



THE RELATIONSHIP BETWEEN CHRONIC DISEASES AND COGNITIVE FUNCTION IN PATIENT WITH CHRONIC DISEASES: AN ANALYSIS BASED ON THE 2014 INDONESIAN FAMILY LIFE SURVEY DATA

Fira Awanis Hazrina^{1*}, Rohman Azzam¹, Yani Sofiani¹, Yuni Shahroh², Bayu Satria Wiratama³, Ninik Yunitri⁴

¹School of Nursing, Universitas Muhammadiyah Jakarta, Jl Cempaka Putih Tengah No.27, Cempaka Putih Timur, Cemp. Putih, Jakarta Pusat, Jakarta 10510, Indonesia.

²Prof. Dr. Sulianti Saroso Hospital for Infectious Diseases, Jl Kalibaru Timur 8F, Cilincing, Jakarta Utara, Jakarta 14110, Indonesia

³Department of Biostatistics, Epidemiology and Population Health, Universitas Gadjah Mada, Bulaksumur, Caturtunggal, Depok, Sleman, Yogyakarta 55281, Indonesia

⁴Mental Health and Psychiatric Nursing Department, Faculty of Nursing, Universitas Muhammadiyah Jakarta, Jl. K.H. Ahmad Dahlan, Cireundeu, Ciputat Timr, Tangerang Selatan, Banten 15419, Indonesia

*firaawanish@gmail.com

ABSTRACT

Cognitive decline known as one of long-term effect of chronic disease. Individuals with cognitive dysfunction tend to show low quality of life. Yet limited studies has been conducted to determine the prevalence of this issue among people with chronic disease in Indonesia. Therefore, this study was aimed to identify the prevalence and relationship between chronic disease and cognitive function. This study also evaluated the factors influencing this relationship. This research was a cross-sectional analysis that utilized data from 2,396 participants with chronic conditions, drawn from the 2014 Indonesian Family Life Survey (IFLS) through purposive sampling. This study focus on people with hipertention, stroke, and diabetes mellitus. Cognitive impairment was measured using Telephone Interview for Cognitive Status (TICS). The data was analysed using analysed using bivariate logistic regression and multivariate Hierarchical Logistic Regression. The relationship between variables indicated based on Odds Ratio (OR), exponential value values β ($e\beta$), and significancy level 5%. The overall prevalence of impaired cognitive function was 2171(61.7%) with the highest frequency found in people with diabetes mellitus (64.5%). Logistic regression test found a significant relationship between chronic disease and impaired function ($p<0.001$). Hierarchical Logistic Regression test on all models found-that age, gender, disease duration, and education had a greater influence, with R^2 0.467 (46.7%). This study showed people with chronic diseases with older age, female, low education, longer disease duration, smoker, and obese have higher risk of developing impaired cognitive function.

Keywords: chronic disease; cognitive function; IFLS

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INTRODUCTION

Chronic diseases are the leading cause of illness, disability and death in the United States (CDC, 2023). An estimated 129 million people in the US have at least one major chronic disease (Boersma et al., 2020). Chronic diseases are among the ten most common and costly diseases (Benavidez et al., 2024). Over the past two decades their prevalence has steadily increased, and this trend is expected to continue (Murray et al., 2018). The number of chronic diseases is increasing in Indonesia. Chronic diseases were the leading cause of death in Indonesia in 2016, accounting for 73% of all deaths (WHO, 2020). The increase in the prevalence of chronic diseases in Indonesia in 2013-2018 from 6.9% to 34.1% in the three highest disease sequences, namely hypertension, stroke, and diabetes mellitus (Riskasdas, 2018). Adults aged 45 years or older experience cognitive function reduction with a prevalence of 37.5% due to stroke and 49.0% due to diabetes mellitus (World Health Organization, 2020). Cognitive function usually refers to how a person processes external and internal information, and how they utilize this information to think and make decisions (Liu et al., 2024).

