INT. J. BIOL. BIOTECH., 8 (3): 437-440, 2011.

SOME BIOCHEMICAL ALTERATIONS IN OBESE CIGARETTE SMOKERS

Waheeb D. M. Alharbi

Department of Physiology, Faculty of Medicine, Umm Al-Qura University, Makkah, Saudi Arabia.
E.Mail: wdharbi@uqu.edu.sa

ABSTRACT

Obesity is a complex and multifactorial disorder, and the association between obesity and smoking has extensively been studied. A variety of the aspects of electrolytes involved in obesity, smoking and combined condition of both have been thoroughly studied. However, it is still controversial to explain the influence of electrolytes in smoking and obesity. The influence of cigarette smoking on electrolytes in obese subjects was studied in a group of obese smoking subjects and compared with those who were neither smokers, nor obese. Statistical comparisons showed significant variations for plasma sodium, potassium and calcium. Fasting glucose concentration in control and obese smokers showed significantly higher percentage of obese smokers with impaired glucose levels. All subjects in control group (non-obese non-smoking controls) showed normal level of fasting glucose. However, further studies are required for elucidating the precise mechanism underlying alterations caused by smoking in obesity.

Key Words: Cigarette smoking, obesity, electrolytes, glucose

INTRODUCTION

Obesity is a complex and multifactorial disorder, and the association between obesity and smoking has extensively been studied. A variety of studies have been carried out to explain the influence of smoking and quitting smoking on body weight in obese smokers (Lycett *et al*., 2010). As weight gain is generally expected to occur after quitting smoking, the decision for quitting smoking becomes more difficult. Higher weight gain occurs in younger ages, and lower socio-economic status (Filozof *et al*., 2004). Tobacco smoking and obesity are considered worldwide as serious health problems in adolescent and young adults (Lamota *et al*., 2007).

Influence of smoking on body weight has been studied by various investigators (Yoshida *et al*., 1990; Iwashima *et al*., 2005; John *et al*., 2005; Vander *et al*., 2008; Xie *et al*., 2010; Beyerlein *el al*., 2011). However, little is known whether obesity alters the risk of fluid and sodium imbalances (Verhave *et al*., 2004; Eijsvogels *et al*., 2011). Elevated plasma solutes mainly sodium were found in overweight and obese subjects (Stookey *et al*., 2007). Furthermore, plasma sodium levels did not change in lean subjects after exercise, whereas plasma sodium increased significantly in overweight and obese subjects (Eijsvogels *et al*., 2011). The obese children had higher intracellular sodium, and lower plasma sodium (Flodmark *et al*., 1992). This alarms that the increase of intracellular sodium may increase the risk of future hypertensive complications.

Role of potassium in smoking and obesity has been investigated in several studies (Edmonds *et al*., 1975; Pahl *et al*., 1988; Salvadori *et al*., 2003). The increase of plasma potassium was less in obesity than in control condition (Salvadori *et al*., 2003). During physical exercise K(+) changes significantly in obese subjects vs normals. In the normal population, total body potassium was significantly correlated with height as well as with weight (Edmonds *et al*., 1975).

Low-calcium intake did not cause obesity and did not show its involvement in raising the body weight set-point (Paradis and Cabanac, 2005). The results reveal that calcium intake probably does not have role in affecting the body weight. Abnormal calcium metabolism is concerned in morbidly obese patients compared to the non-obese, but the clinical value of this is not known (Hamoui *et al*., 2004). The ratio of total serum calcium to plasma ionized calcium (Ca2+) was noted to be significantly correlated to BMI and w/h as well (Lind *et al*., 1993). It was noted that total plasma calcium was higher in the obese subjects (Foldes *et al*., 1992). In contrast to human obesity, obese rats were found hypercalciuric. Calcium intake was found statistically significant correlated with glucose levels in women and men, but not with lipids levels (Pachocka *et al*., 2007).

While studying the Impact of chronic cigarette smoking on the composition of the body and fuel metabolism, plasma glucose was found higher in smokers than in nonsmokers (Jensen et al., 1995). Smoking was found involved in metabolic syndrome (MetS) (Lamota *et al*., 2007; Takeuchi *et al*., 2009) and diabetes mellitus (Liu *et al*., 2001; Takeuchi *et al*., 2009). Glucose metabolism has been associated with the effects of nicotine, smoking and obesity (Jensen *et al*., 1995; Suzuki *el at*., 1996; Nakanishi *et al*., 2000; Liu *et al*., 2001; Lamota *et al*., 2007; Wang *et al*., 2011).

