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Deltamethrin Neurotoxicity and the Safety Profile of Selenium Nanoparticles (SeNPs): Behavioral and Histopathological Evaluation

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Abstract | Selenium nanoparticles (SeNPs) play a crucial structural and functional role in supporting normal cellular processes, growth, and development by maintaining redox homeostasis, enhancing selenoenzyme activity, and preserving structural integrity at the cellular and tissue levels. Therefore, the present in vivo study aimed to investigate the behavioral consequences of Deltamethrin (DM) and nanoselenium in rats after 3 months of oral administration. Thirty adult albino male rats were selected and assigned to three groups using a random allocation method as follows: control, DM-treated (0.3 mg/kg b.w.), selenium nanoparticles SeNPs-treated (1 mg/kg). Rats received DM orally on a daily basis while SeNPs was administered 3 times/ week. At the end of the experiment, rats were euthanized using sodium pentobarbital (≥150 mg/kg, i.p.) and tissue samples were immediately collected. Behavioral assessments included locomotor activity, and anxiety-like responses. Rats exposed to DM displayed reduced locomotor activity, altered exploratory behavior, and increased anxiety indices compared with controls, indicating neurobehavioral toxicity. Conversely, Nanoselenium administration alone did not induce significant behavioral deficits and was associated with mild improvements in locomotor activity. Histopathological assessment of cerebral cortex revealed neuronal degeneration in the DM group, whereas rats receiving SeNPs exhibited preserved cortical architecture with no apparent histopathological alterations, indicating good biocompatibility and the therapeutic potential of SeNPs in preserving neural integrity.

Keywords | Deltametrhrin, Cerebral cortex, Selenium Nanoparticles, Neurodegeneration, Histopathology

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INTRODUCTION

Deltamethrin (DM) is a broad-spectrum, type II synthetic pyrethroid insecticide that has been widely used in both agricultural pest management and public health vector control because of its high efficacy and relative environmental persistence (Rehman *et al.*, 2014). Although pyrethroids are generally considered relatively safe in mammals compared to many other pesticides (Chrustek *et al.*, 2018), DM has been shown in numerous studies to cause neurotoxicity, Particularly following ichronic exposure or at higher doses (Shi *et al.*, 2024). Behavioral and neurological studies in rats have reported that DM exposure leads to alterations in anxiety and locomotor activity dysregulation

of learning and memory processes, changes in neurotransmitter systems, and motor coordination deficits (Souza *et al*., 2022). In addition, DM has been shown to cause marked histopathological alterations in neural tissues including including neuronal degeneration, cytoplasmic vacuolization, and pyknosis (Khalifa *et al*., 2022). DM effectively induces neurodegeneration through multiple mechanisms,including the disruption of sodium channel function, mitochondrial dysfunction, and the induction of regulated cell death (Kumar et al., 2015; Li et al., 2022). Additionally, it promotes neuroinflammation and triggers microglial activation (Hossain et al., 2022). DM presents a complex profile of toxicity, primarily associated with ROS production, as evidenced through (Lu et al., 2019). Various promising agents targeting oxidative stress, redox imbalance, and neuronal loss are under investigation as potential adjunct or alternative therapies for neurotoxicity (Lee et al., 2020).

As an essential trace element, selenium (Se) plays a critical role in diverse physiological processes, notably in maintaining redox equilibrium, supporting antioxidant defense mechanisms, and regulating cellular growth and programmed cell death (Saito, 2022). In the nervous system, selenium has demonstrated neuroprotective potential in several experimental models. For instance, in models of Alzheimer-type dementia induced by intracerebroventricular streptozotocin (ICV-STZ), pre-treatment with sodium selenite (0.1 mg/kg) ameliorated memory deficits (Morris water maze, passive avoidance), decreased oxidative damage, and reversed cholinergic dysfunction (Ishrat et al., 2009). Nanotechnology is breakthrough technology pervading all fields especially in medical research and newer applications of this field (Haleem *et al*., 2023). Nano-selenium (SeNPs) are being explored worldwide due to their potentially improved bioavailability, lower toxicity, and greater capacity to cross blood brain barriers compared to the traditional form of Se (Li et al., 2024) in addition to enhanced antioxidative and anti-inflammatory activity (Prasad et al., 2024).

