Cardiac Risk Factor Management in the Offspring of Patients with Premature Ischemic Heart Disease

by

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Abstract

Objective: To determine the prevalence of cardiac risk factors in offspring of patients with premature ischemic heart disease, their awareness of these risk factors, and to find out what proportion of offspring have had risk factor assessment including cholesterol screening and what proportion have adopted risk factor reducing strategies.

Design: Telephone survey of men less than 50 and women less than 60 years of age with documented coronary heart disease, and of a systematic sample of their offspring. Adults with coronary disease were identified from either the computerized records of the only adult cardiac catheterization laboratory in the region, or the computerized records of all those in seven regional hospitals with a recent myocardial infarction. Offspring over age 15 were interviewed personally, while information was obtained about younger offspring through their parents.

Setting: Ottawa, Ont. and surrounding area.

Participants: 1) A total of 318 patients with premature IHD who lived in the Ottawa-Hull metropolitan area, had a known telephone number, and had a myocardial infarction or positive angiogram between January 1, 1988 and February 28, 1990. 2) One offspring from 298 of the 318 patients.

Main Results: The median age of the offspring was 20 years (range 2 - 39). Among the late adolescent and young adult progeny 37% were smokers, 31% were overweight and 30% exercised less than three times per week. Although 78% had been examined by a physician in the preceding three years, only 97 (44%) reported having had a blood cholesterol measurement performed during that time and only five of the 97 actually knew their cholesterol levels. Fifty-seven percent of the males had had a blood pressure measurement in the previous year. Although all respondents were aware that eating fatty food could contribute to heart disease and cholesterol elevation, few (13%) recognized the role of heredity as a causal factor for heart disease and only 22% reported that they would know how to lower their blood cholesterol.

Conclusions: The low rates of cardiovascular risk factor assessment and management identified in this survey represent missed opportunities for primary prevention. We suggest a coordinated program of education, risk factor assessment and intervention following active identification of family members each time an index patient develops clinically important premature ischemic heart disease.

Acknowledgements

Many people contributed to the successful completion of this study. My heartfelt thanks to all. The survey never would have been done without the encouragement of Dr. Peter Rowe, who collaborated in numerous aspects of the study. Dr. Richard Davies facilitated the survey by providing the patient databases and easing communications at the University of Ottawa Heart Institute. The meta-analysis in the literature review was done under the guidance of and in collaboration with Dr. Sylvie Stachenko and Dr. Andres Petrasovits. Dr. Geoffrey Dunkley kindly provided a copy of the raw data of the Ottawa Carleton Heart Beat Survey so that results of the two surveys could be compared. My involvement with the Ottawa Carleton Heart Beat Program while I was at the Health Department triggered my interest in this whole area. Monica Prince provided accurate data entry. The study could not have been completed without the diligent efforts of all the interviewers. Sylvia Pasher provided valued secretarial assistance. My thesis advisor, Dr. Nick Birkett was forthcoming with many helpful suggestions. The study was supported in part by a grant from Merck Frosst Canada.

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Introduction

Cardiovascular disease remains the leading cause of death in Canada as well as an important cause of morbidity and disability.[1] It is a prime focus for increasing prevention efforts in the area of chronic disease because it has identifiable, modifiable risk factors. Although rates of ischemic heart disease are declining by approximately 3% annually,[2] the potential for prevention is much greater. Further reductions of 10 to 30% in ischemic heart disease events might be obtained with a decline in blood cholesterol levels in the majority of the population.[3,4]

Premature atherosclerotic heart disease in a first degree relative is the strongest predictor of an individual's risk of heart disease. It exceeds by a factor of two the risk posed by an elevated total cholesterol level alone,[5] with family members having up to 13 times the risk of the general population.[5,6,7,8,9,10,11,12,13] The evidence concerning the aggregation of risk factors in families suggests that these offspring will have more modifiable risk factors such as hypertension, smoking and dyslipidemia. A cost-effective primary prevention strategy could focus on screening within identified high risk families. Many studies have examined the aggregation of risk factors in families, and some have examined awareness of risk factors in siblings of people with premature coronary disease.[14,15] Yet, we are not aware of any studies examining the awareness and management of cardiovascular risk factors among offspring of individuals with premature ischemic heart disease.

We conducted a survey of children and young adults in the Ottawa region who are at risk for premature atherosclerosis by virtue of premature ischemic heart disease (IHD) in a parent. We interviewed subjects with known coronary disease - those with either a documented myocardial infarction or angiographically documented coronary disease - and one offspring from each identified family. This report describes the coronary heart disease (CHD) risk factors and health practices in both the parents and the children, the extent of recent changes in those practices, the degree of screening for risk factors and the knowledge levels of the offspring concerning CHD risk factors.

Literature Review

Why focus on families with CHD? What's wrong with a general population approach? Why look at children altogether? Should we be screening anyone - or everyone? Answering questions such as these involves showing the importance of family history as a risk factor for CHD, examining the aggregation of risk factors within families, considering when CHD begins, considering the controversies around population and high risk approaches to risk factor modification, judging the evidence on causation in relation to risk factors and CHD and looking at the efficacy of interventions. The literature on these areas is vast and cannot be comprehensively reviewed in this context. Currently major controversies surround cholesterol screening. The present review will therefore focus to a greater extent on cholesterol, although high risk families need to reduce as many risk factors as possible. As background to the survey, the present review will summarize the literature on 1) the evidence for family history and cholesterol as risk factors for CHD and on the aggregation and segregation of these risk factors, 2) the evidence for CHD beginning in childhood, and 3) cholesterol screening recommendations. It will then address the controversies in cholesterol screening in childhood and the current practices in relation to the recommendations and briefly outline some of the intervention programs that have involved youth and high risk families.

Articles for inclusion were identified for review through Medline and the references of reviews and identified papers. This will tend to identify larger and more frequently cited studies. Because of the volume of the literature and the variety of areas addressed, no attempt was made to identify every study in each area, except in the case of the intervention trials on cholesterol. This is not intended as a comprehensive review of all these areas but rather tries to highlight some of the research that led to the present study.

Evidence for family history as risk factor for CHD

Even before the idea of coronary risk factors arose, Osler observed the clustering of heart attacks within families. Recent studies have shown that family members may have up to 13 times the risk of the general population. However, the relative role ot family history independent from the major known risk factors and its mechanisms of action are still debated[16,17,13] since both premature IHD and the major risk factors - hyperlipidemia, hypertension, smoking and diabetes - cluster in families.[18,19,20,21,22,23,24,25]

This debate is not new. In 1963 Epstein reviewed the literature on hereditary aspects of coronary heart disease.[20] He wondered, given the importance of the topic, why there had been few reported studies concerned with familial aggregations of coronary heart disease. The studies that existed tended to show the presence of aggregations, but told little of the strength of the association. In fact, at that time there was "an unfortunate tendency at times to equate the genetic aspects of coronary heart disease, or for that matter atherosclerosis, with the genetic aspects of hypercholesterolemia." The review concluded that "although the evidence suggests a definite but not striking tendency for coronary disease to cluster in families, a quantitative assessment of the relative importance of familial influences in the genesis of these disorders is not possible at the present time."[20]

The following decades started to provide some evidence which Epstein found lacking. One early study was the Tecumseh Community Health Study, which compared the incidence rates of CHD among men whose parents had died of CHD and men whose parents had died of other causes.[26,19] Men with at least one deceased parent at the time of first examination were followed for a median of 47 months. Among men ages 40 to 59 there was a 12.8% incidence of fatal CHD in the follow-up period among those whose fathers had died of CHD before 65 compared to a 0.7% incidence among men whose fathers had died of other causes. Men whose parents had died of CHD also tended to have higher cholesterol levels and blood glucose levels than men whose parents had died of other causes.

The Western Collaborative Group Study, a prospective study of CHD in 39-59 year old men found an increased incidence of symptomatic MI and angina pectoris in men under age 50 who reported a parental history of heart disease.[10] The relative risks were approximately twofold. Men over age 50 had increased rates of angina, but not MI. When adjusted for other risk factors, there remained a significant association of a positive parental history and the combined incidence of MI and angina in the younger age group. Although the authors analyzed the data in terms of the age of MI of the offspring, there was no information on the age of MI of the parents.

These early studies of CHD thus demonstrated 2-3 times greater incidence of disease in first degree relatives of patients than in control subjects. The Honolulu Heart Study also showed 2.5 times as many fathers of CHD patients dying of CHD, compared to control fathers.[27] But, further analysis of the data showed that young fathers of men with early onset CHD had an 11 fold risk of death. More recent studies have also shown family history to play a much larger relative role in younger patients with CHD, where the parent also had a history of premature CHD. This would suggest a distinct hereditary component in young patients. For example, data from Finland[7] showed that the risk of CHD by age 55 was 11.4, 8.3, 1.3 times greater in South Finland and 6.7, 3.6, 1.8 times greater in East Finland for the brothers of patients than for the brothers of reference subjects depending on whether the MI in the patient was first diagnosed at less than 46, 46 - 50, or 51-55 years of age. High blood pressure and elevated cholesterol levels were most common among relatives of the youngest patients and were less common as patient age increased.

A case control study in Colorado compared 19 variables in 207 cases with MI before age 55 to 621 matched controls. The highest relative risk (10.1) was for a positive family history of CHD before age 55 in a first-degree relative. The next highest was for IHD in a first-degree relative before age 65 (RR = 7.1) This compared to a relative risk of 4.3 for a cholesterol level above 6.7 mmol/L.[5] Although this study examined 19 independent variables, no multivariate analyses were done. The study illustrates the association of family history and CHD, but does not contribute to our knowledge of in the independence of family history as a risk factor.

The Rancho Bernardo study was a large prospective study of 4014 men and women 40 to 79 years old with no known cardiovascular disease. Nine year follow-up showed men under 60 years of age with a family history of heart attack before age 50 to have 10 times higher mortality rate from ischemic heart disease and cardiovascular disease, and twice the all cause death rate of those with no family history of heart attack.[13] There were no ischemic heart disease deaths among the 843 women under the age of 60 during the study period.

Younger men had significantly higher blood pressures and cholesterol levels than older men with CHD. However, there was still a fivefold increased risk of ischemic heart disease death that was independent of all risk factors.

While these studies have provided further evidence of the strength of association between family history and premature ischemic heart disease, many questions remain. In a 1983 review of genetic aspects of CHD, Neufeld and Goldbourt[8] describe how "the complexities of CHD pose extremely difficult problems for analysis of the genetic component." If one considers the entire process of atherosclerosis and the different aspects that might be affected by genetic differences - such as genetic differences in the conduction system of the heart, the distribution pattern of the coronary arterial tree, myocardial metabolism, coronary artery spasm, platelet aggregation, and genetic differences in how the arterial wall reacts to different risk factor levels - it is readily apparent why the relative role of genetics is still being debated. The aggregation of risk factors in families and the examination of the independent effect of family history will be explored in the next two sections. What emerges from this literature, however, is the important role family history plays in *premature* heart disease, as opposed to heart disease occurring later in life.

Familial aggregation of risk factors

Hypertension, elevated lipid levels and smoking are the three major risk factors for premature CHD. Obesity and diabetes also play a role. Hypertension, serum lipid levels, diabetes mellitus and obesity have all shown familial clustering, and have also been associated with a family history of premature CHD. In fact, while not considered in the same way because a genetic component is not thought to be involved, higher smoking rates have also been shown in the children of CHD victims.[28] Children of smoking parents face a double whammy: they have abnormalities of their myocardial oxygen supply demand ratio and they are more likely to smoke themselves.[29]

Familial aggregation of blood pressure was noted in the 1960's in Tecumseh, Michigan,[30] and in the cardiovascular prevalence survey done in Evans County, Georgia in 1960 through 1962.[31] Studies of childhood sibling aggregation of CHD risk factors in Bogalusa, Louisiana noted similar findings among children.[32] The correlations between child siblings for height, weight and blood pressure were on the same order of magnitude as the Evans County study found among adults. For systolic blood pressure, the correlations among adult siblings are approximately 0.20, and between parents and offspring 0.15.[29] Whereas the correlations among fraternal twins are in the order of 0.25, among identical twins they are as high as 0.55. Studies of very young children have shown that familial aggregation of blood pressure begins during infancy.[33] While most of the literature is consistent, some smaller studies, which showed lipoprotein aggregations in families, could not show blood pressure aggregation in family members of patients with a premature MI.[34]

This review will focus primarily on the familial aggregation of cholesterol levels. The evidence is twofold: Elevated cholesterol levels cluster within families, and elevated cholesterol levels are associated with premature CHD in relatives. That works both ways: CHD patients have children with higher cholesterol levels, and children with high cholesterol levels have more relatives with CHD. Each of these approaches will be reviewed.

Aggregation of cholesterol levels in families: The LRC Family Study compared offspring and siblings of probands with severe hypercholesterolemia with offspring and siblings of normal probands. Offspring of hypercholesterolemic probands had total and LDL cholesterol levels in the top decile two to three times as often as did the relatives of normal probands. Siblings' cholesterol levels were in the top decile two to five times as frequently.[35] The Princeton School Study, another aspect of the Cincinnati LRC Program, examined intrafamilial associations of HDL and LDL. Mean levels of HDL and LDL cholesterol progressively increased as parental classification of LDL and HDL progressed from the lowest to the highest.[23,36] For HDL cholesterol there was a significant positive relationship between parents and children ($r = 0.48$) and between siblings ($r = 0.28$). The parents' HDL level accounted for 25% of the variation of the children's levels. LDL cholesterol associations were less consistent. Between parents and children the overall relations were significant ($r = 0.17$) and comparing parents to daughters, mothers to daughters ($r = 0.51$) and siblings to each other ($r = 0.39$) also gave significant positive associations. But, when parents and sons and fathers and daughters were examined the relationships did not hold.

Studies showing CHD mortality predicts progeny's lipid levels: Several case control studies have documented significant familial aggregation of cholesterol. Hennekens and associates showed that men with premature MI had children with greater sibling aggregation of cholesterol levels ($r = 0.71$ vs. 0.56) and higher mean cholesterol levels than children of healthy parents (mean 206 mg/dl vs. 177 mg/dl).[37] Glueck and colleagues also used a case control design to compare lipids and lipoproteins in children from families where one parent had a myocardial infarction before 50 years of age to children from "normal" families.[21]

Cholesterol levels of the children of MI patients not only had a higher mean, but the distribution, instead of being normal, was markedly skewed to the right.

Studies of angiographically diagnosed CHD before age 50 have shown similar results. Among such men, Lee and colleagues found that 65% had a lipid abnormality - either high triglyceride or LDL levels, low HDL levels or some combination. Fifty-one percent of the offspring also had a lipid abnormality, with the distributions of the lipids in the offspring closely resembling those in their fathers.[34] The Framingham Offspring Study[28] and The Lipid Research Clinics Program Prevalence Study[38] also showed that parental (especially paternal) cardiovascular premature mortality predicted the cholesterol levels of their offspring, especially the sons. In the latter study mean cholesterols among men whose fathers had died of CHD before age 60 were 6 mg/dL higher than men whose fathers lived beyond age 60.

Studies identifying children first have also confirmed familial aggregation. Bodurtha and colleagues studied 102 adolescent twin pairs and their parents. The 12% of children with a family history of premature death from IHD had lower levels of HDL2-C than the others.[39]

Although studies in women do not always yield the same results as those in men, a strong hereditary basis for LDL and HDL cholesterol levels has been shown in women. Austin and associates observed this in a study of the relative roles of genetic heritability and shared environmental influences in predicting risk factors for CHD in adult female twins.[40] Among 434 pairs of twins, they found a strong hereditary basis for LDL and HDL cholesterol levels and body weight in women. Blood pressure was much less related to inherited factors. (Using heritability estimates, defined as the genetic variance divided by the total variance, they estimated heritability adjusted for age and environmental influences to be 0.93 for LDL cholesterol, 0.87 for HDL cholesterol and 0.40 and 0.27 for systolic and diastolic blood pressure respectively.) The genetic basis for cholesterol and body weight was strong enough for the authors to suggest it as a reason for the failure of diet modification to significantly alter cholesterol levels and weight in women.

Prospective studies examining CHD in parents according to children's cholesterol levels: Several large prospective studies of coronary risk factors in school children have identified and studied the families of index cases with cholesterol levels above the 95th percentile (high), below the fifth percentile (low), or in between (middle). The Muscatine Study examined relatives of 148 such children. Children with cholesterol levels above the 95th percentile in successive surveys two years apart identified adult relatives with at least double the coronary mortality rate of relatives of children with lower cholesterol levels.[41] Deaths from MI occurred in 6% of male and 2.8% of female relatives age 30-59 of children in the high group. The corresponding frequencies in the middle group were 3% and 0% and in the low group 2.8% and 0.5%.

Similarly, Moll and colleagues studied the families of 100 index cases, ages 6 - 16, from a survey of 3666 children in schools in Rochester, Minnesota.[42] Not only did first degree relatives of high cholesterol cases have higher cholesterol levels, but siblings of the high cholesterol index cases had higher levels of triglycerides and systolic blood pressure as well. Grandfathers of the high cholesterol index cases showed increased rates of coronary mortality (2.5 times increased in those under 65), with HDL and LDL being the best predictors of CHD. Families with high cholesterol levels seemed to have stronger genetic influences on their cholesterol levels than other families. The authors interpret the data to support the hypothesis of a dominant allele with a major effect on cholesterol levels in addition to other additive polygenetic and environmental factors.[42]

As cholesterol measurements have become more sophisticated better predictions have resulted. The Bogalusa Heart Study examined fasting serum lipids, lipoprotein cholesterol and risk factors in 321 parents of children with low or high levels of ß and pre-ß-lipoprotein cholesterol. Children tended to mirror their parents' lipoprotein cholesterol levels. Parents of children with high ß and pre-ß-lipoprotein cholesterol levels differed from parents of children with low levels in several respects: They were more frequently overweight, they had higher ß-lipoprotein levels and they had higher levels of other coronary risk factors as well as clinical premature ischemic heart disease.

These three studies raise additional questions. The Muscatine and Rochester studies show that lipid and lipoprotein levels of children ages six to sixteen from a single cross-sectional survey can identify families at increased risk for CHD. On the other hand, cord blood studies have shown that at birth, neither total nor HDL cholesterol can predict CHD prevalence in adult relatives.[43,44] At what age, then, between birth and six years, do the levels of cholesterol, LDL and HDL become predictive? The Bogalusa study further indicates that while childhood lipid levels are predictive of adult CHD, childhood lipid correlations are lower than those found in adult studies. This may suggest that lipid levels are more responsive to environmental factors during childhood than during adulthood and that early environmental factors have a role to play.[32]

The prospective studies outlined here have demonstrated the association between cholesterol levels in children and CHD rates in their relatives. The importance of actual measurement in studying risk factor aggregation is shown by a study by Becker and colleagues.[15] They measured blood pressure and lipids in 150 coronary disease-free siblings of 86 probands with premature CHD. Forty-eight percent of brothers and 41% of sisters were hypertensive - but 65% of these brothers and 47% of these sisters were unaware of their hypertension. Forty-five percent of brothers and 22% of sisters had a lipid abnormality, most commonly elevated LDL cholesterol - yet 74% of these siblings were unaware of the abnormality. Thus studies of risk factor aggregation will underestimate the effect if they rely on self-reporting of risk factors.

Family history and cholesterol: are they separate risk factors?

We have seen evidence that family history of heart disease increases the risk of coronary heart disease.[8,9,10,11,12,13,45] We have also examined the evidence on familial aggregation of risk factors, which seems marked for cholesterol levels.[36,30,46,47,48,38] Since CHD and its risk factors - especially cholesterol - aggregate in families, to what extent are the risk factors responsible for the increased incidence of cardiovascular disease, and to what extent is positive family history an independent risk factor?

The extent to which family history is a risk factor independent of the other cardiovascular risk factors remains controversial. While some interpret the data on aggregation of risk factors in families with ischemic heart disease to suggest that it is the other risk factors that are responsible for the ischemic heart disease, others suggest an independent effect of family history. Rissanen and Nikkila interpreted their Finnish data on familial aggregation of risk factors to suggest that hyperlipoproteinemia and hypertension could be solely responsible for most of the observed aggregation of coronary artery disease.[6] They assessed the incidence of CHD in 560 relatives of 104 men who had developed angina before age 56 and 498 relatives of 94 controls. CHD mortality rates were five times higher (25%) for fathers of patients and CHD incidence rates were 5.5 times higher for brothers of patients than for brothers of controls. However, hyperlipidemia, hypertension, and clinical diabetes were also more common. Although the interpretation was that this increased prevalence of risk factors was responsible for the increased incidence of CHD, the data were not analyzed in a way that would support or refute this hypothesis.

Several approaches have been used to demonstrate the relationships between familial aggregation and modifiable risk factors. Case control studies have been done both on patients who have undergone coronary angiography,[49,50,51,52] and on patients with clinical endpoints such as MI.[53,54] For example, Shea and associates attempted to examine the independence of family history as a risk factor in CHD patients who had undergone coronary angiography by requesting information about all their first degree relatives.[17] Life table analysis of the data showed four to six times greater probability of MI in the 50 - 65 year age range in relatives of patients compared with relatives of controls. Angina and sudden death were also more common. For the group as a whole, the relative risk of any ischemic endpoint was 2 to 3 for relatives of cases compared to relatives of controls. When they examined CHD incidence among relatives in relation to the level of risk factors in the index patients, they found that if the index patient had coronary heart disease despite a low level of risk factors, the relatives had a higher incidence and earlier age of onset of disease than relatives of patients with CHD who are at higher risk. The cumulative probability of MI in a 50 to 60 year old relative of a patient in the lowest risk quartile was double that of relatives of patients in the upper three quartiles. Life table analyses showed both greater frequency and earlier age of onset of MI in relatives of patients with the fewest risk factors compared to relatives of patients with more risk factors. These results tend to suggest an independent effect of family history.

Prospective studies have compared people with low and high risk levels as to their family history[26,55] and have compared risk levels of people with a family history of CHD to those without a positive family history.[56,57,36,58] The Princeton School study[58] assessed the relationships of parental history of MI, hypertension, stroke, diabetes to CHD risk factors (cholesterol, TG, HDL, LDL, Quetelet index, SBP, DBP) of 556 adult progeny 25 years and older. For the most part, neither univariate nor multivariate comparisons supported the hypothesis that positive family history is associated with elevated risk factors in the children. However, paternal MI (but not maternal) was associated with higher LDL and triglyceride levels in offspring. Multivariate analysis of covariance used a model whereby risk factors were the dependent variables, and the independent variables included age, sex, race and the paternal and maternal history of MI, hypertension, stroke, and diabetes. In this model neither paternal nor maternal history of MI was significantly related to elevated risk factor levels. However, MI, hypertension, stroke and diabetes are not truly independent variables. This may tend to diminish the effects. In addition, offspring in this study had an average age of 40, with the parents on average in their 60's. As discussed earlier, the strongest effects are seen when parents have a history of premature heart disease.

The more convincing prospective studies have been those examining CHD incidence in populations with known risk factor levels and family histories. Most such studies have suggested that family history predicts cardiovascular disease independent of the modifiable risk factors.[10,11,12,13]

In the Framingham Study prospective data on siblings were examined to circumvent the difficulties with obtaining good data on two different generations. Data on 186 pairs of brothers were analyzed using multiple logistic regression to assess the independence of familial aggregation as a risk factor for CHD. The independent variables were the risk factors of the older brother. The dependent variable was the CHD endpoint of the younger brother. The risk of CHD occurring in both brothers of a pair was about 1.5 times what would be expected given their risk factors.[12]

The Paris Prospective Study followed 7484 men ages 43 to 54 for six and a half years. CHD incidence was examined in relation to parental history of CHD and hypertension. Traditional risk factors only explained part of the increased incidence in those with a positive family history. Paternal - but not maternal - history of CHD and paternal history of hypertension contributed independently to the risk of CHD. The relative risk was 3 if both paternal histories were positive.[11]

What emerges from the above studies is the likely existence of an effect of family history on CHD incidence that is independent of cholesterol. However, this does not have to indicate a genetic effect independent of *any* risk factors. It may suggest a sharing of risk factors that are not yet been known to be important. Hopkins and Williams have suggested 246 coronary risk factors.[59] Recently iron levels have been shown to be associated with CHD. Any risk factor either inherited or shared within a family could account for a portion of the results observed. The disentanglement of hereditary and environmental components is ongoing and beyond the scope of this review. However, multigenerational patterns make some hereditary component likely.

An independent genetic component would not diminish the importance of modifying risk factors in those with a positive family history. The Rancho Bernardo Study looked at modifiable risk factors in men under 60 years of age with a family history of heart attack who were at fivefold increased risk. They estimated that approximately 68% of the excess deaths in men with a positive family history were attributable to the interaction of smoking with family history. Comparing those with a positive family history to those without, the relative risks of cardiovascular disease in smokers vs. nonsmokers were 2.5 and 1.1.[60] Thus identifying families at risk can be an important preventive strategy.

Evidence for CHD and cholesterol

The causal relationship between serum cholesterol and coronary heart disease has been established by genetic evidence,[61,62,63] animal studies[64] and a large array of epidemiologic evidence.[65,66,67,68,69,70,71,72,73,74,75,76] The association has been demonstrated in a variety of demographic and ethnic groups and geographic areas, in ecologic studies, migration studies, and cohort studies.[64] The next level of evidence required would be a demonstration that lowering serum cholesterol levels reduces coronary heart disease mortality. This can be used both to show causation and efficacy of intervention. It has been studied in the areas of primary prevention, namely in asymptomatic individuals, and in secondary prevention, that is in persons with coronary heart disease present. This evidence has been extensively reviewed by the Toronto Working Group on Cholesterol Policy.[77] The primary prevention evidence only will be summarized here in a meta-analytic framework. Briefly, three types of primary prevention trials have been done: diet studies, drug studies and multifactorial studies. The diet studies include the New York Anticoronary Club Study,[78] the Finnish Mental Hospital Study[79,80] the Los Angeles Veterans Study,[81] and the Minnesota Coronary Survey.[82,83] Only the latter two were randomized controlled trials. Generally the low fat diets in these trials lowered serum cholesterol levels by 10 to 15% in hypercholesterolemic men. Myocardial infarctions were reduced but there were no reductions in cardiovascular or overall mortality. (See Figures 1 to 3, pages 21 to 23.)

Four randomized controlled trials have been done to assess the potential of cholesterol-lowering drugs to reduce cardiovascular mortality. Each used a different drug. The WHO Cooperative Trial used clofibrate, which was associated with a reduction in nonfatal myocardial infarctions, but an increase in total mortality.[84] The Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) used cholestyramine, and found a reduction in the incidence of myocardial infarction from 8.6% to 7.0% and a reduction in cardiovascular mortality, but no difference in all cause mortality.[85,86] (Figures 1 and 2, pages 21 and 22, show the reduction of risk of fatal and nonfatal myocardial infarction separately, and by two-tailed tests neither is significant. The analysis in the trial uses one-tailed p-values.) The Helsinki Heart Study assessed gemfibrozil therapy and found a 34% reduction in the incidence of cardiac events in the study group, but no difference in overall mortality.[87] A smaller, more methodologically flawed study, was the Upjohn Colestipol Trial.[88] It showed lower CHD morbidity and mortality in men treated with colestipol, especially in those with pre-existing disease.

Several trials have also tried to assess the impact of treating several risk factors for heart disease besides serum cholesterol. The Multiple Risk Factor Intervention Trial used intensive dietary intervention, smoking cessation, hypertension management and exercise and weight reduction in the intervention group. It was unable to detect any differences in mortality between study and control groups.[89] The WHO European Trial intervened on similar risk factors, and had mixed results in various countries.[90,91,92,93] The Oslo trial intervened just on smoking and diet and demonstrated a reduction in coronary events but not in total mortality.[94,95,96,97,98,99]

These studies can be summarized by examining the relative risks of the major outcome measures of these trials. Figure 1 shows the relative risks of fatal coronary heart disease in experimental and control groups in the eight trials that had fatal coronary disease as an outcome measure. This figure also shows pooled data. The data have been pooled in several ways to show drug trials vs. trials using no drugs, and to separate diet trials from multifactorial trials. Finally, all studies are pooled. Although all the pooled relative risks are below 1.0, the confidence limits overlap 1.0 for both the drug studies and the multifactorial studies. For the drug studies, this is primarily the effect of the Clofibrate trial, which had excess mortality in the treated group. For the multifactorial studies, this highlights the results of the MRFIT, which failed to show differences between the two groups. The lowest relative risks are seen with the diet trials. The very low risk of the Finnish Mental Hospital Study, which was unrandomized, contributed to this effect. Combining all the studies, a relative risk of 0.77 is seen for the risk of fatal coronary heart disease in treated groups.

Figure 2 shows the relative risk of nonfatal myocardial infarctions in these same studies. Only six studies are included here, because not all studies used non-fatal MI's as an outcome measure. All the studies show relative risks below one for nonfatal MI's, and the pooled relative risk is 0.71. This 30% reduction in nonfatal MI's together with the 23% reduction in fatal coronary events as a result of cholesterol lowering has been a powerful argument for the widespread promotion of cholesterol lowering.

Those who argue against widespread interventions to lower serum cholesterol often cite data on all cause mortality. Figure 3 summarizes the relative risks of all cause mortality in primary prevention studies of cholesterol lowering. Almost all the confidence limits overlap 1.0, and the pooled relative risk is 0.96. It can be argued that the studies were not designed to measure reductions in all cause mortality and did not have the power or the time span necessary to demonstrate this effect. However the idea of pooling data is in order to yield sufficient numbers to have the power to detect such changes. Moreover, there is a consistent pattern among the studies showing increases in cancer mortality and accidents. Although competing risks may be a factor, the increased neoplasm rates could plausibly either result from cholesterol lowering therapy or from a direct effect of lower cholesterol levels. There is some epidemiological evidence showing increased mortality from cancer in individuals with low cholesterol levels.[100,101]

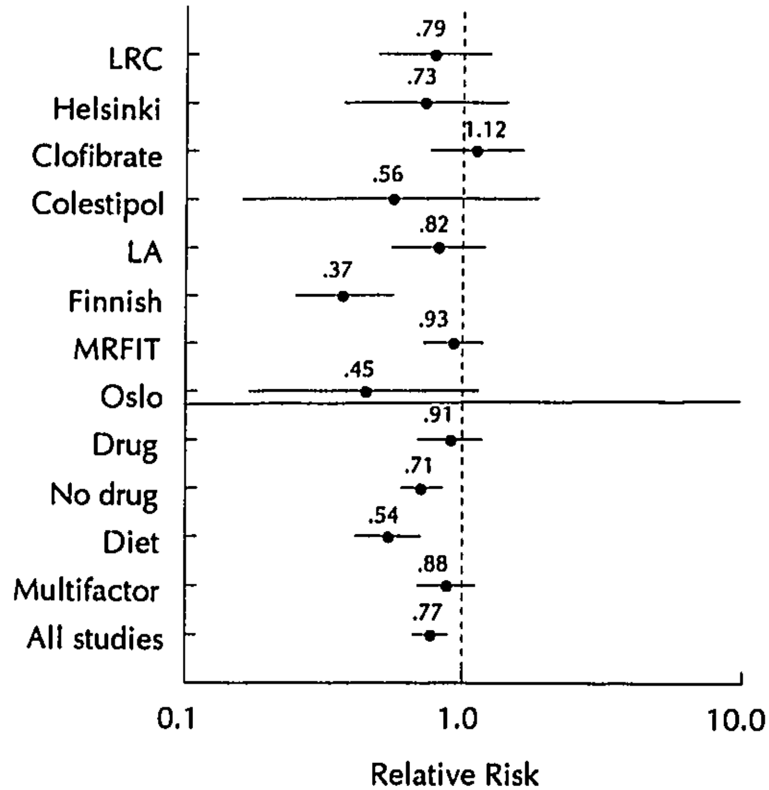

Figure 1. Relative risks of fatal coronary heart disease in primary prevention studies of cholesterol lowering. Pooled data are below the line.

