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Coronary Heart Disease Among The Dariusleut Hutteritesof Alberta
by
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FACULTY OF GRADUATE STUDIES

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled, " Coronary Heart Disease Among the Dariusleut Hutterites of Alberta" by John Howard Brunt in partial fulfillment of the requirements for the degree of Doctor of Philosophy.


December 13, 1989


#### Abstract

Coronary Heart Disease Among the Dariusleut Hutterites of Alberta


The two objectives of this epidemiologic study were: 1) to determine the prevalence of coronary heart disease (CHD) and its risk factors, and 2) to examine the association between CHD and its risk factors, among the Dariusleut Hutterites of Alberta, Canada. The CHD risk factors examined included hypertension, hypercholesterolemia, diabetes, cigarette smoking, elevated Body Mass Index (BMI)., alcohol consumption, psychological stress, physical activity, and family history of CHD.

The methods of investigation used for this cross$s$ sectional survey included interviews, self-administered questionnaires, laboratory measures (random blood cholesterol and post-prandial blood glucose), and physical measures (blood pressure, height, and weight). The data gathered using the questionnaire and interview included demographic information, lifestyle habits, medication and health care utilization, evidence of Rose Angina, past medical history, and family history of CHD.

All Hutterites between 30 and 74 years of age from the 68 Dariusleut colonies in Alberta were invited to participate in the survey; 38 colonies (55.9\%) enrolled in the study. Of the 914 Hutterites from the 38 colonies eligible for inclusion in the survey, $88.7 \%$ ( $n=811$ )
participated, and surrogate information was gathered on the remaining 11.3\% ( $n=103$ ).

Within the Hutterite population, as compared to other North American and/or European prevalence studies: 1) the prevalence of hypertension, hypercholesterolemia, diabetes, and elevated BMI were higher, 2) the prevalence of smoking, physical exercise, and extreme psychological stress were lower, and 3) the prevalence of alcohol consumption was similar. A family history of CHD was reported by over twothirds of the respondents. In the adjusted analysis, the factors significantly associated with CHD, for the males, included hypertension, diabetes and increasing age. Only diabetes was significantly associated with CHD for the females.

Despite the high prevalence of hypertension, hypercholesterolemia, diabetes, and elevated BMI, the prevalence of CHD was not found to be higher than in other prevalence surveys. Possible explanations for this finding include: 1) the very low prevalence of smoking among Hutterites may reduce their risk of CHD by as much as $30 \%$, 2) the moderate alcohol consumption found among the Hutterites may help improve their lipid profiles, 3) the strong social support sŷstems of Hutterites may, through as yet unknown mechanisms, reduce CHD risk, and 4) Hutterites may have higher case-fatality rates for CHD than found in other populations.

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CHAPTER 1: PURPOSE OF THE STUDY AND REVIEW OF CURRENT IITERATURE

## I. Introduction and Purpose of the Study

Despite a three decade long decline in Coronary Heart Disease (CHD) mortality, CHD continues to be the leading cause of death and disability in North America. The reason for the reduction in deaths attributed to CHD is thought to be due, in part, to better medical treatment of prevalent cases (e.g. lowering of case fatality rates) and, in part, through improved lifestyles and preventive measures (e.g. lowering of incidence rates).

There is a substantial body of evidence supporting the role of a number of risk factors in the etiology of CHD. Largely due to population-based epidemiologic research, there is wide acceptance of the association between the development of CHD and the following risk factors: age, male sex, serum lipids, serum glucose, hypertension and cigarette smoking. There are a number of other possible CHD risk factors that are more controversial. These include obesity, physical inactivity, mental stress and family history. However, even when all possible CHD risk factors are taken together, they can only account for approximately half of the incident cases.

There is currently a great deal of interest in exploring the genetic basis of virtually every chronic disease. The rapidly developing technologies associated
with genetic research may provide researchers with powerful new tools for helping explain the missing half of CHD incidence. Discovering populations that may increase the probability of successfully locating the genetic markers of CHD (e.g. genetically isolated populations) could aid in this endeavor.

Such a population, the Hutterian Brethren, or Hutterites, reside in relatively large numbers in the province of Alberta. As a population, the Hutterites have one of the highest coefficients of inbreeding known, making them an ideal group for inclusion in genetic studies of CHD.

The Hutterite way of life is relatively homogeneous both inter- and intra-generationally in terms of lifestylerelated CHD risk factors. This homogeneity of lifestyle decreases the problems associated with attempts to tease out the genetic and lifestyle factors related to multi-factoral diseases such as CHD.

Further enhancing the value of utilizing knowledge about Hutterites in genetic studies is their great interest in health and liberal use of modern health care. Hutterites have in the past cooperated well with medical researchers, particularly for genetic research.

While genetic studies of CHD may prove to be useful in the Hutterite population, to date there is little known about the prevalence of CHD or its major risk factors in this population. Prior to undertaking more costly genetic
studies, it is first necessary to assess the importance of CHD within the Hutterian Brethren.

Thus, the purpose of this study was to help increase our knowledge of the way in which CHD and its risk factors manifest within the Hutterian Brethren. The study's objectives, research questions and hypotheses are described below.

## II. Objectives and Research Questions

This study had two primary objectives:

1) to determine the prevalence and distribution of Coronary Heart Disease and Coronary Heart Disease risk factors in a Hutterite population,
2) to examine the association between Coronary Heart Disease and selected Coronary Heart Disease risk factors in a Hutterite population.

The specific research questions and hypotheses that guided the study are outlined below in relation to each of the main objectives.
A. Research Ouestions for Objective 1

The first objective, "to determine the prevalence and distribution of CHD and CHD risk factors in a Hutterite population", raised a number of research questions. These questions are: in the study population, what was the prevalence and distribution of:

1. CHD ?
2. hypertension ?
3. hypercholesterolemia ?
4. diabetes mellitus ?
5. cigarette smoking ?
6. elevated Body Mass Index (BMI) ?
7. alcohol consumption ?
8. stress ?
9. physical activity ?
10. a positive CHD family history ?
B. Hypotheses for Objective 1

Given the exploratory nature of this study, no formal hypothesis testing was performed for the first objective. However, wherever possible the prevalence of the above factors was compared with other published reports.
C. Research Questions for Objective 2

The second objective, "to examine the association between CHD and selected CHD risk factors in a Hutterite population" posed a number of specific research questions. These are: in the study population, are the following, both independently and in combination with other subsets of risk factors, associated with CHD:

1. hypertension
2. hypercholesterolemia
3. diabetes
4. cigarette smoking
5. elevated Body Mass Index
6. alcohol consumption
7. stress
8. physical activity
9. a positive family history for CHD

## D. Hypotheses for Objective 2

Based on evidence from a number of epidemiologic studies, it was hypothesized that there would be a significant univariate relationship in the study population between CHD and hypertension, diabetes, hypercholesterolemia and cigarette smoking. While there is weaker epidemiologic evidence supporting an association between CHD and alcohol intake, physical activity, elevated BMI, positive family
history and stress, it was hypothesized that these factors will also be related to CHD in the univariate analysis. While there was no a priori hypothesis regarding the multivariate relationships with the above risk factors and CHD, logistic regression was used to provide the set of variables associated with CHD in the adjusted analysis.
III. Review of Current Literature

## A. The Hutterian Brethren

The Hutterite Brethren are a distinctive Anabaptist sect with approximately 30,000 members who live an agrariancommunal lifestyle on the great plains of North America. Approximately $70 \%$ of the population live in the Western Canadian provinces and $30 \%$ in the adjoining American states on communal farms, called colonies (Evans, 1985; Morgan and Holmes, 1982). Alberta has the highest concentration of Hutterites in North America. Two-thirds of the Albertan colonies are Dariusleut and one-third are Leherleut (Hostetler, 1985).

The Hutterite Brethren have essentially remained a genetic isolate with minimal in- or out-migration since the 1870's. The three divisions, or Leut, of the Hutterite Brethren share a common lifestyle and belief system, but inter-leut marriage has been rare in the past and is virtually non-existent today (Hostetler, 1985). Hutterites have one of the highest known fertility rates, with married women having an average of approximately 10 livebirths each (Holmes and Morgan, 1982). While Hutterites are more inbred
than the general population and are more at risk for some relatively rare genetic disorders, proof of their adequate genetic diversity is supported by the fact that their overall incidence of genetic and congenital disorders is no greater than in the general population (Lowry, Morgan, Holmes, and Gilrony, 1985).

In keeping with their relatively homogeneous lifestyles and population growth patterns, there is good evidence that the health status of the three Leut is similar. Cancer rates by site, age, and sex are similar for all three leuts (Martin et al., 1980; Morgan, Holmes, Grace, Kemel, and Robson, 1983) . Similar age-adjusted mortality rates between the Leherleut and Dariusleut provides further evidence of the homogeneity of inter-leut health status (Morgan, 1983).

Hutterite society is highly structured and socialization of its members creates a remarkably similar inter- and intra-generational lifestyle (Hostetler, 1974). Lifestyle habits and personal behavior patterns are strongly influenced by group norms. Smoking is officially forbidden, but there are anecdotal reports of limited smoking in some colonies (personal communication, Mary Fujiwara, 1987). Mild to moderate alcohol consumption is permitted and many colonies are known for their homemade wines. All major meals are taken communally with men, women, and children each eating in separate groups. The usual diet is high in animal fat and regularly includes pork, goose, beef, cheese, fried
potatoes, butter, baked goods, and eggs (Hostetler, 1974). All heavy farm labour jobs are carried out by the men, and women are expected to cook, clean, help with farm chores, and tend children. Hutterite farms are highly efficient and noted for their use of the latest equipment, herbicides, and pesticides (Steinberg, Bleibtreu, Martin, and Kurczinski, 1967).

Hutterites, despite their reluctance to integrate into the society at large, seek modern health care (Hostetler, 1974; Hostetler, 1985; Peter, 1980). While their rural existence does create some obstacles to utilization of tertiary care facilities, if highly technical and specialized care is required they will seek it out (Hostetler, 1985). Community health care services, such as home care for the terminally ill, immunization, and aids to daily living, are also widely utilized (personal communication, Dr. o'Gorman, 1987).

## B. Coronary Heart Disease

Two atherosclerotic diseases, coronary and cerebral artery disease, are the major causes of morbidity and mortality in Western Europe and North America (Scharf and Harker, 1987). There has been a steady decline in CHD mortality over the past three decades, though the reason for this decline is not entirely clear (Levy, 1981; Statistics Canada, 1985). While improved medical therapy has reduced Case fatality from CHD, a significant portion of the decline in mortality has been attributed to earlier detection and
treatment of coronary risk factors through both personal lifestyle modification and preventive medical therapy (Kuller, Perper, Dai, Rutlan, and Traven, 1986; Levy, 1981, Wing, 1984).

CHD and stroke result in $43 \%$ of all deaths and are the leading cause of premature (e.g. before the age of 60) death and disability in Canada (Statistics Canada, 1986). Roughly $25 \%$ of all disability pensions provided by the Canada Pension Plan are due to circulatory diseases, mainly CHD (Canada Pension Plan, 1985). Total health care costs associated with CHD are difficult to estimate, but undoubtedly they take up a large portion of Canada's health care expenses. CHD-related estimates of mortality, morbidity and heath care utilization are unknown for the Hutterites.

The exact mechanism(s) behind the development of the atherosclerotic lesions of CHD is(are) as yet unknown. However existing hypotheses of atherogenesis have received epidemiologic, pathologic and experimental support (Nilsson, 1986). The most widely accepted hypothesis of atherosclerosis is the so-called "Response-to-Injury" hypothesis (Ross and Glomset, 1976). According to this hypothesis there are sequential responses to intimal damage of blood vessels that include: 1) release of factors into the sub-endothelium resulting in the migration and proliferation of smooth muscle cells into the intima, 2) increased synthesis of collagen, of elastic fiber proteins and of proteoglycans by intimal smooth muscle cells, 3).
intracellular and extra cellular lipid accumulation, and 4) thrombus formation at the site of lesions (Scharf and Harker, 1987.) . Based on this hypothesis, formation of atherosclerotic lesions could be influenced by any factor that individually or in combination affected the integrity of the intima, the level of serum lipids, the proliferation of smooth muscle, and/or the function of platelets. The concept of "risk factors" for atherosclerosis has evolved over the course of the last three decades based on findings from epidemiologic studies. Population-based studies have demonstrated an association between CHD and 1). blood pressure, 2) serum and dietary cholesterol, 3) serum glucose, 4) smoking, 5) elevated BMI, 6) alcohol consumption, 7) psychological stress, 8) physical activity, 9) age, 10) sex, and 11) family history (Ross, 1988). While the last three risk factors are not amenable to modification, the first seven can be changed through alterations in lifestyle and personal habits. Thus CHD risk factors have been broadly divided in two categories: modifiable and non-modifiable.

## C. Risk Factors for Coronary Heart Disease

1. Modifiable Coronary Risk Factors.
a. Blood Pressure. While elevated blood pressure has been shown to strongly influence the development of coronary atherosclerotic lesions and to be associated with adverse coronary events (Messerli and Schmieder, 1986; MRFIT Research Group, 1986; Stamler,

Stamler, and Liu, 1985), the effectiveness of antihypertensive treatment in the prevention of CHD events has been called into question (Middeke and Holzgreve, 1988). While most research has evaluated the causative role of hypertension in CHD, there is some evidence that CHD may lead to hypertension in some patients (Hansson and Lundin, 1984) •

The epidemiology of hypertension is poorly understood, although there is substantial evidence linking obesity and excessive alcohol consumption to blood pressure elevation. The role of a variety of cations, including sodium, potassium, calcium and magnesium, in the evolution and treatment of hypertension is still unclear. Evidence linking hypertension to psychological stress, smoking, coffee consumption, fat intake and dietary fiber has been at best inconclusive (Joint National Committee, 1986). More recently, the central distribution of adipose tissue has been found to be predictive of essential hypertension, independent of the overall level of obesity (Selby, Friedman, \& Quesenberry, 1989).

Hypertension affects coronary pathology in two major ways: 1) it is postulated that elevated arterial pressure leads to intimal damage, and thus, the formation of atherosclerotic lesions via the Response to Injury hypothesis (Kannel, 1977), and 2) it directly influences the development of left ventricular hypertrophy (LVH), increasing the risk of congestive heart failure, thrombus
formation and lethal arrhythmias (Fogari and Zoppi, 1989). About half of the deaths in untreated hypertensives can be attributed to congestive heart failure. In contrast, only $10-12 \%$ of the deaths in untreated hypertensives are attributed to acute myocardial infarction (Doyle, 1988).

The risk of CHD continuously rises as blood pressure increases, with no threshold point of protection (Castelli and Anderson, 1986). Evidence from the Framingham Study suggests that hypertension increases cardiovascular mortality and morbidity by a factor of between two and four and the risk of a coronary event is proportional to either the systolic or diastolic blood pressure elevation, regardless of age or sex (Kannel, 1989).

It has been postulated that the decline in CHD mortality over the past three decades may be, in part, due to the lower incidence, and better treatment of hypertension (Stamler and Stamler, 1984). While it has been shown in animal studies that atherosclerosis can be accelerated by increasing blood pressure or retarded by lowering blood pressure (Hansson and Lundin, 1984), the evidence in humans is less convincing (Flamenbaum, 1989; Krzesinski, Carlier and Rorive, 1988). Both the Hypertension Detection and Follow-up Program (1979) and the Australian Therapeutic Trial (Management Committee, 1980) demonstrated that careful control of diastolic hypertension with medications could lower atherosclerosis-related mortality. However, more recent evidence suggests that some commonly used anti-
hypertensive medications (eg.beta-blockers and diuretics) may increase the risk of CHD because of their adverse effects on serum lipids and electrolytes (Fodor, 1986; Houston, 1989). There is better evidence that antihypertensive therapy may have a beneficial influence on hypertensive-related coronary disease related to reduction in left ventricular hypertrophy, not via a reversal of atherosclerotic lesions (Strauer, 1988).

While hypertension independently increases the risk of CHD, there are a number of factors that are believed to synergistically increase its effect. These cofactors include left ventricular hypertrophy, other non-specific ECG abnormalities, established coronary artery disease, hyperlipidemia, hyperglycemia, hyperuricemia, hyperfibrinogenemia, obesity, cigarette smoking, excessive sodium intake and excessive alcohol consumption (Taylor, 1987) .
b. Serum cholesterol. While the exact mechanisms are unknown, serum levels of cholesterol, particularly in its Low Density Lipoprotein (LDL) form, have been shown to be a predictor of CHD events (Castelli, 1986; The Expert Panel, 1988; MRFIT Research Group, 1986; Newman et al., 1986; Simons, 1986). However, there is still considerable controversy regarding the application of what is known about cholesterol to all age and sex-specific groups.

Cholesterol is transported through the body in the blood stream by lipoproteins. The lipoproteins that carry
cholesterol have been divided into five categories based on their relative densities. Thus serum cholesterol is actually a composite of the cholesterol carried by chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL), intermediate density lipoproteins (IDL) and high density lipoproteins (HDL). Approximately seventy percent of all cholesterol is carried by LDL (Rifai, 1986), and indeed, total serum cholesterol is a useful surrogate for LDL (Grundy, Goodman, Rifkind, and Cleeman, 1989).

LDL is the most prevalent fraction of cholesterol in : the circulation and it is readily utilized by cells lining the intima of the arteries. This relationship may help explain why LDL, albeit through mechanisms yet unknown, is implicated in the etiology of CHD (Grundy et al., 1989). The high LDL levels found in western society are thought to be the result of not only a high dietary intake of fat, but possibly due to an inheritable relative lack of LDL receptors (Brown and Goldstein, 1984). While many types of severe hyperlipoproteinemias are genetic, these extreme forms of lipid disorders are relatively rare (Gotto, 1984). Strengthening the argument for an etiological role of LDLcholesterol in CHD etiology, are the results of the clinical trials that demonstrate the beneficial effects of lipid lowering diets and drugs on coronary events in middle aged men (Lipid Research Clinics' Program, 1984; World Health Organization, 1980). However, these same clinical trials
have not shown that a reduction in serum cholesterol has any effect on overall mortality.

Next to LDL-cholesterol, HDL-cholesterol has received the most attention in relation to CHD. Evidence of the protective effects of HDL-cholesterol has come from both population-based and pharmacologic intervention studies. While some population studies have demonstrated an inverse relationship between HDL-cholesterol and CHD (Goldbourt, Holtzman and Neufeld, 1985; Kannel, Castelli, and Gordon, 1979), others have not (Knuiman, West, Katan, Hautvast, 1987; Robinson and Williams, 1979). The salutary effects of HDL-cholesterol have received greater support from clinical trials. The Helsinki Heart study found that the ability of gemfibrozil to lower CHD risk was, in part, due to its ability to increase HDL-cholesterol fractions (Frick et al., 1987). Similarly, the Cholesterol-Lowering Atherosclerosis Study found that diet, in combination with bile acid sequestrants and nicotinic acid, contributed to decreases in LDL-cholesterol and significantly increased HDL-cholesterol fractions, leading to a reduction in atherosclerotic plaques (Blankenhorn et al., 1987).

Recent developments in the field of isolating particular apolipoproteins, proteins bound to the outer membrane of lipoproteins, hold promise for more precisely determining individuals at risk for atherosclerosis (Ross, 1988). Low levels of a particular apolipoprotein, apo-AI, have been demonstrated to be a predictor of atherosclerosis
and may help improve the ability of researchers to investigate CHD in future epidemiologic studies (Kottke et al., 1986). Compared with the more conventional method of measuring the ratio of total cholesterol to HDL cholesterol ratio (Kannel, 1988), the apo-AI to apo-B ratio has been found to be more highly correlated with CHD (Rifai, Chapman, Silverman, and Gwynnes, 1988).

As with hypertension, a graded, continuous relationship between serum cholesterol and CHD events has been demonstrated for middle aged men (Kannel, 1988; Kannel, Castelli, and Gordon, 1979). However, this graded relationship between cholesterol and CHD is not supported for men over 65 years of age or middle aged women (Garber, Sox, and Littenberg, 1989). Women under 65 years of age have significantly lower rates of CHD at the same cholesterol levels as men and only begin to experience an increase in CHD events when their cholesterol is above 6.71 mmol/l (Bush, Fried and Barrett-Conner, 1988). While the relationship between CHD incidence and cholesterol in men over the age of 65 years is not believed to be significant, it is significant for women (Kannel, 1988). More recent evidence from the Framingham Study would suggest that, at least in males 60-70, there is still a significant risk of CHD associated with an elevated cholesterol (Castelli, Wilson, Levy, and Anderson, 1989).

Approximately one third of the cholesterol circulating in the blood is due to dietary intake of food products
containing cholesterol and saturated fats (Ross, 1988). Serum cholesterol can be significantly modified through changes in the diet for individuals with elevated levels (The Expert Panel, 1988). Reductions in dietary intake of saturated fats and cholesterol to $7 \%$ of daily caloric intake has been shown to decrease plasma total cholesterol and LDL by 10-20\% (Kris-Etherton et al., 1988). A number of international population studies have also demonstrated a strong correlation between dietary intake of saturated fat and subsequent coronary events (Phillips, Lemon, Beeson, and Kuzma, 1978; U.S. Department of Health, 1988).
c. Serum glucose and diabetes. Elevated fasting serum glucose, one of the hallmarks of diabetes mellitus, has long been known to accelerate the development of atherosclerosis (Freedman, Guchow, Bamrah, Anderson, and Barboriak, 1988). Epidemiologic evidence from a number of studies has shown a positive relationship between diabetes and coronary events (Assmann and Schulte, 1988; Fuller, Shipley, Rose, Jarrett, and Keen, 1980; Jarrett, McCarney, and Keen, 1982; Kannel and McGee, 1979; Kannel, 1985), regardess of whether the diabetes is of the insulindependent (IDDM) or the non-insulin-dependent type (Laakso, et al., 1988).

Multiple mechanisms have been suggested for explaining the increased susceptibility to atherosclerosis in diabetics. Because diabetes is a disease of metabolism, not only are serum glucose levels elevated, but serum lipids are
affected as well (Ruderman and Haudenschild, 1984). Abnormal blood lipids in diabetics that are associated with an increased risk for CHD include elevated very low density lipoproteins (VIDDI) and triglycerides, lowered $H D L / L D L$ ratios, and lowered HDI (Kannel, 1985). Growth hormones and growth factors which may affect the proliferation of smooth muscle are also elevated in diabetics. Because of microcirculatory changes found in diabetics, particularly in their kidneys, they are more prone to hypertension (Ruderman and Haudenschild, 1984). Changes to the myocardium, independent of the vascular changes seen in diabetes, also put diabetics at greater risk for developing cardiovascular disease, particularly cardiomyopathy (Regan, 1989).

Unlike blood pressure and cholesterol, the relationship between serum glucose and $C H D$ does not show a graded or linear relationship, but appears to have a threshold level above which risk greatly accelerates (Welborn, 1984). Diabetes is closely associated with a number of other CHD risk factors. The Prospective Cardiovascular Munster (PROCAM) study has shown that 1) more than half of all. diabetics are also hypertensive and 2) $30 \%$ of all diabetics have a total serum cholesterol $>7.8$ mmol/l (Assmann and Schulte, 1988). The impact of diabetes on the risk of developing $C H D$ is greater in women than men and predisposes them to higher rates of cardiac failure and coronary death (Kannel, 1985). The risk of recurrent myocardial infarction
in diabetic women is twice the risk in men (Abbott, Donahue, Kannel, and Wilson, 1988).
d. Smoking. While the mechanisms involved in the relationship between coronary events and smoking are as yet unknown, two hypotheses under consideration are that constituents of smoke: 1) accelerate atherosclerosis and/or, 2) initiate occlusion of coronary arteries either by vasospasm and/or thrombosis.

Nicotine has been found to significantly decrease the levels of HDL-cholesterol and, to a lesser extent, increase levels of LDL-cholesterol (McGill, 1988; Tiwari, Gode, and Dubey, 1989). Smoking affects the proliferation of medial smooth muscle in arteries, increasing the possibility of either vasospastic or complicated atherosclerotic occlusion of the coronary arteries (Naeye and Truong, 1977; McGill, 1988).

The increased plasma fibrinogen levels found in smokers (Wilhelmsen et al., 1984) places them at greater risk for both atherogenesis and acute coronary thrombosis (MCGill, 1988). In individuals with fixed atherosclerotic lesions, nicotine-stimulated catecholamine release increases myocardial oxygen demand and may also increase coronary vasospasm leading to acute cardiac ischemia (Fox, 1988).

According to the Canada Health Survey, approximately one-third of all Canadians smoke tobacco (Health and Welfare, Canada, 1981), although more recent surveys would suggest this may be decreasing in all age and sex categories
except for young females (Health and Welfare, Canada, 1988). A long series of epidemiologic studies have shown a doseresponse relationship between coronary events of all kinds and cigarette smoking (Multiple Risk Factor Intervention Trial Research Group, 1986). However, this is not to say that all studies are in agreement.

A recent study from Sweden was unable to show a relationship between smoking and coronary events in women (Lapidus, Bengtsson, Lindquist, Sigurdsson and Rafnsson, 1986). Both the Framingham Study' and the Goteborg Primary Prevention Study have failed to demonstrate a relationship between smoking and angina pectoris (Kannel, McGee, and Castelli, 1984; Wilhelmsen et al., 1986). Evidence from the Framingham study demonstrates that smokers of both sexes are at a significantly higher risk than non smokers for myocardial infarct, sudden cardiac death and overall CHD mortality; however, there were no differences in relative risks for angina pectoris (Kannel et al., 1984).

In a recent review of current studies of smoking and CHD, Wilhelmsen (1988) concluded that the relative risks of nonfatal and fatal myocardial infarct is 2-3 times higher in men who smoke than in male nonsmokers and 1.5-3 times more common in women who smoke than in female nonsmokers. It is estimated that $30 \%$ of all CHD deaths are attributable to cigarette smoking (National Institutes of Health, 1977). e. Elevated BMI. While obesity, or an elevated Body Mass Index (BMI) above $27 \mathrm{~kg} / \mathrm{m}^{2}$ has been linked to
elevated serum cholesterol (Knuiman, West and Burema, 1982), hypertension (Johnson, Cornoni, and Cassel, 1975), and diabetes (Kannel, 1985), there is still controversy over whether it is an independent risk factor for CHD (BarrettConner, 1985; Feinleib, 1985).

The Framingham Heart Study (Hubert, Feinleib, McNamara, and Castelli, 1983) was able to demonstrate that there was a relationship in men between body weight and all disease categories combined, angina, sudden death and congestive heart failure. However, no relationship was found between CHD and either myocardial infarction; or atherothrombotic stroke, or death from cardiovascular disease. In women a relationship was found between weight and all CHD endpoints except sudden death. In a more recent analysis of the Framingham Study data, central torso obesity and BMI were both found to be independent predictors of CHD (Higgins, Kannel, Garrison, Pinsky, and Stokes, 1988).

Despite these findings, in a review of 27 large population studies Stockwell, Periera, White and Coltereau (1985) found that body weight ceases to be an important independent predictor of CHD endpoints when other important CHD risk factors are controlled for in the analysis. These inconsistencies in large scale epidemiologic studies have lead some researchers to conclude that there is insufficient evidence to conclude that obesity is a reliable indicator of CHD risk (Barrett-Conner, 1985).

More recent research would suggest that the central distribution of fat on the body (e.g. the torso) may be more important in predicting CHD than the relative body weight (Ducimetire and Richard, 1989; Lapidus et al., 1984). There is growing evidence that centrally deposited fat may, independent of BMI , predict the risk for developing CHD and increases the risk for an number of other CHD risk factors. Fat deposited on the torso has been associated with elevated serum lipids (Reichley, Mueller, Harris, 1987), hypertension (Blair, Habicht, Sims, Sylvester, and Graham, 1984; Selby et al. 1989; Shear, Freedman, Burke, Harsha, and Berenson, 1987), and diabetes (Ohlson, 1985).

An elevated waist to hip ratio (WHR), indicative of central torso obesity, has been associated with elevated plasma lipid and apolipoprotein concentrations that are predictive of CHD for men (Barakat, Burton, Carpenter, Holbert, and Israel, 1988). The WHR has also been found to be associated with poorer control of non-insulin-dependent diabetes and increases the risk of vascular complications of diabetes (VanGaal, Rillaerts, Creten, and De Leeuw, 1988). f. Alcohol consumption. The effects of alcohol on CHD appear to be closely related to the quantity consumed, though there continues to be controversy over this point. Moderate alcohol intake, defined as 2-3 alcoholic beverages per day, has been associated with a decreased risk of coronary death. Two international studies, one conducted in 18 countries, and one conducted in 20 countries, found.that
there was an inverse relationship between CHD mortality and moderate to heavy alcohol consumption in subjects ages 55-64 years (LaPorte, Cresanta, and Kuller, 1980; St. Leger, Cochrane, and Moore, 1979). Similarly, a study conducted in the Kaiser-Permamente Health Maintenance Organization, found that patients who consumed $1-2$ alcoholic beverages per day were at lower risk for CHD than either abstainers or heavy drinkers (Klatsky, Friedman, and Siegelaub, 1981).

Diabetes could theoretically be influenced by alcohol intake and thus increase the risk of CHD, however, there is little evidence to support this premise. Alcohol abusers are at an increased risk for developing diabetes (Lindegard and Langman, 1985), however no such relationship has been proven for moderate alcohol consumption. After controlling for BMI, women in the Nurses Health Study who drank 5-14.9 g of alcohol per day were at no greater risk for diabetes than non-drinkers (Stampfer, 1988). Similarly, glucose intolerance has not been found to be related to moderate alcohol intake (Nilsson-Ehle, 1979).

