Supplementary Materials

Le Guen et al. Protective association of *HLA-DRB1**04 subtypes in neurodegenerative diseases implicates acetylated tau PHF6 sequences

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Supplementary Methods

QUALITY CONTROL AND ANALYSIS PER DATASET

Alzheimer's Disease – ADSP & ADGC datasets

1) Participants and sources of data

Phenotypic information and genotypes were obtained from publicly released genome-wide association study datasets assembled by the Alzheimer's Disease Genetics Consortium (ADGC) and derived from whole-genome sequencing (WGS) data generated by the Alzheimer Disease Sequencing Project (ADSP), with phenotype and genotype ascertainment described elsewhere. The cohorts' queried accession numbers, as well as the sequencing technology or single nucleotide polymorphism (SNP) genotyping platforms are described in **Supplementary Tables 8 and 9**. The microarray datasets are largely part of the ADGC and as such they will be referred thereafter as the ADGC.

2) Quality control procedures

Prior to HLA imputation, ancestry, principal components and relatedness determinations, variants were excluded in each cohort-platform based on genotyping rate (< 95%), MAF < 1%, and Hardy-Weinberg equilibrium in controls ($p < 10^{-6}$) using PLINK v1.9[1]. GnomAD[2] database-derived information was used to filter out SNPs that met one of the following exclusion criteria[3,4]: (i) located in a low complexity region, (ii) located within common structural variants (MAF > 1%), (iii) multiallelic SNPs with MAF > 1% for at least two alternate alleles, (iv) located within a common insertion/deletion, (v) having any flag different than PASS in gnomADv.3, (vi) having potential probe polymorphisms. The latter are defined as SNPs for which the probe may have variable affinity due to the presence of other SNP(s) within 20 bp and with MAF > 1%. Individuals with more than 5% genotype missingness were excluded. Duplicate individuals were identified with KING[5] and their clinical, diagnostic and pathological data (including age-at-onset of cognitive symptoms, age-at-examination for clinical diagnosis, age-at-last exam, age-at-death), as well as sex, race, and *APOE* genotype were cross-referenced across cohorts. Duplicate entries with irreconcilable phenotype or discordant sex were flagged for exclusion.

3) Ancestry determination

For each cohort, we first determined the ancestry of each individual with SNPWeights v2[6] using reference populations from the 1000 Genomes Consortium[7]. By applying an ancestry percentage cut-off > 75%, the samples were stratified into five super populations: South-Asians, East-Asians, Amerindians, Africans, and Europeans, and an Admixed group composed of individuals not passing the 75% cut-off in any single ancestry (**Supplementary Table 9**)[3]. The analyses were split into three ancestry groups: Europeans, Africans, and Amerindians-Latinos. The first two groups are composed of individuals passing the 75% threshold in their respective ancestry. The Amerindian-Latinos includes individuals in the Amerindians ancestry group (75% cut-off), and individuals in the Admixed group with at least 15% Amerindians and who identified as Hispanic/Latinos ethnicity. The rationale to include these additional individuals is to compensate the paucity of the Amerindians only group and to have a similar ancestry composition as in the Latin American Research Consortium on the Genetics of Parkinson's Disease (LARGE-PD, see below). Last, enriching for Amerindians ancestry enables us to assess the effect of *HLA-DRB1**04:07 since the HLA haplotype DRB1*04:07~DQA1*03:01~DQB1*03:02 is a common haplotype in this ancestry group.

4) Imputation

Each cohort-genotyping platform was imputed on the TOPMed imputation server per ancestry group to obtain an imputation quality (R^2) per ancestry group. For the local-GWAS at the HLA locus we retained variants with $R^2 > 0.30$, MAF > 1%, and present in 50% of the imputed cohorts.

HLA -alleles and -amino-acids were imputed on platform and ancestry specific reference panels available through HIBAG[8] or trained in-house as previously described[9]. In all allele-level analyses, alleles with an imputation posterior probability lower than 0.5 were considered as undetermined as recommended by HIBAG developers, and only allele with carrier frequency above 1% were retained for analysis. For haplotype-level analyses, only individuals with non-missing allele genotypes were included. Three-locus HLA class I or class II haplotypes were determined using the haplo.em function from the R haplo.stats package. Only haplotypes with posterior probability >0.5 and a carrier frequency of >1% were included in the analysis. In the amino-acid-level analyses, HIBAG[8] was used to convert P-coded alleles to amino acid sequences for exons 1-3 of class II genes.

5) Samples retained for analysis

Supplementary Table 10 describes the demographics of individuals retained for analysis. Analyses were implemented into 6 different groups separating WGS data and

TOPMed imputed and by ancestry group: ADSP-European, ADSP-African, ADSP-Amerindian-Latino, ADGC-European, ADGC-African, ADGC-Amerindian-Latino.

6) Statistical analyses

In the following paragraph a variable refers indifferently to a variant in the local-GWAS at HLA locus, an HLA-allele, an HLA-haplotype, or any HLA-amino-acids. The AD risk associated with each variable was estimated using a linear mixed model regression on case-control diagnosis. The HLA -allele, -haplotype, and -amino-acids level analyses were run as dominant model (phenotype frequency, collapsing homozygotes for the minor frequency variable with heterozygotes). All statistical analyses were performed in R (v4.0.2) and adjusted for sex, six genetic principal components estimated with the *PC-Air* method[10] implemented in *GENESIS*[11], and covaried by a sparse genetic relationship matrix estimated with the *PC-Relate* method[12] implemented in *GENESIS*. Case-control analyses were not adjusted for age given that controls were older than cases in some subgroups. Correcting for age when cases are younger than controls leads to the model incorrectly inferring the age effect on AD risk, resulting in statistical power loss[3].

Alzheimer's Disease – UK Biobank dataset

1) Participants, quality control and variant imputation

The UK Biobank data includes 488,377 participants which were genotyped on SNP microarrays and imputed at high resolution using two reference panels: (i) the Haplotype Reference Consortium (HRC) for most variants with minor allele frequency > 0.001 and (ii) the UK10K+1000Genomes for variants not in the HRC panel[13]. The quality control prior to imputation has been extensively described in Bycroft et al.[13]. The proxy-AD phenotype defined in Bellenguez et al.[14] (i.e., cases are individuals who have an ICD10 code linked to AD in their medical record[15] or reported a first degree with Alzheimer's disease, March, 2021 release). We restricted our analysis to 388,051 unrelated individuals after pruning for 3rd degree relatedness using the following criteria to rank order individuals for removal: (i) highest number of relatives, (ii) not a proxy-AD case (iii) and youngest individual.

2) Ancestry determination

Unrelated individuals of the UK Biobank were split into two groups: British and non-British/other ancestries. The British ancestry group corresponds to individuals who self-identified as white British and who clustered on together in the principal ancestry

component analysis performed in Bycroft et al. (field ID: 22006). The British ancestry group was composed of 52,426 proxy-AD cases, and 272,624 controls. The non-British/other ancestries group was composed of 7,840 proxy-AD cases and 55,161 controls. This last group was heterogeneous in term of ancestral origin, but most individuals identified as non-British European.

3) HLA Imputation

HLA -alleles and -amino-acids were imputed on platform and ancestry specific reference panels available through HIBAG[8] or trained in-house as previously described[9]. In allele-level analyses, alleles with an imputation posterior probability lower than 0.5 were considered as undetermined as recommended by HIBAG developers, and only allele with carrier frequency above 1% were retained for analysis. In the haplotype-level analyses, only individuals with non-missing allele genotypes were included in the haplotype level analysis. Three-locus HLA class I or class II haplotypes were determined using the haplo.em function from the R haplo.stats package. Only haplotypes with posterior probability >0.5 and a carrier frequency of >1% were included in the analysis. In the amino-acid-level analyses, HIBAG[8] was used to convert P-coded alleles to amino acid sequences for exons 1 -3 of class II genes.