LRC = Lipid Research Clinics Coronary Primary Prevention Trial
Helsinki = Helsinki Heart Study
Clofibrate = WHO Cooperative Trial on Primary Prevention of IHD
Colestipol = Upjohn Colestipol Trial
LA = Los Angeles Veterans Trial
Finnish = Finnish Mental Hospital Study
MRFIT = Multiple Risk Factor Intervention Trial
Oslo = Oslo Diet and Heart Study

No drug = LA, Finnish, MRFIT, Oslo
Drug = LRC, Helsinki, Clofibrate, Colestipol
Diet = LA, Finnish
Multifactorial = MRFIT, Oslo

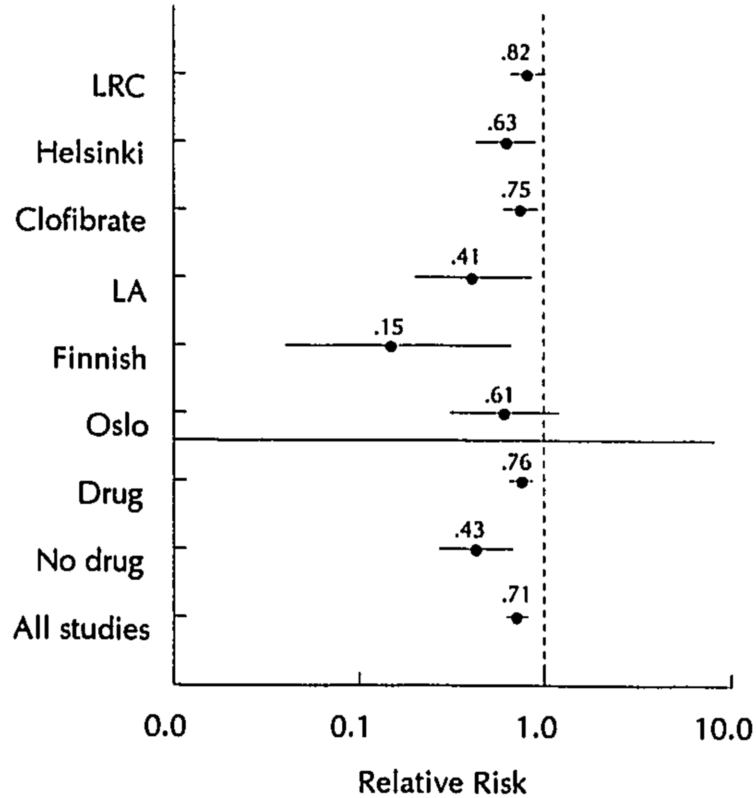

Figure 2. Relative risk of nonfatal myocardial infarction in primary prevention studies of cholesterol lowering. Pooled data below line.

LRC = Lipid Research Clinics Coronary Primary Prevention Trial
Helsinki = Helsinki Heart Study
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Finnish = Finnish Mental Hospital Study
Oslo = Oslo Diet and Heart Study

Drug = LRC, Helsinki, Clofibrate
No drug = LA, Finnish, Oslo

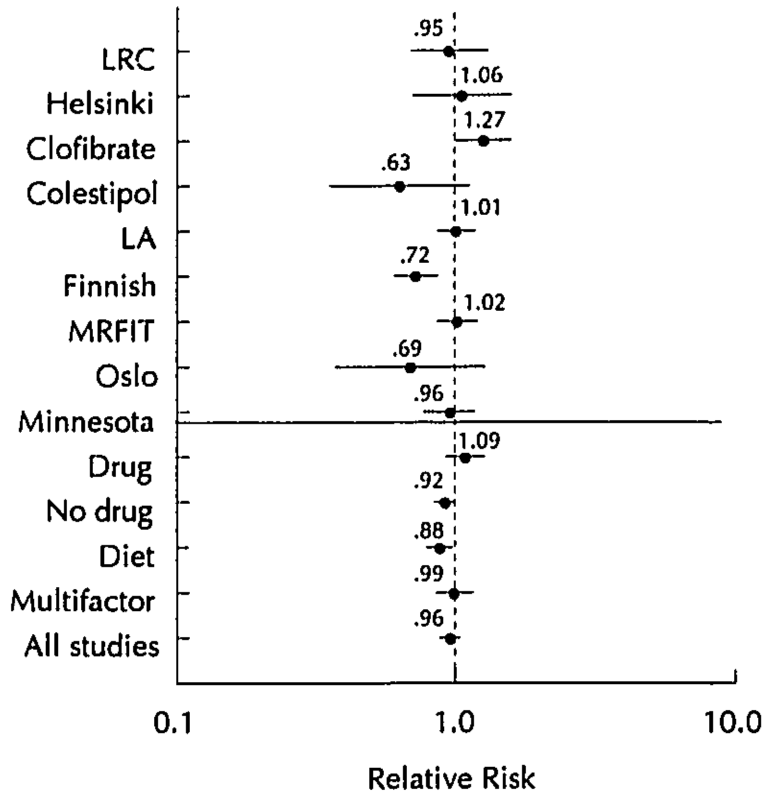

Figure 3. Relative risks of all cause mortality in primary prevention studies of cholesterol lowering. Pooled data below line..

LRC = Lipid Research Clinics Coronary Primary Prevention Trial
Helsinki = Helsinki Heart Study
Clofibrate = WHO Cooperative Trial on Primary Prevention of IHD
Colestipol = Upjohn Colestipol Trial
LA = Los Angeles Veterans Trial
Finnish = Finnish Mental Hospital Study
MRFIT = Multiple Risk Factor Intervention Trial
Oslo = Oslo Diet and Heart Study
Minnesota = Minnesota Heart Study

No drug = LA, Finnish, MRFIT, Oslo, Minnesota
Drug = LRC, Helsinki, Clofibrate, Colestipol
Diet = LA, Finnish, Minnesota
Multifactorial = MRFIT, Oslo

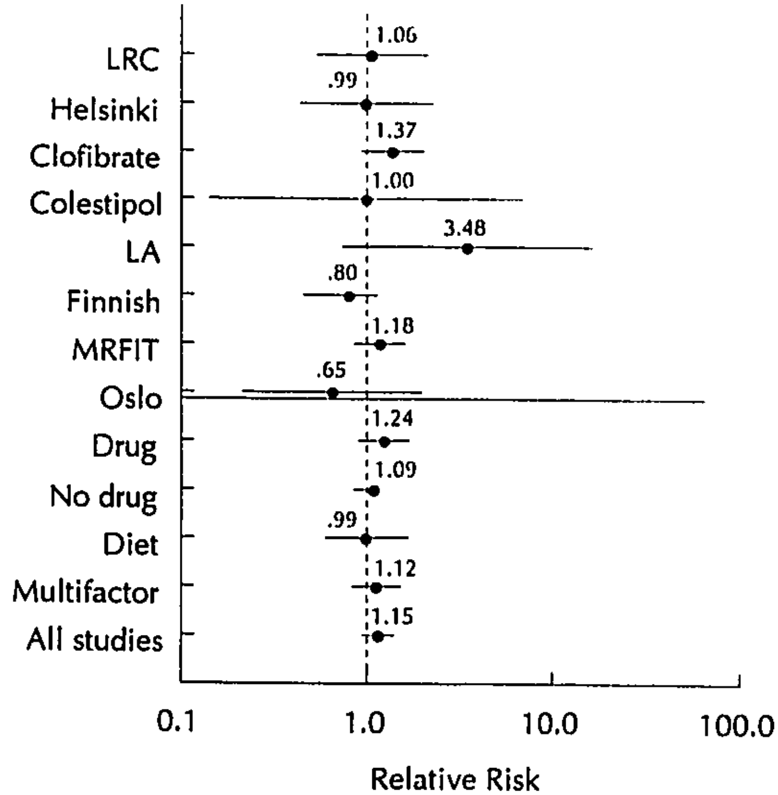

Figure 4. Relative risks of neoplasms in primary prevention studies of cholesterol lowering. Data above line are individual studies, below line are pooled.

LRC = Lipid Research Clinics Coronary Primary Prevention Trial
Helsinki = Helsinki Heart Study
Clofibrate = WHO Cooperative Trial on Primary Prevention of IHD
Colestipol = Upjohn Colestipol Trial
LA = Los Angeles Veterans Trial
Finnish = Finnish Mental Hospital Study
MRFIT = Multiple Risk Factor Intervention Trial
Oslo = Oslo Diet and Heart Study

Drug = LRC, Helsinki, Clofibrate, Colestipol
No drug = LA, Finnish, MRFIT, Oslo, Minnesota
Diet = LA, Finnish
Multifactorial = MRFIT, Oslo

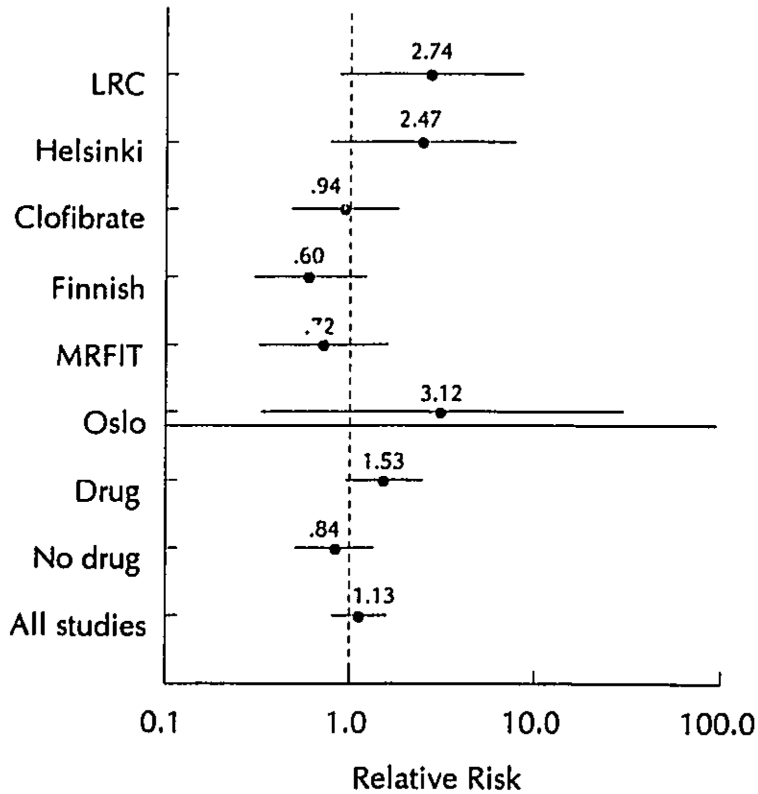

Figure 5. Relative risk of accidents in primary prevention studies on cholesterol lowering. Data above line are individual studies, below line are pooled.

LRC = Lipid Research Clinics Coronary Primary Prevention Trial
Helsinki = Helsinki Heart Study
Clofibrate = WHO Cooperative Trial on Primary Prevention of IHD
Finnish = Finnish Mental Hospital Study
MRFIT = Multiple Risk Factor Intervention Trial
Oslo = Oslo Diet and Heart Study

Drug = LRC, Helsinki, Clofibrate
No drug = Finnish, MRFIT, Oslo

The data on neoplasms and accidents are summarized in Figures 4 and 5 (pages 24 and 25). For neoplasms, the confidence limits of the relative risks of each individual study and all the combinations of pooled studies overlap 1.0. The largest neoplasm rates were in the Los Angeles Veterans trial, a dietary trial, and the clofibrate trial, which also had higher coronary disease mortality rates in the experimental groups. In none of these trials was the increase statistically significant (Figure 4). Fatal accidents were increased in three of the large randomized trials - LRC, Helsinki, and Oslo. Again, the effects were not statistically significant individually or pooled (Figure 5).

Although pooling trials in this combination has not provided real evidence that lowering of cholesterol significantly increases mortality from neoplasms or accidents, combining different groups of studies and different subsets of patients can yield different conclusions.[102] Muldoon and colleagues had more restrictive criteria for their meta-analysis. To be included studies had to be randomized, had to report both total mortality and cause specific mortality and had to intervene only on cholesterol. They analyzed the data only for male subjects on the six randomized trials. The odds ratios for mortality for all causes, CHD, cancer, and accidents were 1.07, 0.85, 1.43 and 1.76 respectively. The latter two were significantly elevated. We estimated relative risks of 0.96, 0.77, 1.15 and 1.13 respectively. Several differences exist between the two meta-analyses. Ours included non-randomized studies. We did not include cause specific data from Minnesota because it was not yet published at the time of our analyses (there was a 14 year lag between the abstract and the publication). Since the mean follow-up time in Minnesota was only a year, it is quite a different study from the others. Sensitivity analysis would need to be performed to analyze the effect of this one study on the results.

The two meta-analyses also differed in some of the raw data used. For example, for the Upjohn Colestipol study, the Muldoon analyses used values of nine and 22 for CHD mortality in the intervention and control groups respectively. We used four and seven. A reexamination of the paper showed that nine and 22 represented total CHD deaths, whereas four and seven represented CHD deaths among men with no CHD at entry to the study. The latter would be more appropriate numbers for an examination of primary prevention.

For the WHO Clofibrate Trial all the numbers differed between the two meta-analyses. Closer examination revealed that we had used the numbers reported as the main endpoints of the trial, namely deaths within the trial and within one year of leaving the trial. Muldoon et al. used only the deaths within the trial. While this gives more favourable results for IHD, it yields higher ratios for neoplasms and accidental deaths. Meta-analyses involve many judgment calls in terms of what to include. As is apparent here, this can lead to divergent conclusions. While the evidence clearly points to a lowering of CHD following reduction of cholesterol levels, the question of whether reducing cholesterol levels leads to an increase in other causes of mortality is bound to remain controversial for some time. Answers will need to come from epidemiological studies and trials designed to test the specific hypotheses.

Evidence for CHD beginning in childhood

The evidence that coronary artery disease has its origins in childhood comes from many sources: epidemiologic evidence, pathologic evidence, familial and genetic evidence, and tracking phenomenon. These have been extensively reviewed by Wynder[103], Kwiterovich[104], Strong and Dennison[105], Cresanta[106] and others and will only be summarized here. The tracking phenomenon, which is a central issue in pediatric screening, will be examined more closely.

The epidemiologic evidence of origins of CHD in childhood is mostly ecologic data that demonstrates high levels of total cholesterol in children in populations with a high incidence of coronary heart disease. Thus multinational studies show the highest cholesterol levels in children from countries with the highest CHD mortality, and the lowest cholesterol levels in countries such as Nigeria, Italy, Greece, Japan, and Kenya, where CHD mortality rates are low.[107,108] Countries that experience dietary changes, such as Japan over the last decade, show increasing levels of cholesterol in the children.[109]

Pathologic evidence of CHD in childhood has existed since the 1921 report of Monckeberg of the "atheromatosis" of the aortic intima of children who died during World War I. Autopsy studies during both the Korean War and the Vietnam War showed fatty streaks and raised lesions in the coronary arteries of 45% to 77% of young soldiers.[110,111] One of the larger pathologic studies was the International Atherosclerosis Project,[112] which examined 4737 autopsied cases, aged 10 to 39 years at death. With increasing age there was a gradual transition from fatty streaks to fibrous plaques. In countries where there was extensive fatty streaking in childhood, there were more extensive raised atherosclerotic lesions in middle age. One other link in the chain of evidence comes from the study of Newman and colleagues[107] who studied the relationship of antemortem cardiovascular disease risk factor levels to early atherosclerotic lesions in coronary arteries. Aortic fatty streaks were strongly related to prior levels of total cholesterol and LDL (r= 0.67), and coronary artery fatty streaks were associated with VLDL (r = 0.41).

The clinical and genetic evidence of origins of CHD in childhood comes from studies that show the association of risk factors in children to risk factors in the parents and to premature CHD in the parents. As described above, these studies have been done in both directions: children selected because of premature CHD in their parents have higher prevalence of hypercholesterolemia[21,113,114] and children with high cholesterol levels have relatives with increased incidence of coronary mortality.[41] Strong correlations between parents and offspring for cholesterol levels have also been shown.[36,41,47,115,116] There is also the clinical entity of familial hypercholesterolemia, an autosomal dominant condition with a gene dosage effect. Familial hypercholesterolemia affects one in 200 to one in 500 children and its effects are completely expressed in early childhood. Heterozygotes have cholesterol levels two to three times normal levels (usually above 7.5 mmol/L), and homozygotes have levels five to six times normal. Homozygous familial hypercholesterolemia is usually clinically evident with xanthomas by the age of five years, and angina and MIs occur in the teens. The heterozygous state is generally asymptomatic in the first decade, and a minority may develop xanthomas in the second decade.[104] Although the prognosis is generally pessimistic for heterozygously affected males, the occasional male heterozygote without premature CHD has been identified. Healthy lifestyle factors might have protected these men.[117]

The tracking phenomenon is the tendency of measurements to follow a pattern whereby early measurements predict later measurements. Tracking refers to "the degree a given measurement tends to retain the same rank order among individuals of the same age."[103] Two major tracking studies over long periods of time have been done in the United States: the Bogalusa Heart Study[107,118,119,120,121,122,123,124] and the Muscatine Study.[34,125,126,127,128,129,130,131]

The Bogalusa Heart Study is an epidemiologic study of risk factors for cardiovascular disease from birth through the age of 26. The study consists of periodic surveys in Bogalusa, Louisiana, a town with a population of approximately 22,000, of whom two-thirds are white and one-third black. The baseline survey took place in 1973-74, when 80% of preschool children (ages 2.5 to 5) and 93% of school children (ages 5 - 14) were screened, forming an initial cohort of 4238 children. Three repeat examinations of the population took place in Years four, six and nine of the study. During these latter surveys, examinations were expanded to include individuals up to age 23. Besides tracking risk factor levels, a local information system was set up in 1978 to obtain permission to autopsy residents who died between the ages of three and 26 years. Since then 87% of known eligible deaths were autopsied. Thus the Bogalusa Heart Study also provides pathological information related to previous risk factor levels.

The Bogalusa study tracked serum total cholesterol, serum triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, very-low-density lipoprotein cholesterol, systolic and diastolic blood pressure, obesity and cigarette smoking. Results can be looked at both in terms of correlations and of the persistence of extreme levels. Correlations among cholesterol levels were highest for LDL cholesterol. At 12 years of follow-up the correlation coefficients for total cholesterol between baseline and follow-up ranged from 0.42 to 0.53 for whites and 0.38 to 0.66 for blacks. For LDL the coefficients ranged from 0.44 to 0.51 for whites and from 0.59 to 0.69 for blacks. Of those children whose baseline cholesterol levels were above the 75^{th} percentile, approximately 50% had cholesterol levels above the 75^{th} percentile 12 years later. The tracking was better for those who were nine to 14 years of age at baseline (55%) than those who were two to eight years old (42%).[121] The best predictor of cholesterol level at 12 years of follow-up was the baseline cholesterol level, with the next best predictor being body mass index at 12 year follow-up. We are thus seeing a combination of hereditary and environmental influences on serum cholesterol. This is further illustrated with the dietary data. Children with consistently high intakes of dietary cholesterol divided by tertiles had significantly increased levels of serum total cholesterol at ages four years (14.3 mg/dl higher) and seven years (13.7 mg/dl higher). Children with consistently high intakes of saturated fat per 1000 kcal had slightly higher levels of serum total cholesterol at ages four years (5.1 mg/dl higher) and seven years (5.4 mg/dl higher), and higher levels of LDL.[118]

The pathological portion of the study related lesions in both the aorta and the coronary arteries to antecedent risk-factor measurements in adolescents and young adults. Rankings for total cholesterol and low-density lipoprotein cholesterol were strongly associated with rankings for aortic fatty streaks but the extent of aortic fatty streaks was not related to triglycerides, high-density lipoprotein cholesterol, systolic blood pressure, or diastolic blood pressure. It did tend to be inversely associated with the high-density lipoprotein ratio. Fatty streaks were also not related to smoking or obesity. The associations of both total cholesterol and low-density lipoprotein cholesterol to the extent of aortic fatty streaks persisted in male subjects of both races but were weaker in white female subjects. There was almost no difference in the magnitudes of associations according to the age at death.[107]

The Muscatine study best addresses the question of using cholesterol measurements in childhood to predict adult hypercholesterolemia.[126] Investigators examined 2367 children aged 8 to 18 between 1971 and 1981 and reexamined them at least once between ages 20 to 30 years. They did not intervene, but informed children and parents of the cholesterol levels. Only those with cholesterol levels over the 95th percentile were told that their levels were high. Fewer than half the children (43%) with cholesterol in the top 10% of the group remained in the top 10% in their 20's, although 81% had levels above the 50th percentile. In comparison, among children with levels below the 90th percentile, only 17% had levels above the 50th percentile when in their 20's and only 3% had levels above the 75th percentile. However, the predictive value of a positive test is still low. In the Muscatine Study there were 1391 children with a cholesterol level below the 90th percentile. If 3% of these have cholesterol levels above 6.2 in their 20's, that yields 42 children. That is more than the 24 children who would have been identified from a positive test (70 positives x 34% with levels above 6.2 in their 20's).[125]

Even if one uses a criterion of 5.2 mmol/L (200 mg/dl) as a cut-point for high cholesterol in the 20's, only half the children will be correctly identified if one uses the 75th percentile level cutpoint for identifying children with high cholesterol levels. About three quarters of the children with cholesterol levels above the 90th percentile will have cholesterol levels over 5.2 mmol/L as adults. Thus although the Muscatine Study shows that childhood cholesterol measurements can predict adults levels, the childhood levels explain only 25% to 50% of the variability of adult cholesterol levels.[126] A portion of the remainder of the variability is accounted for by lifestyle factors such as obesity, smoking and oral contraceptives. Family history of ischemic heart disease was highly correlated with high cholesterol levels. Two conclusions are possible. One is that on an individual basis, prediction is not sufficiently precise to warrant action - we are trying to predict a risk factor for a risk factor and it's too far removed from the final outcome to be important. The other possible conclusion is that since family history correlates well, and that since modifiable risk factors also contribute to adult levels, early counselling and risk factor modification may play an important preventive role in children with a family history of premature ischemic heart disease.

Screening Recommendations: Adults and children

Since 1985 there has been an abundance of recommendations and guidelines concerning the population that should have serum cholesterol measured, how often to screen, how to manage each possible set of results, and what should be the individual and population goals of therapy. Still, very little consensus exists and many issues remain unresolved. One area of greater consensus, although not addressed by all the organizations, is the acceptance of the WHO target of mean population serum cholesterol below 5.2 mmol/L.[132] In terms of general population screening and management there is a tendency for the American recommendations to promote general screening and aggressive management, and for countries with government sponsored health insurance to be more conservative. Thus in Canada, both the Task Force on the Use and Provision of Medical Services in Ontario[77] and the original reports of the Canadian Task Force on the Periodic Health Examination[133] find no evidence for recommending general population screening, and suggest measurement of serum cholesterol in a case finding setting only when other risk factors are present. The Canadian Consensus Conference suggests giving priority to those with risk factors, and measuring cholesterol in other adults as resources permit.[134] New Zealand guidelines also find no justification for general population screening, but recommend measurement of cholesterol as part of regular health exams.[135] Almost all American guidelines recommend periodic measurement of serum cholesterol.[136] The most recent report of the Canadian Task Force recommends screening men between ages 30 and 59.[137]

Screening and follow-up recommendations for cholesterol[a]

	Whom to screen and how often	*Diet therapy*	*Drug therapy*
NIH Consensus Conference 1985[64]	adults q5y if TC < 5.2	TC ≥ 6.2	TC ≥ 6.7
American Heart Association 1987[138]	adults 20-60: TC < 5.2: q5y TC 5.2-6.2: q1-2y	prudent diet for everyone; TC 5.2-6.2: urge adherence to diet	TC > 6.2 unresponsive to intense dietary management or FH
British Hyperlipidaemia Association 1987[139]		TC ≥ 5.2	TC = 6.5-7.8
European Atherosclerosis Society 1987[140]	case finding at first visit	TC 5.2-6.5: dietary advice, correction of RF	
British Cardiac Society 1987[141]		TC ≥ 6.5	TC > 7.8
National Health Foundation of New Zealand 1988[135]	people with RF	advice at TC ≥ 6.0	
National Cholesterol Education Program 1988[136]	adults 20+: TC < 5.2: q5y TC 5.2-6.2: q1y	LDL > 4.1 or LDL > 3.4 if 2 RF	LDL > 4.9 or LDL > 4.1 if 2 RF
Canadian Consensus Conference on Cholesterol 1988[134]	priority: known CHD; family history CHD or FH; HBP, DM, renal failure, obesity; others as resources permit	30+: TC >6.2: AHA II; 5.2-6.2: AHA I LDL >3.4, HDL <0.9, TG >2.3: more intensive	after 6 months adequate trial of rigorous diet modification, failure to reach goals
Ontario Task Force 1989[77]	M 35-59 with ≥ 1 RF M & F 20-69, ≥ 2 RF	advice: ≥ 6.2, < 2 RF therapy: ≥ 6.2, ≥ 2 RF	those with unmodified RF and LDL > 5.5
Canadian Task Force on PHE 1993[137]	men 30-59, others at MD discretion	advice: men 30-59 therapy: M 30-59 TC > 6.85	men 30-59 with TC > 6.85 after diet therapy
US Preventive Services Task Force 1989[142]	middle aged men; q5y prudent in other adults;	≥ 2 RF: TC 5.15-6.15 < 2 RF: TC ≥ 6.2	no RF: TC ≥ 6.2 ≥2 RF: TC ≥ 6.85
American College of Physicians 1989[143]	not more than q5y in healthy adults		

[a]ABBREVIATIONS: AHA= American Heart Association, CHD = Coronary heart disease, FH = familial hypercholesterolemia, HBP = high blood pressure, DM = diabetes mellitus, RF = risk factor(s), TC = total cholesterol, TG = triglycerides, LDL = low density lipoproteins, HDL = high density lipoproteins, q1y = every year

Once a serum cholesterol measurement is performed, the subsequent interpretation of the result and the management of the patient become important issues. Guidelines for patient management have become increasingly complex, since they often rely not only on total cholesterol measurement but also LDL, HDL and triglyceride values. The recommendations for adults are summarized above.

Although many of the earlier consensus reports on screening for cholesterol did not include recommendations for children, several recent reports have done so, and this year the National Cholesterol Education Program in the United States published an expert panel report on blood cholesterol levels in children and adolescents.[144] The major reports with discussions of children are the NIH Consensus Conference[137] (1986), the policy statement of the European Atherosclerosis Society[140] (EAS) (1987), the conclusions of the British Cardiac Society Working Group on Coronary Prevention[141] (1987), the American Academy of Pediatrics[145] (AAP) recommendations (1989), the Committee on Atherosclerosis and Hypertension in Childhood[146] (1986), and the National Cholesterol Education Program (NCEP) Expert Panel Report[144] (1992). There has been considerable agreement among these diverse committees in terms of the identification of children with elevated blood cholesterol levels. Except the American Academy of Pediatrics, which had some reservations, they all agree that atherosclerosis prevention should begin in childhood. They all advocate a population approach to healthy lifestyles and diets in children, with dietary recommendations similar to those in adults. They all recommend an individualized approach to identifying children and adolescents at greatest risk; this generally means screening those whose parents or grandparents had a history of premature coronary heart disease and may include screening children with a parent with hypercholesterolemia. There is some discrepancy in the age at which they recommend that the prudent diet should begin: The NCEP and the AAP suggest two years of age, the British Cardiac Society recommends age five, and the EAS recommends as early as possible.

The recommendations to screen children with a positive family history can be seen as a compromise, the likely result of committee deliberations. This compromise position has been attacked from both sides - by individuals favouring universal screening and by those opposing any screening in childhood. These controversies, with the arguments on both sides, will be discussed in the next section.

Controversies in cholesterol screening in childhood

The controversy concerning which populations should undergo cholesterol screening involves many interrelated issues.[147,148,149,150] A "high risk" approach to cardiovascular disease prevention, in which the small proportion of the population at highest risk of disease is identified and treated, may have a limited impact on a problem such as ischemic heart disease whose risk factors are so prevalent.[151] Accordingly, programs that have used this approach, such as in Goteborg[152] and one community of the Stanford Three Cities[153] project, could not show sustained differences at the community level in incidence of cardiovascular disease.

The "population" approach attempts to shift the risk factor profile of the entire community in a favourable direction. However, even population approaches have often used risk factor measurement as motivating factors in the general population strategy. This is usually justified based on behavioral change models. In many of the behavioral change models, perceived susceptibility to disease is an important factor in determining the likelihood of taking action (the Health Belief Model,[154] the Theory of Reasoned Action,[155] Subjective Expected Utility Theory,[156] Protection Motivation Theory[157]). Perceived susceptibility to disease may well be influenced by risk factor measurement and awareness, and thus can form the basis of motivating change.

There is, however, a down side to screening. Significant side effects are possible from labelling an individual "high risk".[158,159] Possible adverse effects in those with elevated measures might include the psychological consequences of labelling, iatrogenic disease, and physical consequences of increased stress resulting from labelling, and the costs and inconvenience of regular medical care. Although recent studies of labelling effects have found that strategies using positive, supportive approaches can avoid negative consequences,[158,160,161,162] patients may proceed to act without return to medical care. Knowledge of "normal" measures may also have negative consequences by leading to the encouragement of behaviours which increase the individual's risk of disease. Since, with many risk factors, risk of disease increases continuously with risk factor level, there is reason for those in the "normal" range to follow prudent diets and lifestyles as well.

The question of whether children should be routinely screened for high blood cholesterol is especially controversial. Four major positions on cholesterol screening have been advocated: 1) There should be no cholesterol screening of any children. 2) Children with a positive family history of cardiovascular disease should be screened. 3) Children of parents with high blood cholesterol should be screened. 4) There should be universal cholesterol screening. Each of these views has its proponents, with the second and third statements sometimes combined into one position. This issue is currently being hotly debated in the literature. This section will outline the major arguments for and against the various positions.

Those who argue for cholesterol screening[64,144,163,] offer the following arguments:

- Without an objective measure of abnormality, people are less likely to make dietary changes.[144]
- Measuring total cholesterol serves as an introduction for the pediatrician to counsel families about nutrition and lifestyle. One can educate families about cholesterol, but also teach them that cholesterol is not the only important lifestyle factor.
- The evidence for cholesterol screening is sound: There is autopsy evidence of the precursor atherosclerotic lesions coronary artery disease in adolescents. Coronary artery disease is related causally to cholesterol. And there is significant tracking of serum cholesterol through childhood and from childhood into adulthood. (These arguments are disputed below.)
- Although tracking is not perfect, by far the majority of identified youths (the exact numbers depending on the criteria used) will have cholesterol levels in adulthood over the 50th percentile, which is where most CHD deaths occur.
- Waiting to treat elevated cholesterol levels in adulthood has several disadvantages: Lifestyle behaviours are more easily changed in children that in adults. Young adults do not necessarily have routine primary medical care.
- Healthy life-styles initiated in youth are more likely to be continued in adulthood than attempts to alter habitual activity in adulthood.

- Recidivism approximates 90% in most studies of attempts to alter adult behaviors, such as diet for obesity. It would be far easier to establish good habits in children than to try and change them as adults.
- Children with severe inherited lipid problems need early evaluation and follow-up. We need to screen to detect these children.

In the last few years, there have been many proposals that all children be screened for high blood cholesterol levels.[103,106,164,165,166,167] Those who argue for *universal* screening offer the following additional arguments:[168]

- Family history identifies only one third to one half of children with very elevated cholesterol levels. In fact, because of the lack of routine primary medical care in some adult populations, childhood screening can be used to identify parents who are unaware of their elevated cholesterol levels.
- The cost of screening is only a tiny percentage of the amount of money spent on cardiovascular disease.
- Labelling will not be such a problem when current health promotion programs lead to the more general population adoption of the prudent diet. The child not eating properly would then be labelled instead of the other way around.
- Failure to thrive on low fat diets represents overzealous restriction by parents without medical supervision. If screening and counselling were to occur in the physician's office, there would be opportunity to educate in this regard.
- We need universal screening because the necessary societal lifestyle changes have not yet taken place.

On the other hand, the arguments against universal screening are many:

- The cost of screening is high and just not worth it. Only in middle age men is the cost of screening and treatment thought to approach the cost effectiveness of other preventive maneuvers. Even in young adults the costs far exceed those of other preventive maneuvers.[141] In children the costs would be totally out of line with other preventive interventions.
- What are the effect on the children and the cost to families of those who are either false negative or false positive? Labelling of children as "high" risk could have longlasting adverse effects. On the other hand, false negative labelling could also lead children to believe that they need not heed the universal health promotion recommendations.
- The tracking of cholesterol levels from childhood to adulthood is inadequate to make screening worthwhile. As seen above, the Muscatine study showed that a 16 year old with a cholesterol level above the 90th percentile has only a 30% chance of having that high a level at 28.
- Cholesterol measurements performed outside research settings are likely to be less accurate, therefore even less predictive.[169]
- What is the evidence that diet modification works, that the final outcome will be better and no adverse outcomes will result from the proposed modifications?[170]
- We do not have tests with adequate sensitivity and specificity.

These arguments against universal screening can be expanded to argue that no screening should take place. Despite the evidence reviewed above, there are many steps - perhaps too many - between childhood cholesterol and adult coronary heart disease. There are also many areas where knowledge is lacking. Those who argue against any screening play up the incompleteness of the evidence. They argue that:[171]

- Although serum cholesterol is considered a "risk factor" for cardiovascular disease, no causal relationship has been established - it may only be a "marker" for increased risk of cardiovascular events.
- Fat and cholesterol in the diet may not be the determinants of serum lipids. Those who argue this point often quote the high fat diets of the Greenland Eskimos, who have very little coronary artery disease.[171]
- There is no real evidence that risk reduction is more effective if begun in childhood.
- Present recommended levels of cholesterol may be incorrect.
- Laboratory determinations may be wrong. Office measurement of cholesterol is particularly problematic. On a reference standard of 262 mg/dL, present commercially available equipment yields results anywhere from 187 to 379 mg/dL.[171]
- Treatment of those with high levels is not clear.
- Instead of leading to a discussion of other risk factors, measuring serum cholesterol focuses attention only on the one risk factor and may lead to inadequate modification of the other factors. This argument has been used on both sides of the debate. Those who argue in favour of screening maintain that measurement of cholesterol is a terrific lead-in to discussion of other risk factors.
- The fact that only one of three children with high cholesterol levels come from families with a positive history of cardiovascular disease[172,173] may suggest limited association between high cholesterol in childhood and later coronary heart disease.[174]

(Or it may suggest a remarkably high level of association, but still leave room for other factors to play a role.)

