



Toxicological evaluation of *Sclerophrys regularis* venom in Swiss Albino mice

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Abstract

Toads have been used for several purposes over time. It has played several prominent roles in human daily activities, such as in religious rituals, witchcraft, poisoning, hunting, warfare and healing. Toad venom possesses a wide variety of chemical compounds that have potential significance in pharmacology, toxicology and therapeutics. This research was designed to study the toxicological effects of toad venom in Swiss albino mice. The median lethal dose (LD₅₀) of the venom was determined by a modified up-and-down method, haemolytic effect by spectrophotometry, and fibrinolytic effect by the clotting time (in minutes). In mice, in vivo effect was assessed following intraperitoneal injection of 4 times the LD₅₀ of the venom in Swiss albino mice. The LD₅₀ of *Sclerophrys regularis* venom was calculated to be 35.48 mg/kg in this study. A modest fibrinolytic effect was noted, though not statistically significant, and it proved independent of *Sclerophrys* venom concentration. The least pronounced haemolytic activity occurred at 7.5 and 3.75 mg/mL venom concentrations. The histology slides showed marked areas of congestion in the lungs, kidneys, heart, liver and brain. Besides the congestion, the kidneys showed coagulative necrosis, tubular degeneration, and Bowman's space dilation; the heart exhibited haemorrhage, oedema, and vacuolation; the spleen displayed red/white pulp depletion and trabecular dilation; while the liver featured central vein congestion and hepatocyte vacuolation. The lungs and brain reflected oedema and vacuolation. These pathologies underscore the broad, debilitating impact of *Sclerophrys regularis* venom.

Keywords: Bufotoxins, Congestion, Haemolysis, LD₅₀, Necrosis, *Sclerophrys regularis*, Vacuolation

Introduction

Toads have long woven themselves into human myths and medicines, captivating cultures for millennia. From ancient rituals and witchcraft to

hunting aids, warfare tools, and healing remedies, they've shaped daily life across societies (Lyttle et al., 1996).

Only two toad species hold medical and veterinary importance in Nigeria: the arboreal *Sclerophrys perreti* (tree-climbing toad), thriving in southern Guinea savannahs and rainforests first spotted in Idanre, Ondo State, then across the south and the ubiquitous *Sclerophrys regularis* (common land toad), burrowing under logs, rocks, or damp spots nationwide, surfacing near breeding waters (Pauly *et al.*, 2009). The skin of the toad serves not only as a protective barrier against dehydration and pathogens, together with the limbs and lungs, constitutes an integrated adaptive system that has enabled long-term survival and the successful exploitation of a wide range of ecological habitats (Garg *et al.*, 2008; Pauly *et al.*, 2009). The toad's skin secretes a wide variety of chemical compounds that have potential significance in pharmacology, toxicology and therapeutics (Garg *et al.*, 2008; Pauly *et al.*, 2009; Johnny & Bangura, 2024). Recent investigations into toad toxins have highlighted their potential for diverse pharmaceutical applications, extending beyond the management of cardiac failure to include antimicrobial, analgesic, anti-inflammatory, immunoregulatory, and anticancer effects (Qi *et al.*, 2018; Aliyu *et al.*, 2021).

Toads possess prominent parotoid glands located on the dorsum, which secrete venom when the toad is threatened or attacked. This venom contains a range of bioactive compounds, including dopamine, epinephrine, norepinephrine, serotonin, bufotenine, bufogenins, bufotoxins, and indole alkylamines. These compounds are toxicologically active and are rapidly absorbed across mucous membranes into the systemic circulation. Once absorbed, they exert effects on the cardiovascular system, particularly the heart and peripheral vasculature, as well as on the nervous system (Asrorov *et al.*, 2023).

