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Serum creatinine and uric acid levels in adult male smokers versus nonsmokers

Noran Jameel Ibraheem¹, Ghadeer Hamid AL-Ardhi^{2*}

Department of Biology, Faculty of Science for women, University of Babylon Al- Hila, Iraq¹ Department of biology, DNA Research centre, University of Babylon, Al- Hila, Iraq²

Correspondence author: 2*



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ABSTRACT

We performed a prospective study to evaluate the possible effect of smoking on oxidative stress subsequently on kidney working. This study included a smoker and nonsmoker cohort of 140 healthy men, all volunteers subjects aged 25 to 45 years free renal disease and another disease, with 65 of them being nonsmokers and the other being cigarette smokers. Blood specimen was collected to estimate serum uric acid and creatinine levels. There was a significant decrease in uric acid while there was a non-significant difference in creatinine level in the smoker group compared with nonsmoker between the two groups. Both creatinine and uric acid concentration were affected by the duration of smoking compared with the control group. Smoking leads to decrease uric acid, which acts as a valuable antioxidant against oxidative stress, but creatinine levels were independently associated with smokers compared with nonsmoker males.



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1. Introduction

Many things can stimulate stress between people, like a natural body response; various research pieces have considered that exposure to smoking can trigger stress, and prolonged stress worsens or increases the risk of inappropriate renal function and developing kidney disease [17]. The kidney is a vital target organ of smoking-induced damage [18] there are harmful substances found in tobacco smoke, as nicotine is one of these substances that may be acquired through active and passive smoking [3] cigarette smoking is a well-known risk factor for atherosclerosis development through vascular endothelial damage that possibly occurs through oxygen-free radicals production as superoxide radicals and hydroxyl radicals [10] cigarette smoking has increased inflammatory responses that further enhance their oxidative stress [19].

Uric acid is a degradation production from nucleic acids. The final results of the purine oxidation process act as a valuable antioxidant, including against oxidative stress caused by chronic tobacco smoking [7], [6].

Uric acid is the most abundant aqueous antioxidant, accounting for up to 60% of serum-free radical scavenging capacity [1] and is an essential intracellular free radical scavenger during metabolic stress, including smoking [16] therefore, measurement of its serum level reflects the antioxidant capacity.

Creatinine is a waste product made by muscles; it passes into the bloodstream and is usually passed out in the urine [5]. Creatinine concentration was taken as an indication for renal function, so its level in blood is characterized as a valuable guide to renal function state as reported by the [21].

The present study aimed to characterize how current and passive smoking influences stress may impact serum uric acid and creatinine levels.

2. Subjects and Methods

This study was conducted from November to May 2021 in a science college for women at Babylon University. The subjects enrolled in this study were 140 male volunteers aged 25-45 years divided into groups (group I and group II).

Group I, considered a control group, comprised 65 seemingly healthy nonsmokers, ranging from 25 to 45 years.

Group II, considered a smoker group composed of 75 cigarette smokers, shows healthy ages ranging from 25 to 45 years.

A complete history record was obtained, including name, age, average number of daily cigarettes smoked daily (10 cigarettes per day), duration of smoking (1-15 years), dietary habit, past medical and drug history.

Blood samples were obtained from both groups for colourimetric measurement of uric acid and creatinine that were done by Jaffes reaction kinetic method [11] at the biochemistry laboratory of a college of science for women.

The results were expressed as means \pm standard deviation (S.D), for computation uric acid and creatinine level between control and study groups, an independent sample t-test was used, while comparison between smoker and nonsmoker to same parameters in regarded with smoker duration analysis of variance (Dunnett,s test) was applied.

A p-value equal to or less than (0.05) represents a statistically significant difference.

3. The results and discussion

As shown in (table 1), no significant statistical differences in serum creatinine between groups (P>0.05), whereas serum uric acid was significantly lower in the smoking group compared with the control group [p<0.05].

On the other hand, the duration of smoking significantly changes serum uric acid and creatinine concentration, as revealed in (table 2), since a significant increased (p<0.05) was noted in creatinine level was linked with increment smoking duration, while a significant reduction (p<0.05) in uric acid concentration concern with an excess smoking period.

Table 1: Creatinine and uric acid level in smoking men as compared with nonsmoking

Parameters	Mean ± SD	NO	Subjects	Sig
Creatinine(mg\dl)	0.94 ± 0.57	75	smoker	P > 0.05
	0.72 ± 0.40	65	nonsmoker	
Uric acid(mg\dl)	2.12 ± 0.73	75	smoker	P < 0.05
	4.14 ± 1.72	65	nonsmoker	

Table 2: Effect of duration time of smoking on the level of uric acid and creatinine as compared with the



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control group

variables	Duration time	Control		
	1-5yrs 6-10y	rs 11-15yrs		(65)
	A. (25) B.	(26) D.(24)		
Creatinine				
Mg\dl	0.97 ± 0.6	2.1±0.8†	3.55±0.9*	$\boldsymbol{0.72 \pm 0.40}$
Uric acid				
Mg\dl	3.3±1.3	2.9±0.6	1.6±0.3**	4.1±1.7

Mean as expressed as mean \pm SD,p*<0.05 vs (A, B and control group),p*<0.05 vs (A and control group),p†<0.05vs.(A and control group)

The decreasing effect of smoking on serum uric acid was also consistently observed with another study that showed low serum uric acid in regular smokers [7] however, smoking was closely associated with uric acid in females but not in male subjects in Korean population [12].

Cigarette smoke contains harmful substances such as Reactive oxygen species, toxin material, and nicotine, subsequently generating oxidative stress [22], so lower serum uric during smoking may be due to antioxidant action for Reactive oxygen species and free radicals produced by smoking [20] however, uric acid has a potential therapeutic role as an antioxidant [12].

Based on these mechanisms, a dose-dependent effect of smoking on uric acid indicates that oxidative stress increases every cigarette smoked [8]. Another study attributed that antioxidant decreased in current smokers increases susceptibility to oxidative damage after exposure to smoke for five minutes [20]. accordingly, smoking duration was inversely associated with uric acid in smokers as the previous study indicated [9] therefore, high serum uric acid might be protective in situations characterized by increased cardiovascular risk and oxidative stress as smoking, and by reducing its level, it increases susceptibility to oxidative damage and accounts for the excessive free radical production [14].

This suggests that smoking stress and renal function deficiency may be related, this may be a reason for creatinine level increases together with more smoking periods; there was a significant increase in creatinine level from five years until 15 years of smoking, this result may be explained that excess cigarettes substances accumulation can cause severe effects in renal tubules pH and lead to change in glomerular filtration rate [15].

This finding confirms study approached the relationship between smoking and creatinine level in which authors found creatinine levels significantly higher in active smokers [4] besides this, most responders had normal stress levels, the increase in stress hormone has a significant effect on the balance function of the body [13], as a stress hormone-like(adrenaline and cortisol)will interfere with the filtration of kidney and decrease of glomerular filtration rate will lead to a decrease in distal tubular flow rate which leads to increase in creatinine reabsorption [2].

Finally, with more periods of smoking, the elevated value of creatinine has been shown; this finding is in agreement with the previous study [4].

4. Conclusion

Smoking is a more incredible source for oxidation stress, and reduced uric acid was attributed to reducing

endogenous production being influenced adversely in glomerular filtration rate, afterwards elevation in serum creatinine.

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