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Short Communication

Impact of anthropometric adiposity and excessive daytime sleepiness on endothelial function in healthcare workers: A cross-sectional analysis

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Abstract

Obesity and excessive daytime sleepiness (EDS) are known contributors to cardiovascular risk through their impact on endothelial function. Healthcare workers, frequently exposed to shift work, are particularly vulnerable to these risk factors. The aim of this study was to assess the relationship between anthropometric adiposity measures and EDS with endothelial function, measured via flow-mediated dilation (FMD), in healthcare workers. This cross-sectional study included 82 healthcare workers aged 20–50 years without pre-existing cardiovascular conditions. Anthropometric measures such as body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHtR) were collected to assess adiposity. EDS was evaluated using the Epworth sleepiness scale (ESS), with a score ≥10 indicating EDS. Endothelial function was measured via FMD, with values <7.1% indicating dysfunction. Multivariate logistic regression was used to identify predictors of endothelial dysfunction, adjusting for confounders such as age and sex. Collinearity diagnostics, including the Belsley–Kuh–Welsch method, were applied to confirm multicollinearity and refine the regression model. Overweight and obesity, high-risk WC, and increased risk WHtR were associated with endothelial dysfunction (p<0.001), with WHtR showing an independent association (adjusted odds ratio (AOR): 8.48; 95%CI: 2.58–27.86; p<0.001). EDS also showed a significant independent association with impaired FMD outcomes (AOR: 3.73; 95%CI: 1.23–11.26; p=0.020). Pearson correlation analysis revealed significant negative correlations between BMI (r=-0.483, p<0.001), WC (r=-0.473, p<0.001), and WHtR (r=-0.432, p<0.001) with FMD, indicating that higher adiposity levels were linked to poorer endothelial function. Obesity and poor sleep quality, even in the absence of cardiovascular disease, are associated with an increased risk of endothelial dysfunction in healthcare workers. Early intervention focusing on weight management and improving sleep quality could mitigate future cardiovascular risks in this population.

Keywords: Body mass index, endothelial dysfunction, flow-mediated dilation, waist circumference, waist-to-height ratio

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Introduction

Cardiovascular diseases (CVD) remain one of the leading causes of mortality globally, with recent estimates attributing 1.95 million deaths annually to obesity-related conditions in 2021 [1]. Obesity has a well-established connection with the development of endothelial dysfunction, a key precursor to atherosclerosis and other cardiovascular diseases [2]. The global prevalence of

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obesity has increased dramatically, with an estimated 603.7 million adults classified as obese [2]. Anthropometric adiposity, including measures such as body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHtR), provides an accessible and practical way to assess body fat distribution and its associated cardiovascular risk [3]. The relationship between these adiposity markers and endothelial function, assessed by non-invasive techniques like flow-mediated dilation (FMD), is crucial in predicting cardiovascular outcomes [4-6]. Endothelial function can also be evaluated using other non-invasive methods, including pulse wave analysis and finger plethysmography during post-ischemic hyperemia. Among these, FMD is considered the most widely used non-invasive technique due to its ability to provide reliable assessments of endothelial function [7,8].

Similarly, lifestyle factors, such as sleep patterns, play a significant role in cardiovascular health [9]. Excessive daytime sleepiness (EDS) is an easily identifiable marker of poor sleep quality and has been associated with adverse cardiovascular outcomes [10]. Healthcare workers, particularly those engaged in shift work, are prone to sleep disturbances, leading to a higher incidence of EDS. This population is of particular interest, as sleep deprivation and irregular sleep patterns have been shown to negatively affect endothelial function, thereby contributing to early development of CVD [11]. Several tools have been developed to measure EDS, including the multiple sleep latency test (MSLT) [12], maintenance of wakefulness test (MWT) [13], and the Stanford Sleepiness Scale (SSS) [14]. However, these methods can be time-consuming, require specialized equipment, or may not be practical for large-scale studies. Given these limitations, the authors decided to use the Epworth sleepiness scale (ESS) due to its simplicity, ease of administration, and strong correlation with objective sleep measures [12-14].

