International Journal of
Active & Healthy Aging

e-ISSN :3023-6045
https://ndpapublishing.com/index.php/ijaha

Analysis of Inorganic Blood Element Fluctuations in the Context of Hyperglycemia Risk Factors

Mahira Firudin Kizi Amirova[1] and Ellada Eldar Qizi Huseynova[1]

[1]*Azerbaijan Medical University, Biochemistry Department, Azerbaijan*

Article Info
Received: 22 February 2025
Revised: 04 April 2025
Accepted: 26 May 2025
Published: 30 June 2025

Keywords
Hyperglycemia
Iron
Calcium
Magnesium
Microelements

ABSTRACT

Hyperglycemia (HG) is a prevalent condition among patients, necessitating continued efforts to identify effective therapeutic interventions that can improve population health outcomes. Given the extensive side effects associated with chemical pharmacological treatments, scientific research has increasingly focused on biological compounds that align more closely with normal physiological metabolism rather than the metabolism of xenobiotics. In this context, contemporary medicine has turned its attention to the role of macro- and micronutrients, which have the potential to stabilize the microbiota, the primary regulator of metabolic processes, in line with a pathogenetic approach to hyperglycemia. The association between macro- and microelements and various etiological factors of hyperglycemia has been an area of active investigation. Within this framework, the role of inorganic blood elements in the development and progression of hyperglycemia is of particular interest. Our **study aims** to investigate the underlying reasons for discrepancies observed in the literature concerning the impact of micro- and macroelements in hyperglycemia. For this, a **retrospective analysis** was conducted on 2024 biochemical laboratory data from the Azerbaijan Medical University Teaching Surgery Clinic. Our **findings** reveal a statistically significant inverse correlation between hyperglycemia and serum calcium and magnesium levels. Notably, we present **pioneering** evidence that changes in the concentrations of inorganic blood elements were significant (P=0.01 and 0.001 for Ca and Mg, respectively) despite remaining within the generally accepted normal reference ranges.

1. INTRODUCTION

Hyperglycemia (HG) is a multifaceted pathological condition associated with a wide spectrum of systemic complications, including damage to erythrocytes [1], inflammation and oxidative stress of T-lymphocytes [2], duodenal dysbiosis [3], the development of intracranial meningiomas [4], etc. Beyond its direct impact on patient health, hyperglycemia poses significant challenges in the context of surgical interventions [5,6], often exacerbating postoperative complications and leading to long-term disease burden [7].

Despite the widespread use of chemical hypoglycemic agents [8, 9, 10], their therapeutic application is often constrained by adverse effects, including the risk of neonatal hypoglycemia [11]. Consequently, the search for safer and more physiologically compatible approaches to glycemic control remains an urgent priority in contemporary medicine. Emerging strategies include the exploration of polyphenols [12], selenium polysaccharides [13], bioactive compounds from sea cucumber gonads [14], and mulberry leaf extracts [15], among others.

Intriguingly, reports of hyperglycemic episodes following calcium channel blocker administration [16] have prompted hypotheses regarding a potential interrelationship between glycemic homeostasis and the balance of micro- and macroelements in the body [17]. Recent studies have identified subtle but significant correlations between trace element proportions and blood glucose levels [18]. Extending this line of inquiry, some researchers have examined the impact of elemental concentrations on postprandial glucose reduction dynamics [19], while others have observed fluctuations in serum potassium levels

*Corresponding author
e-mail: gerayelmira@gmail.com
ORCID ID: 0000-0001-5598-6995

How to cite this article
Amirova, A. F. K., and Huseynova, E. E. Q. (2025). Analysis of Inorganic Blood Element Fluctuations in the Context of Hyperglycemia Risk Factors. Int. J. Act. Health Aging, 3(1), 19-24

during glucose tolerance testing [20]. These findings collectively suggest a complex interplay between glycemic regulation and inorganic element homeostasis.

Given these observations, we aim to investigate the degree and nature of the relationship between glycemia and the concentrations of inorganic elements in the bloodstream. By elucidating these interactions, our study seeks to provide novel insights into the potential role of micro- and macroelement balance in glycemic control and metabolic stability.

