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CORONARY HEART DISEASE RISK FACTORS IN NEWFOUNDLAND CHILDREN

by

© B. Christofer M. Balram, B.A., B.Sc., M.ScMed.

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ABSTRACT

A cross-sectional epidemiological study was carried out to investigate the distribution of coronary heart disease (CHD) risk factors in children aged 8-10 and 14-16 years living in two regions of Newfoundland characterised by high and low CHD mortality rates among adults. The age-adjusted CHD mortality rates for males and females in the high mortality region (HMR) were 298 and 131 per 100,000 respectively, compared to 198 and 85 per 100,000 population for males and females respectively in the low mortality region (LMR). Participants attended schools, randomly chosen from two communities in each region.

Height, weight, systolic and diastolic blood pressures were measured in Phase I. Also, an EKG was recorded and a questionnaire administered.

In the HMR, 275 (87%) children aged 8-10 and 383 (89%) aged 14-16 years responded positively. In the LMR, 255 (94%) children aged 8-10 and 259 (89%) children aged 14-16 years participated.

Ten-hour fasting blood samples were taken in Phase II. Participants in the first phase were eligible to give blood. Sera were analysed for total and HDL cholesterol, triglyceride, uric acid and glucose. In the HMR, 242 (88%) children aged 8-10 and 350 (91%) aged 14-16 years participated, compared to 240 (94%) children aged 8-10 and 201 (78%) aged 14-16 years from the LMR.

The following significant results were considered to be of epidemiological importance:

1. Boys and girls, aged 8-10 and 14-16 years, in the HMR had significantly higher mean systolic and diastolic blood pressures than those in the LMR.
2. Body mass index (weight/height^2) was significantly greater for boys and girls aged 14-16 years in the HMR than in the LMR. This risk factor was a strong discriminant between this age group in the two mortality regions.
3. The prevalence of elevated levels of systolic blood pressure was significantly higher for boys and girls aged 8-10 years and for boys aged 14-16 in the HMR.
4. Boys and girls aged 14-16 years in the HMR had a significantly higher prevalence of elevated diastolic blood pressure.
5. The total number of boys aged 14-16 years with one and clusters of two and three primary CHD risk factors simultaneously was significantly higher in the HMR.
6. The prevalence of Type IV hyperprebetalipoproteinemia was significantly higher in children living in the HMR.

The risk of developing premature CHD in adulthood appears to be higher for children in the HMR. A primary prevention CHD program should, therefore, be started in this region. This program should employ health education as a means of improving nutrition, weight control, physical activity and preventing the onset of smoking.

The effectiveness of this program, in reducing risk factor levels in a young population, should be evaluated.

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DEDICATED TO

My wife Jennella

son Christofer Anil

daughter Adele Nalini

and

Mum and Dad to whom I owe so much

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CHAPTER ONE

BACKGROUND AND OBJECTIVES OF THE STUDY

(i) Introduction

Coronary heart disease (CHD) exists in epidemic proportions in most Western industrialized countries. In Canada the extent of this epidemic is borne out by the mortality rate for the year 1977 which shows that 334/100,000 men between the ages of 45 and 64 died from this disease (Chronic Diseases in Canada, Vol. 2, No. 2, 1981).

There is evidence which suggests that CHD begins in childhood in the form of lesions that progress through several stages before becoming clinically apparent in the form of myocardial infarction, angina pectoris, sudden death and congestive heart failure in middle and late adult life. The early onset of the pathological process which may lead to CHD in adulthood has been documented by autopsy data collected by the International Atherosclerosis Project (McGill, 1968) as well as by autopsy examinations performed on U.S. casualties during the Korean War (Enos et al 1953) where 77% of young American men, average age 22 years, had gross evidence of coronary atherosclerosis.

In the Vietnamese War (McNamara et al 1971) 45% of men, average age 22 years, had evidence of coronary artery narrowing, including 5% with severe lesions. It was also shown in an anatomoepidemiological study in 5 European cities, coordinated by the WHO that 10% of boys and girls who died when they were between 10-14 years of age already had fibrous plaques in their coronary arteries (Kagan et al 1976).

Results of human epidemiological studies, such as the Albany Cardiovascular Health Centre Study, Chicago People's Gas Company Study, Chicago Western Electric Company Study, the Framingham Study and the Tecumseh Health Study, have established a multifactorial etiology for CHD and the development of the concept of risk factors. By risk factors is meant "those habits, traits and abnormalities associated with a sizeable (i.e. 100% or more) increase in susceptibility to CHD. In particular, they are associated with greater proneness to premature onset of the disease, that is before age 65" (Stamler, 1973).

The studies mentioned above identified three primary CHD risk factors. These are: (1) hypercholesterolemia, (2) cigarette smoking and (3) hypertension. One of the studies, the Framingham Study, which covered a 20-year observation period of a cohort of men and women 30-60 years of age who were living in Framingham, Massachusetts in 1948 and 1950, revealed that serum cholesterol levels of 250 mg%, systolic blood pressure $\geq$ 160 mmHg and smoking one or more packs of cigarettes daily were associated with increased risk of CHD (Truett et al 1967). It was found that each of these factors increased the risk independently, and that all three together increased the risk four-fold over any one of the factors.

In addition, this study showed that the younger the subject, the greater the likelihood that these factors would predict CHD. The Tecumseh Project (Francis and Epstein, 1965) supported the findings of the Framingham Study and, in addition, found that a

young man was more likely to have a coronary event if either of his parents had a myocardial infarction before age 65; his coronary attack was less likely to be fatal if his parents died after 65. However, after a man reached age 60, his family history appeared to have no bearing on his risk of CHD.

The identification of these risk factors led to the development of a public policy to combat the CHD epidemic in North America. The first call for an effort to prevent CHD came from the eminent cardiologist, Paul Dudley White, and others in 1959 when they issued "Statement on Atherosclerosis, Main Cause of Heart Attacks and Strokes". This was followed in 1960 by reports from the American Heart Association on cigarette smoking and cardiovascular disease. The landmark report of the Advisory Committee to the Surgeon General, "Smoking and Health", was published in 1964 (Smoking and Health, 1964). Other efforts in subsequent years not only increased the public awareness of the problem, but provided information on how to reduce the risk of premature CHD through changes in lifestyle, such as better eating habits, cessation of smoking, exercise and control of high blood pressure.

Evidence that the prevention of CHD is possible comes from two randomized control trials which were completed recently. One of these was the Primary Prevention Trial in Oslo, Norway (Hjermann et al 1981). This was a 5-year randomized control trial involving non-hypertensive men aged 40-49 years who were highly prone to the development of CHD because of hypercholesterolemia and cigarette

smoking. Intervention with the experimental group involved nutritional recommendations to lower serum cholesterol. This resulted in a 13% reduction of serum cholesterol in the experimental over the control group, over the 5-year period.

These men were advised to stop smoking. Thirty-one percent in the experimental group did, compared to 19% in the control group. At the end of the observation period, the incidence of myocardial infarction (fatal and non-fatal) and sudden death was 47% lower in the intervention than the control group.

The other primary prevention trial was the Hypertension Detection and Follow-up Program (HDFP) (Hypertension Detection and Follow-up Program Cooperative Group, 1979). This study recorded an overall reduction of more than 20% in mortality due to all causes in its stepped care group, compared to the referred care group over a 5-year period. Since this was not a placebo-controlled trial, a sizeable proportion of the referred care group was treated. This fact, together with the significant results obtained, provide immense possibilities for prevention.

It is stated often in the medical literature that prevention of CHD should begin in childhood. However, there are few studies (Lippert et al 1981; Choay and Morla, 1981; Williams et al 1981) which have reported on the population levels of total cholesterol, lipoprotein cholesterol, blood pressure, smoking habits, body mass, heart rate, triglyceride, uric acid and glucose in children. "The importance of considering all these factors together is suggested

by data from the Framingham Study where a man with three elevated primary risk factors carried greater than ten-fold risk of CHD than a man of the same age with no elevated risk factors. "The pediatric corollary would be that an obese adolescent boy, who smokes cigarettes and has a borderline elevated blood pressure, requires more intervention and medical attention than a thin girl who only has an increased cholesterol level" (Williams et al 1979).

(ii) Problem

There is in Newfoundland a regional difference in age-adjusted CHD mortality. As shown in Figure 1, the CHD mortality for men in the high CHD mortality region is 298/100,000 population compared to 198/100,000 population in the low CHD mortality region. A similar regional difference, although of a lesser magnitude, is observed for females who have CHD mortality rates of 131/100,000 versus 85/100,000 respectively (Mortality Atlas of Canada, Health and Welfare Canada, Vol. 2, 1980).

Total mortality rates are also different for males and females in the two regions. In the high CHD mortality region the rates are 930/100,000 and 540/100,000 population for males and females respectively. In the low CHD mortality region, the total mortality rates are 650/100,000 and 380/100,000 for males and females respectively.

FIGURE 1

Map of Newfoundland showing High and Low CHD Mortality Regions - HMR and LMR - with respective age-adjusted CHD mortality rates for males


LMR
198/100,000
HMR
298/100,000

(iii) General Objectives

The general objective of this study was to collect data on CHD risk factor variables from children living in the high and low adult CHD mortality regions of the Province. This information would enable us to document whether the distribution of these risk factor variables are different significantly between children in the two mortality regions. The data would also provide information on whether or not there is a significant difference in the prevalence and clustering of elevated primary risk factor variables (blood pressure, cholesterol and smoking) in these children. Furthermore, information on the various factors would be used to identify those children who may be at increased risk due to elevated cholesterol and blood pressure levels as well as smoking. It would be possible, therefore, to identify a target group of children who could benefit from a primary prevention program.

The purpose of this study, therefore, was to collect baseline epidemiological data (see below) which are considered important for the implementation of a population-based coronary heart disease primary prevention program in children.

(iv) Specific Objectives

1. To obtain descriptive data in a non-clinical setting on the following variables, from children aged 8-10 and 14-16 living and attending schools in geographical regions distinguished by high and low age-adjusted CHD mortality

rates in adults:

(i) Serum lipid profile consisting of total serum cholesterol, HDL cholesterol, LDL cholesterol, VLDL cholesterol and triglyceride

(ii) Blood pressure

- Systolic
- Diastolic (IV)

(iii) Heart Rate

(iv) Smoking history from 14-16 year olds

(v) Fasting serum glucose

(vi) Serum uric acid

(vii) Anthropometric factors

- Height
- Weight
- Body mass index, e.g. weight/height^3 and weight/height^2

2. To evaluate for each sex and age group the distribution of the factors within and between the high and low CHD mortality regions

3. To determine whether the risk factor variables are similar or different for boys and girls in the 8-10 (prepuberta1) and 14-16 (puberta1) age groups within and between the two mortality regions

4. To ascertain for each mortality region the prevalence and clustering of elevated levels of CHD risk factors, namely,

cholesterol (≥ 200 mg%), systolic blood pressure (≥ 125 mmHg for children 8-10 years and ≥ 140 mmHg for those 14-16 years), diastolic (IV) blood pressure (≥ 80 mmHg for children 8-10 years and ≥ 85 mmHg for children 14-16 years) and cigarette smoking (≥ 11 cigarettes per week for children 14-16 years)

5. To determine the prevalence of hyperlipoproteinemia in the two mortality regions and to ascertain whether the prevalence is different significantly between the regions
6. To identify, using an inferential statistical technique, namely, stepwise discriminant analysis, those CHD factors that are most important in distinguishing between similar age groups in the high and low mortality regions and between prepubertal (8-10 years) and pubertal (14-16 years) age groups in each mortality region
7. To suggest and institute measures through a primary prevention program which might help those children who are identified to be at high risk of developing premature CHD and to evaluate the effectiveness of these measures.

The following chapter will present a review of the scientific literature pertaining to CHD risk factors.

CHAPTER TWO

LITERATURE REVIEW

Introduction

Within the last 30 years or so, epidemiologists have attempted, through cross-sectional, retrospective and prospective studies in the adult population, to identify the basic etiology of coronary heart disease (CHD) which is the leading cause of death in Canada and other developed countries. The results of some of these studies (Chapman and Massey, 1964; Dawber et al 1964; Doyle, 1963; Kannel, 1970; Paul et al 1963) have found certain factors, which increase the risk of individuals developing premature CHD. Those factors identified are: (a) elevated blood lipids (total cholesterol), triglyceride and lipoprotein cholesterol), (2) cigarette smoking, (3) hypertension (high blood pressure), (4) hyperglycemia/diabetes mellitus, (5) hyperuricemia, (6) obesity, (7) inadequate physical activity and (8) elevated resting heart rate.

One of the largest and well-designed epidemiological studies, the Framingham Study, began in 1948 and observed a cohort of men and women who, at entry, were 30-62 years of age. This study showed, through biennial physical examinations and recording of illness or death, that serum cholesterol levels $\geq$ 250 mg%, systolic blood pressure $\geq$ 160 mmHg and smoking $\geq$ 1 pack of cigarettes per day were associated with increased risk of CHD. Each factor separately increased this risk, all three factors together increased the risk four-fold over any one factor alone. Today, these three factors are considered to be the principal risk factors that may lead to CHD. Evidence is now available (Drash, 1972; Friedman, 1972) and these

risk factors may have their beginnings in childhood. In addition, studies of college students (Paffenbarger et al 1966; Paffenbarger and Wing, 1967; Thomas, 1958) show that coronary and stroke mortality can be predicted at an early age.

The literature review which follows will emphasize research, depending on the information available, into the distribution of these factors in children and adolescents. The review will be presented in SECTIONS as follows:

SECTION I: SERUM LIPIDS AND SERUM LIPOPROTEIN CHOLESTEROL
SECTION II: CIGARETTE SMOKING
SECTION III: HYPERTENSION
SECTION IV: HYPERGLYCEMIA/DIABETES MELLITUS
SECTION V: HYPERURICEMIA
SECTION VI: OBESITY
SECTION VII: PHYSICAL ACTIVITY
SECTION VIII: ELEVATED HEART RATE

SECTION 1: SERUM LIPIDS AND SERUM LIPOPROTEIN CHOLESTEROL

(i) Serum Lipids

(a) Cholesterol and triglyceride levels in adults

Total serum cholesterol has been shown to be an independent risk factor for coronary heart disease in adults (Kannel et al 1971; Kannel et al 1971; Kannel, 1974). The National Cooperative Pooling Project (Pooling Project, 1978) which pooled the data from five longitudinal epidemiological studies (Albany, Chicago Gas Company,

Chicago Western Electric Company, Framingham and Tecumseh) not only confirmed this finding but showed in addition that the risk of developing CHD was increased with higher levels of total cholesterol beginning with a level of 180 mg%. Similar findings were documented by Connor (1961).

The relationship between serum triglyceride and CHD is not as clear cut as that of serum cholesterol and CHD. As a matter of fact, there is still an ongoing debate as to whether serum triglyceride is an independent risk factor for CHD. It was suggested by one study (Albrink, 1973) that fasting serum triglyceride concentrations might be of equal or even greater value than cholesterol levels as a predictor of CHD among adults. The results of two prospective studies (Albany and Framingham) contradicted this finding by showing that the predictive power of triglyceride was less than that of cholesterol (Brown et al 1965). In another study (Carlson and Bottiger, 1972) it was shown that men beyond age 50 and for all ages combined the initial serum triglyceride concentrations were of no predictive value for CHD. Heyden et al (1980) in their study showed that triglyceride levels were predictive of CHD mortality only in white females over 50 years of age.

(b) Cholesterol and triglyceride levels in children

Frerichs et al (1976) in Bogalusa, U.S.A. measured 12-14 hour fasting total serum cholesterol and serum triglyceride levels in

3,446 children between 5 and 14 years of age. Of these, 1274 were black (669 boys, 605 girls) and 2,172 were white (1142 boys, 1030 girls). Results showed that blacks had a significantly higher mean total serum cholesterol level than whites - 170 mg% versus 162 mg%. There were no consistent differences in mean cholesterol levels between the sexes of either race. Those children 10 years and younger had relatively constant levels. However, beginning about 11 or 12 years of age there was a slight reduction more noticeably in boys than in girls. Ninety-five percent of white children had cholesterol levels below 210 mg% whereas 95% of black children had values below 226 mg%.

White children had significantly higher serum triglyceride levels than black children. The mean values were 73 mg% and 61 mg% respectively. However, the levels were skewed toward higher values of the distributions for both blacks and whites. The levels of serum triglyceride in these children increased with age in both race and sex groups. The increase, however, was very minimal in blacks. Girls had higher serum triglyceride levels than boys in both racial groups, although these differences were not observed at every age interval. Disregarding sex and age differences, the triglyceride values of 90% of white children fell between 36 mg% (5th percentile) and 136 mg% (95th percentile), whereas the values for 90% of black children fell between 32 mg% (5th percentile) and 105 mg% (95th percentile).

Non-fasting serum cholesterol and triglyceride levels were measured (Lauer et al 1975) in children 6 to 18 years of age who were residents of Muscatine, Iowa in the United States. Of these children 2,346 were boys and 2,483 were girls, 96.4% of whom were white. The mean value of all children was 182 mg%. Differences between the sexes were minimal and 95% of these children had serum cholesterol levels less than 230 mg%.

There were little sex-related differences in serum triglyceride levels. In boys the mean level increased 4.2 mg% per year with age, with the youngest age group having a mean value of 72 mg%. In girls the increase was 1.9 mg% per year. Ninety-five percent of these children had values less than 183 mg%.

In both studies (Frerichs et al 1976; Lauer et al 1975) determination of serum cholesterol and serum triglyceride levels were obtained from venous blood with children in the seated position in the study by Frerichs et al (1976). The position of the children in the study by Lauer et al (1975) was not stated. It has been reported (Tan et al 1973) that serum cholesterol values obtained from blood drawn in the supine position tends to be lower in value than that obtained from blood taken in an upright or sitting position.

Serum samples obtained in the Bogalusa Study were analysed in a laboratory that was designed as "standardized" by the Center for Disease Control (CDC) in Atlanta, Georgia. Both total serum

cholesterol and triglycerides were analysed simultaneously in a Technicon Auto Analyzer II according to the protocol developed by the Lipid Research Clinics in collaboration with the CDC. In the Muscatine Study, determinations of serum cholesterol and triglyceride were done by a standard Auto-Analyzer (AA-1) technique.

In the Bogalusa Study additional aliquots of blood were taken each day in a random order on about 12% of the children in order to assess measurement errors. The coefficients of variation of the measurement errors were 5.5% and 13.3% respectively.

A study of de Groot et al (1977) done in Cincinatti, U.S.A. is perhaps more comparable to that of Frerichs et al (1976) because both studies followed the LRC-Auto-Analyzer II (Technicon) laboratory manual for blood analysis. De Groot et al (1977) obtained blood samples from 7,337 children between 6 and 17 years of age. Their analysis was based on 7,241 children - 4,946 were white (2,602 boys; 2,344 girls) and 1,829 were black (939 boys; 890 girls). Of these children 6,775 fasted for 12 hours or more. Ninety-six were excluded from the analysis - 40 of whom were American Indian and Oriental and 56 were either using oral contraceptives, anti-diabetic, anti-hypertensive, uric acid-lowering or lipid-lowering drug regimes or whose pregnancy status was unknown.

In this study, black boys and girls had a higher cholesterol level than white boys and girls. The 90th percentile value for blacks was 210 mg%, compared to 190 mg% for whites. The mean

values of plasma cholesterol were slightly lower than the mean values reported by Frerichs et al (1976) for white boys and girls in most comparable single age groups. Mean serum triglycerides obtained by Frerichs et al (1976) were higher in all comparable single age groups and sex as compared with plasma triglyceride values reported by de Groot et al (1977). In this study by de Groot et al (1977), it was stated that, although serum cholesterol levels are highly correlated with plasma cholesterol levels, plasma cholesterol values are generally 3-5 mg% lower than serum cholesterol levels.

Six weeks later a 15% random sample of the initial population studies by de Groot et al in 1977 were re-examined. Apart from remeasuring plasma cholesterol and triglyceride, they also measured HDL cholesterol and LDL cholesterol. The methods used to collect and analyse blood samples for cholesterol and triglyceride in this study (Morrison et al 1978) were similar to those used by de Groot et al (1977). Analyses were done on 927 students of whom 243 were black (119 males; 124 females) and 684 were white (358 males; 326 females) between 6 and 17 years of age. They found that between the ages of 6 and 11 there were no significant changes in lipid levels in any of the sex and race groups. These investigators did find that lipid values were different during ages 12 to 17.

In white males grouped according to two year age intervals 10-11, 12-13, 14-15 and 16-17, there was an increase in mean triglyceride in each age group. In black males mean triglycerides

increased between each age group. Results showed mean triglyceride levels were higher in 12-17 year olds than in 6-11 year olds. These differences were significant. Statistically significant differences were also observed between 12-17 and 6-11 year age groups for mean cholesterol levels, the latter age group having higher levels. From ages 10-11 and 16-17, the tendency was for decreasing levels of mean total cholesterol.

Black females showed no significant differences between the two age groups, 6-11 and 12-17 for triglyceride, and total plasma cholesterol levels. White females in the 12-16 year age group had lower total plasma cholesterol that were significantly different from the 6-11 year age group. Mean triglyceride levels were not significantly different between the two age groups for this sex.

White females in the 6-11 age group had higher mean triglyceride than their male counterparts. These differences were statistically significant. Lipid differences were minimal between males and females in the 12-17 year age group. Black females had significantly higher mean triglyceride levels in the 6-11 year age group than black males. No significant differences were observed between black males and females for lipid levels in the 12-16 year age groups.

In 1961 Hames and Greenberg studied residents of Evans County, Georgia, U.S.A. between 6-10 years of age. Serum cholesterol was measured in 1,321 persons. Of these, 837 were white (444

boys, 393 girls) and 484 were black (229 boys, 255 girls). All participants had fasted; however, the length of fast nor the position in which blood was taken was not stated. Measurement of cholesterol was by the method of Zlatkis, Sak and Boyle. In this study, serum cholesterol levels increased with age for each race and sex group. Non-whites had a greater increase per year than whites (3.3 mg% versus 4.3 mg%) versus (2.7 mg% versus 2.8 mg%). Females showed a tendency to increase faster than males, but this was not significant in this group of subjects.

Hodges et al (1965) reported mean cholesterol levels of 167 mg% in Iowa teenagers and noted that 20% of the children had cholesterol levels above an arbitrary normal limit of 200 mg%. Clarke et al (1970) reported that 13% of adolescent children in Vermont had cholesterol levels above 200 mg%. Starr (1971) noted that 6-7% of California school children, aged 6-14 years, had cholesterol concentrations above 200 mg%. McGandy (1971) reported that mean plasma cholesterol levels in Eastern United States school children were in the 180-190 mg% range and concluded that dietary and environmental forces strongly associated with coronary heart disease in middle life begins to operate during adolescence. McGandy (1971) stated also that if coronary heart disease prevention was ever to achieve its maximal effect, attention must be turned to much younger groups.

Pediatric hypercholesterolemia has also been found to be common in unselected middle-class children in Australia and South

Africa. Godfrey et al (1972) reported that 2.5% of Australian school children had plasma cholesterol levels over 238 mg%. Duplessis et al (1967) noted that 8.9% of white South African chidren aged 7-11 years, and 13.9% of those aged 12-15 years, had cholesterol levels greater than 260 mg%. Serum cholesterol greater than 200 mg% and triglycerides greater than 100 mg% were found in 20% and 8% of children respectively. Thirteen percent of the children were obese (greater than 25% body fat).

There are also marked differences in lipid levels of children belonging to different cultures and geographic locations. The mean plasma cholesterol level of rural Mexican children between 5 and 14 years of age was 100 mg%, compared to a mean of 187 mg% in children living in Wisconsin, U.S.A. of comparable age (Golubjatnikov et al 1972). Thus, Wisconsin children have nearly twice the levels of plasma cholesterol of the Mexican children. In addition, what is most striking is that the upper 10% distribution of the plasma cholesterol levels of Mexican children practically can be superimposed on the lower 10% for plasma cholesterol levels of Wisconsin children.

Another study (Connor, 1979) compared cholesterol levels of children living in different geographic locations. This comparison was between the Tarahumara Indian and Iowa children. There was a substantial difference in the mean plasma cholesterol levels between the two groups. In the Tarahumara's, the plasma cholesterol level was 118 mg%, compared to 163 mg% in the children from Iowa.

It was suggested by the authors that the lower mean plasma cholesterol levels in the Indian children probably reflect their dietary habits. Their diet is largely vegetarian with a very active agricultural society. In this society, adult lipid levels are only, on the average, 10-20 mg% higher than those of children.

A study (Kafatos et al 1979) done in another part of the world, on the Greek island of Crete, measured cholesterol levels in youth. This study showed that the mean total cholesterol levels of boys aged 0-4, 5-9, 10-14 and 15-19 were 165 mg%, 168 mg%, 160 mg%, 165 mg% respectively. Urban children had slightly higher mean levels than rural children.

A comparison of mean plasma cholesterol levels of children from societies with a high incidence of CHD versus those from societies with a low incidence of CHD shows the following for sexes combined: 5-9 years of age, 170 mg% versus 135 mg%; 10-14 years of age, 180 mg% versus 140 mg%; and 15-19 years of age, 175 mg% versus 125 mg% (American Health Foundation, 1979). There is evidence to suggest that intervention to lower cholesterol levels among children with high levels can be accomplished. Stein et al (1975) reported that they were able to reduce the cholesterol level of 229 male adolescents attending boarding schools in Johannesburg, South Africa. This was done by providing polyunsaturated fat substitutes in place of saturated fat in the regular diet.

Summary

Elevated serum cholesterol is an independent risk factor for coronary heart disease among adults. Epidemiological studies involving children have shown that there is a high prevalence of elevated cholesterol levels ≥ 200 mg% in societies with high CHD mortality rates. These findings suggest the need to identify at a young age those children who have high cholesterol levels in order that measures may be taken to reduce their elevated cholesterol before it adds to the development of atherosclerosis and subsequent coronary heart diease.

(ii) Serum lipoprotein cholesterol - HDL, LDL and VLDL cholesterol

There is an inverse association between HDL cholesterol and CHD (Castelli et al 1977; Tyroler et al 1975; Glueck et al 1976). This cholesterol fraction is now regarded as having a protective effect against CHD. It has been shown also (Kannel et al 1971; Castelli et al 1977; Tyroler et al 1975) that LDL cholesterol is associated with CHD. Although there is a positive association between VLDL cholesterol and CHD, it's importance as an independent risk factor is not established firmly (Kannel et al 1971; Castelli et al 1977).

The associations referred to above were documented through population studies on adults. There are few studies that have collected data on lipoprotein cholesterol levels in children selected randomly from communities.

Jones et al (1980) found that the mean HDL cholesterol of males and females in the 5-9 year age group were 60 and 57 mg% respectively. In the 10-14 year age group, the mean values were 58 and 55 mg% for boys and girls respectively. Females in the 15-19 year age group had higher mean HDL levels than males - 57 versus 46 mg%.

The mean LDL cholesterol levels were 87 and 97 mg% for males and females respectively in the 5-9 year age group. In the 10-14 and 15-19 year age groups, males had mean LDL cholesterol values of 98 and 96 mg%, whereas the mean values for females were 93 and 91 mg%.

Females in the 5-9 and 10-14 year age groups had identical mean VLDL cholesterol levels - 10 mg%. Males in these age groups had mean VLDL-C corresponding to 7 and 8 mg% respectively. In the 15-19 year age group, the mean VLDL cholesterol levels were 11 and 10 mg% for males and females respectively. The children who participated in this study were from the Toronto-Hamilton area and had fasted 12 hours before giving blood.

Using similar age groupings as in the previous study, Williams et al (1980) reported mean HDL cholesterol values of 56 and 53 mg% for males and females respectively in the 5-9 year age group. Males in the 10-14 and 15-19 year age groups had mean HDL cholesterol values of 55 and 46 mg%. Females in these two age groups had identical mean values - 52 mg%.

The mean LDL cholesterol value of females in the 5-9 year age group was 7 mg% higher than males. Males and females in the 10-14 year age group had similar mean LDL cholesterol values - 97 mg%. In

the 15-19 year age group, the mean LDL cholesterol was 2 mg% higher for females than males whose mean value was 94 mg%.

The Bogalusa Heart Study measured lipoprotein cholesterol levels of children 2-14 years of age. The mean HDL, LDL and VLDL cholesterol levels were 67, 87 and 6 mg% respectively.

Differences in mean lipoprotein cholesterol levels were reported (Connor et al 1978) for children of different cultural and geographical backgrounds. A comparison of HDL, LDL and VLDL cholesterol between Tarahumara and Iowa children showed the following mean levels: 23 versus 50 mg%; 75 versus 103 mg% and 20 versus 8 mg% respectively. The LDL/HDL ratio was lower in the Tarahumaras than the Iowans. It should be pointed out that of the various lipid ratios such as total cholesterol/HDL, total cholesterol/LDL, LDL/HDL and HDL/$\sqrt{\text{LDL}}$, the latter was shown (Green et al 1982) to be the best predictor of CHD.

Summary

There is a paucity of data on lipoprotein cholesterol distribution in children. This information is important in order to identify those children who might be at risk for CHD and to be able to plan a strategy for primary prevention.

SECTION II: CIGARETTE SMOKING

During the 1950's and 60's several large prospective epidemiological studies assessed the mortality risks associated with

cigarette smoking. In the United States, Hammond and Garfinkel (1969) followed 358,500 males and 445,900 females for 6 years, Hammond and Horn (1958) followed 187,800 men for 3½ years, Weir and Dunn (1970) followed 68,200 men for up to 8 years and Paffenbarger and Wing (1969) followed 50,000 men for more than 17 years. In Canada, Best (1966) followed 78,000 men for 6 years and in Britain, Doll and Hill (1964) and Doll and Peto (1976) studied about 34,400 male doctors for 20 years.

These studies showed that death rates from coronary heart disease were higher for smokers than non-smokers or ex-smokers, and that these mortality rates relative to those for non-smokers varied with age and amount smoked (Townsend and Meade, 1979). It has been shown that individuals who smoked 1 pack of cigarettes per day had a 3 times greater risk of developing coronary heart disease than non-smokers (Smoking and Health, 1964; Pinney, 1979; Kannel et al 1966; Kannel et al 1975).

According to the Framingham Study, the number of cigarettes smoked daily and not the duration of the habit increases the risk of CHD. Cigarette smoking is not only independent of other major risk factors but is known to enhance the effect of other coronary risk factors, such as hypertension and hypercholesterolemia, and at any given level of risk, smoking appears to double that existing risk (Borhani, 1977).

Recent evidence linking cigarette smoking to pulmonary and cardiovascular disease has resulted in a slight decrease in adult

smoking, but smoking among children and adolescents appears to be increasing at alarming rates with over 3,000 new adolescents in the United States smoking daily (United States Department of Health, Education and Welfare, 1976; Ramstrom, 1977; American Cancer Society, 1977). In Canada, 19.3% males and 25.5% females between 14-16 years of age reported they smoked daily (Smoking Habits of Canadian School Children, 1980).

A major proportion of the increase in adolescent smoking during the past decade is attributable to a rapid rise in the number of adolescent females acquiring the habit (Lippert et al 1981; Choay and Morla, 1981; Williams et al 1981). Rosenberg et al (1980) investigated the relationship between myocardial infarction (MI) and cigarette smoking in women 30-49 years of age. They found that the relative risk of an MI increased with the amount of cigarettes smoked compared to women who had never smoked. The estimate was 1.8 for smokers of less than 15 cigarettes per day and 6.9 for smokers of 35 or more cigarettes per day. These researchers concluded that the risks faced by those who smoke heavily are appreciable.

The increasing trend in smoking during childhood is of particular concern knowing that the incidence of hypertension and CHD is highest and onset earliest in those who began smoking when they were 20 years of age (Borhani, 1977). When one considers that this is a coronary risk factor which, if avoided or abandoned, will significantly reduce the risk of CHD, one appreciates the importance of preventing the onset of smoking among the young (Salonen, 1980;

Kahn, 1966; Gordon et al 1974).

Summary

The positive relationship between cigarette smoking and CHD is well documented. Furthermore, it is shown that the habit begins in the early adolescent years and at the present time girls are smoking more than boys. In order to affect a program aimed at prevention of CHD, it would be necessary to identify those persons who are at increased risk because of their smoking habits and institute measures to prevent the onset of smoking and to assist those who are smoking to quit the habit.

SECTION III: HYPERTENSION

Introduction

Epidemiological research has shown that essential hypertension, that is, high blood pressure without evident cause, can lead to major complications, such as stroke (Kannel, 1971) and coronary heart disease (Dawber and Kannel, 1961). In one study (Kannel et al 1972) congestive heart failure was shown to be six times more prevalent among hypertensives than normotensives. The extent to which hypertension can be considered a public health problem is shown by the fact that the prevalence of hypertension among adult Americans is 15-20% (Dustan et al 1973). The prevalence of hypertension in the Canadian adult population is estimated to be between 9% and 14% (Fodor, 1982). Several factors have been shown to be associated

with the development of hypertension. Some of the most important factors are discussed below.

(i) Risk factors for hypertension

(a) Age and sex

Studies of adult populations have shown that there is a positive relationship between blood pressure and age (Comstock, 1957; Miall and Lovell, 1967; Pickering, 1968). Investigators (Londe, 1968; Biron et al 1976) have documented a similar finding for children.

Among adults, men have higher pressures than women until age 50 (Kannel and Dawber, 1973). In children, the general tendency is for mean blood pressure levels to show no significant difference between the sexes until about 14 when boys begin to show higher systolic blood pressure levels than girls. Mean diastolic blood pressures are not significantly different between the sexes until age 15 after which boys show a tendency towards higher levels. In women, the incidence of essential hypertension increases after menopause and remains high thereafter (Kannel and Dawber, 1973; Miall and Lovell, 1967; Harris, 1968).

(b) Familial factors

There is considerable evidence from studies done on adults to indicate that there is a strong familial relationship in the development of essential hypertension (Reynolds, 1973; Thomas, 1959; Chazan and Winkelstein, 1964; Gearing et al 1962; Allan, 1933;

Johnson et al 1965). A family history of hypertension and of its secondary and often lethal complications is an important marker for identifying individuals at risk (Blumenthal and Jesse, 1973; Thomas, 1959). Children at increased risk for developing hypertension can be identified by finding out whether they belong to a family with members who have had a history of hypertension, myocardial infarction, cerebrovascular accident or uremia of unknown etiology before they were 50 years of age (Blumenthal, 1973; Blumenthal and Jesse, 1973). Another investigator, Lieberman (1974) pointed out that it is necessary to conduct a thorough family history looking for the presence of hypertension, stroke or CHD in younger family members. If any of these factors are present, these children are at greatest risk for developing hypertension.

Zinner et al (1971) have shown that there is a familial influence of blood pressure in childhood. Their data suggest that familial tendencies for hypertension are established early in life. This study involved 721 children, 2-14 years old from 192 families in Boston, U.S.A. In the study by Londe (1977) of the 132 hypertensive children detected between ages 3-16, 51% had one or both parents with hypertension, whereas only 18% of normotensive children had a hypertensive parent. Other studies (Deutscher et al 1966; Higgins et al 1977) have shown similar associations.

