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Article

The impact of active and passive smoking on IL-6 serum levels for Iraqi healthy people

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ABSTRACT

Cigarette smoking is responsible for respiratory tract disorder by inducing the inflammation response. Interleukin-6 is an essential inflammatory interleukine produced by macrophages and epithelial cells of the airways. Due to the critical gap in our knowledge about the secretion of IL-6 by healthy smokers, this study aimed to evaluate the serum level of interleukin-6 for healthy people due to active and passive smoking and compared them with healthy non-smokers. The research population included (49=n) healthy males aged from 22 to 28 years, classified into 3 groups: non-smokers (n =10), active smokers (n =29), and passive smokers (n =10). An ELISA kit(Peprotech Company) (USA) was used to quantify interleukin-6 in serum levels. The result has displayed significantly increased mean IL-6 serum levels for active smokers (552.7 pg/ml) and passive smokers (614.9 pg/ml) in comparison with non-smokers (332.8 pg/ml) at levels (P <0.01). Both passive and active smoking induce the secretion of IL-6 for healthy smokers significantly higher than non-smokers. So, little contact with cigarette smoke or passive smoking raises the pro-inflammatory IL-6 and active smoking.

Keywords: Healthy active smoking, healthy passive smoking, IL-6

INTRODUCTION

Tobacco smoke is responsible for many diseases and mortality worldwide [1]. Tobacco Smoke has a wide range of toxic components [1], which is responsible for inducing abnormalities in the immune response of smokers, excluding the asymptomatic healthy smokers [2]. The term passive smoker refers to non-smoker people in contact with environments with tobacco burn [1], sometimes termed secondhand smoking [3]. Active and passive smoking are the most vital reasons for many life-threatening diseases. [4]. Smoke delivered over the filter [5]. Shiyia et al. mention that passive smoking significantly increases the threat of health problems, specifically children's disease and cancer [6]; inhaling CS by non-smokers is major health trouble for about 40% of children and 34% of adults worldwide [6]. Epidemiological research refers to the association between exposure to (CS) and susceptibility to respiratory disease [6], specifically among workers in the hospital, employees on flights, and children with parents who smoke [7]. Cigarette smoke affects the immune system [1,8], which acts on alterations

in the innate immunity and antigen presentation, initiating Autoimmunity and immune dysfunction [9]; this health disorder is associated with both types of smoking, active and passive [4]. Wherever the smoke of cigarettes enflames and destroys the respiratory tract [10,11], IL-6 is a critical pro-inflammatory cytokine produced by monocytes, macrophages, and the epithelial cells of the airway [10]. In addition to TNF-alpha and IL-1, IL-6 has been established as a critical marker of pro-inflammatory cytokines, where noticed in a variety of inflammatory disorders elevated the serum levels of IL-6 [10,12]. The inhalation of a low level of tobacco smoking or passive smoking alters the lung cell biology similarly to that of a current smoker [13]. A prior study confirmed the induction of inflammatory response and numerous physiological alterations in the lung airway for passive smoking [PS] [14,15], the recognition of a specific mechanism by which tobacco Smoke induces disease due to the affected host immunity, so this recognizes unique therapeutic approaches for the controlling the smoker disease. The effect of contact with environmental cigarette smoke on respiratory health has received much attention for several years [16,17]; this phenomenon is not restricted to the patients but includes healthy smokers [2]. Many studies have detected pathological changes in healthy smokers. 2 Due to the critical effect of cigarette smoke on the inflammation and immunity of healthy active and passive smokers. The current study will chiefly evaluate the alteration of inflammation cytokine IL-6 serum levels for active and passive healthy smokers.

MATERIALS AND METHODS

According to the past article, contact with passive smoking for one hour is sufficient to increase significantly the inflammation response [18]. This study comprised (n=49) healthy men aged 22 to 28 years. Classified into 3 groups: the first group was non-smokers (n =10), the second group was active smokers (n =29), and the third group was non-smokers who inhaled cigarette smoke in the home and workplace for more than 10 hours(n =10) during the 3 past years. All three Study groups were healthy, without any history of drug or alcohol abuse or history of hospitalization in the recent year.

IL-6 measurement

Interleukin-6 was measured using an ELISA kit from Peprotech Company (USA). Five ml of the vein blood sample from all group members was kept in a vacuum tube containing a clot activator. To separate, the serum was centrifuged in the tube at 1500g for a quarter-hour and stored at -20°C. The levels of IL-6 were determined according to a kit Peprotech Company procedure.

Statistical analysis

The differences between mean values were determined by using SPSS software version 14 (SPSS) for statistical analysis one-way ANOVA; the result data was documented as (mean±SD) and used one-way ANOVA test to compare the serum level data for active and passive smokers with non-smokers, this data calculation recorded significant differences at the level of the p-value<(0.01).

