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Research Article

Hematological profiles and complete blood count alterations in smokers

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Abstract

Objective: The aim of this study was to evaluate the associations between cigarette smoking and hematological parameters in otherwise healthy individuals.

Methods: A total of 109 individuals were included in the study, comprising 49 active smokers and 60 never-smokers. Complete blood count parameters along with related hematological indices were retrospectively evaluated.

Results: A total of 109 participants (49 smokers, 60 non-smokers) were evaluated. In unadjusted analyses, smokers exhibited significantly higher white blood cell (WBC), neutrophil, lymphocyte, eosinophil, and hemoglobin levels ($p<0.05$). However, after adjusting for sex, only WBC ($p=0.002$) and lymphocyte ($p=0.033$) levels remained significantly higher in the smoker group. Correlation analyses showed that cumulative smoking exposure was positively associated with WBC, lymphocyte, and RDW levels ($p<0.05$). Multivariable linear regression further revealed that cumulative smoking exposure was an independent factor associated with higher leukocyte levels ($B=0.004$, $p=0.009$), whereas sex was the primary factor independently associated with red cell distribution width (RDW-SD) ($B=-2.555$, $p<0.001$). The inclusion of smoking exposure improved the adjusted R^2 for the WBC model from 0.015 to 0.113.

Conclusion: This study indicates that smoking is significantly associated with variations in hematological parameters and inflammatory markers. After adjusting for confounding factors such as sex, leukocyte and lymphocyte counts remained significantly elevated in smokers. Notably, cumulative smoking exposure was identified as an independent factor of higher leukocyte levels, increasing the adjusted R^2 of the model from 0.015 to 0.113. These findings suggest that chronic tobacco exposure may contribute to systemic inflammation and alterations in the leukocyte profile.

Keywords: Blood cell count, Hemoglobin, Smoking, Inflammation

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INTRODUCTION

Tobacco use is one of the most important global public health challenges and is associated with numerous causes of morbidity and mortality, particularly chronic obstructive pulmonary disease (COPD), cardiovascular diseases, and various malignancies (Gartner & Hall, 2024). Cigarette smoke contains thousands of toxic, mutagenic, and carcinogenic compounds that directly induce cellular damage. Inhalation of these substances triggers oxidative stress pathways at the cellular level, resulting in the development of persistent low-grade systemic inflammation (Khanna et al., 2013).

The hematopoietic system shows highly sensitive variations in relation to the chronic inflammatory processes and bone marrow stimulation induced by tobacco smoke exposure (Siggins et al., 2014). Previous studies have demonstrated that cigarette smoking is associated with a variety of hematological alterations, most notably leukocytosis (Malenica et al., 2017). However, the relationships between tobacco exposure and hematological/inflammatory markers-such as acute-phase reactants C-reactive protein (CRP) and complete blood count parameters such as white blood cell (WBC), neutrophil, lymphocyte, eosinophil, hemoglobin (Hb), red cell distribution width (RDW-SD), and plateletcrit (PCT) are complex and involve multiple underlying mechanisms (Sharafi et al., 2025). Furthermore, distinguishing the potential confounding effects of demographic factors such as age and sex on these parameters and determining the role of cumulative smoking exposure (pack-years) as an independent predictor are crucial for understanding the extent of tobacco-related systemic damage (Lee et al., 2025).

The present study aimed to evaluate the associations of active smoking and cumulative tobacco exposure on routine hematological parameters and inflammatory markers among healthy individuals attending an occupational health outpatient clinic. In addition, the study sought to investigate the influence of sex on these associations and to determine the independent relationship between cumulative smoking exposure and laboratory parameters using multivariable analytical models.

METHODS

This retrospective study was conducted between September 2025 and March 2026 and included a total of 109 volunteers who attended the Occupational Health Outpatient Clinic of xxx University Faculty of Medicine Hospital for routine health examinations or periodic medical surveillance. The study protocol was approved by the Ethics Committee of xxx University (Approval Date: 13.04.2026, Decision No: 11). Due to the retrospective nature of the study and the complete anonymization of patient data, the requirement for informed consent was officially waived by the ethics committee.

