A family study of coronary risk factors in Geelong

Prasong Tienboon and Ingrid H.E. Rutishauser
Department of Human Nutrition, Deakin University

Mark L. Wahlqvist
Department of Medicine, Monash Medical Centre

Abstract: A study of coronary risk factor relationships was carried out in 213 families with adolescents in Geelong, Victoria, Australia, in 1987. Weight, height, skinfold thicknesses, body circumferences, blood pressure and serum cholesterol were measured in both parents and children and other relevant information was obtained by questionnaire. The study group did not differ significantly from the Geelong population with respect to the proportion of Australian-born parents but contained a significantly higher proportion than expected of fathers who had a university degree or diploma. Twenty-five per cent of adolescents, 30% of mothers and 38% of fathers were positive for at least one of the three major established coronary risk factors. Significant correlations between parents and children, but not between parents, were found for total cholesterol and systolic blood pressure, while significant correlations were observed both between parents and children and between parents for measures of body size, body fatness and body fat distribution. The strongest predictors of risk factor status in adolescence were both parents with total cholesterol at or above 5.5 mmol/l, one or more parents who smoked and one or more grandparents with a history of coronary heart disease. Body mass index in adolescence was unrelated to heart disease risk factor status in adolescence or to a family history of heart disease. During early childhood and adolescence, parental risk factor status, rather than anthropometric or risk factor screening of the children themselves, is likely to provide the best guide to those at risk for an adverse coronary risk factor profile. (*Aust J Public Health* 1992; 16: 20–5)

Coronary heart disease is a leading cause of death in industrialised countries, including Australia. Because there is some evidence to suggest that cardiovascular disease begins early in life, attention is now focused on identifying risk factors in children and adolescents for the early prevention of the disease. Familial and genetic factors play a significant role in the pathogenesis of coronary heart disease. Many studies have documented a tendency for the first-degree relatives of Coronary heart disease patients to have more adverse risk factors than the general population.[1–4] Although no specific genetic mechanism has been identified for many coronary heart disease events, analysis of familial patterns of coronary heart disease and risk factors for them can often provide clues for early diagnosis and identification of symptomatic individuals at potentially high risk. Studies of parents and children provide one approach to the early identification of individuals at risk.[5] In this article we report data from a community-based study in Geelong, Victoria, on the relationships between coronary risk factors in parents and children.

Methods

Study population

The data presented are from a study of the health of Geelong adolescents designed with the aim of investigating the determinants of early risk factors for coronary heart disease. The study included a questionnaire, a physical examination and measurement of blood cholesterol in adolescents and their parents. The project was approved by the Deakin University Ethics Committee and the Victorian State Department of Education. The participants in this study were recruited with the cooperation of the principals of 24 of the 25 secondary schools in the Geelong statistical district, from all families with a child attending one of these schools in 1987 who was born between July and December 1972. This age cohort was chosen because some data from the first year of life were already available for this group from an earlier study.[6,7]

On the basis of the school records provided, 1215 families were eligible for the study and all were invited to participate. A response was received from 493 families and of these 215 agreed to participate. The 213 families who actually participated in the study thus constituted only a one-in-six self-selected sample of those eligible. However, as the main focus of the study was on relationships between coronary risk factors both over time and within families, rather than on assessment of the prevalence of risk factors in this population, a high response rate was not essential for the validity of the data and the sample size of 200 was adequate for analysis of within-family correlations.

Correspondence to Ingrid H.E. Rutishauser, Department of Human Nutrition, Deakin University, Geelong, VIC 3217.

Data collection

Data collection was carried out between July 1987 and March 1988. All measurements were made by the same physician (PT).

Anthropometric measurements

Weight was measured to the nearest 0.1 kg in a swimming costume or hospital gown using a digital platform scale (Soehnle). Height was measured to the nearest millimetre with a Harpenden anthropometer and Harpenden callipers were used to measure skinfold thickness at the left mid-triceps, subscapular, mid-biceps, suprailiac and medial thigh to the nearest 0.1 mm. All of these measurements were performed using standard techniques as described by Weiner and Lourie[8] except that the medial thigh was measured at the intersection of a line drawn from the anterior superior iliac spine to the medial condyle of the femur and the level of the gluteal fold. Waist circumference was measured at the level of umbilicus, and hip circumference at the most prominent part of the buttocks using a metal measuring tape read to the nearest millimetre.

