

Original Research Article

A Study of HbA1c, fasting and 2 hour plasma glucose levels in current smokers presenting at a tertiary care hospital in North India

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ABSTRACT

Background: There is a concern that smokers are at increased risk of diabetes and this association has received surprisingly little attention. This study aims to study the relationship of smoking with three glycaemic variables (HbA1c, fasting and 2 hour post prandial plasma glucose levels) in current smokers and describe the potential impact of smoking in the context of screening for diabetes by using one of these glycaemic variables.

Methods: 150 patients attending the OPD of Santosh Medical College were part of this Cross sectional population based study. One hundred were smokers and fifty non-smokers and non-diabetics. All participants were subjected to fasting plasma glucose level, 2 hour plasma glucose level and HbA1c testing.

Results: Mean values of all three variables are substantially higher in smokers as compared to non-smokers. HbA1c levels show an increasing trend with increasing age among both groups. Mean plasma glucose levels show increasing values with increasing age but the trend is not uniform. Among females, only HbA1c levels whereas among males all three variables show a strong correlation with smoking. No correlation was observed between the number of cigarettes smoked daily or smoking index and the glycaemic variables. All variables show an increasing trend as the number of years of smoking increases.

Conclusions: A higher prevalence of diabetes is seen in current smokers when we consider HbA1c levels indicating the beneficial effect of using HbA1c levels for identifying current smokers at risk of diabetes mellitus.

Keywords: Blood glucose, Glycosylated, Haemoglobin A, Smoking

INTRODUCTION

Diabetes is a major public health problem globally and detecting those who have a high probability of developing type 2 diabetes is a priority so that prevention programmes can be proposed to people at risk.¹

Cigarette smoking is a well-known risk factor for cardiovascular diseases and several cancers. Possible association of smoking and risk of diabetes has been reported in both Asian and non-Asian population.² A number of primary 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 mellitus: smoking therefore may be modifiable risk factor for type 2 diabetes.³⁻⁵ Several studies have found that current smokers have higher glycosylated haemoglobin concentrations as compared to non-smokers.⁶⁻⁸

The World Health Organisation study group has now suggested the evaluation of glycated haemoglobin as an alternative method to plasma glucose measurement for screening of diabetes mellitus.⁹ This provides cumulative estimate of mean blood glucose concentration over

preceding 2-3 months and major glycated haemoglobin A1C (HbA1C) is the most reliable indicator of long term blood glucose.¹⁰⁻²¹

Table 1: Spectrum of glucose homoeostasis and diabetes mellitus.

	Normal glucose tolerance	Impaired Glucose tolerance (pre-diabetes)	Diabetes mellitus
Fasting plasma glucose	< 100 mg /dl (<5.6 mmol/L)	100-125 mg /dl (5.6-6.9 mmol/L)	>126 mg /dl (> 7.0 mmol/L)
2 h plasma glucose	< 140 mg/dl (< 7.8 mmol/L)	140-199 mg /dl (7.8-11 mmol/L)	>200 mg /dl (> 11.1 mmol/L)
Hb A1 c	<5.6%	5.7-6.4%	> 6.5%

METHODS

Study design

This was a cross sectional population based study. The subjects were divided into the following groups:

- Study group/Cases,
- Control.

Sample size

Total 150 subjects aged between 20-70 years were taken. These 150 subjects included 100 smokers and 50 non-smokers non-diabetics. Both these groups were age and gender matched.

Place of study

The study was carried out in Santosh Medical College, Santosh University, Ghaziabad from June 2015 onwards on patients attending the outpatient clinics of the Department of Medicine.

* All the subjects were voluntary participants in the study and informed consent was taken.

Inclusion criteria

- All current smokers (as defined by Centre for Disease Control and Prevention criteria),
- Not known diabetics who were healthy and taking no medication affecting carbohydrate or lipid metabolism.

Exclusion criteria

- All known diabetics,
- Patient on medication interfering with carbohydrate or lipid metabolism,

- Patient with known chronic heart disease, COPD, MI, chronic hypertension,
- All former or never smokers (as defined by Centre for Disease Control and Prevention criteria).
- 150 patients attending the outpatient clinics of Santosh Medical College were part of the study out of which 100 were smokers and 50 were non-smokers and non-diabetics. These groups were age and gender matched and were evaluated clinically, including history, physical examination, blood pressure measurement, questionnaires on life style characteristics such as physical activity and smoking habits.
- Smoking habits were gone into detail. A detailed smoking history was taken, and smoking index was calculated.

Smoking index is product of average number of cigarettes/ bidis smoked per day and the total duration of smoking in years.

Smoking Index =No. of cigarettes per day x total duration (in years).

