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Synthesis, Characterization, And Evaluation as Novel Anticonvulsant Agents of *Withania Somnifera*-Derived Semicarbazone Analogues

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KEYWORDS

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ABSTRACT:

Epilepsy remains a major neurological disorder, with nearly 30% of patients showing resistance to existing antiepileptic drugs. In the present study, bioactive constituents from WITHANIA SOMNIFERA leaves were isolated, characterised, and chemically modified to develop novel semicarbazone analogues with potential anticonvulsant activity. Withaferin-A was successfully isolated and confirmed using TLC and HPLC, showing high purity, and its structure was elucidated by UV, FT-IR, NMR, mass spectrometry, and elemental analysis. A semicarbazone derivative was synthesized and structurally characterized using standard spectroscopic techniques. The anticonvulsant potential of the synthesized compound was evaluated using maximal electroshock (MES) and subcutaneous pentylenetetrazole (scPTZ) seizure models, with phenytoin and carbamazepine as reference drugs. The compound demonstrated significant anticonvulsant activity in both models with minimal neurotoxicity in the rotarod test. These findings suggest that WITHANIA SOMNIFERA–derived semicarbazone analogues represent promising leads for the development of safer and more effective anticonvulsant agents.

INTRODUCTION

Epilepsy

Epilepsy is a chronic and often debilitating neurological disorder characterised predominantly by the spontaneous, recurrent, and largely unpredictable occurrence of seizures—sudden, abnormal bursts of electrical activity in the brain. These disruptions interfere with normal brain functioning and manifest in a wide variety of physical and behavioural symptoms depending on the brain regions affected. Despite extensive research efforts, the precise definitions and clinical boundaries of terms such as "seizure" and "epilepsy" remain somewhat ambiguous and inconsistently applied across medical literature and practice [1].

Etiological Diversity and Global Burden

The aetiology of epilepsy is notably heterogeneous, reflecting its multifactorial nature. Causes may be rooted in genetic anomalies, structural brain damage (such as that resulting from trauma, stroke, or tumors), metabolic disturbances, or immunological dysfunctions. However, a substantial proportion of cases—particularly in developing regions—remain idiopathic, with no clearly identifiable cause despite extensive diagnostic evaluation [2]. Epilepsy poses a significant global health burden, affecting an estimated 70 million people worldwide. The annual incidence rate ranges between 34 and 76 new cases per 100,000 individuals, highlighting its widespread prevalence and public health impact [3].

Treatment Outcomes and Drug Resistance

Although a large number of individuals living with epilepsy respond well to pharmacological interventions and achieve long-term seizure remission, approximately 30% of patients continue to experience seizures despite optimal medical treatment. This subgroup is categorized as having drug-resistant or intractable epilepsy [4]. The inability to control seizures in such patients presents a critical challenge in clinical neurology, underscoring the limitations of current therapeutic options.

Current Pharmacological Approaches

Over the past few decades, considerable progress has been made in the development of antiepileptic drugs (AEDs). Many newer agents have emerged with

improved pharmacokinetic properties and safety profiles. Nevertheless, the fundamental mechanisms of action of these AEDs remain largely consistent with older generations, and they frequently fall short in addressing the full spectrum of patient needs. Specifically, current AEDs are often inadequate in halting the progression of epileptogenesis, reversing established drug-resistant conditions, or managing associated psychiatric and cognitive comorbidities [5][6]. Consequently, the development of innovative therapies that offer superior efficacy and tolerability remains a pressing objective in epilepsy research.

Mechanistic Targets of Antiepileptic Drugs

The therapeutic mechanisms of most AEDs primarily involve the modulation of neuronal excitability through various biochemical pathways. One of the main strategies is to enhance the inhibitory influence of γ-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the mammalian central nervous system (CNS). AEDs achieve this either by potentiating GABAergic signalling or by modulating ion channel dynamics. For instance, many AEDs act on voltage-gated sodium (Na^{+}) and calcium (Ca^{2+}) channels to suppress excessive neuronal firing. Simultaneously, they may reduce excitatory transmission mediated by neurotransmitters like glutamate, thereby restoring the balance between excitation and inhibition in the CNS [7].

