Journal of Health, Population and Nutrition

RESEARCH

Open Access

The association between metabolic syndrome components and cognitive function in community-dwelling middle-aged and older adults: the first wave result of a cohort study

Hsin-Yeong Yen[1†], Yi-Hsuan Lin[1,2†], Yen-Feng Wang[1,3], Jong-Ling Fuh[1,3], Shuu-Jiun Wang[1,3], Harn-Shen Chen[1,4], Shu-Chiung Chiang[1], Sih-Rong Li[5], Ming-Hwai Lin[1,5], Tzeng-Ji Chen[1,5,6,7], Shinn-Jang Hwang[1,5,8] and Hsiao-Ting Chang[1,5*]

Abstract

Background The components of metabolic syndrome (MetS) have previously been demonstrated to be contributors to cognitive decline in older adults as individual factors, but not collectively as a syndrome. This study investigated whether adults ≥ 50 years old who meet the criteria for MetS were more likely to develop impaired cognition than those without MetS.

Methods Adults aged 50 years or older without significant cognitive impairment who received outpatient care at Taipei Veterans General Hospital were recruited. Waist circumference, blood tests for Mets components, and high-sensitivity C-reactive protein (hsCRP) were measured. Demographics, health condition, cognitive function (by Montreal Cognitive Assessment Taiwanese version, MoCA-T, and AD-8), depression symptoms (by Geriatric Depression Scale-15) and functional status (by Barthel's Index, and Lawton & Brody instrumental activities of daily living, IADL) were evaluated. Associations between MetS and cognitive function were analyzed by multivariate logistic regression.

Results Data of 567 participants were analyzed. The prevalence of MetS of the study population was 34.2%. MetS status was not significantly correlated to cognitive decline as indicated by Montreal Cognitive Assessment Taiwan version ($p=0.13$) and AD-8 ($p=0.42$). Mild abdominal obesity decreased the risk of developing impaired cognition in women (adjusted OR = 0.62, 95% CI = 0.42, 0.93, $p=0.02$) but not in men (adjusted OR = 0.84, 95% CI = 0.46, 1.53, $p=0.58$).

Conclusions MetS is not a significant contributory factor to cognitive decline in community-dwelling middle-aged and older adults. An optimal waist circumference in community-dwelling older women is protective against the development of mild dementia.

Keywords Cognition, Older adults, Metabolic syndrome, Middle-aged, Montreal cognitive assessment taiwanese version

†Hsin-Yeong Yen and Yi-Hsuan Lin have equal contribution for this work.

*Correspondence:
Hsiao-Ting Chang
htchang2@vghtpe.gov.tw
Full list of author information is available at the end of the article

Introduction

Metabolic syndrome (MetS) is a constellation of metabolic disorders, including abdominal obesity, hypertension, hyperglycemia, hypertriglyceridemia, and low high-density lipoprotein cholesterol (HDL-C), which are associated with neurocognitive diseases such as stroke and neurodegeneration [1]. MetS and its components have been correlated to cognitive impairment in older adults [2, 3], as neuroendocrine crosstalk plays a major contributory role in the homeostasis of metabolism and neurological function. Neural regulation of metabolism has been well-established [4–6]. Both animal and human studies [7–9] have indicated that aberrant metabolic regulation causes downstream disruption of neurological structures and function, including cerebrovascular endothelial dysfunction in hypertension [10–12] and glucose-mediated neuropathy [13].

According to a systematic review, the global prevalence of cognitive impairment ranged from 5.1% to 41% among adults older than 50 years [14]. Cognitive impairment can lead to difficulties in performing daily tasks, following instructions, and producing appropriate communication [15]. Cognitive impairment is also detrimental to the ability of instrumental activities of daily living (IADL) and contributes to lower quality of life [16, 17]. Furthermore, impairment of cognitive and functional abilities in older adults poses an increased risk of falls [18]. Injuries from falls in older adults tend to be moderate to severe and are a major cause of mortality and loss of independence [19]. Consequently, cognitive impairment causes a substantial health burden and precipitate financial and emotional distress for caregivers and family members [20]. Therefore, identifying factors associated with dementia is crucial for providing insight into appropriate health management and long-term care resources for the global aging society.

Interestingly, several studies have presented evidence suggesting that the relationship between MetS and cognitive deficits may vary depending on age and sex. Although MetS has been linked to the development of cognitive impairment in middle-aged adults [21], this association is less evident in the older old (adults aged 75 years or greater) [22–24]. Additionally, MetS may be more likely to cause cognitive deficits in women than in men [25]. However, a limitation of many studies on the cognitive functions of patients with MetS is the assessment of cognitive ability solely with the Mini–Mental State Examination (MMSE), which may not be sensitive enough to detect mild cognitive impairment [26]. Considering the scarcity of data based on more sensitive assessments of cognitive function and the mixed results regarding age and sex as co-factors of MetS impact on cognition, further investigation of the differences in MetS effects on cognition in different age ranges between men and women is needed.

This research is a community-based cross-sectional study aimed at investigating the association between MetS and cognitive function in middle-aged and older adults. Considering the rising prevalence of MetS [27] and the global aging tendency, understanding this topic is important in facilitating long-term care planning in geriatric medicine. In this study, we use the Montreal Cognitive Assessment Taiwanese version (MoCA-T) and the Eight-item Informant Interview to Differentiate Aging and Dementia (AD-8) to evaluate cognitive function, as both are sensitive tools to identify mild cognitive impairment and dementia [28]. By presenting evidence on this pivotal issue, we aim to illuminate the healthcare challenges faced by an aging society and foster further discussion on preventive medicine directions.

Methods

Study design and participants

This cross-sectional study was the first wave of the cohort study of "The Taipei Veterans General Hospital Precision Medicine Study on Metabolic Syndrome Risk Factors" based on community-dwelling men and women who are at least 50 years old and receive outpatient care in the Taipei Veterans General Hospital (TPVGH). The protocol for this study was approved by the Institutional Review Board (IRB) of TPVGH (protocol No.: 2020–06-001A), and the research implementation adheres completely to the guidelines set forth by the Committee on Human Experimentation and the principles outlined in the Declaration of Helsinki.

