

Mathematical Modelling Of Predator-Prey Equations

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Abstract:-- The paper intends theoretical and mathematical aspects of the known predator-prey problem that are considered by relaxing the assumptions that interaction of a predation leads to little or no effect on growth of the prey population and the prey growth rate parameter is a positive valued function of time. Prototypes may lead to the most engrossing and conspicuous mathematical result but only those prototypes are endurable which can expound envision or regulate the milieu. A variation method is used to build a numerical solution by differential equations. The dependence of amplitude and a frequency of damped vibrations on parameters characterizing the mobility of species is estimated. Derivations and simulation studies are provided in the paper. Analysis of equilibrium points and stability is also included.

keywords:-- Mathematical modelling, variation methods, numerical solution, differential equations and Simulations.

I. INTRODUCTION

Mathematical modelling is the process of converting a Real-world problem into a mathematical problem and solving them with certain circumstances and interpreting those solutions in terms of real world.

The sample of the flow chart for solving a Real-world problem by using mathematical modelling is explicated below in Fig.1

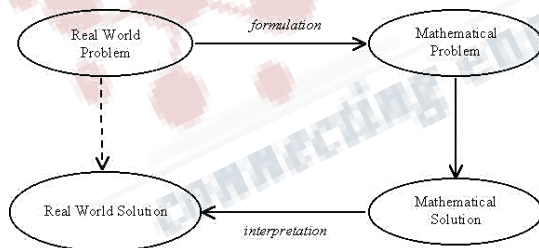


Fig.1

Mathematical modelling in population dynamics has extended a lot of attention and appreciation during the last few decades and among which the models predator-prey systems play an imperative role. The most important element in population models is the “**predator-prey model**” which describes the number (density) of prey consumed per predator per unit time for given quantities (densities) of prey and predator.

The predator-prey model is also called an **Volterra-Lotka model** (or) **Competition model** (One species will depend on other species for their food), to solve this model, we do need a pair of first order, nonlinear, differential equations to describe the population dynamics of biological system in which two species interact, one as predator (Foxes) and other as prey (Rabbits) The population changes through time to the pair of equations:

$$\frac{dx}{dt} = ax + bxy$$

$$\frac{dy}{dt} = dy + cxy$$

Where,

1. x is the Prey population (Rabbits);
2. y is the Predator population (Foxes);
3. xy is the small increment (like distraction by Diseases, earthquakes and etc..)
4. $\frac{dx}{dt}$ and $\frac{dy}{dt}$ Represents the growth rates of the two populations over time;
5. t is the time;
6. a, b, c and d are positive parameters describing the interaction of two species.

This model can be generalized to any number of species competing against each other. One can think

of the populations and growth rates as vectors and the interaction as matrix.

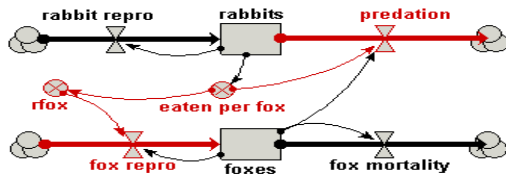


Fig.2

II. HISTORY OF VOLTERRA-LOTKA MODEL

The Volterra-Lotka predator-prey model was initially proposed by Alfred J. Lotka in the theory of autocatalytic chemical reactions in 1910. This was effectively the logistic equation which was originally derived by Pierre François Verhulst. In 1920 Lotka extended the model to "organic systems" using a plant species and an herbivorous animal species as an example and in 1925 he utilized the equations to analyse predator-prey interactions in his book on biomathematics. The same set of equations were published in 1926 by Vito Volterra, a mathematician and a physicist, who took interest in mathematical biology. Volterra's analysis was inspired through his interactions with the marine biologist Umberto D'Ancona who was courting his daughter at the time and later was to become his son-in-law. D'Ancona studied the fish catches in the Adriatic Sea and had noticed that the percentage of predatory fish caught had increased during the years of World War I (1914–18). This puzzled him as the fishing effort had been very much reduced during the war years. Volterra developed his model independently from Lotka and used it to expound D'Ancona's observation.

The model was later extended to include density dependent prey growth and a functional response of the form developed by C.S. Holling; a model that is known as the Rosen Zweig-McArthur model. Both the Lotka-Volterra and Rosen Zweig-MacArthur models were used to explain the dynamics of natural populations of predators and prey, such as the lynx and snowshoe hare data of the Hudson's Bay Company and the moose and wolf populations in Isle Royale National Park.

