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Behavioral and Neuronal Alterations Following Oral Naphthalene Exposure in Rats

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Abstract: Naphthalene (NP), a widely used polycyclic aromatic hydrocarbon, is present in various commercial products and environmental pollutants. Despite its established toxicity, its impact on neuronal integrity and behavior remains relatively underexplored. This study investigates oral NP-induced behavioral and neuronal alterations in Sprague Dawley rats. Twenty-five animals were divided into five groups and oral NP was administered at varying doses (200 mg/kg and 400 mg/kg) for 28 days, with post-treatment evaluations up to 42 days. Behavioral assessments using the open field test revealed initial hyperactivity, followed by a progressive decline in locomotion and increased anxiety-related behavior in high-dose groups. Cresyl violet staining of the basolateral amygdala showed significant neurodegeneration, with pyramidal neuronal damage more pronounced in high-dose groups. Statistical analysis was conducted using one-way ANOVA, and post-hoc Duncan's test was applied to confirm a dose-dependent decrease in neuronal viability ($p < 0.05$). Post-treatment observations indicated partial behavioral recovery but no significant reversal of neuronal damage. The findings suggest that oral NP exposure induces anxiety-associated behavioral changes and neurotoxicity in the amygdala, potentially affecting emotional regulation. Further investigation is required to assess the long-term effects of oral NP exposure on brain function.

Keywords: Napthalene; toxicity; neuronal alteration; behavioral alteration; neurotoxicity

1. Introduction

Naphthalene (NP) is a polycyclic aromatic hydrocarbon, characterized as a white solid that readily sublimates into gas. It is primarily obtained from coal tar. NP is also present in various types of smoke, including cigarette smoke, forest fire emissions, and automobile exhaust (Angu Bala Ganesh et al; 2024).

NP is widely used in the manufacture of commercial goods, most notably as the active component in mothballs (naphthalene balls) or crystals that release toxic vapours to repel insects and animals. Naphthalene was first documented as a pesticide in the United States in 1948 (CDC, 2005). It also produces toilet deodorant blocks, pesticides, leather tanning agents, dyes, resins, and PVC polymers (Baker et al., 2011; Preuss et al., 2003).

NP can be introduced and absorbed through four distinct pathways: orally, by ingesting contaminated water with NP; dermally, by contact with fabrics treated with NP; via inhalation, through low concentrations present in indoor and outdoor air, including emissions from factories during the production and handling of commercial goods, as well as smoke from tobacco, wood, and coal; and through ocular exposure, either from NP vapours or by touching the eyes with contaminated hands (Mugweru et al., 2020; Wang et al., 2020). The absorbed NP metabolites are distributed via the bloodstream to many organs, particularly the heart, lungs, liver, kidneys, and spleen, and are ultimately eliminated through urine and faeces (Waidyanatha et al., 2020; Mugweru et al., 2020).

The basolateral nucleus of the amygdala (BLA) engages in two-way communication with brain regions that influence cognition, motivation, and stress responses, including the prefrontal cortex, hippocampus, nucleus accumbens, and hindbrain areas that activate norepinephrine-mediated stress responses. The hyperexcitability of BLA principal neurons is related to behavioral disorders characterized by excessive fear and anxiety (Etkin et al., 2004, Angu Bala Ganesh., 2024).

Many studies have been conducted and shown that NP toxicity produces histological changes in multiple organs of animal models, such as hyperplasia of hepatic bile ducts (Radoslaw Świercz & Maciej Stępnik, 2011), thickening of the inter-alveolar septa and hepatocellular necrosis (Fang Zhang et al., 2016), vacuolisation in Clara cells (Ching-Yu Lin et al; 2015).As a result of naphthalene-induced hemolysis, newborn neonates are at risk for

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permanent neurological impairment known as kernicterus. Convulsions and, in rare cases, death occur when brain cells absorb more bilirubin. Survivors often suffer from motor abnormalities and dementia. It has been reported that kernicterus occurred in eight of twenty-one Greek newborns who had hemolysis due to naphthalene exposure (Valaes et al., 1963).

More recent case reports have reaffirmed this association, particularly in infants with glucose-6-phosphate dehydrogenase (G6PD) deficiency, who are especially vulnerable to oxidative damage from naphthalene exposure through ingestion of naphthalene-containing mothballs (Bölükbaş et al., 2021; Koca, N., & Doğan, M., 2022).

