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The impact of electronic cigarettes on blood cell composition and immune system performance

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ABSTRACT

The use of e-cigarettes has skyrocketed in recent years due to a lack of awareness about the dangers and problems connected to these devices. This is particularly accurate given that the nicotine content may be adjusted, which over time may induce addiction. In order to ascertain its detrimental effects on the immune system, this study sought to ascertain how it affected several immunological components. 68 participants (male), 56 of whom were smokers and 12 of whom served as control samples, were examined using ELISA and CBC procedures to measure the concentration of these variables. Participant were aged between 19 and 46 years. While the remaining blood components exhibited elevated concentration at ages 30 and above, the results indicated that there were highly significant changes in blood components when compared to the control and for years 19–29. All blood component rates were noticeably higher than in control samples. In comparison to control samples, smokers had higher levels of IL-12, IL-5, and IFN-γ, and people under the age of 29 had higher levels of IL-5 and IFN-γ than people older than them. The levels of IL-12 were comparable to the control samples, but the levels of IL-5 and IFN-γ rose dramatically with longer smoking times and showed a strong, significant connection. While IL-12 levels did not clearly rise in response to rising nicotine concentrations in contrast to control samples, IL-5 and IFN-γ levels did rise noticeably. The study found elevated blood component (IL-12, IL-5, and IFN-γ) in smoker and those under 29 years old, but no significant increase in nicotine content.

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1. INTRODUCTION

E-cigarettes are electronic nicotine delivery systems (ENDS) that spray nicotine solution mixed in vegetable glycerin and propylene glycol. They were created to provide nicotine without exposing users to many of the harmful byproducts of burning tobacco at the same time [1]. E-cigarettes are regarded by some as a safer option to traditional cigarettes due to their ability to burn tobacco without generating as much combustion product [2]. Additionally, individuals who are unable or unwilling to give up smoking may find that e-cigarettes make quitting smoking easier [3]. The entire list of compounds produced by cigarettes that catch fire is in the several thousand range. Numerous substances cause or encourage lung damage, cardiovascular disease, and carcinogenesis, and others are extremely toxic and lethal [4]. Therefore, it makes sense to assume that nicotine administered without reactive molecules would be less dangerous than nicotine delivered with a combination of toxins produced during burning [5]. And from that, it follows that breathing nicotine would be much safer if all of the tar and other combustion products were eliminated. Regretfully, the situation is more problematic than anticipated for a variety of reasons. Regarding these products' safety and long-term effects on health, not much is known. Most products contain nicotine, even if there is a tendency toward the usage of vaporized poly-substances [6].

There are worries that this makes ENDSs more addictive or raises the likelihood that teenagers may use combustible cigarettes in the future. Because nicotine content in certain recent goods has increased, this issue has become much more important [7]. The average concentration of nicotine has been rising over time, with Juul having a significantly higher net nicotine content than cigarettes. More nicotine can be delivered while reducing the negative side effects by carefully controlling the fraction of protonated nicotine. Modifying the protonated fraction may also have an effect on the likelihood of addiction by altering pharmacokinetics and sensory impressions [8]. The menthol taste found in recent nicotine delivery systems may influence the expression of some cytokines, according to certain findings. According to [7] smokers of menthol-flavored e-cigarettes had a significant decrease in the evaluated inflammatory markers in their serum [9]. This study aims to determine the effects of e-cigarette vapor on blood cells, such platelets, red and white blood cells, and others. to determine whether the immune system is impacted by e-cigarettes.

2. MATERIALS AND METHOD.

2.1. Samples collection and ELISA performance

The study includes 68 samples (male) collected from Mosul city, 56 from smokers, and 12 control collections from July to November (2023), with ages ranging from (19 to 46) years. Blood samples overall were taken, the samples were examined using an Auto Hematology Analyzer made in Rayto, China, after two milliliters of venous blood were put in anticoagulant tubes with EDTA. Jell tubes—tightly sealed, dry, and free of anticoagulants—were used to hold the residual blood. Twenty minutes were spent with the tubes at room temperature. Serum was then obtained by centrifuging at 9000 x g for 15 minutes. Before the necessary tests were carried out for this investigation, the serum was kept in a deep freezer at -20°C in dry, sterile Eppendorf tubes, (for the ELISA test, serum samples are utilized). The study was conducted in the research laboratory in the University of Mosul, Department of Biology, College of Sciences. Table (1) illustrates description of kits used in this study.

