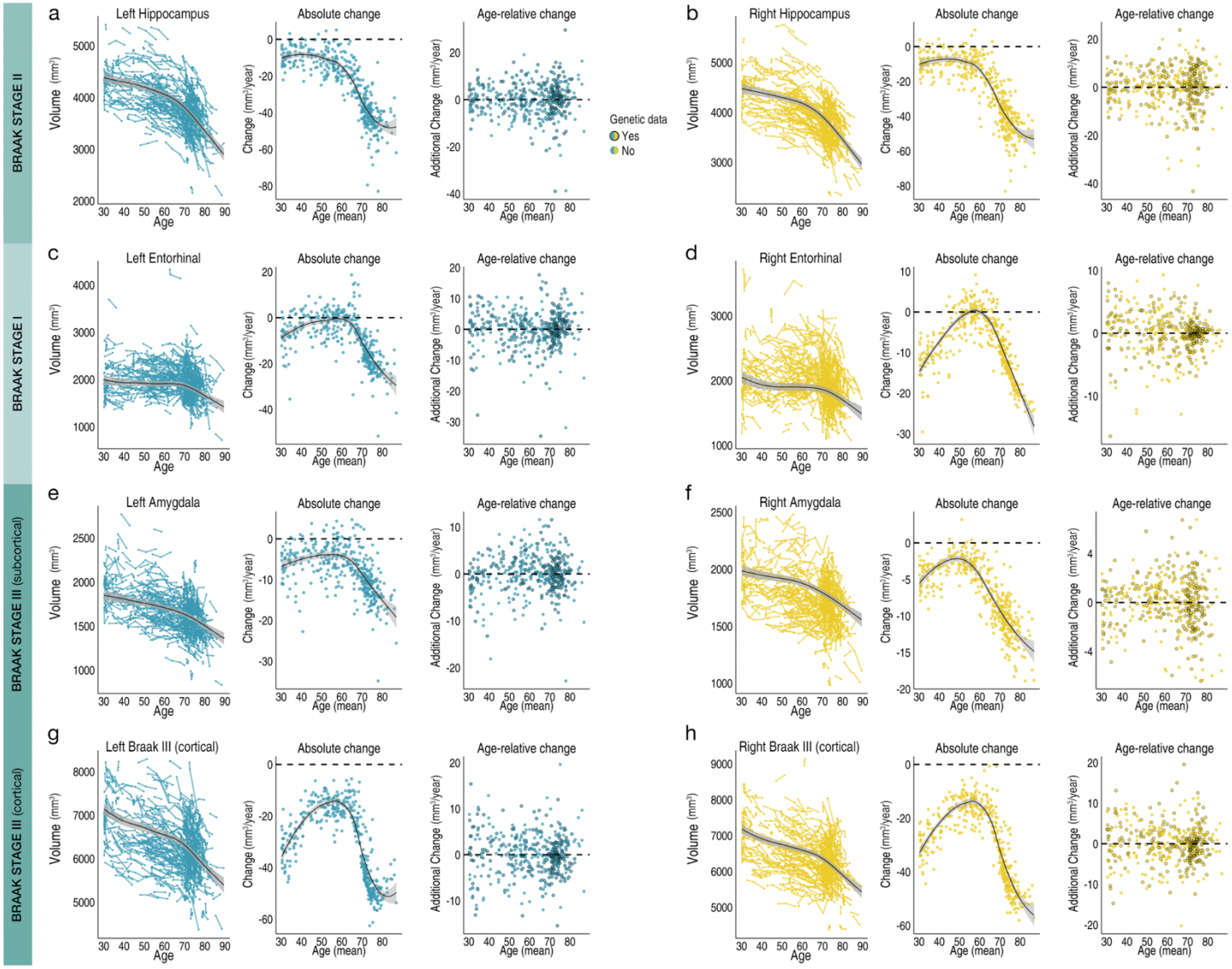
Brain change trajectories in healthy adults correlate with Alzheimer's related genetic variation and memory decline across life

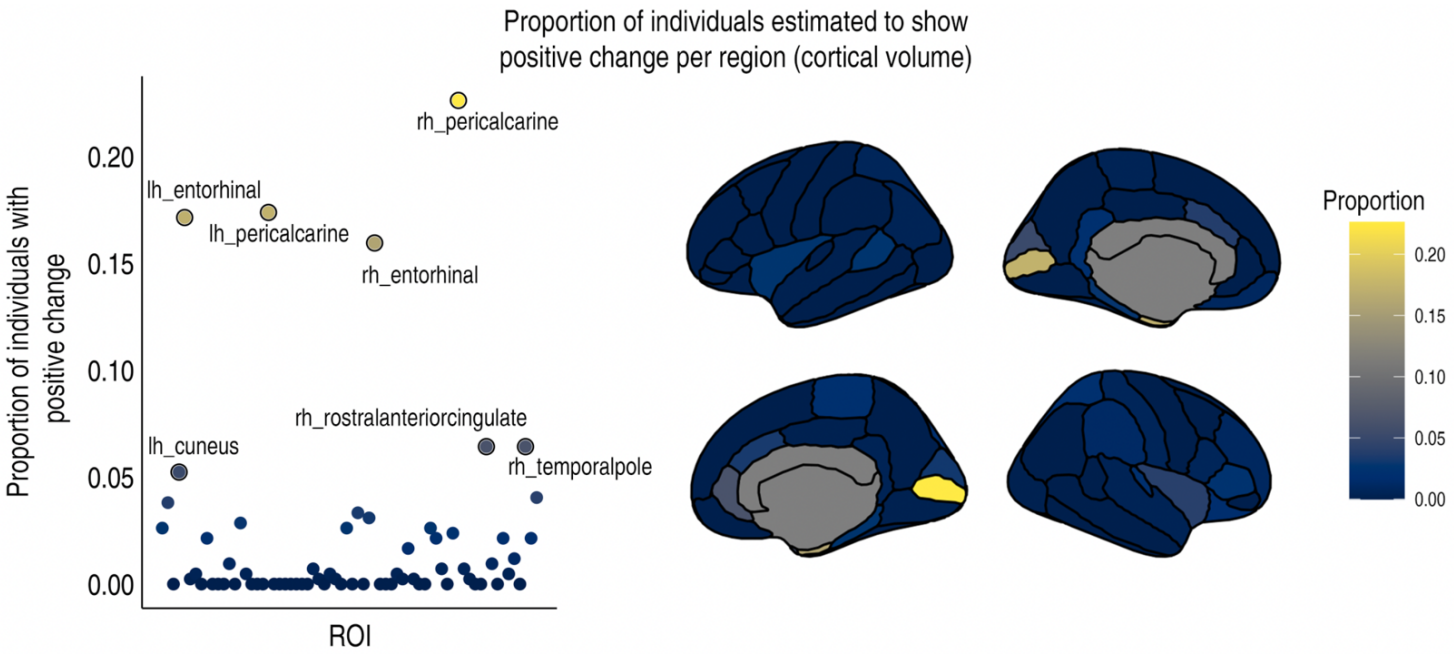
Roe et al.

SUPPLEMENTARY INFORMATION

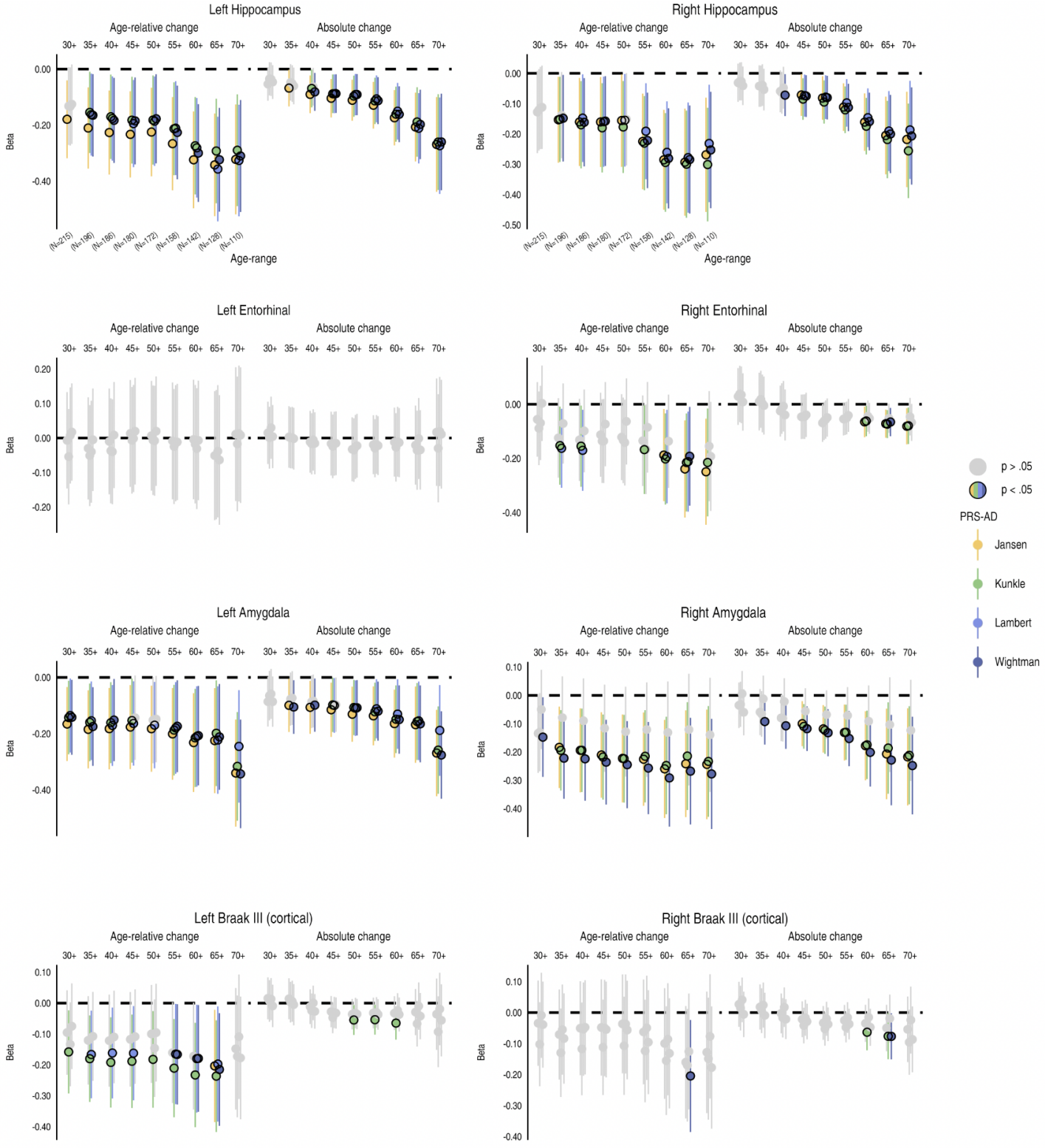


SUPPLEMENTARY FIGURE 1

Adult lifespan trajectories in early Braak regions. Exclusively longitudinal data was used to estimate individual-specific age-relative change in bilateral hippocampus (Braak stage II; **a** left, **b** right), entorhinal cortex (Braak stage I; **c,d**), amygdala (Braak stage III subcortical; **e,f**), and a Braak Stage III cortical region (**g,h**), modelling the adult lifespan trajectories using GAMMs with random individual-specific slopes. Leftmost plots in each: adult lifespan trajectory from 30-89 years (data corrected for sex and scanner, lines connect longitudinal observations). Middle plots: absolute change per individual (datapoints) as a function of their mean age across timepoints. Rightmost plots: estimated age-relative change per individual (i.e. individual-specific slopes) as a function of their mean age across timepoints. Black stroke indicates whether or not genetic data was available per participant and thus whether the datapoint was included in the PRS-AD association tests. Trajectories depict mean measures and error bands depict 95% CI.

