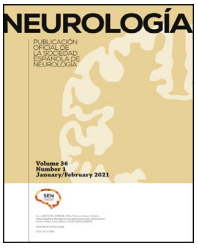
Neurología 40 (2025) 884—914

NEUROLOGÍA

www.elsevier.es/neurologia



REVIEW ARTICLE

Neuropsychological differential diagnosis of Alzheimer's disease and Lewy body dementia: A systematic review

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Received 28 November 2022; accepted 29 March 2023

KEYWORDS
Alzheimer's disease;
Dementia with Lewy bodies;
Differential diagnosis;
Neuropsychological assessment;
Neural correlate

Abstract
Introduction: Alzheimer's disease (AD) reports heterogeneity of neuropsychological symptoms misleading the differential diagnosis with other forms of dementia, such as dementia with Lewy bodies (DLB). About 50% of DLB patients are misdiagnosed as AD cases. Likewise, the diagnosis of both diseases is mainly based on clinical characteristics. However, differentiating AD of those with DLB based on neuropsychological symptoms and anatomical and functional brain changes remains challenging.
Aim: To establish the main neuropsychological, anatomical, and functional similarities and differences in patients with AD and DLB.
Methods: The present study followed the PRISMA guidelines and included studies from the PubMed, Scopus, and Web of Sciences databases, published between January 2000 and July 2022.
Results: 41 articles were included in this systematic review for critical analysis. Our results suggest that the cognitive key domains to consider in the differential diagnosis are memory, executive function, attention, visuospatial/visuoconstructive skills, and verbal fluency (both semantic and phonological). The stage and severity of both diseases would be essential for

DOI of refers to article: https://doi.org/10.1016/j.nrl.2023.03.006.
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https://doi.org/10.1016/j.nrleng.2025.10.001

differential diagnosis. On the other hand, the anatomical and functional changes suggest a similar atrophy pattern between AD and DLB in the frontal, parietal, temporal, hippocampal, and precuneus regions.
Conclusion: The differential diagnosis between AD and DLB is challenging in clinical practice. Therefore, our results suggest exploring cognitive linguistic markers along with correlating these markers with anatomical and functional brain changes.

PALABRAS CLAVES
Enfermedad de Alzheimer;
Demencia con Cuerpos de Lewy;
Diagnóstico diferencial;
Neuropsicología;
Redes neurales

Diagnóstico neuropsicológico diferencial en enfermedad de Alzheimer y demencia por cuerpos de Lewy: una revisión sistemática

Resumen
Introducción: La Enfermedad de Alzheimer (EA) reporta heterogeneidad de síntomas neuropsicológicos. Esto conduce a errores diagnósticos con otras formas de demencia como la demencia por cuerpos de Lewy (DPCL). De hecho, alrededor del 50% de los pacientes con DPCL son confundidos como casos de EA. Si bien el diagnóstico de ambos cuadros se basa principalmente en aspectos clínicos, continúa siendo un desafío su diferenciación en base a los síntomas neuropsicológicos y los patrones de atrofia cortical.
Objetivo: Establecer las principales similitudes y diferencias neuropsicológicas y de atrofia cortical en pacientes con EA y DPCL.
Metodología: La presente revisión sistemática siguió los lineamientos establecidos en la declaración PRISMA, utilizando las bases de datos PubMed, Scopus y Web of Science. La búsqueda estuvo limitada a estudios observacionales analíticos de pruebas diagnósticas, publicados en idioma inglés entre Enero 2000 y Julio 2022.
Resultados: La búsqueda dio como resultado 41 artículos finales. Del total de los artículos identificados se sugiere que los elementos neuropsicológicos claves para el diagnóstico diferencial entre EA y DPCL son la memoria, función ejecutiva, atención, habilidades visuoespaciales/visuoconstructivas, y fluidez verbal (semántica y fonológica) siendo el estadío y grado de severidad de cada cuadro críticos en el proceso diagnóstico. Los resultados además sugieren un patrón de cambios anatomo-funcionales similar entre EA y DPCL en áreas frontal, parietal, temporal, hipocampal y precuneus.
Conclusión: El diagnóstico diferencial entre EA y DPCL es un desafío en la práctica clínica por lo que esta revisión propone explorar marcadores cognitivos con énfasis en los indicadores lingüísticos además de los cambios anatómicos y funcionales de las áreas cerebrales.

Introduction

Alzheimer disease (AD) is the leading neurodegenerative cause of dementia, accounting for 60%—70% of all cases.[1—3] Advances in neuroimaging techniques have enabled the detection of molecular alterations in the brains of patients with AD, with high levels of sensitivity and specificity; specifically, positron emission tomography (PET) studies[4] can detect extracellular β-amyloid (Aβ) plaques and intracellular neurofibrillary tangles of hyperphosphorylated tau protein[5] in frontal, temporal, and parietal regions of the brain.[6,7] The most common methods for early detection of AD include Aβ-PET, tau-PET, [^{18}F]-fluorodeoxyglucose PET (FDG-PET), and volumetric analysis of brain MRI images. In addition to these neuroimaging techniques, cerebrospinal fluid (CSF) biomarkers of Aβ (Aβ42/Aβ40 ratio, Aβ42) and phosphorylated tau (p-tau) have been implemented in clinical practice to support the diagnosis of AD.[8]

Although AD has been the main subject of interest in this area of research, such other diseases as frontotemporal dementia (FTD) and Lewy body dementia (LBD) are also relevant.[9—11] Toloza-Ramírez et al.[12] highlight similarities between the symptoms of AD and behavioural variant FTD in terms of memory, executive function, and linguistic indicators.

LBD, in turn, is characterised by attention fluctuations,[13] and accounts for 0.3%-24.4% of all cases of dementia.[14] Neuropathological differential diagnosis considers α-synuclein deposition,[13,15,16] as well as FDG-PET findings of hypometabolism in the occipital lobe, sparing the posterior cingulate.[5]

In neuropsychological terms, LBD manifests with severe impairment of memory, attention, executive function, and visuoconstructive skills, unlike AD, in which episodic memory is the main domain affected.[17–19] Furthermore, both diseases present with language symptoms (e.g., verbal fluency), negatively affecting expressive language.[20–23]

The heterogeneity of symptoms and patterns of atrophy in AD is a current subject of interest in cognitive neuroscience. Some studies suggest that LBD is often misdiagnosed and mistaken for AD.[24–26] Likewise, post mortem studies reveal that approximately half of patients with LBD present a considerable burden of AD pathology; as a result, LBD is considered to be secondary to AD.[13,27] Oda et al.[28] suggest that cognitive and emotional decline in LBD is explained by the loss of cholinergic neurons, as is also the case in AD.

Various lines of research have focused on the differential diagnosis between AD and LBD, studying the role of patterns of atrophy as a differentiating factor and seeking to identify neuropsychological deficits.[29,30] Neuroimaging studies have demonstrated temporoparietal and occipital hypometabolism in both diseases; however, this sign tends to be milder in AD.[31] Furthermore, the right cingulate cortex is reported to display greater connectivity in LBD, whereas in AD, the left hippocampus shows greater connectivity.[32]

Some authors also describe neuropsychiatric symptoms that are key to differential diagnosis. Several studies suggest that hallucinations, agitation, and sleep alterations are more frequent in LBD than in AD; this is correlated with higher Neuropsychiatric Inventory (NPI) scores in patients with LBD.[33–36] Furthermore, motor symptoms are observed in up to 85% of cases with LBD[13]; although these are not characteristic of typical AD, they do present in atypical cases of AD, such as corticobasal syndrome.[37] In fact, the evidence suggests that these neuropsychiatric symptoms promote greater functional and cognitive impairment in these patients, and even favour axonal degeneration and the pattern of atrophy in brain regions relevant to cognitive function (e.g., frontal and limbic areas).[38–41]

Despite efforts to establish a consensus on the clinical and neuropathological characteristics involved in the differential diagnosis between AD and LBD,[5,13] the neuropsychological deficits that support differential diagnosis remain unclear, particularly those related to cognition and language. Therefore, the aim of this review is to establish the main similarities and differences between AD and LBD in terms of neuropsychological symptoms (particularly in the language domain) and anatomical/functional changes. Thus, improving the assessment of cognitive domains will favour the design of early, appropriate interventions in patients with dementia, and particularly interventions targeting functional status in the activities of daily living.

