

Concurrent exposure to light and traffic noise pollution augments behavioral deficits in rats: Protective effect of curcumin

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ABSTRACT

The current study was aimed to explore the effect of light and traffic noise pollution exposure on behavioral and neurochemical deficits in adult male Wistar rats. We hypothesized that exposure to these pollutants alone or in combination may cause behavioral deficits in rats. Animals were allocated into different groups and were exposed to either varying light cycles (150 lx-5 lx, 14:10 h) or traffic noise with sound level 100 dBA for 6 h/day or both for 28 days. Concurrently, Curcumin (100 mg/kg; per oral; 28 days) was administered in varying light cycles and noise exposed animal groups to study the preventive effect. After the exposure time, the Morris water maze test, elevated plus maze test, novel object recognition test, locomotor activity test, and forced swimming test were conducted. The neurobehavioral results suggested that only combined exposure of light and noise pollution markedly impaired the learning as well as spatial and reference memory in rats. Furthermore, we found significant anxiety (less no. of open arm entries in EPM) and depressive-like behavior (increased immobility time) in light or/and traffic noise pollution exposed group animals in comparison to normal control groups. Curcumin treatment significantly prevented the behavioral anomalies in combined light and noise exposed rats. Furthermore, our results revealed augmentation of acetylcholinesterase, oxidative stress markers and interleukin (IL)-1 β in the hippocampal region of noise and light-exposed groups which were markedly ameliorated by curcumin. In conclusion, our results suggest that curcumin significantly modulates light and noise pollution exposed behavioral and neurochemical anomalies in rats.

Introduction

Pollution is one of the biggest challenges facing humankind, causing irreparable damage to the earth. The uninterrupted rise in industrialization and unsustainable development causes amplification of environmental pollutants, which in turn affects the physiological and mental health of the human [1]. Particularly, light at night and noise are well known influential and widespread sources of pollution that are encountered by people of urban cities in daily life [2]. Industries, traffic, and construction sites are the key source of noise pollution. On the other hand, light pollution occurs due to use of artificial light at night. Noise pollution acts a stressor particularly at intensity of 85–90 dBA or above by evoking the sympathetic nervous system and corticosterone release that eventually contributes to the development of neuropsychiatric illness [3]. Several clinical and preclinical studies reported anxiety,

depression and memory deficits after chronic exposure of Noise stress [4–5]. Extensive research found that oxidative stress, hyperphosphorylation of tau protein, inflammatory cytokines, dendritic atrophy, “hypothalamic pituitary adrenal” (HPA) axis over-activation, modulation of cholinergic system and reduction in hippocampal neurogenesis are the key underlying mechanisms of neuronal damage by noise exposure [2,5–7]. Long-term exposure to high-intensity sound, may modify hippocampus GABA-inhibitory transmission, which may contribute to hippocampal long-term potentiation (LTP) impairment [8, 9]. Noise exposure even in low intensity could also disrupt the sleep cycle that in turn leads to activation of the HPA axis, and augmentation of stress hormones [10].

Similarly, light pollution is also a key causative factor for the progress of neuropsychiatric anomalies. The most common form of light pollution is Dim light at night (dLAN), emitting from sources like street

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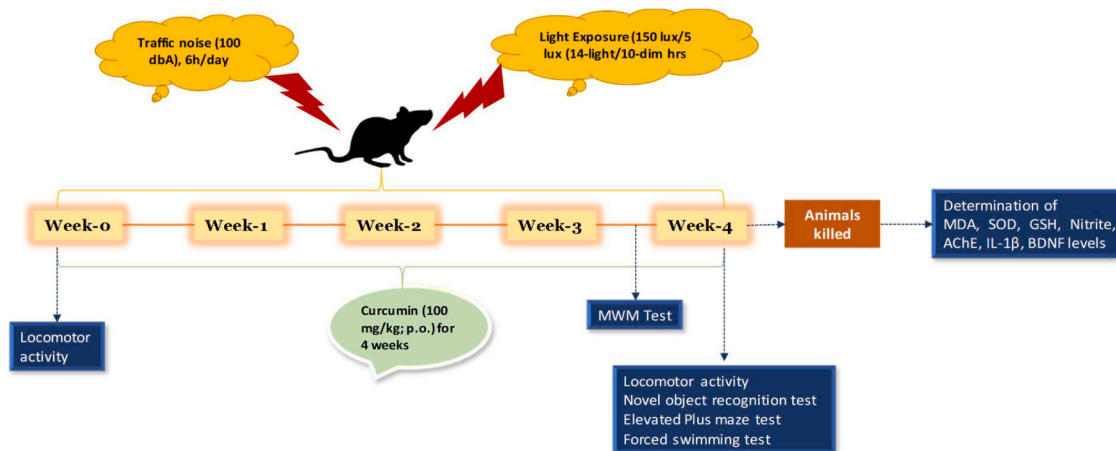


