

## Comparison of Total and Differential Leukocyte Counts and Hemoglobin Oxygen Saturation Between Healthy Smokers and Non-Smokers

Dinesh Kumar<sup>1</sup>, Pansy Lyall<sup>2</sup><sup>1</sup>Associate Professor, Department of Physiology, Gouri Devi Institute of Medical Sciences, Durgapur, West Bengal, India<sup>2</sup>Professor and HOD, Department of Physiology, Gouri Devi Institute of Medical Sciences, Durgapur, West Bengal, India

Received: 10-05-2025 / Revised: 25-06-2025 / Accepted: 28-07-2025

Corresponding Author: Dr. Dinesh Kumar

Conflict of interest: Nil

**Abstract:****Background:** Smoking is a public health problem globally, and it has a significant impact on hematological and respiratory indices. In West Bengal, where the habit of smoking is common, it is important to know the effect of smoking on oxygen saturation (SpO<sub>2</sub>), total leukocyte count (TLC), and differential leukocyte count (DLC) for early detection of the physiologic changes caused by smoking.**Objective:** The objective was to compare TLC, oxygen saturation, and DLC of hemoglobin between non-smokers and smokers from West Bengal.**Methodology:** A cross-sectional comparative study was done among 100 adults (60 smokers, 40 non-smokers) between 21 and 55 years. Venous blood was tested for TLC and DLC on an automated hematology counter, and SpO<sub>2</sub> was measured non-invasively by pulse oximetry. Statistical tests were carried out using independent t-tests at a significant level of  $p < 0.05$ .**Results:** Smokers exhibited significantly higher TLC ( $7.36$  vs.  $6.95 \times 10^3/\mu\text{L}$ ,  $p < 0.001$ ), granulocyte ( $0.589$  vs.  $0.575$ ,  $p = 0.01$ ), and monocyte counts ( $0.056$  vs.  $0.053$ ,  $p = 0.02$ ) compared to non-smokers. Conversely, lymphocyte count ( $0.366$  vs.  $0.371$ ,  $p < 0.001$ ) and oxygen saturation ( $98.2\%$  vs.  $98.7\%$ ,  $p = 0.04$ ) were significantly lower in smokers.**Conclusion:** In healthy subjects, smoking provokes systemic inflammatory responses characterized by leukocytosis and a mild drop in oxygen saturation, which could predispose them to cardiovascular and pulmonary complications later. The observations suggest the imperative for early screening and public intervention for the correction of smoking-induced physiological deficits within the WB population.**Keywords:** West Bengal, Differential Leukocyte Count, Hemoglobin, Oxygen Saturation, Smoking, Total Leukocyte Count.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

**Introduction**

Tobacco smoking remains a universal public health emergency, causing an estimated 8 million deaths every year, 1.2 million of whom are non-smoking victims of second-hand smoke [1]. According to the Global Adult Tobacco Survey (GATS-2), the prevalence rate of tobacco use in WB continues to be high, with more than 25% of the total population actively smoking [2]. Smoking's biochemical effects are not confined to the lung alone but also involve systemic effects, particularly on hematologic and respiratory indices, including white blood cell (WBC) counts and oxygen-carrying capacity of the blood [3].

Cigarette smoke harbors over 4,000 toxic chemicals, most of which are effective oxidants, carcinogens, and immunomodulators, and which include but are not limited, to nicotine, CO, benzene, and polycyclic aromatic hydrocarbons [4]. All these constituents

trigger chronic inflammation, immune dysfunction, and oxidative stress. A number of studies have shown that smoking stimulates leukocytosis and changes the differential leukocyte count (DLC), most notably elevating neutrophils and monocytes as part of the systemic inflammatory response [5,6]. These hematologic irregularities are important since they are both biomarkers for subclinical inflammation and independent predictors of cardiovascular morbidity and mortality [7].

White cells or leukocytes are the central players in host defence. In a healthy situation, their number stays within a tightly regulated range. Cigarette smoke, however, provokes chronic low-grade inflammation, and increased interleukins (e.g., IL-6, IL-8), tumor necrosis factor-alpha (TNF- $\alpha$ ), and granulocyte colony-stimulating factors, which

together act to produce leukocytosis [8]. Nicotine, the major alkaloid present in tobacco, acts by binding to nicotinic acetylcholine receptors (nAChRs) and causing increased sympathetic discharge, release of catecholamines, and finally, leukocyte demargination and mobilization [9].

