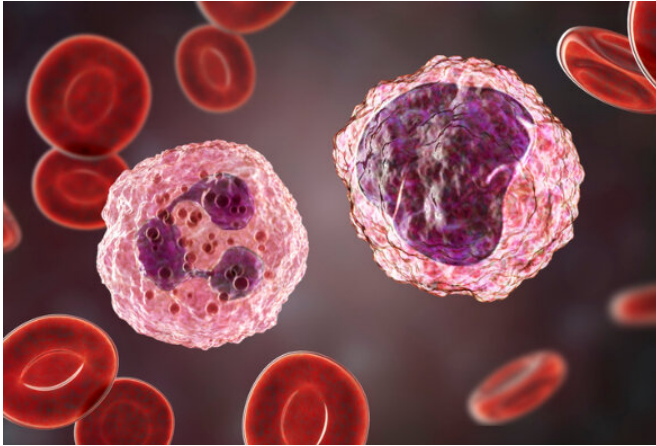


Researchers identify key role of immune cells in brain infection

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Credit: University of Liverpool

A new study has detailed the damaging role played by the immune system in a severe brain condition most commonly caused by the cold sore virus.

Researchers have identified the specific type of immune cell that induces [brain inflammation](#) in herpes simplex virus (HSV) encephalitis. Crucially, they have also determined the signaling protein that calls this immune cell into the [brain](#) from the bloodstream.

The findings, published in *Cell Reports*, could aid the development of targeted treatments for the brain infection, which is the most common cause of viral encephalitis worldwide.

HSV encephalitis takes hold quickly and, despite rapid anti-viral drug treatment, many patients die. Most survivors are left with brain injury due to the inflammation and damage caused by the virus and immune cells gaining access to the brain, breaking down the blood-brain barrier.

"Determining the roles of specific immune cells and

the factors that allow them to cross the protective blood-brain barrier is critical to develop targeted immune-therapies," explains Dr. Benedict Michael, a Senior Clinician Scientist Fellow at the University of Liverpool, who led the research.

Using a [mouse model](#), the researchers showed that neutrophils (a type of immune cell) made the blood-brain barrier more permeable and contributed to the brain damage associated with HSV encephalitis. They also found that these neutrophils were not needed to control the virus.

Meanwhile, monocyte immune cells were found to play a protective role and were needed to control the virus and prevent brain damage.

The researchers also identified the exact signaling protein, called CXCL1, that drove the migration of these damaging neutrophils into the brain during HSV infection. By blocking this CXCL1 protein, neutrophils were prevented from crossing the [blood-brain barrier](#) and causing inflammation which resulted in less severe disease.

The findings make the CXCL1 protein an attractive target for new therapies that can stop the influx of damaging white blood cells without limiting the roles of protective ones.

Dr. Michael said: "There is currently no licensed treatment for the severe brain swelling which occurs despite antiviral therapy in HSV encephalitis. Sometimes steroids are given, but as these suppress the [immune system](#) in a very broad way, there is a risk of uncontrolled viral infection.

"There is an urgent need for targeted treatment that prevents damaging immune [cells](#) from entering the brain without limiting the [immune cells](#) needed to control the [virus](#)."

Now Dr. Michael and colleagues are planning to examine the impact of the CXCL1 signaling protein

in patients who have already had steroids as part of a clinical trial led by Professor Tom Solomon at the University of Liverpool.

More information: Benedict D. Michael et al, Astrocyte- and Neuron-Derived CXCL1 Drives Neutrophil Transmigration and Blood-Brain Barrier Permeability in Viral Encephalitis, *Cell Reports* (2020). [DOI: 10.1016/j.celrep.2020.108150](https://doi.org/10.1016/j.celrep.2020.108150)

Provided by University of Liverpool

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