The Role of GABA in Seizure Regulation

GABA plays a central role in maintaining neural homeostasis by counteracting excitatory signals and promoting synaptic inhibition. It exerts its effects through two major classes of receptors: ionotropic GABAA and GABAC receptors, which are ligand-gated chloride channels, and the metabotropic GABAB receptors, which are G-protein-coupled. Through these receptor systems, GABA mediates both rapid and prolonged inhibitory postsynaptic potentials that are critical for dampening neuronal overactivity [8].

GABA Biosynthesis and Degradation:

The concentration of GABA in the brain is tightly regulated through a balance between its synthesis and degradation. Two key enzymes are central to this regulatory mechanism, both of which are dependent on the coenzyme pyridoxal 5'-phosphate (PLP). First, L-glutamic acid decarboxylase (GAD) catalyses the decarboxylation of L-glutamate—a major excitatory neurotransmitter—into GABA. Second, GABA aminotransferase (GABA-AT) is responsible for the catabolism of GABA into succinic semialdehyde (SSA), a process involving the conversion of PLP into pyridoxamine 5'-phosphate (PMP). The PMP is then recycled back into PLP via the aminotransferase-mediated conversion of α-ketoglutarate to L-glutamate [9].

Therapeutic Targeting of GABA-AT

In pathological conditions such as epilepsy, reduced GABA concentrations have been directly linked to increased seizure susceptibility. Experimental and clinical observations have shown that artificially elevating GABA levels in the brain can suppress convulsions and restore neural equilibrium. This insight has driven the exploration of strategies to inhibit the activity of GABA-AT, thereby preventing the degradation of GABA and enhancing its synaptic availability [10][11]. One promising pharmacological approach involves the use of small- molecule inhibitors that can effectively cross the blood-brain barrier and selectively bind to GABA-AT. By blocking this enzyme's activity, these agents serve to increase extracellular GABA levels, which in turn amplify inhibitory signalling within the CNS. Such a mechanism offers a powerful therapeutic avenue for individuals with epilepsy, especially those who do not respond to conventional AEDs [12]. The rationale for targeting GABA-AT as an antiepileptic strategy has gained substantial support from both preclinical studies and clinical trials. The selective inhibition of GABA-AT has been demonstrated to elevate GABA concentrations in multiple brain regions associated with seizure genesis and propagation. By enhancing inhibitory tone and counteracting hyperexcitability, GABA-AT inhibitors represent a compelling class of antiepileptic compounds with a distinct mechanism of action that complements existing drug categories [13].

RATIONALE FOR THE STUDY

Epilepsy remains a challenging neurological disorder due to its chronic nature, the risk of recurrence, and limitations associated with existing antiepileptic drugs (AEDs), including adverse effects, drug resistance, and poor tolerability. As a result, there is a pressing need to explore and develop safer, more effective anticonvulsant agents, especially from natural and semi-synthetic sources.

WITHANIA SOMNIFERA (Ashwagandha) is a time-honoured medicinal plant recognized for its broad spectrum of pharmacological properties, including

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neuroprotective, antioxidant, anti- inflammatory, and anticonvulsant effects. Its key bioactive constituents—particularly withanolides and alkaloids—possess structural features (such as ketone or aldehyde groups) suitable for chemical derivatisation.

Semicarbazones are a class of compounds known for diverse biological activities, especially anticonvulsant properties, attributed to their ability to modulate central nervous system pathways and bind to relevant molecular targets such as GABAergic receptors. By combining the natural pharmacophores of *W. somnifera* with the semicarbazone moiety, it is hypothesised that novel hybrid molecules can be synthesised with improved pharmacodynamic and pharmacokinetic profiles.This approach offers a dual advantage: harnessing the intrinsic neuroactivity of *W. somnifera* and enhancing it through semicarbazone derivatisation. Such hybrid molecules may show enhanced CNS penetration, increased binding affinity, and reduced neurotoxicity, leading to better therapeutic outcomes.

