

the “drift”. Our predictions are that stroke deaths are likely to continue to decrease up to at least 2015. For 2015, our predictions of deaths from stroke varied from 34 429 to 46 538 (compared with 47 213 deaths in 2005) under the different plausible model scenarios. We also noted that there were some potentially interesting trends in mortality amongst recent birth cohorts. In particular, mortality was higher in older age groups, but the difference between the older and younger age groups appears to have decreased over time for both sexes. Modelling of the subgroup aged 40–69 suggested a relative rate increase in mortality amongst those born since the mid-1940s compared with earlier cohorts; this trend appears to have been sustained in men.

Conclusions: Downward trends in stroke mortality are set to continue overall to at least 2015. However, these trends appear to be levelling off in middle-aged people, particularly men. This raises concerns that stroke mortality might increase as these cohorts grow older.

030 CHOOSING METRICS IN PUBLIC HEALTH ASSESSMENTS: ATTRIBUTING CREDIT FOR THE RECENT LARGE CORONARY HEART DISEASE MORTALITY DECLINE IN THE US POPULATION

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Background: Most explanations of falls in coronary heart disease (CHD) mortality limit themselves to event based metrics (such as fewer deaths). However, time based metrics (such as life-years gained (LYG)) promise to capture more of the social value attached to the deaths averted. We have assessed the sensitivity of conclusions about the relative contributions of treatments and risk factor changes to the choice of metric.

Methods: Using a validated CHD mortality model (IMPACT), we integrated data on the number of CHD patients, treatment uptake, treatment effectiveness, risk factor trends, and median survival among US adults aged 25–84 between 1980 and 2000, in order to estimate fewer deaths and LYGs. LYG were estimated using the US general population life expectancy for CHD onsets averted in 2000 and the median survival rates from Medicare for the additional survivors after CHD onset. (All the latter gains are currently attributed to treatments). We examined how uncertainty within the model may vary according to choice of metric.

Results: Between 1980 and 2000, CHD mortality-rates halved resulting in approximately 341 745 fewer deaths in 2000; approximately 47% of the fall was attributed to treatments in patients (after clinical presentation) and 44% to population-wide risk factor reductions (independent of medication). However, this split was altered to 35%/65% when LYG was used. Taking smoking as an example, those who did not experience a smoking attributable CHD death in 2000 because they did not smoke have been given the average US life expectancy (at the age of averted death) in 2000. The life-expectancy of the hypothetical non-smoker, however, would be expected to be higher. Applying these extended years to those deaths avoided by not smoking can add more than 50 000 more life years attributable to the decline of smoking in the population. Furthermore, while the model attributes all gains from increased survival post CHD onset to improved treatments, an increasing proportion of non-smokers among CHD patients could result in additional LYG attributable to smoking reduction.

Discussion and Conclusions: Using life-years gained rather than deaths avoided strengthens the case for primary prevention by risk factor reduction, because it captures more fully the social gains that result. The additional assumptions required are all relatively minor. It seems that past versions of the model may understate the relative

importance of risk factor changes when better assessed using the metric of life-years gained.

031 EXPLAINING THE MASSIVE DECLINE IN CORONARY HEART DISEASE MORTALITY IN ICELAND BETWEEN 1981 AND 2006

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Background: Coronary heart disease mortality rates have been decreasing in Iceland since the 1980s. We used the validated IMPACT model to examine how much of the 80% decrease in Iceland between 1981 and 2006 could be attributed to medical and surgical treatments and how much to changes in cardiovascular risk factors.

Methods: The previously validated IMPACT mortality model was used to combine and analyse data on uptake and effectiveness of cardiological treatments and risk factor trends in the entire Iceland population. The main data sources were official statistics, national quality registers, published trials and meta-analyses, clinical audits and a series of national population surveys. Sensitivity analyses were then conducted.

