

Plasma lipids in Turkish children: impact of puberty, socioeconomic status, and nutrition on plasma cholesterol and HDL

Robert W. Mahley,^{1,*†,§,***} Perihan Arslan,^{§§} Gülden Pekcan,^{§§} Guy M. Pépin,^{*,††} Ayşen Ağaçdiken,^{***} Nilgün Karaağaoğlu,^{§§} Neslişah Rakıcioğlu,^{§§} Berat Nursal,^{§§} Pinar Dayanıklı,^{††} K. Erhan Palaoglu,^{††} and Thomas P. Bersot^{*,†,§}

Gladstone Institute of Cardiovascular Disease,^{*} Cardiovascular Research Institute,[†] and Departments of Medicine[§] and Pathology,^{**} University of California, San Francisco, CA 94141-9100; American Hospital,^{††} Istanbul 80200, Turkey; Department of Nutrition and Dietetics,^{§§} Hacettepe University, Ankara, Turkey; and Department of Cardiology,^{***} Kocaeli University, Izmit, Turkey

Abstract In Turkish adults, HDL cholesterol (HDL-C) levels are 10–15 mg/dl lower than those of adults in western Europe and the United States. In this study, we determined whether HDL-C levels in Turks are low from birth to adulthood and assessed the effect of socioeconomic status (SES) on plasma lipids and lipoproteins. Analyses of cord blood from 105 Turkish newborns showed low levels of plasma cholesterol (~60 mg/dl) and HDL-C (~30 mg/dl), consistent with results from other Western ethnic groups. Prepubescent 8- to 10-year-old Turkish boys and girls of upper (n = 82) and lower (n = 143) SES had high HDL-C levels (50–60 mg/dl) similar to those of western European children. However, the cholesterol (154–158 mg/dl) and HDL-C (55–58 mg/dl) levels of upper SES children were ~25 and ~12 mg/dl higher, respectively, than those of lower SES children. Height, weight, skinfold thickness, and estimated body fat were greater in the upper SES children and appeared to reflect dietary differences. Upper SES children consumed more total fat (~35% vs. 25% of total calories), including more saturated fat of animal origin, and less carbohydrate (~50% vs. 62% of total calories), consistent with their elevated plasma cholesterol levels. Carbohydrate intake correlated inversely with the HDL-C level. The HDL-C levels in the prepubescent children, especially those of higher SES, who consumed diets more like western Europeans, decreased markedly to adult levels, with males exhibiting a ~20 mg/dl decrease (from 58 to 37 mg/dl) and females a ~13 mg/dl decrease (from 55 to 42 mg/dl). SES did not affect HDL-C levels in adults. **□** The profound decrease may reflect alterations in androgen/estrogen balance in Turks at puberty and a modulation of hepatic lipase affecting HDL-C levels.—Mahley, R. W., P. Arslan, G. Pekcan, G. M. Pépin, A. Ağaçdiken, N. Karaağaoğlu, N. Rakıcioğlu, B. Nursal, P. Dayanıklı, K. E. Palaoglu, and T. P. Bersot. **Plasma lipids in Turkish children: impact of puberty, socioeconomic status, and nutrition on plasma cholesterol and HDL.** *J. Lipid Res.* 2001. 42: 1996–2006.

Supplementary key words dietary carbohydrates • saturated fat • androgens • triglycerides • neonates • cord blood lipids

HDL cholesterol (HDL-C) levels are typically 10–15 mg/dl lower in Turkish adults than in Europeans and North Americans, and >50% of Turkish men and 25% of Turkish women have levels <35 mg/dl (1). These findings have been confirmed by Onat and associates (2, 3). These low HDL-C levels appear to be largely of genetic origin because they are also observed in Turks living in Germany and the United States (4, 5). Furthermore, in both Turkish men and women, the low HDL-C level is associated with 25–30% greater hepatic lipase activity than in white American controls (5) and is characterized by low levels of HDL₂ and LpA-I, consistent with elevated hepatic lipase levels (6). In addition, socioeconomic status (SES) has a major impact on fasting cholesterol levels, which are 30–40 mg/dl higher in Turkish men and women of high SES than in those of low SES. However, SES does not affect HDL-C levels in adults (1).

In this study, we sought to determine if HDL-C levels in Turks are low throughout life. We also assessed the effects of SES on plasma lipids and lipoproteins. To achieve these goals, we measured lipid and lipoprotein levels in neonates and 8- to 10-year-old school children and compared the values with those in older Turkish subjects reported previously (1). As will be shown, Turkish newborns and prepubescent children have HDL-C levels similar to those of other ethnic groups of western European origin. After puberty, however, the HDL-C levels decrease markedly to the levels seen in Turkish adults. SES was associated with significant differences in plasma cholesterol and HDL-C levels in the prepubescent children.

Abbreviations: BMI, body mass index; HDL-C, HDL cholesterol; RDA, recommended daily allowance; SES, socioeconomic status.

¹To whom correspondence should be addressed at the Gladstone Institute of Cardiovascular Disease, P.O. Box 419100, San Francisco, CA 94141-9100.

EXPERIMENTAL PROCEDURES

Study participants

Two groups of subjects were studied: full-term, healthy Turkish newborns delivered by cesarean section without complications at the American Hospital in Istanbul, Turkey, and prepubescent 8- to 10-year-old Turkish school children from Ankara, Turkey. The study protocol was approved by the Ethics Committee of Hacettepe University, the Turkish Ministry of Education, and the Committee on Human Research of the University of California, San Francisco. Written informed consent was obtained from the parents of all participants before the study.

The American Hospital is a private hospital, and the patients are typically of higher SES. The 8- to 10-year-olds of lower and higher SES were recruited from two schools. The lower SES children were from a public school serving a region of Ankara where employment status, educational levels, living conditions, and lifestyles were classified as low SES. The upper SES children were from a private competitive school serving high-income families. Interviewers visited the classrooms to answer questions. Biodata concerning lifestyle and health status were obtained by interviewing the parents and children. None of the 8- to 10-year-old girls had experienced menarche.

For studies in newborns, cord blood was obtained at delivery in EDTA-containing tubes. For studies in school children, blood samples were obtained after a 12-h fast (no food after 9 PM); the blood was drawn into tubes containing EDTA (for lipid analyses) or fluoride (for glucose analysis). All samples were immediately placed on ice and centrifuged within 4 h and then were stored at -70°C and transported to the laboratory on dry ice. On the same day blood was drawn, anthropometric measurements (height, weight, waist, hip, and mid-upper arm circumferences, and skinfold thickness) were made by a trained examiner.

Blood lipid and glucose analyses

Plasma cholesterol, triglyceride, and HDL-C concentrations were measured enzymatically in the lipid diagnostic laboratory of the American Hospital. This laboratory has been certified as a lipid reference laboratory by the Centers for Disease Control (1). HDL-C levels were measured after VLDL and LDL were precipitated with phosphotungstic acid and magnesium. Kits for the lipid and glucose assays were from Boehringer-Mannheim (Mannheim, Germany). A multichannel analyzer (Hitachi, Tokyo, Japan) was used for the colorimetric enzymatic determinations of cholesterol (Monotest Cholesterol, CHOD-PAP), triglyceride (Peridochrom Triglyceride, GPO-PAP), and glucose (Glucose, GOD-PAP) (5, 6). The Friedewald calculation was used to estimate LDL cholesterol (LDL-C) values (7).

Anthropometric measurements

The weight of children wearing minimal clothing was measured to the nearest 0.1 kg with a portable electronic scale (Tanita). Each time it was moved, the scale was recalibrated with standardized

weights. Height to the nearest 0.1 cm was measured with a fiberglass tape. Body mass index (BMI) was calculated as weight (kg)/height (m^2). Body circumferences were measured with subjects in the standing position. Hip and waist (just above the iliac crest) circumferences were measured to the nearest 0.1 cm. Triceps, biceps, subscapular, and suprailiac skinfold thicknesses were measured to the nearest 1.0 mm with a Holtain caliper. All measurements were obtained as described by Lohman, Roche, and Martorell (8).

The percentage of body fat was estimated from triceps and subscapular skinfold thicknesses by using the equations of Slaughter et al. (9): $1.21(\text{triceps} + \text{subscapular}) - 0.008(\text{triceps} + \text{subscapular})^2 - 1.7$ for males and $1.33(\text{triceps} + \text{subscapular}) - 0.013(\text{triceps} + \text{subscapular})^2 - 2.5$ for females. If the sum of the triceps and subscapular measurements was >35 mm, the following equations were used: $0.783(\text{triceps} + \text{subscapular}) + 1.6$ for males and $0.546(\text{triceps} + \text{subscapular}) + 9.7$ for females.