4) Statistical analyses

In the following paragraph, a variable refers indifferently to a variant in the local-GWAS at HLA locus, an HLA-allele, an HLA-haplotype, or a specific HLA-amino-acid. HLA -allele, -haplotype, and -amino-acids level analyses were run as dominant model (phenotype frequency, collapsing homozygotes for the minor frequency variable with heterozygotes). Proxy-AD association were tested with plink2 (v2.00a2LM) using the –glm flag covarying for age at last visit, sex, genotyping array, assessment center and the first 20 PCs provided by the UK Biobank.

Alzheimer's Disease – EADB, GR@ACE, GERAD, EADI, DemGene, Bonn, CCHS datasets

Demographics, quality control and GWAS analysis are fully described in Bellengez et al. [14] and demographics are also shown in **Supplementary Table 11**. The HLA analyses were conducted plink2 (v2.00a2LM) using the –glm flag covariates per cohort were described in Bellengez et al. [14].

Alzheimer's Disease – NCGG dataset

The National Center for Geriatrics Gerontology (NCGG) Biobank was established as a geriatric hospital-based Biobank in 2012. The NCGG Biobank is one of the facilities belonging to the National Center Biobank Network. The NCGG Biobank cohort of the study consisted of 2974 patients (female, 64%; mean age, 78.0) with LOAD and 3096 controls (female, 53%; mean age, 71.1) who were recruited from the NCGG Biobank. All subjects were of Japanese origin. Genotyping data were downloaded from the NCGG Biobank database. All subjects were genotyped by using the Affymetrix Japonica Array. Demographics, quality control and GWAS analysis are fully described in Shigemizu et al.[16]. The HLA analyses were conducted plink2 (v2.00a2LM) using the –glm flag covariates per cohort were described in Shigemizu et al.[16].

Alzheimer's Disease – GARD dataset

Phenotypic information and genotypes were obtained from the Gwangju Alzheimer's & Related Dementias (GARD) cohort database portal (http://gard.nrcd.re.kr:8080/), with phenotype and genotype ascertainment, as well as ethnical review described elsewhere. Briefly, all cases were LOAD and fulfilled the NINCDS-ADRDA criteria and met the pathological criteria (scanned amyloid beta PET). Genotyping was conducted with the blood species using the Korea Biobank Array, a microarray platform customized for Koreans. Demographics, quality control and GWAS analysis are fully described in Kang et al.[17] and summarized in **Supplementary Table 12**. The HLA analyses were conducted plink2 (v2.00a2LM) using the –glm flag covariates per cohort were described in Kang et al.[17].

Alzheimer's Disease –JGSCAD dataset

Demographics, quality control and GWAS analysis are fully described in Miyashita et al.[18] and summarized in **Supplementary Table 12**. The HLA analyses were conducted plink2

(v2.00a2LM) using the –glm flag covariates per cohort were described in Miyashita et al.[18].

Alzheimer's Disease Neuropathology – NACC and RUSH datasets

1) Participants and sources of data

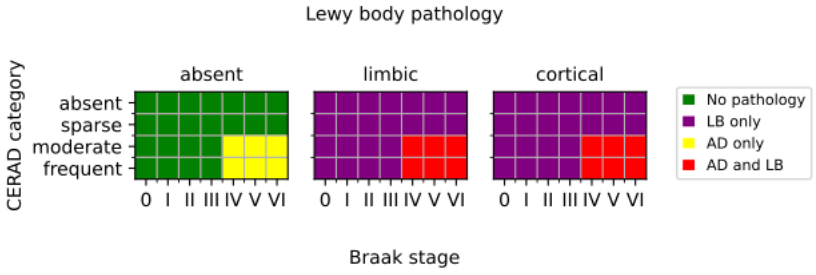
Participants were enrolled and followed up at one of Alzheimer's Disease Center (ADC) across the US. Genetic data were obtained from the Rush Religious Orders Study and Memory and Aging Project (ROSMAP)[19] and from the Alzheimer's Disease Center (ADC) cohorts 1 to 7 parts of the ADGC[20] (see **Supplementary Table 3** for data accession number). ROSMAP samples were assessed by the Rush ADC and their neuropathological assessment followed procedures described respectively in Schneider et al.[21]. Neuropathological assessment for samples with genotyping from ADGC was obtained from National Alzheimer's Coordinating Center (NACC) and followed postmortem evaluation protocol[22].

2) Quality control procedures, ancestry determination, and imputation

The content of this section is identical to the corresponding sections in "Alzheimer's Disease – ADSP & ADGC datasets" given that these samples were included in the association with AD status.

3) Samples retained for analysis

Supplementary Table 13 describes the demographics of individuals retained for neuropathology analyses: Tau Braak staging, neuritic plaques density. We also defined three categories: AD pathology only, Lewy body (LB) pathology only, and dual pathology (AD and LB) and compared these against controls without AD and LB pathologies. The schematic below describes these categories and follows the classification defined in Tsuang et al.[23].



Supplementary Table 14 provides the demographics and number of individuals per category.

4) Statistical analyses

The statistical analyses follow the method described in the "Alzheimer's Disease – ADSP & ADGC datasets" corresponding section.

Alzheimer's Disease Cerebrospinal Fluid – EADB and Swedish datasets

1) Participants and sources of data.

EADB participants (as described above) for which cerebrospinal fluid (CSF) amyloid beta and/or (phosphorylated) tau measurements were available were included. The Swedish cohorts originate from Gothenburg H70 Birth cohort studies and are clinical AD samples from Sweden all gathered and analyzed in Gothenburg. Genetic data for EADB cohorts has been processed using a consistent approach[14], in which the Illumina Infinium Global Screening Array (GSA, GSAsharedCUSTOM_24+v1.0) was predominantly used in addition to the Axiom 815K Spanish biobank array (Thermo Fisher). The genetic data for the Swedish cohorts were generated with the Illumina Neurochip array.

2) Quality control procedures and imputation

Quality control procedures of the EADB datasets are described here in Bellenguez et al.[14]. For the Swedish datasets, QC and imputation procedures are described elsewhere[24]. In short, low-quality variants were excluded based on call rate, minor alle frequency (MAF< 0.01) and Hardy-Weinberg disequilibrium ($P < 1 \times 10^{-6}$). Individuals were removed based on per-sample call rate, sex mismatch, excessive heterozygosity or non-European ancestry. The Sanger imputation service was used to impute post-QC, using the reference panel of Haplotype Reference Consortium data (HRC1.1). The UCSC LiftOver program (https://genome-store.ucsc.edu/) and Plink v2.0 (www.cog-genomics.org/plink/2.0/) were used to lift the GRCh37 genomic positions to GRCh38, the genomic build for all other datasets.

3) Samples retained for analysis

Supplementary Table 15 describes the demographics of individuals retained for analysis. The association analyses with HLA haplotypes, alleles and amino acids were only performed for those individuals for which genotype-level data was available (rather

than GWAS summary statistics). For rs601945 association analyses, all cohorts were included.

4) Statistical analyses

For HLA-locus, -allele, haplotype, and amino acid association analyses, similar association analysis procedures were performed. For continuous phenotypes Aβ42, tau and pTau, linear regression was performed within each cohort using PLINK v2.0. Association tests were adjusted for gender, age, assay type (if applicable), and ten ancestry principal components. METAL was used for meta-analysis of the per cohort association results, applying the default approach that utilizes p-value and direction of effect, weighted according to sample size.

Association analyses were repeated for subgroups, stratified according to diagnosis status, resulting in a group including only AD subjects, and one including individuals with no or mild cognitive impairment. Covariates were those described for the main analyses above.

