World Nutrition Journal |eISSN 2580-7013

ORIGINAL PAPER

Association of vitamin E intake and malondialdehyde (MDA) levels with cognitive function in elderly residing at the Budi Mulia Tresna Werdha nursing home, East DKI Jakarta

Ria Angelia Putri[1], Novi Silvia Hardiany[2], Erfi Prafiantini[1]

1. *Department of Nutrition, Faculty of Medicine, Universitas Indonesia*
2. *Department of Biochemistry and Molecular Biology, Faculty of Medicine, Universitas Indonesia*

Received 29 June 2026
Accepted 24 August 2026
Published 31 August 2026

Link to DOI:
10.25220/WNJ.V10.i1.0015

Citation: Putri R A, Hardiany N S, Prafiantini E. Association of vitamin E intake and malondialdehyde (MDA) levels with cognitive function in elderly residing at the Budi Mulia Tresna Werdha nursing home, East DKI Jakarta. World Nutrition Journal.2026 August 31,10(i1): 175-184.

Abstract

Background: Cognitive dysfunction is one of the health problems associated with elderly. Oxidative stress is thought to play a role in the neurodegenerative process through increased production of free radicals that cause nerve cell damage. Vitamin E is an antioxidant that protects cell membranes from oxidative damage, whereas malondialdehyde (MDA) is a biomarker of oxidative stress produced by lipid peroxidation. The objective of this study is to analyze the association of vitamin E intake and MDA levels with cognitive function among elderly residents of Budi Mulia 1 Nursing Home in DKI Jakarta, Indonesia.

Methods: This secondary study employed a cross-sectional design using data derived from a primary study investigating the association between zinc intake, superoxide dismutase activity, and cognitive function among elderly. A total of 85 participants met the inclusion and exclusion criteria. Vitamin E intake was assessed using a Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ), plasma MDA levels were measured from stored plasma samples, and cognitive function was evaluated using the Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina).

Results: The median age of the participants was 69 years (range: 60–91 years). The median vitamin E intake, plasma MDA level, and cognitive function score were 3.7 mg/day (2.5–3.8), 2.85 nmol/mL (1.88–6.77), and 16 (1–30), respectively. No significant association was found between vitamin E intake and cognitive function ($r = 0.027$; $p = 0.806$), nor between plasma MDA levels and cognitive function ($r = 0.096$; $p = 0.380$).

Conclusion: There was no significant association between vitamin E intake or plasma MDA levels and cognitive function among elderly.

Keywords: vitamin E, malondialdehyde, oxidative stress, cognitive function, elderly

Corresponding author:
Novi Silvia Hardiany
Department of Biochemistry and Molecular Biology, Faculty of Medicine, Universitas Indonesia
Email: novi.silvia@ui.ac.id, novish98@gmail.com

Introduction

Elderly represent one of the population groups experiencing the most significant demographic shift worldwide. According to Indonesian Law Number 13 of 1998 concerning the Welfare of Older Persons, elderly are individuals aged 60 years and above.[1] It is estimated that by 2030, one in six people globally will be aged 60 years or older, and the number of individuals aged 60 years and above is projected to reach 2.1 billion by 2050.[2] Indonesia is currently entering an ageing population period, characterized by approximately 12% of its population being classified as elderly in 2024.[3] In DKI Jakarta, the proportion of elderly has reached 10.64% of the total population.[4]

The growing older adult population presents both opportunities and challenges for national development. If elderly remain healthy and productive, this demographic transition may provide Indonesia with a second demographic dividend.[5] However, the increasing number of elderly is accompanied by a higher prevalence of cognitive decline, characterized by impairments in memory, attention, language, and problem-solving abilities.[6,7] Such decline may limit individuals' participation in family and social activities, ultimately increasing the burden on caregivers and society.[8]

Micronutrients, including vitamins, minerals, and antioxidant compounds found in food, such as vitamins A, C, D, and E, as well as selenium, have received considerable attention because of their potential role in neuroprotection and the maintenance of cognitive function.[9] Devarshi, et al.[10] reported that higher intakes of vitamins C, D, and E, folate, and carotenoids among elderly were associated with better cognitive performance, particularly in memory and executive function. In addition, Devore, et al.[11] found that adequate intakes of vitamins C and E were associated with a lower risk of cognitive decline and Alzheimer's disease over time.

