Supplementary Table 1. Summary of the 73 studies selected from the reviewing process.

Reference	Population (N)	MRI Sequences/Techniques	Main outcome measures	Main findings
Clinical trials				
1. Egan et al., 2019[1]	1454 mild to moderate AD. Randomized, double-blind, placebo-controlled trial (treatment: verubecestat)	T1-weighted sequences. Techniques: Hippocampal volumetry.	Changes in CDR scores, cognition and daily life functioning. Among the secondary outcomes: hippocampal volumes.	The trial was terminated for futility after 104 weeks; verubecestat did not improve clinical ratings and some measures suggested worse cognitive functioning in patients undertaking medication. Hippocampal volumes were lower at the end of trial compared to baseline in both groups.
2. Cheng et al., 2019[2]	11 mild to moderate AD 11 HC. Open-label study (treatment: donepezil).	T1-weighted and rs-fMRI sequences. Technique: Analysis with ROIs.	Therapeutic mechanism of donepezil on brain functioning (ReHo) and cognitive performance.	After treatment with donepezil, AD patients showed improvements in ADAS-cog and MMSE, and decreased ReHo in the right gyrus rectus, right precentral gyrus and left superior temporal gyrus compared with HC.
3. De Jong et al., 2019[3]	58 mild-to-moderate AD. 6-months randomized, double-blind, placebo-controlled study (treatment: Nilvadipine)	T1-weighted, FLAIR, SWI and ASL sequences. Techniques: whole-brain CBF, GM volumetry, CT of regions of interest.	Effect of Nilvadipine on whole-brain and ROI CBF.	With Nilvadipine treatment, systolic blood pressure was lower, GM CBF remained stable in the whole-brain and increased in the hippocampus.

4.	Kehoe et al., 2018[4]	228 mild to moderate AD. Randomized, double-blind, placebo-controlled trial (treatment: RADAR).	T1-weighted sequences at baseline and after 12 months. Techniques: Semi-automated methods to derive brain structure volumes from single time-point MRI and rates of atrophy from serial MRI.	Whole brain atrophy, WMH volume, CBF, cognitive performance and quality of life.	/
5.	Boespflug et al., 2018[5]	16 MCI. Randomized, double-blind, placebo-controlled trial (treatment: blueberry supplementation).	T1-weighted and task-based fMRI sequences. Technique: voxel-wise task-related activity.	Effect of blueberry supplementation on fMRI activity and working memory.	Compared to placebo, blueberry-treated MCI exhibited increased fMRI activation in the left pre-central gyrus, left middle frontal gyrus, and left inferior parietal lobe during working memory load conditions. No clear evidence of working memory improvement due to blueberry supply.
6.	Huhn et al., 2018[6]	60 elderly participants. Double-blind, randomized controlled trial (treatment: Resveratrol).	T1-weighted, rs-fMRI and DWI sequences. Techniques: seed-based functional connectivity and WM microstructure of hippocampal subfields.	Memory performance, blood-based biomarkers, hippocampal connectivity and microstructure, glucose metabolism.	Negative findings.
7.	Feng et al., 2018[7]	25 aMCI. Single blind, prospective study (treatment: SD or MD cognitive training).	T1-weighted and rs-fMRI sequences. Technique: GM volumetry and voxel-wise ReHo analysis.	GM atrophy, ReHo and cognitive changes.	MD compared to SD group showed: larger GM volumes of the middle frontal gyrus, superior parietal lobule, and inferior temporal gyrus; and higher ReHo in the putamen, calcarine and inferior temporal gyrus. In the MD group only, the GM integrity

				of the precuneus was positively related to language scores, and that of the amygdala, fusiform gyrus, and hippocampus had a positive relationship with delayed memory performances.
8. Zhang et al., 2019[8]	17 aMCI. Open label study. (treatment: computerized multi-domain cognitive training)	T1-weighted sequences. Technique: VBM.	Post- treatment changes in GM volumes and cognitive performances.	In all sample, GM volume increased in the right angular gyrus and other parietal subareas near the intraparietal sulcus; no significant changes in neuropsychological scores.
9. Lin et al., 2018[9]	600-800 MCI. Multicentre, randomized, single-blind prospective clinical trial (treatment: computerized cognitive training).	T1-weighted and rs-fMRI sequences. Technique: /	Effects of training on the conversion rate of MCI to AD and brain activity within 36 months of follow-up.	/
Predementia stages				
10. Kang et al., 2018[10]	34 aMCI 38 HC	T1-weighted and rs-fMRI sequences. Techniques: seed-based functional connectivity analysis.	Intra- and inter-regional ReHo.	Significantly higher ReHo in the left putamen and lower ReHo in the left inferior temporal gyrus in aMCI patients *vs* HC.
11. Qian et al., 2018[11]	59 MCI (21 sMCI; 38 pMCI)	T1 weighted sequence. Techniques: volumetry analysis.	Cognitive scores; GM and WM volumes.	pMCI patients *vs* sMCI showed: lower scores in memory, language, executive and visual spatial domains, and reduced volume