- Cholesterol reduction is effective at preventing hypercholesterolemia-related CHD, even when not begun until middle age, so why start so early?[171]
- One meta-analysis of primary prevention trials has shown a significant increase in injury and cancer deaths - enough to offset any decrease in CHD deaths caused by reduced cholesterol levels.[102] Even in high risk middle age men the two causes of death even out. Since injury deaths are far more common in children and young adults than CHD deaths, if these results are generalizable, cholesterol intervention might lead to an increase in total mortality. Although the extrapolation from adults may not be valid, it can be argued that it is the same type of extrapolation that proponents of cholesterol testing use in suggesting the benefits of screening and treatment.[175,176] Of course, as we have seen above, the meta-analysis may not be entirely valid either.
- The adverse effects of labelling and of possible family conflicts may be longlasting and serious.[175] The evidence is poor that screening and intervention can be accomplished with little risk to the child. Of 40 children referred for dietary treatment of high blood cholesterol levels found on routine screening,[177] 8 (20%) had evidence of growth failure due to unsupervised application of low-fat, low-cholesterol diets recommended by pediatrician. Thus medical recommendation of diets is no assurance that there will not be overzealous application.
- Poor compliance with follow-up recommendations may lead to false positive labelling, with no opportunities for benefits. Lannon and Earp recommended follow-up lipid panels and dietitian advice for children whose total cholesterol was above the 75th

percentile.[178] Only 47% returned for the recommended lipid panel, and only 29% consulted with the dietitian. Much of the "non-compliance" was appropriate: 73% of children with screening cholesterol values > 199 mg/dL returned for lipid panels, but only 37% of children with screening cholesterol values between 176 and 199 mg/dL. However, this may further increase the false labelling effect. Labelling was a definite issue among the parents, as a third of the noncompliant parents were concerned that confirmation of high cholesterol levels would cause their children to worry too much. Anecdotal evidence of excessive concern from the single initial measurement was cited as well.[178]

- While knowledge of elevated cholesterol levels may not lead to dietary change, knowledge of "normal" levels could lead to the encouragement of behaviours which increase the individual's risk of disease. Since risk of disease increases continuously with risk factor level, there is reason for those in the "normal" range to follow prudent diets and lifestyles. So why screen?

If universal screening is not thought to be warranted, then any screening advocated would be based on family history. One can further argue against the family history approach that family history is frequently inaccurate[179] and that it has a low predictive value. Depending on the cutpoints, this approach will either miss a high percentage of children or will label everybody.[180,181,182,183]

In Canada there has not been support for universal screening even among adults. The question of universal screening in childhood would thus seem rather moot. The Toronto Working Group on Cholesterol Policy[77] and the Task Force on the Periodic Health

Examination[137] have questioned even the screening of males in the 20 to 29 year age range with a family history of premature heart disease and the screening of women at any age. The question of any pediatric screening would then be totally irrelevant. Yet, for many, questions remain about whether children should never be tested, or whether children at high risk because of positive family history should have a cholesterol measurement. The present study attempts to shed light on this dilemma by exploring the age groups of offspring of patients with recently identified coronary heart disease, and the changes being made in the presence and absence of cholesterol testing. Dietary and cholesterol modification is not the only behaviour change needed in these children. Smoking prevention is of vital importance. Does measurement of cholesterol and identification of an additional risk factor aid in the primary prevention of other risk factors? Or does it focus too much attention on the one factor to the detriment of the modification of other factors?

Are screening recommendations followed?

Widely publicized disagreements among members of the scientific community and among the recommendations of professional societies[184,185] may be responsible for some of the confusion and divergence of practices in this area of medicine. In addition there is confusion between the recommended approach to the identified high-risk patient with hypercholesterolemia and the approach to the general population. American studies have shown that among pediatricians, 90% routinely inquire about familial cardiovascular disease history,[186] but fewer than half measure serum cholesterol in most of their high-risk patients. In line with American recommendations, 90% of the primary care pediatricians did not include testing of lipid levels during well-child visits unless the child was at high risk. But only 70% counsel against smoking or give dietary advice.[187] In one American study 48% of physicians required an exercise history as part of the initial examination and 91% encouraged their patients to participate in regular exercise programs. However, only 3% had any training related to prescribing exercise.[188] Examining the management of elevated cholesterol levels by New Hampshire primary care physicians,[189] Vivier et al. found that 37% of family physicians and 48% of internists assess cholesterol levels at least once every five years in asymptomatic 20-39 year olds. Many physicians were uncertain about what approach to take in the pediatric age group.

A slightly earlier study of family practice residents' compliance with preventive medicine recommendations[190] showed that only 6% screened for serum cholesterol at least once every five years.

In Canada, there have been several surveys of cholesterol management by family physicians. In an Ottawa telephone survey of family physicians, 56% claimed to routinely measure serum cholesterol levels in all patients over the age of 30 years.[191] However, the average cholesterol level at which physicians began dietary therapy was 6.95 mmol/L (270 mg/dl). Tannenbaum and colleagues[192] found in a national mail survey that physicians were conservative in their practices - more so than recommended by the Canadian Consensus Conference on Cholesterol. In the 20 to 39 age group, over half the physicians reported measuring serum cholesterol on fewer than half their patients who presented with one risk factor. Close to a third measured levels on fewer than half their patients who presented with more than one risk factor. Not everyone in this age group sees a physician. What proportion of offspring of patients with premature CHD are being missed in terms of screening and counselling by the present approaches of family physicians? The present study sought to ask the high risk family members themselves.

Interventions in Youth and High Risk Families

Screening is only worth while if there are effective interventions. Since CHD is multifactorial, the interventions also have to address multiple risk factors. The impact of intervening on cholesterol in middle aged men was reviewed above. In younger age groups, the time lag is too long, so that intermediate endpoints are generally used - amount of reduction of cholesterol levels or proportion who do not start to smoke. Several school and community based Heart Health programs have evaluated their programs. Programs such as the North Karelia Youth Project,[193] the Oslo Youth Study,[194] the International Know Your Body Studies,[195] the Heart Smart Program,[196] and the SEGEV Program[197] have used combinations of high risk and population approaches, school and/or community and/or medical delivery vehicles, individual change strategies and environmental change strategies. In general they have modified knowledge, dietary and smoking practices, and have had only small (but measurable) effects on serum cholesterol. Community wide Heart Health Programs, such as in Pawtucket,[198,199] Minnesota[200,201,202,203] and Stanford,[204,205] have recognized that behaviours associated with increased risk, such as eating patterns, exercise and smoking are well formed by adulthood. Children and adolescents not only constitute a large proportion of the population, but also strongly influence the health behaviours of their families. Thus these programs have included major components directed at youth, their families and the school system.

Although it is often impossible to segregate intervention strategies with the most impact, several studies have evaluated family components separately. In North Karelia, besides the community and school-based programs, two family-based studies were performed.[193] During a 12 week intervention period, the families' diets were changed so that the proportion of fat in the diet was reduced from 35% to 24%. Cholesterol levels decreased by 15%. This compares to the less than 3% reductions achieved with the community wide programs. In Minnesota the addition of a home-based component to the school program also had a significant beneficial effect on dietary patterns. Thus there are some suggestions that family based programs can have an impact when they accompany larger community programs.

Families of CHD patients should be even more prepared to adopt risk-reducing behaviours. Patients themselves quit smoking at high rates (43%) following a myocardial infarct.[206] Do intervention programs in this setting work? One recent study of adolescents was not particularly encouraging, but several circumstances could have accounted for the results. Walker and colleagues in Australia invited MI patients with teenage children to participate in an intervention trial.[207] Of the 192 families that were approached, 95 refused to participate. The remaining 51% were randomized, with half allocated to an early intervention group and half to late intervention (after one year). A further 43 families of patients with other diagnoses formed a second control group. Seventeen complete families and fourteen additional teenagers withdrew before 12 month outcomes were measured. The intervention program consisted of three home visits, a group discussion session, follow-up telephone calls and letters and a 6-month screening session. After one year, self-reported exercise participation and smoking rates did not change in any of the groups. Teenagers in the early group reported reduced intakes of total and saturated fat compared with teenagers in the late group, but as there was no difference in cholesterol levels, this may have been false reporting. There were differences in cholesterol levels between the control group and the late intervention group, suggesting that the participating families were already highly motivated. There were no changes in self-reported exercise or smoking rates in any of the groups. This is a small study with a high refusal rate, a high dropout rate and a relatively weak intervention. It may well be a pre-selected group from whom the results are not generalizable. Although other studies have shown more beneficial effects when addressing the family unit, during the adolescent years teens may be less amenable to an intervention given solely at home. Clearly the ideal intervention strategies in this high risk group have yet to be worked out. But first we need to know what is happening to them now.

Conclusions

Using decision analysis, Krahn and colleagues have compared the aggressive US strategy and the conservative Canadian policy for cholesterol screening and treatment.[208] The result was a "toss-up" - the number of expected quality adjusted life years was similar for both programs at all time intervals. However, interventionist programs are more costly. Thus, there may be advantages to the conservative approach of the Canadian Task Force on the Periodic Health Examination of only doing cholesterol screening in men ages 35 to 59 and in others at high risk. The literature reviewed here has shown that early onset ischemic heart disease clusters in families and increases the risk of the offspring and that even the most conservative approach to screening for cholesterol recommends screening in these high risk families. And although a high risk approach may not have a large impact on the overall incidence of ischemic heart disease, whose risk factors are so prevalent, there is a large potential impact for high risk families and for the incidence of premature coronary disease. But does this approach work? Are Canadian high risk individuals being identified and counselled? Are these high risk offspring aware of their risk factors and do they know what they can do about them? The present study was designed to answer these questions.

Objectives

The objectives of the study were to determine:

1. The prevalence and awareness of risk factors in offspring of patients with premature ischemic heart disease (and to compare when possible to general population)
2. The proportion of offspring who have had risk factor assessment including cholesterol screening, and the proportion who have adopted risk factor reducing strategies (non-smoking, exercise, diet), both in the presence and in the absence of testing
3. Determinants of variation in offspring practices
4. Risk factor awareness and behavioral changes in patients with premature coronary disease, and determinants of those behavioral changes.

Design and methods

Design

We conducted a telephone survey in a random sample of subjects with premature ischemic heart disease and their offspring. The questionnaire solicited information first from the patients with heart disease about risk factors for IHD, awareness of cholesterol levels, recent lifestyle modifications, and demographic features. We then asked permission to conduct a similar interview of one offspring in each family. The offspring from each family greater than two years of age who, at the time the interview was conducted, had had the most recent birthday was selected for interview. Offspring over the age of 16 years were interviewed directly while the parent most familiar with the child's habits responded for children less than 16 years.

Study population

Patients with premature IHD who lived in the Ottawa-Hull metropolitan area, had a known telephone number, and had a myocardial infarction or positive angiogram between January 1, 1988 and February 28, 1990 were eligible for the study. Males were eligible if they were less than 50 years of age and females if they were under 60 years. To qualify as a myocardial infarction, patients required a new Q wave on EKG or a positive CK with MB fraction greater than 5%. To qualify as a positive angiogram, there had to be a stenosis of at least 50% in at least one major coronary artery, as read by the cardiologist who performed the angiogram.

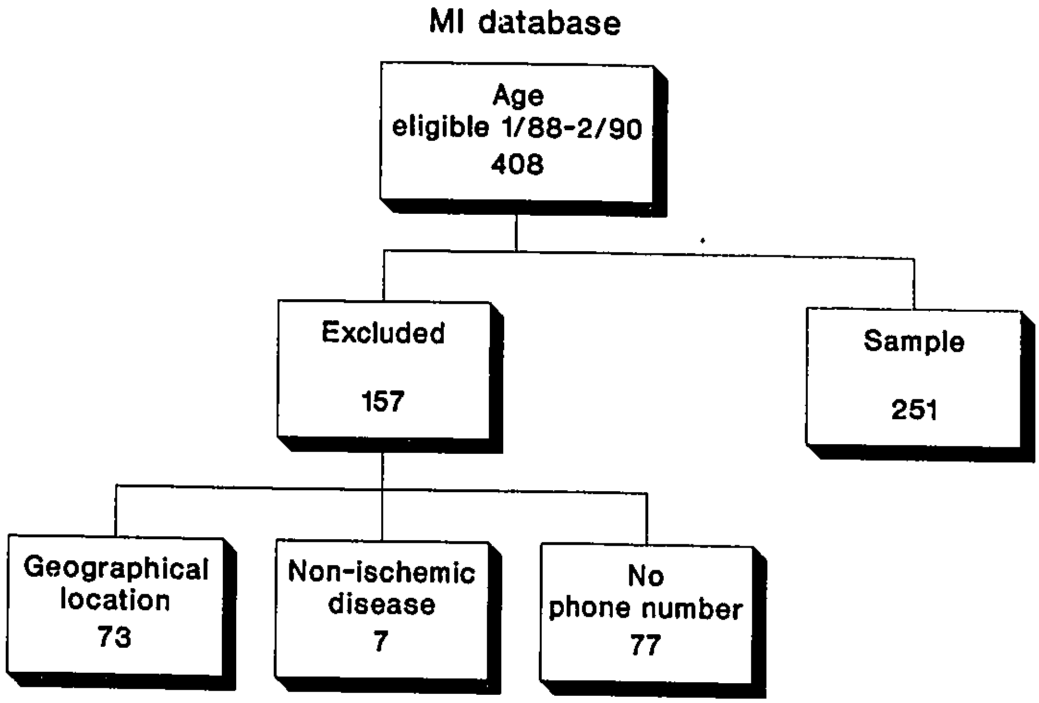

Figure 6. Flow charts of sample selection, eligibility and exclusions from the MI database.

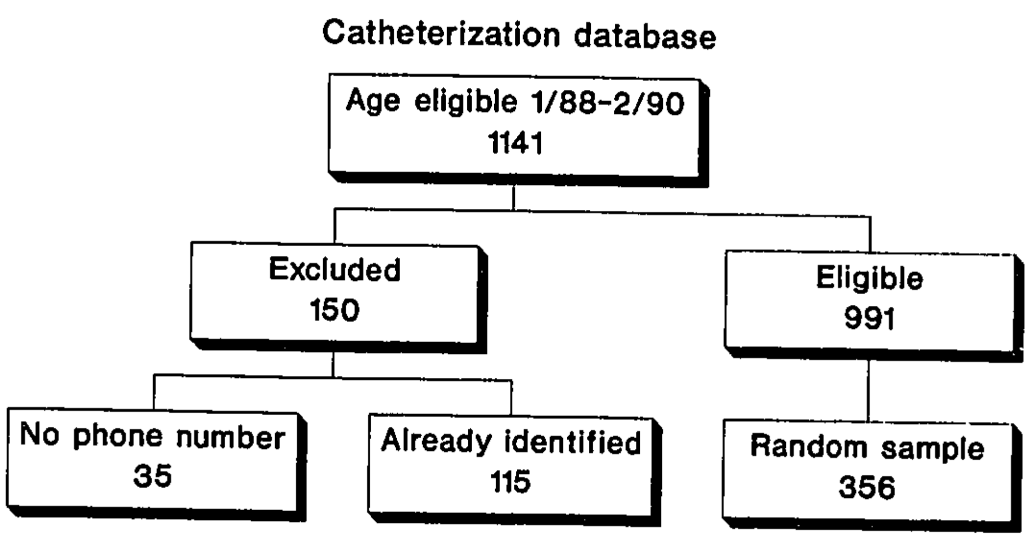

Figure 7. Flow chart of sample selection, eligibility and exclusions from the catheterization laboratory database.

We identified the study population using two local databases. As a participating centre in the Cardiac Arrhythmia Suppression Trial (CAST), the University of Ottawa Heart Institute identifies most patients in the Ottawa-Carleton area with a new myocardial infarction (MI), and maintains a database of patients screened but excluded from CAST. This constitutes over 90% of the MI population since CAST eligibility required more than six premature ventricular beats per hour on a 24 hr. Holter monitor as well as an ejection fraction of less than 40%. The second database constitutes the computerized records from the University of Ottawa Heart Institute's cardiac catheterization laboratories. The Heart Institute carries out coronary angiography on approximately 4000 patients per year.

The MI database included a total of 1879 patients whose MI's occurred between January 1, 1988 and February 28, 1990. We excluded 1471 who did not meet the age eligibility criteria, and a further 157 for the following reasons: inaccessible geographical location (73), clinically significant preexisting nonischemic cardiac conditions (7) and lack of telephone number (77). We attempted to contact the remaining 251 patients (Figure 6).

The Catheterization Laboratory database included 1141 age and angiogram eligible patients during the same period. Thirty-five lacked a phone number and 115 had already been identified from the CAST database. From the remaining 991 we selected a random sample (Figure 7) using a computer-generated list of random numbers: Patients were assigned consecutive numbers via automatic numbering. EpiInfo was used to generate the random numbers. Patients were selected if their number appeared on the random number list. At the end of each patient interview, patients were asked for the names and birth dates of all their children. The interviewer selected the offspring with the birthday closest to and preceding the date of the interview and asked for permission to contact that offspring if over 15 years of age. If the identified offspring was under 16 years of age, the interviewer asked which parent would be most familiar with the child's diet, and asked for a further interview with that parent. A questionnaire was completed on only one offspring per family.

Questionnaire

The questionnaire was developed for this study using some original questions, but mostly questions previously validated in larger health surveys including the US Health and Diet Survey, the Ottawa-Carleton Heart Beat Survey,[209] the Ontario Health Survey, the Health Promotion Survey,[210] the Ottawa Carleton Worksite Survey and the Ottawa Carleton Community Health Survey.[211] We specifically sought questions from Ottawa Carleton surveys so that we could compare the results of high risk individuals to the general population in the same community.

We designed three questionnaires: one for the index patient, one for offspring over age 15, and one for interview of the parents about their children under age 16. The patient questionnaire consisted of standardized short answer questions on general health, family history of heart disease, dietary habits and changes in the previous two years, cholesterol screening attempts at modification, stress, weight and weight loss, hypertension, smoking demographic data. The questionnaire for the offspring over 15 was similar, but added knowledge questions on the causes of heart disease and high cholesterol, potential lifestyle changes if the cholesterol level were known and physical exercise. The questionnaire for the children under 16 was more limited in scope. The topics were similar, but detailed data on smoking and weight were not sought. (See Appendix B for English version of questionnaire.)

The translation department of the Children's Hospital of ' astern Ontario translated the questionnaire into French. The French questionnaire was reviewed by the investigators, and pretested on health care professionals for clarity, content coverage and face validity.

Eight interviewers (three medical students, five medical secretaries) were trained and conducted the interviews over a twelve week period beginning in July 19Ṣ'0. Afte: familiarizing themselves with the questionnaire, interviewers practised interviewing the investigators both individually and in groups. The entire group, including investigators, would code responses to ensure consistency among interviewers. After the first several telephone interviews, completed forms were collected and reviewed by the investigators for correctness and completeness of coding. Since interviewers were permitted to make calls from home, there was no direct supervision of the actual interviews.

Each interviewer received a questionnaire booklet, containing copies of the three colour coded questionnaires in both English and French; colour coded coding forms for recording answers (in English only); and patient log sheets. The patient log sheets were preprinted with the name, phone number and ID number of the patient whom they were to contact. On this sheet the interviewers recorded the interviewer name, the date of completion of the parent and child interviews or the reasons for non-completion, the dates and times of all calls attempted, the names and phone numbers of children not living at home, and the name of the child with the most recent birthdate. Interviewers were given additional batches of patient log sheets as they satisfactorily completed earlier ones.

Six telephone calls at varying times of day over a two week period were attempted before eligible individuals were regarded as unreachable. All attempts were logged. In the interests of confidentiality, no effort was made to locate through the phone book new phone numbers of patients with wrong or no longer listed numbers if there was more than one listing under that name (the majority of cases). Completion of each parent interview required approximately 10 minutes, while interviews with offspring over age 15 took about 15 minutes.

Sample size calculations

The chief outcome measure in this study was to be the proportion of offspring who had had blood cholesterol measurements done. We estimated this proportion to be 25%. To be 95% confident that the true proportion of offspring who had cholesterol measurements done would be within 5% of this estimate, we calculated that we would need a total of 300 respondents (from both databases combined). We estimated the non-response to be 50% (20% with no offspring, 20% unreachable and 10% refusal rate). We thus decided that we would need to approach 600 patients to obtain 300 interviews.

Data entry and analysis

Data were entered into the computer using EpiInfo (version 5), then converted, using DBMSCopy (Conceptual Software) to the statistical package SYSTAT (Systat Corporation, Evanston, IL) for analysis. Comparisons between groups were conducted using contingency tables and chi-square statistics for categorical variables and two-tailed t-tests or analysis of variance for interval or continuous data. The Fisher exact test was employed for categorical analysis when warranted by small cell sizes. Chi square tests for two by two tables are Yates corrected unless otherwise stated. Because of the large number of comparisons, only p-values < 0.01 were considered statistically significant. However, p-values below 0.05 are identified as trends and relative risks (also called prevalence ratios when used to compare prevalences of risk factors in males and females) with 95% confidence intervals are used to compare males and females on certain factors. Logistic regression analysis was employed to examine determinants of cholesterol measurement in offspring and determinants of attempts at risk factor modification. This analysis was performed using SYSTAT's Logit Module.

Definitions

The following risk factors for CHD were asked about in the survey: blood pressure, cholesterol, smoking, exercise, obesity and family history of heart disease. Patients and offspring were considered hypertensive if they reported that they were ever told by a physician, nurse or other health professional that they were hypertensive. Cholesterol was similarly considered a risk factor on the basis of such self report, but patients were also asked to specify the level of their last cholesterol measurement. However, these values were not used to classify patients. Respondents were considered never smokers if they had smoked less than 100 cigarettes in their lifetime. If they had smoked at least 100 cigarettes and reported that they presently smoke "not at all", they were considered ex-smokers. Offspring were considered to be at risk from smoking if they smoked at the time of the survey. Patients were considered to have had smoking as a risk factor for their coronary disease if they were smokers or had quit less than five years previous to the study. Exercise was assessed by the question "How many times per week do you exercise for at least 15 minutes?". (It was described to the respondents as vigorous activities such as calisthenics, jogging, racquet sports, team sports, dance classes and brisk walking.) Lack of exercise was considered a risk factor if respondents answered that they exercise less than three times per week. Body Mass Index (BMI) was calculated as weight (kg) / height^2 (m). A BMI of 20 to 24.9 is considered optimal, 25 to 26.9 is moderate overweight, and 27 or greater is considered to be overweight that may have adverse health consequences. Patients with premature CHD were considered to have a positive family history if they identified close relatives (i.e. brothers, sister, parents, aunts, uncles, grandparents) who had had a heart attack under the age of 55. The same cutpoint was used for males and females to facilitate answers.

Ethical considerations

We obtained permission from all of the cardiologists at the Heart Institute to contact their patients by telephone. We did not attempt to contact anyone whose phone number was not available on file, or whose number had changed. Interviewers had no information about the people they were calling except their names and phone numbers and the obvious fact, given the study, that there was some history of cardiovascular disease. Interviewers were instructed in confidentiality, although, being medical students and medical secretaries, they were already privy to similar confidential information. Verbal consent was obtained from the patient before the interview proceeded. Confidentiality was maintained by assigning each potential respondent a unique identifying number. The log sheets containing names and phone numbers were kept separate from the data entry forms, and only the ID number was recorded on the data entry form. Ethical approval for the study was obtained from the Research Ethics Committee of the Ottawa Civic Hospital.

Results

Description of the study population

From the selected sample of 607 patients, results are reported on 318 completed patient interviews. A total of 297 interviews were completed both by the parent and the child, 21 interviews were completed by the parent only and one interview was completed by an adult child of a deceased patient. Of the 298 offspring interviews, 219 were interviews on offspring 16 and over (interviewed personally) and 79 were on younger children (interviewed through the parent).

Figure 8 shows the response rates and reasons for non-response. Seventy-three patients were excluded because they had no children (57) or were deceased (16). Two patients were found upon interview to be age ineligible. Non-response was due to lack of a phone number (number not in service, wrong number: n = 120), refusal to participate (n = 48) no answer despite six attempts (n = 29) and a variety of miscellaneous reasons each accounting for fewer than five cases (eg. parent or child in hospital, inability to speak English or French, on holidays).

We compared respondents, non-respondents and ineligible patients from both databases regarding age and gender. In the MI database we also had information on history of smoking, diabetes, congestive heart failure (CHF) and a previous MI. Table 1 compares these values. For patients selected from the catheterization database, we had data on the presence of left main coronary artery disease, left ventricular class, and the number of vessels

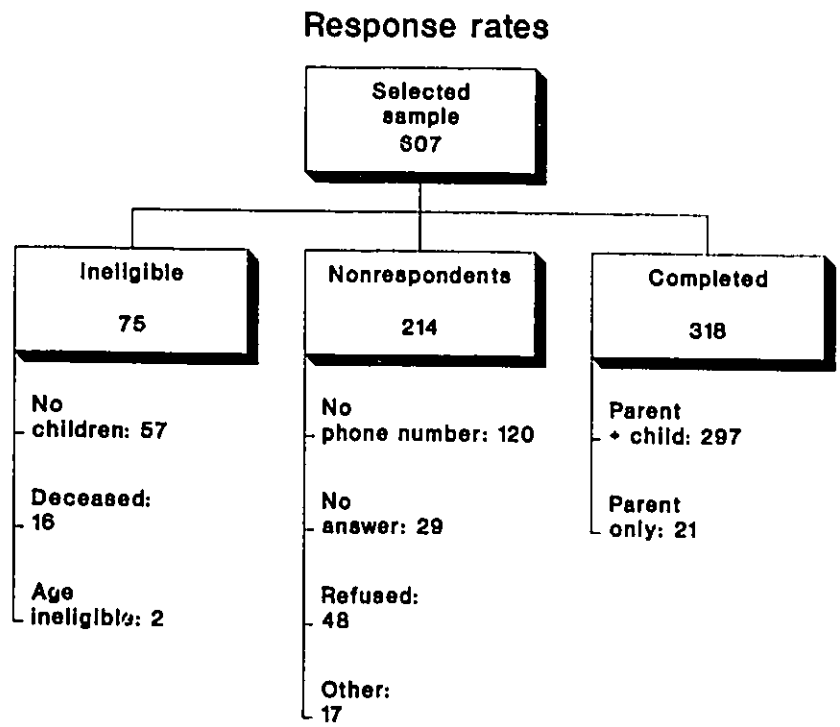

Figure 8. Response rates, exclusions and reasons for non-response. One child of a deceased parent was also interviewed.

Table 1. *Comparison of respondents and non-respondents from the MI database (CAST exclusions).*

	Completed Interview *N = 122**	Ineligible *N = 38***	Non-respondent *N = 89*	χ^2	p-value
Age: mean ± SEM*	45.4 ± .39	45.4 ± 1.3	44.4 ± .83		0.613
Gender: % male	71.3%	63.2%	70.8%	0.97	0.616
Current smokers	57.9%	56.8%	65.1%	1.33	0.514
History diabetes	12.6%	13.6%	11.6%	0.07	0.965
History CHF*	0.84%	5.3%	1.2%	3.63	0.163
Previous MI	13.3%	15.8%	16.7%	0.46	0.793

*Completed interview = completed parent interview, SEM = standard error of the mean, CHF = congestive heart failure, MI = myocardial infarction

**Does not include the two patients with completed interviews who were age ineligible.

affected. We compared respondents to both non-respondents and ineligible patients (Table

2); and the randomly selected sample to those not selected (Table 3). The groups did not differ with regard to either the prevalence of self reported risk factors or angiographic measures of heart disease severity.

Table 2. *Respondents and non-respondents from the catheterization lab database.*

	Completed Interview *N = 196*	Ineligible *N = 35*	Non-respondents *N = 125*	χ^2	p-value
Age: mean ± SEM	47.1 ± .45	45.9 ± 1.46	46.1 ± .6		0.361
Gender: % male	61.2%	62.9%	61.6%	0.03	0.983
3 vessel disease	27.7%	31.4%	34.4%	1.64	0.441
LV Class 3 or 4*	13.3%	22.9%	17.6%	2.57	0.277
Left main disease	2.0%	2.9%	2.4%	0.11	0.946
LVEDP > 20*	31.1%	34.3%	34.4%	0.42	0.809

*LV = left ventricular; LVEDP = left ventricular end diastolic pressure

Table 3. *Comparison of patients randomly selected from the catheterization lab database to those not selected.*

	Randomly selected *N = 356*	*Not selected* *N = 785*	χ^2	*p-value*
Age: mean ± SEM	46.5 ± .37	45.9 ± .25		0.153
Gender: % male	61.5% (55.4-67.2)	67.4% (63.5-71.1)	3.48	0.062
3 vessel disease	30.3% (24.3-36.7)	31.2% (27.1-35.5)	0.10	0.750
LV Class 3 or 4*	15.7% (11.6-20.5)	17.2% (14.3-20.4)	0.28	0.596
Left main disease	2.3% (0.8-4.7)	4.1% (2.6-5.9)	1.91	0.167
LVEDP > 20*	32.6% (27.0-38.4)	28.8% (25.2-32.5)	1.50	0.220

*LV = left ventricular; LVEDP = left ventricular end diastolic pressure

1. *Patients with premature ischemic heart disease*

Description of population

We interviewed 318 patients with premature coronary heart disease: 204 men (64.2%) and 114 women (35.8%). Since the cutoff age for men was lower (50 years at diagnosis) than for women (60 years), the mean age of the men was lower (45.9 years vs. 53.2 years: For age distribution see Appendix A, Table A1). Eighty-five percent of the respondents were married, 5% separated, 5% divorced, 4% widowed and 0.6% were single. Thirty-five percent had some post-secondary education. The language of interview was English for 247 respondents (80.5%) and French for 60 respondents (19.5%). There were significant differences between male and female respondents in educational level (RR of no university education for females = 2.5: CI 1.4 - 4.4), marital status (no men were widowed, 11 women were) and language of interview (26.8% of women were interviewed in French vs. 15.3% of men, p = 0.01). English respondents were more likely to have attended university (25% vs. 8%, p = 0.005). Males were more likely to have had a MI (83.9% vs. 72.8%, p = 0.02).

Presence of risk factors

Table 4 shows the frequency of risk factors by gender of the interviewed patients. The five risk factors included were family history of premature coronary disease, smoking, body mass index (BMI) > 25 and having been told of a high cholesterol or blood pressure level. Smoking was considered a risk factor if the individual was either a current smoker or quit smoking less than five years prior to interview (i.e. two to three years prior to MI or cardiac catheterization). Ninety percent of the premature ischemic heart disease patients had two or more risk factors.

Table 4. *Number of cardiovascular risk factors by sex of respondent (excluding gender).*

# of risk factors	Total		Males		Females	
	N	%	N	%	N	%
0	5	1.6	3	1.5	2	1.8
1	26	8.2	17	8.3	9	7.9
2	85	26.7	52	25.5	33	29.0
3	99	31.1	64	31.4	35	30.7
4	85	26.7	56	27.5	29	25.4
5	18	5.7	12	5.9	6	5.3

Table 5. *Percentage of respondents with risk factors for coronary heart disease, by gender and ratio of prevalence in males compared to females.*

	Total *(N = 318)*		Males *(N=204)*	Females *(N=114)*	Prevalence Ratio *(95% Confidence Limits)*
	N	%	%	%	
Smokers	99	31.1	32.8	28.1	1.17 (0.82-1.67)
Ex-smokers (all)	174	54.7	58.8	47.4	1.24 (0.99-1.56)
Quit < 5 yrs. ago (% of those who quit)	125	71.8	71.8	71.7	0.99 (0.81-1.21)
Overweight					
BMI 25 - 26.9	63	20.0	21.2	17.9	1.20 (0.74-1.94)
BMI ≥ 27	172	54.6	57.1	50.0	1.16 (0.93-1.44)
Hypertension	116	36.9	29.4	50.5	0.60 (0.45-0.79)
High cholesterol	175	54.7	56.9	53.6	1.07 (0.87-1.32)
Family history					
1 relative	112	35.2	36.3	33.3	1.09 (0.79-1.49)
2 or more	64	20.1	19.1	21.9	0.87 (0.56-1.36)

Table 5 shows the distributions of the individual risk factors. Except for hypertension, all were present in at least 50% of this population. A family history of premature coronary disease was reported by 176 of the 318 patients (55%). They reported a total of 276 close relatives who had suffered heart attacks before the age of 55. Thiry-five percent had one relative with an MI, and 20% more than one relative with an early MI. Family history of early coronary disease was equally prevalent in men and women.