Cognitive function and oxidative stress are closely related, as oxidative stress may adversely affect cognitive performance. Oxidative stress is defined as an imbalance between free radicals (pro-oxidants) and antioxidants that can lead to tissue damage. [12] One of the biomarkers of oxidative stress is malondialdehyde (MDA), the final product of cellular lipid peroxidation derived from polyunsaturated fatty acids. [13,14] Numerous studies have demonstrated that MDA is a stable and reliable indicator of lipid peroxidation and serves as an accurate biomarker of oxidative stress.[15]

Vitamin E is a potent antioxidant capable of donating a hydrogen ion from its hydroxyl (OH) group to neutralize free radicals. Through this mechanism, vitamin E terminates lipid peroxidation chain reactions, protects cells from oxidative damage, and helps reduce oxidative stress and inflammation associated with ageing and age-related diseases.[16,17] Power, et al.[13] reported that vitamin E supplementation may provide beneficial effects on working memory among elderly. Furthermore, Ortega, et al.[18] demonstrated an association between vitamin E status and cognitive function in elderly aged 65–91 years.

Previous research conducted using the same cohort demonstrated that cognitive impairment was present in 84.7% of elderly, while superoxide dismutase (SOD) activity explained 15.9% of the variation in cognitive function. However, no significant association was observed between zinc intake and either SOD activity or cognitive function among elderly.[19] Since the primary study focused on zinc intake and SOD activity, the potential contribution of vitamin E intake, a major lipid-soluble antioxidant, and malondialdehyde (MDA), a biomarker of lipid peroxidation and oxidative stress, to cognitive function remains unclear.

The present study is a secondary analysis derived from the same cohort but addresses a different scientific question by examining the association between vitamin E intake, plasma MDA levels, and cognitive function. Unlike the primary study, which investigated zinc intake and SOD activity,[19] this study evaluates dietary antioxidant intake together with a biomarker of oxidative stress, thereby providing complementary evidence regarding the potential role of antioxidant nutrition and lipid peroxidation in cognitive ageing. Consequently, this secondary analysis extends the findings of the primary study by providing additional scientific evidence on nutritional and oxidative stress-related factors associated with cognitive function among elderly.

Therefore, this study aimed to analyze the association between vitamin E intake and malondialdehyde (MDA) levels, as a biomarker of oxidative stress, and cognitive function among elderly.

Methods

This secondary study employed a cross-sectional design to analyze the association between vitamin E intake, malondialdehyde (MDA) levels, and cognitive function among elderly. Both primary and secondary data were utilized. The primary data consisted of stored biological samples obtained from the primary study, specifically blood plasma samples used for the measurement of plasma MDA levels. Secondary data were derived from the primary study and included participant characteristics, energy intake, vitamin E intake, cognitive function, and physical activity.

The primary study was conducted at the Budi Mulia 1 Nursing Home in DKI Jakarta, Indonesia, between July and September 2024. Measurement of plasma MDA levels was performed at the Department of Biochemistry and Molecular Biology Laboratory, Faculty of Medicine, Universitas Indonesia.

The target population comprised all residents of the Budi Mulia 1 Nursing Home who had been enrolled as participants in the primary study. Study participants were selected from the target population based on the primary study inclusion criteria and the availability of stored plasma samples that met the required quality standards. Total sampling was applied to all eligible participants from the primary study who fulfilled the inclusion and exclusion criteria.