The primary objective of this study is to assess the variations in key microelements (Ca, Mg, and Fe) blood serum levels in hyperglycemic individuals, analyzing their statistical correlations with glucose concentrations. By identifying these fluctuations, we aim to elucidate potential dietary or therapeutic interventions to mitigate hyperglycemia- associated risks.

2. MATERIALS AND METHODS

2.1. Participant

The average age of the examined patients was 49.52±17.53. All patients were stratified to 2 groups: with normal glucose levels (control group) and hyperglycemic (II group).

2.2. Biochemical Analysis

A retrospective analysis was performed on biochemical laboratory data obtained in 2024 from the Teaching Surgery Clinic of Azerbaijan Medical University (AMU). Ca, P, and Fe were measured using Clinical Chemistry reagents for semi auto chemistry analyzer (Mindray BA-88A). The glucose assay was conducted using the ortho-toluidine method.

2.3. Statistical Analysis

Statistical analysis was conducted using Microsoft Excel to evaluate correlations and regression trends between serum glucose and mineral concentrations. A significance threshold of $p < 0.05$ was considered statistically significant.

3. RESULTS

Data from 188 patients were subjected to statistical analysis. The study cohort had a mean age of 57.7 ± 12.32 years. All demographic data are presented in Tables 1 and 2. The significance of differences in macroelement blood concentrations between normoglycemic (NG, N=30) and hyperglycemic (HG, N=158) patients, within established reference ranges, was evaluated.

Table 1. Demographic characteristics of the Hyperglycemic (HG) participants

Parameters	Young	Adult	Elderly	
Women	41	28	25	94 (59.5%)
Men	17	23	21	64 (40.5%)
Total	58	51	46	158

Remarkably, across all hyperglycemic age groups (especially youth), the number of women exceeded that of men.

Table 2. Height and weight (M ± SD, min–max) of Hyperglycemic (HG) patients

Age Group	Sex	Height (cm)	Weight (kg)
Young	Women	140–160	40–60
	Men	150-170	50-75
Adult	Women	165–175	70–90
	Men	165-180	80-95
Elderly	Women	160–170	65–80
	Men	162-176	66-87

Table 4. Blood macroelements fluctuations in hyperglycemic (HG) patients with an assessment of the statistical significance of changes

Parameter	Sample size	FG, mg/dL	Ca, mg/dL	Mg, mg/dL	Fe, μг/дл
Group NG	30	83±16.67 (70;100)	9.75±0.5 (8.91;10.71),	2.16+/-0.29 (1.81;2.77)	85.9+/-45.11 (31;199.28)
Group HG	158	136± 55.66 (102; 300)	9.3±0.68 (8.14;10.6)	1.93+/-0.2 (1.33;2.15),	92.36+/-32.5 (18;144),
P		0.001	0.01	0.001	0.29

Note. FG – Fasting glucose, NG – normoglycemia, HG – hyperglycemia. The *p*-value was determined for the comparison between the hyperglycemic (HG) and normoglycemic (NG) groups

The findings presented suggest that although the calcium levels in patients with HG did not exceed the reference range, there was a notable discrepancy in the variability of blood calcium when compared to the control group with normal glucose levels.

Specifically, the distribution of calcium levels in the NG group exhibited a standard deviation of 0.5 mg/dL, indicating relatively tighter clustering around the mean value of 9.75 mg/dL. In contrast, patients with HG showed a wider spread in their calcium levels, with a standard deviation of 0.68 mg/dL and an average of 9.3 mg/dL (Table 4).

Magnesium, an essential cofactor in glucose metabolism, exhibited a notable reduction in hyperglycemic individuals (2.84 ± 0.21 mmol/L) compared to normoglycemic subjects (2.99 ± 0.29 mmol/L). These results reinforce previous studies linking Mg deficiency to insulin resistance and impaired glucose regulation.

