PAPERS AND ORIGINALS

Changes in blood lipids and blood pressure during adolescence

T J ORCHARD, M RODGERS, A J HEDLEY, J R A MITCHELL

Summary and conclusions

A total of 625 adolescents from three general practices participated in a cross-sectional study of cardiovascular disease risk factors. The girls had higher serum total and high density lipoprotein (HDL) cholesterol concentrations than the boys, while the boys had higher serum triglyceride concentrations. Smoking (equally prevalent in both sexes) was associated with lower HDL cholesterol concentrations, particularly in boys, while in girls use of oral contraceptives was associated with higher total cholesterol and lower HDL cholesterol concentrations. HDL and total cholesterol concentrations showed striking associations with age, height, and sexual maturation in boys, but not in girls. Triglyceride concentrations were associated with age in boys. Systolic blood pressure and serum urate concentrations were higher in boys and rose steeply with age, but no age association was seen for urate concentrations or systolic blood pressure in girls or for diastolic pressures in either sex. Girls, however, had higher diastolic pressures. There was a strong association between urate concentration and the other cardiovascular disease risk factors, especially HDL cholesterol.

Adolescence is associated with considerable changes in cardiovascular disease risk factors, and there are striking sex differences in these changes.

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Introduction

Men are more likely to suffer and die from coronary disease than women, and the sex difference is most striking when young men are compared with young women.[1] This disparity cannot be explained by known sex differences in the prevalence of risk factors such as smoking, hypertension, and hyperlipidaemia.[2] Premenopausal women were thought to be protected by their ovarian hormones but this concept has recently been challenged, and it has been suggested that men are placed at greater risk by their testicular hormones.[3] Attention has also been redirected to sex differences in the high density lipoproteins (HDL),[4 5] and prospective studies have shown a strong predictive link between HDL cholesterol and ischaemic heart disease.[6 7]

To clarify the relation between risk factors and sexual and physical maturation we have conducted a cross-sectional study of cardiovascular disease risk factors in adolescence. We now present our results for serum lipids, arterial blood pressure, and serum urate.

Subjects and methods

The subjects, aged 13-18 years, were identified from the age-sex registers of three general practices. In practices 1 (suburban Nottingham) and 3 (semi-rural Derbyshire) all those 13-18 year olds currently residing in the practice areas were eligible to participate, while in practice 2 (Nottingham city centre), which was about twice as large, a one-in-two systematic age-sex stratified sample of those registered was used. A letter of invitation was sent to subjects aged 16 or over, while for those aged 13-15 the parents were first asked to return a signed consent form.

A total of 1179 teenagers was initially considered eligible to participate. Of these 1179, 154 were rejected because they had left the area or because their date of birth had been incorrectly recorded, while 21 subjects were excluded for medical reasons (severe mental handicap, 15; recent operations, 2; Hodgkin's disease, 1; late pregnancy, 2; and hospitalisation, 1). Thirty subjects from practice 1 participated in a pilot study, so were excluded from the main study. Thus 974 adolescents remained eligible, of whom 625 (64%) agreed to participate. The subjects were seen at specially arranged clinics held at their general practitioner's surgery in an evening (5-8 pm) or on a Saturday morning (9-12 am). The survey team spent two months in each practice starting in mid-November 1978 (practice 1) and ending in mid-May 1979 (practice 3).

A full account of our methods (training procedures, measures of repeatability, interobserver and intraobserver variation, laboratory quality control, and external standardisation) will be published later, but the essential features were as follows.

