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Subclinical auditory dysfunction in a genetic rat model of childhood absence epilepsy

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ABSTRACT

OBJECTIVE: Childhood absence epilepsy (CAE) is defined by cognitive, behavioral, and neurophysiological abnormalities, but its impact on auditory function remains underexplored. Using Genetic Absence Epilepsy Rats from Strasbourg (GAERS), this study aimed to examine both peripheral and central auditory pathways function to better understand the auditory consequences of CAE.

METHODS: The experimental and control groups consisted of six-month-old male GAERS (n=6) and Wistar (n=6) rats. Auditory functions were assessed using distortion product otoacoustic emissions (DPOAE) and auditory brainstem responses (ABR). DPOAE amplitudes and ABR wave thresholds, latencies, and amplitudes were analyzed for group comparison.

RESULTS: DPOAE amplitudes were significantly reduced across all tested frequencies in epileptic rats compared to controls. In addition, ABR thresholds were slightly elevated compared to controls, but remained within clinically normal limits. Prolonged latencies were obtained in all waves except Wave I, indicating subcortical auditory pathway involvement in the epileptic group. A distinct ABR amplitude pattern emerged in epilepsy, characterized by increased Wave I–II amplitudes, a marked reduction at Wave III, and subsequent increases at Waves IV–V.

CONCLUSION: The results showed both peripheral and central auditory nervous system dysfunction in GAERS rats, providing translational insight into CAE. Subclinical abnormalities in auditory neuronal processing may contribute to speech-in-noise difficulties, language impairments, and learning problems in affected children. Early detection and follow-up are valuable for the clinical management of auditory involvement in pediatric epilepsy.

Keywords: Childhood absence epilepsy; hearing loss; otorhinolaryngology; pediatric epilepsy; subclinical dysfunction.

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Childhood absence epilepsy (CAE) is one of the most common pediatric epilepsy syndromes, seen primarily in school-aged children, more frequently in girls, and accounting for approximately 10–18% of epilepsy diagnoses in this age group [1–4]. Typical absence seizures in children are characterized by a sudden and brief loss of consciousness (absence) [5]. During the seizure, the child stares fixedly at a point, their facial expression becomes blank, and any ongoing activity is abruptly interrupted [6]. In most cases, automatisms such as lip-smacking or hand movements can be observed, along with blinking or mild facial/ocular twitching [2]. The seizure generally lasts 3–20 seconds and occurs several times throughout the day [2, 7]. Children rapidly return to normal afterward, although a brief period of confusion may occur.

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CAE has traditionally been considered a "benign" condition because it generally carries a good prognosis, responds well to antiepileptic medications [8, 9], and occurs in approximately half of patients within two years of onset or by early adolescence [2]. However, a comprehensive review has shown that children with absence epilepsy may exhibit comorbidities such as attention deficit hyperactivity disorder (ADHD), learning difficulties, impairments in cognitive and executive functioning, and problems with verbal skills and language acquisition ([10] for details). Due to these associated comorbidities, the notion that CAE is a benign disorder is now being reconsidered. Moreover, because absence seizures often occur without warning and can frequently go unnoticed, a detailed characterization of the condition is essential for clinical management as well as for the well-being of both the child and the family.

The Genetic Absence Epilepsy Rats from Strasbourg (GAERS), derived from a Wistar rat colony in Strasbourg, France, represent a well-established genetic model of absence epilepsy [11, 12]. These animals display the complete spectrum of electroencephalographic and behavioral features characteristic of the disorder [13]. Absence seizures are thought to result from abnormal electrical activity within thalamocortical circuits, which increases thalamic inhibition and disrupts consciousness [14, 15].

This study aimed to provide the first detailed characterization of the auditory profile of absence epilepsy in rats. Validating this approach in a preclinical model offers a valuable foundation and support for future clinical studies on absence epilepsy, particularly in identifying subclinical signs that might remain unnoticed.