- All the patients were subjected to fasting plasma glucose level, 2 hours post prandial plasma glucose level and HbA1c level testing.
- Plasma glucose was estimated by GOD-POD (Glucose oxidase peroxidase) End point method.

HbA1c was estimated by Ion Exchange Resin method.

RESULTS

Table 2: Comparison of mean HbA1c levels between cases and controls.

	Controls	Cases	P value
	Mean ± sd	Mean ± sd	
HbA1c (%)	5.36 ± 0.49	6.39 ± 1.09	<0.001*

The mean HbA1c value in cases (6.39% +1.09) is higher than the mean HbA1c value in controls (5.36% + 0.49). The difference in the values is statistically significant ('p-value' < 0.05).

Table 3: Comparison of mean plasma glucose levels (fasting and post prandial) between cases and controls.

	Controls	Cases	
	Mean ± SD	Mean ± SD	
Plasma glucose (F) (mg/dl)	94.42±9.18	104.87±35.00	0.006*
Plasma glucose (PP) (mg/dl)	103.98±16.79	125.52±61.45	0.001*

Mean fasting plasma glucose level is higher in cases (104.87 mg /dl +35) as compared to controls (94.42 mg /dl + 9.18). The mean post prandial plasma glucose level

in cases (125.52 mg /dl + 61.45) is higher than in controls (103.98 mg/dl + 16.79). The difference in values is statistically significant ('p-value' < 0.05).

Table 4: Comparison of mean HbA1c levels between cases and controls in different age groups.

Hba1c (%)					
Age groups	Controls		Cases		P value
	n	Mean ± SD	n	Mean ± SD	
21 - 30 yrs	11	5.24 ± 0.42	14	5.93 ± 0.60	0.003
31 - 40 yrs	18	5.43 ± 0.40	22	5.97 ± 0.73	0.008
41 - 50 yrs	8	5.19 ± 0.54	29	6.55 ± 01.32	0.008
51 - 60 yrs	7	5.43 ± 0.66	24	6.67 ± 1.00	0.005
61 - 70 yrs	6	5.55 ± 0.60	11	6.75 ± 1.34	0.058

In all age groups the mean HbA1c levels in smokers are higher than the non- smokers. The difference is statistically significant in all age groups ('p-value' < 0.05) except 61-70 years age group. In the 61-70 years age

group the 'p-value' is only slightly greater than 0.05. This discrepancy may be due to the small sample size of controls and cases in this age group because of which even one or few value(s) can affect the mean value significantly.

Table 5: Comparison of mean plasma glucose levels (fasting and post-prandial) between cases and controls in different age groups.

Age groups		n	Controls		Cases		P value
			Mean ± SD	n	Mean ± SD		
21 - 30 yrs	Plasma glucose (F) (mg/dl)	11	90.82 ± 9.55	14	92.00 ± 9.12	0.756	0.791
	Plasma glucose (PP) (mg/dl)		106.00 ± 15.84		108.21 ± 23.42		
31 - 40 yrs	Plasma glucose (F) (mg/dl)	18	95.11 ± 6.50	22	94.32 ± 9.21	0.760	0.322
	Plasma glucose (PP) (mg/dl)		102.06 ± 15.40		107.36 ± 17.60		
41 - 50 yrs	Plasma glucose (F) (mg/dl)	8	98.88 ± 6.62	29	111.72 ± 48.69	0.179	0.332
	Plasma glucose (PP) (mg/dl)		108.50 ± 18.23		136.59 ± 79.33		
51 - 60 yrs	Plasma glucose (F) (mg/dl)	7	90.29 ± 12.64	24	106.71 ± 23.65	0.091	0.208
	Plasma glucose (PP) (mg/dl)		94.43 ± 17.16		131.54 ± 74.80		
61 - 70 yrs	Plasma glucose (F) (mg/dl)	6	97.83 ± 12.12	11	120.27 ± 55.11	0.347	0.256
	Plasma glucose (PP) (mg/dl)		111.17 ± 19.77		141.55 ± 60.48		

Table 6: Gender wise comparison of mean HbA1c and plasma glucose (fasting and post-prandial) levels between cases and controls.

	Controls	Cases	P value
	Mean ± SD	Mean ± SD	
Female			
HbA1c (%)	5.30 ± 0.46	6.71 ± 0.81	0.003*
Plasma glucose (F) (mg/dl)	93.20 ± 12.21	105.91 ± 32.71	0.421
Plasma glucose (PP) (mg/dl)	119 ± 14.16	132.00 ± 36.27	0.458
Male			
HbA1c (%)	5.37 ± 0.50	6.35 ± 1.11	<0.001*
Plasma glucose (F) (mg/dl)	94.56 ± 8.95	104.74 ± 35.45	0.012*
Plasma glucose (PP) (mg/dl)	102.31 ± 16.35	124.72 ± 63.97	0.002*

The general trend in this table shows an increase in the values as we move from the lower to higher age groups.

