleled those for HDL and LDL cholesterol, respectively, results for apo A-I and apo B are not presented here.

In the Hispanic group separately, we examined the prevalence of dyslipidemia and plasma concentrations of lipids by diabetes status and evaluated the magnitude of the odds ratios of having selected lipid risk factors for CHD.

RESULTS

Descriptive characteristics

A comparison of the descriptive characteristics of Hispanics and non-Hispanic whites from the same neighborhoods is shown in **Table 1**. The Hispanics were younger than the non-Hispanic whites and had fewer years of education ($P < 0.001$). Poverty was also more prevalent among the Hispanics. Significantly fewer Hispanics than non-Hispanic whites were current smokers. No significant differences were observed in physical activity, BMI, or waist circumference between the 2 groups. The prevalence of diabetes was significantly higher in Hispanic men and women than in non-Hispanic whites.

Macronutrient intakes

No significant differences in energy intake were observed between ethnic groups (**Table 2**). After adjustment for age and

TABLE 2Energy and macronutrient intakes, by sex, of elderly Hispanics and non-Hispanic whites¹

| | Hispanics | Non-Hispanic whites |
|---|-------------------------|---------------------|
| Women² | | |
| <i>n</i> | 279 | 96 |
| Energy (kJ/d) ³ | 6673 ± 172 | 6594 ± 293 |
| Cholesterol (mg/d) ⁴ | 185 ± 5 ⁵ | 221 ± 9 |
| Total fat (% of energy) ³ | 30.1 ± 0.4 ³ | 32.9 ± 0.6 |
| MUFA (% of energy) ³ | 9.5 ± 0.1 ⁵ | 11.6 ± 0.2 |
| SFA (% of energy) ³ | 9.6 ± 0.2 ⁵ | 12.3 ± 0.3 |
| PUFA (% of energy) ³ | 8.7 ± 0.1 ⁵ | 6.3 ± 0.3 |
| PUFA:SFA ³ | 1.0 ± 0.02 ⁵ | 0.6 ± 0.04 |
| Carbohydrate (% of energy) ³ | 55.4 ± 0.4 ⁵ | 51.6 ± 0.8 |
| Alcohol (% of energy) ³ | 0.1 ± 0.1 ⁵ | 1.0 ± 0.1 |
| Men² | | |
| <i>n</i> | 196 | 65 |
| Energy (kJ/d) ³ | 8092 ± 222 | 7933 ± 381 |
| Cholesterol (mg/d) ⁴ | 234 ± 9 ⁵ | 291 ± 15 |
| Total fat (% of energy) ³ | 31.3 ± 0.4 ⁶ | 33.5 ± 0.8 |
| MUFA (% of energy) ³ | 9.9 ± 0.2 ⁵ | 12.0 ± 0.3 |
| SFA (% of energy) ³ | 10.0 ± 0.2 ⁷ | 12.0 ± 0.4 |
| PUFA (% of energy) ³ | 9.1 ± 0.2 ⁵ | 6.8 ± 0.3 |
| PUFA:SFA ³ | 1.0 ± 0.02 ⁵ | 0.6 ± 0.04 |
| Carbohydrate (% of energy) ³ | 53.1 ± 0.6 ⁵ | 48.2 ± 1.0 |
| Alcohol (% of energy) ³ | 0.8 ± 0.3 ⁵ | 4.8 ± 0.6 |

¹MUFA, monounsaturated fatty acid; SFA, saturated fatty acid; PUFA, polyunsaturated fatty acid.

²Data for 17 subjects (10 Hispanic women, 5 Hispanic men, and 2 non-Hispanic men) were excluded because they were identified as outliers on the basis of energy intake.

³ $\bar{x} \pm SE$ adjusted for age.

⁴ $\bar{x} \pm SE$ adjusted for age and for total energy intake.

⁵⁻⁷Significantly different from non-Hispanic whites (general linear models): ⁵ $P < 0.001$, ⁶ $P < 0.05$, ⁷ $P < 0.01$.

energy intake, the Hispanic men and women consumed significantly less cholesterol than did the non-Hispanic white men and women. Within sex, the age-adjusted mean percentage of energy from total fat, MUFA, SFA, and alcohol were also significantly lower in Hispanics than in non-Hispanic whites. Hispanics were more likely to consume more energy from carbohydrates and PUFAs than were non-Hispanic whites, and the ratio of PUFA to SFA was higher in the Hispanics than in the non-Hispanic whites. Compared with non-Hispanic white women, Hispanic women had a lower mean contribution of energy from total fat.

