

# Reducing Diabetes Mellitus Incidence: A Prediabetes Progression Reduction Approach

**Dr. George Gborienemi S.** (Corresponding Author)

Department of Medical Laboratory Science, Niger Delta University, Bayelsa State, Nigeria

Email: [ozunugborie@gmail.com](mailto:ozunugborie@gmail.com)

**Ombor Jerry A.**

Department of Chemical Pathology, Federal Medical Center, Yenagoa, Bayelsa State, Nigeria

## Article History

**Received:** 26 October, 2019


**Revised:** 3 March, 2022

**Accepted:** 5 May, 2022

**Published:** 14 May, 2022

Copyright © 2022 ARPG & Author

This work is licensed under the Creative Commons Attribution International

 **CC BY: Creative Commons Attribution License 4.0**

## Abstract

The critical nature of the narrow window between Prediabetes and Diabetes and a possible progressive transformation were the prompting for this research. 300 out-patients visiting 3 hospitals in Yenagoa city of Bayelsa State, Nigeria were the subjects. By adopting enzymatic and spectrophotometric methods, fasting blood glucose samples collected were estimated and evaluated along with some other parameters. Results show that 45(15%) fall within the Prediabetic range (6.0-9.0mmol/l); 30(10%) were Diabetic (>10.0mmol/l); 213(71%) were within normal reference range (3.5-5.5mmol/l), while 12(4%) had glucose values compatible with hypoglycemia (2.0-3.3mmol/l). Considering positive and negative factors that can influence progression to the diabetic state, we suggest aggressive monitoring model to avert the transformation from prediabetes to diabetes and reduce both the disease prevalence, burden and lethality.

**Keywords:** Diabetes mellitus; Incidence; Prediabetes; Reduction.

## 1. Introduction

Treatment of patients afflicted with Diabetes Mellitus remains a mirage as sufferers are still being managed to ameliorate the condition. Efforts aimed at discovering suitable therapeutics to resolve this debilitating condition are on-going. Of grave concern is the complications associated with Diabetes Mellitus which includes retinopathy nephropathy, neuropathy etc, with attendant blindness, loss of libido and ulceration of the foot and several cases leading to amputation.

Concerns for sufferers imposes on us the need to study conditions that can precipitate this disease condition. Prediabetes has been identified as a state of intermediate hyperglycemia using parameters of Impaired Fasting Glucose (IFG), Impaired Glucose Tolerance (IGT) and Oral Glucose Tolerance Test (OGTT) for those that fall within the grey zone between normal and diabetes (WHO,2006). This condition which is a precursor to diabetes mellitus has been variously described as Insulin resistance syndrome, syndrome X or metabolic syndrome [1].

There is now understanding that a moderate to marked increase in Type 2 diabetes persist predominantly due to changes in life style that manifest in obesity, overweight and noticeable decline in physical activity.

These factors result in activation of features that exacerbate a superimposition on genetic predisposition, elevation in insulin level which in tandem with progressive B-cell inactivity result in rising glycemia among those considered as nondiabetic [2].

The pathophysiology of prediabetics may involve many organs like muscle, liver, pancreas, gut, brain, kidney and adipose tissues and their extent of involvement in prediabetes are being studied by various researchers [3-5]. Previous population based studies that relates to health risk assessment have shown that there is ease of progression of Prediabetes to Diabetes. As shown by Pearson and Pronk [6], there was an incidence of 0.7% in the low risk group and 3.5% in high risk group. A cross sectional population based study have also shown that undiagnosed diabetes constitute a significant percent of 28.8% [7]. Studies by Carolina, *et al.* [8] show that several individuals have abnormal glucose metabolism which they are not aware of. An increase in Prediabetes when the batteries of test included glycated hemoglobin (HbA1c) revealed higher prevalence of Prediabetics in men than in women [9]. A correlation prevalence of prediabetes and diabetes mellitus have also been shown by Weyer, *et al.* [10].

Several studies have shown a continuous and contiguous relationship of glucose in the grey zone and the possible development of type 2 diabetes mellitus. In this research we evaluated the prevalence to ascertain the incidence and proffer proactive solution to stem the progressive rate from Prediabetes to Diabetes Mellitus.