Highlight key points

- Subclinical auditory dysfunction was objectively identified in a genetic rat model of childhood absence epilepsy, affecting auditory pathways.
- ABRs presented distinct patterns of prolonged wave latencies and increased amplitudes in epilepsy, although within the normal range for hearing.
- DPOAE and ABR reveal functional abnormalities that may underlie speech-in-noise difficulties and learning impairments in pediatric epilepsy.

MATERIALS AND METHODS

Experimental Animals

The experimental and control groups consisted of six-month-old male GAERS (n=6) and Wistar (n=6) rats. All animals were housed in standard cages in a temperature-controlled room with 45–65% relative humidity and maintained under a 12-hour light/dark cycle. Rats had ad libitum access to standard chow and water. All animal procedures were approved by the Marmara University Institutional Animal Care and Use Committee (8.2025mar6) and conducted in accordance with the Guide for the Care and Use of Laboratory Animals to minimize animal suffering and reduce the number of animals used.

Auditory Brainstem Response (ABR) Protocol

Auditory Brainstem Response (ABR) test was conducted under anesthesia, which was induced via intraperitoneal injection of ketamine (100 mg/kg) and xylazine (10 mg/kg). Stimuli were presented at 2, 4, and 8 kHz test frequencies. Stainless steel subcutaneous needle electrodes were positioned at the vertex (active), behind the stimulated ear (reference), and non-stimulated ear (ground). Tonal stimuli with a rise/fall time of 1,000 ms and alternating polarity were delivered via ER3A insert earphones at a rate of 25.1 tone-pips per second.

The electroencephalography (EEG) signals were amplified (×100,000), band-pass filtered between 100 Hz and 3,000 Hz, and averaged over 1024 sweeps. ABR thresholds were determined by identifying the lowest stimulus intensity that produced a replicable waveform, initially using 10-dB steps and then refined with 5-dB steps near the threshold, particularly following peak II (16). All recordings were performed using the SmartEP data acquisition system (Intelligent Hearing Systems, Miami, FL, USA).

Distortion Product Otoacoustic Emission (DPOAE) Protocol

Distortion Product Otoacoustic Emissions (DPOAE) testing was performed on rats under anesthesia to assess auditory function and hearing loss at the level of outer hair cells (OHC). During the DPOAE test, two pure-tone stimuli were presented with a frequency ratio of f2/f1=1.22, and the 2f1–f2 frequency combination was used for emission analysis. The intensity levels of the primary tones were set at L1=65 dB SPL for f1 and L2=55 dB SPL for f2. The test frequencies included 2, 2.8, and 4 kHz. The signal-to-noise ratio (SNR) at each frequency was analyzed in dB SPL. Recordings were conducted using the Otodynamics ILO-V6 system (Otodynamics, London).