Dietary intake

Food intake was assessed by dietary records over three consecutive days, including one weekend day. Nutritionists with extensive experience in determining food intake in Turkey instructed the children individually on how to complete the food records and how to estimate or measure the food portions. The food records were reviewed daily by the dietitians during an interview with the subject and, if needed, with the parents. The data were analyzed with SPSS version 9 statistical software. Food composition tables were used to calculate the energy and nutrient intakes (10). The data were compared with the recommended daily allowances (RDA) (11).

Comparison data

In a previous study (1), we measured lipid and lipoprotein levels in Turkish men and women 15–19, 20–24, 35–39, and 50–54 years of age. In conjunction with the data from Turkish newborns and prepubescent school children, these values provide a picture of the changes in lipid and lipoproteins in Turks from birth through adulthood.

Statistical analyses

All values are reported as the mean \pm SD. The Shapiro-Wilk test was used to determine whether outcome variables were normally distributed (12). Because this analysis showed that most variables were not distributed normally, the Mann-Whitney U test was used. A P value <0.05 was considered significant. Pearson correlation coefficients were used to assess associations between independent variables.

RESULTS

Anthropometric measurements and lipid levels

Neonates. One hundred five newborns were enrolled in the study. Their anthropometric data and plasma lipid and lipoprotein levels are shown in **Table 1**. The total cholesterol (~ 60 mg/dl), triglycerides (~ 24 mg/dl), LDL-C

TABLE 1. Anthropometric data and plasma lipid levels (mean \pm SD) in Turkish newborns delivered by cesarean section

	Gestation	Weight	Length	BMI	Total Cholesterol (range)	LDL-C	HDL-C (range)	Triglycerides (range)
	weeks	kg	cm	kg/m ²			mg/dl	
Males (n = 68)	38 \pm 2	3.37 \pm 0.52	50 \pm 2	14 \pm 1	56 \pm 15 (34–98)	23 \pm 9	29 \pm 9 (11–52)	24 \pm 9 (10–46)
Females (n = 37)	38 \pm 2	3.24 \pm 0.44	49 \pm 2	14 \pm 1	61 \pm 18 (37–124)	24 \pm 10	32 \pm 12 (15–68)	23 \pm 9 (11–42)

BMI, body mass index; LDL-C, LDL cholesterol; HDL-C, HDL cholesterol.

TABLE 2. Age and anthropometric data (mean \pm SD) in 8- to 10-year-old Turkish children according to gender and socioeconomic status (SES)

	Boys (n = 102) ^a		Girls (n = 121) ^b	
	Upper SES	Lower SES	Upper SES	Lower SES
Age (months)	109.4 \pm 10.6 ^c	110.3 \pm 9.8 ^c	110.0 \pm 9.7 ^d	110.1 \pm 9.7 ^d
Height (cm)	138.3 \pm 7.3	129.7 \pm 6.2	137.5 \pm 7.0	130.5 \pm 7.1
<50th percentile (%)	30	71	32	64
>50th percentile (%)	70	29	68	36
>75th percentile (%)	46	5	36	12
Weight (kg)	33.5 \pm 9.1	27.0 \pm 4.2	34.1 \pm 8.1	27.9 \pm 5.8
<50th percentile (%)	38	71	37	68
>50th percentile (%)	62	29	63	32
>75th percentile (%)	46	12	40	12
BMI (kg/m ²)	17.3 \pm 3.5 ^c	16.0 \pm 1.6 ^c	17.9 \pm 3.3	16.3 \pm 2.3
BMI \geq 19 kg/m ² (%) ^e	22	8	33	9
BMI \geq 23 kg/m ² (%)	11	0	9	5
Waist (cm)	61.3 \pm 9.6	55.9 \pm 4.4	59.5 \pm 7.1	55.6 \pm 5.5
Hip (cm)	73.7 \pm 8.9	66.8 \pm 4.3	74.0 \pm 7.5	67.8 \pm 6.1
Waist/hip ratio	0.8 \pm 0.05 ^c	0.8 \pm 0.04 ^c	0.8 \pm 0.05 ^d	0.8 \pm 0.04 ^d
Arm circumference (cm)	20.2 \pm 3.6	17.3 \pm 1.8	20.4 \pm 2.9	18.0 \pm 2.3
Triceps (mm)	11.0 \pm 6.1	6.6 \pm 2.3	12.6 \pm 4.7	8.7 \pm 4.0
Biceps (mm)	6.6 \pm 4.2	4.3 \pm 1.7	7.8 \pm 3.5	5.7 \pm 2.5
Subscapular (mm)	8.4 \pm 6.8	5.0 \pm 1.7	9.6 \pm 5.9	6.8 \pm 4.9
Suprailiac (mm)	10.3 \pm 9.5	5.2 \pm 2.2	12.5 \pm 8.0	7.8 \pm 5.7
Predicted body fat (%)	17.7 \pm 9.9	11.1 \pm 3.8	19.7 \pm 6.9	14.3 \pm 6.2

^a Data were incomplete for one boy.

^b Data were incomplete for one girl.

^c Not significantly different comparing the upper and lower SES boys ($P > 0.05$); other values are significantly different ($P < 0.05$).

^d Not significantly different comparing the upper and lower SES girls ($P > 0.05$); other values are significantly different ($P < 0.05$).

^e Internationally acceptable definition of overweight and obese in a 9-year-old male or female child is ≥ 19 and ≥ 23 kg/m², respectively (20).

(~24 mg/dl), and HDL-C (~30 mg/dl) levels in cord blood were low and similar to those observed in other populations (13–18). There were no significant gender differences in lipid levels or in anthropometric measurements.

Prepubescent school children. Two hundred twenty-five 8- to 10-year-olds participated in the study. Their anthropometric data are shown in **Table 2**, and their plasma lipid and lipoprotein levels are shown in **Table 3**. There were 143 children (65 boys and 78 girls) in the lower SES group and 82 children (38 boys and 44 girls) in the upper SES group. Biodata and fasting blood samples were obtained from all participants.

The mean ages of the upper and lower SES groups were similar, but most of the anthropometric measures were lower in the lower SES group (Table 2). Upper SES children tended to be taller and heavier; about 60–70% were above the 50th percentile for height and weight, as determined from standard growth curves (19), compared with 30–40% of lower SES children. Upper SES children had higher BMIs, but the difference was significant only in girls ($P = 0.0025$). Nevertheless, higher percentages of upper SES boys and girls were overweight (≥ 19 kg/m²) or obese (≥ 23 kg/m²), as defined by international standards (20). Skinfold thickness and arm circumference were greater in the upper SES boys and girls than in the lower SES children, as was the estimated percentage of body fat, but the

waist/hip ratio was not. These differences in anthropometric measurements suggested significant differences in nutrition between the two SES groups.

Plasma lipid and lipoprotein levels increased significantly during childhood (Table 3). Compared with values in Turkish newborns and consistent with other observations worldwide (16, 21–26), total cholesterol and triglyceride levels more than doubled to ~140 and 65 mg/dl, respectively, in Turkish 8- to 10-year-olds. HDL-C levels also increased from ~30 mg/dl in newborns to ~50 mg/dl in both boys and girls. However, there were striking differ-

TABLE 3. Plasma lipid and glucose levels (mean mg/dl \pm SD) of 8- to 10-year-old Turkish children

	Total Cholesterol	HDL-C	Triglycerides	Glucose
	mg/dl			
Boys				
Total group (n = 103)	138 \pm 26	50 \pm 13	65 \pm 29	85 \pm 8
Upper SES (n = 38)	154 \pm 20 ^a	58 \pm 14 ^a	58 \pm 18	85 \pm 7
Lower SES (n = 65)	129 \pm 25	45 \pm 10	70 \pm 33	85 \pm 8
Girls				
Total group (n = 122)	143 \pm 26	49 \pm 12	68 \pm 24	82 \pm 8
Upper SES (n = 44)	158 \pm 25 ^a	55 \pm 12 ^a	67 \pm 24	84 \pm 6
Lower SES (n = 78)	134 \pm 22	45 \pm 10	69 \pm 25	81 \pm 9

^a $P < 0.01$, upper versus lower SES.

