

Anti-inflammatory drug blocks brain plaques

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Brain destruction in Alzheimer's disease is caused by the build-up of a protein called amyloid beta in the brain, which triggers damaging inflammation and the destruction of nerve cells. Scientists had previously shown that preventing individual amyloid beta proteins from sticking to one another minimized brain lesions and protected nerve cells against damage.

The new study—a collaborative effort by researchers in Germany and the US—shows that an anti-inflammatory drug (called CNI-1493) may have the same effect. The drug—already tested in humans for the treatment of inflammatory diseases—protected nerve cells against amyloid beta—induced damage in culture. In mice prone to developing an Alzheimer's-like disease, the drug decreased brain inflammation and improved memory and cognitive function.

Other anti-inflammatory drugs, such as ibuprofen, have been shown to reduce Alzheimer's disease lesions in the brains of rodents, but CNI-1493 appears to be faster and more effective. If these results hold up in humans, CNI-1493 may provide a more effective alternative to current Alzheimer's therapies, which temporarily prolong the function of nerve cells but do not prevent their destruction.

Source: Journal of Experimental Medicine

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