Fig. 1. Schematic representation of the experimental design timeline.

lights, television, and vehicle lights. The exposure of dLAN has been reported to be associated with immunosuppression, metabolic disruption, anxiety, depression and alteration of circadian system [11]. These effects are mediated through activation of several deleterious pathways that are associated with suppression of melatonin, and HPA axis over-activation [12]. Substantial amount of evidences suggested that neurobehavioral anomalies in dLAN exposed animals caused by oxidative stress, downregulation of hippocampal BDNF, cAMP-response element binding protein (CREB), and Synapsin II genes [13,14]. Thus, the previous studies indicated the detrimental role of noise and light pollution on the behavioral function. Moreover, there are some common and interactive pathways underlying the light and noise mediated behavioral anomalies such as oxidative stress [15], HPA-overactivation, reduction in melatonin level, augmentation of pro-inflammatory cytokines [16], and impairment of BDNF signaling in the hippocampus and other parts of brain [17]. To the best of our understanding, no prior investigation has reported so far on the assessment of interactive effects of light and noise pollution. Thus, the major goal of designed research was to explore the neurobehavioral function of rats exposed to light and noise.

The natural polyphenolic compound Curcumin (diferuloylmethane), also known as “Indian Saffron”, found in rhizomes of tropical plant, *Curcuma longa*. Numerous research evidence catalogued its antioxidant, anti-apoptotic, anti-inflammatory, neuroprotective hepatoprotective, anti-microbial and nephroprotective effects [18–22]. Studies have suggested that neuroprotection exhibited by curcumin is mediated through regulation of HPA-axis, BDNF signaling, and attenuation of pro-inflammatory cytokines production in the hippocampus [18,22]. These properties of curcumin have gained a great deal of attention in light and noise exposure induced neurobehavioral anomalies. Therefore, we also aimed to explore the neuroprotective potential of curcumin on animal behavior, oxidative stress, and associated inflammation in the hippocampal region of light and noise exposed rats.

Material and methods

Drug and chemicals

Curcumin was purchased from Sigma Aldrich, India. Curcumin solution was prepared in 2% gum acacia solution. Interleukin-1 (IL-1) and BDNF kits were purchased from Elabscience, India. All further reagents used in the experiment were of analytical grade.

Animals and experimental design

The NIB, Noida, India, provided male young Wistar Rats (200–230

g). The departmental Institutional Animal Ethic Committee (IAEC) examined and approved the experimental procedure (Approval number. IAEC/KSOP/E/19/10). All of the experiments were performed in accord to the standards set by the Committee for Control and Supervision of Experiments on Animals (CCSEA), India. Throughout the trial, animals were maintained in a room with a temperature within range of $22 \pm 3^\circ\text{C}$ and a humidity of 70%. The animals were allowed unrestricted access to a routine feed and water. The male Wistar rats were allocated into five experimental groups, each with six animals in it, as follows: Group 1 serves as the standard control group. Group 2 as Light group: rats were exposed to 150 lux and 5 lux light intensities for 14 h bright and 10 dim light hours (dLAN), respectively for 28 days. Group 3 as Noise group: rats were exposed to noise level (100 dBA) for 28 days (6 h/day). Group 4 and 5 as a light+noise and light+noise+curcumin groups, respectively: rats were simultaneously exposed to 150 lux and 5 lux light intensities (14 h bright and 10 dim light hours) and 100 dBA (6 h/day) noise level for 28 days. In addition, curcumin (100 mg/kg; per oral) was administered in the group 5 for 28 days. On day-0 and day-28, locomotor activity was measured through the Actophotometer apparatus. Novel object recognition test (NORT) and Morris water maze test (MWM test) were conducted on the last five and three days of the study, respectively. Forced swimming test (FST), Elevated plus maze test (EPM test) and estimation of IL-1 β and AChE were also used to assess the better understanding of the anxiety and depressive-like behavior and physiological response towards the end of the trial. All of the neurobehavioral testing took place in a dimly lit environment. Noise induces neuro-inflammation, microglial activation and BDNF depletion in the hippocampus and that this effect was analysed via detection of the expression of the IL-1 β and BDNF [24]. Animals were euthanized on the 29th day of the experiment for neurochemical assessment. The hippocampal tissue was instantly dissected and homogenized in chilly phosphate buffer saline. Fig. 1 depicts a design for an experimental investigation.

