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Review Article

From Seizure Control to Parasite Management: Antiepileptic Drugs and Neurocysticercosis

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ABSTRACT

Epilepsy is a chronic brain condition that affects more than 65 million people worldwide, with 30% of patients unable to control seizures, despite progress in Antiepileptic Drugs (AEDs). This article exploring the history of AEDs, focusing on new AEDs such as cenobamate and fenfluramine, and it also explores the reason behind the withdrawal of certain drugs like felbamate, trimethadione, vigabatrin, and phenacemide. These withdrawals were primarily for some of the adverse effects, including liver failure, aplastic anemia, and visual disturbances that emerged after these drugs entered the market. The paper also addresses epilepsy caused by neurocysticercosis, a parasite infection from the tapeworm Taenia solium, discusses treatment strategies involving cysticidal drugs like praziquantel and albendazole, and along with prevention measures such as improved hygiene and vaccination. The paper emphasizes the importance of ongoing research for developing safer and more effective treatments.

Keywords: Cenobamate, Epilepsy, Felbamate, Neurocysticercosis, Trimethadione, Vigabatrin.

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INTRODUCTION

Epilepsy is a neurological disorder (Botros *et al*., 2013). According to the World Health Organization (WHO), 65 million people worldwide suffer from epilepsy, with 80% of those affected residing in low-and middle-income countries. Epilepsy is an uncontrollable neurological disorder causing recurrent and unpredictable seizures, occurring seven times more frequently than other chronic illnesses (Bhor *et al*., 2024). The US spends approximately $15.5 billion annually on medical costs related to seizures (Fisher *et al*., 2005). To reduce treatment costs, safer and more efficient anticonvulsants are required, especially considering the increasing prevalence of epilepsy in the US and India. Many countries are actively working to develop novel, safe, and effective medications for epilepsy (Bhor *et al*., 2024). Epilepsy is commonly seen in patients with other neurological conditions like prolonged febrile seizures, Parkinson's disease, traumatic brain injury, and stroke. It can also result from brain infections, severe brain damage, and other cerebral illnesses (Cretin *et al*., 2017) and Common causes of encephalitis include TB, neurocysticercosis, cerebral toxoplasmosis, herpes simplex, human herpes virus (Bhor *et al*., 2024), and bacterial meningitis. Brain tumor, head injuries, alcohol use, discontinuation of antiepileptic medication, and metabolic disturbances like acidosis, organ failure, and electrolyte imbalances can also contribute to epilepsy (Tan *et al*., 2010) (Table 1). Epilepsy often leads to cognitive deficits, particularly affecting learning and memory due to hippocampal injury (Baxendale, 2020). Studies show that epileptic patients have higher death rates, dying 2–10 years earlier than the general population (Beghi, 2020) and uncontrollable storms and excessive, fleeting neuronal discharges were the causes of epilepsy. League Against Epilepsy (LAE) criteria include two unprovoked seizures during the next decade, one unprovoked (or reflex) seizure, and the possibility of more seizures based on the overall likelihood of recurrence (at least 60%). The mid-1800s saw the first reports of potassium bromide, which was the first anticonvulsant medication. Since then, a variety of AEDs have been developed and authorized by researchers and medical professionals, becoming a first-line treatment for individuals with epilepsy. The novel anticonvulsants used to treat focal seizures are the result of modern research. However, not all types of epilepsy can be cured with these newer medications. There are several types of epilepsy, including focal and generalized seizures, clonic seizures, absence seizures, and others (Bhor *et al*., 2024).

The primary diagnostic tool for individuals presenting with seizures is the 2017 revision of the International League Against Epilepsy (ILAE) classification scheme (Hakami, 2021).

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Innovations in the Treatment of Epilepsy: FDA Approvals of Fenfluramine and Cenobamate

Over twenty second-generation AEDs have been approved during the previous three decades; from 2008 to 2020, 10 new AEDs were launched. More than thirty percent of epileptics still struggle to control their seizures even with progress in medicine (Table 2). Even worse results occur in certain epilepsy disorders. More modern and potent therapies are desperately needed. Creating treatments with increased safety and tolerability is also crucial (Bialer *et al.*, 2020). In recent years, two new antiseizure medications, cenobamate and fenfluramine, have been approved by the U.S. Food and Drug Administration (FDA).

