Methods Quality and Outcomes Framework (QOF) electronic records until May 2016 were linked to mortality data. COPD Prevalence was calculated by sex, age group (10-year age bands), and SES using SIMD quintiles. Smoking status (ever smoked and current smoker) was also collected by the QOF. Population estimates for smoking status by age sex and SIMD for GGC were calculated using three Scottish Household Survey rounds, 2013, 2014 and 2015. COPD prevalence rates by SIMD quintile were calculated, adjusting for age, sex, and smoking status.

Results Crude prevalence of COPD among all ages in GGC is 2.74% and among those aged 40 years+ in GGC was 5.67%, higher in females 5.95% than males, 5.36%. Comparing prevalence of COPD between males and females, rates were higher for males until age 39 and equivalent for ages 40–49 years. However, for 50–59 year olds prevalence among females was 3.84 compared with 3.15 among males, and for 60–69 year olds, prevalence was 8.15% for females compared with 7.26% for males. Thereafter prevalence was greater among males; for 70–79, 80–89 and 90+ years, prevalence among males was 11.81%, 12.03% and 7.56% respectively, compared with 11.76%, 10.58% and 6.38% among females. Prevalence of COPD in SIMD 1 (most deprived) was almost 3.5 times of that in SIMD 5 (least deprived). Adjusting for age and sex, SES inequalities in COPD increased with SIMD1 prevalence 4.8 times that of SIMD5. After adjustment for age sex and ever smoked, SIMD1 prevalence was 3.1 times that of SIMD5. After adjustment for age, sex and current smoking, SIMD1 prevalence was 2.45 times that of SIMD5.

Conclusion Prevalence of COPD in GGC is higher than previously estimated. It is also higher among females than males at ages 50–70 years. Inequalities in COPD are evident and become greater on adjustment for age and sex. Smoking accounts for around half of the gap in prevalence of COPD between most and least deprived, however inequalities in COPD persist after adjustment for smoking status.

LIFE COURSE SOCIOECONOMIC CIRCUMSTANCES AND DEMENTIA PREVALENCE: EVIDENCE FROM THE ENGLISH LONGITUDINAL STUDY OF AGEING

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Background Dementia represents a major public health impact. Previous work showed that higher socio-economic status (SES) is protective against dementia, through mentally engaging and socially interactive occupations and activities. However, the associations with childhood SES and social mobility are not well understood. We examined four SES indicators (father's social class, own education, occupational class and wealth) as well as social mobility across life, in relation to dementia prevalence.

Methods The data used are from 2032 men and women aged ≥65 years at recruitment, from the English Longitudinal Study of Ageing (ELSA), an ongoing, representative prospective cohort study. Seven waves of data between 2002/03(wave 1) and 2014/15 (wave 7) were analysed. 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. Education has been grouped into university degree, A-levels and no education, and occupation into professional, intermediate, routine and manual. Wealth (property, savings, or other financial assets) was divided in quintiles, while social mobility was derived by combining childhood SES and adult occupational class into three stable SES levels (low, medium, high), and upwards or downwards trends. The highest SES indicator or stable-high were used as reference groups. Multivariable logistic regressions were employed to estimate the associations between each baseline SES indicator and dementia prevalence by wave 7, while controlling for age, sex, marital status, long-standing limiting illness, and subsequent gradual adjustment for all other SES markers.

Results During the 12-year follow-up, 25% of sample developed dementia. Lower childhood SES was associated with a higher dementia risk (Odds Ratio (OR)=1.39 (95% Confidence Intervals (CI) 1.02–1.89), but explained by education. Education did not show a protective effect, but the lower occupational class was associated with higher dementia risk (OR=1.39 (95% CI) 1.02 to 1.89). However, this association was subsequently explained by wealth. Lowest wealth was a strong predictor of dementia, independent of other SES markers (OR=2.81 (95% CI) 1.83 to 4.32) in contrast to the wealthiest counterparts. Those in the stable-low category across life showed a higher risk (OR=1.65 (95% CI) 1.01 to 2.68) in contrast with those in stable-high SES. Upward or downward SES mobility trends did not show an impact.

Conclusion In an English, nationally representative sample, the incidence of dementia appeared to be socioeconomically patterned, primarily by the level of wealth and long-term SES disadvantage. Public health strategies for dementia prevention should target the socioeconomic gap to reduce health disparities and protect those who are particularly disadvantaged.

DOES MOVING INTO SOCIAL, INTERMEDIATE AND MARKET-RENT ACCOMMODATION IN EAST VILLAGE (THE FORMER LONDON 2012 OLYMPIC ATHLETES VILLAGE) IMPROVE SELF-RATED MENTAL HEALTH, WELL-BEING AND NEIGHBOURHOOD PERCEPTIONS? EVALUATION OF A NATURAL EXPERIMENT

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Background Evidence suggests that where we live might be an important determinant of mental health and well-being, especially amongst the more disadvantaged. However, longitudinal evidence is limited. The Examining Neighbourhood Activities in Built Living Environments in London (ENABLE London) study aimed to establish whether mental health, well-being, and neighbourhood perceptions improved among adults relocating to East Village, purposely designed for healthy active living, when compared with a control population who lived outside East Village throughout.

