



Journal of Medical Sciences

ISSN 1682-4474

science
alert

ANSI*net*
an open access publisher
<http://ansinet.com>

JMS (ISSN 1682-4474) is an International, peer-reviewed scientific journal that publishes original article in experimental & clinical medicine and related disciplines such as molecular biology, biochemistry, genetics, biophysics, bio-and medical technology. JMS is issued eight times per year on paper and in electronic format.

For further information about this article or if you need reprints, please contact:

E.E.J. Iweala
Covenant University Public
Health and Wellbeing Research
Group (CUPHWERG),
Covenant University,
Ota, Ogun State, Nigeria

Tel: +234(0)8036738625

Propensity for Diabetes and Correlation of its Predisposing Factors in Ota, Nigeria

^{1,5}E.E.J. Iweala, ^{1,5}S.N. Chinedu, ^{1,5}I.S. Afolabi, ^{1,5}O.O. Ogunlana,
^{2,5}D.E. Azuh, ^{3,5}V.C. Osamor and ^{4,5}T.A. Toogun

Body Mass Index (BMI) and Random Blood Glucose (RBG) are considered important predisposing factors for type 2 diabetes mellitus in adults. This study assessed the propensity to become diabetic based on the relationship between Body Mass Index (BMI), Random Blood Glucose (RBG), gender and age in a community in South west Nigeria. The study included a convenient sample size of 140 healthy adult individuals who met the inclusion criteria. Anthropometric indices including height and weight were measured and Blood samples analyzed for random blood glucose. A significant positive correlation was observed ($r = +0.32$) between BMI and RBG in females while there was no correlation in the males ($r = -0.05$). The males were found to be less likely to be diabetic than the females. The relationship between age and RBG was significantly positive in both males and females. The study confirms the hypothesis that a positive correlation exist between BMI and RBG but only in women. This suggests that other causes including sex could predispose to diabetes and reiterates the diabetogenic effect of adiposity.

Key words: Body mass index, obesity, random blood sugar, age, diabetes mellitus

¹Department of Biological Sciences,

²Department of Economics and Development Studies,

³Department of Computer and Information Sciences,

⁴Covenant University Health Center,

⁵Covenant University Public Health and Wellbeing Research Group (CUPHWERG), Covenant University, Ota, Ogun State, Nigeria

INTRODUCTION

Body Mass Index (BMI) associated with a healthy body weight is the ratio of weight in kilograms to the square of height in meters (kg m^{-2}) (Hu, 2008). Persons can be categorized as underweight, normal, overweight and obese based on BMI ranges of ≤ 18.4 , 18.5-24.9, 25 to 29.9 and ≥ 30 , respectively (Sturm, 2007; World Health Organization, 1997). A high BMI value is due to genetic and environmental factors and is considered a risk factor for several diseases and medical problems including heart disease, stroke and carcinomas (Whitlock *et al.*, 2009; Yang *et al.*, 2007; Grundy, 2004). A raised fasting or random blood glucose level is associated with type 2 diabetes mellitus, a disease caused by both genetic factors and lifestyle linked to obesity (Shoelson *et al.*, 2007; Bray, 2004). A positive correlation is generally assumed to exist between BMI and Random Blood Glucose (RBG) levels (Diaz *et al.*, 2007). According to a World Health Organization (WHO) study group and American Diabetes Association (ADA), various unstandardized random blood sugar values can be used in diagnosis and classification of diabetes. RBG above 200 mg dL^{-1} is classified as likely to be diabetic, less than 200 mg dL^{-1} as uncertain to be diabetic and less than 80 mg dL^{-1} as unlikely to be diabetic (World Health Organization, 1985). RBG between 70-140 mg dL^{-1} is normal, 140-200 mg dL^{-1} is pre-diabetes and above 200 mg dL^{-1} is diabetes (American Diabetes Association, 2006). Generally prevalence and complications of diabetes is more pronounced in females than males as result of gender associated adiposity (World Health Organization, 2002; Icks *et al.*, 1997; Howard *et al.*, 1998; Wishner, 1996). Overweight and obesity are risk factors in the pathogenesis of type 2 diabetes due to insulin resistance and excess cortisol (Steppan *et al.*, 2001; Connell *et al.*, 1986). Obesity and diabetes have become major global public health challenges (Fauci *et al.*, 2008; World Health Organization, 2005). Globally, there is increase in incidence of type 2 diabetes especially in developing countries due to changing trend of urbanization and lifestyles (Wild *et al.*, 2004; Kenny *et al.*, 1995). This study was undertaken to assess the propensity for diabetes based on the association between BMI and Random Blood Glucose (RBG) in an adult community in Nigeria.

