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Stability Analysis of the Mathematical Model for the Dynamics of Diabetic Population under the Combine Effect of Birth Rate and Treatment

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ABSTRACT

In this paper a mathematical model for the dynamics of diabetic population under the combined effect of birth rate and treatment was developed to study the stability and disease free equilibrium of the diabetic population. In developing the model, we splitted the population of diabetics into; Diabetics without complications, diabetics with complications, diabetics with regulated glucose as a result of treatment, and severely disabled sub-populations, we then modeled the dynamics between the first three sub-populations, this ushered in a model consisting of three differential equations. We obtained the disease free equilibrium state of the model and carried out stability analysis. The result of the study established that; birth rate and the number of diabetics with complications determine the equilibrium solutions, and that the contribution of birth rate to the dynamics of the diabetic population depends on the dynamics of the diabetic gene and life style. This is in tandem with the dynamics of the diabetics since diabetes is not contagious, rather, a genetic disorder with chromosomes 1q21-1q24, 2q37, 12q24, and chromosome20, as the locus that harbor the gene that causes diabetes. The work recommended that efforts should be geared towards controlling life style and reproductive practice of a population to curtail the genetic spread of the disease.

Key Words: *Panmictic, Population, Diabetes, Birth Rate, Fasting Blood Sugar, Random Blood sugar*

2010 Subject classification: 92B05, 92D25, 92D30, 93D05, 34K20, 34K25

1. INTRODUCTION

Diabetes was previously considered a disease of minor significance to world health. However, there has been an explosive increase in the number of people diagnosed with diabetes worldwide in the last few decades (Hans *et al.*, 2003). It is now commonly admitted that diabetes is sweeping the globe as a silent epidemic largely contributing to the growing burden of non-communicable disease and mainly encouraged by decreasing level of activities and increasing prevalence of obesity, recent reports released by the World Health Organization (WHO) are alarming, in 2003, it was estimated that 194 million people were diabetic, representing a global prevalence exceeding 3% of the world population, the trend is increasing and the number is expected to reach 333 million (16.3%) by the year 2025. Moreover, for the first time, an estimation of 314 million (8.2%) is given for people in the pre-diabetic stage, which constitute a compartment from which at least one third will evolve to the diabetic stage after ten years (Bouteyab *et al.*, 2004).

Glucose is the main source of fuel for our body. Foods that affect blood glucose are called carbohydrates. Carbohydrates, when digested, change to glucose. Glucose is then absorbed into the blood and is used by the cells for energy. In order for glucose to be transferred from the blood into the cells, the hormone – insulin (*which is produced by the beta cells in the pancreas*) is needed. In individuals with diabetes, this process is impaired www.diabeteswellness.net,

The term diabetes mellitus describes a metabolic disorder of multiple etiologies, characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action or both. The effects of diabetes include long-term damage, dysfunction and failure of various organs (WHO, 1999).

According to WHO (2003), diabetes can be classified into two based on the aetiology of the disease; viz; Type I diabetes and Type II diabetes. Type I diabetes indicates the process of β-cell destruction that may ultimately lead to diabetes, in which insulin is required for survival, the other name for it is Insulin Dependent Diabetes Mellitus (IDDM) (WHO, 2003) while Type II Diabetes is characterized by disorders in insulin action, insulin secretion or both. The other name of it is Non-Insulin Dependent Diabetes (NIDDM) (WHO, 2003).

Adamu *et al.*, (2005) stated that type II diabetes is a heterogeneous disorder characterized by chronic hyperglycemia due to dynamic interactions between varying defects of insulin secretion and insulin resistance. Either of these defects may be the predominant feature in a particular case. According to world table published by WHO in 2000, 3.4% Nigerians age 20 and above are diabetic. This global diagnosis being given, it is essential to stress that, much of the cost of diabetes treatment is attributable to long term complications such as blindness, kidney failure, heart disease, amputations and their economic and social consequences (like; care, hospitalization, absenteeism, e.t.c.). According to

Ebenezer *et al.*, (2003), the prevalence of type II diabetes in Port Harcourt is fairly high. 40% of the subjects with diabetes are undiagnosed, over 80% of these are asymptomatic. Some of the identified risk factors for type II diabetes are modifiable, making type II diabetes a potentially preventable disease. It would be prudent, therefore, to recommend screening of subjects at risk to reduce the prevalence of type II diabetes in Port Harcourt. Sarah *et al.*, (2004) opined that; the prevalence of diabetes for all age-groups worldwide was estimated to be 2.8% in 2000 and 4.4% in 2030. The total number of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030, and the prevalence of diabetes is higher in men than women, but there are more women with diabetes than men. Sarah *et al.*, (2004) further stated that, the most important demographic change to diabetes prevalence across the world appears to be on the increase in the proportion of people 65 years of age. These findings indicate that the "diabetes epidemic" will continue even if levels of obesity remain constant. David (2010) opined that a diabetes prevalence survey attracted the attention of the world's general media, but the number of people in China estimated to have diabetes now was so large that it took the news world by surprise. The findings of the study have a number of important implications for China and beyond.

