Social Mobility and Inflammatory and Metabolic Markers at Older Ages:

The English Longitudinal Study of Ageing

Supplementary material

SUPPLEMENTARY METHODS

Data source and studied population: non-response analysis

In a non-response analysis (Table S1), non-responders defined as those who had missing values on, at least, one outcome variable were more likely to be older, more obese, have lower SEP, current smokers, less physically active, more frequent alcohol drinkers, have elevated depressive symptoms, and have a higher prevalence of both cardiovascular and non-cardiovascular comorbidities than responders who had all outcome measurements (all p-value < 0.001). This non-responder profile was consistent with previous ELSA reports on attrition and non-response, in which the attrition in studies was related to older age, increased morbidity, elevated depressive symptoms, and lower SEP.[1-3] Moreover, responders were likely to have more favourable inflammatory and metabolic profiles characterised by lower levels of CRP, fibrinogen, and HbA1c, and higher levels of HDL, compared with those in non-responders.

Table S1. Non-response analysis

Characteristics	Non-responders 4,060 (46.2)	Responders 4,720 (53.8)	Total 8,780 (100)	P-value[a]
Women	2,217 (54.6)	2,614 (55.4)	4,831 (55.0)	0.53
Age (year)				
Mean (SD)	67.82 (9.68)	65.42 (9.15)	66.52 (9.48)	< 0.001
Median (IQR)	67 (59-75)	64 (58-72)	65 (58-74)	< 0.001
BMI (kg/m^2)				
Mean (SD)	28.80 (5.40)	27.40 (4.48)	27.93 (4.89)	< 0.001
< 25 kg/m^2	641 (15.8)	1367 (29.0)	2008 (22.9)	< 0.001
25 – 30 kg/m^2	1087 (26.8)	2045 (43.3)	3132 (35.7)	
> 30 kg/m^2	972 (23.9)	1113 (23.6)	2085 (23.7)	
Missing	1360 (33.5)	195 (4.1)	1555 (17.7)	
Waist (cm)				
Mean (SD)	93.15 (25.82)	92.12 (18.30)	92.52 (21.51)	0.058
Normal waist	492 (12.1)	1148 (24.3)	1640 (18.7)	< 0.001
Increased risk	624 (15.4)	1313 (27.8)	1937 (22.1)	
Substantially increased	1677 (41.3)	2162 (45.8)	3839 (43.7)	
Missing	1267 (31.2)	97 (2.1)	1364 (15.5)	
Waist to hip ratio				
Mean (SD)	0.80 (0.45)	0.84 (0.29)	0.83 (0.36)	< 0.001
Normal	880 (21.7)	1,915 (40.6)	2,795 (31.8)	< 0.001
Increased risk	1,901 (46.8)	2,698 (57.2)	4,599 (52.4)	
Missing	1,279 (31.5)	107 (2.2)	1,386 (15.8)	

Characteristics	Non-responders	Responders	Total	P-value[a]
	4,060 (46.2)	4,720 (53.8)	8,780 (100)	
Social mobility				
Stable high	1,395 (36.8)	1,963 (43.8)	3,358 (40.6)	< 0.001
Low to high	536 (14.1)	714 (15.9)	1,250 (15.1)	
High to low	965 (25.5)	1,005 (22.4)	1,970 (23.8)	
Stable low	896 (23.6)	805 (17.9)	1,701 (22.5)	
Physical activity				
No on weekly basis	499 (12.6)	249 (5.3)	748 (8.6)	< 0.001
Mild	804 (20.3)	591 (12.5)	1,395 (16.0)	
Moderate	1,836 (46.3)	2,364 (50.1)	4,200 (48.4)	
Vigorous	828 (20.9)	1,514 (32.1)	2,342 (27.0)	
Alcohol consumption				
Daily/ almost daily	696 (17.1)	1,105 (23.4)	1,801 (20.5)	< 0.001
1-2 times/week	1,129 (27.8)	1,707 (36.2)	2,836 (32.3)	
1-2 times/month	633 (15.6)	801 (17.0)	1,434 (16.3)	
Never/ almost never	808 (19.9)	720 (15.2)	1,528 (17.4)	
Missing	794 (19.6)	387 (8.2)	1,181 (13.5)	
Smoking status				
Current smoker	724 (17.9)	605 (12.8)	1,329 (15.1)	< 0.001
Ex-smoker	1,956 (48.2)	2,307 (48.9)	4,263 (48.6)	
Never smoked	1,377 (33.9)	1,805 (38.3)	3,182 (36.3)	
CESD score ≥ 4				
Yes	758 (18.7)	582 (12.3)	1,340 (15.3)	< 0.001
No	3,126 (77.0)	4,093 (86.7)	7,219 (82.2)	
Missing	176 (4.3)	45 (1.0)	221 (2.5)	
Prevalent CVD	1,641 (40.4)	1,074 (22.8)	2,715 (30.9)	< 0.001

