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FITTING A GAMMA DISTRIBUTED RANDOM-EFFECTS MODEL FOR THE TYPE-2 DIABETES MELLITUS PATIENTS - A COMPARATIVE STUDY

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ABSTRACT

The aim of the present study is to reduce the morbidity and mortality by improving adherence to important recommendation for preventing, detecting, and managing diabetes patients with complication. This study determines the comparison of TYPE 2 Diabetes Mellitus (T2DM) patients of two regions and analyzed their prevalence of risk factors. A gamma distributed random- effects model is fitted and the estimates, standard error and Wald's 95% confidence limits of the parameters are obtained using SAS. Also the LR statistics for each parameter of the two regions are obtained to study the significance of the risk factors and derived the findings.

Key words: Diabetes Mellitus (DM), Risk factor, Gamma distribution, LR statistics.

INTRODUCTION

Diabetes Mellitus (DM) is a metabolic disorder resulting from deficiency/absence of insulin either due to secretion or action or both. Insulin deficiency in turn leads to hyperglycemia with disturbances of carbohydrate, fat and protein metabolism [1-3]. As the disease progresses it causes damage to tissues of various systems. Thus, it affects all the major systems of our body namely cardiovascular, nerves and renal system leading to many complications. So, it is a multi system disorder though the defect is only in insulin.

Type 2 diabetes, (formerly known as Non – Insulin- Dependent Diabetes (NIDDM)) accounts for most of the cases of DM worldwide. It is estimated that in 2000 there were approximately 150 million individuals with the disease and that this number is likely to double by 2025 [4]. Type 2 diabetes is the fourth of five leading causes of death in most developed countries and there is growing evidence that it has reached epidemic proportions in many developing and newly industrialized countries [5]. Diabetes is the condition in which the body does not properly process food for use as energy. Most of the food we eat is turned into glucose, or sugar by our system to get energy the pancreas, an organ that lies near the stomach, secretes a hormone called insulin to help glucose get into the cells of our bodies. When we have diabetes, our body either doesn't make enough insulin or can't use its own insulin as well as it should. This causes raise in blood sugar level. Even though the level of glucose in the blood is high, body cannot use it without the help of insulin.

There are two main types of diabetes. Type 1 DM, totally insulin dependent and has early onset. T2DM is late onset and due to either deficiency or insulin resistance. Type 2 diabetes is a complex, slowly progressive silent killer disease. This destroys multiple organs by damaging and clogging the small capillaries – micro vascular system. Thus, it leads to the damage of heart, brain, feet, kidneys and eyes, commonly refer to as Neuropathy, Nephropathy, Retinopathy all due to mainly Micro vascular Angiopathy.

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As the disease progresses, the majority of diabetics develop two or more of the following complications:

- ✓ Additional weight (fat) gain, leading to low energy and fatigue, obesity, high blood pressure, dehydration, high cholesterol, arthritis, toxicity and other acidic, inflammatory ailments.
- ✓ Kidney disease (nephropathy), leading to kidney failure and dialysis.
- ✓ Eye disease (retinopathy), leading to blindness.
- ✓ Nerve damage (neuropathy), leading to amputation of a lower limb; and other nerve-related conditions.
- ✓ Gum (periodontal) disease, leading to the loss of teeth and other infections.
- ✓ Heart disease (due to high blood pressure, high homocysteine, thick blood), leading to a heart attack or stroke.
- ✓ A higher susceptibility to other infections and diseases due to an underlying mechanism of internal inflammation and a weakened immune system.

Under normal circumstances, when we eat food, it is broken down and converted to glucose, and the glucose level in blood begins to rise. This signals the pancreas to secrete insulin into bloodstream. The cells in the body, such as the fat cells and muscle cells, contain these "doors" (insulin receptors) that sense the presence of insulin. Insulin acts like a "key" and causes these "doors" to open. When these "doors" open, the glucose in blood is transported into cells and processed to provide with energy. Any extra glucose is stored as glycogen in liver and muscle cells for future use (e.g. exercise). At this point, the glucose level in blood lowers and returns to normal, usually within 2 hours after eating.