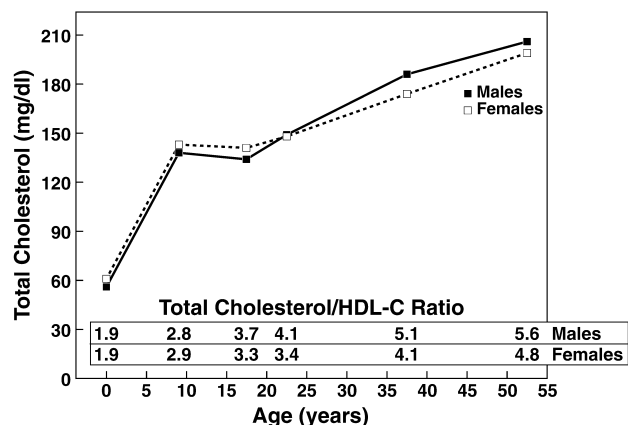


Fig. 1. Total plasma cholesterol levels increase markedly from birth to prepuberty (8–10 years of age) and slowly thereafter in adulthood. The total cholesterol/HDL-C ratio increases to high levels in adulthood as plasma cholesterol levels rise. The total cholesterol levels for ages 15–19, 20–24, 35–39, and 50–54 years have been published previously (1).

ences between the SES groups. Total cholesterol levels were ~20 mg/dl higher and HDL-C levels were 10–13 mg/dl higher in the upper SES children. Triglyceride levels in the SES groups were similar, as were blood glucose levels. Within the SES groups, there were no major gender differences in any of the lipid parameters.

Changes in plasma lipids with age

The changes in plasma total cholesterol levels in neonates, prepubescent children, and adults are illustrated in **Fig. 1**. After puberty, the cholesterol levels did not increase significantly in the 15- to 19-year-olds but did increase after 20–24 years of age and thereafter. Similar

TABLE 4. Effect of being overweight on plasma total cholesterol and HDL-C levels (mg/dl) and on the total cholesterol/HDL-C ratio in 8- to 10-year-old Turkish children

	Boys		Girls	
	Upper SES	Lower SES	Upper SES	Lower SES
Total cholesterol				
BMI ≥ 19 kg/m ²	161 \pm 26 (n = 8)	137 \pm 33 (n = 5)	163 \pm 24 (n = 14)	136 \pm 25 (n = 7)
BMI <19 kg/m ²	152 \pm 18 (n = 29)	128 \pm 24 (n = 60)	154 \pm 24 (n = 29)	136 \pm 22 (n = 71)
HDL-C				
BMI ≥ 19 kg/m ²	52 \pm 14 (n = 8)	41 \pm 5 (n = 5)	50 \pm 9 ^a (n = 14)	45 \pm 8 (n = 7)
BMI <19 kg/m ²	60 \pm 13 (n = 29)	45 \pm 10 (n = 60)	58 \pm 12 (n = 29)	45 \pm 8 (n = 71)
Total cholesterol/HDL-C				
BMI ≥ 19 kg/m ²	3.2 \pm 0.7 ^b (n = 8)	3.4 \pm 0.8 (n = 5)	3.4 \pm 0.7 ^c (n = 14)	3.1 \pm 0.7 (n = 7)
BMI <19 kg/m ²	2.6 \pm 0.6 (n = 29)	2.9 \pm 0.7 (n = 60)	2.8 \pm 0.9 (n = 29)	3.1 \pm 0.8 (n = 71)

^a $P = 0.05$ versus BMI <19 kg/m².

^b $P = 0.039$ versus BMI <19 kg/m².

^c $P = 0.008$ versus BMI <19 kg/m².

P values calculated with the Mann-Whitney ranking test.

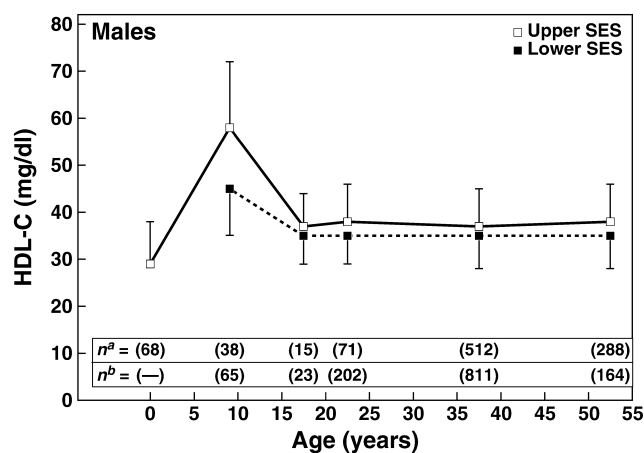


Fig. 2. Plasma HDL-C levels (mean \pm SD) for males are plotted against age in years and according to upper and lower SES. The cord blood values are from newborns delivered at the private American Hospital in Istanbul, at which most patients are of upper SES. SES of subjects 15–19, 20–24, 35–39, and 50–54 years of age was determined as previously described (1) and is based on family salaries in the upper versus lower half of typical Turkish family salaries. n^a , number of samples from the upper SES group; n^b , number of samples from the lower SES group.

plasma cholesterol levels have been reported for Turkish adults (27). The total cholesterol/HDL-C ratio remained low in prepubescent children but rose significantly after puberty, reflecting both an increase in total cholesterol and a decrease in HDL-C levels. This ratio was also significantly increased in upper SES 8- to 10-year-olds who were overweight (BMI ≥ 19 kg/m²) (**Table 4**). These results reflect the trend toward higher total cholesterol levels and lower HDL-C levels in these overweight boys and girls.

Figures 2 and 3 illustrate the increase in HDL-C levels in Turkish boys and girls from birth to 8–10 years of age and the marked postpubescent decrease to adult levels.

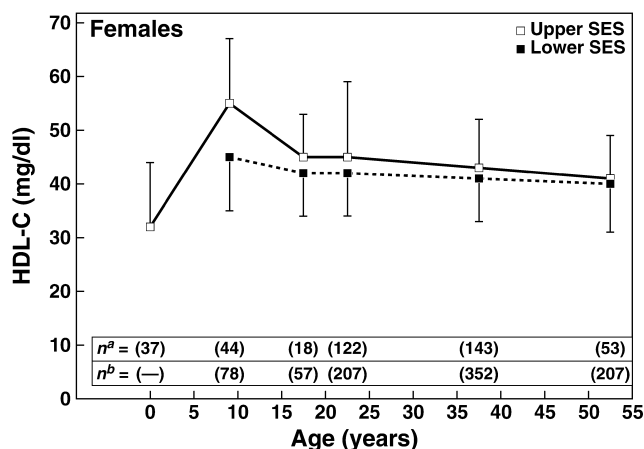


Fig. 3. Plasma HDL-C levels (mean \pm SD) for females are plotted against age in years and according to upper and lower SES, as described in the legend to **Fig. 2**. n^a , number of samples from the upper SES group; n^b , number of samples from the lower SES group.

In upper SES males, HDL-C levels were 21 mg/dl lower in 15- to 19-year-olds than in 8- to 10-year-olds (58 vs. 37 mg/dl); in lower SES males, HDL-C was ~10 mg/dl lower in the older group than in the younger group (45 vs. 35 mg/dl) (Fig. 2). In upper SES females, the HDL-C levels were 11 mg/dl lower in the 15- to 19-year-olds than in the 8- to 10-year-olds (55 vs. 44 mg/dl); in lower SES females, however, the HDL-C levels were only 3 mg/dl lower in the older group than in the younger group (45 vs. 42 mg/dl) (Fig. 3). Interestingly, SES did not affect HDL-C levels in adult males or females (age >15 years) (Figs. 2 and 3).

Prepubescent school children: socioeconomics and dietary differences

Analysis of the food records of the 8- to 10-year-old participants in the study revealed significant differences between the upper and lower SES groups (Table 5). Energy intake was higher in the upper SES children than in the lower SES children. Despite this difference, the average caloric intake was >80% of RDA in both groups. Total protein intake, especially of animal protein, was significantly higher in the upper SES boys and girls. The RDA for total protein exceeded 100% for all children in both groups. Furthermore, upper SES children consumed more total fat, especially animal fat, than did the lower SES children.

