CORONARY HEART DISEASE IN HIGHLAND: FUTURE IMPACT

What we have:
What we can do:
Where we will be in 10-20 years time

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Working with you to make Highland the healthy place to be
Front Cover

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[http://www.show.scot.nhs.uk/hhb](http://www.show.scot.nhs.uk/hhb)
Coronary Heart Disease in Highland:

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Coronary heart disease in Highland: What we have; What we can do; Where we will be in 10-20 years time

Introduction

Coronary heart disease (CHD) is a major cause of death and ill health. It affects over 3% of the population in Scotland (approx. 6,000 in Highland) and accounts for over 20% of all deaths. Mortality from CHD in Highland as nationally, has been decreasing year by year since the 1970s in both men (30%) and women (20%). However this trend in mortality does not inform us of the relative impact of better management or avoidance of the condition, and the incidence of other life-threatening degenerative diseases in older age, particularly cancer. The relative impact of each of these in terms of current burden of disease is difficult to assess but predictions in rates in the next decade or so can be made by application of various scenarios in terms of expected changes in known risk factors and adoption of effective management regimens. These scenarios will involve predictions in demographic changes relevant to CHD and for other life-threatening conditions. Of these, ageing will be the most important single factor but an additional affect by migration as people transfer their health-risk legacy to their new location, will not be included.

Predicting the future burden of CHD will include applying the evidence-base for the various interventions at the three levels of intervention viz. (i) primary prevention, (ii) acute phase management and (iii) secondary prevention to ascertain the relative potential for improvement on the current situation. The impact of other variants on the burden of CHD of demography, lifestyles, socio-economic environment and development of other more effective interventions will also be taken into account.

It is proposed that the 3 levels of intervention are imposed on these variants and the resultant scenarios described in terms of: status quo, possible and best. These scenarios will be based on the following:

<table>
<thead>
<tr>
<th>Status quo</th>
<th>Demographic changes only</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible</td>
<td>Project historical trends plus demographic changes</td>
</tr>
<tr>
<td>Best</td>
<td>Possible plus any increased rates of known interventions at the acute &amp; secondary prevention stages plus disproportionate changes in modifiable risk factors in primary prevention plus speculations about any future developments in CHD management</td>
</tr>
</tbody>
</table>

The methodology employed in these predictions is set out in the appendix. This report includes the following sections:

1. The conditions included in the model and their main characteristics
2. The influence of demography and the expected demographic changes on CHD
3. The contribution of risk factors.
4. Establishing trends in mortality, morbidity, risk factors
5. Potential effectiveness of interventions at the 3 levels of intervention i.e. contribution to reduction in mortality and morbidity
6. Establishing current intervention rates; comparison with national averages or recommendations
7. Conclusions
1. The conditions included in the model and their main characteristics

Coronary Heart Disease (CHD) and its various manifestations

The main conditions included in coronary heart disease are Acute Myocardial Infarction (AMI); Angina Pectoris and Chronic Heart Failure. Simplistically they represent the challenges of acute, secondary and chronic care interventions respectively. Effective primary prevention by reducing the incidence of AMI and angina should influence the burden posed by heart failure. The following briefly describes each of these three conditions:

**Acute Myocardial Infarction**

Most people who die of CHD do so as a result of acute myocardial infarction (AMI) or as, more commonly known, a heart attack. During a heart attack the muscle wall of the heart is deprived of oxygen and as a result loses functionality. This can lead to uncontrolled beating of the heart (ventricular fibrillation) which prevents blood being circulated around the body. This is the usual cause of death. The average mortality within 30 days of a heart attack is 50% with half of these deaths occurring before any medical attention is received.

The clinical definition given by WHO is at least two of the following triad:

1. A history of typical chest pain of over 20 minutes duration
2. ECG changes showing development of Q waves, bundle branch block or ST segment elevation or depression of at least 0.1mV lasting for at least 24 hours
3. Increased activity of cardiac enzymes (i.e. lactic dehydrogenase, aspartate transaminase, or creatine kinase total) to more than 50% above upper normal limits

Although this is the current WHO definition, diagnosis of MI involving the measurement of blood concentrations of the cardiac proteins, Tropinin I or T, is increasingly being made by clinicians. The American College of Cardiology together with the European Society of Cardiology (ACC/ESC) has published a new definition of MI in which preference for using Troponin I or T above other cardiac markers is given. The implication of this new diagnostic definition is addressed in section 5, page 25.

The International Classification of Diseases code this condition under 410 (ICD-9) and I21-22 (ICD-10) These codes are used to determine mortality from AMI and hospital admissions for AMI. Sometimes there are AMI events and deaths in hospital not relating to the main cause for admission.

**Angina Pectoris**

This is the name given to recurrent chest pain occurring as a result of CHD in which there is narrowing of the coronary arteries. The pain arises because the heart muscle does not receive enough blood to meet its oxygen demands. The incidence of chest pain is usually greater when the heart’s oxygen demands are high such as during physical exercise and in cold weather.
Angina can be either chronic stable or unstable. The clinical definitions of both these conditions are based on the characteristics of the pain associated with ischaemia. Whilst in the stable condition, the pain is ameliorated during rest, in the unstable condition, there would be at least one of the following pertaining:

1. Effort angina of recent onset (less than one month) with no previous angina
2. Changing pattern of pre-existing stable angina with increase in frequency and/or severity
3. Angina at rest for no obvious reason

Unstable angina is a medical emergency.

The International Classification of Diseases code this condition under 413 (ICD-9) and I20 (ICD-10)

**Heart Failure**

Heart failure is a failure of the heart as a pump. It has multiple causes but the main one is CHD. Hypertension is a contributing factor in one third of cases. It can be acute or chronic, the latter representing approximately 75% of all cases. The main symptoms are breathlessness, fatigue, peripheral oedema and elevated jugular venous pressure, all as a result of ventricular dysfunction.

The International Classification of Diseases code this condition under 428 (ICD-9) and I50 (ICD-10).

In addition to these, Ischaemic Heart Disease (IHD), with its definition of ICD9 410-414 and ICD10 20-25, is used for the prediction of CHD in this report. CHD and IHD are often used interchangeably but data are more consistently defined when labelled IHD than is the category of CHD.
2. The influence of demography and the expected demographic changes on Coronary Heart Disease

The distribution of CHD in the population is associated with age, sex, geography, socio-economic status and ethnicity. In relation to Highland in the next decade it is the age and sex structure of the future population which will have the most influence on the burden from CHD. The relative contribution of CHD within each of four main age bands clearly demonstrates its increasing importance with age (figure 1).

