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EVALUATION OF ANTIDEPRESSANT EFFECTS OF ZINC ACETATE IN MICE

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ABSTRACT: Background: Depression is a debilitating mental health disorder that causes changes in physical activity, appetite, sleep and weight. Many studies shown that zinc may have an important role in the pathogenesis of depression. In this study we aimed to evaluate the antidepressant effects of Zinc acetate in mice and to investigate the synergistic effect of zinc acetate with fluoxetine, a standard antidepressant. **Methods:** After getting approval from ethics committee, 36 male swiss albino mice weighing 20-30 gm were randomly allocated into 6 groups with 6 in each group. First group received normal saline (control), second group received fluoxetine 10 mg/kg (standard). Third, fourth and fifth group received Zinc acetate 10 mg/kg, 20 mg/kg and 40 mg/kg respectively. Sixth group received fluoxetine 10 mg/kg with zinc acetate 10 mg/kg. All the drugs were given through oral route. The effects of the drugs were assessed by recording the immobility time in Tail Suspension Test (TST) and the results were analysed by one way ANOVA followed by Bonferroni post hoc test. **Results:** The mean immobility time for Control is 236 ± 14 seconds which is significantly higher than that of the Standard Fluoxetine 10 mg/kg group 155 ± 13 seconds ($p < 0.001$). The mean immobility time for zinc acetate 10 mg/kg, 20 mg/kg and 40 mg/kg group were 226±9 seconds, 219±11 seconds and 196 ± 9 seconds respectively. The mean values of these groups were not statistically lower when compared to that of control group. The zinc acetate 40 mg/kg group did not differ significantly from either the control ($p=0.228$) or fluoxetine ($p=0.206$), reflecting an intermediate numerical trend. **Conclusion:** From this study, a numerical dose dependent trend toward reduced immobility time was observed with zinc acetate, most pronounced at 40 mg/kg and warrants further investigation at higher doses or with chronic administration. Also, there is no additional benefit of Zinc acetate over Fluoxetine at the tested dose.

INTRODUCTION: Depression is a complex and debilitating mental health disorder that affects millions of people worldwide and represents a major public health concern.

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It is characterized by persistent sadness, loss of interest or pleasure in daily activities, disturbed sleep, altered appetite, reduced energy levels, and impaired cognitive function.

The pathophysiology of depression is multifactorial and involves several biological mechanisms, including dysregulation of neurotransmitter systems, impaired synaptic plasticity, altered neurotrophic signaling, and increased oxidative stress [1].

These mechanisms contribute to structural and functional changes in brain regions involved in mood regulation, such as the hippocampus, prefrontal cortex, and limbic system.

Conventional antidepressant drugs, including selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs), remain the mainstay of treatment for depression. However, the therapeutic response to these agents varies among individuals, and some patients may experience adverse side effects that limit their clinical utility [2]. Moreover, currently available therapeutic strategies primarily target monoaminergic neurotransmission and do not fully address the multifactorial nature of depression. Therefore, there is a growing need to explore novel therapeutic approaches that target multiple mechanisms involved in the pathogenesis of the disorder. In this context, strategies that incorporate antioxidant mechanisms appear particularly promising [3].

In recent years, increasing attention has been directed toward the role of micronutrients, particularly zinc, in the pathophysiology and treatment of depression. Zinc is an essential trace element involved in numerous physiological processes in the central nervous system, including neurotransmission, neurogenesis, synaptic plasticity, and antioxidant defense mechanisms. It is abundantly present in the brain, particularly in presynaptic vesicles of glutamatergic neurons located in regions such as the cerebral cortex, hippocampus, and amygdala. Upon neuronal activation, zinc is released into the synaptic cleft, where it modulates the activity of several receptors, including NMDA receptors and monoaminergic receptors, thereby influencing neuronal signaling and synaptic plasticity [4, 5].

Clinical and experimental studies have reported altered zinc levels in individuals with depression, suggesting a potential association between zinc deficiency and depressive symptoms. Furthermore, zinc supplementation has demonstrated promising antidepressant-like effects in both preclinical and clinical studies and has been shown to enhance the efficacy of conventional antidepressant drugs when used as adjunctive therapy. Recent studies have identified GPR39, a G-protein-coupled receptor activated by zinc, which is predominantly located on the postsynaptic membrane of neurons in the CA3 region of the hippocampus. Activation of GPR39 stimulates cAMP response element-dependent gene transcription, leading to an increase in the synthesis of brain-derived neurotrophic factor (BDNF), a key molecule involved in neuronal survival, synaptic plasticity, and antidepressant effects [6]. Given this evidence, the present study was undertaken to evaluate the antidepressant potential of zinc acetate in mice at different doses, and to assess its interaction with fluoxetine.

Aim: To evaluate the antidepressant-like activity of Zinc acetate in Swiss albino mice and to investigate its potential synergistic interaction with Fluoxetine using the Tail Suspension Test.

Objectives:

1. To assess the antidepressant-like activity of Zinc acetate at three dose levels (10, 20, and 40 mg/kg) in Swiss albino mice using the Tail Suspension Test (TST).
2. To compare the antidepressant-like effect of zinc acetate at the effective dose with that of the standard antidepressant Fluoxetine (10 mg/kg).
3. To evaluate the effect of combining a subtherapeutic dose of Zinc acetate (10 mg/kg) with fluoxetine (10 mg/kg) on immobility time in the TST, to assess any synergistic or additive interaction.

MATERIALS AND METHODS:

Animal Care and Housing: A total of 36 male Swiss albino mice weighing 20–30 g were sourced from TANUVAS, Madhavaram, Chennai and kept at the Animal house of our college. Animals were housed in standard polypropylene cages with 5 per cage. Paddy husk was used for bedding and were changed every 48 hours. Temperature maintained at 24°C and humidity at 50 to 60%. They were provided with standard pellet diet and water *ad libitum*. A 12-hour light–dark cycle was maintained for 1 week before the initiation of the experimental procedures. Animals were fasted for 4 hours before the procedure with free access to water. The study was approved by the Institutional Animal Ethics Committee with approval

no.99/GKMC/IAEC/2018 and conducted in accordance with CPCSEA guidelines. All animals were acclimatized to laboratory conditions for one week.

Study Design: On the day of the experiment, animals were allowed to adapt to the experimental environment for 1–2 hours prior to the procedure. Then animals were randomly allocated into six groups, with six in each [7]. Group I (control) received normal saline orally (p.o.), while Group II (standard) was administered with fluoxetine at a dose of 10 mg/kg. Groups III, IV, and V received zinc acetate orally at doses of 10 mg/kg, 20 mg/kg and 40 mg/kg respectively. Group VI was treated with zinc acetate 10 mg/kg along with fluoxetine 10 mg/kg as shown in **Table 1**. The dose of Zinc acetate was selected based on the study by Rosa *et al.* (2003) [8]. Rosa *et al* have used Zinc chloride 10-30 mg/kg through intra peritoneal route whereas we have chosen Zinc acetate 10-40 mg/kg per oral.

TABLE 1: TREATMENT GIVEN FOR EACH GROUP

Group	Number of mice	Drug	Dose
Group I	6	Normal saline	10ml/kg
Group II	6	Fluoxetine	10 mg/kg
Group III	6	Zinc acetate	10 mg/kg
Group IV	6	Zinc acetate	20 mg/kg
Group V	6	Zinc acetate	40 mg/kg
Group VI	6	Zinc acetate and Fluoxetine	10 mg/kg each

Preparation of Drugs: Zinc acetate is available as 50 mg tablet, procured from Zuventus Healthcare Ltd (Mumbai, Maharashtra) which was crushed using pill grinder and dissolved in normal saline. Fluoxetine is available as 10 mg capsule, procured from Cadila Pharmaceuticals Ltd (Ankleshwar, Gujarat) which was dissolved in normal saline. Drug solutions were prepared just before the procedure and were administered orally using an appropriate gauge gavage needle and syringe. All groups received equal volumes of 10 ml/kg to eliminate volume related confounders.

Experimental Procedure: In accordance with the ethics guidelines for experimentation on animals, animals were randomly allocated using computer generated random numbers into 6 groups with 6 animals in each. Random allocation was done by one investigator, and the experiments were carried out by the other.

Assessment with Tail suspension Method: The Tail Suspension Test (TST), originally described by Steru *et al.* (1985) [9], is a widely used and validated experimental model for assessing antidepressant activity.

It is based on the principle that when rodents are exposed to short-term, unavoidable stress, they initially exhibit escape-oriented movements followed by a period of immobility, which is considered a behavioral correlate of depressive-like states. In this procedure, each mouse is suspended 35 cm above the surface of a table using adhesive tape placed approximately 2 cm from the tip of the tail. The tape is attached to a horizontal rod supported by two metallic stands as shown in **Fig. 1**.

The total duration of immobility is recorded in seconds over a 5-minute observation period. Immobility is defined as the absence of active escape-directed movements reflecting a state in which the animal ceases attempts to struggle. Passive swinging or pendular motion of the suspended animal was not counted as immobility.

Any mouse that climbed its own tail was excluded from analysis and replaced. The apparatus was thoroughly cleaned with 70% ethanol and allowed to air dry between successive animals to eliminate olfactory cues. Each mouse is tested individually and only once, ensuring that it remains out of sight of other animals during the assessment. All testing sessions were conducted between 10:00 and 14:00 hours to minimize circadian variation.

A decrease in immobility time is indicative of antidepressant-like activity and correlates well with clinically effective antidepressant agents. Thirty minutes after oral drug administration [10], mice were subjected to the Tail Suspension Test. Immobility time was recorded over 5 minutes, and the data obtained were entered in an Excel sheet.

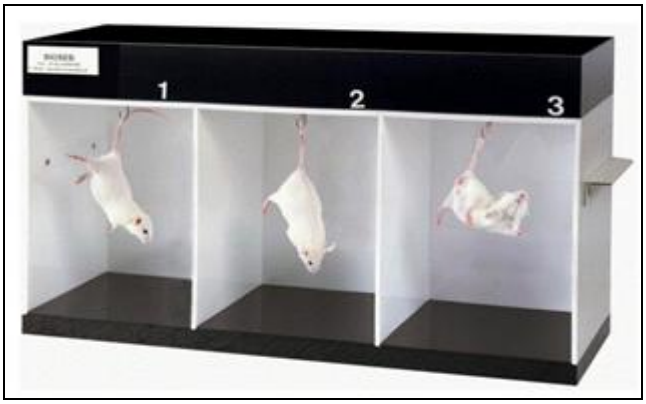

FIG. 1: TAIL SUSPENSION TEST

Statistical Analysis: Statistical analysis was carried out by mean±standard error of mean (SEM) for quantitative variables. Immobility time in Tail Suspension test was considered as primary outcome. Comparison between the groups were performed using one way ANOVA followed by Bonferroni post hoc test and p value <0.05 was considered significant. All analyses were done using SPSS software version 20.0 (IBM Corp) for windows.

RESULTS: In this study, 36 Swiss albino mice were evaluated for Antidepressant effect using Tail Suspension test. The immobility time in seconds was recorded during the five-minute period. The mean values of immobility time were given in **Table 2**.

TABLE 2: MEAN IMMOBILITY TIME IN TAIL SUSPENSION TEST (N=6 PER GROUP)

Group	Treatment	Mean±SEM (Seconds)
I	Control group (Normal saline 10 ml/kg)	236±14
II	Standard -Fluoxetine 10 mg/kg	155±13
III	Zinc acetate 10 mg/kg	226±9
IV	Zinc acetate 20 mg/kg	219±11
V	Zinc acetate 40 mg/kg	196±9
VI	Zinc acetate 10 mg/kg and Fluoxetine 10 mg/kg	164±9

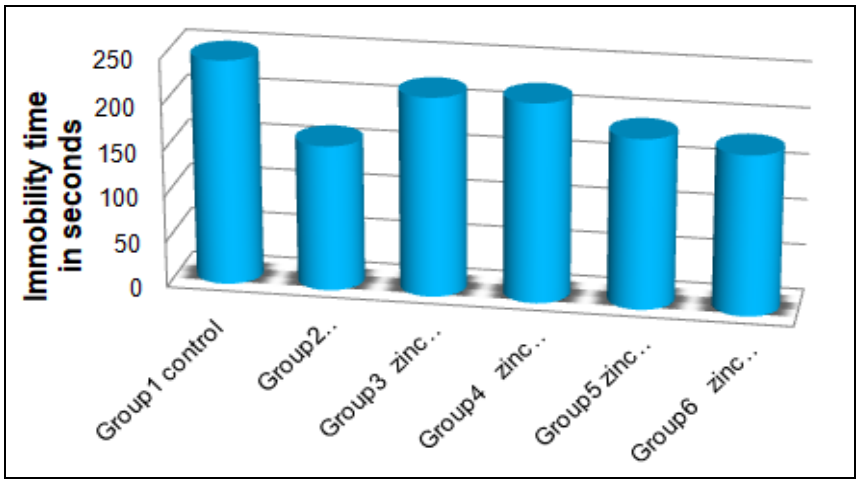

FIG. 2: RESULT OF TAIL SUSPENSION TEST SHOWING MEAN OF IMMOBILITY TIME

Effect of Zinc Acetate and Fluoxetine on Immobility Time in the TST: The mean immobility time (seconds) for all groups are presented in **Table 2** and illustrated in **Fig.2**. Homogeneity of variances across groups was confirmed by Levene's test (F = 0.601, p = 0.700), satisfying the assumption for one-way ANOVA.

One-way ANOVA revealed a statistically significant overall difference in immobility time among the six groups [$F(5, 30) = 9.314$, $p < 0.001$, $\eta^2 = 0.608$, indicating a large effect size] and it is presented in **Table 3.**

TABLE 3: ONE WAY ANOVA – IMMOBILITY TIME IN SECONDS

Source of variation	Sum of squares	df	Mean square	F	p value
Between groups	33,988.889	5	6,797.778	9.314	< 0.001
Within groups	21,895.667	30	729.856	—	—
Total	55,884.556	35	—	—	—

Overall: $F(5, 30) = 9.314$, $p < 0.001$. Effect size: $\eta^2 = 0.608$ (large; 95% CI: 0.281–0.700); $\varepsilon^2 = 0.543$; $\omega^2 = 0.536$.

Bonferroni post hoc analysis showed that standard group (Fluoxetine 10 mg/kg; 155 ± 13 s) showed a significantly lower immobility time compared to control (236 ± 14 s; $p < 0.001$). Administration of zinc acetate at doses of 10 mg/kg (226 ± 9 s; $p = 1.000$), 20 mg/kg (219 ± 11 s; $p = 1.000$), and 40 mg/kg (196 ± 9 s; $p = 0.228$) did not produce a statistically significant reduction in immobility time compared to the control group after Bonferroni correction. Although a numerical trend toward reduced immobility was observed with increasing doses of zinc acetate, none of the zinc acetate treated groups reached statistical significance versus control.

When compared with the standard (fluoxetine 10 mg/kg), the zinc acetate 10 mg/kg group ($p = 0.001$) and zinc acetate 20 mg/kg group ($p = 0.004$) showed significantly higher immobility times, indicating inferior antidepressant-like activity. The zinc acetate 40 mg/kg group did not differ significantly from either control ($p=0.228$) or fluoxetine 10 mg/kg group ($p = 0.206$), reflecting an intermediate numerical trend.

Effect of Combination Group on Immobility Time in the TST: The combination group (Zinc acetate 10 mg/kg + Fluoxetine 10 mg/kg; 164 ± 9 s) showed a significantly lower immobility time compared to control ($p = 0.001$). However, the combination group did not differ significantly from fluoxetine alone (155 ± 13 s; $p = 1.000$), indicating that the addition of zinc acetate 10 mg/kg to fluoxetine 10 mg/kg did not produce any additive or synergistic reduction in immobility time. The p values for all comparisons are presented in **Table 4.**

TABLE 4: BONFERRONI POST HOC TEST — PAIRWISE COMPARISONS

Comparison	Mean difference (seconds)	95% Confidence Interval	Bonferroni p value	Significance
Control vs Fluoxetine 10 mg/kg (Standard)	81.00	31 to 130	< 0.001	Significant *
Control vs Zinc acetate 10 mg/kg	10.17	-40 to 60	1.000	NS
Control vs Zinc acetate 20 mg/kg	17.17	-33 to 67	1.000	NS
Control vs Zinc acetate 40 mg/kg	40.17	-10 to 90	0.228	NS
Control vs Zinc 10 mg/kg + Fluoxetine 10 mg/kg	72.17	22 to 122	0.001	Significant *
Fluoxetine 10 mg/kg vs Zinc acetate 10 mg/kg	-70.83	-121 to -21	0.001	Significant *
Fluoxetine 10 mg/kg vs Zinc acetate 20 mg/kg	-63.83	-114 to -14	0.004	Significant *
Fluoxetine 10 mg/kg vs Zinc acetate 40 mg/kg	-40.83	-91 to 9	0.206	NS
Fluoxetine 10 mg/kg vs Zinc 10 mg/kg + Fluoxetine 10 mg/kg	-8.83	-59 to 41	1.000	NS
Zinc acetate 40 mg/kg vs Zinc 10 mg/kg + Fluoxetine 10 mg/kg	32.00	-18 to 82	0.735	NS

* Significant at $p < 0.05$ after Bonferroni correction for multiple comparisons. NS = Not significant.

