

Appendix 5: Full adjusted association between interleukin-6 levels at baseline (1997–99) and over the 5-yr exposure period (5 yr before baseline and at baseline) and subsequent aging phenotype at 10-yr follow-up within a single analytic setting, with normal aging as the common reference point for all three outcomes ($n = 3044$)*

	Successful aging	Fatal or nonfatal CVD events	Non-CVD death
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Interleukin-6 levels at baseline			
Low ($n = 756$)	1.00 (ref)	1.00 (ref)	1.00 (ref)
Intermediate ($n = 1456$)	0.73 (0.59–0.89)	1.27 (0.90–1.80)	1.20 (0.70–2.05)
High ($n = 832$)	0.50 (0.38–0.65)	1.46 (1.00–2.14)	2.20 (1.27–3.80)
No. of times interleukin-6 was high over the 5-yr exposure period†			
0 ($n = 1867$)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1 ($n = 791$)	0.69 (0.55–0.86)	1.27 (0.96–1.68)	1.23 (0.82–1.86)
2 ($n = 386$)	0.60 (0.43–0.83)	1.48 (1.04–2.11)	2.13 (1.36–3.35)

Note: CI = confidence interval, CVD = cardiovascular, OR = odds ratio.

*To examine potential competing risk bias, the association between inflammation and aging phenotypes was assessed using multinomial regression. We estimated odds of successful aging and the two unhealthy aging outcomes (CVD events and non-CVD death) within a single analysis, with normal aging (the “non-case” category for each of the other categories) as the common reference point for all three outcomes, thus avoiding the substantial overlap in the different health components of aging. Models were adjusted for sex, age, socioeconomic status, smoking status, physical activity, acute inflammation and use of anti-inflammatory drugs.

†Interleukin-6 was measured twice (5 yr before baseline and at baseline); 0 = neither measurement was high, 1 = either measurement was high, 2 = both measurements were high.