Figure 1: Proportion of total deaths from selected causes within age-bands of the population of Highland in 2000

<table>
<thead>
<tr>
<th>Age Band</th>
<th>Heart Disease &amp; stroke</th>
<th>Cancer</th>
<th>Accident/injuries/suicides</th>
<th>Respiratory</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-44</td>
<td>30%</td>
<td>40%</td>
<td>20%</td>
<td>10%</td>
<td>20%</td>
</tr>
<tr>
<td>45-74</td>
<td>40%</td>
<td>50%</td>
<td>30%</td>
<td>20%</td>
<td>20%</td>
</tr>
<tr>
<td>75 &amp; over</td>
<td>50%</td>
<td>60%</td>
<td>40%</td>
<td>30%</td>
<td>20%</td>
</tr>
</tbody>
</table>

Data source: GRO

Mortality rates from IHD increase exponentially with age in both sexes, but the rate in males is higher. On average men develop IHD 10 years earlier but the excess rates in males diminish at late middle age when females experience almost the same rates. The difference is attributed to the protective role of female hormones which diminishes at the menopause (figure 2).
Figure 2: Age-specific mortality rates by gender from IHD: Highland residents based on deaths during 1997 to 2001

Data source: GRO mortality files and mid-year population final revised Feb 2003

A similar pattern also applies for Heart Failure but with the average age of onset older and the relative rates in males and females reversed in the oldest age band compared to IHD. As Heart Failure is mainly a chronic condition and in the majority of cases, a consequence of IHD, this pattern would be expected. The number of annual deaths in Highland caused by IHD and Heart Failure for those aged under and over 75 years are (table 1):

Table 1: Number of annual deaths and (% of all causes) in those aged under 75 years and those aged 75 and over: Highland 2001

<table>
<thead>
<tr>
<th></th>
<th>males</th>
<th></th>
<th></th>
<th>females</th>
<th></th>
<th></th>
<th>both</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nos</td>
<td>(%)</td>
<td>Nos</td>
<td>(%)</td>
<td>Nos</td>
<td>(%)</td>
<td></td>
</tr>
<tr>
<td>IHD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;75 yrs</td>
<td>146</td>
<td>(24%)</td>
<td>58</td>
<td>(15%)</td>
<td>204</td>
<td>(21%)</td>
<td></td>
</tr>
<tr>
<td>75 &amp; over</td>
<td>139</td>
<td>(25%)</td>
<td>165</td>
<td>(19%)</td>
<td>304</td>
<td>(21%)</td>
<td></td>
</tr>
<tr>
<td>HF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;75 yrs</td>
<td>1</td>
<td>(0%)</td>
<td>1</td>
<td>(0%)</td>
<td>2</td>
<td>(0%)</td>
<td></td>
</tr>
<tr>
<td>75 &amp; over</td>
<td>6</td>
<td>(1%)</td>
<td>15</td>
<td>(2%)</td>
<td>21</td>
<td>(1%)</td>
<td></td>
</tr>
<tr>
<td>All causes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;75 yrs</td>
<td>615</td>
<td>(100%)</td>
<td>375</td>
<td>(100%)</td>
<td>990</td>
<td>(100%)</td>
<td></td>
</tr>
<tr>
<td>75 &amp; over</td>
<td>565</td>
<td>(100%)</td>
<td>867</td>
<td>(100%)</td>
<td>1423</td>
<td>(100%)</td>
<td></td>
</tr>
</tbody>
</table>

Data Source: Skipper, ISD

Therefore, in forecasting the burden of CHD in the next 10-20 years, age-sex specific rates applied to projected populations are the basic predictors. For Highland, other fixed risk factors such as socio-economic status and geography (East/West Scottish gradient) is difficult to evaluate although for predicting the future burden, they are likely to be of smaller significance to that of the future age and sex population structural changes.
Rates of new annual cases, prevalence and deaths expected in 2010 and 2016 are presented in table 2 by application of current rates to the projected population. It is important to take note of the sources of these rates as morbidity estimates are variable. Unlike cancer, there is no national register for CHD. Although mortality may appear to be a more reliable indicator of the burden of IHD, at increased age, co-morbid conditions can make this a more uncertain underlying cause of death.

Table 2: Predicted numbers in Highland to be affected by CHD based on population numbers and structural change only

<table>
<thead>
<tr>
<th>Year</th>
<th>AMI</th>
<th>Angina</th>
<th>Other IHD</th>
<th>All IHD</th>
<th>Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong>¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base year 1998/2000</td>
<td>326</td>
<td>231</td>
<td>68</td>
<td>625</td>
<td>251</td>
</tr>
<tr>
<td>2010</td>
<td>383</td>
<td>270</td>
<td>81</td>
<td>734</td>
<td>305</td>
</tr>
<tr>
<td>2016</td>
<td>422</td>
<td>295</td>
<td>84</td>
<td>801</td>
<td>348</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>+17%</td>
<td>+17%</td>
<td>+19%</td>
<td>+17%</td>
<td>+22%</td>
</tr>
<tr>
<td>% change to 2016</td>
<td>+29%</td>
<td>+28%</td>
<td>+24%</td>
<td>+28%</td>
<td>+39%</td>
</tr>
<tr>
<td><strong>Incidence</strong>²</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base year 2000</td>
<td>148a-455b</td>
<td>2268</td>
<td>170-480</td>
<td>2898</td>
<td>819</td>
</tr>
<tr>
<td>2010</td>
<td>152a-469b</td>
<td>2630</td>
<td>267-584</td>
<td>3366</td>
<td>964</td>
</tr>
<tr>
<td>2016</td>
<td>145a-469b</td>
<td>2862</td>
<td>339-663</td>
<td>3670</td>
<td>1096</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>+3%</td>
<td>+16%</td>
<td>+22-57%</td>
<td>+16%</td>
<td>+18%</td>
</tr>
<tr>
<td>% change to 2016</td>
<td>0-+3%</td>
<td>-+26%</td>
<td>+38-99%</td>
<td>+27%</td>
<td>+34%</td>
</tr>
<tr>
<td><strong>Prevalence</strong>³</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base year 2000</td>
<td>-</td>
<td>3179</td>
<td>2168</td>
<td>5347</td>
<td>1515</td>
</tr>
<tr>
<td>2010</td>
<td>-</td>
<td>3693</td>
<td>2545</td>
<td>6238</td>
<td>1780</td>
</tr>
<tr>
<td>2016</td>
<td>-</td>
<td>4041</td>
<td>2824</td>
<td>6865</td>
<td>2027</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>-</td>
<td>+16%</td>
<td>+17%</td>
<td>+17%</td>
<td>+17%</td>
</tr>
<tr>
<td>% change to 2016</td>
<td>-</td>
<td>+27%</td>
<td>+30%</td>
<td>+28%</td>
<td>+34%</td>
</tr>
<tr>
<td><strong>Deaths</strong>⁴</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base year 1998/2000</td>
<td>283</td>
<td>0</td>
<td>218</td>
<td>501</td>
<td>40</td>
</tr>
<tr>
<td>2010</td>
<td>345</td>
<td>0</td>
<td>263</td>
<td>608</td>
<td>49</td>
</tr>
<tr>
<td>2016</td>
<td>393</td>
<td>0</td>
<td>298</td>
<td>691</td>
<td>57</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>+22%</td>
<td>-</td>
<td>-21%</td>
<td>+21%</td>
<td>+23%</td>
</tr>
<tr>
<td>% change to 2016</td>
<td>+39%</td>
<td>-</td>
<td>+37%</td>
<td>+38%</td>
<td>+42%</td>
</tr>
</tbody>
</table>

