Greater dietary intake of simple carbohydrate is associated with lower concentrations of high-density-lipoprotein cholesterol in hypercholesterolemic children

Thomas J Starc, Steven Shea, Lisa C Cohn, Lori Mosca, Welton M Gersony, and Richard J Deckelbaum

ABSTRACT  Hypercholesterolemic children are increasingly being treated with lipid-lowering diets, but little research has focused on the effects of specific dietary substitutions on HDL cholesterol. We examined the relation between carbohydrate intake and HDL cholesterol in hypercholesterolemic children consuming fat-restricted diets. We obtained 3-d food records for 67 children (mean age: 5.8 ± 2.5 y) referred for the treatment of hypercholesterolemia. Mean plasma HDL cholesterol was 1.12 ± 0.21 mmol/L and total cholesterol was 5.99 ± 1.39 mmol/L. Dietary intake comprised (percentage of total energy) 24.9 ± 5.1% fat, 59.9 ± 6.5% carbohydrate, and 16.5 ± 3.4% protein. Carbohydrate intake included 30.7 ± 7.4% from simple and 22.6 ± 6.2% from complex carbohydrates. HDL cholesterol was positively correlated with intake of total fat (r = 0.44, P < 0.001) and saturated fatty acids (r = 0.43, P < 0.001) and inversely correlated with intake of total carbohydrate (r = −0.55, P < 0.001) and simple carbohydrate (r = −0.40, P < 0.001), but not with complex carbohydrate (r = −0.02). The significant inverse relation between simple carbohydrate intake and HDL cholesterol remained after intakes of saturated, monounsaturated, and polyunsaturated fatty acids; intake of complex carbohydrates; dietary cholesterol; plasma triacylglycerol; and age were adjusted for with multivariate techniques. In summary, higher dietary intake of simple carbohydrates was associated with lower HDL-cholesterol concentrations in hypercholesterolemic children consuming reduced-fat diets. Am J Clin Nutr 1998;67:1147–54.

KEY WORDS  HDL cholesterol, children, diets, hypercholesterolemia, simple carbohydrate, complex carbohydrate, low-fat diet

INTRODUCTION

Recommendations for the treatment of hypercholesterolemic children aged > 2 y suggest lowering intake of total fat to < 30% of energy (1). Lowering dietary fat intake is associated with reductions in plasma total and LDL cholesterol in adults (2) as well as children (3–5). However, the effect of low-fat diets on other lipids such as HDL cholesterol has not been studied as extensively. Because low concentrations of HDL cholesterol are an important coronary risk factor in adults (6), it is desirable to consider dietary components that alter HDL cholesterol when planning dietary therapy for hypercholesterolemic children.

Reduced-fat diets have been associated with lower HDL-cholesterol concentrations in adults (7). Studies of the effects of reduced-fat diets on HDL-cholesterol concentrations in children have been less consistent, ranging from no effect (4, 8) to a small lowering effect on HDL cholesterol (9). Investigators have also reported that carbohydrate intake is inversely related to HDL-cholesterol concentrations in adults (10). Similarly, an international population study of children suggested an inverse association between total carbohydrate intake and HDL cholesterol (11), but this relation has not been investigated in hypercholesterolemic children in a single population. Furthermore, it is unknown whether these effects on HDL cholesterol are associated with simple or complex carbohydrates. We studied the relations between dietary carbohydrate intake and HDL cholesterol in a group of hyperlipidemic children consuming reduced-fat diets. We hypothesized that dietary intakes of total and simple carbohydrates would be inversely related to HDL-cholesterol concentrations.

SUBJECTS AND METHODS

Settings and subjects

Between December 1987 and February 1993, 279 children aged 2–10 y were referred for evaluation at the Children’s Cardiovascular Health Center at Columbia-Presbyterian Medical Center. The center is a referral program for the diagnosis and treatment of children with hyperlipidemia. Patients were usually referred by a pri-

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mary care health care provider after the initial diagnosis of hyperlipidemia was made. The child’s medical history, results of previous cholesterol testing, and consent to participate in the center program were obtained at the time of the first visit. The study was approved by the Columbia-Presbyterian Institutional Review Board.