In oxygen transport, hemoglobin saturation (SpO<sub>2</sub>) supplies an objective indicator of oxygen binding to hemoglobin units within the circulatory system. While under normal circumstances hemoglobin binds about 1.34 mL of O<sub>2</sub> per gram, smoking severely depresses this activity through the high plasma concentrations of carboxyhemoglobin generated by CO breathing [10]. Carboxyhemoglobin contains more than 200 times as much affinity for hemoglobin as oxygen, decreasing areas available for binding and reducing oxygenation of the tissues. Tissue hypoxia and malfunctioning organs may be caused by chronic smokers, who can show reduced peripheral oxygenation [11].

In environmentally heterogeneous as well as resource-poor environments like WB, both passive and active smoking is common, and knowing the hematological and physiologic effects of smoking is crucial for both clinical and public health practice. Furthermore, rural West Bengal frequently experience other environmental stresses like biomass fuel combustion indoors and industrial pollution, which could enhance the harmful effects of tobacco smoke on leukocyte behavior and pulmonary gas exchange [12].

Despite numerous international studies being carried out on the hematologic effects of smoking, there is a scarcity of region-specific data for Eastern India but more so for West Bengal. Genetic background, way of life, and tobacco composition (e.g., bidi smoking vs. cigarettes smoking) could lead to varying biological responses [13]. The use of region-specific data for clinical diagnosis avoids contextual inaccuracies, increases the predictiveness of hematological predictors, and allows for targeted prevention.

The present study plans to compare and assess the Oxygen saturation (SpO<sub>2</sub>), total leukocyte count (TLC), and differential leukocyte count (DLC) between healthy non-smokers and smokers within the West Bengal population. Through comparison of these factors within a demographically representative population, the study aims to outline the physiological signature of smoking and provide evidence-based recommendations that could be used to enhance diagnosis and public health policy.

Ultimately, detection of early hematologic and respiratory anomalies among asymptomatic people can open doors to prevent strategies against smoking morbidities. These efforts are particularly crucial in WB, which lags in health care accessibility and preventive screening. This study then adds to the existing science discussion on the systemic effects of

smoking, focusing on the necessity for site-specific studies within public health research.

### Methodology

**Study Design:** This was a comparative cross-sectional study aimed at evaluating the differences in Oxygen saturation (SpO<sub>2</sub>), total leukocyte count (TLC), and differential leukocyte count (DLC) of hemoglobin between healthy non-smokers and smokers.

**Study Area:** The study was conducted in the Department of Physiology, Gouri Devi Institute of Medical Sciences, Durgapur, West Bengal, India. However, participants included in the study were selected from the West Bengal region to specifically assess the physiological effects of smoking in that population.

**Study Duration:** The study was carried out over a one period, allowing sufficient time for recruitment, data collection, and analysis.

**Sample Size:** A total of 100 adult volunteers, aged between 21 and 55 years, were enrolled in the study. Participants were divided into two groups:

- **Smokers (n = 60):** Individuals who smoked cigarettes or bidis daily for at least one year.
- **Non-Smokers (n = 40):** Individuals who did not smoke any tobacco products.

**Data Collection:** Data was collected through anthropometric and physiological examinations. The height and weight of participants were first recorded, and participants' BMI (Body Mass Index) was determined by using the standard formula. A prepared questionnaire was used to collect detailed histories of participants' smoking experience, i.e., duration and frequency of using tobacco. Three milliliters of venous blood was drawn under aseptic conditions from the antecubital vein into EDTA vacutainers. Samples collected through this procedure were then counted using the MS-9 automated haematology cell counter to compute TLC (total leukocyte count) expressed as thousands and DLC (differential leukocyte count) expressed as percentages. All samples were analysed on the same day within 3–5 hours of their collection.

Hemoglobin oxygen saturation was detected non-invasively by a pulse oximeter (fingertip, Nidek Medical 5300). Prior to measurement, each subject was required to be seated and rest quietly for 5 to 15 minutes to obtain stabilized physiology. The sensor was attached to the index finger, or an alternate one, as necessary. Following an initial reading on the screen, six readings over 10-second intervals were recorded consecutively. The overall average over the six recordings was determined and was reported as the individual's final oxygen saturation.

### Inclusion Criteria

- Adults aged 21–55 years.
- Clinically healthy individuals based on history and clinical examination.
- Non-smokers who have never smoked or used tobacco products.
- Smokers with a consistent smoking history (daily for at least one year).

### Exclusion Criteria

- Ex-smokers or individuals with a past history of smoking.
- Individuals with acute or chronic illnesses, bleeding disorders, or drug addiction.
- Pregnant women or those who had delivered within the last three months.
- Subjects who had donated blood in the last six months.”

