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Protective pathway identified to counter toxicity associated with Alzheimer's disease

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New research led by Marco Prado, PhD, of Western University has identified a pathway used by the brain to try to protect itself from toxicity that occurs with Alzheimer's disease (AD). Prado and his colleagues at the Robarts Research Institute and at the A.C. Camargo Cancer Center in Brazil have done extensive work on the role of prion protein. They found that toxicity of amyloid-β peptides, one of the major culprits in AD, can be decreased by preventing it from interacting with the prion protein. When a protein called stress-inducible phosphoprotein 1 (STI1) interacts with the prion protein preventing the interaction of amyloid-β peptides, it protects neurons. The research is now published in the *Journal of Neuroscience*.

"What's interesting is when we studied the brains of people with AD, we saw that, compared to age and gender-matched controls, STI1 levels are increased. We think of this increase as a compensatory response that could protect against insults from this amyloid-β. So we tested it in mouse neurons to see what happens if you increase or decrease the levels of STI1," says Prado, a professor in the Departments of Physiology and Pharmacology and Anatomy and Cell Biology at Western's Schulich School of Medicine & Dentistry. "When we decreased the levels of STI1, the neurons became more sensitive to attack by amyloid-β, and when we increased the STI1, it seemed to mitigate or decrease the toxicity of amyloid-β."

"You have to think of AD as one big puzzle. Every year scientists are putting new pieces in, and now we found that STI1 is one of the

pathways in the puzzle," says Prado. "The disease is very complex, but new clues on how we may be able to help the brain to resist the toxins in AD are starting to emerge. We are starting to see the big picture. It may still take years, but we're going to put this puzzle together and find a way to slow down or cure AD."

Provided by University of Western Ontario