Journal of Carcinogenesis
Covering Genesis to Therapeutics

Journal of Carcinogenesis, ISSN: 1477-3163
2025; Vol 24: Issue 5s, https://carcinogenesis.com/

Positive Effects of Camel's Milk for Alzheimer's Patients

Faris Q.B.Alenzi[1]

[1]Department of Medical Laboratory Sciences, College of Applied Medical Sciences, Prince Sattam bin Abdulaziz University (PSAU), Alkharj, Saudi Arabia

***Corresponding Author:**

Faris Q. Alenzi

Email ID: f.alenzi@psau.edu.sa

ABSTRACT

Background: Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline, memory loss, and impaired daily functioning. In recent years, a growing interest has developed in finding natural and dietary interventions for AD. The present study aimed to assess the effects of camel milk on patients with AD.

Methods: A case-control, hospital-based study design was used for this study. The study was conducted in Al azhar Faculty of Medicine, Cario, Egypt between June 2011- June 2012. Patients diagnosed as AD (clinically probable)-according to the criteria of the National Institute of Neurological and Communication Disorders and AD and Related Disorders Association (NINCDS/AD RDA), with Hachiniski Ischemic Score (HIS)<4 were included in the treatment group. All subjects were given the Mini-Mental State Examination (MMSE) to assess cognitive functions.

Results: A total of 40 participants were included in the study, with 15 (37.5%) participants in the control group and 25 (62.5%) participants in treatment group. The male and female ratio in the study was 1:1. The mean age in the control group was 67±5.06 and 70.7±5.38 in the treatment group. There was a significant difference in serum parameters between the control and treatment groups prior to the treatment. After treatment, amyloid beta 42, HSP 90, MDA, nitric oxide, IL-6, cholesterol, noradrenaline, and dopamine levels showed a significant decrease (P=0.000 for all). However, GSH, total antioxidants, acetylcholine, and serotonin levels were significantly improved after treatment (P=0.000 for all). There was a significant increase in MMSE score after treatment (15.28±5.78 vs. 26±9.85; P=0.000).

Conclusion: The findings of the present study showed that camel's milk has a significant positive impact on patients with AD, regarding improvement in laboratory parameters and cognitive functions.

Keywords: *Cognitive function, amyloid beta 42, Heat shock proteins, Neurodegenerative disorder*

How to Cite: Faris Q.B.Alenzi, (2025) Positive Effects of Camel's Milk for Alzheimer's Patients, *Journal of Carcinogenesis*, *Vol.24, No.5s*, 284-292

1. INTRODUCTION

The global prevalence of dementia is on the rise, with approximately 50 million people suffering from this debilitating condition. Among these, about two-thirds suffer from Alzheimer's disease (AD) (1). Some estimates suggest that by 2050, the number of people with dementia will rise to 150 million. In low- and middle-income countries, the projected rise is expected to be 68% by 2050 (2). AD is a progressive neurodegenerative disorder characterized by cognitive decline, memory loss, and impaired daily functioning. The disease was first reported in 1906 by Alois Alzheimer when he found amyloid plaques in the brain of a patient who suffered from memory loss prior to his death (3). The pathology of AD is multifactorial, involving amyloid-beta (Aβ) plaque deposition, neurofibrillary tangles composed of hyperphosphorylated tau protein, oxidative stress, and neuroinflammation. Most of the pathological changes are seen in the hippocampus and cerebral cortex region. These changes result in synapse loss and neuronal atrophy (4). The symptoms of AD vary depending on the stage of the disease; however, there is continuum of symptoms which ultimately culminate in limiting the performance of daily activities of affected individuals. The duration of AD symptoms depends on age, gender, and genetics of an individual (5).

The pathogenesis of AD involves several metabolic and biochemical alterations. Dysregulation of biochemical markers such as amyloid-beta (Aβ42) (6), heat shock protein 90 (HSP90) (7), malondialdehyde (MDA), glutathione (GSH), nitric oxide (NO) (8), total oxidant status, and interleukin-6 (IL-6) have been implicated in AD (9). Due to the multifactorial

pathology of AD, the management of AD is quite complicated. No definitive treatment exists so far and the symptomatic treatments remain the main approach in clinical practice. However, research is focused on finding treatments that can slow down or halt the progression of the disease. Two categories of drugs including cholinesterase inhibitors and partial N-methyl D-aspartate (NMDA) antagonists have been approved for the treatment of AD. Due to the lack of available treatment options, there has been growing interest in exploring natural and dietary interventions for AD. The interesting aspect of these nutraceuticals is that they exert little adverse effects on humans irrespective of their potential benefits. For example, olive and its phenolic compounds have demonstrated neuroprotective effects (10). Similarly, curcumin is found in turmeric which belongs to ginger family. Turmeric has been used for centuries as a staple of oriental medicine in Indian countries. As the prevelance of AD is lower in Indian countries, it is believed that diet rich in curcumin has beneficial effect on AD (11).

