**Original Research Article**

# A study of impact of smoking on glycemic index status among patients who are attending Government Dharmapuri Medical College Hospital, Dharmapuri

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## Abstract

**Introduction:** Tobacco use has long been known to be a major risk factor for cardiovascular disease. Recent studies have identified a positive association between smoking and incidence of diabetes. The evidence that smoking is an independent risk factor for the development of diabetes is still considered preliminary. Some studies have shown a dose response association between smoking and incidence of diabetes.

**Aim of The Study:** To assess whether smokers are more likely than non-smokers to develop clinically relevant glucose intolerance or diabetes and to assess whether total pack years' correlates with the severity of glucose intolerance/ diabetes.

**Materials and Methods:** We have evaluated Hundred smokers and FIFTY nonsmokers Baseline characteristics of the study were as follows; age, sex, occupation, Education status, family history of diabetes, comorbid illness of smoking. Our Baseline examination included Ht, Wt, BMI, BP, HC, WC, Waist Hip ratio (anthropometric measurements), Lab investigation – Blood sugar (F and PP) and Lipid profile. Participants were defined as current smokers and former smokers. Former smoker were those who, at baseline, reported previously using cigarettes but denied current smoking.

**Results:** Nearly 5% of the case population were obese. Among the control population, obesity accounted for 2% of them. There was a significant statistical difference between cases and controls with respect to BMI ('p' = 0.0116). With regard to Blood Pressure, there was no significant statistical difference between cases and controls ('p' = 0.4989). Glucose Intolerance among case population

were found to be 29% and among the control population, found to be 22%. Thus there was no significant statistical difference between cases and controls ('p' = 0.4727). 53 cases of the study group (53%) had high Triglyceride. 46% of the control group had high TG. Thus, there was no significant statistical association between study and control group (p =0.52) 13 cases of the study group (13%) had low HDL. 14% of the control group also had low HDL. Thus there was no significant statistical association between study and control group (p = 0.93). 10% of the case population had metabolic syndrome and 6% of the control population had the same. There was no significant statistical association. 10% of the case population had metabolic syndrome and 6% of the control population had the same. There was no significant statistical association.

**Conclusion:** Smoking stimulate symathetic system, which inturnleads to anelevated catecholamine levels and there by insulin resistence.Smoking influences visceral adipose tissue and there by insulin resistance.Smokers(especially heavy smokers) are prone for unhealthy food habits and low physical activity which in turn leads to visceral fat accumulation and insulin resistance.

## Key words

Glycemic index, Smoking, Insulin resistance, Obesity, Lipid profile.

## Introduction

Several Hypothesis have been proposed to link tobacco use and incidence of diabetes. Smoking has been linked to impaired response to glucose tolerance tests and insulin resistance. Although, smoking cessation can result in modest weight gain, smoking is related to a more unhealthy distribution of upper body weight and greater waist – hip ratio [1]. Smoking has also been associated with risk of chronic pancreatitis and pancreatic cancer, suggesting that tobacco smoke may be directly toxic to pancreas. "Heavy smokers are more likely to get diabetes over time than are lighter smokers who are in turn are more likely to get diabetes than non-smokers," said by Dr. William Ghali, one of the review authors [2].

