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A study on the Behavioural alteration (Depression and Anxiety) in experimental rats exposed to five different noise intensities

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ABSTRACT

The present study was designed to assess the altered behavioural activity of Wistar rats exposed to five different noise intensities. Wistar rat was divided into five groups such as EG1, EG2, EG3, EG4, and EG5 and in respect to that 60, 80, 100, 120, and 140 dB noise exposure (1hr/day) was assigned for 30 days, and post noise effects of noise stress were also evaluated on 7th and 14th day by employed Actophotometer and Elevated Plus Maze respectively. The experimental rats of all the groups showed a significant ($p<0.05$) reduction in locomotor activity and increased anxiety level after 1st day of noise exposure of different noise intensities, but animals of group EG1 and EG2 also showed an adaptive response towards their respective noise intensities on 15th, 30th and post noise day i.e. 7th and 14th day, while a consistent reduction in locomotion and increased anxiety level were observed in EG3, EG4 and EG5 groups on animals on noise stressed exposure and post noise days respectively. The present study results suggested that noise stress can alter behavioural activity and may potentiate the risk of neurodegenerative disorders.

Keywords: Noise stress, noise intensities, Behavioral alteration, dB (Decibel), Adaptation

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INTRODUCTION

Noise is ubiquitous in many modern communities which have become relentless stress and thus leisure individual day to day life. Various studies indicated that noise can impose its effects by altering both auditory and non-auditory systems[1]. Furthermore, different studies also suggested that experiences with acute and chronic noise stress affects the central nervous system and shows the deleterious effects on brain structure, cognition[2, 3], therefore, promote the neuropsychiatric disorders[4]. Exposure of noise exceeds up to 90 dB which is the main source of stressor[5]. Although the mechanism of cognitive impairment by the exposure of noise is not yet clear but various evidence suggested that exposure of noise instigate the oxidative reaction which has been involved in the memory impairment and oxidative reactions lead to the free radicals production and in the plasma and tissue it decreases the activity of the antioxidant enzymes[6, 7]. It has been shown in previous studies that elevated oxidative stress is the cause for neuronal degeneration in auditory nuclei together with the brain regions important for cognitive functions[8, 9, 10].

The induction of noise causes modification in the free radical scavenging enzymes as well as lipid peroxidation distinct the regions of the brain[11]. The brain acknowledges the level of sound and distinguishes the level of stress. The brain is the most important organ which illustrates and reacts to potential stressors[12]. Therefore, noise can work as a non-specific stressor persuading stress reactions that are in line with the general stress model. Furthermore, noise is the most common cause of sleep disturbance and leads to acoustic stimuli. Hence, elevated heart rate is caused via autonomic responses[13]. Moreover, such a minimal level of disturbance that may be troubling during sleep might not have significant impact on wakefulness. Consequently, even with the suppressive impact of sleep state on the stress system and plasma level of stress hormones sleep disruption induces higher activation of these stress mechanisms typical of what is seen in the wakeful state[14].

A study investigated that noise stress alters the functions of the immune by damaging natural killer cells[15]. Prenatal exposure study also suggested that the noise stress in the gestation period gives rise to impaired cognition, low weight birth, and postnatal growth to noise stress in animals[16]. Chronic experience of unavoidable noise stress induces tiredness due to deregulations of various feedback mechanisms and leads to decreased muscle movement and social interactions[17]. Studies of noisy activities have studied in mice and undertaken the detrimental consequences of traffic noise experience on the brain, although the harmful effects of noise stress on wellbeing and behavioral role have been widely expressed in both animals and humans, there is no approaching cohort research to understand if the nocturnal noise sensitivity has the same effects as recorded for the exposure of daytime. The influence of the period of exposure in the everyday circadian cycle is important to know since the impact of environmental stress depends on the form, time, and length of exposure, as well as age, sex, and the test conditions [18, 19]. Provided that, chronic stress can severely impact neural plasticity in diverse brain regions, particularly in limbic structures, i.e., the hippocampal development, mPFC, and amygdala[20]. Collectively, exposure to noise stress in daily life can affect the biological systems to a greater extent.

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MATERIALS AND METHODS

Experimental animals

The research was commenced after taking permission from the Institutional Animal Ethical Committee of Kamla Nehru Institute of Management and Technology, Sultanpur, Uttar Pradesh, India. The Wistar strain of animals (291–296 g) was employed in the present study. Before starting the protocol of the experiment to the experimental animals, seven days of acclimatization period was provided in normal condition. In an open wire cage, Wistar rats were kept in a 12 h light-dark cycle and a temperature-controlled room with free access to standard laboratory rat chow and tap water were provided. Habituation of animals with some undesired stresses, such as handling and habitat, imposed during the acclimatization period, except these, experimental animals were not exposed to any other stresses.

NOISE TREATMENT

To generate a noise environment, a rectangular special noise chamber was designed. Inside the chamber, speakers were fixed with an amplifier and a noise meter was arranged and set to record noise level inside the acoustic chamber and a controller knob was set to ensure the release of desired sound intensities in dB i.e. 60, 80, 100, 120 and 140 dB respectively. The animals were placed inside the noise chamber with their home cage. Before the commencement of the procedure, the animals were kept within the noise chamber without sound to get familiarized with the interior of the chamber for the duration of the acclimatization period.

Study design

In this study, male Wistar rats (291–296 g) were employed to assess the alteration in behavior. The experimental animals were divided into five and six groups (n=6) as EG1, EG2, EG3, EG4, and EG5 for locomotion activity and Elevated Plus Maze based on noise stress exposure of 60, 80, 100, 120, and 140 dB for 1 h/day for 30 days, respectively. The time of noise stress exposure (11:00 AM–12: 00 PM) was kept constant for each exposed group throughout the end of the experiment. A sham control group was also constructed that was not exposed to any kind of the noise except surrounding habitat, but it was kept in experimental conditions with the noise-exposed groups. Assessment of locomotor and anxiety was done by using Actophotometer and Elevated Plus Maze respectively. After the exposure of noise stress (1hr/day for 30 days) firstly, locomotion activity was evaluated and determination of anxiety level was followed by after 5 minutes on 1^{st}, 15^{th}, and 30^{th} day, a post noise effect was also evaluated for every group on 7^{th} and 14^{th} day respectively.

Assessment of Locomotor Activity Using Actophotometer test

The locomotor behavior is known as an indicator of consciousness and was tested by holding the rats in the Actophotometer separately. The locomotor operation was measured in terms of counts every 10 minutes in an Actophotometer, the action of the animal interrupts the light beam dropping on the photocell and a count is registered digitally. Therefore, the amount of counts is directly linked to the movement of the animal within the Actophotometer chamber[21]. The animals were divided into five groups namely EG1, EG2, EG3, EG4, and EG5, and each group consisting six animals. Before starting the noise procedure, the basal count (B/COUNT) of locomotion was taken on an Actophotometer of each group of animals and then the locomotion test was conducted at 12:10 PM after 10 minutes of noise stress exposure of different intensities assigned to the respective group for 10 minutes cut off time respectively.

Assessment of Anxiety using Elevated Plus Maze

Elevated Plus Maze composed of two open arms (50 cm×10 cm) facing each other; two sealed arms of the same proportions with walls 40 cm high crossed the open arms. A central rectangle, 10 cm×10 cm were linked with both arms to giving the apparatus a plus sign appearance. The maze was raised 70 cm above the floor in a dimly lit space. Briefly, rodents have a natural disdain towards high and wide spaces and prefer enclosed arms, which have a burrow like an ambiance and thus spend a larger amount of time in the enclosed arm. When introduced to the novel maze alley, the animals encounter an approach-avoidance confrontation, which is greater in the open arm when opposed to the enclosed arms[22].

