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Neuroanatomical Determinants of Personality Alteration in Epilepsy

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AUTHOR BIOGRAPHY

Sophia Zhang is a junior at Tenafly High School (NJ) and a dedicated neuroscientific researcher with experience at premier institutions, including NYU Langone and Columbia University Irving Medical Center. Her work explores the intersection of molecular biology, neuroscience, and psychology, with the ultimate goal of pursuing a career in neurosurgery. An accomplished violinist, Sophia is also a student at the Juilliard Pre-College Division and a member of the Grammy-winning New York Youth Symphony.

ABSTRACT

Epilepsy, a chronic neurological disorder characterized by recurrent unprovoked seizures, is increasingly recognized as a multifaceted condition with profound implications for personality and behavioral stability. This is particularly evident when seizure activity originates in brain regions critical for emotional regulation, social cognition, and executive function. This paper investigates the complex mechanisms underlying personality changes in epilepsy patients, focusing on the interplay between neuroanatomy, chronic seizure activity, and various treatment interventions.

By synthesizing neurobiological research, I analyze the bidirectional relationship between seizure activity and personality structure, with a specific emphasis on the distinct neuroanatomical traits of temporal lobe epilepsy (TLE) and frontal lobe epilepsy (FLE). The study also explores how chronic dysregulation in these regions can lead to behavioral syndromes such as heightened emotionality, impulsivity, or social withdrawal. Furthermore, the analysis evaluates the effects of common antiepileptic medications and surgical outcomes on a patient's overall temperament. Ultimately, this paper provides a comprehensive framework for understanding epilepsy, arguing that personality changes are the result of cumulative impacts on both neurological and psychological aspects of the brain.

Keywords: *Temporal Lobe Epilepsy, Frontal Lobe Epilepsy, Neuropsychiatry, Personality Change, Behavioral Phenotypes, Limbic System, Neuroinflammation, Antiepileptic Drugs, Surgical Intervention*

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INTRODUCTION

Epilepsy is a neurological condition characterized by recurrent, unprovoked seizures that result from abnormal electrical activity in the brain (Karoly et al., 2021). Affecting approximately 50 million people, it is one of the most prevalent neurological disorders worldwide (World Health Organization, 2024). Common symptoms range from convulsions to altered consciousness, and potential causes include genetic predispositions, structural brain abnormalities, and trauma (Mayo Clinic, 2021). Seizures often disrupt neural circuits critical for cognition, emotion, and behavior, leading to intense psychological changes and social challenges. One of the most significant disruptions for patients is personality change, which appears to be associated with neuroanatomical damage, chronic disease management, and societal stigma (Li et al., 2024).

Personality comprises stable patterns of thoughts, emotions, and behaviors that develop through genetic and environmental influences (Mammadov, 2022). While healthy personality traits allow individuals to adapt to life circumstances, maladaptive traits can hinder social functioning and coping mechanisms (Tijdink et al., 2016). These changes are evident in patients with temporal lobe epilepsy (TLE) and frontal lobe epilepsy (FLE), where key areas involved in emotional regulation and executive function are affected (Iurina et al., 2021).

The intersection of epilepsy and behavioral change is a critical point to highlight analyzing the condition comprehensively. Evidence suggests that patients with epilepsy face an increased risk for formal personality disorders, including those categorized under DSM-5 Clusters B (dramatic/emotional) and C (anxious/fearful) (Viola et al., 2024). Moreover, treatment approaches, including antiepileptic drugs (AEDs) and surgical interventions, can further influence trajectories (Iurina et al., 2021). Understanding these dynamics is essential for optimizing strategies for enhancing patient quality of life.

The objective of this paper is to investigate the mechanisms underlying personality changes in epilepsy, with a particular focus on the roles of neuroanatomy, seizure activity, and treatment interventions. By exploring the multifaceted nature of these shifts, this research highlights the need for tailored therapeutic approaches that address both the neurological and social consequences of the disorder.

