

Comparison of Carboxyhemoglobin Levels, Pulmonary Functions, and Nicotine Dependence Among Smoking Healthcare Professionals in the Operating Theater

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Cite as: Kavak İ, Şen S, Özen C, Akalın MZ, Şirinyıldız F, Kurt Ömürlü İ. Comparison of Carboxyhemoglobin Levels, Pulmonary Functions, and Nicotine Dependence Among Smoking Healthcare Professionals in the Operating Theater. *J Cukurova Anesth Surg.* 2026;9(3):898-908.

doi:10.36516/jocass.1977439

Received: 23.06.2026, **Accepted:** 18.09.2026, **Published:** 30.09.2026, CC BY-NC 4.0

Abstract

Objective: While the individual impacts of tobacco and surgical smoke are known, their combined effect on the respiratory health of healthcare professionals remains undocumented. This study aimed to fill this gap by providing the first integrated evaluation of COHb levels and pulmonary function parameters among smoking personnel exposed to varying degrees of occupational hazards in operating theaters (OT) and internal medicine wards. **Material and Methods:** In this cross-sectional study, smoking healthcare personnel were categorized by work environment: OT (Group O) and internal medicine wards (Group M). To assess the "distance-to-hazard" gradient, Group O was sub-analyzed professional role (doctors, nurses, technicians). Nicotine dependence was controlled via the Fagerström Test. Measurements included pulse co-oximetry (Masimo Rad-7) for COHb and SpO₂, and standardized spirometry for FEV₁, FVC, FEV₁/FVC. **Results:** Group O exhibited significantly higher COHb levels and lower FVC values compared to Group M (p<0.05). Within the OT, doctors—maintaining the closest proximity to surgical smoke—showed significantly higher COHb levels than nurses and other personnel (p=0.001). Logistic regression identified OT exposure as a powerful independent predictor for elevated COHb (p=0.002) and reduced FVC (p<0.001). COHb levels negatively correlated with SpO₂ and FVC, confirming a synergistic physiological burden.

Keywords: Respiratory function test, Carboxyhemoglobin, Healthcare worker.

1. Introduction

Tobacco use remains one of the primary causes of preventable morbidity and mortality worldwide¹. Globally, approximately 1.5 billion individuals consume tobacco. While China, India, and Indonesia lead in total volume, Turkey ranks among the top ten countries with the highest prevalence, involving 17–18 million users (31.2% of the

population aged ≥ 15). Collectively, these ten high-prevalence nations account for nearly two-thirds of global tobacco consumption^{1,2}.

Carbon monoxide (CO) in cigarette smoke significantly impairs oxygen delivery to tissues by increasing the formation of carboxyhemoglobin (COHb) due to its high affinity for hemoglobin³. This condition may induce more pronounced physiological effects in occupational groups working under high work load, irregular breaks, and stress, such as healthcare professionals⁴. Operating theaters and inpatient wards represent two critical clinical settings that warrant comparison regarding respiratory functions and COHb levels, owing to disparities in working conditions and exposure patterns⁵.

Tobacco use directly compromises respiratory mechanical integrity beyond systemic intoxication. Elevated COHb levels are often accompanied by impaired lung ventilation, increased airway resistance, and diminished respiratory capacity. In addition to declining spirometric parameters, the degree of nicotine dependence significantly correlates with both consumption volume and the severity of physiological damage^{6,7}.

It is reported that electrocautery (surgical) smoke consists of approximately 150 different toxic chemicals, including phenol, ethanol, chloroform, benzene, toluene, ethylbenzene, xylenes, and naphthalene^{8,9}. It is suggested that these components can trigger genetic mutations and lead to carcinogenesis. In experimental studies, tumor formations have been reported following the administration of surgical smoke to laboratory animals¹⁰. Furthermore, analyses have demonstrated that the carcinogenic potential of specific chemical compounds within surgical smoke significantly exceeds established threshold values^{9,11}.