The animals were divided into six groups namely EG1, EG2, EG3, EG4, and EG5, and SC (Sham Control) group, and each group consisting six animals. The sham control (SC) group was not exposed to any type of noise stress but it was routinely kept in the noise chamber for 1hr/day for 30 days. The animals of all groups primarily imposed to habituation on EPM arena for 5 minutes and noise exposure was initiated after habituation imposed. The effect of noise onto the anxiety level was then assessed at 1:45 PM to 2:15 PM for a 5 minute cut off time period for all the group respectively.

Results

Effect of 60 dB Noise stress onto locomotor activity

Animals of group EG1showed a significant ($p<0.0001$) reduction in locomotion activity on the 1st day after noise exposure while the average locomotion on the 15th and 30th day and even on post noise days i.e. 7th and 14th day were found to increased and these increment were not significant ($p>0.05$) when compared with their basal locomotion count taken prior to the noise exposure respectively. Results showed that there were no further reductions in locomotor activity were observed between 1st day versus 15th, 30th, and even on post noise days i.e. 7th and 14th days, on the contrary, locomotion activity were significantly ($p<0.05$) increased on 15th, 30th and post noise days i.e. 7th and 14th day when compared to locomotion activity on 1st day respectively. Similarly, no further reduction in locomotion activity was observed between 15th versus 7th& 14th day (post noise days); 30th day versus 7th& 14th day, and also between 7th and 14th day respectively. (Fig 1)

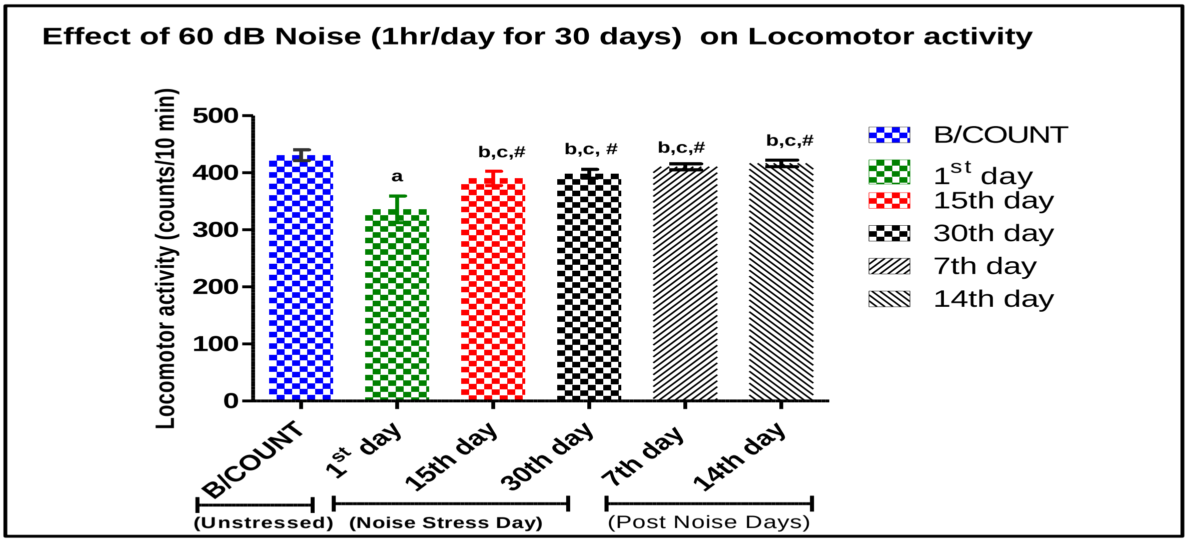

Fig 1: Effect of 60 dB noise stress on locomotor activity of EG1 group in terms of count in 10 minutes time interval in the Actophotometer test. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{a}p<0.05$ versus B/COUNT; $^{b}p>0.05$ versus B/COUNT; $^{c}p<0.05$ versus 1st day; $^{\#}p>0.05$: 15th versus 30th versus 7th versus 14th day respectively.

Effect of 80 dB noise stress onto locomotor activity

Animals of group EG2 were exposed to 80 dB noise intensity showed a significant ($p<0.0001$) reduction in their locomotor activity on the 1st and 15th day, while average increment was observed in locomotion on the 30th day and also on post noise days i.e. 7th and 14th day and it was not significant ($p>0.05$) when compared to their basal count taken prior to the noise exposure respectively. When locomotion was compared between 1st day versus 15th day no significant difference ($p>0.05$) in locomotion was found, whereas a significant increase in locomotion on 30th and post noise days i.e. 7th and 14th were observed when compared to 1st day respectively, similarly no any significant difference in locomotion was observed between 15th day, 30th and 7th day (post noise day), while significant ($p<0.05$) increment in locomotor activity was observed on 14th day (post noise day) when compared to 15th day. On the contrary, no significant differences in locomotion were observed between the 30th and 7th& 14th days (post noise day) and also between post noise days i.e. 7th and 14th respectively. (Fig 2)

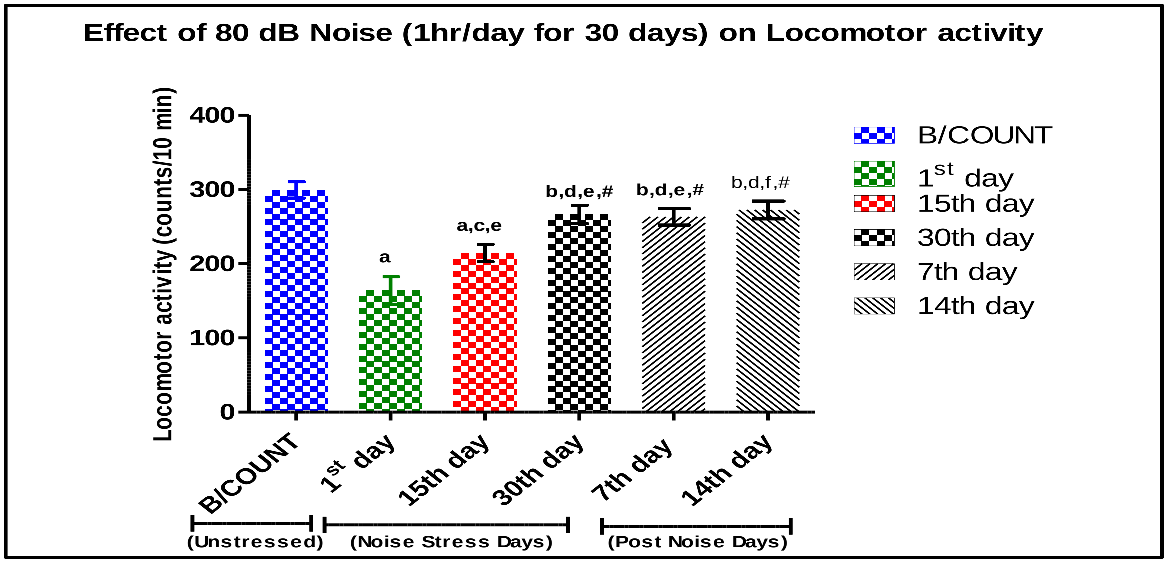

Fig 2: Effect of 80 dB noise stress on locomotor activity of EG2 group in terms of count in 10 minutes time interval in the Actophotometer test. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{a}p<0.05$ versus B/COUNT; $^{b}p>0.05$ versus B/COUNT; $^{c}p>0.05$ versus 1st day; $^{d}p>0.05$ versus 1st day, $^{e}p>0.05$ versus 15th, 30th, 7th day; $^{f}p<0.05$ versus 14th day and $^{\#}p>0.05$ versus 30th, 7th and 14th day respectively.

