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Pharmacological and Toxicological Evaluation of 5-(p-Aminophenyl)-1,3,4-oxadiazole-2-thione Potassium Salt: Acute Toxicity and Anxiolytic Activity in Experimental Models

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Abstract Anxiety disorders are among the most prevalent neuropsychiatric conditions worldwide, affecting a significant proportion of the population and substantially impairing quality of life. Although benzodiazepines and other anxiolytic agents are widely used in clinical practice, adverse effects such as sedation, tolerance, dependence, and withdrawal symptoms limit their therapeutic application. Therefore, the search for novel anxiolytic compounds with improved safety and efficacy profiles remains an important objective in modern pharmacology. Heterocyclic compounds containing the 1,3,4-oxadiazole ring have attracted considerable interest due to their broad spectrum of biological activities and potential central nervous system effects.

Keywords Heterocyclic compounds, 1,3,4-oxadiazole, Anxiolytic effect, Behavioural models, Acute toxicity, Experimental pharmacology

1. Introduction

Anxiety disorders represent one of the most prevalent groups of neuropsychiatric conditions worldwide and constitute a significant medical and social problem. According to epidemiological studies, anxiety-related disorders affect a substantial proportion of the global population and are associated with impaired cognitive function, decreased productivity, and reduced quality of life [1-3]. Current anxiolytic drugs are effective in reducing symptoms of anxiety; however, their clinical use is frequently limited by significant adverse effects. Benzodiazepines, although widely prescribed, may cause sedation, cognitive impairment, tolerance, dependence, and withdrawal symptoms after prolonged administration. Selective serotonin reuptake inhibitors and serotonin–norepinephrine reuptake inhibitors have a delayed onset of action and are often associated with gastrointestinal disturbances, insomnia, and sexual dysfunction. Barbiturates produce marked central nervous system depression and possess a high risk of respiratory suppression and toxicity. Therefore, the development of novel anxiolytic agents with improved efficacy and safety remains an important objective in modern pharmacology [2,17].

Drug group	Representative drugs	Main limitation and adverse effects
Benzodiazepines	Diazepam, Alprazolam	Sedation, tolerance, dependence, memory impairment
SSRIs/SNRIs	Fluoxetine, Sertraline, Venlafaxine	Delayed onset of action, insomnia, nausea, sexual dysfunction
Barbiturates	Phenobarbital, Thiopental	Marked CNS depression, respiratory suppression, toxicity
Azapirones	Buspirone	Slow onset of effect, dizziness, limited efficacy in severe anxiety

"These limitations justify the search for novel anxiolytic agents with improved safety profiles."

In recent years, heterocyclic compounds have attracted considerable attention due to their diverse biological activities and favourable pharmacodynamic characteristics. The 1,3,4-oxadiazole ring system is known to confer a wide spectrum of pharmacological effects, including antimicrobial, anti-inflammatory, anticonvulsant, antidepressant, and central

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nervous system–modulating activities. Structural modification of this heterocyclic nucleus allows the design of molecules with enhanced lipophilicity, metabolic stability, and the ability to penetrate the blood–brain barrier [4-6].

Several experimental studies have suggested that 1,3,4-oxadiazole derivatives may exhibit potential anxiolytic properties while demonstrating reduced sedative effects compared to classical benzodiazepines. However, the pharmacological and toxicological characteristics of many newly synthesized oxadiazole derivatives remain insufficiently investigated, particularly regarding their safety profile and behavioural effects. These findings indicate that the 1,3,4-oxadiazole scaffold represents a promising platform for the development of safer anxiolytic agents [4-6].

In this context, the present study focuses on 5-(p-amino phenyl)-1,3,4-oxadiazole-2-thione potassium salt, a heterocyclic compound with a potentially promising neuropharmacological profile. The selection of 5-(p-aminophenyl)-1,3,4-oxadiazole-2-thione potassium salt (D-360) for the present study was based on several structural considerations. The p-aminophenyl substituent may enhance the affinity of the molecule toward central nervous system targets involved in anxiety regulation. In addition, the presence of the 2-thione group is known to increase the pharmacological activity of heterocyclic compounds through stronger interactions with receptor binding sites. Transformation of the compound into its potassium salt form was expected to improve water solubility, bioavailability, and consequently its pharmacological potential [19-22].

