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Research Article

Glucose and insulin levels' linkages with breast cancer factors

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Abstract

Serum lipid and glucose metabolisms are considered as the intermediary mechanisms for connecting obesity and Breast Cancer (BC). The article aims to identify the linkages between diabetes biochemical factors such as glucose & insulin and BC biochemical factors such as Monocyte Chemoattractant Protein-1 (MCP-1), adiponectin, Homeostasis Model Assessment score Insulin Resistance (HOMA-IR), resistin, leptin. These objectives can be focused on the two separate statistical models of glucose & insulin based on the remaining factors. From the mean model of glucose, it is observed that mean glucose level is directly linked with BC women (P= 0.023), leptin (P<0.001), HOMA-IR (P<0.001), while it is inversely linked with the interaction effects between HOMA-IR & leptin (i.e., HOMA-IR*leptin (P<0.001)) and adiponectin & leptin (i.e., adiponectin* leptin (P= 0.088)). Similarly, from the mean model of insulin, it is observed that mean insulin level is directly linked with HOMA-IR (P<0.001), leptin (P<0.001), and the interaction effects Age*MCP-1 (P= 0.091), glucose*adiponectin (P= 0.043), glucose*resistin (P<0.001), MCP-1*HOMA-IR (P<0.001), while it is reciprocally linked with MCP-1 (P= 0.026), resistin (P<0.001), adiponectin (P= 0.078), HOMA-IR*body mass index (BMI) (P<0.001), glucose*HOMA-IR (P<0.001), leptin*adiponectin (P= 0.071). There are more linkages of the variances of insulin and glucose with the BC biochemical factors. Based on the summarized results it is interpreted that diabetes biochemical factors such as insulin and glucose are highly linked with BC biomarkers such as MCP-1, HOMA-IR, leptin, resistin, and adiponectin.

Introduction

The most common diseases such as diabetes and cancer have horrible effects on human health all over the world. Epidemiologic researchers have pointed out that diabetes patients are always at higher risk of cancer [1-4]. Some researchers have established that Body Mass Index (BMI) and BC disease is closely associated [4-7]. The linkages between BMI and BC biochemical factors are focused on in a current article [7]. The linkages between the BC prognosis and metabolic syndrome have been reported in [8]. It is well known that metabolic syndrome is a union of a minimum of three of the following metabolic risks such as visceral obesity, elevated serum triglycerides, raised serum glucose, reduced serum insulin, raised blood pressure, reduced high-density lipoprotein cholesterol [8-10]. The definite linkages between BC prognosis and metabolic syndrome are still controversial [10-15]. Glucose metabolisms, Insulin-Like Growth Factors (IGF), resistin, lipid, and leptin have been claimed as probable intermediate mechanisms that are liable for connection between BMI and BC risk biochemical factors [9,11,13,15-18]. Some researchers have pointed out that the adverse hormonal profile (elevated insulin, estrogen, or leptin) is connected with the low levels of high-density lipoprotein that is considered to increase BC risk [19,20].

Most of the earlier cancer research studies are performed using basic statistical tools such as simple, or multiple correlation & regression [1-3,8,15,16], Kaplan Meier analysis [20], Cox model analyses [4] and Logistic regression, that are not always suitable statistical tools for finding the linkages of physiological heterogeneous, positive, non-normal continuous risk factors. Very little is known about the linkages between diabetes factors (glucose and insulin) and BC factors (MCP-1, HOMA-IR, adiponectin, resistin, leptin). The current

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article aims to give some clear linkages between diabetes factors (glucose and insulin) and BC factors (MCP-1, HOMA-IR, adiponectin, resistin, leptin) adopting Joint Generalized Linear Models (JGLMs) using a gamma distribution.

The above-stated article's aim is a *hypothesis-testing* medical science research query . Generally, in medical science cause-and-effect (i.e., causal relationship) research problems among the factors/ variables can be studied based on the probabilistic model of the effect (response or dependent variable) with the causal factors (independent or explanatory factors/variables). It is the *only* scientific technique for examining cause-and-effect medical science-related problems. In general, the physiological response variables are always heteroscedastic in medical science research studies, as there are many hidden unknown sources of variation. The derived response variable model should be examined by several model diagnostic tools before accepting it. Therefore, the article aims to examine the following research questions.

