Diabetes Case Reports
ISSN: 2572-5629
Saiedullah, Diabetes Case Rep 2016,1:1
DOI: 10.4172/2572-5629.1000e102
Editorial
Open Access

Insulin Sensitivity or Resistance in Type 2 Diabetes Mellitus with Obesity

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Type 2 diabetes mellitus, a state of hyperglycemia associated with beta cell dysfunction or insulin action on target cells. This state is often mediated or sustained by obesity linked inflammatrory cytokines. Tumor necrosis factor-alpha (TNF-α), most important inflammatory cytokine plays critical role in the pathogenesis of type 2 diabetes mellitus as evidenced from higher circulating levels of TNF-α in type 2 diabetes mellitus compared to controls [1,2] and its negative influence on insulin secretion and insulin sensitivity in type 2 diabetes mellitus [1,3]. Positive association of TNF-α with insulin secretion in subjects with impaired glucose regulation [4] indicates its causal relationship beta-cell dysfunction, insulin resistance or sensitivity. In obese subjects with type 2 diabetes mellitus, we observed stronger and graded relationship between TNF-α and obesity [3].

It is well established that in response to inflammation, biosynthesis of inflammatory molecules are stimulated [5] which are common in obese subjects [6]. Higher TNF-α may downregulate the genes that encodes proteins/factors required for normal insulin action and involved in negative regulation of an insulin-sensitizing nuclear factor, modulation of insulin signaling pathway or induction of free fatty acids [7].

Thus management of obesity and obesity-mediated inflammation or its mediators like TNF-α may help in reduction of burden and hazards related diabetes or its complications.

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Received: June 28, 2016; **Accepted:** June 29, 2016; **Published**: June 30, 2016

Citation: Saiedullah M (2016) Insulin Sensitivity or Resistance in Type 2 Diabetes Mellitus with Obesity. Diabetes Case Rep 1:e102. doi: 10.4172/2572-5629.1000e102