

Evaluation of Cardiovascular Status of Apparently Healthy Sedentary Subjects in Nnewi, Anambra State Using Framingham Risk Score Calculator

Ezeugwunne IP^{1*}, Ogbodo EC², Nwankwo EC¹, Analike RA³, Onah CE³, Okwara JE², Amah UK², Oguaka VN¹, Asebioyo SJ⁴, Meludu SC^{1,2}.

¹Department of Human Biochemistry; College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Nigeria

²Department of Medical Laboratory, College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Nigeria

³Department of Chemical Pathology, College of Health Sciences, Nnamdi Azikiwe University Teaching Hospital, Nnewi Campus, Nigeria.

⁴Department of Human Biochemistry; College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Nigeria

* Corresponding author: Dr. I.P. Ezeugwunne, Email: goodnessifeoma007@yahoo.com

Abstract:

Introduction: Sedentary lifestyle is a lifestyle with no or irregular physical activity, found in both individuals that live in developed and developing world.

Objective: The study was done to evaluate the cardiovascular status of apparently healthy subjects who reside in Nnewi, South Eastern, Nigeria.

Method: Based on the fasting blood sugar (FBS), subjects were classified into diabetics and non-diabetics. Using standard routine analysis for FBS the subjects were grouped into 50 adult diabetic and 50 non-diabetic subjects which serve as control group. Ethical approval was obtained from the Ethics Review Committee and written informed consent was sort from each participant. Also, questionnaire was used to obtain their Biodata and risk levels of both modifiable and non-modifiable factors. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using sphyngnomanometer. Fasting lipid profile was assayed using standard routine analysis. The cardiovascular risk score was calculated using Framingham risk calculator to determine the risk of the subjects to cardiovascular diseases in the next 10 years. Student t-test was used for data analysis.

Results: The serum level of FBS was significantly higher in diabetic subjects than in control subjects ($p < 0.05$). The SBP and DBP values were significantly higher in diabetic subjects than in control subjects ($p < 0.05$). The mean age was significantly higher in diabetic subjects than in control subjects ($p < 0.05$). The serum levels of Triglycerides (TG), Low density lipoprotein (LDL) and Total cholesterol (T chol) were significantly higher in diabetic subjects than in control subjects ($p < 0.05$). But

the serum level of High density lipoprotein (HDL) was significantly lower in diabetic subjects than in control subjects ($p < 0.05$). The Framingham cardiovascular risk score was significantly higher in diabetic subjects than in control subjects ($p < 0.05$).

Conclusion: The findings showed that diabetic subjects may likely die from cardiovascular diseases than apparently healthy individuals.

Keywords: Cardiovascular diseases, sedentary lifestyle, Framingham risk calculator.

1. INTRODUCTION

A lifestyle denotes the interests, opinions, behaviours of an individual, group or culture [1]. A sedentary lifestyle is a life an individual lives that lacks physical activity and it is a risk factor for anxiety, lipid disorders, [2] cardiovascular diseases, [3] depression, diabetes, high blood pressure [3], [4] and obesity [5].

A sedentary activity include sitting, reading, watching television, playing video games and computer use for much of the day with little or no vigorous physical exercise [6]. A healthy or unhealthy lifestyle will most likely be transmitted across generations [7]. Reports have it that high income parents are more likely to eat organic food, have time to excise and provide the best living condition to their children than low income parents who are more likely to engage in unhealthy activities like smoking, alcohol consumption to help them release poverty-related stress and depression [7].

Coronary heart disease (CHD) begins with damage to the lining and inner layers of the heart arteries. Factors due to lifestyles such as smoking, high fats content in food and cholesterol, high blood pressure, high blood

sugar due to insulin resistance or diabetes [5], [8] cause coronary heart diseases. CHDs are the number one cause of death globally.⁹ Most heart diseases can be prevented by addressing behavioural risk factors such as tobacco use, unhealthy diet, physical inactivity, obesity and harmful alcohol intake [8].

Diabetes mellitus (DM) is a chronic metabolic disorder caused by an increase in blood glucose level due to either partial or complete absence of insulin secretion, which affects more than 300 million people in the world [10]. Type 2 diabetes (90- 95% of cases) results from a combination of the inability of muscle cells to respond to insulin properly (insulin resistance) and inadequate compensatory insulin secretion. diabetes can also result from genetic defects in insulin action [11] and infections [12].

