

Association Between Hyperglycemia and Brain Stroke

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Abstract

The article focuses on the role of average blood glucose level (GLU) on some brain stroke patients based on 750 real study subjects consisting of both normal and brain stroke patients. The current outcomes have been derived herein using joint statistical modeling. It is derived herein that mean GLU level is positively associated with the joint interaction effect (JIE) of age and stroke (STR) i.e., AGE*STR ($P=0.0432$), JIE of hypertension (HYP) and residence type (RES) i.e., HYP*RES ($P=0.0261$), while it is negatively associated with the JIE of smoking (SMO) status and HYP i.e., SMO*HYP ($P=0.0206$), ever married (MAR) and heart disease (HRT) status i.e., MAR*HRT ($P<0.0001$). In addition, variance of GLU level is positively associated with HYP ($P<0.0001$), HRT ($P=0.0327$), JIE of MAR and STR i.e., MAR*STR ($P=0.0327$), while it is negatively associated with the JIE of HYP and MAR i.e., HYP*MAR ($P=0.0001$). There are many more associations of GLU levels with many other factors for brain stroke patients in the both mean and variance models. It can be concluded that average GLU levels maintain very complicated roles with several heart disease risk factors, physical and lifestyle factors. The brain stroke treatment process may be benefitted using the present derived complicated associations of glucose levels with the other factors. For all common people, the report informs about the controlling of GLU levels, BMI and smoking at older ages.

Keywords: average blood glucose (glu); body mass index (bmi); brain stroke (str); hypertension (hyp); heart disease (hrt) status; joint generalized linear models (jglm)

1. Introduction

Hyperglycemia (or elevated average blood glucose levels) is one of the most common the early phases of comorbidities in ischemic stroke, which is associated with brain infarct growth, worsened neurological outcomes and hemorrhagic transformation [1-5]. Many clinical and experimental stroke research studies have established thromboinflammation as a key mediator of ischemic stroke brain damage [6,7]. The hyperglycemia prevalence, defined as blood glucose level > 6.0 mmol/L (or 108 mg/dL), has been commonly observed in two thirds of all ischemic stroke subtypes on admission and in at least 50% in each subtype including lacunar strokes [4,5,8]. It is considered that hyperglycemia facilitates thromboinflammation by exciting the endothelium, neutrophils and platelets [9, 10]. In the setting of stroke, it was shown to weaken post-stroke cerebral blood flow, smash the blood-brain barrier, and cause hemorrhagic transformation [4, 5, 11-14]. Even so, the exact mechanisms underlying these investigations are incompletely understood [7,15, 16]. Brain stroke symptoms and signs may include an inability to move fully, or feel on one side of the body, speaking & understanding problems, or one side vision loss, or dizziness etc. These brain stroke symptoms and

signs often appear soon after the stroke has happened. If these symptoms and signs stay less than one or two hours, the brain stroke is a transient ischemic attack (TIA), also known as a mini-stroke. Note that a hemorrhagic stroke may also be related to a severe headache [17,18]. Then these brain stroke symptoms and signs can be extended for a long time. Long-term complications may include loss of bladder control and pneumonia [19, 20]. High blood pressure is the main risk factor for stroke. There are many other risk factors such as high blood cholesterol, obesity, tobacco smoking, diabetes mellitus, atrial fibrillation, end-stage kidney disease, etc. An ischemic stroke is generally caused by a blood vessel blockage, though there are also less common causes [8, 17, 18, 21]. On the other hand, a hemorrhagic stroke is caused by either bleeding into the space between the brain's membranes or directly into the brain. Bleeding may generally occur due to a ruptured brain aneurysm [9, 10, 19-21]. The diagnosis of a brain stroke is generally based on a physical examination, which is supported by medical imaging such as an MRI scan or CT scan. Note that a CT scan can leave out bleeding, but may not necessarily leave out ischemia, which early on generally does not show up on a CT scan.

