



A Monthly e Magazine
ISSN:2583-2212

Popular Article

April, 2026 Vol.6(4), 1132-1138

Lantana camara as a Hepatotoxic and Photosensitizing Weed: Mechanism of Toxicity, Clinical Features, and Implications for Animal Health

Anjitha SV, Soorya Gayathri PV, Shafalika Sasi KTK, Kashmeera S Nair, Nandana Ramesh VP, Misriya KA, Ranjith D

College of Veterinary and Animal Sciences, Pookode, Wayanad, Kerala.

Corresponding author: ranjith946@gmail.com

doi.org/10.5281/ScienceWorld.20074309

Abstract

Lantana camara is a noxious and highly toxic weed widely distributed across tropical and subtropical regions, posing a serious threat to livestock health and productivity. It contains toxic pentacyclic triterpenoids (lantadenes), which are primarily responsible for its deleterious effects in ruminants. Ingestion of the plant leads to hepatotoxicity characterized by bile duct damage, cholestasis, and impaired liver function. Clinically, affected animals exhibit anorexia, icterus, constipation, and marked photosensitization due to the accumulation of phylloerythrin in the bloodstream. Biochemically, lantana poisoning is associated with a significant elevation of hepatic enzymes, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP), indicating liver injury. In addition to hepatic damage, renal involvement is evident through increased blood urea nitrogen (BUN) and creatinine levels, suggesting concurrent nephrotoxicity. The combined hepatic and renal dysfunction contribute to reduced productivity, increased morbidity, and occasional mortality in affected livestock. Poisonous plants such as *Lantana camara* and their secondary metabolites are responsible for considerable economic losses in the livestock sector worldwide. Understanding the toxicodynamics, clinical manifestations, and biochemical alterations associated with lantana poisoning is essential for early diagnosis, effective management, and prevention strategies.

Keywords: *Lantana camara*, photosensitization, hepatic toxicity.

Introduction

Lantana camara Linn. (Family: Verbenaceae) is a widely invasive, perennial shrub that was originally introduced as an ornamental plant but has since become a persistent and problematic weed across tropical and subtropical regions of the world. Introduced into India during the early nineteenth century, it has aggressively spread in pastures, forest margins, roadsides, and wastelands, posing a significant threat to livestock production systems and



biodiversity. The plant typically grows as a low, erect, or sub-scandent vigorous shrub, reaching a height of 2–4 meters. Its leaves are ovate, arranged in opposite pairs, bright green, rough in texture, finely pubescent, and possess serrated margins. When crushed, the leaves emit a characteristic pungent odour. The stem is often non-thorny or sparsely prickly. Flowering may occur seasonally from August to March or throughout the year under favourable environmental conditions such as adequate moisture and sunlight. The plant produces small, clustered flowers that vary in colour—red, pink, white, yellow, or mixed shades—followed by fleshy fruits that turn dark purple or black upon ripening. These fruits are readily consumed by birds, facilitating widespread seed dispersal, and are occasionally ingested by humans in certain regions.

The toxic potential of *Lantana camara* is attributed to the presence of pentacyclic triterpenoids known as lantadenes. The major toxic constituents include lantadene A, B, C, and D, while reduced lantadenes A and B occur in smaller quantities. Among these, lantadene A and B are primarily responsible for hepatotoxicity and photosensitization in grazing animals such as sheep, goats, cattle, and horses. These toxins exhibit structural similarity to cholesterol, and their absorption is believed to be associated with esterification processes like those involved in cholesterol metabolism. Following ingestion, lantadenes disrupt hepatic function by inducing intrahepatic cholestasis, impairing bile flow, and causing degeneration of bile canaliculi along with gallbladder dysfunction.

Clinically, lantana poisoning is characterized by anorexia, constipation or ruminal stasis, icterus, and marked photosensitization, commonly referred to as “pink nose disease” in cattle. The accumulation of phylloerythrin, a photodynamic agent derived from chlorophyll metabolism, leads to severe skin lesions upon exposure to sunlight. Hematological alterations include increased haematocrit, elevated red blood cell counts, and neutrophilia, accompanied by a reduction in platelet numbers. Biochemically, significant elevations in liver enzymes such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), and alkaline phosphatase (ALP) are observed, reflecting hepatic injury. In addition to its direct toxic effects on livestock, *Lantana camara* exerts allelopathic effects, as extracts from its roots, stems, leaves, and inflorescences inhibit the growth of surrounding vegetation, including ferns. This characteristic contributes to its invasive success and ecological dominance. Heavy outbreaks of lantana poisoning are often reported during drought conditions when the availability of palatable forage is limited, forcing animals to consume this otherwise avoided plant. The widespread distribution, high toxicity, and significant economic impact of *Lantana camara*



make it an important subject of study in veterinary toxicology, necessitating a comprehensive understanding of its toxic principles, pathogenesis, clinical manifestations, and preventive strategies.