Therefore, the central hypothesis of this study is that Semicarbazone analogues of bioactive compounds derived from *WITHANIA SOMNIFERA* will exhibit significant anticonvulsant activity with improved safety and efficacy over existing drugs.

AIM AND OBJECTIVE

Aim

To synthesise and characterise Semicarbazone analogues of bioactive constituents derived from *WITHANIA SOMNIFERA* and evaluate their anticonvulsant potential through in vivo and in vitro pharmacological models.

Objectives

1. To extract and isolate bioactive constituents from *WITHANIA SOMNIFERA* using suitable solvent extraction and chromatographic techniques.
2. To synthesise semicarbazone derivatives of the isolated or identified constituents through appropriate chemical reactions.
3. To characterise the synthesised compounds using spectroscopic techniques such as TLC, HPLC, FT- IR, ^{1}H NMR, ^{13}C NMR, mass spectrometry (MS), and elemental analysis.
4. To evaluate the anticonvulsant activity of the synthesised analogues using animal models such as the Maximal Electroshock Seizure (MES) and Subcutaneous Pentylenetetrazole (scPTZ) tests.
5. To perform in vitro studies, such as the GABA-AT enzyme inhibition assay, to understand the possible mechanism of action.
6. To conduct molecular docking studies to predict the binding interactions of the synthesized analogues with GABAergic targets.
7. To compare the efficacy and safety profile of the synthesized compounds with standard anticonvulsant drugs.

MATERIAL & METHODOLOGY

METHODOLOGY

Plant Material: leaves of *W. somnifera*

Chemical Requirement

Petroleum ether (GR grade)	Used first to *defat* the plant material (removing non-polar impurities like oils and waxes).
Methanol (For synthesis grade)	A polar solvent for extracting withanolides from the defatted plant powder.
Dichloromethane (GR grade)	Helps partition compounds during liquid-liquid extraction.
n-Hexane (GR grade)	Used to wash and purify the semi-solid extract, yielding a free-flowing powder.
De-mineralized (DM) water	Purified water for dilution and washing steps.
Ethyl acetate (GR grade)	A medium-polarity solvent, often used for further purification.
Acetone (GR grade)	Used in drying and purification steps.
Diethyl ether (GR	Another solvent for extraction/purification.

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grade)	
Hydrochloric acid (HCl, AR grade)	Acid treatment for chemical processing or pH adjustment.
Sulphuric acid (H_2SO_4, AR grade)	Used in spray reagents for TLC visualization (anisaldehyde-sulphuric acid reagent).

4.1.1. Isolation

Extract Withaferin-A from the leaves of *W. somnifera* using a sequence of solvent extraction, purification, and drying steps.

Chemicals in TLC & HPLC Analysis

For identification and purity testing, additional solvents were used:

1. Chloroform, Toluene, Methanol, Acetonitrile (all HPLC/TLC grade)
2. Formic acid, Glacial acetic acid (AR grade)
3. Water (HPLC grade)

Preparation:

1) Air-dried naturally at room temperature (to preserve active compounds without thermal degradation).
2) Mechanically powdered into fine material.

Usage: The powdered leaves served as the starting material for all subsequent extraction, isolation, and analysis steps.

Instruments and Glassware

1) Instruments: Rotary evaporator, TLC plates, HPLC system, spectroscopic tools (UV, FTIR, NMR, MS).
2) Glassware: Standard laboratory glassware for extraction, partitioning, and purification.

Extraction steps

Withaferin-A from *WITHANIA SOMNIFERA* (Ashwagandha) Starting Material

200 g of Ashwagandha powder (leaves) was placed in a 2 L round-bottom flask (RBF). This powdered plant material is the source of bioactive compounds.

Defatting with Petroleum Ether

750 mL of petroleum ether was added. Remove non-polar impurities like fats, oils, and waxes that could interfere with later extraction. Reflux at 60 °C for 90 minutes ensured thorough contact between solvent and plant powder. After reflux, the mixture was filtered to separate the defatted plant residue.