Alcohol consumption has been linked to hypertension, yet there is still uncertainty of whether this increases CHD risk. A recent survey found that there was a statistically significant association between blood pressure and alcohol intake, but only in those consuming more than one drink per day (Weissfeld, Johnson, Brock, and Hawthorne, 1988). A dose-response between alcohol and CHD risk has been observed, with risk of hypertension increasing only above an
average alcohol consumption of more than two drinks per day (Klatsky, Friedman, Siegelaub, and Gerard, 1977).

There is considerable evidence that moderate alcohol consumption improves lipid profiles, particularly for HDLcholesterol (Kris-Etherton et al., 1988). Two large epidemiologic studies, the Cooperative Lipoprotein Phenotyping Study and the Multiple Risk Factor Intervention Trial demonstrated a direct relationship between alcohol Consumption and HDL-cholesterol (Castelli et al, 1977; Hulley, Ashman, Kuller, Lasser and Sherwin, 1979). There is growing evidence that alcohol's beneficial effect on HDL- . cholesterol results from its ability to increase the microsomal enzyme activity of the liver (Luoma, 1988).
g. Psychological stress. The role of
psychological stress in atherogenesis and coronary events remains controversial. While stress has been shown to acutely elevate serum catecholamines, serum cholesterol, serum glucose, and blood pressure, there is little evidence that multiple short term changes in these factors lead to chronic conditions such as hyperlipidemia, diabetes, or hypertension (Mathews and Haynes, 1986). In a recent investigation, no relationship was found between the pressor response to stress and three measures of Type-A personality (TAP) (Jenkins Activity Survey, Framingham Type-A Scale and the Bortner Type-A Scale) (Gallagher, Beswick, Jones, Turkington, 1988).

Despite findings from a number of studies in the 1970 s that coronary events were significantly related to the stressful TAP (Brand, Rosenman, Sholz, Friedman, 1976; Rosenman et al., 1975), more recent evidence has failed to confirm these results (Cinciripini, 1986; Ragland and Brand, 1988).

TAP, characterized by competitiveness, impatience, hostility and time urgency, has been used as a measure of stress and as a predictor of CHD for a number of years (Helgeson, 1989; Jenkins, Zyzanski, Rosenman, 1979).

While early reports from both the Framingham Study and the Western Collaborative Study Group (WCSG) fueled speculation that there was a link between CHD and TAP, more recent evidence would suggest that subsets of TAP individuals may be more at risk than others, particularly those at the extreme end of the TAP scale (ShohamYakubovich, Ragland, Brand, and Syme, 1988). It has been suggested that the measurement of hostility, one of the subcomponents of TAP, may be the most important single measure of CHD risk (Hecker, Chesney, Black and Frautschi, 1988; Reunanen, 1988).

More research will be needed, using tools that are more sensitive to variations in stress, before the role of psychological stress in CHD etiology can be established. h. Physical Activity Physical activity is thought to have both direct and indirect effects on CHD etiology (Blair, Kannel, Kohl, Goodyear and Wilson, 1989).

The lack of cardiovascular conditioning associated with a sedentary lifestyle may place the myocardium at increased risk of ischemia during strenuous exertion (Johansson et al, 1988). Beyond this direct effect of physical inactivity on cardiovascular disease, a sedentary lifestyle increases the risk for developing other CHD risk factors such as obesity, hyperlipidemia, hypertension and diabetes (Blair and Oberman, 1987).

Exercise training has been shown to favorably affect a number of coronary risk factors including: 1) reduction in resting blood pressure, 2) weight loss, and 3) increasing the HDL/LDI serum cholesterol ratio (Butler and Goldberg, 1989; Hartley, 1985). Methodological problems in quantifying and validating exercise levels have hampered population based studies (Wilson, Paffenbarger, Morris, Havlik, 1986). As complicated as measurement of physical activity may be, Blair et al. (1989) have recently shown that other factors such as relative weight, resting heart rate and vital capacity may serve as useful surrogates for the measurement of physical activity and physical fitness in population surveys.

Findings from the Framingham Study (Kannel, Belanger, D'Agostino, and Isreal, 1986) suggest that there is an inverse relationship between 24-hour activity levels both on and off the job and coronary mortality in men of all ages. Paffenbarger, Wing, and Hyde (1978), in a longitudinal study of over 16,00 Harvard alumni, have also shown an inverse
relationship between exercise and coronary events. Both the Framingham Study and Paffenbarger et al.'s study indicate that the amount of exercise needed to provide a protective effect is relatively low. Hagan (1988) suggests that the minimal exercise required for optimizing the caloric input/output ratio is $20-30$ minutes of exercise at $60 \%$ maximal heart rate, 3 times per week.

Recently released findings from the Framingham Offspring study show that there is an inverse relationship between physical activity and cardiovascular risk (Dannenberg, Keller, Wilson and Castelli, 1989). In this. same study, there was a direct relationship between an increase in physical activity and increase in HDL cholesterol, lower heart rate, lower BMI and less cigarette use for both sexes. This report also stresses the importance of taking seasonal variation in physical activity into account when examining the relationship between physical activity and cardiovascular disease.

The positive effects of exercise on CHD risk have not been demonstrated in all population-based studies. Results from a large cohort study in Finland found that, while leisure time exercise was significantly associated with reduced risk for $C H D$ in the univariate analysis, this effect was no longer present after controlling for other risk factors (Salonen, Slater, Tuomilehto, and Rauramaa, 1988).

## 2. Non-Modifiable CHD Risk Factors

a. Age and Sex. CHD events and coronary
mortality rates begin to rise from the third decade of life at a progressive rate until the seventh or eighth decade of Iife, where they plateau and then fall with further aging (Dawber, 1980). The prevalence of CHD in pre-menopausal women is significantly lower than in men, but postmenopausal women begin to have similar rates to men around the sixth decade of life (Bengtsson, 1973; Wilcosky, Harris, and Weissfeld., 1987). Indeed, cardiovascular diseases account for a higher proportion of age-adjusted deaths among women than men (52\% vs $46 \%$ ) (Lerner and Kannel, 1986).

Presumably, aging affects CHD risk through a number of mechanisms. Atherosclerotic lesions are known to progress in number and complexity as we age. The prevalence of the following CHD risk factors are known to rise with age: 1) obesity, 2) hypertension, 3) diabetes, and 4) decreasing physical activity (Ross, 1988).
b. Family History and Coronary Heart Disease.

While there is little question that CHD clusters in families, debate revolves around the mechanism of transmission (Perkins, 1986). There are two schools of thought in this regard, each with evidence to support their respective viewpoints. The first posits that the relationship is risk factor dependent, i.e. that the familial clustering of $C H D$ results from a clustering of other known coronary risk factors such as hypertension;
diabetes, elevated serum cholesterol and smoking. The second holds that family history is a risk factor independent of familial clustering of other CHD risk factors.

In support of the 'risk factor-dependent' view a number of epidemiologic studies have found that when other known coronary risk factors are controlled for in the analysis, the effect of a positive family history on CHD transmission is statistically non-significant (Conroy, Mulcahy, Hickey, and Daly, 1985; Rosenman et al. 1976; Rissanen, 1979b). In a case-control comparison of 203 men who had a positive family history with 106 controls, Rissanen (1979b) concluded that. family history is an important, but not an independent, risk factor for CHD. Conroy et al. (1985) in a cross-sectional design compared the family histories of 792 patients who had survived a coronary event and found that there was no evidence that a positive family history was an independent risk factor for CHD. In a prospective study of CHD, Rosenman et al. (1976b) found that family history was not an independent risk factor for ischemic heart disease in 257 patients who suffered a coronary event.

However, the evidence supporting a 'risk factorindependent' viewpoint is equally compelling, with a number of studies finding that family history is a statistically significant predictor of CHD after adjusting for the presence of other risk factors (Colditz et al.., 1986; Epstein, 1976; Nora, Lortscher, Spangler, Nora, and Kimberling, 1980; Shea, Ottman, Gabrieli, Stein, and.

Nichols, 1984). Khaw and Barrett-Conner (1986) examined the role of family history in CHD in a prospective study of 4014 adults and concluded that, when other important risk factors were taken into account, family history was an independent predictor of coronary events. Shea et al. (1984), in a comparison of 223 CHD patients with a family history of CHD with 57 control subjects, found that family history is an independent risk factor for coronary artery disease. They also concluded that family history may be most important in subjects who are otherwise at low risk for CHD. Snowden et al. (1982) report that the incidence of myocardial infarction in siblings is strongly, and independently, related, even after controlling for other important risk factors such as smoking, cholesterol; and blood pressure. Sholtz, Rosenman, and Brand (1975) found an independent relationship between parental history of CHD and CHD in participants in the prospective Western Collaborative Group study.

Perkins (1986) has criticized the results of all studies of family history of CHD on methodologic grounds. His concerns with prior research include 1) incomplete assessment of other known risk factors, 2) failure to control for other known risk factors in the analysis, and 3) lack of validation of diagnoses. Another major methodological problem with previous CHD-family history research has been an inability to adequately control for
potentially important inter-generational differences in lifestyle factors such as diet, exercise, and smoking.

Ample evidence exists to support the important role played by genetics in CHD transmission through either known, or as yet unrecognized, risk factors (Goldbourt and Neufeld, 1986). At the same time, lifestyle factors such as exercise and diet have been shown to significantly influence the phenotypic expression of obesity (Simic, 1983), of serum lipids (Hautvast, Knuiman, West, Brussard, and Katan, 1983; Grundy, 1984), and of blood pressure (Joossens and Geboers, 1983) -

When this issue of the relative contribution to disease of lifestyle and genetics is examined in general population studies, the inter- and intra-generational variability of lifestyle factors frequently obscures the results (Edwards, 1969). Thus well defined populations with homogeneous lifestyles, such as the Hutterites, are better suited than the general population for examining the relative role of genetics and lifestyle in the etiology of disease (Hauck and Martin, 1984). While the present study does not propose to directly examine the issue of lifestyle vs. genetics, it is anticipated that future studies in the Hutterite population may do so.

Most epidemiologic studies have treated 'family history' as a categorical variable, with CHD in

1) a parent (Deutscher, Ostrander, and Epstein, 1970; Sholtz et al., 1975i),
2) a sibling (Snowden et al., 1982),
3) a parent, sibling, or a grandparent (Hamsten and de Faire, 1987; Rissanen and Nikkila, 1979; Rose, 1964; Slack and Evans, 1966; Shea et al., 1984; Conroy et al., 1985; Khaw and Barrett-Conner, 1986),
defining a case of "positive family history". The age of onset of CHD in the first degree relative has also been used in stratifying family history. A CHD event in a relative, . particularly in a parent, before the age of 55 years is commonly used as the operational definition of premature CHD (Khaw and Barrett-Conner, 1986; Rissanen, 1979a; Conroy et al., 1985; Deutscher et al, 1970).

## D. CHD and Coronary Risk Factors in the Hutterite

## Population

Despite anecdotal reports that CHD may be an important health problem among Hutterites (personal communication, Dr. Fowlow, 1988, Dr. Morgan, 1987), the CHD incidence, prevalence and mortality among the Hutterites has never been reported. However, clues to the prevalence of coronary risk factors in the Hutterite population do exist. Data gathered in the late 1950s suggested that Hutterites had generally similar age and sex-specific distributions of serum cholesterol and blood pressures as the population of a typical North American city, Tecumseh, Michigan (Goldbarg,

Kurczynski, Hellerstein, and Seinberg, 1970). These researchers also reported a higher prevalence of ECG abnormalities possibly indicative of ischemic heart disease among the Hutterites, but no statistical comparisons were made with the reference group. Anthropometric measures indicate that central torso obesity is a common observation among the Hutterites as they approach middle age (Howells and Bleibtreu, 1970). However, because only age and sexspecific group means have been published from these studies, there is no way to determine the exact prevalences of hypercholesterolemia, hypertension, abnormal ECG and obesity. Similarly, while there is anecdotal information suggesting that diabetes may be prevalent in some Hutterite colonies (Fowlow, 1973), no prevalence or incidence rates have ever been published.

A recent study found that Hutterites had significantly higher diastolic and systolic blood pressures and had higher BMIs than non-Hutterites (Schlenker, Parry, and McMillan, 1989). In this same study, the prevalence of a diastolic blood pressure $>90 \mathrm{~mm}$ Hg ranged from $27.5 \%$ to $37.9 \%$ for males depending on age, and ranged from $6.0 \%$ to $45.2 \%$ for the females.

Other evidence bearing on the discussion of CHD risk factors among the Hutterites comes from their lifestyle habits and beliefs. As previously mentioned, smoking is rare among the Hutterites due to social norms. Schlenker et al.
(1989) found the active smoking rate to be about $5.0 \%$.

Adherence to this precept is validated by the Hutterite's very low incidence of lung cancer (Gaudette, Holmes, Laing, Morgan, and Grace, 1978; Martin et al., 1980).

The Hutterite diet, high in meat and fat content (Rokusek-Kennedy, Parry, and Schlenker, 1987), has been shown in other populations to be a strong correlate of CHD mortality (Berkel and de Waard, 1983; Phillips et al., 1978; Snowden, Phillips, and Fraser, 1984).

While the role of stress in CHD etiology remains controversial, there is evidence that the Hutterite lifestyle may be marked by less anxiety and stress than found in the general population (Eaton, Weil, and Kaplan, 1951).

Nothing is known about the aerobic fitness of Hutterites, though recreational weight lifting among the young men of some colonies has been reported (personal communication, Dr. Fowlow, 1988).

The inbreeding observed among the Hutterites would suggest that the potential for homozygosity of otherwise rare metabolic or functional correlates of CHD may be higher than in the general population (Hartyl, 1981). However, this assumption has never been examined.
E. Determining CHD Prevalence

Prevalence rates of CHD, as for all diseases, are related to the type of measures used to determine cases (Wilcosky et al., 1987). To ascertain CHD cases, studies have used either symptoms alone (Health and Welfare, Canada,

1981; Reid, 1966; Rose, 1962), symptoms in combination with corroborative physical exams or laboratory tests (Bengtsson, 1973; Lapidus, Begtsson, Lindquist, Sigurdsson, and Rafnsson, 1985; Reunanen et al., 1983), or medical histories of coronary events (Heyden et al., 1971; Lapidus et al., 1985; Wilcosky et al., 1987) . Medical histories of myocardial infarction, abnormal resting or exercise ECGs, and abnormal angiography have been used to either validate or ascertain CHD cases in prevalence studies (Areskog, Tibbling, and Bengt, 1981; Wilcosky et al, 1987; Heyden et al., 1971; Dawber, 1980). However, such in-depth exams are often not feasible due to cost and/or personnel restraints when conducting population studies or public screening programs (Friedman and Byington, 1985).

The need for a valid and reliable tool for ascertaining CHD cases in epidemiologic studies was recognized by Rose and resulted in his development of the Rose Angina Questionnaire (RAQ) (Rose, 1962). Rose Angina is said to be present when the respondent reports all of the following symptoms or actions:

1) ever had pain in the chest
2) chest pain when walking or in a hurry
3) stops or slows down when this pain occurs
4) pain is relieved by standing still
5) pain goes away within 10 minutes
6) location of pain is substernal or in the left arm and left anterior chest

The RAQ has been widely used in a variety of settings and geographical locations (Areskog et al.,1981; Bengtsson, 1973; Friedman and Byington, 1985; Heyden et al., 1971;

Lapidus et al., 1985; Reid, 1966; Reunanen et al., 1983; Rose, 1962; Rose, McCartney, and Reid, 1977; Wilcosky et al., 1987). As a predictor of future CHD mortality and morbidity, the RAQ has been shown to be as good as a physician's exam and medical history (Friedman et al, 1985; Heyden et al., 1971). The RAQ's validity is borne out by its ability to show expected relationships between angina and other selected CHD risk factors such as previous MI, age, sex, and smoking status (Reid, 1966; Wilcosky et al., 1987). The reported sensitivity, specificity, and predictive value of the RAQ in a number of population-based studies supports its value for epidemiologic research (Friedman et al., 1985; Heyden et al, 1971).

While the original RAQ was administered by an interviewer, a self-administered form has been developed and found to be just as useful in predicting future coronary events (Rose et al., 1977; Wilcosky et al., 1987). By using the RAQ in either its self- or interviewer-administered form a researcher is able to make comparisons between the population of interest and other groups previously studied with this instrument at a relatively low cost.

The prevalence of Rose Angina in populations has consistently been demonstrated to increase with age in both men and women (Reunanen et al., 1983; Bengtsson,1973; Wilcosky et al., 1987). The overall prevalence of Rose Angina ranges from $3-12 \%$, depending on the population under
study and the version of the RAQ used(Reid et al., 1966;
Reunanen et al., 1983, Wilcosky et al., 1987).

## CHAPTER 2: METHODS USED FOR THE STUDY

## I. Study Design

A cross-sectional survey was utilized to collect information from adult Dariusleut Hutterites ages 30-74 years residing on 38 colonies in Alberta regarding the prevalence of Coronary Heart Disease (CHD) and its associated risk factors. Physician verification of selfreported medical histories of CHD, cerebrovascular accident, diabetes mellitus, hypertension and hyperlipidemia was also sought.

## II. Inclusion Criteria

In order to be eligible for the study a participant was: a) a resident of a Dariusleut colony with an Alberta postal code, b) between thirty years and seventy-four years of age by December 31, 1989, and c) present at the time of the survey. The operational definition of a "resident" was that the participant's regular and usual residence was in a colony that had elected to be included in the study.
III. Instruments Used for Measurement

## A. The Personal Health Survey (PHS)

The PHS (Appendix 1), specifically developed for this study, is a self-administered questionnaire that surveys six dimensions of a participant's past and present health status:

1. demographic characteristics,
2. lifestyle and personal habits,
3. medication and health care utilization,
4. evidence of Rose Angina,
5. past medical history, and
6. family history of heart disease.

Content validity was provided by a review of the questionnaire by Faculty and Graduate Students in the Faculty of Medicine at the University of Calgary. A pilot test of the PHS was conducted on August 22, 1988 to assess whether it was an appropriate instrument for use in the Hutterite population. It was particularly important to determine whether the questions were written at an appropriate cognitive level given some concerns regarding the Hutterite's education in English as a written language.

Five elders from one of the participating colonies (colony \#61) completed the questionnaire under the direction of a research assistant. Both the general format and the content of the PHS proved to be satisfactory, although some minor revisions in layout were recommended by the elders. The elders had minimal difficulty understanding the questions as written in English. Based on the recommendations from the elders, however the layout of the questionnaire was revised to facilitate its ease of completion.

The demographic characteristics measured included:

1) birthdate,
2) birth colony,
3) duration of residence in present colony,
4) sex,
5) marital status, and
6) work responsibilities.

Information was gathered regarding a number of lifestyle factors and habits related to risk of CHD:

1) alcohol consumption,
2) tobacco usage,
3) dietary intake,
4) sodium intake,
5) work related exercise, and
6) leisure-time exercise.

Participants were also asked to rate how stressed they usually felt in their daily lives.

Detailed information was gathered about the usage of both prescription and non-prescription medications. All hospitalizations, except for normal childbirth, were documented as to their purpose and year of admission. Participants were also requested to provide information about any contacts they have had with a physician in the last year, whether it led to hospitalization or not.

The Rose Angina Questionnaire, a questionnaire widely used in population studies, was used to identify participants with the symptoms of typical angina pectoris.

The participant's past medical history was gathered using a check list for a number of common medical conditions. This check list gathered information similar to that gathered in the Canada Health Survey (Health and

Welfare Canada, 1981). In addition to general medical histories, indepth histories were gathered for anyone with a history of heart disease, stroke, diabetes, hypertension and/or hyperlipidemia; the information gathered included the onset of the disease, hospitalizations, and past and current therapy modalities.

Information about a family history of CHD was gathered for all first, second, and third degree relatives (parents, grandparents, aunts and uncles, and siblings). For each relative with a history of CHD the respondents were also requested to specify whether its onset was before, or after, the age of 55 .

## B. Cholesterol Measurement

Non-fasting total cholesterol was measured using the REFLOTRON dry chemistry reflectance photometer (Boehringer Mannheim Inc.) . Advantages of the REFLOTRON over conventional wet lab measures include:

1. ease of blood sample procurement
2. results are available within 3 minutes
3. reduces the problems of inter-laboratory variation
4. low cost per test.

The accuracy and precision of the REFLOTRON have been demonstrated in both industry supported (Boehringer Mannheim, 1985) and independent studies (Burke and Fisher, 1988). While the REFLOTRON has many desirable features which are ideal for population screening, Greenland (1988) has reported that this system tends to provide
systematically higher cholesterol values than venous samples tested by CDC standardized equipment.

## C. Glucose Measurement

Two hour post-prandial capillary glucose was measured using the GLUCOMETER II/GLUCOSTIX SYSTEM (Ames Division, Miles Laboratories, Ltd.). This system is widely used by both individuals and institutions to monitor diabetic control. Compared to other self-testing devices and wet lab results, the GLUCOMETER II has proven to be both accurate and precise (Brooks, Rawal \& Henderson, 1986; Peterson, Peterson, Dudley, Kilo, and Ellis, 1988).

## D. Blood Pressure Measurement

Blood pressure was measured using a mercury sphygmomanometer and a stethoscope.
E. Height and Weight Measurement

A Seca Medical Weight Scale was used to measure the respondents weight. Height was measured using a wall-mounted measuring tape.
IV. Referral Criteria and Follow-up Procedures

Each participant and, with the participant's consent, their physician, was provided with a copy of results of their blood pressure, cholesterol, glucose and body mass index. Based on the subject's measurements, recommendations were made for whether and when to seek further physician follow-up.

The recommendations for diastolic blood pressure were based on the guidelines developed by the canadian Coalition
for the Prevention and Control of High Blood Pressure (1988). To be as safe as possible, referrals were based on the highest diastolic pressure recorded. Because of concerns on the part of the Thesis Supervisory Committee, one modification was made to the recommended guidelines: if the diastolic pressure is between 100 mm Hg and 114 mm Hg , the Canadian Coalition calls for physician follow-up within one month; this was reduced to two weeks for participants in this study. As there is no consensus on referral guidelines for systolic pressure at the present time, no referrals were made for isolated elevated systolic blood pressure.

Based on the recommendations of Dr. Stuart Ross, a diabetic specialist at the Foothills Hospital, and the National Diabetes Data Group (1979), participants with a two hour post-prandial glucose above $11.1 \mathrm{mmol} / \mathrm{L}$ were advised to see their physicians within two months for further studies. These same guidelines have been recently recommended by the American Diabetes Association (1989).

Following referral guidelines developed by the Canadian Consensus Conference on Cholesterol (1988), any participant with a random total cholesterol above 6.2 mmol/L was advised to talk with their physician within four to six weeks about having a fasting cholesterol drawn.

While no specific referral guidelines exist for an elevated body mass index (BMI), a BMI greater than $27 \mathrm{Kg} / \mathrm{m}^{2}$ is associated with a significant increase in the risk of a number of chronic diseases including CHD and cancer (Health
and Welfare Canada, 1988). Based on these findings, it was deemed prudent to recommend that participants with a BMI above 27 seek assistance to help them lower their weight. If a participant provided consent, we sent a copy of their results and recommendations to their family physician. In addition, if a participant was found to have a diastolic pressure above 114 mm Hg we immediately sought their permission to phone their physician directly from the colony.

## V. Data Collection

## A. Recruitment of Colonies

A list of Dariusleut colonies in Alberta was provided by Michael Tschetter of Fairview Hutterite colony on June 26, 1987. Letters were sent to the ministers in each of the 68 Dariusleut colonies on September 29, 1987 inviting the participation of their colony in the study. A second letter was mailed on March 16, 1989 to colonies that had not responded to the initial letter. While it was anticipated that 20-30 colonies would ultimately agree to participate, a total 35 colonies responded affirmatively to the initial and second mailing. At their request, an additional 3 colonies were added as the study was underway. Three other colonies also requested that they be included in the study late in the data collection period; however, due to budget constraints, they were not added to the sample.

## B. Questionnaire Administration

Although the PHS was designed to be self-administered, it was also used during interviews of participants who were unable, for any reason, to complete the questionnaire themselves. The PHS was coded with a unique identification number for each participant in order to protect their anonymity. The questionnaire was administered to the entire colony in a common meeting room, most commonly the dining hall or school. Only rarely was it necessary to make a special visit to someone's apartment in order to administer the questionnaire.

Younger members of the colony often circulated among the participants to help them complete the questionnaire. It was also common to have participants talk amongst themselves while completing the questionnaire. On average, the PHS required between 30 and 60 minutes to complete.

When a participant was finished with the PHS the Principal Investigator reviewed the entire questionnaire with him/her in order to make certain that all of the questions had been answered. In addition, this exit interview provided the participants and the researcher with the opportunity to ask further questions about the PHS and to seek clarification of any ambiguous answers.
C. Procedures Used for Physical and Laboratory Measures

## 1. Cholesterol

Cholesterol testing was performed by a trained Research Assistant or by the Principal Investigator. The precision
and accuracy of the REFLOTRON was tested using a manufacturer recommended procedure prior to gathering data in every colony. Briefly, a REFLOTRON-CHEK strip was used in order to determine if the optical components of the system were operating within a pre-determined range of accuracy. In addition to the quality control tests performed during each colony visit, Precinorm $U$ control sera were used, as specified by the manufacturer, to test the reliability of the REFLOTRON approximately once a month.

The procedures recommended by the manufacturer for procuring and testing blood samples for cholesterol were performed on each consenting participant. The procedures that were followed included:

1. an AUTOCLIX lancet was used to obtain the sample,
2. 30 micro-liters of blood were drawn into a micropipette,
3. the blood was placed on the dry chemical reagent strip,
4. the strip was placed in the machine, and
5. the reading was recorded in mmol/L on a coding sheet next to each participant's unique study identification number.
6. Glucose. Glucose testing was performed by a trained Research Assistant or by the Principal Investigator. The accuracy of the GLUCOMETER II was checked prior to each colony visit with a manufacturer supplied check paddle, and once a month with DEXTRO-CHEK control material. The procedures recommended by the manufacturer for procurement and measurement of the blood samples were followed.

Briefly, the steps for using the GLUCOMETER II involved:

1. an AUTOCLIX lancet was used to obtain the sample,
2. the start button was depressed and the sample was placed on the reagent strip at the sound of the tone,
3. at the sound of the next tone, the reagent strip was dry blotted with a tissue,
4. the reagent strip was placed in the machine, and
5. the reading was recorded in mmol/L on a coding sheet next to each participant's unique ID number
6. Blood Pressure. Blood pressure was measured by a trained Research Assistant or by the Principal Investigator. The Principal Investigator trained the Research Assistant in the techniques to be followed and was able to determine that their inter-rater reliability was within 2 mm Hg . Standard mercury sphygmomanometers and stethoscopes were used. Cuff sizes, as recommended by the Canadian Coalition for the Prevention and Control of High Blood Pressure (1987), were used.

Participants were seated for a minimum of 5 minutes before their pressures were recorded from their left arm. The first and fifth Korotkoff sound were recorded on two successive readings taken 30 seconds to 1 minute apart. Where no fifth sound was distinguishable, the fourth (muffling) sound was recorded. The systolic and diastolic measurements from both readings were recorded next to the participants unique study identification number.
4. BMI. The participant's weight was measured fully clothed but with shoes removed using a SECA medical scale. One kilogram was deducted from each participant's weight to compensate for clothing weight. Height was measured with
the participant's shoes removed using a tape measure attached to a convenient wall. A level was used for accurately determining the top of the participant's head. In rare cases where a participant was unable to stand due to infirmity, an estimated height and weight was recorded. The BMI for each participant was calculated by dividing their weight in kilograms by their height in meters squared.

## D. Validation of Self-reported Conditions

The consent form requested the participants' permission to contact their physician in order to confirm reported medical histories of heart disease, pharmacologic treatment for hypertension, stroke, diabetes and/or hyperlipidemia. A Medical Release of Information Form was signed by participant's who agreed to have their medical diagnoses confirmed by their physician.

Consenting participants supplied the researcher with the name and place of practice of their physician. The physician's addresses were taken from the Alberta Medical Directory. Letters were sent to the participants' physicians outlining the purpose of the study and requesting their cooperation in confirming their patients' diagnoses.

Physicians who agreed to validate their patient's diagnoses received a second letter with a list of their patient's names and reported diagnoses . Physicians indicated on a form whether they confirmed or did not confirm their patient's self-reported diagnoses.
E. Informant Information

Information regarding a) age, b) sex, and c) history of heart disease, hypertension, diabetes, stroke and/or hyperlipidemia was gathered for Hutterites unable to personally meet with researchers from informants. Informants interviewed were either the spouse of the subject or the elders of the colony.
VI. Data Management

All of the physical measurements were recorded on a summary sheet and entered directly into the computer using the sPSS Data Entry system. Information from the PHS was entered using the same data entry program. Because each participant had been assigned a unique study number, it was possible to link these two files together for data analysis. All data was verified after initial entry using the SPSS data entry verification procedure.

It was necessary to develop special coding schemes for some of the data contained in the PHS. Because of the wide variety of medications prescribed for health problems, the ICD-9-CM Volume 1 (Commission on Professional and Hospital Activities,1980a) was used to code the purpose of the medication (i.e. the disease the medication was prescribed for), not the medication itself. However, in some cases this scheme was not practical (i.e. vitamins prescribed for. no particular disorder). In such cases, the ICD-9-CM Volume 2 Table of Drugs and Chemicals (Commission on Professional
and Hospital Activities, 1980b) was used to classify the medication for its therapeutic use.

