Results A one standard deviation (SD) increase in weight gain between 7 and 15 years was associated with 0.50 years (95% CI: 0.21, 0.80) higher AgeAccelGrim and, with 0.22 years (95% CI: -0.11, 0.55) higher AgeAccelLevine. This was replicated in NCDS. For linear growth, there was some evidence that more rapid growth between 2 and 4 years was associated with lower AgeAccelLevine (-0.39 years [95% CI: -0.74, -0.050]) and AgeAccelGrim (-0.24 years [95% CI: -0.54, 0.06]). There was no evidence that relative weight gain and linear growth during childhood was associated with any other AgeAccel biomarker. There was no relationship between pubertal timing in men and any of the AgeAccel biomarkers at 33 years. Women who reached menarche ≥12 years had 1.20 years (95% CI: 0.15, 2.24) higher AgeAccelGrim on average than women who reached menarche <12 years; however this was not replicated in NCDS.

Conclusion Our findings support the use of the second generation DNAAge biomarkers as markers of ageing and reinforces the idea that faster gains in weight during adolescence has lasting implications for healthy ageing.

Background There has been a long argument over whether or not schizophrenia is a neurodegenerative disorder associated with progressive brain changes and increasing cognitive impairment. Building on the fact that schizophrenia is highly heritable and overlaps genetically with general cognitive ability, we investigated whether common genetic variants associated with schizophrenia additively confer a stable deficit in cognitive ability, a greater risk of cognitive decline over time, or both, over the 8-year follow-up, independently from the effects of the apolipoprotein E gene (APOE-ε4), in phenotypically well-defined sample of healthy older adults.

Methods We used data from the English Longitudinal Study of Ageing study which encompassed 6817 population-representative English adults who were followed-up for 8 years. Cognitive function was measured with well-established tests of memory (tests of immediate and delayed word recall combined into a single measure of correctly recalled words) and executive function (a test of verbal fluency where participants name as many animals as possible in a minute). Polygenic score for schizophrenia (SZ-PGS) was calculated based on the results from Psychiatric Genomics Consortium. Linear mixed effect models with maximum likelihood estimation were used to estimate baseline status and rate of change in cognition associated with SZ-PGS.

Results The sample baseline mean age was 64.3 years old (standard deviation (SD)=9.3, range=50–101); 25.3% (N=1724) of participants were carriers of APOE-ε4 and 46.2% (N=3159) were men. The average baseline memory score was 10.4 (SD=3.4) and executive function score was 20.7 (SD=6.3). One standard deviation increase in SZ-PGS was associated with a lower baseline executive function score (-0.23, 95%CI-0.38 – -0.08) but not memory. SZ-PGS was not associated with rates of change in these cognitive domains during the 8-year follow-up period. However, APOE-ε4, tobacco smoking and lower wealth were associated with a decrease in the rate of memory and executive function during follow-up.

Discussion Common genetic variants associated with schizophrenia additively confer a stable deficit in cognitive ability but not cognitive ageing. The fact that we observed cognitive decline in our sample over the 8-year follow-up, associated with APOE-ε4, tobacco smoking and lower wealth demonstrates that our study had the capacity to show cognitive deficit but not further cognitive decline in healthy older adults.

Schizophrenia Polygenic Risk Predicts General Cognitive Deficit but Not Further Cognitive Decline in Healthy Older Adults

1.2.0 Ajnakina*,1A Kępiński,3J MacCabe,1D Cadar,1A Steptoe,4R Murray,1Department of Behavioural Science and Health, Institute of Epidemiology and Health Care, University College London, London, UK; 2Department of Biostatistics and Health Informatics, Institute of Psychiatry, Psychology and Neuroscience, King’s College London, University of London, London, UK; 3Department of Psychiatry Studies, Institute of Psychiatry, Psychology and Neuroscience, King’s College London, London, UK

10.1136/jech-2020-SSMabstracts.102

Schizophrenia Polygenic Risk Predicts General Cognitive Deficit but Not Further Cognitive Decline in Healthy Older Adults

1O Ajnakina*,1A Kępiński,3J MacCabe,1D Cadar,1A Steptoe,4R Murray,1Department of Behavioural Science and Health, Institute of Epidemiology and Health Care, University College London, London, UK; 2Department of Biostatistics and Health Informatics, Institute of Psychiatry, Psychology and Neuroscience, King’s College London, University of London, London, UK; 3Department of Psychiatry Studies, Institute of Psychiatry, Psychology and Neuroscience, King’s College London, London, UK

10.1136/jech-2020-SSMabstracts.102