Figure 1: Morphological characteristics of *Lantana camara*.

Poisoning due to *Lantana camara* represents a significant constraint to livestock production, particularly in tropical and subtropical regions where the plant is widely distributed. The impact of lantana on animal health and pasture ecology is two-fold. Firstly, it exerts direct toxic effects on grazing animals, primarily causing hepatotoxicity and secondary photosensitization. Secondly, it displays strong allelopathic properties that suppress the growth of desirable pasture species, thereby reducing the availability of palatable fodder and predisposing animals to accidental ingestion. The incidence of lantana poisoning varies from sporadic cases to severe outbreaks, often associated with environmental stress conditions such as droughts or floods, when normal forage becomes scarce. Under such circumstances, animals are forced to consume lantana leaves, which are otherwise avoided due to their pungent odor and unpalatable nature. Additionally, cases have been reported when livestock are transported from lantana-free areas to regions heavily infested with the plant, where unfamiliarity increases the risk of ingestion. Young, naive, or hungry animals are particularly susceptible. Clinically, lantana poisoning is characterized by progressive anorexia, constipation, ruminal stasis, dehydration, and reduced productivity. Affected animals may exhibit jaundice, weakness, and reluctance to move. One of the most distinctive features is photosensitization, manifested as erythema, edema, and necrosis of non-pigmented or sparsely haired skin, particularly in areas exposed to sunlight such as the muzzle, eyelids, ears, and teats. In cattle, this condition is often referred to as “pink nose disease.” Secondary complications such as skin cracking, ulceration, and fly infestation may further aggravate the condition.



From a pathological standpoint, lantana toxicity is associated with significant hepatic dysfunction and alterations in metabolic processes. Studies have demonstrated a reduction in hepatic mitochondrial protein content, along with notable changes in lipid composition, including increased cholesterol-to-protein and cholesterol-to-phospholipid ratios. These alterations reflect substantial cellular and subcellular disturbances in affected animals. Overall, lantana poisoning acts as a “double-edged sword” by simultaneously diminishing pasture quality and directly affecting animal health, leading to decreased productivity, increased morbidity, and economic losses in the livestock sector.

Mechanism of Toxicity

Ingestion of lantana foliage leads to hepatotoxicity and secondary photosensitization. Lantana exhibits its toxicity in animals through three phases: the gastrointestinal phase, the hepatic phase, and the post-hepatic phase. In lantana-poisoned animals, the rumen contents become more liquid, enhancing absorption. In sheep, absorption sufficient to cause toxicity occurs from both the small and large intestines, with the small intestine being quantitatively the most significant. It was observed that the passage of ingesta into the small intestine decreased markedly a few hours after lantana leaf powder was administered. The absorption of the toxin from all regions of the gastrointestinal tract is crucial for the progression of toxicity. When bile was diverted from the small intestine, the animals remained intoxicated, indicating that bile is not essential for the absorption of lantana toxins. After absorption, the toxins are transported to the liver via portal blood, as lymph drainage in sheep did not prevent intoxication. An intravenous dose greater than 3 mg/kg induced hepatic necrosis rather than the typical cholestatic lesions of lantana poisoning. A single intravenous dose of lantana induces only mild cholestasis, but if the dose is administered twice daily for several days, typical cholestasis of lantana poisoning occurs. Continuous absorption of the toxin is required for the disease to persist over a long period. Some evidence using guinea pigs and female rats as laboratory animal models is available for the biotransformation of lantadene A and reduced lantadene A to polar metabolites. Ingestion of lantana toxins causes paralysis of the gallbladder and closure of bile canaliculi, which likely leads to decreased bile flow during lantana poisoning. Cholestasis results in the regurgitation of bile, causing a marked increase in the levels of bilirubin and phylloerythrin (the biodegradation product of chlorophyll) in the blood. Both bilirubin and phylloerythrin bind to proteins and undergo photochemical reactions upon exposure to light, causing photosensitization and associated skin lesions.



