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Mathematical Modeling for detecting diabetes in bloods

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Abstract
Diabetes is a syndrome of disordered metabolism, usually due to a combination of hereditary and environmental causes, resulting in abnormally high blood sugar levels. Various hormones in our body such as insulin, growth hormone, and glucagon control blood glucose levels, epinephrine best known as adrenaline, glucose corticoids and thyroxin.The two most common forms of diabetes are due to eighter a diminished production of insulin (Type1 diabetes).Both lead to hyperglycemia, which largely causes the acute signs of diabetes: excessive urine production. Resulting compensatory thirst and increased fluid intake, blurred vision, unexplained weight loss, lethargy, and changes in energy metabolism. We will explain how the hormone, insulin is activated and how it affects glucose levels in blood. We present a mathematical model that determines diabetes in patients based in the results on the glucose intolerance test of 5 hours. The model is in line with the one proposed by E. Ackerman (1969). The model is based on a 2×2system of non-homogenous ordinary differential equation. A compartmental model is used to determine the coefficient parameters of the system based on actual data from GTT. The simultaneous also provide an indicator similar to the one proposed by E. Ackerman (1969), to diagnose a diabetic condition.

Keywords: Differential equation, diabetes, glucose, insulin, compartmental model

Introduction

Diabetes is a disease of the metabolism where the body cannot adequately respond to the ingestion of any type of carbohydrate. This is the result of the inability of the body to adequately produce the hormone insulin, which allows cells to absorb glucose from the blood. The source of this inadequacy varies between the types of diabetes. In type 1 diabetes, the cells of the pancreas responsible for producing insulin (the islets of Langer hans) are destroyed by the body's own immune system and the production of insulin is therefore completely absent. In type 2 diabetes, insulin is still produced by the body, but the body is very resistant to its erects. The result in both cases is the same: high levels of glucose in the blood, also known as hyperglycemia. Because this effect is much more drastic and therefore more obvious and easier to diagnose in type 1 diabetes, this model will be constrained to the detection and diagnosis of type 2 diabetes.

Type-1 diabetes

Type –1diabetes is also called insulin dependent diabetes mellitus because this disease is characterized by an absolute deficiency of insulin. Beta cells are destructed due to invasion by virus, action of chemical toxins or due to action of autoimmune antibodies. This beta cell necrosis is causes insulin deficiency and caused Type-1 diabetes

Type-2 diabetes

Non- insulin dependent diabetes mellitus or Type-2 diabetes is frequently accompanied by target organ insulin resistance that limits responsiveness to both endogenous and exogenous insulin

Type- 3 diabetes

This type of diabetes is caused by chronic pancreatitis or chronic drug therapy with glucocorticoids, thiazids diuretics, diazotize, and growth hormone and with some protease inhibitors (e.g. saquinavir).

Type- 4 diabetes

This type of diabetes is observed in approximately 4-5% of all pregnancies, due to placental hormones that promotes insulin resistance [5].

For more study about diabetes, rodents such as rat, mouse, hamster, guinea pigs and the rabbits are suitable models. They are used for natural development of study. At present time best and quickest way to induce diabetes is with use of chemicals (alloxan, streptozotocin, dithizone, monosodium glutamates etc.), viruses and genetically diabetic rats. In recent years, scientists and technologists have worked toward Refining techniques that have led to the discovery of chemical agents that physiologically alter the function of the pancreas. The main advantage of using such chemicals is that body changes during and after the induction of diabetes can be observed. The five major diabetogenic agents are chemicals,

Biological agents, peptides, potentiators, and steroids but most commonly used chemicals agents are alloxan and streptozotocin

2. Formulation of Mathematical Model:

It has been formulated in two steps.

Step 1

In the first step, assumptions have been stated, have to identify suitable variables and give the law governing the performance of BGRS.

(i) It is assuming that the following two concentrations adequately describe the performance of BGRS. (1)Concentration of glucose in the blood (G).

(2) Net Hormonal concentration (I).

By net hormonal concentration, it means the cumulative effect of all the relevant hormones with the following sign convection: those hormones which decrease bold glucose concentration (BGC) for example Insulin, are regarded to increase H and hence their contribution to H is taken with positive sign while those hormones which increase BGC for example, Cortical, contribute negatively to I.

(ii) Since, G and I changes with time, we are considering G and I as dependent variables while t as the independent variable.

(iii) From the elementary consideration of the biological facts, which is stated above, the logistic law which is governing the performance of BGRS may be written

$\frac{dc_g}{dt}$ = -$m_1 c_g$-$m_2 c_i$+G (t

$\frac{dc_i}{dt}$ = -$m_3 c_i$-$m_4 c_g$+I (t)

3. Compartmental model for diabetes mellitus:

This model is illustrated in Fig.10.6. This method is determined by the following considerations:

(i) The food that we take gives glucose to the digestive system and from there it goes to blood. The glucose from blood can also go to the digestive system when blood circulates through it.

