Biochemistry & Physiology: Open Access
ISSN: 2168-9652

Balbaa, Biochem Physiol 2016, 5:3
DOI: 10.4172/2168-9652.1000e151

Editorial OMICS International

Diabetes and Cell Signaling

Mahmoud Balbaa*

Department of Biochemistry, Faculty of Science, Alexandria University, Alexandria, Egypt

Diabetes mellitus is a metabolic disease which affects not only the glucose metabolism, but also lipid and protein metabolism. There are two main types of diabetes, hereafter are termed as type I and type II. Type I diabetes is characterized by destruction of the β-cells of the pancreas and insulin is not produced, whereas type II diabetes mellitus is characterized by a progressive impairment of insulin secretion and relative decreased sensitivity of target tissues to the action of this hormone [1,2]. Diabetes mellitus is a major worldwide health problem predisposing to markedly increased cardiovascular mortality. Many serious problems are related to the development of nephropathy, neuropathy, and retinopathy [2-7]. In addition, the pathogenesis of diabetes mellitus was not only implicated in the oxidative stress but also the hyperglycemia-induced protein glycation that generates superoxide free radicals [2,3,6,7]. The generation of reactive oxygen species may lead to lipid peroxidation and formation of reactive products, which may be involved in severe damage of cell molecules and structures. As type II diabetes mellitus continues to increase world widely, there is an enhanced need for effective disease management [2].

In diabetes, the insulin resistance is partly mediated by lowering insulin receptor (IR) expression level [8]. This is followed by impaired tyrosine phosphorylation of IR and subsequent tyrosine phosphorylation of IRS-1 and the attenuated association the regulatory subunit (P85) of phosphoinositide-3 kinase (PI3K) with IRS-1. This results in subsequent deactivation of its catalytic subunit (P110) [9]. Therefore, when the reduction of PI3K signaling pathway occurs, the protein kinase Akt will be activated and the reduction of glucose transport will occur [10]. Consequently, the plasma free fatty acids increase and the insulin-stimulated glucose uptake will be reduced [11]. Moreover, in the development of insulin resistance, an increased release of pro-inflammatory cytokines occurs [12]. The stimulated TNF-α production contributes to ß-cell degradation [13]. Hyperglycemia increases the activities of matrix metalloproteinases such as ADAM17, which may be linked to the unbalanced expression of the tissue inhibitor of metalloproteinase 3 (TIMP3) [14]. Therefore, the reduction of TIPM3 results in an increase of ADAM17 and TNF-α [15]. In addition, the reduction of antioxidants and elevation of reactive oxygen species in diabetes mellitus lead to stimulation of the tumor necrosis factor TNF-α and ADAM17 production [16-18]. We have an ongoing intensive investigation of insulin-induced signaling molecules in liver and brain tissues during the treatment of streptozotocin-induced diabetic rats by *Nigella sativa* oil (NSO) in combination with some anti-diabetic drugs. The anti-diabetic effect of NSO was confirmed through a linked investigation of lipid profile, antioxidant activity and signaling molecules in the absence and presence of some anti-diabetic drugs [19]. In conclusion, a chance of interaction between herbs and drug was suggested during the treatment of diabetes.