Characteristics	Non-responders 4,060 (46.2)	Responders 4,720 (53.8)	Total 8,780 (100)	P-value[a]
Prevalent non-CVD	2,503 (61.6)	2,590 (54.9)	5,093 (58.0)	< 0.001
Outcome variables				
CRP (ln mg/liter), mean (SD)	0.62 (0.85)	0.46 (0.90)	0.48 (0.90)	< 0.001
Fibrinogen (ln g/liter), mean (SD)	1.28 (0.25)	1.11 (0.20)	1.15 (0.22)	< 0.001
HbA1c (ln %), mean (SD)	1.70 (0.07)	1.69 (0.06)	1.70 (0.06)	< 0.001
HDL (ln mmol/liter), mean (SD)	0.30 (0.25)	0.41 (0.24)	0.39 (0.25)	< 0.001

n (%) unless specified elsewhere.

[a]One-way ANOVA and chi-square for continuous and categorical variables, respectively, and p-value did not take into account the effect of missing group.

Abbreviations: SD, standard deviation; CESD, Center for Epidemiology Studies Depression Scale; CVD, cardiovascular disease; non-CVD, non-cardiovascular disease; HbA1c, haemoglobin A, glycosylated; HDL, high-density lipoprotein cholesterol.

SUPPLEMENTARY DISCUSSION

Implication of our findings: how are health behaviours associated with social mobility?

To our knowledge, there are two main mechanisms that explain the association between social mobility and health behaviours. The social causation theory explains that social mobility influences health behaviours. For example, people who experience a downward social drift may encounter massive stress, which makes them engage in smoking and heavy alcohol drinking. In contrast, people who engage in heavy alcohol drinking may later confront a financial problem and mental illness, which result in a lower SEP, as they cannot study or work properly. This is known as the social (or health) selection theory.[4]

Although a combination of social causation and social selection processes may account for the association between SEP and health behaviours, the social causation theory is mostly endorsed,[4-13] and many studies simply define risky health behaviours as potential mediators for the association between life course SEP and clinical outcomes.[14, 15] A study by Burrow *et al.* showed that British men who upwardly mobile from manual background to the middle class were less likely to engage in risky health behaviours in terms of smoking and binge drinking, while those middle-class men who have remained within their class of origin have a greater odds of engaging in risky health behaviours. This would undermine a claim of the social selection process.[7] These findings are consistent with a study on alcohol use during adolescence, which shows that children on the downward drift in the family income trajectory, measured from 1 month of age through 15 years, were more likely to drink alcohol when they are 15 years.[6]

Social causation theory also explains the associations between social mobility and mental health and obesity. Heraclides *et al.* suggested that downward socially mobile women had a higher prevalence of overweight and obesity in adulthood than the socially stable of high SEP (52.0% vs. 36.1%). The results were still consistent, even after excluding participants, who had been obese as adolescents, to reduce the possibility of reverse causality.[5] Johnson *et al.* suggested that a low parental occupational status since offspring was born was significantly associated with an increased

risk for depressive disorders in adolescent children, and these findings support the social causation theory because of an apparent time sequence.[4]

Due to the fact that our study used cross-sectional data, we cannot establish a clear temporal sequence and draw a causal conclusion about the exact role of unhealthy behaviours. Therefore, our explanation, on which social mobility and health behaviours interplay with each other, is based mainly on previous literature. Although existing evidence is likely to support that health behaviours are mediators (social causation theory), further studies, using a prospective design and a large community sample, are still required to address this issue. Nonetheless, we need to take health behaviours into consideration when we investigate the association, between social mobility and clinical outcomes, as what has been done successfully in previous studies.[14-16]

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