Australian Government

NHMRC National Institute for Dementia Research

AUSTRALIAN DEMENTIA FORUM

Abstracts

Accelerating research. Enhancing collaboration. Creating change.

Hotel Grand Chancellor Hobart, 13-14 June 2019

nnidr.gov.au

Contact

NHMRC National Institute for Dementia Research (NNIDR)
Level 1, 16 Marcus Clarke Street
Canberra City ACT 2601

Phone: 02 6217 9170
Email: nnidr@nnidr.gov.au
www.nnidr.gov.au

CONTENTS

All inflammatory markers were significantly upregulated at 12 months ($p<0.05$), demonstrating heterogenic inflammation of both anti- and pro-inflammatory profiles. The increased inflammatory profiles occur simultaneously with morphological shifts suggesting a potential relation at late stage plaque development. Spatially, at 12 months there was significant increase in inflammation directly correlating to plaque location ($p<0.05$). Overall, these data suggest microglia display a heterogenic inflammatory profile throughout disease progression, which impacts future research in designing potential therapeutics for inflammatory cascades activated AD.

DR ABRAHAM KUOT

Flinders University Rural Health South Australia

Harmony in the Bush: An innovative personalised care model for dementia in rural residential care

Dr Abraham Kuot[1], Dr Vivian Isaac[1], Mrs Margaret Kimani[1], Dr Mohammad Hamiduzzaman[1], Professor Jennene Greenhill[1]

[1]Flinders University Rural Health South Australia, Renmark, Australia

There are creative ways to improve the quality of life, and decrease the stress, carer burden and staff workloads in residential care facilities. Harmony in the Bush is an innovative research study aims to co-design an effective model of care for dementia in residential facilities. Approximately 30% of Australians live in rural communities. Dementia is a major concern for many rural communities where there are ageing populations with poor access to health services. Many people rely on aged care facilities as their relatives experience progressive decline, particularly when they experience behavioural and psychological symptoms of dementia such as agitation and wandering. These symptoms are complex, stressful and costly aspects of care. Institutionalisation and antipsychotic medications have limited efficacy but are widely used in residential aged care. The study is funded by the Australian Government Dementia and Aged Care Services grant, and is a two-year, longitudinal, quasi-experimental design involving behaviour measurements, interviews, and focus groups in five different kinds of residential facilities to evaluate the model's effectiveness in various rural health contexts. They include small and large, private, public, not for profit, people from multicultural backgrounds and an Aboriginal specific facility. Our 'Ageing Well in Harmony' is a new model of care incorporating personalised care, non-pharmacological interventions and music for people with dementia (PWD). This presentation will include an overview of the study design and preliminary findings. This personalised model of care will have long-term positive outcomes for rural communities especially beneficial for PWD, carers, aged care staff and their workplaces.

MR ROSS LANGLEY

Wicking Dementia Research and Education Centre

A new method of tracking microglia motility and synapse interactions in vivo

Mr Ross Langley[1], Dr Jessica Collins[1], Associate Professor Alison Canty[1], Associate Professor Anna King[1], Dr Jenna Ziebell[1]

[1]Wicking, Hobart, Australia

Microglia are highly motile immune cells found within the brain and their dysfunction has been implicated in the progression of neurodegenerative diseases such as Alzheimer's disease (AD). As the brain's primary immune cell, they constantly survey their surroundings for signs of infection or any "waste" that needs to be cleared. In addition to this they also play a role in the wiring of the brain and have been found to periodically contact synapses, the communication points between neurons. Studies suggest that this is to monitor the functionality of the synapses. It is hypothesised that microglial processes may slow down in AD resulting in a build up of waste and disrupted synapse maintenance. We have developed a protocol that allows us to measure the movements of microglia *in vivo* using mice that express fluorescent proteins on both microglia and neurons allowing these synaptic interactions to be visualised. Following an imaging session, the movement of the microglia is tracked using a program known as Trackmate. This collects data on the speed of the processes, the distance covered and the contacts made with synapses. This is tracked throughout the animal's lifetime allowing us to see if microglia dysfunction occurs with ageing and/or at which stage of AD it occurs. This protocol is the first to allow for the collection of high volumes of microglia movement and synapse interaction data. This has the potential to give us new targets for therapeutics as well as a time period that these treatments will be most effective.

DR JACQUELINE YK LEUNG

Wicking Dementia Research and Education Centre

The role of TAR DNA binding protein 43 (TDP43) in white matter degeneration in dementia.

Dr Jacqueline YK Leung[1], Mr Samuel T Dwyer[1], Dr Rachel Atkinson[1], Professor James Vickers[1], Associate Professor Anna King[1]

[1]Wicking Dementia Research and Education Centre, Hobart, Australia

White matter degeneration is a pathological feature of frontotemporal dementia (FTD), although the mechanism of this degeneration is currently unknown. TAR DNA binding protein 43 (TDP43) aggregates have been found in oligodendrocytes; however, the role of TDP43 in oligodendrocytes and its effect on oligodendrocytes development and myelin production has not been determined. Our research hypothesises that TDP43 has a direct role in oligodendrocytes development; hence the dysfunction of TDP43 might potentially contribute to white matter degeneration observed in FTD. To examine this, we have utilised primary cultured oligodendrocyte precursor cells, where the TDP43 expression is manipulated using lentivirus (either overexpression of wild type; TDP43-WT, or nuclear-localisation signal mutation; TDP43-NLS). The morphology of cells was analysed using immunohistochemistry and tracing in Image-J.

Our preliminary data indicate that overexpression of TDP43-WT leads to a significantly ($p<0.05$) more complex cell morphology compared to non-transduced control cells ($n=20$). The expression of a mutant form of TDP43 leads to a less complex morphology when compared to non-transduced control ($n=10$). To examine this further in vivo, we utilise the Sox10-iCre/ERT2 transgenic mouse and AAV carrying the lox-sequence (e.g., lox-TDP43) to create a mouse model where the TDP43 expression is altered in an oligodendrocytes-specific manner. We aim to use this model to study the long-term changes in oligodendrocyte development and myelination in the presence of pathologic TDP-43.

Australian Government

NHMRC National Institute for Dementia Research

NHMRC National Institute for Dementia Research (NNIDR)
Level 1, 16 Marcus Clarke Street
Canberra City ACT 2601

Phone: 02 6217 9170
Email: nnidr@dementia.org.au