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EVALUATION OF ROTENONE INDUCE BEHAVIORAL DEFICITS, MEMORY AND COGNITIVE IMPAIRMENT RATS MODEL OF PARKINSON'S DISEASE

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Abstract: Rotenone is a toxic plant chemical that is used commonly in pesticides. It is highly lipophilic in nature, so it can readily penetrate the blood-brain barrier and build up inside the brain. According to previous studies administration of rotenone produced clear behavior deficits. Repetitive administration of low to moderate doses of rotenone induced neurotoxicity which is characterized by behavioral and motor abnormalities which are commonly observed in patients with Parkinson's disease. These abnormalities are due to increased generation of reactive oxygen species. ROS produced oxidative damage cause degeneration of nigrostriatal pathway, where dopaminergic neurons are widely distributed. Degeneration of this pathway causes loss of dopaminergic neurons which are involved in the development of neurodegenerative disease such as Parkinson's. In present study we found that repetitive administration of 10mg/ml/kg/day dose of rotenone for 14 days decreased food intake and bodyweight of rats. Locomotor activity in home cage was also found to be decrease after 14th day of rotenone administration. In open field activity latency time was increased and number of square crossed decreased. These changes were found after 14th day of administration. Hence rotenone administration reduced bodyweight and all behavioral parameters and produced anxiety and depression like symptoms. We may conclude from all of the current study's results, as well as from earlier research, that repeated administration of rotenone causes behavioral abnormalities and locomotor impairment, as well as symptoms like those of PD.

Key words: Parkinson's disease, Toxicity, Rotenone, Neuropharmacological deficits

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INTRODUCTION

Parkinsonism is a neuropathological condition that results in severe motor symptoms as bradykinesia, stiffness, uncontrollable tremors, and postural instability. The primary pathogenic feature of parkinsonism is the loss of dopamine at higher level (lotharius & Brundin, 2002). After Alzheimer's disease, PD is the worldwide neurological illness that accumulate 0.5-1% those aged 65 to 69 and 1-3% of People aged 80 and up (Nussbaum and Ellis, 2003). PD eventually leads to death that impacts an estimated 1–3% of people worldwide who are over 60 years old (Ball *et al*., 2019). PD is characterized by the presence of Lewy bodies, which are intracellular inclusions made up of aggregated α-synuclein and associated Lewy neurites. These proteinaceous inclusions are found within neuronal cell bodies and processes and are considered a defining feature of the disease's pathology (Eriksen *et al.,* (2005). As a result, In PD, like in other neurodegenerative illnesses, cognitive impairment indicates the common involvement of neurodegeneration as well as PD-specific damage in certain advanced cognitive brain regions, which is followed by well-defined clinical manifestations (Yang *et al*., 2016). The causes of late-onset idiopathic PD are unknown, but it is thought to be the result of inheretant and environment impairments and their interaction as the brain ages (Chena, and Beate, 2018). Rotenone is a highly lipophilic insecticide that easily penetrates in the blood-brain barrier and enters neurons, where it aggregates within mitochondria and suppresses complex I of the electron transport chain (NADH: ubiquinone oxidoreductase). Although supression of complex I lead to reduced ATP formation, ATP depletion alone is not considered the primary cause of rotenone-induced neurotoxicity. Instead, toxicity is largely attributed to the extravagant formation of reactive oxygen species (ROS) resulting from glutathione depletion, which promotes oxidative stress. Oxidative damage, evidenced by increased protein carbonyl formation, has been observed in the midbrain, olfactory bulb, striatum, and cortex of rotenone-treated rats regions similarly affected in post-mortem Parkinson's disease (PD) brains (Duty and Jenner, 2011).

