# A Mathematical Model for Recovery of Myocardial Infarction Due Diabetes

A.A Khajehnasiri, N. Khajehnasiri, S. Shahmorad

Department of mathematics, payame noor University of Tabriz, Tabriz- Iran
Department of natural sciences, University of Tabriz, Tabriz- Iran
Department of applied mathematics, University of Tabriz, Tabriz- Iran

E-mail:a.khajehnasiri@gmail.com

E-mail: Naz_khajeh89@ms.tabrizu.ac.ir

E-mail:shahmord@tabrizu.ac.ir

**Abstract**

The numbers of diabetic patients are increased very fast in over the world. Diabetes causes myocardial infarction which it is very important inadequacy of heart. In the present paper, solving an ordinary differential equations (ODE) are used to find the number of individuals with myocardial infarction and recovery of that.

Keyword**:** Ordinary Differential Equations (ODE), Diabetes, Myocardial infarction.

## 1 Introduction:

Diabetes mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM exist and are caused by a complex interaction of genetics and environmental factors. Depending on the etiology of the DM, factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization, and increased glucose production [9].

[*] **AMO - Advanced Modeling and Optimization. ISSN: 1841-4311**

In general, two forms of diabetes are considered: 1) Type I diabetes or Insulin Dependent Diabetes Mellitus (IDDM), which due to hack insulin secretion [10]. 2) Type II diabetes or Non-Insulin Dependent Diabetes Mellitus (NIDDM) [10]. Type 2 DM is a heterogeneous group of disorders characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production [9].

Two features of the current classification of DM diverge from previous classifications. First, the terms insulin-dependent diabetes mellitus (IDDM) and noninsulin-dependent diabetes mellitus (NIDDM) are obsolete. Since many individuals with type 2 DM eventually require insulin treatment for control of glycemia, the use of the term NIDDM generated considerable confusion. A second difference is that age is not a criterion in the classification system. Although type 1 DM most commonly develops before the age of 30, an autoimmune beta cell destructive process can develop at any age. It is estimated that between 5% and 10% of individuals who develop DM after age 30 have type 1 DM. Likewise, type 2 DM more typically develops with increasing age but is now being diagnosed more frequently in children and young adults, particularly in obese adolescents [ 9].

The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system [9].

