**Appendix 2.** Calculation of polygenic risk score for mental disorders, mental health, and cognitive functioning

Raw genotype and phenotype data were processed through the same quality control and analysis pipeline.

**Quality control**: Respondents with low genotyping rate (<98%) and SNPs showing significant deviation from Hardy-Weinberg equilibrium (HWE, P-value < 1×10-5), a low minor allele frequency (MAF <10%) or high rates of missing data (>5%) were excluded. Mismatch between recorded and genotypic sex were also excluded.

**Imputation**: We used IMPUTE v2 to account for variation in SNP coverage across genotyping platforms, generating data on ~8 million SNPs on the basis of the UK10K data release (Wain et al., 2015) and the 1,000 Genomes Project version June 2010. Imputed SNPs with posterior probability averages < 90% for the most likely genotype were excluded. Principal component analysis (PCA) (Price et al., 2006) was used to identify, and control for, population stratification in our models.

**Statistical analysis**: After linkage disequilibrium pruning and frequency filtering of the genotyped SNPs, we exploited publically available data from the Cross-Disorder working group of the psychiatric genomic consortium (PGC) to account for polygenic effects on risk of common mental disorders within the offspring cohort. We generated polygenic risk scores (PRS) derived from analyses of selected mental disorders (PRScross) (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013). Assoicatioon of PRSs with phenotype status were performed with logistic regression. Nagelkerke’s pseudo-R2 was calculated to measure the proportion of variance explained. To estimate heritability (i.e., variance explained at the liability scale) assuming a liability-threshold model, a lifetime risk of 25% of poor mental health, 20% of mental disorders, and 10% cognitive dysfunction, the Genetic Anaysis Repository (<http://sourceforge.net/p/gbchen/wiki/GEAR/>) was used. For each analysis, ten different polygenic risk scores with ten different level of significance in logistic regression were fitted. The equivalent number of effective tests using the correlation matrix (<http://gump.qimr.edu.au/general/daleN/matSpD/>) and Bonferroni correction were also used on the sum of effective tests across parents and offspring cohort.

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