

# Distinguishing Alzheimer's disease from Parkinson's disease dementia using Enhanced Cued Recall test subscores

Ezgi Yetim<sup>1</sup>, Ezgi Demirel<sup>1</sup>, Ayse Akyay<sup>1</sup>, Gul Yalcin Cakmakli<sup>1</sup>, Esen Saka<sup>1</sup>

Department of Neurology, Hacettepe University Faculty of Medicine, Ankara, Türkiye

## ABSTRACT

**Objectives:** This study aimed to investigate the utility of differences in Enhanced Cued Recall (ECR) subscores in distinguishing cognitive impairment associated with Alzheimer's disease (AD) and Parkinson's disease (PD).

**Patients and methods:** The prospective study included 50 patients (34 females, 17 males; mean age: 71.3±8.6 years; range, 51 to 82 years) with AD and 25 patients (13 females, 12 males; mean age: 70.0±9.3 years; range, 53 to 93 years) with PD with associated cognitive impairment between January 2023 and June 2023. The counts of items within the free recall segments of the three ECR test trials were individually assessed and compared as repeated measurements between the respective groups. The difference between the number of items in the third and the first free recall trials was calculated and evaluated as a prospective predictive instrument.

**Results:** In addition to the total ECR score, the number of items in each of the three free recall trials was significantly lower in the AD group compared to the PD group. While these scores remained relatively consistent across trials in the AD group, the PD group progressively recalled more item names. The increasing positive difference between the third trial and the first trial, when subtracted, was found to be a significant predictor in favor of the PD group, with a sensitivity of 84% and specificity of 68%.

**Conclusion:** The examination of the trajectory of ECR test subscores, in addition to the total score, can be a useful method for the differential diagnosis of cognitive impairment associated with AD and PD.

**Keywords:** Alzheimer's disease, cognitive decline, enhanced cued recall test, Parkinson's disease.

Dementia is the leading cause of disability and dependency among the elderly. Among the spectrum of dementia, Alzheimer's disease (AD) emerges as the predominant primary neurodegenerative etiology, characterized by the accumulation of amyloid-beta and aberrantly phosphorylated microtubule-associated protein tau. On the other hand, Parkinson's disease (PD) is the second most common neurodegenerative disease following AD, in which patients suffer cognitive decline, depression, anxiety, sleep disorders, and dysautonomia in addition to the primary motor findings of the disease. The pathophysiological hallmark of the disease comprises Lewy bodies

and intracytoplasmic neuronal inclusions formed by phosphorylated alpha-synuclein protein.

From the perspective of cognition, typical cognitive manifestations characterizing AD encompass deficits in recent memory, challenges in visuospatial orientation, apraxia, and language dysfunction. In contrast, patients diagnosed with PD tend to exhibit a cognitive impairment more closely aligned with frontal-subcortical involvement. Consequently, cognitive profile in PD predominantly correlates with impairments in attention and executive functions, with a comparatively lesser degree of impact on memory and language domains.<sup>[1-4]</sup> Nevertheless, it is not uncommon

**Correspondence:** Ezgi Yetim, MD. Hacettepe Üniversitesi Tıp Fakültesi Nöroloji Anabilim Dalı, 06230 Altındağ, Ankara, Türkiye.

**E-mail:** ezgiyetim@hacettepe.edu.tr

**Received:** August 22, 2023 **Accepted:** December 27, 2024 **Published online:** March 05, 2025

**Cite this article as:** Yetim E, Demirel E, Akyay A, Yalcin Cakmakli G, Saka E. Distinguishing Alzheimer's disease from Parkinson's disease dementia using Enhanced Cued Recall test subscores. Turk J Neurol 2025;31(1):62-68. doi: 10.55697/tnd.2025.136.