Methods

This systematic review observed the criteria and flow diagram established in the PRISMA statement.[42] The protocol was registered on the PROSPERO database (code CRD42021261164). The PRISMA checklist is included in the Supplementary material (Appendix 1).

Search strategy for identifying studies

The literature search was conducted on the following electronic databases, in the following order: PubMed, Scopus, and Web of Science. The search was limited to articles published between January 2000 and July 2022. This time period was selected due to the fact that research in the last 10 years has identified atypical profiles of AD; therefore, the search start date had to be extended to the year 2000 to obtain a general perspective of both typical and atypical AD. The general search strategy was as follows: (Alzheimer disease AND Lewy body dementia) AND (cognitive function OR memory OR executive function OR attention OR visuoconstructive skills OR visuospatial skills OR processing speed OR language) AND (neuropsychological assessment OR cognitive tests OR neuroimaging OR differential diagnosis). All terms were adapted to each database. The complete search strategy for each database is included in the Supplementary material (Appendix 2).

Study selection and inclusion/exclusion criteria

We applied the following inclusion criteria for screening by title and abstract: *a)* analytical observational studies of diagnostic tests, reporting neuropsychological assessment and/or neuroimaging study results; *b)* studies written only in English; and *c)* including patients aged at least 60 years with a clinical diagnosis of AD or LBD. We excluded the following types of article: *a)* editorials, experimental studies, systematic reviews with or without meta-analyses, study protocols, and theses; *b)* studies including patients with mixed dementia or pseudodementia; and *c)* studies including patients with history of psychiatric disorders (e.g., psychosis, schizophrenia).

Extraction of data

Studies were imported to the Rayyan software[43] to eliminate duplicate articles. Subsequently, 2 reviewers (VMC and DTR) applied the inclusion and exclusion criteria to the titles and abstracts of all articles. In the event that decisions could not be made based solely on the title and abstract, full texts of the articles were retrieved. A third reviewer (TJR) participated in the final selection of articles included for review, resolving disagreements through discussion where necessary.

Assessment of risk of bias and methodological quality

To evaluate the methodological quality and risk of bias of the articles selected, we used the QUADAS-2 tool,[44] an instrument designed and validated for the independent evaluation of methodological quality and risk of bias in studies of diagnostic accuracy. This instrument evaluates 4 key domains:

patient selection, the index test, the reference standard, and the patient flow and timing of the study (the latter 2 aspects are grouped together into the domain ''flow and timing''). Studies were classified into one of 3 risk categories: high, low, or unclear. Methodological quality was assessed according to the domains patient selection, index test, and reference standard, and was classified as high, low, or unclear.

Data synthesis strategy

Table 1 presents a narrative synthesis of our findings. This synthesis summarises the general characteristics of the studies reviewed, such as study population, mean age, and the instruments/measures used. It also presents the main findings regarding the neuropsychological similarities and differences between AD and LBD, with the neuroimaging profiles reported.

Results

Literature search

The PRISMA flow diagram in Fig. 1 illustrates the article selection process.[42] We identified the titles and abstracts of 447 articles, 50 of which were duplicates. We excluded 202 articles after application of the selection criteria. Of the 195 full-text articles screened for eligibility, 154 were excluded. Therefore, a total of 41 articles were included in the systematic review for qualitative analysis.

Imaging study characteristics

Table 1 shows the general characteristics of the 41 articles included for review. The year in which the greatest number of articles were published was 2017 (6 articles), with the years 2019, 2007, 2006, 2005, 2004, 2001 presenting the lowest number of articles (one each). The selected studies included a total of 8780 patients, with a mean age of 75.2 years (range, 60—80; standard deviation [SD]: 7.0). A total of 4498 (57%) were men.

Regarding the main neuropsychological findings, the results suggest that the cognitive domains of memory, attention, executive function, visuospatial/visuoconstructive skills, and verbal fluency (both semantic and phonological) are key aspects in the differential diagnosis of AD and LBD. From an anatomical/functional perspective, the pattern of atrophy in the frontal, parietal, and temporal lobes, as well as the hippocampus and precuneus, seems to enable differential diagnosis between the 2 disorders. However, the results underscore that the level of functional connectivity is reduced in sensorimotor, temporal, basal ganglia, thalamic, insular, and anterior cingulate networks in LBD, whereas in AD it is reduced in frontal and temporal areas, with increased connectivity in the posterior cingulate and hippocampus. This is a relevant finding, as this may be a useful indicator for establishing an early, accurate diagnosis.

Finally, the neuropsychological instruments and measures most frequently used to diagnose AD and LBD were the Mini—Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), Boston Naming Test (BNT), Trail-Making Test (TMT) parts A and B, Frontal Assessment Battery (FAB), FAS verbal fluency test, and Cambridge Cognition Examination (CAMCOG). The most frequently used neuroimaging techniques were MRI, fMRI, and PET, which are combined to establish an anatomical-functional correlate (Table 1).

Risk of bias and methodological quality assessment

The 41 articles selected were analysed using the QUADAS-2 tool (Fig. 2).[44] In the risk of bias assessment, the domains patient selection and flow and timing presented low risk of bias in 95% of studies and unclear risk in 5%. In the index test domain, 90% of studies presented low risk, 5% presented high risk, and 5% presented unclear risk. In the reference standard domain, 93% of studies presented low risk, 5% presented high risk, and 2% presented unclear risk. In turn, in the assessment of methodological quality, 95% of articles presented high quality for patient selection, compared to 90% and 93% for index test and reference standard, respectively.

Discussion

The objective of this review was to establish the main neuropsychological and anatomical/functional similaritiesand differences between AD and LBD. Our results suggest that memory, attention, executive function, visuospatial/visuoconstructive skills, and semantic and phonological verbal fluency may be considered additional indicators supporting differential diagnosis. Several studies[84—87] report differences in the progression of overall cognitive impairment in AD and LBD, suggesting that progression is faster in LBD. In fact, Giil and Aarsland[88] report that LBD progresses to advanced cognitive impairment within 5 years of follow-up. They also note that the main characteristic of AD is the heterogeneity of the cognitive domains affected. Regarding anatomical/functional changes, specific patterns of atrophy enabling differentiation between AD and LBD are reported in frontal, parietal, and temporal regions, the hippocampus, and the precuneus.

Episodic memory

Our findings suggest that episodic memory is impaired in both conditions. However, the deficit is more severe in AD; this is consistent with the results of previous studies.[13,89] In contrast, Calderon et al.[20] report that episodic memory impairment does not differ between the 2 diseases; in fact, they suggest that working memory is more severely

Table 1 Summary of the characteristics of the reviewed studies.