Of the 318 patients, 310 said that they had had their cholesterol checked in the last five years. One hundred and seventy-eight patients (54.7%) had been told at some time that their serum cholesterol level was elevated. Of these, 57 (32%) recalled their cholesterol levels from the last measurement. It was reported as < 5.2 mmol/L in 21%, between 5.2 and 6.1 in 34%, between 6.1 and 7.1 in another 27% and ≥ 7.2 in 18% (97% of patients reported in SI units). Of the 129 patients who denied being told their cholesterol was high, only 18 (14%) remembered the last level: in half it was ≥ 5.2 mmol/L. Figure 9 shows in flow chart form what patients remembered about their cholesterol levels.

Over 85% of respondents at some point smoked at least 100 cigarettes. The mean age patients had started smoking was 16.5 ± 4.4 years. Thirty-one percent of the patients still smoke, averaging 20 cigarettes per day. Of the current smokers, 92% reported that they've tried to stop or decrease smoking in the last year.

Hypertension was the only risk factor whose reported prevalence was significantly higher in females. Since age could be a confounder in this relationship, the analysis was repeated using only women under 50 at diagnosis with similar results (Appendix A, Table A4).

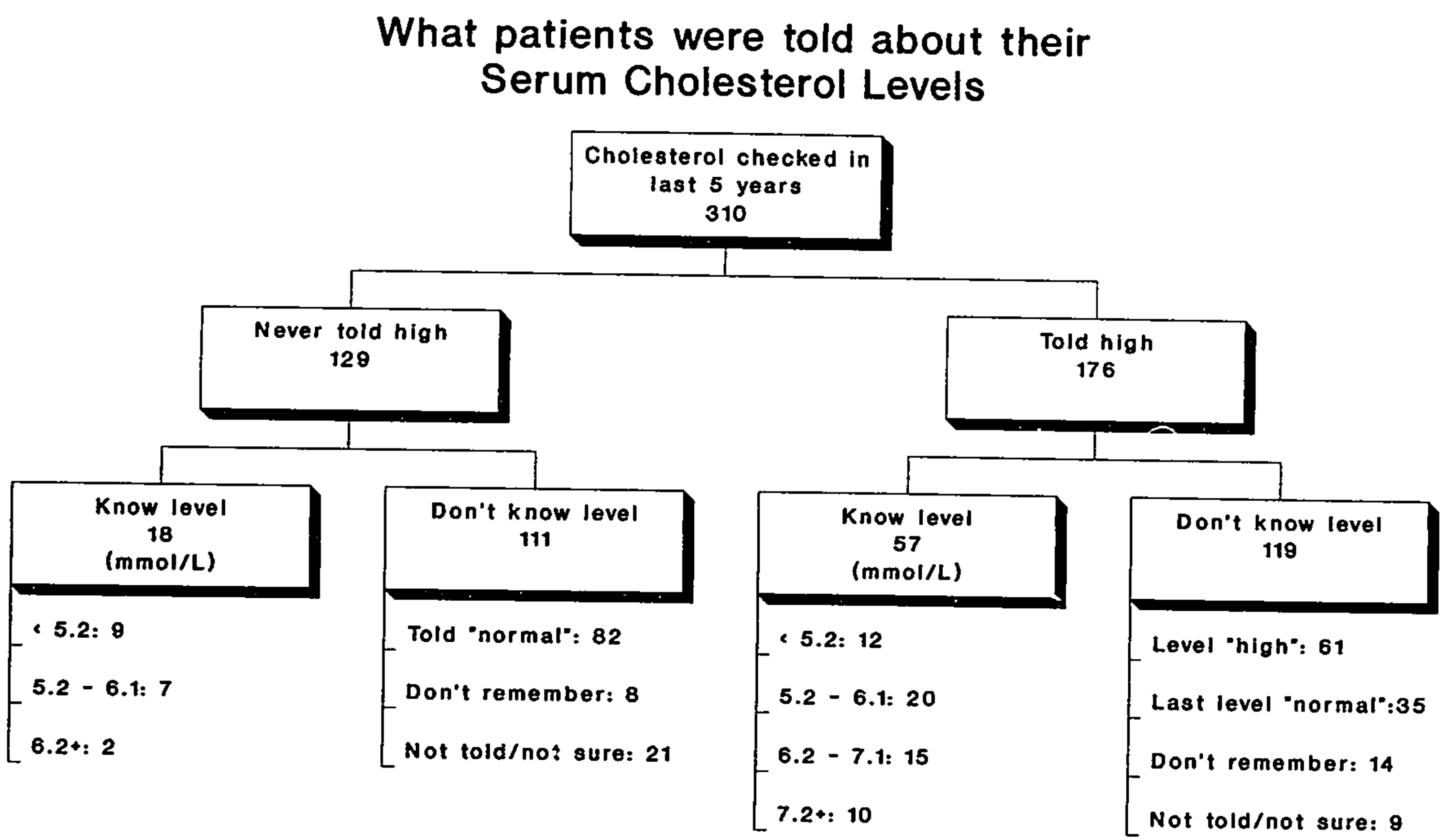

Figure 9. What patients said they were told and what they remembered about their serum cholesterol levels. Of the 318 patients, 310 remembered having their serum cholesterol checked within the previous five years. Of the 310, five did not know if they were told that their serum cholesterol was high, yielding data on knowledge on 305 patients.

Changes in risk factors

Of the 122 patients on the MI database, 36% had had their MI four to twelve months prior to the date we began the survey, 45% from one to two years earlier, and 19% from two to two and a half years earlier. Thirteen percent had also had a previous MI.

Almost 93% of the patients with premature ischemic heart disease reported having done something in the previous year to improve their health. The largest proportion (97/318 or 30.5%) increased exercise. Twenty-one percent mentioned improving their eating habits, and 17.6% said the most important thing they had done was to quit smoking. Twelve respondents who had quit smoking in the past year did not consider this the most important improvement to health and mentioned other changes instead: half mentioned increasing exercise.

Eighty percent of patients also had plans to improve their health in the following year (Figure 10). Increased exercise was again at the top of the list (46.9%) followed by improvements in eating habits and weight loss. Only 30% of smokers planned to quit smoking.

Eighty-seven percent of respondents reported having changed the way they eat in the previous two years. Figures 11 and 12 show these changes. Patients reported eating less fat, meat, dairy fat, eggs, salt, fried foods and sweets. They report eating more vegetables, fruits, poultry, fish and whole grains. Among the 36 respondents who reported not changing their dietary habits, 27 considered the things they presently eat to be healthy as one reason for not changing. The reason most often given for not changing was enjoyment of present food (16 considered it a major reason and 13 a partial reason).

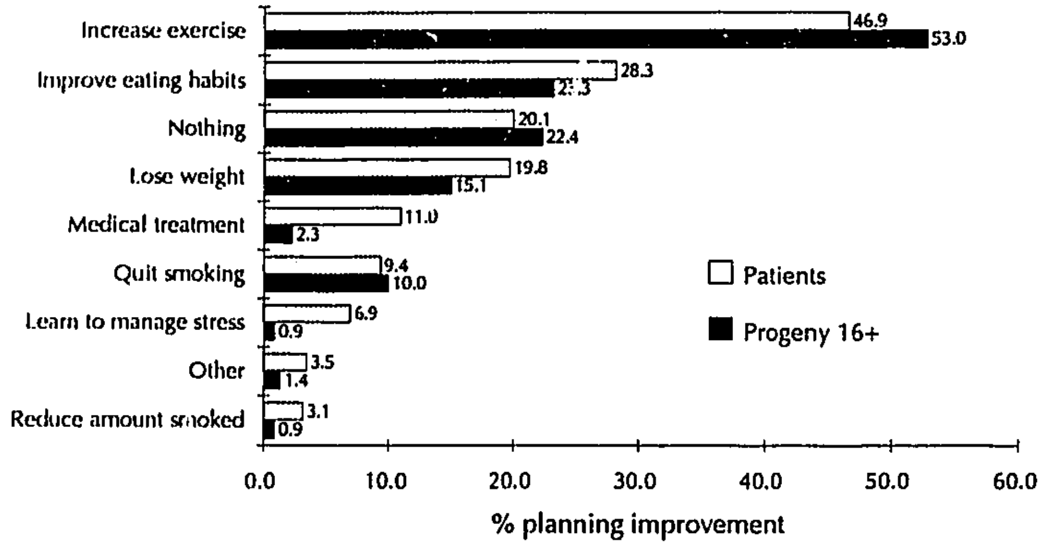

Figure 10. Plans for health improvements during the next year. Patients = 318; progeny 16+ = 219. Data for progeny 16 and over refer to Section 2, and are included here for comparison.

Three quarters of patients with premature ischemic heart disease had a BMI of 25 or more (normal BMI: 20-25): 68% of the women and 78% of the men. Seventy percent of the respondents were trying to lose weight: 29% of those with a BMI < 25, 79% of those with a BMI between 25 and 27, and 87% of those with a BMI of 27 or more. Of those trying to lose weight, 72% have been trying for more than six months, and only 13% for less than a month. Seventy-six percent say they have lost weight. Among those patients not currently trying to lose weight, 56% have tried to do so in the past, and 81% of these claim to have been successful.

Patients reported making several changes in response to instructions to lowering blood pressure or cholesterol levels. The differences in instructions are noteworthy. The primary

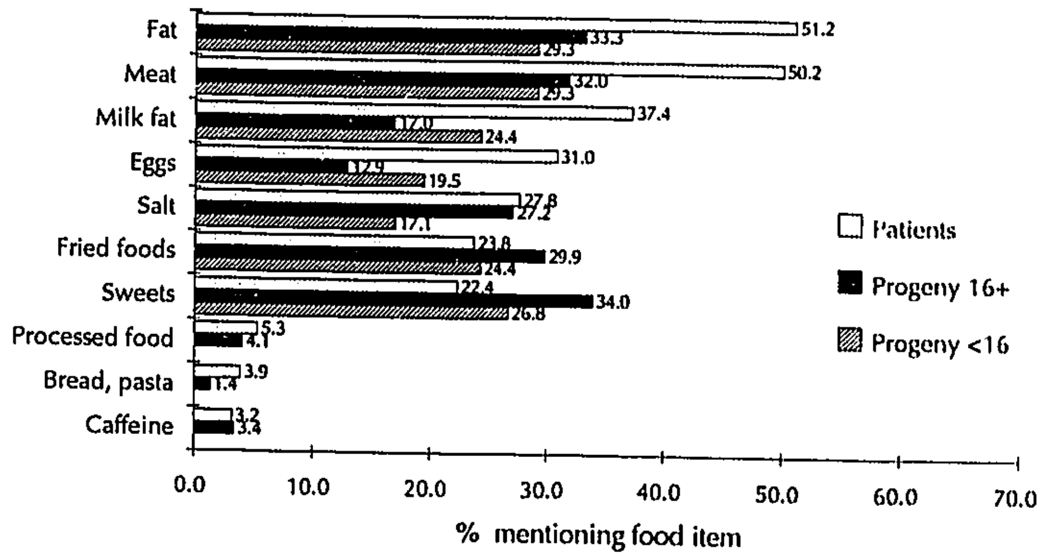

Figure 11. Changes to diet in the last 2 years: Foods respondents are eating less of, among those who have changed their diets. Respondents could give up to four answers. For patients, N = 281, for progeny 16+, N = 147, for progeny <16, N = 41.

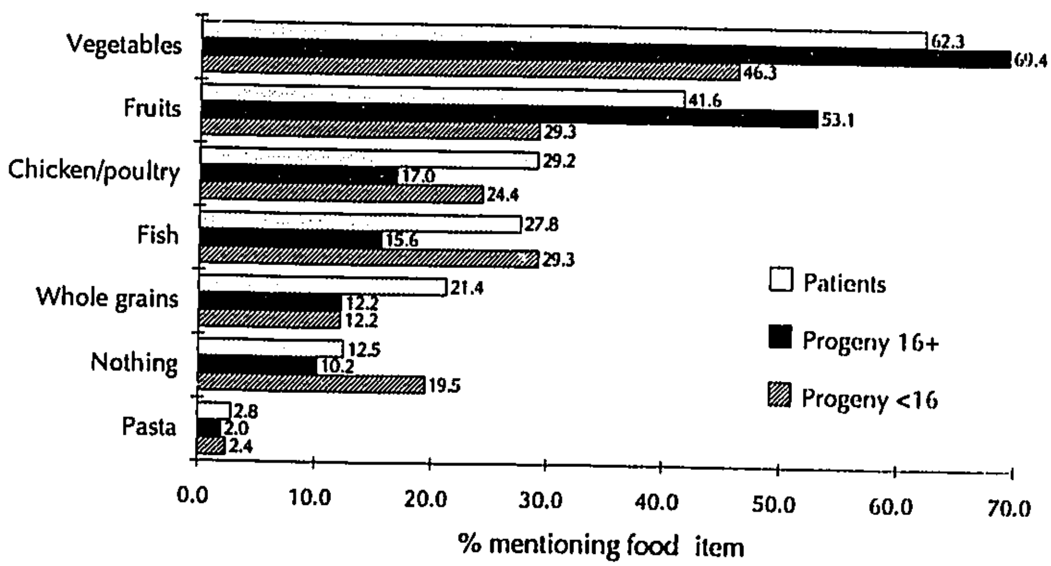

Figure 12. Changes to diet in the last 2 years: Foods respondents are eating more of, among those who have changed their diets. Respondents could give up to four answers. For patients, N = 281, for progeny 16+, N = 147, for progeny <16, N = 41.

treatment of elevated blood pressure remains pharmacological: almost 60% of patients were told to take medicine for their high blood pressure (Figure 13). They were also instructed to avoid stress, lose weight and follow a low salt diet. Compliance was high for medication use (88%) and exercise prescriptions (88%: 14/16), slightly lower for stress avoidance (64%), weight loss (71%), low salt diet (71%); and very low for stopping smoking (1/11: 9%) and reducing alcohol intake (0/7).

For lowering blood cholesterol, the primary therapeutic modality was dietary change (Figure 14): over three-quarters of the patients specifically mentioned being instructed to reduce saturated fat intake, and half mentioned dietary change in general. Just over one quarter (26.8%) were prescribed medicine. Exercise was advocated to 18.8% of patients. Figure 14 shows that reported compliance was high for all but the recommendations to reduce saturated fat and to stop smoking.

Determinants of behavioral change

We hypothesized that different manifestations of coronary heart disease might lead to different rates of behavioral change: that those with an acute event such as a myocardial infarction might be more likely to change than others. We therefore compared patients identified from the MI database to those identified from the catheterization database. We found no significant differences in dietary changes, smoking habits, weight loss, or therapies of cholesterol or blood pressure (Appendix A, Table A2). Since many patients on the catheterization database had also had infarcts, we compared those who had a heart attack to those who did not. Again there were no significant differences.

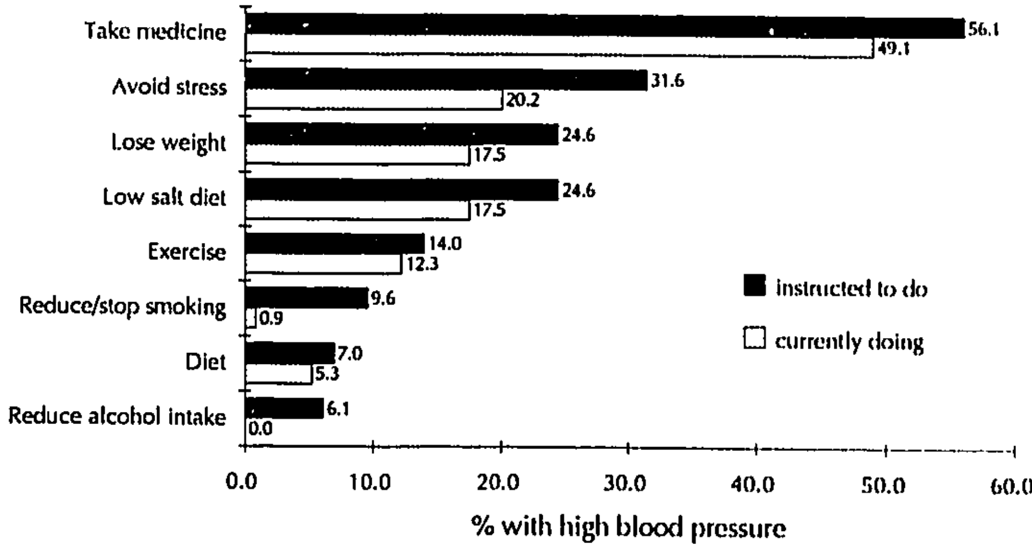

Figure 13. What patients were instructed to do and what they were actually doing to lower their blood pressure. Question was asked only of those who said they had been told their blood pressure was high. N = 114.

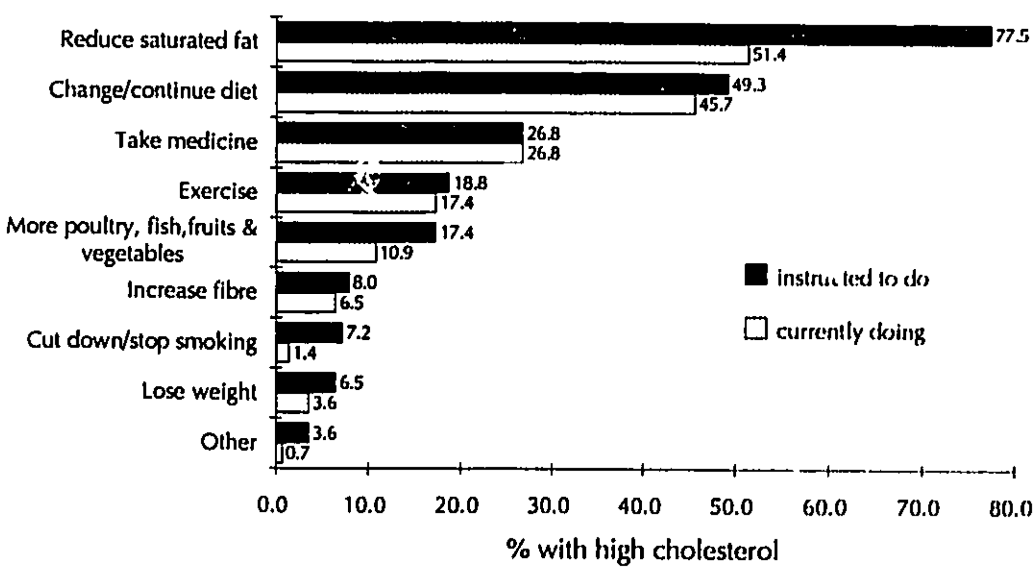

Figure 14. What patients were instructed to do and what they were actually doing to lower their blood cholesterol. Question was asked only of those who said they had been told their cholesterol was high. N = 138.

We looked at socio-demographic variables in relation to behaviour change: education, gender and language. There was a trend for those who attended university to smoke less than other patients (78% vs. 88%, $p = 0.02$) and to be more likely to quit. No other variables in relation to behavioral change differed by educational status (See Appendix A, Table A3).

Gender was an important determinant of perceptions of health: 58% of women but only 35% of men considered their state of health to be fair or poor. This held true when we excluded women over age 50 at the time of their MI or cardiac catheterization. Gender was also a determinant of dietary changes. Men were almost twice as likely as women not to have changed their diets. They were more than three times as likely to report that they probably should try to lower their cholesterol levels but haven't really tried, or to be doing nothing if their level was high. However, there was no significant difference in the proportion trying to lose weight, or in the proportion of hypertensives who were doing something to control their blood pressure. A far greater percentage of women were told that they had high blood pressure. Again this held true when women over 50 years of age at the time of their MI or cardiac catheterization were excluded from the analysis. More men had smoked at some time, but there was no significant difference in the proportion currently smoking (Table 6; for the same comparisons excluding women over 50 at diagnosis, taking into consideration the possibility of confounding by age, see Appendix A, Table A4).

None of the variables indicative of behaviour change varied significantly according to language of interview, despite the difference in sex and education distributions between English and French respondents.

Table 6. *Rates of behavioral change and male to female prevalence ratios.*

	Rate in men (%)	Rate in women (%)	Prevalence ratio (95% confidence interval)
Feel health fair or poor	34.6	57.9	0.60 (0.47-0.76)*
No change in diet	15.2	7.9	1.92 (0.95-3.9)
Should try to lower cholesterol level but haven't	10.9	2.9	3.85 (1.17-12.6)*
Not doing anything to lower cholesterol	15.3	3.9	3.77 (.89-16.03)
Trying to lose weight	73.7	67.3	1.04 (0.99-1.10)
Trying to control hypertension (among hypertensives)	64.4	74.6	0.86 (0.68-1.10)
Informed blood pressure high	29.4	50.5	0.61 (0.45-0.81)*
Smoked at some time	91.7	75.0	1.22 (1.09-1.36)
Current smoker	32.8	28.1	1.17 (0.82-1.67)

*95% confidence limits do not overlap 1.0.

Smoking status was related to some dietary change variables. Current smokers were somewhat less likely to have changed their diets than current nonsmokers. They were also more likely to feel that they probably should try to lower their cholesterol levels, but haven't really tried. They were twice as likely as nonsmokers not to be doing anything to control their cholesterol levels. There was no significant difference between smokers and nonsmokers in the proportion actively trying to control their blood pressure (Table 7).

Table 7. *Rates of behavioral change and current smokers to nonsmokers relative risks.*

	Current smokers (%)	Current nonsmokers (%)	Relative Risk (95% confidence interval)
No change in diet	18.2	10.1	1.80 (1.01-3.20)
Should try to lower cholesterol, but haven't	15.1	4.9	3.06 (1.41-6.64)
Not doing anything to lower cholesterol	17.8	7.7	2.30 (0.89-5.97)
Trying to control blood pressure	66.7	71.62	0.93 (0.71-1.21)

2. *Offspring (age 16+) of patients with early ischemic heart disease*

Description of population

We interviewed 298 progeny of parents with premature coronary disease: 168 (56%) were female. The age-sex distribution is shown in Table 6. This section reports on those offspring age 16 and over who were interviewed personally. The mean age was 23.9 (SD ± 5.7). Marital status was single for 117 (53.4%: median age 21) respondents, married for 93 (42.5%: median age 27), the other nine being divorced (6), separated (2) or widowed (1). Eighty-eight respondents (40%) had some post-secondary education. Of those respondents age 20 or over, 51% had some post-secondary education. The language of interview was English for 174 respondents (79.5%)

Table 8. *Age and sex distribution of the progeny interviewed.*

Age group	Females N (%)	Males N (%)
2 - 4	2 (1.2)	3 (2.3)
5 - 9	14 (8.3)	12 (9.2)
10 - 14	22 (13.1)	14 (10.8)
15 - 19	34 (20.2)	36 (27.7)
20 - 24	40 (23.8)	33 (25.4)
25 - 29	30 (17.9)	17 (13.1)
30 - 34	15 (8.9)	12 (9.2)
35 - 39	11 (6.5)	3 (2.3)

and French for 45. There were no significant differences in mean age, marital status or educational level between English and French respondents.

Table 9. *Demographic comparisons of English and French respondents.*

	English *(N = 174)*	French *(N = 45)*	Relative Risk (English:French)	Statistic	p-value
Age (mean + SD)	23.6±5.6	25.1±6.2		F=2.56	.10
Married (%)	44.2%	35.6%	1.24(.81-1.19)	χ^2=.38	.78
Post high school education (%)	39.7%	42.2%	.94(.64-1.38)	χ^2=.10	.89

Presence of risk factors

By definition, all subjects have at least one important risk factor for coronary heart disease, namely a family history of premature ischemic heart disease. From the survey questions we defined the following self-reported additional risk factors: currently smoking (having smoked at least 100 cigarettes and still smoking), being overweight (body mass index > 25), having been informed of high blood pressure, and having been informed of a high cholesterol level. By these criteria, 97 (44.2%) respondents had one additional risk factor, 38 (17.4%) had two and 7 (3.2%) had three additional risk factors (Table 10). (None had four, likely due to the very small numbers of individuals who knew their cholesterol levels.) Including exercise less than three times per week as a risk factor would give 165 (75.3%) with at least one additional risk factor: 88 (40.2%) with only one, 63 (28.8%) with two, and 14 (6.4%) with three or more. Even with this high prevalence of risk factors, we will underestimate risk for three reasons. More than half the sample has never had a serum cholesterol measurement.

We did not ask about a history of diabetes. And the greater rate of hypertension in females suggests that the rate in males is underestimated.

Table 10. *Number of risk factors besides family history and male sex by gender of respondent.*

# of risk factors	Total		Males		Females	
	N	%	N	%	N	%
0	77	35.2	24	25.8	53	42.1
1	97	44.3	50	53.8	47	37.3
2	38	17.4	15	16.1	23	18.3
3	7	3.2	4	4.3	3	2.4

Table 11. *Number and percentage of respondents reporting each of the classic risk factors, and those exercising less than three times a week.*

	Total (N = 219)		Males (N = 93)		Females (N = 126)		χ^2	p-value
	N	%	N	%	N	%		
Smokers	82	37.4	42	45.1	40	31.7	3.56	.059
Ex-smokers	29	13.2	8	8.6	21	16.7		
Overweight								
BMI 25 - 26.9	29	13.3	22	23.7	7	5.6	13.72	.000
BMI ≥ 27	39	17.9	16	17.2	23	18.4	.04	.982
Hypertension	24	11.0	6	5.4	18	14.3	2.61	.106
High cholesterol	20	9.1	6	6.5	14	11.1	.89	.344
Exercise less than 3x/wk	65	29.7	15	16.1	50	39.7	13.12	.000

The most prevalent among the individual risk factors was smoking. A total of 50.6% of the sample (111 respondents) had at some point smoked more than 100 cigarettes and 37.4%

(82) still smoked. Table 11 shows these results by sex and age. Table 11 shows a higher prevalence of current smokers among the men in our sample, and higher rates of having quit among the females. Smoking status also varied by education. The national tendency toward lower smoking rates in those with a university education is seen to a greater degree in this small subgroup (Table 12). Smokers and non-smokers were equally satisfied with their health. Still, 22 smokers planned to quit smoking in the next year to improve health.

Table 12. *Smoking status by education level attained.*

	N	Never smoked %	Former smokers %	Current smokers %
Less than high school	66	45	11	44
High school completed	65	41	14	44
Some post-high school	69	49	17	33
University degree	18	89	5	5

*Current smokers vs. others: $\chi^2 = 11.5$, $p = 0.01$

Overweight, defined as a body mass index over 25, is also present in 31% of the sample. Approximately one sixth of the sample (17% of males and 18% of females) meets the alternate definition of overweight as a BMI over 27 kg/m^2.

Knowledge of risk factors

We asked two questions about the causes of heart attacks and prevention of heart disease that were also asked on the Ottawa-Carleton Heart Beat Survey (for description see section 4, page 92). These questions had coded responses which were not read to the respondents. (See questionnaire III, Appendix B, questions B1, B3.) Figures 15 and 16 compare the two sets of survey results. The Heart Beat Survey results are based on the survey report, so are given for the entire survey population. Despite the different age distributions of these two populations, the answers are, for the most part, strikingly similar. For the major causes of heart attacks, the most frequent responses in both surveys appeared to be smoking and stress. However, 99% mentioned some dietary factor: the answers were split among poor diet (31%), cholesterol (29%), overeating (26%), and high fat diet (16%). Smoking was cited as a cause of heart attacks in 72% (59/82) of current smokers, 59% (17/29) of ex-smokers and 49% (53/108) of nonsmokers, a significant difference ($\chi^2 = 10$, $p = 0.006$). One notable difference in responses between the two surveys was that a smaller proportion in the present survey mentioned lack of exercise as a major cause of heart attacks. Only 13% of offspring of patients with premature ischemic heart disease mentioned heredity as a major cause of heart attacks, and only 9% mentioned high blood pressure. On the other hand, 5% mentioned fatigue, and 4% salt. The level of knowledge among offspring of CHD patients seems quite comparable to that in the general population.

The answers to the question on preventing heart disease were in sharp contrast to the answers about the causes of heart attacks. Whereas only 22% mentioned exercise as a cause of heart attacks, 64% mentioned more exercise as something a person can do to prevent heart disease. For smoking the reverse happened. Only 32% mentioned reducing smoking as a preventive maneuver, a smaller proportion than those who mentioned it as a cause of heart disease, and a smaller proportion than in the Heart Beat Survey. The responses concerning diet in the present survey were more specific: 15% mentioned improving diet, but 47% mentioned less high fat food. The responses were lifestyle-oriented: only one respondent cited lowering high blood pressure as a preventive maneuver.

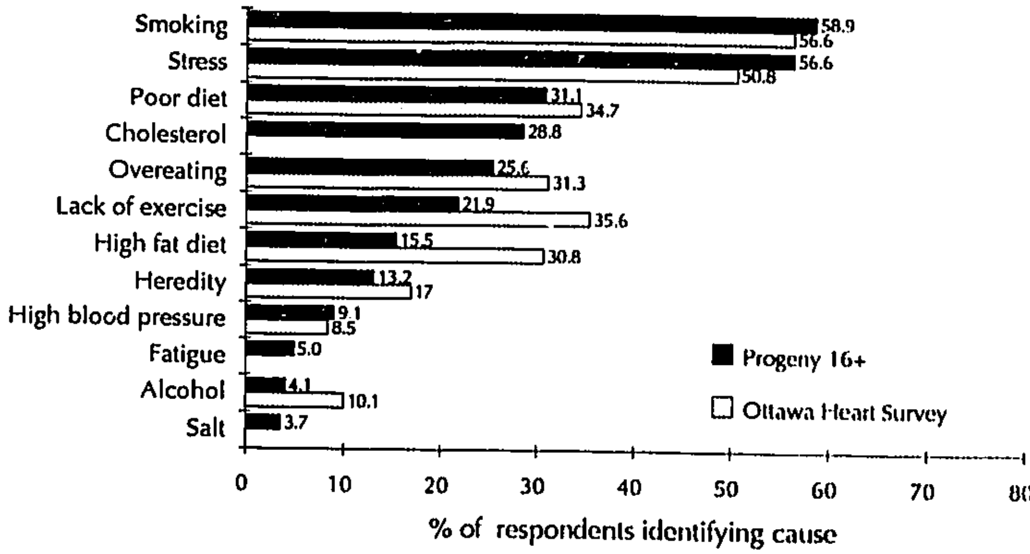

Figure 15. Major causes of heart attacks, according to progeny 16+ of CHD patients (N = 219) and in Heart Beat Survey (N = 1229). Respondents could give up to four answers.

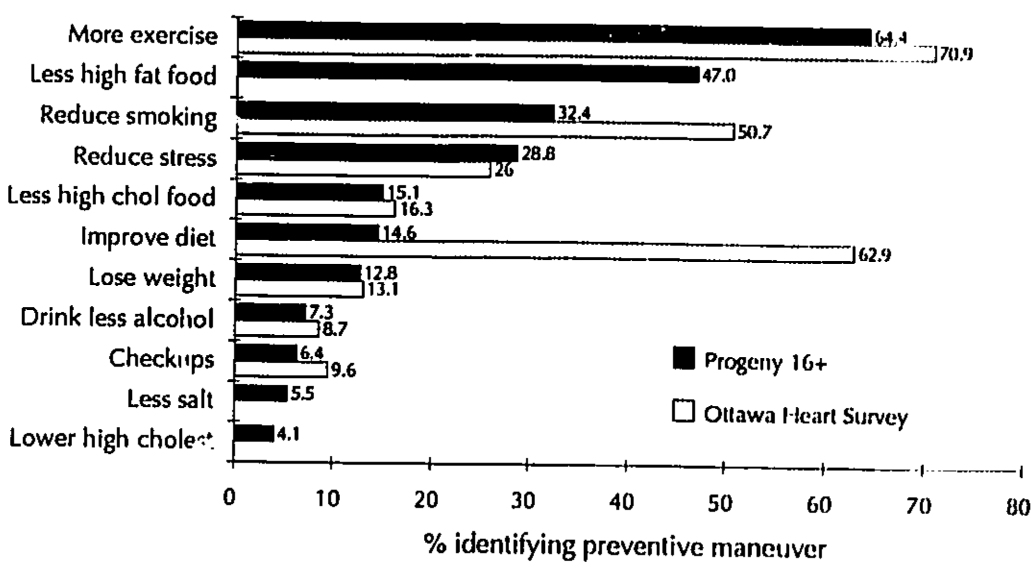

Figure 16. Methods of preventing heart disease, according to progeny 16+ of CHD patient (N = 219) and in Heart Beat Survey (N = 1229).

Every respondent mentioned either fat, cholesterol, saturated fat or fried foods as food related to heart disease. In addition 55% cited alcohol in response to the same question. Every respondent also cited foods high in fat, poor diet, foods high in cholesterol or foods high in saturated fat as causes of high blood cholesterol. Only 8% cited heredity as a cause of high cholesterol levels (Figures 17 and 18).

Respondents were not entirely confident of their knowledge and ability to lower their cholesterol levels. Only 22% (49) of the progeny age 16 and over said they would know how to lower their blood cholesterol if they wanted to, 40% (88) said they had some idea but were not really sure, and 35% (76) said they would not know.