Patients with Developmental and Epileptic Encephalopathies (DEEs) are treated with Fenfluramine (FFA), an Antiseizure Medicine (ASM) having serotonergic and sigma-1 receptor action. It is approved in the US to treat seizures linked to Lennox-Gastaut Syndrome (LGS) and Dravet Syndrome (DS) in patients ≥2 years old. In the EU, UK, and Japan, it is also approved to treat seizures linked to DS and LGS in patients of a similar age (Wirrell *et al.*, 2024).

Adult patients with partial-onset seizures (seizures that involve only one part of the brain) can be treated with cenobamate, a recently FDA-approved medication (Latimer *et al.*, 2021).

Phenytoin is an anticonvulsant medication primarily used in the treatment of epilepsy and seizure disorders. Its mechanism of action involves the following key processes:

Adverse Effects

Phenytoin has been prescribed for about 75 years, and while there are numerous known side effects-some occurring at therapeutic plasma concentrations after prolonged use and others manifesting as toxicity due to overdose-few new negative impacts have emerged recently. Many of these side effects resemble overdose symptoms, and some are clearly related to the drug's concentration in the body (So and Penry, 1981).

Therapeutic-level adverse effects

Phenytoin can cause gum hypertrophy, hirsutism, hypersensitivity reactions, and megaloblastic anemia since it reduces folate absorption and increases its excretion, leading to this condition. Osteomalacia Hyperglycaemia can be caused by phenytoin, which inhibits insulin release, resulting in elevated blood sugar levels (livanainen and Savolainen, 1983). Fetal hydantoin (Table 3) syndrome can also be caused by phenytoin taken during pregnancy, which features hypoplastic phalanges, cleft palate, hare lip, and microcephaly, most likely due to its arene oxide metabolite (Hanson and Smith, 1975).

Adverse Effects at high plasma levels (dose-related toxicity)

The most obvious symptoms are vestibular and cerebellar signs such as nystagmus, diplopia, vertigo, and ataxia. Patients may also experience drowsiness, hallucinations, mental confusion, stiffness, and strange conduct. When taken with food, the medication helps reduce nausea, vomiting, and epigastric pain (Wu and Lim, 2013). Because intravenous injection might cause local vascular damage, which can lead to vein thrombosis, edema, and discoloration of the injected limb, the injection rate shouldn't exceed 50 mg/min. In most cases, only intravenous injections result in cardiac arrhythmias and a drop-in blood pressure (Eadie, 2015).

Withdrawal of Anticonvulsant Medications

There are a few reasons why some anticonvulsant medications have been taken off the market, but the main one is that the drugs are not readily available in local markets, forcing patients to travel to nearby towns to get their medications, which results in lost working days, lost income, and increased travel expenses. Another reason is that the high cost of treatment puts a financial strain on families, which also causes therapy to be discontinued. Finding unexpected but substantial side effects that might not show up until after extended exposure or in particular patient subgroups that were not included in registration trials is another crucial consideration. Routine clinical use and drug surveillance systems may uncover these side effects, which were missed in the initial clinical studies, and could result in the drug being taken off the market (Das *et al.*, 2007; Zaccara *et al.*, 2007).

Felbamate

The first antiepileptic drug to be proven effective in controlled trials is felbamate (Figure 1) which is approved to treat both focal and generalized seizures in children with Lennox-Gastaut syndrome as well as focal seizures in adults. There are several ways that felbamate operates. At therapeutic dosages, it inhibits NMDA receptors and, like barbiturates, increases GABAergic neurotransmission. Additionally, it inhibits Ca2+ and Na+ voltage-gated channels in a use-dependent fashion. GABA potentiation is the most plausible mechanism for its antiseizure impact, while seizure generalization is influenced by Na+ channel effects (Löscher *et al.*, 2021a).

