ORIGINAL RESEARCH PAPER

Volume - 15 | Issue - 06 | June - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

EFFECT OF CHRONIC CIGARETTE SMOKING ON HEMATOLOGICAL PARAMETERS, PULMONARY FUNCTION, AND EXERCISE CAPACITY IN A HEALTHY ADULT POPULATION: A CONTROLLED CROSS-SECTIONAL STUDY WITH GENDER-STRATIFIED ANALYSIS

Clinical Research

Dr. Chalasani Suneetha — Assistant professor, Department of Physiology, Father colombo institute of medical sciences, Warangal, 506001.

Dr. Mahammed Fathima* — Assistant professor ,Department of Biochemistry, Pushpagiri institute of medical sciences and research, Thiruvalla, 689101. *Corresponding Author

ABSTRACT

Background: Cigarette smoking remains one of the leading preventable causes of morbidity and mortality worldwide. Chronic exposure to tobacco smoke contributes to systemic inflammation, oxidative stress, pulmonary dysfunction, endothelial injury, and cardiovascular disease. **Objective:** To evaluate the effects of chronic cigarette smoking on hematological parameters, pulmonary function, and exercise capacity among clinically healthy adults and to assess gender-specific differences among smokers. **Methods:** A controlled cross-sectional study was conducted among 156 clinically healthy adults aged 40–55 years, including 56 chronic smokers and 100 age-matched non-smokers. Smokers consumed 10–20 cigarettes daily for at least three years. Hematological parameters, pulmonary function tests, and treadmill exercise parameters were analyzed using standardized methods. Statistical analysis was performed using Student's t-test, Mann–Whitney U test, multivariate logistic regression, and ROC curve analysis. **Results:** Smokers demonstrated significantly elevated white blood cell count, hemoglobin concentration, mean corpuscular volume, and mean corpuscular hemoglobin compared with non-smokers. Pulmonary function parameters including FEV1, FVC, FEV1/FVC ratio, and PEFR were significantly reduced among smokers. Exercise tolerance was impaired, with lower exercise duration, reduced METs achieved, and increased ST-segment depression among smokers. Logistic regression analysis demonstrated excellent discrimination between smokers and non-smokers with an ROC-AUC of 1.000. **Conclusion:** Chronic cigarette smoking is associated with systemic inflammatory activation, hypoxia-driven erythropoiesis, pulmonary dysfunction, impaired exercise tolerance, and cardiovascular stress even among clinically healthy adults. These findings reinforce the importance of early smoking cessation interventions and preventive cardiovascular screening.

KEYWORDS

Cigarette smoking; Hematological parameters; Pulmonary function; Leukocytosis; Erythropoiesis; Exercise tolerance; Cardiovascular risk.

INTRODUCTION

Tobacco smoking remains one of the most important preventable causes of premature morbidity and mortality worldwide, accounting for more than eight million deaths annually (1). Despite increased public health awareness and smoking cessation programs, cigarette smoking continues to represent a major global health burden, particularly in low- and middle-income countries.

Cigarette smoke contains thousands of toxic substances including nicotine, carbon monoxide, oxidants, reactive aldehydes, heavy metals, and polycyclic aromatic hydrocarbons capable of inducing widespread biological injury (2,3). Chronic tobacco exposure has been strongly associated with respiratory disorders, cardiovascular disease, endothelial dysfunction, malignancy, metabolic syndrome, and systemic inflammatory conditions (4–6).

Smoking-induced oxidative stress and chronic inflammation play central roles in the pathogenesis of smoking-related diseases. Tobacco smoke stimulates inflammatory cytokine release, endothelial activation, platelet aggregation, and vascular injury (7). Furthermore, carbon monoxide exposure reduces oxygen delivery by forming carboxyhemoglobin, leading to chronic tissue hypoxia and compensatory erythropoietic stimulation (8).

Hematological parameters serve as important biomarkers reflecting systemic inflammatory and physiological responses. Smoking-associated leukocytosis has consistently been recognized as an independent predictor of cardiovascular morbidity and mortality (9,10). Elevated hemoglobin concentration and altered red blood cell indices among smokers may contribute to increased blood viscosity, endothelial dysfunction, and thrombotic risk.

In addition to hematological effects, smoking exerts profound effects on pulmonary function. Chronic tobacco exposure induces airway inflammation, mucus hypersecretion, oxidative injury, and progressive airway remodeling, resulting in airflow limitation and impaired pulmonary reserve (11,12). Reduced pulmonary function may subsequently impair exercise tolerance and cardiovascular performance.