Parkinson's Disease – IPDGC, McGill, NINDS, NGRC, Oslo, PPMI, APDGC, UK Biobank datasets

Demographics, phenotyping, quality control, imputation and analysis of the European ancestry cohorts part of local-GWAS at HLA summary statistics have been extensively described in Nalls et al[25]. Similarly, the phenotyping, quality control and HLA imputation PD cohorts used in the HLA -alleles, -haplotypes, and -amino-acids level analysis were previously described in Yu et al.[9]. Demographics are presented in **Supplementary Table 16**.

Parkinson's Disease – EastAsians-PD and 23andMe datasets

For the EastAsians-PD and 23andMe cohorts, HLA alleles, haplotypes, amino acids statistics were derived from GWAS summary statistics data using the DISH software[26] as described in Naito et al.[27]. Demographics, quality control and GWAS analysis were previously described[27–29] and available demographics are reported in **Supplementary Table 17**.

Parkinson's Disease – LARGE-PD dataset

Demographics, quality control and GWAS analysis are fully described in Loesch et al.[30] and demographics are also shown in **Supplementary Table 17**.

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Supplementary Table 1. Number of individuals per disease and ancestry group included in the meta-analyses.

	Ancestry	N cases/ proxy-cases	N controls	Reference doi
Parkinson's disease				
European datasets in Yu et al. 2021	European	33984	490861	doi:10.1038/s41531-021-00231-5
23andMe individuals (i) in Nalls et al. 2014	European	3261	29499	doi:10.1038/ng.3043
23andMe individuals (ii) in Nalls et al. 2014	European	866	32538	doi:10.1038/ng.3043
23andMe individuals in Chang et al. 2017	European	6476	302042	doi:10.1038/ng.3955
23andMe individuals in Nalls et al. 2019	European	2448	571441	doi:10.1016/s1474-4422(19)30320-5
Japaneses in Naito et al. 2021	East Asian	988	2521	doi:10.1002/mds.28583
Taiwaneses in Foo et al. 2020	East Asian	216	225	doi:10.1001/jamaneurol.2020.0428
Singaporeans/Malays in Foo et al. 2020	East Asian	2536	21840	doi:10.1001/jamaneurol.2020.0428
South Koreans in Foo et al. 2020	East Asian	1494	599	doi:10.1001/jamaneurol.2020.0428
Hong-Kongers in Foo et al. 2020	East Asian	199	166	doi:10.1001/jamaneurol.2020.0428
Chineses in Foo et al. 2020	East Asian	2279	2021	doi:10.1001/jamaneurol.2020.0428
Amerindian/European-Latinos in Loesch et al. 2021	Amerindian-European	807	690	doi:10.1002/ana.26153
	Total	**55554**	**1454443**	
Alzheimer's disease				
ADGC-European TOPMed imputed	European	13027	12748	doi:10.1038/s41588-019-0358-2
ADSP-European WGS	European	4127	3020	doi:10.1038/s41588-019-0358-2
UK Biobank – British ancestry	European	52426	272624	doi:10.1038/s41586-018-0579-z
UK Biobank – non-British/Other ancestries	European	7840	55161	doi:10.1038/s41586-018-0579-z
EADB, GR@ACE, GERAD, EADI, DemGene, Bonn, CCHS	European	35084	55762	doi:10.1101/2020.10.01.20200659
ADGC-African TOPMed imputed	African	253	1837	doi:10.1001/jamaneurol.2020.3536
ADSP-African WGS	African	849	1240	doi:10.1001/jamaneurol.2020.3536
South Koreans – GARD	East Asian	872	895	doi:10.1101/2020.07.02.20145557
Japanese – JGSCAD	East Asian	1008	376	doi:10.1371/journal.pone.0058618
Japanese – NCGG	East Asian	2974	1016	doi:10.1038/s41398-021-01272-3
ADGC-Amerindian-Latino TOPMed imputed	Amerindian-European	1542	1906	unpublished
ADSP-Amerindian-Latino WGS	Amerindian-European	1233	2327	unpublished
	Total	**121235**	**408912**	

Supplementary Table 2. HLA -alleles, -haplotypes, -amino-acids levels association across tested variables in Alzheimer's Disease.

External spreadsheet.

Supplementary Table 3. HLA -alleles, -haplotypes, -amino-acids levels association across tested variables in Parkinson's Disease. Frequencies are missing for 23andMe samples as DISH analysis on GWAS summary statistics did not require these and these were not provided.

External spreadsheet.

Supplementary Table 4. (on the next page)

Supplementary Table 5. List of tau peptides that were tested for binding with *HLA-DRB104:01, *HLA-DRB1**04:04, and *HLA-DRB1**04:05.**

External spreadsheet.

Supplementary Table 6. List of α-synuclein peptides that were tested for binding with *HLA-DRB104:01, *HLA-DRB1**04:04, and *HLA-DRB1**04:05.**

External spreadsheet.

Supplementary Table 4. *HLA-DRB104 alleles are associated with reduced tau and neurofibrillary tangles but not with Amyloid-β or neuritic plaques, when testing their association with Alzheimer's disease neuropathology and cerebrospinal fluid biomarkers.** p-tau: phosphorylated tau, t-tau: total tau, N: number of individuals, MAF: minor allele frequency, OR: odds ratio, β: parameter estimate, CI: confidence interval. Braak: Tau Braak staging, Neur: Neuritic plaques density.

		DRB1*04:01				DRB1*04:04				DRB1 H13			
	Phenotype	**N**	**Freq**	**β [95% CI]**	**pval**	**N**	**Freq**	**β [95% CI]**	**pval**	**N**	**Freq**	**β [95% CI]**	**pval**
All individuals	**Tau Braak staging**	6804	0.164	-0.17[-0.28; -0.07]	1.1E-03	6804	0.058	0.06[-0.11; 0.22]	0.5	7456	0.293	-0.13[-0.21; -0.05]	1.4E-03
	Neuritic plaques density	5385	0.165	-0.06[-0.14; 0.01]	0.11	5385	0.057	-0.02[-0.14; 0.1]	0.72	5876	0.292	-0.04[-0.1; 0.02]	0.19
	total-tau in CSF	5392	0.16	-0.05[-0.12; 0.02]	0.17	5392	0.052	-0.24[-0.36; -0.12]	1.1E-04	5289	0.232	-0.11[-0.17; -0.05]	5.5E-04
	p-tau in CSF	5371	0.16	-0.02[-0.09; 0.06]	0.66	5371	0.052	-0.29[-0.41; -0.17]	2.1E-06	5269	0.234	-0.08[-0.14; -0.02]	1.0E-02
	Aβ42 in CSF	5471	0.16	0.07[-0.01; 0.14]	0.07	5471	0.051	0.09[-0.03; 0.21]	0.16	5368	0.232	0.08[0.01; 0.14]	0.02
Dx adjusted	**Tau Braak staging**	5826	0.16	-0.08[-0.16; -0.01]	0.02	5826	0.057	0.05[-0.06; 0.16]	0.38	6388	0.287	-0.05[-0.1; 0.01]	0.08
	Neuritic plaques density	3796	0.163	-0.02[-0.07; 0.03]	0.42	4602	0.057	-0.03[-0.1; 0.04]	0.43	5020	0.289	-0.01[-0.06; 0.03]	0.63
	total-tau in CSF	5364	0.16	-0.04[-0.11; 0.02]	0.2	5264	0.052	-0.22[-0.32; -0.11]	5.0E-05	5263	0.233	-0.09[-0.14; -0.03]	1.6E-03
	p-tau in CSF	5343	0.161	-0.02[-0.08; 0.05]	0.61	5242	0.052	-0.27[-0.37; -0.16]	1.7E-06	5243	0.234	-0.06[-0.12; -0.01]	0.02
	Aβ42 in CSF	5443	0.16	0.07[0.01; 0.13]	0.03	5342	0.051	0.08[-0.03; 0.18]	0.15	5342	0.232	0.07[0.01; 0.12]	1.0E-02
Cases	**Tau Braak staging**	4689	0.157	-0.1[-0.17; -0.03]	5.7E-03	4689	0.057	0.07[-0.04; 0.18]	0.21	5126	0.283	-0.07[-0.12; -0.01]	0.02
	Neuritic plaques density	3796	0.162	-0.02[-0.05; 0.02]	0.38	3796	0.057	-0.01[-0.06; 0.05]	0.85	4124	0.289	-0.01[-0.04; 0.02]	0.39
	total-tau in CSF	-	0.157	-0.06[-0.15; 0.04]	0.25	-	0.055	-0.32[-0.48; -0.16]	9.6E-05	-	0.228	-0.14[-0.23; -0.06]	8.2E-04
	p-tau in CSF	-	0.159	-0.02[-0.12; 0.08]	0.67	-	0.055	-0.33[-0.49; -0.17]	8.2E-05	-	0.228	-0.1[-0.19; -0.01]	0.02
	Aβ42 in CSF	-	0.159	0.10[0.02; 0.18]	0.02	-	0.055	-0.01[-0.14; 0.12]	0.85	-	0.227	0.09[0.02; 0.16]	1.0E-02
	Age-at-AD-onset	11315	0.152	-0.07[-0.54; 0.41]	0.78	11315	0.054	0.89[0.15; 1.63]	0.02	11900	0.278	0.39[0.03; 0.76]	0.03
Controls	**Tau Braak staging**	1137	0.173	0.08[-0.12; 0.28]	0.46	1137	0.058	-0.14[-0.46; 0.18]	0.39	1262	0.303	0.06[-0.09; 0.2]	0.45
	total-tau in CSF	-	0.171	0.05[-0.05; 0.15]	0.31	-	0.047	-0.24[-0.42; -0.07]	6.6E-03	-	0.244	0.01[-0.08; 0.09]	0.85
	p-tau in CSF	-	0.171	0.06[-0.04; 0.17]	0.25	-	0.047	-0.26[-0.44; -0.08]	5.5E-03	-	0.246	0.03[-0.06; 0.12]	0.56
	Aβ42 in CSF	-	0.17	0.05[-0.07; 0.16]	0.42	-	0.046	0.16[-0.04; 0.35]	0.11	-	0.243	0.06[-0.04; 0.15]	0.25

