

## Role of Insulin Level on Pregnant American (Pima) Indians

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The worldwide escalation of obesity (or body mass index (BMI)) and diabetes mellitus (DM) in economically developed, or developing countries poses a great health challenge. BMI is one of the principal causes of type 2 diabetes. Type 1 diabetes is mainly due to the autoimmune-mediated waste of pancreatic beta cells leading to insulin deficiency [1-3]. Generally, type 1, gestational, type 2 diabetes are noticed in practice all over the world. Due to some unusualness of human organs, if the pancreas cannot produce insulin, type 1 DM occurs, which is termed as juvenile diabetes [4,5-7]. Some gestational women (GW) are frequently observed with high glucose levels during pregnancy, and they are affected with diabetes that is called gestational diabetes (GD). Later on, the GD reduces to type 2 diabetes [3,4,8,9]. The present Editorial note examines the following hypotheses related to some gestational American (Pima) Indian heritage females.

- Is there any effect of insulin level on gestational American (Pima) Indian heritage women?
- If it is affirmative, what are the effects of insulin level on the GW?
- What are the roles of insulin level on the other factors of GW?
- What is the relationship of insulin level with the other factors?

The above hypotheses are searched in the Editorial note based on a real data set of 768 American (Pima) Indian GW at least 21 years old. The data set is available in the UCI Machine Learning Repository, and it contains 9 study characters, which are as follows.

- Body mass index (BMI),
- Age (in years),
- Diastolic blood pressure (BP) (mm Hg),
- Plasma glucose concentration level over 2 hours in an oral glucose tolerance test (Glucose),
- Investigation unit type (IUT) (1=non-diabetic, 2= diabetic),

- Triceps skin-fold thickness (TST) (mm),
- Number of pregnancies (NOP),
- 2-hours serum insulin ( $\mu$ U/ml) (Insulin),
- Diabetes pedigree function (DPF).

The above collected characteristics are variables, except IUT, which is an attribute character. DPF is a function that estimates diabetes likelihood depending on family history.

The above hypotheses can be studied by deriving an appropriate model of insulin level on the remaining eight independent variables. It is clear that insulin level is a continuous unequal variance positive response variable that can be modeled properly applying joint generalized linear models (JGLMs) under gamma distributions [10,11]. Joint gamma model is briefly presented in a current article by Das [12]. JGLMs of insulin level under gamma distribution show better outcomes, therefore only the joint gamma fit outcomes of insulin level are presented in Table 1.

The data generated insulin level joint gamma fit models (Table 1) are diagnosed by Figure 1. Figure 1 represents the absolute insulin level gamma fit residuals plot against the predicted values. It is observed that all absolute residual points are randomly dispersed against a single point, except a point at the right boundary. Figure 2 shows the insulin level gamma fitted mean model (Table 1) normal probability plot that does not show any lack of fit. The two plots Figure 1 and Figure 2 show that the insulin level fitted joint gamma models are nearly the unknown true models.

From Table 1, joint gamma insulin level fitted mean ( $\hat{\mu}$ ) model is  $\hat{\mu} = \exp(-1.93 + 0.02 \text{ Glucose} - 0.13 \text{ Pregnancies} + 1.07 \text{ DPF} + 0.25 \text{ Pregnancies} * \text{ DPF} - 0.05 \text{ Age} + 0.07 \text{ BP} + 0.39 \text{ TST} - 0.01 \text{ BP} * \text{ TST} - 0.14 \text{ TST} * \text{ DPF} - 0.14 \text{ BMI} + 0.08 \text{ BMI} * \text{ DPF})$ , and also from Table 1, the joint gamma insulin level fitted dispersion ( $\hat{\sigma}^2$ ) model is  $\hat{\sigma}^2 = \exp(3.57 + 0.14 \text{ Pregnancies} + 0.02 \text{ Age} - 0.01 \text{ Pregnancies} * \text{ Age} - 0.05 \text{ BMI} - 0.34 \text{ DPF} - 0.01 \text{ Glucose} + 0.01 \text{ Glucose} * \text{ BMI})$ .

From the insulin level fitted mean & dispersion models as the above (or Table 1), the following relationships of insulin level with

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Table 1: Joint gamma fit of insulin level mean and dispersion models for pregnant women.

