

## Research Report

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# The Role of Nutrition and Inflammation on Cognition in a High-Risk Group for Alzheimer's Disease

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### Abstract.

**Background:** Alzheimer's disease (AD) is a prevalent neurodegenerative disease. Treatments are necessary to target people at high risk for AD. Inflammation, particularly tumor necrosis factor alpha (TNF $\alpha$ ), appears to be an important marker associated with the development of AD pathophysiology. Consuming a high-fat diet induces tissue expression of TNF $\alpha$ .

**Objective:** This study investigates the relationship between nutrition, circulating inflammation, and cognition in African American women (age:  $M = 59.5 (\pm 8.20)$  [42–73] years) at risk for developing AD.

**Methods:** Participants were split into high-fat and low-fat groups based on total dietary fat consumption self-reported on the Lower Mississippi Delta Nutrition Intervention Research Initiative Food Frequency Questionnaire (Delta NIRI FFQ).

**Results:** A high-fat diet was associated with increased blood serum TNF $\alpha$  ( $p = 0.02$ ) compared to the low-fat diet. In addition, global cognition scores were 9.0% better in those who consumed a higher fat diet ( $p = 0.04$ ). No significant differences across groups were noted for executive function, dual-tasking, and visuospatial performance.

**Conclusion:** These results indicate that there may be multiple biological pathways involved in AD development, suggesting the need for more holistic approaches to mitigate AD-development risk.

**Keywords:** African American, Alzheimer's disease, cognition, executive function, inflammation, nutrition, tumor necrosis alpha

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## INTRODUCTION

Alzheimer's disease (AD) is the 6th leading cause of death in the United States [1]. AD is a disorder that gradually and progressively destroys cognitive function. While the disease first affects declarative memory, it progresses to affect nondeclarative memory and ultimately leads to dementia [2]. AD has become more prevalent in recent years, and its prevalence is expected to increase. By 2050 it is predicted that one in 85 people world-wide will have AD, including 13 million Americans [3, 4]. This increase in prevalence makes it imperative to develop better diagnostic measures and more efficient screening procedures for those in early stages of the disease [5, 6].

Ethnic minorities, female sex, and family history are aggravating factors of AD. The Alzheimer's Association estimates that the prevalence of AD and other dementias in African Americans (AA) above the age of 65 years is about twice the rate of older Whites [7]. Similarly, women are disproportionately affected by AD. Women are two to three times more likely to develop AD than men [8] and are also more likely to become caregivers to AD patients. Genetic factors such as Apolipoprotein E (*APOE*)  $\epsilon 4$  allele increases the frequency of development of AD [9, 10]. Recent data shows that a higher percentage of AA have at least one copy of the  $\epsilon 4$  allele compared to European Americans [1]. Independent of genetic factors, those with a parental history of AD have a greater risk of developing dementia [11, 12]. Having one or both parents with dementia has been associated with greater cognitive impairment, thus contributing to disease severity [9]. Important next steps in investigating treatment for AD is to target individuals who are at a higher risk for AD and institute preventative strategies to provide early intervention [13].

Because ethnicity and biological sex cannot be changed, identifying modifiable risk factors in high risk groups is needed to reduce risk of cognitive decline and AD. One modifiable lifestyle factor that has received relatively little attention in the pathophysiology of AD is nutrition. In animal models, a high-fat (Western) diet has been shown to exacerbate AD-related pathology [14], but less is known in humans.

A high-fat diet can alter the immune system [15]. Alterations in the immune system could contribute to the pathophysiology of AD [16]. Elevated pro-inflammatory cytokines, particularly tumor necrosis

factor alpha (TNF $\alpha$ ), are risk factors for the onset and progression of AD [17, 18]. On average, people with AD have higher levels of serum TNF $\alpha$  compared to healthy adults [19]. High levels of circulating TNF $\alpha$  correlate with poor performance on cognitive tasks [20]. Levels of TNF $\alpha$  expression can be altered with diet. A diet high in saturated fat increases the amount of TNF $\alpha$  present in the plasma compared to a diet high in unsaturated fats [21]. In addition, production of pro-inflammatory cytokines is further augmented after an inflammatory stimulus in people with high trans-fat diets [22, 23]. Currently unexplored is the relationship of a high-fat diet on characteristics of AD, specifically in people who are predisposed for developing AD. This study included African American women who are predisposed for AD (have a parental history) to determine if individuals who consume a high-fat diet show early signs of memory deficits and systemic inflammation.

