

Break on through to the other side: How HIV penetrates the blood-brain barrier

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Although it is known that HIV can enter the brain early during infection, causing inflammation and memory/cognitive problems, exactly how this occurs has been largely unknown. A new research report appearing in the February 2015 issue of the *Journal of Leukocyte Biology* solves this mystery by showing that HIV relies on proteins expressed by a type of immune cell, called "mature monocytes," to enter the brain. These proteins are a likely drug target for preventing HIV from reaching brain cells. Although not a direct focus of this research, these proteins might also shed light on novel mechanisms for helping drugs penetrate the blood-brain barrier.

"I hope this study brings awareness to the need for adjunctive therapies targeting monocyte influx into the brain as a means to decrease HIV entry into the brain and HIV-associated neurocognitive disorder," said Dionna W. Williams, Ph.D., a researcher involved in the work from the Department of Pathology at the Albert Einstein College of Medicine in Bronx, New York.

To make their discovery, scientists received blood from two groups of people—people infected by HIV and people who were not infected. Mature monocytes were obtained from the blood of people from both groups and researchers determined how many of these cells were present, what proteins the cells expressed and also characterized how they entered into the brain. The researchers found that the mature monocytes had an increased ability to enter into the brain due to the unique proteins they expressed, which could lead to HIV infecting the

brain.

"Monocytes are normal part of the immune system's defense against viruses, but these cells have also been known to act like 'Trojan horses' and carry viruses from the site of initial infection to other parts of the body," said John Wherry, Ph.D., Deputy Editor of the *Journal of Leukocyte Biology*. "Identifying how these cells facilitate HIV entry into the [brain](#) might provide new treatment opportunities not only for HIV but other neurological diseases."

More information: Dionna W. Williams, Kathryn Anastos, Susan Morgello, and Joan W. Berman. JAM-A and ALCAM are therapeutic targets to inhibit diapedesis across the BBB of CD14+CD16+ monocytes in HIV-infected individuals. *J. Leukoc. Biol.* February 2015 97:401-412; DOI: [10.1189/jlb.5A0714-347R](https://doi.org/10.1189/jlb.5A0714-347R)

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