But, in the case of diabetes, the "doors" do not respond to the "key" insulin and do not open and let in the glucose. Although some of the glucose is stored as glycogen by the liver and muscle cells, the majority of the glucose begins to "back up" in the blood causing the blood glucose level to continue to rise. The pancreas senses that the blood glucose level is still rising, so the pancreas ramps up and secretes more and more insulin to try to "push" the glucose into the cells and bring the glucose level down.

As the glucose level continues to rise, the body is unable to store glucose and become resistant to insulin. The excess glucose is converted into fat and deposited at various sites of the body. This leads to obesity, atherosclerosis etc., At the same time, when the blood sugar level increases beyond the renal threshold value, it starts excreting glucose. By osmotic effect, naturally water is excreted resulting in poly urea. This again leads to excess thirst and increase appetite – polydipsia and polyphagia. The excess fat cells release chemicals called cytokines that block the insulin receptors, leading the pancreas to churn out two to three times more insulin. After years of high insulin levels, due to over stimulation, insulin secreting beta cells burn out. This, in turn, causes insulin levels to fall, leading to a further rise in the glucose level. This vicious cycle goes on and finally ends in organ shut down and hyperglycemic coma.

As depicted in the following diagram, the lack of response from the cell receptors ("doors") to open up and let in the glucose leads to low energy, fatigue and an increased resistance to insulin. However, diabetes is more than "insulin resistance". Ongoing diabetes leads to an increase of oxidation (free radical damage), inflammation (tissue damage), and toxicity (poisoning and acidity).

Symptoms of diabetes

- ✓ Frequent urination
- ✓ Excessive thirst
- ✓ Unexplained weight loss
- ✓ Extreme hunger
- ✓ Sudden vision changes
- ✓ Feeling very tired much of the time
- ✓ Very dry skin
- ✓ Sores that are slow to heal

Risk factor for Type 2 DM

The major risk factor for DM type 2 could be categorized as mentioned below

1. Non-Modifiable risk factor
2. Modifiable risk factors

Non- Modifiable risk factors

- ✓ Age
- ✓ Sex
- ✓ Genetic factors
- ✓ Family history
- ✓ Gestational diabetes

Modifiable risk factors

- ✓ Obesity
- ✓ Life style changes
- ✓ Eating habits
- ✓ High saturated fat intake
- ✓ Lack of fiber
- ✓ Alcohol
- ✓ Stress
- ✓ Other factor – Marital status, socio economic etc.,

Diagnosis of diabetes

DM is confirmed by estimating the fasting and post-prandial blood and urine glucose supported by the levels of HbA1c.

MATERIALS AND METHODS

For this study two independent samples of each from 450 patients were observed from two regions such as Region 1 (Chennai) and Region 2 (Kumbakonam) in Tamilnadu. These data were collected from those who received treatment for Type 2 diabetes with their clinical measurements such as FBS, BMI, SBP, TCHO, HDL,

LDL, TGL and creatinine from the patients visit the clinical centre. These data were analyzed using SAS software.

Life data are very much appropriate for modeling with gamma distribution. PROC GENMOD in SAS can be used for modeling complete data with the gamma distribution and it provides a statistical test for the exponential distribution against gamma distribution alternatives [6,7] for applications of the gamma distribution to life data.

The Type 3 analysis in GENMOD Procedure, contrast is provided for each effect specified in the MODEL statement. The default analysis is to compare likelihood ratio statistics for the contrasts or score statistics for GEEs. Wald statistics are computed if the WALD option is also specified.

1. GENERALIZED LINEAR MODEL

The class of generalized linear models is an extension of traditional linear models that allows the mean of a population to depend on a linear predictor through a nonlinear link function and allows the response probability distribution to be any member of an exponential family of distributions [8]. Many widely used statistical models are generalized linear models. These include classical linear models with normal errors, logistic and probit models for binary data, and log-linear models for multinomial data. Many other useful statistical models can be formulated as generalized linear models by the selection of an appropriate link function and response probability distribution.

The Statistical modeling using generalized linear models is discussed in [9]. Many examples of applications of generalized linear models are provided in [10,11]. An overview of generalized linear models is provided in [12].