Epidemiological studies showed connection of chronic rotenone exposure to an increased cause of Parkinson's disease in humans. In experimental models, rotenone induces neuropathological and behavioral phenotypes in rats that closely resemble those of PD. At low doses, both in vitro and in vivo studies demonstrate that rotenone disrupts multiple pathogenic pathways implicated in PD, including altered calcium signaling, oxidative stress, mitochondrial dysfunction, apoptosis, proteasomal impairment, nigral iron accumulation, and loss of tyrosine hydroxylase. Additionally, rotenone promotes the formation of fibrillar cytoplasmic inclusions containing ubiquitin and α-synuclein. Consistent with these pathological changes, rotenone-treated animals exhibit motor and behavioral abnormalities characteristic of Parkinson's disease, such as muscular rigidity (catalepsy), bradykinesia, postural instability, gait disturbances, and sleep impairments (Wrangel *et al*., 2015).

The aim of this reseach was to determine the effectiveness of rotenone which induced Parkinsonism in Albino Wister rodents. Rotenone induced symptoms similar to Parkinson's disease and had an impact on movement and food intake. Rotenone also induced some stress and anxiety in the animal which change its behavior. These parameters and behavioral changing helps to study the Parkinson's disease induced by rotenone which can be treat with the administration of Levodopa and some novel drugs like caffeic Acid.

1.

MATERIALS AND METHODS

2.

Research Animals

Twenty-four (24) male Albino Wister rats weighed between 190 and 220 grams were acquired from Dow University in Karachi. A comprehensive handbook for the care and management of laboratory animals (Institute of Laboratory Animals Resource of Life Science, US National Research Council, 1996) was used to ensure that experiments were conducted in a well-organized and stringent manner. All of the rats were housed in 12-hour light and dark settings, at a maintained temperature of 22±2° C, for at least three days prior to the experiment. They were also given full access to standard rodent food and use distilled tap water for habituation.

Drug and Chemicals

Each of chemicals used in the behavioral analysis research of rats were obtained from Sigma chemicals (USA), BDH chemicals pool (England), Merck business, or Research Biochemical (RBI, USA) and was verified by particular action of drug and different parameters.

Experimental Protocol

Twenty-four rodents were classified into two equal groups in this study: Controls and Tests. Twelve rodents are in each of these groups. The rats in the control group administered 0.9 % normal saline, whereas the rats in the test group were given Rotenone (10mg/ml/kg) till 14 days. The next day of the 1st, 7th, and 14th days of administration, all behavioral parameters were determined.

Behavioral Assessment

Food Intake

A weighted portion of a recently created standard laboratory meal with 30%fat, 30% protein and 40% carbs was given to the rats. Food consumption was measured the day after the first day of therapy and then on a regular basis by measuring the quantity of food remained in the hoopers in each cage.

Growth Rate (Body Weight)

To determine whether a medication or meal had an impact on growth, body weights were examined. Prior to the experiment initiate, on the first day following an hour-long neurotoxic dose, and then on a regular basis to track changes

Open Field Activity

Open field test is typically utilized to assess general exploratory and locomotor activity in rodents when exposed to a novel environment. The apparatus consists of opaque plastic walls measuring 42 cm in height, enclosing a square arena of 76 × 76 cm. The walls are designed to prevent the animals from escaping. The floor of the apparatus is divided into 25 equal-sized square compartments to facilitate the assessment of locomotor activity. The arena is considered a novel environment for the experimental animals. Each rat is kept in the central square of the arena, and the following parameters are recorded during a 5-minute observation period:

Latency time: The time taken by the rat to leave the central square after being placed in the arena.

Number of crossings: The number of squares the rat crosses with all 4 paws**.**

Pole Test

The pole test evaluates an animal's agility and could be used to gauge bradykinesia. The home cage was filled with a vertical, rough-surfaced wooden pole that measured 50 cm in height and 1 cm in diameter. After positioning their head atop the pole, the rat orients themselves downward and descends the pole back into their cage. Animals underwent five trials on test day, and the amount of time they took to orient downward (u-turn) and descend was recorded. The default value was used as the maximum level of impairment if the animal fell, slipped, or was unable to turn completely downward (Fleming *et al*., 2004).