Medical conditions and special medical procedures (EG. surgery.) were coded according to the ICD-9-CM Volume 3 (Commission on Professional and Hospital Activities, 1980c) classifications. While these coding schemes were useful for most of the participants, occasionally the self-reported conditions or procedures were difficult to code. In such cases a special code number was assigned to serve as a nonstandard ICD classification (EG. 888.88 was used to designate a surgery of unknown type).

All forms used to gather or summarize data were arranged by colony and stored in a secure office space and locked in a filing cabinet. Computer disks were, likewise, stored in a locked filing cabinet, when not in use. VII. Data Analysis

Data was analyzed using the BMDP (Dixon et al., 1985) and the SPSS $^{\mathrm{X}}$ (SPSS Inc., 1986) software statistical packages on the NOS-VE mainframe system at the University of Calgary. Both descriptive and analytic analyses were performed.

## A. Descriptive Analysis

Descriptive analysis was performed on all variables in order to describe their 1) frequencies and frequency distributions and, where appropriate, 2) their means, standard deviations, standard errors of the mean, and confidence intervals.

The prevalence of CHD, CVA, hypertension, diabetes, and hyperlipidemia was calculated by dividing the occurrence of the disorder by the total number of persons at risk for the disorder (Hennekens \& Buring, 1987, p.57). Both overall and age/sex-specific prevalence was calculated.

After examining a number of possible age-groupings, it was decided to use the following age categories for the presentation of the study results: 30-44, 45-59 and 60-74. This age-categorization scheme provided equal intervals with sufficient numbers of respondents in each age category to permit statistical comparisons by age and sex for the major variables of interest. The lower age limit (30 years) was chosen, in part, because of limited funds available for the project and the need to limit enrollment and, in part, because finding any CHD cases before this age was very unlikely. The upper age limit ( 74 years) was chosen, in part, because it reflected the upper range of age limits used in many of the studies of CHD prevalence reviewed in the literature and, in part, because of the small number of subjects available in the over-74 age category.

Because of the close relationship between the definition of disorder's occurrence and prevalence, disorders were analyzed using multiple definitions to assess the effect on the resulting prevalence. Table 2-1 lists the prevalence definitions for hypertension, hypercholesterolemia, diabetes and CHD.

TABLE 2-1
SPECIFIC OPERATIONAL DEFINITIONS USED TO DEFINE THE PREVALENCE OF HYPERTENSION, HYPERCHOLESTEROLEMIA, DIABETES AND CHD

| Disorder | Definition |
| :---: | :---: |
| Hypertension: | 1) Self-reported history of treatment for hypertension <br> 2) Diastolic Pressure $>90 \mathrm{~mm} \mathrm{Hg}$ and $>100 \mathrm{~mm} \mathrm{Hg}$ <br> 3) Self-reported history of treatment for hypertension and/or diastolic pressure $>99 \mathrm{~mm} \mathrm{Hg}$ |
| Hypercholesterolemia: | 1) Self-reported history of treatment for hypercholesterolemia <br> 2) Blood cholesterol $>6.20 \mathrm{mmol} / \mathrm{l}$ <br> 3) Self-reported history of treatment for hypercholesterolemia and/or blood cholesterol > $6.20 \mathrm{mmol} / \mathrm{l}$ |
| Diabetes: <br> (Hyperglycemia) | 1) Self-reported history of treatment for diabetes <br> 2) Two hour post-prandial blood glucose > $11.19 \mathrm{mmol} / 1$ <br> 3) Self-reported history of treatment for diabetes and/or blood glucose> $11.19 \mathrm{mmol} / \mathrm{l}$ |
| Coronary Heart Disease: | 1) Self-reported history of treatment for myocardial infarction, angina, coronary artery bypass graft, and/or angioplasty <br> 2) Rose Angina <br> 3) Self-reported history of treatment for CHD and/or Rose Angina |

The prevalence of CHD risk factors that are not medical disorders were also calculated. Based on the recommendations of Health and Welfare for BMI described above, the prevalence of a BMI for each category was calculated by age and sex. The prevalences of smoking, alcohol consumption and sedentary lifestyle were also calculated.

The age and sex-specific denominators for calculating prevalence are based on the age and sex-specific census performed in each colony. The surrogate data was analyzed separately from the respondent data.

## B. Analytic Analyses

An alpha of 0.05 was used as the criteria for significance in all statistical analyses. Both univariate and multivariate statistical techniques were used to examine the relationships between the independent and the dependent variables.

1. Univariate techniques. The relationship between categorical variables were examined using the Chi-square Test with the Yates correction applied. The Chi-square Test is ideally suited for analyzing categorical data that meet the following criteria: 1) there must be an adequate sample size and distribution of the data (a minimum of 5 expected occurrences per cell), 2) the measurements must be independent of one another and 3) there must be some theoretical basis for the categorization scheme (Munro, Visintainer, \& Page, 1986, p.127).

All of the categorical variables of interest were stratified by age and sex when using the Chi-square Test. Because of the increased likelihood of a Type I error when multiple comparisons are performed, the Bonferroniadjustment was made to the calculated $p$-value in the following ways (Mathews and Farewell, 1985, p. 169). First, the p-value for each age stratum (the age by sex comparisons) was adjusted (multiplied) by a factor of 3 to account for the three distinct age categories. The overall age category of 30-74 was not adjusted as it was treated as the age category of primary interest. Second, the p-value. for the two sexes (the sex by age comparisons) was adjusted by a factor of 2 . All p-values listed in the tables reflect these adjustments. The convention used in the tables, when tests of significance using the Chi-square Test could not be performed due to small expected cell frequencies, was to simply not list the results.

Logistic regression was also used to evaluate the univariate relationship between CHD and the risk factors of interest. Variables found to be significantly related to CHD in the univariate analysis were then entered into the multivariate analysis in order to adjust for any confounding effects that may have been present.

Comparisons of variables with continuous values (EG. blood cholesterol) between categories of discrete variables (EG. sex) were made using a number of univariate techniques
including t-tests, 95\% confidence intervals and logistic regression.
2. Multivariate techniques. Logistic regression was used to adjust for any confounding between CHD and the independent variables (Kleinbaum, Kupper and Morgenstern, 1982, p. 423). Where the relationship between independent variables and CHD was possibly confounded by other factors, logistic regression was used. The coding of the categorical variables used in the multivariate analyses for CHD are found in Table 2-2. A detailed discussion of the operational definitions used in the multivariate analysis for hypertension, diabetes and hypercholesterolemia may be found in Chapter 5. The analysis of the factors associated with CHD was done in three ways, each using a different operational definition of CHD. CHD was defined as 1) a history of myocardial infarction, angina, coronary bypass surgery and/or angioplasty, 2) Rose Angina, and 3) either Rose Angina or a history of myocardial infarction, angina, coronary bypass surgery and/or angioplasty. Because of the survey design used for the study, the adjusted odds ratios calculated using logistic regression reflect the coexistence of CHD and the variables of interest; they are not predictive of future events (Kelsey, Thompson and Evans, 1986).

TABLE 2-2
CODING SCHEME FOR MULTIVARIATE ANALYSES FOUND IN CHAPTER 5

| Variable | coding |
| :---: | :---: |
| Hypertension, Diabetes, Hyperlipidemia | $\begin{aligned} & 0=\text { no, } I=y e s \quad \text { (this scheme was used } \\ & \text { for each of the ways in } \\ & \text { which a case was } \\ & \text { defined) } \end{aligned}$ |
| History of CHD | $0=$ no, $1=y e s \quad$ (this scheme was used for each of the ways in which a case was defined) |
| Alcohol <br> Intake/Week | $\begin{aligned} & 0=\text { none, } 1=\text { one drink, } 2=2-3 \text { drinks, } \\ & 3=4-6 \text { drinks, } 4=7-13 \text { drinks, } \\ & 5=14+\text { drinks } \end{aligned}$ |
| Exercise | $0=$ no regular exercise, <br> $1=$ regular exercise |
| Family History | $0=$ no, $1=y e s$ (this scheme used for all combinations of relatives and age of onset) |
| Obesity | $0=$ no, $1=y e s$ |
| History of Tobacco Use | $0=$ no, $1=y e s$ |

## VIII. Ethics

Ethical approval for the design of the study was granted by the Conjoint Medical Ethics Committee of the Faculty of Medicine, University of Calgary. Because of the unique social structure of the Hutterite Brethren, it was necessary to receive consent from both the colony elders and the individual participant. Consent was first sought from the colony's minister and elders to include the colony in the study. The methods for informing the colony elders have been reviewed above.

After receiving a verbal presentation about the purpose and procedures involved in the study from the Principal Investigator, participants were asked to sign a consent form. The consent form described the purpose of the study, detailed the tests to be done, assured confidentiality and anonymity, and assured the participants that they could withdraw from, or refuse to participate in, the study without any prejudice on the part of the researchers.

A second form, the Release of Medical Information Form, gave a) the participants the opportunity to give their consent to have a copy of their physical measures sent to their physician, and b) the researchers permission to contact their physician to have their self-reported medical histories verified.

CHAPTER 3 : DESCRIPTION OF THE SAMPLE, NON-MODIFIABLE RISK<br>FACTORS AND LIFESTYLE-RELATED CHD RISK FACTORS

The purpose of this chapter is to describe the study population, paying particular attention to the prevalence of non-modifiable and modifiable CHD risk factors.
I. Participation in the Survey

The number of persons actually seen during the colony visits was 811; these individuals will subsequently be referred to as 'respondents'. Based on colony-supplied census data, $88.7 \%$ of all potential respondents were enrolled in the study as respondents. Because of concerns about biases that may have resulted from missing potential subjects, information was also gathered on all Hutterites aged 30-74 who were not present at the time of the colony visit ( $\mathrm{n}=103$ ). Those absent will be referred to as 'surrogates'. The sources of surrogate information, the informants, were colony elders and family members.

The informants were asked for the age, sex, and presence or absence of medical treatment for hypertension, diabetes, hypercholesterolemia, stroke, or CHD (defined as having had angina, myocardial infarction, coronary bypass surgery or coronary angioplasty) of the surrogates.

There were no significant differences found between surrogates and respondents in the age by sex distributions
or with respect to any of the disease conditions. The mean age of the surrogates was 3.0 years less than that of the respondents ( 43.2 vs. 46.2 years, respectively, $\mathrm{p}<0.01$ ).

Despite our efforts to limit data collection to those 30-74 years of age, four individuals under the age of 30 and 32 individuals over the age of 74 , completed the questionnaires. These individuals were not included in the data analysis. Two additional individuals from colonies not participating in the study were also excluded from the analysis.
II. Non-modifiable CHD Risk Factors Among Respondents .

## A. Age and Sex

The age- and sex-specific distributions of the study population are detailed in Table 3-1. There were no significant differences found between the males and the females in either the age distribution or the mean age. B. Family History of CHD

Table 3-2 summarizes the prevalence of respondents reporting that at least one of their first, second, or third degree relatives (parent, sibling, grandparent, aunt or uncle) had a history of CHD, irrespective of its age of onset. Hereafter, the term "relative" will refer to a first, second, or third degree relative. Over two-thirds of the respondents reported having at least one relative with a history of CHD. Table 3-3 details the prevalence of CHD in a relative before the age of 55 .

Approximately a third of the respondents (30.9\% of

TABLE 3-1
AGE DISTRIBUTION AND MEAN AGES OF RESPONDENTS BY SEX

| Age Group | Female |  | Male |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\begin{gathered} n \\ (N=421) \end{gathered}$ | \% | $\underset{(N=390)}{n}$ | \% |
| 30-44 | 222 | 52.7 | 214 | 54.9 |
| 45-59 | 133 | 31.6 | 116 | 29.7 |
| 60-74 | 66 | 15.7 | 60 | 15.4 |
| Test of Significance in Difference in Age Distribution Between Males and Females: |  |  |  |  |
| Mean Age:$47.4$$t=0.08, d f=841, p=0.936$ |  |  |  |  |

TABLE 3-2
PREVAIENCE OF RESPONDENTS HAVING A RELATIVE WITH HEART DISEASE* BY AGE AND SEX

| Age <br> Group | Male |  | Sex Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $n / N$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | $p$ |
| 30-44 | 140/211 | 66.4 | 151/219 | 68.9 | 0.22 | 1 | 1.000 |
| 45-59 | 85/114 | 74.6 | 95/133 | 71.4 | 0.17 | 1 | 1.000 |
| 60-74 | 40/60 | 66.7 | 40/66 | 60.6 | 0.27 | 1 | 1.000 |
| 30-74 | 265/385 | 68.8 | 286/418 | 68.4 | 0.00 | 1 | 0.961 |

TABLE 3-3
PREVALENCE OF RESPONDENTS HAVING A
RELATIVE WITH HEART DISEASE*, ONSET BEFORE AGE 55, BY AGE AND SEX

| Age Group | Male |  | Sex <br> Female |  | ```Test of }\mp@subsup{x}{}{2 Significance Between Sexes``` |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | n/N | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | $d f$ | p |
| 30-44 | 63/211 | 29.9 | 88/219 | 40.2 | 4.58 | 1 | 0.100 |
| 45-59 | 42/114 | 36.8 | 46/133 | 34.6 | 0.06 | 1 | 1.000 |
| 60-74 | 14/60 | 23.3 | 21/66 | 31.8 | 0.74 | 1 | 1.000 |
| 30-74 | 119/385 | 30.9 | 155/418 | 37.1 | 3.13 | 1 | 0.077 |

Test of $x^{2}$ Significance by age Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 3.60 | 2.04 |
| df | 2 | 2 |
| p | 0.330 | $<0.720$ |

* in parents, siblings, aunts or uncles, or grandparents
males and $37.1 \%$ of females) have a relative with a history of CHD before the age of 55 years. Regardless of the age of onset, there were no differences found either between age groups or the sexes in the prevalence of having a relative with CHD.

Because CHD in a parent with onset before 55 years of age is considered to be highly predictive of CHD, Table 3-4 provides information about the prevalence of a "premature" parental history of $C H D$ in the study population. Slightly more females than males (11.3\% vs $8.5 \%$ ) had a parent with premature onset of CHD, however this difference was not significant in the overall population or in any of the agespecific categories.

The prevalence of having a parent with CHD onset after the age of 55 years was approximately three times greater than premature onset for both the males ( $30.3 \%$ vs 11.3\%) and the females (25.5\% vs 8.5\%) (Table 3-5). Again however, there was no difference in the prevalence of a parental history of CHD after age 55 years by either age or sex.
III. Modifiable Lifestyle-related CHD Risk Factors

## A. Alcohol Consumption

1. Present use. As shown in Table $3-6,90.5 \%$ of males and $55.0 \%$ of females reported consuming an alcoholic beverage at least once a month during the previous twelve months. Males, in all age groups, reported regular consumption of alcohol significantly more often than

TABLE 3-4
PREVALENCE OF RESPONDENTS HAVING A PARENT WITH HEART DISEASE ONSET BEFORE AGE 55 BY AGE AND SEX

| Age Group | Male |  | Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | p |
| 30-44 | 25/212 | 11.8 | 32/219 | 14.6 | 0.52 | 1 | 1.000 |
| 45-59 | 7/115 | 6.1 | 11/132 | 8.3 | 0.19 | 1 | 1.000 |
| 60-74 | 1/59 | 1.7 | 4/65 | 6.2 | 0.65 | 1 | 1.000 |
| 30-74 | 33/386 | 8.5 | 47/416 | 11.3 | 1.39 | 1 | 0.238 |

TABLE 3-5
PREVALENCE OF RESPONDENTS HAVING A PARENT WITH HEART DISEASE ONSET AFTER AGE 55 BY AGE AND SEX

| Age Group | Male |  | Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | $p$ |
| 30-44 | 50/212 | 23.6 | 44/219 | 20.1 | 0.58 | 1 | 1.000 |
| 45-59 | 45/115 | 39.1 | 43/132 | 32.6 | 0.88 | 1 | 1.000 |
| 60-74 | 22/59 | 37.3 | 17/65 | 26.2 | 1.29 | 1 | 0.762 |
| 30-74 | 117/386 | 30.3 | 104/416 | 25.0 | 2.57 | 1 | 0.110 |

TABLE 3-6
RESPONDENT ALCOHOL CONSUMPTION* BY AGE AND SEX

| Age Group | Male |  | Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | $p$ |
| 30-44 | 198/214 | 92.5 | 135/220 | 61.4 | 57.3 | 1 | $<0.001$ |
| 45-59 | 100/116 | 86.2 | 63/133 | 47.4 | 39.6 | 1 | $<0.001$ |
| 60-74 | 55/60 | 91.7 | $32 / 65$ | 49.2 | 24.6 | 1 | $<0.001$ |
| 30-74 | 353/390 | 90.5 | 230/418 | 55.0 | 124.7 | 1 | $<0.001$ |

Test of $\mathrm{x}^{2}$ Significance Between Ages Within Each Sex Group

| Male | Female |
| :---: | :---: |
| 3.6 | 7.6 |
| 2 | 2 |
| 0.338 | 0.044 |

* during past year had an alcoholic beverage more than once a month
females. There was a significant decrease in alcohol consumption with aging in females, but not in males.

2. Type of product. As seen in Table 3-7, men reported drinking both wine and spirits significantly more often than women, although there was no difference in beer consumption. There was a significant difference in the type of beverage consumed by both males and females, with both sexes preferring in order: beer, wine and spirits.
3. Frequency of use. There was a significant difference between males and females in the frequency of alcohol consumption, with men drinking more frequently than women (Table 3-8). Of those who reported that they drank alcohol at least once a month, daily consumption of at least one alcoholic beverage was reported by $50.4 \%$ of the males and $13.4 \%$ of the females. Most of the women ( $69.1 \%$ ) who consumed alcoholic beverages did so once a week or less. B. Use of Tobacco Products
4. Past use. No women in the study, and just $14.6 \%$ ( $n=57$ ) of men reported ever having been regular smokers. There was a significant difference in past smoking by age, with men in the 30-44 age group reporting the greatest past use (Table 3-9). ,
5. Type of product. Of those men who reported having ever been regular users of tobacco, $91.2 \%$ smoked cigarettes and $12.3 \%$ used smokeless tobacco products (chewing tobacco

TABLE 3-7
OF RESPONDENTS AGED WHO CONSUMED ALCOHOL IN LAST YEAR, PREVALENCE OF DRINKING BEER, WINE AND SPIRITS BY SEX

| Beverage | Male |  | Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\underset{(\mathrm{N}=353}{\mathrm{n}}$ | \% | $\underset{(N=230)}{n}$ | \% | $\mathrm{x}^{2}$ | df | p |
| Beer | 260 | 73.7 | 158 | 67.8 | 2.1 | 1 | 0.151 |
| Wine | 222 | 62.9 | 114 | 48.9 | 10.6 | 1 | 0.001 |
| Spirits | 138 | 39.1 | 51 | 21.9 | 18.2 |  | $<0.001$ |

Test of $x^{2}$ Significance Within Each Sex Group for the Type of Beverage Consumed

|  | Male | Female |
| :--- | :--- | :---: |
| $\mathrm{x}^{2}$ | 90.9 | 99.9 |
| df | 2 | 2 |
| p | $<0.001$ | $<0.001$ |

TABLE 3-8
OF RESPONDENTS WHO CONSUMED ALCOHOL IN PAST YEAR, . FREQUENCY OF DRINKING BY SEX

| Usual Number of Drinks Consumed | Males |  | Females |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\underset{(N=35}{n}$ | \% | $\underset{(N-2}{n}$ | \% |
| $2+/ D A Y$ | 46 | 13.0 | 1 | 0.4 |
| 1/DAY | 132 | 37.4 | 30 | 13.0 |
| 4-6/WEEK | 28 | 7.9 | 7 | 3.0 |
| 2-3/WEEK | 67 | 19.0 | 33 | 14.3 |
| 1/WEEK OR LESS | 80 | 22.7 | 159 | 69.1 |

TABLE 3-9
DISTRIBUTION OF MALE RESPONDENT SMOKING* BY AGE

| Age | $\mathrm{n} / \mathrm{N}$ | \% |
| :---: | :---: | :---: |
| Group |  |  |
| 30-44 | $42 / 214$ | 19.6 |
| 45-59 | 9/116 | 4.2 |
| 60-74 | $6 / 60$ | 10.0 |
| 30-74 | 57/390 | 14.6 |
| Between Age Test of Significance: |  |  |
| $\mathrm{x}^{2}=$ | $=2, \mathrm{p}$ | 0.008 |

* those reporting that they have ever been regular smokers
and snuff). No men reported regular use of either cigars or pipes.

3. Present use. Of the 57 men who had regularly used tobacco at some time in the past, $75.5 \%$ ( $n=43$ ) have quit, $8.8 \%(n=5)$ continue to regularly use it, and $15.8 \%$ ( $n=9$ ) still occasionally use tobacco products. Thus, of all Hutterite males, only $3.6 \%$ (14/390) report even occasional use of tobacco at the present time.
4. Quantity used. Of the 57 males who had ever smoked, only $3.6 \%$ smoked a package of cigarettes or more per day, and $72.7 \%$ smoked under 5 cigarettes per day.
5. Duration of use. Of those who ever regularly used tobacco products, $39.3 \%$ did so for at least 10 years. The most prevalent duration of tobacco use in the group of regular smokers was 5-9 years (47.4\%).

## C. Dietary Intake of Selected Foods

The study participants were asked to report how often they ate a variety of selected foods. These foods may be roughly divided into two categories: 1) those with higher fat content (Table 3-10) and 2) those with lower fat content (Table 3-11). This diet survey was not rigorous enough to permit calculation of the fat content in the Hutterite diet. Rather, it was intended to provide a crude estimate of how frequently participants ate foods higher or lower in fat content.

Of the high fat foods, dairy products were reported to be eaten most frequently, though all types of high fat foods

TABLE 3-10
FREQUENCY OF INTAKE OF SELECTED HIGH FAT FOODS BY SEX

| Food Type | Frequency |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\begin{gathered} 1+\text { Times } \\ \text { a Day } \end{gathered}$ |  | 4-6 Times a Week |  | 2-3 Times a Week |  | $<1$ Time <br> a Week |  | Never |  |
|  | n | \% | n | \% | n | \% | n | \% | n | \% |
| Red Meat |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=364$ ) | 107 | 29.4 | 50 | 13.7 | 132 | 36.3 | 71 | 19.5 | 4 | 1.1 |
| F ( $\mathrm{N}=399$ ) | 77 | 19.3 | 35 | 8.8 | 157 | 39.3 | 128 | 32.1 | 2 | 0.5 |
| Margarine |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=328$ ) | 190 | 57.9 | 28 | 8.5 | 24 | 7.3 | 29 | 8.8 | 57 | 17.4 |
| F ( $\mathrm{N}=376$ ) | 227 | 60.4 | 31 | 8.2 | 19 | 5.1 | 36 | 9.6 | 63 | 16.8 |
| Butter |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=291$ ) | 151 | 51.9 | 23 | 7.9 | 13 | 4.5 | 23 | 7.9 | 81 | 27.8 |
| F ( $\mathrm{N}=316$ ) | 167 | 52.8 | 18 | 5.7 | 11 | 3.5 | 10 | 3.2 | 110 | 34.8 |
| Eggs |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=365$ ) | 130 | 35.6 | 61 | 16.7 | 128 | 35.1 | 42 | 11.5 | 4 | 1.1 |
| F ( $\mathrm{N}=399$ ) | 149 | 37.3 | 44 | 11.0 | 145 | 36.3 | 49 | 12.3 | 12 | 3.0 |
| Whole Milk |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=355$ ) | 272 | 76.6 | 29 | 8.2 | 22 | 6.2 | 17 | 4.8 | 15 | 4.2 |
| F ( $\mathrm{N}=386$ ) | 287 | 74.4 | 25 | 6.5 | 18 | 4.7 | 15 | 3.9 | 41 | 10.6 |
| Dairy |  |  |  |  |  |  |  |  |  |  |
| Products* |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=342$ ) | . 168 | 49.1 | 43 | 12.6 | 71 | 20.6 | 33 | 9.7 | 28 | 8.0 |
| F ( $\mathrm{N}=373$ ) | 189 | 50.7 | 31 | 8.2 | 77 | 20.7 | 32 | 8.6 | 44 | 11.8 |
| Cheese |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=358$ ) | 119 | 33.2 | 59 | 16.5 | 119 | 33.2 | 51 | 14.2 | 10 | 2.8 |
| F ( $\mathrm{N}=390$ ) | 153 | 39.2 | 35 | 9.0 | 135 | 34.6 | 54 | 13.8 | 13 | 3.3 |
| Fried Foods |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=356$ ) | 109 | 30.6 | 81 | 22.8 | 110 | 30.9 | 49 | 13.8 | 7 | 2.0 |
| $F \quad(\mathrm{~N}=40 \mathrm{I})$ | 107 | 26.7 | 70 | 17.5 | 160 | 39.9 | 54 | 13.5 | 10 | 2.5 |
| Gravy |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=343$ ) | 24 | 7.0 | 30 | 8.7 | 98 | 28.6 | 148 | 43.1 | 43 | 12.5 |
| F ( $\mathrm{N}=353$ ) | 30 | 8.5 | 12 | 3.4 | 64 | 18.1 | 142 | 40.2 | 105 | 29.7 |

[^0]
## TABLE 3-11

FREQUENCY OF INTAKE OF SELECTED LOW FAT FOODS BY SEX

| Food Type | Frequency |  |  |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | ```1 + Times 4-6 Times a Day a Week``` |  |  |  | 2-3 Times a Week |  | $\begin{gathered} <\text { I Time } \\ \text { a Week } \end{gathered}$ |  | Never |  |
|  | n | \% | n | \% | n | \% | n | \% | n | \% |
| Fish |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=333$ ) | 11 | 3.3 | 4 | 1.2 | 15 | 4.5 | 276 | 82.9 | 27 | 8.1 |
| F ( $\mathrm{N}=364$ ) | 16 | 4.4 | 4 | 1.1 | 15 | 4.1 | 301 | 82.7 | 28 | 7.7 |
| Chicken |  |  |  |  |  |  |  |  |  |  |
| $\mathrm{M}(\mathrm{N}=358)$ | 11 | 3.1 | 24 | 6.7 | 197 | 55.0 | 125 | 34.9 | 1 | 0.3 |
| F ( $\mathrm{N}=410$ ) | 14 | 3.4 | 15 | 3.7 | 27.1 | 66.1 | 110 | 26.8 | 0 | 0.0 |
| Bread |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=365$ ) | 298 | 81.6 | 44 | 12.1 | 13 | 3.6 | 7 | 1.9 | 3 | 0.8 |
| F ( $\mathrm{N}=410$ ) | 353 | 86.1 | 29 | 7.1 | 16 | 3.9 | 8 | 2.0 | 4 | 1.0 |
| Vegetables |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=367$ ) | 269 | 73.3 | 69 | 18.8 | 20 | 5.4 | 8 | 2.2 | 1 | 0.3 |
| F ( $\mathrm{N}=415$ ) | 335 | 80.7 | 63 | 15.2 | 14 | 3.4 | 2 | 0.5 | 1 | 0.2 |
| Fruit |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=361$ ) | 171 | 47.4 | 59 | 16.3 | 83 | 23.0 | 46 | 12.7 | 2 | 0.6 |
| F $\quad(\mathrm{N}=403)$ | 250 | 62.0 | 40 | 9.9 | 78 | 19.4 | 35 | 8.7 | 0 | 0.0 |
| Cereals |  |  |  |  |  |  |  |  |  |  |
| M ( $\mathrm{N}=346$ ) | 44 | 12.7 | 15 | 4.3 | 85 | 24.6 | 178 | 51.4 | 24 | 6.9 |
| F ( $\mathrm{N}=391$ ) | 70 | 17.9 | 11 | 2.8 | 134 | 34.3 | 163 | 41.7 | 13 | 3.3 |

are clearly part of the regular Hutterite diet. Of the lower fat foods, fruits, vegetables, chicken and bread are regularly eaten, though fish and cereal are infrequently consumed.

In order to better understand any differences in dietary habits between the males and the females, the frequency data was dichotomized into 'once a week or less' and 'more than once a week'. Using this approach to data analysis, the males reported consuming significantly more red meat, milk and gravy, but less chicken, vegetables and cereal, than females.

## D. Use of Salt at the Table

Table 3-12 summarizes the reported use of salt at the table by males and females in the study. Regular salt use at the table, as defined by adding salt to prepared food 'always' or 'usually', was reported by $28.2 \%$ of men and $12.6 \%$ of women. Occasional salt use was reported by $30.3 \%$ of male and $29.5 \%$ of female respondents. Negligible salt use, as defined by adding salt 'never' or 'hardly ever', was reported by $41.5 \%$ in males and $57.8 \%$ by females. There was a significant difference in the use of salt at the table between men and women in both the 30-44 age group ( $\mathrm{p}=$ 0.006 ) and in the 45-59 age group ( $p=0.004$ ), with men reporting greater salt use than women. There was also a significant age differential found in salt use, with salt use declining as age increases for both males ( $p=0.017$ ), and females ( $\mathrm{p}<0.001$ ).