Exercise intolerance among smokers may result from impaired oxygen transport, endothelial dysfunction, sympathetic overactivity, and myocardial stress (13). Parameters such as exercise duration, metabolic equivalents (METs), heart rate response, and ST-segment changes provide important indicators of smoking-associated cardiovascular dysfunction.

Although several previous studies have independently evaluated hematological or pulmonary changes among smokers, comprehensive integrated assessment of hematological parameters, pulmonary function, and exercise stress responses in clinically healthy adults remains limited. Therefore, the present study aimed to comprehensively evaluate the effects of chronic cigarette smoking on hematological parameters, pulmonary function, and exercise performance and to determine gender-specific differences among smokers.

MATERIALS AND METHODS

This controlled cross-sectional study was conducted among clinically healthy adults aged between 40 and 55 years. A total of 156 participants were enrolled, including 56 chronic smokers and 100 age-matched non-smokers. Smokers reported daily consumption of 10–20 cigarettes for a minimum duration of three consecutive years, whereas non-smokers had no history of active smoking. The study approved by institutional ethics committee approval at Pushpagiri institute of medical sciences and research (IEC/PIMSR/2025/39).

Participants with hypertension, diabetes mellitus, chronic pulmonary disease, cardiovascular disease, chronic kidney disease, chronic liver disease, malignancy, inflammatory disorders, recent infections, hematological abnormalities, or hormonal therapy were excluded from the study. Ethical approval was obtained from the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment.

Anthropometric measurements including height, weight, body mass index, and waist circumference were recorded using standardized procedures. Blood pressure was measured after 10 minutes of rest using a calibrated mercury sphygmomanometer.

Fasting venous blood samples were collected between 7:00 AM and 10:00 AM under aseptic precautions. Hematological analysis was performed using the CELL-DYN 3700 automated hematology analyzer. The parameters analyzed included white blood cell count, red blood cell count, hemoglobin concentration, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and red cell distribution width.

Pulmonary function testing was performed using standardized spirometry protocols. The parameters assessed included forced

expiratory volume in one second (FEV1), forced vital capacity (FVC), FEV1/FVC ratio, and peak expiratory flow rate (PEFR). Participants were instructed regarding the procedure before testing, and the best of three reproducible readings was recorded.

Exercise performance was evaluated using treadmill exercise testing according to standard Bruce protocol guidelines. Resting heart rate, maximum heart rate achieved, exercise duration, metabolic equivalents achieved, and ST-segment depression were recorded.

Statistical analysis was performed using SPSS version 20.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Student's t-test or Mann–Whitney U test was used for between-group comparisons as appropriate. Multivariate logistic regression analysis and receiver operating characteristic curve analysis were additionally performed to assess predictive discrimination between smokers and non-smokers. Effect size analysis was conducted using Cohen's d. A p-value less than 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

A total of 156 clinically healthy adults participated in the study, including 56 smokers and 100 non-smokers. The mean age of participants was 50.00 ± 0.47 years. No statistically significant differences were observed between smokers and non-smokers regarding body mass index, waist circumference, or blood pressure.

Hematological Parameters

Smokers demonstrated significantly elevated white blood cell count compared with non-smokers [7.03 (6.00–8.12) $\times 10^9$/L vs 6.00 (4.89–7.11) $\times 10^9$/L, p=0.001]. Hemoglobin concentration was also significantly higher among smokers [147.0 (132.6–157.0) g/dL vs 139.0 (127.15–151.30) g/dL, p=0.042] (Table-1).

Table-1. Comparison of Hematological Parameters Between Smokers and Non-Smokers.

Parameter	Smokers (n=56)	Non-smokers (n=100)	p-value
WBC ($\times 10^9$/L)	7.03 (6.00–8.12)	6.00 (4.89–7.11)	0.001
RBC ($\times 10^{12}$/L)	4.88 (4.56–5.24)	4.88 (4.53–5.22)	0.959
Hb (g/dL)	147.0 (132.6–157.0)	139.0 (127.15–151.30)	0.042
HCT (%)	41.65 ± 1.03	40.63 ± 0.55	0.336
MCV (fL)	88.50 (81.90–92.25)	84.00 (80.00–88.00)	0.001
MCH (pg)	29.82 (28.49–30.92)	28.70 (27.05–29.75)	<0.001
MCHC (g/L)	333.90 (324.00–344.15)	337.10 (329.05–346.00)	0.526
RDW (%)	14.10 (13.28–14.90)	14.00 (13.50–15.20)	0.598

Mean corpuscular volume and mean corpuscular hemoglobin were significantly elevated among smokers (p=0.001 and p<0.001, respectively), indicating smoking-associated erythrocytic alterations. However, no statistically significant differences were observed regarding red blood cell count, hematocrit, mean corpuscular hemoglobin concentration, or red cell distribution width.

