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Association of Exhaled Carbon Monoxide (CO) and Serum Malondialdehyde (MDA) With Spirometric Impairment in Active Smokers; A Cross-Sectional Study

Andi Isra Azraeni Mustari[1], Sitti Nurisyah[1,3*], Harry Akza Putrawan[1,2], Nur Ahmad Tabri[1,2], Erwin Arief[1,3], Joko Hendarto[1]

[1]Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
[2]Dr. Wahidin Sudirohusodo Hospital, Makassar, South Sulawesi, Indonesia
[3]Dr. Tadjuddin Chalid Hospital, Makassar, South Sulawesi, Indonesia
Corresponding Author*: Sitti Nurisyah. Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia. Perintis Kemerdekaan Street, Km 10, Tamalanrea, Makassar, South Sulawesi, Indonesia. Email: ichanurisyah@unhas.ac.id.

ABSTRACT: Smoking-related oxidative injury may cause early lung function impairment before respiratory symptoms develop. This study evaluated the associations of exhaled carbon monoxide and serum malondialdehyde with spirometric impairment in asymptomatic active smokers. This cross-sectional study included 70 active-smoking hospital security personnel in Makassar, Indonesia. Smoking history, time since the last cigarette, exhaled carbon monoxide, serum malondialdehyde, and spirometric parameters were assessed. Associations were evaluated using Spearman correlation, group comparisons, receiver operating characteristic analysis, and multivariable Firth logistic regression. Spirometric impairment was identified in 30 participants (42.9%), with reduced $FEF_{25–75}$ as the most frequent abnormality (22.9%). Time since the last cigarette was associated with exhaled carbon monoxide category ($p=0.006$). The Brinkman Index correlated positively with exhaled carbon monoxide ($rho=0.628$, $p<0.001$) and serum malondialdehyde ($rho=0.537$, $p<0.001$). Exhaled carbon monoxide was inversely associated with FEV_1, FEV_1/FVC, and $FEF_{25–75}$, whereas serum malondialdehyde was inversely associated with all spirometric parameters, most strongly with $FEF_{25–75}$ ($rho=−0.616$, $p<0.001$). Both biomarkers were higher in participants with spirometric impairment. Serum malondialdehyde showed better discrimination for composite spirometric impairment than exhaled carbon monoxide (area under the curve: 0.814 vs 0.745) and was independently associated with composite impairment and reduced $FEF_{25–75}$. Exhaled carbon monoxide and serum malondialdehyde provide complementary information in asymptomatic active smokers. Exhaled carbon monoxide reflects recent smoking exposure, whereas serum malondialdehyde more consistently reflects oxidative injury associated with early spirometric impairment, particularly $FEF_{25–75}$.

1. INTRODUCTION

Tobacco smoking remains a major global health problem, with more than one billion tobacco users worldwide and a persistently high burden in several low- and middle-income countries [1]. In Indonesia, smoking is particularly prevalent among adult men,

creating a substantial population at risk of smoking-related respiratory impairment [2]. Cigarette smoke is a major exogenous source of carbon monoxide (CO), and exhaled CO provides a rapid, non-invasive indicator of recent smoking exposure [3].

Beyond exposure assessment, smoking promotes oxidative stress through an imbalance between oxidant generation and antioxidant defence, contributing to airway and parenchymal injury [4]. Malondialdehyde (MDA), a product of lipid peroxidation, is commonly used as a biomarker of oxidative stress and has been reported at higher concentrations among tobacco users than among non-users [5]. In active smokers with chronic obstructive pulmonary disease, higher serum MDA levels have also been associated with poorer spirometric indices, including forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and the FEV1/FVC ratio [6].

Spirometry is widely used to assess pulmonary function and identify airflow limitation. Although forced expiratory flow between 25% and 75% of forced vital capacity ($FEF_{25–75}$) may provide additional information regarding mid-expiratory airflow, its clinical interpretation is limited by variability and dependence on FVC. Therefore, reduced $FEF_{25–75}$ should be interpreted cautiously and cannot independently establish small-airway disease [7]. Although exhaled CO and serum MDA represent complementary pathways—recent smoke exposure and oxidative injury, respectively—their concurrent relationship with spirometric impairment in asymptomatic active smokers remains insufficiently characterised. This cross-sectional study therefore evaluated the associations of exhaled CO and serum MDA with spirometric impairment among active-smoking security personnel.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This analytical cross-sectional study was conducted among active-smoking security personnel working in hospitals in Makassar, Indonesia, from March to May 2026. The study evaluated the associations of exhaled CO and serum MDA with spirometric impairment.