Blood pressure

Systolic and diastolic blood pressure were measured in the sitting position after three minutes at rest with a Copal digital sphygmomanometer using standard technique and recorded to the nearest 1 mm Hg.[9,10] A second measurement was obtained after at least one minute's rest. The average value of both measurements was used for the analyses. The 95 per cent confidence interval for the difference between blood pressure measurements between two occasions one week apart was similar to that for measurements taken on one occasion and of the order of 4 mm Hg for both systolic and diastolic blood pressure.

Total cholesterol

Total cholesterol was measured on a fasting fingerprick sample of blood using the Reflotron (Boehringer Mannheim) dry chemistry system.[11–14] Using this system the coefficient of variation for total cholesterol over the range 2.33 to 12.9 mmol/l is 3 to 4 per cent (Boehringer Mannheim).

Questionnaire

All participants completed a questionnaire about smoking and sociodemographic characteristics. For the parents this also included questions on the presence or absence of a history in first-degree relatives of a number of conditions associated with coronary heart disease: overweight, high blood pressure, high blood fat, diabetes mellitus and heart attack. Repeat questionnaires completed after an interval of six months by 20 families gave 100 per cent agreement for all sociodemographic questions and 94 per cent agreement for those relating to a family history of coronary heart disease.

Data analysis

Values above the 90th centile for age of the Australian Council for Health, Physical Education and Recreation data for weight, blood pressure and cholesterol were used to categorise adolescents as risk-factor positive, as at present there are no generally accepted cut-off levels for coronary risk factors in this age group.[15] Statistical analysis was carried out using Minitab release 7.2, 1990. Pearson product-moment correlation coefficients were used to assess relationships and variables were standardised whenever data for both sexes were combined for this purpose. The appropriate Student's *t*-test formula was used to compare coronary risk factors and anthropometric measurements between groups. Confidence intervals for relative risks were calculated using the method of Katz as described by Kahn and Sempos.[16]

Results

Study subjects

Although the 213 families who participated were a self-selected rather than a random sample of Geelong families with adolescents aged 15 years, the proportion of Australian-born parents did not differ significantly ($\chi^2 = 2.8$, 2 df) from that for the population of Geelong at the 1986 Census.[17] However, in terms of educational level there was a bias, with more of the study fathers than expected having a university degree or diploma ($\chi^2 = 11.1$, 3 df). The median household size of the families was five, and in 90 per cent both natural parents were living in the household. Forty-seven fathers from the 213 families did not participate in the study at all and 30 provided only questionnaire data. Within-family comparisons were, therefore, limited to 166 families for smoking and 136 families for other data, except for total cholesterol, for which the database was complete for only 126 families. Because the significance of correlation coefficients is sensitive to sample size all correlations described, except for smoking, were restricted to these 126 families. Distribution by country of birth and educational level did not differ significantly between the fathers from the 126 families used for correlation analysis and the 213 previously described. There were also no significant differences in any of the children's measurements between these two groups.

The mean and standard deviation for age, weight, height, coronary risk factors and related anthropometric measurements in the study group are shown in Table 1. In the adolescents there were significant sex differences at the 1 per cent level in all measurements except for age and body mass index, and in the parents there were similar significant differences with sex in all measurements except for diastolic blood pressure.

Risk factor profile of the study population

The proportion of adolescents and parents with risk factor values at or above clinically relevant cut-off levels is shown in Tables 2 and 3. Table 2 shows the percentage of adolescents who smoked and who had risk factor levels above the 90th centile for a national sample of the Australian adolescent population. The percentage of adolescents who smoked was 15 per cent and the percentage with values above the 90th

Table 1: Age, weight, height and coronary risk factors in adolescents and parents