Cognitive deficits adversely affect people with chronic diseases in the community, hindering them from performing daily physical activities (Kim et al., 2019). Chronic disease can cause physical disabilities or functional impairments that limit individual’s ability to lead a healthy and productive life . A person with cognitive dysfunction has difficulty acquiring new information, remembering, focusing, and making decisions (Handajani, 2020). Cognitive function is important for individuals to continue their lives effectively, both in health and illness (Barutcu & Gokce, 2020). Previous studies have included populations with hypertension (Anto et al., 2019; Bao et al., 2022), diabetes mellitus (Teixeira et al., 2020), and stroke (Aam et al., 2020). However to date, no studies have evaluated the overall changes in cognitive function in people with chronic diseases and determine variables that might affect them. Therefore, this study objective were to comprehend the relationship between chronic diseases and cognitive function and to analyze the influencing factors.

METHOD

This is quantitative study, using cross-sectional research design. This study utilizes secondary data sourced from IFLS data of 2014. The research was conducted on September 2024. The study includes people suffering from chronic diseases (hypertension, stroke, and diabetes mellitus) accounting 8003 respondents. The sample was chosen based on inclusion criteria: 1) ≥ 45 years old; 2) diagnosed with chronic diseases (hypertension, stroke, and diabetes mellitus); and 3) measured by TICS. Respondents with missing data were excluded from the results so that the number of samples analyzed was 2396 using the purposive sampling technique. Sociodemographic data were collected using questionnaires from IFLS related to age, gender, education, smoking habits, duration of illness and BMI. This study calculated BMI in kg/m² and adopted the WHO BMI criteria with the Indonesian cut-off points of underweight: <18.4, normal: 18.5-24.9, pre-obese: 25-29.9, obese: ≥30 (WHO, 2004). Smoking habits were summarized into non-smoking and smoking. Education level was categorized into no schooling, low (≤12 years of schooling) and high (>12 years of schooling). This research protocol has been reviewed and approved by the Nursing Research Ethics Committee, Faculty of Nursing Science, University of Muhammadiyah Jakarta Indonesia No. 1774/XII/F,9-UMJ/2024. Data analysis in this study to test the data using univariate analysis, namely the characteristics of respondents. Bivariate analysis of this study used logistic regression, to identify the relationship between chronic diseases and cognitive function indicated by the Odds Ratio (OR) or exponential value β (eβ) and seen from the Pvalue <0.001. As well as conducting multivariate analysis using logistic regression analysis with a Hierarchical Logistic Regression approach to determine the effect of moderators on the relationship of two variables and their effect on the direction or strength of the relationship between variables of age, gender, education, smoking habits, length of suffering and Body Mass Index (BMI) with cognitive function with interpretation R² has a coefficient of determination between 0 and 1.

RESULT

Table 1.
General characteristics of respondents (n=2.396)

Respondent Characteristics		f	%
Gender	Male	875	36.5
	Female	1.521	63.5
Education	Not in School	174	7.3
	≤ 12 Years of Schooling	1.600	66.8
	>12 Years of School	622	25.9
Smoking Habit	No	1.529	63.8
	Yes	867	36.2
<i>Body Mass Index (BMI)</i>			
	Which	274	11.4
	Normal	931	38.8
	Pre-Obesity	835	34.8
	Obesity	356	14.8

Table 2.
Bivariate logistic regression test of age, gender, education, smoking habit, duration of illness and Body Mass Index (BMI) with cognitive function (n=2.396)

	β	Pvalue	OR	95%CI for Exp (B)	
				Lower	Upper
Age (Years)	0.061	<0.001	1.063	1.051	1.075
Duration of Illness (Years)	0.009	0.042	1.009	1.000	1.018
Gender					
Female	1.235	<0.001	3.438	2.880	4.103
Education					
≤12 Years of Education	1.382	<0.001	0.251	0.160	0.394
>12 Years of Education	-5.014	<0.001	0.007	0.004	0.012
Smoking Habit					
Yes	0.352	<0.001	1.422	1.203	1.681
Body Mass Index (BMI)					
Normal body weight	-0.281	0.042	0.755	0.576	0.990
Pre-obesity weight	0.350	0.012	1.420	1.080	1.866
Obesity weight	0.411	0.011	1.509	1.099	2.071

Table 3.
Bivariate logistic regression test of chronic diseases (hypertension, stroke, and diabetes mellitus) with cognitive function (n=2.396).