Model	Covariate	estimate	Standard error.	t-value	P-value
Mean	constant	-1.93	1.44	-1.34	0.18
	Glucose	0.02	0.01	2.78	0.01
	Pregnancies	-0.13	0.10	-1.32	0.19
	DPF	1.07	1.71	0.63	0.53
	Pregnancies *DPF	0.25	0.15	1.72	0.09
	Age	-0.05	0.02	-2.60	0.01
	BP	0.07	0.01	6.45	<0.01
	TST	0.39	0.04	8.86	<0.01
	BP*TST	-0.01	<0.01	-4.16	<0.01
	DPF*TST	-0.14	0.03	-4.48	<0.01
	BMI	-0.14	0.04	-3.91	<0.01
	DPF*BMI	0.08	0.05	1.51	0.13
Dispersion	Constant	3.57	0.96	3.72	<0.01
	Pregnancies	0.14	0.06	2.36	0.02
	Age	0.02	0.01	3.01	<0.01
	Pregnancies*Age	-0.01	<0.01	-2.04	0.04
	BMI	-0.05	0.03	-1.79	0.07
	DPF	-0.34	0.16	-2.16	0.03
	Glucose	-0.01	0.01	-1.46	0.14
	Glucose*BMI	0.01	<0.01	1.45	0.15
AIC			7195.53		

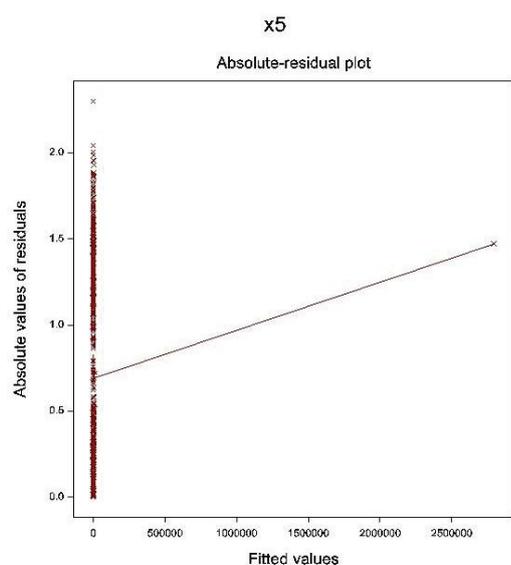


Figure 1: For the joint gamma fitted insulin level (Table 1) and the absolute residuals plot against the insulin level fitted values.

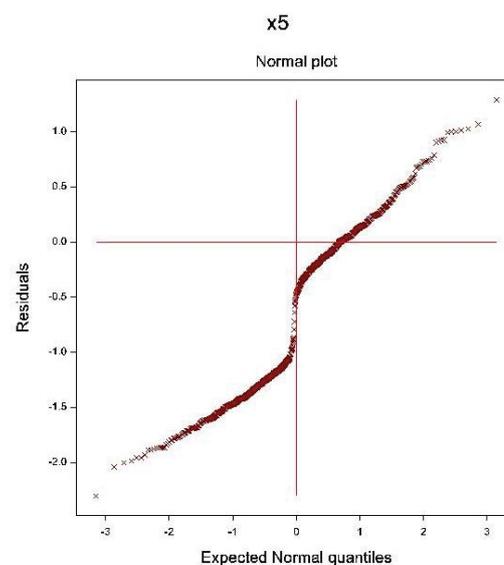


Figure 2: For the joint gamma fitted insulin level (Table 1) and the normal probability plot for the insulin level mean model.

the rest factors can be reported as follows.

- For GW, mean insulin level is directly related to glucose level ( $P=0.01$ ), implying that insulin level rises as the glucose level increases, which is the usual functional activity of insulin.
- Mean insulin level is inversely related to age ( $P=0.01$ ), interpreting that insulin level reduces at older ages of GW, which is observed in practice.
- Mean insulin level for GW is partially directly connected with Pregnancies\*DPF ( $P=0.09$ ), concluding that it rises as the joint effect of DPF and pregnancies rises. It means that when DPF and pregnancies are higher, insulin level is also higher. It is noted that insulin level is free of the marginal

effects of DPF ( $P=0.53$ ) and pregnancies ( $P=0.19$ ), but it is influenced by their joint effect.

- Mean insulin level is directly connected with BP ( $P<0.01$ ) and TST ( $P<0.01$ ), but it is inversely connected with the joint interaction effect BP\*TST ( $P<0.01$ ), concluding that it rises as the BP and TST rises, but it does not rise proportionally as it is negatively associated with the joint effect BP\*TST ( $P<0.01$ ).
- Mean insulin level is inversely related to the joint effect DPF\*TST ( $P<0.01$ ), but DPF is free of insulin level. So, the mean insulin level can't rise proportionally as TST, or BP rises.