	Boys ($n = 113$)		Girls ($n = 100$)		Fathers ($n = 136$)		Mothers ($n = 193$)	
	Mean	SD[a]	Mean	SD	Mean	SD	Mean	SD
Age (years)	14.9	0.2	14.9	0.2	44.8	5.9	41.8	5.3
Weight (kg)	58.5	10.8	54.9	7.1	79.6	10.7	65.7	12.9
Height (cm)	168.5	8.8	160.9	5.5	172.9	6.8	160.1	6.3
Diastolic blood pressure (mm Hg)	61.3	10.1	66.8	8.2	75.7	10.8	74.7	9.3
Systolic blood pressure (mm Hg)	122.9	11.3	114.3	11.8	129.7	14.5	122.1	16.4
Serum cholesterol[b] (mmol/l)	4.01	0.75	4.34	0.79	5.57	1.13	5.32	0.98
Body mass index (kg/m^2)	20.5	2.8	21.2	2.6	26.6	2.9	25.6	4.7
Waist–hip ratio	0.84	0.05	0.76	0.05	0.93	0.05	0.81	0.07

Notes
(a) Standard deviation. (b) For cholesterol only, $n = 129$ for fathers and 183 for mothers.

Table 2: Percentage of Geelong adolescents who smoked or had risk factor levels above the 90th centile for age and sex of an Australian adolesecent population[15]

Risk factor	Boys ($n = 113$) %	Girls ($n = 100$) %
Major established factors		
Smoking	13.3	15.0
Diastolic blood pressure	1.7	3.0
Serum cholesterol	12.4	9.0
Other factors		
Weight for age	11.0	4.0
Systolic blood pressure	12.4	12.0

Table 3: The percentage of parents who smoked or had risk factor levels at or above accepted cut-off levels for overweight, hypertension and hypercholesterolaemia[18]

Risk factor	Fathers ($n = 136$) %	Mothers ($n = 193$) %
Major established factors		
Smoking	24.4	21.2
Diastolic blood pressure > 90 mm Hg	8.9	7.2
> 95 mm Hg	5.8	3.6
Serum cholesterol[a] > 5.5 mmol/l	51.8	39.6
> 6.5 mmol/l	15.5	10.5
Other factors		
Body mass index > 25 kg/m^2	70.5	44.1
> 30 kg/m^2	12.6	15.1
Systolic blood pressure > 140 mm Hg	19.8	13.9
> 160 mm Hg	3.6	2.0

Note: (a) $n = 129$ for fathers and 183 for mothers.

Table 4: Adolescent smoking status relative to parents' smoking status

Parents' smoking status	Adolescent's smoking status: Smoker	Nonsmoker	Total
Smoker in family	15	44	59
No smokers	7	100	107
Total	22	144	166

Note: Relative risk for adolescent smoking 3.9, 95% CI 1.7 to 9.0.

centile ranged from 2 per cent for diastolic blood pressure to 12 per cent for serum cholesterol and systolic blood pressure. Table 3 shows the percentage of parents who smoked and who had risk factor levels at or above accepted cut-off values for overweight, hypertension and hypercholesterolaemia in adults. Six per cent of fathers and 4 per cent of mothers had diastolic blood pressure at or above 95 mm Hg, and 13 per cent of fathers and 15 per cent of mothers were obese (had a body mass index of 30 or greater).

Clustering of risk factors

No adolescents were positive for all three major established coronary risk factors (elevated total cholesterol and diastolic blood pressure and smoking) and only two of the girls (2 per cent) were positive for two risk factors. In the mothers the degree of clustering of risk factors was similar to that found in the 1983 National Heart Foundation of Australia risk factor prevalence study (4 per cent with two risk factors); it was slightly higher in the fathers (7 per cent with two risk factors). About one in four of the adolescents (27 per cent of the boys, 23 per cent of the girls), 30 per cent of the mothers and 38 per cent of the fathers were positive for at least one of the three major established risk factors.

Risk factor levels in adolescents according to parental risk factor status

Data on smoking were available for both fathers and mothers in 166 families (Table 4). Adolescents who had one or more parents who smoked were themselves at significantly higher risk for smoking (relative risk 3.9; 95 per cent confidence interval (CI) 1.7 to 9.0). In contrast, if one or both parents had values for total cholesterol or blood pressure (either systolic or diastolic) at or above the lower cut-off values in Table 3, the respective relative risks for total cholesterol or blood pressure values above the 90th centile for age and sex were only 2.1 and 1.8 respectively, and not significantly different from 1. However, if both parents had total cholesterol values at or above 5.5 mmol/l (Table 5), the adolescent was at a significantly higher risk for total cholesterol above the 90th centile for age and sex (relative risk 6.2; CI 2.3 to

16.5). Obesity in one or both parents was not associated with a significantly higher risk for a body weight above the 90th centile in adolescents aged 15 years (relative risk 0.3; CI 0.1 to 2.5), but adolescents with a parent who had a body mass index over 25 had, on average, a body mass index which was one unit higher than their peers, although this difference was not statistically significant.