Community residents visiting outpatient clinics were invited to participate in the study. After a full explanation of the study, subjects who could understand and agreed to participate signed a written informed consent. The recruitment period for the study was from 1 June 2020 to 31 May 2023. The exclusion criteria were as follows: 1) individuals with significant cognitive impairment (unable to understand the study and could not independently sign the participation consent form after an interview with and explanation of the study and informed consent by well-trained research personnel. The research personnel would also discuss the evaluation of cognitive function with the principal investigator to decide if the subject was eligible for recruitment. The subject was excluded if he/she had already been diagnosed with moderate to severe dementia.), and 2) residents of long-term care facilities or nursing homes. Figure 1 illustrates the participant selection process and subsequent data collection for this study.

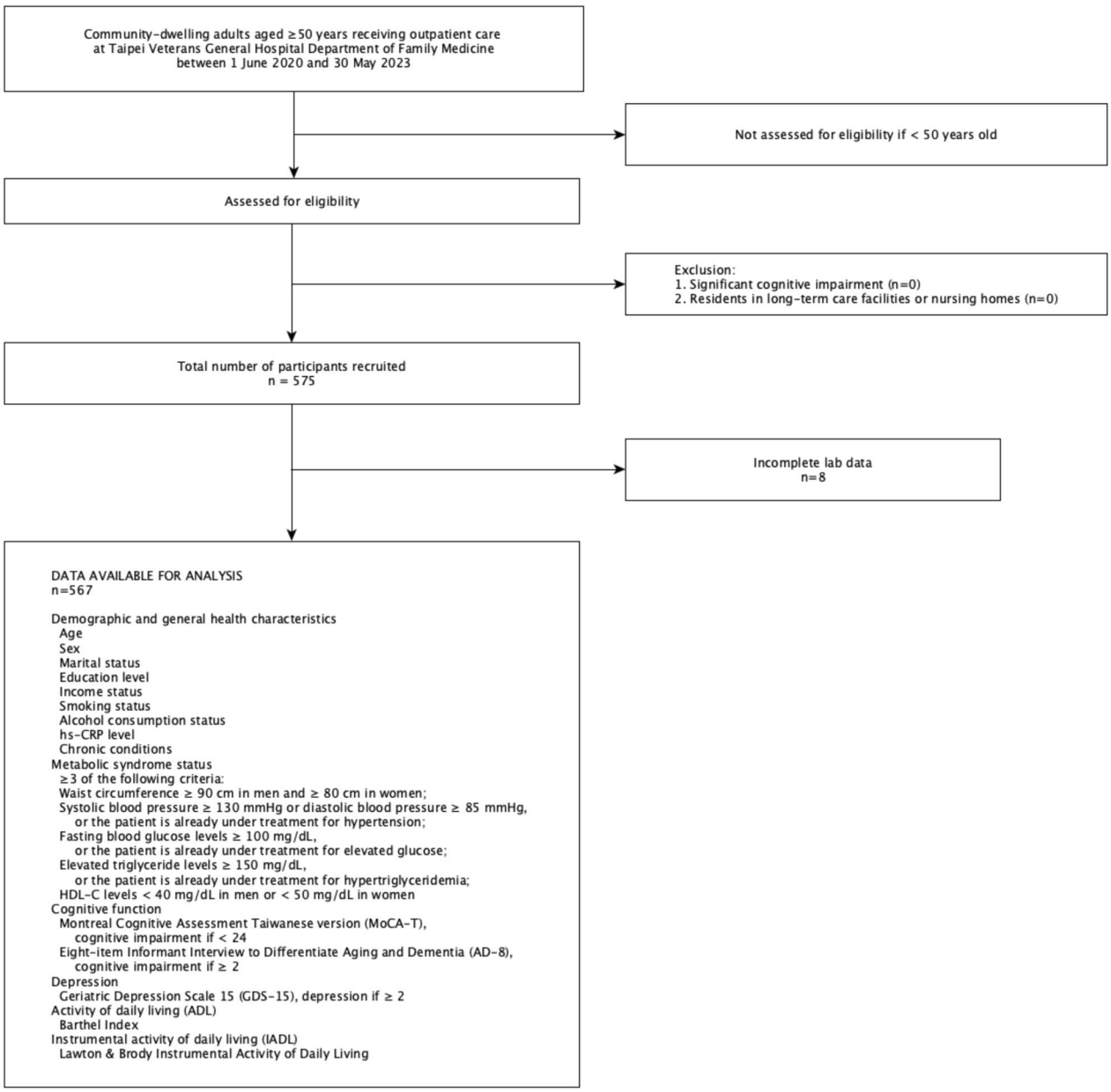

Fig. 1 Study participant selection and data collection

Measurements and questionnaires

Participants received a comprehensive health exam, including height (cm), weight (kg), body mass index (kg/m^2), waist circumference (cm), physical examination, and blood tests. Demographic information was collected for each participant, including sex, age, education level, marital status, status of cigarette smoking, alcohol consumption, income, and medical history of hypertension, type 2 DM, hypertriglyceridemia, low HDL-C, stroke, lung diseases, depression, degenerative joints diseases (DJD), malignancy, pain, and medication history. Smoking status for current smokers was further described as < 20 pack-years or ≥ 20 pack -years according to the 2025 Lung Cancer Screening guidelines recommended by the National Comprehensive Cancer Network (NCCN) for adults ≥ age 50 [29]. Heavy drinking was described as 15 or more drinks per week for men or 8 or more drinks per week for women, according to the National Institute on Alcohol Abuse and Alcoholism (NIAAA) [30].

Depression was evaluated with the Geriatric Depression Scale 15 (GDS-15) [31]. The participants' activity of daily living (ADL) status was assessed with Barthel's

Index [32], and instrumental activity of daily living (IADL) was measured with the Lawton & Brody Instrumental Activity of Daily Living [33]. Blood tests included liver function, renal function, fasting blood glucose level, total cholesterol, high-density and low-density lipoprotein cholesterol levels, triglyceride level, uric acid level, complete blood count, and high-sensitivity C-reactive protein (hsCRP).