(c) Genetic factors

Platt (1947) felt that hereditary factors are the primary cause of essential hypertension and that it relates to a single autosomal gene. On the other hand, Pickering (1968) felt that blood pressure is influenced by a constellation of genetic factors which are modified by environmental influences.

Biron et al (1975) in a well-designed study in Montreal, Quebec, Canada, showed that correlation of blood pressure levels between natural children and their natural parents were highly significant for both systolic and diastolic pressures. There was no parent-child correlation for adopted children up to the age of 6. A small non-significant parent-child correlation was seen when adoption had occurred 7 or more years earlier. A total of 274 families participated and included 379 adopted and 129 natural children. In a subsequent re-analysis of their data (Annest et al 1979) to determine genetic and environmental factors in determining blood pressure levels, they found that both correlated each to a greater degree with diastolic, than with systolic pressures.

(d) Obesity

A relationship between obesity and hypertension has long been recognized (Pickering, 1955; Hahn, 1952; Kannel and Dawber, 1973; Shah, 1967). In a comprehensive study relating adiposity and blood pressure, Kannel et al (1967) reported that there was a higher prevalence of hypertension in the obese patient, but that obesity

was not necessarily the chief determinant factor in those patients who were hypertensive. They also showed that while existing hypertension was related to relative weight, obese patients who were normotensive initially, subsequently developed hypertensive cardiovascular disease at an increased rate.

In Londe's study (1971) which reported essential hypertension in apparently normal children, it was found that of 132 children with elevated blood pressure at ages 3-16 over a period of 3-9 years, there were 55% who were overweight, compared to only 15% of normotensive children who were overweight. Heyden et al (1969) did a 7-year follow-up of 30 hypertensive adolescents and were able to show that overweight or subsequent weight gain was associated with the development of hypertension. Paffenbarger et al (1968) have shown also a relationship between weight and subsequent development of hypertension.

(e) Salt

This is another factor which has been implicated as being associated in the development of essential hypertension. Dahl et al (1954, 1957, 1972, 1968) have published several studies which suggest that the level of dietary salt intake may play a role in the development of essential hypertension. Dahl and Love (1954) postulated that excessive salt intake in infants may predispose them to essential hypertension as adults and, thus, have suggested that salt additives to processed baby foods be discontinued.

Studies of Eskimos who have a low salt intake and a low incidence of hypertension (Knudsen and Dahl, 1966) in comparison with Northern Japanese who have an average consumption of 26 g of salt daily and a 40% incidence of hypertension in individuals over 40 years of age provide some support for Dahl's hypothesis. There is still some doubt as to whether salt induces hypertension in populations at large. Miall (1959) and Swaye et al (1972) could not correlate the levels of systolic and diastolic pressure with the amount of salt ingested in random samples of some populations. Guthrie (1968) suggested that infant foods be manufactured which are markedly lower in salt than those currently available.

The study by Prior et al (1968) of two Polynesian populations with different salt intake supported Dahl's hypothesis that increased salt intake and higher blood pressures are related. A majority of people, however, ingest a large amount of salt without developing hypertension and many hypertensive patients remain hypertensive despite severe salt restriction. Denton (1965) and Blair-West et al (1970) concluded that the appetite for salt is acquired and not instinctive. If this is the case, it must be culturally determined, must develop early in life and must be subject to modification.

In a recent article by the Committee on Nutrition, American Academy of Pediatrics (1974) reviewing current thinking about salt intake and blood pressure in children, the following statements were made: "(1) The Committee recommends actions that reduce or

avoid increasing the present level of salt intake by children in the population at large; (2) Children with a family history of hypertension may benefit from a low salt diet, although the evidence is incomplete; (3) There is a reasonable possibility that a low salt intake begun early in life may protect, to some extent, persons at risk from developing hypertension". Thus, there is suggestive, but not conclusive, evidence that salt intake may play a role in the genesis of essential hypertension. It is more than likely that excess intake of dietary salt may be one of the main contributing factors in those children who may be genetically predisposed to hypertension.

(f) Race

Rose (1965) recorded blood pressures of 277 black children in the 10th grade and compared his results with published data on white children. He found that the mean pressures of the black children were low by comparison with those of the white children. In a more comprehensive and meaningful study, Comstock (1957) showed little difference in blood pressures between blacks and whites up to the age of 15, but thereafter at all ages blacks had higher mean blood pressures than whites. These studies would suggest that black and white children have comparable blood pressures, but at some time in young adulthood, the differences become significant.

(g) Other factors

Several studies have shown (Moriyama et al 1971; Stamler et al 1967; Hypertension Detection and Follow-Up Program Cooperative Group (HDFP), 1977) that there is a relationship between hypertension and socioeconomic factors. In the HDFP study, the prevalence of hypertension was inversely related to educational level. This relationship was stronger in the younger age group. The results of the HANES survey showed that when blood pressure was correlated with family income levels, the values for the children showed a trend towards higher blood pressure levels in lower economic groups.

Paffenbarger et al (1968) who did a follow-up study of men 22-31 years after college, found a correlation between pulse rate and blood pressure. Miller and Shekelle (1976) found a sizeable correlation (> 0.3) between pulse rate and systolic blood pressure among grade 10 students. The association between heart rate and blood pressure has been documented also by Stamler (1975).

Although Paffenbarger (1968) has presented data which suggests a correlation between lack of exercise in youth and risk of subsequently developing hypertension, his analysis did not control for weight. In a study (Montoye et al 1972) which involved 1,700 males 16 years of age and older, it was found that systolic and diastolic pressures were lower in active individuals.

(ii) Natural history of hypertension in children

The natural history of hypertension in children is documented

poorly. Some investigators feel that elevated blood pressures in a child do not necessarily predict a future potential for hypertension (Stewart, 1971; Julius and Schork, 1971). Others (Perera, 1950; Paul, 1971) have documented somewhat conflicting evidence regarding the prognosis of elevated blood pressure in children and young adults. Most of these studies involved young adults, however, rather than children.

Perera (1950) followed 30 young adults with essential hypertension for an average of 20 years. Eight patients (37%) died after a mean survival period of 21 years after diagnosis. Of the remaining 22 patients, 15 had hypertension complications and this figure, combined with the 8 deaths, would suggest that elevated blood pressures at an early age is a poor prognosis. Diehl and Hesdorffer (1933) reported on the follow-up of men who were found to be hypertensive in college and found that those with elevated systolic blood pressures initially were more likely to have elevated systolic blood pressures than those who were normal initially.

Julius et al (1964) followed 208 male college students prospectively. After an average 20-year interval, they found that: (1) the overall incidence of hypertension initially was the same - 13% - as it was after a 20-year follow-up (14%); (2) there was poor correlation between initial and final readings for an individual; (3) there appeared to be a relationship between obesity and hypertension. In perhaps the most meaningful study pertaining to children, Heyden et al (1969) studied a group of 435 adolescents

looking for evidence of elevated blood pressures. Fifty adolescents (11%) were found to have hypertension: systolic levels > 140 mmHg and/or diastolic levels > 90 mmHg. Thirty people were re-evaluated 7 years later; 12 (40%) had lower blood pressure, 7 (23%) had pressures which were unchanged and the remaining 11 (37%) had either systolic (> 160 mmHg and/or > 95' mmHg) hypertension or vascular complications (including death in two patients). This study would suggest that hypertension discovered at a young age can be indicative of future hypertensive disease.

The evidence is conflicting, but it would appear that although a significant percentage of young people with a single elevated blood pressure reading will not develop hypertension as adults, an unusually higher percentage of those with elevated blood pressure will develop frank hypertension and the complications associated with this entity.

(iii) Prevalence of essential hypertension in children

Perhaps the study that had the most impact in the documentation of the prevalence of essential hypertension in children was that done by Londe et al (1971). The children were 4-15 years of age of whom 735 were boys and 738 were girls. Blood pressures were taken in the supine position from the right arm and the diastolic blood pressure corresponded to the disappearance of the sound and, if this were not possible, then the muffling of the sound was used. The criteria used for defining hypertension in this study was blood

pressure levels greater than the 95th percentile. Using this criteria, Londe documented a prevalence of hypertension that was 2.3%.

Since then, Kilcoyne et al (1974) studied 3,537 high school students in New York between the ages of 14 and 19 and documented a prevalence of 5.4% systolic hypertension and 7.8% diastolic hypertension using a criteria of systolic blood pressures $\geq$ 140 mmHg and/or diastolic blood pressures $\geq$ 90 mmHg for the 5th phase. Blood pressures in this study were made by one examiner and they were taken in the sitting position from the left arm.

Silverberg et al (1975) in a study of 15,594 high school students between the ages of 15 and 20 in Edmonton, Alberta, Canada, documented a $\geq$ 0.2% prevalence of essential hypertension. Their definition of hypertension was systolic blood pressures $\geq$ 150 mmHg and/or diastolic blood pressures $\geq$ 95 mmHg. In this study, more than one examiner measured blood pressure from the sitting position on the right arm. Diastolic blood pressure corresponded to the 5th phase. However, if no disappearance was detected, the point of muffling was used.

Reichman et al (1975) took blood pressure measurements from 1,863 students in New York, U.S.A. These students were between the ages of 12-20 of whom 90% were between the ages of 14-17. This investigator documented a 5.9% prevalence of systolic hypertension and 2.5% prevalence of diastolic hypertension using the criteria of systolic blood pressure $\geq$ 140 mmHg and/or diastolic blood pressure

≥ 90 mmHg. In this study, blood pressures were measured from the left arm by more than one individual, with children in the sitting position and the diastolic blood pressure corresponding to the 5th phase.

In 1975 Lauer et al measured blood pressure levels in 4,829 school children in Muscatine, Iowa, U.S.A. These children were between 6 and 18 years of age. They documented a prevalence of 8.9% systolic hypertension and 12.2% diastolic hypertension with a combined prevalence of 4.4% for systolic and diastolic hypertension. The criteria for defining hypertension in these children was systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg. Blood pressures were taken by more than one examiner in this study, with children in the sitting position and measurements made from both arms and the average of two readings used. The diastolic blood pressure corresponded to the 4th phase.

In another study (Cassimos et al 1977), blood pressure measurements were made from 4,428 school-children who were of Greek extraction. These children were between the ages of 7 and 18 years of age. The reported prevalence of essential hypertension in those children between 14 and 18 years of age was 3.1%. The criteria used for defining hypertension in this study was blood pressure levels greater than the 95th percentile. In this study blood pressures were taken by one examiner, however, the position of the child when the blood pressures were measured was not stated. Blood pressures were taken from the right arm and the diastolic blood pressure was

recorded at the disappearance of the sound.

The prevalence of essential hypertension reported in the studies mentioned above ranged from 2.2% to 4.4%. The prevalence of diastolic hypertension ranged from 2.4% to 12.2%. However, there are several points worth mentioning with relation to the reported prevalence in this age group.

One of these is that different criteria was used for establishing the presence of hypertension. Some researchers (Lauer et al 1975; Reichman et al 1975; Kilcoyne et al 1974) used a reading of $\geq$ 140 mmHg of systolic and/or $\geq$ 90 mmHg of diastolic, while others (Cassimos et al 1977; Londe et al 1971) used readings greater than the 95th percentile to indicate essential hypertension. In two studies (Cassimos et al 1977; Londe et al 1971) pressures were also taken by more than one examiner. Blood pressure measurements were also taken in different positions. The sitting position was the most common (Lauer et al 1975; Reichman et al 1975; Silverberg et al 1975). The supine position was used in one study (Londe et al 1971). In other studies (Cassimos et al 1977; Reichman et al 1975; Kilcoyne et al 1974) the point of disappearance of the 5th phase diastolic was taken as the diastolic blood pressure. Blood pressures were recorded from the right arm in some studies (Cassimos et al 1977; Silverberg et al 1975; Londe et al 1971). In one study, it was recorded from the left arm (Reichman et al 1975) and in another study (Lauer et al 1975) blood pressures were recorded from both arms and the mean of the two readings used. The

cuff sizes used to measure blood pressures were not stated.

It should be mentioned that there are certain inherent errors in measuring blood pressure with mercury manometers. For example, the tendency for preferences of certain terminal digits usually 0 or 5, the reading back to the point of visual oscillations from that at which the auscultatory endpoints occur with overcompensation, the possibility of pressure values being forgotten between the moment of reading and tabulation, the examiner's behaviour and the circumstances surrounding the measurement, and the possible auditory problems of the examiner.

It is also acknowledged widely that a great deal of importance should be placed on using an appropriate cuff size whenever a mercury manometer is used for taking blood pressure measurements. This fact has been pointed out by Woodbury et al (1938) and Robinow et al (1939) who concluded, after measuring blood pressure in infants and older children using the indirect and direct methods, that the smaller the arm the narrower should be the cuff. In yet another study by Karvonen et al (1964), it was found that too short a cuff bladder like too narrow a cuff can produce artificially high readings. King (1967) also found that too short a blood pressure bladder produced artificially higher readings, but concluded that once the cuff had encircled the arm, the effect of the cuff length on the reading was minimal. He stressed the importance of these findings for pediatric studies. Long, Dunlop and Holland (1971) found that factors such as upper arm length, arm circumference and

skin fold thickness did not have very much influence on the level of blood pressure recorded in children.

However, they found that in the same child, when cuffs of differing sizes were used, smaller cuffs gave consistently higher readings. They pointed out that it was possible to minimize the errors in cuff selection by using the largest cuff which will snugly fit the subject's arm. In addition, the inflatable bladder contained within the cuff should completely encircle the arm without overlapping. Additional difficulties in the measurement of blood pressure with mercury manometer leads to the inflation of an improperly applied cuff which may result in systematically higher readings (Rose and Blackburn, 1968). Variations in the speed of cuff inflation and deflation may result in differences in blood pressure recorded (Holland and Humerfelt, 1964).

Summary

Population studies of blood pressure levels do show that essential hypertension in children is more common than was once believed. In addition, epidemiological studies are able to identify those children who may be at increased risk of developing hypertension later in life because of blood pressure levels that are higher than those considered to be acceptable for their age and sex.

Primary prevention of essential hypertension is very practicable, since factors such as excess salt and overweight which are positively associated with its development can be manipulated

without causing discomfort to the individual. It is, therefore, of public health importance that those children who may be at increased risk of developing hypertension as adults be identified. This will facilitate the implementation of appropriate preventive strategies to reduce or eliminate this risk.

SECTION IV: HYPERGLYCEMIA/DIABETES MELLITUS

Clinical and pathological studies (Stamler and Epstein, 1972; Miller and Miller, 1975; Abenavoli et al 1981; Strong et al 1973; Gordon et al 1977) have shown positive associations between diabetes and coronary heart disease. Other studies (Rhoads et al 1976; Stamler and Epstein, 1972) have indicated that persons with atherosclerotic disease have a greater frequency of abnormal glucose tolerance than controls. There is also evidence (Gordon et al 1977) which suggests that there is a significant relationship between diabetes and hypertension, serum lipids and obesity.

Roberts and Crall (1977) recently studied coronary artery disease in autopsy cases of nine juvenile diabetic patients and compared their findings with a similar non-diabetic group. They found more extramural luminal narrowing by atherosclerotic plaques in the diabetic group.

Studies of incidence and prevalence of diabetes in children are few. Those studies which have been done lack uniformity as to epidemiological methodology. In a retrospective study done in

Sweden (Sterky et al 1978) it was found that the mean yearly incidence of diabetes in children 0-14 years old was 19.6/100,000. In another Swedish study (Dahlquist et al 1982), the incidence was 23/100,000 for children 0-14 years old.

In Denmark the incidence of diabetes in children (Christau et al 1979; Christau et al 1981) was reported to be 13.7/100,000 and in northern Norway (Bratlid, 1976; Joner and Sovik, 1981), 6.8/100,000. The reported incidence rate in Finland for the age group 0-19 years was 29/100,000 (Christau et al 1981; Koivisto et al 1976).

Summary

Research findings indicate positive association between diabetes and CHD as well as hypertension, serum lipids and obesity. Available data also show a geographical variation in the prevalence and incidence of diabetes in children. The distribution of this variable in a healthy population of children is not well documented.

SECTION V: HYPERURICEMIA

It is still not clear whether elevated uric acid is associated with an increased prevalence or incidence of coronary heart disease. As early as 1951 it was reported (Gertler et al 1951) that young men with CHD had higher mean serum uric acid levels than healthy controls. This finding was supported by other case control studies (Kohn and Prozan, 1959; Benedik, 1967; Jacobs, 1972; Shoshkes, 1976). However, the number of persons participating in these studies was

small.

Larger epidemiological studies have produced conflicting results. One such study, the Israeli Prevalence Study (Medalie, 1973) found no association between uric acid level and CHD. However, an Australian prevalence study (Welborn et al 1969) did show an association between uric acid level and CHD in men but not in women. This association persisted when other variables were controlled, but diuretic usage was not mentioned. Incidence data from Honolulu (Kagan et al 1975) showed a significant association between baseline uric acid level and CHD after two years of follow-up. This study, however, failed to control for use of diuretics. It has been shown in other studies that the association between uric acid level and CHD disappears when the use of diuretics is controlled for (Shurtleff, 1970; Gordon et al 1971; Myers et al 1968; Klein et al 1973; Isomaki, 1969; Bengtsson and Tibblin, 1974; The Coronary Drug Project Research Group, 1976; Yano et al 1977).

In the Framingham Study, except for an increased incidence of myocardial infarction in men aged 65-74 years with elevated uric acid levels, there were no associations in other age groups between baseline uric acid and 16 year incidence of CHD (Shurtleff, 1970). In the Tecumseh Study (Myers et al 1968), no association was found between uric acid and CHD prevalence when age and weight were controlled for. In the Evans County Study (Klein et al 1973), a significant association was found between uric acid and CHD prevalence for white women and black men. These associations lost

their significance when blood pressure levels and cardiovascular medication were controlled for.

Some studies (Breckenridge, 1966; Isomaki, 1969; Lanese et al 1969; Thiele et al 1971) have noted an association between hypertension and high serum uric acid levels. Others (Evans et al 1969; Hall, 1965; Myers et al 1968) have found little or no association. Associations between serum uric acid and body weight have been reported by several researchers (Acheson and O'Brien, 1968; Evans et al 1969; Hall et al 1967; Lanese et al 1969; O'Sullivan, 1972; Thiele et al 1971).

Summary

The relationship between elevated uric acid levels and CHD, as well as hypertension, needs further clarification. In addition, little or no epidemiological research has attempted to evaluate the relationship of uric acid levels in children, especially children living in geographical regions, that have a substantial difference in CHD mortality.

SECTION VI: OBESITY

Epidemiological studies, as well as statistics kept by life insurance companies, indicate an association between obesity and the risk of coronary heart disease (Kannel et al 1966; Marks, 1960; Kannel et al 1961; Kannel et al 1976). It is the concensus of epidemiologists that the risk associated with an obese person

developing CHD is less than that associated with hypertension, hypercholesterolemia or smoking. The risk is due largely to the likelihood of the obese person having hypercholesterolemia, to be sedentary, to have carbohydrate intolerance and hypertension.

The association between obesity and hypertension has been well documented in children (Kannel et al 1961; Kannel et al 1967; Chiang et al 1969; Court et al 1974; Webber et al 1977; Stamler et al 1978; Londe, 1971). Whatever the association, obesity is not an attractive characteristic and correction may prevent or modify the coronary risk with which it is associated indirectly. Weight reduction, for example, has been shown to affect beneficially elevated blood pressure levels and glucose tolerance. Other studies (Mossberg, 1948; Mullins, 1958; Eid, 1970; Asher, 1966) have indicated that there is an association between childhood and adult obesity. In addition, there is a suggestion that overweight children become overweight adults (Charney et al 1976).

The positive correlation between blood pressure and ponderosity is well established (Chiang et al 1969). For adults this correlation was higher in younger than in older age groups (Epstein et al 1965; Chiang et al 1969). A similar relationship was noted for young populations in Evans County, Georgia (Johnson et al 1975), Muscatine, Iowa (Lauer et al 1975) and Bogalusa, Louisiana (Voors et al 1976).

The importance of obesity in adolescents for the development of hypertension in adulthood was suggested by the data from Oberman,

et al (1967), Paffenbarger et al (1968) and Abraham et al (1971), although the latter stated that their data, which were highly suggestive, did not reach statistical significance.

Epidemiological observations have indicated also a relationship between weight gained and blood pressure. Ashley and Kannel (1974) found that changes over time in blood pressure in the Framingham population were more closely related to gain in relative weight than to actual weight. Positive correlations between weight gain and blood pressure were reported by Oberman et al (1967), Stamler et al (1967), Heyden et al (1969), and Johnson et al (1973). Positive correlations between blood pressure and body weight in young populations were also shown by Levy et al (1946), Paffenbarger et al (1968), Londe and Goldring (1972) and Court et al (1974). In the last-named study, the rise in blood pressure lagged a number of years behind the weight increase.

Summary

Obesity is associated indirectly with the development of both CHD and hypertension. Furthermore, overweight children are likely to become overweight adults. The condition can be corrected or prevented through the practice of good nutritional habits.

SECTION VII: PHYSICAL ACTIVITY

The positive effects of physical activity on specific risk factors for CHD have been documented by some studies (Cooper et al 1976; Hickey et al 1975). Nevertheless, there is no conclusive evidence that can demonstrate a relationship between physical activity and coronary heart disease. Those studies which have provided evidence in the direction that physical activity may reduce the incidence of CHD have had methodological problems and, as a result, there is some doubt about their findings (Breslow and Buell, 1960; Kahn, 1963; Morris et al 1953; Morris and Crawford, 1958; Stamler et al 1960; Taylor et al 1962).

An inherent problem of these studies was that physical activity level was classified based upon the most recent job of the participant. This classification was probably incorrect because it did not take into account how long the job was held, nor did it allow consideration of leisure or recreational activity. Physical activity was not quantified and no assessment was made of the most productive activity.

Another problem was that old men and women with CHD often transferred to less active jobs and this fact was disregarded in some studies (Breslow and Buell, 1960; McDonough et al 1965; Morris et al 1953; Stamler et al 1960; Taylor et al 1962). Another methodological problem of these studies (Brunner, 1966; Frank et al 1966; Kahn, 1963; Morris et al 1953; Paffenbarger et al 1970; Taylor et al 1962; Zukel et al 1959) was that they did not take

into account CHD risk factors when making comparison between active and sedentary groups.

Results of the Health Insurance Plan Study in New York (Frank, 1968) showed that the early mortality of less active men was more than twice that of the most active subjects. This relationship persisted when the effects of smoking habits, previous CHD and elevated blood pressure were considered. Men who smoked cigarettes and were inactive had a five times greater probability of fatal myocardial infarction than men of the same age who did not smoke and were habitually more active.

A Finnish study (Karvonen et al 1961) found that middle-aged lumberjacks differed from other occupations only by fewer ECG changes indicative of past CHD or present myocardial ischemia. No differences were found in cholesterol levels, blood pressure and smoking habits so that the extreme heavy work of the lumberjacks seemed to offer protection from CHD in spite of higher risks. It was not reported if any former lumberjacks were included who had changed to less physically demanding occupations because of CHD.

The study of the San Francisco longshoremen also showed a protective effect of vigorous activity but the same was not true of moderate and light work levels. A lack of high energy expenditure work was found to contribute an effect on CHD risks which was at least partially independent of smoking habits, level of systolic blood pressure, diagnosed heart disease and relative body weight.

Negative results between physical activity and reduction of

CHD were shown in a large retrospective study by Stamler et al (1960). These negative results were probably due to very little activity gradient within the study population. Their prospective study of employees of a Chicago Utility Company (Stamler et al 1970) was also a negative possibility for the same reason. The Los Angeles prospective study of Chapman and Massey (1964) found no difference in the incidence of CHD according to level of physical activity as determined from the job title.

The effects of physical activity on specific CHD risk factors have been investigated by serveral researchers. The factors most often considered have been: serum cholesterol and lipids, blood pressure and body composition.

Hellerstein et al (1969) found that decreased serum cholesterol was associated with physical activity but also pointed out that some subjects followed dietary restriction of fats. Campbell (1968) reported decreased serum cholesterol in obese college-aged men but not in other men. The obese young men had significant decrease but no dietary change. Alterkruse and Wilmore (1973) found a significant decrease in serum cholesterol in a group of 39 male subjects following a ten-week run or jog program. The subjects did not change significantly in body weight and maintained a constant dietary pattern before and throughout the study.

Wood et al (1974) found total cholesterol and LDL cholesterol to be lower in a group of 41 middle-aged runners than in 743 randomly selected controls from the same geographical area. The

runners had averaged at least 15 miles per week for the previous year. Naughton and Balke (1964) found a decrease in serum cholesterol in 6 young men following 16 weeks of training. They also noted a transient cholesterol increase following strenuous active exercising. Decreases in cholesterol levels have also been noted in other training studies (Garrett et al 1966; Siegel et al 1970; Tzankoff et al 1972) of young to middle-aged men in which diet was not regulated or controlled and a significant weight loss was not found. It seems that the studies indicate that exercise training held little effect on cholesterol in individuals with normal levels to begin with (Campbell, 1968; Milesis, 1974).

Triglycerides have been found to decline with training in many studies (Alterkruse and Wilmore, 1973; Garrett et al 1966; Oscai et al 1972; Pollock et al 1969; Wood et al 1974) but in others have remained the same or increased (Allard et al 1973; Hunter et al 1968; Mann et al 1971; Terjung et al 1973).

The effect of physical training on blood pressure has been almost as inconclusive as that of cholesterol. Montoye et al (1972), in a population study of 1,700 males, 16 years of age and older, found systolic and diastolic pressures lower in active individuals. Boyer and Kasch (1970) trained hypertensive and normotensive subjects for 6 months and obtained significant diastolic and systolic pressure decreases in a normal group. Borderline hypertensive and normotensive subjects showed declines in both systolic and diastolic pressure following 6 months of training by Choquette and Ferguson

(1973). This was accomplished without significant weight loss.

Garrett et al (1966) found a decrease in diastolic pressure in 12 subjects following an intensive 6-week training program. Hunter and others (1968) found no change in blood pressure despite a significant weight loss following a 20-week jogging program. No change in non-elevated blood pressure was found by Taylor et al (1973) and by one group trained by Tzankoff et al (1972). Decreased normal range systolic pressures were noted by Wilmore et al (1970), Naughton and Nagle (1965), Mann et al (1969) and Hellerstein (1969). Decreased diastolic pressures were found by Pollock et al (1971).

Body weight and composition may be favourably affected by endurance exercise training as indicated by the results of numerous studies. Most of these have reported significant weight reductions or decreases in body fat or skin fold thicknesses associated with increased energy expenditures over a sufficient period of time. (Boileau et al 1971; Carter and Phillips, 1969; DeVries, 1970; Garrett et al 1966; Harris et al 1967; Hunter et al 1968; Joseph, 1968; Kasch and Carter, 1970). The period of training necessary to obtain these body composition changes and weight reductions varied from as short as 6 weeks to as long as 2 years. Some studies have reported no changes in weight or body composition following endurance training (Alterkruse and Wilmore, 1973; Boileau et al 1973; Siegel et al 1970; Terjung et al 1973).

Exercise may reduce coronary risk by altering plasma lipid and lipoprotein levels. Two of the major lipoprotein cholesterol

are LDL and HDL. High levels of HDL cholesterol may protect against CHD (Cooper et al 1976; DeVries, 1970; Fardy and Ilarinen, 1975; Frank et al 1966; Frank, 1968). There has been increased interest in HDL cholesterol because its negative association with CHD is stronger than the positive relationship between CHD and either total cholesterol or LDL (Miller et al 1977; Gordon et al 1977).

Highly trained athletes have lower levels of total and LDL cholesterol and higher levels of HDL cholesterol than do sedentary persons of the same age and sex. This is true of runners (Wood et al 1976; Wood et al 1974; Hartung et al 1980; Lehtonen and Viikari, 1978), skiers (Lehtonen and Viikari, 1978; Enger et al 1977) and swimmers (Zinet al 1972; Johnson and Wong, 1961). Hartung et al (1980) found that marathon runners have higher HDL cholesterol levels than joggers who, in turn, have higher levels than sedentary persons.

Summary

Although the role of exercise and its relationship to coronary heart disease and its various risk factors seem to be conflicting, there is no doubt that regular exercise, if done vigorously enough, can improve greatly cardiopulmonary function and exercise tolerance in healthy individuals. Most boys and girls have a positive attitude towards physical exercise. This attitude should be maintained.

SECTION VIII: ELEVATED RESTING HEART RATE

Life insurance actuaries (Society of Actuaries, 1959) have shown that a rapid, resting heart rate is associated with increased risk of coronary mortality. The Framingham and Western Electric Study have documented results which support the finding of the life insurance actuaries (Kannel, 1967; Paul et al 1963). A more detailed analysis of this variable as it relates to CHD was presented by data from the People's Gas Company's Study (Berkson et al 1970) which showed that the resting heart rates of 80 beats per minute or greater at initial examination in 1958 were associated with higher 10 year CHD mortality rates. This relationship of heart rate to risk was apparently independent of and additive to the effects of hypercholesterolemia, hypertension and cigarette smoking.

Elevated resting heart rate is also a predictor for the development of essential hypertension that can lead to a more persistent elevated arterial pressure and peripheral resistance (Julius, 1977). Other studies (Stamler et al 1975; Stamler et al 1975; Stamler et al 1975; Berglund and Wilhelmsen, 1975; Kahn et al 1972; Paffenbarger et al 1968) have shown that heart rate is a correlate of systolic and diastolic blood pressure and hypertension in addition to being a risk factor for hypertension.

Summary

Elevated resting heart rate has been shown to be associated

with increased CHD mortality and development of hypertension in adults. The distribution of this risk factor variable in children is not well documented.

Comments

In the foregoing literature review, it was shown that children, both black and white, do have elevated levels of cholesterol and lipoprotein cholesterol. Elevated systolic and diastolic blood pressures were also documented for children by various researchers. The distribution of other risk factor variables such as glucose, uric acid, obesity, smoking and heart rate were less well studied in children. Furthermore, none of the studies reviewed investigated the distribution of these risk factor variables in children living in regions that differed in adult CHD mortality rates.

The next chapter will deal with the materials and methods used in this study.

CHAPTER THREE

MATERIALS AND METHODS

Introduction

The materials and methods used in this study will be described in four sections as follows:

SECTION I - PREPARATIONS FOR CONDUCTING THE STUDY

SECTION II - DATA COLLECTION

SECTION III - BIOCHEMICAL METHODS

SECTION IV - STATISTICAL METHODS

SECTION I: PREPARATIONS FOR CONDUCTING THE STUDY

(i) Participating communities in high and low CHD mortality regions

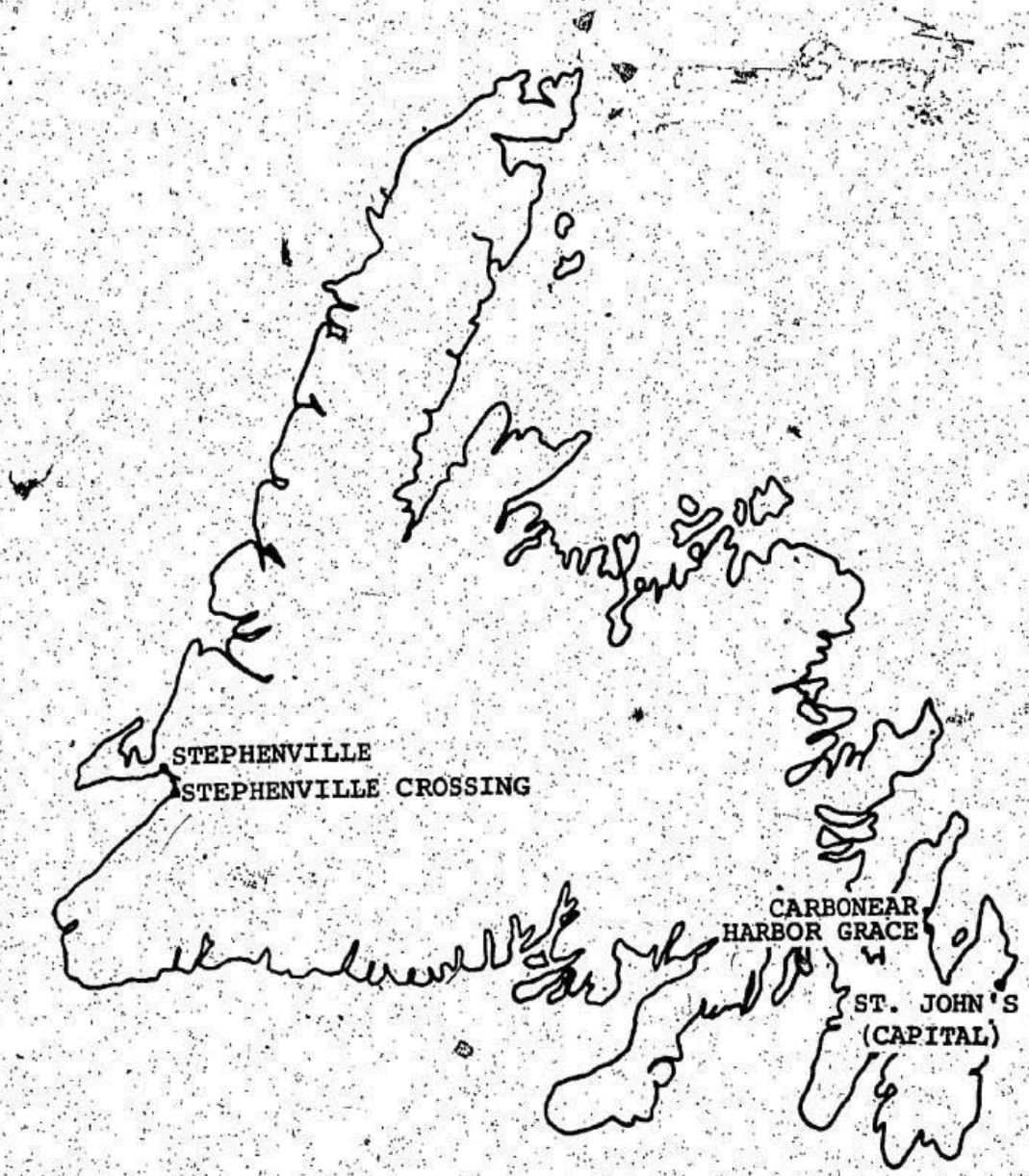
Children involved in this study attended schools chosen randomly. Schools were located in the twin communities of Carbonear and Harbour Grace located in the high coronary heart disease mortality region (Figure 2). Schools in the low mortality region were in the communities of Stephenville and Stephenville Crossing (Figure 2).

Carbonear and Harbour Grace are in close proximity to each other and are about 65 miles from the capital city, St. John's (Figure 2). The two communities in the low mortality region are approximately 495 miles from the capital city.