Plasma lipids, lipoproteins, and apolipoproteins

The age-adjusted mean concentrations of TC, HDL cholesterol, apo A-I, LDL cholesterol, and apo B were significantly lower in Hispanic women than in non-Hispanic white women (**Table 3**). The mean concentration of Lp(a) cholesterol was higher (0.24 ± 0.01 mmol/L) in Hispanic women than in non-Hispanic white women (0.19 ± 0.02 mmol/L), although the differences were not significant after adjustment for age. Except for significantly higher apo A-I values, no significant differences in mean plasma lipid values were observed between Hispanic men and non-Hispanic white men.

Age-adjusted prevalences of risk categories for plasma lipids, by sex, are presented in **Figures 1** and **2**. The proportion of Hispanic women (Figure 1) with high TC (17%) was significantly

TABLE 3

Age-adjusted fasting plasma lipid, apolipoprotein (apo) A-I, apo B, and lipoprotein(a) [Lp(a)] concentrations, by sex, in elderly Hispanics and non-Hispanic whites¹

| Plasma variable | Hispanics | Non-Hispanic whites |
|----------------------------|--------------------------|---------------------|
| Women | | |
| <i>n</i> | 221 | 78 |
| Triacylglycerols (mmol/L) | 1.72 ± 0.06 | 1.91 ± 0.11 |
| Total cholesterol (mmol/L) | 5.25 ± 0.08 ² | 5.76 ± 0.13 |
| HDL cholesterol (mmol/L) | 1.26 ± 0.02 ² | 1.42 ± 0.04 |
| LDL cholesterol (mmol/L) | 3.20 ± 0.07 ³ | 3.47 ± 0.12 |
| Lp(a) cholesterol (mmol/L) | 0.24 ± 0.01 | 0.19 ± 0.02 |
| Apo A-I (mmol/L) | 3.40 ± 0.05 ² | 3.75 ± 0.08 |
| Apo B (mmol/L) | 2.48 ± 0.05 ³ | 2.69 ± 0.08 |
| Men | | |
| <i>n</i> | 151 | 54 |
| Triacylglycerols (mmol/L) | 1.66 ± 0.08 | 1.73 ± 0.13 |
| Total cholesterol (mmol/L) | 4.97 ± 0.08 | 5.15 ± 0.13 |
| HDL cholesterol (mmol/L) | 1.09 ± 0.03 | 1.11 ± 0.04 |
| LDL cholesterol (mmol/L) | 3.12 ± 0.07 | 3.25 ± 0.12 |
| Lp(a) cholesterol (mmol/L) | 0.22 ± 0.02 | 0.19 ± 0.03 |
| Apo A-I (mmol/L) | 3.03 ± 0.05 ³ | 3.23 ± 0.08 |
| Apo B (mmol/L) | 2.42 ± 0.05 | 2.51 ± 0.09 |

¹In comparisons between Hispanics and non-Hispanic whites, within sex groups, values were adjusted for age. Triacylglycerol and Lp(a) were log transformed for statistical testing.

^{2,3}Significantly different from non-Hispanic whites: ² $P < 0.001$, ³ $P < 0.05$.

lower than that observed among non-Hispanic white women (35%). However, more Hispanic women (34%) than non-Hispanic white women (18%) had high Lp(a) cholesterol. No significant differences in the age-adjusted prevalences of risk categories of plasma lipids were found between Hispanic men and non-Hispanic white men (Figure 2).

Association of ethnicity and dietary factors with plasma lipids

The association of ethnicity (Hispanic or non-Hispanic white) with triacylglycerol, HDL cholesterol, and LDL cholesterol was

evaluated, with adjustment for age, sex, years of education, smoking status, waist circumference, BMI, and use of antihyperlipidemic drugs. In these adjusted models (data not shown), non-Hispanic whites had significantly higher HDL-cholesterol concentrations than did Hispanics, and Hispanics had higher concentrations of LDL cholesterol than did non-Hispanic whites ($P < 0.05$). No ethnic effect was observed with triacylglycerol.