Human case studies have documented neurological symptoms associated with naphthalene ingestion, including disorientation, altered sensorium, listlessness, lethargy, and vertigo. Individuals who ingested naphthalene exhibited severe symptoms such as convulsions, muscle fasciculations, reduced reactivity to painful stimuli, and coma prior to death. During autopsy, the brain exhibited edema, accompanied by histological signs of axonal disconnection and inflammation of the myelin sheaths (Sharma et al., 2024). Cerebral oedema, which was most likely caused by abrupt hemolysis, could be the cause of the neurologic symptoms observed after naphthalene exposure (Shaikh et al., 2023). However, previous research that did not focus on behavioral and neuronal changes due to NP toxicity was inconclusive. Hence, this study was conducted to analyse the effect of naphthalene on behavior and neuronal cell integrity.

2. Materials and methods

Chemicals: Naphthalene balls were obtained from Research Lab Fine Chemical Industries. All other chemicals were obtained from a local supplier of standard quality.

Animals: Male Sprague Dawley rats less than 9 weeks old were obtained from Invivo Biosciences, No. 23, Kodigehalli village, Eshwanthpur hobli, Bangalore North taluk, Bangalore. All animals were kept under 12-hour light and dark conditions. All experimental animals were randomly assigned to polypropylene cages and bedded with autoclaved paddy husk. The cages were covered with stainless-steel grid tops. Cages were changed on alternate days, and cage grilling occurred once a week. The animals were fed commercial rodent pelleted feed obtained from M/s. VRK Nutritional Solutions, Pune, and water ad libitum. The Institutional Animal Ethical Committee (IAEC) granted ethical permission via ethics certificate IAEC/60/SRIHER/674/2019. All experiments complied with IAEC and Sri Ramachandra Institute of Higher Education and Research procedures.

Test item preparation: Naphthalene balls were placed in a mortar, crushed, and ground into fine powder. The required test material was weighed, mixed with a small amount of vehicle, and formed into a fine pasty liquid using a pestle. This pasty liquid was transferred to a graduated measuring cylinder and adjusted to the desired volume by adding vehicle.

Induction of Naphthalene Toxicity: A total of 25 experimental animals were allowed five days of acclimatisation before being randomly assigned to one of five groups. The experimental period lasted 42 days (28 days for the main group and 14 days for post-treatment observation for satellite groups). The test item was administered daily as described below, with the dosage determined by body weight.

Dose Volume = (Body weight of the animal (g) × Dose) / (1000 × Concentration (mg/ml)). Control animals received the vehicle at a 5 ml/kg body weight dose. The test item was delivered orally through gavage.

The LD50 value, which represents the lethal dose that causes death in 50% of a set of test animals, is a crucial factor in toxicology studies. Naphthalene has an oral LD50 of over 2000 mg/kg/bw in rats. In this study, the LD50 value for naphthalene in rats was used to determine safe doses, with selected doses being 1/10 and 2/10 of the LD50 value. Table 1 presents the experimental design in this work.

Table 1. Experimental Design

S.No	Animal Groups	Treatment,	No	Dose and Duration
1	Group 1	(Control- Vehicle)	5	5ml corn oil/kg/for the period of 28 days
2	Group 2	Low Dose Naphthalene	5	200mg/kg/ for the period of 28 days
3	Group 3	High Dose Naphthalene	5	400mg/kg/day for 28 days
4	Group 4 (Satellite- Control Vehicle)	(Satellite Control- Vehicle)	5	5ml corn oil/kg/day for 28 days and 14 days post treatment observation period.
5	Group 5 (Satellite- High Dose)	(Satellite- High Dose Naphthalene)	5	400/mg/kg/ for the period of 28 days and 14 days of post treatment observation period.

2.1 Behavioural test

Open field test

Open field tests were conducted for all animals on days 0, 7, 14, 21, and 28 for the leading group and on days 35 and 42 for the satellite groups.

The open field apparatus consisted of a wooden box with dimensions of 60x60x60 cm and painted black except for the analysis area, which was equally divided into sixteen squares by drawing lines on the floor region, where the central analysis area included four squares. At the time of open field testing, the animals were placed in the right arena corner, and their movement around the analysis field was recorded. The experimental arena was cleaned with 70% alcohol after each group, and the following animal behaviour was analysed using the recorded video.

Locomotion measurement: Number of lines crossed by each animal during the given 5-minute interval.

Central latency: The time taken (in seconds) by an individual rat to enter the central square of the apparatus, defined as head entry and placement of two front paws, was measured in seconds.