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Buciuc et al.[45] (2021)	Total sample of 20 participants: 6 with logopenic variant primary progressive aphasia and LBD, 7 with logopenic variant primary progressive aphasia and typical AD, 5 with logopenic variant primary progressive aphasia and hippocampal variant AD, and 5 with logopenic variant primary progressive aphasia and frontotemporal lobar degeneration	61.3 (—)	√	√	√	4 (20)/16 (80)	MMSE MoCA MDS-UPDRS III FBI NPI WAB BNT PPT TT WMS-III VOSP TMT-A D-KEFS ST ROCF FDG-PET PiB-PET	Patients with logopenic variant primary progressive aphasia and LBD displayed global cognitive impairment affecting memory, attention, executive function, and semantic and phonological verbal fluency; similar deficits are reported in patients with typical AD and the same type of aphasia. However, severity was greater in the first group. From a linguistic perspective, patients with LBD presented severe anomia and deficits in phonological fluency; this would be a differentiating factor with respect to the typical AD group. From an anatomical/functional perspective, patients with LBD presented moderate-severe neuronal loss in the substantia nigra; this sign was milder in the typical AD group. Greater parietal hypometabolism was observed in patients with LBD, whereas in AD the pattern of atrophy is heterogeneous, affecting temporal, parietal, and occipital areas.
Tak et al.[46] (2020)	Total sample of 76 participants: 38 with LBD and 38 controls	74.9 (6.2)	√	√	—	38 (50)/38 (50)	MMSE CDR DS TMT-A TMT-B eTIV MRI	In neuropsychological testing, patients with LBD displayed severe impairment of working and short-term memory, as well as significant alterations to executive function and attention performance. Marked atrophy was observed in the pulvinar nucleus, particularly in the left medial region; this alteration occurs at early stages, promoting executive deficits in LBD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Schumacher et al.[47] (2018)	Total sample of 91 participants: 31 with LBD, 29 with AD, and 31 controls	76.6 (7.5)	√	√	–	30 (33)/61 (67)	MMSE CAMCOG UPDRS-III CAF NPI fMRI	Neuropsychological evaluation suggested slight cognitive differences between LBD and AD, with the latter showing greater impairment of orientation to space and time, expressive language (verbal fluency), recent and distant memory, learning, attention, executive function, and perceptual abilities. Patients with LBD presented impaired functional connectivity of sensorimotor, temporal, basal ganglia, thalamic, insular, and anterior cingulate networks. AD was associated with reduced connectivity in frontal and temporal areas. However, comparison between the 2 revealed slight differences in frontal and temporal atrophy, which are key to the initial diagnosis of both diseases.
Donaghy et al.[48] (2018)	Total sample of 77 participants: 37 with LBD, 20 with AD, and 20 controls	75.9 (6.9)	√	√	–	14 (18)/63 (82)	MRI PET CT CSF ACE-R RAVLT TMT-A TMT-B FAS GNT IADL BADL NPI DCFS CAF GDS MDS-UPDRS motor score BP	Patients with LBD and positivity for beta amyloid present impaired orientation to time and place, attention, memory, semantic and phonological verbal fluency, visuospatial skills, delayed recall, and executive function. However, these deficits tend to be less pronounced in patients who are not positive for beta amyloid, suggesting that they may play a key role in the differential diagnosis between LBD and AD. AD was associated with greater atrophy of frontal, temporal, and parietal regions. Compared with AD, LBD presented greater volume of the hippocampus and medial temporal lobe, as well as greater orbitofrontal perfusion, which was not observed in AD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Elder et al.[49] (2017)	Total sample of 204 participants: 65 with LBD, 76 with AD, and 63 controls	77.8 (6.8)	√	√	—	75 (37)/129 (63)	MMSE CAMCOG UPDRS-III NPI CAF MRI	No significant differences in overall cognitive function were observed between AD and LBD; however, patients with AD presented better memory performance. Patients with LBD presented moderate atrophy of temporal, hippocampal, parahippocampal, and entorhinal cortical regions, compared to severe atrophy in AD.
Kemp et al.[50] (2017)	Total sample of 66 participants: 37 with LBD and 29 controls	68 (8.3)	√	√	—	33 (50)/33 (50)	CSF MRI IADL ADL MMSE FCSRT DMS-48 DS FAB TMT-A TMT-B DSB DMS-48 DO80 FLE DSym SG FSE ROCF VOSP Mini-SEA FPRT FER RMET	Patients with LBD displayed overall impairment of cognitive function. The memory domains of free recall and delayed recall were particularly affected. Furthermore, executive function, language (naming and semantic fluency), and visuoconstructive skills displayed severe impairment in this group. From an anatomical/functional perspective, patients with LBD presented bilateral hippocampal atrophy and positivity for beta amyloid and tau markers.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Peraza et al.[51] (2016)	Total sample of 53 participants: 19 with LBD, 18 with AD, and 16 controls	76.2 (6.9)	√	√	√	12 (23)/41 (77)	MRI fMRI MMSE UPDRS CAMCOG CAF NPI	Patients with LBD and AD displayed impairment in the cognitive domains of remote memory, recent memory, episodic and short-term memory, orientation, language (semantic and phonological verbal fluency), attention, abstract reasoning, executive function, and visuoconstructive skills. Although no significant differences were observed, neuropsychological test scores were lower in patients with AD. The patterns of brain involvement in LBD and AD were similar in such areas as the precuneus, posterior cingulate, and frontal lobe. Furthermore, patients with LBD displayed greater atrophy in the cerebellum, right precuneus, left lingula, left thalamus, and left postcentral gyrus.
Kobeleva et al.[52] (2017)	Total sample of 71 participants: 30 with LBD, 20 with AD, and 21 controls	75.4 (6.8)	√	√	–	13 (18)/58 (82)	MMSE CAMCOG FAS UPDRS CDS CAF NPI fMRI	Compared to LBD, AD was associated with greater impairment in the domains of memory, language, attention, perception, and abstract reasoning. However, executive function was severely impaired in LBD. These patients also showed greater impairment of phonological verbal fluency compared to those with AD. The anatomical/functional study identified increased functional connectivity in the posterior cingulate in AD and decreased connectivity in the left medial prefrontal cortex in LBD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Schumacher et al.[53] (2019)	Total sample of 89 participants: 39 with LBD, 28 with AD, and 22 controls	76 (6.3)	√	√	–	18 (20)/71 (80)	MMSE CAMCOG UPDRS CAF NPI ANT VBM MRI	Patients with LBD present less marked cognitive impairment than those with AD. Cognitive impairment is more evident in AD, affecting the domains of orientation, recent memory, remote memory, learning, language comprehension and speech, verbal fluency, attention, perception, and abstract reasoning. Similarly, patients with LBD presented more frequent cognitive fluctuations than those with AD. Furthermore, the pattern of atrophy in AD showed lower grey matter volume in the temporal, lingual, and left frontal cortex, as well as the precuneus. In contrast, LBD was associated with greater atrophy in the frontal cortex, particularly in the right hemisphere.
Heitz et al.[54] (2016)	Total sample of 64 participants: 33 with LBD, 15 with AD, and 16 controls	69.1 (10.0)	√	√	–	33 (52)/31 (48)	MMSE IADL CAF FCSRT DMS-48 DO80 FAB DS TMT-A TMT-B DSST FLE ROCF VOSP MRI VBM Mini-SEA RMET PF SF	Patients with AD presented impairment of immediate and delayed recall, attention, executive function, and semantic verbal fluency. In contrast, patients with LBD showed deficits in phonological verbal fluency, praxis, and abstract reasoning. Patients with AD presented greater atrophy in the left medial frontal gyrus, bilateral parietal lobe (including the precuneus), right cuneus, left lingual gyrus, and bilateral medial temporal lobe. Patients with LBD presented greater atrophy of the left cingulate gyrus and right medial frontal gyrus.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Chabran et al.[55] (2020)	Total sample of 159 participants: 79 with LBD, 58 with AD, and 22 controls	70.2 (8.5)	√	√	–	83 (52)/76 (48)	MMSE MCFS MRI fMRI VBM DMN FPN DAN	Patients with LBD and AD presented significant impairment of general cognition; however, AD was associated with greater involvement of memory, attention, visuoconstructive skills, and executive function. From an anatomical/functional perspective, patients with AD presented greater atrophy of the medial temporal lobe, including the parahippocampal gyrus, hippocampus, and amygdala. In LBD, greater atrophy was also observed in temporal lobe structures, as well as the insula and frontal lobes.