Definition of metabolic syndrome

Participants were screened for MetS according to the diagnostic criteria proposed by the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III [34, 35] and the World Health Organization (WHO) obesity criteria for Asian populations [36]. The criteria were as follows: waist circumference ≥ 90 cm in men and ≥ 80 cm in women; systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg, or the patient is already under treatment for hypertension; fasting blood glucose levels ≥ 100 mg/dL, or the patient is already under treatment for elevated glucose; elevated triglyceride levels ≥ 150 mg/dL, or the patient is already under treatment for hypertriglyceridemia; HDL-C levels < 40 mg/dL in men or < 50 mg/dL in women. Participants who met three or more of the above criteria were categorized as having MetS.

Evaluation of cognitive function

Cognitive function was evaluated with the Eight-item Informant Interview to Differentiate Aging and Dementia (AD-8) [37, 38] and Montreal Cognitive Assessment (MoCA) Taiwanese Version (MoCA-T) [39, 40]. AD-8 is a widely used instrument to screen for early dementia. The AD-8 test assesses cognitive abilities including memory, orientation, judgment, and function. Scores of 2 or higher on the AD-8, indicating cognitive impairment [37], have been previously validated for the Taiwanese population [41]. The MoCA evaluates short-term memory recall, visuospatial function, executive function, sustained attention, language, and orientation. It is highly sensitive and specific for differentiating between normal cognitive aging, mild cognitive impairment, and Alzheimer's disease [40]. According to a validation study of the MoCA-T on the Taiwanese population, a cut-off value of 23/24 was found to be optimal for differentiating mild cognitive impairment [39]. In this study, MoCA-T was utilized rather than MMSE, as a previous study demonstrated MoCA to be more sensitive and specific for screening cognitive impairment in older adults [26].

Statistical analysis

Descriptive data were presented as mean ± standard deviation (SD). Categorical variables were presented as number (%) of subjects and were analyzed using Chi-square tests. Comparisons between abnormal and normal populations in each category of MetS criteria and AD-8 and MoCA-T results were analyzed with the Chi-square test. Cognitive function was categorized by MoCA-T and AD-8 as normal (MoCA-T ≥ 24, AD-8 < 2) or cognitive impairment (MoCA-T < 24, AD-8 ≥ 2). Multivariate logistic regression models were used to analyze the associations between MetS status and cognitive function. Possible confounders that may affect cognitive function were adjusted, including age, sex, education level, marital status, income status, history of stroke, and scores on IADL and GDS-15. The statistical analyses were conducted using IBM SPSS version 20.0 (IBM Corporation, Armonk, NY, USA). A two-tailed p value < 0.05 was considered as statistically significant.

Results

We recruited 567 patients who were older than 50 years old. The mean age of the study population is 66.3 ± 9.1 years (Table 1). Of the participants, 323 (56.97%) were older people (65 years or older). 223 (39.3%) participants were male. 415 participants (73.2%) were married. 311 participants (54.9%) had an education level of university and above. More than half of the participants did not have income (63.5%). On average, the participants had good physical function (mean score ± SD of Barthel index and IADL were 99.6 ± 3.5 and 23.5 ± 2.3, respectively) and fair cognitive level (mean score ± SD of MoCA-T and AD-8 were 25.2 ± 4.1 and 0.59 ± 1.46, respectively). Participants did not have a tendency of depression or inflammation (mean score ± SD of GDS-15 and hs-CRP were 1.7 ± 2.4 and 0.13 ± 0.17, respectively). However, about half of the participants (54.3%) reported pain.

The prevalence of MetS of the study population was 34.2% (n = 194, Table 2). The prevalence of MetS components (abdominal obesity, hypertension or on drug treatment; hyperglycemia or on drug treatment; hypertriglyceridemia or on drug treatment; and low HDL-C or on drug treatment) in the study population was 44.8% (n = 254), 59.4% (n = 337), 37.9% (n = 215), 24.7% (n = 140), and 33.9% (n = 192), respectively. Whether cognitive function is categorized by MoCA-T or AD-8, there were no differences between cognitive function status in abdominal obesity, hypertension or on drug treatment, hyperglycemia or on drug treatment, hypertriglyceridemia or on drug treatment, low HDL-C or on drug treatment, and MetS status.

Table 3 showed the association between MetS status with cognitive function. The odds of mild cognitive impairment increased with age (aOR = 1.08, 95% CI = 1.04–1.11, p < 0.001). Conversely, a higher education

Table 1 Basic characteristics of study population

	Total (*n* = 567)
Age (year), mean (SD)	66.3 (9.1)
Male, *n* (%)	223 (39.3)
Married, *n* (%)	415 (73.2)
Education level, *n* (%)	
Below junior high school	95 (16.8)
Senior high school	161 (28.4)
University and above	311 (54.9)
No income, *n* (%)	360 (63.5)
Smoking status, *n* (%)	
Never smoker	484 (85.4)
Secondhand smoker	6 (1.1)
Current smoker	34 (6)
< 20 pack-years	16 (2.8)
≥ 20 pack-years	18 (3.2)
Former smoker	43 (7.6)
Alcohol consumption, *n* (%)	105 (18.5)
Heavy drinking (15 or more drinks/week for men; 8 or more drinks/week for women)	9 (1.6)
Barthel index, mean (SD)	99.6 (3.5)
IADL score, mean (SD)	23.5 (2.3)
MoCA-T, mean (SD)	25.2 (4.1)
AD-8, mean (SD)	0.59 (1.46)
GDS-15, mean (SD)	1.7 (2.4)
Level of hs-CRP (mg/dL), mean (SD)	0.13 (0.17)
Chronic conditions, *n* (%)	
Pain	308 (54.3)
Degenerative joint disease	108 (19.0)
Malignancy	72 (12.7)
Depression	44 (7.8)
Lung diseases	25 (4.4)
Stroke	6 (1.1)

Abbreviations: IADL, instrumental activities of daily living; MoCA-T, the Montreal Cognitive Assessment, Taiwanese version; AD-8, The Eight-item Informant Interview to Differentiate Aging and Dementia; GDS-15, Geriatric Depression Scale-15; hs-CRP, high sensitivity C-reactive protein

level, including college and graduate school, and a higher score on the IADL were found to be protective factors against mild cognitive impairment [aOR (95% CI) were 0.44 (0.36–0.55) and 0.74 (0.61–0.91), respectively, $p < 0.05$]. Similar to the results for mild cognitive impairment, a higher education level of college and graduate school, and a higher score on the IADL were also identified as protective factors against mild dementia [aOR with 95% CI were 0.66 (0.48–0.91) and 0.73 (0.63–0.84), respectively, $p < 0.05$]. Additionally, participants with a higher score on the GDS-15 were more likely to have mild dementia (aOR = 1.59, 95% CI = 1.40–1.80, $p < 0.001$). There were no significant associations between MetS and mild cognitive impairment or mild dementia.