References

1. Burcelin R, Rolland E, Dolci W, Germain S, Carrel V, et al. (1999) Encapsulated, genetically engineered cells, searching glucagons-like peptide-1 for the treatment of non-insulin-dependent diabetes mellitus. Ann NY Acad Sci 875: 277-362.
2. Kaushik G, Satya S, Khandelwal RK, Naik SN (2010) Commonly consumed Indian plant food materials in the management of diabetes mellitus. Diab Metabol Syndr: Clin Res Rev 4: 21-40.
3. Atalay M, Laaksonen DE (2002) Diabetes, oxidative stress and physical exercise. J Sports Sci Med 1: 1-14.
4. Bringer J, Fontaine P, Detournay B, Nachit-Ouinekh F, Brami G, et al. (2009) Prevalence of diagnosed type 2 diabetes mellitus in the French general population: The INSTANT study. Diabetes Metab 35: 25-31.
5. King H, Aubert RE, Herman WH (1998) Global burden of diabetes, 1995-2025: Prevalence, numerical estimates and projections. Diabetes Care 21: 1414-1431.
6. MemisoÄŸullari R, Taysi S, Bakan E, Capoglu I (2003) Antioxidant status and lipid peroxidation in type II diabetes mellitus. Cell Biochem Funct 21: 291-296.
7. Raskin P, Jovanovic L, Berger S, Schwartz S, Woo V, et al. (2000) Repaglinide/troglitazone combination therapy: Improved glycemic control in type 2 diabetes. Diabetes Care 23: 979-983.
8. Virkamäki A, Ueki K, Kahn CR (1999) Protein-protein interaction in insulin signaling and the molecular mechanisms of insulin resistance. J Clin Invest 103: 931-943.
9. Højlund K (2014) Metabolism and insulin signaling in common metabolic disorders and inherited insulin resistance. Dan Med J 61: B4890.
10. Anai M, Funaki M, Ogihara T, Terasaki J, Inukai K, et al. (1998) Altered expression levels and impaired steps in the pathway to phosphatidylinositol 3-kinase activation via insulin receptor substrate 1 and 2 in Zucker fatty rats. Diabetes 47: 13-23.
11. Moller DE (2001) New drug targets for type 2 diabetes and the metabolic syndrome. Nature 414: 821-827.
12. Boden G (2008) Obesity and free fatty acids. Endocrinol Metab Clin North Am 37: 635-646, viii-ix.
13. Yuan HD, Chung SH (2010) Protective effects of fermented ginseng on streptozotocin-induced pancreatic beta-cell damage through inhibition of NF-kappaB. Int J Mol Med 25: 53-58.
14. Federici M, Menghini R, Mauriello A, Hribal ML, Ferrelli F, et al. (2002) Insulin-dependent activation of endothelial nitric oxide synthase is impaired by O-linked glycosylation modification of signaling proteins in human coronary endothelial cells. Circulation 106: 466-472.
15. Menghini R, Menini S, Amoruso R, Fiorentino L, Casagrande V, et al. (2009) Tissue inhibitor of metalloproteinase 3 deficiency causes hepatic steatosis and adipose tissue inflammation in mice. Gastroenterology 136: 663-672.
16. Niture NT, Ansari AA, Naik SR (2014) Anti-hyperglycemic activity of rutin in streptozotocin-induced diabetic rats: An effect mediated through cytokines, antioxidants and lipid biomarkers. Indian J Exp Biol 52: 720-727.
17. Saud AM (2014) Serum levels of tumor necrosis factor alpha and interleukin-12 in some Iraqi diabetic patients type I. Int J Curr Microbiol App Sci 3: 260-268.
18. Bartoli M, Lamoke F, Shaw S, Gutsaeva D, Baban B (2014) Modulation of cytokine trans-signaling by microRNA-21 (miR-21)-mediated regulation of TACE/ADAM17 in the diabetic retina. Invest Ophthalmol Vis Sci 55: 1030.
19. Balbaa M, El-Zeftawy M, Ghareeb D, Taha N, Mandour AW (2016) *Nigella sativa* relieves the altered insulin receptor signaling in streptozotocin-induced diabetic rats fed with a high-fat diet. Oxid Med Cell Longev, p: 2492107.

***Corresponding author:** Mahmoud Balbaa, Department of Biochemistry, Faculty of Science, Alexandria University, Alexandria, Egypt, E-mail: mahmoud.balbaa@alexu.edu.eg

Received October 19, 2016; **Accepted** October 20, 2016; **Published** October 25, 2016

Citation: Balbaa M (2016) Diabetes and Cell Signaling. Biochem Physiol 5: e151. doi: 10.4172/2168-9652.1000e151