to observe AD pathology that accompanies PD pathology. This scenario can pose a challenge in distinguishing between the two conditions during clinical evaluation. Various animal studies have demonstrated that Lewy-related pathology can stimulate the development of amyloid-beta plaques.<sup>[5,6]</sup> As a result of this neuropathological interaction, cognitive decline exhibited in PD has a heterogenous feature. Even in the early stages, some individuals with PD may exhibit prominent episodic memory deficits that mimic the clinical profile of AD.<sup>[7]</sup>

The differential diagnosis of dementia hinges upon clinical history, a detailed neurological examination focused on mental status, a standardized basic laboratory work-up, and structural brain imaging.<sup>[8]</sup> In this aspect, the clinical neuropsychological assessment serves as a pivotal tool for early dementia detection and differential diagnosis. Previous studies have employed numerous tests to distinguish between AD and PD. In one of them, commission errors on the experimental visual recognition task and commission errors on the word list recognition task differed between the two groups, indicating a more pronounced impact on episodic memory.<sup>[9]</sup> Additionally, a different study showed that patients with probable AD demonstrated a more significant decline in the three-word recall test than matched patients with PD.<sup>[10]</sup> Furthermore, a general cognitive assessment tool, the Dementia Rating Scale, was investigated in a separate study, demonstrating subcortical type cognitive impairment and relatively preserved memory functions in patients with PD.<sup>[11]</sup> The present study focused on the Enhanced Cued Recall (ECR) test, which evaluates episodic memory through the recall of 16 drawings using both free recall and cued recall methods, relying on the association of these drawings with semantic cues. A prior study showed its value as a screening test in the diagnosis of AD and mild cognitive impairment in the Turkish population.<sup>[12]</sup> Enhanced Cued Recall, beyond its potential for early dementia detection, displays variations in performance across different dementia types. Our group previously demonstrated a more pronounced memory impairment, as evaluated by ECR, in AD compared to PD-related dementia, despite similar Mini-Mental State Examination (MMSE) scores.<sup>[13]</sup> Although the total score of ECR was evaluated for its discriminatory potential between AD and PD-related cognitive impairment, there is a lack of studies that focus on the explicit assessment of ECR subscores obtained during each trial. In

this study, the aim was to investigate the utility of using differences in subscores of ECR to distinguish between patients with AD and those with PD.

## PATIENTS AND METHODS

The prospective study included 50 patients (34 females, 17 males; mean age: 71.3±8.6 years; range, 51 to 82 years) with AD and 25 patients (13 females, 12 males; mean age: 70.0±9.3 years; range, 53 to 93 years) with PD who were admitted to the Hacettepe University Faculty of Medicine, outpatient clinic of Movement Disorders and Behavioral Neurology, between January 2023 and June 2023. The diagnoses of AD were based on the National Institute on Aging-Alzheimer's Association diagnostic guidelines, and the diagnoses of PD were based on the Movement Disorder Society clinical diagnostic criteria.<sup>[14,15]</sup> The PD patients included in the study reported cognitive impairment that had a significant impact on their daily living activities without any other condition that might interfere with cognitive abilities. In addition, consecutive series of individuals with AD were included, unless any other condition that could potentially exacerbate cognitive impairment was identified. Neurological examination, MMSE, geriatric depression scale, clock drawing, ECR, reciting months forward and backward, letter fluency, semantic fluency (animals in 1 min), Stroop test, and digit span were administered to the whole group. Impairment of activities of daily living was assessed with Lawton and Brody's<sup>[16]</sup> Instrumental Activities of Daily Living Scale. Brain magnetic resonance imaging, complete blood count, blood biochemistry, thyroid function tests, and vitamin B12 levels were studied to exclude other causes of cognitive decline. The analyses were restricted to patients ≥50 years of age with a total MMSE score of 24 and lower in both groups. This prospective study was approved by the Hacettepe University Non-Interventional Clinical Research Ethics committee (date: 07.02.2023, no: GO 23/91). Written informed consent was obtained from all participants and/or legal guardians of the participants. The study was conducted in accordance with the principles of the Declaration of Helsinki.