TABLE 3-12
RESPONDENTS SALT USE AT THE TABLE BY SEX

| SALT USE | MALES |  | FEMALES |  |
| ---: | ---: | ---: | ---: | ---: |
|  | $n$ <br> $(N=390)$ | $n$ <br> $(N=420)$ | $\%$ |  |
| ALWAYS | 56 | 14.4 | 24 | 5.7 |
| USUALLY | 54 | 13.8 | 29 | 6.9 |
| SOMETIMES | 118 | 30.3 | 124 | 29.5 |
| HARDLY EVER | 75 | 19.2 | 103 | 24.5 |
| NEVER | 87 | 22.3 | 140 | 33.3 |
| $x^{2}=36.2, \mathrm{df}=4, \mathrm{p}<0.001$ |  |  |  |  |

## E. Physical Exertion

1. Exercise at work. Work was classified by whether it usually 1) was sedentary, 2) required walking but little lifting, 3) required light lifting and some climbing, or 3) required heavy lifting or a great deal of exertion. Men reported significantly higher levels of work-related exercise than women (Table 3-13). The amount of work-related exercise significantly declined with age for both males and females
2. Leisure-time activity. As seen in Table 3-14, $34.0 \%$ of males and $32.3 \%$ of females reported that they engaged in leisure-time exercise at least once per week. There were no significant differences between the sexes in reported leisure-time exercise for any of the age groups. For both sexes there was a significant difference in leisure-time exercise between the age groups. More exercise was reported by males in the 45-59 age group than in the $30-$ 44. or 60-74 age groups, whereas females in the 30-44 and 4559 age groups exercised more than those in the 60-74 age group.
3. Strenuousness of exercise. Only $11.8 \%$ of males and $13.0 \%$ of females who regularly exercise usually exert themselves enough to cause either sweating or heavy breathing. There were no significant differences either by sex or age in the strenuousness of leisure-time exercise.
4. Duration of exercise. Of the respondents who did regularly exercise, $40.5 \%$ of males and $61.5 \%$ of females

TABLE 3-13
WORK-RELATED EXERCISE OF RESPONDENTS BY SEX

| Work <br> Activity | Male |  | Female |  |
| :--- | ---: | ---: | ---: | ---: |
|  | $n$ <br> $(N=390)$ | $n$ <br> $(N=419)$ |  |  |
|  | 18 | 4.6 | 18 | 4.3 |
| Sedentary | 168 | 43.1 | 232 | 55.4 |
| Walk | 106 | 27.2 | 118 | 28.2 |
| Climb/Lift | 98 | 25.1 | 51 | 12.2 |
| Heavy Work |  |  |  |  |

Between sex Test of Significance:
$x^{2}=24.7, d f=3, p<0.001$

Test of $x^{2}$ Significance by Age for Sexes:
Males: $\mathrm{x}^{2}=60.5, \mathrm{df}=6, \mathrm{p}<0.001$ )
Females: $x^{2}=46.6$, df $=6, p<0.001$ ).

TABLE 3-14

## RESPONDENTS ENGAGING IN REGULAR LEISURE-TIME EXERCISE* BY AGE AND SEX

| Age Group | Male |  | Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | p |
| 30-44 | 59/213 | 27.7 | 77/218 | 35.3 | 2.6 |  | 0.329 |
| 45-59 | 52/115 | 45.2 | 46/131 | 35.1 | 2.2 |  | 0.413 |
| 60-74 | 21/60 | 35.0 | 11/66 | 16.7 | 4.6 |  | 0.093 |
| 30-74 | 132/388 | 34.0 | 134/415 | 32.3 | 0.2 | 1 | 0.656 |

Test of $x^{2}$ Significance Between Ages Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 10.2 | 8.8 |
| df | 2 | 2 |
| p | 0.012 | 0.025 |

* exercised at least once a week during past month
reported that their usual exercise lasted for less than 15 minutes per day. There was an overall significant sex differential in the duration of exercise, with men reporting that they exercised for longer periods than women (Table 315).

5. Type of exercise. Walking, usually from home to work or to and from the communal dining hall, was the most frequently reported form of exercise. A handful of respondents reported riding exercise bicycles or using other types of aerobic apparatus (i.e. trampoline, rowing machine).
6. Relationship between work-related and leisurerelated exercise. There was no relationship found between leisure and work-related exercise for either the males or the females, e.g. respondents at any level of work-related exercise were just as likely to exercise in their free time as not.

## F. Self-reported Levels of Stress

Respondents were asked to rate their usual level of stress along a continuum from relaxed to extremely tense. As seen in Table 3-16, there was no significant difference in self-reported stress between males and females in any age group. There was no significant relationship between level of stress and age in either males or females.

TABLE 3-15
DURATION OF EXERCISE OF RESPONDENTS WHO ENGAGE IN LEISURE-TIME EXERCISE BY SEX

| DURATION | MALE |  | FEMALES |  |
| :---: | :---: | :---: | :---: | :---: |
|  | $\begin{gathered} n \\ (N=126) \end{gathered}$ | \% | $\begin{gathered} n \\ N=130) \end{gathered}$ | \% |
| < 15 MINUTES | 51 | 40.5 | 80 | 61.5 |
| 15-30 MINUTES | 46 | 36.5 | 35 | 26.9 |
| 31-60 MINUTES | 19 | 15.1 | 6 | 4.6 |
| > 60 MINUTES | 10 | 7.9 | 9 | 6.9 |

Between Sex Test of Significance:
$x^{2}=14.7, d f=3, p=0.002$

```
TABLE 3-16
RESPONDENTS' SELF-ASSESSMENT OF USUAL LEVEL OF STRESS BY AGE AND SEX
```

| Age Group SelfAssessment | Male |  | Female |  | Test of $x^{2}$ <br> Significance <br> Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | $d f$ | $p$ |
| 30-44 |  |  |  |  | 5.2 | 4 | $0.804^{\text {A }}$ |
| Relaxed | 27/209 | 12.9 | 18/221 | 8.1 |  |  |  |
| Calm | 89/209 | 42.6 | 83/221 | 37.6 |  |  |  |
| Anxious | 30/209 | 14.4 | 41/221 | 18.6 |  |  |  |
| Tense | 60/209 | 28.7 | 75/221 | 33.9 |  |  |  |
| Extremely Tense | 3/209 | 1.4 | 4/221 | 1.8 |  |  |  |
| 45-59 |  |  |  |  | 3.6 | 4 | 1.000. |
| Relaxed | 12/113 | 10.6 | 9/132 | 6.8 |  |  |  |
| Calm | 42/113 | 37.2 | 48/132 | 36.4 |  |  |  |
| Anxious | 23/113 | 20.4 | 24/132 | 18.2 |  |  |  |
| Tense | 32/113 | 28.3 | 49/132 | 37.1 |  |  |  |
| Extremely Tense | 4/113 | 3.5 | 2/132 | 1.5 |  |  |  |
| 60-74 |  |  |  |  | 0.9 | 4 | $1.000^{\text {A }}$ |
| Relaxed | 5/55 | 9.1 | 8/61 | 13.1 |  |  |  |
| Calm | 26/55 | 47.3 | 30/61 | 49.2 |  |  |  |
| Anxious | 11/55 | 20.0 | 9/61 | 14.8 |  |  |  |
| Tense | 10/55 | 18.2 | 11/61 | 18.0 |  |  |  |
| Extremely Tense | 3/55 | 5.5 | 3/61 | 4.9 |  |  |  |
| 30-74 |  |  |  |  | 4.7 | 4 | 0.316 |
| Relaxed | 44/377 | 11.1 | 35/414 | 8.5 |  |  |  |
| Calm | 157/377 | 41.6 | 161/414 | 38.9 |  |  |  |
| Anxious | 64/377 | 17.0 | 74/414 | 17.9 |  |  |  |
| Tense | 102/377 | 27.1 | 135/414 | 32.6 |  |  |  |
| Extremely Tense | 10/377 | 2.7 | 9/414 | 2.2 |  |  |  |

## CHAPTER 4: HEALTH CONDITIONS RELATED TO CHD

The purpose of this chapter is to describe the prevalence of a number of health-related conditions that are associated with an increased risk for CHD. The conditions discussed will include: A) hypertension, B) diabetes, C) hypercholesterolemia, D) elevated BMI, and E) stroke. Where appropriate, prevalence will be discussed in relation to physical measurements and medical treatments. This chapter will also discuss the physician verification process.

## I. Hypertension

## A. Prevalence of Treatment for Hypertension

1. Distribution by Age and Sex. Table 4-1 summarizes the prevalence of hypertension treatment at any time in the past or at present for the study participants. Approximately one-fifth of all respondents reported that they had been under treatment at some time for hypertension. There were no significant differences in self-reported hypertension prevalence between the males and females in any of the age groups. However, for both sexes there was a significant increase in the prevalence of hypertension with aging.
2. Treatment modalities for hypertension. Table 4-2 describes the use of multiple treatment modalities for respondents with hypertension. Of those under treatment for hypertension, significantly more anti-hypertensive

TABLE 4-1
PREVALENCE OF RESPONDENTS REPORTING HAVING EVER BEEN TREATED FOR HYPERTENSION

| Age Group | Male |  | Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | $d f$ | p |
| 30-44 | 25/214 | 11.7 | 13/222 | 5.9 | 3.9 | 1 | 0.141 |
| 45-59 | 38/116 | 32.8 | 38/133 | 28.6 | 0.3 | 1 | 1.000 |
| 60-74 | 24/60 | 40.0 | 37/66 | 56.1 | 2.6 | 1 | 0.314 |
| 30-74 | 87/390 | 22.3 | 88/421 | 20.9 | 0.2 | 1 | 0.689 |

Test of $x^{2}$ Significance Between Ages Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 32.1 | 84.5 |
| df | $<0.001$ | $<0.001$ |
| p | $<0.001$ |  |

TABLE 4-2
USE OF TREATMENT MODALITIES FOR HYPERTENSION AT THE PRESENT TIME IN RESPONDENTS BY SEX

| Treatment Type | $\begin{array}{r} \text { Males } \\ (\mathrm{N}=87) \\ \mathrm{n} \quad \% \end{array}$ |  | $\begin{aligned} & \text { Females } \\ & (N=88) \end{aligned}$ |  | Between Sex Test of Significance $x^{2}$ df $p$ |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Lose Weight Alone | 1 | 1.1 | NR |  |  |  |  |
| Low Na Diet Alone | 4 | 4.6 | 2 | 2.3 |  |  |  |
| Medications Alone | 22 | 25.3 | 25 | 28.4 | 0.09 | 1 | 1.000 |
| Lose Weight Plus Low Na Diet | 2 | 2.3 | 1 | 1.1 |  |  |  |
| Lose Weight Plus Medications | 4 | 4.6 | 12 | 13.6 | 3.28 | 1 | 0.349 |
| Low Na Diet Plus Medications | 11 | 12.6 | 14 | 15.9 | 0.16 | 1 | 1.000 |
| Lose Weight Plus Low Na Diet Plus Medications | 22 | 25.3 | 26 | 29.5 | 0.21 | 1 | 1.000 |
| No Treatment | 21 | 24.1 | 8 | 9.1 | 6.12 | 1 | 0.067 |
| Weight Ioss in Total | 29 | 33.3 | 39 | 44.3 | 1.78 | 1 | 0.182 |
| Low Na Diet in Total | 39 | 44.3 | 43 | 48.9 | . 15 | 1 | 0.701 |
| Medications in Total | 59 | 67.8 | 79 | 89.8 | 11.37 | 1 | $<.001$ |

medication, either alone, or in combination with other forms of therapy, was reported by the females ( $n=79,89.8 \%$ ) than by the males ( $n=59,67.8 \%$ ). There were no significant differences between males and females for any of the other treatment modality combinations. In the males, there was identical use (25.3\%) of medication therapy alone, or medication therapy in combination with both sodium restriction and weight loss. Females were also more likely to report being treated either by medications alone (28.4\%) or a combination of medications and weight lose and sodium restriction (29.5\%). Nearly a quarter (24.1\%) of the males, but only 9.1\% of the females, who had been under treatment for hypertension at some time in the past, reported that they were under no treatment at present.

The finding that significantly more females than males with known hypertension were treated with anti-hypertensive medications ( $89.8 \%$ vs $67.8 \%$ ) suggests that either females are more vigorously treated, or they are more compliant with their treatment regimens than the males.
3. Physician Validation of Self-reported Histories.

The validity of the self-reported treatment for hypertension is strongly supported by physician validation. Of the 136 respondents reporting that. they were taking antihypertensive medications, physician validation was ultimately received for 107 respondents. Of these 107 respondents, 104 were verified as actually under care for hypertension, yielding a positive verification rate of $97.2 \%$
for hypertension. The details of the physician validation process is described in Section VI later in this Chapter.
4. Health Care Utilization and Hypertension. Of all prescribed medications taken by respondents, antihypertensive medications were the most prevalent pharmacologic agents reported, with 59 males (15.1\%) and 79 females (18.8\%) taking anti-hypertensive drugs. Hypertension treatment was also the most frequently cited reason for making a visit to the respondents' personal physician during the previous twelve months, with 67 males (17.2\%) and 73 females (17.5\%) reporting such a visit.
B. Diastolic Blood Pressure

1. Distribution by Age and Sex. The distribution of diastolic blood pressure by age and sex is presented in Table 4-3 and a more detailed histogram of diastolic pressure may be found in Appendix II. Because of the small number of respondents with a diastolic blood pressure above 114 mm Hg , this category was combined with the $100-114 \mathrm{~mm} \mathrm{Hg}$ category for the statistical comparisons of age and sex categories.

Of all respondents, $57.4 \%$ of the males ( $n=217$ ) and 29.5\% ( $n=121$ ) of the females had a mean diastolic pressure above $90 \mathrm{~mm} \mathrm{Hg} ; 26 \%(n=98)$ of the males and $12.2 \%(n=50)$ of the females had a diastolic pressure above 99 mm Hg . The differences between the sexes in the proportion of respondents above both the 90 mm Hg and the 99 mm Hg cutpoints were significant, i.e. more males in upper ranges
of diastolic pressure. In all age groups there were fewer males than females with diastolic pressures under 90 mm Hg ;
TABLE 4-3
DISTRIBUTION OF MEAN DIASTOLIC BLOOD PRESSURE (mm Hg )
BY AGE AND SEX

| $\begin{gathered} \text { Age Group } \\ \text { MDBP } \\ \text { (mm Hg) } \end{gathered}$ | Male |  | - Female |  | Between Sex Test of Significance |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | $p$ |
| 30-44 |  |  |  |  |  |  |  |
| < 90 | 108/206 | 52.4 | 185/217 | 85.3 | 53.58 | 2 | < 0.001* |
| 90-99 | 58/206 | 28.2 | 20/217 | 9.2 |  |  |  |
| 100-114 | 40/206 | 19.4 | 12/217 | 5.5 |  |  |  |
| $115+$ | NR |  | NR |  |  |  |  |
| 45-59 |  |  |  |  |  |  |  |
| < 90 | 36/113 | 31.9 | 72/129 | 55.8 | 42.52 | 2 | < 0.001* |
| 90-99 | 38/113 | 33.6 | 32/129 | 24.8 |  |  |  |
| 100-114 | 37/113 | 32.7 | 24/129 | 18.6 |  |  |  |
| $115+$ | 2/113 | 1.8 | 1/129 | 0.8 |  |  |  |
| 60-74 |  |  |  |  |  |  |  |
| < 90 | 17/59 | 28.8 | 32/64 | 50.0 | 5.90 | 2 | 0.156* |
| 90-99 | 23/59 | 39.0 | 19/64 | 29.7 |  |  |  |
| 100-114 | 17/59 | 28.8 | 12/64 | 18.8 |  |  |  |
| $115+$ | 2/59 | 3.4 | 1/64 | 1.6 |  |  |  |
| 30-74 |  |  |  |  |  |  |  |
| $<90$ | 161/378 | 42.6 | 289/410 | 70.5 | 62.91 | 2 | < 0.001* |
| 90-99 | 119/378 | 31.5 | 71/410 | 17.3 |  |  |  |
| 100-114 | 94/378 | 24.9 | 48/410 | 11.7 |  |  |  |
| $115+$ | 4/378 | 1.1 | 2/410 | 0.5 |  |  |  |

Test of $x^{2}$ Significance Between Ages* Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 17.9 | 47.6 |
| df | 4 | 4 |
| p | 0.003 | $<0.001$ |

*The 115 + category was collapsed into the 100-114 category
these differences were significant in the 30-44 and 45-59 age groups. There was a significant shift in the distributions to higher diastolic pressures for both the males and the females with aging.

The mean diastolic pressure for males was 91.5 mm Hg and for females it was 84.1 mm Hg (Table 4-4). As can be seen from the $95 \%$ confidence intervals for male diastolic pressures, the 30-44 age group had a significantly lower mean diastolic pressure than the $45-59$ age group. For the females, the $95 \%$ confidence intervals reveals that the 30-44 age group had a significantly lower mean diastolic pressure than either the 45-59 or 60-74 age groups. As evidenced by their non-overlapping 95\% confidence intervals, males had significantly higher diastolic pressures than females in both the 30-44 and 45-59 age groups. Mean diastolic pressures in the 60-74 age group were not significantly different between the sexes.
2. Treatment and control of hypertension. Based on diastolic blood pressure measurements, both the males and the females who were under a physicians care for hypertension were relatively well controlled. Nevertheless, as will be discussed below, a large number of Hutterites with evidence of hypertension are not under treatment.

More females than males with an elevated diastolic pressure were under a physicians care for hypertension. of the 87 males under treatment for hypertension $67 \%$ ( $n=59$ ) had a diastolic pressure under 90 mm Hg ; for the 88 females the
corresponding control rate was 71.6\% (n=63). Of the 119 males with a diastolic pressure $90-99 \mathrm{~mm} \mathrm{Hg}, 79.8 \% ~(n=95)$ were under no treatment for hypertension; the corresponding rate for women was $53.2 \%$ (42/71). Of the 98 males with a diastolic pressure $>99 \mathrm{~mm} \mathrm{Hg}, 52.0 \% ~(\mathrm{n}=51)$ were not under a physicians care for hypertension; the corresponding rate for women was $32.0 \%(16 / 50)$. C. Prevalence of Hypertension When Defined as "Under Medical

Treatment or Having a Diastolic Pressure $>99 \mathrm{~mm} \mathrm{Hg}{ }^{\prime \prime}$ When the definition of hypertension is broadened to include all of those under medical treatment for hypertension and/or having a diastolic pressure above 99 mm Hg , the prevalence in males is $39.2 \%$ ( $\mathrm{n}=153$ ), and in females is $24.9 \%$ ( $n=105$ ) (Table 4-5). Using this definition of hypertension, males have significantly more hypertension than females in both the 45-59 and the 60-74 age categories and there was also a significant increase in the prevalence of hypertension for both sexes with aging. It should be noted that the expanded definition of hypertension nearly doubles the prevalence of hypertension for the males when compared to those only under treatment (39.2\% vs $22.3 \%$ ), but has a much smaller effect for the females (24.9\% vs 20.9\%). D. Systolic Pressure

1. Distribution by age and sex. The distribution of systolic blood pressure by age and sex is presented in Table 4-6 and a more detailed histogram of systolic pressure is contained in Appendix III. Because of the small number of

TABLE 4-4
MEAN DIASTOLIC BLOOD PRESSURE ( mm Hg ) BY AGE AND SEX

| $\begin{aligned} & \text { Age } \\ & \text { Group } \end{aligned}$ | Males |  |  |  | Females |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | n | Mean | S.E. | 95\% CI | n | Mean | S.E. | 95\% CI |
| 30-44 | 206 | 89.32 | 0.76 | 87.82-90.82 | 217 | 80.10 | 0.69 | 78.74-81.46 |
| 45-59 | 113 | 94.28 | 1.11 | 92.09-96.47 | 129 | 87.80 | 1.04 | 85.74-89.86 |
| 60-74 | 59 | 93.79 | 1.48 | 90.82-96.76 | 64 | 90.34 | 1.39 | 87.57-93.11 |
| 30-74 | 378 | 91.50 | 0.58 | 90.34-92.66 | 410 | 84.12 | 0.58 | 82.99-85.26 |

TABLE 4-5
PREVALENCE OF HYPERTENSION AS DEFINED BY TREATMENT FOR HYPERTENSION AND/OR HAVING A DIASTOLIC PRESSURE > 99 mm Hg

| Age Group | Male |  | Sex Female |  | Test of $\mathrm{x}^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | n/N | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | p |
| 30-44 | 58/214 | 27.1 | 19/222 | 8.6 | 24.5 | 1 | $<.001$ |
| 45-59 | 61/116 | 52.6 | 48/133 | 36.1 | 6.2 | 1 | . 039 |
| 60-74 | 34/60 | 56.7 | 38/66 | 57.6 | . 0 | 1 | 1.000 |
| 30-74 | 153/390 | 39.2 | 105/421 | 24.9 | . 18.41 | 1 | <. 001 |

Test of $x^{2}$ Significance Between Ages Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $x^{2}$ | 29.5 | 78.2 |
| df | 2 | $2^{2}$ |
| p | $<0.001$ | $\cdot<0.001$ |

TABLE 4-6
DISTRIBUTION OF MEAN SYSTOLIC BLOOD PRESSURE (mm Hg ) BY AGE AND SEX

| Age Group MSBP | Male |  | Female |  | Between Sex Test of Significance |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | $\%$ | $\mathrm{n} / \mathrm{N}$ | \% | $x^{2}$ | df | p |
| 30-44 |  |  |  |  |  |  |  |
| < 160 | 198/206 | 96.1 | 213/217 | 98.2 | 0.94 | 1 | < $0.996 *$ |
| 160-189 | 8/206 | 4.9 | 4/217 | 1.8 |  |  |  |
| $190+$ | NR |  | NR |  |  |  |  |
| 45-59 |  |  |  |  |  |  |  |
| $<160$ | 97/113 | 85.8 | 112/129 | 86.8 | 0.04 | 1 | $<1.000 *$ |
| 160-189 | 14/113 | 12.4 | 15/129 | 11.6 |  |  |  |
| $190+$ | 2/113 | 1.8 | 2/129 | 1.6 |  |  |  |
| 60-74 |  |  |  |  |  |  |  |
| < 160 | 34/59 | 57.6 | 47/64 | 73.4 | 2.75 | 1 | 0.293* |
| 160-189 | 21/59 | 35.6 | 15/64 | 23.4 |  |  |  |
| $190+$ | 4/59 | 6.8 | 2/64 | 3.1 |  |  |  |
| 30-74 |  |  |  |  |  |  |  |
| < 160 | 329/378 | 87.0 | 372/410 | 90.7 | 2.37 | 1 | 0.124 |
| 160-189 | - $43 / 378$ | 11.4 | 34/410 | 8.3 |  |  |  |
| $190+$ | 6/378 | 1.6 | 4/410 | 1.0 |  |  |  |

Test of $x^{2}$ Significance Between Ages* Within Each Sex Group

|  | Male | Female |
| :---: | :---: | :---: |
| $\mathrm{x}^{2}$ | 50.1 | 34.3 |
| df | 2 | 2 |
| p | $<0.001$ | $<0.001$ |
| *The 190 | y coll | o the 1 |

respondents with a systolic pressure above 189 mm Hg , this category was added to the $160-189 \mathrm{~mm} \mathrm{Hg}$ category for the statistical comparisons of age and sex categories.

The prevalence in the study population of systolic pressure greater than 160 mm Hg was $13.0 \%$ for the males and $9.3 \%$ for the females. There was no significant difference in the distribution of systolic pressure between the males and the females in any age category. There was, however, a significant increase with aging in the proportion of both males and females who had a systolic pressure above 160 mm Hg .

As evidenced by the non-overlapping. 95\% confidence intervals in Table 4-7, there was a significant increase in the mean systolic blood pressure with aging for both sexes. While males had significantly higher mean systolic pressures than females in the 30-44 age group, there were no differences in either the 45-59 or 60-74 age categories.
2. Isolated systolic hypertension. As hypertension was not defined in terms of systolic pressure, the prevalence of isolated systolic pressure (elevated systolic pressure in the absence of elevated diastolic pressure) was examined to see if this decision led to missing a significant number of possible hypertension cases. There was not one case of a systolic pressure above 189 mm Hg in respondents who had a diastolic pressure below 90 mm Hg . The number of cases of systolic pressure $160-189 \mathrm{~mm} \mathrm{Hg}$ in respondents with a diastolic pressure $<90 \mathrm{~mm} \mathrm{Hg}$ was also

TABLE 4-7
MEAN SYSTOLIC BLOOD PRESSURE (mm Hg) BY AGE AND SEX

| Age |  |  |  | Males |  | Fem | males |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group |  | $n \quad$ Mean | S.E. | . 95\% CI |  | n Mean | S S.E. | . $95 \% \mathrm{CI}$ |
| 30-44 | 206 | 132.07 | 1.00 | 130.09-134.05 | 217 | 120.13 | 0.971 | 118.21-122.05 |
| 45-59 | 113 | 139.20 | 1.84 | 135.55-142.84 | 129 | 134.84 | 1.911 | 131.06-138.62 |
| 60-74 | 59 | 154.61 | 3.041 | 148.52-160.70 |  | 147.97 | 2.531 | 142.92-153.01 |
| 30-74 | 378 | 137.72 | . 991 | 135.76-139.67 | 410 | 129.10 | 1.021 | 127.10-131.11 |

TABLE 4-8
PREVALENCE OF RESPONDENTS REPORTING HAVING EVER BEEN TREATED FOR DIABETES MELLITUS

| Age Group | Male |  | Sex <br> Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | n/N | $\%$ | $\mathrm{x}^{2}$ | $d f$ | p |
| 30-44 | 7/214 | 3.3 | 9/222 | 4.1 | 0.3 | 1 | 1.000 |
| 45-59 | 9/116 | 7.8 | 13/133 | 9.8 | 0.1 | 1 | 1.000 |
| 60-74 | 12/60 | 20.0 | 10/66 | 15.2 | 0.2 | 1 | 1.000 |
| 30-74 | 28/390 | 7.2 | 32/421 | 7.6 | 0.01 | 1 | 0.924 |

Test $x^{2}$ Significance Between Ages Within Each Sex Group

|  | Male | Female |
| :---: | :---: | :---: |
| $\mathrm{x}^{2}$ |  |  |
| df | 19.8 | 10.2 |
| p | $<0.001$ | 2 |
|  |  | 0.012 |

small; 5 females and 2 males were found. Of these, 3 of the females and 1 of the males were in the highest age category. Thus, only 3 respondents under the age of 60 years were found to have isolated systolic hypertension. This small number of cases supports the use of diastolic pressure as the criterion for hypertensive classification.
II. Diabetes Mellitus

## A. Prevalence of Treatment for Diabetes

1. Distribution by age and sex. Table 4-8 summarizes the prevalence of treatment for diabetes at any time in the past, or at present, in the study population. In the overall study population, $7.2 \%$ of the males $(n=28)$ and $7.6 \%$ of the females ( $n=32$ ) were under a physician's care for diabetes. There were no significant differences in the prevalence of diabetes between the men and the women for any of the age groups. However, there was a significant increase in the prevalence of diabetes with aging for both sexes.

While it was not possible to distinguish between Type I (Insulin-Dependent) and Type II (Non-Insulin-Dependent) diabetes, diabetes with onset after 30 years of age is primarily Type II (American Diabetes Association, 1989). Of the 28 males with a history of diabetes treatment, $17.9 \%$ ( $n=5$ ) reported that their disease onset was before thirty years of age; $31.3 \%$ of the females (10/32) reported diabetes onset before thirty years of age. While a higher proportion of females had diabetic onset before age 30 , it was not statistically different from the males ( $p=.37$ ).
2. Treatment modalities for diabetes. Of those under a phýsician's care for diabetes, $57.1 \%$ of the males ( $n=16$ ), and $43.8 \%$ of the females ( $n=14$ ), were taking daily insulin injections at the time of the survey. The distribution of multiple modalities of therapy for diabetes is described in Table 4-9. There were no significant differences between the males and females in their use of any of the treatment modalities. The most frequent form of therapy reported by both the male and the female diabetics was the combination of diet and insulin (males: 46.4\%, $\mathrm{n}=13$; females: 31.3\%, $n=10$ ). Six of the known diabetics, or $10.0 \%$ of the diabetic population, were still monitored by their physicians but were under no form of therapy (diet or medications) at the time of the survey. The four females who were no longer under active treatment for diabetes had all been diagnosed with gestational diabetes; it was not clear why the two males were no longer receiving therapy.
3. Physician Validation of Diabetes Treatment. The self-reported histories of diabetes were well supported by physician verification. Of the 54 diabetics who reported that they were under either pharmacologic or dietary treatment for diabetes, 36 physician verifications were ultimately received. Of these 36 verifications, 33 supported the respondents self-reported history, for a positive verification rate of $91.7 \%$
4. Diabetes and Health Care Utilization. Of all prescribed medications taken by the study population,

| TABLE 4-9 <br> USE OF TREATMENT MODALITIES FOR DIABETES AT THE PRESENT TIME IN RESPONDENTS BY SEX |  |  |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Treatment Type | n | 28) | Fern | \%) | Test Betwe $x^{2}$ |  | nificance $p$ |
| Diet Alone | 5 | 17.9 | 8 | 25.0 | 0.13 | 1 | 1.000 |
| Insulin Alone | 3 | 10.7 | 4 | 12.5 |  |  |  |
| Oral Agents Alone | 1 | 3.6 | 2 | 6.3 |  |  |  |
| Diet plus Insulin | 13 | 46.4 | 10 | 31.3 | 0.88 | 1 | 0.794 |
| Diet Plus oral Agents | 4 | 14.3 | 4 | 12.5 |  |  |  |
| No Treatment | 2 | 7.1 | 4 | 12.5 |  |  |  |
| Diet in Total | 22 | 78.6 | 22 | 68.8 | 0.32 | 1 | 0.572 |
| Oral Agents <br> in Total | 5 | 17.9 | 6 | 18.8 | 0.06 | 1 | 0.806 |
| Insulin in Total | 16 | 57.1 | 14 | 43.8 | 0.60 | 1 | 0.438 |

diabetic agents (insulin and oral hypoglycemics) were the second most prevalent for the males ( $n=21,5.4 \%$ ) and the sixth most prevalent for the females ( $\mathrm{n}=20,4.0 \%$ ). Diabetes was the second leading cause of hospitalizations for a medical condition for both the males and the females; with 14 males ( $3.6 \%$ ) and 9 females (2.1\%) having been hospitalized for diabetes, at some time, in their life. Diabetes was the third most frequent reason given for making a visit to a physician's office during the previous twelve months for both the males and the females; with 21 of the males ( $5.4 \%$ ) and 23 of the females ( $5.5 \%$ ) having made such a visit.