Experimental procedures

Light and noise exposure

For the exposure of light pollution, rats were exposed to two distinctive lightening phase conditions (150 lux and 5 lux light intensities for 14 h bright and 10 dim light hours (dLAN) respectively) for 28 days [25]. The intensities of light cycles were maintained by means of light-emitting diodes (LEDs) bulbs purchased locally. Bulbs were placed in the center of the rack cages of rats to provide uniform light exposure. Light intensity was measured by lux meter. In another experimental group, animals were exposed to noise level (Intensity: 100 dBA) for 28 days (6 h/day) at daytime through a loudspeaker [26]. The loudspeaker and cages were 30 cm distant apart from each other. Animals were

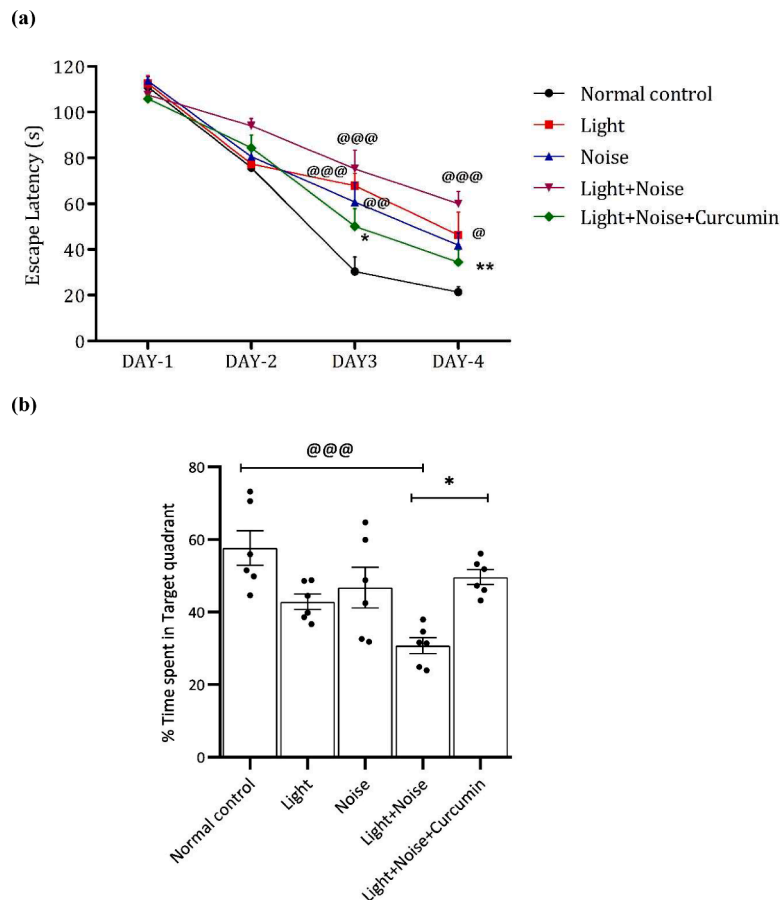


Fig. 2. Effects of Light and Noise pollution exposures and curcumin treatment on (a) Escape latency, and (b) percentage time spent in the target quadrant in the Morris water maze (MWM). Data are expressed as mean $\pm$ S.E.M. for 6 rats in each group. @@@ $p \leq 0.001$, @@ $p \leq 0.01$, @ $p \leq 0.05$ significantly different compared to the control group. ** $p \leq 0.01$, * $p \leq 0.05$ significantly different compared to the light-noise exposed group.

exposed to artificial traffic noise on highways and high-speed railways. We recorded the traffic noise for the experiments at Ghaziabad-Meerut Highway (NH-58) for 3 h and rail noise was collected from Delhi-Dehradun railroad for 3 h. The noise level was measured using a Phoenix sound level meter. 100 dBA intensity of noise was chosen in the experiment because it is equivalent with noise level produced in nightclubs, ceremonies, heavy traffic jams and industrial workplaces. During the light and noise exposures, the animals were given unlimited food and water.

Measurement of locomotor activity

A laser beam digital Actophotometer was utilized to examine the consequence of light and noise exposure on exploratory behavior. The number of laser beam interruption is directly proportional to activity of the animal. Locomotor activity was expressed as photocell counts in 5 min [27].

Morris water maze (MWM) test

In the MWM test, a circular black coloured tank (diameter 180 cm and height 40 cm) was filled with drinking water ($25 \pm 1^\circ\text{C}$) up to 30 cm. The tank was divided into four quadrants of equal size. In one quadrant, a 13 cm square platform was positioned 1 cm beneath the water's surface. The animal was placed in the pool for 120 s (facing toward tank wall) during the acquisition and was free to seek out the submerged platform. The rat was positioned on the platform for 30 s if it did not locate the platform till 120 s. Each animal was put through four trials per day for continuous four days, with a ten-minute interval between them. The average escape latency, or the time taken by rat to find the platform was recorded and reported across trials. A probe trial was also done on

the fifth day in the same water-filled circular tank with no platform. The time spent in the target quadrant in the MWM tank was calculated and reported [28].