We further evaluated linear regression models of triacylglycerol, HDL cholesterol, and LDL cholesterol, with dietary variables as independent factors, in Hispanics and non-Hispanic whites separately (Table 4), with adjustment for age, sex, smoking status, physical activity, waist circumference, and energy intake. Age, sex, and energy intake were retained in all models. Other non-significant variables ($P \geq 0.15$) were removed sequentially. The models explained the plasma concentrations of triacylglycerol, HDL cholesterol, and LDL cholesterol more completely among non-Hispanic whites (higher adjusted R^2 values) than among the Hispanics.

In Hispanics, a borderline negative association ($P = 0.08$) between triacylglycerol and MUFA intake was observed (Table 4). In addition, the proportion of total energy intake from MUFA and from alcohol and TC were each positively associated with HDL cholesterol, but none of the tested dietary variables were associated with LDL cholesterol. In non-Hispanic whites, high concentrations of triacylglycerol were negatively associated with the proportion of energy intake from carbohydrate and alcohol, whereas the only dietary variable that tended to be associated with HDL cholesterol was alcohol (positively), although the association was not significant ($P = 0.10$). Lower LDL-cholesterol concentrations were associated with the proportion of energy from alcohol ($P < 0.001$), MUFA ($P < 0.05$), and carbohydrate ($P < 0.10$).

Diabetes and plasma lipid profiles

The prevalence of dyslipidemia and other lipid risk factors is presented by diabetes status for the Hispanics in Table 5. We tested logistic models with diabetes as the dependent variable and dyslipidemia or other lipid risk factors as independent variables, with adjustment for age, physical activity, smoking status, and waist circumference (which was a stronger predictor of plasma

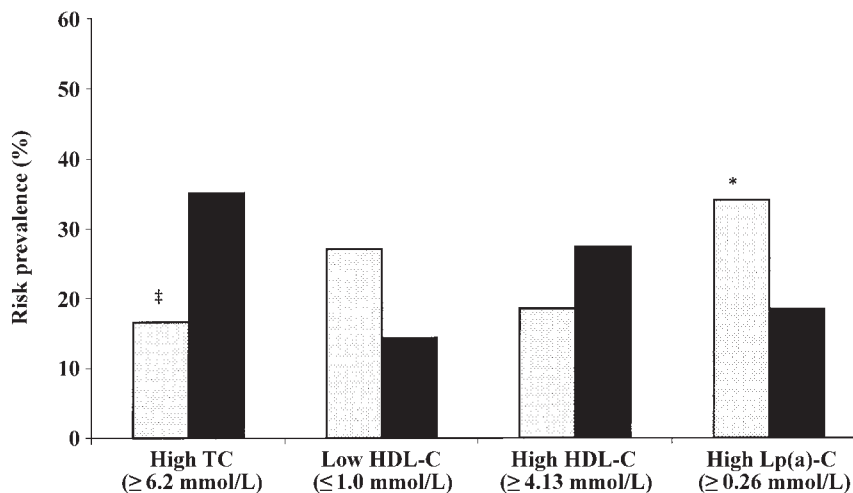


FIGURE 1. Prevalence of high total cholesterol (TC), low HDL cholesterol (HDL-C), high HDL-C, and high lipoprotein(a) cholesterol [Lp(a)-C] in Hispanic women (□; $n = 221$) and non-Hispanic white women (■; $n = 78$) in Massachusetts. Values were adjusted for age and use of antihyperlipidemic agents with logistic regression models. ^{*} $P < 0.05$, [‡] $P < 0.001$.