2.2 Participants and Sampling

Participants were recruited using consecutive sampling: all hospital security personnel who met the eligibility criteria during the study period were approached and invited to participate. The sample size was determined based on an anticipated correlation coefficient of 0.35, a two-sided significance level of 0.05, and 80% statistical power. After finite population correction and allowance for incomplete data, the minimum planned sample size was 58 participants. A total of 70 participants were included in the final analysis. Eligible participants were aged ≥18 years, had smoked at least 100 cigarettes during their lifetime, were current smokers, had no respiratory symptoms during the preceding 2 weeks, and provided written informed consent. Participants were excluded if they used electronic cigarettes; had dyspnoea, haemoptysis, or cough lasting >2 weeks; had chronic pulmonary disease, including asthma, chronic obstructive pulmonary disease, active pulmonary tuberculosis, or post-tuberculosis lung damage; or were unable to perform acceptable spirometry manoeuvres.

Participants with conditions that could affect exhaled CO, serum MDA, or lung function were also excluded. These included haemolytic anaemia, pregnancy, menstruation, vigorous exercise within 12 h before examination, diabetes mellitus, hypertension, cardiovascular disease, lung cancer, kidney disease, relevant occupational or environmental smoke exposure, and other major respiratory comorbidities. A total of 70 participants were included in the final analysis.

2.3 Clinical Assessment and Smoking History

Demographic characteristics, body mass index, smoking duration, and daily cigarette consumption were recorded using a structured, interviewer-administered questionnaire. Smoking history was self-reported and included current smoking status, duration of smoking, and average cigarettes smoked per day. The Brinkman Index was calculated as the number of cigarettes smoked per day multiplied by smoking duration in years. Time since the last cigarette was recorded in hours immediately before exhaled CO measurement and categorised as <3 h, 3–8 h, or >8 h.

2.4 Exhaled CO Measurement

Participants rested in a seated position for 5–10 min before measurement. Exhaled CO was measured using a Smokerlyzer® piCO™ Breath Carbon Monoxide Monitor (Bedfont® Scientific Ltd., Maidstone, UK) equipped with an electrochemical sensor. After a maximal inhalation, participants held their breath for approximately 15 s and then exhaled slowly and completely

through a single-use mouthpiece. Measurements were recorded in parts per million together with the examination time and time since the last cigarette.

2.5 Serum MDA Measurement

Participants fasted for approximately 12 h before venous blood collection; water intake was permitted. Approximately 3 mL of venous blood was collected into a plain tube, allowed to clot at room temperature for 20–30 min, and processed to obtain serum. Samples were centrifuged within 1 h and stored at −20 °C to −80 °C until analysis. Serum MDA was quantified by high-performance liquid chromatography using an Agilent 1200 Series system at Prodia Laboratory, Makassar. The analytical method had been developed and validated by the Mass Spectrometry Laboratory, Prodia.

2.6 Spirometry

Spirometry was performed using a calibrated spirometer according to American Thoracic Society/European Respiratory Society standards. The device used was a portable digital CONTEC SP80B spirometer (Contec Medical Systems Co., Ltd., China), equipped with an infrared turbine sensor; its volume measurement range was 0–10 L, with an accuracy of ±3% or ±0.05 L. Participants performed the test in a comfortable upright position, with a nose clip used to minimise nasal air leakage. After maximal inspiration, participants exhaled as forcefully and rapidly as possible until no further air could be expelled. Each participant performed at least three forced expiratory manoeuvres, and the best technically acceptable values were recorded. Spirometric acceptability and repeatability were evaluated according to ATS/ERS technical standards. FEV_1, FVC, FEV_1/FVC, and $FEF_{25–75}$ were recorded. Reduced values were defined a priori as FEV_1 and FVC <80% predicted, FEV_1/FVC <70%, and $FEF_{25–75}$ <65% predicted. Composite spirometric impairment was defined as the presence of at least one of these prespecified abnormalities. Because $FEF_{25–75}$ is effort-dependent, variable, and influenced by FVC, a reduced value was treated as an exploratory marker of altered mid-expiratory flow and was not interpreted as diagnostic of isolated small-airway disease.