Methods During 2013–2015, 1278 adults seeking accommodation in East Village were recruited (participation rate 70%, n=1278/1819); 520 were seeking social housing, 524 intermediate (affordable) and 234 market-rent accommodation. Participants were followed-up after 2 years; those who moved into East Village formed the intervention group, and those who did not move to East Village controls. Self-reported mental-health (depression, anxiety), subjective well-being (life satisfaction, worthwhile, happiness), and neighbourhood perceptions (quality and crime-free) were assessed by questionnaire. Multilevel linear regression models examined change in these outcomes adjusted for age, sex, ethnicity and household (random effect), comparing those in the intervention group with controls, overall and by housing sector.

Results Of 1278 adults recruited at baseline, 877 (69%) adults were followed-up after two years; half (n=440/877) had moved to East Village. There were marginally lower levels of depression and anxiety amongst those who moved to East Village, compared with those who did not, but differences were not statistically significant. Overall levels of positive well-being (including life satisfaction, worthwhile, happiness) were marginally higher amongst participants who moved to East Village, but again differences were not statistically significant. However, increases in life satisfaction and happiness amongst those living in intermediate accommodation in East Village were stronger (p=0.01, p=0.05 respectively). The most marked differences were in neighbourhood perceptions, where sizeable increases in both quality and crime-free status were observed amongst those living in East Village compared with those who were not, overall and in each housing sector (all P values <0.01). There was also the suggestion that improvements in crime-free perceptions were stronger in the social and intermediate sectors compared to those in market-rent accommodation (test for interaction, p=0.04).

Conclusion East Village had modest effects on measures of well-being, but appreciable effects on neighbourhood perceptions. Longer-term exposure to better neighbourhoods could plausibly have beneficial effects for health, particularly for both mental health and well-being, more so among those from less privileged circumstances, who potentially have the most to gain.

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Background Systemic inflammation has been associated with the onset of depressive symptoms. However, the exact mechanisms underlying the relationship between inflammation and depression remain elusive. This study examined whether sleep explained the association between elevated levels of inflammatory markers and subsequent depressive symptoms in an English nationally representative sample.

Methods The sample consisted of 2953 men and women (aged 50+) recruited from the English Longitudinal Study of Ageing (ELSA) an ongoing, open, representative prospective cohort study. Four waves of data between 2008/09 (wave 4) and 2016/17 (wave 8) were analysed. Serum levels of inflammatory markers (C-reactive protein (CRP)) and covariates (age, sex, education, wealth, body mass index, smoking, cholesterol, triglyceride) were measured at wave 4 (considered here as the baseline). Self-reported sleep disturbance (vs no sleep disturbance) was examined via three items of the Jenkins Sleep Problems Scale (difficulty falling asleep, trouble remaining asleep and morning tiredness) at a four-year follow-up (wave 6, 2012/13). Depressive symptoms were assessed at baseline and six years later (wave 7, 2014/15) using the 8-item version of the Centre for Epidemiological Studies Depression Scale (CES-D) (excl. the item on sleep). Binary mediation analysis was used to investigate whether sleep mediated the relationship between systemic inflammation and depressive symptoms, adjusting for the full set of covariates.

Results High baseline levels of CRP were significantly associated with greater odds of subsequent depressive symptoms, independent of age, sex and baseline depressive symptoms (Odds Ratio (OR)=1.32 (95% Confidence Intervals (CI)) 1.02–1.70). Further adjustment for socio-economic variables (education, wealth status) attenuated this relationship to non-significance (OR=1.17 (95% CI) 0.90 to 1.53). Moreover, high CRP levels at baseline were significantly related to higher odds of reporting sleep disturbance at wave 6 (OR=1.44 (95% CI) 1.14 to 1.82). Sleep disturbance was associated with greater odds of subsequent depressive symptoms (OR=2.69 (95% CI) 2.03 to 3.57). Mediation analyses revealed that sleep problems mediated the relationship between high CRP and depressive symptoms, explaining a total of 65.61% of this association.

Conclusion Our results showed that sleep acted as a strong partial mediator of the relationship between elevated levels of CRP and subsequent depressive symptoms in an English nationally representative sample. Targeting sleep disturbance via tailored interventions may be effective in alleviating inflammation-associated depressive symptoms.

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Background Associations between depression and cardiovascular disease have been reported in a number of studies. However, in many of these, the results have not been adjusted for potentially important confounding factors. With the large amount of data collected, the UK Biobank offers a unique opportunity to investigate to what extent different measures of depression remain independent risk factors for major cardiovascular events (MCVE) after controlling for a number of potential confounding factors.

Methods We used data from 275,759 UK Biobank participants without a history of cardiovascular diseases and major mental disorders other than depression who had complete data available. In primary analyses, depression was defined as one or more of self-reported depression, antidepressant use, or hospital admission for depression. In secondary analyses, the effects of each of the subcategories of depression were analysed separately. MCVE were defined as first-ever fatal or non-fatal stroke or myocardial infarction ascertained from hospital