Ageing and cardiovascular disease

OP21 PREDICTION OF FUTURE ISCHEMIC STROKE TRENDS IN SWEDEN T0 2030: A MODELLING STUDY

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10.1136/jech-2018-SSMabstracts.21

Background The burden of cardiovascular disease and death has plummeted over recent decades in Sweden and comparable countries. But might it soon increase as a result of population aging? Few studies have formally investigated future stroke trends. The aim of this study was therefore to quantify the future burden of ischemic stroke in Sweden until 2030.

Methods We developed and validated a discrete open cohort Markov model for ischemic stroke (IS) in the Swedish population. We used population data from Statistics Sweden, the Swedish inpatient registry and cause-specific death registries to calculate IS prevalence and absolute numbers from 2000 to 2010. We then estimated future trends in IS incidence and mortality rates until 2030 using a BAPC (Bayesian aged-period cohort) approach. We also conducted sensitivity analyses to better quantify uncertainty around model inputs and outputs.

Results Overall IS prevalence was predicted to decrease by approximately 20% between 2010 and 2030, from 170 to 137 per 10 000 population and from 183 to 157 per 10 000 in men and from 157 to 116 per 10 000 in women.

During the same period, the overall number of IS patients might fall by just 3%, from 1 27 000 in 2010 to 1 23 000 in 2030 (decreasing from 59 000 to 52 000 in women, and increasing modestly in men, from 68 000 to 71,000).

Worryingly, the prevalence of IS in young people aged <45 years was predicted to increase from 5.4 to 6.7 per 10 000 population (the number of IS patients correspondingly increasing from 1970 to 2,610).

IS prevalence in elderly people aged >85 years was predicted to fall by a third, from 1180 to 800 per 10 000 population. However, the actual number of IS patients might increase from 29 470 to 31,800, reflecting a growing elderly population.

Conclusion Ischaemic stroke prevalence in Sweden might well fall by 20% between 2010 and 2030. However, the overall number of IS patients could remain above 1 20 000 per year. More worrying still, IS cases among the older citizens will increase due to population ageing, as will morbidity among the youngest groups. To reduce the future burden of stroke, Sweden needs a more ambitious and comprehensive prevention strategy.

OP22 ALLOSTATIC LOAD IS ASSOCIATED WITH CORONARY HEART DISEASE, BUT NOT WITH DEMENTIA: EVIDENCE FROM A 12-YEAR FOLLOW-UP IN THE ENGLISH LONGITUDINAL STUDY OF AGEING

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10.1136/jech-2018-SSMabstracts.22

Background Allostatic load (AL) has been proposed as a conceptualisation of cumulative biological burden on the body that emerges through attempts to adapt to life's demands. Using a multisystem summary measure of AL, we evaluated its associations with subsequent coronary heart disease (CHD) and dementia.

Methods The data used for these analyses are from 4335 men and women aged ≥50 years at recruitment from the English Longitudinal Study of Ageing (ELSA), an ongoing, representative prospective cohort study. Seven waves of data between 2004/2005 (wave 2) and 2016/2017 (wave 8) were analysed. CHD events were defined as the fatal and non-fatal myocardial infarction (MI) and Angina occurring after the study entry. Dementia was determined by doctor-diagnosis combined with a score above the threshold of 3.38 on the Informant Questionnaire on Cognitive Decline in the Elderly. The AL index included 4 biomarker risk groups covering cardiovascular (systolic and diastolic blood pressure, pulse rate), metabolic (total cholesterol-to-HDL ratio, Hba1c, triglycerides), immune (CRP, fibrinogen) and anthropometric systems (waist to height ratio, underweight), measured at wave 2. Each biomarker was grouped into high (1) vs low (0) risk. Except for underweight, the highest gender-specific quartile of the distribution for each biomarker was scored with 1. Multivariable logistic regressions were used to estimate the associations between the AL index and subsequent CVD or dementia prevalence, while controlling for confounders (age, sex, marital status, education, wealth) and potential mediators (alcohol, smoking, fruit and vegetable consumption, and physical activity).