Table 1: description kits used in this study

Parameters	Company name	Type assay	Catalog number or code
Interferon γ	BT.LAB	Sandwich ELISA	E0056Mo
	Bio assay Technology Laboratory		
Interleukin 12	BT.LAB	Sandwich ELISA	E0036Mo
	Bio assay Technology Laboratory		

2.2. Statistical analysis

With the employing of SPSS version 21, the data was analyzed using the t-test to compare the concentrations of IL-5, IL-12, and IFN- γ between control and smoking individuals at a significance level of $p \leq 0.05$. Alphabetic letters were used to indicate the many important components at the 1% probability level using the Duncan's multiple range test.

3. RESULTS AND DISCUSSION

3.1. Effect E.C smoking on blood count according to the age

Table (2) shows the blood cell parameters WBC, Monocyte %, Lymphocyte %, Hb, PLT, and RBC for those who mistake e-cigarettes as safe and non-smokers, divided by age (over 30 and from 19-29 years) so that the differences can be seen clearly.

Table 2: Effect of electronic cigarettes smoking on hematological parameters by age group

Smoker above 30 years	control	Smoker 19-29 years	Control	Parameters
8.953±0.193	6.0653±0.231	9.275±0.528	7.052±0.586	W.B.C 10^3/µL
0.743±0.019	0.544±0.042	0.773±0.128	0.561±0.029	Monocyte %
2.791±0.135	1.834±0.097	2.81±0.124	1.898±0.089	Lymphocyte %
17.33±0.35 b	14.56±0.35	16.03±0.86 cd	14.56±0.35	Hb g/dL
269.33±36.47 a	201.0±51.50 a	260.0±52.25 a	201.0±51.50 a	PLT 10^3/µL
5.77±0.22 a	4.46±0.19 b	5.31±0.10 a	4.46±0.19 b	RBC 10^6/µL

Blood parameters rate were considerably higher in smokers over 30 than in control individuals, and smokers between the ages of 19 and 29 had higher blood cell counts, while smokers over 30 had higher rates of the other blood components. When blood components (Hb, PLT, and RBC) appear at high rates in smokers, it indicates that there is a persistent inflammatory state due to the effects of nicotine on the body. This state of inflammation causes blood component levels to rise in comparison to control subjects.

The substantial difference in the number of red blood cells compared to the control samples shows that smoking directly affects red blood cells and their function because nicotine damages and distorts red blood cell membranes, especially the phospholipid presents in the membrane. Additionally, smokers' blood levels of carbon dioxide rise more quickly [10].

According to another study, the effect of electronic cigarettes is responsible for the rise in hemoglobin concentration and the percentage of packed blood cell volume. The study found that the carbon monoxide inhaled from cigarettes combines with hemoglobin and prevents it from combining with oxygen, which causes erythropoietin to be secreted and hemoglobin to increase in order to make up for the oxygen shortage [11]. It is also possible that the substances included in electronic cigarettes are the cause of the rise in hemoglobin concentration and the percentage of packed blood cell volume. Electronic cigarettes are known to contain several metals, including copper, nickel, and cadmium. Cobalt and nickel chloride promote the heme protein's synthesis of erythropoietin [12].

Table (3) gives you important values for blood cells, which are White Blood Cells (WBC), Monocyte %, Lymphocyte %, Hemoglobin (Hb), Platelets (PLT), and Red Blood Cells (RBC). The study gives the average with standard error for each group: e-cigarette smokers and non-smokers, who are further compared by age at 30 or more and 19-29 years. It becomes easy to assess how smoking electronic cigarettes could impact blood makeup in individuals from different age groups.

Table 3: Effect of electronic cigarettes use on specific interleukins by age group.

	Age	N	Mean	t-value	sig	Std. Deviation	Std. Error Mean
IL-12(pg/ml)	control	12	0.29	/	/	0.06281	/
	less than 29 years	28	0.3110	1.474	0.152	0.08887	0.02375
	more than 30 years	28	0.4279	1.474	0.160	0.28297	0.07563
IL-5(pg/ml)	control	12	9.55	/	/	6.59756	/
	less than 29 years	28	51.7876	1.563	0.130	74.70465	19.96566
	more than 30 years	28	18.9526	1.563	0.138	24.39755	6.52052
IFN- γ (ng/L)	control	12	52.69	/	/	8.44523	/
	less than 29 years	28	82.3667	0.902	0.375	129.34225	34.56817
	more than 30 years	28	13.715	0.902	0.376	186.95421	49.96561

The levels of specific cytokines in smokers, the control group, and both age groups of research participants are shown in Table No. (2). The levels of interleukin 5 and interferon α were higher in those under 29 than in those over 30 and higher than the control in all age groups. In contrast, the levels of interleukin 12 were very close to the control samples in both age groups. The current study's findings demonstrated that both age groups' levels of IFN- α had significantly increased. When compared to other investigations, the findings of the prior study revealed different findings regarding the level of IFN- γ [13]. According to some research, a healthy 46-year-old man experienced weight loss, fever, night sweats, and respiratory discomfort after using e-cigarettes for a month [14].

There is increasing evidence that lymphocytes and macrophages play an important part as immunocytes in the chronic inflammatory process brought on by cigarette smoking. IFN- γ, which functions as a macrophage-activating factor, may be produced by activated T cells. Numerous inflammatory proteins secreted by activated macrophages may coordinate the inflammatory process. Macrophages can release the chemokines IFN-inducible protein (IP-10), IFN-inducible T-cell α-chemoattractant (I-TAC), and monokine induced by IFN- γ (Mig). These chemokines may be chemotactic for CD8+ T cells through their interaction with smokers' CCR5 (markers of T helper 1 cells) receptor-CCL5 [15].

Table (4) explores the impact of e-cigarette use on particular immune system markers called interleukin-12 (IL-12), interleukin-5 (IL-5) and Interferon-gamma (IFN-γ). An analysis was made according to how long people used e-cigarettes: a Control group, and people who smoked e-cigarettes for 1-3 years or 4-7 years. By looking at the table's Mean, N, and Std. Deviation for every group and parameter, a comparison may be made to check if smoking e-cigarettes changes these immune responses.

Table 4: Relationship between duration of E-cigarette use and cytokine level

period smoking		IL-12 (pg/ml)	IL-5 (pg/ml)	IFN- γ (ng/L)
Control	Mean	0.2930 a	9.5557 a	52.6900 a
	N	12	12	12
	Std. Deviation	0.06281	6.59756	8.44523
1-3 year	Mean	0.4134 a	33.1607 a	114.18 a
	N	30	30	30
	Std. Deviation	0.27413	58.8279	158.707
4-7 year	Mean	0.3407 a	62.2314 a	149.21 a
	N	14	14	14
	Std. Deviation	0.12755	70.7640	226.992

When smoking for longer periods of time, nicotine accumulate in the body in direct proportion, and this accumulation rises as more nicotine enters the body. Table 3 illustrates the correlation between the rates of the cytokines under investigation in smokers and the length of time they smoked in comparison to the control samples. Both IL- 5 and IFN- γ seemed to rise in direct proportion to the length of smoking, appearing to peak in smokers for 4–7 years. Although the rate of IL-12 showed several distinct and significant changes, it occurred at a close level with the increase in smoking duration when compared to the control [16]. IFN-γ is mainly released by natural killer (NK) cells and activated T cells. It can promote macrophage activation, mediate antiviral and antibacterial immunity, improve antigen presentation, coordinate lymphocyte-endothelium interaction, orchestrate innate immune system activation, regulate Th1/Th2 balance, and control cellular proliferation and apoptosis [16].