				of the left thalamus, bilateral hippocampus, posterior and central corpus callosum.
12. Luo et al., 2018[12]	49 HC 32 SD-aMCI 32 MD-aMCI	T1-weighted and rs-fMRI sequences. Techniques: ReHo voxel-wise analysis, automatic segmentation of WML.	ReHo, quantitative WML, and CSF biomarkers.	SD-aMCI *vs* HC showed decreased ReHo in medial temporal gyrus and these changes were related to the CSF Aβ levels. MD-aMCI *vs* HC and SD-aMCI showed decreased ReHo in precuneus, lingual gyrus and postcentral gyrus.
13. Farrar et al., 2018[13]	31 MCI (15 with high executive function performance; 16 with low executive function performance)	T1-weighted and DTI sequences. Techniques: WM connectome and graph analysis.	Alterations of WM connectivity within the brain structural connectome.	High executive ability was associated with greater network size, density and clustering coefficient, greater fractional anisotropy in the inferior and superior longitudinal fasciculi bilaterally.
14. Heinrich et al., 2018[14]	75 MCI	T1-weighted, T2 FLAIR, and proton density/T2 sequences. Techniques: visual rating.	Olfactory performances; Hippocampal atrophy; periventricular and deep WM lesions and their relationship.	In all patients, significant relationships between lower olfaction with older age, worse cognitive performance, hippocampal atrophy, periventricular and deep WM lesions.
15. Shu et al., 2018[15]	36 SCD 51 HC	DTI sequence. Techniques: WM connectome and graph analysis.	Alterations of WM connectivity within the brain structural connectome.	SCD *vs* HC showed reduced global and local efficiency and reduced regional efficiency in bilateral PFC and left thalamus; in SCD

				diminished nodal strength was related with impaired memory performance.
16. Zhang et al., 2018[16]	26 early MCI 19 late MCI 23 HC	Rs-fMRI sequences. Techniques: Seed-based functional connectivity.	Intra- and -inter-network dysfunctions.	In MCI patients, aberrant intra- and inter-network dysfunctions in DMN and extra-DMNs.
17. Esposito et al., 2018[17]	27 young HC 26 elderly HC 17 MCI	T1-weighted and rs-fMRI sequences. Techniques: seed-based functional connectivity; VBM.	Functional connectivity alterations between DMN and Dorsal Attention Network, and voxel-wise GM alterations.	Reduced anticorrelated activity between DMN and Dorsal Attention Network in MCI compared to HC.
18. Liu et al., 2018[18]	16 MCI with depressive symptoms; 18 MCI without depressive symptoms	T1-weighted and rs-fMRI sequences. Techniques: voxel-based functional connectivity analysis.	ALFF and functional connectivity density.	In depressed MCI *vs* non-depressed MCI: higher ALFF in the left middle PFC and lower in the right precentral gyrus; higher functional connectivity density values in the left medial temporal gyrus.
19. Melrose et al., 2018[19]	25 aMCI 19 HC	T1-weighted and task-based fMRI sequences. Techniques: FEAT and FLAME approaches.	Task-related alterations.	Compared to HC, MCI showed: hypoactivation of right frontoparietal regions and hyperactivation of left prefrontal cortex, coupled with attenuation of DMN, during the working memory task; hypoactivation of parietal regions, coupled with attenuation of anterior DMN and increased deactivation of posterior DMN during the Reasoning task.