Clinical Signs: Clinical signs include hepatotoxicity, cholestasis, and photosensitization. The hepatotoxins are present only in the leaves of the lantana plant. There is ruminal stasis, and animals go off-feed within a couple of hours of consuming lantana foliage, becoming severely constipated. The conjunctiva of the eyes and mucous membranes of the vagina and rectum become icteric. The eyelids swell, and fissures appear on the muzzle, ear tips, and non-hairy parts of the body. Terminal renal failure also occurs in affected animals. In light-skinned animals exposed to bright sunlight, photosensitization is also observed.



Figure 2: Photograph showing the signs and effects of *Lantana camara* poisoning in cattle.

Treatment and Management

Management of *Lantana camara* poisoning in livestock is primarily supportive and symptomatic, as no specific antidote is available. Early recognition and prompt intervention are critical to minimize the severity of toxicosis and improve clinical outcomes. The foremost step in management involves the immediate removal of affected animals from lantana-infested pastures to prevent further ingestion of the toxic plant. Animals should be shifted to shaded areas or housed indoors to reduce exposure to sunlight and limit the severity of photosensitization.

Rumen decontamination constitutes a key therapeutic approach in ruminants. This can be achieved by evacuation of rumen contents (rumenotomy or stomach tubing where feasible), followed by replacement with fresh, healthy rumen liquor obtained from donor animals. This procedure helps restore normal ruminal microflora and improves digestive function. Administration of adsorbents such as activated charcoal is recommended to bind residual toxins within the gastrointestinal tract and reduce systemic absorption. In sheep, activated charcoal is commonly administered orally, while in cattle it is given via stomach tube at a



dose rate of 5 g/kg body weight, typically suspended in water along with electrolyte solutions to correct dehydration and maintain fluid balance.

Supportive therapy plays a crucial role in recovery. Fluid therapy using isotonic electrolyte solutions is indicated to counter dehydration and support renal function. Hepatoprotective agents, including antioxidants and liver tonics, may be administered to mitigate hepatic damage and promote regeneration. Laxatives can be used judiciously to facilitate the elimination of toxins from the gastrointestinal tract. In cases exhibiting severe photosensitization, topical antiseptics and soothing agents should be applied to affected skin areas to prevent secondary infections, while systemic antibiotics may be considered if complications arise.

Nutritional management is equally important, with provision of easily digestible, high-quality feed to support recovery. Preventive strategies include regular monitoring and eradication of lantana from grazing areas, especially during periods of fodder scarcity, and gradual acclimatization of animals when transported to new environments. Collectively, these measures are essential to reduce morbidity, enhance recovery, and minimize economic losses associated with lantana poisoning in livestock.

Conclusion

Lantana Camara poisoning remains a significant toxicological concern in livestock, particularly cattle, sheep, and goats in tropical and subtropical regions, including India. The plant's hepatotoxicity, primarily attributed to pentacyclic triterpenoids such as lantadenes A and B, manifests as cholestasis, photosensitization, and severe jaundice, often leading to high mortality in untreated cases.

References:

- Sharma, O. P., Sharma, S., Pattabhi, V., Mahato, S. B., & Sharma, P. D. (2007). A Review of the Hepatotoxic Plant *Lantana camara*. *Critical Reviews in Toxicology*, 37(4), 313–352. <https://doi.org/10.1080/10408440601177863>.
- Sharma, O. P., Makkar, H. P. S., Dawra, R. K., & Negi, S. S. (1981). A review of the toxicity of *Lantana camara* (Linn) in animals. *Clinical Toxicology*, 18(9), 1077–1094. <https://doi.org/10.3109/15563658108990337>.
- Kato-Noguchi, H., & Kato, M. (2025). Compounds Involved in the Invasive Characteristics of *Lantana camara*. *Molecules* (Basel, Switzerland), 30(2), 411. <https://doi.org/10.3390/molecules30020411>.
- Pour, B. M., Latha, L. Y., & Sasidharan, S. (2011). Cytotoxicity and Oral Acute Toxicity Studies of *Lantana camara* Leaf Extract. *Molecules*, 16(5), 3663–3674. <https://doi.org/10.3390/molecules16053663>.



Khairan, K., Maulydia, N. B., Faddillah, V., Tallei, T. E., Fauzi, F. M., & Idroes, R. (2024). Uncovering anti-inflammatory potential of *Lantana camara* Linn: Network pharmacology and in vitro studies. *Narra J*, 4(2), e894. <https://doi.org/10.52225/narra.v4i2.894>.

