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'Longevity gene' helps prevent memory decline and dementia (w/ Video)

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Scientists at Albert Einstein College of Medicine of Yeshiva University have found that a "longevity gene" helps to slow age-related decline in brain function in older adults. Drugs that mimic the gene's effect are now under development, the researchers note, and could help protect against Alzheimer's disease.

The paper describing the Einstein study is published in the January 13 edition of the Journal of the American Medical Association.

"Most work on the genetics of Alzheimer's disease has focused on factors that increase the danger," said Richard B. Lipton, M.D., the Lotti and Bernard Benson Faculty Scholar in Alzheimer's Disease and professor and vice chair in the Saul R. Korey Department of Neurology at Einstein and senior author of the paper. As an example, he cites APOE ε4, a gene variant involved in cholesterol metabolism that is known to increase the risk of Alzheimer's among those who carry it.

"We reversed this approach," says Dr. Lipton, "and instead focused on a genetic factor that protects against age-related illnesses, including both memory decline and Alzheimer's disease."

In a 2003 study, Dr. Lipton and his colleagues identified the cholesteryl ester transfer protein (CETP) gene variant as a "longevity gene" in a population of Ashkenazi Jews. The favorable CETP gene variant increases blood levels of high-density lipoprotein (HDL) - the so-called good cholesterol - and also results in larger-than-average HDL and low-

density lipoprotein (LDL) particles.

The researchers of the current study hypothesized that the CETP longevity gene might also be associated with less cognitive decline as people grow older. To find out, they examined data from 523 participants from the Einstein Aging Study, an ongoing federally funded project that has followed a racially and ethnically diverse population of elderly Bronx residents for 25 years.

At the beginning of the study, the 523 participants - all of them 70 or over - were cognitively healthy, and their blood samples were analyzed to determine which CETP gene variant they carried. They were then followed for an average of four years and tested annually to assess their rates of cognitive decline, the incidence of Alzheimer's disease and other changes.

"We found that people with two copies of the longevity variant of CETP had slower memory decline and a lower risk for developing dementia and Alzheimer's disease," says Amy E. Sanders, M.D., assistant professor in the Saul R. Korey Department of Neurology at Einstein and lead author of the paper. "More specifically, those participants who carried two copies of the favorable CETP variant had a 70 percent reduction in their risk for developing Alzheimer's disease compared with participants who carried no copies of this gene variant."

The favorable gene variant alters CETP so that the protein functions less well than usual. Dr. Lipton notes that drugs are now being developed that duplicate this effect on the CETP protein. "These agents should be tested for their ability to promote successful aging and prevent Alzheimer's disease," he recommends.

Provided by Albert Einstein College of Medicine

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