Results From the overall sample, 11% developed CHD and 8% dementia during 12 year study period. After controlling for sociodemographic factors, we found that an increase in the AL index was associated with a higher risk of CHD (Odds Ratio (OR)=1.13 (95% Confidence Intervals (CI) 1.06–1.20)); but not with dementia (OR)=1.03 (95% CI) 0.97 to 1.10)). Further adjustment for the role of lifestyle behaviours slightly attenuated the association with CHD (OR)=1.09 (95% CI) 1.02 to 1.17), but did not explain it fully.

Conclusion Our results showed that a higher cumulative physiological burden represents a predictor of subsequent CHD, supporting the hypothesis that a cumulative measure of 'biological dysregulation' could act as an early determinant of atherosclerosis and CHD. However, our results do not indicate that the cumulative biological risk plays a pivotal role in the aetiology of dementia. The fact that dementia is slighlty underestimated in this study, may mask the real association with specific metabolic and inflammatory markers.

OP23 ASSOCIATIONS BETWEEN LIFETIME HAZARDOUS DRINKING AND ASSOCIATIONS BETWEEN LIFETIME HAZARDOUS DRINKING AND BIOMARKERS OF CARDIOMETABOLIC HEALTH AND LIVER FUNCTION AMONG OLDER ADULTS: FINDINGS FROM THE WHITEHALL II PROSPECTIVE COHORT STUDY

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10.1136/jech-2018-SSMabstracts.23

Background Hazardous drinking among older adults is a growing concern, however there is limited research on the effect of chronic versus acute hazardous drinking among older people, and how the effects vary across life. This study among older adults, explores the association of positive AUDIT-C

scores across life with objective biomarkers of cardio-metabolic health.

Methods Analyses were undertaken among 4820 civil servants aged 59–83 years, within the prospective Whitehall II study, who answered the life-grid AUDIT-C inventory during the 2011–2012 survey and provided biological measurements (264 non-drinkers were excluded). Lifetime hazardous drinking groups were defined using a threshold of ≥5, at each decade of life from age 16 to 80+. These groups were as follows; never hazardous-drinker (reference), former hazardous-drinker$_1$ (before age 50), former hazardous-drinker$_2$ (after age 50), current hazardous-drinker (past hazardous-drinker sporadically), stable hazardous-drinker (hazardous-drinker in every decade). Similar groups were created for lifetime binge-drinking categories; never/former/current/stable binge-drinker (AUDIT-3 ≥2). Fully-adjusted linear regression was carried out on cardio-metabolic biomarkers including: waist circumference (WC, measured in cm), body mass index (BMI, kg/m^2), total cholesterol (TC, mmol/L), systolic (SBP, mmHg) and diastolic (DBP, mmHg) blood pressure, gamma-glutamyl transferase (GGT), fatty-liver index scores (FLI) and lifetime hazardous/binge drinking as exposure, using STATA15. Covariates included sex, age, socio-economic position, ethnicity, smoking status, physical activity, BMI and fruit and vegetable consumption.

Results Over half of the sample had been a hazardous-drinker at some point; Current hazardous-drinkers (21%), former hazardous-drinkers$_1$ (<age 50) (19%), former hazardous-drinkers$_2$ (≥age 50) (11%) stable hazardous-drinker (5%). After adjusting for co-variates, hazardous-drinkers had a larger WC than never hazardous-drinkers (former hazardous-drinkers$_1$ (β=1.17 [95% CI 0.25, 2.08]); (former hazardous-drinkers$_2$ (1.88 [95% CI 0.77, 2.98]); current hazardous-drinkers (2.44 [1.55, 3.34]) and stable hazardous-drinkers (3.85 [2.23, 5.47])). A similar linear association along more current and frequent hazardous-drinking was also found with BMI. Current hazardous-drinkers had higher SBP (2.44 [1.19, 3.68]), log (GGT) (22.64 [18.27,27.02]) and FLI scores (4.05 [2.92, 5.18]) than never hazardous-drinkers, and so did stable hazardous-drinkers (sbp (2.78 [0.53, 5.04]), log(GGT) (17.94 [10.12, 25.75]), FLI (3.76, [1.75, 5.77])). Similar associations with waist, sbp, GGT, and FLI outcomes were found for lifetime binge-drinkers.