After completion of 28 days, the rats in groups 1 to 3 were euthanised by deep anaesthesia with intraperitoneal injection of ketamine (50 mg/kg, b.wt) + xylazine (5 mg/kg, b.wt); death was confirmed via fixative perfusion with 10% neutral buffered

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formalin and the cessation of heartbeat. In groups 4 and 5 (satellite groups), the same procedure was followed for euthanasia after completion of 42 days.

2.2 Sample collection and Preparation

The excised rat brain tissues were immersed in ice-cold saline. The brain tissues intended for histological analysis were thoroughly washed with normal saline and then immersed in 10% formalin solution for one week. The brain samples were subsequently processed to reveal histological changes. Using a rotary microtome, tissue sections of 5 µm thickness were obtained, after which they were stained with Cresyl Violet to observe the neurodegenerative cells in the deep temporal region of the basolateral nucleus of the amygdaloid region.

Cresyl Violet Staining

The portions were deparaffinized and rehydrated in distilled water. The slides were incubated with a 0.1% cresyl violet stain for several minutes and washed with distilled water. The specimen was rapidly dehydrated in absolute alcohol, cleared in xylene, and mounted in synthetic resin. The cresyl violet-stained sections of the basolateral amygdala were examined using a 40x objective lens, and a random selection procedure quantified the neurons through the microscope.

Statistical Analysis

The open-field test results for central latency and locomotion were presented as mean ± SD. ANOVA was conducted for G1, G2, and G3, whereas paired t-tests were applied for the satellite groups (G4 and G5). Cresyl violet results were presented as mean ± SD with one-way ANOVA and post-hoc Duncan's test for group comparison. All analyses were statistically significant at $p < 0.05$.

3. Results

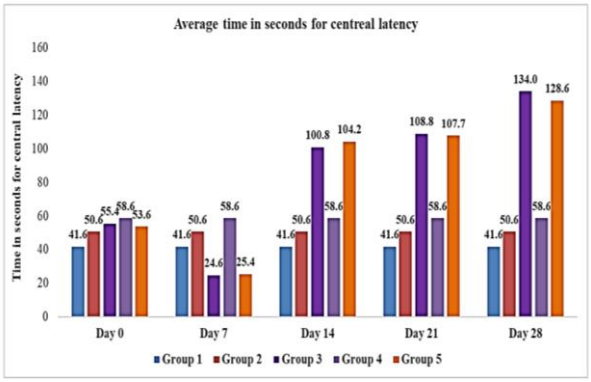

Figure 1. Behavioural changes in time in seconds for central latency in rats induced with naphthalene toxicity. Represented values are expressed as average (Mean) for five rats in each group. p<0.05* denotes significantly different compared to normal control rats.

3.1 Effect of Naphthalene on behaviour and locomotion

On the seventh day, central latency was shorter in G3 and G5 compared to G1, G2, and G4, and on the 14th, 21st, and 28th day, a gradual decrease in the central latency, i.e., delay in time to enter the central square, was noted in G3 and G5 (Figure 1).

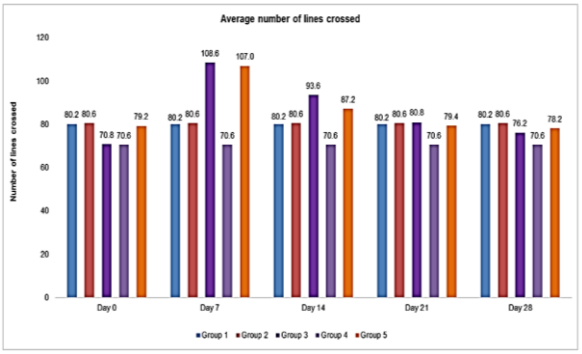

Figure 2. Behavioural changes in the number of lines crossed in rats induced by naphthalene toxicity. Represented values are expressed as average (Mean) for five rats in each group, and p<0.05* denotes significantly different compared to normal control rats.

It was found that, on day 7, there was an increase in the locomotor activity in G3 and G5 compared to G1, G2, and G4, and on the 14th, 21st, and 28th day, the mean number of lines crossed gradually decreased compared to day 7, which was noted in G3 and G5 (Figure 2).

Post-Treatment Observation: Central latency was shorter in both groups on the 42nd day than on the 35th day. G4 and G5 locomotor activity increased on the 42nd day compared to the 35th day. In the post-treatment observation period, no delayed withdrawal effects were noted in all the parameters of the open field test on G5.