The FDA approved felbamate in 1993, which was a huge advancement for individuals whose seizures were not well controlled or who were experiencing severe adverse effects from previous medicines. In less than a year, 126,000 patients received prescriptions for it, many of whom had total seizure control for the first time. In addition, many users reported an improvement in quality of life due to felbamate's lack of CNS depressive effects. However, the FDA warned about a higher-than-expected incidence of liver failure and aplastic anemia linked to felbamate

in September 1994. Based on some study on Felbamate's chemical structure, which consists of a 1,3-dioxolane ring attached to a phenyl group and an amide functional group, suggest that it may induce liver damage as well as aplastic anemia. The amide group (-CONH2) undergoes metabolic activation by cytochrome P450 enzymes in the liver, producing toxic species that enhance the damage to the liver as well as suppress bone marrow activity. Further to this, the aryl ring also reacts with the liver enzymes via electrophilic reactions and forms toxic products that worsen the degree of liver injury. The presence of a 1,3-dioxolane ring impacts the pharmacokinetic properties of the drug on its absorption and metabolism. As a result, when 33 cases of aplastic anemia and 18 cases of hepatic failure were reported between January and October 1994, most physicians stopped prescribing the drug. At the time, only 12,000 people were still on the drug, and this number has not changed. Remarkably, since then, neither new patients nor those who continued using felbamate have been reported to develop aplastic anemia. While some have maintained that complicating factors may have exacerbated the initial risks, others opine that long-term exposure diminishes the probability of substantial adverse outcomes (French *et al*., 1999).

Trimethadione

It was first approved in the United States in 1946 for this particular purpose. It was effective in treating absence seizures and acts as a blocker of T-type calcium channels at a period when there were few alternative options. Moreover, it possesses neuroprotective properties. The chemical structure of trimethadione (Figure 2) itself does not directly explain its withdrawal. Instead, its metabolism, reactivity, and associated severe side effects, along with the development of better alternatives, led to the decision to remove it from the market. Some studies suggest that trimethadione was withdrawn due to its limited use, with only about 150 patients remaining on the drug, and changes in medical practice that favored newer Antiepileptic Drugs (AEDs) with improved benefit/risk profiles. Additionally, the manufacturer faced challenges in maintaining the supply of trimethadione due to complex manufacturing processes and regulatory validation issues (Caplan *et al*., 2019; Kopecky *et al*., 2014).

Sabril (Vigabatrin)

Vigabatrin is a structural analogue of Gamma-Aminobutyric acid (GABA) that increases brain GABA levels by irreversibly inhibiting GABA-transaminase (GABA-T). It is used to treat partial seizures of refractory complex and infantile spasms. GABA-T is irreversibly inhibited by vigabatrin (Figure 3) which functions as a structural analogue of GABA and raises GABA levels in the brain. This process amplifies GABA's CNS inhibitory effects, which helps explain why it has anticonvulsant qualities.

Vigabatrin is severely limited in several nations, including the US and Europe, but has not been completely removed from the market. The drug's negative effects, including peripheral Visual Field Abnormalities (VFD) and retinal dysfunction, were the main reason it was removed rather than just its chemical makeup. Its action as an irreversible inhibitor of GABA-T is responsible for this, as it raises GABA levels in the brain. Some people may develop peripheral VFD as a result of altered GABA levels; the prevalence varies according to age and level of medication exposure. While central visual acuity usually is maintained, the specific negative effects frequently include bilateral nose abnormalities, which can develop into concentric bilateral field defects. As a result of these serious concerns about its safety profile, particularly regarding visual side effects, vigabatrin was withdrawn from the market. The FDA rescinded the approvable letter granted to Hoechst Marion Roussel in 1997, highlighting regulatory apprehensions about the drug's association with these adverse effects (James Willmore *et al*., 2009).

Phenacemide

Phenytoin's ring-opened analog is phenacemide (Figure 4). Structure of Although there are no well-controlled clinical trials that have confirmed its efficacy, it is known to be beneficial