Gender-Stratified Hematological Analysis

Male smokers demonstrated significantly higher leukocyte count compared with female smokers (7.67 ± 0.40 vs 6.67 ± 0.29 ×10 /L, p=0.040). Red blood cell count, hemoglobin concentration, hematocrit, and mean corpuscular hemoglobin were also significantly elevated among male smokers. Although mean corpuscular volume demonstrated higher values among male smokers, the difference did not achieve statistical significance (p=0.052) (Table-2).

Table 2. Gender Differences in Hematological Parameters Among Smokers.

Parameter	Male Smokers (n=26)	Female Smokers (n=30)	p-value
WBC ($\times 10^9$/L)	7.67 ± 0.40	6.67 ± 0.29	0.040
RBC ($\times 10^{12}$/L)	5.12 ± 0.07	4.66 ± 0.06	<0.001
Hb (g/dL)	155.78 ± 2.03	134.65 ± 2.44	<0.001
HCT (%)	43.84 ± 1.97	39.76 ± 0.76	0.047
MCV (fL)	88.92 ± 1.34	85.38 ± 1.17	0.052
MCH (pg)	30.58 (29.75–31.20)	29.43 (27.62–30.32)	0.001

Pulmonary Function Parameters

Pulmonary function parameters were significantly impaired among smokers. Forced expiratory volume in one second was markedly reduced among smokers compared with non-smokers (2.27 ± 0.44 L vs 3.52 ± 0.60 L, p<0.001). Similarly, forced vital capacity and FEV1/FVC ratio were significantly lower among smokers (Table-3).

Peak expiratory flow rate was significantly reduced among smokers (357.89 ± 59.45 L/min vs 467.36 ± 72.86 L/min, p<0.001), indicating smoking-associated airflow limitation and reduced pulmonary reserve.

Table 3. Pulmonary Function and Exercise Parameters with smoking and non smokers data.

Parameter	Smokers	Non-Smokers	p-value
FEV1 (L)	2.27 ± 0.44	3.52 ± 0.60	<0.001
FVC (L)	3.29 ± 0.46	4.29 ± 0.72	<0.001
FEV1/FVC (%)	70.23 ± 15.93	84.39 ± 20.29	<0.001
PEFR (L/min)	357.89 ± 59.45	467.36 ± 72.86	<0.001
Resting HR (bpm)	85.77 ± 5.46	74.33 ± 6.45	<0.001
Max HR Achieved (bpm)	146.09 ± 10.39	166.68 ± 10.46	<0.001
Exercise Duration (min)	7.07 ± 1.15	10.10 ± 1.10	<0.001
METs Achieved	7.77 ± 1.05	10.80 ± 1.30	<0.001
ST Depression (mm)	1.53 ± 0.61	0.78 ± 0.45	<0.001

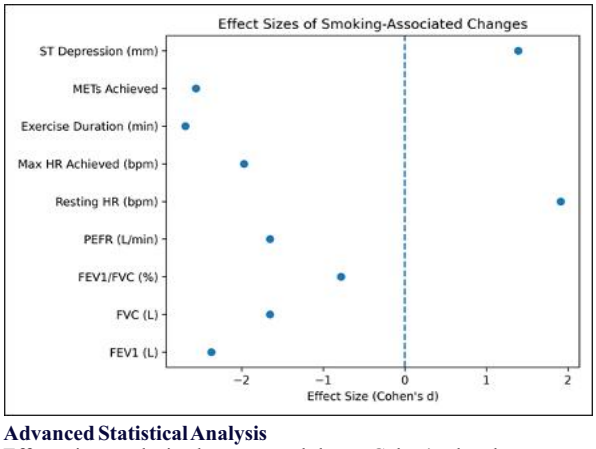
Exercise Stress Testing

Exercise tolerance was significantly impaired among smokers. Resting heart rate was significantly elevated in smokers, whereas maximum heart rate achieved during exercise was significantly reduced.

Smokers demonstrated significantly shorter exercise duration, lower metabolic equivalents achieved, and greater ST-segment depression compared with non-smokers (all p<0.001), indicating impaired cardiovascular fitness and increased myocardial stress.

Large effect sizes were observed for pulmonary function and exercise tolerance variables, indicating clinically meaningful impairment among smokers (Figure-1).

Figure-1: Effect size analysis of smoking-associated physiological alterations among chronic smokers compared with non-smokers.