1.3. Burden of Diabetes

According to Bouteyab *et al.*, (2004) diabetes is;
- The leading cause of end stage kidney failure necessitating dialysis or transplantation.

- The leading cause of blindness in people of working age.

- The leading cause of amputation.

- The first cause (*with other basic factors*) of mortality and mobility by Cardio vascular diseases

-Bouteyab *et al.*, (2004) stated that, the exact cost of diabetes is not easy to pin down but estimation can be obtained according to 3 levels as follows:

i) Cost directly related to the diagnosis and management of diabetes, this includes theinpatient andoutpatient care, means of treatment by insulin or tablets and equipment of self-control (*blood and urine testing*).

ii) Cost generated by complications of diabetes, these are difficult to quantify because diabetes is linked to
micro and macro vascular diseases such as heart disease, kidney failure, eye disease and amputation; moreover diabetes may add a cost of care by complicating other unrelated medical situations like infections, accident and surgery.

iii) Indirect cost correlated to the quality of life and the economic productivity, which can be somehow estimated by the degree of disability

1.4. How is it spread?

Diabetes is spread through heredity as reported by David *et al.*, (2003), they opined that diabetes mellitus is a recognized consequence of hereditary haemochromatosis, furthermore experimental results from the work of Florence *et al.*, (2003) on genomic wide scans for linkage, have reported a number of chromosomal regions that may harbor genes involved in type II diabetes, with the most promising, replicating findings on chromosomes 1q21-q24, 2q37, 12q24 and chromosome 20.

1.5. How is it controlled?

Once diabetes is diagnosed, treatment consists of controlling the amount of glucose in the blood and preventing complications, depending on the type of diabetes, this can be accomplished through regular physical exercise, a carefully controlled diet, and medication. Individuals with Type I diabetes must receive insulin, often two to four times a day, to provide the body with the hormone it does not produce. For persons with type II diabetes, treatment begins with diet control, exercise, and weight reduction, although over time this treatment may not be adequate. People with Type II diabetes typically work with nutritionists to formulate a diet plan that regulates blood sugar levels so that they do not rise too swiftly after a meal. A recommended meal is usually low in fat (30 percent or less of total calories), provides moderate protein (10 to 20 percent of total calories), and contains a variety of carbohydrates, such as beans, vegetables, and grains. Regular exercise helps body cells absorb glucose, even ten minutes of exercise a day can be effective. Diet control and exercise may also play a role in weight reduction, which appears to partially reverse the body's inability to use insulin, Microsoft ® Encarta ® (2008), this is supported by Christian *et al.*, (2001), who reported that; considerable information has recently accumulated regarding postprandial glucose homeostasis, it appears that about one-third of ingested carbohydrate is immediately taken up by splanchnic tissues, of the remaining two-thirds of the ingested carbohydrate that enters the systemic circulation, some is extracted by the liver, and 40% of the ingested carbohydrate is taken up by the skeletal muscles (Christian *et al.*, 2001). Symptoms of diabetes include some or all of the following.

- Blurred vision
- Unusual thirst
- Frequent urination
- Slow-healing of cuts
- Unexplained tiredness
- Unexplained rapid weight loss
- Erectile dysfunction
- Numbness or tingling in hands or feet

Symptoms may occur rapidly with Type 1 diabetes; however, with Type 2 diabetes, the onset is more insidious and may not be noticed (*Diabetes research & Wellness Foundation (DRWF)*), www.diabeteswellness.net, pp 2-3.

Ibrahim (2012) developed a Mathematical Model for attenuating the spread of Diabetes and its Management in a population. The result of the study suggested; 1) We need to look into the dynamics of the genetics of transmission of the gene(s) responsible for the evolution of diabetes. 2) Look into the effects of physical activity and or dieting on the dynamics of glucose metabolism so as gain an insight on how to manage diabetic patients. The combined effect of birth rate and treatment was not considered by Ibrahim (2012) in developing the model. Ibrahim *et al.,* (2014) developed a Mathematical model for the dynamics of glucose regulatory system under the combined use of dieting and physical activity. The model was used to investigate the effect of dieting and physical activity on glucose and insulin homeostasis, The result of the study showed that; dieting and physical activity has great impact on the regulation of plasma glucose and insulin concentration, and that dieting and physical activity can be used on any population for the management of glucose and insulin homeostasis. Furthermore, their study corroborates the clinical trial studies by Margarita *et al.,* (2005), Jean (1999), that; dieting and physical activity improves insulin and glucose effectiveness on a wider scope rather than on a specific population as required by clinical trial studies. Ibrahim *et al.,* (2014) concluded as follows; 1) Medical practitioners should encourage a combined physical activity and calorie restriction therapy for the management of diabetes, 2) Government, NGO's, and stake holders should improve on investment in physical activity facilities and production of low calorie diet for diabetic patients. Such therapy will attenuate the development of diabetes and transition from diabetes without complications to diabetes with complications in a diabetic population. Also, the combined effect of birth rate and treatment was not considered by Ibrahim *et al.,* (2014) in developing the model.