Camel milk has also shown potential neuroprotective and therapeutic benefits. The bioactive compounds found in camel milk are the main source of its therapeutic effects. Camel's milk is also enriched with immunoglobulins, lysozyme, lactoferrin, and peptidoglycan recognition protein (PGRP) (12). Lactoferrin has antibacterial, antiviral, immunomodulatory, and anti-inflammatory properties. Antibodies present in camel milk can provide beneficial effects in autoimmune diseases like AD and multiple sclerosis (13). Camel milk has been used in the treatment of autism spectrum disorder (ASD), Parkinson's disease, and AD (14). A study by Khatoon et al. reported that patients treated with camel milk demonstrated significant seizure protection including delay in seizure onset, decrease in conclusion duration and mortality rates ($P \leq 0.001$ for all) (15). Camel milk has also been associated with the modulation of biogenic amines (16).

Oxidative stress has been implicated in the pathology of several diseases including diabetes, atherosclerosis, rheumatoid arthritis, and AD (17). Due to the role of oxidative stress in AD, the need for naturally occurring antioxidants in the treatment becomes crucial. Currently, there is a paucity of clinical evidence on the efficacy of camel's milk in the treatment of AD and most of the findings are from animal studies. For example, research involving animal models has indicated that camel's milk can improve cognitive function and reduce the levels of amyloid-beta peptides, which are implicated in the development of Alzheimer's pathology (18). Most studies to date have been conducted in animal models, and human clinical trials are needed to assess the efficacy and safety of camel's milk consumption in individuals with AD. Therefore, the present study aimed to investigate the potential impact of camel's milk on patients with AD.

2. SUBJECTS AND METHODS

2.1 Study Questions:

Is there a biochemical profile disturbance in the AD patients? Is there a correlation between these biochemical markers, and severity of cognitive function? Could these cognitive functions improve after ingestion of camel milk?

2.2 Study Design:

A case-control, hospital based design was used to investigate the current research problem.

2.3 Study Setting:

This study was conducted in the Department of Pathology *Al azhar Faculty of Medicine, Cario, Egypt*

2.4 Study Sample:

A total of 80 patients of both sexes were included in our study, diagnosed as AD (clinically probable)-according to the criteria of National Institute of Neurological and Communication Disorders and AD and Related Disorders Association (NINCDS/AD RDA), with Hachiniski Ischemic Score (HIS)<4 . 30 healthy subjects' age and sex matched served as controls.All subjects were screened by medical history, general and neurological examination, electrocardiogram, Magnetic resonance imaging (MRI) and laboratory evaluation of serum electrolytes, glucose, renal and hepatic functions, complete blood count and thyroid functions studies. All subjects were given the Mini Mental State Examination (MMSE) to assess cognitive functions. The assigned scores ranging from 30 (for normal function) down to 5 (in case of severe dementia). These assessments were further supported by tests of cognitive function in accord with the Global Deterioration Scale (GDS) and the Clinical Dementia Rating (CDR).

All the AD patients must be fulfilling the following inclusion criteria: 1) Age of patients 60-70 years, 2) Patients were divided into 3 groups regarding severity of dementia according to the MMSE. Group I: included mild cases where MMSE ranged from 19-23.Group II: included moderate cases ranging from 10-19. Group III: included severe cases with MMSE <10. Also, all patients recruited in this study have fulfilled the following specific exclusion criteria: 1) Past or present major psychiatric disorders, 2) Neurological diseases other than AD, 3) Serious head trauma, 4) CNS infections, 5) B12 deficiency, 6) Liver disease, 7) Kidney disease, and 8) Hypoxia. 20 patients start medical treatments for 3 months. After three months of treatment they were observed for their cognitive functions by MMSE test and tested for of Aβ42, HSP 90,

MDA GSH, Nitric oxide, Total Oxidant, IL6, Cholesterol ,Noradrenalin, Acetylcholine, serotonin and Dopamine . The Remaining 20 patients ingest camel milk 200 ml in the morning and 200 ml in the evening for 3 successive months. After three months of ingestion of camel milk daily, they were observed for their cognitive functions by MMSE test and tested for Aβ42, HSP 90, MDA GSH, Nitric oxide Total Oxidant, IL6, Cholesterol ,Noradrenalin, Acetylcholine, serotonin and Dopamine .

