THE AUCKLAND HEART STUDY:
A Case-Control Study Of Coronary Heart Disease

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SUMMARY

Coronary heart disease is the leading cause of death and a major cause of morbidity in New Zealand. Although the mortality rates are now declining, they remain high by international standards and there is considerable scope for the prevention of coronary heart disease. There is a paucity of New Zealand data on the aetiology of this disease.

The Auckland Heart Study is a case-control study of coronary heart disease which was designed to determine whether a range of variables including; smoking, passive smoking, hypertension, exercise, alcohol, serum lipids, and dietary and psychosocial factors, are related to coronary heart disease in New Zealand. Other factors examined included respiratory infection, sex hormones and serum vitamins. The study also documented the prevalence of the major risk factors in the Auckland adult population so as to examine trends in risk factors since the 1982 Auckland Risk Factor Study.

The study was conducted between 1 March 1986 and 3 May 1988. There were two case groups: non fatal myocardial infarction cases and coronary death cases; and two control groups: myocardial infarction controls and coronary death controls. Participants were aged 25-64 years.

An ongoing register of cardiovascular disease, the Auckland Region Coronary or Stroke (ARCOS) Study provided the cases for the study. Controls for the myocardial infarction cases were randomly selected from the 22 central Auckland general electoral rolls and group matched to the

cases by age and sex. Approximately one control was selected for each male case and two for each female case. Both myocardial infarction cases and controls were interviewed in one study centre by the same group of interviewers.

Coronary death controls were initially selected directly from the electoral rolls in an identical fashion to the myocardial infarction controls. They were asked if a close friend or relative could be interviewed about them because information on coronary death cases came from a similar source. Three months into the study the procedure was changed because of low response rates. Myocardial infarction controls were asked, at the time of their interviews, if the study team could also interview one of their close friends or relatives at a later date. Therefore two sets of data were collected on the myocardial infarction controls, one set directly from the control and a second set from a close friend or relative (as coronary death controls).

A total of 2,496 people participated in the Auckland Heart Study. 2,301 Pakeha (i.e., non Maori and non Pacific Island Polynesian) participants were available for the analyses. The response rates in the Pakeha group were 80%(n=561) and 85%(n=379) in male and female myocardial infarction controls respectively. In myocardial infarction cases they were 90%(n=359) in men and 76%(n=122) in women. For coronary death controls the response rates were 68%(n=355) in men and 66%(n=226) in women, and for coronary death cases the rates were 83%(n=249) in men and 79%(n=50) in women.

Cigarette smoking is a major risk factor for CHD in Auckland. In both men and women, smokers had three to four times the risk of myocardial infarction and coronary death as never smokers. There was a dose response effect with people smoking over 30 cigarettes per day having 8-14 times the risk of CHD as never smokers. It was estimated that approximately 25% of fatal and non fatal CHD events in New Zealand are attributable to cigarette smoking. There has been a 22% decline in smoking prevalence in men aged 40-64 years and a 9% decline in women in the same age group between 1982 and 1986/8.

Exposure to passive smoking was associated with an increased risk of myocardial infarction and coronary death in both men and women. Women exposed to passive smoking at home had four times the risk of CHD as those who were not exposed. Passive smoking exposure at work was associated with a 50% increase in CHD risk in men. Approximately 38% of never smoking men and 36% of never smoking women in this study reported that they were regularly exposed to passive smoking at home or work.

Hypertension was a major risk factor for both myocardial infarction and coronary death in this study. Using antihypertensive drug treatment as a proxy measure, hypertension was associated with a two to four fold increase in CHD risk. In 1986/8 21.4% of men and 20.4% of women aged 40-64 years were defined as hypertensive compared with 25.8% of men and 23.2% of women in 1982. This change was mainly due to a reduction in the number of people on drug treatment as there was no consistent change in mean blood pressure levels over the five year period. Approximately

15% of women and 10% of men aged 40-64 years were on regular antihypertensive drug therapy at the time of the Auckland Heart Study.

In this study the well described beneficial association between leisure time physical activity (LTPA) and CHD was found to be markedly weakened after adjusting for potential confounding, in particular cigarette smoking and, unexpectedly, alcohol consumption. People taking regular exercise were more likely to be light drinkers and less likely to be never drinkers than their sedentary counterparts. No consistent difference in benefit was found between vigorous and moderate LTPA.

This study shows a strong and consistent beneficial effect of alcohol consumption on both fatal and non fatal CHD risk. After controlling for possible confounding, people who drank alcohol more than once per month, had approximately a 30-60% reduction in CHD risk. The effect could not be explained by people with medical conditions reducing their alcohol consumption. Alcohol consumption was also shown to acutely reduce the risk of CHD. Among people who drank at least once per month, CHD risk was halved during the 24 hours after a drinking episode.

Finally, the expected associations between serum total, HDL and LDL cholesterol and CHD risk were also found in this study. HDL cholesterol levels in women were 25-30% higher than in men, and this difference was considerably greater than case-control differences in HDL. There were no other major sex differences in the main CHD risk factors examined and the strengths of association between risk factors and CHD were in general similar between sexes.

The Auckland Heart Study has provided comprehensive information on the aetiology of CHD in a large community based population sample for the first time in New Zealand. This thesis has examined only part of the extensive data collected and analyses are expected to continue for several years.

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