Tables 6 and 7 compare correlations for risk factor and related anthropometric measurements between parents, and between parents and children, to provide additional information on the relationships observed between risk factor levels in parents and children. Correlations in risk factor levels observed between parents cannot be of genetic origin, whereas correlations observed in parents and children may have both an environmental and a genetic component and might therefore be expected to be stronger than any environmental or selective associations observed in parents.

Both tables show associations between parents and children of both sexes, since no differences were found for boys and girls when considered separately. For coronary risk factors (Table 6) significant correlations ($P < 0.01$) were observed between parents and children for total cholesterol and systolic blood pressure and for waist–hip ratio between parents ($r = 0.22$; $P < 0.01$). For both total cholesterol and systolic blood pressure the correlation was stronger between children and fathers than between children and mothers. In Table 7 the strongest correlation observed was that for height between parents and children ($r = 0.55$; $P < 0.001$) and between parents ($r = 0.25$; $P < 0.01$). The only other anthropometric measurements for which the correlations were significantly higher between parents and children than between parents were body weight ($r = 0.32$; $P < 0.001$) and mid-biceps ($r = 0.29$; $P < 0.001$), although this was true only in the case of fathers. The correlation for medial thigh was similar between parents and between parents and children ($r = 0.28$ to 0.32; $P < 0.001$).

Risk factor status in adolescents according to family history of coronary heart disease

In this study adolescents aged 15 years who had one or more grandparents with a history of coronary heart disease (high blood pressure, high blood fat or a heart attack) were four times as likely to have a total cholesterol or blood pressure level above the 90th centile for age and sex (relative risk 4.0; CI 1.5 to 10.8) than those from families without such a history (Table 8). If a history of heart attack alone was considered, the relative risk of a total cholesterol or blood pressure above the 90th centile in adolescence was reduced to 1.8 (CI 1.0 to 3.5). Similarly if adolescents whose body weight was above the 90th centile for age and sex were also regarded as risk-factor positive, the relative risk was reduced to 1.2 and no longer significantly different from that of adolescents whose grandparents did not have a history of coronary heart disease. This finding suggests that in adolescence a body weight above the 90th

Table 5: Adolescent serum total cholesterol level relative to parents' cholesterol level[a]

Parents' cholesterol category	Adolescent's cholesterol category: >90th centile[b]	<90th centile[b]	Total
Both > 5.5 mmol/l	11	22	33
One > 5.5 mmol/l	5	88	93
Total	16	110	126

Notes
(a) Relative risk for elevated cholesterol 6.2, 95% CI 2.3 to 16.5.
(b) Of a national sample of Australian adolescents.[15]

Table 6: Correlations for coronary risk factors between parents and between parents and adolescents

Variables	Fathers and mothers ($n = 126$)	Fathers and children ($n = 126$)	Mothers and children ($n = 126$)
Diastolic blood pressure	0.107	0.149	0.087
Systolic blood pressure	0.002	0.238†	0.197*
Serum cholesterol	0.144	0.282‡	0.172*
Body mass index	0.109	0.213*	0.043
Waist–hip ratio	0.220†	0.082	0.141

Note: *$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$.

Table 7: Correlations for anthropometric measurements between parents and between parents and adolescents

Variables	Fathers and mothers ($n = 126$)	Fathers and children ($n = 126$)	Mothers and children ($n = 126$)
Weight	0.227†	0.319‡	0.238†
Height	0.250†	0.467‡	0.549‡
Triceps skinfold	0.042	0.103	0.007
Subscapular skinfold	0.128	0.188*	0.026
Biceps skinfold	0.071	0.291‡	0.087
Suprailiac skinfold	0.147	0.138	0.098
Thigh skinfold	0.318‡	0.298‡	0.341‡

Note: *$P < 0.05$; †$P < 0.01$; ‡$P < 0.001$.