Supplementary Table 7. Association of HLA haplotypes in linkage with *HLA-DRB401:03.** Lack of association of DRB1*07:01~DQA1*02:01~DQB1*02:02 and DRB1*09:01~DQA1*03:02~DQB1*03:03 advocates against HLA-DRB4*01:03 involvement in the protective effect observed in AD and PD. Effect sizes are reported as odds ratio (OR), with 95% confidence interval [CI], and significance (p-value). FreqC: frequency of carriers, N: number of individuals

		Parkinson's Disease				Alzheimer's Disease				AD + PD		
HLA-DRB3/4/5	HLA haplotype	FreqC	N	OR	pval	FreqC	N	OR	pval	OR	pval	p_het
DRB4*01:03	DRB1*04:01~DQA1*03:01~DQB1*03:02	0.088	1473386	0.95[0.92; 0.98]	3.6E-03	0.097	521560	0.91[0.88; 0.94]	1.1E-06	0.93[0.91; 0.96]	5.2E-08	0.11
DRB4*01:03	DRB1*04:01~DQA1*03:03~DQB1*03:01	0.12	1481518	0.98[0.97; 0.99]	5.2E-04	0.146	532448	0.97[0.94; 1.0]	0.08	0.98[0.97; 0.99]	1.1E-04	0.66
DRB4*01:03	DRB1*04:02~DQA1*03:01~DQB1*03:02	0.019	1478393	0.92[0.85; 0.99]	0.04	0.019	85940	0.96[0.86; 1.08]	0.53	0.93[0.88; 1.0]	0.04	0.50
DRB4*01:03	DRB1*04:03~DQA1*03:01~DQB1*03:02	0.077	35369	0.82[0.73; 0.92]	9.6E-04	0.077	10995	1.06[0.92; 1.22]	0.42	0.91[0.83; 1.0]	0.04	0.01
DRB4*01:03	DRB1*04:04~DQA1*03:01~DQB1*03:02	0.077	1475734	0.84[0.8; 0.89]	1.7E-10	0.092	524205	0.85[0.82; 0.89]	3.1E-14	0.85[0.82; 0.88]	3.5E-23	0.80
DRB4*01:03	DRB1*04:05~DQA1*03:03~DQB1*04:01	0.11	35369	0.99[0.92; 1.06]	0.76	0.227	10995	1.08[0.98; 1.18]	0.11	1.02[0.97; 1.08]	0.46	0.15
DRB4*01:03	DRB1*04:06~DQA1*03:01~DQB1*03:02	0.047	35369	0.95[0.83; 1.09]	0.45	0.058	10995	0.9[0.77; 1.06]	0.21	0.93[0.84; 1.03]	0.16	0.63
DRB4*01:03	DRB1*04:07~DQA1*03:01~DQB1*03:02	0.198	1498	0.58[0.44; 0.76]	6.1E-05	0.063	1865	0.75[0.47; 1.21]	0.23	0.62[0.49; 0.78]	4.6E-05	0.36
DRB4*01:03	DRB1*04:07~DQA1*03:03~DQB1*03:01	0.021	524845	0.87[0.73; 1.04]	0.12	0.019	519510	0.88[0.81; 0.96]	4.0E-03	0.88[0.81; 0.95]	1.1E-03	0.93
DRB4*01:03	DRB1*04:10~DQA1*03:03~DQB1*04:02	0.039	5007	0.91[0.67; 1.24]	0.55	0.037	6846	1.24[0.96; 1.6]	0.1	1.09[0.9; 1.33]	0.37	0.13
DRB4*01:03 (2/3) /DRB4*01:01 (1/3)	DRB1*07:01~DQA1*02:01~DQB1*02:02	0.201	1506744	1.01[0.97; 1.05]	0.65	0.186	92453	1.01[0.97; 1.04]	0.75	1.01[0.98; 1.03]	0.58	0.92
DRB4*01:03N	DRB1*07:01~DQA1*02:01~DQB1*03:03	0.08	1485027	1.2[1.12; 1.28]	2.1E-07	0.065	95130	1.08[1.02; 1.14]	8.1E-03	1.12[1.08; 1.17]	9.4E-08	0.02
DRB4*01:03	DRB1*09:01~DQA1*03:02~DQB1*03:03	0.03	1510253	1.02[0.97; 1.07]	0.43	0.048	96309	1.04[0.97; 1.11]	0.29	1.03[0.99; 1.07]	0.2	0.70

Supplementary Table 8. Queried US based cohorts' part of the Alzheimer's disease ADSP and ADGC analyses.