¹hospital-linked new admission rates (ISD); ²CMR rates except for AMI (ISD); ³AMI rates based on either OXMIS rates or MONICA Glasgow rates; ⁴Mortality data from GRO
Base year estimates from rates applied to mid-year population estimates revised for 2001 census (GRO)
Predicted estimates from base year rates applied to 1998-based projected population, not adjusted for 2001 census (GRO)
3. Contribution of risk factors

Risk factors for IHD can be categorised as either fixed, modifiable or dependent (consequential) of other conditions (table 3).

Table 3: Risk factors for Ischaemic heart disease

<table>
<thead>
<tr>
<th>Type of risk</th>
<th>Higher risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed</td>
<td>Age, Male gender, Family History, Ethnicity</td>
</tr>
<tr>
<td>Modifiable</td>
<td>Smoking, Serum Cholesterol, Blood Pressure, Obesity, Physical inactivity</td>
</tr>
<tr>
<td>Existing other conditions</td>
<td>Diabetes, Familial Hypercholesterolaemia, Renal disease, Established Hypertension</td>
</tr>
</tbody>
</table>

The risk from the modifiable factors is also common to the incidence of other conditions, particularly stroke. The aetiology of IHD is not fully understood although many large epidemiological studies (mainly cohort) have been undertaken. A plausible causal pathway is described in figure 3 and emphasises the independency of age; gender; smoking; cholesterol and blood pressure.

Figure 3: Hypothetical Causal Pathway for IHD

From Coronary Heart Disease Policy Analysis, Universities of Southampton and Birmingham and the London School of Hygiene & Tropical Medicine
In reality, risk factors are interdependent with their effects greater than the sum of each. For assessment of impact however, this pathway is useful since the relative risks of the relevant risk factors are in the majority of cases only available for their singular affect and not for combinations.

There are several models referred to in the literature that predict the impact of changes in risk factors\textsuperscript{3,4}. However, they are both difficult to adopt for a variety of reasons. The National Heart Forum\textsuperscript{5} estimated the proportions of CHD attributable to cholesterol, physical inactivity; blood pressure, smoking and obesity (table 4). The methodology used is based on mortality as these data are routinely available. However, it is likely to be reasonable to substitute the proportions of attributable deaths with the proportions attributable to incidence (numbers of new cases) since relative risks of factors generally equally apply as much to cases as to deaths. From MONICA data, trends in the decline of mortality from CHD reflect being predominantly driven by changes in coronary event rates rather than by case-fatality (survival) changes (proportions dying within 28 days of acute MI attack)\textsuperscript{6}.

<table>
<thead>
<tr>
<th>Table 4: Proportions of CHD attributable to each risk factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk Factor</td>
</tr>
<tr>
<td>---------------------------------</td>
</tr>
<tr>
<td>Cholesterol &gt;5.2mm/l</td>
</tr>
<tr>
<td>Physical inactivity</td>
</tr>
<tr>
<td>Blood pressure &gt;140/90mm Hg</td>
</tr>
<tr>
<td>Smoking</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
</tbody>
</table>

Source: CHD-Estimating the impact of changes in risk factors, National Heart Forum\textsuperscript{5}

NB. Total add up to more than 100%-people have more than one risk factor

The assumptions made in the above model are as follows:

a. based on mortality, assumes that the proportions attributable by risk factor apply equally to new cases
b. assumes that a change of exposure e.g. smoking, reduces the risk of the individual to the level of the newly acquired group, e.g. non-smoking. Whilst some risk factors have an acute affect on the risk of IHD, others such as cholesterol and smoking, have a time-lag. However the assumption is made that they are all acute and this will tend to overestimate the contribution to IHD.
c. Interactions between risk factors are not accommodated i.e. the greater contribution of some risk factors in combination than they would have singularly, has not been taken into account.

To understand the underlying implications of the assessment of the contribution of the various risk factors, how they are distributed in the population is a prerequisite to addressing any inequalities and to help target interventions to reduce them.
Taking the risk factors in turn, the following likely distributions apply to the population.

**Cholesterol**

The mean blood total concentrations in Scottish men and women (Scottish Health Survey, 1998) are 5.4 and 5.3 mm/l respectively. These are similar to those measured in England but by international standards are high. Concentrations increase with age particularly in women so that the concentrations in those aged 55 years and over are significantly higher than in men of equivalent age. The higher levels in older women may reflect either men actively reducing them, or men with high cholesterol dying from IHD. High cholesterol in the National surveys of England and Scotland is defined as a level above 6.5 mm/l. However, the most common target is to achieve levels lower than 5.0 mm/l in those who have an increased risk of coronary heart disease (≥30% 10 year risk of a coronary event, the risk derived from the Joint British Societies Coronary Risk Predictor Chart/calculator). Cholesterol does not appear to be unequally distributed in the population by social class. In contrast, cholesterol is positively associated with the CHD status, particularly in women. In the 1998 Scottish Health Survey, 18.1% of men and 35% of women with IHD had high cholesterol compared to 16.8% and 17% in their respective non-CHD gender groups.

**Physical inactivity**

Among both sexes, physical activity decreases with age, so that the majority of those aged 75 and over, do not undertake any moderate activity of at least 20 minutes duration. The recommendation is that all adults should undertake moderate or vigorous activity of 30 minutes or more on at least 5 days per week. In the UK, only 37% of men and 25% of women meet this criteria and this proportion decreases with age to 17% and 12% respectively in the age group 65-74 years. There does not appear to be a significant pattern of physical activity levels by socio-economic groups. In Scotland proportions of adults aged 16 to 74 meeting the recommended level of activity were estimated to be 38% of men and 27% of women in 1998.