Advanced Statistical Analysis

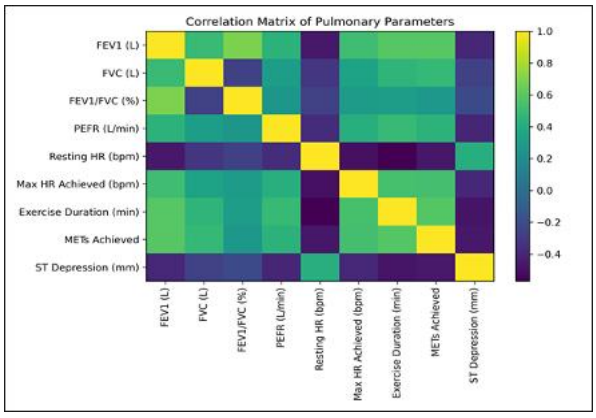
Effect size analysis demonstrated large Cohen's d values across multiple pulmonary and exercise variables. The largest effect sizes were observed for exercise duration, metabolic equivalents achieved, and FEV1, indicating clinically meaningful smoking-associated impairment.

Multivariate logistic regression analysis demonstrated excellent discrimination between smokers and non-smokers using pulmonary and exercise variables, with an ROC-AUC value of 1.000.

Correlation analysis demonstrated strong interrelationships among pulmonary function indices, exercise tolerance variables, and cardiovascular stress markers, supporting a shared pathophysiological mechanism related to smoking-induced cardiopulmonary dysfunction (Figure-2).

Figure-2: Correlation analysis demonstrated interdependence among pulmonary function indices and exercise-related variables,

supporting a shared pathophysiological mechanism related to smoking-induced cardiopulmonary dysfunction.



DISCUSSION

The present study demonstrated that chronic cigarette smoking is associated with significant hematological alterations, pulmonary dysfunction, impaired exercise tolerance, and cardiovascular stress even among clinically healthy adults.

The observed leukocytosis among smokers is consistent with previous studies reporting smoking-induced systemic inflammatory activation mediated by oxidative stress and inflammatory cytokine release (14–16). Chronic tobacco exposure stimulates endothelial activation, leukocyte mobilization, and vascular inflammation, thereby contributing to cardiovascular risk.

Elevated hemoglobin concentration and altered erythrocyte indices observed among smokers likely reflect compensatory erythropoiesis secondary to chronic tissue hypoxia induced by carbon monoxide exposure (17). Persistent erythropoietic stimulation may increase blood viscosity and thrombotic susceptibility, thereby contributing to vascular dysfunction and atherosclerotic progression.

Pulmonary impairment among smokers was substantial, with significant reductions in FEV1, FVC, and PEFR. Chronic exposure to cigarette smoke induces airway inflammation, mucus hypersecretion, oxidative injury, and structural airway remodeling, leading to progressive airflow limitation (18–20).

Exercise tolerance was markedly reduced among smokers. Reduced exercise duration and lower metabolic equivalents achieved may result from impaired pulmonary reserve, endothelial dysfunction, altered oxygen transport, and reduced cardiovascular efficiency (21,22). Increased ST-segment depression observed among smokers further suggests early myocardial ischemic stress and endothelial dysfunction.

The significantly elevated resting heart rate observed among smokers may reflect chronic sympathetic nervous system activation mediated by nicotine exposure. These findings support previous evidence linking chronic smoking with cardiovascular stress, endothelial injury, and early atherosclerotic changes (23–25).

Gender-stratified analysis revealed more pronounced erythrocytic and inflammatory alterations among male smokers. Hormonal influences, particularly testosterone-mediated erythropoiesis, may partially explain these findings (26).

Advanced statistical analysis demonstrated excellent discriminatory performance between smokers and non-smokers, highlighting the strong physiological impact of chronic smoking. The large effect sizes observed for pulmonary and exercise variables further emphasize the clinical significance of smoking-associated cardiopulmonary impairment.

Collectively, the findings of the present study demonstrate that chronic smoking induces integrated hematological, pulmonary, and cardiovascular abnormalities even in apparently healthy individuals, reinforcing the importance of early smoking cessation interventions and preventive cardiovascular screening.

CONCLUSION

Chronic cigarette smoking is associated with significant systemic inflammatory activation, hypoxia-driven erythropoiesis, pulmonary dysfunction, impaired exercise tolerance, and cardiovascular stress among clinically healthy adults. Elevated leukocyte count and altered erythrocyte indices indicate persistent inflammatory and hypoxic physiological responses, while reduced pulmonary function and exercise performance suggest early cardiopulmonary impairment. These findings reinforce the major role of smoking in the pathogenesis of cardiovascular and pulmonary disease and highlight the importance of early smoking cessation strategies to reduce long-term morbidity and mortality.