SeNPs serve as a bioavailable selenium source for the synthesis of selenoproteins such as glutathione peroxidases and thioredoxin reductases, which neutralize reactive oxygen species and maintain glutathione homeostasis (Kondaparthi et al., 2019). Moreover, The integration of selenium into reductases enhances its vital function in preventing oxidative alterations to essential biomolecules such as lipids, proteins, and nucleic acids (Shahidin et al., 2025; Zhang *et al*., 2020). In parallel, SeNPs upregulating endogenous antioxidant enzymes (SOD, CAT, GPx) and directly scavenging free radicals (Chhabria and Desai, 2016). They also exert a significant property to mitigate the inflammation by suppressing NF-κB signaling, thereby lowering levels of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) and reducing COX-2 and iNOS expression (Bai *et al*., 2025). At the mitochondrial level, SeNPs stabilize membrane potential, preserve ATP production, inhibit cytochrome c release, and shift the balance toward cell survival by increasing Bcl-2 and decreasing Bax expression, ultimately reducing caspase-mediated apoptosis (Ansari *et al*., 2024).

Khalil *et al*. (2022) reported that SeNPs at 0.5 mg/kg administered three times per week over two months significantly did not induce behavioral deficits, maintaining neuronal structure, and normal histopathological characteristics in male rats. similarly Hadrup et al. (2019) found that treatment with selenium nanoparticles had no significant impact on neurochemical markers, hematological indices, or hepatic histopathology, confirming their good tolerability in experimental animals. At safe exposure levels, SeNPs preserve neuronal integrity, and maintain normal behavioral deficits (Yuan *et al*., 2020); however, particle size, prolonged or excessive administration may lead to tissue accumulation and shift their role from antioxidant to pro-oxidant, thereby inducing oxidative stress and organ damage (Wang *et al*., 2020). The present study aimed to evaluate the neurotoxic effects of DM and safety of selenium nanoparticle (SeNP) adminsteration on neurobehavioral and histopathological alterations.

MATERIALS AND METHODS

Experimental animals

A total of thirty non-medicated, clinically healthy adult male albino rats (10-12 weeks) weighing 190–210 g were purchased from the Animal Lab facility of Agricultural Research Center, housed in groups of ten per cage and allowed to acclimatize to laboratory conditions for two weeks. They had free access to basic laboratory diet throughout the study period. The experimental protocol followed the ethical standards outlined in the Guide for the Care and Use of Laboratory Animals (Care and Animals, 1986), ensuring proper handling and welfare of all animals used in the study. This was also officially approved by the animal care and use committee of the Faculty of Veterinary Medicine, Suez Canal University.

Drugs and chemicals

Deltamethrin (Butox®, 50 mg/mL), ($C_{22}H_{19}Br_2NO_3$, Na_2SeO_3, CAS: No.52918-63- 5), a pure cis-isomer (purity >97%) commercial formulation for veterinary use was obtained from Intervet Co. (Paris, France) and dissolved in corn oil.

Selenium nanoparticles (SeNPs) were synthesized via a wet chemical reduction method using sodium selenite (Na_2SeO_3, 99%) employed as the selenium source

(precursor) and ascorbic acid as the reducing agent, and dextrin as the stabilizing agents. The resulting nanoparticles exhibited an average core diameter of 81.25 nm, as reported by Transmission Electron Microscopy (TEM) (Figure 1A) and a histogram showing different size distribution of selenium nanoparticles (Figure 1B). The synthesis protocol followed the procedure of Dhawan *et al.* (2021) was consistent with the method previously described by Arulnathan *et al.* (2016).

