



RESEARCH ARTICLE

BIOCHEMISTRY

**EFFECT OF CIGARETTE SMOKING ON BLOOD SODIUM AND POTASSIUM LEVELS IN SUDANESE SUBJECTS****EMAD-ALDIN I. OSMAN<sup>\*1</sup>, ABDULLAH A. DAFALLAH<sup>2</sup>, WALEED H. OMER<sup>3</sup> AND GAMAL M. ELIMIRI<sup>4</sup>**

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**ABSTRACT**

This study aimed to investigate the effect of cigarette smoking on plasma sodium and potassium levels. Atomic emission flame spectrophotometry was used to measure plasma sodium and potassium in sixty subjects, thirty smokers and thirty non-smokers as matched controls. The plasma sodium and potassium were analyzed before and after smoking in the smokers, while the analysis was done once in non-smoker cohort. There was a significant increase in sodium level immediately after smoking among smokers ( $p < 0.05$ ) and no significant change in potassium level. No change was found in plasma sodium level between smokers (before smoking) and non-smokers. In addition, there is a decrease in plasma potassium level among smokers (before smoking) in comparison to non-smokers; however, it is statistically not significant. This study reveals that cigarette smoking has an immediate increasing effect on plasma sodium level. It is recommended that cigarette smoking should be avoided especially before blood sodium measurement.



## KEYWORDS

Sudan, Shendi, Electrolytes, smokers, sodium, potassium.

## INTRODUCTION

Cigarette smoke contains over 4000 different chemicals, 400 of which are proven carcinogens; it also contains various oxidants, volatile aldehydes<sup>1</sup>, polycyclic aromatic hydrocarbons, nitroso compounds and aromatic amines<sup>2,3</sup>. Smokers are at greater risk of cardiovascular diseases, respiratory disorders, cancer, peptic ulcers and gastroesophageal reflux disease<sup>4,5</sup>. There is now ample evidence that tobacco smoking causes endocrine changes in humans. Many studies have demonstrated that exposure to cigarette smoke produces marked neuroendocrine changes. The effects of nicotine on the hypothalamic–pituitary axis (HPA) were first investigated by Balfour<sup>6</sup>, who showed that nicotine was a potent activator of the HPA. Cigarette smoking also causes an acute increase in circulating levels of Adrenocorticotrophic hormone (ACTH); intense smoking is necessary to induce these changes<sup>7,8</sup>. In contrast to the acute effect of smoking on ACTH levels, the latter are not altered in chronic smokers<sup>9</sup>. There is also extensive evidence that cigarette smoking acutely increases circulating cortisol levels<sup>7,10,11</sup>.

Sodium is the major cation of extracellular fluid. Because it represents approximately 90% of inorganic cations in plasma, Sodium is responsible for almost one half of the osmotic strength of plasma. It therefore has a central function in maintaining the normal distribution of water and osmotic pressure in the extracellular fluid. Potassium is the major intracellular cation. It is filtered through the glomeruli and almost completely reabsorbed in the proximal tubules and is secreted in the distal tubules in exchange for Sodium under the influence of aldosterone<sup>12</sup>. Plasma potassium values exhibit a circadian rhythm (average peak-to-trough difference approximately 0.60 mmol/L, with lowest values

at night)<sup>13,14</sup> and also decrease postprandially because of insulin released in response to an ingested carbohydrate load<sup>15</sup>. Potassium is critical to the maintenance of cardiovascular health and the normokalemic state is vital to the prevention of potentially serious sequelae, especially in the at-risk cardiovascular patient. The hypokalemic state is highly arrhythmogenic, and may contribute to arrhythmic deaths in cardiac patients. In contrast, hyperkalemia causes slowed conduction and conduction block which, if sufficiently progressive, can result in asystole<sup>15,16</sup>.

## MATERIAL AND METHODS

Sixty healthy Sudanese volunteers aged between 18 and 45 years, residing in Shendi city, Sudan, taking local diet were chosen for the study. Thirty of them, were smokers (29 male and 1 female), the remaining 30 were non-smokers control group (sex and age matched). All the smokers were using a locally manufactured cigarette.

All the subjects signed informed consents and filled questionnaires. Institutional ethical committee approved this study.