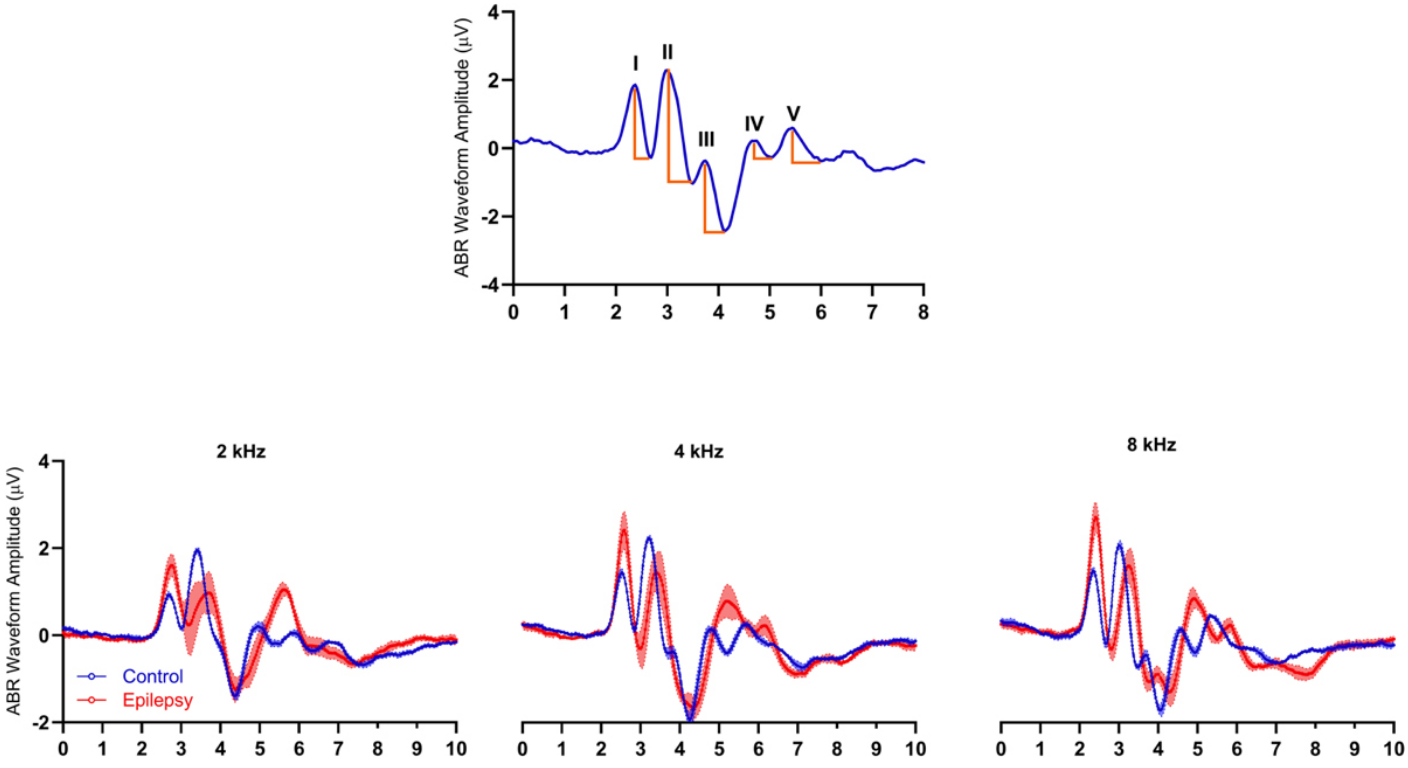


FIGURE 1. Auditory brainstem responses reveal altered subclinical auditory dysfunction in epileptic rats. Representative ABR waveform is shown with characteristic peaks (I–V), and amplitudes are measured from the top to the bottom right of each peak. Comparison of ABR waveforms at 2, 4, and 8 kHz revealed altered response morphology and increased amplitudes in the epileptic group (red) relative to controls (blue), consistent with frequency-dependent auditory deficits. The shaded red and blue areas represent the standard error.

Statistical Analysis

Statistical analysis and graph preparation were performed with GraphPad Prism (version 10.2.2; GraphPad Software). Data distribution was first examined to decide whether parametric or nonparametric methods were appropriate. Group comparisons were carried out using two-way ANOVA followed by Tukey's post hoc multiple comparisons test for each parameter. Statistical significance was defined as $p<0.05$.

RESULTS

Auditory function was evaluated in GAERS rats and age-matched Wistar controls by recording DPOAEs and ABRs. ABR waveforms (Fig. 1) reflect the neural activity evoked by acoustic stimulation along the auditory pathway, beginning with inner hair cells and spiral ganglion neuron responses in the cochlea and extending through the brainstem to the inferior colliculus. Representative ABR waveforms at 2, 4, and 8 kHz demonstrate larger amplitudes and delayed peak latencies in the epileptic group compared to controls, visually supporting the quantified latency and amplitude findings.

ABR thresholds were significantly elevated in GAERS across all tested frequencies compared to controls ($F(1, 10)=49.91$, $p<0.0001$), although they remained within clinically normal limits (below 25 dB SPL) (Fig. 2). These results suggest a relative decrease in auditory sensitivity and a potential vulnerability to subclinical hearing impairment in the epileptic group.

