Common Virus Linked to Possible Alzheimer's Risk

Description

A recent investigation has unveiled an intriguing correlation between a persistent gastrointestinal infection instigated by a ubiquitous virus and the potential onset of Alzheimer's disease in specific populations.

Most individuals encounter cytomegalovirus (CMV) during their formative years. Following the initial viral invasion, CMV establishes itself within the host, typically entering a state of dormancy that may endure for the entirety of life.

By the age of 80, nine out of ten individuals will exhibit the virus's characteristic antibodies within their circulatory system. This pathogen, classified among the herpesvirus family, transmits through various bodily fluids, including breast milk, saliva, blood, and semen, yet remains dormant unless reactivated.

Notably, the study elucidated that in a select cohort, the virus may exploit a biological circumvent, enabling it to sustain an active state for a protracted duration. This activity allows CMV to traverse the gut-brain axis, colloquially termed the vagus nerve.

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Upon reaching cerebral territories, the activated virus possesses the capacity to provoke an immune response, potentially exacerbating the pathophysiological processes contributing to Alzheimer's disease.

This prospect is alarming; however, it connotes that antiviral therapies could play a pivotal role in thwarting the onset of Alzheimer's, particularly if researchers can devise reliable serological assays to promptly identify active CMV infections within the gastrointestinal tract.

Earlier endeavors by researchers from Arizona State University identified a subtype of microglia linked to Alzheimer's disease, designated CD83(+), which correlates with elevated levels of immunoglobulin G4 in the transverse colon, intimating an infectious etiology.

Microglia, the vigilant sentinels of the central nervous system, endeavor to maintain homeostasis by remediating plaques and cellular debris. Nonetheless, incessant activation of these immune cells may culminate in neuronal damage, a hallmark of Alzheimer's pathology. The research was published in *Alzheimer's & Dementia: The Journal of the Alzheimer's Association.*

CATEGORY

1. Health - LEVEL6

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