Changes in risk factors: diet

Two thirds of the progeny age 16 and over (60% of males and 72% of females) reported having changed the way they eat in the past two years to reduce their chances of getting heart disease. Univariate analyses showed no significant differences between those who did and did not change on any of the following variables: age, education, marital status, smoking status, BMI, physical exam within the previous year, blood pressure check within the previous year (Appendix A, Table A5). The amount of worry about future heart disease was significantly related to dietary change. Worry was expressed on a Likert type scale from one to seven (least to most). Only 52% of those who had a worry rating of less than four had changed their diets, compared to 76% of those who worried more ($\chi^2 = 13.5$, $p = 0.001$).

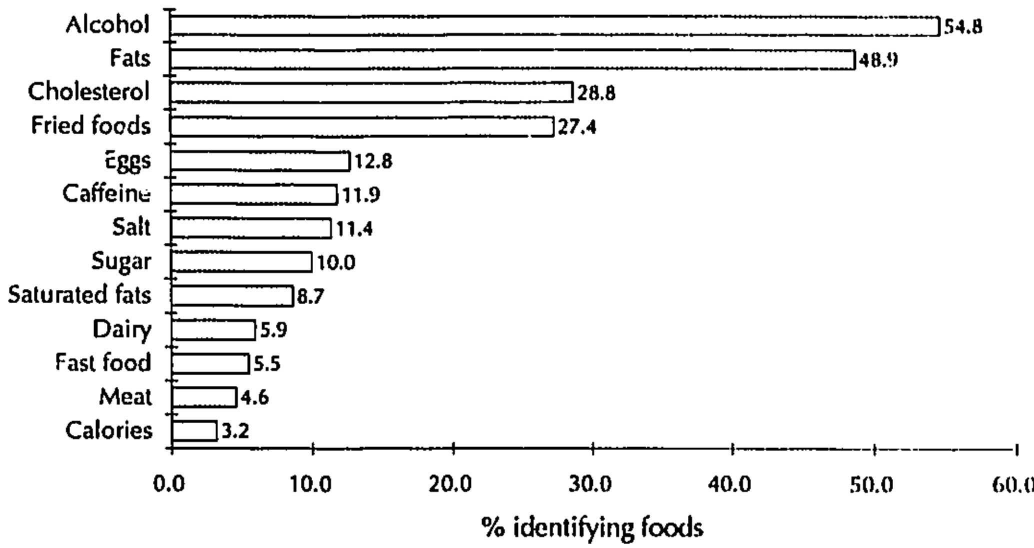

Figure 17. Foods and drinks related to heart disease, according to progeny 16+. Respondents could give up to four answers. (N = 219).

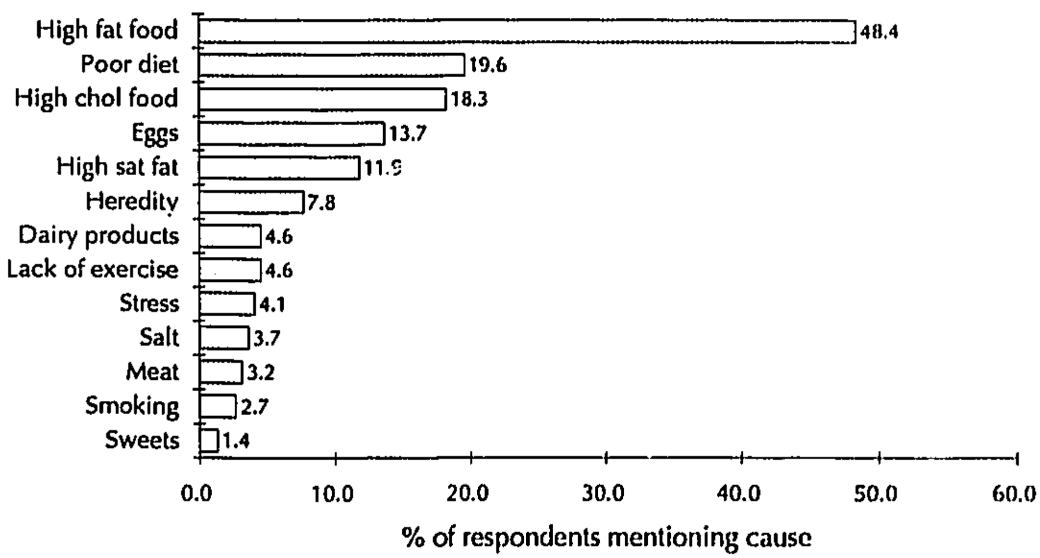

Figure 18. Major causes of high blood cholesterol, according to progeny 16+. Respondents could give up to four answers. (N = 219).

Table 13 shows the responses to some reasons for not changing given by the other 72 respondents. Enjoyment of the foods they presently eat was the most common reason given for not changing (73%). Sixty percent felt that the foods they presently eat are healthy. However, 30% believe that what they eat makes no difference.

Figures 11 and 12 (page 67) show the changes to diet that were reported. Most mentioned eating more fruits and vegetables while a minority increased consumption of poultry, fish, and whole grains. A quarter to a third of respondents reported eating less of each of the following: sweets, fat, meat, fried foods and salt.

Table 13. *Reasons for not making dietary changes.*

Reason	Major Reason N (%)	Part of reason N (%)	Not a reason N (%)
Making changes would be expensive	3 *(4.2)*	7 *(9.7)*	62*(86.1)*
I enjoy the things I eat and don't want to change	24*(33.3)*	29*(40.3)*	19*(26.4)*
The things I eat and drink now are healthy	22*(30.6)*	21*(29.2)*	29*(40.3)*
It would be too much trouble to change	10*(13.9)*	16*(22.2)*	46*(63.9)*
There are so many recommendations, I don't know what's good or bad	15*(20.8)*	15*(20.8)*	42*(58.3)*
I don't believe that what I eat makes a difference	12*(16.7)*	10*(13.9)*	50*(69.4)*
Someone else fixes my meals	13*(18.1)*	20*(27.8)*	39*(54.2)*
Making changes would be hard: I eat out so much	10*(13.9)*	16*(22.2)*	46*(63.9)*

Females who changed their diets within the last two years were on average 18 lbs. heavier than those who did not report such changes (means ± SEM: 145.6 ± 3.8 vs. 127.3 ± 3.9, t statistic = 2.75, p = 0.007). This relation held for BMI as well as weight in women (means ± SEM: 24.7 ± .56 vs. 21.8 ± .54, t-statistic = 2.83, p = 0.004) but was not true for males (change vs. no change: mean weight (lbs) ± SEM: 170.02 ± 3.4 vs. 173.2 ± 5.8, t-statistic = .509, p = 0.612).

Changes in other risk factors

When asked for the single most important thing the respondents had done in the past year to improve their health, 34% (73) mentioned exercise, 22% (48) said they improved their eating habits, 7% (14) said they lost weight and 4% (9) said they had quit smoking. 18% (39) did nothing (see Figure 14). In terms of the coming year, 22% (49) plan to do nothing, but 53% (115) plan to increase exercise (46 of whom already did so in the past year), 23% (51) plan to improve eating habits, 15% (33) plan to lose weight and 10% (22) plan to quit smoking.

We examined by univariate analysis variables that might be related to making lifestyle changes. In answer to the question "What is the single most important thing you have done in the past year to improve you health?" we compared respondents who said "nothing" to those who had made a change. Current smokers were almost three times as likely as non-smokers to have done nothing (29.6% vs. 11%; RR = 2.69, CI = 1.5 - 4.8; χ^2 = 10.69, p = 0.001). A trend seen in univariate analysis for those who exercised less than three times a week to make no changes (26.2% vs. 14.5%, p = 0.05) became significant when smoking was controlled for in a stratified analysis (Mantel Haenszel χ^2 = 6.5, p = 0.01).

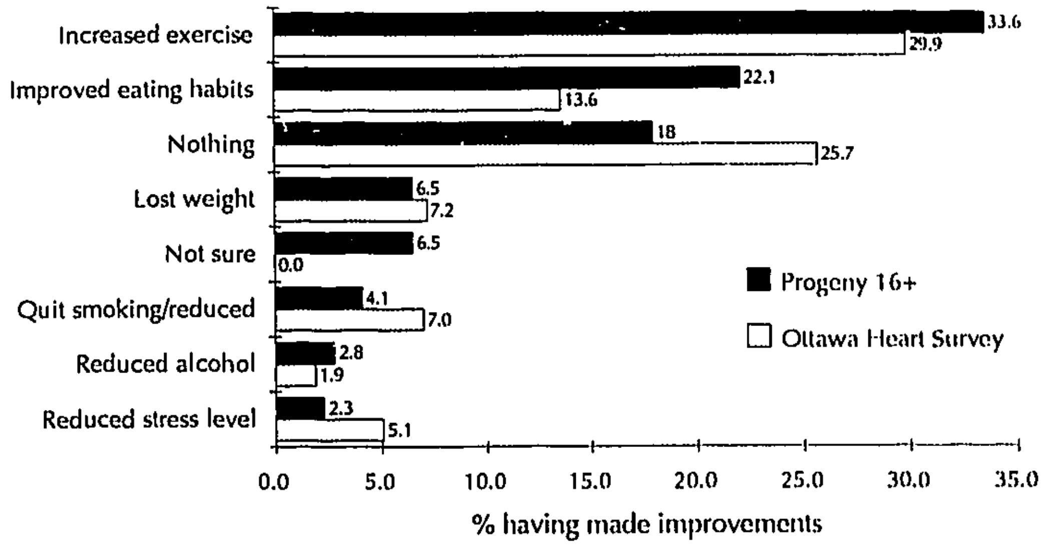

Figure 19. The single most important thing done in the past year to improve their health by progeny 16+ of CHD patients (N = 219) and in the Heart Health Survey (N = 1231).

There was considerable emphasis upon weight loss among the study participants: 47% (104) were currently trying to lose weight and a further 28% (62) had tried to lose weight in the past (86% of the latter said they were successful). Forty-one percent of those with a BMI of less than 25, 48% of those with a BMI between 25 and 27, and 64% of those with a BMI over 27 were trying to lose weight ($\chi^2 = 9.47$, $p = 0.009$). Seventy percent of those with a BMI less than 25 said that they were trying to lose weight primarily for appearance, while only 36% of those with a BMI over 27 gave that reason: 56% gave health reasons as their first answer. Almost equal numbers reported dieting (77) and exercising (72) as the method they are using to lose weight. Three quarters report having lost weight. Only 40% (88) of respondents felt they were getting as much exercise as they need.

Measurement of risk factors: Serum cholesterol measurement

Although 78% of the offspring 16 and over have had a physical exam within the last three years, 45% (97/217) have never had their cholesterol checked, and a further 12 respondents (5.5%) were not sure if they had it measured (Figure 20). This was not influenced by whether the parents had high cholesterol levels. More females (53%) than males (42%) had cholesterol measured, but this was not statistically significant ($\chi^2 = 1.9$, $p = 0.168$). Only five respondents actually knew their cholesterol levels and could tell us the number: of these two had levels over 7 mmol/L (270 mg/dL), one of whom had "never been told it was high".

Logistic regression analysis was used to assess factors related to whether or not offspring had had their serum cholesterol measured in the previous five years. In a model that examined age, BMI, sex, smoking status, physical examination within one year, amount of worry about cardiovascular disease and the number of relatives with premature CHD, only age and physical exam were related to cholesterol measurement. Those who were older ($p < 0.01$) and who had undergone a physical examination within the last year (Odds ratio 3.78, 95% CI 2.08-6.89; $p < 0.0001$) were more likely to have had a cholesterol measurement performed.

Table 14. *Logistic regression parameters for model of serum cholesterol measurement = constant + physical exam + age. Log likelihood of model = -134.45, of constants only model = -148.4. 2LLR = 27.87, 2df, p = .000. (Physical exam within 1 year binary coded and age by year.)*

Parameter	Estimate	S.E.	T-ratio	P-value	Odds ratio	Upper 95	Lower 95
Constant	-2.54999	0.67834	-3.75915	0.00017			
Physical exam	1.33023	0.30585	4.34936	0.00001	3.78	6.89	2.08
Age	0.06299	0.0261	2.41318	0.01581	1.07	1.12	1.01

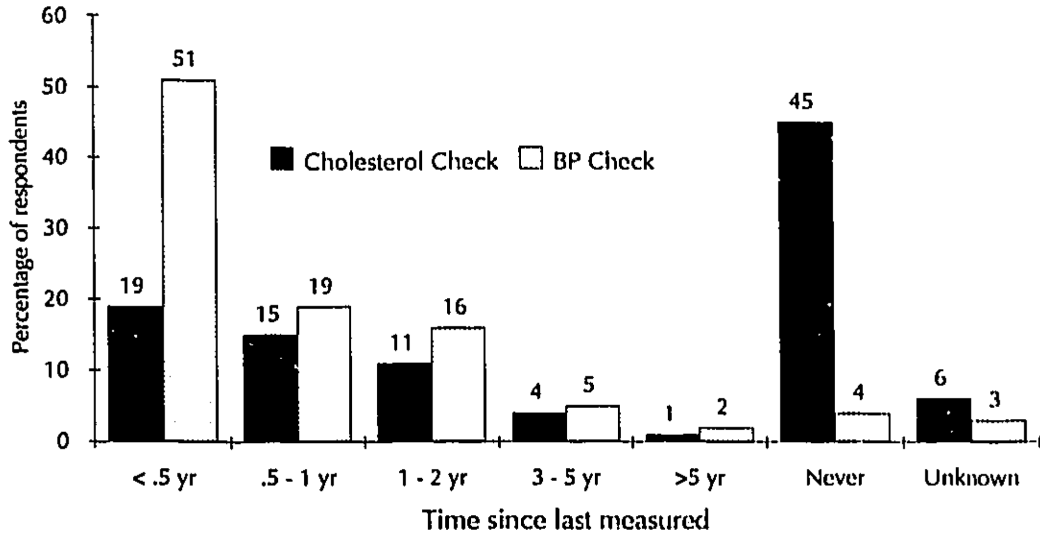

Figure 20. Time of last measurement of serum cholesterol and blood pressure. Offspring 16+, N = 219.

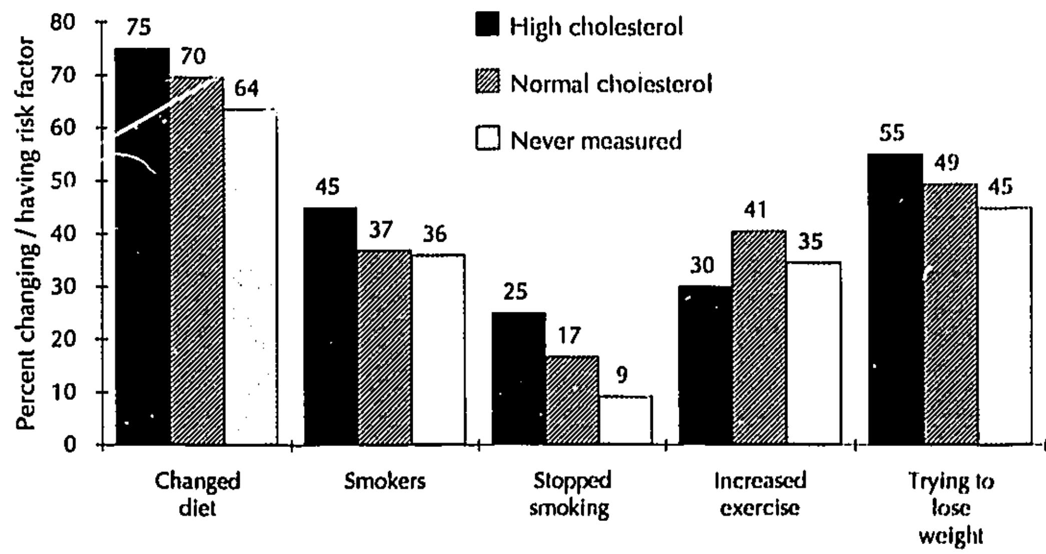

Figure 21. Risk factors and changes according to whether respondents' serum cholesterol was measured, and if measured, found to be "high" or "normal". Never measured: n = 110, told cholesterol was "high": N = 20, told cholesterol was "normal": N = 89.

Twenty offspring over age 15 were told at some point that their cholesterol levels were high: 20% of those who had it measured. Measurement and knowledge of results did not seem to affect risk factor changes. We compared those who were told their cholesterol levels were high, those who were told it was normal and those never had it measured. There was no significant difference in the proportion who changed their diets ($\chi^2 = 1.43$, $p = 0.49$), stopped smoking ($\chi^2 = 4.54$, $p = 0.10$), increased exercise ($\chi^2 = 1.14$, $p = 0.56$), or were trying to lose weight ($\chi^2 = 0.97$, $p = 0.62$). Very slightly greater changes in diet and attempts at weight loss were noted in those with high cholesterol, but there was insufficient power to detect significant changes of this magnitude. Figure 21 shows these results.

Of the 20 respondents who knew their cholesterol levels were high, 25% (5) were not especially concerned about it, 30% (6) felt they probably should try to lower it but haven't really tried, and 40% (8) have been trying to reduce their cholesterol levels.

Among those who had never had their cholesterol measured, 71% (82) stated that they would be interested in finding out their cholesterol levels, whereas 15% (17) would prefer not to know. The remaining 14% (16) were unsure. Yet, 81% (87) reported that they would make changes to their lifestyle, and 76% (81) stated they would make changes to their diets if they found out that their cholesterol levels were high. They would make the following lifestyle changes: 33% (35) would get more exercise, 33% (35) would eat fewer high fat foods, 21% (22) would eat fewer high cholesterol foods, 15% (16) would follow doctor's orders, and 6% (6 of 39 smokers) would stop or reduce smoking (Figure 22). Specific dietary changes mentioned were to eat less fat, meat and fried foods, and to eat more fruits and vegetables (see figure 23). Except for the dietary changes, these numbers are lower than

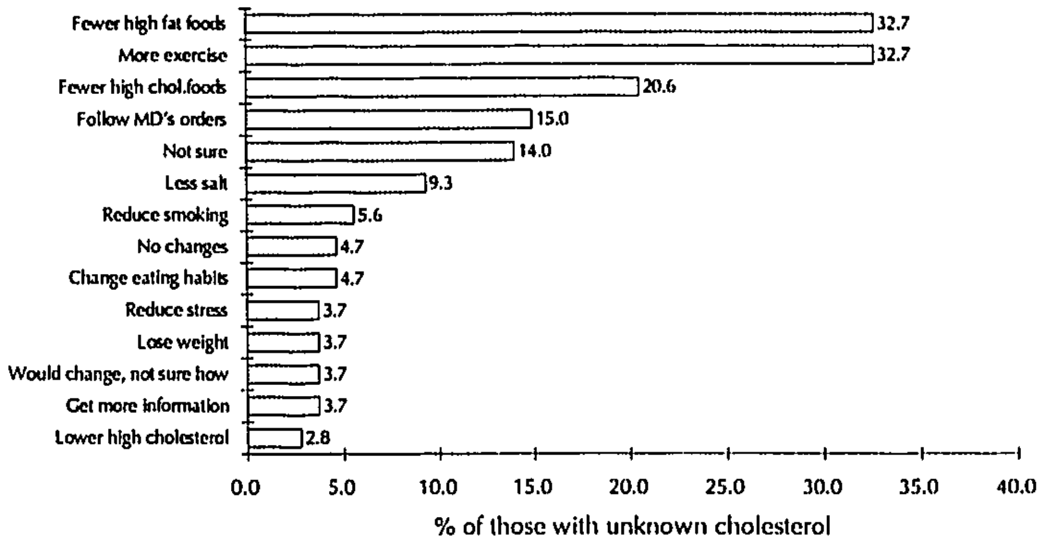

Figure 22. Changes to present lifestyle which would be made by progeny 16+ if they found their cholesterol level to be high. Question was only asked of those who had never had their cholesterol measured, or were unsure (N = 107).

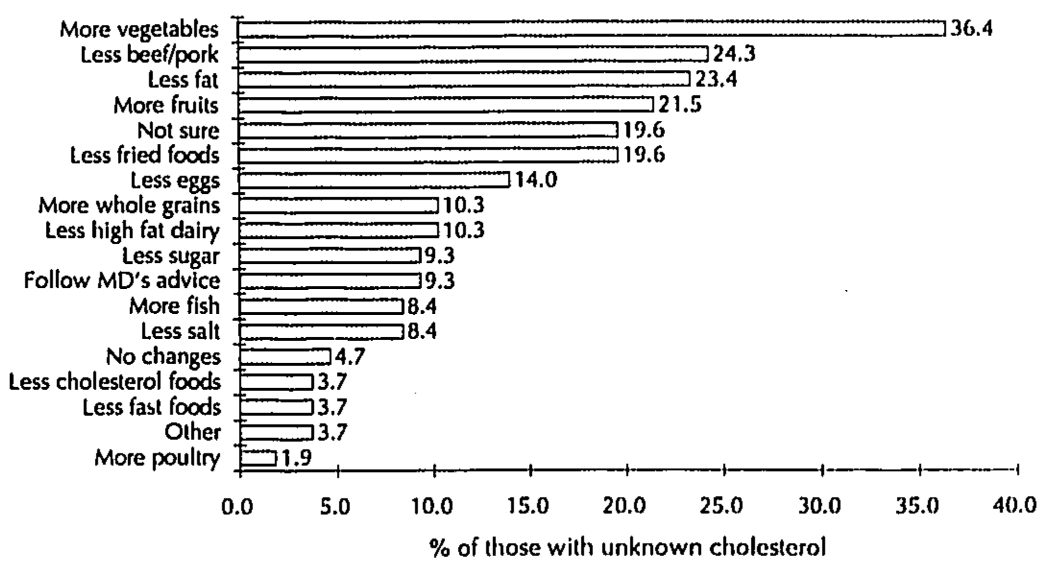

Figure 23. Changes to present diet which would be made by progeny 16+ if they found their cholesterol level to be high. Question was only asked of those who had never had their serum cholesterol measured, or were unsure (N = 107).

those who said that they plan to make those lifestyle changes anyways in the coming year.

Differences in responses by language of interview, sex of respondent

Women were more likely than men to have seen a general practitioner and to have had a physical exam in the previous year. They reported having had their blood pressure checked more recently: 80% of women and 57% of men in the survey had it checked within the last year. This probably accounts for the fact that more women reported high blood pressure. Women were somewhat more likely to have had their cholesterol checked, but the difference was not statistically significant.

With respect to risk factors, men more often felt that they got enough exercise. 84% of the men and 60% of the women reported exercising for more than 15 minutes at least three times a week. Although similar proportions of males and females were once smokers, women reported quitting at over twice the rate of men: 21/60 women had quit vs. 8/50 men. Women also more frequently reported trying to lose weight. More women than men reported changing their diet to prevent heart disease, but this fell short of statistical significance (Table 15).

There were several differences between respondents interviewed in English and those who chose to be interviewed in French. English respondents were more likely to report changing their eating habits. Among those who have not changed their diets in the last two years, French respondents were more likely to say that they doubt what they eat really makes much difference in the chances of getting heart disease, and that making changes would be too hard because of eating out. English respondents were more likely to feel that the things they presently eat were healthy so that there was no reason to change (Table 16).

Table 15. *Rates of risk factor measurement and changes and male to female prevalence ratios.*

	Rate in men (%)	Rate in women (%)	Prevalence ratio (95% confidence interval)
Physical exam within 1 year	44.1	69.9	0.64 (0.49-0.82)
Blood pressure checked within 1 year	57.0	80.2	0.71 (0.58-0.87)
Cholesterol checked within 1 year	28.3	38.4	0.74 (0.50-1.09)
Informed of high blood pressure	7.1	14.6	0.48 (0.20-1.16)
Enough exercise	57.0	27.8	2.05 (1.45-2.86)
Exercise more than 15 minutes at least 3 times a week	83.9	60.0	1.39 (1.18-1.64)
Quit smoking	8.6	16.7	0.46 (0.23-0.96)
Trying to lose weight	50.0	73.3	0.68 (0.46-1.00)
Changed diet	60.2	72.2	0.83 (0.68-1.02)

English respondents expressed more interest in finding out their cholesterol levels if they did not already know them, worried more about future risk of heart disease, and were more often trying to lose weight. There was no statistically significant difference in smoking or exercise rates.

Table 16. *Changes in diet and reasons for not changing: English vs. French respondents.*

	English respondents % *(n)*	French respondents % *(n)*	Relative Risk (95% Confidence Interval)
Changed eating habits	70.7% *(123)*	53.3% *(24)*	1.33 (0.99-1.77)
Reasons for not changing:			
Always ate well	68.6% *(35)*	38.1% *(8)*	1.80 (1.01-3.20)
It doesn't matter	25.5% *(13)*	42.9% *(9)*	0.59 (0.30-1.18)
Eat out too much	29.4% *(15)*	52.4% *(11)*	0.56 (0.31-1.01)
Interested in cholesterol level	76.3% *(71)*	50.0% *(11)*	1.53 (0.99-2.35)
Trying to lose weight	51.5% *(89)*	33.3% *(15)*	1.54 (1.00-2.39)

3. *Offspring under age 16*

We received some information about 79 offspring aged two to 16 from their parents (nine parents of 16 year olds answered for their 16 year olds). In these "proxy" interviews we asked primarily about cholesterol screening and dietary management, and the extent to which knowledge of serum cholesterol levels might change that management. Subgroup analyses are difficult to do for the children under 16 because of the small sample size.

Seventy percent of these children had seen a physician for a physical exam in the preceding year, and 87% in the preceding two years. Only 16/79 children (20.3%) had ever undergone a cholesterol measurement. Three (19%), age 13 to 16, were informed that their levels were high: a similar proportion to those 16 and over. The only serum cholesterol level remembered was that of a seven-year old, whose level was 5.3 mmol/L, and the parents were not told that this was high.

Fifty-six percent (44/79) of the children had had a blood pressure measurement in the previous year: 80% of the number who had been seen by a physician for a physical exam. Only three were told that it was high.

Fifty-one percent had reportedly changed their diets in the previous two years to reduce their chances of getting heart disease. As shown in Figures 11 and 12 (page 67), the changes mostly parallel those of the older offspring, especially for fat, meat and sweets. Parents less often reported that children under 16 were eating more fruits and vegetables, compared to the self reports of the older offspring (fruits: 29.3 vs. 53.1%, $\chi^2 = 7.61$, $p = 0.005$; vegetables: 46.3% vs. 69.4%, $\chi^2 = 6.5$, $p = 0.01$). This is not unexpected for young children.

Over 90% (56/62) of the parents indicated that they would try to make further changes to their child's lifestyle (63% dietary) if they found out that their child's cholesterol level was high. Only three would try to make changes if they found out that their child's cholesterol level was normal.

Most parents were satisfied with their children's weight and level of exercise. Seventy-two percent felt that their child's weight was about right and seventy-five percent felt they were getting enough exercise. Ninety percent said that their children exercised at least 15 minutes at least three times a week.

We asked parents to rate on a scale of one to seven their concern and their child's concern about having a heart attack in the future. A rating of one was given the explanation "never thinks about it". Results showed the two variables were not correlated ($r = +0.12$, 95% CI: -0.11 to +0.34). Parents rated their own concern higher than their children's (mean parents = 2.84, mean children = 1.76, paired $t = -4.4$, $p = 0.000$), although the level of concern was generally low. Parents rated their child's concern as one or two in 62/77 cases (80.5%) and only in five cases did they rate their child's concern as five or above. Parents rated their own concern as one or two in 43/74 cases (58.1%), and as five or above in 15 cases (20.3%).

4. Comparison of offspring to Ottawa-Carleton Heart Beat Survey

We tried to assess whether there were any greater efforts at screening in the families of patients with premature heart disease than in the general population, and whether the offspring differed in terms of risk factors known to them. To do this, we obtained the data set from the Ottawa-Carleton Heart Beat Survey of 1987, a general community survey of risk factors for heart disease. This survey randomly sampled residential telephone numbers in the community and conducted personal interviews on 1233 individuals aged 19 to 64. This represented 71% of the eligible study sample. There was a slight under-representation of individuals age 20-29. Physical measures such as weight and blood pressure were obtained at the time of interview. The two surveys overlapped in the age group of 19 to 39, so we report analyses based on this age group. In this age group there were 690 respondents in the Heart Beat Survey (44% male) and 180 in the current survey (41% male).

Table 17. *Risk factor measurement and changes: comparison of Ottawa-Carleton Heart Beat Survey (1987) and current survey (1990). Respondents 19 to 39 years of age. In this age group, for the Heart Beat Survey N = 690, for the Family Survey N = 180.*

	Heart Beat Survey Family History	No Family History	High Risk Survey	χ^2 (2 df)	p-value
Feel health fair or poor	6.1	5.0	8.9	3.25	.197
Blood pressure checked within 1 yr	78.8	78.5	74.5	1.44	.488
Cholesterol checked within 1 yr	22.7	25.3	38.2	14.57	.001
Informed of high blood pressure	16.6	13.6	13.3	1.43	.490
Enough exercise	26.5	33.3	36.7	5.94	.051
Exercise more than 15 minutes at least 3 times a week	54.8	60.3	68.3	8.32	.016
Smoking					
Never smoked	47.7	48.0	44.4	0.68	.714
Ever smoked				5.02	.08
Quit smoking	20.1	14.5	15.6		
Current smoker	32.3	37.5	40.0		
Tried to stop	71.3	66.5	68.1	0.63	.729
BMI ≥ 25	29.6	24.9	35.2	6.56	.038
19-24	16.2	13.2	26.4	4.74	.093
25-39	31.4	27.5	44.3	9.16	.010
Trying to lose weight					
Everybody	46.0	40.1	49.7	5.31	.070
Men	35.7	27.7	33.3	3.13	.209
Women	54.6	50.0	58.4	2.00	.368
BMI ≥ 25	67.1	57.6	61.9	1.72	.424

Table 17 compares the current survey of offspring of patients with premature coronary disease to the respondents in the Heart Beat Survey reporting a history of heart problems in the immediate family and those reporting no such problems. The only significant difference between the two populations was in the percentage who had their cholesterol checked in the previous year: close to 40% of those in the present survey, but less than 25% of those in the community survey. There were trends toward differences in two other variables: a higher proportion in the present survey had a BMI of 25 or greater, and more claimed to be exercising at least three times a week. Overall, there were minimal differences between the present survey and the Heart Beat Survey, and within the Heart Beat Survey between those who had a family history of heart disease and those who did not.

The present survey contained a greater proportion of younger respondents than the Heart Beat Survey: 51% were in the 19 to 24 age group, compared to 17% of respondents in this age group in the Heart Beat Survey. We therefore examined the potential effects of age as a confounder by calculating rates separately for the 19 to 24 age group and the 25 to 39 age group. These groups were chosen to yield approximately equal groups in the smaller survey. In both surveys, there were two significant differences between the two age groups. BMI was greater in the older age group and there were differences in smoking rates between the two age groups. Comparing within each of the two age groups, BMI was higher in the present survey than in the Heart Beat Survey (for 19-24: $\chi^2 = 4.1$, $p = 0.04$, RR = 1.88; for 25-39: $\chi^2 = 7.6$, $p = 0.006$, RR = 1.53). The three way comparison of the Heart Beat survey (family history and no family history), and the present survey are shown in Table 17. There is considerably higher prevalence of obesity among our population of high risk offspring. The other comparisons by age group are shown in Appendix A, Table A6. Although smoking rates differed between age groups within surveys, the differences between surveys were not significant in either age group. (For the comparison never smokers vs. ever smokers, in the 19 to 24 age group $\chi^2 = .01$, $p = 0.94$; in the 25 to 39 age group $\chi^2 = 3.4$, $p = .063$.)

Recently data have also become available from nine of the provinces in the Canadian Heart Health Survey. Published reports provide data for 18-34 year olds in some areas. To compare the results with the present survey, we examined the data on the 186 offspring aged 18-34 and applied the reported age-sex rates from the Canadian Heart Health Surveys to the age-sex distribution in our survey (18-24: M - 27%, F - 33%; 25-34: M - 16%, F - 34%). Table 18 shows the comparison of these results. The most notable difference is the higher reporting of hypertension in women in our sample. Despite this the total number of risk factors, excluding family history, is only slightly higher in the present survey. This is likely due to the fact that we did not actually measure risk factors.

Table 18. *Risk factors: comparison of current survey and Canada Heart Health Surveys, age-gender standardized for respondents age 18-34.*

	Current Survey	Canada Heart Health Surveys
Smoking		
Never smoked	48.4	35.7
Former smokers	14.0	25.1
Current smokers	37.6	38.6
Mean no. of cigarettes/day	16.6	17.4
Prevalence/awareness of HBP		
Men	3.4	3.3 / 1.4*
Women	8.4	1.3 / 1.1
Body mass index		
< 25	67.6	71.1
25-26	14.6	11.3
27+	17.8	18.0
Number of major Risk factors (excluding family history CHD)		
0	50.0	54.7
1	41.4	36.7
2	8.6	8.1
3	0.0	0.2

*The first figure is prevalence as measured, the second is awareness and compares to the present survey.