From an organizational point of view, it was convenient to do this study in these communities since laboratory technicians from hospitals in Carbonear and Stephenville were available to take blood from the children. Separation of the serum was done at the hospital laboratories at minimal costs. In addition, field workers in these

FIGURE 2

Map of Newfoundland showing location of communities, in High and Low CHD mortality regions, in which schools were located


STEPHENVILLE
STEPHENVILLE CROSSING
CARBONEAR
HARBOR GRACE
ST. JOHN'S
(CAPITAL)

communities were recruited without any difficulty.

(ii) Contact with school superintendents and school principals

The school superintendents and school principals were informed by letter about our intention to carry out this study. The purpose of the study was later explained to each school principal during a meeting.

At this meeting they were informed that boys and girls 8, 9, 10, 14, 15 and 16 years of age would be eligible to participate.

It was pointed out that consent from the parent of each child would be required. All principals indicated their willingness to cooperate and assured the author that parental consent would not be a problem.

(iii) Selection of schools

Five schools were chosen randomly from among ten schools in the high mortality region. A similar procedure was followed in choosing four from among the eleven schools in the low mortality region. Schools were chosen randomly in order to have a minimum sample size (see below) of 96 students of each sex in the 8-10 year age group and 96 of each sex in the 14-16 year age group from each mortality region. Entire classes consisting of children in the desired age groups were examined since it was felt that the response rate would be higher than if children were chosen individually.

(iv) Sample size determination

The sample size was calculated based on the variability of total serum cholesterol which was obtained from the literature. This variable has the greatest variability of all the other variables investigated in this study.

The sample size was calculated using the following formula:

$$n = \frac{z^2\alpha^2}{d^2}$$

where n = sample size

z = 1.96 corresponding to the 95% confidence level

α = 30 mg% corresponding to the standard deviation of cholesterol measurements in the population

d = 6 mg% - the need to have the sample mean within 6 mg% of the population mean

Inserting these values for the unknowns in the equation above, the value of n is 96. This is the minimum sample size required for each sex and age group.

A sample size of 96 would provide this study with an 89% chance of detecting a clinically relevant difference of 20 mg% in total serum cholesterol levels between two individuals at the 5% level. It is recommended (Altman, 1980) that significance tests should have a power of 80-90% in order to detect a true difference.

(v) Obtaining parental consent for Phase I of the study

Parental consent was requested two weeks before the survey took place in a school. Each eligible participant was given a letter to

which was attached a consent form to take home (Appendices A and B). Consent forms were returned by the children to their respective class teachers.

(vi) Response rate for Phase I of the study

In the high mortality region the participation rates of the 8-10 and 14-16 year olds were 87% and 89% respectively (Table 1). Non-participants did not consent or were absent from school during the time the survey team was on location.

There were 94% and 89% of the eligible 8-10 and 14-16 year olds respectively who participated in the low mortality region. As was the case in the high mortality region, the non-participants either did not consent or were absent from school at the time the survey took place.

(vii) Obtaining parental consent for Phase II of the study

Phase II of the study consisted of obtaining blood samples from the children who had participated in Phase I. Parental consent for this phase was obtained by sending a letter to which was attached a consent form (Appendices C and D).

(viii) Response rate for Phase II of the study

As mentioned above, only those children involved in the first phase of the study were eligible to give blood. Table 2 shows the participation rates of the 8-10 and 14-16 year olds in the high and

TABLE 1

RESPONSE RATE TO PHASE I OF THE STUDY
ACCORDING TO AGE GROUPS AND MORTALITY REGIONS

	AGED 8-10 YEARS		AGED 14-16 YEARS	
	No. Eligible	No. Participated	No. Elfgible	No. Participated
High Mortality Region	318	275 (87%)	429	383 (89%)
Low Mortality Region	271	255 (94%)	292	259 (89%)

low mortality regions were 88%, 91%, 94% and 78% respectively.

The higher non-response in the low mortality region was due to a major outbreak of influenza in the schools which resulted in many students being absent. An attempt was made to return to the schools at a later date but this was not possible since students were writing their year-end examinations.

SECTION II: DATA COLLECTION

(i) Preparation of children

The day before the actual examination, students were invited to a demonstration session. At this time students met members of the survey team and familiarized themselves with the various instruments. The author explained the purpose of the study and what would be done when they returned the next day.

Volunteers were requested from the class to participate in these demonstrations. Children were shown how their blood pressure would be taken and were allowed to use, under supervision, the blood pressure instruments. In addition, they were shown how an EKG recording was made.

Some expressed concern about giving blood. However, an attempt was made to alleviate their concerns by telling them that the procedure was not painful. They left with less anxiety.

(ii) Phase I of data collection

During this part of the data collection, members of the survey

TABLE 2

RESPONSE RATE TO PHASE II OF THE STUDY ACCORDING TO AGE GROUPS AND MORTALITY REGIONS

	AGED 8-10 YEARS		AGED 14-16 YEARS	
	No. Eligible	No. Participated	No. Eligible	No. Participated
High Mortality Region	275	242 (88%)	383	350 (91%)
Low Mortality Region	255	240 (94%)	259	201 (78%)

team were assisted by teachers in the examination of the younger children. They assisted in allocating identification numbers and measuring heights and weights. The presence of their teachers kept the children relaxed. Each participant sat for about 10-15 minutes after he/she entered the examination room. At this time, each of them was given a questionnaire with an identification number.

The sequence followed in measuring blood pressure, height, weight and recording of the electrocardiogram is shown in Figure 3. The questionnaire was administered by a nurse and was used to obtain information relating to family, length of residence, medical history and smoking habits (Appendix E).

(iii) Preparation of children for blood pressure measurements

In all schools blood pressure measurements were made in a spacious room in a comfortable environment. The students were advised by their teachers not to run and were encouraged to void before coming to the examining room. They were asked to remove their shoes and excess clothing, such as sweaters and coats.

(iv) Measurement of blood pressure with the Physiometrics SR-II Automatic Blood Pressure Recorder

This instrument was calibrated daily before use with the mercury column of a well-illuminated mercury baunamanometer. The Physiometrics Automatic Blood Pressure Recorder (Figure 4) is equipped with a standard rigid cuff designed for use on the upper

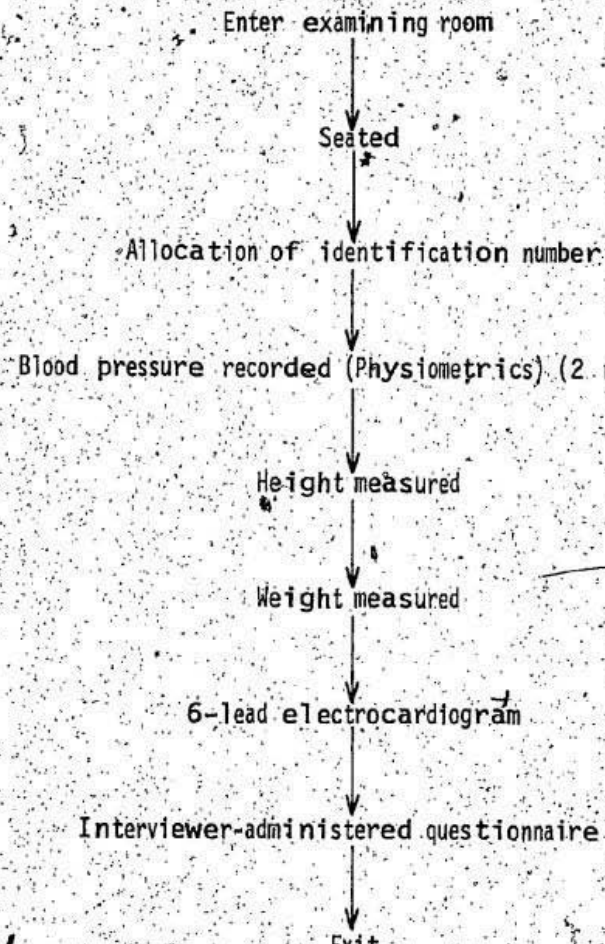


Figure 3

Sequence followed in the examination of children

FIGURE 4

Physiometrics Automatic Blood Pressure Recorder with Cuff



right arm. The cuff contains a microphone located under the fabric on the inside of the cuff which detects and records 1st and 4th phase Korotkoff sounds. The blood pressure was measured from the base upper right arm which was placed in the cuff with the microphone in the cuff located on the distal side (towards the elbow) and over the brachial artery.

After the arm was placed properly, the cuff was closed and the metal hook and velcro fastener were secured. The cuff was rapidly inflated until the pen indicated a reading of approximately 30 mm mercury higher than the anticipated systolic pressure. The cuff was then deflated at a rate of 2 mmHg per second. The inflation and deflation rates were pre-set on this instrument.

Two blood pressure measurements were taken five minutes apart from each child after he/she had been seated quietly for approximately 10-15 minutes. The mean of the two blood pressure readings was used in all analyses. Care was taken to make sure that after the child was seated, the arm was resting at heart level. Before the blood pressure was taken, the procedure of taking the blood pressure and what to expect was explained to each student.

Blood pressures were recorded on a circular 2 mm graduated disc. Each of the recordings was identified with the child's name, identification number and class grade and then stapled together. The blood pressure recording on the disc was read as follows: the first clear pen stroke which was followed by a series of strokes regularly spaced was the systolic blood pressure.

Diastolic blood pressure (4th phase) was read at the first sharp reduction in amplitude following the usual series of pen strokes at the maximum amplitude (Figure 5).

(v) Measurement of anthropometric factors

(a) Height

Height was measured using a steel tape fixed to a wall with the bottom end touching the ground. The child stood without shoes, with the back square against the wall and eyes looking straight ahead.

Each child stood as upright as possible, with heels on the ground. A measuring square was placed on the scalp and against the wall. Height was measured once to the nearest 0.5 cm and recorded in the appropriate section of the questionnaire.

(b) Weight

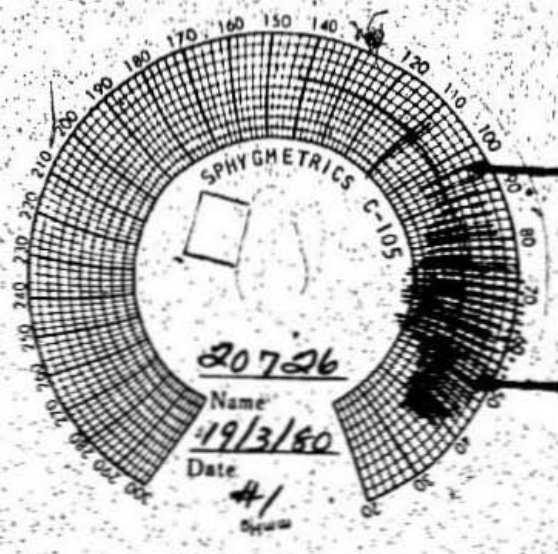
Weight was measured once with a floor model scale (SECA) to the nearest 0.5 kg without shoes and in the minimum amount of clothing. The scale was calibrated daily with a 5 kg weight. Everyone was instructed to stand in the middle of the scale without stooping. Measurements were recorded in the appropriate section of the questionnaire.

(vi) Electrocardiogram

A six-lead electrocardiogram was taken from each child with a direct writing single channel instrument (Hewlett Packard, Model

FIGURE 5

Blood Pressure Recording Disc showing points at which Systolic and Diastolic (IV) Blood Pressures were read



SYSTOLIC B.P.

DIASTOLIC (IV) B.P.

1500) at a paper speed of 25 mm per second. The conventional standardization of 1 mm amplitude for each 0.1 millivolt was used. This means that the sensitivity control of the recorder was set to give a set 10 mm deflection for 1 millivolt of potential.

The standard limb leads - 1, 2 and 3, as well as the unipolar AVR, AVL and AVF, were recorded after the child had been lying for approximately five minutes. Each recorded lead was coded and the name and grade of the student were written on the electrocardiogram for identification purposes. The recording was then stapled to the student's questionnaire.

(vii) Blood sampling

The blood drawing technique was explained to all participants. Attempts were made to alleviate whatever anxiety existed by assuring each child that the procedure is not painful and takes only a few minutes. Each child fasted for ten hours and was told that he/she would be given breakfast after blood was taken. Blood was drawn with the child in the supine position by trained technicians from community hospitals in the region. The effect of venous stasis was countered by releasing stasis as soon as the needle entered the vein.

In a few cases, blood flow into the vacutainer had to be helped by reapplying slight stasis on the vein. Two 10 ml samples of blood were taken in red top silicone coated tubes from each child. One of these samples was used to determine uric acid and

glucose levels and the other to measure triglyceride, total and HDL cholesterol concentrations.

Both blood samples from each person were labelled with the same identification number used in the first phase of the study. Samples were shipped about an hour after they were taken to the biochemistry laboratory of the hospital in the community for separation of the serum. They were centrifuged at 2000 revolutions per minute for ten minutes. The serum was transferred to plastic tubes with screw tops and labelled with the appropriate identification number.

After the serum was obtained, two individuals from each of the schools in the high and low mortality regions were selected randomly and both of their serum samples were split in half. Thus, each contributed four samples. Two of these samples were given a fictitious identification number that was known only to the author. These samples with the fictitious identification number were used as internal controls to test the accuracy of the lipid, uric acid and glucose analyses. There were ten such samples from the high mortality region and eight samples from the low mortality region. The results of the coefficient of variation of the measurement error between these samples, that is, the correctly labelled and the fictitiously labelled samples for the different variables are shown in Appendices F, H, I, K and L.

Serum samples obtained from children in the high mortality region were transported by the author to the Biochemistry Laboratory at the Sir Charles A. Janeway Children's Hospital in St.

John's for lipid analysis. Samples for uric acid and glucose determinations were taken by the author to the Biochemistry Laboratory at the Health Sciences Centre in St. John's.

The samples from the low mortality region were transported by air. They were picked up at the St. John's Airport and delivered to the respective biochemistry laboratories for analysis. Samples were kept at $\leq 4^{o}C$ during shipment.

All samples were frozen at the biochemistry laboratories until they were ready to be analysed. However, samples were never frozen, thawed and then refrozen. The lipid analyses, as well as serum uric acid and glucose analyses, were done within three weeks of the samples being delivered to the respective laboratories.

SECTION III: BIOCHEMICAL METHODS

(i) Determination of total serum cholesterol

Total serum cholesterol was determined by the CHOD-PAP method which is an Enzymatic Colorimetric Test designed for automated analysis by Boehringer Mannheim. The biochemical reactions of this test are as follows:

Cholesterol ester + H_2O $\xrightarrow{\text{cholesterol esterase}}$ cholesterol + fatty acid

Cholesterol + O_2 $\xrightarrow{\text{cholesterol oxidase}}$ ∇^4- cholestenone + H_2O_2

$2H_2O_2$ + 4-aminophenazone + phenol $\xrightarrow{\text{POD}}$ 4-(p-benzoquinone-monoimino) phenazone + $4H_2O$

In the first reaction, there is esterification of cholesterol ester by cholesterol esterase. In the second reaction, there is oxidation of cholesterol by cholesterol oxidase and formation of hydrogen peroxide. In the third reaction, hydrogen peroxide is determined by the peroxidase catalysed coupling of phenol with 4-aminophenazone. In this procedure, a 0.5 ml sample of sera was used to determine the cholesterol concentration. The range of the test was 0-500 mg cholesterol/100 ml.

Conventional method of standardization included the use of control sera Precinorm U, obtained from Boehringer Mannheim GmbH Diagnostica. Additional internal quality control was carried out by supplying the laboratory with eighteen blind duplicate samples for analysis as mentioned on page 77. The coefficient of variation of the measurement of the error between the samples is shown in Appendix F. External quality control was performed by obtaining lyophilized cholesterol sera of different concentrations from the Centre for Disease Control (CDC) in Atlanta, Georgia, U.S.A. The coefficient of variation for measurement error between values of the samples from CDC and the values of these samples obtained by the local laboratory is shown in Appendix G.

(ii) Determination of HDL-serum cholesterol

HDL-serum cholesterol was determined by the Heparin/$MnCl_2$ precipitation method. The procedure involved the preparation of reagent tubes for each sample and control to be assayed. The

reagents were heparin, manganese chloride and stabilizers. To each of the tubes was added 500 ul of well-mixed serum sample. The tubes were vortexed and left to sit at room temperature for 10 minutes. The next step involved centrifugation of the samples for 15 minutes at 3000 rpm.

The combination of heparin and manganese chloride bring about the precipitation of the lower density lipoprotein (LDL) and the very low density lipoprotein (VLDL) leaving the HDL fraction in solution. The solution containing the HDL fraction is then assayed for cholesterol using a spectrophotometer.

Internal quality control consisted of providing the laboratory with eighteen blind duplicate samples as mentioned on page 77. The coefficient of variation of the measurement error is shown in Appendix H.

(iii) Determination of serum triglyceride

Serum triglyceride analysis was done by the Kinetic UV method as designed for Automated Analysis by Boehringer Mannheim. The biochemical reactions of this test are as follows:

Triglycerides $\xrightarrow{\text{lipase/esterase}}$ glycerol + fatty acids

Glycerol + ATP $\xrightarrow{\text{GK}}$ glycerol-3-phosphate + ADP

ADP + PEP $\xrightarrow{\text{PK}}$ pyruvate + ATP

Pyruvate + NADH + H $\xrightarrow{\text{LDH}}$ lactate + NAD

The oxidation of NADH was followed at 340 nm. The reaction was performed at 30°C, pH 7.0 and was assumed to be complete after

10 minutes.

In addition, blind duplicate samples were analysed as mentioned on page 77, which gave the amount of error associated with the measurement of these samples (Appendix I). External quality control was carried out using samples of sera of known concentrations from the Centre for Disease Control in Atlanta. The sera were reconstituted for analysis. The values were compared with the known values to obtain the coefficient of variation of the measurement error as shown in Appendix J.

(iv) Determination of serum uric acid

Serum uric acid concentrations were determined by the Alkaline Phosphotungstate Method. In this method, sera were diluted with NaCl and then dialyzed against a sodium tungstate hydroxylamine solution. Phosphotungstic acid was added and the absorbance of the color produced in the analytical stream was measured at 660 nm.

In order to test the accuracy of the method, eighteen blind duplicate samples were analysed as mentioned on page 77. The coefficient of variation of the measurement error of these blind duplicate samples is shown in Appendix K.

(v) Determination of serum glucose

The glucose concentration in the sera was determined by the Technicon bound-hexokinase method. This procedure used an immobilized enzyme combination of hexokinase and glucose-6-

phosphate-dehydrogenase as catalysts. The reaction occurs in two steps: (1) hexokinase catalyses the phosphorylation of glucose by adenosine-5-triphosphate and (2) the catalytic dehydrogenation by glucose-6-phosphate dehydrogenase by the glucose-6-phosphate produced in the first reaction, coupled with the reduction of nicotinamide-adenine-dinucleotide, forms 6-phospho-glucose-8-lactone and NADH. The amount of NADH produced is in direct proportion to the concentration of glucose present in the serum sample. The absorbance was read at 340 nm in a flow cell.

In order to test the accuracy of this method, eighteen blind duplicate samples were sent to the laboratory for analysis as mentioned on page 77. The coefficient of variation of the measurement error associated with these samples is shown in Appendix L.

SECTION IV: STATISTICAL METHODS

Introduction

All data were transferred to punch cards and analyses were done using the Statistical Package for the Social Sciences (Nie et al 1975). Two-tailed tests of statistical significance were used throughout and the level for statistical significance chosen as $p \leq 0.05$.

Whenever there were two measurements of a variable per child, as in the case of systolic and diastolic blood pressure levels, the mean of the two measurements was used in the analysis of the data.

(i) Age

The age of each subject was calculated based upon the age at last birthday. The birth date of each child which was obtained from school registers was made available to the author by the respective class teachers on an information sheet (Appendix M).

(ii) Grouping of children into sex and age groups

Data were analysed for each sex according to two age groups: 8-10 years and 14-16 years. These age groups were chosen to assess whether there are differences in the distribution of CHD risk factor variables between prepubertal and pubertal children within and between high and low CHD mortality regions.

(iii) Distribution of variables

All the continuous variables were plotted to ascertain the shape of their distribution. It was found that triglyceride levels were not distributed normally but were skewed towards the right. A log transformation was done on this variable to normalise its distribution. However, the transformed values gave the same results as the untransformed. Other researchers reported similar findings (Goldstein et al 1973; Berenson et al 1980). The central limit theorem states that for a random sample from a distribution with finite variance, the distribution of a sample mean approaches normality as the sample size increases. Therefore, the original data were used in the statistical analysis.

(iv) Quetelet index (weight/height^3 and weight/height^2)

The quetelet index, which is an index of body mass, was computed to provide a measure of weight corrected for height. In this computation, weight was measured in kilograms and height in centimeters. The index, weight/height^3, was used to compute the body mass of children in the 8-10 year age group. This index was one of the five (see below) that was least correlated with height.

Weight/height^2 was used as an index of body mass for children in the 14-16 year age group. This index was chosen from among the five (see below) tested because it was least correlated with the height of children in this age group.

The following indices were tested: (1) weight/height, (2) weight/height^2, (3) weight/height^3, (4) weight/height$^{2.77}$, and (5) weight$^{0.425}$ x height$^{0.725}$ x 0.007184.

(v) Resting heart rate (beats per minute)

Each electrocardiogram was checked for irregularity of rhythm and other abnormalities. None was found. Resting heart rate was then calculated from lead 2 by determining the number of fifths of a second between consecutive beats. The R-R complex was chosen and the number of fifths elapsing before the same complex recurred was counted. Since there are only 300 fifths of a second in a minute, the heart rate (number of beats per minute) was determined by the number of fifths counted. For example, 1/5 second between consecutive beats would give a rate of 300 beats

per minute, 3/5 would give a rate of 100 beats per minute. The number of fifths was obtained with the aid of a caliper. This enabled measurements to be moved away from the EKG graph to a more convenient place on the graph paper, thereby facilitating the calculation of the heart rate.

(vi) Estimation of LDL cholesterol

The LDL cholesterol of each individual was calculated using the formula of Friedewald, Levy and Fredrickson (1972). The formula is: LDL cholesterol equals total cholesterol minus (HDL cholesterol + total triglyceride divided by 5).

(vii) Estimation of VLDL cholesterol

An estimate of VLDL cholesterol concentration for each individual was obtained by dividing the total serum triglyceride level by 5.

(viii) Calculation of lipid ratios

Two ratios were calculated. The total cholesterol to HDL-C ratio was obtained by dividing the total serum cholesterol by the HDL-cholesterol. The other ratio was derived by dividing the HDL-C by the square root of the LDL cholesterol.

(ix) Definition of elevated primary CHD risk factors

(a) Systolic blood pressure

Readings ≥ 125 mmHg were considered elevated for children in the 8-10 year age group. For the 14-16 year age group, readings ≥ 140 mmHg were designated elevated. These readings are considered to be hypertensive levels for these age groups (Dialogues in Hypertension, Vol. 2, No. 1, 1975).

(b) Diastolic (IV) blood pressure

Those children in the 8-10 year age group with diastolic (IV) blood pressure levels ≥ 80 mmHg were considered to have elevated blood pressure. Readings ≥ 85 mmHg were considered elevated for the 14-16 year age group. These readings are considered abnormal for children in these age groups (Dialogues in Hypertension, Vol. 2, No. 1, 1975).

(c) Total serum cholesterol

Anyone in the two age groups with cholesterol levels ≥ 200 mg% was considered to have elevated cholesterol. This is the cut-off value suggested for children in this age group (Drash 1972).

(d) Smoking

Data on this variable were collected from the 14-16 year age group only. For the purposes of this study those smoking ≥ 11 cigarettes per week were considered to be smoking excessively. This

was an arbitrary cut-off value.

(x) Descriptive statistics

(a) Mean and standard deviation

The mean and standard deviation of the following variables were calculated for each age and sex group: (1) systolic blood pressure, (2) diastolic (IV) blood pressure, (3) height, (4) weight, (5) quetelet index, (6) heart rate, (7) total serum cholesterol, (8) HDL-C, (9) LDL-C, (10) VLDL-C, (11) serum triglyceride, (12) serum glucose, (13) serum uric acid, (14) HDL-C/$\sqrt{\text{LDL-C}}$ and (15) total cholesterol/HDL-C.

(b) Prevalence and clustering

The prevalence and clustering of elevated primary CHD risk factors were calculated based on the levels considered elevated for the respective age groups (see page 86). These factors were: (1) systolic blood pressure, (2) diastolic (IV) blood pressure, (3) total serum cholesterol and (4) smoking $\geq$ 11 cigarettes per week (for the 14-16 year olds). The prevalence rate (%) of each risk factor was determined by dividing the number of individuals with elevated levels by the total number of individuals multiplied by 100.

Clustering of elevated primary risk factors was ascertained by identifying the number of children with one elevated factor, two elevated factors and three elevated factors respectively.

(xi) Inferential statistics

(a) t-test

The two-tailed t-test was used to test for differences between two means. It was based on the hypothesis that the sample means were equal in both regions.

(b) Differences between proportions

The testing of differences between proportions is theoretically analogous to the chi square test (Zar, 1974). The hypothesis tested was that the proportions of individuals with elevated prevalence and clustering of primary CHD risk factors were equal in both regions. The formula is shown below:

$$Z = \frac{P_1 - P_2}{\sqrt{P(1-P)\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}}$$

$$P_1 = \frac{a_1}{n_1} \; ; \quad P_2 = \frac{a_2}{n_2}$$

$$P = \frac{a_1 + a_2}{n_1 + n_2}$$

where n_1 is the sample of a number of individuals of which a_1 have a certain characteristic, where n_2 is the sample of a number of individuals of which a_2 have the same characteristic.

(c) Stepwise discriminant analysis

The WILKS method of stepwise discriminant analysis was used to identify those risk factor variables that were the best discriminators between similar age groups in the high and low mortality

regions. The analysis was also used to determine the important discriminating variables between the 8-10 and 14-16 year age groups within the two mortality regions.

"The stepwise procedure begins by selecting the single best discriminating variable according to a user determined criterion. A second discriminating variable is selected as the variable best able to improve the value of discrimination criterion in combination with the first variable. The third and subsequent variables are similarly selected according to their ability to contribute to further discrimination. At each step, variables already selected may be removed if they are found to reduce discrimination when combined with more recently selected variables. Eventually, either all variables will have been selected or it will be found that the remaining variables are no longer able to contribute to further discrimination" (Statistical Package for the Social Sciences, page 436).

Based upon the value of the Standardised Discriminant Function Coefficients obtained from the analysis, one is able to rank the variables with the most discriminating power in order of importance in their ability to discriminate between the two groups. The percentage contribution of each variable was obtained by dividing the coefficient of that variable by the sum of the coefficients for all the variables and multiplying by 100.

In this procedure each person must have data on each of the variables entered in the analysis and the general rule is that there should be ten persons to each variable.

The following variables were entered: (1) systolic blood pressure, (2) diastolic (IV) blood pressure, (3) height, (4) weight, (5) quetelet index (weight/height^3 for the 8-10 year age group and weight/height^2 for the 14-16 year age group), (6) heart rate, (7) total serum cholesterol, (8) HDL-C, (9) LDL-C, (10) VLDL-C, (11) serum triglyceride, (12) serum glucose, (13) serum uric acid, (14) HDL-C/$\sqrt{\text{LDL-C}}$, (15) total cholesterol/HDL-C, (16) sex and (17) smoking for the 14-16 year age group.

Smoking was coded as a dummy variable as follows: those smoking 1-10 cigarettes per week = 0 and those smoking $\geq$ 11 cigarettes per week = 1.

Height and weight were not included in the analysis between age groups within mortality regions since these two variables logically would be very important distinguishing factors in groups with such age differences - 8-10 versus 14-16 years. Smoking also was not included because data on this variable were collected from the 14-16 year age group only.

The results of the study are presented in the next chapter.

CHAPTER FOUR

RESULTS

Introduction

The results of this study will be presented in FOURTEEN SECTIONS. The mean values of the following variables will be reported in SECTIONS 1-8: (i) Systolic blood pressure, (ii) Diastolic (IV) blood pressure, (iii) Heart rate, (iv) Height, (v) Weight, (vi) Quetelet index, (vii) Total serum cholesterol, (viii) HDL cholesterol, (ix) LDL cholesterol, (x) VLDL cholesterol, (xi) Total cholesterol/HDL-C, (xii) HDL-C/$\sqrt{\text{LDL-C}}$, (xiii) Serum triglyceride, (xiv) Serum uric acid, and (xv) Serum glucose.

In each section these variables will be described under four main subheadings for both the 8-10 and 14-16 year age groups. These subheadings are indicated below using systolic blood pressure as an example:

(i) Mean systolic blood pressure levels of boys and girls in the 8-10 year age group in the high CHD mortality region

(ii) Mean systolic blood pressure levels of boys and girls in the 8-10 year age group in the low CHD mortality region

(iii) Mean systolic blood pressure levels of boys in the 8-10 year age group in high and low CHD mortality regions

(iv) Mean systolic blood pressure levels of girls in the 8-10 year age group in high and low CHD mortality regions.

Section 9 will report the results of stepwise discriminant analysis which identified those risk factor variables listed above that were most important in distinguishing between children in the 8-10 year age groups in the two regions and between age groups

within each region.

The remaining sections will cover areas as indicated below, using the four subheadings as outlined above. In Sections 10 and 11 - Prevalence of elevated CHD risk factor variables among 8-10 and 14-16 year olds, Sections 12 and 13 - Clustering of CHD risk factor variables for the 8-10 and 14-16 year olds and Section 14 - Frequency of hyperlipoproteinemia in the combined age groups in the two mortality regions.

SECTION 1

MEAN SYSTOLIC AND DIASTOLIC (IV) BLOOD PRESSURE LEVELS AND HEART RATE OF BOYS AND GIRLS IN THE 8-10 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean systolic blood pressure levels of boys and girls in the 8-10 year age group in the high CHD mortality region

As shown in Table 3, the mean systolic blood pressure of girls in the high mortality region was 1.7 mmHg higher than that of boys in the same region. The difference, however, was not statistically significant.

(ii) Mean systolic blood pressure levels of boys and girls in the 8-10 year age group in the low CHD mortality region

There was a small difference in the mean systolic blood pressure level between the sexes in the low mortality region. As indicated

TABLE 3

MEAN AND STANDARD DEVIATION (S.D.) OF BLOOD PRESSURE LEVELS AND HEART RATE OF BOYS AND GIRLS AGED 8-10 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 8-10 YEARS				
	Boys(H): n=133 Boys(L): n=129		Girls(H): n=142 Girls(L): n=126		
	Mean	S.D.	Mean	S.D.	p*
Systolic (mmHg)					
H	106.7	10.4	108.4	12.1	NS**
L	104.2	8.6	104.1	8.3	NS**
Diastolic (IV) (mmHg)					
H	68.3	7.3	70.4	8.3	<0.05
L	64.9	7.7	65.4	8.6	NS**
Heart Rate (beats/min)					
H	84.7	12.4	90.0	14.8	<0.01
L	76.2	11.6	84.9	12.8	<0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

in Table 3, the mean levels were almost identical - 104.2 mmHg for boys compared to 104.1 mmHg for girls.

(iii) Mean systolic blood pressure levels of boys in the 8-10 year age group in the high and low CHD mortality regions

As shown in Table 4, the mean systolic blood pressure level of boys was 2.5 mmHg higher in the high mortality region than in the low mortality region. The difference between the two groups was statistically significant at the 5% level.

(iv) Mean systolic blood pressure levels of girls in the 8-10 year age group in the high and low CHD mortality regions

Girls in the high mortality region had a higher mean systolic blood pressure level than girls in the low mortality region. The difference was 4.3 mmHg and was highly significant ($p<0.001$) as shown in Table 4.

Summary

The mean systolic blood pressure level of girls was slightly higher than that of boys in the high mortality region, however, the difference was not statistically significant. In the low mortality region, the mean systolic blood pressure levels of boys and girls were almost identical.

A comparison between regions showed that boys and girls in the high mortality region had significantly higher mean systolic blood

TABLE 4

MEAN AND STANDARD DEVIATION (S.D.) OF BLOOD PRESSURE LEVELS AND HEART RATE BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 8-10 YEARS

	AGED 8-10 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=133 Girls: n=142		Boys: n=129 Girls: n=126		
	Mean	S.D.	Mean	S.D.	p*
Systolic (mmHg)					
Boys	106.7	10.4	104.2	8.6	< 0.05
Girls	108.4	12.1	104.1	8.3	< 0.001
Diastolic (IV) (mmHg)					
Boys	68.3	7.3	64.9	7.7	< 0.001
Girls	70.4	8.3	65.4	8.6	< 0.001
Heart Rate (beats/min)					
Boys	84.7	12.4	76.2	11.6	< 0.001
Girls	90.0	14.8	84.9	12.8	< 0.01

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

pressure levels than their counterparts in the low mortality region.

(v) Mean diastolic (IV) blood pressure levels of boys and girls in the 8-10 year age group in the high CHD mortality region

The mean diastolic blood pressure of girls in the high mortality region was 2.1 mmHg higher than that of boys in this region. The difference was statistically significant at the 5% level as shown in Table 3.

(vi) Mean diastolic (IV) blood pressure levels of boys and girls in the 8-10 year age group in the low CHD mortality region

In the low mortality region, there was a 0.5 mmHg difference in the mean diastolic blood pressure between boys and girls. This difference was not statistically significant as shown in Table 3.

(vii) Mean diastolic (IV) blood pressure levels of boys in the 8-10 year age group in the high and low CHD mortality regions

Table 4 shows that the mean diastolic blood pressure level of boys in the high mortality region was 3.4 mmHg higher than that of their counterparts in the low mortality region. This difference was highly significant ($p<0.001$).

(viii) Mean diastolic (IV) blood pressure levels of girls in the 8-10 year age group in the high and low CHD mortality regions

Girls in the high mortality region had a mean diastolic blood pressure level that was 5.0 mmHg higher than that of girls in the low mortality region. This difference was highly significant ($p<0.001$) as indicated in Table 4.

Summary

In the high mortality region, girls had a significantly higher mean diastolic blood pressure level than boys. There was no significant difference in the mean diastolic blood pressure levels between boys and girls in the low mortality region.

A comparison of mean diastolic blood pressure levels between the regions show that boys and girls in the high mortality region had significantly higher mean diastolic blood pressure levels than their counterparts in the low mortality region.

(ix) Mean heart rate of boys and girls in the 8-10 year age group in the high CHD mortality region

Table 3 shows that in the high mortality region girls had a mean heart rate that was 5.3 beats per minute faster than that of boys. This difference was statistically significant ($p<0.01$).

(x) Mean heart rate of boys and girls in the 8-10 year age group in the low CHD mortality region.

Girls in the low mortality region had a mean heart rate that was 8.7 beats per minute faster than that of boys. This difference was highly significant ($p<0.001$) as indicated in Table 3.

(xi) Mean heart rate of boys in the 8-10 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a mean heart rate that was 8.5 beats per minute faster than boys in the low mortality region. This difference in the mean heart rate, as shown in Table 4, was highly significant ($p<0.001$).