In the late 1980s an alternative to the Lotka-Volterra predator-prey model (and its common prey dependent generalizations) emerged, the ratio dependent or Arditi-

Ginzburg model. The validity of prey or ratio depends on the models that has been debated.

III. PREDICTIONS OF THE MODEL

There is a lot of available space, the foxes are depending on rabbits for their food and obviously rabbits feed on anything.

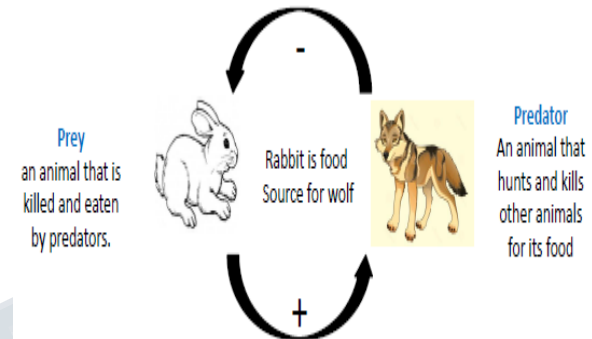


Fig.3

1. If Rabbits are more, Foxes population will increase logistically
2. If no Rabbits, Foxes population will decrease
3. If no Foxes, Rabbits population will increase
4. If less Foxes, less Rabbits can be supported, normal Rabbits population begins



Fig.4

In the absence of fox, the rabbit population grows at a rate proportional to its current population; thus $\frac{dx}{dt} = ax$ when $y = 0$ with $a > 0$, it results in the increase in rabbit population.

In the absence of Rabbits, the foxes die out; thus $\frac{dy}{dt} = -dy$ where $x=0$ $d>0$, it results in decrease in the fox Population,

-ve sign represents decay in the fox population.

The results of Both the assumptions are shown in the Fig.5

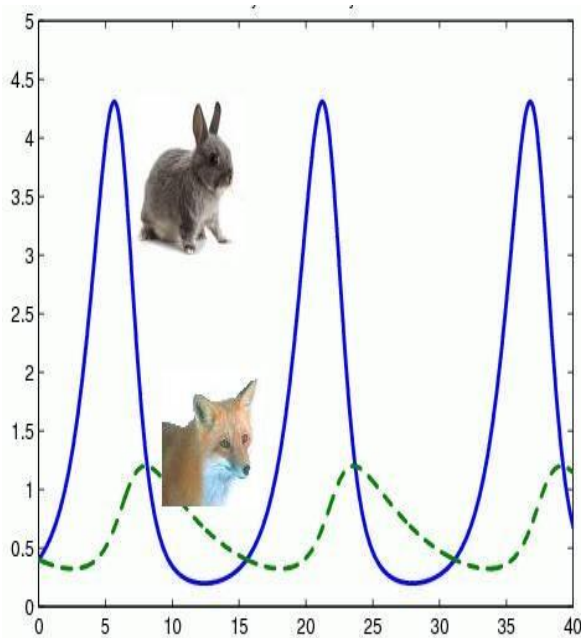


Fig.5

The number of encounters between the species is proportional to the product of their populations. Each encounter tends to increase y and decrease x . Thus, the growth rate of x includes a term of the form bxy and that of y includes a term of the form cxy , where b and d are positive. The parameters a, b, c and d are independent of time t .

These assumptions lead to the equations:

$$\frac{dx}{dt} = ax + bxy = x(a + by)$$

$$\frac{dy}{dt} = dy + cxy = y(d + cx)$$

IV. COMPARISON OF PREDATOR AND PREY POPULATION

Comparison of Predator and prey population is shown in Fig.6

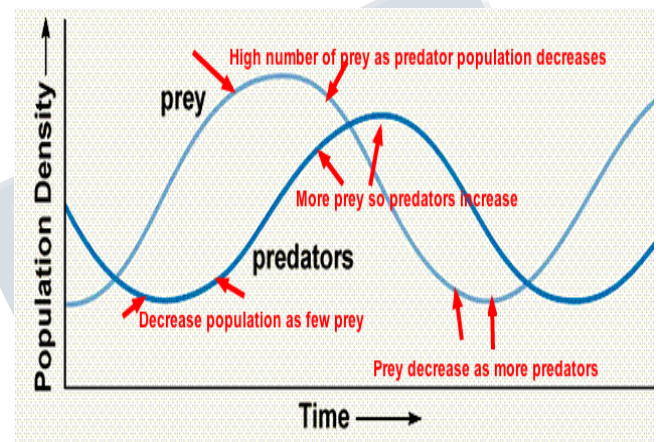


Fig.6

V. THE ACURACY OF THE MODEL

Hopefully we now have a little insight to the thinking that was behind the creation of the Volterra-Lotka model for predator -prey interaction. In practice, actual field studies of this types of biological systems show that the Volterra-Lotka model is a very good predictor of what actually occurs.