RESULTS

This study includes 49 men participating, ages between 20 and 28 years. They are categorized into 3 groups (non-smokers, passive and active smokers). The result of ELISA displayed the mean of IL-6 serum levels (pg/ml) ±SD non-smoker (332.8±91.03), passive smoker(614.9±218.3), and active-smoker (552.7±242.7), Table (1) explained high significantly increased in IL-6 serum level for passive and active-smoker in comparison with non-smoker group at ($P < 0.01$).

GROUPS	No.	Median (pg/ml) ±SD	(range) (pg/ml)	P value
				Compare with non-smoker
non-smoker	10	332.8±91.03	111.4-421.05	1
passive-smoker	10	614.9±218.3	435.6-1019.5	0.001**
active -smoker	29	552.7±242.7	246.5-1172.2	0.008**
****= p-value<0.01**				

Table 1. Describe the comparison of the mean IL-6 serum level between smokers (active and passive) and non-smokers.

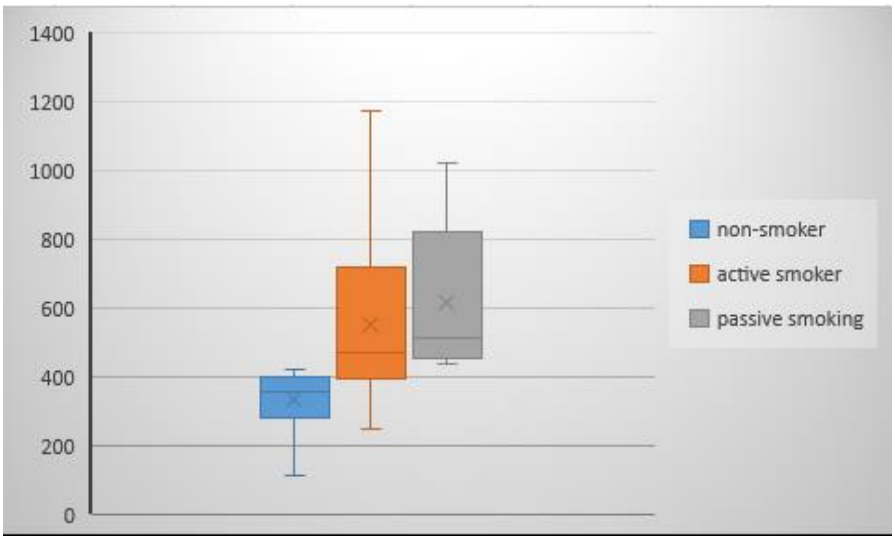

Figure 1. Explain the comparison of IL-6 serum levels among 3 study groups (non-smokers, active smokers, and passive smokers).

Whereas the Correlation coefficient between the period of active smoking and Il-6 serum concentration is a positive relationship but is non-significant (P-value = 0.8) as described in Table (2) below

Parameters	Correlation coefficient (r)	Level sig.
IL-6& smoking years	0.059	**0.8 NS**
NS: Non-significant		

Table 2. Describe the correlation coefficient between il-6 serum level and smoking period.

When analyzing The effect of the smoking period on the IL-6 serum level, it is made clear that the increase in IL-6 serum level with an increased period of smoking in the groups 1-3 years and 3-5 years of active smoking significantly increased at (p-value<0.05), where the more than 5 years of active smoking high significantly increased at (p <0.01), that is mean the concentration of IL-6 signif-

icantly increased to all periods of smoking. In contrast, passive smoking increased the IL-6 at a higher significant raise ($p<0.001$) than in another group study, as shown in Table 3.

GROUPS	No.	Median (pg/ml) ±SD	(range) (pg/ml)	P-value
				Compare with non-smoker
1-3 years of active smoking	12	562.4±245	243.4-1172.2	0.01*
3-5 years of active smoking	7	554.7±255.6	270.24-998.32	0.02*
more than 5 years of active smoking	10	607.9±225.1	353.84-998.32	0.002**
passive-smoker	10	614.9±218.3	435.6-1019.5	0.001**
non-smoker	10	332.8±91.03	111.4-421.05	1
(* =p-value<0.05),(= p-value<0.01)**				

Table 3. Describe the IL-6 serum level according to the period of smoking and passive smoking in comparison with the non-smoker group.

