Poster Presentation

A Review of Animal Models of Absence Epilepsy

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Abstract

The most common type of childhood-onset epilepsy syndrome is childhood absence epilepsy (CAE) with well-defined electro clinical features but unknown pathological basis. The incidence of absence epilepsy is about 2 and 8 out of every 100 000 children up to the age of 16, and the prevalence is 2 and 10% of children with any form of epilepsy. Children with CAE suffer from high rate of pretreatment attention deficits that persist despite seizure freedom. Many researchers have still focused on the phenomenon of the absence seizure because of the unclear mechanisms involved in its pathophysiology. Although several models used for screening, quantification and evaluation of absence epilepsy but the key issue is reproducibility of the full clinical syndrome and pathogenesis as well as its different etiology. Considering that each substance has limited duration of action and specific time to observe the seizure. This review includes pharmacological animal models (Systemic Penicillin, Low-dose pentylentetrazole, tetrahydroisoxazolo pyridine and gamma-Hydroxy-butyrate, AY-9944 and methylazzoxymethanol acetate (MAM)-AY-9944 models and genetic animal models (tottering, lethargic, stargazer, mocha, slow-wave epilepsy and ducky mouse, and WAG/Rij, GEARS and Legacies rats). As regards, childhood absence epilepsy has variable genetic etiology; it seems that genetic animal models are more suitable than chemical models, as close correlation of EEG features and behaviors of genetic animal models to the human condition. Among genetic models in mousses and rats the GAERS and the WAG/Rij strains of Wistar, have asserted to be valid and predictive of human absence epilepsy. Most publications were designed based on the WAG/Rij rats. Altogether in both models the thalamocortical circuits obviously involved as the critical generator of absence seizures. Multidisciplinary studies of these two strains, lead to find wealth information about the role of the cortex and the thalamus, and other subcortical circuits.

Keywords: Absence epilepsy, Genetic models, Pharmacological models

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