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Prevalence of Insulin Resistance among Cigarette Smokers in Sokoto Metropolis

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Authors' contributions

This work was carried out in collaboration among all authors. Author SLK designed the study, wrote the protocol, and wrote the first draft of the manuscript. Authors TO, MKD, HB, CU and AIA managed the analyses of the study and literature searches, Author AIA performed the statistical analysis. All authors read and approved the final manuscript.

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ABSTRACT

Introduction: Cigarette smoking (CS) is a well-known risk factor for the development of metabolic diseases, various forms of cancer as well as insulin resistance (IR). IR is considered as an underlying derangement which very commonly aggravates metabolic syndrome.
Aim: This study assessed the prevalence of IR in cigarette smokers in Sokoto metropolis using selected surrogate markers.
Methodology: This cross sectional study was conducted in Sokoto among 108 subjects. Fasting venous blood samples were collected for plasma glucose, triglycerides and insulin estimation. Plasma glucose and serum triglycerides were analysed using enzymatic methods while insulin was assayed using ELISA method. Homeostasis model of assessment-IR (HOMA-IR), Quantitative insulin sensitivity check index (QUICKI), Mc Auley (McA) and fasting IR index (FIRI)

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were calculated using standard formula and IR cut-off of >2.5, <0.339, >5.8 and >2.3 respectively were used.
Results: Based on the cut off mark, the prevalence of IR for HOMA-IR, QUICKI, McA, FIRI indices were 62(57.4%), 66(61.1%), 39(36.1%) and 60(55.6%) respectively. There was a significant correlation between HOMA-IR and FIRI ($p< 0.05$, $r = 0.999$). HOMA-IR also had a significant correlation with McA ($p<0.05$ $r = -0.506$). QUICKI had a significant correlation with McA ($p<0.05$ and $r = 0.243$).
Conclusion: This study established a significantly high prevalence of IR among CS. Importantly, it can be concluded that cigarette smokers may be predisposed to the development of metabolic disease.

Keywords: HOMA-IR; QUICKI; Mc Auley; FIRI.

1. INTRODUCTION

Cigarette smoking (CS) remains a huge health burden and is still one of the leading preventable causes of morbidity and mortality globally [1]. It is a very wide spread activity and it consumption has reached the level of global epidemic [2]. It has been associated with an extensive list of disorder as well as reduction of life expectancy [3,4]. On average, cigarette smokers lose about 15 years of their life and an estimated 4 million cigarette smokers die worldwide annually [5]. CS kills more Americans than alcohol, car accidents, HIV, guns, and illegal drugs combined [6].

Globally, the World Health Organization (WHO) reported that nearly 47% and 12% of men and women respectively smokes cigarettes [7]. Although cigarette smoking is the most commonly used form of tobacco, the prevalence of cigarette smoking amongst adults has been declining in recent years. The prevalence of cigarette smoking has declined in the developed world while it is increasing rapidly throughout the developing world and is a major threat to current and future global health [8].

According to the 2015 National Health Interview Survey, the percentage of adults aged ≥18 years who smokes cigarettes was 15.1% in 2015, a decrease from 20.9% in 2005 [9]. In Nigeria, the percentage of current smokers has been put at 7.2% and 0.3% for men and women respectively [8]. However, Kaoje et al. (2015) reported the prevalence of cigarette smoking in Sokoto North Western Nigeria to be 7.1% [10].

CS affects the human body in myriad of ways, causing the development of chronic metabolic diseases like diabetes, heart disease, cancer because it induces insulin resistance (IR) and hypertriglyceridemia. Govindarajan et al. (2006) reported that about 25–47% of individuals with hypertension were insulin resistance. Similarly, there is considerable evidence for the increase prevalence of hypertension in diseases associated with insulin resistance like type 2 diabetes mellitus [11]. The effect of cigarette smoking on health of an individual depends on smoking duration over the years and exposure to cigarette smoke. Exposure to free radicals leading to increased oxidative stress, inflammation, and DNA damage are the possible mechanism by which cigarette smoke causes adverse health challenges [12].