Some other tests such as blood tests and an electrocardiogram (ECG) are performed to locate the risk factors and leave out the other possible causes. Also, low blood sugar may cause the same symptoms. Many earlier studies have focused on the blood glucose (GLU) level effects on the brain stroke patients using simple bivariate correlation, meta-analysis, multiple regression analysis, and machine learning techniques etc. [2,4,5,8,17,19, 21]. The current data set is a physiological data set, which is generally heteroscedastic in nature. The previous reports do not consider that the considered brain stroke data set is of a heteroscedastic nature. So, most of the earlier reports invite many debates and doubts. Moreover, the previous reports do not use any appropriate model fitting diagnostic tools on their final selected models, which may be doubtful. So, the research may not have a good faith on all the outcomes related to the earlier doubtful models. The roles of average blood glucose levels on the brain stroke patients are very few investigated based on probabilistic modeling. The current report searches for the following research hypotheses.

- Is there any association of GLU levels with cardiac risk factors, physical and lifestyle factors for brain stroke patients?
- If it is affirmative, how can we derive the most probable GLU levels association model?
- What is the most probable GLU levels statistical model?
- What are the effects of GLU levels on the brain stroke patients?

The current report searches the above research hypotheses considering the following sections such as materials & methods, statistical analysis & results, discussions, and conclusions. The current derived GLU levels statistical model is shown in Table 1 using the considered data set that is reported in the materials section. The statistical GLU levels mean and variance models are developed by joint generalized linear models (JGLMs), which is shortly reported in the methods section. The current derived outcomes are illustrated in the results section, while the present results are illustrated in the discussion section. Based on the present derived GLU levels mean and variance statistical models, the present report has drawn some necessary information that are reported in the conclusions section.

2. Materials and Methods

2.1 Materials

The current GLU levels statistical model is derived herein from a subset of 750 random sample objects out of 4981 brain stroke and normal sample units. The present considered brain-stroke (a medical condition) data set is available in the site- <https://www.kaggle.com/datasets/jillanisofttech/brain-stroke-dataset/data>. The data set contains basically normal subjects and patients of the brain strokes, which are mainly two types, one is stroke ischemic, due to lack of blood flow, and the other is hemorrhagic, due to bleeding. These two types of brain strokes cause parts of the brain to stop functioning properly. The current considered brain stroke data set contains 11 study characters such as gender (or sex) (male=0; female=1), age, hypertension (HYP) (no hypertension=0, hypertension=1), heart disease (HRT) (no heart disease=0, heart disease =1), ever-married (MAR) (no married=0; married=1), work type (WOK) (Govt job=1; private=2; self-employed=3; children=0), residence-type (RES) (rural=0; urban=1), average glucose level in blood (GLU), body mass index (BMI), smoking-status (SMO) (never smoke=1; former=1; smoker=3), stroke (STR) (no stroke=0; stroke=1).

2.2 Statistical Methods

The present study considers average blood glucose (GLU) level is the targeted response random variable that is to be modeled with the remaining

cardiac, lifestyle and physical characteristics. It is examined that the response GLU level is non-normally and heteroscedastic distributed random variable. The variance of GLU level can't be stabilized with the help of any suitable transformation, therefore it is modeled in the current article using joint generalized linear models (JGLMs) under both the gamma and log-normal distribution that is clearly described in [22-25]. A detailed discussion about JGLMs is given in the book by Lee, Nelder and Pawitan [22]. JGLMS for both the log-normal and gamma distribution are shortly reported herein. JGLMs for log-normal distribution: For the positive response Y_i (=GLU) with $E(Y_i=GLU) = \mu_i$ (mean) and $Var(Y_i=GLU) = \mu_i^2 = \sigma^2$ say, where μ_i 's are dispersion parameters and σ^2 reveals the variance function. Generally, log transformation $Z_i = \log(Y_i=GLU)$ is adopted to stabilize the variance $Var(Z_i) \approx \sigma^2$, but the variance may not always be stabilized [26]. For developing a GLU improved model, JGLMs for the mean and dispersion are considered. For the response GLU, assuming log-normal distribution, JGL mean and dispersion models (with $Z_i = \log(Y_i=GLU)$) are as follows:

$$E(Z_i) = \mu_i \text{ and } Var(Z_i) = \sigma^2,$$

$$\mu_i = x_i \beta \text{ and } \log(\sigma^2) = g_i \gamma,$$

where x_i and g_i are the explanatory factors/variables vectors of GLU associated with the mean regression coefficients β and dispersion regression coefficients γ , respectively.