Cohort/Project	Genotyping Platform	Cohort-Platform ID	Sample (N)	Data Repository and Access ID
ADSP WGS	Whole Genome Sequencing	ADSP_WGS	16906	NIAGADS DSS (NG00067.v5) / NACC
ACT	Illumina Human 660W-Quad	ACT	2790	NIAGADS (NG00034) / dbGaP (phs000234)
ADC1	Illumina Human 660W-Quad	ADC1	2731	NIAGADS (NG00022) / NACC
ADC2	Illumina Human 660W-Quad	ADC2	928	NIAGADS (NG00023) / NACC
ADC3	Illumina Human OmniExpress	ADC3	1526	NIAGADS (NG00024) / NACC
ADC4	Illumina Human OmniExpress	ADC4	1054	NIAGADS (NG00068) / NACC
ADC5	Illumina Human OmniExpress	ADC5	1224	NIAGADS (NG00069) / NACC
ADC6	Illumina Human OmniExpress	ADC6	1333	NIAGADS (NG00070) / NACC
ADC7	Illumina Infinium Human OmniExpressExome	ADC7	1462	NIAGADS (NG00071) / NACC
ADDNEUROMED	Illumina Human 610-Quad	ADM_Q	315	Synapse AddNeuroMed (syn4907804)
	Illumina Human OmniExpress	ADM_O	329	Synapse AddNeuroMed (syn4907804)
ADNI	Illumina Human 610-Quad	ADNI_Q	757	LONI ADNI
	Illumina Human OmniExpress	ADNI_OE	361	LONI ADNI
	Illumina Omni 2.5	ADNI_O25	812	LONI ADNI
	Illumina Human OmniExpress	ADNI_DOD	204	LONI ADNIDOD
ADNI3	Illumina Global Screening Array (GSA)	ADNI3	327	LONI ADNI
IIDP African Americans	Illumina Human 1M-Duo	IIDP_AA	1175	NIAGADS (NG00047)
IIDP Yorubans	Illumina Human 1M-Duo	IIDP_YOR	1264	NIAGADS (NG00047) / cf. gaaindata.org/partner/IIDP
CIDR	Illumina Human Omni1-Quad	CIDR	3101	NIAGADS (NG00015) / dbGAP (phs000160)
GenADA	Affymetrix 500K	GSK	1571	dbGaP (phs000219)
LATC	Illumina Multi-Ethnic – BU	LATC	63	RADC Rush / Latino CORE Study
NIA-LOAD	Illumina Human 610-Quad	LOAD	5220	NIAGADS (NG00020)
MARS	Illumina Multi-Ethnic – BU	MARS	708	RADC Rush / Minority Aging Research Study
MAYO	Illumina Human Hap300	MAYO_1	2099	Synapse AMP-AD (syn5591675)

MAYO2	Illumina Omni 2.5	MAYO_2	314	Synapse AMP-AD (syn5550404)
MIRAGE	Illumina Human CNV370-Duo	MIRAGE_370	397	NIAGADS (NG00031)
	Illumina Human 610-Quad	MIRAGE_610	1105	NIAGADS (NG00031)
MTC	Illumina Human OmniExpress	MTC	542	NIAGADS (NG00096)
OHSU	Illumina Human CNV370-Duo	OHSU	647	NIAGADS (NG00017)
ROSMAP	Affymetrix GeneChip 6.0 - Broad Institute	ROSMAP_1B	1126	RADC Rush / Synapse AMP-AD (syn3219045)
	Affymetrix GeneChip 6.0 - TGen	ROSMAP_1T	582	RADC Rush / Synapse AMP-AD (syn3219045)
	Illumina Human OmniExpress 12 - Chop	ROSMAP_2C	382	RADC Rush / Synapse AMP-AD (syn7824841)
	Illumina Multi-Ethnic - BU	ROSMAP_3BU	494	RADC Rush
TARCC	Affymetrix 6.0	TARCC	2718	NIAGADS (NG00097) / TARCC study
TGEN2	Affymetrix 6.0	TGEN	1599	NIAGADS (NG00028)
UPITT	Illumina Human Omni1-Quad	UPITT	2440	NIAGADS (NG00026)
UM-VU-MSSM	Illumina Human 1M-Duo, Illumina 1M	UVM_A	1153	NIAGADS (NG00042)
	Affymetrix 6.0	UVM_B	864	NIAGADS (NG00042)
	Illumina Human 550K. Illumina Human 610-Quad	UVM_C	445	NIAGADS (NG00042)
WASHU	Illumina Human 610-Quad	WASHU_1	670	NIAGADS (NG00030)
WASHU2	Illumina Human OmniExpress	WASHU_2	235	NIAGADS (NG00087)
WHICAP	Illumina Human OmniExpress	WHICAP	647	NIAGADS (NG00093)

Supplementary Table 9. Demographics of the cohorts queried among the ADSP and ADGC in-house analyses in Alzheimer's disease. AFR: African, AMR: American (central and south; admixed), EAS: East Asian, SAS South Asian, EUR: European, otherwise ADMIX: admixed of these super ancestry categories.

		Ancestry						Diagnosis		Sex - Females		Age	
Cohort	N total	AFR N	ADMIX N	AMR N	EAS N	SAS N	EUR N	CN N	AD N	CN N(%)	AD N(%)	CN μ(σ)	AD μ(σ)
ADSP WGS	16906	2240	4012	58	68	19	10509	6717	6434	4510(67.1)	3896(60.6)	78.2(8.5)	74.1(10.5)
ACT	2790	70	64	7	73	0	2576	1833	713	1000(54.6)	462(64.8)	82.9(6.5)	82.1(6.6)
ADC1	2731	92	58	47	20	0	2514	603	1946	354(58.7)	1039(53.4)	79.8(10.8)	70.7(9.5)
ADC2	928	0	2	0	0	0	926	124	707	87(70.2)	366(51.8)	80.1(9.2)	72.9(7.1)
ADC3	1526	0	5	0	0	0	1521	482	858	305(63.3)	468(54.5)	79.6(9.6)	72.5(10.3)
ADC4	1054	6	10	1	0	0	1037	420	452	257(61.2)	237(52.4)	79.2(8.7)	72.6(9.0)
ADC5	1224	0	1	0	0	0	1223	579	415	376(64.9)	226(54.5)	82.0(8.9)	74.1(8.7)
ADC6	1333	0	2	0	0	0	1331	352	567	238(67.6)	304(53.6)	80.1(8.9)	66.9(12.0)
ADC7	1462	0	4	0	0	0	1458	763	536	493(64.6)	281(52.4)	78.0(7.9)	72.8(7.7)
ADDNEURO	644	0	2	0	0	0	642	186	256	105(56.5)	164(64.1)	76.4(6.6)	73.0(6.7)
ADNI	2134	63	69	21	30	5	1945	606	761	260(42.9)	330(43.4)	78.5(7.8)	74.1(7.4)
ADNI3	327	4	12	1	4	0	306	228	24	142(62.3)	10(41.7)	72.5(6.1)	72.7(9.5)
CIDR	3101	93	2780	70	0	0	158	1505	1530	1033(68.6)	986(64.4)	74.5(9.4)	75.5(9.6)
GSK	1571	0	1	1	0	0	1569	773	798	497(64.3)	459(57.5)	73.4(7.9)	72.5(8.6)
IIDP AA	1175	815	359	0	0	0	1	1001	172	663(66.2)	107(62.2)	83.3(5.3)	83.6(6.7)
IIDP YOR	1264	1253	10	0	0	0	1	1145	104	732(63.9)	79(76.0)	82.6(5.9)	77.9(7.2)
LATC	63	13	23	24	0	0	0	15	2	15(100.0)	2(100.0)	77.4(5.4)	78.0(0.0)
MARS	708	423	275	1	0	0	1	463	79	392(84.7)	54(68.4)	79.6(6.1)	77.3(7.1)
MAYO	2413	7	24	2	4	0	2335	1225	948	642(52.4)	546(57.6)	75.5(6.5)	74.0(6.0)
MIRAGE	1502	1	28	2	0	0	1471	738	601	436(59.1)	366(60.9)	72.1(7.3)	68.8(8.6)
MTC	542	5	29	12	0	0	496	202	272	130(64.4)	157(57.7)	71.7(8.9)	72.6(9.3)
NIA-LOAD	5220	112	642	13	8	0	4445	2091	2351	1278(61.1)	1546(65.8)	70.6(12.6)	73.6(7.8)
OHSU	647	3	2	0	1	0	635	379	201	205(54.1)	127(63.2)	85.7(7.5)	85.0(6.9)
ROSMAP	2584	13	50	28	9	0	2451	1102	951	795(72.1)	690(72.6)	85.4(7.4)	84.1(6.5)
TARCC	2718	75	218	821	7	2	1557	1124	908	788(70.1)	502(55.3)	70.1(9.8)	70.1(8.9)
TGEN2	1599	0	9	1	0	1	1512	573	1005	255(44.5)	640(63.7)	80.8(8.7)	72.8(8.0)
UM-VU-MSSM	2462	5	16	0	0	0	2441	1195	1206	724(60.6)	778(64.5)	74.1(8.2)	74.2(7.9)
UPITT	2440	7	8	1	0	0	2355	896	1406	563(62.8)	908(64.6)	75.6(6.2)	73.2(6.6)
WASHU	670	0	0	0	0	0	670	202	429	125(61.9)	239(55.7)	77.9(8.7)	74.0(9.6)
WASHU2	235	10	1	0	0	0	224	116	68	65(56.0)	38(55.9)	73.7(8.6)	74.0(8.1)
WHICAP	647	0	7	0	0	0	640	554	85	335(60.5)	60(70.6)	82.7(6.7)	84.1(7.5)

Supplementary Table 10. Demographics by ancestry of ADSP and ADGC individuals included in the analyses. AAD: age-at death, AAL: age-at-last-exam, AAE: age-at-exam, AAO: age-at-onset.