Sixty-seven children who returned complete 3-d diet records within 2 wk of the initial blood tests were included in this study. Children whose diet records described time periods more distant from the time of the blood sample, as well as children taking lipid-lowering medications, were excluded. We also excluded children with triacylglycerol concentrations > 4.52 mmol/L (n = 1), homozygous LDL-cholesterol receptor deficiency (n = 2), cholesterol ester storage disease (n = 1), and secondary lipid disorders such as nephrotic syndrome (n = 1) and thyroid disease (n = 1). Most study children (47/67; 70%) reported following a cholesterol-lowering diet before coming to the center.

Dietary analysis

At the time of the first visit, all patients were requested to provide a food record to assess the effects of diet on lipids and to facilitate dietary counseling. Parents and children were instructed in recording food intake and estimating food quantity, preparation techniques, and brand information by a registered dietician specializing in dietary therapy of children. Parents were asked to record the child’s food intake over a 3–7-d period. Parents and children were advised to record the child’s food intake at the time of the first visit. Diet records were mailed to the center or brought in at the next center visit. Records were reviewed by the nutritionist and, when appropriate, families were questioned concerning details on incomplete food items. Only the first 3 d of the diet records were used in this study so that the number of days analyzed was the same for all 67 children. Forty-five of 67 (67%) of the 3-d records included ≥ 1 weekend day.

Nutrient analyses were performed with the Minnesota NUTRITION DATA SYSTEM (NDS) software, developed by the Nutrition Coordinating Center at the University of Minnesota (12). Records between 1987 and 1990 (n = 43) were analyzed by using Food Database version 1.3 and Nutrient Database Version 16 and records between 1991 and 1993 (n = 24) were analyzed by using Food Database version 2.3 and Nutrient Database Version 20. In the earlier version (1.3), all foods were numerically coded by the nutritionist and then entered into the computer. In version 2.3, the coding was automatic. Diets were analyzed for total energy and for the percentage of total energy provided by fats, carbohydrates, and proteins. Fat intake was subdivided into saturated, monounsaturated, and polyunsaturated fatty acids and carbohydrates were reported as either simple or complex. Total simple carbohydrates included the sum of energy from sucrose, fructose, glucose, and lactose. Complex carbohydrates included the sum of energy from starch and glycogen. The dietary sources of 6.6 ± 2.0% of energy in the total carbohydrate group were not individually identified in the Minnesota NDS and included items such as galactose, maltose, and other organic acids.

The comparability of NDS versions 1.3 and 2.3 was assessed by selecting a random sample of 10 of the 43 (23%) food records analyzed with NDS version 1.3. These food records were reanalyzed with NDS version 2.3. Mean values for energy and nutrient densities were similar with both NDS versions. Total fat intake was 24.5 ± 5.9% according to NDS version 1.3 compared with 24.2 ± 6.7% according to NDS version 2.3. Total carbohydrate intake was 58.2 ± 8.9% according to NDS version 1.3 compared with 58.7 ± 9.1% according to NDS version 2.3. The Pearson correlation coefficients ranged from 0.70 for polyunsaturated fatty acid intake to 0.99 for protein. The intraclass correlation coefficients ranged from 0.71 for polyunsaturated fatty acid to 0.98 for complex carbohydrate intake.

Lipid analysis

A fasting venous blood sample was obtained on the morning of the child’s first visit to the center. Plasma was separated by centrifugation at 3000 rpm for 15 min at room temperature and stored at 4°C before being analyzed. Plasma total cholesterol and triacylglycerol concentrations were determined by enzymatic methods using Lipid Research Clinics techniques and an automated spectrophotometer (13). HDL cholesterol was measured in the same manner after the precipitation of apolipoprotein B–containing lipoproteins with magnesium and phosphotungstate (14). Our laboratory participates in the Lipid Research Clinics quality control program administered by the Centers for Disease Control and Prevention. The interassay CVs in our laboratory were < 2% for total cholesterol and < 5% for HDL cholesterol. LDL cholesterol was calculated by using the standard Friedewald equation as recommended in children (1):

\[
\text{LDL cholesterol (mg/dL)} = \text{total cholesterol (mg/dL)} - \text{HDL cholesterol (mg/dL)} - \left[\frac{\text{triacylglycerols (mg/dL)}}{5}\right] 
\]

Data analysis

Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Data from lipid profiles and dietary composition were analyzed on personal computers with SPSS (15) and SAS computer software (16). Diet data are reported in terms of nutrient density (percentage of total energy) and grams per day adjusted for energy intake by use of the regression method described by Willett and Stampfer (17).

