DOI: 10.21276/sjams

Scholars Journal of Applied Medical Sciences (SJAMS) **ISSN 2320-6691 (Online)**
Sch. J. App. Med. Sci., 2017; 5(5C):1879-1882 **ISSN 2347-954X (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublisher.com

Original Research Article

Study of insulin secretory response after oral glucose load in smokers

Ramesh Kumar Tilwani[1*], Khemlata Tilwani[2] , Priya Jangid[3], Madhurima Maheshwari[4]
[1]Senior Specialist Medicine, E S I Dispensary, Pali, Rajasthan, India
[2, 3] Assistant Professor, Department of Physiology S. N. Medical College, Jodhpur, Rajasthan, India
[4]Medical officer, Department of Physiology, S. N. Medical College, Jodhpur, Rajasthan, India

***Corresponding author**
Ramesh Kumar Tilwani
Email: dr_rtilwani@yahoo.co.in

Abstract: The objective is to study of insulin secretory response after oral glucose load in smokers. Study was conducted on normal BMI matched healthy 40 nonsmokers and 60 smokers (divided into 3 groups according to amount of smoking) of age group of 20-50 years. venous blood was drawn after an overnight fasting and fasting blood sugar level, at ½ hour,1 hour , 1 ½ hour and 2 hour after an oral glucose load of 75 gm and Serum insulin estimation (radio- immune assay method) After 75 gm oral glucose load at ½ hour,1 hour , 1 ½ hour and 2 hours interval are estimated. Smokers have relatively increased blood glucose levels and hyperinsulinemia as compared to non-smokers. Smoking is associated with relative glucose intolerance, hyperinsulinemia and a slower insulin response which is independent of age and BMI. The alteration is directly proportional to the amount of smoking.
Keywords: smokers, insulin secretary response, fasting blood sugar, OGTT

INTRODUCTION

Smoking is a worldwide public health problem. It not only substantially increases the risk of many disabling and potentially fatal disease but is the leading avoidable cause of death. Smoking increase the risk of ischemic heart disease(IHD), cerebro-vascular accident(CVA), acute dissecting aortic aneurysm , peripheral vascular disease(PVD), chronic obstructive pulmonary disease(COPD), acid peptic disease(APD) and malignancies like bronchogenic carcinoma , carcinoma of head and neck , urinary tract , pancreas etc. (US'DHHS , 1989).

The woman smokers are also at greater risk of developing IHD than non-smokers [1]. In a study of British male doctors the death rate for IHD among men under 60 years of age was 166/1,00,000 for non-smokers , 278 per 1,00,000 for those smoking 1 to 14 cigarettes per day and 427 per 1,00,000 for those smoking 25 or more cigarettes per day [2]. Study on women the relative risk of fatal IHD was 1.9 for those smoking 1 to 14 cigarettes per day , 4.3 for those smoking 14 to 25 cigarettes per day and 5.4 for those smoking more than 25 cigarettes per day [3]. The underlying mechanism as how smoking affects coronary architecture is not known with certainty (Criqui et al , 1980), and several mechanisms have been suggested including impaired blood coagulation (Mc Gill et al , 1979), impaired integrity of arterial wall (Topping, 1980) and changes in blood lipid and lipoprotein concentration (Freedman et al , 1986) .

The cigarette smoke contains more than 4,000 substances and the tissue and organ system responses to cigarette smoke inhalation are multiple and complex. Most studies in human beings have dealt with exposure to whole smoke or nicotine and carbon-monoxide which are thought to pose greatest risk to health [1]. The relation between many of the numerous compounds in cigarette smoke and anthropogenesis is not simple [4]. Carbon-monoxide produces hypoxia of arterial endothelium interfering its function as barrier to lipid infiltration of intima (Kannel, 1987). Nicotine increases work load and oxygen demand of myocardium by increasing pulse rate and blood pressure resulting in myocardial ischemia (Ball, 1974).

Nicotine stimulates release of adrenaline leading to increase in circulating levels of free fatty acids which are well known stimulant of hepatic

secretion of VLDL; hence triglycerides and serum HDL concentrations vary inversely with VLDL concentration (Criag et al, 1983). Since similar changes are also found in conditions consequent to resistance to insulin mediated glucose uptake or hyperinsulinemia (Dalefgky et al 1974, Tobey et al 1981 and Raven 1988) there may be a relationship between smoking and hyperinsulinemia. However it has been evaluated scarcely and the reports are scantly and conflicting. Hence the present study was conducted to explore any relationship between smoking and insulin metabolism.

MATERIAL AND METHODS

The present study was done in healthy male subjects of 20-50 years age; normal BMI matched healthy nonsmokers and 60 smokers. General physical examination of the subject including anthropometric measures such, as height, weight and Blood Pressure were taken.

The following study groups were made viz:

Group 1 (control group, n=40):- Healthy male subjects who either had never smoked.