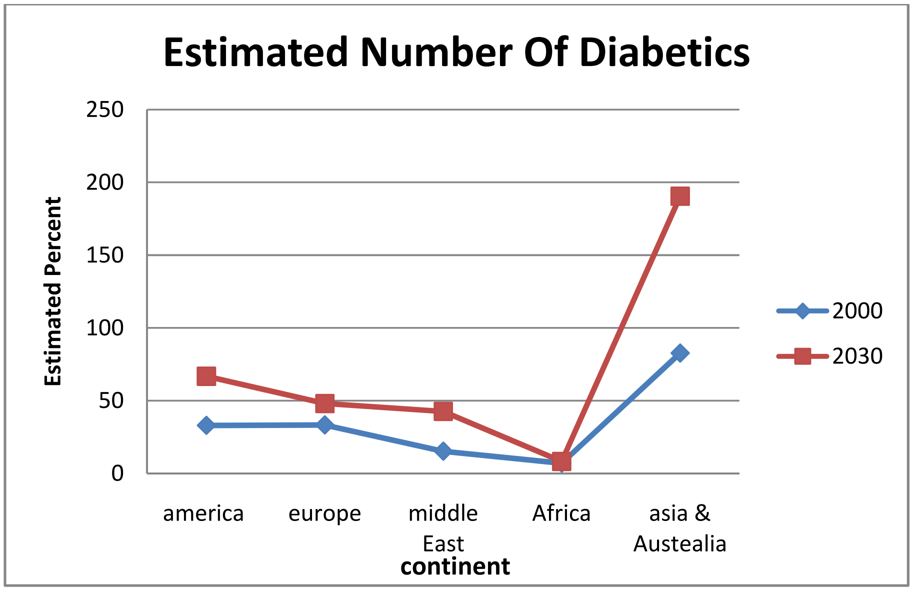
**Complications of Diabetes**

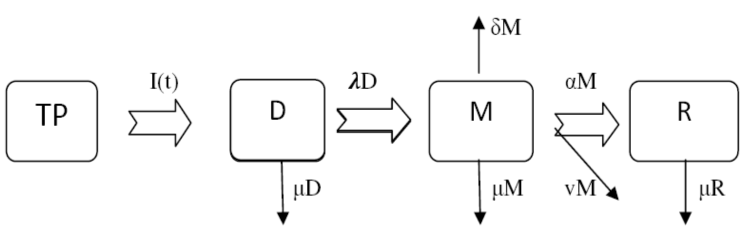
Diabetes have a lot of complications such as**:**

- Bladder Control Problems for Women
- Diabetes, Heart Disease and Stroke
- Diabetic Neuropathies: The Nerve Damage of Diabetes
- Diabetic Retinopathy (Eye Disease)
- Erectile Dysfunction
- Erection Problems
- Feet Can Last a Lifetime kit (PDF, 387 KB)
- Hypoglycemia (Low Blood Glucose)
- Kidney Disease of Diabetes
- Kidney Failure: Choosing a Treatment That’s Right for You
- Make the Kidney Connection
- Sexual and Urologic Problems of Diabetes
- Stomach Nerve Damage (Gastro paresis)

Immediately after an acute coronary occlusion, blood flow ceases in coronary vessels beyond the occlusion except for small amount of collateral flow form surrounding vessels. The area of muscle that has either zero flow or so little flow that it cannot sustain cardiac muscle function is said to infarcted. The overall process is called a myocardial infarction. We must mention it if there is even as much as 15 to 30 per cent of normal resting coronary blood flows, the muscle will not die. However blood flow is less than this, so that this muscle does die [10]. The four major causes of death following acute myocardial infarction are decreased cardiac output; damming of blood in the pulmonary or systemic veins with death resulting from edema, especially pulmonary edema; fibrillation of the heart; and occasionally, rupture of the heart. But myocardial infarction after a few months can be recovers as following: after the occlusion, the muscle fibers die in the very center of ischemic area. Then during the ensuring days, this area of dead fibers grows because of the marginal fibers finally succumb to the prolonged ischemia. At the same time, owing to the enlargement of the collateral arterial channels growing into the outer rim of the infarcted area, the nonfunctional area of muscle become smaller and smaller. After a few day to 3 weeks, most of the nonfunctional area of muscle become functional again or dead one or the other. In the meantime, fibrous tissue begins developing among the dead fibers, for ischemia stimulates growth of fibroblasts and promotes development of greater than normal quantities of fibrous tissue. Therefore, the dead muscular tissue is gradually replaced by fibrous tissue then, because it is a general property of fibrous tissue to undergo progressive electrometric and dissolution, the size of the fibrous scar may became smaller over a period of several months a year [10]. During the last decades, a huge number of papers were published on different aspects of diabetes and its complications. In particular, an interesting literature has been devoted to studies collecting, analyzing and validating data concerning diabetes populations. A variety of mathematical models, statistical methods and computer algorithms have been proposed in order to understand different aspects of diabetes such as: glucose-insulin dynamics, epidemiology of diabetes and its complications, cost of diabetes and cost-effectiveness of strategies dealing with diabetes. The numbers of diabetic patients are increased very fast in over the world, because of that in this paper we try to model the recovery from myocardial infarction on diabetic patients.

diagram1: Estimated number of diabetes




Figurer 1: The mathematical model

## 2 Method

Suppose that R=R (t), M=M (t) and D=D (t) represent the number of recovery individual from myocardial infarction, myocardial infarction individual and diabetic individual, respectively, and let N=N (t) =R (t) +M (t) +D (t) denote the size of the population of diabetics at time *t,* model parameters to be incorporated are μ (the natural mortality rate), λ (the probability of a diabetic person have myocardial infarction), ν (the rate at which diabetic patients with myocardial infarction become severely disabled) and δ (the Mortality rate due to myocardial infarction). α (the rate at which diabetic patient with myocardial infarction who recovered).