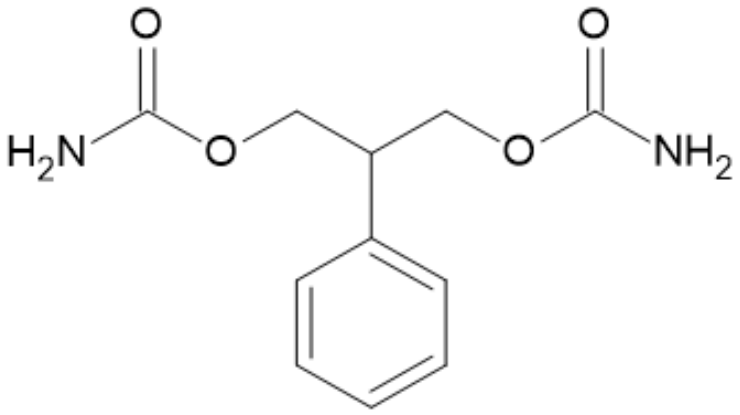


Figure 1: Structure of Felbamate(Palmer and McTavish, 1993).

against both localized and generalized tonic-clonic seizures. Phenacemide mainly functions by stabilizing the neuronal membrane, decreasing neuronal excitability, and blocking voltage-gated sodium channels. Additionally, it may improve GABA and Adrenergic inhibition and alter glutamate receptors to reduce seizure activity. Phenacemide has been taken off the market because of serious safety issues, such as links to hepatic failure, aplastic anemia, and Congenital Abnormalities (CAs) such as cleft palate and other deformities. These hazards played a role in the decision to remove it, especially when considering AEDs and their possible teratogenic consequences (Czeizel and Bnhidy, 2011).

Clinical details of Neurocysticercosis (NCC)

NCC is the most chronic parasite infection that affects the Central Nervous System (CNS) of humans. The larval form of Taenia solium, also referred to as the "pork tapeworm," is the cause. The illness known as "taeniasis," which is brought on by adult tapeworms, must be separated from NCC. Ingestion of food or water tainted with *T. solium* eggs results in NCC. These larvae move throughout the body after hatching in the intestines, but they have a special preference for the CNS, though they may penetrate other tissues. Depending on the size and number of cysticerci, their developmental stage, and their location in the brain, the clinical signs and symptoms of NCC might differ significantly. It is difficult to diagnose and stage the disease accurately because of this diversity. In emerging Asia, Latin America, and sub-Saharan Africa, NCC is very prevalent. According to estimates from the WHO, over 2 million people in these areas have epilepsy brought on by a *T. solium* infection, with cysticercosis being the most frequently preventable cause of epilepsy. In these regions, symptomatic neurocysticercosis is responsible for about one-third of seizure disorders. Other neurological disorders, such as recurring and persistent headaches, are also linked to NCC and are probably underdiagnosed. Reducing the burden of NCC and its related problems requires efficient detection and treatment due to its substantial public health impact (Gripper and Welburn, 2017; Fogang *et al.*, 2015). The transmission and life cycle of NCC were depicted in (El-Kady *et al.*, 2021a) (Figure 4).

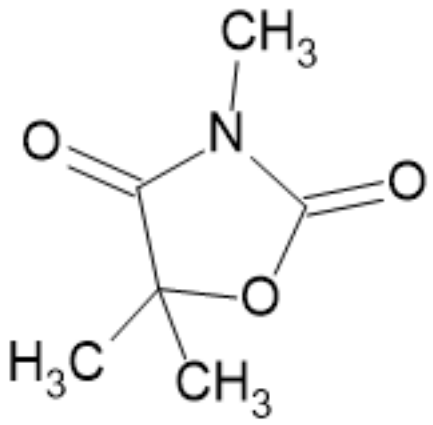


Figure 2: Structure of Trimethadione (Wells *et al.*, 1989).

Table 1: Classification of seizures.

Types of Epilepsy	Subcategory	Symptoms	Severity
Focal Epilepsy	Focal Aware Seizures	Unusual sensory experience (e.g., tingling and smell)	Generally mild; person is aware.
	Focal Impaired Awareness Seizures	Confusion, loss of awareness; may perform automatic behaviors.	Moderate; awareness impaired.
	Focal to Bilateral Tonic-Clonic Seizures	Begins localized, then generalizes, causing convulsions.	Loss of consciousness.
Generalized Epilepsy	Generalized Tonic-Clonic Seizures	Stiffening of the body followed by rhythmic jerking.	Loss of consciousness.
	Myoclonic Seizures	Sudden, brief jerks of muscles may affect one or more limbs.	Mild to moderate; often brief.
	Absence Seizures	Brief lapses in awareness, staring episodes.	Generally mild; can disrupt daily activities.
	Atonic Seizures	Sudden loss of muscle tone, causing falls.	risk of injury.
	Infantile Spasms	Sudden jerking movements in infants, often in clusters.	can impact development.
Combined Generalized and Focal Epilepsy		Mixed symptoms from both focal and generalized types.	can range from mild to severe.
Unknown Onset Epilepsy		Symptoms unclear; may resemble either focal or generalized.	severity unknown.