However, the increase is not uniform. Among controls, highest levels of fasting plasma glucose if seen in the 41-

50 years age group and highest level of post-prandial plasma glucose is seen in the 61-70 years age groups. Among cases, highest level of fasting and post-prandial plasma glucose is seen in the 61-70 years age group.

Among female participants of the study the mean levels of all variables are higher in smokers as compared to non-smokers and in the case of HbA1c this difference is statistically significant.

Among male participants of the study the mean levels of all variables are higher in smokers as compared to non-smokers. In the case of all three variables this difference is statistically significant.

Table 7: Comparison of mean HbA1c and plasma glucose (fasting and post-prandial) levels between bidi smokers and cigarette smokers.

	Bidi(n=61)		Cigarette (n=39)		P value
	Mean ± sd	Mean ± sd	Mean ± sd	Mean ± sd	
HbA1c (%)	6.40 ± 1.07	6.36	±1.12		0.852
Plasma glucose (F) (mg/dl)	101.39 ± 27.70	110.31	±43.94		0.216
Plasma glucose (PP) (mg/dl)	122.36 ± 50.78	130.46	±75.66		0.523

The mean HbA1c level in Bidi smokers is higher than the mean HbA1c in Cigarette smokers. However, the difference is NOT statistically significant (p-value>0.05). The mean Plasma Glucose (Fasting and Post-Prandial) levels are higher in cigarette smokers than the corresponding levels in Bidi smokers. However, the difference is NOT statistically significant (p-value > 0.05 in both cases).

Table 8: Comparison of mean HbA1c and plasma glucose (fasting and post-prandial) levels on the basis of number of bidis/cigarettes smokers per day.

	Numbers of bidi/cigarette per day				P value
	1- 5	6 - 10	>10		
	Mean ± SD	Mean ± SD	Mean ± SD		
HbA1c (%)	6.32 ± 0.89	6.47 ± 1.36	6.39 ± 0.90		0.821
Plasma glucose (F) (mg/dl)	103.91 ± 31.96	109.82 ± 41.71	89.67 ± 9.69		0.290
Plasma glucose (PP) (mg/dl)	126.51 ± 57.77	128.95 ± 72.25	105.22 ± 19.00		0.578

The highest mean HbA1c values are seen in smokers who smoke 6-10 bidis /cigarettes per day (6.47% + 1.36) while the lowest mean HbA1c values are seen in smokers who smoke 1-5 bidis/cigarettes per day (6.32% + 0.89).

Highest values of mean Plasma Glucose levels (fasting and post prandial) are seen in smokers who smoke 6-10 bidis/cigarettes per day (109.82 mg/dl + 41.71 and 128.95 mg /dl + 72.25 respectively) and lowest value in smokers who smoke more than 10 bidis /cigarettes per day (89.67 mg/dl +9.69 and 105.22 mg /dl + 19 respectively). However, the difference in the values of three variables across the three sub groups of smokers is NOT significant statistically.

Table 9: Comparison of mean HbA1c and plasma glucose (fasting and post-prandial) levels between different groups of smokers (cases) divided on the basis of years since smoking.

Age groups	HbA1c (%)	Plasma glucose (F) (mg/dl)	Plasma glucose (PP) (mg/dl)
1 - 5 yrs	Mean 5.96 ± SD ± 0.55	92.00 ± 7.53	104.94 ± 20.79
6 - 10 yrs	Mean 6.13 ± SD 0.88	103 ± 24.95	115.71 ± 30.28
11 - 15 yrs	Mean 6.00 ± SD ± 0.40	94.25 ± 11.06	108.25 ± 12.75
16 - 20 yrs	Mean 6.94 ± SD ± 1.68	124.21 ± 56.18	166.53 ± 115.82
21 - 30 yrs	Mean 6.42 ± SD ± 0.77	99.11 ± 16.57	115.17 ± 32.95
>30 yrs	Mean 6.89 ± SD ± 1.30	116.92 ± 52.32	140.92 ± 57.22
P value	0.017*	0.035*	0.016*

Table 10: Classification and comparison of HbA1c profiles between cases and controls.

	Controls		Cases		
<5.6	35	70%	20	20%	
5.7 - 6.4	15	30%	53	53%	
>6.5	0	0%	27	27%	<0.001*
Total	50	100%	100	100%	

The mean values of HbA1c and Plasma Glucose (Fasting and Post-prandial) levels show an increasing trend with increasing number of years.

The increase is statistically significant (because 'p-value' in all three cases <0.05). The increase in the mean levels of the three variables is not uniform. The maximum values of the 3 variables are seen in the group of smokers who have been smoking for the past 16-20 years.