The ESS, introduced by authors, is a widely used self-administered questionnaire designed to assess an individual's general level of EDS [15]. The ESS presents eight common daily situations, such as sitting and reading or watching television, and asks respondents to rate their likelihood of dozing off in each scenario on a scale from 0 (would never doze) to 3 (high chance of dozing) [15]. The cumulative score, ranging from 0 to 24, provides an estimate of a person's average sleep propensity, with higher scores indicating greater daytime sleepiness [16]. Due to its simplicity and strong correlation with objective sleepiness measures, the ESS has become a valuable tool in both clinical and research settings for identifying individuals who may require further evaluation for sleep disorders [16].

Obesity has been linked to EDS through several physiological mechanisms, notably systemic inflammation, metabolic dysregulation, and autonomic dysfunction [17-19]. Adipose tissue in obese individuals secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), leading to a chronic low-grade inflammatory state [17]. This inflammation can disrupt sleep architecture, resulting in increased sleep fragmentation and subsequent EDS [20]. Metabolic dysregulation, characterized by insulin resistance and impaired glucose metabolism, is prevalent in obesity and has been associated with sleep disorders, further contributing to daytime sleepiness [18]. Additionally, obesity is often accompanied by autonomic dysfunction, including heightened sympathetic activity and reduced parasympathetic tone, which can impair sleep quality and promote EDS [19]. Collectively, these findings highlight the complex association between adiposity, sleep disturbances, and cardiovascular dysfunction.

Despite the growing body of evidence linking obesity and sleep patterns to cardiovascular outcomes, there remains a gap in understanding the effects of anthropometric adiposity and excessive daytime sleepiness on endothelial function [4,21]. Therefore, the aim of this study was to investigate the relationship between these risk factors-adiposity and sleepiness-and endothelial dysfunction, as measured by FMD, in healthcare workers. Understanding these relationships may provide further insight into modifiable risk factors for cardiovascular disease, particularly in populations at high risk due to occupational stress and lifestyle.

Methods

Study design

This cross-sectional study was conducted to examine the relationship between anthropometric adiposity, EDS, and endothelial function, measured using FMD, in healthcare workers at Siloam

Hospital Lippo Village from May 4, 2024, to July 23, 2024. This manuscript followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline for cross-sectional studies.

Population and sample

The study's inclusion and exclusion criteria were carefully designed to ensure a specific and relevant participant pool. Healthcare workers aged 20 to 50 years were eligible for inclusion if they had no history of cardiovascular disease, dyslipidemia, peripheral artery disease, diabetes mellitus, or chronic inflammatory diseases. The information on participants' health status was obtained through a combination of self-reported medical history and objective laboratory assessments. All self-reported medical histories were subsequently verified against their medical records, which were obtained from routine medical check-ups conducted by their employers. Additionally, participants needed to be available for FMD measurement and capable of completing the ESS. Exclusion criteria included participants with current or prior smoking history, pregnant individuals, those currently using medications that could affect endothelial function, such as vasodilators, and individuals who declined to provide informed consent.