JGLMs for gamma distribution: In the above stated Y_i 's (=GLU), the variance has two portions such as (based on the mean parameters μ_i 's) and (free of μ_i 's). The variance function $V(\cdot)$ displays the GLM family distributions. For instance, if $V(\cdot) = 1$, it is normal, Poisson if $V(\cdot) = 1$, and gamma if $V(\cdot) = \mu_i$ etc. Gamma JGLMs means and dispersion models of GLU are as follows: and, where and are the GLM link functions attached with the mean and dispersion linear predictors respectively, and, are the explanatory factors/variables vectors of GLU attached with the mean and dispersion parameters respectively. Maximum likelihood (ML) method is used for estimating the mean parameters, while the restricted ML (REML) method is applied for estimating the dispersion parameters, which are explicitly stated in the book by Lee, Nelder and Pawitan [22].

3. Statistical analysis & Results

3.1 Statistical Analysis

The report aims to derive the effects of average blood glucose (GLU) levels on the brain stroke patients. Probabilistic model of GLU levels has been derived on the remaining 10 explanatory variables such as heart disease related parameters (hypertension (HYP), subject's heart disease status (HRT), subject's stroke status (STR)), physical parameters (SEX or GEN, AGE, BMI), social & lifestyle parameters (residence type (RES), work type (WRK), ever married (MAR), smoking status (SMO)). Final GLU levels model has been accepted based on the smallest Akaike information criterion (AIC) value (within each class) that reduces both the squared error loss and predicted additive errors [27, p. 203--204]. Based on the AIC rule, JGLMs Log-normal fit (AIC= 6387.836) is better than gamma fit (AIC=6413.285). Table 1 presents the summarized JGLMs results of the GLU levels analysis of both the mean and variance models under both the log-normal and gamma distribution.

In both the mean and variance models some insignificant marginal effects such as SEX (or GEN), SMO, HYP (in mean model) and AGE, BMI (in variance model) are included in the Log-normal fitted model due to the marginality rule of Nelder [28]. According to the marginality of Nelder [28], if any higher order interaction effect is significant, then all its lower order interaction effects and marginal effects should be included in the model. For example, in the Log-normal fitted mean model (Table 1),

SEX*SMO is significant ($P=0.0078$), so the insignificant marginal effects SEX ($P=0.7704$) and SMO ($P=0.2132$) should be included in the model. Similarly, for other insignificant effects in the final selected Log-normal model. The generated GLU levels Log-normal fitted probabilistic JGLM (Table 1) is a data derived model that is to be examined by model checking tools. All the valid conclusions about GLU levels are obtained from the data derived Log-normal fitted GLU levels probabilistic model (Table 1) that should be taken based on appropriate graphical diagnostic tools, which is displayed in Figure 1. Figure 1(a) presents the absolute residuals plot for the Log-normal fitted GLU levels model (Table 1) with respect to the fitted

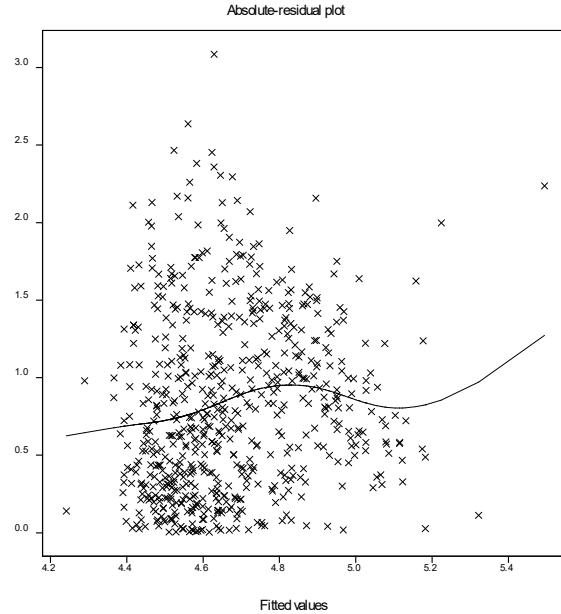


Figure 1 (a)

values, which is almost flat linear except the right tail, indicating that variance is constant with the running means. Note that the right tail is little increasing as a large absolute residual value is located at the right boundary. Figure 1(b) reveals the normal probability plot for the Log-normal fitted GLU levels mean model (Table 1) that does not reflect any lack of fit. So, both the figures 1(a) and (1b) do not show any discrepancy in the Log-normal fitted GLU levels model (Table 1). The above Figure 1(a) and Figure 1(b) confirm that the Log-normal fitted GLU levels model is an approximate form of the unknown true GLU levels model.