For the associations between MetS components and cognitive function, almost all components of MetS showed no significant correlation with mild cognitive impairment or mild dementia. However, abdominal obesity exhibited a protective effect against mild dementia after adjusting age, sex, education level, marital status, income status, stroke history, and scores of IADL and GDS-15 (aOR = 0.68, 95% CI = 0.50–0.95, $p = 0.02$, Table 4). We further conducted a subgroup analysis stratified by sex. Table 5 demonstrates that abdominal obesity has a significant protective effect against mild dementia in women but not in men (aOR = 0.62, 95% CI = 0.42–0.93, $p = 0.02$ in women).

We additionally conducted a subgroup analysis to explore the association between waist circumference and the risk of mild dementia, stratified by sex. Both men and women were quintiled based on the sample size in each group. Figure 1 illustrates that women with a waist circumference ranging from 82 to 89 cm had a reduced risk of mild dementia compared to women with a waist circumference of less than 73 cm (aOR = 0.32, 95% CI = 0.12, 0.84, $p = 0.02$). No significant association was found between other quintiles of waist circumference and the risk of mild dementia (Fig. 2).

Discussion

In this study, we did not observe a significant correlation between MetS status and impaired cognition as indicated by both MoCA-T and AD-8. However, we did find that an optimal waist circumference served as a protective factor against mild dementia in female community-dwelling older adults. Our study revealed gender differences in the effects of MetS components on cognition, a finding less explored in prior research.

MetS was not associated with mild cognitive impairment (MCI) (MoCA < 24) and mild dementia (AD-8 ≥ 2) in this cross-sectional study. Our study population was predominantly older people, which may explain the insignificant correlation between MetS and cognition in our study. In prior published findings, MetS increased the risk for developing MCI [42] and for the progression of MCI to dementia in middle-aged adults (55 years or older) [21] but not in the oldest old (75 years or older) [22, 24]. Another study regarding the influence of MetS in patients with Alzheimer's disease and at least 70 years of age found that cognitive performance was worse but not statistically significant in the group with MetS [43]. Because low measurements of blood pressure, adiposity, and serum lipid levels are associated with poor physical health and cognitive performance in older people [44], MetS in older age may not exert significant detrimental effects on cognition as it does in younger ages. Therefore, older adults with MetS in our study population may be

Table 2 The associations between status of metabolic syndrome and cognitive function by Chi-square tests

	MoCA-T			AD-8			Total *n* = 567
	Normal (MoCA-T ≥ 24) *n* = 394 (69.5%) *n* (%)	Cognitive impairment (MoCA-T < 24) *n* = 173 (30.5%) *n* (%)	*p*	Normal (AD-8 < 2) n = 494 (87.1%) *n* (%)	Cognitive impairment (AD-8 ≥ 2) 73 (12.9%) *n* (%)	*p*	
Abdominal obesity							
Normal	228 (40.2)	85 (15.0)	0.05	272 (48.0)	41 (7.2)	0.86	313 (55.2)
Abnormal	166 (29.3)	88 (15.5)		222 (39.2)	32 (5.6)		254 (44.8)
Hypertension or on drug treatment							
Normal	170 (30.0)	60 (10.6)	0.06	202 (35.6)	28 (4.9)	0.68	230 (40.6)
Abnormal	224 (39.5)	113 (19.9)		292 (51.5)	45 (7.9)		337 (59.4)
Hyperglycemia or on drug treatment							
Normal	248 (43.7)	104 (18.3)	0.52	312 (55.0)	40 (7.1)	0.17	352 (62.1)
Abnormal	146 (25.8)	69 (12.2)		182 (32.1)	33 (5.8)		215 (37.9)
Hypertriglyceridemia or on drug treatment							
Normal	295 (52.0)	132 (23.3)	0.72	377 (66.5)	50 (8.8)	0.15	427 (75.3)
Abnormal	99 (17.5)	41 (7.2)		117 (20.6)	23 (4.1)		140 (24.7)
Low HDL-C or on drug treatment							
Normal	264 (46.6)	111 (19.6)	0.51	326 (57.5)	49 (8.6)	0.85	375 (66.1)
Abnormal	130 (22.9)	62 (10.9)		168 (29.3)	24 (4.2)		192 (33.9)
Metabolic syndrome							
No	267 (47.1)	106 (18.7)	0.13	328 (57.9)	45 (7.9)	0.42	373 (65.8)
Yes	127 (22.4)	67 (11.8)		166 (29.3)	28 (4.9)		194 (34.2)

Abbreviations: MoCA-T, the Montreal cognitive assessment, Taiwanese version; AD-8, the eight-item informant interview to differentiate aging and dementia; HDL-C, high-density lipoprotein cholesterol

less susceptible to the effects of MetS on general health and cognitive function.