From the forgone introduction, the combined effect of birth rate and treatment on the dynamics of a diabetic population was not studied. In this paper a mathematical model for the dynamics of diabetic population incorporating birth rate and treatment was developed, the stability of the disease free equilibrium state was analyzed and established.

2. METHODOLOGY

2.1. Assumption

Some assumptions as adopted from Ibrahim *et al.,* (2014) with new additions are given below;

- The population of diabetics is finite and panmictic
- Birth rate is a function of time
- Rate of developing complications is constant
- Rate of recovery from Complications is constant
- Rate of returning to unregulated glucose is constant
- Natural death is constant
- Death due to complications is constant
- Rate of achieving glucose regulation is constant
- Incidence of the disease occurs without complications
- Complications are developed with time
- At birth, a child is susceptible with time

2.2. Notations

Notations adopted from Ibrahim *et al.,* (2014) with new additions are given below;

- μ Natural Death
- δ Death due to complications
- λ Rate of developing complications
- γ Rate of recovery from complications
- ν Rate of developing disability
- ρ(t) Birth rate.
- DC(t) Number of diabetics without complications
- C(t) Number of diabetics with complications
- DN(t) Number of diabetics with regulated glucose level
- SD(t) Number of diabetics with disability
- t Time as continuous variable
- Ex Level of exercise measured in calories
- d Level of dietary intake measured in calories
- $h = \frac{Ex}{d}$ Rate of achieving glucose regulation
- k Rate of returning to unregulated glucose
- FBS, RBS Fasting Blood Sugar, Random Blood Sugar respectively

2.3 Model Development

2.3.1 The Dynamics

A person is born into the susceptible population, he/she may develop the disease without complications and develop complications with time or have his/her blood sugar normalized through some control measure or die naturally. A diabetic person with complications may die naturally or as a result of complications, he/she may develop severe disability and die naturally or as a result of the disability. On this basis, we have the following dynamics; the number of diabetics without complications DC(t) depletes by $\mu DC(t)$, $\beta DC(t)$ and $hDC(t)$ as a result of natural death, transition to the state of diabetes with complications and transition to the state of normalized blood sugar respectively, and increase by $\gamma C(t), \rho(t)$ *and* k as a result of recovery from complications, birth rate, and lost of normalized blood sugar respectively. The number of diabetes with complications $C(t)$ depletes by

$C(t), \nu C(t), \delta C(t)$ *and* $\gamma C(t)$ as a result of natural death, developing disability, death from complications and recovery from complications, while it increases by$\beta D(t)$ as a result of developing complications from D(t). the above dynamics can be schematically represented as follows;