## B. Post-prandial Glucose

I. Distribution by age and sex. The distribution of the two hour post-prandial glucose by age and sex is presented in Table 4-10 and a more detailed histogram of glucose may be found in Appendix IV. The prevalence of a post-prandial glucose $11.20 \mathrm{mmol} / \mathrm{l}$ or greater was $2.7 \%$ for males and 4.3\% for females. Of the 20 individuals with an elevated glucose, 15 (75.0\%) were known diabetics. Due to the small number of respondents with a glucose above 11.20 mmol/l, the only statistical test performed on the age and sex distribution was the comparison of glucose between the sexes in the 30-74 age category. There was no difference between the males and the females in the distribution of glucose.

TABLE 4-10

| Age Group Glucose | Male |  | Female |  | Between Sex Test of Significance |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $x^{2}$ | df | p |
| 30-44 |  |  |  |  |  |  |  |
| $<11.20$ | 137/139 | 98.6 | 150/155 | 96.8 3.2 |  |  |  |
| $11.20+$ | 2/139 | 1.4 | 5/155 |  |  |  |  |
| 45-59 |  |  |  |  |  |  |  |
| < 11.20 | 74/77 | 96.1 | 96/100 | 96.0 |  |  |  |
| $11.20+$ | 3/77 | 3.9 | 4/100 | 4.0 |  |  |  |
| 60-74 |  |  |  |  |  |  |  |
| $<11.20$ | 38/40 | 95.0 | 43/47 | 91.5 |  |  |  |
| $11.20+$ | 2/40 | 5.0 | 4/47 | 8.5 |  |  |  |
| 30-74 |  |  |  |  | 0.59 | 1 | 0.444 |
| $<11.20$ | 249/256 | 97.3 | 289/302 | 95.7 4.3 | 0.59 | 1 | 0.444 |
| 11.20 + | 7/256 | 2.7 | 13/302 | 4.3 |  |  |  |

TABLE 4-11
MEAN POST-PRANDIAL GLUCOSE (mmol/l) BY AGE AND SEX

| Age group | Males |  |  |  |  | Fen Mean | Females | 95\% CI |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 30-44 | 139 | 5.71 | 0.14 | 5.44-5.98 | 155 | 5.84 | 0.16 | 5.52-6.16 |
| 45-59 | 77 | 5.75 | 0.23 | 5.29-6.21 | 100 | 6.31 | 0.23 | 5.86-6.76 |
| 60-74 | 40 | 6.61 | 0.38 | 5.83-7.39 | 47 | 6.91 | 0.48 | 5.95-7.87 |
| 30-74 | 256 | 5.86 | 0.12 | 5.63-6.10 | 302 | 6.16 | 0.14 | 5.90-6.43 |

Table 4-11 details the means, standard errors and 95\% confidence intervals of the two hour post-prandial glucose by age and sex. As evidenced by the overlapping 95\% confidence intervals, no significant differences in glucose were noted either by age for each sex, or between the sexes in any age strata.
2. Control of diabetes. Based on the post-prandial glucose measurements, male diabetics were found to have better diabetic control than the females. Of the 28 males under a physician's care for diabetes, 85.7\% ( $\mathrm{n}=24$ ) had a post-prandial glucose under $11.20 \mathrm{mmol} / 1$; the corresponding rate for the 32 diabetic females was $65.6 \%$ ( $n=21$ ). Thus, the rate of non-control for diabetic females was more than twice that of the males ( $34.4 \%$ vs $14.3 \%$ ). Only five respondents who were not previously diagnosed with diabetes were found to have a blood glucose > $11.19 \mathrm{mmol} / \mathrm{l}$. C. Prevalence of Diabetes When the Definition is Expanded to Include Both Those Under Treatment and Those with a Blood Glucose > $11.19 \mathrm{mmol} / \mathrm{l}$

When diabetes is more broadly defined to include both those under treatment and/or those with a blood glucose > $11.19 \mathrm{mmol} / \mathrm{l}$ there is only a small increase in prevalence over and above those just under treatment (Table 4-12). For the males the prevalence increases from 7.2\% to 7.9\% (n=31), and for the females the increase is from $7.6 \%$ to $8.3 \%$ ( $n=35$ ). Just as was found in the more limited definition of diabetes (treatment alone), there are no differences between

TABLE 4-12
PREVALENCE OF DIABETES AS DEFINED BY DIABETIC TREATMENT AND/OR BLOOD GLUCOSE > $11.19 \mathrm{mmol} / \mathrm{L}$

| Age Group | Male |  | x Female |  | ```Test of x Significance Between Sexes``` |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | n/N | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | $d f$ | $p$ |
| 30-44 | 7/214 | 3.3 | 11/222 | 5.0 | 0.4 | 1 | 1.000 |
| 45-59 | 12/116 | 10.3 | 14/133 | 10.5 | 0.1 | 1 | 1.000 |
| 60-74 | 12/60 | 20.0 | 10/66 | 15.2 | 0.2 | 1 | 1.000 |
| 30-74 | $31 / 390$ | 7.9 | 35/421 | 8.3 | 0.01 | 1 | 0.951 |

Test $\mathrm{x}^{2}$ Significance Between Ages Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 19.2 | 8.19 |
| df | 2 | 2 |
| p | $<0.001$ | 0.039 |

the sexes at any age in diabetes prevalence, however, there is a significant increase for both sexes with aging.

## III. Hypercholesterolemia

## A. Prevalence of Treatment for Hypercholesterolemia

1. Distribution by age and sex. The prevalence of treatment for hypercholesterolemia in the study population at present, or at sometime in the past, is presented in Table 4-13. Physician prescribed treatment (diet and/or medications) for hypercholesterolemia was reported by $5.4 \%$ of the males and by $6.2 \%$ of the females. There were no differences found for hypercholesterolemia treatment between males and females in any of the age groups examined. There was a significant increase in reported hypercholesterolemia with aging for the males, but not for the females.
2. Treatment modalities for hypercholesterolemia. As seen in Table. 4-14, dietary treatment alone was used for hypercholesterolemia by $52.4 \%$ of the males ( $n=11$ ) and $76.9 \%$ of the females ( $n=20$ ). Of those with hypercholesterolemia, none of the females, and $19.0 \%$ of the males ( $n=4$ ) were on pharmacologic therapy. Approximately a quarter of the respondents ( $28.6 \%$ of males, $23.1 \%$ of females) who reported hypercholesterolemia were under no treatment.

## 3. Physician validation of treatment for

hypercholesterolemia. Physician verification of the selfreported treatment for hypercholesterolemia supports the reliability of the respondent's data. Of the 35 respondents who indicated that they were under treatment for

TABLE 4-13
PREVALENCE OF RESPONDENTS REPORTING HAVING EVER BEEN TREATED FOR HYPERCHOLESTEROLEMIA

| Age Group | Male |  | X Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | p |
| 30-44 | 7/214 | 3.3 | 8/222 | 3.6 | 0.0 | 1 | 1.000 |
| 45-59 | 14/116 | 12.1 | 12/133 | 9.0 | 0.3 | 1 | 1.000 |
| 60-74 | 0/60 |  | 6/66 | 9.1 |  |  |  |
| 30-74 | 21/390 | 5.4 | 26/421 | 6.2 | 0.1 | 1 | 0.740 |

Test of $x^{2}$ Significance by Age Within Each Sex Group

|  | Male | Female |
| :---: | :---: | :---: |
| $\mathrm{x}^{2}$ | 15.5 | 5.4 |
| df | 2 | 2 |
| p | $<0.001$ | 0.137 |

TABLE 4-14
USE OF TREATMENT MODALITIES FOR HYPERCHOLESTEROLEMIA AT THE PRESENT TIME IN RESPONDENTS BY SEX

| Modality Type | $\begin{gathered} \text { Males } \\ (\mathrm{N}=21) \end{gathered}$ |  | $\begin{aligned} & \text { Females } \\ & (N=26) \end{aligned}$ |  | Test of Significance Between Modalities |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | n | \% | n | \% | $\mathrm{x}^{2}$ | df | p |
| Diet Alone | 11 | 52.4 | 20 | 76.9 | 3.12 | 1 | 0.155 |
| Medications Alone | 2 | 9.5 | NR |  |  |  |  |
| Diet and Medication | 2 | 9.5 | NR |  |  |  |  |
| No Treatment | 6 | 28.6 | 6 | 23.1 | 0.01 | 1 | 1.000 |

hypercholesterolemia, 11 physician verifications were ultimately received. While this low return rate was disappointing, 10 of the 11 verifications supported the respondent's reported treatment, for a positive verification rate of $90.9 \%$.
4. Health care utilization and hypercholesterolemia. No hospitalizations or physician visits in the previous 12 months were ascribed specifically to hypercholesterolemia. Undoubtedly, those under pharmacologic treatment for hypercholesterolemia were seen by their physicians, but no visits were reported.

## B. Blood Cholesterol

1. Distribution by age and sex. The distribution of serum cholesterol by age and sex is presented in Table 4-15 and a more detailed histogram of cholesterol is contained in Appendix $V$. Almost half of the males (46.6\%) and females ( $42.1 \%$ ) had a blood cholesterol of $6.20 \mathrm{mmol} / \mathrm{l}$ or greater. There was a similar distribution of cholesterol for the males and the females in all but the 30-44 age category; in this age group significantly more females had cholesterol levels under $5.70 \mathrm{mmol} / \mathrm{l}$. There was a significant shift to higher levels of cholesterol with aging for the females, but not for the males.

Table 4-16 details the means, standard errors and 95\% confidence intervals of cholesterol by age and sex for the study population. The males had a mean cholesterol of 6.14 mol/l and the females mean cholesterol was $5.99 \mathrm{mmol} / \mathrm{l}$.

TABLE 4-15
DISTRIBUTION OF CHOLESTEROL (mmol/l) BY AGE AND SEX

| Age Group CHOL | Male |  | Female |  | Between Sex Test of Significance |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | 6 | $n / \mathrm{N}$ | q | $x^{2}$ | df | p |
| 30-44 |  |  |  |  |  |  |  |
| < 5.70 | 79/195 | 40.5 | 119/205 | 58.0 | 12.39 | 4 | 0.044 |
| 5.70-6.19 | 37/195 | 19.0 | 28/205 | 13.7 |  |  |  |
| 6.20-7.19 | 62/195 | 31.8 | 45/205 | 22.0 |  |  |  |
| 7.20-8.19 | 10/195 | 5.1 | 7/205 | 3.4 |  |  |  |
| $8.20+$ | 7/195 | 3.6 | 6/205 | 2.9 |  |  |  |
| 45-59 |  |  |  |  |  |  |  |
| $<5.70$ | 26/107 | 24.3 | 40/127 | 31.5 | 2.47 | 4 | 1.000 |
| 5.70-6.19 | 22/107 | 20.6 | 19/127 | 15.0 |  |  |  |
| 6.20-7.19 | 37/107 | 34.6 | 45/127 | 35.4 |  |  |  |
| 7.20-8.19 | 14/107 | 13.1 | 16/127 | 12.6 |  |  |  |
| $8.20+$ | 8/107 | 7.5 | 7/127 | 5.5 |  |  |  |
| 60-74 |  |  |  |  |  |  |  |
| < 5.70 | 19/56 | 33.9 | 11/60 | 18.3 | 3.99 | 4 | 1.000 |
| 5.70-6.19 | 8/56 | 14.3 | 10/60 | 16.7 |  |  |  |
| 6.20-7.19 | 17/56 | 30.4 | 23/60 | 38.3 |  |  |  |
| 7.20-8.19 | 8/56 | 14.3 | 9/60 | 15.0 |  |  |  |
| $8.20+$ | 4/56 | 7.1 | 7/60 | 11.7 |  |  |  |
| 30-74 |  |  |  |  |  |  |  |
| $<5.70$ | 124/358 | 34.6 | 170/392 | 43.4 | 6.54 | 4 | 0.162 |
| 5.70-6.19 | 67/358 | 18.7 | 57/392 | 14.5 |  |  |  |
| 6.20-7.19 | 116/358 | 32.4 | 113/392 | 28.8 |  |  |  |
| 7.20-8.19 | 32 ¢ 358 | 8.9 | 32/392 | 8.2 |  |  |  |
| $8.20+$ | 19/358 | 5.3 | 20/392 | 5.1 |  |  |  |

Test of $x^{2}$ Significance by Age Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 15.7 | 49.6 |
| df | 8 | 8 |
| p | 0.094 | $<0.001$ |

TABLE 4-16
MEAN CHOLESTEROL (mmol/l) BY AGE AND SEX

| Age |  |  | Males |  | Females |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Group | n | Mean | S.E. | 95\% CI | n | Mean | S.E. | 95\% CI |
| 30-44 | 195 | 5.95 | 0.08 | 5.79-6.10 | 205 | 5.60 | 0.08 | 5.43-5.76 |
| 45-59 | 107 | 6.47 | 0.12 | 6.23-6.72 | 127 | 6.28 | 0.10 | 6.09-6.48 |
| 60-74 | 56 | 6.18 | 0.18 | 5.82-6.54 | 60 | 6.71 | 0.15 | 6.40-7.02 |
| 30-74 | 358 | 6.14 | 0.06 | 6.01-6.27 | 392 | 5.99 | 0.06 | 5.87-6.11 |

For both sexes there was a significant difference in cholesterol between the age groups, though the patterns of difference were not the same.

As evidenced by non-overlapping $95 \%$ confidence intervals, males $30-44$ years old had significantly lower mean cholesterol levels than the 45-59 age group, but werenot significantly different from the 60-74 age group. In contrast, females aged 30-44 had significantly lower cholesterols than either the 45-59 or the 60-74 age groups. Only in the 30-44 age group was there a significant difference in mean cholesterol levels between males and females, with males having significantly higher values than females.
2. Control of hypercholesterolemia. Of the 21 males who reported that they had been diagnosed with hypercholestexolemia, $52.4 \%(n=11)$ had a cholesterol below $6.20 \mathrm{mmol} /$; the corresponding rate for the 26 females was. considerably lower, $23.1 \% ~(n=6)$.
C. The Prevalence of Hypercholesterolemia When it is

Defined as Those Under Treatment and/or Those with a

Blood Cholesterol $>6.19 \mathrm{mmol} / 1$

When the definition of hypercholesterolemia is expanded to include both those under treatment and those with a blood cholesterol above $6.19 \mathrm{mmol} / \mathrm{l}$, there is a substantial increase in hypercholesterolemia prevalence over and above the treatment-only definition (Table 4-17). Using the expanded definition, the prevalence of hypercholesterolemia

TABLE $\dot{4}-17$
PREVALENCE OF HYPERCHOLESTEROLEMIA AS DEFINED BY TREATMENT FOR HYPERCHOLESTEROLEMIA AND/OR BLOOD CHOLESTEROL > 6.19

| Age Group | Male |  | Fex Female |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $n / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | $p$ |
| 30-44 | 82/195 | 42.1 | 60/205 | 29.3 | 5.8 | 1 | . 045 |
| 45-59 | 66/107 | 61.7 | 71/127 | 55.9 | 0.2 | 1 | 1.000 |
| 60-74 | 28/56 | 50.0 | 40/60 | 66.7 | 1.9 | 1 | . 495 |
| 30-74 | 176/358 | 49.2 | 171/392 | 43.6 | 2.5 | 1 | 0.220 |

Test of $x^{2}$ Significance by Age Within Each Sex Group
Male Female
$x^{2}$
df
p
10.55

2
.012
36.92

2
$<.001$
is $49.2 \%$ for the males ( $\mathrm{n}=176$ ), and $43.6 \%$ ( $\mathrm{n}=171$ ) for the females. As might be expected by the small number of respondents under treatment for hypercholesterolemia, this prevalence rate is more closely related to the general distribution of cholesterol found in Table 4-15. While there were no differences in the prevalence of hypercholesterolemia between the sexes in any age category when using the more expanded definition, there was a significant increase in prevalence for both sexes with aging.

## IV. Elevated Body Mass Index

The distribution of Body Mass Index (BMI) by age and sex is presented in Table 4-18 and a more detailed histogram of BMI is contained in Appendix VI. As there were so few respondents with a BMI under $20 \mathrm{~kg} / \mathrm{m}^{2}$ except in the $30-44$ age group, this category was collapsed with the 20-24.9 group for the sex comparisons in the 45-59 and 60-74 age groups.

Over half of the respondents, $52.8 \%$ of males and $53.6 \%$ of females, had a BMI greater than $27 \mathrm{~kg} / \mathrm{m}^{2}$. There was a significant difference in the BMI distribution between the sexes in the 30-44 age group, with more females than males distributed in the lower BMI categories. While there was no significant difference in the BMI distribution with aging for the males, there was for the females.

The means, standard errors and $95 \%$ confidence intervals for BMI by age and sex are detailed in Table 4-19. The mean

TABLE 4-18
DISTRIBUTION OF BODY MASS INDEX $\left(\mathrm{kg} / \mathrm{m}^{2}\right)$ BY AGE AND SEX

| $\begin{aligned} & \text { Age Group } \\ & \cdot \mathrm{BMI} \\ & \left(\mathrm{~kg} / \mathrm{m}^{2}\right) \end{aligned}$ | Male |  | Female |  | Between Sex Test of Significance |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $n / N$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | $d f$ | p |
| 30-44 |  |  |  |  |  |  |  |
| $<20.0$ | 5/206 | 2.4 | 24/215 | 1.1 .2 | 15.24 | 3 | 0.005 |
| 20.0-24.9 | 63/206 | 30.6 | 74/215 | 34.4 |  |  |  |
| 25.0-26.9 | 40/206 | 19.4 | 38/215 | 17.7 |  |  |  |
| $27.0+$ | 98/206 | 47.6 | 79/215 | 36.7 |  |  |  |
| 45-59 |  |  |  |  |  |  |  |
| $<20.0$ | 4/112 | 3.6 | 4/129 | 3.1 | 4.79 | 2 | 0.274 * |
| 20.0-24.9 | 18/112 | 16.1 | 19/129 | 14.7 |  |  |  |
| 25.0-26.9 | 27/112 | 24.1 | 18/129 | 14.0 |  |  |  |
| $27.0+$ | $63 / 112$ | 56.3 | 88/129 | 68.2 |  |  |  |
| 60-74 |  |  |  |  |  |  |  |
| $<20.0$ | NR |  | 1/63 | 1.5 | 4.23 | 2 | $0.361 *$ |
| 20.0-24.9 | 12/59 | 20.3 | 6/63 | 9.5 |  |  |  |
| 25.0-26.9 | 9/59 | 15.3 | 5/63 | 7.9 |  |  |  |
| $27.0^{\circ}+$ | 38/59 | 64.4 | $51 / 63$ | 81.0 |  |  |  |
| 30-74 |  |  |  |  |  |  |  |
| < $<20.0$ | 9/377 | 2.4 | 29/407 | 7.1 | 12.09 | 3 | 0.007 |
| 20.0-24.9 | 93/377 | 24.7 | 99/407 | 24.3 |  |  |  |
| 25.0-26.9 | 76/377 | 20.2 | 61/407 | 15.0 |  |  |  |
| $27.0+$ | 199/377 | 52.8 | 218/407 | 53.6 |  |  |  |

Test of $x^{2}$ Significance by Age Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 11.2 | 58.7 |
| df | 4 | 4 |
| p | 0.051 | $<0.001$ |

* The < 20.0 category collapsed into the 20-24.99 category

TABLE 4-19
BODY MASS INDEX $\left(\mathrm{kg} / \mathrm{m}^{2}\right)$ BY AGE AND SEX

| Age Group | n | Males |  |  | Females |  |  | 95\% CI |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Mean | S.E. | 95\% CI | n | Mean | S.E. |  |
| 30-44 | 206 | 27.07 | 0.29 | 26.51-27.63 | 215 | 26.40 | 0.39 | 25.63-27.17 |
| 45-59 | 112 | 28.42 | 0.45 | 27.52-29.32 | 129 | 29.91 | 0.48 | 28.96-30.86 |
| 60-74 | 59 | 28.91 | 0.65 | 27.61-30.20 | 63 | 32.32 | 0.83 | 30.65-33.98 |
| 30-74 | 377 | 27.76 | 0.23 | 27.30-28.22 | 407 | 28.43 | 0.31 | 27.82-29.03 |

BMI for males was 27.8, for females it was 28.4. As can be seen from the $95 \%$ confidence intervals, there is no significant difference in BMI between any of the age groups for the males. In contrast, the females 30-44 have a significantly lower BMI than either the 45-59 or 60-74 age groups. While there is no significant difference between the sexes in BMI for the $30-44$ and $45-59$ age groups, thefemales have significantly higher BMIs than males in the 60-74 age group.

## V. Cerebrovascular Disease

There were a total of three self-reported strokes in the study population. The prevalence of stroke by age and sex is presented in Table 4-20. Only one of the three respondents with a stroke also had a history of CHD, and this history was verified by the respondent's physician. Because of the small number of respondents with stroke, no further analysis was performed for this health condition. However, in light of the relatively high prevalence of hypertension, it is noteworthy that the prevalence of stroke was so low in those aged 30-74 years.
VI. Summary of the Physician Validation Procedure There was an attempt made to receive physicianverification of every respondent-reported case of a cardiac disease of any type. In addition, verification was sought for all respondents who reported that they 1) were on medication for hypertension, 2) were under a physician's active care for diabetes or hypercholesterolemia and 3), had

Table 4-20
PREVALENCE OF RESPONDENTS HAVING EVER BEEN TREATED FOR A STROKE (CEREBROVASCULAR ACCIDENT)

| Age Group | Male |  | Female |  | Total |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% |
| 30-44 | 0/214 | 0.00 | 1/222 | 4.50 | 1/436 | 2.29 |
| 45-59 | 0/116 | 0.00 | $0 / 133$ | 0.00 | $0 / 249$ | 0.00 |
| 60-74 | 1/60 | 1.67 | 1/66 | 1.52 | 2/126 | 1.59 |
| 30-74 | 1/390 | . 26 | 2/421 | . 48 | 3/811 | .37 |

a history of stroke. There was a remarkable degree of cooperation on both the part of the respondents and their physicians in this verification process. The verification process may be broken down into five component parts:

1. the number of conditions of each type requiring verification
2. of these conditions, the proportion that the respondents had given permission to verify
3. of the conditions that respondents gave permission to verify, the proportion that the physicians agreed to verify
4. of the conditions that the physicians agreed to verify, the proportion of verifications that were ever received from the physicians
5. Of the physician verifications received, the proportion that agreed with the respondentreported histories

The results of this verification procedure are presented in Table 4-21.

Based on the participants' RELEASE OF MEDICAL INFORMATION FORMS, a total of 166 physicians were eligible to provide verification of their patients' self-reported history of CHD, stroke, diabetes, hypertension, and/or hypercholesterolemia. Of these 166 physicians, 127 (76.5\%) agreed to provide verification of their patients conditions, 3 (1.8\%) refused, 2 ( $1.2 \%$ ) no longer practiced medicine and 34 (20.5\%) did not respond after two reminder letters. Thus, physician interest in the study was reasonably high.

In total, there were 339 conditions requiring physician verification. Of these 339 , the respondents had provided permission to the researchers to contact their physician for
$86.7 \%$ of the verifiable conditions $(n=294)$. Of these 294 , physicians had agreed to verify 256 conditions. Ultimately, verification was received for 233 conditions. Thus, of the 339 conditions requiring verification, $68.7 \%$ ( $n=233$ ) of the verifications were ultimately received.

The reliability of self-reported conditions as measured against physician verification varied by condition (Table 421, far right column). There was high concordance (> 90\%) between respondents and physicians for hypertension, diabetes, ventricular hypertrophy, rheumatic heart disease, stroke and hypercholesterolemia. The concordance rates were lower for angina, myocardial infarction, congestive heart failure and arrhythmias (see Chapter 5 for discussion). However, there was reasonably good agreement between physicians and respondents who reported any type of CHD (66.7\%).

TABLE 4-21

## VERIFICATION STATUS OF SELECTED CARDIOVASCULAR CONDITIONS AND RISK FACTORS REPORTED BY RESPONDENTS

|  | Number To Be Verified | Permission Given To Contact MD | MD Agrees To Verify | MD VerificaReturned | Condition Verified |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Condition | N | $\underset{\%}{n / N}$ | $\underset{\%}{n / N}$ | $\underset{8}{\mathrm{n} / \mathrm{N}}$ | $\underset{\%}{\mathrm{n} / \mathrm{N}}$ |
| Heart Valve Problem | 5 | 5/5 | 3/5 | 3/3 | 1/3 |
|  |  | 100.0 | 60.0 | 100.0 | 33.3 |
| Myocardial Infarct | 12 | 12/12 | 12/12 | 10/12 | 5/10 |
|  |  | 100.0 | 100.0 | 83.3 | 50.0 |
| Angina | 23 | 23/23 | 20/23 | 17/20 | 11/17 |
|  |  | 100.0 | 87.0 | 85.0 | 64.7 |
| CHD* | 28 | 28/28 | 25/28 | 21/25 | 14/21 |
|  |  | 100.0 | 89.3 | 84.0 | 66.7 |
| Congestive <br> Heart Failure | 7 | 7/7 | 5/7 | 5/5 | 3/5 |
|  |  | 100.0 | 71.4 | 100.0 | 60.0 |
| Hypertrophy | 8 | 7/8 | 5/7 | 4/5 | 4/4 |
|  |  | 87.5 | 71.4 | 80.0 | 100.0 |
| Arrhythmia | 20 | 18/20 | 12/18 | 9/12 | 4/9 |
|  |  | 90.0 | 66.7 | 75.0 | 44.4 |
| Murmur | 6 | 5/6 | 3/5 | 2/3 | 1/2 |
|  |  | 83.3 | 60.0 | 75.0 | 50.0 |
| Rheumatic <br> Heart Disease | 2 | 2/2 | 1/2 | 1/1 | 1/1 |
|  |  | 100.0 | 50.0 | 100.0 | 100.0 |
| Hypertension | 136 | 130/136 | 119/130 | 107/119 | 104/107 |
|  |  | 95.6 | 91.5 | 89.9 | 97.2 |
| Diabetes | 54 | 43/54 | 39/43 | 36/39 | 33/36 |
|  |  | 79.6 | 90.7 | 92.3 | 91.7 |
| Hyperchol | 35 | 12/35 | 11/12 | 11/11 | 10/11 |
|  |  | 34.3 | 91.7 | 100.0 | 90.9 |
| Stroke | 3 | 2/3 | 1/2 | 1/1 | 1/1 |
|  |  | 66.7 | 50.0 | 100.0 | 100.0 |

[^1]
## CHAPTER 5: THE PREVALENCE OF CHD

The purpose of this chapter is to describe the prevalence of heart disease in the study population. In order to place the importance of CHD in context, the prevalence of other cardiovascular disorders will also be described. Just as hypertension, diabetes and hypercholesterolemia can be defined in different ways, CHD prevalence may also be defined according to history and/or measurement. In this case, Rose angina was the subjective measure used for CHD. Therefore the analyses of CHD prevalence, univariate relationships and multivariate relationships, were all conducted in three ways: 1) CHD by history alone, 2) CHD by Rose angina history, and 3) CHD history and/or Rose angina.

## I. The Prevalence of Selected Cardiovascular Conditions

In order to place CHD in context with other cardiac disorders, Table 5-1 summarizes the prevalence of respondent-reported cardiac health problems of any type. The overall prevalence of cardiovascular disorders of any kind was $6.2 \%$ for the males and $6.4 \%$ for the females. While there were no significant differences in these prevalences by sex for any of the age groups, there was a significant increase in prevalence with aging for both the males and the females.