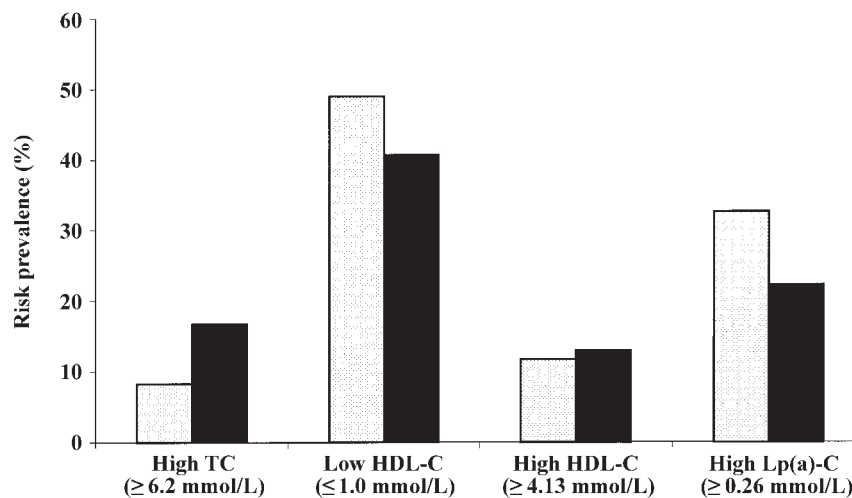


FIGURE 2. Prevalence of high total cholesterol (TC), low HDL cholesterol (HDL-C), high HDL-C, and high lipoprotein(a) cholesterol [Lp(a)-C] in Hispanic men (□; $n = 151$) and non-Hispanic (■; $n = 54$) white men in Massachusetts. Values were adjusted for age and use of antihyperlipidemic agents with logistic regression models. There were no significant differences between the ethnic groups.

concentrations of lipids and lipoproteins than was BMI in our previous analysis). A significantly higher proportion of women with (29.6%) than without (17.8%) diabetes had high triacylglycerol concentrations; this difference remained significant after adjustment for potential confounders. There was a trend toward more dyslipidemia among women with diabetes than among those without it, but the difference (44.3% compared with $\approx 35.6\%$) was not significant. Hispanic men with diabetes were significantly more likely to have dyslipidemia, high triacylglycerol, and low HDL cholesterol (68.2%, 37.9%, and 62.1%, respectively) than were those without diabetes (51.1%, 14.8%, and 45.9%, respectively).

TABLE 4
Association of dietary factors with fasting plasma lipids in elderly Hispanics and non-Hispanic whites¹

| Factor | Triacylglycerol ² | HDL cholesterol | LDL cholesterol |
|-----------------------------------|------------------------------|--------------------|--------------------|
| Hispanics ($n = 372$) | | | |
| MUFA (% of energy) | -0.01 | 0.03 ³ | |
| Carbohydrates (% of energy) | | 0.008 | |
| Alcohol (% of energy) | | 0.04 ⁴ | |
| Cholesterol (mg/d) | | 0.001 ⁴ | |
| Adjusted R^2 | 0.04 | 0.14 | 0.01 |
| Non-Hispanic whites ($n = 132$) | | | |
| MUFA (% of energy) | -0.02 | | -0.13 ³ |
| Carbohydrate (% of energy) | -0.008 ³ | | -0.03 |
| Alcohol (% of energy) | -0.01 ³ | 0.01 | -0.08 ⁵ |
| Cholesterol (mg/d) | | | |
| Adjusted R^2 | 0.08 | 0.33 | 0.15 |

¹ Values are β coefficients. Other variables were tested and removed because they were nonsignificant at $P < 0.15$ (BMI, years of education, use of antihyperlipidemic drugs, physical activity, and energy from total fat and polyunsaturated and saturated fatty acids). Sex, age, diabetes, and total energy intake were retained in all models.

² Log transformed.

³ $P < 0.05$.

⁴ $P < 0.01$.

⁵ $P < 0.001$.

Waist circumference was strongly related to diabetes status in all of the models tested.

DISCUSSION

This article presents data on dietary intakes and plasma lipid concentrations of Hispanics of Puerto Rican and Dominican origin residing in Massachusetts, a group for whom information on CVD risk factors is limited. We also examined patterns of dietary intake and plasma lipids by diabetes status. Hispanic women were less likely than non-Hispanic whites to have elevated concentrations of TC, LDL cholesterol, and apo B. However, Hispanic women also had lower concentrations of the CVD-protective factors HDL cholesterol and apo A-I than did non-Hispanic white women. Hispanic men had lower concentrations of apo A-I than did their non-Hispanic white counterparts. Among men, mean plasma TC and HDL-cholesterol concentrations followed the same trends as observed among women, although differences between the ethnic groups were not significant after age adjustment.