The inclusion criteria were: being between 18 and 65 years of age, having undergone periodic occupational health surveillance at the outpatient clinic during the study period, and having complete laboratory data available. Based on tobacco use status, participants were classified into two groups: active smokers,

defined as individuals who smoked at least one cigarette per day (n = 49), and never-smokers, defined as individuals who had never used cigarettes throughout their lifetime (n = 60). Following final database validation, the cohort sizes were confirmed as n = 49 for smokers and n = 60 for non-smokers (total N = 109), and all subsequent statistical analyses, including ANCOVA models, were executed using these verified cohort sizes.

The exclusion criteria included a history of acute or chronic infection, malignancy, autoimmune or chronic inflammatory systemic disease, hematological malignancy or disorders affecting bone marrow function, use of immunomodulatory medications (e.g., systemic corticosteroids) within the previous month, chronic medication use for major systemic diseases, regular or heavy alcohol consumption and pregnancy, all of which could directly influence hematological parameters and inflammatory processes.

Information regarding tobacco use status, type of exposure, and smoking characteristics, including the number of cigarettes smoked per day, duration of smoking, and cumulative exposure expressed as cumulative smoking exposure, was obtained from medical records. Specifically, these detailed smoking histories were extracted from the standardized medical anamnesis records documented by the examining physicians during face-to-face clinical interviews at the time of the outpatient clinic visit. Complete blood count parameters routinely assessed during occupational health surveillance, including WBC, neutrophil count, lymphocyte count, eosinophil count, Hb, platelet count (PLT), RDW, PCT, and immature granulocyte (IG) count, were retrospectively retrieved from the hospital electronic medical record system.

Statistical analyses

Statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA), and graphs were generated using GraphPad Prism version 10.4.1 (GraphPad Software, LLC, San Diego, CA, USA). Data distribution was assessed using the Kolmogorov–Smirnov test and visual inspection methods. Normally distributed continuous variables were presented as mean ± standard deviation, whereas non-normally distributed variables were expressed as median (25th–75th percentile). Categorical variables were presented as n (%).

Independent samples t-test was used for comparisons of normally distributed continuous variables, while Pearson's chi-square test was used for categorical variables according to expected cell frequencies. Since the proportion of females differed significantly between groups, sex was considered a potential confounding factor in the comparison of hematological parameters, and analysis of covariance (ANCOVA) was performed.

Spearman correlation analysis was used to evaluate the relationships between smoking-related variables and hematological parameters.

Multiple linear regression analyses were performed to determine the association of

smoking exposure with WBC and RDW levels, as these parameters showed significant unadjusted correlations with cumulative smoking exposure. Because lymphocyte count is a component of the total WBC count and was moderately to strongly correlated with WBC, WBC was selected as the representative leukocyte parameter to avoid redundancy in the regression model. Two regression models were constructed: Model 1 included age and sex, whereas Model 2 additionally included cumulative smoking exposure. Assumptions of linear regression analyses, including linearity, normality of residuals, homoscedasticity, independence of errors, and absence of multicollinearity, were evaluated and confirmed before analysis. A two-tailed p value < 0.05 was considered statistically significant.

RESULTS

A total of 109 participants were included in the study, consisting of 49 active smokers and 60 non-smokers. As shown in **Table 1**, the proportion of females was significantly higher in the non-smoker group compared to the smoker group (p = 0.020). No significant between-group differences were observed in age. Among smokers, the median number of cigarettes smoked per day was 10.0 (7.0–20.0), median smoking duration was 10.0 (5.0–13.8) years, and median cumulative smoking exposure was 100.0 (42.0–195.0).