Table 2: Mechanism of action of Phenytoin.

Mechanisms	Target	Effect	Outcome
Phenytoin inhibits voltage-gated sodium channels by binding preferentially to their inactivated state and delaying the conformational recovery of the channels.	Sodium Channels (Voltage-Gated)	Decreases the sodium current flowing inward. Delays the process of recovering from the inactivated condition.	Reduces the chance of seizures by stabilizing neuronal membranes and limiting recurrent neuronal activation (White *et al.*, 2007).
Phenytoin inhibits glutamate release and calcium influx into neurons by acting as an antagonist at high-voltage-activated calcium (Ca2+) channels.	Calcium Channels (High Voltage)	Prevents calcium from entering. Glutamate, an excitatory neurotransmitter, is released less frequently.	Minimizes excitatory neurotransmission. Prevents seizures and excessive firing of Neurons (Sills and Rogawski, 2020).
Phenytoin increases GABA levels in the brain and increases chloride ion conductance through GABA receptors to improve GABAergic function.	GABAergic System	Increases the brain's GABA levels. Enhances post-synaptic inhibition mediated by GABA. Through GABA-A receptors, chloride inflow is enhanced.	Enhances GABAergic inhibition to promote CNS stability; increases inhibitory signals to decrease excitability (Chweh *et al.*, 1986).
By inhibiting sodium channels and presynaptically modifying calcium channels, phenytoin lowers glutamate release.	Glutamate Release	Prevents glutamate from being released. Decreases the amount of calcium that enters neurons.	prevents excitotoxicity, especially in regions such as the retina and optic nerve. Prevents damage to neurons (Davies, 1995; Bartollino *et al.*, 2018).
Potassium (K^+) channels are modulated by phenytoin, which lowers K^+ current and influences the repolarization phase of action potentials.	Potassium Channels (Voltage-Dependent)	Action potentials are prolonged by lowering potassium current. Repolarization and membrane stability are impacted.	decreases the likelihood of seizures by stabilizing the membrane potential and reducing neuronal excitability (Sills and Rogawski, 2020).

Humans are the definitive hosts for the adult Taenia solium tapeworm, which resides in the human intestines. These adult worms produce eggs that are excreted in human feces. Pigs become intermediate hosts when they consume food, water, or environmental sources contaminated with T. solium eggs, typically in unsanitary conditions. The eggs develop into oncospheres after consumption, which penetrate the intestinal wall and grow into pig muscles called cysticerci. By eating undercooked pork that has these cysticerci, humans can contract the infection. In addition, unsanitary surroundings can spread the disease from person to person because contaminated food, water, or surfaces can harbor T. solium eggs. Human cysticerci have the ability to mature into adult tapeworms in the intestines, where they release eggs to carry on the parasite's life cycle (Butala *et al.*, 2021a; Bonnet *et al.*, 2022).

The symptoms of NCC could differ according to where the cyst is located in the central nervous system. Common symptoms include headaches (50%), fatigue (22.2%), papilledema (47.2%), elevated intracranial pressure (up to 75%), seizures (50-80% of those with parenchymal cysts), and cysticercosis meningitis (72.2%). Indications of meningeal irritation, mental symptoms (such as depression, disorientation, and psychosis), and cognitive impairment are also common. Hydrocephalus (20-30%), cranial nerve deficits (13.9%), ataxia (5.6%), focal deficits, and modest motor impairments are possible additional neurological problems. Elevated intracranial pressure is a possible consequence, and in extreme situations, hydrocephalus, cognitive impairment, and persistent meningitis may develop (El-Kady *et al.*, 2021b; Bazan *et al.*, 2016).

