A mathematical model on the effect of non-adherence to drugs on diabetes control

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Abstract—A compartmental mathematical model for diabetes is developed. The model describes the dynamics of the spread of Type-2 diabetes. A theoretical investigation in the non-adherence to drugs is investigated. A system of differential equations is analysed by stability analysis, the non-trivial critical point obtained is locally asymptotically stable under the given conditions. In-host mathematical model for glucose tolerance test (GTT) is considered, actual glucose data values are fitted using Matlab least squares curve fitting technique. Two methods are used to numerically compute the distributions of steady states of diabetic sub-populations. The Gauss-Seidel method is more accurate than the Jacobi method. The results show that more than 50% of clinical diagnosis effort need to be applied to have more diagnosed population than undiagnosed. Non-adherence to drugs make the control of diabetes difficult. Other non-clinical activities such as campaigns against unhealthy lifestyles can help control diabetes. The GTT model show that if strict diet and medication is followed diabetes can be controlled.

Keywords—Type-2 Diabetes, non-adherence, Gauss-Seidel Method, Jacobi Method, GTT model

I. Introduction

DIABETES is a disease that is caused by the body's failure to produce insulin which regulates the amount of blood sugar [1]. Insulin is produced by beta cells that are in the pancreas, when these beta cells die, the amount of insulin produced is low. This is normally caused by lack of physical activity and obesity [2]. Diabetes is also hereditary, it develops on people who are genetically susceptible and is now an epidemic [3] The failure to control blood sugar levels leads to more complications. The details on how diabetes is caused is not clearly understood [4].

About 3% of the world's population is diagnosed with diabetes. Most diabetes deaths are related to undiagnosed cases and non-adherence to drugs. The world's health care cost on diabetes is increasing as the number of cases keeps on increasing, this makes the disease difficult to control. Research has shown that 9.9% of the world's population will have diabetes by 2030. Diabetes is responsible for between 2.5-15% total healthcare expenditure, costing about 153 billion dollars and is expected to double by 2025 [5].

The diagnoses of diabetes disrupt one's own life and involves a lot of work in terms of taking medication and glucose monitoring [6]. This brings about the aspect of non-adherence. On first diagnoses some people fail to accept this condition and do not take medication immediately. This is sometimes referred to as the denial stage. Other non-adherence issues arise from religious affiliation; In some African religious beliefs, taking medication is regarded as a sin and among some African Americans, there is an expectation that devine intervention can help healing diabetes [7]. This creates a large number of non-adherence cases. 50% of people are diagnosed with diabetes at is complication stage [8]. These complications include kidney failure, amputation, blindness, cardiovascular diseases [9].

The study of diabetes mathematical models has been studied by among others, Enagi et al. [10] who studied diabetes complications using the Homotopy perturbation method. Appuhammy et al. [11] investigated age-specific diabetes incidence using body mass index. Rusado studied mathematical models for detecting diabetes. Duun-Henriksen et al. [12] developed a stochastic differential equation based on Grey-box models in diabetes, Coll et al. [13] used a matrix model to study and estimate future diabetes prevalence, these studies advanced the study of diabetes and its complications.

Diabetes can be treated but patients in most cases do not recover completely; the treatment involves taking medication in the form of tablets in type 2 diabetes. Patients with type 1 diabetes need to inject themselves with insulin shots [14]. The treatment if adhered to regulates the amount of blood sugar to acceptable levels. The treatment of diabetes might be delayed until complications occur, it might also be difficult to distinguish between two types of diabetes diseases [15]. The treatment involves taking measurement using a glucometer to monitor blood sugar levels on a day to day basis. The control and taking of medication has been advanced in some cases patients wear special watches that send glucose signals to mobile phones. Some systems automatically inject insulin as and when is required [16].

The study of diabetes can also be considered at cell level. The glucose-insulin mathematical models give a clear understanding of what take place in the blood stream glucose levels. Noguchi et al. [17] considered a glucose-insulin model metabolism for Type 1 diabetes with digestion and absorption of carbohydrates. Wang et al. [18]considered the glucose–insulin system, therapies and its applications. Kwach et al. [19] investigated mathematical modeling of insulin therapies in patients with Type 2 diabetes. Singh [20] studied mathematical modelling for detecting diabetes. Kuma and Kumar [21] investigated mathematical models for glucose-insulin regulatory system of diabetes, The glucose tolerance test model (GTT) is considered in Ackerman et al. [22]. This model is used to determine if a person is diabetic or not by considering data recordings of glucose levels.