Effect of 100 dB noise stress onto locomotor activity

The locomotion activity of group EG3 was significantly ($p<0.0001$)reduced on the 1st, 15th, 30th day, and even on post noise days i.e. 7th and 14th day when compared to their basal count taken prior to the noise exposure respectively. Similarly, significant reductions in locomotion were also observed on the 15th, 30th day. An average locomotion activity was recovered after post noise i.e. 7th and 14th day but it was still lower than when compared to 1st day respectively. On the contrary, no significant differences ($p>0.05$) in locomotion were observed between the 15th, 30th, and 7th& 14th days (post noise day) and also between post noise days i.e. 7th and 14th respectively. (Fig 3)

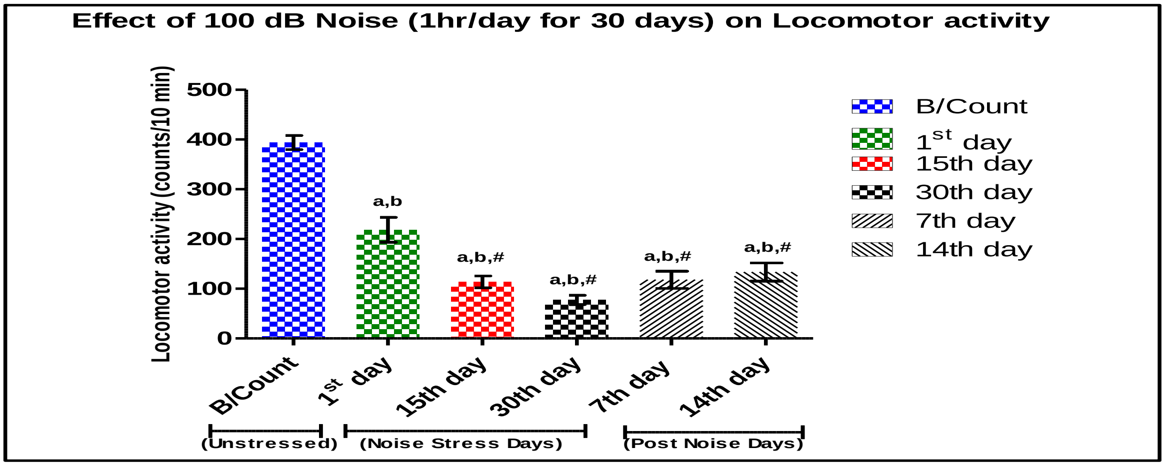

Fig 3: Effect of 100 dB noise stress on locomotor activity of EG3 group in terms of count in 10 minutes time interval in the Actophotometer test. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one-way ANOVA followed by Tukey's multiple range tests; [a]p<0.05 versus B/COUNT; [b]p<0.05 versus 1st day and [#]p>0.05 versus 15th, 30th, 7th, and 14th day respectively.

Effect of 120 dB and 140 dB noise stress onto locomotor activity

The animals of group EG4 and EG5 showed a similar response in their locomotion. Locomotion activity was significantly (p<0.0001)reduced on 1st, 15th, 30th, and on post noise days i.e. 7th and 14th when compared to their basal count taken prior to the noise exposure in both animal groups respectively. Similarly, significant (p<0.0001) reductions in locomotor activity were observed between the 15th, 30th, and post noise days i.e. 7th and 14th day when compared to locomotor activity on 1st day in both experimental groups respectively, while an average improvement was observed on post noise days i.e. 7th and 14th in both of experimental groups but that improvement in locomotion was not significant in both experimental group with each other and with 15th and 30th day respectively. (Fig 4 and Fig 5)

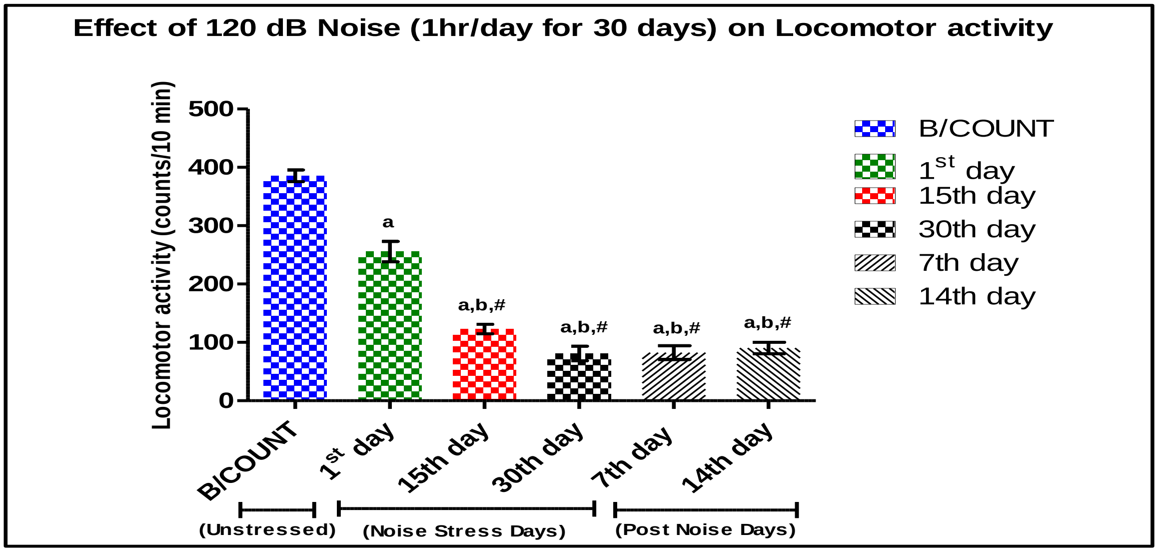

Fig 4: Effect of 120 dB noise stress on locomotor activity of EG3 group in terms of count in 10 minutes time interval in the Actophotometer test. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one-way ANOVA followed by Tukey's multiple range tests; $^{a}p<0.05$ versus B/COUNT; $^{b}p<0.05$ versus 1st day and $^{\#}p>0.05$ versus 15th, 30th, 7th, and 14th day respectively.

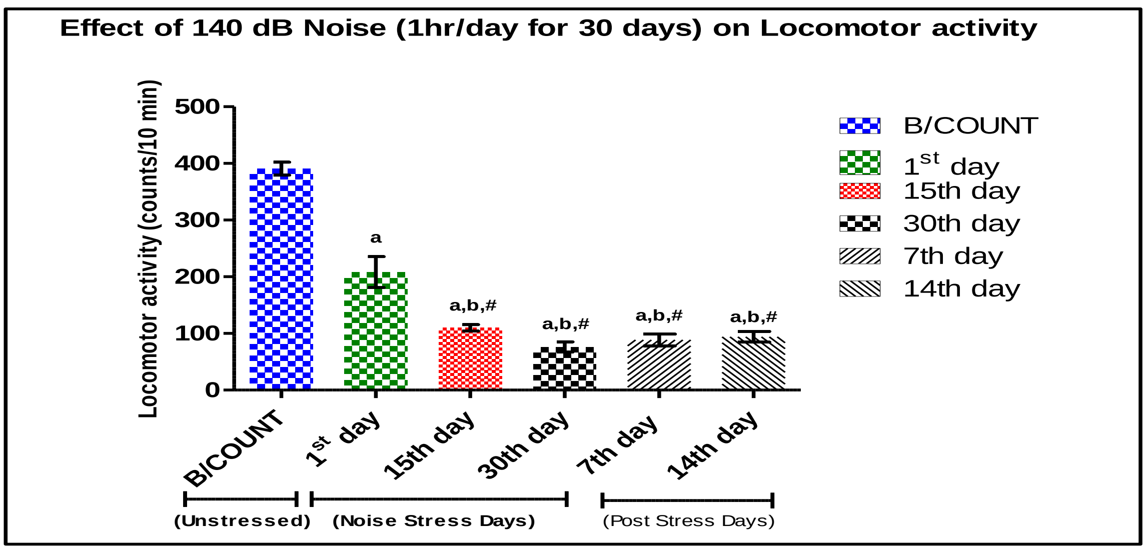

Fig 5: Effect of 140 dB noise stress on locomotor activity of EG3 group in terms of count in 10 minutes time interval in the Actophotometer test. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one-way ANOVA followed by Tukey's multiple range tests; $^{a}p<0.05$ versus B/COUNT; $^{b}p<0.05$ versus 1st day and $^{\#}p>0.05$ versus 15th, 30th, 7th, and 14th day respectively.