Table 5: Impact of nicotine concentration in E-cigarette on cytokine expression

Percentage-nicotine		IL-12 (pg/ml)	IL-5 (pg/ml)	IFN- γ (ng/L)
control	Mean	0.2930 a	9.5557 a	52.6900 a
	N	12	12	12
	Std. Deviation	0.06281	6.59756	8.44523
less than 15 %	Mean	0.4117 a	31.9142 a	107.16 a
	N	26	26	26
	Std. Deviation	0.29493	41.8159	168.776
more than 16 %	Mean	0.3593 a	57.5718 a	151.56 a
	N	18	18	18
	Std. Deviation	0.12069	85.1800	198.537

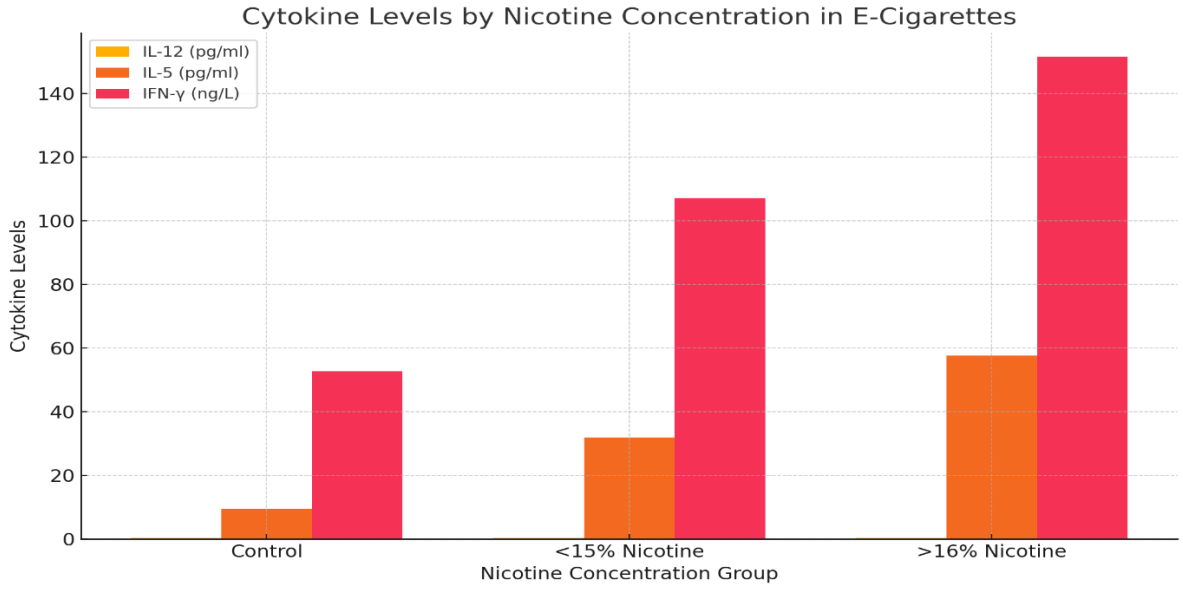

Figure 1. Show cytokine level by nicotine concentration in E-cigarettes

Dendritic cells, neutrophils, and macrophages all release IL-12 in response to antigen stimulation. Because of its ability to stimulate T cells and NK cells, IL-12 is categorized as a cytotoxic lymphocyte maturation factor (CLMF) and an NK cell stimulator factor (NKSF). Furthermore, IFN-γ is positively correlated with IL-12 secretion, and IL-12 increases its expression [17]. T-cells produce interleukin-5 (IL-5), which is crucial for the eosinophil response to allergens and parasites [18]. Illustrating how quickly e-cigarettes can cause negative effects. After a year of e-cigarette use, adolescents' rates of chronic bronchitis symptoms, such as a persistent cough, phlegm, or bronchitis, doubled [19].

