

## **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 | Responders    | Total         | P-value <sup>a</sup> |
|---------------------------|----------------|---------------|---------------|----------------------|
|                           | 4,060 (46.2)   | 4,720 (53.8)  | 8,780 (100)   |                      |
| 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 <sup>2</sup> )  |                |               |               |                      |
| Mean (SD)                 | 28.80 (5.40)   | 27.40 (4.48)  | 27.93 (4.89)  | < 0.001              |
| < 25 kg/m <sup>2</sup>    | 641 (15.8)     | 1367 (29.0)   | 2008 (22.9)   | < 0.001              |
| 25 – 30 kg/m <sup>2</sup> | 1087 (26.8)    | 2045 (43.3)   | 3132 (35.7)   |                      |
| > 30 kg/m <sup>2</sup>    | 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 <sup>a</sup> |
|---------------------------------------|----------------|--------------|--------------|----------------------|
|                                       | 4,060 (46.2)   | 4,720 (53.8) | 8,780 (100)  |                      |
| <b>Social mobility</b>                |                |              |              |                      |
| 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) |                      |
| <b>Physical activity</b>              |                |              |              |                      |
| 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) |                      |
| <b>Alcohol consumption</b>            |                |              |              |                      |
| 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) |                      |
| <b>Smoking status</b>                 |                |              |              |                      |
| 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) |                      |
| <b>CESD score <math>\geq 4</math></b> |                |              |              |                      |
| 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 | Responders   | Total       | P-value <sup>a</sup> |
|------------------------------------|----------------|--------------|-------------|----------------------|
| Prevalent non-CVD                  | 4,060 (46.2)   | 4,720 (53.8) | 8,780 (100) | < 0.001              |
| <b>Outcome variables</b>           |                |              |             |                      |
| 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.

<sup>a</sup>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]

## REFERENCES

1. Pikhartova J, Blane D, Netuveli G. The role of childhood social position in adult type 2 diabetes: evidence from the English Longitudinal Study of Ageing. *BMC Public Health* 2014;**14**:505.
2. Demakakos P, Marmot M, Steptoe A. Socioeconomic position and the incidence of type 2 diabetes: the ELSA study. *Eur J Epidemiol* 2012;**27**(5):367-78.
3. James Banks, Elizabeth Breeze, Carli Lessof, et al. Retirement, health and relationships of the older population in England: ELSA 2004 (wave 2). Secondary Retirement, health and relationships of the older population in England: ELSA 2004 (wave 2) [Book] July 2006. <http://www.elsa-project.ac.uk/publicationDetails/id/3658>.
4. Johnson JG, Cohen P, Dohrenwend BP, et al. A longitudinal investigation of social causation and social selection processes involved in the association between socioeconomic status and psychiatric disorders. *J Abnorm Psychol* 1999;**108**(3):490-9.
5. Heraclides A, Brunner E. Social mobility and social accumulation across the life course in relation to adult overweight and obesity: the Whitehall II study. *J Epidemiol Community Health* 2010;**64**(8):714-9.

6. Poonawalla IB, Kendzor DE, Owen MT, et al. Family income trajectory during childhood is associated with adolescent cigarette smoking and alcohol use. *Addict Behav* 2014;**39**(10):1383-8.
7. Burrows R, Nettleton S. Going against the grain: smoking and 'heavy' drinking amongst the British middle classes. *Sociol Health Illn* 1995;**17**(5):668-80.
8. Catalano R, Dooley D, Wilson G, et al. Job loss and alcohol abuse: a test using data from the Epidemiologic Catchment Area project. *J Health Soc Behav* 1993;**34**(3):215-25.
9. Dodge KA, Pettit GS, Bates JE. Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Dev* 1994;**65**(2 Spec No):649-65.
10. Fenwick R, Tausig M. The macroeconomic context of job stress. *J Health Soc Behav* 1994;**35**(3):266-82.
11. Hamilton VL, Broman CL, Hoffman WS, et al. Hard times and vulnerable people: initial effects of plant closing on autoworkers' mental health. *J Health Soc Behav* 1990;**31**(2):123-40.
12. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav* 1995;**Spec No**:80-94.
13. Airaksinen J, Hakulinen C, Pulkki-Raback L, et al. Neighbourhood effects in health behaviours: a test of social causation with repeat-measurement longitudinal data. *Eur J Public Health* 2016;**26**(3):417-21.
14. Loucks EB, Pilote L, Lynch JW, et al. Life course socioeconomic position is associated with inflammatory markers: the Framingham Offspring Study. *Soc Sci Med* 2010;**71**(1):187-95.
15. Camelo LV, Giatti L, Neves JA, et al. Life course socioeconomic position and C-reactive protein: mediating role of health-risk behaviors and metabolic alterations. The Brazilian Longitudinal Study of Adult Health (ELSA-Brasil). *PLoS One* 2014;**9**(10):e108426.
16. Castagne R, Delpierre C, Kelly-Irving M, et al. A life course approach to explore the biological embedding of socioeconomic position and social mobility through circulating inflammatory markers. *Sci Rep* 2016;**6**:25170.