

# Novel monoclonal antibodies show promise for Alzheimer's disease treatment

20 July 2015

Scientists at NYU Langone Medical Center's Center for Cognitive Neurology have evidence that monoclonal antibodies they developed may provide the blueprint for effective treatments for Alzheimer's disease and other neurodegenerative diseases, such as Parkinson's disease.

A team led by Fernando Goni, PhD, an adjunct associate professor of Neurology, and Thomas Wisniewski MD, director of the Center for Cognitive Neurology at NYU Langone, showed that a novel class of [monoclonal antibodies](#) successfully targeted proteins that change shape and misfold, becoming toxic and triggering the hallmark beta-amyloid plaques and abnormal tau proteins that are known to accumulate in Alzheimer's and other neurodegenerative conditions. The monoclonal antibodies were also successful at targeting the proteins linked to Parkinson's development.

The research team's findings are to be released at the Alzheimer's Association International Conference 2015, July 19, 2015 in Washington, D.C.

Monoclonal antibodies are antibodies produced by only one single, specific type of cell and all have the same activity. They can be purified and infused in any organism to produce a desired effect.

The new research suggests that monoclonal antibodies designed to specifically target these misfolding proteins in soluble, aggregated states, may be ideally suited to treating [neurodegenerative diseases](#).

"There is a commonality underlying the misfolding in many neurodegenerative diseases and we are targeting it. We are confident this is the right strategy and our monoclonals are showing they are up to the task," says Dr Goni. "There is potential for specific therapeutic agents for neurodegenerative diseases."

Previous research has established that most neurodegenerative diseases including Alzheimer's, Lewy Body and other dementias, Parkinson's and prion diseases develop and progress along similar paths. In each disease, a particular protein undergoes a change in its shape from a soluble, physiologically functional protein to a protein that has lost the ability to perform its required tasks in the brain, starting off a chain reaction of binding to each other with little control. These aggregates become toxic to brain cells.

In previous studies, Drs. Goni and Wisniewski tested their theory that attacking an early form of these proteins when they change their shape could prevent their formation into aggregates that lead to plaques and tangles, or neutralize their capacity to spread throughout the brain, and stop the progression of a particular neurological disease. They found that their monoclonal antibodies reacted to an intermediate, or "oligomer" state of the amyloid and tau proteins seen in Alzheimer's disease, as well as to prion disease proteins.

In their new study, they determined the monoclonal antibodies' binding specificity to oligomeric forms of a protein called alpha-synuclein, which accumulates and presents in Lewy body containing neurons of Parkinson's disease patients.

Researchers tested three different monoclonal antibodies, each of which binds to amyloid and tau and also reverses Alzheimer's-like damage to brain tissue in animals. They found that all three monoclonal antibodies bind to the oligomeric forms of alpha-synuclein. Then, they confirmed the antibodies' affinity for these structures within neurons using brain tissue samples from people with Parkinson's disease and Alzheimer's.

"We have been developing this strategy for many years, and now we have results. Other labs are trying similar strategies", says Dr Wisniewski, the Lulu P. and David J. Levidow Professor of

Neurology and a professor of Pathology and Psychiatry. "The importance of this concept is being increasingly recognized."

Researchers have plans for further animal tests of their monoclonal antibody regimen, on its own and in combination with other approaches, before proceeding to clinical trials.

Alzheimer's disease and other dementias affect 47 million people worldwide and 5.3 million Americans, numbers that are expected to triple by 2050, according to the Alzheimer's Association. This surge is expected to put a tremendous strain on the health care system unless better screening and treatment methods are identified.

Provided by New York University School of Medicine

APA citation: Novel monoclonal antibodies show promise for Alzheimer's disease treatment (2015, July 20) retrieved 12 October 2022 from <https://medicalxpress.com/news/2015-07-monoclonal-antibodies-alzheimer-disease-treatment.html>

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