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Effect of Tobacco Smoking on Some Blood Parameters in Males in Al-Bayda City, Libya

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Abstract:
Smoking tobacco is considered the worst human habit. Many harmful chemicals included in cigarettes have been connected to specific types of cancer. Numerous biochemical and hematological processes can be disrupted by cigarette smoking, according to laboratory tests. Determining the influence of tobacco use on some blood in males in Al-Bayda City, Libya, is the goal of the current study. 119 people, aged 20 to 55, participated in the study; 61 of them smoked, while the remaining 58 did not. They surveyed from October 20, 2023, until January 30, 2024. The smokers' durations ranged from six months to forty years. The participants were free of major health conditions, had never used drugs, and had not given or received blood in the previous six months. Medical data, health histories, and other relevant information were collected from participants through in-person interviews. The findings demonstrated that there were considerable alterations in the hematological parameters of experimental males, including MCV, MCHC, WBC, LYM, MON, GR, PCT, MPV, RDW-1, and PDW. The PLT was meaningfully lower in smokers than in non-smokers, but there was no important difference in the RBC, HGB, MCH, RDW, HCT, MPV, or PDW. The current study concluded that there were noteworthy differences between smokers and non-smokers in some hematological parameters. These differences may be associated with several future diseases, including an increase in atherosclerosis, PV, COPD, and/or CVDs.
Keywords: Cigarettes; hematology; males, El Bayda, smoke.

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تأثير تدخين التبغ على بعض معايير الدم لدى الذكور في مدينة البيضاء، ليبيا

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الملخص:

يُعتبر تدخين التبغ أسوأ عادة بشرية. وقد رُبطت العديد من المواد الكيميائية الضارة الموجودة في السجائر بأنواع مُحددة من السرطان. ووفقًا للاختبارات المعملية، يُمكن أن يُعطّل تدخين السجائر العديد من العمليات الكيميائية الحيوية والدموية. يهدف هذا البحث إلى تحديد تأثير تعاطي التبغ على بعض معايير الدم لدى الذكور في مدينة البيضاء، ليبيا. شارك في الدراسة 119 شخصًا، تتراوح أعمارهم بين 20 و55 عامًا؛ كان 61 منهم مدخنين بينما 58 غير مدخنين. أجريت الدراسة في الفترة من 20 أكتوبر 2023 إلى 30 يناير 2024. وتراوحت مُدّة التدخين لدى المُدخّنين بين ستة أشهر وأربعين عامًا. وكان المُشاركون ليس لديهم أي مشاكل صحية خطيرة، ولم يسبق لهم تعاطي المخدرات، ولم يتبرعوا أو يتلقوا دمًا في الأشهر الستة السابقة للدراسة. وقد تم الحصول على البيانات السريرية والتاريخ الطبي والمعلومات الأخرى ذات الصلة من المُشاركين من خلال مُقابلات شخصية معهم. أظهرت النتائج وجود اختلافات واضحة ما بين المدخنين وغير المدخنين في المعايير التالية: MCV، MCHC، WBC، LYM، MON، GR، PCT، MPV، RDW−1، وPDW.. بينما كان PLT أقل بكثير لدى المدخنين منه لدى غير المدخنين، ولكن لم يكن هناك فرق معنوي كبير في RBC، HGB، MCH، RDW، HCT، MPV، أو PDW. خلصت الدراسة الحالية إلى وجود اختلافات كبيرة بين المدخنين وغير المدخنين في بعض المعايير الدموية والتي قد ترتبط بعدة أمراض مستقبلية منها ارتفاع بزيادة خطر الإصابة بتصلب الشرايين، كثرة الحمر الحقيقية، مرض الانسداد الرئوي المزمن، و/أو أمراض القلب والأوعية الدموية.

الكلمات المفتاحية: السجائر، أمراض الدم، ذكور، البيضاء، دخان.

Introduction:

One of the leading causes of death worldwide and a major public health problem is tobacco smoking (TS) [1]. Smoking may have both immediate and long-term effects on hematological markers [2].