Published data from the Framingham Offspring Study (28) showed HDL cholesterol and apo A-I values that were similar to those reported in the present study for non-Hispanic whites. They also reported that apo A-I concentrations < 3.10 mmol/L may be associated with an increased risk of CVD (28). The mean concentration of this apolipoprotein was below this cutoff point in Hispanic men. Men and women from the Framingham Offspring Study (27) had higher concentrations of LDL cholesterol and apo B than did the non-Hispanic whites in the present study. Increased LDL cholesterol and elevated apo B are associated with an increased risk of CVD (27). Our data suggest that Hispanic men may have an increased risk of CVD because of low concentrations of HDL cholesterol and apo A-I, whereas the risk of CVD in non-Hispanic whites may be increased because of elevated concentrations of LDL cholesterol and apo B.

Mexican Americans from San Antonio, TX, have been shown to have lower plasma concentrations of TC than do non-Hispanic whites (5–7). On the other hand, results from the Laredo (6), San Antonio (5, 8), and San Luis Valley (9) studies showed that

TABLE 5

Dyslipidemia and other risk factors in elderly Hispanics, by sex and diabetes status¹

| | No diabetes: risk | | Diabetes | |
|---|-------------------|---|----------|--------------------------------|
| | % | % | Risk | OR (95% CI) |
| Women | | | | |
| <i>n</i> | 174 | | 115 | |
| Dyslipidemia ² | 35.6 | | 44.3 | 1.55 (0.09, 2.45) |
| High triacylglycerols (≥ 2.3 mmol/L) | 17.8 | | 29.6 | 2.21 (1.23, 3.96) ³ |
| High total cholesterol (≥ 6.2 mmol/L) | 18.4 | | 12.2 | 0.61 (0.30, 1.22) |
| Low HDL cholesterol (≤ 1.0 mmol/L) | 27.0 | | 29.6 | 1.13 (0.66, 1.95) |
| Men | | | | |
| <i>n</i> | 135 | | 66 | |
| Dyslipidemia ² | 51.1 | | 68.2 | 1.99 (1.05, 3.77) ⁴ |
| High triacylglycerols (≥ 2.3 mmol/L) | 14.8 | | 37.9 | 3.29 (1.64, 6.60) ⁵ |
| High total cholesterol (≥ 6.2 mmol/L) | 8.1 | | 4.5 | 0.52 (0.14, 1.97) |
| Low HDL cholesterol (≤ 1.0 mmol/L) | 45.9 | | 62.1 | 1.93 (1.04, 3.58) ⁴ |

¹ Determined in fasting and nonfasting samples. Differences between groups were tested with logistic regression models, with adjustment for age, waist circumference, and smoking status. OR, odds ratio.

² Defined as a triacylglycerol concentration > 2.3 mmol/L, an HDL-cholesterol concentration < 1.0 mmol/L, or both.

³ $P < 0.01$

⁴ $P < 0.05$.

⁵ $P < 0.001$.

Mexican Americans were more likely to have higher triacylglycerol concentrations than were non-Hispanic whites. Hispanic men and women from the San Luis Valley also had significantly higher plasma concentrations of apo B than did their non-Hispanic counterparts (38). We observed no significant differences in triacylglycerol concentrations by ethnicity.

Elevated plasma Lp(a) is an independent risk factor for CHD (15, 39, 40), and it has been shown that Lp(a) distribution in plasma differs significantly across ethnic groups. Plasma Lp(a) concentrations were significantly higher in Hispanic men of Mexican origin than in non-Hispanic white men in the San Luis Valley (38). We observed higher concentrations of Lp(a) cholesterol in Hispanic women than in non-Hispanic white women, although the differences were not significant after adjustment for age. No significant differences in Lp(a) concentrations were seen between Hispanic men and non-Hispanic white men. In the Framingham Offspring Study, Seman et al (15) reported mean concentrations of Lp(a) in men and women aged > 60 y that were similar to those observed in the non-Hispanic men and women in the present study.