Risk and protective factors				
20. Cherbuin et al., 2019[20]	461 HC	T1-weighted sequence. Technique: ROI analysis.	Estimation of the risk to develop cognitive decline and of the effectiveness of the ANU-ADRI on total and regional brain volumes.	In all sample, higher risk estimates with the ANU-ADRI were associated with lower cortical GM (particularly in the DMN regions).
21. Mosconi et al., 2018[21]	116 HC	T1-weighted sequence. Technique: CT of ROIs.	CT changes in relation with lifestyle, vascular risk, and cognition.	The adherence to a Mediterranean diet and insulin sensitivity explained CT in key brain regions for AD; EC thickness predicted memory performance.
22. Rizvi et al., 2018[22]	519 HC	T1-weighted and T2-FLAIR sequences. Techniques: conditional process analysis modeling techniques.	Effect of WMH on global cognition mediated by cortical thinning.	In all sample, increased total WMH volume was associated with poorer global cognition and memory. Global CT and medial temporal lobe thickness mediate this relationship.
23. Ding et al., 2018[23]	200 HC 26 MCI	T1-weighed, T2-FLAIR sequences. Techniques: Visual rating.	Prediction of the risk of cognitive decline due to WMH.	In MCI, increasing severity of WMH and the presence of lacunes at baseline were independent predictors of incident cognitive decline.
24. Chen et al., 2018[24]	57 HC 50 AD 53 aMCI 40 dysexecutive MCI	T1-weighted and T2- FLAIR sequences. Techniques: visual ratings.	The interactive effects of WMH and medial temporal lobe atrophy on longitudinal clinical decline.	In all subjects, periventricular WMH were correlated with medial temporal lobe atrophy, and both were

				independent predictors of clinical decline.
25. Smith et al., 2018[25]	Hypertensive patients, 22 MCI and 45 SCD	T1-weighted, FLAIR, dual-echo T2-weighted and proton density-weighted, and SWI sequences. Technique: automatic WM lesion segmentation.	Cognitive performances; WM lesions; Aβ deposition.	Relationship between worse episodic memory performance in MCI and SCD hypertensive participants and higher Aβ deposition.
26. Zhang et al., 2019[26]	203 MCI	T1-weighted, T2-weighted and FLAIR sequences. Techniques: visual rating.	Impact of periventricular WMH and serum cystatin C on MCI.	Older age and hypertension were predictors of severe periventricular WMH, while age alone predicted deep WMH. Periventricular severity was independently associated with MCI condition, executive function and processing speed. Deep WMH had no significant effect on cognitive function. Cystatin C only affected the overall cognitive level, and the relationship with WMH severity was not significant.
27. Cantero et al., 2018[27]	57 subjects (HC=20; positive $A\beta_{1-42}$=19; positive p-tau=18)	T1-weighted sequence. Techniques: CT; VBM; Graph-theory.	CSF in relation to cerebral integrity, at local and network levels. Differences in CT, WM volume, and properties of structural networks.	Subjects with abnormal $A\beta_{1-42}$ had cortical thinning of several AD-brain regions (such as precuneus, PCC, hippocampus and parahippocampal gyrus) at baseline. These subjects

				presented with WM atrophy of the anterior and posterior cingulate bundle and more segregated cortical networks, with the Aβ positive group showing heightened isolation of cingulate and temporal cortices.
28. Falcon et al., 2018[28]	Two independent cohorts: 1) 22 HC + 9 asymptomatic subjects with low CSF-$A\beta_{42}$ levels. 2) 22 HC + 17 asymptomatic subjects with low CSF-$A\beta_{42}$ levels. from ADNI dataset.	T1-weighted sequence. Techniques: VBM.	Estimation of rates of GM volume changes in association with baseline core CSF AD-like biomarkers and determination of whether these differences are sample dependent.	In the whole sample, associations were observed between CSF levels and GM atrophy rates. Specifically: $A\beta_{42}$ and medial and orbital frontal, precuneus, cingulate, medial temporal regions and cerebellum; p-tau and left hippocampus, parahippocampus and striatal nuclei; p-tau/$A\beta_{42}$ and ventral and medial temporal areas.
29. Voevodskaya et al., 2018[29]	299 HC with and without amyloid pathology	T1-weighted sequence. Technique: CT; Graph theory.	CT; Global and local network changes.	Compared to Aβ-negative, Aβ-positive group exhibited: an altered global network organization, including decreased global efficiency and modularity; fewer and more disorganized modules as well as a loss of hubs at the local level.