This paper aims at developing a compartmental model of diabetes which include those diagnosed, not diagnosed, those under treatment and non-adherence cases. A unique mathemat-

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ical model in the form of differential equations is developed. Stability analysis is carried out to investigate the conditions under which the disease persist. A numerical method is used to predict the population distribution for all the four groups. The data from glucose level of a diabetic individual is fitted using MATLAB least squares fitting. The data is used to determine if the individual is indeed diabetic or not. Data from eating low GI and high GI foods is considered and its effect on glucose levels is investigated. The GTT model is used to perform these investigations.

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II. Mathematical formulation

A compartmental model for diabetes is developed, the population is divided into four, those diagnosed with diabetes x_1, undiagnosed cases x_2, those under treatment x_3 and non-adherence cases x_4. (See Figure 1)

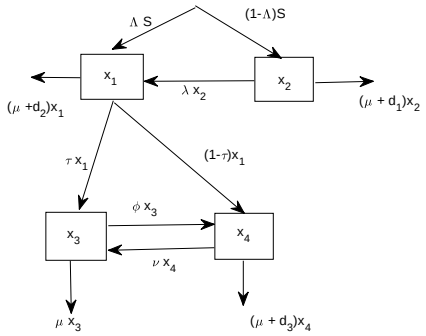

Fig. 1. Schematic for diabetes compartmental model

The differential equations describing the dynamics in Figure 1 is given by

$$\dot{x_1} = \Lambda S - (\mu + d_2 + 1)x_1 + \lambda x_2, \quad (1)$$
$$\dot{x_2} = (1-\Lambda)S - (\mu + d_1 + \lambda)x_2, \quad (2)$$
$$\dot{x_3} = \tau x_1 - (\mu + \phi)x_3 + \nu x_4, \quad (3)$$
$$\dot{x_4} = (1-\tau)x_1 + \phi x_3 - (\mu + d_3 + \nu)x_4, \quad (4)$$

with initial conditions

$$t > 0, x_1(0) = x_{10}, x_2(0) = x_{20}, x_3(0) = x_{30},$$
$$x_4(0) = x_{40}. \quad (5)$$

where Λ is the rate of diagnosis, μ is the natural mortality rate, d_1 is the death due to undiagnosed cases, d_2 is the disease induced death in diagnosed cases. τ is the rate of treatment from the diagnosed cases. ϕ is the rate of non-adherence to drugs, ν is the rate of movement from the non-adherence class to the treatment class. d_3 is the disease induced death due to non-adherence to drugs. It is assumed that those undiagnosed stay a long time in this class and will move to the diagnosed class at a rate λ. Those in the diagnosed class stay in this class for a short time and immediately move to the classes of treatment and non-adherence to drugs. The total population is given by

$$N(t) = x_1(t) + x_2(t) + x_3(t) + x_4(t) \quad (6)$$

at time t, this population is approximately 3% of the world's population.

A. Stability analysis

In the above model, S is the steady-state value of the incidence of diabetes. The model reaches its critical point when $\dot{x_1}, \dot{x_2}, \dot{x_3}\dot{x_4}$ in eqns (1)-(4) vanish together,

$$\Lambda S - (\mu + d_2 + 1)x_1 + x_2 = 0, \quad (7)$$
$$(1-\Lambda)S - (\mu + d_1 + \lambda)x_2 = 0, \quad (8)$$
$$\tau x_1 - (\mu + \phi)x_3 + \nu x_4 = 0, \quad (9)$$
$$(1-\tau)x_1 - (\mu + d_3 + \nu)x_3 + \phi x_3 = 0, \quad (10)$$

