# GRIN2A related syndrome

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Language dysfunction is a common and serious comorbidity of epilepsy, especially in individuals with epilepsy aphasia spectrum syndromes. Childhood epilepsy with centrotemporal spikes is on the mild end of the spectrum, while epileptic encephalopathy with continuous spike-and-wave during sleep syndrome is on the severe end. Traditional antiseizure medicines and immunotherapy are currently used to treat severely affected patients, but the results are usually disappointing. The discovery that GRIN2A is the primary monogenic etiology of these diseases has opened the door to precision treatments. The GRIN2A gene encodes GluN2A protein, which constitutes a subunit of the NMDA receptor (NMDAR). The GRIN2A pathogenic variants cause gain or loss of function of NMDAR; the former can be treated with uncompetitive NMDAR antagonists, such as memantine, while the latter with NMDAR co-agonist serine. Hyper-precision therapies with various other effective agents are likely to be developed shortly to target the diverse functional effects of different variants. Precision treatments for GRIN2A-related disorders will benefit those who suffer from the condition and pave the way for new therapeutic approaches to a variety of other NMDAR-linked neurodegenerative and psychiatric diseases (schizophrenia, Parkinson's disease, Alzheimer's disease, and so on). Furthermore, more research into GRIN2A-related disorders will help us better understand the neuroinflammatory and neuroimmunological basis of epilepsy, as well as the pathological and physiological network activation mechanisms that cause sleep activation of central-temporal spikes and language impairment.