We found women with a waist circumference of 82 to 89 cm were less likely to have mild dementia. Similarly, a study investigating the correlation of BMI and waist circumference with impaired cognition in older Chinese adults found that abdominal obesity defined by waist circumference [45] was a protective factor against cognitive decline in both women and men. Past literature has shown that nutritional intake patterns are important factors to be considered. Consumption of omega-6 and omega-3 polyunsaturated fatty acids, including docosahexaenoic acid, eicosapentaenoic acid, and arachidonic acid, may contribute to central obesity but are crucial for maintaining nervous system metabolism and function [46]. Our study found that the protective effects of waist circumference on mild dementia were observed within the range of 82–89 cm, rather than with unlimited increases in waist circumference. This indicates that optimal nutrition is beneficial to cognitive function. A randomized controlled trial that evaluated nutritional habits in middle-aged and older women and assessed their cognitive function throughout the duration of the study [47] found that differences in consumption levels of different fat types, rather than of total fat, affected cognitive changes in aging. Observations from this study indicated that higher intake of saturated fatty acids correlated to poor cognitive trajectories in older age, while larger intake of mono-unsaturated fatty acids was protective against impaired cognition. Unlimited abdominal obesity poses risks for cognitive decline, while appropriate nutritional support is helpful for neuronal survival and neurotransmission, thus modulating the decline of cognitive function in aging [48].

Our observation of central adiposity acting as a protective factor against cognitive decline specifically in women aligned with evidence from a study in an Icelandic population [49]. This study found that larger amounts of subcutaneous fat in the abdomen and thigh and late-life obesity decreased the likelihood of developing dementia for women, while greater amounts of visceral fat contributed to the deterioration of cognitive function. Another possible mechanism of the inverse relationship between central obesity and development of impaired cognition

Table 3 Factors associated with cognitive impairment by logistic regression

	Cognitive impairment by MoCA-T		Cognitive impairment by AD-8	
	aOR (95% CI)	***p***	**aOR (95% CI)**	***p***
Age	1.08 (1.04, 1.11)	< 0.001	1.00 (0.96, 1.04)	0.84
Sex				
Female	Ref		Ref	
Male	0.87 (0.69, 1.10)	0.24	1.14 (0.82, 1.60)	0.44
Education level				
Below high school	Ref		Ref	
College and graduate school	0.44 (0.36, 0.55)	< 0.001	0.66 (0.48, 0.91)	0.01
Marital status				
Married	Ref		Ref	
Single, widowed, divorced, and other	1.21 (0.96, 1.53)	0.1	1.34 (0.96, 1.86)	0.08
Monthly income				
No income	Ref		Ref	
Have income	1.18 (0.89, 1.55)	0.25	1.01 (0.68, 1.52)	0.95
IADL score	0.74 (0.61, 0.91)	0.005	0.73 (0.63, 0.84)	< 0.001
GDS-15 score	1.05 (0.96, 1.14)	0.25	1.59 (1.40, 1.80)	< 0.001
Stroke history	1.15 (0.43, 3.12)	0.78	1.60 (0.53, 4.87)	0.40
Metabolic syndrome	0.92 (0.74, 1.15)	0.47	0.80 (0.58, 1.12)	0.20

Mild cognitive impairment was diagnosed as MoCA-T score < 24. Mild dementia was screened as AD-8 ≥ 2

Abbreviations: aOR, adjusted odds ratio; IADL, instrumental activities of daily living; GDS-15, Geriatric Depression Scale-15; MoCA-T, the Montreal Cognitive Assessment, Taiwanese version; AD-8, The Eight-item Informant Interview to Differentiate Aging and Dementia

Table 4 The association between components of metabolic syndrome and cognitive function

	Cognitive impairment by MoCA-T		Cognitive impairment by AD-8	
	aOR (95% CI)	***p***	**aOR (95% CI)**	***p***
Abdominal obesity	0.98 (0.79, 1.20)	0.81	0.68 (0.50, 0.95)	0.02
Hypertension or on drug treatment	0.97 (0.78, 1.20)	0.77	0.99 (0.72, 1.37)	0.96
Hyperglycemia or on drug treatment	0.98 (0.79, 1.22)	0.87	1.18 (0.87, 1.61)	0.29
Hypertriglyceridemia or on drug treatment	0.87 (0.68, 1.12)	0.28	1.01 (0.72, 1.41)	0.95
Low HDL-C or on drug treatment	1.01 (0.82, 1.26)	0.91	0.88 (0.64, 1.22)	0.44

Cognitive impairment was defined as MoCA-T < 24 and AD-8 ≥ 2

The statistics were performed using logistic regression models, adjusting for age, sex, education level, marital status, income status, stroke history, and scores of instrumental activities of daily living and Geriatric Depression Scale-15

Abbreviations: aOR, adjusted odds ratio; HDL-C, high-density lipoprotein cholesterol; MoCA-T, the Montreal Cognitive Assessment, Taiwanese version; AD-8, The Eight-item Informant Interview to Differentiate Aging and Dementia

in women is the production of endogenous hormones by adipose tissue [50], which is the main hormone production mechanism in postmenopause. Two endogenous hormones secreted by adipose, estrogen [51] and leptin [52], have been demonstrated to lower the risk of cognitive decline via their involvement in memory and learning pathways in the brain. Production of estrogen and leptin may be greater in older women with abdominal obesity, thus offering protection against cognitive decline that women without central obesity and men would not have. Currently, it is unclear whether ethnicity affects abdominal obesity as a protective factor against cognitive decline, and our study contributes to the evidence from Asia.

There are some limitations in this study. First, the sample population of this study is limited to adults who receive care at the outpatient clinic at TVGH, which restricts the generalizability of our findings to other community-dwelling adults. Second, dietary habits are important factors correlated with cognitive function but are unavailable in this study. Not analyzing dietary habits may lead to a misinterpretation of the protective effect

Table 5 The associations of abdominal obesity and cognitive impairment stratified by sex

	Men ($n=223$)		Women ($n=344$)	
	aOR (95% CI)	***p***	**aOR (95% CI)**	***p***
Age	1.01 (0.94, 1.08)	0.84	0.98 (0.93, 1.04)	0.59
Education level				
Below high school			ref	
College and graduate school	0.66 (0.38, 1.14)	0.14	0.63 (0.42, 0.95)	0.03
Marital status				
Married			ref	
Single, widowed, divorced, and other	0.58 (0.20, 1.65)	0.31	1.62 (1.11, 2.38)	0.01
Monthly income				
No income			ref	
Have income	0.82 (0.40, 1.70)	0.60	1.02 (0.61, 1.71)	0.93
IADL score	0.75 (0.61, 0.93)	0.008	0.72 (0.59, 0.88)	0.001
GDS-15 score	1.68 (1.32, 2.13)	< 0.001	1.57 (1.35, 1.84)	< 0.001
Stroke history	2.33 (0.43, 12.5)	0.33	1.25 (0.22, 7.04)	0.80
Abdominal obesity	0.84 (0.46, 1.53)	0.58	0.62 (0.42, 0.93)	0.02