To determine the minimum sample size for an analytical comparative categorical study with unpaired data, a specific statistical formula is used [22]. The calculation relied on parameters such as the significance level (α), power (1-β), and the expected proportions in each group. The significance level (α) was set at 5%, corresponding to a Zα value of 1.96. The statistical power was set at 80%, meaning a Type II error (β) of 20%, with a corresponding Zβ value of 0.84. The minimum required sample size (n) was obtained by first summing the product of the critical value for Type I error (Zα) and the square root of twice the product of the pooled proportion (P) and its complement (Q), with the product of the critical value for Type II error (Zβ) and the square root of the sum of the products of the proportions in each group (P1 and Q1, as well as P2 and Q2). This entire sum is then squared, and the result is divided by the square of the difference between the proportions in the two groups (P1-P2) [22]. Based on a previous study [6], the expected proportion of patients with a WHtR ≥0.5 and FMD <4.8% (P1) is determined by dividing the number of patients in this category (515) by the total population (637) [6], yielding 0.81. Consequently, the complement (Q1) is calculated as 1-P1=0.19. Similarly, the expected proportion of patients with a waist circumference (WC) ≥90 cm and FMD <4.8% (P2) is obtained by dividing the number of patients in this group (69) by the total population (209) [6], resulting in 0.34. The complement (Q2) is then calculated as 1-P2=0.66. Thus, a minimum sample size of at least 50 participants was determined to provide sufficient statistical power to detect a significant relationship between the variables, assuming a 5% significance level and 80% power.

Data collection

Anthropometric measurements were utilized to assess adiposity, focusing on three key parameters. First, BMI was calculated as the participant's weight in kilograms divided by their height in meters squared (kg/m^2), and participants were categorized as normal, overweight, or obese based on World Health Organization (WHO) standards. Individuals with a BMI of less than 18.5 were classified as underweight, those with a BMI between 18.5 and 22.9 were considered to have normal weight, individuals with a BMI ranging from 23.0 to 24.9 were classified as overweight, and a BMI of 25.0 or higher was categorized as obese [23]. The tools used for weight measurement included the AZKO® Kris Digital Body Scale 10P, while height was measured using the OneMed® Stature Meter. Second, WC was measured using a flexible measuring tape at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest, with classifications into high-risk or normal categories. For males, a WC of less than 90 cm was categorized as low risk, while measurements of 90 cm or more were classified as high risk. For females, a WC of less than 80 cm was considered low risk, whereas 80 cm or more indicated high risk [24]. Lastly, the WHtR was determined by dividing WC by height, where a ratio of ≥0.5 was considered indicative of increased cardiovascular risk [25].

Excessive daytime sleepiness was measured using the ESS, a validated self-administered questionnaire that evaluates the likelihood of falling asleep during eight different daytime situations. A total ESS score of ≥10 was considered indicative of EDS. Participants completed the questionnaire in a quiet setting under the supervision of research staff [15]. The original ESS

questionnaire was in English and translated into Indonesian in a previous publication [26], in which its validity and reliability were tested on 30 undergraduate students from the 2015 regular cohort of Indonesian University. This questionnaire has also been utilized in another publication [27], confirming that all items in the Indonesian version of the ESS are valid ($r \geq 0.4$) and that the reliability tests affirm the ESS's consistency. Sleep duration data were also collected. Sleep duration was categorized as <7 hours and ≥7 hours. The cutoff of 7 hours was chosen based on the American Heart Association (AHA) recommendation, which emphasizes that a minimum of seven hours of sleep per night is essential for optimal health and reducing the risk of cardiovascular disease [21].

Endothelial function was assessed non-invasively using FMD (Philips® iE33 Color Doppler Ultrasound), a widely used measure of vascular endothelial health. The FMD procedure was performed on the brachial artery in the non-dominant arm, following established protocols [28], Briefly, a blood pressure cuff was placed on the participant's arm and inflated to 50 mmHg above systolic pressure for 5 minutes to occlude blood flow. Upon release of the cuff, the brachial artery diameter was measured using high-resolution ultrasound to assess the degree of vasodilation in response to the increased blood flow (reactive hyperemia). An FMD value of <7.1% was considered indicative of endothelial dysfunction [29]. The FMD measurement was performed by two independent cardiologists, each conducting separate assessments to ensure reliability and minimize inter-observer variability. Both cardiologists were blinded to each other's measurements to prevent bias. In cases where discrepancies arose between the two cardiologists' measurements, a consensus discussion was conducted. This involved a thorough review of the recorded ultrasound images and Doppler data, followed by a joint re-evaluation of the artery diameter measurements. If a disagreement persisted, a final consensus decision was reached through mutual agreement.