Associations between dietary variables and HDL cholesterol were assessed by using bivariate analyses with the dietary variable as the independent variable and HDL cholesterol as the dependent variable. Because BMI, age, triacylglycerols, and sex have been shown to affect HDL cholesterol in population-based studies of normal children (18), the effect of these variables on HDL cholesterol was also tested by using bivariate regression analyses.

A multiple regression model with HDL-cholesterol concentration as the dependent variable and dietary intakes of simple and complex carbohydrates; intakes of saturated, monounsaturated, and polyunsaturated fatty acids; dietary cholesterol; log of the triacylglycerol concentration; and age was fitted by using enter testing multiple regression analysis (15). Dietary variables for carbohydrates, fats, cholesterol, log of the triacylglycerol concentration, and age were included in this model if they were significant in the bivariate model or if they changed the β coefficient of the independent variable of primary interest (simple carbohydrate) by > 10%.

As expected, intercorrelations existed between various dietary variables. The two highest correlations between nutrients were between saturated and monounsaturated fatty acids (r = 0.70, P < 0.001) and between simple and complex carbohydrates (r = −0.62, P < 0.001). Other correlations between dietary variables ranged from r = −0.51 for monounsaturated fatty acid and...
simple carbohydrates to \( r = 0.38 \) for monounsaturated and polyunsaturated fatty acids. In the multivariate model, nutrient values were adjusted with centering techniques by subtracting the mean from each value. Collinearity diagnostics after centering showed a variance inflation factor < 5.24, condition number = 4.98, Eigenvalues > 0.105, and variance proportions < 0.935, indicating an acceptable level of freedom from major collinearity problems (19).

HDL-cholesterol concentrations in children with high compared with low intakes of simple carbohydrates were also compared among groups of children divided into quartiles of intake of simple carbohydrates. We performed one-way analysis of variance to test for overall significance among groups, and differences between groups were tested for significance by using Duncan’s multiple-range test. Data are presented as means ± 1 SD. Because the distribution of plasma triacylglycerols was skewed toward the higher values, a logarithmic transformation of this variable was used in regression analyses. Noncontinuous variables were compared with the chi-square test or Fisher’s exact test.

RESULTS

The mean age of the 67 children was 5.8 ± 2.5 y and ranged from 2 to 10.9 y (Table 1). The racial makeup of the children was 87% white, 10% Hispanic, and 3% Asian. In this group of children referred for treatment of hypercholesterolemia, the mean total cholesterol concentration was 5.99 ± 1.39 mmol/L (232 ± 54 mg/dL) and the mean LDL-cholesterol concentration was 4.34 ± 1.47 mmol/L (168 ± 57 mg/dL). Both of these values are well above the 95th percentile for children aged 2–10 y (20).

TABLE 1
Demographics and lipid concentrations of participating and nonparticipating children

<table>
<thead>
<tr>
<th></th>
<th>Participants (n = 67)</th>
<th>Nonparticipants (n = 212)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>5.8 ± 2.5(^1)</td>
<td>6.6 ± 2.5(^1)</td>
</tr>
<tr>
<td>Sex (% boys)</td>
<td>34.3</td>
<td>39.6</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>16.6 ± 2.6</td>
<td>17.8 ± 3.8</td>
</tr>
<tr>
<td>Girls</td>
<td>16.0 ± 1.9</td>
<td>18.0 ± 3.6(^2)</td>
</tr>
<tr>
<td>Race [n(%)]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>2/67 (3)</td>
<td>7/212 (3)</td>
</tr>
<tr>
<td>Black</td>
<td>0/67 (0)</td>
<td>10/212 (5)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>7/67 (10)</td>
<td>93/212 (44)(^4)</td>
</tr>
<tr>
<td>White</td>
<td>58/67 (87)</td>
<td>96/212 (45)(^4)</td>
</tr>
<tr>
<td>Not identified</td>
<td>0/67 (0)</td>
<td>6/212 (3)</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>5.99 ± 1.39</td>
<td>5.77 ± 1.40</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>4.34 ± 1.47</td>
<td>4.05 ± 1.42</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.12 ± 0.21</td>
<td>1.20 ± 0.31</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/L)</td>
<td>1.15 ± 0.51</td>
<td>1.14 ± 0.62</td>
</tr>
</tbody>
</table>

\(^1\)\(^x\) ± SD; \(^2\)Significantly different from participants: \(^3\)\(^P < 0.05\), \(^4\)\(^P < 0.01\).