(ii) Surplus glucose from blood is stored in livers glycogen and, when blood needs glucose, it can be released from the liver and given to the blood compartment.

(iii) Pancreas gives insulin to blood.this insulin is necessary for metabolizing glucose for useful work in tissues.

(iv) Glucose can be directly injected in to the digestive system or blood. Insulin can also be injected in to the blood stream.

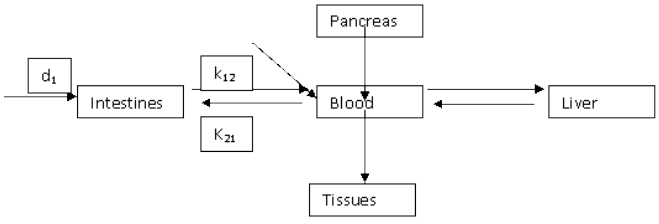

Fig10.6 compartmental model for diabetic mellitus.

We now consider of glucose and insulin in blood.Let c_g and c_i is the excesses of concentration of Glucose and Insulin Respectively, at time t over their equilibrium values. Then we get the differential equations

$$\frac{dc_g}{dt} = m_1 c_g - m_2 c_i + G(t) \quad (1)$$

$$\frac{dc_i}{dt} = -m_3 c_i - m_4 c_g + I(t) \quad (2)$$

Wherem_1, m_2, m_3, m_4 >0 for the following reasons.

(i) if there is excess glucose, it tends to disappear to the liver and to the tissues so that m_1>0

(ii) If there is excess insulin, it helps in metabolizing glucose for the tissues so that m_2 > 0\

(iii) If there is excess glucose, pancreas are induced to secrete insulin so that m_4 > 0

(iv) If there is excess insulin, it tends to disappear so that m_3 >0

Differentiating (1) with respect to t

$$\frac{d^2c_g}{dt^2} = -m_1 \frac{dc_g}{dt} - m_2 \frac{dc_i}{dt} + \frac{dG(t)}{dt} \quad (3)$$

Equation (2) substitute in (3) we get

$$\frac{d^2c_g}{dt^2} = -m_1 \frac{dc_g}{dt} - m_2[-m_3 c_i - m_4 c_g + \text{I (t)}] + \frac{dG(t)}{dt}$$

$$\frac{d^2c_g}{dt^2} = -m_1 \frac{dc_g}{dt} + m_2 m_3 c_i + m_2 m_4 c_g - m_2 I(t) + \frac{dG(t)}{dt}$$

$$\frac{d^2c_g}{dt^2} = -m_1 \frac{dc_g}{dt} + m_3 [-m_1 c_g - \frac{dc_g}{dt} +$$

$$\text{G (t)}] + m_2 m_4 c_g - m_2 I(\text{t}) + \frac{dG(t)}{dt}$$

$$\frac{d^2c_g}{dt^2} + \frac{dc_g}{dt}(m_1 + m_3) + c_g [\ m_3 m_1 + m_4] - m_3 \text{G(t)} - m_2 \text{I(t)} + \frac{dG(t)}{dt} \quad (4)$$

Similarly differentiating equation (2) with respect to t we get

$$\frac{d^2c_i}{dt^2} + \frac{dc_i}{dt}(m_1 + m_3) + c_i [\ m_3 m_{1+} m_2 m_4] = m_1 I(t) + m_4 I(t) + \frac{dI(t)}{dt} \quad (5)\backslash$$

$$\frac{d^2c_g}{dt^2} + 2\alpha \frac{dc_g}{dt} + {\omega_0}^2 c_g = s_1(t) \quad (6)$$

$$\frac{d^2c_i}{dt^2} + 2\alpha \frac{dc_i}{dt} + {\omega_0}^2 c_i = s_2(t) \quad (7)$$

Where $2\alpha = m_1 + m_3$ (8)

${\omega_0}^2 = m_1 m_3 + m_2$ (9)

$s_1(t) = m_3 G(t).m_2 I(t) + \frac{dG(t)}{dt}$ (10)

$s_2(t) = m_1 I(t) + m_4 G()$ t

The solutions of (6) and (7) are

$$c_g = e^{-\alpha t}(A_1 \cos \omega t + A_2 \sin \omega t) + \frac{e^{-\alpha t}}{\omega}[\sin \omega t \int_0^t e^{\alpha t} \cos \omega t\, s_1(t)\, dt - \cos \omega t \int_0^t e^{\alpha t} \sin \omega t\, s_1(t)\, dt] \quad (11)$$

$c_i = e^{-\alpha t}(A_3 \cos \omega t + A_4 \sin \omega t) + \frac{e^{-\alpha t}}{\omega}[\sin \omega t \int_0^t e^{\alpha t}$

$\cos \omega t \, s_1(t)\, dt - \cos \omega t \int_0^t e^{\alpha t} \sin \omega t \, s_2(t\, dt$ (12)

Where $\omega^2 = {\omega_0}^2 - \alpha^2$ (13)