A schematic representation of the model is shown in Figure1.

The diagram shows that $I = I(t)$ cases are diagnosed in a time interval of length $t$ and are assumed to have no myocardial infarction upon diagnosis. In that same time interval, the number of sufferers without myocardial infarction, TP is total population, D = $D(t)$, is seen to decrease by the amounts $\mu D$ (natural mortality) and $\lambda D$ (sufferers who have myocardial infarction). During this time interval, the number of diabetics with myocardial infarction is increased by the amount $\lambda D$ (the probability of a diabetic person who have myocardial infarction, and decrease by $\nu M$ (patients who become severely disabled and whose disabilities cannot be cured) and $\delta M$ (those who die from their myocardial infarction)and μM (the natural mortality rate), and αM (the rate at which diabetic patient with myocardial infarction who recovered) also the number of myocardial infarction patients who recovery increase by αM (the rate at which diabetic patient with myocardial infarction who recovered), and decrease by $\mu R$ (natural mortality).

These rates of change are formalized by the ordinary differential equations (ODEs).

$$D'(t) = \frac{dD(t)}{dt} = I - \lambda D(t) - \mu D(t) = I - (\lambda + \mu)D(t)$$

$$M'(t) = \frac{dM(t)}{dt} = \lambda D(t) - \delta M(t) - \alpha M(t) - v M(t) - \mu M(t)$$
$$= \lambda D(t) - (\delta + \alpha + v + \mu)M(t) = \lambda D(t) - \theta M(t)$$

$$\acute{R}(t) = \frac{dR(t)}{dt} = \alpha M(t) - \mu R(t)$$

With initial conditions {M (0) = $M_0$, R (0) = $R_0$, N (0) = $N_0$} since $N(t)$ = R (t) + M (t) +$D(t)$, give rise to the initial value problem (IVP).

$$\acute{D} = I - (\lambda + \mu)N(t) + (\lambda + \mu)R(t) + (\lambda + \mu)M(t)$$

$$\acute{M} = \lambda N(t) - \lambda R(t) - (\lambda + \theta)M(t) \qquad t > 0 \ , \ M(0) = M_0 \qquad (1)$$

$$\acute{R} = \alpha M(t) - \mu R(t) \qquad t > 0 \ , \quad R(0) = R_0 \qquad (2)$$

$$N' = I - \mu N(t) - (\delta + v)M(t) \qquad t > 0 \ , \ N(0) = N_0 \qquad (3)$$

Where $\theta = \alpha + \mu + \nu + \delta$, $N'(t) = \frac{dN(t)}{dt}$ and $N_0$ , $R_0$ are the initial values of $R(t)$ and $N(t)$, respectively.

The model equations (1), (2), (3) are linear in N and R, and θ is a constant.

**Critical point and eigenvalues**

The probability of developing a myocardial infarction, α, will be estimated to have the constant value:

$$\alpha = \frac{R_0}{N_0} \tag{4}$$

The initial-value problem (1), (2), (3) may consequently be written in matrix-vector form as:

$$J'(t) = AJ(t) + b(t) \qquad t > 0\,, J(0) = J_0 \tag{5}$$

$$J(t) = \begin{bmatrix} M(t) \\ R(t) \\ N(t) \end{bmatrix}, \qquad A = \begin{bmatrix} -(\lambda+\theta) & -\lambda & \lambda \\ \alpha & -\mu & 0 \\ -(\delta+\nu) & 0 & -\mu \end{bmatrix}$$

In which,

$$b(t) = \begin{bmatrix} 0 \\ 0 \\ I(t) \end{bmatrix}, \qquad J_0 = \begin{bmatrix} M_0 \\ R_0 \\ N_0 \end{bmatrix} \tag{6}$$

The fallowing equations are given from (1), (2), (3).

$$\alpha\lambda N - (\lambda\alpha + \lambda\mu + \theta\mu)R = 0 \tag{7}$$

$$\alpha I - \mu\alpha N - (\delta\mu + \nu\mu)R = 0 \tag{8}$$

Solving (7) and (8) gives:

$$R^* = \frac{\lambda\alpha I}{\mu\lambda\alpha + \mu^2\lambda + \theta\mu^2 + \lambda\delta\mu + \lambda\nu\mu}$$