			Sex	Age	Age Type			
Cohort	**Diagnosis**	**N**	**Female (%)**	**Age μ(σ)**	**AAD**	**AAL**	**AAE**	**AAO**
African ancestry ADSP WGS	AD	849	70.0%	75.0(8.8)	-	-	77.0(7.7)[1.1%]	74.9(8.8)[98.8%]
	CN	1240	75.1%	75.7(9.0)	82.5(8.9)[5.6%]	75.3(8.9)[94.3%]	-	-
European ancestry ADSP WGS	AD	4127	56.6%	73.8(11.0)	69.0(-)[0.0%]	86.2(4.1)[0.1%]	78.8(8.5)[20.0%]	72.5(11.2)[79.2%]
	CN	3020	60.8%	81.2(7.2)	85.2(7.2)[23.4%]	80.0(6.8)[76.2%]	-	-
Amerindian-Latino ancestry ADSP WGS	AD	355	69.9%	76.4(10.0)	-	-	73.4(9.1)[2.8%]	76.5(10.0)[92.4%]
	CN	1366	69.2%	74.6(7.3)	88.0(7.0)[0.3%]	74.5(7.3)[82.1%]	-	-
African ancestry ADGC imputed	AD	253	74.3%	76.8(9.3)	74.2(3.3)[2.0%]	-	79.2(8.4)[64.8%]	72.3(9.6)[32.0%]
	CN	1837	65.8%	81.9(6.6)	80.3(8.0)[2.6%]	81.9(6.5)[97.4%]	-	-
European ancestry ADGC imputed	AD	7151	58.3%	74.2(9.9)	71.5(8.1)[13.0%]	85.9(6.4)[0.7%]	83.9(6.9)[11.4%]	73.0(9.7)[74.9%]
	CN	3277	54.0%	83.5(9.1)	83.5(9.1)[100.0%]	-	-	-
Amerindian-Latino ancestry ADGC imputed	AD	1051	64.6%	74.3(10.2)	78.0(2.8)[0.2%]	87.2(8.3)[0.6%]	76.4(9.7)[0.5%]	74.2(10.2)[98.8%]
	CN	1354	70.4%	69.9(10.1)	81.4(7.5)[1.1%]	69.8(10.1)[98.9%]	-	-

Supplementary Table 11. Demographic descriptions of the different meta-analyzed GWAS from Bellenguez et al. and subset included in the HLA -level analysis (EADB, GR@ACE, GERAD, EADI, DemGene, Bonn, CCHS).

	AD or proxy-ADD cases					Controls			
	N	% females	Age	Age at onset	APOE e4 allele frequency	N	% females	Age	APOE e4 allele frequency
EADB-TOPMed	20,301	61.7	72.0±10.4	*71.1±10.5*	32.6	21,839	57.3	67.0±14.3	13.2
Belgium	*1,230*	*64.6*	*78.7±5.9*	*78.3±5.9*	*31.6*	*1,474*	*61.8*	*70.1±8.4*	*13.6*
Bulgaria	*164*	*54.9*	*65.0±8.6*	*65.1±8.6*	*22.9*	-	-	-	-
Switzerland	*182*	*64.3*	*76.0±6.6*	*76.9±6.0*	*19.2*	*388*	*55.9*	*74.8±4.0*	*10.1*
Czech Republic	*183*	*60.7*	*75.8±7.8*	-	*31.7*	*61*	*65.6*	*66.9±7.2*	*10.7*
Denmark	*403*	*57.1*	*79.6±7.8*	*79.6±7.8*	*33.7*	*654*	*54.4*	*73.1±8.5*	*15.4*
Spain	*3,273*	*67.0*	*75.3±9.0*	*75.2±9.0*	*27.2*	*1,685*	*63.3*	*69.3±12.0*	*10.0*
Finland	*1,151*	*64.0*	*70.9±8.8*	*69.8±8.5*	*42.0*	*1,806*	*51.4*	*71.8±7.1*	*15.9*
France	*1,664*	*60.2*	*67.4±11.9*	*63.2±10.8*	*33.3*	*3,106*	*63.8*	*44.9±15.4*	*11.5*
Germany	*1,628*	*60.3*	*74.8±9.4*	*74.6±9.8*	*33.1*	*2,050*	*56.1*	*74.2±8.0*	*12.3*
Greece	*614*	*63.0*	*73.1±8.0*	*72.9±8.0*	*23.8*	*1,246*	*57.3*	*73.1±5.6*	*9.2*
Italy	*3,271*	*68.1*	*73.7±8.9*	*72.2±8.7*	*25.0*	*1,317*	*56.8*	*72.2±10.5*	*8.6*
The Netherlands	*2,438*	*55.8*	*66.2±10.7*	*65.6±10.5*	*41.9*	*2,389*	*47.5*	*60.1±12.0*	*17.9*
Portugal	*80*	*75.0*	*69.9±9.2*	*69.2±8.9*	*30.0*	*74*	*75.7*	*67.2±6.8*	*17.6*
Sweden	*1,533*	*63.0*	*72.8±11.2*	*72.8±11.2*	*40.7*	*3,089*	*61.8*	*70.6±9.8*	*15.6*
United Kingdom	*2,487*	*51.1*	*68.1±10.7*	*66.4±10.1*	*34.4*	*2,500*	*51.8*	*74.4±7.2*	*12.8*
EADB-HRC	163	54.0	71.5±7.9	71.5±7.9	31.8	405	48.2	77.2±2.1	14.1
EADI	2,400	65.6	74.3±10.1	73.9±10.1	29.4	6,338	60.3	80.0±7.6	10.5
GERAD	3,030	63.2	78.1±9.3	77.8±9.3	35.1	7,153	51.2	50.7±11.7	15.4
Bonn	635	65.5	77.8±9.8	77.8±9.3	30.1	1,210	54.8	69.9±9.3	12.6
RS1	1,165	72.9	83.7±0.2	83.7±0.2	33.4	4,739	56.7	82.8±0.1	12.9
RS2	141	59.6	82.8±0.6	82.8±0.6	27.1	1,961	54.1	73.3±0.2	14.1
GR@ACE/DEGESCO	6,497	64.1	81.8±8.8	81.8±8.8	23.0	6,785	49.1	55.9±15.8	11.0
DemGene	1,693	65.5	72.2±8.8	71.6±8.8	39.5	5,926	47.7	68.5±11.1	18.2
CCHS	365	68.5	82.7±6.9	82.7±6.9	31.3	6,106	54.3	58.5±13.7	15.8
NxC	269	72.4	78.7±6.9	78.7±6.9	26.0	675	44.4	51.9±8.9	10.0

Supplementary Table 12. Demographics of the GARD and JGSCAD cohorts.

