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Assessing the Influence of Smoking on Blood Components and Inflammation Factors in Humans

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ABSTRACT

There was an investigation done to observe whether smoking (cigarettes and/or arghile) would have an effect on the hemogram (or blood) and any correlation with blood type distribution. Out of 80 men in total, there were 40 smokers and 40 non-smokers in the control group. Data was collected about and analyzed to allow for comparisons between the groups in terms of age, years of smoking (duration), blood type, and assessed hematological parameters. The results indicated that there were more smokers aged 30-40 than young men aged 19 or younger in the control group. Men that smoked cigarettes predominantly had shorter smoking durations, while men that smoked arghile predominantly had longer smoking durations. There were no significant differences between smoking and blood type distribution (ABO and Rh). Some of the hematological parameters had statistically significant differences between smokers and non-smokers. Smokers had greater mean values for Hb, PCV, WBC counts, and ESR than non-smokers ($P < 0.05$). Meanwhile, there were no statistically significant differences noted in RBC counts and PLT. Generally, smoking appears to induce definite hematological changes in smokers (especially related to inflammation and oxygen transport) and may indirectly increase the risk of cardiovascular and inflammatory disease in smokers.

Keywords: Smoking, HB, PCV, Leukocyte Count, ESR.

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INTRODUCTION

When tobacco is burned, a large number of organic and inorganic compounds are created that make up tobacco smoke. It is estimated that over 4,000 chemicals exist in tobacco smoke, and at least 60 of them have been identified as carcinogens. Various others have also been proven to be associated with serious diseases leading to cardiovascular as well as respiratory illnesses [1]. Another important component of tobacco is nicotine, which creates a strong dependency and causes problems for people trying to quit. Once nicotine is inhaled, it is absorbed into the blood and quickly reaches the brain. Once in the brain, it activates certain neurotransmitters that are responsible for mood and behaviours.

Nicotine is a naturally produced chemical that is found in various plants, particularly within the nightshade family of plants, such as tobacco plants (Nicotiana tabacum) and Duboisia hopwoodii [1].

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In terms of pharmacology, nicotine predominantly behaves as an agonist on most of the nicotinic acetylcholine receptors (nAChR), but may also behave as an antagonist on certain subtypes of nAChR, including nAChR-9 and nAChR-10 [2–4]. Nicotine was first identified and isolated from tobacco in 1828 by the scientists Posselt and Reimann, and was named after Jean Nicot, who is credited with introducing tobacco to the French royal court in 1560 [5].

Hiccups, skin irritation, dizziness, nausea, gastrointestinal problems (e.g. diarrhea/constipation), sleep problems, heart palpitations or chest pain are some of the side effects that can occur when using Nicotine Replacement Therapy (NRT),. Some individuals may experience nicotine toxicity at higher doses; symptoms include severe vomiting, profuse excess saliva, severe diarrhoea, problems with heart rhythms; seizures; slowing of breath; and even death.

Nicotine also causes a wide range of harmful physiological effects on the body. More specifically, it causes an increase in heart rate; blood pressure; myocardial oxygen demand (i.e. the amount of oxygen required by the heart to pump blood) [6]. vasoconstriction (i.e. constriction of blood vessels); and rigidness/curvature of arteries. Thus, all these combined with nicotine create a substantial risk factor in developing atherosclerosis (i.e. hardening and narrowing of arteries) and myocardial infarction [7]. The time required for nicotine to get out of the human body range anywhere from several hours to several days depending on one's frequency of smoking practice. Ultimately, if one continues to smoke on a regular basis, the cumulative impact of nicotine (which can be toxic) poses a far greater illness/death risk exclusively associated only with cardiovascular disease/stroke and/or lung cancer as compared to somebody who doesn't smoke and/or has never smoked [8].