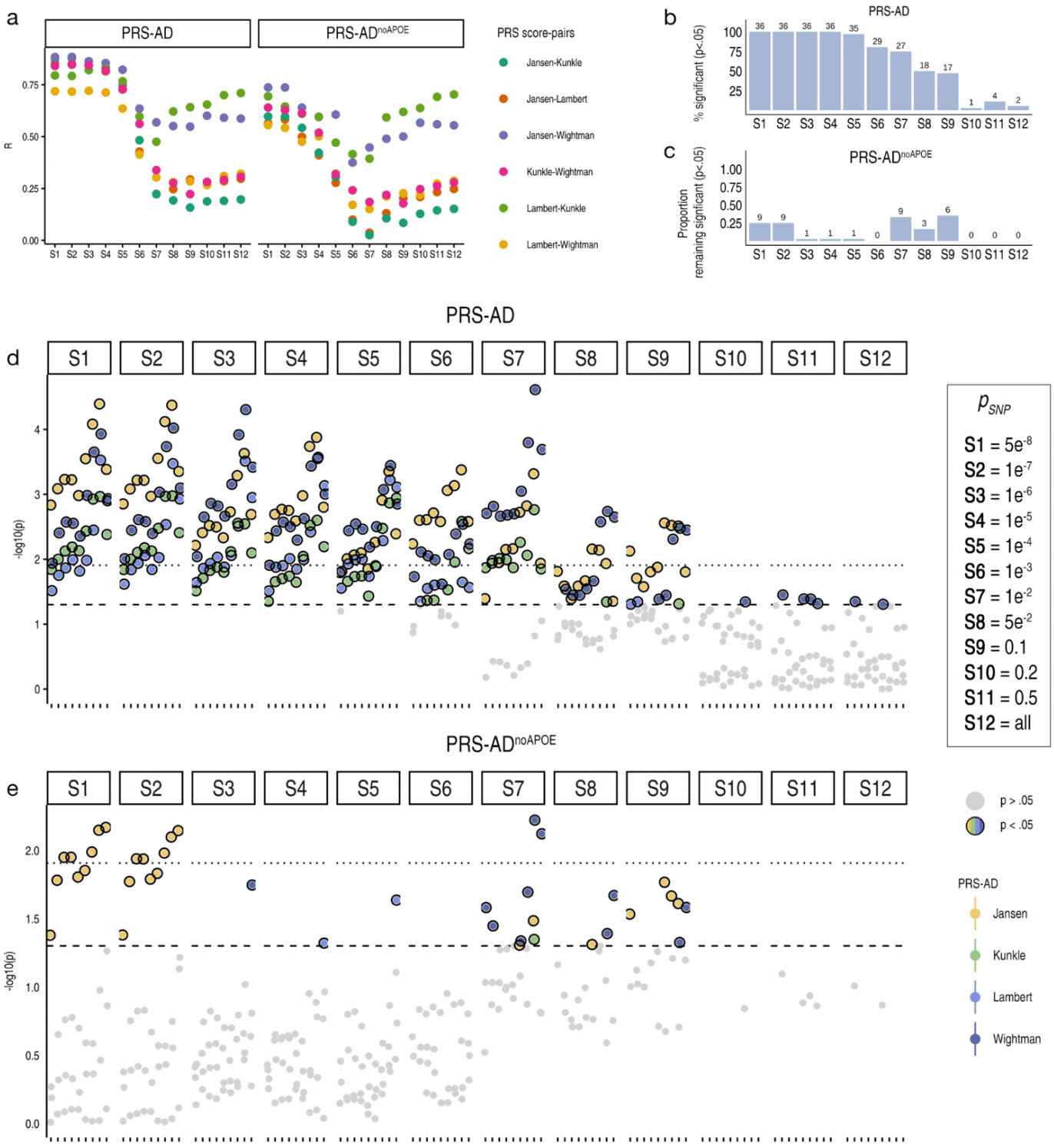


SUPPLEMENTARY FIGURE 2
Assessment of brain change estimates. We estimated the proportion of individuals estimated to show positive absolute change in cortical volume (i.e., growth) across the cortex. The median proportion of individuals estimated to show growth across cortical regions was 0.002. Left and right entorhinal cortex were clear outliers, suggesting these measures may be less reliable. Cortical volume change was estimated using the adult lifespan discovery sample (1430 scans from 420 individuals aged 30 to 89 years).



SUPPLEMENTARY FIGURE 3

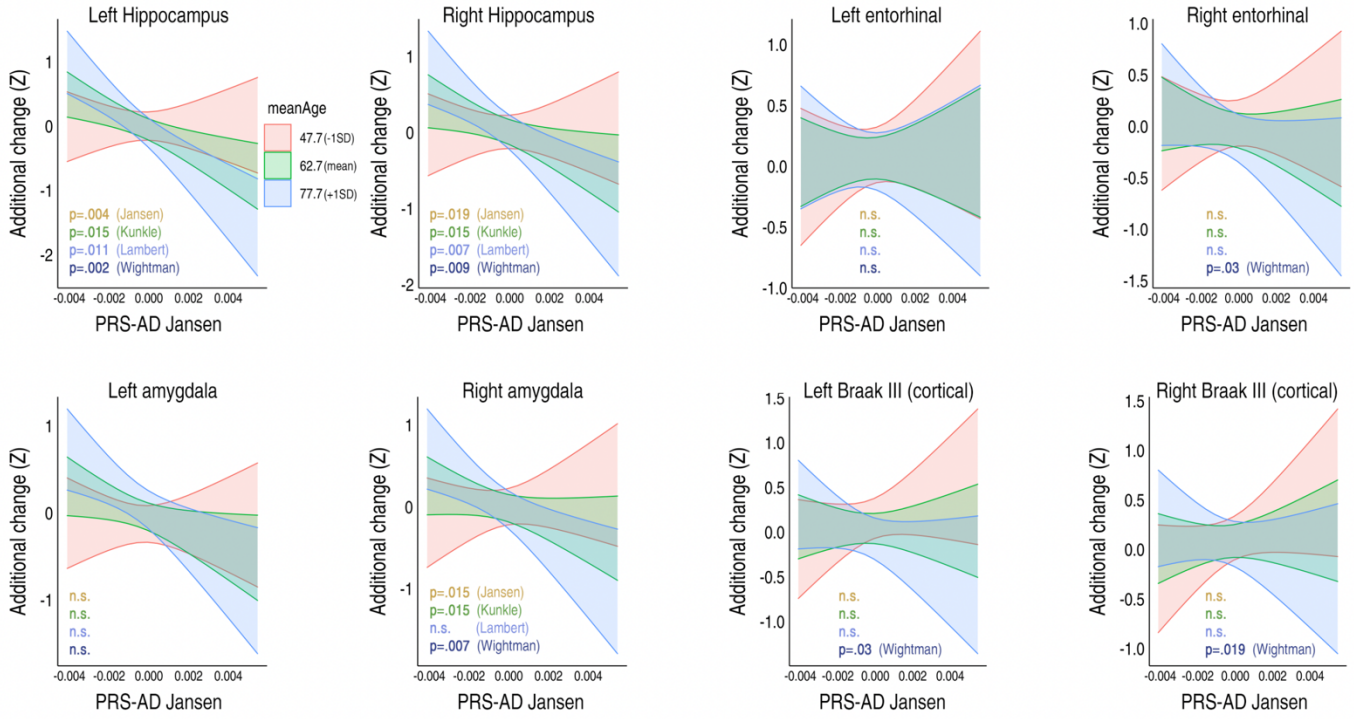
Sensitivity analysis: quality control. Univariate associations after discarding data from subjects with observations >3SD in any of the 8 a priori ROIs (maximum genetic N = 215; exact sample size per age-range subset is shown in top row). The set of 576 PRS-AD beta estimates correlated at r = .95 with the results reported in the main paper (See Figures 1-2). Error bars depict 95% CI.



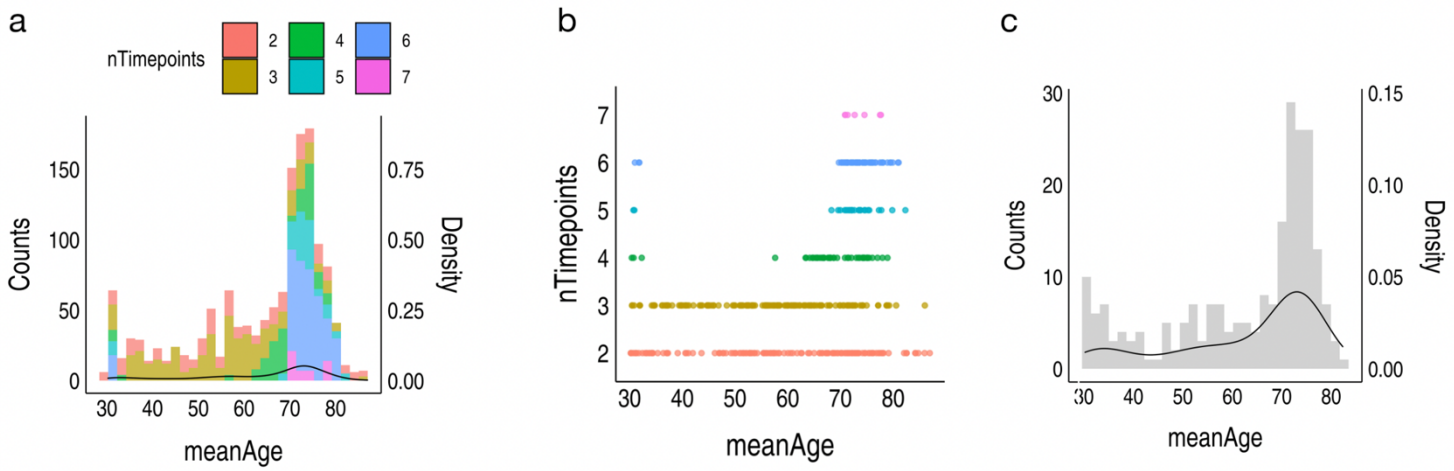
SUPPLEMENTARY FIGURE 4

Sensitivity analysis: 12 alternative PRS-AD thresholds. a The correlation between PRS-AD scores dropped when including more SNPs in the PRS, rendering the results between scores less comparable at more liberal thresholds. **b** The percentage count (y-axis) of significant PRS-AD associations with age-relative change in left hippocampus for different PRS computation thresholds ($p < .05$ [uncorrected]; 9 age-subsets x 4 scores). **c** Where the PRS-AD association was significant ($p < .05$ [uncorrected]) we tested whether it remained significant with PRS-AD^{noAPOE} ($p < .05$ [uncorrected]). The plot illustrates the proportion of PRS-AD associations in b that remained significant with PRS-AD^{noAPOE} and their counts. **d-e** The associations visualized (9 age-subsets on dashed x-axis). The first column depicts the p-values using genome-wide significant SNPs as in the main paper. Coloured points/black outline depicts associations at $p<.05$ (uncorrected). As these post-hoc tests are for illustrative purposes and not independent of our initial main analysis, the

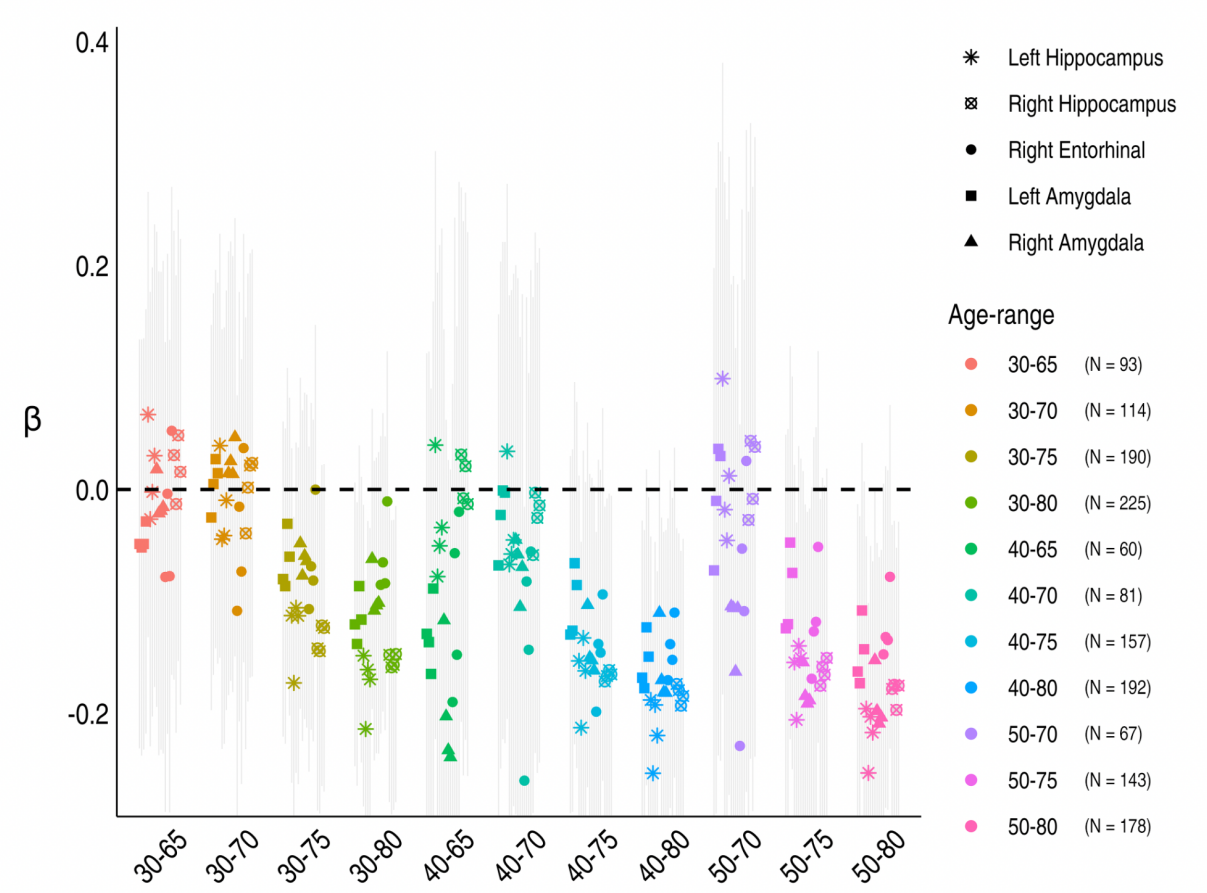
FDR-correction level applied is the same as across the 576 PRS-AD tests in the main paper. Dashed and dotted horizontal lines depict uncorrected and corrected significance levels.