Nutrient intake as a percentage of total calories consumed by 8- to 10-year-old children in the upper and

TABLE 5. Energy and macronutrient intakes (mean \pm SD) for 8- to 10-year-old Turkish school children according to SES

	Upper SES	Lower SES
Boys (n = 101) ^a		
Energy (kcal)	2,002 \pm 479	1,840 \pm 600
% RDA	100 \pm 24	92 \pm 30
Total protein (g) ^c	68 \pm 15	57 \pm 18
% RDA	244 \pm 52	204 \pm 64
Animal protein (g) ^b	42 \pm 13	22 \pm 11
Plant protein (g) ^b	26 \pm 7	35 \pm 12
Total fat (g) ^b	79 \pm 25	53 \pm 23
Animal fat ^b	39 \pm 15	22 \pm 12
Plant fat ^c	40 \pm 15	31 \pm 16
Carbohydrates (g)	262 \pm 76	285 \pm 99
Crude fiber (g)	3.6 \pm 1.2	3.9 \pm 1.9
Cholesterol (mg) ^d	274 \pm 110	218 \pm 167
Girls (n = 118) ^a		
Energy (kcal)	1,851 \pm 363	1,597 \pm 437
% RDA	93 \pm 18	80 \pm 22
Total protein (g) ^b	65 \pm 15	50 \pm 16
% RDA	230 \pm 53	179 \pm 57
Animal protein (g) ^b	42 \pm 12	20 \pm 11
Plant protein (g) ^b	23 \pm 6	30 \pm 11
Total fat (g) ^b	79 \pm 18	47 \pm 17
Animal fat (g) ^b	40 \pm 12	20 \pm 12
Plant fat (g) ^b	39 \pm 12	27 \pm 12
Carbohydrates (g)	228 \pm 51	246 \pm 74
Crude fiber (g)	3.5 \pm 1.3	3.7 \pm 1.7
Cholesterol (mg) ^c	261 \pm 106	185 \pm 101

RDA, recommended daily allowance.

^a Data were incomplete for two boys and four girls.

^b $P < 0.0001$ by Mann-Whitney test (upper versus lower SES).

^c $P < 0.001$ by Mann-Whitney test (upper versus lower SES).

^d $P < 0.01$ by Mann-Whitney test (upper versus lower SES).

TABLE 6. Macronutrient intake as a percentage of total calories (mean \pm SD) for 8- to 10-year-old Turkish children according to SES

	Upper SES	Lower SES	P^a
	%	%	
Boys (n = 101) ^b			
Total protein	13.9 \pm 1.8	12.5 \pm 1.9	0.0004
Animal protein	8.6 \pm 2.1	4.9 \pm 8.6	<0.0001
Plant protein	5.3 \pm 1.1	7.6 \pm 1.5	<0.0001
Total fat	35.1 \pm 5.8	26.1 \pm 7.0	<0.0001
Animal fat	17.5 \pm 5.1	10.8 \pm 5.2	<0.0001
Plant fat	17.7 \pm 3.9	15.3 \pm 5.4	0.008
Carbohydrate	52.4 \pm 6.2	61.7 \pm 7.1	<0.0001
Girls (n = 118) ^b			
Total protein	13.9 \pm 1.9	12.6 \pm 1.9	0.0006
Animal protein	9.0 \pm 2.2	5.0 \pm 2.2	<0.0001
Plant protein	4.9 \pm 0.9	7.6 \pm 1.4	<0.0001
Total fat	38.3 \pm 4.8	26.5 \pm 6.2	<0.0001
Animal fat	19.2 \pm 4.7	11.2 \pm 5.7	<0.0001
Plant fat	19.1 \pm 3.9	15.3 \pm 5.3	<0.0001
Carbohydrate	49.3 \pm 6.0	61.5 \pm 6.1	<0.0001

^a Comparing upper versus lower SES children (Mann-Whitney test).

^b Data were incomplete for two boys and four girls.

lower SES groups is shown in Table 6. Total protein constituted ~13% of calories in both groups, but protein consumption was slightly but significantly higher in the upper SES group. The major difference, however, was the higher percentage of calories from animal protein in the upper SES group. The percentage of calories from fat, especially animal fat, was also strikingly higher in the upper SES group, who consumed ~10% more fat calories than did lower SES children. Importantly, the difference in the total caloric intake in the lower SES group was made up by carbohydrate. Carbohydrate represented ~62% of total calories in the lower SES boys and girls compared with 52% for the upper SES boys and 49% for the upper SES girls. Thus, the diets of the upper SES children were relatively high in fat, especially animal fat, and low in carbohydrate, as is typical of many western European diets, whereas the diets of the lower SES children were relatively high in carbohydrates and low in fat.

Correlations between dietary constituents and plasma lipids and lipoproteins of 8- to 10-year-old Turkish children

As shown in Table 3, the upper SES boys and girls had significantly higher total cholesterol levels than did the lower SES children. Consistent with previous studies (28–32), the higher fat content and especially the higher animal fat content (which would equate to higher levels of saturated fats) correlated positively with the total cholesterol levels (Table 7). In addition, there was a positive association between the higher total and animal fat consumption and higher HDL-C levels seen in boys and girls (Table 7). Plasma triglycerides were little affected by the content or type of fat.

In Figs. 4 and 5, the values for total cholesterol and HDL-C are plotted against total dietary fat (percentage of calories) for the upper and lower SES boys and girls. The distribution patterns for the lipid/lipoprotein values for

TABLE 7. Pearson correlation coefficients between dietary constituents (as a percentage of total energy intake) and plasma lipids and lipoproteins of 8- to 10-year-old Turkish children^a

	Total Cholesterol		HDL-C		Triglyceride	
	Boys	Girls	Boys	Girls	Boys	Girls
Carbohydrate	-0.33 ^b	-0.34 ^b	-0.31 ^c	-0.41 ^c	0.19	0.13
Total fat	0.37 ^b	0.35 ^b	0.30 ^c	0.38 ^c	-0.16	-0.11
Animal fat	0.32 ^b	0.36 ^b	0.34 ^b	0.35 ^b	-0.14	-0.07
Plant fat	0.19	0.08	0.07	0.16	-0.08	-0.09

^a Data were incomplete for two boys and four girls.

^b $P \leq 0.001$.

^c $P \leq 0.01$.

the upper and lower SES children illustrate the effect of dietary fat intake on these two parameters.

On the other hand, as shown in Table 7, dietary intake of carbohydrates was inversely associated with total cholesterol and HDL-C. Carbohydrate intake was not significantly associated with triglyceride levels in the 8- to 10-year-old children. **Figure 6** clearly illustrates the inverse relationship between HDL-C levels and carbohydrate as a percentage of calories in the diet in the upper and lower SES children. As reported by Katan and associates (32, 33), the higher the carbohydrate content of a diet, the lower the HDL-C levels. The lower SES boys and girls, who consumed ~10% more carbohydrates than did the upper SES children, had ~10 mg/dl lower HDL-C levels.

DISCUSSION

The results of numerous studies of western European populations and whites in the United States are summarized in **Table 8**, which shows the typical changes in plasma total cholesterol and HDL-C levels with age (13–18, 21–26). Typically, newborns have low total cholesterol and HDL-C levels, with males and females having similar levels. Very similar results were obtained for Turkish newborns. In older children, plasma total cholesterol and HDL-C levels are higher, but there are no major gender

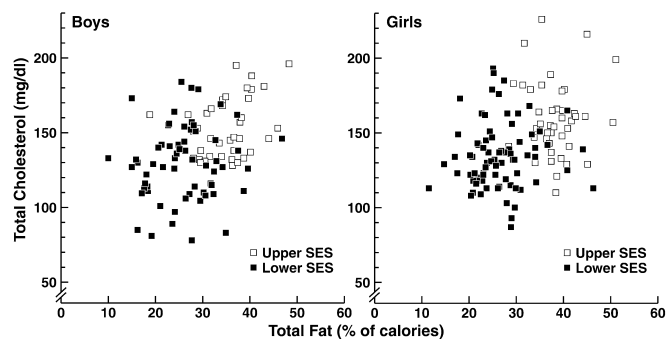


Fig. 4. The total plasma cholesterol levels for 8- to 10-year-old boys and girls are plotted against the percentage of total calories consumed as total fat. The lower SES boys and girls tended to have lower cholesterol levels and a lower fat diet than upper SES boys and girls.

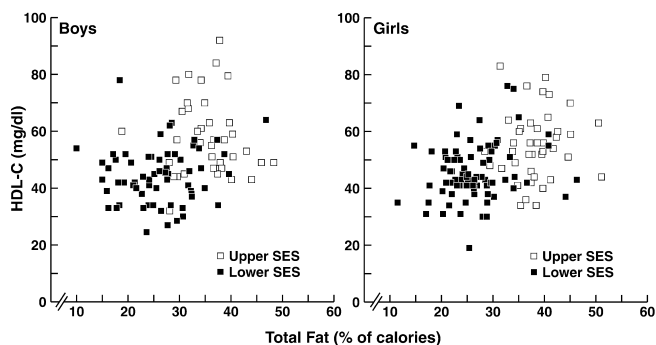


Fig. 5. The HDL-C levels for 8- to 10-year-old boys and girls are plotted against the percentage of total calories consumed as total fat. The lower SES boys and girls tended to have lower HDL-C levels and a lower fat diet than the upper SES boys and girls.

differences before puberty. For example, Webber et al. (34) found that the total cholesterol levels in 8- to 9-year-old Anglo-American children from four states (California, Louisiana, Minnesota, and Texas) were ~165–170 mg/dl, and their HDL-C levels were 50–60 mg/dl irrespective of gender. Similar values were observed in African-American and Latino children (34). In **Fig. 7**, the HDL-C levels for pre- and postpubescent upper and lower SES groups of Turkish males and females are compared with HDL-C levels before and after puberty in U.S., Australian, and Japanese populations (35). The HDL-C levels for prepubescent males and females in the United States and Australia were very similar to those of the upper SES Turkish children (56 and 58 vs. 58 mg/dl for males; 53 and 57 vs. 55 mg/dl for females, respectively) and were lower than those in Japanese 8- to 10-year-olds (61–62 mg/dl). It is likely that the Turkish upper SES groups are most appropriate for comparison with other populations because the diets of the upper SES children are most similar to the typical European diet, as will be discussed.