	β	Pvalue	OR	95%CI for Exp (B)	
				Lower	Upper
Hypertension					
Yes	0.700	<0.001	2.014	1.711	2.370
Stroke					
Yes	1.458	<0.001	4.297	3.619	5.102
Diabetes Mellitus					
Yes	1.358	<0.001	3.887	3.280	4.605

Table 4.
Multivariate test of age, gender, duration of illness, education, smoking habit, and Body Mass Index (BMI) with cognitive function (n=2.396)

Variable	Model 1		Model 2		Model 3		Model 4		Model 5	
	β	OR	β	OR	β	OR	β	OR	β	OR
Age	0.070	1.072	0.069	1.072	0.068	1.071	0.068	1.070	0.074	1.077
Gender	1.346	3.842	1.346	3.841	1.267	3.549	1.248	3.484	1.179	3.250
Duration of Suffering			0.006	1.006	0.015	1.015	0.015	1.015	0.016	1.016
Education					-2.983	0.051	-2.973	0.051	-3.020	0.049
Smoking Habit							0.182	1.200	0.186	1.205
Body Mass Index (BMI)									0.380	1.463
P Value	< 0.001		<0.001		<0.001		<0.001		<0.001	
R ²	0.174		0.175		0.467		0.468		0.482	

DISCUSSION

It was found that most of the respondents were middle age. Middle age approximately occurs in the fifth and sixth decades of human life. Brain structures continue to evolve throughout adulthood, and this is characterized by linear and non-linear trajectories bounded by turning points that may reflect a shifting balance between degenerative and developmental processes. The hippocampus and adjacent parahippocampal gyrus are two regions where volumetric turning points occur in humans during the fifth to sixth decades, and some hippocampal subfields shrink faster and earlier than others. This accelerated decline and interindividual differences in magnitude are associated with overall cognitive status and longitudinal episodic memory decline during the transition from middle age to old age. On a larger scale, brain white matter and ventricular volume both show non-linear decline and growth, respectively, at around the sixth decade of life, and individual variations in

these structures are differentially associated with mild cognitive impairment and Alzheimer's disease (AD) case-control status (Dohm-Hansen et al., 2024). The majority of the study's findings were female. Women may experience faster cognitive decline than men due to differences in sex hormones, structural brain development, genetics, psychosocial factors, lifestyle factors, functional connectivity, and pathology. Women may have a greater burden of small vessel disease, including hyperintense white matter volume, and less structural integrity of axons which in turn leads to more rapid cognitive decline especially in executive function and processing speed. Women also appear to have lower gray matter volumes, so they may be more susceptible to the accelerated gray volume loss that occurs with age and the differential volume loss in certain brain regions that occurs with neurodegenerative diseases (Levine et al., 2021).

In line with previous research, it was found that most respondents had ≤ 12 years of schooling. Education improves cognitive function and reduces the risk of dementia in old age (Lövdén et al., 2020). Low education never surpasses higher education in terms of its impact on cognitive function (Widanarti Setyaningsih et al., 2023). Education provides regular and continuous stimulation for the development of cognitive skills such as logic and reasoning, abstract thinking, as well as the ability to prevent loss of connectivity and improve neuron-to-neuron connections (Rachmawaty, 2022). The higher a person's education, the faster and more responsive the ability to digest information (Wiranti & Sofiana, 2022). It was found that most respondents had suffered a stroke for 4 years. High blood pressure experienced over 10 years is associated with cognitive decline. Hypertension experienced with a longer exposure time is more detrimental than transient hypertension for cognitive function (Sanchez Hoffmann et al., 2020). A decline in cognitive function of 10% or more that occurs between one week and three months post-stroke is associated with worse long-term outcomes, including an approximately two-fold increase in mortality, depressive symptoms, loss of independence, and hospital admissions within one year of the stroke event (Obaid et al., 2020).

