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Editorial
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Role of Glucose on Breast Cancer Patients

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Editorial

Cancer and diabetes mellitus (DM) are frequently occurring diseases with frightening impact on human health worldwide. In epidemiological studies, it is observed that people with DM are always at higher risk for several types of cancer [1-4]. Some research reports have pointed that there is a direct link between overweight and cruelty of breast cancer (BC) [5-7]. Correlation between the BC prognosis and metabolic syndrome has been reported in [8]. The following inquiries are surveyed in the present editorial report.

i. Are there any effects of glucose with BC patients and BC markers?

ii. If it is affirmative, what are the relations of glucose with BC patients and BC markers?

iii. What are the effects of glucose on BC patients and BC markers?

The above queries are searched in the editorial note based on a real data set of 116 normal and BC women along with 10 study variables, and the data set is available in the UCI Machine Learning Repository, and it is clearly illustrated in [9,10]. For immediate applications, the 10 study variables are presented as follows.

a. Age (years),

b. BMI (kg/m^2),

c. Homeostasis model assessment score insulin resistance (HOMA-IR),

d. Insulin (μU/mL),

e. Glucose (mg/ dL),

f. Resistin (ng/mL),

g. Adiponectin (μg /mL),

h. Monocyte chemoattractant protein-1 (MCP-1) (pg/dL),

i. Leptin (ng/mL),

j. Patient type (PT) (1=Healthy controls; 2= BC patients).

The above inquiries can only be surveyed by modeling glucose on the rest factors. Note that glucose is a positive continuous heteroscedastic random variable that can be modeled by joint generalized linear models (JGLMs) under both the lognormal and gamma distributions [11,12]. Joint gamma glucose model fit gives better results than lognormal fit, so only the gamma model fit outcomes are displayed in Table 1. In addition, the data generated glucose gamma model fit is justified by the graphical plots in Figure 1. Figure 1(a) reveals the absolute residuals plot with respect to glucose predicted values, which shows that all absolute residuals are randomly located at a single point except only two residuals. The smooth fitted line is exactly a flat straight line, except the right end which is increasing as a higher residual is located at the right boundary. Figure 1(b) presents the mean normal probability plot of glucose in Table 1. These two plots do not reveal any lack of fit. So, the gamma fitted glucose model (Table 1) is close to its true model. Gamma fitted mean & dispersion models of glucose are as follows.

Gamma fitted glucose mean () model (from Table 1) is

= exp(4.3933 + 0.625 HOMA-IR – 0.128 Insulin + 0.022 Tyes of patients + 0.004 LEPT

+ 0.001 ADIP – 0.002 HOMA-IR*LEPT – 0.001 LEPT*ADIP),

and the Gamma fitted glucose variance () model (from Table 1) is

= exp(–8.844 + 2.005 HOMA-IR + 0.053 Age – 0.007 Age*HOMA-IR – 0.162 Insulin

+ 0.129 REST – 0.039 LEPT – 0.002 Age*REST– 0.001 MCP-1 – 0.011Insulin*HOMA-IR).