Smokers not only suffer from the effects of nicotine but are also absent from environmentally toxic substances present in cigarette smoke including carbon monoxide, ammonia, benzene, formaldehyde, hydrogen cyanide, etc, [9]. Cigarette smoke carbon monoxide attaches strongly to blood's haemoglobin molecules causing reduced blood oxygen-carrying capacity and adversely affecting tissues' ability to receive enough oxygen [10]. Additionally, the presence of other toxic substances in cigarette smoke can attack/lodge in the liver, kidneys, nerves, spinal cord etc., to either create irreversible damage or permanent dysfunction (e.g. effects of methanol on the central nervous system and/or optic nerve) [11-14].
The health effects of tobacco depend on the way it's used, how long someone has been exposed, and whether it was from smoking, chewing, or breathing in someone else's smoke. The most common way that tobacco harms health is through smoking, making smoking one of the top reasons for people to get sick and die from preventable causes. Smoking causes COPD (chronic obstructive pulmonary disease), which includes both emphysema and chronic bronchitis, as well as heart disease, stroke, and a variety of cancers, especially of the lungs, larynx, mouth, and pancreas [15,16].

In addition to these diseases, smoking can lead to diseases of the vascular system, including peripheral vascular disease, high blood pressure, and progressive endothelial dysfunction. All of these diseases are influenced by how long a person has been smoking, how much they smoke, and how old they were when they started smoking. Smoking at an early age and using cigarettes with a lot of tar increases the chance a person will develop these diseases. Secondhand smoke is another source of health problems related to tobacco, and people at any age can suffer health problems from secondhand smoke. Therefore, smoking has public health consequences that extend beyond the individual who smokes or uses tobacco products [17].

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The tobacco that is smoked has over 5,000 chemical compounds in it such as carbon monoxide (CO) and tar; many are harmful to the health of humans [18, 19]. CO has a much greater affinity than oxygen for hemoglobin. Smoking creates carboxyhemoglobin, leading to a reduction in the amount of oxygen that can be delivered to tissues. As a result, smoking causes a relative decrease in tissue's oxygen level (Hypoxia), thus causing a compensatory elevation in hemoglobin and RBC count [20].

Smokers have a greater concentration of hemoglobin, hematocrit, and RBC's; there is a direct relationship between elevation levels of these cells and smoking the number of cigarettes smoked daily [21]. Smoking also causes an increase in WBC count, including neutrophils and lymphocytes, due to the smoker's inflammatory response.

As well as the elevation of leukocytes, Smoking elevates platelet activity, raises cholesterol levels, and accelerates the progression of atherosclerosis and increases the risk of CV disease [22]. Additionally, smoking also causes lung function loss more quickly than would occur naturally due to aging. There is also a strong correlation between smoking and the development of chronic respiratory diseases such as COPD [23,24].

The likelihood of developing a smoking-related disease is determined by many different factors including the duration of smoking, the number of cigarettes smoked per day, the early age when someone starts to smoke, and the level of exposure to chemicals and carcinogens found in tobacco smoke [25-28].

METHODOLOGY

Study Design and Participants

This study was conducted during the period of October 2024 to April 2025 using a total of 80 male participants, which included both smoker (cigarette, hookah, or electronic cigarette) and healthy non-smokers as a control group (40 in each). The study was conducted to investigate how smoking affects selected hematologic parameters (Red Blood Cell Count, White Blood Cell Count, Hemoglobin Concentration, Erythrocyte Sedimentation Rate and Blood Group Distribution).

Materials and Equipment

The following were used in the study: Westergren glass tubes, EDTA tubes, sodium citrate tubes, pipettes, Westergren tube holder/rack, microscope slides, and anti-A, anti-B and anti-D reagents.

Sample Collection

Two mls of venous blood from each participant were collected via the standard technique of venipuncture with an aseptic technique. Each blood sample was placed into an EDTA tube and treated gently to avoid clotting and processed for hematological analysis as quickly as possible.

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Erythrocyte Sedimentation Rate (ESR)

ESR was determined using the Westergren method. Anticoagulated blood was collected into a sodium citrate tube and then drawn into a Westergren pipette to the zero mark. The pipette was then placed vertically in the holder/rack and left undisturbed for one hour. The ESR level was determined after that hour by measuring how many millimeters the erythrocytes settled within that hour.

Blood Group Determination

Blood groupings by ABO and Rh factor were accomplished through the Slide Agglutination Method, which consisted of placing blood drops from each individual onto a clean glass slide and mixing them with Anti-A, Anti-B, and Anti-D serum directly on the slide and/or using a gentle rocking motion to perform the slide agglutination reaction for 90 seconds. The presence or absence of agglutinated cells from each of the reagents was used to identify each subject's blood type.