Cognitive impairment was screened as AD-8 ≥ 2. Cognitive impairment indicated by MoCA-T score was not significantly associated with abdominal obesity (Table 4), and therefore was not included in this analysis

Abbreviations: aOR, adjusted odds ratio; IADL, instrumental activities of daily living; GDS-15, Geriatric Depression Scale-15; AD-8, The Eight-item Informant Interview to Differentiate Aging and Dementia

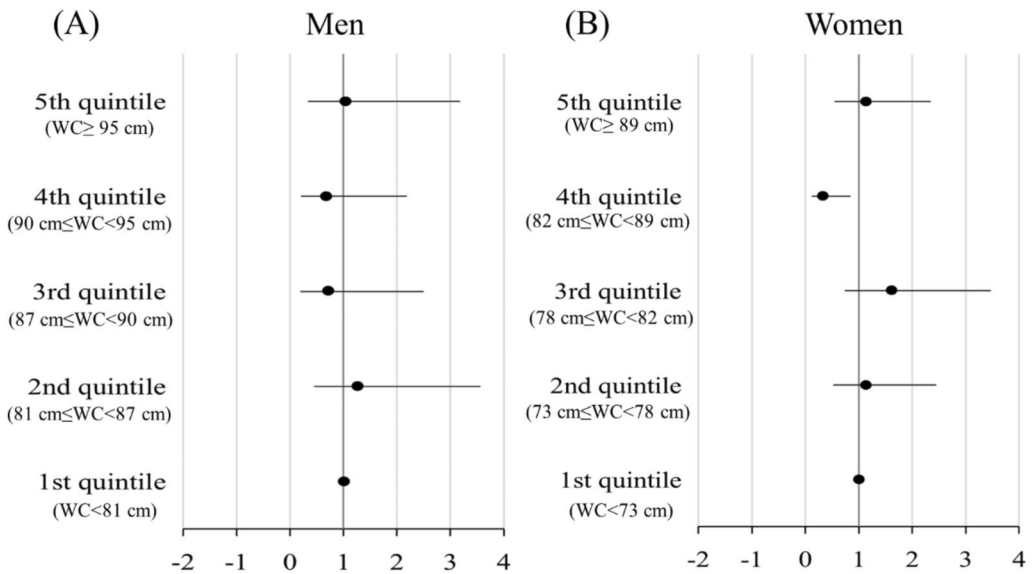

Fig. 2 Subgroup analysis of the association between waist circumference and the risk of cognitive impairment stratified by sex, expressed as adjusted odds ratio and 95% CI. The waist circumference was divided into five groups based on the sample size in each group. Cognitive impairment was screened as AD-8 ≥ 2. The statistics were performed using logistic regression models, adjusting for age, sex, education level, marital status, income status, stroke history, and scores of instrumental activities of daily living and Geriatric Depression Scale-15. Abbreviations: WC, Waist circumference; AD-8, The Eight-Item Informant Interview to Differentiate Aging and Dementia

of abdominal obesity on mild dementia in female participants. Finally, the cross-sectional design did not allow for the interpretation of causal inferences. Despite these limitations, the study has several strengths. First, we used MoCA-T and AD-8 to evaluate cognitive function. These tools are sensitive and well-validated in the Taiwanese population. Second, we adjusted for important confounders related to cognitive function in the analytical models.

Finally, we found an optimal waist circumference is correlated to a lower risk of mild dementia in women, which is a novel finding in this field.

Conclusion

We identified an optimal waist circumference as a protective factor against mild dementia in female community-dwelling older adults. The potential roles of dietary factors and hormones in neuroprotection warrant further investigation. Our findings contribute to preventive strategies of cognitive impairment in the global aging society.

Abbreviations

AD-8	Eight-item informant interview to differentiate aging and dementia
ADL	Activity of daily living
BMI	Body mass index
DJD	Degenerative joint diseases
DM	Diabetes mellitus
GDS-15	Geriatric depression Scale 15
HDL-C	High-density lipoprotein cholesterol
HFD	High fat diet
hsCRP	High-sensitivity C-reactive protein
IADL	Independent activities of daily living
IQR	Interquartile range
MCI	Mild cognitive impairment
MetS	Metabolic syndrome
MMSE	Mini-mental state examination
MoCA	Montreal cognitive assessment
MoCA-T	Montreal cognitive assessment taiwanese version
NCCN	National comprehensive cancer network
NIAAA	National institute on alcohol abuse and alcoholism
SD	Standard deviation
T1DM	Type 1 DM
T2DM	Type 2 DM
TVGH	Taipei veterans general hospital
WHO	World health organization

Acknowledgements

The authors would like to thank the participants for their time and contribution to the study, and the Taipei Veterans General Hospital for the use of its facilities. No external organizations had roles in the study design, data collection and analysis, or manuscript preparation.

Author contributions

HTC, SJH, SJW, and JLF contributed to conceptualization and funding acquisition for this study. SRL acquired and processed the data. YHL, HTC, SRL, and SCC carried out statistical analyses. HYY, YHL, SRL, MHL, TJC, and HTC contributed to the interpretation of the results. HYY prepared the manuscript with important feedback and suggestions from HTC, YFW, and YHL.

Funding

This study was funded by Taipei Veterans General Hospital, [Grant number: V109E-005–4, V110E-005–4, V111E-006–4, V112E-004–4].

Availability of data and materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The protocol of this study was approved by the Research Ethics Committee of Taipei Veterans General Hospital (protocol code: 2020–06-001A).

Consent for publication

Not applicable.

Competing Interests

The authors declare no competing interests.