30. Foster et al., 2018[30]	181 HC	Task-based fMRI (spatial distance-judgment task) sequence.	Relationship between increasing Aβ burden and alterations in functional activation during high demanding cognitive tasks.	During the task, individuals with slightly elevated Aβ burden exhibited hyperactivation, whereas individuals with higher Aβ burden showed hypoactivation. In high-Aβ individuals, Aβ load moderated the effect of functional activation on behavioural task performance: in lower-elevation, greater deactivation was associated with better accuracy, in higher-elevation, greater deactivation was associated with poorer accuracy during the task.
31. Tardif et al., 2018[31]	217 HC	T1-weighted, T2-FLAIR sequences. Technique: automated segmentation; T1/T2 FLAIR intensity ratio.	Structural integrity of components of the hippocampal circuit, (including subfields and extra-hippocampal WM structure) in relation to CSF levels.	Subjects with abnormal CSF $A\beta_{42}$ and tau levels exhibited lower subiculum volume, lower fornix microstructural integrity, and lower cognitive performances than individuals who showed only reduction in CSF $A\beta_{42}$.
32. Mecca et al., 2018[32]	3 groups of HC with a first-degree AD positive family history and different APOE status (N=45):	T1-weighted sequence. Techniques: ROI modelling-based approach.	Association between positive family history and APOE genotype to Aβ burden in brain regions preferentially	APOEε4ε4 participants demonstrated significantly higher cortical Aβ burden than APOEε4ε3 and

	1) APOE ε4ε4 (n=15) 2) APOE ε3ε4 (n=15) 3) APOE ε3ε3 (n=15)		affected by AD and the association between Aβ burden, GM volume and episodic memory performance.	APOEε3ε3; In these subjects, Aβ burden was inversely associated with GM mean volume but not with episodic memory performance.
33. Li et al., 2018[33]	Two independent datasets: 1) 360 HC 2) 323 HC	T1-weighted sequence. Techniques: VBM.	Association between the effect of AD polygenic risk score on the entire brain and the influence of this score on cognitive functioning.	In all subjects, an elevated AD polygenic risk score was associated with a smaller precuneal volume. No correlation was observed between polygenic risk score and any cognitive measure.
34. Lee et al., 2019[34]	34 HC 21 AD 32 MCI	T1-weighted and rs-fMRI sequences. Techniques: Graph analysis.	Cognitive reserve estimation; relationship between network topological characteristics and cognitive reserve.	Cognitive reserve marker was associated with education and occupation complexity; in all subjects it was correlated with global efficiency of the entire network, and nodal clustering coefficient; in AD and MCI it was related with the local efficiency of the right middle-temporal pole; in HC it was related with the efficiency of the right precentral gyrus.
35. Wolf et al., 2019[35]	ADNI-2 cohort: 158 HC 299 MCI	T1-weighted sequence. Techniques: VBM.	Relationship between resilience mechanisms and pathology.	Hippocampus is proposed as a dynamic resilience factor.
36. Duncan et al., 2018[36]	34 monolingual MCI, 34 multilingual MCI,	T1-weighted sequences. Techniques: CT and VBM.	CT and tissue density.	In areas related to language and cognitive control, both

	13 monolingual AD, 13 multilingual AD			multilingual MCI and AD patients had thicker cortex than the monolinguals. Multilingual patients showed a correlation between CT in regions related to language and cognitive control and their performance on episodic memory.
37. Ding et al., 2018[37]	26 HC 55 aMCI	DTI and T1-weighted sequences. Techniques: TBSS and ROI-based analyses.	WM integrity; cardiorespiratory fitness; cognitive performance.	In all subjects, global WM fiber integrity was associated with high cardiorespiratory fitness levels and executive function performance.
New brain regions of interest				
38. Su et al., 2018[38]	13 AD 22 MCI 27 HC	T1-weighted and T2-weighted sequences. Techniques: VBM; manual segmentation of hippocampus subfields.	Hippocampal subfields thickness.	AD and MCI compared to HC showed: CA1 and subiculum thinning; AD compared to HC: SRLM thinning. In all patients CA1, CA3/dentate gyrus areas, and SRLM integrity were related with clinical and cognitive measures.
39. Butler et al., 2018[39]	18 future AD (MCI which became AD 2.8 years later) 40 AD/MCI 89 HC	T1-weighted sequences. Technique: Manual tracing.	Septal nucleus volumes.	HC who were destined to develop AD had enlarged septal nuclei as compared to both HC and patients with current MCI/AD.