Generally, in medical science research problems for studying a cause-and-effect relationship among the variables/ factors, the researchers need to derive an appropriate mathematical model which can reveal the relationships among the variables. This is the *only* scientific method for studying causal epidemiological relationship problems. To focus on the article's aims, one needs to examine the following queries.

- What are the linkages of glucose levels with the BC factors?
- What are the linkages of insulin levels with the BC factors?
- How can the linkages be derived?
- What are the roles of the BC causal factors on the response variables glucose and insulin levels?

The above queries are addressed in the article adopting the sections materials & methods, statistical analysis & results, discussions, and conclusions. Glucose and insulin models along with their causal factors are reported in Tables 1,2, respectively. Moreover, JGLMs (discussed in the methods section) of glucose and insulin are displayed in the result section based on a real data set reported in the materials section. Finally, the roles of the causal factors are displayed in the discussion section.

Table 1: Glucose Level Gamma Fitted JGLMs.

Model	Factors	Estimate	Standard error	t-value	P-value
Mean	Constant	4.393	0.020	218.02	<0.001
	Insulin	-0.128	0.006	-22.58	<0.001
	HOMA-IR	0.625	0.028	22.03	<0.001
	Subject's type	0.022	0.009	2.32	0.023
	Leptin	0.004	0.001	6.21	<0.001
	HOMA-IR *Leptin	-0.002	0.002	-7.12	<0.001
	Adiponectin	0.001	0.001	0.78	0.437
	Leptin*Adiponectin	-0.001	0.001	-1.72	0.088
Dispersion	Constant	-8.844	1.019	-8.68	<0.001
	Age	0.053	0.016	3.31	0.001
	HOMA-IR	2.005	0.393	5.11	<0.001
	Age* HOMA-IR	-0.007	0.003	-2.19	0.031
	Insulin	-0.162	0.065	-2.51	0.014
	Leptin	-0.039	0.009	-4.29	<0.001
	Resistin	0.129	0.055	2.33	0.022
	Age*Resistin	-0.002	0.001	-2.08	0.040
	Insulin*HOMA-IR	-0.011	0.003	-3.79	<0.001
	MCP-1	-0.001	0.001	-2.57	0.012
AIC		771.87			

Table 2: Insulin Level Gamma Fitted JGLMs.

Model	Factors	Estimate	Standard error	t-value	P-value
Mean	Constant	0.669	0.227	2.95	0.004
	Age	-0.003	0.002	-1.64	0.104
	HOMA-IR	1.324	0.092	14.38	<0.001
	MCP-1	-0.001	0.001	-2.25	0.026
	MCP-1*Age	0.001	0.001	1.71	0.091
	BMI	0.030	0.005	5.97	<0.001
	HOMA-1R* BMI	-0.016	0.003	-6.01	<0.001
	Resistin	-0.033	0.004	-8.80	<0.001
	Glucose	-0.003	0.002	-1.86	0.067
	Glucose*HOMA-1R	-0.005	0.001	-23.69	<0.001
	Adiponectin	-0.027	0.015	-1.78	0.078
	Glucose*Adiponectin	0.001	0.001	2.05	0.043
	Glucose*Resistin	0.003	0.001	9.21	<0.001
	HOMA-1R*MCP-1	0.001	0.001	7.28	<0.001
	Leptin	0.005	0.001	3.41	<0.001
	Leptin*Adiponectin	-0.002	0.001	-1.82	0.071
Dispersion	Constant	-3.755	1.432	-2.62	0.010
	Age	-0.022	0.009	-2.44	0.017
	Glucose	0.004	0.015	0.24	0.808
	MCP-1	0.007	0.002	3.29	0.002
	Glucose*MCP-1	-0.001	0.001	-3.76	<0.001
	HOMA-IR	0.417	0.060	6.94	<0.001
	Leptin	-0.014	0.008	-1.75	0.083
	Subject's type	1.165	0.308	3.78	<0.001
AIC		318.77			