Effect of noise stress on anxiety level using an elevated plus-maze

Effect of noise stress 60 dB on anxiety level

Before the noise exposure, the Basal Time Spent [BTS] (sec.) and the Number of Entries [NOE] in the open arm of Sham control (SC) and EG1 were not found significant ($p>0.05$) respectively. After exposure to the noise stress, time spent [TS] (sec.) in open arm reduced on 1st ($p<0.0001$), 15th ($p<0.05$) day whereas, reduction on 30th day was found not significant ($p>0.05$) and the NOE in open arm reduced significantly ($p<0.05$) on 1st day but no significant reduction ($p>0.05$) were observed on 15th and 30th day and also on post noise day, there was no any significant ($p>0.05$) reduction were observed on 7th and 14th day when compared to sham control (SC) respectively.

The BTS(sec.) and the NOE in the open arm of group EG1 were analyzed before and after the noise stress exposure (1hr/day/30) day and the TS (sec) in the open arm on 1st, 15th and 30th day was significantly reduced ($p<0.05$) and the NOE in open arm significantly reduced on 1st day only whereas, no any significant ($p>0.05$) reduction was observed on 15th and 30th day and also on post noise day i.e. 7th and 14th day when compared to BTS(sec.) and the NOE of group EG1 animals respectively.

During the noise stress exposure, the TS(sec.) and the NOE in open arm between 1st day versus 15th and 30th day, 15th day versus 30th day of group EG1 was not significantly ($p>0.05$) affected but a significant ($p<0.05$) increment were observed after post noise day, i.e. on 7th and 14th day when compared to 1st and 15th day, in contrast, there is no any significant difference found on 30th (stressed) day versus 7th and 14th day (post noise) and 7th day versus 14th day respectively. (Fig 6 and 7)

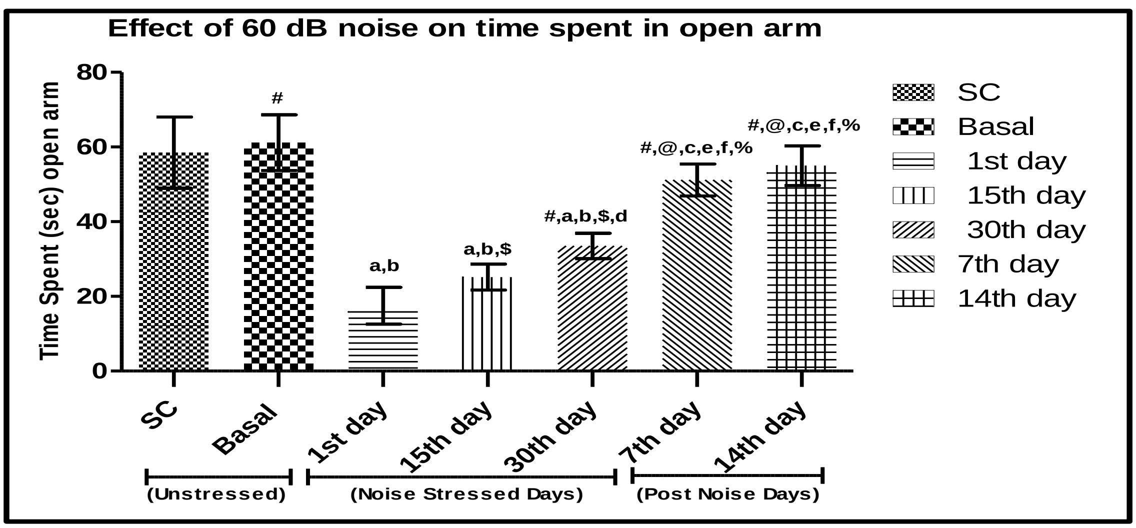

Fig 6: Effect of 60 dB noise stress on TS in the open arm of EG1 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{\#}p>0.05$ versus sham control (SC); $^{a}p<0.05$ versus SC; $^{b}p<0.05$ versus basal; $^{@}p>0.05$ versus basal; $^{\$}p>0.05$ versus 1st day; $^{c}p<0.05$ versus 1st day; $^{d}p>0.05$ versus 15th day; $^{e}p>0.05$ versus 15th day; $^{f}p>0.05$ versus 30th day; $^{\%}p>0.05$ between each other respectively.

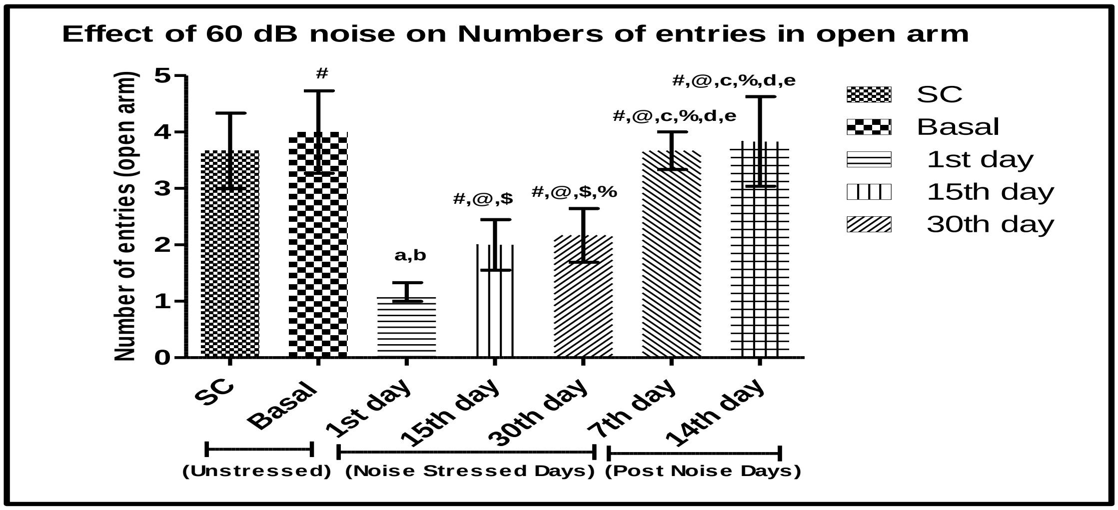

Fig 7: Effect of 60 dB noise stress on NOE in the open arm of EG1 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{\#}p>0.05$ versus sham control (SC); $^{a}p<0.05$ versus SC; $^{b}p<0.05$ versus basal; $^{@}p>0.05$ versus basal; $^{\$}p>0.05$ versus 1st day; $^{c}p<0.05$ versus 1st day; $^{\%}p>0.05$ versus 15th day; $^{d}p>0.05$ versus 30th day; $^{e}p>0.05$ between each other respectively.

Effect of 80 dB noise stress on anxiety level

Before the noise exposure, the BTS(sec.) and the NOE in the open arm of Sham control (SC) and test group EG2 were not found significant ($p>0.05$) respectively. After exposure to the noise stress, TS (sec.) in the open arm reduced on 1st ($p<0.0001$), 15th ($p<0.05$), and 30th ($p<0.05$) day, and the NOE in the open arm reduced significantly ($p<0.05$) on 1st day but no significant reduction ($p>0.05$) were observed on 15th and 30th day and also on post noise day, there was no any significant ($p>0.05$) reduction were observed on 7th and 14th day when compared to sham control (SC) respectively.

The BTS(sec.) and the NOE in the open arm of group EG2 were analyzed before and after the noise stress exposure (1hr/day/30) day and the TS (sec) in the open arm significantly reduced ($p<0.05$) on 1st and 15th but on 30th day and post noise day i.e. 7th and 14th day, no significantly reduced ($p>0.05$) was observed and the NOE in open arm significantly ($p<0.05$) reduced on 1st day only whereas, no any significant ($p>0.05$) reduction was observed on 15th and 30th day and also on post noise day i.e. 7th and 14th day when compared to BTS(sec.) and the NOE of group EG2 animals respectively.