Yamamoto et al.[56] (2017)	Total sample of 130 participants: 57 with AD and 73 with LBD	72.6 (8.7)	√	√	–	99 (76)/31 (24)	MoCA	Patients with AD presented greater impairment of visuoconstructive skills and semantic and phonological verbal fluency. LBD was associated with significant memory impairment, particularly affecting delayed recall. Compared to AD, LBD was associated with a greater deficit in naming.
Azar et al.[30] (2020)	Total sample of 51 participants: 34 with AD and 17 with AD + LBD	74.7 (8.4)	√	√	√	29 (59)/22 (41)	MMSE CDR Modified MMSE HVLT BNT CFL VF TMT-A TMT-B PC CSF CT MRI	Patients with AD performed better for visuoconstructive skills and information processing speed. Regarding language, the AD group performed better for phonological verbal fluency. Therefore, these 3 domains are proposed as factors for study in the differential diagnosis of AD and LBD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Firbank et al.[57] (2016)	Total sample of 78 participants: 23 with LBD, 32 with AD, and 23 controls	75.7 (6.7)	√	√	–	16 (20)/62 (80)	MMSE UPDRS CAMCOG MCFS CAF NPI FAS ANT DMN fMRI	No significant differences in overall cognitive impairment were observed between AD and LBD. However, LBD was associated with greater impairment of executive function and poorer performance in phonological verbal fluency tasks. The anatomical/functional study revealed similar involvement in both groups in the frontal, parietal, and occipital lobes, with an impact on the connectivity of these areas.
Peraza et al.[58] (2015)	Total sample of 54 participants: 18 with LBD, 19 with AD, and 17 controls	76.2 (6.8)	√	√	–	11 (20)/43 (80)	MMSE UPDRS CAMCOG CAF NPI fMRI	Compared to LBD, AD was associated with greater deficits in the cognitive domains of orientation, recent memory, remote memory, learning, concentration, perception, abstraction, and language (comprehension and expression). From an anatomical/functional perspective, patients with LBD presented greater involvement of parietal and posterior occipital regions. In contrast, the most affected areas in AD were the temporal cortex, right occipital lobe, and right frontal lobe.
Brenowitz et al.[59] (2017)	Total sample of 1603 participants: 193 without major neuropathology, 195 with stroke, 110 with LBD, 51 with LBD + stroke, 450 with AD, 292 with AD + LBD, 217 with AD + stroke, and 95 with AD + LBD + stroke	83 (9.9)	√	√	√	706 (44)/897 (56)	CDR LMS-A DS SF PF BNT DSym TMT-A TMT-B MRI	Patients with AD present greater impairment of episodic and working memory than those with LBD. No significant differences between groups were observed for attention, executive function, or language (semantic verbal fluency and naming); however, faster decline was observed in patients with AD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Pizzi et al.[60] (2015)	Total sample of 45 participants: 16 with AD, 16 with LBD, and 13 controls	75.6 (4.8)	√	√	—	23 (51)/22 (49)	CDR MMSE DRS FAB CAF UPDRS-III NPI MRI SPECT EEG	In terms of overall cognitive performance, patients with AD presented greater deficits in the domains of memory, orientation to space and time, delayed recall, visuoconstructive skills, and executive function, compared to the LBD group. AD was associated with greater atrophy of the bilateral thalamus and temporal cortex. In contrast, the pattern of atrophy in LBD involved the bilateral thalamus and projections to the prefrontal, parietal, and occipital cortex bilaterally.
Jiménez-Huete et al.[61] (2014)	Total sample of 301 participants: 199 with AD, 65 with frontotemporal lobar degeneration, and 37 with LBD	75 (8.0)	√	√	√	160 (53)/141 (47)	MEC CFL PF TMT-A TMT-B CDT SS-IQCODE FAQ GDS-15 SRT	Patients with AD presented difficulties with naming, free recall, cued recall, and attention. Patients with LBD presented alterations in phonological verbal fluency, executive function, and visuoconstructive skills. Both diseases presented similar levels of impairment in semantic verbal fluency and short-term and working memory.
Nervi et al.[62] (2008)	Total sample of 591 participants: 157 with AD, 70 with LBD, and 364 controls	80 (7.6)	√	√	√	396 (67)/195 (33)	CDR SRT BVRT RDT MMSE SF PF BNT BDAE WAIS-R UPDRS APOE-ε4 genotype	Patients with AD presented impairment of delayed recall, visuoconstructive skills, orientation, naming, and semantic and phonological verbal fluency, compared to patients with LBD. From a diagnostic perspective, no significant differences were observed between groups for presence of APOE-ε4.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Bradshaw et al.[63] (2004)	Total sample of 25 participants: 13 with LBD and 12 with AD	78.3 (4.5)	√	√	√	12 (48)/13 (52)	MMSE CDR NART UPDRS HADS BPRS FSIQ VIQ PIQ CAF WASI BI	Significant differences in neuropsychological test performance were observed between groups. Patients with AD presented greater memory impairment, whereas those with LBD presented greater impairment of attention. These deficits play a key role in patients' functional difficulties.
Colloby et al.[64] (2017)	Total sample of 127 participants: 41 with LBD, 47 with AD, and 39 controls	78.3 (7.1)	√	√	–	43 (34)/84 (66)	MMSE CAMCOG UPDRS-III NPI CAF TIV MRI CSF VBM	No significant differences were observed between AD and LBD in orientation, short- or long-term memory, recent memory, language, attention, abstract reasoning, perception, and visuoconstructive skills. Both diseases showed a similar pattern of atrophy in the hippocampus bilaterally.
Colloby et al.[65] (2014)	Total sample of 127 participants: 41 with LBD, 47 with AD, and 39 controls	78.3 (7.1)	√	√	√	43 (34)/84 (66)	MMSE CAMCOG NPI RBD UPDRS-III CAF TIV VBM MRI	No significant differences were observed between AD and LBD in impairment of memory, orientation, language, visuoconstructive skills, or attention. From an anatomical/functional perspective, patients with AD present greater grey matter loss in the cerebellum and bilateral temporal lobe. AD is also associated with white matter loss in the bilateral middle cerebellar peduncle. Patients with LBD presented grey matter loss in the cerebellum, although this was not significant compared to AD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Kraybill et al.[66] (2005)	Total sample of 135 participants: 48 with AD, 22 with LBD, and 65 EA plus LBD	76 (6.8)	√	√	—	77 (57)/58 (43)	MMSE DRS TMT-A WAIS-R DS CERAD-N FOME WMS WAIS-R TMT-A TMT-B ASN	Patients with AD presented greater impairment of memory (delayed recall) and naming. LBD was associated with greater deficits in cognitive function and attention. No significant differences between groups were observed for visuoconstructive skills.
Gomperts et al.[67] (2008)	Total sample of 78 participants: 8 with LBD, 7 with Parkinson's disease dementia, 11 with Parkinson's disease, 15 with AD, and 37 controls	71.3 (2.1)	√	√	—	39 (50)/39 (50)	CDR UPDRS MMSE BDS DS TMT-A TMT-B CFL PF BNT	LBD was associated with greater impairment of attention, executive function, delayed recall, episodic memory, naming, and semantic and phonological verbal fluency, compared to AD.
							LM FRSRT FCSRT BVDT MCFS WAIS-R NPI GDS AMNART-VIQ PET PiB-PET	Patients with AD presented greater temporal lobe atrophy and less marked occipital lobe involvement. Patients with LBD presented greater involvement of the lateral parietal lobe, precuneus, and posterior cingulate cortex.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Taylor et al.[68] (2013)	Total sample of 42 participants: 23 with AD and 19 with LBD	77.5 (10.0)	√	√	√	19 (45)/23 (55)	MMSE CAMCOG UPDRS-III CAF CRT SPECT	The most affected domains in AD are memory, language, orientation, and abstract reasoning. Patients with LBD presented greater impairment of attention. From an anatomical/functional perspective, both groups presented atrophy of the bilateral thalamus, putamen, and precuneus, and of the left occipital lobe. Patients with AD also presented atrophy of the bilateral pre- and post-central gyri and cerebellum, as well as the left temporal lobe. Patients with LBD displayed atrophy of the bilateral anterior cingulate cortex and caudate nucleus.