40. Matsuoka et al., 2018[40]	63 AD 33 MCI 24 HC	T1-weighted sequences. Techniques: VBM.	Pineal gland and pineal parenchymal volume and correlation with cognitive testing.	Mean pineal gland volume in patients with AD was significantly smaller than in HC. The mean pineal parenchymal volume in AD was significantly smaller than in MCI and HC. In all subjects, MMSE score and total intracranial volume were significant independent predictors of both pineal gland volume and pineal parenchymal volume.
41. Zidan et al., 2019[41]	26 AD 20 aMCI 20 HC	T1-weighted, T2-weighted FLAIR, T2-weighted and DTI. Techniques: morphometric analysis.	Thalamic brain volume loss.	AD showed lower global GM, volumes of thalamus and hippocampus compared to aMCI and HC. aMCI showed lower hippocampal volume than HC.
42. Persson et al., 2018[42]	36 SCD 60 MCI 120 AD 5 VD	T1-weighted sequences. Technique: automated segmentation.	Caudate nucleus volumes.	Compared to MCI and SCD, AD patients had larger caudate nucleus volumes and smaller hippocampal volumes.
43. Zhao et al., 2018[43]	209 HC 208 early (stage) MCI 183 advanced MCI 121 AD	Rs-fMRI sequences. Techniques: sparse representation of concatenated multiple brain signals.	Functional connectivity of the executive control and frontoparietal networks.	AD *vs* HC: increased functional connectivity of the superior and middle frontal gyrus in the executive control network and decreased of the

				superior parietal gyrus in the frontoparietal network.
44. Yu et al., 2019[44]	30 AD 14 aMCI 18 HC	Rs-fMRI sequences. Techniques: Granger causality analysis.	Identification of neuroimaging biomarkers with high sensitivity to aMCI.	Differences in DMN, SN and ECN were found in the three groups; aMCI patients showed inhibitory activity within the DMN connectivity from the PCC to the hippocampal formation, and from the thalamus to the PCC, as well as excitatory activity within the SN connectivity from the dorsal anterior cingulate cortex to the striatum, from the ECN to the DMN, and from the SN to the ECN.
MRI and cognition				
45. Arighi et al., 2018[45]	14 MCI	T1-weighted sequences. Techniques: VBM.	Differences in FCSRT-word and FCSRT-picture version performances and their relationship with GM integrity.	In MCI patients, performances at FCSRT-picture were related with atrophy in areas involved in visual stimuli processing, while performances at FCSRT-word were related to hippocampal atrophy.
46. Slachevsky et al., 2018[46]	35 AD 34 HC	T1-weighted sequences. Techniques: VBM.	Relationship between the integrity of GM and performances at word and picture versions of FCSRT.	AD patients performed worse in both versions of the test than HC, and both groups performed higher in the

				picture version; significant correlations between free recall of either version with atrophy of the temporal pole and hippocampal regions.
47. Petok et al., 2018[47]	33 asymptomatic autosomal-dominant mutation carriers, 11 non carrier kin	T1-weighted sequence. Techniques: CT.	Assessment of generalization ability, learning and retention; relationship between memory generalization and cortical volumes.	Preclinical mutation carriers made significantly more errors during generalization than non-carrier kin, and this impairment was correlated to the hippocampal volume loss.
48. Delazer et al., 2019[48]	13 AD 3 MCI	3D T1-weighted, FLAIR and T2-weighted sequences. Techniques: morphometric analysis.	Relationship between hippocampal degeneration and arithmetic fact retrieval.	Both AD and MCI showed marked bilateral hippocampal atrophy and deficits in episodic memory; 13 out of 16 patients showed intact arithmetic facts retrieval.
49. Verfaillie et al., 2018[49]	233 SCD	T1-weighted sequences. Techniques: CT.	Associations between regional CT and rate of decline over time in memory, attention, executive function, and language.	In SCD, no association was found between CT and baseline cognition, but a faster subsequent rate of memory loss was associated with thinner cortex of the frontal, temporal and occipital cortices.
50. Van der Stigchel et al., 2018[50]	41 AD 38 aMCI 7 SCD	T1-weighted sequences. Techniques: GM semiautomated segmentation.	Correlation of GM volumes with performance on the pentagon copying test.	In all subjects, total score and subscores of the pentagon copying test was associated

				with parietal and subparietal GM volumes.
51. Matsuoka et al., 2018[51]	30 AD 10 MCI 18 HC	T1-weighted sequences. Techniques: VBM	Relationship between cortical volumes and performances at the executive functions.	In all subjects: EXIT25 score correlated inversely with the regional GM volume of the left lateral frontal lobe; CLOX1 score correlated positively with the regional GM volume of the right orbitofrontal cortex and the left supramarginal gyrus. FAB score correlated positively with the regional GM volume of the right precentral gyrus. The bilateral lateral frontal gyri were identified as common brain regions that showed association with all executive tests.
52. Brown et al., 2019[52]	32 HC	T1-weighted, T2-weighted, FLAIR, task-based fMRI (visual working memory paradigm), and DTI. Techniques: linear regression models with fractional anisotropy and functional activation/deactivation means within DMN and ECN.	Contribution of executive function performance to functional and structural profiles within the ECN and DMN longitudinally.	Low DMN deactivation, high ECN activation and WMH burden were the main predictors of executive function scores at baseline; poor DMN and ECN WM microstructure and higher AD pathology predicted greater annual decline in executive function scores.

