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Virus-induced diabetes: Diabetogenic viruses and host susceptibility genes and vice versa

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Explosive increase of patients with diabetes in the world must be, at least in part, due to environmental factors, especially viruses. Experimental evidence had shown that even diabetogenic encephalomyocarditis strain D (EMC-D) virus-induced diabetes is inducible only in limited strains of mice such as SJL, DBA/2 and SWR backgrounds. We could show that natural mutation of Tyrosine kinase 2 (TYK2) gene: Interferon (IFN) receptor-associated signaling molecule is responsible for susceptibility to murine EMC-D virus-induced diabetes, with deteriorated IFN dependent anti-viral response in pancreatic beta-cells. Moreover, a human TYK2 gene haplotype with multiple genetic polymorphisms at promoter region in complete linkage disequilibrium, named TYK2 promoter variant (NCBI ClinVar, variation ID: 440728), is associated with an increased risk for type-1 diabetes (T1D), particularly for anti-glutamic acid decarboxylase antibody (GADAb) negative T1D associated with flu-like syndrome at onset (Odds ratio: 3.6). Surprisingly, the risk is high in non-obese patients with type-2 diabetes (T2D) (Odds ratio: 2.4). We also found that patients with T1D had increased serum IgE levels, however, those with flu-like syndrome at the onset showed no relation to increased IgE nor anti-GAD, but with TYK2 promoter variant. These experimental and clinical evidences taken together indicated that TYK2 gene serves a virus-induced diabetes susceptibility gene, common to mice and humans. Conversely, these findings suggested that latent diabetogenic viruses may induce diabetes only in small fraction of people possessing virus-induced susceptibility gene. Moreover, the concept could extend to other infectious diseases such as Zika virus infection complicated with microcephalic infants among a few pregnant women. Thus, these observations should enhance the research for the identification of latently diabetogenic viruses using rodents with multiple virus-induced diabetes susceptibility genes in high sensitivity. Those studies will convincingly lead to the development of anti-diabetogenic virus vaccine not only to prevent T1D but also to reduce the risk of T2D, among patients due to viral infection.

Biography

Seiho Nagafuchi has graduated from Medical School of Kyushu University, Japan. He was the Resident in Department of Medicine, Kyushu University Hospital (1975) and the Visiting Fellow (1978), National Institute of Health, USA. He was the Chief Internist of Internal Medicine at Fukuoka-Teishin Hospital (1983) and was the Associate Professor (1998), Department of Health Sciences, Kyushu University, Japan. He is presently a Visiting Research Scientist at Department of Hepatology, Faculty of Medicine, Saga University and a Professor Emeritus, Kyushu University, Japan since 2016.

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