Beam Walking Test

The inverted screen test was employed to evaluate the neuromuscular strength in the experimental rats. Each rat was placed on a horizontal wire grid positioned 20 cm above the floor. The animal was allowed to firmly grasp the grid with all four paws, after which the screen was carefully inverted to an upside-down position (Kondziella, 1964). The duration for which the rat remained suspended on the inverted grid before falling was recorded, with a maximum observation period of 30 seconds. The hanging latency was expressed in seconds.

Statistical Analysis

Two Way ANOVA (Repeated measured design) data of drug administration of control and test rats were studied. The Analysis program utilized was SPSS (version 17).

RESULTS

Impact of repeated rotenone administration on food intake in rats over a 14-day period was shown in Fig. 1. Food intake was assessed on the day following the first drug administration and subsequently monitored at weekly intervals. Two-way ANOVA with repeated measures revealed significant effects of rotenone treatment ($F = 143.12$, $df = 1,21$, $p < 0.01$), days of treatment ($F = 172.45$, $df = 1,21$, $p < 0.01$), and the relation among the treatment and days ($F = 87.31$, $df = 1,21$, $p < 0.01$). Post hoc analysis using the Newman–Keuls test demonstrated that rotenone consumption significantly reduced food intake in rats following both single and repeated administration compared with saline-treated animals. A significant reduction was observed on day 7 ($p < 0.05$) and day 14 ($p < 0.01$) of treatment. Furthermore, when compared with the food intake recorded on the first day of administration, rotenone-treated rats exhibited a further decline in food intake over the treatment period, with a significant reduction observed on day 14 ($p < 0.01$).

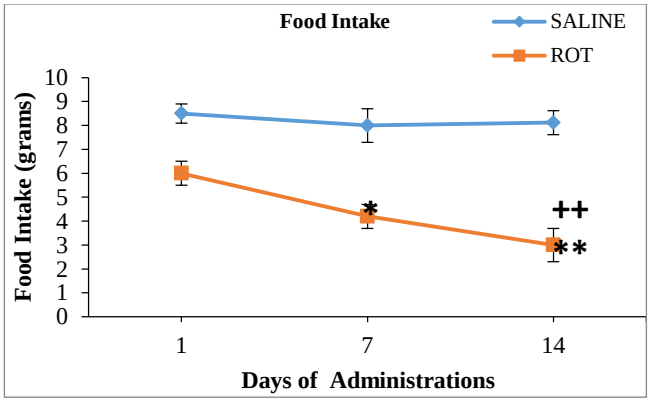


Fig 1: Effects of Rotenone on Food Intake of rats for 14 days
Data as analyzed 24 hrs after 1st, 7th and 14th day of administrations. *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration.

Figure 2 showed the effect of continuous Rotenone dosage on the food ingestion of rats over a 14-day period, with monitoring occurring the day after the initial drug dose and then weekly thereafter. Analysis of the data using two-way ANOVA (a repeated measures design) revealed a significant effect of Rotenone ($F=75.23$, $df=1, 21$, $p<0.01$) and a significant impact of periodic monitoring ($F=58.12$, $df=1, 21$, $p<0.01$), while the interaction effect among the drug and the duration ($F=2.54$, $df=1, 21$) was not significant. A post hoc analysis via the Newman-Keuls test indicated that the dosage of Rotenone led to a reduction in growth rate both acutely and with repeated dosing in rats when compared to those administered saline. A significant decrease was observed after the 7th day ($p<0.05$) and the 14th day ($p<0.01$) of administration. This was in contrast to the rats that received similar treatments.