Cohort	N total	Diagnosis		Sex - Females		Age	
		CN	AD	CN	AD	CN	AD
		N	N	N(%)	N(%)	μ(σ)	μ(σ)
GARD	2127	1079	1048	604(56.0)	666(63.5)	76.1(4.1)	74.6(6.9)
JGSCAD	2022	1015	1007	583(57.4)	722(71.7)	77.0(5.9)	73.0(4.3)

Supplementary Table 13. Demographics by ancestry of ROSMAP and ADGC individuals included in the neuropathology analyses. AAD: age-at death, AAL: age-at-last-exam, AAE: age-at-exam, AAO: age-at-onset.

Phenotype	Diagnosis	N	Phenotype	Sex	Age	Age Type			
			μ(σ)	Female (%)	Age μ(σ)	AAD	AAL	AAE	AAO
Tau Braak staging	AD	5148	5.14(0.91)	2926(56.8%)	73.5(10.2)	71.0(8.1)[12.5%]	87.0(5.0)[0.4%]	83.8(7.0)[14.2%]	71.8(9.8)[72.9%]
	CN	1262	2.44(1.32)	733(58.1%)	86.3(7.7)	86.3(7.7)[100.0%]	-	-	-
	MCI/Others	1072	2.98(1.62)	553(51.6%)	79.8(10.1)	76.9(8.3)[10.3%]	84.5(7.2)[4.3%]	83.8(6.8)[22.7%]	78.2(11.3)[49.8%]
Neuritic plaques density	AD	4146	2.77(0.42)	2279(55.0%)	73.6(10.5)	69.7(3.1)[0.1%]	86.8(5.1)[0.4%]	84.1(6.8)[15.8%]	71.5(9.9)[83.7%]
	CN	896	1.04(1.10)	543(60.6%)	87.7(7.4)	87.7(7.4)[100.0%]	-	-	-
	MCI/Others	860	0.86(0.92)	447(52.0%)	80.5(10.4)	85.2(7.4)[3.3%]	84.5(7.2)[5.3%]	83.8(6.8)[28.3%]	78.2(11.5)[57.4%]

Supplementary Table 14. Demographics by ancestry of ROSMAP and ADGC individuals included in the dual-pathology analyses. AAD: age-at death. AD-LB-: without AD and LB pathology, AD+LB-: only AD pathology without LB pathology, AD-LB+: only LB pathology without AD pathology, AD+LB+: dual pathology (AD and LB).

		Sex	AAD
Phenotype	**N**	**Female (%)**	**Age μ(σ)**
AD-LB-	1290	722(56.0%)	87.5(8.2)
AD+LB-	2641	1478(56.0%)	83.0(9.4)
AD-LB+	298	146(49.0%)	87.0(8.2)
AD+LB+	889	453(51.0%)	82.4(9.3)

Supplementary Table 15. Demographic information on cohorts of Cerebrospinal Fluid analyses.

					Diagnoses				
Country	**Cohort**	**n**	**age (sd)**	**male**	**AD**	**MCI**	**control**	**other dem**	**data type**
Belgium	DEM	587	78.3 (7.3)	40%	72%	27%	1%	0%	Genotype level
Finland	ADGEN	226	70.2 (8.0)	34%	89%	1%	10%	0%	Genotype level
France1	BALTAZAR	420	77.0 (6.7)	45%	43%	57%	0%	0%	Genotype level
France2	MEMENTO	389	69.2 (8.9)	47%	0%	100%	0%	0%	Summary statistics
France3	CNRMAJ-Rouen	127	66.0 (8.7)	47%	100%	0%	0%	0%	Genotype level
Germany1	Delcode	465	71.7 (5.9)	52%	13%	23%	64%	0%	Summary statistics
Germany2	KND	309	67.3 (8.7)	57%	18%	82%	0%	0%	Genotype level
Germany3	TUM	151	70.2 (9.2)	48%	98%	1%	0%	1%	Genotype level
Germany4	PAGES	136	73.4 (7.7)	40%	70%	30%	0%	0%	Genotype level
Germany5	UHB	111	70.3 (7.2)	42%	69%	30%	1%	0%	Genotype level
Netherlands	ADC & Pearl ND	2936	64.1 (8.9)	59%	42%	10%	24%	23%	Genotype level
Spain1	ACE	609	72.7 (8.2)	43%	27%	59%	8%	6%	Summary statistics
Spain2	SIGNAL & SPIN	394	70.6 (8.0)	43%	34%	45%	19%	2%	Genotype level
Spain3	Valdecilla	98	67.0(9.0)	39%	10%	37%	45%	8%	Summary statistics
Sweden1	Birth cohort & Clin. AD	856	75.0 (9.4)	45%	51%	0%	49%	0%	Genotype level
Sweden2	Uppsala university	260	71.0 (6.3)	46%	58%	37%	0%	6%	Genotype level

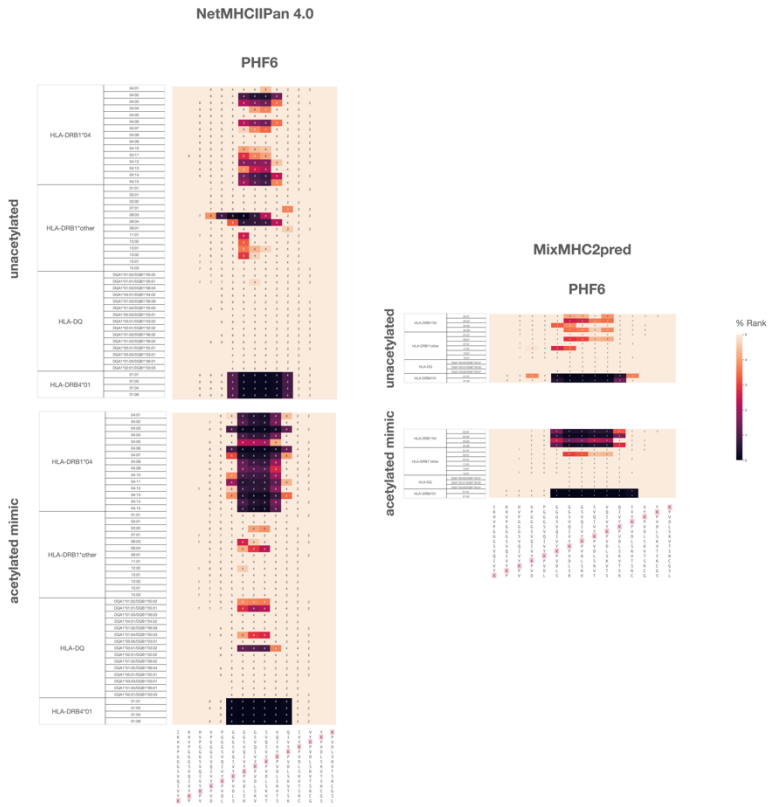
DEM=Antwerp prospective dementia cohort. All, except the Swedish Birth cohort & clinical AD samples, are part of EADB.

Supplementary Table 16. Demographics for the IPDGC, McGill, NINDS, NGRC, Oslo, PPMI, APDGC, UK Biobank datasets included in Yu et al. (2021) analysis[9].