The total score of MMSE was utilized in the analyses, and when necessary, an appropriate version for individuals with limited education was employed. A threshold of 24 was used to include participants in both groups. It is known that

cognitive decline in PD spares the memory domain at early stages. Therefore, any participant with PD who had an MMSE score greater than 24 was eliminated, despite the presence of cognitive impairment. This was done to specifically identify individuals with more pronounced memory impairment, which could then be compared to AD patients who were already anticipated to have memory difficulties.

Enhanced Cued Recall was administered and scored as previously presented by Saka et al.<sup>[12]</sup> Briefly, 16 black and white drawings in total in four different cards were presented to subjects one by one. While the subject was looking at the card, the examiner gave a unique semantic cue for every item and asked them to recognize the corresponding drawing. For example, one of the items on Card 1 was grapes. The examiner inquired, 'Can you identify the fruit on this card?' If the patient responded with 'grapes,' the examiner proceeded to the next item. However, if the patient failed to recognize 'grapes,' the examiner provided the correct answer, 'those are grapes,' and then moved on to the next item. Once all four items in the card were studied, the examiner removed the card and an immediate recall testing for those four items was performed by presenting the unique semantic cues. If the subject recalled

all of them correctly, the examiner proceeded to the next card containing another set of four items. In case the subject was unable to recall any of the items, the same card was subsequently presented once more, and the iterative process was repeated. Irrespective of the patient's persistent inability to recall any of the items, the card was not studied for a third time. Upon completion of all the cards, three recall trials were implemented, separated from each other either by counting the months forward and backward or clock drawing tests. In each recall trial, both free recall (without a semantic cue) and cued recall (with the presentation of semantic cues) were assessed. The total score was the sum of correctly recalled items in both free and cued recall trials, with a maximum total score of 48.

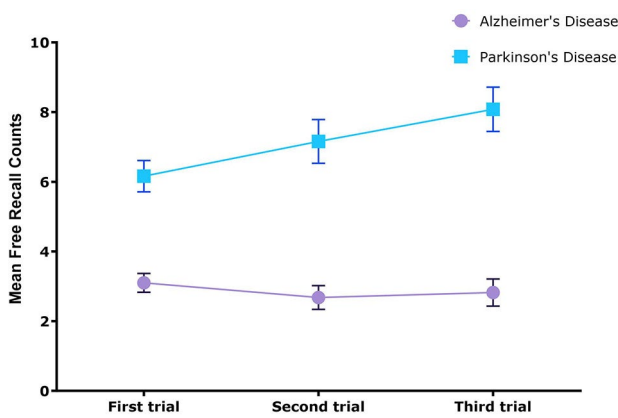
### Statistical analysis

Statistical analysis was performed using the IBM SPSS version 25.0 software (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as frequency (percentage) and continuous variables as mean  $\pm$  standard deviation (SD) or median (interquartile range [IQR]) depending on the distribution feature of the variable. Group-wise comparisons were performed by the chi-square test for categorical variables

**TABLE 1**  
Summary and comparison of demographic and clinical variables of the groups

	Alzheimer's disease (n=50)					Parkinson's disease (n=25)					p
	n	%	Mean $\pm$ SD	Median	IQR	n	%	Mean $\pm$ SD	Median	IQR	
Age (year)			71.3 $\pm$ 8.6					70.0 $\pm$ 9.3			0.818
Sex											
Female	34	68				13	52				0.180
Year of education			8.4 $\pm$ 4.9					5.6 $\pm$ 3.7			<0.05
Disease severity											
Mild	24	48						Hoehn & Yahr			
Moderate	18	36						2.9 $\pm$ 1.1			
Severe	8	16									
MMSE score			20.0 $\pm$ 3.4					21.6 $\pm$ 2.3			<0.05
Total ECR score				25	4-46				45	22-48	<0.001
First trial free recall				3	2.0-4.0				6	4.5-8.0	<0.001
Second trial free recall				2	0.8-4.3				6	5.0-9.5	<0.001
Third trial free recall				2	0.0-4.0				7	5.5-11.0	<0.001
The difference between third and first trial free recall counts				0	-1.3-1.0				2	1.0-3.0	<0.001

SD: Standard deviation; IQR: Interquartile range; MMSE: Mini-Mental State Examination; ECR: Enhanced Cued Recall. p-values denote statistical significance of group-wise comparisons conducted by chi-square test for categorical variables and by Independent Samples T-test or Mann-Whitney U test for continuous variables.