## MATERIALS AND METHODS

### *Study and participants*

The Institutional Review Board at Emory University School of Medicine and the Research and Development Committee of the Atlanta VA approved this work. Participants provided written informed consent before participating. Participants were recruited from the Emory Alzheimer's Disease Research Center from registries derived from previous studies of individuals with parental history of AD. Forty-two individuals were recruited for the study. Participants who completed less than half of the nutritional questionnaire ( $n=11$ ) and participants whose biomarker data was not completed ( $n=5$ ) were excluded from analyses resulting in a sample of 26 individuals. There were no significant differences between excluded and included participants on key demographics and clinical characteristics (Age (y): Included 59.5, Excluded 63.7; Education (y): Included 15.9; Excluded:16.3; BMI ( $\text{kg}/\text{m}^2$ ): Included 30.6, Excluded 29.3; Number of falls (no.): Included 0.583, Excluded 0.3).

Participants were at least 40 years of age and self-identified as African American (AA) and a woman. The participants' parents had a diagnosis of probable AD as defined by National Institute of Neurological Disorders and Stroke-Alzheimer's Disease and Related Disorders Association (NINDS-ADRDA) criteria, which was verified using the validated

Dementia Questionnaire and medical records when available [24]. Because these women had a parent with likely AD, these women were considered to be at greater risk for developing AD.

### Nutrition data

Lower Mississippi Delta Nutrition Intervention Research Initiative Food Frequency Questionnaire (Delta NIRI FFQ) was used to assess dietary habits because it highlighted the unique food, preparation, and portion sizes often consumed by AA adults from the Southern region [25]. Participants self-administered the 158-item (short FFQ) Delta NIRI FFQ. Participants were instructed to report how often, on average, certain foods were consumed across the past year. This short FFQ was designed to include all foods on the longer FFQ, by collapsing similar foods to combine line items and simplifying adjustment questions and has been validated (Table 1) [26].

The Delta NIRI FFQ was used to categorize participants into low-fat or high-fat diets. Participants' self-reported dietary consumption habits were converted into an overall fat intake score based on dietary consumption frequency and portion sizes of 13 select high-fat food categories: 1) poultry and game (i.e., fried dark versus light meat), 2) beef and pork, 3) luncheon meat, sausage, hotdog, and eggs, 4) other meat (i.e., organ meat, ham hock), 5) fish (fried and fatty varieties), 6) vegetables (i.e., fried potatoes; fried onions), 7) mixed dishes, pizza, and pasta dishes, 8) milk (full versus low fat varieties), 9) non-beverage dairy products (i.e., cheese, yogurt), 10) condiments (i.e., salad dressing, gravy), 11) dessert, sweets, and snacks (i.e., chips; nuts), 12) added fats (i.e., oils), and 13) omega-3 fatty acid supplement. For each category, subjects were assigned a score of 0 (low fat) or 1 (high fat) based upon whether their intake was  $\leq$  or  $>$  the median intake of the group. All categories were summed to calculate an overall dietary fat intake score, with 0 representing the lowest fat intake and 13 representing the highest fat intake. Overall dietary fat intake scores of  $\leq 6$  were considered "low-fat" and  $> 6$  were considered "high-fat".

To further clarify, food groups that are high in fat were assigned a score by assessing frequency of consumption and typical serving size consumed. Further, the food groups that we chose to evaluate were high in total fat, regardless of the type of fat.

Table 1  
Food Grouping Used in Dietary Pattern Analysis

Food Groups	Food Items
Fried Potatoes/Onion	French Fries, Fried Potatoes, Potato Logs, Fried Onion Rings
Poultry and Game	Fried Chicken, "How often do you eat the skin on the chicken"
Lunch Meat, Bacon, and Eggs	Hot Dogs, Sausage (non-breakfast type), Ham, Bologna, Salami, Potted Meat, Luncheon Meat, Bacon, Breakfast Sausage, Eggs
Mixed Meat, Pizza, and Pasta	Mixed Dishes with Cheese (Macaroni and Cheese, Lasagna, Broccoli and Rice Casserole), Pizza
Dairy	Cottage Cheese, Cheese Spread and Dips, Yogurt (Not Frozen)
Beef and Pork	Fried Beef, Pork (chops, roasts, spareribs), "How often do you trim the fat from the meat"
Other Meat	Neck bones, Ham Hock, Pig's Feet, Liver
Fish	Fried Fish or Fish Sandwich (including catfish), Sardines, Maceral, Canned Salmon, Tuna Salad, Tuna Casserole,
Condiments	Gravy, Salad Dressing (Regular, Light, and Fat-Free), Mayonnaise
Desserts, Sweets, and Snacks	Ice Cream, Pudding, Custard, Cheesecake, Doughnuts, Cookies, Sweet Rolls, Muffins, Potato Chips, Corn Chips, Cracklings, Popcorn, Peanuts, Other Nuts, and Peanut Butter
Oils	Stick Margarine, Soft Tub Margarine, Butter, Salt Pork, Bacon Fat, Olive Oil, Canola Oil, Vegetable Oil
Vitamins	Omega 3 Fatty Acids
Other Beverages	Milk