Bufogenins and bufotoxins are cardioactive steroids commonly classified as digitalis-like substances. Bufogenins exert their effects by inhibiting the membrane-bound Na^+/K^+ -ATPase, a key enzyme responsible for maintaining intracellular sodium and potassium gradients. Inhibition of this enzyme leads to an increase in intracellular sodium, which subsequently reduces the activity of the $\text{Na}^+/\text{Ca}^{2+}$ exchanger, resulting in elevated intracellular calcium concentrations and enhanced cardiac contractility (Asrorov *et al.*, 2023). In addition to these cardiotoxic effects, toad venom contains indole alkylamines such as bufotenine (5-hydroxy-N, N-dimethyltryptamine), which has been associated with hallucinogenic or psychedelic effects. These neuroactive effects may be related to its serotonergic activity and systemic

physiological actions (Asrorov *et al.*, 2023). Due to its psychoactive properties and potential for misuse, bufotenine has been classified as a Schedule I substance by the United States Food and Drug Administration (USFDA) since 1967 (Lyttle, 1993). Young animals, particularly canids and felids, are highly susceptible to bufotoxosis following exposure to toad secretions, typically through mouthing, biting, or chewing toads of the genus *Bufo*. Smaller animals are especially vulnerable and may exhibit more severe clinical signs following exposure (Eubig, 2001). The clinical presentation of toad poisoning is variable, ranging from localized to systemic effects. Local effects are usually rapid in onset and include profuse frothy salivation, anxiety, and vomiting. In severe cases, death may occur within 15 minutes of exposure (Eubig, 2001). Additional clinical signs include hyperaemic mucous membranes, recumbency, collapse, and tachypnoea. Neurological manifestations are common and may include convulsions, ataxia, nystagmus, and stupor (Gadelha *et al.*, 2014). Cardiac involvement is also frequently reported, with arrhythmias such as bradycardia, sinus tachycardia, and sinus arrhythmias (Chen and Huang, 2013). Furthermore, signs of pulmonary oedema, hyperthermia, and hyperkalaemia have been documented in affected animals (Chen & Huang, 2013).

This research aimed to evaluate the acute and systemic toxicological effects of venom from *Sclerophrys regularis* in Swiss albino mice, including determination of the median lethal dose (LD_{50}) and assessment of its haemolytic, fibrinolytic, haemorrhagic, and histopathological impacts.

Materials and Methods

Experimental animals

Six adult toads of the genus *Sclerophrys regularis* (150-280g) were captured from the premises of the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria. They were transported to the Toxicology Laboratory in an aerated plastic box and identified in the Zootoxicology Unit of the Department of Veterinary Pharmacology and Toxicology, Ahmadu Bello University, Zaria, Nigeria. All procedures were conducted in accordance with Ahmadu Bello University animal care guidelines.

Milking

Extraction of the toad venom was carried out following the methods of El-Naggar *et al.* (2024). However, a few modifications were done. After about 3 hours of rest, they were placed in a wide bowl half-

filled with clean water. The toads were allowed to swim for 30 minutes to loosen dirt around the venom glands, after which cotton wool soaked in 10% ethanol was used to clean the glands of dirt and microbes. The toads were then restrained from the dorsum with the head between the thumb and the index finger (i.e., the thumb on one side and the remaining four fingers wrapped around it from the other side). The venom glands on the back were gently massaged with the free hand to milk the venom into a Petri dish. Milked venom was pooled together and dehydrated using a desiccator with activated silica gel as the desiccant and stored at 4°C.

*Determination of median lethal dose (LD₅₀) of venom of *Sclerophrys regularis**

The up-and-down method introduced by Dixon & Mood (1948), modified by Bruce (1987) and remodified by Depass (1989) was used to determine the median lethal dose (LD₅₀). In this method, Swiss albino mice were dosed one at a time starting with an estimated LD₅₀ dose (30 mg/kg). The next mouse received a higher dose by a constant multiplicative factor of 1.5. Doses were usually adjusted by a constant multiplicative factor of 1.5. The dose for each successive mouse was adjusted up depending on the outcome for the previous mouse.

Determination of the fibrinolytic activity

The fibrinolytic activity of venom obtained from *Sclerophrys regularis* was evaluated using a plasma clotting assay, following the method of Theakston & Reid (1983) with slight modifications. Fresh caprine blood (from Uda breed) was collected into tubes containing 3.8% sodium citrate (9:1, v/v) as an anticoagulant. The samples were centrifuged at 2500 rpm for 15 minutes at 4°C to obtain platelet-poor plasma (PPP), which was used immediately for the assay.

Crude venom was prepared at different concentrations (30, 15, 7.5, 3.75, 1.575 and 0.7875 mg/mL). An aliquot of 0.2 mL of citrated plasma (2.0 mg/mL) was placed in a test tube and incubated at 37 ± 1°C. Thereafter, 50 µL of venom solution was added, followed immediately by 50 µL of calcium chloride (CaCl₂, 25 mM) to initiate clot formation. The time taken for clot formation was recorded in minutes. Fibrinolytic activity was expressed as the inverse of the clotting time (1/clotting time, min⁻¹), with shorter clotting times indicating higher fibrinolytic activity. All assays were performed under identical conditions, and appropriate controls were included (Theakston & Reid, 1983).