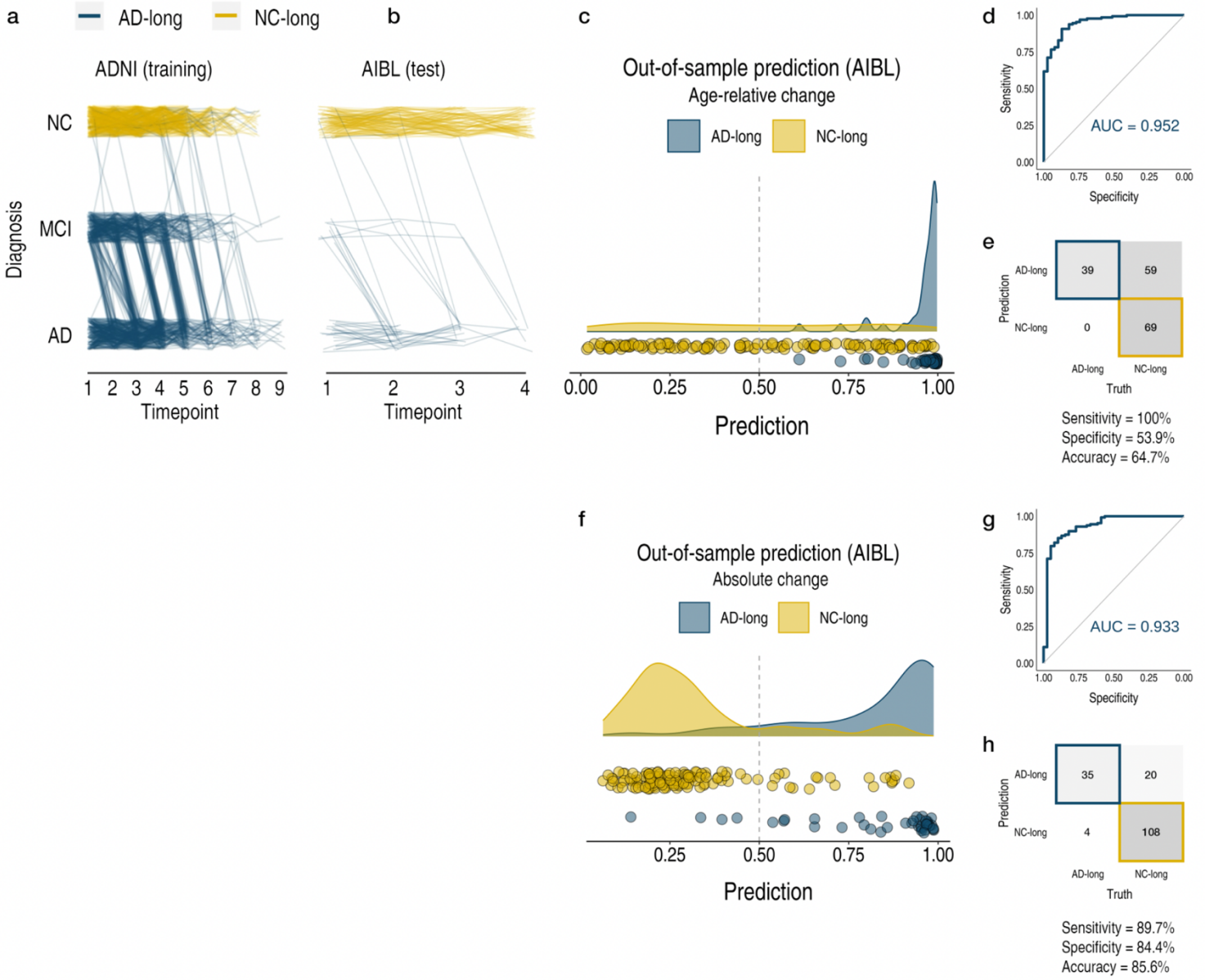
SUPPLEMENTARY FIGURE 5
PRS-AD x mean age interactions. Results from alternative analyses dependent on power across the full age-range (30-89 years). Linear PRS-AD * mean age interactions upon age-relative change that were significant at p < .05 (uncorrected) are in bold. None of the tests survived FDR-correction across the 32 tests performed in this analysis (see Supplementary Table 6). Error bands depict 95% CI.



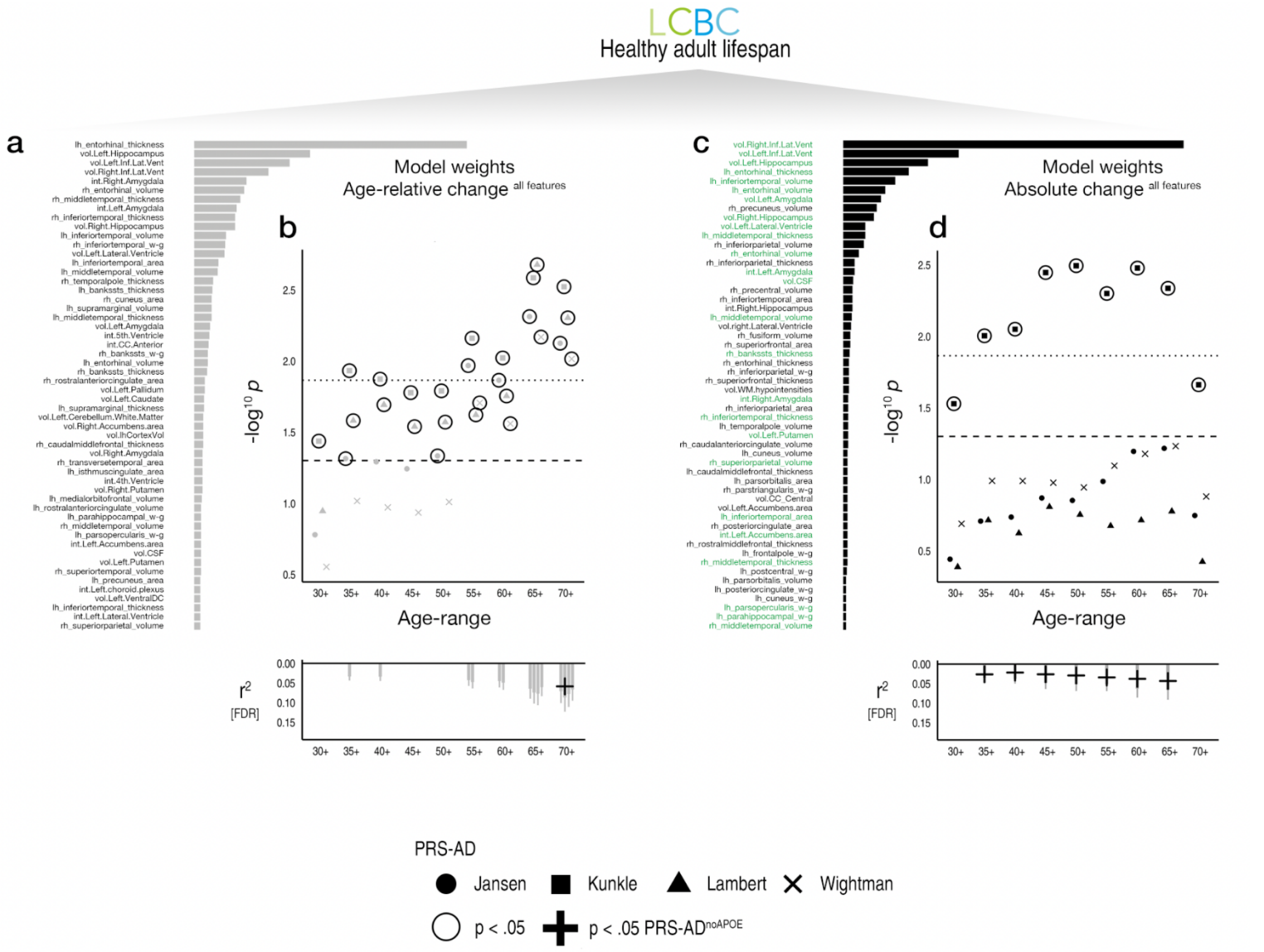
SUPPLEMENTARY FIGURE 6
Age and timepoint distributions. a Age and **b** timepoint distribution of the LCBC discovery cohort used to estimate individual-specific brain change (obs = 1430; N = 420). **c** Age distribution of the genetic observations in this sample (N = 229).



SUPPLEMENTARY FIGURE 7
Beta estimates for linear PRS-AD associations with age-relative change in early Braak stage regions, tested across alternative age subsets discarding the data from the oldest individuals. Estimates are shown for Braak regions where PRS-AD was associated with change in the main analyses. All analyses were corrected for mean age, sex, N timepoints, interval between first and last timepoint, and 10 genetic PCs. Error bars depict 95% CI. The data indicated the genetic associations were not driven by only the oldest-old, though older adults likely contributed more of the individual differences in brain change signal. Error bars depict 95% CI.