Group 2 (smoker group, n=60) :- Healthy male subjects who were smoking 5 or more cigarettes and /or bidi per day for at least 5 years or more, at the time of study. These were further sub classified as:-

Group 2A (n=20):- Subjects smoking 5 to 10 cigarettes and / or bidi per day (Light Smokers).

Group 2B (n=20):- Subjects smoking 11 to 20 cigarettes and/or bidi per day (Moderate Smokers).

Group 2C (n=20):- Subjects smoking >20 cigarettes and/or bidi per day (Heavy Smokers).

Test done are-

a) Blood glucose (Methods of Folin and Wu ,1956)
   1. in fasting state
   2. at ½ hour,1 hour , 1 ½ hour and 2 hour after an oral glucose load of 75 gm.

b) Serum insulin estimation (radio- immune assay method)

After 75 gm oral glucose load at ½ hour, 1 hour , 1½ hour and 2 hours interval.

Time	**Non smokers (Gr I, n=40)**	**Light smokers (Gr II, n=20)**	**Moderate smokers (Gr I, n=20)**	**Heavy smokers** (Gr II, n=20)	**Total** (Gr II, n=60)
Fasting	72.5 ± 8.7	79.9 ± 5.3	86.8 ± 2.6	90.4 ± 4.6	85.8 ± 11.7
½ hour	95.0 ± 8.0	99.8 ± 10.5	105.6 ± 4.8	109.8 ± 4.7	105.0 ± 5.6
1 hour	105.6 ± 9.9	116.6± 3.8	124.4 ± 7.4	105.5 ± 4.7	122.1 ± 6.8
1½ hour	91.1 ± 10.1	100.4 ± 9.0	104.7 ± 5.1	106.5 ± 5.8	103.8 ± 6.4
2 hour	72.2 ± 6.2	79.6 ± 5.0	86.8 ± 5.4	89.9 ± 4.4	85.3 ± 6.6

OBSERVATION AND RESULT

Table 1: Oral glucose tolerance test in subjects studied

Statistical significance	Fasting	½ hour	1 hour	1½ hour	2 hour
Group I V/S Group IIA	P≤ 0.001	P≤ 0.05	P≤ 0.001	P≤ 0.02	P≤ 0.01
Group I V/S Group IIB	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
Group I V/S Group IIC	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
Group I V/S Group II	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001

Blood sugar levels (Mean± S.D) in mg/dl

Table 1- shows the mean fasting blood sugar levels were significantly higher in smoke than in non-smokers and it rose progressively from light smokers to heavy smokers. Similarly the mean fasting blood sugar levels after ½ hour, 1hour, 1 ½ hour and 2 hour were significantly higher in smoke than in non-smokers and it rose progressively from light smokers to heavy smokers.

Table 2: Serum insulin level after oral GTT of subjects studied Serum insulin levels (Mean ± S.D) µ/ml

Time	Non smokers (Gr I, n=40)	Light smokers (Gr II, n=20)	Moderate smokers (Gr I, n=20)	Heavy smokers (Gr II, n=20)	Total (Gr II, n=60)
Fasting	11.3 ± 7.8	43.0 ± 7.3	44.8 ± 4.6	49.1 ± 9.5	45.6 ± 7.4
½ hour	26.1 ± 13.4	59.2 ± 7.4	61.1 ± 6.7	67.8 ± 9.5	62.0 ± 8.4
1 hour	36.2 ± 15.0	77.3 ± 8.0	79.5 ± 7.8	68.8 ± 12.3	81.8 ± 10.8
1½ hour	27.5 ± 13.7	58.0 ± 7.7	59.4 ± 8.5	63.6 ± 10.7	60.4 ± 9.2
2 hour	16.4 ± 9.2	44.6 ± 10.6	43.3 ± 7.5	51.4 ± 10.3	47.8 ± 10.8

Statistical significance	Fasting	½ hour	1 hour	1½ hour	2 hour
Group I V/S Group IIA	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
Group I V/S Group IIB	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
Group I V/S Group IIC	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001
Group I V/S Group II	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001	P≤ 0.001

Table 2- shows the mean fasting serum insulin levels were significantly higher in smokers than in non-smokers and it rose progressively from light smokers to heavy smokers. Similarly the mean fasting serum insulin levels after ½ hour, 1 hour, 1 ½ hour and 2 hour were significantly higher in smokers than in non-smokers and it rose progressively from light smokers to heavy smokers. There was a definite dose dependent response between amount of smoking and serum insulin levels after glucose load as the levels increased progressively from light smokers to heavy smokers.

DISCUSSION:

Sandberg et al (1973) found that blood sugar level after smoking 3 cigarettes in rapid succession was significantly higher in smokers than in non-smokers (P<.05) at 30 minutes while no difference was observed at 0, 60, 90,and 120 minutes intervals between blood sugar of smokers and non-smokers, however, oral glucose tolerance test was not performed. Janzon et al (1979 & 1983) and Trell et al (1980) measured blood glucose at 5, 20, 40, 60, 90, and 120 minutes after performing oral and intravenous glucose tolerance tests in non-smokers and smokers.