One early piece of research involved an analysis of the inventory of animal pelts purchased from fur trappers by the Hudson Bay Company over a lengthy period of time. Specifically, some of the pelts that were being purchased fell into a tidy predator-prey grouping, namely silver foxes and snow-shoe hares.

If the assumption was made that the pelts being sold formed a representative sample of the total size of each population, then the company's extensive records could be used to from a long-term profile of the sizes of both the

predator and prey populations. When this was done and compared with the predictions made by the Volterra-Lotka, the correlation was surprisingly good.

VI. APPLICATIONS OF THE PREDATOR- PREY MODEL

One of the most interesting application of systems of differential equations is the predator-prey problem. In this laboratory we will consider an environment containing two related populations: a prey population, such as rabbits and predator population such as foxes. Clearly it reasonable to expect that two populations react in such a way as to influence each other's size.

VII. CONCLUSION

This Lotka-Volterra Predator-Prey Model is an indispensable model of the complex ecology of this world. It espouses just one prey for the predator and vice versa. It also advocates no outside influences like diseases, changing conditions, pollution, and so on. However, the model can be extended to include other variables. we can polish the equations by adding additional variables and procure a better picture of the ecology. But with additional variables, the model becomes more intricate and entails additional intelligence or computer resources. It also exhibits an exceptional association between biology and mathematics.

VIII. REFERENCES

1. Dr.Frank Hoppenstedt, Courant Institute Of Mathematical Sciences, NYU, Newyork, NY.
2. The text book of predator prey model, University of Tuebingen, Germany.
3. Int.J.Contemp.Math.Science, Vol 7, 2012, No 10489-488.
4. Marting and S.Ruan, Prey Predator models with time delay and prey harvesting.J.Math.Biol 43(2001), 247-287.

therefore that the body will interpret this as an extreme emergency and thereafter the hormones epinephrine and glucagon come in play. Epinephrine is secreted by the adrenal medulla in response to acute stress (fight or flight response), Duff and Jason. Important effects of epinephrine, some of which are highlighted in the appendix, include;

- (a) Increased glucose production from glycogen breakdown
- (b) Increased glucose production from lactate and amino acids
- (c) Increased fat mobilization by stimulation of hormone sensitive lipase
- (d) Small net stimulation of insulin secretion from pancreatic β -cells.

III. BLOOD GLUCOSE REGULATORY SYSTEM

diagnosed by means of a glucose tolerance test (GTT). Today, there are over 20 million diabetics in America, six million of whom must take injections of insulin daily, Reporter. It was established by the Kenya Diabetes Management and Information Centre during the free diabetes screening exercise at M. P. Shah hospital, Nairobi, Kenya that 3.3 million Kenyans suffer from diabetes, Okwemba. Cases of diabetes in the country have increased from 3.5 to 10 per cent of the population in the past one year. Every 2 years 921 new cases are diagnosed in various clinics in Nairobi, Coast, Central, Nyanza, Eastern and Rift Valley provinces. Diabetic patients require supplement of insulin in the form of regular injections and tablets in addition to modified diet to regulate glucose input, Krimmel et al. The normal blood glucose concentration level in humans is in a narrow range (70–110 mg/dl). Exogenous factors that affect the blood glucose concentration level include food intake, rate of digestion, exercise, reproductive state.