The inclusion criteria were: (1) male or female residents aged ≥60 years who had participated in the primary study; (2) willingness to participate as indicated by written informed consent; and (3) availability of stored plasma samples of adequate quality, defined as samples that had been stored continuously at −20°C since collection (approximately one year), had not undergone repeated freeze–thaw cycles, had sufficient volume for laboratory analysis, and showed no visible hemolysis. The exclusion criteria were: (1) acute illness or severe pain at the time of the primary study; (2) coagulation disorders; (3) smoking or alcohol consumption within one year before the primary study; (4) refusal to participate; and (5) plasma samples that showed hemolysis, had not been consistently stored at −20°C, had undergone repeated freeze–thaw cycles, or had insufficient volume for MDA measurement.

A total of 96 elderly were initially screened for participation in the primary study. Of these, 86 fulfilled the eligibility criteria and provided written informed consent. During the study period, one participant withdrew because they permanently left the nursing home, resulting in a final sample of 85 participants included in the present secondary analysis. No missing data were identified for variables included in the present analysis; therefore, all 85 participants were included in the statistical analyses.

Vitamin E intake was assessed using the validated Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ). The SQ-FFQ was used to estimate habitual vitamin E intake based on the frequency and portion size of foods consumed. To improve the accuracy of dietary assessment, food photographs, food models, standard household measures, and the monthly nursing home menu were used during the interview. Daily nutrient intake was analyzed using NutriSurvey 2007 software, and information regarding the type, product, and daily dosage of dietary supplements was also recorded to estimate total nutrient intake.

Cognitive function was evaluated using the Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina), a validated cognitive screening instrument with a total score ranging from 0 to 30, where higher scores indicate better cognitive function. Physical activity was assessed using the Physical Activity Scale for the Elderly (PASE), which evaluates leisure, household, and occupational activities performed during the previous seven days. PASE score was categorized as inactive (<125) or active (≥125).

Educational level, history of chronic diseases, age, and sex were obtained through interviewer-administered questionnaires completed during the primary study. Anthropometric measurements, including body weight and height (or estimated height using knee height for participants unable to stand upright), were performed by trained research personnel following standardized procedures. Body mass index (BMI) was calculated as body weight (kg) divided by height squared (m^2).

Plasma MDA levels were measured using stored plasma samples using the Wills thiobarbituric acid reactive substances (TBARS) spectrophotometric method. Plasma samples were reacted with thiobarbituric acid (TBA), and absorbance was measured at of 530 nm. MDA concentrations were calculated using a standard calibration curve and expressed as nmol/mL.

Data analysis was performed using IBM Statistical Package for the Social Sciences (SPSS) version 29. Data normality was assessed using the Kolmogorov–Smirnov test. Normally distributed continuous variables were presented as mean ± standard deviation, whereas non-normally distributed variables were presented as median (minimum–maximum). Categorical variables were expressed as frequencies and percentages. The associations between vitamin E intake and cognitive function, as well as between plasma MDA levels and cognitive function, were analyzed using Spearman's rank correlation test. A p-value of less than 0.05 was considered statistically significant.

This secondary study received ethical approval from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Indonesia (Approval No. KET-496/UN2.F1/ETIK/ PPM.00.02/2026). Written informed consent had been obtained from all participants prior to their involvement in the study.

Results

A total of 85 participants were included in this study. Participant characteristics included age, sex, educational level, history of chronic diseases, body mass index (BMI), nutritional status, energy intake, energy intake status, fat intake, and physical activity as shown in **Table 1**. The data were obtained from our previous study.[19] The median age of the participants was 69 years (range: 60–91 years). The majority of participants were female (61.2%) and had a primary level of education (72.9%). Most participants had a history of chronic disease (87.1%), with hypertension being the most common condition (47.1%). The median BMI was 21 kg/m² (range: 17–35 kg/m²), and nearly half of the participants (49.4%) had normal nutritional status. The median energy intake was 1,928 kcal/day (range: 1,342–2,167 kcal/day), and most participants (76.5%) had adequate energy intake. In addition, the median fat intake was 64 g/day (range: 48–68 g/day). Based on physical activity level, the majority of participants (88.2%) were classified as physically inactive.