Data analysis

All collected data were entered into SPSS version 24.0 (IBM, New York, USA) for statistical analysis. Descriptive data for continuous variables were presented as mean and standard deviation (SD) for normally distributed data, and median and minimum-maximum (min-max) for non-normally distributed data. The independent t-test was used to compare normally distributed variables between participants with normal and dysfunctional FMD. For non-normally distributed variables, the Wilcoxon signed-rank test was applied. The relationships between the independent variables (BMI, WC, WHtR, sleep duration, and ESS scores) and the dependent variable (FMD values) were evaluated using chi-square tests for categorical variables and Pearson's correlation for continuous variables. A p-value of <0.05 was considered statistically significant.

Spearman's correlation analysis was to assess the relationship between each category of the ESS and FMD. The ESS categories included sitting and reading, watching TV, sitting inactive in a public place, being a passenger in a car for an hour without a break, lying down to rest in the afternoon when possible, sitting and talking to someone, sitting quietly after lunch (without alcohol), and being in a car while stopped in traffic. Each category was scored on a four-point scale, where 0=no chance of falling asleep, 1=slight chance, 2=moderate chance, and 3=high chance of falling asleep.

For multivariate analysis, logistic regression was conducted to adjust for potential confounders such as age, sex, waist-to-height ratio, sleep duration, ESS, and family history of cardiovascular disease. Before proceeding with the regression analysis, collinearity diagnostics were performed to detect potential multicollinearity among independent variables. Variance inflation factors (VIF) and tolerance values were calculated, with a VIF greater than 10 and a tolerance value less than 0.1 indicating the presence of multicollinearity [30]. In addition to the standard VIF and tolerance values, the Belsley-Kuh-Welsch collinearity diagnostic was also employed. This diagnostic provides a more detailed assessment by analyzing the condition index and variance proportions for each variable. A condition index above 30, combined with variance proportions for two or more variables exceeding 0.5, indicated a multicollinearity problem. Variables exhibiting high multicollinearity were excluded from the model to ensure the stability of the regression estimates [31]. The final model included independent variables free of

collinearity issues, providing reliable adjusted odds ratios (OR) with 95% confidence intervals (CI) for the predictors of endothelial dysfunction.

Results

A total of 101 respondents were recruited, and 82 of them met the inclusion criteria. Nineteen respondents were excluded from the study due to a history of cardiovascular disease, hypertension, diabetes mellitus, stroke, dyslipidemia, and smoking history (**Figure 1**). In this study, a total of 82 healthcare workers participated (**Table 1**), of which the majority were female (81.7%). The participants' ages ranged from 23 to 48 years, with a mean age of 27.48 years (SD: 4.72). The mean BMI was 24.47 kg/m² (SD: 4.60), with values spanning from 17.38 to 35.43 kg/m². WC measurements varied between 59 cm and 103 cm, with a mean of 79.70 cm (SD: 11.08). WHtR had a mean value of 0.501 (SD: 0.065), ranging from 0.38 to 0.63.

Participants' BMI distribution indicated that 43.9% were categorized as underweight or normal, while 56.1% were classified as overweight or obese. The underweight and normal BMI categories were combined due to the limited number of underweight participants (5 individuals). Furthermore, existing literature indicates that endothelial function, as assessed by FMD, does not significantly differ between underweight and normal-weight individuals. For instance, a previous study also grouped together participants with normal and underweight (BMI <25 kg/m²) categories, suggesting comparable endothelial function between normal and underweight groups [32]. WC measurements showed that 51.2% of participants were in the low-risk category, while 48.8% were at high risk, which is consistent with their WHtR, where 48.8% had a normal ratio, and 51.2% had an increased risk (>0.5).