DISCUSSION

The association between smoking and these three glycaemic variables has been studied but few have studied all three variables in the same population.²⁴ The aim of this study was to assess and quantify the influence of smoking on mean values of HbA1c and Plasma

Glucose (Fasting and Post- Prandial) in the population under study and to describe the potential impact of

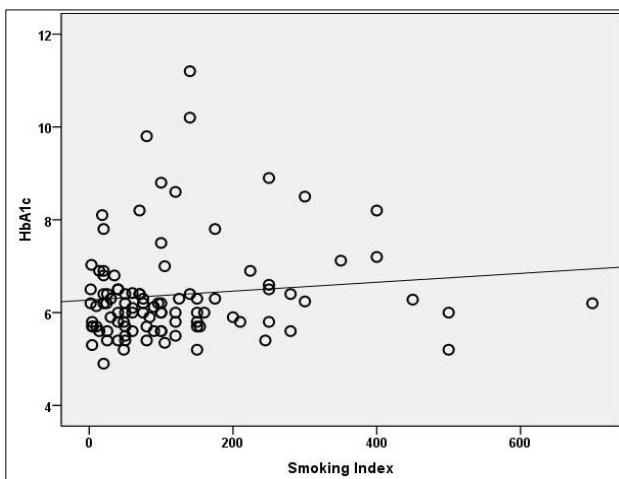
smoking in the context of screening for diabetes by one of these glycaemic variables.

Table 11: Classification and comparison of plasma glucose (fasting) profiles of cases and controls.

Plasma glucose (F) (mg/dl)	Controls		Cases		
	Frequency	%	Frequency	%	
<100	36	72%	61	61%	
100 - 125	14	28%	29	29%	
>126	0	0%	10	10%	0.060
Total	50	100%	100	100%	

Table 12: Classification and comparison of plasma glucose (post-prandial) profiles of cases and controls.

Plasma Glucose (PP) (mg/dl)	Controls		Cases		p value
	Frequency	%	Frequency	%	
<140	49	98%	81	81%	
140 - 199	1	2%	13	13%	0.015*
>200	0	0%	6	6%	
Total	50	100%	100	100%	



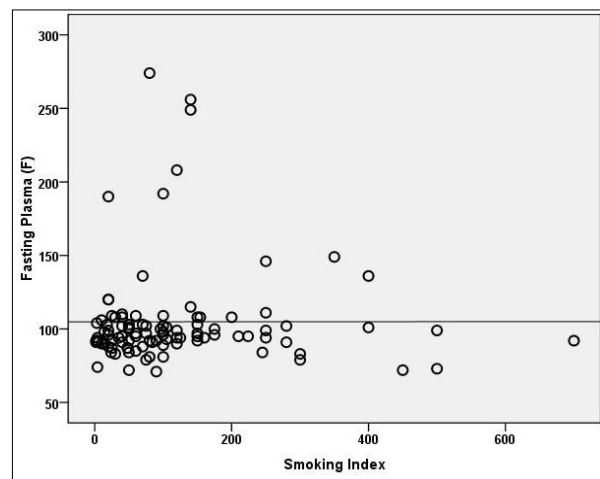
There was no correlation between Smoking Index and HbA1c (%) in smokers ($r=0.115$, $p=0.254$).

Figure 1: Correlation between smoking index and hba1c (%) in cases.

In our study the mean HbA1c level in smokers was substantially higher than mean HbA1c level in non-smokers. Similar findings have been reported in almost all studies that have compared HbA1c levels between smokers and non-smokers.^{28,29}

A 'p-value' < 0.05 indicates strong evidence against the null hypothesis and therefore in our study, the 'p- value' < 0.001 in this comparison indicates a strong correlation between smoking and the observed increase in mean HbA1c levels.

Various studies have observed higher fasting plasma glucose levels in smokers as compared to non-smokers.²⁸



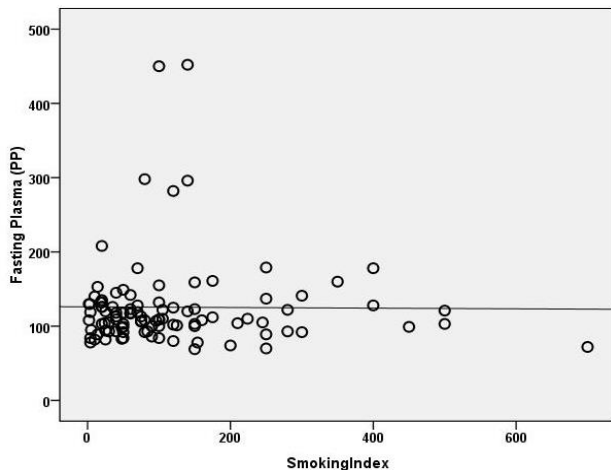
There was no correlation between Smoking Index and Plasma Glucose (Fasting) in smokers ($r=0.089$, $p=0.380$).