Hamilton et al.[69] (2008)	Total sample of 66 participants: 22 with LBD and 44 with AD	72.7 (5.9)	√	√	–	32 (48)/34 (52)	MMSE MDRS WISC-R BD BNT ADL BI	Patients with LBD presented severely impaired visuoconstructive skills, whereas AD was associated with greater impairment of language abilities. These alterations were predictive of progression to severe overall cognitive impairment in both groups.
Fong et al.[70] (2011)	Total sample of 46 participants: 14 with LBD, 16 with AD, and 16 controls	70.7 (9.5)	√	√	–	21 (46)/25 (54)	MMSE CT MRI CERAD COWAT BNT RAVLT SF FAS DS	Patients with LBD performed better in delayed recall, recognition, and phonological verbal fluency, compared to those with AD. Patients with AD presented greater impairment in naming and semantic verbal fluency tasks. The anatomical/functional study revealed hypoperfusion of the bilateral frontal lobe, precuneus, and parietal and occipital lobes in LBD. The AD group showed frontal, parietal, and occipital hypoperfusion; however, this alteration was more severe in LBD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Ballard et al.[71] (2001)	Total sample of 190 participants: 85 with LBD, 80 with AD, and 35 controls	77 (6.8)	√	√	–	103 (54)/87 (46)	MMSE CDS M-UPDRS COGDRAS-D CRT CogRT DS WAIS-R VIG	While both LBD and AD present significant general cognitive impairment, the differentiating factor is the deficit and fluctuations in attention performance, which are more severe in LBD.
Kawai et al.[72] (2013)	Total sample of 440 participants: 402 with AD and 38 with LBD	78.8 (6.3)	√	√	–	313 (71)/127 (29)	MMSE ADAS-Jcog FAB RCPM DS LM WMS-R MRI CT	Patients with AD presented greater impairment of orientation to time and space, as well as delayed recall. In turn, patients with LBD showed greater impairment of attention, executive function, visuoconstructive skills, and phonological verbal fluency. These domains are proposed as key factors for differential diagnosis of AD and LBD.
Ryman et al.[73] (2021)	Total sample of 2433 participants: 111 with LBD, 741 with AD plus LBD, 1357 with AD, and 224 controls	80.1 (9.5)	√	√	√	1086 (45)/1347 (55)	MMSE CERAD TMT-A TMT-B DSym VF SF BNT DS BI	LBD was associated with severe impairment of executive function and visuospatial skills. The AD group showed significant deficits in memory and language. Both groups showed similar levels of attention impairment.
Watson et al.[74] (2015)	Total sample of 94 participants: 31 with LBD, 30 with AD, and 33 controls	77.5 (6.0)	√	√	–	33 (35)/61 (65)	MMSE	Patients with AD presented greater overall cognitive impairment than those with LBD. They also presented severe delays in attention, memory, orientation to space and time, language, executive function, and visuoconstructive skills.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
							CAMCOG UPDRS-III NPI B-ADL MRI CSF	In the AD group, the anatomical/functional study revealed atrophy of temporal and parietal areas, extending to frontal regions. These patients presented severe atrophy of the hippocampus, precuneus, and entorhinal cortex. Patients with LBD displayed atrophy in the inferior parietal region, posterior cingulate, and fusiform gyrus.
Prats-Sedano et al.[75] (2021)	Total sample of 182 participants: 76 with LBD, 40 with AD, and 66 controls	73.8 (7.1)	√	√	–	48 (26)/134 (74)	MMSE ACE-R BI	Patients with AD presented greater memory impairment. Patients with LBD showed greater deficits in visuospatial abilities. No significant differences were observed between groups for attention, orientation, or language. Patients with LBD performed slightly worse in verbal fluency than those with AD.
Mitolo et al.[76] (2016)	Total sample of 228 participants: 28 with LBD, 115 with AD, and 85 controls	73.1 (8.6)	√	√	–	–/–	MMSE MDRS VOT CDT WISC-R BD BNT SF CVLT WMS-R LM	Patients with LBD presented greater deficits in visuospatial and visuoconstructive abilities, semantic verbal fluency, and language. While language was also impaired in AD, episodic memory was most significantly affected in this group.
Kenny et al.[32] (2012)	Total sample of 47 participants: 15 with LBD, 16 with AD, and 16 controls	78.1 (7.7)	√	√	–	–/–	MMSE UPDRS-III GDS	Despite mild differences in scores, there was no significant difference between patients with AD and LBD in overall cognitive performance. Therefore, the results demonstrate similar levels of impairment in both groups. However, memory is severely affected in patients with AD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
							NPI CAMCOG CAF fMRI	The anatomical/functional study revealed greater connectivity in the left hippocampus, right insula, and left inferior parietal lobe in patients with AD. Patients with LBD presented greater connectivity in the right posterior cingulate cortex, limbic area (left anterior cingulate), right globus pallidus, right culmen, and right cerebellar tonsil. Both groups showed similar connectivity in the insula, thalamus, and caudate nucleus.
Stavitsky et al.[77] (2006)	Total sample of 83 participants: 55 with AD and 28 with LBD	73.3 (7.9)	√	√	√	43 (52)/40 (48)	Modified MMSE DS HVLT-R IADL ADL DDS	Patients with AD presented greater memory impairment, whereas those with LBD presented greater impairment of visuoconstructive skills. Furthermore, patients with AD present greater behavioural alterations and increased visual hallucinations; in LBD, these symptoms tend to remain stable over the course of the disease. No significant difference between groups was observed for language deficits.
Mak et al.[78] (2016)	Total sample of 104 participants: 35 with LBD, 34 with AD, and 35 controls	77.7 (6.0)	√	√	–	38 (36)/66 (64)	MMSE CAMCOG CAF UPDRS-III NPI HVLT BVMT MRI	AD was associated with greater impairment of delayed recall, information retention capacity, verbal memory, and learning, compared to LBD. Patients with LBD presented greater impairment of recent memory, and greater cognitive fluctuations than patients with AD. Patients with LBD displayed greater atrophy of the medial hippocampus. Although hippocampal atrophy is a characteristic feature of AD, the results suggest that atrophy of the CA1 region is a key factor in differentiating AD and LBD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Park et al.[79] (2011)	Total sample of 103 participants: 10 with LBD, 76 with AD, and 17 with Parkinson's disease dementia	70.2 (7.2)	√	√	—	68 (66)/35 (34)	K-MMSE CDR GDS BADL DS K-BNT ROCF COWAT ST SVLT MRI	Patients with LBD present greater impairment of executive function and attention than those with AD. Both diseases were associated with impairment of episodic memory. Language and naming were impaired in both groups, although patients with LBD presented lower scores.
Breitve et al.[80] (2018)	Total sample of 186 participants: 119 with AD and 67 with LBD	75.6 (7.5)	√	√	√	116 (62)/70 (38)	MMSE CDR CVLT-II TMT-A TMT-B COWAT BNT VOSP ST I-FP-CIT SPECT	Patients with AD performed worse than those with LBD in attention, language (semantic verbal fluency), executive function, and memory (delayed recall). However, during the follow-up period, severe attention impairment in LBD was predictive of progression and deterioration in this patient group; this was not the case in AD.
Mak et al.[81] (2015)	Total sample of 69 participants: 13 with LBD, 23 with AD, and 33 controls	76.7 (6.3)	√	√	√	24 (35)/45 (65)	MMSE	No significant differences were observed at baseline or during follow-up between LBD and AD in the domains memory, attention, executive function, visuoconstructive skills, perception, abstract reasoning, or verbal fluency. However, greater cognitive fluctuations were observed in LBD than in AD.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
							BADL UPDRS-III NPI CogFluct CAMCOG MRI	The anatomical/functional study revealed a pattern of atrophy involving the left superior and middle temporal gyri, extending to the left lingual gyrus. In contrast, patients with LBD displayed frontal and parietal atrophy.
Firbank et al.[82] (2007)	Total sample of 46 participants: 16 with LBD, 15 with AD, and 15 controls	75.6 (7.3)	√	√	–	18 (39)/28 (61)	MMSE CAMCOG UPDRS NPI CDS UPDRS-III CT MRI	Overall cognitive performance was severely impaired in LBD, with alterations in attention, orientation, memory, language (verbal fluency), visuoconstructive skills, perception, abstract reasoning, and executive function. In the anatomical/functional study, LBD was associated with atrophy of the right postcentral gyrus. In contrast, the pattern of atrophy in AD involved the inferior and medial anterior temporal lobe, as well as the inferior longitudinal and uncinate fasciculus.