Novel object recognition test (NORT)

The NORT procedure is divided into three phases: habituation, familiarization, and testing. All phases of the test were conducted in a black open box arena (50 cm x 50 cm x 36 cm). The individual animal was positioned in an empty open box for five minutes during the habituation phase. The animal was then allowed 10 min to investigate a plastic ball and a woody cylindrical object that had been placed within the box. The woody cylindrical object was replaced with a triangle-shaped object in the third phase, and the animal was given three minutes to explore the items. The extent of time spent for exploring each thing, both new and old, was noted. The animal's recognition index was then calculated by dividing the period spent examining the novel object by the period spent exploring both things. The % recognition index was used to calculate the results [29].

Elevated plus maze (EPM) test

A plus-shaped labyrinth with two open along with two close arms was used in the EPM device ($35 \times 5 \text{ cm}^2$). EPM was 50 cm above the ground level. Individual rats were placed in the EPM's center region, and the number of entrances and total period consumed in the open and closed arms were noted for five minutes. The amount of time spent in open arm and the percentage of open arm entries were calculated and presented [30].

Table 1
Effect of Light and Noise pollution exposure and curcumin treatment on (a) Recognition memory in Novel object recognition test, (b) Locomotor activity, (c) Anxiety-like behavior in Elevated plus maze test, and (d) Depressive-like behavior in Forced swimming test.

Groups	Parameters	Normal control	Light	Noise	Light+Noise	Light+Noise+ Curcumin
	Recognition index (%)	61.34±6.78	33.39±6.05 ^a	40.71±4.19	27.44±4.65 ^b	51.18±5.10 ^c
Locomotor activity (Photocell counts in 5 min.)	Day-0	352.26±19.05	333.15±21.31	298.09±10.24	307.66±17.79	362.42±8.94
	Day-28	327.38±10.89	300.80± 23.44	337.02±30.94	291.84±28.88	298±17.47
	% Time spent in open arms	36.06±6.78	15.61±3.85 ^a	14.22±3.23 ^a	10.72±2.50 ^b	29.39±4.25 ^c
	% No. of open arm entries	63.80±3.93	32.55±7.10 ^b	38.33±6.73 ^a	26.09±4.71 ^b	52.97±6.12 ^c
	Immobility time (in sec)	79.21±5.64	139.80±10.41 ^a	143.49±16.56 ^b	157.44±7.18 ^b	101.73±17.19 ^c

Data are expressed as mean ± S.E.M. for 6 rats in each group. ^a $p \leq 0.05$
^b $p \leq 0.01$ significantly different compared to the control group. ^c $p \leq 0.05$ significantly different compared to the light-noise exposed group.

Forced swimming test (FST)

In this experiment, a cylindrical container of 25 cm height with 10cm diameter was employed, which was filled with drinking water up to the level of 20 cm and 25±1 °C temperature was maintained. The animal was kept in the tank for 6 min, during which time its last 5 min of activity was recorded and presented [31].

Measurement of oxido-nitrosative stress parameters

As previously reported [32], the level of malondialdehyde (MDA) in the hippocampus was estimated using the thiobarbituric acid reactive substances (TBARS) spectrophotometric technique. The MDA level in the hippocampal cortex was measured and represented in micromoles per mg of total protein. Furthermore, superoxide dismutase (SOD) level was also measured to assess the endogenous antioxidant level [33]. The test is based on SOD's capacity to prevent the chemical nitroblue tetrazolium (NBT) from being photo-reduced. The results were computed and presented as units per milligram of protein. The Lowry method was followed to determine the protein level in the hippocampus [34]. The hippocampal GSH level was estimated using a modified Ellman method described in a previous report [33]. The data were calculated and presented as micromoles per mg of protein. In nitrite level estimation, equal volume of Griess reagent and hippocampal supernatant mixed and stored at room temperature in a dark room. After that, the mixture's absorbance was noted at 540 nm, and the results obtained were represented in micro-moles per mg of total protein [33].

AChE estimation

The activity of AChE in isolated hippocampus was measured using Ellman's reagent colorimetric test, as previously reported [33]. In a brief, 100 µl of homogenised hippocampal samples were combined with 0.5 mM 5,5-dithiobis (2-nitrobenzoic acid) (DTNB) and 0.1 M, 650 µl phosphate buffer. Absorbance was noted at 412 nm after 5 µl of acetylthiocholine was added with the mixture. The activity of AChE was estimated and presented in micromoles per minute per milligramme of protein.

Quantification of IL-1β and BDNF

The levels of IL-1β and BDNF in the hippocampus were quantified using ELISA kits as per the manufacturer's directions. The results of IL-1β and BDNF were computed and represented as picogram/mg of protein and nanogram/mg of protein, respectively.

