How culprit Alzheimer's protein wreaks havoc

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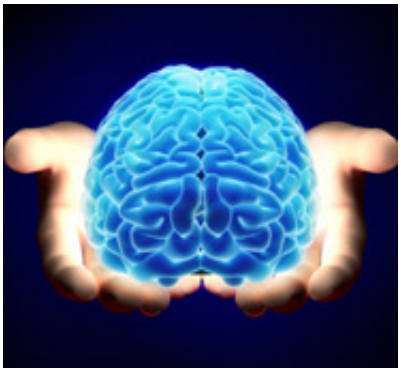

(PhysOrg.com) -- How the toxic protein, amyloid, sets off a chain of events that leads to brain cell death during Alzheimer's disease is described in new detail in a study from the University of Bristol published today in *Nature Neuroscience*. The research, part-funded by Alzheimer's Research UK, uncovers a raft of new targets for treatment development.

The hallmark amyloid protein has been a focus for worldwide Alzheimer's research since it was first linked to the disease in the 1980s. Now, the Bristol team has shed light on how amyloid causes other proteins to change their behaviour in nerve cells. This ultimately leads the nerve cells to stop functioning properly in brain. The study, overseen by Professor Kei Cho, was conducted in part by promising PhD student

Daniel Whitcomb, also funded by Alzheimer's Research UK.

Professor Kei Cho, who led the research at the University of Bristol, explains: "We have discovered a critical chain of events, triggered by amyloid, which damages nerve cells. We found that the toxic amyloid protein affects the behaviour of other proteins in the brain, causing them to malfunction. This finding could help to explain the memory deficit that has such a profound effect on people with Alzheimer's. Each of these newly linked proteins provide important clues for treatment development -- if we can disrupt this fateful chain of events in the brain, we might be able to protect against Alzheimer's disease."

Dr Simon Ridley, Head of Research at Alzheimer's Research UK, the UK's leading dementia research charity, said: "It's essential to find out how our brains work, how nerve cells function, and how these processes go wrong in diseases like Alzheimer's. Armed with this knowledge we can develop new and effective treatments that are so desperately needed.

"To develop new treatments for Alzheimer's and other dementias, we must invest in research now. Over 4000 people in Bristol alone have dementia, a number forecast to grow as our population ages."

Provided by University of Bristol