Total fat concentrations were 1.12 ± 0.25 mmol/L in boys (\(n = 23\)) and 1.12 ± 0.20 mmol/L in girls (\(n = 44\); \(P = 0.97\)). Total and LDL cholesterol as well as triacylglycerol were also similar in boys and girls. Twenty-seven children were presumed to have heterozygous familial hypercholesterolemia on the basis of an LDL-cholesterol concentration > 4.24 mmol/L (21). HDL cholesterol was 1.09 ± 0.21 mmol/L in those with presumed heterozygous familial hyperlipidemia, similar to the concentration in the others (1.15 ± 0.22 mmol/L; \(P = 0.29\)).

BMI was calculated in 64 children and was 16.2 ± 2.2, between the 50th and 75th percentile for 6-y-old children (22). There was no systematic relation between age- and sex-matched BMI percentiles and HDL cholesterol. HDL cholesterol was 1.16 ± 0.16 mmol/L in children with a BMI greater than the 75th percentile for age compared with 1.13 ± 0.23 mmol/L in those with BMI less than the 25th percentile for age (\(P > 0.05\)).

The children in the study group were generally comparable with the other children referred to the center who did not meet the criteria for inclusion in the present study (Table 1). Self-reports of educational status of the mothers of the children in the center were available for 38 families seen during the last 14 mo of the study. Mothers of participants were more highly educated: 15/16 participant mothers had completed at least high school compared with only 10/22 in the nonparticipant group (\(P < 0.05\)).

Nutrient intake

The contributions of dietary components to energy intake are shown in Table 2. Mean energy intake was 5870 ± 1439 kJ/d. The mean total fat intake was 24.9 ± 5.1% of total energy. Carbohydrate intake was 59.9 ± 6.5% of energy and protein made up the remainder. Saturated fatty acids provided 8.5 ± 2.5%, monounsaturated fatty acids 8.9 ± 2.1%, and polyunsaturated fatty acids 5.4 ± 1.6% of total energy. Simple carbohydrates made up 30.7 ± 7.4% of total energy, whereas 22.6 ± 6.2% of total energy was derived from complex carbohydrates. The intake of individual simple sugars are shown in Table 2.
Fat intake and HDL cholesterol

Total fat intake was positively correlated with HDL cholesterol \((r = 0.44, P < 0.001)\) (Figure 1 and Table 3). Saturated fatty acids and monounsaturated fatty acids were also positively correlated with HDL cholesterol. In addition, the intake of dietary cholesterol was positively correlated with HDL cholesterol. In this group of hypercholesterolemic children, most of whom were consuming reduced-fat diets, no relation was found between dietary fat and total or LDL cholesterol.

Dietary intake and other lipoproteins

Dietary intake of total carbohydrate was positively correlated with plasma triacylglycerol \((r = 0.31, P < 0.02)\). In addition, the intake of total fat was positively correlated with plasma triacylglycerol \((r = 0.25, P < 0.05)\). Plasma triacylglycerol was inversely correlated with HDL cholesterol \((r = -0.34; P < 0.01)\). There were no significant relations between intake of fats and carbohydrates and either total or LDL cholesterol.

In the multivariate model, the relation between simple carbohydrate intake and HDL cholesterol remained significant \((P = \ldots)\).

HDL cholesterol and nondietary variables

HDL cholesterol was directly correlated with the age of the children \((r = 0.26, P < 0.05)\). No significant relation was seen between HDL cholesterol and intake of total energy, BMI, or sex.

Carbohydrate intake and HDL cholesterol

There was a significant inverse relation between total carbohydrate intake and HDL cholesterol \((r = -0.55, P < 0.001)\); Figure 2). Simple carbohydrate intake and HDL cholesterol were also inversely related \((r = -0.40, P < 0.001)\); Figure 3), as were fructose and glucose intake and HDL cholesterol. No significant relation was seen between sucrose or lactose intake and HDL cholesterol, nor between the intake of complex carbohydrate or dietary fiber and HDL cholesterol.

The relation between intake of simple carbohydrate and HDL cholesterol was further investigated by comparing plasma HDL cholesterol in children grouped by quartile of simple carbohydrate intake (Figure 4). Mean HDL cholesterol was 1.21 ± 0.18 mmol/L in the quartile with the lowest intake of simple carbohydrate compared with 1.03 ± 0.16 mmol/L in the quartile of children with the highest intake of simple carbohydrate \((P < 0.05)\). The mean HDL-cholesterol concentration was also lower in children in the second-highest quartile of simple carbohydrate intake than in those in the lowest quartile \((P < 0.05)\).