Discussion

This survey of the offspring of adults with premature ischemic heart disease has identified a low rate of compliance with preventive guidelines. Both the Canadian Consensus Conference on Cholesterol and the Ontario Task Force Report recommend screening of those with a family history of coronary heart disease. Yet 44% of late adolescent and young adult offspring had never undergone such testing. Screening for hypertension was also poor, particularly for the group at highest risk for coronary heart disease. Only 57% of males over age 15 reported having had their blood pressure measured in the preceding year.

The rates of self-reported modifiable risk factors for ischemic heart disease were high among the late adolescent and young adult offspring: 37% were current smokers, 31% were overweight, and 39% exercised less frequently than three times per week. The high rate of smoking is particularly alarming in light of evidence that smoking multiplies the adverse effect of a family history of ischemic heart disease. The true prevalence of these risk factors may be higher, as smoking and obesity may have been underreported and exercise overreported. Blood pressure and cholesterol were not measured in a large proportion of this group. The prevalence of cardiovascular risk factors other than family history must therefore be regarded as the minimal estimates for this group.

Despite a health care system with no financial barriers to medical examination, the results of primary prevention in this high risk group are disappointing. The reasons for this are undoubtedly complex. One important factor is the low rate of contact with physicians of the study population, which is also true of young adults in general. Another is the lack of knowledge regarding the importance of family history, and the resulting inaccurate perception by the offspring of their actual cardiovascular risk. In the present study, most offspring seemed unaware of the increased risk posed by their family histories. This has also been noted in the siblings of patients with coronary heart disease.[14,15] It is also clear from these results that practitioners caring for these high risk patients do not comply with existing recommendations regarding lipoprotein measurement. It is unclear whether this is because practitioners are unaware of the guidelines, because they disagree with them, or because the health care delivery system is not set up in a way that facilitates the identification and treatment of this high risk group. Another explanation is that adoption is occurring, but slowly.

The low rates of cardiovascular risk factor assessment and management identified in this survey represent missed opportunities for the primary prevention of premature coronary heart disease. Individuals with a family history of early coronary heart disease constitute a substantial proportion of those with early myocardial infarction. In one population-based study, 50% of those with a myocardial infarction before age 50 were members of only 5% of families.[212] In the present study 55% of the patients with premature CHD reported premature myocardial infarction among their close relatives. It thus appears that this increased risk has either received insufficient attention over two generations, or that some form of denial is present.

Education about health risks does not translate directly into either lifestyle change or compliance with medical advice.[213] Nonetheless, the potential benefits of identifying individuals at increased cardiovascular risk include detecting and treating hypertension and dyslipoproteinemias, and counselling patients to avoid or stop smoking. If smoking multiplies the risk of ischemic heart disease in those with a positive family history,[212,214] one could anticipate that gains in life expectancy from coronary artery risk factor modification would be more substantial in such families than in the general population.[215] Other potential benefits of identifying this population include the opportunity to influence those who have either adopted excessively restrictive diets[216] or who have developed unrealistic perceptions of their risk of heart disease.[217]

Among the offspring personally interviewed heredity was rarely mentioned as a cause of heart disease. Several explanations are possible. Individuals may know about the link between heredity and heart disease, yet not consider heredity a "cause". Or there may be a true lack of awareness due to "optimistic bias" - the tendency to underestimate one's own risk of developing disease.[218,219,220] These explanations would have implications for modification of risk factors. Perceived susceptibility to disease is an important component of models of health behaviour (the Health Belief Model,[154] the Theory of Reasoned Action,[155] Subjective Expected Utility Theory,[156] Protection Motivation Theory[157]). These models all suggest that major lifestyle modifications will be unlikely in the absence of perceived susceptibility to disease. Lack of awareness of susceptibility to coronary disease through lack of knowledge or optimistic bias could lead to less than optimal risk factor modification.

Would measurement of risk factors such as cholesterol change this perception? A recent review of the impact of cardiac risk factor measurement concluded that there is no support in the literature for the contention that risk factor measurement accompanied by supportive information is of itself sufficient to achieve sustained behaviour change.[221] It suggested that risk factor measurement might reduce optimistic bias and alter intentions and could perhaps change behaviour if combined with environmental and individual interventions which enable actions on the altered intentions. Avis and associates found that those at highest risk were the least accurate in their perceptions of risk, but that feedback of Health Hazard Appraisal results somewhat modified that perception.[217] We were unable to detect any relationship between the degree of concern about future heart disease and whether offspring had unknown cholesterol levels or feedback of high or normal levels. This may have been due to insufficient power since the group with known high cholesterol levels was very small. Level of concern was also not associated with any of the other risk factors or total number of risk factors, although it was associated with reported stress levels and dietary change.

Most of the progeny in our survey were late adolescents and young adults, a group in whom there has been little debate about the value of screening in those with a positive family history. However, we also gathered some information about younger children. The Canadian guidelines have not generally dealt with cholesterol screening in children. In the US there has been vigorous debate about whether all children should be screened,[103,106,164,165,167,168] or only those with a family history of ischemic heart disease or hyperlipidemia,[145,137] or none at all.[175] One of the concerns with measurement of risk factors in children is the labelling effect, although children who have a parent with early heart disease may already be "labelled".[175] We were unable to document this labelling effect in terms of the level of concern of about future heart disease in children in whom a parent is known to have heart disease. We found that the large majority of children never even think about their own risk.

Because of the personal and social costs of early coronary heart disease, and because some of the risk factors in those with a familial predisposition can be modified, it is important to develop more effective methods to identify the offspring of those with early CHD and enhance their adoption of preventive measures. To function well, the present system demands either an exemplary effort by practitioners to schedule assessments of offspring (who may not be members of the physician's practice) once premature CHD is recognized in a parent, or an unusual prescience among offspring about the risks entailed by the parent's disease, which then leads them to request an appointment. An alternative to the current method would be a program of education, risk factor assessment, and intervention - initiated perhaps by a public health group, a heart association, or a cardiac facility - which would actively offer risk factor assessment and counselling to other family members soon after an index case in a family developed clinically important premature CHD. Research into this or other preventive strategies, however, has the obligation to measure whether the intervention leads to the desired changes in behavior, without adding an unhealthy psychological burden to those identified as being at increased risk.

Risk factor management in patients

The survey confirmed the important role of smoking in premature coronary disease: only 8% of the male patients had never smoked. While almost two thirds of smokers (64%) have quit smoking, mostly in the last five years, 31% of this very high risk population continue to smoke. Given than 92% of these smokers have tried unsuccessfully to quit in the previous year, but less than a third mention quitting smoking as their major plan for health improvement in the coming year, this would comprise an excellent target group for smoking cessation interventions. Although we did not directly ask whether their physicians had advised them to stop smoking, the fact that less than a third of smokers mentioned this as part of the advice they were given for hypertension and hypercholesterolemia suggests that physicians may also need to play a greater role in this regard. Since the offspring of current smokers are approximately twice as likely to be current smokers (OR = 2.3), parental quitting might also encourage higher quit rates among the offspring.

Continued smoking may be a marker for lack of compliance with other health instructions. We found that current smokers were less likely to have changed their diets, more likely to report that they should be trying to lower cholesterol levels but haven't really tried, and more likely to be doing nothing to control their cholesterol levels when so advised. However, there was no significant difference in the proportion actively trying to control their blood pressure, a primarily pharmacologic intervention. Failure of a cardiac patient to quit smoking should thus trigger in the physician's mind that this patient may also require more intensive intervention to achieve compliance with other behavioral changes.

Virtually all the index cases had had blood pressure and cholesterol measurements within the previous two years. The difference in approaches to treatment for those with high levels of cholesterol and blood pressure is noteworthy. The primary mode of therapy for hypertension remains pharmacologic, with only a minority of patients having been advised on nonpharmacologic methods such as diet, weight loss, and stress avoidance. For high cholesterol the primary therapeutic modality was reduction of saturated fat and other dietary changes, with only a minority advised to take medicine.

Comparisons to other surveys

Previous surveys of physicians have shown low screening rates for serum cholesterol in adults ages 20 to 39. Langner and colleagues found that 56% of physicians in the Ottawa area routinely measured cholesterol levels in adults over the age of 30, but a further 24% measured serum cholesterol levels in a more limited age range, usually 40 to 60 years.[191] Similarly, Tannenbaum et al. found in a national survey that most physicians tested less than half of their patients aged 20 to 39 who have only one CHD risk factor.[192] For patients with more than one CHD risk factor 75% of physicians reported testing most of their male patients and 62% most of their female patients. Our findings from interviewing individuals in this age group with CHD risk factors tend to corroborate the low screening rates reported by the physicians. One may argue though that the low screening rates by family physicians are appropriate, since screening should be restricted to high risk cases. Our results show that screening is not taking place efficiently even in high risk families.

We were able to compare the present survey to the Ottawa Carleton Heart Beat Survey for individuals of the same age range. We were also able to examine within that community survey those who had a family history of heart disease and those who did not. The one area in which a difference was noted was in the higher rates of cholesterol screening in the current survey. Apart from chance or bias, two explanations are possible. The difference may reflect more intensive screening of family members than other members of the population, in accordance with the recommendations outlined in the Report on the Detection and Management of Asymptomatic Hypercholesterolemia by the Task Force on the Use and Provision of Medical Services.[77] Alternatively, it may reflect a general increase in the level of cholesterol screening over time, since the present survey was conducted three years later.

The fact that there was no difference in cholesterol screening rates within the Ottawa Carleton Heart Beat Survey between those with and without a family history of heart disease may suggest that the latter explanation is more likely. This may reflect gradual adoption of lipid guidelines.

The prevalence of smoking in the present survey can also be compared to national data. "Canadians and Smoking: An Update" presents smoking data by education, age and sex for Canada in 1989.[222] Nationally, 32% of the population are current smokers. In the 15-34 age group 33.4% are smokers and 18.2% are former smokers. Higher rates of smoking are seen in the Heart Health Survey data available from nine provinces: only 34% of those in the 18 to 34 year age group never smoked, 27% are former smokers, and 39% current cigarette smokers.[223] Comparing our survey to the national data, 37% are smokers but only 13.2% are former smokers. Thus smoking rates are similar to the national population with a trend toward lower rates of quitting. However, there may be further reason for concern about smoking rates in this high risk population. Although rates are similar between the Ottawa Carleton Heart Beat Survey and the present survey, more recent telephone surveys in Ottawa Carleton, including those done at the same time as the present survey, have shown a marked decline in smoking. The general population rates fell from 33% in 1987 to 20% in 1990 (unpublished data: G. Dunkley). In that case, our findings would suggest a substantially higher smoking rate in this high risk group than in the general population.

The Canadian Heart Health Survey also provided some data on knowledge of smoking as a cause of heart disease. Fifty-eight percent of smokers and 41% of nonsmokers knew that smoking causes heart disease. Unfortunately these figures were only available age-standardized to the 1986 Canadian population. In our survey, among 16 to 39 year olds, 59% listed smoking as a cause of heart disease.

Methodologic considerations

The present study has some limitations. The results may not be applicable to other communities and in particular other countries which practice more aggressive screening strategies. The limited sample size precluded some multivariate analyses. We excluded families of patients who had died. Premature death from coronary heart disease could be a stronger motivator for risk factor assessment and behaviour change than simply illness. Nevertheless, the offspring of living parents with premature IHD are also at high risk.

We obtained information on children under age 16 through their parents. This limited the type and amount of information we could obtain, and some may be inaccurate, particularly in the areas of diet and smoking. We lacked actual measurements of risk factors and had to rely only on self-report. Weight is known to be underestimated, and men are known to overestimate the amount of exercise they do. We had no independent validation of responses, and reported dietary changes may have been based on changes respondents thought they should have made, rather than what they actually did. However, we did get proxy responses from parents about their children under 16. Reduction of fat in the diet was reported by 33% of offspring 16 years and over by self- report, and 29% of offspring under 16 by proxy report. The similarity of reporting of changes among the older and younger offspring was noted with other variables as well, even when the parents' own reports of changes were quite different. There is thus reason to believe that the results are within reasonable ranges of the true values.

Although the questionnaire used questions from previously validated surveys, there was no independent validation of the questionnaire as a whole (other than face validity) and no test-retest reliability measures. Offspring were not interviewed twice to examine the reliability of their responses, and physician records were not examined to validate the reported rates of cholesterol and blood pressure measurement. However, even if the latter measurements had been performed in a higher proportion, few of the offspring were aware of the results.

One methodologic strength of the study is the enhanced generalizability through the ability to target most of the MI's in a city and the availability of data on non-respondents. Information on the databases from which we drew our study population (coronary angiography and post-MI patients) enabled us to characterize those who could not be contacted or who did not respond. Since the refusal rate was low and no clinically or statistically important differences emerged, the survey was likely representative of adults with premature ischemic heart disease and their offspring.

An additional strength was the ability to compare respondents on numerous variables to a city-wide random sample of respondents of similar age. Overall, there were minimal differences between the two groups. The one difference which emerged was in rates of cholesterol screening, which could be due to the time delay between the two surveys.

Conclusions

The present study shows that the Canadian approach to selective cholesterol screening is not sufficiently identifying high risk families. The low rates of cardiovascular risk factor assessment and management identified in this survey represent missed opportunities for primary prevention. However, there are alternatives to universal screening. We suggest active identification of family members each time an index patient develops clinically important premature ischemic heart disease. This should be followed by a coordinated program of education, risk factor assessment and intervention. Further research is needed to determine the efficacy of different interventions in improving the risk factor profile and to develop education tools that achieve the desired effects without adding psychological burden to those identified as being at increased risk.

References

1. Nichols E, Nair C, MacWilliam L et al. Cardiovascular Disease in Canada (cat no 82-544), Statistics Canada, Ottawa, 1986.

2. Federal Provincial Working Group on the Prevention and Control of Cardiovascular Disease in Canada. Promoting Heart Health in Canada: Report of the Federal Provincial Working Group on the Prevention and Control of Cardiovascular Disease in Canada. Department of National Health and Welfare, Ottawa, 1987.

3. Tyroler HA. Total serum cholesterol and ischemic heart disease risk in clinical trials and observational studies. Am J Prev Med 1985;1:18-24.

4. Tyroler HA. Review of lipid lowering clinical trials in relation to observational epimemiological studies. Circulation 1987;76(3):515-522.

5. Nora JJ, Lortscher RH, Spangler RD, Nora AH, Kimberling WJ. Genetic-epidemiologic study of early-onset ischemic heart disease. Circulation 1980;61:503-508.

6. Rissanen AM, Nikkila E. Coronary artery disease and its risk factors in families of young men with angina pectoris and in controls. Br Heart J 1977;39:875-933.

7. Rissanen AM. Familial occurrence of coronary heart disease: effect of age at diagnosis. Am J Cardiol 1979;44:60-66.

8. Neufeld HN, Goldbourt U. Coronary heart disease: genetic aspects. Circulation 1983;67:943.

9. Phillips RL, Lilienfeld AM, Diamond EL, Kagan A. Frequency of coronary heart disease and cerebrovascular accidents in parents and sons of coronary heart disease index cases and controls. Am J Epidemiol 1974;100:87-100.

10. Scholtz RI, Rosenman RH, Brand RJ. The relationship of reported family history to the incidence of coronary heart disease in the Western Collaborative Group Study. Am J Epidemiol 1975;102:350-356.

11. Cambien F, Richard JL, Ducimetiere P. Familial history of coronary heart diseases and high blood pressure in relation to the prevalence of risk factors, and the incidence of coronary heart disease. Rev Epidemiol Sante Publ 1980;28:21-37.

12. Snowden CB, McNamara PM, Garrison M, Feinleib M, Kannel WB, Epstein FH. Predicting coronary heart disease in siblings - A multivariate assessment. Am J Epidemiol 1982;115:217-222.

13. Barrett-Connor E, Khaw KT. Family history of heart attack as an independent predictor of death due to cardiovascular disease. Circulation 1984;69:1065.

14. Becker DM, Levine DM. Risk perception, knowledge, and lifestyles in siblings of people with premature coronary disease. Am J Prev Med 1987;3(1):45-50.

15. Becker DM, Becker LC, Pearson TA, Fintel DJ, Levine DM, Kwiterovich PO. Risk factors in siblings of people with premature coronary heart disease. J Am Coll Cardiol. 1988 Nov, 12(5): 1273-1280.

16. Perkins, KA. Family history of coronary heart disease: is it an independent risk factor? Am J Epidemiol 1986;124:182-193.

17. Shea S, Ottman R, Gabrieli C, Stein Z, Nichols A. Family history as an independent risk factor for coronary artery disease. J Am Coll Cardiol 1984;4:793-801.

18. Berg K, Dahlen G, Borrensen AL. Lp(a) phenotypes, other lipoprotein parameters, and a family history of coronary heart disease in middle-aged males. Clin Genet 1979;16:347-352.

19. Deutscher S, Ostrander LD, Epstein FH. Familial factors in premature coronary heart disease - A preliminary report from the Tecumseh Community Health Study. Am J Epidemiol 1970;91:223-243.

20. Epstein FH. Hereditary aspects of coronary heart disease. Am Heart J 1964;67:445-456.

21. Glueck CJ, Fallat RW, Tsang R, Buncher CR. Hyperlipidemia in progeny of parents with myocardial infarction before age 50. Am J Dis Child 1974;127:70-75.

22. Slack J, Evans KA. The increased risk of death from ischemic heart disease in first degree relatives of 121 men and 96 women with ischemic heart disease. J Med Genet 1966;3:239-257.

23. Morrison JA, Khoury P, Mellies MJ, Kelly K, Glueck CJ. Identifying CHD risk factors in children: Intrafamilial lipoprotein correlations. Prev Med 1980;9:484-495.

24. Stamler R, Stamler J, Reidlinger WF, Algera G, Roberts RH. Family (parental) history and prevalence of hypertension. JAMA 1979;241:43-46.

25. Feinleib M, Garrison JR, Stallones L. A comparison of blood pressure, total cholesterol and cigarette smoking in patients in 1950 and their children in 1979. Am J Epidemiol 1979;110:291-303.

26. Deutscher S, Epstein FH, Keller JB. Relationships between familial aggregation of coronary heart disease and risk factors in the general population. Am J Epidemiol 1969;89:510-520.

27. Phillips RL, Lilienfeld AM, Diamond EL, Kagan A. Frequency of coronary heart disease and cerebrovascular accidents in parents and sons of coronary heart disease index cases and controls. Am J Epidemiol 1974;100:87-100.

28. Feinleib M, Kannel WB. Relation of parental history of coronary heart disease to risk factors in young adults. The Framingham Offspring Study. (abstr) Circulation 1976;54(suppl II):II-52.

29. Schieken RM. Identification of high risk relatives for coronary heart disease. J Am Coll Cardiol 1988;12(4): 1110-3.

30. Deutscher S, Epstein FH, Kjelsberg MO. Familial aggregation of factors associated with coronary heart disease. Circulation 1966;33:911-924.

31. Hayes C, Tyroler H, Cassel J. Family aggregation of blood pressure in Evans County, Georgia. Arch Intern Med 1971;128:965-75.

32. Shear CL, Frerichs RR, Weinberg R, Berenson GS. Childhood sibling aggregation of coronary artery disease risk factor variables in a biracial community. Am J Epidemiol 1978;107:522-528.

33. Zinner SH, Levy PS, Kass EH. Familial aggregation of blood pressure in childhood. New Engl J Med 1971;284:401-404.

34. Lee J, Lauer RM, Clarke WR. Lipoproteins in the progeny of young men with coronary artery disease: Children with increased risk. Pediatrics 1986;78(2):330-7.

35. Morrison JA, Namboodiri K, Green P, Martin J, Glueck CJ. Familial aggregation of lipids and lipoproteins and early identification of dyslipoproteinemia: The Collaborative Lipid Research Clinics Study. JAMA 1983;250:1860-8.

36. Morrison JA, Horvitz R, Khoury YP, et al. Parental history of coronary heart disease, hypertension, diabetes and stroke: relationship to coronary heart disease risk factor variables in their adult children. Prev Med 1980;9:773-86.

37. Hennekens CH, Levine RS, Rosner B, et al. Aggregation of cholesterol among young families of men with premature myocardial infarction. J Chron Dis 1980;33:359-664.

38. Glueck CJ, Laskarzewski PM, Suchindran CM, et al. Progeny's lipid and lipoprotein levels by parental mortality. The Lipid Research Clinics Program Prevalence Study. Circulation 1986;73(suppl I):I51-I61.

39. Bodurtha JN, Schieken RM, Segrest J, Nance WE. HDL cholesterol subfractions in adolescent twins. Pediat 1987;79:181-189.

40. Austin MA, King MC, Bawol RD, et al. Risk factors for coronary heart disease in adult female twins: genetic heritability and shared environmental influences. Am J Epidemiol 1987;125:308-318.

41. Schrott HB, Clarke WR, Wiebe DA, et al. The Muscatine Family Studies Program: Increased coronary mortality in relatives of hypercholesterolemic school children. Circulation 1979;59:320-326.

42. Moll PP, Sing CF, Weidman WH, et al. Total cholesterol and lipoproteings in school children: Prediction of coronary heart disease in adult relatives. Circulation 1983;67(1):127-34.

43. Goldstein JL, Alber JJ, Schrott HG, Hazzard WR, Bierman E, Motulsky AG. Plasma lipid levels and coronary heart disease in adult relatives of newborns with normal and elevated cord blood lipids. Am J Hum Genet 1974;26:727.

44. Miller NE, Nestel PJ, Boulton TJC, Dwyer T, Leitch D. Cord blood high density lipoprotein concentration in 1797 births: Relationship to family history of coronary heart disease. J Chronic Dis 1981;34:119.

45. Rose G. Familial patterns in ischemic heart disease. Br J Prev Soc Med 1964;18:75-80.

46. Nikkila EA, Aro A. Family study of serum lipids and lipoproteins in coronary heart disease. Lancet 1973;1:954-958.

47. Garrison RJ, Castelli WP, Feinleib M, et al. The association of total cholesterol, triglycerides and plasma lipoprotein cholesterol levels in first degree relatives and spouse pairs. Am J Epidemiol 1979;110:313-321.

48. Sosenko JM, Breslow JL, Ellison RC, Miettinen OS. Familial aggregation of total cholesterol, high density lipoprotein cholesterol and total triglyceride levels in plasma. Am J Epidemiol 1980;112:656-660.

49. Anderson AJ, Loeffler RF, Barboriak JJ, Rimm AA. Occlusive coronary artery disease and parental history of myocardial infarction. Prev Med 1979;8:419-428.

50. Vlietstra RE, Frye RL, Kronmal RA, Sim DA, Tristan FE, Killip T. Tisk factors and angiographic coronary artery diseas: a report from the Coronary Artery Surgery Study. Circulation 1980;62:254-261.

51. Holmes DR, Elveback LR, Frye RL, Kottke BA, Ellefson RD. Association of risk factor variables and coronary artery disease documented with angiography. Circulation 1981;63:293-299.

52. Hamby RI. Hereditary aspects of cornary artery disease. Am Heart J 1981;101:639-649.

53. Ten Kate LP, Boman H, Daiger SP, Motulsky AG. Familial aggregation of coronary heart disease and its relation to known genetic risk factors. Am J Cardiol 1982;50:945-53.

54. Rissanen AM, Nikkila EA. Aggregation of coronary risk factors in families of men with fatal and non-fatal coronary heart disease. Br Heart J 1979;42:373-380.

55. Hedstrand H, Aberg H. Familial history in males at low and high risk for cardiovascular disease. Prev Med 1978;7:15-21.

56. Forde OH, Thelle DS. The Tromso Heart Study: risk factors for coronary heart disease related to the occurrence of myocardial infarction in first degree relatives. Am J Epidemiol 1977;105:192-199.

57. Thelle DS, Forde OH. The cardiovascular study in Finmark Country: coronary risk facotrs and the occurrence of myocardial infarction in first degree relatives and in subjects of different ethnic origin. Am J Epidemiol 1979;110:708-715.

58. Laskarzewski P, Morrison JA, Horvitz R, et al. The relationship of parental history of myocardial infarction, hypertension, diabetes and stroke to coronary heart disease risk factors in their adult progeny. Am J Epidemiol 1981;113:290-306.

59. Hopkins PN, Williams RR. A survey of 246 suggested coronary risk factors. Atherosclerosis 1981;40:1-52.

60. Khaw K, Barrett-Conner E. Family history of heart attack: a modifiable risk factor? Circulation 1986;74:239-44.

61. Kwiterovich PO, Frederickson Ds, Levy RI. Familial hypercholestrolemia: A study of its biochemical, genetic and clinical presentation in childhood. J Clin Invest 1974;53:1237-1249.

62. Goldstein JL, Schrott HG, Hazzard WR, et al. Hyperlipidemia in coronary heart disease: II. Genetic analysis of lipid levels in 176 families and delineation of a new inherited disorder, combined hyperlipidemia. J Clin Invest 1973;52:1544-1568.

63. Goldstein JL, Brown MS. Familial hypercholesterolemia. In: Stanbury JB, Wyngaarden JB, Fredrickson DS, Goldstein JL, Brown MS. The Metabolic Basis of Inherited Disease, 5th ed. New York: McGraw-Hill, 1983;672-712.

64. Consensus Conference. Lowering blood cholesterol to prevent heart disease. JAMA 1985; 253:2080-2090.

65. Castelli WP, Garrison RJ, Wilson PWF, et al. Incidence of coronary heart disease and lipoprotein cholesterol levels: The Framingham Study. JAMA 1986; 256:2835-2838.

66. Dyer AR, Stamler J, Paul O, et al. Serum cholesterol and risk of death from cancer and other causes in three Chicago epidemiologic studies. J Chronic Dis 1981; 34:249-260.

67. Gordon T, Garcia-Palmieri MR, Kagan A, et al. Differences in coronary heart disease in Framingham, Honolulu and Puerto Rico. J Chronic Dis 1974; 27:239-244.

68. Keys A. Coronary heart disease in seven countries. Circulation 1970; 41(Suppl I):1-211.

69. Kozarevic D, McGee D, Vojvodic N, et al. Serum cholesterol and mortality: The Yugoslavia Cardiovascular Disease Study. Am J Epidemiol 1981; 114:21-28.

70. Kleinbaum DG, Kupper LL, Cassel JC, et al. Multivariate analysis of risk of coronary heart disease in Evans County, Georgia. Arch Intern Med 1971; 128:943-948.

71. Pelkonen R, Nikkila EA, Koskinen S, et al. Association of serum lipids and obesity with cardiovascular mortality. Br Med J 1977; 2:1185-1187.

72. Rhoads GG, Gulbrandsen CL, Kagan A. Serum lipoproteins and coronary heart disease in a population of Hawaii Japanese men. N Engl J Med 1976; 294:293-298.

73. The Pooling Project Research Group. Relationship of blood pressure, serum cholesterol, smoking habits, relative weight and ECG abnormalities to incidence of major coronary events: final report of the Pooling Project. J Chronic Dis 1978; 31:201-306.

74. Wallace RB, Anderson RA. Blood lipids, lipid-related measures, and the risk of atherosclerotic cardiovascular disease. Epidemiol Rev 1987; 9:95-119.

75. Simons LA. Interrelations of lipids and lipoproteins with coronary mortality in 19 countries. Am J Cardiol 1986; 57:5G-10G.

76. LaRosa JC, Hunninghake D, Bush D, et al. The Cholesterol Facts: A summary of the evidence relating dietary fats, serum cholesterol, and coronary heart disease. A Joint statement by the American Heart Association and the National Heart, Lung, and Blood Institute. Circulation 1990;81(5):1721-1733.

77. Toronto Working Group on Cholesterol Policy. Detection and management of asymptomatic hypercholesterolemia. A policy document prepared for The Task Force on the Use and Provision of Medical Services. The Ontario Ministry of Health and the Ontario Medical Association. March, 1989.

78. Christakis G, Rinzler SH, Archer M, et al. The Anti-Coronary Club: A Dietary Approach to the Prevention of Coronary Heart Disease - A Seven-year Report. Am J Public Health 1966; 56:299-314.

79. Miettinen M, Turpeinen O, Karvonen M, Elosuo R, Paavilainen E. Effect of Cholesterol-Lowering Diet on Mortality from Coronary Heart-Disease and Other Causes: A Twelve-year Clinical Trial in Men and Women. Lancet 1972; 2:835-838.

80. Turpeinen O, Karvonen M, Pekkarinen M, Miettinen M, Elosuo R, Paavilainen E. Dietary Prevention of Coronary Heart Disease The Finnish Mental Hospital Study. Int J Epid 1979; 8:99-118.

81. Dayton S, Pearce ML, Hashimoto S, Dixon W, Tomiyasu U. A Controlled Clinical Trial of a Diet High in Unsaturated Fat in Preventing Complications of Atherosclerosis. Circulation 1969;39/40:(Suppl.2)1-63.

82. Brewer ER, Ashman PL, Kuba K. The Minnesota Coronary Survey: Composition of the diets, adherence and serum lipid response. Circulation 1975; 52(II):269.

83. Frantz ID, Dawson EA, Ashman PL et al. Test of effect of lipid lowering by diet on cardiovascular risk. Arteriosclerosis 1989;9:129-135.

84. Committee of Principal Investigators. W.H.O. Cooperative Trial on Primary Prevention of Ischemic Heart Disease Using Clofibrate to Lower Serum Cholesterol: Mortality Follow-Up. Lancet 1980; 2:379-385.

85. Lipid Research Clinics Program. The Lipid Research Clinics Coronary Primary Prevention Trial Results. I. Reduction in Incidence of Coronary Heart Disease. JAMA 1984; 251:351-364.

86. Lipid Research Clinics Program. The Lipid Research Clinics Coronary Primary Prevention Trial Results. II. The Relationship of Reduction in Incidence of Coronary Heart Disease to Cholesterol Lowering. JAMA 1984; 251:365-374.

87. Manninen V, Elo O, Frick MH, et al. Lipid Alterations and Decline in the Incidence of Coronary Heart Disease in the Helsinki Heart Study. JAMA 1988; 260:641-651.

88. Dorr AE, Gundersen K, Schneider JC, Spencer TW, Martin WB. Colestipol Hydrochloride in hypercholesterolemic patients - Effect on serum cholesterol and mortality. J Chronic Dis 1978; 31:5-14.

89. Multiple Risk Factor Intervention Trial Research Group. Multiple Risk Factor Intervention Trial: Risk Factor Changes and Mortality Results. JAMA 1982; 248:1465-1477.

90. Kornitzer M, DeBacker G, Dramizx M et a;. Belgian Heart Disease Prevention Project: Incidence and Mortality Results. Lancet 1983; 1:1066-1070.

91. Rose G, Tunstall-Pedoe HD, Heller RF. UK Heart Disease Prevention Project: Incidence and Mortality Results. Lancet 1983; 1:1062-1065.

92. World Health Organisation European Collaborative Group. European Collaborative Trial of Multifactorial Prevention of Coronary Heart Disease: Final Report on the 6-Year Results. Lancet 1986; 1:869-872.

93. World Health Organisation European Collaborative Group. Multifactorial trial in the prevention of coronary heart disease: 3. Incidence and mortality results. Euro Heart J 1983; 4:141-147.

94. Hjermann I, Byre KV, Holme I, Leren P. Effect of diet and smoking intervention on the incidence of coronary heart disease: Report from the Oslo Study Group of a Randomised Trial in Healthy Men. Lancet 1981; 2:1303-1810.

95. Hjermann I, Holme I, Leren P. Oslo Study Diet and Antismoking Trial: Results after 102 months. Am J Med 1986; 80(suppl 2A):7-11.

96. Holme I, Enger SC, Helgeland A, et al. Risk factors and raised atherosclerotic lesions in coronary and cerebral arteries: Statistical analysis from the Oslo Study. Arteriosclerosis 1981;1:250-256.

97. Holme I, Hjermann I, Helgelane A, Leren P. The Oslo Study: Diet and Antismoking Advice. Additional Results from a 5-Year Primary Preventive Trial in Middle-Aged Men. Prev Med 1985; 14:279-292.

98. Leren P, Helgeland A, Hjermann I, Holme I. The Oslo study: CHD risk factors, socioeconomic influences, and intervention. Am Heart J 1983; 106:1200-1206.

99. Leren P. The Oslo diet-heart study: eleven-year report. Circulation 1970;42:35-42.

100. Schatzkin A, Hoover RN, Taylor PR et al: Serum cholesterol and cancer in the NHANES I epidemiologic followup study. Lancet 1987;2:298-301.

101. Isles CG, Hole DJ, Gillis CR et al: Plasma cholesterol, coronary heart disease, and cancer in the Renfrew and Paisley survey. BMJ 1989;298:920-924.

102. Muldoon M, Manuck S, Matthews K. Lowering cholesterol concentrations and mortality: a quantitative review of primary prevention trials. Br Med J 1990;301:309-314.

103. Wynder EL, Berenson GS, Strong WB, Williams C, eds. Coronary artery disease prevention: cholesterol, a pediatric perspective. Prev Med 1989;18:323-409.

104. Kwiterovich PO. Biochemical, clinical, epidemiologic, genetic, and pathologic data in the pediatric age group relevant to the cholesterol hypothesis. Pediatrics 1986;78(2):349-361.