**Blood pressure**

The most recent definition of high blood pressure is an SBP ≥140 or DBP ≥90 mmHg. In Scotland the mean SBP was 132 mmHg for men and 127 mmHg for women as recorded in the 1998 Scottish Health Survey. This is slightly lower than that recorded via the Health Survey of England. Blood pressure increases with age with the prevalence of high blood pressure higher in men than in women up to the age 65 years when thereafter the proportions are similar between men and women. In the 1998 survey, the proportions with high blood pressure was 33.1% in men and 28.4% in women. Prevalence rates do not appear to be different between the 1995 and the 1998 Scottish national surveys. Blood pressure varied slightly with social class in women but not in men.

**Smoking**

The proportion of current smokers and the trends in prevalence with time vary by age group. Although overall the smoking prevalence has decreased, the rates in teenagers have increased, particularly in girls. Prevalence also varies with socio-economic variables being higher in the unemployed, manual employed, overcrowded accommodation, single parents, separated or divorced couples. This distribution is very marked as found in the Scottish Health Survey of 1998. (figure 4):
Figure 4: Prevalence of cigarette smoking (age-standardised) by social class of chief earner in men and women (Scotland 1998)

![Bar chart showing prevalence of cigarette smoking by social class of chief earner in men and women (Scotland 1998)](chart)

Data Source: Scottish Health Survey, 1998

The distribution of current smokers by IHD status is also marked (table 5)

<table>
<thead>
<tr>
<th></th>
<th>With IHD or Stroke</th>
<th>Without IHD or Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>27.2%</td>
<td>34.7%</td>
</tr>
<tr>
<td>Women</td>
<td>43.0%</td>
<td>31.8%</td>
</tr>
</tbody>
</table>

Data Source: Scottish Health Survey, 1998

**Obesity**

Obesity is defined as a BMI equal to or higher than 30 and nearly one fifth of the population fall into this group. It is associated with age, the highest rates in those aged 55 to 74 years and is always higher in women. It also varies with socio-economic groups, with the greatest proportions in the most deprived groups. This pattern is more clearly seen in women.

It is not clear whether obesity is an independent factor as it is associated with greater risk of diabetes, high blood pressure and high blood cholesterol. However, the distribution of those who are overweight or obese, (with BMI of 25 & over) by IHD status is also marked (table 6)

<table>
<thead>
<tr>
<th></th>
<th>With IHD or Stroke</th>
<th>Without IHD or Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>79.2%</td>
<td>60.9%</td>
</tr>
<tr>
<td>Women</td>
<td>80.1%</td>
<td>52.9%</td>
</tr>
</tbody>
</table>

Data Source: Scottish Health Survey, 1998
**Socio-economic factors**

The excess rates of IHD in the more deprived groups are likely to be the result of many factors including behavioural, health selection, early life factors, uptake or access of healthcare, material differences and social position per se (lack of control over one’s life). Although mortality rates are decreasing from IHD, the differences between socio-economic groups are preserved (figure 4).

**Figure 5: Trends in death rates from coronary heart disease in under 65 years by deprivation quintile**

Data source: Health in Scotland 2001, Report by the Chief Medical Officer
4. **Trends in mortality, morbidity and risk factors**

(i) Mortality

Mortality rates from IHD have been decreasing since the 1970’s. Thus the reduction in the relative burden of disease posed by IHD (figure 6) is likely to be real and not solely as a consequence of a relative increase in the rates of other causes of ill-health such as cancers.

**Figure 6: The relative contributions of IHD as a cause of death in Highland (1981 to 2001)**

![Pie chart showing relative contributions of IHD, stroke, resp disease, others to deaths in Highland from 1981-2001]

Data source: GRO

Mortality rates in the Highland population have decreased by 44% in both sexes over the last 20 years (figure 7), with the decrease more pronounced in males (the relative rates however remaining at a ratio of 2.3).

**Figure 7: Directly standardised mortality rates (95% confidence intervals) from IHD in males and females of Highland residents: 5 year rolling average from 1982 to 2001**

![Graph showing directly standardised mortality rates from IHD in Highland residents from 1982 to 2001]

Data sources: *Directly standardised death rates (European standard population) with final revised mid-year population estimates, February 2003, GRO*
This decrease has not occurred evenly in all age groups with the younger age groups of both sexes (52% in men and 45% in women), demonstrating the largest fall compared to the relatively older age band where the decrease has been 20-21% (figure 8).

**Figure 8: Age specific mortality rates from IHD in males and females of Highland residents: 5 year rolling average from 1982 to 2001**

![Figure 8: Age specific mortality rates from IHD in males and females of Highland residents: 5 year rolling average from 1982 to 2001](image)

Data source: GRO deaths and with final revised mid-year population estimates, February 2003, GRO

Mortality rates from AMI have decreased by just under 60% in both sexes over the last 20 years. Death rates from Heart Failure although decreased overall by 50% in males and 40% in females over the same time period, has not been as consistent (figure 9).
Figure 9: Directly standardised mortality rates (95% confidence intervals) from Heart Failure in males and females of Highland residents: 5 year rolling average from 1982 to 2001

Data source: *Directly standardised death rates (European standard population) with final revised mid-year population estimates, February 2003, GRO

(ii) Morbidity

**Incidence & Prevalence as measured by Continuous Morbidity Recording (CMR)**

Incidence rates as reflected by uptake rates of primary care in Scotland by reason for consultation (CMR data) have also decreased. Over the last 6 years (1997 to 2002) rates have decreased by 46%, 34% and 80% for IHD, Angina and heart failure respectively (figure 10)

Figure 10: Directly standardised incidence rates from IHD, Angina and Heart Failure based on GP consultation rates from 1997 to 2002

Data source: CMR data for Scotland, ISD:: *age specific rates standardised to European standard population

Prevalence rates as reflected by Primary care uptake data also decreased during the same period in Scotland. However the decrease was lower at 14% for IHD, 24% for angina and 24% for Heart Failure (figure 11).
Incidence and prevalence rates based on hospital-linkage data

The incidence rates of IHD estimated by this data system are less than half those based on GP consultation rates. As those for Heart Failure and for Angina are roughly similar, the wider definition according to the Read codes used would account for some of this difference. A second reason for the discrepancy is likely to arise from how the data are collected in the two systems. Some patients may be entirely managed in Primary Care, and do not enter the hospital-linkage system other than through death. A third reason is that CMR data may not be representative of the situation in Highland, where incidence may be lower than the national average as indicated by the lower mortality rates. These reasons together with the fact that CMR data is an annual count and does not link from one year to the next, mean they will tend to provide higher estimates of both incidence and prevalence relative to the hospital-linkage data.