All subjects were fasted overnight and morning venous blood samples were taken in lithium heparin containers and plasma was separated within half an hour of collection. Each subject of the smokers group was given a cigarette and another venous blood sample was taken after half an hour of finishing the cigarette.

Determination of plasma Sodium (Na<sup>+</sup>) and Potassium (K<sup>+</sup>) levels

The plasma samples were analyzed by Atomic Emission Flame Spectrophotometer (Flame Photometer) using standard procedures for determination of Sodium (Na<sup>+</sup>) and potassium (K<sup>+</sup>).



### Statistical analysis

Two tailed paired student's t-test was performed to find a significant difference between means of the groups, while Chi square method was used for categorical variables. *P*-value was considered significant when it less than or equal 0.05.

## RESULTS AND DISCUSSION

The means of sodium and potassium levels for smokers before and after smoking and for non-smokers group is shown in table 1.

**Table 1**

**Comparison of plasma sodium and potassium between smokers (before and after smoking) and non-smokers controls**

Parameter	Cigarette smokers		Non-smokers (control)
	Before smoking	After smoking	
Sodium mmol/L	140.1±1.35	143.9±1.68	140.2±1.32
Potassium mmol/L	3.6±0.72	3.65±0.41	3.94±0.26

*Values are means ± S.D. of 30 healthy subjects in each group. Students's t-test was performed to find significant difference among groups.*

In this study we found a significant increase in sodium plasma levels and no significant alteration in potassium plasma levels among smokers after smoking in comparison to the pre-smoking levels. No difference was found between plasma sodium levels in smokers (before smoking) and non-smokers. Moreover, there was a decrease in Plasma potassium levels among smokers in comparison to non-smoker, however it is statistically insignificant. Cigarette smoke consists of many chemicals, including nicotine, tar and many carcinogens and gaseous compounds<sup>8</sup>, some of these compounds have neuroendocrine effects. In this study, the acute and chronic effect of cigarette smoking on plasma sodium and potassium levels was investigated in 30 smoking subjects before and after smoking and 30 non-smoking control subjects. Plasma sodium level was significantly increased among smokers immediately after smoking and this may be due to stimulation of adrenal cortex, which leads to increase of circulatory cortisol<sup>17</sup> that increase sodium retention and urinary potassium loss and also increase the glomerular filtration rate (GFR). In addition, cigarette smoke stimulates the adrenal medulla to secrete adrenaline that increases the GFR via its action on blood vessels and also may

facilitate pituitary ACTH secretion<sup>18</sup>. However, No significant change was found on plasma sodium levels between smokers (before smoking) and non-smokers and this is in agreement with another study<sup>19</sup>. Moreover, 27% of smokers were felt thirsty immediately after smoking; significant correlation ( $p < 0.05$ ) was found between the increased plasma sodium level after smoking and polydipsia. The immediate increase in sodium level stimulates the thirst centre and so promote oral fluid intake, minimizing changes in plasma sodium concentration on long-term basis.

In this study, there is a decrease in plasma potassium level among smokers (before smoking) in comparison to non smokers; however, it is statistically insignificant. Cigarette smoke contains glycyrrhizinic acid that mimics aldosterone action which increases urinary potassium excretion<sup>20</sup>. However, this result is contrast to a study<sup>19</sup> which found an elevated plasma potassium level in chronic smokers in comparison with non smokers and this may be due to the method used in that study. The insignificant decrease in plasma potassium level among the smokers group in this study may be attributed to the continuous repletion of potassium ions from intracellular compartment which contributes to correction of



plasma potassium level. Longstanding intracellular potassium depletion causes extracellular alkalosis. Prolonged potassium depletion impairs the renal concentrating mechanism and may cause polyuria with potassium depletion<sup>20</sup>.

In conclusion, cigarette smoking has an acute increasing effect on plasma sodium level which

may lead to misinterpretation of blood sodium test results. It's recommended that chronic influence of cigarette smoking on plasma potassium level would be investigated in a larger number of samples with the measurement of plasma bicarbonate level as well.