The difference, when comparing the upper SES Turkish HDL-C levels with those of the U. S. and Australian (and especially the Japanese) populations, was the strikingly greater decrease in the HDL-C levels of the Turks after puberty. After puberty, HDL-C levels in American and Australian males decrease to typical adult levels of ~47 mg/dl,

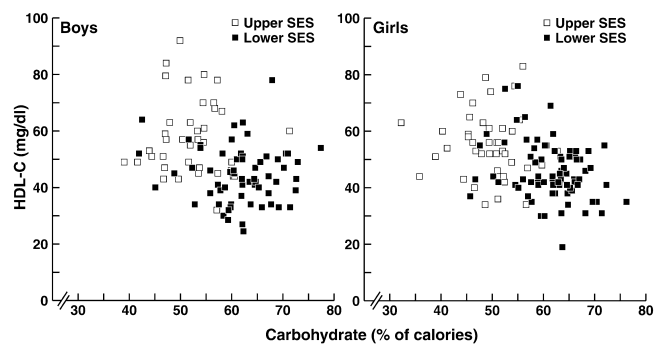


Fig. 6. The lower SES boys and girls had lower HDL-C levels and consumed more carbohydrates than the upper SES boys and girls.

TABLE 8. Changes in plasma lipid levels with age in western Europeans and Turks

	Western Europeans		Turks ^a	
	Total Cholesterol	HDL-C	Total Cholesterol	HDL-C
	<i>mg/dl</i>		<i>mg/dl</i>	
Newborns	<100	~30	~60	~30
Male/female differences	None	None	None	None
Children	135–165	55–60	~155	55–58
Male/female differences	None	None	None	None
Young Adults	>160	47–57	>160	37–42
Male/female differences	M>F	M:55–60→47 F:55–60→~55	M>F	M:58→37 F:55→42

^a Data obtained from the upper SES groups.

whereas in females they decrease much less to typical adult levels of ~55–57 mg/dl (Fig. 7). Japanese males and females continue to have higher HDL-C levels even after puberty. On the other hand, the HDL-C levels in Turkish boys dropped from a group mean of ~50 mg/dl to 37 mg/dl and remained at 36–37 mg/dl during adulthood. The HDL-C levels in Turkish girls decreased to ~42 mg/dl and remained at 40–42 mg/dl in adulthood regardless of SES.

Total cholesterol levels of upper SES Turks, before and after puberty, are comparable to typical values for western European and white U.S. populations, as shown in Table 8 (21–26, 34–38). This observation is of concern because the greatly reduced HDL-C levels of postpubescent Turks would be predicted to substantially increase coronary heart disease risk compared with other populations with similar total cholesterol levels but higher HDL-C levels (39–42).

The mechanism for the significant reduction in HDL-C levels after puberty remains to be determined. It has been postulated that androgen production may play a major role in modulating HDL-C levels at puberty (43–45), and increased androgen production in both males and females is one of the prominent changes at this developmental stage. Consistent with a possible role for androgens in the modulation of HDL-C levels in Turks is the observation by Hergenç et al. (46) that Turks have lower levels of sex hormone binding globulin, which would result in increased blood levels of free bioactive testosterone in both males and females. Hepatic lipase production is regulated by androgens and to a lesser extent by estrogens. High levels of androgens are associated with increased levels and activity of hepatic lipase (43–45). Because high levels of hepatic lipase activity and protein mass are characteristic of Turkish males and females (5), the dynamic balance between androgens and estrogens in Turks may prove to be important in explaining their low HDL-C levels.

The effect of socioeconomic status on plasma lipids in the Turkish society is intriguing. Previously, we demonstrated that cholesterol levels are higher in Turks with higher salaries and higher levels of education (i.e., higher SES) (1). This is not typically seen in western European or American populations; in fact, plasma cholesterol levels are often inversely associated with salary and education (47–49). The more affluent Turks appear to consume diets higher in saturated fats, which is supported by the increase in saturated fatty acid content of plasma cholesterol esters and triglycerides in the affluent Turks (1). However, SES has no effect on mean HDL-C levels in Turkish adults (Figs. 2 and 3).

Unlike Turkish adults, differences in SES were associated with profound effects on the plasma levels of HDL-C in prepubescent 8- to 10-year-old Turkish children. Both the plasma cholesterol and HDL-C levels were lower in

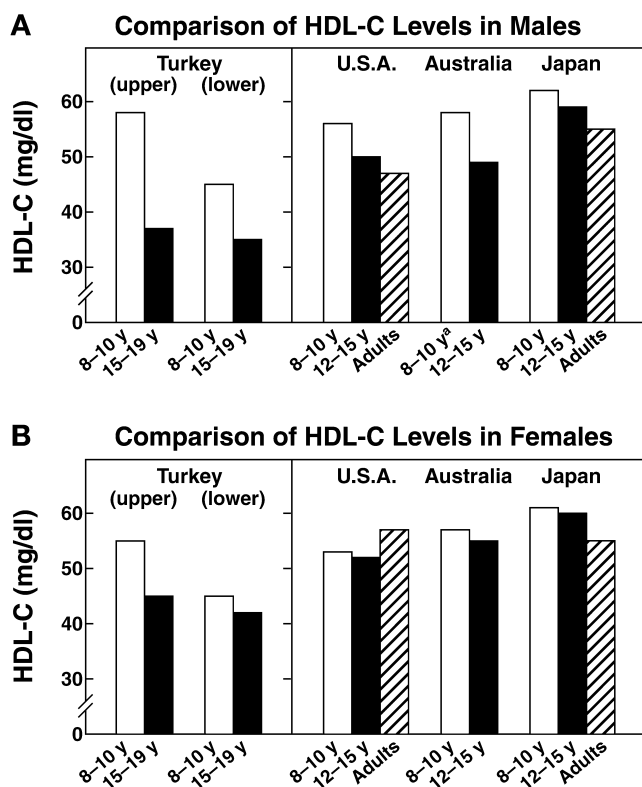


Fig. 7. Plasma HDL-C levels in males (A) and females (B) from Turkey are compared with values from the United States, Australia, and Japan. Data for 8- to 10-year-old males and females from the United States, Australia, and Japan are from the study by Dwyer et al. (35) and are shown for comparison with data from this study of 8- to 10-year-old Turkish males and females and from a previous study of 15- to 19-year-old Turks (1). Adult HDL-C levels for the U.S. population [NHANES data; >20 years of age (71)], and the adult Japanese population [40–69 years of age (72)] are shown for comparison.

the lower SES boys and girls. In fact, in prepubescent boys and girls of lower SES, the HDL-C levels were only 45 mg/dl. Thus, the lower SES Turkish girls have relatively low HDL-C before puberty (~45 mg/dl) and only a small decrease in HDL-C levels after puberty (~42 mg/dl), whereas the lower SES boys have a 10 mg/dl decrease in HDL-C after puberty. By contrast, the upper SES boys have a 21 mg/dl decrease and the lower SES boys have a 10–13 mg/dl decrease in HDL-C levels after puberty.

Both developmental status and nutritional status were clearly different in the upper and lower SES children. Most of the anthropometric measurements were lower in the lower SES boys and girls (Table 2). For example, many in the lower SES group tended to be underweight and short in stature. Whereas 46% of the upper SES boys and 40% of the upper SES girls were above the 75th percentile for weight for their ages, only 12% of the lower SES boys and girls were above the 75th percentile for weight. Likewise, 46% of the upper SES boys and 36% of the upper SES girls were above the 75th percentile for height, compared with only 5% of the lower SES boys and 12% of the lower SES girls. Similar anthropometric differences related to SES have been observed in Turkey (50–52) and elsewhere in the world (53).

