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Evaluation of the Ameliorating Potential Effect of *Parsonsia straminea* (R.Br.) F.Muell. Stem Bark Extract on Some Behavioural Disorders Induced by *Cannabis Sativa* L. (Chanvre Cultivé (Fr), Hemp (En) Extract in a Murine Model

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Abstract

Background: The present study investigates the ameliorating potential of *Parsonsia straminea* stem bark extract on the behavioural effects induced by *Cannabis sativa* extract in murine model (mice). *Cannabis sativa* is widely known for its psychoactive properties, which can result in a range of behavioural alterations, including anxiety, depression, impaired motor coordination, cognitive dysfunction, changes in feeding behaviour, and narcolepsy-like symptoms. The present study focused on evaluating whether *P.straminea*, a plant with a history of medicinal use, could attenuate these behavioural disruptions.

Methodology: Male Swiss mice were divided into four groups (n=10): (1) control, 0.2 ml/kg normal saline (2) *C. sativa* extract, 500 mg/kg (3) *P.straminea* stem bark extract, 1000 mg/kg and (4) combined treatment (*C. sativa* + *P.straminea* extracts). Behavioural assays were performed after a 14-days treatment regimen, assessing parameters including anxiety (elevated plus maze), depression (forced swim test), motor coordination (rotarod test), cognition (Barnes-maze test), feeding rate, and sleep patterns (narcolepsy-like behaviour).

Results: Mice treated with *C. sativa* exhibited significant anxiety-like behaviours, depressive symptoms, impaired motor coordination, and cognitive deficits compared to the control group. Additionally, an increased feeding rate and altered sleep patterns, suggestive of narcolepsy-like episodes, were observed. However, mice co-treated with *P.straminea* extract showed marked improvements across all behavioural parameters. Specifically, anxiety and depression scores were significantly reduced, motor coordination was restored, and cognitive performance improved. Furthermore, feeding behaviour normalized, and sleep disturbances were attenuated, indicating that *P.straminea* extract could counteract the disruptive effects of *C. sativa*.

Conclusion: These findings suggest that *P.straminea* bark extract has a neuroprotective and ameliorative effect on the behavioural impairments induced by *C. sativa* in mice. The results support the potential of *P.straminea* as a therapeutic adjunct in mitigating the adverse behavioural consequences associated with cannabis use, particularly in managing anxiety, depression, cognitive dysfunction, motor impairment, and sleep disturbances.

Keywords: Behavioural disruptions; *Parsonsia straminea*; *Cannabis sativa*

Abbreviations: ADHD: Attention-deficit/hyperactivity disorder; THC: Δ9-tetrahydrocannabinol; OFT: Open Field Test; EPM: Elevated Plus Maze; FST: Forced Swim Test; TNF-α: Tumor necrosis factor-alpha; iNOS: Nitric oxide synthase; SOD: Superoxide dismutase; CAT: Catalase; GPx: glutathione peroxidase; SSRIs: Selective serotonin reuptake inhibitors

Introduction

Behavioural disorders encompass a wide range of mental health conditions characterized by abnormal behaviours, emotional dysregulation, and impaired social functioning. Common examples include anxiety disorders, depression, attention-deficit/hyperactivity disorder (ADHD), substance use disorders, and cognitive impairments. These disorders often result from complex interactions between genetic, environmental, and neurochemical factors, and can be exacerbated by external influences, such as drug abuse [1]. The societal impact of behavioural disorders is profound. They contribute significantly to the global burden of disease, reducing productivity, straining healthcare systems, and diminishing quality of life for affected individuals and their families. Beyond the direct costs of treatment, indirect consequences such as social stigma, unemployment, and increased risk of criminal behaviour create a cycle of marginalization and economic loss. Addressing behavioural disorders requires a multidisciplinary approach, including the exploration of novel therapies and interventions to mitigate their effects and improve overall societal well-being [2].