A questionnaire was completed by the subjects when they arrived at the clinic, which included questions on biographical details; alcohol consumption[8]; smoking[9]; and personal, medical, and drug histories. Questions on leisure activities and sports were adapted from the method of Morris *et al*,[10] coded according to oxygen consumption and energy output,[11] and graded on a five-point scale. Social class was determined from the father's occupation.[12]

Examinations were performed by TJO, AJH, and a State-registered nurse. The pulse was counted for 30 seconds and then the blood pressure recorded on the left arm,[13] systolic and diastolic phase IV and V pressures being read to the nearest 2 mm Hg below. A random-zero sphygmomanometer (Hawksley Ltd, Northampton) was used for all recordings[14] with a cuff of appropriate size.[15] A second set of pulse and blood pressure readings was taken after a further five minutes' sitting; the mean of these two recordings was used in the analyses, both sets of blood pressures being recorded by the same observer for each patient. Height (to nearest 0·1 cm) and weight (to nearest 100 g below) were measured using a portable Harpenden stadiometer and Avery balance. Triceps and subscapular skinfold thicknesses were recorded to the nearest 0·1 mm using Harpenden skin callipers[16] and transformed to a logarithmic scale.[17] Sexual maturity[16] was assessed on a five-point scale by TJO for the boys (pubic hair and genital development) and by the nurse for the girls (pubic hair and breast development). Finally, 25 ml of venous blood was obtained from the antecubital fossa. Subjects attending on Saturday mornings were asked to fast for 12 hours before attendance.

Laboratory estimations—Serum HDL cholesterol was measured by the LRC method,[18] modified by Warnick and Albers.[19] In the pilot study there was no significant difference in HDL cholesterol concentration according to fasting status (fasting mean = 1·04 mmol/l, 40·0 mg/100 ml; two-hours postprandial mean = 1·05 mmol/l, 40·5 mg/100 ml), so all HDL cholesterol results were used irrespective of fasting status. Samples were analysed within six weeks of collection; most were precipitated within one week and the cholesterol estimations performed within two weeks. Before analysis samples were stored at 4°C for a week then frozen. All estimations were made with the CentrifiChem centrifugal analyser (Union Carbide Corporation, New York). Cholesterol concentrations were measured by an adaptation of the method of Roeschlau *et al*[20] and serum triglyceride concentrations with the Eskalab Triglyceride Reagent (Smith Kline Instruments, Welwyn Garden City, Herts) by a modification of Bucolo and David's method.[21] A uricase kit (Roche, Welwyn Garden City, Herts) was used for estimations of serum urate concentration.[22]

Statistical analyses[23]—Distributions and percentiles were based on the whole population, while for all other analyses non-white participants and those subjects taking drugs known to interfere with lipid metabolism were excluded, reducing the population available for analysis to 584-590 depending on the parameter studied. Statistical tests of significance, unless otherwise stated, were χ^2 (for qualitative variables) or one-way analyses of variance (for quantitative measures), an unpaired Student's *t* test being used where there was only one degree of freedom. Correlation coefficients were all Pearson product moment coefficients. Standardisation was achieved by using standard deviation scores based on the formula $(a_v - \bar{x}_v)/SD_v$, where $\bar{x}_v$ is the mean value of the variable being standardised in the class being standardised for, a_v is the individual's value, and SD_v is the standard deviation of all values in the relevant class being standardised for.

Results

Our response rate (mean 64%) varied with practice, age, and, to a lesser extent, sex (table I). Of the participants, 96% were British-born whites, 30% had already left school, and 17% were currently taking medication (including the contraceptive pill, which was used by a quarter of all 16 to 18-year-old girls).

The prevalence of smoking was similar in boys and girls (table II) and was positively associated with a history of minor illness such as upper respiratory tract infection, or with gastrointestinal disturbances (which had occurred in the past two weeks in 63% of smokers and 52% of non-smokers; $p < 0.05$).

Figure 1 shows the mean lipid results by age and sex. The girls had higher total cholesterol concentrations, largely reflecting their higher HDL concentrations. The lipid differences between the sexes were all significant ($p < 0.001$) but the only significant age association was with random triglyceride concentrations in boys, and similar patterns were seen when non-fasted subjects were excluded. Smokers had lower total and HDL cholesterol concentrations (table III), this trend being particularly apparent in the boys, but there was no difference in triglyceride concentration between smokers and non-smokers. Alcohol consumption was not related to any of the lipids on univariate analysis. The 16 to 18-year-old girls taking oral contraceptives had significantly higher total cholesterol and marginally lower HDL cholesterol concentrations, but no significant difference in triglyceride values was seen (table IV).