Statistical analysis

The mean ± standard error of mean was used to represent all of the findings. The learning in the MWM test was assessed using a two-way analysis of variance (ANOVA) followed by a Bonferroni test. The study's other data was subjected to a one-way ANOVA followed by a Tukey's test using GraphPad Prism (8.0 Version for Windows, Graph Pad Software). The statistical significance values were set at $p \leq 0.05$, $p \leq 01$, and $p \leq 001$.

Results

Effects of light and noise exposures and curcumin treatment on learning and memory assessed through Morris water maze test and novel object recognition test

In MWM, time taken by the animals to locate and mount the platform was measured during acquisition phase. Escape latency was measured to assess the learning in different experimental groups. As reported in Fig. 2 (a), there was a significant difference in the escape latency of light ($p \leq 0.001$), noise ($p \leq 0.01$) and light-noise ($p \leq 0.001$) exposed rats in comparison to normal control group on day-3. However, on the fourth day, escape latency was found significantly longer in light ($p \leq 0.05$) and light+noise ($p \leq 0.001$) exposed animals when compared with normal control group. The concurrent treatment of curcumin significantly shortened the escape latency in light+noise exposed animals on the third day ($p \leq 0.05$), and fourth day ($p \leq 0.01$) of MWM test. Only the light+noise group animals spent considerably ($p \leq 0.001$) less period in the target quadrant during probe testing than the normal control group (Fig. 2b). However, the part of period spent in the target quadrant in rats receiving curcumin was markedly ($p \leq 0.05$) shorter in comparison to the light+noise exposed animals.

The locomotor activity was also assessed in different experimental groups to assess the effects of different exposures and treatment on locomotor activity. Between day 0 and day 28, there was no significant difference in the locomotive activity of the various experimental groups.

In addition, the recognition memory was evaluated using NORT. Our findings revealed that when rats were exposed to light or light+noise, their recognition index (percent) decreased significantly ($p \leq 0.05$ and $p \leq 0.01$ respectively) as compared to the control group (Table 1). Concurrent treatment with curcumin markedly ($p \leq 0.05$) prevents the decline in recognition index (%) caused by light+noise exposure. Overall, the MWM test and NORT results show that curcumin can prevent learning as well as memory deficits in rats exposed to light and noise. Moreover, simultaneous exposure of light and noise caused profound cognitive deficits in comparison to light or noise exposure alone.

Effects of light and noise exposures and curcumin treatment on anxiety, and depressive-like behavior assessed through elevated plus maze test and forced swimming test

The EPM test was used to evaluate anxiety-like behavior. As demonstrated in Table 1, rats from the light ($p \leq 0.05$; $p \leq 0.01$), noise ($p \leq 0.05$; $p \leq 0.05$) and light+noise ($p \leq 0.01$; $p \leq 0.01$) groups spent considerably less time in open arms (percent) and had a markedly lesser number of open arm entries (percent) than normal controls and which is significantly different compared to the light-noise exposed group. However, in the curcumin treated rats, all of the alterations induced by light+noise was reversed ($p \leq 0.05$), indicating anxiolytic potential of curcumin. In addition, FST was used to assess depressive-like behavior in several experimental groups. The immobility time (%) was considerably increased in light ($p \leq 0.05$), noise ($p \leq 0.01$) and light+noise ($p \leq 0.01$) exposed animals in comparison to normal controls, indicative