Determination of the hemolytic activity

The haemolytic activity of venom obtained from *Sclerophrys regularis* was evaluated in vitro using caprine erythrocytes, following the method of Gomes & De (1999) with slight modifications.

Fresh caprine blood (20 mL) was collected into tubes containing sodium citrate as an anticoagulant. The blood was centrifuged at 2400 rpm for 10 minutes, and the plasma was carefully discarded. The packed erythrocytes were washed three times with normal saline (0.9% NaCl) by resuspension and centrifugation under the same conditions to obtain plasma-free red blood cells.

A 1% erythrocyte suspension was prepared in normal saline. Aliquots of this suspension (10 mL) were incubated with varying concentrations of crude venom (30, 15, 7.5, 3.75, 1.575 and 0.7875 mg/mL) at 37 ± 1°C for 60 minutes. Following incubation, the reaction was terminated by the addition of 3 mL of chilled phosphate-buffered saline (PBS), and the mixtures were centrifuged at 2000 rpm for 10 minutes.

The absorbance of the supernatant was measured at 540 nm using a spectrophotometer. Complete haemolysis (100%) was determined using erythrocytes treated with distilled water, while erythrocytes incubated with normal saline served as the negative control (0% haemolysis).

Haemolytic activity was calculated as a percentage using the formula:

Percentage haemolysis =

$$\frac{A_{\text{sample}} - A_{\text{blank}}}{A_{\text{positive control}} - A_{\text{blank}}} \times 100$$

where:

A_{sample} = absorbance of test sample

A_{blank} = absorbance of saline control

$A_{\text{positive control}}$ = absorbance of distilled water-treated sample.

(Theakston & Reid, 1983; Gomes & De, 1999).

In vivo Toxicity Assessment

The acute systemic toxicity of *Sclerophrys regularis* venom was evaluated in mice following intraperitoneal administration, as described by Dong *et al.* (2003) with slight modifications. Ten mice were randomly assigned to two groups (n = 5 per group). Mice in the treatment group received a single intraperitoneal injection of the venom at a dose equivalent to four times the median lethal dose (4 × LD₅₀), while the control group received 0.2 mL of normal saline.

Following administration, the animals were closely observed for 24 hours for the development of clinical signs of toxicity and mortality. At the end of the observation period, two animals from each group were humanely euthanised and subjected to histopathological examination.

All procedures were conducted in accordance with Ahmadu Bello University animal care guidelines.

Histopathology

Two of the five mice from each group were sacrificed, and their Organs (kidney, brain, lungs, liver, spleen and heart) were harvested in 10% formalin and taken to the Anatomy Laboratory of Ahmadu Bello University, Zaria, for preparation of histology slides. Sections were embedded in paraffin according to routine histological processing. Sections of 6µm thickness were stained using haematoxylin and eosin (HE) as described by Bancroft & Steven (1982).

Data analysis

The LD₅₀ was determined using non-linear correlation analysis to plot percentage mortality against dose. Other data were analysed using One-Way Analysis of

Variance (ANOVA), and results were recorded as Mean ± SD. Data analysis was carried out using GraphPad Prism version 6.0 for Windows. The results were presented as histograms and tables, and values of $p \leq 0.05$ were considered to be statistically significant.

Results

Median lethal dose

The LD₅₀ was calculated using non-linear regression analysis to plot percentage mortality (%) against log dose (mg/kg). The log dose that corresponded to 50% mortality was 1.55. The antilog of which is 35.48. Hence the LD₅₀ was determined to be approximately 35.48 mg/kg as shown in Figure 1.

Fibrinolytic activity of the venom

There was a slight fibrinolytic effect, which was concentration-dependent, with venom concentrations of 30 mg/ml, 15 mg/ml and 7.5 mg/ml showing slightly higher fibrinolytic effects. These effects were however, not statistically significant (Table 1).

Haemolytic effects of the venom

Haemolytic effects were observed at all the venom concentrations used. The least haemolytic effects were observed at concentrations of 7.5 mg/mL and 3.75 mg/mL of the *Sclerophrys regularis* venom (Table 2).