The controls enrolled in the study have fulfilled the following inclusion criteria: 1) Age 60-70 years, 2) Have no history of Neurological diseases or CNS infection, and 3) Result Of MMSE > 23, and 4) Have fulfilled the same specific exclusion criteria used for the patients group.

2.5 Ethical Consideration:

The purpose of the study and procedures to be performed were explained to the cases and controls with an oral consent to participate in the study were taken accordingly.

2.6 Laboratory investigations:

Laboratory examinations were done for the AD cases and controls. Fasting venous blood samples, 10 ml, were taken for laboratory examinations. These examinations were Aβ42, HSP 90, MDA GSH, Nitric oxide Total Oxidant, IL6, Cholesterol, Noradrenalin, Acetylcholine, serotonin and Dopamine.

3. RESULTS

A total of 40 participants were included in the study, with a ratio of 1:1 between males and females. A total of 15 (37.5%) participants were included in the control group whereas 25 (62.5%) were included in the patient group (Table 1 and Figure 1).

Table 1. Sex distribution between controls and patients

Sex	Controls	Patients
Male	7 (46.7 %)	13(52.0 %)
Female	8 (53.3 %)	12(48.0 %)
Total	15 (100.0 %)	25 (100.0 %)

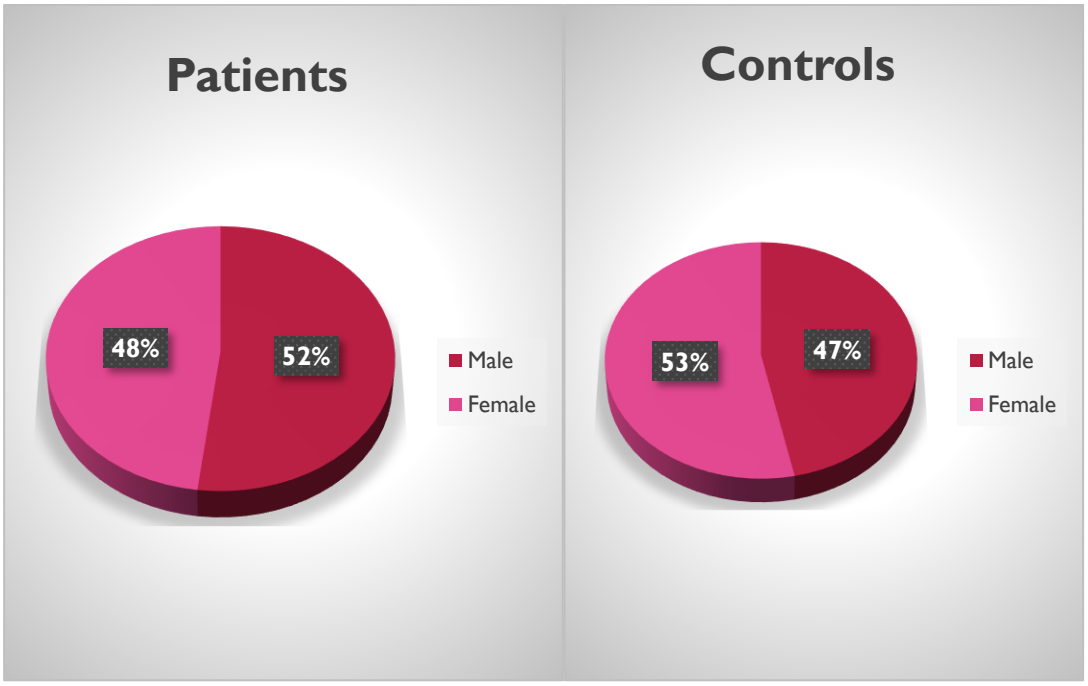

Figure 1. Sex distribution between controls and patients

Table 2 shows the relationship between the control and patient groups. The control group had a mean age of 67±5.06 years, whereas the patient group had a slightly higher mean age of 70.7±5.38 years. The difference between the two groups was statistically significant (P=0.038).

Table 2. Relation between the age of the controls and patients

	Mean±SD	t- test value	P- value
Age (control)	67±5.06	2.146	0.038
Age (patients	70.7±5.38		

Table 3 presents a comparative analysis of various serum parameters between control subjects and patients before treatment. All measured parameters showed significant differences between the two groups, with p-values less than 0.001.