Author details

[1]School of Medicine, College of Medicine, National Yang Ming Chiao Tung University, Taipei, Taiwan. [2]Department of Medical Education, Taipei Veterans General Hospital, Taipei, Taiwan. [3]Department of Neurology, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan. [4]Department of Internal Medicine, Taipei Veterans General Hospital, Taipei, Taiwan. [5]Department of Family Medicine, Taipei Veterans General Hospital, Taipei, Taiwan. [6]Department of Family Medicine, Taipei Veterans General Hospital Hsinchu Branch, Hsinchu County, Taiwan. [7]Department of Post-Baccalaureate Medicine, National Chung Hsing University, Taichung, Taiwan. [8]Department of Family Medicine, En Chu Kong Hospital, New Taipei City, Taiwan.

Received: 25 July 2024 Accepted: 11 March 2025
Published online: 29 March 2025

References

1. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement: Executive Summary. Crit Pathw Cardiol. 2005;4(4):198–203.
2. Martinez-Miller EE, Kohl HW 3rd, Barlow CE, Willis BL, DeFina LF. Metabolic Syndrome and Cognitive Impairment among High Socioeconomic, Nondemented Older US Adults. J Am Geriatr Soc. 2019;67(7):1437–43.
3. Wahl D, Solon-Biet SM, Cogger VC, Fontana L, Simpson SJ, Le Couteur DG, et al. Aging, lifestyle and dementia. Neurobiol Dis. 2019;130:104481.
4. Xu J, Bartolome CL, Kong D. Synaptic regulation of metabolism. Adv Exp Med Biol. 2018;1090:49–77.
5. Chen Y, Knight ZA. Making sense of the sensory regulation of hunger neurons. BioEssays. 2016;38(4):316–24.
6. Chen Y, Lin YC, Zimmerman CA, Essner RA, Knight ZA. Hunger neurons drive feeding through a sustained, positive reinforcement signal. Elife. 2016;5:18640.
7. Atti AR, Valente S, Iodice A, Caramella I, Ferrari B, Albert U, et al. Metabolic syndrome, mild cognitive impairment, and dementia: a meta-analysis of longitudinal studies. Am J Geriatr Psych. 2019;27(6):625–37.
8. Tsai CK, Kao TW, Lee JT, Wu CJ, Hueng DY, Liang CS, et al. Increased risk of cognitive impairment in patients with components of metabolic syndrome. Medicine (Baltimore). 2016;95(36):e4791.
9. Yaffe K. Metabolic syndrome and cognitive decline. Curr Alzheimer Res. 2007;4(2):123–6.
10. Fulop T, Witkowski JM, Olivieri F, Larbi A. The integration of inflammaging in age-related diseases. Semin Immunol. 2018;40:17–35.
11. Toth P, Tarantini S, Csiszar A, Ungvari Z. Functional vascular contributions to cognitive impairment and dementia: mechanisms and consequences of cerebral autoregulatory dysfunction, endothelial impairment, and neurovascular uncoupling in aging. Am J Physiol Heart Circ Physiol. 2017;312(1):H1–20.
12. Akiguchi I, Yamamoto Y. Vascular mechanisms of cognitive impairment: roles of hypertension and subsequent small vessel disease under sympathetic influences. Hypertens Res. 2010;33(1):29–31.
13. Toth C, Schmidt AM, Tuor UI, Francis G, Foniok T, Brussee V, et al. Diabetes, leukoencephalopathy and rage. Neurobiol Dis. 2006;23(2):445–61.
14. Pais R, Ruano L, Carvalho OP, Barros H. Global cognitive impairment prevalence and incidence in community dwelling older adults-a systematic review. Geriatrics (Basel). 2020;5(4):84.
15. Pérez Palmer N, Trejo Ortega B, Joshi P. Cognitive impairment in older adults: epidemiology, diagnosis, and treatment. Psychiatr Clin North Am. 2022;45(4):639–61.
16. Overdorp EJ, Kessels RP, Claassen JA, Oosterman JM. The combined effect of neuropsychological and neuropathological deficits on instrumental activities of daily living in older adults: a systematic review. Neuropsychol Rev. 2016;26(1):92–106.
17. Artero S, Touchon J, Ritchie K. Disability and mild cognitive impairment: a longitudinal population-based study. Int J Geriatr Psych. 2001;16(11):1092–7.