## 2. Materials and Methods

A total of 300 subjects were randomly selected among those visiting 3 hospitals in Federal Medical Centre Yenagoa, Diète Koki Memorial Hospital and Niger Delta University, Teaching Hospital, Okolobiri. The study

received Ethical Committee approval from all institutions involved and were done in the principles expressed in the Declaration of Helsinki prior to data collection. Study patient's sociodemographic characteristics include Gender, male 150(50%), female 150(50%), and an average age of 40-70 years. Parameters evaluated were glucose, triglyceride, cholesterol, insulin and glycated hemoglobin (HbA1c). Glucose was determined by the GOD-PAP method (A product of Human Biochemica Diagnostica, Germany.) Enzymatic colorimetric method was used for determination of Triglyceride using lipid clearing factor (LCF) GPO-PAP method (a product of PCC, Germany). Glycated hemoglobin (HbA1c) was measured through a chromatographic and spectrophotometric method (a product of Techo Diagnostics, California, USA). Measurement was taken at 415nm of total hemoglobin fraction to obtain a ratio of the two fractions and the glycoprotein. Insulin was measured by the automated Elisa method whereby radioactive iodine <sup>125</sup> I-labelled insulin antibodies react with unlabeled antibodies. Separation of bound from free ligand was accomplished by double antibody precipitation and measurements were taken.

### 3. Results

Results of analysis of the Sample for all parameters used are as shown in [Table 1](#).

**Table-1.** Variation in concentration of Parameters among the Various glucose metabolic conditions.

	Group A	Group B	Group C	Group D
Parameters				
Fasting Blood Glucose(mmol/l)	4.5±0.5	1.8±0.7	6.2±0.8	15.4±3.8
Fasting plasma insulin (µiu/l)	22.0±1.5	8.5±1.3	28.0±0.9	26.0±4.0
Glycated hemoglobin (mmol/mol)	38.0±5.0	20.0±3.0	46.0±6.0	82.0±8.2
Triglyceride (mmol/l)	1.5±0.6	1.0±0.3	3.1±0.9	3.3±3.2
Cholesterol (mmol/l)	3.0±1.3	2.1±0.06	3.8±1.3	6.5±2.4

Variations in the blood glucose concentration are shown for the respective conditions. They correlated positively with the concentration of glycated hemoglobin which buttress the fact that glycation took place. Compared with the normal state, we observed a slight increase in the concentration of insulin in prediabetes and diabetes with a mild to moderate elevation of the lipid profiles.

### 4. Discussion

The complications that arise from Diabetes Mellitus is a well-founded ground for sufferers and non-sufferers to be afraid of. This fact has imposed on researchers the need to develop strategies that are proactive in nature so as to avoid escalation.

Our interest in Prediabetes studies is anchored on the understanding that a progression from Prediabetes to Diabetes Mellitus is possible while a pre-emptive action can reduce both the prevalence and incidence with a cumulative effect of reducing the entire burden of the disease.

Outcomes emanating from recent trials have given vent to the realization that delayed progression to type 2 Diabetes Mellitus demonstrates the benefits of identifying patients at risk and implementing aggressive intervention [11].

Although, there is a commonality of evidence to support the fact that a dedicated life style pattern and pharmacological intervention outcome in either delaying, preventing or outrightly inhibiting progression to Diabetes Mellitus were possibilities, there are still doubts that has to do with the issue of cost effectiveness which is still a worry when it comes to situating these interventions into ideal and primary settings. Previous studies have shown that entry into the prodromal phase which patient manifest impaired glucose metabolism has been identified as prediabetes [Meigs, et al. \[12\]](#) and [ADA \[13\]](#).

[Table 1](#) show sample clinical variables of various conditions related to glucose metabolic derangement. We observed moderate to marked variations in insulin levels and Triglyceride level among prediabetics. The clinical pearls of Prediabetes includes the diagnosis of fasting glucose level to demonstrate impaired glucose tolerance (IGT), impaired fasting glucose (IFG) at (7.1mmol/l), or oral Glucose Tolerance Test(OGTT) at (7.77-11.11mmol/l). These observation are in tandem with previous studies of [Pollavsky, et al. \[14\]](#); [Richard, et al. \[11\]](#); [Tuomilehto, et al. \[15\]](#).