**Procedure:** All the procedures followed standard clinical and laboratory protocols. Participants were initially screened for eligibility according to the inclusion and exclusion criteria. Following verbal or written consent, anthropometric information and smoking status were recorded. Blood samples and oxygen saturations were then taken according to the set procedures.

**Statistical Analysis:** Statistical analysis was carried out using SPSS software version 26 (SPSS Inc., Chicago, IL, USA). Normality was checked using the

Shapiro–Wilk and Kolmogorov–Smirnov tests, and through examination of skewness, kurtosis, and the use of probability plots. According to data distribution, parametric tests were employed. For comparison between means of continuous data like SpO<sub>2</sub>, DLC, and TLC between non-smokers and smokers, the independent t-test was employed. Pearson’s correlation analysis was used to establish relationships between the selected hematological indices and oxygen saturation values. We used two tailed tests and a cut-off p-value less than 0.05 for determining statistical significance. Results were reported as mean ± standard deviation (SD) to present an effective summary of the data’s central tendency and dispersion.

### Result

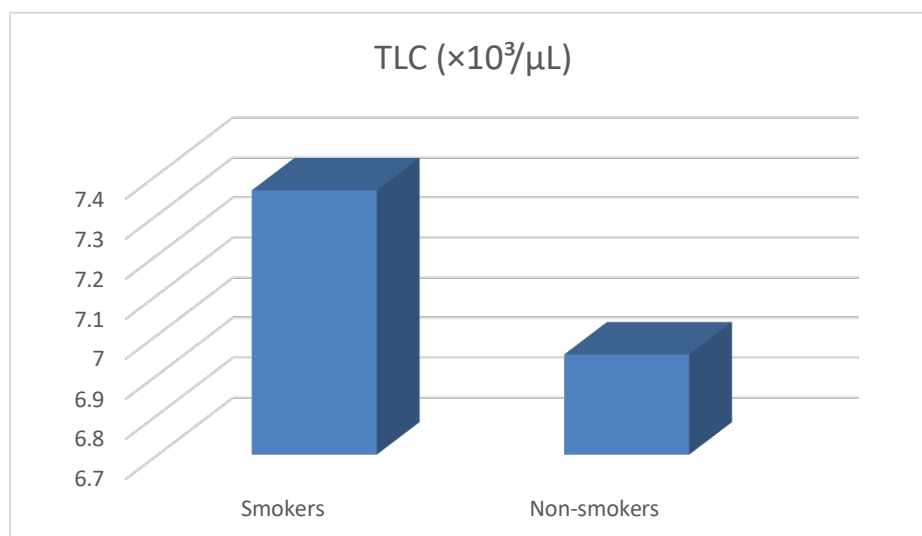
Table 1 shows the comparison between smokers and non-smokers' baseline demographic characteristics. Smokers had a minimally higher mean age at 32.8 years (SD = 8.2) than non-smokers, who had a mean age of 31.7 years (SD = 9.25). The minimum to maximum ages for smokers and non-smokers were 29 years (range 21–50) and 35 years (range 20–55), respectively. Smokers had a higher, albeit minimally so, mean BMI at 25.2 (SD = 2.97) and a range of 11.69 (range 18.0–29.7) while the non-smokers had a mean BMI and range at 24.1 (SD = 3.12) and 20.42 (range 16.5–36.0). All values indicate that the groups were similarly distributed demographically with slight deviation between the ages and the BMIs.

Smoking Status	Parameter	n	Range	Minimum	Maximum	Mean	Standard Deviation
Non-smoker	Age	40	35	20	55	31.7	9.25
	BMI	40	20.42	16.5	36	24.1	3.12
Smoker	Age	60	29	21	50	32.8	8.2
	BMI	60	11.69	18	29.7	25.2	2.97

Table 2 presents the comparison of oxygen saturation, total count of leukocytes (TLC), and differential leukocyte counts (DLC) between smokers and nonsmokers. Smokers had a lesser mean oxygen saturation (SpO<sub>2</sub>) at 98.2% compared to 98.7% among nonsmokers, which was statistically significant (p = 0.04). The count of granulocytes was more among smokers (0.589) than nonsmokers (0.575), with a p-value 0.01, which reflects increased inflammatory activity. As was the case for count of monocytes, which was more among smokers (0.056) than

nonsmokers (0.053), it was also significant (p = 0.02). On the contrary, lymphocytes count was less among smokers (0.366) than nonsmokers (0.371), but the p-value was high (<0.001). Total leukocyte count was also increased among smokers (7.36 × 10<sup>3</sup>/μL) relative to nonsmokers (6.95 × 10<sup>3</sup>/μL), and statistical significance was high (p < 0.001). The findings indicate the effects of smoking on immune cells distribution and oxygen saturation among healthy individuals too.