MATERIALS AND METHODS

Study subjects and setting: This study was done in April, 2012 at Iyesi, a small semi urban community located in Ota, Ogun State, South west Nigeria after obtaining

approval and permission from relevant government agencies and Covenant University Institutional ethics Committee.

Inclusion criteria: The study group included 140 apparently healthy individuals aged between 20-70 years old and randomly selected to participate in a community health impact programme. They were enlisted on a voluntary basis after obtaining their informed consent. Pregnant women and psychiatric subjects were excluded from the study.

Anthropometric measurement: The weight in kilogram (kg) was taken with a Produex™ digital balance and height was measured in meters (m) with a standard meter rule. The BMI for each subject was calculated by using the standard formula (weight in kilograms divided by square of height in meters).

Random blood glucose measurement: Blood samples were collected by pricking the finger and Random Blood Glucose (RBG) levels estimated using one touch® glucometer.

Statistical analysis: Data is expressed as Mean±SD and analyzed with SPSS version 15. One way ANOVA followed by Tukey’s test and Pearson’s correlation coefficient (r) were used for comparing the variables determined. The $p < 0.05$ was considered statistically significant.

RESULTS

Out of the total subjects assessed, 34% were male and 66% were female with a mean age of 42.4 ± 11.04 and 38.0 ± 11.78 years, respectively. From Table 1, the mean RBG and BMI of the female subjects were $109.8 \pm 39.5 \text{ mg dL}^{-1}$ and $26.1 \pm 5.84 \text{ kg m}^{-2}$ while that of the male subjects were $122.5 \pm 93.6 \text{ mg dL}^{-1}$ and $26.5 \pm 6.84 \text{ kg m}^{-2}$, respectively. The results showed that the female had lower RBG levels and BMI than the males. Table 2 shows that 12.4% of females were less likely to be diabetic as compared to 86.3% of males. Generally most of

Table 1: Measures of predisposing factors for diabetes in the subjects

	Total (N)	Minimum	Maximum	Mean±SD
Females				
BMI (kg m^{-2})	89	17.04	46.39	26.1±5.950
RBG (mg dL^{-1})	89	57.00	303.00	109.8±39.52
AGE (years)	89	20.00	70.00	38.0±11.78
Males				
BMI (kg m^{-2})	51	19.01	61.57	25.9±6.140
RBG (mg dL^{-1})	51	67.00	590.00	122.5±93.59
AGE (years)	51	22.00	64.00	42.4±11.05

Table 2: Percentage classification of diabetes in the subjects (World Health Organization, 1985; American Diabetes Association, 2006)

	RBG (mg dL ⁻¹) (World Health Organization, 1985)			RBG (mg dL ⁻¹) (ADA)				
	N	Less likely to be diabetic (≤ 80)	Uncertain to be diabetic (≤ 200)	Likely to be diabetic (>200)	Below normal (≤ 70)	Normal (70-140)	Pre-diabetes (140-200)	Diabetes (>200)
Females	89	12.4	85.4	2.2	2.3	84.2	11.2	2.3
Males	51	86.3	7.8	5.9	2.0	84.3	7.8	5.9

Table 3: Percentage classification of obesity in the subjects (World Health Organization, 1985)

	BMI (kg m ⁻²)			
	Underweight (≤ 18.4)	Normal (18.5-24.9)	Overweight (25-29.9)	Obese (≤ 30)
Females	5.6	37.1	39.3	18.0
Males	0	41.2	49.0	9.8

Table 4: Relationship between predisposing factors for diabetes in the subjects

	RBG (mg dL ⁻¹)	Age (years)
Females		
BMI	+0.32**	+0.21
RBG (mg dL ⁻¹)	-	+0.35**
Males		
BMI	-0.05	-0.11
RBG (mg dL ⁻¹)	-	+0.37**

**Correlation coefficient (r) significant at p<0.01 level (2-tailed)

the male and female subjects had normal RBG while few were pre-diabetic. The female subjects were more obese than males. Higher percentages of both males and females were overweight. The 5.6% of females were underweight while no male was underweight (Table 3).