(xii) Mean heart rate of girls in the 8-10 year age group in the high and low CHD mortality regions

The mean heart rate of girls in the high mortality region was 5.1 beats per minute faster than that of girls in the low mortality region. The difference was statistically significant at the 1% level as shown in Table 4.

Summary

Within the high and low mortality regions, girls had a significantly faster mean heart rate than boys.

A comparison of the mean heart rate between mortality regions showed that boys in the high mortality region had a significantly

faster heart rate than boys in the low mortality region. Similarly, girls in the high mortality region had a significantly faster heart rate than girls in the low mortality region.

SECTION 2

MEAN SYSTOLIC AND DIASTOLIC (IV) BLOOD PRESSURE LEVELS AND HEART RATE OF BOYS AND GIRLS IN THE 14-16 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean systolic blood pressure levels of boys and girls in the 14-16 year age group in the high mortality region

In the high mortality region, boys had a higher mean systolic blood pressure level than girls. As shown in Table 5., the mean difference was 5.7 mmHg and was highly significant ($p<0.001$).

(ii) Mean systolic blood pressure levels of boys and girls in the 14-16 year age group in the low CHD mortality region

Boys in the low mortality region also had a significantly higher mean systolic blood pressure level than girls. The difference in mean levels between the sexes was 6.2 mmHg and was highly significant ($p<0.001$) as shown in Table 5.

TABLE 5

MEAN AND STANDARD DEVIATION (S.D.) OF BLOOD PRESSURE LEVELS AND HEART RATE OF BOYS AND GIRLS AGED 14-16 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 14-16 YEARS				
	Boys(H): n=187 Boys(L): n=124		Girls(H): n=196 Girls(L): n=135		
	Mean	S.D.	Mean	S.D.	p*
Systolic (mmHg)					
H	121.8	17.5	116.1	13.4	<0.001
L	116.3	13.6	110.1	10.9	<0.001
Diastolic (IV) (mmHg)					
H	74.7	10.6	74.9	10.0	NS**
L	68.5	8.4	68.4	9.3	NS**
Heart Rate (beats/min)					
H	68.7	13.8	73.2	11.3	<0.001
L	69.9	12.1	73.6	12.5	<0.02

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(iii) Mean systolic blood pressure levels of boys in the 14-16 year age group in the high and low CHD mortality regions

Table 6 shows that the mean systolic blood pressure level of boys in the high mortality region was 5.5 mmHg higher than that of boys in the low mortality region. This difference between the two groups was statistically significant at the 1% level.

(iv) Mean systolic blood pressure levels of girls in the 14-16 year age group in the high and low CHD mortality regions

The mean systolic blood pressure level of girls in the high mortality region was significantly higher than that of girls in the low mortality region. The mean difference between the two groups was 6.0 mmHg and was highly significant ($p<0.001$) as indicated in Table 6.

Summary

The mean systolic blood pressure level of boys in the high mortality region was significantly higher than that of girls. Boys in the low mortality region also had a significantly higher mean systolic blood pressure level than girls.

A comparison of the mean systolic blood pressure levels between the two regions shows that boys and girls in the high mortality region had significantly higher mean systolic blood pressure levels than their counterparts in the low mortality region.

TABLE 6

MEAN AND STANDARD DEVIATION (S.D.) OF BLOOD PRESSURE LEVELS AND HEART RATE BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 14-16 YEARS

	AGED 14-16 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=187 Girls: n=196		Boys: n=124 Girls: n=135		
	Mean	S.D.	Mean	S.D.	p*
Systolic (mmHg)					
Boys	121.8	17.5	116.3	13.6	<0.01
Girls	116.1	13.4	110.1	10.9	<0.001
Diastolic (IV) (mmHg)					
Boys	74.7	10.6	68.5	8.4	<0.001
Girls	74.9	10.0	68.4	9.3	<0.001
Heart Rate (beats/min)					
Boys	68.7	13.8	69.9	12.1	NS**
Girls	73.2	11.3	73.6	12.5	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(v) Mean diastolic (IV) blood pressure levels of boys and girls in the 14-16 year age group in the high CHD mortality region

The mean difference in diastolic blood pressure levels between the sexes in the high mortality region was small. The difference was 0.2 mmHg and, as shown in Table 5 , it was not statistically significant.

(vi) Mean diastolic (IV) blood pressure levels of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the difference in the mean diastolic blood pressure level between boys and girls was 0.1 mmHg. As shown in Table 5 , this difference was not statistically significant.

(vii) Mean diastolic (IV) blood pressure levels of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a mean diastolic blood pressure level that was 6.2 mmHg higher than their counterparts in the low mortality region. This difference was highly significant ($p<0.001$) as shown in Table 6.

(viii) Mean diastolic (IV) blood pressure levels of girls in the 14-16 year age group in the high and low CHD mortality regions

As shown in Table 6, girls in the high mortality region had a

higher mean diastolic blood pressure level than their counterparts in the low mortality region. The difference was 6.5 mmHg and was highly significant ($p<0.001$).

Summary

No significant differences were found in the mean diastolic blood pressure levels of boys and girls in the high mortality region. A similar finding was noted for boys and girls in the low mortality region.

A comparison of the mean diastolic blood pressure levels of boys and girls in the high versus low mortality region showed that both boys and girls in the high mortality region had a significantly higher mean diastolic blood pressure level than their counterparts in the low mortality region.

(ix) Mean heart rate of boys and girls in the 14-16 year age group in the high CHD mortality region

There was a highly significant difference ($p<0.001$) between the mean heart rate of boys and girls in the high mortality region. As shown in Table 5, girls in the high mortality region had a mean heart rate that was 4.5 beats per minute faster than boys.

(x) Mean heart rate of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the mean heart rate of girls was 3.7 beats per minute faster than that of boys. This difference

was statistically significant at the 2% level as indicated in Table 5.

(xi) Mean heart rate of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the low mortality region had a mean heart rate that was 1.2 beats per minute faster than their counterparts in the high mortality region. The difference, as indicated in Table 6 was not statistically significant.

(xii) Mean heart rate of girls in the 14-16 year age group in the high and low CHD mortality regions

There was a small difference between the mean heart rate of girls in the two mortality regions. As shown in Table 6 , girls in the low mortality region had a mean heart rate that was 0.4 beats per minute faster than those in the high mortality region. Similarly, as was found for boys, the difference was not statistically significant.

Summary

The mean heart rate of girls in the high mortality region was significantly higher than that of boys. Likewise, in the low mortality region, girls had a significantly higher mean heart rate than boys.

A comparison between mortality regions showed that there was no difference in the mean heart rate between boys. A similar result was obtained for girls.

SECTION 3

MEAN ANTHROPOMETRIC FACTORS (HEIGHT, WEIGHT AND QUETELET INDEX) OF BOYS AND GIRLS IN THE 8-10 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean height of boys and girls in the 8-10 year age group in the high CHD mortality region

As shown in Table 7 , the mean height of boys and girls in the high mortality region was identical - 133.2 cm.

(ii) Mean height of boys and girls in the 8-10 year age group in the low CHD mortality region

In the low mortality region, as indicated in Table 7 , girls were 1.2 cm taller than boys. However, this difference was not statistically significant.

(iii) Mean height of boys in the 8-10 year age group in the high and low CHD mortality regions

There was a difference of 0.8 cm in the mean height of boys in the high and low mortality regions. The difference was not

TABLE 7

MEAN AND STANDARD DEVIATION (S.D.) OF ANTHROPOMETRIC FACTORS OF BOYS AND GIRLS AGED 8-10 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 8-10 YEARS				
	Boys(H): n=133 Boys(L): n=129		Girls(H): n=142 Girls(L): n=126		
	Mean	S.D.	Mean	S.D.	p*
Height (cm)					
H	133.2	7.2	133.2	7.5	NS**
L	134.0	7.0	135.2	7.8	NS**
Weight (kg)					
H	31.5	8.2	31.7	7.7	NS**
L	32.5	7.4	33.2	8.0	NS**
Quetelet Index (Wt/Ht^3)					
H	13.2	1.9	13.2	2.0	NS**
L	13.4	2.4	13.3	1.8	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

statistically significant as indicated in Table 8.

(iv) Mean height of girls in the 8-10 year age group in the high and low CHD mortality regions

Table 8 shows that girls in the low mortality region had a mean height which was 2.0 cm more than that of girls in the high mortality region. This difference was statistically significant at the 5% level.

Summary

The mean height of boys and girls in the high mortality region was the same. In the low mortality region, girls were slightly taller than boys, but the difference was not statistically significant.

There was no significant difference in the mean height of boys in the two mortality regions. Girls in the low mortality region, however, were significantly taller than their counterparts in the high mortality region.

(v) Mean weight of boys and girls in the 8-10 year age group in the high CHD mortality region

In the high mortality region, girls were heavier than boys. The difference was 0.2 kg but was not statistically significant as indicated in Table 7.

TABLE 8

MEAN AND STANDARD DEVIATION (S.D.) OF ANTHROPOMETRIC FACTORS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 8-10 YEARS

	AGED 8-10 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=133 Girls: n=142		Boys: n=129 Girls: n=126		
	Mean	S.D.	Mean	S.D.	p*
Height (cm)					
Boys	133.2	7.2	134.0	7.0	NS**
Girls	133.2	7.5	135.2	7.8	<0.05
Weight (kg)					
Boys	31.5	8.2	32.5	7.4	NS**
Girls	31.7	7.7	33.2	8.0	NS**
Quetelet Index (Wt/Ht^3)					
Boys	13.2	1.9	13.4	2.4	NS**
Girls	13.2	2.0	13.3	1.8	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(vi) Mean weight of boys and girls in the 8-10 year age group in the low CHD mortality region

In the low mortality region girls were 0.7 kg heavier than boys in the same region. This difference was not statistically significant as shown in Table 7.

(vii) Mean weight of boys in the 8-10 year age group in the high and low CHD mortality regions

Table 8 shows that boys in the low mortality region were 1.0 kg heavier than boys in the high mortality region. This difference was not statistically significant.

(viii) Mean weight of girls in the 8-10 year age group in the high and low CHD mortality regions

As shown in Table 8 the average weight of girls in the low mortality region was 1.5 kg greater than that of girls in the high mortality region. This difference was not statistically significant.

Summary

There were no statistically significant differences between the mean weight of boys and girls in the two mortality regions.

Boys in the two mortality regions showed no statistically significant differences in their mean weights. The same was true for girls.

(ix) Mean quetelet index (weight/height^3) of boys and girls in the 8-10 year age group in the high CHD mortality region

As shown in Table 7, the mean quetelet index was identical for boys and girls in the high mortality region.

(x) Mean quetelet index (weight/height^3) of boys and girls in the 8-10 year age group in the low CHD mortality region

Table 7 shows that in the low mortality region boys had a slightly higher mean quetelet index than girls. The difference was 0.1 and was not statistically significant.

(xi) Mean quetelet index (weight/height^3) of boys in the 8-10 year age group in the high and low CHD mortality regions

Table 8 shows that for boys the mean quetelet index was 0.2 higher in the low than in the high mortality region.

(xii) Mean quetelet index (weight/height^3) of girls in the 8-10 year age group in the high and low CHD mortality regions

The mean quetelet index was 0.1 higher for girls in the low than in the high mortality region. The difference was not statistically significant as indicated in Table 8.

Summary

No significant differences in the mean quetelet indices were found between the sexes within the mortality regions.

There was also no significant difference in the mean quetelet index between boys in the high and low mortality regions. A similar result was obtained for girls.

SECTION 4

MEAN ANTHROPOMETRIC FACTORS (HEIGHT, WEIGHT AND QUETELET INDEX) OF BOYS AND GIRLS IN THE 14-16 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean height of boys and girls in the 14-16 year age group in the high CHD mortality region

In the high mortality region, boys were 6.9 cm taller than girls. As indicated in Table 9 , the difference was highly significant ($p<0.001$).

(ii) Mean height of boys and girls in the 14-16 year age group in the low CHD mortality region

Boys in the low mortality region were 9.6 cm taller than girls. The difference (Table 9) was highly significant ($p<0.001$).

(iii) Mean height of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the low mortality region were 4.0 cm taller than boys in the high mortality region. The difference was highly significant

TABLE 9

MEAN AND STANDARD DEVIATION (S.D.) OF ANTHROPOMETRIC FACTORS OF BOYS AND GIRLS AGED 14-16 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 14-16 YEARS				
	Boys(H): n=187 Boys(L): n=124		Girls(H): n=196 Girls(L): n=135		
	Mean	S.D.	Mean	S.D.	p*
Height (cm)					
H	165.7	9.1	158.8	6.1	<0.001
L	169.7	7.3	160.1	6.1	<0.001
Weight (kg)					
H	61.0	11.6	57.1	10.4	<0.001
L	59.3	8.9	54.2	8.5	<0.001
Quetelet Index (Wt/Ht^2)					
H	22.2	4.0	22.6	3.7	NS**
L	20.5	2.4	21.2	3.0	<0.05

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

($p<0.001$) as shown in Table 10.

(iv) Mean height of girls in the 14-16 year age group in the high and low CHD mortality regions

Girls in the low mortality region were 1.3 cm taller than their counterparts in the high mortality region. This difference was not statistically significant (Table 10).

Summary

Within both mortality regions, boys were significantly taller than girls.

A comparison of the mean heights between regions showed that boys in the low mortality region were significantly taller than those in the high mortality region. In the case of girls, those in the low mortality region were taller, although not significantly, than those in the high mortality region.

(v) Mean weight of boys and girls in the 14-16 year age group in the high CHD mortality region

In the high mortality region, boys were 3.9 kg heavier than girls. The difference was highly significant ($p<0.001$) as shown in Table 9.

TABLE 10

MEAN AND STANDARD DEVIATION (S.D.) OF ANTHROPOMETRIC FACTORS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 14-16 YEARS

	AGED 14-16 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=187 Girls: n=196		Boys: n=124 Girls: n=135		
	Mean	S.D.	Mean	S.D.	p*
Height (cm)					
Boys	165.7	9.1	169.7	7.3	<0.001
Girls	158.8	6.1	160.1	6.1	NS**
Weight (kg)					
Boys	61.0	11.6	59.3	8.9	NS**
Girls	57.1	10.4	54.2	8.5	<0.01
Quetelet Index (Wt/Ht^2)					
Boys	22.2	4.0	20.5	2.4	<0.001
Girls	22.6	3.7	21.2	3.0	<0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(vi) Mean weight of boys and girls in the 14-16 year age group in the low CHD mortality region

Boys in the low mortality region were 5.1 kg heavier than girls. The difference was highly significant ($p<0.001$) (Table 9).

(vii) Mean weight of boys in the 14-16 year age group in the high and low CHD mortality regions

The mean weight of boys in the high mortality region was 1.7 kg more than their counterparts in the low mortality region. As indicated in Table 10, this difference was not statistically significant.

(viii) Mean weight of girls in the 14-16 year age group in the high and low CHD mortality regions

Girls in the high mortality region were 2.9 kg heavier than their counterparts in the low mortality region. The difference was statistically significant at the 1% level (Table 10).

Summary

Within both mortality regions, boys were significantly heavier than girls.

A comparison of the mean weights of boys between the two mortality regions showed that those in the high mortality region were slightly heavier, although not significantly, than those in the low mortality region. However, girls in the high mortality region were

significantly heavier than their counterparts in the low mortality region.

(ix) Mean quetelet index (weight/height^2) of boys and girls in the 14-16 year age group in the high CHD mortality region

The mean quetelet index was 0.4 higher for girls than boys in the high mortality region. The difference (Table 9) was not statistically significant.

(x) Mean quetelet index (weight/height^2) of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the mean quetelet index was 0.7 higher for girls than boys. The difference was significant at the 5% level (Table 9).

(xi) Mean quetelet index (weight/height^2) of boys in the 14-16 year age group in the high and low CHD mortality regions

The mean quetelet index of boys in the high mortality region was 1.7 higher than that of their counterparts in the low mortality region. The difference was highly significant ($p<0.001$) (Table 10).

(xii) Mean quetelet index (weight/height^2) of girls in the 14-16 year age group in the high and low CHD mortality regions

In the high mortality region, the mean quetelet index of girls was 1.4 higher than those in the low mortality region. As shown in

Table 10, this difference was highly significant ($p<0.001$).

Summary

The mean quetelet index was slightly higher for girls than boys in the high mortality region, but the difference was not statistically significant. In the low mortality region, girls had a significantly higher mean quetelet index than boys.

Boys and girls in the high mortality region had significantly higher mean quetelet indices than their counterparts in the low mortality region.

SECTION 5

MEAN LEVELS OF SERUM LIPIDS AND SERUM LIPOPROTEIN CHOLESTEROL OF BOYS AND GIRLS IN THE 8-10 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean total serum cholesterol levels of boys and girls in the 8-10 year age group in the high CHD mortality region

Table 11 shows that the mean total serum cholesterol level of girls in the high mortality region was 4.0 mg% higher than that of boys in the same region. The difference was not statistically significant.

TABLE 11

MEAN AND STANDARD DEVIATION (S.D.) OF TOTAL CHOLESTEROL AND SERUM LIPOPROTEIN CHOLESTEROL LEVELS OF BOYS AND GIRLS AGED 8-10 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 8-10 YEARS				
	Boys(H): n=115 Boys(L): n=121		Girls(H): n=127 Girls(L): n=118		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol					
H	176	26	180	31	NS**
L	180	27	192	31	<0.01
HDL-Cholesterol					
H	66	12	67	13	NS**
L	68	11	68	11	NS**
LDL-Cholesterol					
H	96	24	98	26	NS**
L	104	27	113	27	<0.01
VLDL-Cholesterol					
H	15	7	15	7	NS**
L	9	3	11	5	<0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant.

(ii) Mean total serum cholesterol levels of boys and girls in the 8-10 year age group in the low CHD mortality region

In the low mortality region, the mean total serum cholesterol level was 12.0 mg% higher for girls than boys. This difference was statistically significant at the 1% level (Table 11).

(iii) Mean total serum cholesterol levels of boys in the 8-10 year age group in the high and low CHD mortality region

Table 12 indicates that the mean total serum cholesterol level of boys in the low mortality region was 4.0 mg% higher than that of their counterparts in the high mortality region. The difference was not statistically significant.

(iv) Mean total serum cholesterol levels of girls in the 8-10 year age group in the high and low CHD mortality regions

Girls in the low mortality region had a mean total serum cholesterol level that was 12.0 mg% higher than those in the high mortality region. This difference was statistically significant at the 1% level (Table 12).

Summary

In the high mortality region, there was no significant difference in the mean serum cholesterol levels between boys and girls. In the low mortality region, however, the mean serum cholesterol level was significantly higher for girls than boys.

TABLE 12

MEAN AND STANDARD DEVIATION (S.D.) OF TOTAL CHOLESTEROL AND SERUM LIPOPROTEIN CHOLESTEROL LEVELS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 8-10 YEARS

	AGED 8-10 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=115 Girls: n=127		Boys: n=121 Girls: n=118		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol					
Boys	176	26	180	27	NS**
Girls	180	31	192	31	<0.01
HDL-Cholesterol					
Boys	66	12	68	11	NS**
Girls	67	13	68	11	NS**
LDL-Cholesterol					
Boys	96	24	104	27	<0.02
Girls	98	26	113	27	<0.001
VLDL-Cholesterol					
Boys	15	7	9	3	<0.001
Girls	15	7	11	5	<0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

No significant difference was found in the mean total serum cholesterol levels between boys in the high and low mortality regions. However, girls in the low mortality region had a significantly higher mean total serum cholesterol level than their counterparts in the high mortality region.

(v) Mean HDL cholesterol levels of boys and girls in the 8-10 year age group in the high CHD mortality region

The mean HDL cholesterol level of girls in the high mortality region was 1.0 mg% higher than that of boys in this region. This difference was not statistically significant (Table 11).

(vi) Mean HDL cholesterol levels of boys and girls in the 8-10 year age group in the low CHD mortality region

Boys and girls in the low mortality region had identical mean HDL cholesterol levels (Table 11).

(vii) Mean HDL cholesterol levels of boys in the 8-10 year age group in the high and low CHD mortality regions

There was a 2.0 mg% higher level of HDL cholesterol for boys in the low mortality region compared to those in the high mortality region. The difference was not statistically significant (Table 12).

(viii) Mean HDL cholesterol levels of girls in the 8-10 year age group in the high and low CHD mortality regions

In the low mortality region, girls had a 1.0 mg% higher mean HDL cholesterol level than their counterparts in the high mortality region. The difference was not statistically significant (Table 12).

Summary

There was no significant difference in the mean HDL cholesterol levels between boys and girls in the high mortality region. The finding was similar for the sexes in the low mortality region.

The mean HDL cholesterol levels were not significantly different between boys in the two mortality regions. The results were similar for girls.

(ix) Mean LDL cholesterol levels of boys and girls in the 8-10 year age group in the high CHD mortality region

The mean LDL cholesterol level was 2.0 mg% higher for girls than boys in the high mortality region. This difference was not statistically significant (Table 11).

(x) Mean LDL cholesterol levels of boys and girls in the 8-10 year age group in the low CHD mortality region

In the low mortality region, the mean LDL cholesterol level was 9.0 mg% higher for girls than boys. This difference was

statistically significant at the 1% level as shown in Table 11.

(xi) Mean LDL cholesterol levels of boys in the 8-10 year age group in the high and low CHD mortality regions

The mean LDL cholesterol level was 8.0 mg% higher for boys in the low mortality region compared to those in the high mortality region. This difference was statistically significant at the 2% level (Table 12).

(xii) Mean LDL cholesterol levels of girls in the 8-10 year age group in the high and low CHD mortality regions

The mean LDL cholesterol level was 5.0 mg% higher for girls in the low mortality region than their counterparts in the high mortality region. This difference was highly significant (Table 12).

Summary

In the high mortality region, there was no significant difference in the mean LDL cholesterol between the sexes. However, in the low mortality region, girls had a significantly higher mean LDL cholesterol level than boys.

The mean LDL cholesterol level was significantly higher for boys in the low mortality region compared to those in the high mortality region. A regional difference in the same direction was found for girls.

(xiii) Mean VLDL cholesterol levels of boys and girls in the 8-10 year age group in the high CHD mortality region

Identical mean values of VLDL cholesterol were obtained for boys and girls in the high mortality region (Table 11).

(xiv) Mean VLDL cholesterol levels of boys and girls in the 8-10 year age group in the low CHD mortality region

The 2.0 mg% higher mean VLDL cholesterol level for girls than boys in the low mortality region was highly significant ($p<0.001$) (Table 11).

(xv) Mean VLDL cholesterol levels of boys in the 8-10 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a mean VLDL cholesterol level that was 6.0 mg% higher than their counterparts in the low mortality region. This was a significant difference (Table 12).

(xvi) Mean VLDL cholesterol levels of girls in the 8-10 year age group in the high and low CHD mortality regions

Girls in the high mortality region had a mean VLDL cholesterol level that was 4.0 mg% higher than that of girls in the high mortality region. This difference was highly significant ($p<0.001$) (Table 12).

Summary

There was no difference in the mean levels of VLDL cholesterol between the sexes in the high mortality region. In the low mortality region, the mean VLDL cholesterol level was significantly higher for girls than boys.

Boys and girls in the high mortality region had significantly higher mean VLDL cholesterol levels than their counterparts in the low mortality region.

(xvii) Mean total cholesterol/HDL-C ratios of boys and girls in the 8-10 year age group in the high CHD mortality region

The mean total cholesterol/HDL-C ratio was almost the same, 2.4 versus 2,5, for boys and girls in the 8-10 year age group in the high mortality region (Table 13).

(xviii) Mean total cholesterol/HDL-C ratios of boys and girls in the 8-10 year age group in the low CHD mortality region

There was no significant difference in the mean total cholesterol/HDL-C ratios of boys and girls in the low CHD region in this age group (Table 13).

(xfx) Mean total cholesterol/HDL-C ratios of boys in the 8-10 year age group in the high and low CHD mortality regions

The mean lipid ratios did not differ significantly between mortality region for boys in this age group (Table 14).

TABLE 13

MEAN AND STANDARD DEVIATION (S.D.) OF LIPID RATIOS AND SERUM TRIGLYCERIDE LEVELS OF BOYS AND GIRLS AGED 8-10 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 8-10 YEARS				
	Boys(H): n=115 Boys(L): n=121		Girls(H): n=127 Girls(L): n=118		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol/HDL-C					
H	2.4	1.1	2.5	1.0	NS**
L	2.5	0.8	2.7	0.8	NS**
HDL-C/$\sqrt{\text{LDL-C}}$					
H	4.1	0.7	4.1	0.7	NS**
L	4.2	0.6	4.1	0.5	NS**
Triglyceride					
H	73	33	77	34	NS**
L	43	14	54	24	<0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

TABLE 14

MEAN AND STANDARD DEVIATION (S.D.) OF LIPID RATIOS AND SERUM TRIGLYCERIDE LEVELS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 8-10 YEARS

	AGED 8-10 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=115 Girls: n=127		Boys: n=121 Girls: n=118		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol/HDL-C					
Boys	2.4	1.1	2.5	0.8	NS**
Girls	2.5	1.0	2.7	0.8	NS**
$HDL\text{-}C/\sqrt{LDL\text{-}C}$					
Boys	4.1	0.7	4.2	0.6	NS**
Girls	4.1	0.7	4.1	0.5	NS**
Triglyceride					
Boys	73	33	43	14	<0.001
Girls	77	34	54	24	<0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(xx) Mean total cholesterol/HDL-C ratios of girls in the 8-10 year age group in the high and low CHD mortality regions

The total cholesterol/HDL-C ratio of girls in the low mortality region was 0.2 higher than those in the high mortality region. The difference was not significantly different (Table 14).

(xxi) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of boys and girls in the 8-10 year age group in the high CHD mortality region

Boys and girls in the high mortality region had identical mean HDL-C/ LDL-C ratios (Table 13).

(xxii) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of boys and girls in the 8-10 year age group in the low CHD mortality region

The mean lipid ratio was 4.2 for boys compared to 4.1 for girls. The difference was not significant (Table 13).

(xxiii) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of boys in the 8-10 year age group in high and low CHD mortality regions

There was a 0.1 difference which was not significant in the mean HDL-C/ LDL-C ratio between boys in the two mortality regions (Table 14).

(xxiv) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of girls in the 8-10 year age group in high and low CHD mortality regions

Girls in both mortality regions had a mean ratio of 4.1 (Table 14).

Summary

There was no significant difference in the mean lipid ratios between boys and girls in either mortality region.

Similarly, there was no significant difference in the mean values between mortality regions for boys or girls.

(xxv) Mean serum triglyceride levels of boys and girls in the 8-10 year age group in the high CHD mortality region

The mean serum triglyceride level was 4.0 mg% higher for girls than boys in the high mortality region. This difference was not statistically significant (Table 13).

(xxvi) Mean serum triglyceride levels of boys and girls in the 8-10 year age group in the low CHD mortality region

In the low mortality region, the mean serum triglyceride level was 11.0 mg% higher for girls than boys. As shown in Table 13, this difference was highly significant ($p<0.001$).

(xxvii) Mean serum triglyceride levels of boys in the 8-10 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a mean serum triglyceride level that was 30.0 mg% higher than their counterparts in the low mortality region. This difference was statistically significant ($p<0.001$) (Table 14).

(xxviii) Mean serum triglyceride levels of girls in the 8-10 year age group in the high and low CHD mortality regions

As shown in Table 14, the mean serum triglyceride level of girls in the high mortality region was 23.0 mg% higher than that of girls in the low mortality region. This was a significant difference ($p<0.001$).

Summary

Mean serum triglyceride levels were not significantly different between boys and girls in the high mortality region. In the low mortality region, however, the mean serum triglyceride level was significantly higher for girls than boys.

Regional comparison of the mean serum triglyceride levels showed that boys in the high mortality region had a significantly higher mean level than their counterparts in the low mortality region. A similar finding was obtained for girls.

SECTION 6

MEAN LEVELS OF SERUM LIPIDS AND SERUM LIPOPROTEIN CHOLESTEROL OF BOYS AND GIRLS IN THE 14-16 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean total serum cholesterol levels of boys and girls in the 14-16 year age group in the high CHD mortality region

Table 15 shows that the mean total cholesterol level of girls

TABLE 15

MEAN AND STANDARD DEVIATION (S.D.) OF TOTAL CHOLESTEROL AND SERUM LIPOPROTIN CHOLESTEROL LEVELS OF BOYS AND GIRLS AGED 14-16 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 14-16 YEARS				
	Boys(H): n=173 Boys(L): n=100		Girls(H): n=177 Girls(L): n=101		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol					
H	166	26	175	27	< 0.01
L	172	29	180	27	< 0.05
HDL-Cholesterol					
H	57	11	62	10	<0.001
L	58	13	63	12	< 0.01
LDL-Cholesterol					
H	98	23	102	26	<0.001
L	101	32	104	32	NS**
VLDL-Cholesterol					
H	11	6	10	4	NS**
L	11	5	12	5	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

was 9.0 mg% higher than that of boys in the high mortality region. The difference was statistically significant at the 1% level.

(ii) Mean total serum cholesterol levels of boys and girls in the 14-16 year age group in the low CHD mortality region

The mean total serum cholesterol level of girls in the low mortality region was 8.0 mg% higher than that of boys. The difference was significant at the 5% level (Table 15).

(iii) Mean total serum cholesterol levels of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the low mortality region had a mean total serum cholesterol level that was 6.0 mg% higher than their counterparts in the high mortality region. This difference, however, was not statistically significant (Table 16).

(iv) Mean total serum cholesterol levels of girls in the 14-16 year age group in the high and low CHD mortality regions

The mean total serum cholesterol level of girls in the low mortality region was 5.0 mg% higher than that of girls in the high mortality region. The difference was not statistically significant (Table 16).

Summary

Within both mortality regions, girls had a significantly higher

TABLE 16

MEAN AND STANDARD DEVIATION (S.D.) OF TOTAL CHOLESTEROL AND SERUM LIPOPROTEIN CHOLESTEROL LEVELS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 14-16 YEARS

	AGED 14-16 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=173 Girls: n=177		Boys: n=100 Girls: n=101		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol					
Boys	166	26	172	29	NS**
Girls	175	27	180	27	NS**
HDL-Cholesterol					
Boys	57	11	58	13	NS**
Girls	62	10	63	12	NS**
LDL-Cholesterol					
Boys	98	23	101	32	NS**
Girls	102	26	104	32	NS**
VLDL-Cholesterol					
Boys	11	6	11	5	NS**
Girls	10	4	12	5	< 0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

mean level of total serum cholesterol than boys.

There was no significant difference in the mean total serum cholesterol levels between boys in the two mortality regions. A similar result was obtained for girls.

(v) Mean HDL cholesterol levels of boys and girls in the 14-16 year age group in the high CHD mortality region

The mean HDL cholesterol level was 5.0 mg% higher for girls than boys in the high mortality region. This difference was highly significant ($p<0.001$) (Table 15).

(vi) Mean HDL cholesterol levels of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the mean HDL cholesterol level was 5.0 mg% higher for girls than boys. The difference was statistically significant at the 1% level (Table 15).

(vii) Mean HDL cholesterol levels of boys in the 14-16 year age group in the high and low CHD mortality regions

The mean HDL cholesterol level of boys in the low mortality region was 1.0 mg% higher than their counterparts in the high mortality region. This difference, as shown in Table 16, was not statistically significant.

(viii) Mean HDL cholesterol levels of girls in the 14-16 year age group in the high and low CHD mortality regions

Girls in the low mortality region had a mean HDL cholesterol level which was 1.0 mg% higher than their counterparts in the high mortality region. The difference was not statistically significant (Table 16).

Summary

Within both mortality regions, the mean HDL cholesterol level was significantly higher for girls than boys.

There was no significant difference in the mean HDL cholesterol levels for boys in the two regions. A similar result was obtained for girls.

(ix) Mean LDL cholesterol levels of boys and girls in the 14-16 year age group in the high CHD mortality region

The mean LDL cholesterol level was 4.0 mg% higher for girls than boys in the high mortality region. This difference was highly significant ($p<0.001$) (Table 15).

(x) Mean LDL cholesterol levels of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, girls had a mean LDL cholesterol level which was 3.0 mg% higher than boys. The differehce was not statistically significant (Table 15).

(xi) Mean LDL cholesterol levels for boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the low mortality region had a mean LDL cholesterol level which was 3.0 mg% higher than boys in the high mortality region. The difference was not statistically significant (Table 16).

(xii) Mean LDL cholesterol levels of girls in the 14-16 year age group in the high and low CHD mortality regions

There was a 2.0 mg% higher mean level of LDL cholesterol for girls in the low mortality region compared to those in the high mortality region. The difference was not statistically significant (Table 16).

Summary

The mean LDL cholesterol level was significantly higher for girls than boys in the high mortality region. No significant difference between the sexes was observed in the low mortality region.

There was no significant difference in the mean LDL cholesterol levels between mortality regions for boys or girls.

(xiii) Mean VLDL cholesterol levels of boys and girls in the 14-16 year age group in the high CHD mortality region

Boys in the high mortality region had a mean VLDL cholesterol level that was 1.0 mg% higher than that of girls. As indicated in Table 15, this difference was not statistically significant.

(xiv) Mean VLDL cholesterol levels of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the mean VLDL cholesterol level was 1.0 mg% higher for girls than boys. This difference was not statistically significant (Table 15).

(xv) Mean VLDL cholesterol levels of boys in the 14-16 year age group in high and low CHD mortality regions

The mean VLDL cholesterol level of boys in the high and low mortality regions was identical as shown in Table 16.

(xvi) Mean VLDL cholesterol levels of girls in the 14-16 year age group in high and low CHD mortality regions

The mean VLDL cholesterol level of girls in the low mortality region was 2.0 mg% higher than their counterparts in the high mortality region. The difference was highly significant ($p<0.001$) (Table 16).

Summary

There was no significant difference in the mean VLDL cholesterol levels between the sexes within mortality regions.

Boys in both regions had the same mean VLDL cholesterol level. The mean VLDL cholesterol level was significantly higher for girls in the low mortality region compared to those in the high mortality region.

(xvii) Mean total cholesterol/HDL-C ratios of boys and girls in the 14-16 year age group in the high CHD mortality region

As shown in Table 17, there was no significant difference between the two mean lipid ratios of boys and girls in the high mortality region.

(xviii) Mean total cholesterol/HDL-C ratios of boys and girls in the 14-16 year age group in the low CHD mortality region

There was no significant difference between the sexes in the mean lipid ratios (Table 17).

(xix) Mean total cholesterol/HDL-C ratios of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a slightly higher lipid ratio than their counterparts in the low mortality region. The difference, however, was not significant (Table 18).