Figure 2: Correlation between smoking index and plasma glucose (fasting) in cases.

Some studies have reported contrary findings whereas other have observed no significant difference in plasma glucose level between smokers and non-smokers.^{26,25,30} In our study 'p values' < 0.05 indicates a strong correlation between smoking and the observed increase in mean fasting plasma glucose level in smokers.

In our study, mean post prandial plasma glucose levels are significantly higher in smokers as compared to non-smokers. Similar findings have been reported by the India, CURES study by Deepa M et al which is the largest population based studies to be done in India in the field of diabetes and its complications.³¹ However most studies conducted outside in India have reported lower

post-prandial plasma glucose level in smokers as compared to non-smokers.^{25,26,28-30}



There was no correlation between Smoking Index and Plasma Glucose (PP) in smokers ($r=0.001$, $p=0.993$).

Figure 3: Correlation between smoking index and plasma glucose (post-prandial) in cases.

The transient increase in blood glucose associated with smoking represents one of the many well-known acute effects of nicotine as shown by Milson A S et al.³² Cigarette smoking has been associated with increased resistance to insulin stimulated glucose uptake than non-smokers.³³ This could be due to the direct effects of nicotine, carbon monoxide or other agents in tobacco smoke.³⁴ In 1993 Attval et Al showed that smoking impaired insulin sensitivity and acutely impaired insulin action in otherwise healthy subjects.³⁵

In our study mean HbA1c values show an increasing trend with increasing age in both smokers (cases) and non-smokers (controls). While this can be attributed (even if, partially) to the effects of smoking in smokers (cases) the same cannot be said from the observed increase in non-smokers (controls). Mokded AH et al observed that HbA1c increases linearly with age in both sexes.³⁶ Arnetz BB et al also demonstrated an increase in HbA1c with ageing in non-diabetic subjects.³⁷

Similarly, we observe an increasing trend of mean plasma glucose (fasting and post prandial) values as we move from lower to higher age groups in both smokers (cases) and non-smokers (controls). This could be due to impaired glucose regulation with increasing age.²⁵ This impairment of glucose tolerance with advancing age is a well-known phenomenon first reported by Spence JC.³⁸ Some studies have reported that a large proportion of elderly subjects aged 65 years and over have either impaired glucose tolerance or overt diabetes.^{39,40}

In the case of all three variables we have observed an increase in mean values in smokers (cases) as compared to non-smokers (controls) in every age group. However, a strong correlation between smoking and the observed

increase exists only in the case of HbA1c ('p-value' almost equal to 0.05 in 61-70 years age group and < 0.05 in all other age groups).

When we compare mean Plasma Glucose (fasting and post-prandial) values between smokers and non-smokers in different age groups a strong correlation between smoking and the observed increase in mean value cannot be made out because 'p-value' in every age group is > 0.05 .

On tabulating the gender wise comparison we observe that the mean values of all three variables are higher in smokers (cases) than non-smokers (controls) in both female and male participants of the study.

In the comparison between Bidi smokers and Cigarette smokers we observe that Bidi smoking and Cigarette smoking are equally harmful.

Previous studies have reported a positive dose-response relationship between HbA1c levels and both the number of cigarettes smoked per day and with total smoking as measured by pack years. Heavy smokers (≥ 20 cigarettes per day) have been shown to have higher plasma glucose levels than smokers smoking < 20 cigarettes per day. Various studies have reported a dose response relationship between cigarettes smoked per day and the incidence of diabetes mellitus in both men and women. The number of pack years has also been observed to be positively related to the development of diabetes mellitus in a dose dependent manner as reported by Uchimoto S et al.

However, we could not derive any such association in our study. We observe the mean values of the three variables are highest in the group of smokers who smoke 6-10 bidis/cigarettes per day. In the group of smokers smoking > 10 bidis /cigarettes per day the mean plasma glucose (fasting and post-prandial) values are the lowest, lower even than the group of smokers smoking 1-5 bidis/cigarettes per day, while the mean Hb A1c level in this group is only marginally higher than the corresponding mean HbA1c level in the group of smokers smoking 1-5 bidis/cigarettes per day. Similarly, in our study no correlation was observed between the smoking index and mean HbA1c or plasma glucose (fasting and post-prandial) levels.