METHODOLOGY

This paper presents a synthesis of findings from the Science Direct database using a systematically-devised search strategy by the author. Initial search terms were set as [personality change and epilepsy], and filters were applied for open-access research articles published between 2002 and 2025 within the neuroscience domain. From an initial pool of 537 papers, a final cohort of 22 empirical studies were selected based on specific inclusion criteria through a multi-stage screening process. First, a primary screening of titles

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and abstracts was performed to remove duplicates and irrelevant literature. Second, the full text of the remaining papers was evaluated against strict eligibility criteria.

Studies needed to demonstrate a clear clinical correlation by explicitly investigating the link between neuroanatomical seizures—particularly in the temporal and frontal lobes—and long-term behavioral or personality alterations. This initial analysis was first done by a brief overview of the abstract, introduction, and method section of each paper by the author.

Furthermore, papers had to use previously validated metrics in methods, such as MRI volumetric analysis of Gray Matter Volume (GMV) or recognized personality categorizations, such as the Big Five or DSM-5 criteria, to ensure data reliability. In addition to these structural requirements, preference was given to papers that provided interventional insight through longitudinal or comparative studies. Specifically, ones about treatment modalities, including antiepileptic drugs or surgical resections, and how they impacted personality trajectories over time, were highly preferred.

Conversely, papers were excluded if they focused solely on generalized psychiatric changes, such as isolated depression, without addressing the broader personality profile or neurological mechanisms that led to such cases. This multi-layered selection process ensured that the data directly addressed the goals of the paper, meaning mechanisms behind personality alteration rather than general psychological distress, which may happen in any neurological disorder, unspecific to epilepsy.

After analyzing and extracting relevant information from the 22 included research articles, descriptive categories for epilepsy types, affected cerebral lobes, personality profiles, and treatment methods were constructed.

LIMBIC HYPERCONNECTIVITY AND THE "HYPER-EMOTIONAL" PROFILE

Patients with epilepsy (PWE) exhibit a broad spectrum of personality changes influenced by seizure type, frequency, and treatment interventions. Temporal Lobe Epilepsy (TLE) emerges as a focal point in personality studies such as an Arabian investigative study in which patients with epilepsy were observed in accordance with psychological disorders (Khachan et al., 2024). TLE is especially prevalent due to its connection with the limbic system, a brain region integral to emotion and behavior regulation. TLE patients consistently display heightened neuroticism, anxiety, and social withdrawal, often coupled with psychiatric comorbidities such as mood and anxiety disorders (Khachan et al., 2024).

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Beyond these standard psychological markers, the Sensory-Limbic Hyperconnection Theory also offers an explanation for the profound shifts in personality often seen in TLE. According to this theory, chronic seizure activity in the temporal lobe "strengthens" or creates abnormal functional pathways between sensory processing areas and the limbic system, specifically the amygdala and hippocampus (Bear, 1979). This neurological "kindling" can lead to a state of hyper-emotionality, where patients ascribe deep, often spiritual or philosophical significance to otherwise mundane events. This phenomenon is hence frequently linked to the Gastaut-Geschwind syndrome, characterized by traits such as hypergraphia (an intensive drive to write), hyper-religiosity, and intensified emotional responses. These cases further illustrate how chronic electrical disruption in neurological wiring can fundamentally shape an individual's personality (Colak et al., 2021).

GREY MATTER VOLUME

Recent structural imaging data, with insights from the global Epilepsy Connectome Project, support these observations by linking personality traits to the physical morphology of the brain. Research indicates that increased neuroticism in patients with epilepsy is directly correlated with reduced Gray Matter Volume (GMV) in the prefrontal cortex and specific subcortical regions like the amygdala (Bonet et al., 2019). Structural deficits in areas responsible for top-down emotional regulation suggest that personality changes are not merely a psychological reaction to the diagnosis. Instead, they may reflect underlying structural neuro-pathology of the disorder. The reduction in GMV within the prefrontal-limbic network likely impairs the brain's ability to inhibit emotional distress, potentially stabilizing "high-neuroticism" traits as a chronic feature of the patient's persona profile.