Effects of venom on tissue histo-architecture

The venom of *Sclerophrys regularis* caused congestion in the lungs, kidneys, liver, heart and brain of poisoned mice. Additionally, the kidneys showed evidence of coagulative necrosis, vacuolations, dilation of the Bowman's space, tubular necrosis, and tubular degeneration. The heart showed

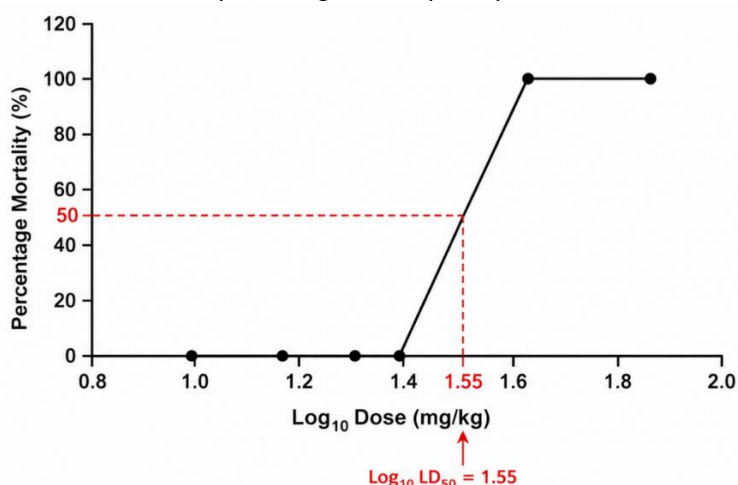


Figure 1: Median lethal dose of *Sclerophrys regularis* venom in mice using the up and down method

Table 1: Mean fibrinolytic effects of the venom of *Sclerophrys regularis*

Venom Concentration (mg/mL)	Mean Fibrinolytic Effect
30	0.0909 ± 0.0139
15	0.0909 ± 0.0121
7.5	0.0909 ± 0.0104
3.75	0.0833 ± 0.0156
1.875	0.0833 ± 0.0173
0.9375	0.0833 ± 0.0191

Table 2: Mean haemolytic effects of the venom of *Sclerophrys regularis*

Venom Concentration (mg/mL)	Mean Haemolytic Effect
30	2.146 ± 0.0970 ^a
15	1.211 ± 0.1507 ^b
7.5	0.548 ± 0.1403 ^c
3.75	0.566 ± 0.1351 ^c
1.875	1.344 ± 0.1368 ^b
0.9375	1.428 ± 0.1351 ^b

Means with different superscripts are statistically significant ($p < 0.05$) within column

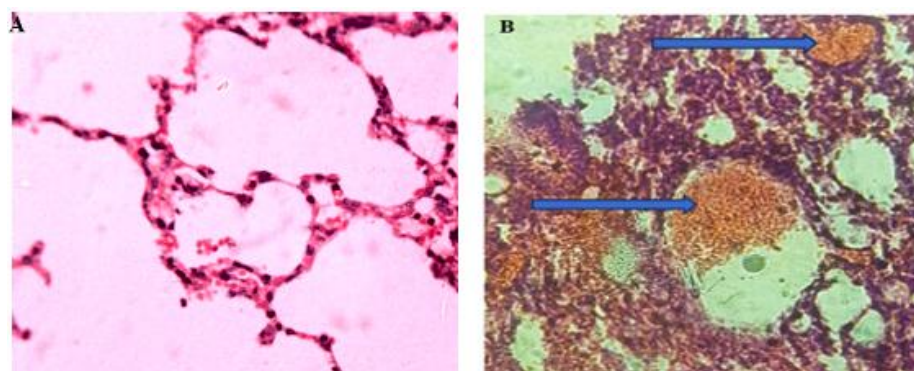


Plate I: A. normal lungs. B. Photomicrograph of lungs of envenomated rats: arrow showing congestion (H and E $\times 400$)