Despite the fact that energy and macronutrient intakes approached the established RDA for age and gender for the children in both SES groups, the diets were very different. The upper SES groups consumed a diet that was higher in fat, especially animal fat, which would be expected to be high in saturated fatty acids. On the other hand, the children in the lower SES groups consumed a diet higher in carbohydrates. Previously, other surveys conducted in Ankara and elsewhere in Turkey demonstrated that upper SES children consumed diets high in animal protein and fat (especially lamb), whereas the typical diet of the lower SES children was enriched in carbohydrates, including cereal grains (flour in bread, rice, and bulgur) and sugar (51, 54, 55).

The effect of diet on plasma lipids and lipoproteins has been studied extensively in clinical trials and free-living populations, and although diet is clearly an important determinant, various environmental and metabolic factors intervene to modulate the dietary effects (53). However, it is reasonable to discuss the differences in the lipids and lipoproteins (especially the HDL-C levels) of the upper and lower SES 8- to 10-year-old boys and girls in the context of the dietary differences observed.

The effect on HDL-C levels of the higher carbohydrate content (62% vs. 52% of total calories) of the diet consumed by the lower SES children is consistent with prior observations. Low HDL-C levels associated with high-carbohydrate diets were reported as early as 1966 by Levy, Lees, and Fredrickson (56). Subsequently, numerous trials of healthy individuals have established that exchanging carbohydrates for fat decreases HDL-C levels significantly (57–63). It appears that the effect of carbohydrates on HDL-C occurs not only with the consumption of simple sugars but also with complex carbohydrates (64, 65). The impact of high-carbohydrate, low-fat diets on atherogene-

sis is, however, controversial (66, 67). Although such a diet reduces LDL-C levels as well as HDL-C levels, replacing dietary saturated fat with a balanced intake of mono- and polyunsaturated fat, rather than carbohydrates, may be more appropriate (68).

In a meta-analysis of 27 trials, Mensink and Katan (32) examined the effects of exchanging fat in the diet for carbohydrates, including isocaloric exchanges of saturated and unsaturated fatty acids. Increased consumption of fat at the expense of carbohydrate was associated with an increase in HDL-C levels, with saturated fatty acids having the greatest effects (~5 mg/dl per 10% of energy replaced) and increasing unsaturation having lesser effects (~3 mg/dl per 10% of energy replaced). Increased saturated fat content was also associated with increased LDL-C, so there was no change in the ratio of HDL-C to LDL-C despite the increase in HDL-C associated with replacing carbohydrates with saturated fat. Replacement of carbohydrate by unsaturated fat raised HDL-C less than did replacement with saturated fat, but the HDL-C to LDL-C ratio increased because unsaturated fat did not raise LDL-C levels (32). When carbohydrate was replaced by fat, triglyceride levels declined independently of the type of fat. In our study, the upper SES 8- to 10-year-old boys and girls consumed diets ~10% higher in fat content than did the lower SES children, and the exchange was accounted for almost entirely by decreased carbohydrate and increased saturated fatty acid-rich animal fats in the upper SES children.

Knuiman et al. (36, 57) examined the effects of different diets on HDL-C levels in 8- to 9-year-old prepubescent boys from various countries and reviewed data from studies of 7- to 10-year-old boys. They concluded that HDL-C levels correlate strongly and negatively with the percentage of energy derived from carbohydrates and correlate positively with the proportion of energy derived from fats, especially saturated fats. Multiple regression analyses revealed that the strongest predictor of the HDL-C levels is the carbohydrate content of the diet and not the fat content. In these prepubescent boys, differences in obesity and physical activity were slight and did not affect the HDL-C differences associated with the carbohydrate content of the diet. Knuiman et al. (57) compared HDL-C levels with the percentage of calories derived from carbohydrates in prepubescent boys from five different countries (Finland, the Netherlands, Italy, Ghana, and the Philippines). As shown in **Fig. 8**, these data demonstrate that the higher the carbohydrate content of the diet, the lower the HDL-C levels. A 10% change in energy derived from carbohydrate is associated with a ~10 mg/dl difference in HDL-C levels across several different ethnic groups.

The HDL-C levels of the 8- to 10-year-old Turkish boys from our study are entirely in agreement with those plotted in Fig. 8. Others have shown that diets high in starch and sucrose are associated with lower HDL-C levels at comparable body weight (69). SES is far more complex than diet alone, and it is likely that many factors interact to produce the differences in HDL-C levels that we have observed (53, 70). Future studies will attempt to

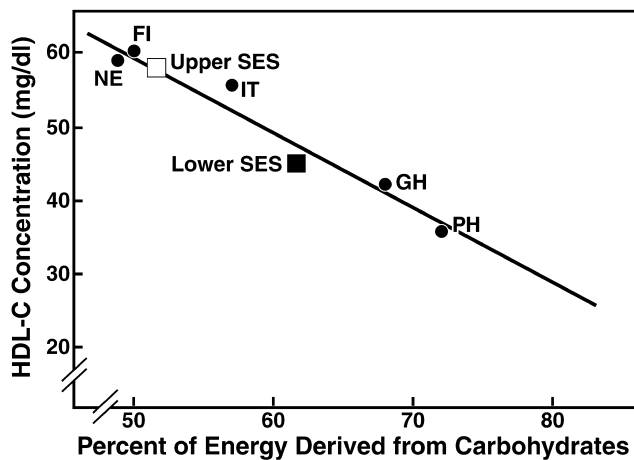


Fig. 8. Data from Knuiman et al. [modified from reference (57)] illustrate that 8- to 10-year-old boys in different countries who consume diets that are higher in the percentage of energy derived from carbohydrates are associated with lower levels of HDL-C. Data from the upper and lower SES Turkish 8- to 10-year-old boys are plotted on the graph. The data point represented by the percentage of total calories as carbohydrates (52%) and the HDL-C levels (58 mg/dl) of the upper SES Turkish boys (open square) falls exactly on the regression line. Likewise, the data point corresponding to the carbohydrate content (62%) and HDL-C level (45 mg/dl) of the lower SES 8- to 10-year-old Turkish boys (filled square) is in good agreement. FI, Finland; NE, the Netherlands; IT, Italy; GH, Ghana; PH, the Philippines.

elucidate psychosocial, environmental, educational, physical activity, and metabolic differences among the groups. [14](#)

We are indebted to our associates at the American Hospital, Istanbul, and to Sibel Tanir in the Gladstone Research Laboratory (Istanbul). We thank Sylvia Richmond and Catharine Evans for manuscript preparation and Stephen Ordway and Gary Howard for editorial assistance. We wish to acknowledge the generous support of the American Hospital, the J. David Gladstone Institutes, and Merck Sharp and Dohme, Turkey.

Manuscript received 10 May 2001 and in revised form 6 August 2001.

REFERENCES

- Mahley, R. W., K. E. Palaoglu, Z. Atak, J. Dawson-Pepin, A-M. Langlois, V. Cheung, H. Onat, P. Fulks, L. L. Mahley, F. Vakar, S. Özbayrakçı, O. Gökdemir, and W. Winkler. 1995. Turkish Heart Study: lipids, lipoproteins, and apolipoproteins. *J. Lipid Res.* **36**: 839–859.
- Onat, A., M. A. Büyükbese, E. Ural, I. Keles, D. Ural, E. Ince, B. Kurban, and V. Sansoy. 1997. Marmara bölgesi hakkında HDL-kolesterol ile fibrinojen düzeyleri ve bazı etkenlerle ilişkileri. *Arch. Turk. Soc. Cardiol.* **25**: 520–525.
- Onat, A., B. Yıldırım, N. Uslu, N. Gürbüz, I. Keles, A. Çetinkaya, H. Aksu, Ö. Uysal, and V. Sansoy. 1999. Plasma lipoproteins and apolipoproteins in Turkish adults: overall levels, associations with other risk parameters and HDL's role as a marker of coronary risk in women. *Arch. Turk. Soc. Cardiol.* **27**: 72–79.
- Lüttmann, S., A. von Eckardstein, W. Wei, H. Funke, E. Köhler, R. W. Mahley, and G. Assmann. 1994. Electrophoretic screening for genetic variation in apolipoprotein C-III: identification of a novel apoC-III variant, apoC-III(Asp45→Asn), in a Turkish patient. *J. Lipid Res.* **35**: 1431–1440.