TABLE I—*Response rate by practice, age, and sex (percentages in parentheses)*

Practice or age group	Boys	Girls	Total
Practice 1	103 (66)	109 (64)	212 (65)
Practice 2	110 (76)	95 (71)	205 (74)
Practice 3	106 (61)	102 (52)	208 (56)
13–15 years	196 (75)	162 (65)	358 (70)
16–18 years	123 (57)	144 (58)	267 (57)
Total	319 (67)	306 (62)	625 (64)

TABLE II—*Percentage of smokers by age and sex*

Sex	Age group (years)		Total
	13–15	16–18	
Boys	10	25	16
Girls	9	27	18

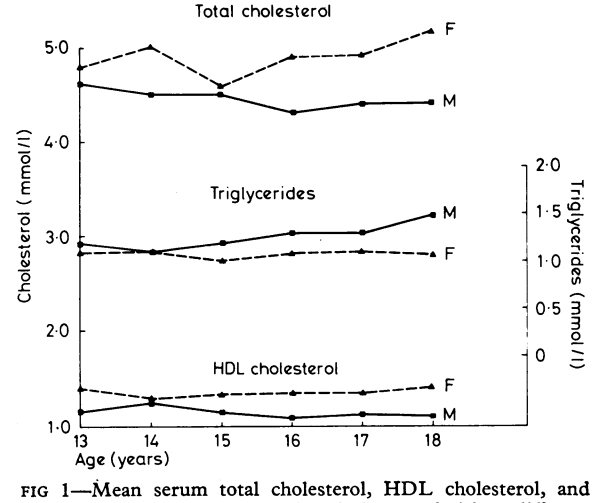

FIG 1—Mean serum total cholesterol, HDL cholesterol, and random triglyceride concentrations in boys and girls at different ages.

Conversion: SI to traditional units—Serum cholesterol: 1 mmol/l ≈ 38·7 mg/100 ml. Serum triglyceride: 1 mmol/l ≈ 88·6 mg/100 ml.

TABLE III—*Mean serum HDL and total cholesterol concentrations by age, sex, and smoking habit*

Age group (years) or sex	HDL cholesterol (mmol/l)		Total cholesterol (mmol/l)		No of cases
	Smokers	Non-smokers	Smokers	Non-smokers	
13–15	1·10*	1·27*	4·58	4·69	331
16–18	1·19	1·26	4·47*	4·80*	253
Boys	1·01**	1·17**	4·24*	4·56*	300
Girls	1·30	1·36	4·78	4·92	284
Total	1·16*	1·26*	4·50*	4·73*	584

Significance of differences between smokers and non-smokers: *0·05 > p > 0·01; **p < 0·001.

Conversion: SI to traditional units—Serum cholesterol: 1 mmol/l ≈ 38·7 mg/100 ml.

TABLE IV—*Mean serum total and HDL cholesterol concentrations and random and fasting serum triglyceride concentrations (mmol/l), and use of oral contraceptives in 16-18-year-old girls (numbers of subjects in parentheses)*

	Users	Non-users	Significance
Total cholesterol	5·44 (34)	4·85 (102)	0·01>p>0·001
HDL cholesterol	1·28 (34)	1·38 (101)	NS
Random triglyceride	1·16 (34)	1·04 (102)	NS
Fasting triglyceride	1·03 (7)	0·74 (27)	NS

Conversion: SI to traditional units—Serum cholesterol: 1 mmol/l ≈ 38·7 mg/100 ml. Serum triglyceride: 1 mmol/l ≈ 88·6 mg/100 ml.