Since inhaled anesthetic gases—particularly nitrous oxide (N₂O), sevoflurane, desflurane, and isoflurane—are largely excreted unchanged via exhalation, they can accumulate in operating theaters and post-anesthesia care units, leading to chronic occupational exposure among healthcare professionals¹². Occupational exposure to these agents has been associated with potential adverse effects on the reproductive, neurological, and respiratory systems. The chemical properties and atmospheric residence times of these gases vary; specifically, the prolonged atmospheric life of desflurane and N₂O is thought to contribute to the cumulative risk profile associated with their use^{13,14}. Inhalational gases may impair lung capacity and spirometric indices, with effects potentially exacerbated in healthcare workers who smoke¹³.

While previous studies have investigated the effects of tobacco use, surgical smoke, and waste anesthetic gases separately, limited data are available regarding their combined impact on respiratory function and COHb levels among healthcare personnel. Therefore, this study aimed to compare COHb levels, pulmonary function parameters, and nicotine dependence among smoking healthcare workers employed in operating theaters and inpatient wards. Additionally, the study sought to evaluate the potential association between occupational exposure in the operating theater environment and respiratory health outcomes.

2. Materials and Methods

2.1. Study Design and Setting

This cross-sectional study was conducted at Aydın Adnan Menderes University Hospital between October and November 2025. Prior to the commencement of the study, approval was obtained from the local ethics committee (Protocol No: 2025/274). Additionally, informed consent was obtained from all participants before the measurements were performed. The study was conducted in accordance with the principles of the Declaration of Helsinki.

2.2. Study Population

This cross-sectional study compared COHb levels, respiratory functions, and nicotine dependence among smoking healthcare personnel in operating theaters and inpatient wards. Inclusion criteria comprised active smokers aged 25–60, classified as ASA I–II, who provided written informed consent. Participants were required to be free of asthma, COPD, or any upper respiratory tract infection (URTI) within the previous two weeks. Conversely, individuals outside the age range, non-smokers, those with chronic pulmonary diseases, or those having recent URIs were excluded. Furthermore, participants with occupational or environmental factors that could confound carbon monoxide exposure, including cigarette smoking, biomass smoke exposure, occupational exposure to combustion products, and significant environmental air pollution, were also excluded.

2.3. Assessment Parameters

The demographic and clinical characteristics of the participants were recorded using a standardized data collection form. In this context, the following parameters were documented: work environment (operating theater/inpatient ward), gender, age, height, body weight, body mass index (BMI), education level, years of employment, occupational group, pregnancy status, history of chronic disease, daily cigarette consumption, duration of smoking (years), time elapsed since the last cigarette smoked, time of last caffeine intake, physical activity level, heart rate, peripheral oxygen saturation (SpO₂), pulmonary function tests (FEV₁, FVC, PEF, FEV₁/FVC), Fagerström Test for Nicotine Dependence (FTND), and COHb levels. To minimize inter-measurement variability, all measurements were performed by the same doctoral student in physiology (table 1).

Table 1: Demographic and Smoking Characteristics of Operating Theater and Non-Operating Theater Personnel.

Parameters	Operating Theater (n=141)	Internal Medicine Ward (n=140)	p-value
Gender (Female/Male)	63/78	71/69	0.311
Age (years) (mean±SD))	33 (28-42)	33 (28-43)	-
Body Mass Index (median± 25-75) (kg/m ²)	24.91 (22.7-26.9)	25.66 (22.84-29.11)	0.136
Years of Employment	10 (3.25-15)	10 (4-16.75)	0.717
Occupation (MD/AT/Nurse/HA)*	32/27/39/43	24/24 (45/47)	0.589
Daily Cigarette Consumption (n)	20 (30-20)	20 (10-20)	0.109
Time Since Last Cigarette (min)	45 (30-90)	45 (30-77.5)	0.511
Duration of Smoking (years)	10 (5-19)	11 (6-20)	0.242

* Median/Anesthesia technician /Nurse/Health Atendant

2.3.1. Measurement of Carboxyhemoglobin

The COHb levels of the participants were measured non-invasively using the Masimo Rad-7® pulse CO-oximeter (Masimo Corp., Irvine, CA, USA). Measurements were performed while the participants were in a resting position, in a well-ventilated room with stable ambient temperature and humidity to ensure identical environmental conditions, and in accordance with the manufacturer's standard measurement protocol. The sensor was shielded from high-intensity ambient light to prevent potential optical interference during pulse CO-oximetry.

