Diabetes and Cell Signaling

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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 membranes 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 (PS5) of phosphoinositide-3 kinase (PI3K) with IRS-1. This results in subsequent deactivation of its catalytic subunit (PI110) [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 β-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 TIMP3 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


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