II. MATHEMATICAL MODELLING

Glucose plays an important role in the food metabolism of any vertebrate tissue since it is a source of energy for all tissues and organs, Middleman. The majority of

mathematical models were devoted to the dynamics of glucose-insulin, including Intra Venous Glucose Tolerance Test (IVGTT), Oral Glucose Tolerance Test, (OGTT) and Frequently Sampled Intra Venous Glucose Tolerance Test (FSIVGTT). It is quite conceivable,

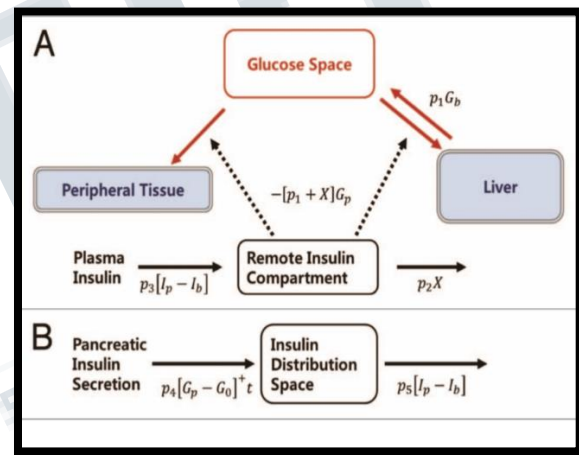


Fig.1

To understand the glucose homeostasis mechanism in the human body, many researchers have studied various mathematical models for the blood glucose regulation dynamics in the human whole-body system. With the help of those models, it has been reproduced that the plasma glucose concentration returns to its normal level from the hyperglycaemic state during the intravenous glucose tolerance test (IVGTT). In particular, since the study of the glucose insulin feedback action of the whole-body glucose regulation system by means of a simple model, a number of artificial models have been developed for understanding the glucose homeostasis in the human body. In the simple model by Bolie, plasma insulin increases due to production in the pancreas and exogenous insulin infusion, whereas plasma glucose diminishes due to various glucose consuming organs such as the liver and peripheral tissues.

If one's glucose concentration level is constantly out of the range (70–110 mg/dl), this person is considered to have blood glucose problems known as hyperglycaemia or hypoglycaemia. Diabetes mellitus is a disease of the glucose-insulin regulatory system hyperglycaemia. (see fig.1)

IV. GLUCOSE TOLERANCE TEST

The glucose tolerance test is a medical test in which glucose is given and blood samples taken afterward to determine how quickly it is cleared from the blood. The test is usually used to test for diabetes, insulin resistance, impaired beta cell function, and sometimes reactive hypoglycaemia and acromegaly, or rarer disorders of carbohydrate metabolism. In the most commonly performed version of the test, an oral glucose tolerance test (OGTT), a standard dose of glucose is ingested by mouth and blood levels are checked two hours later. Many variations of the GTT have been devised over the years for various purposes, with different standard doses of glucose, different routes of administration, different intervals and durations of sampling, and various substances measured in addition to blood glucose. (see Fig.2)

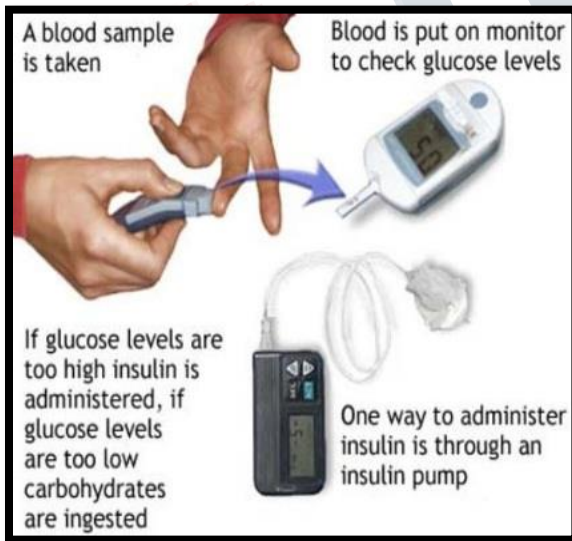


Fig.2

V. ORDINARY DIFFERENTIAL EQUATION

In mathematics, an ordinary differential equation (ODE) is a differential equation containing one or more functions of one independent variable and its derivatives. The term ordinary is used in contrast with the term partial

differential equation which may be with respect to more than one independent variable.

ODEs that are linear differential equations have exact closed-form solutions that can be added and multiplied by coefficients. By contrast, ODEs that lack additive solutions are nonlinear, and solving them is far more intricate, as one can rarely represent them by elementary functions in closed form: Instead, exact and analytic solutions of ODEs are in series or integral form. Graphical and numerical methods, applied by hand or by computer, may approximate solutions of ODEs and perhaps yield useful information, often sufficing in the absence of exact, analytic solutions.

