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Original Article

Association Between Smoking and Central Obesity in Individuals Aged ≥40 Years Based on the Indonesian Family Life Survey-5

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Abstract

Background: Obesity and smoking are major public health concerns and leading causes of preventable morbidity and mortality. Smoking may contribute to central obesity through multiple mechanisms, and individuals aged ≥40 years are at higher risk. This study aimed to examine the association between smoking and central obesity, considering sociodemographic factors, using data from the Indonesian Family Life Survey-5 (IFLS-5).

Methods: A cross-sectional study was conducted using secondary data from IFLS-5. Participants included Indonesians aged ≥40 years with complete data on smoking and waist circumference. Smoking status was assessed using the Brinkman Index (BI) and classified as light (0–199), moderate (200–599), or heavy (>600). Central obesity was defined as waist circumference ≥90 cm for male and ≥80 cm for female. Associations were analyzed using Chi-square tests at 95% confidence interval.

Results: Of 3,391 participants, the majority were male (95.4%) with a mean age of 52.76 ± 9.78 years (range 40–101). The prevalence of central obesity was 22.3% (n=790), and the largest group of participants were moderate smokers (54.9%). Central obesity was significantly associated with smoking status (p=0.003), age (p<0.001), gender (p<0.001), and education level (p<0.001). No significant associations were found with marital status (p=0.826), occupation (p=0.374), or residence (p=0.750).

Conclusion: Smoking, age, gender, and education level are significantly associated with central obesity among Indonesians aged ≥40 years. Preventive measures, including smoking cessation and lifestyle interventions, are essential to reduce central obesity and the risk of cardiovascular disease..

Keywords: Active smoker, Brinkman Index, central obesity, smoking status, IFLS-5

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Introduction

Obesity and smoking are two of the most common health issues and remain leading causes of preventable morbidity and mortality worldwide.[1] In Indonesia, more than 30% of adults are classified as obese, making the country one of the highest in Southeast Asia.[2] Central obesity, characterized by fat accumulation around the waist and abdominal cavity, is a stronger predictor of cardiovascular disease risk than body mass index (BMI).[3,4] This condition is influenced by genetics, environment, and lifestyle, and contributes to an estimated 73% of total global deaths.[4]

Multiple factors influence central obesity, including physical activity, excessive stress, unhealthy diets, age, gender, marital status, and smoking.[5] Interestingly, while active smokers generally have a lower BMI and are less likely to be classified as obese, they may still develop central obesity due to fat accumulation in the abdominal area.[6,7] This matter highlights the complex relationship between smoking and fat distribution.

Although the Indonesian Family Life

Survey (IFLS) provides a large and nationally representative dataset, its application in public health research on central obesity and smoking remains underutilized. Given the increasing burden of obesity-related diseases in Indonesia, further investigation is needed to clarify this association. Therefore, this study aimed to determine the relationship between smoking and central obesity among Indonesians aged ≥40 years, by considering sociodemographic factors, using data from the Indonesian Family Life Survey-5 (IFLS-5).

Methods

This study applied a cross-sectional design using secondary data from the Indonesian Family Life Survey-5 (IFLS-5), conducted between October 2014 and April 2015. This study adhered to ethical standards, and received approval from the Research Ethics Committee of the Faculty of Medicine and Health Sciences, Atma Jaya Catholic University of Indonesia (Approval No: 14/09/KEP-FKIKUAJ/2024).

IFLS was a large-scale, population-based longitudinal survey coordinated by a consortium including the RAND Corporation, University of California Los Angeles (UCLA), the Demographic Institute, and the University of Indonesia. The survey covered around 30,000 individuals from 13 provinces, representing approximately 83% of the Indonesian population, collects extensive demographic, socioeconomic, and health-related information.[8]

The study population included all respondents aged ≥40 years who participated

Table 1 Characteristics of Respondents Based on Indonesian Family Life Survey-5 (IFLS-5) (n= 3,391)

Variables	Category	Frequency (n)	%
Gender	Male	3.236	95.4
	Female	155	4.6
Smoking status	Light smoker	986	29.1
	Moderate smoker	1.861	54.9
	Heavy smoker	544	16.0
	Active smoker		
	Male	3.213	94.8
	Female	178	5.2
Central obesity	Yes	790	22.3
	No	2.601	76.7
	Male smoker		
	Central obese	689	20.3
	Not central obese	2.702	79.7
	Female smoker		
	Central obese	104	3.1
	Not central obese	3.287	96.9
Marital status	Not married	79	2.3
	Married	3.077	90.7
	Divorced	81	2.4
	Widowed	154	4.5
Education	Elementary school	1.719	50.7
	Junior high school	524	15.5
	Senior high school	854	25.2
	University	294	8.7
Occupation	Self employed	1.824	53.8
	Government employee	213	6.3
	Private sector employee	781	23.0
	Family worker	101	3.0
	Agricultural	147	4.3
	Others	325	9.6
Residence	Urban	1.786	52.7
	Rural	1.605	47.3

in IFLS-5. Participants were included if they had complete data on smoking status, waist circumference, weight, and BMI. Exclusion criteria were applied to women who were pregnant or using contraceptive methods that could affect body weight or fat distribution.