Regarding sleep patterns, the majority (67.1%) reported sleeping less than seven hours per night, while 43.9% exhibited EDS based on the ESS, with scores greater than 10. A notable proportion of participants (17.1%) had a family history of cardiovascular disease. The endothelial function, measured by FMD, was normal (≥7.1%) in 68.3% of the participants, while 31.7% showed endothelial dysfunction (FMD <7.1%). These findings highlight the high prevalence of cardiovascular risk factors, such as obesity, high WC, insufficient sleep, and EDS, among the healthcare workers included in this study.

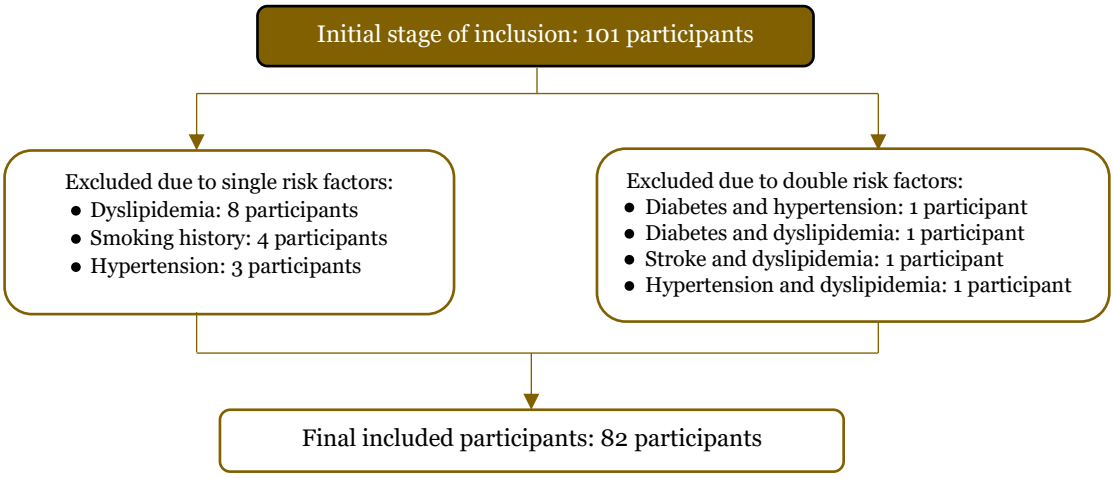

Figure 1. Flow diagram of participants inclusion.

A comparison of participants by FMD status is presented in (**Table 2**), showing significant differences between the normal (n=56) and endothelial dysfunction (n=26) groups. The dysfunction group had a higher median BMI (30.34 (17.38–35.43) vs 22.56 (17.58–32.19), p<0.001), WC (88.42 cm (84.83–92.01) vs. 75.64 cm (73.07–78.22), p<0.001), and WHtR (0.55 (0.53–0.58) vs 0.48 (0.46–0.49), p<0.001). Additionally, the dysfunction group had higher daytime sleepiness scores (ESS 13.00 (1.00–24.00) vs 8.00 (1.00–23.00), p=0.049), though no significant difference in sleep duration was found (5.75 (5.14–6.35) vs 6.24 (5.86–6.62), p=0.155).

Table 1. Characteristics of included participants (n=82)

Characteristic	Sample (n)	Percentage (%)
Sex		
Male	15	18.3
Female	67	81.7
Body mass index		
Underweight + normal	36	43.9
Overweight + obesity	46	56.1
Waist circumference		
Low risk (male <90 cm, female <80cm)	42	51.2
High risk (male ≥90 cm, female ≥80cm)	40	48.8
Waist-to-height ratio		
Normal (≤0.5)	40	48.8
Increased risk (>0.5)	42	51.2
Sleep duration		
≥7 hours	27	32.9
<7 hours	55	67.1
Epworth sleepiness scale		
Normal daytime sleepiness (≤10)	46	56.1
Excessive daytime sleepiness (>10)	36	43.9
Family history of cardiovascular disease		
No	68	82.9
Yes	14	17.1
Flow-mediated dilation		
≥7.1% (normal)	56	68.3
<7.1% (dysfunction)	26	31.7
Total participants	82	100