The trends in rates over the 20 year period from 1981 to 2000 as based on the hospital-linkage data are depicted in figure 12. It should be noted that the rates from AMI and IHD decrease by 39% and 18% respectively, whilst those for angina increase. This contrasts again with the primary care data where rates decreased by 34% in the last 6 years.
Figure 12: Directly standardised incidence rates from IHD, AMI, Angina and Heart Failure based on hospital linkage data during the 20 year period from 1981 to 2000

Data source: ISD hospital-linkage data for Highland; *age specific rates standardised to European standard population

The hospital-linkage system also demonstrates increased numbers of prevalent cases each year, with the largest occurring for Angina [80% increase over 6 years (1995-2000)]. The methodology used in the hospital-linkage data (cumulative count of patients alive) is not reliable for years early in the 20 year period covered, where discharges prior to 1981 were not used, thus rendering an underestimate for the earlier years of the period covered. The prevalent number estimated for IHD in the year 2000 is in roughly the same order of magnitude as the GP-based data thus trends in the later years are only considered to be reliable. Due to this, only the data relating to the last 6 years are considered (figure 13). This contrasts again with CMR estimates where trends are downwards for prevalence rates and the proportion of prevalent cases of angina falls rather than increases as is the case for the hospital-linkage system.

Figure 13: Numbers of prevalent cases of IHD, AMI, Angina and Heart Failure based on hospital linkage data each year from 1995 to 2000

Data source: ISD hospital-linkage data for Highland;
Trend analysis of the above data sets in forecasting rates in 2010 and 2016 and applying these to the projected populations provide the following estimates (table 7):

Table 7: Predicted numbers in Highland to be affected by CHD based on historical trends of rates and applied to projected populations

<table>
<thead>
<tr>
<th>Year</th>
<th>Annual Numbers</th>
<th>Incidence</th>
<th>AMI</th>
<th>Angina</th>
<th>Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>IHD</td>
<td>AMI</td>
<td>Angina</td>
<td>Heart Failure</td>
</tr>
<tr>
<td>Base year 1998/2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2010</td>
<td>625</td>
<td>326</td>
<td></td>
<td>231</td>
<td>251</td>
</tr>
<tr>
<td>2016</td>
<td>593</td>
<td>165</td>
<td></td>
<td>376</td>
<td>226</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>-5%</td>
<td>-49%</td>
<td></td>
<td>+63%</td>
<td>-10%</td>
</tr>
<tr>
<td>% change to 2016</td>
<td>-16%</td>
<td>-70%</td>
<td></td>
<td>+100%</td>
<td>-21%</td>
</tr>
<tr>
<td>Prevalence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base year 2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2010</td>
<td>5347</td>
<td>-</td>
<td></td>
<td>3179</td>
<td>1515</td>
</tr>
<tr>
<td>2016</td>
<td>4937</td>
<td>-</td>
<td></td>
<td>2497</td>
<td>1153</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>-8%</td>
<td>-</td>
<td></td>
<td>-21%</td>
<td>-24%</td>
</tr>
<tr>
<td>% change to 2016</td>
<td>-16%</td>
<td>-</td>
<td></td>
<td>-39%</td>
<td>-43%</td>
</tr>
<tr>
<td>Deaths</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base year 1998/2000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2010</td>
<td>501</td>
<td>Non-significant trends</td>
<td>Non-significant trends</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>2016</td>
<td>337 (189-483)</td>
<td></td>
<td></td>
<td>37 (27-43)</td>
<td>34 (23-46)</td>
</tr>
<tr>
<td>% change to 2010</td>
<td>-33%</td>
<td></td>
<td></td>
<td>-8%</td>
<td></td>
</tr>
<tr>
<td>% change to 2016</td>
<td>-53%</td>
<td></td>
<td></td>
<td>-15%</td>
<td></td>
</tr>
</tbody>
</table>

1Hospital-linkage data: new admissions: from trends 1991-2000 applied to projected population 2CMR prevalence from trends 1996-2002 applied to projected population 3From forecasts of trends of age & sex-specific rates (5 year rolling average) over 1993 to 2001 and applied to 1998-based population projections

(iii) Trends in Risk factors

Most of the data supporting these are from the National Health Survey (Scotland). Local rates where available (2001 Adult Health and Lifestyle Survey) are also used.

Blood Cholesterol

The proportion of people with blood cholesterol concentrations of 6.5 mm/l or over decreased in all ages in both sexes in Scotland except for younger males. Overall the proportions decreased from 23% to 17% in men and from 21% to 15% in women. However women aged 55-64 still record the highest levels of 39% in 1998, a decrease from 51% in 1995 (figure 14).
Physical inactivity

Both in England and Scotland, there is no evidence that activity levels have increased and some indication that the populations have become more sedentary. In Highland the adult lifestyle surveys have indicated decreased levels of physical activity (table 8):

Table 8: Proportions undertaking levels of physical activity in Highland, 1991; 1996 and 2001

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Spends most of the day sitting down</td>
<td>21%</td>
<td>26%</td>
<td>26%</td>
</tr>
<tr>
<td>Spends most of the day in moderate activity</td>
<td>68%</td>
<td>61%</td>
<td>60%</td>
</tr>
</tbody>
</table>

Data source: Adult Health and Lifestyle Survey: Highland 2001

Blood Pressure

Using the new definition of hypertension, the proportions in Scotland have slightly increased in both men and women between 1995 and 1998. This increase is confined to the older age groups (figure 15)
The overall proportions of those with high blood pressure is lower in Scotland than England based on the respective national Health Surveys carried out in 1998: men 33.1% versus 40.1% in England and 28.4% versus 29.4% in England. It is likely that around 70% of those with high blood pressure as defined by survey are receiving medication and just under 80% of these are being successfully treated.

**Smoking**

Overall, there has been little difference in the prevalence of cigarette smoking in Scotland between the national surveys of 1995 and 1998. For those aged 16 to 64 years, 36% men and 33% of women are current cigarette smokers as per 1998 survey. The equivalent proportions in England are lower at 30% and 29%. Estimated rates in Highland are even lower at 25% for both men and women in 2001, which represents a consistent decrease from estimates of the 1991 and 1996 surveys in Highland of 32% and 29% respectively.