Materials and methods

Materials

Study subjects & design: The present study considers a secondary data set that is obtainable in the UCI Machine Learning Repository, and the data set is clearly described in the articles [21,22]. Very shortly, it is reproduced as follows. The study participants were 154 Portuguese women who were freshly diagnosed with BC disease, and they were considered from the Gynaecology Department of the University Hospital Centre of Coimbra (CHUC) between 2009 and 2013. The participants were grouped into 4 study groups depending on their BMI. The groups are (1) Without BC with BMI <25kg/m^2, n= 29; (2) Without BC with BMI >25kg/m^2, n= 48; (3) With BC with BMI <25kg/m^2, n= 30; (4) With BC with BMI >25kg/m^2, n= 47. The normal BMI (<25kg/m^2) females were taken from the Internal Medicine Department in an annual check-up of the aforementioned hospital. Obesity women (BMI >25 kg/m^2) were also taken from the same Department. These subjects had never been treated as malignant or benign, and they had no BC family history. Also, BC women subjects with normal BMI or obesity were considered from the surgically treated at the Gynaecology Department of the same hospital. There were 38 subjects with BMI >40kg/m^2, which were excluded finally from the study. So, the final study included 116 subjects out of which 64 were BC disease and 52 without BC disease. The data set

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includes the information on some factors/ variables such as age (in years), insulin (µU/mL), HOMA-IR, glucose (mg/dL), BMI, adiponectin (µg/mL), resistin (ng/mL), MCP-1, leptin(ng/mL), subject's types (1= healthy controls; 2= patients).

Statistical methods

In the above data set, there are two diabetes-related biochemical factors such as glucose and insulin levels, and five BC disease-related biochemical factors such as adiponectin (µg/mL), resistin (ng/mL), MCP-1, leptin(ng/mL), and HOMA-IR. The article aims to examine the linkages of diabetes-related biochemical factors with BC disease-related biochemical factors. Therefore, there are two response variables as glucose and insulin levels. It is identified that both the responses are heterogeneous, so they are modeled using gamma Joint Generalized Linear Models (JGLMs), which are clearly illustrated in the book by Lee, et al. [23]. Very shortly gamma JGLMs are described herein for immediate applications.

JGLMs under gamma distribution: For the above two stated Y_i's (=glucose, or insulin level), with $E(Yi)=\mu_i$ (mean) and $Var\left(Y_i\right)=\sigma_i^2\mu^2{}_i=\sigma_i^2V(\mu_i)$ say, where σ_i^2's are dispersion parameters and V(.) represents the variance function, while the variance has two elements such as V(.) (depending on the mean parameters) and σ_i^2 (free of μ_i's). The variance function $V(\mu_i)$ shows the GLM family distributions. For example, if $V(\mu) = \mu$, it is Poisson, gamma if $V(\mu) = \mu^2$ normal if $V(\mu) = 1$, etc. Gamma JGLMs mean and dispersion models are as follows:

$$\eta_i = g(\mu_i) = x_i^\tau \beta \text{ and } \in_i = h(\sigma_i^2) = w_i^\tau \gamma$$

where g (.) and h (.) are the GLM link functions connected to the mean and dispersion linear predictors respectively and x_i^τ, w_i^τ are the independent factors/variables vectors connected to the mean and dispersion parameters respectively. The maximum likelihood (ML) method is adopted for estimating mean parameters, while the restricted ML (REML) method is used for estimating dispersion parameters [23].

Statitical analysis & results

Statistical and graphical analysis

The response variable glucose level (or insulin level) is modeled using gamma JGLMs based on the remaining independent variables. The best gamma JGLMs have been considered depending on the lowest Akaike information criterion (AIC= 771.87 for glucose level) (or AIC= 318.77 for insulin level) value (within each class) that reduces both the squared error loss and predicted additive errors [24]. For both the response variables modeling, some partially significant or insignificant effects are taken in both the models due to the marginality rule given by Nelder [25] and also for better fitting [24]. Tables 1,2 provide the glucose level and insulin level gamma JGLMs fitted models, respectively.

The derived gamma fitted glucose level JGLMs in Table 1 have been examined by Figure 1. In Figure 1(a), the glucose level fitted absolute residuals are plotted against the fitted values, which shows that all the points are distributed randomly at a single point except only two points. It shows that the variance is constant with the running means. Figure 1(b) displays the mean glucose level gamma fitted JGLMs normal probability plot (Table 1) that does not present any lack of fit discrepancy. Figures 1(a) and 1(b) confirm that the gamma fitted glucose level JGLMs (Table 1) are nearly true modes.

