Editorial Alzheimer's could be stopped from Progressing

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Alzheimer's disease spreads in a predictable pattern like an infection, going from one brain cell to another along linked circuits known as synapses, researchers say.

The findings, published in the online journal PloS One, suggest that blocking the process early on may keep the disease from spreading.

'This is a phenomenon that is increasingly recognised and potentially very important,' said Dr Samuel Gandy, of the Mount Sinai Alzheimer's Disease Research Center in New York.

'If we understood this process, we could potentially arrest progression at an early stage.'

Imaging studies in people have suggested that Alzheimer's spreads from region to region in the brain rather than popping up spontaneously in different areas, but the evidence was not strong enough to say for sure.

'Everyone talks about Alzheimer's 'spreading', but there really has not been a standard theory,' study authors Dr Karen Duff and Dr Scott Small from the Columbia University Medical Center in New York, said.

'In the past, we have asked many of our colleagues in the field of Alzheimer's research what they mean when they say 'spread'. Most think that the disease just pops up in different areas of the brain over time, not that the disease actively jumps from one area to the next,' they said.

'Our findings show for the first time that the latter might be true.'

More than five million Americans and 465,000 people in the UK suffer from Alzheimer's, a brain disease that causes dementia.

Despite costly efforts, no drug has been found that can keep the disease from progressing.

There is currently no cure for Alzheimer's, which is a progressive condition and most common in people over 65.

For their study, the team used mice that were genetically engineered to accumulate deposits of tau in a key memory center of the brain known as the entorhinal cortex, which is where that toxic protein starts to deposit in people.

Their aim was to map the progression of tau, an abnormal protein that forms tangles of protein fibers in the brains of people with Alzheimer's disease.

The team analysed the brains of the mice periodically over a period of 22 months to see how the disease progressed.

They found that as the mice aged, the abnormal human tau spread along a linked pathway, traveling from the entorhinal cortex to the hippocampus to the neocortex, areas of the brain needed to form and store memories.

That pattern closely follows the progression of Alzheimer's as it passes through various stages in people, Dr Duff said.

The team also saw signs that tau moved from brain cell to brain cell across synapses, connection points that allow nerve cells to communicate.

The researchers think those findings suggest new strategies for diagnosing and treating Alzheimer's disease.

'First, it would suggest that imaging tools that can detect entorhinal cortex dysfunction will be particularly helpful in diagnosing the earliest stages of the disease,' they said.

'More importantly, it might suggest ways of improving treatment.

'The implication of our study is that if it were possible to 'treat' Alzheimer's when it was first detected in the entorhinal cortex, this would prevent spread,' they said.

They likened the approach to treating cancer early, when it is still in one spot, and not waiting until it has spread.

The study may bring a new focus to diagnostics and treatments that focus on tau, rather than amyloid, the protein that causes plaques to form in the brain.

Current imaging agents used with PET scanners can identify amyloid deposits in the brain, but not tau.

Most late-stage Alzheimer's drugs, including Eli Lilly and Co's solanezumab, and Johnson & Johnson and Pfizer's bapineuzumab, take aim at amyloid, which accumulates silently 15 to 20 years before signs of dementia appear.