
**eMethods.** Supplemental methods

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This supplementary material has been provided by the authors to give readers additional information about their work.
Quantification of AD pathological indices

Brains were removed and weighed following a standard procedure. One hemisphere was cut coronally into 1cm slabs and fixed in 4% paraformaldehyde for dissection of diagnostic blocks. Six micron sections were cut for modified Bielshowsky silver stain for assessment of neuritic plaques, diffuse plaques, and neurofibrillary tangles in the frontal, temporal, parietal, entorhinal cortex, and the hippocampus. Each case received a neuropathological AD diagnosis based on the corresponding Braak staging for neurofibrillary tangle pathology and the CERAD estimates of neuritic plaque pathology, as recommended by the National Institute on Aging-Reagan criteria. AD diagnosis by Reagan criteria required either an intermediate likelihood AD (i.e., at least Braak stage 3 or 4 and CERAD moderate plaques) or a high likelihood (i.e., at least Braak stage 5 or 6 and CERAD frequent plaques). Neuropathological diagnoses were made by a board-certified neuropathologist blinded to age and all clinical data.

Immunohistochemistry was performed using the ABC method with alkaline phoshatase as the color developer. Multiple tissue blocks from 8 brain regions, including entorhinal cortex, CA1/subiculum of the hippocampus, superior frontal cortex, mid prefrontal cortex, inferior temporal cortex, angular gyrus, anterior cingulate cortex, and calcarine cortex were cut into 20µm sections to assess amyloid load and PHFtau positive tangles. Briefly, 20 (for smaller regions) to 90 (for cortex) video images of Aβ stained sections were captured and processed in an automated, multistage computational image analysis protocol. Mean percentage (percentage area positive for amyloid) was computed within and across brain regions. Quantification of tau tangle density was performed with the stereological mapping station and yielded a measure of the density of tangles (per mm²), averaged within and across brain regions.
Quality control of brain DNA methylation data

Raw data from Illumina 450k platform were processed following a rigorous pipeline for data quality control (QC). Briefly, original TIFF images and IDAT files were converted to beta-values for each probe, ranging from 0 (no methylation) to 1 (full methylation). Corresponding detection p-values ($p > 0.01$) were used to filter out probes with poor quality. In addition, we removed cross-reactive and polymorphic probes. Missing beta-values were imputed using k-nearest neighbor algorithm. Samples with less than 450,000 high quality probes or low quality bisulfite conversion rates were excluded from the final dataset. Duplicate or mis-annotated samples were detected and removed using principal component analysis.

In general, methylation level at individual CpG sites follows a bell-shaped unimodal distribution. By contrast, the global distribution of mean methylation across the genome follows a distinct bimodal distribution, where roughly a third of CpGs are extremely hyper-methylated and a third are extremely hypo-methylated. Details on global patterns of DNA methylation in human brain has been described in a recent review.

RNA-Seq Transcript Expression Profiling

RNA was extracted using Qiagen's miRNeasy mini kit and the RNase free DNase Set. Samples were quantified by Nanodrop and quality evaluated by Agilent Bioanalyzer. RNA-Seq library was prepared on Broad Institute’s Genomics Platform using strand specific dUTP method with poly-A selection. Each sample has been sequenced using Illumina HiSeq platform to a depth of 50 million paired-end reads of 101 bp each. At the time of analyses, 541 samples met quality (Bioanalyzer RNA integrity (RIN) score $> 5$) and quantity thresholds (5ug). RNA expression of
coding genes in the vicinity of the reported AD loci were queried and examined for association with the AD pathologies, as well as correlations with DNA methylation.

**Statistical testing for \( \psi \)**

The omnibus statistic \( \psi \) assesses a hypothesis that none of CpGs was associated with the outcome of interest, and has advantages of reducing the burden for multiple testing as well as enhancing power. The asymptotic distribution of \( \psi \) under original Fisher’s method did not apply since individual CpGs within each target loci were correlated with each other. Therefore the statistical significance of \( \psi \) was tested by using random permutations of the outcome while preserving the correlation structure of CpGs. An estimate from the actual data was compared with the null distribution generated by random permutations, such that an estimate distant/far from the mean would be unlikely under the null hypothesis. Briefly, the outcome was randomly permuted and used in the regression analysis for each CpG. The resulting \( p \)-values for individual CpGs were combined to derive the estimate for \( \psi \). The process was repeated, which gives a permuted distribution of \( \psi \) under the null hypothesis. Maximum permutation limit was set up to 1,000,000. A permuted \( p \)-value was estimated as the proportion of permutations for which the estimates for \( \psi \) were greater than or equal to the observed value from the actual data.

**References**

**eTable 1.** CpGs in *SORL1* locus nominally associated with pathological AD

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**eTable 2.** CpGs in *SORL1* locus nominally associated with Aβ load

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**Table 3.** CpGs in *SORL1* locus nominally associated with PHF tau tangle density

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**Table 6.** CpGs in *ABCA7* locus nominally associated with PHF tau tangle density

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eTable 7. CpGs in *HLA-DRB5* locus nominally associated with pathological AD

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**Table 8.** CpGs in *HLA-DRB5* locus nominally associated with Aβ load

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Table 9. CpGs in *HLA-DRB5* locus nominally associated with PHF tau tangle density

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eTable 10. CpGs in SLC24A4 locus nominally associated with pathological AD

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**eTable 11.** CpGs in *SLC24A4* locus nominally associated with Aβ load

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**eTable 12.** CpGs in *SLC24A4* locus nominally associated with PHF tau tangle density

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**Table 13.** CpGs in *BIN1* locus nominally associated with pathological AD

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**Table 15.** CpGs in *BIN1* locus nominally associated with PHF tau tangle density

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**eTable 16.** Association of *BIN1* transcript expression with Aβ load

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eFigure 1. Correlation of SORL1 DNA methylation and RNA expression
eFigure 2. Correlation of \textit{ABCA7} DNA methylation and RNA expression
eFigure 3. Correlation of $BIN1$ DNA methylation and RNA expression