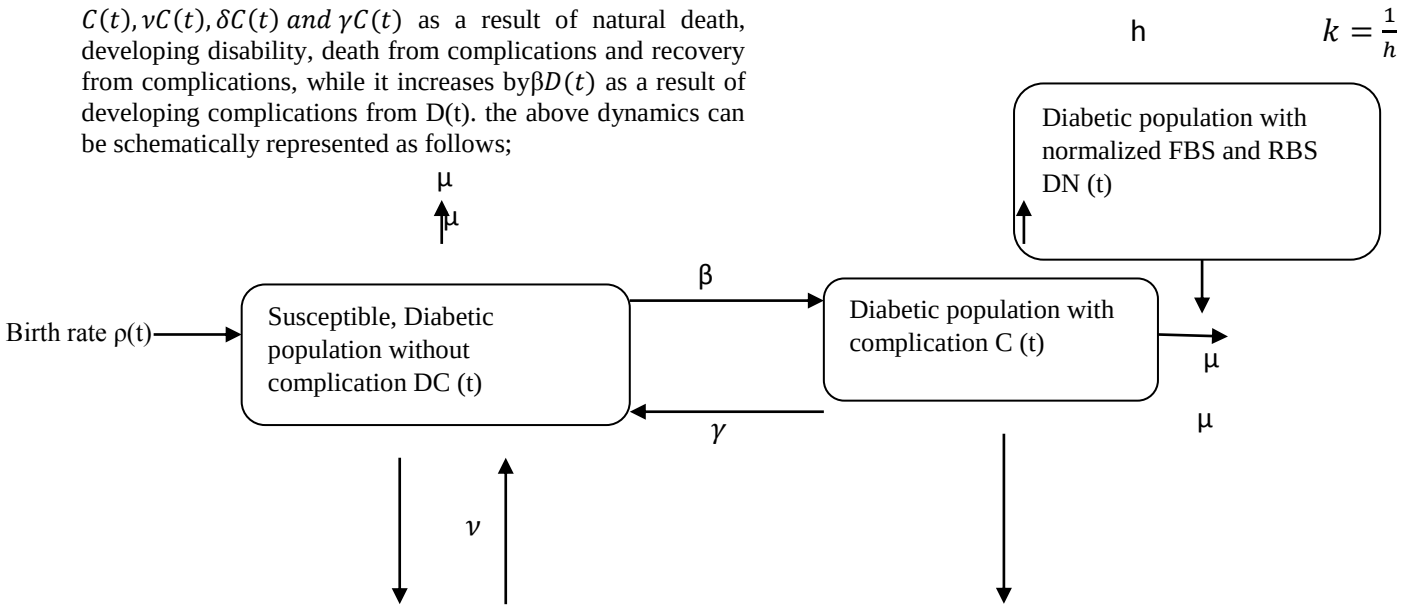

Fig. 1: Schematic Model for the dynamics described above

2.3.2 Mathematical Formulation

In developing the mathematical model, we shall only model the dynamics between DC(t), DN(t) and C(t), this is because the subpopulation SD(t) of disabled diabetic persons constitutes a class that cannot revert back to DC(t), DN(t) or C(t) and therefore considered unproductive. Hence they are excluded from the dynamics and further analysis. On the basis of the exclusion, and the schematic model described above, the dynamics of the diabetic population under the combined effect of birth rate and treatment was mathematically modeled by the system of Ordinary Differential Equations given below:

$$\left.\begin{aligned} \frac{dDC\{t)}{dt} &= \rho(t) - \beta DC(t) + \gamma C(t) - hDC(t) + KDN(t) - \mu DC(t) \\ \frac{dDN\{t)}{dt} &= hDC(t) - \mu DN(t) - KDN(t) \\ \frac{dC\{t)}{dt} &= \beta DC(t) - \gamma C(t) - \nu C(t) - \mu C(t) - \delta C(t) \end{aligned}\right\} - - - (1)$$

Since we assumed that incidence of the disease occurs without complications and DC(t) is recharged by $\rho(t)$, we have the following initial conditions

C(0) = 0, DC(0) = C_0 and DN(0) = D_0

Existence and Uniqueness of Solution

The existence and uniqueness theorem requires that the right hand side terms of system (1) and their partial derivatives with respect to DC(t), C(t) and DN(t) be continuous.

Looking at system, we see that the right hand side terms of system (1) and their partial derivatives with respect to. DC(t), C(t) and DN(t) are continuous. Hence solutions to system (1) exist and are unique.

Positivity of solutions

The presented model above describes the dynamics of a population of Diabetics under the combined effect of birth rate and treatment, it is important to prove that all quantities (*Diabetics without complications DC(t); Diabetics with complications C(t); Diabetics with regulated glucose DN(t)*) will be non negative for all times. Intuitively, we want to prove that all solutions of the system of eqn (1) with positive initial data will remain positive for all times t>0.

Lemma 1: Let the initial data be $C(0) \geq 0, DC(0) \geq C_0$ and $DN(0) \geq D_0$ for all $t \in [0, \infty)$. Then the solution (C(t), DC(t), DN(t)) of the model remain positive $\forall\, t \geq 0$.

Proof: From the third equation of system (1), we have

$$\frac{dC\{t)}{dt} = \beta DC(t) - \gamma C(t) - \nu C(t) - \mu C(t) - \delta C(t)$$

$$= \beta DC(t) - (\gamma + \nu + \mu + \delta)C(t)$$

Clearly $\frac{dC\{t)}{dt} \geq -(\gamma + \nu + \mu + \delta)C(t) = -\emptyset C(t)$; where $\emptyset = (\gamma + \nu + \mu + \delta)$

Thus; $\frac{dC\{t)}{dt} \geq -\emptyset C(t)$. Solving this differential inequality we have $C(t) \geq k_1 e^{-\theta t} > 0 \; \forall \; t \geq 0$

Similar reasoning on the remaining equations yields $DN(t) \geq k_2 e^{-(\mu+k)t} > 0 \; \forall \; t \geq 0$ and $DC(t) \geq k_3 e^{-(\beta+\mu+h)t} > 0 \; \forall \; t \geq 0$. Thus the solutions to the model equations are positive $\forall \; t \geq 0$.