Interestingly, mean Fe levels were similar between normoglycemic (85.93 ± 45.11 µg/dL) and hyperglycemic (84.37 ± 39.89 µg/dL) groups. However, the lowest Fe concentration in hyperglycemic individuals (30.81 µg/dL) was higher than that in normoglycemic individuals (24.09 µg/dL). Despite this, both values remained below the reference normal threshold of 37 µg/dL. Moreover, a positive correlation between Fe and glucose levels was identified, suggesting that increased iron levels may contribute to higher blood glucose concentrations. This finding is consistent with emerging evidence linking iron overload to oxidative stress and insulin resistance, although the precise mechanistic pathways remain under investigation.

Based on the results obtained, it can be concluded that our study highlights the fluctuations in macronutrient concentrations within normal reference ranges in hyperglycemia, a factor of critical importance when prescribing supplements to hyperglycemic patients.

4. DISCUSSION

A significant inverse linear correlation was observed between serum Ca and glucose levels, indicating that lower Ca concentrations were associated with elevated blood glucose levels. This finding aligns with existing literature suggesting that calcium signaling is crucial for insulin secretion and glucose homeostasis.

The findings presented suggest that although the calcium levels in patients with HG did not exceed the reference range, there was a notable discrepancy in the variability of blood calcium when compared to the control group with normal glucose levels. Specifically, the distribution of calcium levels in the NG group exhibited a standard deviation of 0.5 mg/dL, indicating relatively tighter clustering around the mean value of 9.75 mg/dL. In contrast, patients with HG showed a wider spread in their calcium levels, with a standard deviation of 0.68 mg/dL and an average of 9.3 mg/dL.

Despite both groups presenting calcium values that fell within the accepted reference range, the statistical analysis revealed a significant difference between these two groups. A p-value of 0.01 at a 97% confidence level (Z-score = -2.3) indicates that the calcium levels in the HG group are significantly lower, even though they remain within the typical reference limits.

This observation suggests that the commonly accepted reference ranges for blood calcium may be too broad and may mask subtle but clinically significant fluctuations in calcium levels, particularly in the context of HG. The wider spread in calcium levels within the HG group implies that the typical "normal" range for calcium may fail to accurately capture the true physiological state of patients with hyperglycemia. This finding warrants further investigation into whether the current reference ranges for calcium are appropriate for individuals with conditions like hyperglycemia, where underlying biochemical changes may not be sufficiently reflected in standard laboratory measurements. While both the NG and HG groups exhibited calcium levels within the standard reference range, the significant statistical difference between these groups supports the hypothesis that the current laboratory reference ranges for blood calcium may not adequately reflect subtle yet important fluctuations in calcium homeostasis in patients with HG. Therefore, it may be necessary to reconsider the adequacy of these reference ranges in HG to better detect and understand the underlying biochemical shifts in patients with disordered metabolic conditions.

The observed reduction in serum magnesium (Mg) levels in hyperglycemic individuals (2.84 ± 0.21 mmol/L) compared to normoglycemic subjects (2.99 ± 0.29 mmol/L) provides further support for the growing body of literature linking magnesium deficiency to impaired glucose metabolism and insulin resistance. Magnesium plays a pivotal role in several biochemical processes, including those involved in glucose regulation, insulin signaling, and cellular energy production [21]. It serves as a cofactor for enzymes involved in glucose transport, glycolysis, and the activation of insulin receptors, which are crucial for maintaining normal glucose homeostasis.

Magnesium deficiency has been increasingly recognized as a key player in the pathophysiology of type 2 diabetes and other metabolic disorders. A growing body of evidence suggests that low magnesium levels may impair the function of insulin receptors and reduce the efficiency of insulin action, thereby promoting insulin resistance

[22]. Insulin resistance is a hallmark of hyperglycemia and is characterized by the diminished ability to respond to insulin, leading to elevated blood glucose levels. As a result, the decrease in magnesium levels observed in the HG cohort aligns with the concept that magnesium deficiency may exacerbate the pathogenesis of glucose dysregulation by further impairing insulin sensitivity.