Despite the growing interest in oxadiazole-containing compounds, the pharmacological profile of D-360 has not yet been sufficiently investigated. To the best of our knowledge, no previous study has comprehensively evaluated both the acute toxicity and anxiolytic activity of this compound in vivo. Therefore, the present work aimed to determine the acute toxicity of the compound and to investigate its anxiolytic activity using established experimental models.

2. Materials and Methods

Experimental Animals

The study was conducted on adult white laboratory mice weighing 20–25 grams. The animals were housed under standard laboratory conditions, with free access to food and water, and maintained on a 12-hour light/dark cycle. All experimental procedures were conducted in accordance with internationally accepted principles for the care and use of laboratory animals.

3. Experimental Design

The animals were randomly divided into experimental groups (n = 10 per group). The study included:

- Control group (received physiological saline)
- Est compound groups administered at doses ranging from 0.1 to 60 mg/kg
- Reference drug groups (two dose levels)

The compound D-360 was administered orally which dissolve with Twin solution. The reference drug was administered at therapeutically relevant doses. The compound 5-(p-amino phenyl)-1,3,4-oxadiazole-2-thione potassium salt, synthesized in our laboratory, was used in the present study and is hereafter designated as D-360.

Acute Toxicity Study - acute toxicity was evaluated by determining the median lethal dose (LD_{50}) following single-dose administration. Animals were observed for behavioural changes, signs of intoxication, and mortality during the first 24 hours and for an additional observation period. LD_{50} values were calculated using standard statistical methods [14,15].

Behavioural Studies

Open Field Test - the Open Field test was used to assess locomotor and exploratory activity. The number of crossed squares, rearing episodes, and grooming behaviour were recorded during the observation period [8,16].

Elevated Plus Maze (EPM) Test

Anxiolytic activity was evaluated using the Elevated Plus Maze model. The following parameters were recorded: time spent in open arms, number of entries into open and closed arms, and total locomotor activity. An increase in time spent in open arms was interpreted as an anxiolytic-like effect [7].

Statistical Analysis

Data were expressed as mean ± standard error of the mean (M ± SEM). Statistical analysis was performed using Student's t-test. Differences were considered statistically significant at $P < 0.05$.

4. Results

Determination of acute toxicity (LD_{50}) by the Kerber method. Acute toxicity was assessed after a single administration of the test compound at graded dose levels in mice (n = 10 per group). Mortality was recorded during the first 24 h (and throughout the observation period, if applicable). The median lethal dose (LD_{50}) was calculated using the Kerber method that was 1975 mg/kg. The compound demonstrated moderate acute toxicity according to standard toxicity classification criteria.

Open Field Test

In the Open Field test, administration of the test compound produced dose-dependent changes in behavioural activity. In the Open Field test, D-360 at moderate doses (1–10 mg/kg) increased exploratory and locomotor activity, as evidenced by a higher number of crossed squares and rearing episodes, while reducing the number of defecation acts. These changes indicate an anxiolytic-like effect. Importantly, locomotor activity was not markedly suppressed, suggesting that the compound did not produce significant sedative effects. The

most pronounced activity was observed at doses between 1 and 10 mg/kg, whereas higher doses were less effective.

(Table 1. Effects of the compound on behavioural parameters in the Open Field test).

D-360 produced dose-dependent changes in locomotor and exploratory activity in the Open Field test. Statistically significant differences were observed at several dose levels compared to the control group ($p \leq 0.05$).

Elevated Plus Maze (EPM) Test

Effects of D-360 on behavioural parameters in the Elevated Plus Maze test is represented Table 2.

In the control group, the time spent in the light chamber was 53.6 ± 8.89 s, while the time spent in the dark chamber was 126.4 ± 8.89 s. The duration of stay in the central zone was 19.4 ± 3.5 s, the number of transitions was 2.2 ± 0.42, and the anxiety index (K index) was 0.42.