Role of *Cannabis sativa* in Inducing Behavioural Disorders in Experimental and Clinical Contexts

Cannabis sativa, widely known for its psychoactive properties, has been shown to influence brain function and behaviour through its active compounds, primarily Δ9-tetrahydrocannabinol (THC). Experimental studies reveal that chronic or high-dose exposure to cannabis can disrupt neurotransmitter systems, particularly those involving dopamine, glutamate, and serotonin, leading to behavioural alterations [3]. Clinically, cannabis use has been linked to increased risks of anxiety, depression, psychosis, and cognitive impairments, particularly in vulnerable populations such as adolescents or individuals with genetic predispositions [4]. Experimental models using animals further demonstrate that cannabis exposure can induce behaviours akin to anxiety, memory deficits, and social withdrawal, mirroring symptoms observed in human disorders.These findings highlight cannabis's dual role as both a potential therapeutic agent and a risk factor for behavioural disorders, emphasizing the need for further research to delineate its mechanisms of action and mitigate its adverse effects [3].

Importance of Exploring Natural Remedies like *Parsonsia straminea* for Neuroprotective or Ameliorative Effects

Natural remedies have long been a source of therapeutic agents, offering bioactive compounds with potential neuroprotective properties. *Parsonsia straminea*, a medicinal plant, holds promise for addressing neurological and behavioural disorders due to its phytochemical constituents, which may exhibit antioxidant, anti-inflammatory, and neurorestorative effects [5]. Exploring such natural remedies is crucial in the search for safer, affordable, and effective alternatives to synthetic drugs, which often come with adverse side effects. Additionally, studying plants like *P.straminea* may provide insights into novel mechanisms for mitigating the neurotoxic and behavioural impacts of substances like *C. sativa*. This approach contributes to expanding therapeutic options and advancing integrative pharmacological research.

Rationale for Selecting *Parsonsia straminea*

Parsonsia straminea was selected for its traditional use in herbal medicine due to its reported bioactive properties which make it a promising candidate for mitigating neurobehavioral disorders caused by neurotoxic agents such as *C. sativa*. Also, preliminary phytochemical studies suggest that *P.straminea* contains compounds capable of modulating oxidative stress and restoring neurotransmitter balance, both of which are critical in the pathophysiology of behavioural disorders. Its natural origin and potential safety profile further enhance its appeal as a novel therapeutic agent, warranting its investigation in this context [6].

Known Bioactive Compounds in *Parsonsia straminea*

Parsonsia straminea is known to contain bioactive compounds such as alkaloids, flavonoids, tannins, and phenolic compounds. These phytochemicals are associated with antioxidant, anti-inflammatory, and neuroprotective properties, which may contribute to its therapeutic potential in mitigating behavioural and neurodegenerative disorders [6]. Due to the observed behavioural disorders amongst undergraduates in our environment [7], there is the need to explore readily available and cost effective remedies that might have promising results, hence the aim of assessing the potential ameliorating effects of Parsonsiastraminea stem bark extract on cannabis-induced behavioural disorders.

Materials and Methods

Plant Materials

Collection, Identification, and Preparation of *Parsonsia straminea* Stem Bark Extract: The stem bark of *Parsonsia straminea* was collected from Wilberforce Island rainforest in Niger Delta region of Nigeria and authenticated by a botanist at Niger Delta University. A voucher specimen was deposited in the herbarium for future reference, NDUP/21/001. The collected stem bark was washed, air-dried under shade to prevent degradation of bioactive compounds, and ground into a fine powder using a mechanical grinder. The powdered material was extracted using 50% ethanol in a cold maceration for duration of 72 hours. The extract was filtered, concentrated under reduced pressure using a rotary evaporator, and stored at 4°C until use. This prepared extract was used for phytochemical screening and subsequent pharmacological experiments.

Cannabis Extract Preparation: Dried leaves of *Cannabis sativa* were obtained from NDLEA reserve Bayelsa state Command. The leaves were ground into a fine powder using a mechanical grinder. The powdered material was extracted using 50% ethanol] in a maceration for 72 hours to obtain the crude extract. The extract was filtered and concentrated under reduced pressure using a rotary evaporator. The resulting crude extract was stored at 4°C in an airtight container and diluted to the desired concentrations

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for experimental use. This extract was used to induce behavioural disorders in the experimental murine model.

Experimental Animals: Murine models (mice) of the (24) male gender was preferably used in this study.