In view of discrepancies found in various reports for the involvement of blood levels of glucose, sodium, potassium and calcium in smokers having obesity, it was planned to carry out a basic study to evaluate the variations

of glucose and electrolytes in obese smokers, and to explain the role and pathophysiological interpretation of these variations.

MATERIALS AND METHODS

The subjects studied in the present study were the male obese smokers (n: 40) and male controls (n: 42). These subjects (obese smokers and controls) were consulted in Umm Al-Qura University and related institutions in Makkah, Saudi Arabia.

Age range of these subjects was 17-26 years (mean: 22.5 years). The obese smokers in this group were those smoking 3-5 cigarettes/ day with an average of 4 cigarettes/ day; and the average duration of the smoking habit was 2.0 years.

The details about the smoking habits, body weight and height, criteria for the evaluation of subjects with obesity, other physical/ physiological measurements and information in the form of a standard questionnaire, was employed before collecting blood for the estimation of plasma electrolytes (sodium, potassium and calcium) and glucose by standard methods in obese smokers and control (non-smoking non-obese male subjects). Each subject provided the details of his age, blood pressure, BBT, and other physical measurements. The criteria of the selection of obese smokers was that no one should have the medical complication such as hypertension, ischemic heart disease, stroke, diabetes or any such disorder. Hence, all male subjects included in the present study are the normal healthy subjects except that the study group comprised subjects with confirmed obesity.

The fasting blood glucose was determined in all subjects (control and obese smokers), and number of subjects were counted for specific range of glucose levels to understand the involvement of plasma glucose in obese smokers. Normal levels of plasma glucose were considered as: 80-100 mg/ dl, whereas impaired glucose level was 101- 130 mg/ dl. However, no any subject showed the fasting glucose concentration more than 130 mg/ dl or below 80 mg/ dl. The number of subjects in each group was converted into percentage for comparison purposes. The data was collected and analyzed statistically using student t test. The significance values (p) for various comparisons were found and the results were interpreted.

RESULTS AND DISCUSSION

The influence of cigarette smoking on electrolytes was studied in a group of obese smoking subjects and compared with those subjects that were neither smokers, nor obese.

Plasma electrolytes (mmol/L; sodium, potassium and calcium) respectively in this part of study were (142.68±2.58, 4.36±0.58 and 2.49±0.12) for control male subjects and (147.51±2.60, 4.92±0.56 and 2.10±0.120) in obese smoking male subjects. These results are presented in Table 1. Statistical comparisons showed significant variations for plasma sodium, potassium and calcium ($P < 0.0001$; Table.1).

Table 1. Plasma electrolytes in obese cigarette smokers.

Blood levels	Control (n: 42)	Obese smokers (n: 40)	Significance Level (p)
Sodium (mmol/L)	142.68 ± 2.58 SE = 0.398	147.51 ± 2.60 SE = 0.411	p< 0.0001
Potassium (mmol/L)	4.36 ± 0.58 SE = 0,089	4.92 ± 0.56 SE = 0.088	p< 0.0001
Calcium (mmol/L)	2.49 ± 0.12 SE = 0.018	2.10 ± 0.12 SE = 0.018	p< 0.0001

The values are mean ± SD; n: number of subjects; SE , Standard Error

Fasting glucose concentration (mg/dl) in control and obese smokers showed significantly higher percent of obese smokers with impaired glucose levels (Table 2). However, all subjects in control group (non-obese non-smoking controls) showed normal level of fasting glucose.

Association between adiponectin and smoking was found in men (Iwashima *et al*., 2005) that predicts the involvement of adverse change in smokers. Whereas, the impairment of the glucose tolerance and insulin sensitivity is another important aspect in smokers (Filozof *et al*., 2004). Efficacy of various lifestyle changes in smokers at increased risk for increased weight and cardio vascular events might be helpful (Vander *et al*., 2008). This part of

investigations also relates with the alteration in glucose metabolism obtained in the present study. Increased level of plasma glucose in obese smokers can be interpreted through several reports (Pahl *et al*., 1988; Jensen *et al*., 1995; Nakanishi *et al*., 2000; Liu *et al*., 2001; Pachocka *et al*., 2007). These studies emphasize the metabolic role and alterations in glucose concentrations which lead to alterations in body weight and health and wellness.