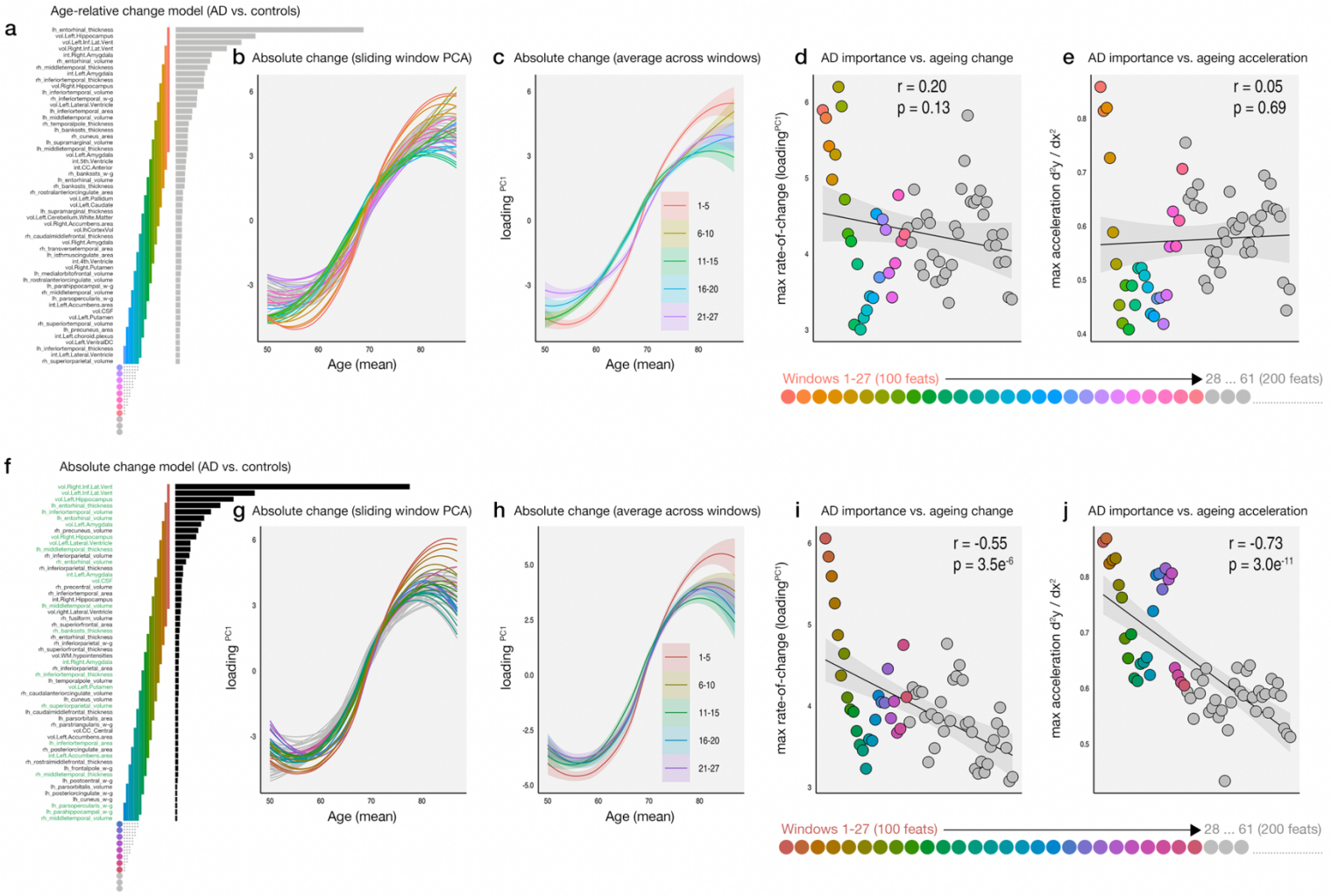


SUPPLEMENTARY FIGURE 8
Longitudinal ADNI training and AIBL test data. **a** Longitudinal grouping in ADNI training data (as in Fig. 3A) **b** Longitudinal grouping in the independent AIBL test data. **c-e** Out-of-sample prediction (AIBL) for the binary classifier based on age-relative change, including receiver operator curve (d), confusion matrix and performance metrics (e). **f-h** Out-of-sample results for an alternative binary classifier based on absolute change for comparison.



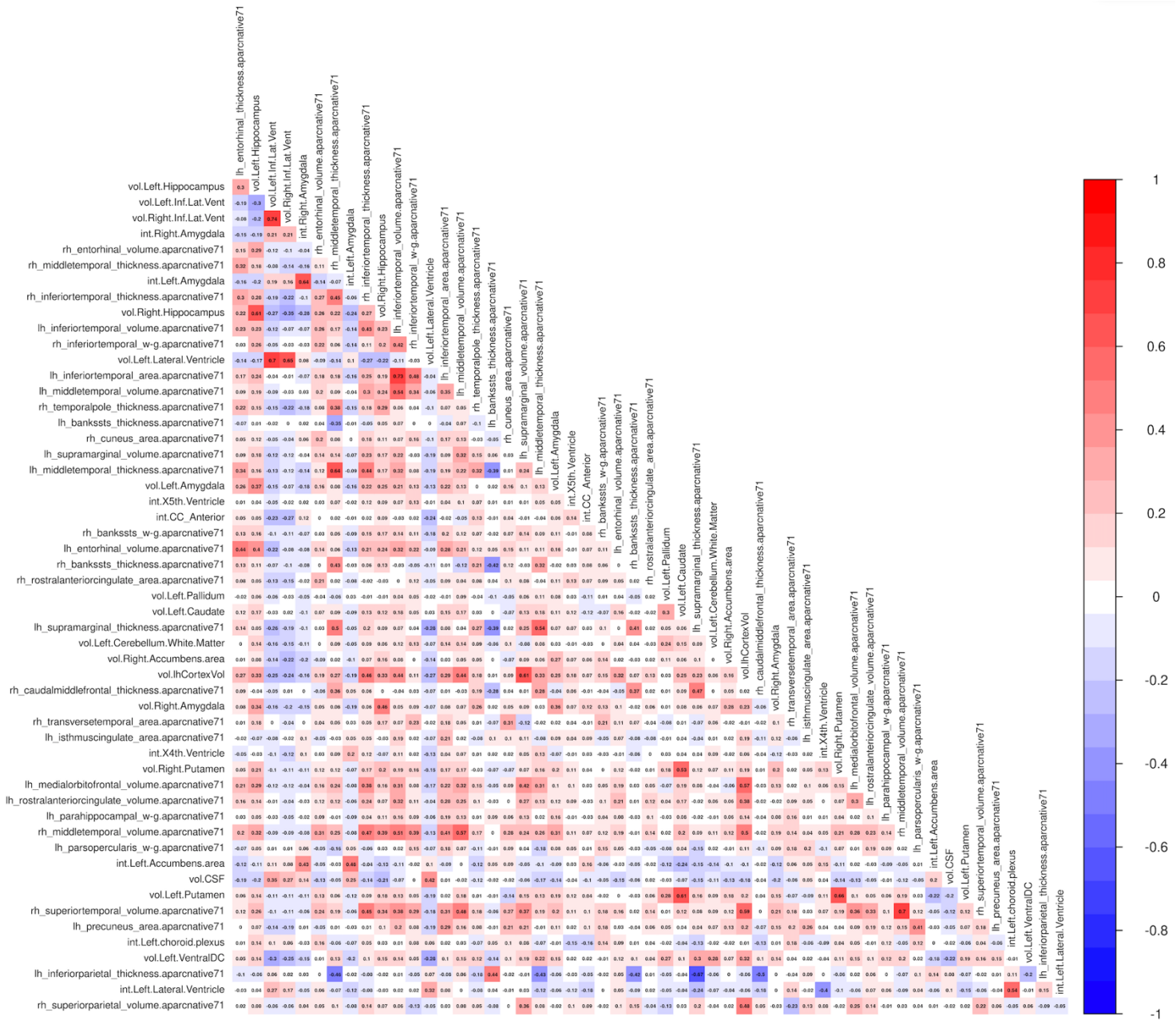
SUPPLEMENTARY FIGURE 9

ADNI-derived ML models applied to the healthy adult lifespan. a Top features for classifying *AD-long* from *NC-long* individuals in ADNI data based on age-relative change (as in Fig. 4A). We directly applied the ADNI-derived model weights to the LCBC healthy adult lifespan dataset. **b** PRS-AD associations in the LCBC healthy adult lifespan dataset after predicting with the learned model weights from the ADNI-derived model (i.e. LCBC as test data). The dependent variable is the model-implied log odds of having AD ($probAD^{relChange}$). Datapoints show (-log10) p-values for PRS-AD associations with $probAD^{relChange}$, tested at progressively older age-ranges, for all four scores. Dashed line indicates p=.05, and datapoints with black stroke depict significant PRS-AD associations at p<.05. Datapoints above the dotted line are significant at p(FDR)<.05 (FDR-correction applied across all 72 PRS-AD tests in this analysis). Bottom plot shows partial r^2 for PRS-AD where the association survived FDR-correction. Where FDR-corrected significant, we retested the association after removing *APOE* ($PRS\text{-}AD^{noAPOE}$). Partial r^2 of $PRS\text{-}AD^{noAPOE}$ is depicted by a black cross if the association remained significant ($p < .05$). **c** Top features for classifying *NC-long* from *AD-long* individuals in ADNI data based on absolute change. Green text indicates whether the feature was present or not in the list of top features from the model shown in a. **d** PRS-AD associations in the LCBC dataset after predicting with ADNI-derived model weights based on absolute change. Here, the dependent variable is the model-implied log odds of having AD ($probAD^{absChange}$). Bottom plot shows partial r^2 for PRS-AD where the association survived FDR-correction, and partial r^2 of $PRS\text{-}AD^{noAPOE}$ is depicted by a black cross if the association remained significant after removing *APOE* ($p < .05$). Error bars depict 95% CI. lh=left hemisphere, rh=right hemisphere, vol=volume (subcortical); int=intensity (subcortical); w-g=grey/white matter contrast. Subcortical features (aseg atlas) are delineated with ".", whereas cortical features (aparc atlas) are delineated with "_". Error bars depict 95% CI.