Table 4 shows the relationship between BMI, RBG and age. The correlation coefficient between BMI and RBG of the female subjects was strongly positive (r = +0.32) while that of the males was negative (r = -0.05). The relationship between BMI and age was positive for females (r = +0.21) and negative for males (r = -0.11). There was a strong positive relationship between RBG and age in both males (r = +0.37) and females (r = +0.35).

DISCUSSION

BMI is a good measure of adiposity and by extension overweight and obesity which are major predisposing factors to type 2 diabetes through resistance to insulin-mediated glucose uptake and decreased sensitivity of the beta-cells to glucose (Kahn *et al.*, 2006; DeFronzo and Ferramini, 1991). Insulin resistance is due to adipose tissue-derived hormone-like compounds such as resistin, leptin and adiponectin and other compounds including retinol-binding protein 4, free fatty acids, tumour necrosis factor-alpha, plasminogen activator inhibitor 1 (Steppan *et al.*, 2001; Niswender and Magnuson, 2007; Kadowaki *et al.*, 2006). Diabetes is diagnosed as a consistently high level of blood glucose (Taylor and Agius, 1998).

This study showed that BMI was positively correlated with RBG in female subjects while it was negative for males. This is similar to observations reported by other researchers (Jhanghorbani *et al.*, 1992; Bakari *et al.*, 2006; Shrivastava *et al.*, 2011). Gender and genetic differences in glucocorticoids homeostasis, sex hormones and tissue metabolism may affect the relationship between obesity and diabetes which may explain the different types of association observed in males and females (Andrew *et al.*, 1998; Raven and Taylor, 1995). Other factors including race may also suggest this trend in Nigerians (Bakari and Onyemelukwe, 2004). Obesity is usually more pronounced in females than males (World Health Organization, 2002; Kumar, 1996). Overweight and obese women are more at risk for diabetes than males due to genetic and environmental factors (Guh *et al.*, 2009; Lasky *et al.*, 2002). The observed positive correlation between BMI and RBG in female subjects underlies a strong relationship between overweight and impaired glucose regulation. This observation indicates the role of normal weight in the prevention of diabetes and overall maintenance of good health. The deleterious changes seen with type 2 diabetes is usually reversed by weight loss managed by nutrition and exercise (Felber, 1992; Knowler *et al.*, 1991). The strong association between age and RBG is indirectly related to increase in BMI with age which results in insulin resistance to glucose (Kahn *et al.*, 2006).

The study also revealed isolated high RBG levels in some of the subjects which can be associated with type 2 diabetes. Such subjects could be unaware of their undiagnosed diabetic condition as a result of lack of access to constant medical screening and check up. Most of the subjects including males and females were overweight and therefore have a propensity to become diabetic. This could be attributed to patterns of dietary habits and lifestyles. However, the males were less likely to be diabetic than the females which could be due to their engagement in more physical lifestyles as compared to females (Henriksson, 1995; Lau *et al.*, 2007). This was seen in the negative correlation between BMI and RBG in the male subjects. Generally the low level of diabetes in spite of the high level of overweight could be due to the obesity-survival paradox associated with certain persons (Schmidt and Salahudeen, 2007). A good

number of the subjects had normal weight which is associated with good health and low mortality (Berrington de Gonzalez *et al.*, 2010). Only a small number (5.6%) of females were underweight which could generally be attributed to malnutrition or eating disorders.

CONCLUSION

In conclusion, BMI increased with RBG in the female subjects only while RBG increased with age in both sexes. The prevalence of obesity and diabetes is rising all over the world including developing countries such as Nigeria due to unhealthy lifestyles and dietary habits (Pelletier and Rahn, 1998; World Health Organization, 2005). Also many cases of diabetes are undiagnosed in Nigeria due to poverty and lack of adequate access to health facilities. There is thus need to discourage lifestyle patterns especially in women that may predispose to obesity and type 2 diabetes and thus reduce their associated socio-economic and health consequences (Strycher, 2006; Tate *et al.*, 2007; Braun *et al.*, 1995). There is also need to enlighten the populace about diabetes and need for healthy lifestyles and routine medical screening since the complications of diabetes are far less common and less severe in people who have well-managed blood glucose levels.

ACKNOWLEDGMENT

We appreciate the management of Covenant University, Ota and Ado-Odo/Ota local government area of Nigeria for providing logistic support for this study.