NEURO-INFLAMMATORY PRIMING

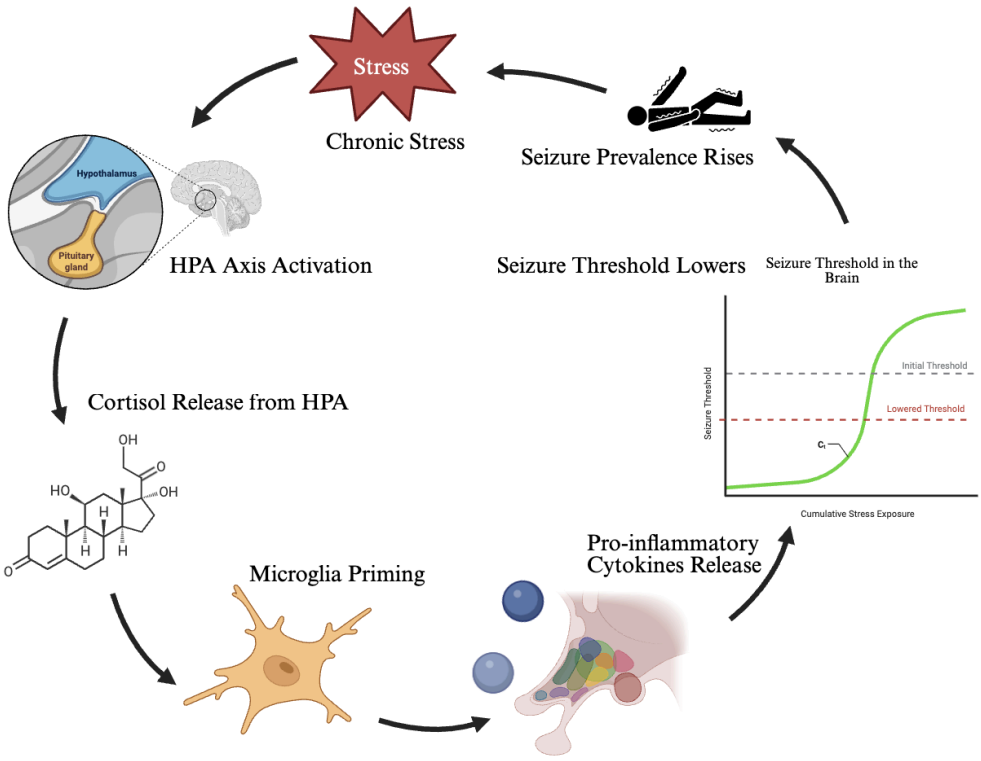
In addition, the relationship between personality and epilepsy is deeply influenced by a bidirectional feedback loop involving stress and neuroinflammation. Chronic stress acts as a potent seizure trigger by activating the hypothalamic-pituitary-adrenal (HPA) axis and releasing cortisol. This hormonal surge does more than just increase neuronal excitability; it also effectively prepares the brain's resident immune cells, known as microglia. Under chronic stress, these primed microglia shift into a pro-inflammatory state, releasing cytokines (such as IL-1β) that further lower the seizure threshold. This creates a devastating cycle: the anxious or neurotic personality traits associated with epilepsy lead to a state of chronic stress, which then fuels neuroinflammation, subsequently increasing seizure frequency and further reinforcing maladaptive behavioral traits (Espinosa-García et al., 2021).

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Figure 1



Note. This diagram illustrates the bidirectional feedback loop between psychological, external stress and neurobiological seizure triggers. The cycle starts with chronic stress, leading to HPA axis activation and the systemic release of cortisol. These hormones induce microglial priming, with the subsequent release of pro-inflammatory cytokines (e.g., IL-1β) that reduce the seizure threshold and facilitate seizure activity. The resulting neurological trauma and burden only reinforce chronic stress, reinitiating the cycle. Produced by author (2026) on BioRender based on data from (Espinosa-García et al., 2021).

PREFRONTAL DISRUPTION AND SOCIAL COGNITION DEFICITS

In contrast, Frontal Lobe Epilepsy (FLE) is more closely associated with impulsivity, executive dysfunction, and disinhibited behavior, reflecting the disruption of the frontal neural circuits responsible for

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decision-making and emotional control (Helmstaedter, 2001). Specific cases of FLE reveal significant challenges, such as addictive behaviors and moral reasoning deficits, particularly when the prefrontal cortex was affected.