DPOAE amplitudes were significantly reduced in the epileptic group at all tested frequencies ($F(1, 10)=10.70$; $p<0.01$), suggesting impaired OHC function compared to controls (Fig. 2). However, all SNRs tested were above 6 dB in both groups. These results may reflect a subclinical peripheral deficit likely contributing to the slightly elevated ABR thresholds observed.

The ABR amplitudes of Peaks I, II, IV, and V were significantly higher in the epileptic group as shown in the Figure 3 (Peak I, $F(1, 10)=32.04$, $p<0.001$; Peak II, $F(1, 10)=8.892$, $p<0.05$; Peak IV, $F(1, 10)=17.21$,

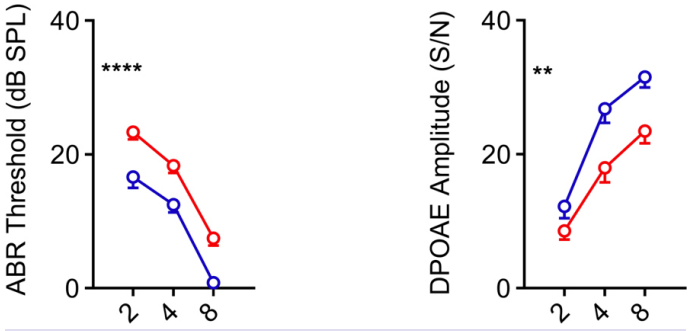


FIGURE 2. Elevated ABR thresholds and reduced DPOAE amplitudes indicate impaired auditory function in epileptic rats. ABR thresholds were significantly elevated in the epileptic group (red) compared with controls (blue) across all tested frequencies, indicating impaired auditory sensitivity. DPOAE amplitudes were markedly reduced in the epileptic group, compared with controls. Data are shown as mean±SEM. Statistical significance is indicated as follows: **p<0.01, ****p<0.0001.

p<0.01; Peak V, F (1, 10)=22.83, p<0.001), while Peak III showed no significant difference (F (1, 10)=2.825 p>0.05) compared to controls. The I/V amplitude ratio was also significantly larger in the epileptic group (F (1, 10)=5.786, p<0.05), suggesting abnormal central neural synchrony or altered auditory pathway gain.

Significantly prolonged latencies were observed in GAERS at Peaks II to V (sequentially; F (1, 10)=9.544; F (1, 10)=12.65; F (1, 10)=14.13; F (1, 10)=20.73), while Peak I latency remained unchanged (F (1, 10)=0.3276, p>0.05) (Fig. 4). This indicates a disruption in neural conduction time in the retro-cochlear auditory pathways.

DISCUSSION

This study provides detailed evidence of subclinical auditory dysfunction in GAERS rats, a genetic model of absence epilepsy. These results offer new insights into auditory alterations associated with CAE. While previous research has primarily focused on the cognitive, behavioral, and neurophysiological aspects of CAE [10, 16–18], its influence on the auditory system remains comparatively unknown. In the following sections, potential mechanisms that may underlie auditory dysfunction are discussed in detail.

Peripheral Auditory Findings and Outer Hair Cell Dysfunction

DPOAE amplitudes results revealed significant reductions across all tested frequencies, suggesting impaired OHC function in epileptic rats compared to controls. Notably, all epileptic rats passed the DPOAE tests according to SNRs at all tested frequencies. Given that OHCs are essential for cochlear amplification and fine-tuned frequency selectivity [19], the impairment could result in elevated auditory thresholds and diminished frequency resolution [20, 21]. This mechanism may