Gamma distribution of random effects models

A random-effects model parameterizes the random effects according to an assumed distribution for which the parameters of the distribution are estimated. These models are called subject-specific models, since the likelihood models the individual observations instead of the marginal distribution of the panels. As in the case of conditional fixed-effects models, our derivation begins with an assumed distribution and, thus, does not address the quasi-likelihoods of GLMs.

The log-likelihood for a random-effects model is

$$\mathcal{L} = log\prod_{i=1}^{n}\int_{-\infty}^{\infty} f(\epsilon_i)\left\{\prod_{t=1}^{n_i} f_y\left(x_{it}\beta+\epsilon_i\right)\right\}d\epsilon_i \quad (1)$$

where f_y is the assumed density for the overall model (the outcome) and f is the density of the i.i.d random effects ϵ_i. The estimating equation is the derivative of the log-likelihood in terms of β and the parameters of the assumed random-effects distribution.

By inspection, obtaining the estimating equation might be a formidable task. There are cases for which an analytic solution of the integral is possible and for which the resulting estimating equation may be easily calculated. This depends on both the distribution of the outcome variable and the distribution of the random effect. There are also cases for which numeric integration techniques, e.g., quadrature formulae, may be implemented in order to calculate the estimating equation. In the following, we present an example of each of these approaches.

A random effects model may be derived assuming a gamma distribution for the random effect with Poisson setting [13, 14, 15]. This choice of distribution leads to an analytic solution of the integral in the likelihood.

For a random effects specification, we know that

$$\Pr\left(y_{i1},\ldots,y_{in_i}\middle|\alpha_i,x_{i1},\ldots,x_{in_i}\right) = \left(\prod_{t=1}^{n_i}\frac{\lambda_{it}^{y_{it}}}{y_{it}!}\right)exp\left\{-exp(\alpha_i)\sum_{t=1}^{n_i}\lambda_{it}\right\}exp\left(\alpha_i\sum_{t=1}^{n_i}y_{it}\right) \quad (2)$$

In the usual Poisson model we hypothesize that the mean of the outcome variable y is given by $\boldsymbol{\lambda}_{it}=\exp(x_{it}\beta)$. In the panel setting we assume that each panel has a different mean that is given by $\exp(x_{it}\beta)= \boldsymbol{\lambda}_{it}$. As such, we refer to the random effect as entering multiplicatively rather than additively, as is the case in random-effects linear regression.

Since the random effect $\epsilon_i=\exp(\alpha_i)$ is positive, we select a gamma distribution adding the restriction that the mean of the random effects equals one. We do this so that there is only one additional parameter θ to estimate.

$$f(\epsilon_i) = \frac{\theta^{\theta}}{\Gamma(\theta)}\epsilon_i^{\theta-1}\exp(-\theta\epsilon_i) \quad (3)$$

The conditional mean of the outcome given the random effect is Poisson, and the random effect is distributed Gamma (θ,θ). Therefore, we take the product to obtain the joint density function for the observations of a single panel given by

$$Pr\left(y_{i1},\ldots,y_{in_i}|\epsilon_i,x_{i1}\ldots,x_{in_i}\right) = \left\{\prod_{t=1}^{n_i}\frac{(\lambda_{it}\epsilon_{it})^{y_{it}}}{y_{it}!}\right\}exp\left(-\sum_{t=1}^{n_i}\lambda_{it}\epsilon_{it}\right)$$

$$= \left\{\prod_{t=1}^{n_i}\frac{\lambda_{it}^{y_{it}}}{y_{it}!}\right\}exp\left(-\epsilon_i\sum_{t=1}^{n_i}\lambda_{it}\right)\epsilon_i^{\sum_{t=1}^{n_i}y_{it}} \quad (4)$$

Moreover, since the panels are all independent, the joint density for all of the panels combined is the product of the density of each of the panels.

