Objective To investigate how lifetime socioeconomic position (SEP) is associated with later-life crystallised cognitive function, accounting for different missing data mechanisms.

Participants A nationally representative population sample born in 1946 (MRC National Survey of Health and Development; NSHD, N = 5362), and a sample of British civil servants (Whitehall II; WHII, N = 10,308).

Methods Novel structured statistical approach to distinguish between accumulation and sensitive period life course models using SEP measures from childhood, early-adulthood and midlife. Results of complete case (CC) (assuming missing completely at random), multiple imputation (MI) (missing at random) and a Heckman selection model (missing not at random) were compared.

Outcomes National Adult Reading Test, age 53 (NSHD); Mill Hill Selection Test, age 55 (WHII); National Adult Reading Test, age 53 (NSHD); Mill Hill Selection Test, age 55 (WHII); National Adult Reading Test, age 53 (NSHD); Mill Hill Selection Test, age 55 (WHII).

Results NSHD: After adjusting for childhood cognitive function, the best fitting model was an accumulation model allowing SEP at each time point to have its own estimate. However, estimates varied by missing data method (women: childhood SEP: CC: coefficient = 1.11 (95% CI 0.15 to 2.06), MI: coefficient = 1.22 (95% CI 0.87 to 2.76), Heckman: coefficient = 0.70 (95% CI 0.38 to 1.78)). WHII (not adjusted for childhood cognition): the best fitting model represented accumulation in adulthood only, with childhood SEP not significant.

Conclusion Despite adjustment for childhood cognitive score, childhood SEP remains important in NSHD, whereas in Whitehall II childhood SEP was not associated with cognitive function. These differences may be due to recall bias of early SEP in WHII.

Genome wide association (GWS) studies have identified single nucleotide polymorphisms (SNPs) related to body mass index (BMI: kg/m²). Associations have also been reported between fetal malnutrition and BMI. We use the circumstances of the Dutch Hunger Winter of 1944–1945 to further examine these relations. We studied 348 adult men and women born in affected cities in the western Netherlands who had been exposed to famine during pregnancy, 294 born before or after the famine as time-controls, and 305 same-sex unexposed siblings of above groups as family controls. Mean age at examination was 58 years. We evaluated common variants in the FTO, TMEM18, MC4R, GNPDA2, DBNF, SEC16B, NECR1, SH2B1, SFRS10, MTCH2, and KCTD15 genes related to BMI. A genetic risk score was calculated for each individual by summing the number of risk alleles in these genes. Scores were also weighted using recent GWAS estimates of gene specific changes in BMI per risk allele. Institutional ethics committees gave the appropriate approvals for the study. Genetic risk scores had a mean of 11.4 (SD 2.2) and were not related to prenatal famine. Adult BMI was 1.34 units higher among famine exposed (95% CI 0.15 to 2.06), MI: coefﬁcient = 1.22 (95% CI 0.87 to 2.76), Heckman: coefﬁcient = 0.70 (95% CI 0.38 to 1.78)).