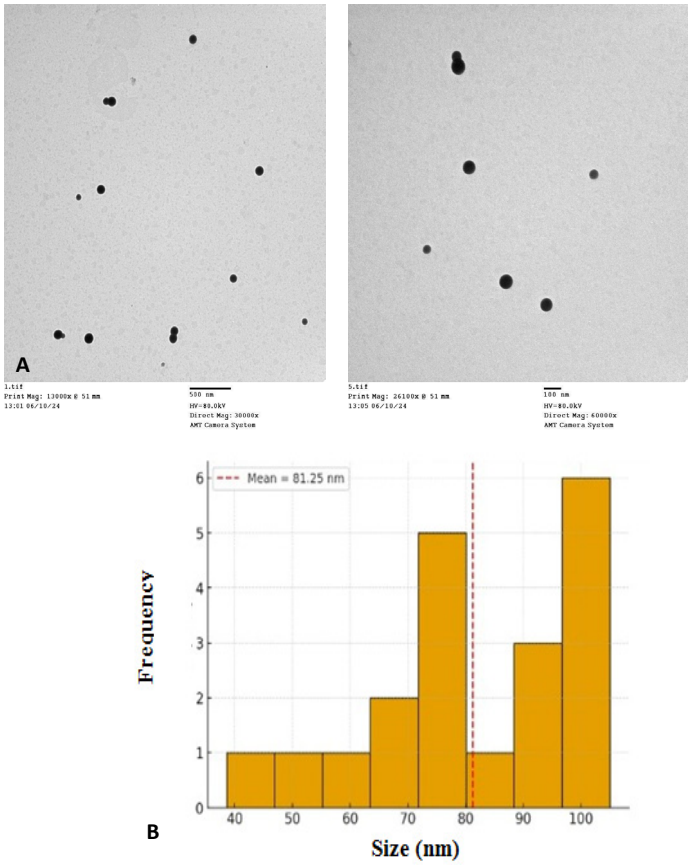

Figure 1: A: Transmission electron microscope image of nano-selenium size of 49-104 nm, average mean size 81.25 nm and spherical shape. B: Histogram of Selenium Nanoparticles showing the full size distribution.

Synrthesis and characterization of SeNPs

Selenium nanoparticles can be synthesized efficiently by a wet chemical method using sodium selenite (selenium precursor). In this process, ascorbic acid acts as a mild agent for reduction, converting Se (IV) ions, where selenium exists in the +4 oxidation state, to elemental selenium under controlled conditions. Dextrin was added simultaneously as a stabilizing and capping agent, preventing particle aggregation and promoting uniform nucleation. The reaction mixture was typically stirred until a distinct color change indicates nanoparticle formation. The resulting colloid was washed and purified to remove unreacted reagents, yielding stable nanoselenium dispersion.

Transmission electron microscopy (TEM) was employed to characterize selenium nanoparticle size, morphology, and crystallinity of nanoparticles. In order to assess the morphology of the nanoparticles, a drop of the prepared solution was carefully deposited onto carbon-coated copper grids (CCG) and dried at room temperature by allowing the solvent to evaporate naturally. Electron micrographs were obtained using JEOL JEM-1010 TEM at 80 kV at The Regional Center for Mycology and Biotechnology, (RCMB) Al-Azhar University (Amin *et al.*, 2021). Transmission electron microscope image of nanoparticles revealed Nano selenium as discrete, nearly spherical structures with sizes in the range depending on the synthesis conditions. The spherical shaped selenium nanoparticle appeared well dispersed and the diameter measured between 49-104 nm.

Experimental design

Thirty rats were assigned to three experimental groups randomly, each comprising ten animals. First Group (Control): Rats received corn oil by oral gavage. Second Group (Deltamethrin): Rats were orally were administered dissolved deltamethrin in corn oil at a dose of 0.3 mg/kg bw once daily. Third Group (Nanoselenium): Rats were orally gavaged with selenium nanoparticles (SeNPs) synthesized as described previously at a dose of 1mg/kg three times weekly. All treatments were given for 90 consecutive days, and doses were freshly prepared and adjusted to the individual body weight of each rat.

Behavioral and neurological assessment

By the end of treatment period, all groups underwent behavioral testing one day after the final dose, performed sequentially as previously described. The behavioral assessment were monitored and documented with an HD video camera (Model No. B80, Yes-original. co, China) for behavioral recording and scoring, The behavioral data were evaluated with the Behavioral Observation Research Interactive Software (BORIS), v. 2.95, University of Torino, Torino, Italy.

The open field test (OFT)

The behavior of Rats was conducted by evaluating their locomotion, anxiety-related, and exploration activities according to (Voikar and Stanford, 2022). The animals were introduced into the central area of a transparent open-field Plexiglas chamber measuring 40 cm × 60 cm × 30 cm (W × L × H) divided into 36 equal squares outlined with white lines to facilitate locomotion tracking. Each setup was enclosed with a cardboard cover to block visual contact with other animals and the surroundings. Each rat was introduced into the central square of the apparatus, marking the commencement of a 10-minute session. The behavior of each rat included immobility duration, walking duration, exploratory activities. The behavioral activities

duration was recorded according to (Seibenhener and Wooten, 2015).