2.3.2. Pulmonary Function Tests

Pulmonary functions were assessed using a portable spirometer, calibrated daily with a 3-L syringe according to manufacturer guidelines. Spirometric maneuvers followed American Thoracic Society/ European Respiratory Society (ATS/ERS) technical

standards, with at least three acceptable maneuvers obtained per participant to ensure reproducibility. FEV₁, FVC, FEV₁/FVC ratio, and PEF were recorded, with the highest values utilized for analysis. All volumes were automatically corrected to BTPS (Body Temperature and Pressure, Saturated with water vapor) conditions by the device.

2.3.3. Assessment of Nicotine Dependence

The nicotine dependence levels of the participants were evaluated using standardized and validated scales

2.3.4. Fagerström Test for Nicotine Dependence

Nicotine dependence was assessed using the Fagerström Test for Nicotine Dependence (FTND), which yields scores from 0 to 10. Based on established criteria, scores were categorized as very low/none (0–3), moderate biological dependence (4–6), or high biological and psychological dependence (7–10)¹⁵.

2.4. Data Collection and Statistical Analysis

Data distribution was assessed using the Kolmogorov-Smirnov test. Continuous variables are presented as mean $\pm$ standard deviation or median (25th–75th percentiles) based on normality, while categorical data are expressed as frequencies and percentages. Inter-group comparisons were performed using the independent samples t-test or Mann-Whitney U test for two groups, and the Kruskal-Wallis test with post-hoc Bonferroni correction for multiple group comparisons. Categorical variables were analyzed using Chi-square tests. The relationship between physiological parameters and smoking variables was evaluated via Spearman correlation analysis. A logistic regression model and Classification and Regression Tree (CART) analysis were employed to identify independent predictors of the operating theater environment and COHb levels. Statistical significance was set at $p < 0.05$.

2.5. Sample Size

Sample size was calculated using G*Power (v3.1.9.2). Based on preliminary data for distinguishing operating theater effects on respiratory function (sensitivity: 69.3%; specificity: 68.1%), a minimum of 135–140 participants per group was estimated to achieve 80% power at a 5% Type I error rate ($\alpha = 0.05$). To compensate for potential data loss, the total sample was increased to 300 participants.

3. Results

A total of 300 healthcare professionals were initially enrolled; however, 281 participants (141 operating theater [OT] staff and 140 internal medicine ward staff) were included in the final analysis due to data completeness. Demographic characteristics, including age, height, years of employment, and baseline smoking habits, were comparable between groups ($p > 0.05$). Similarly, no significant differences were observed in baseline SpO₂, FEV₁, PEF, and nicotine dependence scores (FTND and SAI) ($p > 0.05$). In contrast, OT staff exhibited significantly lower FVC percentages and FEV₁/FVC ratios, alongside markedly higher COHb levels compared to the ward staff ($p < 0.05$) (table 2).

Table 2: Physiological and Respiratory Parameters by Work Environment

Parameters	Operating Theater (Group1) Median (25th-75th)	Non-Operating Theater (Group 2) Median (25th-75th)	p-value
Heart rate (bpm)	87 (76-95)	87 (77.25-94)	0.628
SpO ₂ (%)	98 (97-99)	98 (97-99)	0.237
FEV ₁ (L)	2.17 (1.69-3.21)	2.20 (1.67-2.79)	0.198
FVC (L)	2.4 (1.69-3.21)	3.04 (2.40-3.77)	0.0001

Parameters	Operating Theater (Group1) Median (25th-75th)	Non-Operating Theater (Group 2) Median (25th-75th)	p-value
FVC (% predicted)	65 (44-78)	73 (51.25-88.5)	0.002
PEF (L/s)	4.90 (3.63-6.10)	4.45 (3.31-6.41)	0.222
PEF(% predicted)	63 (45-82)	57.5 (42-83)	0.049
FEV1/FVC	0.96 (0.90-0.98)	0.97 (0.93-0.99)	0.0001
COHb (%)	0 (0-2)	0 (0-1)	0.0001
FTND (score)	5 (3-6)	5 (3-6)	0.0001
SAI (score)	3 (2-4)	3 (2-3.75)	0.253