VI. PARTIAL DIFFERENTIAL EQUATIONS

In mathematics, a partial differential equation (PDE) is a differential equation that contains unknown multivariable functions and their partial derivatives. (A special case are ordinary differential equations (ODEs), which deal with functions of a single variable and their derivatives.) PDEs are used to formulate problems involving functions of several variables, and are either solved by hand, or used to create a relevant computer model.

PDEs can be used to describe a wide variety of phenomena such as sound, heat, electrostatics, electrodynamics, fluid dynamics, elasticity, or quantum mechanics. These seemingly distinct physical phenomena can be formalised similarly in terms of PDEs. Just as ordinary differential equations often model one-dimensional dynamical systems, partial differential equations often model multidimensional systems. PDEs find their generalisation in stochastic partial differential equations.

VII. MATHEMATICAL PART

Provided there is no recent digestion, glucose and insulin concentration will be in equilibrium. If g is taken to be excess glucose concentration and h is excess insulin concentration at time t , then at equilibrium, $g = h = 0$; positive value of g or h corresponds to concentrations greater than the equilibrium values while negative values corresponds to concentrations less than equilibrium values. If h or g is a non-zero value then the body tries to restore the equilibrium. It is assumed that the rate of change of Type equation here. these quantities depend only on the values of g and h . If there is an internal rate at which the blood glucose concentration is being increased,

epinephrine is included as a separate variable in this model of blood glucose regulatory system. Thus, if it is assumed that there is no recent digestion, the following systems of differential equations results, Ackerman et al;
 $g = -ag -bh +fe$

$$h = cg -dh +ke$$

$$e = -lg -mh +ne$$

where e represents epinephrine.

Thus, a, b, c, d, f, k, l, m and n are constants.

From the model in equation

$$\frac{dq}{dt} = -ag -bh + fe \dots\dots\dots(1)$$

Differentiating (1) with respect to t,

$$\frac{d^2q}{dt^2} = -a \frac{dg}{dt} -b \frac{dh}{dt} + f \frac{de}{dt} \dots\dots\dots(2)$$

Substituting for $\frac{dh}{dt} = cg -dh + ke \dots\dots\dots(3)$ and

$$\frac{de}{dt} = -lg -mh + ne \dots\dots\dots(4)$$
 in (1)

$$\frac{d^2q}{dt^2} + a \frac{dg}{dt} + (bc + fl)g + (bk - fn)e + (fm - bd)h = 0 \dots\dots\dots(5)$$

From equation (1) and assuming $h=0$

$$e = \frac{1}{f} \left(\frac{dg}{dt} + ag \right) \dots\dots\dots(6)$$

Substituting (6) in (5)

$$\frac{d^2q}{dt^2} + a \frac{dg}{dt} + (bc + fl)g + (bk - fn) \left(\frac{1}{f} \left[\frac{dg}{dt} + ag \right] \right) + (fm - bd) \times 0 = 0$$

$$\frac{d^2q}{dt^2} + \left(\frac{bk}{f} + a - n \right) \frac{dg}{dt} + \left(bc + fl + \frac{bka}{f} - na \right) g = 0 \dots\dots\dots(7)$$

The above expression is of the form,

$$\frac{d^2q}{dt^2} + 2\alpha \frac{dg}{dt} + \omega_0^2 g = 0 \dots\dots\dots(8)$$

Where $\alpha = \frac{bk}{f} + a - n$ and $\omega_0^2 = bc + fl + \frac{bka}{f} - na$

where the value of ω_0 , which is the system natural frequency is the basic descriptor of the response to a GTT. The model certainly conforms to reality in predicting that the blood glucose concentration tends to return eventually to its optimal concentration (1). It is assumed that $\alpha^2 - \omega_0^2 < 0$ is negative, so $\alpha^2 - \omega_0^2 < 0$. This means that characteristic equation of (2) has complex roots. If $\alpha^2 - \omega_0^2 > 0$, then $g(t)$ drops very rapidly from a fairly high value to negative ones below the equilibrium value. The body will interpret this as an extreme emergency and large amounts of epinephrine will be secreted.