Table 1 (*Continued*)

Lead author (year)	Population	Age	Age range			Sex	Instruments /measures	Main findings
		Mean (SD)	(years)			♀/♂		
			60-70	71-80	> 80	*n* (%)		
Watson et al.[83] (2012)	Total sample of 106 participants: 35 with LBD, 36 with AD, and 35 controls	77.8 (6.0)	√	√	√	38 (36)/68 (64)	MMSE CAMCOG VF BADL NPI UPDRS-III MRI	Both AD and LBD were associated with episodic memory deficits. Furthermore, phonological verbal fluency was more severely impaired in LBD than in AD. The anatomical/functional study showed that LBD was associated with atrophy of parietal, occipital, and temporal areas, with extension to the frontal lobe. AD tended to show a more generalised pattern of atrophy, involving the same areas as LBD but with greater temporal lobe atrophy.

√: included; —: not reported.

ACE-R: Addenbrooke's Cognitive Examination-revised; ADAS-Jcog: Alzheimer's Disease Assessment Scale-Cognitive Component-Japanese version; ADL: Activities of Daily Living; AMNART-VIQ: American version of the National Adult Reading Test, verbal IQ; ANT: Attention Network Test; ASN: α-synuclein; BADL: Bristol Activities of Daily Living Scale; BDAE: Boston Diagnostic Aphasia Examination; BDS: Blessed Dementia Scale; BI: brain imaging; BNT: Boston Naming Test; BP: blood pressure; BPRS: Brief Psychiatric Rating Scale; BVDT: Benton Visual Form Discrimination Test; BVMT: Brief Visuospatial Memory Test; BVRT: Benton Visual Retention Test; CAF: Clinician Assessment of Fluctuation; CAMCOG: Cambridge Cognitive Examination; CASI: Cognitive Abilities Screening Instrument; CDR: Clinical Dementia Rating Scale; CDS: Cornell Depression Scale; CDT: Clock Drawing Test; CERAD: Consortium to Establish a Registry for Alzheimer's Disease Neuropsychological Battery; CERAD-N: CERAD naming subtest; CFL: category fluency; COGDRAS-D: Cognitive Drug Research Computerized Assessment System for Dementia Patients; CogFluct: Cognitive Fluctuation Scale; CogRT: cognitive reaction time; COWAT: Controlled Oral Word Association Test; CRT: choice reaction time; CSF: cerebrospinal fluid; CT: computed tomography; CVLT: California Verbal Learning Test; CVLT-II: CVLT, second edition; DAN: dorsal attention network; DCFS: Dementia Cognitive Fluctuation Scale; DDS: Dependence Scale sum; D-KEFS ST: Delis-Kaplan Executive Function Sorting Test; DMN: default mode network; DMS-48: Delayed Matching to Sample-48 items; DO80: Oral Denomination-80 items; DRS: Dementia Rating Scale; DS: digit span; DSB: digit span backward; DSST: Digit Symbol Substitution Test; DSym: digit symbol; EEG: electroencephalography; eTIV: estimated total intracranial volume; FAB: Frontal Assessment Battery; FAQ: Functional Assessment Questionnaire; FAS: FAS verbal fluency; FBI: Frontal Behavioral Inventory; FCSRT: Free and Cued Selective Reminding Test; FDG-PET: [^{18}F]-fluorodeoxyglucose positron emission tomography; FER: facial emotion recognition; FLE: Lexical Evocation; fMRI: functional magnetic resonance imaging; FOME: Fuld Object Memory Evaluation; FPN: frontoparietal network; FPRT: Faux Pas Recognition Test; FRSRT: Free Selective Reminding Test; FSE: formal semantic evocation; FSIQ: Full Scale IQ; GDS: Geriatric Depression Scale; GDS-15: Geriatric Depression Scale-15 items; GNT: Graded Naming Test; HADS: Hospital Anxiety and Depression Scale; HDRS: Hamilton Depression Rating Scale; HVLT: Hopkins Verbal Learning Test; HVLT-R: Hopkins Verbal Learning Test-Revised; IADL: Instrumental Activities of Daily Living scale; I-FP-CIT SPECT: ioflupane single-photon emission computed tomography; K-BNT: Korean version of the Boston Naming Test; K-MMSE: Korean version of the Mini—Mental State Examination; LM: logical memory; LMS-A: Logical Memory Story A-immediate and delayed recall; MCFS: Mayo Clinic Fluctuation Scale; MDRS: Mattis Dementia Rating Scale; MDS-UPDRS: Movement Disorders Society Unified Parkinson's Disease Rating Scale motor sub-scale; MEC: Miniexamen Cognoscitivo; MFCS: Mayo Fluctuation Composite Score; Mini-SEA: Mini-Social Cognition and Emotional Assessment; mMMSE: modified Mini—Mental State Examination; MMSE: Mini—Mental State Examination; MoCA: Montreal Cognitive Assessment; MRI: magnetic resonance imaging; M-UPDRS: Modified Unified Parkinson's Disease Rating Scale; NA: neuropsychological assessment; NART: National Adult Reading Test; NPI: Neuropsychiatric Inventory; PC: Pentagon Copy; PET: positron emission tomography; PF: phonological fluency; PiB-PET: Pittsburgh compound B positron emission tomography; PIQ: Performance IQ; PPT: Pyramids and Palm Trees test; RAVLT: Rey Auditory Verbal Learning Test; RBD: REM sleep behaviour disorder; RCPM: Raven's Coloured Progressive Matrices; RDT: Rosen drawing test; RMET: Reading the Mind in the Eyes Test; ROCF: Rey-Osterrieth Complex Figure; SD: standard deviation; SF: semantic fluency; SG: symbol gesture; SPECT: single-photon emission computed tomography; SRT: Selective Reminding Test; SS-IQCODE: Shortened Spanish-Informant Questionnaire on Cognitive Decline in the Elderly; ST: Stroop Test; SVLT: Seoul Verbal Learning Test; TIV: total intracranial volume; TMT: Trail-Making Test; TT: token test; UPDRS: Unified Parkinson's Disease Rating Scale; UPDRS-III: Unified Parkinson's Disease Rating Scale Part III; VBM: voxel-based morphometry; VF: verbal fluency; VH: visual hallucinations; VIG: Tasks and Digit Vigilance; VIQ: Verbal IQ; VOSP: Visual Object and Space Perception Battery; VOT: Visual Organization Test; WAB: Western Aphasia Battery; WAIS-R: Wechsler Adult Intelligence Scale, revised; WASI: Wechsler Abbreviated Scale of Intelligence; WCST: Wisconsin Card Sorting Test; WISC-R BD: Wechsler Intelligence Test for Children-Revised, block design subtest; WMS: Wechsler Memory Scale; WMS-R: Wechsler Memory Scale-Revised.