solving (7)-(10) we obtain

$$x_1^* = \frac{\Lambda S(\mu + d_1) + \lambda S}{(1 + \mu + d_2)(\mu + d_1 + \lambda)}, \quad (11)$$
$$x_2^* = \frac{(1-\Lambda)S}{(\mu + d_1 + \lambda)}, \quad (12)$$
$$x_3^* = B^* G, \quad (13)$$
$$x_4^* = B^* \left[\frac{R((1-\tau)\mu + \phi)}{(R-1)}\right], \quad (14)$$

where

$$B^* = \frac{\Lambda S(\mu + d_1) + \lambda S}{(1 + \mu + d_2)^2(\mu + d_1 + \lambda)(\mu + \phi)^2},$$
$$G = \frac{R((1-\tau)\mu + \phi) + \tau(\mu + \phi)(R-1)(\mu + d_2 + 1)}{(R-1)}.$$

we write the equations (1)-(4) in the form

$$x(t) = Ax(t) + h(t), t > 0, \quad (15)$$

subject to $x(0)$ as given in (5), where

$$x(t) = \begin{pmatrix} x_1 \\ x_2 \\ x_3 \\ x_4 \end{pmatrix},$$

$$A = \begin{pmatrix} -(\mu + d_2 + 1) & \lambda & 0 & 0 \\ 0 & -(\mu + d_1 + \lambda) & 0 & 0 \\ \tau & 0 & -\mu + \phi & \nu \\ (1-\tau) & 0 & \phi & -(\mu + d_3 + \nu) \end{pmatrix}$$

$$h(t) = \begin{pmatrix} \Lambda S \\ (1-\Lambda)S \\ 0 \\ 0 \end{pmatrix}$$

The characteristic equation of the matrix A is given by

$$x^4 + \xi_0 x^3 + \xi_1 x^2 + \xi_2 x + \xi_3 = 0, \quad (16)$$

where

$$\begin{aligned}
\xi_0 &= 4\mu + d_1 + d_2 + d_3 + \lambda + \phi + \nu + 1,\\
\xi_1 &= (2\mu + d_1 + d_2 + \lambda + 1)(2\mu + \phi + d_3 + \nu)\\
&\quad + (\mu + d_2 + 1)(\mu + d_1 + \lambda) + (R - 1)\\
\xi_2 &= (\mu + d_2 + 1)(\mu + d_1 + \lambda)(2\mu + \phi + d_3 + \nu)\\
&\quad + (2\mu + d_1 + d_2 + \lambda + 1)(R - 1)\\
\xi_3 &= (\mu + d_2 + 1)(\mu + d_1 + \lambda)(R - 1) \qquad (17)
\end{aligned}$$

where $R = (\mu + \phi)(\mu + d_3 + \nu)/\nu\phi$. R is the threshold value at which the stability of the system changes. If $R < 1$ the disease does not persist and if $R > 1$ the disease persist.

The roots of the characteristic equation correspond to the eigenvalues of the matrix A, if $-x$ is substituted in the characteristic polynomial (16). There are four sign changes indicating that there are four negative roots. This result indicates that the non-trivial point shown in equations (11)-(14) is asymptotically stable when $R > 1$.

III. Numerical Solution

In this section we compute the diabetes population distributions using numerical methods, we compare two numerical solutions for solving linear equations. The two methods considered are the Jacobi and Gauss-Seidel methods. The methods are used to solve systems of the form $Ax = b$. Equation (12) takes this form at steady states $\dot{x}(t) = 0$, this becomes a system of linear equations of the form

$$a_{11}x_1 + a_{12}x_2 + a_{13}x_3 + a_{14}x_4 = R_1, \qquad (18)$$
$$a_{21}x_1 + a_{22}x_2 + a_{23}x_3 + a_{24}x_4 = R_2, \qquad (19)$$
$$a_{31}x_1 + a_{32}x_2 + a_{33}x_3 + a_{34}x_4 = 0, \qquad (20)$$
$$a_{41}x_1 + a_{42}x_2 + a_{43}x_3 + a_{44}x_4 = a, \qquad (21)$$

Where

$$a_{11} = -(\mu + d_2 + 1), a_{12} = \lambda, a_{13} = 0, a_{14} = 0, \qquad (22)$$
$$a_{21} = 0, a_{22} = -(\mu + d_1 + \lambda), a_{23} = 0, a_{24} = 0, \qquad (23)$$
$$a_{31} = \tau, a_{32} = 0, a_{33} = -(\mu + \phi), a_{34} = \nu, \qquad (24)$$
$$a_{41} = 1 - \tau, a_{42} = 0, a_{43} = \phi, a_{44} = -(\mu + d_3 + \nu), \qquad (25)$$

Parameters used in the model $\mu = 0.01,\ d_1 = 0.03,\ d_2 = 0.02,\ d_3 = 0.01,\ \lambda = 0.1,\ \phi = 0.2,\ \nu = 0.03,\ \tau = 0.3,\ \Lambda = 0.5$ as in [15].