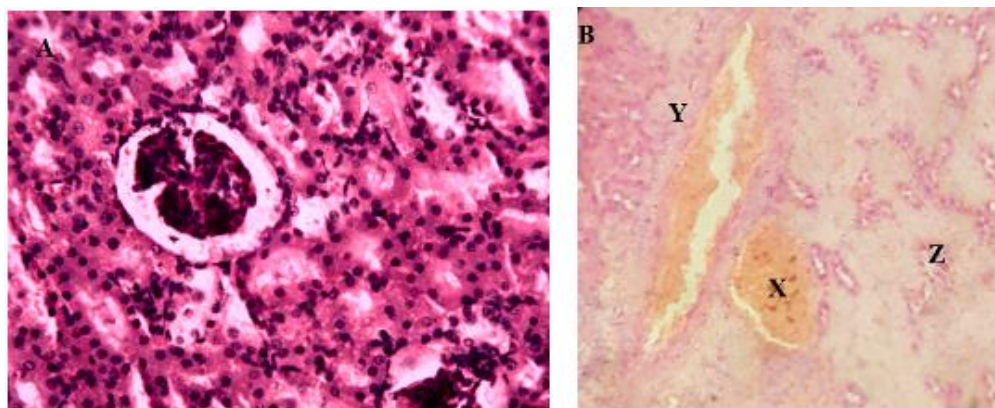


Plate II: A. normal kidney, B. Photomicrograph of the kidney of envenomated rats: X showing congestion, Y showing coagulative necrosis, and Z showing vacuolations, dilation of the bowman's space, tubular necrosis, and tubular degeneration (H and E $\times 400$)

evidence of haemorrhage, oedema and vacuolations. The spleen had massive edema and dilatation of trabeculae with evidence of depletion of red and white pulp. The congestion of the liver was in the central vein and vacuolation of hepatocytes was observed. Vacuolation, pseudocyst and vascular edema were also observed in the envenomated brain (Plates I – VI).

Discussion

The present study provides the first systematic toxicological characterization of *Sclerophrys regularis*

venom under experimental conditions representative of acute envenomation, generating data on lethal potency, haematotoxicity, and multiorgan histopathology. These findings add substantially to the limited available literature on African bufonid toxicology and establish a comparative framework against which future studies can be evaluated.

The determined LD_{50} of 35.48 mg/kg places *Sclerophrys regularis* venom within the moderately toxic range for members of the family Bufonidae.

This value is broadly consistent with previously reported LD_{50} values for related species. Qian *et al.*

(2015) reported an intravenous LD₅₀ of approximately 40 mg/kg for Bufo bufo-derived bufotoxin preparations in rodents. The slightly lower value obtained in the present study, using the intraperitoneal route, likely reflects both route-related differences in bioavailability and intraspecific variation attributable to geographic origin, as is well

established for venomous taxa (Calvete *et al.*, 2010). Geographic variation in venom composition is a well-recognised phenomenon in both snakes and toads, arising from differences in diet, ecological pressures, and local adaptations that influence the biosynthetic pathways responsible for toxin production (Casewell *et al.*, 2020). The primary toxic agents responsible for

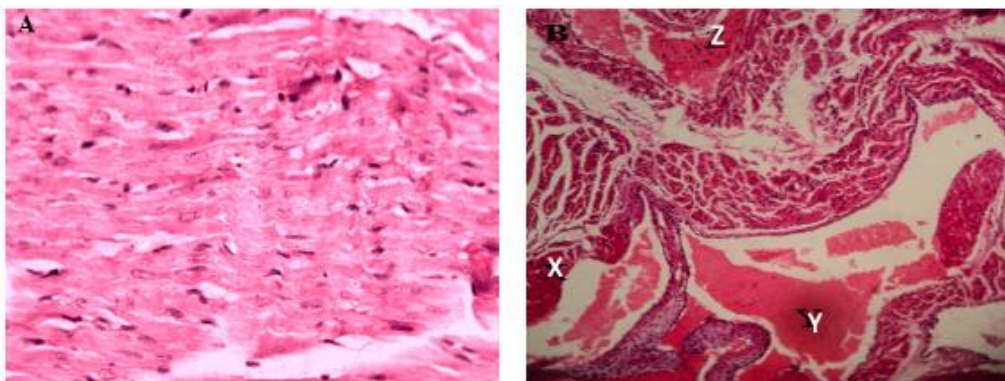


Plate III: A. Normal heart B. envenomated heart X showing haemorrhage, Y showing congestion, oedema, and Z showing necrosis (H and E ×400)