## REFERENCES

1. Yeh, C.C.; Graham Barr, R.; Powell, C.A.; Mesia-Vela, S.; Wang, Y.; Hamade, N.K.; Austin, J.H.; and Santella, R.M, No effect of cigarette smoking dose on oxidized plasma proteins. *Environ Res*, 106(2):219-25, (2008).
2. Utiger, R.D, Effects of smoking on thyroid function. *Eur J Endocrinol*, 138(4):368-9, (1998).
3. Shiverick, K.T., and Salafia, C, Cigarette smoking and pregnancy I: ovarian, uterine and placental effects. *Placenta*, 20(4):265-72, (1999).
4. Witschi, H, A short history of lung cancer. *Toxicol Sci*, 64(1):4-6, (2001).
5. Spiro, S.G., and Silvestri, G.A, One hundred years of lung cancer. *Am J Respir Crit Care Med*, 172(5):523-9, (2005).
6. Balfour, D.J, Influence of nicotine on the release of monoamines in the brain. *Prog Brain Res*, 79:165-72, (1989).
7. Seyler, L.E., Jr.; Fertig, J.; Pomerleau, O.; Hunt, D.; and Parker, K, The effects of smoking on ACTH and cortisol secretion. *Life Sci*, 34(1):57-65, (1984).
8. Pomerleau, O.F, Nicotine and the central nervous system: biobehavioral effects of cigarette smoking. *Am J Med*, 93(1A):2S-7S, (1992).
9. del Arbol, J.L.; Munoz, J.R.; Ojeda, L.; Cascales, A.L.; Irlles, J.R.; Miranda, M.T.; Ruiz Requena, M.E.; and Aguirre, J.C, Plasma concentrations of beta-endorphin in smokers who consume different numbers of cigarettes per day. *Pharmacol Biochem Behav*, 67(1):25-8, (2000).
10. Winternitz, W.W., and Quillen, D, Acute hormonal response to cigarette smoking. *J Clin Pharmacol*, 17(7):389-97, (1977).
11. Wilkins, J.N.; Carlson, H.E.; Van Vunakis, H.; Hill, M.A.; Gritz, E.; and Jarvik, M.E, Nicotine from cigarette smoking increases circulating levels of cortisol, growth hormone, and prolactin in male chronic smokers. *Psychopharmacology (Berl)*, 78(4):305-8, (1982).
12. Carl A. Burtis, Edward R. Ashwood, David E. Bruns, Barbara G. Sawyer. *Tietz Fundamentals of Clinical Chemistry*, 6<sup>th</sup> Edn, Saunders, an imprint of Elsevier Inc: 432-433, (2008).
13. Kawano, Y.; Minami, J.; Takishita, S.; and Omae, T, Effects of potassium supplementation on office, home, and 24-h blood pressure in patients with essential hypertension. *Am J Hypertens*, 11(10):1141-6, (1998).
14. Solomon, R.; Weinberg, M.S.; and Dubey, A, The diurnal rhythm of plasma potassium: relationship to diuretic therapy. *J Cardiovasc Pharmacol*, 17(5):854-9, (1991).
15. Sica, D.A.; Struthers, A.D.; Cushman, W.C.; Wood, M.; Banas, J.S., Jr.; and Epstein, M, Importance of potassium in cardiovascular disease. *J Clin Hypertens (Greenwich)*, 4(3):198-206, (2002).
16. Yang, T., and Roden, D.M, Extracellular potassium modulation of drug block of IKr. Implications for torsade de pointes



- and reverse use-dependence. *Circulation*, 93(3):407-11, (1996).
17. Tziomalos, K., and Charsoulis, F, Endocrine effects of tobacco smoking. *Clin Endocrinol (Oxf)*, 61(6):664-74, (2004).
  18. T.D. Reisine, E, Mezey, M. Palkovits, S. Heisler and J. Axelrod, Beta adrenergic control of adrenocorticotrophic hormone release from the anterior pituitary. In: E. Usdin, A. Carlsson, A. Dahlstrom and J. Engel (eds), *Catecholamines: Neuropharmacology and Central Nervous System Theoretical Aspects*, Alan R. Liss, New York, 1984, pp. 419–423.
  19. Pannuru Padmavathi, Vaddi D. Reddy and Nallanchakravarthula Varadacharyulu, Influence of chronic cigarette smoking on serum biochemical profile in male human volunteers, *J health Sci*, 55(2): 265-270, (2009)
  20. Philip D. Mayne, *Clinical Chemistry in Diagnosis and Treatment*, 6<sup>th</sup> Edn, ELBS Publication: p 63-66, (1994)