IR is a condition characterized by lack of physiological response of peripheral tissues to insulin action [13]. It prevalence in the general population varies with the criteria used for its definition and the measurement adopted [14]. Govindarajan et al. (2006) reported an estimate that IR is prevalent in 30% of the adult population but acknowledges that IR is greater in metabolic diseases such as diabetes and cardiometabolic syndrome [14]. Accurate assessment of insulin sensitivity helps to identify individuals at increased risk of diseases, and may help target preventive and therapeutic efforts more effectively. The gold standard method is hyperinsulinemic euglycemic clamp**.** For epidemiologic and clinical studies surrogate markers are employed [12] which include measurement of Homeostasis model assessment-insulin resistance (HOMA-IR), Quantitative insulin sensitivity check index (QUICKI), McAuley (McA) index and Fasting insulin resistance index (FIRI).

2. MATERIALS AND METHODS

2.1 Subjects

In this cross sectional study, a total of 108 cigarette smokers of 18-59 years were recruited from different locations within Sokoto metropolis, Nigeria, between May and August, 2019.

2.2 Sample Collection and Analysis

After an overnight fast for 8-12 hours, 5ml of venous blood sample was collected aseptically. About 3ml and 2ml of the samples were dispensed into plain and fluoride oxalate containers for triglyceride (TG), insulin and glucose estimation respectively. The samples were spurned at 3000rpm for 5 minutes and unhaemolyzed plasma and serum were obtained respectively. These were harvested into labelled cryo-vials and then stored at -20^{o}C in a refrigerator until required for analysis.

Serum TG and plasma glucose were analysed using enzymatic methods [15], and serum insulin was assayed using ELISA [16].

2.3 Calculation of Selected Surrogate Markers of Insulin Resistance

2.3.1 HOMA-IR

HOMA-IR is calculated with the following mathematical expression;

IR_{HOMA} = [Fasting insulin (mU/l) × Fasting glucose (mmol/l)]/22.5

Normal value = < 2.5

Subjects were considered IR with value above the cut-off 2.5 [17].

2.3.2 QUICKI

QUICKI = 1/[(log fasting plasma insulin (μU/ml) + log fasting plasma glucose (mg/dl)]

Normal value = 0.382±0.007

Subjects were considered IR when the cut-off is ≤ 0.339 [12].

2.3.3 McA index

McA = $e^{(2.63-0.28\ln(\mathrm{Io})-0.31\ln(\mathrm{TGo})}$

Normal value = > 5.8

Subjects were considered insulin resistance with cut-off value less than 5.8 [12].

2.3.4 Fasting insulin resistance index (FIRI)

The fasting insulin resistance index (FIRI) was formulated [18]

FIRI is calculated as = (fasting glucose × fasting insulin)/25.

Normal value = < 2.3

Subjects were considered insulin resistance if the cut-off value is greater than 2.3 [19].

2.4 Statistical Analysis

The data generated were analyzed using SPSS Software Version 25. The results were expressed as MEAN±SD. p-value ≤ 0.05 was considered statistically significant. Pearson's correlation was used to determine the strength of relationship between insulin resistance surrogate markers and other variables. Frequency distribution was used to determine the percentage prevalence of the variable in the study participants.

3. RESULTS

This study established a significantly high prevalence of IR among CS. Based on the cut off mark, the prevalence of IR for HOMA-IR, QUICKI, McA, FIRI indices were recorded.