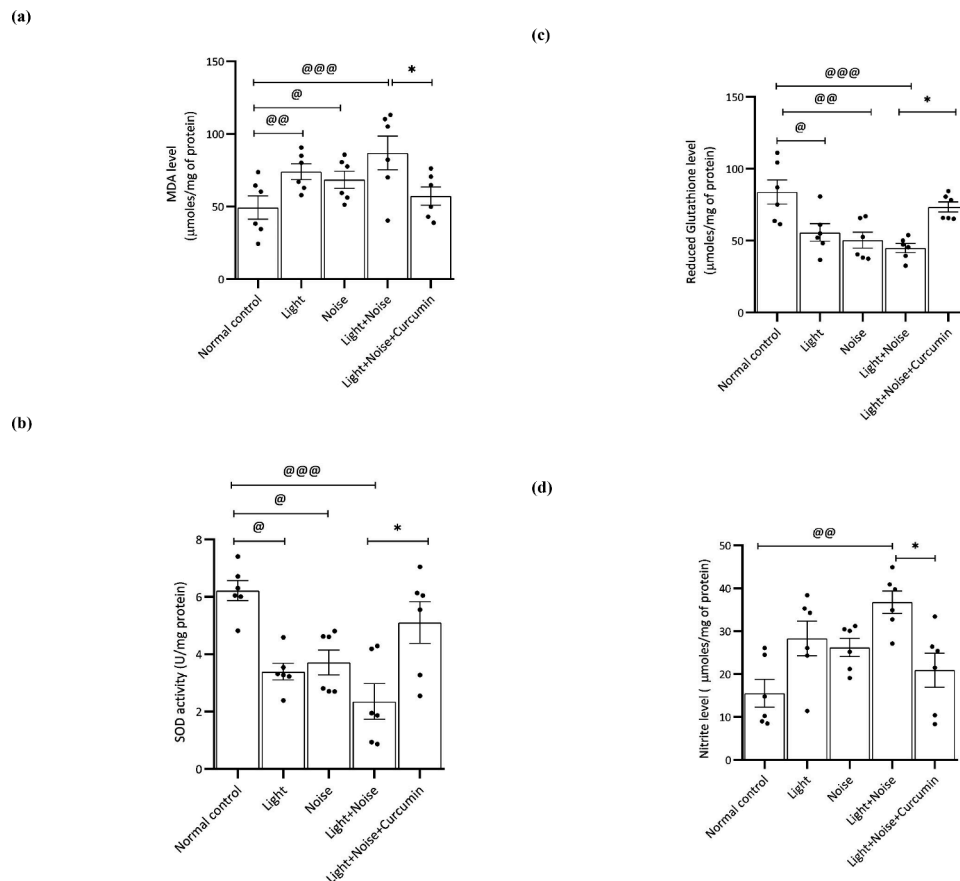


Fig. 3. Effects of Light and Noise pollution exposures and curcumin treatment on (a) Malondialdehyde (MDA), (b) Superoxide dismutase (SOD), (c) Reduced glutathione, and (d) Nitric oxide (NO) in the hippocampus. Data are expressed as mean $\pm$ S.E.M. for 6 rats in each group. @@@ $p \leq 0.001$, @@ $p \leq 0.01$, @ $p \leq 0.05$ significantly different compared to the control group. * $p \leq 0.05$ significantly different compared to the light-noise exposed group.

depressive-like behavior. However, in the light+noise group, immobility time was found to be considerably ($p \leq 0.05$) reduced following curcumin therapy (Table 1), confirming curcumin's anti-depressant effect.

Effects of light and noise exposures and curcumin treatment on oxidative stress markers in the hippocampus

As shown in Fig. 2, a significant rise in hippocampal levels of MDA was observed in light ($p \leq 0.01$), noise ($p \leq 0.05$), and light+noise ($p \leq 0.001$) exposed animals in comparison to controls. Moreover, results revealed that exposure to light, noise and light+noise caused substantial reduction in SOD activity ($p \leq 0.05$, $p \leq 0.05$, $p \leq 0.001$, respectively) and GSH level ($p \leq 0.05$, $p \leq 0.01$, $p \leq 0.001$, respectively) in animals compared to normal controls (Fig. 2b and c). As compared to the normal control group, NO levels were noted to be remarkably lower ($p \leq 0.01$) only in the light+noise exposed animals (Fig. 2d). Thus, our results revealed that light+noise exposure caused marked elevation in oxido-nitrosative stress in comparison to noise or light alone. Concurrent administration of curcumin, on the other hand, significantly ($p \leq 0.05$) reduced oxido-nitrosative stress, as evidenced by lower MDA and NO levels and higher GSH and SOD levels.

Effects of light and noise exposures and curcumin treatment on AChE, IL-1 β and BDNF levels in the hippocampus

The study results showed that the group of animals exposed with light and light+noise exhibited a significant ($p \leq 0.05$ and $p \leq 0.001$) increase in hippocampal AChE activity as compared with the control

group (Fig. 3a). Curcumin therapy, on the other hand, significantly ($p \leq 0.01$) declined AChE augmentation in light+noise exposed mice. There was no significant effect observed in noise-exposed animals while comparison with normal control rats. Furthermore, when compared to normal controls, the level of IL-1 in the hippocampal region of light ($p \leq 0.05$), noise ($p \leq 0.05$), and light+noise ($p \leq 0.001$) exposed rats increased (Fig. 3b). In the light+noise group, however, curcumin therapy significantly ($p \leq 0.01$) reduced hippocampus IL-1 levels. As shown in Fig. 3c, hippocampal BDNF level was found significantly lowered after light ($p \leq 0.01$), noise ($p \leq 0.05$) and light+noise ($p \leq 0.001$) exposure in different experimental groups. On the other hand, a significant ($p \leq 0.05$) augmentation in BDNF level was observed in curcumin-treated light+noise animals. Collectively, we found that light+noise exposure caused significantly neurochemical anomalies which were prevented by curcumin administration for 28 days.