Table 8: Adolescent risk factor status for total cholesterol and/or blood pressure relative to a history of coronary heart disease in grandparents[a]

Family history of coronary heart disease	Adolescent risk factors status: >90th centile[b]	<90th centile[b]	Total
History in one or more grandparents	28	52	80
No history	4	42	46
Total	32	94	126

Notes
(a) Relative risk for an elevated risk factor level 4.0, 95% CI 1.5 to 10.8.
(b) Of a national sample of Australian adolescents.[15]

centile for age, in the absence of elevated total cholesterol or blood pressure, is unrelated to a family history of coronary heart disease.

Discussion

The mean values for weight, height, body mass index, systolic blood pressure and diastolic blood pressure in the adolescents were similar to values from other Australian studies for this age group,[19–22] while the prevalence of smoking was somewhat lower than in other Australian studies of the same age group.[20–4] In the parents the prevalence of obesity (body mass index over 30) in both fathers and mothers (13 and 15 per cent respectively) was higher than in the same age group in a national sample of Australians (8 and 10 per cent) resident in state capital cities. The study fathers also had a higher prevalence of hypertension and hypercholesterolaemia, but not smoking, than the national sample.[18]

In this study the strongest parental influences on adolescent coronary heart disease risk factor status at age 15 were a cholesterol level at or above 5.5 mmol/l in both parents and smoking by one or both parents. Positive correlations between serum lipids in parents and their offspring are well recognised.[25,26] While cholesterol levels were significantly correlated between parents and adolescents, so that on average adolescents who had a parent with total cholesterol of 5.5 mmol/l or above had a total cholesterol level about 0.5 mmol/l higher than those who did not, there was no significant correlation between the total cholesterol values of parents despite 15 years of cohabitation.

Several studies have also observed that there is a significant resemblance between the blood pressure of parents and children even in the first weeks of life.[27–9] In the Bogalusa heart study, for example, highly statistically significant increases in systolic blood pressure and diastolic blood pressure were observed in children aged five to 17 in the presence of maternal or paternal hypertension.[5] In our study, however, a significant correlation between parents and children was found only for systolic blood pressure, with adolescents who had a parent with systolic blood pressure at or above 140 mm Hg having on average systolic blood pressure values 6 to 7 mm Hg higher than those of their peers with normotensive parents. In addition the daughters, but not sons, of parents with diastolic blood pressure at or above 90 mm Hg had diastolic blood pressure levels which were an average 6 to 9 mm Hg above those of their peers. The reason for this difference between daughters and sons is not clear, as approximately the same number of boys and girls had parents with a diastolic blood pressure at or above 90 mm Hg. As with total cholesterol, there was no significant correlation between the blood pressure of parents, although concordance of blood pressure in parents is reported to be a function of the duration of marriage[30–3] and the study parents had all shared the same family environment for at least 15 years. This observation suggests that, in comparison with genetic factors, the family environment had exerted little effect on parents' blood pressure or total cholesterol.

Overall the highest correlations between parents and children were seen for anthropometric measurements, in particular height ($r = 0.5$). It is possible that in this study the effect of day-to-day variability in blood pressure and cholesterol as compared with the stability of anthropometric measurements might have acted to reduce the within-family correlations for the former. However, blood pressure measurements repeated after one week provided no support for this possibility. Significant correlations between parents and children were also found for measures of body fatness and fat distribution such as weight and thigh skinfold but, in marked contrast to height, the magnitude of these correlations tended to be similar between parents and between parents and children and could be interpreted primarily as a consequence of a shared environment. The fact that weight, height, waist–hip ratio and thigh skinfold were all significantly correlated at a similar level in parents, however, also suggests the possibility of assortative mating for body build and body fat distribution. If the influence of assortative mating is significant, metabolic characteristics associated with a particular body build or body fat distribution will tend to be transmitted mainly by a genetic mechanism rather than through a shared family environment.

In this study body weight at 15 years of age was essentially unrelated to risk factor status except for a positive association with diastolic blood pressure in girls ($r = 0.3$). In the 126 adolescents for whom complete family data were available, only one of the 19 who had a body weight above the 90th centile for age and sex was also above the 90th centile for total cholesterol and diastolic blood pressure and one was a current smoker. A body weight above the 90th centile for age and sex was also unrelated to a history of coronary heart disease in a grandparent. However, adolescents who had one or more grandparents with a history of coronary heart disease were on average four times as likely to have a total cholesterol or blood pressure at or above the 90th centile for age and sex. This is in agreement with the findings of others that a family history of coronary heart disease is an independent risk factor for coronary heart disease.[34–7] Dennison et al., for example, found a higher risk ($P < 0.05$) of cholesterol values above the 95th centile in adolescents aged 11 to 17 years with a parental history of heart attack than in those with without such a history.[37] Although in our study the family history was based only on reports, these were found to be highly reproducible in the subsample of 20 families who repeated the questionnaire after an interval of six months.