Exercise and diet are very essential in the management of type 2 diabetes caused by insulin resistance by controlling high glucose level, lipid and blood pressure abnormalities and losing excess weight [13]. Exercise causes increased glucose uptake into active muscles to fuel muscular activity as intensity of exercise increases [13]. Contraction of muscle during exercise increase glucose transport into the muscle without insulin and increased GLUT4 to the plasma membrane to increase the effectiveness of insulin [14], [15].

Early exercise training improves the metabolic status and insulin sensitivity of people, thereby reducing their risk of developing type 2 diabetes later in life [16]. Numerous reports have shown that increased daily exercise can improve glucose control and reduce medication requirements in those with type 2 diabetes [17]. Hence, a lifestyle that is complementary with moderate exercise will improve the overall health and wellness and reduce the risk of heart disease and stroke [18].

Multivariate risk scores have been used to predict CVD risk in individuals with diabetes [19], although fasting or random fasting blood sugar values are not part of the Framingham risk score calculation. There are a large number of scores for the general population but the most commonly used score is that of Framingham risk score [20], [21].

2. MATERIALS AND METHODS

A total of 100 adults apparently healthy individuals aged 35 years and above, residing in Nnewi, Anambra State were randomly recruited for this study. In the course of study, diabetics were discovered based on

test results. So, 50 diabetics were identified and 50 non-diabetics were also identified and served as controls. Questionnaire was used to collect individual biodata and lifestyle status which was used in the Framingham risk calculator.

Five (5) millilitres of blood sample were collected from each subject and serum FBS was analysed immediately using a glucometer (Life Scan Inc, Johnson- Johnson Company, USA) by the one- touch blood glucose monitoring system [22] and the presence of diabetes was defined using WHO recommendations [23].

The remaining blood sample was introduced into a plain tube, allowed to clot, centrifuged and the serum aspirated into plain tube for lipid profile assay. Total cholesterol was estimated by enzymatic hydrolysis and oxidation of cholesterol by the method of Allain *et al*, [24] Serum Triglyceride was estimated by enzymatic hydrolysis by the method of Buccolo and David [25], HDL was estimated by precipitation method described by Assmann *et al*, [26] and LDL was estimated and was calculated as described by Kaplan *et al*, [27] Randox Laboratory Ltd., UK, commercially prepared the reagents used.

High blood pressure was defined according to the WHO guideline: a SBP \geq 140mm/Hg or DBP \geq 90mm/Hg or being on treatment [28]. The SBP and DBP of the subjects were measured using sphygmomanometer and recorded accordingly.

The data collected from this study were keyed into the risk score Framingham calculator, which provides coronary heart disease risk at 10 years in percentage [29]. The statistical analysis was carried out using SPSS version 16. Data were expressed as mean \pm SD. Test of significance of mean was by Levene's test for equality of variance. The acceptable level of significance was $p < 0.05$.

3. RESULTS

Table-1 shows the mean levels of the Framingham cardiovascular risk score in the subjects studied. The mean serum level of FBS was significantly higher in diabetic subjects than in control subjects ($p < 0.05$). The SBP and DBP values were significantly higher in diabetic subjects than in control subjects ($p < 0.05$). The mean age was significantly higher in diabetic subjects than in control subjects ($p < 0.05$). The serum levels of TG and Total cholesterol were significantly higher in diabetic subjects than in control subjects ($p < 0.05$) respectively. The serum level of HDL was significantly

lower in diabetic subjects than in control subjects ($p < 0.05$). The Framingham cardiovascular risk score was significantly higher in diabetic subjects than in control subjects ($p < 0.05$).

Table 1: Mean Framingham Cardiovascular Risk Factors

Risk factors	Diabetics (n=50)	Controls (n=50)	p-value
TG (mmol/l)	1.33 ± 0.41	0.92 ± 0.46	<0.05
LDL (mmol/l)	2.43 ± 0.74	2.34 ± 1.01	>0.05
Total chol (mmol/l)	4.68 ± 0.09	4.47 ± 1.39	<0.05
HDL (mmol/l)	0.89 ± 0.42	1.40 ± 0.45	<0.05
FBS (mmol/l)	6.67 ± 3.08	4.74 ± 0.86	<0.05
SBP (mm/Hg)	136.62 ± 77.48	130.48 ± 54.70	<0.05
DBP (mmol/l)	77.48 ± 13.65	70.14 ± 8.39	<0.05
Framingham Score (%)	19.16 ± 8.72	5.67 ± 4.40	<0.05
Age (Years)	55.54 ± 7.97	46.70 ± 12.24	<0.05