Discussion

The current study aimed to investigate the individual and combined effects of light and traffic noise pollution on behavioural and neurochemical deficits in adult male Wistar rats, with a focus on exploring the potential protective role of curcumin. Our findings indicate that concurrent exposure to light and noise pollution for four weeks resulted in significant behavioural deficits in rats, accompanied by biochemical alterations indicative of oxidative stress and neuro-inflammation in the hippocampus. Notably, curcumin administration mitigated these adverse effects, suggesting its protective potential against combined light and noise pollution-induced neurobehavioral anomalies. Several studies have highlighted the detrimental impact of environmental

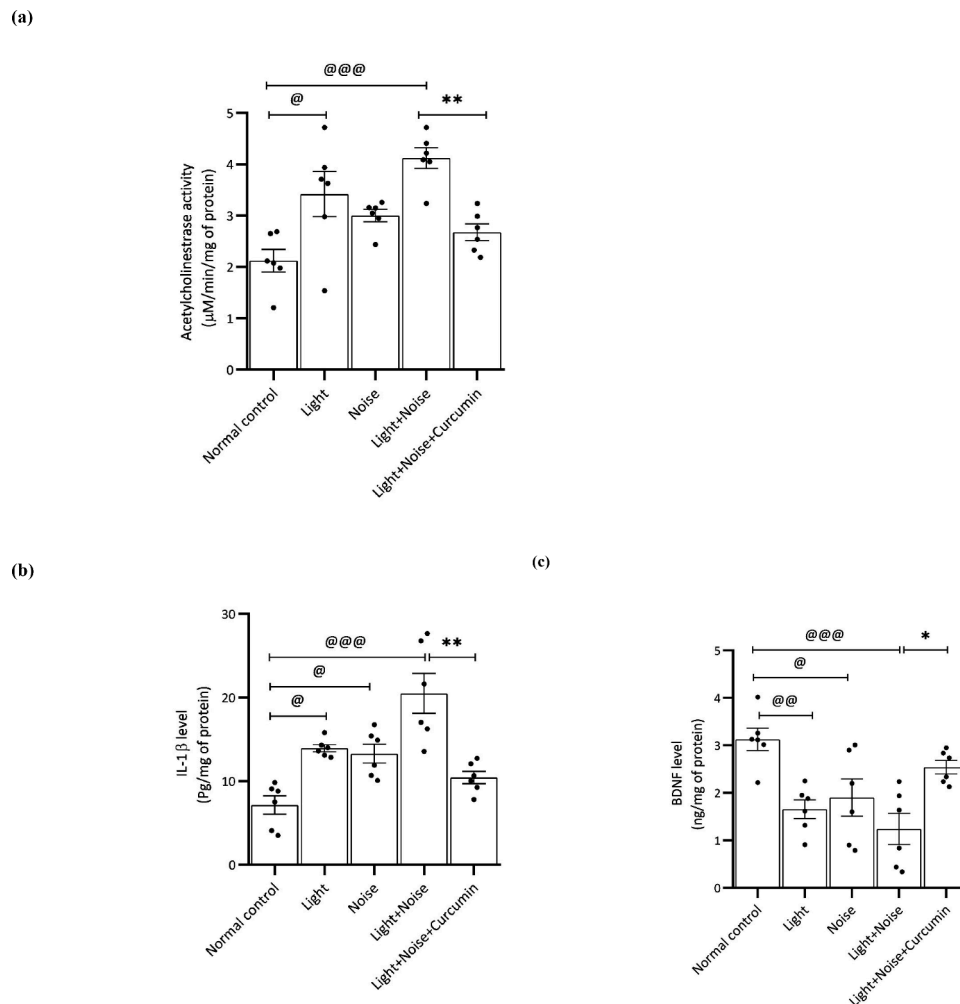


Fig. 4. Effects of Light and Noise pollution exposures and curcumin treatment on (a) Acetylcholinesterase (AChE), (b) Interleukin (IL)-1 β , and (c) BDNF in the hippocampus of light-noise exposed group animals. Data are expressed as mean $\pm$ S.E.M. for 6 rats in each group. @@@ $p \leq 0.001$, @@ $p \leq 0.01$, @ $p \leq 0.05$ significantly different compared to the control group. ** $p \leq 0.01$, * $p \leq 0.05$ significantly different compared to the light-noise exposed group.

pollutants, including air, light, and noise pollution, on human health, particularly with regard to psychological disturbances such as depression, anxiety, and memory deficits [5,11,14,35]. While extensive research has focused on understanding the mechanisms underlying air pollution-induced behavioural dysfunctions [35–37], less attention has been given to the interactive effects of light and noise pollution on behavioural function. Thus, our study fills this gap by demonstrating the deleterious effects of concurrent exposure to light and noise pollution on behavioural performance in rats.