3. QUALITATIVE ANALYSIS

3.1 Equilibrium Point

The null clines of the above system are given by:

$$\left.\begin{aligned} \frac{dDC(t)}{dt} = 0 = \; & \rho(t) - \beta DC(t) + \gamma C(t) - hDC(t) + kDN(t) - \mu DC(t) \\ \frac{dDN(t)}{dt} = 0 = \; & hDC(t) - \mu DN(t) - kDN(t) \\ \frac{dC(t)}{dt} = 0 = \; & \beta DC(t) - \gamma C(t) - \nu C(t) - \mu C(t) - \delta C(t) \end{aligned}\right\} \ldots\ldots\ldots\ldots\ldots (2)$$

From $\frac{dDN(t)}{dt} = 0$, we have $DN(t) = \frac{hDC(t)}{\mu+k}$ ……(3)

Using (3) in $\frac{dDC(t)}{dt} = 0$, and solving for $\rho(t)$, we have;

$$\rho(t) = \left(\beta + h + \mu - \frac{kh}{\mu+k}\right) DC(t) - \gamma C(t).$$

From which we have $DC(t) = \frac{(\rho(t)+\gamma C(t))\,(\mu+k)}{(\beta+h+\mu)\,(\mu+k)-kh} = \frac{(\rho(t)+\gamma C(t))}{(\beta+h+\mu)\,-\frac{kh}{(\mu+k)}}$ ……(4)

From $\frac{dC(t)}{dt} = 0$, we have $\beta DC(t) - C(t)(\gamma + \upsilon + \delta) = 0$. Whence C(t) = $\frac{\beta DC(t)}{(\gamma+\upsilon+\delta)}$ …… (5)

Using equation (4) in (3) and simplifying we have:

$$DN(t) = \frac{h(\rho(t) + \gamma C(t))}{(\mu + k)\left[(\beta + h + \mu) - \frac{kh}{\mu+k}\right]}$$

$$C(t) = \frac{\beta\rho(t)}{\left[(\gamma + \upsilon + \mu + \delta)\left[(\beta + h + \mu) - \frac{kh}{\mu+k}\right]\right]}$$

Therefore at equilibrium point we have:

$$DC(t) = \frac{(\rho(t) + \gamma C(t))}{(\beta + h + \mu)\; - \frac{kh}{(\mu+k)}}$$

$$DN(t) = \frac{h(\rho(t) + \gamma C(t))}{(\mu + k)\left[(\beta + h + \mu) - \frac{kh}{\mu+k}\right]}$$

$$C(t) = \frac{\beta(\rho(t) + \gamma C(t))}{\left[(\gamma + \upsilon + \delta)\left[(\beta + h + \mu) - \frac{kh}{\mu+k}\right]\right]}$$

Clearly at equilibrium points; $DC(t)$ and $C(t)$ are influenced by $\rho(t)$, $C(t)$

Therefore disease free equilibrium state is given by; $E_0 = \left(\frac{\rho(t)\,(\mu+k)}{(\beta+h+\mu)\,(\mu+k)-kh}, \frac{h\rho(t)}{(\gamma+\upsilon+\delta)\left[(\beta+h+\mu)-\frac{kh}{\mu+k}\right]}, \frac{\beta\rho(t)}{\left[(\gamma+\upsilon+\delta)\left[(\beta+h+\mu)-\frac{kh}{\mu+k}\right]-\gamma\right]}\right)$ which clearly depends on $\rho(t)$. With $\rho(t) = 0$, E_0 evaluates to $E_0 = (0,0,0)$.

From the first equation of (2), we see that birth rate $\rho(t)$, is a major positive contributor to diabetics with complications. This suggests that; at birth a child may have a genetic disposition for the disease (*As stated in our assumptions*).

This position is confirmed by the work of Florence *et al* (2003) who identified chromosome 1q21-1q24, 2q37, 12q24 and chromosome 20 as the locus that harbor the gene that causes diabetes

3.2 Stability Analysis

The above system of equations can be represented as follows:

$$f(DC, C, DN) = \frac{dDC\{t\}}{dt} = \rho(t) - \beta DC(t) + \gamma C(t) - hDC(t) + KDN(t) - \mu DC(t)$$
$$g(DC, C, DN) = \frac{dDN\{t\}}{dt} = hDC(t) - \mu DN(t) - KDN(t)$$
$$l(DC, C, DN) = \frac{dC\{t\}}{dt} = \beta DC(t) - \gamma C(t) - \nu C(t) - \mu C(t) - \delta C(t)$$

Now the Jocobian is given by:

$$JAC = \begin{pmatrix} \frac{\partial f}{\partial DC} & \frac{\partial f}{\partial C} & \frac{\partial f}{\partial DN} \\ \frac{\partial g}{\partial DC} & \frac{\partial g}{\partial C} & \frac{\partial g}{\partial DN} \\ \frac{\partial l}{\partial DC} & \frac{\partial l}{\partial C} & \frac{\partial l}{\partial DN} \end{pmatrix} = \begin{pmatrix} -(\beta + h + \mu) & \gamma & k \\ h & 0 & -(\mu + k) \\ \beta - \gamma & -(\nu + \mu + \delta) & \beta \end{pmatrix}$$