Simon D et al reported that although their study showed an increase in HbA1c in smokers and that this shift was more evident in heavier smokers, the correlation coefficient between HbA1c and tobacco consumption was not significant.²⁶ Norto Kawakami et al observed that the increase in NIDDM risk related to number of cigarettes smoked per day was modest when the number of cigarettes smoked per day was greater than 25. A similar pattern was observed by Attvall S et al.³⁵ The authors suggested that smoking a certain number of cigarettes per day is sufficient for developing NIDDM

and there is no further increase in risk beyond that amount. Similar “saturated” effects of number of cigarettes smoked per day have been reported from studies of smoking and serum lipoprotein disturbances and smoking and plasma fibrinogen levels. Other studies have shown that the number of cigarettes smoked daily was related to the risk of developing diabetes but not monotonically. Rimm EB et al and Kawakami N et al showed that there was no relationship between subjects smoking only a few cigarettes per day and the risk of diabetes mellitus. Ohlson LO et al and Perry IJ et al also did not find any positive association between cigarettes smoked per day and the incidence of diabetes mellitus in both men and women.

In our study we observe that the mean values of HbA1c and plasma glucose (fasting and post-prandial) show an increasing trend (although not uniform) as the number of years since the smokers (cases) have been smoking increases.

The changes acutely caused by smoking were found to be more pronounced in chronic smokers by Fratje et al. The risk of diabetes has been found to be higher among those who started smoking at a younger age as also seen by Kawakami N et al. Smoking related changes in triglyceride, low density lipoprotein cholesterol and high density lipoprotein cholesterol levels have been reported to be greater in smokers who begin smoking at an early age. Smoking might cause more insulin resistance in younger people than in adults. The increased risk of NIDDM in those who start smoking at a younger age might be attributable to increased effects of smoking on other physiological factors, such as body fat distribution, in younger people. Starting smoking at a younger age might be associated with greater dependence and heavy smoking patterns.

When we classify the glycaemic profiles separately for the three variables we note a higher prevalence of diabetes in current smokers when we consider Hb A1c levels (27% prevalence) than when we use fasting plasma glucose levels (10% prevalence) or post-prandial plasma glucose levels (only 6% prevalence). Similar result was observed by Dunstan DW et al in which the authors note that beneficial effect of preferentially identifying current smokers with high HbA1c and thus provide the possibility for early preventive measures with an encouragement to stop smoking.

Similar results were observed by Soulimane S et al, Inoue K and Borg R et al. They reported that identifying undiagnosed diabetes by HbA1c resulted in a 60% increase in prevalence in the study population. The authors found that HbA1c tended to be better at distinguishing between individuals at high and low risk of developing diabetes over a 10 years period as compared to fasting and 2 hours post-prandial plasma glucose levels. HbA1c assays are not affected by fasting status,

show little within day or day to day variation and assays are reliable and have high precision.

Smoking has been shown to be associated with an increased risk of developing Non-Insulin Dependent Diabetes Mellitus even after taking into account different confounding factors viz Rimm EB, Pery IJ and Feskens DJ et al, but a causal relation cannot be assumed. We need further evidence on the consistency of the association in different populations. We need data on the reversibility of the effect given the tendency towards weight gain on quitting smoking and well-designed clinical studies of the effects of acute and chronic smoking on insulin resistance. Several studies have shown that smoking reduces insulin sensitivity and this may be due to nicotine as it stimulates the secretion of insulin -antagonising hormones, which impair the action of insulin.³⁴ It is also probable that smokers have other unhealthy behaviours which are well recognised risk factors for diabetes. Therefore a simple causal model is unlikely. It is possible that smoking may ultimately emerge as a causal factor in diabetes in its own right via effects on glucose homeostasis and as a marker for additional causal factors such as physical inactivity and an atherogenic diet. The atherogenic effects of smoking may contribute to the effect of smoking on the risk of diabetes. Type 2 diabetes and cardiovascular disease are increasingly regarded as overlapping syndromes with common causal factors. Smoking is also in the frame and should be presumed guilty until proven innocent. Given the increasing prevalence of smoking in our country and a high burden of Non-Insulin Dependent Diabetes Mellitus along with a high genetic vulnerability to the disease these findings have public health implications and suggest that cessation of smoking should be considered as one of the key factors for diabetes prevention and treatment programmes.

CONCLUSION

Smoking Index was calculated for all smokers (smoking Index = number of bidis/cigarettes smoked per day x Total duration in years). The average smoking Index of the smokers (cases) in our study is 80.

Mean HbA1c fasting plasma glucose and 2 hour post prandial plasma glucose levels in smokers are substantially higher in smokers (cases) as compared to non-smokers (controls). P-values <0.05 in case of all three variable indicates a strong correlation between smoking and the observed increase in the values.

Mean HbA1c levels show an increasing trend with increasing age among both controls (non-smokers) and cases (smokers). Within any age group substantially higher mean HbA1c levels are seen among smokers as compared to non-smokers indicating a strong correlation between smoking and the observed increase in the values. Highest mean HbA1c values in case of both smokers and non-smokers are seen in the 61-70 years age group.