Table 2. Fasting glucose variations in obese smokers.

Fasting glucose levels (mg/ dL)	Controls (n:42)		Obese smokers (n:40)	
	#	%	#	%
Normal (80- 100)	42	100.00	22	55
Impaired (101-130)	0	0.00	18	45

Similarly, the results obtained by various authors for the role of electrolytes (Paradis and Cabanac, 2005; Stookey *et al*., 2007; Eijsvogels *et al*., 2011) in obesity and smoking might differ, but the basic mechanism wherein plasma sodium, potassium and calcium are involved have the specified significance and are considered important in the cause of obesity and consequences of cigarette smoking (Edmonds *et al*., 1975; Flodmark *et al*., 1992; Salvadori *et al*., 2003; Hamoui *et al*., 2004; Verhave *et al*., 2004; Paradis and Cabanac, 2005; Stookey *et al*., 2007; Eijsvogels *et al*., 2011). The current study hence, provides information for carrying out further related work to investigate and correlate the involvement of blood glucose and electrolytes in obese smokers.

REFERENCES

Beyerlein, A., S. Rückinger, A.M. Toschke, A. Schaffrath Rosario and R. von Kries (2011). Is low birth weight in the causal pathway of the association between maternal smoking in pregnancy and higher BMI in the offspring? *Eur. J. Epidemiol*., 26(5):413-20.

Edmonds, C.J., B.M. Jasani and T.Smith (1975). Total body potassium and body fat estimation in relationship to height, sex, age, malnutrition and obesity. *Clin.Sci.Mol. Med*., 48(5):431-40.

Eijsvogels, T.M., M.T. Veltmeijer, T.H. Schreuder, F. Poelkens, D.H. Thijssen and M.T. Hopman (2011). The impact of obesity on physiological responses during prolonged exercise. *Int. J. Obes* (Lond)., [Epub ahead of print].

Filozof, C., M.C. Fernández Pinilla and A. Fernández-Cruz (2004). Smoking cessation and weight gain.*Obes. Rev*., 5(2):95-103.

Foldes, J., M.S. Shih and J. Levy (1992). Bone structure and calcium metabolism in obese Zucker rats. *Int. J. Obes. Relat. Metab. Disord*., 16(2):95-102.

Flodmark, C.E., N.C. Henningsen and T. Sveger (1992). Red cell sodium-potassium adenosine triphosphatase sites and intracellular sodium increased in obese school children. *Miner Electrolyte Metab*.,18(1): 6-8.

Hamoui, N., G. Anthone, and P.F. Crookes (2004). Calcium metabolism in the morbidly obese. *Obes. Surg*.,14(1):9-12.

Iwashima, Y., T. Katsuya, K. Ishikawa, I. Kida, M. Ohishi, T. Horio, N. Ouchi, K. Ohashi, S. Kihara, T. Funahashi, H. Rakugi and T. Ogihara (2005). Association of hypoadiponectinemia with smoking habit in men. *Hypertension*., 45(6):1094-100.

John, U., M. Hanke, H.J. Rumpf, J.R. Thyrian (2005). Smoking status, cigarettes per day, and their relationship to overweight and obesity among former and current smokers in a national adult general population sample.*Int. J. Obes* (Lond)., 29(10):1289-94.

Jensen, E.X., C. Fusch, P. Jaeger, E. Peheim and F.F. Horber (1995). Impact of chronic cigarette smoking on body composition and fuel metabolism. *J. Clin. Endocrinol. Metab.*, 80(7):2181-5.

Lamota, L., F.J. Bermudez-Silva, E.M.Marco, R.Llorente, A.Gallego, F.Rodríguez de Fonseca and M.P. Viveros (2008). Effects of adolescent nicotine and SR 147778 (Surinabant) administration on food intake, somatic growth and metabolic parameters in rats. *Neuropharmacology*., 54(1):194-205.

Lind, L., H.Lithell, A. Hvarfner, T.Pollare and S. Ljunghall (1993). On the relationships between mineral metabolism, obesity and fat distribution. *Eur. J. Clin. Invest*., 23(5):307-10.