TABLE 5-1
PREVALENCE OF RESPONDENTS REPORTING HAVING EVER BEEN TREATED FOR CARDIAC PROBLEMS OF ANY TYPE

| Age <br> Group | Male Sex Female |  |  |  | Test of $x^{2}$ Significance Between Sexes |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | p |
| 30-44 | 3/214 | 1.4 | 6/222 | 2.7 |  |  |  |
| 45-59 | 6/116 | 5.2 | 11/133 | 8.3 | 0.5 | 1 | 1.000 |
| 60-74 | 15/60 | 25.0 | 10/66 | 15.2 | 1.3 | 1 | 0.737 |
| 30-74 | 24/390 | 6.2 | 27/421 | 6.4 | 0.0 | 1 | 0.994 |

Test of $x^{2}$ Significance by Age Within Each Sex Group

|  | Male | Female |
| :--- | :---: | :---: |
| $\mathrm{x}^{2}$ | 45.5 | 14.3 |
| df | 2 | 2 |
| p | $<0.001$ | $<0.001$ |

The prevalence of each of the selected cardiovascular conditions, as self-reported by respondents, is presented in Table 5-2. There were no significant differences in the prevalence of any of the selected conditions between the sexes. The most prevalent self-reported cardiovascular condition was CHD for the males (3.3\%) and arrhythmias for the females (2.9\%).
II. Prevalence of Coronary Heart Disease by Self-reported

## History

A. Distribution by Age and Sex

Table 5-3 details the prevalence of self-reported medical histories of CHD. The numerators for this analysis include respondents reporting a history of angina, myocardial infarction, coronary bypass surgery and/or angioplasty. Each respondent is only counted once in the numerator, i.e. even if respondents report both angina and myocardial infarct, they were only counted once. The prevalence of CHD was $4.4 \%$ for the males, and $2.6 \%$ for the females. There were no significant differences in the prevalence of CHD between the sexes. Although the prevalence of CHD was higher in the 60-74 age group compared to the 45-59 age group for both sexes, the difference was significant only for the males.
B. Treatment Modalities for Self-reported CHD

Of the 17 males with a history of CHD, $82.2 \% ~(n=14)$ were taking medications for their condition; of the 11 females with a history of CHD $63.6 \%$ ( $n=7$ ) were taking CHD

## TABLE 5-2

## PREVALENCE OF SELECTED CARDIOVASCULAR CONDITIONS SELF-REPORTED BY RESPONDENTS BY SEX*

| Male <br> $(N=390)$ | Female <br> $(N=421)$ | Test of Significance |  |  |  |
| :--- | :--- | :--- | :--- | :--- | :--- |
| $n$ | $\%$ | $n$ | $\%$ | $x^{2}$ | $d f$ |

Related to CHD

| Myocardial <br> Infarct | 9 | 2.3 | 3 | 0.7 | 2.52 | 1. | 0.112 |
| :--- | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Angina | 13 | 3.3 | 10 | 2.4 | 0.37 | 1 | 0.542 |
| Coronary <br> Bypass | 1 | 0.3 | 0 | 0.0 |  |  |  |
| Angioplasty | 1 | 0.3 | 0 | 0.0 |  |  |  |
| CHD** | 17 | 4.4 | 11 | 2.6 | 1.37 | 1 | 0.243 |
| Congestive <br> Heart Failure | 2 | 0.5 | 5 | 1.2 |  |  |  |
| Arrhythmia | 8 | 2.1 | 12 | 2.9 | 0.26 | 1 | 0.612 |
| Other Cardiac |  |  |  |  |  |  |  |

problems

| Hypertrophy | 3 | 0.8 | 5 | 1.2 |
| :--- | :--- | :--- | :--- | :--- |
| Murmur | 1 | 0.3 | 5 | 1.2 |
| Rheumatic <br> Heart Disease | 1 | 0.3 | 1 | 0.2 |
| Heart Valve | 1 | 0.3 | 4 | 1.0 |

[^2]TABLE 5-3
PREVALENCE OF RESPONDENTS REPORTING HAVING EVER BEEN TREATED FOR CORONARY HEART DISEASE*


Test of $x^{2}$ Significance by Age $* *$ Within Each Sex Group
Male Female

| $x^{2}$ | 7.34 | 0.26 |
| :---: | :---: | :---: |
| $d f$ | 1 | 1 |
| $p$ | 0.014 | 1.00 |

* angina, myocardial infarct, coronary bypass, angioplasty ** 45-59 age category compared with 60-74 age category
related medications. One male reported a history of coronary artery bypass graft surgery, and another male had undergone coronary angioplasty. C. Physician Verification of Self-reported CHD

Of the 28 cases of self-reported CHD, physician verification was received for a 21 cases (75\%). Of these 21, physicians gave a positive verification for 66.7\% ( $\mathrm{n}=14$ ). Six of the cases for whom no physician verification was received were taking medications consistent with anginal therapy. Thus, of the 28 self-reported CHD cases, there was confirmatory evidence, either by physician verification or. medication use, for 20 cases (71.4\%).
D. Health Care Utilization for Self-reported CHD

Hospitalization at any time in the past for a myocardial infarct was reported by 7 male and 2 female respondents; angina was responsible for 3 male and 1 female admissions. Thirteen males and 12 females reported that they visited. their physician in the previous 12 months for a problem related to CHD.

## III. Prevalence of Rose Angina

The prevalence of Rose Angina by age and sex is presented in Table 5-4. Men had significantly more Rose angina than women (5.9\% vs 2.9\%). There was also a significant increase in the prevalence of Rose angina with aging for the men, but not for the women.

There was significantly more Rose angina among respondents of both sexes with a history of CHD than those

without CHD. Of the males without a history of CHD, 4.5\% ( $n=16$ ) were positive for Rose angina; $41.2 \% ~(n=7)$ of the males with a history of CHD were positive for Rose angina (p $<0.001$ ). The corresponding values for the women were $2.2 \%$ in those without a history of CHD and $27.3 \%$ in those with a history of CHD ( $p<.001$ ). Of the 18 respondents reporting that they were taking medications for angina, 6 (33.3\%) were positive for Rose angina.
IV. Prevalence of CHD as Defined by Self-reported History and/or Rose Angina

When CHD is more broadly defined to include those respondents reporting a history of CHD and/or having Rose angina, the prevalence for males is $8.5 \% \quad(n=33)$ and for females is $4.8 \%(n=20)(T a b l e 5-5)$. Overall, males had significantly more CHD by this broader definition than females. CHD also significantly increased with aging for the males, but not for the females.
V. Univariate Analysis of CHD and Rose Angina
A. Case Definitions Used for Hypertension, Diabetes,

Hypercholesterolemia, and CHD
As detailed in Chapter 4, the prevalence for hypertension, diabetes and hypercholesterolemia was calculated based on 1) treatment history alone and 2) treatment history and/or an elevated physical or laboratory measurements. An analysis was conducted to see whether there was a difference in the association between CHD and each of the three conditions based on how the condition was defined.

TABLE 5-5
PREVALENCE OF CHD AS DEFINED BY SELF-REPORTED HISTORY AND/OR ROSE ANGINA IN RESPONDENTS BY SEX

| Age Group | Male Sex Female |  |  |  | Test of $x^{2}$ Significance |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\mathrm{n} / \mathrm{N}$ | $\%$ | $\mathrm{n} / \mathrm{N}$ | \% | $\mathrm{x}^{2}$ | df | p |
| 30-44 | 5/214 | 2.3 | 7/222 | 3.2 | 0.05 | 1 | 1.000 |
| 45-59 | 13/116 | 11.2 | 7/133 | 5.3 | 2.21 | 1 | . 410 |
| 60-74 | 15/60 | 25.0 | 6/66 | 9.1 | 4.64 | 1 | 0.093 |
| 30-74 | $33 / 390$ | 8.5 | 20/421 | 4.8 | 3.98 | 1. | 0.046 |
| Test of $x^{2}$ Significance by Age Within Each Sex Group |  |  |  |  |  |  |  |
| Male Fem |  |  |  |  |  |  |  |
| $x^{2}$ | 32. |  |  |  |  |  |  |
| df | 2 |  |  |  |  |  |  |
| p | $<0.0$ |  |  | 60 |  |  |  |

As can be seen from Table 5-6, for each of the three Conditions their relationship with CHD was similar whether the definition was based on history of treatment alone or on a history of treatment and/or the appropriate level of the physical or laboratory measurement. For both forms of the definition, hypertension and diabetes were significantly associated with CHD, while hypercholesterolemia was not.

As both forms of the definition for each of the three conditions were related to CHD in the same way, and, as the definitions that include both treatment history and/or measurement captured more information than the definition based on history alone, the former definition was used in all subsequent analyses. Therefore, in all subsequent analyses the terms hypertension, diabetes and hypercholesterolemia are based on their broader definitions of history and/or measurement.

In order to provide a reference point for interpreting the adjusted odds ratios (OR) for the continuous variables, the following conventions were followed: 1) The OR for BMI was based on the 7 Index point difference between the lower end of the healthy BMI scale (20) and the lower end of the unhealthy scale (27), 2) the OR for age was based on a 10year difference and 3) the OR for systolic pressure was based on a 10 mm Hg difference in pressure.

TABLE 5-6
DEFINITION-DEPENDENT RELATIONSHIP BETWEEN CHD AND HYPERTENSION, DIABETES AND HYPERCHOLESTEROLEMIA. USING LOGISITIC REGRESSION

| Relationship with CHD by Condition's Definition ${ }^{1}$ |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| Definition | beta | $\frac{\text { beta }}{\text { s.E. (b) }}$ | beta | $\frac{\text { beta }}{\mathrm{s} \cdot \mathrm{E} \cdot(\mathrm{~b})}$ |
| Hypertension |  |  |  |  |
| a) Treatment alone | . 724 | 2.883** | . 941 | 3.223** |
| b) Treatment and/or DBp > 99 mm Hg | . 844 | 2.902** | . 995 | 3.250** |
| Diabetes |  |  |  |  |
| a) Treatment alone | 1.058 | 3.839*** | . 683 | 1.992* |
| b) Treatment and/ or 1.0 Glucose $>11.19 \mathrm{mmol} / 1$ | 1.014 | 3.698*** | . 844 | 2.681** |
| Hypercholesterolemia |  |  |  |  |
| a) Treatment alone | -3.589 | . 3289 | . 533 | 1.339 |
| b) Treatment and/or Cholesterol > $6.19 \mathrm{mmol} / \mathrm{I}$ | $-.084$ | . 335 | .276 | . 975 |
| * $\mathrm{p}<0.05, * * \mathrm{p}<.01, * * * \mathrm{p}<.001$ |  |  |  |  |
| 1 two definitions, one based on treatment alone and one based on treatment and/or measurement, for each of the three conditions (hypertension, diabetes and hypercholesterolemia) were entered into a logistic regression with CHD as the outcome variable |  |  |  |  |

B. Univariate and multivariate analysis of CHD by History

## Alone

1. Univariate analysis. All of the variables that significantly related to CHD, by history alone, for either the males or the females, are listed in Table 5-7. For both the males and the females there was a significant relationship between CHD and:
1) BMI in $\mathrm{kg} / \mathrm{m}^{2}$
2) hypertension
3) diabetes
4) age in years
5) systolic pressure in mm Hg

The amount of regular exercise and obesity were significantly related to CHD for the females, but not for the males.

In addition, the following factors were not significantly related to $C H D$ in the univariate (unadjusted) analysis for either the males or the females:

1) diastolic pressure in mm Hg
2) blood glucose in mmol/l
3) blood cholesterol in mmol/l
4) hypercholesterolemia
5) a positive family history of CHD in any relative or in parent before the age of 55 years, or in a parent after the age of 55 years
6) having ever smoked
7) amount of alcohol consumed regularly
8) usual amount of stress

None of the interaction terms for age and treatment for hypertension, diabetes or hypercholesterolemia were significant.

TABLE 5-7
UNIVARIATE RELATIONSHIPS BETWEEN CORONARY
HEART DISEASE AS DEEINED BY HISTORY ONLY AND CHD RISK FACTORS BY SEX USING LOGISTIC REGRESSION*

| Risk Factor | Univariate <br> Beta |  | Relatio $\frac{\text { Beta }}{S E(b)}$ | ship: <br> p | and CH <br> Odds <br> Ratio | D Risk Factors <br> 95\% CI of Odds Ratio |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Hypertension | M | . 844 | 2.902 | . 004 | 2.33 | (1.32-4.11) |
|  | F | . 995 | 3.250 | . 001 | 2.70 | (1.48-2.70) |
| Diabetes | M | 1.014 | 3.698 | $<.001$ | 2.76 | (1.61-4.72) |
|  | F | . 844 | 2.681 | . 008 | 2.32 | (1.25-4.31) |
| BMI in $\mathrm{kg} / \mathrm{m}^{2}$ | M | 1.465 | 1.987 | . 046 | 1.62 | (1.17-2.24) ${ }^{1}$ |
|  | F | . 049 | 2.790 | . 006 | 1.42 | $(1.11-1.81)^{1}$ |
| Age in years | M | . 074 | 5.023 | $<.001$ | 2.09 | (1.57-2.79) ${ }^{2}$ |
|  | F | . 049 | 3.806 | $<.001$ | 1.64 | $(1.27-2.11)^{2}$ |
| Systolic Pres- | M | . 014 | 2.610 | . 009 | 1.14 | $(1.03-1.27)^{3}$ |
| sure in mm Hg | F | . 017 | 3.176 | . 001 | 1.19 | $(1.07-1.32)^{3}$ |
| Obesity | M | . 435 | 1.605 | . 120 | 1.55 | (.91-2.63) |
|  | F | . 838 | 2.162 | . 030 | 2.31 | (1.08-4.94) |
| Regular | M | . 286 | 1.147 | . 250 | 1.33 | ( . 82-2.17) |
| Exercise | F | . 627 | 2.162 | . 030 | 1.87 | (1.06-3.31) |

* a) only variables significant for either males or females are included in the Table. (See text for non-significant variables)
b) CHD only by history of myocardial infarction, angina, coronary bypass surgery and/or angioplasty

1: based on an Index difference of 7 points (i.e. a BMI of 20 vs 27)
2: based on an age difference of 10 years (i.e. 50 vs 60)
3 : based on a systolic pressure difference of 10 mm Hg (i.e. 140 vs 150)
2. Multivariate analysis. When the variables that were significantly related to CHD in the univariate analysis are entered into the multivariate analysis (Table 5-8):

1) in the males: regular exercise becomes significantly related to CHD, and systolic blood ceases to be significantly related to CHD
2) in the females: hypertension, diabetes, BMI in $\mathrm{kg} / \mathrm{m}^{2}$, obesity and systolic pressure in mm Hg cease to be significantly related to CHD

In the adjusted analysis, the males with CHD were more likely to be hypertensive ( $O R=2.69$ ), have diabetes ( $O R=2.21$ ) and exercise regularly ( $O R=2.12$ ) than those without CHD. The odds of males having CHD also increase with aging (OR=2.51) and increasing BMI (OR=2.16).

The females with CHD were more likely to engage in regular exercise ( $\mathrm{OR}=3.36$ ) than those without CHD and the association with CHD also increased with age (OR=1.79). C. Univariate and Multivariate Analysis of Rose Angina

1. Univariate Analysis. The factors significantly associated with Rose Angina in the univariate (unadjusted) analysis were largely similar to those found for cHD for the males, but not for the females (Table 5-9). Only one factor, diabetes, was significantly related to Rose Angina for the females. The variables significantly associated with Rose Angina for the males included:
1) BMI in $\mathrm{kg} / \mathrm{m}^{2}$
2) hypertension
3) systolic pressure in mm Hg
4) age in years
5) diabetes

TABLE 5-8
MULTIVARIATE RELATIONSHIPS BETWEEN CORONARY HEART DISEASE AS DEFINED BY HISTORY ONLY AND CHD RISK FACTORS BY SEX USING LOGISTIC REGRESSION

| Risk Factor |  | Beta | $\frac{\text { Beta }}{S E(b)}$ | $p$ | Odds Ratio | 95\% CI of Odds Ratio |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Hypertension | M | . 991 | 2.349 | . 018 | 2.69, | (1.18-6.16) |
|  | F | . 314 | . 828 | . 406 | 1.37 , | ( . 68-2.74) |
| Diabetes | M | . 791 | 2.192 | . 028 | 2.21, | (1.09-4.46) |
|  | F | . 645 | 1.818 | . 068 | 1.91, | ( .95-3.82) |
| BMI in $\mathrm{kg} / \mathrm{m}^{2}$ | M | . 110 | 2.644 | . 008 | 2.16, | (1.22-3.80) ${ }^{1}$ |
|  | $F$ | . 023 | . 765 | . 442 | 1.18, | $(.78-1.78)^{1}$ |
| Age in years | M | . 092 | 4.375 | $<.001$ | 2.51, | (1.66-3.77) ${ }^{2}$ |
|  | F | . 058 | 3.119 | . 002 | 1.79, | (1.24-2.59) ${ }^{2}$ |
| Systolic Pressure in m mg | M | -. 014 | 1.806 | . 070 | . 87. | (.75-1.01) ${ }^{3}$ |
|  | F | -. 002 | . 281 | . 780 | . 98 , | $(.83-1.15)^{3}$ |
| Regular <br> Exercise | M | . 753 | 2.186 | . 046 | 2.12, | (1.08-4.17) |
|  | F | 1.212 | 3.243 | . 001 | 3.36, | (1.62-6.99) |

1: based on an Index difference of 7 points (i.e. a BMI of 20 Vs 27 )
2: based on an rage difference of 10 years (i.e. 50 vs 60)
3: based on a systolic pressure difference of 10 mm Hg (i.e. 140 vs 150)

TABLE 5-9
UNIVARIATE RELATIONSHIPS BETWEEN ROSE
ANGINA AND CHD RISK FACTORS BY SEX
USING LOGISTIC REGRESSION*

| Risk Factor |  | Beta | $\frac{\text { Beta }}{\operatorname{se}(b)}$ | $p$ | ocdds <br> Ratio | 95\% CI of Odds Ratio |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Hypertension | M | . 786 | 3.226 | . 001 | 2.19 | (1.36-3.54) |
|  | F | . 211 | . 677 | . 596 | 1.23 | ( . 67-2.27) |
| Diabetes | M | . 917 | 3.666 | $<.001$ | ,2.50 | (1.53-4.08) |
|  | F | . 904 | 2.824 | . 004 | 2.47 | (1.32-4.62) |
| $B M I$ in $\mathrm{kg} / \mathrm{m}^{2}$ | M | . 047 | 2.193 | . 028 | 1.38 | (1.04-1.86) ${ }^{1}$ |
|  | F | . 020 | . 956 | . 338 | 1.15 | $(.86-1.54)^{1}$ |
| Age in years | M | . 037 | 4.157 | $<.001$ | 1.45 | $(1.22-1.73)^{2}$ |
|  | F | . 009 | . 719 | . 572 | 1.09 | $(.86-1.38)^{2}$ |
| Systolic Dressure in mm Hg | M | . 014 | 3.101 | . 002 | 1.15 | $(1.05-1.26)^{3}$ |
|  | F | . 003 | . 479 | . 732 | 1.03 | $(.90-1.19)^{3}$ |

* a) only variables significant for either males or females are included in the Table. (See text for non-significant variables)

1: based on an Index difference of 7 points (i.e. a BMI of 20 vs 27)
2: based on an age difference of 10 years (i.e. 50 vs 60)
3: based on a systolic pressure difference of 10 mm Hg (i.e. 140 vs 150)

The factors not significantly related to Rose Angina for either the males or the females were similar to those found in the univariate-CHD analysis, and included:

1) blood glucose in mmol/l
2) blood cholesterol in mmol/l
3) hypercholesterolemia
4) obesity
5) regular exercise
6) a positive family history of CHD in any relative or in parent before the age of 55 years, or in a parent after the age of 55 years
7) having ever smoked
8) amount of alcohol consumed regularly
9) usual amount of stress

In addition, none of the interaction terms for age and treatment for hypertension, diabetes and hypercholesterolemia were significant.
2. Multivariate analysis. In the adjusted analysis (Table 5-10), both BMI and systolic pressure cease to be significantly related to Rose Angina for the males. Males with Rose angina were more likely to be hypertensive ( $O R=1.77$ ) and diabetic ( $O R=2.72$ ). For males there was also an increase in Rose angina with aging (OR=1.28).

For the females, only diabetes ( $O R=2$.72) was associated with Rose angina. The lack of other significant relationships with Rose angina in the females may, in part, be due to its extremely low prevalence.

## D. Univariate and Multivariate Analysis of CHD Cases

Combined with Those Having Rose Angina

1. Univariate analysis. When the operational definition of CHD is broadened to include all of those with either Rose Angina or a history of CHD, the factors

## TABLE 5-10

## MULIIVARIATE RELATIONSHIPS BETWEEN ROSE ANGINA CHD RISK FACTORS BY SEX USING LOGISTIC REGRESSION



1: based on an Index difference of 7 points (i.e. a BMI of 20 vs 27)
2: based on an age difference of 10 years (i.e. 50 vs 60)
3: based on a systolic pressure difference of 10 mm Hg (i.e. 140 vs 160)
associated with the more broadly defined CHD in the univariate (unadjusted) analysis is almost identical to that described above for the more limited definition of CHD cases (only a history of myocardial infarction etc.). The results of the univariate analysis is presented in Table 5-11. The only differences between the two univariate analyses (more narrowly vs. more broadly defined CHD) are that, in the later, 1) obesity is significantly associated with CHD for both the sexes, not just the females, and 2) regular exercise ceases to be significantly related to CHD for the females.

Therefore, factors associated with the more broadly defined form of CHD for both sexes included:

1) hypertension
2) diabetes
3) BMI
4) age
5) systolic blood pressure
6) obesity
2. Multivariate analysis. The results of the multivariate analysis for the more broadly defined CHD are. presented in Table 5-12. In the adjusted analysis for the males, hypertension ( $O R=2.23$ ) and diabetes ( $O R=2.63$ ) were significantly associated with CHD. The odds of having CHD for males also increased with aging ( $O R=1.42$ ).

Just as in the adjusted analysis of Rose Angina for the females, only diabetes is significantly related to the more broadly defined CHD, with an adjusted odds ratio of 2.04 .

TABLE 5-11
UNIVARIATE RELATIONSHIPS BETWEEN CORONARY
HEART DISEASE AS DEFINED BY EITHER A CHD HISTORY AND/OR ROSE ANGINA AND CHD RISK FACTORS By SEX USING LOGISTIC REGRESSION*


[^3]1: based on an Index difference of 7 points (i.e. a BMI of 20 vs 27)
2: based on an age difference of 10 years (i.e. 50 vs 60 )
3: based on a systolic pressure difference of 10 mm Hg (i.e. 140 vs 150)

TABLE 5-12
MULTIVARIATE RELATIONSHIPS BETWEEN CORONARY
HEART DISEASE AS DEFINED BY EITHER A CHD HISTORY AND/OR ROSE ANGINA AND CHD RISK FACTORS
BY SEX USING LOGISTIC REGRESSION*


1: based on an Index difference of 7 points (i.e. a BMI of 20 vs 27)
2: based on an age difference of 10 years (i.e. 50 vs 60)
3: based on a systolic pressure difference of 10 mm Hg (i.e. 140 vs

## CHAPTER 6: DISCUSSION OF THE STUDY RESULTS

I. The Prevalence of Selected CHD Risk Factors in the Study

## Population

A. The Prevalence of Hypertension

When compared to either Canadian or other western industrialized populations, the prevalence of hypertension was surprisingly high in the Hutterite population. As shown in Chapter 4, the prevalence of hypertension is closely related to the definition of hypertension used. Depending on the criteria used, the prevalence (\%) of hypertension in Hutterite males and females was:

Criteria

1) Treatment alone
2) Diastolic $\mathrm{Bp}>90$
3) Diastolic Bp > 99
4) Diastolic Bp > 99
and/or under treatment

Male Prevalence Female Prevalence 22.3 20.9
57.5
29.5
26.0
12.2
39.2
24.9

Regardless of the definition used, and as found in most population surveys, the prevalence of hypertension increased for both males and females with age. Of known hypertensives, Hutterite females were more likely to be under treatment for hypertension than males, a common finding in other population surveys (Federal/Provincial Working Group, 1986).

In order to place the Hutterites' prevalence of hypertension in perspective, as shown in Table 6-1, comparisons were made with other published rates. It is

TABLE 6-1
COMPARISON OF THE PREVALENCE OF HYPERTENSION FOUND IN ALBERTA HUTTERITES AND OTHER POPULATION SURVEYS

| Comparison Study and Criteria Used | Age Groups | Other <br> Prev <br> Male <br> \% | study <br> lence <br> Female \% | Hutt Male \% | rite <br> Female \% |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Canadian Blood |  |  |  |  |  |
| Pressure Survey (1) |  |  |  |  |  |
| b) $\mathrm{DBp}>90 \mathrm{~mm} \mathrm{Hg}$ | 45-64 | 21 | 11 | 67 | 44 |
| Nova Scotia Heart |  |  |  |  |  |
| Survey (2) |  |  |  |  |  |
| a) $\mathrm{DBp}>90 \mathrm{~mm} \mathrm{Hg}$ and/or on treatment | 35-64 | 25 | 21 | 49 | 3.3 |
| South Dakota |  |  |  |  |  |
| Hutterites(3) |  |  |  |  |  |
| a) $\mathrm{DBp}>90 \mathrm{~mm} \mathrm{Hg}$ | 30-39 | 28 | 6 | 36 | 10 |
|  | 40-49 | 41 | 28 | 52 | 29 |
|  | 50-59 | 56 | 28 | 62 | 42 |
|  | 60+ | 38 | 45 | 58 | 45 |
| South Dakota |  |  |  |  |  |
| Non-Hutterites(3) |  |  |  |  |  |
| a) $\mathrm{DBp}>90 \mathrm{~mm} \mathrm{Hg}$ | 30-39 | 7 | 3 | 36 | 10 |
|  | 40-49 | 37 | 4 | 52 | 29 |
|  | 50-59 | 20 | 13 | 62 | 42 |
|  | $60+$ | 8 | 7 | 58 | 45 |
| North Karelia(4) |  |  |  |  |  |
| a) DBp $>99 \mathrm{~mm} \mathrm{Hg}$ | 40-44 | 24 | 14 | 35 | 14 |
| and/or $\mathrm{SBp}>$ | 45-49 | 28 | 29 | 48 | 24 |
| 174 mm Hg and/ | 50-54 | 36 | 27 | 53 | 52 |
| or on treatment | t 55-59 | 44 | 38 | 58 | 43 |

(1) Health and Welfare Canada, 1989
(2) MacLean; 1986
(3) Schlenker et al., 1989
(4) Nissinen et al., 1988
important to bear in mind that most published reports do not provide the information necessary to make statistical comparisons with the study population, i.e. age distributions, standard errors etc.. Therefore the comparisons are limited to age- and sex-specific data from published reports. It is also necessary to realize that there is a lack of consistency in age-groupings used by various researchers. Therefore, wherever necessary, the study data was re-analyzed to match age groups with specific comparison studies.

Depending on the definition of hypertension used in the Canadian Blood Pressure Survey (CBPS) (Health and Welfare Canada, 1989), the prevalence of hypertension among the Hutterites was between two and four times greater than found in the general Canadian population. The differences in the ratios of Hutterite to Canadian hypertension prevalence for the two definitions of hypertension would suggest that Hutterites with hypertension are less likely to be under control than non-Hutterite Canadians. Indeed, as will be seen below, this is the case.

Nova Scotia has one of the highest prevalence rates of hypertension in Canada (MacLean, 1986), consequently the Nova Scotia Heart Health Survey (NSHHS) provides an interesting comparison group for the Hutterites. As can be seen in Table 6-1, the Hutterite males had almost twice the hypertension prevalence of the Nova Scotian males, and the Hutterite females had a prevalence 63\% higher than the Nova

Scotian females.
A comparison of the prevalence of a diastolic pressure $>90 \mathrm{~mm} \mathrm{Hg}$ in the present study with the study of South Dakota Hutterites and non-Hutterite controls by Schlenker et al. (1989), is also presented in Table 6-1. The age- and sex- specific prevalence of diastolic pressure $>90 \mathrm{~mm} \mathrm{Hg}$ is remarkably similar for the Hutterites from both South Dakota and Alberta. The non-Hutterite controls from South Dakota had a significantly lower prevalence of diastolic pressure > 90 mm Hg than the South Dakota Hutterites, and, as can be seen in Table 6-1, the Alberta Hutterites had an even higher prevalence than the South Dakota Hutterites.

Table 6-1 also provides a comparison of the prevalence of hypertension between the Alberta Hutterites and residents of an area known for its high hypertension prevalence, North Karelia, Finland (Nissinen et al., 1988). The North Karelia program provides an interesting comparison for the Hutterites as it represents the prevalence of hypertension in a population that has undergone an intensive community hypertension intervention program for many years. As can be seen, Hutterite males have a higher age-specific prevalence of hypertension in each age group as compared to the North Karelia males. Female Hutterites, on the other hand, have a either a similar or lower prevalence of hypertension than the North Karelia females prior to age 50; then the Hutterite females overtake the North Karelia females. Indeed, after age 50, the female Hutterites and the male

Hutterites have a similar prevalence of hypertension. A similar relationship was found by Schlenker et al. (1989) in South Dakota Hutterites.

A final comparison of hypertension prevalence will be made between Hutterite females and females in the Nurses Health Study (Fiebach et al., 1989). The definition of hypertension used in the Nurses Health Study was based on self-reports of having ever been diagnosed with hypertension. The Hutterite females had a similar prevalence of hypertension until age 50 and then their prevalence is twice that of the nurses (42.4\% vs 20.0\%) i. this finding is similar to the results comparing Hutterite and non-Hutterite residents of South Dakota described above (Schlenker et al., 1989).

Both the CBPS and the NSHHS evaluated the awareness, treatment status and control status of survey participants who were either under a physician's care for hypertension or had a diastolic pressure greater than or equal to 90 mm Hg . The same analysis was conducted for the Hutterites and the results are presented in Table 6-2. Compared to the other two Canadian studies, the Hutterites were less likely to be aware of their elevated diastolic pressure, less likely to be under treatment for hypertension and, even if under treatment, they were less likely to be under control. Of those under treatment for hypertension, $76 \%$ of the Hutterites $(26 \% /(26 \%+8.4 \%))$ were uncontrolled (diastolic
pressure $>90 \mathrm{~mm} \mathrm{Hg})$; by comparison only $27 \%$ of those in the CBPS and 19\% of those in the NSHHS were uncontrolled.