**Figure 1.** Mean free recall counts across three trials in the ECR test for patients with AD and PD. AD patients exhibited consistently lower recall performance across trials, with no significant improvement. In contrast, PD patients demonstrated better recall performance overall and showed progressive improvement across trials. Error bars represent standard deviations.

ECR: Enhanced Cued Recall; AD: Alzheimer's disease; PD: Parkinson's disease.

and the Mann-Whitney U test or independent samples t-test for continuous variables. Logistic regression models were constructed to determine independent factors related to distinguishing between AD and PD, and included the difference between the scores in the first and last free recall trials, patient age, years of education, and MMSE scores. A generalized linear mixed model was used to evaluate repeated measures through all three trials in ECR to test the difference between trajectories of performance in both groups. In this model, loglinear link was used with the linear model as the target had a Poisson distribution in which the target represents a

count of occurrences in a fixed period of time. A receiver operating characteristic (ROC) curve was generated to investigate the diagnostic yield of the difference between the third and first free recall trial counts to differentiate the two groups. The ROC curve was produced by plotting the true positive rate (sensitivity) against the false positive rate (100-specificity) at various cutoff points. Area under the curve (AUC), its 95% confidence intervals (CIs), and the standard error (SE) were calculated. A p-value <0.05 was considered statistically significant.

### RESULTS

The baseline characteristics of the cohort are summarized in Table 1. There was no significant difference in age (p=0.818) and sex distribution (p=0.180) between the two groups. When assessing disease severity through their respective scales, the PD group exhibited a mild to moderate clinical presentation as indicated by the Hoehn and Yahr score (2.9±1.1). Similarly, the AD group consisted mainly of patients in mild and moderate clinical stages, with 48% (n=24) classified as mild, 36% (n=18) classified as moderate, and 16% (n=8) classified as severe. Patients with PD had a lesser degree of education (5.6±3.7 vs. 8.4±4.9; p<0.05); however, MMSE test scores were significantly higher in the PD group compared to the AD group (21.6±2.3 vs. 20.0±3.4; p<0.05). Patients with AD, expectedly, had lower ECR scores either in total score or in each trial individually compared to patients with PD.

Figure 1 illustrates the mean values of the recalled item counts obtained from free recall trials of ECR in patients with AD and PD

**TABLE 2**  
Binary logistic regression analysis results

	Multivariate (Enter) <sup>1</sup>			Multivariate (Wald) <sup>2</sup>		
	OR	95% CI	p	OR	95% CI	p
Constant	0.169			1.000		
Age	0.982	0.912-1.058	0.630			
Year of education	0.825	0.713-0.954	<b>&lt;0.05</b>	0.829	0.720-0.954	<b>&lt;0.05</b>
MMSE score	1.156	0.909-1.470	0.238	0.844		0.763
The difference between the third and first trial	2.254	1.436-3.539	<b>&lt;0.001</b>	2.480	1.578-3.898	<b>&lt;0.001</b>

CI: Confidence interval; MMSE: Mini-Mental State Examination.