As presented in **Table 2**, smokers had significantly higher WBC ($p < 0.001$), neutrophil ($p = 0.044$) lymphocyte ($p = 0.007$), eosinophil ($p < 0.001$), and Hb ($p = 0.004$) levels, whereas PCT ($p = 0.042$) levels were significantly lower

Table 1. Comparison of demographic characteristics, comorbidities, and smoking-related variables between active smokers and non-smokers

	Active smokers N=49	Non-smokers N=60	Effect size	p-value
Age, years[a]	32.0±6.3	31.7±7.7	NA	0.807
Sex, F[b]	16 (32.7)	33 (55.0)	0.220	**0.020**
Comorbidity HT DM Asthma COPD CAD Other	None	None	NA	NA
Cigarettes/day[c]	10.0 (7.0 ,20.0)	NA	NA	NA
Smoking duration,years[c]	10.0 (5.0 ,13.8)	NA	NA	NA
Exposure* [c]	100.0 (42.0 ,195.0)	NA	NA	NA

(*): Cigarettes/day x Smoking duration/years, a: mean ± standard deviation, b: n (%), c: median (25% - 75%)
Abbreviations: F, Female; HT, Hypertension; DM, Diabetes Mellitus; COPD, Chronic Obstructive Pulmonary Disease; CAD, Coronary Artery Disease

compared to non-smokers in the unadjusted analyses. However, after adjustment for sex, only WBC (p = 0.002, partial η^2= 0.09, moderate effect size) and lymphocyte levels (p = 0.033, partial eta squared = 0.04, low-effect size) remained significantly higher in smokers. No significant between-group differences were observed for neutrophil, RDW, HB, PLT, and IG levels after sex adjustment.

As shown in **Table 3**, cigarettes/day showed significant positive correlations with WBC (rho = 0.493, p < 0.001) and neutrophil levels (rho = 0.309, p = 0.033). Smoking duration was positively correlated with Hb levels (rho = 0.297, p = 0.042). In addition, cumulative smoking exposure was significantly correlated with WBC (rho = 0.391, p = 0.007), lymphocyte (rho = 0.311, p = 0.033), and RDW levels (rho = 0.315, p = 0.031). No other significant correlations were observed between smoking-related variables and hematological parameters.

Table 2. Comparison of hematological parameters between active smokers and non-smokers

	Active smokers N=49	Non-smokers N= 60	Effect size (Partial Eta Squared)	p-value[c]	p-value (adjusted for sex)[d]
WBC (×10³/µL)[a]	8.55 ± 1.84	7.34 ± 1.71	0.090	<0.001	0.002
Neutrophil (×10³/µL)[a]	4.82 ± 1.59	4.27 ± 1.49	0.030	0.044	0.064
Lymphocyte (×10³/µL)[a]	2.74 ± 0.74	2.35 ± 0.74	0.040	0.007	0.033
Eosinophil (×10³/µL)[b]	0.22 (0.13, 0.36)	0.13 (0.07 – 0.24)	0.030	<0.001	0.093
PCT (%)[a]	0.25 ± 0.05	0.27 ± 0.05	0.030	0.042	0.096
RDW-SD (fL)[a]	40.96 ± 2.59	41.16 ± 3.10	0.000	0.724	0.569
HB (g/dL)[a]	14.98 ± 1.59	14.09 ± 1.54	0.030	0.004	0.081
PLT (×10³/µL)[a]	253.63 ± 45.60	268.93 ± 54.11	0.020	0.118	0.197
IG (%)[b]	0.02 (0.01, 0.03)	0.02 (0.01,0.03)	0.020	0.138	0.221

a: mean ± standard deviation, b: median (25% - 75%), (c): Independent samples t test or Mann Whitney – U test, (d): ANCOVA
Abbreviations: SD, standard deviation; WBC, white blood cell count; PCT, plateletcrit; RDW, red cell distribution width; HB, hemoglobin; PLT, platelet count; IG, immature granulocyte

Table 3. Correlations between smoking exposure variables and hematological parameters in active smokers

