New finding will help target multiple sclerosis immune response

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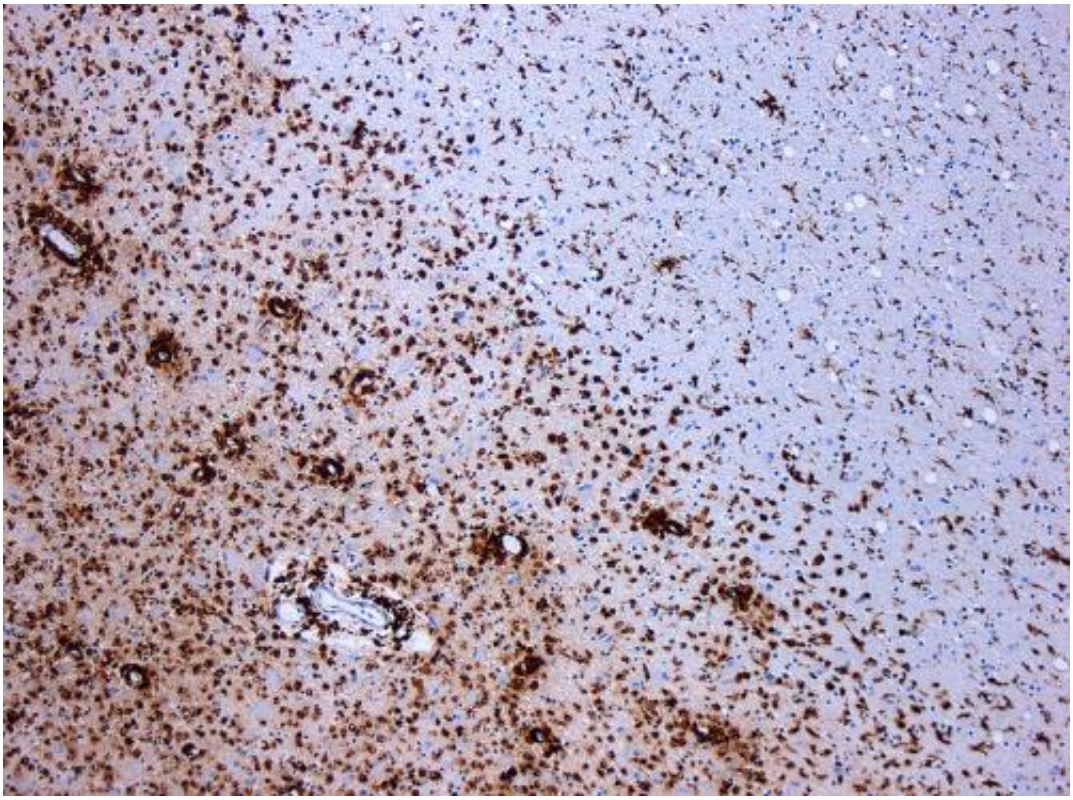

Demyelination by MS. The CD68 colored tissue shows several macrophages in the area of the lesion. Original scale 1:100. Credit: CC BY-SA 3.0 Marvin 101/Wikipedia

Researchers have made another important step in the progress towards being able to block the development of multiple sclerosis (MS) and other autoimmune diseases.

Published today in the journal *Nature Communications*, the researchers at the University of Adelaide have identified a key protein involved in a 'super-inflammatory' immune response that drives the progression of MS and other autoimmune diseases.

The protein is a specific 'chemokine receptor' involved in moving the body's immune response cells, the T-cells, around the body when they are in the super-inflammatory mode needed to fight persistent infections or conversely, as in the case of autoimmune diseases like MS, attacking the body's own tissues. This chemokine receptor, called CCR2, is a different receptor than was widely assumed to be involved.

"Everybody has been focussing on the CCR6 receptor as the one to target to control this inflammatory response," says project leader Professor Shaun McColl, Director of the Centre for Molecular Pathology at the University of Adelaide.

"We've now shown that the receptor to target is actually CCR2. Blocking CCR6 makes the disease worse. If we can find an antagonist to block the CCR2 receptor specifically on these T-cells, we should be able to control the progression of MS."

MS is an incurable neurodegenerative disease, currently affecting 23,000 people in Australia and the most common disease of the central nervous system in young adults.

"We still can't control MS well, there's a great need for new therapies," says Professor McColl.

The University of Adelaide research was conducted by PhD student Ervin Kara under the supervision of Professor McColl and research fellow Dr Iain Comerford, also in the University's School of Biological Sciences.

Another potential benefit of the research is in making improved vaccines to fight infection.

"Unlike in autoimmune diseases, where the body's immune response is destroying its own cells and the aim is to block T-cell migration, with persistent infection we want to turn on the super-inflammatory response and enhance the migration of the immune cells to sites where they are needed," says Professor McColl. "This research may help guide development of vaccines that can better force that immune response."

Provided by University of Adelaide