Liu, R.H., T. Kurose and S. Matsukura (2001). Oral nicotine administration decreases tumor necrosis factor- alpha expression in fat tissues in obese rats. *Metabolism*., 50(1):79-85.

Lycett, D., M. Munafò, E. Johnstone, M. Murphy and P. Aveyard (2011). Associations between weight change over 8 years and baseline body mass index in a cohort of continuing and quitting smokers. *Addiction*.,106(1): 188-96.

Nagaya, T., H.Yoshida, H.Takahashi and M.Kawai (2008). Heavy smoking raises risk for type 2 diabetes mellitus in obese men; but, light smoking reduces the risk in lean men: a follow-up study in Japan. *Ann. Epidemiol*., 18(2): 113-8.

Nakanishi, N., K.Nakamura, Y.Matsuo, K.Suzuki and K.Tatara (2000).Cigarette smoking and risk for impaired fasting glucose and type 2 diabetes in middle-aged Japanese men. *Ann Intern. Med*., 133(3): 183-91.

Pachocka, L.M., A.Nowak, U.Targosz, L.Kłosiewicz-Latoszek and I. Stolarska (2007). Calcium intake and glucose and lipids concetrations in overweight and obese patients. *Rocz. Panstw. Zakl. Hig.*, 58(1): 315-20.

Pahl, M.V., N.D. Vaziri, F. Akbarpour, A.Afrasiabi and R. Friis (1988). Effect of rapid weight loss with supplemented fasting on serum electrolytes, lipids, and blood pressure. *J. Natl. Med. Assoc*., 80(7): 803-9.

Paradis, S. and M. Cabanac (2005). Calcium deficiency cannot induce obesity in rats. *Physiol. Behav*., 85(3): 259-64.

Salvadori, A., P. Fanari, E. Giacomotti, P. Palmulli, G.Bolla, I. Tovaglieri, L. Luzi and E. Longhini (2003). Kinetics of catecholamines and potassium, and heart rate during exercise testing in obese subjects. Heart rate regulation in obesity during exercise. *Eur. J. Nutr*., 42(4): 181-7.

Stookey, J.D., D. Barclay, A. Arieff, and B.M. Popkin (2007). The altered fluid distribution in obesity may reflect plasma hypertonicity. *Eur. J. Clin. Nutr*., 61(2): 190-9.

Suzuki, H., H. Ikenaga, T.Hayashida, K.Otsuka, Y.Kanno, Y.Ohno, H.Ikeda and T. Saruta (1996). Sodium balance and hypertension in obese and fatty rats. *Kidney Int. Suppl*., 55: S150-3.

Takeuchi, T., M.Nakao, K. Nomura and E.Yano (2009). Association of metabolic syndrome with smoking and alcohol intake in Japanese men. *Nicotine Tob Res*., 11(9): 1093-8.

Vander Weg, M.W., R.C. Klesges, J.O.Ebbert, E.J. Lichty, M.DeBon, F.North, D.R. Schroeder and P.M. Dubbert (2008). Trial design: blood pressure control and weight gain prevention in prehypertensive and hypertensive smokers: the treatment and prevention study. Contemp. *Clin. Trials*., 29(2): 281-92.

Verhave, J.C., H.L. Hillege, J.G. Burgerhof, W.M. Janssen, R.T. Gansevoort, G.J. Navis, D. de Zeeuw and P.E. de Jong ; PREVEND Study Group (2004). Sodium intake affects urinary albumin excretion especially in overweight subjects. *J. Intern. Med*., 256(4): 324-30.

Wang, J.W., D.Y. Hu, Y.H. Sun, J.H. Wang and J.Xie (2011). Gender difference in association between smoking and metabolic risks among community adults]. *Zhonghua Yi Xue Za Zhi*., 91(12): 805-9.

Xie, B., P.H. Palmer, Z. Pang, P.Sun, H.Duan and C.A. Johnson (2010). Environmental tobacco use and indicators of metabolic syndrome in Chinese adults. *Nicotine Tob Res*., 12(3): 198- 206.

Yoshida, T., K.Yoshioka, N. Hiraoka and M. Kondo (1990). Effect of nicotine on norepinephrine turnover and thermogenesis in brown adipose tissue and metabolic rate in MSG obese mice. *J. Nutr. Sci. Vitaminol*. (Tokyo), 36(2): 123-30.

(*Accepted for publication June 2011*)