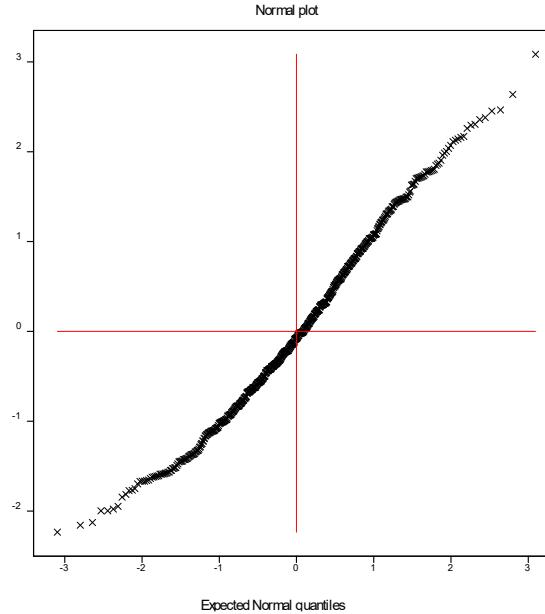


Figure 1 (b)

Figure 1: For the joint Log-normal fitted models of average glucose level in blood (glucose) (Table 1), the (a) absolute residual plot with the fitted values, and (b) the normal probability plot for mean model

3.2 Results

Table 1 displays the summarized results. Based on AIC rule, Log-normal fitted JGLM gives better results than Gamma fitted model. So, the final selected GLU levels model is Log-normal fitted JGLM. There are some discrepancies between these two fitted GLU levels models (in Table 1). The general discrepancies between Log-normal and Gamma fitted models are well discussed in [29, 30]. Herein the Log-normal fitted (Table 1) outcomes are presented, as its AIC value is lower than the Gamma fit.

The associations between the mean GLU levels and heart disease related parameters are illustrated in the following lines. It is derived herein that mean GLU levels is positively associated with the joint interaction effect (JIE) of age and stroke (STR) i.e., AGE*STR ($P=0.0432$), while both the marginal effects AGE ($P=0.0004$) and stroke (STR) ($P=0.0942$) are negatively associated with the mean GLU levels. Mean GLU levels is negatively associated with the JIE of smoking status (SMO) and hypertension (HYP) i.e., SMO*HYP ($P=0.0206$), while both the marginal effects SMO ($P=0.2132$) and HYP ($P=0.1681$) are insignificant. Mean GLU levels is negatively associated with the JIE of ever married (MAR) and subject's heart disease status (HRT) i.e. MAR*HRT ($P<0.0001$), while it is negatively associated with the marginal effect MAR ($P=0.0002$), and it is positively associated with the marginal effect HRT ($P<0.0001$). Mean GLU levels is positively associated with the JIE of HYP and subject's residence type (RES) i.e., HYP*RES ($P=0.0261$), while it is negatively associated with the marginal effect RES ($P=0.0295$), but it is insignificant of HYP ($P=0.1681$). The associations between the mean GLU levels and physical & social parameters are given in the following lines. Mean GLU levels is positively associated with the JIE of age and BMI i.e., AGE*BMI

