

Researchers link Huntington's disease to overactive immune response in the brain

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The damage to brain tissue seen in Huntington's disease may be caused by an overactive immune response in the bloodstream and the brain, according to new findings from two teams of researchers at the University of Washington in Seattle and University College London. The findings will be published online July 14 in the *Journal of Experimental Medicine*.

Working separately, the two teams found evidence in both brain cells and the bloodstream suggesting an important link between the immune system's response and Huntington's disease. Together, the findings may help scientists find biological markers for monitoring the disease progression earlier and with more accuracy, and could help them develop new treatments for the disease. Huntington's is a fatal inherited neurodegenerative disorder for which there is currently no effective treatment.

The UW team, lead by Dr. Thomas Moeller, research associate professor of neurology, had previously studied the role of inflammation and immune response in neurodegenerative diseases like Huntington's and ALS, also known as Lou Gehrig's disease. In this study, they found that patients with Huntington's had higher levels of immune-system signaling molecules, called cytokines, in their brain tissue.

The UW researchers then looked at a mouse-based model of the disease, studying the response of microglia, the immune cells of the nervous system. When the microglia were treated with a molecule triggering an

immune response, the microglia from Huntington's mice produced much higher levels of cytokines, the immune system molecules. That finding suggests that the protein produced by the Huntington's disease genetic mutation, a protein called huntingtin, is causing the immune cells to be overactive. The researchers think that overly strong immune response may be the mechanism through which the disease causes damage to neurons in the brain.

"When we found increased levels of cytokines in the brains of Huntington's disease patients, we were very excited," Moeller said. "Inflammation in the brain has been increasingly recognized as an important component in other neurodegenerative diseases such as Alzheimer's or Parkinson's disease. These findings might open the door to novel therapeutic approaches for Huntington's disease that target inflammation."

The team at University College London focused their work on immune cells in the bloodstream, and found similar results linking the disease to the body's immune response.

"The similar effect in the blood of Huntington's patients suggests that we have discovered a new pathway in the disease by which the mutant protein could cause damage," Moeller explained. "The protein could be causing damage through an abnormally overactive immune system in both the blood and the brain. While damage from Huntington's is typically seen in the brain, this new pathway is quite easy to detect in the blood of patients, so we may have found a unique window from the blood into what the disease is doing in the brain."

The immune response in the blood may also help researchers use immune-system molecules as biological markers for the disease, which can be difficult to diagnose in early stages. Better tracking of Huntington's disease progression may help researchers to fine-tune

interventions aimed at slowing the disease before it has affected as much brain tissue.

Huntington's affects an estimated 30,000 people in the United States. It is characterized by loss of motor control and cognitive functions, as well as by depression or other psychiatric problems.

Source: University of Washington

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