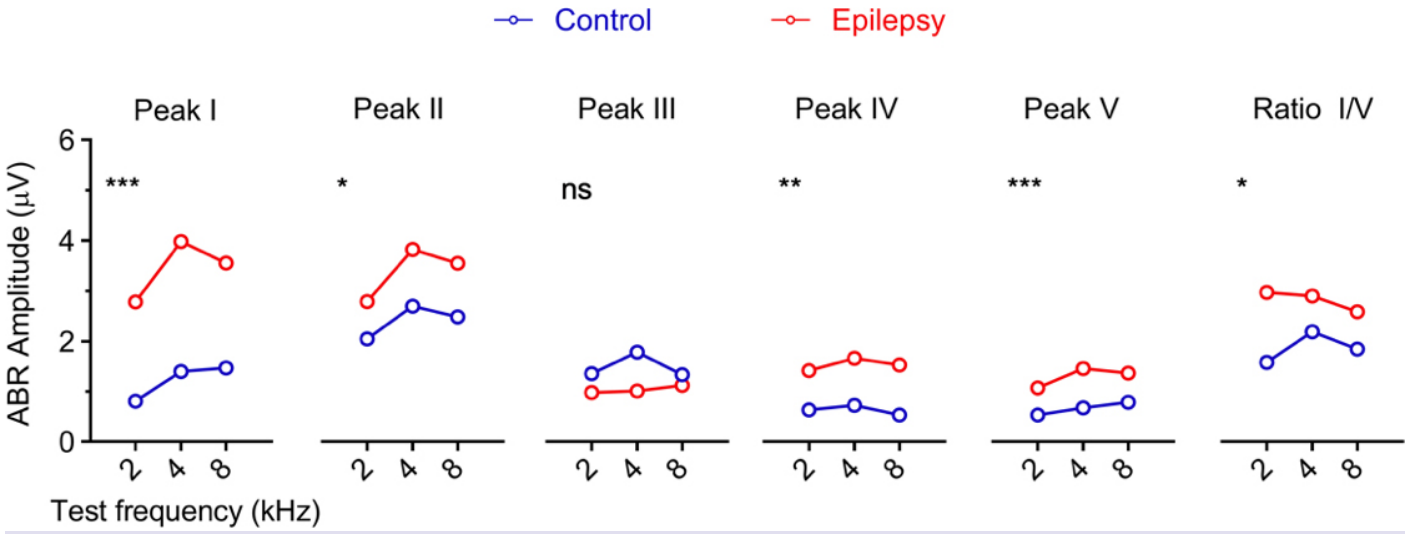


FIGURE 3. ABR amplitudes revealed hyperexcitability and abnormal gain control in the epileptic rats. Analysis of wave amplitudes showed increased responses across all peaks except Wave III, indicating hyperexcitability in the auditory neuronal responses. Increased I/V amplitudes may reflect that epilepsy predominantly affects retro-cochlear auditory pathways rather than the auditory nerve. Statistical significance is indicated as follows: *p<0.05, **p<0.01, ***p<0.001, ns: not significant.

