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 53 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 DNAm age biomarkers as markers of ageing and reinforces the idea that faster gains in weight during adolescence has lasting implications for healthy ageing.

P05 PERFORMANCE ON DIFFERENT COGNITIVE TESTS PREDICT FUTURE DEMENTIA: FIFTEEN YEARS OF FOLLOW-UP IN A BRITISH COHORT STUDY

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Background Current policies do not support screening for future dementia among apparently healthy individuals. This is due to the lack of certainty of the clinical outcome and effective interventions. Numerous dementia risk models have been developed, currently, all within the research setting only. Studies have shown impairment in multiple cognitive domains several years before a clinical diagnosis of dementia.
Methods We examined the utility of an extensive battery assessing a range of specific cognitive abilities as well as a composite global score, to predict dementia. Dementia outcomes were ascertained using electronic health record linkage in 8581 individuals (aged 48–92 years) taking part in the EPIC-Norfolk study. Participants were followed for 15 years (2004–2019). Risk of dementia was estimated using Cox proportional hazard models adjusting for sociodemographic, lifestyle and health variables, evaluating discriminative accuracy of the models by analysing receiver operating characteristic (ROC) curves.
Results Poor cognition was predictive of incident dementia, even after adjustment for co-variates. Those with a poor performance score for global cognition (bottom 10%) were almost four times as likely to get a dementia diagnosis than those who performed well (HR=3.51 (95%CI 2.61, 4.71) P<0.001). Associations were observed for specific as well as global cognitive abilities. The test for episodic (verbal) memory outperformed other tests and was comparable to global cognition scores. Poor cognition in four or more tests was associated with 10-fold increased risk of developing dementia compared to those not performing poorly in any test (HR=10.82 (95% CI 6.85, 17.10) P<0.001)). Cognitive measures strengthen prediction models of dementia (AUC = 0.85 (95%CI 0.82, 0.87) P<0.001).
Discussion This study provides further insight on poor cognition predicting future dementia. This association was observed for global cognition and specific abilities, particularly for verbal episodic memory. Impairment occurs in multiple domains several years prior to a clinical diagnosis, and the more pervasive and greater the variability, the higher the risk of dementia. Deficits across multiple domains predict over and above individual test scores.

P06 SCHIZOPHRENIA POLYGENIC RISK PREDICTS GENERAL COGNITIVE DEFICIT BUT NOT FURTHER COGNITIVE DECLINE IN HEALTHY OLDER ADULTS

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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 of APOE-ϵ4, tobacco smoking and lower wealth demonstrates that our study had the capacity to show cognitive

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