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There are almost 4,000 distinct chemicals in cigarette smoke (CS). Among other dangerous compounds, smokers are also exposed to carbon monoxide (CO), free radicals (FRs), nicotine (NCT), and airborne particles [3, 4]. Moreover, reactive oxygen species (ROS) are produced in large quantities by people as a result of CS products [5]. Furthermore, smokers have higher white blood cell (WBC) counts than nonsmokers, according to several studies [3, 6]. However, some studies found a link between smoking and red blood cell (RBC) numbers in smokers [7]. Additionally, tobacco use has been connected to numerous serious morphological and biological problems in humans [3]. However, carcinogenesis and clonal hematopoiesis are also linked to extremely potent inflammatory stimulants that are linked to hazardous TS components that are associated with smoking [8]. Furthermore, it is estimated that TS accounts for 8.8% of all deaths globally and nearly four million deaths each year [9]. In addition, the WOH predicts that TS causes eight million deaths annually by 2030, with more than 80% of those deaths attractive room in low- and middle-income countries [10]. Several studies demonstrate that TS use significantly alters the lipid profile from its normal state and that smoking cigarettes is a main risk issue for a number of heart-related conditions, including peripheral vascular diseases, coronary artery disease, atherosclerosis, and others [11]. Furthermore, rheumatoid arthritis and immune system dysfunction have been connected to smoking [12]. Many studies have shown that smoking is bad for overall health and increases the risk of developing many diseases and pathological conditions, such as cancer, metabolic syndrome, gastrointestinal disorders, pancreatitis, and chronic obstructive pulmonary disease (COPD) [4]. NCT impairs vascular function, fosters endothelial dysfunction, and results in a clot forming in the coronary arteries. Moreover, hypoxia and sub-endothelial oedema may be caused by elevated carboxyhemoglobin (COHb) levels due to variations in vascular penetrability and lipid accumulation [13]. Furthermore, there is no question that the FRs and peroxides found in TS play a part in the etiology of a number of diseases, such as cancer, atherosclerosis, and inflammation, as well as physiological processes like thromboxane and prostaglandin synthesis [4]. So, the current study was conducted on the effect of TS on blood parameters in males in Al-Bayda City, Libya.

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Materials and methods:

Subjects:

The current investigation sought to ascertain the effects of smoking on the hematological indicators of a sample of clinically healthy volunteers. Of the 119 individuals in the study, who were between the ages of 20 and 55, 61 smoked, and 58 were non-smokers. The study period was from October 20, 2023, to January 30, 2024. The smokers ranged in age from six months to forty years. The participants were free of major health conditions, had never used drugs, and had not given or received blood in the previous six months. Medical data, health histories, and other relevant information were collected from participants through in-person interviews.

Laboratory test:

Venous blood samples were transferred into tubes with 15% EDTA. The complete blood count was calculated using a fully automated hematological analyzer within one to two hours of blood collection. A commercially available, standardized calibration kit was used to calibrate the hematological analysis. The CBC count.

WBC, RBC, hemoglobin (Hb), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), hematocrit (HCT), red cell distribution width (RDW), platelets (PLT), platelet criticality (PCT), mean platelet volume (MPV), and platelet distribution width (PDW) were among the parameters measured in this study.

Statistical examination:

The SPSS program for Windows was used to examine the data reported in this study using the 2-way ANOVA and the post hoc descriptive tests. Smokers' and nonsmokers' hematological levels were compared using an unpaired "t" test. Statistical significance was clear as a P value < 0.05. Standard error of means (SEM) is used to depict data.

Results:

All hematological parameter results among experimental males are reported in Tables 1, 2, and 3, and all parameters are stated using the mean and SEM.

Hematological indices such as MCV, MCHC, WBC, LYM, MON, GR, PCT, MPV, RDW-1, and PDW were considerably greater in smokers than in non-smokers, according to the tables. Although the RBC, Hb, MCH, RDW, HCT, MPV, and PDW did not significantly

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differ between smokers and non-smokers, the PLT did reveal a considerably lower value.

Table 1: Shows a comparison of RBC, Hb, MCV, MCH, MCHC, HCT, and RDW parameters in smokers and non-smokers.

Parameters	Smoking (Mean ± SEM)	Non-smoking
RBC	5.1± 0.04 A	5.09±0.05 A
Hb	15.9±0.16 A	14.6±0.20 A
HCT	45.3±0.45 A	44.0±0.51 A
MCV	88.1±0.82 A	84.7±0.90 B
MCH	29.3±0.31 A	28.5±0.31 A
MCHC	38.5±5.31 A	33.1±0.26 B
RDW	11.5±0.14 A	11.9±0.16 A
RDW-1	41.6±0.86 A	39.9±0.61 B

- Red blood cell (RBC), hemoglobin (Hb), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), hematocrit (HCT), red cell distribution width (RDW), and red cell distribution width-1 (RDW-1).

Table 2: Shows a comparison of WBC, LYM, MON, and GR parameters in smokers and non-smokers.

Parameters	Smoking (Mean ± SEM)	Non-smoking
WBC	7.9± 0.23 A	7.4±0.31 B
LYM	37.0±1.43 A	27.2±2.37 B
MON	4.5±0.19 A	2.78±0.34 B
GR	58.3±1.53 A	52.0±2.18 B

- White blood cell (WBC), lymphocytes (LYM), monocytes (MON), and granulocytes (GR).

Table 3: Shows a comparison of PLT, PCT, MPV, and PDW parameters in smokers and non-smokers.