In the case of mean plasma glucose (fasting and post-prandial) levels the general trend is an increase in values with increasing age but the trend is not uniform. Among controls highest fasting and post-prandial glucose levels are observed in 41-50 and 61-70 years age group respectively. Among cases highest values of both variables are observed in the 61-70 years age group.

In the gender wise comparison the values of mean HbA1c and plasma glucose (fasting and post-prandial) levels are higher in smokers as compared to non-smokers among both female and male participants. Among females the increase in mean HbA1c values is statistically significant and shows a strong correlation with smoking whereas among males the increase in values of all three variables is significant and shows a strong correlation with smoking.

Mean HbA1c levels are higher in bidis smokers as compared to cigarette smokers and mean plasma glucose (fasting and post prandial) levels are higher in cigarette smokers as compared to bidis smokers. However the difference is not statistically significant and both bidis and cigarette smoking is equally harmful.

No correlation can be observed between the number of bidis/cigarettes smoked per day and the values of the three variables under study. Mean values of the three variables are highest among smokers who smoke 6-10 bidis/cigarettes per day. Similarly, in our study NO correlation was observed between the smoking index and mean HbA1c and plasma glucose (fasting and post prandial) levels.

Mean values of HbA1c and plasma glucose (fasting and post prandial) levels show an increasing trend as the number of years since the smokers have been smoking increases however this trend is not uniform. Highest values of the three variables are observed in the group of smokers who have been smoking for the past 16-20 years.

On classifying the glycaemic profile separately for the three variables we note a higher prevalence of diabetes in current smokers when we consider HbA1c levels (27% prevalence) than when we use fasting plasma glucose levels (10% prevalence) or 2 hours post prandial plasma glucose levels (6% prevalence). This indicates the beneficial effect of using HbA1c levels for identifying current smoker at risk of diabetes.

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REFERENCES

1. Narayan KM, Boyle JP, Geiss LS, Saddine JB, Thompson TJ. Impact of recent increase in incidence on future diabetes burden: U.S 2005-2050. *Diabetes care.* 2006;29:2114-6.
2. Shai I, Liang R, Manson JE. Ethnicity, obesity, and risk of type 2 diabetes in women: a 20-years follow up study. *Diabetes Care.* 2006;29:1585-90.
3. Ehasson B. Cigarette smoking and diabetes. *Prog Cardiovasc Dis.* 2003;45(5):405-13.
4. Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *American J Clinic Nutrit.* 2008;87(4):801-9.
5. Haire-Joshu D, Glasgow RE, Tibbs TL. Smoking and diabetes. *Diabetes care.* 2004;27(suppl. 1):574-5.
6. Simon D, Senam C, Saint GP, Paul M, Papoz I. Epidemiological features of glycated haemoglobin A1c- distribution in healthy population. The telecom study. *Diabetol.* 1989;32:864-9.
7. Modan M, Meytes D, Rozerman P, Significance of high Hb A1c levels in normal glucose tolerance. *Diabetes care.* 1988;11:422-8.
8. Nilsson PM, Land L, Pollare T, Berne C, Lithell HO. Increased level of hemoglobin A1c but not impaired insulin sensitivity found in hypertension and normotensive smokers *Metabolism.* 1995;44:557-61.
9. WHO Expert Committee on Diabetes Mellitus Technical Report Series 727, World Health Organisation, Geneva; 1985.
10. Nathan DM, Singer DE, Hurxthal K, Goodson JD. The clinical information value of the glycosylated hemoglobin assay. *N Engl J Med.* 1984;310:341-6.
11. Goldstein DE. Is glycosylated hemoglobin clinically useful? *N Engl J Med.* 1984;310:384-5.
12. Health and public policy committee, American college of physicians Glycosylated hemoglobin assays in the management and diagnosis of diabetes mellitus. *Ann Intern Med.* 1984;101:710-3.
13. Baynes JW, Bunn HF, Goldstein DE, Harris M, Martin DB, Peterson C, et al. National Diabetes Data Group : report of the expert committee on glycosylated hemoglobin. *Diabetes Care.* 1984;7: 602-6.
14. Santiago JV, Davis JE, Fisher F. Hemoglobin A1c levels in a diabetes detection program. *J Clin Endocrinol Metab.* 1978;47:578-80.
15. Dunn PJ, Cole RA, Soeldner JS, Gleason RE. Reproducibility of hemoglobin A1c and sensitivity to various degrees of glucose intolerance. *Ann Int Med.* 1979;91:390-6.
16. Kesson CM, Young RE, Talmar D, Whitelaw JW, Robb DA Glycosylated hemoglobin in the diagnosis of non-insulin dependent diabetes mellitus. *Diabetes Care.* 1982;5:395-8.
17. Simon D, Coignet MC, Thibult N, Senan C, Eschwege E. Comparison of glycosylated