We now assume that ϵ_i follows a gamma distribution with mean one and variance 1/θ so that unconditional on ϵ_i

$$Pr\left(y_{i1},\ldots,y_{in_i}|X_i\right) = \frac{\theta^{\theta}}{\Gamma(\theta)}\left(\prod_{t=1}^{n_i}\frac{\lambda_{it}^{y_{it}}}{y_{it}!}\right)\int_0^{\infty}\exp\left(-\epsilon_i\sum_{t=1}^{n_i}\lambda_{it}\right)\epsilon_i^{\sum_{t=1}^{n_i}y_{it}}\epsilon_i^{\theta-1}exp(-\theta\epsilon_i)d\epsilon_i$$

$$= \frac{\theta^{\theta}}{\Gamma(\theta)}\left(\prod_{t=1}^{n_i}\frac{\lambda_{it}^{y_{it}}}{y_{it}!}\right)\int_0^{\infty}\exp\left\{-\epsilon_i\left(\theta+\sum_{t=1}^{n_i}\lambda_{it}\right)\right\}\epsilon_i^{\theta+\sum_{t=1}^{n_i}y_{it}-1}d\epsilon_i \quad (5)$$

$$= \left(\prod_{t=1}^{n_i}\frac{\lambda_{it}^{y_{it}}}{y_{it}!}\right)\frac{\Gamma\left(\theta+\sum_{t=1}^{n_i}y_{it}\right)}{\Gamma(\theta)}\left(\frac{\theta}{\theta+\sum_{t=1}^{n_i}\lambda_{it}}\right)^{\theta}\left(\frac{1}{\theta+\sum_{t=1}^{n_i}\lambda_{it}}\right)^{\sum_{t=1}^{n_i}y_{it}} \quad (6)$$

for $X_i = \left(x_{i1},\ldots,x_{in_i}\right)$.

The log likelihood (assuming gamma heterogeneity) is then derived using

$$u_i = \frac{\theta}{\theta + \sum_{t=1}^{n_i} \lambda_{it}} \qquad and \quad \lambda_{it} = exp(x_{it}\beta) \tag{7}$$

$$Pr\left(Y_{i1} = y_{i1}, \ldots, Y_{in_i} = y_{in_i} | X_i\right) = \frac{\prod_{t=1}^{n_i} \lambda_{it}^{y_{it}} \Gamma\left(\theta + \sum_{t=1}^{n_i} y_{it}\right)}{\prod_{t=1}^{n_i} y_{it}! \Gamma(\theta) \left(\sum_{t=1}^{n_i} \lambda_{it}\right)^{\sum_{t=1}^{n_i} y_{it}}} u_i^{\theta} (i - u_i)^{\sum_{t=1}^{n_i} y_{it}} \tag{8}$$

The log-likelihood for gamma distributed random effects may then be derived by integrating over ϵ_i. We note that by rearranging terms in the joint density, the integral term may be simplified to one since it is the integral of another gamma random variable. After simplification and collection of terms, we substitute our preferred $\boldsymbol{\lambda}_i$ notation for the expected value $\boldsymbol{\lambda}$ for consistency and to address the goal of introducing covariates. The log-likelihood is then specified as

$$\mathcal{L} = \sum_{i=1}^{n} w_i \left\{ log\Gamma\left(\theta + \sum_{i=1}^{n_i} y_{it}\right) - \sum_{t=1}^{n_i} log\Gamma(1 + y_{it}) - log\,\Gamma(\theta) + \theta \log u_i \right.$$
$$\left. + log(1 - u_i)\left(\sum_{t=1}^{n_i} y_{it}\right) + \sum_{t=1}^{n_i} y_{it}(x_{it}\beta) \right\} - \left(\sum_{t=1}^{n_i} y_{it}\right) log\left(\sum_{t=1}^{n_i} \lambda_{it}\right) \tag{9}$$

Where * w_i is the user – specified weight for panel i; if no weights are specified, w_i=1. The estimating equation $\psi(\Theta = \psi(\beta, \theta)$ for a gamma distributed random effects Poisson model is then given by setting the derivative of the log-likelihood to zero

$$\left[\begin{Bmatrix} \frac{\partial \mathcal{L}}{\partial \beta_j} \\ \frac{\partial \mathcal{L}}{\partial \theta} \end{Bmatrix}\right]_{(p+1)\times 1} = [0]_{(p+1)\times 1} \tag{10}$$

where

$$\frac{\partial \mathcal{L}}{\partial \beta_j} = \sum_{i=1}^{n} \sum_{t=1}^{n_i} x_{jit} \left[y_{it} + \lambda_{it} \left((u_i - 1) \frac{\sum_{l=1}^{n_i} y_{il}}{\sum_{l=1}^{n_i} \lambda_{il}} - u_i \right) \right] \left(\frac{\partial \lambda}{\partial \alpha}\right)_{it} \tag{11}$$

$$\frac{\partial \mathcal{L}}{\partial \theta} = \sum_{i=1}^{n} \left[\psi\left(\theta + \sum_{i=1}^{n_i} y_{it}\right) - \psi(\theta) + ln\, u_i + (1 - u_i) - \frac{u_i}{\theta} \sum_{i=1}^{n_i} y_{it} \right] \tag{12}$$

and u_i is defined in equation (7), in the derivative with respect to θ equation (12).