During the noise stress exposure, the TS (sec.) and the NOE in open arm between 1st day versus 15th and 30th day, 15th day versus 30th day of group EG2 was not significantly ($p>0.05$) affected respectively and similarly, TS (sec.) in the open arm was not significantly ($p>0.05$) reduced and the NOE in open arm increased significantly ($p<0.05$) between 1st day versus post noise day i.e. 7th respectively, whereas, TS (sec.) and the NOE in open arm increased significantly ($p<0.05$) between 1st day versus post noise day i.e. 14th respectively. In contrast, TS (sec.) and the NOE in open arm in between 15th versus post noise day i.e. 7th& 14th day, and between 30th versus post noise day i.e. 7th& 14th day was found not significant ($p>0.05$) respectively. (Fig 8 and 9)

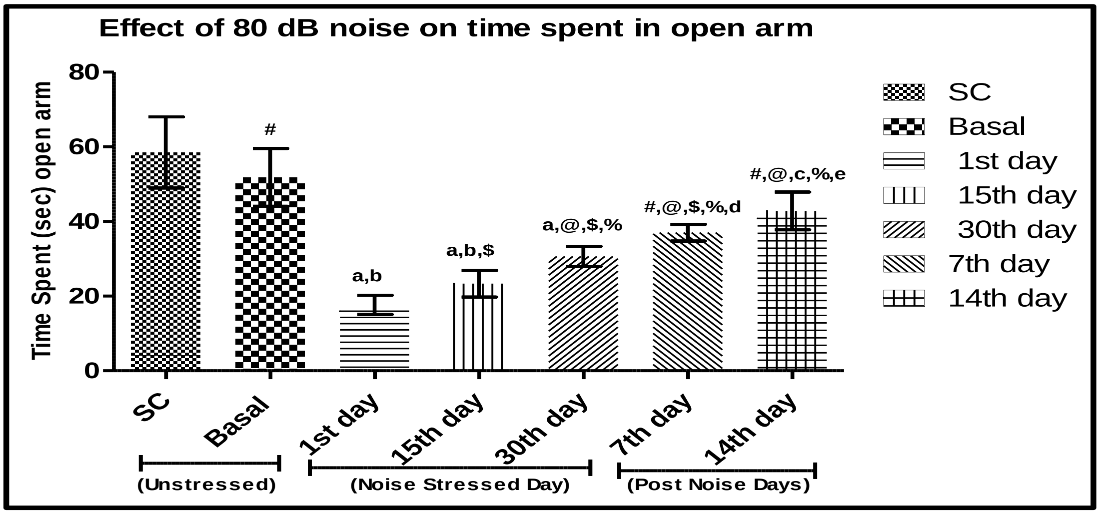

Fig 8: Effect of 80 dB noise stress on TS in the open arm of EG2 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{\#}p>0.05$ versus sham control (SC); $^{a}p<0.05$ versus SC; $^{b}p<0.05$ versus basal; $^{@}p>0.05$ versus basal; $^{\$}p>0.05$ versus 1st day; $^{c}p<0.05$ versus 1st day; $^{\%}p>0.05$ versus 15th day; $^{d}p>0.05$ versus 30th day; $^{e}p>0.05$ between each other respectively.

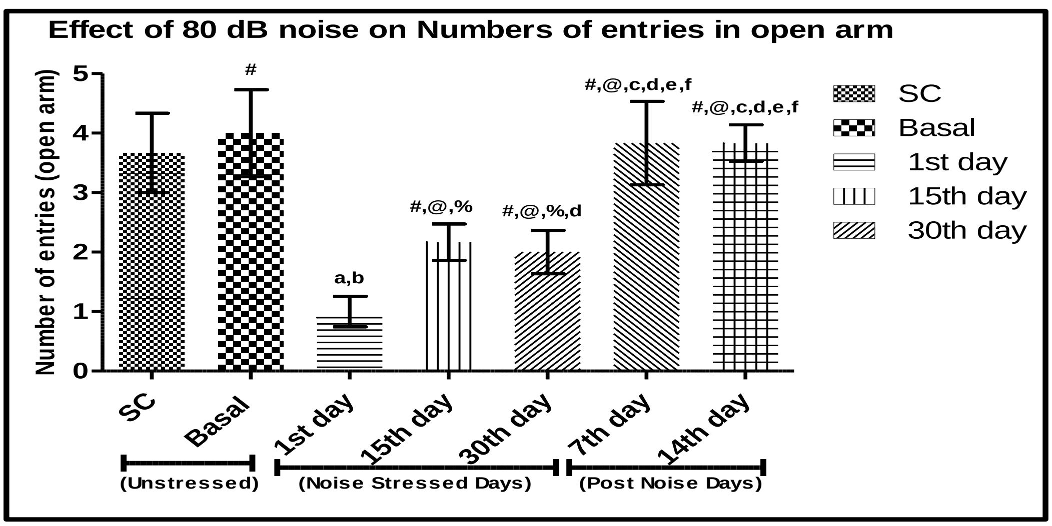

Fig 9: Effect of 80 dB noise stress on NOE in the open arm of EG2 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{\#}p>0.05$ versus sham control (SC); $^{a}p<0.05$ versus SC; $^{b}p<0.05$ versus basal; $^{@}p>0.05$ versus basal; $^{\%}p>0.05$ versus 1st day; $^{c}p<0.05$ versus 1st day; $^{d}p>0.05$ versus 15th day; $^{e}p>0.05$ versus 30th day; $^{f}p>0.05$ between each other respectively.

Effect of 100 dB noise stress on anxiety level

Before the noise exposure, the BTS(sec.) and the NOE in the open arm of Sham control (SC) and test group EG3 were not found significant ($p>0.05$) respectively.

After exposure to the noise stress, TS (sec.) in the open arm reduced on 1st ($p<0.0001$), 15th ($p<0.05$), and 30th ($p<0.05$) day, and the NOE in the open arm reduced significantly ($p<0.05$) only on 1st day but no significant reduction ($p>0.05$) were observed on 15th and 30th day and also on post noise day, there was no any significant ($p>0.05$) reduction were observed on 7th and 14th day when compared to sham control (SC) respectively.

The BTS (sec.) and the NOE in the open arm of group EG3 were analyzed before and after the noise stress exposure (1hr/day/30) day respectively and the TS (sec) and the NOE in open arm significantly reduced on 1st ($p<0.0001$), 15th ($p<0.05$) and 30th ($p<0.05$) day, whereas reduction on post noise day i.e. 7th and 14th day was not found significant ($p>0.05$) respectively.

During the noise stress exposure, the TS(sec.) and the NOE in the open arm between 1st day versus 15th and 30th day, 15th day versus 30th day of group EG3 was not significantly ($p>0.05$) affected respectively. TS (sec.) in the open arm was not significantly ($p>0.05$) reduced and the NOE in the open arm increased significantly ($p<0.05$) between 1st day versus post noise day i.e. 7th respectively, whereas, TS (sec.) and the NOE in open arm increased significantly ($p<0.05$) between 1st day versus post noise day i.e. 14th respectively. In contrast, TS(sec.) and the NOE in open arm in between 15th versus post noise day i.e. 7th& 14th day and between 30th versus post noise day i.e. 7th& 14th day was found not significant ($p>0.05$) respectively, whereas TS (sec.) in open arm significantly not affected on post noise days i.e. 7th and 14th day but the NOE in open arm, on post noise days i.e. 7th and 14th significantly ($p<0.05$) increased when compared to 1st day (stressed) of group EG3 respectively, whereas no any significant increment ($p>0.05$) in TS (sec.) and the NOE of group EG3 were found on post noise days i.e. 7th and 14th day when compared to 15th and 30th day respectively. (Fig 10 and 11)