The median vitamin E intake among participants was 3.7 mg/day (range: 2.5–3.8 mg/day). Based on the Indonesian Recommended Dietary Allowance (RDA), all participants (100%) had inadequate vitamin E intake. The median cognitive function score was 16 (range: 1–30), and 72 participants (84.7%) were classified as having cognitive impairment. Vitamin E intake and cognitive function were obtained from our previous study.[19] Spearman's correlation analysis showed no significant association between vitamin E intake and cognitive function ($r = 0.027$; $p = 0.806$) as shown in **Table 2**.

Table 1. Characteristics of study participants (n = 85)

Variable	n%	Mean±SD / Median (min-max)
Age, *years*		69 (60-91)
Sex		
Male	33 (38.8)	
Female	52 (61.2)	
Educational level		
No formal education	13 (15.3)	
Primary education	62 (72.9)	
Secondary education	7 (8.2)	
Higher education	3 (3.5)	
History of chronic disease		
No	11 (12.9)	
Yes	74 (87.1)	
Schizophrenia	29 (34.1)	
Asthma	3 (3.5)	
Hypertension	40 (47.1)	
Hyperuricemia	21 (24.7)	
Dyslipidaemia	6 (7.1)	
Diabetes mellitus	7 (8.2)	
Gastritis	1 (1.2)	
Vertigo	1 (1.2)	
Parkinson's disease	1 (1.2)	
Dermatitis	1 (1.2)	
Heart disease	1 (1.2)	
Body mass index, kg/m^2		21 (17-35)
Nutritional status		
Underweight	19 (22.4)	
Normal	42 (49.4)	
Overweight	9 (10.6)	
Obese	15 (17.6)	
Energy intake, kcal/day		1928 (1342-2167)
Energy intake status		
Inadequate	20 (23.5)	
Adequate	65 (76.5)	
Fat intake, g/day		64 (48-68)
Physical activity		
Inactive	75 (88.2)	
Active	10 (11.8)	

**Data are presented as median (minimum–maximum).*

Table 2. Association between vitamin E intake and cognitive function

Variable	Cognitive function	
	r	p-value
Vitamin E intake, *mg/day*	0.027	0.806

r = correlation coefficient; p-value = level of statistical significance (p < 0.05); Spearman's rank correlation test.

The median plasma MDA level among participants was 2.85 nmol/mL (1.88–6.77 nmol/mL), while the median cognitive function score was 16 (range: 1–30). Spearman's correlation analysis demonstrated no significant association between plasma MDA levels and cognitive function (r = 0.096; p = 0.380) as shown in **Table 3**.

Table 3. Association between plasma malondialdehyde (MDA) levels and cognitive function

Variable	Cognitive function	
	r	p-value
MDA, *nmol/ml*	0.096	0.380

MDA = malondialdehyde; r = correlation coefficient; p-value = level of statistical significance (p < 0.05); Spearman's rank correlation test.

Discussion

This study found no significant association between vitamin E intake and cognitive function, as measured by the Indonesian version of the Montreal Cognitive Assessment (MoCA-Ina), among elderly (r = 0.027; p = 0.806). This finding is consistent with the study conducted by Hartati, which reported no significant association between serum vitamin E levels and cognitive function among elderly in Indonesia.[20] Theoretically, vitamin E is the primary lipid-soluble antioxidant that protects cell membranes from free radical-induced damage.[21] In the central nervous system, vitamin E inhibits lipid peroxidation in neuronal membranes, thereby helping to maintain neuronal integrity and function.[16,17] Long-term vitamin E deficiency may increase neuronal susceptibility to oxidative damage, potentially accelerating neurodegenerative processes and cognitive decline.[17,21]

The present findings are also in agreement with those reported by Cetin, et al.,[22] who demonstrated that increased vitamin E levels following supplementation were not accompanied by greater improvements in cognitive function compared with the control group. Similarly, a systematic review by Farina, et al.,[23] concluded that vitamin E supplementation did not significantly improve cognitive performance or reduce the risk of progression from mild cognitive impairment to dementia. Furthermore, Grundman reported that vitamin E has not been proven effective in preventing the progression of mild cognitive impairment to Alzheimer's disease.,[24] These findings suggest that cognitive function in elderly is a multifactorial condition influenced by various factors, including physical activity, health status, educational attainment, chronic diseases, genetic factors, and oxidative stress levels.