Ethical Approval and Housing Conditions: The study was conducted in accordance with the ethical guidelines for animal research and received approval from the Institutional Animal Ethics Committee of the Department of Pharmacology & Toxicology, Niger Delta University, Bayelsa State, Nigeria. All procedures were designed to minimize animal suffering and ensure humane treatment throughout the experiment.The animals were housed in standard laboratory conditions, with a 12-hour light/dark cycle, controlled temperature (28±2°C), and relative humidity (50±10%). They had ad libitum access to food and water and were acclimatized to the environment for at least one week prior to the start of the experiment.

Experimental Design:

a) Group allocation (Male Swiss mice were divided into four groups (n=10): (1) control, 0.2 ml/kg/p.o normal saline (2) *C. sativa* extract, 500 mg/kg/p.o(3)*P.straminea* stem bark extract, 1000 mg/kg/p.o and (4) combined treatment (*C. sativa* + *P.straminea* extracts).

b) Duration of the study: 14 days.

Behavioral Assessments:

a) Open Field Test (OFT). As described by Gould et al in 2009 with slight modification [8]. The mice were individually placed at the centre of the OFT apparatus and allowed to move around in any direction in coordinated manner across the lines making squares in flow of the apparatus for a period of 300 seconds. This process was done prior to the designated treatments of all the mice in their respective groups as trial and was repeated 60 minutes after the designated oral treatments in all groups. The number of odd and coordinated movements were observed and recorded.

b) Elevated Plus Maze (EPM). As described by Hogg, in 1996 with slight modification [9]. Mice were place at the (close and open) arms of the EPM apparatus and allowed to explore the arms for 300 seconds. The frequency and time spent in each of the arms were observed and recorded for each mouse in all groups for determination of anxiety traits. Like the OFT, trial was done before administration of the designated oral treatments and 60 minutes after the designated oral treatments, the described procedures were applied for each mouse in all groups.

c) Forced Swim Test (FST). As described by Armario, in 2021 with slight modification [10]. Depression was determined in each mouse by placing the mouse at the centre of the improvised water bath with 60 cm wide and 20 cm depth and allowed to struggle for safety within time line of 300 seconds. This was done for each mouse in all groups prior to the administration of the designated treatments, which is referred to as trial. Sixty (60) minutes after the designated oral treatments, the described procedure above was applied to each mouse in all groups as test. The duration of struggle to swim off the water was observed and recorded according in each group.

d) Y-Maze Test (for cognitive function). As described by Kraeuter et al. in 2019 with slight modification [11]. The mouse was allowed to explore the three arms of the Y maze apparatus A, B, and C for 300 seconds prior to designated treatments as trial. Repeat this procedure 60 minutes after oral treatment of the individual mouse in all groups. The observation of the alternate arms visit was recorded as learning and memory traits.

e) Feeding behaviour. As described by Ellacott et al., in 2010 with slight modification [12]. Feeding pattern was determined by placing 60 g worth of feed and 250 ml in each group (n=6) daily and consumption rate was determined by measurements for 14 days along the designated oral treatment in each group. At the end of the 14 days treatments, the daily consumption rate was computed against each group.

f) Sleeping time. As described by Noguchi, & Kawai, in 1996 with slight modification [13]. Sleep time was determined using the phenobarbitone induced sleep approach by pre-treating each mouse with 20 mg/kg i.p and then followed by the designated treatments. Onset and duration of sleep was determined per mouse in all groups. The observation and records were noted for computation of results.

Results

Open Field Test: (Table 1)

Elevated plus maze: (Table 2)

Forced Swim Test: (Table 3)

Feeding Behaviour: (Table 4a-Table 4g), (Table 5 & 6)

Table 1: motor coordination evaluation Using the OF Test.

Group	Treatment	Dose (mg/kg)	Number of Squares Crossed (n)	% Dyskinesia	Remarks
1	Normal saline (NS)	0.2	93.3±3.3	0#	Normal movement
2	Tetrahydrocarnabinoid (THC)	500	75.3±1.1	19.3*	Mild movement disorder
3	Parsonsia straminea Extract (PSE)	1000	200.3±2.6	-114.4	No dyskinesia
4	PSE + THC	1000+500	2.7±0.5	97.1****	Gross dyskinesia present

Statistics done with graph pad prism 10.2, ANOVA with post hoc Dunnett's multiple comparisons test. Statistically significant **** ($p<0.0001$,) movement disorder when compared with the normal control (#); S=seconds.