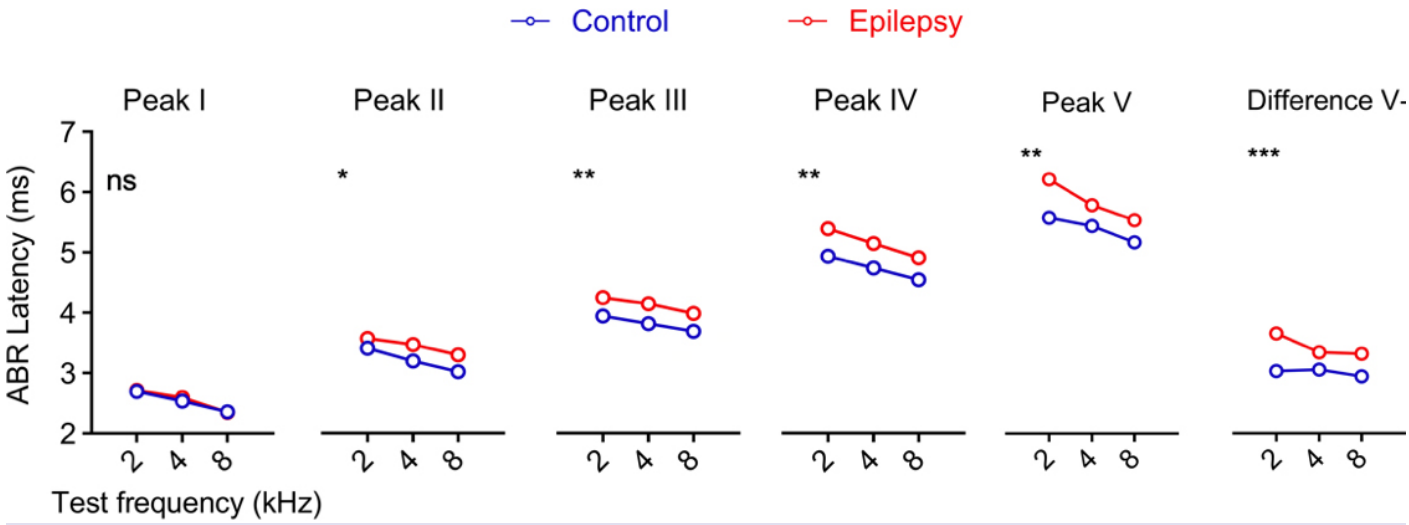


FIGURE 4. ABR latencies prolonged from Wave II onward. The preserved Wave I latency suggests that epilepsy primarily reflects disruptions in retro-cochlear neural circuits rather than effects on the auditory nerve. Additionally, the increased V-I interpeak latency compared to controls supports this inference. Statistical significance is indicated as follows: *p<0.05, **p<0.01, ***p<0.001, ns: not significant.

explain the slightly elevated ABR thresholds observed in this study. The presence of auditory deficits in other epilepsy models [22] and in human epilepsy populations [23–26] further supports to the findings with changing degrees of hearing loss.

Central Auditory Findings: Latency Prolongation and Amplitude Patterns

ABR analysis revealed prolonged latencies beginning at Wave II, despite preserved Wave I latency, indicating that auditory dysfunction in epilepsy predominantly involves retro-cochlear and subcortical neurotransmission circuits rather than the auditory nerve. Moreover, an increased Wave I/V amplitude ratio suggests the dysfunction within central auditory pathways. The increased prolonged Wave V–I interpeak latency further indicates impaired neurotransmission at the brainstem and higher level of auditory stations.

Analysis of wave amplitudes showed increased responses across all peaks except Wave III. The characteristic pattern (amplitude increases in Waves I–II, a marked reduction at Wave III, followed by recovery in Waves IV–V) was observed in the epileptic rats. These patterns may reflect the interplay of multiple mechanisms, including structural alterations within auditory circuits, neurotransmitter imbalances, ion channel dysregulation, and superior olivary complex (SOC) mediated inhibitory processes.

First, dendritic remodeling in GAERS rats, including increased branching, length, and spine density [27], may enhance postsynaptic receptor availability and glutamatergic transmission, contributing to hyperexcitability [28]. This mechanism is consistent with the increased ABR amplitudes.

Second, the neurotransmitters like gamma-aminobutyric acid (GABA) and acetylcholine are essential in auditory inhibition and modulation [29, 30]. Disruption of these neurotransmitter systems has been implicated in generating epileptic discharges [31, 32] and asynchronous auditory brainstem responses [23]. Such an imbalance may contribute to the increased amplitude by weakening inhibitory processes within the auditory pathway.

Third, increased expression of T-type calcium channels has been reported in GAERS rats [33, 34]. These channels critically regulate neuronal excitability within the auditory system, and knockout models demonstrate ABR amplitude reductions and elevated thresholds when disrupted [35]. Accordingly, T-type channel dysregulation could contribute to both latency prolongation and amplitude alterations observed in the present study.

Finally, the abrupt amplitude reduction at Wave III may reflect SOC-mediated inhibitory influences. In the absence epilepsy patients, preictal oscillations arising at the level of Wave III have been documented as potential "seizure precursors" [36]. These findings position ventral brainstem structures as a key inhibitory station within

epileptic networks. The subsequent increase in Wave IV–V amplitudes may, in turn, represent compensatory hyperexcitability.

