ORIGINAL ARTICLE

Impact of Dietary Acid Load on Pancreatic β-cells Function and Insulin Resistance in Type 2 Diabetic Nigerians

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ABSTRACT

Background: The possible impact of high intake of dietary acid on pancreatic β-cells dysfunction and insulin resistance in type 2 diabetic patients has been suggested from previous studies; but findings across different study groups are conflicting. **Objective:** To determine the impact of dietary acid load on pancreatic β-cell dysfunction and insulin resistance in a group of patients with uncomplicated type 2 diabetes mellitus. **Method:** Study subjects were categorized in to four quartiles according to their dietary acid intake. Assessment of dietary intake was done using a food frequency questionnaire and the Nigerian Food Composition Table. Acid forming potential of our local diets were estimated as Potential Renal Acid Load (PRAL) scores. Pancreatic β-cell function and insulin resistance were estimated as HOMA-β and HOMA-IR respectively. **Results:** Degree of pancreatic β-cells function was observed to be significantly lower in subjects in the highest quartile of the PRAL score (*p for trend* < *0.05*). There was a statistically significant trend with higher intake of dietary acid associated with increased degree of insulin resistance (*p for trend*< 0.05). Dietary acid load was found to be a significant predictor of pancreatic β-cells dysfunction among the study subjects. **Conclusion:** Among subjects with type 2 diabetes mellitus in this study, consumption of a diet loaded with high acid forming potential food items was associated with greater insulin resistance and lower insulin secretion ability. High intake of dietary acid might be an additive mechanism contributing to deterioration of glycemic control in type 2 diabetic patients in our setting.

Keywords: Dietary acid; Diabetes mellitus; Pancreatic β-cell function

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Introduction

Relative pancreatic β-cell failure is described to be present at the diagnosis of type 2 diabetes mellitus and progressively deteriorate with disease duration. The progressive failure of the pancreatic β-cells to secrete enough insulin to compensate for the decrease of tissue sensitivity to insulin is associated with worsening of glycemic control and therapeutic failure; however, the progression is variable and potentially influenced by genetic and environmental factors.[1-3]A number of mechanisms underlying the progressive decline in pancreatic β-cell function among patients with type 2 diabetes mellitus have been proposed.[4-6]

Recently, there is increasing evidence, from studies mainly done among Caucasians, to suggest that regular consumption of diet with high acid load is associated with the development of chronic mild metabolic acidosis,[7] which has been proposed to be one of the potential predictors of decreased insulin secretion and sensitivity.[8-12] However, the definitive relationship between dietary acid load and indices of

insulin secretion and resistance, in diabetic patients and non-diabetics, is still controversial.[13-21]

Given that substantial differences in intake quality and quantities exist between Western and African settings,[22] studies investigating the independent effects of diet induced acid-base imbalance on the indices of insulin secretion and resistance among type 2 diabetic patients in our setting are warranted: with the hope of identifying targets for the optimization of treatment and reduction of the complications of the disease in our setting. Generally, food items such as red meat and eggs have high acid forming potential, while vegetables and fruits have low acid forming potential.

We aim to examine the impact of consumption of dietary acid load, as determined by potential renal acid load (PRAL) scores, on the degree of pancreatic β-cells dysfunction and insulin resistance in a group of Nigerian patients with uncomplicated type 2 diabetes mellitus.

Method

Study population

The participants for this cross-sectional analytical study were one hundred and ninety-two (192) patients with uncomplicated type 2 diabetes mellitus attending an outpatient clinic in the Gombe State Specialist Hospital, Gombe, Nigeria for routine follow up or for the first time. Ethical approval for the study was obtained from the Health Research Ethics Committee of the Gombe State Ministry of Health, Gombe, Nigeria and all the study participants gave informed consent.

WHO diagnostic criteria were used for the diagnosis of diabetes mellitus, and participants were classified as having type 2 diabetes mellitus based on clinical ground of non-dependence on insulin therapy for survival.[1] The study participants were grouped in to four quartiles according to their dietary PRAL scores (mEq/day). All study participants are Nigerians and living in Gombe State. The exclusion criteria were clinical and/or biochemical evidence of secondary diabetes mellitus or any other pre-existing disease, pregnancy, breastfeeding or use of contraceptives. Smoking and/or ingestion of alcohol, insulin dependence, current therapy with insulin or other medications known to interfere with insulin metabolism were also part of the exclusion criteria. Under-reporters, (≤ 800 kcal/day) or over-reporters (> 4,200 kcal/day) of dietary intake and those on special diets during the recruitment were also excluded.