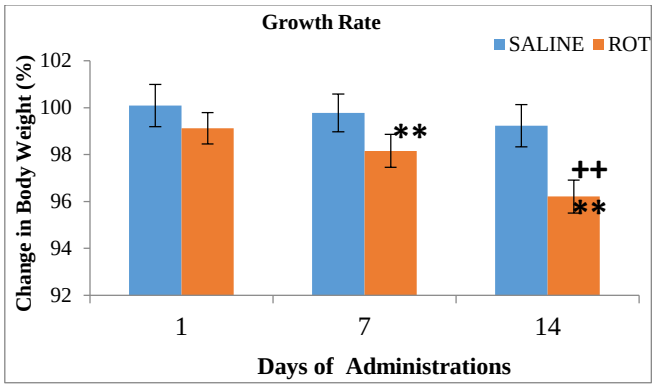


Fig 2: Effects of Rotenone on growth rate of rats for 14 days.
Data as analyzed 24 hrs after 1st, 7th and 14th day of administrations. *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration.

Figure 3 is presented the impact of continuous Rotenone administration on locomotor activity in a novel open-field environment (measured as latency time) over a 14-day period. A two-way repeated-measures ANOVA demonstrated substantial major impact for both days (F 49.12,$df=1,21$, $p < 0.01$) and Rotenone treatment ($F=45.91$, $df=1,21$, $p < 0.01$), as well as a significant interaction between Rotenone treatment and days ($F= 2.88$, $df=1,2$, $p < 0.01$). Subsequent post hoc analysis using the Newman–Keuls test revealed that both acute and chronic Rotenone dosage noticeably reduced latency time in the open field compared to vehicle-treated (saline) controls. Specifically, this reduction was statistically significant on day 7 ($p < 0.05$) and day 14 ($p < 0.01$). Furthermore, intra-group comparisons showed that Rotenone-treated rats exhibited a progressive decrease in latency time across the study duration, reaching a significant reduction on day 14 relative to day 1 baseline values ($p < 0.01$).

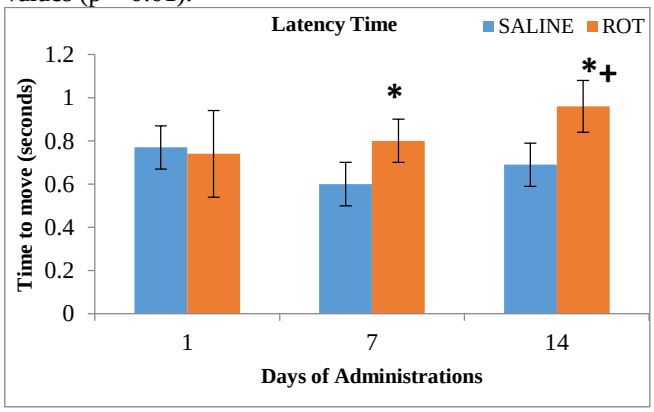


Fig 3: Effects of Rotenone on open field (latency) of rats for 14 days
Data as analyzed 24 hrs after 1st, 7th and 14th day of administrations *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration.

Figure 4 is showing the impact of continuous Rotenone administration on locomotor activity in a novel open-field environment (measured as the number of squares crossed) over 14 days. Two-way repeated-measures ANOVA shown noticeable major impact for both days ($F=87.46$, $df=1,21$, $p<0.01$) and Rotenone treatment ($F=121.68$, $df=1,21$, $p<0.01$) , as well as a significant interaction between Rotenone treatment and days ($F=81.97$, $df=1,21$. $p<0.01$). Post hoc analysis using the Newman–Keuls test demonstrated that both acute (day 1) and repeated Rotenone administration significantly reduced open-field activity compared to vehicle-treated (saline) controls, with significant decreases observed at week 1 ($p < 0.01$) and week 2 ($p < 0.01$). Furthermore, intra-group analysis revealed that Rotenone-treated rats exhibited a progressive decrease in grid crossings over time, reaching a statistically significant reduction on day 14 relative to their initial day 1 baseline values ($p < 0.01$). significant decreases observed at week 1 ($p < 0.01$) and week 2 ($p < 0.01$). Furthermore, intra-group analysis revealed that Rotenone-treated rats exhibited a progressive decrease in grid crossings over time, reaching a statistically significant reduction on day 14 relative to their initial day 1 baseline values ($p < 0.01$).