Table 3: Relation of serum parameters of the controls and patients before treatment

Parameters		Mean±SD	t- test value	P- value
Amyloid beta 42 pg/ ml	Controls	20.48±2.11	25.770	0.000
	Pt before tt	130.36±16.34		
HSP 90 Pg/ml	Controls	71.43±10.73	18.288	0.000
	Pt before tt	195.4±24.70		
MDA nmol/ml	Controls	18.62±3.14	18.571	0.000
	Pt before tt	114.07±18.4		
GSH µmol/ml	Controls	12.7±1.86	18.763	0.000
	Pt before tt	4.4±1.024		
Nitric oxide µmol/ml	Controls	59.6± 9.1	13.823	0.000
	Pt before tt	177.2±23.3		
Total anti-oxidant µM/ml	Controls	88.4333±14.4	21.309	0.000
	Pt before tt	28.5±5.3		
IL -6 Pg/ml	Controls	7.51±1.34	16.221	0.000
	Pt before tt	34.71±7.51		
Chol .mg/dl	Controls	163.9±11.95	28.591	0.000
	Pt before tt	249.6±12.5		
Noradrenaline nmol/ml	Controls	17.8±2.741	24.044	0.000
	Pt before tt	76.12±13.67		
Acetylcholine ng/ml	Controls	42.8±5.7	16.241	0.000
	Pt before tt	8.4±1.4		
Serotonin ng/ml	Controls	63.94±9.96	25.770	0.000
	Pt before tt	14.66±2.11		
Dopamine ng/ml	Controls	19.4±2.27	18.352	0.000
	Pt before	76.26±13.37		

Amyloid beta 42, HSP 90, MDA, nitric oxide, IL-6, cholesterol, noradrenaline, and dopamine levels showed a significant decrease after treatment (P=0.000 for all). Conversely, GSH, total antioxidants, acetylcholine, and serotonin levels were significantly increased after treatment (P=0.000 for all) (Table 4).

Table 4: Relation of serum parameters of the patients before and after treatment

		Mean±SD	t- test value	P- value
Amyloid beta 42 pg/ ml	Pt before tt	130.36±16.34	17.326	0.000
	Pt after tt	61.4±11.34		
HSP 90 Pg/ml	Pt before tt	195.4±24.70	17.820	0.000
	Pt after tt	99.7±10.48		
MDA nmol/ml	Pt before tt	114.07±18.4	11.87	0.000
	Pt after tt	58.7±14.2		
GSH µmol/ml	Pt before tt	4.4±1.024	-11.07	0.000
	Pt after tt	8.45±1.5		
Nitric oxide µmol/ml	Pt before tt	177.2±23.3	12.48	0.000
	Pt after tt	107.6±15.1		
Total anti-oxidant µM/ml	Pt before tt	28.5±5.3	12.25	0.000
	Pt after tt	51.66±7.74		
IL -6 Pg/ml	Pt before tt	34.71±7.51	9.362	0.000
	Pt after tt	18.17±4.6		
Chol .mg/dl	Pt before tt	249.6±12.5	17.016	0.000
	Pt after tt	186.6±13.61		
Noradrenaline nmol/ml	Pt before tt	76.12±13.67	9.780	0.000
	Pt after tt	46.4±6.56		
Acetylcholine ng/ml	Pt before tt	8.4±1.4	16.347	0.000
	Pt after tt	20±3.2		
Serotonin ng/ml	Pt before tt	14.66±2.11	27.680	0.000
	Pt after tt	46.4±5.34		
Dopamine ng/ml	Pt before	76.26±13.37	8.895	0.000
	Pt after	44.88±11.49		

Table 5 shows the relation between MMSE values of the controls and patients before treatment. There was a significant difference between controls and patients regarding MMSE values before treatment.

Table 5. Relation between MMSE values of the controls and patients before treatment

	Mean±SD	t- test value	P- value
MMSE (control)	26.73±1.75	7.43	0.000
MMSE (patients)	15.28±5.78		

Table 6 presents the MMSE grades of patients before and after treatment. Initially, the distribution of patients across different MMSE categories was as follows: 1 patient (4.0%) had normal cognitive function, 8 patients (32.0%) had mild

cognitive impairment, 9 patients (36.0%) had moderate cognitive impairment, and 7 patients (28.0%) had severe cognitive impairment.