Neural Network Perspective: Cortex–Thalamus–Brainstem Interactions

Functional Magnetic Resonance Imaging (fMRI) studies in CAE patients demonstrate that the brainstem reticular formation operates in concert with the thalamus and cortex as part of the "core network" sustaining absence seizures [37, 38]. According to the corticoreticular theory, absence seizures emerge from reciprocal interactions among the cortex, thalamus, and brainstem [39]. Findings of this study suggest that brainstem auditory pathways may also participate in this network, broadening the framework of seizure-related circuitry.

Clinical Implications and Subclinical Dysfunction

There are relatively few reports investigating the relationship between epilepsy and hearing impairment. Previous studies has reported that children with epilepsy present with normal ABR thresholds in response to click stimuli, while showing delayed responses to speech stimuli [40]. In another study, individuals with temporal lobe epilepsy demonstrated reduced performance in auditory temporal ordering tasks, including sequential and tonal pattern discrimination, as well as in dichotic listening, despite having normal pure-tone audiometric thresholds [41]. These studies suggest that central auditory processing deficits may be leading to clinically relevant "hearing difficulties" that are not detectable through standard audiometry, especially affecting the temporal lobe of the brain.

In addition, long-term use of certain antiepileptic drugs has been associated with auditory side effects such as tinnitus, sensorineural hearing loss, hyperacusis, and delayed auditory brainstem conduction, indicating potential cochlear and/or retrocochlear toxicity [42]. These effects highlight the need to consider medication-related auditory dysfunction when interpreting hearing outcomes in individuals with epilepsy.

Furthermore, mutations in ion channel and transporter genes may give rise to syndromic conditions that combine epilepsy with sensorineural hearing loss [43, 44]. For example, variants in CACNA1H, which encodes a T-type calcium channel, and KCNJ10, implicated in EAST (Epilepsy, ataxia, sensorineural hearing loss, and renal tubulopathy) syndrome, have been reported in patients presenting with both epileptic seizures and early-onset sensorineural hearing loss. Together, these genetic findings support the notion of shared pathophysiological mechanisms underlying auditory and epileptic phenotypes.

In the present study, although thresholds were elevated compared with controls, they remained within the clinically normal range. In contrast, prolonged latencies and altered amplitude patterns point toward subclinical auditory dysfunction. These findings may help explain common clinical complaints in children with absence epilepsy, such as speech-in-noise difficulties, language processing deficits, and learning problems, providing a neurophysiological basis for these challenges.

Conclusion

The present findings reveal crucial translational implications for CAE. Subclinical auditory dysfunction may contribute to speech-in-noise perception difficulties, delayed language development, and learning impairments, which are frequently reported in affected children. Early identification of such deficits could inform patient management and guide the development of targeted rehabilitation strategies. Objective measures of auditory function may provide valuable insight into screening for auditory involvement in pediatric epilepsy.

Ethics Committee Approval: The Marmara University Local Ethics Committee for Animal Experiments approved for this study (date: 20.08.2025, number: 8.2025mar6).

Informed Consent: This study does not involve any human subjects and therefore does not require informed consent.

Conflict of Interest: The authors declare that there is no conflict of interest.

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Use of AI for Writing Assistance: The authors declared that artificial intelligence-supported technologies were not used in the production of the study.

Authorship Contributions: Concept – FNK; Design –FNK, YG; Supervision – AC, YG; Fundings – FNK; Materials – MFB; Data collection and/or processing – MFB, FNK; Analysis and/or interpretation – FNK, AC; Literature search – FNK, MFB; Writing – FNK, YG, MFB, AC; Critical review – YG, AC.

Data Availability: The authors declared that all datasets generated for this study are included in the manuscript. Additional data supporting this study's findings are available from the corresponding author upon reasonable request.

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