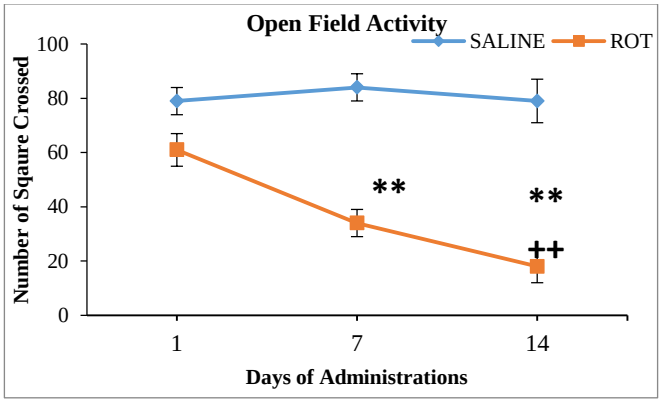


Fig 4: Effects of Rotenone on open field (boxes crossed) of rats for 14 days

Data as analyzed 24 hrs after 1st , 7th and 14th day of administrations. *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration.

Figure 5 presents the effect of continuous rotenone administration on performance in the pole test (time) in rats over 14 days. The animals were assessed on the day following the first drug administration and subsequently monitored weekly. Two-way ANOVA with repeated measures revealed a significant impact of days ($F = 65.28$, $df = 1, 21$, $p < 0.01$) and a significant impact of rotenone treatment ($F = 39.21$, $df = 1, 21$, $p < 0.01$). However, the interaction between rotenone treatment and days was not significant ($F = 1.51$, $df = 1, 2$). Post hoc analysis using the Newman–Keuls test showed that rotenone dosage noticeable altered pole-test execution compared with saline-treated rats. A significant reduction in performance was observed after the 7th ($p < 0.01$) and 14th ($p < 0.01$) days of rotenone administration.

When compared with the corresponding measurements obtained after the first administration, the time recorded in the pole test was significantly reduced in rotenone-treated rats. This reduction was significant on the 7th day ($p < 0.05$) and further increased by the 14th day ($p < 0.01$) of administration.

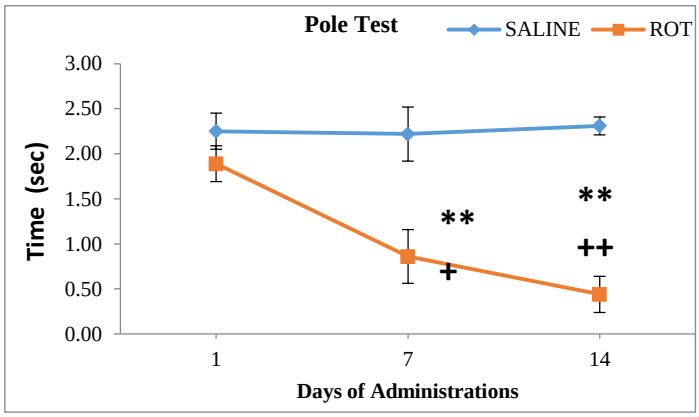


Figure 5: Effects of Rotenone on Pole test of rats for 14 days.

Data as analyzed 24 hrs after 1st , 7th and 14th day of administrations. *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration.