Complete Blood Count (CBC)

Automated hematology analyzers were used to analyze complete blood counts. All blood tubes containing EDTA were stored upright until use and placed into a roller mixer for no longer than two minutes, after which they were used directly with the automated analyzer in accordance with the manufacturer's specifications for sample aspiration into the automated analyzer. Hematological parameters, including complete blood counts (CBC), such as RBC count, WBC count, hemoglobin (Hb) and platelet (PLT) count, were measured by the analyzer, and results were displayed automatically by the analyzer and printed within one minute following result measurement.

Statistical Analysis

The data were analyzed using appropriate statistics, as indicated in the above methods. For each subject, the mean and standard deviation (SD) of the dependent variable (dependent) for smokers and controls were determined. Independent sample tests were applied to evaluate differences between smoker and control groups, with a P-value < 0.05 considered to be statistically significant.

RESULTS

The distribution of smokers by age showed a distinctly different pattern than that of the controls. Smokers aged 30 - 40 comprised the largest proportion of smokers (40.0%), followed by those aged > 40 (35.0%), while smokers aged < 30 comprised the smallest group (25.0%).

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On the other hand, controls showed a larger proportion of persons aged < 30 as compared to persons aged 30 - 40, and a smaller proportion of persons aged > 40 compared to persons in the age range of < 30 (50.0%, 30.0%, 20.0%, respectively). This observation suggests that there is a relationship between an increase in smoking prevalence and increasing age and that the greatest smoking prevalence is found among middle-aged individuals.

Table 1: Distribution of Age Groups Among Smokers and Controls

Age Group (years)	Smokers (N=40)	%	Controls (N=40)	%
< 30	10	25.0	20	50.0
30–40	16	40.0	12	30.0
> 40	14	35.0	8	20.0

Duration of using smoking products

Trends were observed based on length of time using smoking products between users of arghile (waterpipe) and cigarette smokers. Most of arghile users had smoked for between 10-20 years (42.0%); followed by >20 years (33.0%); >10 years (25.0%). Most of cigarette users had smoked <10 years (52.0%); followed by 10-20 years (32.0%); followed by >20 years (16.0%). With these results, there are more cigarette smokers that are newer; compared to longer time arghile users.

Table 2: Distribution of Smoking Duration

Duration (years)	Arghile Smokers (N=21)	%	Cigarette Smokers (N=19)	%
< 10	5	25.0	10	52.0
10–20	9	42.0	6	32.0
> 20	7	33.0	3	16.0

Hematological Characteristics and Blood Groups

In regard to whether or not a preference for a specific ABO blood group exists in the distribution of blood groups among smokers, the data did not suggest any unequivocal marked preference for one of the ABO blood groups. Specifically, while both arghile and cigarette smokers exhibited a slightly increased incidence of blood group O when compared to the other blood groups, both groups showed a relatively low frequency of blood group AB.

In addition, most of the individuals in each of the two groups had Rh-positive blood (although the majority of the individuals from both groups had Rh-negative blood).

Thus, smoking behaviour and smoking-related hematological alteration do not appear to be strongly associated with blood group distribution [20] [30–33].

Table 3: Distribution of Blood Groups

Blood Group	Arghile Smokers	Cigarette Smokers
A	4	5
B	5	4
AB	3	3
O	9	7
Rh positive	17	16
Rh negative	4	3
Total	21	19

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Comparison of Hematological Parameters

There were a number of significant differences between smokers and non-smokers with regards to hematological parameters compared to controls.

Smokers had higher hemoglobin (Hb), packed cell volume (PCV) and white blood cells (WBC) than either controls or non-smokers ($P < 0.05$). These increases may represent physiological adjustments due to chronic hypoxia, as well as the impact of inflammation caused by smoking.

Smokers had a higher mean red blood cell (RBC) count compared to non-smokers but it was not statistically significant. There was also no significant difference in the platelet count (PLT) between smokers and non-smokers. In contrast, the erythrocyte sedimentation rate (ESR) was elevated moderately among smokers, suggesting some degree of low-grade inflammation [34].