Table 1. Sociodemographic distribution of the study participants

Variables	Frequency (n=108)
Gender	**n (%)**
Male	102 (94.4%)
Female	6 (5.6%)
Age group	
<20	1 (0.9%)
20-29	52 (48.1%)
30-39	35 (32.4%)
40-49	13 (12%)
50>	7 (6.5%)
Ethnicity	
Hausa	84 (77.8%)
Yoruba	6 (5.6%)
Igbo	3 (2.8%)
Others	15 (13.9%)
Occupation	
Farming	3 (2.8%)
Pettytrader	47 (43.5%)
Civilservice	36 (33.3%)
Unemployed	7 (6.5%)
Student	15 (13.9%)

. n=total number

4. DISCUSSION

IR is increasing not only in African population but globally due to sedentary lifestyle including

Table 2. Prevalence of insulin resistance

Surrogate markers	Insulin resistance (n=108)	Non- insulin resistance (n=108)
HOMA-IR	62 (57.4%)	46 (41.8%)
QUICKI	66 (61.1%)	42 (38.9%)
Mc Auley	39 (36.1%)	69 (63.9%)
FIRI	60 (55.6%)	48 (44.4%)

HOMA-IR= Homeostasis model of assessment insulin resistance; QUICKI = Quantitative insulin sensitivity check index; FIRI= Fasting insulin resistance index

Table 3. Correlation between the biomarkers of insulin resistance

		HOMA-IR	QUICKI	McAuley	FIRI
HOMA-IR	r-value	1	0.059	-0.506**	0.999**
	P-value		0.542	0.000	0.000
QUICKI	r-value	0.059	1	0.243*	0.062
	P-value	0.542		0.011	0.525
McAuley	r-value	-0.506**	0.243	1	-0.504**
	P-value	0.000	0.011		0.000
FIRI	r-value	0.999**	0.062	-0.504**	1
	P-value	0.000	0.525	0.000	

*r=Pearson correlation; **Correlation is significant at the 0.01 level (2-tailed); *Correlation is significant at the 0.05 level (2-tailed)*

cigarette smoking, a major health concern and a well-known risk factor for metabolic disease. Nicotine content of cigarette is known to increase sympathetic activity, raise circulating levels of catecholamines, growth hormone, adrenocorticotropic hormone, cortisol, prolactin, and beta-endorphin, and decrease oestrogen levels all which are strongly antagonistic to insulin's action. Thus, smoking leads to decrease insulin production, decrease glucose catabolism and increase glucose accumulation in the body.

This study showed a high prevalence of IR base on calculation of HOMA-IR, FIRI, and QUICKI using the standard cut off point, while a lower prevalence rate was observed in McA index with 39 (36.1%) of participants. This is in contrast to the result [20] who reported higher prevalence of IR in type 2 diabetes mellitus base on McA (81%), and HOMA-IR and QUICKI (93%).

The differences might be due to different study participants and there seems to be paucity of published data on the prevalence of insulin resistance in cigarette smokers. This could also be due to the fact that the prevalence of insulin resistance in the general population varies with the criteria used for its definition and the measurement adopted.

The result further shows that HOMA-IR had a significant negative correlation with McA and a significant positive correlation with FIRI ($p< 0.05$, $r = -0.506$ and $p< 0.05$, $r = 0.999$) respectively. QUICKI had a significant positive correlation with McA ($p<0.05$, $r = 0.234$), while McA has shown to have a significant negative correlation with FIRI index ($p<0.05$, $r = -0.505$).

5. CONCLUSION

There is high prevalence of insulin resistance in cigarettes smokers in Sokoto metropolis. Hence, it can be deduced that cigarette smoking is one of the risk factors in the development of T2DM, and also associated with the prediction of cardiovascular disease.