2.7 Statistical Analysis

Data were summarised using descriptive statistics. Associations between categorical variables were assessed using the Fisher–Freeman–Halton test, whereas correlations between continuous or ordinal variables were examined using Spearman's rank correlation. Exhaled CO and serum MDA levels were compared between normal and impaired spirometric groups using the Mann–Whitney U test. Receiver operating characteristic curve analysis was used to evaluate the discriminatory performance of exhaled CO and serum MDA for composite spirometric impairment and individual reduced spirometric parameters. Multivariable Firth logistic regression included age, Brinkman Index, exhaled CO, and serum MDA; these covariates were selected a priori as clinically relevant potential confounders representing age, cumulative smoking exposure, and recent smoke exposure. Firth penalisation was used to reduce small-sample bias and mitigate potential separation in this modest-sized dataset with sparse abnormal spirometric categories. Odds ratios were expressed per 1-standard-deviation increase. A two-sided p value <0.05 was considered statistically significant.

3. RESULTS AND DISCUSSION

3.1 Participant Characteristics and Spirometric Status

Seventy asymptomatic active smokers were included. Participant characteristics, smoking exposure, biomarker levels, and spirometric measures are summarised in Table 1. The median Brinkman Index was 225.00, and the mean exhaled CO and serum MDA concentrations were 14.24 ppm and 1.20 nmol/mL, respectively. Study-defined composite spirometric impairment, defined as at least one prespecified abnormal parameter, was identified in 30 participants (42.9%). Reduced $FEF_{25–75}$, using the prespecified threshold of <65% predicted, was the most frequent abnormality (22.9%), followed by reduced FVC (20.0%) and FEV_1 (17.1%); no participant had an FEV_1/FVC ratio <70%.

Table 1. Participant characteristics, smoking exposure, biomarker concentrations, and spirometric measures (n=70).

Variable	Value
Age, years	38.46 ± 10.16
Body mass index, kg/m²	23.98 ± 3.74
Smoking duration, years	17.76 ± 9.00

Cigarettes/day	13.89 ± 8.66
Brinkman Index	225.00 (4.00–1360.00)
Exhaled CO, ppm	14.24 ± 8.71
Serum MDA, nmol/mL	1.20 ± 0.43
FEV_1, % predicted	86.37 ± 8.08
FVC, % predicted	86.51 ± 7.88
FEV_1/FVC, %	86.22 ± 5.56
$FEF_{25–75}$, % predicted	74.56 ± 12.73
Composite spirometric impairment	30 (42.9)
Reduced FEV_1	12 (17.1)
Reduced FVC	14 (20.0)
Reduced $FEF_{25–75}$	16 (22.9)

Data are mean ± standard deviation, median (range), or n (%), as appropriate.

3.2 Smoking Exposure, Exhaled CO, and Serum MDA

Time since the last cigarette was significantly associated with exhaled CO category (p=0.006; Table 2 and Figure 1). Participants examined within 3 h of smoking were predominantly classified as having moderate or heavy exhaled CO levels. The Brinkman Index correlated positively with exhaled CO (rho=0.628, p<0.001) and serum MDA (rho=0.537, p<0.001), and inversely with FEV_1 (rho=−0.421, p<0.001) and $FEF_{25–75}$ (rho=−0.319, p=0.007) (Table 3).

Table 2. Association Between Time Since the Last Cigarette and Exhaled Carbon Monoxide Categories..

Time since last cigarette	Normal <7 ppm	Mild 7–10 ppm	Moderate >10–20 ppm	Heavy >20 ppm	p value
<3 h (n=24)	1 (4.2)	1 (4.2)	16 (66.7)	6 (25.0)	0.006
3–8 h (n=23)	8 (34.8)	2 (8.7)	11 (47.8)	2 (8.7)	
>8 h (n=23)	7 (30.4)	5 (21.7)	5 (21.7)	6 (26.1)	

Values are n (%); Fisher–Freeman–Halton test.

