

MCFIP - The document affixed to this article provides a testable explanation for how and why presenilin can be a regulator for familial AD.

<https://medicalxpress.com/news/2019-10-key-gene-familial-alzheimer-disease.html>

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Key gene in familial Alzheimer's disease regulates neuronal development

by Autonomous University of Barcelona

An international team of researchers led by the Institute of Neuroscience at the Universitat Autònoma de Barcelona describes a new route that promotes differentiation of neurons. The discovery demonstrates that there is a common mechanism between the neurodegeneration occurring in Alzheimer's and the proliferation of cancer cells.

Age is the most important factor in the development of neurodegenerative diseases such as Alzheimer's and cancer. Moreover, [epidemiological studies](#) indicate that there is an inverse correlation between suffering from Alzheimer's disease and cancer, although the mechanisms connecting these two pathologies remain unknown.

An international team of researchers led by Dr. Carles Saura from the Institute of Neuroscience at UAB has unravel a mechanism crucial for the growth of neuron axons, a key process for the correct development of the [brain](#).

According to the study, published in *eLife*, this mechanism requires presenilin, the main gene mutated in familial Alzheimer's disease. Its malfunction in this disease is what causes the abnormal accumulation of beta-amyloid in the brain in Alzheimer's [disease](#) patients. In this study, researchers demonstrate that presenilin is not only essential for regulating growth of neurons during [brain development](#), but that it achieves this through the EphA3 receptor, a protein involved in several cancers. The relevance of this study is that it demonstrates the existence of a new cell mechanism that connects neurodegeneration with cancer.

"The discovery of this new signalling route is very relevant in the study of neurological disorders in which the morphology of the neuron axon is altered. Indeed, the implications of the study go beyond the brain given that the mechanism involves the EphA receptors, which play a key role in cancer", explains first author of the study Dr. Míriam Javier. According to Dr. Saura, "this research allows us to be optimistic about the development of common therapeutic strategies to fight neurological disorders and [cancer](#)."

Presenilin

Presenilin is comprised of a trefoil that includes amyloid precursor protein (APP), PSEN-1 and PSEN-2.

The following should be used for discussion with qualified computational biologists.

Cell Alignment: For Explanation and Discussion	
TNF-Alpha: TGF- Alpha (Calnexin) Density (CD-4)	
Calcium - threonine - magnesium (BRCA1)	p16
Calcium - serine - magnesium (BRCA2)	p18
Calcium - cysteine - magnesium (BRCA3)	p19
TNF-Beta: TGF-Beta (Calmodulin) Motility (CD-8)	
Calcium - phenylalanine - magnesium (HRas)	p21
Calcium - tyrosine - magnesium (KRas)	p27
Calcium - tryptophan - magnesium (NRas)	p57
TNF-Gamma: TGF-Gamma [VEGF] (Calcineurin) Modulatory Enzyme (CD-25)	
Iron - serine - Manganese	
Iron - cysteine - Manganese	
Iron - threonine - Manganese	

These are examples of the "enzymes" that have evolved with various designations; e.g. AKT, mTOR, PTEN, NF-kB, and MYC.

APP can be verified as being calcineurin.

PSEN1 can be verified as being calnexin.

PSEN2 can be verified as being calmodulin.