4. DISCUSSION

This study revealed that serum level of FBS was elevated in diabetic subjects than in apparently healthy individuals. The mean FBS of the control group was within this normal range. Globally, estimated 422 million adults are living with diabetes mellitus [11], [30]. Reports have it that individuals with diabetes have two-fold higher risk of developing coronary heart disease than non-diabetic subjects [31]. diabetes mellitus is a group of metabolic disease with high blood sugar over a prolonged period [11], [30]. The most prevalent form of diabetes mellitus is type 2, which accounts for at least 90 % of all cases of diabetes mellitus [32]. Type 1 account for 10 % of all cases of diabetes mellitus, caused by a complex disease process where genetic and environmental factors lead to an autoimmune response that remains to be fully elucidated. The process contributes to the destruction of beta cells of the pancreas with no production of insulin [33], so the body needed daily insulin injection. It is diagnosed during childhood or early adolescence and it affects about 1 in every 600 children [34].

Type 2 diabetes mellitus is a heterogeneous disorder caused by a combination of genetic factors related to impaired insulin secretion, insulin resistance and environmental factors such as obesity, over eating, lack of exercise, stress and aging [35]. In developing countries, people aged 45 to 65 years are affected most, compared with those older than 60 years in developed countries [35].

Studies have it that longstanding diabetes causes cardiovascular diseases but tight glycaemic control

helps prevent long- term cardiovascular events in people newly diagnosed with type 2 diabetes but has a limited effect on cardiovascular outcomes in the short-term (3-5 year) [36]. The normal FBS range for an adult without diabetes is 3.8 – 5.5 mmol/l [13].

In this study, the mean SBP and DBP values were significantly higher in diabetic individuals than in the control subjects. Hypertension or high blood pressure, sometimes called arterial hypertension is a chronic medical condition in which the blood pressure in the arteries is elevated. This condition requires the heart to work harder than normal to circulate blood through the blood vessels. Blood pressure involves two measurements, systolic- which is the blood pressure when the muscle is contracting and diastolic- which is the blood pressure when the muscle is relaxing. High blood pressure is said to be present if it is persistently at or above 140/90 mmHg. Hypertension is a major risk factor for stroke, myocardial infarction (heart attacks), peripheral arterial disease and is a cause of chronic kidney disease [37]. The serum levels of TG, LDL and total cholesterol were significantly higher in diabetic subjects than in control subjects. But the serum level of HDL was significantly lower in diabetic subjects than in control subjects. Clinically, dyslipidemia has been correlated with atherosclerosis and up to 97 % of patients with diabetes are dyslipidemic [38].

The mean age was significantly higher in diabetic subjects than in the control subjects. Age is among the important factor in developing cardiovascular disease with approximately a tripling of risk with each decade of life [39]. One of the reasons is related to serum total cholesterol level. In most population, the serum total cholesterol level increases as age increases [40], [41]. Ageing is also associated with loss of arterial elasticity and reduced arterial compliance and may lead to coronary artery disease [42].

The Framingham risk score is a gender-specific algorithm used to estimate the 10 year cardiovascular risk of an individual. The framingham cardiovascular risk score calculation in % was equated to points total in both men and women. The calculation took cognizance of age, total cholesterol (mg/dl), cigarette smokers and non-smokers, HDL cholesterol (mg/dl), SBP (mmHg) with respect to treatment and untreated subjects [43],[44].The framingham cardiovascular risk score was significantly higher in diabetic subjects than in control subjects after the computation of risk factors obtained in the study.

The modifiable risk factors such as FBS, SBP, LDL and total cholesterol that lead to cardiovascular diseases were found to be raised in diabetic subjects than in apparently healthy individuals. Also, age the non-modifiable risk factor was significantly observed to contribute to increase in Framingham risk score in diabetic subjects than in apparently healthy individuals. This may indicate that diabetic subjects are more likely to die of cardiovascular diseases within 10 years if their conditions are not well managed than in apparently healthy individuals.

5. CONCLUSION

An individual can prevent risk for future cardiovascular events by modifying their sedentary lifestyle by eating healthy diet low in sugar, alcohol, have regular exercise and have preventive medical treatment for hypertension and diabetes mellitus.