18. Fernando E, Fraser M, Hendriksen J, Kim CH, Muir-Hunter SW. Risk Factors associated with falls in older adults with dementia: a systematic review. Physiother Can. 2017;69(2):161–70.
19. Ambrose AF, Paul G, Hausdorff JM. Risk factors for falls among older adults: a review of the literature. Maturitas. 2013;75(1):51–61.
20. Grande G, Qiu C, Fratiglioni L. Prevention of dementia in an ageing world: Evidence and biological rationale. Ageing Res Rev. 2020;64:101045.
21. Ng TP, Feng L, Nyunt MSZ, Feng L, Gao Q, Lim ML, et al. Metabolic syndrome and the risk of mild cognitive impairment and progression to dementia: follow-up of the Singapore longitudinal ageing study cohort. JAMA Neurol. 2016;73(4):456–63.
22. Van den Berg E, Biessels G, De Craen A, Gussekloo J, Westendorp R. The metabolic syndrome is associated with decelerated cognitive decline in the oldest old. Neurology. 2007;69(10):979–85.
23. Alsuwaidi HN, Ahmed AI, Alkorbi HA, Ali SM, Altarawneh LN, Uddin SI, et al. Association between metabolic syndrome and decline in cognitive function: a cross-sectional study. Diabetes Metab Syndr Obes. 2023;16:849–59.
24. Liu C-L, Lin M-H, Peng L-N, Chen L-K, Su C-T, Liu L-K, et al. Late-life metabolic syndrome prevents cognitive decline among older men aged 75 years and over: one-year prospective cohort study. J Nutr Health Aging. 2013;17(6):523–6.
25. Chang Y-M, Lee C-L, Wang J-S. Sex disparity in the association of metabolic syndrome with cognitive impairment. J Clin Med. 2024;13(9):2571.
26. Ciesielska N, Sokolowski R, Mazur E, Podhorecka M, Polak-Szabela A, Kedziora-Kornatowska K. Is the montreal cognitive assessment (MoCA) test better suited than the mini-mental state examination (MMSE) in mild cognitive impairment (MCI) detection among people aged over 60? Meta-Analysis Psychiatr Pol. 2016;50(5):1039–52.
27. Hirode G, Wong RJ. Trends in the prevalence of metabolic syndrome in the United States, 2011–2016. JAMA. 2020;323(24):2526–8.
28. Zhuang L, Yang Y, Gao J. Cognitive assessment tools for mild cognitive impairment screening. J Neurol. 2021;268(5):1615–22.
29. Wood DE, Kazerooni EA, Aberle DR, Argento C, Baines J, Boer B, et al. NCCN guidelines® insights: lung cancer screening, version 1 2025. J Natl Comprehens Cancer Netw. 2025;23(1):0002.
30. National Institute on Alcohol Abuse and Alcoholism (NIAAA). Drinking Levels Defined 2025 [updated February 2025. Available from: https://www.niaaa.nih.gov/alcohols-effects-health/alcohol-drinking-patterns.
31. Sheikh JI, Yesavage JA. Geriatric depression scale (GDS): recent evidence and development of a shorter version. Clin Gerontol J Aging Mental Health. 1986;5:165–73.
32. Mahoney FI, Barthel DW. Functional evaluation: the barthel index. Md State Med J. 1965;14:61–5.
33. Lawton MP, Brody EM. Assessment of older people: self-maintaining and instrumental activities of daily living. Gerontologist. 1969;9(3):179–86.
34. National Cholesterol Education Program Expert Panel on Detection E, Treatment of High Blood Cholesterol in A. Third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. Circulation. 2002;106(25):3143–421.
35. Expert Panel on Detection E. Executive summary of the third report of gram (NCEP) expert panel on detection, evaluation, and treatment of hithe national cholesterol education progh blood cholesterol in adults (adult treatment panel III). Jama. 2001;285(19):2486–97.
36. Inoue S, Zimmet P, Caterson I, Chunming C, Ikeda Y, Khalid A, et al. The Asia-Pacific perspective: redefining obesity and its treatment. Sydney: Health Communications Australia Pty Ltd.; 2000.
37. Hendry K, Green C, McShane R, Noel-Storr AH, Stott DJ, Anwer S, et al. AD-8 for detection of dementia across a variety of healthcare settings. Cochr Database Syst Rev. 2019;3(3):11121.
38. Chen HH, Sun FJ, Yeh TL, Liu HE, Huang HL, Kuo BI, et al. The diagnostic accuracy of the Ascertain Dementia 8 questionnaire for detecting cognitive impairment in primary care in the community, clinics and hospitals: a systematic review and meta-analysis. Fam Pract. 2018;35(3):239–46.
39. Tsai CF, Lee WJ, Wang SJ, Shia BC, Nasreddine Z, Fuh JL. Psychometrics of the montreal cognitive assessment (MoCA) and its subscales: validation of the Taiwanese version of the MoCA and an item response theory analysis. Int Psychogeriatr. 2012;24(4):651–8.
40. Nasreddine ZS, Phillips NA, Bedirian V, Charbonneau S, Whitehead V, Collin I, et al. The montreal cognitive assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005;53(4):695–9.
41. Yang Y-H, Galvin JE, Morris JC, Lai C-L, Chou M-C, Liu C-K. Application of AD8 questionnaire to screen very mild dementia in Taiwanese. Am J Alzheimer's Dis Other Dement. 2011;26(2):134–8.
42. Marseglia A, Darin-Mattsson A, Skoog J, Ryden L, Hadarsson-Bodin T, Kern S, et al. Metabolic syndrome is associated with poor cognition: a population-based study of 70-year-old adults without dementia. J Gerontol A Biol Sci Med Sci. 2021;76(12):2275–83.
43. Hishikawa N, Fukui Y, Sato K, Kono S, Yamashita T, Ohta Y, et al. Cognitive and affective functions in Alzheimer's disease patients with metabolic syndrome. Eur J Neurol. 2016;23(2):339–45.
44. Forti P, Pisacane N, Rietti E, Lucicesare A, Olivelli V, Mariani E, et al. Metabolic syndrome and risk of dementia in older adults. J Am Geriatr Soc. 2010;58(3):487–92.
45. Ren Z, Li Y, Li X, Shi H, Zhao H, He M, et al. Associations of body mass index, waist circumference and waist-to-height ratio with cognitive impairment among Chinese older adults: Based on the CLHLS. J Affect Disord. 2021;295:463–70.
46. Goyal MS, Iannotti LL, Raichle ME. Brain Nutrition: A Life Span Approach. Annu Rev Nutr. 2018;38:381–99.
47. Okereke OI, Rosner BA, Kim DH, Kang JH, Cook NR, Manson JE, et al. Dietary fat types and 4-year cognitive change in community-dwelling older women. Ann Neurol. 2012;72(1):124–34.
48. Bazinet RP, Laye S. Polyunsaturated fatty acids and their metabolites in brain function and disease. Nat Rev Neurosci. 2014;15(12):771–85.
49. Spauwen PJ, Murphy RA, Jonsson PV, Sigurdsson S, Garcia ME, Eiriksdottir G, et al. Associations of fat and muscle tissue with cognitive status in older adults: the AGES-Reykjavik Study. Age Ageing. 2017;46(2):250–7.
50. Szymczak J, Milewicz A, Thijssen JH, Blankenstein MA, Daroszewski J. Concentration of sex steroids in adipose tissue after menopause. Steroids. 1998;63(5–6):319–21.
51. Yaffe K, Lui LY, Grady D, Cauley J, Kramer J, Cummings SR. Cognitive decline in women in relation to non-protein-bound oestradiol concentrations. Lancet. 2000;356(9231):708–12.
52. Gustafson DR. Adiposity and cognitive decline: underlying mechanisms. J Alzheimers Dis. 2012;30(Suppl 2):S97-112.

Publisher's Note