Following treatment, there was a significant shift in the distribution of MMSE grades. The number of patients with normal cognitive function increased markedly to 10 patients (40.0%). Those with mild cognitive impairment slightly increased to 9 patients (36.0%). Conversely, the number of patients with moderate cognitive impairment decreased to 5 patients (20.0%), and those with severe cognitive impairment dropped substantially to 1 patient (4%).

Table 6. Relation between MMSE grades of the patients before and after treatment

MMSE grades	MMSE (before treatment)	MMSE (after treatment)	□2	P- value
Normal	1(4.0%)	10 (40.0 %)	13.065	0.004
Mild	8 (32.0%)	9 (36.0 %)		
Moderate	9 (36.0%)	5 (20.0 %)		
Severe	7 (28.0%)	1 (4.0 %)		
Total	25 (100.0%)	25(100.0%)		

Prior to the treatment, patients had an average MMSE score of 15.28±5.78 whereas after treatment the score was 26±9.85, showing significant improvement (P=0.000) (Table 7).

Table 7. Relation between MMSE values of the patients before and after treatment

MMSE	Mean±SD	t- test value	P- value
MMSE (patients before treatment)	15.28±5.78	4.69	0.000
MMSE (patients after treatment)	26±9.85		

The results of the correlation analysis between the MMSE values of patients before and after treatment reveal a strong positive relationship (Table 8 and Figure 2).

Table 8. Correlation between MMSE values of the patients before and after treatment

MMSE	r - value	P- value
MMSE (patients before treatment)	0.903	0.000
MMSE (patients after treatment)		

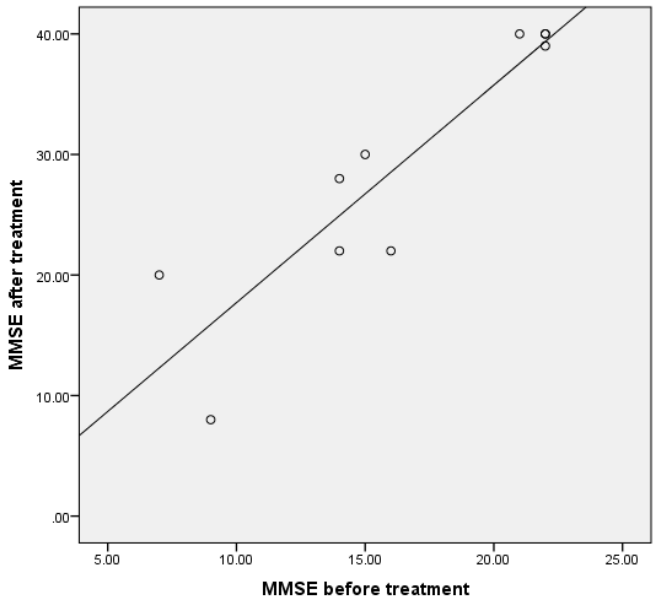

Figure 2. Correlation between MMSE values of the patients before and after treatment

4. DISCUSSION

To the best of our knowledge, this is the first study to date to report the beneficial properties of camel milk in AD patients. The findings of the present study show that camel's milk has therapeutic potential regarding the treatment of AD. Following treatment with camel milk, a significant change in biochemical parameters was observed in the current study. For example, a significant decrease in Aβ42 was reported after treatment with camel milk. These findings are supported by a previous study by Abdulkadir et al. who reported that camel milk and taurine resulted in a significant reduction in Aβ peptide compared to controls ($P < 0.05$) (19). The ratio of plasma Aβ42 and Aβ40 is an accurate measure to detect amyloid plaques and determine the future risk of AD-related dementia in patients (20). AD is closely associated with increased oxidative stress and a compromised antioxidant defence system. Oxidative stress is a critical factor in the pathogenesis of AD which contributes to neuronal damage and cognitive decline. In the present study, camel milk demonstrated antioxidant effects by increasing the GSH levels after treatment. GSH is the most abundant antioxidant that is present in the brain. GSH reacts with reactive oxygen species (ROS) and neutralizes it (21).

In AD, there is a decrease in the level of GSH. A meta-analysis by Chen et al. reported that brain GSH is reduced in AD patients (22). Furthermore, GSH levels are also decreased with age and oxidative stress which are also associated factors of AD. The decrease in GSH levels is also witnessed in peripheral lymphocytes in AD patients and there is an increase in oxidized glutathione (GSSG) (21). The ratio of GSSG to GSH is usually used to detect oxidative stress, which is also seen in AD. A linear correlation between increased GSSG and cognitive decline in AD patients was reported by Lloret et al. (23). The significant increase in GSH observed in the present study indicates that camel's milk increases the overall antioxidant capacity of the patients which can potentially mitigate the oxidate stress to neuronal cells. The reduction in malondialdehyde (MDA) levels further supports the antioxidant properties of camel's milk. MDA is considered one of the markers for identifying oxidative stress (24).