Data collection

The demographic and clinical characteristics of the participants, including age, sex, dietary intake and duration of diabetes mellitus were obtained and recorded. Each participant underwent a routine physical examination; body height and weight were taken with each participant standing erect without shoes or headgear and with only undergarment. Body mass index (BMI) was calculated as weight in kilogram (kg) divided by height in meters squared (m^2) and expressed as kg/m^2. Blood pressure measurements were done with the participants in a sitting position with the arm placed at the level of the heart using a mercury sphygmomanometer.

Assessment of Dietary Intake

A semi-quantitative food frequency questionnaire (FFQ), that included locally available foods, was used to collect data on dietary intake in all the participants. The frequency of consumption and portion sizes of foods and drinks each of the participants consumed over the past week were obtained and recorded during the interview. Frequencies of consumption of rare foods were estimated as monthly consumption frequency and then converted to frequency per week. The frequencies were then all converted to daily frequency of consumption and using household measures, the portion sizes were converted to grams. The energy and nutrient content of the food items consumed were then determined using the Nigerian food composition table.[23]

Laboratory analysis

Fasting venous blood samples were collected in the morning following 10-12 hours overnight fasting into heparin bottles. Blood samples were immediately centrifuged for 15 minutes for separation of plasma, which was stored in aliquots at -20^0C until analysis. Plasma glucose was measured using glucose oxidase method (Agappe Diagnostics Limited, India). Plasma insulin was assayed using a commercially available human insulin enzyme-linked immunosorbent assay (ELISA) kit (Monobind Inc. USA). All laboratory analyses were done at the Chemical Pathology laboratory of Gombe State University/Federal Teaching Hospital, Gombe.

Estimation of dietary acid intake and pancreatic β-cell function

The dietary acid intake for each subject was determined using the Potential Renal Acid Load (PRAL) score. The PRAL score was calculated using the following equation:[24]

PRAL (mEq/day) = 0.4888 × dietary Protein (g/day) + 0.0366 × dietary Phosphorus (mg/day) − 0.0205 × dietary Potassium (mg/day) − 0.0125 × dietary Calcium (mg/day) − 0.0263 × dietary Magnesium (mg/day).[24]

PRAL is an estimate of the capacity of any food to release acids or alkalis to the body. Food items with positive PRAL score increase the generation of acid precursors and those food items with negative PRAL scores increase the generation of alkali precursors in the body.[24]

Pancreatic β-cell function was determined by the Homeostasis Model Assessment-Beta (HOMA-β) index.

HOMA-β = [20 × Fasting Plasma Insulin (μU/mL)] / [Fasting Plasma Glucose (mmol/L) - 3.5][25]

Insulin resistance was calculated as Homeostatic Model Assessment-Insulin Resistance (HOMA-IR);

HOMA-IR = Fasting Plasma Insulin (mU/mL) × Fasting Plasma Glucose (mmol/L) / 22.5[25]

Insulin resistance was defined as HOMA-IR ≥ 2.0[26]

Statistical analysis

Statistical analysis was done using the Statistical Package for Social Sciences (SPSS) software version 20.0. Quantitative variables (age, duration of diabetes, body mass index, blood pressure, PRAL score, fasting plasma glucose, fasting plasma insulin, HOMA-IR, HOMA-β and dietary intakes of energy, protein, calcium, magnesium, phosphorus and potassium) were presented using measures of central tendency and dispersion and their mean values were compared across the PRAL quartiles categories using the ANOVA test. We considered all the p-values (two-sided) as significant if less than 0.05.

Results

Demographic and dietary parameters of the study subjects are given in Table 1. The study included one hundred and ninety-two (192) type 2 diabetic subjects categorized in to four quartiles according to their median dietary acid intake. The median PRAL scores in first, second, third and fourth quartiles were -21.5mEq/day, +6.6mEq/day, +12.2mEq/day and +55.0mEq/day respectively. Mean duration of type 2 diabetes was not significantly different across the quartiles (*pfor trend* > *0.05*). The mean age and body mass index of the study subjects were also not observed to be significantly different across the study groups (*p for trend* > *0.05*).