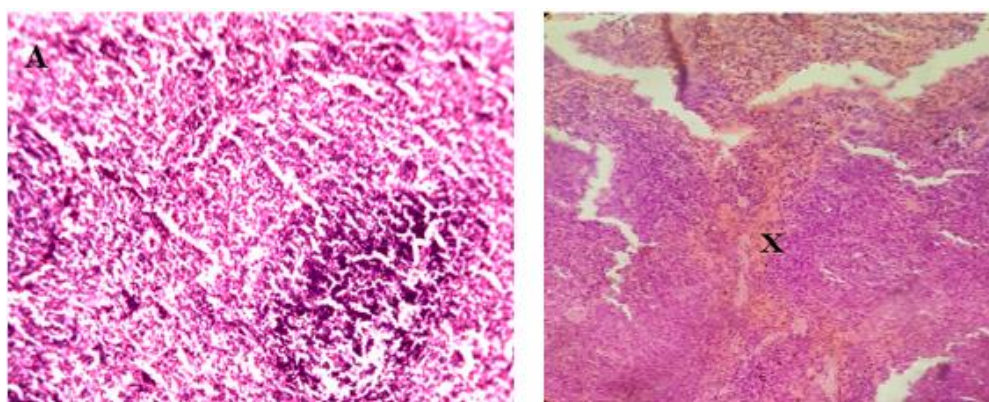


Plate IV: A. Normal spleen B. Photomicrograph of the spleen of envenomated rats: X showing massive edema and dilatation of trabeculae. There is a depletion of red and white pulp (H and E ×400)

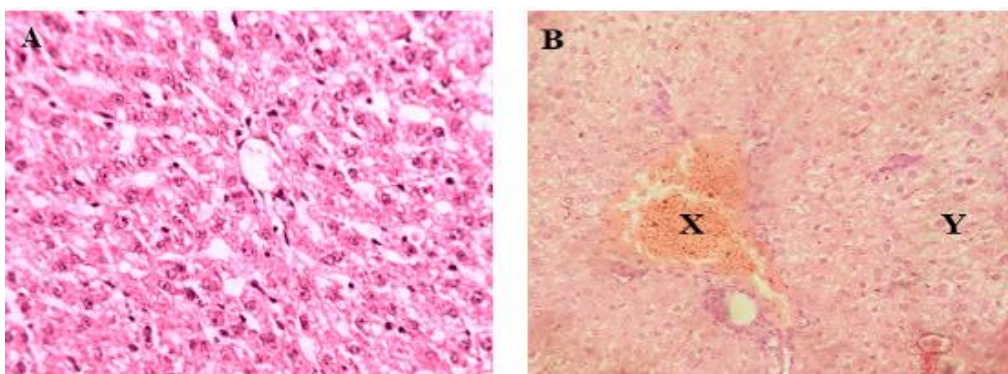


Plate V: A. Normal liver. B. Photomicrograph of the liver of envenomated rats: X showing congestion of central vein and Y showing vacuolation of hepatocytes (H and E ×400)

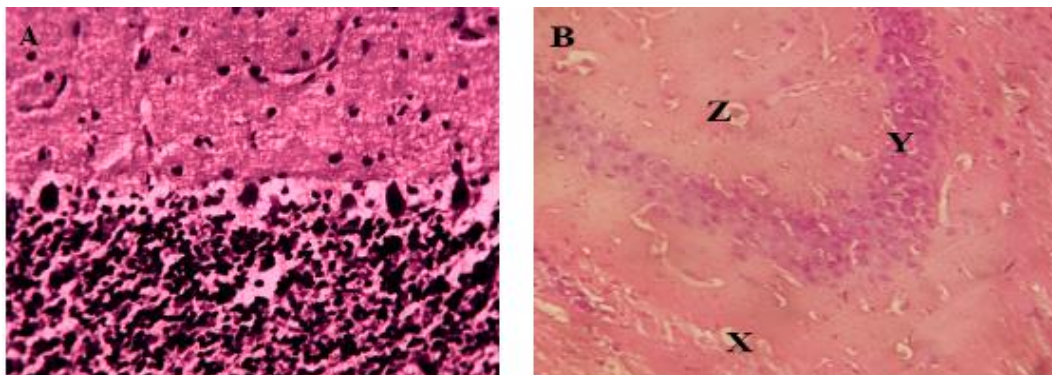


Plate VI: A. Normal brain. B. Photomicrograph of the brain of envenomated rats: X showing vacuolation, pseudocyst, Y showing congestion and Z Showing vascular edema (H and E $\times 400$)

the observed lethality are most likely the bufadienolides, which inhibit the Na^+/K^+ -ATPase pump, elevate intracellular calcium, and impair membrane ion transport in cardiomyocytes and neurons, consistent with mechanisms described across multiple bufonid species (Li *et al.*, 2021; Liu *et al.*, 2025).