While researchers are hesitant to directly link smoking to the onset of diabetes, they theorize that smoking may "lead to insulin resistance or inadequate compensatory insulin secretion responses," according to the authors, primarily from the University of Lausanne in Switzerland. Insulin resistance means [3]. The body is less able to both store and process glucose, causing blood glucose levels to rise and leading to the development of Type 2 diabetes. A number of preliminary studies have assessed the association between smoking and incidence of glucose abnormalities, suggesting that active smoking could be independently associated with glucose intolerance, impaired fasting glucose, and type 2 Diabetes [4]. There is a growing body of evidence that smoking is an Independent risk factor for diabetes, and that among people with diabetes, smoking aggravates the risk of serious disease and premature death Cigarette smoking remains the most important cause of preventable morbidity and early mortality [5]. In 2000, there was an estimated 4.8 million premature deaths in the world attributable to smoking, 2.8 million in developing countries and 2 million in industrialized countries. More than 3/4 (3.8 million) of these deaths were in men. Nicotine is highly addictive, raises the Brain Level of Dopamine and alters the Bioavailability of Dopamine and Seratonin. It produces withdrawal symptoms on discontinuation. Cigarettes, cigars, spit and pipe tobacco is made from dried tobacco leaves, as well as ingredients added for flavor and other reasons [5]. More than 4,000 different chemicals have been found in tobacco and tobacco smoke. Among them more than 60 chemicals known to cause cancer (carcinogens).There are hundreds of substances added to cigarettes by manufacturers to enhance the flavor or to make smoking more pleasant. Some of the compounds found in tobacco smoke include ammonia, tar, and carbon monoxide. Exactly what effects these substances have on the

cigarette [6]. Diabetes Mellitus is a metabolic disorder characterized by hyperglycemia resulting from defects in Insulin secretion, Insulin action on both. The prevalence of type and diabetes is increasing all over the world particularly in the developing countries. It has emerged as a major public health problem in our country. The WHO estimated that there were 31.7 million persons with diabetes in India in 2000 and that the number is likely to be 71.4 million in 2030 [7]. India has the distinction of having the largest number of diabetes in the world. Studies in 1980 highest prevalence rates of type and diabetes among migrant ethnic groups, suggesting that Indians as an ethnic group had a genetic propensity to develop diabetes which was precipitated by lifestyle changes. Current prevalence rates are 12.1% in the urban population. There is evidence that the prevalence of type 2 diabetes in increasing in rural population also. Type 2 diabetes amongst Indians occurs at a younger age, the age at diagnosis being a decade earlier than in the west. Body mass indeed is lower by 4 kg/m2 in male and 6 kg/$m^2$ in female. However, abdominal obesity with increased waist to hip ratio is more common [8].

## Materials and methods

This cross-sectional study was conducted in December 2016 – July 2017. We have evaluated Hundred smokers and Fifty non-smokers Baseline characteristics of the study were as follows; age, sex, occupation, Education status, family history of diabetes, comorbid illness of smoking. Our Baseline examination included Ht, Wt, BMI, BP, HC, WC, Waist Hip ratio (anthropometric measurements), Lab investigation – Blood sugar (F and PP) and Lipid profile. Participants were defined as current smokers and former smokers. Former smoker were those who, at baseline, reported previously using cigarettes but denied current smoking. Informed consent was obtained from all Patients or relatives.

**Inclusion criteria**

All smokers attending Government Dharmapuri Medical College Dharmapuri from 18 – 60 years of age.

**Exclusion criteria**

- Known diabetes
- Alcoholics
- Pregnancy
- Age < 18 and > 60 years.
- Acute stressful situations such as Myocardial infarction, Trauma, Severe Infection
- Patients on drugs such as Steroids, OCP, Thiazide diuretics

## Results

Nearly 5% of the case population was obese. Among the control population, obesity accounted for 2% of them. There was a significant statistical difference between cases and controls with respect to BMI ('p' = 0.0116). With regard to Blood Pressure, there was no significant statistical difference between cases and controls ('p' = 0.4989). Glucose Intolerance among case population was found to be 29% and among the control population, found to be 22%. Thus there was no significant statistical difference between cases and controls ('p' = 0.4727) as per **Table - 1**.

53 cases of the study group (53%) had high Triglyceride. 46% of the control group had high TG. Thus, there was no significant statistical association between study and control group (p =0.52). 13 cases of the study group (13%) had low HDL. 14% of the control group also had low HDL. Thus there was no significant statistical association between study and control group (p = 0.93) as per **Table – 2**.

10% of the case population had metabolic syndrome and 6% of the control population had the same. There was no significant statistical association (**Table – 3**).

There was a significant statistical association between smoking and glycemic status (p = 0.0048). There was no statistically significant difference between current smokers and former smokers (**Table – 4**).

