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

Authors: Dorina Cadar[1], Jessica Abell[1], Ruth A. Hackett[1], Andrew Steptoe[1]

[1] Department of Behavioural Science and Health, University College London, UK

[2] Mental Health Research Group, University of Exeter Medical School, University of Exeter, UK

[3] Department of Epidemiology and Public Health, University College London, UK

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 4,335 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 2004/05(wave 2) and 2016/17 (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) 0.97-1.10)). Further adjustment for the role of lifestyle behaviours slightly attenuated the association with CHD (OR)=1.09 (95% CI) 1.02-1.17), but did not explain it fully.

Conclusions:

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.

Keywords: Allostatic load, Coronary Heart Disease, Dementia