REFERENCES

- [1] Lynn R and Close AG. Consumer Behaviour Knowledge for Effective Sports and Events. New York: Routledge 2011; ISBN 978-0-415-87358-1.
- [2] World Health Organization (WHO). Physical activity. 2011; Retrieved May, 2010.
- [3] World Health Organization (WHO). Physical activity a leading cause of disease and disability. 2010b; Retrieved June, 2010.
- [4] National Institute of Health. Who is at risk for high blood pressure?. 2010; Retrieved April, 2010.
- [5] Kabiri N, Asgary S, Madani H, Mahzouni P. Effects of *Amaranthus caudatus* extract and lovastatin on atherosclerosis in hypercholesterolemic rabbits. *Journal of Medicinal plants Respiration* 2010; 4 (5): 355-381.
- [6] Lopez A.D, Mathers C.D, Ezzati M, Jamison D.T, Murray C.J. Global and regional burden of disease and risk factors, 2001 systemic analysis of population health data. *Lancet* 2001; 367 (9524): 1747- 1757.
- [7] Case A, Lubotsky D, Paxson C. Economic Status and Health in Childhood: The origins of the Gradient. *The American Economic Review* 2002; 92 (5): 1308 – 1337.
- [8] Guldiken S, Demir M, Anka E, Tugrul, A. The level of serum high sensitive C-Reactive Protein in women with hyperthyroidism. *Journal of Clinical Endocrinology and Metabolism* 2005;3: 85-88.
- [9] Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, Abraham J, Adair T, Aggarwal R, Ahn SY, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; 380 (9859): 2095–20128.
- [10] Sherwin R, Jastreboff AM. Year in diabetes 2012: The diabetes tsunami. *Journal of clinical endocrinology metabolism* 2012; 97: 4293 – 4301.
- [11] WHO, 2014. About Diabetes. Retrieved April, 2014.
- [12] Atkins R.C and Zimmet P. Diabetic kidney disease: act now or pay later. *Saudi Journal of kidney and transplantation* 2010; 21 (2): 217 – 221.
- [13] American Diabetes Association, Standards of Medical Care in Diabetes. *Diabetes Care* 2011; 34 (suppl): 511 – 561.
- [14] Goodyear L.J. AMP-activated protein kinase (AMPK) is activated in muscle of subjects with type 2 diabetes during exercise. *Diabetes* 2001; 50: 921 – 927.
- [15] Wilkin T.J. The accelerator hypothesis: a unifying explanation for type-1 and type-2 diabetes. *Nestle Nutrition Workshop Service in Clinical Performance Programme* 2006; 11: 139 – 150; DISCUSSION 150- 153 (pmid: 16820737 DOI: 10.1159/000094447).
- [16] Hu F.B, Manson J.E, Stampfer M.J, Colditz G, Liu S, Solomon C.G, Willet W.C, Manson S, Colditz L, Solomon W. Diet, lifestyle and the risk of type 2 diabetes mellitus in women. *The New England Journal of Medicine* 2001; 345 (11): 790 – 797.
- [17] Slentz C.A, Tanner C.J, Bateman LA, Durham M.T, Huffman K.M, et al). Effect of exercise training intensity on pancreatic beta-cell function. *Diabetes Care* 2009: 1807 – 1811.
- [18] Stampfer M.J, Hu F.B, Manson J.E, Rimm E.B, Willett W.C et al. Primary Prevention of Coronary Heart Disease in Women through Diet and lifestyle. *New England Journal of Medicine* 2000; 343 (1): 16 – 22.
- [19] Juutilainen A, Lehto S, Ronnema T, Pyörälä K, Laakso M. Type-2 Diabetes as a coronary heart disease equivalent: an 18 years prospective population-based study in Finnish subjects. *Diabetes Care* 2005; 28 : 2901 – 2907.