In contrast, the findings of this study differ from those reported by Ortega, et al.,[18] who found that higher vitamin E status was associated with better cognitive function among elderly. This discrepancy may be explained by differences in the methods used to assess vitamin E status. In the present study, vitamin E intake was estimated from dietary intake using a Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ), which may not accurately reflect actual vitamin E status in the body because it is influenced by absorption, metabolism, distribution, and storage processes.[18,25] Furthermore, the median vitamin E intake of participants was only 3.7 mg/day, substantially lower than the recommended dietary allowance for elderly, which is 15 mg/day.[26]

This study also found no significant association between plasma malondialdehyde (MDA) levels and cognitive function among elderly (r = 0.096; p = 0.380). The median plasma MDA concentration was 2.85 nmol/mL, with values ranging from 1.88 to 6.77 nmol/mL. MDA is a final product of lipid peroxidation and is widely used as a biomarker of oxidative stress. Elevated MDA levels reflect increased oxidative damage within the body and are thought to contribute to neuronal membrane damage, impaired cellular function, and accelerated neurodegenerative processes associated with cognitive decline.[27-29] Nevertheless, the relationship between MDA levels and cognitive function has not been consistently demonstrated across studies, as cognitive performance is influenced by multiple factors, including age, sex, chronic diseases, nutritional status, antioxidant capacity, and genetic predisposition.[28]

The present findings are consistent with those of Abdul Sani, et al.,[30] who reported no significant association between MDA levels and cognitive function among elderly. In contrast, Papathanasiou, et al.,[29] found that individuals with higher MDA levels tended to have poorer cognitive performance or more severe cognitive impairment. In the current study, most participants had a history of chronic diseases (87.1%), low educational attainment (72.9% with primary education), and low levels of physical activity (88.2%), all of which may influence cognitive function through mechanisms that are not necessarily directly related to MDA levels.

This study has several limitations. First, the cross-sectional design only allows assessment of associations between variables at a single point in time and therefore cannot establish causal relationships. Second, vitamin E intake was assessed using a Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ), which relies on participants' ability to recall their habitual dietary intake and may therefore be subject to recall bias. Third, the study evaluated vitamin E intake solely from dietary sources and did not measure circulating vitamin E concentrations, which may better reflect vitamin E status in the body. Finally, oxidative stress was assessed using plasma MDA as a single biomarker, which may not fully represent the overall oxidative stress burden and antioxidant defense system.

Conclusion

The findings showed no significant association between vitamin E intake and cognitive function among elderly (r = 0.027; p = 0.806). Likewise, no significant association was observed between plasma MDA levels and cognitive function among elderly (r = 0.096; p = 0.380). These results indicate that vitamin E intake and plasma MDA levels were not associated with cognitive function among elderly residing at the Budi Mulia 1 Nursing Home in DKI Jakarta, Indonesia.

Conflict of interest

The authors declared no conflict of interest regarding this study.

Acknowledgement

The authors would like to express their sincere gratitude to all study participants for their valuable contribution to this research. The authors also thank the Budi Mulia 1 Nursing Home in DKI Jakarta, Indonesia, for granting permission and providing support throughout the study.

Appreciation is also extended to the Department of Biochemistry and Molecular Biology, Faculty of Medicine, Universitas Indonesia, for providing laboratory facilities used in this research. Furthermore, the authors acknowledge the research team of the primary study entitled "*The Correlation Between Zinc Intake and Superoxide Dismutase Activity with Cognitive Function in The Elderly*", which served as the data source for the present study.