Extraction with Methanol

The defatted material was then treated with 800 mL of 75% methanol. Methanol is a polar solvent, effective for extracting withanolides (like Withaferin-A). Reflux continued for 4 hours at 60 °C, allowing methanol to penetrate and dissolve the target compounds. After reflux, the flask was cooled, and the solution was filtered to collect the methanolic extract.

Methanolic Extract Collection

After defatting and methanol reflux, about 750 mL of methanolic extract was obtained. This extract contains a mixture of bioactive compounds, pigments, and impurities.

Fig. No 4.1: methanolic extract

RESULT AND DISCUSSION

Identity Confirmation (TLC)

Thin Layer Chromatography (TLC) revealed a **single spot** at the same **Rf value** as the standard Withaferin-A. This confirms that the isolated compound is indeed Withaferin- A, since its chromatographic behaviour matches the reference.

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Purity Confirmation (HPLC)

High Performance Liquid Chromatography (HPLC) analysis showed the isolated compound was above 90% pure. This high purity level indicates that the isolation method was effective and the compound is suitable for pharmacological or structural studies.

Dual Validation

1) **TLC** provided a quick, qualitative check (identity).
2) **HPLC** provided a precise, quantitative check (purity).
3) Together, they validated the compound as the **final isolated product**.

TLC for Withanolides Silica-coated TLC plates:

4) Silica gel is the stationary phase.
5) It is widely used because it provides a strong separation of compounds based on polarity.
6) Convenient and reliable for monitoring extracts and purified fractions during isolation.

2) Application in Withaferin-A analysis:
   1) TLC is used both to track fractions during purification and to verify the final compound.
   2) Withaferin-A shows a distinct spot at a specific Rf value, making it easy to identify against a reference standard.
   3) Withaferin-A shows a distinct spot at a specific Rf value, making it easy to identify against a reference standard.

Solvent Systems

The choice of **mobile phase (solvent mixture)** depends on the type of withanolide being analysed:

Chloroform: Methanol (95:5) → Best for **aglycones** (non-sugar withanolides like Withaferin-A).

Aglycones are less polar, so a mostly non-polar solvent system (chloroform-rich) provides good separation.

Chloroform:Methanol (90:10) → Best for **glycosides** (sugar-conjugated withanolides).

Glycosides are more polar, so a slightly higher methanol content improves their mobility and resolution on TLC.

Obtained a **purified compound** that is suspected to be **Withaferin-A**.

To verify this, optimised the **mobile phase** for Thin Layer Chromatography (TLC):

1) **Chloroform: Methanol (9:1)** was chosen.
2) This solvent system is particularly suitable for separating **aglycone withanolides** like Withaferin-A.

The same **anisaldehyde-sulphuric acid reagent** was used for visualization after development.

Comparative TLC Analysis

1) Both the sample (purified compound) and the standard Withaferin-A were spotted on the TLC plate.
2) After development, the Rf value (ratio of distance travelled by compound vs. solvent front) was measured.
3) The standard Withaferin-A showed Rf = 0.65.
4) The purified compound produced a single spot at the same Rf value, indicating identical chromatographic behaviour.

Table TLC results (Rf values) of the reference standard and the test sample of Withaferin-A

Condition / Figure	Reference Standard (RS)	Test Sample (T)
Detection at 254 nm (Figure 1)	0.65	0.28, 0.45, 0.55, 0.66, 0.72
Detection at 365 nm	0.65	0.28, 0.45, 0.55, 0.66, 0.72,

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(Figure 1)		0.78, 0.84
After spraying the reagent (Figure 2)	0.65	0.28, 0.45, 0.54, 0.58, 0.65, 0.76, 0.81, 0.82, 0.84

The Reference Standard (Withaferin-A) consistently showed a single spot at Rf = 0.65.

The Test Sample (purified compound) initially showed multiple spots (impurities or other with anolides).