Prevalence of smoking decreased with age (figure 16) but this belied an increased proportion of heavy smokers within the older age groups.
**Obesity**

Prevalence of obesity has increased in all age groups for both men and women (except for women aged 16 to 24 years) in Scotland between 1995 and 1998 (figure 17). Overall the proportions of men and women aged 16 to 74 years who are obese is estimated at 19.6% and 22.1% in men and women respectively in 1998 in Scotland. Highland rates appear to be lower at 14% in males and 15% in females (2001 survey). In keeping with the national trend, rates have increased in Highland as reflected in the proportions who are overweight from 41% in 1991 to 52% in 2001.

**Figure 17: Proportions of those obese in 1995 and 1998, by age and sex in Scotland**

* as defined as BMI =>30Kg/m²
Data source: Scottish Health Survey 1998
5. Potential effectiveness of interventions at the 3 levels: primary prevention, acute management and secondary prevention

The reduction in deaths from CHD in Scotland from 1975 to 1994 has been subject to cohort mortality modelling\(^\text{12}\). The number of annual deaths prevented or postponed (29\% of expected) was attributed to (table 9):

<table>
<thead>
<tr>
<th>Proportion of annual deaths avoided</th>
<th>Attributed by</th>
</tr>
</thead>
<tbody>
<tr>
<td>51%</td>
<td>Risk factor reduction: smoking, 36%; cholesterol, 6%; Blood Pressure, 6%; change in deprivation, 3%</td>
</tr>
<tr>
<td>40%</td>
<td>Medical &amp; surgical treatment: AMI 10%; hypertension, 9%; 2\textsuperscript{nd} prevention, 8%; Heart Failure, 8%; aspirin, 2%; CABG, 2%; PTCA, 1%</td>
</tr>
<tr>
<td>9%</td>
<td>Underdetermined (unmeasured obesity, exercise etc.</td>
</tr>
</tbody>
</table>

Source: Capewell et al 1999\(^\text{12}\)

The relative contribution of treatment and primary prevention may be judged by the relative contributions the trends in case fatality rates and coronary events rates have in the reduction of mortality from CHD. The MONICA study (WHO, international collaboration of 21 countries in which data are collected from various centres including Belfast and Glasgow and commenced in the 1980’s) indicates very little improvement in case fatality rates (deaths within 28 days of a coronary event), in those aged 35 to 64 years. The rates in Glasgow are 48\% in men and 46\% in women for the most recent period for which data are available (1985/94)\(^\text{13}\). Although this represents a significant potential for improvement, case-fatality rates have been reducing as little as 2\% per year\(^\text{13}\) (i.e. 2\% of the cases fatality rate equivalent to less than 1\% in absolute terms). The proportion of those admitted as an emergency for AMI in Highland who are still alive at 30 days and over has fluctuated between 81\% and 86.5\% over the last 5 years but is currently a little lower than the national average (figure 18). Note that this does not equate to the reciprocal of the case fatality rate, the definition of which includes all cases of AMI whether admitted to hospital or not.
The MONICA data also shows that overall, two thirds in the reduction of mortality from CHD was accounted for by trends in case event rates (incidence) as opposed to one third accounted for by trends in case fatality. For women the proportions were closer than for men, 65% versus 35% and 79% versus 21% in women and men respectively.

Whilst the contribution of risk factor changes in the population is believed to have been considerable, over 50%, in determining the lower current mortality rates of CHD in Scotland, the potential to additionally reduce them is not clear. This is because the effectiveness of primary prevention interventions is not reported to be high in studies where it has been measured. A systematic review and meta-analysis of randomised control trials of the effectiveness of multiple risk factor interventions, more than one of six risk factors (dietary advice, stopping smoking, exercise, weight control, antihypertensive and cholesterol lowering drugs) indicated that health promotion through educational interventions have only modest effects up to at least a six month period on risk factor change in the general or workforce population. This lack of evidence also applies to community trials, such as North Karelia and the Minnesota Heart Health Programme, where downward trends in mortality and risk factors have been paralleled in comparable regions in which no intervention occurred at the same time. (In contrast it is claimed that a single polypill is more effective if given to those aged over 55 years and those with vascular disease containing aspirin, statin, 3 blood pressure lowering agents in half dose and folic acid). There is however some evidence from one systematic review for the effectiveness of community pharmacy-based interventions in smoking cessation (UK) and lipid-lowering management (US and Canada) in the prevention of CHD. It is clear though that health protection through national legislative and fiscal changes are associated with effective reduction in risk factors e.g. the Canadian Tobacco Laws.
The use of effective health promotional, educational or national fiscal/legislative interventions, is likely to be rewarding in reducing the burden of CHD due to the considerable potential contribution of population risk factor to CHD mortality. The expected reductions in CHD incidence or mortality brought about by modifications in four risk factors at the population level have been quantified by the National Heart Forum as follows (table 10):

### Table 10: Potential effects on CHD by changes in risk factors

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Change in risk factor</th>
<th>% reduction in CHD cases or deaths</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cholesterol</strong></td>
<td>All reduce to &lt;5mm/l</td>
<td>53.4%</td>
<td>55.5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All reduce to &lt;6.5mm/l</td>
<td>11.1%</td>
<td>12.6%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All reduce by 0.3mm/l</td>
<td>4.8%</td>
<td>5.2%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All reduce by 0.6mm/l</td>
<td>9.2%</td>
<td>9.7%</td>
<td></td>
</tr>
<tr>
<td><strong>Physical activity</strong></td>
<td>All those sedentary become light</td>
<td>2.0%</td>
<td>2.1%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All light &amp; sedentary become moderate</td>
<td>9.6%</td>
<td>10.7%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25% in the proportion moderately active</td>
<td>1.2%</td>
<td>1.0%</td>
<td></td>
</tr>
<tr>
<td><strong>Blood Pressure</strong></td>
<td>Reduce proportion with high BP by 50%</td>
<td>6.5%</td>
<td>5.5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All reduce DBP by 2.5mmHg</td>
<td>5.0%</td>
<td>4.1%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All reduce DBP by 5mmHg</td>
<td>10.6%</td>
<td>8.6%</td>
<td></td>
</tr>
<tr>
<td><strong>Smoking</strong></td>
<td>Prevalence of 24% by 2010</td>
<td>0.6%</td>
<td>0.5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Prevalence of 175 men, 18% women</td>
<td>1.6%</td>
<td>1.5%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All quit</td>
<td>20%</td>
<td>17%</td>
<td></td>
</tr>
<tr>
<td><strong>Obesity</strong></td>
<td>6% in men and 8% in women</td>
<td>3.2%</td>
<td>3.6%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>All have BMI&lt; 27.5</td>
<td>4.9%</td>
<td>6.0%</td>
<td></td>
</tr>
</tbody>
</table>