While TLE is defined by emotional "over-signification," personality changes in FLE are often characterized by a breakdown of executive control and social function. Empirical psychometric profiling has revealed that localized network paths result in entirely divergent clinical pathologies: while temporal lobe epilepsy exhibits distinct correlation with obsessive-compulsive personality profiles, frontal lobe epilepsy is uniquely characterized by prevalences in dramatic, histrionic personality traits (Parente & Giovagnoli, 2022). Recent research utilizing advanced neuroimaging suggests that FLE disrupts the ventromedial prefrontal cortex (vmPFC) and the orbitofrontal cortex (OFC), regions essential for reward processing and social decision-making (Caciagli et al., 2022). Chronic seizure activity in these circuits often results in a deficit in Theory of Mind (ToM)—the ability to understand the mental states, intentions, and emotions of others. This cognitive blindness leads to personality shifts marked by apparent apathy, reduced empathy, and social inappropriateness (Giovagnoli, 2014), which can be frequently misdiagnosed as primary personality disorders, such as pseudo-psychopathy. Furthermore, the connectivity gap caused by frontal seizures disrupts the inhibitory pathways that normally suppress impulsive urges, which is a potential explanation for the high rates of aggression and behavioral disinhibition observed in this specific population.

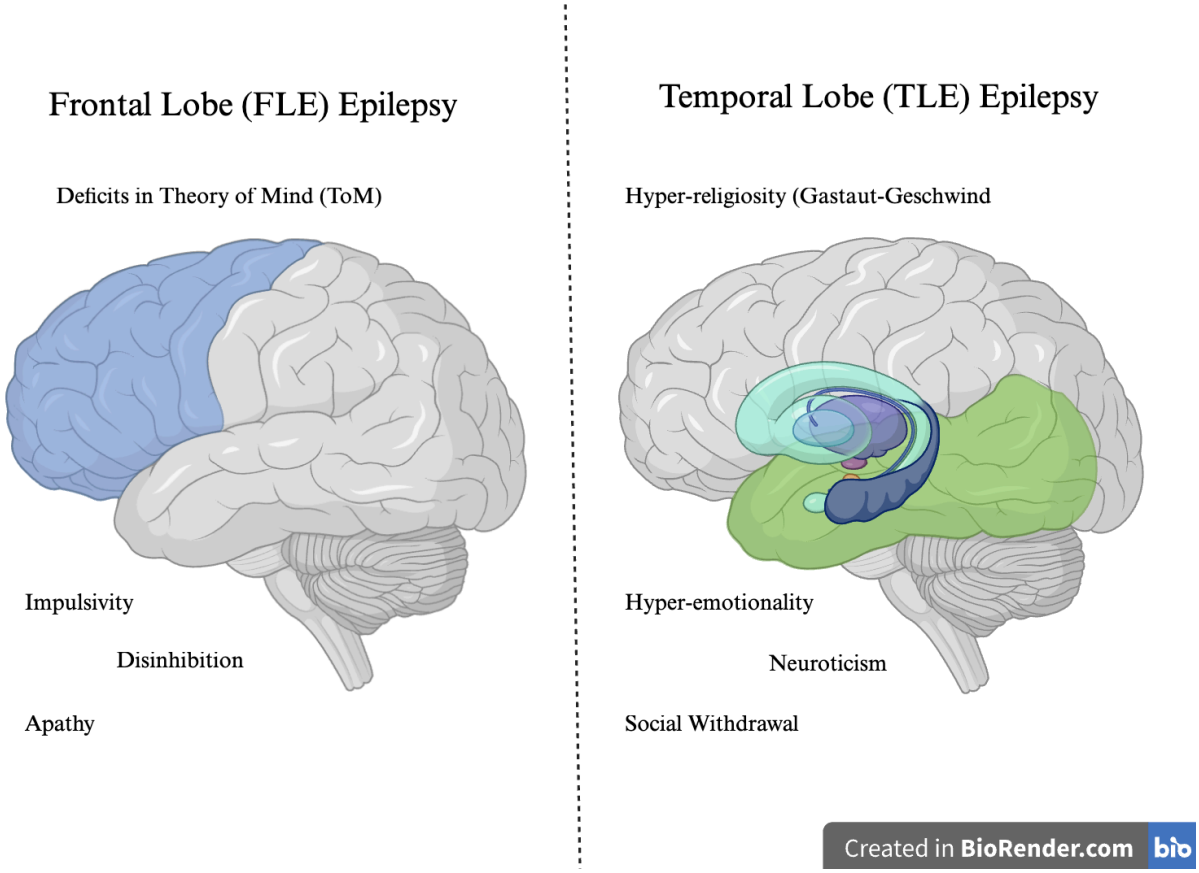
To clinically distinguish these complex frontal-subcortical circuit disruptions from standard psychiatric conditions, researchers have utilized specialized neuro-behavioral inventories. Specifically, the Orbitofrontal Behavioral Rating Scale (OBRS) has proven to be highly sensitive in quantifying precise progressions of disinhibition, executive apathy, and emotional shifts unique to orbitofrontal circuit damage (Chow, 2000). Ultimately, capturing these localized frontal deficits shows how structural network failures govern the distinct behavioral phenotypes observed in FLE.