Conclusion Hazardous-drinking is common among older adults and may increase cardio-metabolic risk factors, this may be compounded by persistent hazardous-drinking across life. Population reductions in hazardous-drinking is likely to have immediate improvements in elderly, but also long lasting improvements with early intervention in the life course, particularly with weight gain. Future analyses will assess risk of lifetime hazardous-drinking and cardiovascular events and mortality.

OP24 SUBCLINICAL CARDIOVASCULAR DISEASE AND FRAILTY AMONG BRITISH OLDER MEN WITHOUT A DIAGNOSIS OF CARDIOVASCULAR DISEASE: A POPULATION-BASED STUDY

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10.1136/jech-2018-SSMabstracts.24

Background Frailty is associated with incident cardiovascular disease (CVD). However, its association with subclinical CVD, which precedes the onset of a CVD event, remains uncertain. Non-invasive vascular markers provide valuable proxy indicators of CVD risk, permitting investigation of risk burden and the development of subclinical CVD. N-terminal pro brain natriuretic peptide (NT-proBNP) and cardiac troponin T (cTNT) are markers of cardiac injury and are strong risk predictors of CVD events such as heart failure and myocardial infarction respectively. Our aim was to examine the association of subclinical cardiovascular markers with frailty in men without a diagnosis of CVD.

Methods In 2010–2012, 1622 surviving men aged 71–92 years were examined and completed a questionnaire as part of a cross-sectional study from a cohort of men from 24 British towns initially recruited in 1978–1980. Using the Fried phenotype, frailty was defined by the presence of ≥3 of the following components: unintentional weight loss, low grip strength, low physical activity, slow walking pace and exhaustion. Carotid intima media thickness (CIMT) and carotid distensibility coefficient (DC) were measured using ultrasound. A Vicorder device was used to measure pulse wave velocity (PWV) and ankle brachial pressure index (ABPI). Fasting blood samples were analysed for NT-proBNP and cTNT. Multivariable logistic regression was used for analyses.

Results Three hundred and three participants (19%) were frail and 876 (54%) were pre-frail. In men without CVD, frail individuals were significantly more likely to be older, physically inactive, have a smoking history and co-morbidities such as peripheral vascular disease and diabetes compared to non-frail individuals ($p<0.05$ for all associations). Multivariable analyses of baseline demographics and diabetes showed that frailty was positively and significantly associated with high NT-proBNP (odds ratio [OR]=3.48; 95% confidence interval [CI]=2.13–5.67), high cTNT (OR=3.33; 95% CI 2.03 to 5.45) and low DC (reflecting greater arterial stiffness; OR=1.79; 95% CI 1.11 to 2.88). Some subclinical vascular markers (ABPI <0.9 or >1.2, PWV and CIMT) were not associated with frailty ($p>0.05$). Pre-frailty was also associated with raised cardiac markers ($p<0.05$ for all associations).

Conclusion Frailty is strongly associated with subclinical CVD markers (NT-proBNP, cTNT and DC), even in the absence of prevalent CVD. Further research is needed to determine whether this association is causal and to understand the underlying mechanisms. Our study findings support the need for screening older individuals with frailty, with the objective of identifying those with an underlying increased risk of CVD and its adverse outcomes.

OP25 SOCIOECONOMIC FACTORS ASSOCIATED WITH FRAILTY: RESULTS FROM TWO STUDIES OF OLDER BRITISH POPULATIONS

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10.1136/jech-2018-SSMabstracts.25

Background Frailty is a state of increased vulnerability to stressors in older age, which increases risks of disability, falls and mortality. Prevalence of frailty is very high in older populations. The extent to which socioeconomic factors are