IV. The Jacobi method

To solve the system using the Jacobi method, the coefficient matrix does not have zeros in its diagonal entries. If there are zeros, then row operations must be performed to avoid zeros in the diagonal. To solve this system using the Jacobi method we solve each equation for x_1, x_2, x_3 and x_4. We use the first approximation usually $(0, 0, 0, 0)$ if there is no available first approximations. The values that are obtained in the first iteration are then used in the second iteration and so on until a solution is achieved. In this case we use the procedure as follows;

$$x_1 = \frac{1}{a_{11}}\left(R_1 - a_{12}x_2 - a_{13}x_3 - a_{14}x_4\right), \qquad (26)$$
$$x_2 = \frac{1}{a_{22}}\left(R_1 - a_{21}x_1 - a_{23}x_3 - a_{24}x_4\right), \qquad (27)$$
$$x_3 = \frac{1}{a_{33}}\left(-a_{31}x_1 - a_{32}x_2 + a_{34}x_4\right), \qquad (28)$$
$$x_4 = \frac{1}{a_{44}}\left(-a_{41}x_1 - a_{42}x_2 - a_{43}x_3\right). \qquad (29)$$

By using the parameters in Table I we obtain the results shown below

TABLE I
NUMERICAL DIABETES DISTRIBUTIONS OBTAINED BY THE JACOBI METHOD, VALUES$\times 10^6$

Iter	x_1	x_2	x_3	x_4
1	2	950	0	0
2	11	950	66	14
3	11	950	83	73
4	11	950	83	73
5	11	950	83	73

V. Gauss-Seidel Method

The Gauss-Seidel method is different from the Jacobi method. The new values are used as soon as they are known. If x_1 is obtained in the first equation it is immediately used in the second equation to get x_2. Next when x_2 is obtained it is used in the next equation to obtain x_3 and so on. The numerical scheme can be represented as follows;

$$x_1^{k+1} = \frac{1}{a_{11}}\left(R_1 - a_{12}x_2^k - a_{13}x_3^k - a_{14}x_4^k\right) \qquad (30)$$
$$x_2^{k+1} = \frac{1}{a_{22}}\left(R_1 - a_{21}x_1^{K+1} - a_{23}x_3^k - a_{24}x_4^k\right) \qquad (31)$$
$$x_3^{k+1} = \frac{1}{a_{33}}\left(-a_{31}x_1^{k+1} - a_{32}x_2^{k+1} + a_{34}x_4^k\right) \qquad (32)$$
$$x_4^{k+1} = \frac{1}{a_{44}}\left(-a_{41}x_1^{k+1} - a_{42}x_2^{k+1} - a_{43}x_3^{k+1}\right) \qquad (33)$$

By using the parameters in Table II we obtain the following results.

The last row shown from both the Jacobi and Gauss-Seidel methods is the steady state values of the distributions for all the four classes of diabetics.

TABLE II
NUMERICAL DIABETES DISTRIBUTIONS OBTAINED BY THE GAUSS-SEIDEL METHOD, VALUES$\times 10^6$

Iter	x_1	x_2	x_3	x_4
1	2	950	12	14
2	11	950	66	73
3	11	950	83	73
4	11	950	83	73

Tables I and II show the numerical steady states for the populations $(x_1^*, x_2^*, x_3^*, x_4^*) = (11, 950, 83, 73)$ values are of the order of 10^6.