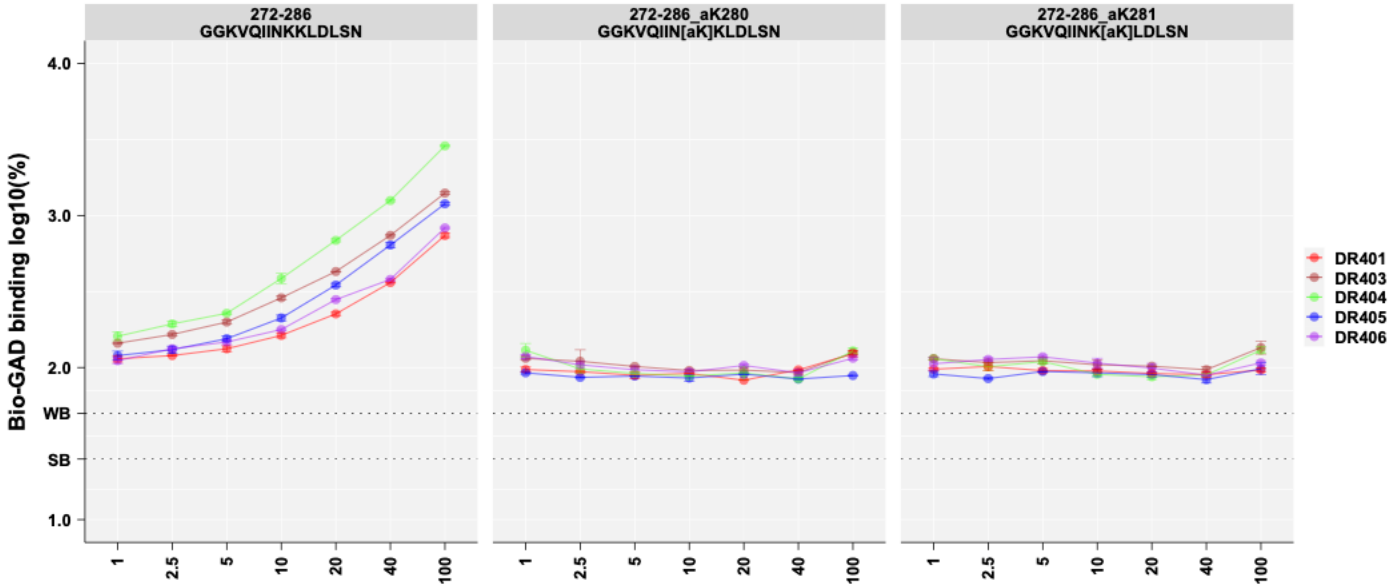
Cohort	Diagnosis	N	Female (%)	Age at onset	Age at recruitment or last visit
APDGC	PD	603	27.7	77.4 (8.4)	
	CN	295	50.3		81.9 (12.7)
IPDGC	PD	5163	35.6	61.2 (12.6)	
	CN	5389	44.2		64.3 (14.8)
McGill	PD	1240	36.8	58.5 (10.6)	
	CN	995	49.0		44.15 (14.7)
NINDS	PD	847	40.4	66.1 (11.1)	
	CN	773	58.1		58.7 (16.4)
NGRC	PD	1922	32.6	58.3 (12.0)	
	CN	1938	61.2		70.3 (14.1)
Oslo	PD	474	35.9	55.7 (11.3)	
	CN	459	42.5		61.8 (11.0)
PPMI	PD	398	33.3	59.8 (9.9)	
	CN	159	33.5		61.1 (10.7)
UKB Proxy	Proxy-PD	14422	56.8		58.5 (7.1)
	CN	308694	53.6		56.8 (8.0)
UKB cases	PD	1490	37.1		62.8 (5.4)
	CN	33251	53.7		56.8 (8.0)

Supplementary Table 17. Demographics for the 23andMe, East-Asians-PD, and LARGE-PD datasets. Sex and Age were partly not available for the 23andMe individuals.

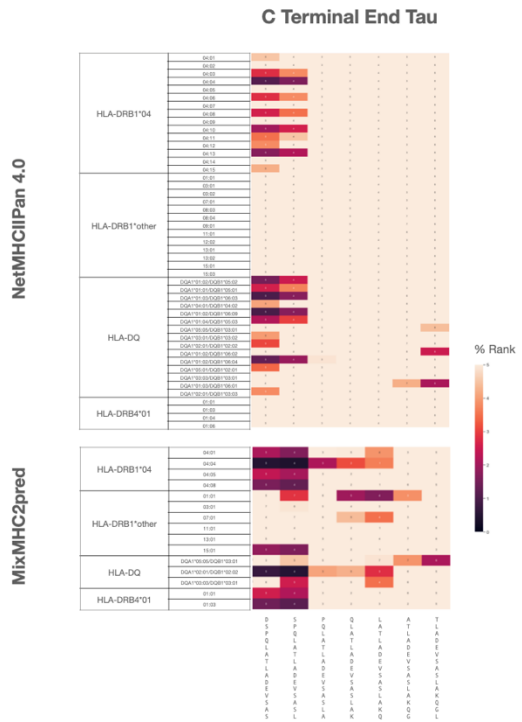
			Sex	Age
Cohort	**Diagnosis**	**N**	**Female (%)**	**Age μ(σ)**
European ancestry	PD	3261	-	-
23andMe	CN	29499	-	-
European ancestry	PD	866	-	-
23andMe	CN	32538	-	-
European ancestry	PD	6476	38.9%	-
23andMe	CN	302042	48.2%	-
European ancestry	PD	2448	39.1%	-
23andMe	CN	571411	54.9%	-
East Asian ancestry	PD	988	55.0%	58.8(10.1)
Japaneses (Naito et al. 2021)	CN	2521	45.2%	49.9(14.2)
East Asian ancestry	PD	2279	42.9%	59.7(10.7)
Chineses (Foo et al., 2020)	CN	2021	45.0%	52.6(13.5)
East Asian ancestry	PD	2536	43.0%	66.6(9.7)
Singaporeans/Malays (Foo et al., 2020)	CN	21840	55.2%	60.8(7.6)
East Asian ancestry	PD	1494	54.2%	66.1(9.4)
South Koreans (Foo et al., 2020)	CN	599	56.4%	71.0(9.3)
East Asian ancestry	PD	199	35.2%	63.8(10.0)
Hong-Kongers (Foo et al., 2020)	CN	166	32.5%	61.9(7.9)
East Asian ancestry	PD	216	51.9%	72.4(9.5)
Taiwaneses (Foo et al., 2020)	CN	225	55.6%	72.4(7.7)
Amerindian/European-Latinos	PD	807	47.0%	61.7(12.8)
South Americans (Loesch et al., 2021)	CN	690	67.0%	56.5(14.6)



Supplementary Figure 1. HLA binding predictions for PHF6*. Predictions were made using NetMHCIIPan 4.0[31] (left) and using Mixed MHC pred 2 Server[32] (right). Cmap indicates a percentile rank generated by comparing the peptide's score against the scores of five million random 15 mers selected from SWISSPROT database (best score = 0%; worst score = 100%). 15mer sequences incorporating 15mer sequences incorporating PHF6* in its unacetylated (bottom) or K2380 mimic acetylated (top) form. Annotations within heat map show the position of the lysines of interest (K280 for PHF6*, see highlighted sequences) within the 9mer core that is predicted to bind to the HLA molecule. 9mer cores excluding the lysine of interest were not predicted for binding and left blank.



Supplementary Figure 2. PHF6* peptides do not bind *HLA-DRB104 sybtypes**. Each tau peptide at different concentrations competed with biotinylated peptide binding to *HLA-DRB1**04:01, *HLA-DRB1**04:03, *HLA-DRB1**04:04, *HLA-DRB1**04:05, and *HLA-DRB1**04:06. Tau peptides with fluorescence that was lower than 25% and 25-50% of biotinylated peptide are considered strong (SB) and weak (WB) binders, respectively. None of the peptides bind these subtypes.



Supplementary Figure 3. HLA binding predictions for tau C terminal end. Predicted binding of 15mer sequences of the C terminal end of tau for various HLA molecules is shown using (a) NetMHCIIPan 4.0 [31]. Cmap indicates a percentile rank generated by comparing the peptide's score against the scores of five million random 15 mers selected from SWISSPROT database (best score = 0%; worst score = 100%). (b) Mixed MHC pred 2 Server [32]. Cmap indicates the percent of random peptides that would have a score higher than the peptide provided among peptides of sizes 12-25 amino acids (best score = 0%; worst score = 100%).