

Evaluation of Hemoglobin Levels and Oxygen Saturation of Hemoglobin in Healthy Male Smokers and Nonsmokers

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ABSTRACT

Background: Cigarette smoking has been well documented as a significant contributor to hypoxia, primarily due to carbon monoxide (CO) exposure, which reduces the oxygen-carrying capacity of hemoglobin (Hb) by binding with it. This compensatory effect often leads to changes in Hb levels and oxygen saturation (SpO₂). This study aims to compare these parameters in healthy male smokers and nonsmokers to assess the impact of smoking on blood oxygenation.

Objective: The objective of this study is to evaluate and compare Hb levels and SpO₂ of hemoglobin in healthy male smokers and nonsmokers.

Materials and methods: This analytical cross-sectional study was conducted at the Government Cuddalore Medical College and Hospital. The study comprised two groups, each with 50 participants aged 20–45 years—group A included healthy male smokers, and group B consisted of healthy male nonsmokers, with participants selected through purposive sampling. Hb levels were measured using an automated hematology analyzer, while SpO₂ was assessed via fingertip pulse oximetry. Data were analyzed using SPSS software, with descriptive statistics and independent *t*-tests to compare mean Hb levels and SpO₂ between groups. A *p*-value of <0.05 was considered statistically significant.

Results: Findings revealed that smokers had significantly lower mean SpO₂ levels (96.67 ± 1.56) compared to nonsmokers (99.30 ± 1.64) (*p* < 0.05). Additionally, a significant difference in mean Hb levels between smokers (15.25 ± 1.4) and nonsmokers (13.81 ± 0.21) was observed, with higher Hb concentration among smokers, indicating compensatory erythrocytosis. In the current study, there was no statistically significant influence of factors such as duration of smoking, current smoking status, or smoking intensity on the Hb and hematocrit (Hct) levels in smokers.

Conclusion: The study demonstrates that cigarette smoking results in increased Hb levels and decreased SpO₂, indicating a potential risk for chronic hypoxia and subsequent cardiovascular diseases. These findings underscore the importance of smoking cessation for maintaining optimal oxygen transport and cardiovascular health.

Keywords: Cardiovascular risk, Hemoglobin, Hypoxia, Oxygen saturation, Smoking.

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INTRODUCTION

Smoking remains a leading public health concern, widely recognized for its detrimental effects on health and its significant role in the development of various diseases. The World Health Organization (WHO) estimates that tobacco use causes >8 million deaths annually, making it one of the primary preventable causes of morbidity and mortality worldwide. The harmful effects of smoking extend beyond respiratory illnesses, influencing multiple systems within the body, including the cardiovascular and hematological systems.

One of the critical physiological impacts of smoking is its contribution to hypoxia, a condition characterized by inadequate oxygen supply to tissues. Hypoxia can arise from several mechanisms related to smoking, with carbon monoxide (CO) exposure being a significant factor. When inhaled, CO binds with hemoglobin (Hb) in red blood cells, forming carboxyhemoglobin, which reduces the oxygen-carrying capacity of Hb. This binding impairs the ability of Hb to transport oxygen, leading to decreased oxygen availability for vital bodily functions.

Alterations in Hb levels and oxygen saturation (SpO₂) are common consequences of smoking and have profound implications on health.¹ Decreased SpO₂ can lead to increased strain on the cardiovascular system and may contribute to the development of various smoking-related diseases, including chronic obstructive

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pulmonary disease (COPD), lung cancer, and cardiovascular disorders. Moreover, these physiological changes can significantly impact the overall quality of life and functional capacity of individuals who smoke.

Assessing Hb and SpO₂ levels in smokers compared to nonsmokers provides valuable insights into the extent of these physiological impacts.² Understanding these differences can aid in clinical diagnosis, guide risk assessments for smoking-related diseases, and inform strategies for health interventions, including smoking cessation programs.

Aims and Objectives

Aim

To evaluate and compare the Hb levels and SpO₂ in healthy male smokers and nonsmokers.

Objectives

- To evaluate the Hb levels in smokers and nonsmokers.
- To evaluate SpO₂ levels in smokers and nonsmokers.
- To classify the smokers into three classes—mild, moderate, and severe smoking—and to correlate their Hb and SpO₂ values.

MATERIALS AND METHODS

After obtaining ethical committee approval from the Institutional Ethics Committee (IEC), the study was conducted at the Government Cuddalore Medical College and Hospital, employing a cross-sectional, comparative observational design.