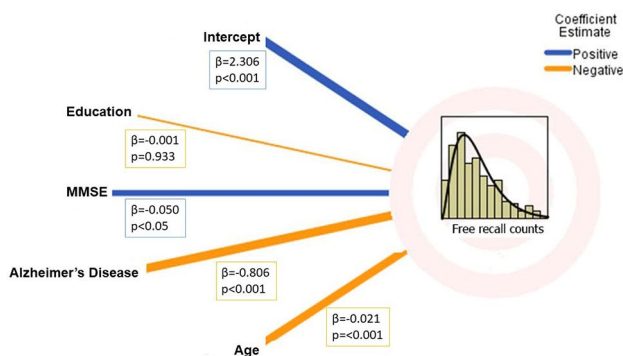
Dependent variable: Parkinson's disease (reference variable: Alzheimer's disease)

Year of education and the difference between the third and first free recall trial variables predict PD versus AD in both multivariate models utilizing Enter and Wald methods

<sup>1</sup>: Cox-Snell R<sup>2</sup>=0,367; Nagelkerke R<sup>2</sup>=0,510; Hosmer and Lemeshow Chi-square=4.170, p=0.760; Percentage correct=84%

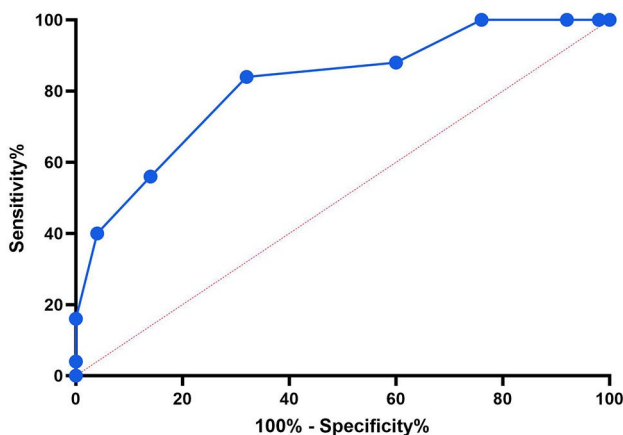
<sup>2</sup>: Cox-Snell R<sup>2</sup>=0,352; Nagelkerke R<sup>2</sup>=0,489; Hosmer and Lemeshow Chi-square=7.576, p=0.476; Percentage correct=83%

(3.1, 2.7, and 2.8 in the AD group; 6.2, 7.2, and 8.1 in the PD group, respectively). Patients with PD were more likely to show an increase across trials, whereas freely recalled item counts slightly differed between trials in patients with AD. In the multivariate analysis conducted for repeated measures, a significant difference was observed between the two groups, with this difference being associated with a more negative outcome in the AD group ( $\beta=-0.806$ ;  $p<0.001$ ) compared to the PD group, even after adjusting for age,



**Figure 2.** General linear model analysis predicting free recall counts across ECR test trials adjusted for age, education, MMSE score, and having AD diagnosis. Blue lines indicate positive effects, and orange lines indicate negative effects. The histogram inset represents the distribution of free recall counts.

ECR: Enhanced Cued Recall; MMSE: Mini-Mental State Examination; AD: Alzheimer's disease.



**Figure 3.** Receiver operating characteristic curve for the difference between the third and first free recall item counts in ECR test, assessing its ability to discriminate between AD and PD. The analysis demonstrated a high area under the curve (AUC=0.816, SE=0.052, 95% CI: 0.714-0.919,  $p<0.001$ ), indicating strong discriminatory power.

ECR: Enhanced Cued Recall; AD: Alzheimer's disease; PD: Parkinson's disease; SE: Standard error; CI: Confidence interval.

education, and MMSE score (Figure 2).

In logistic regression models, the prediction of PD versus AD was accomplished through multivariate models incorporating the variables of education (odds ratio [OR]=0.825, 95% CI: 0.713-0.954,  $p<0.05$ ) and the difference between the third and first trial free recall counts (OR=2.254, 95% CI: 1.436-3.539,  $p<0.001$ ; Table 2).

The ROC curve analysis demonstrated that the difference between the third and first free recalled item counts was useful for discrimination of AD and PD with a high AUC value (AUC=0.816, SE=0.052, 95% CI: 0.714-0.919,  $p<0.001$ ; Figure 3). With a cutoff value of one more item recalled correctly in the third trial compared to the first trial, cognitive impairment in PD could accurately be differentiated from AD with 84% sensitivity and 68% specificity.