Note: we use ψ( ) to denote the derivative of the log of the Gamma function (the psi-function). This is a standard notation for this function and should not be confused with our use of $\boldsymbol{\Psi}$( ) (Capital Psi) to denote the estimating equation.

STATISTICAL ANALYSIS AND INTERPRETATION

The results of fitting a gamma distributed random-effects model for the DM patients data are presented as in the following Table 3 to Table 10.

Table 3 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.0001 for the chi- square statistic in the Type 3 analysis table 3(a) indicates that the parameter of FBS is highly significant between the two regions.

Table 4 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.0001 for the chi- square statistic in the Type 3 analysis table 4(a) indicates that the parameter of PPBS is highly significant between the two regions.

Table 5 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.0026 for the chi- square statistic in the Type 3 analysis table 5(a) indicates that the parameter of BMI is highly significant between the two regions.

Table 6 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.0314 for the chi- square statistic in the Type 3 analysis table 6(a) indicates that the parameter of BMI is highly significant between the two regions.

Table 7 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.0763 for the chi- square statistic in the Type 3 analysis table 7(a) indicates that the parameter of TCHO is insignificant between the two regions.

Table 8 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.1365 for the chi- square statistic in the Type 3 analysis table 8(a) indicates that the parameter of LDL is insignificant between the two regions.

Table 9 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.0110 for the chi- square statistic in the Type 3 analysis table 9(a) indicates that the parameter of LDL is highly significant between the two regions.

Table 10 shows that the fitting of linear model for the parameter of gamma distribution in GLM. Further, the P – value of 0.1081 for the chi- square statistic in the Type 3 analysis table 10(a) indicates that the parameter of HDL is insignificant between the two regions.

Table 1. Characteristics of Study Parameters for Region 1 and Region 2 T2DM patients

Parameters	Region- 1		Region-2		Both Region	
	No. of Person	Percentage	No. of Person	Percentage	Total	Percentage
Gender						
Female	269	59.8	202	44.9	471	52.3
Male	181	40.2	248	55.1	429	47.7
Age(Years)						
<50	261	58.0	197	43.8	458	50.9
≥50	189	42.0	253	56.2	442	49.1

BMI						
<18.5	22	4.9	65	14.4	87	09.7
18.5-24.9	90	20.0	136	30.2	226	25.1
25-29.9	180	40.0	164	36.5	344	38.2
30 or more	158	35.1	85	18.9	243	27.0
Duration (years)						
<5	246	54.7	199	44.2	445	49.4
5-10	116	25.7	165	36.7	281	31.2
10-15	53	11.8	41	9.1	94	10.4
≥15	35	7.8	45	10.0	80	08.9