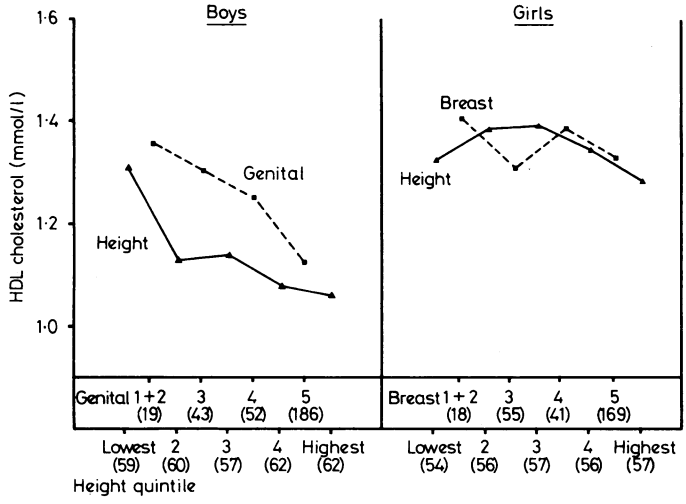

FIG 2—Mean serum HDL cholesterol concentrations according to height quintile and breast or genital development. Numbers of cases in parentheses.
Conversion: SI to traditional units—Serum cholesterol: 1 mmol/l ≈ 38·7 mg/100 ml.

volume of alcohol consumed by boys (0·29; p < 0·001), was lost on age standardisation.

Systolic blood pressure rose with age in boys but not in girls (fig 4). This sex difference and the difference by age in boys were significant (p < 0·001). Diastolic pressures did not change with age and were higher in girls (p < 0·001). Smoking habit did not correlate with blood pressure except for a lower systolic pressure in girl smokers (p < 0·05).

FIG 3—Mean serum urate concentrations in boys and girls at different ages.
Conversion: SI to traditional units—Serum urate: 1 mmol/l ≈ 16·8 mg/100 ml.

Higher sports-activity scores were associated with lower diastolic blood pressures in boys, even after age standardisation (p < 0·001). Age-standardised systolic blood pressures were related to height (r = +0·16; 0·01 > p > 0·001), to genital development (r = +0·14; 0·01 > p > 0·001), and to Quetelet index (r = +0·18; p < 0·001) in boys but not in girls. Diastolic pressure showed no such association in either sex. Urate concentrations correlated significantly with age-standardised systolic blood pressures in girls (r = +0·14; 0·05 > p > 0·01) but not in boys.

TABLE V—*Correlation coefficients for relation between serum lipid concentration (mmol/l) and developmental variables*

Lipid	Boys				Girls			
	Age	Height	Genital	Pubic hair	Age	Height	Breast	Pubic hair
Total cholesterol	−0·10*	−0·20**	−0·28**	−0·22**	+0·10*	−0·01	+0·07	+0·06
HDL cholesterol	−0·13*	−0·28**	−0·28**	−0·23**	+0·02	−0·03	−0·03	−0·02
Random triglyceride	+0·20**	+0·01	+0·05	+0·07	−0·02	−0·05	−0·03	−0·11*
Fasting triglyceride	+0·34**	+0·04	+0·10	+0·13	−0·00	+0·17	−0·05	−0·02

Significance levels: *0·05 > p > 0·01; **p < 0·001.
Conversion: SI to traditional units—Serum cholesterol: 1 mmol/l ≈ 38·7 mg/100 ml. Serum triglyceride: 1 mmol/l ≈ 88·6 mg/100 ml.

Figure 2 shows the relation between HDL cholesterol concentrations and height quintile and sexual maturation. A similar pattern was seen for total cholesterol concentrations, while triglyceride concentrations showed no relation in either sex. HDL cholesterol values fell with increasing height and sexual maturity in boys (p < 0·001) but not in girls. The correlation coefficients shown in table V confirm these associations and also show that triglyceride concentrations in boys were associated with age rather than with indices of sexual maturity.