Moreover, magnesium is essential for maintaining intracellular calcium homeostasis, and imbalances in these ions can disrupt insulin secretion from pancreatic β-cells and insulin action in peripheral tissues such as muscle and adipose tissue. It is hypothesized that magnesium deficiency may contribute to inflammation, alterations in calcium signaling, further compromising glucose metabolism and exacerbating the cycle of hyperglycemia and insulin resistance [23, 24].

Several clinical studies have reported an inverse relationship between serum magnesium levels and the risk of developing insulin resistance and type 2 diabetes [25]. For instance, lower magnesium intake has been associated with an increased risk of insulin resistance and elevated fasting glucose levels, which are key indicators of impaired glucose metabolism. These findings suggest that magnesium supplementation might have therapeutic potential for improving insulin sensitivity and glycemic control, particularly in individuals at risk of metabolic dysfunction.

The reduction in magnesium levels observed in HG individuals could also reflect a maladaptive response to chronic hyperglycemia [26]. Prolonged high blood glucose levels may increase renal magnesium excretion [27], leading to a depletion of extracellular magnesium stores, further exacerbating the deficiency. The interplay between magnesium depletion and glucose dysregulation thus appears to be cyclical, where magnesium deficiency may impair glucose metabolism, and in turn, hyperglycemia may perpetuate magnesium loss, potentially accelerating the progression of metabolic disorders. The lower magnesium levels observed in individuals with HG strengthen the hypothesis that magnesium deficiency contributes to the pathogenesis of insulin resistance and impaired glucose regulation. These findings emphasize the importance of magnesium as an essential nutrient in glucose metabolism and highlight the need for further research into the therapeutic potential of magnesium supplementation in preventing or managing hyperglycemia and associated metabolic disorders. Moreover, these results underline the importance of considering micronutrient status, including Mg, in the context of metabolic health, as it may provide a crucial therapeutic target for improving insulin sensitivity and overall glucose homeostasis in individuals with or at risk for HG and type 2 diabetes.

The observed data indicating that mean serum iron (Fe) levels in both normoglycemic (85.93 ± 45.11 µg/dL) and hyperglycemic (84.37 ± 39.89 µg/dL) individuals were similar, but with a higher minimum Fe concentration in hyperglycemic individuals (30.81 µg/dL) compared to normoglycemic individuals (24.09 µg/dL), presents an intriguing paradox. Although both groups have Fe bottom concentrations that fall below the typical reference threshold of 37 µg/dL, the broader distribution of Fe values and the observed positive correlation between Fe and glucose levels warrant further investigation into the role of iron metabolism in glucose dysregulation and insulin resistance.

The results suggest that while the average iron levels in HG and NG individuals are comparable, the distribution of Fe within the HG group demonstrates variability that could reflect more complex alterations in iron homeostasis associated with HG. Despite this variability, the fact that both groups bottom Fe fall below the lower reference range could indicate that suboptimal iron status is present across the board, which is of particular interest because iron plays an essential role in a variety of biological processes, including oxygen transport, mitochondrial function, and cellular metabolism.

The positive correlation between Fe and blood glucose levels observed in this study is especially noteworthy. This finding aligns with growing evidence suggesting that iron overload, particularly in tissues such as the liver, heart, and pancreas, can contribute to the development of insulin resistance. Iron accumulation in cells leads to the generation of reactive oxygen species, a hallmark of oxidative stress. Oxidative stress, in turn, is known to impair insulin signaling pathways [28], leading to reduced insulin sensitivity and the promotion of hyperglycemia.

Moreover, iron-induced oxidative stress can negatively impact pancreatic β-cell function [29], further compromising insulin secretion. In hyperglycemic individuals, iron overload may exacerbate the already elevated oxidative stress, leading to a vicious cycle where excess iron contributes to insulin resistance, which in turn may worsen the glycemic profile.

While the precise pathways linking iron overload and insulin resistance remain under investigation, several studies have suggested that increased tissue iron levels may alter the activity of key signaling molecules involved in glucose

metabolism, such as AMP-activated protein kinase (AMPK) and protein kinase C (PKC) [30, 31], both of which are involved in insulin action and glucose uptake. Additionally, iron accumulation can affect the function of antioxidant systems within the body, impairing the ability to neutralize reactive oxygen species and thereby exacerbating the inflammatory response that is often observed in insulin resistance [32].