In summary in this study both parents with total cholesterol at or above 5.5 mmol/l, a history of coronary heart disease in one or more grandparents, and one or more parents who smoked were the strongest predictors for the occurrence in adolescence of three major established risk factors for coronary heart disease. In contrast, a body weight at 15 years of age above the 90th centile for age and sex appeared to be unrelated to the presence of coronary heart disease risk factors at this age or to a family history of coronary heart disease, although there was evidence for

familial transmission of both fatness and body fat distribution. The findings of this study, with respect to transmission of familial patterns of fatness, blood pressure and total cholesterol, are in contrast to a previous Australian study in younger children which found no evidence for transmission of coronary risk factors or risk factor precursors in children aged eight years.[38] The main implications for public health, of the results both from that study and our own, are that during early childhood and adolescence parental risk factor status, rather than anthropometric or risk factor screening of the children themselves, is likely to provide a better guide to those individuals at high risk of developing risk factors for coronary heart disease in later years.

Acknowledgments

This work was in part supported by a grant from Mead Johnson, Thailand. Also gratefully acknowledged are the help and cooperation of the participating secondary schools and subjects in this study.

References

1. Deutscher S, Ostrander LD, Epstein FH. Familial factors in premature coronary heart disease: a preliminary report from the Tecumseh community health study. *Am J Epidemiol* 1970; 91: 233–7.
2. Morrison JA, Horvitz R, Khoury P, Laskarzewsa P, Gartside PS. Parental history of coronary heart disease risk factor variables in their adult children. *Prev Med* 1980; 9: 773–86.
3. Hamby RI. Hereditary aspects of coronary artery disease. *Am Heart J* 1981; 101: 639–49.
4. Glueck CJ, Fallat RW, Tsang R, Buncher CR. Hyperlipidemia in progeny of parents with myocardial infarction before age 50. *Am J Dis Child* 1974; 127: 70–5.
5. Shear CL, Webber LS, Freedman DS, Srinivasan SR, Berenson GS. The relationship between parental history of vascular disease and cardiovascular disease risk factors in children: the Bogalusa heart study. *Am J Epidemiol* 1985; 122: 762–71.
6. Rutishauser IHE, Hunter S. The Geelong study. *Proc Nutr Soc Aust* 1980; 5: 79–87.
7. Rutishauser IHE. Weight, height and weight for height in 6–7 year-old children. 1. Relationship with demographic variables. *Aust Paediatr J* 1984; 20: 35–8.
8. Weiner JS, Lourie JA. *Human biology: a guide to field methods.* Philadelphia: FA Davis Co., 1969.
9. Rogers S, Smith GD, Doyle W. Field evaluation of the Copal UA-231 automatic sphygmomanometer. *J Epidemiol Community Health* 1988; 42: 321–4.
10. Petrie JC, O'Brien ET, Littler WA, de Swiet M. Recommendations on blood pressure measurement. *Br Med J* 1986; 293: 611–5.
11. Dennis PM, Carr SG, Balazs NDH. Screening for hypercholesterolaemia in a self-selected population using the Reflotron system. *Clin Biochem Rev* 1986; 7: 99.
12. Sedor FA, Holleman CM, Schneider KA, Heyden S. Evaluation of the Reflotron as a cholesterol screening system. *Clin Chem* 1987; 33: 895.
13. James R, Tyler C, Henrikson D. An evaluation of the accuracy of the Reflotron system in the field. *Med J Aust* 1988; 149: 130–1.
14. Pain R. Are your laboratory's cholesterol results reliable enough? *Med J Aust* 1988; 149: 113–5.
15. Australian Council for Health, Physical Education and Recreation. *Australian health and fitness survey 1985: the fitness, health and physical performance of Australian school students aged 7–15 years.* 1st edn. Adelaide: ACHPER, 1987.