The relationship between dietary acid intake and degree of pancreatic β-cells function as measured by HOMA-β was examined (Table 2, Figures 1 and 2). Subjects in the higher quartiles of PRAL score had a significantly higher intake of proteins, calcium and phosphorus compared to those in the lower quartiles (*p for all* < *0.05*), while subjects in the lower PRAL quartiles consumed significantly higher energy, potassium and magnesium than the subjects in the higher PRAL quartiles (*p for all* < *0.05*).

Degree of pancreatic β-cells function was observed to be significantly lower in subjects in the highest quartile of the PRAL compared to those in the lower quartiles (*p for trend* < *0.05*). There was a statistically significant trend with higher intake of dietary acid associated with increased degree of insulin resistance among the study subjects (*p for trend*< 0.05). Dietary acid load was found to be a significant predictor of pancreatic β-cells function, as measured by HOMA-β, among the study subjects.

Table 1: Characteristics of the study subjects across the quartiles of PRAL scores

	Dietary PRAL Scores				
	Q1 (m ± SD)	**Q2** (m ± SD)	**Q3** (m ± SD)	**Q4** (m ± SD)	***p*-value**
Sample size (n)	48	48	48	48	
Age (years)	53.4 ± 5.3	54.3 ± 5.2	54.8 ± 5.3	52.9 ± 5.3	0.318
Sex ratio (Male/Female)	27/21	27/21	28/20	24/24	-
Duration of type 2 DM (years)	5.4 ± 1.5	5.2 ± 1.6	5.4 ± 1.4	5.3 ± 1.4	0.900
Body Mass Index (kg/m²)	23.4 ± 3.4	23.6 ± 4.0	24.7 ± 3.7	25.0 ± 4.1	0.111
Systolic BP (mmHg)	120 ± 5.4	118 ± 6.0	118 ± 5.8	120 ± 3.3	0.219
Diastolic BP (mmHg)	77 ± 4.5	79 ± 3.7	79 ± 3.0	79 ± 3.7	0.051
PRAL (mEq/day)*	-21.5	+6.6	+12.2	+55.0	0.000#
Fasting PG (mmol/L)	10.4 ± 2.3	10.0 ± 2.3	10.4 ± 2.5	12.9 ± 1.1	0.000
Plasma Insulin (mU/mL)	14.5 ± 5.3	13.2 ± 4.5	13.8 ± 5.7	18.2 ± 4.2	0.000

m, mean
SD, standard deviation
DM, diabetes mellitus
BP, blood pressure
PG, plasma glucose
PRAL, potential renal acid load
*Median values
#Kruskal-Wallis test
Data are presented as proportion or m ± SD.

Table 2: Energy and Nutrient Intake of the study subjects across the quartiles of PRAL scores

	Dietary PRAL Scores				
	Q1 (m ± SD)	**Q2** (m ± SD)	**Q3** (m ± SD)	**Q4** (m ± SD)	***p*-value**
Sample size (n)	48	48	48	48	
Energy (kcal/day)	1957 ± 556	2395 ± 192	2410 ± 177	1888 ± 630	0.000
Protein (g/day)	74.6 ± 20.9	89.8 ± 7.2	90.4 ± 6.6	112.8 ± 21.5	0.000
Calcium (mg/day)	446 ± 182	575 ± 106	543 ± 133	604 ± 45	0.000
Phosphorus (mg/day)	819 ± 211	973 ± 78	979 ± 72	1206 ± 217	0.000
Potassium (mg/day)	3488 ± 738	3144 ± 679	2898 ± 622	2944 ± 614	0.000
Magnesium (mg/day)	1357 ± 167	1002 ± 564	480 ± 582	236 ± 97	0.000
HOMA-β	41.9 ± 5.5	40.8 ± 6.0	39.9 ± 6.5	38.6 ± 6.5	0.047
HOMA-IR	2.9 ± 1.7	2.5 ± 1.4	2.8 ± 1.7	4.3 ± 1.2	0.000

m, mean
SD, standard deviation
PRAL, potential renal acid load
HOMA-IR, Homeostasis Model Assessment-Insulin Resistance
HOMA-β, Homeostasis Model Assessment-Beta
Data are presented as m ± SD.

