Leading Alzheimer's theory challenged by mounting research

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Drugs that target beta-amyloid, the molecule widely considered the cause of Alzheimer's disease, make neurons function worse — at least in mice — explaining why anti-amyloid compounds have failed to improve memory or cognition in clinical trials.

For nearly a quarter-century, the leading explanation of Alzheimer's disease has been that a protein called beta-amyloid (written ?-amyloid) is deposited around brain neurons, forming sticky "plaques" that eventually kill cells, destroy synapses, erase memory, and cripple cognition. The amyloid hypothesis has been so dominant — virtually every pharmaceutical and biotech company trying to develop an Alzheimer's drug is on the amyloid warpath — that scientists pursuing other explanations have warned that "The Church of the Holy Amyloid" has choked off research on competing ideas. The real tragedy has been the repeated failures of experimental compounds targeting amyloid: They often clear away amyloid but don't help symptoms.

In a new study, scientists injected a form of the Pfizer-J&J anti-amyloid antibody, or a similar one from Novartis, into mice whose brains were riddled with ?-amyloid plaques. The Pfizer-J&J compound sopped up amyloid in the mice; Novartis's had less effect. But the shocker was that both antibodies made neurons become hyperactive, reported Marc Busche of Technical University of Munich and colleagues (including two from Novartis) in Nature Neuroscience.

Read full, original post: Top Alzheimer's approach makes (mice) brains worse