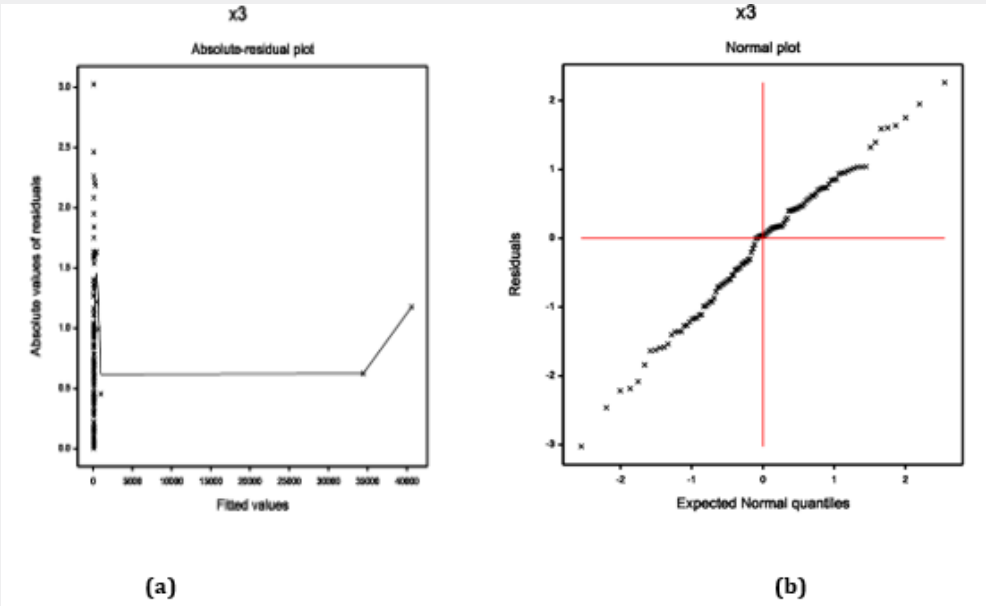

Figure 1: For the joint gamma glucose fitted model (Table 1), the (a) absolute residuals plot with respect to the glucose fitted values, and (b) the normal probability plot for the glucose mean model.

From the above glucose fitted mean & dispersion models (i.e., Table 1), the following relationships of glucose with BC markers can be reported.

Table 1: Results for mean and dispersion models of glucose fitting.

Model	Variables	From gamma fit			
		Estimate	s.e.	t-value	p-value
Mean	Constant	4.393	0.020	218.02	<0.001
	Insulin (INSU)	-0.128	0.006	-22.58	<0.001
	HOMA-IR	0.625	0.028	22.03	<0.001
	TYOP	0.022	0.009	2.32	0.022
	LEPT	0.004	0.001	6.21	<0.001
	HOMA-IR *LEPT	-0.002	0.001	-7.12	<0.001
	ADIP	0.001	0.001	0.78	0.437
	LEPT*ADIP	-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 (INSU)	-0.162	0.065	-2.51	0.014
	LEPT	-0.039	0.009	-4.29	<0.001
	REST	0.129	0.055	2.33	0.022
	Age* REST	-0.002	0.001	-2.08	0.040
	INSU* HOMA-IR	-0.011	0.003	-3.79	0.002
	MCP-1	-0.001	0.001	-2.57	0.012

Mean glucose levels are directly related with patient type (P=0.022), interpreting that mean glucose levels are higher for BC women than normal. It concludes that BC women have greater risk of DM. Mean glucose levels are directly related with leptin (P<0.001), implying that mean glucose levels rise as leptin levels increase. Mean glucose levels are directly related with

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HOMA-IR(P<0.001), concluding that glucose levels rise as HOMA-IR levels increase. Mean glucose levels are inversely related to the interaction effect of HOMA-IR and leptin (HOMA-IR*LEPT) (P<0.0001), indicating that mean glucose levels increase as HOMA-IR*LEPT decreases. Mean glucose levels are inversely related to the interaction effect of leptin and adiponectin (LEPT*ADIP) (P=0.088), concluding that mean glucose levels rise as the interaction effect LEPT*ADIP decreases. Mean glucose levels are inversely related with Insulin levels (P<0.001), concluding that mean glucose levels rise as insulin levels decrease, which is occurring in practice. Variance of glucose levels is directly related with HOMA-IR (P<0.001), indicating that subjects with higher HOMA-IR levels have highly scattered glucose levels. Variance of glucose levels is directly related with resist in levels (P=0.022), indicating that subjects with higher resistin levels have highly scattered glucose levels.

Variance of glucose levels is inversely related with leptin levels (P<0.001), concluding that subjects with lower leptin levels have highly scattered glucose levels. Variance of glucose levels is inversely related with MCP-1 levels (P=0.012), interpreting that subjects with lower MCP-1 levels have highly scattered glucose levels. Variance of glucose levels is inversely related with Age*HOMA-IR (P=0.031), or INSU* HOMA-IR (P=0.002), or Age*REST (P=0.040), concluding that variance of glucose levels is rises as Age*HOMA-IR, or INSU* HOMA-IR, or Age*REST decreases.

All the above conclusions are drawn from the appropriate glucose levels fitting model (Table 1), where the standard errors of all the estimates (Table 1) are very small, indicating that estimates are stable. From the above it is observed that mean and variance of glucose levels are highly associated with many BC markers and their joint interaction effects. It can be concluded that glucose levels rise for breast cancer women, along with the increase of HOMA-IR, leptin, and decrease of insulin, HOMA-IR*leptin, leptin*adiponectin. From this report medical practitioners and BC women will be benefitted. BC women should always care about their glucose levels regularly.

Conflict of Interest

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

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