VI. Results and Discussions

From the stability analysis carried out in this study, the critical point is stable. This means that the system tends to this state even if the system is disturbed or perturbed. The steady state was also alternatively calculated by using the two methods that are used to solve linear system of equations. These methods are the Jacobi and Gauss-Seidel methods. The comparisons show that the Gauss-Seidel method is more accurate than the Jacobi method. The solution is obtained using fewer iterations in the Gauss-Seidel method than in the Jacobi method. Simulations for the effect of varying some parameters on the variable is performed and shown in the form of figures.

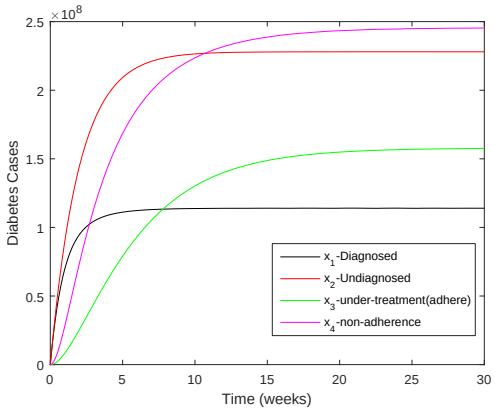

Fig. 2. Diabetes population simulations when rate of diagnosis $\Lambda = 0.5$.

Figure 2 shows the diabetes population simulations for the case when the disease related death rates $d_1 = d_3 > d_2$. If clinical diagnosis efforts are $\Lambda = 0.5$, the diagnosed cases are less than the undiagnosed cases. More effort has to be applied to get more diagnosed cases than undiagnosed cases. It is also noted that under these assumptions the treated cases are less than the number of non-adherence cases.

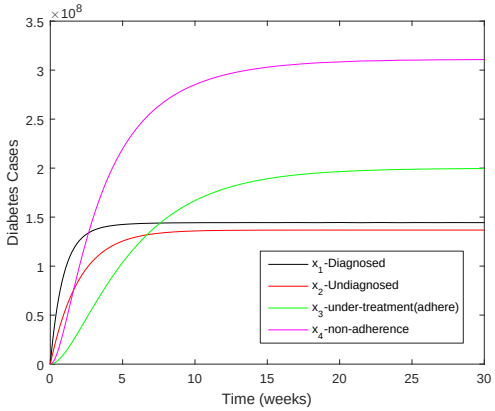

Fig. 3. Diabetes population simulations when rate of diagnosis $\Lambda = 0.7$.

Increasing clinical diagnosis effort to $\Lambda = 0.7$ result in more diagnosed cases that undiagnosed cases as shown in Figure 3. Further increasing this effort to $\Lambda = 0.8$ result in even more diagnosed cases as shown in Figure 7. The existence of non-adherence to drugs cases make it difficult to increase treated cases.

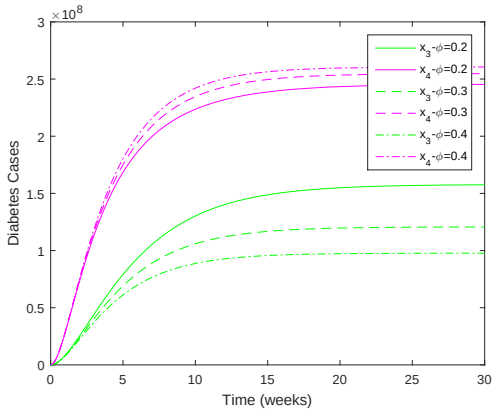

Fig. 4. Effect of varying non-adherence rate ϕ on treated and non-adhering populations

Figure 4 shows the effect of increasing non-adherence to drugs rate ϕ on the treated and non-adherence to drugs cases. Increasing ϕ result in the decrease in the treated cases and increase non-adherence cases. Under these assumptions it is noted that the decrease of treated cases is more enhanced than increasing non-adherence cases. Religious beliefs also contribute to non-adherence to drugs resulting in high mortality rate among those not adhering to their medication. This is attributed to the fact that there are some non-adherence cases that begin to take their drugs again with the rate ν. This means that non-clinical efforts to diagnose people is also very effective.