Table 2. Clinical Parameters of Region 1 and Region 2 T2DM patients

Parameters	Region- 1		Region-2		Both Region	
	No. of Person	Percentage	No. of Person	Percentage	Total	Percentage
FBS						
<100	35	07.8	54	12.0	89	09.9
100-125	82	18.2	112	24.9	194	21.6
≥125	333	74.0	284	63.1	617	68.6
PPBS						
<150	53	11.8	39	08.6	92	10.2
150-250	132	29.3	170	37.8	302	33.6
≥250	265	58.9	241	53.6	506	56.2
Creatinine (mg/dl)						
<1.5	371	82.4	333	74.0	704	78.2
≥1.5	079	17.6	117	26.0	196	21.8
Cholesterol (mg/dl)						
<150	080	17.8	121	26.9	201	22.3
150-200	157	34.9	149	33.1	306	34.0
≥200	213	47.3	180	40.0	393	43.7
HDL(mg/dl)						
<35	080	17.8	100	22.2	180	20.0
35-55	344	76.4	292	64.9	636	70.7
≥55	026	05.8	058	12.9	84	09.3
Triglycerides(mg/dl)						
<150	189	42.0	173	38.4	362	40.2
150-350	228	50.7	220	48.9	448	49.8
≥350	033	07.3	057	12.7	90	10.0
LDL(mg/dl)						
<150	390	86.7	421	93.6	811	90.1
≥150	060	13.3	029	06.4	89	09.9
SBP						
<120	170	37.8	125	27.8	295	32.8
120-160	224	49.8	252	56.0	476	52.9
>160	56	12.4	73	16.2	129	14.3
DBP						
<80	185	41.1	150	33.4	335	37.2
80-99	167	37.1	217	48.2	384	42.7
>100	98	21.8	83	18.4	181	20.1
HbA1c (%)						
<6.5	123	27.3	91	20.2	214	23.8
6.5-8.7	281	62.4	294	65.3	575	63.9
≥8.7	46	10.3	65	14.5	111	12.3

* BMI-body mass index, FBS-Fasting blood sugar, PPBS-Post prandial blood sugar, HDL- High density lipoprotein , LDL-Low density Lipoprotein, SBP- systolic blood pressure, DBP-diastolic blood pressure, HbA1c- Hemoglobin average blood glucose.

Table 3. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	5.0779	0.0267	5.0255	5.1302	36122.1	<0.0001
Both region (FBS)	1	0.3076	0.0378	0.2335	0.3816	66.26	<0.0001
Scale	1	3.1132	0.1396	2.8512	3.3992	-	-

Table 3(a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Fasting Blood Sugar Level)	1	63.91	<0.0001

Table 4. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	5.6201	0.0154	5.5899	5.6503	13288.9	< 0.0001
Both region (PPBS)	1	-0.1567	0.0218	-0.1994	-0.1140	51.65	< 0.001
Scale	1	9.3495	0.4331	8.5380	10.2381	-	-

Table 4(a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Post Prandial Blood Sugar)	1	50.22	< 0.0001

Table 5. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	0.9547	0.0164	0.9225	0.9868	3380.75	< 0.0001
Both region(BMI)	1	0.0702	0.0232	0.0247	0.1157	9.14	0.0025
Scale	1	8.2434	0.3810	7.5295	9.0250	-	-

Table 5 (a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Body Mass Index)	1	9.09	0.0026

Table 6. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	4.9212	0.0094	4.9028	4.9397	27421.5	< 0.0001
Both region (SBP)	1	-0.0286	0.0133	-0.0547	-0.0026	4.64	< 0.0312
Scale	1	25.1610	1.1783	22.9544	27.5798	-	-

Table 6(a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Systolic Blood Pressure Level)	1	4.63	0.0314

Table 7. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	5.2671	0.0097	5.2480	5.2862	29293.5	<0.0001
Both region (TCHO)	1	-0.0244	0.0137	-0.0513	-0.0026	3.15	0.0760
Scale	1	24.0526	1.1382	21.9222	26.3901	-	-

Table 7(a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Low Density Lipoprotein Level)	1	3.14	0.0763

Table 8. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	4.7344	0.0151	4.7047	4.7641	97890.4	< 0.0001
Both region (LDL)	1	-0.0319	0.0214	-0.0738	0.0101	2.22	0.1362
Scale	1	9.7050	0.4499	8.8622	10.6280	-	-

Table 8(a) - LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Low Density Lipoprotein Level)	1	2.22	0.1365

Table 9. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	5.2913	0.0240	5.2444	5.3383	48759.5	< 0.0001
Both region(TGL)	1	-0.0863	0.0339	-0.1528	-0.0199	6.49	< 0.0109
Scale	1	3.8700	0.1751	3.5416	4.2290	-	-

Table 9(a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (Triglycerides Level)	1	6.47	0.0110

