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

Inflammation is a defense mechanism against foreign invaders but, because the brain is enclosed by the cranium, inflammation within the central nervous system (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. It is known that interleukin (IL)-10 is anti-inflammatory and we previously demonstrated that the related cytokine IL-19 also has anti-inflammatory characteristics. However, very little is known about the function of other IL-10 family members, IL-20 and IL-24, in CNS inflammation. Recently data from our research team has shown that glial cells express IL-20 and IL-24 after bacterial infection, and that IL-24 is produced in a delayed way. In the present study we provide evidence that IL-24 attenuates IL-6 production and that it acts as an anti-inflammatory agent following bacterial challenge. However, the effect of IL-20 on inflammation is, at present, inconclusive. The production of IL-24 by major glial cell types, could serve a protective function by limiting potentially damaging inflammation.