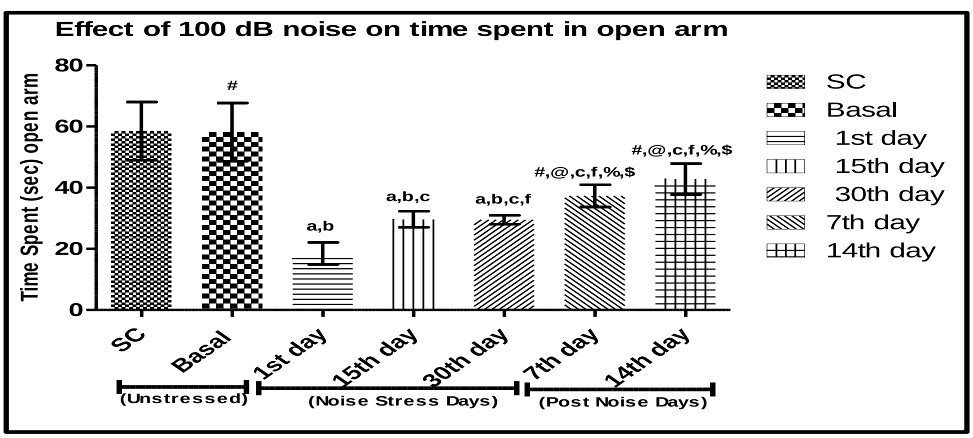

Fig 10: Effect of 100 dB noise stress on TS in the open arm of EG3 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; [#]$p>0.05$ versus sham control (SC); [a]$p<0.05$

versus SC; [b]p<0.05 versus basal; [@]p>0.05 versus basal; [c]p>0.05 versus 1st day; [f]p<0.05 versus 15th day; [%]p>0.05 versus 30th day; [$]p>0.05 between each other respectively.

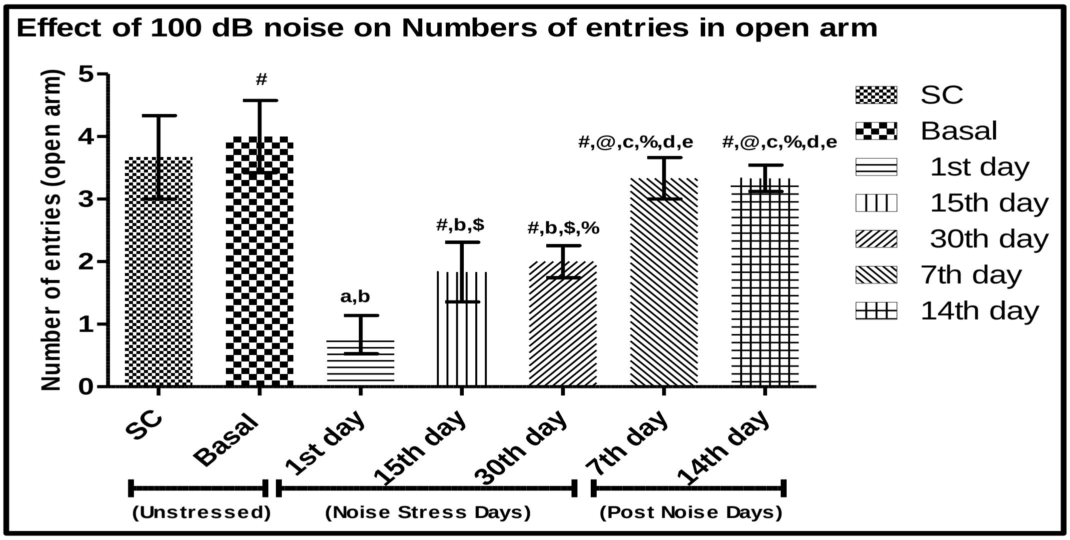

Fig 11: Effect of 100 dB noise stress on NOE in the open arm of EG3 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; [#]p>0.05 versus sham control (SC); [a]p<0.05 versus SC; [b]p<0.05 versus basal; [@]p>0.05 versus basal; [$]p>0.05 versus 1st day; [c]p<0.05 versus 1st day; [%]p>0.05 versus 15th day; [d]p>0.05 versus 30th day; [e]p>0.05 between each other respectively.

Effect of 120 dB noise stress on anxiety level

Before the noise exposure, the BTS(sec.) and the NOE in the open arm of Sham control (SC) and test group EG4 were not found significant (p>0.05) respectively.

After exposure to the noise stress, TS(sec.) and the NOE in open arm significantly (p<0.0001) reduced on 1st, 15th, and 30th day and also on post noise day i.e. 7th and 14th day when compared to sham control (EQPC) respectively. In a similar manner, the BTS(sec.) and the NOE in the open arm of group EG4 were analyzed before and after the noise stress exposure (1hr/day/30) day respectively, and the TS (sec.) and the NOE in open arm significantly reduced on 1st, 15th and 30th day, and also on post noise day i.e. 7th and 14thday, when compared to TS(sec.) and the NOE in the open arm of group EG4 respectively. In contrast, during the noise stress exposure and post noise days, the reduction was found not significant for TS(sec.) and the NOE in the open arm between 1st day versus 15th, 30th day & post noise days (7th and 14th day), 15th day versus 30th day & post noise days (7th and 14th day), 13th day versus post noise days (7th and 14th day) and post noise days i.e. 7th versus 14th day of group EG4 respectively (Fig 12 and 13).

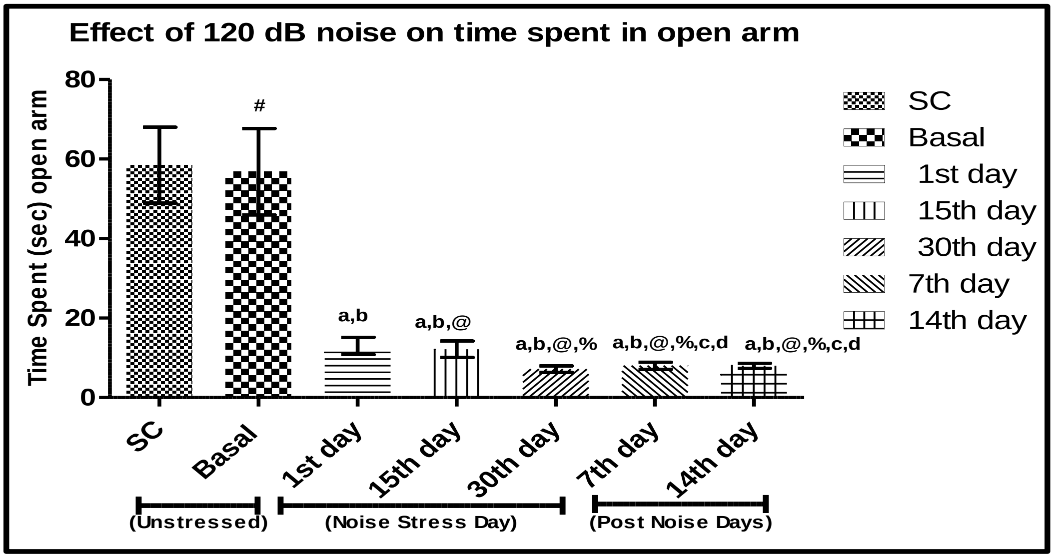

Fig 12: Effect of 120 dB noise stress on TS in the open arm of EG4 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; [#]p>0.05 versus sham control (SC); [a]p<0.05 versus SC; [b]p<0.05 versus basal; [@]p>0.05 versus 1st day; [%]p>0.05 versus 15th day; [c]p<0.05 versus 30th day; [d]p>0.05 between each other respectively.