Table 4: Comparison of Hematological Parameters

Parameter	Smokers (Mean ± SD)	Controls (Mean ± SD)	P-value
PCV (%)	46.10 ± 3.80	42.90 ± 2.60	0.012
Hb (g/dL)	15.10 ± 1.30	13.80 ± 1.10	0.004
WBC (10^3/µL)	9.10 ± 1.70	7.00 ± 1.50	<0.001
RBC (million/µL)	5.30 ± 0.50	5.10 ± 0.48	0.080
ESR (mm/hr)	18.50 ± 10.20	13.20 ± 7.90	0.020
PLT (10^3/µL)	250.40 ± 65.30	262.10 ± 58.40	0.120

Discussion

The findings of this study confirm that smoking significantly impacts various hematological parameters including those related to delivery of oxygen and inflammation.

The elevated white blood cell (WBC) counts noted in smokers support the concept that smoking promotes a chronic inflammatory response, possibly from irritation of the airways and subsequent systemic immune response associated with cigarettes. An elevated WBC count can also increase the likelihood of developing cardiovascular disease(s).

The elevated levels of hemoglobin and packed cell volume (PCV) in smokers can also be explained by the physiological adjustments (or compensatory mechanisms) that occur due to decreased availability of oxygen resulting from exposure to carbon monoxide at low levels. Elevated blood viscosity as a consequence of elevated hemoglobin/PCV level can increase the risk of developing thrombosis, leading to vascular complications.

Even though there was only a small increase in the number of red blood cells (RBCs) observed, a sustained elevation of RBC counts over time might lead to the development of secondary polycythemia. The increase in erythrocyte sedimentation rate (ESR) that was also observed, suggests that a greater degree of inflammation is present in smokers compared to non-smokers.

In comparison, there were no significant differences noted between the platelet indices for smokers versus non-smokers, indicating that smoking likely has an effect on platelet function rather than platelet counts.

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Combined, the results of this study are consistent with the literature demonstrating that smoking produces changes in hematological parameters that put individuals at risk for the development of both cardiovascular and inflammatory disease(s).

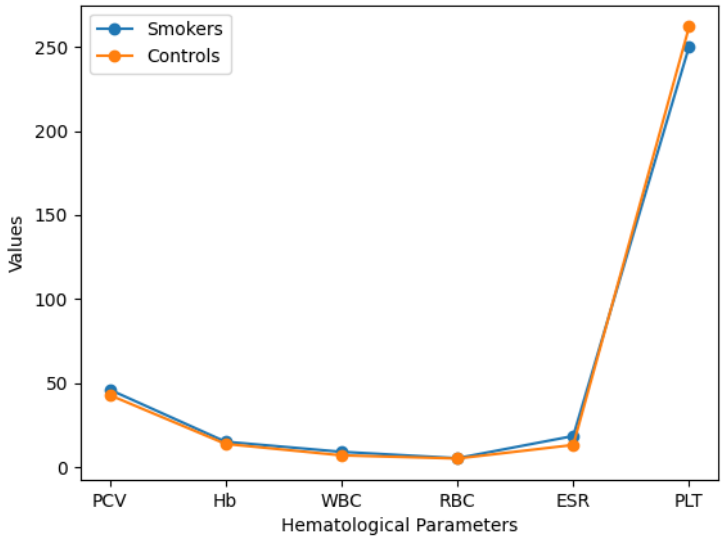

Figure 1: Illustrates a comparison of these hematological parameters for the Control group vs. the Smoking group.

CONCLUSION

In this research, we have found that smoking has a significant impact on multiple parameters of the blood system, even in fairly healthy individuals. For example, smoking has resulted in increases in hemoglobin levels and packed cell volume (his blood makes - as a result of a person's body trying to compensate for less oxygen reaching that person's body). Elevated levels of white blood cells (WBC) are indicative of an increased risk of chronic inflammation associated with the use of tobacco products.One area where no strong correlation between smoking and distribution of blood groups was observed. This is consistent with the notion that a person's genetic blood type does not influence their body's ability to respond hematologically to tobacco products. Given the extent of changes in the body due to smoking will ultimately have harmful effects on cardiovascular diseases and chronic inflammatory diseases. Therefore, periodic monitoring of various blood parameters will be an effective way to identify the presence of smoking-related complications at a relatively low cost.

Future studies with larger sample sizes should be conducted to investigate the long-term effects smoking has on blood parameters and explore the identification of other biological markers (biomarkers) that may be affected by being exposed to tobacco.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest regarding the publication of this manuscript. There are no financial, personal, or institutional relationships that could influence or be perceived to influence the work reported in this study.

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