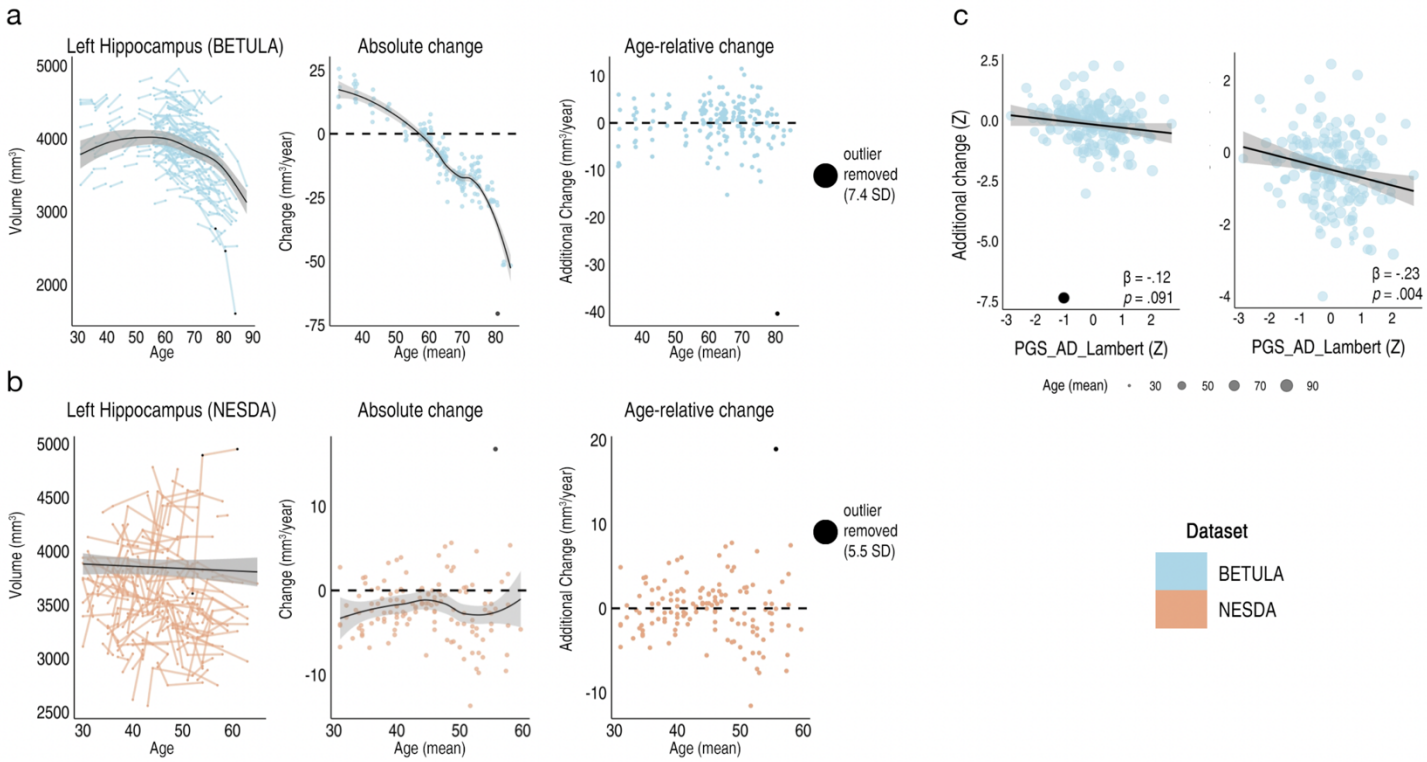


SUPPLEMENTARY FIGURE 10

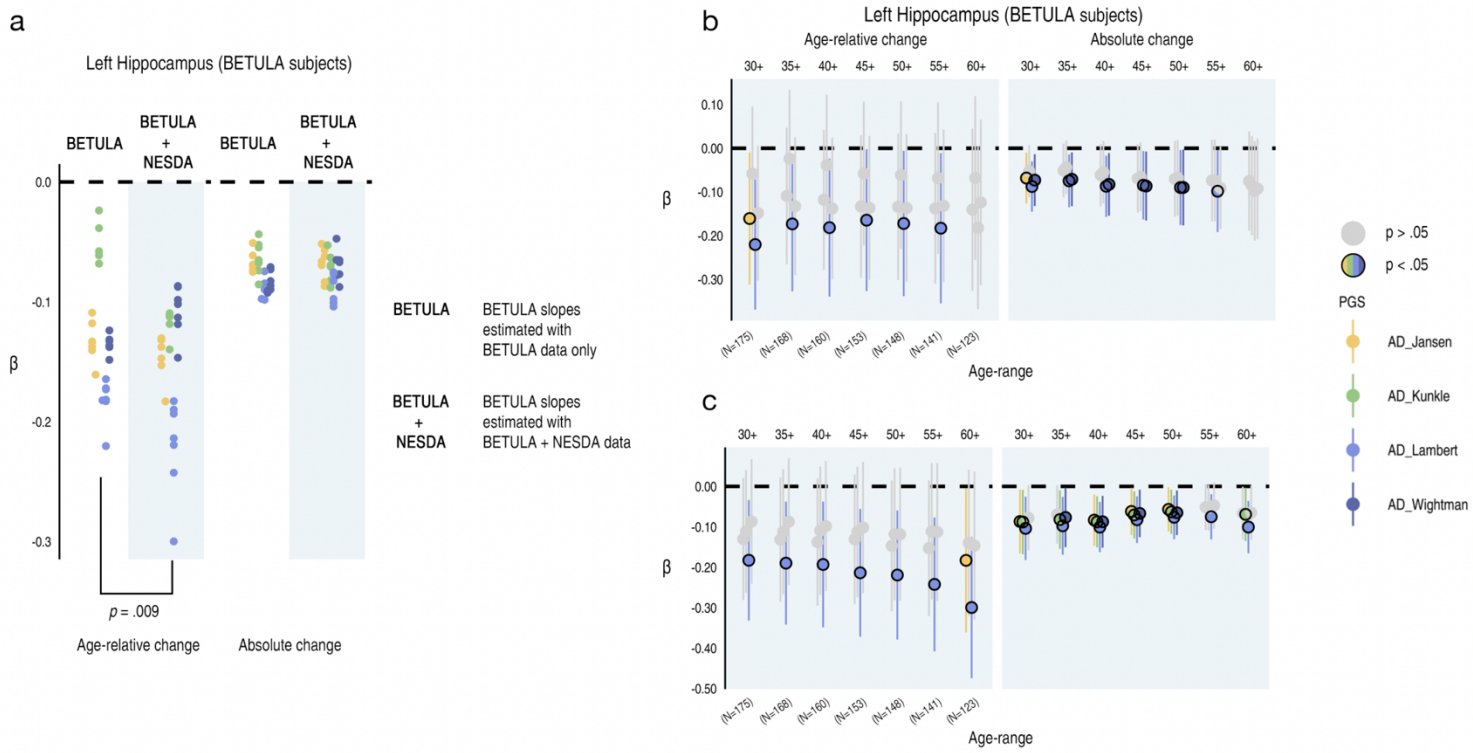
PCA-based sliding window trajectory analysis. Within the 50-89 years age-range, we ran a sliding window PCA, iteratively calculating PC1 across 20 features with a step size of 3. We calculated the trajectory of absolute change within each window. Colours depict the selection of features across which we calculated PC1 (here including hippocampal and amygdala volumes) and link with the subsequent plots. Feature selections and colours high up in the importance matrix thus represent features that contribute most to separating AD patients from controls (i.e., AD importance), whereas selections that are comparatively lower down contribute less to this prediction. **a** Top features for classifying *AD-long* from *NC-long* individuals in ADNI data based on age-relative change (as in Fig. 4A). **b** Absolute change as a function of mean age (across timepoints) within each window. Since the y-axis in b, c, g and h represents change, the slope of the curves represents acceleration. Feature selections across the top 100 features are shown in colour (27 windows), whereas trajectories in grey represent feature selections beyond the first 100 features (up to 200 features; 61 windows). Features most important for separating AD patients from controls showed the highest rate-of-change and steepest acceleration in healthy individuals. **c** As in b, except the trajectories are averaged across the feature windows depicted in the key to better show their differences. Ribbons depict 95% CI. **d** Maximum rate-of-change and **e** acceleration of the brain aging trajectories in healthy individuals, plotted against AD-importance (x-axis shows the feature selections and thus implicitly represents AD-importance). Note that the plots confirm that maximal rate-of-change and acceleration in healthy individuals is found in brain features that are most important for separating AD patients from controls. **f-j** As in a-e, except feature selections follow the order of importance of features for classifying *AD-long* from *NC-long* individuals based on absolute change (i.e. as in Supplementary Fig. 9C). Again, maximal rate-of-change and acceleration in healthy individuals was found in brain features that are most important for separating AD patients from controls. Error bands depict 95% CI.



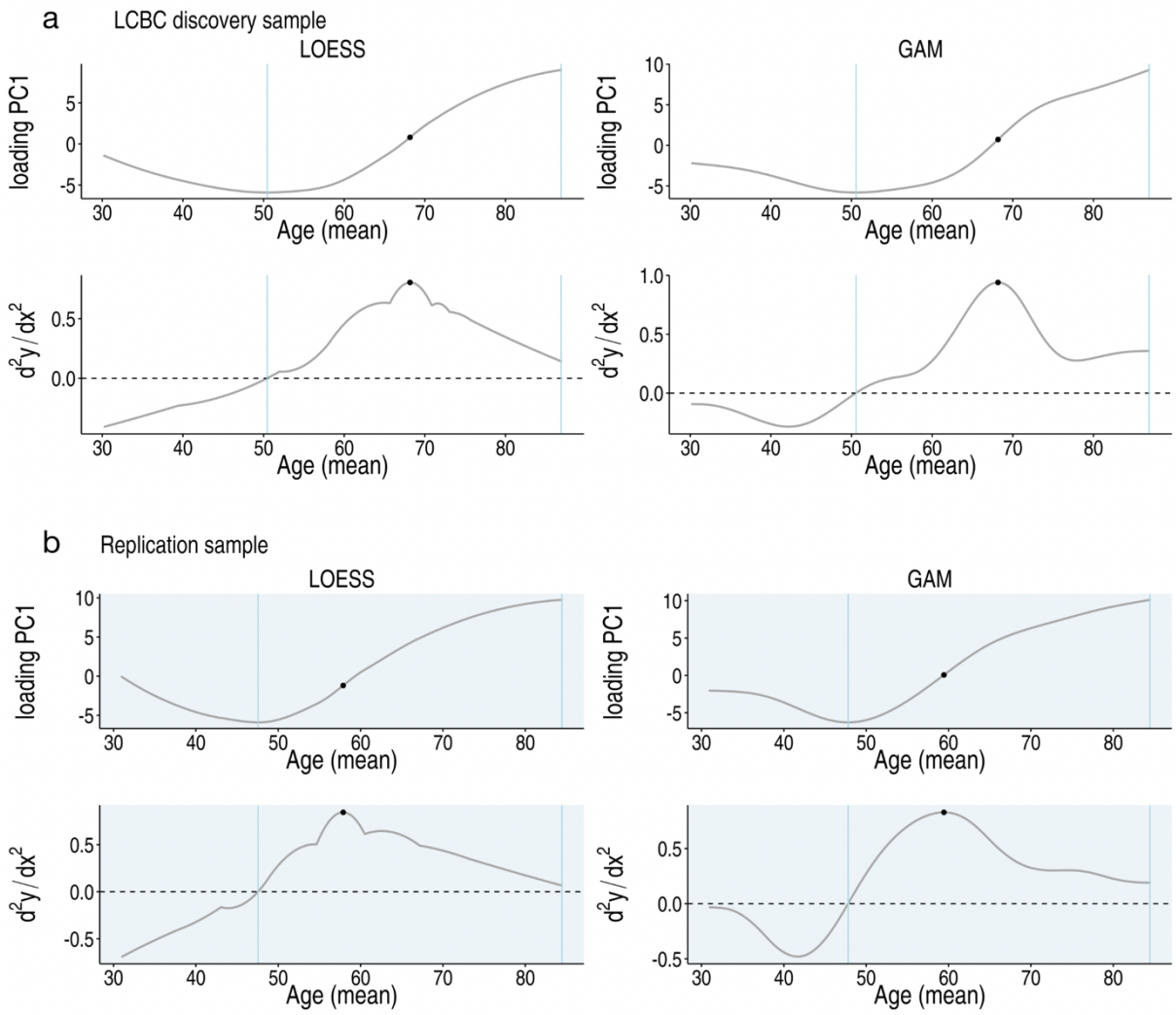
SUPPLEMENTARY FIGURE 11
Correlations between age-relative change estimates in the top 54 AD-accelerated features in LCBC data. Correlations are shown for the age-range 50-89 years. 54 features are shown due to the inclusion of hippocampal and amygdala volumes not otherwise included in PC1^{relChange}.



SUPPLEMENTARY FIGURE 12
Initial GAMM trajectory analysis separately in the BETULA and NESDA longitudinal replication samples (max 3 timepoints) revealed a strong outlier in the left hippocampal slope data in each sample (datapoint in black indicates the large negative outlier in BETULA data [7.4 SD; top row], and the large positive outlier in NESDA data [5.5 SD; bottom row]). **a-b** These outliers are shown for each of their timepoints (leftmost plots), and absolute (middle plots) and age-relative change estimates (rightmost plots). **c** In BETULA, the strong outlier had a large influence on the tested PRS-AD association with age-relative change, shown before (β = -.12, *p*=.091; left plot) and after removal of this outlier (β = -.23; p=.004; right plot; one example score). The two samples were then collated into a single replication dataset and these outliers were removed. Error bands depict 95% CI.

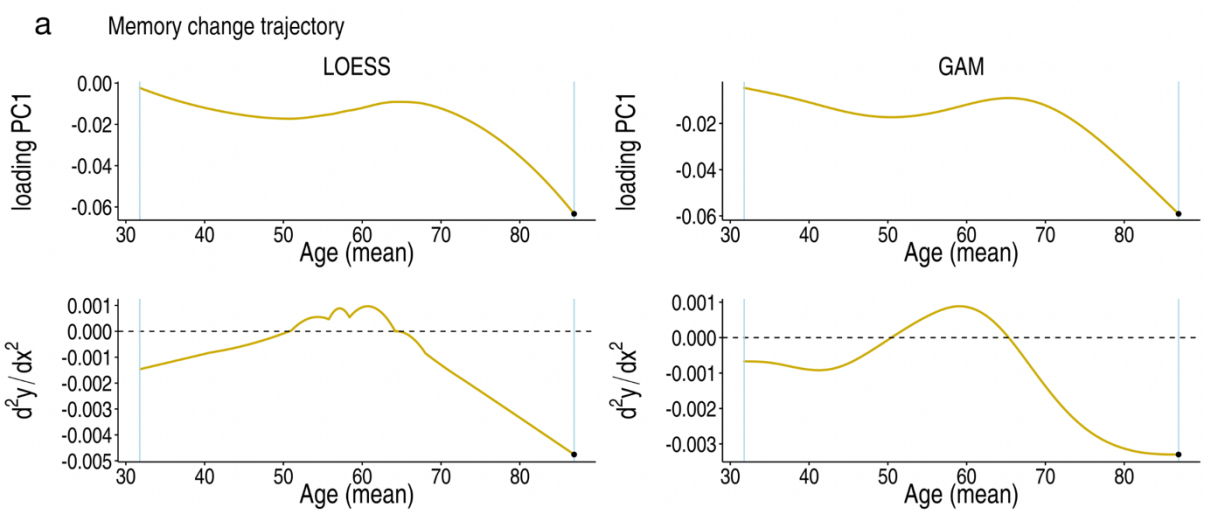


SUPPLEMENTARY FIGURE 13
a In the same 175 individuals in BETULA, PRS-AD beta estimates with age-relative change in left hippocampus were significantly lower (p = .009 [one-sided]) when their individual-specific slopes were estimated together with NESDA data, relative to when estimated using only BETULA data. **b** Beta estimates for individual-specific slopes (BETULA subjects) estimated from a GAMM using only BETULA data. **c** Beta estimates for individual-specific slopes (same 175 subjects) estimated from a GAMM across BETULA and NESDA data. Including more longitudinal observations in the GAMM helped optimize the estimation of individual-specific slopes, boosting the power to detect PRS-AD associations in the same individuals. Error bars depict 95% CI.



