pISSN:2349-5707 Volume:13,Issue 3, March -2026 eISSN:2349-5715

ETIOPATHOGENESIS OF FEBRILE SEIZURES

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Abstract: Febrile seizures (FS) are the most common seizure disorder in young children. They typically happen between 6 months and 5 years of age when a child has a fever — but **no** brain infection, metabolic problem, or history of seizures without fever.

The causes are complex and involve several things working together: a child's genes, how their brain is still developing, and the body's natural inflammatory response to infection. Recent research has spotlighted one key player — a chemical called interleukin-1β (IL-1β) — that ramps up brain cell activity and makes seizures more likely. This article explains the main triggers and mechanisms in plain language.

Keywords: febril seizure, mechanism, brain, factor, sytocin, fever

Introduction Febrile seizures are the most frequent neurological issue seen in early childhood, affecting roughly 2–14% of kids worldwide. They occur during a feverish illness without any sign of infection inside the brain or sudden chemical imbalance in the body.

Although most children outgrow them and do very well afterward, doctors and researchers still study them closely. That's because febrile seizures can sometimes be linked to later epilepsy or other neurological concerns. At their core, these seizures happen when fever, inflammation, genetic factors, and the developing brain all interact in just the right (or wrong) way.

What Triggers Febrile Seizures?

1. Infections The usual spark is an infection that causes fever. Common culprits include viruses such as influenza, human herpesvirus-6 (HHV-6), adenovirus, and ordinary respiratory viruses. These germs kick off the immune system, which then releases inflammatory chemicals that can affect the brain and lower the seizure threshold.

2. Genetic Factors Family history matters a lot. If parents or siblings had febrile seizures, a child is more likely to have them too. Certain gene changes — especially ones affecting sodium channels in brain cells (like the SCN1A gene) — have been linked to febrile seizures and related conditions such as Genetic Epilepsy with Febrile Seizures Plus (GEFS+). These genetic differences can make the brain more excitable when fever hits.

3. Brain Development Young children's brains are still maturing. Their "brakes" (the inhibitory GABA system) aren't fully developed yet, so it's easier for over-excitation to tip into a seizure. This immaturity explains why febrile seizures almost never happen after age 5 or 6.

4. Other Contributing Factors Things like low iron levels or quick shifts in electrolytes can make a child more vulnerable. Interestingly, it's often the **rapid rise** in temperature — rather than how high the fever actually gets — that acts as the trigger.

How Do Febrile Seizures Actually Happen? (The Mechanisms)

pISSN:2349-5707 Volume:13,Issue 3, March -2026 eISSN:2349-5715

The Role of Fever and Inflammation When the body fights an infection, immune cells release pro-inflammatory cytokines — especially IL-1β, IL-6, and TNF-α. These chemicals can cross or open up the blood–brain barrier, letting inflammatory signals reach the brain.

IL-1β stands out as a major culprit. It directly increases the excitability of neurons, making it much easier for a seizure to start. Studies consistently show that higher IL-1β activity lowers the seizure threshold during fever.

The Fever Pathway Cytokines tell an enzyme called COX-2 to produce prostaglandin E2 (PGE2). PGE2 travels to the brain's thermostat (the hypothalamus) and raises body temperature. The resulting fever plus the inflammatory chemicals then further excite brain cells.

Disrupting the Brain's Chemical Balance Inflammation tilts the scales: it boosts excitatory signals (glutamate) while weakening inhibitory ones (GABA). This imbalance creates a perfect storm for hyperexcitable neurons and seizures.

Microglia Join the Party Microglial cells (the brain's immune guards) get activated by the inflammatory signals. They release even more cytokines, amplifying the inflammation inside the brain and making seizures more likely.

Genes + Inflammation = The Perfect Storm The latest understanding is that febrile seizures usually result from a **combination** of genetic vulnerability and a strong inflammatory response. Variations in the genes that control cytokines (IL-1β, IL-6, TNF-α) can make some children's inflammatory reaction more intense, dramatically raising their risk during a simple viral fever

Conclusion.

Febrile seizures are truly multifactorial — infections, genes, an immature brain, and the body's inflammatory response all play important roles. The spotlight today is on cytokine-driven neuroinflammation, particularly IL-1β, which appears to be the key that unlocks neuronal hyperexcitability

References:

1. Gaffarova Visola Furqatovna Evaluate the Neuropsychological, Clinical Neurological and Neurophysiological Characteristics of Febrile and Afebrile Seizures// American Journal of Science and Learning for Development Volume 2 | No 2 | February -2023. P187-192.
2.Кожинова А.В. Фармакотерапия больных, перенесших ишемический инсульт, в период реабилитации / А.В. Кожинова, О.С. Левин // Современная терапия в психиатрии и неврологии. — 2015. — 4-11. — с. 236-242.
3.Пирадов М.А. Инсульт. Пошаговая инструкция. – 1-е изд. / М.А. Пирадов, М.Ю. Максимова, М.М. Танашян — М.: ГЭОТАР-Медиа, 2019. — 272 с.

4.Габашвили, В.М. Эпилептические припадки при сосудистых заболеваниях головного мозга / В.М.

5.Zhao Y, Li X, Zhang K, Tong T, Cui R. The Progress of epilepsy after stroke. Curr Neuropharmacol. 2018;16(1):71–8.

6. Chung JM. Seizures in the acute stroke setting. Neurol Res. 2014;36(5):403–6.

pISSN:2349-5707 Volume:13,Issue 3, March -2026 eISSN:2349-5715

7. tokum JA, Gerzanich V, Simard JM. Molecular pathophysiology of cerebral edema. J Cereb Blood Flow Metab. 2016;36(3):513–38.

8. Denier C, Masnou P, Mapoure Y, Souillard-Scemama R, Guedj T, Theaudin M, et al. Watershed infarctions are more prone than other cortical infarcts to cause early-onset seizures. Arch Neurol. 2010;67(10):1219–23.