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Figure 2



Note. Divergent behavior phenotypes associated with Temporal Lobe Epilepsy (TLE) and Frontal Lobe Epilepsy (FLE). Panel 1 (TLE) highlights the sensory-limbic hyperconnection that involves the amygdala and hippocampus, correlated with symptoms such as hyper-emotionality and Gastaut-Geschwind syndrome. Panel 2 (FLE) shows disruptions in the vmPFC and OFC, which correlates with executive dysfunction, social problems, and ToM deficits. Produced by the author (2026) with BioRender based on synthesized data from (Khachan et al., 2024) and (Caciagli et al., 2022).

SURGICAL OUTCOMES REGARDING LATERALIZATION AND SEIZURE FREEDOM

Surgical treatment for refractory epilepsy provided both opportunities and challenges for personality outcomes. A study by (Iurina et al., 2021) revealed that patients achieving seizure freedom (SF) often experienced significant reductions in neuroticism, impulsivity, and paranoia, along with improved emotional

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regulation and social functioning. Notably, patients undergoing temporal lobe resection exhibited varying outcomes depending on the lateralization of the epileptic focus (Haeusermann et al., 2024) . Left-sided resections were associated with enhanced emotional stability, whereas right-sided resections showed pronounced reductions in impulsivity and anxiety (Iurina et al., 2021). However, not all interventions yielded favorable results. Insula resections, for instance, were linked to heightened irritability and emotional instability in some patients (Bailles et al., 2021). Furthermore, non-seizure-free (NSF) patients reported more transient personality improvements, often reverting to pre-surgical baselines within a year (Steinbrenner et al., 2022). Longitudinal studies confirmed that while SF patients sustained their gains in emotional and social domains, NSF patients demonstrated limited or unstable changes, underscoring the importance of seizure control in long-term personality management.

SOCIODEMOGRAPHIC MEDIATORS OF BEHAVIORAL ADAPTATION

In more complex scenarios, personality outcomes varied by age and comorbidities. For example, childhood-onset epilepsy often interfered with the development of social and moral reasoning, leading to long-lasting personality traits characterized by dependency and rigidity (Berg et al., 2012). Meanwhile, elderly patients with epilepsy, frequently burdened by cognitive decline, exhibited amplified personality changes, particularly apathy and emotional detachment. Women with epilepsy (WWE) presented unique challenges, often reporting heightened vulnerability, insecurity, and feelings of social isolation (Eden et al., 2024). These emotional burdens were compounded by societal stigma, difficulties in self-confidence, and the unpredictability of seizures, emphasizing the need for gender-specific psychological interventions (Mangaard et al., 2021).

Furthermore, personality traits like neuroticism did not uniformly change across all studies. Some seizure-free patients exhibited reductions in anxiety and impulsivity subscales, while others showed no significant post-treatment differences, suggesting underlying genetic or neurodevelopmental factors in personality expression (Iurina et al., 2021).

