

Is There a Link Between Herpes and Certain Degenerative Diseases?

Study findings suggest that herpes simplex may trigger the development of Alzheimer's disease, Lou Gehrig's disease and glaucoma.

September 27, 2021 By Jeanette L. Pinnace

In most people with [herpes simplex virus type 1](#) (HSV-1), the virus is quashed before it harms the central nervous system. Now, thanks to [study findings published in the journal Nature](#), researchers have a better understanding of why this happens and how impairment of the mechanism that suppresses the damage might precipitate the development of [neurodegenerative](#) illnesses such as [Alzheimer's disease](#), Lou Gehrig's disease and [glaucoma](#), states [a press release from the University of Illinois at Chicago](#) (UIC).

For the study, scientists used mice from which a protein coding gene called optineurin (OPTN) was excised and infected them with HSV-1. Researchers noted that their [brains](#) showed much higher growth of the [virus](#), which destroyed [neurons](#) and ultimately led to their death.

"Where you have mutated OPTN plus herpes, you have the recipe to create a disaster in terms of neurodegeneration," said Deepak Shukla, PhD, the Marion H. Schenk Esq. Professor in Ophthalmology for Research of the [Aging](#) at UIC.

"The study also shows there is an impairment of immune response when there is a deficiency in OPTN," said Chandrashekhar Patil, PhD, a visiting scholar at UIC's department of ophthalmology and visual science and a coauthor of the study. "OPTN is needed to signal an influx of proper immune cells at the site of infection. When you don't have it, you have issues."

Researchers believe that some of these problems might prompt neurodegenerative illnesses because HSV-1 remains fixed in neurons, setting off a continuous [inflammatory](#) response that damages these cells.

In addition, scientists found that after 30 days, the infected mice were unable to identify familiar objects. Shukla suggested that this inability might show that a deficiency in OPTN in the presence of HSV-1 could speed up injury to the neurons and lead to impaired [cognition](#).

"Part of our translational research can be how can we correct the problems with OPTN so that we don't have issues with neurodegeneration," Shukla said.

Scientists also think that the findings from this inquiry will show that when OPTN is present in sufficient quantity, the gene will be able to suppress the eight herpes viruses that usually affect humans.

Read “Real Health Basics: Alzheimer’s Disease” to learn more about this common neurodegenerative disease and “[Alzheimer’s Disease and African Americans: What You Need to Know.](#)”

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