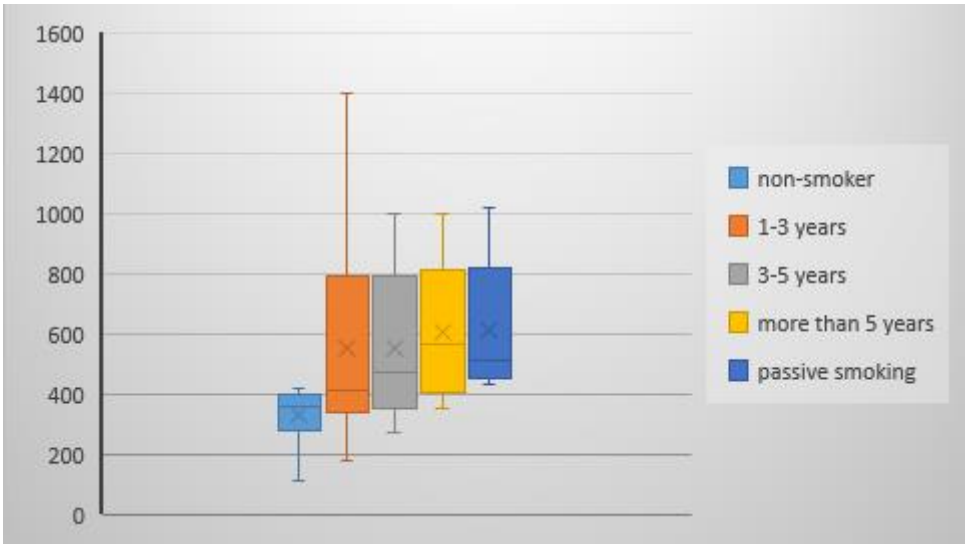

Figure 2. Explain the IL-6 serum level for study groups

DISCUSSION

More than 4500 chemicals material known as toxic, mutagenic, and carcinogenic effects have been contained in cigarette smoke [2]. The smoker and individuals nearby have inhaled this toxic material 19. Nonetheless, there is little research dealing with abnormalities induced by smoking for people known as healthy smokers who have a normal physical examination and are asymptomatic and may be considered a healthy control group in many research studies; this study was designed to assess the effect of both type of smoking (active &passive) on the pro-inflammatory IL-6 serum level as a critical cytokine for inflammation re-

sponse, which is elevated in inflammatory disease in addition to the IL-1β and TNF-α cytokines [10]. The result clarified the significant increase in the IL-6 serum level for the active and passive smokers in comparison with the non-smokers at ($p < 0.01$). This agreed with previous studies, which indicate that little contact with cigarette smoke or passive smoking is responsible for an alteration in the cell lung biology in a similar way that occurs with active smoking [13]; other studies presented a positive association between smoking and IL-6 serum level, Wu and his group noticed the stimulation by tobacco smoke extract increased IL-6 and IL-8 production by mononuclear cells of chronic obstructive pulmonary disease (COPD) [20]. where Herfs et al remarked the respiratory epithelium cell for smokers elevated the expression of IL-6 compared with non-smokers [21], and so the same result published by Moretti et al, where mentioned that smokers' IL-6 serum levels were higher than non-smokers at level ($p = 0.01$) [22], another study explained the contact to cigarette smoking (CS)for a long time increased the level of inflammatory cytokines IL-17Aand IL-6 in the lung in addition to defect the innate and adaptive response to infectious disease [14], cigarette smoking significantly stimuli inflammation by modifications the signaling pathway [23], so increase neutrophils, dendritic and macrophage cells numbers in the airway for smokers and animals in contact with smoking several-fold than non-somking [24], which are acting on the increased secretion of IL-6 due to the release of proteolytic enzymes and generate oxidants acting on tissue damage and induce immune reactions [25], other studies record the exposure to [CS] modifies T-cell responses to the chronic inflammation by altering the IL-17 signaling axis that leading to severe pulmonary damage [22], the third research published that exposure to CS lead to oxidative stress due to the imbalance between oxidant and antioxidants, which act on the increasing the mucosal inflammation and elevated the inflammatory interleukine-6, interleukine-8 and TNF-α [26]. Table 2 clarifies The non-significant correlation between the active smoking periods group and the IL-6 serum level; this result confirms other research on the fact that a short period of smoking or a low level of Cigarette smoking plays a role in the activation of the inflammatory cells and linked with signs of systemic inflammation [13]. In contrast, Table 3 refers to a significant increase in IL-6 serum levels for all study groups compared to a non-smoker group. The result approves the idea that short contact with CS is enough to proliferate the IL-6 secretion compared to non-smokers; our result agrees with other studies that confirm that even short exposure to CS increases the pro-inflammatory cytokines [27,28].

CONCLUSION

Our results show that exposure to cigarette smoke, either passive or active smoking, induces a higher secretion of IL-6 in smokers than non-smokers. So, little contact with cigarette smoke or passive smoking, raising the inflammatory interleukine-6 as well as active smoking, does. So, an assumption is to avoid healthy smokers, either passive or active, in a study as the control group.

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