The sample size was 100, calculated for an analytical cross-sectional study. A total of 100 clinically healthy male volunteers aged 20–45 years who attended the daily outpatient department (OPD) were selected by purposive sampling. After obtaining informed consent, they were divided into two groups—group A (smokers), which included 50 individuals with a history of smoking for at least 12 months, and group B (nonsmokers), comprising 50 individuals who affirmed they had never smoked.

Exclusion criteria included participants with a history of any acute or chronic illness, bronchial asthma, COPD, tuberculosis, heart failure, anemia, bleeding disorders, drug addiction, or those who had undergone recent blood donation within the past 6 months.

Data collection involved informed consent, gathering smoking history through a pretested questionnaire that captured current smoking status, the number of cigarettes smoked per day, and the duration of smoking in years, and pack year was calculated. Pack years is a measure of the amount a person has smoked over a long period of time. It is calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked.³

Participants were classified based on their smoking intensity into three categories—mild smokers (10–14 pack years), moderate smokers (15–19 pack years), and heavy smokers (20 or more pack years). About 5 mL of venous blood sample was collected from the cubital vein in an ethylenediaminetetraacetic acid (EDTA)-coated tube, and Hb levels were measured using an automated hematology analyzer, while SpO₂ was assessed *via* fingertip pulse oximetry.

RESULTS

For data analysis, was performed using SPSS 19 version. Descriptive statistics, independent t-tests, and ANOVA were performed, with a significance level set at a *p*-value of <0.05 (Table 1).

These results indicate a statistically significant difference in both Hb and SpO₂ levels between smokers and nonsmokers. Smokers had a higher mean Hb level of 15.25 gm/dL compared to nonsmokers, whose mean was 13.81 gm/dL. Additionally, the mean SpO₂ for smokers was 96.67%, significantly lower than the 99.30% observed in nonsmokers.

Furthermore, we assessed Hb and SpO₂ levels across different smoking intensity categories. The analysis revealed no statistically significant differences in Hb levels (*p* = 0.247) among the different smoker groups. However, there was a highly significant difference in SpO₂ levels (*p* = 6.68 × 10⁻¹⁹), indicating that heavier smoking intensity correlates with lower SpO₂ (Table 2).

DISCUSSION

The findings of this study reveal critical insights into the physiological impacts of smoking on hematological and respiratory parameters. The significantly higher Hb levels in smokers may be a compensatory response to chronic hypoxia caused by smoking. As CO binds to Hb, it can result in reduced oxygen delivery to tissues, prompting the body to produce more Hb to enhance oxygen transport.⁴ However, the resulting increase in Hb does not correlate with improved SpO₂, as evidenced by the lower SpO₂ levels in smokers compared to nonsmokers.

The statistically significant difference in SpO₂ further emphasizes the adverse effects of smoking on respiratory function. The reduced SpO₂ levels in smokers reflect compromised oxygenation due to impaired gas exchange, potentially leading to a range of health issues, including increased cardiovascular strain and a higher risk of respiratory diseases.⁵

While our analysis showed no significant differences in Hb levels across the mild, moderate, and heavy smoker groups, the notable differences in SpO₂ levels suggest that smoking intensity has a profound impact on SpO₂. The data support the understanding that heavier smokers experience greater hypoxia and highlight the need for targeted interventions to address the health risks associated with smoking.

These results align with findings from studies by Anandha et al.⁶ and Saranya and Krishnaveni,⁷ reinforcing the understanding of the physiological effects of smoking on oxygen transport and utilization.^{8–12}

Table 1: Comparison of Hb and SpO₂ values between smokers and nonsmokers

Parameter	Smokers (Mean ± SD)	Nonsmokers (Mean ± SD)	<i>p</i> -value
Hb (gm/dL)	15.25 ± 1.4	13.81 ± 0.21	<0.05
SpO ₂ (%)	96.67 ± 1.56	99.30 ± 1.64	<0.05

Table 2: Comparison of Hb and SpO₂ values between various degrees of smokers

Parameter	Mild smokers (pack years 10–14) (Mean ± SD)	Moderate smokers (pack years 15–19) (Mean ± SD)	Heavy smokers (pack years 20 and above) (Mean ± SD)	<i>p</i> -value
Hb (gm/dL)	15.06 ± 1.28	14.98 ± 1.39	14.54 ± 1.43	0.247
SpO ₂ (%)	96.9 ± 1.33	95.6 ± 1.42	93.4 ± 1.23	<0.05

CONCLUSION

Overall, this study underscores the importance of regular monitoring of Hb and SpO₂ levels in smokers, as these parameters can serve as valuable indicators of smoking-related health risks. Health interventions, including smoking cessation programs, should consider these physiological alterations to effectively address the health challenges associated with tobacco use.

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