High levels of MDA have also been reported in patients with AD. Some authors suggest that MDA in the erythrocytes should be the biomarker for AD identification (25). MDA is a biomarker of lipid peroxidation. A study by Rani et al. reported that MDA levels were higher in AD patients which indicates the lipid peroxidation in AD pathology (26). The findings of the present study are also consistent with previous studies that have reported that camel's milk decreased MDA (27). Chronic inflammation is another hallmark of AD which contributes to neurodegeneration and cognitive impairment. This study observed a significant decrease in interleukin-6 (IL-6) levels following treatment with camel's milk. IL-6 is a pro-inflammatory cytokine that plays a pivotal role in the inflammatory response associated with AD. Post-mortem AD brains have demonstrated an increased level of IL-6 (28). Similar to our study, Ming et al. reported that camel's milk reduced the production of IL-6 and TNF-α (29). Another study by Alhaider et al. revealed that camel milk inhibits inflammatory angiogenesis by decreasing the production of proinflammatory cytokines such as IL-6, IL-17, and TNF-α (30). These anti-inflammatory effects may be attributed to the bioactive compounds in camel's milk, such as lactoferrin and immunoglobulins which have shown to reduce IL-6 (31).

Neurotransmitter imbalance is a characteristic feature of AD that can highlight the neurobiological basis of symptoms seen in AD patients. In the present study, the increase in serotonin and acetylcholine levels and the decrease in dopamine and noradrenaline levels suggest a rebalancing of neurotransmitter activity. AD patients generally have lower levels of serotonin. A recent study by Smith et al. reported that people with mild cognitive impairment have a loss of serotonin transporters (32). Similarly, several studies have found that AD patients have reduced levels of acetylcholine (33, 34). Acetylcholine is essential for cognitive processes, and its increase suggests an enhancement in cholinergic function which is typically impaired in AD. Hence, the findings of the present study are significant as an increase in acetylcholine and serotonin can help reduce AD symptoms. AD is also associated with increased levels of noradrenaline metabolites (35). The most significant clinical outcome of this study is the improvement in cognitive function, as evidenced by the increase in MMSE scores and the shift in MMSE grades. Cognitive decline is a debilitating aspect of AD. Previously, no study has investigated the cognitive improvement with camel milk in AD patients. However, several studies have reported the beneficial effects of camel milk on autism.

A study reported a significant increase in oxidative stress biomarkers and autistic behaviour after treatment with camel's milk (36). Similarly, significant differences were noted in autistic behaviour after consumption of camel's milk for two weeks in another study (37). Camel's milk has also demonstrated the anti-Parkinson's properties (38). The mechanisms underlying the improvement in various brain disorders are likely multifaceted. The antioxidant and anti-inflammatory properties of camel's milk may protect neuronal cells from damage and promote neuronal health. The improvement in cognitive outcomes can be attributed to improvement in neurotransmitter balance.

There are several limitations of this study as well which should be considered while interpreting the findings of the study. The main limitation of this study is the small sample size. The included population, including 15 controls and 25 camel's milk-treated patients does not represent the broader population. Furthermore, the control and patient groups were not matched appropriately as there was a significant difference in ages of the both groups. Therefore, these groups have inherent differences between them. Another limitation of the study is that it just presents pre- and post-treatment data which does not show the long-term effects of the camel's milk treatments.

5. CONCLUSION

In conclusion, this study shows that camel milk has significant positive improvements in biochemical and cognitive aspects in patients with AD. The significant decrease observed in Amyloid-beta 42, HSP 90, MDA, nitric oxide, IL-6, cholesterol, noradrenaline, and dopamine indicates the potential of camel's milk to reduce oxidative stress in AD. Furthermore, an increase in biomarkers like GSH, total antioxidant capacity, acetylcholine, and serotonin, suggests improvement in antioxidant potential, lipid profiles, and balance of neurotransmitter function. All these parameters observed were also translated into an improvement in cognitive function as well in the present study. However, a small sample size remains the main limitation of the present study. Future research should further investigate these findings in larger trials.