Smoking status was assessed using the Brinkman Index (BI), calculated as the number of cigarettes smoked per day multiplied by the duration of smoking in years. Based on BI scores, respondents were classified as light smokers (BI 0–199), moderate smokers (BI 200–599), and heavy smokers (BI ≥600).

Central obesity was defined Asian-specific cut-off values for waist circumference: ≥90 cm for male and ≥80 cm for female.[9] The primary independent variable was smoking status, while the dependent variable was central obesity. Covariates included age group, gender, education level, marital status, occupation, and type of residence (urban or rural). These variables were selected based on previous evidence of their association with obesity risk.[1,10]

Descriptive statistics were used to summarize participant characteristics. Continuous variables expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Associations between smoking status, sociodemographic factors, and central obesity were assessed using Chi-square tests with a 95% confidence interval. A p-value <0.05 was considered statistically significant. Analyses were conducted using STATA version 15 (StataCorp, College Station, TX, USA).

Table 2 Association Between Central Obesity and Sociodemographic Variables

Variables	Central Obesity				Total		p-value
	Yes		No				
	n	%	n	%	n	%	
Smoking status							0.003
Light smoker	250	31.6	736	29.1	986	29.0	
Moderate smoker	393	49.8	1,468	54.9	1,861	54.9	
Heavy smoker	147	18.6	379	16.0	544	16.1	
Age group							0.000
40–59 years	666	84.3	2,025	77.9	2,691	79.4	
>60 years	124	15.7	576	22.1	700	20.6	
Gender							0.000
Male	694	87.8	2,542	97.7	3,236	95.4	
Female	96	12.2	59	2.3	155	4.6	
Education							0.000
Elementary school	297	37.6	1,422	54.7	1,719	50.7	
Junior high school	121	15.3	403	15.5	524	15.5	
Senior high school	250	31.6	604	23.2	854	25.2	
University	122	15.5	172	6.6	2.944	8.6	
Marital status							0.826
Not married	19	2.4	60	2.3	79	2.3	
Married	714	90.4	2,363	90.8	3,077	90.7	
Divorced	17	2.1	64	2.5	81	2.4	
Widowed	40	5.1	114	4.4	154	4.6	
Occupation							0.374
Self employed	412	52.2	1,412	54.3	1,824	53.8	
Government employee	58	7.3	155	6.0	213	6.3	
Private sector employee	193	24.4	588	22.6	781	23.0	
Family worker	26	3.2	76	2.9	101	3.0	
Agricultural	27	3.4	120	4.6	147	4.3	
Others	75	9.5	250	9.6	325	9.6	
Residence							0.750
Urban	420	53.2	1,366	52.5	1,786	52.7	
Rural	370	46.8	1,235	47.5	1,605	47.3	
Total	790	100	2.601	100	3.391	100	

Results

Out of 12,276 respondents aged ≥40 years, a total of 3,391 individuals met the inclusion criteria and were included in the final analysis. The majority were male (95.4%) and the mean age was 52.76±9.78 years (range 40–101). The prevalence of central obesity was 22.3% (n=790), with the largest proportion of participants were moderate smokers (54.9%) (Table 1).

Bivariate analysis showed that smoking status, age group, gender, and education level were significantly associated with central obesity ($p<0.05$). Among smokers, 31.6% of light smokers and 49.8% of moderate smokers were centrally obese, compared with 18.6% of heavy smokers. This indicates that moderate smokers had the highest proportion of central obesity. Respondents aged 40–59 years were more likely to have central obesity compared with those aged ≥60 years (84.3% vs. 15.7%).

Central obesity was also significantly associated with gender and education. Although male comprised the majority of respondents, central obesity was proportionally higher among females (12.2% vs 87.8%, $p<0.001$). Education showed a strong association, with elementary school graduates having the highest proportion of central obesity compared with higher education levels. No significant associations were observed between central obesity and marital status ($p=0.826$), occupation ($p=0.374$), or residence ($p=0.750$) (Table 2).

Discussion

This study demonstrated a significant association between smoking and central obesity in individuals aged ≥40 years in Indonesia, with moderate smokers showing the highest prevalence of central obesity. The relationship between smoking and fat distribution is complex. Active smokers, particularly heavy smokers, tend to have a higher likelihood of central obesity compared to those who have never smoked, even though their body mass index (BMI) is generally lower.[5,10] This may be attributed to the effects of nicotine. Initially, nicotine suppresses appetite, however, over time, the brain develops tolerance, requiring higher amounts to achieve the same effect.[11] In addition, hormonal imbalances can increase cortisol levels, which enhance lipolysis and lead to elevated free fatty acid levels, subsequently contributing central obesity.[10] Nicotine in cigarettes stimulates the sympathetic nervous system, which promotes lipolysis and contributes to weight loss. However, prolonged nicotine exposure results in insulin resistance, impairs glycogen synthesis, and reduce the activity of lipoprotein lipase (LPL) in adipocytes. LPL plays a key role in breaking down fat, and when its function is inhibited, free fatty acids accumulate, leading to the release of inflammatory cytokines such as IL-6, TNFα, and leptin.[12] These cytokines can exacerbate inflammation and contribute to the development of metabolic syndrome, thereby increasing the risk of type 2 diabetes, cardiovascular diseases, and non-alcoholic fatty liver disease (NAFLD).[13]