They concluded that in early phase of OGTT blood sugar levels are higher in smokers while 2 hours values are lower, as compared to non-smokers and ex-smokers. Fracnini *et al.;* in 1992 found that although the plasma glucose after oral glucose load was similar in smokers and nonsmokers, the steady state plasma glucose (SSPG) in response to continuous infusion of insulin, glucose & somatostatin was significantly higher (8.4 in smokers than non-smokers, indicating insulin resistance. In contrast Godsland *et al.;* in 1992 [5] found no evidence of any effect of smoking on IVGTT glucose dependent glucose disposal. Roos *et al.;* in 1993 [6] in a study of 52 pregnant females (15 smokers & 37 nonsmokers) found slight difference between fasting blood glucose of smokers (80.34 ± 8.58 mg/dl) and non-smokers (84.48 ± 7.92 mg/dl, P< 0.05). Zavaroni *et al.;* in 1994 [9] in 152 healthy subjects matched for age, sex &BMI found that smokers (n=76) had impaired glucose tolerance as compared to non-smokers (n=76). However, Szczechowska *et al.;* in 1994 [7] and Eliasson *et al.;* in 1995 [8] did not find any difference in blood glucose levels of smokers and non-smokers.

C. meisinger *et al.;* in 2006 [10] study demonstrated a significant dose–response relationship between nicotine and tar intake and the development of type 2 diabetes in men. Kim *et al.;* in 2014 [11] early onset of smoking increases type II diabetic risk among men in South Korea and the U.S., and type II diabetic risk increases with higher pack-years in U.S. men, however, no associations were found in women population.

CONCLUSION:

The blood sugar level after ½ hour, 1 hour, 1 ½ hour and 2 hour comprehend a definite relationship between blood sugar and number of cigarettes and/or bidi smoked per day. The serum insulin levels were significantly higher in smokers than in non-smokers both at fasting and after ½ hour and 2 hours after oral glucose load and it increased progressively from light to moderate and heavy smokers, showing a positive dose response relationship with number of cigarettes and/or bidi smoked per day. The insulin secretary response as revealed by magnitude of rise in serum insulin levels from fasting to 1 ½ hour and 2 hours of oral glucose load was slower in smokers than in non-smokers showing insulin resistance.

REFERENCES

1. Holbrook JH: Nicotine addiction. In Harrisons principle of Internal medicine, vol.2, 13th edition 1993: 2432.
2. Doll R, Peto R. Mortality in relation to smoking: 20 years' observations on male British doctors. Br med J. 1976 Dec 25; 2(6051):1525-36.
3. Willett WC, Green A, Stampfer MJ, Speizer FE, Colditz GA, Rosner B, Monson RR, Stason W, Hennekens CH. Relative and absolute excess risks of coronary heart disease among women who smoke cigarettes. New England Journal of Medicine. 1987 Nov 19; 317(21):1303-9.
4. Facchini FS, Hollenbeck CB, Jeppesen J, Chen YD, Reaven GM. Insulin resistance and cigarette smoking. The Lancet. 1992 May 9; 339(8802):1128-30.
5. Godsland I, Wynn V, Walton C, Stevenson JC. Insulin resistance and cigarette smoking. The Lancet. 1992 Jun 27; 339(8809):1619-20.
6. Langhoff-Roos J, Wibell L, Gebre-Medhin M, Lindmark G. Effect of smoking on maternal glucose metabolism. Gynecologic and obstetric investigation. 1993; 36(1):8-11.
7. Szczeckowska SE, Grzeszczakw, Urbanm et al. Effect of cigarette smoking on carbohydrate metabolism of healthy subjects' .Pol. Arch Med Wewn 1994 Jan; 19(1) 27-32.
8. Eliasson M, Asplund K, Evrin PE, Lundblad D. Relationship of cigarette smoking and snuff dipping to plasma fibrinogen, fibrinolytic variables and serum insulin. The Northern Sweden MONICA Study. Atherosclerosis. 1995 Feb 28; 113(1):41-53.
9. Zavaroni I, Bonini L, Gasparini P, Dall'Aglio E, Passeri M, Reaven GM. Cigarette smokers are relatively glucose intolerant, hyper insulinemic and dyslipidemic. The American journal of cardiology. 1994 May 1; 73(12):904-5.
10. Meisinger C, Döring A, Thorand B, Löwel H. Association of cigarette smoking and tar and nicotine intake with development of type 2 diabetes mellitus in men and women from the general population: the MONICA/KORA Augsburg Cohort Study. Diabetologia. 2006 Aug 1; 49(8):1770-6.
11. Kim SJ, Jee SH, Nam JM, Cho WH, Kim JH, Park EC. Do early onset and pack-years of smoking increase risk of type II diabetes? BMC public health. 2014 Feb 19; 14(1):178.