Parameters	Smoking (Mean ± SEM)	Non-smoking
PLT	238.6±8.88 B	269.8±10.32 A
PCT	0.20±0.008 A	0.24±0.01 A
MPV	8.5±0.13 A	8.95±0.13 A
PDW	15.7±0.77 A	14.3±0.39 A

- Platelets (PLT), platelet criticality (PCT), mean platelet volume (MPV), and platelet distribution width (PDW).

Discussion:

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CS comprises the majority of the harmful and cancer-causing substances found in mainstream smoke, which have an impact on human health due to oxidative stress, FRs, NCT, CO, and other substances [5]. Furthermore, some toxic substances, particularly free radicals like HOCL, superoxide anions, hydroxyl radicals, and H_2O_2, which are found in CS, can harm cellular mechanisms and cause severe inflammation. These ROS can also impair lipids, proteins, and DNA, altering the structure and biological role of cells [14, 15].

In this study, the local population's smokers and non-smokers were compared using the hematological parameters, which the parameters of the participants varied greatly, according to the experimental results; smokers had significantly greater MCV, MCHC, WBC, LYM, MON, GR, PCT, MPV, RDW-1, and PDW, while non-smokers had much lower PLT. RBC, HGB, MCH, RDW, HCT, MPV, and PDW levels did not significantly differ, according to the data. The precise processes underlying the high epidemiological associations between CS and cancer, COPD, and cardiovascular disease (CVDs) are still unclear. The development of endothelial damage is one of the negative consequences of smoking [16, 17], acceleration of the development of new lesions and coronary progression [18], as well as general changes in the hemostatic and lipid systems [19]. The most helpful stage in the primary analysis of atherosclerosis may be identifying endothelial damage. Even while the endothelium releases a large number of chemicals into the bloodstream and arterial wall, not all of these compounds are endothelium-specific, making them less useful for research or diagnosis [20]. Furthermore, the WBC count is arguably the most practical, affordable, and straightforward biomarker for endothelial injury [15]. According to the study's findings, habitual smokers' WBC levels were noticeably greater than those of non-smokers. According to other published results [15, 21, 22], male smokers in this study had a high WBC count. According to Freedman et al. [21], current smokers had a median total WBC count that was 36% greater than that of non-smokers [6]. It's unclear what process causes smoking to raise WBC counts. NCT may cause a rise in blood lymphocytes; however, it has also been proposed that inflammatory inspiration of the bronchial tract causes a rise in inflammatory indicators in the blood [17, 23, 24]. Although leukocytosis might only be a sign of tissue injury brought on by smoking, a high count might contribute to CVDs through a number

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of pathologic processes, including inflammation, microvasculature blockage, hypercoagulability, and infarct growth [25, 26]. Atherosclerosis and CVDs can be independently predicted by WBC count, according to several studies [27, 28]. Male smokers may be more susceptible to atherosclerosis and cardiovascular diseases than non-smokers, as indicated by their elevated WBC count.

In CS, partial combustion of carbon-containing material results in the production of CO. In comparison to O2, CO has a relatively high affinity for Hb (around 200 times) [29]. Accordingly, CO causes RBCs to create COHb by dislodging O2 from Hb, which lowers the amount of O2 released into tissues [30]. Smokers have been shown to have higher levels of HCT and Hb, which are probably compensatory for CO exposure [31]. A hypercoagulable state may be exacerbated by elevated HCT and Hb concentrations seen in smokers [30, 32]. On the other hand, the study revealed that the smoker group's MCV and MCHC levels were considerably lower ($p<0.05$) than those of the non-smoker group. Asif et al. [3] discovered that smokers have higher MCV. MCV indicates RBC size, and anemia is present if there are RBCs that are either smaller or larger than normal. Our study's elevated MCV levels suggest that participants may have megaloblastic, hemolytic, pernicious, or macrocytic anemia, which is naturally caused by iron and folic acid deficiencies. Additionally, this might be because CS damages the bone marrow, causing immunological reactions from inflammation after years of smoking [14]. According to the study, smokers had much greater MCHC. This may be due to the hypothesis that thyroid issues, vitamin B12 deficiency, or folic acid could be the cause of the elevated MCHC [5, 33]. According to the study, there was no important difference in MPV, PDW, or PCT between smokers and nonsmokers. However, there was no discernible difference between the smoker and nonsmoker groups in terms of HGH, RBCs, and HCT levels. These results were consistent with those of [4] and [34] but not with those of Nadia et al. [34] and Roethig et al. [31], who discovered that the smoking group had significantly higher levels of these parameters.

Conclusion:

The study concluded that there were important alterations between smokers and non-smokers in the following hematological parameters: MCV, MCHC, WBC, LYM, MON, GR, PCT, MPV, RDW-1, and PDW. These differences may be associated with

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several future diseases, including an increase in atherosclerosis, PV, COPD, and/or CVDs.

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