The results on marital status contradict several previous studies that suggested a significant influence on central obesity, with some reporting that married individuals have a 2.5-fold higher risk.[14] However, central obesity is influenced by a complex interplay of factors such as dietary habits, psychological health, genetics, and physical activity, each varying from person to person, regardless of marital status. Therefore, its impact is not absolute.[15] Lifestyle choices, cultural influences, education level, and stress also contribute significantly. Ultimately, personal motivation and discipline in maintaining a healthy diet and regular physical activity often have a stronger effect than marital status alone. Additionally, cultural norms and values may further influence how individuals approach health and nutrition, making marital status less relevant in certain contexts.[16]

As individuals age, they often become more health-conscious, adopting healthier lifestyles due to life experiences and an increased focus on well-being. Chronic conditions such as hypertension, diabetes, and arthritis further heighten awareness, requiring more proactive health management. In contrast, younger individuals tend to engage in riskier behaviours, including sedentary lifestyles and poor dietary habits such as consuming fast food and sugary drinks. These patterns contribute significantly to weight gain and abdominal fat accumulation. This contrast highlights how life stages and health challenges influence behaviours that shapelong-term health outcomes.[17] Evidence shows a rising prevalence of central obesity among younger adults, increasing from 16.3% to 33.9% in individuals aged 15–40 between 1985 and 2014. More recently, the 2023 Indonesian Health Survey (*Survei Kesehatan Indonesia,* SKI) reported an increase in central obesity prevalence from 39.2 to 44.7% in the 35–44 age group, and from 29.6 to 36.2% in

the 25–34 age group over just five years.[18,19] These findings highlight the urgent need for targeted health interventions for younger populations. Westernization, characterized by the adoption of Western dietary and lifestyle patterns, has also contributed to the rise in central obesity. Increased consumption of high glycaemic index carbohydrates, sugar, and unhealthy fats promotes insulin resistance and low-grade inflammation.[20]

Biologically, women generally have a higher percentage of body fat than men. The 45–54 age group coincides with menopause, during which reduced estrogen levels contribute to a shift toward abdominal fat distribution.[21] Smoking has an anti-estrogenic effect, further disturbing hormonal balance and leading to altered fat metabolism and increased abdominal fat in women.[22] In men, smoking increases cortisol levels, which affects fat distribution and appetite regulation. Excess cortisol promotes insulin resistance and fat accumulation in the abdominal region.[23] In postmenopausal women, decreased estrogen levels amplify this effect, resulting in greater susceptibility to central obesity.

Education also plays a protective role. Higher education levels are associated with lower rates of central obesity. Individuals with higher education levels often have better access to information about nutrition and physical activity, which fosters healthier behaviours.[24] Conversely, individuals with lower education levels may have limited access to such information, increasing the likelihood of smoking and other unhealthy behaviours. This aligns with findings from South Africa, where smoking prevalence was higher among individuals with lower educational attainment.[25]

Occupation was not significantly associated with central obesity in this study. This finding contrasts with research from Southeastern Ethiopia, which found that self-employed individuals were more likely to experience central obesity.[25] Sedentary work habits, irregular eating patterns, stress, and lack of physical activity among self-employed individuals could explain this association.[26] Prolonged sitting, in particular, has been linked to worse central obesity outcomes.[27] However, occupation-related activity levels can vary widely, while some self-employed individuals may have physically demanding jobs, others may spend much of their time in sedentary desk work, which elevates risk.

Urbanization is another important factor. A study from Eastern Ethiopia found that urban adults had a higher prevalence of central obesity than rural adults.[28] In China, rapid economic growth and urbanization have significantly altered dietary patterns and reduced physical activity, particularly in cities.[29] Urban residents, with higher incomes, are more likely to consume processed foods and lead sedentary lifestyles, further increasing obesity prevalence. Better access to healthcare in urban areas also contributes to higher reporting and diagnosis rates. These findings emphasize the multifaceted social, economic, and healthcare dynamics driving central obesity in urban populations.

This study has limitations. The data were drawn from pre-existing sources, limiting the ability to add or modify variables. Controlling for confounding factors was also difficult due to unavailable information on potential influences.

In conclusion, smoking status, age, gender, and education level are significantly associated with central obesity, however, marital status, occupation, and residence show no significant association. Preventive and curative measures should be implemented at the primary healthcare level, including smoking cessation programs and lifestyle interventions, to mitigate the risks of central obesity and related cardiovascular diseases.

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