DISCUSSION: Zinc is an essential trace element involved in numerous biological functions. In adults, the total body zinc content is approximately 2–3 g, with the majority stored in skeletal muscle and bone, and only about 0.1% is present in plasma [11]. In the brain, less than 5% of total zinc is localized within synaptic vesicles of zinc-containing neuronal terminals. The concentration of vesicular zinc in the hippocampal mossy fiber's ranges from 300 to 350 μM. This vesicular zinc is believed to participate in synaptic neurotransmission and functions as an endogenous neuromodulator in the mammalian brain [12].

Several studies have reported that there is reduced serum zinc levels in patients with depression, particularly in those resistant to treatment. Other investigations have shown that serum zinc concentrations normalize during antidepressant therapy [13]. Additionally, a significant correlation has been observed between serum zinc levels and the severity of depressive symptoms. Elham Ranjbar *et al.* reported that the mean dietary zinc intake among patients with major depression was approximately 6.5–7 mg/day, which is considerably lower than the intake reported by Ghasemi *et al.*, who found average daily zinc consumption in healthy men and women to be 15.8 ± 11 mg and 14.7 ± 11 mg, respectively [14, 15]. These findings collectively suggest that zinc may play a meaningful role in the management of depression.

The Tail Suspension Test (TST) is commonly employed to evaluate the antidepressant potential

of novel compounds. It is sensitive to major classes of antidepressants, including tricyclic antidepressants, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, and atypical agents, and serves as a valuable tool for exploring the neurobiological mechanisms underlying antidepressant responses. Effective antidepressant treatments are associated with reduced immobility time in the TST [9].

Multiple studies have implicated zinc in both the pathophysiology and treatment of depression. Rosa *et al.* demonstrated that zinc chloride administered intraperitoneally at doses of 10 and 30 mg/kg produced antidepressant-like effects [8]. Similarly, Cunha MP *et al.* reported that systemic oral administration of $ZnCl_2$ (10 and 30 mg/kg) significantly reduced immobility time in the TST, findings consistent with earlier observations by Steru *et al* [9, 16]. In contrast, our study showed that zinc acetate produced some antidepressant-like activity only at a dose of 40 mg/kg, whereas doses of 10 mg/kg and 20 mg/kg were ineffective. These results indicate that zinc acetate, when given as a single dose, is not effective at doses below 40 mg/kg. Notably, at 40 mg/kg, it did not differ significantly from fluoxetine, but the difference from the control is also not statistically significant. The dose we used remained well below the reported LD_{50} of zinc acetate (287 mg/kg in mice, as reported by Domingo *et al*.), suggesting a favorable safety margin [17].

Janko *et al.*, in a study evaluating the anxiolytic and antidepressant effects of zinc in rats, found that zinc histidine dehydrates produced significant antidepressant effects at doses of 10 mg/kg and 20 mg/kg without affecting anxiety, whereas a dose of 30 mg/kg exhibited both antidepressant and anxiogenic properties [18]. In the present study, the anxiolytic effects of zinc were not assessed.

Synergistic interactions between zinc and various antidepressants have been demonstrated in several studies. Sowa-kucma *et al.* observed enhanced antidepressant responses when zinc chloride was combined with different classes of antidepressants [19]. Similarly, Kroczka *et al.* and Szewczyk *et al.* reported synergistic effects when zinc was co-administered intraperitoneally with imipramine in the mouse Forced Swim Test (FST) [20, 21].

Furthermore, Cieślik *et al.* showed that co-administration of zinc and imipramine, each at subeffective doses, produced antidepressant-like effects in a chronic unpredictable stress model of depression [22]. However, in our study, there is no additional benefit over Fluoxetine at the tested dose.

Limitations: The major limitation of the study is, only the TST was employed as the behavioral model; corroboration with a second validated model such as the Forced Swim Test would strengthen the conclusions. Second, the drugs were administered as a single dose, which limits the translational relevance of the findings given the chronic nature of clinical depression. Third, the study did not include an open field test to assess locomotor activity, which is necessary to rule out non-specific motor stimulation as a potential confounder for the observed reduction in immobility. Finally, the study was not blinded, which may introduce observer bias in the immobility time measurements.

CONCLUSION: Based on the findings of our study, a numerical dose dependent trend toward reduced immobility time was observed with zinc acetate, most pronounced at 40 mg/kg: however, none of the zinc acetate group reached statistical significance versus control. Zinc acetate 40 mg/kg demonstrated an antidepressant-like effect that did not differ significantly from Fluoxetine suggesting a trend that warrants further investigation with adequate sample size, chronic dosing, different behavioral model and motor function assessment.

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CONFLICTS OF INTEREST: The authors declare no conflicts of interest.

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