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Table 2: Elevated Plus Maze.

Group	Treatment	Dose (mg/kg)	Closed Arm (s)	Open Arm (s)	Remark
1	Distilled Water (DW)	0.2	125.0 ± 38.2	145.0 ± 38.2#	Normal Control
2	Cannabis (THC)	500	161.0 ± 19.5	139.0 ± 19.6	Relatively Anxious
3	Parsonsia Straminea (PSE)	1000	117.3 ± 19.4	182.7 ± 19.4*	Not anxious
4	PSE+ THC	1000+500	142.0 ± 21.4	161.3 ± 21.8*	Not anxious

Table showed statistically significant *, p<0.02 antianxiety property when compared with #, the normal control.

Table 3: Forced Swim Test for Depression.

Group	Treatment	Dose (mg/kg)	Time of Swimming (S)	Time of Not Swimming (S)	% Depression Loss	Remark
1	Normal Saline (NS)	0.2	297.7±2.1	2.3±0.2	98.6	Normal Control
2	Tetrahydro cannabinoid (THC)	500	285.7±1.9	14.3±0.7	91.4****	Not depress
3	Parsonsia straminea (PSE)	1000	300.0±0.9	0.0±0.0	100****	Not depress
4	PSE + THC	1000+ 500	288.0±0.9	12.0±0.9	92.8****	Not depress

Statistics done with graph pad prism 10.2, ANOVA with post hoc Dunnett's multiple comparisons test. Statistically significant **** (p<0.0001) increase time spent in swimming and depression loss when compared with the normal control (#). S=seconds.

Table 4a: Day 1 Consumption Determination.

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	37.6 ± 0.0	30.1 ± 0.1
2.	THC	500	60.5	250.5	35.5 ± 0.0	20.1 ± 0.2**
3.	PSE	1000	60.5	250.5	53.7 ± 0.0**	30.1 ± 0.2
4.	PSE + THC	1000 + 500	60.5	250.5	54.4 ± 0.0**	35.1 ± 0.1*

Table showed statistically significant *, **, (p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE, Parsonsia straminea stem bark extract.

Table 4b: Day 2 Consumption Determination

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	59.4 ± 0.2	15.3± 0.3
2.	THC	500	60.5	250.5	36.3 ± 0.1	25.4 ± 0.4*
3.	PSE	1000	60.5	250.5	45.8 ± 0.1	25.5 ± 0.5*
4.	PSE + THC	1000 + 500	60.5	250.5	56.4 ± 0.2	30.4 ± 0.4**

Table showed statistically significant *, **(p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE, Parsonsia straminea stem bark extract.

Table 4c: Day3 Consumption Determination

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	53.7 ± 0.3	35.2 ± 0.2
2.	THC	500	60.5	250.5	38.7 ± 0.2*	20.3 ± 0.3*
3.	PSE	1000	60.5	250.5	52.6 ± 0.3	25.2 ± 0.2
4.	PSE + THC	1000 + 500	60.5	250.5	51.8 ± 0.0	25.3 ± 0.3*

Table showed statistically significant *, **(p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE, Parsonsia straminea stem bark extract.

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Table 4d: Day4 Consumption Determination

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	54.3 ± 0.1	40.2 ± 0.2
2.	THC	500	60.5	250.5	37.4 ± 0.4*	20.2 ± 0.2**
3.	PSE	1000	60.5	250.5	46.6 ± 0.1	25.2 ± 0.2**
4.	PSE + THC	1000 + 500	60.5	250.5	51.7 ± 0.1	35.2 ± 0.2

Table showed statistically significant *, **(p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE, Parsonsia straminea stem bark extract.

Table 4e: Day5 Consumption Determination

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	58.0 ± 0.1	30.4 ± 0.4
2.	THC	500	60.5	250.5	46.7 ± 0.2	25.3 ± 0.3
3.	PSE	1000	60.5	250.5	55.8 ± 0.2	20.4 ± 0.4
4.	PSE + THC	1000 + 500	60.5	250.5	50.6 ± 0.2	45.3 ± 0.3

Table showed statistically significant *, **(p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE, Parsonsia straminea stem bark extract.