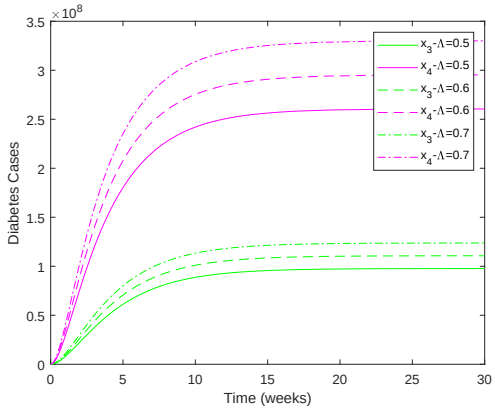

Fig. 5. Effect of varying clinical diagnosis efforts Λ on treated and non-adhering populations

Figure 5 shows the effect of increasing clinical diagnosis effort on the treated and non-adherence cases. Increasing Λ result in the increase of treated cases. This also increase non-adherence to drugs cases. The reason why non-adherence cases increase is due to those individuals who stop taking their drugs by the rate ϕ.

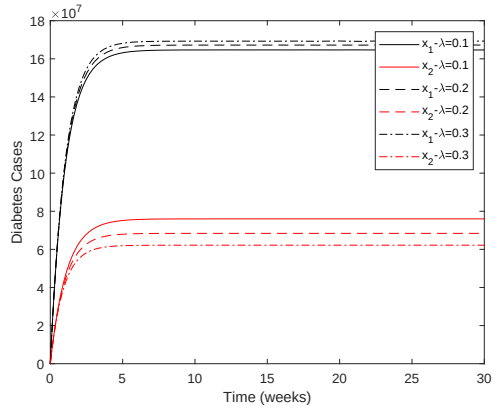

Fig. 6. Effect of varying non-clinical diagnosis efforts λ on diagnosed and non-diagnosed populations

Figure 6 depicts the effect of increasing non-clinical efforts on diagnosed and undiagnosed cases. Increasing λ result in the decrease in undiagnosed cases and increasing diagnosed cases. These interventions help to reach those who will have missed clinical diagnosis and end up being diagnosed. The effect is more enhanced for the case of undiagnosed cases.

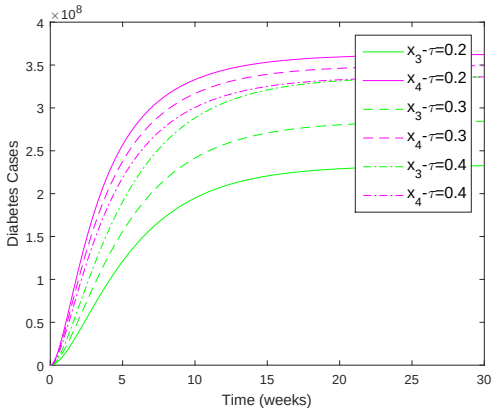

Fig. 7. Effect of varying treatment efforts τ on treated and non-adhering populations

In Figure 7 increasing the treatment effort result in increased treated populations and decrease non-adherence cases. If there is 100% adherence to drugs there will be no non-adherence cases. The in-host mathematical models for the group taking medication and on strict diabetic diet and change of diet will be discussed in the next section.

VII. In-host diabetes models

In the previous section we discussed the human population for diabetes. These included the diagnosed, not diagnosed and those taking treatment and those not adhering to medication. In-host models are those that consider what exactly happens at cell level. In this section we consider the glucose-insulin mathematical model.

The problem of diabetes emanates from the glucose-insulin imbalance, this is caused by the death of $\beta-$ cells in the pancreas [20], [19]. The production of insulin is therefore impeded and hence the control of blood sugar level is affected [18]. When insulin is produced it affects glucose concentration in a number of ways; enhances glucose transport through cell membranes, it helps conversion of glucose to glycogen in the liver which is stored for future use [17]. During this process a hormone called adrenalin is produced to convert glycogen back into glucose [21].