The derived gamma fitted insulin level JGLMs in Table 2 have been examined by Figure 2. In Figure 2(a), the insulin level fitted absolute residuals are plotted against the fitted values, which shows that all the points are distributed randomly at a single point except only two points, while one lower absolute residual is located at the right boundary. As a result, the smooth curve is decreasing. It shows that the variance is constant with the running means. Figure 2(b) displays the mean insulin level gamma fitted JGLMs normal probability plot (Table 2) that does not indicate any lack of fit discrepancy. Figures 2(a) and 2(b) confirm that the gamma fitted insulin level JGLMs (Table 2) are nearly true modes.

Statistical analysis results

Glucose level gamma fitted JGLMs summarized outcomes are shown in Table 1. From the glucose fitted mean model, it is derived that mean glucose level is directly linked with BC women (P= 0.023), leptin (P<0.001), HOMA-IR (P<0.001), while it is inversely linked with the interaction effects HOMA-IR*leptin (P<0.001) and leptin*adiponectin (P= 0.088). From the glucose fitted variance model it is derived that variance of glucose is directly linked with HOMA-IR (P<0.001) and resistin (P= 0.022), while it is inversely linked with age*HOMA-IR (P= 0.031), leptin (P<0.001), age*resistin (P= 0.040), insulin*HOMA-IR (P<0.001) and MCP-1 (P= 0.012).

From Table 1, glucose level gamma fitted mean ($\hat{\mu}$) model is $\hat{\mu}$ = exp(4.393−0.128 Insulin + 0.625 HOMA-IR + 0.022 Subject's type + 0.004 Leptin − 0.002 HOMA-IR*Leptin + 0.001 Adiponectin − 0.001 Leptin*Adiponectin) and the glucose level gamma fitted variance ($\hat{\sigma}^2$) model is $\hat{\sigma}^2$ = exp(−8.844 + 2.005 HOMA-IR + 0.053 Age − 0.162 Insulin − 0.007 Age*HOMA-IR − 0.039 Leptin + 0.129 Resistin − 0.011 Insulin*HOMA-IR − 0.002 Age*Resistin − 0.001 MCP-1).

Insulin level gamma fitted JGLMs summarized outcomes are shown in Table 2. From the insulin fitted mean model it is derived that mean insulin level is directly linked with HOMA-IR (P<0.001), leptin (P<0.001), and the interaction effects MCP-1*Age (P= 0.091), glucose*adiponectin (P= 0.043), glucose*resistin (P<0.001), MCP-1*HOMA-IR (P<0.001), while it is reciprocally linked with MCP-1 (P= 0.026), resistin (P<0.001), adiponectin (P= 0.078), HOMA-IR*BMI (P<0.001), glucose*HOMA-IR (P<0.001), leptin*adiponectin (P= 0.071). From the insulin fitted variance model it is derived that variance of insulin is directly linked with MCP-1 (P= 0.002), HOMA-IR (P<0.001), Subject's type (P<0.001), while it is inversely linked with leptin (P= 0.083) and glucose*MCP-1 (P<0.001).

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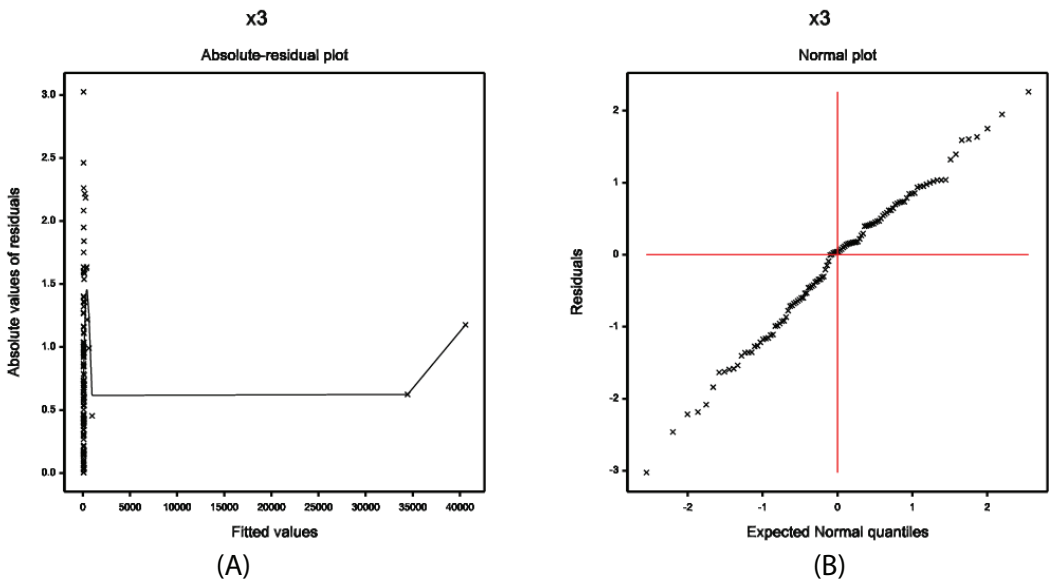

Figure 1: For the glucose level gamma fitted JGLMs (Table 1), the (a) absolute residuals plot against the fitted values, and (b) the normal probability plot for the mean model.

