Layperson Summary

To determine the effects of IL-20 and IL-24 on glial immune functions stimulated by bacterial products

Background
Bacterial meningitis is inflammation of the central nervous system (CNS), more specifically the brain, caused by infection with these microorganisms. It is a severe occurrence during many bacterial infections that can result in long-term neurological dysfunction or even death. Organisms such as *Streptococcus pneumonia* and *Neisseria meningitidis* are the most common causes of bacterial meningitis. Inflammation is a defense mechanism against foreign invaders but because the brain is enclosed by the cranium inflammation within the CNS can be deadly. As bacteria accumulate within the brain, the inflammatory response increases and elevations in intracranial pressure can have lethal consequences. Indeed it is this inflammation that is the root cause of death following infection rather than the bacteria themselves. CNS inflammation occurs due to the release of immune mediators by resident cells of the brain and recruited immune cells. These immune mediators include molecules known as cytokines. To date, the identity and function of many of these cytokines has not been defined in bacterial meningitis. Examples of this include newly identified members of the interleukin (IL)-10 family of cytokines, IL-20 and IL-24. While it has been recently shown that resident brain cells can produce IL-20 and IL-24, it is not clear whether these molecules are helpful or harmful following infection of the brain.

Objective
My project focuses on determining whether IL-20 and/or IL-24 augment or inhibit the inflammatory responses of isolated mouse brain cells to bacterial challenge.

Expected outcome
It is anticipated that the presence of IL-20 and/or IL-24 will alter the production of inflammatory mediators by isolated brain cells following exposure to bacteria. While it is not yet clear whether these cytokines will increase or decrease such responses, the previous demonstration that IL-20 and IL-24 are rapidly produced by brain cells is more consistent with a role in promoting inflammation than the delayed production usually seen with anti-inflammatory mediators. These studies will therefore shed new light on the role of these immune molecules in conditions such as bacterial meningitis.