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Effectiveness of smoking on ACE-2, IL-6 levels in men

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Abstract

Smoking is a significant risk factor for various cardiovascular and respiratory diseases, impacting key biomarkers such as Angiotensin-Converting Enzyme 2 (ACE-2) and Interleukin-6 (IL-6). ACE-2 is crucial for regulating blood pressure and cardiovascular health, while IL-6 is a cytokine involved in inflammatory responses. Recent research indicates that smoking disrupts the renin-angiotensin system, potentially lowering ACE-2 levels and exacerbating systemic inflammation. This study aimed to investigate the relationship between smoking and these biomarkers, shedding light on the underlying pathophysiological processes and informing targeted interventions for smokers at risk of inflammatory and cardiovascular diseases. The study involved 100 participants, including 50 smokers and 50 healthy controls, with significant differences observed in physiological and biochemical parameters such as systolic blood pressure, hemoglobin, packed cell volume, ACE-2, and IL-6 levels. Results showed that smokers had higher systolic blood pressure, hemoglobin, and PCV, alongside elevated ACE-2 and IL-6 levels, indicating chronic inflammation and cardiovascular risk. Furthermore, positive correlations were found between smoking frequency and duration with these health parameters, emphasizing the detrimental effects of smoking on overall health. These findings underscore the urgent need for smoking cessation programs to mitigate the associated health risks.

Keywords: ACE-2, IL-5, smoking, cardio vascular disease, atherosclerosis

Introduction

Smoking is a well-documented risk factor for a range of cardiovascular and respiratory diseases. Among its numerous detrimental effects, smoking influences several key biomarkers involved in inflammatory and regulatory processes within the body ^[1]. Two such biomarkers are Angiotensin-Converting Enzyme 2 (ACE-2) and Interleukin-6 (IL-6). ACE-2 plays a critical role in the renin-angiotensin system, regulating blood pressure and maintaining cardiovascular homeostasis, while IL-6 is a cytokine central to inflammatory responses and immune system regulation ^[2].

By blocking the effects of Angiotensin II, a peptide that can worsen inflammation and vasoconstriction, ACE-2 is well-known for its preventive properties against hypertension and cardiovascular disease. On the other hand, IL-6 has been connected to chronic illnesses like diabetes and cardiovascular disease and is frequently increased in a variety of inflammatory situations. The way these molecules interact can have a big impact on how diseases develop and how healthy people are overall ^[3].

The possible effects of smoking on these biomarkers have been brought to light by recent research. It has been demonstrated that smoking upsets the renin-angiotensin system's equilibrium, which may lower ACE-2 levels and impede the system's protective effects. Furthermore, smoking causes systemic inflammation ^[4].

This study aims to explore the relationship between smoking and these biomarkers, providing insights into the underlying pathophysiological processes and contributing to the development of targeted interventions for smokers at risk of inflammatory and cardiovascular diseases ^[5].

Materials and Methods

Statement of Ethics

Written informed consent was provided by each individual to take part in the research and was a student at Hilla University College, located in the province of Babil. The study was carried out in 2024 with approval from Al-Hilla University College's Ethics Committee.

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Selection of Smokers and controls

The study involved 100 people in all, 50 of whom were smokers and 50 of whom were healthy controls from Hilla University College. A pulmonologist and an endocrinologist collected each participant's clinical history and illness status 1.

Smokers' body weights were recorded using a digital scale that had an accuracy of 1000 g. With a 3 cm accuracy level, the patient's height was measured with a measuring tape while their shoulders were in their normal position. Weight (kg)/(height (cm)) was the formula used to determine body mass index (BMI) 2. A conventional mercury column manometer was used to measure blood pressure. The pulmonologist took all measurements from the left arm. Samples of venous blood were taken. None of the smokers or healthy people had a history of infectious, malignant, autoimmune, or allergy conditions. The average age of smokers was 26.8 ± 7.8 , while the average age of controls was 26.6 ± 6.8 years. In the patient group, there was no discernible age difference between smokers and non-smokers; however, the controls were not age matched ($p < 0.001$).

Biomarkers assay

Each patient had two milliliters of blood drawn into an EDTA tube and three milliliters drawn into a gel tube. Prior

to further investigation and an instant CBC of the whole blood, played being separated, the serum was stored at -20°C . ELISA, an enzyme linked immunosorbent test, was examined After to quantify serum IL-6 and TNF- α levels in accordance with the manufacturer's instructions (BT Lab, China). The BT Lab ELISA kits' analytical sensitivity ranges for serum samples were 0.05-20 ng/m pg/ml for ACE-2 and 2-600 ng/L pg/ml for IL-6 7.