Now; $Det(JAC - \lambda I) = |JAC - \lambda I| = 0$ gives:

$$\begin{vmatrix} -(\beta + h + \mu) - \lambda & \gamma & k \\ h & -\lambda & -(\mu + k) \\ \beta - \gamma & -(\nu + \mu + \delta) & \beta - \lambda \end{vmatrix} = 0$$

$$\equiv (-(\beta + h + \mu) - \lambda)(-\lambda(\beta - \lambda) - (\mu + k)(\nu + \mu + \delta)) - \gamma\big(h(\beta - \lambda) + (\mu + k)(\beta - \gamma)\big) + k\big(-h(\nu + \mu + \delta) + \lambda(\beta - \gamma)\big) = 0$$

Let $\beta + h + \mu = \alpha;\ \mu + k = \pi\ ;\ \nu + \mu + \delta = \sigma,\ \beta - \gamma = \theta$

Therefore we have:

$(-\alpha - \lambda)(-\lambda(\beta - \lambda) - \pi\sigma) - \gamma(h(\beta - \lambda) + \pi\theta) + k(-h\sigma + \lambda\theta) = 0$……(6)

Next; we investigate the stability of the system by applying the result of Bellman and Coke (1963) to (6). We first present a fundamental theorem for stability analysis of the characteristic equation as follows:

3.2.1 Bellman And Coke's Theorem

Let $H(Z) = P(Z, e^z)$ where P a polynomial with principal term. Suppose $H(Z),$, is separated into its real and imaginary parts as follows;

$H(Z) = F(y) + iG(y)$ …… (7)

If all zeros of $H(Z)$ have negative real parts, then the zeros of $F(y)\ and\ G(y)$ are real, simple and alternate and $J = G'(0)F(0) - G(0)F'(0) > 0$ ... (8)

Conversely, all zeros of $H(Z)$ will be in the left half plane provided that either of the following conditions is satisfied.

- All the zeros of $F(y)\ and\ G(y)$ are real, simple, and alternate; and the inequality in (8) is satisfied
- All the zeros of $F(y)$ are real, and for each zero the relation (7) is satisfied All the zeros of $G(y)$ are real, and for each zero, the relation (7) is satisfied

Thus the condition for stability according to Bellman and Cooke's theorem is given by
$J = G'(0)F(0) - G(0)F'(0) > 0$ Hence the disease free equilibrium state will be stable if $J > 0$

3.3 Applying Bellman And Coke Theorem To The System

Let (6) take the form;

$$H(\lambda) = -(\alpha + \lambda)(-\lambda(\beta - \lambda) - \pi\sigma) - \gamma(h(\beta - \lambda) + \pi\theta) + k(-h\delta + \lambda\theta)$$

Expanding and rearranging in ascending powers of λ we obtained;

$$H(\lambda) = \lambda^3 - (\beta - \alpha)\lambda^2 - (\alpha\beta + \pi\sigma + \gamma h + K\theta)\lambda - (\alpha\sigma - \gamma\theta)\pi - (\gamma\beta + k\sigma)h \quad ... (9)$$

We now set $\lambda = iw$ in equation (9) to have;

$$H(\lambda) = (iw)^3 - (\beta - \alpha)(iw)^2 - (\alpha\beta + \pi\sigma + \gamma h + K\theta)iw - (\alpha\sigma - \gamma\theta)\pi - (\gamma\beta + k\sigma)h \quad ... (10)$$

Separating the expression into real and imaginary parts we have;

$$H(\lambda) = (\beta - \alpha)w^2 - (\alpha\sigma - \gamma\theta)\pi - (\gamma\beta + k\sigma)h - (w^3\ (\alpha\beta + \pi\sigma + \gamma h + K\theta)w)i$$

We then apply the result of Bellman and Coke (1963) to analyze the disease free equilibrium state for stability or otherwise. Resolving (10) into real and imaginary parts we have; $H(iw) = F(w) + iG(w)$, where $F(w)\ and\ G(w)$ are given, respectively, by;

$$F(w) = w^2(\beta - \alpha) - (\alpha\sigma - \gamma\theta)\pi - (\gamma\beta + k\sigma)h \ and \ G(w) = -w^3 - (\alpha\beta + \pi\sigma - \gamma h + K\theta)w$$