And

$$N^* = \frac{(\lambda\alpha + \lambda\mu + \theta\mu)I}{\mu\lambda\alpha + \mu^2\lambda + \theta\mu^2 + \lambda\delta\mu + \lambda\nu\mu} \tag{9}$$

The eigenvalues of the matrix A, are the roots of the following equation (the characteristic equation).

$$x^3-(-2\mu-\lambda-\theta)x^2-(-\lambda\delta-\lambda\nu-2\lambda\mu-\mu^2-2\theta\mu-\alpha\lambda)x+\lambda\delta\mu+\lambda\nu\mu+\alpha\lambda\mu+\lambda\mu^2+\theta\mu^2=0$$

That is:

$$-\mu,-\frac{\mu}{2}-\frac{\lambda}{2}-\frac{\theta}{2}+\frac{\sqrt{\mu^2-2\mu\lambda-2\theta\mu+\lambda^2+2\lambda\theta+\theta^2-4\lambda\delta-4\lambda\nu-4\alpha\lambda}}{2},-\frac{\mu}{2}-\frac{\lambda}{2}-\frac{\theta}{2}-\frac{\sqrt{\mu^2-2\mu\lambda-2\theta\mu+\lambda^2+2\lambda\theta+\theta^2-4\lambda\delta-4\lambda\nu-4\alpha\lambda}}{2}$$

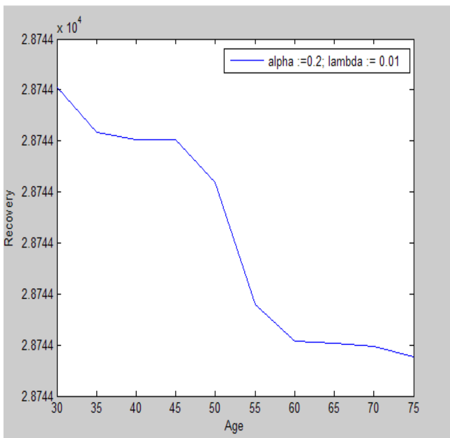
To accede purpose, the IVP (1), (2), (3) was solved by ordinary differential equations method $rk_4$.[2]
We put the following parameter values in (1), (2), (3) and then use Mat lab software to draw bellow graphs.
μ=0.7, δ=0.04, α= 0.2, v= 0.04, $\boldsymbol{\lambda}$= 0.01 or0.7

## 3 Result

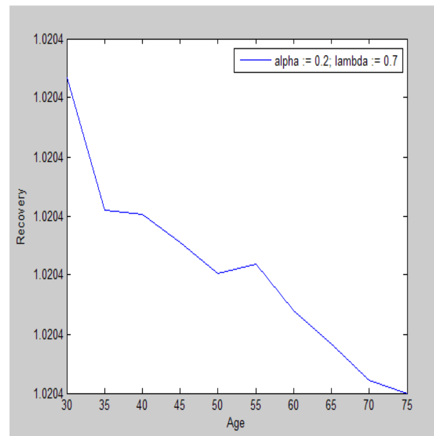
Two scenarios of diabetic patients were considered. One scenario has small myocardial infarction that means, damage area is small. In second scenario damage area is big, because they have a big myocardial infarction. All patients in both scenarios have same care. There is not any difference among the patients in each scenario, except their ages. In this paper, we show that, when the patients are old, rate of recovery decrease (figure2, A, B). Also in older patients, the rate of myocardial infarction decrease (figure 2, C, D). Because in old patients revascularization occurred and collateral vessels are formed. On the other hand, physical activity in old individuals is low, so their heart needs a little amount of oxygen and probability of ischemia is reduced. When one of the coronary vessels become gradually blocked the collateral vessels are formed. The forming of these vessels takes time. If one of the coronary vessels in old patients blocks, then these collateral vessels wrought the myocardial cells that can get oxygen. These ingredients decrease the rate of myocardial infarction. Also in old cases the regeneration of all tissues is low especially in heart,

