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Cellular Inflammation in the etiology and treatment of Type-2 Diabetes

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Abstract

Cellular inflammation mediated by NFκβ activation and subsequent inflammasome play an important role in the initiation and progression of Type 2 diabetes (T2D). Inflammation is triggered by predetermined genetic trait followed by lifestyle and environmental factors.

It is hypothesized that prediabetic state results in response to increased hepatic inflammation due to high dietary fructose levels as well as other di- etary components triggering microbial dysbiosis. This gut dysbiosis decreases hepatic insulin clear- ance and increases lipogenesis which lead to hy- perinsulinemia and dyslipedemia thereby altering insulin sensitivity of myocytes.

Persistent inflammatory factors lead to hyper- glycemia and dysfunctionality of the β-cell pul- satile insulin secretion causing a vicious cycle be- tween all these organs. Eventually, this process also leads to adipocyte insulin resistance. In some individuals, these changes lead to obesity without concomitant hyperglycemia.

Although these processes are common to T2D etiology, genetic, nutritional, environmental stress factors as well as demographic factors influence specific outcome. Hence, it is proposed that the treatment regime should consider these factors ultimately designing molecular therapy to mini- mize all kinds of cellular inflammation. Specifical- ly, antioxidants such as pyrroloquinoline quinone and tocotrienol along with anti-inflammatory nanocurcumin and itaconate compounds need to be used for diabetes treatment. Population specif- ic probiotic consortia is needed for ameliorating the deleterious effects of gut dysbiosis.

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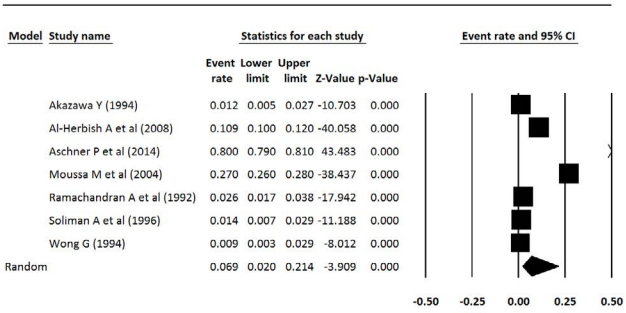

Figure 1. Prevalence of type 1 diabetes in Asia