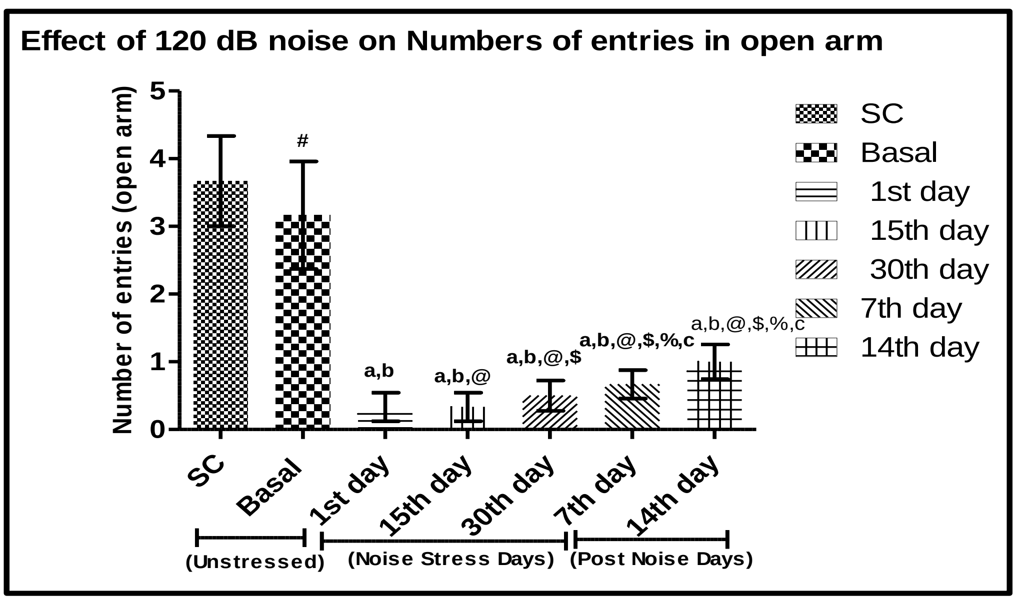

Fig 13: Effect of 120 dB noise stress on NOE in the open arm of EG4 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; [#]p>0.05 versus sham control (SC); [a]p<0.05 versus SC; [b]p<0.05 versus basal; [@]p>0.05 versus basal; [$]p>0.05 versus 1st day; [c]p<0.05

versus 1st day; $^{\%}$p>0.05 versus 15th day; ^{d}p>0.05 versus 30th day; ^{e}p>0.05 between each other respectively.

Effect of 140 dB noise stress on anxiety level

Before the noise exposure, the BTS(sec.) and the NOE in the open arm of Sham control (SC) and test group EG5 were not found significant (p>0.05) respectively.

After exposure to the noise stress, TS(sec.) and the NOE in open arm significantly (p<0.0001) decreased on 1st, 15th, and 30th day and also on post noise day i.e. 7th and 14th day when compared to sham control (EQPC) respectively. In a similar manner, the BTS (sec.) and the NOE in the open arm of group EG5 were analyzed before and after the noise stress exposure (1hr/day/30) day respectively, and the TS (sec) and the NOE in open arm significantly reduced on 1st, 15th and 30thday, and also on post noise day i.e. 7th and 14th day, when compared to TS (sec.) and the NOE in the open arm of group EG5 respectively. In contrast, during the noise stress exposure and post noise days, the reduction was found not significant for TS (sec.) and the NOE in the open arm between 1st day versus 15th, 30th day & post noise days (7th and 14th day), 15th day versus 30th day & post noise days (7th and 14th day), 13th day versus post noise days (7th and 14th day) and post noise days i.e. 7th versus 14th day of group EG5 respectively (Fig 14 and 15).

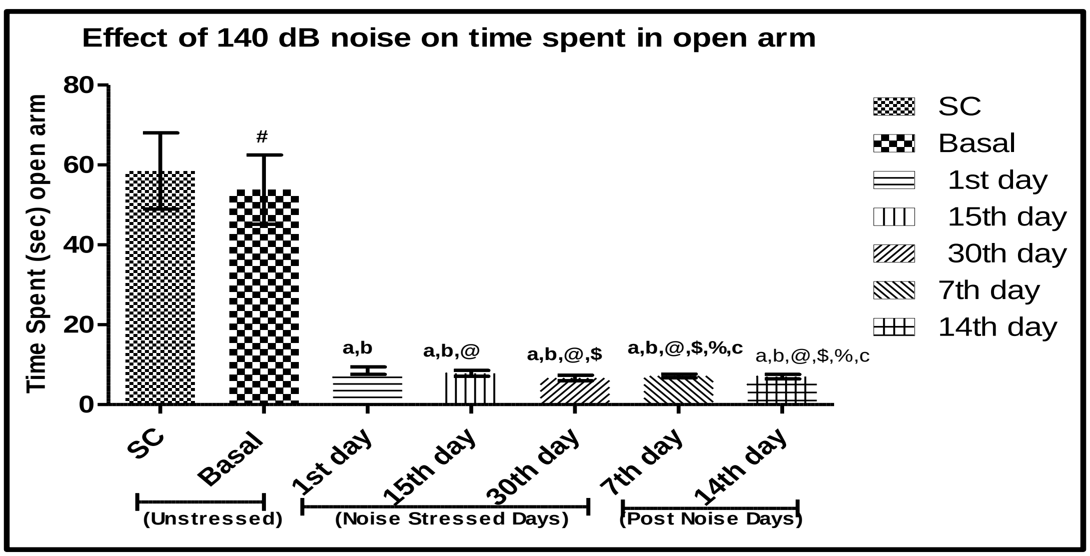

Fig 14: Effect of 140 dB noise stress on TS in the open arm of EG5 group in terms of count in 5 minutes interval of time in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically analyzed by using one way ANOVA followed by Tukey's multiple range tests; $^{\#}$p>0.05 versus sham control (SC); ^{a}p<0.05 versus SC; ^{b}p<0.05 versus basal; $^{@}$p>0.05 versus 1st day; $^{\$}$p>0.05 versus 15th day; $^{\%}$p<0.05 versus 30th day; ^{c}p>0.05 between each other respectively.

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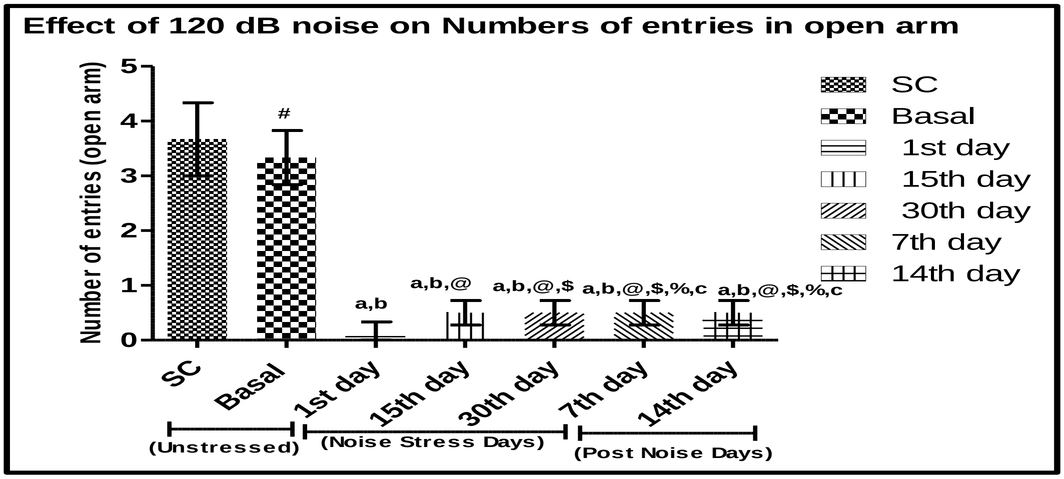

Fig 15: Effect of 140 dB noise stress on NOE in the open arm of EG5 group in terms of count in 5 minutes time interval in the Elevated Plus Maze. Values in the graph are mean±s.e.m of six rats. Data were statistically evaluated by using one way ANOVA followed by Tukey's multiple range tests; $^{\#}p>0.05$ versus sham control (SC); $^{a}p<0.05$ versus SC; $^{b}p<0.05$ versus basal; $^{@}p>0.05$ versus 1st day; $^{\$}p>0.05$ versus 15th day; $^{\%}p<0.05$ versus 30th day; $^{c}p>0.05$ between each other respectively.