Food groupings were determine based on their section placement in the Lower Mississippi Delta Nutrition Intervention Research Initiative Food Frequency Questionnaire (Delta NIRI FFQ).

### Cytokine data

Participants underwent blood draws for inflammatory cytokines after an 8-hour overnight fast. Plasma was collected by members of the research team. Four panels of biomarkers were measured in plasma using singleplex or multiplex assays in a Luminex 200 platform. Measured biomarkers included those that have been linked to AD and family caregiver stress [20, 27, 28].

### Cognitive function data

Cognitive testing was performed by trained, experienced and qualified research assistants. This study evaluated global and executive function, using

the Montreal Cognitive Assessment (MoCA), the Reverse Corsi task (Corsi), the Trails Making Task (TMT), and the D-KEFS Tower Test. Widely-accepted methods of reporting the data for each assessment were employed, including raw scores and scaled scores based on age norms when appropriate [29].

MoCA is a global cognitive screening tool for mild cognitive impairment [30]. It includes eight cognitive domains assessing clock drawing and 3-D cube copy task, TMT B task, phonemic fluency task, two-item verbal abstraction task, sustained attentional task, serial subtraction task, naming task, repetition of sentence task, and orientation to time and place task. Throughout the assessment, a score is given out of 30. One point is added for those with less than 12 years of education. A score greater than or equal to 26 is considered normal [30]. This is based on previous research which found that a cutoff score of 26 detected 90% of MCI subjects. Several papers have found that the minimal clinically important difference (MCID) for the MoCA test is 2 points [31–33].

Corsi task assesses short-term and working memory using nonverbal analog. The task consists of a board containing nine cubes at fixed, pseudorandom positions. The blocks are labeled with numbers only visible to the experimenter. The experimenter taps a certain number of blocks, after which the participant has to tap this block sequence in the reverse order as presented. The block sequences gradually increase in length, and the score that is obtained is generally the number of correctly remembered sequences or the length of the longest sequence that was remembered correctly [34].

Tower Test assesses planning ability by having participants move five discs of different sizes on pegs in specific arrangements printed on cards that were presented by the administrator [35]. They were told to attempt to make the arrangement in the least amount of moves possible. The number of moves as well as time was recorded.

TMT assesses executive function, visual attention and task switching among participants. This task contains two parts: part A and part B. In TMT A, participants need to connect 25 numbered circles continuously in an ascending order. In TMT B, 13 numbers and 12 letters must be alternately connected in ascending numerical and alphabetical order. The test is terminated after 5 minutes even if not completed. Once complete, a difference score (TMT B – TMT A) is calculated [36].

### Statistical analysis

Descriptive analyses were used to assess demographics of the study population using *t*-tests or Fisher's exact tests. For outcome variables, data were tested for normality using the Shapiro-Wilk test. Based on normality tests and the type of data (continuous or ordinal), Mann-Whitney U tests or *t*-tests were utilized to assess the association between dietary fat intake and scores on cognitive tests (MoCA, Tower Test, Corsi, and TMT) or serum cytokine levels. All analyses were conducted using SAS (SAS version 9.4, 2013, SAS Institute, Cary, NC). Significance for all analyses was set at the 0.05 level. A point-biserial correlation was performed between TNF $\alpha$  and the classification of diet (high-fat/low-fat diet). Pearson correlations were performed between cytokines.

## RESULTS

Twenty-six women with first degree parental history of AD ( $59.5 \pm 8.20$  years) were included in the sample analyses. Participants in the high-fat group ( $n=7$ ) and low-fat group ( $n=19$ ) did not differ significantly in the following measures: education, quality of life, number of medications, age, number of falls, BMI, learning disabilities, comorbidities, level of exercise, marital status, occupation, and income (Table 2).