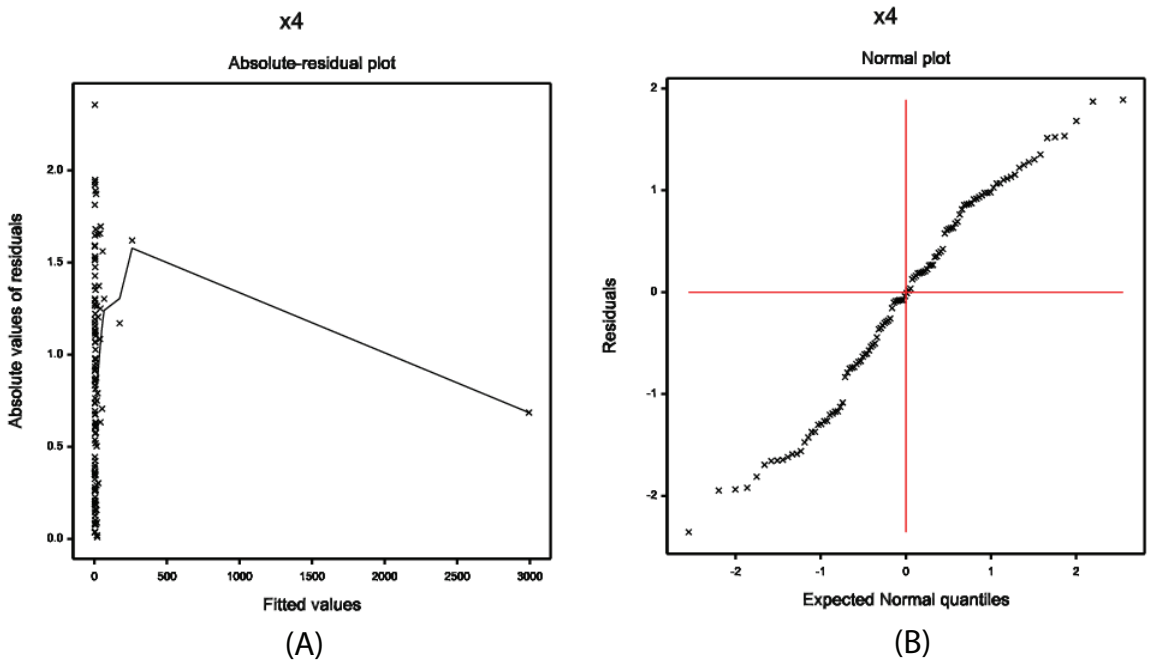

Figure 2: For the insulin level gamma fitted JGLMs (Table 2), the (a) absolute residuals plot against the fitted values, and (b) the normal probability plot for the mean model.

From Table 2, insulin level gamma fitted mean ($\hat{\mu}$) model is $\hat{\mu}$ = exp(0.669 + 1.324 HOMA-IR – 0.003 Age – 0.001 MCP-1 + 0.001 MCP-1*Age + 0.03 BMI – 0.016 HOMA-IR*BMI - 0.033 Resistin - 0.003 Glucose – 0.005 Glucose*HOMA-IR – 0.027 Adiponectin + 0.001 Glucose*Adiponectin + 0.001 Glucose*Resistin + 0.001 HOMA-IR*MCP-1 + 0.005 Leptin - 0.002 Leptin*Adiponectin) and the insulin level gamma fitted variance ($\hat{\sigma}^2$) model is $\hat{\sigma}^2$ = exp(–3.755 – 0.022 Age + 0.004 Glucose + 0.007 MCP-1 – 0.001 Glucose*MCP-1 + 0.417 HOMA-IR – 0.014 Leptin + 1.165 Subject's type).