## DISCUSSION

The current study investigated differences in the ECR test subscores between patients with AD and those with PD. Our findings underscored the importance of ECR in distinguishing between these two neurodegenerative conditions and shed light on the potential utility of specific subscores in this discrimination.

In accordance with prior literature, we observed that as a memory assessment test, ECR resulted in notably lower scores in patients with AD compared to those with PD. Saka et al.<sup>[12]</sup> conducted a comparison of ECR performance in amnesic mild cognitive impairment (MCI), AD, PD-MCI, and Parkinson's disease dementia (PD-D) patients with similar MMSE scores. Their findings revealed significantly higher ECR test scores in the PD-D group compared to the AD group. Additionally, the amnesic MCI group displayed a slightly higher performance than the PD-MCI group. Their investigations suggested that the total ECR score could be a valuable tool in distinguishing AD from PD-D and amnesic MCI from PD-MCI.<sup>[13]</sup> The current study added to these observations that patients with PD consistently achieved higher scores compared to patients with AD across all trials and significantly improved their recall counts across repetitive efforts. This outcome supports the widely accepted hypothesis that cognitive impairment in PD is characterized by a less prominent impact on memory consolidation than AD.<sup>[17,18]</sup> In this study, even though the patients with AD were of similar age and had a better level

of education, they exhibited poorer performance on the MMSE and ECR tests. This is because, although education has a protective effect, it is evident that in the clinical course of AD, cognitive function, particularly memory, is much more affected compared to PD. Prior studies suggested that PD patients were prone to benefit from cued recall resulting in similar total recall scores with controls, indicating that they were able to utilize cues for memory retrieval.<sup>[13,19]</sup> As a wide variety of studies all agreed, the progression of Lewy body pathology from subcortical to limbic areas paved the way for memory impairment in PD.<sup>[20-23]</sup> Several investigators studied recollection and familiarity, which are subcomponents of retrieval, to better understand episodic recognition memory deficits in PD. Most of these studies suggested an impaired recollection as the primarily responsible deficit, whereas familiarity did not differ from normal controls.<sup>[24-26]</sup> The nature of the ECR test involves semantic cues and repetition; therefore, it provides subjects with the opportunity to retrieve learned information. This feature helps to stratify episodic memory subcomponents and distinguish episodic memory impairment from attention deficit. In our study, patients with PD demonstrated an increasing ability to consolidate information with each trial, thereby getting familiar with a progressively higher number of items with each effort. Conversely, patients with AD were unable to consolidate information due to learning deficits, resulting in an absence of significant differences between trials. Moreover, the difference in the number of items correctly recalled between the third and first free recall trials can be a useful discriminator between AD and cognitive impairment in PD. This parameter yielded a promising sensitivity of 84% and specificity of 68% in distinguishing cognitive impairment in PD from AD. These results emphasized the practical utility of ECR as a valuable tool for clinical practitioners to aid in the differential diagnosis of these two prevalent neurodegenerative conditions.

To our knowledge, this study is one of the initial attempts to examine the individual subscores of ECR in the context of AD and PD. While previous studies have primarily focused on total ECR scores, analyzing subscores of each recall phase offers a nuanced perspective on episodic memory subcomponent patterns in these conditions. Our investigation highlighted the potential of these subscores in aiding discrimination between patients with AD and PD, demonstrating the intricate differences in their cognitive profiles.

In conclusion, the present study emphasized that the distinct patterns in free recall counts across the three trials in the ECR test contributed to the differentiation between AD and PD. Moreover, the notable link between the change in free recall counts from the first to the third trial and the diagnostic classification underscored its clinical relevance and potential as a useful diagnostic tool in clinical practice.

**Data Sharing Statement:** The data that support the findings of this study are available from the corresponding author upon reasonable request.