**Table – 1:** Physical profile of patients.

| Parameter | | Cases (Smokers) | | Controls (Non Smokers) | |
|---|---|---|---|---|---|
| | | **No** | **%** | **No** | **%** |
| **A. Body mass index (BMI)** | | | | | |
| i) | Normal (< 25) | 70 | 70 | 45 | 90 |
| ii) | Over weight (25 – 29.9) | 25 | 25 | 4 | 8 |
| iii) | obese (30 and above) | 5 | 5 | 1 | 2 |
| 'P' | | 0.0116 Significant | | | |
| **B. Blood pressure(BP)** | | | | | |
| i) | Normal | 80 | 80 | 43 | 86 |
| ii) | Abnormal | 20 | 20 | 7 | 14 |
| 'P' | | 0.4989 Not significant | | | |
| **C. Waist Circumference** | | | | | |
| i) | Normal (m < 102 F < 88) | 97 | 97 | 50 | 100 |
| ii) | Abnormal (m > 102 F > 88) | 3 | 3 | - | - |
| 'P' | | 0.5511 Not Significant | | | |
| **D. Fasting Blood Sugar** | | | | | |
| i) | Normal (Less than 100mg/dl) | 71 | 71 | 39 | 78 |
| ii) | Impaired (100 – 125) | 12 | 12 | 11 | 22 |
| iii) | Diabetes (more than 125) | 17 | 17 | - | - |
| 'P' | | 0.4727 Not significant | | | |

**Table – 2:** Lipid profile changes among patients.

| Parameter | | Cases (Smokers) | | Controls (Non Smokers) | |
|---|---|---|---|---|---|
| | | **No** | **%** | **No** | **%** |
| **A. Triglyceride(TGL)** | | | | | |
| i) | Normal (≤ 150) | 47 | 47 | 27 | 54 |
| ii) | Abnormal (> 150) | 53 | 53 | 23 | 46 |
| 'P' | | 0.5253 (Not Significant) | | | |
| **B. HDL** | | | | | |
| i) | Normal (≥ 40) | 87 | 87 | 43 | 86 |
| ii) | Abnormal (< 40) | 13 | 13 | 7 | 14 |
| 'P' | | 0.9323 (Not Significant) | | | |

## Discussion

Among the 150 participants in my study, 100 of them were smokers. Among the 100 smokers, 83 were current smokers and 17 were former smokers. The mean age of the participants was 47 (SD 10.7) years; majority of the smokers had an occupation of daily wages laborers and a low educational status [9]. In my study, Tobacco consumption in the form of smoking was observed more among low educational status (Up to $5^{th}$ standard – 45%) and illiterates (21%), compared to those educated higher (>$5^{th}$ standard – 34%) In one study, the greatest tobacco consumption was observed among illiterate (60%) and low education status (51%), compared to more literate ($6^{th}$ -$10^{th}$ and > 10 years of formal education-46 and 36% respectively). This difference in the above could be explained as

