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Editorial
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Role of HOMA-IR on Breast Cancer Women

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Editorial

Insulin resistance is suggested as a mediator of the onward cancer incidence and mortality related to obesity. Obesity is linked to increased breast cancer (BC) incidence, and high insulin circulating levels may inversely impact on cancer incidence. The association of insulin and homeostasis model assessment of insulin resistance (HOMA-IR) with BC and all-causes related to cancer mortality has been examined in many observational studies with mixed results [1-4]. The role HOMA-IR on BC biomarkers such as monocyte chemoattractant protein-1 (MCP-1), leptin, adiponectin, and resistin is little focused in the medical literature based on statistical modeling [5-7]. Relationship of HOMA-IR with BC has been reported in many articles [8, 9]. The following queries are investigated in the current editorial report.

- Are there any effects of HOMA-IR on BC biomarkers for BC patients?
- For the affirmative case, what are the probable relations of HOMA-IR with BC markers?
- What are the roles of HOMA-IR on BC biomarkers for BC patients?

The above-mentioned inquiries are studied in the current editorial report with the help of a real data set containing 116 BC patients & normal women with related 10 study characters. The data set can be viewed in the UCI Machine Learning Repository. A clear illustration of the data is given in [10,11]. For necessary use of the 10 study characters, they are reported as follows.

- Age (years),
- Body mass index (BMI) (kg/m^2),
- HOMA-IR,
- Insulin (μU/mL),
- MCP-1 (pg/dL),
- Glucose (mg/ dL),
- Resistin (ng/mL),
- Leptin (ng/mL),
- Adiponectin (μg /mL),
- Study unit type (SUT) (1=Healthy controls; 2= BC women).

The above questions can only be properly studied based on a probabilistic model of HOMA-IR on the remaining explanatory variables. It is noted that HOMA-IR is a continuous heteroscedastic positive random dependent variable that can be modeled using joint generalized linear models (JGLMs) under both the lognormal and gamma distributions [12,13]. Very shortly the joint gamma model is displayed in a recent article by Das [14]. JGLMs of HOMA-IR under gamma distribution give better results than lognormal fit, therefore, only the gamma model fit findings of HOMA-IR are displayed in Table 1.

The data developed for the HOMA-IR gamma model fit is diagnosed by Figure 1. Figure 1(a) shows the absolute residuals plot against the HOMA-IR predicted values, which reveals 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 is decreasing as a lower residual is located at the right boundary. Figure 1(b) shows the mean normal probability plot of HOMA-IR in Table 1. Figures 1(a) & 1(b) do not show any lack of fit. So, the gamma JGLMs fitted HOMA-IR model (Table 1) is an approximate true model (Figure 1).

Table 1: JGLMs model for HOMA-IR from Gamma fit Results.

Model	Covariate	Gamma fit			
		estimate	Standard error.	t-value	P-value
Mean	Constant	-3.18	0.11	-28.6	0.02
	BMI	0.02	0.01	5.4	<0.01
	Insulin	0.42	0.02	20.1	<0.01
	Insulin*BMI	-0.01	<0.01	-5.5	<0.01
	Glucose	0.02	<0.01	44.4	<0.01
	Insulin* Glucose	-0.01	<0.01	-31.7	<0.01
	Leptin	0.01	0.01	1.8	0.07
	Leptin* Insulin	-0.01	<0.01	-3.2	<0.01
	Study Unit type	-0.05	0.01	-4.0	<0.01
Dispersion	Constant	-15.79	3.54	-4.4	<0.01
	Leptin	-0.01	0.01	-0.5	0.59
	Glucose	0.13	0.04	3.3	<0.01
	Adiponectin	-0.46	0.23	-2.0	0.04
	Adiponectin* Glucose	0.01	0.01	2.2	0.03
	Age	0.17	0.06	2.6	0.01
	Glucose* Age	-0.01	<0.01	-3.5	<0.01
	Insulin	0.24	0.12	2.1	0.04
	Insulin* Age	0.01	<0.01	4.3	<0.01
	Insulin* Glucose	-0.01	<0.01	-3.2	<0.01
	Resistin	0.03	0.02	1.8	0.08
AIC		-56.8			