Interestingly, even though iron levels did not exceed the reference normal threshold, the presence of a higher minimum Fe concentration in the HG vs NG group suggests that iron metabolism could still play a role in influencing glucose regulation, even within the bounds of what is considered normal for the population. This suggests that more subtle alterations in iron homeostasis may be at play, warranting further investigation into how even relatively modest deviations in iron levels might contribute to metabolic dysfunction. Despite the fact that the mean iron levels in NG and HG groups were similar, the positive correlation between iron and glucose levels supports the hypothesis that dysregulation of iron metabolism may play a role in the development of insulin resistance and hyperglycemia[33]. Further research is needed to elucidate the mechanistic pathways by which iron overload leads to oxidative stress and impairs insulin action. Understanding these processes may open new avenues for therapeutic interventions aimed at modulating iron levels or mitigating oxidative stress to improve glucose control in individuals with metabolic disorders such as HG.

5. Conclusion

Our study confirms a statistically significant negative correlation between hyperglycemia and serum Ca and Mg levels, reinforcing their potential roles in glucose metabolism and insulin function. Conversely, while Fe levels did not exhibit a clear differentiation between normoglycemic and hyperglycemic subjects, a positive correlation with fasting glucose suggests that iron metabolism may influence glycemic control. Further research with larger sample sizes and mechanistic studies is required to elucidate the clinical implications of these mineral fluctuations and their potential utility in managing hyperglycemia.

Findings

Both Ca and Mg levels significantly drop in HG, yet remaining within the normal reference range. This suggests that HG may influence these elements levels, or conversely, that these elements may have an impact on HG levels.

Conflict of Interest

No conflict of interest is declared by tehe authors. In addition, no financial support was received.

Ethics Committee

The study was conducted retrospectively and, as such, did not require prior approval from an Ethics Committee.

Author Contributions

Conception and design of the study: EEQH; Data collection: EEQH; Data analysis: MFKA; Data Interpretation: MFKA; Drafting the article and/or its critical revision: MFKA, EEQH; All authors have read and agreed to the published version of the manuscript.

REFERENCES

1. Alamri, B. N., Bahabri, A., Aldereihim, A. A., Alabduljabbar, M., Alsubaie, M. M., Alnaqeb D., Almogbel, E., Metias, N. S., Alotaibi, O. A., & Al-Rubeaan, K. (**2019**). Hyperglycemia effect on red blood cells indices. *Eur Rev Med Pharmacol Sci*, 23(5), 2139-2150. [CrossRef] [PubMed]
2. Stentz, F. B. (**2021**). Hyperglycemia- and Hyperlipidemia-Induced Inflammation and Oxidative Stress through Human T Lymphocytes and Human Aortic Endothelial Cells (HAEC). *Intech Open*, 598-606. [CrossRef]
3. Darra, A., Singh, V., Jena, A., Popli, P., Nada, R., Gupta, P. et al. (**2023**). Hyperglycemia is associated with duodenal dysbiosis and altered duodenal microenvironment. *Sci Rep,* 13, 11038 [CrossRef]
4. Orešković, D., Madero Pohlen, A., Cvitković, I. *et al.*, (**2024**). Chronic hyperglycemia and intracranial meningiomas. *BMC Cancer,* 24, 488. [CrossRef]
5. Mamadova, V., M., K., Abdullaeva, A., M., K., Amirova, M., F., K., & Nasirova, V., B., K. (**2024**). A novel treatment for ptosis complication after preseptal cellulitis in diabetic patient. *J of Law and sustainable development*. Miami12(10): 01-13/e03847. [CrossRef]
6. Morales J, Schneider D. (**2014**). Hypoglycemia. The American Journal of Medicine, *Am J Med*, 127(10 Suppl):S17-24. [CrossRef] [PubMed]
7. Martin ET, Kaye KS, Knott C, Nguyen H, Santarossa M, Evans R, Bertran E, Jaber L. (**2016**). Diabetes and Risk of Surgical Site Infection: A Systematic Review and Meta-analysis. *Infect Control Hosp Epidemiol*, 37(1):88-99. [CrossRef] [PubMed]
8. Tyurenkov I. N., Kurkin D. V., Bakulin D. A., Volotova E. V., Morkovin E. I., Chafeev M. A. et al., (2018). Chemistry and Hypoglycemic Activity of GPR119 Agonist ZB-16. *Frontiers in Endocrinology*, 9:543. [CrossRef] [PubMed]
9. Roberta B, and Corsini A. (**2011**). Pharmacology of dipeptidyl peptidase-4 inhibitors." *Drugs*, 71(11), 1441-1467. [CrossRef] [PubMed]
10. Farmanova N T, Farmanov Sh I, & Kadirov M A. (**2017**). Pharmacological activity and studying of the chemical compound of hypoglycemic gathering. *Austrian Journal of Technical and Natural Sciences*, (3-4), 37-41.

