ORAL PQE

Establishing an in vitro ageing model in human pluripotent stem cells and neural derivatives to model neuronal aging

Aging is an inevitable phenomenon in every living cell. With aging, comes deterioration in cellular processes like cellular senescence, telomere attrition, genomic instability, metabolic dysfunction and epigenetic alterations among others. Since aging is the largest risk factor of neurodegenerative diseases, we theorise that these aging hallmarks contribute to neuronal degradation and death. The exact causes of neurodegenerative diseases that are mainly sporadic like Amyotrophic Lateral Sclerosis and Frontotemporal Dementia are still unknown. Physiological aging has been evidenced to be contributed by several genes like telomerase and sirtuins. Current animal aging models do not explain the pathology of neurodegenerative diseases. The use of human pluripotent stem cells (hPSCs) have been widely used in disease modelling. Hence, we aim to use hPSCs with specific mutations of the aging genes to generate cells with the aged hallmarks and discover specific molecular pathways in which aging-related processes directly cause neurodegeneration. Therefore, with our accelerated aging system, it will accelerate aging of neurons and will allow us to study the molecular contributions of aging to neurodegeneration.

Friday
21 June 2019
12.30pm – 2.00pm
Seminar Room, MD10
Level 2, Anatomy Museum

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