

Obesity and Prediction of Type 2 Diabetes

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Obesity, in particular, central or abdominal obesity is a recognized pro-inflammatory state prone for broad alterations of the metabolic milieu, which include insulin resistance, impaired glucose tolerance, dyslipidemia and hypertension. It is unclear whether the pro-inflammatory state determines the insulin resistance condition or insulin resistance causes increased systemic inflammation [1]. Central obesity is defined according to International Diabetes Federation (IDF), by the measure of waist circumference. An excessive accumulation of abdominal fats is most tightly associated with type 2 diabetes [2]. The association of obesity with development of type II diabetes may be partly mediated by altered secretion of adipokines by adipose tissue.

Adiponectin represents one of the most abundant and well-studied adipokines that has been implicated as a major protective factor against the adverse metabolic and cardiovascular consequences of obesity [3].

The main-insulin sensitizing action of adiponectin results from decrease in hepatic gluconeogenesis by acting on hepatocytes via AMP activated protein kinase (AMPK) to inhibit glucose production and reduce intracellular triglycerides and on myocytes via AMPK to stimulates glucose uptake and reduce intracellular triglycerides and insulin resistance. Secondly from enhancement of energy consumption and fatty acid oxidation in peripheral tissues with the aim of increasing ATP production [3].

The association of obesity with insulin resistance syndrome is not only related to the degree of obesity but also seems to be critically dependent on body fat distribution.

Thus, individual with greater degree of central adiposity develop this syndrome more frequently than do those with a peripheral body fat distribution [4].

Obesity is strongly associated with elevated concentrations of circulating markers of inflammation, such as C-reactive protein (CRP) and Apo-lipoprotein B (Apo-B).

Elevated plasma levels of CRP have been associated with increased risk of coronary heart disease and type II diabetes [5]. As synthesis of CRP is regulated by adipocyte cytokines, such as tumor necrosis factor (TNF- α), elevated levels of CRP up-regulated with increase of adipocyte burden in case of obesity [6]. ApolipoproteinB (ApoB) is synthesized by liver and is responsible for carrying cholesterol to the tissues [6].

It is a key structural component of all atherogenic lipoprotein particles. Each of these particles carries only one ApoB molecule, thus the total ApoB level represents the total number of circulating atherogenic lipoprotein particles [6]. There is considerable evidence that levels of ApoB are a better indicator of heart disease and type II diabetes risks than total cholesterol or LDL [6]. Hyper ApoB was found to increase the risk of developing the incidence of diabetes [6].

There is a change in CRP concentration and IR with exercise. Exercise is known to increase insulin auto-phosphorylation, glucose transporter 4 expressions and glucose transport. It increase glucose uptake by muscle cells which consequently increase the oxidation of glucose leading to improvement in Insulin resistance (IR).

Exercise directly reduce cytokine production in fat, muscle and mononuclear cells, and Indirectly by increasing, insulin sensitivity, improving endothelial function causing reduction of nitric oxide synthase activity improving in insulin resistance and reducing body weight [7].

As a conclusions lifestyle modification by eating a healthy diet and being more physically active with reducing and maintaining a healthy weight, we can reverse insulin resistance and prediabetic status to prevent or delaying type 2 diabetes mellitus.

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