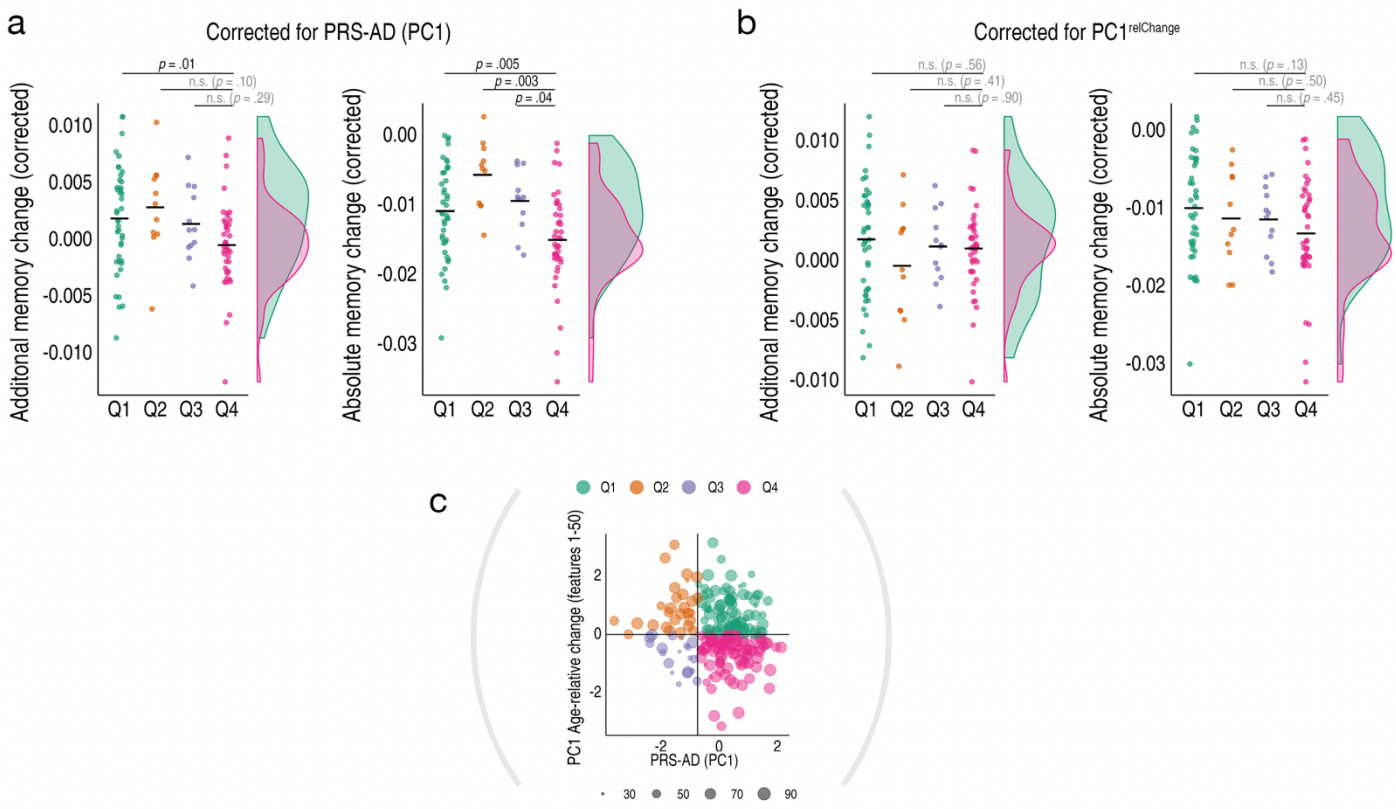
SUPPLEMENTARY FIGURE 14

Multivariate change in AD features ($PC1^{absChange}$) in healthy adults
a For the LCBC adult lifespan discovery sample, top row shows the rate of absolute change ($PC1^{absChange}$) which reflects the first derivative (i.e., the y-axis represents rate-of-change; see Methods). Bottom row shows the derivative of this curve, which therefore represents acceleration (i.e., second derivative). Left column: estimated using a Locally Estimated Scatterplot Smoothing (LOESS) model (as in Fig. 4 and Fig. 5). Right column: estimated using a General Additive Model (GAM). The first and second blue line markers indicate the onset of negative change (crossing to positive on the second derivative) and the point of maximum rate-of-change in AD features in healthy adults. Black points indicate the point of maximum accelerated change. **b** As above, shown for the adult lifespan replication sample.



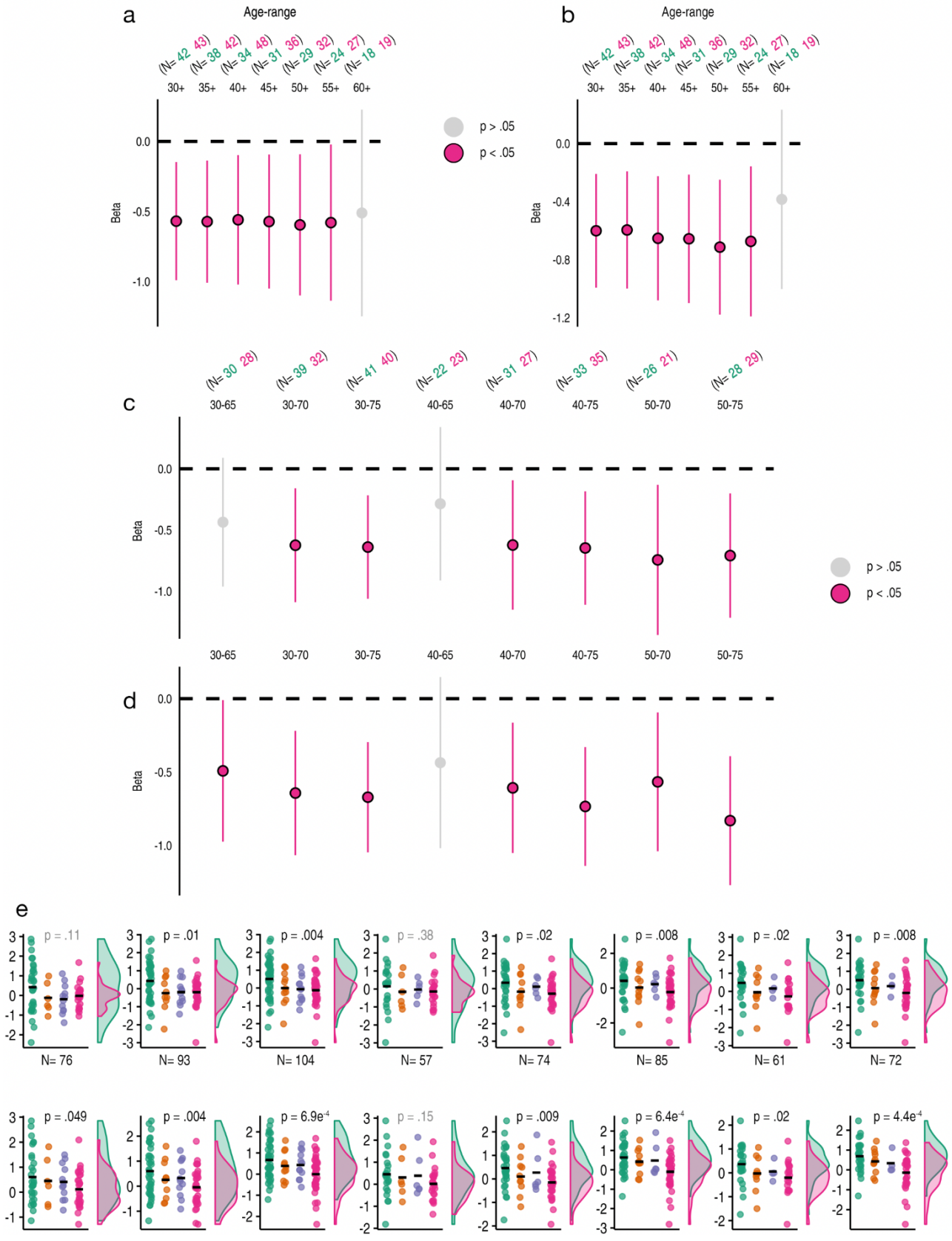
SUPPLEMENTARY FIGURE 15

Memory change trajectory across the healthy adult lifespan
a Top row shows the rate of absolute memory change which reflects the first derivative (i.e., the y-axis represents rate of change; see Methods). Bottom row shows the derivative of this curve, which therefore represents acceleration (i.e., second derivative). Left column: estimated using a Locally Estimated Scatterplot Smoothing (LOESS) model (as in Fig. 6). Right column: estimated using a General Additive Model (GAM). The first and second blue line markers indicate the onset of negative memory change (estimated at the minimum mean age of our sample) and the point of maximum rate-of-change in memory in healthy adults (estimated at the maximum mean age of our sample). Black points indicate the point of maximum accelerated negative memory change.



SUPPLEMENTARY FIGURE 16

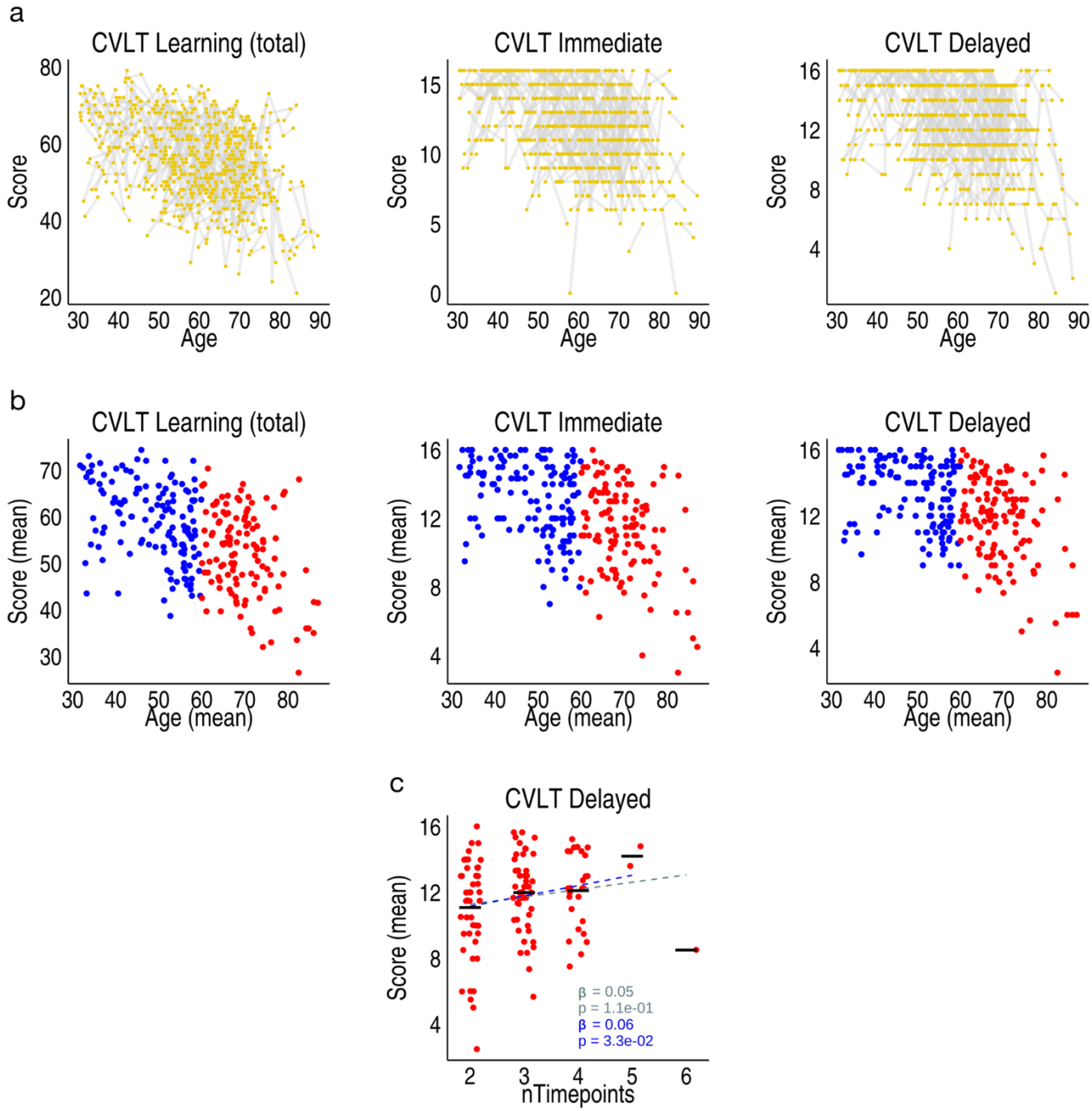
Memory change results for alternative models correcting for additional covariates. a. Correcting for group-differences in genetic risk (PRS-AD; PC1 across four scores), and **b.** group-differences in multivariate brain change across AD-accelerated features (PC1^{relChange}). Group-differences in memory change persisted when controlling for differences in genetic risk but not when correcting for group-differences in brain change. Similar results were found correcting for the number of *APOE*-ε4 alleles. Other covariates: mean age, sex, number of timepoints, interval between first and last timepoint. **c.** Shown again for comparison, the association that was used to define four quadrant-groups representing the conjunction of brain and genetic risk factors (as in Fig. 4D; pink group depicts individuals high on both).