Parameter	Smokers (n=60)	Non-smokers (n=40)	P-value
SpO <sub>2</sub> (%)	98.2	98.7	0.04
Granulocyte count	0.589	0.575	0.01
Monocyte count	0.056	0.053	0.02
Lymphocyte count	0.366	0.371	<0.001
TLC (×10 <sup>3</sup> /μL)	7.36	6.95	<0.001



**Figure 1: Total leukocyte count (10<sup>3</sup>/mm<sup>3</sup>) in non-smokers and smokers**

### Discussion

The baseline demographic comparison carried out during the present study showed no notable difference between the distribution of smokers ( $32.8 \pm 8.2$  years) and non-smokers ( $31.7 \pm 9.25$  years), which corroborates data from similar population studies. For example, Lakshmi (2018) reported a similar mean age of  $34.5 \pm 7.8$  years for smokers and  $33.2 \pm 8.1$  years for non-smokers among a South Indian population, confirming the same homogeneity between smoking status and age for this region [14]. A study by Hasan et al., (2018) for the West Bengal region also reported similar mean ages for smokers and non-smokers as  $31.2 \pm 9.5$  years and  $30.8 \pm 8.9$  years, respectively, further attesting to the same findings [15]. The mild increase in BMI for smokers ( $25.2 \pm 2.97$ ) relative to non-smokers ( $24.1 \pm 3.12$ ) corroborates different and contrary data globally, which correlate smoking to lesser BMI (Chiolerio et al., 2008) [16], although West Bengal regional lifestyles and socioeconomic patterns may modulate this trend. For instance, research on rural Indian populations by Hasan et al. (2020) reported smokers to have a slightly higher BMI, which can be attributed to varying eating habits and forms of tobacco consumption [17].

The increased total leukocyte count (TLC) between smokers ( $7.36 \times 10^3/\mu\text{L}$ ) and non-smokers ( $6.95 \times 10^3/\mu\text{L}$ ,  $p < 0.001$ ) attests to an established pro-inflammatory condition due to chronic tobacco use. This systemic leukocytosis is consistent with observations by Mohamed et al. (2023), who found a mean TLC value for smokers to be  $7.2 \times 10^3/\mu\text{L}$  and for non-smokers to be  $6.7 \times 10^3/\mu\text{L}$ , attributing the rise to chronic inflammatory stimulation by tobacco poisons [18]. The increased counts for the granulocytes ( $0.589$  vs.  $0.575$ ,  $p = 0.01$ ) and for the monocytes ( $0.056$  vs.  $0.053$ ,  $p = 0.02$ ) for smokers add to evidence for engagement of the innate immune

mechanisms, which aligns with the findings by Goel et al. (2020) for an Indian population, for the percentage of granulocytes being significantly higher among smokers (59.3%) than among non-smokers (56.8%) [19].

In comparison, the modest but significant decrement in lymphocyte count for smokers ( $0.366$  vs.  $0.371$ ,  $p < 0.001$ ) indicates immunomodulation through adaptive immunity, supported by findings by Dahdah et al. (2022) who observed lymphopenia among chronic smokers, which may be attributed to defective immune surveillance [20]. For oxygen saturation, the modest reduction for smokers (98.2% vs. 98.7%,  $p = 0.04$ ) agrees with findings by Helal (2014), who reported decreased arterial oxygenation for chronic smokers despite normal clinical values, emphasizing early pulmonary functional impairment [21]. Together, the hematological and physiological changes indicate the subtle but significant systemic effects of smoking in West Bengal, consistent with trends seen globally but modulated by regional environmental and population considerations.

Overall, the findings indicate the systemic inflammatory and physiological changes that are linked to chronic smoking, even among healthy subjects. The increased leukocyte counts, and reduced oxygen saturation are early markers for underlying pathophysiological stress that may predispose smokers for worse cardiovascular and pulmonary outcomes in the long term.

### Conclusion

This research concluded that smoking has a quantifiable effect on hematological indices and oxygen saturation among healthy subjects. Smokers showed an increased total leukocyte count and number of granulocytes and an increased, though, to a lesser extent, number of monocytes, when compared to

non-smokers. On the contrary, lymphocyte count and oxygenation capacity of hemoglobin were observed to decrease among smokers. These findings indicate that, independent of frank disease, smoking causes system-wide changes, possibly as an indicator for an underlying inflammatory state and inefficient oxygen carriage. This study emphasizes the role of early screening and public health promotion to counteract the subclinical actions of smoking on immune function and respiratory health.