	Cigarettes/day			Smoking duration			Exposure		
	rho	p-value	BH-FDR adjusted p-value	rho	p-value	BH-FDR adjusted p-value	rho	p-value	BH-FDR adjusted p-value
WBC	0.493	<0.001	0.009	-0.055	0.712	0.745	0.391	0.007	0.063
Neutrophil	0.309	0.033	0.149	-0.241	0.102	0.459	0.095	0.527	0.716
Lymphocyte	0.158	0.283	0.637	0.125	0.401	0.745	0.311	0.033	0.149
Eosinophil	0.129	0.381	0.668	0.178	0.231	0.693	0.281	0.056	0.168
PCT	0.113	0.445	0.668	-0.049	0.745	0.745	0.088	0.557	0.716
RDW	0.251	0.085	0.255	0.078	0.604	0.745	0.315	0.031	0.149
HB	0.051	0.728	0.728	0.297	0.042	0.378	0.199	0.180	0.405
PLT	0.067	0.651	0.728	-0.096	0.519	0.745	-0.004	0.978	0.978
IG	0.079	0.591	0.728	0.088	0.557	0.745	0.175	0.240	0.432

Abbreviations: WBC, white blood cell count; PCT, plateletcrit; RDW, red cell distribution width; HB, hemoglobin; PLT, platelet count; IG, immature granulocyte; BH-FDR, Benjamini–Hochberg False Discovery Rate.

After applying the Benjamini–Hochberg false discovery rate (BH-FDR) correction, only the positive correlation between cigarettes/day and WBC remained statistically significant (rho = 0.009). None of the other correlations remained significant after correction for multiple comparisons (all rho > 0.05).

As presented in **Table 4**, multivariable linear regression analyses were performed to determine the independent associations of age, sex, and cumulative smoking exposure with WBC and RDW levels in active smokers. In Model 1, which included only age and sex, neither age nor sex was significantly associated with WBC levels. However, sex was independently associated with RDW levels (B = -2.278, p = 0.003). In Model 2, after adding cumulative smoking exposure to the model, smoking exposure was found to be independently associated with higher WBC levels (B = 0.004, 95% CI: 0.001 to 0.008, p = 0.009), whereas age and sex remained non-significant . For RDW,

Table 4. Multiple linear regression analysis of factors associated with WBC and RDW levels in active smokers adjusted for age and sex

	WBC		RDW	
Model 1	B (95% CI)	p-value	B (95% CI)	p-value
Age	-0.045 (-0.134 to 0.044)	0.318	0.179 (0.071 to 0.288)	0.002
Sex*	0.457 (-0.731 to 1.646)	0.442	-2.278 (-3.726 to -0.830)	0.003
Adjusted R^2	0.015		0.242	
Model 2	B (95% CI)	p-value	B (95% CI)	p-value
Age	-0.060 (-0.144 to 0.024)	0.159	0.167 (0.060 to 0.274)	**0.003**
Sex*	0.115 (-1.025 to 1.255)	0.839	-2.555 (-4.009 to -1.102)	**<0.001**
Exposure	0.004 (0.001 to 0.008)	**0.009**	0.004 (-0.001 to 0.008)	0.089
Adjusted R^2	0.113		0.275	

(*): Sex was coded as 1 = female and 2 = male.
Abbreviations: B, Unstandardized Coefficients; WBC, white blood cell count; RDW, red cell distribution width

sex remained a significant independent factor (B = -2.555, $p < 0.001$), while cumulative smoking exposure was not independently associated with RDW levels ($p = 0.089$). The adjusted R^2 value increased from 0.015 to 0.113 for WBC and from 0.242 to 0.275 for RDW after inclusion of cumulative smoking exposure in the models. All predictors showed acceptable collinearity, with tolerance values ranging from 0.897 to 0.926 and VIF values ranging from 1.080 to 1.115, indicating no evidence of multicollinearity.