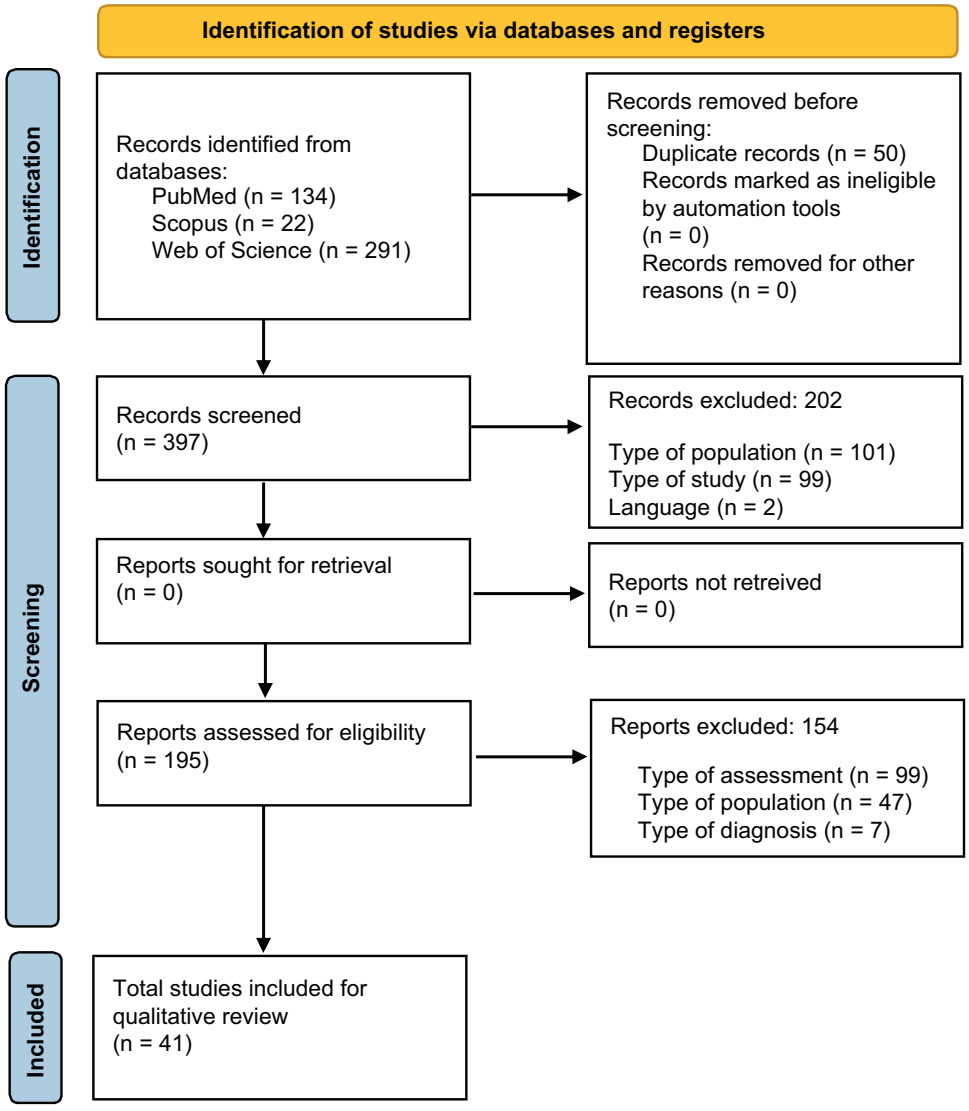

Figure 1 PRISMA flow chart.

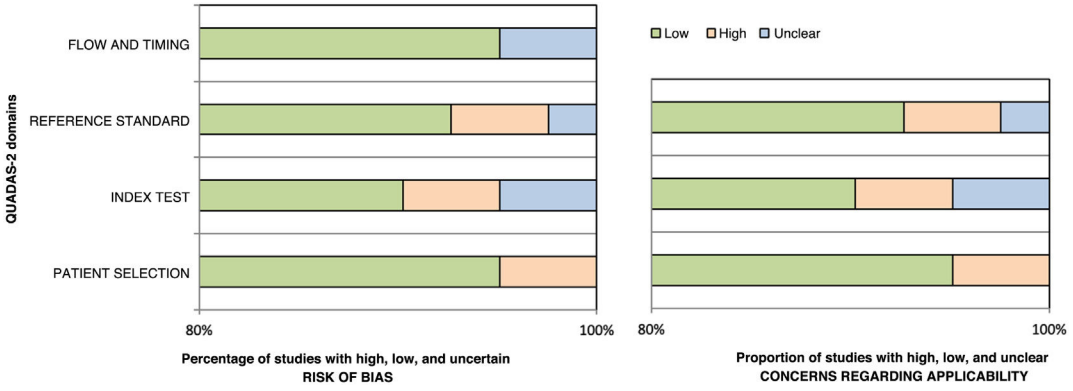

Figure 2 Assessment of risk of bias and methodological quality.

affected in LBD. Significant differences were also observed in the ability to consolidate information, with AD showing severe deficits mainly affecting delayed recall and verbal memory.[45]

Another recent study[90] notes that analysis during the pre-dementia stage is key to differentiating between patients who will progress to AD or LBD. The results suggest that patients in the mild cognitive impairment stage who present

mild memory deficits tend to progress to LBD, whereas progression to AD is associated with severe memory impairment. Previous studies[91–93] report that this performance is also observed in patients with subjective memory complaints. Similarly, Lemstra et al.[94] highlight the relevance of LBD co-occurring with AD, as memory decline tends to be severe in these patients, even at early stages. Nonetheless, the results reported in the literature are contradictory, with other studies suggesting that memory performance is better in patients with AD plus LBD than in those with pure AD[19,95]; on the other hand, Azar et al.[30] suggest that memory impairment is no different in patients with AD and concomitant LBD. These discrepancies may be explained by differences in sample size. It should also be noted that there is a clear overlap in the cognitive symptoms and patterns of brain involvement between AD and AD plus LBD; as a result, differential diagnosis is relatively imprecise.[77,96]

Attention and executive function

Attention deficit and executive function decline were identified as cognitive markers for the differential diagnosis of AD and LBD. In line with our own findings, Xu et al.[97] report that both domains are more severely impaired in LBD than in AD. Furthermore, several authors[98–100] suggest that selective attention is a differential cognitive marker in early stages. Although attention deficit manifests early in AD, it is more evident in moderate and severe disease.[71] However, other studies present contradictory results,[101–103] suggesting that divided attention is key to differentiating between these diseases, as it is more severely impaired in LBD.

Regarding executive function, specific subdomains are proposed for differential diagnosis. For instance, the evidence suggests that inhibitory control[97,104,105] and cognitive flexibility[50,106–109] are affected in LBD. In contrast, Bailon et al.[110] and Bussè et al.[103] suggest that cognitive flexibility does not present significant impairment in LBD. Similarly, discrepancies were also observed regarding performance in abstract reasoning tasks and conceptual skills. While some authors assert that these domains are unaffected,[105,108] Perri et al.[111] report severe impairment in patients with LBD compared to those with AD.

Visuocognitive skills

Visuoconstructive and visuospatial deficits are observed in both AD and LBD. Ferman et al.[107] note that visuoconstructive impairment enables accurate differentiation between LBD, AD, and healthy elderly adults (sensitivity: 80%; specificity: 90%). Similarly, other studies suggest that visuoconstructive skills are severely impaired in LBD, whereas AD is associated with severe impairment of episodic memory, favouring differential diagnosis.[112–114] Furthermore, these studies report that early stages are crucial in identifying the deficit, as motor symptoms in advanced stages of LBD contribute to diagnostic errors.