Discussion

Stressors in any sense, have an impactful control upon human Psychology like mood, our sense of well-being, behavior, and health. The acute and chronic stressor is the noble threat to the biological system and the response to the stressor is called the "stress response." However induced stress provokes an adaptive cascade in the biological system. In this regard, Selye observed that severe, expanded stress responses, insult tissue and it defiantly commenced disease pathogenesis. Stress can alter psychological well-being and initiates neuronal responses resulting in a behavioral disorder like depression and anxiety. [23, 24]

In this context, noise is an environmental factor that inducing stress and leads to neurochemical alteration that includes the imbalance of neurotransmitters in the brain. [25]

In the present study, animals (Wistar rats) were intentionally subjected to five different noise intensities, and the effects of these were assessed to evaluate the behavioral changes by employed two experimental models of depression and anxiety.

Impaired locomotion is an indicative parameter of poor motor coordination and Actophotometer is a well established and reliable methods to evaluates the same, presented study showed that all five noise intensities (60, 80,100,120, and 140 dB) successfully decreased the locomotion activity on 1st day of noise stress exposure in all experimental groups compared to their basal locomotion count before the noise stress treatment. The presented results supported the past study conducted on higher levels of noise intensities. Responses onto the locomotor activity of the five different noise intensities were varied, when the locomotor activity evaluated on the 15th and 30th day, the animals of group EG1 and EG2 showed a mean average increased in locomotor responses and the responses were not significant with locomotion on 1st day and even with their

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basal count before the noise stress, therefore, this may suggest the concept of general adaptation with the repeated stressful stimuli described by Hans Selye, according to his concept of general adaption, the three-stage process is alarm reaction, adaptation stage and exhaustion stage [25]It means the animals of group EG1 and EG2 adapted themselves according to the stressful situation and an average recovery in the locomotion activity was observed, which was not significantly differ with their basal locomotion count before the noise exposure. It is well accepted and documented in the literature that quantity, types, and exposure (duration) of stressful stimuli elicit the outcomes, here, in our study group EG1 and EG2 were exposed to lower noise intensities that may present in the surrounding biological system in daily routine, it is also well established that acute stress potentiating neuroendocrine and neurotransmitter levels in the brain as an adaptive response to work against the effect of stress, while prolonged chronic stress changes in neurotransmitter levels in response to stress may insults the homeostasis [22]and this happened to our study in other reaming three noise stress exposed groups i.e. group EG3, EG4, and EG5 as these group exposed to higher noise intensities and results showed that the locomotor activity potentially decreased on 1st, 15th and 30th day and there were no average increments in the locomotor count observed when compared to their basal count before the noise stress exposure. These events suggested that the brain area participates in motor control, such as the cerebellum, basal ganglia, and motor cortex. In the locomotion, through basal ganglia, the well known dopaminergic neurotransmitter plays an important role in locomotion and movement. Furthermore, the cerebellum responds to motor regulation and synchronization operation by multiplying motor production with the continuing sensory reaction. During noise, enhanced dopamine promoted the free radical damage in the cerebellum contributes to hurt motor control, and also, cerebellar Purkinje cells discharge inhibitory neurotransmitters i.e. GABA which can decrease the impulse transmission. Activation of glutamatergic and GABAergic neurotransmission systems also changes motor coordination [26]. So, fruitful locomotor activity is akey to attentiveness, and a reduction in it indicating an increase in sedative activity. [27]

In another behavioral parameter of an elevated plus-maze that was employed to test the induction of anxiety in animals of group EG1, EG2, EG3, EG4, and EG5 showed that after the noise stress exposure a decrease in TS and NOE in open arm were significantly reduced on after 1st-day exposure of different noise intensities compared to the sham control group that was not exposed to any noise stress but an average increment in TS and NOE in open arm were observed in animals of group EG1 and EG2 and the increment were not Statistically differing to their basal count and sham control group. These changes again suggested the general adaptation of the animals to repeated stressful stimuli. The results were also showed the disruption of adaption in animals exposed to higher intensities of group namely EG3, EG4, and EG5, and the average TS and NOE in the open arm significantly decreased on the 15th and 30th day compared to their basal and sham group respectively. The present research findings are in accordance with the previous study concluded that acute stress, caused by some other approach including immobilization stress causes behavioral changes like a reduction in locomotor function, social behaviour, and exploratory behavior 28. Latest human research have indicated that fear or maternal tension for the duration of childbirth adversely modifies the neuro-motor growth of infants. [29, 30]

Induction of stress by a stressor is regulated by more than one brain region and mainly involvement of brain region to controlling behaviors during stress are the amygdala, the hypothalamus, the hippocampus, the cingulated gyrus, the fornix, and anterior pituitary (as HPA axis) [31, 32]. Any research have indicated that these stress-receptive regions of the brain had more corticosteroid receptors when opposed to some other regions of the brain. Thus, these are highly vulnerable to the belongings of stress. It is important to remember that the amygdala, hippocampus, and prefrontal cortex are often involved with the HPA axis to monitor the discharge of CRH through CRH neurons through the hypophyseal portal system, which controls signals in the pituitary gland to trigger the release of ACTH. ACTH arouses the adrenal cortex to produce cortisol in humans and corticosterone in rodents. [33]

Furthermore, many studies demonstrate the role of stress in the development of depressive-like symptoms via neurochemical alterations [34] and also endogenous corticosterone and other glucocorticoids in stressful conditions influencing gene expression and regulation all over the body, and this enhancement in corticosterone levels provokes several behavior alterations like memory deficits, fear, and anxiety. [35, 36, 37]

Thus, corticosterone is the final result attributable to the activation of the hypothalamic-pituitary-adrenal axis.As a result, stress conditions are associated with an increase in plasma corticosterone levels. [38]Many studies in past revealed that in stressful events hippocampus is the center of attraction and stress modifies hippocampal neurons and results in underlying cognitive and memory deficits. [39, 40] Neuronal releases induced by noise stress results in the secretion of glutamate [41, 42] and if the chronic level of noise stress has been revealed to raise glutamate in the hippocampus. [43, 44] Past studies suggested that a high density of glucocorticoids present in the hippocampus [45, 46] and stress-induced glucocorticoid discharge of glutamate has been shown to encourage neuronal damage. [47] This resultant initiation of glutamate increases glutamine by enhancing glutamine synthetase activity in all brain regions [24] and this glial specific enzyme is responsive to glutamate and regulates its level at the synaptic cleft [48, 49]. A study showed that in the restraint stress animal model the prolonged activation of glutamine synthetase leads to increased glutamate toxicity in the brain and increased glutamine synthetase activity is responsible to decrease glutamate clearance[50]. Stress also has an impact on GABA, which is also stressor specific, in a chronic cold stress study, a decrease in GABA content was observed in the hypothalamus, cortex, and olfactory bulb, whereas chronic noise stress reported decreasing GABA in the hippocampus [51].

CONCLUSION

The biological systems are surrounded by a numbers of stressful triggers and impacts of the same are uncountable health defects. Noise stress is already an existing stress stimulus in well developed and developing countries across the world, where industrial and traffic noise is a major issue and living system meet with different intensities of noise in that surrounding environment in much sense. If the exposure of noise persists prolonged or if the noise is the part of daily life, it may leads to pathogenesis of many diseases. The present study revealed that the lower and higher noise intensities if persist for longer duration can alter the behavioral integrity of the rats and impaired the locomotion and potentially induced anxiety in the rats. This could be possible due to change in hormonal (Corticosterone), neuronal and cellular pathways. A well established futuristic approach may warrant for the development of an animal model with noise stress that mimicking the effects of acute and chronic exposure of noise intensities in the human beings in respect to evaluate negative outcomes of noise exposure and drug development for the same.

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AUTHORS CONTRIBUTION

The first and corresponding author carried out the behavioral study and paper writing. Second and third authors designed the work and critical revision of the manuscript.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest concerning this research article.

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