- Bersot, T. P., G. L. Vega, S. M. Grundy, K. E. Palaoglu, P. Atagündüz, S. Özbayrakçı, O. Gökdemir, and R. W. Mahley. 1999. Elevated hepatic lipase activity and low levels of high density lipoprotein in a normotriglyceridemic, nonobese Turkish population. *J. Lipid Res.* **40**: 432–438.
- Mahley, R. W., J. Pépin, K. E. Palaoglu, M. J. Malloy, J. P. Kane, and T. P. Bersot. 2000. Low levels of high density lipoproteins in Turks, a population with elevated hepatic lipase: high density lipoprotein characterization and gender-specific effects of apolipoprotein E genotype. *J. Lipid Res.* **41**: 1290–1301.
- Friedewald, W. T., R. I. Levy, and D. S. Fredrickson. 1972. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin. Chem.* **18**: 499–502.
- Lohman, T. G., A. F. Roche, and R. Martorell. 1988. Anthropometric Standardization Reference Manual. Human Kinetics Books, Champaign, IL.
- Slaughter, M. H., T. G. Lohman, R. A. Boileau, C. A. Horswill, R. J. Stillman, M. D. Van Loan, and D. A. Bembien. 1988. Skinfold equations for estimation of body fatness in children and youth. *Hum. Biol.* **60**: 709–723.
- Paul, A. A., and D. A. T. Southgate. 1978. McCance and Widdowson's The Composition of Foods. 4th revised and extended edition. Elsevier/North-Holland Biomedical Press, Amsterdam.
- National Research Council. 1989. Recommended Dietary Allowances. 10th edition. National Academy Press, Washington, D.C.
- Shapiro, S. S., and M. B. Wilk. 1965. An analysis of variance test for normality (complete samples). *Biometrika.* **52**: 591–611.
- Winkler, L., B. Schlag, and E. Goetze. 1977. Concentration and composition of the lipoprotein classes in human umbilical cord serum. *Clin. Chim. Acta.* **76**: 187–191.
- Hardell, L. I. 1981. Serum lipids and lipoproteins at birth based on a study of 2815 newborn infants. Concentrations and distributions of triglycerides and cholesterol. *Acta Paediatr. Scand. Suppl.* **285**: 5–10.
- Davis, P. A., T. M. Forte, A. V. Nichols, and C. B. Blum. 1983. Umbilical cord blood lipoproteins. Isolation and characterization of high density lipoproteins. *Arteriosclerosis.* **3**: 357–365.
- Viikari, J., H. K. Åkerblom, T. Nikkari, A. Seppänen, M. Uhari, E. Pesonen, M. Dahl, P-L. Lähde, M. Pietikäinen, and P. Suoninen. 1985. Atherosclerosis precursors in Finnish children and adolescents. IV. Serum lipids in newborns, children and adolescents. *Acta Paediatr. Scand. Suppl.* **318**: 103–109.
- Diaz, M., C. Leal, J. Ramon y Cajal, M. D. Jimenez, H. Martinez, M. Pocovi, and F. Grande. 1989. Cord blood lipoprotein-cholesterol: relationship birth weight and gestational age of newborns. *Metabolism.* **38**: 435–438.
- Loughrey, C. M., E. Rimm, G. Heiss, and N. Rifai. 2000. Race and gender differences in cord blood lipoproteins. *Atherosclerosis.* **148**: 57–65.
- Kuczmarski, R. J., C. L. Ogden, L. M. Grummer-Strawn, K. M. Flegal, S. S. Guo, R. Wei, Z. Mei, L. R. Curtin, A. F. Roche, and C. L. Johnson. 2000. CDC growth charts: United States. Advance data from vital and health statistics, no. 314. National Center for Health Statistics, Hyattsville, MD.
- Cole, T. J., M. C. Bellizzi, K. M. Flegal, and W. H. Dietz. 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. *Br. Med. J.* **320**: 1240–1243.
- Lipid Research Clinics. 1980. Population Studies Data Book. The Prevalence Study. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health (NIH Publ. No. 80–1527), Bethesda, MD.
- Freedman, D. S., S. R. Srinivasan, J. L. Cresanta, L. S. Webber, and G. S. Berenson. 1987. Serum lipids and lipoproteins. *Pediatrics.* **80**(Suppl.): 789–796.
- Webber, L. S., S. R. Srinivasan, W. A. Wattigney, and G. S. Berenson. 1991. Tracking of serum lipids and lipoproteins from childhood to adulthood. The Bogalusa Heart Study. *Am. J. Epidemiol.* **133**: 884–899.
- Brotans, C., A. Ribera, R. M. Perich, D. Abrodos, P. Magaña, S. Pablo, D. Terradas, F. Fernández, and G. Permanyer. 1998. Worldwide distribution of blood lipids and lipoproteins in childhood and adolescence: a review study. *Atherosclerosis.* **139**: 1–9.
- Berenson, G. S., S. R. Srinivasan, R. R. Frerichs, and L. S. Webber. 1979. Serum high density lipoprotein and its relationship to car-

