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Special Issue on

# Environmental Factors and the Risk of Development of Type 1 and Type 2 Diabetes Mellitus

CALL FOR PAPERS

Diabetes as a social and civilization disease has reached an epidemic level at the turn of the 20th and 21st centuries. The most recent International Diabetes Federation (IDF) data, published on November 14, 2013, indicate that diabetes affects 383 million people all over the world. As estimated by the IDF, the number of diabetic patients is likely to reach 592 million by 2035. Although the patomechanisms of diabetes have been investigated in numerous studies, there are many factors, including the involvement of the immune system, that have not been elucidated. Therefore, it is very important that the interactions and mechanisms associated with the development of type 1 and type 2 diabetes mellitus are fully understood. It is well known that both type 1 and type 2 diabetes mellitus are characterized by progressive pancreatic beta cell damage. However, the mechanisms that contribute to the development of each of these two types of diabetes are different. Currently, there are several hypotheses that explain the mechanisms of diabetes development. Among them there is an accelerator hypothesis that suggests a common background for type 1 and type 2 diabetes. An association has been shown between Pro12Ala polymorphism in the peroxisome proliferator-activated receptor (PPAR) gamma2 gene and the onset of type 1 and type 2 diabetes. Moreover, the accelerator hypothesis suggests that excess adipose tissue and insulin resistance can accelerate beta cell destruction through the primary or secondary effects on the beta cell apoptosis, thereby initiating relevant autoimmune processes in genetically predisposed subjects. Another popular hypothesis is the "overload hypothesis" presented by Dahquist as a kind of completion to the accelerator hypothesis. The hypothesis assumes that there are some environmental factors that contribute to the increased susceptibility of beta cells to necrosis or/and apoptosis.

Potential topics include, but are not limited to:

- The effect of viral infections on the inset of diabetes
- The effect of overfeeding of children and their excess body mass gain during the first years of life on the onset of diabetes
- Low physical activity that initiates insulin resistance leading to beta cell hyperactivity
- Low vitamin D3 level and the onset of diabetes
- Food as a risk factor for diabetes
- Effect of probiotics in modulating parameters related to diabetes
- Psychological stress and the onset of type 1 diabetes
- Immune system dysregulation and the onset of diabetes
- Chitinase 3-like-1, an emerging immunomodulator involved in diabetic complications

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