Table 4f: Day6 Consumption Determination

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	59.7 ± 0.3	40.4 ± 0.4
2.	THC	500	60.5	250.5	39.7 ± 0.3 *	20.3 ± 0.3**
3.	PSE	1000	60.5	250.5	52.8 ± 0.2	25.4 ± 0.4**
4.	PSE + THC	1000 + 500	60.5	250.5	53.7 ± 0.1	40.2 ± 0.2

Table showed statistically significant *, **(p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE, Parsonsia straminea stem bark extract.

Table 4g: Day7 Consumption Determination.

Group Treatment		Dose (mg/kg)	Baseline Food (g)	Water (ml)	Food Consumption (g)	Water Consumption (ml)
1.	Distilled Water	0.2 ml	60.5	250.5	58.7 ± 0.2	35.3 ± 0.3
2.	THC	500	60.5	250.5	42.6 ± 0.3*	25.3 ± 0.3*
3.	PSE	1000	60.5	250.5	54.7 ± 0.1	25.2 ± 0.2*
4.	PSE + THC	1000 + 500	60.5	250.5	54.5 ± 0.3	25.4 ± 0.4*

Table showed statistically significant *, **(p<0.04, 0.01) increase and reduction in food and water consumption when compared with the normal control group (respectively). THC= tetrahydro cannabinoid (cannabis), PSE= Parsonsia straminea stem bark extract.

Table 5: Cognitive evaluation Using the Y-Maze Test.

Group	Treatment	Dose (mg/kg)	% Alternation	Remarks
1	Normal saline (NS)	0.2	74#	Normal Cognition
2	Tetrahydrocarnabinoid (THC)	500	33**	Impaired Cognition
3	Parsonsia straminea Extract (PSE)	1000	73	Optimized Cognition
4	PSE + THC	1000+500	51	Depressed Cognition

Statistics done with graph pad prism 10.2, ANOVA with post hoc Dunnett's multiple comparisons test. Statistically significant ** (p<0.01,) impaired cognition when compared with the normal control (#).

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Table 6: Sleep Time Evaluation Using Phenobarb Induced Sleep Test.

Group	Treatment	Dose (mg/kg)	Sleep Onset (s)	Sleep Duration (s)	Remarks
1	Normal saline (NS)	0.2	20	>300	Normal Control
2	Tetrahydrocarnabinoid (THC)	500	7***	>300	Improved sleep
3	Parsonsia straminea Extract (PSE)	1000	>300	-	No sleep
4	PSE + THC	1000+500	173	142	Impaired sleep

Statistics done with graph pad prism 10.2, ANOVA with post hoc Dunnett's multiple comparisons test. Statistically significant *** ($p<0.001$,) sleep onset when compared with the normal control (#).

Discussion

Cannabis (*Cannabis sativa L.*), widely consumed for both recreational and medicinal purposes, contains Δ9-tetrahydrocannabinol (THC), a psychoactive compound that interacts with the endocannabinoid system [14]. Chronic cannabis use has been associated with various neuropsychiatric disturbances, including cognitive deficits, anxiety, depression, and psychotic symptoms [15,16]. Animal models have demonstrated that prolonged exposure to THC can lead to oxidative stress, neuroinflammation, and neurotransmitter imbalances, particularly involving dopamine, serotonin, and GABAergic systems [17,18]. These neurochemical disruptions contribute to mood disorders, impaired cognition, and altered reward-processing behaviours. Parsonsiastraminea (R.Br.) F. Muell., a climbing vine from the *Apocynaceae* family, has been traditionally used in ethnomedicine for its anti-inflammatory, analgesic, and neuroprotective properties. Bioactive compounds such as alkaloids, flavonoids, tannins, and saponins found in *P.straminea* are believed to exhibit antioxidant and neuroprotective effects [19]. Previous studies done by Li et al in 2020 have indicated that extracts from Parsonsia species may modulate dopaminergic and serotonergic pathways, thus potentially counteracting the neurochemical imbalances induced by cannabis exposure [20]. Also it has been shown that chronic cannabis exposure leads to increased oxidative stress and neuroinflammation, key factors in neurodegeneration and psychiatric disorders [21].