3.3 Associations With Spirometric Parameters

Exhaled CO was inversely correlated with FEV_1, FEV_1/FVC, and $FEF_{25–75}$, but not with FVC. Serum MDA was inversely correlated with all spirometric parameters, with the strongest association observed for $FEF_{25–75}$ (rho=−0.616, p<0.001) (Table 3 and Figure 2). Exhaled CO and serum MDA were not significantly associated with each other (R^2=0.017, p=0.281).

Table 3. Spearman correlations of smoking exposure and biomarkers with spirometric parameters

Association	Spearman rho	p value
Brinkman Index–exhaled CO	0.628	<0.001
Brinkman Index–serum MDA	0.537	<0.001
Brinkman Index–FEV_1	−0.421	<0.001
Brinkman Index–FVC	−0.189	0.118
Brinkman Index–FEV_1/FVC	−0.183	0.128
Brinkman Index–$FEF_{25–75}$	−0.319	0.007
Exhaled CO–FEV_1	−0.420	<0.001

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Exhaled CO–FVC	−0.199	0.099
Exhaled CO–FEV_1/FVC	−0.477	<0.001
Exhaled CO–$FEF_{25–75}$	−0.378	0.001
Serum MDA–FEV_1	−0.426	<0.001
Serum MDA–FVC	−0.302	0.011
Serum MDA–FEV_1/FVC	−0.365	0.002
Serum MDA–$FEF_{25–75}$	−0.616	<0.001

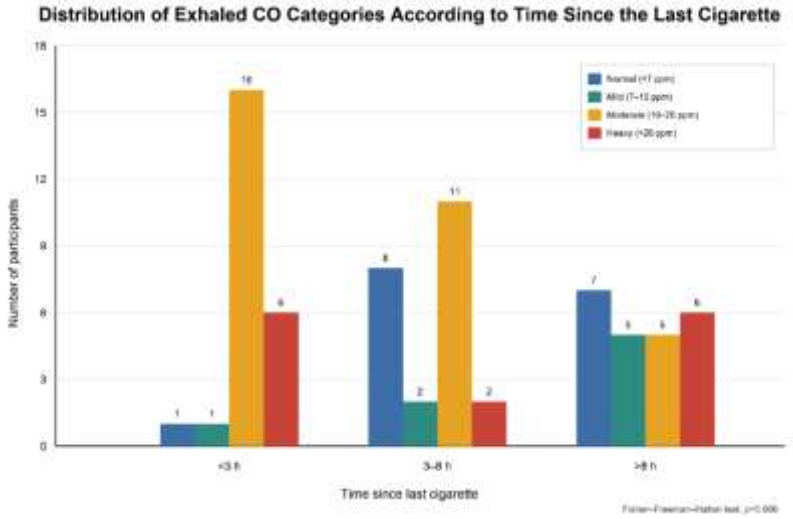

Figure 1. Distribution of exhaled carbon monoxide categories according to time since the last cigarette. Bars represent the number of participants in each exhaled CO category across the three smoking intervals (<3 h, 3–8 h, and >8 h).

