

Epilepsy and brain pathology linked together by the protein ADK

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The brain of individuals who suffer from epilepsy is characterized by astrogliosis, a brain pathology evidenced by a complex series of changes in the morphology and function of brain cells known as astrocytes. Little is known about how astrogliosis relates to the dysfunction of brain cells known as neurons in individuals with epilepsy, but filling in the blanks in our knowledge could lead to new possibilities for therapeutic intervention. A study using mice by Detlev Boison and colleagues at Legacy Clinical Research, Portland, has now identified the protein ADK in astrocytes as a molecular link between astrogliosis and neuronal dysfunction in epilepsy.

The authors observed in a mouse model of epilepsy that ADK upregulation and spontaneous seizures occurred in the region of the brain affected by astrogliosis. In addition, overexpression of ADK in a specific region of the brain triggered seizures in the absence of astrogliosis. Conversely, mice engineered to express less ADK in specific regions of the brain were protected from chemical-induced epilepsy. Furthermore, as ADK-deficient ES cell-derived implants protected normal mice from chemical-induced astrogliosis, ADK upregulation, and seizures, it was suggested that ADK-based treatment strategies might provide a new approach for the treatment of individuals with epilepsy.

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