- hemoglobin and fasting plasma glucose with two-hour post-load plasma glucose in the detection of diabetes mellitus *Am J Epidemiol.* 1985;122:589-93.
18. Little RR, England JD, Wiedmeyer HM, Mc Kenzie EM, Pettitt DJ, Knowler WC, et al. Relationship of glycosylated hemoglobin to oral glucose tolerance. Implications for diabetes screening. *Diabetes.* 1988;37:60-4.
 19. Bunn F. Evaluation of glycosylated haemoglobin in diabetic patients. *Diabetes.* 1981;30:613-7.
 20. Goldstein DE, Parker KM, England JD, England Jr JE, Wied-meyer HM, Rawlings SS, Hess R, Little RR, Simonds JF, Breyfogle RP Clinical application of glycosylated hemoglobin measurements. *Diabetes.* 1982;31(Suppl 3):70-8.
 21. Mortensen HB, Nielsen L, Seogaard U, Svendsen PAA, Nerup J. Comparison of six assays for glycosylated haemoglobin determination. *Scand J Clin Lab Invest.* 1983;43:357-62.
 22. Prignot J. Quantification and chemical markers of tobacco exposure. *Europ J Resp Dis.* 1987;70:1-7.
 23. Pakhale SS, Jayant K, Sanghvi LD. Chemical constituents of tobacco smoke in relation to habits prevalent in India. *Ind J Chest Dis and Alli Sci. (Special No.)* 1982:36-43.
 24. Soulimane S. Hb A1c fasting and 2 h plasma glucose in current, ex-and never smokers: a meta - analysis. *Diabetol.* 2014;57:30-9.
 25. Glumer C, Jogensen T, Borch-Johnson K. Prevalences of diabetes and impaired glucose regulation in a Danish population: the inter 99 study. *Diabetes care.* 2003;26:2335-40.
 26. Simon D, Senan C, Garnier P, Saint-Paul M, Papoz L Epidemiological features of glycated haemoglobin A1c- distribution in a healthy population. The Telecom Study. *Diabetol.* 1989;32:864-9.
 27. Herman WH, Ali MA, Aubert RE. Diabetes mellitus in Egypt: risk factors and prevalence. *Diabet Med.* 1995;12:1126-31.
 28. Gimeno SG, Ferreira SR, Franco LJ Hirai AT, Matsumura L, Moises RS. Prevalence and 7 -years incidence of type II diabetes mellitus in a Japanese-Brazilian population: an alarming public health problem *Diabetol.* 2002;45:1635-8.
 29. Stengard JH, Tuomilehto J, Pekkanen J Diabetes mellitus, impaired glucose tolerance and mortality among elderly men: the Finnish cohorts of the Seven Countries Study. *Diabetol.* 1992;35:760-5.
 30. Dunstan DW, Zimmet PZ, Welborn TA. The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)-methods and response rates. *Diabetes Res Clin Pract.* 2002;57:119-29.
 31. Deepa M, Pradeepa R, Rena M. The Chennai Urban Rural Epidemiology Study (CURES)-study design and methodology (urban component) (CURES-1). *J Assoc Physicians India.* 2003;51:863-70.
 32. Milson AS. The effect of nicotine on blood glucose levels and plasma non-esterified fatty acid levels in the intact and adrenalectomized rat. *Br J Pharmacol.* 1966;26:256-9.
 33. Sandberg H, Roman L, Zavodnick J, Kupers N. The effect of smoking on serum somatotropin, immunoreactive insulin and blood glucose levels of young adult males. *J Pharmacol Exp Ther.* 1973;184(3): 787-91.
 34. Facchini FS, Hollenbeck CB, Jeppesen J, Chen YD, Reaven GM. Insulin resistance and cigarette smoking *Lancet.* 1992;339:1128-30.
 35. Attvall S, Fowelin J, Lager I. Smoking induces insulin resistance - a potential link with the insulin resistance syndrome. *J Inter Med.* 1993;233:327-32.
 36. Mokded AH, Ford ES, Bowman BA. The continuing increase of diabetes in the US. *Diabetes Care.* 2001;24(2):412.
 37. Arnetz BB, Kallner A, Theorell T The influence of aging on hemoglobin A1c (Hb A1c). *J Gerontol.* 1982;37:648-50.
 38. Spence JC. Some observations on sugar tolerance with a special reference to variations found at different ages. *Quart J Med.* 1921;4:314-26.
 39. Tuomilehto J, Nissinen A, Kivela SL. Prevalence of diabetes mellitus in elderly men aged 65 to 84 years in eastern and western Finland. *Diabetol.* 1986;29:611-5.
 40. Wingard DL, Sinheimer P, Barrett-Connor EL, McPhillips JB. Community based study of prevalence of NIDDM in older adults. *Diabetes Care.* 1990;(Suppl 2):3-8.

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