Regarding whether dietary fat consumption was associated with performance on cognitive tests, participants in the high-fat diet group performed better on the MoCA ( $p=0.04$ ;  $\eta^2=0.148$ ) compared to the low-fat diet group (Table 3). However, there was no significant difference between the groups in performance on the TMT, Tower Test, and Corsi blocks test (Table 3).

Regarding the effect of dietary fat intake on plasma cytokine levels, individuals who consumed a high-fat diet exhibited increased plasma levels of TNF $\alpha$  compared to the low-fat diet group ( $p=0.02$ ;  $\eta^2=0.196$ ) (Table 4). Further, a correlation was observed between TNF $\alpha$  and fat diet category ( $r=0.44$ ,  $p=0.024$ ). There were no differences in any other plasma cytokine levels (CRP, IL-10, and IL-8) between the high-fat and low-fat groups. A significant positive correlation was observed between TNF $\alpha$  and high IL-10 concentration ( $r=0.401$ ,  $p=0.04$ ).

Table 2  
 Characteristics of 26 African-American Women with Parental History of AD

	Overall (n (%))	High-Fat	Low-Fat	P
BMI (kg/m <sup>2</sup> )	30.65(4.86)	32.48 (5.51)	29.98 (4.57)	0.253
Age (y)	59.5 (8.20)	55.57 (5.38)	60.95 (8.67)	0.141
Number of Medications	5.85 (4.24)	6.43 (5.09)	5.63 (4.02)	0.680
Number of Falls	0.583 (0.88)	0.714 (0.78)	0.529 (0.94)	0.650
Years of Education	15.50 (3.22)	15.43 (0.98)	16.21 (2.10)	0.356
Occupation Status				
Disabled	3 (11.54)	1 (14.29)	2 (10.53)	0.197
Retired	11 (42.31)	1 (14.29)	10 (52.63)	
Unemployed/seeking employment	2 (7.69)	0 (0)	2 (10.53)	
Work full-time	7 (26.92)	4 (57.14)	3 (15.79)	
Work part-time	3 (11.54)	1 (14.29)	2 (10.53)	
Type of Housing				
House/Apartment/Condominium	24 (92.31)	6 (85.71)	18 (94.74)	0.473
Relatives Home	2 (7.69)	1 (14.29)	1 (5.26)	
Income				
\$19,000 or less	5 (19.23)	2 (28.57)	3 (15.79)	0.697
\$20,000–\$39,000	6 (23.08)	2 (28.57)	4 (21.05)	
\$40,000–\$59,000	8 (30.77)	1 (14.29)	7 (36.84)	
\$60,000–\$79,000	2 (7.69)	0 (0)	2 (10.53)	
\$80,000 or more	5 (19.23)	2 (28.57)	3 (15.79)	
Marital Status				
Married/partnered	7 (26.92)	1 (14.29)	6 (31.58)	0.430
Separated/Divorced	12 (46.15)	3 (42.86)	9 (47.37)	
Single	4 (15.38)	1 (14.29)	3 (15.79)	
Widowed	3 (11.54)	2 (28.57)	1 (5.26)	
Education				
Years of Education	15.50 (3.22)	15.43 (0.98)	16.21 (2.10)	0.356
High school graduate/GED	1 (3.85)	0 (0)	1 (5.26)	0.086
Vocational training	2 (7.69)	2 (28.57)	0 (0)	
Some college/associate’s degree	4 (15.38)	0 (0)	4 (21.05)	
Bachelor’s Degree (BA, BS)	13 (50)	5 (71.43)	8 (42.11)	
Master’s degree (or other post-graduate training)	4 (15.38)	0 (0)	6 (21.05)	
Doctoral degree (PhD, MD, EdD, DDS, JD, etc.)	2 (7.69)	0 (0)	2 (10.53)	
Exercise Activity				
Never	2 (7.69)	0 (0)	2 (7.69)	0.932
Once a month	2 (7.69)	1 (14.29)	1 (3.85)	
1–4 times per month	6 (23.08)	2 (28.57)	4 (15.38)	
Greater than once a week	15 (57.69)	4 (57.14)	11 (57.89)	
Don’t know	1 (3.85)	0 (0)	1 (5.26)	
Smoker				
Yes	7 (26.92)	3 (42.86)	8 (42.11)	1
No	19 (73.08)	4 (57.14)	11 (57.89)	
Alcohol in the past month				
Yes	7 (26.92)	1 (14.29)	12 (63.16)	0.375
No	19 (73.08)	6 (85.71)	7 (36.84)	
High Cholesterol				
No	8 (30.77)	3 (42.86)	5 (26.32)	0.301
Yes	16 (61.54)	3 (42.86)	13 (68.42)	
Unsure	2 (7.69)	1 (14.29)	1 (5.26)	
Have you ever been told you have high blood pressure?				
Yes	18 (62.23)	4 (57.14)	14 (73.68)	0.635
No	8 (30.77)	3 (42.86)	5 (26.32)	
Do you have diabetes?				
Yes	5 (19.23)	1 (14.29)	4 (21.05)	1
No	19 (73.08)	6 (85.71)	13 (68.42)	
Unsure	2 (7.69)	0(0)	2 (10.53)	
In general, how would you rate your quality of life?				
Low	2 (7.69)	2 (28.57)	0 (0)	0.068
Moderate	13 (50)	3 (42.86)	10 (52.63)	
High	9 (34.62)	2 (28.57)	7 (36.84)	
Very High	2 (7.69)	0 (0)	2 (10.53)	