VIII. APPLICATION

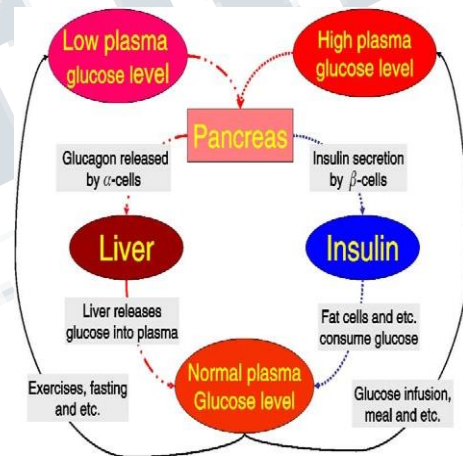


Fig.3

If one's glucose concentration level is constantly out of the range (70–110 mg/dl), this person is considered to have blood glucose problems known as hyperglycaemia or hypoglycaemia. Diabetes mellitus is a disease of the glucose-insulin regulatory system [1,3] which is referred to as hyperglycaemia.

(See Fig.3 for plasma glucose-insulin interaction loops.) Diabetes is classified into two main categories: Type 1 diabetes, juvenile onset and insulin-dependent, and type 2 diabetes, adult onset and insulin-independent. The relative interaction and contribution in the pathogenesis of this disease of various defects of the glucose-insulin regulatory system associated for example with β-cells

mass, the responsiveness level of β -cells to glucose and the sensitivity of tissues to insulin, remains to be clarified [2,4]. Complications of the disease include retinopathy, nephropathy, peripheral neuropathy and blindness [7,8]. There are many diabetic patients in the world and diabetes mellitus is becoming one of the worst diseases with respect to the size of the affected population. This motivates many researchers to study the glucose-insulin endocrine regulatory system.

Various in-vivo and in-vitro experiments have shown that the insulin secretion rate (ISR) from pancreatic islets, oscillates in a number of different time scales: The fastest oscillations have a period of tens of seconds and they have been shown to be in phase with oscillations in the free Ca^{2+} concentration of β -cells; the second fast or rapid oscillations have a period of 5–15 minutes and the slow oscillations referred to usually as ultradian oscillations, have a period within the range of 50–120 minutes [5,6,7]. In addition to these types of oscillations, circadian rhythms have been also observed (cf. [10], originally Peschke and Peschke (1998) [5]).

The rapid oscillations are caused by coordinate periodic secretory insulin bursting from the β -cells. These bursts are the dominant mechanism of insulin release at basal states [5,7]. According to Bertram et al. (2004) [1,7], in some cases compound bursting occurs, the term referred to episodic bursts clustered together and they propose that the compound bursting is responsible for insulin oscillations with a period of approximately 5 minutes. The ultradian oscillations of insulin concentration are associated to similar oscillations of the plasma glucose concentration, and they are best seen after meal ingestion, oral glucose intake, continuous enteral nutrition or intravenous glucose infusion [3,5].

Many mathematical models have been developed to better understand the mechanisms of the glucose insulin regulatory system. The most noticeable model is the so-called “minimal model” which contains minimal number of parameters [12,14] and it is widely used in physiological research work to estimate glucose effectiveness (SG) and insulin sensitivity (SI) from intravenous glucose tolerance test (IVGTT) data by sampling over certain periods. The IVGTT focuses on the metabolism of glucose in a short time period starting from the infusion of a big bolus (0.33 g/kg) of glucose at time $t = 0$. Models addressing insulin secretion oscillations include these presented in the papers [4,5,8,9]. A few models are based on the control through meals and

exercise (cf. [8,9]). See also a review paper by Mari [5] for a classification of models.

Types of models which have been used in the literature can be classified mathematically as: ordinary differential equations (ODEs), delay differential equations (DDEs), partial differential equations (PDES), Fredholm integral equations (FIES) (in the estimation of parameters problem), stochastic differential equations (SDEs) and integro-differential equations (IDEs). Different software packages can be used for different types of models for numerical analysis and simulations.