If $s_1 = B\,\delta(t)$, $s_2(t) = 0$, then (11) and (12) give

$c_g = e^{-\alpha t}(A_1 \cos \omega t + A_2 \sin \omega t) + \frac{e^{-\alpha t}}{\omega} B \sin \omega t$ (14)

$c_i = e^{-\alpha t}(A_3 \cos \omega t + A_4 \sin \omega t)$

$c_i = e^{-\alpha t}(A_3 \cos \omega t + A_4 \sin \omega t)$

If t=0, then , also, integrating (3), we get

$(\frac{dc_g}{dt})\, 0 + 2\alpha(c_g)\, 0 + {\omega^2}_0 \int_0^{\omega} c_g \, dt = B \int_0^{\omega} \delta(t)\, dt$

Which gives

$A_2 = 0$

And leads to the solution

$c_g = (B/\omega)e^{-\alpha t} \sin \omega t.$

For $\alpha = 0.014\ min^{-1}$ and ${\omega_0}^2 = 0.0019 min^{-2}$, the graph of c_g is shown in Fig.10.7. This shows damped oscillations. Other normal persons may show a critical damping, where the original equilibrium may be asymptotically reached in about two hours.Diabetic patients take a longer time to recover and the graph corresponding to $\alpha = 0.0048 min^{-1}$, ${\omega_0}^2 = 0.0003 min^{-2}$ is shown in Fig.10.8

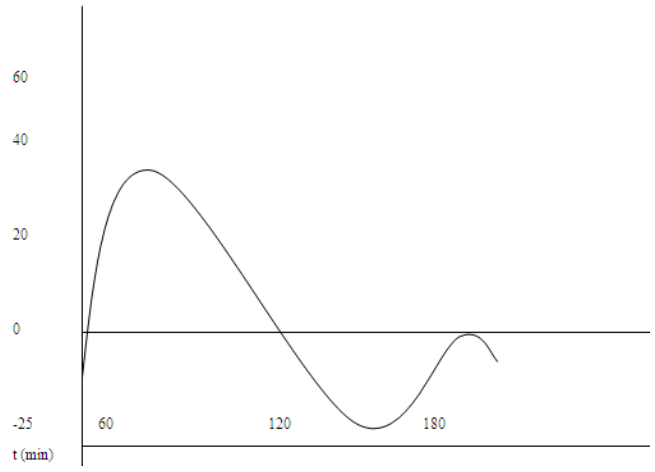

Fig.10.7 Variation of glucose concentration for normal persons

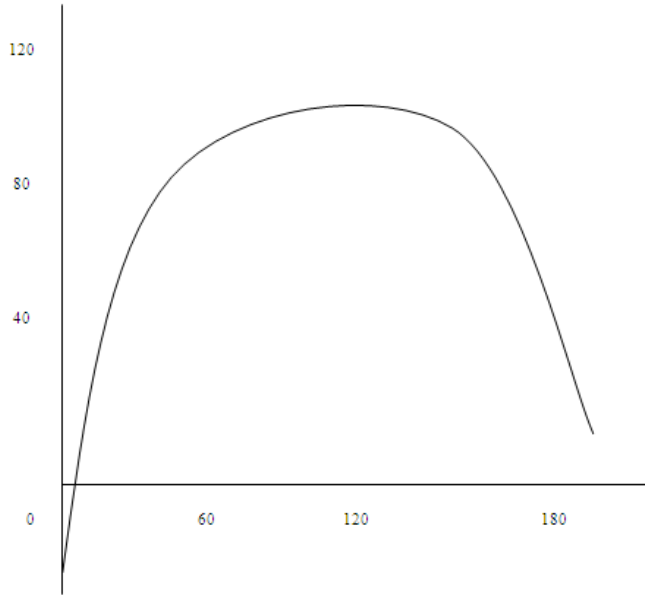

Fig.10.8 variation of glucose concentration for diabetic patients

It has been suggested that ω_0 should be treated as the most significant parameter for distinguishing diabetic persons from normal persons and a period $2\pi/\omega_0$ should be chosen as a classification parameter. Most normal persons show a natural period of less than three hours, whereas most diabetic patients show a natural period greater than five years.

We can also take G (t) $= 0$ and I (t)$\neq 0$, i.e., we can give insulin to normal persons and diabetics and compare their glucose –time curves.

Conclusion:

This model proved to be effective in the diagnosis of mild to moderate type 2 diabetes mellitus using the results of the glucose tolerance test. It was found that the body's blood glucose regulation mechanisms could be likened to a hormonal spring, where too low a blood glucose led to pressure in the upward direction, and too high a blood glucose led to pressure in the downward direction. The model struggles to diagnose severe type 2 diabetes, especially in the time period three to five hours after glucose is ingested. The model is also completely ineffective in the diagnosis of 1diabetes, but the symptoms of type 1diabetes usually become prominent to the point where they are impossible to ignore. The implications made by this model as to the correlation between the natural amount of time it takes to reach equilibrium in blood and the timing of meals in American culture are fascinating, and more research should be done in other cultures to provide better understanding.

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