Note: Data are presented as median (25th–75th percentiles). For COHb (%), values are reported as median (25th–75th percentiles) of the whole-number percentages measured by the Masimo RAD-7 pulse CO-oximeter (e.g., 0 [0–2] indicates a median COHb value of 0%, with the 25th and 75th percentiles of 0% and 2%, respectively). FTND scores represent the sum of questionnaire item scores and are therefore expressed as integer values. COHb: Carboxyhemoglobin; FEV₁: Forced Expiratory Volume in 1 second; FTND: Fagerström Test for Nicotine Dependence; FVC: Forced Vital Capacity; PEF: Peak Expiratory Flow; SAI: Smokeless Artificial Intelligence (Dependence) Score; SpO₂: Peripheral Oxygen Saturation.

Predictors of Elevated COHb Levels Logistic regression analysis was employed to identify factors associated with elevated COHb levels. Working in the OT environment, advanced age, and shorter time elapsed since the last cigarette were identified as independent significant predictors ($p < 0.05$). Notably, working in the OT environment increased the probability of high COHb levels approximately fivefold (OR = 4.976; 95% CI: 2.92–8.49; $p < 0.001$).

Correlation Between Physiological and Smoking Variables Spearman correlation analysis revealed that COHb levels positively correlated with tobacco-related parameters, including daily cigarette consumption ($r = 0.365$), smoking duration ($r = 0.247$), and nicotine dependence scores (FTND: $r = 0.365$; SAI: $r = 0.247$) ($p < 0.05$). Regarding physiological parameters, COHb levels showed a significant negative correlation with SpO₂ ($r = -0.247$, $p = 0.003$) and a weak negative correlation with FEV₁ ($r = -0.184$, $p = 0.029$). No significant correlations were found between COHb and other spirometric indices or physical activity levels ($p > 0.05$).

Occupational Groups and Environmental Influence Kruskal-Wallis analysis demonstrated significant variations in COHb levels across occupational roles ($p = 0.001$). Post-hoc pairwise comparisons revealed that medical doctors had significantly higher COHb levels compared to nurses and other healthcare staff ($p < 0.05$). Subgroup analysis indicated that this occupational discrepancy was exclusive to the OT environment ($p < 0.05$), whereas no such difference existed within internal medicine wards ($p > 0.05$).

Environmental Modeling and Decision Tree Analysis The logistic regression model for predicting the OT environment achieved an overall classification accuracy of 78.3%. COHb levels were independently associated with the OT environment (OR = 1.232; 95% CI: 1.083–1.402; $p = 0.002$), while FVC exhibited a strong inverse relationship (OR = 0.099; 95% CI: 0.055–0.179; $p < 0.001$). Classification and Regression Tree (CART) analysis further supported these findings, identifying FVC as the most influential variable in distinguishing OT personnel from ward staff (relative importance = 100%), followed by the FEV₁/FVC ratio (relative importance = 50%). In addition, COHb levels emerged as one of the major variables contributing to the classification of participants according to their working environment (Figure 1).