In this section we analyse the glucose-insulin models for diabetics who are diagnosed and do not take medication and those that take medication. The mathematical model that we consider is the one proposed by Ackerman et al. [22]. The model is derived from a system of first order differential equations that are stated in [19], [20] as

$$\dot{G}(t) = f_1(G, I) + J(t) \tag{34}$$
$$\dot{I}(t) = f_2(G, I) \tag{35}$$

Where $G(t)$ and $I(t)$ are the glucose and insulin concentrations at any time t. $J(t)$ is the source term for food input. This model is the linearised and written in matrix form. The characteristic equation of the coefficient matrix of this system has complex eigenvalues with negative real parts. This means that the solution of the differential equation is of the form

$$G = e^{-\alpha t}(A\cos(\omega t) + B\sin(\omega t)) \tag{36}$$

This is clearly an oscillation or spring-like process, this equation (36) can then be written as

$$G = G_{eq} + Ae^{-\alpha t}\cos(\omega(t-\delta)) \tag{37}$$

where G is the glucose concentration, G_{eq} is the equilibrium level of glucose concentration, $A, \alpha, \omega, \delta$ are constants to be determined. In this model we need to determine the time it takes for an individual's glucose level to get back to normal. If it takes a less time it means the individual is normal and if it takes more time then it means that the individual is likely to be diabetic. From the data that was taken from a diabetic individual under medication and the other one not diabetic we calculate the period. The critical period $T_c = 2\pi/\omega_c$. where $\omega_c^2 = \omega^2 + \alpha^2$. According to Ackerman et al. [22] If $T_c < 4$ then the individual is normal and if $T_c > 4$ then the individual is diabetic. At this stage two theoretical cases are considered, one is a comparison of a normal individual and Type-2 diabetic, the other one is a comparison of a normal and Type-1 diabetic individuals.

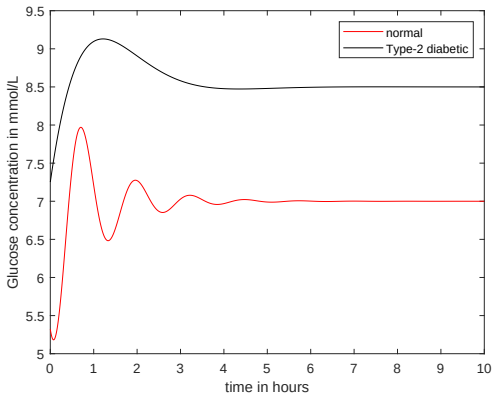

Fig. 8. GTT on normal and Type-2 diabetic

In Figure 8, the glucose tolerance test model shows that a normal individual takes a shorter time to reach the equilibrium glucose concentration, the concentration oscillates several times about this value $G_{eq} = 7$. In this case $T_c = 1.26$. A Type-2 individual takes longer to reach the equilibrium value of $G_{eq} = 8.5$ and $T_c = 6.26$.

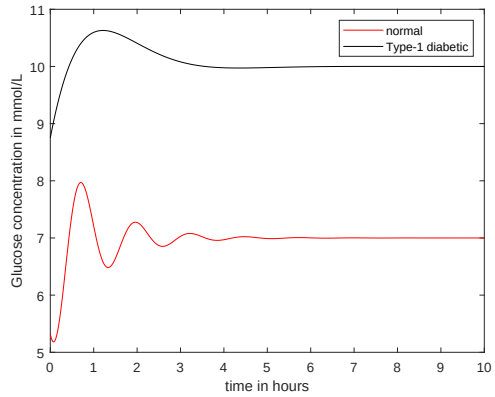

Fig. 9. GTT on normal and Type-1 diabetic

In Figure 9 an individual with Type-1 diabetes takes longer to reach an equilibrium value $G_{eq} = 10$. The GTT model can be used to check actual data from any individual to determine if they are diabetic. In this paper we use data obtained from the author, this data is then fitted using MATLAB least squares curve fit. The data is was obtained when different kinds of foods were consumed. The following tables show different data sets and their corresponding fitted models. From the GTT model shown in equation (37), least squares has to determine the constants $G_{eq}, A, \alpha, \omega$ and δ. The determined values are the ones that describe the glucose concentration profile for an individual with the given data.