## References

1. Bashir BA, Gibreel MO, Abdalatif HM, Mohamed MA, Ahmed EA, Mohamed MS, Hamid KA. Impact of tobacco cigarette smoking on hematologic parameters among male subjects in port Sudan Ahlia College, Sudan. *Sch J Appl Med Sci* 2016; 4:1124-8.
2. Malenica M, Prnjavorac B, Bego T, Dujic T, Semiz S, Skrbo S, et al. Effect of cigarette smoking on haematological parameters in healthy population. *Med Arch* 2017; 71:132-36.
3. Salih SI. Studying the effect of smoking on some blood parameters in young adult male smokers. *Karbala J Med* 2015; 8:2287-91
4. Anandhalakshmi S, Kalaivani A, Shivasekar G, Saravanan A. Evaluation of the impact of cigarette smoking on platelet parameters. *Natl J Physiol Pharm Pharmacol* 2015; 5:426-430
5. Jena SK, Purohit KC, Misra AK. Effect of chronic smoking on hematological parameters. *Int J Curr Res* 2013; 5:279-82
6. Ozdal M, Pancar Z, Çinar V, Bilgic M. Effect of smoking on oxygen saturation in healthy sedentary men and women. *EC Pulmonol Respir Med* 2017; 4:178-82.
7. Madjid M, Awan I, Willerson JT, Casscells SW. Leukocyte count and coronary heart disease: implications for risk assessment. *Journal of the American College of Cardiology*. 2004 Nov 16;44(10):1945-56.
8. SHENWAI MR, Aundhakar NV. Effect of cigarette smoking on various hematological parameters in young male smokers. *Indian journal of basic & applied medical research*. 2012;2(5).
9. Benowitz NL. Nicotine addiction. *Primary Care: Clinics in Office Practice*. 1999 Sep 1;26(3):611-31.
10. West JB. *Respiratory physiology: the essentials*. Lippincott Williams & Wilkins; 2012.
11. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. *Journal of the American college of cardiology*. 2004 May 19; 43(10): 1731-7.
12. Smith KR, Mehta S, Maeusezahl-Feuz M. Indoor air pollution from household use of solid fuels. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. 2004; 2:1435-93.
13. Bano R, Ahmad N, Mahagaonkar AM, Latti RG. Study of lung functions in smokers and non-smokers in rural India. *Indian J Physiol Pharmacol*. 2011 Jan 1;55(1):84-8.
14. Lakshmi VS. Comparative study of hematological profile among smokers and non-smokers in rural part of South India. *IAIM*. 2018 Sep 1; 5:34-8.
15. Hasan AR, Poddar CK, Prasad BK, Singh MN. Comparative study of pulmonary function tests among smokers and non-smokers in a tertiary care hospital in Koshi region (Northern Bihar), India. *J Evolution Med Dent Sci*. 2018 Mar 12;7(11):1341-6.
16. Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *The American journal of clinical nutrition*. 2008 Apr 1;87(4):801-9.
17. Hasan MZ, Cohen JE, Bishai D, Kennedy CE, Rao KD, Ahuja A, Gupta S. Social capital and peer influence of tobacco consumption: a cross-sectional study among household heads in rural Uttar Pradesh, India. *BMJ open*. 2020 Jun 1;10(6):e037202.
18. Mohamed SE, Eid HA, Moazen EM Abd El-Fattah DA. Impact of chronic cigarette smoking on blood count indices, erythrocyte sedimentation rate and C-reactive protein as inflammatory markers in healthy individuals. *Journal of Recent Advances in Medicine*. 2023 Jan 1;4(1):74-85.
19. Goel A, Gupta P, Deepak D, Pandey HS, Moinuddin A. Total and differential leukocyte count and oxygen saturation of hemoglobin changes in healthy smokers and non-smokers. *National Journal of Physiology, Pharmacy and Pharmacology*. 2020 Aug 31;10(9):726-.
20. Dahdah A, Jagers RM, Sreejit G, Johnson J, Kanuri B, Murphy AJ, Nagareddy PR. Immunological insights into cigarette smoking-induced cardiovascular disease risk. *Cells*. 2022 Oct 11;11(20):3190.
21. Helal OF. Impact of smoking on adults lung age and ventilatory function. *Int J Physiother Res*. 2014;2(2):453-59.