HDL cholesterol and nondietary variables

HDL cholesterol was directly correlated with the age of the children \((r = 0.26, P < 0.05)\). No significant relation was seen between HDL cholesterol and intake of total energy, BMI, or sex.

### Table 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fat</td>
<td>0.44</td>
</tr>
<tr>
<td>Saturated fatty acid</td>
<td>0.43</td>
</tr>
<tr>
<td>Monounsaturated fatty acid</td>
<td>0.30</td>
</tr>
<tr>
<td>Polyunsaturated fatty acid</td>
<td>0.24</td>
</tr>
<tr>
<td>Total carbohydrate</td>
<td>-0.55</td>
</tr>
<tr>
<td>Complex</td>
<td>-0.02</td>
</tr>
<tr>
<td>Simple</td>
<td>-0.40</td>
</tr>
<tr>
<td>Sucrose</td>
<td>-0.08</td>
</tr>
<tr>
<td>Fructose</td>
<td>-0.38</td>
</tr>
<tr>
<td>Glucose</td>
<td>-0.33</td>
</tr>
<tr>
<td>Lactose</td>
<td>-0.01</td>
</tr>
<tr>
<td>Protein intake</td>
<td>0.28</td>
</tr>
<tr>
<td>Cholesterol intake</td>
<td>0.25</td>
</tr>
<tr>
<td>Dietary fiber intake</td>
<td>0.18</td>
</tr>
<tr>
<td>Age</td>
<td>0.26</td>
</tr>
<tr>
<td>BMI</td>
<td>-0.01</td>
</tr>
<tr>
<td>Sex</td>
<td>0.00</td>
</tr>
</tbody>
</table>

\(^1 n = 67.\) 
\(^2 P < 0.001.\) 
\(^3 P < 0.05.\) 
\(^4 P < 0.01.\)
HDL cholesterol at the time of the first visit was \(-0.61\) \((P < 0.05)\), indirectly suggesting that the children had changed their diets before coming to the center.

**DISCUSSION**

The results of this study indicate that the type and amount of carbohydrate ingested were associated with HDL-cholesterol concentrations in 67 hypercholesterolemic children consuming reduced-fat diets. Specifically, we found an inverse relation between intake of simple carbohydrates and plasma HDL-cholesterol concentrations in a bivariate analysis and in a multivariate analysis after intake of saturated, monounsaturated, and polyunsaturated fatty acids; intake of complex carbohydrates; dietary carbohydrate; plasma triacylglycerol; and age were adjusted. Children in the highest quartile of simple carbohydrate intake had a mean plasma HDL-cholesterol concentration that was \(\approx 20\%\) lower than the concentrations for children in the lowest quartile of simple carbohydrate intake.

International population studies have found that HDL cholesterol is directly correlated with dietary fat intake in groups of children from different countries (11, 23). Although some children following cholesterol-lowering diets have been reported to have low HDL cholesterol (9), other investigators reported a small increase or no change in HDL cholesterol in children consuming cholesterol-lowering diets (4, 8, 24, 25).

In most dietary surveys, carbohydrate intake is inversely proportional to fat intake. An international population study in children suggested an inverse relation between total carbohydrate intake and HDL cholesterol (11). The results of cross-sectional studies of children in the United States, however, have been inconsistent. In one study of 10-y-old children in which 24-h dietary recalls were used (the Bogalusa Heart Study), an inverse relation was found between total carbohydrate intake and HDL cholesterol \((P < 0.05)\) as well as between sucrose and HDL cholesterol \((P < 0.001)\) (26). Similarly, Morrison et al (27) reported an inverse relation between sucrose and HDL cholesterol in children. In contrast, and similar to our findings, the Lipid Research Clinics Program Prevalence Study found no significant relation between sucrose and HDL-cholesterol intake in children (28).