TABLE 17

MEAN AND STANDARD DEVIATION (S.D.) OF LIPID RATIOS AND SERUM TRIGLYCERIDE LEVELS OF BOYS AND GIRLS AGED 14-16 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 14-16 YEARS				
	Boys(H): n=173 Boys(L): n=100		Girls(H): n=177 Girls(L): n=101		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol/HDL-C					
H	2.7	1.0	2.6	1.1	NS**
L	2.5	1.4	2.4	1.4	NS**
HDL-C/$\sqrt{\text{LDL-C}}$					
H	3.9	1.5	4.0	0.6	NS**
L	3.8	0.8	4.0	0.8	NS**
Triglyceride					
H	55	32	52	22	NS**
L	55	25	61	24	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

TABLE 18

MEAN AND STANDARD DEVIATION (S.D.) OF LIPID RATIOS AND SERUM TRIGLYCERIDE LEVELS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 14-16 YEARS

	AGED 14-16 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=173 Girls: n=177		Boys: n=100 Girls: n=101		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Total Cholesterol/HDL-C					
Boys	2.7	1.0	2.5	1.4	NS**
Girls	2.6	1.0	2.4	1.4	NS**
HDL-C/LDL-C					
Boys	3.9	1.5	3.8	0.8	NS**
Girls	4.0	0.6	4.0	0.8	NS**
Triglyceride					
Boys	55	32	55	25	NS**
Girls	52	22	61	24	<0.01

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(xx) Mean total cholesterol/HDL-C ratios of girls in the 14-16 year age group in the high and low CHD mortality regions

Although girls in the high mortality region had a higher mean lipid ratio than girls in the low mortality region, the difference did not reach statistical significance (Table 18).

(xxi) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of boys and girls in the 14-16 year age group in the high CHD mortality region

The mean lipid ratios of boys and girls were 3.9 and 4.0 respectively as shown in Table 17.

(xxii) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of boys and girls in the 14-16 year age group in the low CHD mortality region

Although the mean ratio was 0.2 higher for girls than boys in this region, the difference was not significant (Table 17).

(xxiii) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of boys in the 14-16 year age group in high and low CHD mortality regions

As shown in Table 18, the mean lipid ratios of boys in the high and low mortality regions were 3.9 and 3.8 respectively and were not significantly different.

(xxiv) Mean HDL-C/$\sqrt{\text{LDL-C}}$ ratios of girls in the 14-16 year age group in high and low CHD mortality regions

Girls in the two mortality regions had identical mean lipid

ratios of 4.0 (Table 18).

Summary

There was no significant difference in the mean lipid ratios between boys and girls in either mortality region.

The mean lipid ratios between mortality regions were not significantly different for boys or girls.

(xxv) Mean serum triglyceride levels of boys and girls in the 14-16 year age group in the high CHD mortality region

In the high mortality region, the mean serum triglyceride level of boys was 3.0 mg% higher than that of girls. This difference was not statistically significant (Table 17).

(xxvi) Mean serum triglyceride levels of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the mean serum triglyceride level of girls was 6.0 mg% higher than that of boys. This difference was not statistically significant (Table 17).

(xxvii) Mean serum triglyceride levels of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the high and low mortality regions had identical mean serum triglyceride levels (Table 18).

(xxviii) Mean serum triglyceride levels of girls in the 14-16 year age group in the high and low CHD mortality regions

The mean serum triglyceride level of girls in the low mortality region was 9.0 mg% higher than in the high mortality region. This difference was statistically significant at the 1% level (Table 18).

Summary

There was no statistically significant difference in the mean seurm triglyceride levels between the sexes within the mortality regions.

Boys in both regions had identical mean triglyceride levels. However, the mean level was significantly higher for girls in the low than in the high mortality region.

SECTION 7

MEAN SERUM URIC ACID AND SERUM GLUCOSE LEVELS OF BOYS AND GIRLS IN THE 8-10 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean serum uric acid levels of boys and girls in the 8-10 year age group in the high CHD mortality region

In the high mortality region, the mean serum uric acid levels of boys and girls were identical (Table 19).

TABLE 19

MEAN AND STANDARD DEVIATION (S.D.) OF SERUM URIC ACID AND SERUM GLUCOSE LEVELS OF BOYS AND GIRLS AGED 8-10 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 8-10 YEARS				
	Boys(H): n=112 Boys(L): n=119		Girls(H): n=125 Girls(L): n=114		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Uric Acid					
H	4.6	0.7	4.6	0.7	NS**
L	4.1	0.7	4.4	0.8	< 0.01
Glucose					
H	94	6.0	91	6.0	< 0.001
L	87	7.0	85	8.0	< 0.05

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(ii) Mean serum uric acid levels of boys and girls in the 8-10 year age group in the low CHD mortality region

The mean serum uric acid level of girls in the low mortality region was 0.3 mg% higher than that of boys. The difference was statistically significant at the 1% level (Table 19).

(iii) Mean serum uric acid levels of boys in the 8-10 year age group in the high and low CHD mortality regions

The mean serum uric acid level of boys in the high mortality region was 0.5 mg% higher than that of boys in the low mortality region. The difference was highly significant ($p<0.001$) (Table 20).

(iv) Mean serum uric acid levels of girls in the 8-10 year age group in the high and low CHD mortality regions

The mean serum uric acid level of girls in the high mortality region was 0.2 mg% higher than that of girls in the low mortality region.

This difference was statistically significant at the 5% level (Table 20).

Summary

There was no significant difference in the mean serum uric acid levels between boys and girls in the high mortality region. In the low mortality region, girls had a significantly higher mean serum uric acid level than boys.

TABLE 20

MEAN AND STANDARD DEVIATION (S.D.) OF SERUM URIC ACID AND SERUM GLUCOSE LEVELS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 8-10 YEARS

	AGED 8-10 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=112 Girls: n=125		Boys: n=119 Girls: n=114		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Uric Acid					
Boys	4.6	0.7	4.1	0.7	< 0.001
Girls	4.6	0.7	4.4	0.8	< 0.05
Glucose					
Boys	94	6.0	87	7.0	< 0.001
Girls	91	6.0	85	8.0	< 0.001

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

Regional comparisons showed that boys and girls in the high mortality region had significantly higher mean uric acid levels than their counterparts in the low mortality region.

(v) Mean serum glucose levels of boys and girls in the 8-10 year age group in the high CHD mortality region

The mean serum glucose level of boys in the high mortality region was 3.0 mg% higher than that of girls. The difference was highly significant ($p<0.001$) (Table 19).

(vi) Mean serum glucose levels of boys and girls in the 8-10 year age group in the low CHD mortality region

The mean serum glucose level of boys in the low mortality region was 2.0 mg% higher than that of girls. The difference was statistically significant at the 5% level (Table 19).

(vii) Mean serum glucose levels of boys in the 8-10 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a mean serum glucose level that was 7.0 mg% higher than their counterparts in the low mortality region. The difference was highly significant ($p<0.001$) (Table 20).

(viii) Mean serum glucose levels of girls in the 8-10 year age group in the high and low CHD mortality regions

In the high mortality region, girls had a mean serum glucose level that was 6.0 mg% higher than those in the low mortality region. The difference was highly significant ($p<0.001$) (Table 20).

Summary

Within both mortality regions, boys had a significantly higher mean serum glucose level than girls.

The mean serum glucose level of boys in the high mortality region was significantly higher than that of boys in the low mortality region. A similar result was obtained for girls.

SECTION B

MEAN SERUM URIC ACID AND SERUM GLUCOSE LEVELS OF BOYS AND GIRLS IN THE 14-16 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Mean serum uric acid levels of boys and girls in the 14-16 year age group in the high CHD mortality region

The mean serum uric acid level of boys in the high mortality region was 1.1 mg% higher than that of girls. The difference was highly significant ($p<0.001$) (Table 21).

TABLE 21

MEAN AND STANDARD DEVIATION (S.D.) OF SERUM URIC ACID AND SERUM GLUCOSE LEVELS OF BOYS AND GIRLS AGED 14-16 YEARS IN HIGH (H) AND LOW (L) CORONARY HEART DISEASE MORTALITY REGIONS

	AGED 14-16 YEARS				
	Boys(H): n=171 Boys(L): n= 94		Girls(H): n=177 Girls(L): n=101		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Uric Acid					
H	5.9	0.9	4.8	0.8	<0.001
L	6.0	1.0	4.7	0.7	<0.001
Glucose					
H	91	10	86	9	<0.001
L	88	11	86	11	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions

** Not statistically significant

(ii) Mean serum uric acid levels of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, the mean serum uric acid level was 1.3 mg% higher for boys than girls. The difference was highly significant ($p<0.001$) (Table 21).

(iii) Mean serum uric acid levels of boys in the 14-16 year age group in the high and low CHD mortality regions

The mean serum uric acid level of boys in the low mortality region was 0.1 mg% higher than their counterparts in the high mortality region. The difference was not statistically significant (Table 22).

(iv) Mean serum uric acid levels of girls in the 14-16 year age group in the high and low CHD mortality regions

The mean serum uric acid level of girls in the high mortality region was 0.1 mg% higher than that of girls in the low mortality region. However, as found for boys, the difference was not statistically significant (Table 22).

Summary

Boys within both mortality regions had a significantly higher mean serum uric acid level than girls.

There was no significant difference in the mean serum uric acid levels between boys or girls in the two mortality regions.

TABLE 22

MEAN AND STANDARD DEVIATION (S.D.) OF SERUM URIC ACID AND SERUM GLUCOSE LEVELS BETWEEN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS OF BOYS AND GIRLS AGED 14-16 YEARS

	AGED 14-16 YEARS				
	High Mortality Region		Low Mortality Region		
	Boys: n=171 Girls: n=177		Boys: n= 94 Girls: n=101		
Level, mg%	Mean	S.D.	Mean	S.D.	p*
Uric Acid					
Boys	5.9	0.9	6.0	1.0	NS**
Girls	4.8	0.8	4.7	0.7	NS**
Glucose					
Boys	91	10	88	11	<0.05
Girls	86	9	86	11	NS**

* Based on a two-tailed t-test of the hypothesis that the sample means are equal in both regions.

** Not statistically significant

(v) Mean serum glucose levels of boys and girls in the 14-16 year age group in the high CHD mortality region

In the high mortality region, boys had a 5.0 mg% higher mean serum glucose level than girls. The difference was highly significant ($p<0.001$) (Table 21).

(vi) Mean serum glucose levels of boys and girls in the 14-16 year age group in the low CHD mortality region

The mean serum glucose level of boys in the low mortality region was 2.0 mg% higher than that of girls. This difference was not statistically significant (Table 21).

(vii) Mean serum glucose levels of boys in the 14-16 year age group in the high and low CHD mortality regions

Boys in the high mortality region had a mean serum glucose level that was 3.0 mg% higher than that of boys in the low mortality region. As shown in Table 22, this difference was statistically significant at the 5% level.

(viii) Mean serum glucose levels of girls in the 14-16 year age group in the high and low CHD mortality regions

Girls in the high and low mortality regions had identical mean serum glucose levels as shown in Table 22.

Summary

Boys had a significantly higher mean serum glucose level than girls in the high mortality region. There was no significant difference in the mean serum glucose levels between the sexes in the low mortality region.

Boys in the high mortality region had a significantly higher mean serum glucose level than boys in the low mortality region. In the case of girls, the mean serum glucose levels were the same in both mortality regions.

General Summary

The significant findings documented in the preceding sections 1-8 are summarised in tables 23, 24, 25 and 26.

Table 23 shows that except for glucose there were no consistent risk factor variables that were significantly higher for boys than girls in the 8-10 (prepubertal) and 14-16 (pubertal) year age groups within the high mortality region. This table also shows that within this mortality region, heart rate was the only risk factor variable that was significantly higher for girls than boys in both age groups. Within the low mortality region, glucose was significantly higher for boys than girls in both age groups and heart rate and total serum cholesterol were significantly higher for girls than boys in the 8-10 and 14-16 year age groups (Table 24).

These results suggest that differences in risk factor variables between the age groups within both regions become more pronounced

TABLE 23

SUMMARY OF RISK FACTOR VARIABLES WHICH WERE SIGNIFICANTLY HIGHER FOR BOYS OR GIRLS AGED 8-10 AND 14-16 YEARS WITHIN HIGH CHD MORTALITY REGION

AGED 8-10 YEARS WITHIN HIGH CHD MORTALITY REGION	AGED 14-16 YEARS WITHIN HIGH CHD MORTALITY REGION
(a) Risk Factor Variables Significantly Higher for Boys than Girls	(a) Risk Factor Variables Significantly Higher for Boys than Girls
1. Uric Acid	1. Systolic Blood Pressure
2. Glucose	2. Height
	3. Weight
	4. Glucose
(b) Risk Factor Variables Significantly Higher for Girls than Boys	(b) Risk Factor Variables Significantly Higher for Girls than Boys
1. Heart Rate	1. Heart Rate
2. Diastolic (IV) Blood Pressure	2. Total Serum Cholesterol
	3. HDL-C
	4. VLDL-C

TABLE 24

SUMMARY OF RISK FACTOR VARIABLES WHICH WERE SIGNIFICANTLY HIGHER FOR BOYS OR GIRLS AGED 8-10 AND 14-16 YEARS WITHIN THE LOW CHD MORTALITY REGION

AGED 8-10 YEARS WITHIN LOW CHD MORTALITY REGION	AGED 14-16 YEARS WITHIN LOW CHD MORTALITY REGION
(a) Risk Factor Variables Significantly Higher for Boys than Girls	(a) Risk Factor Variables Significantly Higher for Boys than Girls
1. Uric Acid	1. Systolic Blood Pressure
2. Glucose	2. Height
	3. Weight
	4. Glucose
(b) Risk Factor Variables Significantly Higher for Girls than Boys	(b) Risk Factor Variables Significantly Higher for Girls than Boys
1. Heart Rate	1. Heart Rate
2. Total Serum Cholesterol	2. Total Serum Cholesterol
3. LDL-C	3. HDL-C
4. VLDL-C	4. Quetelet Index (Wt/Ht^2)
5. Triglyceride	5. Uric Acid

as they get older. These observed differences that seem to occur with increase in age could be due to maturational differences between the two groups within both regions. It should be noted, however, that for boys in both age groups within the two regions, the same risk factor variables were significantly higher than girls.

Two risk factor variables were identified as being consistently different between the two mortality regions in both age groups. As shown in table 25 and 26, systolic and diastolic blood pressures were significantly higher for both boys and girls in the high, as opposed to the low, mortality region. The finding occurred in both the 8-10 and 14-16 year age groups. There were no other risk factor variables that indicated differences between the mortality regions for both age groups.

SECTION 9

DISCRIMINATING RISK FACTOR VARIABLES BETWEEN CHILDREN IN THE 8-10 AND 14-16 YEAR AGE GROUPS IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Discriminating risk factor variables between children in the 8-10 and 14-16 year age groups in the high CHD mortality region

Eight risk factor variables were chosen by the discriminant function analysis that discriminated best between the two age groups in the high CHD mortality region. As shown in Table 27, the

TABLE 25

SUMMARY OF RISK FACTOR VARIABLES WHICH WERE SIGNIFICANTLY HIGHER IN EITHER HIGH OR LOW CHD MORTALITY REGION FOR BOYS AND/OR GIRLS AGED 8-10 YEARS

AGED 8-10 YEARS

Risk Factor Variables Significantly Higher in the High Mortality Region than the Low Mortality Region	Risk Factor Variables Significantly Higher in the Low Mortality Region than the High Mortality Region
(a) For Boys and Girls	(a) For Boys and Girls
1. Systolic Blood Pressure	1. LDL-C
2. Diastolic (IV) Blood Pressure	
3. Heart Rate	
4. Triglyceride	
5. Uric Acid	
6. Glucose	
7. VLDL-C	
(b) For Girls Only	(b) For Girls Only
--	1. Total Cholesterol
	2. Height

TABLE 26

SUMMARY OF RISK FACTOR VARIABLES WHICH WERE SIGNIFICANTLY HIGHER IN EITHER HIGH OR LOW CHD MORTALITY REGION FOR BOYS AND/OR GIRLS AGED 14-16 YEARS

AGED 14-16 Years

Risk Factor Variables Significantly Higher in the High Mortality Region than the Low Mortality Region	Risk Factor Variables Significantly Higher in the Low Mortality Region than the High Mortality Region
(a) For Boys and Girls	(a) For Boys and Girls
1. Systolic Blood Pressure	--
2. Diastolic (IV) Blood Pressure	
3. Quetelet Index (Wt/Ht^2)	
(b) For Boys Only	(b) For Boys Only
1. Glucose	1. Height
(c) For Girls Only	(c) For Girls Only
1. Weight	1. VLDL-C
	2. Triglyceride

TABLE 27

STANDARDISED DISCRIMINANT FUNCTION COEFFICIENTS AND PERCENTAGE CONTRIBUTION OF VARIABLES, LISTED IN DESCENDING ORDER OF IMPORTANCE, WHICH DISCRIMINATE BETWEEN CHILDREN IN THE 8-10 AND 14-16 YEAR AGE GROUPS IN THE HIGH CHD MORTALITY REGION

Risk Factor Variables	Standardised Discriminant Function Coefficient	% Contribution
1. Heart Rate	0.70754*	24.1
2. Systolic B.P.	0.56400*	19.2
3. Triglyceride	0.54451*	18.5
4. Uric Acid	0.32302*	11.0
5. Sex	0.27032*	9.2
6. HDL-C	0.22833*	7.8
7. Total Chol/HDL-C	0.16411*	5.6
8. Diastolic (IV) B.P.	0.13469*	4.6

* $p<0.0001$

No. of children in the 8-10 year age group correctly classified by these variables = 213/235 = 91%

No. of children in the 14-16 year age group correctly classified by these variables = 307/340 = 90%

most important was heart rate followed by systolic blood pressure, triglyceride, uric acid, sex, HDL-C, total cholesterol/HDL-C and diastolic (IV) blood pressure.

The percentage contribution of each variable is also shown (Table 27). These variables classified correctly 91% and 90% of the children in the 8-10 and 14-16 year age groups respectively.

(ii) Discriminating risk factor variables between children in the 8-10 and 14-16 year age groups in the low CHD mortality region

As indicated by the magnitude of the standardised discriminant function coefficients, eight variables were most important in distinguishing between the 8-10 and 14-16 year age groups in the low CHD mortality region. The most important risk factor variable was uric acid followed by HDL-C, HDL-C/$\sqrt{\text{LDL-C}}$, heart rate, systolic blood pressure, sex, total cholesterol/HDL-C and triglyceride (Table 28). As indicated in the table, the percentage contribution of the variables ranged from 17.1% for uric acid to 4.3% for triglyceride. Eighty-seven percent of the children in the 8-10 year age group were classified correctly by these variables, compared to 86% for the 14-16 year age group.

(iii) Discriminating risk factor variables between children in the 8-10 year age groups in the high and low CHD mortality regions

Table 29 shows the eight variables identified as most important in distinguishing between the 8-10 year age groups in the high and

TABLE 28

STANDARDISED DISCRIMINANT FUNCTION COEFFICIENTS AND PERCENTAGE CONTRIBUTION OF VARIABLES, LISTED IN DESCENDING ORDER OF IMPORTANCE, WHICH DISCRIMINATE BETWEEN CHILDREN IN THE 8-10 AND 14-16 YEAR AGE GROUPS IN THE LOW CHD MORTALITY REGION

Risk Factor Variables	Standardised Discriminant Function Coefficients	% Contribution
1. Uric Acid	0.64654*	17.1
2. HDL-C	0.62844*	16.6
3. HDL-C/$\sqrt{\text{LDL-C}}$	0.60325*	16.0
4. Heart Rate	0.58241*	15.4
5. Systolic B.P.	0.47465*	12.5
6. Sex	0.42288*	11.2
7. Total Chol/HDL-C	0.26002*	6.9
8. Triglyceride	0.16174*	4.3

* $p<0.0001$

No. of children in the 8-10 year age group correctly classified by these variables = 200/230 = 87%

No. of children in the 14-16 year age group correctly classified by these variables = 164/191 = 86%

TABLE 29

STANDARDISED DISCRIMINANT FUNCTION COEFFICIENTS AND PERCENTAGE CONTRIBUTION OF RISK FACTOR VARIABLES, LISTED IN DESCENDING ORDER OF IMPORTANCE, WHICH DISCRMINATE BETWEEN CHILDREN IN THE 8-10 YEAR AGE GROUPS IN HIGH AND LOW CHD MORTALITY REGIONS

Risk Factor Variables	Standardised Discriminant Function Coefficients	% Contribution
1. Triglyceride	0.75580*	24.0
2. Total Cholesterol	0.51164*	16.2
3. Glucose	0.44832*	14.2
4. Weight	0.40907*	13.0
5. Uric Acid	0.33283*	10.6
6. Heart Rate	0.28822*	9.2
7. Diastolic (IV) B.P.	0.21587*	6.9
8. HDL-C	0.18609*	5.9

* $p<0.0001$

No. of children in the high mortality region correctly classified by these variables = 192/235 = 82%

No. of children in the low mortality region correctly classified by these variables = 195/229 = 85%

low mortality regions. As indicated by the magnitude of the standardised discriminant function coefficients, triglyceride was the most important of the eight followed by total cholesterol, glucose, weight, uric acid, heart rate, diastolic (IV) blood pressure and HDL-C. The percentage contribution of each of these variables is shown also in the table. These variables correctly classified 82% of the children in the high mortality region and 85% in the low mortality region.

(iv) Discriminating risk factor variables between children in the 14-16 year age groups in the high and low CHD mortality regions

Ten risk factor variables were identified as being most important in distinguishing between the 14-16 year age groups in the high and low CHD mortality regions (Table 30). In descending order of importance, they were: weight, height, quetelet index (weight/height^2), diastolic (IV) blood pressure, sex, systolic blood pressure, total cholesterol, heart rate, triglyceride and uric acid. Although weight and height had a greater percentage contribution as discriminating factors than quetelet index, the latter removes the effect of height on weight. These ten variables classified correctly 68% of the children in the high mortality region and 76% in the low mortality region.

TABLE 30

STANDARDISED DISCRIMINANT FUNCTION COEFFICIENTS, AND PERCENTAGE CONTRIBUTION OF RISK FACTOR VARIABLES, LISTED IN DESCENDING ORDER OF IMPORTANCE, WHICH DISCRIMINATE BETWEEN CHILDREN IN THE 14-16 YEAR AGE GROUPS IN HIGH AND LOW CHD MORTALITY REGIONS

Risk Factor Variables	Standardised Discriminant Function Coefficients	% Contribution
1. Weight	1.99016*	28.3
2. Height	1.76486*	25.1
3. Quetelet Index (Wt/Ht^2)	1.38123*	19.6
4. Diastolic (IV) B.P.	0.63212*	9.0
5. Sex	0.29480*	4.2
6. Systolic B.P.	0.26199*	3.7
7. Total Cholesterol	0.23778*	3.4
8. Heart Rate	0.17665*	2.5
9. Triglyceride	0.16658*	2.4
10. Uric Acid	0.12770*	1.8

* $p<0.0001$

No. of children in the high mortality region correctly classified by these variables = 232/340 = 68%

No. of children in the low mortality region correctly classified by these variables = 145/191 = 76%

SECTION 10

PREVALENCE OF ELEVATED CHD RISK FACTORS (TOTAL SERUM CHOLESTEROL, SYSTOLIC AND DIASTOLIC (IV) BLOOD PRESSURE) IN THE 8-10 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure levels for boys and girls in the 8-10 year age group in the high CHD mortality region

In the high mortality region, 6.7% more girls than boys had total serum cholesterol levels $\geq$ 200 mg%. This difference was not statistically significant (Figure 6). Systolic blood pressure levels $\geq$ 125 mmHg were 3.2% more prevalent for girls than boys in this mortality region. This difference was not statistically significant (Figure 6). In this mortality region, 4.6% more girls than boys had elevated diastolic blood pressure ($\geq$80 mmHg) and, as shown in Figure 6 , this difference was not significant..

(ii) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure levels for boys and girls in the 8-10 year age group in the low CHD mortality region

The prevalence of elevated total serum cholesterol levels ($\geq$200 mg%) was 9.1% higher for girls than boys in the low mortality region. However, the difference was not statistically significant as shown in Figure 7. In this region, 0.8% of boys had elevated

FIGURE 6

Prevalence of elevated primary CHD risk factors for boys and girls aged 8-10 years in the high mortality region

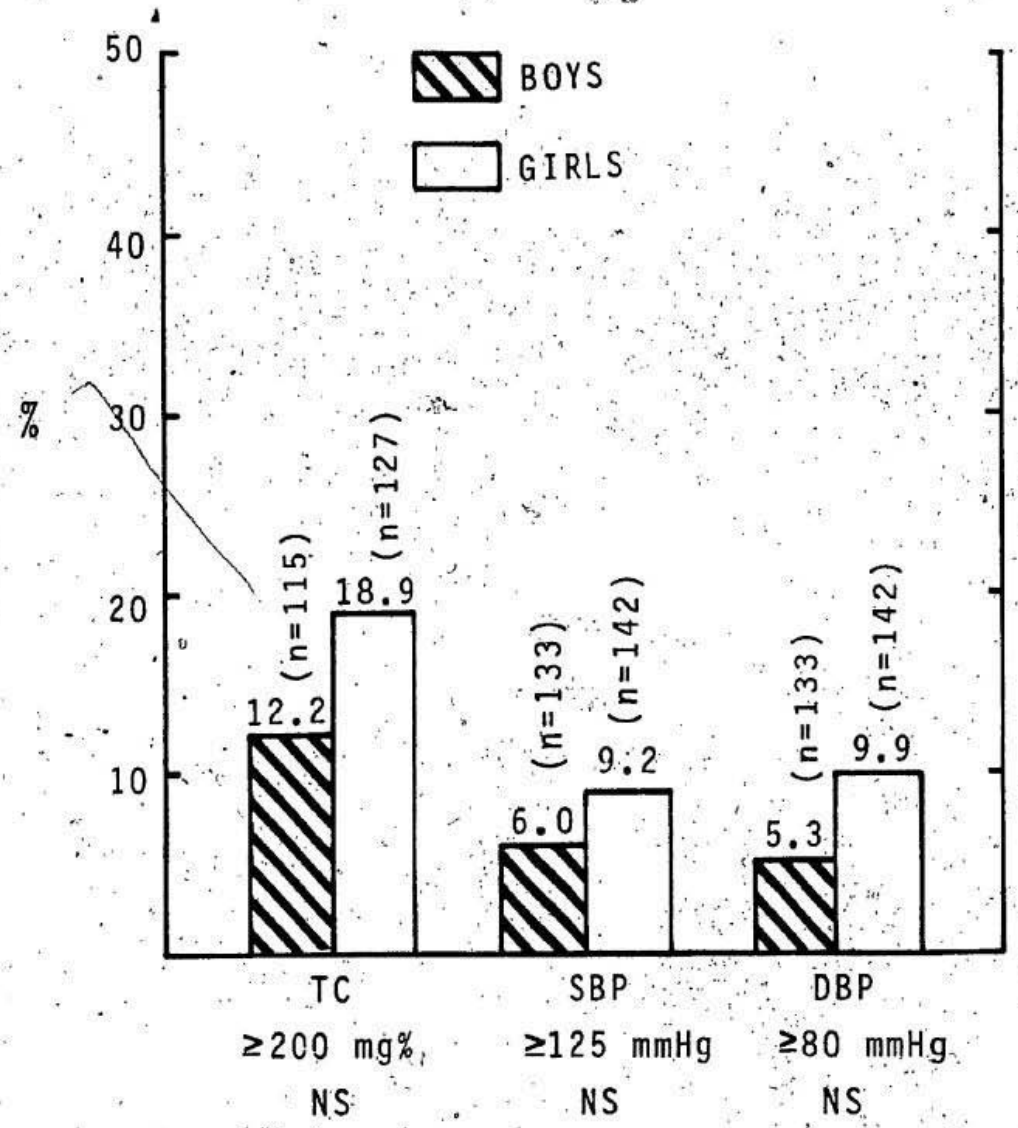
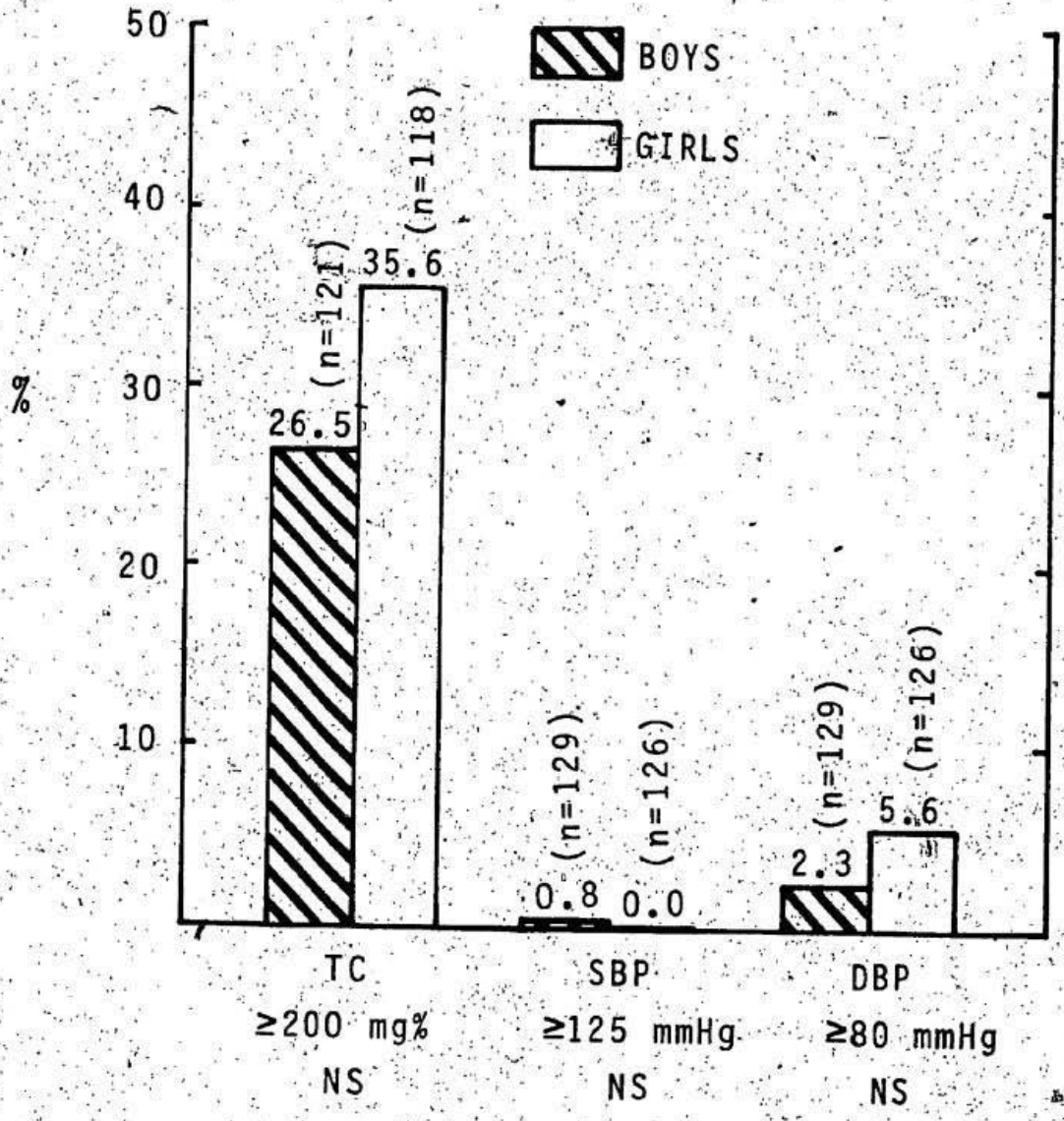

50
40
30
20
10
%
BOYS
GIRLS
12.2
(n=115)
18.9
(n=127)
6.0
(n=133)
9.2
(n=142)
5.3
(n=133)
9.9
(n=142)
TC
≥200 mg%
NS
SBP
≥125 mmHg
NS
DBP
≥80 mmHg
NS

FIGURE 7

Prevalence of elevated primary CHD risk factors for boys and girls aged 8-10 years in the low CHD mortality region


50
40
30
20
10
%
BOYS
GIRLS
26.5
(n=121)
35.6
(n=118)
0.8
(n=129)
0.0
(n=126)
2.3
(n=129)
5.6
(n=126)
TC
≥200 mg%
NS
SBP
≥125 mmHg
NS
DBP
≥80 mmHg
NS

systolic blood pressure (≥ 125 mmHg), whereas none of the girls had such levels. The prevalence of elevated diastolic blood pressure (≥ 80 mmHg) was 3.3% more for girls than boys. This was not a significant difference as indicated in Figure 7.

(iii) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure levels for boys in the 8-10 year age group in high and low CHD mortality regions

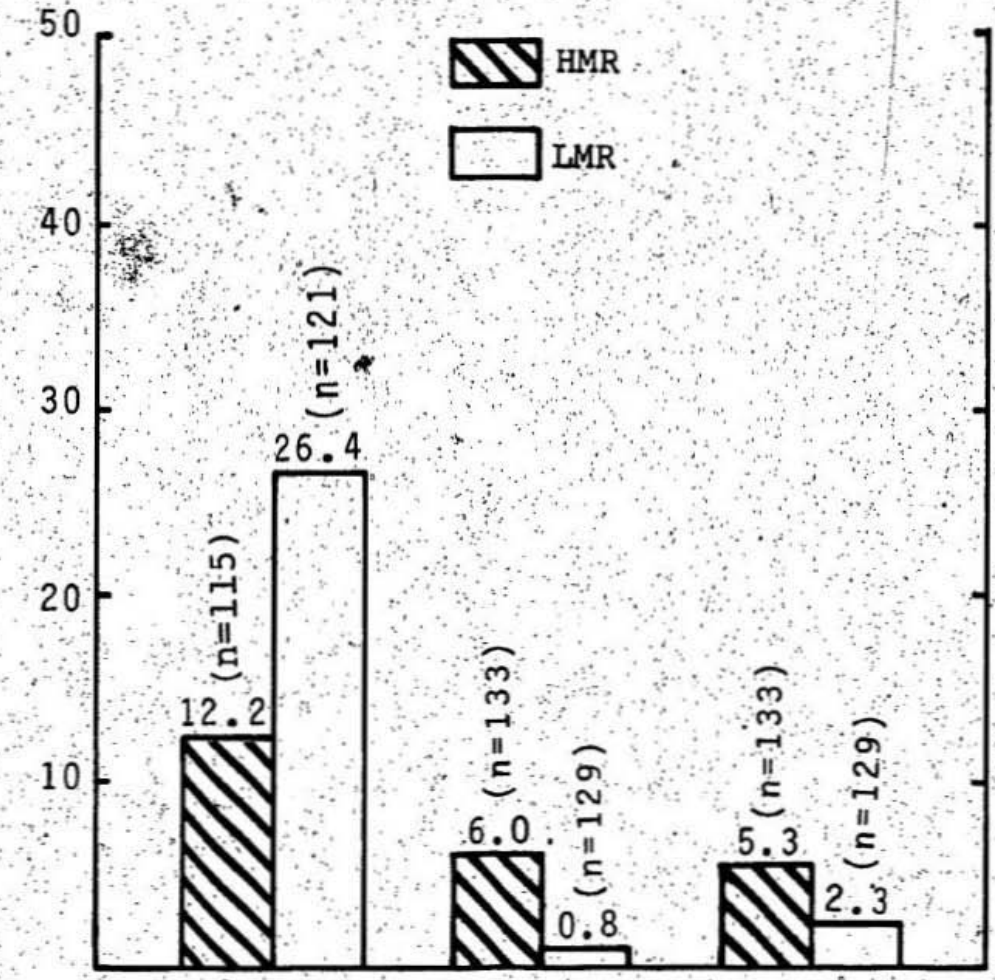
Boys in the low mortality region had a 14.2% higher prevalence of elevated total serum cholesterol (≥ 200 mg%) than boys in the high mortality region. This difference was statistically significant at the 1% level (Figure 8). However, 5.2% more boys in the high than in the low mortality region had elevated systolic blood pressure levels ≥ 125 mmHg. This difference was statistically significant at the 2% level (Figure 8). The prevalence of elevated diastolic blood pressure (≥ 80 mmHg) was 3.0% greater for boys in the high mortality region compared to boys in the low mortality region. This difference, however, was not statistically significant (Figure 8).