Table 2. Participants' characteristics based on flow-mediated dilation group (n=82)

Characteristic	Flow-mediated dilation (FMD)		*p*-value
	Normal (n=56)	Dysfunction (n=26)	
Age (median, min-max), years	27.00 (23.00–48.00)	26.00 (23.00–35.00)	0.064[a]
Body mass index (median, min-max), kg/m²	22.56 (17.58–32.19)	30.34 (17.38–35.43)	<0.001[a*]
Waist circumference (mean, SD), cm	75.64 (9.61)	88.42 (8.89)	<0.001[b*]
Waist-to-height ratio (mean, SD)	0.48 (0.06)	0.55 (0.05)	<0.001[b*]
Epworth sleepiness scale (median, min-max), score (0–24)	8.00 (1.00–23.00)	13.00 (1.00–24.00)	0.049[a*]
Sleep duration (mean, SD), hours	6.24 (1.41)	5.75 (1.50)	0.155[b]

Max: maximum; Min: minimum; SD: standard deviation
[*]Statistically significant at *p*-value <0.05
[a]Analyzed using wilcoxon signed-rank test
[b]Analyzed using independent t-test

The correlation coefficients (*r* values) and corresponding *p*-values between clinical characteristics and FMD are presented in (**Table 3**). Age showed a positive correlation with FMD (r=0.297, p=0.007), indicating that increasing age was associated with better endothelial function. In contrast, BMI (r=-0.475, p<0.001), WC (r=-0.473, p<0.001), and WHtR (r=-0.432, p<0.001) were all negatively correlated with FMD, suggesting that higher adiposity measures are linked to poorer endothelial function. Neither ESS (r=-0.161, p=0.150) nor sleep duration (r=0.079, p=0.482) showed significant correlations with FMD.

Table 3. Correlation for each clinical characteristic and flow-mediated dilation scores (n=82)

Characteristic	Correlation *r*-value	*p*-value
Age (years)	0.297	0.007[a*]
Body mass index (kg/m²)	-0.475	<0.001[a*]
Waist circumference (cm)	-0.473	<0.001[b*]
Waist-to-height ratio	-0.432	<0.001[b*]
Epworth sleepiness scale (score 0-24)	-0.161	0.150[a]
Sleep duration (hours)	0.079	0.482[b]

[*]Statistically significant at *p*-value <0.05
[a]Analyzed using Spearman correlation test
[b]Analyzed using Pearson correlation test

Both unadjusted and adjusted analyses for predictors of endothelial dysfunction based on FMD status are presented in (**Table 4**). In the unadjusted analysis, several characteristics were significantly associated with endothelial dysfunction. Participants with overweight or obesity had a higher risk of dysfunction compared to those with underweight or normal BMI (OR: 7.33 (95%CI: 2.23–24.10), $p<0.001$). Similarly, participants with high-risk WC had increased odds of dysfunction (OR: 6.00 (95%CI: 2.07–17.38), $p=0.00$), as did those with an increased WHtR (OR: 7.00 (95%CI: 2.30–21.35), $p<0.001$). EDS was also significantly associated with endothelial dysfunction (OR: 2.88 (95%CI: 1.10–7.53), $p=0.028$). However, age, sex, sleep duration, and family history of cardiovascular disease were not significantly associated with endothelial dysfunction ($p>0.05$). In the adjusted analysis, which controlled for potential confounders (age, sex, WHtR, sleep duration, ESS, and family history of cardiovascular disease), the WHtR remained a strong independent predictor of endothelial dysfunction (adjusted OR (AOR): 8.48 (95%CI: 2.58–27.86), $p<0.001$). EDS also remained a significant independent predictor (AOR: 3.73 (95%CI: 1.23–11.26), $p=0.020$).