Administration of 0.1 mg/kg of the test compound did not markedly change the distribution between chambers, with light chamber time of 53.5 ± 3.73 s and dark chamber time of 126.5 ± 3.73 s. The K index remained at 0.42. At 0.5 mg/kg, the time spent in the light chamber increased to 62.1 ± 5.58 s, while dark chamber time decreased to 117.9 ± 5.58 s. The K index increased to 0.53. Administration of 1.0 mg/kg resulted in a more pronounced increase in light chamber time (80.0 ± 3.86 s) and reduction in dark chamber time (100.0 ± 3.86 s), with a K index of 0.80. At 5.0 mg/kg, light chamber time further increased to 83.5 ± 4.01 s and dark chamber time decreased to 96.5 ± 4.01 s. The K index reached 0.87. The 10.0 mg/kg dose produced light chamber time of 75.6 ± 17.03 s and dark chamber time of 104.4 ± 17.03 s, with a K index of 0.72. At 30.0 mg/kg, light chamber time was 56.5 ± 3.41 s, while dark chamber time was 123.5 ± 3.41 s, and the K index decreased to 0.46. The highest dose (60.0 mg/kg) showed light chamber time of 58.4 ± 6.27 s and dark chamber time of 121.6 ± 6.27 s, with a K index of 0.48.

The reference drug diazepam demonstrated a marked increase in light chamber time at both tested doses (136.6 ± 2.96 s and 185.5 ± 7.7 s), with corresponding K index values of 0.836 and 1.62, respectively.

Effect of D-360 on Anxiety-like Behaviour in the Pentylenetetrazol Model

The results represented table 3.

In the control group treated with pentylenetetrazol (20 mg/kg), the time spent in the light compartment was 37.0 ± 5.44 s, while the time spent in the dark compartment was 83.0 ± 5.44 s. The K index (light/dark ratio) was 0.45.

Table 1. Assessment of the anxiolytic activity of D-360 substance in the Open field test

Groups and Treatments	Dose (mg/kg)	Locomotor Activity	Exploratory Activity	Number of Defecation Acts	Effect (%) Compared to Control		
Control Group	Distilled Water	7.4 ±0.62	4.8 ±0.59	8.0 ±0.33	Locomotor Activity	Exploratory Activity	Number of Defecation Acts
D-360	0,1	6.5 ±0.50*	5.3 ±0.42*	8.1 ±0.53	-12,16	10,42	1,25
	0,5	6.3 ±0.76*	5.5 ±0.40*	5.8 ±0.39*	-14,86	14,58	-27,5
	1.0	8.4 ±0.69*	5.3 ±0.42*	5.1 ±0.31*	13,51	10,42	-36,25
	5.0	6.9 ±0.60*	5.8 ±0.47*	6.1 ±0.53*	-6,76	20,83	-23,75
	10.0	8.0 ±0.67*	6.2 ±0.55*	6.3 ±0.45*	8,11	29,17	-21,25
	30.0	8.4 ±0.73*	4.2 ±0.25	6.2 ±0.55*	13,51	-12,5	-22,5
	60.0	6.6 ±0.58*	5.6 ±0.75*	7.2 ±0.53*	-10,81	16,67	-10.0

*Values are expressed as M ± SEM. $p \leq 0.05$ vs. control group.

Table 2. Assessment of the anxiolytic activity of D-360 substance in the Elevated plus maze

Groups and Treatments	Dose (mg/kg)	Time Spent in Light Chamber (s)	Time Spent in Dark Chamber (s)	Time Spent in Central Zone (s)	Number of Transitions	Anxiety Index (K index)
Control Group		53.6 ±8.89	126.4 ±8.89	19.4 ±3.5	2.2 ±0.42	0.42
D-360	0.1	53.5 ±3.73*	126.5 ±3.73*	19.4 ±3.5*	1.5 ±0.34*	0.42
	0.5	62.1 ±5.58*	117.9 ±5.58*	14.5 ±3.29*	2.2 ±0.29*	0.53
	1.0	80.0 ±3.86*	100.0 ±3.86*	13.0 ±2.60*	1.6 ±0.4*	0.80
	5.0	83.5 ±4.01*	96.5 ±4.01*	11.7 ±2.89*	1.7 ±0.26*	0.87
	10.0	75.6 ±17.03*	104.4 ±17.03*	18.0 ±4.26*	1.2 ±0.25*	0.72
	30.0	56.5 ±3.41*	123.5 ±3.41*	11.2 ±3.85*	1.0 ±0.45*	0.46
	60.0	58.4 ±6.27*	121.6 ±6.27*	32.6 ±3.99*	2.6 ±0.27*	0.48
Diazepam	0.5	136.6 ±2.96*	163.4 ±2.96*	10.0 ±0.39*	2.2 ±0.29*	0.836
	1.0	185.5 ±7.7*	114.5 ±7.7*	15.5 ±1.57*	1.4 ±0.16*	1.62

*Values are expressed as M ± SEM. $p \leq 0.05$ vs. control group.