11. Khangholi, S., Abdul Majid F.A., Ahmed Berwary N.J., Ahmad, F., & Bin Abd Aziz, R. (**2016**). The Mechanisms of Inhibition of Advanced Glycation End Products Formation through Polyphenols in Hyperglycemic Condition. *Planta Med*, 82(01/02): 32-45. [CrossRef] [PubMed]
12. Ramos, G. A., Hanley, A. A., Aguayo, J., Warshak, C. R., & Kim, J. H., (**2012**). Neonatal chemical hypoglycemia in newborns from pregnancies complicated by type 2 and gestational diabetes mellitus - the importance of neonatal ponderal index. *J Matern Fetal Neonatal Med*, 25(3):267-71. [CrossRef] [PubMed]
13. Duan, W. X., Yang, X. H., Zhang, H. F, Feng, J., & Zhang M. Y. (**2022**). Chemical Structure, Hypoglycemic Activity, and Mechanism of Action of Selenium Polysaccharides. *Biol Trace Elem Res*, 200(10):4404-4418. [CrossRef] [PubMed]
14. Jingjie, W., Fangfei, L., Zeng, D., Baohua, K., Hao, W., & Xiufang, X. (**2022**). Physicochemical properties and antioxidant activity of polysaccharides obtained from sea cucumber gonads via ultrasound-assisted enzymatic techniques. *Food Science and Technology*, 160:1096-1127. [CrossRef]
15. Levine, M., Boyer, E. W., Pozner, C. N., Geib, A. J., Thomsen, T., Mick, N., & Thomas, S. H. (**2007**). Assessment of hyperglycemia after calcium channel blocker overdoses involving diltiazem or verapamil. *Crit Care Med*, 35(9):2071-5. [CrossRef] [PubMed]
16. Chen, S. M, Gao, F., Li, M., Dong, T. W., & Geng, Z. (**2023**). Evaluation of mulberry leaves' hypoglycemic properties and hypoglycemic mechanisms.Front. Pharmacol., *Front Pharmacol*, 6:14:1045309. [CrossRef] [PubMed]
17. Hu, Y., Zhang, Y., Cui, X., Wang, D., Hu, Y., & Wang, C. (**2024**). Structure-function relationship and biological activity of polysaccharides from mulberry leaves: A review.Int J Biol Macromol, 268(Pt 1):131701. [CrossRef] [PubMed]
18. Gao, M., & Xu, S. (**1999**). Advances in the study of hypoglycemic effective monomer elements in natural pharmaceutical materials and their pharmacological action. *Zhong Yao Cai*, 22(10):542-5. [PubMed]
19. van Dijk, P. R., Schutten, J. C., Jeyarajah, E. J, Kootstra-Ros, J. E, Connelly, M. A., Bakker, S. J. L, Dullaart, R. P. F. (**2019**). Blood Mg^{2+} is more closely associated with hyperglycaemia than with hypertriacylglycerolaemia: the PREVEND study. *Diabetologia*. 62(9), 1732-1734. [CrossRef] [PubMed]
20. Obeid, O. (**2019**). Effect of Bread Fortification with Phosphorus and Lysine on Postprandial Glycemia and Thermogenesis. American University of Beirut Medical Center. https://trial.medpath.com/clinical-trial/a7f3a452d1db1072/effect-bread-fortification - phosphorus-lysine-glycaemia-thermogenesis
21. Pavie, J., Scemla, A., Bouldouyre, M. A., Pillebout, E., Verine, J., & Molina, J. M. (**2011**). Severe acute renal failure in an HIV-infected patient after only 2 weeks of tenofovir-based antiretroviral therapy. *AIDS Patient Care STDS*, 25(8):457-60. [CrossRef] [PubMed]