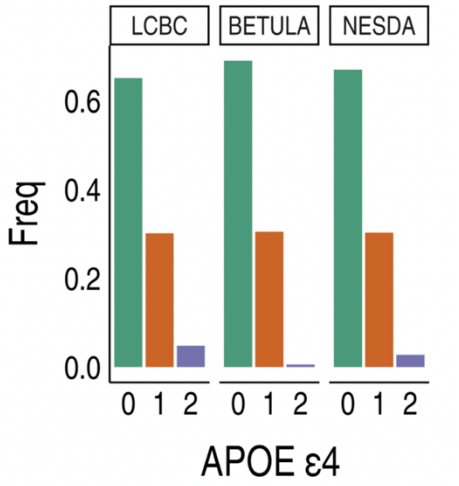
SUPPLEMENTARY FIGURE 17

Memory change results for models across different age-ranges. a Group-differences in age-relative and **b** absolute memory change tested at progressively older age-ranges (main model). **c** Group-differences in age-relative and **d** absolute memory change across alternative age subsets (redundant subsets removed; standardized betas, 95% CI error bars). Colour depicts significance at p < .05 [uncorrected]. As these tests shed light on group-differences that survived FDR-correction in our main hypothesis-driven model, no further correction was applied. **e** The distribution of

group differences in age-relative (upper row) and absolute memory change (lower) within each age subset in C and D (Y axis shows Z values). The p-value of the group-difference is given above each plot. The reported group differences in memory change were not driven by the oldest adults but evident within the age-ranges 30-65 and 30-70 (all models and datapoints corrected for covariates: mean age, *APOE*-ε4 carriership, sex, N timepoints, and interval between first and last timepoint). The N of the two groups wherein the difference was found (a-d) and N of the overall model is given (e).



SUPPLEMENTARY FIGURE 18
a Spaghetti plot of raw memory scores on the CVLT learning subtests for the subset of individuals used to estimate memory change (707 observations of 261 individuals). **b** Individual-level mean scores across memory timepoints. Colour depicts mean age cut-off of 60 to highlight the overlapping distribution with younger participants. **C** In participants aged 60 and above, the data indicated that individuals with more repeat visits tended to have better mean CVLT Delayed scores (most challenging subtest). However, note the association was not significant across the full data, but became significant when discarding the only individual with 6 timepoints (who seemingly detracted from this pattern). For reader interpretation, models, estimates and p-values are shown, with and without this individual included.



SUPPLEMENTARY FIGURE 19
Frequency of *APOE*-ε4 genotypes per sample. Fishers exact tests indicated there were no significant differences between samples in the number of ε4 carriers (all p > .45). A significant difference in *APOE*-ε4 genotype was found between LCBC and BETULA only (p = .03), likely reflecting more ε4 homozygotes in LCBC (see also Supplementary Table 8).

Dataset	Cohort	N Unique	N obs	N Timepoints 2, 3, 4, 5, 6, 7	Mean Time Interval (SD)	Interval Range	Mean Age (SD)	Age-Range	Sex (f/m)	N Genetic
Discovery	LCBC	420	1430	135, 147, 45, 26, 60, 7	2.1(2.8)	0.14 – 11.1	63.7 (14.4)	30.1 – 89.4	248 / 172	229
Replication	BETULA	182	449	97,85, -----	3.0 (2.7)	3.5 – 7.7	64.3 (11.9)	30.9 – 87.9	85 / 97	175
	NESDA	138	331	83, 55, -----	2.5 (3.3)	1 – 10	45.1 (7.9)	30 – 65	91 / 47	118

SUPPLEMENTARY TABLE 1
Description of the LCBC discovery healthy adult lifespan sample and the two Lifebrain cohorts that comprised the replication sample. Intervals are in years.

Cognitive Test		Mean (SD)	N	N obs
Mean MMSE (SD)	**Age < 60**	29.2 (0.7)	170	380
N = 417, obs = 1266	**Age > 60**	28.8 (1.0)	247	886
Mean FSIQ (SD)	**Age < 60**	117.3 (8.0)	166	341
N = 415, obs = 857	**Age > 60**	119.5 (10.2)	249	516
Mean CVLT Learning (SD)	**Age < 60**	57.7 (10.1)	138	350
N = 261, obs = 707	**Age > 60**	51.0 (10.2)	123	357
Mean CVLT Immediate (SD)	**Age < 60**	12.9 (10.0)	''	''
''	**Age > 60**	11.0 (3.1)	''	''
Mean CVLT Delayed (SD)	**Age < 60**	13.1 (2.5)	''	''
''	**Age > 60**	11.6 (3.0)	''	''

SUPPLEMENTARY TABLE 2
Cognitive test scores in the LCBC discovery sample. Individual-level scores are calculated as the mean across their timepoints. Mini Mental State Examination (MMSE) and Full Scale IQ (Weschler Abbreviated Scale of Intelligence) are given for all participants from the adult lifespan sample used to estimate brain change for which the data was available. CVLT scores are given for the subset used to estimate memory change (i.e., after discarding data from participants in memory training projects; Methods; Supplementary Note 1).

Dataset	Group	N unique	N Obs	N Timepoints 2	3	4	5	6	7	8	9	Mean Time Interval (SD)	Interval Range	Mean Age (SD)	Age-Range	Sex (f/m)	Mean MMSE All Timepoints	Mean MMSE Longitudinal Timepoints	Mean Clinical Dementia Rating (CDR) All Timepoints	Mean Clinical Dementia Rating (CDR) Longitudinal Timepoints
ADNI	NC-long	372	1680	37	60	82	110	47	22	14	0	1.65 (1.47)	0.05-6.66	75.4 (6.1)	59.7 - 95	196 / 176	29.03	29.02	0.08	0.09
	AD-long	606	2730	55	86	197	147	53	35	21	12	1.5 (1.32)	0.07-6.48	75.4 (7.3)	55 - 92.9	257 / 349	23.94	23.53	3.95	4.24
AIBL (test)	NC-long	128	435	21	34	73						2.9 (1.4)	1.1-6.5	73 (7)	60.5 - 90.2	65 / 63	28.95	28.95	0.02	0.01
	AD-long	39	107	17	15	7						2.5 (1.1)	1.3-5.2	74.9 (7.6)	55 - 89.2	20 / 19	21.91	20.5	0.74	0.84

SUPPLEMENTARY TABLE 3
Description of the longitudinal ADNI training and AIBL test data. Intervals are in years.

Sample	Scanner	Tesla	Sequence parameters
LCBC	Avanto Siemens	1.5	TR: 2,400 ms, TE: 3.61 ms, TI: 1,000 ms, flip angle: 8°, slice thickness: 1.2 mm, FoV: 240 × 240 m, 160 slices, iPat = 2
	Avanto Siemens	1.5	TR: 2,400 ms, TE = 3.79 ms, TI = 1,000 ms, flip angle = 8, slice thickness: 1.2 mm, FoV: 240 × 240 mm, 160 slices
	Skyra Siemens	3.0	TR: 2,300 ms, TE: 2.98 ms, TI: 850 ms, flip angle: 8°, slice thickness: 1 mm, FoV: 256 × 256 mm, 176 slices
BETULA	Discovery GE	3.0	TR: 8.19 ms, TE: 3.2 ms, TI: 450 ms, flip angle: 12°, slice thickness: 1 mm, FOV 250 × 250 mm, 180 slices
NESDA	Phillips	3.0	TR: 9 ms; TE: 3.5 ms; slice thickness: 1 mm, FOV: 256 x 256 mm, 170 slices.

SUPPLEMENTARY TABLE 4
MRI parameters
FoV = field of view, iPat = in-plane acceleration, TE = echo time, TI = inversion time, TR = repetition time.

	nSNPs	
Score	**PRS-AD**	**PRS-AD^{noAPOE}**
Jansen	59	27
Kunkle	42	16
Lambert	33	13
Wightman	81	42

SUPPLEMENTARY TABLE 5
The number of SNPs used to construct each PRS score.

stage	struct	score	effect	beta	stderr	t	p	df
Braak II	Left Hippocampus	Jansen	age_interaction	-0.21	0.07	-2.92	**0.00387**	211
Braak II	Left Hippocampus	Lambert	age_interaction	-0.18	0.07	-2.58	**0.01062**	211
Braak II	Left Hippocampus	Kunkle	age_interaction	-0.18	0.07	-2.46	**0.01487**	211
Braak II	Left Hippocampus	Wightman	age_interaction	-0.22	0.07	-3.1	**0.00219**	211
Braak II	Right Hippocampus	Jansen	age_interaction	-0.17	0.07	-2.36	**0.01925**	211
Braak II	Right Hippocampus	Lambert	age_interaction	-0.14	0.07	-2.14	**0.03351**	211
Braak II	Right Hippocampus	Kunkle	age_interaction	-0.19	0.07	-2.74	**0.00676**	211
Braak II	Right Hippocampus	Wightman	age_interaction	-0.18	0.07	-2.63	**0.00928**	211
Braak I	Left entorhinal	Jansen	age_interaction	-0.06	0.08	-0.76	0.44868	211
Braak I	Left entorhinal	Lambert	age_interaction	-0.05	0.07	-0.66	0.50907	211
Braak I	Left entorhinal	Kunkle	age_interaction	0	0.07	-0.01	0.98997	211
Braak I	Left entorhinal	Wightman	age_interaction	-0.1	0.07	-1.33	0.18490	211
Braak I	Right entorhinal	Jansen	age_interaction	-0.1	0.07	-1.37	0.17311	211
Braak I	Right entorhinal	Lambert	age_interaction	-0.05	0.07	-0.75	0.45180	211
Braak I	Right entorhinal	Kunkle	age_interaction	-0.06	0.07	-0.82	0.41544	211
Braak I	Right entorhinal	Wightman	age_interaction	-0.16	0.07	-2.21	**0.02834**	211
Braak III (subcortical)	Left Amygdala	Jansen	age_interaction	-0.13	0.07	-1.88	0.06098	211
Braak III (subcortical)	Left Amygdala	Lambert	age_interaction	-0.1	0.06	-1.54	0.12478	211
Braak III (subcortical)	Left Amygdala	Kunkle	age_interaction	-0.11	0.07	-1.57	0.11864	211
Braak III (subcortical)	Left Amygdala	Wightman	age_interaction	-0.13	0.07	-1.89	0.06026	211
Braak III (subcortical)	Right Amygdala	Jansen	age_interaction	-0.18	0.07	-2.46	**0.01459**	211
Braak III (subcortical)	Right Amygdala	Lambert	age_interaction	-0.11	0.07	-1.68	0.09410	211
Braak III (subcortical)	Right Amygdala	Kunkle	age_interaction	-0.17	0.07	-2.46	**0.01485**	211
Braak III (subcortical)	Right Amygdala	Wightman	age_interaction	-0.19	0.07	-2.7	**0.00743**	211
Braak III (cortical)	Left temporal	Jansen	age_interaction	-0.14	0.07	-1.89	0.06079	211
Braak III (cortical)	Left temporal	Lambert	age_interaction	-0.07	0.07	-1.07	0.28520	211
Braak III (cortical)	Left temporal	Kunkle	age_interaction	-0.12	0.07	-1.72	0.08707	211
Braak III (cortical)	Left temporal	Wightman	age_interaction	-0.16	0.07	-2.21	**0.02817**	211
Braak III (cortical)	Right temporal	Jansen	age_interaction	-0.13	0.07	-1.78	0.07726	211
Braak III (cortical)	Right temporal	Lambert	age_interaction	-0.12	0.07	-1.84	0.06663	211
Braak III (cortical)	Right temporal	Kunkle	age_interaction	-0.13	0.07	-1.83	0.06887	211
Braak III (cortical)	Right temporal	Wightman	age_interaction	-0.16	0.07	-2.37	**0.01893**	211