Based on clinical evaluation, which includes the patient's history, neurological examination, neuroimaging (CT or MRI), and symptoms like seizures or visual problems, neurocysticercosis is diagnosed. Neurocysticercosis in the CNS can be diagnosed with both CT and MRI. However, when employing 3D volumetric sequences, MRI is more effective than CT at detecting intraventricular and extra parenchymal cysts, including those in the CSF. Neurocysticercosis can also be identified by serological assays such as quantitative Polymerase Chain Reaction (qPCR), antibody-based antigen detection, EITB, and ELISA (Carpio *et al.*, 2018; Brutto, 2022; Garg *et al.*, 2024). NCC can be controlled with or without cyst removal. To eradicate the parasites, chemotherapy usually uses cysticidal medications like Praziquantel (PZQ) and/

or Albendazole (ABZ). In order to stop the symptoms brought on by heightened inflammatory responses from getting worse, corticosteroids are also advised, at least for the first four days of treatment (Butala *et al.*, 2021b; Filho *et al.*, 2021).

Drug-based treatments have been successful in lowering the prevalence of cysticercosis and taeniasis in both humans and pigs. Human and pig cysticercosis cases are significantly reduced by annual chemotherapy, which prevents 94% of human cases and 74% of pig cases. Although they are less effective, practices including improved animal husbandry, sanitation, and meat inspection can also lower the prevalence of disease, either by themselves or in conjunction with medication treatments. Initiatives for health education have been successful in encouraging behavioural changes to prevent taeniasis and cysticercosis, even though their long-term viability is questionable. Research on vaccination, especially with synthetic peptide vaccines, has demonstrated potential in preventing human neurocysticercosis by managing porcine cysticercosis. Finally, managing and maybe eliminating taeniasis and cysticercosis can be accomplished through the application of mathematical modelling (Winskill *et al.*, 2017).

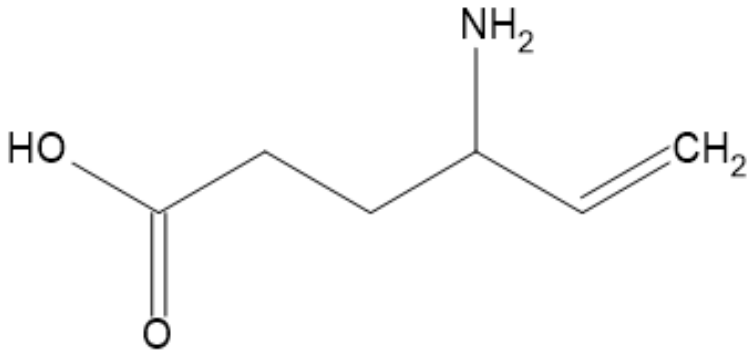


Figure 3: Structure of Sabril (Vigabatrin)(Ben-Menachem, 2011).

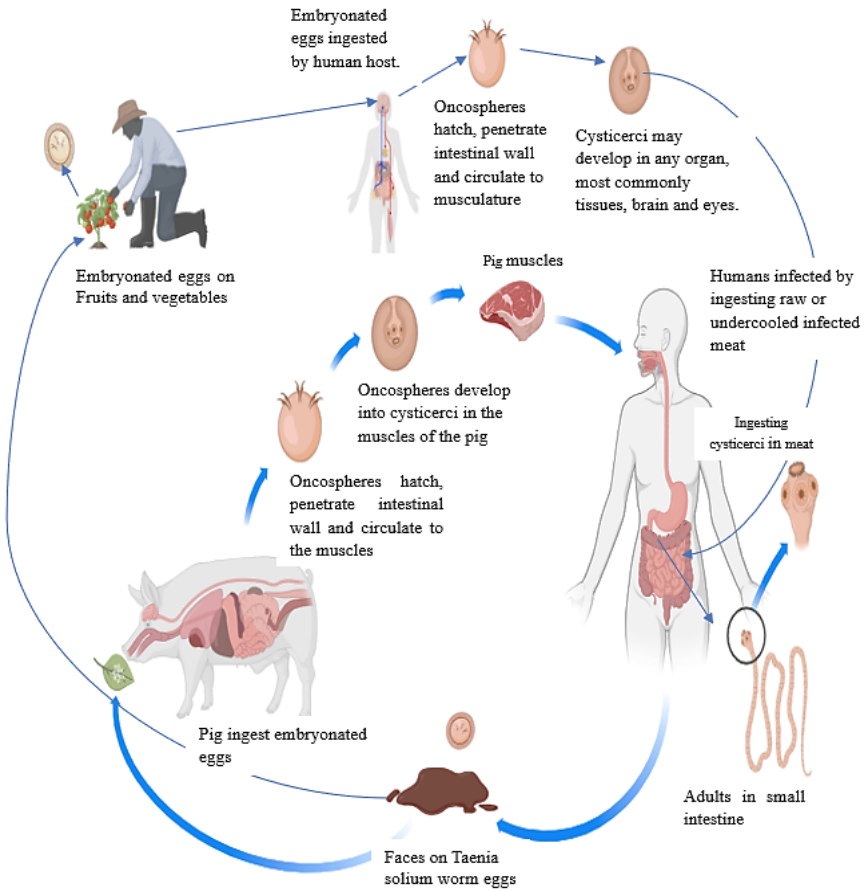
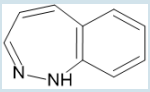
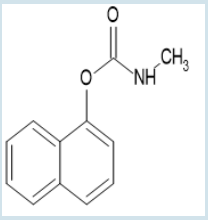
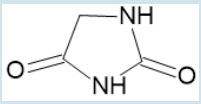
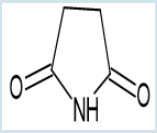