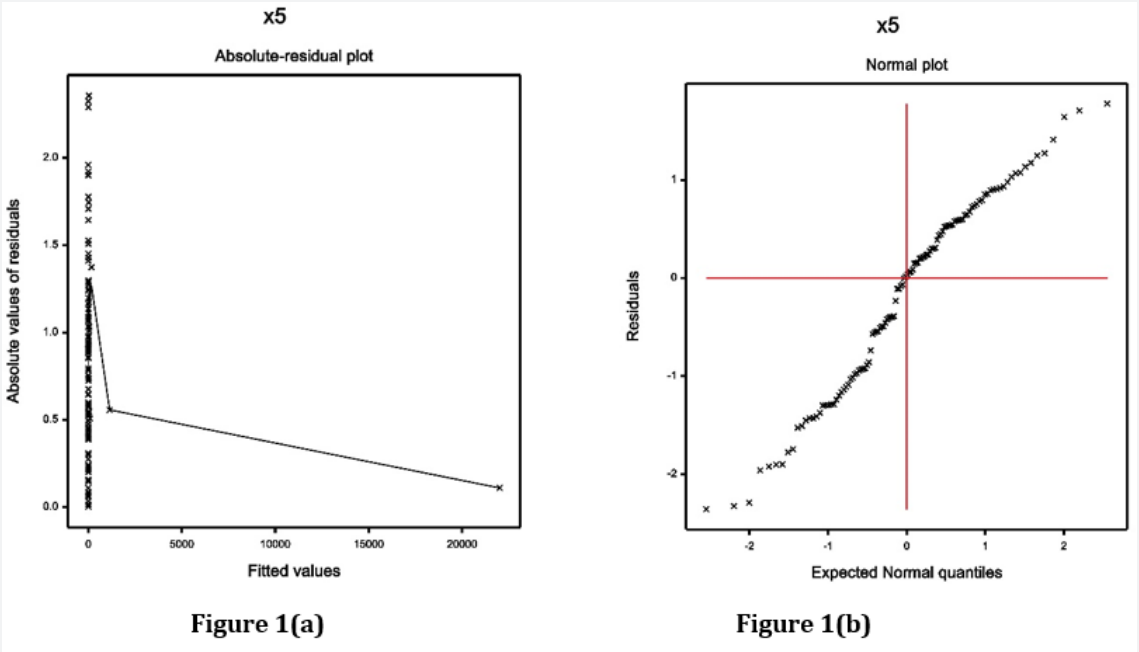

Figure 1(a) Figure 1(b)

Figure 1: For the gamma fitted JGLMs of HOMA-IR (Table 1), the (a) absolute residuals plot against the fitted values, and (b) the mean model normal probability plot of HOMA-IR.

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Gamma fitted mean & dispersion models of HOMA-IR are as follows.

Gamma fitted HOMA-IR mean () model (from Table 1) is

= exp(--3.18 + 0.02 BMI + 0.42 Insulin -- 0.05 Study Unit type – 0.01 Insulin*BMI + 0.02 Glucose – 0.01 Insulin*Glucose + 0.01 Leptin -- 0.01 Leptin*Insulin),

and the gamma fitted HOMA-IR variance () model (from Table 1) is

= exp(–15.79 – 0.01 Leptin + 0.13 Glucose – 0.46 Adiponectin + 0.01 Adiponectin*Glucose + 0.17 Age – 0.01 Glucose*Age + 0.24 Insulin + 0.01 Insulin*Age + 0.03 Resistin -- 0.01 Insulin*Glucose).

From the HOMA-IR fitted mean & dispersion models in Table 1, the following associations of HOMA-IR with BC markers can be noted.

i. Mean HOMA-IR is inversely related to the study unit type (1=Healthy controls; 2= BC women) (P<0.01), interpreting that HOMA-IR is higher for healthy women than BC patients.

ii. Mean HOMA-IR is directly linked to BMI (P<0.01), implying that mean HOMA-IR rises as BMI increases.

iii. Mean HOMA-IR is directly linked to insulin level (P<0.001), concluding that HOMA-IR rises as insulin levels increase.

iv. Mean HOMA-IR is inversely connected to the interaction effect BMI*insulin (P<0.001), concluding that HOMA-IR rises as BMI*insulin decreases. Note that BMI and insulin levels are directly connected to HOMA-IR, while their joint interaction effect is inversely connected to it.

v. Mean HOMA-IR is directly connected to glucose levels (P<0.01), indicating that mean HOMA-IR increases as glucose levels rise.

vi. Mean HOMA-IR is directly related to insulin and glucose levels, while it is inversely related to their joint interaction effect insulin*glucose (P<0.01), concluding that mean HOMA-IR rises as the interaction effect insulin*glucose decreases.

vii. Mean HOMA-IR is directly related to leptin (P=0.07), concluding that mean HOMA-IR rises as leptin levels increase.

viii. Mean HOMA-IR is directly related to leptin and insulin, while it is inversely related to their interaction effect leptin*insulin (P<0.01), implying that HOMA-IR rises as the interaction effect leptin*insulin decreases.

ix. Variance of HOMA-IR is directly related with glucose (P<0.01), insulin (P=0.04) and age (P=0.01), while their interaction effect insulin*age (P<0.01) is directly, and glucose*age (P<0.01) & insulin*glucose (P<0.01) are inversely related to the variance of HOMA-IR. Therefore, insulin, glucose and age have a very complex association with the variance of HOMA-IR.

x. Variance of HOMA-IR is directly related with glucose (P<0.01), and inversely related with adiponectin (P=0.04), while it is directly linked to their joint interaction effect glucose*adiponectin (P=0.03). In addition, it is directly related to resistin (P=0.08).

All the above interpretations are derived from the developed HOMA-IR gamma fitted model in Table 1, while the standard errors of all the estimates of HOMA-IR (Table 1) are very small, concluding that estimates are stable. From Table 1, it is noted that mean and variance of HOMA-IR are highly linked to many BC biomarkers and their joint interaction effects. Mean HOMA-IR is directly connected with BMI, insulin, glucose, leptin, while it is inversely related with insulin*BMI, insulin*glucose and insulin*leptin. It is concluded here that HOMA-IR is higher for normal women than BC women, and it increases as BMI, or glucose, or insulin increases, or insulin*BMI, or insulin*glucose, or insulin*leptin decreases. BC women, medical researchers & practitioners will be benefited from the report. HOMA-IR, glucose, insulin, leptin levels along with BMI are regularly examined for BC women.

Conflict of Interest

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

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