SUPPLEMENTARY TABLE 6
Results from alternative analyses dependent on power across the full age-range (30-89 years; N = 229). Linear PRS-AD * mean age interactions upon age-relative change that were significant at $p < .05$ (uncorrected) are in bold. None of the tests survived FDR-correction across the 32 tests in this analysis.

stage	struct	score	effect	beta	stderr	t	p	df
-	$PC1^{relChange}$	Jansen	age_interaction	0.23	0.07	3.21	* **0.00154**	211
-	$PC1^{relChange}$	AD	age_interaction	0.19	0.07	2.74	* **0.00664**	211
-	$PC1^{relChange}$	Kunkle	age_interaction	0.22	0.07	3.07	* **0.00243**	211
-	$PC1^{relChange}$	Wightman	age_interaction	0.22	0.07	3.2	* **0.00159**	211

SUPPLEMENTARY TABLE 7
Results from alternative analyses dependent upon power across the full age-range (30-89 years; N = 229). FDR-corrected significant linear PRS-AD * mean age interactions (in bold denoted with *) upon $PC1^{relChange}$ were found for all four GWAS-derived scores, indicative of steeper effects of PRS-AD upon multivariate age-relative change across AD-sensitive features in older ages. Note that hippocampal and amygdala volumes were not included in $PC1^{relChange}$ to ensure these did not drive the multivariate effect (see Fig. 4A). FDR-correction was applied across all 4 tests performed in this analysis.

		APOE genotypes					
Sample	N Genetic	e2 / e2	e2 / e3	e2 / e4	e3 / e3	e3 / e4	e4 / e4
LCBC	229	26	-	8	123	61	11
BETULA	175*	-	18	5	102	48	1
NESDA	118*	-	-	-	73	33	3

SUPPLEMENTARY TABLE 8
APOE genotypes per sample. *Because *APOE* alleles were derived using imputed genotypes (1000 Genomes Project Phase 3; hg19 build), 9 individuals in NESDA, and 1 individual in BETULA had ambiguous genotypes due to not having direct access to the two *APOE* indexing SNPs.

SUPPLEMENTARY NOTE 1

LCBC Samples

The main discovery sample consisted of magnetic resonance imaging (MRI) data collected across 5 projects at the Center for Lifespan Changes in Brain and Cognition (LCBC; Department of Psychology, University of Oslo). For the brain change analysis, to optimize individual-specific change estimates for all individuals, we used as many longitudinal observations as we could gather. Notably, the samples described below were all used in the brain change analysis, but many of these individuals had no genetic observations, and thus were not included in the PRS-AD association tests (see below). Brain change data over the following projects were used, and the number of individuals included in the genetic analysis [max N = 229] is also given per project. For the memory change analysis, to optimize individual-specific memory slopes for all we also used as many usable longitudinal memory observations as we could. However, we discarded all data for individuals involved in memory training projects with on-off designs over time, deemed likely to induce non-linear effects upon the individual-specific memory slopes.

Cognition and Plasticity Through the Lifespan (MemP):

For the brain change analysis, **537** scans of 185 individuals (mean age = 59.5 ± 13.0, age-range = 30.6 – 89.4, females = 110, 2–7 timepoints [TP's]), collected on 3 different scanners were used (N 2TP's = 57, 3TP's = 93, 4TP's = 33, 5TP's = 1, 7TP's = 1). The number of scans used here collected on each scanner was 489 (1.5T Avanto), 46 (3.0T Skyra) and 2 (3.0T Prisma), respectively.
*For the genetic analysis, **81** individuals originated from this project.*
*To estimate individual-specific memory slopes, **522** observations from **185** individuals originated from this project,*

This is an ongoing longitudinal study where cognitively healthy adults have undergone MRI scanning and neuropsychological evaluation. The study consists of four main recruitment waves (though note that the number of longitudinal timepoints ranges from 2-7). New participants were recruited at waves 1, 3 and 4. Participants were scanned up to 11.1 years after the initial scan. The interval between waves was approximately 3.5, 4.4 and, 1.7 years, respectively. Data acquisition took place between 2006 and 2023 at the center for LCBC. For more details see [1,2]. Volunteers were initially recruited by newspaper advertisements and later contacted by mail for follow-ups. MRI sequences were acquired across three scanners (1.5T Avanto, 3.0T Skyra, 3.0T Prisma).

Constructive Memory (MemC):

For the brain change analysis, **85** scans of **42** individuals (mean age = 53.9 ± 13.4, age-range = 30.5 – 80.8, females = 24, 2-3 timepoints [TP's]), collected across two scanners were used (N 2TP's = 41, 3TP's = 1). The number of scans here collected on each scanner was 84 (3.0T Skyra) and 1 (3.0T Prisma), respectively.
*For the genetic analysis, **30** individuals originated from this project.*
*To estimate individual-specific memory slopes, **80** observations from **41** individuals originated from this project.*

This project is a cross-sectional study where cognitively healthy adults underwent an fMRI source-item memory task. The protocol also included MRI scanning and neuropsychological evaluation. The sample was collected at the center for LCBC. The project is nested within the longitudinal Cognition and Plasticity through the Lifespan project. Data acquisition took place between 2013 and 2015. MRI sequences were acquired with a 3.0T Skyra scanner. See [3,4] for more details.

Neurocognitive Plasticity (NCP):

For the brain change analysis, **637** scans of **130** individuals (mean age = 70.5 ± 12.3, age-range = 30.1 – 84.0, females = 79; 2-3 timepoints [TP's]), collected on a single scanner (3.0T Skyra) were used (N 2TP's = 17, 3TP's = 11, 4TP's = 12, 5TP's = 24, 6TP's = 60, 7TP's = 6).
*For the genetic analysis, **117** individuals originated from this project.*
*To estimate individual-specific memory slopes, **0** observations originated from this project (due to the complex on-off memory training design over time likely to induce non-linear effects upon individual-specific memory slopes; see below)*

This study consists of an experimental project of memory training with the "method of loci", employing an on-off training design over time. The study includes two groups of participants (young and old) that underwent an ABAB design where a batch in each group started with a resting condition, and the other started with memory training (after a baseline test). The study also includes an active group without memory training. The participants were initially scanned up to 6 times, five of them before/after a block of training while the sixth time point consisted of a follow-up ≈ 2 years after the intervention (note that several participants also have a seventh timepoint). Participants were recruited through newspaper and web page adverts and were screened with a health interview. Participants were required to be either young or older (in or around their 20s or 70s, respectively) healthy adults. Data acquisition took place between 2013 and 2018. MRI sequences were acquired with a 3.0T Skyra scanner. See [5,6] for more details.

Method of Loci (Loci):
For the brain change analysis, **112** scans of **41** individuals (mean age = 63.2 ± 9.1, age-range = 41.9 – 82.6, females = 22; 2-5 timepoints [TP's]), collected across two scanners were used (N 2TP's = 13, 3TP's = 27, 5TP's = 1). The number of scans used here collected on each scanner was 110 (1.5T Avanto) and 2 (3.0T Skyra), respectively.
*For the genetic analysis, **1** individual originated from this project (individual with 5 timepoints; see below).*
*To estimate individual-specific memory slopes, **111** observations from **41** individuals originated from this project (due to the simple pre-post memory training design [see below], individual-specific slopes are likely to be linear; note also that as only 1 individual here had genetic data, results of the longitudinal memory change analyses in the main paper – requiring individuals with both genetic and memory data – are unaffected by including this project, except insofar as the additional longitudinal observations may boost power to estimate individual-specific memory slopes for all; see below)*

This study consists of an eight-week simple pre-post memory training experiment focused on improving verbal recall memory by implementing the mnemonic technique "method of loci". Participants were scanned three times as part of the project: pre and post-training and a follow-up after 5 years. The number of timepoints included here therefore typically ranges from 2-3, though we note one participant originating from this project and included in the genetic analyses had 5 timepoints, as they were assimilated into (or later found to be a duplicate ID) of a later LCBC project (MemC). The full project included both healthy controls and memory clinic patients. Only data from healthy controls was used here. Healthy volunteers were recruited through a local newspaper ad, screened by a structured interview, and randomly assigned to either an intervention group or a control group serving as passive controls. Cognitive assessments and the memory training program were conducted at the center for LCBC. Data acquisition for waves 1 and 2 took place between 2007 and 2008 while the third wave was acquired in 2013. MRI sequences were acquired with a 1.5T Avanto scanner. See [7,8] for more details.

Set to Change (S2C):
For the brain change analysis, **59** scans of **22** individuals (mean age = 44.6 ± 16.8, age-range = 30.2 – 79.0, females = 13), collected on a single scanner (3.0T Prisma) were used (N 2TP's = 7, 3TP's = 15).
*For the genetic analysis, **0** individuals originated from this project (individuals were twins; see below).*
*To estimate individual-specific memory slopes, **0** observations originated from this project (due to the complex on-off memory training design over time likely to induce non-linear effects upon individual-specific memory slopes; see below)*

This is the first LCBC twin project, where twins were invited to participate, as recruited through either the Norwegian twin registry or via ads on social media platforms. The project consists of a targeted experimental approach to test differences in neurocognitive plasticity by training of younger and older adult mono- (MZ) and dizygotic (DZ) twins. The project employs a memory intervention using navigation training while cycling in virtual reality through a bespoke virtual city. Twins are assessed with brain MRI, and cognitive measures at multiple time points across 2.5 years pre- and post- a 10 week intervention in a AB/BA crossover design. Data acquisition began in 2019 and is ongoing. MRI sequences were acquired with a 3.0T Prisma scanner. Of the 22 individuals included in the brain change analysis here, 14 were MZ whereas 8 were DZ twins.

Common information:
Common exclusion criteria across all LCBC projects are outlined in the main paper. Written informed consent was obtained from all participants, and all studies were approved by the Regional Ethical Committee of South Norway.

SUPPLEMENTARY NOTE 2

Dataset access

Requests for access to the raw data can be directed to the Principal Investigators of the contributing studies: LCBC (Anders M. Fjell; anders.m.fjell@psykologi.uio.no), BETULA (Lars Nyberg; lars.nyberg@umu.se), NESDA (Brenda Penninx; b.penninx@amsterdamumc.nl). ADNI and AIBL (https://adni.loni.usc.edu/data-samples/access-data/).

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Acknowledgements

The Australian Imaging, Biomarker and Lifestyle Flagship Study of Ageing (AIBL)

Colin L. Masters[25], Christopher C. Rowe[25, 26], Jurgen Fripp[29], Ralph Martins[28], David Ames[31], Christopher Fowler[25], Stephanie Rainey-Smith[28], Vincent Dore[30], Paul Maruff[34], Kevin Taddei[28], Simon Laws[27], Ashley I. Bush[25], Jo Robertson[25], Brett Trounson[25], Qiao-Xin Li[25], Alyce Russell[27], Madeline Peretti[27], Lidija Milicic[27], Lucy Lim[28], Mark Rodrigues[28], Tania Taddei[28], Eugene Hone[28], Florence Lim[28], Shane Fernandez[28], Steve Pedrini[28], James Doecke[29], Pierrick Bourgeat[29], Simon Gibson[29], Hugo Leroux[29], David Hanson[29], Ping Zhang[30], Elizabeth Cyarto[31], Brendan Silbert[32], Belinda Brown[33], Harmid Sohrabi[33].

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