3.2 Effect of Naphthalene in the Depth of the Temporal lobe in the basolateral amygdaloid region

The standard control group shows intact neuronal cell bodies with the typical morphological characteristics of pyramidal neuronal cells present in the depth of the temporal lobe in the basolateral amygdaloid region. At the same time, mild toxicity was observed in low-dose toxicity groups with subtle morphological abnormalities on the pyramidal cells. In the case of high-dose naphthalene toxicity, degenerated pyramidal cellular morphology with cell death was observed. The satellite control group was similar to the control group. Necrotic neurons were observed in the delayed toxicity group, similar to the naphthalene toxicity group (Figure 3).

Post-hoc Duncan's test for group comparison (pairwise comparison of groups) shows a mean difference in the total number of surviving neurons across all pairs. However, a statistically significant (p<0.05) mean difference was found between all pairs except for G1 and G4 and G3 and G5 of animals (Figure 4). It indicates that low, high, and satellite-high doses reduced the number of surviving neurons. In the post-treatment

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observation period, minor and delayed withdrawal effects were noted in the satellite-high doses.

4. Discussion

Naphthalene is well known for its cytotoxicity and has even been used as a therapeutic agent for its cytotoxic properties (Anwar et al., 2021). When naphthalene is administered as a drug,

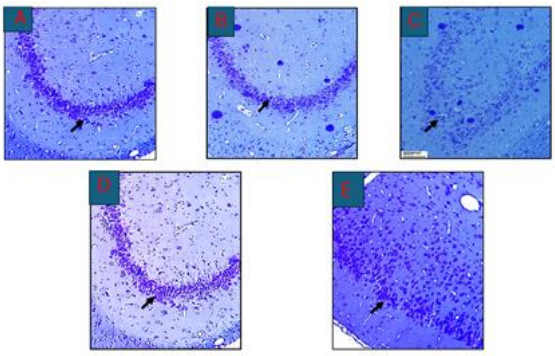

Figure 3. Cresyl violet staining analysis of naphthalene toxicityrats (magnification 40x). G1(A). G2(B), G3(C), G4 (D), G5 (E)

it converts into its reactive metabolites, like NP epoxides and NP-quinones. These active breakdown products are associated with cysteine residues of several intracellular proteins and cause toxicity. Primarily, NP oxides interact with the sulfhydryl group of cysteine and produce naphthoquinones (Jing et al., 2020).

Naphthalene, which is commonly used in households for pest control and deodorizing, has become an important yet problematic substance due to its widespread domestic use and subsequent emergence as a major environmental pollutant. This highlights the necessity for extensive toxicity testing in mammals (Pannu & Singla, 2020). Chlorinated forms of naphthalene enter edible products, especially animal food, which is considered an early exposure source in the general population. In human biological samples such as plasma, serum, milk, and adipose

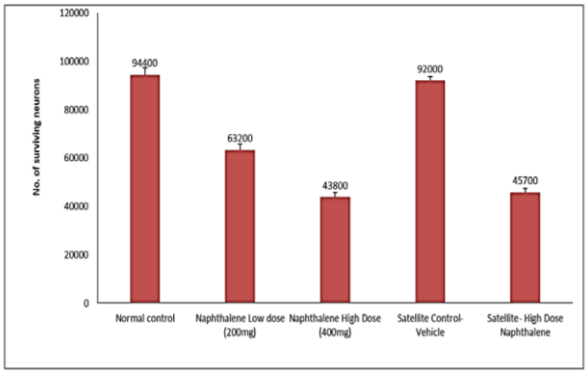

Figure 4. Quantification of surviving neurons across all groups using cresyl violet staining. Data are presented as Mean ± SD (n = 5 rats per group).

tissue, the chlorinated form of naphthalene has been detected (Jin et al., 2019; Li et al., 2021; Waheed et al., 2020).

The open field test is a well-established method for studying anxiety-like behavior and evaluating drug-related toxic effects on animal behavior (Zimcikova et al., 2017). This test can also be used to assess the levels of anxiety effects on movement activity under drug influence. On repeated exposure to the open field test, animals adapt to the open field area, and their movements tend to decrease (Choleris et al., 2001).

The total number of entries to the central square, the time spent in the central square, and the latency to enter the central square are indicators of exploratory behavior and anxiety (Zimcikova et al., 2017). Figure 2 illustrates the change in central latency in rats subjected to NP toxicity. It was found that, on day 7, central latency was shorter in G3 and G5 compared to G1, G2, and G4. Moreover, on days 14, 21, and 28, a gradual reduction in the central latency (in seconds), i.e., delay in time to enter the central square, was observed in the G3 and G5 groups.

On the seventh day of the test, the high-dose and high-dose satellite groups exhibited increased exploratory behavior and a quicker time to enter the central square. On days 14, 21, and 28, exploratory behavior decreased, and the time to enter the central square was prolonged. On days 35 and 42, the satellite high-dose group showed a slight increase in exploratory behavior.