(iv) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure levels for girls in the 8-10 year age group in the high and low CHD mortality regions

In the low mortality region, 16.7% more girls had elevated total serum cholesterol ≥ 200 mg% than in the high mortality region. This

FIGURE 8

Prevalence of elevated primary CHD risk factors for boys aged 8-10 years in the high and low CHD mortality regions - HMR and LMR


50
40
30
20
10
%
HMR
LMR
12.2
(n=115)
26.4
(n=121)
6.0
(n=133)
0.8
(n=129)
5.3
(n=133)
2.3
(n=129)
TC
≥200 mg%
p < 0.01
SBP
≥125 mmHg
p < 0.02
DBP
≥80 mmHg
NS

difference was significant at the 1% level (Figure 9). In the high mortality region, 9.2% of girls had systolic blood pressures ≥125 mmHg. No one in the low mortality region had such levels. The difference was statistically significant ($p<0.001$) as indicated in Figure 9 . The prevalence of elevated diastolic blood pressure (≥ 80 mmHg) was 4.3% higher for girls in the high than in the low mortality region. This difference, however, was not statistically significant (Figure 9).

Summary

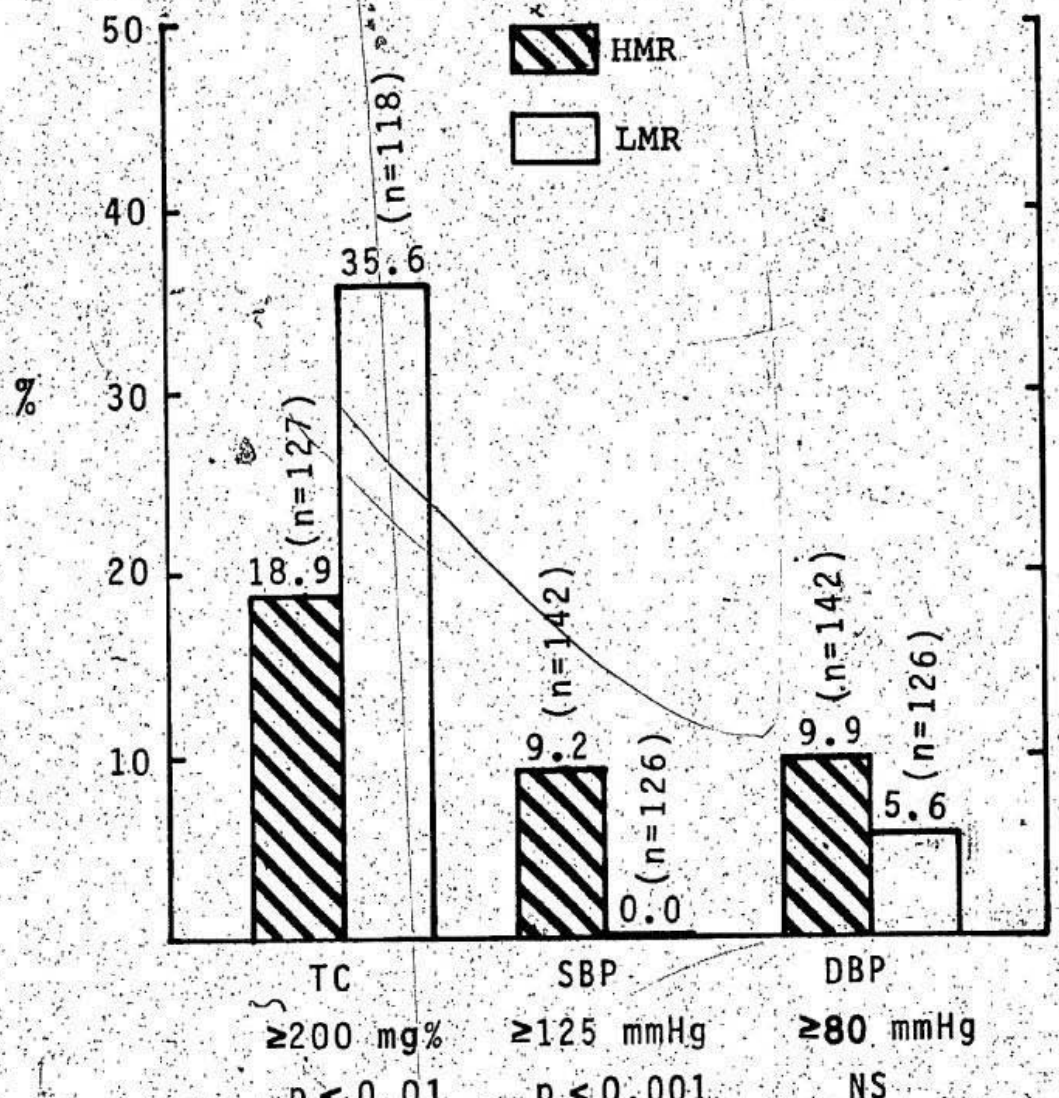
In the high mortality region, girls in this age group had a non-significant but higher prevalence of elevated total serum cholesterol, elevated systolic blood pressure and elevated diastolic (IV) blood pressure than boys. Girls in the low mortality region had a non-significant but higher prevalence of elevated total serum cholesterol and elevated diastolic (IV) blood pressure than boys. Although boys had a higher prevalence of elevated systolic blood pressure than girls in the low mortality region, the difference was not significant.

There was a significantly higher prevalence of elevated total serum cholesterol for boys in the low mortality region compared to those in the high mortality region. Elevated levels of systolic blood pressure were significantly more prevalent for boys in the high mortality region compared to boys in the low mortality region. Although elevated diastolic (IV) blood pressure levels were more



FIGURE 9

Prevalence of elevated primary CHD risk factors for girls aged 8-10 years in the high and low CHD mortality regions - HMR and LMR


50
40
30
20
10
%
HMR
LMR
18.9 (n=127)
35.6 (n=118)
9.2 (n=142)
0.0 (n=126)
9.9 (n=142)
5.6 (n=126)
TC
≥200 mg%
p < 0.01
SBP
≥125 mmHg
p < 0.001
DBP
≥80 mmHg
NS

prevalent for boys in the high than in the low mortality region, the difference was not statistically significant.

The prevalence of elevated total serum cholesterol was significantly higher for girls in the low than in the high mortality region. Elevated systolic blood pressure levels were significantly more prevalent for girls in the high than in the low mortality region. The prevalence of elevated diastolic (IV) blood pressure was higher for girls in the high than in the low mortality region; however, the difference was not statistically significant.

SECTION 11

PREVALENCE OF ELEVATED CHD RISK FACTORS (TOTAL SERUM CHOLESTEROL, SYSTOLIC AND DIASTOLIC (IV) BLOOD PRESSURE AND SMOKING) IN THE 14-16 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure and smoking for boys and girls in the 14-16 year age group in the high CHD mortality region

The prevalence of elevated total serum cholesterol ($\geq$ 200 mg%) was 8.3% higher for girls than boys in the high mortality region. This difference was significant at the 5% level (Figure 10). Elevated systolic blood pressure levels ($\geq$ 140 mmHg) were 10.8% higher for boys than girls in this region. The difference was highly

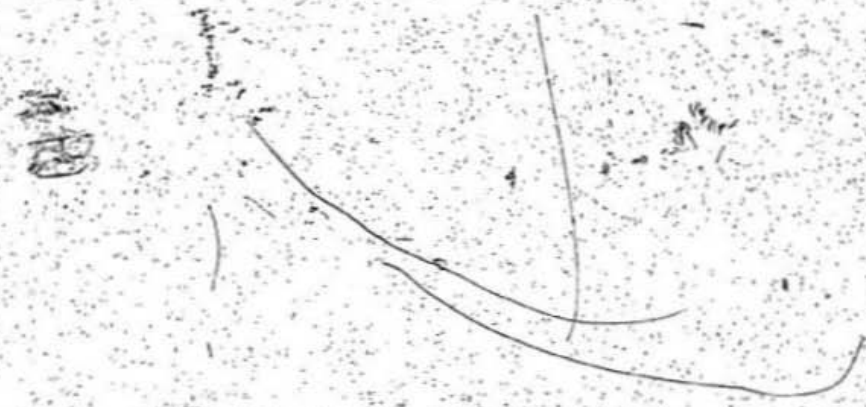
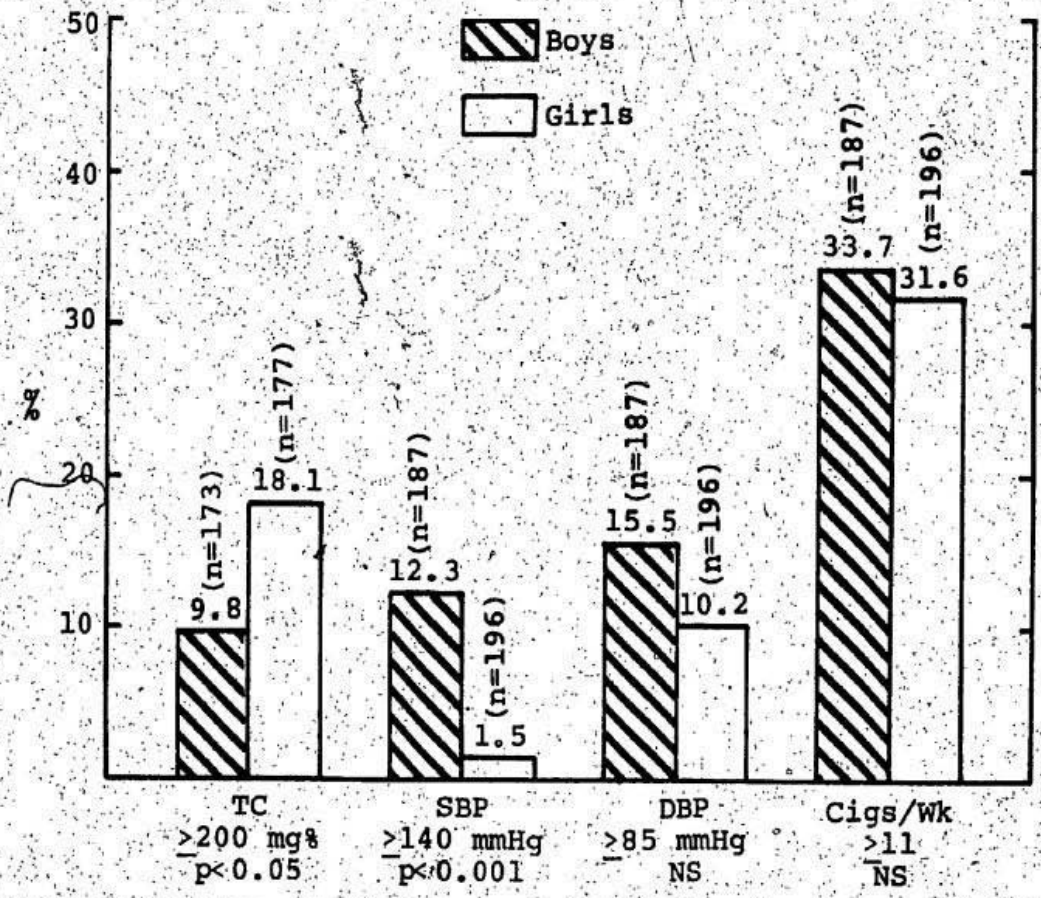


FIGURE 10

Prevalence of elevated primary CHD risk factors for boys and girls aged 14-16 years in the high CHD mortality region


Boys
Girls
%
50
40
30
20
10
9.8 (n=173)
18.1 (n=177)
12.3 (n=187)
1.5 (n=196)
15.5 (n=187)
10.2 (n=196)
33.7 (n=187)
31.6 (n=196)
TC
≥200 mg%
p<0.05
SBP
≥140 mmHg
p<0.001
DBP
≥85 mmHg
NS
Cigs/Wk
≥11
NS

significant ($p<0.001$) (Figure 10).

In this mortality region, 5.3% more boys than girls had elevated diastolic blood pressure levels ($\geq$ 85 mmHg). This was not a significant difference as shown in Figure 10. The prevalence of those smoking $\geq$ 11 cigarettes per week was 2.1% greater for boys than girls in this region. This was not a significant difference (Figure 10).

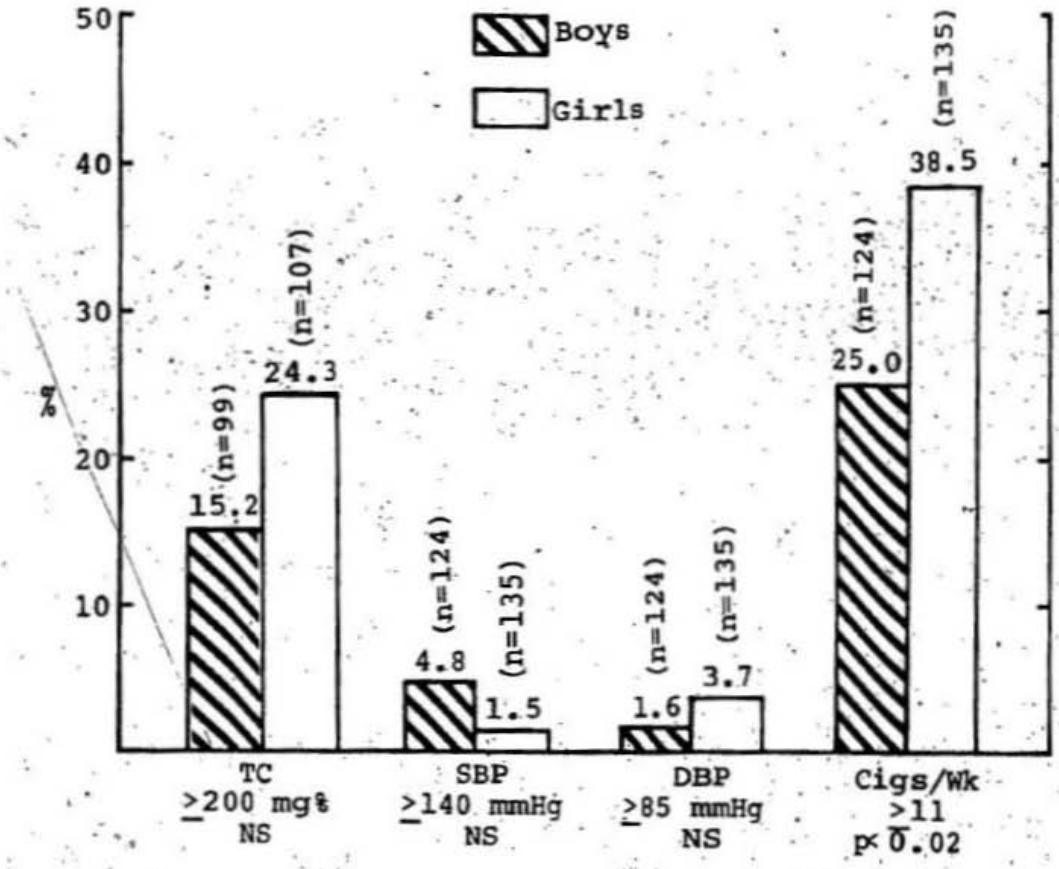
(ii) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure and smoking for boys and girls in the 14-16 year age group in the low mortality region

In the low mortality region, 9.1% more girls than boys had elevated total serum cholesterol levels $\geq$ 200 mg%. This difference was not statistically significant (Figure 11). The prevalence of elevated systolic blood pressure was 3.3% higher for boys than girls in the low mortality region. This difference was not statistically significant (Figure 11).

There were 2.1% more girls than boys who had elevated diastolic blood pressure levels $\geq$ 85 mmHg. The difference was not statistically significant (Figure 11). In this mortality region, 13.5% more girls than boys smoked $\geq$ 11 cigarettes per week. This difference was statistically significant at the 2% level (Figure 11).

FIGURE 11

Prevalence of elevated primary CHD risk factors for boys and girls aged 14-16 years in the low CHD mortality region


Boys
Girls
%
50
40
30
20
10
15.2
(n=99)
24.3
(n=107)
4.8
(n=124)
1.5
(n=135)
1.6
(n=124)
3.7
(n=135)
25.0
(n=124)
38.5
(n=135)
TC
≥200 mg%
NS
SBP
≥140 mmHg
NS
DBP
≥85 mmHg
NS
Cigs/Wk
>11
p< 0.02

(iii) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure and smoking for boys in the 14-16 year age group in the high and low CHD mortality regions

In the low mortality region, 5.4% more boys had elevated total serum cholesterol $\geq$ 200 mg% than in the high mortality region. This was not a significant difference (Figure 12). There were 7.5% more boys in the high than in the low mortality region with elevated systolic blood pressure $\geq$ 140 mmHg. This difference was statistically significant at the 5% level (Figure 12).

The prevalence of elevated diastolic blood pressure levels ($\geq$ 85 mmHg) was 13.9% higher for boys in the high than in the low mortality region. This was a significant difference ($p<0.001$) (Figure 12).

The prevalence of smoking $\geq$ 11 cigarettes per week was 8.7% higher for boys in the high than in the low mortality region. The difference was not significant (Figure 12).

(iv) Prevalence of elevated total serum cholesterol, systolic and diastolic (IV) blood pressure and smoking for girls in the 14-16 year age group in the high and low CHD mortality regions

Elevated serum cholesterol levels $\geq$ 200 mg% were 6.2% more prevalent for girls in the low than in the high mortality region. The difference was not significant (Figure 13). In both regions 1.5% of girls had elevated systolic blood pressure levels ($\geq$ 140 mmHg) (Figure 13).

FIGURE 12

Prevalence of elevated primary CHD risk factors for boys aged 14-16 years in the high and low CHD mortality regions

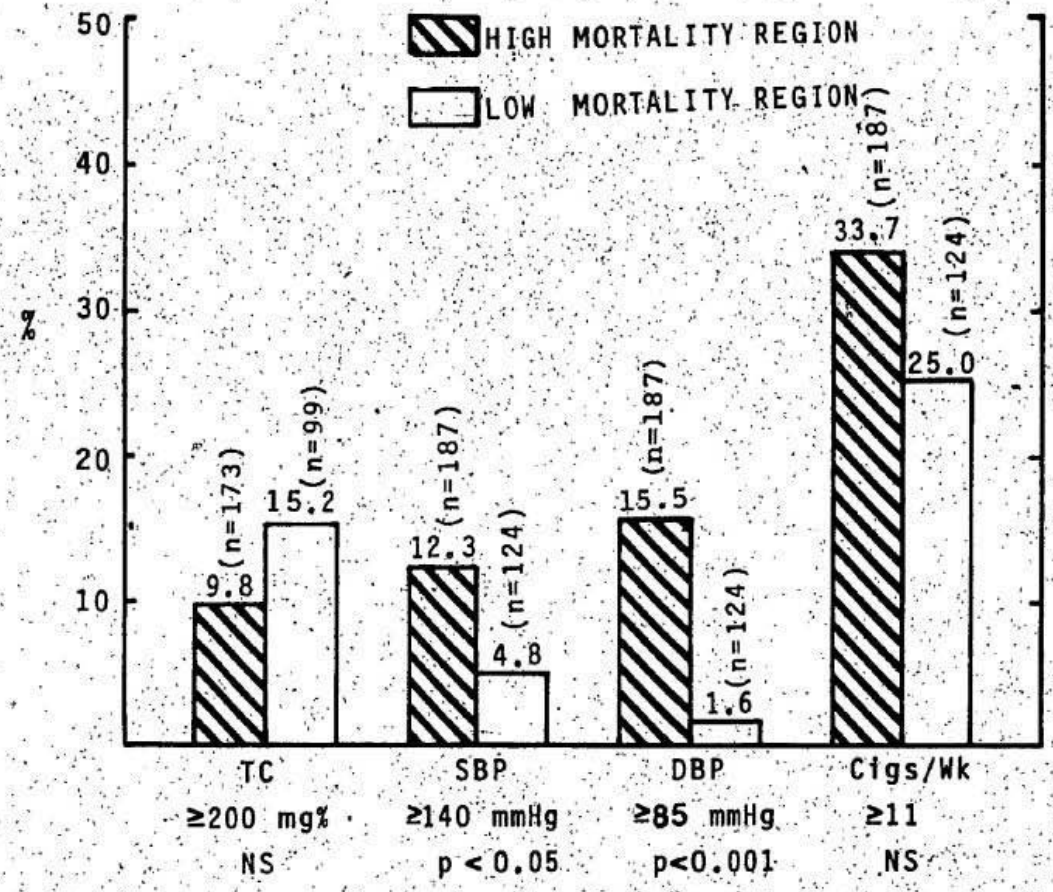
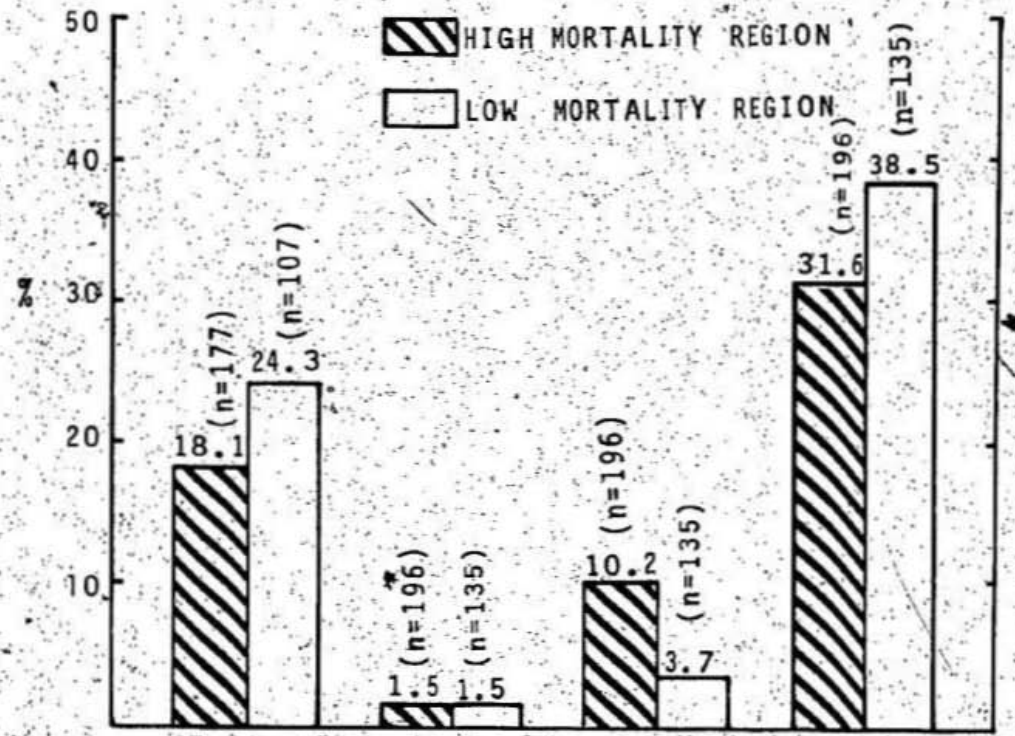

HIGH MORTALITY REGION
LOW MORTALITY REGION
%
50
40
30
20
10
9.8 (n=173)
15.2 (n=99)
12.3 (n=187)
4.8 (n=124)
15.5 (n=187)
1.6 (n=124)
33.7 (n=187)
25.0 (n=124)
TC
≥200 mg%
NS
SBP
≥140 mmHg
p < 0.05
DBP
≥85 mmHg
p<0.001
Cigs/Wk
≥11
NS

FIGURE 13

Prevalence of elevated primary CHD risk factors for girls aged 14-16 years in the high and low CHD mortality regions


HIGH MORTALITY REGION
LOW MORTALITY REGION
50
40
30
20
10
%
18.1 (n=177)
24.3 (n=107)
1.5 (n=196)
1.5 (n=135)
10.2 (n=196)
3.7 (n=135)
31.6 (n=196)
38.5 (n=135)
TC
≥200 mg%
NS
SBP
≥140 mmHg
NS
DBP
≥85 mmHg
p<0.05
Cigs. Wk.
≥11
NS

There were 6.5% more girls in the high than in the low mortality region with elevated diastolic blood pressure levels ($\geq$ 85 mmHg). This difference was statistically significant at the 5% level (Figure 13). The prevalence of smoking $\geq$ 11 cigarettes per week was 6.9% higher for girls in the low than in the high mortality region. This was not a significant difference (Figure 13).

Summary

Girls had a significantly higher prevalence of elevated total serum cholesterol than boys in the high mortality region. The prevalence of elevated systolic and diastolic blood pressure as well as smoking $\geq$ 11 cigarettes per week were higher for boys than girls with elevated systolic blood pressure being significantly higher.

The prevalence of elevated total serum cholesterol and diastolic blood pressure levels was higher for girls than boys in the low mortality region. Boys in this region had a higher prevalence of elevated systolic blood pressure than girls. A significantly higher number of girls than boys in the low mortality region smoked $\geq$ 11 cigarettes per week.

Boys in the high mortality region had a significantly higher prevalence of elevated systolic and diastolic blood pressures. The prevalence of elevated total serum cholesterol was greater, but not significantly, for boys in the low than in the high mortality region. There was an insignificant but higher prevalence of smoking $\geq$ 11

cigarettes per week for boys in the high than in the low mortality region.

Girls in the low, as opposed to the high, mortality region had a higher prevalence which was not significant of elevated total serum cholesterol. The prevalence of elevated systolic blood pressure was the same for girls in both regions, however, those in the high mortality region had significantly higher prevalence of elevated diastolic blood pressure. Girls in the low than in the high mortality region had a higher prevalence of smoking 11 or more cigarettes per week. This difference was not significant.

SECTION 12

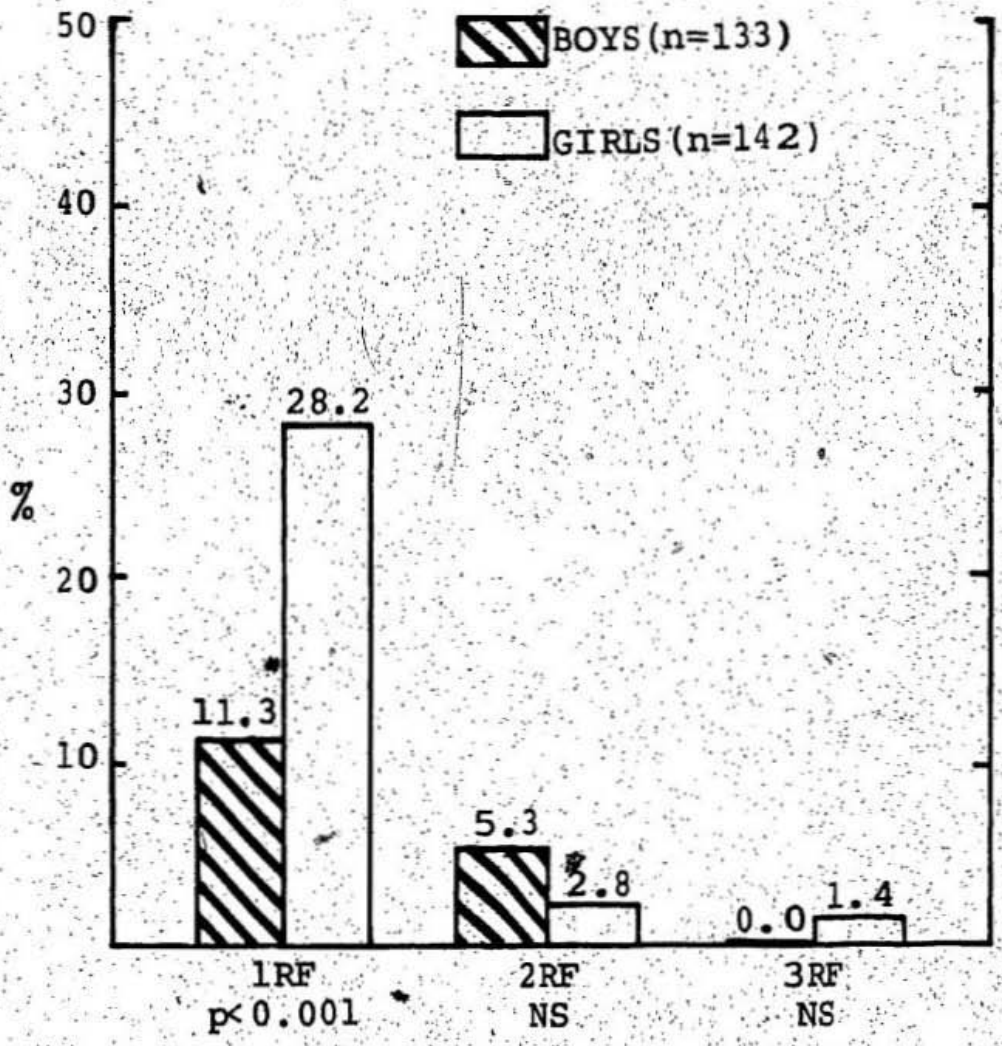
CLUSTERING OF PRIMARY CHD RISK FACTORS IN BOYS AND GIRLS IN THE 8-10 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Clustering of CHD risk factors of boys and girls in the 8-10 year age group in the high CHD mortality region

As shown in Figure 14, 11.3% of boys as compared to 28.2% of girls in the high mortality region had a single risk factor. This was a highly significant difference ($p<0.001$). This figure shows also that 5.3% of boys had two risk factors compared to 2.8% of girls. The difference was not statistically significant. In this region, 1.4% of girls but none of the boys had three risk factors.

FIGURE 14

Clustering of elevated primary CHD risk factors (RF) for boys and girls aged 8-10 years in high CHD mortality region


BOYS (n=133)
GIRLS (n=142)
50
40
30
20
10
%
28.2
11.3
5.3
2.8
0.0
1.4
1RF
p< 0.001
2RF
NS
3RF
NS

This difference was not significant (Figure 14).

(ii) Clustering of CHD risk factors of boys and girls in the 8-10 year age group in the low CHD mortality region.

There were 14.1% more girls than boys in the low mortality region with one risk factor. This was a significant difference ($p<0.02$) (Figure 15). In this region, 1.6% boys compared to 0.8% of girls had two risk factors present. This difference was not statistically significant (Figure 15). No one of either sex had three risk factors.

(iii) Clustering of CHD risk factors for boys in the 8-10 year age group in the high and low CHD mortality regions

There were 24.0% of boys in the low compared to 11.3% in the high mortality region who had one risk factor present. The difference was significant at the 1% level (Figure 16). In the high mortality region, 5.3% of boys compared to 1.6% in the low mortality region had two risk factors present. This difference was not significant (Figure 16). No one had three CHD risk factors present.

