LGI1

The Leucine-rich glioma inactivated 1 (LGI1) protein is thought to be implicated in malignant progression of glioma tumors, and mutations in the encoding gene, LGI1, cause autosomal dominant lateral temporal epilepsy, a genetic focal epilepsy syndrome. The aim of a study was to investigate the possible involvement of LGI1 in high grade glioma-associated epilepsy by analyzing its expression in tumor specimens of patients with and without epilepsy and by searching for LGI1 autoantibodies in the sera these patients.

They examined tumor tissue samples from 24 patients with high-grade gliomas (12 with and 12 without epilepsy) by immunoblot and detected variable amounts of LGI1 in tumor tissues from 9/24 (37%) patients.

LGI1 was detected in 7/12 (58%) patients with epilepsy and in 2/12 (16%) patients without epilepsy (p = 0.0894; Fisher's exact test). Moreover, testing blood sera of five patients for antibodies against LGI1 revealed LGI1 autoantibodies in two patients, both suffering from epilepsy and expressing LGI1 in tumor tissue.

The findings suggest that there may be a preferential expression of LGI1 in high-grade glioma tumors of patients with epilepsy. They also unveil the presence of serum LGI1 autoantibodies in some patients with high-grade gliomas, where they might play an epileptogenic role. ¹⁾.

The protein NgR1 is encoded by RTN4R, a gene linked to schizophrenia.

Thomas et al, previously reported NgR1 as receptor for the epilepsy-linked protein LGI1. NgR1 regulates synapse number and synaptic plasticity, whereas LGI1 antagonizes NgR1 signaling and promotes synapse formation. Impairments in synapse formation are common in neurological disease and we hypothesized that an LGI1-NgR1 signaling pathway may contribute to the development of schizophrenia.

Variants in NgR1 and LGI1 may be associated with schizophrenia and variants in NgR1 found in schizophrenic patients have impaired LGI1-NgR1 signaling. Impaired LGI1-NgR1 signaling may contribute to disease progression ²⁾.

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