6. ACKNOWLEDGMENTS

The authors extend their appreciation to Prince Sattam bin Abdulaziz University (PSAU) for funding this research work through the project number 2023/03/26310.

REFERENCES

[1] Sheppard O, Coleman M. Alzheimer's disease: etiology, neuropathology and pathogenesis. Exon Publications. 2020:1-21.

[2] Kerwin D, Abdelnour C, Caramelli P, Ogunniyi A, Shi J, Zetterberg H, et al. Alzheimer's disease diagnosis and management: Perspectives from around the world. Alzheimers Dement (Amst). 2022;14(1):e12334.

[3] Breijyeh Z, Karaman R. Comprehensive Review on Alzheimer's Disease: Causes and Treatment. Molecules. 2020;25(24).

[4] Trejo-Lopez JA, Yachnis AT, Prokop S. Neuropathology of Alzheimer's disease. Neurotherapeutics. 2023;19(1):173-85.

[5] Vermunt L, Sikkes SAM, van den Hout A, Handels R, Bos I, van der Flier WM, et al. Duration of preclinical, prodromal, and dementia stages of Alzheimer's disease in relation to age, sex, and APOE genotype. Alzheimers Dement. 2019;15(7):888-98.

[6] Yang J, Wu S, Yang J, Zhang Q, Dong X. Amyloid beta-correlated plasma metabolite dysregulation in Alzheimer's disease: an untargeted metabolism exploration using high-resolution mass spectrometry toward future clinical diagnosis. Frontiers in Aging Neuroscience. 2023;15.

[7] Ou JR, Tan MS, Xie AM, Yu JT, Tan L. Heat shock protein 90 in Alzheimer's disease. Biomed Res Int. 2014;2014:796869.

[8] Guven A, Dalginli KY, Culhaoglu H, Huseyinoglu N, Alp SI. Investigation of the Levels of Blood MDA, GSH and Nitric Oxide Levels in Patients with Probable Alzheimer's Disease. Kafkas Journal of Medical Sciences, Kafkas Tip Bilimleri Dergisi. 2020;10(3).

[9] Maciejewska K, Czarnecka K, Szymański P. A review of the mechanisms underlying selected comorbidities in Alzheimer's disease. Pharmacol Rep. 2021;73(6):1565-81.

[10] Kamil K, Kumar J, Yazid MD, Idrus RBH. Olive and its phenolic compound as the promising neuroprotective agent. Sains Malays. 2018;47:2811-20.

[11] Mecocci P, Tinarelli C, Schulz RJ, Polidori MC. Nutraceuticals in cognitive impairment and Alzheimer's disease. Front Pharmacol. 2014;5:147.

[12] HASSEN KA. Review on nutritional, medicinal value of camel Milk and its public health importance. International Journal on Integrated Education. 2020;3(12):348-63.

[13] Awais MA, Arif A, Asif R, Hussain MI, Niaz A. A Review on Camel Milk and Its Medicinal Properties. Riphah Journal of Allied Health Sciences. 2022;1(1).

[14] Adams CM. Patient report: autism spectrum disorder treated with camel milk. Glob Adv Health Med. 2013;2(6):78-80.

[15] Khatoon H, Najam R, Mirza T, Sikandar B, Ishaq H, Anser H. Evaluation of anticonvulsant and neuroprotective effects of camel milk in strychnine-induced seizure model. Asian Pacific Journal of Tropical Disease. 2015;5(10):817-20.

[16] Khatoon H, Ikram R, Abbas G. Neuropharmacological effects of camel milk related to modulation of biogenic amines in experimental animals. Pak J Pharm Sci. 2019;32(6):2633-41.

[17] Dhapola R, Beura SK, Sharma P, Singh SK, HariKrishnaReddy D. Oxidative stress in Alzheimer's disease: current knowledge of signaling pathways and therapeutics. Molecular Biology Reports. 2024;51(1):48.

[18] Abdulkadir TS, Dawud FA, Isa AS, Ayo JO. Taurine and Camel Milk Modulate Neurobehavioral and

Biochemical Changes in Aluminum Chloride-Induced Alzheimer's Disease in Rats. J Alzheimers Dis. 2021;84(1):291-302.

[19] Abdulkadir TS, Isa AS, Dawud FA, Ayo JO, Mohammed MD. Effect of taurine and camel milk on amyloid beta peptide concentration and oxidative stress changes in aluminium chloride-induced Alzheimer's disease rats. Alzheimer's & Dementia. 2021;17(S12):e058642.