Sample collection

At the end of the experiment, animals were humanely euthanized with sodium pentobarbital (≥150 mg/kg, i.p.) in accordance with the recommendations of the Institutional Animal Care and Use Committee (IACUC) and the AVMA Guidelines for the Euthanasia of Animals, The brains were then carefully dissected along the coronal (frontal) plane to obtain serial sections for morphological assessments. A portion of the tissue was fixed in a 4% paraformaldehyde fixative for a period of 24–48 hours for subsequent histopathological evaluation.

Histopathological assessment

Formalin-fixed tissue sections of the brain were routinely for histology, sections at thickness of 4–5 µm, stained with hematoxylin and eosin (H and E) following standard protocols outlined by (Bancroft and Gamble, 2008; Denoix *et al*., 2022) for general histological examination and scoring. Sections were examined to identify characteristic neurodegenerative lesions in the cerebral cortex using a LEICA (DFC290 HD) camera connected to the light microscope.

Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS, version 22.0; IBM Corp., Chicago, IL, USA). The Shapiro–Wilk test was used to assess data normality. Differences among the experimental groups were evaluated by one-way analysis of variance (ANOVA) for both biochemical and behavioral parameters. When significant differences were detected, Duncan's multiple range test (DMR) was applied as a post hoc procedure. Results are presented as mean ± standard error (SE), and significance was set at $p < 0.05$

RESULTS AND DISCUSSION

Effect of deltamethrin on histological assessment and scoring

The cerebrum sections were stained with H and E stain for general histological examination. In Figure 2, the cortical cross sections of the cerebrum with HandE staining showed normal architecture. They revealed an intact covering of attached piamater layer, and a normal composition of the cortical layers. The latter consisted of normally organized six layers: molecular, external granular, external pyramidal, internal granular, internal pyramidal and polymorphic layers (Figure 2A). Each layer contained normal basophilic neurons with vesicular nuclei, neuroglial cells, and blood capillaries (Figure 2A, B). In the SeNPs group, the cerebral cortex appeared normally organized with intact cells and capillaries as in the control group (Figure 2C, D). Neurons appeared with normal morphology and preserved neuropil. No structural disruption was detected. Findings consistent with Khalil *et al*. (2022). According to the previous study, the current study further demonstrates their safety at the tested dose and regimen. The preserved cortical and preserved hippocampal histoarchitecture, along with maintaining normal behavioral performance in rats, supports the absence of neurotoxicity following repeated SeNP administration that may attributed to higher bioavailability, lower toxicity, and stronger free radical scavenging ability.