Independent *t*-tests (Mean ± SD) or Fisher’s exact tests (n(%)) were used to determine significant differences between groups.

Table 3

Cognitive performance of 26 African-American Women with Parental History of AD

Variable		<i>M</i> ( <i>SD</i> )	$\eta^2$	<i>P</i>
MoCA (/30)	Low-fat Intake	24.7 (2.7)	0.148 <sup>§</sup>	0.04 <sup>†</sup>
	High-fat Intake	27.0 (2.2)		
Tower Test (total achievement score)	Low-fat Intake	9.6 (2.3)	0.002	0.81
	High-fat Intake	9.9 (1.4)		
TMT Diff. (s) (Trails B – A)	Low-fat Intake	50.3 (23.3)	0.025	0.34 <sup>†</sup>
	High-fat Intake	41.8 (28.0)		
Corsi (number of correct trials)	Low-fat Intake	9.6 (2.3)	0.002	0.81
	High-fat Intake	9.9 (1.4)		

*M*, mean; *SD*, standard deviation. <sup>§</sup>Moderate effect size according to partial eta squared. <sup>†</sup>*p*-values determined with Mann-Whitney U test.

Table 4

Inflammatory Variables of 26 African-American Women with Parental History of AD

Variable		<i>M</i> ( <i>SD</i> )	$\eta^2$	<i>P</i>
TNF $\alpha$	Low-fat diet	5.17 (1.3)	0.196 <sup>§</sup>	0.02
	High-fat diet	6.82 (2.0)		
CRP <sup>^</sup>	Low-fat diet	0.394 (0.52)	0.000	0.69 <sup>†</sup>
	High-fat diet	0.371 (0.35)		
IL-10	Low-fat diet	10.96 (5.41)	0.002	0.62 <sup>†</sup>
	High-fat diet	11.47 (4.37)		
IL-8	Low-fat diet	16.51 (38.18)	0.028	0.56 <sup>†</sup>
	High-fat diet	4.24 (2.02)		

*M*, mean; *SD*, standard deviation; IL, Interleukin; CRP, C-reactive protein; TNF $\alpha$ , Tumor necrosis factor alpha;  $\eta^2$ , partial effect size. <sup>†</sup>*p*-values determined with Mann-Whitney U test. <sup>^</sup>measured in ng/mL, all others measured in pg/mL.

## DISCUSSION

This study demonstrated better global cognition and higher plasma TNF $\alpha$  levels in AA women with a family history of AD who consume a high-fat diet compared to a low-fat diet. While changes in global cognition were found between dietary fat groups, there were no significant differences in performance on other measures of cognitive function, including the Corsi, Tower Test, and the TMT.