53. Fernaeus et al., 2018[53]	30 HC 45 AD 7 NOS 4 VD 39 MCI 9 SCD 3 FTD 3 other diagnoses	T1-weighted and T2-weighted sequences. Technique: WMH visual rating; media temporal lobe manual tracing.	Discriminative ability of WAIS-R Similarities in distinguishing AD, MCI and HC. Relationship between WAIS-R Similarities scores, WMHs and temporal lobe volumes.	Semiautomatic lexical access and conceptual elaboration scores discriminated between MCI, AD, and HC. In all subjects, high levels of periventricular WMHs predicted factor scores of direct lexical access but not those of conceptual elaboration, which were predicted only by medial and lateral temporal lobe volumes.
54. Peters-Founshtein et al., 2018[54]	20 AD 20 MCI 20 HC	T1-weighted and task-based fMRI sequences. Techniques: group-level random-effects general linear model analysis.	Mental orientation task performance in the diagnosis of MCI, AD and healthy aging compared to standard tests (MMSE, ACE-R).	Compared to standard tests, mental orientation task had a high discrimination ability in distinguishing AD, MCI, and HC. This task preferentially recruits brain regions exhibiting early AD-related atrophy.
55. Yamashita et al., 2019[55]	25 AD (divided in good and poor oriented in time) 10 HC	T1-weighted and rs-fMRI sequences. Techniques: Seed-based functional connectivity.	PCC functional connectivity changes in relation to impairment of orientation in time.	Compared to the other groups, AD patients with poor orientation in time showed reduced PCC connectivity with right dorsal frontal lobe, lateral parietal and lateral temporal lobe; compared to AD with a good orientation in time, AD with poor orientation showed

				reduced PCC connectivity with right posterior middle temporal gyrus.
56. McLachlan et al., 2018[56]	104 patients with probable AD (47 psychotic and 57 non-psychotic).	T1-weighted sequences. Techniques: CT; GM volumetry; ROI analysis.	CT and volumetric measures in visuo-perceptual and frontal networks associated to psychotic symptoms.	Psychosis subtypes are associated to reduced left parahippocampal volume and ventral visual stream.
57. Guo et al., 2018[57]	21 non-depressed AD 21 depressed AD	T1-weighted and rs-fMRI sequences. Techniques: seed-based functional connectivity.	Changes in functional connectivity between the amygdala and frontal regions and their association with depressive symptoms.	Compared to non-depressed AD patients, depressed patients showed: increased functional connectivity between amygdala and orbitofrontal cortex; decreased functional connectivity between amygdala and medial PFC and inferior frontal gyrus.
58. Wang et al., 2019[58]	70 aMCI 28 AD	Rs-fMRI sequences. Techniques: multivariate pattern analysis.	Neural circuits involved in neuropsychiatric symptoms.	Functional connectivity of fronto-limbic circuit was linked to neuropsychiatric symptoms and AD pathology.
59. Ren et al., 2018[59]	55 aMCI	Rs-fMRI and DTI sequences. Techniques: Seed-based functional and structural connectivity.	Relationship between functional and structural connectivity and locus of control.	Functional and structural connectivity between the medial prefrontal cortex and amygdala significantly correlated with external locus of control.
Novel biomarkers				