(iv) Clustering of CHD risk factors for girls in the 8-10 year age group in the high and low CHD mortality regions

There were 9.9% more girls in the low as compared to the high mortality region with one risk factor. The difference was not

FIGURE 15

Clustering of elevated primary CHD risk factors (RF) for boys and girls aged 8-10 years in the low CHD mortality region

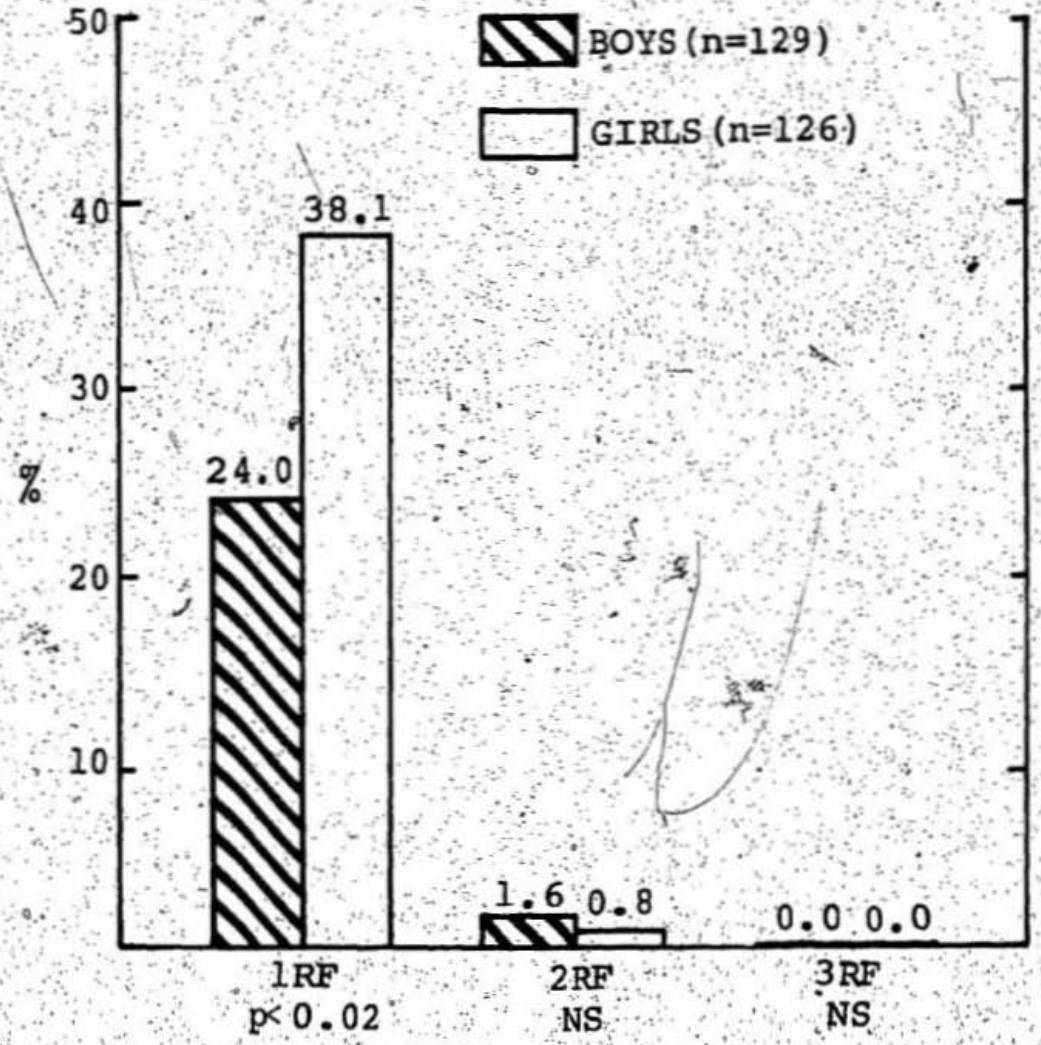
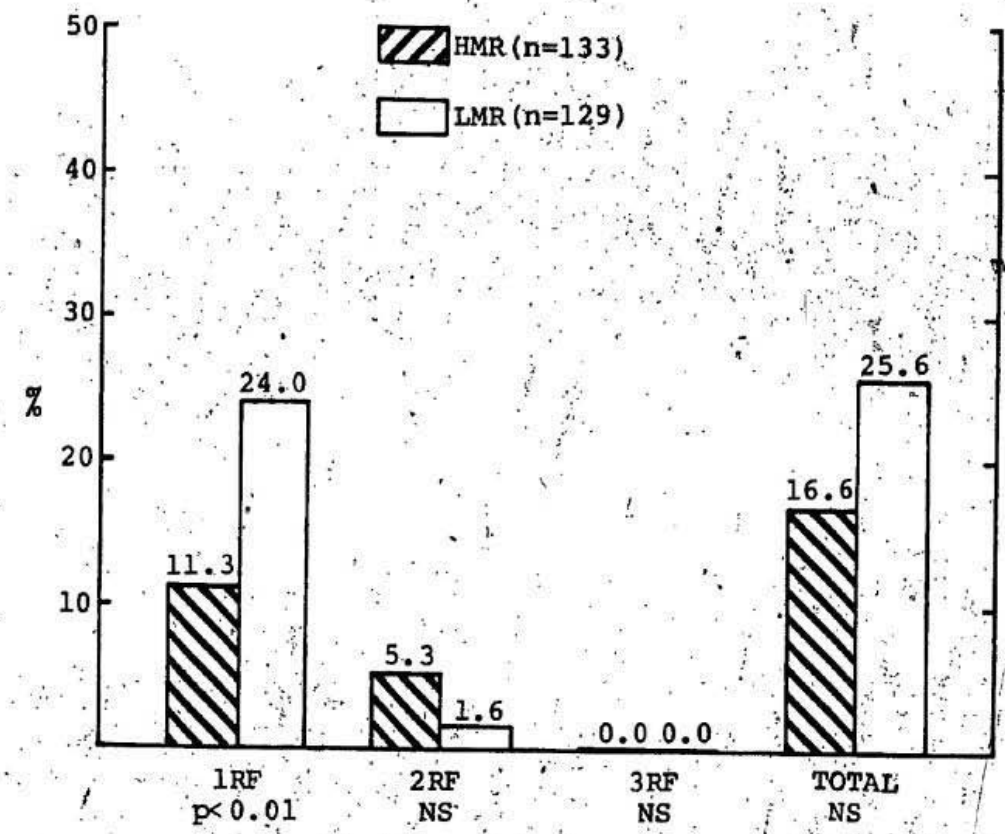

BOYS (n=129)
GIRLS (n=126)
50
40
30
20
10
%
38.1
24.0
1.6
0.8
0.0
0.0
1RF
p< 0.02
2RF
NS
3RF
NS

FIGURE 16

Clustering of elevated primary CHD risk factors (RF) for boys aged 8-10 years in the high and low CHD mortality regions - HMR and LMR


HMR (n=133)
LMR (n=129)
50
40
30
20
10
%
24.0
11.3
5.3
1.6
0.0 0.0
16.6
25.6
1RF
p< 0.01
2RF
NS
3RF
NS
TOTAL
NS

significant (Figure 17). Two risk factors were present in 2.8% girls in the high compared to 0.8% in the low mortality region. This difference was not significant (Figure 17). In the high mortality region, 1.4% of girls had three risk factors present. No one had three risk factors in the low mortality region as shown in Figure 17.

Summary

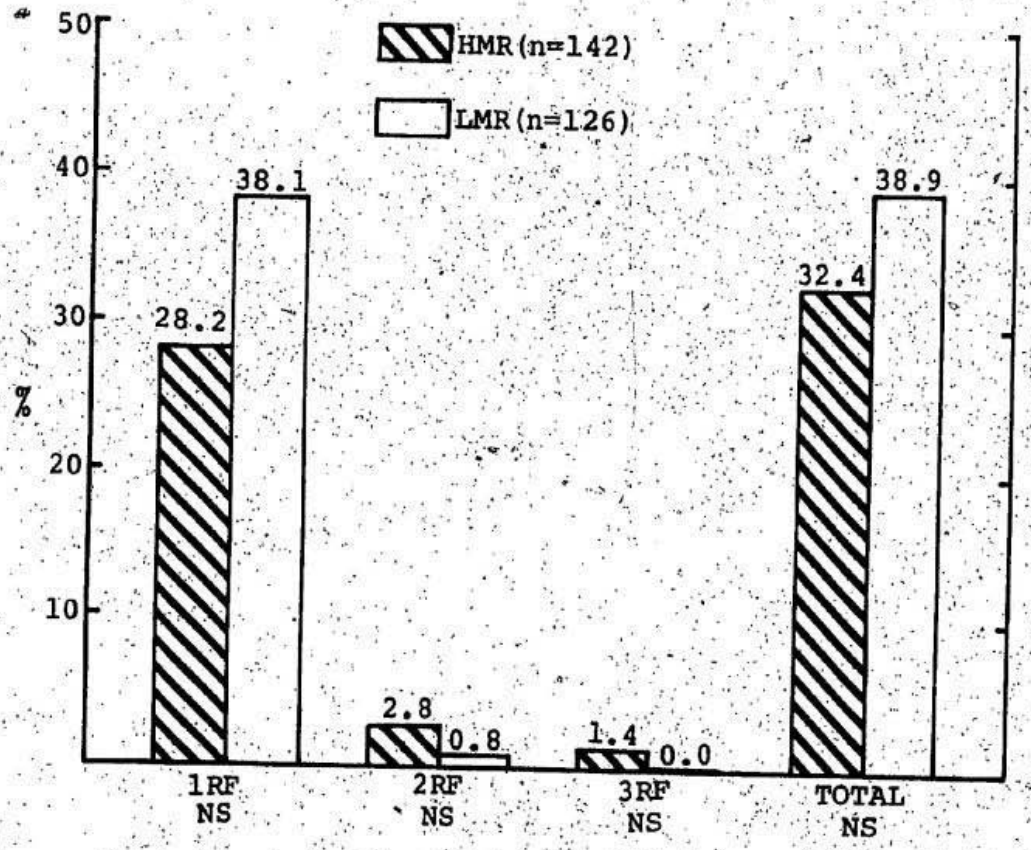
In the high mortality region, there was a higher prevalence of girls than boys with one and three risk factors present. However, more boys than girls in this region had two risk factors. In the low mortality region, a significantly higher percentage of girls than boys had a single risk factor present. Twice as many boys as compared to girls in this region had two risk factors. None of the boys or girls had three risk factors.

The percentage of boys with a single risk factor was higher in the low than in the high mortality region. There was a higher percentage of boys in the high than in the low mortality region with two risk factors. No one in either region had three risk factors.

There was a higher percentage of girls in the low compared to the high mortality region with a single risk factor. More girls in the high than in the low mortality region had two and three risk factors respectively.

FIGURE 17

Clustering of elevated primary CHD risk factors (RF) for girls aged 8-10 years in the high and low CHD mortality regions - HMR and LMR


50
40
30
20
10
%
HMR (n=142)
LMR (n=126)
28.2
38.1
2.8
0.8
1.4
0.0
32.4
38.9
1RF NS
2RF NS
3RF NS
TOTAL NS

SECTION 13

CLUSTERING OF PRIMARY CHD RISK FACTORS IN BOYS AND GIRLS IN THE 14-16 YEAR AGE GROUP IN THE HIGH AND LOW CHD MORTALITY REGIONS

(i) Clustering of CHD risk factors of boys and girls in the 14-16 year age group in the high CHD mortality region

Almost the same percentage of boys (44.9%) and girls (44.4%) in the high mortality region had one risk factor present (Figure 18). In this region, 8.6% of boys had two risk factors compared to 6.6% of girls. The difference of 2.0% was not statistically significant. The number of boys and girls in the high mortality region with three risk factors was 1.6% and 0.5% respectively. No one of either sex had four risk factors.

(ii) Clustering of CHD risk factors of boys and girls in the 14-16 year age group in the low CHD mortality region

In the low mortality region, 10.5% more girls than boys had one risk factor present. The difference was not statistically significant (Figure 19). Also, almost twice as many girls as boys in this region had two risk factors present. The difference was not significant (Figure 19). There was no one of either sex in this mortality region with three or four risk factors.

FIGURE 18

Clustering of elevated primary CHD risk factors (RF) for boys and girls aged 14-16 years in the high CHD mortality region

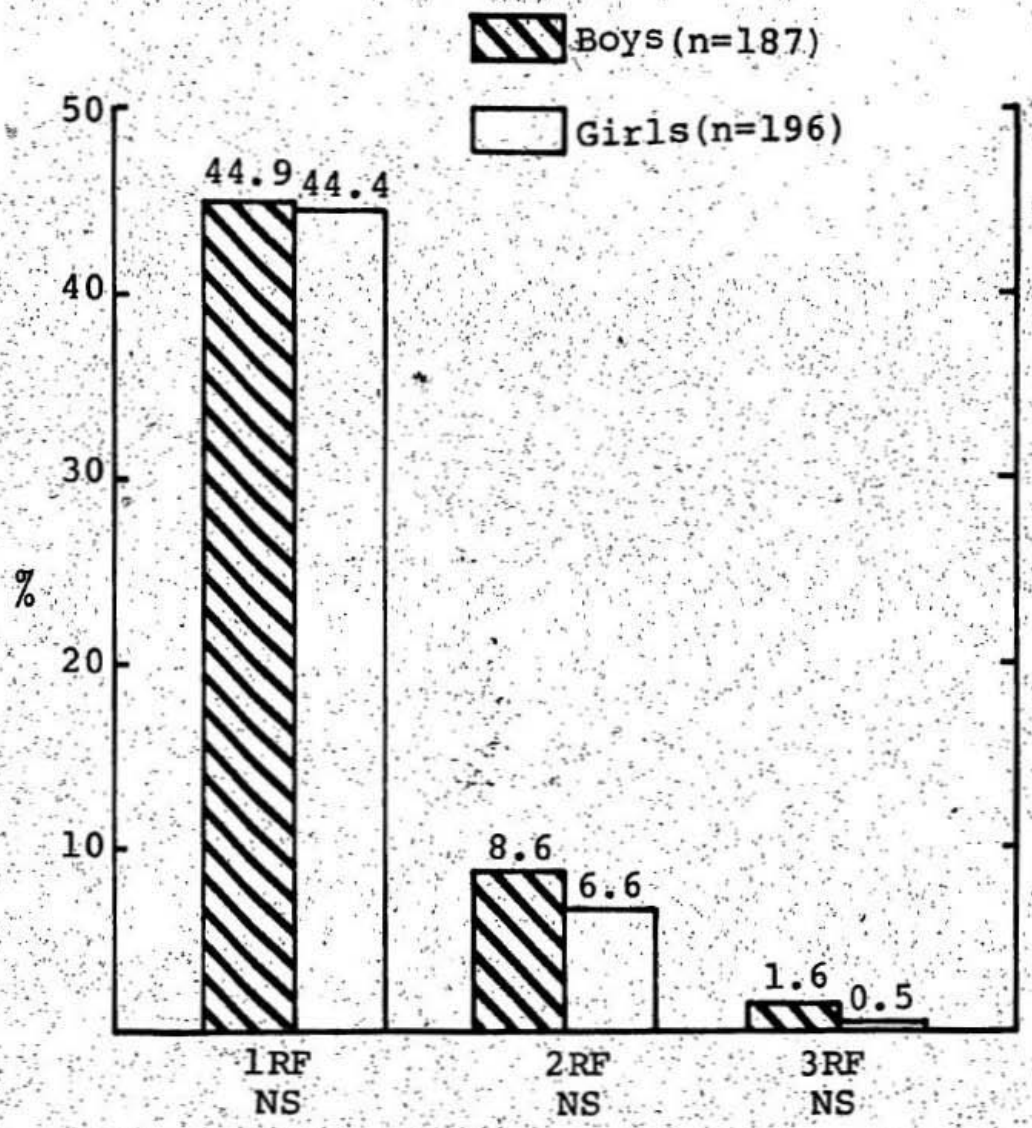
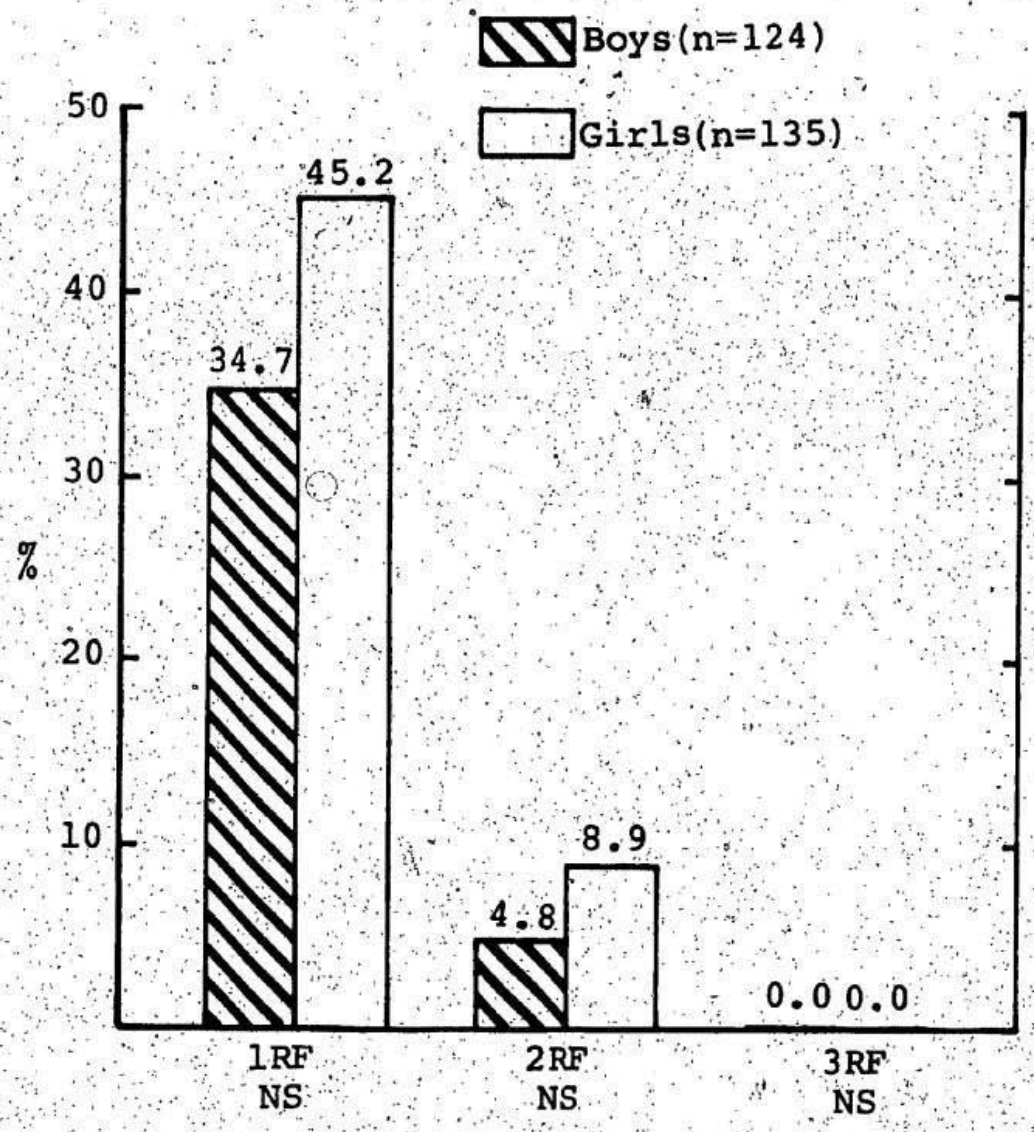

Boys(n=187)
Girls(n=196)
50
40
30
%
20
10
44.9
44.4
8.6
6.6
1.6
0.5
1RF
NS
2RF
NS
3RF
NS

FIGURE 19

Clustering of elevated primary CHD risk factors (RF) for boys and girls aged 14-16 years in the low CHD mortality region


Boys(n=124)
Girls(n=135)
50
40
30
20
10
%
34.7
45.2
4.8
8.9
0.0
0.0
1RF
NS
2RF
NS
3RF
NS

(iii) Clustering of CHD risk factors of boys in the 14-16 year age group in high and low CHD mortality regions

There were 10.2% more boys in the high than in the low mortality region with one risk factor. The difference was not significant (Figure 20). The number of boys with two risk factors present were 8.6% and 4.8% in the high and low mortality regions respectively. The difference was not significant. Three risk factors were present in boys (1.6%) in the high mortality region only. No one in either region had four risk factors present.

(iv) Clustering of CHD risk factors of girls in the 14-16 year age group in the high and low CHD mortality regions

The percentage of girls in the high and low mortality regions with one risk factor was 44.4% and 45.2% respectively. The difference was not significant (Figure 21). In the low mortality region, 2.3% more girls had two risk factors present than girls in the high mortality region. The difference was not significant (Figure 21). Only girls (0.5%) in the high mortality region had three risk factors present. There was no one in either region with four risk factors.

Summary

One risk factor was present in the same number of boys and girls in the high mortality region. A higher percentage of boys than girls in this mortality region had two and three risk factors

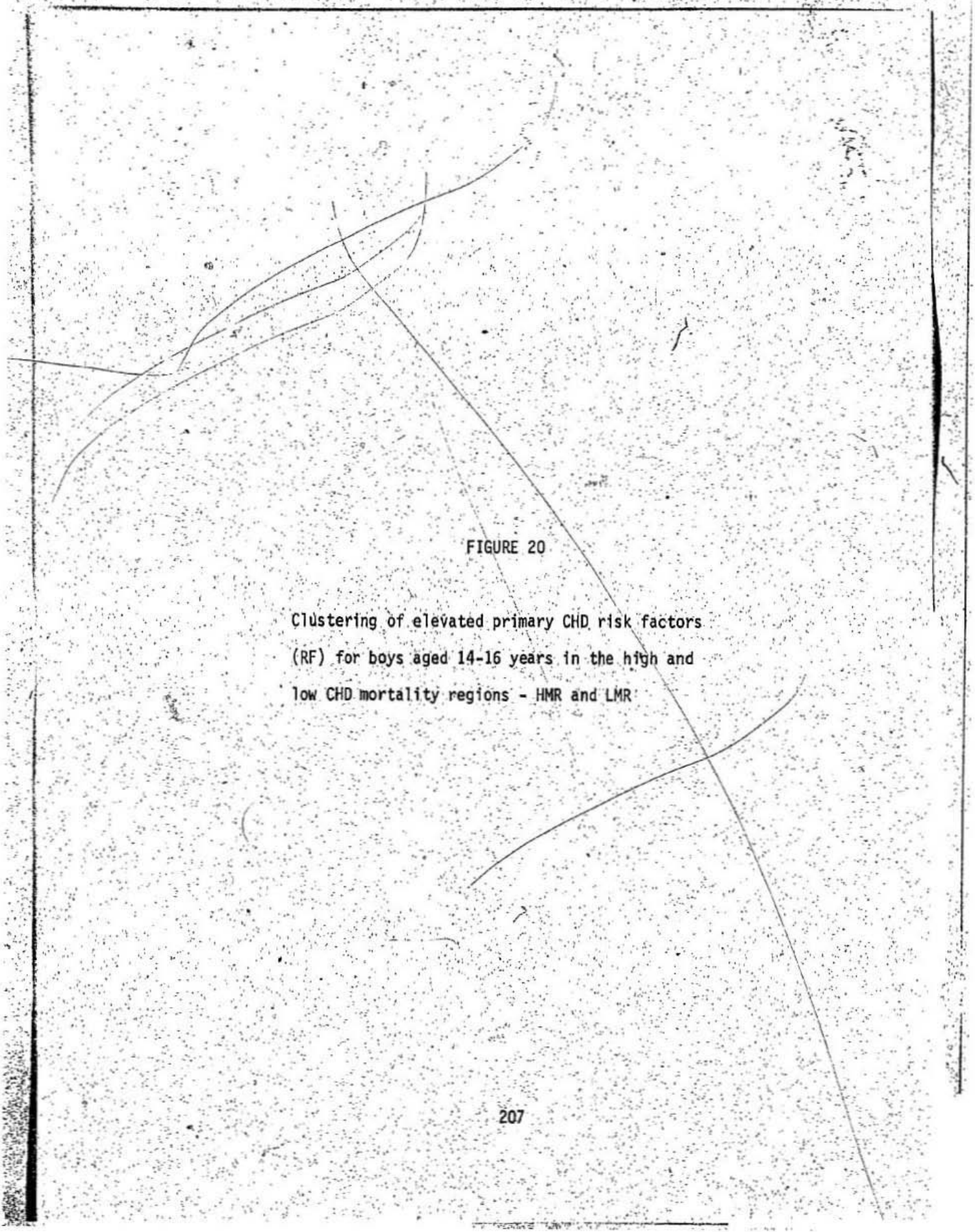


FIGURE 20

Clustering of elevated primary CHD risk factors (RF) for boys aged 14-16 years in the high and low CHD mortality regions - HMR and LMR

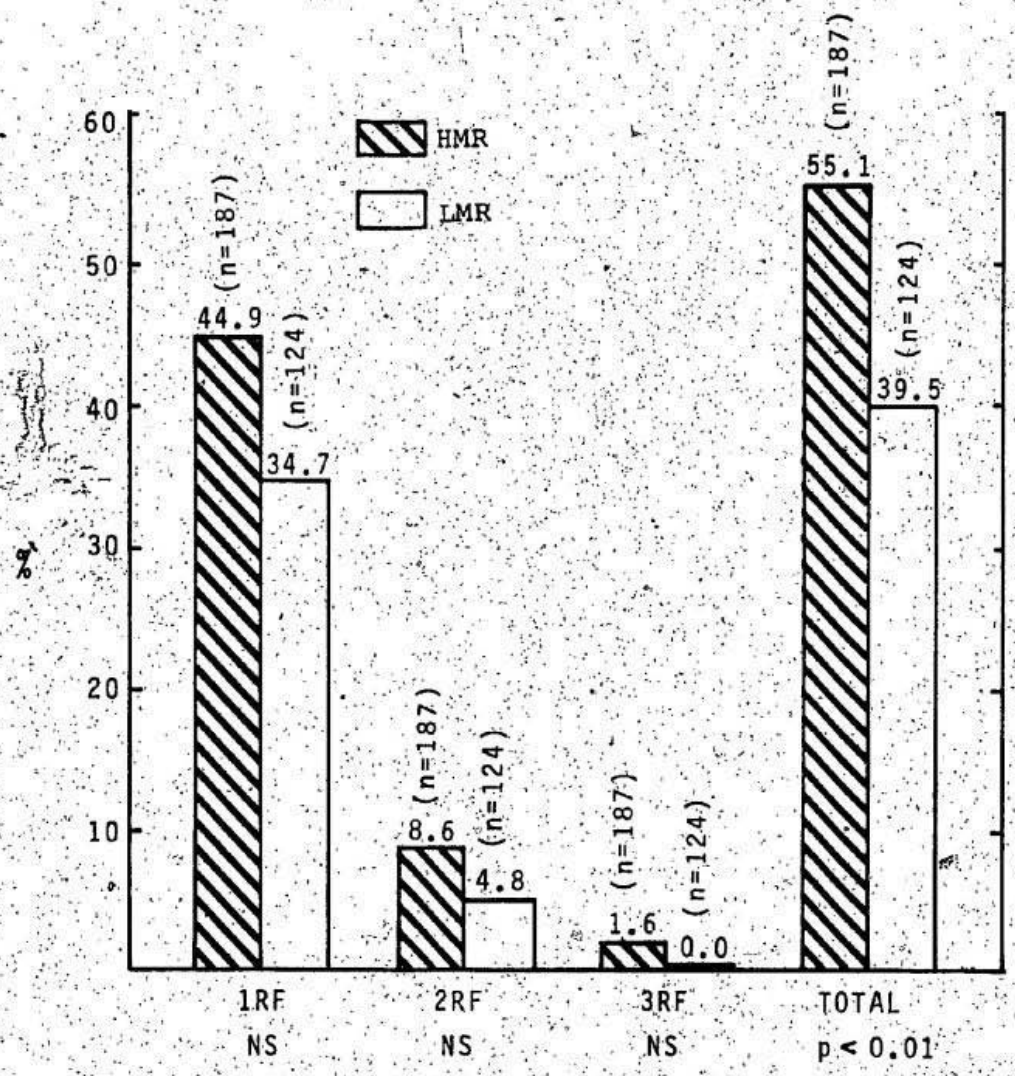
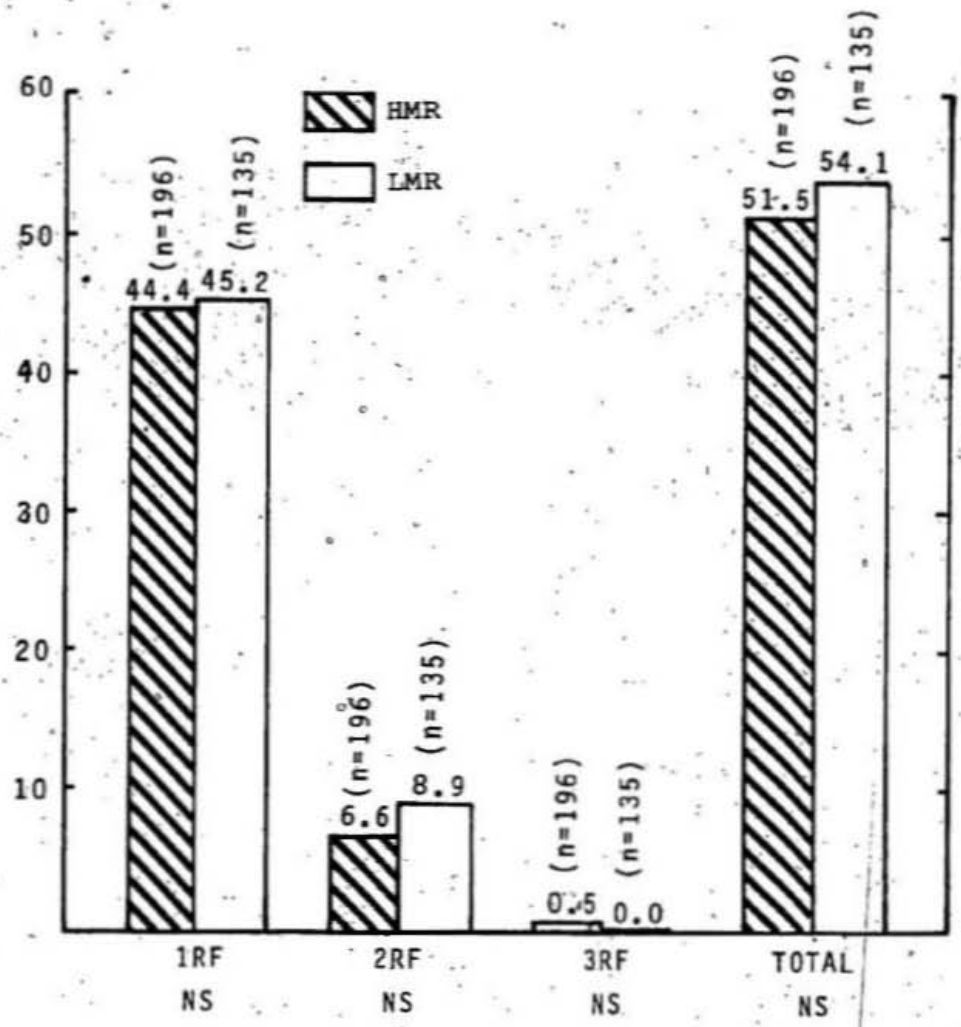

HMR
LMR
%
60
50
40
30
20
10
44.9 (n=187)
34.7 (n=124)
8.6 (n=187)
4.8 (n=124)
1.6 (n=187)
0.0 (n=124)
55.1 (n=187)
39.5 (n=124)
1RF
NS
2RF
NS
3RF
NS
TOTAL
p < 0.01

FIGURE 21

Clustering of elevated primary CHD risk factors (RF) for girls aged 14-16 years in the high and low CHD mortality regions - HMR and LMR


60
50
40
30
20
10
%
HMR
LMR
(n=196)
(n=135)
44.4
45.2
6.6
8.9
0.5
0.0
51.5
54.1
1RF
NS
2RF
NS
3RF
NS
TOTAL
NS

respectively. In the low mortality region, a higher percentage of girls than boys had one and two risk factors present. No one of either sex had three or four risk factors.

A greater percentage of boys in the high than in the low mortality region had one, two and three risk factors present. The percentage of girls with one risk factor was almost identical in the two mortality regions. There was a slightly higher percentage with two risk factors in the low mortality region as compared to the high mortality region. Only one person in the high mortality region had three risk factors present.

SECTION 14

FREQUENCY OF HYPERLIPOPROTEINEMIA AMONG CHILDREN IN THE COMBINED AGE GROUPS 8-10 AND 14-16 IN THE HIGH AND LOW CHD MORTALITY REGIONS

Table 31 shows that only 0.7% of the children in the high mortality region exceeded the beta hyperlioprotein cholesterol level of greater than 170 mg% and could, therefore, be considered hyperbetalipoproteinemic (Type 2A). In the low mortality region, the prevalence of this Type 2A disorder was 1.3%. The difference was not significant.

The same table shows that 3.9% of the children in the high mortality region showed hyper-pre-betalipoproteinemia (pre-beta-

lipoprotein cholesterol levels greater than 25 mg%) without associated hyperbetalipoproteinemia (Type 4). In the low mortality region, the prevalence of hyper-pre-betalipoproteinemia was 1.1%. The difference between the two mortality regions was significant at the 1% level.

Table 31 shows also that the combined hyperlipoproteinemia, without both beta- and pre-betalipoprotein cholesterol levels exceeding the cut-off point as shown in the table, was seen in only 0.2% of the children in the high mortality region. No one in the low mortality region had combined hyperlipoproteinemia.

Summary

The frequency of hyperlipoproteinemia in this population was calculated based on the currently recommended upper normal limits by Fredrickson et al 1967. The results showed that 0.7% of children in the high mortality region exceeded the upper beta-lipoprotein cholesterol level of 170 mg% and could be considered hyperbetalipoproteinemic (Type 2A). In the low mortality region, 1.3% of children exceeded this level and could, thus, be considered hyperbetalipoproteinemic (Type 2A). On the other hand, 3.9% of children in the high mortality region and 1.1% in the low mortality region showed hyper-pre-betalipoproteinemia (Type 4) (pre-betalipoprotein cholesterol level > 25 mg%) without associated hyperbetalipoproteinemia. Combined hyperlipoproteinemia, with both beta- and pre-betalipoprotein cholesterol levels exceeding the cut-off points as

TABLE 31

FREQUENCY OF HYPERLIPOPROTEINEMIA USING INDICATED CUT-OFF VALUES AMONG CHILDREN IN THE COMBINED 8-10 AND 14-16 YEAR AGE GROUP IN HIGH AND LOW CORONARY HEART DISEASE MORTALITY REGIONS

	FREQUENCY		
LIPOPROTEIN ELEVATED	HIGH MORTALITY REGION	LOW MORTALITY REGION	p
LDL-C > 170 mg%	4/592 = 0.7%	6/474 = 1.3%	N.S.
VLDL-C > 25 mg%	23/592 = 3.9%	5/474 = 1.1%	<0.01
BOTH { LDL-C > 170 mg% / VLDL-C > 25 mg%	1/592 = 0.2%		

indicated in the table, was seen only in the high mortality region where there were 0.2% of the children with such levels.

The significant epidemiological findings of this study are discussed in the following chapter.

CHAPTER FIVE

DISCUSSION

Introduction

This is the first epidemiological study, at least in North America, to the author's knowledge, which has investigated the distribution of CHD risk factor variables in children living in two regions of an island characterised by high and low adult CHD mortality rates. The study was undertaken with the following research questions in mind:

1. Are there significant differences in the distribution of risk factor variables between children 8-10 years of age (prepubertal) and those 14-16 years of age (pubertal) within the high and low CHD mortality regions?
2. Are there significant regional differences in the distribution of these variables between children of comparable age groups in the two regions? If there are significant differences in risk factor variables, are the differences the same for both sex and age groups and in which region are they significantly different?
3. Which of these risk factor variables are most important in distinguishing between children in the high and low CHD mortality regions?
4. Is there a group of children in one or both mortality regions who may be at increased risk of developing premature CHD based upon their risk factor profile?

The answers to these questions are necessary in order to design an effective primary prevention CHD program in children. Such a program would be focused on those children who have been identified as having the greatest likelihood of developing CHD. This would be the most effective strategy because it is during childhood that it is easiest to change deleterious lifestyle habits.

1. Study design and methods

One of the major objectives of this study was to determine whether there is a group of children who have a greater likelihood of developing CHD later in life based on their risk factor levels. It is important, therefore, to note the following design and methods which serve to enhance the validity of the results which will be discussed below.

The study compared the distribution of risk factor variables of children in strictly defined narrow age groups of a considerably homogenous population. It is, therefore, unlikely that these factors could have confounded the results in any meaningful way.

Children in the two regions who participated in the study were from schools that were selected randomly. Furthermore, the response rate of children from both regions was higher than most school-based epidemiological studies (Orchard et al 1980; McGandy, 1971; Lauser and Schutt, 1978; Morrison et al 1979; Berenson et al 1979), and the number of children that responded met the prerequisite of a minimum sample size of 96 (Chapter 3, page 63) for each

age and sex group for all continuous variables measured.

Analyses of serum uric acid, serum glucose and lipid samples from the two mortality regions were done in the same biochemical laboratories, using the same biochemical methods. The coefficient of variation of measurement error for blind duplicate samples of lipids, uric acid and glucose did not exceed 4.8%. This compares very favorably with the coefficient of variation of measurement error reported by other researchers for these variables (Viikari et al 1982; Frerichs et al 1976).

Blood pressures were measured with an automatic blood pressure recorder in both regions under similar conditions. The use of this instrument for measuring blood pressure removes several biases inherent in taking blood pressures with a mercury sphygmomanometer as mentioned in Chapter 2, page 42. Blood pressure measurements were not made on the day venipuncture was done since it has been shown (United States Vital and Health Statistics, Series 11, 1977) that if these two procedures are done on the same day, blood pressure levels tend to be higher due to anxiety of the children.