**Author Contributions:** Contributed to the study concept and design, and they also provided supervision: E.Y., G.Y.Ç., E.S.; Data collection and processing were conducted: E.Y., E.D., A.A.; Analysis and interpretation were performed, and they also contributed to the literature review: E.Y., E.D., G.Y.Ç.; Manuscript writing was carried out: E.Y., E.S., E.D., A.A.; Critical review was provided: E.S., G.Y.Ç., E.Y. All authors reviewed and approved the final version of the manuscript.

**Conflict of Interest:** The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

**Funding:** The authors received no financial support for the research and/or authorship of this article.

## REFERENCES

1. Stern Y, Richards M, Sano M, Mayeux R. Comparison of cognitive changes in patients with Alzheimer's and Parkinson's disease. *Arch Neurol* 1993;50:1040-5. doi: 10.1001/archneur.1993.00540100035011.
2. Cummings JL. The dementias of Parkinson's disease: Prevalence, characteristics, neurobiology, and comparison with dementia of the Alzheimer type. *Eur Neurol* 1988;28 Suppl 1:15-23.
3. Cahn-Weiner DA, Grace J, Ott BR, Fernandez HH, Friedman JH. Cognitive and behavioral features discriminate between Alzheimer's and Parkinson's disease. *Neuropsychiatry Neuropsychol Behav Neurol* 2002;15:79-87.
4. Bronnick K, Emre M, Lane R, Tekin S, Aarsland D. Profile of cognitive impairment in dementia associated with Parkinson's disease compared with Alzheimer's disease. *J Neurol Neurosurg Psychiatry* 2007;78:1064-8. doi: 10.1136/jnnp.2006.108076.
5. Braak H, Braak E, Yilmazer D, de Vos RA, Jansen EN, Bohl J. Pattern of brain destruction in Parkinson's and Alzheimer's diseases. *J Neural Transm (Vienna)* 1996;103:455-90. doi: 10.1007/BF01276421.
6. Clinton LK, Blurton-Jones M, Myczek K, Trojanowski JQ, LaFerla FM. Synergistic interactions between Abeta, tau, and alpha-synuclein: Acceleration of neuropathology and cognitive decline. *J Neurosci* 2010;30:7281-9. doi: 10.1523/JNEUROSCI.0490-10.2010.