Differentiating with respect to w, we have; $F'(w) = 2w(\beta - \alpha)\ and$
$G'(w) = -3w - (\alpha\beta + \beta\sigma - \gamma h + K\theta)$
Setting $w = 0$, we get;
$F(0) = -(\alpha\sigma + \gamma\theta)\beta + (\gamma\beta + k\sigma)h,\ G(0) = 0, F'(0) = 2w(\beta - \alpha) = 0\ and\ G'(0) = -(\alpha\beta + \beta\sigma - \gamma h + K\theta)$

According to the result by Bellman and Coke (1963), the condition for stability is given by

$$J = G'(0)F(0) - G(0)F'(0) > 0, \forall\, y \in \mathbb{R}$$

Now, since $G(0) = F'(0) = 0$ We have $G'(0)F(0) = 0$, thus we have

$$J = G'(0)F(0) = (\alpha\sigma + \pi\sigma + \gamma h + k\theta)(\sigma\alpha - \gamma\theta)\pi - (\gamma\beta + k\theta)h \quad ... (10)$$

Now if J evaluates to positive (+ve) value for values of the parameters, then the system is stable else unstable. Adopting the definition of h from Ibrahim *et al.,* (2012), given as, $h = \frac{Ex}{d}$, and defined $k = \frac{1}{h}$ and using the values for the parameters from A. Bouteyab *et al.,* (2004) given in Table 1 below for the stability analysis:

Table 1: Adopted from A. Bouteyab *et al* (2004) pp5

S/N	PARAMETER	VALUE
1	μ	0.02
2	γ	0.08
3	υ	0.05
4	δ	0.05

We have the computed values of J by varying the values of β and h as follows:

Table 2: Simulated results for stability analysis

S/N	β	μ	γ	ν	h	$k=\frac{1}{h}$	δ	J	Remark
1	0.01	0.02	0.08	0.05	0.01	100	0.05	4.6043	Stable
2	0.05	0.02	0.08	0.05	0.06	16.67	0.05	0.2728	Stable
3	0.09	0.02	0.08	0.05	0.11	9.09	0.05	0.1364	Stable
4	0.13	0.02	0.08	0.05	0.16	6.25	0.05	0.0967	Stable
5	0.17	0.02	0.08	0.05	0.21	4.76	0.05	0.0891	Stable
6	0.21	0.02	0.08	0.05	0.26	3.85	0.05	0.0655	Stable
7	0.25	0.02	0.08	0.05	0.31	3.23	0.05	0.0617	Stable

4. RESULTS AND DISCUSSION

In this work, we have developed a mathematical model for the study of the dynamics of diabetic population under the combined effect of birth rate and treatment given as follows;

$$\frac{dDC\{t)}{dt} = \rho(t) - \beta DC(t) + \gamma C(t) - hDC(t) + kDN(t) - \mu DC(t)$$
$$\frac{dDN\{t)}{dt} = hDC(t) - \mu DN(t) - KDN(t)$$
$$\frac{dC\{t)}{dt} = \beta DC(t) - \gamma C(t) - \nu C(t) - \mu C(t) - \delta C(t)$$

With equilibrium point solution given as;

$$DC(t) = \frac{(\rho(t) + \gamma C(t))}{(\beta + h + \mu) - \frac{kh}{(\mu+k)}}$$

$$DN(t) = \frac{h(\rho(t) + \gamma C(t))}{(\mu + k)\left[(\beta + h + \mu) - \frac{kh}{\mu+k}\right]}$$

$$C(t) = \frac{\beta(\rho(t) + \gamma C(t))}{\left[(\gamma + \upsilon + \delta)\left[(\beta + h + \mu) - \frac{kh}{\mu+k}\right]\right]}$$

The above expressions indicate that, perturbation of the equilibrium point will be governed, more importantly, by birth rate $\rho(t)$ and sub- population of diabetics with complications $C(t)$.

From the result in table 2, we see that the disease free equilibrium state of the diabetic population is stable. However the stability decreases with growing values of β and *h.* The meaning of this is that; the dynamics of the system will be stable and predictable for smaller values of β and *h.* This implies that with low level of transiting to $C(t)$ and high level of exercise and low calorie

intake $\left(h = \frac{Ex}{d}\right)$, the system will be stable and hence the menace of diabetes can be controlled with time. However, as β and h increases, the stability of the disease free equilibrium decreases and may ultimately be unstable at some point. This means at that point, it will be difficult to control the menace of diabetes. This is in tandem with the dynamics of diabetics, since, high level of h implies low exercise and higher calorie intake and, high β implies high rate of transiting to $C(t)$, and as a result $C(t)$ will explode. This is dangerous to the population.

5. CONCLUSION

The result of the study established that; birth rate and the number of diabetics with complications determine the equilibrium solutions and that the contribution of birth rate to the dynamics of the diabetic population depends on the dynamics of the diabetic gene and life style. This is in tandem with the dynamics of the diabetics since diabetes is not contagious, rather, a genetic disorder with chromosomes 1q21-1q24, 2q37, 12q24, and chromosome20, as the locus that harbor the gene that causes diabetes. The work recommended that efforts should be geared towards controlling life style and reproductive practice of a population to manage Diabetes and curtail the genetic spread of the disease respectively.