Figure 4: Transmission and life cycle of NCC (El-Kady *et al.*, 2021a).

Table 3: Popular, safety, and efficacy nucleus leads to anticonvulsant activity.

Nucleus	Structure Description
Benzodiazepine 	The bicyclic benzodiazepine nucleus is formed by fusing a seven-membered 1,4-diazepine ring with three double bonds and two nitrogen atoms to a benzene ring (5-phenyl-1,3-dihydrobenzo[e] [1,4] diazepine)(Saari *et al.*, 2011; El-Gamal *et al.*, 2022).
Carbamate 	The carbamate group is the carbamate nucleus, which is formed up of a carbonyl group (C=O) joined to a nitrogen atom (NH) to form a –NH-C=O bond. Alkyl groups, such as methyl or ethyl, are attached to the nitrogen in alkyl carbamates. Monocarbamates and dicarbamates can have distinct structures. In order to increase GABA A receptor activation and produce anticonvulsant effects, this nucleus plays a key role (Löscher *et al.*, 2021b).
Hydantoin 	Imidazolidine-2,4-dione is a five-membered heterocyclic structure that contains two nitrogen atoms and two carbonyl groups. Functional sites are available in positions 1, 3, and 5 (Gawas *et al.*, 2024; He *et al.*, 2010).
Succinimide 	A five-membered ring (pyrrolidine-2,5-dione) with two carbonyl groups (C=O) and a nitrogen atom builds up the succinamide nucleus. With functional groups at positions two and five, the structure is cyclic. Its anticonvulsant qualities may be affected by ring modifications, such as the addition of substituents on the phenyl group. The action of anticonvulsants based on succinimide depends on this nucleus (Sadiq *et al.*, 2015; Kocharov *et al.*, 2019).

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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ABBREVIATIONS

AEDs: Antiepileptic drugs; **WHO:** World Health Organization; **LAE:** League against Epilepsy; **ILAE:** International League against Epilepsy; **FDA:** Food and Drug Administration; **DEEs:** Developmental and epileptic encephalopathies; **FFA:** Fenfluramine; **ASM:** Antiseizure medicine; **LGS:** Lennox-Gastaut syndrome; **DS:** Dravet syndrome; **Ca2+:** Calcium; **K^+:** Potassium; **C=O:** Carbonyl Group; **N:** Nitrogen; **CONH2:** Amide group; **GABA:** Gamma-aminobutyric acid; **GABA-T:** GABA-transaminase; **VFD:** Visual field abnormalities; **CAs:** Congenital Abnormalities; **NCC:** Neurocysticercosis; **CNS:** Central nervous system; **PZQ:** Praziquantel; **ABZ:** Albendazole; **qPCR:** Quantitative Polymerase Chain Reaction.

ETHICAL STATEMENT

This study did not involve any human or animal subjects requiring ethical approval.

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