Low socioeconomic status (SES) during childhood and adolescence has been found to predict greater susceptibility to common cold viruses in adults. Here, we test whether low childhood SES is associated with shorter leukocyte telomere length in adulthood, and whether telomere length mediates the association between childhood SES and susceptibility to acute upper respiratory disease in adulthood.

At baseline, 196 healthy volunteers reported whether they currently owned their home and, for each year of their childhood, whether their parents owned the family home. Volunteers also had blood drawn for assessment of specific antibody to the challenge virus, and for CD8+CD28− T-lymphocyte telomere length (in a subset, n = 135). They were subsequently quarantined in a hotel, exposed to a virus (rhinovirus [RV] 39) that causes a common cold and followed for infection and illness (clinical cold) over five post-exposure days.

Lower childhood SES as measured by fewer years of parental home ownership was associated with shorter adult CD8+CD28− telomere length and with an increased probability of developing infection and clinical illness when exposed to a common cold virus in adulthood. These associations were independent of adult SES, age, sex, race, body mass, neuroticism, and childhood family characteristics. Associations with infections and colds were also independent of pre-challenge viral-specific antibody and season. Further analyses do not support mediating roles for smoking, alcohol consumption or physical activity but suggest that CD8+CD28− cell telomere length may act as a partial mediator of the associations between childhood SES and infection and childhood SES and colds.

Lower SES is associated with lower IQ, which in turn may be caused by increased genetic load, so I’m wondering whether increased genetic load might also be responsible for shorter telomeres. If so, this could explain some of the association between IQ and life expectancy.