Performance on cognitive tasks between the dietary fat groups contradicted the original hypothesis that higher fat intake results in decreased cognitive ability. Participants who consumed a high-fat diet performed better on the MoCA (~27 points) compared to those with a lower intake of dietary fat (~25 points). A cut-off score of 26 is typically used to diagnose MCI with 90% accuracy [30]. Using this cutoff, individuals consuming a low-fat diet would be considered having an MCI compared to high-fat diet. Further, the mean difference between the high-

fat diet and the low-fat diet is 2.32. This is higher than the reported MCID (2 points) for the MoCA indicating a difference in clinical treatment and functional meaning between the two groups. While no statistical difference was observed between nutrition groups for TMT, our findings may be clinically relevant. A study examined well-educated (education = 12–19 years), community-dwelling residents with no CNS trauma, alcohol abuse, or cardiac problems [37]. 105 females (45 years of age or older) performed an average TMT difference of 39.6 s based on averaging subtracted means (B–A). Based on these values, only a slight impairment was observed for the high-fat diet group (41.8 s), but the low-fat group exhibited a more considerable impairment (50.3 s). As a result, this may indicate mild cognitive impairment in abilities such as working memory, executive function, and cognitive flexibility. Deficiencies in these areas may impair everyday normative functions such as driving which requires moderate cognitive flexibility.

This finding raises new questions about the role of diet and cognitive function. Researchers have often examined the role of nutrition on global cognition, yet present contrasting conclusions. These differing conclusions could be due to varying fatty acid profiles within the diet. For example, a prospective cohort study from 1988–2016 found that adhering to a Mediterranean diet or a plant based polyunsaturated fatty acid diet increases global cognitive function and executive function support [38]. Whereas another prospective cohort, a higher intake of a Western style diet (typically higher in saturated fats) was correlated with worse cognitive performance on visual spatial learning, long-term memory, and reaction time tasks [39]. While the exact composition of saturated and unsaturated fats was not calculated in this paper, our data suggests that a high-fat diet can be beneficial for cognition. Similar findings have been found in rodents on a ketogenic diet that was high in fats [40].

Elevated levels of the proinflammatory cytokine TNF $\alpha$  were observed in the plasma for individuals eating a high-fat diet compared to low-fat diet. These results replicate previous findings that a high-fat diet increases the amount of TNF $\alpha$  present in the plasma [21]. Further, this study found that levels of TNF $\alpha$  correlated with IL-10, an anti-inflammatory cytokine. The increase in an anti-inflammatory cytokine might be in response to the increase in TNF $\alpha$ .

This study had several limitations. This study was underpowered therefore the results should be interpreted cautiously. Specifically, the ability to detect significant correlations was limited due to

the small sample sizes. However, it is important to disseminate the results of this pilot study to begin to build a foundation of knowledge regarding this unique, underserved and understudied population. Furthermore, USDA Food Composition Database was temporarily affected by political factors and not updated regularly. This lapse may have affected the total fat average on the various food options which was used to compute the overall fat score. Another limitation is that this study did not categorize fat intake by saturated or unsaturated fats. Previous research has shown the difference that fatty acids can have on cognition [38, 39] and cytokine levels [21]. Many of the high fat foods listed were high in saturated fats, with the exception of the omega 3 supplement. This study did not specifically consider the omega 3 supplement's impact on fat content or cognitive status. It is possible that taking an omega 3 supplement alone is associated with better cognitive function. Future studies should investigate the interrelationship of cognitive function with omega 3 and fat intake. Caregiver status was also not considered. Caregiver status may influence inflammatory variables measured. Many studies have suggested a link between family caregiving and adverse mental and physical health consequences. However, family caregiving may also have positive effects on health and well-being. Several population-based studies have found longer lifespans for family caregivers compared to individuals who did not have family caregiver responsibilities [41]. However, AD caregiving is associated with higher levels of subjective stress measures, as well as elevated levels of C-reactive protein and inflammatory cytokines [27, 28].

AD is the most prevalent age-related neurodegenerative disease and is clinically characterized by a progressive loss of memory and other cognitive functions. Although it is difficult to identify which environmental factors induce AD and other dementia-based disorders, it is important to take preventative measures to delay cognitive impairments in high-risk individuals. Deficits in cognition lead to impairments in everyday activities, which require processes involved in reasoning, synthesis and problem-solving. In this study, we rejected the original hypothesis that consuming a high-fat diet would cause cognitive impairments. Yet, it is interesting to note that nutrition science often has various conflicting information involved in what affects the human body. Nutritional evidence-based science data often reverse or contradict one another over time. This

may explain why we saw more prominent cognitive deficits in the low-fat consumption group. Ultimately, this is a pilot study to power future studies involved in nutrition and cognitive function. By understanding which dietary modifications are needed to delay or mitigate the onset of AD, more nutritional based programs can be implemented. Subsequently, diet and nutrition-based interventions for those at risk for developing AD may result in decreasing mortality rates linked to AD, especially in regions where nutrition influences culture.

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## CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

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