The fibrinolytic assay revealed marginally elevated activity at higher venom concentrations that did not reach statistical significance, suggesting that direct modulation of the coagulation cascade is not a primary mechanism of *Sclerophrys regularis* venom toxicity. This contrasts with the fibrinolytic and procoagulant properties described for certain snake venoms (Chippaux, 2017; Warrell, 2019) and suggests that the haematotoxic potential of this toad venom operates through mechanisms other than direct enzymatic disruption of fibrin polymerization. Qian *et al.* (2015) and Candelario *et al.* (2020) have proposed that bufotoxin preparations affect the haemostatic system in variable ways depending on species and concentration, though neither study quantified fibrinolytic activity in the manner employed here. Li *et al.* (2021) noted that indirectly mediated haemostatic changes may arise from vascular injury and systemic inflammatory responses triggered by venom components rather than from primary enzymatic fibrinolysis. Accordingly, the modest fibrinolytic signal observed in this study may reflect a low-level, non-specific perturbation of the coagulation system secondary to cell membrane disruption, rather than a direct enzymatic effect of venom proteases.

The haemolytic activity of *Sclerophrys regularis* venom was demonstrable across all tested concentrations, with statistically significant differences between groups and peak disruption at 30

mg/mL. The observation that intermediate concentrations (7.5 and 3.75 mg/mL) produced relatively lower haemolytic activity than both higher and lower concentrations suggests a non-linear, potentially biphasic, dose-response relationship. Such patterns have been described for membrane-active toxins whose mechanism of action involves pore formation or lipid disruption at concentrations sufficient to aggregate and saturate membrane targets, with reduced per-molecule efficiency at intermediate concentrations owing to steric or thermodynamic constraints (Bhakdi *et al.*, 1984; Ponmalar *et al.*, 2021). The haemolytic findings of this study are consistent with those reported for *Duttaphrynus melanostictus* by Patnaik & Sahoo (2021), who demonstrated that venom from the Asian common toad increased erythrocyte osmotic fragility, impaired haemoglobin integrity, and induced oxidative stress characterized by increased glutathione production. These findings collectively indicate that erythrocyte membrane disruption and oxidative mechanisms are shared features of bufonid venom haematotoxicity, which may underlie some of the systemic clinical signs associated with clinical bufotoxycosis (Gadelha *et al.*, 2014). The haemolytic activity documented here also has broader implications for the proposed antimicrobial potential of bufonid secretions, as membrane-disruptive activity is mechanistically compatible with bactericidal or antifungal effects through analogous cytolytic action on microbial cell membranes (Qi *et al.*, 2018).

The histopathological evaluation constitutes the most substantive component of the present study and reveals the anatomical basis of the multisystem toxicity induced by *Sclerophrys regularis* venom. The widespread vascular congestion observed across the

lungs, kidneys, heart, liver, and brain is consistent with systemic haemodynamic failure, most likely mediated by the cardiotoxic and vasotonic components of the venom. Bufadienolides are known to elicit profound cardiovascular effects, including arrhythmia, reduced cardiac output, and peripheral vasoconstriction, all of which can result in the passive congestion of multiple organ systems (Asrorov *et al.*, 2023; Liu *et al.*, 2025). Ameh *et al.* (2019) and Yusuf *et al.* (2024) have similarly attributed multiorgan congestion and haemorrhage in envenomated animals to venom-mediated haemotoxicity and cardiovascular disruption, observations that align closely with the present findings.

The renal pathology documented in this study, encompassing coagulative necrosis, tubular degeneration, tubular necrosis, and dilation of Bowman's space, is particularly noteworthy. The kidney is one of the most vulnerable organs to systemic venom toxicity owing to its high perfusion rate and the concentration of toxins during excretion (Visith, 2006; Pinho *et al.*, 2008;). Coagulative necrosis of tubular epithelium represents a histopathological marker of acute tubular injury that has been extensively documented in the context of snake envenomation, where it correlates clinically with acute renal failure (Koh *et al.*, 2006; World Health Organization, 2010; Yusuf *et al.*, 2015). The presence of analogous lesions in *Sclerophrys regularis*-envenomated animals indicates that acute nephrotoxicity may be a clinically significant feature of bufotoxycosis in mammals, and this finding has direct relevance to the clinical management of envenomated patients and animals, where renal function monitoring and nephroprotective interventions may be warranted.