PHARMACO-PSYCHOLOGY AND THE DRUG FINGERPRINT

The role of antiepileptic drugs (AEDs) further complicated the picture. While effective in managing seizures, their unique mechanisms of action can induce behavioral alterations that are frequently misidentified as permanent personality shifts (Caplan, 2012). For instance, levetiracetam is often associated with "Keppra-rage," a profile of increased irritability and hostility thought to result from the drug's binding to the synaptic vesicle protein SV2A, which can disinhibit aggressive impulses within the limbic system (Halma et al., 2014). Conversely, topiramate is notorious for cognitive dulling, where multi-channel inhibition can disrupt the high-frequency neuronal firing required for complex language processing. This in turn manifests as anomia

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(word-finding difficulty) and a flattened affect that can be mistaken for an apathetic or passive personality (Mula et al., 2003). On the other hand, some AEDs exert a restorative effect, such as lamotrigine. By inhibiting the release of the excitatory neurotransmitter glutamate, lamotrigine acts as a potent mood stabilizer, effectively reducing the emotional spikes and neuroticism common in TLE and assisting the patient in returning to a stable, pre-morbid state (Ade et al., 2024). The dual-edged nature of AEDs highlights the delicate balance clinicians must navigate in optimizing both seizure control and quality of life for both patients and families, ensuring that the treatment itself does not become a primary driver of maladaptive personality change.

Taken together, these findings illuminate the intricate interplay of neurological, psychological, and social factors in shaping personality changes in epilepsy patients. While advancements in surgical and pharmacological treatments have improved outcomes for many, the variability in results underscores the need for personalized approaches that consider the patient's age, seizure type, comorbidities, and social context. Future research should aim to refine diagnostic frameworks and therapeutic strategies to better address the multifaceted nature of personality changes in epilepsy.

Summary Chart

Lobe	Key Neuroanatomical Driver	Typical Personality Shift	Supporting Treatment Outcomes
Temporal Lobe Epilepsy (TLE)	Sensory-Limbic Hyperconnectivity and reduced Gray Matter Volume (GMV) in the amygdala and hippocampus	"Hyper-emotional" profile: heightened neuroticism, anxiety, social withdrawal, and Gastaut-Geschwind syndrome (hyper-religiosity/hypergraphia)	Surgical: Left-sided resections often enhance emotional stability Pharmacological: Lamotrigine acts as a mood stabilizer by inhibiting glutamate
Frontal Lobe Epilepsy (FLE)	Disruption of the vmPFC and OFC circuits; impairment of the inhibitory pathways and Theory of Mind (ToM)	Executive dysfunction: Impulsivity, social disinhibition, apparent apathy, and reduced empathy (pseudo-psychopathy)	Surgical: Right-sided resections significantly reduce impulsivity and anxiety Pharmacological: Topiramate may worsen shifts via cognitive "dulling"

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LIMITATIONS

Several limitations should be considered when interpreting these findings. First, the screening, data extraction, and quality assessment processes were conducted by a single reviewer, which may introduce slight subjective selection bias. Second, the literature search was restricted to a single database, ScienceDirect; consequently, relevant studies indexed exclusively in other biomedical and psychological databases, such as PubMed or Embase, were omitted. Finally, the search was also constrained by an open-access filter, which likely excluded certain high-quality, subscription-restricted data. These combined factors somewhat limit the overall comprehensiveness of this review and may introduce publication bias.

CONCLUSION

This paper investigated the complex mechanisms behind personality changes in epilepsy, particularly focusing on the roles of neuroanatomy, seizure activity, and treatment interventions. The evidence suggests that personality alteration is not a singular phenomenon but a divergence strongly influenced by localized seizure activity; while temporal lobe involvement predicts heightened neuroticism and a “hyper-emotional” profile driven by both limbic hyperconnectivity and neuro-inflammatory cycles, frontal lobe involvement typically manifests as executive dysfunction and impaired social cognition.

Furthermore, identifying a bi-directional feedback loop between chronic stress and microglial priming in the brain highlights that personality traits such as neuroticism are active participants in lowering seizure thresholds. The added complexity of the pharmacological “fingerprint” imposed by AEDs further adds challenge in concrete diagnoses. Ultimately, these findings advocate for a shift towards a more personalized neuro-behavioral framework in epilepsy management. By integrating predictive growth tailored therapeutic or drug-based strategies, clinicians may be able to better address the multifaceted nature of personality changes, and move closer to a model of care that preserves the individual's sense of self.

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