($P=0.0032$), while it is negatively associated with both AGE ($P=0.0004$) and BMI ($P=0.0090$). Mean GLU levels is negatively associated with the JIE of sex (or gender) and SMO i.e., SEX*SMO ($P=0.0078$), while it is insignificant of both the SEX ($P=0.7704$) and SMO ($P=0.2132$). Mean GLU levels is positively associated with the JIE of AGE and MAR i.e., AGE*MAR ($P<0.0001$), while it is negatively associated with both the marginal effects AGE ($P=0.0004$) and MAR ($P=0.0002$). Also mean GLU levels is positively associated with the JIE of BMI and MAR i.e., BMI*MAR ($P=0.0037$), while it is negatively associated both of BMI ($P=0.0090$) and MAR ($P=0.0002$). Mean GLU levels is positively associated with the JIE of SMO and RES i.e., SMO*RES ($P=0.0427$), while it is negatively associated with RES ($P=0.0295$) and indifferent of SMO ($P=0.2132$). The associations between the GLU levels' variance and heart disease related parameters are illustrated in the following lines. Variance of GLU levels is positively associated with HRT ($P=0.0327$). It is negatively associated with the JIE of HYP and MAR i.e., HYP*MAR ($P=0.0001$), while it is positively associated with the marginal effect HYP ($P<0.0001$) and negatively associated with MAR ($P=0.0022$). Also, variance of GLU levels is positively associated with the JIE of MAR and STR i.e., MAR*STR ($P=0.0327$), while it is negatively associated with both the marginal effects of MAR ($P=0.0022$) and STR ($P=0.0123$). The associations between the GLU levels' variance and physical & social parameters are illustrated in the following lines. Variance of GLU levels is positively associated with the JIE of AGE and SEX i.e., AGE*SEX ($P=0.0789$), while it is negatively associated with the marginal effects SEX ($P=0.0739$) and insignificant of AGE ($P=0.1561$). Variance of GLU levels is positively associated with the JIE of AGE and MAR i.e., AGE*MAR ($P=0.0010$), while it is negatively associated with MAR ($P=0.0022$) and

insignificant of AGE ($P=0.1561$). Also, variance of GLU levels is positively associated with the JIE of BMI & MAR i.e., BMI*MAR ($P=0.0579$), while it is negatively associated with the marginal effect of MAR ($P=0.0022$) and insignificant of BMI ($P=0.8337$). From Table1, Log-normal fitted GLU levels mean (z) model is $z = 5.0861 - 0.0123 \text{ age} - 0.0162 \text{ bmi} + 0.0003 \text{ age*bmi} - 0.0100 \text{ gen2} + 0.0642 \text{ smoking2} - 0.1448 \text{ gen2*smoking2} - 0.2124 \text{ stroke2} - 0.5640 \text{ emarried2} + 0.0056 \text{ age*emarried2} + 0.0041 \text{ age*stroke} + 0.1114 \text{ hypert2} - 0.2251 \text{ smoking2*hypert2} + 0.6517 \text{ heartd2} - 0.5672 \text{ emarried2*heartd2} + 0.0135 \text{ bmi*emarried2} - 0.0718 \text{ resi2} + 0.1063 \text{ smoking2*resi2} + 0.2135 \text{ hypert2*resi2}$, and from Table 1, the Log-normal fitted GLU levels

variance (z) model is $z = \exp. (-1.901 - 0.0124 \text{ age} - 0.0047 \text{ bmi} + 2.419 \text{ hypert2} + 0.453 \text{ heartd2} - 0.634 \text{ gen2} + 0.0108 \text{ age*gen2} - 2.369 \text{ emarried2} + 0.0294 \text{ age*emarried2} - 2.136 \text{ hypert2*emarried2} + 0.0458 \text{ bmi*emarried2} - 1.208 \text{ stroke2} + 1.078 \text{ emarried2*stroke2})$. From the above GLU levels mean (z) and variance (z) models, it is observed that mean GLU levels is explained by many factors and their interaction effects such as age*bmi, gen2*smoking2, age*emarried2, age*stroke, smoking2*hypert2, emarried2*heartd2, bmi*emarried2, smoking2*resi2, hypert2*resi2, while the GLU levels variance is explained by age*gen2, age*emarried2, hypert2*emarried2, bmi*emarried2, and emarried2*stroke2.