60. Maeno, 2019[60]	21 AD 20 MCI 15 HC	T1 weighted sequences. Techniques: ROI analysis; VBM.	Association between $A\beta_{42}$ deposits and local atrophy of corresponding brain regions using BF-227 uptake (PET imaging).	In AD, $A\beta_{42}$ deposition was found in the inferior temporal gyrus and was related to GM atrophy in this region, while $A\beta_{42}$ deposition in the precuneus was associated with atrophy in the right occipital-temporal region. In HC, $A\beta_{42}$ deposition in the inferior temporal gyrus was associated with atrophy in the precuneus.
61. Preische et al., 2019[61]	243 mutation carriers, 162 non-carriers of APP, PSEN1, and PSEN2 mutations.	T1-weighted images. Techniques: CT.	NfL chain levels in CSF and serum. Association between NfL levels, CT and cognitive impairment.	Compared to non-carriers, mutation carriers showed higher NfL; the rate of NfL changes in this group discriminate them from non-mutation carriers almost a decade earlier than cross-sectional absolute NfL levels; serum NfL rate of change peaked in participants converting from the presymptomatic to the symptomatic stage was associated with CT, but not with amyloid load.
62. Rozzini et al., 2018[62]	44 AD 36 MCI 28 HC	T1-weighted sequence. Technique: visual rating.	Serum copper levels as measured with a newly developed method to detect	Retrieval of higher copper concentrations in MCI and dementia due to AD compared to controls. No

			serum copper free from proteins.	correlation between copper levels, cognitive and MRI measures were observed.
63. Varma et al., 2018[63]	MRI sample [from ADNI]: 767 prodromal AD and 207 preclinical AD.	T1-weighted sequences. Techniques: ROI analysis (SPARE-AD index).	Identification of brain and blood metabolites associated with disease pathology and progression.	Blood metabolites concentration was associated with AD-like patterns of brain atrophy (SPARE-AD index) and CSF biomarkers of AD pathology.
64. Nation et al., 2018[64]	10 MCI 22 HC	T1-weighted, T2- FLAIR sequences. Techniques: CT; VBM; ROI analysis; manual WML load.	Progenitor cell levels in MCI and HC; association between circulating levels to memory, brain volume, WML volume and cerebral perfusion.	MCI exhibited depletion of all circulating progenitor cells markers relative to HC; the depletion of CD34+ cells correlated with memory performance, left PCC CT and bilateral hippocampal perfusion.
65. Ning et al., 2018[65]	138 AD 225 HC 358 MCI	T1-weighted sequence. Techniques: ROIs analysis.	To test the effectiveness of neural network frameworks to classify and distinguish AD from MCI and HC through brain imaging and genetics.	Structural and genetic features taken together better predicted the distinction between AD and HC and the conversion from MCI to AD, with the most important predictors being the APOEε4 allele, and the volumes of the left middle temporal gyrus, the left hippocampus, and the right EC.

66. Ferrari et al., 2019[66]	38 suspected AD	FDG-PET-CT assessment and MRI: T1-weighted, DWI, FLAIR, T2-weighted sequences. Techniques: hippocampal volumetry.	Hippocampal volumetry and glucose metabolism for the diagnosis of AD using quantitative clinical tools.	Qualitative FDG-PET-CT analysis showed greater accuracy (0.87), sensitivity (0.76), and negative predictive value (0.77) than quantitative PET analysis and hippocampal MRI volumetry.
67. Moreland et al., 2018[67]	244 HC 241 AD	T1-weighted sequences. Techniques: predictive model computerized tool.	Prognostic psychometric and imaging data for the progression to dementia in patients at risk for AD.	The disease similarity index was able to estimate that the risk of progression within 3 years was higher for patients with both amyloid deposition and neurodegeneration than for patients with the former factor but not the latter.
68. Belathur Suresh et al., 2018[68]	269 HC 137 AD	T1-weighted sequences. Techniques: CT.	Identification of potential factors contributing to misclassification of HC as AD patients based on structural imaging.	A portion of HC which were clinically unimpaired (likely due to 'cognitive reserve') but harboured AD biomarkers of pathology were misclassified as AD patients from structural imaging. Other HC were misclassified from CT and had increased WMH volume compared to the those correctly classified.
69. Filippi et al., 2020[69]	94 AD 47 aMCI (25 converters within 36 months)	T1 weighted and rs-fMRI sequences. Techniques: Graph-analysis.	Structural and functional brain network architecture in AD and aMCI; relationship	In AD and aMCI converters, the point of maximal atrophy was the left hippocampus