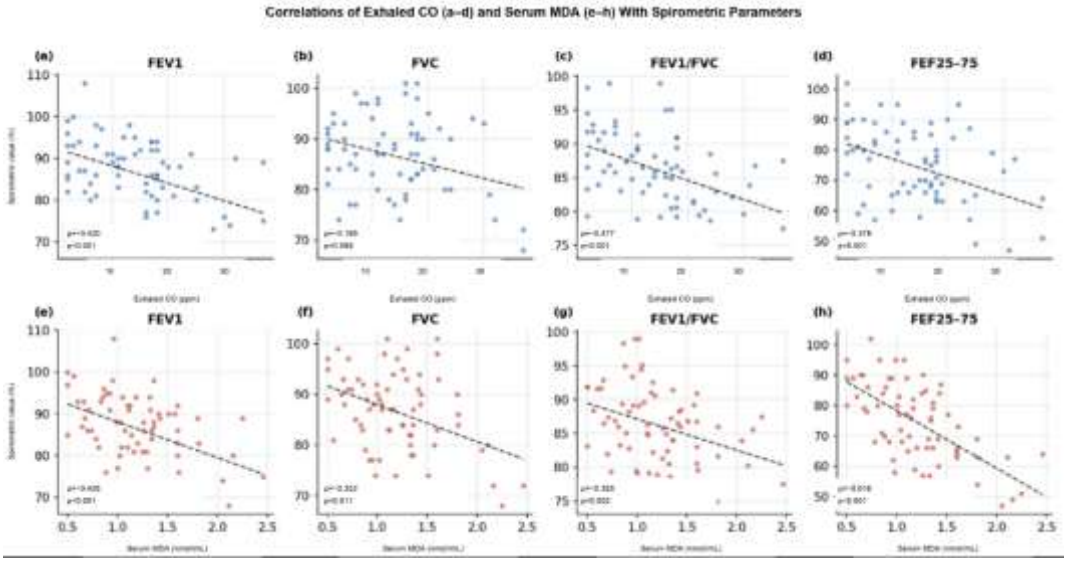

Figure 2. Associations of exhaled carbon monoxide and serum malondialdehyde with spirometric parameters. Panels (a–d) illustrate the relationships between exhaled CO and FEV_1, FVC, FEV_1/FVC, and $FEF_{25–75}$, respectively. Panels (e–h) illustrate the corresponding relationships between serum MDA and the same spirometric parameters. Spearman correlation coefficients and p values are presented.

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3.4 Biomarker Levels and Discriminatory Performance

Both biomarkers were higher among participants with spirometric impairment than among those with normal spirometry (Table 4). Serum MDA demonstrated a numerically higher AUC than exhaled CO for composite spirometric impairment (0.814 vs. 0.745). It also showed the strongest discrimination for reduced $FEF_{25–75}$ (area under the curve, 0.848; optimal cut-off, 1.42 nmol/mL), compared with exhaled CO (area under the curve, 0.690; optimal cut-off, 18.00 ppm) (Table 5 and Figure 3). Conversely, exhaled CO showed greater discrimination than serum MDA for reduced FEV_1.

Table 4. Comparison of Exhaled Carbon Monoxide and Serum Malondialdehyde Concentrations According to Spirometric Status.

Biomarker	Normal spirometry (n=40)	Spirometric impairment (n=30)	p value
Exhaled CO, ppm	11.00 (4.75–17.00)	18.00 (13.25–24.75)	<0.001
Serum MDA, nmol/mL	1.03 (0.73–1.21)	1.42 (1.17–1.61)	<0.001

Values are median (interquartile range); Mann–Whitney U test.

Table 5. Discriminatory performance of exhaled CO and serum MDA for spirometric impairment

Outcome / biomarker	AUC	Optimal cut-off	Sensitivity, %	Specificity, %	p value
Composite impairment: exhaled CO	0.745	13.00 ppm	76.7	60.0	<0.001
Composite impairment: serum MDA	0.814	1.30 nmol/mL	66.7	82.5	<0.001
Reduced FEV_1: exhaled CO	0.816	14.00 ppm	100.0	55.2	<0.001
Reduced FEV_1: serum MDA	0.727	1.43 nmol/mL	58.3	82.8	0.014
Reduced $FEF_{25–75}$: exhaled CO	0.690	18.00 ppm	62.5	74.1	0.022
Reduced $FEF_{25–75}$: serum MDA	0.848	1.42 nmol/mL	68.8	87.0	<0.001

AUC, area under the receiver operating characteristic curve.