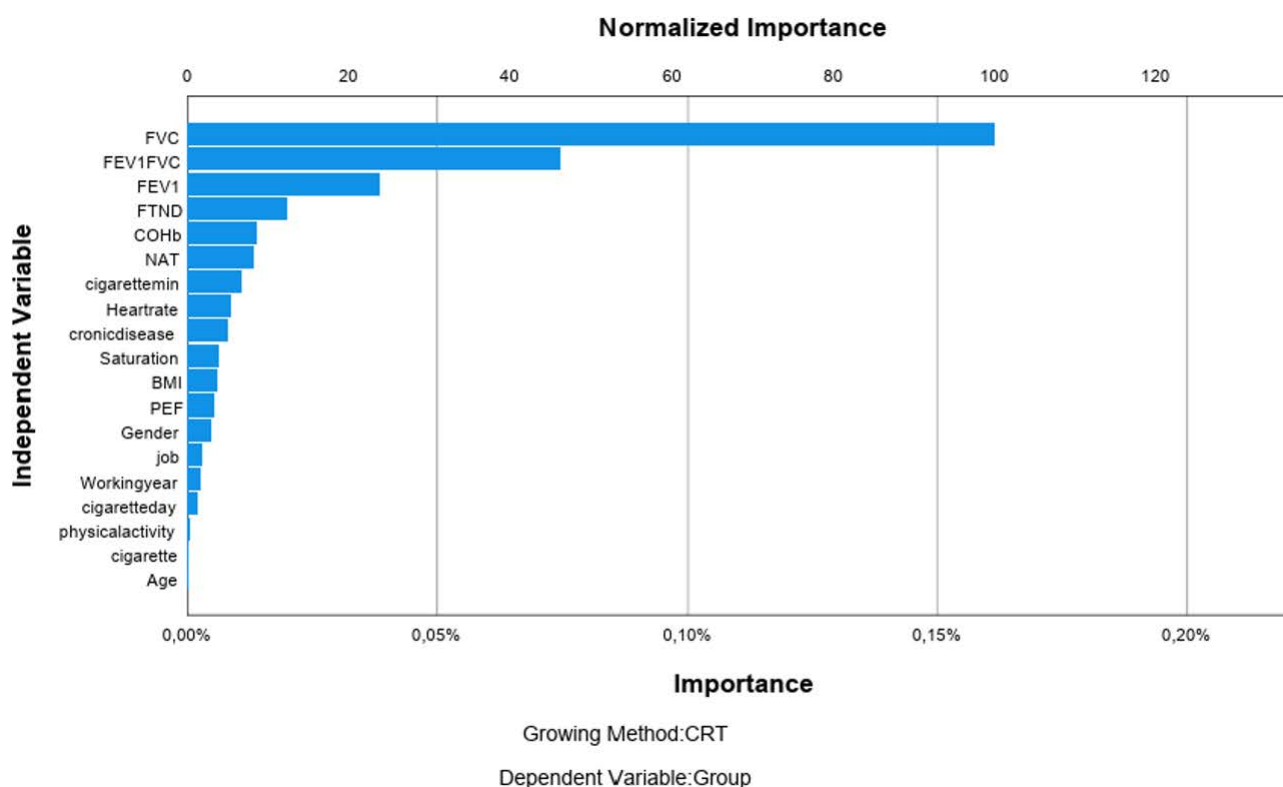


Figure 1: Variable importance plot illustrating the relative significance of pulmonary function tests (FVC, FEV1/FEV1/FVC and de) mographic/clinical data within the model.

4. Discussion

Operating theater personnel exhibited significantly higher COHb levels and reduced FVC, indicating a distinct functional respiratory impairment. Our findings suggest that the OT microenvironment acts as a primary determinant of restrictive ventilatory patterns, independently affecting FVC parameters. Notably, elevated COHb levels remained associated with OT work after adjusting for smoking habits, pointing toward an exogenous carbon monoxide burden from surgical smoke. These results support the hypothesis that occupational exposure to surgical plumes and waste gases induces cumulative biophysiological stress, potentially compromising alveolar-capillary gas exchange efficiency¹⁶.

Elevated COHb levels in the OT group suggest a physiological burden exceeding tobacco consumption alone, likely involving synergistic contributions from electrocautery-induced surgical smoke and waste anesthetic gases. Our data align with literature documenting higher COHb concentrations among healthcare workers in high-circulation, closed clinical settings¹⁷⁻¹⁹. These findings reinforce the premise that the OT microenvironment constitutes an independent occupational risk factor, imposing an exogenous carbon monoxide load that exceeds the physiological effects of individual smoking behavior alone.