	53 HC		between healthy brain network functional connectivity and the topography of brain atrophy in all patients.	(disease-epicenter). Brain regions most strongly connected with the disease-epicenter in the healthy functional connectome were also the most atrophic in AD and aMCI converters.
70. Meijboom et al., 2019[70]	11 AD 12 bvFTD 18 HC	3D T1-weighted, DTI, rs-fMRI sequences. Techniques: VBM, tractography, ICA.	Functional connectivity and tract-specific microstructural WM differences between AD and bvFTD.	The functional and WM differences between bvFTD and AD diminished at follow-up, but abnormalities were more pronounced in the bvFTD group.
71. Song et al., 2018[71]	52 HC	T1-weighted and DWI sequences. Techniques: Tractography.	Integrity of the fornix and the parahippocampal cingulum tracts in the asymptomatic phase of AD; assessment of amyloid load in healthy aging.	Increased neocortical Aβ burden over time was associated with increased radial diffusivity in the fornix but not in the parahippocampal cingulate bundle.
72. Huang et al., 2019[72]	40 aMCI 40 AD 40 HC	T1-weighted and SWI sequences. Techniques: visual rating.	Evaluation of perfusion imaging and SWI for early-disease sensitive markers of conversion from MCI to AD.	AD compared to MCI and HC: lower MoCA scores and widespread pattern of cerebral blood flow values. On SWI, the average numbers of haemorrhages in regions of lobes and bilateral basal ganglia/thalamus were significantly higher in AD, followed by aMCI and HC.

73. Park et al., 2019[73]	32 AD 95 MCI 127 HC	T2-weighted, T1-weighted, FLAIR, and SWI sequences. Techniques: visual rating and quantitative estimation of phase shift in the posterior bank of the motor cortex.	Prevalence and characteristics of motor cortex hypointensity and clinical significance.	Motor cortex hypointensity on SWI was more frequently found in MCI than HC and was positively associated with age.

Abbreviations: $A\beta_{42}$=Beta-Amyloid (β_{42}); ACE-R=Addenbrooke's cognitive examination-revised; AD=Alzheimer's disease; ADAS-cog=Alzheimer's Disease Assessment Scale-Cognitive; ADNI=Alzheimer's Disease Neuroimaging Initiative; ALFF=amplitude of low-frequency fluctuations; aMCI=amnestic mild cognitive impairment; ANU-ADRI=ANU Alzheimer's Disease Risk Index; APOE=apolipoprotein E; APP=Amyloid Precursor Protein; ASL=arterial spin labeling; BF-227=fluoro-ethoxy benzoxazole; bvFTD=behavioural variant of fronto-temporal dementia; CA=Cornu Ammonis; CBF=cerebral blood flow; CDR=Clinical Dementia Raring Scale; CLOX1=executive clock-drawing task; CSF=cerebrospinal fluid; CT=cortical thickness; DLB=Dementia with Lewy Bodies; DMN=Default Mode Network; DTI=diffusion tensor imaging; DWI=diffusion-weighted imaging; EC=entorhinal cortex; ECN=executive control network; EPI=echo-planar imaging; EXIT25=Executive Interview; FAB=Frontal Assessment Battery; FCSRT=Free and Cued Selective Reminding Test; FDG-PET-CT=fluorodeoxyglucose positron emission tomography-computed tomography; FEAT=Functional MRI Expert Analysis Tool; FLAIR=fluid attenuation inversion recovery; FLAME=FMRI's Local Analysis of Mixed Effects; fMRI=functional magnetic resonance imaging; FSL=FMRIB Software Library; FTD=frontotemporal dementia; FTLD=frontotemporal lobar degeneration; GM=gray matter; HC=healthy controls; ICA=Independent Component Analysis; MCI=mild cognitive impairment; MD=multi-domain; MMSE=Mini-Mental State Examination; MoCA=Montreal Cognitive Assessment; MRI=magnetic resonance imaging; N=number; NfL=Neurofilament Light chain; NOS=unspecified dementia; PCC=posterior cingulate cortex; PET=positron emission tomography; PFC=prefrontal cortex; pMCI=progressive MCI; PSEN1/2=presenilin1/2; RADAR=Reducing Pathology in Alzheimer's Disease through Angiotensin Targeting; pMCI=progressive memory impairment; ReHo=Regional Homogeneity; ROI=region of interest; rs-fMRI=resting state-functional magnetic resonance imaging; SCD=subjective cognitive decline; SD=single-domain; sMCI=stable mild cognitive impairment; SN=salience network; SPARE-AD=Spatial Pattern of Abnormality for Recognition of Early Alzheimer's disease; SPM=spatial parametric mapping; SRLM=stratum radiatum, lacunosum and moleculare; SWI= susceptibility weighted imaging; TBSS=Tract-based Spatial Statistics; VBM=voxel-based morphometry; VD=vascular dementia; WAIS=Wechsler Adult Intelligence Scale; WM=white matter; WMH=white matter hyperintensity; WML=white matter lesions.

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