Cardiac pathology in the envenomated animals, manifesting as haemorrhage, myocardial oedema, and vacuolation, reflects the well-established cardiotoxic mechanism of bufadienolides acting through Na⁺/K⁺-ATPase inhibition and intracellular calcium overload (Li *et al.*, 2021; Asrorov *et al.*, 2023). Myocardial vacuolation is a recognized morphological consequence of calcium-mediated myofibrillar disruption and mitochondrial swelling in cardiomyocytes subjected to toxic injury. The splenic findings, particularly the depletion of red and white pulp with trabecular dilation, suggest immunological compromise and disruption of haematopoietic function, consistent with the haemolytic activity documented in vitro and with reported suppression of immune cell populations by bufadienolide compounds in experimental settings (Wang *et al.*,

2014). Hepatic central vein congestion and hepatocellular vacuolation indicate passive venous congestion secondary to right heart failure and direct hepatotoxic effects, a pattern that is recognized in clinical toxicology as a consequence of systemic cardiovascular collapse.

Among the most significant findings of this study are the cerebral histopathological changes, including neuronal vacuolation, pseudocyst formation, and vascular oedema. These lesions indicate that *Sclerophrys regularis* venom possesses meaningful neurotoxic potential, consistent with the known neuroactive properties of bufotenine and 5-methoxy-N,N-dimethyltryptamine (5-MeO-DMT), both of which are constituents of bufonid skin secretions and act as potent agonists of serotonin (5-HT) receptors in the central nervous system (Qi *et al.*, 2018; Liu *et al.*, 2025). Wang *et al.* (2014) and Bi *et al.* (2016) demonstrated that bufadienolides can overexcite and injure central neurons, potentially inducing neuroinflammatory cascades and structural damage. Ye *et al.* (2023) further showed that hippocampal neuronal death resulting from toad venom exposure is associated with deficits in spatial learning and memory in rodent models. These neurological effects provide a mechanistic basis for the psychedelic and neurological symptoms, including hallucinations, altered perception, seizures, and impaired consciousness, that have been reported in human cases of toad venom abuse and clinical bufotoxycosis (Belperio *et al.*, 2021). The documentation of corresponding neuropathological lesions in an African bufonid species is both novel and clinically significant, as it underscores the neurotoxic risk associated with exposure to *Sclerophrys regularis* in the regional context.

Collectively, the results of this study demonstrate that *Sclerophrys regularis* venom is a complex, multitoxin mixture capable of inducing concurrent cardiotoxic, haematotoxic, nephrotoxic, hepatotoxic, and neurotoxic injury in mammalian experimental models. The breadth of organ involvement observed following a single acute exposure underscores the clinical urgency of developing evidence-based management protocols for human and veterinary bufotoxycosis in regions where this species is prevalent. Furthermore, the demonstrated cytolytic, haemolytic, and membrane-active properties of the venom highlight its potential as a source of bioactive leads for antimicrobial, anticancer, and pharmacological research, consistent with a growing body of literature advocating for the biomedical exploitation of amphibian secretions (Lin *et al.*, 2021).

This study provides the first experimental toxicological characterization of *Sclerophrys regularis* venom, establishing an intraperitoneal LD₅₀ of 35.48 mg/kg in Swiss albino mice. The venom exhibited haemolytic activity across all tested concentrations, with a non-linear dose-response pattern, and demonstrated marginal fibrinolytic activity that was not statistically significant. Histopathological examination confirmed multiorgan toxicity affecting the kidneys, heart, liver, lungs, spleen, and brain, with lesions consistent with combined haemodynamic, cytotoxic, and neurotoxic mechanisms. These findings establish that *Sclerophrys regularis* venom poses a significant risk of acute multisystem injury and identifies the kidney and brain as particularly vulnerable targets. The data provide an empirical basis for future investigations into the molecular mechanisms of toxicity, the pharmacological potential of venom components, and the development of therapeutic countermeasures for bufotoxosis in the West African subregion.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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