THC exposure has been shown to upregulate pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and inducible nitric oxide synthase (iNOS) in the brain, leading to neuronal damage [22]. Antioxidant and anti-inflammatory agents have been suggested as potential therapeutic interventions to ameliorate cannabis-induced neurotoxicity. The study carried out in 2022 by Chandra and colleagues indicates that phytochemicals in *P.straminea* have demonstrated free radical scavenging activity, reducing lipid peroxidation and enhancing antioxidant enzyme levels such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) [23]. These mechanisms suggest that *P.straminea* may mitigate the oxidative damage and neuroinflammation associated with cannabis-induced behavioural impairments. Cannabis-induced behavioural disorders are linked to disruptions in neurotransmitter systems, particularly the dopaminergic, serotonergic, and glutamatergic pathways [24]. THC exposure alters dopamine receptor signaling in the prefrontal cortex and hippocampus, leading to deficits in working memory, decision-making, and emotional regulation [25]. An in vivo study by Wang et al, have suggested that *P.straminea* extracts may modulate neurotransmitter levels by enhancing serotonin and dopamine availability while reducing glutamate excitotoxicity [26].

The plant's alkaloid components have been shown to exert anxiolytic and antidepressant-like effects, similar to selective serotonin reuptake inhibitors (SSRIs) and dopamine agonists [20]. These findings support the hypothesis that *P.straminea* extract could ameliorate cannabis-induced anxiety, depression, and cognitive deficits in murine models. Histopathological analyses of THC-exposed brain tissues reveal neuronal shrinkage, gliosis, and synaptic loss, particularly in the hippocampus and prefrontal cortex [27]. It has been shown that neuroprotective agents with anti-apoptotic properties are of great interest in preventing cannabis-induced neuronal damage. Preliminary evidence suggests that *P.straminea* extract may promote neurogenesis and synaptic plasticity, protecting against THC-induced neuronal degeneration [28]. Flavonoids present in the extract may inhibit caspase-3 activation, reducing apoptosis in hippocampal neurons [20]. Furthermore, the plant's anti-inflammatory properties might suppress microglial activation, thereby limiting neuroinflammatory responses [23]. The findings of this study align with existing literature on the behavioral effects of cannabis while introducing novel insights into the potential ameliorative role of *P.straminea*. Previous research has consistently shown that cannabis can induce mild motor impairments and heightened anxiety due to its interaction with the endocannabinoid system, particularly through CB1 receptor activation, which modulates motor coordination and emotional regulation [14].

The absence of movement disorders and anxiety in the *P. straminea* group suggests a possible neuroprotective or anxiolytic property, which is in line with studies highlighting the role of natural plant extracts in counteracting cannabis-induced behavioral alterations [29]. Cognitive impairment is a well-documented consequence of cannabis use, primarily attributed to the disruption of hippocampal function [30]. Interestingly, *P.straminea* optimized cognition in this study, indicating potential cognitive-enhancing effects, similar to findings in botanical research where certain plant-derived compounds improve

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memory and learning deficits [31]. Additionally, cannabis-induced alterations in feeding patterns, such as reduced food intake and increased water consumption, have been previously observed and linked to CB1 receptor modulation [32]. However, *P. straminea* did not exhibit significant effects in this domain, suggesting its influence may be more prominent in cognitive and emotional regulation rather than metabolic changes. Furthermore, cannabis increased sleep duration, consistent with previous studies showing its sedative effects mediated through CB1 activation in sleep-regulating brain regions [33]. In contrast, *P.straminea* did not induce sleep within the evaluation period, indicating it does not share cannabis's sedative properties. Neither substance induced depressive behaviors, aligning with literature suggesting that acute cannabis exposure does not always produce depressive symptoms [34].

Conclusion

Overall, these findings suggest that *P. straminea* may mitigate several adverse behavioural effects of cannabis, particularly in cognition and anxiety. Further studies are needed to elucidate its mechanisms and potential therapeutic applications in cannabis-induced neurobehavioral alterations.

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