Results: Between 1981 and 2006, coronary heart disease mortality rates in Iceland decreased by 80% in men and women aged 25 to 74 years. This fall resulted in 295 fewer deaths in 2006. Approximately one quarter of this decrease (27%) was attributable to treatments in individuals (including some 11% to secondary prevention, 5% to heart failure treatments, 5% to initial treatments of acute coronary syndrome, 2% to hypertension treatments and less than 1% to lipid reduction). Almost three quarters of the mortality decrease (72%) was attributable to population risk factor reductions (principally cholesterol, 35%; smoking, 22%; systolic blood pressure, 22% and physical activity, 5%). Adverse trends were seen for diabetes (–5%), and obesity (–4%).

Conclusions: Almost three quarters of the large coronary heart disease mortality decrease in Iceland between 1981 and 2006 was attributable to reductions in major cardiovascular risk factors in population (mainly decreases in total serum cholesterol, smoking and blood pressure levels). Very little was attributable to primary prevention medications. These findings emphasise the value of a comprehensive strategy that promotes tobacco control and a healthier diet. It also highlights the potential importance of effective, evidence based medical treatments.

032 20-YEAR TRENDS IN MAJOR CORONARY RISK FACTORS IN OLDER BRITISH MEN: ASSESSING THE IMPACT OF MEDICATION USE

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Background: Favourable trends in blood pressure (BP) and blood lipids have contributed to the falling incidence of coronary heart disease (CHD) in recent decades. The role of medication in the BP and blood lipid trends is unknown.

Objectives: To investigate the impact of medication on 20-year trends in BP and blood lipids in older British men.

Design: Longitudinal study.

Setting: 24 British towns.

Participants: 4231 men from a socially and geographically representative cohort of older British men, examined at baseline (1978–80, aged 40–59 years) and after 20 years (1998–2000).

Main Outcome Measures: 20-year trends in systolic blood pressure (SBP), diastolic blood pressure (DBP), and total, non-HDL and HDL cholesterol according to medication use, derived from linear regression with generalised estimating equations, adjusting for age and risk factors for dyslipidemia and hypertension.

Results: Over 20 years from 1978–80 to 1998–2000, overall age-adjusted mean SBP fell: mean change -7.6 mm Hg (95% CI -9.7 to -5.4), whilst mean DBP rose: mean change $+3.3$ mm Hg ($+2.2$ to $+4.5$). Among 1573 men (37%) who reported taking BP-lowering medication during the follow-up, mean SBP fell significantly by -12.3 mm Hg (-14.7 to -9.9) and DBP fell non-significantly by -1.2 mm Hg (-2.5 to $+0.07$). In contrast, men not using BP-lowering medication experienced a non-significant fall in SBP of -1.6 mm Hg (-3.7 to $+0.5$) and an increase in DBP of $+7.7$ mm Hg ($+6.6$ to $+8.8$) ($p < 0.001$ for medication-time interaction for both SBP and DBP). Overall mean total cholesterol fell by -0.2 mmol/L (-0.3 to -0.08), and mean non-HDL cholesterol fell by -0.4 mmol/L (-0.5 to -0.2). Total and non-HDL cholesterol fell significantly by -1.6 mmol/L (-1.8 to -1.4) and -1.8 mmol/L (-2.0 to -1.6) respectively among 302 men (8%) reporting use of lipid-regulating drugs, but fell by only -0.1 mmol/L (-0.2 to $+0.03$) and -0.2 mmol/L (-0.4 to -0.1) respectively among men not using lipid-regulating medication ($p < 0.001$ for medication-time interactions). Mean HDL cholesterol levels rose overall by $+0.16$ mmol/L ($+0.13$ to $+0.19$). The trend did not vary appreciably according to lipid-regulating drug use ($p = 0.15$ for interaction).

Conclusions: A marked cohort-wide increase in HDL levels occurred, independent of medication use, therefore probably reflecting changes in health behaviours. Decreases in SBP and total and non-HDL cholesterol were largely confined to medication users suggesting limited implementation of population-wide lifestyle strategies to reduce these coronary risk factors. Greater efforts are needed to reduce BP and cholesterol among the general population if the potential benefits of population strategies for CHD prevention are to be realised.