The present results revealed that exposure to light and noise pollution, either individually or in combination, led to oxidative-nitrosative stress, increased pro-inflammatory cytokines, reduced BDNF levels, and altered AChE activity in the hippocampus of exposed animals. These findings are consistent with previous studies linking environmental pollutants to neurochemical alterations and cognitive impairments [6, 13,38]. Importantly, curcumin treatment attenuated these biochemical changes, suggesting its potential neuroprotective effects against pollution-induced hippocampal dysfunction. Behavioral assessment using a battery of tests including the Morris water maze, novel object recognition, elevated plus maze, and forced swimming tests revealed significant impairments in learning, memory, anxiety-like behavior, and depressive-like behavior in rats exposed to light and/or noise pollution. Notably, simultaneous exposure to light and noise pollution exacerbated these behavioral deficits compared to exposure to either pollutant alone. However, curcumin treatment effectively prevented these behavioural

anomalies, indicating its ability to mitigate the adverse effects of combined pollution exposure on behavior. The observed behavioural deficits in light and noise-exposed rats are consistent with previous studies highlighting the neurobehavioral effects of environmental pollutants [39–42]. Furthermore, our findings suggest that while noise exposure alone did not induce cognitive deficits, it augmented the deleterious effects of light pollution on cognitive function, emphasizing the interactive nature of these pollutants in influencing behavior. Oxidative-nitrosative stress emerged as a key mechanism underlying the neurotoxic effects of light and noise pollution, particularly in the hippocampus, a region highly susceptible to oxidative insults [43,44]. Curcumin's antioxidant properties likely contributed to its protective effects by reducing markers of oxidative stress and inflammation in the hippocampus, consistent with previous studies highlighting curcumin's neuroprotective mechanisms [18,23,45]. Cholinergic deficits, characterized by increased AChE activity, have also been implicated in cognitive dysfunction [46]. Our findings of elevated AChE activity in light and noise-exposed rats suggest impairment of cholinergic signaling, which may contribute to memory deficits observed in these animals. However, curcumin treatment attenuated AChE activity, supporting its role in preserving cholinergic function and cognitive performance in polluted environments. Similarly, reductions in BDNF levels observed in light and noise-exposed rats are consistent with previous reports linking environmental pollutants to neurotrophic factor depletion and impaired neurogenesis [6,13]. Curcumin administration

restored BDNF levels, underscoring its potential to enhance neurotrophic support and promote hippocampal plasticity in polluted environments. In conclusion, our study provides evidence of the detrimental effects of concurrent exposure to light and traffic noise pollution on behavioural and neurochemical parameters in rats. Moreover, we demonstrate the protective effects of curcumin against these adverse effects, suggesting its potential as a therapeutic intervention for mitigating pollution-induced neurobehavioral anomalies. These findings contribute to our understanding of the interactive effects of environmental pollutants on brain function and highlight the importance of exploring natural compounds like curcumin as potential neuroprotective agents in polluted environments. Further research is warranted to elucidate the precise mechanisms underlying curcumin's protective effects and to evaluate its therapeutic potential in clinical settings.

Conclusion

In summary, our results revealed that light, noise and light+noise exposures which are common in cities cause memory deficits, anxiety and depressive-like behavior. Thus, people who are exposed to noise and dLAN are at higher risk of psychological disturbances. These effects may have been mediated through suppression of antioxidant defense system, ROS generation and release of inflammatory cytokines in the hippocampus. Further, these hippocampal insults eventually caused augmentation of AChE activity and depletion of BDNF level. Importantly, the present results depicted that simultaneous exposure of noise and light caused profound behavioral anomalies and neurochemical insults. The curcumin treatment is likely able to prevent the development of aforementioned behavioral deficits through strengthening the antioxidant defense system, reducing the pro-inflammatory cytokines, and restoration of AChE activity and BDNF level. Thus, the present study may provide additional insights in the utilization of curcumin for the management of behavioral disorders. Fig. 4

Ethical declaration

All of the experiments were performed in accord to the standards set by the CPCSEA, India. The departmental IAEC examined and approved the experimental procedure (Approval number. IAEC/KSOP/E/19/10).

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CRediT authorship contribution statement

Sonal Sharma: Data curation, Investigation, Methodology, Visualization. **Jiten Singh:** Formal analysis, Writing – original draft, Writing – review & editing. **Dinesh Kumar:** Conceptualization, Data curation, Formal analysis, Software, Writing – review & editing. **Mandeep Kumar Arora:** Data curation, Formal analysis, Validation, Writing – review & editing. **Ashok Jangra:** Conceptualization, Supervision, Writing – review & editing.

Declaration of competing interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

Data availability

My manuscript has no associated data.

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