16. Kahn AH, Sempos CT. *Statistical methods in epidemiology.* New York: Oxford University Press, 1989: 62–3.
17. Geelong Regional Commission. *Geelong region demographic characteristics of municipalities.* Geelong: Geelong Regional Commission, 1987.
18. National Heart Foundation of Australia. Risk factor prevalence study: report no. 2, 1983. Canberra: NHF, 1985.
19. Court JM, Dunlop M, Reynolds M, Russell J, Griffiths L. Growth and development of fat in adolescent school children in Victoria. Part 1. Normal growth values and prevalence of obesity. *Aust Paediatr J* 1976; 12: 296–304.
20. Gracey M, Hitchcock NE, Wearne KL, Garcia-Webb P, Lewis R. The 1977 Busselton children's survey. *Med J Aust* 1979; 2: 265–7.
21. Simons LA, Anderson N, Simons J, Whish P. Health attitudes and knowledge, and coronary risk factors in high-school children, Sydney and Inverell. *Med J Aust* 1982; 2: 178–83.
22. Hitchcock NE, Maller RA, Gilmour AI. Body size of young Australians aged five to 16 years. *Med J Aust* 1986; 145: 368–72.
23. Hill D, Willcox S, Gardner G, Houston J. Tobacco and alcohol use among Australian secondary schoolchildren. *Med J Aust* 1987; 146: 125–30.
24. Gliksman MD, Dwyer T, Wlodarczyk J, Pierce JP. Cigarette smoking in Australian schoolchildren. *Med J Aust* 1989; 150: 81–4.
25. Schrott HG, Clarke WR, Wiebe DA, Connor WE, Lauer RM. Increased coronary mortality in relatives of hypercholesterolemic school children: the Muscatine study. *Circulation* 1979; 59: 320–6.
26. Lee J, Lauer RM, Clarke WR. Lipoproteins in the progeny of young men with coronary artery disease: children with increased risk. *Pediatrics* 1986; 78: 330–7.
27. Biron P, Mongeau JG. Familial aggregation of blood pressure and its components. *Pediatr Clin North Am* 1978; 25: 29–33.
28. Mongeau JG, Biron P, Bertrand D. Familial aggregation of blood pressure and body weight. In: New MI, Levine LS, eds. *Juvenile hypertension.* New York: Raven Press, 1977: 39–44.
29. Ibsen KK, Gronkaek M. Familial aggregation of blood pressure levels in newborn infants and their mothers. In: Giovannelli G et al., eds. *Hypertension in children and adolescents.* New York: Raven Press, 1981: 329–31.
30. Winkelstein W Jr, Kantor S, Ibrahim M et al. Familial aggregation of blood pressure. *JAMA* 1966; 195: 160–2.
31. Borhani NO, Slansky D, Gaffey W et al. Familial aggregation of blood pressure. *Am J Epidemiol* 1969; 89: 537–46.
32. Sackett DL, Anderson GD, Milner R et al. Concordance for coronary risk factors amongst spouses. *Circulation* 1975; 52: 589–95.
33. Suarez L, Criqui MH, Barrett-Connor E. Spouse concordance for systolic and diastolic blood pressure. *Am J Epidemiol* 1983; 118: 345–51.
34. Snowden EB, McNamara PM, Garrison RJ, Feinleib M, Kannel WB, Epstein FH. Predicting coronary heart disease in siblings—a multivariate assessment. *Am J Epidemiol* 1982; 115: 212–7.
35. Barrett-Connor E, Khaw KT. Family history of heart attack as an independent predictor of death due to cardiovascular disease. *Circulation* 1984; 69: 1065–9.
36. Khaw KT, Barrett-Connor E. Family history of heart attack: a modifiable risk factor? *Circulation* 1986; 74: 239–44.
37. Dennison BA, Kikuchi DA, Srinivasan SR, Webber LS, Berenson GS. Parental history of cardiovascular disease as an indication for screening for lipoprotein abnormalities in children. *J Pediatr* 1989; 115: 186–94.
38. Boulton J, Magarey A. Precursors of cardiovascular risk factors in childhood: family studies in Australia. In: Hetzel B, Berenson GS, eds. *Cardiovascular risk factors in childhood: epidemiology and prevention.* Amsterdam: Elsevier, 1987: 43–65.