7. Oltra-Cucarella J, Ferrer-Cascales R, Alegret M, Gasparini R, Díaz-Ortiz LM, Ríos R, et al. Risk of progression to Alzheimer's disease for different neuropsychological Mild Cognitive Impairment subtypes: A hierarchical meta-analysis of longitudinal studies. *Psychol Aging* 2018;33:1007-21. doi: 10.1037/pag0000294.
8. Gale SA, Acar D, Daffner KR. Dementia. *Am J Med* 2018;131:1161-9. doi: 10.1016/j.amjmed.2018.01.022.
9. Hildebrandt H, Fink F, Kastrup A, Haupts M, Eling P. Cognitive profiles of patients with mild cognitive impairment or dementia in Alzheimer's or Parkinson's disease. *Dement Geriatr Cogn Dis Extra* 2013;3:102-12.
10. Song IU, Kim JS, Yoo JY, Song HJ, Lee KS. Cognitive dysfunctions in mild Parkinson's disease dementia: Comparison with patients having mild Alzheimer's disease and normal controls. *Eur Neurol* 2008;59:49-54. doi: 10.1159/000109261.
11. Janvin CC, Larsen JP, Salmon DP, Galasko D, Hugdahl K, Aarsland D. Cognitive profiles of individual patients with Parkinson's disease and dementia: Comparison with dementia with lewy bodies and Alzheimer's disease. *Mov Disord* 2006;21:337-42. doi: 10.1002/mds.20726.
12. Saka E, Mihci E, Topcuoglu MA, Balkan S. Enhanced cued recall has a high utility as a screening test in the diagnosis of Alzheimer's disease and mild cognitive impairment in Turkish people. *Arch Clin Neuropsychol* 2006;21:745-51. doi: 10.1016/j.acn.2006.08.007.
13. Saka E, Elibol B. Enhanced cued recall and clock drawing test performances differ in Parkinson's and Alzheimer's disease-related cognitive dysfunction. *Parkinsonism Relat Disord* 2009;15:688-91. doi: 10.1016/j.parkreldis.2009.04.008.
14. McKhann GM, Knopman DS, Chertkow H, Hyman BT, Jack CR Jr, Kawas CH, et al. The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimers Dement* 2011;7:263-9. doi: 10.1016/j.jalz.2011.03.005.
15. Postuma RB, Berg D, Stern M, Poewe W, Olanow CW, Oertel W, et al. MDS clinical diagnostic criteria for Parkinson's disease. *Mov Disord* 2015;30:1591-601. doi: 10.1002/mds.26424.
16. Lawton MP, Brody EM. Assessment of older people: Self-maintaining and instrumental activities of daily living. *Gerontologist* 1969;9:179-86.
17. Siquier A, Andrés P. Episodic memory impairment in Parkinson's disease: Disentangling the role of encoding and retrieval. *J Int Neuropsychol Soc* 2021;27:261-9. doi: 10.1017/S1355617720000909.
18. Zokaei N, Sillence A, Kienast A, Drew D, Plant O, Slavkova E, et al. Different patterns of short-term memory deficit in Alzheimer's disease, Parkinson's disease and subjective cognitive impairment. *Cortex* 2020;132:41-50. doi: 10.1016/j.cortex.2020.06.016.
19. Grober E, Lipton RB, Hall C, Crystal H. Memory impairment on free and cued selective reminding predicts dementia. *Neurology* 2000;54:827-32. doi: 10.1212/wnl.54.4.827.
20. Hely MA, Morris JG, Traficante R, Reid WG, O'Sullivan DJ, Williamson PM. The Sydney Multicentre Study of Parkinson's disease: Progression and mortality at 10 years. *J Neurol Neurosurg Psychiatry* 1999;67:300-7. doi: 10.1136/jnnp.67.3.300.
21. Galvin JE, Uryu K, Lee VM, Trojanowski JQ. Axon pathology in Parkinson's disease and Lewy body dementia hippocampus contains alpha-, beta-, and gamma-synuclein. *Proc Natl Acad Sci U S A* 1999;96:13450-5. doi: 10.1073/pnas.96.23.13450.
22. Adamowicz DH, Roy S, Salmon DP, Galasko DR, Hansen LA, Masliah E, et al. Hippocampal  $\alpha$ -Synuclein in dementia with lewy bodies contributes to memory impairment and is consistent with spread of pathology. *J Neurosci* 2017;37:1675-84. doi: 10.1523/JNEUROSCI.3047-16.2016.
23. Das T, Hwang JJ, Poston KL. Episodic recognition memory and the hippocampus in Parkinson's disease: A review. *Cortex* 2019;113:191-209. doi: 10.1016/j.cortex.2018.11.021.
24. Edelstyn NM, Mayes AR, Condon L, Tunnicliffe M, Ellis SJ. Recognition, recollection, familiarity and executive function in medicated patients with moderate Parkinson's disease. *J Neuropsychol* 2007;1:131-47. doi: 10.1348/174866407x182565.
25. Algarabel S, Rodríguez LA, Escudero J, Fuentes M, Peset V, Pitarque A, et al. Recognition by familiarity is preserved in Parkinson's without dementia and Lewy-Body disease. *Neuropsychology* 2010;24:599-607. doi: 10.1037/a0019221.
26. Rodríguez LA, Algarabel S, Escudero J. Exploring recollection and familiarity impairments in Parkinson's disease. *J Clin Exp Neuropsychol* 2014;36:494-506. doi: 10.1080/13803395.2014.909386.