105. Strong WB, Dennison BA. Pediatric preventive cardiology: atherosclerosis and coronary heart disease. Pediatr Rev 1988;9(10):303-314.

106. Cresanta JL, Burke GL, Downey AM, Feedman DS, Berenson GS. Prevention of atherosclerosis in childhood. Pediatr Clin North Am 1986;33:835-838.

107. Newman WP, Freedman DS, Voors AW, et al. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis. The Bogalusa Heart Study. N Engl J Med 1986;314(3):138-144.

108. Knuiman JT, Hermus RJJ, Hautvast JGAJ. Serum total and high-density lipoprotein cholesterol concentrations in rural and urban boys from 16 countries. Atheroslerosis 1980;36:529-537.

109. Okuni M, Hayashi K, Kiryu S, Yamauchi K. Risk factors of arteriosclerosis in Japanese children. Japan Circ J 1986;44:69-75.

110. Enos W, Holmes R, Beyer J. Coronary disease among United States soldiers killed in action in Korea. JAMA 1953;152:1090-1092.

111. McNamara JJ, Molat MA, Stremple JF, et al. Coronary artery disease in combat casualties in Vietnam. JAMA 1974;216:1185-1187.

112. Strong JP, McGill HC Jr. The pediatric aspects of atherosclerosis. J Atheroscler Res 1969;9:251-266.

113. Tamir I, Bojamower Y, Leutow O, et al. Serum lipids and lipoproteins in children from families with early coronary disease. Arch Dis Child 1972;23:808-810.

114. Chase HP, O'Quin RJ, O'Brien P. Screening for hyperlipidemia in childhood. JAMA 1974;230:1535-1537.

115. Weinberg R, Webber LS, Berenson GS. Hereditary and environmental influences on cardiovascular risk factors for children. Am J Epidemiol 1982;116:385-393.

116. Van Natta P, Jenkin J, Chase G, et al. The East Baltimore Study. II. Familial aggregation of plasma lipids in juveniles in a black inner city population. Amer J Epidemiol 1981;114:385-397.

117. Williams RR, Hasstedt SJ, Wilson DE, et al. Evidence that men with familial hypercholesterolemia can avoid early coronary death. An analysis of 77 gene carriers in four Utah pedigrees. JAMA 1986:255:219-224.

118. Nicklas TA, Farris RP, Smoak CG, et al. Dietary factors relate to cardiovascular risk factors in early life. Bogalusa Heart Study. Arteriosclerosis. 1988 Mar-Apr, 8(2): 193-199.

119. Nicklas TA, Farris RP, Major C, et al. Cardiovascular risk factors from birth to seven years of age: Bogalusa Heart Study. V. Dietary intakes. Pediatrics 1987;80(suppl 5, pt 2):797-806.

120. Webber LS, Cresanta JL, Voors AW, Berenson GS. Tracking of cardiovascular disease risk factor variables in school-age children. J Chronic Dis 1983;36:647-660.

121. Webber LS, Srinivasan SR, Wattigney WA, Berenson GS. Tracking of serum lipids and lipoproteins from childhood to adulthood: The Bogalusa Heart Study. Am J Epidemiol 1991;133(9):884-899.

122. Freedman DS, Shear CL, Srinivasan SR, Webber LS, Berenson GS. Tracking of serum lipids and lipoprotiens over an eight-year period: The Bogalusa Heart Study. Prev Med 1985;14:203-216.

123. Freedman DS, Srinivasan SR, Shear CL, et al. Cigarette smoking initiation and longitudinal changes in serum lipids and lipoproteins in early adulthood: The Bogalusa Heart Study. Am J Epidemiol 1986;124:207-219.

124. Freedman DS, Srinivasan SR, Harsha DW, Webber LS, Berenson GS. Relation of body fat patterning to lipid and lipoprotein concentrations in children and adolescents: the Bogalusa Heart Study. Am J Clin Nutr 1989; 50:930-939.

125. Lauer RM, Clarke WR. Use of cholesterol measurements in childhood for the prediction of adult hypercholesterolemia. JAMA 1990;264(23):3034-3038.

126. Lauer RM, Lee J, Clarke WR. Factors affecting the relationship between childhood and adult cholesterol levels: The Muscatine Study. Pediatrics 1988;82(3):309-318.

127. Lauer RM, Lee J, Clarke WR. Predicting adult cholesterol levels from measurements in childhood and adolescence: The Muscatine Study. Bull N Y Acad Med 1989;65(10):1127-1142, discussion 1154-1160.

128. Clarke WR, Schrott HG, Leaverton PE, Connor WE, Lauer RM. Tracking of blood lipids and blood pressure in school-age children: The Muscatine Study. Circulation 1978;58:626-634.

129. Burns TL, Moll PP, Lauer RM. Increased familial cardiovascular mortality in obese schoolchildren. The Muscatine Ponderosity Family Study. Pediatrics 1992;89(2):262-268.

130. Burns TL, Moll PP, Lauer RM. The relation between ponderosity and coronary risk factors in children and their relatives. The Muscatine Ponderosity Family Study. Am J Epidemiol. 1989 May, 129(5): 973-987.

131. Clarke WR, Schrott HG, Leaverton PE, Connor WE, Lauer RM. Tracking of blood lipids and blood pressure in school-age children: The Muscatine Study. Circulation 1978;58:626-634.

132. Report of a World Health Organization expert committee. Prevention of coronary heart disease. WHO Tech Rep Ser 1982; 678:i-53.

133. Summary of the presentation to the Candian Consensus Conference on Cholesterol, March 9-10, 1988, Ottawa, Canada. 1988. Paper 10: Logan A. The conclusions of the Canadian Task Force on the Periodic Health Examination. Update on cholesterol.

134. Canadian Consensus Conference on Cholesterol: Final Report. The Canadian Consensus Conference on the Prevention of Heart and Vascular Disease by Altering Serum Cholesterol and Lipoprotein Risk Factors. Can Med Assoc J 1988; 139(Suppl):1-8.

135. National Health Foundation of New Zealand Advisory Group. Guidelines on blood lipid screening and management of hyperlipidaemia. NZ Med J 1988; 101:629-631.

136. Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Arch Intern Med 1988; 148:36-69.

137. Canadian Task Force on the Periodic Health Examination. Periodic health examination, 1993 update: 2. Lowering the blood total cholesterol level to prevent coronary heart disease. CMAJ 1993;148(4):521-538.

138. Grundy SM, Greenland P, Herd A, et al. Cardiovascular and risk factor evaluation of healthy American adults: A statement for physicians by an ad hoc committee appointed by the Steering Committee, American Heart Association. Circulation 1987; 75:1340A-1360A.

139. Shepherd J, Betteridge DJ, Durrington P et al. Strategies for reducing coronary heart disease and desirable limits for blood lipid concentrations: guidelines of the British Hyperlipidaemia Association. Br Med J 1987; 295:1245-1246.

140. Study Group, European Atherosclerosis Society. Strategies for the prevention of coronary heart disease: A policy statement of the European Atherosclerosis Society. Eur Heart J 1987; 8:77-88.

141. The British Cardiac Society Working Group on Coronary Prevention: conclusions and recommendations. Br Heart J 1987; 57:188-189.

142. Report of the US Preventive Services Task Force. Guide to Clinical Preventive Services: An assessment of the effectiveness of 169 interventions. Baltimore, Md.: Williams and Wilkins, 1989.

143. Garber AM, Sox HC, Littenberg B. Screening asymptomatic adults for cardiac risk factors: the serum cholesterol level. Ann Intern Med 1989; 110:622-639.

144. National Cholesterol Education Program. Report of the expert panel on blood cholesterol levels in children and adolescents. Pediatrics 1992;89(3):(suppl)525-577.

145. American Academy of Pediatrics Committee on Nutrition. Indications for cholesterol testing in children. Pediatrics 1989;83:141-142.

146. A Joint Statement for Physicians by the Committee on Atherosclerosis and Hypertension in Childhood of the Council of Cardiovascular Disease in the Young and the Nutrition Committee, American Heart Association. Diagnosis and treatment of primary hyperlipidemia in childhood. Circulation 1986;74(5):1181A-1188A.

147. Rose G. Sick individuals and sick populations. Int J Epid 1985;14:32-38.

148. Slater CH, Carlton W, Ramirez G, Ashton C. Heart disease - Strategies for prevention. Evaluation and the Health Professions 1987;10:255-286.

149. McCormick J, Skrabenek P. Coronary heart disease is not preventable by population interventions. Lancet 1988;ii:839-841.

150. Gunning-Schepers LJ, Barendregt JJ, van der Maas PJ. Population interventions reassessed. Lancet 1989;i:479-481.

151. Kottke TE, Gatewood LC, Wu SC, Park HA. Preventing heart disease: is treating the high risk sufficient? J Clin Epidemiol 1988;41:1083-1093.

152. Wilhelmsen L, Berglund G, Elmfeldt D et al. The multifactor primary prevention trial in Goteborg, Sweden. Euro Heart J 1986;7:279-288.

153. Farquhar JW, Maccoby N, Wood PD, et al. Community education for cardiovascular health. Lancet 1977;i:1192-1195.

154. Becker MH (ed). The Health Belief Model and Personal Health Behavior. Health Educ Monogr 1974;2:326-473.

155. Ajzen I, Fishbein M. Understanding attitudes and predicting behavior. Englewood Cliffs, NJ: Prentice Hall, 1980.

156. Sutton SR: Fear arousing communications: A critical examination of theory and research. In: Eiser JR (ed): Social Psychology and Behavioral Medicine. New York: Wiley, 1982.

157. Rogers RW. A protection motivation theory of fear appeals and attitude change. J Psychol 1975;91:93-114.

158. Macdonald LA, Sackett DL, Haynes and Taylor DW. Labelling in hypertension: a review of the behavioural and psychological consequences. J Chron Dis 1984;37:933-942.

159. Monk M. Blood pressure awareness and psychological well-being in the Health and Nutrition Examination Survey. Clin Invest Med 1981;4:183-190.

160. Mann A. Factors affecting psychological state during one year on a hypertension trial. Clin Invest Med 1981;4:197-200.

161. Polk BF, Harlan LC, Cooper SP et al. Disability days associated with detection and treatment in a hypertension control program. Am J Epidemiol 1984;119:44-53.

162. Lefebvre RC, Hursey KG, Carleton RA. Labeling of participants in high blood pressure screening programs: Implications for blood cholesterol screenings. Arch Intern Med 1988;148:1993-1997.

163. Committee on Nutrition. Indications for cholesterol testing in children. Pediatrics 1989;83(1):141-142.

164. Forster JH. Elevated lipids: whom to screen and how to treat. Contemp Pediatr, April 1985;22-32.

165. Long TJ. Routine screening tests for the adolescent. Prim Care 1987;14:41-47.

166. Strong WB. You are a preventive cardiologist: The scope of pediatric preventive cardiology. AJDC 1989;143:1145.

167. Garcia RE, Moodie DS. Routine cholesterol screening in childhood. Pediatrics 1989;84:751-755.

168. Strong WB. Point-counterpoint: to test or not to test in pediatric practice. Pediatr Rev 1990;12:36-38.

169. Newman TB, Browner WS, Hulley SB. Children should not be routinely screened for high blood cholesterol [letter] Am J Dis Child 1990;144(8):851-852.

170. Robertson LD Jr. Routine cholesterol screening in childhood [letter] Pediatrics. 1990;85(5):894-895.

171. Barness LA. Point-counterpoint: to test or not to test in pediatric practice. Pediatr Rev 1990;12:35-36.

172. Davidson DM, Bradley BJ, Landry SM, Iftner CA, Bramblett SN. School-based blood cholesterol screening. J Pediatr Health Care 1989;3:3-8.

173. Dennison BA. Cholesterol screening? [letter] Pediatrics 1990;85(6):1125-1126.

174. Staines A. Another view of cholesterol screening [letter]. Pediatrics 1990;86(2):327-328.

175. Newman TB, Browner WS, Hulley SB. The case against childhood cholesterol screening. JAMA 1990;264(23):3039-3043.

176. Hulley SB, Newman TB. Position statement: Cholesterol screening in children is not indicated, even with positive family history. J Amer Coll Nutrition 1992;11(S):20S-22S.

177. Lifshitz F, Moses N. Growth failure: A complication of dietary treatment of hypercholesterolemia. Am J Dis Child 1989;143:537-542.

178. Lannon CM, Earp J. Parents' behavior and attitudes toward screening children for high serum cholesterol levels. Pediatrics 1992;89:1159-1163.

179. Hastrup JL, Hotchkiss AP, Johnson CA. Accuracy of knowledge of family history of cardiovascular disorders. Health Psychol 1989;3:3-8.

180. Meyerson DA, Santanello NC. Pediatric lipid screening based upon criteria of high-risk family history fails to identify two-thirds of higher-risk children. (Abstract) Circulation 1988;78(suppl II):383.

181. Dennison BA, Kikuchi DA, Srinivasan SR, Webber LS, Berenson GS. Parental history of cardiovascular disease as an indication for screening for lipoprotein abnormalities in children. J Pediatr 1989;115:186-194.

182. Dennison BA, Kikuchi DA, Srinivasan SR, Webber LS, Berenson GS. Serum total cholesterol screening for the detection of elevated low-density lipoprotein in children and adolescents: the Bogalusa Heart Study. Pediatrics 1990;85(4):472-479.

183. Griffin TC, Christoffel KK, Binns HJ, McGuire PR, Pediatric Practice Research Group. Family history as a predictive screen for childhood hypercholesterolemia. Pediatrics 1989;84:365-373.

184. Nauer AM. Pediatrician attends the National Institutes of Health consensus conference on lowering blood cholesterol. Pediatrics 1985;76:125-126.

185. Glueck CJ, McGill H, Shank RE, et al. Value and safety of diet modification to control hyperlipidemia in childhood and adolescence. Circulation 1978;58:381A-385A.

186. Nader PR, Tara HL, Sallis JF, et al. Adult heart disease prevention in childhood: a national survey of pediatricians' practices and attitudes. Pediatrics 1987;79:843-850.

187. Jacobson MS, Lillienfeld DE. The pediatrician's role in atherosclerosis prevention. J Pediatrics 1988;112(5):836-841.

188. Williford HN, Barfield BR, Lazenby RB, Olson MS. A survey of physicians' attitudes and practices related to exercise promotion. Prev Med 1992;21:630-636.

189. Vivier PM, Dietrich AJ, Witters LA. Management of elevated cholesterol levels by New Hampshire primary care physicians. J Fam Pract 1988;27(2):167-172.

190. Morris PD, Morris ER. Family practice residents' compliance with preventive medicine recommendations. Am J Prev Med 1988;4(3):161-165.

191. Langner NR, Hasselback PD, Dunkley GC, Corber SJ. Attitudes and practices of primary care physicians in the management of elevated serum cholesterol levels. Can Med Assoc J 1989;141:33-38.

192. Tannenbaum TN, Sampalis JS, Battista RN, Rosenberg S, Joseph L. Early detection and treatment of hyperlipidemia: Physician practices in Canada CMAJ 1990;875-81.

193. Vartiainen E, Tossavainen K, Viri L, Niskanen E, Puska P. The North Karelia Youth Programs. Ann NY Acad Sci 1991;623:332-349.

194. Tell GS, Vellar OD. Physical fitness, physical activity, and cardiovascular disease risk factors in adolescents: the Oslo Youth Study. Prev Med 1988;17(1):12-24.

195. Resnicow K, Cross D, Wynder E. The role of comprehensive school-based interventions: The results of four Know Your Body Studies. Ann NY Acad Sci 1991;623:285-298.

196. Berenson GS, Arbeit ML, Hunter SM, Johnson CC, Nicklas TA. Cardiovascular health promotion for elementary school children: The Heart Smart Program. Ann NY Acad Sci 1991;623:299-313.

197. Tamir D, Feurstein A, Brunner S, Halfon ST, Reshef A, Palti H. Primary prevention of cardiovascular diseases in childhood: changes in serum total cholesterol, high density lipoprotein, and body mass index after 2 years of intervention in Jerusalem schoolchildren age 7-9 years. Prev Med 1990;19(1):22-30.

198. Carleton RA, Sennett L, Gans KM et al. The Pawtucket Heart Health Program: Influencing adolescent eating patterns. Ann NY Acad Sci 1991;623:322-326.

199. Lefebvre RC, Peterson GS, McGraw SA, et al. Community intervention to lower blood cholesterol: The "Know Your Cholesterol" Campaign in Pawtucket RI. Health Educ Q 1986;13(2):117-129.

200. Luepker RV, Perry CL. The Minnesota Heart Health Program: Education for youth and parents. Ann NY Acad Sci 1991;623:314-321.

201. Mittelmark MB, Luepker RV, Jacobs DR et al. Community-wide prevention of cardiovascular disease: education strategies of the Minnesota Heart Health Program. Prev Med. 1986;15:1-17.

202. Perry CL, Klepp KI, Sillers C. Community-wide strategies for cardiovascular health: the Minnesota Heart Health Program youth program. Health Educ Res 1989;4:87-101.

203. Perry CL, Luepker RV, Murray DM et al. Parent involvement with children's health promotion: the Minnesota Home Team. Am J Publ Health 1988;78:1156-1160.

204. Stern MP, Farquhar JW, Maccoby N, Russell SH. Results of a two-year health education campaign on dietary behavior: The Stanford Three Community Study. Circulation 1976;54(5):826-833.

205. Farquhar JW. The Stanford Cardiovascular Disease Prevention Programs. Ann NY Acad Sci 1991;623:327-331.

206. Dobson AJ, Givvered RW, Leeder SR, Alexander HM, Young AF, Lloyd DM. Ischaemic heart disease in the Hunter Region of New South Wales, Australia: 1979-1985. Am J Epidemiol 1988;128:106-115.

207. Walker R, Heller R, Redman S, O'Connell D, Boulton J. Reduction of ischemic heart disease risk markers in the teenage children of heart attack patients. Prev Med 1992;21:616-629.

208. Krahn M, Naylor CD, Basinski AS, Detsky AS. Comparison of an aggressive (U.S.) and a less aggressive (Canadian) policy for cholesterol screening and treatment. Ann Int Med 1991;115(4):249-255.

209. Ottawa Carleton Regional Health Department. Report on the Ottawa-Carleton Heart Beat Survey. November, 1988.

210. Department of National Health and Welfare. The Active Health Report - Perspectives on Canada's Health Promotion Survey 1985. Ministry of Supply and Services 1987.

211. Corber S. A Report on the Health Status of Adults in Ottawa-Carleton. The Ottawa-Carleton Regional Health Unit Community Survey. Ottawa-Carleton Health Unit 1985.

212. Hopkins PN, Williams RR, Hunt SC. Magnified risks from cigarette smoking for coronary prone families in Utah. West J Med 1984;141:196-202.

213. Avis NE, McKinlay JB, Smith KW. Is cardiovascular risk factor knowledge sufficient to influence behavior? Am J Prev Med 1990;6:137-44.

214. Khaw K. Barrett-Conner E. Family history of heart attack: a modifiable risk factor? Circulation 1986;74:239-44.

215. Tsevat J, Weinstein MC, Williams LW, Tosteson ANA, Goldman L. Expected gains in life expectancy from various coronary heart disease risk factor modifications. Circulation 1991;83:1194-1201.

216. Lifshitz F, Moses N. Growth failure: a complication of dietary treatment of hypercholesterolemia. Am J Dis Child 1989;143:537-42.

217. Avis NE, Smith KW, McKinlay JB. Accuracy of perceptions of heart attack risk: What influences perceptions and can they be changed? Am J Public Health 1989;17:1608-12.

218. Kulik JA, Mahler HIM. Health status perceptions of risk and prevention interest for health and non-health problems. Health Psych 1987;6:15-27.

219. Weinstein ND. Unrealistic optimism about susceptibility to health problems. J Behav Med 1982;5:441-60.

220. Weinstein N. Unrealistic optimism about susceptibility to health problems: Conclusions from a community-wide sample. J Behav Med 1987;10:481-499.

221. Dunkley GC, Langner NR, Hotz SB, Birkett NJ, Spasoff RA. Risk Factor Measurement in the Promotion of Cardiovascular Health. NHRDP Ref # 6606-4450-CH (L). Ottawa, 1991.

222. Health and Welfare Canada. Canadians and Smoking: An Update. Minister of Supply and Services Canada, Cat. No. H39-214/1991E. Ottawa, 1991.

223. Stachenko SJ, Reeder BA, Lindsay E, Donovan C, Lessard R, Balram C and the Canadian Heart Health Surveys Research Group. Smoking prevalence and associated risk factors in Canadian adults. CMAJ 1992;146(11):1989-1996.

Appendix A.

Table A1. Age distribution of patients at time of survey

Table A2. Risk factor changes by MI or cath lab identification

Table A3. Risk factor changes by patients' educational level

Table A4. Rates of behavioral change and male to female prevalence ratios, excluding women over age 50 at time of entry eligible event

Table A5. Variables not associated with dietary change in offspring

Table A6. Risk factor measurement and changes in high risk survey and in Ottawa Carleton Heart Beat Survey by age groups

Table A1. *Age distribution of patients at time of survey (patients were 0.5 to 2.7 years younger at time of entry eligible event (MI or catheterization)).*

Age group	Males No. (%)	Females No (%)
< 35	1 (0.5)	3 (2.6)
35-39	15 (7.4)	0 (0.0)
40-44	48 (23.5)	7 (6.1)
45-49	98 (48.0)	21 (18.4)
50-54	42 (20.6)	22 (19.3)
55-59	0 (0.0)	43 (37.7)
60-64	0 (0.0)	18 (15.8)
Total	204	114

Table A2. *Risk factor changes according to whether the patient was identified from the MI or the catheterization laboratory database.*

	MI No. (Total)	Cath Lab No. (Total)	χ^2	p-value
Changed diet	110 (121)	167 (196)	1.72	.190
Quit smoking (as proportion of all smokers)	72 (105)	102 (168)	.61	.433
Lost weight (among those with BMI > 25)	57 (68)	96 (124)	.75	.386

Table A3. *Risk factor changes by patients' educational level.*

	Education			
	No university (N)	University (N)	χ^2	p-value
Changed diet	217 (248)	57 (66)	0.0	.969
Quit smoking (as proportion of all smokers)	132 (218)	40 (52)	4.19	.041
Lost weight (among those with BMI > 25)	127 (154)	24(36)	3.55	.060

Table A4. *Rates of behavioral change and male to female prevalence ratios, excluding women over age 50 at time of entry eligible event.*

	Rate in men (%) n=204	Rate in women (%) n=42	Relative Risk (95% confidence interval)
Feel health fair or poor	34.6	57.1	0.61 (0.44-0.84)*
No change in diet	15.2	9.5	1.60 (0.59-4.28)
Should try to lower cholesterol level but haven't	10.9	2.4	4.31 (0.60-31.11)
Not doing anything to lower cholesterol	15.3	10.5	1.45 (0.36-5.91)
Trying to lose weight	73.7	83.3	0.81 (0.68-0.95)*
Trying to control hypertension (among hypertensives)	64.4	70.0	0.92 (0.65-1.30)
Informed blood pressure high	29.4	47.6	0.62 (0.42-0.90)*
Smoked at some time	91.7	81.0	1.13 (0.97-1.32)
Current smoker	32.8	26.2	1.25 (0.73-2.16)

*95% confidence limits do not overlap 1.0.

Table A5. *Variables not associated with offspring changing their diets.*

	Changed (N)	Did not change (N)	χ^2	p-value
Age: mean ±SEM	23.68 ±.463	24.47 ±.704		
Marital status	59	34	.724	.395
Physical exam within the previous year	86	43	.001	.979
Blood pressure check withing the previous year	105	49	.127	.722
BMI			.97	.617
<25	98	52		
25-27	21	8		
27+	28	11		
Smoking			1.26	.531
smoker	55	27		
exsmoker	22	7		
never smoked	70	38		
Education			.44	.507
- no university	127	59		
- university	20	13		

Table A6. *Risk factor measurement and changes: comparison of Ottawa-Carleton Heart Beat Survey (1987) and current survey (1990), respondents 19 to 24 years of age compared to 25 to 39 years. For the Heart Beat Survey total N = 690, for the Family Survey N = 180.*

	Heart Beat Survey		High Risk Survey	
	19-24 N = 115	25-39 N = 575	19-24 N = 92	25-39 N = 88
Feel health fair or poor	3.5	5.7	7.6	10.2
Blood pressure checked within 1 yr	82.6	77.9	75.0	73.9
Cholesterol checked within 1 yr	21.7	24.9	34.8	41.9
Informed of high blood pressure	14.8	15.1	10.2	16.5
Enough exercise	34.8	29.7	38.0	35.2
Exercise > 15 minutes at least 3x/week	59.1	57.6	69.6	67.1
Smoking				
Never smoked	54.8	46.4	53.3	35.2
Quit smoking	6.1	19.0	9.8	21.6
Current smoker	39.1	34.6	37.0	43.2

Appendix B.

Questionnaire I. Questionnaire for patients with premature IHD.

Questionnaire II. Questionnaire for offspring under age 16.

Questionnaire III. Questionnaire for offspring age 16 and over.

Section I: Questionnaire for parents

A. General Health

A.1. In general, compared to other persons your age, would you say your health is..[1]

1 ○ excellent
2 ○ very good
3 ○ good
4 ○ fair
5 ○ poor

A.2. Is there anything you plan to do over the next year to improve your health?[1] (DO NOT READ)

RECORD FIRST 4 ANSWERS

1 ○ nothing
2 ○ increase exercise
3 ○ lose weight
4 ○ improve eating habits
5 ○ quit smoking
6 ○ reduce amount smoked
7 ○ reduce drug/medication use
8 ○ drink less alcohol
9 ○ have blood pressure checked
10 ○ attempt to control blood pressure
11 ○ learn to manage stress / reduce stress level
12 ○ receive medical treatment
13 ○ change jobs
14 ○ other ______________________________

A.3. What is the single most important thing you have done in the past year to improve your health?[2,3]

1 ○ nothing
2 ○ increased exercise
3 ○ lost weight
4 ○ improved eating habits
5 ○ quit smoking
6 ○ reduced amount smoked
7 ○ reduced drug/medication use
8 ○ drank less alcohol
9 ○ had blood pressure checked
10 ○ attempted to control blood pressure
11 ○ learned to manage stress / reduced stress level
12 ○ received medical treatment
13 ○ changed jobs
14 ○ other ______________________________

A.4. Has a doctor ever told you that you have had a heart attack or a myocardial infarction?[2]

1 ○ yes
2 ○ no
8 ○ can't remember / don't know

B. Family History

B.1. Think about the other members of your household. Do any of them have high blood cholesterol?[4]

1 ○ yes
2 ○ no
3 ○ there is no one else in the household

B.2. Has anyone else in your family (brothers, sisters, parents, aunts, uncles) had a heart attack under the age of 55?

	Relation	Age at first heart attack
1	____________	____________
2	____________	____________
3	____________	____________
4	____________	____________
5	____________	____________
6	____________	____________

C. Dietary habits/changes

C.1. Have you changed the way you eat in the past 2 years?[4]

1 ○ yes **GO TO C.3**
2 ○ no/not sure

C.2. People have given us many reasons for not changing the things they eat and drink. I'm going to read some of those. For each, tell me if that is a major reason why you have no made changes, just part of the reason, or not at all a reason.[4]

CODE EACH ANSWER:
1 = MAJOR REASON
2 = PART OF REASON
3 = NOT A REASON
8 = NOT SURE

1 ○ Making changes in the kinds of food I eat would be expensive
2 ○ I enjoy the things I eat and drink and don't want to change
3 ○ The things I eat and drink now are healthy so there's no reason for me to make changes
4 ○ I've been eating this way for years. It would be too much trouble to start selecting different foods or preparing them in different ways
5 ○ There are so many recommendations about healthy ways to eat, I don't know what is good or bad
6 ○ I don't believe that what I eat really makes that much difference
7 ○ Someone else fixes my meals
8 ○ Making changes would be too hard, because I eat out so much

GO TO D.1

C.3. What changes have you made? First, are there any things you're eating less of?[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ fat/saturated fat (*ask for specifics*)
2 ○ meat/red meat/beef/pork
3 ○ fried foods
4 ○ eggs
5 ○ butter/whole milk/cheese
6 ○ salt/salty foods/sodium
7 ○ sweets/sugar
8 ○ processed foods/refined foods
9 ○ other ______________________
10 ○ nothing I'm eating less of
88 ○ not sure /don't know

C.4. Are there any things you're eating more of?[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ chicken/poultry
2 ○ fish
3 ○ fruits
4 ○ vegetables
5 ○ whole grains/fibre/oat bran
6 ○ pasta
7 ○ vegetable oil/olive oil
8 ○ other ______________________________
9 ○ nothing I'm eating more of
88 ○ not sure/don't know

C.5. Have you made any other changes in the way you eat to reduce your chances of getting heart disease?[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ use margarine/oil instead of butter
2 ○ eat lean cuts instead of fattier cuts of meat
3 ○ use skim or lowfat milk
4 ○ trim fat from meat
5 ○ other ______________________________
8 ○ not sure
9 ○ no other changes

D. Cholesterol

D.1. Have you ever been told by a doctor, nurse or other health professional that you have a high cholesterol level? [2,4,5,6] (PROMPT FOR HEALTH PROFESSIONAL)

1 ○ no
2 ○ yes, told by a doctor
3 ○ yes, told by a nurse
4 ○ yes, told by other health professional
8 ○ don't know

D.2. When did you last have your serum (blood) cholesterol checked?[2]

1 ○ within last 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never **GO TO E.1**
8 ○ not sure/don't know

D.3. Were you told what your blood cholesterol level was?[2]

1 ○ yes Specify ____________ [Were you told that that was high or normal?]
2 ○ was told only that it was high
3 ○ was told only that it was normal
4 ○ was told, but doesn't remember
5 ○ no/not sure

D.4. Which of these statements best describes your feelings about your blood cholesterol level?[4]

1 ○ I am not especially concerned about it
2 ○ ! probably should try to lower it but haven't really tried
3 ○ I have been trying to reduce my blood cholesterol level
4 ○ Not sure (DO NOT READ)

NORMAL CHOLESTEROL OR UNKNOWN: GO TO E.1

D.5. What were you told to do to lower your blood cholesterol? (PROBE: Anything else you were told to do to lower you blood cholesterol?)[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ changing or continuing diet (*ask for specifics*)
2 ○ reducing fat: ↓ meat, ↓ eggs, ↓ dairy fat
3 ○ increasing chicken, fish, fruits and vegetables
4 ○ increasing fiber/oat bran
5 ○ losing weight
6 ○ controlling stress and fatigue
7 ○ cutting down or stopping smoking
8 ○ taking medicine
9 ○ exercising
10 ○ rechecking it
11 ○ other ______________________________
88 ○ not sure/don't know

D.6. How much advice did you get from a doctor or other health professional about how you could lower your cholesterol level? Would you say you were given[4]

1 ○ all the advice you wanter'
2 ○ some advice but not really enough
3 ○ you were not given any advice at all
8 ○ not sure

D.7. From whom did you receive this information? Was it a doctor, a nurse, a dietitian, or who?[4]

1 ○ doctor
2 ○ nurse
3 ○ dietitian
4 ○ weight-loss counsellor
5 ○ other ______________________________

D.8. Are you currently doing anything to control your cholesterol level?[2]

1 ○ yes
2 ○ no **GO TO E.1**

D.9. What are you doing?[2] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ changing or continuing diet
2 ○ reducing fat: ↓ meat, ↓ eggs, ↓ dairy fat
3 ○ increasing chicken, fish, fruits and vegetables
4 ○ increasing fiber/oat bran
5 ○ losing weight
6 ○ controlling stress and fatigue
7 ○ cutting down or stopping smoking
8 ○ taking medicine
9 ○ exercising
10 ○ rechecking it
11 ○ other ______________________________
88 ○ not sure/don't know

E. Stress

E.1. On a scale of 1 to 7, how would you rate the amount of stress you've been under in the last 2 weeks? "No stress" would be 1 and a "great deal of stress" would be 7.[2]

1 2 3 4 5 6 7
+--------+--------+---------+---------+--------+--------+

F. Weight

F.1. How tall are you without shoes?[2,3]

____ inches
____ cm

F.2. How much do you weigh now?[2,3]

____ lbs
____ kgs

F.3. Are you now trying to lose weight, gain weight or neither?[5]

1 ○ lose weight
2 ○ gain weight } GO TO F.6
3 ○ neither }

F.4. How long have you been trying to lose weight?[5]

1 ○ < 1 week
2 ○ 1 - 2 weeks
3 ○ < 1 month
4 ○ < 6 months
5 ○ > 6 months

F.5. Have you lost weight?[5]

1 ○ yes GO TO G.1
2 ○ no
8 ○ not sure

F.6. In the past have you tried to lose weight?[5]

1 ○ yes
2 ○ no } GO TO
8 ○ not sure } G.1

F.7. Were you successful?[5]

1 ○ yes
2 ○ no
8 ○ not sure

G. Hypertension

G.1. When did you last have your blood pressure checked?[2,3]

1 ○ within last 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never **GO TO H.1**
8 ○ not sure/don't know

G.2. Have you ever been told by a doctor, nurse, or other health professional that you have high blood pressure? (Probe for doctor, nurse or other health professional)[2]

1 ○ no **GO TO H.1**
2 ○ yes, told by a doctor
3 ○ yes, told by a nurse
4 ○ yes, told by other health professional
8 ○ not sure/don't know

G.3. What were you told you should do for your high blood pressure?[2,6] (4 ANSWERS)

1 ○ take medicine
2 ○ go on a low salt diet
3 ○ watch weight/lose weight
4 ○ avoid stress, slow down and relax
5 ○ cut down or stop smoking
6 ○ cut down on alcohol intake
7 ○ start an exercise program
8 ○ use biofeedback
9 ○ none
10 ○ other ______________________________
88 ○ not sure/don't know

G.4. Are you currently doing anything to control your blood pressure?[2,3]
1 ○ yes
2 ○ no **GO TO H.1**

G.5. What are you doing?[2,3] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)
1 ○ take medicine
2 ○ go on a low salt diet
3 ○ watch weight/lose weight
4 ○ avoid stress, slow down and relax
5 ○ cut down or stop smoking
6 ○ cut down on alcohol intake
7 ○ start an exercise program
8 ○ use biofeedback
9 ○ none
10 ○ other ______________________________
88 ○ don't know

H. Smoking

H.1. Have you smoked at least 100 cigarettes in your life?[5]

1 ○ yes
2 ○ no **GO TO I.1**
8 ○ not sure

H.2. How old were you when you started smoking?[5]

____ years old
88 not sure

H.3. At the present time, how often do you smoke cigarettes?[5]

1 ○ everyday
2 ○ occasionally
3 ○ not at all **GO TO H.6**

H.4. On the average, how many cigarettes do you smoke a day?[2]

____ cigarettes
88 don't know

H.5. Have you tried to stop or decrease smoking in the last year?[7,2]

1 ○ yes
2 ○ no
8 ○ not sure

GO TO I.1

H.6. How long ago did you stop smoking cigarettes?[5]

1 ○ < 1 year ago ENTER NUMBER: ______ months
2 ○ > 1 year ago ENTER NUMBER: ______ years
88 ○ not sure

I. Demographic data

I.1. What is the highest grade or level of education you have completed?[2,3] (DO NOT READ)

1 ○ no schooling
2 ○ elementary
3 ○ some secondary
4 ○ completed secondary
5 ○ some community/technical college
6 ○ completed community/technical college
7 ○ some university
8 ○ completed university
9 ○ post-graduate
10 ○ other

I.2. What is your marital status?[2]

1 ○ married
2 ○ separated
3 ○ divorced
4 ○ widowed
5 ○ single

I.3. What is your birth date?[2,3]

______________________, 19____
Month Day Year

I.4. (Record language of interview)

E English
F French

This concludes the questions we have about you. We would now like to find out similar information about your children.