TABLE 6-2

HIGH BLOOD PRESSURE AWARENESS, TREATMENT, AND CONTROL STATUS*: COMPARISON BETWEEN ALBERTA HUTTERITES, THE NOVA SCOTIA HEART SURVEY (NSHHS), AND THE CANADIAN BLOOD PRESSURE SURVEY (CBPS)

| Status | NSHHS <br> $\%$ | CBPS <br> $\%$ | Hutterites <br> $\%$ |
| :--- | :---: | :---: | :---: |
| Aware <br> Treated and <br> controlled (a) | 55.0 | 43.0 | 8.4 |
| Treated and <br> not controlled (b) | 13.0 | 16.0 | 26.1 |
| Not treated and <br> not controlled (c) | 15.0 | 5.0 | 9.8 |
| Not Aware(d) | 17.0 | 36.0 | 55.7 |
| Total | 100.0 | 100.0 | 100.0 |

* the denominator includes respondents who were ever told that they had hypertension or had a diastolic pressure greater than or equal to 90 mm Hg. Excluded are those who had been told that they had hypertension but were not on treatment and had a pressure $<90 \mathrm{~mm} \mathrm{Hg}$
a) told hypertension present, on treatment, diastolic pressure < 90 mm Hg
b) told hypertension present, on treatment, diastolic pressure $>$ or equal to 90 mm Hg
c) told hypertension present, not on treatment, diastolic pressure $>$ or equal to 90 mm Hg
d) never told hypertension present, diastolic pressure > or equal to 90 mm Hg .

There are a number of possible explanations for the unusually high prevalence of hypertension found in the Hutterites. Elevated BMI, particularly when associated with central torso distribution of fat, is known to have a strong relationship with hypertension (Selby et al., 1989). As discussed in Chapter 4 , the prevalence of a BMI $>27 \mathrm{~kg} / \mathrm{m} 2$ is over $50 \%$ for the Hutterite population.

Alcohol consumption of more than one to two beverages per day has also been associated with hypertension (Weissfeld et al., 1988). As described in Chapter 3, over half of the males and $13 \%$ of the females reported that they consumed at least one alcoholic beverage per day.

Dietary intake of sodium and potassium has also been linked to hypertension prevalence (Joint National Committee, 1986). While measurement of these cations was not done, $a$ previous study of Hutterite diet (Roukusek-Kennedy et al., 1987) found that the sodium to potassium ratio was 4:1, a ratio believed to favor elevation of blood pressure (Huttunen, Pietinen, Nissinen, Puska, 1985).

Finally, there is considerable evidence supporting a genetic role in the familial aggregation of hypertension (Clark, Schrott, Burns, Sing, and Lauer, 1986). While there was no genetic analysis performed on the Hutterite data, the high coefficient of inbreeding known to exist in this population may play a part in the high prevalence of hypertension.

## B. The Prevalence of Hypercholesterolemia

The prevalence of hypercholesterolemia in the study population was found to be consistently higher than in other studies of cholesterol in Canada and other industrialized nations. The prevalence (\%) of hypercholesterolemia was strongly influenced by the criteria used:

Criteria

1) Treatment alone
2) Cholesterol > 6.20
3) Treatment and/or Cholesterol > 6.20

Male Prevalence Female Prevalence
5.4
6.2
46.6
42.1
43.6

Despite the high proportion of Hutterites with an elevated cholesterol, a very small percentage were under any form of treatment for hypercholesterolemia. Of the 21 male and 26 female respondents who were under a physicians care for elevated cholesterol, the majority were conservatively treated by diet modification alone (52.4\% of males, $76.9 \%$ of females). No females and just $19 \%$ of the males under treatment for elevated cholesterol were taking medications for the condition.

Table 6-3 provides a comparison of the prevalence of elevated serum cholesterol between Hutterites and a number of recent North American population studies. One of the largest North American studies examined the serum

TABLE 6-3
COMPARISON OF THE PREVALENCE OF ELEVATED SERUM CHOLESTEROL FOUND IN ALBERTA HUTTERITES AND OTHER POPULATION SURVEYS

| Comparison Study <br> and Criteria Used | Age Groups |
| :--- | :--- | | Other Study |
| :---: |
| Prevalence |
| Male Female |
| $\%$ |$\quad$| Hutterite |
| :---: |
| $\%$ | | Male Female |
| :---: |
| $\%$ |

U.S. Population(1)

| a) Cholesterol | $35-44$ | 27.9 | 21.1 | 36.1 | 26.8 |
| :--- | :--- | :--- | :--- | :--- | :--- |
| $>6.20$ mmol/I | $45-54$ | 36.5 | 40.6 | 58.8 | 46.9 |
|  | $55-64$ | 37.3 | 53.7 | 38.3 | 57.4 |
|  | $65-74$ | 32.4 | 52.1 | 50.0 | 65.7 |
|  |  |  |  |  |  |
| Ova scotia Heart |  | 10.0 | 11.0 | 28.5 | 22.8 |
| urvey(2) |  |  |  |  |  |
| a) Cholesterol | $35-64$ | 10.0 | 21.0 | 33.3 | 45.7 |
| $>6.72$ mmol $/ \mathrm{l}$ | $65-74$ |  |  |  |  |

Maritime Telephone and Telegraph(3)
$\begin{array}{cccccc}\begin{array}{c}\text { Cholesterol } \\ >6.20 \mathrm{mmol} / 1\end{array} & 35-64 & 14.0 & 12.0 & 43.0 & 40.0\end{array}$

## Canada Health

Survey (4)
a) Cholesterol
35-44
$>6.45 \mathrm{mmol} / \mathrm{l}$
45-64
$11.3 \quad 0.0$
27.118 .1
22.611 .8
42.137 .8

## Hartford Connecticut(5)

a) Cholesterol
31-40
17.89 .1
$36.1 \quad 25.7$
$>6.20 \mathrm{mmol} / \mathrm{l}$
41-50
$27.8 \quad 19.0$
$49.0 \quad 37.7$
51-60
$27.2 \quad 36.5$
$49.3 \quad 60.0$
$>60$
27.644 .9
$43.2 \quad 54.9$
(1) National Center for Health Statistics, 1986
(2) MacLean, 1986
(3) Occupational Health Services, 1989
(4) Health and Welfare Canada, 1981
(5) Wynder et al., 1989
cholesterol values in a sample of the United states population (National Center for Health Statistics, 1986). As seen in Table 6-3, the Hutterites had a higher age and sexspecific prevalence of hypercholesterolemia than the U.S. population. The prevalence of hypercholesterolemia for women after the child-bearing years, in both the Hutterite study and the U.S. study, is higher than found in men.

Table 6-3 also summarizes the prevalence of hypercholesterolemia found in the Hutterites and three other Canadian studies. Regardless of the definition of hypercholesterolemia used, or the age categories specified, Hutterites have a higher prevalence of hypercholesterolemia than non-Hutterites in all three Canadian investigations. Compared with the NSHHS (MacLean, 1986), Hutterite males have three times, and Hutterite females have two times, the prevalence of hypercholesterolemia (blood cholesterol > 6.72 mmol/l). Compared to the Maritime Telephone and Telegraph Health Survey (MTMHS) (Occupational Health Services, 1989), Hutterite males and females both have three times the prevalence of hypercholesterolemia (blood cholesterol >6. 20 mmol/l). Just as was found in both the NSHHS and the MTTHS, compared with Canadians in the Canada Health survey (Health and Welfare Canada, 1981), Hutterites have between two and three times the prevalence of hypercholesterolemia (blood cholesterol $>6.45 \mathrm{mmol} / \mathrm{l})$.

A final comparison of hypercholesterolemia prevalence in Hutterites and residents of Hartford, connecticut
(Wynder, Harris, and Hayley, 1989) is presented in Table 63. The Hartford population provides an interesting reference for the Hutterites because the screening and measurement procedures were similar in both studies. Compared to the Hartford population, both the male and the female Hutterites have between 1.5 and 2 times the prevalence of hypercholesterolemia in every age group. Both the Hutterite and Hartford females have lower prevalences of elevated cholesterol until age 50 , at which time they overtake the males.

There are a number of possible explanations for why the Hutterites have such a high prevalence of elevated cholesterol. Dietary lipids, particularly saturated fats, are known to play an important role in the production of circulating cholesterol (The Expert Panel, 1988). As discussed in Chapt 3 of this study, and confirmed by the analysis of Hutterite diets by Rokusek-Kennedy et al. (1987), the Hutterite diet is high in saturated fats. Deep frying in animal lard is a standard dietary practice among the Hutterites, as is frequent consumption of dairy products and red meat. Elevated BMI, such as found in over half of the study population, has also been shown to have a strong association with serum lipids (National Center for Health Statistics, 1983). Aerobic exercise, inversely associated with elevated BMI, was not found to be routinely used by the Hutterites and may contribute to the elevated cholesterol levels observed. While the role of genetics in relatively
rare hyperlipoproteinemias is well known, there is growing evidence that genetics play a role in less severe forms of lipid disorders as well (Austin, King, Bawol, Hulley, and Friedman, 1987; Berg, 1985). Again, because of the high coefficient of in-breeding found among the Hutterites, it is entirely possible that the high prevalence of elevated cholesterol is, in part, genetic in origin.

## C. The Prevalence of Diabetes Mellitus

Regardless of the criteria use, the prevalence (\%) of diabetes was similar for males and females in all age groups:

Criteria

1) Treatment alone
2) Glucose > 11.19
3) Treatment and/or

Glucose > 11.19

Male Prevalence Female Prevalence
7.4
7.6
2.7
4.3
7.9
8.3

There was a significant increase in the prevalence of diabetes with age for both sexes. Three-quarters of respondents with a serum glucose $>11.19$ mmol/l were known diabetics. There was some evidence that male diabetics were under better control than the female diabetics: $85.7 \%$ of the males had a random glucose less than $11.19 \mathrm{mmol} / 1$, whereas the corresponding rate for females was $65.6 \%$.

Virtually nothing is known about the prevalence of diabetes in Canada (Knowler, Everhart, and Bennett, 1985; personal communication, S. Ross, 1989). The most comprehensive examination of the prevalence of diabetes in North America was conducted in the United States as part of
the Second National Health and Nutrition Examination Survey (NHANES II) (Harris, Hadden, Knowler, and Bennett, 1987).

Table 6-4 compares the prevalence of diabetes by age and sex in the Hutterites with the NHANES II data. The definition used for diabetes was simply a self-report of a history of diabetes. Compared to the white U.S. population, the prevalence of diabetes is higher for the Hutterites in all age groups and for both sexes. The Hutterites males also have a higher prevalence of diabetes than U.S. blacks in all age groups, though the differences are not as great as for the U.S. white males. The Hutterite females have a prevalence of diabetes similar to black females in the 45-54 and 55-64 age groups, but have almost double the rate in the 64-74 age category.

Undoubtedly, the high prevalence of elevated BMI found in the Hutterite population strongly contributes to the prevalence of diabetes (Bray, 1987; Health and Welfare Canada, 1988b).

Another possible explanation is that the prevalence of diabetes among the Hutterites is related to their high coefficient of inbreeding. Diabetes has long been known to be a familial disorder (Rotter, Vadheim, Raffel, and Rimoin, 1984), and, while genetic analysis was not conducted in the present study, diabetes did cluster in certain colonies.

TABLE 6-4
COMPARISON OF THE PREVALENCE OF DIABETES* BETWEEN HUTTERITES AND THE NHANES II STUDY**

|  |  | NHANE | II |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | ites | Bla | ks | Hutte | ites |
| Age Group | $\underset{\%}{\text { Male }}$ | $\begin{gathered} \text { Female } \\ \% \end{gathered}$ | $\begin{aligned} & \text { Male } \\ & \% \end{aligned}$ | Female | $\begin{aligned} & \text { Male } \\ & \% \end{aligned}$ | Female \% |
| 45-54 | 4.5 | 3.9 | 3.6 | 7.5 | 6.3 | 7.3 |
| 55-64 | 5.3 | 6.6 | 9.2 | 16.3 | 13.3 | 14.7 |
| 65-74 | 9.1 | 8.8 | 17.2 | 10.8 | 25.0 | 17.1 |
| * by self <br> ** Harris | porte al., | $\begin{aligned} & \text { history } \\ & 987 \end{aligned}$ |  |  |  |  |

Such colony-related clustering of diabetes may serve as a surrogate for familial clustering. The clustering of diabetes in colonies was also observed by Fowlow (1983). D. The Prevalence of Cigarette Smoking

The prevalence of cigarette smoking was very low among the Hutterites, only $3.6 \%$ of the males, and none of the females, reported even occasional tobacco use. This low prevalence of smoking was similar to the $5 \%$ reported in South Dakota Hutterites by Schlenker et al. (1989). By comparison, $36 \%$ of Canadian males and $32 \%$ of Canadian females are regular smokers (Health and Welfare Canada, 1988a). The social sanctions against smoking in the Hutterite community likely contributes to smoking's low prevalence (Hostetler, 1974).
E. The Prevalence of Elevated Body Mass Index

Just over half of the males (52.8\%) and females (53.6\%) in the study had a BMI greater than $27 \mathrm{~kg} / \mathrm{m}^{2}$. BMI increased significantly with aging for the females, but not for the males. Compared to other studies, both male and female Hutterites have higher BMIs than the Canadian population.

Table 6-5 compares the prevalence of a BMI greater than $27 \mathrm{~kg} / \mathrm{m}^{2}$ in Hutterites and Canadians who participated in the Canada Fitness Survey (CFS) (Fitness Canada, 1981) and the Canada Health Survey (CHS) (Health and Welfare Canada, 1981). While the methodology used in the CFS was similar to that used in the Hutterite study, a certain amount of caution must be taken in assessing the differences between

TABLE 6-5
COMPARISON OF THE PREVALENCE OF A BMI $>27 \mathrm{~kg} / \mathrm{m}^{2}$ BETWEEN HUTTERITES AND CANADIANS IN THE CANADA FITNESS SURVEY (CFS)* AND THE CANADA HEALTH SURVEY (CHS)**

| Age | CFS |  | CHS |  | Hutterites |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Male Female | Male <br> $\%$ | Female <br> $\%$ | Male <br> $\%$ | Female <br> $\%$ |  |
| $30-39$ | 27.1 | 15.3 | 31.9 | 19.4 | 48.7 | 34.5 |
| $40-49$ | 38.4 | 24.1 | 38.8 | 33.2 | 45.0 | 51.3 |
| $50-59$ | 38.3 | 32.7 | 42.5 | 41.7 | 55.7 | 70.0 |
| $60-69$ | 41.5 | 36.7 | 41.3 | 49.6 | 61.9 | 73.1 |

* Fitness Canada, 1981
** Health and Welfare Canada, 1981
the Hutterites and the participants in the CHS. A major problem with interpretation of the CHS data resulted from the grouping of extreme values into predetermined cutoff points. Both Canadian studies have also been criticized because of the possible bias introduced by selective noncompliance with anthropometric measures (Health and Welfare Canada, 1988b). Having taken these caveats into account, the prevalence of elevated BMI is considerably higher in the Hutterite study for all age groups and both sexes.

Table 6-6 compares the percentage of the Hutterites who are overweight (BMI >27.8 for males and $>27.3$ for females) with the U.S. white and black population (Gray, 1989). While Hutterite males display a steady increase with aging in the prevalence of being overweight, the proportion of overweight white and black males in the U.S. actually declines with aging. While the proportion of overweight females increases in all three groups of women with aging, the Hutterite and black U.S. females are twice as likely to be overweight than the U.S. white females in every age category. The increased prevalence of elevated BMI among Hutterite and Black females vis a vis White females may be due, in part, to their higher fertility rates (Ohlin and Rossner, 1989).

There are a number of possible explanations for why the prevalence of elevated BMI is so great among the Hutterites. The average caloric intake of Hutterites is high, above 2500 kcal/day (Rokusek-Kennedy et al, 1987). Physical exercise,

TABLE 6-6
COMPARISON OF THE PREVALENCE OF ELEVATED BMI* BETWEEN THE HUTTERITES AND THE U.S. WHITE AND BLACK POPULATIONS

| Age | U.S. White <br> $\%$ | Female <br> $\%$ | Male <br> $\%$ | Black <br> Female <br> $\%$ | Mutcterites <br> $\%$ |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| $35-44$ | 28.2 | 24.8 | 40.9 | 40.8 | 41.0 | 36.2 |
| $45-54$ | 30.5 | 29.8 | 41.4 | 61.2 | 42.5 | 61.5 |
| $55-64$ | 28.6 | 34.8 | 26.0 | 59.4 | 51.7 | 69.1 |
| $60-69$ | 25.8 | 36.5 | 26.4 | 60.8 | 63.9 | 85.7 |

* BMI > 27.8 for males, $>27.3$ for females
** Gray, 1989
both at work and during leisure-time were, found to be quite low in the present study. Thus, the imbalance of high caloric intake with low energy expenditure puts Hutterites at risk for insidious weight gain. The large number of pregnancies among Hutterite females has also been shown to increase the probability of obesity (Ohlin et al., 1989). The familial nature of obesity is also well known and probably results from an interaction of socio-behavioral and genetic factors (Venters and Mullis, 1984).
F. The Prevalence of Alcohol Consumption

While alcohol is widely used by the Hutterites, particularly as an aperitif, there was little evidence of widespread alcohol abuse. Hutterites produce their own beer and fortified wine, consumption of stronger spirits is less common. Almost all of the males (90.5\%) and just over half of the females (55.0\%) reported that they consume alcohol on a regular basis, although most of the women (69.1\%) drink once a week or less. Daily alcohol consumption was reported by $50.4 \%$ of the males and $13.4 \%$ of the females.

A similar proportion of males in the Canadian Health Promotion Survey (CHPS) (Health and Welfare Canada, 1988a) and the Hutterite study regularly consumed alcohol (86\% and 90.5\%, respectively), although a higher proportion of females in the CPHS than in the Hutterite study regularly consumed alcohol ( $77 \%$ vs. $55.0 \%$, respectively). In comparison to the Hutterites, daily consumption of alcohol
was reported by only $9 \%$ of the males, and by just $5 \%$ of the females in the CHPS. However, it must be remembered that the CHPS included all Canadians over the age of 15 , thus there may be an underestimation of daily alcohol consumption in Canadians 30-74. Just as found with the Hutterites, the CHPS found that Canadian males drink more frequently than females.

## G. The Prevalence of Psychological Stress

The bucolic Hutterite lifestyle does not preclude or prevent stress. Roughly a third of Hutterite males (29.8\%) and females (34.8\%) reported that they generally feel pressured and tense. At the other end of the spectrum, approximately half of the males (52.7\%) and females (47.4\%) reported that they were generally relaxed or calm. As described by Eaton and Weil (1953), Hutterite society is not free of neuroses, stress, or psychoses. However, the strong social support available to Hutterites, provides an ideal therapeutic environment for psychologic problems.

The distribution of psychological stress among the Hutterites differs from Canadians examined in the CHPS only at the extremes of the scale (Health and Welfare, 1988a). Almost one in ten Hutterites (9.7\%) describe their lives as relaxed and not at all stressful, this compares to just one in one hundred (1.3\%) in the CHPS. At the other end of the spectrum, only $2.3 \%$ of Hutterites describe their lives as extremely stressful, whereas almost 1 in ten (8.8\%) Canadians in the CHPS did so.

## H. The Prevalence of Physical Activity

Moderate to vigorous aerobic physical activity, either on the job or during leisure-time, was uncommon in the study population. It must be remembered that, while Hutterites are farmers, their farms are some of the most highly mechanized and efficient in the world and do not demand excessive physical labor. Hutterite adults do not usually engage in social activities that require exercise (Hostetler, 1974) and walking between buildings on the colony was the most frequent form of exercise reported.

While approximately half of the Hutterite males (52.3\%) and females (40.4\%) reported moderate to heavy exercise on the job, little of this work was aerobic in nature. Approximately a third of Hutterite males (34\%) and females ( $32.3 \%$ ) reported that they engage in regular leisure-time exercise, though just over half of these exercise for less than 15 minutes (51.2\%). By comparison, $54 \%$ of Canadians in the CHPS (Health and Welfare, 1988a) regularly exercise for 15 minutes or more.
I. The Prevalence of a Positive Family History of CHD

In light of the high coefficient of inbreeding reported among Hutterites, it is not surprizing that a family history of a relative with CHD is virtually ubiquitous in the Hutterite population. Just over two-thirds of the Hutterite males (68.8\%) and females (68.4\%) reported a family history of CHD. A family history of premature CHD (onset prior to age 55) was reported by $30.9 \%$ of the males and by $37.1 \%$ of
the females. A parental history of CHD was reported by $38.9 \%$ of the males and by $36.3 \%$ of the females in the study. A parental history of premature CHD was reported by $8.5 \%$ of the males and 11.3\% of the females.

The Hutterites had a higher prevalence of family history than found in other population studies. For example, the prevalence of a parental history of CHD among the Hutterite males was more than twice that reported in the Western Collaborative Group Study (WCGS) (Rosenman et al., 1975), $38.9 \%$ vs 18.3\%. Similarly, compared to women in the Nurses Health Study (Colditz et al., 1986), the Hutterite females had a higher prevalence of parental history of CHD, $36.3 \%$ vs. $26.5 \%$. Williams et al. (1988), using a complicated formula, have estimated that between $3.7 \%$ and $7.5 \%$ of the 24,332 families included in their study of "family trees' have a high-risk family history for CHD; this compares to just over a third of the Hutterites reporting a high risk family history of premature CHD.

# II. The Prevalence and Correlates of CHD Among the <br> Hutterites <br> A. The Prevalence of CHD 

As with hypertension, hypercholesterolemia, and diabetes, the prevalence (\%) of CHD varied by the definition used:

Criteria

1) Medical History alone
2) Rose Angina
3) Medical History and/or Rose Angina

Male Prevalence
4.4
5.9
8.5
4.8

The details of CHD prevalence are found in Chapter 5. Regardless of the definition used, males had a higher prevalence of CHD than females and the prevalence increased for both sexes with aging.

Only two studies were located that provided the prevalence of CHD based on medical history alone. However, there have been numerous examinations of the prevalence of Rose angina in populations. Based on the medical history prevalence rates, the Hutterites would not appear to have a higher prevalence of CHD than the comparison populations of the United States and Finland. Finland provides an interesting comparison population for the Hutterites as the incidence and mortality rate of CHD in Finland is one of the highest in the world (Statistics Canada, 1986). However, the
prevalence of Rose angina among male Hutterites was found to be consistently higher than in other North American studies.

Table 6-7 compares the prevalence of CHD by history and Rose angina in the Hutterites with other North American and western European studies. In both of the studies based on medical history (Kannel and Thom, 1986; Reunanen et al., 1983), Hutterite males have a lower prevalence of CHD, while Hutterite females are similar to the non-Hutterites. The reverse is true of the studies based on Rose angina (Lacroix, Haynes, Savage, and Havlik, 1989; Wilcosky, Harris, and Weissfeld, 1987). Hutterite males in both comparative studies have a higher prevalence of Rose angina than the Hutterite females, the non-Hutterite females, and the non-Hutterite males.

It should be noted that the physician-supplied verification rate for respondent-reported CHD was 66.7\%, thus it is possible that the prevalence of CHD in this population, however defined, is over-estimated.

In light of the high prevalence of hypertension, diabetes, and hypercholesterolemia, it is surprizing that the prevalence of medically diagnosed CHD is not higher in the Hutterite population. Then again, because of the relatively high prevalence of Rose angina among Hutterite males, a number of undiagnosed CHD cases may actually exist in the population. However, even taking into account the prevalence of CHD by medical history and/or Rose angina, there does not appear to be an excessive prevalence of CHD

TABLE 6-7
COMPARISON OF CHD PREVALENCE BETWEEN HUTTERITES, THE UNITED STATES POPULATION, AND THE POPULATION OF FINLAND

| Comparison Study <br> and Criteria Used Age Groups | Other Study <br> Prevalence <br> Male Female <br> $\%$ <br> $\%$ | Hutterite <br> $\%$ | Male Female <br> $\%$ |
| :--- | :--- | :--- | :--- |
| U. S. Population(1) <br> a) Medical history | $45-64$ | 7.6 | 3.4 |

## Social Insurance

## Institution(2)

a) Medical history

45-49

| $50-54$ | 8.7 | 3.4 | 3.0 | 5.9 |
| :--- | :--- | :--- | :--- | :--- |


| $55-59$ | 12.9 | 5.8 | 8.1 | 5.6 |
| :--- | :--- | :--- | :--- | :--- |

U.S. White Population(3)
a) Rose Angina

35-54
55-74
3.16 .1
$4.0 \quad 2.1$
6.97 .5
12.5
3.9
U.S. Black Population(4)
a) Rose Angina
35-54
7.3 6.8
4.0
2.1
55-74
6.08 .5
12.5
3.9

Lipids Research Clinic Program(5)

| $30-39$ | .8 | 4.0 | 2.0 | 2.4 |
| :--- | ---: | :--- | :--- | :--- |
| $40-49$ | 1.5 | 5.5 | 5.5 | 3.4 |
| $50-59$ | 5.0 | 5.5 | 5.7 | 1.4 |
| $60-69$ | 7.8 | 6.0 | 9.5 | 1.9 |

(1) Kannel et. al, 1986
(2) Reunanen et al., 1983
(3) Lacroix et al., 1989
(4) Lacroix et al., 1989
(5) Wilcosky et al., 1987
in the Hutterites studied.
There are a number of possible explanations for this finding. As the attributable risk for smoking in relation to CHD has been estimated to be as high as $30 \%$ (National Institutes of Health, 1977), the low prevalence of smoking among the Hutterites may significantly reduce their risk of developing CHD. There is good evidence that the Hutterites are at low risk for other smoking related diseases such as lung cancer (Gaudette et al., 1978).

The moderate consumption of alcohol among the Hutterites, in spite of its possible deleterious effects on blood pressure, may improve the HDL/LDI cholesterol fractions and provide some protection against atherosclerosis (Kris-Etherton et al., 1988). However, compared to salubrious effects of non-smoking, the protection afforded by moderate alcohol consumption may be quite small.

While the relationship between psychological stress, personality type, and CHD remains controversial, it is entirely possible that future research will help elucidate the effects of stress on CHD etiology. The strong social support and virtual lack of anomie found among the Hutterites may, in the future, prove to reduce CHD risk (Radley, 1984).

The relatively low prevalence of CHD found among the Hutterites may also result from a higher rate of sudden cardiovascular-related deaths. In other words, Hutterites
may have fewer prevalent cases of CHD because they have a higher case-fatality rate related to cardiac or vascular disease. The high prevalence of hypertension, such as found among the Hutterites, is associated with an increased risk of premature death from cardiac failure, arrhythmias and stroke (Castelli and Anderson, 1986). Because Hutterite colonies are generally far removed from specialized tertiary care settings, they may also be at a higher risk of dying during the critical first hours of acute myocardial infarction and stroke.

Because of the genetic isolation of Hutterites, they may have either increased genetic protection from CHD or', alternatively, they may have an increased susceptibility to sudden cardiac death. Schlenker et al. (1989) report that they have some unpublished evidence that the life expectancy of Hutterites is an average of ten years less than that of non-Hutterites. If their information is correct, it would support the hypothesis of an increased premature casefatality rate among the Hutterites. Further studies will be needed to assess whether, 1) Hutterites are indeed more prone to premature death, and 2) whether this is due to genetic and/or lifestyle factors.
B. The Correlates of CHD in the Hutterite population

After adjustment for possible confounding, there were more variables associated with CHD, regardless of how it was defined, for the males than the females. Hypertension, diabetes, and increasing age were associated with all three
operational definitions of CHD for the males. For the males, the inability to discriminate between CHD cases and non-cases in relation to family history, obesity, and hypercholesterolemia may be partially explained by their ubiquitous prevalence. As discussed above, and described in detail in Chapters Three and Four, over two-thirds of Hutterite males had a relative with CHD, almost threequarters of Hutterite males over the age of 45 had a BMI above $27 \mathrm{~kg} / \mathrm{m}^{2}$, and over half of the males over the age of 45 had a cholesterol above $6.20 \mathrm{mmol} / 1$. When the prevalence of a risk factor is so high, it is difficult to use statistical tests of risk. Conversely, the very low prevalence of smoking among males, less than $4 \%$, also effectively removes smoking as a risk factor in this population.

In contrast to the males, no variables were associated with CHD for all three definitions for the females. However, diabetes was associated with both Rose angina and the more broadly defined form of cHD for the females. The inability to discriminate between CHD cases and non-cases in relation to other factors may be due, in part, to the relatively small number of females with CHD , and, as for the males, due to the ubiquitous (or absent) prevalence of other CHD risk factors.

## IV Strengths and Limitations of the study

A. Strengths of the Study

A major strength of this study was the high rate of participation by individual Hutterites (88.7\%) from the colonies included in the investigation. Surrogate data related to the main end points of the study was gathered on all remaining participants eligible for inclusion in the study; thus data collection approached $100 \%$ for the calculation of prevalence of CHD, hypertension, diabetes and hypercholesterolemia. Fortunately, there was no evidence of selection bias: there was no difference in the prevalence of the main end points of the prevalence study between the respondents and the surrogates. While the colonies included in the study were not randomly selected, the fact that over half of all Dariusleut colonies in Alberta participated, strengthens the study.

The use of physician validation for self-reported cases of CHD, hypertension, diabetes, and hypercholesterolemia also strengthened the study design. The cooperation of the physicians, and the willingness of the respondents to have their medical histories verified, was quite good: of the 339 conditions requiring verification, $68.7 \%$ ( $n=233$ ) were ultimately verified. The concordance between respondentreported medical histories and physician-supplied verifications were above $90 \%$ for hypertension, diabetes, and hypercholesterolemia. However, physician verification for CHD was not as high (66.7\%). It is possible that respondents
have labelled themselves as having angina or myocardial infarcts because of other symptoms they erroneously believed indicated CHD e.g. heartburn, gall bladder disease, hiatal hernia etc.. Conversely, it is possible that some physicians were not aware of a previous diagnosis of CHD in some of the respondents.