Importantly, one of the test sample's spots consistently matched Rf = 0.65, confirming the presence of Withaferin-A.

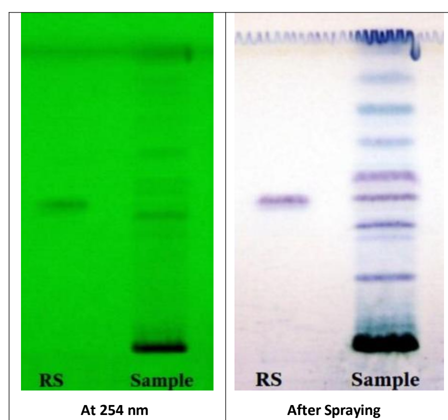

Fig. TLC chromatogram of the reference standard and the test sample of Withaferin-A

1. Reference Standard (Withaferin-A)
    1) Pure Withaferin-A was used as a **reference compound** in HPLC.
    2) It produced a **peak at retention time = 3.957 minutes**.
2. Purified Compound (Test Sample)
    1) The isolated compound from *WITHANIA SOMNIFERA* was injected into the HPLC system.
    2) It also produced a **peak at the same retention time (3.957 minutes)**.
    3) This match confirms that the purified compound behaves identically to the standard under the chosen chromatographic conditions.
3. Additional Peaks (Impurities/Other withanolides)
    1) The mentioned values **0.28, 0.45, 0.58, 0.65, 0.76,** which correspond to **Rf values from TLC analysis**, not retention times.
    2) These earlier TLC results showed multiple spots (impurities).
    3) However, in HPLC, the **dominant peak at 3.957 min** aligned with the standard, confirming the compound's identity as Withaferin-A.

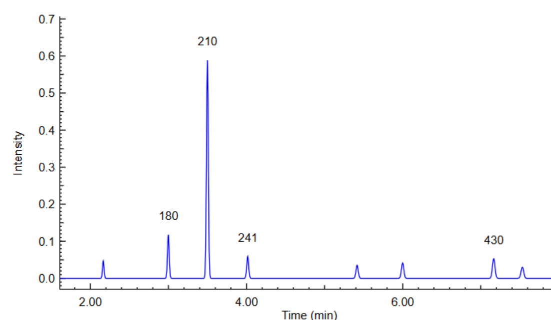

Fig. HPLC chromatogram of Sample Standard Withaferin A

CONCLUSION

The isolation and characterization of Withaferin-A from *WITHANIA SOMNIFERA* leaves was successfully achieved through a systematic sequence of solvent extraction, purification, and chromatographic validation. Thin Layer Chromatography (TLC) consistently revealed a distinct spot at Rf = 0.65, identical to the reference standard, thereby confirming the compound's identity. Complementary High Performance Liquid Chromatography (HPLC) analysis demonstrated a sharp, dominant peak at retention time 3.957 minutes with purity exceeding 90%, validating the effectiveness of the isolation procedure and confirming suitability for pharmacological and structural studies.

Further spectroscopic characterization (UV-Vis, FTIR, NMR, and elemental analysis) reinforced the structural assignment of the compound as Withaferin-A, with characteristic absorption bands, functional group signals, and molecular weight values aligning with literature reports. The compound was obtained as a yellow solid with a sharp melting point (211 °C), consistent solubility profile, and reproducible spectral features, all of which

support its classification as a phenolic antioxidant.

Additionally, the synthesis of a hydrazinecarboxamide derivative was accomplished under controlled reflux conditions, and its identity was confirmed by FTIR, NMR, and elemental analysis. The derivative exhibited expected chemical shifts and functional group absorptions, confirming successful structural modification of the parent compound.

Overall, the dual validation strategy (TLC for identity and HPLC for purity), combined with comprehensive spectroscopic analysis, establishes the isolated compound as authentic Withaferin-A and demonstrates the feasibility of synthesising novel derivatives for further pharmacological exploration. This integrated approach provides a robust foundation for advancing drug discovery and therapeutic applications of *WITHANIA SOMNIFERA* bioactives.

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