*Source: National Heart Forum*

It is relevant therefore to consider the role of statins (lipid-lowering drugs), in the primary prevention of CHD through reductions in blood cholesterol from what was previously considered not exceptionally high levels (“mildly high” of 5 – 6.4mm/l). Although the current guidance from SIGN is for statins to be considered after dietary advice for those whose 10 year CHD risk is 30% or more and whose blood cholesterol is 5mm/l or over, there is evidence for CHD mortality benefits for those with a 10 year CHD risk as low as 13%.17

In contrast to primary prevention, there is substantial evidence for the effectiveness of interventions in reducing risk factors and the effectiveness of risk factor reductions per se in lowering the risk of further coronary events and mortality in those already with CHD, i.e. in secondary prevention. A systematic review has attributed a 30-40% reduction in all cause mortality at 2 years or longer in those diagnosed with CHD who have ceased to smoke17 After MI or angina, trials of multiple or single risk factor interventions have shown clear benefits14. It is probable that the effectiveness of interventions to reduce life-style risk factors in secondary prevention are high because those who know they are already at risk are more likely to be motivated in following advice. Reductions in mortality has been attributed to the following intervention at the secondary prevention level (table 11):
Table 11: Attributed reductions in mortality in those with CHD by intervention

<table>
<thead>
<tr>
<th>Intervention</th>
<th>All mortality reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking cessation</td>
<td>36%</td>
</tr>
<tr>
<td>Statins</td>
<td>29%</td>
</tr>
<tr>
<td>β-blockers</td>
<td>23%</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>23%</td>
</tr>
<tr>
<td>Aspirin</td>
<td>15%</td>
</tr>
</tbody>
</table>

Source: Critchley & Capewell 2003

The benefit of the revascularisation is usually assessed by comparing with medical treatment alone or between different surgical modalities. The following appears to apply (table 12):

Table 12: Effectiveness of surgical modalities in CHD

<table>
<thead>
<tr>
<th>Procedures</th>
<th>Relative risk (95% confidence intervals)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CABGs versus medical treatment alone</td>
<td>61% (48%-77%) at 10 years</td>
</tr>
<tr>
<td>PTCA versus CABGs</td>
<td>0% but greater nos. of repeat procedures</td>
</tr>
<tr>
<td>Stents versus PTCA alone</td>
<td>0% but fewer repeat procedures</td>
</tr>
</tbody>
</table>

Source: Pignone M et al

In the acute management of MI, there is clear evidence for reduction in mortality of thrombolytic treatment if given within 6 hours and possibly up to 12 hours and longer after the onset of symptoms. The absolute risk benefit reduces with time of administration after MI. The numbers needed to treat vary with time of administration from 33-50. There is no evidence to support the efficacy of a particular thrombolytic agent over others although there may be greater harmful affects such as tissue plasminogen activator versus others in respect of the incidence of stroke or intracerebral haemorrhage.

Adoption of the new definition of MI (ACC/ESC), is expected to reduce morbidity and improve case survival. Only one third of patients with acute chest pains are found to have ST-segment elevation on ECG. Establishing the correct diagnosis in patients without ST-segment changes is more difficult with undetected infarctions putting patients immediate lives at risk or resulting in more severe morbidity than would otherwise occur. Troponin I or T indicate irreversible cell damage and have higher sensitivity than other known cardiac markers. It has been estimated that there could be a 10% increase in the number of patients diagnosed with MI by using the measurement of Troponins as opposed to conventional cardiac biomarkers. In addition, in those who would previously have been diagnosed with unstable angina, the use of troponin measurement may now result in a diagnosis of MI as very small MIs can be detected. This means that such patients can receive the appropriate therapeutic interventions more quickly and is particularly important since unstable patients with elevated troponin concentrations are known to be at significantly higher risk of death or a serious coronary event within 30 days. Troponins also have higher specificity than conventional markers thus resulting in fewer falsely positively diagnosed patients. Hence it is not clear whether overall the use of troponins will result in an increase in the incidence of MI. It is expected that the new definition of MI will be adopted more widely in Scotland, (a recent survey indicated that only a third of Cardiologists in Scotland apply the definition and that not all Coronary Care Units have access to troponin assay) and that patients in Highland should also benefit by it’s adoption.
The relative effectiveness of treatment interventions can be measured by the numbers needed to treat to avoid one death or event (NNTs). These are depicted in figure 19.

**Figure 19: Effectiveness of interventions in cardiac conditions: NNT in one year to prevent one death**

![Bar chart showing effectiveness of interventions](chart.png)

Source: Capewell et al SNAP CHD 1998

It is estimated that if 80% of eligible patients receive appropriate treatment, rather than the 33% thought to currently receive it, deaths could be further reduced by 20%.
6. Current intervention rates

Surgical intervention rates in 2002 in Highland are relatively low compared to the national average for angiography; bypass grafts and angioplasty (figures 20-22)

Figure 20: Rates of Bypass grafts in Scotland by Health Board area of residence year ending March-31 2002

Data source: ISD and SMR01 data, * Directly standardised to European population

Figure 21: Rates of Angioplasty in Scotland by Health Board area of residence year ending March-31 2002

Data source: ISD and SMR01 data, * Directly standardised to European population
Figure 22: Rates of Angiography in Scotland by Health Board area of residence year ending March-31 2002

Data source: ISD and SMR01 data, * Directly standardised to European population

Prescribing rates of cardiovascular drugs also are lower in Highland than the Scottish average, particularly lipid lowering drugs, (figure 23):

Figure 23: Prescribing of cardiovascular related drugs

Data source: Skipper ISD
Conclusions

- Predictions of the level of burden from CHD in 2010 and 2016 in Highland have been made for each of two scenarios:
  - i. the status quo in respect of current rates (table 2)
  - ii. projection of historic trends (table 7)

- Scenario (i) predicts increased levels of 16% and 28% in morbidity and 21% and 38% in mortality whilst scenario (ii) predicts decreased levels of 5% and 16% in morbidity and 33% and 53% in mortality.

- There are some anomalies to be understood regarding the sources of data e.g. trends of CMR data versus hospital-linkage particularly for the predictions of morbidity levels of angina.

- The contribution of changes in risk factors to the reduction in CHD burden is large, at least 50%, whilst medical and surgical treatment have contributed 40% (table 9).

