Medical Xpress

Researchers find protein deposits linked to Alzheimer's disease behave like prions

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(Medical Xpress) -- Researchers from the University of California have found that a peptide that forms deposits in the human brain and is thought to be responsible for the onset of Alzheimer's disease, behaves in ways that are very similar to the way prions behave when propagating through mammalian neurological tissue. In their paper published in the *Proceedings of the National Academy of Sciences* describing their research into the ways amyloid-β (Aβ) peptides form deposits, the team found that they propagate across brain tissue in much the say way prions do when causing ailments such as Creutzfeldt-Jakob disease (CJD).

Prions, short for "protein infection" are neither bacterial nor virus and instead are defined as a somewhat mysterious condition, rather than as an infection, despite the fact that diseases that are caused by them are communicable, e.g. mad cow disease. Instead of an infectious agent, cells in the body simply react to the sudden presence of an abnormally folded protein by folding in a likewise manner, propagating across brain or nerve tissue until the victim succumbs. Sadly, scientists don't yet know how they really do their work and thus can offer no cure for those afflicted.

In this new research, the team introduced the peptide amyloid-β (Aβ) along with a florescent molecule, into just one side of the brain of several mice and then watched what happened over nearly a year's time. Because of the florescent molecule, the team was able to track the progress of the peptide as it propagated to the other side of the brain, eventually damaging the entire structure. This, the team says, suggests

that Aβ is either a prion or something that acts an awful lot like one. There's one hitch though, diseases caused by prions are generally contagious and thus far there is no reason to believe that Alzheimer's disease can be passed from person to person.

Whether it is a prion or isn't, researchers will likely approach research into Alzheimer's disease with a different view now that it's known that the disease starts in one part of the brain and propagates to others, rather than simply cropping up in small bits all over the brain and progressing to a worse state as time passes, as has been thought to be the case up till now.

More information: Purified and synthetic Alzheimer's amyloid beta (Aβ) prions, *PNAS*, Published online before print June 18, 2012, doi: 10.1073/pnas.1206555109

Abstract
The aggregation and deposition of amyloid-β (Aβ) peptides are believed to be central events in the pathogenesis of Alzheimer's disease (AD). Inoculation of brain homogenates containing Aβ aggregates into susceptible transgenic mice accelerated Aβ deposition, suggesting that Aβ aggregates are capable of self-propagation and hence might be prions. Recently, we demonstrated that Aβ deposition can be monitored in live mice using bioluminescence imaging (BLI). Here, we use BLI to probe the ability of Aβ aggregates to self-propagate following inoculation into bigenic mice. We report compelling evidence that Aβ aggregates are prions by demonstrating widespread cerebral β-amyloidosis induced by inoculation of either purified Aβ aggregates derived from brain or aggregates composed of synthetic Aβ. Although synthetic Aβ aggregates were sufficient to induce Aβ deposition in vivo, they exhibited lower specific biological activity compared with brain-derived Aβ aggregates. Our results create an experimental paradigm that should lead to identification of self-propagating Aβ conformations,

which could represent novel targets for interrupting the spread of Aβ deposition in AD patients.

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