Feeding studies in healthy adults have shown that diets rich in simple carbohydrates are associated with declines in HDL cholesterol as well as total cholesterol, but have not established whether this association is due to reduced fat intake, increased carbohydrate intake, or both (29). Studies on the effects of simple and complex carbohydrates on HDL cholesterol in adults have had conflicting results. Mensink and Katan (10), in a study that did not compare simple with complex carbohydrates, described lower HDL-cholesterol concentrations in adults consuming a high-complex-carbohydrate diet compared with a high-monounsaturated-fatty-acid diet. A feeding study of adults in which gradually increased amounts of complex carbohydrates were consumed showed a trend toward lower HDL cholesterol; however, no statistically significant changes in HDL cholesterol were seen (30). The effect of individual simple sugars on HDL cholesterol is also incompletely understood. In short-term feeding studies, a high-sucrose diet was associated with decreased HDL cholesterol (31). In contrast, a high-fructose diet was associated with higher fasting HDL-cholesterol concentrations than was a high-starch diet (32). Others have shown no differences in HDL cholesterol in subjects consuming diets high in fructose compared with diets high in amylose (33).

**FIGURE 2.** Relation between total carbohydrate intake and HDL cholesterol in 67 children referred for hypercholesterolemia (\(n = 67\); \(r = -0.55, P < 0.001\)).

**TABLE 4**

Results of multiple linear regression analysis of predictors of plasma HDL cholesterol (in mmol/L) in 67 children referred for hypercholesterolemia

<table>
<thead>
<tr>
<th>Variable</th>
<th>B (95% CI)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple carbohydrate (g/d)</td>
<td>(-0.0045 (-0.0088, -0.0003))</td>
<td>0.036</td>
</tr>
<tr>
<td>Complex carbohydrate (g/d)</td>
<td>(-0.0037 (-0.0088, 0.0007))</td>
<td>0.100</td>
</tr>
<tr>
<td>Saturated fatty acid (g/d)</td>
<td>0.0154 (-0.0040, 0.0347)</td>
<td>0.118</td>
</tr>
<tr>
<td>Monounsaturated fatty acid (g/d)</td>
<td>(-0.0207 (-0.0456, 0.0040))</td>
<td>0.099</td>
</tr>
<tr>
<td>Polyunsaturated fatty acid (g/d)</td>
<td>0.0189 (-0.0112, 0.0330)</td>
<td>0.328</td>
</tr>
<tr>
<td>Dietary carbohydrate (mg/d)</td>
<td>0.0001 (-0.0006, 0.0008)</td>
<td>0.711</td>
</tr>
<tr>
<td>Log triacylglycerol (mmol/L)</td>
<td>(-0.1969 (-0.4954, 0.1016))</td>
<td>0.192</td>
</tr>
<tr>
<td>Age (y)</td>
<td>0.1397 (-0.0047, 0.3277)</td>
<td>0.15</td>
</tr>
</tbody>
</table>

*Multiple \(R^2 = 0.376\) and \(P = 0.0004\) for the overall regression model including the above variables. All nutrients were energy adjusted.
These differences may be due in part to variability in the type of simple and complex carbohydrates ingested and the type and amount of fat in the diet. In our study, the relation between complex carbohydrates and HDL cholesterol was not significant in either the bivariate or multiple regression models. Although the 95% CIs of the regression coefficients for complex and simple carbohydrates overlapped in our multiple regression model, an independent effect of complex carbohydrate is not supported by our data. Nevertheless, our study does not exclude an independent effect of complex carbohydrates on HDL cholesterol. Further studies to describe the effects of complex and simple carbohydrates on lipids in children may uncover relations not observed in our study.

Metabolic studies have shown that diets high in polyunsaturated fatty acids compared with saturated fatty acids lead to a marked decrease in HDL cholesterol in adult volunteers (34). When monounsaturated fatty acid is exchanged for saturated fatty acid, HDL cholesterol tends not to fall (35). Further dietary studies are needed to identify types of fat and carbohydrate intakes that will lead to optimal LDL-cholesterol as well as HDL-cholesterol concentrations (36–38).

One factor that may have contributed to our finding of an inverse association between simple carbohydrate intake and HDL cholesterol is that most (84%) of the study children were consuming fat-restricted diets. Restriction of the variability in total fat and saturated fatty acid intake may have reduced the influence of these dietary factors in our study group, thereby permitting detection of the inverse relation between simple carbohydrate intake and HDL cholesterol. Additional research is needed to address this hypothesis.