DISCUSSION

In the present study, hematological parameters were compared between healthy active smokers and healthy non-smoking controls, and the independent effects of tobacco smoke exposure on leukocyte profiles and erythrocyte indices were investigated using multivariate analyses. The principal finding of our study was that, in unadjusted analyses, smokers exhibited significantly higher WBC, neutrophil, lymphocyte, eosinophil, and Hb levels, whereas PCT levels were significantly lower. However, after controlling for sex—a factor frequently overlooked in the literature despite its substantial influence on hematological parameters-via ANCOVA, the independent effect of smoking on WBC and lymphocyte counts remained significant, whereas the significance of differences observed in other parameters disappeared. These findings highlight the importance of considering sex distribution as a potential confounding factor in studies investigating the relationship between smoking and hematological indices (Campesi et al., 2020; Zhao et al., 2026).

Previous studies have consistently reported that cigarette smoking increases peripheral total WBC counts (Chee et al., 2025). Inhalation of tobacco smoke activates alveolar macrophages in the lungs, leading to the release of pro-inflammatory cytokines such as IL-1, IL-6, and TNF-α, which subsequently stimulate leukocyte mobilization from the bone marrow (Sehlikoğlu et al., 2024). Our findings are consistent with this pathophysiological mechanism, as WBC and lymphocyte levels remained significantly elevated among smokers even after adjustment for sex ($p = 0.002$ and $p = 0.033$, respectively).

Furthermore, unadjusted correlation analyses revealed a significant positive association between the number of cigarettes smoked per day and WBC count. In our multivariable linear regression model (Model 2), cumulative smoking exposure was independently associated with higher WBC levels after adjustment for age and sex. Notably, inclusion of cumulative smoking exposure substantially improved the model's explanatory power, increasing the adjusted R^2 value for WBC from 0.015 to 0.113 (representing an increase in explained variance from 1.5% to 11.3%). It should be noted that this dose-response linear regression analysis evaluating pack-years was conducted exclusively within the active smoking subgroup (n = 49), rather than the overall study population. Consistent with these findings, Smith et al. (2021) also demonstrated a significant association between tobacco use and asymptomatic leukocytosis, while Higuchi et al. (2016) highlighted the substantial impact of smoking status on mathematical models of leukocyte variance. These results further suggest the association between increasing chronic tobacco exposure and greater degrees of subclinical systemic inflammation.

Although neutrophil, eosinophil, and Hb levels were significantly higher in smokers in unadjusted analyses, these differences lost statistical significance after adjustment for sex. Cigarette smoking is known to induce tissue hypoxia, stimulating erythropoietin production and thereby increasing hemoglobin levels. However, our findings suggest that the observed difference in Hb levels was largely attributable to sex-related variation, as males physiologically exhibit higher Hb concentrations and were overrepresented in the smoking group (Beleslin-Cokic et al., 2004). Nevertheless, the positive correlation observed in unadjusted analyses between smoking duration and Hb levels (rho = 0.297, p = 0.042) suggests that prolonged exposure to tobacco smoke may be linked to a hypoxia-driven stimulatory pattern in erythrocyte mass. However, because the between-group difference in Hb disappeared after sex adjustment and the correlation was no longer significant after BH-FDR correction, this finding should be interpreted with caution.

RDW has recently gained attention not only as a marker for differential diagnosis of anemia but also as a biomarker of chronic inflammation and cardiovascular risk (Marzouk et al., 2020). In our study, no significant difference in RDW was observed between smokers and non-smokers. However, among smokers, cumulative smoking exposure demonstrated a significant positive correlation in unadjusted analysis with RDW (rho = 0.315, p = 0.031). Further investigation using multivariable regression analysis yielded an interesting finding: although cumulative smoking exposure showed a borderline association with RDW after adjustment (p = 0.089), sex emerged as the strongest and most significant independent factor related to RDW-SD ($B = -2.555$, $p < 0.001$). These results suggest that the potential adverse effects of smoking on erythropoiesis, such as inflammation-induced anisocytosis, may be overshadowed by physiological variations associated with biological sex (Erik et al., 2025).

