Massive transcriptome analysis of genes in the hippocampus, a brain region involved in encoding and storage of declarative memory that is affected in AD, revealed altered expression of a gene program essential for neuronal function coinciding with the first memory deficits. These findings were verified also in human brains at early pathological stages of the disease. Surprisingly, the researchers found that the molecule that regulates this set of genes is the transcriptional coactivador Crtc1, a protein that regulates genes involved in glucose metabolism and cancer. In the hippocampus, Crtc1 is activated during memory processing, but its inactivation causes alteration of a gene program essential for memory storage disturbing the capability of a person to remember correctly says Dr. Saura. According to this scientist “this study opens new perspectives in the prevention and therapeutic treatment of AD, since we have demonstrated that a gene therapy that activates Crtc1 is effective to prevent memory impairment in a mouse model of AD”. The study states that one of the future goals for the treatment of this devastating disease is the development of pharmacological therapies that activate Crtc1 to prevent, ameliorate or reverse cognitive impairments in AD patients. These future investigations will be possible after funding by an American foundation aimed to combat AD and other neurological disorders.

Publication of reference: