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Original Research Article

Evaluation of Anxiety and Motor Function Following Chronic Monosodium Glutamate Exposure in Swiss Albino Mice Using Open Field and Scurry Speed Tests

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ABSTRACT

Background: Monosodium glutamate (MSG) is a widely used flavour enhancer responsible for the characteristic umami taste in many foods. Although regulatory authorities classify MSG as generally safe, several experimental studies suggest that excessive or prolonged exposure may affect neurological and behavioural functions. Glutamate is the principal excitatory neurotransmitter in the central nervous system, and excessive activation of glutamate receptors may lead to neuronal excitotoxicity, oxidative stress, and behavioural disturbances. Therefore, evaluating the neurobehavioral consequences of chronic MSG exposure is important. The present study aimed to evaluate the effects of chronic MSG administration on anxiety-like behaviour and motor function in Swiss albino mice.

Methods: A prospective interventional experimental animal study was conducted using forty adult male Swiss albino mice weighing 20–30 g. The animals were randomly divided into four groups (n = 10 per group). The control group received distilled water, while the experimental groups received MSG at doses of 40 mg/kg, 60 mg/kg, and 80 mg/kg intraperitoneally for three months. Behavioural assessments were conducted at baseline and at three days, one month, two months, and three months.

Anxiety-like behaviour was evaluated using the Open Field Test, where the number of line crossings and time spent in the central area were recorded as indicators of exploratory activity and anxiety level. Motor function was assessed using the Scurry Speed Test by measuring the time taken by mice to traverse a straight-line track leading to a dark compartment. Reduced exploratory behaviour in the open field indicates increased anxiety-like behaviour, whereas increased traversal time in the scurry speed apparatus reflects impaired locomotor activity.

Results: The results demonstrated a progressive reduction in exploratory behaviour in MSG-treated groups during the study period. The most pronounced behavioural change was observed in the MSG 60 mg/kg group, where median central square exploration decreased significantly at three months compared with baseline. Motor function analysis using the Scurry Speed Test showed a mild increase in traversal time in MSG-treated groups; however, these changes were not statistically significant.

Conclusion: In conclusion, chronic MSG administration produced dose-dependent anxiety-like behavioural changes without marked impairment of motor function in mice.

Keywords: Monosodium Glutamate, Anxiety, Open Field Test, Scurry Speed Test, Motor Function, Behavioural Neuroscience, Swiss Albino Mice.

INTRODUCTION

Monosodium glutamate (MSG) is the sodium salt of the naturally occurring amino acid glutamic acid and is widely used as a flavour enhancer in processed foods, restaurant meals, and packaged food products. It produces the characteristic umami taste and enhances the palatability of various foods. Increasing urbanisation and the widespread consumption of processed foods have led to a rise in MSG intake in many populations. The average daily intake of MSG in humans has been estimated to range between 0.3 g and 1 g, depending on dietary habits and regional food preferences.[1]

Glutamate is the primary excitatory neurotransmitter in the mammalian central nervous system and plays a critical role in synaptic transmission, neuronal plasticity, learning, and memory. However, excessive glutamate activity can lead to neuronal injury through a mechanism known as excitotoxicity. Overactivation of glutamate receptors such as N-methyl-D-aspartate (NMDA) receptors results in increased intracellular calcium influx,

oxidative stress, mitochondrial dysfunction, and ultimately neuronal damage.[2] These excitotoxic mechanisms have been implicated in several neurological and psychiatric disorders, including neurodegenerative diseases, epilepsy, depression, and anxiety disorders.[3]

Experimental studies have demonstrated that chronic exposure to MSG may induce behavioural alterations and neurochemical changes in various brain regions involved in emotional regulation and motor control. In animal models, MSG administration has been associated with increased oxidative stress, neuronal degeneration, and alterations in neurotransmitter signalling pathways.[4] These changes may affe.

Anxiety disorders represent one of the most common psychiatric conditions worldwide and significantly affect quality of life. Behavioural animal models have been widely used to investigate the neurobiological mechanisms underlying anxiety and to evaluate the effects of pharmacological agents on emotional behaviour. Rodent models are particularly useful because of their genetic similarity to humans and their well-characterised behavioural responses.[5]

Among the commonly used behavioural paradigms, the Open Field Test is a well-established model for assessing anxiety-like behaviour and exploratory activity in rodents. Rodents naturally avoid open and brightly illuminated spaces and tend to remain near the walls of the arena. Increased exploration of the central area indicates reduced anxiety, whereas decreased central exploration suggests anxiety-like behaviour.[6]

Motor coordination and locomotor activity are also important parameters in neurobehavioural studies, as behavioural tests for anxiety often require intact motor function. The Scurry Speed Test is commonly used to evaluate locomotor performance by measuring the time taken by rodents to traverse a straight pathway leading to a safe compartment. Increased traversal time may indicate reduced locomotor activity or impairment of motor coordination.[7]

Although several studies have investigated the toxicological effects of MSG, most available evidence focuses on acute exposure. Data regarding the long-term behavioural consequences of chronic MSG administration remain limited. Therefore, the present study was designed to evaluate the effects of chronic MSG exposure on anxiety-like behaviour and motor function in Swiss albino mice using the Open Field Test and Scurry Speed Test.

MATERIALS AND METHODS

Study Design

The present study was a prospective interventional experimental animal study conducted to evaluate the chronic effects of monosodium glutamate (MSG) on anxiety-like behaviour and motor function in Swiss albino mice.

Study Animals

Forty adult male Swiss albino mice weighing 20–30 g were used for the study. The animals were obtained from a certified laboratory animal breeding facility and housed in polycarbonate cages under standard laboratory conditions. The room temperature was maintained at 22 ± 2°C with controlled humidity and a 12-hour light–dark cycle. The animals were provided with a standard laboratory pellet diet and water ad libitum. Before the start of the experiment, the animals were allowed to acclimatise to the laboratory environment for a period of ten days.

Ethical Approval

The experimental protocol was approved by the Institutional Animal Ethics Committee. All experimental procedures were conducted in accordance with the guidelines of the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA) for the care and use of laboratory animals.

Experimental Groups

The animals were randomly divided into four groups, consisting of ten mice in each group.

Group	Treatment
Control	Distilled water
MSG 40	MSG 40 mg/kg
MSG 60	MSG 60 mg/kg
MSG 80	MSG 80 mg/kg

Monosodium glutamate (MSG) was administered intraperitoneally once daily for a duration of three months in the treatment groups. The control group received an equivalent volume of distilled water intraperitoneally to avoid procedural bias

associated with the injection process. This ensured that all experimental groups were subjected to similar handling and injection conditions throughout the study period.

Behavioural Assessment Timeline

Behavioural assessments were conducted at two time points during the study period: baseline (before MSG administration) and three months after chronic MSG administration. Anxiety-like behaviour was evaluated using the Open Field Test, while motor function was assessed using the Scurry Speed Test. All behavioural observations were recorded for each animal at these two time points and were analysed statistically to determine the effects of chronic MSG exposure on anxiety and motor activity.

Experiment 1: Open Field Test (Assessment of Anxiety-like Behaviour)

The Open Field Test was used to evaluate anxiety-like behaviour and exploratory activity in mice. The apparatus consisted of a square wooden arena measuring approximately 60 cm × 60 cm with walls 40 cm high, and the floor of the arena was divided into equal squares using black lines.

Each mouse was placed gently in the centre of the arena and allowed to explore freely for five minutes. During the observation period, the following parameters were recorded:

1. **Open Field Line Crossings** – the number of times the animal crossed from one square to another with all four paws.
2. **Central Square Time (seconds)** – the amount of time spent by the animal in the central squares of the arena.

Rodents naturally prefer to remain close to the walls of the arena (thigmotaxis). Therefore, reduced line crossings and reduced time spent in the central area are considered indicators of increased anxiety-like behaviour, whereas increased exploration of the central area indicates reduced anxiety.

The apparatus was cleaned with 70% ethanol between each trial to eliminate olfactory cues that might influence the behaviour of subsequent animals.

Experiment 2: Scurry Speed Test (Assessment of Motor Function)

Motor function and locomotor performance were assessed using the Scurry Speed Test. The apparatus consisted of a straight track approximately 80 cm in length leading to a dark enclosed compartment at the end of the track.

Each mouse was placed at the starting point of the track and allowed to move toward the dark compartment, which served as a natural shelter. The time taken by the mouse to traverse the entire length of the track and reach the dark compartment was recorded using a stopwatch.

Each animal was given three trials, and the average traversal time was recorded for analysis. Increased traversal time indicated reduced locomotor activity or impaired motor coordination, whereas shorter traversal time reflected normal motor function and locomotor activity.

The track was cleaned between trials to prevent behavioural interference due to scent cues.

Statistical Analysis

The behavioural data obtained from the Open Field Test and Scurry Speed Test were expressed as median values with interquartile range (IQR). The normality of the data distribution was assessed using the Shapiro–Wilk test. As the data did not follow a normal distribution, non-parametric statistical tests were used for analysis.

Within-group comparisons between baseline and three-month measurements were performed using the Wilcoxon signed-rank test. Between-group comparisons among the control group and the different MSG-treated groups were conducted using the Kruskal–Wallis test. When a statistically significant difference was detected between groups, the Mann–Whitney U test was used for post-hoc pairwise comparisons.

All statistical analyses were performed using appropriate statistical software, and a p-value of less than 0.05 was considered statistically significant. The results were presented in tabular form to compare the effects of chronic MSG administration on anxiety-like behaviour and motor function in Swiss albino mice.

RESULTS

Baseline Characteristics of Experimental Groups

The baseline behavioural parameters recorded in the Open Field Test and Scurry Speed Test were comparable among all experimental groups. No statistically significant differences were observed between the control group and the MSG-treated groups before the initiation of the experimental intervention.

Group	Open Field Line Crossings Median (IQR)	Central Square Time (Sec) Median (IQR)	Scurry Speed Time (Sec) Median (IQR)

Control	45 (40–52)	18 (15–22)	4.5 (4–5)
MSG 40 Mg/Kg	44 (39–50)	17 (14–21)	4.6 (4.2–5.2)
MSG 60 Mg/Kg	43 (38–49)	17 (14–20)	4.7 (4.3–5.3)
MSG 80 Mg/Kg	42 (37–48)	16 (13–20)	4.8 (4.5–5.4)

Table 1. Baseline Characteristics of Behavioral Parameters

These findings indicate that locomotor activity and exploratory behavior were comparable across all groups at baseline.

Effect of MSG on Anxiety (Open Field Test)
Baseline vs Three-Month Comparison

Chronic administration of MSG resulted in a reduction in exploratory behaviour in the Open Field Test at the three-month time point. This reduction was reflected by a decrease in the number of line crossings and reduced time spent in the central squares of the open field arena.

Group	Parameter	Baseline Median (IQR)	3 Months Median (IQR)	Z	P Value
Control	Line Crossings	45 (40–52)	42 (38–48)	1.72	0.06
	Central Square Time	18 (15–22)	16 (14–20)	1.54	0.08
MSG 40 Mg/Kg	Line Crossings	44 (39–50)	36 (32–42)	2.04	0.04*
	Central Square Time	17 (14–21)	13 (11–16)	2.10	0.04*
MSG 60 Mg/Kg	Line Crossings	43 (38–49)	30 (26–36)	2.70	0.01*
	Central Square Time	17 (14–20)	9 (7–12)	2.68	0.01*
MSG 80 Mg/Kg	Line Crossings	42 (37–48)	29 (24–34)	2.63	0.01*
	Central Square Time	16 (13–20)	8 (6–11)	2.59	0.01*

Table 2. Effect of Chronic Msg Administration on the Open Field Test

*Statistically Significant Difference Noted Using The Wilcoxon Signed-Rank Test

The reduction in exploratory behaviour was most pronounced in the MSG 60 mg/kg and MSG 80 mg/kg groups, indicating increased anxiety-like behaviour following chronic MSG exposure.

Effect of MSG on Motor Function (Scurry Speed Test)

Motor function was evaluated using the Scurry Speed Test by measuring the time required for mice to traverse a straight track leading to a dark compartment.

Baseline vs Three-Month Comparison

A mild increase in traversal time was observed in MSG-treated groups following three months of administration; however, these changes did not reach statistical significance.

Group	Baseline Median (Sec)	3 Months Median (Sec)	Z	P Value
Control	4.5 (4–5)	4.7 (4.2–5.3)	0.94	0.33
MSG 40 Mg/Kg	4.6 (4.2–5.2)	5.0 (4.5–5.8)	1.06	0.29
MSG 60 Mg/Kg	4.7 (4.3–5.3)	5.3 (4.8–6.1)	1.24	0.21
MSG 80 Mg/Kg	4.8 (4.5–5.4)	5.4 (4.9–6.3)	1.30	0.19

Table 3. Effect of Chronic Msg Administration on Scurry Speed Test

These findings indicate that chronic MSG administration did not produce statistically significant impairment in locomotor activity or motor coordination as assessed by the Scurry Speed Test.

DISCUSSION

The present study evaluated the chronic effects of monosodium glutamate (MSG) administration on anxiety-like behaviour and motor function in Swiss albino mice using the Open Field Test and Scurry Speed Test. The results demonstrated that prolonged exposure to MSG produced a significant reduction in exploratory behaviour in the Open Field Test, indicating increased anxiety-like behaviour. In contrast, motor function assessed using the Scurry Speed Test did not show statistically

significant impairment across the experimental groups.

The Open Field Test is a well-established behavioural paradigm used to assess anxiety-like behaviour and locomotor exploration in rodents. Animals placed in a novel open arena generally exhibit exploratory behaviour characterised by increased line crossings and movement across different zones of the apparatus. Reduced exploration, particularly reduced entry into or time spent in the central area of the arena, is commonly interpreted as an indicator of anxiety-like behaviour.[8] In the present study, both parameters evaluated in the Open Field Test—namely line crossings and time spent in the central square—showed a significant reduction in the MSG-treated groups at the three-month time point. These findings suggest that chronic MSG administration resulted in behavioural changes consistent with increased anxiety.

The observed anxiogenic effect of MSG may be related to alterations in glutamatergic neurotransmission within the central nervous system. Glutamate is the primary excitatory neurotransmitter in the brain and plays a critical role in regulating neuronal communication, synaptic plasticity, learning, and memory. However, excessive activation of glutamate receptors can lead to excitotoxic neuronal damage.[9] Overstimulation of NMDA receptors increases intracellular calcium influx, which can trigger oxidative stress, mitochondrial dysfunction, and neuronal injury. Such neurobiological alterations may disrupt the normal functioning of brain regions involved in emotional regulation, including the hippocampus, amygdala, and prefrontal cortex. The dose-dependent pattern observed in the present study further supports the role of MSG in modulating behavioural responses. The most pronounced reduction in exploratory behaviour was observed in the MSG 60 mg/kg and MSG 80 mg/kg groups, suggesting that higher doses of MSG may produce greater neurobehavioural alterations. Similar dose-dependent behavioural effects have been reported in previous experimental studies investigating MSG exposure in rodents. Chronic MSG administration has been associated with increased oxidative stress and neuronal degeneration in brain regions responsible for behavioural regulation.[10] These structural and biochemical changes may contribute to disturbances in emotional behaviour.

Another possible mechanism underlying the behavioural alterations observed in this study involves oxidative stress and inflammatory processes in neural tissue. Experimental evidence suggests that MSG administration can increase lipid peroxidation and decrease antioxidant enzyme activity in the brain.[11] Oxidative stress can impair neuronal signalling and synaptic transmission, which may contribute to behavioural abnormalities, including anxiety-like responses. Furthermore, alterations in glutamatergic signalling pathways have been implicated in the pathophysiology of anxiety disorders and other neuropsychiatric conditions.[12]

Despite the significant changes observed in anxiety-related behaviour, the Scurry Speed Test did not reveal statistically significant differences in locomotor performance between groups. The Scurry Speed Test measures the time required for rodents to traverse a straight track leading to a dark compartment and is commonly used to evaluate locomotor activity and motor coordination. The absence of significant changes in traversal time suggests that chronic MSG exposure at the doses used in the present study did not markedly impair motor function.

These findings are important because behavioural tests used to evaluate anxiety-like behaviour require intact motor function for accurate interpretation. If locomotor impairment were present, reduced exploration in the Open Field Test could be attributed to motor deficits rather than emotional changes. The preservation of locomotor activity observed in the present study, therefore, indicates that the behavioural changes detected in the Open Field Test are more likely related to alterations in emotional behaviour rather than motor impairment.[13]

The preservation of motor function may also suggest that the neural circuits responsible for locomotor control were not significantly affected by the administered doses of MSG. Motor coordination primarily depends on the integrity of cerebellar and basal ganglia pathways, whereas anxiety-related behaviour is largely regulated by structures within the limbic system, including the amygdala and hippocampus.[14] Disruption of neurotransmission within limbic circuits may therefore result in anxiety-like behaviour without producing substantial changes in locomotor activity.

In addition to functional changes, chronic MSG exposure has also been associated with structural alterations in neural tissue. Histopathological studies have reported

neuronal degeneration and astrocytic activation in various brain regions following MSG administration.[15] Increased expression of glial fibrillary acidic protein (GFAP), a marker of astrocyte activation, has been observed in experimental models of glutamate-induced neurotoxicity. Astrocytic activation may alter neuronal signalling and contribute to the behavioural abnormalities observed following chronic MSG exposure.

Overall, the findings of the present study suggest that chronic MSG administration may influence emotional behaviour through mechanisms involving glutamatergic excitotoxicity and oxidative stress. The observed reduction in exploratory behaviour in the Open Field Test indicates increased anxiety-like behaviour following prolonged MSG exposure. However, the absence of significant motor impairment in the Scurry Speed Test suggests that locomotor function remained largely preserved at the doses used in this study. Further investigations involving neurochemical analysis, oxidative stress markers, and histopathological evaluation of brain tissue are required to better understand the mechanisms underlying MSG-induced behavioural alterations.

CONCLUSION

Chronic administration of monosodium glutamate (MSG) produced dose-dependent anxiety-like behavioural changes in Swiss albino mice as evidenced by reduced exploratory behaviour in the Open Field Test. However, motor function assessed using the Scurry Speed Test did not show significant impairment. These findings suggest that prolonged MSG exposure may influence emotional behaviour without markedly affecting locomotor activity.

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