Molecule linked to Alzheimer's

Israeli researchers find way to improve memory in lab rats

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Researchers from the University of Haifa have found a way to improve memory through manipulation of a molecule known to malfunction in old age - and that is closely linked to Alzheimer's disease.

In their research, which was carried out on rats by the neurobiology department at the university and recently published in the Journal of Neuroscience, the researchers report that they managed for the first time to maneuver the activity of the molecule, a protein called PERK, without doing harm to cognitive functioning.

"We know that in Alzheimer's disease, the PERK protein acts improperly," explained Kobi Rosenblum, who directs the University of Haifa laboratory where the study was carried out. "Our success in maneuvering its expression without causing harm to normal brain function also opens the way for us to improve memory and perhaps also slow development of the pathology of diseases such as Alzheimer's."

Prior studies show that the process of creating memory in the brain is connected to protein synthesis, in which the rapid production of proteins leads to "strong" memory capabilities that are maintained over the long term, while slower protein production results in "weak" or fuzzy memory that in many instances never coalesces into long-term memory and in practice is forgotten.

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In the current study, researchers Hadile Ounallah-Saad and Viendra Sharma from Rosenblum's lab sought to research the activity of the elF2 alpha protein, which is known to set the pace at which proteins in the brain are synthesized when memory is created. Prior studies demonstrated that three molecules act on the protein, either activating or delaying it.

As a result, in the first stage of the current research, the study examined the relative effect of each of the molecules that control the elF2 alpha protein, and then what the effects would be on the creation of memory. After investigating at the level of cell tissue, the researchers discovered that the main molecule that controls the protein's activity is PERK.

"The fact that we identified PERK as the main controlling [substance] was particularly important," said Dr. Ounallah-Saad. "First we identified the dominant component. Second, from prior studies, we already knew that in degenerative diseases such as Alzheimer's, the PERK malfunctions. Thirdly, the PERK functions in various cells, including neurons, as a monitor of metabolic stress. In other words, we found a molecule of central importance in the process of creation and consolidation of memory and that we know malfunctions in Alzheimer's disease."

The researchers were then able to stimulate the PERK activity, resulting in a 30% improvement of memory in the laboratory rats. "Because it involves a molecule that malfunctions in Alzheimer's [patients], and is related to aging, we are in effect opening up the possibility of developing medications that can slow the progress of incurable illnesses such as degenerative brain diseases."