Gene regulator plays role in Alzheimer’s

Along with symptoms of cognitive decline, Alzheimer’s disease patients often have an accumulation of plaques and tangles of proteins in parts of their brains. But a long-standing question in neurology is why some elderly people develop dementia and others do not. Researchers are also left to wonder why some people have Alzheimer’s disease-like brain pathology yet show no cognitive symptoms.

A study published March 19 in Nature provides new clues that could help solve both puzzles, showing that a previously unknown stress response kicks in later in life to protect aging neurons. Researchers at Harvard Medical School found that a protein called REST, which is well characterized as a transcription factor that represses neuron-specific genes during embryogenesis, is switched on during middle- and late-adulthood, helping to protect neurons of the hippocampus and cortex from oxidative stress and the aggregated and misfolded proteins characteristic of Alzheimer’s and other neurodegenerative diseases.

“This work establishes REST as a regulator protein we have to pay a lot of attention to in the context of neurodegeneration,” said Susan Lindquist, a molecular biologist at the MIT Whitehead Institute for Biomedical Research, who was not involved in the study.

Read the full, original story: Protein Protects Aging Brain