The effects of dietary macronutrients on plasma lipid concentrations and their effects on cardiovascular health have been extensively studied (41–43). However, few studies have examined associations between dietary macronutrient intakes and plasma lipids in Hispanics of Caribbean origin. Almost 30 y ago, the Puerto Rican Heart Study found lower mean concentrations of TC and triacylglycerol in Puerto Ricans than in subjects in the Framingham Heart Study (44). Among urban Puerto Rican men, TC was positively associated with the percentage of energy from total fat, SFAs, simple sugars, and protein and negatively associated with the percentage of energy from PUFAs, total carbohydrate, and PUFA:SFA (44).

The Hispanics in the present study reported macronutrient intakes that appear to be more protective than those of the non-Hispanic whites on the basis of recent guidelines from the American Heart Association (14). However, the percentage of energy from MUFAs (which protect the cardiovascular system) was lower in the Hispanics than in the non-Hispanic whites. In the Hispanic


Health and Nutrition Examination Survey (HHANES), elderly (60–74 y) Puerto Ricans reported lower intakes of total fat, MUFAs, and PUFAs and higher intakes of carbohydrates than did Mexican Americans and higher intakes of PUFAs than did the Cubans (45).

Among the Hispanics in the present study, alcohol intake was associated with higher plasma HDL cholesterol. Among non-Hispanic whites, alcohol was associated with lower plasma LDL cholesterol and tended to be associated with higher HDL cholesterol. An inverse relation between alcohol intake and CHD was reported in Puerto Rican men in Puerto Rico (46). Increases in HDL cholesterol due to alcohol consumption may be caused by the increased transport of the HDL apo A-I and apo A-II (47). It was shown that alcohol intake influences circulating lipid and lipoprotein concentrations (43), which in turn tend to reduce the risk of CHD (41, 42).

A potential beneficial effect of dietary MUFAs on HDL cholesterol has been suggested (41). We saw a significant association between MUFAs and plasma HDL cholesterol among the Hispanics but not among the non-Hispanic whites. However, MUFA intake was negatively associated with LDL cholesterol in the non-Hispanic whites.

Diabetes is highly prevalent among Hispanics (16–20). We previously documented not only the high prevalence of diabetes among Hispanic groups of Caribbean origin but also the elevated rates of uncontrolled diabetes (21). Data from the first National Health and Nutrition Examination Survey (NHANES I) conducted in 1971–1974 and the NHANES I Epidemiologic Follow-up Survey (1982–1984) showed that, whereas age-adjusted heart disease mortality declined for the general population (36% for men and 27% for women), it decreased at a lower rate in men with diabetes (13% decrease) and increased in women with diabetes (23%) (48). The American Heart Association has identified type 2 diabetes as a risk factor for CVD (49). Type 2 diabetes, as a component of the metabolic syndrome, is commonly accompanied by other CVD risk factors such as dyslipidemia, hypertension, obesity, and prothrombotic factors.

As reported for other Hispanic groups (50), dyslipidemia in our study was more prevalent among those with diabetes than among those without this chronic condition. Hispanics with diabetes were more likely to have high triacylglycerol concentrations than were those without diabetes. Low HDL cholesterol was more prevalent among Hispanic men with than without diabetes. As documented previously (51), differences in the prevalence of dyslipidemia between the elderly with and without diabetes were not completely explained by differences in central obesity. Haffner (51) suggested that subjects with diabetes may have impaired catabolism of VLDLs, decreased plasma lipoprotein lipase (EC 3.1.1.34) activity, and increased catabolism of HDL because of increased hepatic triacylglycerol lipase (EC 3.1.1.3) activity. The HDL particles are also triacylglycerol-rich and cholesterol-poor. We observed no significant differences in the absolute concentration of LDL cholesterol by diabetes status, as was documented in one study (51). However, many metabolic and compositional changes in LDL have been described. In normotriglyceridemic subjects with type 2 diabetes, both LDL synthesis and catabolism may be increased; however, in more severe hypertriglyceridemic subjects, LDL catabolism may be impaired because of LDL glycation (52, 53).

Although the results of the present study are suggestive, its cross-sectional design prevents us from making causal inferences on the relation between diet and plasma lipid risk factors for CVD for the total group and for the subgroup with diabetes. Our study population may have received dietary counseling that influenced their dietary patterns. Longitudinal and intervention studies are needed to determine the magnitude of ethnic differences in dietary and plasma factors and to provide information on the management of plasma lipids through diet to reduce CVD morbidity and mortality, particularly in persons with diabetes. 

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