22. Veronese, N., Watutantrige-Fernando, S., Luchini, C., Solmi, M., Sartore, G., Sergi, G., Manzato, E., Barbagallo, M., Maggi, S., & Stubbs, B. (**2016**). Effect of magnesium supplementation on glucose metabolism in people with or at risk of diabetes: a systematic review and meta-analysis of double-blind randomized controlled trials. *Eur J Clin Nutr*. 2016 Dec;70(12):1354-1359. [CrossRef] [PubMed]
23. Kostov, K. (**2019**). Effects of magnesium deficiency on mechanisms of insulin resistance in type 2 diabetes: Focusing on the processes of insulin secretion and signaling. *Int J Mol Sci, 20(6), 1351.* [CrossRef] [PubMed]
24. Rayssiguier, Y., Libako, P., Nowacki, W., & Rock, E. (2010). Magnesium deficiency and metabolic syndrome: Stress and inflammation may reflect calcium activation. *Magnes Res, 23(2), 73-80.* [CrossRef] [PubMed]
25. Henkin, R. I. (**2018**). Calcium and Magnesium Levels Change in Relationship to Variations in Usual Dietary Nutrient Intake. *J Nutrition Health Food Sci*, 6(5), 1-17. [CrossRef]
26. Takaya, J., Higashino, H., & Kobayashi, Y. (**2004**). Intracellular magnesium and insulin resistance. *Magnes Res, 17(2), 126-36.* [PubMed]
27. Botturi, A., Ciappolino, V., Delvecchio, G., Boscutti, A., Viscardi, B., & Brambilla, P. (**2004**). The role and the effect of magnesium in mental disorders: A systematic review. *Nutrients, 12(6), 1661.* [CrossRef] [PubMed]
28. Djurhuus, M. S., Skøtt, P., Vaag, A., Hother-Nielsen, O., Andersen, P., Parving, H. H., & Klitgaard, N. A. (**2000**). Hyperglycaemia enhances renal magnesium excretion in type 1 diabetic patients. *Scand J Clin Lab Invest, 60(5), 403-9.* [CrossRef] [PubMed]
29. Rains, J. L., & Jain, S. K. (**2011**). Oxidative stress, insulin signaling, and diabetes. *Free Radic Biol Med, 50(5), 567-75.* [CrossRef] [PubMed]
30. Backe, M. B., Moen, I. W., Ellervik, C., Hansen, J. B., & Mandrup-Poulsen, T. (**2016**). Iron regulation of pancreatic beta-cell functions and oxidative stress. *Annu Rev Nutr, 36:241-73.* [CrossRef] [PubMed]
31. Alcantara, O., Obeid, L., Hannun, Y., Ponka, P., & Boldt, D. H. (**1994**). Regulation of protein kinase C (PKC) expression by iron: Effect of different iron compounds on PKC-β and PKC-α gene expression and role of the 5′-flanking region of the PKC-β gene in the response to ferric transferrin. *Blood, (10), 3510-7.* [PubMed]
32. Kuvibidila, S. R., Kitchens, D., & Baliga, B. S. (**1999**). In vivo and in vitro iron deficiency reduces protein kinase C activity and translocation in murine splenic and purified T cells. *J Cell Biochem, 74(3), 468-78.* [PubMed]
33. Mellor, K. M., Ritchie, R. H., & Delbridge, L. M. D. (**2010**). Reactive oxygen species and insulin-resistant cardiomyopathy. *Clin Exp Pharmacol Physiol, 37(2), 222-8.* [CrossRef] [PubMed]