The ascertainment of the prevalence of all major, and most minor, CHD risk factors also provided an important dimension to the study. The prevalence of CHD can be placed in context with other associated factors such as hypertension, hypercholesterolemia, diabetes, cigarette use, elevated BMI, alcohol consumption, psychological stress, physical activity, and family history. In addition, many of the risk factors were examined in multiple ways: by medical history, by physical and/or laboratory findings, by medication use, and by hospitalization. This comprehensive examination of $C H D$ and its risk factors will be particularly useful for generating future research (see below).

Providing the respondents and their physicians with the results of the physical and laboratory measures paid large dividends in terms of cooperation and credibility. It should be noted that initially only 26 colonies had agreed to participate in the study. The recruitment of an additional twelve colonies, for a total of 38 colonies, was greatly facilitated by "word of mouth" recommendations from colonies visited during the first few weeks of the study.

## B. Limitations of the Study

This study shared the two major weaknesses inherent in cross-sectional designs: 1) the difficulty in separating cause and effect and, 2) the loss of potential prevalent cases due to either a short duration of disease or death (Kelsey et al., 1986). Because of the large body of available literature regarding the causal pathways of CHD and risk factors such as hypertension and diabetes, and because the major purpose of this study was to examine association, not causality, the first weakness of crosssectional designs is not of great concern. However, there is the possibility that the apparently low prevalence of CHD among the Hutterites may, as discussed earlier, result from loss of prevalent cases through death. One way to ascertain whether there was a censoring bias due to death would be to conduct a mortality study of CHD-related deaths among the Hutterites. However, due to the relatively small number of Hutterites, this would necessarily need to be conducted over an extended period of time, e.g at least 20 years. It would be more feasible to conduct a retrospective review of death certificates for evidence of excessive CHD mortality rates. Ascertainment of CHD prevalence could have been done with greater precision if more vigorous case-finding and case-validating techniques had been utilized. For example, electrocardiograms (ECGs) at rest and/or with stress would have helped diagnose myocardial ischemia and infarction. Rather than relying on physician verification, office and
hospital records could have been carefully reviewed for references to medical treatment for ischemic heart disease.

Just as CHD prevalence could have been more vigorously examined, the same could be said for all of the associated CHD risk factors. It is possible that if blood pressures had been measured more often, or at different times of the day, the prevalence of hypertension could have been either higher, or lower (Health and Welfare Canada, 1989).

While a random total cholesterol was measured, it would have been desirable to have measured the total cholesterol, HDL, LDL, and apolipoprotein fractions in a fasting state. It must also be remembered that there is some evidence that measurement of capillary cholesterol may over-estimate serum cholesterol. In one study of 30 subjects, the simultaneous measurement of venous and capillary cholesterol found that capillary cholesterol was at least $5 \%$ greater than venous cholesterol in 7 of the subjects (Greenland, Sparks, Bomley, and Micklejohn, 1988). Similarly, either a fasting serum glucose or a glucose tolerance test would have more precisely measured and/or predicted diabetic status (American Diabetes Association, 1989).

While BMI was measured in the standard way, in light of recent interest in the distribution of body fat, it would have been useful to also measure the waist to hip ratio (WHR) (Health and Welfare Canada, 1988b).

Suffice it to say that all of the lifestyle-related CHD risk factors, e.g. cigarette smoking, alcohol consumption,
psychological stress, and diet could have been more intensively examined. Assuring accurate measurement and/or disclosure of lifestyle habits, on both self-administered and interviewer-administered questionnaires, remains a difficult task (McDowell and Newell, 1987).

Lastly, the difficulties associated with the measurement of a family history of disease are well known (Perkins, 1986). Even among the Hutterites, where family ties are close and life-long contact with relatives is the norm, knowledge of the health status of even the closest relatives is, in all likelihood, far from accurate. Ideally, the medical records and/or death certificates of relatives should be examined in order to verify reported family histories. While the logistics of such an indepth review of family records is fraught with difficulty, it is, at least theoretically, possible.

In summary, the weaknesses of the present study resulted largely from either practical or pecuniary considerations. However, even taking into account the problems outlined above, the study adequately met its objectives. For the first time there is a body of valuable information available for further hypothesis generation and health promotion planning in this unique population. Despite the relatively high prevalence of hypertension, hypercholesterolemia, elevated BMI, and diabetes, there is no good evidence that, in relation to other western populations, Hutterites suffer from an excessive prevalence
of CHD. Nevertheless, it must be remembered that hypertension, diabetes, and elevated BMI each significantly increase the risk of negative health outcomes other than CHD, e.g. renal failure, retinopathy, cerebrovascular disease, and joint degeneration. Future research will hopefully provide a more comprehensive picture of the factors that contribute to the health status of Hutterites.

## V. Directions for Further Research

Ideally the present study, integrated with many of the improvements and suggestions described below, would be replicated in another population of Hutterites. Such a: replication would provide the information necessary to plan and evaluate health promotional programming in the Hutterite population. CHD case finding in the replication study should employ electrocardiography and review of medical charts.

Perhaps the most pressing questions raised by this investigation relate to the high prevalence of hypertension among the Hutterites. It would be helpful to more intensively investigate the possible causes of hypertension in this population. While elevated BMI, and possibly alcohol consumption, contribute to the Hutterites' hypertension, there are undoubtedly other important factors worthy of investigation, e.g. diet, exercise patterns, water content, parity and genetics. In addition, there was good evidence that Hutterites are under-treated for hypertension; it would be helpful to better understand this
phenomenon in both the physiologic and socio-behavioral context.

The prevalence of ventricular hypertrophy and its sequelae should most certainly be explored in light of the hypertension found among the Hutterites. Such an investigation should use not only electrocardiography, but also the more sophisticated and precise technology of echocardiography (Savage and Devereux, 1981).

Future investigations should also utilize more precise and detailed measures of lipid profiles, e.g. cholesterol sub-fractions. The factors associated with hypercholesterolemia, specifically dietary intake of saturated fats and genetics, deserve greater attention in future studies.

While the higher than expected prevalence of noninsulin dependent diabetes mellitus (NIDDM) in this population may largely result from the high prevalence of elevated BMI, there was some evidence that insulin dependent diabetes mellitus (IDDM) may be more prevalent that expected. This phenomenon should be examined in more detail.

The high prevalence of elevated BMI found among the Hutterites can only be partially explained on the basis of diet; the present study did nothing to investigate the socio-behavioral aspects of elevated BMI: Impressions from the present study would suggest that Hutterites do not engage in aerobic exercise. It would be very useful to
conduct a detailed study of the energy expenditure and exercise physiology of the Hutterites to either confirm or refute this impression.

Because the measurements of stress and alcohol made in the study were not as detailed or as precise as they could be, more precise studies of stress and alcohol consumption among the Hutterites should also be conducted. Such a study could be integrated into a larger examination of the influence of socio-behavioral factors on Hutterite health.

Lastly, research studies related to health promotion among the Hutterites are necessary, particularly in light of the high prevalence of hypertension, diabetes, elevated BMI, and diabetes in this population. The cultural relevance of methods applied to other, non-Hutterite, health promotion programs could be examined. Such research would undoubtedly be challenging and could help improve the provision of health promotion programs to other culturally unique populations.

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## APPENOIX I <br> Prevalence of Heart Disease Study, 1988 <br> PERSONAL HEALTH SURVEY QUESTIONNAIRE

SECTION A: QUESTIONS ABOUT YOU
(1) What is your birthdate?
month $\overline{\text { day }}$
2) In which colony were you born? $\qquad$
(3) What is the name of your present colony? $\qquad$
(4) How long have you lived in this colony? $\qquad$ (years)
5) Are you: $\quad \square$ Male
(6) Are you now (checkonly one answer please):
$\square$ i single, never been married
$\square 2$ married
$\square 3$ widowed
$\square 4$ separated or divorced
(7) What is your height in

8) What is your weight in $\qquad$ or $\qquad$ pounds kilograms
9) What is your one major work responsibility in the colony? (check $r$ only one answer please):
$]_{1}$ minister, work boss, manager, head cook or teacher
$\square_{2}$ childcare
$\square 3$ :cooking or housekeeping
$\square]_{4}$ tending animals
—s gardening or general farm labor
$\square 6$ skilled work (carpenter, mechanic, electrician etc.)
$\square 7$ retired
$\square g$ other $\qquad$
(please specify)
10) What other kinds of work do you do? $\qquad$

SECTION B: QUESTIONS ABOUT YOUR LIFESTYLE AND HABITS

1) In the last year did you drink beer, wine, or spirits more than ence a month?
$\qquad$ I NO (please an on to question 2)
—_, YES (please answer Ouestions la and lb):
la) What do you usually drink (please check $\sqrt{\text { y }}$ yes or no)?


1b) On average, during the last year, how of ten have you taken at least one drink of wine, beer, or liquor? (check Jonly one answer please):


1 two or more times a day
2 once a day
$\square, 4$ to 6 times a week
${ }_{4} 2$ or 3 times a week
$\square$ s once a wenk or less
2) Have you ever regularly used tobacco products?
[. MO (please go on to Question 3 on the next page)
$\square]_{2}$ YES (please answer Questions 2a, 2b, 2c and 2d):

- 2a) Have you ever regularly used the following tobacco products? (please check $\sqrt{ }$ yes or no).

Cigarettes
Cigars
Pipe
Smokeless tobacco (i.e. chewing tobacco)

2b) What is your present tobacco habit? (check $\sqrt{ }$ only one answer please):
$\square 1$ I am still using tobacco regularly
2 I use tobacco only occasionally
3 quit using tobacco in the last year
4 I quit using tobacco 1 to 5 years ago
5 quit using tobacco more that 5 years ago

2c) How of ten did you regularly use tobacco?
(check $\sqrt{ }$ only one please):
[ $\square 1$ daily, more than 20 times a day
$\square 2$ daily, 5 to 20 times a day
daily, 1 to 5 times a day
$\square$ a few times a week
$1{ }_{5}$ once a month or less
2d) For how many vears did you regularly use tobacco products? (checkronly one please):
$\left.\square 1 \begin{array}{l}20 \text { years or mere } \\ 10 \text { to } 19 \text { years } \\ 2\end{array}\right)$
5 to 9 years
$\square 1$ to 4 years
$\square$ less than 1 year
3) In your regular diet how of ten do you have (check (only one answer for each food type):

| FREQUENCY |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
| FOOD TYPE | at least once $\underset{i}{\text { a day }}$ | $\begin{aligned} & \text { 4-6 times } \\ & \text { a week } \\ & 2 \end{aligned}$ | $\begin{aligned} & 2-3 \text { times } \\ & \text { a week } \end{aligned}$ | once a week or less. | ${ }_{5}$ |
| red meat |  |  |  |  |  |
| fish |  |  |  |  |  |
| chicken |  |  | . |  |  |
| goose or duck |  |  |  |  |  |
| bread |  |  |  |  |  |
| sweet taked goods |  |  |  |  |  |
| vegetables |  |  |  |  |  |
| fruit |  |  |  |  |  |
| whole grain cereals |  |  |  |  |  |
| margarine |  |  |  |  |  |
| butter |  |  |  |  |  |
| eggs |  |  |  |  |  |
| milk |  |  |  |  |  |
| cheese |  |  |  |  |  |
| fried foods |  |  |  |  |  |
| gravy |  |  |  |  |  |

4) "How often do you add salt to your food at the table? (check only one answer please):
$\square 1$ always
$\square 2$ usually
$\square 3$ sometimes
$\square 4$ hardly ever
$\square 5$ never
5) Which bf the following choices best describes the work or other activities you usually do? (check only one answer please): $\square 1$ I am usually sitting during the day and do not walk about very much
2 I stand or walk about quite a lot during the day, but I do not carry or lift things very often
3 I usually lift or carry light loads or I have to climb stairs or hills often
$\square 4$ I do heavy work or carry heavy loads
6) Do you regularly do physical exercises such as brisk walking durire ycur non-working time? (By regularly we mean at least once a week during the past n:enth):1 NO (please gn on to (question 1 in Section $C$ on this page)
$\square 2$ YES (please answer Ga, 6b and 6c):
6a) How much of this exercise is hard enough to cause: sweating or heavy breathing (checkVonly one please):
$\square 2$ most of it
2 some of it
3 nore of it

6b) How long do you usually exercise? (check
$\square$ I less than 15 minutes
$=15$ to 30 minutes
j 31 to 60 minutes
4 more than 60 minutes
6c) What do you do for physical exercise?

## SECTION C: MEDICATIONS AND USE OF HEALTH CARE

1) Are you regularly taking any medications (pills, ointments, drops, injections, syrups etc.) that are prescribed by a Ooctor?1 NO (please on go to Question 2 on the next page)
$\square 2$ YES (please answer Question la and 1b)
la) Please list the names of the medications prescribed by a Doctor that you are taking. Please also decribe what you are taking them for:
$\qquad$ WHAT IT IS FOR
2) 

$\qquad$
$\qquad$
2)
$\qquad$
4) $\qquad$
5) $\qquad$
$\qquad$
6) $\qquad$
$\qquad$
7) $\qquad$
$\qquad$
8) $\qquad$
9) $\qquad$
$\qquad$
10) $\qquad$

1b) Do you take any medications prescribed by a Doctor for any of the following health problems? (please check $\checkmark$ yes, no, or not sure):

| NO | YES | NOT SURE |  |
| :---: | :---: | :---: | :---: |
| $\square$ |  |  | heart trouble <br> A STROKE <br> HIGH BLOOD PRESSURE DIABETES |

2) Do you reqularly tike any medications that are not prescribed by a doctor? (Such as co!d remedios, linaments, herbal medicines etc.)
$\square 1$ Mn (flease go on to Question 3)
$\square_{2}$ YFS (please answer Question 2a)
2e) On you take any of following types of medications that are not prescribed by a Doctor? (flease check $\checkmark$ yes, no, or not sure):


ASPIRIN (headaches, other pains)
TYLENOI. (headaches, other pains)
LAXATIVES (move bowels)
vitamins
ANTACIDS (heartburn, stomach pain)
COLD OR COUGH REMEDIES
LINAMENTS (muscle or joint pain)
herral remedies $\qquad$
type of herbs
$\square \square$ OTHERS (please 1 ist) $\qquad$
3) Have you ever stayed overnight or longer in a hospital as a patient for $a$ health. problem (do not include normal childbirth)?
$\square$ NO (please go on to Question 4 on the next page)

| YES (please answer Questions 3 a and 3 b ) <br> 3a) For each hospital stay, please provide the following information: |  |  |  |
| :---: | :---: | :---: | :---: |
|  | THE REASON | THE YEAR | THE HOSPITAL |
| most recent stay |  |  |  |
| next most recent |  |  |  |
| next most recent |  |  |  |
| next most recent |  |  |  |
| next most recent |  |  |  |

3b) Have you ever stayed in the hospital for any of the following health problems (please checkyes, no, or not. sure):

heart trouble
A STROKE
HIGIf BLOOD PRESSURE
diabetes
4) Have you seer vour doctor in the last year for a health problem ? $\square: N O$ (olease go on to (luestion 5)

5) In general, compared to other persons your age would you say your health is: (please check $\sqrt{\text { only }}$ one answer)
$\square$ Excellent
$\square]_{2}$ Very food
$\square_{3}$ Good
$\square_{4}$ Fair${ }_{5}$ Poor

SECTION D: QUESTIONS ABOUT YOUR HEALTH AND YOUR FAMILY'S HEALTH

1) Hás a doctor ever said that you have (please check $\underline{\text { all }}$ that apply):
$\square$ Anemia (285.9)
Skin allergies (692.9)
Too much weight (278.0)
Kidney disease (586.0)Hay fever or other type of allergy (477.9)Stomach or duodenal ulcer (532.9) Thyroid trouble or goiter (240.9)Asthma (493.9)High Blood Pressure (400.9)Arthritis or Arthritis or
Rheumatism (716.9)

Migrane headaches (346.9)
Paralysis of any kind (344.9)Heart Trouble (414.0)
Cancer (239.9)
Epilepsy (345.9)Cerebral palsy (343.9)Emphysema or chronic bronchitis (492.8)Diabetes (250.0)
Stroke (436.0) Any emotional disorders (300.9)Parkinsons Disease (332.0)


Mental retardation (319.0)
Any other conditions
i.) Have you ever had amy pain or discomfort in vour chest?
—u in (onerase ofe en to (Uuestion 3)


YFS (please g̣o to Question 2a)
2a! Do you get this pain or discomfort when you walk uphill or when you walk in a hurry?1 NO (please go on to Question 4 on the next page)
$\square$, YES (please answer 2b, 2c, 2d, 2e, $2 f$ and 2g):

2b) Do you get it when you walk at an ordinary pace on level ground?NOYES

2c) When you get this pain or discomfort in your chest what do you do?STOP MOVINGSLOW DOWN3 COntinue at the same pace

2d) Does it go away when you stand still?1 NO2 YES

2e) How quickly does it go away?
$\qquad$ IN TEN MINUTES OR LESSin more than ten minutes
2f) Where do you get this pain or discomfort? Mark the place(s) with an ' $X$ ' on the picture:


2g) Have you ever had a severe pain across the front of your chest lasting for half an hour or more?


NO
$\square-]_{2}$ YES
3) What is your usual level of stress? (check Vonly one answer please)RELAXED, PARELY TENSE OR ANXIOUS2 MOSTLY CALMANXIOUS: TENSE 2 TO 3 TIMES A DAYTENSE: USUALLY FEEL RUSHEDE EXTREMELY TENSE
(4) Have your fanEllts or GRAHDPARENTS pver had any type of Heari Trouble? NO (please go on to Question 5)


YES (plpase answer Questions $4 a, 4 b, 4 c, 4 d, 4 e$ and $4 f ;:$
na) Did your FATHER (please check only one answer):
$\square$ never have either a heart attack or angina (shest pains)
$\square$ have either a heart attack or atigina before aoe 55
L. have either a heart attack or angina after turning 55
$\square$ I am not sure if he had a heart attack or angina
4b) Did your HOTHER (please check Jonly one answer):
[]: never have either a heart attack or angina (chest pains)
-, have elther a heart attack or angina before age 55
$\square$, have either a heart attack or angina after turning 55
[], I am not sure if she had a heart attack or angina
4c) Did your FATHER'S FATHER (please check. only one answer): ? haver have either a heart attack or angina before age 55 ?, have either a heart attack or angina after turning 55 - I am not sure if he had a heart attack or angina

4d) Did your FATHER'S MOTHER (please check. Only one answer): $\square 1$ never have either a heart attack or angina (chest pains) —: have either a heart attack or angina before age 55 have either a heart attack or angina after turnina 55 $\square$ I am not sure if she had a heart attack or angina
4e) Did your MOTHER'S FATHER (please check Vonly one answer): $\square 1$ never have either a heart attack or angina (chest pains) I2. have either a heart attack or angina before age 55 have either a heart attack or angina after turning 55 [. 1 am not sure if he had a heart attack or angina
4f) Oid your MOTHER'S MOTHER (please checkioniv one answer): $\square 1$ never have either a heart attack or angina (chest pains) 2 have either a heart attack or angina before age 55 ,' have either a heart attack or angina after turning 55 . I am not sure if she had a heart attack or angina
5) Haixe any of your 8ROTHERS or SISTERS ever had a heart attack or angina (chest pains):
$\square: N O$ (please go on to Question 6)
12 YES (please answer Questions 5a, 5b, 5c and 5d):
5a) How many 8ROTHERS (including both living and dead) do you have?
5b) How many of your BROTHERS ever had a heart attack or angina before age 55? $\qquad$ , after turning 55?
5c) How many SISTERS (including both living and dead) do you have?
5d) How many of your SISTERS ever had a heart attack or angina before age 55? $\qquad$ , after turning 55?
6) Have any of your AUNTS OR UNCLES ever hod a heart attack or angina?I 110 (please go on to Question 7 on the next page)
$\square \square_{2}$ YES (please answer Questions 6a and 6b):
6a) How many AUNTS/UHCLES (including both living and dead) do you have? AUNTS: $\qquad$ , UNCLES:

6b) How many of your AUITS/UACLES ever had a heart attack or angina before age 55? $\qquad$ , after turning 55? $\qquad$
7) Has a doctor ever told you that you have any of the following health problems? (pleasercheck yes, no, or not sure):

heart trouble A stroke
borderline or actual high blood pressure BORDERLINE OR ACTUAL DIABETES
TOO MUCH CHOLESTEROL (FAT) IN YOUR BLOOD

If you checked "not sure" or "yes" to even one of the health problems in question 7, then please go on to the next section of the questionnaire.

BUT
IF YOU Checked "no" to every one of the health problems in question 7 , then you are finished with this questionnaire. thank you for four COOPERATION
please go on to page 10 If you think you may have had any of the gollowing: any type of heart trouble, or

- had A STROKE, OR

HIGH BLOOD PRESSURE, OR
DIABETES, OR
TOO MUCH CHOLESTEROL IN YOUR BLOOD

SECTION E: QUESTIONS ABOUT YOUR RISK FACTORS FOR HEART DISEASE

1) Have you ever been treated by a doctor for HEART TROUBLE? $\mathrm{I}_{1}$ NO (please go on to Question 2 on the next page)
$\square 2$ YES (please answer 1a, $1 \mathrm{~b}, \mathrm{lc}$, and 1 d ):
1a) What type(s) of HEART TROUBLE do you have? How long have


1b) Were you ever hospitalized for HEART TROUBLE?


1c) Have you ever had (please check/yes, no, or not sure):

heart surgery to repair valves
heart bypass surgery
Angioplasty, "Clogged arteries opened WITH A BALLOON" medications prescribed for heart trouble my heart trouble has never been treated WITH ANYTHING

1d) Are you now taking any medications for HEART TROUBLE?

a) Have you ever been treated by a doctor for A STROKE (blood clots or bleeding ir the brain) ?
[1 MO (please go on to OUestion 3)
[_1, YES (please answer 2a, 2b, 2c, and 2d):
(i.a) llive you ever had: (please check yes, ne, or not sure)

| ${ }_{1}$ | YES | $\begin{aligned} & \text { NOT } \\ & \text { SURE } \\ & \hline \end{aligned}$ |  |
| :---: | :---: | :---: | :---: |
|  | $\square$ | $\ddot{\square}$ | medications prescribed to prevent or <br> treat a stroke <br> surgery to treat or prevent a stroke nothing has been used to treat or PREVEHT MY STROKE |

2b) Has your STROKE left you with (please check fyes, no, or not sure):


PROBLEMS WITH TALKING OR UNDERSTAHDIMG OTHERS
PARALYSIS OR WEAKNESS III ARMS OR HAROS PARALYSIS OR WEAKNESS IN LEGS OR FEET ANY OTHER OISABILITY?
(please specify)
2c) Have you ever been hospitalized for problems resulting from a STROKE?
$\square 2: \begin{array}{ll}\mathrm{NO} \\ 2\end{array}$
2d) Are you now taking any pills to help treat or prevent a STROKE?
$\square 1$ YO
3). Have you ever been told by a doctor that you have DIABETES (sugar in - your urine or eleyated sugar in the blood)?
$\square_{1}$ NO (please go on to Question 4 on the next page)
$\square 2$ YES (please answer 3a, 3b, 3c, 3d, and 3e):
3a) How long have you had DIABETES? $\qquad$ (in years)

- 3b) Has your DIABETES ever been treated by a doctor with (please check yes, no, or not sure):


A DIET
PILLS
PILLS
IMSULIN INJECTIONS

3c) Are you now on a diet to treat DIABETES?


3d) Are you now taking injections of insulin?
$\square: 10$
3e) Are you now taking pills for olapetes?
$\square:$ YO
4) Have vou ever been told by a doctor that you have HIGH BLOOD PRESSURE (Hypertension) ?
—I NO (please go on to question 5)
[]2 YES (please answer 4a, 4b, 4c and 4d):

- 4) How long have you had HIGIl BLOOD PRESSURE? $\qquad$ (in years)
- 4b) Has your HIGH BL000 PRESSURE ever been treated with the following (please check $\sqrt{\text { yes, }}$ no, or not sure):


A DOCTOR'S ADVICE TO LOSE WEIGHT A DOCTOR'S ADYICE TO USE A LOW SALT OIET PILLS PRESCRIBED BY A DOCTOR NOTHING HAS BEEN DONE TO TREAT MY BLOOD PRESSURE

- 4c) Is your HIGH BLOOD PRESSURE now being treated with (please check yes, no, or not sure):


A DIET TO LOSE WEIGHT a LOW SALT DIET
PILLS PRESCRIBED BY A DOCTOR NOTHING IS 8EING DONE TO TREAT MY BLOOD PRESSURE

4d) Are you presently taking any pills prescribed by your doctor to treat HIGH BLOOD PRESSURE?
$\square 1$ YO $\square_{2}$ YES
5) Have you ever been told by a doctor that you have too much CHOLESTEROL (fat) IM YOUR BLOOD ?
$\square$ NO (You are now done with the Questionnaire, thank you.)
$\square 2$ YES (please answer 5a, 5b, 5c and 5d):
5a) How long have you known that you have too much CHOLESTEROL in your blood? $\qquad$ (in years)

5b) Has your CHOLESTEROL ever been treated with (please check yes, no, or not sure):


A DIET
PILLS
NOTHING HAS BEEN OONE TO TREAT MY CHOLESTEROL

5c) Are you now on a diet to control your CHOLESTEROL?


H0
2 YES
5d) Are you now taking pilts prescribed by a doctor for Cholesterot?


THANK YOU, YOU ARE NOW DONE WITH THIS QUESTIONNAIRE.

## APPENDIX II <br> HISTOGRAM OF DIASTOLIC PRESSURE (DBP) WITH NORMAL CURVE SUPERIMPOSED*

COUNT DBp ( mm Hg )

| 1 | 58 | * |
| :---: | :---: | :---: |
| 4 | 62 | ** |
| 12 | 66 | ****** |
| 35 | 70 | ***************** |
| 29 | 74 | *************** |
| 43 | 78 | ********************** |
| 90 | 82 | ******************************************** |
| 92 | 86 | ********************************************* |
| 82 | 90 | ***************************************** |
| 62 | 94 | ******************************* |
| 57 | 98 | ***************************** |
| 60 | 102 | ********************** |
| 27 | 106 | ************** |
| 37 | 110 | ***************** |
| 3 | 114 | ** |
| 3 | 118 |  |
| 1 | 122 | 1 |
| 0 | 126 |  |
| 0 | 130 |  |
| 1 | 134 | * |
| 1 | 138 | * |
|  |  | I....t....I....t....I....t....I....t....I....t |
|  |  | 0206000 |

[^4]
## APPENDIX III <br> HISTOGRAM OF SYSTOLIC PRESSURE (SBP) WITH NORMAL CURVE SUPERIMPOSED*

| COUNT | $\mathrm{SBp}(\mathrm{mm} \mathrm{Hg})$ |  |  |  |
| :---: | :---: | :---: | :---: | :---: |
| 5 | 93 | * |  |  |
| 18 | 100 | **** |  |  |
| 36 | 107 | ********. |  |  |
| 47 | 114 | ************ |  |  |
| 104 | 121 | ************************ |  |  |
| 117 | 128 | *************************** |  |  |
| 54 | 135 | ************** |  |  |
| 79 | 142 | ******************* |  |  |
| 68 | 149 | ****************. |  |  |
| 31 | 156 | ******** |  |  |
| 29 | 163 | ******* |  |  |
| 22 | 170 | ****** |  | . |
| 11 | 177 | **. |  |  |
| 7 | 184 | :* |  |  |
| 5 | 191 | : |  |  |
| 3 | 198 | * |  |  |
| 0 | 205 |  |  |  |
| 1 | 212 |  |  |  |
| 2 | 219 | * |  |  |
| 0 | 226 |  |  |  |
| 1 | 233 |  |  |  |
|  | I....+....I....+....I....t....I....t.....I....t.....I |  |  |  |
|  |  | 04080 | 160 | 200 |
|  | HISTOGRAM FREQUENCY |  |  |  |

* Histogram Generated by SPSS-X

One Symbol Equals Approximately Four Occurences $\mathrm{N}=788$

## APPENDIX IV <br> HISTOGRAM OF RANDOM GLUCOSE WITH NORMAL CURVE SUPERIMPOSED*



* Histogram Generated by SPSS-X

One Symbol Equals Approximately Four Occurences
$N=558$

## APPENDIX V <br> HISTOGRAM OF. BLOOD CHOLESTEROL WITH NORMAL CURVE SUPERIMPOSED*

COUNT Cholesterol (mmol/l)


* Histogram Generated by SPSS-X

One Symbol Equals Approximately Four Occurences
$\mathrm{N}=750$

## APPENDIX VI <br> HISTOGRAM OF BODY MASS INDEX (BMI) WITH NORMAL CURVE SUPERIMPOSED*



* Histogram Generated by SPSS-X

One Symbol Equals Approximately Four Occurences
$N=784$


[^0]:    * an average of butter, milk, and cheese

[^1]:    * Includes those reporting angina, myocardial infarct, bypass, and/or angioplasty

[^2]:    * a single respondent may be counted for more than one condition
    ** myocardial infarct, angina, coronary bypass and/or angioplasty

[^3]:    a) only variables significant for either males or females are included in the Table. (See text for non-significant variables)
    b) CHD by history and/or Rose Angina

[^4]:    * Histogram Generated by SpSS-X

    One Symbol Equals Approximately Two Occurences
    $N=788$

