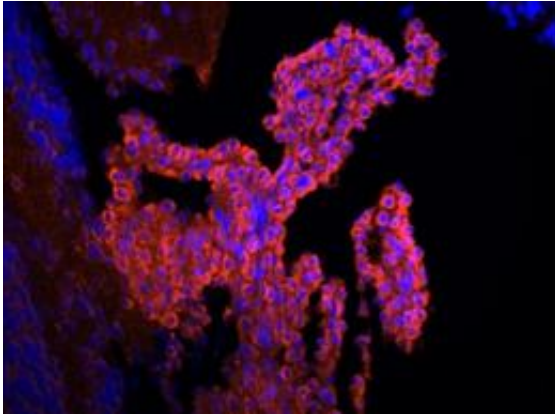


Receptor may hold key to multiple sclerosis treatment

11 June 2012, By Krishna Ramanujan



This image shows the A2A adenosine receptor (in red) abundantly expressed in the central nervous system (CNS). The blue color identifies the nuclei of cells in the CNS.

(Medical Xpress) -- A receptor recently discovered to control the movement of immune cells across central nervous system barriers (including the blood-brain barrier) may hold the key to treating multiple sclerosis (MS), a neuroinflammatory disease of the central nervous system.

In MS, immune cells enter the [central nervous system](#) and attack and destroy the [myelin sheath](#) surrounding the [axons](#) of [nerve cells](#) in the brain and spinal cord, resulting in blindness, paralysis, incontinence and many more symptoms.

The research, appearing last month online and in print June 1 in the *Journal of Immunology*, reveals how the A2A adenosine receptor expressed on blood-brain barrier cells acts as a gateway, allowing immune cells to enter the brain, where they can cause havoc in people with MS.

The blood-brain barrier is composed of specialized cells that selectively prevent substances from passing from the [bloodstream](#) into the brain.

"We found that expression of this A2A adenosine receptor is important for regulating the entry of cells into the brain; whereby its activation allows immune cell entry and its [inhibition](#) blocks entry," said Margaret Bynoe, associate professor of immunology at Cornell's College of Veterinary Medicine and senior author of the paper, which was also selected as a featured publication in the "In This Issue" section of the journal, where the top 10 percent of manuscripts are featured. Jeffrey Mills, a postdoctoral associate in Bynoe's lab, is the paper's lead author.

In this study, the researchers used mice where the A2A adenosine receptor was knocked out and then infused those mice with normal immune cells from wild-type mice expressing the A2A adenosine receptor. This produced chimeric mice expressing the A2A receptor on immune cells, but not on blood-brain barrier cells. Without A2A receptor on blood-brain barrier cells, the normal [immune cells](#) failed to effectively infiltrate the central nervous system, and thus, these mice were protected and developed less severe symptoms of MS-like disease.

"The absence of the A2A receptor on blood-brain barrier cells is similar to the effect of pharmacologically blocking the receptor with antagonists [drugs], which also protected mice from MS-like disease," Bynoe said.

"The implications of these findings are that, potentially, modulation of this receptor can be beneficial for future treatment of MS," she added.

The study was funded by the National Institutes of Health.

Provided by Cornell University

APA citation: Receptor may hold key to multiple sclerosis treatment (2012, June 11) retrieved 24 November 2020 from <https://medicalxpress.com/news/2012-06-receptor-key-multiple-sclerosis-treatment.html>

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