Epilepsy drug shows potential for Alzheimer's treatment

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A drug commonly used to treat epilepsy could help clear the plaques in the brain associated with Alzheimer's disease, according to researchers at the University of Leeds. The plaques are known to lead to the progressive death of nerve cells in the brain linked to many forms of dementia.

Sodium valproate - which is marketed as the anti-seizure drug Epilim - has been shown by scientists at the University of Leeds to reactivate the body's own defences against a small protein called amyloid beta peptide, which is the main component of the brain plaques characteristic in Alzheimer's. Their work was funded by the Medical Research Council.

"The fact that we've been able to show that a well-established, safe and relatively inexpensive drug could help treat Alzheimer's is an extremely exciting development," says lead researcher Professor Tony Turner from the University's Faculty of Biological Sciences. "We hope colleagues will be able to progress this research with clinical trials in the near future."

Alzheimer's disease is the most common form of dementia and has no cure. In the UK today there a half a million people living with Alzheimer's – and this is likely to double within a generation unless new treatments are found.

Sodium valproate has been used for many years to suppress epileptic seizures and the many sufferers of epilepsy have been taking the drug for decades with few side effects.

The development of Alzheimer's is widely believed to be caused by the gradual accumulation in the brain of amyloid-beta peptide which is toxic to nerve cells. This is thought to be caused by a key enzyme called neprilysin or NEP gradually switching off in later life. One of NEP's roles is to clear the toxic peptide from the brain, and plaques begin to form as it gradually switches off, leading to the death of the brain's nerve cells.

The research team examined changes in chromatin – the 'packaging' that genes are contained within - and surmised that these changes might be involved in switching off NEP. The team found clear differences (acetylation) in key proteins within the chromatin when they compared normal nerve cells against those that failed to produce NEP.

"From there it was relatively simple to stimulate the expression of NEP with sodium valproate, which was seen to prevent the acetylation," says Professor Turner. "We were elated when we saw the results."

Source: University of Leeds