years, with secondary prevention patients automatically considered high risk. The guidelines in Scotland, and elsewhere, state that statins should be offered in secondary prevention regardless of their plasma lipid concentration and that annual monitoring of lipid concentration represents good practice. This descriptive analysis investigated the demographics of a secondary prevention population, their estimated adherence to statin medication and whether targets for cholesterol reduction were achieved.

Methods Data was extracted from the NHS Greater Glasgow and Clyde (NHS GGC) Safe Haven for all patients with a record of a non-fatal myocardial infarction (MI) occurring between 1st March 2008 and 1st March 2014, together with their demographics, lipid concentrations, and statin dispensing records up to 1st March 2017. Estimated adherence for each year post MI was calculated from the dispensing records using the Medication Possession Ratio (MPR) and averaged across the years available for each patient. Descriptive statistics were presented using raw counts and percentages, and means and standard deviations, for categorical and continuous variables respectively. Logistic regression examined associations between adherence and cholesterol targets, and adherence and mortality, and were also adjusted for age, sex, year of MI and deprivation quintile, with results presented as odds ratios (OR).

Results In the population, 11,568 patients had a previous non-fatal MI, and were predominantly male (7,002, 60.5%), had a mean age at admission of 66.9 years (SD: 13.9), and approximately a third died before 1st March 2017 (4,053, 35.0%). 10,469 patients had at least one year's follow-up allowing average adherence to be estimated and one third (3,360, 32.1%) had an average statin adherence <80%. Patients with <80% adherence were associated with lower odds of achieving target 40% non-HDL reduction (OR: 0.387 [95% CI: 0.336, 0.446]), which were unaltered after adjustment (OR 0.392 [95% CI: 0.339, 0.452]). These patients were also associated with higher odds of mortality (OR: 1.717 [95% CI: 1.571, 1.877]), with some attenuation following adjustment (OR: 1.653 [95% CI: 1.486, 1.839]).

Conclusion In the NHS GGC post MI population, patients with lower statin adherence were associated with lower odds of achieving cholesterol targets set by current guidelines and higher odds of mortality. However, a large proportion of patients achieved targets and had excellent adherence, questioning the need for regular lipid monitoring.

OP17

BIOPSYCHOSOCIAL DETERMINANTS OF CARDIOVASCULAR AND DEMENTIA RISK: AN EXAMINATION OF DIFFERENTIALS IN A REPRESENTATIVE STUDY OF THE ENGLISH POPULATION

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10.1136/jech-2019-SSMabstracts.17

Background Growing evidence supports a strong and likely causal association between cardiovascular disease (CVD) and dementia incidence. Several cardiometabolic risk factors including smoking, sedentary behaviour, hypertension, and diabetes are common to both conditions, but less clear is the predictive value of inflammatory markers, alcohol consumption or depression. We investigated the congruity and difference across 10 CVD and dementia risk factors in a representative cohort of the English population.

Methods Data come from the English Longitudinal Study of Ageing, an ongoing, open, prospective cohort study. CVD was defined as the fatal and non-fatal myocardial infarction, angina pectoris and stroke. Dementia was determined using doctor-diagnosis combined with a score above the threshold of 3.38 on the Informant Questionnaire on Cognitive Decline in the Elderly. A triangulation method was also used to derive these outcomes using the Hospital Episode Statistics (HES) records. We investigated 10 risk factors: alcohol, smoking, sedentary behaviour, hypertension, diabetes, depressive symptoms, obesity (defined as ≥ 30 body mass index (BMI)), HDL/total cholesterol and inflammatory markers (serum fibrinogen and C-reactive protein [CRP]) in 3,981 men and women, free of CVD or dementia and aged 50+ at baseline (2008–09). Multinomial logistic regression models were fitted to estimate the relationship (relative risk ratios [RRR] and 95% confidence intervals [CI]) between each factor, and the risk of CVD, dementia or both.

Results From the analytical sample, 13% developed CVD, 4% dementia and 1.5% both CVD and dementia during the eight-year follow-up period. After controlling for sociodemographic factors (age, sex, education and wealth), we found that hypertension (RRR=1.66, 95% CI 1.35–2.04), depressive symptoms (RRR=1.55, 95% CI 1.15–2.08), and obesity (RRR=1.37, 95% CI 1.03–1.83) were predictive of an increased risk of CVD; smoking (RRR=2.04, 95% CI 1.14–3.65) was associated with a higher risk of dementia; whilst smoking (RRR=2.34, 95% CI 1.03–5.27) and depressive symptoms (RRR=4.03, 95% CI 2.12–7.66) were predictive of developing both CVD and dementia. We found no associations between inflammatory markers, alcohol consumption or sedentary behaviour and these outcomes.

Conclusion Our findings indicate that smoking and depressive symptoms are associated with an increased risk of developing both CVD and dementia in later life, while obesity and hypertension are specific to CVD. There is relatively limited congruency across the predictive values of the 10 biopsychosocial risk factors investigated for these conditions. However, larger studies with longer periods of follow-up are necessary to extend these findings.

OP18

WHAT EXPLAINS THE EFFECT OF EDUCATION ON CARDIOVASCULAR DISEASE? APPLYING MENDELIAN RANDOMISATION TO IDENTIFY THE CONSEQUENCES OF EDUCATION INEQUALITY

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10.1136/jech-2019-SSMabstracts.18

Background Studies have demonstrated causal effects of educational attainment on cardiovascular disease (CVD). We aimed to investigate the role of body mass index (BMI), systolic blood pressure (SBP) and smoking in explaining the effect of education on risk of CVD outcomes triangulating across multi-variable regression analysis of observational data and one- and

two-sample Mendelian randomisation (MR) analysis; an instrumental variable approach more robust to bias from confounding and reverse causation.