Mean serum urate concentrations rose with age in boys (p < 0·001) but not in girls (fig 3). A similar, but inverse, figure to figure 2 could be drawn for boys, relating urate concentrations to height and maturity. The age-standardised correlation coefficient in boys was +0·26 (p < 0·001) between height and urate concentrations, +0·20 (p < 0·001) between urate concentrations and genital development, and −0·17 (0·01 > p > 0·001) between urate concentrations and HDL cholesterol concentrations. The results in girls showed no such correlations. Total cholesterol concentrations were not related to urate concentrations in either sex, while only in boys did fasting triglyceride concentrations correlate with urate concentrations (+0·22; 0·05 > p > 0·01). Urate values among smokers and non-smokers were similar, and a positive association between urate values and the

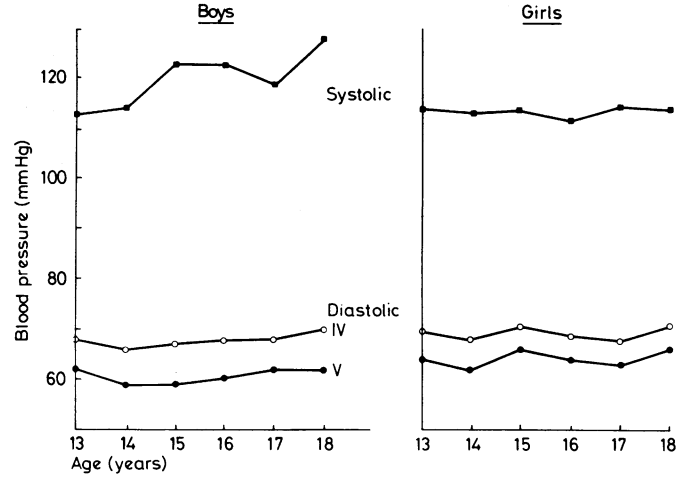

FIG 4—Mean systolic and diastolic (phase IV and V) blood pressures in boys and girls at different ages.

Discussion

Our response rate of 64% is comparable with most school-based and community-based studies.[24-30] Two school-based studies with higher response rates[31 32] did not include 18 year olds or provide appropriate age-specific response rates for comparison purposes. The response rate in our study was lowest in the older age groups and in practice 3.

Our study is the first sizable survey of cardiovascular disease risk factors in British adolescents. Our total cholesterol values are similar to those reported in many studies.[24 27-30] Our HDL cholesterol values are also broadly comparable with those in the LRC[26] and Rochester[25] studies, but the Cincinnati[31] and Bogalusa[32] studies showed higher HDL cholesterol values. The lower HDL cholesterol concentration in our cigarette smokers is also consistent with the findings in other studies,[33 34] though in our study the association was particularly strong for boys, which raises the possibility of differences either in inhalation patterns or in susceptibility between the sexes.

We have shown a relation between HDL cholesterol concentration and age, maturity, and height in boys but not in girls. This does not necessarily imply that male sexual maturation is solely responsible for the fall in HDL cholesterol concentration or that the correlations with age, height, and weight are merely secondary associations. The correlation between height and HDL cholesterol concentration in boys may be more than just a reflection of sexual development and may represent a direct influence of the growth spurt itself. If growth itself is important it is interesting to speculate whether the increased susceptibility of short-stature men to ischaemic heart disease[35 36] results from their growth pattern, since early growth spurts are known to be more intense and to result in lower adult height.[16] Further evidence that influences other than sexual maturation may be responsible for a fall in HDL cholesterol concentration in boys comes from a study of male Pima Indians aged 10-22 years, which failed to show any association between HDL cholesterol and the oestradiol:testosterone ratio.[37] Similarly, the Bogalusa study,[32 38] which showed a fall in HDL cholesterol values in boys with increasing maturity, also showed as great an effect with age, suggesting that sexual maturation alone cannot be the overriding influence.

In addition to the potentially unfavourable fall in HDL cholesterol concentration during adolescence, the boys in our study also showed adverse alterations in other cardiovascular risk factors, since during this period systolic blood pressure and serum urate concentration rose steeply. The association between urate concentration and HDL cholesterol concentration is particularly interesting and potentially significant because we have found that in boys urate concentration varied with age, sex, height, and maturity in the opposite way to HDL cholesterol concentration. These results, plus the recent evidence suggesting that urate concentration may be an independent risk factor in women,[39] and that a uricosuric agent (sulphinpyrazone) reduces mortality after myocardial infarction,[40] make it vitally important to clarify the role of uric acid in ischaemic heart disease.