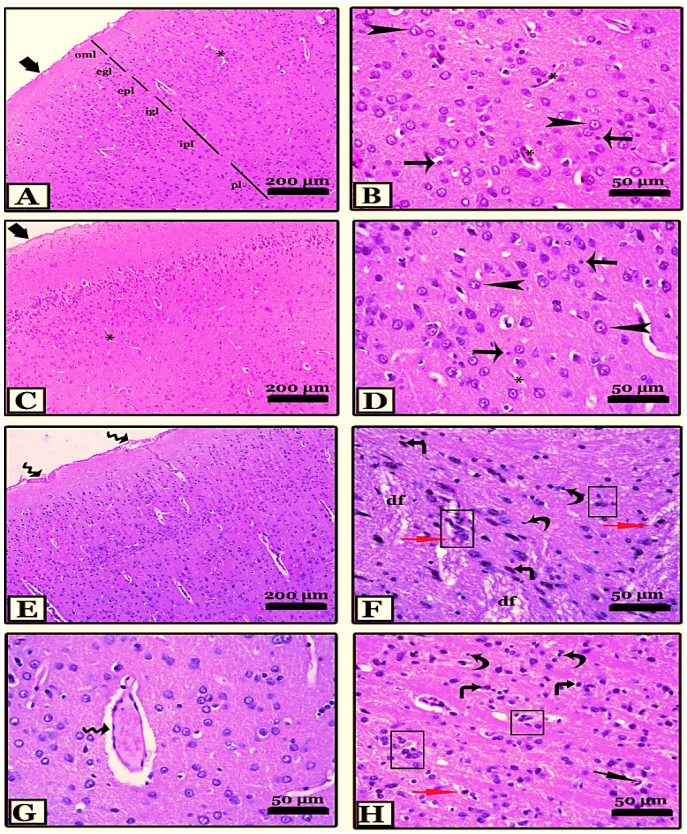

Figure 2: Representative photomicrographs of the cerebral cortex cross sections in adult male albino rats. The control group (A and B) and SeNPs group (C and D) showing normal piamater (thick arrows), normal cortical six layers: outer molecular (oml), external granular (egl), external pyramidal (epl), internal granular (igl), internal pyramidal (ipl), and polymorphic (pl)) layers containing normal neurons (arrowheads), normal neuroglia cells (thin arrows), and normal blood capillaries (*). The deltamethrin group (E, F, G and H) showing degenerated neurons (turned arrows), degenerated neuroglia cells (curved arrows), congested and dilated blood capillaries (zigzagged arrows), degenerated nerve fibers (df), inflammatory cells (red arrows), and neurophagia (squares). H and E stain, cerebrum X100 par 200µm, and X400 par 50µm.

On the other hand, the cerebral cortex in the deltamethrin group was affected by the administration of deltamethrin,

which revealed extensive pathological lesions. The cerebral sections showed degeneration in the meningeal covering with capillary dilatation and congestion (Figure 2E), severe degeneration of the neuropil, the gliocytes, and deterioration of cerebral neurons. Apoptotic neurons appeared contracted, intensely stained (pyknotic) nuclei, accompanied by perineuronal vacuolation (Figure 2E, F). In addition to inflammatory cells infiltration and neuroglia cell cluster surrounding degenerated neurons until their disappearance leads to many neurophagia, as well as cerebral capillary dilatation and congestion (Figure 2G, H).

Our findings are consistent with those reported by Ali *et al.* (2017) and Hussein *et al.* (2018). The observed similarity may be ascribed to the compound's capacity to delay the closure of voltage-gated sodium channels, thereby inducing sustained depolarization, neuronal hyperexcitability, and eventual excitotoxicity. Such ionic imbalance triggers a cascade involving oxidative stress, lipid peroxidation, and mitochondrial impairment, ultimately resulting in neuronal degeneration (Khalatbary *et al.*, 2015; Saoudi *et al.*, 2017). Another possible explanation could be the reduction in both enzymatic and non-enzymatic antioxidant levels, associated with elevated thiobarbituric acid reactive substances and heightened oxidative stress, which together contribute to neurotoxicity (Ogaly *et al.*, 2015).

Effect of deltamethrin on behavior parameters

Regarding open field test, the obtained data revealed that deltamethrin (DM)-treated rats showed significant behavioral alterations compared with the control group. Specifically, DM markedly increased the latency to the first step (4.62 ±0.92 s vs. 1.94 ±0.06 s in control) and duration of freezing episodes (567.39 ±4.79 s), while it significantly reduced duration of crossing lines and rearing episodes (9.25 ±2.09 s) and exploratory activities (10.58 ±3.23) (Table 1). These observations corroborate the results of Gasmi *et al.* (2017), who assessed locomotor activity in an open field test after inducing neurotoxicity with deltamethrin. Interestingly, grooming activity was markedly reduced in DM-treated rats (8.16 ±2.12) compared to the control group (21.63 ±5.66). These outcomes are consistent with earlier reports in the literature of Sadeghi-Hashjin *et al.* (2011), which confirmed the neurotoxic effect of agents such as cypermethrin, and permethrin on behavior in mice. Ghasemi *et al.* (2022) reported that anxiety-like behaviors are associated with cellular and structural alterations within the hippocampus. The manifestation of anxiety-like behaviors following DM exposure could be ascribed to its neurotoxic effects through the overproduction of ROS, resulting in lipid peroxidation and subsequent neuronal damage, particularly within the hippocampus, along with disruption of neurotransmitter homeostasis (Hossain *et al.*, 2022). These neurochemical and structural disturbances compromise inhibition of GABAergic signaling and potentiate glutamatergic excitotoxicity (Hossain *et al.*, 2008), in addition to inducing dopaminergic system dysfunction and impairments in synaptic plasticity (Souza *et al.*, 2022). Collectively, these alterations contribute to the expression of anxiety-like phenotypes, as evidenced by using the open field and elevated plus maze behavioral evaluations. Several studies have reported that DM promotes reactive oxygen species (ROS) generation, in agreement with our results (Khalifa *et al.*, 2022). These studies reported that exposure to pyrethroids including DM induces neurotoxicity and impairs short-term memory in rats, accompanied by anxiety-like behavior, locomotor disturbances, and oxidative stress.

