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Research Article

Assessment Of Sutherlandia Frutescens Neuroprotective Potential In A Rotenone-Induced Parkinsons Disease Rat Model.

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Abstract

In South Africa, the herb Sutherlandia frutescens (SF) has long been used to treat a variety of illnesses, including neurological diseases. The loss of dopaminergic neurons in the substantia nigra is the hallmark of Parkinson's disease (PD), a progressive neurodegenerative illness that causes motor symptoms. Both in vitro and in vivo investigations have connected the pesticide rotenone to symptoms similar to Parkinson's disease. However, there hasn't been much research done on how SF specifically affects PD symptoms. This study used an open-field test to evaluate motor behavior and in vivo electrical recordings from the hippocampus to examine the possible neuroprotective effects of SF against rotenone-induced Parkinson's disease.Rats were split up into three groups: one that received sunflower oil as a control, one that received rotenone treatment, and one that received SF extract that was hydroponically cultivated. Motor behavior was assessed using an open-field test. Rats given SF showed noticeably more motor activity than rats given either sunflower oil or rotenone, indicating that SF has an activating effect on motor behavior. The rotenone group, on the other hand, showed decreased levels of activity and exploratory behavior, underscoring the drug's inhibitory effect on motor function. According to these results, SF may provide neuroprotective benefits against rotenone-induced PD-like symptoms by modulating hippocampus neuronal activity. In an arotenone-induced Parkinson's disease model, SF, a plant with traditional medicinal uses, exhibits promise in modifying motor behavior and hippocampus neuronal activity.

Keywords : *Sutherlandia Frutescen, Open-Field Testing, Parkinson's Disease, In Vivo Electrophysiology.*

INTRODUCTION

Overview Sutherlandia frutescens (L.) (SF), also known as Lessertia frutescens in taxonomy, is a member of the Fabaceae family of legumes. For ages, different ethnic groups in southern Africa have utilized it in traditional medicine to treat a variety of illnesses, such as urogenital, gastrointestial, and gynecological conditions. The review notes that no negative treatment results have been documented from its traditional use, which is noteworthy [1]. The anticancer capabilities of SF have been the subject of numerous studies. The antiproliferative effects of SF extracts on diabetic and cancer cells have been demonstrated in vitro [2, 3]. Although SF's effectiveness as a cancer treatment has not been definitively demonstrated in human research, a few case reports indicate that it may help cancer patients feel less fatigued [4]. SF is one of the herbal medications used to treat NDDs, which include amyotrophic lateral sclerosis, Parkinson's disease (PD), and Alzheimer's diseammatory responses [8, 9]. Consuming SF was shown to reduce microglial activity in the striatum and hippocampal regions of animals with ischemic brains [10]. More clinical study is required to completely comprehend the safety and effectiveness of SF in treating a variety of illnesses, despite its lengthy history of traditional use and encouraging scientific findings. However, the evidence that is now available indicates that SF is a plant with significant therapeutic promise that merits more research.

FINDINGS

Findings In this work, the neuroprotective effects of SF were examined in a rat model of Parkinson's disease caused by arotenone. The following are the measurement parameters: 1. Electrophysiological recordings In order to evaluate

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synaptic plasticity, extracellular hippocampal spike activity was measured following high-frequency stimulation (HFS) of the entorhinal cortex (EC). 2. OFT, or open-field test: Total distance traveled and line crossings were two behavioral tests used to gauge locomotor activity and inquisitive behavior.

CONVERSATION

Conversation Although no model accurately simulates the human state, animal models are crucial for Parkinson's disease research. In animals, certain neurotoxins can cause symptoms similar to Parkinson's disease, such as motor deficits and loss of dopaminergic neurons in the substantia nigra pars compacta [29]. Protease dysfunction, oxidative stress, and mitochondrial impairment are only a few of the intricate interactions between environmental and genetic factors that contribute to the pathophysiology of Parkinson's disease. The neurodegenerative process of Parkinson's disease (PD) has been linked to excessive ROS generation, which results in the death of dopaminergic neurons [30].A 33% incidence of TD neurons was seen in the CSF group in our study (Figure 2), indicating a reduction of synaptic transmission. Complex modulation of synaptic plasticity, possibly involving both depression and potentiation pathways, is indicated by the presence of TD-PTD at 22.95% and TD-PTP at 44.3%. A strong inhibition of synaptic activity was indicated by the significantly larger percentage of TD neurons (93%) in the sunflower oil-treated group (SO, Figure 3). Seven percent of the neurons are nonreactive, which indicates that they are not responding to the applied stimuli. The percentage of TD neurons decreased to 17.74% after rotenone (R) treatment (Figure 1), suggesting a partial reversal of the synaptic suppression seen in the SO group (Figure 3). In rats, rotenone, a common pesticide, selectively degenerates nigral dopamine neurons and produces symptoms similar to Parkinson's disease [36]. Dopaminergic and nondo-paminergic neurons, as well as other brain cell types including astrocytes, are known to experience progressive neurodegeneration as a result. According to studies, rotenone can cause Parkinson's disease pathology at brain concentrations of up to 30 nM [37]. Hippocampal atrophy may be a biomarker for cognitive deterioration in Parkinson's disease, according to new research. Research has demonstrated that people with Parkinson's disease (PD) have changed hippocampal functional connectivity, including reduced connectivity with areas like the paracingulate gyrus [38].

CONCLUSIONS

Although the effectiveness of existing treatments in neurotoxin-induced animal models of Parkinson's disease (PD) is limited, there is hope that behavioral phenotyping in these animals will open the door to future treatments that are more effective. Our findings point to hydroponic SF's potential as a therapeutic agent for neurological conditions by indicating that it may modulate hippocampus activity through GABAergic systems. To fully investigate the therapeutic potential of hydroponic SF in the context of Parkinson's disease and other neurological diseases, as well as to clarify the precise pathways involved, more research is necessary.

RESOURCES AND PROCEDURES

Declarations of Ethics. The Ethics Committee of Yerevan State Medical University in Yerevan, Armenia, approved all animal studies, which were carried out in accordance with the guidelines set forth in the National Institutes of Health's (NIH) Guide for the Care and Use of Laboratory Animals (ethicalapproval number: N4 IRB). During the trial phase, every attempt was made to minimize the suffering of the animals.1. Group CSF (control + Sutherlandia): For three weeks, commencing on day 1, the rats in this group were given hydroponically administered Sutherlandia (82.6 mg/kg/day, oral administration) on alternate days. 2. Group R (rotenone): For five weeks, rats were given rotenone dissolved in sunflower oil at a dose of 2.0 mg/kg/day, subcutaneously. Group SO (sunflower oil): For five weeks, rats were given the vehicle (sunflower oil, 1 mL/kg/day, intramuscularly) every day. 4. Rotenone + Sutherlandia Group RSF: For five weeks, rats in this group were given rotenone (2 mg/kg/day, subcutaneously), and then they were given hydroponically administered Sutherlandia (82.6 mg/kg/day, oral administration) for three weeks.

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