

# Dementia study sheds light on how damage spreads through brain

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Insights into how a key chemical disrupts brain cells in a common type of dementia have been revealed by scientists.

Brain tissue from people with Dementia with Lewy Bodies (DLB)

showed that the protein builds up in vital parts of neurons that connect [cells](#) and may jump from one cell to another through these connections.

Scientists say the findings shed light on the causes of DLB and will help to accelerate the search for a treatment.

The study, co-led by the University of Edinburgh, focused on synapses - shared connection points between [brain](#) cells that allow chemical and electrical signals to flow between cells. These signals are vital for forming memories and are key to brain health, experts say.

Researchers showed that synapses in five people who had died with DLB contained clumps of the damaging protein, known as [alpha-synuclein](#), which could contribute to [dementia](#) symptoms.

Toxic alpha-synuclein was spotted in both sides of the synapses, suggesting that it may jump between cells through these connections. This sheds light on how damage could be spread through the brain.

Similar findings were not seen in [brain tissue](#) from people who had died with Alzheimer's disease or those without dementia.

The discovery was made with extremely powerful technology, used in DLB for the first time, which allowed the scientists to view detailed images of over one million single synapses. Individual synapses are around 5000 times smaller than the thickness of a sheet of paper.

Although alpha-synuclein clumps had been previously identified in DLB, their effects on synapses were unknown because of difficulties in studying them due to their tiny size.

DLB is the third most common form of dementia after Alzheimer's and vascular dementias, affecting around 100,000 people in the UK. It can

cause severe memory loss as well as movement problems and there is no cure.

Professor Tara Spire-Jones, Programme Lead at the UK Dementia Research Institute at the University of Edinburgh, who co-led the study, said: "DLB is a devastating condition and our findings suggest that it is at least partly driven by damage to synapses. These discoveries should invigorate the search for therapies aimed at reducing synaptic damage and open the possibility of targeting the spread of alpha-synuclein through the brain, which could stop disease progression in its tracks."

Dr Rosa Sancho, Head of Research at Alzheimer's Research UK, said: "This exciting research using cutting-edge technology sheds new light on the progression of DLB in the brain. The results provide convincing, measurable and visual evidence that toxic alpha-synuclein is disrupting [synapses](#) that could potentially contribute to the devastating symptoms of the disease.

"We are extremely pleased our funding has helped produce these important results which demonstrate potential avenues for much-needed new treatments for people who are living with DLB. The research we fund would not be possible without the work of our tireless supporters who go to extraordinary lengths to allow talented researchers to make important new discoveries like these."

**More information:** *Brain* (2017). [DOI: 10.1093/brain/awx275](https://doi.org/10.1093/brain/awx275)

Provided by University of Edinburgh

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