REFERENCES

A. Bouteyab EH. Twizell K. Achouayb, and A. Chetouani(2004). A Mathematical Model for
the burden of diabetes and its complications, *Biomedical Engineering online*, Vol. 3, No 20., pp 1-8

Adamu G. Bakari and Geofrey C. Onyemelukwe (2005) *Int J Diabetes & Metabolism* 13:24-27

Bellman and Coke (1963). Differential Difference Equations, *A report prepared for United States Air force Project Rand* copyright© (1963) by the RAND Corporation, Printed in the United States of America

Bouteyab A., Twizell Eh, Achouayb K, And Chetouani (2004). *Biomedical engineering online*, Vol. 3, No 20.

Brian T., Keith P., Gerda D., Robert M. M., and Diane T. F. (2000). A Model of b-Cell Mass,Insulin, and Glucose Kinetics: Pathways to Diabetes, *Journal of theoretical biology*; vol. 206.

Christian M., Jean M. D., Stephen I. W., and John E. G. (2001), Role of human liver, kidney, and skeletal muscle in postprandial glucose homeostasis, *Am J Physiol Endocrinol Metab* 282: E419–E427, 2002; 10.1152/ajpendo.00032.2001.

David J. H., Ian M., Jian'an L., Timothy M. C. and Nicholas J. W. (2003); Typical Type II
diabetes mellitus and HFE gene mutations a population based case- control study; *Human Molecular Genetics*, Vol. 12, No. 12, pp 1361-1365

David Whiting (2010); The implications of the new Chinese prevalence study, *Diabetes Voice*, Volume 55, issue 1

EBenezer A. N., Osaretin J. O. Anele E. I.,Aaron O.,Seye B. (2003); Type II diabetes in adult
Nigerians: a study of its prevalence and risk factors in Port Harcourt, Nigeria. *Diabetes Research and Clinical Practice* 62 (2003) 177_/185

Florence D., Timo K., Cecelia M. L., Steven W., Stephen G. Candice D., Peter A., Marketa, S., Andrew H., Christian D., Tiinamaiji, T., Mark I. M., Philippe F. and Leif C. G. (2003)

Meta analysis of four European genome screens, *Human Molecular Genetics*, vol. 12, No. 15, pp 1865- 1873.

Hans S. I., Jonathan S., Paul Z. (2003) Guidelines for the detection of Diabetes Mellitus-Diagnostic criteria & rational for screening. *Clin Biochem Rev*., 24, Pp77-80

Ibrahim I.A., Yusuf H., E.J.D. Garbal (2009) Mathematical Model for the dynamics of diabetic population, *ICASTOR Journal*, vol. 3, no. 1, Pp85-100

Ibrahim I. A., Yusuf H., E.J.Garbal (2012) A Mathematical Model for attenuating the spread of diabetes and its management in a population, *Global Journal of Mathematical Sciences*, vol. 11, no.1 & 2 , Pp 1-14

Ibrahim I. A., Yusuf H. and E.J.D. Garba (2014). Mathematical Model for the Dynamics of Glucose
Regulatory System under the Combined Effect of Dieting and Physical Activity. *Int. J. Pure Appl. Sci. Technol*., 20(1) (2014), pp. 88-100

Jean-Jacques G.(1999). Interaction of physical activity and diet: implications for insulin- glucose dynamics, *Journal of Public Health Nutrition*; 2(3a), pp 363–368

Margarita T., Garcia T. R., Robert A. K., D. C. Rao, and Claude B. (2005). Endurance training-induced changes in insulin sensitivity and gene expression, *Am J Physiol Endocrinol Metab* 288: E1168– E1178.

Microsoft ® Encarta ®(2008) Diabetes complications, *Encarta Encyclopedia*

Sarah W., Gojka R., Anders G., Richard S., Hilary K.(2004), Global Prevalence of Diabetes,Estimates for the year 2000 and projections for 2030; *Diabetes Care* 27:1047–1053, 2004

World Health Organisation, WHO (1999), Report of WHO consultation,WHO/NCD/NCS/99.2 on diagnosis and classification of diabetes Mellitus.

World Health Organisation, WHO (2000) World Data Table, Pp 88

World Health Organisation, WHO (2003) Today's Challenges, Geneva world health organization. http://www.who.int/whr/2003/en
www.diabeteswellness.net,

APPENDIX A

Table 2: Adopted from A. Bouteyab *et al* (2004) pp5

S/N	PARAMETER	VALUE
1	μ	0.02
2	γ	0.08
3	υ	0.05
4	δ	0.05