Supported by the results of previous studies that most respondents do not have a smoking habit and have a smoking habit. Older people who smoke have a 24 percent higher chance of experiencing cognitive impairment than older people who do not smoke (Muhammad et al., 2021). Smoking can cause damage to blood vessels, increase inflammation, and reduce blood flow to the brain, leading to damage to brain cells and cognitive impairment (Elendu et al., 2023). Quitting smoking significantly improves cardiovascular health and reduces the risk of stroke and cognitive decline (Ovbiagele et al., 2013). Quitting smoking for more than four years can significantly reduce the risk of dementia for up to eight years (Choi et al., 2018). In the world about 1.3 million non-smokers are exposed to cigarette smoke (WHO, 2023). More than 70% of Indonesian active smokers smoke 2-3 times a day in their homes near their non-smoking family members or so-called passive smokers (Holipah et al., 2020). Passive smoking has been associated with the incidence of Aortic Arch Calcification (AAC). AAC has been implicated as a major risk factor for coronary heart disease. Chronic thickening of blood vessels by chemicals from inhaled smoke can constrict blood vessels and cause an increase in heart pressure to supply blood, increasing the risk of hypertension (Akpa et al., 2021). Impaired cognitive function is not limited to individuals with smoking habits. Exposure to secondhand smoke can also damage the cardiovascular system by increasing platelet coagulability, leading to endothelial dysfunction. This endothelial dysfunction may be associated with improper clearance of amyloid beta-peptides through the blood-brain barrier, which plays an important role in cognitive decline (He et al., 2020). This is not much different from the results obtained in previous studies that most respondents have a Body Mass Index (BMI) of normal weight. Pre-obese body weight. Obese individuals show abnormal neural responses to food, taste, and odor, as well as social connectivity, sedentary activity, and functional and cognitive tasks such as decision-making, inhibitory control, learning/memory, and attention. In addition, obesity is associated with altered cortical morphometry, lower gray matter volume, and compromised white matter integrity (Li et al., 2023).

Cognitive impairment in patients with hypertension is associated with an imbalance in autoregulation of cerebral blood flow and cerebral vascular changes. These changes may further lead to brain atrophy or damage caused by macro and micro infarcts, which are predictors of cognitive dysfunction. Infarcts that occur in areas involved in cognition, such as the hippocampus, medial thalamus and frontal lobes, can result in cognitive impairment even when the volume of damage is relatively small (Ladecola & Gottesman, 2019). Furthermore, white matter lesions are defined as damage to the white matter of the brain caused by reduced blood flow to the tissue, this condition can also affect cognition by disrupting connectivity between the anterior thalamus and frontal cortex. Another factor that may lead to cognitive dysfunction in individuals with hypertension is the accumulation of β -amyloid protein in the brain, which may serve as a potential mechanism for cognitive decline (Benjamin et al., 2018). Stroke patients experience impaired cognitive function occurs when in response to cerebral ischemia, inflammatory processes are activated within the brain. Activated microglia and astrocytes release pro-inflammatory cytokines, such as interleukin- 1β (IL- 1β), tumor necrosis factor-alpha (TNF- α), and interleukin-6 (IL-6). Neuroinflammation exacerbates neuronal damage, disrupts synaptic connections, and contributes to neurodegeneration, ultimately leading to cognitive impairment (Elendu et al., 2023). Mechanisms of white matter damage Ischemic stroke can also damage white matter, which is a significant factor in cognitive impairment. Axon injury, demyelination, and disruption of white matter pathways disrupt neuronal connectivity and communication. White matter lesions, such as leukoaraiosis and lacunae infarction, are frequently observed in post-stroke cognitive impairment and contribute to executive dysfunction, information processing deficits, and slowed cognitive speed (Elendu et al., 2023). Cardiac conditions such as angina pectoris, myocardial infarction, coronary heart disease, and other vascular diseases are also associated with decreased cognitive function (Rachmawaty, 2022).