Discussion

The linkages of glucose and insulin levels with BC biochemical parameters are summarized in Tables 1,2, along with their mean and variance models as in above. From the glucose fitted mean model (Table 1), it is derived that mean glucose level is directly linked with the subject's type (1= healthy controls; 2=patients) (P= 0.023), interpreting that glucose level is greater for BC females than normal. Also, mean glucose level is directly linked with leptin level (P<0.001) and HOMA-IR (P<0.001), implying that glucose level rises as leptin level or HOMA-IR increases. Even though both leptin

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level and HOMA-IR are directly linked with mean glucose level, their interaction effect HOMA-IR*leptin (P<0.001) is inversely linked with the mean glucose level. This shows that mean glucose level can not always increase as leptin level or HOMA-IR increases. In addition, mean glucose level is inversely linked with leptin*adiponectin (P=0.088), indicating that mean glucose level rises as the interaction effect leptin*adiponectin decreases. Note that mean glucose level is directly linked with leptin and independent with adiponectin.

From the glucose fitted variance model (Table 1), it is derived that variance of glucose is directly linked with HOMA-IR (P<0.001) and resistin (P= 0.022), implying that the glucose level of the subjects are highly scattered if their HOMA-IR or resistin level rises. In addition, the variance of glucose is inversely linked with age*HOMA-IR (P= 0.031), or leptin level (P<0.001), or age*resistin (P= 0.040), or insulin*HOMA-IR (P<0.001), or MCP-1 (P= 0.012), concluding that glucose level of the subjects is highly scattered if the effect of any one of age*HOMA-IR, leptin level, age*resistin, insulin*HOMA-IR, MCP-1 decreases.

From the insulin fitted mean model (Table 2), it is derived that the mean insulin level is directly linked with HOMA-IR (P<0.001), leptin level (P<0.001), or the interaction effects MCP-1*Age (P=0.091), or glucose*adiponectin (P= 0.043), or glucose*resistin (P<0.001), or MCP-1*HOMA-IR (P<0.001), concluding that mean insulin level rises as the effect of any one of them HOMA-IR, leptin level, MCP-1*Age, glucose*adiponectin, glucose*resistin, MCP-1*HOMA-IR rises. Also, it is derived that the mean insulin level is reciprocally linked with MCP-1 (P= 0.026), resistin level (P<0.001), adiponectin level (P= 0.078), HOMA-IR*BMI (P<0.001), glucose*HOMA-IR (P<0.001), leptin*adiponectin (P= 0.071), concluding that mean insulin level rises as the effect of any one of them MCP-1, resistin level, adiponectin level, HOMA-IR*BMI, glucose*HOMA-IR, leptin*adiponectin decreases.

From the insulin fitted variance model (Table 2), it is derived that the variance of insulin is directly linked with MCP-1 (P= 0.002), HOMA-IR (P<0.001), or Subject's type (P<0.001), indicating that insulin level is highly scattered for the BC women, and also for the subjects with a higher level of MCP-1 or HOMA-IR. Also, the variance of insulin level is inversely linked with leptin level (P= 0.083) and glucose*MCP-1 (P<0.001), interpreting that insulin level is highly scattered for the subjects with a lower level of leptin, or glucose*MCP-1. Relationships of MCP-1, adiponectin, leptin, and resistin with diabetes-related factors such as insulin and glucose are focused on in the articles [26-29].

The above linkages of glucose and insulin levels are derived from their joint gamma fitted mean and variance models. Note that both the fitted modes are accepted based on graphical diagnosis, and models are almost true. Therefore, the above linkages of glucose and insulin levels with BC biochemical factors such as HOMA-IR, adiponectin, resistin, MCP-1, leptin are almost true. The article establishes very complex linkages of glucose and insulin levels with BC biochemical factors.

Conclusion

The linkages of glucose and insulin levels with BC biochemical factors are derived herein using gamma JGLMs, while the best-fitted models are considered herein using the lowest AIC, small standard error of the estimates along with model diagnostic examinations. Therefore, the research should have higher faith in the derived linkages of glucose and insulin levels with BC biochemical factors. It is interpreted that diabetes biochemical factors such as insulin and glucose are highly linked with BC biomarkers such as MCP-1, HOMA-IR, leptin, resistin, and adiponectin. Medical researchers and practitioners can interpret glucose and insulin levels based on BC biochemical factors. Diabetic females are advised to take care of BC biochemical factors.

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