**Appendix e-1: additional methods information**

**PEG study**

If a PD diagnosis was deemed only ‘possible’ at baseline, a diagnostic re-assessment was scheduled. In addition, if necessary, disease classifications were updated after further follow-up visits with the neurologists. Recruitment occurred in two phases; during 2001-2007 (PEG1) and 2010-2017 (PEG2). At PEG1 baseline, 359 incident PD patients were seen within three years of their diagnosis, but the presence of hallucinations was first assessed during follow-up appointments (N=251), after on average 6 years of disease duration. The second recruitment (PEG2) enrolled patients diagnosed with PD any time after 2001 from the same communities. They were first seen by UCLA movement disorder specialists after 2010. PEG2 enrolled 388 idiopathic PD patients, and at the end of 2019, 183 had completed a follow-up visit.

**Genetic data**

Pre-imputation quality control consisted of removal of individuals who were excluded due to sex discrepancy, excessive heterozygosity (F>± 0.3), or had genotyped SNPs less than 98% (N=63 PEG patients, no PW patients). We removed SNPs that were not in Hardy Weinberg Equilibrium (HWE) <1x10-7 (PEG: N=2,968 SNPs; PW: N=364 SNPs) and SNPs from the sex chromosomes (PW: N=16,723 SNPs), leaving 516,448 and 667,866 SNPs for imputation in PEG and PW, respectively.

After imputation, SNPs that were not bi-allelic (N=3,760 PEG; N=7,165 PW), with HWE less than 1x10-7 (among those from European ancestry) or with a Minor Allele Frequency (MAF) of less than 0.02 were excluded, leaving 6,767,726 SNPs for PEG and 6,778,623 SNPs for PW. We further eliminated SNPs that had a call-rate of less than 90% (N=25,020 PEG; N=17,282 PW), or any SNPs that were of poor quality, based on a low R-square (less than 0.90) with the imputed variant (N=831,664 PEG; N=621,580 PW) leaving a total of 5,911,042 SNPs in PEG and 6,139,755 SNPs in PW. The hardcoded genotypes from the imputation were used.

**Creation of polygenic risk scores**

The AD-PRS: used the results of the previously performed IGAP GWAS and *APOE* status. *APOE* allele status is a well-known genetic risk factor for AD, and allele status is best estimated using specific combinations of two SNPs (rs429358 and rs7412). Although GWAS results have been known to be highly associated with *APOE* allele status, some measurement bias could still occur when using GWAS data instead of estimating the *APOE* haplotype status based on these two SNPs. Thus, we chose to use the *APOE* haplotype status instead of using the GWAS results for this specific gene region. We identified *APOE* haplotypes, and among the European ancestry population, 49 individuals had at least one *APOE*-*e2* allele (based on the minor allele of rs7412), and 98 individuals had at least one *APOE*-*e4* allele (based on the minor allele of rs429358). The *APOE-*PRS was then calculated using the betas of the odds ratio for the *APOE-e2* and *APOE-e4* alleles and AD, estimated from the meta-analysis of 28 AD case-control studies.1 For European ancestry, this meta-analysis estimated the OR to be 0.55 (β: -0.598) for each *APOE*-*e2* allele; and 3.77 (β: 1.327) for each *APOE*-*e4* allele compared with the *APOE*-*e3* allele.2

The IGAP GWAS analysis was performed on 54,142 individuals of European descent (17,008 AD cases; 37,134 controls) and consisted of over 7 million SNPs.3,4 We first removed the SNPs that were located in the *APOE* gene or were in linkage disequilibrium with the two *APOE*-related SNPs (rs429358 and rs7412) previously mentioned. We then restricted to those SNPs that were available in the PEG and PW study and more than 5.17 million variants remained. After ‘clumping’ 383,050 SNPs remained for the AD-PRS. We then restricted the PRS to variants that had a GWAS P-value of less than 5x10-8 in the IGAP GWAS, retaining 92 SNPs (including the 2 *APOE* SNPs).

The schizophrenia (SZ)-PRS: used the results of the SZ working group of the Psychiatric Genomics consortium.26 This group performed a GWAS analysis on 77,096 individuals of mixed ethnicity, although the majority of the individuals were of European descent (24 out of 28 studies) and the remaining individuals were from East-Asian populations.5 After restricting to those that were available for the PEG and PW populations, there were almost 5.4 million variants. Subsequent clumping of the data, using a R-square threshold of 0.5, resulted in a total of 394,125 SNPs for the SZ-PRS. Restricting to a P-value of less than 5x10-8 in the SZ-GWAS resulted in a SZ-PRS based on 328 SNPs.

The Parkinson’s disease (PD)-PRS used a previous meta-analysis GWAS of 11.9 million SNPs on 416,000 individuals in European ancestry populations.6,7 Of the top 9,830 variants of this GWAS, for which summary statistics are available for downloading, 8,123 variants overlapped with our genetic data. After clumping, 688 variants remained. 181 variants had a P-value of less than 5x10-8 in the original GWAS analysis and were used to calculate the PRS.

The height PRS: was created using a GWAS meta-analysis using data from the GIANT consortium and the UK Biobank (2.3 million).8 This GWAS consisted of ~700,000 individuals of European descent.21 When comparing with our dataset, 2.0 million SNPs remained. Subsequent clumping of the data restricted the PRS to 267,658 SNPs, with 12,688 SNPs below the P-value threshold of 5x10-8. These 12,688 SNPs were used to construct the PRS.

**References appendix e-1**

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