- Mean insulin level is inversely related to BMI ( $P < 0.01$ ), concluding that it decreases as the BMI rises. Also, mean insulin level is directly partially related to the interaction effect BMI\*DPF ( $P = 0.13$ ), but DPF is independent of insulin level. So, for GW with high DPF along with high BMI, insulin level may rise a very little.
- Insulin level's variance is directly related to pregnancies ( $P = 0.02$ ) and age ( $P < 0.01$ ), but it is inversely related to their joint interaction effect Age\*Pregnancies ( $P = 0.04$ ), concluding that insulin level is not highly scattered at older ages along with higher pregnancies.
- Insulin level's variance is partially inversely related to BMI ( $P = 0.07$ ), interpreting that insulin level is highly scattered for GW with lower BMI.
- Insulin level's variance is inversely related to DPF ( $P = 0.03$ ), implying that insulin level is highly scattered for the GW with smaller DPF values.
- Insulin level's variance is partially inversely related to glucose level ( $P = 0.14$ ), and it is partially directly related to their joint interaction effect Glucose\*BMI ( $P = 0.15$ ), concluding that insulin level variance has a complex relationship with the other factors.

The Editorial note has focused on the relationships of insulin level with age, BMI, TST, DPF, BP, glucose level, IUT, NOP for American (Pima) Indians GW. The report has focused on a complicated insulin level's relationship with the rest of the explanatory factors. Mean & dispersion models of insulin level have displayed many uncommon findings that are really new to the GW study literature. Medical experts, researchers, practitioners and GW will be benefited from the current Editorial note. The report concludes that for GW, insulin level rises with the increase of glucose level, TST, BP, and the interaction effects DPF\*Pregnancies & DPF\*BMI, and it decreases with the increase of BMI, age, and the interaction effects TST\*BP & TST\*DPF. Pregnant females should be careful of her BMI, BP, glucose level, and TST regularly.

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

## REFERENCES

1. Das RN, Lee Y, Mukherjee S, Oh S. Relationship of body mass index with diabetes & breast cancer biomarkers. *J Diab Manag.* 2019; 9:163-168.
2. Mishra M, Ndisang JF. A critical and comprehensive insight on heme oxygenase and related products including carbon monoxide, bilirubin, biliverdin and ferritin in type-1 and type-2 diabetes. *Curr Pharm Des.* 2014; 20:1370-1391.
3. Petersen MC, Vatner DF, Shulman GI. Regulation of hepatic glucose metabolism in health and disease. *Nat Rev Endocrinol.* 2017; 13: 572-587.
4. Marchetti P, Dotta F, Lauro D, Purrello F. An overview of pancreatic beta-cell defects in human type 2 diabetes: implications for treatment. *Regul Pept.* 2008; 146:4-11.
5. Kido Y, Nakae J, Accili D. Clinical review 125: The insulin receptor and its cellular targets. *J Clin Endocrinol Metab.* 2001; 86:972-979.
6. Gray N, Picone G, Yashkin A. The relationship between BMI and onset of diabetes mellitus and its Complications. *South Med J.* 2015; 108: 29-36.
7. Odeleye OE, Courten M, Pettitt DJ, Ravussian E. Fasting hyperinsulinemia is a predictor of increased body weight and obesity in Pima Indian children. *Diabetes* 1997; 46:1341-1345.
8. Knowler WC, Pettitt DJ, Saad MF, Bennett PH. Diabetes mellitus in Pima Indians incidence risk factors and pathogenesis. *Diabetes Metab Rev.* 1990; 6:1-27.
9. Bennett PH, Burch TA, Miller M. Diabetes mellitus in American (Pima) Indians. *Lancet.* 1971; 2: 125-128.
10. Das RN, Lee Y. Log-normal versus gamma models for analyzing data from quality-improvement experiments. *Qual Eng.* 2009; 21:79-87.
11. Lee Y, Nelder JA, Pawitan Y. Generalized Linear Models with Random Effects (Unified Analysis via H-likelihood) (second edition). Chapman & Hall, London, 2017.
12. Das M. Induced Abortion Trends for Senior Women ( $\geq 35$  Years Old) in New Zealand between 2000-2019. *Act Sci Clin Case Rep.* 2021; 2:24-30.