[20] Li Y, Schindler SE, Bollinger JG, Ovod V, Mawuenyega KG, Weiner MW, et al. Validation of plasma amyloid-β 42/40 for detecting Alzheimer disease amyloid plaques. Neurology. 2022;98(7):e688-e99.

[21] Pocernich CB, Butterfield DA. Elevation of glutathione as a therapeutic strategy in Alzheimer disease. Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease. 2012;1822(5):625-30.

[22] Chen JJ, Thiyagarajah M, Song J, Chen C, Herrmann N, Gallagher D, et al. Altered central and blood glutathione in Alzheimer's disease and mild cognitive impairment: a meta-analysis. Alzheimers Res Ther. 2022;14(1):23.

[23] Lloret A, Badia M-C, Mora NJ, Pallardó FV, Alonso M-D, Vina J. Vitamin E paradox in Alzheimer's disease: it does not prevent loss of cognition and may even be detrimental. Journal of Alzheimer's Disease. 2009;17(1):143-9.

[24] 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.

[25] Bermejo P, Gómez-Serranillos P, Santos J, Pastor E, Gil P, Martín-Aragón S. Determination of malonaldehyde in Alzheimer's disease: a comparative study of high-performance liquid chromatography and thiobarbituric acid test. Gerontology. 1997;43(4):218-22.

[26] Rani P, Krishnan S, Rani Cathrine C. Study on Analysis of Peripheral Biomarkers for Alzheimer's Disease Diagnosis. Frontiers in Neurology. 2017;8.

[27] Althwab SA, Alamro SA, Al Abdulmonem W, Allemailem KS, Alarifi SA, Hamad EM. Fermented camel milk enriched with plant sterols improves lipid profile and atherogenic index in rats fed high -fat and -cholesterol diets. Heliyon. 2022;8(10):e10871.

[28] Lyra e Silva NM, Gonçalves RA, Pascoal TA, Lima-Filho RAS, Resende EdPF, Vieira ELM, et al. Pro-inflammatory interleukin-6 signaling links cognitive impairments and peripheral metabolic alterations in Alzheimer's disease. Translational Psychiatry. 2021;11(1):251.

[29] Ming L, Qi B, Hao S, Ji R. Camel milk ameliorates inflammatory mechanisms in an alcohol-induced liver injury mouse model. Scientific Reports. 2021;11(1):22811.

[30] Alhaider AA, Abdel Gader AG, Almeshaal N, Saraswati S. Camel milk inhibits inflammatory angiogenesis via downregulation of proangiogenic and proinflammatory cytokines in mice. Apmis. 2014;122(7):599-607.

[31] Widjaja NA, Hamidah A, Purnomo MT, Ardianah E. Effect of lactoferrin in oral nutrition supplement (ONS) towards IL-6 and IL-10 in failure to thrive children with infection. F1000Res. 2023;12:897.

[32] Smith GS, Kuwabara H, Yan H, Nassery N, Yoon M, Kamath V, et al. Serotonin Degeneration and Amyloid-β Deposition in Mild Cognitive Impairment: Relationship to Cognitive Deficits. Journal of Alzheimer's Disease. 2023;96:215-27.

[33] Chen ZR, Huang JB, Yang SL, Hong FF. Role of Cholinergic Signaling in Alzheimer's Disease. Molecules. 2022;27(6).

[34] Fullwood NJ. Neural stem cells, acetylcholine and Alzheimer's disease. Nature Chemical Biology. 2007;3(8):435-.

[35] van Hooren RWE, Verhey FRJ, Ramakers IHGB, Jansen WJ, Jacobs HIL. Elevated norepinephrine metabolism is linked to cortical thickness in the context of Alzheimer's disease pathology. Neurobiology of Aging. 2021;102:17-22.

[36] Al-Ayadhi LY, Elamin NE. Camel Milk as a Potential Therapy as an Antioxidant in Autism Spectrum Disorder (ASD). Evid Based Complement Alternat Med. 2013;2013:602834.

[37] Al-Ayadhi LY, Halepoto DM, Al-Dress AM, Mitwali Y, Zainah R. Behavioral Benefits of Camel Milk in Subjects with Autism Spectrum Disorder. J Coll Physicians Surg Pak. 2015;25(11):819-23.

[38] Khatoon H, Najam R, Mirza T, Sikandar B. Beneficial anti-Parkinson effects of camel milk in Chlorpromazineinduced animal model: Behavioural and histopathological study. Pak J Pharm Sci. 2016;29(5):1525-9.