I.5. Could you give me the sex and birthday of each of your children, starting with the oldest?

	Month	Day	Year	SEX	(If over 16, at home?) Y / N
1	____	____	19 ___	___	___
2	____	____	19 ___	___	___
3	____	____	19 ___	___	___
4	____	____	19 ___	___	___
5	____	____	19 ___	___	___
6	____	____	19 ___	___	___

I.6. [*If most recent birthday is a child under 16*]
I'd like to ask some additional questions about your child with the birthday on ______________ [mention most recent birthday].

What is his/name? ______________________

These questions should be answered by the parent most familiar with the child, and his/her diet.

Who would that be? ______________________ [May I please speak to... and reintroduce if necessary]

GO TO Section II.

I.7. [*If most recent birthday is a child 16 or over*]
We would like permission to contact your children over 16 to ask them some similar questions. Do you think this would be possible?

1 ○ yes
2 ○ no

I.8. Could you in that case give me their names and phone numbers? (RECORD ON FAMILY COVER SHEET)

NAME ______________________ PHONE NO. (___)_____-_________

NAME ______________________ PHONE NO. (___)_____-_________

NAME ______________________ PHONE NO. (___)_____-_________

NAME ______________________ PHONE NO. (___)_____-_________

NAME ______________________ PHONE NO. (___)_____-_________

NAME ______________________ PHONE NO. (___)_____-_________

Section II: Questionnaire to be asked of parents for children under 16

A. General Health

A.1. In general, compared to other children that age, would you say _______'s [name of child] health is..[1,2]

1 ○ excellent
2 ○ very good
3 ○ good
4 ○ fair
5 ○ poor

A.2. Does _______ [name of child] have a chronic health problem which requires regular supervision by a doctor?

1 ○ yes SPECIFY _____________
2 ○ no

A.3. Has ________ [name of child] seen or talked to any of the following health professionals during the past 12 months?[1] (READ LIST)

		# times
1 ○	General practitioner	_______
2 ○	Specialist (paediatrician..)	_______
3 ○	Nurse	_______
4 ○	Nutritionist	_______

B. Dietary habits/changes

B.1. Has ________ [name of child] changed the way (s)he eats in the past 2 years to reduce the chances of getting heart disease?[4]

1 ○ yes
2 ○ no/not sure **GO TO C.1**

B.2. What changes has ________ [name of child] made? First, are there any things (s)he is eating less of?[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ fat (*ask for specifics*)
2 ○ meat/red meat/beef/pork
3 ○ fried foods
4 ○ eggs
5 ○ butter/whole milk/cheese
6 ○ salt/salty foods/sodium
7 ○ sweets/sugar
8 ○ processed foods/refined foods
9 ○ other ______________________________
10 ○ nothing eating less of
88 ○ not sure/don't know

B.3. Are there any things (s)he is eating more of?[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ chicken/poultry
2 ○ fish
3 ○ fruits
4 ○ vegetables
5 ○ whole grains/fibre/oat bran
6 ○ pasta
7 ○ vegetable oil/olive oil
8 ○ other ____________________
9 ○ nothing eating more of
88 ○ not sure/don't know

B.4. Has ________ [name of child] made any other changes in the way (s)he eats to reduce the chances of getting heart disease?[4] (PROBE) Anything else (DO NOT READ CHOICES)

1 ○ use margarine instead of butter
2 ○ eat lean cuts instead of fattier cuts of meat
3 ○ use skim or lowfat milk
4 ○ trim fat from meat
5 ○ other ____________________
8 ○ not sure
9 ○ no other changes

C. Cholesterol

C.1. Have you ever been told by a doctor, nurse or other health professional that ________ [name of child] has a high cholesterol level? [2,5,4,6] (PROBE FOR HEALTH PROFESSIONAL)

1 ○ no
2 ○ yes, told by a doctor
3 ○ yes, told by a nurse
4 ○ yes, told by other health professional
8 ○ don't know

C.2. About how long ago did ________ [name of child] last have a physical examination?[4]

1 ○ within the past 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never
8 ○ not sure/don't know

C.3. When did _________ [name of child] last have a serum (blood) cholesterol check?[2]

1 ○ within last 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never **GO TO C.5**
8 ○ not sure/don't know

C.4. Were you told what his/her blood cholesterol level was?

1 ○ yes Specify ________ [Were you told if it was high or normal?]
2 ○ was told only that it was high
3 ○ was told only that it was normal
4 ○ was told, but doesn't remember
5 ○ no/not sure

HIGH CHOLESTEROL: **GO TO C.9**
NORMAL CHOLESTEROL: **GO TO D.1**
DON'T KNOW: CONTINUE

C.5. If you found out that _________'s [name of child] cholesterol was *high*, would you try to make any changes to his/her present *lifestyle*? What would they be?

1 ○ lose weight
2 ○ reduce smoking
3 ○ lower high blood pressure
4 ○ dietary changes to lower high cholesterol levels
5 ○ drink less alcohol
6 ○ reduce stress
7 ○ get more exercise
8 ○ eat less salt
9 ○ eat fewer high fat foods
10 ○ eat fewer high cholesterol foods
11 ○ no changes
12 ○ other ______________________________
88 ○ not sure

C.6. If you found out that ________'s [name of child] cholesterol was *high*, would you try to make any changes to his/her present *diet*? What would they be? (RECORD 6 RESPONSES)

Eat less

1 ◯ fat (*ask for specifics*)
2 ◯ meat/red meat/beef/pork
3 ◯ fried foods
4 ◯ eggs
5 ◯ butter/whole milk/cheese
6 ◯ salt/salty foods/sodium
7 ◯ sweets/sugar
8 ◯ processed foods/refined foods

Eat more

9 ◯ chicken/poultry
10 ◯ fish
11 ◯ fruits
12 ◯ vegetables
13 ◯ whole grains/fibre/oat bran
14 ◯ pasta
15 ◯ vegetable oil/olive oil

Other changes

16 ◯ use margarine instead of butter
17 ◯ eat lean cuts instead of fattier cuts of meat
18 ◯ use skim or lowfat milk
19 ◯ trim fat from meat
20 ◯ no changes
21 ◯ other ________________________________
88 ◯ not sure

C.7. If you found out that ________'s [name of child] cholesterol level was *normal*, would you try to make any changes to his/her present *lifestyle*? What would they be?

1 ◯ changes (specify)____
____

2 ◯ no changes
8 ◯ not sure

C.8. If you found out that ________'s [name of child] cholesterol level was *normal*, would you try to make any changes to his/her present *diet*? What would they be?

1 ◯ changes (specify)____
____

2 ◯ no changes
8 ◯ not sure

GO TO D.1

C.9. What were you told to do to lower ________'s [name of child] blood cholesterol? (PROBE: Anything else you were toid to do to lower ________'s [name of child] blood cholesterol?)[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ changing or continuing diet (*ask for specifics*)
2 ○ reducing fat: ↓ meat, ↓ eggs, ↓ dairy fat
3 ○ increasing chicken, fish, fruits and vegetables
4 ○ increasing fiber/oat bran
5 ○ losing weight
6 ○ controlling stress and fatigue
7 ○ cutting down or stopping smoking
8 ○ taking medicine
9 ○ exercising
10 ○ rechecking it
11 ○ other ________________________________
88 ○ not sure/don't know

C.10. How much advice did you get from a doctor or other health professional about how you could lower ________'s [name of child] cholesterol level? Would you say you were given[4]

1 ○ all the advice you wanted
2 ○ some advice but not really enough
3 ○ you were not given any advice at all } **GO TO**
4 ○ (s)he did not see a health professional (DO NOT READ) } **C.12**
8 ○ not sure (DO NOT READ) }

C.11. From whom did you receive this information? Was it a doctor, a nurse, a dietitian, or who?[4]

1 ○ doctor
2 ○ nurse
3 ○ dietitian
4 ○ weight-loss counsellor
5 ○ other ________________________________

C.12. Is ________ [name of child] currently doing anything to control his/her cholesterol level?[2]

1 ○ yes
2 ○ no **GO TO D.1**

C.13. What is (s)he doing?[2] (DO NOT READ CHOICES)

1 ◯ changing or continuing diet
2 ◯ reducing fat: ↓ meat, ↓ eggs, ↓ dairy fat
3 ◯ increasing chicken, fish, fruits and vegetables
4 ◯ increasing fiber/oat bran
5 ◯ losing weight
6 ◯ controlling stress and fatigue
7 ◯ cutting down or stopping smoking
8 ◯ taking medicine
9 ◯ exercising
10 ◯ rechecking it
11 ◯ other ______________________________
88 ◯ not sure/don't know

D. Stress (For children over 6)

D.1. On a scale of 1 to 7, how would you rate the amount of stress ________ [name of child] has been under in the last 2 weeks? "No stress" would be 1 and a "great deal of stress" would be 7.[2]

1 2 3 4 5 6 7
+--------+--------+---------+---------+--------+--------+

D.2. On a scale of 1 to 7, how would you rate your concern about ________ [name of child] having a heart attack in the future? "Never think about it" would be 1 and "lose a lot of sleep over it" would be 7.

1 2 3 4 5 6 7
+--------+--------+---------+---------+--------+--------+

D.3. On a scale of 1 to 7, how would you rate your child's own concern about ________ [name of child] having a heart attack in the future? "Never think about it" would be 1 and "lose a lot of sleep over it" would be 7.

1 2 3 4 5 6 7
+--------+--------+---------+---------+--------+--------+

E. Weight

E.1. Do you consider ________ [name of child] to be overweight, underweight, or about the proper weight?

1 ◯ overweight
2 ◯ underweight
3 ◯ proper
8 ◯ don't know

F. Hypertension

F.1. When did ________ [name of child] last have his/her blood pressure checked?[2,3]

1 ○ within last 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never **GO TO G.1**
8 ○ don't know

F.2. Have you ever been told by a doctor, nurse, or other health professional that ________ [name of child] has high blood pressure? (Probe for doctor, nurse or other health professional)[2]

1 ○ no **GO TO G.1**
2 ○ yes, told by a doctor
3 ○ yes, told by a nurse
4 ○ yes, told by other health professional
8 ○ not sure/don't know

F.3. What were you told to do for ________'s [name of child] high blood pressure?[2,6] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ take medicine
2 ○ go on a low salt diet
3 ○ watch weight/lose weight
4 ○ avoid stress, slow down and relax
5 ○ cut down or stop smoking
6 ○ cut down on alcohol intake
7 ○ start an exercise program
8 ○ use biofeedback
9 ○ nothing
10 ○ other ________________________________
88 ○ not sure/don't know

F.4. Is ________ [name of child] currently doing anything to control his/her blood pressure?[2,3]

1 ○ yes
2 ○ no **GO TO G.1**

F.5. What is (s)he doing?[2,3] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ take medicine
2 ○ go on a low salt diet
3 ○ watch weight/lose weight
4 ○ avoid stress, slow down and relax
5 ○ cut down or stop smoking
6 ○ cut down on alcohol intake
7 ○ start an exercise program
8 ○ use biofeedback
9 ○ none
10 ○ other ______________________________
88 ○ not sure/don't know

G. Physical exercise (For children over 6)

G.1. Exercise includes vigorous activities such as calisthenics, jogging, racquet sports, team sports, dance classes, or brisk walking. Do you feel that ________ [name of child] gets as much exercise as (s)he needs or less than (s)he needs?[2]

1 ○ as much as needed
2 ○ less than needed
8 ○ not sure/don't know

G.2. How many times per week does ________ [name of child] exercise for at least 15 minutes?[2]

1 ○ daily
2 ○ 5-6 times a week
3 ○ 3-4 times a week
4 ○ 1-2 times a week
5 ○ less than once a week
6 ○ never
8 ○ not sure/don't know

G.3. Compared to 2 years ago, does (s)he now exercise more, less or about the same amount of time per week?

1 ○ more
2 ○ less
3 ○ about the same
8 ○ not sure/don't know

H. Smoking (For children over 9)

H.1. Does ________ [name of child] smoke cigarettes on a regular basis?

1 ○ yes
2 ○ no
8 ○ not sure/don't know

Section III: Questionnaire for children 16+

A. General Health

A.1. In general, compared to other persons your age, would you say your health is..[1,2,3]

1 ◯ excellent
2 ◯ very good
3 ◯ good
4 ◯ fair
5 ◯ poor

A.2. As a whole, would you describe your life as..[1]

1 ◯ very stressful
2 ◯ fairly stressful
3 ◯ not very stressful
4 ◯ not at all stressful

A.3. How satisfied are you with your health?[1]

1 ◯ very satisfied
2 ◯ somewhat satisfied
3 ◯ not too satisfied
4 ◯ not at all satisfied

A.4. Is there anything you plan to do over the next year to improve your health?[1] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ◯ nothing
2 ◯ increase exercise
3 ◯ lose weight
4 ◯ improve eating habits
5 ◯ quit smoking
6 ◯ reduce amount smoked
7 ◯ reduce drug/medication use
8 ◯ drink less alcohol
9 ◯ have blood pressure checked
10 ◯ attempt to control blood pressure
11 ◯ learn to manage stress
12 ◯ reduce stress level
13 ◯ receive medical treatment
14 ◯ change jobs
15 ◯ other ______________________________

A.5. What is the single most important thing you have done in the past year to improve your health?[2,3] (DO NOT READ CHOICES: ONE ANSWER ONLY)

1 ○ nothing
2 ○ increased exercise
3 ○ lost weight
4 ○ improved eating habits
5 ○ quit smoking/reduced amount smoked
6 ○ reduced drug/medication use
7 ○ drank less alcohol
8 ○ had blood pressure checked
9 ○ attempted to control blood pressure
10 ○ learned to manage stress
11 ○ reduced stress level
12 ○ received medical treatment
13 ○ other ______________________
88 ○ not sure

A.6. Have you seen or talked to any of the following health professionals during the past 12 months?[1] (READ EACH CHOICE, PROMPT FOR NUMBER OF TIMES)

		# times
1 ○	General practitioner	______
2 ○	Specialist / internist	______
3 ○	Nurse	______
4 ○	Nutritionist	______

B. Causes of Heart Disease

B.1. As you understand it, what are the major causes of heart disease or heart attacks? Are there any others you can think of?[4] (DO NOT READ CHOICES: RECORD FIRST 4 ANSWERS)

1 ◯ diet
2 ◯ overeating/eating too much
3 ◯ fats/excessive fats/fatty foods/greasy foods
4 ◯ saturated fats/animal fats
5 ◯ salt/sodium/salty foods
6 ◯ cholesterol
7 ◯ stress/worry/tension/anxiety/emotional strain
8 ◯ fatigue/overwork/not enough relaxation
9 ◯ lack of exercise
10 ◯ smoking
11 ◯ alcohol
12 ◯ high blood pressure
13 ◯ heredity/family history/genes
14 ◯ other ______________________________
88 ◯ don't know/not sure

B.2. What things that people eat or drink might be related to heart disease or heart attacks?[4] (DO NOT READ CHOICES: RECORD FIRST 4 ANSWERS)

1 ◯ fats
2 ◯ saturated fats
3 ◯ fried foods/greasy foods/oily foods
4 ◯ cholesterol
5 ◯ calories/eating too much/overweight
6 ◯ eggs
7 ◯ dairy products/milk/cheese
8 ◯ sugar/sweet foods
9 ◯ caffeine/coffee
10 ◯ salt/salty foods/sodium
11 ◯ alcohol
12 ◯ other ______________________________
88 ◯ not sure/don't know

B.3. In your opinion, what can a person do to prevent heart disease?[2] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ lose weight
2 ○ reduce smoking
3 ○ lower high blood pressure
4 ○ lower high cholesterol levels
5 ○ drink less alcohol
6 ○ reduce stress
7 ○ get more exercise
8 ○ eat less salt
9 ○ eat fewer high fat foods
10 ○ eat fewer high cholesterol foods
11 ○ other ______________________________
88 ○ not sure/don't know

C. Dietary habits/changes

C.1. Have you changed the way you eat over the past year to reduce your chances of getting heart disease?[4]

1 ○ yes **GO TO C.3**
2 ○ no/not sure

C.2. People have given us many reasons for not changing the things they eat and drink. I'm going to read some of those. For each, tell me if that is a major reason why you have no made changes, just part of the reason, or not at all a reason.[4]

CODE EACH ANSWER:
1 = MAJOR REASON
2 = PART OF REASON
3 = NOT A REASON
8 = NOT SURE

1 ○ Making changes in the kinds of food I eat would be expensive
2 ○ I enjoy the things I eat and drink and don't want to change
3 ○ The things I eat and drink now are healthy so there's no reason for me to make changes
4 ○ I've been eating this way for years. It would too much trouble to start selecting different foods or preparing them in different ways
5 ○ There are so many recommendations about healthy ways to eat, I don't know what is good or bad
6 ○ I don't believe that what I eat really makes that much difference in my chances of getting heart disease
7 ○ Someone else fixes my meals
8 ○ Making changes would be too hard, because I eat out so much

GO TO D.1

C.3. What changes have you made? First, are there any things you're eating less of?[4] (DO NOT READ CHOICES: RECORD FIRST 4 ANSWERS)

1 ◯ fat (*ask for specifics*)
2 ◯ meat/red meat/beef/pork
3 ◯ fried foods
4 ◯ eggs
5 ◯ butter/whole milk/cheese
6 ◯ salt/salty foods/sodium
7 ◯ sweets/sugar
8 ◯ processed foods/refined foods
9 ◯ not sure
10 ◯ nothing I'm eating less of
10 ◯ other ______________________

C.4. Are there any things you're eating more of?[4] (DO NOT READ CHOICES)

1 ◯ chicken/poultry
2 ◯ fish
3 ◯ fruits
4 ◯ vegetables
5 ◯ whole grains/fibre/oat bran
6 ◯ pasta
7 ◯ vegetable oil/olive oil
8 ◯ other ______________________
9 ◯ not sure
10 ◯ nothing I'm eating more of

C.5. Have you made any other changes in the way you eat to reduce your chances of getting heart disease?[4] (DO NOT READ CHOICES: RECORD FIRST 4 ANSWERS)

1 ◯ use margarine/oil instead of butter
2 ◯ eat lean cuts instead of fattier cuts of meat
3 ◯ use skim or lowfat milk
4 ◯ trim fat from meat
5 ◯ other ______________________
8 ◯ not sure
9 ◯ no other changes

D. Cholesterol

D.1. As you understand it, what are the major causes of high blood cholesterol? (DO NOT READ CHOICES but PROMPT:) Are there any other causes of high blood cholesterol?[4]

1 ◯ diet/poor diet (ask for specifics)
2 ◯ eating foods high in cholesterol
3 ◯ eating foods high in fat
4 ◯ eating foods high in saturated fats
5 ◯ overweight/overeating
6 ◯ eggs
7 ◯ meat
8 ◯ dairy products
9 ◯ heredity/family history/genes
10 ◯ lack of exercise
11 ◯ other ______________________________
88 ◯ not sure

D.2. If you wanted to lower your blood cholesterol, would you know how to do it, or not?[4]

1 ◯ would know
2 ◯ have some idea, but not really sure
3 ◯ would not know
8 ◯ not sure

D.3. Have you ever been told by a doctor, nurse or other health professional that you have a high cholesterol level? [2,5,4,6] (PROMPT FOR HEALTH PROFESSIONAL)

1 ◯ no
2 ◯ yes, told by a doctor
3 ◯ yes, told by a nurse
4 ◯ yes, told by other health professional
8 ◯ don't know

D.4. About how long ago did you last have a physical examination?[4] (DO NOT READ CHOICES)

1 ◯ within the past 6 months
2 ◯ 6-12 months ago
3 ◯ 1-2 years ago
4 ◯ 3-5 years ago
5 ◯ more than 5 years ago
6 ◯ never
8 ◯ not sure/don't know

D.5. When did you last have your serum (blood) cholesterol checked?[2]

1 ○ within last 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never } **GO TO D.8**
8 ○ not sure/don't know

D.6. Were you told what you blood cholesterol level was?

1 ○ yes Specify ________ [Were you told that it was high or normal?]
2 ○ was told only that it was high
3 ○ was told only that it was normal **GO TO D.18**
4 ○ was told, but doesn't remember
5 ○ no/not sure **GO TO D.8**

D.7. Which of these statements best describes your feelings about your blood cholesterol level?[4]

1 ○ I am not especially concerned about it
2 ○ I probably should try to lower it but haven't really tried
3 ○ I have been trying to reduce my blood cholesterol level
4 ○ Not sure (DO NOT READ)

GO TO D.13

D.8. [*If doesn't know*] Would you be interested in finding out your blood cholesterol level, or would you prefer not to know?

1 ○ would be interested
2 ○ would prefer not to know
8 ○ not sure/don't know

D.9. If you f... out that your cholesterol was *high,* would you make any changes to your present *lifestyle*? What would they be? (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ lose weight
2 ○ reduce smoking
3 ○ lower high blood pressure
4 ○ lower high cholesterol levels
5 ○ drink less alcohol
6 ○ reduce stress
7 ○ get more exercise
8 ○ eat less salt
9 ○ eat fewer high fat foods
10 ○ eat fewer high cholesterol foods
11 ○ no changes
12 ○ other ________________________________

D.10. If you found out that your cholesterol was *high,* would you make any changes to your present *diet*? What would they be? (DO NOT READ CHOICES, RECORD FIRST 6 ANSWERS)

eat less

1 ○ fat (*ask for specifics)*
2 ○ meat/red meat/beef/pork
3 ○ fried foods
4 ○ eggs
5 ○ butter/whole milk/cheese
6 ○ salt/salty foods/sodium
7 ○ sweets/sugar
8 ○ processed foods/refined foods

eat more

9 ○ chicken/poultry
10 ○ fish
11 ○ fruits
12 ○ vegetables
13 ○ whole grains/fibre/oat bran
14 ○ pasta
15 ○ vegetable oil/olive oil

16 ○ use margarine instead of butter
17 ○ eat lean cuts instead of fattier cuts of meat
18 ○ use skim or lowfat milk
19 ○ trim fat from meat
20 ○ no changes
21 ○ other ________________________
88 ○ not sure

D.11. If you found out that your cholesterol level was *normal*, would you make any changes to your present *lifestyle*? What would they be?

1 ○ changes: specify ______________________________
____

2 ○ no changes
8 ○ not sure

D.12. If you found out that your cholesterol level was *normal*, would you make any changes to your present *diet*? What would they be?

1 ○ changes: specify ______________________________
____

2 ○ no changes
8 ○ not sure

D.13. What were you told to do to lower your blood cholesterol? (PROBE: Anything else you were told to do to lower you blood cholesterol?)[4] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ changing or continuing diet (*ask for specifics*)
2 ○ reducing fat: ↓ meat, ↓ eggs, ↓ dairy fat
3 ○ increasing chicken, fish, fruits and vegetables
4 ○ increasing fiber/oat bran
5 ○ losing weight
6 ○ controlling stress and fatigue
7 ○ cutting down or stopping smoking
8 ○ taking medicine
9 ○ exercising
10 ○ rechecking it
11 ○ other ______________________________
88 ○ not sure/don't know

D.14. How much advice did you get from a doctor or other health professional about how you could lower your cholesterol level? Would you say you were given[4]

1 ○ all the advice you wanted
2 ○ some advice but not really enough
3 ○ you were not given any advice at all }
} **GO TO D.16**
4 ○ did not see health professional (DO NOT READ) }
8 ○ not sure }

D.15. From whom did you receive this information? Was it a doctor, a nurse, a dietitian, or who?[4]

1 ○ doctor
2 ○ nurse
3 ○ dietitian
4 ○ weight-loss counsellor
5 ○ other ____________________

D.16. Are you currently doing anything to control your cholesterol level?[2]

1 ○ yes
2 ○ no **GO TO D.18**

D.17. What are you doing?[2] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ changing or continuing diet
2 ○ reducing fat: ↓ meat, ↓ eggs, ↓ dairy fat
3 ○ increasing chicken, fish, fruits and vegetables
4 ○ increasing fiber/oat bran
5 ○ losing weight
6 ○ controlling stress and fatigue
7 ○ cutting down or stopping smoking
8 ○ taking medicine
9 ○ exercising
10 ○ rechecking it
11 ○ other ____________________
88 ○ not sure/don't know

D.18. Think about the other members of your household. Do any of them have high blood cholesterol?[4]

1 ○ yes
2 ○ no
3 ○ there is no one else in the household

E. Stress

E.1. On a scale of 1 to 7, how would you rate the amount of stress you've been under in the last 2 weeks? "No stress" would be 1 and a "great deal of stress" would be 7.[2]

```
1        2        3         4         5        6        7
+--------+--------+---------+---------+--------+--------+
```

E.2. On a scale of 1 to 7, how would you rate your concern about having a heart attack in the future? "Never think about it" would be 1 and "lose a lot of sleep over it" would be 7.

```
1        2        3         4         5        6        7
+--------+--------+---------+---------+--------+--------+
```

F. Weight

F.1. How tall are you without shoes?[2,3]

_____ inches
_____ cm

F.2. How much do you weigh now?[2,3]

_____ lbs
_____ kgs

F.3. Are you now trying to lose weight, gain weight or neither?[5]

1 ○ lose weight
2 ○ gain weight } **GO TO**
3 ○ neither } **F.8**

F.4. Why do you think you should lose weight? Are there any other reasons why you think you should lose weight?[4] (DO NOT READ CHOICES)

1 ○ appearance/attractiveness
2 ○ general health
3 ○ heart disease
4 ○ high blood pressure
5 ○ high cholesterol
6 ○ cancer
7 ○ diabetes
8 ○ not sure
9 ○ other ____

F.5. What are you doing in order to lose weight?[5] (DO NOT READ CHOICES)

1 ○ dieting
2 ○ exercising
3 ○ skipping meals
4 ○ taking diet pills
5 ○ going to weight control programs
6 ○ other

F.6. How long have you been trying to lose weight?[5]

1 ○ < 1 week
2 ○ 1 - 2 weeks
3 ○ < 1 month
4 ○ < 6 months
5 ○ > 6 months

F.7. Have you lost weight?[5]

1 ○ yes **GO TO G.1**
2 ○ no
8 ○ not sure

F.8. In the past have you tried to lose weight?[5]

1 ○ yes
2 ○ no } **GO TO G.1**
8 ○ not sure }

F.9. Were you successful?[5]

1 ○ yes
2 ○ no
8 ○ not sure

G. Hypertension

G.1. When did you last have your blood pressure checked?[2,3]

1 ○ within last 6 months
2 ○ 6-12 months ago
3 ○ 1-2 years ago
4 ○ 3-5 years ago
5 ○ more than 5 years ago
6 ○ never **GO TO H.1**
8 ○ don't know

G.2. Have you ever been told by a doctor, nurse, or other health professional that you have high blood pressure? (Probe for doctor, nurse or other health professional)[2]

1 ○ no **GO TO H.1**
2 ○ yes, told by a doctor
3 ○ yes, told by a nurse
4 ○ yes, told by other health professional
8 ○ don't know

G.3. What were you told you should do for your high blood pressure?[2,6] (FIRST 4 ANSWERS)

1 ○ take medicine
2 ○ go on a low salt diet
3 ○ watch weight/lose weight
4 ○ avoid stress, slow down and relax
5 ○ cut down or stop smoking
6 ○ cut down on alcohol intake
7 ○ start an exercise program
8 ○ use biofeedback
9 ○ nothing
10 ○ other ____________________
88 ○ don't know

G.4. Are you currently doing anything to control your blood pressure?[2,3]

1 ○ yes
2 ○ no **GO TO H.1**

G.5. What are you doing?[2,3] (DO NOT READ CHOICES, RECORD FIRST 4 ANSWERS)

1 ○ take medicine
2 ○ go on a low salt diet
3 ○ watch weight/lose weight
4 ○ avoid stress, slow down and relax
5 ○ cut down or stop smoking
6 ○ cut down on alcohol intake
7 ○ start an exercise program
8 ○ use biofeedback
9 ○ none
10 ○ other ______________________
88 ○ don't know

H. Physical exercise

H.1. Exercise includes vigorous activities such as calisthenics, jogging, racquet sports, team sports, dance classes, or brisk walking. Do you feel that you get as much exercise as you need or less than you need?[2]

1 ○ as much as needed
2 ○ less than needed
8 ○ don't know

H.2. How many times per week do you exercise for at least 15 minutes?[2]

1 ○ daily
2 ○ 5-6 times a week
3 ○ 3-4 times a week
4 ○ 1-2 times a week
5 ○ less than once a week
6 ○ never
8 ○ don't know

H.3. Compared to 2 years ago, do you now exercise more, less or about the same amount of time per week?

1 ○ more
2 ○ less
3 ○ about the same
8 ○ not sure

I. Smoking

I.1. Have you smoked at least 100 cigarettes in your life?[5]

1 ○ yes
2 ○ no **GO TO J.1**
8 ○ not sure

I.2. How old were you when you started smoking?[5]

____ years old
88 not sure

I.3. At the present time, how often do you smoke cigarettes?[5]

1 ○ everyday
2 ○ occasionally
3 ○ not at all **GO TO I.6**

I.4. On the average, how many cigarettes do you smoke a day?[2]

____ cigarettes
88 don't know

I.5. Have you tried to stop or decrease smoking in the last year?[2,7]

1 ○ yes }
2 ○ no } **GO TO J.1**
8 ○ not sure }

I.6. How long ago did you stop smoking cigarettes?[5]

1 ○ < 1 year ago ENTER NUMBER: ______ months
2 ○ > 1 year ago ENTER NUMBER: ______ years
88 ○ not sure

J. Demographic data

J.1. What is the highest grade or level of education you have completed?[2,3] (DO NOT READ)

1 ○ no schooling
2 ○ elementary
3 ○ some secondary
4 ○ completed secondary
5 ○ some community/technical college
6 ○ completed community/technical college
7 ○ some university
8 ○ completed university
9 ○ post-graduate
10 ○ other

J.2. What is your marital status?[2]

1 ○ married
2 ○ separated
3 ○ divorced
4 ○ widowed
5 ○ single

J.3. What is your birth date?[2,3]

______________, 19___
Month Day Year

J.4. (Record language of interview)

E English
F French

References

1. Ontario Health Survey
2. Ottawa Carleton Heart Beat Survey
3. Health Promotion Survey
4. US Health and Diet Survey, 1986
5. Ottawa Carleton Worksite Survey, 1989
6. Nova Scotia Heart Health Survey
7. Ottawa Carleton Community Health Survey, 1985