REFERENCES

- American Diabetes Association, 2006. Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 29: S43-S48.
- Andrew, R., D.I.W. Phillips and B.R. Walker, 1998. Obesity and gender influence cortisol secretion and metabolism in man. *J. Clin. Endocrinol. Metab.*, 83: 1806-1809.
- Bakari, A.G. and G.C. Onyemelukwe, 2004. Glucose intolerance among apparently healthy Hausa-Fulani northern Nigerians. *Ann. Afr. Med.*, 3: 32-34.
- Bakari, A.G., G.C. Onyemelukwe, B.G. Sani, I.S. Aliyu, S.S. Hassan and T.M. Aliyi, 2006. Relationship between random blood sugar and body mass index in an Africa population. *Int. J. Diabetes Metab.*, 14: 144-145.
- Berrington de Gonzalez, A., P. Hartge, J.R. Cerhan, A.J. Flint and L. Hannan *et al.*, 2010. Body-mass index and mortality among 1.46 million white adults. *N. Engl. J. Med.*, 363: 2211-2219.
- Braun, B., M.B. Zimmermann and N. Kretcher, 1995. Effects of exercise intensity on insulin sensitivity in women with non-insulin dependent diabetes mellitus (NIDDM). *J. Applied Physiol.*, 78: 300-306.
- Bray, G.A., 2004. Medical consequences of obesity. *J. Clin. Endocrinol. Metab.*, 89: 2583-2589.
- Connell, J.M.C., J.A. Whitworth, D.L. Davies, A.F. Lever, A.M. Richards and R. Fraser, 1986. Effects of ACTH and cortisol administration on blood pressure, electrolyte metabolism, atrial natriuretic peptide and renal function in normal man. *J. Hypertens.*, 5: 425-433.
- DeFronzo, R.A. and E. Ferrannini, 1991. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia and atherosclerotic cardiovascular disease. *Diabetes Care*, 14: 173-194.
- Diaz, V.A., A.G. Mainous, R. Baker, M. Carnemolla and A. Majeed, 2007. How does ethnicity affect the association between obesity and diabetes? *Diabetic Med.*, 24: 1199-1204.
- Fauci, A., E. Braunwald, D. Kasper, S. Hauser, D. Longo, J. Jameson and J. Loscalzo, 2008. *Harrisons Principles of Internal Medicine*. 17th Edn., McGraw-Hill Professional, New York, ISBN-10: 0071466339, Pages: 2958.
- Felber, J.P., 1992. From obesity to diabetes. Pathophysiological considerations. *Int. J. Obes. Relat. Metab. Disord.*, 16: 937-952.
- Grundy, S.M., 2004. Obesity, metabolic syndrome and cardiovascular disease. *J. Clin. Endocrinol. Metab.*, 89: 2595-2600.
- Guh, D.P., W. Zhang, N. Bansback, Z. Amarsi, C.L. Birmingham and A.H. Anis, 2009. The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. *BMC Public Health*, Vol. 9. 10.1186/1471-2458-9-88
- Henriksson, J., 1995. Influence of exercise on insulin sensitivity. *J. Cardiovasc. Risk*, 2: 303-309.
- Howard, B.V., L.D. Cowan, O. Go, T.K. Welty, D.C. Robbins and E.T. Lee, 1998. Adverse effects of diabetes on multiple cardiovascular disease risk factors in women. The strong heart study. *Diabetes Care*, 21: 1258-1265.
- Hu, F., 2008. *Obesity Epidemiology*. Oxford University Press, Oxford, pp: 87-97.