TABLE III
DATA FOR GLUCOSE LEVELS FOR DIABETIC INDIVIDUAL ON DIET AND MEDICATION

Time	0	1	2	3	4	5	6
Glucose (mmol/L)	3.8	8.7	5.9	5.7	4.3	4.8	5.3
Time	7	8	9	10	11	12	13
Glucose (mmol/L)	4.2	5.9	7.0	8.1	5.3	5.3	5.1

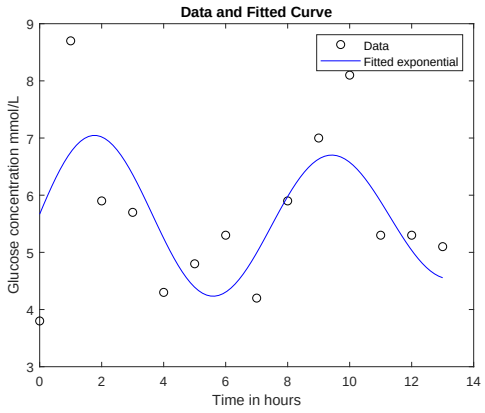

Fig. 10. GTT on Type-2 diabetic on medication and diet

The glucose concentration of the data in Table III was fitted using MATLAB least squares curve fit and the parameters were obtained as follows;

$$G_{eq} = 5.55,\ A = 1.59,\ \alpha = 0.034,\ \omega = 0.82,\ \delta = 1.82$$

The glucose concentration mathematical model for this individual taking medication and on strict diabetic diet is given as

$$G(t) = 5.55 + 1.59e^{-0.034t}\cos[0.82(t - 1.82)]. \quad (38)$$

From this data the value of critical period $T_c = 7.65$, this indicates that the individual is indeed diabetic. A second data set was considered in which the individual changed diet from low GI (Glycaemic Index) food to high GI food. The data set is recorded in Table IV

TABLE IV
DATA FOR GLUCOSE LEVELS FOR LOW GI FOOD

Time	0	1	2	3	4	5
Glucose (mmol/L)	4.1	6.9	5.3	5.2	4.7	7.0

TABLE V
DATA FOR GLUCOSE LEVELS FOR HIGH GI FOOD

Time	0	1	2	3	4	5
Glucose (mmol/L)	9.5	4.3	3.4	7.3	5.4	10.7
Time	6	7	8			
Glucose (mmol/L)	3.5	5.2	9.1			

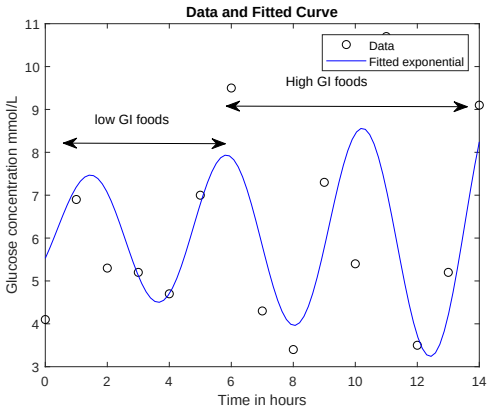

Fig. 11. GTT on Type-2 diabetic on change of diet from low GI to high GI

Table IV shows data in which the diabetic individual was on medication and was on low GI diet, this included brown rice, brown bread, mabele (sorghum mealie-meal),oats porridge. The diet was then changed to high GI foods which included white bread, white pap, red meat, the data for glucose levels is recorded in Table V. The amplitude of the fitted model increased with increasing time. Glucose levels increased with high GI foods and it became difficult to control.

VIII. Conclusion

The problem of diabetes control caused by non-adherence to drugs is studied. A system of differential equations is analyzed by considering the stability at steady states. The results show that the equilibrium point is asymptotically stable. The resulting system of linear equations is solved numerically to obtain the population distribution of different diabetic groups. The two methods were used and compared. The Gauss-Seidel method is more accurate than the Jacobi method. The Gauss-Seidel converges much faster than the Jacobi method converging after three iterations while the Jacobi after two iterations. To effectively control diabetes, more than 70% clinical diagnosis effort is required. Non-adherence to drugs and failure to complement diabetes awareness with non-clinical efforts make the control of diabetes more difficult. Treatment efforts need to be stepped up much more than non-adherence. From the GTT model analysis, taking low GI foods and medication effectively control diabetes (This produce a cosine wave with constant amplitude). Foods with high GI spike glucose levels. The glucose concentration data can used to determine if a patient is diabetic or not. This method considers data over a long period of time as opposed to a single testing method. Mabele (sorghum mealie-meal) can be used to effectively control blood sugar levels.

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