Figure 6 demonstrates the effect of continuous rotenone administration on performance in the beam-walking test (time) in rats over a period of 14 days. The animals were assessed on the day following the first drug administration and then monitored on a weekly basis. Two-way ANOVA with repeated measures revealed a significant effect of days ($F = 91.48$, $df = 1, 21$, $p < 0.01$), a significant impact of rotenone treatment ($F = 53.41$, $df = 1, 21$, $p < 0.01$), and a significant relation among rotenone treatment and days ($F = 42.11$, $df = 1, 21$, $p < 0.01$). Post hoc analysis using the Newman–Keuls test revealed that continuous rotenone dosage significantly altered performance in the beam-walking test compared with saline-treated rats. A significant decrease in the recorded activity was observed after the 7th and 14th days of rotenone dose ($p < 0.01$). Furthermore, when compared with the corresponding measurements obtained after the first administration, rotenone-treated rats showed a significant reduction in activity over time. This reduction was evident after the 7th day and became more pronounced by the 14th day of administration ($p < 0.01$).

Figure 7 illustrates the effect of continuous rotenone administration on performance in the inverted screen test (hanging time) in rats over a period of 14 days. The animals were assessed on the day following the first drug administration and subsequently monitored weekly. Two-way ANOVA with repeated measures demonstrated a significant impact of days ($F = 77.79$, $df = 1, 21$, $p < 0.01$), a significant impact of rotenone treatment ($F = 67.32$, $df = 1, 21$, $p < 0.01$), and a significant interaction between rotenone treatment and days ($F = 88.49$, $df = 1, 2$, $p < 0.01$). Post hoc analysis using the Newman–Keuls test revealed that

continuous rotenone dosage significantly reduced performance in the inverted screen test, as indicated by decreased hanging time, compared with saline-treated rats. A main reduction ($p < 0.01$) was observed after the 7th and 14th days of rotenone dose. Furthermore, compared with the corresponding values recorded after the first administration, rotenone-treated rats revealed a main reduction in hanging latency over the course of treatment. This reduction was significant after the 7th and 14th days of administration ($p < 0.01$).

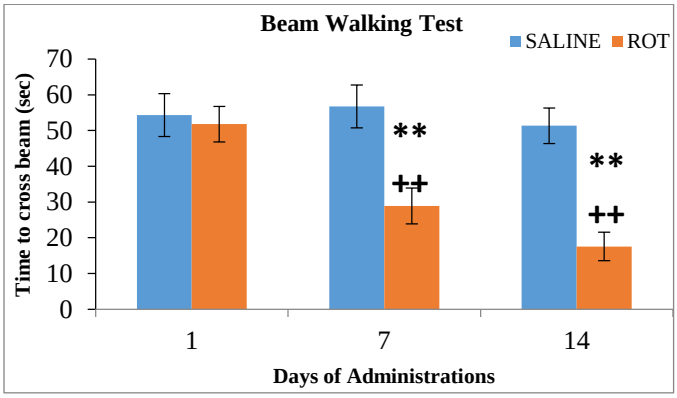


Fig 6. Effects of Rotenone on Beam Walking Test of rats for 14 days.

Data as analyzed 24 hrs after 1st , 7th and 14th day of administrations. *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration

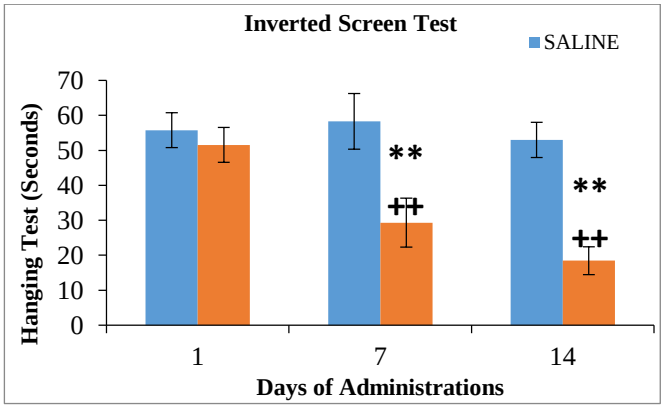


Fig 7. Effects of Rotenone on Inverted Screen Test of rats for 14 days.