The same examination procedure was followed in all schools in both regions. Another important factor in this study was that identical cut off values were used to designate the presence of elevated primary CHD risk factor variables in children of the same age group belonging to the two mortality regions.

In essence, the results of this study were not biased by using a different design and methods in the collection and analysis of data from the two mortality regions.

2. Significant epidemiological findings

In my view, the most noteworthy findings of this study were that boys and girls in the high CHD mortality region had significantly higher systolic and diastolic blood pressures than their counterparts in the low mortality region. These were the only risk factor variables that were significantly higher in the 8-10 year age group that continued to be significantly higher in the 14-16 year age group in the high mortality region as shown in tables 25 and 26 on pages 159 and 160. No other studied risk factor displayed the same unequivocal trend in both age groups and both regions.

One can infer from these findings that blood pressure levels of children in the high mortality region remain in the same "track" since these variables continued to be identifiers in the older ages. This finding was also supported by discriminant function analysis which showed that both systolic and diastolic blood pressures were important discriminating variables between children 14-16 years of age belonging to the high and low mortality regions. The magnitude and statistical significance ($p<0.0001$) of the standardised discriminant function coefficients for these two risk factor variables and their percentage contribution indicate that these findings were not due to chance.

Additional evidence indicating the importance of these findings was obtained from the results of the prevalence data. These results showed a significantly higher prevalence of elevated systolic blood pressure for both boys and girls 8-10 years of age in the high mortality region (Figure 8 page 174 and Figure 9 page 177), and a significantly higher prevalence of this variable for boys in the older age group in the high mortality region.

In the younger age group, both boys and girls in the high mortality region also had a higher prevalence of elevated diastolic blood pressure, although these differences were not statistically significant. In the older age group, the prevalence of elevated diastolic blood pressure was significantly higher for both boys and girls in the high mortality region (Figure 12 page 186 and Figure 13 page 188).

The accumulated evidence point to systolic and diastolic blood pressures as important factors in the development of CHD in children from the high CHD mortality region. It is further of interest that the body mass (weight/height^2) of both boys and girls in the older age group in the high mortality region was significantly greater than their counterparts in the low mortality region. The importance of body mass in distinguishing between older children from the two regions was shown in Table 30 , page 166. It can be seen that this was a very important risk factor variable by virtue of its ranking and standardised discriminant function coefficient.

One of the possible effects that increased body mass may be having on these children is probably related to the finding that children in the high mortality region have a significantly higher prevalence of Type IV hyperprebetalipoproteinemia (Table 31 , page 213). The latter disorder is associated with overweight. It has been stated that 40% of children with this disorder are obese and the opinion is that obesity might facilitate the childhood manifestation of this lipid abnormality (Glueck et al 1977). The relationship between this disorder and the incidence of CHD is not clear. One study (Carlson and Bottiger, 1972) did show a positive relationship, another did not (Brunzell et al 1976).

Further evidence that children in the high mortality region have a higher exposure to primary CHD risk factors comes from results of the clustering of risk factors. These results showed that a significantly higher number of boys in the high mortality region had one, two and three elevated primary CHD risk factors (Figure 20, page 208).

The impact of multiple CHD risk factors was demonstrated clearly by the results of the National Cooperative Pooling Project which investigated the effects of combinations of CHD risk factors upon individuals. It was found that when only one risk factor was present as compared to none, there was an association with a substantial increase in the probability of a major event over the next decade. This means an increase in risk of almost 100% for the fatal endpoints including total mortality (Inter Society Commission for Heart Disease

Resources, 1970).

When a person had any two or all three present, susceptibility to overt CHD and fatal disease was substantially higher, reaching levels four or five times greater than for the group with none of the three risk factors present. It would be appropriate, therefore, to consider these children with elevated levels of multiple CHD risk factors to be at high risk for developing premature CHD.

3. Public Health implications of findings

(i) Elevated blood pressure

The public health implications of the finding that children in the high mortality region have significantly higher blood pressures are quite substantial. In every major epidemiological study (Galyean, 1978; Stamler, 1971; Keys, 1970; Medalie et al 1973; Robertson et al 1977; Rosenman et al 1976; Wilhelmsen et al 1973; Ostrander and Lamphiears, 1976; Kagan et al 1974; Kannel, 1976; Dolder and Oliver, 1975; Robertson et al 1977; Marmot et al 1975; Hollander, 1976) elevation of blood pressure, both systolic and diastolic, were correlated strongly with the risk of CHD. The point has been made often that risk of CHD associated with blood pressure rises smoothly as blood pressure increases and that there is not a clear cut level at which risk becomes manifest.

The International Atherosclerosis Pooling Project (McGill, 1968) as well as the study by Davis and Klainer (1940) showed that hypertensives had greater numbers and severity of raised atherosclerotic

lesions in their coronary arteries than normotensives. The hypothesis put forward to explain the accelerated development of CHD as seen in those with high blood pressure is that the distending pressure of the blood within the vessel exerts a shearing force that damages the intima making the intima more permeable to lipid deposits (Glagov, 1972).

The significance of elevated blood pressures in children should not be ignored. In the Evans County, Georgia Study, Heyden et al (1969) found on initial evaluation, using a single blood pressure measurement, that 11% of 435 adolescents were hypertensive (diastolic $\geq$ 90 mmHg). Seven years later, they examined 30 of these 435 patients from the hypertensive group and found 11 of them to have persistent hypertension, with 6 of them having some form of vascular complication in early adulthood including 2 deaths. Many studies (Zinner et al 1975; Buck, 1973; Beaglehole, 1977) have demonstrated a "tracking" effect of blood pressure in children.

(ii) Obesity

The possible implications of the finding that boys and girls in the high mortality region were significantly more obese can be derived from the results of the Manitoba Study (Rabkin et al 1977). This was a prospective study with 26 years of follow-up that showed a positive correlation between body weight and CHD after confounding factors, such as age and blood pressure, were accounted for. The study demonstrated the strongest relationship for men under 40 and

obesity was significantly associated with myocardial infarction, sudden death and suspected myocardial infarction but not angina pectoris. It was concluded that obesity was a definite risk factor in younger men.

The Los Angeles Heart Study (Chapman et al 1971) supported this finding. It documented after a 15-year follow-up, using discriminant function analysis, that weight was a risk factor for myocardial infarction and sudden death, but not angina pectoris in men less than 40 years of age. Reduction in body weight is known to reduce the level of other risk factors (Kannel, 1976; Gordon and Kannel, 1973; Ashley and Kannel, 1974).

Obesity increases the venous return and the preload of the left ventricle. This may become an important factor in persons with essential hypertension in whom the left ventricle is already burdened by an increased afterload. This probably explains why increased left ventricle diastolic dimensions and diffused myocardial hypertrophy have been reported in obese persons (Woodard et al 1978; Smith, 1928).

(iii) Obesity and blood pressure

As mentioned above, this study found a significantly increased level of obesity (weight/height^2) and blood pressure in children from the high CHD mortality region. The importance of this finding can be appreciated by discussing the findings of other studies, as they relate to these two risk factor variables. It was shown that

in younger adults, blood pressure and body mass were highly correlated (Chiang et al 1969; Epstein et al 1965). Similar results were obtained for young populations in Evans County (Johnson et al 1975), Muscatine (Lauer et al 1975) and Bogalusa (Voors et al 1976) and by Londe and Goldring (1972), Levy et al (1946) and Court et al (1974).

Oberman et al (1967), Paffenbarger et al (1968) and Abraham et al (1971) suggested that obesity and elevated blood pressure in adolescence are positively associated with the development of hypertension in adulthood. In their review entitled "Overweight and Hypertension", Chiang et al (1969) mentioned 14 studies in which weight reduction through diet was accompanied by a decrease in blood pressure levels.

Thus, there is substantial evidence which indicate that obesity and elevated blood pressure are highly correlated and, if observed in the young, as was found in this study, they are likely to result in hypertension in adulthood.

4. Possible explanations of findings

The obvious question which need to be addressed is "Why do children in the high mortality region have significantly higher systolic and diastolic blood pressures and, in the older ages, are more obese than their counterparts in the low mortality region?". In order to answer this question, three factors may be relevant. They are: (i) dietary salt, (ii) other nutritional factors and

(iii) physical activity.

(i) Dietary salt

As early as 1904 two young French physicians, Ambard and Beaujard, postulated that salt restriction will lower blood pressure. This idea was followed up with clinical applications by Allen (1925) in the United States, and salt restriction was widely advocated after the Second World War with the development of the Kempner (1948) rice-fruit diet.

Page (1976) has stated that the prevalence of hypertension is not related to the fact whether the individual eats meat or is a vegetarian. The crucial factor is the amount of salt eaten. Rauh et al (1981), in one of the few studies that investigated the effects of lowering salt intake in hypertensive children, found that a 10 mEq sodium diet significantly lowered both systolic and diastolic blood pressures.

The association between high salt intake and hypertension in genetically susceptible individuals has been discussed by Dahl (1972) and a direct correlation between average sodium intake and the prevalence of hypertension has been documented (Knudsen and Dahl, 1966). These researchers provided evidence to show that Eskimos who have a low salt intake have almost no hypertension. However, groups such as the Northern Japanese with high salt diets have above 40% prevalence of hypertension.

It is quite conceivable that these children living in coastal communities in the high mortality region consume larger amounts of

salt in their diet than those in the low mortality region. Fodor, Abbott and Rusted (1973) found that in coastland communities of Newfoundland the salt intake was around 150 mEq per day compared to 120 mEq per day for inland communities.

It is believed that the population residing in the communities in the high mortality region from which these children were selected consume a high amount of salt because of traditional customs developed prior to refrigeration. Salt was used to preserve fish which was the main source of food. The daily diet also consisted of salted beef and pork. As a result, the acquired appetite for unusual quantities of salt in the food was transmitted from one generation to another through common cooking and eating habits in the family. In the low mortality region, the traditional food has been fresh meat obtained from hunting; as a result, there has been less salting to preserve foods.

(ii) Other nutritional factors

Traditionally agriculture has been successful only in the low mortality region. This part of the island has better quality soil for growing vegetables and other fresh produce as well as better climatic conditions which permit a longer growing season. These conditions permit the production of home grown green vegetables and fruits which are the main source of dietary potassium and would lead one to speculate that the dietary intake of potassium would be higher in the low than in the high mortality region. Certainly the

more favourable agricultural and climatic conditions in the low mortality region should make this possible.

Another more interesting difference between the two regions is their geological formations. Sedimentary rocks are present only in the low mortality region and only in this part of the Island does the soil have any significant amount of magnesium and very likely potassium. Fodor, Pfeiffer and Papezik (1973) have documented that the drinking water is about thirty times harder in the low than in the high mortality region. They suggested that this may be a reason for the difference in total and cardiovascular mortality between the two regions.

One cannot help but hypothesise that the significantly lower blood pressures of children in the low mortality region may be due to an increased intake of potassium obtained from a higher consumption of green vegetables and fruits together with a quality of drinking water that contains higher concentrations of minerals. These thoughts probably fit in well with the dictum of Addison (1928) who with the sharp insight of an extremely perceptive internist suggested that "high salt and low potash environment" could be a reason for high blood pressure.

The postulated role of sodium (salt) and potassium in the pathogenesis of hypertension is worth noting.

(a) Salt and hypertension

The physiological relationship of salt on blood pressure has been investigated by Guyton et al (1974), Tobian (1972), Borst (1963), Ledingham (1953) and Freis (1976). Guyton et al (1974) have proposed a theory which suggests that essential hypertension develops from a complex hemeostatic response to chronic increase in extracellular fluid (ECF). The salt which is consumed is distributed mainly in the ECF, excess quantities are excreted by the kidneys. However, if the ECF expands venous filling pressure increases which results in a number of hemodynamic changes leading to increased blood pressure in order to increase urinary output and salt excretion.

Those persons who consume unusual amounts of salt have increased ECF relative to those with low salt intake. When a person who consumes too much salt is put on a salt restricted diet, the ECF drops and blood pressure falls. Freis (1976) believes that a reduction of salt in the diet to below 2 g per day would result in the prevention of essential hypertension; however, Tobian (1978) disagrees by pointing out that there are many people who eat a lot of salt and still have normal blood pressures.

This paradox can be explained by the finding that there are individuals who are salt sensitive as opposed to others who are salt insensitive (Fujita, 1980; Kawaski, 1978). These researchers observed patients first on a 9 mEq sodium diet and then increased the amount to 250 mEq per day. They identified the two groups of

patients noted above. The salt-sensitive patients gained more weight, retained more sodium and had a greater increase in cardiac output on a high salt diet. Those patients who were salt insensitive had significant decreases in plasma renin activity, plasma aldosterone concentration and urinary prostagladin levels.

(b) Potassium and hypertension

Although the role of potassium in the epidemiology of essential hypertension is not clear, recent studies by Langford and Watson (1971; 1973; 1975) as well as Blaustein (1977) have shown that the prevalence of hypertension is inversely related to calcium and potassium intake. The results of other studies (Langford and Watson, 1975; Grim et al 1970) suggest that the ratio of sodium to potassium excretion might be associated with blood pressure levels.

The diet of our Western society is high in sodium but low in potassium (Meneely and Battarbee, 1976). Parfrey et al (1981) showed recently that a diet low in sodium and high in potassium reduced blood pressure in patients with mild hypertension. In addition, they found that the sons of hypertensive patients responded to increased dietary potassium with a significant fall in blood pressure while the sons of normotensive patients responded with a slight, but non-significant, rise in pressure.

They suggested that a genetically determined susceptibility to the pressor effect of low dietary potassium is important in the early stages of essential hypertension and that the increased

susceptibility to dietary sodium develops later. They also proposed that the potassium effect is mediated by the autonomic nervous system. This probably supports the view that autonomic nervous overactivity is important in the early stages of the disease (Ledingham, 1971; Brown et al 1976). It has been suggested by Priddle (1962) that potassium acts as a neutraliser on the hypertensiogenic effect of sodium.

If these views regarding the relationship between potassium and blood pressure are correct, then they will most likely explain why children living in a region which probably has a much higher dietary potassium intake because of the availability of fresh green vegetables and fruits and a better quality drinking water have significantly lower blood pressure.

Our own interest in the role of sodium and potassium as explanatory factors of the observed blood pressure differences has prompted us to embark on the study of a small sample of children from both regions to determine whether their excretion of sodium and potassium are significantly different.

(iii) Physical activity

An explanation as to why children in the high mortality region are significantly more obese than those in the low mortality region can be found in several etiological factors of childhood obesity. One such factor is based on the thermodynamic principle that obesity develops when there is an imbalance between caloric input

and output. This can take place by increasing caloric input or decreasing physical activity. At the present time, there is no evidence to indicate which is more important in the primary development of obesity - physical inactivity or overeating.

There is no available data which show that children in the high mortality region eat more than children in the opposite mortality region. However, the author observed while in the communities that children in the low mortality region engaged in more physical activities than their counterparts in the high mortality region. One reason is that there are more sport facilities available in this region which were leftover from the former American base.

Heredity and social factors, for example, income levels, have also been implicated in the development of obesity (Mayer, 1975; Garn and Clark, 1976; Bruch, 1939). The extent, if any, of the influence these factors may have on the children in the high mortality region are not known. Other suggested reasons for increased obesity in children from the high mortality regions are: (1) higher birth weight - obese children have been shown to have higher birth weights (Helve et al 1971; Shukla et al 1972; Sveger et al 1975; Borjeson, 1962), (2) bottle feeding versus breast feeding - it has been found that bottle-fed infants gain weight more rapidly than breast-fed infants (Taitz, 1971).

Other factors known to be associated with obesity such as hypothalmic tumours, sexual immaturity (Frohlich, 1901) and endocrine causes such as hypothyroidism, Cushing's syndrome and

Turner's syndrome (Court, 1977; Jones, 1972) are most unlikely explanatory factors for the increased obesity observed in this group of children in the high mortality region.

It is quite feasible, however, that children in this region, apart from being less physically active, consume a greater quantity of refined carbohydrates, as part of their daily diet.

5. Need for a prevention program in childhood

One of the primary objectives of this study was to determine whether there was a group of children in the mortality region who may be at high risk for the development of premature CHD. The results have shown that the children in the high mortality region are at increased risk for developing CHD in later life based upon the finding that they have significantly higher systolic and diastolic blood pressure levels as well as obesity than their counterparts in the low mortality region of the Province. Since these risk factor variables have been shown to be strongly related to the development of coronary heart disease in adults, it is probably very necessary to institute a program that will reach these children in order to modify the influence of these risk factors.

While we do not have hard evidence to show that longevity can be extended, or morbid events reduced by controlling risk factor levels at an early age or in healthy populations, there is considerable evidence to support the benefits to be had by reducing

risk factor levels from prevention trials which have been just concluded.

One such program was the Hypertension Detection Follow-up Program which showed that it was possible to screen thousands of individuals in their homes and evaluate them medically and enrol them into a program whose main objective was to reduce blood pressure levels. This program also showed that there were significant benefits to be had from the reduction of blood pressure.

The Chicago Coronary Prevention Evaluation Program also showed that it was possible to normalise blood pressures through nutritional hygienic methods, that is through moderate weight reduction, improved cardiopulmonary fitness through regular, frequent, moderate rhythmic exercise without use of drugs (Stamler et al 1980). These results would indicate the possibility of achieving primary prevention of hypertension by such methods on large numbers of individuals.

It is of interest to note the comments of some researchers about the implications of leaving children with elevated blood pressures to grow up without any risk factor modification. Holman et al (1958) are of the opinion that if today's children were to grow up like their parents, 20-30% of them will have hypertension as adults. Ninety percent will develop significant atherosclerotic lesions, and it was further stated by Gordon and Devine (1966) that over 50% will die from hypertension and atherosclerosis.

One of the vehicles that can be used to impart primary prevention strategies is education. This can be done by emphasising the role of health education in schools and public education through the mass media and community groups. It is probably necessary that consistent changes in the desireable age groups be accomplished and this could be done by focusing our attention in changing behaviour through behaviour modification techniques.

There is no doubt that there are great potential benefits to be had in comprehensive programs directed towards improving cardiovascular health in the young age group. By making this information available to children through the schools, one can provide them with the knowledge about coronary heart disease followed by the development of an attitude for healthy behaviour, since there is no assurance children will develop lifestyles that will enhance the cardiovascular system based upon the knowledge they have obtained (Kelman, 1961).

In addition to learning new facts about coronary heart disease, its consequences and the associated risk factors and developing new attitudes in regard to more healthy living patterns, children must be taught new physical skills. They must be taught how to jog, to run, to walk briskly and to maintain conditioning.

The more important aspects that should be included in any health education program are the following:

(i) Diet

(a) It should be explained that salt should not be added to food at the table. The processed foods which we consume have more than the required physiological salt requirements of 0.4-0.8 gram per day (Weinsier, 1976; Grollman, 1961). The salt shaker should, therefore, be kept out of reach.

(b) The benefits of balanced nutrition should be stressed. The harmful effects of foods with high refined carbohydrates and fat content, such as french fries, cookies, chocolate, milk shakes and particularly soft drinks, which contain a great amount of sugar and sodium should be mentioned.

(c) The relationship between "poor" diet and CHD should be explained, particularly in relation to obesity. The role of saturated animal fats in the diet should be discussed.

(ii) Weight control

(a) The need to maintain proper weight

(b) How this can be achieved

(iii) Exercise

(a) Exercise should be encouraged

(iv) Smoking

(a) This habit should be discouraged by making children aware of its long-term effects

The final chapter contains a summary and the conclusions arrived at in this study.

CHAPTER SIX

SUMMARY AND CONCLUSIONS

Summary

A cross-sectional epidemiological study was carried out to investigate the distribution of CHD risk factors in children 8-10 and 14-16 years of age. These children resided in two regions of Newfoundland, one characterised by high and the other by low total and standardised CHD mortality rates.

In addition to the study of the distribution of risk factors in these children, the prevalence of elevated systolic and diastolic blood pressures, elevated total cholesterol and smoking habits in the older age group were compared in both geographical regions. The clustering of these elevated CHD risk factors in each child was determined, as well as, the frequency of hyperlipoproteinemia in the two mortality regions.

All children attended schools that were randomly chosen from two communities in each mortality region. The study was conducted in two phases. During the first phase, height, weight, systolic and diastolic blood pressures were measured. In addition, an EKG was recorded and a questionnaire administered.

Of the number of children in the high mortality region who were eligible to participate in the study, 275 (87%) aged 8-10 years and 383 (89%) aged 14-16 years responded positively. In the low mortality region, 255 (94%) children aged 8-10 years and 259 (89%) children aged 14-16 years participated from among those who were eligible.

In the second phase of the study, two 10 ml, 10-hour fasting blood samples were taken. Only children who participated in the first phase of the study were eligible to give blood. The sera were analysed for total cholesterol, HDL cholesterol, triglyceride, uric acid and glucose. In the high mortality region, 242 (88%) children in the 8-10 and 350 (91%) children in the 14-16 year age groups participated in this phase of the study. Blood samples were taken from 240 (94%) children aged 8-10 years and 201 (78%) children aged 14-16 years in the low mortality region.

The following significant results were considered to be of epidemiological importance:

1. Boys and girls, 8-10 and 14-16 years of age, in the high CHD mortality region had significantly higher mean systolic and diastolic blood pressures than their counterparts in the low CHD mortality region.
2. Body mass index (weight/height^2) was significantly greater for boys and girls in the 14-16 year age group in the high than in the low CHD mortality region. This risk factor was also identified as a strong discriminant between this age group in the two mortality regions.
3. The prevalence of elevated levels of systolic blood pressure was significantly higher for boys and girls 8-10 years of age and for boys 14-16 years of age in the high CHD mortality region.

4. Boys and girls in the 14-16 year age group in the high CHD mortality region had a significantly higher prevalence of elevated diastolic blood pressure.
5. The total number of boys 14-16 years of age who had one or clusters of two and three primary CHD risk factors simultaneously was significantly higher in the high mortality region.
6. The prevalence of Type IV hyperprebetalipoproteinemia was significantly higher in children living in the high CHD mortality region.

Conclusions

In a region of Newfoundland, which is known to have the highest adult mortality rate for CHD, we were able to document that children at the young age of 8-10 years and continuing into adolescence (14-16) have significantly higher levels of systolic and diastolic blood pressures. The children in the older age group in the high mortality region were also significantly more obese. Furthermore, this increased obesity may be related to the additional finding that the prevalence of Type IV hyperprebetalipoproteinemia is significantly higher in children living in the high mortality region.

It would appear, based on these findings, that children in the high CHD mortality region may have a higher risk of developing premature CHD in adulthood. It would be prudent, therefore, to

establish a program, directed at children in the region, that would reduce this risk. Such a program should focus on health education aimed at improving nutrition, weight control, physical activity and preventing the onset of smoking.

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Appendix A

Letter to Parent/Guardian

January, 1980

Dear Parent/Guardian:

We are requesting your consent to have your child participate in a health survey to be carried out at the school he/she is attending.

The purpose of this health survey is to help determine which children are most likely to develop heart disease when they grow older. In order for us to do this, we would like to measure your child's blood pressure, height, weight, record a heart tracing, and administer a questionnaire.

At a later date, we would like to take a small 10-hour fasting blood sample for measuring the fat level in his/her blood. This will be done by a qualified person from the hospital in your area. We will provide a breakfast in school for your child after the fasting blood samples have been taken. During the next month or so, we will also be contacting you personally to obtain information on your family's health.

If you agree or disagree to have your child participate in this health survey, please place an X in the appropriate box on the Consent Form, sign it and have your child return it to the school principal.

We would like to point out that your school board and school principal have granted us permission to carry out this health survey in their school.

Thank you for your cooperation.

Sincerely yours,

Appendix B

Consent Form.

CONSENT FORM

My child/children will have his/her blood pressure and an electrocardiogram taken, as well as measurements of height, weight and answer questions in response to a questionnaire. At a later date a small 10-hour fasting blood sample will be taken by a qualified person from the hospital in the community.

I agree ☐ disagree ☐ to have my child/children participate in this health survey to be carried out in his/her school.

SIGNED: _____

Parent/Guardian

Appendix C

Letter to Parent/Guardian

Dear Parent/Guardian:

We wish to express deep appreciation for the way in which both parents and students associated with the school health survey have cooperated so readily with our efforts thus far.

As mentioned in our first letter, which described briefly the investigations to be used, the final test of each child having received parental consent will be to draw a 10-hour fasting blood sample. Because of such large numbers involved, not all individuals can be tested together on the same day. Your child's blood test will be carried out tomorrow morning.

We ask that the following instructions be followed carefully:

(i) Please do not allow your child to eat food or drink any liquids, except water, after 11:00 p.m. tonight.

(ii) Your child should not eat breakfast in the morning. Again, remember that water may be taken if he/she is thirsty.

NOTE: After a blood sample has been drawn on arrival at school, all children tested will be treated to a breakfast party. Please, then, remind your son or daughter how very important it is that he/she eat nothing on the way to school.

(iii) The attached form should be completed and returned tomorrow morning. If, for a particular health problem or any other reason, you would not have your child go without eating until after he/she reaches school, please indicate

this by checking next to the appropriate statement.

In order that accurate information might be obtained in answer to certain questions relating to the family's health, a personal interview will be arranged with each parent/guardian over the next two months.

Many thanks again for your interest and consideration.

Yours sincerely,

Appendix D

Consent Form

CORONARY HEART DISEASE RISK FACTOR STUDY

Please check one of the following:

(1) To the best of my knowledge, the child named below has has not ☐ eaten food or taken liquids (except water) since 11:00 p.m. last night.

(2) Instructions for fasting were not followed due to health or other reasons.

NAME OF CHILD: _____

SIGNATURE OF PARENT/GUARDIAN: _____

DATE: _____

Appendix E

Questionnaire 1979-1980

QUESTIONNAIRE 1979-1980

EPIDEMIOLOGICAL STUDY OF CORONARY HEART DISEASE RISK FACTORS IN NEWFOUNDLAND CHILDREN

PART I: PERSONAL INFORMATION

1. Subject's I.D. No. ☐☐☐☐☐
2. Date of Examination ☐☐☐☐☐☐
3. Name of Community:
   - Harbour Grace ☐
   - Carbonear ☐
   - Stephenville ☐
   - Stephenville Crossing ☐
4. Name of School _____
5. Grade ☐
6. Who are you living with?
   - Father/Mother ☐
   - Guardian ☐
7. Home Address _____
8. Phone No. ☐☐☐☐☐☐☐
9. Where were you born? _____
10. How long have you lived in this community?
    - Less than a year ☐
    - 1-4 years ☐
    - 5-8 years ☐
    - 9-12 years ☐

10. cont'd

13-16 years ☐

17-20 years ☐

21-24 years ☐

11. Sex

Male ☐

Female ☐

12. Age at last birthday ☐☐

13. Date of Birth ☐☐ ☐☐ ☐☐

14. Do you have any brother(s) or sister(s) attending this school?

No ☐

Yes ☐

If yes, how many brothers? ☐

How many sisters? ☐

15. Do you have any brother(s) or sister(s) attending another school in Newfoundland?

No ☐

Yes ☐

If yes, name of school(s): _____

_____

16. What is the name of your family doctor?

_____

17. Above information verified by parent(s)?

No ☐

Yes ☐

PART II: MEDICAL INFORMATION

18. Are you being looked after by a doctor for any illness?

No ☐

Yes ☐

If yes, what kind of illness? _____

19. Do you take any medicine regularly?

No ☐

Yes ☐

If yes, what kind?

Pills ☐

Liquid ☐

Name of medicine _____

Don't know the name ☐

20. Above information verified by family doctor?

No ☐

Yes ☐

PART III: SMOKING HABITS

21. Subject is too young to be asked ☐

22. Do you smoke?

No ☐

If no, go to item 24

Subject does not wish to answer, go to item 25

23. If yes, how many cigarettes (including thin cigars) do you smoke per week?

1-5	☐
6-10	☐
11-15	☐
16-20	☐
21-25	☐
Don't Know	☐

24. Although you do not smoke, have you ever smoked or lit a cigarette or cigar?

No	☐
Yes	☐

PART IV: PHYSICAL EXAMINATION

25. Standing Height (cm) ☐☐☐

26. Body Weight (kg) ☐☐☐

BTood pressure readings (mmHg) (Physiometrics Recorder)

	1st reading	2nd reading
27. Systolic	☐☐☐	☐☐☐
28. Diastolic (IV)	☐☐☐	☐☐☐

Appendix F

Measurement Error in Blind Duplicate Measurements

Serum Total Cholesterol (mg%)

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

SERUM TOTAL CHOLESTEROL (mg%)

READING				
1	2	SUM	MEAN	ss
187	200	387	193.5	84.5
150	145	295	147.5	12.5
119	110	229	114.5	40.5
134	136	270	135.0	2.0
138	147	285	142.5	40.5
170	178	348	174.0	32.0
177	181	358	179.0	8.0
148	145	293	146.5	4.5
239	219	458	229.0	200.0
226	229	455	227.5	4.5
178	185	363	181.5	24.5
162	168	330	165.0	18.0
235	210	445	222.5	312.5
172	177	349	174.5	12.5
193	196	389	194.5	4.5
201	214	415	207.5	84.5
182	197	379	189.5	12.5
157	167	324	162.0	112.5
		6372	177.0	1010.5

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{7.493}{177.0} \times 100\%$

$CV_{me} = 4.2\%$

Appendix G

Measurement Error in Blind Duplicate Measurements

Serum Total Cholesterol (mg%)

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

SERUM TOTAL CHOLESTEROL (mg%)

READING				
*CDC	LOCAL			
1	2	SUM	MEAN	ss
351	377	728	364	338
248	252	500	250	8
127	127	254	127	0
df = 3		1482	247	346

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{10.74}{247} \times 100\%$

$CV_{me} = 4.3\%$

* Samples from Center for Disease Control in Atlanta, Georgia, U.S.A.

Appendix H

Measurement Error in Blind Duplicate Measurements

HDL Serum Cholesterol

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

HDL SERUM CHOLESTEROL

READING				
1	2	SUM	MEAN	ss
75	78	153	76.5	4.5
68	66	134	67.0	2.0
37	40	77	38.5	4.5
46	47	93	46.5	0.5
52	57	109	54.5	12.5
58	60	118	59.0	2.0
60	64	124	62.0	8.0
39	38	77	38.5	0.5
71	72	143	71.5	0.5
72	70	142	71.0	2.0
58	59	117	58.5	0.5
58	61	119	59.5	4.5
82	80	162	81.0	2.0
55	54	109	54.5	0.5
75	71	146	73.0	8.0
54	56	110	55.0	2.0
64	61	125	62.5	4.5
51	53	104	52.0	2.0
df = 18		2162	60.1	61.0

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{1.84}{60.06} \times 100\%$

$CV_{me} = 3.1\%$

Appendix 1

Measurement Error in Blind Duplicate Measurements

Serum Triglyceride (mg%)

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

SERUM TRIGLYCERIDE (mg%)

READING 1	2	SUM	MEAN	ss
64	66	130	65.0	2.0
67	71	138	69.0	8.0
62	61	123	61.5	0.5
36	31	67	33.5	12.5
79	78	157	78.5	0.5
122	116	238	119.0	18.0
64	66	130	65.0	2.0
76	79	155	77.5	4.5
38	38	76	38.0	0.0
57	54	111	55.5	4.5
74	76	150	75.0	2.0
20	24	44	22.0	8.0
44	48	92	46.0	8.0
62	60	122	61.0	2.0
46	44	90	45.0	2.0
58	60	118	59.0	2.0
47	43	90	45.0	8.0
36	34	70	35.0	2.0
df = 18		2101	58.4	86.5

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{2.19}{58.36} \times 100\%$

$CV_{me} = 3.7\%$

Appendix J

Measurement Error in Blind Duplicate Measurements

Serum Triglyceride (mg%)

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

SERUM TRIGLYCERIDE (mg%)

READING				
*CDC	LOCAL			
1	2	SUM	MEAN	ss
56	61	117	58.5	12.5
160	156	316	158.0	8.0
239	229	468	234.0	50.0
		901	150.2	70.5

df = 3

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{4.85}{150.17} \times 100\%$

$CV_{me} = 3.2\%$

* Samples from Center for Disease Control in Atlanta, Georgia, U.S.A.

Appendix K

Measurement Error in Blind Duplicate Measurements

Serum Uric Acid (mg%)

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

SERUM URIC ACID (mg%)

READING				
1	2	SUM	MEAN	ss
4.1	4.3	8.4	4.20	0.020
5.3	5.5	10.8	5.40	0.020
4.3	4.6	8.9	4.45	0.045
3.8	3.8	7.6	3.80	0.000
3.7	3.6	7.3	3.65	0.005
7.6	7.8	15.4	7.70	0.020
7.1	7.0	14.1	7.05	0.005
4.9	5.0	9.9	4.95	0.005
5.5	5.8	11.3	5.65	0.045
3.7	3.6	7.3	3.65	0.005
3.9	3.9	7.8	3.90	0.000
4.6	4.8	9.4	4.70	0.020
4.8	4.9	9.7	4.85	0.005
4.2	4.0	8.2	4.10	0.020
6.4	6.2	12.6	6.30	0.020
4.8	4.8	9.6	4.80	0.000
4.5	4.7	9.2	4.60	0.020
3.3	3.0	6.3	3.15	0.045
df = 18		173.8	4.83	0.300

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{.13}{4.83} \times 100\%$

$CV_{me} = 2.7\%$

Appendix L

Measurement Error in Blind Duplicate Measurements

Serum Glucose (mg%)

MEASUREMENT ERROR IN BLIND DUPLICATE MEASUREMENTS

SERUM GLUCOSE (mg%)

READING 1	READING 2	SUM	MEAN	ss
98	104	202	101.0	18.0
92	88	180	90.0	8.0
89	96	185	92.5	24.5
89	88	177	88.5	0.5
88	100	188	94.0	72.0
81	82	163	81.5	0.5
83	89	172	86.0	18.0
88	79	167	83.5	40.5
81	80	161	80.5	0.5
85	83	168	84.0	2.0
85	91	176	88.0	18.0
93	86	179	89.5	24.5
94	99	193	96.5	12.5
89	79	168	84.0	50.0
107	107	214	107.0	0.0
75	77	152	76.0	2.0
78	84	162	81.0	18.0
83	89	172	86.0	18.0
df = 18		3179	88.3	327.5

CV for measurement error = $\frac{s}{\bar{x}}$

where s = standard deviation for measurement error

$CV_{me} = \frac{4.26}{88.31} \times 100\%$

$CV_{me} = 4.8\%$

Appendix M

Information Sheet

CORONARY HEART DISEASE RISK FACTORS IN NEWFOUNDLAND CHILDREN

SCHOOL: PRINCIPAL:

ADDRESS: DATE:

PHONE NO.:

STUDENT'S AGE:

NAME(Surname First)	ADDRESS	DATE OF BIRTH	PHONE NO.

"A journey of a million miles
begins with a single step"

Chinese Proverb