- [20] Stevens R.J, Coleman R.L, Holman R.R (2005). Framingham risk equations underestimate coronary heart disease risk in diabetes. *Diabetes Medicine* 22: 228.
- [21] Eichler K, Puhon M.A, Steurer J, Bachmann L.M. Prediction of coronary events with the Framingham score: a systematic review. *Journal of American Heart* 2007; 153: 722 – 731.
- [22] Mark S.V, Dawson A. Rapid stick methods for determining of blood glucose concentration. *British Medical Journal* 1965;1: 293.
- [23] World Health Organization (WHO). Part 1: Diagnosis and Classification of Diabetes mellitus, Report of WHO consultation. Geneva: WHO 1999.
- [24] Alain B. Leucocyte typing: Human leucocyte differentiation antigens detected by monoclonal antibodies: Specification, classification, nomenclature. Report on the first international references workshop sponsored by INSERM, WHO and IUIS, Berlin. Springer 1974. 45-48.
- [25] Buccolo G and David H. Quantitative determination of serum triglycerides by the use of Enzymes. *Journal of Clinical Chemistry* 1973; 19: 476-482.
- [26] Assmann G, Jabs H U, Nolte W, Schriewer H. LDL-cholesterol determination in blood serum following precipitation of LDL with polyvinyl sulphate. *Clinica Chimica Acta* 1984; 140: 77 -88.
- [27] Kaplan A, Szabo L, Opheim K. Clinical chemistry interpretation and techniques. 3rd ed. Published by Lea and Febiger at 600 Washington Square Philadelphia, USA 1983; pp 307 – 316.
- [28] World Health Organization (WHO). International society of Hypertension (ISH). Statement on management of Hypertension. *Journal of Hypertension* 2003; 21: 1983 – 1992.
- [29] Aberg JA. Lipid Management in Patients Who Have HIV and Are Receiving HIV Therapy. *Endocrinology and metabolism clinics of North America* 2009; 38 (1): 207 – 222.
- [30] WHO, 2016. Global Report on Diabetes. Accessed 30 August, 2016.
- [31] Kannel W.B. Historic perspectives on the relative contributions of diastolic/systolic blood pressure elevation to cardiovascular risk profile. *American Heart Journal* 1999; 138: 205 – 210.
- [32] Williams textbook of endocrinology. Philadelphia: ELSEVIER/ Saunders. 12th ed. Pp. 1371- 1435. ISBN 978114377 03245.
- [33] Shoback, edited by David G. Gardner D. Greenspan's basic and clinical endocrinology. New York: McGraw- Hill Medical 2011; 9th ed. Chapter 17. ISBN 0071622438.
- [34] Stang J and Story M. Guidelines for Adolescent Nutrition Services 2005; 167.
- [35] Centers for Disease Control and Prevention (CDC). National Diabetes Statistics Report: Estimates of Diabetes and its Burden in the United States 2014; Atlanta, GA, US Department of Health and Human Services.
- [36] Turnbull F.M, Abaira C, Anderson R.J et al. Intensive glucose control and macrovascular outcomes in type 2 diabetes. *Diabetologia* 2009; 52:2288 – 2298.
- [37] Carretero O.A and Suzanne O. Essential hypertension. *Circulation* 2000; 101: 329 – 335.
- [38] Fagot-Campagna A, Rolka DB, Beckles GL, Gregg E.W, Narayan K.M. Prevalence of lipid abnormalities, awareness and treatment in US adults with diabetes . *Diabetes* 2000; 49 (Suppl): A78.
- [39] Finegold J.A and Ascaria P, F. Mortality from ischaemic heart disease by country region and age statistics from world Health Organisation and United Nation. *International Journal of Cardiology* 2002; 168 (2): 934 - 945.
- [40] Finks SW, Airee A, Chow SL, Macaulay TE, Moranville MP, Rogers KC, Trujillo TC. Key articles of dietary interventions that influence cardiovascular mortality. *Pharmacotherapy* 2012; 32 (4): e54-87.
- [41] Micha R, Michas G, Mozaffarian D. Unprocessed and processed meats and risk of coronary artery disease and type 2 Diabetes- an updated review of the evidence. *Current atherosclerosis reports* 2012; 14 (6): 515-524.
- [42] Go A.S, Mozaffarian D, Roger V.L, Benjamin E.J, Berry J.D, Borden W.B, Bravata D.M. Heart disease and stroke statistics 2013 update: a report from the American Heart Association. *Circulation* 2013; 127 (1): e6- e245.

- [43] Wilson P.W, D'Agostino R.B, Levey D, Belanger A.M, Silbershatz H, Kannel W.B. Predictions of coronary heart disease using risk factors categories. *Circulation* 1998; 97 (18): 1837 – 1847.
- [44] D'Agostino R.B, Vasani R.S, Pencina M.J, Wolf P.A, Cabain M, Massaro J.M, Kannel W.B. General cardiovascular risk profile for use in primary care: the Framingham Heart study. *Circulation* 2008; 117 (6): 743 – 753