Unlocking Blood-Brain Barrier May Improve Neurological Treatment Outcomes

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Researchers at Cornell's College of Veterinary Medicine may have solved a problem that has long vexed neurologists, including those involved in treating and studying HIV/AIDS-related neurological problems. According to a new report published in the September 14 issue of *The Journal of Neuroscience*, the Cornell team has found a way to open and close the blood-brain barrier, which may allow for more effective treatment of a variety of brain-centered diseases and complications, such as those associated with HIV/AIDS.

The researchers found that adenosine, a molecule produced by the body, can modulate the entry of large molecules—such as those that make up drugs needed to treat neurological disorders, infectious diseases and cancers—into the brain. They discovered that when adenosine receptors are activated on cells that comprise the blood-brain barrier, a gateway through the blood-brain barrier can be established.

Although the study was done on mice, the Cornell team, under the direction of Margaret Bynoe, PhD, also found that adenosine receptors affect the same cells in humans. What's more, they confirmed that an existing FDA-approved drug called Lexiscan (regadenoson), an adenosine-based drug used in heart imaging in very ill patients, can briefly open the gateway across the blood-brain barrier.

The biggest hurdle for every neurological disease—including those related to HIV infection, such as HIV-associated neurocognitive disorder (HAND), AIDS-related dementia and a host of AIDS-related opportunistic infections—is the inability to easily deliver drugs into the brain. This is largely because of the blood-brain barrier, which consists of specialized cells that make up the brain's blood vessels. It selectively prevents substances from entering the blood and brain, only allowing such essential molecules as amino acids, oxygen, glucose and water to pass through.

Researchers have tried to deliver drugs to the brain by modifying them so they would bind to receptors and "piggyback" onto other molecules to get across the barrier. But so far, this modification process has decreased the drugs' efficacy.

According to the paper authored by Bynoe and her colleagues, the researchers were able to

transport large molecules, such as antibodies, into the mice brains. No drugs used to treat any neurological diseases, including antiretrovirals or medications used to manage brain-related opportunistic infections, were studied.

The researchers did, however, successfully deliver anti-beta amyloid antibodies across the blood-brain barrier. These antibodies bind to beta-amyloid plaques, which are known to cause Alzheimer’s disease, at least in the mouse model.

Although there are many known antagonists—drugs or proteins that specifically block signaling—for adenosine receptors in mice, Bynoe and her colleagues note that future work will be needed to identify such drugs for humans.

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