Open Access

Author Contributions

All authors contributed equally to the study conception and design, data acquisition, analysis and interpretation, manuscript drafting and critical revision, and final approval of the manuscript. All authors reviewed and approved the final version of the manuscript.

References

1. Undang-Undang Republik Indonesia Nomor 13 Tahun 1998 tentang Kesejahteraan Lanjut Usia.
2. World Health Organization. Ageing and health [Internet]. Geneva: World Health Organization; [cited 2025 Feb]. Available from: https://www.who.int/news-room/fact-sheets/detail/ageing-and-health.
3. Sari NR, Nugroho SW, Sulistyowati R, Agustina R, Yulianto KT, Anggraeni G. *Statistik Penduduk Lanjut Usia 2024*. Vol. 21. Jakarta: Badan Pusat Statistik; 2024.
4. Badan Pusat Statistik Provinsi DKI Jakarta. *Jumlah lembaga pelayanan kesejahteraan sosial milik masyarakat/swasta dan binaan menurut jenis lembaga di Provinsi DKI Jakarta*, 2018–2021 [Internet]. Jakarta: BPS Provinsi DKI Jakarta; [cited 2025 Feb]. Available from: https://jakarta.bps.go.id/id/statistics-table/2/NjE4IzI=/jumlah-lembaga-pelayanan.
5. Wahyudi MZ. *Siapkah menyongsong era lansia*? [Internet]. Jakarta: Kompas; 2019 [cited 2025 Feb]. Available from: https://interaktif.kompas.id/baca/siapkah-menyongsong-era-lansia.
6. Reynolds CF 3rd, Jeste DV, Sachdev PS, Blazer DG. Mental health care for older adults: recent advances and new directions in clinical practice and research. World Psychiatry. 2022; 21(3): 336–363.
7. Kirova AM, Bays RB, Lagalwar S. Working memory and executive function decline across normal aging, mild cognitive impairment, and Alzheimer's disease. Biomed Res Int. 2015;2015:1-9.
8. Rekawati E, Eriska W, Rachmawati U, Wati DNK, Sahar J, Andriyanti A, et al. Cognitive function and its determinants in elderly Indonesians residing in long-term care: Insights from a cross-sectional study. F1000Research. 2024, 13:1384.
9. Akhgarjand C, Hashemi R, Amini M, Rasekhi H, Farazandeh D, Etesam F, et al., The relationship between micronutrients and cognitive ability in an elderly population with mild cognitive impairment and Alzheimer's disease: a cross-sectional study. BMC Neurol. 2024;24:416.
10. Devarshi PP, Gustafson K, Grant RW, Mitmesser SH. Higher intake of certain nutrients among older adults is associated with better cognitive function: an analysis of NHANES 2011–2014. BMC Nutr. 2023;9(1):142.