The 3-d diet records for the children in our study were kept by a parent or guardian, so that effects of self-reporting by children were minimized. When properly completed, diet records provide a detailed estimate of dietary intakes; however, the 3-d record may not provide an accurate estimate of habitual food intake because of sampling error (39). Nevertheless, our dietary findings using food records were comparable with those seen in other studies in children. Using 3-d food records in hypercholesterolemic children following dietary protocols, Kuehl et al (24) reported fat intakes varying from 24.2% to 28.1% of total energy. Using 24-h dietary recalls, the dietary intervention group of the Dietary Intervention Study in Children (4) study reported an intake of 28.5 ± 5.8% of energy from fat, also similar to the 24.9 ± 5.2% of energy from fat observed in our study.

Our study examined the effect of diet in hypercholesterolemic children aged 2–10 y. This age range was chosen to minimize the effects of smoking, alcohol ingestion (40), and puberty. We assumed that our younger study subjects had negligible rates of cigarette smoking and alcohol use. Children from 2 to 10 y of age are almost always prepubertal, thereby excluding the effects on HDL cholesterol associated with puberty (41, 42). Although boys tend to have slightly higher HDL-cholesterol concentrations than girls in the age range of the children we studied, there are no dramatic changes in HDL-cholesterol concentrations associated with growth and development between 2 and 10 y of age (18, 20).

The mechanisms whereby diet influences HDL cholesterol are unknown. An increase in carbohydrate ingestion may increase production of VLDL cholesterol (2), leading to an elevation of triacylglycerols, which is in turn associated with decreases in HDL cholesterol (43, 44). In our study subjects, the mean triacylglycerol concentration was 1.15 ± 0.51 mmol/L, so that elevated triacylglycerol concentrations were not a major contributor to the low HDL-cholesterol concentration.

Several limitations of our study should be considered in interpreting the findings. A single measurement of plasma lipids was used although day-to-day variations in total and HDL cholesterol are known to occur (45). Additionally, subjects in our study were highly motivated to enter the study and their families were able to complete 3-d diet records. Such subjects may not represent the general population (39). We did not attempt to validate the diets of these children by home visits or other dietary assessments. The potential effects of sampling errors from use of the food record are likely to be random. These random measurement errors would have attenuated rather than accounted for the inverse association seen between simple carbohydrate intake and plasma HDL cholesterol.

We examined the relation between diet and HDL cholesterol in hyperlipidemic children only. Similar findings might not be...

![Figure 3](https://academic.oup.com/ajcn/article-abstract/67/6/1147/4666043/blob/1152_STARC_ET_AL_Figure3)

**FIGURE 3.** Relation between simple carbohydrate intake and HDL cholesterol in hypercholesterolemic children ($n = 67; r = -0.40, P < 0.001$).

![Figure 4](https://academic.oup.com/ajcn/article-abstract/67/6/1147/4666043/blob/1152_STARC_ET_AL_Figure4)

**FIGURE 4.** Relation between simple carbohydrate intake and HDL cholesterol in hypercholesterolemic children. Children were divided into quartiles based on intake of simple carbohydrate. Means and 95% CIs of HDL-cholesterol concentrations are shown. Quartile 1 ($n = 17$) was significantly different ($P < 0.05$) from both quartile 4 ($n = 17$) and quartile 3 ($n = 16$).
seen in children with normal lipid concentrations. The children in our study were almost all white and the relation between simple carbohydrate intake and plasma HDL cholesterol in other races, especially African Americans, who are known to have higher HDL cholesterol (18), is unknown. In addition, trans fatty acids may lower HDL cholesterol (46). Because intakes of trans fatty acids were not calculated in this study, their potential effects on HDL cholesterol and other lipoproteins are not known. Furthermore, this study did not address how a low-fat diet would affect the ratio of LDL to HDL cholesterol in hypercholesterolemic children. A low-fat, high-carbohydrate diet may improve the ratio of LDL to HDL cholesterol, leading to a more favorable lipid profile despite reductions in HDL cholesterol. Finally, our study was cross-sectional in design so that we do not know how changes in diet would affect HDL cholesterol in an intervention trial; therefore, specific dietary recommendations should not be made on the basis of this study.

The main finding in our study was an inverse relation between intakes of simple carbohydrate and concentrations of HDL cholesterol in hyperlipidemic children consuming reduced-fat diets. Current recommendations for the dietary management of hypercholesterolemic children are based on reducing total and saturated fatty acids and cholesterol intakes to reduce LDL-cholesterol concentrations. Because diet may also affect other components of the lipid profile, further studies to investigate these relations are needed.

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