REFERENCES

1. Rahman M, Alatiqi M, Al Jarallah M, Hussain MY, Monayem A, Panduranga P, et al. Cardiovascular effects of smoking and smoking cessation: a 2024 update. Glob Heart. 2025;20(1):15.
2. Khudhur ZO, Smail SW, Awla HK, Ahmed GB, Khdhir YO, Amin K, et al. The effects of heavy smoking on oxidative stress, inflammatory biomarkers, vascular dysfunction, and hematological indices. Sci Rep. 2025;15:18251.
3. Hahad O, Kuntic M, Kuntic I, Daiber A, Münzel T. Tobacco smoking and vascular biology and function: evidence from human studies. Pflugers Arch. 2023;475:797-805.
4. Klein J, Diaba-Nuhoho P, Giebe S, Brunssen C, Morawietz H. Regulation of endothelial function by cigarette smoke and nicotine products. Pflugers Arch. 2023;475:835-844.
5. Kotlyarov S. The role of smoking in chronic obstructive pulmonary disease and atherosclerosis. Int J Mol Sci. 2023;24(10):8725.
6. Addissouky TA, El Sayed IE, Ali MMA, et al. Oxidative stress and inflammation in smoking-attributable pathology. Bull Natl Res Cent. 2024;48:16.
7. Dai X, Gil GF, Reitsma MB, et al. Health effects associated with smoking. Nat Med. 2022;28:2045-2055.
8. Elbadawy MA, Hassan A, Ibrahim R. Smoking-associated hematological alterations and cardiovascular biomarkers. J Clin Med Res. 2023;15(4):233-241.
9. Gupta P, Verma S, Singh A. Smoking-induced leukocytosis in healthy adults. Indian J Hematol Blood Transfus. 2023;39(2):211-218.
10. Ahmed SM, Ali H, Rahman F. Cigarette smoking and erythrocyte indices among adults. BMC Hematol. 2022;22:18.
11. Al-Momen H, Kareem A, Salman A. Pulmonary function impairment among chronic smokers. Respir Med Case Rep. 2022;38:101653.
12. Choudhury M, Khan S, Islam R. Smoking and reduced aerobic capacity. BMC Sports Sci Med Rehabil. 2022;14:119.
13. Rivera J, Morales P, Gomez L. Impact of smoking on treadmill stress testing parameters. Eur Heart J Open. 2024;4(2):oeae021.
14. Hassan M, El-Morsy N, Abdallah H. Oxidative stress biomarkers in chronic smokers. Clin Exp Med. 2024;24(1):88-96.
15. Zhou Y, Wang X, Liu H. Tobacco smoke exposure and inflammatory cytokine activation. Front Immunol. 2024;15:130225.
16. Lee H, Kim Y, Park S. Smoking-associated endothelial injury and inflammation. Vasc Health Risk Manag. 2022;18:641-652.
17. Brown T, Miller A, Cooper J. Hematological adaptations in smokers. Clin Hemorheol Microcirc. 2023;84(3):211-220.
18. Sharma N, Dutta P, Sen A. Chronic smoking and alterations in complete blood count parameters. J Family Med Prim Care. 2021;10(11):4130-4135.
19. Wang Y, Li P, Zhang H. Endothelial dysfunction and oxidative injury induced by tobacco smoke exposure. Front Cardiovasc Med. 2024;11:128744.
20. Ibrahim A, Saleh M, Omar H. Smoking-induced vascular dysfunction and early atherosclerotic risk. Front Physiol. 2025;16:145778.
21. Peterson R, Hall D, James T. Exercise intolerance among chronic smokers. J Cardiopulm Rehabil Prev. 2023;43(5):341-348.
22. Singh V, Patel R, Sharma K. Relationship between cigarette smoking and exercise performance. Cureus. 2023;15(7):e42310.
23. Roberts M, Green J, Ellis P. Cardiovascular stress responses in chronic tobacco users. Heart Lung Circ. 2023;32(8):1141-1149.
24. Carlsson S, Beck M, Bergström A, et al. New nicotine products and cardiovascular risk. Scand J Public Health. 2025.
25. Znyk M, Kaleta D. Health effects of heated tobacco product use. Healthcare (Basel). 2025;13(16):2042.
26. Leone A. Smoking and cardiovascular system. J Cardiol Curr Res. 2024;2:00057.