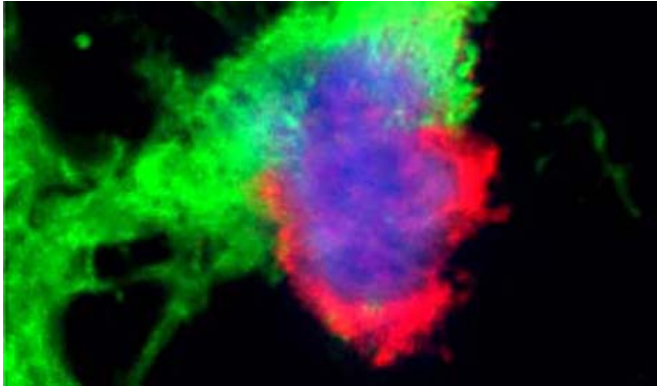


Immune cells are an ally, not enemy, in battle against Alzheimer's

29 January 2015, by Bill Hathaway



The cell in green is a microglia, an immune system cell that is blocking free floating beta amyloid (in red) as it binds to amyloid plaque (blue), the hallmark of Alzheimer's Disease. Yale researchers found that rather than a marker for damaging inflammation, these cells protect the brain from free floating amyloid.

Beta-amyloid is a sticky protein that aggregates and forms small plaques in the brains of the elderly and is thought to be a cause of Alzheimer's disease. Because specialized immune cells always surround these plaques, many have theorized that these cells are responsible for inflammation and damage to surrounding brain cells.

That theory appears to be wrong, according to a new study by Yale School of Medicine researchers published in the Jan. 29 issue of *Nature Communications*. Instead of causing damage, these brain [immune cells](#)—called microglia—seem to protect the brain by keeping [amyloid plaques](#) corralled, the paper shows.

Previous research had shown that some people with large accumulations of plaques do not necessarily have symptoms of dementia, which led researchers to search for other causes of [cognitive decline](#). Inflammation was one of potential culprits identified by scientists. However Alzheimer's drugs

that target inflammation in the brain have failed to show any benefit.

"The idea that [inflammation](#) is always bad is a simplistic view and is probably wrong when talking about Alzheimer's," said Jaime Grutzendler, associate professor in the Department of Neurology and senior author of the study. "In fact, as we age we lose microglia and become less able to confine plaques, leading to the release of plaque toxins that destroy the connections between neurons."

The new study using high-resolution imaging technology revealed that in the brains of mice, microglia actually act as a physical barrier that slows the expansion of plaques and blocks the ability of free-floating beta-amyloid proteins to bind to the plaques and cause toxicity.

"One possibility is that microglia nicely insulate the rest of the brain from plaques and may explain why some people with them do not experience severe cognitive decline," Grutzendler said. "By improving microglia's shielding function, we were able to reduce toxicity to neurons."

This insight could lead to new treatments for the disease, he added.

Provided by Yale University

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