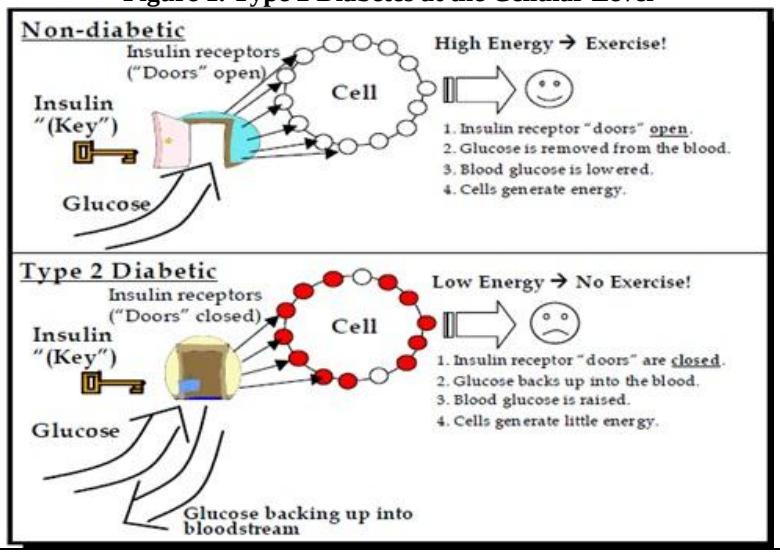
Table 10. Analysis of Parameter Estimates

Parameter	DF	Estimate	Standard Error	Wald 95% Confidence Limits		Chi- Square	p-value
Intercept	1	3.7757	0.0108	3.7545	3.7970	121331.0	< 0.0001
Both region (HDL)	1	-0.0246	0.0153	-0.0547	0.0054	2.59	< 0.1079
Scale	1	18.9130	0.8838	17.2577	20.7270	-	-

Table 10(a). LR Statistics for Type 3 Analysis

Source	DF	Chi- Square	p-value
Both region (High Density Lipoprotein Level)	1	2.58	0.1081

Figure 1. Type 2 Diabetes at the Cellular Level



DISCUSSION

The fitting of gamma distribution results as displayed in Table 3a to Table 10a shows that the two regions patient's clinical factor ranges are varying between Region 1 and Region 2 in Tables 3a, 4a, 5a, 6a and 9a, but not varying in Tables 7a, 8a, 10a. From this study, two regions clinical dataset conclude that the T2DM patients

were admitted to a good compliance of the risk factors which however contrasted with their actual health status. From this, T2DM prevalence in Region 1 found to be higher than the Region 2. Hence it is intended to find out whether the patients from Region 1 are more risk to have an impaired health status by comparing with Region 2. While comparing these two regions T2DM patients it is showed that the lipid pattern differs. The proportions of patients from Region 1 with elevated triglycerides are more frequent than those with elevated cholesterol values.

Obesity has been accompanied by an increasing prevalence of Region 1 patients. Since obesity is such a strong predictor of diabetes incidence, it appears that the rapid increases in the prevalence of T2DM seen in Region 1 patients are almost certainly related to increasing obesity. This is due to the change in their lifestyle and food pattern as it is a metropolitan city. This suggests that the highest risk of diabetes occurs among Region 1 patients. When the number of persons with BMI less than 25 is taken for consideration, there is a significant difference between the two regions.

A significant difference exists between the two regions people with inverse relation between T2DM and their physical activity. These two groups with high physical activity had the lowest prevalence. But their physical activity does not independently affect prevalence but through its associate with other factor such as obesity and stress level. The T2DM patients who had regular physical exercise have good control over their disease. In our study, the prevalence of T2DM is related to educational level significantly, though it does not have a direct role, the risk factor decreases due to healthier life style.

PREVENTION

The following tips can help to reduce risk of developing Type 2 diabetes and keep it low:

- Eat regular meals to keep blood glucose and blood pressure levels stable.
- Eat more fruits and green leafy vegetables
- Regular exercise at least 30 minutes daily, it brings about relaxation and reduces stress and also weight control
- To avoid alcohol consumption
- To avoid smoking and use of tobacco related products
- Reduce fatty foods – eat less amount of foods fried in oil and use less oil in diet
- Limit unhealthy snacks such as high in salt, yoghurt, reduce fat cheese and wholegrain crackers or unsalted nuts.
- Treatment typically includes diet control
- Home blood glucose testing and in some cases, oral medication and/or insulin.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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