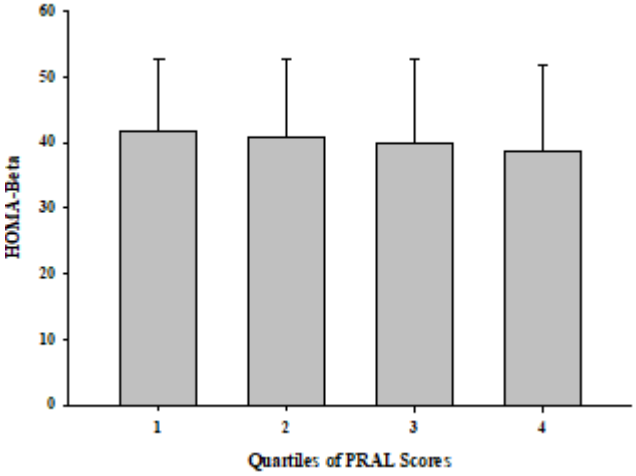

Figure 1: HOMA-β of the study subject across the quartiles of PRAL Scores.
Mean and standard deviation are shown.
HOMA-β, Homeostasis Model Assessment-Beta

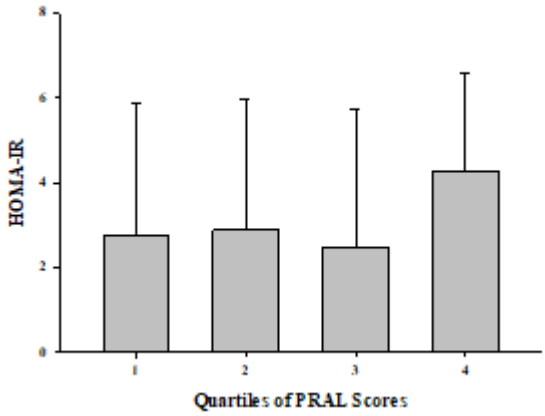

Figure 2: HOMA-IR of the study subject across the quartiles of PRAL Scores.
Mean and standard deviation are shown.
HOMA-IR, Homeostasis Model Assessment-Insulin Resistance

Discussion

In this present study, we investigated the potential relationship between consumption of diet with high acid forming potential, as determined by dietary acid load score (PRAL), and the indices of insulin secretion (as determined by HOMA-β) and resistance (as determined by HOMA-IR) in a group of patients with uncomplicated type 2 diabetes mellitus at a specialist hospital in north eastern Nigeria.

We observed that HOMA-β is significantly lower among subjects in the group with highest intake of dietary acid compared to those in the group with lowest intake of dietary acid. We also found that lower dietary acid load scores is associated with increased HOMA-βamong the study subjects independent of total energy intake, age, gender, duration of diabetes mellitus and body weight status. Degree of insulin resistance was observed to be higher in subjects in the highest quartile of the PRAL compared to those in the lower quartiles.

Our results are in line with reports from previous studies wherein indices of dietary acid load were found to be significant predictors of pancreatic beta cell dysfunction and insulin resistance.[13-16] In addition, Gæde et al observed, in a prospective study conducted among cohort of Danish men and women, that consumption of acidic diets is associated with a higher risk of diabetes mellitus.[17]

However, Xu et al, in a prospective study involving elderly Swedish men, did not find a significant relationship between dietary acid load, pancreatic β-cells function and insulin sensitivity.[18] Furthermore, recent findings from a systematic review and meta-analysis of studies by Dehghan et al, also suggested no significant association between indices of dietary acid load and degree of insulin resistance.[19] Also, adding to the controversy, Harris et al[20] and Kozan et al[21] reported that alkalizing treatment has no effect on insulin sensitivity following high acid load meal in healthy individuals.[20,21]

Heterogeneity of the study participants, including differences in age, gender proportion and body weight status, and differences in the assay methods used in analysis of plasma insulin levels and the definition of insulin resistance used in the various studies might explained the inconsistencies.

Potential mechanisms by which diet induced low grade metabolic acidosis could contribute to the development of pancreatic β-cells dysfunction and insulin resistance have been proposed. Binding affinity of insulin to insulin receptors is decreased in acidosis[27] and while in pancreatic β-cells, glycolysis critically regulates insulin secretion by coupling glucose-stimulated insulin secretion, acidosis inhibits the expression of the glycolytic enzymes and switches the cellular metabolism from glycolysis to other metabolic pathways, thereby reducing insulin release.[8,28,29] Also, the controlling mechanism that regulates the recycling of insulin receptors and insulin

is dependent on the cellular hydrogen ion concentration [H^+]. Therefore, extracellular acidosis, which is associated with changes in the lysosomal pH, could affect secretion of insulin and sensitivity of tissues to insulin.[8,30,31] Extracellular acidosis is also known to be associated with inflammation.[32] Increased transcription and expression of inflammatory cytokines including TGF-β, is associated with decreased tissue sensitivity to insulin and could cause pancreatic β-cells dysfunction.[33-35]