CONSENT AND ETHICAL APPROVAL

Ethical approval was obtained from the Sokoto State Ministry of Health (SKREC/037/018). Written informed consent was obtained from all participants prior to the sample collection by filling a standard informed consent form by themselves or through an interpreter.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Mpabulung L, Muula AS. Tobacco use among high school student in Kampala, Ugandar questionnaire study. Croatica Medical Journal. 2004;43:80-83.
2. Can G, Topbes M, Ozutuna F, Ozgun S, Can E, Yavuzyilma A. Factors contributing to regular smoking in adolescent in Turker. Journal School of Health. 2009;79:93-97.
3. Detel R. Oxford textbook of public health. 4th Ed. Oxford University Press Oxford, United Kingdom. 2002;40.
4. Doll R, Boreham PJ, Sutherland I. Mortality in relation to smoking, 50 years observation on male British doctors. 2004; 238(10):1136.
5. Global Youth Tobacco Survey Collaborative Group (GYTS). Tobacco use among youth. A cross country comparison tobacco control. 2002;11:252-270.
6. America cancer society America cancer society position paper on electronic cigarettes cancer; 2018. Available:org/healthy/stay-away-from-tobacco/e-cigarette-position-statement.html
7. Bago BJ. Prevalence of cigarette smoking and its associated risk factors among students of Hawassan University, college of medicine and health sciences. Journal of Addiction Research and Therapy. 2017; 8(4):331.
8. Adunmo GO, Adesokan AA, Biliaminu SA, Abdulazeez IM, Adunmo EO. Effect of chronic cigarette smoking on lipid profile – A pilot study in North-Eastern Nigeria. Sokoto Journal of Medical Laboratory Science. 2016;1(1):94 – 99.
9. Onor ICO, Stirling DL, Williams SR, Bediako D, Borghol A, Harris MB, Darensburg TB, Clay SD, Okpechi SC, and Sarpong DF. Clinical effects of cigarette smoking: Epidemiologic impact and review of pharmacotherapy options. International Journal of Environmental Research and Public Health. 2017;14: 1147.
10. Kaoje AU, Sabir AA, Yusuf S, Jimoh AO, Raji MO, Ango UM, Magaji BA. Tobacco consumption prevalence and pattern among residents of Sokoto Metropolis, Northwestern Nigeria. African Journals Online. 2015;12(1):981-987.
11. Govindarajan G, Gill H, Rovetto M, Sowers JR. What is insulin resistance? University of Missouri School of Medicine, Departments of Internal Medicine and Medical Pharmocology and Physiology, and Harry S. Truman VA Medical Center, Columbia, MO, USA. Heart Metabolism. 2006;30:30–34.
12. United State Department of Health and Human Services (USDHHS). The health consequences of smoking-50 years of progress: A report of the surgeon general; Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, office on smoking and health: Atlanta, GA, USA. 2014;1–36.
13. Ascaso JF, Parbo S, Real JT, Lorente RL, Priego A, Carmena R. Diagnosing insulin resistance by simple quantitative methods in subjects with normal glucose metabolism. Diabetes Care. 2003;20(12): 3320-3325.
14. Yi KH, Hwang JS, Kim EY. Prevalence of insulin resistance and cardiometabolic risk in Korean children and adolescents: A population-based study. Diabetes Research Clinical Practice. 2014;103(1): 106-113.
15. Trinder P. Determination of blood glucose using an oxidase-peroxidase system with a non- carcinogenic chromogen. Journal of Clinical Pathology. 1969;22(2):158-161.
16. Turkington RW, Estkowksi A, Link M. Secretion of insulin or connecting pepetide: A predictor of insulin dependence of obese diabetics. Archives of Internal Medicine. 1982;142:1102-1105.
17. Gutch M, Kumar S, Razi SM, Gupta KK, Gupta A. Assessment of insulin sensitivity and resistnce. Indian Journal of Endocrinology Meatbolism. 2015;19(1): 160-164.
18. Duncan MH, Singh BM, Wise PH, Carter G, Alaghband- Zadeh J. A simple measure of insulin resistance. Lancet. 1995;346: 120-121.
19. Bhowmik B, Siddique T, Mujumder A, Rajib MMR, Das CK, Khen MC, Khen AKA, Hussein A. Identifying insulin resistance by fasting blood samples in Bangladesh population with normal blood glucose. Journal of Diabetology. 2016; 7(3):4.

20. Hettihewa M, Palansasingle S, Jayasingle, SS, Gunasekara SW, Weerarathna TP Comparison of insulin resistance by indirect method, homeostasis model assessment, quantitative insulin sensitivity check index and McAuley with Fasting insulin in patient with type 2 diabetes mellitus in Gelle, Sri lanka; A pilot study. Online Journal of Health Allied Science. 2006;5(1):0972-5997.

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