We have shown that in a British population puberty and adolescence have profound effects on cardiovascular disease risk factors. During adolescence boys develop lower HDL cholesterol concentrations, higher serum urate concentrations and systolic blood pressures, and show a greater adverse effect of smoking on HDL cholesterol concentrations than girls. Further studies on cardiovascular disease risk factors in adolescence are thus required, since the appreciable differences that emerge between the sexes during adolescence may help to explain the later sex differential in cardiovascular disease. We believe that our findings in adolescents of the interactions between risk factors and activities such as smoking, use of oral contraceptives, and exercise could have major implications in prevention and health education for doctors, teachers, and the teenagers themselves.

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Value of repeated blood pressure measurements in children—the Brompton study

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Summary and conclusions

Systolic blood pressures were measured in 1797 infants aged 4 days and then repeated at 6 weeks, 6 months, 1 year, and then yearly until 4 years of age. The mean pressure rose from 76 mm Hg at 4 days to 96 mm Hg at 6 weeks but did not vary appreciably between subsequent measurements. Serial correlation coefficients of blood pressure adjusted for weight and degree of consciousness were calculated, comparing measurements at each age. At ages under 1 year the correlation coefficients were relatively weak, though most were significant ($r < 0.2$). As the children grew older these serial correlations became stronger, so that the correlation coefficient in blood pressure between ages 3 and 4 years was 0·47.

These observations suggest that "tracking" for blood pressure starts at about 1 year and is much stronger by 4 years. Taken in conjunction with the findings of other long-term follow-up studies in older children, this suggests that children develop blood pressures indicative of their adult values between 1 and 4 years.

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Introduction

Casual measurements showing raised blood pressures in adults, a potent risk factor for cardiovascular disease, are of considerable importance. Values usually continue to be raised when the blood pressure is measured subsequently. The correlation coefficient of repeated blood pressure measurements in adults is between 0·6 and 0·7.[1] Zinner *et al*,[2] comparing blood pressure measurements 4-8 years apart, have shown that such a correlation coefficient is achieved by about 18 years of age. Though we found significant correlations in blood pressure in a group of infants aged between 4 days and 6 weeks,[3] the correlation coefficient was weak ($r = 0.17$). We have therefore continued to measure blood pressure in infants initially aged 4 days to determine at what age the correlations become stronger.

TABLE I—*Numbers of children and blood pressure measurements, and causes and numbers of exclusions at ages 4 days to 4 years*

	4 Days	6 Weeks	6 Months	1 Year	2 Years	3 Years*	4 Years*
Initial No of children	1895†	1797	1777	1738	1681		
No moved from area or withdrawn	88	20	34	57	77	122	11
Deaths	10	0	5	0	2	1	1
Total remaining	1797	1777	1738	1681	1602	1050	346
Failed to contact or too uncooperative	39	32	27	47	146	120	20
Ill at time of measurement	16	104	153	188	153	108	34
No of measurements analysed	1742	1641	865‡	1341‡	1303	822	292

*Data incomplete.
†Excluding 105 born prematurely.
‡Excluding measurements with 5 cm cuff (693 at 6 months and 105 at 1 year).

Subjects and methods

POPULATION

The parents of 2000 eligible infants born consecutively at Farnborough Hospital, Kent, were approached for permission to enter their children to the study. The criteria for entry were that the parents were residents of the London borough of Bromley and did not expect to leave the area within six months. We excluded 105 infants born before 38 weeks' gestation (table I) but will report our findings on these separately. The parents of 88 infants refused to co-operate, and 10 infants born at or after 38 weeks' gestation died in the neonatal period. Thus 1797 infants, including 32 born after twin pregnancy and recruited between 1 May 1975 and 21 June 1977, were available for study. Of the 1797 infants, 1734 (96·5%) were white.

Some children subsequently moved from the area or were with-