Model	Covariates	LOG-NORMAL FIT				GAMMA FIT			
		estimate	s.e.	t(618)	P-value	estimate	s.e.	t(618)	P-value
Mean	constant	5.0861	0.1783	28.526	<0.0001	5.1904	0.1817	28.563	<0.0001
	age	-0.0123	0.0034	-3.559	0.0004	-0.0137	0.0035	-3.964	<0.0001
	bmi	-0.0162	0.0062	-2.621	0.0090	-0.0183	0.0063	-2.906	0.0038
	age*bmi	0.0003	0.0001	2.962	0.0032	0.0004	0.0001	3.286	0.0011
	gen 2	-0.0100	0.0342	-0.292	0.7704	-0.0076	0.0348	-0.218	0.8275
	smoking 2	0.0642	0.0515	1.246	0.2132	0.0726	0.0525	1.382	0.1675
	gen 2*smoking 2	-0.1448	0.0543	-2.667	0.0078	-0.1587	0.0552	-2.876	0.0042
	stroke 2	-0.2124	0.1267	-1.676	0.0942	-0.2479	0.1273	-1.947	0.0520
	emarried 2	-0.5640	0.1484	-3.799	0.0002	-0.6891	0.1508	-4.569	<0.0001
	age*emarried 2	0.0056	0.0014	4.038	<0.0001	0.0072	0.0014	5.146	<0.0001
	age*stroke 2	0.0041	0.0020	2.026	0.0432	0.0045	0.0020	2.196	0.0285
	hypert 2	0.1114	0.0807	1.380	0.1681	0.1487	0.0788	1.887	0.0596
	smoking 2*hypert 2	-0.2251	0.0970	-2.321	0.0206	-0.2525	0.0946	-2.667	0.0078
	heartd 2	0.6517	0.1223	5.328	<0.0001	0.6898	0.1174	5.875	<0.0001
	emarried 2 *heartd 2	-0.5672	0.1435	-3.953	<0.0001	-0.5794	0.1384	-4.187	<0.0001
	bmi*emarried 2	0.0135	0.0046	2.911	0.0037	0.0160	0.0047	3.421	0.0007
	resi 2	-0.0718	0.0329	-2.182	0.0295	-0.0762	0.0336	-2.271	0.0235
	smoking 2 *resi 2	0.1063	0.0523	2.031	0.0427	0.1135	0.0532	2.135	0.0331
	hypert 2 *resi 2	0.2135	0.0957	2.230	0.0261	0.2080	0.0934	2.227	0.0263
Dispersion	constant	-1.901	0.691	-2.75	0.0061	-1.786	0.686	-2.61	0.0093
	age	-0.0124	0.0087	-1.42	0.1561	-0.0137	0.0087	-1.58	0.1146
	bmi	-0.0047	0.0219	-0.21	0.8337	-0.0060	0.0217	-0.28	0.7796
	hypert 2	2.419	0.524	4.61	<0.0001	2.314	0.513	4.51	<0.0001
	heartd 2	0.453	0.211	2.14	0.0327	0.385	0.207	1.86	0.0634
	gen 2	-0.634	0.354	-1.79	0.0739	-0.680	0.351	-1.94	0.0528
	age*gen 2	0.0108	0.0061	1.76	0.0789	0.0115	0.0061	1.89	0.0592
	emarried 2	-2.369	0.773	-3.07	0.0022	-2.246	0.766	-2.93	0.0035
	age*emarried 2	0.0294	0.0089	3.29	0.0010	0.0295	0.0089	3.32	0.0009
	hypert 2 *emarried 2	-2.136	0.554	-3.86	0.0001	-2.098	0.542	-3.87	0.0001
	bmi*emarried 2	0.0458	0.0241	1.90	0.0579	0.0426	0.0239	1.78	0.0756
	stroke 2	-1.208	0.481	-2.51	0.0123	-1.232	0.477	-2.59	0.0098
	emarried 2 *stroke 2	1.078	0.504	2.14	0.0327	1.076	0.499	2.15	0.0319
	AIC	6387.836				6413.285			

Table 1: Results for mean and dispersion models for glucose from Log-normal & Gamma fit.

4. Discussions

The summarized GLU levels analysis outcomes are displayed in Table 1. Based on Table 1, the most appropriate GLU levels Log-normal fitted mean and variance models are displayed in the above results section. These two GLU level models show the different complicated associations of GLU levels with heart disease related risk factors and along with the other physical, social and life-style factors. These different associations of GLU levels are discussed in the following paragraphs. It is derived herein that mean GLU levels is positively associated with the JIE of age and STR i.e.,