Table 3. Assessment of the anxiolytic activity of D-360 on Anxiety-like Behaviour in the Pentylenetetrazol Model

Experimental Groups	Dose (mg/kg)	Time Spent in Light Compartment (s)	Time Spent in Dark Compartment (s)	K Index (Light/Dark Ratio)
Control Group (Pentylenetetrazol, 20 mg/kg)	20	37.0 ±5.44	83.0 ±5.44	0.45
D-360	60.0	47.0 ±8.5*	73.0 ±8.5*	0.64
	30.0	56.0 ±5.1*	64.0 ±5.1*	0.88
	10.0	59.0 ±6.23*	61.0 ±6.23*	0.97
	5.0	63.0±2.86*	57.0±2.86*	1.10
	1.0	74.0±3.64*	46.0±3.64*	1.61
	0.5	70.0±4.59*	50.0±4.59*	1.40
	0.1	58.0±3.89*	62.0±3.89*	0.94

*Values are expressed as M ± SEM. $p \leq 0.05$ vs. control group.

Table 4. Effect of D-360 on Haloperidol-Induced Catalepsy

Test Compound	Dose (mg/kg)	Baseline	60 min	120 min	180 min	240 min	300 min
Control Group (Haloperidol-induced catalepsy)	0.5	0 ±0	103.5 ±4.28	117.0 ±4.9	123.0 ±4.48	110.0 ±2.98	100 ±2.69
D-360	0.1	0 ±0	78.5 ±3.76*	79.5 ±3.65*	60.8 ±3.5*	77 ±3.96*	92.0 ±2.69*
	0.5	0 ±0	64.5 ±6.56*	71.0 ±4.64*	76.0 ±4.76*	72.0 ±3.27*	75.2 ±2.69*
	1.0	0 ±0	72.5 ±5.49*	78.0 ±4.36*	86.0 ±4.76*	83.0 ±3.67*	76.0 ±3.14*
	5.0	0 ±0	45.5 ±3.37*	54.7 ±2.79*	66.5 ±4.28*	74.5 ±3.61*	81.1 ±3.14*
	10.0	0 ±0	31.5 ±4.83*	36.5 ±4.02*	43.0 ±4.42*	48.5 ±3.58*	55.5 ±3.29*

*Values are expressed as M ± SEM. $p \leq 0.05$ vs. control group.

Administration of D-360 at 60.0 mg/kg increased the time spent in the light compartment to 47.0 ±8.5 s and reduced the time in the dark compartment to 73.0 ±8.5 s, with a K index of 0.64. At 30.0 mg/kg, light compartment time increased to 56.0 ±5.1 s, while dark compartment time decreased to 64.0 ±5.1 s, resulting in a K index of 0.88. The 10.0 mg/kg dose produced light compartment time of 59.0 ±6.23 s and dark compartment time of 61.0 ±6.23 s, with a K index of 0.97. At 5.0 mg/kg, a further increase in light compartment time was observed (63.0 ± 2.86 s), with a decrease in dark compartment time (57.0 ±2.86 s). The K index reached 1.10. The 1.0 mg/kg dose demonstrated light compartment time of 74.0 ±3.64 s and dark compartment time of 46.0 ±3.64 s, with the highest recorded K index of 1.61. At 0.5 mg/kg, light compartment time was 70.0 ± 4.59 s and dark compartment time was 50.0 ±4.59 s, with a K index of 1.40. Administration of 0.1 mg/kg resulted in light compartment time of 58.0 ±3.89 s and dark compartment time of 62.0 ± 3.89 s, with a K index of 0.94. Statistically significant differences compared to the control group were observed at multiple dose levels ($p \leq 0.05$).

Effect of D-360 on Haloperidol-Induced Catalepsy in Mice

Effect of D-360 on Haloperidol-Induced Catalepsy. In the control group treated with haloperidol (0.5 mg/kg), cataleptic response progressively increased over time, reaching 123.0 ± 4.48 at 180 minutes, followed by gradual reduction at later time points.