Methods Individual level data from UK Biobank (N=217 013) was used for multivariable analyses and one-sample MR. Summary statistics from genome-wide association studies were used in two-sample MR.

The total effect of education on risk of coronary heart disease (CHD), CVD (all subtypes), myocardial infarction (MI) and stroke (all measured in odds ratio, OR) was assessed using multivariable regression and univariable Mendelian randomization (MR).

The degree to which this effect is mediated through BMI, SBP and smoking respectively (the indirect effect and proportion mediated) was estimated using the product of coefficients method, where the effect of education on each mediator, and each mediator on each outcome was assessed using multivariable regression and Network MR. The joint contribution of all three risk factors was assessed via the difference method, using multivariable regression or multivariable MR.

Results Each additional standard deviation of education (3.6 years) associated with 13% lower risk of coronary heart disease (OR 0.87, 95% confidence interval [CI] 0.84 to 0.89) in observational analysis and 37% lower risk (OR 0.63, 95% CI 0.60 to 0.67) in MR analysis. As a proportion of the total risk reduction, BMI mediated 15% (95% CI 13% to 17%) and 18% (95% CI 14% to 23%) in the observational and MR estimates respectively. Corresponding estimates for SBP were 11% (95% CI 9% to 13%) and 21% (95% CI 15% to 27%), and for smoking, 19% (15% to 22%) and 34% (95% CI 17% to 50%). All three risk factors combined mediated 42% (95% CI 36% to 48%) and 36% (95% CI 16% to 63%) of the effect of education on CHD in observational and MR respectively. Similar results were obtained for risk of stroke, MI and all-cause CVD.

Conclusion BMI, SBP and smoking mediate a substantial proportion of the protective effect of education on risk of cardiovascular outcomes. Intervening on these would reduce cases of CVD attributable to lower education. However, more than half of the protective effect of education remains unexplained.

OP19 WILL SOCIAL CARE NEED MORE RESOURCES? A MODELLING STUDY OF HEALTH AND SOCIAL COSTS IN ENGLAND AND WALES FOR ALTERNATIVE FUTURE CARDIOVASCULAR DISEASE SCENARIOS

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10.1136/jech-2019-SSMabstracts.19

Background Cardiovascular disease (CVD) contributes to dementia and disability risk. It also affects the cost of care. The English NHS long-term plan targets preventing 150,000 CVD events from 2019–2029. However, after decades of declines in CVD mortality in England, CVD mortality improvements have slowed since 2011, which may indicate a slowdown in incidence reduction from around 2006. Therefore, there is uncertainty about how CVD burden and

associated health and social care costs might evolve in the next decade.

Methods Simulations for people aged 35–100 in England and Wales were carried out using the IMPACT Better Aging Model (BAM), an open-cohort, stochastic Markov model which synthesises observed trends in CVD incidence and mortality, dementia and disability in the English Longitudinal Study of Ageing (ELSA) and national ONS data. The synthesised trends were projected to 2029.

We modelled undiscounted health and social care costs and quality adjusted life years (QALYs) for 2019–2029 under two scenarios:

1. Basecase – age-specific CVD incidence continue to decline, following the long-term trends;
2. Age-specific CVD incidence do not decline after 2006, following recent trends.

Healthcare costs were based on hospital episode statistics (HES) data, matched to ELSA participants and calibrated to Office for Budget Responsibility healthcare cost estimates. Age-related social care costs were estimated using reported social care contact hours from ELSA combined with PSSRU unit costs. Utility weights for QALYs were from EQ-5D MEPS catalogue and Health Survey for England.

Results In the basecase scenario 1, median healthcare costs (2019 prices) are projected to increase by ~12% between 2019–2029, from £93.0bn to £104.6bn per year. Social care costs are projected to increase by ~27%, from £8.0bn to £10.2bn per year.

In the CVD flat-lining scenario 2, median healthcare costs increased by ~15% from £95.3bn in 2019 to £109.6bn in 2029, and social care costs increased by ~30% from £8.2bn in 2019 to £10.7bn in 2029.

When compared with scenario 2, the basecase scenario would generate ~200,000 additional QALYs/year by 2029, which, valued at UK Treasury rate, would be worth some £12billion per year.

Conclusion This study projects future health and social care costs resulting from the recent slowdown in CVD incidence and mortality declines. We predict that social care costs will grow twice as fast as healthcare costs over the next decade, regardless of future improvements. Total funding policy therefore need to be urgently addressed, which may prove politically challenging.

OP20 CARDIOVASCULAR DISEASE BIOMARKER PROFILES AMONG HARMFUL AND HAZARDOUS DRINKERS: A CROSS-SECTIONAL STUDY FROM NORTH WEST RUSSIA

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10.1136/jech-2019-SSMabstracts.20

Background Heavy alcohol drinking is increasingly recognized as a risk factor for cardiovascular disease (CVD), although the mechanisms underlying this are not well understood. Moderate alcohol consumption is associated with changes in many blood biomarkers of cardiometabolic risk. There are however few studies of the impact of harmful and hazardous drinking on