## GENETIC FACTOR IN ETIOLOGY OF FEBRILE SEIZURES

Investigators from Istanbul, Turkey, studied R43Q mutations of the gamma-aminobutyric acid A receptor (GABRG2) gene, located on the long arm of chromosome 5, in 44 children with febrile seizure (FS) and 49 without. FSs were simple in 28 (63.6%) and complex in 16 (36.4%). Heterogeneous R43Q mutation of gamma-aminobutyric acid A receptor g2 subunit occurred significantly more often in the patient group (36%) than in the control group (2%); $p<0.001$. The homozygous mutation carrier status was not different in the 2 groups. Family history of febrile convulsion and epilepsy was significantly higher in the study group than in controls ($p<0.01$). (Hancili S, Onal ZE, Ata P, et al. The GABAa receptor g2 subunit (R43Q) mutation in febrile seizures. **Pediatr Neurol** 2014 Apr;50(4):353-6).

COMMENTARY. The febrile seizure trait is inherited as a polygenic or multifactorial model or an autosomal dominant pattern with reduced penetrance [1]. Mutations of several genes have been linked to febrile seizures, including voltage-gated sodium, calcium, and potassium, and ligand-gated ion channels, nicotinic cholinergic receptor and gamma-aminobutyric acid A (GABAa) receptor. R43Q mutation of the GABAa receptor g2-subunit is involved in the cause of absence epilepsy and febrile seizure [2]. Twin studies reveal distinct genetic factors for different FS subtypes and sub-syndromes, especially FS+ [3].

**References.**


1. Nakayama J. Brain Dev. 2009 May;31(5):359-65.
2. Wallace RH, et al. Nat Genet. 2001 May;28(1):49-52.
3. Eckhaus J, et al. Epilepsy Res. 2013 Jul;105(1-2):103-9.


## ENVIRONMENTAL FACTORS ASSOCIATED WITH FEBRILE SEIZURES

Investigators from Taichung, Taiwan, conducted a nationwide population-based retrospective study of the association between febrile seizure (FS) and allergic rhinitis. During an average 6.7 years follow-up of 1304 children with FSs, the incidence of allergic rhinitis in the FS group was higher, and after 11 years, the allergic rhinitis incidence was 4% higher than controls ($p<0.0001$). Risk of allergic rhinitis in the FS group is 1.21 times higher than in the control group, and the risk is even higher (18.9) in patients with more than 3 FS-related medical visits. Both disorders have similar cytokine profiles and viral infection association. (Lin W-Y, Muo C-H, Ku Y-C, Sung F-C, Kao C-H. Increased association between febrile convulsion and allergic rhinitis in children: a nationwide population-based retrospective cohort study. **Pediatr Neurol** 2014 Apr;50(4):329-33).

COMMENTARY. Fever and height of the body temperature as a measure of the FS threshold have an essential role in the mechanism of the FS. The cause of fever is almost always viral, most frequently HHV-6 in the United States and influenza in Japan. Some viruses have neurotropic properties, leading to the theory of a transient encephalitic