Conclusion

We conclude that among subjects with type 2 diabetes mellitus in this study, consumption of a diet loaded with high acid forming potential food items was associated with greater insulin resistance and lower insulin secretion ability. Poor control of dietary acid load might be an additive mechanism contributing to deterioration of glycaemic control in type 2 diabetic patients in our setting. Further interventional studies are required to determine whether specific dietary interventions to control dietary acid intake may be a useful and achievable approach to improve pancreatic β-cells function, insulin resistance and glycaemic control among type 2 diabetics in our setting. We also recommend further studies in other ethnic groups with different dietary habits to validate these findings.

Limitations

The study is cross-sectional in nature; therefore, we cannot be certain of causality. Also, HOMA-β and HOMA-IR which are surrogate markers of insulin secretion and resistance respectively were used in the study rather than the gold standard methods that precisely measures insulin secretion and resistance. The degree to which these might have influenced the findings of this study requires further investigation. The assessment of dietary intake was done using a self-reported food frequency questionnaire which might be affected by recall bias.

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Conflict of interest

Nil

The Health Research Ethics Committee of the Gombe State Ministry of Health, Gombe, Nigeria approved the study protocol.

References

1. WHO Global Report on Diabetes. Geneva; World Health Organization; 2016.
2. Fasanmade OA, Dagogo-Jack S. Diabetes Care in Nigeria. *Annals of Global Health.* 2015; 81(6): 821 – 829.
3. Oputa RN, Chinenye S. Diabetes in Nigeria – a translational medicine approach. *African Journal of Diabetes Medicine.* 2015; 23: 7 – 10.
4. U.K. prospective diabetes study 16. Overview of 6 years' therapy of type II diabetes: a progressive disease. U.K. Prospective Diabetes Study Group. *Diabetes.* 1995; 44: 1248 – 1258.
5. Kahn SE. The relative contributions of insulin resistance and beta-cell dysfunction to the pathophysiology of Type 2 diabetes. *Diabetologia.* 2003; 46: 3 – 19.
6. Yoshifumi Saisho. β-cell dysfunction: Its critical role in prevention and management of type 2 diabetes. *World J Diabetes.* 2015; 6(1): 109 – 124.
7. Adeva MM, Souto G. Diet-induced metabolic acidosis. *Clin Nutr.* 2011; 30: 416 – 421.
8. Baldini N, Avnet S. The Effects of Systemic and Local Acidosis on Insulin Resistance and Signaling.*Int. J. Mol. Sci.*2019; 20: 126.
9. Rebolledo OR, Hernandez RE, Zanetta AC, Gagliardino JJ. Insulin secretion during acid-base alterations. *Am J Physiol.* 1978; 234: E426 – E429.
10. Mak, R.H. Effect of metabolic acidosis on insulin action and secretion in uremia. *Kidney Int.*1998; 54: 603 –607.
11. Nabe K, Fujimoto S, Shimodahira M, Kominato R, Nishi Y, Funakoshi S, *et al.* Diphenylhydantoin suppresses glucose-induced insulin release by decreasing cytoplasmic H+ concentration in pancreatic islets. *Endocrinology.*2006; 147: 2717 – 2727.
12. Gunawardana SC, Rocheleau JV, Head WS, Piston DW. Nutrient-stimulated insulin secretion in mouse islets is critically dependent on intracellular pH. *BMC Endocr. Disord.* 2004; 4: 1.
13. Akter S, Eguchi M, Kuwahara K, Kochi T, Ito R, Kurotani K, *et al.* High dietary acid load is associated with insulin resistance: The Furukawa

