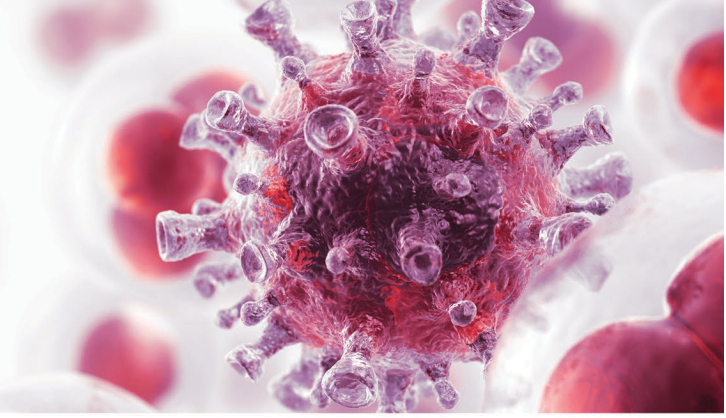
Griffith UNIVERSITY
Institute for Glycomics
Queensland, Australia



2021 Lecture Series

A/Prof Jenny Ekberg

Clem Jones Centre for Neurobiology and Stem Cell Research
School of Pharmacy and Medical Sciences
Menzies Health Institute Queensland
Griffith Institute for Drug Discovery

will present a seminar entitled

The nose-to-brain nerve path to central nervous system infection

Friday 22 October 2021, 11am

Institute for Glycomics
Lecture Theatre (G26 4.09) or via Zoom

(No food or drink allowed in the lecture theatre)

Abstract

Two cranial nerves connect the nasal cavity and the brain. These are the olfactory nerve, which mediates the sense of smell, and the trigeminal nerve (intranasal branches) that is responsible for sensation inside the nose. These two nerves also constitute paths by which certain microbes can invade the central nervous system (CNS). The olfactory and trigeminal nerves connect with the olfactory bulb and brainstem, respectively, within the CNS. These two brain regions are the first to show signs of neurodegeneration in both Alzheimer's and Parkinson's diseases, and it has been suggested that infectious agents can contribute to neurodegeneration.

We have investigated whether certain bacteria, known to be capable of causing brain infection, can reach the CNS via the olfactory/trigeminal nerves. We have recently focussed in particular on *Chlamydia pneumoniae*, a respiratory pathogen linked to neurodegeneration. We found that *C. pneumoniae* could rapidly invade the brain CNS via the olfactory/trigeminal nerves within 24-72 hours after intranasal inoculation. Amyloid beta accumulations, one hallmark of neurodegeneration, were detected adjacent to bacterial inclusions in the olfactory nervous system at 7 days post inoculation. We are now determining whether *C. pneumoniae* contributes to neurodegeneration in the long-term.

But why, if cranial nerves can serve as paths to rapid CNS infection, are such infections relatively rare? We have also over many years studied immune responses by the glial cells of these nerves, and found that these glial cells are key innate immune cells with strong phagocytic activity. We have found that the bacteria that are capable of infecting the CNS via cranial nerves can infect and survive inside glial cells, accompanied by features of neuropathology and inflammation. This is likely a key mechanism behind microbial CNS invasion via peripheral nerves, which may also contribute to neuropathologies.

For further information, contact

P +61 7 555 28051 **E** glycomics@griffith.edu.au griffith.edu.au/glycomics