Visuospatial skills are also proposed as a key element in differential diagnosis. Classically, severe visuospatial deficit has been considered a differentiating factor in LBD[66,77,95,98]; however, interesting findings are also reported in AD. It has been suggested that deficits in these skills may be the first cognitive manifestations of AD, with the initial stage being fundamental to distinguishing it from LBD.[115–118]

Changes in visuoperceptual skills have been associated with these diseases; among the different types of visuocognitive ability, these skills stand out for their high complexity. Visuoperceptual skills are reported to be significantly impaired in LBD, enabling differentiation from AD at early stages.[22,54,62,76,119,120] However, other studies report diverging results, with these deficits also having been observed in preclinical and mild AD.[121,122]

Semantic and phonological verbal fluency

Various studies[62,105,106,123] report that patients with LBD present severe impairment of semantic and phonological verbal fluency, as well as deficits in naming. Ralph et al.[22] support this hypothesis, but note that patients with typical AD present better phonological performance. Other researchers[124–126] argue that the deficit in semantic verbal fluency in AD is due to a loss of semantic knowledge. Similarly, patients with LBD also show language impairment, but this differs from that observed in AD in that lexical and semantic abilities are spared.[127] Additionally, it should be noted that verbal fluency tests involving verbs have also been proposed as cognitive/linguistic markers. For instance, Delbeuck et al.[128] found that patients with LBD were able to produce higher numbers of verbs than those with AD.

Neuropsychological assessment tools

The studies reviewed frequently used the MMSE and MoCA test to evaluate cognitive performance. The MMSE is a brief, simple cognitive screening test, but is unable to exhaustively assess the different cognitive domains. However, its use is encouraged in clinical practice as it is able to detect progression of cognitive impairment and to differentiate AD from LBD. In fact, patients with LBD show significant reductions in scores at annual follow-up visits compared to those with AD.[84,88,129] Similarly, longitudinal studies[80,130,131] highlight the rapid decline observed in MMSE scores in LBD. Although the MMSE is more useful than the MoCA test for identifying cognitive impairment in LBD,[132] it is not recommended for studying progression of the diagnosis and cognitive decline in longitudinal studies.[133] Using the MMSE to monitor disease progression is only helpful in more advanced stages of LBD (General Deterioration Scale scores > 4), as results are often normal in earlier stages, when abnormal results indicate an amnestic profile suggesting copresence of AD. Furthermore, variation is reported in MMSE scores, which differ significantly in LBD (lower scores on the MoCA test and higher scores on the MMSE).[134]

Our results showed that the Free and Cued Selective Reminding Test is not frequently used, despite its demonstrated efficacy in identifying patients with AD.[135] Bussè et al.[136] studied the test's effectiveness for differentiating LBD from AD, finding that although patients with LBD pre-

sented greater overall performance, total recall and delayed free recall were key to differentiating the 2 diseases.

Language evaluation is mainly based on such tests as the BNT, FAS, semantic verbal fluency, and the language subdomains of the CAMCOG. Although these tools offer a general view of language performance, they do not assess specific subdomains in detail. For instance, the Mini—Linguistic State Examination (MLSE),[137] recently validated for classifying primary progressive aphasia in Spanish-speaking populations,[138] may be useful for better characterising language deficits in AD and LBD based on phonology, semantics, and syntax (including working memory). However, language evaluation should also consider spontaneous language, which is thought to play a key role in differentiating between these 2 diseases.[139]

Neuroimaging techniques and atrophy pattern

Structural MRI is frequently used to diagnose AD and LBD, and is able to identify structural damage and grey matter loss. Previous studies[140—142] confirm these findings, highlighting the overlap between the patterns of atrophy in LBD and in AD. However, atrophy is more diffuse in LBD, with moderate preservation of the medial temporal lobe. Similarly, several other studies[143—145] report that atrophy in AD involves not only the temporal lobe, but also the cingulate, parahippocampal regions, and the precuneus. In LBD, cortical thinning is reported to affect superior temporo-occipital regions, the lateral orbitofrontal cortex, and the posterior and medial cingulate cortex.[143—145]

Furthermore, fMRI studies show greater connectivity in the inferior parietal cortex and putamen in LBD, whereas patients with AD display greater connectivity in frontoparietal areas, the medial prefrontal cortex, the primary visual cortex, and the left hippocampus.[146,147] In contrast, Schumacher et al.[47] recommend caution in the interpretation of fMRI results, as the increase in frontotemporal connectivity in LBD overlaps with that reported in AD.

The growing interest in establishing an accurate differential diagnosis between AD and LBD has promoted the use of PET studies. This technique is useful for identifying AD and for distinguishing it from LBD, with the hippocampus being the region of interest. Tau-PET reveals greater concentration of tau protein in the hippocampus in AD, with more moderate concentrations in LBD.[148—150] Furthermore, FDG-PET results show hypometabolism in the occipital region in LBD, with sparing of the posterior cingulate, whereas hypometabolism in AD affects temporo-occipital regions.[5] Finally, Marquié et al.[151] suggest that the radiotracer ^{18}F-AV-1451 in tau-PET studies may be a potential biomarker for differential diagnosis, as it presents high affinity for tau neurofibrillary pathology in AD.

Limitations

This study presents 4 significant limitations. Firstly, the review does not include a meta-analysis, which would improve the interpretation of the neuropsychological and imaging findings in AD and LBD. Secondly, only 3 databases were consulted (PubMed, Scopus, and Web of Science). Future studies should search a larger number of databases to obtain a global view of the differential diagnosis of these diseases. Thirdly, the search strategy was limited, focusing only on observational studies of diagnostic tests and excluding other study designs and even neuropathological diagnosis. Similarly, given the range of years of publication, we consider that there is a need for future studies in this area to take into account updates to diagnostic criteria. Finally, we excluded atypical AD profiles, such as primary progressive aphasia and posterior cortical atrophy, as numerous studies suggest that these share symptoms with LBD.[152—155]

Contributions to clinical practice

To our knowledge, this is the first systematic review addressing differential diagnosis between AD and LBD that considers both the neuropsychological profile and the anatomical/functional correlates of each disease. These findings are valuable beyond the research setting, contributing directly to improving screening and accurate differential diagnosis in the early stages of both diseases.

Future lines of research

Future studies should consider using fMRI to comprehensively analyse semantic and phonological processing and to analyse connectivity patterns in brain areas responsible for language processing in AD and LBD. A final future challenge will be to promote studies that expand clinical diagnosis beyond neuropsychological symptoms and the pattern of atrophy, seeking to characterise the complete neuropsychiatric picture of each disease. Regarding the characteristics of psychoemotional interaction and social cognition, there is a need for deeper understanding of abilities related to the theory of mind, as few studies have examined the relationship between these processes and neuropsychiatric symptoms in early stages of dementia.[156,157]

Conclusion

This review identified the main similarities and differences in neuropsychological symptoms and patterns of cortical atrophy between patients with AD and LBD. Although the domains of memory, attention, executive function, visuospatial/visuoconstructive skills, and semantic and phonological verbal fluency are proposed as important cognitive markers, the level of severity at early stages seems to be key to the differential diagnosis of these 2 diseases. The pattern of fronto-parieto-temporal, hippocampal, and precuneus atrophy tends to overlap in both diseases. In this regard, the identification of functional alterations using fMRI would play an important role in early and preclinical stages of both neurodegenerative diseases.

Finally, language is increasingly recognised as another potential marker supporting differential diagnosis in AD. Nonetheless, assessment of spontaneous language should be promoted as a complementary resource for early, accurate differentiation between AD and LBD.

Funding

This study has received no specific funding from any public, private, or non-profit organisation.

Declaration of competing interest

None.

Acknowledgements

David Toloza-Ramirez thanks the Chilean National Agency for Research and Development (ANID)-Subdirección de Capital Humano/Doctorado Nacional/2021-21212181, and Teresa Julio-Ramos thanks ANID-Subdirección de Capital Humano/Doctorado Nacional/2023-21230591. The authors also thank Daniela Aguirre of the neuroscience masters programme at Universidad Autónoma de Chile, Temuco campus, for her support in the preliminary theoretical construction of this study.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi: https://doi.org/10.1016/j.nrleng.2025.10.001.

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