[2] Four stages Ronge kotta

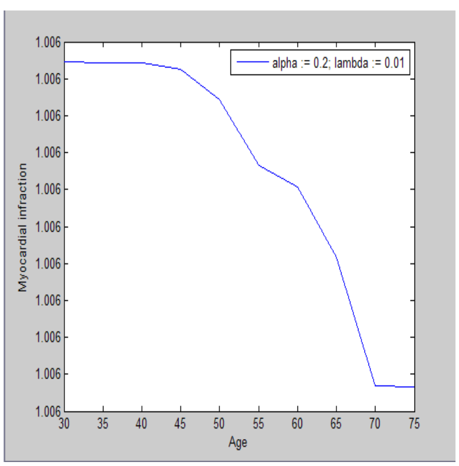
therefore the rate of recovery is reduced. Conversely in young patient rate of big myocardial infarction more than small myocardial infarction (figure 2C, D).



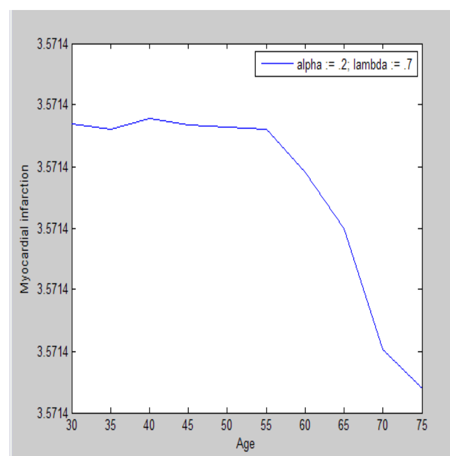

A




B




C




D

Figure2: These graphs from A to D compare the rate of myocardial infarction and recovery of it, in different age group of Diabetics patients.

## References:

[1] Makroglou, A. and J.Li and Y. Kuang, (2006) *Mathematical models and software tools for the glucose-insulin regulatory system and diabetes: an overview*. Applied Numerical Mathematics, 56, pp. 559-573.

[2] Boutayeb, A., and E.H. Twizell, (2004)*An age structured model for complications of diabetes mellitus in Morocco* Simulations Modeling Practice Theory, 12, pp. 77-87.

[3] Bergman, R.N.(2002)*Pathogenesis and prediction of diabetes mellitus, Lessons from integrative physiology*,in: Irving L. Schwartz Lecture, Mount Sinai J. Medicine 60,pp. 280-290.

[4] Metascreen Writing Committee,(2006)*The metabolic syndrome is a risk indicator of microvascular and macrovascular complications in diabetes, Results from Metascreen, a multicenter diabetes clinic-based survey*,Diabetes Care 29,pp. 2701-2708.

[5] Hermans, M.P, (2007)*Diabetic macro and microvascular disease in type 2 diabetes*, Diabetes Vasc Dis Res 4,pp.7-11.

[6] UK Prospective Diabetes Study Group,(1998)*Tight blood pressure control and risk of Macrovascular and microvascular complications in type 2 diabetes*,BMJ 317,pp. 703-713.

[7] Staines, A. and HJ. Bodanshy and H.E.B. Lill and ey, C Stephenson and R.J.Q McNally And R.A. Cartwright, (1993) *The epidemiology of diabetes mellitus in the United Kingdom*, Diabetologia 36, pp. 1282-1287.

[8] Lehmann, E.D. and T.D.eutsch, (1995) *Applications of computers in diabetes care a review*, Med Inform, 20, pp.303-329.

[9] Fauci, A.S. and D.L. Longo's, (2008) *Principles of internal medicine*.(17th edition).The McGraw-Hill Companies.

[10] Guyton, A. and J. Hall,(2006)*Medical physiology*.(11th edition).Elsevier Saunders.

[11] O. Casis and E. Echevarria, (2004) *Diabetic Cardiomyopathy: Electromechanical Cellular Alterations* Current Vascular Pharmacology, 2, 237-248.