- Icks, A., C. Trautner, B. Haastert, M. Berger and G. Giani, 1997. Blindness due to diabetes: Population-based age-and sex-specific incidence rates. *Diabetic Med.*, 14: 571-575.
- Jhanghorbani, M., A.J. Hedley, R.B. Jones and W.H. Gilmour, 1992. Is the association between glucose level and all causes and cardiovascular mortality risk dependent on body mass index? *Med. J. I.R.I.*, 6: 205-212.
- Kadowaki, T., T. Yamauchi, N. Kubota, K. Hara, K. Ueki and K. Tobe, 2006. Adiponectin and adiponectin receptors in insulin resistance, diabetes and the metabolic syndrome. *J. Clin. Invest.*, 116: 1784-1792.
- Kahn, S.E., R.L. Hull and K.M. Utzschneider, 2006. Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature*, 44: 840-846.
- Kenny, S.J., R.E. Aubert and M.A. Geiss, 1995. Prevalence and Incidence of Non-Insulin-Dependent Diabetes. In: *Diabetes in America*, Aubert, R.E. (Ed.). 2nd Edn., National Diabetes Data Group, National Institutes of Health, Washington, DC., pp: 47-67.
- Knowler, W.C., D.J. Pettitt, M.F. Saad, M.A. Charles and R.G. Nelson *et al.*, 1991. Obesity in the Pima Indians: Its magnitude and relationship with diabetes. *Am. J. Clin. Nutr.*, 53: 1543S-1551S.
- Kumar, K.M.P., 1996. Gender difference in diabetes mellitus. *Int. J. Diab. Dev. Countries*, 16: 103-104.
- Lasky, D., E. Becerra, W. Boto, M. Otim and J. Ntambi, 2002. Obesity and gender differences in the risk of type 2 diabetes mellitus in Uganda. *Nutrition*, 18: 417-421.
- Lau, D.C.W., J.D. Douketis, K.M. Morrison, I.M. Hramiak, A.M. Sharma and E. Ur, 2007. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children. *Can. Med. Assoc. J.*, 176: S1-S13.
- Niswender, K.D. and M.A. Magnuson, 2007. Obesity and the β cell: Lessons from leptin. *J. Clin. Invest.*, 117: 2753-2756.
- Pelletier, D.L. and M. Rahn, 1998. Trends in body mass index in developing countries. *Food Nutr. Bull.*, 19: 191-289.
- Raven, P.W. and N.F. Taylor, 1995. Steroid metabolism in healthy men and women. *J. Endocrinol.*, 147: 100-100.
- Schmidt, D.S. and A.K. Salahudeen, 2007. Obesity-survival paradox-still a controversy? *Semin Dial.*, 20: 486-492.
- Shoelson, S.E., L. Herrero and A. Naaz, 2007. Obesity, inflammation and insulin resistance. *Gastroenterology*, 132: 2169-2180.
- Shrivastava, M., R. Singhal, G. Shrivastava and A. Gupta, 2011. Obesity and gender differences in risk of type 2 diabetes mellitus in Rewa district. *J. Pharm. Res.*, 4: 2643-2644.
- Steppan, C.M., S.T. Bailey, S. Bhat, E.J. Brown and R.R. Banerjee *et al.*, 2001. The hormone resistin links obesity to diabetes. *Nature*, 409: 307-312.
- Strycher, I., 2006. Diet in the management of weight loss. *Can. Med. Assoc. J.*, 174: 56-63.
- Sturm, R., 2007. Increases in morbid obesity in the USA: 2000-2005. *Public Health*, 121: 492-496.
- Tate, D.F., R.W. Jeffery, N.E. Sherwood and R.R. Wing, 2007. Long-term weight losses associated with prescription of higher physical activity goals. Are higher levels of physical activity protective against weight regain. *Am. J. Clin. Nutr.*, 85: 954-959.
- Taylor, R. and L. Agius, 1998. The biochemistry of diabetes. *Biochem. J.*, 250: 625-640.
- Whitlock, G., S. Lewington, P. Sherliker, R. Clarke and J. Emberson *et al.*, 2009. Body-mass index and cause-specific mortality in 900 000 adults: Collaborative analyses of 57 prospective studies. *Lancet*, 373: 1083-1096.
- Wild, S., G. Roglic, A. Green, R. Sicree and H. King, 2004. Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. *Diabetes Care*, 27: 1047-1053.
- Wishner, K.L., 1996. Diabetes mellitus: Its impact on women. *Int. J. Fertil. Menopausal Stud.*, 41: 177-186.
- World Health Organization, 1985. *Diabetes mellitus. Report of a WHO Study Group*, Geneva.
- World Health Organization, 1997. *Obesity: Preventing and Managing the Global Epidemic*. World Health Organization, Geneva pp: 786-987.
- World Health Organization, 2002. *Global health observatory*. *Obes. Res.*, 10: 345-350.
- World Health Organization, 2005. *Surveillance of Chronic Diseases and Risk Factors: Country Level Data and Comparable Estimates*. WHO Global InfoBase Team, Geneva.
- Yang, W., T. Kelly and J. He, 2007. Genetic epidemiology of obesity. *Epidemiol. Rev.*, 29: 49-61.