11. Devore EE, Grodstein F, van Rooij FJA, Hofman A, Stampfer MJ, Witteman JCM, et al. Dietary antioxidants and long-term risk of dementia. Arch Neurol. 2010;67(7):819-825.
12. Ayudiah RU, Dinda TS, Felly T. *Stres oksidatif dan penyakitnya* [Internet]. 2023 Jan [cited 2025 Feb]. Available from: https://www.researchgate.net/publication/366903090
13. Power R, Nolan JM, Prado-Cabrero A, Roche W, Coen R, Power T, et al. Omega-3 fatty acid, carotenoid and vitamin E supplementation improves working memory in older adults: a randomised clinical trial. Clin Nutr. 2022;41(2):405-414.
14. Bergin P, Leggett A, Cardwell CR, Woodside JV, Thakkinstian A, Maxwell AP, et al. The effects of vitamin E supplementation on malondialdehyde as a biomarker of oxidative stress in haemodialysis patients: a systematic review and meta-analysis. BMC Nephrol. 2021;22(1):126.
15. Ayala A, Muñoz M, Argüelles S. Lipid peroxidation: production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal. Oxid Med Cell Longev. 2014;2014:360438.
16. Vasei FM, Zamanian MY, Golmohammadi M, Mahmoodi M, Khademalhosseini M, Tavakoli T, et al. The impact of vitamin E supplementation on oxidative stress, cognitive functions, and aging-related gene expression in aged mice. Food Sci Nutr. 2024;12:9834-9845.
17. Gropper SS, Smith JL, Carr TP. *Advanced Nutrition and Human Metabolism*. 8th ed. Boston (MA): Cengage Learning; 2022.
18. Ortega RM, Requejo AM, López-Sobaler AM, Andrés P, Navia B, Perea JM, et al. Cognitive function in elderly people is influenced by vitamin E status. J Nutr. 2002;132(7):2065-2068.
19. Khuzaimah R, Witjaksono F, Hardiany NS. The correlation between zinc intake and superoxide dismutase activity with cognitive function in the elderly. Sultan Qaboos Univ Med J. 2025;25(1):506-514. doi:10.18295/2075-0528.2866.
20. Hartati NS. Hubungan kadar vitamin E serum dengan fungsi kognitif pada lanjut usia [tesis]. Jakarta: Universitas Indonesia; 2015.
21. Rolfes SR, Pinna K, Whitney E. Understanding normal and clinical nutrition. 10th ed. Belmont (CA): Wadsworth; 2015.
22. Çetin E, Top EC, Şahin G, Ozkaya YG, Aydin H, Toraman F. Effect of vitamin E supplementation with exercise on cognitive functions and total antioxidant capacity in older people. J Nutr Health Aging. 2010;14(9):763-9. doi:10.1007/s12603-010-0256-x.
23. Farina N, Llewellyn D, Isaac MGEKN, Tabet N. Vitamin E for Alzheimer's dementia and mild cognitive impairment. Cochrane Database Syst Rev. 2017;4(4):CD002854. doi:10.1002/14651858.CD002854.pub5.
24. Grundman M. Vitamin E and Alzheimer disease: the basis for additional clinical trials. Am J Clin Nutr. 2000;71(2 Suppl):630S-636S. doi:10.1093/ajcn/71.2.630S.
25. Zingg JM. Vitamin E [Internet]. In: Gibson RS, editor. Principles of nutritional assessment. 3rd ed. Dunedin. University of Otago; 2026 [cited 2026 Jun]. Available from: https://nutritionalassessment.org/vitamine/.
26. Kementerian Kesehatan Republik Indonesia. Peraturan Menteri Kesehatan Republik Indonesia Nomor 28 Tahun 2019 tentang Angka Kecukupan Gizi yang dianjurkan untuk Masyarakat Indonesia. Jakarta: Kementerian Kesehatan Republik Indonesia; 2019.
27. Cordiano R, Di Gioacchino M, Mangifesta R, Panzera C, Gangemi S, Minciullo PL. Malondialdehyde as a potential oxidative stress marker for allergy-oriented diseases: an update. Molecules. 2023;28(16):5979. doi:10.3390/molecules28165979.
28. Ioannidou S, Tsolaki M, Ginoudis A, Tamvakis A, Tsolaki A, Makedou K, et al. Oxidative stress biomarkers as preclinical markers of mild cognitive impairment: the impact of age and sex. J Pers Med. 2025;15(5):171. doi:10.3390/jpm15050171.
29. Papathanasiou IV, Fradelos EC, Malli F, Stefanidis I, Zintzaras E, Doxani C. A systematic review of observational studies assessing the impact of oxidative stress in cognitive decline. Wiad Lek. 2021;74(8):1995-2003.
30. Abdul Sani NF, Ahmad Damanhuri HA, Amir Hamzah AIZ, Abu Bakar ZH, Tan JK, Noor Aripin KN, et al. DNA damage and protein oxidation associated with ageing correlate with

cognitive dysfunction in a Malaysian population. Free Radic Res. 2018;52(9):1000-1009. doi:10.1080/10715762.2018.1504300.