Data as analyzed 24 hrs after 1st , 7th and 14th day of administrations. *p<0.05, **p<0.01 from control administrated animals; +p<0.05, ++p<0.01 from same group control or Rotenone treated rodents of 1st administration

DISCUSSION

The present study evaluated the behavioral, motor, and cognitive impacts of acute and repeated Rotenone administration in rats using established neurobehavioral paradigms, including body weight growth rate, food intake, open-field locomotion, and the pole test. Because Rotenone is highly lipophilic, it readily penetrates in the blood–brain barrier (BBB) and bypasses the dopamine transporter (DAT) for neuronal entry (Sharma *et al*., 2016). Intracellularly, Rotenone accumulates within subcellular organelles specifically inhibiting Complex I of the mitochondrial electron transport chain, thus disrupting cellular respiration and elevating reactive oxygen species generations and following oxidative stress (Nicholls, 2008; Nisticò *et al.*, 2011). In this context, the Rotenone-induced rat model provided a reliable model to explore progressive behavioral alterations relevant to Parkinson's disease (PD) pathogenesis.

Repeated Rotenone administration over 14 days led to a significant decline in growth rate and a concurrent reduction in daily food intake compared to saline-treated controls. This hypophagic response mirrors non-motor gastrointestinal and metabolic manifestations common in PD, such as early satiety, constipation, and weight loss (Pfeiffer, 2003). Furthermore, 14-day Rotenone treatment significantly impaired motor performance, as evidenced by reduced locomotor activity (hypokinesia), diminished rotarod retention times, weakened grip strength, and compromised motor coordination. In the open-field apparatus which measures both initiation of movement (latency to move) and exploratory drive (grid-crossing frequency). Rotenone-treated animals exhibited marked hypoactivity across multiple indices, including reduced square crossings, grooming, rearing, and sitting behavior. Collectively, these neurobehavioral impairments substantiate that chronic Rotenone toxicity effectively replicates core motor and systemic features of PD pathogenesis. The test group repeatedly administered with Rotenone showed decreased latency time with reference to its locomotive impairment while control group administered with normal saline showed otherwise. Also this decrease in latency time is quite significant after the 14th day. On the other hand the number of squares crossed by the Rotenone administered rats was decreased significantly after 14th day, showing locomotive impairment and rigidity in the animal. All the above mentioned results revealed that acute as well as repeated administration of Rotenone (10 mg/ml/kg) induce behavioral deficits in animal model of rat as compared to control group given with normal saline (0.9% NaCl). These findings shows that, in rats, rotenone causes loss of dopamine producing neuron which reduce the dopamine level and produce symptoms similar to Parkinson's disease including some changes in behavior and locomotion. The different parameters also revealed that the rotenone also induced some anxiety in rats as well as their growth rate and food intake was also affected. This behavioral decline could also be characterized by the locomotor impairment and motor dysfunction as seen in previous studied Rotenone induced Parkinson's model of rats. Overall, these data and previous study showed that Rotenone cause behavioral deficits and locomotor impairment upon repeated administration and also produce symptoms similar to Parkinson's disease.

CONCLUSION

This study shows that rotenone can successfully reduce behavioral defects and treat depressive-like behavior in rats. These results demonstrate that rotenone results in the death of dopamine-producing neurons, which lowers dopamine levels and induces symptoms resembling those of Parkinson's disease, including as behavioral and movement abnormalities. The various metrics also showed that the rats' growth rate, food intake, and anxiety levels were all impacted by the rotenone. The locomotor impairment and motor dysfunction observed in the previously published Rotenone-induced Parkinson's model of rats may also be indicative of this behavioral deterioration. Overall, these findings and earlier research demonstrated that repeated injection of rotenone results in behavioral abnormalities, locomotor impairment, and symptoms like those of Parkinson's disease.

Conflict of interest

Authors declare no conflict of interest.

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