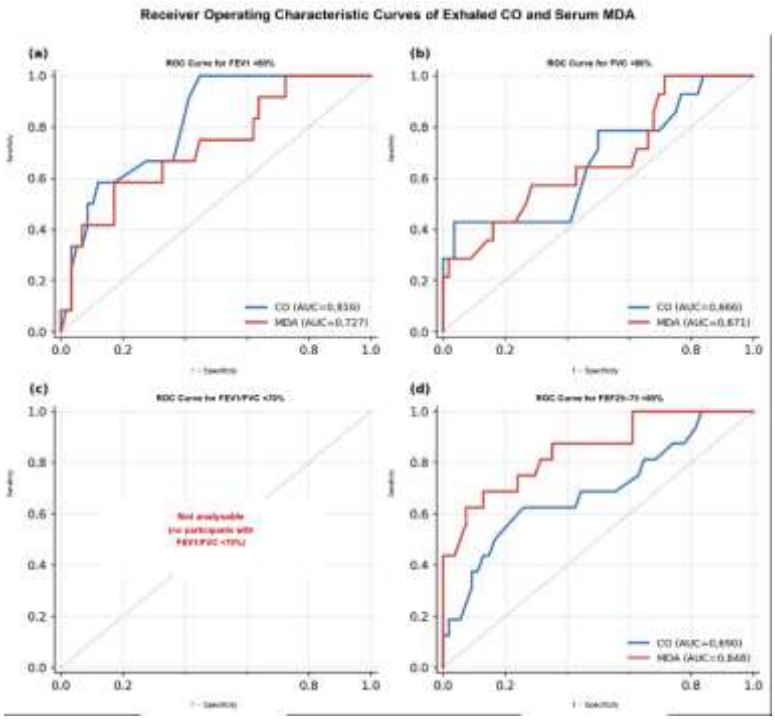

Figure 3. Receiver operating characteristic curves comparing the discriminatory performance of exhaled carbon monoxide and serum malondialdehyde for study-defined spirometric impairment. Panel (a) shows composite spirometric impairment, while panel (b) shows reduced $FEF_{25–75}$. The corresponding area under the curve values are presented for each biomarker.

3.4 Multivariable Analysis

In multivariable Firth logistic regression, serum MDA remained independently associated with composite spirometric impairment after adjustment for age, Brinkman Index, and exhaled CO (odds ratio per 1-standard-deviation increase, 2.94; 95% confidence interval, 1.19–8.17; p=0.019). Serum MDA was also independently associated with reduced $FEF_{25–75}$ (odds ratio per 1-standard-deviation increase, 8.84; 95% confidence interval, 2.60–22.74; p<0.001), whereas age, Brinkman Index, and exhaled CO were not independently associated with either outcome (Table 6).

Table 6. Multivariable Firth logistic regression for spirometric impairment.

Outcome / variable	OR per 1-SD increase	95% CI	p value
Composite spirometric impairment			
Age	0.71	0.30–1.60	0.409
Brinkman Index	1.85	0.55–7.49	0.330
Exhaled CO	1.29	0.56–3.12	0.552
Serum MDA	2.94	1.19–8.17	0.019
Reduced $FEF_{25–75}$			
Age	0.59	0.19–1.73	0.340
Brinkman Index	1.67	0.45–7.64	0.454
Exhaled CO	0.40	0.10–1.25	0.120
Serum MDA	8.84	2.60–22.74	<0.001

OR, odds ratio; CI, confidence interval; SD, standard deviation.

3.4 Discussion

In this cross-sectional study of asymptomatic active smokers, study-defined spirometric impairment was evident despite the absence of respiratory symptoms, with the most frequent prespecified abnormality involving $FEF_{25–75}$. The relatively frequent reduction in $FEF_{25–75}$ may indicate altered mid-expiratory airflow among asymptomatic active smokers. However, because $FEF_{25–75}$ is variable, effort-dependent, and influenced by FVC, this finding should be interpreted cautiously and cannot by itself confirm small-airway disease. Further evaluation using more specific physiological techniques is warranted. These findings are consistent with Chami et al. [8], who reported increased airway resistance measured by impulse oscillometry among exclusive waterpipe smokers despite relatively preserved conventional spirometric parameters. Although the type of tobacco exposure differed from that in the present study, their findings suggest that physiological airway abnormalities may be detectable even when conventional spirometric indices remain within normal limits. The predominance of elevated exhaled CO also accords with findings among workers in Makassar, where smoking behaviour was associated with higher CO exposure [9].

The association between shorter time since the last cigarette and higher exhaled CO confirms that this measurement principally reflects recent and dynamic smoke exposure. This interpretation is consistent with Vinnikov et al. [3] and Herath et al. [10], both of whom reported that exhaled CO is strongly influenced by recent smoking practices. Accordingly, exhaled CO should be interpreted together with time since the last cigarette rather than as an isolated marker of chronic pulmonary injury. Its association with the Brinkman Index nevertheless indicates that acute exposure is partly related to cumulative smoking burden, although the two measures capture different time scales of exposure.