Administration of D-360 produced a dose-dependent attenuation of haloperidol-induced catalepsy. At 0.1 mg/kg, catalepsy scores were significantly reduced at all time points compared to the control group, with values of 78.5 ±3.76 at 60 min and 60.8 ±3.5 at 180 min. The 0.5 mg/kg dose further reduced cataleptic response, particularly at 60 and 120 minutes. Administration of 1.0 mg/kg demonstrated moderate suppression of catalepsy, with values remaining significantly lower than the control group throughout the observation period. At 5.0 mg/kg, cataleptic response was markedly reduced at early time points (45.5 ±3.37 at 60 min), with gradual increase over time. The highest dose (10.0 mg/kg) showed the most pronounced reduction in cataleptic scores, particularly at 60 and 120 minutes. Statistically significant differences compared to the haloperidol control group were observed at multiple time points ($p \leq 0.05$). The result demonstrated table 4.

5. Discussion

The present study comprehensively evaluated the pharmacological profile of D-360, including its acute toxicity and neurobehavioral effects in several experimental models.

The acute toxicity assessment demonstrated a relatively wide safety margin, as reflected by the LD_{50} value. This finding indicates that the compound possesses moderate to low acute toxicity, which is an important prerequisite for further pharmacological development [14,15].

In the Open Field test, D-360 produced dose-dependent alterations in locomotor and exploratory activity. Moderate

doses increased exploratory behaviour without pronounced suppression of motor activity. The reduction in defecation acts at several dose levels suggests decreased anxiety-related autonomic responses. Importantly, the absence of marked locomotor inhibition indicates that the observed behavioural effects are unlikely to be attributable to sedation.

In the Elevated Plus Maze model, D-360 increased time spent in the light/open compartment and elevated the K index at moderate doses, indicating a significant anxiolytic-like effect. Notably, the dose–response relationship demonstrated an inverted U-shaped pattern, with optimal effects observed at intermediate doses, while higher doses showed reduced efficacy. Such a pattern is frequently observed in central nervous system–active compounds.

In the pentylenetetrazol-induced anxiety model, D-360 attenuated PTZ-induced anxiety-like behaviour, as evidenced by increased time in the light compartment and elevated light/dark ratio. These findings further support the compound's anxiolytic potential under chemically induced stress conditions [9-11].

Additionally, D-360 significantly reduced haloperidol -induced catalepsy. Since haloperidol-induced catalepsy is primarily associated with dopaminergic blockade in the nigrostriatal pathway, attenuation of this effect suggests that D-360 may modulate dopaminergic neurotransmission or exert neuroprotective properties without inducing extrapyramidal side effects [12,13].

Taken together, the results obtained across multiple behavioural paradigms consistently indicate that D-360 exhibits pronounced anxiolytic activity with a favourable safety profile. The absence of significant motor suppression and the ability to counteract haloperidol-induced catalepsy further highlight its potential advantages over classical anxiolytic agents.

Although the precise mechanism of action remains to be elucidated, the behavioural profile observed in this study suggests possible involvement of GABAergic and/or dopaminergic pathways. Further biochemical and receptor-binding studies are warranted to clarify the underlying mechanisms.

6. Conclusions

The present study demonstrated that D-360 exhibits significant anxiolytic activity across multiple experimental models. The compound produced dose-dependent behavioural effects in the Open Field and Elevated Plus Maze tests, as well as in the pentylenetetrazol-induced anxiety model.

Moderate doses showed the most pronounced anxiolytic effects without marked suppression of locomotor activity, indicating the absence of significant sedative properties. Additionally, D-360 attenuated haloperidol-induced catalepsy, suggesting a modulatory effect on dopaminergic pathways and a potentially favourable neurological safety profile.

The calculated LD_{50} value indicates a relatively wide therapeutic window, supporting the compound's safety in acute exposure conditions.

Overall, the obtained findings suggest that D-360 may represent a promising candidate for further pharmacological investigation as a potential anxiolytic agent [17,18].

REFERENCES

[1] Bandelow B, Michaelis S. Epidemiology of anxiety disorders in the 21st century. Dialogues Clin Neurosci. 2015; 17(3): 327–335.

