



Do viral infections set a ticking clock towards neurodegenerative diseases?

Hyderabad, 29th September, 2026: Several emerging studies have linked Influenza and COVID-19 to elevated risks of neurodegenerative diseases like Parkinson's disease. However, the molecular mechanisms underlying this correlation remain unclear. Both Influenza and COVID-19 are infections caused by a class of viruses called RNA viruses that use RNA instead of DNA as their genetic material. A recent [study](#) published in Cell Reports from Dr. Swasti Raychaudhuri's lab at CSIR-Centre for Cellular and Molecular Biology, Hyderabad, explores how these RNA virus infections trigger molecular changes that lead to neurodegeneration and identifies a molecular safeguard against this damage.

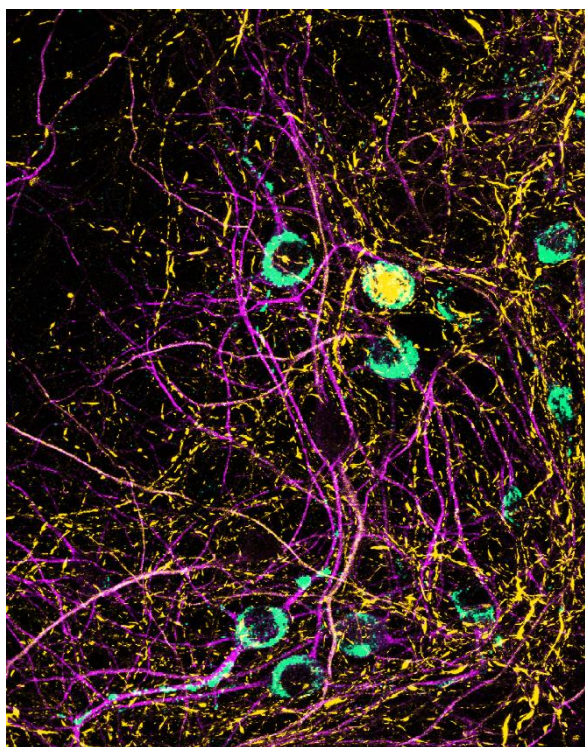


Figure 1: SARS-CoV-2 infected neurons in magenta, viral RNA in teal and α -Synuclein aggregates in yellow

Neurodegenerative diseases are marked by amyloid deposits in the brain – abnormal clumps formed when proteins aggregate together. These amyloids hamper information transmission across neurons, leading to impaired neuronal functioning. One such protein, α -Synuclein, forms amyloid clumps in the brains of patients with Parkinson's disease. This new study dives into how an RNA virus could trigger an increase in α -Synuclein amyloid formation.

RNA viruses fold their genetic material into special secondary structures. These RNAs are not simple thread-like molecules; they have distinct 3D structures called RNA G-quadruplexes (rG4s). When viruses infect cells, these special secondary structures bind to α -Synuclein, accelerating amyloid clump formation, a known hallmark of Parkinson's disease.

Infected cells also have a protective mechanism against the virus. Upon infection, a helicase protein called DDX39A, usually housed in the cell's nucleus, is released into the cytoplasm and binds to α -Synuclein and rG4s. This unwinds the viral rG4s, and offers a line of protection against viral infection. “The virus fails to replicate with its RNA structures dismantled, and thus, the viral load in the cells decreases,” explained Aanchal, the first author of the study. “At the same time, the unwinding of viral RNA's secondary structure effectively slows down α -Synuclein amyloid formation.”

Cells operate with a fine balance of molecules in them, and like every process in life, there exists a trade-off in these biomolecular interactions. “Even if there are protective mechanisms in the cells to prevent viral infections and avoid amyloid formation, there are situations that favor one kind of reaction more than the other. These decide the final outcomes in cells, and sometimes amyloid formation is accelerated in virus-infected cells,” detailed Dr. Raychaudhuri. “So, while not every viral infection

leads to increased amyloid formation and neurodegenerative disease, repeated exposure might tilt the odds against us”. Dr. Raychaudhuri's lab is next working to uncover the finer details of how a routine viral infection might quietly set the stage for Parkinson's disease years later.



Figure 2: Aanchal (left) and Swasti Raychaudhuri (right)