A higher Brinkman Index was associated with higher exhaled CO and serum MDA, as well as poorer airflow-related spirometric indices. This pattern is biologically plausible because cumulative smoke exposure increases oxidant burden, depletes antioxidant defences, and promotes lipid peroxidation. The association between smoking exposure and oxidative stress is consistent with the findings of Chauhan et al. [5], Tualeka et al. [11], and Pandi et al. [12]. The preferential association with FEV_1 and $FEF_{25–}$

$_{75}$, rather than all spirometric indices, suggests an association with airflow-related abnormalities; the $FEF_{25–75}$ finding should not be taken as proof of isolated small-airway dysfunction.

Exhaled CO and serum MDA showed distinct relationships with pulmonary function. Exhaled CO was more closely associated with airflow measures, whereas serum MDA was consistently associated with all spirometric parameters and showed the strongest relationship with $FEF_{25–75}$. These findings are consistent with Ranjana et al. [6], who reported an inverse association between serum MDA and spirometric indices in active smokers with chronic obstructive pulmonary disease. This difference is coherent with their biological roles: exhaled CO is a marker of recent smoking exposure, whereas MDA reflects the downstream oxidative injury produced by persistent lipid peroxidation and inflammation [13].

Both markers were higher among participants with spirometric impairment, supporting a continuum from smoke exposure to oxidative injury and altered lung function. However, serum MDA demonstrated better overall discriminatory ability, particularly for reduced $FEF_{25–75}$. This observation is supported by evidence that lipid peroxidation products reflect respiratory oxidative injury and may capture biological effects that are not represented by acute exposure markers alone [14,15]. Conversely, the relatively stronger performance of exhaled CO for reduced FEV_1 suggests that it may remain useful as a rapid, non-invasive indicator of recent exposure associated with airflow impairment.

After adjustment for age, cumulative smoking burden, and exhaled CO, serum MDA remained independently associated with spirometric impairment, especially reduced $FEF_{25–75}$. These findings suggest that systemic lipid peroxidation, as reflected by serum MDA concentrations, is associated with study-defined reduction in mid-expiratory flow among asymptomatic active smokers. The loss of independent associations for the Brinkman Index and exhaled CO after adjustment is compatible with MDA representing a downstream biological consequence of smoking-related injury, rather than merely the intensity of exposure. Taken together, the findings support serum MDA as a potential adjunct to spirometry for identifying smoking-related oxidative injury and abnormal mid-expiratory flow, while not establishing isolated small-airway disease.

This study has several limitations. Lung function was assessed solely by spirometry; therefore, reduced FVC could not be confirmed as restrictive impairment without measurement of total lung capacity or diffusing capacity. Abnormal mid-expiratory flow was assessed using $FEF_{25–75}$ without confirmation by more specific techniques, such as impulse oscillometry. Smoking history used to calculate the Brinkman Index was self-reported and may have been affected by recall bias. In addition, exposure was not verified using an alternative biomarker, such as urinary cotinine.

4. CONCLUSION

In this cross-sectional study of asymptomatic active smokers, exhaled carbon monoxide and serum malondialdehyde were associated with several spirometric indices. Serum malondialdehyde demonstrated a stronger inverse correlation with $FEF_{25–75}$ and a numerically higher discriminatory performance for study-defined spirometric impairment than exhaled carbon monoxide. After adjustment for age, cumulative smoking exposure, and exhaled carbon monoxide, serum malondialdehyde remained independently associated with reduced $FEF_{25–75}$. These findings suggest that systemic oxidative stress may be associated with alterations in pulmonary function among asymptomatic smokers, although the $FEF_{25–75}$ findings should be interpreted cautiously and do not establish isolated small-airway disease. However, larger prospective studies incorporating more specific assessments of small-airway function are needed to confirm these findings and evaluate the potential clinical utility of these biomarkers.

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AUTHOR CONTRIBUTIONS

A.I.A.M.: Conceptualization, methodology, investigation, data curation, formal analysis, visualization, writing—original draft. **S.N. and H.A.P.:** Conceptualization, methodology, supervision, writing—review and editing. **N.A.T., E.A., and J.H.:** Investigation, data curation, writing—review and editing. All authors approved the final manuscript.

CONFLICT OF INTEREST

The authors declare no competing interests.

ETHICS STATEMENT

The study was approved by the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia (No. 188/UN4.6.4.5.31/PP36/2026). All participants provided written informed consent before enrolment.

DATA AVAILABILITY STATEMENT

The de-identified data supporting the findings of this study are available from the corresponding author on reasonable request.

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