Epilepsy is a burdensome neurological disease which is difficult to treat and is a serious epidemiological problem. In Europe the number of patients affected by different forms of epilepsy is close to 6 million (every minute adds one new case). The death rate among epileptic patients is 2-3 times higher, the mean life expectancy 2-10 years lower and incidence of other diseases 4-times higher than in the whole population. About 30% of epilepsy cases are refractory to any kind of medication and more than half of the patients feel stigmatized (data from Baulac et al., Epilepsia, 56, 1687-1695, 2015). Temporal lobe epilepsy (TLE) is the most frequent form of epilepsy and its treatment appears relatively least effective. Recurrent epileptic seizures characterizing chronic TLE are preceded by durable (often lasting for years) symptom-free period which severely hampers identification of the causative factors and delineation of the initiation phase, understood as a set of primary epileptic seizures preceding the asymptomatic, latent period. Unavailability of prevention methods plus the absence of reliable biomarkers of the TLE onset substantiate the need to characterize the initiation phase of TLE in experimental settings. The aim of the proposed research is to get insight into one of the critical biochemical mechanisms determining the duration and severity of symptoms of the initial phase of TLE. More specifically, attempts will be made to unravel the mechanism responsible for maintenance of the elevated activity of excitatory neurotransmission beyond the period of stimulation by a primary trigger. The hypothesis to be tested is that maintenance of epileptic seizures is facilitated by accelerated renewal of glutamate, a neurotransmitter amino acid responsible for excitation. Studies will be conducted in an animal model of TLE reproducing the juvenile form of the disease. The incidence of TLE in children is very high, constituting one of the major challenges in pediatric neurology.