Drugs being developed to tackle CJD could also help block Alzheimer's, research shows

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(Medical Xpress) -- Scientists funded by the Medical Research Council UK (MRC) and Science Foundation Ireland (SFI) have identified two antibodies which could help block the onset of Alzheimer's disease in the brain.

The antibodies, ICSM-18 and ICSM-35, were already known to play a crucial role in preventing 'protein misfolding'; the main cause of Creutzfeldt–Jakob disease (CJD), the human form of mad cow disease.

This discovery represents a significant step forward in the battle to develop drugs to treat Alzheimer's disease- a devastating neurodegenerative illness which affects more than 20 million people worldwide.

The work was carried at the Medical Research Council Prion Unit at University College London, in collaboration with colleagues at the Laboratory for Neurodegeneration at University College Dublin, and Trinity College Dublin.

The study findings published in Nature Communications has shown, using mice, that these antibodies can block damaging effects on brain tissue caused by a toxic substance called 'amyloid beta'. Cumulatively, amyloid beta becomes attached to the surface of nerve cells in the brain, stopping them from communicating effectively and causing memory loss. The results showed that the antibodies stopped the amyloid beta proteins from taking hold and damaging the brain.

The study also confirms findings from a 2009 paper by Yale researchers, which first indicated that prion proteins, the proteins which can change their shape and cause CJD, may be involved in Alzheimer's.

"With an ageing population and increasing numbers of families affected by Alzheimer's disease, there is an urgent need for new drugs which will help to preserve brain function and prevent memory loss, the symptom which most characterises the devastating impact of Alzheimer's," said Professor John Collinge, Director of the MRC Prion Unit at University College London, who led the study.

"We're thrilled that this discovery shows in mice that these two antibodies which we are developing to treat CJD may also have a role in treating more common forms of dementia like Alzheimer's disease. If these antibody drugs prove to be safe in use to treat CJD we will consider whether studies in Alzheimer's disease should be carried out."

"A unique aspect of this study is that we used amyloid beta extracted from human brain, the same material we believe is causing memory loss in patients with this devastating disease and we identified two antibodies that could block this effect," said Professor Dominic Walsh, Professor of Neurodegeneration at the UCD School of Biomolecular and Biomedical Science, and the UCD Conway Institute, University College Dublin who was co-corresponding author on the research.

"The use of these specific antibodies is particularly exciting since they have already undergone extensive pre-clinical testing for use in treating CJD. Thus a lot of basic work has already been done and could fast-track these antibodies for use in humans. The next step is further validation in other disease models of Alzheimer's and then safety trials in humans," he added.

Clinical trials to see whether drugs based on these antibodies can

mitigate the damage caused to the human brain as a treatment for patients with CJD are due to begin in 2012.

More information: "Interaction between prion protein and toxic amyloid beta assemblies can be therapeutically targeted at multiple sites" is published online in *Nature Communications.* www.nature.com/ncomms/index.html

Provided by University College Dublin