

Fixing a faulty molecular 'transport hub' could slow brain degeneration

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Molecular 'transport hub' protein structure

University of Queensland researchers have gained new insights into how the body sorts and transports protein 'cargo' within our cells, in a finding that could eventually lead to treatments for neurodegenerative diseases

such as Alzheimer's and Parkinson's.

An international research team co-led by Dr Brett Collins from UQ's Institute for Molecular Bioscience has revealed the structure of a molecular transport hub that sorts, directs and transports [protein](#) to correct destinations in the cell.

Dr Collins said protein cargoes that failed to reach the correct destinations in cells created 'traffic jams' that could affect neuronal activity and brain function.

"Having an understanding of how these proteins work together to sort and transport cargo could be the first step in developing drugs that reverse the effects of toxic protein accumulation in neurodegenerative disease," he said.

Dr Collins has been studying how cargo is sorted, packaged, and trafficked within human cells for more than a decade.

He said that developing drugs that fix faulty proteins such as the transport hub was a relatively new and exciting approach to treatment.

"Traditionally, drugs are developed to try to block or inhibit the function of proteins in the body," Dr Collins said.

"The problem with drugs that completely stop the function of a protein is that you often get harmful side-effects."

Dr Collins said the promising finding provided new avenues to target multiple parts of the transport hub to enhance its function by stabilising the protein.

"If we can enhance or improve the function of this protein we could

potentially slow down the brain degeneration that occurs in diseases such as Alzheimer's and Parkinson's," he said.

The study, published in leading scientific journal *Proceedings of the National Academy of Science*, involved scientists from IMB, the University of Bristol's School of Biochemistry, and The Henry Wellcome Integrated Signalling Laboratories.

More information: Matthew Gallon, Thomas Clairfeuille, Florian Steinberg, Caroline Mas, Rajesh Ghai, Richard B. Sessions, Rohan D. Teasdale, Brett M. Collins, and Peter J. Cullen. "A unique PDZ domain and arrestin-like fold interaction reveals mechanistic details of endocytic recycling by SNX27-retromer." *PNAS* 2014 111 (35) E3604-E3613; published ahead of print August 18, 2014, [DOI: 10.1073/pnas.1410552111](https://doi.org/10.1073/pnas.1410552111)

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