Alterations in OT personnel's pulmonary function likely reflect chronic irritation and oxidative stress from chemical and particulate exposure, augmenting the effects of smoking. While high-risk environments are associated with respiratory decline¹⁹⁻²¹, our results suggest that FEV₁/FVC variations represent subclinical functional shifts rather than overt obstruction. Notably, FVC impairment correlated more strongly with the OT environment than with COHb levels, indicating that respiratory changes may be a distinct

physiological response to inhaled particulate matter, independent of systemic carbon monoxide burden.

In a comparative cross-sectional study, Ibrahim et al. (2025) reported significantly lower FVC, FEV₁, and PEF values among healthcare personnel working in operating theaters compared to those in other units^{12,22}. This cross-study consistency supports the hypothesis that surgical respiratory irritants adversely affect pulmonary mechanics. Aligning with recent literature, these findings identify the OT microenvironment as a potential risk zone for subclinical lung impairment, highlighting the need for continuous physiological monitoring of surgical teams.

Mirsaeed et al. (2013) linked reduced FVC to restrictive patterns involving chronic inflammation and decreased lung compliance secondary to inhalation exposures²³. Similarly, the lower FVC values in our OT cohort suggest comparable pathophysiological mechanisms. While previous research emphasized subjective symptoms of surgical smoke^{24,25}, this study provides an objective quantification by correlating spirometric indices with COHb. Notably, these functional shifts appear distinct from acute carbon monoxide effects, suggesting a direct environmental impact on pulmonary mechanics.

In the logistic regression analysis, COHb, FEV₁, and FVC were identified as independent factors associated with the operating theater environment. Supporting this observation, recent research has indicated that non-traditional inhalation exposures among OT personnel are linked to significant reductions in spirometric parameters, with a more pronounced decline as exposure duration increases^{21,26}. The statistical independence of these variables suggests that observed pulmonary alterations represent a direct physiological response to the complex surgical microenvironment, rather than a secondary effect of systemic carbon monoxide elevation.

Higher COHb levels and distinct pulmonary profiles in smoking OT personnel align with the chemical and biological hazard frameworks of Barrett and Garber. Toxic components in surgical smoke, mirroring those in tobacco, suggest that OT staff face a dual respiratory burden from combined cigarette and occupational smoke exposure²⁷⁻²⁹. Impairments in volume-based parameters, such as FVC, likely reflect chronic adaptive responses or irritative damage through pathways distinct from systemic COHb elevation. These findings identify the surgical environment as a discrete contributor to long-term functional respiratory shifts.

Elevated COHb and distinct pulmonary profiles in smoking OT personnel align with literature characterizing surgical smoke as an under-recognized occupational hazard. Ulmer et al. (2008) emphasize that concurrent exposure to surgical smoke and tobacco may induce cumulative biophysiological effects on respiratory health³⁰. Our findings lend support to the hypothesis that this dual exposure contributes to the observed variations in functional respiratory parameters, highlighting the need for enhanced protective measures in surgical settings.

The time-dependent relationship between smoking and COHb levels is well-established in the literature³¹. Results identify smoking recency as a critical determinant of COHb, reflecting the acute dynamics of carbon monoxide kinetics. The inverse relationship between the time elapsed since the last cigarette and COHb levels aligns with documented post-inhalation spikes followed by gradual elimination. As an independent predictor, smoking recency highlights the need for standardized timing protocols in occupational screenings to accurately differentiate tobacco-derived from environment-induced CO exposure and avoid clinical misinterpretation.

Recent literature identifies surgical smoke as a proximity-dependent inhalation hazard. Zhou et al. (2023) highlighted that toxic gases, including carbon monoxide and particulate matter, are more concentrated at the surgical field's center³²⁻³⁴. Consistently, our study reveals significantly higher COHb levels in medical doctors—those in closest

proximity to the smoke source—compared to other OT staff. This disparity was absent in ward settings, confirming that elevated COHb is driven by localized environmental exposure rather than smoking habits alone. These findings identify the surgical suite as a primary site of localized toxic inhalation.