From our findings in this study, a diagnosis of Prediabetes underlies the fact that insulin resistance exists as a factor. Insulin resistance is known to prevent glucose from entering the cells to perform its function of producing adenosine triphosphate (ATP), a potent energy source which the body dearly requires. When this is not possible, the pancreas ends up producing excess insulin as a compensatory mechanism. We observed elevated insulin among the prediabetes subjects. There was also a significant elevation of Triglycerides among the prediabetics when compared with normoglycemics, diabetics and hypoglycemic cases in our study.

Generally, fats are important sources of energy for the body. Constantly, the body store of fat is broken down and reassembled to balance the body's energy need with food available. Some specific enzymes are known to help breakdown the processing of fats. Certain derangement in these groups of enzymes can lead to an increase or elevation of specific fatty substances that normally would have been broken down by enzymes. An accumulation of these substances can be detrimental to organs such as pancreas, kidney and the liver, coupled with problems linked to fatty acid disorders. We would lend credence to the increase in triglyceride to these factors. We also observed a

positive correlation of glucose level with glycated hemoglobin. An observation that agrees with the findings of Sonne and Hemmingen [16] and Bang, *et al.* [17].

It is important to recognize the fact that risk factors and symptoms of Prediabetes are similar to those of type 2 diabetes mellitus and appears to make the progression easy. Factors such as obesity, overweight, lack of exercise and age above 45 years have been proved to be extant sources from studies of Li, *et al.* [18] and James, *et al.* [19]. Those previously being diagnosed with gestational diabetes and susceptibility related to family history, genetics, being Pacific Islanders, American Indians, Asian American, African- Americans are cases in point as shown by (HOPESI, 2001, Diabetes Prevention Group, 2002). A few of the subjects in this study presented with dark skin on the necks, armpits, elbows, knees and knuckles. Our findings reveal a ratio of 15% of persons who are prediabetics. This is supported by previous findings of Wandell and Tovi [20], Fong, *et al.* [21], [13] in which a ratio of 1 out of 3 prediabetics who may progress to diabetics were found. We also observed a small percentage of hypoglycemic patients who run the risk of hypoglycemic shock.

We propose a Proactive Screening Model that will prompt individuals for routine and regular glucose Monitoring to identify those at risk as a prelude for preventive action and control.

## 5. Conclusion

This study re-iterated the need to raise the awareness of the populace on the complications of diabetes and the compelling need to take pre-emptive and preventive decision driven by the urgent desire for a comprehensive proactive policy decision for prevention of diabetes.