[2] Slee A, Nazareth I, Bondaronek P, Liu Y, Cheng Z, Freemantle N. Pharmacological treatments for generalised anxiety disorder: a systematic review and network meta-analysis. Lancet. 2019; 393(10173): 768–777.

[3] Calhoon GG, Tye KM. Resolving the neural circuits of anxiety. Nat Neurosci. 2015; 18(10): 1394–1404.

[4] Kaur R, Kaur M, Singh J. Enduring role of 1,3,4-oxadiazole moiety in medicinal chemistry: a review. Bioorg Chem. 2021; 113: 105024.

[5] Verma G, Marella A, Shaquiquzzaman M, Akhtar M, Ali MR, Alam MM. 1,3,4-Oxadiazole derivatives: synthesis and biological activity. Mini Rev Med Chem. 2017; 17(4): 326–351.

[6] Bhandari SV, Bothara KG, Raut MK, Patil AA. Design and evaluation of novel oxadiazole derivatives as CNS active agents. Eur J Med Chem. 2016; 124: 987–1000.

[7] Kraeuter AK, Guest PC, Sarnyai Z. The Elevated Plus Maze test for measuring anxiety-like behavior in rodents. Methods Mol Biol. 2019; 1916: 69–74.

[8] Seibenhener ML, Wooten MC. Use of the Open Field Maze to measure locomotor and anxiety-like behavior in mice. J Vis Exp. 2015; (96): e52434.

[9] Bourin M, Hascoët M. The light/dark box test. Eur J Pharmacol. 2003; 463(1–3): 55–65.

[10] Löscher W. Animal models of seizures and epilepsy: past, present, and future role for the discovery of antiseizure drugs. Neurochem Res. 2017; 42(7): 1873–1888.

[11] Farooq RK, Asghar K, Kanwal S, Zulqernain A. Role of pentylenetetrazole-induced kindling in epilepsy and anxiety research. Neurosci Biobehav Rev. 2016; 71: 544–558.

[12] Sanberg PR, Bunsey MD, Giordano M, Norman AB. The catalepsy test: its ups and downs. Behav Neurosci. 1988; 102(5): 748–759.

[13] Bishnoi M, Chopra K, Kulkarni SK. Modulatory effect of dopaminergic mechanisms in anxiety models. Pharmacol Biochem Behav. 2018; 169: 42–49.

[14] OECD. Test No. 423: Acute Oral Toxicity – Acute Toxic Class Method. OECD Guidelines for the Testing of Chemicals. Paris: OECD Publishing; 2019.

[15] Erhirhie EO, Ihekwereme CP, Ilodigwe EE. Advances in acute toxicity testing: strengths, weaknesses and regulatory acceptance. Interdiscip Toxicol. 2018; 11(1): 5–12.

[16] Kalueff AV, Stewart AM, Song C, Berridge KC, Graybiel

AM, Fentress JC. Neurobiology of rodent self-grooming and its value for translational neuroscience. Nat Rev Neurosci. 2016; 17(1): 45–59.

[17] Griebel G, Holmes A. 50 years of hurdles and hope in anxiolytic drug discovery. Nat Rev Drug Discov. 2013; 12(9): 667–687.

[18] Al-Harbi NO, Imam F, Al-Harbi MM, et al. Neuroprotective and behavioral effects of heterocyclic compounds in anxiety models. Front Pharmacol. 2020; 11: 1234.

[19] Sanoev Zafar, Sultanov Sardor et al. Relevance and prospects of searching for drugs with anxiolytic activity based on synthetic active substances containing 1,3,4-oxadiazole ring. Universal Index Library of the Eurasian Journal of Medical and Natural Sciences 5.10 (2025): 130-138.

[20] Sultanov SA, Khalilov HD. Biological activity of 1,3,4-oxadiazole derivatives and their significance in medicine. Latin American Journal of Education. 2025; 5(6): 605–614.

[21] Sultanov SA, Sanoyev ZI, Sirov VN. Anxiolytic activity of heterocyclic ring-containing compounds. Latin American Journal of Education. 2025; 5(7): 470–478.

[22] Sultanov, Sardor Allayar Ugli, and Zafar Isomiddinovich Sanoev. "Evaluation of the Neurotropic Activity of Synthetic Heterocyclic Compounds (Literature Review)." Re-health journal 4 (24) (2024): 33-38.