Reports by Smith et al. (2018) indicate that even low-level exposure to volatile toxic substances, such as carbon monoxide, can induce measurable physiological effects³⁵. This suggests that the elevated COHb levels in smoking operating theater personnel likely reflect an additional carbon monoxide load derived from surgical smoke. Furthermore, Yu et al. (2022) demonstrated the adverse effects of electrocautery-induced plumes on biological and hematological parameters, supporting the premise that these elevations are not solely attributable to tobacco use³⁶. Notably, the systemic irritative effects of electrocautery-derived smoke may influence respiratory functions through mechanisms independent of COHb levels, such as oxidative stress or airway remodeling. Consequently, this group exhibits a higher risk profile compared to smoking healthcare professionals in non-surgical settings.

Barletta and Canicoba (2025) emphasize that chronic exposure to surgical smoke induces significant respiratory and systemic effects, representing a substantial occupational risk³⁷. Elevated COHb levels and altered pulmonary profiles in our OT cohort reinforce the necessity of recognizing the operating theater as a distinct environment. These findings demonstrate that occupational pollutants exacerbate the physiological burden of tobacco use, necessitating targeted respiratory protection strategies.

The observed adverse effect of COHb on oxygen saturation aligns with established physiological frameworks. Binny et al. (2024) identified COHb as a clinically relevant biomarker for tobacco exposure, linking elevated levels to decreased saturation³⁸. Our data confirm that the negative correlation between COHb and SpO₂ reflects competitive carbon monoxide binding, which impairs oxygen-carrying capacity. These findings underscore the risk of systemic oxygenation deficits in high-exposure environments like operating theaters, especially for professionals with pre-existing tobacco burdens.

4.1. Limitations of the Study

This study has several limitations. Its cross-sectional design precludes causal inferences regarding the long-term respiratory impact of the operating theater (OT) environment. The single-center nature of the sampling and single-point COHb assessments may limit broader generalizability and fail to capture diurnal fluctuations in exposure. Crucially, this study specifically targeted smoking healthcare professionals to isolate and quantify the incremental physiological burden imposed by the OT environment on an already compromised respiratory system. Consequently, a non-smoking control group was not included, as the primary objective was to evaluate the synergistic risk rather than the baseline effects of tobacco. Furthermore, while the absence of direct environmental monitoring for ambient pollutants prevents a precise dose-response analysis, the utilization of professional roles as a proxy for "distance-to-hazard" provides a significant indirect assessment of exposure levels. Despite these limitations, the application of robust statistical modeling, including logistic regression, alongside standardized physiological measurements, offers an objective and novel framework for understanding occupational health in surgical settings.

5. Conclusion

In conclusion, smoking healthcare personnel working in operating theaters exhibited higher COHb levels and lower FVC values compared with smoking personnel working in inpatient wards. These findings suggest that occupational exposure within the operating theater environment may contribute to additional respiratory burden beyond the

effects of tobacco smoking alone. The observed association between the operating theater environment, elevated COHb levels, and altered pulmonary function highlights the importance of preventive strategies, including effective smoke evacuation systems and appropriate occupational health surveillance. Further prospective and longitudinal studies are needed to clarify the long-term respiratory consequences of combined exposure to tobacco smoke, surgical smoke, and anesthetic gases.

genAI: During the preparation of this manuscript, the authors used ChatGPT for English language editing. After using this service, the authors thoroughly reviewed and revised the content as needed, and they take full responsibility for the final published version of the manuscript.

Data Availability Statement: The datasets generated and/or analyzed during the current study are not publicly available due to privacy and ethical restrictions involving human participants but are available from the corresponding author on reasonable request.

Conflict Of Interest: The authors declare that they have no competing interests or financial conflicts of interest related to this study.

Author Contributions: Idea/Conceptualization: SŞ, İK, FŞ. Methodology: SŞ, FŞ. Investigation: İK, CÖ, MZA. Formal Analysis: İKÖ, SŞ. Writing - Original Draft: İK, FŞ, SŞ. Writing - Review & Editing: SŞ, FŞ. Supervision: FŞ, SŞ. Critical Review: SŞ, İK, FŞ, CÖ, MZA, İKÖ. Final Approval: All authors have read and agreed to the published version of the manuscript.

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