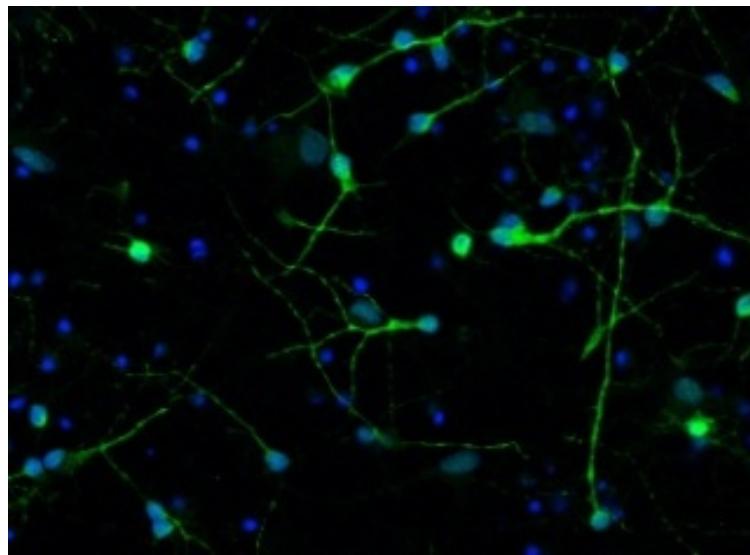


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Identified a Gene with a Key Role in Neuronal Survival



Researchers at the Institute of Neurosciences at Universitat Autònoma de Barcelona (INc-UAB) identified the fundamental role played by the Nurr1 gene in neuron survival associated with synaptic activity. The discovery, published in the *Journal of Biological Chemistry*, allows scientists to study a new target that could help to understand the relationship between alterations in neural connections, which are known to cause early cognitive deficit, and the neurodegeneration characteristic of Alzheimer's disease.

During the development of the brain, hundreds of thousands of neurons die if they do not establish the necessary connections - synapses - with their cell targets. The process of regulating neuron survival and death is fundamental in the organization of brain connections forming the adult brain.

The effect of synaptic activity on the survival of these neurons however is not limited to the developing brain; it is also fundamental in the adult brain. The loss of synaptic activity, which results into the characteristic cognitive impairment seen in neurodegenerative diseases such as Alzheimer's, precedes and contributes to the neuronal death observed in these pathologies. Despite the importance of this process, there is no exact knowledge of the molecular mechanisms implied in neuron survival generated by this activity.

In the study directed by José Rodríguez Álvarez, researcher of the UAB Institute of Neurosciences, scientists determined the relation of a gene and the neuron survival regulated by synaptic activity. Through a massive analysis of gene activity, researchers identified several dozens of genes whose functions are regulated by this activity. Of all the genes, the research demonstrates the key role played by the *Nurr1* gene in the survival of neurons. Among the discoveries made, researchers observed that when the activity of this gene is silenced, the neuron dies. The research concludes that this identification provides a better understanding into the relationship between early synaptic deficits and the posterior neurodegeneration observed in Alzheimer's disease.

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