In contrast, rats administered SeNPs exhibited normalized walking time, exploratory activity, along with normal grooming and immobility activity comparable to the control group, indicating a decline in stress-related behaviors observed in the deltamethrin treated group. SeNPs adminsteration preserved normal morphology and neuronal integrity particularly within brain regions essential for motor coordination, movement, and emotional regulation such as the hippocampus, cerebral cortex thereby preserving neuronal networks and synaptic integrity necessary for maintaining normal locomotor and exploratory activities. Our findings are consistent with the previous report by Khalil *et al.* (2022), which demonstrated that selenium nanoparticles preserved normal histological and behavioral patterns.

Table 1: Behavioral observations (sec.) of rats reared at different treatments at the open field test.

Behavioral observation OFT	Latency till first step	Walking duration	Immobility duration	Exploratory activities	Grooming activities
Control	1.94±0.06^{b}	56.46±10.41^{a}	476.67±26.30^{c}	43.30±11.11^{a}	21.63±5.66^{b}
Deltamethrin	4.62±0.92^{a}	9.25±2.09^{d}	567.39±4.79^{a}	10.58±3.23^{b}	8.16±2.12^{b}
SeNPs	2.20±0.44^{b}	34.10±5.70^{bc}	524.55±7.43^{abc}	23.82±5.67^{b}	15.33±3.42^{b}
p-value	0.04	<0.001	0.002	0.004	<0.001

Results are presented as Mean ± standard error. Superscripts indicating different values are considered substantially different at a threshold of significance of $P < 0.05$, as determined by the Duncan multiple test.

CONCLUSION AND RECOMMENDATIONS

Deltamethrin (DM) exposure induced marked neurobehavioral impairments, including reduced locomotion, increased immobility, and diminished exploratory activity, accompanied by neuronal degeneration in the cerebral cortex. In contrast, Nanoselenium administered alone did not cause adverse behavioral or histological alterations, maintaining normal locomotor patterns and preserved cortical architecture. These findings highlight the protoxic potential of DM on neural tissues and behavior. Meanwhile, Nanoselenium appears safe at the tested dose, exerting no harmful effects on brain histology or function. It is recommended that using of DM must be carefully regulated and monitored, particularly in environments with potential for chronic human or animal exposure. Future studies should investigate the dose-dependent protective mechanisms of SeNPs, explore molecular pathways underlying their neuroprotective effects, and assess their long-term safety and comparative efficacy against other antioxidant agents.

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NOVELTY STATEMENT

This study provides new insights into the neurotoxic effects of deltamethrin and the safety profile of selenium nanoparticles (SeNPs) through behavioral and histopathological evaluations. By assessing SeNPs independently, the work establishes their neurobehavioral safety at the tested dose and regimen. The findings contribute novel baseline data for the non-toxic nature of SeNPs, highlighting their ability to preserve neuronal integrity and behavioral performance. supporting their future use in neurotoxicological and therapeutic investigations.

AUTHOR'S CONTRIBUTION

Taiseer M. Mohamed writing original draft preparation and editing. Bassant A. Elbaz: Analyzing the behavioral assessment. Amina A. Dessouki, Hamdi A. Fetaih, Ahmed S. Eltahan: supervision. All authors have read and agreed to the published version of the manuscript.

Ethical statement

All experimental animal procedures were conducted at the Animal Research Laboratory, Faculty of Pharmacy, Suez Canal University, Egypt. The study followed the guidelines of the Institutional Research Committee and the Animal Ethics Committee, Faculty of Veterinary Medicine, Suez Canal University, Egypt.

Generative AI and AI-assisted technology statement

All authors of this work declare that generative AI technologies including large language models (e.g., ChatGPT, Copilot) and text-to-image generators were not utilized in any capacity during the preparation, writing, or editing of this manuscript.

Conflict of interest

The authors have declared no conflict of interest.

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