Statistics

SPSS software version 16 was used to conduct statistical analyses, which included independent t-test, Fisher's exact test, chi-square, one-way analysis of variance (ANOVA), and descriptive statistics. Statistical significance was defined as P values below 0.05.

Results

The results provided in the study show significant differences in various physiological and biochemical parameters between smokers and non-smokers. These differences are particularly evident in systolic blood pressure (sys), hemoglobin (Hg), packed cell volume (PCV), angiotensin-converting enzyme 2 (ACE-2), and interleukin-6 (IL-6). The analysis also includes correlations between the duration and frequency of smoking with these parameters among smokers [8].

Smokers showed higher blood pressure, hemoglobin, PCV, ACE-2 and IL-6.

Group Statistics					
	Group	N	Mean	Std. Deviation	P. value (Independent t test)
sys	Smoker	37	124.6	10.8	0.007 *
	Control	50	119.7	5.7	
Dia	Smoker	37	82.7	6.1	0.365
	Control	50	81.60	5.2	
Hg	Smoker	37	14.7	1.7	0.001 *
	Control	50	13.6	1.1	
PCV	Smoker	37	43.6	4.03	<0.0001 *
	Control	50	38.7	3.34	
ACE-2 (pg/ml)	Smoker	37	4.39	0.8	<0.0001
	Control	50	1.98	0.7	
IL-6 (pg/ml)	Smoker	37	30.86	13.9	<0.0001 *
	Control	50	14.04	9.1	
Age	Smoker	37	26.8	7.8	0.931
	Control	50	26.6	6.8	
BMI	Smoker	37	25.2	5.5	0.935
	Control	50	25.3	4.4	

The mean systolic blood pressure was significantly higher in smokers (124.6 mmHg) with a p-value of 0.007 in contrast to the control group (119.7 mmHg). This suggests that systolic blood pressure, an established risk factor for cardiovascular illnesses, may be raised as a result of smoking [9].

A statistically significant p-value of 0.001 indicated that the mean hemoglobin level of smokers was higher (14.7 g/dL) than that of the control group (13.6 g/dL). Elevated hemoglobin levels in smokers could be a response to prolonged exposure to cigarette smoke's carbon monoxide, which lowers oxygen delivery to tissues and causes the body to make more hemoglobin to make up for it¹⁰. With a p-value of less than 0.0001, the mean PCV was significantly greater in smokers (43.6%) than in the control group (38.7%). Another measure of the body's reaction to

smoking-induced hypoxia, which raises the risk of thrombotic events, is an increase in PCV, also referred to as hematocrit [11].

The study found a significant increase in ACE-2 levels in smokers (4.39 pg/ml) compared to the control group (1.98 pg/ml) with a p-value of <0.0001. ACE-2 plays a crucial role in the cardiovascular system and has also been implicated in the pathogenesis of COVID-19, making this finding particularly relevant in understanding the potential health impacts of smoking [13].

With a p-value of less than 0.0001, smokers' IL-6 levels were significantly higher (30.86 pg/ml) than those of the control group (14.04 pg/ml). Smokers have higher levels of the pro-inflammatory cytokine IL-6, which is a sign of chronic inflammation and a risk factor for several chronic conditions, including as heart disease and cancer [12].

Discussion

The data shows significant positive correlations between the frequency of smoking and systolic blood pressure ($r = 0.564$, $p < 0.0001$), hemoglobin ($r = 0.712$, $p < 0.0001$), PCV ($r = 0.542$, $p = 0.001$), ACE-2 ($r = 0.689$, $p < 0.0001$), and IL-6 ($r = 0.731$, $p < 0.0001$). According to these associations, smoking more frequently is linked to poorer health outcomes in these areas. Additionally, there was a strong positive connection between the length of smoking and levels of ACE-2 ($r = 0.394$, $p = 0.016$) and IL-6 ($r = 0.513$, $p = 0.001$), indicating that higher levels of these markers are associated with longer exposure to smoking¹³.

Conclusion

Smoking has a profound effect on the body, as seen by the notable variations in systolic blood pressure, hemoglobin, PCV, ACE-2, and IL-6 levels between smokers and non-smokers. The dangers of continuing to smoke are further highlighted by the positive associations found between smoking frequency and duration with these characteristics. These findings support the importance of smoking cessation therapies and add to the increasing amount of data that shows smoking is associated with negative health outcomes [14, 15].

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