- diovascular disease risk factor variables in children—the Bogalusa Heart Study. *Lipids*. **14**: 91–98.
26. Aguilera, F., L. Lupiañez, D. Magaña, E. Planells, F. J. Mataix, and J. Llopis. 1996. Lipid status in a population of Spanish schoolchildren. *Eur. J. Epidemiol.* **12**: 135–140.
 27. Onat, A., G. Surdum-Avcı, M. Senocak, E. Örneç, and Y. Gözükar. 1992. Plasma lipids and their interrelationship in Turkish adults. *J. Epidemiol. Community Health*. **46**: 470–476.
 28. Keys, A., J. T. Anderson, and F. Grande. 1965. Serum cholesterol response to changes in the diet. IV. Particular saturated fatty acids in the diet. *Metabolism*. **14**: 776–787.
 29. Hegsted, D. M., L. M. Ausman, J. A. Johnson, and G. E. Dallal. 1993. Dietary fat and serum lipids: an evaluation of the experimental data. *Am. J. Clin. Nutr.* **57**: 875–883.
 30. Yu, S., J. Derr, T. D. Etherton, and P. M. Kris-Etherton. 1995. Plasma cholesterol—predictive equations demonstrate that stearic acid is neutral and monounsaturated fatty acids are hypocholesterolemic. *Am. J. Clin. Nutr.* **61**: 1129–1139.
 31. Hegsted, D. M., R. B. McGandy, M. L. Myers, and F. J. Stare. 1965. Quantitative effects of dietary fat on serum cholesterol in man. *Am. J. Clin. Nutr.* **17**: 281–295.
 32. Mensink, R. P., and M. B. Katan. 1992. Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. *Arterioscler. Thromb.* **12**: 911–919.
 33. Katan, M. B. 1998. Effect of low-fat diets on plasma high-density lipoprotein concentrations. *Am. J. Clin. Nutr.* **67**(Suppl.): 573S–576S.
 34. Webber, L. S., V. Osganian, R. V. Luepker, H. A. Feldman, E. J. Stone, J. P. Elder, C. L. Perry, P. R. Nader, G. S. Parcel, S. L. Broyles, and S. M. McKinlay. 1995. Cardiovascular risk factors among third grade children in four regions of the United States. The CATCH study. *Am. J. Epidemiol.* **141**: 428–439.
 35. Dwyer, T., H. Iwane, K. Dean, Y. Odagiri, T. Shimomitsu, L. Blizard, S. Srinivasan, T. Nicklas, W. Wattigney, M. Riley, and G. Berenson. 1997. Differences in HDL cholesterol concentrations in Japanese, American, and Australian children. *Circulation*. **96**: 2830–2836.
 36. Knuiman, J. T., R. J. J. Hermus, and J. G. A. J. Hautvast. 1980. Serum total and high density lipoprotein (HDL) cholesterol concentrations in rural and urban boys from 16 countries. *Atherosclerosis*. **36**: 529–537.
 37. Freedman, D. S., S. R. Srinivasan, L. S. Webber, and G. S. Berenson. 1987. Divergent levels of high density lipoprotein cholesterol and apolipoprotein A-I in children. The Bogalusa Heart Study. *Arteriosclerosis*. **7**: 347–353.
 38. Webber, L. S., D. W. Harsha, G. T. Phillips, S. R. Srinivasan, J. W. Simpson, and G. S. Berenson. 1991. Cardiovascular risk factors in Hispanic, white, and black children: the Brooks County and Bogalusa Heart Studies. *Am. J. Epidemiol.* **133**: 704–714.
 39. Miller, N. E. 1987. Associations of high-density lipoprotein subclasses and apolipoproteins with ischemic heart disease and coronary atherosclerosis. *Am. Heart J.* **113**: 589–597.
 40. Gordon, D. J., J. L. Probstfield, R. J. Garrison, J. D. Neaton, W. P. Castelli, J. D. Knoke, D. R. Jacobs, Jr., S. Bangdiwala, and H. A. Tyroler. 1989. High-density lipoprotein cholesterol and cardiovascular disease. Four prospective American studies. *Circulation*. **79**: 8–15.
 41. Assmann, G., and H. Schulte. 1992. Relation of high-density lipoprotein cholesterol and triglycerides to incidence of atherosclerotic coronary artery disease (the PROCAM experience). *Am. J. Cardiol.* **70**: 733–737.
 42. Vega, G. L., and S. M. Grundy. 1996. Hypoalphalipoproteinemia (low high density lipoprotein) as a risk factor for coronary heart disease. *Curr. Opin. Lipidol.* **7**: 209–216.
 43. Boberg, J., M. Boberg, R. Gross, J. D. Turner, J. Augustin, and W. V. Brown. 1979. Hepatic triglyceride and lipoprotein lipase activities of post-heparin plasma in normals and hypertriglyceridemics. *Uppsala J. Med. Sci.* **84**: 215–227.
 44. Kantor, M. A., A. Bianchini, D. Bernier, S. P. Sady, and P. D. Thompson. 1985. Androgens reduce HDL₂-cholesterol and increase hepatic triglyceride lipase activity. *Med. Sci. Sports Exerc.* **17**: 462–465.
 45. Applebaum-Bowden, D., S. M. Haffner, and W. R. Hazzard. 1987. The dyslipoproteinemia of anabolic steroid therapy: increase in hepatic triglyceride lipase precedes the decrease in high density lipoproteins, cholesterol. *Metabolism*. **36**: 949–952.
 46. Hergenç, G., H. Schulte, G. Assmann, and A. von Eckardstein. 1999. Associations of obesity markers, insulin, and sex hormones with HDL-cholesterol levels in Turkish and German individuals. *Atherosclerosis*. **145**: 147–156.
 47. Fulwood, R., W. Kalsbeek, B. Rifkind, R. Russell-Briefel, R. Muesing, J. LaRosa, and K. Lippel. 1986. Total serum cholesterol levels of adults 20–74 years of age. United States, 1976–80. In *Vital and Health Statistics*. National Center for Health Statistics, Series 11, No. 236. DHHS Pub. No. (PHS) 86–1686, U.S. Government Printing Office, Washington, DC.
 48. Liu, K., L. B. Cedres, J. Stamler, A. Dyer, R. Stamler, S. Nanas, D. M. Berkson, O. Paul, M. Lepper, H. A. Lindberg, J. Marquardt, E. Stevens, J. A. Schoenberger, R. B. Shekelle, P. Collette, S. Shekelle, and D. Garside. 1982. Relationship of education to major risk factors and death from coronary heart disease, cardiovascular diseases and all causes. Findings of three Chicago epidemiologic studies. *Circulation*. **66**: 1308–1314.
 49. Winkleby, M. A., D. E. Jatulis, E. Frank, and S. P. Fortmann. 1992. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am. J. Public Health*. **82**: 816–820.
 50. 15 Provinces Nutrition Education Surveillance Project. 1995. Ministry of Health and Hacettepe University Department of Nutrition and Diagnostics, Ankara, Turkey.
 51. Kocaoglu, B. A. 1996. Effects of Socioeconomic Conditions on Growth, Development and Obesity among Adolescents in Turkey. PhD Dissertation. Institute of Health Science, Nutrition and Diagnostics Programme, Hacettepe University, Ankara, Turkey.
 52. Pekcan, G. 1983. A survey on the nutritional and health status of school children between the ages of 10–12 years. (In Turkish.). *J. Nutr. Diet.* **12**: 43–56.
 53. Kaplan, G. A., and J. E. Keil. 1993. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. **88**: 1973–1998.
 54. Arslan, P., and I. Mert. 1997. Nutrition Education and Consumer Awareness. Inter-Country Workshop, Turkey. Food and Agriculture Organization of United Nations Near East Regional Office, Cairo, Egypt.
 55. Baysal, A. 1991. Food and nutrition policies in Turkey. In *Food and Nutrition Policy in Mediterranean Europe*. Proceedings of a WHO Symposium, Rome, 1990. A. Ferro-Luzzi, E. Cialfa, and C. Leclercq, editors. National Institute of Nutrition, Rome, Italy. 131–145.
 56. Levy, R. I., R. S. Lees, and D. S. Fredrickson. 1966. The nature of pre-beta (very low density) lipoproteins. *J. Clin. Invest.* **45**: 63–77.
 57. Knuiman, J. T., C. E. West, M. B. Katan, and J. G. A. J. Hautvast. 1987. Total cholesterol and high density lipoprotein cholesterol levels in populations differing in fat and carbohydrate intake. *Arteriosclerosis*. **7**: 612–619.
 58. Blum, C. B., R. I. Levy, S. Eisenberg, M. Hall III, R. H. Goebel, and M. Berman. 1977. High density lipoprotein metabolism in man. *J. Clin. Invest.* **60**: 795–807.
 59. Antonis, A., and I. Bersohn. 1961. The influence of diet on serum-triglycerides in South African white and Bantu prisoners. *Lancet*. **1**: 3–9.
 60. Brunner, D., J. Weissbort, M. Fischer, J. E. Bearman, K. Loebel, S. Schwartz, and S. Levin. 1979. Serum lipid response to a high-caloric, high-fat diet in agricultural workers during 12 months. *Am. J. Clin. Nutr.* **32**: 1342–1349.
 61. Brussaard, J. H., G. Dallinga-Thie, P. H. E. Groot, and M. B. Katan. 1980. Effects of amount and type of dietary fat on serum lipids, lipoproteins and apolipoproteins in man. A controlled 8-week trial. *Atherosclerosis*. **36**: 515–527.
 62. Kashyap, M. L., R. L. Barnhart, L. S. Srivastava, G. Perisutti, P. Vink, C. Allen, E. Hogg, D. Brady, C. J. Glueck, and R. L. Jackson. 1982. Effects of dietary carbohydrate and fat on plasma lipoproteins and apolipoproteins C-II and C-III in healthy men. *J. Lipid Res.* **23**: 877–886.
 63. Williams, P. T., R. M. Krauss, P. D. Wood, J. J. Albers, D. Dreon, and N. Ellsworth. 1985. Associations of diet and alcohol intake with high-density lipoprotein subclasses. *Metabolism*. **34**: 524–530.
 64. Mensink, R. P., and M. B. Katan. 1987. Effect of monounsaturated fatty acids versus complex carbohydrates on high-density lipoproteins in healthy men and women. *Lancet*. **1**: 122–125.
 65. Mensink, R. P., M. J. M. de Groot, L. T. van den Broeke, A. P. Severijnen-Nobels, P. N. M. Demacker, and M. B. Katan. 1989. Effects of monounsaturated fatty acids *v* complex carbohydrates on serum

lipoproteins and apoproteins in healthy men and women. *Metabolism*. **38**: 172–178.

66. Connor, W. E., and S. L. Connor. 1997. Should a low-fat, high-carbohydrate diet be recommended for everyone? The case for a low-fat, high-carbohydrate diet. *N. Engl. J. Med.* **337**: 562–563.
67. Katan, M. B., S. M. Grundy, and W. C. Willett. 1997. Should a low-fat, high-carbohydrate diet be recommended for everyone? Beyond low-fat diets. *N. Engl. J. Med.* **337**: 563–566.
68. Grundy, S. M. 1999. The optimal ratio of fat-to-carbohydrate in the diet. *Annu. Rev. Nutr.* **19**: 325–341.
69. Ernst, N., M. Fisher, W. Smith, T. Gordon, B. M. Rifkind, J. A. Little, M. A. Mishkel, and O. D. Williams. 1980. The association of plasma high-density lipoprotein cholesterol with dietary intake and alcohol consumption. The Lipid Research Clinics Program Prevalence Study. *Circulation*. **62**: IV-41–IV-52.
70. Heiss, G., W. Haskell, R. Mowery, M. H. Criqui, M. Brockway, and H. A. Tyroler. 1980. Plasma high-density lipoprotein cholesterol and socioeconomic status. The Lipid Research Clinics Program Prevalence Study. *Circulation*. **62**: IV-108–IV-115.
71. National Cholesterol Education Program. 1994. Second report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *Circulation*. **89**: 1333–1445.
72. Ueshima, H., M. Iida, T. Shimamoto, M. Konishi, M. Tanigaki, N. Nakanishi, Y. Takayama, H. Ozawa, S. Kojima, and Y. Komachi. 1982. High-density lipoprotein-cholesterol levels in Japan. *J. Am. Med. Assoc.* **247**: 1985–1987.