AGE*STR ($P=0.0432$), while both the marginal effects AGE ($P=0.0004$) and STR ($P=0.0942$) are negatively associated with the mean GLU levels. This indicates that if GLU levels increase as the joint effect of AGE*STR also increases. In other words, it implies that for higher GLU levels subjects have greater brain stroke effects at older ages. It is noted that if the joint effect is significant, the marginal effects are unimportant. Therefore, the role of marginal effects is not discussed when the joint effect is significant. Mean GLU levels is negatively associated with the JIE of SMO (never smoke=1; former=1; smoker=2) and HYP (no hypertension=0, hypertension=1) i.e., SMO*HYP ($P=0.0206$), while both

the marginal effects SMO ($P=0.2132$) and HYP ($P=0.1681$) are insignificant. This implies that GLU levels increase when the joint effect of smoking and hypertension (i.e. SMO*HYP) decreases. It shows that subjects with lower effect of SMO*HYP may have higher GLU levels. Mean GLU levels is negatively associated with the JIE of MAR (no married=0; married=1), and subject's HRT (no heart disease= 0, heart disease =1) status i.e. MAR*HRT ($P<0.0001$), while it is negatively associated with the marginal effect MAR ($P=0.0002$), and it is positively associated with the marginal effect HRT ($P<0.0001$). This implies that GLU levels increase when the joint effect of ever married and heart disease status (i.e. MAR*HRT) decreases. This indicates that subjects with no marriage and no heart disease may have higher GLU levels. Mean GLU levels is positively associated with the JIE of HYP (no hypertension=0, hypertension=1) and subject's residence type (RES) (rural=0; urban=1) i.e., HYP*RES ($P=0.0261$), while it is negatively associated with the marginal effect RES ($P=0.0295$), but it is insignificant of HYP ($P=0.1681$). This shows that GLU levels increase as the joint effect of HYP*RES increases. It indicates that subjects residing in urban areas with hypertension may have higher GLU levels. This is observed in practice [7,13]. Note that the above four paragraphs focus the effects of GLU levels with different heart disease related parameters. The associations between the mean GLU levels and physical, lifestyle & social parameters are given in the following lines. Mean GLU levels is positively associated with the JIE of age and BMI i.e., AGE*BMI ($P=0.0032$), while it is negatively associated with both AGE ($P=0.0004$) and BMI ($P=0.0090$). It indicates that GLU levels increase as the joint effect AGE*BMI increases. It implies that older subjects with higher BMI levels may have higher GLU levels, which are observed in practice. Mean GLU levels is negatively associated with the JIE of sex (male=0; female=1) and SMO (never smoke=1; former=1; smoker=2) i.e., SEX*SMO ($P=0.0078$), while it is insignificant of both the SEX ($P=0.7704$) and SMO ($P=0.2132$). This shows that GLU levels increase as the joint effect SEX*SMO decreases. It implies that male subjects with no smoking may have higher GLU levels. This is a strange finding. It may be verified in similar data sets in the future research. This is not reported in any earlier articles. Mean GLU levels is positively associated with the JIE of AGE and MAR i.e., AGE*MAR ($P<0.0001$), while it is negatively associated with both the marginal effects AGE ($P=0.0004$) and MAR ($P=0.0002$). It indicates that mean GLU levels increase as the joint effect AGE*MAR increases. It implies that older married subjects may have higher GLU levels, which are observed in practice. Also mean GLU levels is positively associated with the JIE of BMI and MAR i.e., BMI*MAR ($P=0.0037$), while it is negatively associated both of BMI ($P=0.0090$) and MAR ($P=0.0002$). It implies that mean GLU levels increase as the joint effect of BMI*MAR increases. This indicates that married subjects with higher BMI levels may have higher GLU levels, which are commonly observed in the real society. Mean GLU levels is positively associated with the JIE of SMO and RES i.e., SMO*RES ($P=0.0427$), while it is negatively associated with RES ($P=0.0295$) and indifferent of SMO ($P=0.2132$). It implies that GLU levels increase as the joint effect SMO*RES increases. This indicates that smoker subjects residing in urban areas may have higher GLU levels. The associations between the GLU levels' variance and heart disease related parameters are illustrated in the following lines. Variance of GLU levels is positively associated with HRT ($P=0.0327$). It implies that GLU level values are highly scattered of the subjects with higher HRT. In addition, GLU levels' variance is negatively associated with the JIE of HYP and MAR i.e., HYP*MAR ($P=0.0001$), while it is positively associated with the marginal effect HYP ($P<0.0001$) and negatively associated with MAR ($P=0.0022$). This indicates that GLU level values are highly scattered of the subjects with lower joint effect HYP*MAR. Also, variance of GLU levels is positively associated with the JIE of MAR and STR i.e.,