Nutrition and Health Study. *Clin. Nutr.*2016; 35: 453 – 459.
14. Lee KW, Shin D. Positive association between dietary acid load and future insulin resistance risk: findings from the Korean Genome and Epidemiology Study. *Nutr J*. 2020; 19:137.
15. Moghadam SK, Bahadoran Z, Mirmiran P, Tohidi M, Azizi F. Association between Dietary Acid Load and Insulin Resistance: Tehran Lipid and Glucose Study. *Prev. Nutr. Food Sci.* 2016; 21(2): 104 – 109.
16. Emamat H, Tangestani H, Bahadoran Z, Khalili-Moghadam S, Mirmiran P. The associations of dietary acid load with insulin resistance and type 2 diabetes: a systematic review of existing human studies. Recent Pat Food Nutr Agric. 2019; 10(1): 27 – 33.
17. Gæde J, Nielsen T, Madsen ML, Toft U, Jørgensen T, Overvad K, *et al.* Population-based studies of relationships between dietary acidity load, insulin resistance and incident diabetes in Danes. *Nutr J*. 2018; 17: 91.
18. Xu H, Jia T, Huang X, Risérus U, Cederholm T, Arnlöv J, *et al.* Dietary acid load, insulin sensitivity and risk of type 2 diabetes in community-dwelling older men. *Diabetologia*.2014; 57:1561 – 1568.
19. Dehghan P, Abbasalizad Farhangi M. Dietary acid load, blood pressure, fasting blood sugar and biomarkers of insulin resistance among adults: Findings from an updated systematic review and meta-analysis. *Int J Clin Pract*. 2020; 74: e13471.
20. Harris SS, Dawson-Hughes B. No effect of bicarbonate treatment on insulin sensitivity and glucose control in non-diabetic older adults. *Endocrine*.2010; 38: 221 – 226.
21. Kozan P, Blythe JC, Greenfield JR, Samocha-Bonet D. The Effect of Buffering High Acid Load Meal with Sodium Bicarbonate on Postprandial Glucose Metabolism in Humans – A Randomized Placebo-Controlled Study. *Nutrients*.2017; 9: 861.
22. Delisle H. Findings on dietary patterns in different groups of African origin undergoing nutrition transition.*Appl. Physiol. Nutr. Metab.* 2010; 35: 224 – 228.
23. Nigeria Food Composition Available from http://nigeriafooddata.ui.edu.ng/Database.
24. Remer T, Dimitriou T, Manz F. Dietary potential renal acid load and renal net acid excretion in healthy, free-living children and adolescents. *Am J Clin Nutr.* 2003; 77:1255 – 1260.
25. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Truner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia.* 1985; 28: 412 – 419.
26. Oli JM, Adeyemo AA, Okafor GO, Ofoegbu EN, Onyenekwe B, Chukwuka CJ, *et al.* (2009) Basal insulin resistance and secretion in Nigerians with type 2 diabetes mellitus. *Metab Syndr Relat Disord*. 7: 595-599.
27. Whittaker J, Cuthbert C, Hammond VA, Alberti KG. The effects of metabolic acidosis in vivo on insulin binding to isolated rat adipocytes. *Metabolism.*1982; 31(6): 553 – 557.
28. Komatsu M, Takei M, Ishii H, Sato Y. Glucose-stimulated insulin secretion: A newer perspective. *J Diabetes Investig*. 2013; 4(6): 511 – 516.
29. Bevington A, Walls J. Protein catabolism in metabolic acidosis: inhibition of glycolysis by low pH suggests a role for glucose. *Biochem Soc Trans.* 1995; 23(3): 464S.
30. Glunde K, Guggino SE, Solaiyappan M, Pathak AP, Ichikawa Y, Bhujwalla ZM. Extracellular acidification alters lysosomal trafficking in human breast cancer cells. *Neoplasia*. 2003; 5(6): 533 – 545.
31. Haeusler RA, McGraw TE, Accili D. Biochemical and cellular properties of insulin receptor signalling. *Nat Rev Mol Cell Biol.*2018; 19(1): 31 – 44.
32. Casimir GJ, Lefèvre N, Corazza F, Duchateau J, Chamekh M. The Acid–Base Balance and Gender in Inflammation: A Mini-Review. *Front. Immunol.* 2018; 9: 475.
33. Dhawan S, Dirice E, Kulkarni RN, Bhushan A. Inhibition of TGF-b Signaling Promotes Human Pancreatic b-Cell Replication. *Diabetes.* 2016; 65: 1208 – 1218.
34. Lin HM, Lee JH, Yadav H, Kamaraju AK, Liu E, Zhigang D, *et al.* Transforming Growth Factor-β/Smad3 Signaling Regulates Insulin Gene Transcription and Pancreatic Islet β-Cell Function. *J Biol Chem.* 2009; 284(18): 12246 – 12257.
35. Heydarpour F, Sajadimajd S, Mirzarazi E, Haratipour P, Joshi T, Farzaei MH, *et al.* Involvement of TGF-β and Autophagy Pathways in Pathogenesis of Diabetes: A Comprehensive

Review on Biological and Pharmacological Insights. *Front. Pharmacol.* 2020; 11: 498758.

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