## References

- [1] Troughton, R. W., Leigh, J. E., Michael, A. B., Christine, S. M., Wendy, A. S., Mark, A. R., Chris, F., and Vicky, A. C., 2008. "Genomic selection of reference genes for real time PCR in human myocardium." *BMC Medical Genomics*, vol. 1, p. 64. Available: [10.1186/1755-8794-1-64](https://doi.org/10.1186/1755-8794-1-64)
- [2] Vendrame, F. and Peter, A. G., 2004. "Prediabetes: Prediction and Prevention trials." *Endocrinology and metabolism clinic of North America*, vol. 33, pp. 75-95.
- [3] Defronzo, A., 2004. "Medical clinic North America." vol. 88, pp. 787-835.
- [4] Balkau, B., Hu, G., Qiao, Q., Tuomilehto, J., Borch-Johnson, K., and Pyorala, K., 2004. "Prediction of the risk of cardiovascular mortality using a score that includes glucose as a risk factors." *Diabetologia*, vol. 47, pp. 2118-2128.
- [5] Faerch, K., Vaag, A., Borch-Johnsen, P. O., Holst, C., and Glumer, O., 2008. "Impaired fasting glycemia vs impaired glucose tolerance: similar impairment of pancreatic alpha and beta cell function but differential roles of incretin hormones and insulin action." *Diabetologia*, vol. 51, pp. 853-861.
- [6] Pearson, L. and Pronk, N., 2003. "Identifying individuals at risk for development of type 2 diabetes mellitus." *American Journal of Management Care*, vol. 9, p. 57.
- [7] Abbas, M., Header, W., Ibrahim, H. A., Keal, A., and Hameed, W., 2008. "Diabetes screening in basrah, Iraq. A population based cross-sectional study." *Diabetes Research and Clinical Practice*, vol. 79, pp. 567-574.
- [8] Carolina, L., Romina, L., Beverly, O., Joan, F., Zhi, C., Valeria, K., Jean, H., and Kenneth, C., 2012. "Prevalence of prediabetes and diabetes and metabolic profile of patients with nonalcoholic fatty liver disease." *Diabetes care*, vol. 35, pp. 873-878.
- [9] Sicree, R., Shaw, J., and Zimmet, P., 2009. "Global estimates of the prevalence of diabetes for 2010 and 2030." *Journal of Diabetes*, vol. 87, pp. 4-14.
- [10] Weyer, C., Bogardus, C., and Pratel, R., 1999. "Metabolic characteristics of individuals with impaired fasting glucose and/or impaired glucose tolerance." *Diabetes*, vol. 48, pp. 1714-34.
- [11] Richard, J., Shrot, Francis, M. S., James, H., and Bronlee, J., 2004. "Case study: Screening and treatment of prediabetics in primary case." *Clinical Diabetes*, vol. 22, pp. 98-100. Available: <http://doi.org/10.2337/diaclin22.2.98>
- [12] Meigs, J., Muller, D., Natha, D., Blake, D., and Andres, R., 2003. "The natural history of progression from normal glucose tolerance to type 2 diabetes in the baltimore longitudinal study g aging." *Diabetes*, vol. 52, pp. 1475-1484.
- [13] ADA, N., 2004. "Diabetes Clinical Trials: What is New at NIDDK?" *Clin Diabetes*, vol. 22, pp. 109-112.
- [14] Pollavsky, E., Kim, D. J., and Bang, H., 2016. "Comparison of screening score for diabetes and prediabetes." *Diabetes Res. clin. Pract.*, vol. 118, pp. 146-153.
- [15] Tuomilehto, J., Lindstrom, J., Ericson, J. G., Valle, T. T., Hamalainen, H., Illane-Parikka, P., Keinanen-Kiukaanmeni, S., Laasko, M., Loueranta, A., *et al.*, 2001. "Prevention of type 2 diabetes mellitus by changes in life style among subjects with imposed glucose tolerance." *N. Engl. J. Med.*, vol. 344, pp. 1343-1350.
- [16] Sonne, D. P. and Hemmingen, B., 2017. "Comment on american diabetic association. Standards of medical care in diabetes." *Diabetes Care*, vol. 40, pp. 40e92-e93 51-5135. Available: <http://doi.org/10.2337/dc17-0299>
- [17] Bang, H., Edwards, A. M., and Bombuck, A. S., 2009. "Development and validation of a patient self assesment. Score for diabetes risk." *An intern Med.*, vol. 151, pp. 775-783.

- [18] Li, G., Zhang, P., and Wang, J., 2016. "Cardiovascular mortality, all-cause mortality, and diabetes incidence after lifestyle intervention for people with impaired glucose tolerance in the da qing diabetes prevention study: 23-years follow-up study." *Lancet Diabetes*, Available: [https://doi.org/10.1016/S2213-858\(16\)30185-1](https://doi.org/10.1016/S2213-858(16)30185-1)
- [19] James, C., Bullard, K. M., and Rolka, D. B., 2011. "Implications of alternative definition of prediabetes for prevalence in US adults." *Diabetes Care*, vol. 34, pp. 387-391.
- [20] Wandell, P. E. and Tovi, J., 2000. "The quality of life of elderly diabetic patients." *Diabetes Complications*, vol. 14, pp. 25-30.
- [21] Fong, D. S., Aiello, L. P., Ferris, F. L., and Klein, R., 2004. "Diabetic retinopathy." *Diabetes Care*, vol. 27, pp. 2540-2553.