IX. CONCLUSION

This paper presents a model for detecting diabetes Mellitus in the blood described by equation. Epinephrine has been successfully incorporated as a third variable in this model of blood glucose regulatory system (BGRS). The importance of this third variable lies in its ability to help in conducting a reliable test for detecting diabetes in the blood. This leads to a system of linear homogenous equations, which are expressed in the form $Y = AY$ and whose solution provides the blood glucose concentrations for diabetics and non-diabetics. This model has been found to be asymptotically stable since the eigenvalues of the coefficient matrix are complex numbers with negative real parts. Furthermore, the resonance period for this model which is $T_0 = 2.9847134$ hrs, is far less than $T_0 = 3.5232581$ hrs for the existing model. This shows that the glucose concentration returns to normal level within a shorter time. It is worth noting that the model developed in this study only considered an internal rate at which the blood glucose concentration is being increased. Future research may take into consideration an external rate at which the blood glucose concentration is being increased. The model predicts that oscillations occur if there is sufficient diffusion (values of a scaled diffusion parameter ($\delta > 0.1$)) to create adequate concentrations mixing in the reacting layers of the cells. With insufficient such mixing, the oscillations are inhibited. An ‘unsolved dilemma’ having to do with difficulty to produce large enough δ values ($\delta > 0.1$) from experimental values of the scaling parameters V , L_{bed} , where L_{bed} is the length of the islet bed and V is the velocity of the steady flow of the solution along the 1-dimensional reactor, and large physical diffusion (large DI coefficient) which is needed for the model to predict oscillations, is mentioned at the end of the paper. The software package AUTO97 was used for the simulations

The process of obesity could be described by the whole-body glucose regulation model. Accordingly, with the

shortcomings improved, the approach based on modelling is very promising and expected to be beneficial to diabetic patients.

REFERENCE

- [1] J. Li, Y. Kuang, C. Mason, Modelling the glucose-insulin regulatory system and ultradian insulin secretory oscillations with two-time delays, submitted for publication.
- [2] J. Li, Y. Kuang, B. Li, Analysis of IVGTT glucose-insulin interaction models with time delay, *Discrete Contin. Dyn. Syst. Ser. B* 1 (1) (2001) 103–124.
- [3] L.W. Maki, J. Keizer, Mathematical analysis of a proposed mechanism for oscillatory insulin secretion in perfused HIT-15 cells, *Bull. Math. Biol.* 57 (1995) 569–591.
- [4] Mari, Mathematical modeling in glucose metabolism and insulin secretion, *Curr. Opin. Clin. Nutr. Metab. Care* 5 (2002) 495–501.
- [5] Mukhopadhyay, A. De Gaetano, O. Arino, Modeling the intra-venous glucose tolerance test: A global study for a single-distributed-delay model, *Discrete Contin. Dyn. Syst. Ser. B* 4 (2) (2004) 407–417.
- [6] G. Nucci, C. Cobelli, Models of subcutaneous insulin kinetics. A critical review, *Comput. Methods Programs Biomedicine* 62 (2000) 249–257.
- [7] G. Pacini, R.N. Bergman, MINMOD: A computer program to calculate insulin sensitivity and pancreatic responsivity from the frequently sampled intravenous glucose tolerance test, *Comput. Methods Programs Biomedicine* 23 (1986) 113–122.
- [8] R.S. Parker, F.J. Doyle III, N.A. Peppas, The intravenous route to blood glucose control, *IEEE Engrg. Medicine Biol.* 20 (2001) 65–73.
- [9] E. Peschke, D. Peschke, Evidence for a circadian rhythm of insulin release from perfused rat pancreatic islets, *Diabetologia* 41 (1998) 1085–1092.
- [10] G. Pilonetto, G. Sparacino, C. Cobelli, Numerical non-identifiability regions of the minimal model of glucose kinetics: Superiority of Bayesian estimation, *Math. Biosci.* 184 (2003) 53–67.
- [11] N. Pørksen, M. Hollingdal, C. Juhl, P. Butler, J.D. Veldhuis, O. Schmitz, Pulsatile insulin secretion: Detection, regulation, and role in diabetes, *Diabetes* 51 (Suppl. 1) (2002) S245–S254.
- [12] W.R. Puckett, E.N. Lightfoot, A model for multiple subcutaneous insulin injections developed from individual diabetic patient data, *Amer. J. Physiol.* 269 (1995) E1115–E1124, or *Endocrinol. Metab.* 32 (1995) E1115–E1124.
- [13] E. Ruiz-Velázquez, R. Femat, D.U. Campos-Delgado, Blood glucose control for type I diabetes mellitus: A robust tracking H_∞ problem, *Control Engrg. Practice* 12 (2004) 1179–1195.
- [14] Simon, G. Brandenberger, Ultradian oscillations of insulin secretion in humans, *Diabetes* 51 (Suppl. 1) (2002) S258–S261.