Effect of diabetes on hippocampal gene expression

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Diabetes is a metabolic disorder that leads to other health complications over a period of time and is the cause for considerable morbidity and mortality world-wide. The complications of diabetes in the peripheral tissues are well characterized including coronary heart disease, retinal degeneration, renal disorders, and also micro and macro vascular complications. Over the last few decades much emphasis has been placed on the complications of diabetes that occur in the central nervous system (CNS). One such neuropathology due to diabetes in CNS is cognitive impairment. The hippocampus, the limbic structure, is involved in higher brain functions and appears to be particularly vulnerable to diabetes presents a study on the comparison of gene expression profile in the hippocampus of a streptozotocin (STZ) induced diabetic mice and a vehicle treated control animals. Here it is demonstrated that diabetes causes significant alterations in the genes that plays a crucial role in synaptic function and plasticity and also for neurogenesis, both of which are required for normal cognitive functions. Six weeks after diabetes was established in these mice, a number of genes had altered expression including genes involved in epigenetic regulation, and this included histone deacetylase (Hdac) 4, 9 and 11. Interestingly Hdac 4 and 9 are abundantly present in the hippocampus and are required for hippocampal dependent learning, memory and synaptic plasticity. Glycogen synthase kinase beta (Gsk3β) which has been shown to have a crucial role in metabolic and neurodevelopmental functions and considered to be an important regulator of synaptic functions, also exhibited significant decrease in the STZ induced diabetic mice as opposed to the buffer treated control group. The marked decrease of these genes that are crucial for higher functions in the hippocampus underscores the impact of uncontrolled diabetes on the hippocampus. Furthermore genes that have been linked to neurological disorder and cognitive dysfunction such as apolipoprotien E (ApoE) showed increased expression in diabetic mice as opposed to the non-diabetic control group. These findings implicate the abnormal transcription of genes which could disrupt normal cognitive functions in the hippocampus and also underscores epigenetic mechanisms involved in disease conditions like diabetes.

Biography

Jency Thomas is an early career researcher and a teaching academic at La Trobe University. In a short career span she has 6 papers in A* Journals with one invited book chapter. She is an early career researcher building on from her PhD work. She is developing her interest on metabolic diseases and mental health outcomes. She has made significant contribution and added new knowledge in the field of diabetes and cognitive impairment. She has significant experience working with range of antioxidants including omega-3 fatty acid and polyphenols. As an early career researcher, she is keen to understand the effect of metabolic syndrome such as obesity, hypertension, dyslipidemia and diseases like cardiovascular and diabetes on mental health outcomes. Her paper “Dietary Supplementation with resveratrol and/or docosahexaenoic acid after hippocampal gene-expression in adult C57Bl/6 mice”. Journal of Nutrition Biochemistry was awarded the best original research paper, by University of Newcastle 2013. Her achievement is also recognized by invitation to write book chapter on “diabetes and gene expression”. Currently she is involved in number of projects, including a project on Australian Longitudinal Study on Women’s Health Data (ALSWH).

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