According to the findings of one study, e-cigarettes may have a comparable effect on the lungs' defense proteins and neutrophilic response as CS users [20]. Vapors from electronic cigarettes modify several cell types, including the generation of pro-inflammatory cytokines, morphological changes, decrease cell viability, and impairment of defense against germs and viruses [21]. Results from table (5) link the amounts of cytokines (IL-12, IL-5) and Interferon-gamma (IFN-γ) to various nicotine concentrations in e-cigarettes as show in Figure (1). The researchers examined lung function similarly in three groups: (1) non-smokers; and (2), (3) users of e-liquids with less than 16% nicotine and those with more than 16% nicotine. Averages, numbers of participants, and the range of values are provided in the table, so you may see how different nicotine levels in e-cigarettes could influence immune factors across the groups.

Electronic cigarettes, as opposed to regular cigarettes, let users control how much nicotine they take in. In order to determine the rates of the cytokines under investigation in relation to the amount of nicotine consumed, the research first divided the participants into two groups: those who consumed less than 15% and those who consumed more than 16%. In comparison to the control samples, the second group's levels of interleukin 5 and IFN- γ were higher than the first's. In the two groups and the control group, the level of interleukin 12 appeared at a similar rate, although the level of interferon gamma was higher. Both T cells and macrophages, which are involved in humoral and cellular immunity, release this cytokine in response to any foreign body that enters the body. The quantity of nicotine that entered the body was closely correlated with the impact of these cells.

Nicotine and other cigarette smoke components have been connected to weakened human immunity, impacting both immunosuppressive and immunoactivity processes, increased susceptibility to autoimmune disorders, and lung infections. It has been demonstrated that nicotine suppresses immunological responses. raise inflammation and weaken antibacterial defenses in in vitro experiments. Therefore, there are worries about the immunological effects of e-cigarettes [22]. When vapors were inhaled, the amount of macrophages going up in the airways was like what was seen after exposure to cigarette smoke, suggesting a small inflammatory reaction [23]. It was seen that the protein surface markers CD11b and CD66b, which signal neutrophil reactions, were present more on neutrophils after exposure to e-cigarette smoke [24].

It was found by one study that sub-chronic exposure to nicotine elevates many cytokines and chemokines in bronchoalveolar lavage fluid (BALF) through alpha7 nicotinic receptors. IL-1α, IL-2, IL-9, IFN-γ, GM-CSF, and MCP-1/CCL2 are considered to be some of the inflammatory cytokines. Levels are managed by activation receiver, expression of normal T cells, and the secretion of (RANTES/CCLS), and the improvements in IL-1β, IL-5, and TNFα are not because of nAChRα7 [25].

Research limitations limited biomarkers assessed: the study only looked at a few basic hematological indicators and immune-related biomarkers (IL-12, IL-5, and IFN-γ). Other significant immunological and inflammatory markers that could offer a more thorough knowledge of immune system disturbance were not investigated.

4. CONCLUSSION

According to this study, male users between the ages of 19 and 46 who use electronic cigarettes have drastically changed blood cell composition and immune system markers. In particular, smokers had higher levels of two important immunological cytokines—IL-5 and IFN-γ—than non-smokers, especially those who were younger (less than 29 years old), had been exposed for a longer period of time (4–7 years), or had higher nicotine concentrations. IL-5 and IFN-γ levels increased in proportion to both smoking duration and nicotine %, indicating a dose-response relationship, while IL-12 levels did not consistently rise in relation to nicotine content. Furthermore, smokers had markedly higher levels of hematological parameters such hemoglobin, red blood cells, and white blood cells, suggesting a systemic inflammatory response that may have been brought on by nicotine and other chemicals in e-cigarette vapor. These results cast doubt on the perceived safety of e-cigarettes in comparison to conventional tobacco products and emphasize the immunomodulatory and pro-inflammatory effects of e-cigarette usage. To evaluate the long-term health impacts of e-cigarette exposure and to ascertain its chronic effects on human immunity, more longitudinal and mechanistic research is necessary.

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