Discussion

The findings demonstrate a significant association between measures of adiposity-such as BMI, WC, and WHtR-and impaired endothelial function. Additionally, excessive daytime sleepiness emerged as a predictor of endothelial dysfunction, independent of other factors. These results reinforce the growing body of literature that highlights the detrimental impact of obesity and poor sleep quality on cardiovascular health.

Our findings are consistent with previous studies that have established obesity as a significant contributor to cardiovascular risk [33,34]. Higher BMI, increased WC, and elevated WHtR were strongly associated with impaired endothelial function, as evidenced by lower FMD values. This aligns with the understanding that visceral fat contributes to systemic inflammation, oxidative stress, and insulin resistance, which negatively affect endothelial health [33,34]. Similar results were observed in other studies, where adiposity markers such as BMI and WC were linked to lower FMD values, supporting the notion that central obesity is a critical factor in predicting cardiovascular events [4,5,35]. The significant relationship between WHtR and FMD found in this study further underscores the importance of this adiposity marker in cardiovascular risk assessment, as it provides a more accurate measure of fat distribution compared to BMI alone [6].

Endothelial dysfunction is a pivotal factor in the pathophysiology of CVD. The endothelium, a monolayer of cells lining the blood vessels, regulates vascular tone, maintains blood fluidity, and controls inflammatory responses [36]. When endothelial function is compromised, these regulatory mechanisms are disrupted, leading to various pathological conditions. One key mechanism is the reduced bioavailability of nitric oxide (NO), a vasodilator that inhibits vascular smooth muscle contraction, platelet aggregation, and leukocyte adhesion [37,38]. This reduction, often due to oxidative stress, leads to vasoconstriction, thrombosis, and inflammation, all of which are central to CVD development [39]. Additionally, endothelial dysfunction triggers the expression of adhesion molecules and cytokines, facilitating the recruitment of inflammatory cells into the vessel wall, and thereby accelerating atherosclerosis [40]. Chronic endothelial dysfunction also leads to structural changes in the vasculature, such as increased arterial stiffness and reduced elasticity, impairing the vessels' ability to respond to hemodynamic stresses. Thus, these alterations highlight the central role of endothelial dysfunction in initiating and progressing cardiovascular diseases [36].

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It is noteworthy that the study population was comprised of healthcare workers who had no history of cardiovascular disease, dyslipidemia, peripheral artery disease, diabetes mellitus, or chronic inflammatory conditions. Furthermore, none of the participants were current or former smokers, pregnant, or using medications that could influence endothelial function. This enhances the validity of our findings by illustrating that, even in the absence of pre-existing cardiovascular conditions, factors such as obesity and poor sleep quality are still associated with an increased risk of endothelial dysfunction. These findings highlight the possibility of early vascular changes related to adiposity and sleep disturbances, which may occur prior to the development of clinical cardiovascular disease [2,41].

In this study, EDS, measured by the ESS, was significantly associated with endothelial dysfunction. Participants with higher ESS scores had lower FMD values, suggesting a direct correlation between sleep disturbances and vascular health. This is in line with previous research that has shown how sleep deprivation and irregular sleep patterns, common among healthcare workers, contribute to endothelial dysfunction through mechanisms such as heightened sympathetic activity, increased blood pressure, and elevated inflammatory markers [21,42]. Healthcare workers, due to their demanding work schedules and exposure to shift work, are particularly vulnerable to sleep disorders, making this finding especially relevant in this population [43].

Given the high prevalence of obesity and EDS among healthcare workers, as demonstrated by this study, targeted interventions aimed at improving sleep quality and reducing adiposity could be essential in mitigating cardiovascular risks in this population. Implementing workplace wellness programs focusing on healthy weight management and adequate sleep hygiene practices, along with strategies to reduce occupational stress, may help improve both sleep patterns and cardiovascular outcomes in healthcare professionals.