Regarding platelet indices, unadjusted analyses demonstrated significantly lower PCT levels among smokers (p = 0.042). PCT reflects total platelet mass as the proportion of platelet volume within the total blood volume. Similar to the findings observed for leukocyte-related parameters, the significance of this difference disappeared after adjustment for sex (p = 0.096) (Yuan et al., 2026). Furthermore, no significant differences were identified in PLT or IG-percentages between groups, nor were any significant correlations observed with smoking exposure variables. These findings suggest that, within the study population, the systemic inflammatory response associated with smoking is primarily mediated through lymphoid and myeloid leukocyte pathways, whereas the megakaryocytic lineage may not yet be substantially involved in the early subclinical inflammatory process (El-Mahdy et al., 2020).

One of the major strengths of our study is the rigorous methodological control of key confounding variables, particularly age and sex, through both ANCOVA and multivariable linear regression analyses. This approach helps clarify some of the inconsistencies reported in previous studies. Nevertheless, several limitations should be acknowledged. First, the study was conducted at a single center with a relatively small sample size (n = 109), and the study population consisted of relatively young, healthy individuals attending an occupational health outpatient clinic, which may limit the generalizability of our findings to broader or clinical populations. Second, a key limitation of our study is the potential for residual confounding due to unmeasured lifestyle and physiological variables. Specifically, parameters such as body mass index (BMI), dietary habits, alcohol consumption, and physical activity levels were unavailable for adjustment. BMI and nutritional status are well-established modulators of low-grade systemic inflammation and leukocyte counts, while physical activity and chronic alcohol exposure can independently influence subclinical inflammatory markers and hematological profiles. The inability to control for these potential confounders means that residual confounding cannot be completely ruled out, and part of the observed associations could be influenced by unmeasured baseline differences between smokers and non-smokers. Third, the correlation and dose-response regression analyses evaluating smoking exposure parameters were performed exclusively within the subgroup of active smokers (n = 49), rather than the general cohort, and therefore should be considered exploratory. Fourth, major acute-phase reactants such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) could not be comprehensively analyzed due to insufficient data availability, limiting our ability to establish the direct relationship between humoral inflammatory responses and cellular inflammatory markers such as WBC and lymphocyte counts (Elisia et al., 2020). Future prospective studies with larger sample sizes and comprehensive adjustment for lifestyle confounders are warranted to validate these findings.

CONCLUSION

This study suggests that chronic tobacco use is associated with alterations in hematological parameters and markers of systemic inflammation. Even after controlling for potential confounding factors such as sex, WBC and lymphocyte counts remained significantly higher in smokers, aligning with the strong associations between cigarette smoking on immune function and inflammatory processes. While sex emerged as the primary factor independently associated with RDW-SD in the overall cohort, cumulative smoking exposure (pack-years) was identified as a strong independent factor associated with elevated WBC levels strictly within the active smoking subgroup (n = 49), increasing the proportion of explained variance for leukocyte profiles from 1.5% to 11.3%.

In clinical practice, smoking history and cumulative tobacco exposure should be carefully considered when interpreting complete blood count results in smokers. The physiological and pathological tendency toward increased leukocyte and lymphocyte counts may reflect chronic low-grade subclinical inflammation; failing to account for this exposure may otherwise lead to unnecessary diagnostic evaluations and investigations in otherwise healthy individuals.

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Conflict of Interest

The authors declare that they have no conflict of interests regarding content of this article.

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Ethical Declaration

This study was conducted with the decision of Kahramanmaraş Sütçü İmam University Faculty of Medicine Ethics Committee dated 13.04.2026 and numbered 2026/11. As this was a retrospective study, the requirement for written informed consent was waived by the committee. The study was conducted in accordance with the principles of the Declaration of Helsinki and all patient data were anonymized prior to analysis to ensure confidentiality

Author Contributions

Concept: ŞŞB,HŞ Design: ŞŞB,HŞ, Supervising: ŞŞB,HŞ, Financing and equipment: ŞŞB, HŞ, Data collection and entry:ŞŞB, HŞ, Analysis and interpretation:ŞŞB, HŞ, Literature search: ŞŞB,HŞ, Writing: ŞŞB,HŞ, Critical review: ŞŞB, HŞ

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