To examine the neurotoxic effects, measures such as the number of line crossings and depressive and anxiety behaviors in animals were assessed using the open field test. Figure 3 shows the change in locomotor activity (average number of lines crossed) in rats induced with naphthalene toxicity. It was found that, on day 7, locomotor activity increased in G3 and G5 compared to G1, G2, and G4, and on days 14, 21, and 28, the mean number of lines crossed gradually decreased compared to day 7 in G3 and G5 groups. Prolonged NP exposure in animals and humans can cause pathological changes in brain function, resulting in maladaptive behavior (Lupien et al., 2018; Mineur et al., 2006).

These effects are demonstrated by studying animals' responses to stress stimuli in novel environments (Ramos & Mormède, 1998). Glutamatergic neurons and inhibitory interneurons are located in the BLA (Krabbe et al., 2018).

BLA encompasses a dynamic interaction between excitatory glutamatergic neurons and inhibitory interneurons, both essential for modulating emotional and behavioral responses. Naphthalene exposure, recognized for inducing oxidative stress and neuroinflammation, may disrupt the excitatory-inhibitory balance within the BLA. This disruption may contribute to the behavioral changes observed in this study, including symptoms of anxiety and cognitive impairments. Injury to glutamatergic neurons or disturbance of interneuronal inhibition may contribute to the abnormal brain signaling linked to naphthalene neurotoxicity (Moriceau et al., 2006). The principal neurons are pyramidal-like projection neurons with small dendrites that use glutamate as an excitatory neurotransmitter. In contrast, nonpyramidal neurons of the basolateral amygdala are spine-sparse interneurons that use the amino acid GABA as an inhibitory neurotransmitter. The majority (~80%) of neuronal cells are principal glutamatergic, and

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the remainder (~20%) are GABAergic inhibitory interneurons (Spampanato et al., 2011). Stress in any form affects pyramidal neurons in multiple ways, mainly altering the morphology of the dendritic tree, dendritic process length, and synapse spine density (Chocyk et al., 2013b; Muhammad et al., 2011; Monroy et al., 2010).

The deep temporal region of the basolateral part of the amygdala in naphthalene-treated animals exhibited intense neurodegeneration. These changes occurred due to naphthalene exposure, scattered with asymmetric neurons stained dark by cresyl violet. The degenerative cells appeared shrunken with distorted morphology and stained darker compared to normal cells.

The neuronal arrangement of the basolateral amygdala region appeared symmetrical in control rats. Normal and healthy cells were identified by spherical morphology with a visible nucleus. The low toxicity group showed pyramidal cells with minor abnormalities in morphology compared to the standard controls. The high naphthalene toxicity group demonstrated that more pyramidal cells of the basolateral amygdala exhibited degenerated morphology, indicating cell death. It was also observed that the pyramidal cells of the basolateral amygdala were replaced by abundant cells of unidentified origin, resembling inflammatory cells based on morphology. The satellite control group resembled the control group. Necrotic neurons were observed in the delayed toxicity group, similar to the naphthalene toxicity group. From the results obtained, extensive neurodegeneration occurred following treatment with naphthalene. These types of changes were studied three decades earlier, revealing that neuronal damage features condensed neurons scattered darkly in all brain regions (Sugimoto et al., 1990). That study also identifies three main characteristics for damaged neurons: uneven cellular outlines; increased chromatin density in both cytoplasm and nucleus; and profoundly and consistently stained nuclei. All these characteristics were observed in the neurons of naphthalene-treated basolateral amygdala region rats. The morphological abnormalities in pyramidal cells after a high dose of naphthalene confirm neurodegeneration.

5. Conclusion

The open field test revealed that the high dose and high dosage satellite groups exhibited increased anxiety-like behaviour on days 14, 21, and 28. In contrast, the high-dose satellite group showed a slight reduction in anxiety-like behaviour during post-treatment observation periods on days 35 and 42. There were no delayed withdrawal effects in any open-field test parameters for the high-dose and high-dosage satellite groups. Cresyl violet staining indicated that apoptosis and the number of surviving neurons decreased in the low, high, and satellite high dosage groups. The satellite high-dose group experienced no significant reversible or delayed withdrawal effects following treatment. This study concluded that naphthalene oral toxicity damages numerous amygdala basolateral nuclei cells, resulting in anxiety or mood changes. More studies are necessary to validate structural abnormalities associated with oral naphthalene intake and neurological disorders in other brain regions.

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