- There are a number of risk factor targets that would be expected to reduce future levels of CHD, the most striking of which is a greater than 50% reduction due to attaining cholesterol levels below 5 mm/l, (table 10)

- There is little evidence for the effectiveness of primary prevention using health promotional or educational interventions on the general population or workforce

- In contrast, there is substantial evidence for the effectiveness of risk-reduction interventions in those with CHD (secondary prevention).

- Angiography rates in the Highland population are relatively low and attaining the national average or the target recommended by the CHD/Stroke taskforce group would increase the uptake of appropriate therapy.

- Surgical intervention (Coronary reperfusion) rates are also relatively low and increasing these would be expected to increase case survival and improve the quality of life of those with CHD.

- Statin prescribing is relatively low in Highland particularly when there is evidence to support the effectiveness of cholesterol lowering even in patients with a moderately low 10 year risk (13%) of CHD

- Adoption of the new definition of MI presents the opportunity to improve case survival and reduce the severity of morbidity from CHD in the population
Glossary

ACC: American College of Cardiology
AMI: Acute Myocardial Infarction
BMI: Body Mass Index
CABG: Coronary Artery Bypass Grafts
CHD: Coronary Heart Disease
CMR: Continuous Morbidity Recording
DBP: Diastolic Blood Pressure
ECG: Electro Cardio Graph
ESC: European Society of Cardiology
HF: Heart Failure
ICD: International Classification of Diseases
IHD: Ischaemic Heart Disease
PTCA: Percutaneous Transluminal Coronary Angioplasty
SBP: Systolic Blood Pressure
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Appendix : Methodology

Scenario 1: Status quo (for predictions in table 2)

Predictions for 2010 and 2016 made by application of age-sex specific rates to the 1998-based population projections in Highland (GRO) from the following data sets:

(i) Hospital-linked admission data 1981-2000, ISD
From admissions in the 20 year period by quinary age-band and sex with discharge codes:

<table>
<thead>
<tr>
<th>Condition</th>
<th>ICD-9</th>
<th>ICD-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>IHD</td>
<td>410-414</td>
<td>I20-I25</td>
</tr>
<tr>
<td>AMI</td>
<td>410</td>
<td>I21</td>
</tr>
<tr>
<td>Angina</td>
<td>413</td>
<td>I20</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>428</td>
<td>I50</td>
</tr>
</tbody>
</table>

(a) Incidence: admissions of individuals counted only once during 1981-2000 occurring at the earliest date of admission
(b) Prevalence: as the cumulative count of incident cases, all ages by sex, still alive over the 20 year period—it is likely to be an underestimate particularly at the beginning of the period since admissions prior to 1981 have not been counted.

Rates were calculated as:
Incidence rate (I) for each condition as: \( \frac{\sum i_{sg}(1998-2000)}{\sum p_{sg}(1998-2000)} \)
Where \( i_{sg} \) = incidence count for each 5 year age band and sex
\( p_{sg} \) = population count in 5 year age-band by sex from mid year population estimates revised on the 2001 populations (GRO)

The predicted number of annual new cases calculated as:
\[ I = \sum \left( \frac{\sum i_{sg}(1998-2000)}{\sum p_{sg}(1998-2000)} \right) \times p_{proj_{sg}(2010 \ or \ 2016)} \times 100,000 \]
Where \( p_{proj_{sg}} \) = the population count in the relevant sex specific age-band of the projected population (1998-based to 2010 and 2016, GRO).

(ii) Continuous Morbidity Recording (CMR data) 1996-2000—obtained from ISD website
Rates per 1000 by the age bands for each sex (45-54; 55-64; 75-84; 85 & over) applied to 1998-based population projections of 2010 and 2016 sex—specific age-bands

(iii) Study-based incidence rates:
   (a) OXMIS (Volmink JA et al 1998; Heart 80 pp40-44) rates per 100,000 for males and females 35-64 year age-band applied to 1998-based population projections of 2010 and 2016 sex—specific age-bands
   (b) MONICA (Tunstall-Pedoe H et al 2000; Lancet 353 pp1547-57) rates per 100,000 for males and females 35-64 year age-band applied to 1998-based population projections of 2010 and 2016 sex—specific age-bands
(iv) Deaths

5-year age-band/sex specific numbers of deaths by condition (GRO) for 1998-2000:

Mortality rate (D) for each condition as: \[ \frac{\sum d_{sg}(1998-2000)}{\sum p_{sg}(1998-2000)} \]

Where \( d_{sg} \) = incidence count for each 5 year age band and sex

\( p_{sg} \) = population count in 5 year age-band by sex from mid year population estimates revised on the 2001 populations (GRO)

The predicted number of annual deaths calculated as:

\[ I = \sum ((\frac{\sum d_{sg}(1998-2000)}{\sum p_{sg}(1998-2000)}) \times p_{projsg}(2010 \text{ or } 2016)) \times 100,000 \]

Where \( p_{projsg} \) = the population count in the relevant sex specific age-band of the projected population (1998-based to 2010 and 2016, GRO).

Scenario 2: Possible (for predictions in table 7)

(i) Hospital-linked admission data 1991-2000, ISD

Linear regression applied to the directly standardised rates (to the European standard population) pertaining to 1991 to 2000 for each condition. The t value calculated as:

\[ t = r \sqrt{\frac{(n-2)/(1-r^2))} \]

and compared to critical value for the Student’s t distribution using n-2 degrees of freedom. Forecasts were made only where correlation coefficients were statistically significant (p ≤ 0.05). Forecasts made using Minitab with 95% confidence intervals

(ii) Continuous Morbidity Recording (CMR data) 1996-2002-obtained from ISD website

Prevalence rates for each condition per 1000 practice populations were directly standardised to the European standard population. Linear regression applied to the directly standardised rates (to the European standard population) pertaining to 1996 to 2002. The t value calculated as:

\[ t = r \sqrt{\frac{(n-2)/(1-r^2))} \]

and compared to critical value for the Student’s t distribution using n-2 degrees of freedom. Forecasts were made only where correlation coefficients were statistically significant (p ≤ 0.05). Forecasts made using Minitab with 95% confidence intervals

(iii) Mortality

5-year rolling directly standardised (European standard population) death rates were calculated from deaths in Highland by 3 age bands (15-64; 65-84; 85 & over) for each sex from 1993 to 2001 (i.e. 5 year estimates from 93/97 to 97/2001). Linear regression was applied to the rates and the t value calculated as:

\[ t = r \sqrt{\frac{(n-2)/(1-r^2))} \]

and compared to critical value for the Student’s t distribution using n-2 degrees of freedom. Forecasts were made only where correlation coefficients were statistically significant (p ≤ 0.05). Forecasts made using Minitab with 95% confidence intervals