



A Molecular Link Between Diabetes and Dementia: Implications for Prevention



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Type 1 diabetes (T1D) patients have lost almost all their insulin-producing beta cells in the pancreatic islets owing to their autoimmune destruction by the body's own immune system. However, recent studies suggest that intrinsic non-immune beta-cell dysfunction or degeneration could play a central role in the disease process, initiated by anti-islet autoimmunity, but then proceeding independently of the immune system.

Genome-wide association studies (GWAS) analysis of T1D suggests that the neurodegenerative disease gene, MAPT, encoding microtubule-associated protein TAU, has a beta-cell degenerative function. There is evidence that TAU is expressed in islets and in beta-cell lines and hence factors such as inflammation in T1D and beta-cell stress owing to high sugar or fat diets and insulin resistance in type 2 diabetes (T2D) and their associated hyperglycaemia and glucotoxicity, could lead to beta-cell tauopathy, compromised protein homeostasis, disrupted insulin secretion and apoptosis.

Prolonged exposure to hyperglycaemia in diabetes and ageing could induce TAU toxicity in other extra-cerebral sites and cause nephropathy and neuropathy. Therapies targeting TAU and improving protein homeostasis may be of clinical benefit in diabetes.

Finally, diabetes is more common in Alzheimer's patients and this could be due to TAU-associated malfunction and degeneration of the beta cells. In this lecture, Professor John Todd will discuss on the molecular implication that links diabetes to dementia.

Date:
10 April 2017
Monday

Time:
5:30 PM – 7:00 PM

Venue:
Auditorium JC 2, Level 1,
Sunway University

All Are Invited

FREE ADMISSION

For registration, please visit:

<https://university.sunway.edu.my/jcdss>