Several limitations must be considered when interpreting the findings of this study. The cross-sectional design precludes any inference of causality between adiposity, EDS, and endothelial dysfunction. Additionally, the reliance on self-reported sleep measures, such as the ESS, may introduce bias. Finally, the small sample size, particularly among older individuals and those with a family history of cardiovascular disease, limits the generalizability of these results. Future research should focus on larger, more diverse samples and employ longitudinal designs to better understand the relationships between adiposity, sleep, and endothelial health over time.

Table 4. Predictors of endothelial dysfunction based on flow-mediated dilation (FMD)

Characteristic	Flow mediated dilation (FMD)				OR (95%CI)	*p*-value	Adjusted OR** (95%CI)	Adjusted *p*-value	Beta coefficients
	Normal (n=56)		Dysfunction (n=26)						
	n	%	n	%					
Age									
<40 years old	53	67.1	26	32.9					
≥40 years old	3	100	0	0	NA	0.548	NA	NA	NA
Sex									
Female	49	73.1	18	26.9					
Male	7	46.7	8	53.3	3.11 (0.99–9.82)	0.066	NA	NA	NA
Body mass index									
Underweight + normal	32	88.9	4	11.1					
Overweight + obesity	24	52.2	22	47.8	7.33 (2.23–24.10)	<0.001*	NA	NA	NA
Waist circumference									
Low risk	36	85.7	6	14.3					
High risk	20	50	20	50	6.00 (2.07–17.38)	0.001*	NA	NA	NA
Waist-to-height ratio									
Normal	35	87.5	5	12,5					
Increased risk	21	50	21	50	7.00 (2.30–21.35)	<0.001*	8.48 (2.58–27.86)	<0.001*	2.14
Sleep duration									
≥7 hours	21	77.8	6	22.2					
<7 hours	35	63.6	20	36.4	2.00 (0.69–5.78)	0.196	NA	NA	NA
Epworth sleepiness scale									
Normal	36	78.3	10	21.7					
Excessive	20	55.6	16	44.4	2.88 (1.10–7.53)	0.028*	3.73 (1.23–11.26)	0.020*	1.32
Family history of cardiovascular disease									
No	48	70.6	20	29.4					
Yes	8	57.1	6	42.9	1.80 (0.55–5.86)	0.355	NA	NA	NA

Unadjusted predictors were analyzed using the Pearson chi-square test, while independent predictors (adjusted) were identified through logistic regression analysis using backward stepwise (likelihood ratio) selection. CI: confidence interval; NA: not available; OR: odds ratio

*Statistically significant at *p* value <0.05

**Factors included within the model building: age, sex, waist-to-height ratio, sleep duration, Epworth sleepiness scale, and family history of cardiovascular disease (Factors were selected without multicollinearity problem based on collinearity statistics)

Conclusion

This study demonstrates that both anthropometric adiposity and excessive daytime sleepiness are significantly associated with endothelial dysfunction among healthcare workers. These findings suggest that interventions aimed at improving sleep and reducing obesity in health workers should play a key role in preventing cardiovascular diseases in this specific high-risk population.

Ethics approval

Ethical approval for the study was obtained from the Ethics Committee of Universitas Pelita Harapan (Number 115/K-LKJ/ETIK/II/2024 and Number 125/K-LKJ/ETIK/II/2024). All participants provided written informed consent before participating in the study. The study adhered to the ethical principles outlined in the Declaration of Helsinki.

Acknowledgments

None

Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the online digital repository (Appendix 1: http://dx.doi.org/10.6084/m9.figshare.28532816 and Appendix 2: http://dx.doi.org/10.6084/m9.figshare.28536680).

Declaration of artificial intelligence use

We hereby confirm that no artificial intelligence (AI) tools or methodologies were utilized at any stage of this study, including during data collection, analysis, visualization, or manuscript preparation. All work presented in this study was conducted manually by the authors without the assistance of AI-based tools or systems.

How to cite

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