follows: My study included 100 cases, compared to their study (3148 cases) [10]. My study included tobacco consumption only in the form of smoking in contrast to their study which included other forms of tobacco use in addition to smoking. In my study, 12 participants out of 100 smokers had impaired fasting glucose and 17 cases had diabetes. The prevalence of glucose intolerance was also higher among smokers than is non-smokers (p. =0.004) [11]. One study showed a graded association between smoking exposure and the development of glucose Intolerance. The 15 year incidence of glucose intolerance was highest among smokers (21.8%) followed by never smokes with passive smoking exposure (17.2%), and then previous smokers (14.4%); It was lowest for never smokers with no passive exposure (11.5%). Thus, their study ended up by stating that current smokers and never smokers with passive smoking exposure were at higher risk than never smokers without passive smokers exposure and risk in previous smokers was similar to that is never smokers without passive smoking exposure. In my study, which was a cross sectional study, there was a significant statistical association between smoking and glycemic status (p' 0.0048). My study also showed that a statistically significant association between current smokers and non-smokers (p' 0.0055) [12]. But, in contrary to one study, which said that risk is previous smokers was similar to that of never smokers without passive smoking exposure, my study showed a significant statistical association between former smokers and non-smokers ('p' 0.0142). This may be due to the fact that, my study did not subcategorized never smokers as, those with passive exposure and without passive exposure. But, there are certain studies which supported the fact that former smokers were also at risk for Diabetes [14]. Systematic review and meta-analysis of studies describing the association between active smoking and incidence of diabetes or other glucose intolerance which also indicated that active smokers had 44% increased risk for developing type 2 diabetes compared with Non-smokers [15]. They also described a significant association between former smokers and incidence of diabetes. In my study, the pack years among participants with normal glycemic status had a mean of 9.3 (S.D 8.4); participants with impaired fasting glucose had a mean of 12.2 (S.D 8.9) and those with diabetes had a mean of 13.6 (S.D 8.7). Thus, increase in pack years of smoking among the participate was associated with an increased risk of developing glucose intolerance. Of the components of the metabolic syndrome, we could find a significant association for fasting plasma glucose but we could not find a significant association with other factors [16]. In my study, thus smoking did not show statistically significant correlation to metabolic syndrome compared to non-smokers. This study is somewhat contrary to the general concept that cigarette smoking is independently associated with metabolic syndrome. We suppose that the cross sectional design of this study limited its ability to detect these associations [17].

**Table – 3:** Metabolic syndrome among patients.

| **Abnormalities** | **Smokers** | | **Non Smokers** | |
|---|---|---|---|---|
| | **No** | **%** | **No** | **%** |
| a) Central obesity (Waist circumference M > 102 cm, F > 88 cm) | 3 | 3 | - | - |
| b) Abnormal TGL (> 150) | 53 | 53 | 23 | 46 |
| c) Abnormal HDL (< 40) | 13 | 13 | 7 | 14 |
| d) Hypertension (systolic B.P ≥ 130, Diastolic B.P ≥85) | 20 | 20 | 7 | 14 |
| e) Fasting blood glucose (≥100) | 29 | 29 | 11 | 22 |
| Metabolic syndrome(any 3 of the above) | 10 | 10 | 3 | 6 |
| 'P' | 0.3127 Not Significant | | | |

(TGL: Triglyceride, HDL: High Density Lipoprotein, B.P: Blood Pressure)

**Table – 4:** Glycemic status and smoking among patients.

| Glycemic Status | Smokers | | | | Total Smokers | | Non Smokers | |
|---|---|---|---|---|---|---|---|---|
| | Current | | Former | | | | | |
| | Mean | % | Mean | % | Mean | % | Mean | % |
| Normal (110) | 60 | 54.5 | 11 | 10 | 71 | 64.5 | 39 | 35.5 |
| Impaired (23) | 9 | 39.1 | 3 | 13 | 12 | 52.5 | 11 | 47.8 |
| Diabetes (17) | 14 | 82.4 | 3 | 7.6 | 17 | 100 | - | - |
| **'p'** smokers vs non-smokers **0.0048 Significant** | | | | | | | | |
| **'p'** Current smokers vs non-smokers **0.0055 (Significant)** | | | | | | | | |
| **'p'** Former smokers vs non-smokers **0.0142 (Significant)** | | | | | | | | |
| **'p'** Current smokers vs Former smokers **0.5884 ( Not significant)** | | | | | | | | |

## Conclusion

Smokers (especially heavy smokers) are prone for unhealthy food habits and low physical activity which in turn leads to visceral fat accumulation and insulin resistance. Smoking directly influences insulin sensitivity and impairs insulin action. The pack years of exposure showed a significant positive association with glucose intolerance/ diabetes. The mean pack years among patients with glucose intolerance is 11.8 and the mean pack years among patients with frank diabetes is 12.7. Thus, the risk of diabetes directly correlates with the pack years in my study. As the pack years increases, the risk of diabetes also increases [18].

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