MAR*STR ($P=0.0327$), while it is negatively associated with both the marginal effects of MAR ($P=0.0022$) and STR ($P=0.0123$). It implies that GLU level values are highly scattered of the subjects with higher joint effects of MAR*STR. There are a few more significant effects in the variance models, which are stated in the results section. These are not discussed herein as the information may not be used in medical sciences. Yet, the variance model has its own interpretations, which are important for dispersion of the response variable. Note that based on the variance model, the dispersion values of the response variable GLU levels of the subjects can be interpreted, which are associated with heart disease related risk factors, physical, lifestyle and social parameters. These can be interpreted similarly as above. In medical sciences, mean model interpretations are important for understanding the effects of the response variable. Many important outcomes have been pointed out in the above. It is derived that higher GLU levels have a greater risk of brain stroke at older ages, which are observed in practice. Marginal effect of smoking (SMO) ($P=0.2132$) is insignificant with the response GLU levels, while it has many joint effects such as (with hypertension (HYP)) SMO*HYP ($P=0.0206$), (with sex) SEX*SMO ($P=0.0078$), (with residence type) SMO*RES ($P=0.0427$) with the GLU levels. These joint effects are discussed in the above. It is noted that some joint effects (SMO*HYP and SEX*SMO) have negative association with the GLU levels, while SMO*RES has positive association with the GLU levels. It indicates that SMO*HYP and SEX*SMO may be treated as the protective effects, while SMO*RES may be viewed as a risk factor for GLU levels. So, it is better to avoid smoking. In addition, the marginal association of BMI ($P=0.0090$) is negative with the GLU levels, while its joint effects AGE*BMI ($P=0.0032$) and BMI*MAR ($P=0.0037$) are positively associated with the GLU levels, which are treated as the risk factors for it. So, the subject's BMI is very important for the brain stroke problem. In the above, the report has focused on many more factors and the joint effects which are related with the GLU levels.

5. Conclusions

The current article has derived the effects of GLU levels on the brain stroke patients along with the heart disease related factors, other physical, social and lifetime factors. The fitted GLU levels probabilistic model has been selected herein based on the smallest AIC rule, on comparison of joint Log-normal and Gamma models, standard error of the estimates and graphical diagnostic checking plots (Figure 1). Table 1 shows both the Log-normal and Gamma fitted models with similar interpretations. The interpretations about the effects of GLU levels on the brain stroke patients have been discussed above based on the fitted Log-normal model. Most of the derived findings herein focus on the real facts that are observed in practice. The obtained findings regarding GLU levels effects on the brain stroke patients herein though not completely eventual but are expressive. Modern scientific research methods should have complete faith on these obtained findings as the fitted models have been selected with graphical diagnostic checking and comparison of two different models.

The fitted GLU levels models (Table 1) are derived from the data set as reported in the material section. For any similar data sets of GLU levels on the brain stroke patients, the findings will be almost similar to the present findings, which are not verified herein as similar data sets are not available. The current outcomes reveal many real facts, which are rarely reported in the earlier articles. Most of the findings in the report are completely new in the brain stroke literature. In addition, the report may help all the people, brain stroke patients, medical practitioners and researchers. It is concluded that GLU levels have very complex functional roles (Table 1) on the brain stroke patients that should be known to the practitioners for appropriate

treatment processes. For all common people, the report informs about the controlling of GLU levels, BMI and smoking at older ages.

Abbreviations

AIC Akaike information criterion

BMI Body mass index

GLU Average blood glucose

HYP Hypertension

HRT Heart disease

JIE Joint interaction effect

JGLMs Joint generalized linear models

MAR Ever married

RES Residence type

SMO Smoking

STR Brain stroke

TIA Transient ischemic attack

Declarations: The article is an original interesting research report that has been prepared based on advance statistical data analysis using, which has not been submitted in any journal for publication.

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Conflict of interest: The authors confirm that this article content has no conflict of interest.

Ethical approval: Note that the current study has been performed based on a secondary data set, which is available in the site-
<https://www.kaggle.com/datasets/jillanisofttech/brain-stroke-dataset/data>
 The ethics approval and the subject consents are not required for a secondary published data set.

Data availability statement:

The data is available in the site-
<https://www.kaggle.com/datasets/jillanisofttech/brain-stroke-dataset/data>

Informed consent statement

Not applicable

Sample availability

The authors declare no physical samples were used in the study

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