The decline in cognitive function associated with diabetes can be attributed to the fact that glucose and insulin are important regulators of cognitive function (Wrighten et al., 2009). It is known that both acute and chronic hyperglycemia are involved in the outcome of diabetes. Acute hyperglycemia causes pathogenic changes in brain neurons by altering regional cerebral blood flow, leading to oxidative pathogens and subsequent neurological damage. On the other hand, chronic hyperglycemia mediates neurological damage by forming AGEs, which in turn leads to the formation of reactive oxygen species (ROS) and the production of pro-inflammatory cytokines, resulting in microvascular changes and systemic inflammation (Ehtewish et al., 2022). Insulin resistance (IR) and its pathogenesis are widely identified as risk factors for cognitive impairment and dementia, including Alzheimer's disease (AD), independent of diabetes (Ehtewish et al., 2022; Kuusisto et al., 1997). Insulin has multiple functions in the brain, including food intake and modulation of energy homeostasis (Biessels et al., 2006). In addition, insulin receptors are abundantly distributed throughout the hippocampus, entorhinal cortex, and frontal lobes, whose functions are involved in memory, attention, and execution. Impaired insulin signaling affects several brain neurotransmitter systems (including pathways indirectly related to acetylcholine) and may contribute to cognitive impairment (Bartolomeis et al., 2023). Alterations in insulin signaling pathways, phosphorylation of insulin receptor substrate 1, and changes in insulin-like growth factor-1 observed in the brains of Alzheimer Disease (AD) patients suggest a potential role for insulin in the pathogenesis of cognitive dysfunction (Ehtewish et al., 2022). Cognitive deficits also adversely affect people with chronic diseases in the community, hindering them from performing daily physical activities (Kim et al., 2019).

The marked decline in cognitive function that occurs in patients after a heart attack, or as a result of atrial fibrillation, diabetes mellitus, hypertension, obesity, or obstructive sleep apnea, is due in part to hippocampal atrophy. Individuals of all ages with diabetes mellitus who have high levels of hemoglobin A (hbA) are at high risk of impaired cognitive function and show considerable hippocampal atrophy or where the condition of brain cells in the hippocampus shrinks and decreases resulting in decreased function such as memory loss and disorientation (Korf et al., 2006).

Individuals with hypertension who have never received antihypertensive treatment show a significantly smaller hippocampus compared to those who have undergone such treatment. However, low diastolic blood pressure in patients treated with antihypertensive agents is also associated with hippocampal atrophy (Korf et al., 2004). In addition, higher educational attainment correlates with better initial performance on cognitive function and tends to slow the decline in mental status. The slowdown in overall mental state decline results in active cognitive reserve, which suggests that individuals with higher educational attainment can process tasks more efficiently. These individuals use their brain networks more effectively, the same amount of cognitive impairment results in a smaller decline in their cognitive function compared to those with lower levels of education.

CONCLUSION

The results of the study indicate a relationship between chronic diseases (hypertension, stroke, and diabetes mellitus) with cognitive function. This study found a significant relationship between chronic diseases and cognitive function. There is a relationship between age, gender, education, duration of suffering, smoking habits, and Body Mass Index (BMI) with cognitive function. Hierarchical Logistic Regression, the third model that has a significant influence on respondent characteristic variables such as age, gender, duration of suffering, and education obtained a risk of impacting cognitive function disorders of 28.9%, a significant increase was obtained in the second model that added the direct influence of education.

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