Alcohol

033 ALCOHOL CONSUMPTION AND THE U-SHAPE RELATIONSHIP WITH MORTALITY: 8-YEAR FOLLOW-UP OF MORE THAN 6000 OLDER MEN IN THE WHITEHALL STUDY

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Objective: To assess the relevance of alcohol consumption for cause-specific mortality in old age.

Design: Prospective cohort study of 6157 older men (mean age: 77 yrs) followed for cause-specific mortality for an average of 8 years.

Methods: Current and past alcohol consumption were collected by means of a postal questionnaire in 1997/98 among surviving participants in the 1970 Whitehall study of London Civil Servants. Mortality rates in each of six alcohol consumption groups (including a group for reduced or ex-drinking due to ill-health) were estimated after standardisation for age, smoking and employment grade. Cox proportional hazards models were used to estimate hazard ratios.

Results: Of the 6157 men, 1578 men (26%) with a prior history of vascular disease (myocardial infarction, angina or stroke) were excluded. About one quarter of the remaining men reported drinking less than they had done 5 years earlier, with half of these having reduced or given up alcohol for health reasons ("sick quitters"). Alcohol consumption was strongly related to plasma HDL cholesterol concentrations (1% (0.01 mmol/L) higher concentration per unit per week), and also with higher levels of blood pressure and rates of cigarette smoking. During follow-up, 2220 men died (annual rate: 56/1000/yr), including 825 from a vascular cause (21/1000/yr) and 1395 from a non-vascular cause (35/1000/yr). There was a U-shaped relation between alcohol consumption and mortality from all-causes and vascular causes, with the highest mortality observed among sick quitters and men who drank more than 28 units a week. Compared with men who drank 1–7 units a week (standardised death rate: 48/1000/yr), the adjusted HR (95% CI) for all-cause mortality was 1.45 (1.26 to 1.66) for sick quitters, 1.25 (1.11 to 1.41) for non-drinkers, and 1.32 (1.11 to 1.57) for those who drank more than 28 units a week. The risks associated with heavy drinking were even more extreme for vascular mortality (HR 1.48, 95% CI 1.13 to 1.95).

Conclusion: While the excess mortality among non-drinkers may not be causal, as is very likely for sick quitters, the excess mortality for heavy drinkers may well be causal. If so, some of this 50% excess vascular mortality among the 1 in 10 older men who drank more than 28 units per week could have been avoided.

034 LIGHT DRINKING DURING PREGNANCY: STILL NO RISK FOR SOCIOEMOTIONAL DIFFICULTIES OR COGNITIVE DEFICITS AT 5 YEARS OF AGE? FINDINGS FROM THE UK MILLENNIUM COHORT STUDY

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Background: We recently reported that light alcohol consumption during pregnancy was not associated with an increased risk of behavioural difficulties or cognitive deficits at 3 years of age. However, it is not clear whether these associations remain constant or change over time.

Objective: To examine the relationship between light drinking during pregnancy and the risk of socio-emotional problems and cognitive deficits at age 5 years.

Design and Setting: Data from sweeps 1 and 3 of the nationally representative prospective UK Millennium Cohort Study were used.

Participants: 11 512 white singleton cohort members born in 2000–2002.

Comparison Groups: Cohort members were grouped according to mothers' reported alcohol consumption during pregnancy: abstainer; light, not more than 1–2 units per week or per occasion; moderate, not more than 3–6 units per week or 3–5 units per occasion; heavy/binge, 7 or more units per week or 6 or more units per occasion.

Main Outcome Measures: At age 5 the Strengths and Difficulties Questionnaire (SDQ) and British Ability Scales (BAS) tests were administered during home interviews. Behavioural problems were indicated by scores falling above defined clinically relevant cut-offs on the SDQ. Standardised scores for the BAS Naming Vocabulary, Pattern Construction and Picture Similarities scales were used.

Results: Boys and girls born to light drinkers were less likely to have high total difficulties (for boys 6.6 vs 10.0%, OR 0.65, for girls 4.2 vs 6.3%, OR 0.67) and hyperactivity (for boys 10.1 vs 13.7%, OR