DOI: https://doi.org/10.52756/ijerr.2023.v33spl.004 ***Int. J. Exp. Res. Rev.*, Vol. 33: 30-40 (2023)**

Original Article | *Peer Reviewed* | *Open Access*

International Journal of Experimental Research and Review (IJERR)

ISSN: 2455-4855 (Online)
www.iaph.in
ISSN 2455-4855

An Exemplary Computational Approach to Investigate Lumpy Skin Disease in Indian Cattle

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Article History:

Received: 29th Jun., 2023
Accepted: 18th Sep., 2023
Published: 30th Sep., 2023

Keywords:
Differential equation, epidemiology, LSD, LSDV, mathematical model

How to cite this Article:
Deepak Kumar, Bhanu Sharma and Pooja Khurana (2023). An exemplary computational approach to investigate lumpy skin disease in Indian cattle. *International Journal of Experimental Research and Review*. 33: 30-40.
DOI: https://doi.org/10.52756/ijerr.2023.v33spl.004

Abstract: Lumpy Skin Disease (LSD) is a highly consequential infectious ailment that affects cattle caused by the Lumpy Skin Disease Virus (LSDV), which is a DNA virus classified under the Capripoxvirus genus of the Poxviridae family. This affliction presents a significant obstacle in the cattle industries of several Asian and African countries. Although the virus may be found in many physiological fluids and excretions, such as sperm, skin lesions are the primary source of infection. Hematophagous arthropods, such as biting flies, mosquitoes, and ticks, act as mechanical vectors for transmission. The geographical expansion of LSDV is believed to have been assisted by vector-borne transmission. However, a lack of quantitative understanding of the transmission of LSDV hinders the implementation of efficient disease control measures. Obstacles as mentioned earlier, this work focuses on developing a mathematical model to analyze the phenomenon of Lumpy Skin Disease, with a specific emphasis on its implications for the cattle sectors in African and Asian countries. The model encompasses a set of differential equations incorporating the influences of infection forces originating from sick and asymptomatic cattle, along with infected vectors. The research demonstrates a plausible correlation between Lumpy Skin Disease and increased death rates in cattle. Employing rigorous examination and computational modelling, a valuable understanding of the fundamental dynamics of disease outbreaks is obtained. The mathematical model shows potential for predicting the future transmission of Lumpy Skin Disease. This presents a prospective opportunity for understanding and alleviating the disease's effects on bovine populations. Moreover, the analysis reveals that the impact of direct contact transmission on the spread of LSDV during documented outbreaks was negligible, therefore providing valuable insights into the dynamics of disease transmission. As the model is rigorously analyzed to gain insights into its qualitative dynamics of epidemics, which are present in the numerical simulations, this mathematical model can be allowed to estimate the future spread of the disease.

Introduction

Lumpy skin disease (LSD) has been considered a severe infectious condition affecting cattle. It is triggered through the Lumpy skin disease virus (LSDV), a DNA virus of the Poxviridea family and genus Capripox virus. This illness is one of the most serious health issues afflicting the livestock industries of most Asian and African nations. The epidemiology, pathology, clinical symptoms, diagnosis, therapy, & and control of LSD in India are all well covered in this review paper (Choudhary, 2023). The spatiotemporal clusters, risk factors, and high-risk regions of outbreaks of Lumpy skin disease in Asia are thus provided by this study, which can aid in developing more effective disease prevention and control strategies (Li et. al, 2023). Although the virus is expelled through several bodily fluids and excretions, including sperm, skin sores are the most common infection causes. Thus, vulnerable hosts are mostly

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infected mechanically by hematophagous arthropods such as biting flies, mosquitoes, and ticks. The vector-borne form of LSDV transmission is thought to have aided in the virus's fast "geographic expansion"; nonetheless, a lack of quantitative knowledge concerning LSDV transmission has impeded effective disease control. Keeping this in mind, the focus of the study is on finding the transmission route of the disease, viz short and long transmission routes, with the help of a mathematical model. The system of non-linear differential equations has been formulated and analyzed considering the forces of infection from symptomatic and asymptomatic cattle and infected vectors.

Lumpy skin disease in cattle

It is caused by the lumpy skin disease virus (LSDV), of which the Neethling strain is a model. This DNA virus consists of around “150 kilobase pairs” and comparatively huge diameters ranging from 230 nm to 260 nm encased inside a "lipid envelope". It is a member of the family Poxviridae and genus Capripoxvirus and is thus linked congenitally with the viruses of Goat and Sheep pox (Buller et al., 2005; Bhanuprakash et al., 2006; Givens, 2018). The capsid/ nucleo-capsid of LSDV has an elliptical form and contains genome and lateral bodies.

Clinicopathology

The clinical signs of Lumpy skin disease include high temperature, satiation, runny nose, nasal discharge, sialorrhea, decreased body weight, swelling in lymph-nodes lacrimation, and reduced production of milk (Babiuk et al., 2008; Abutarbush et al., 2013; Tasioudi et al., 2016; Annandale et al., 2014). Moreover, the condition has been distinguished with rigid, elevated and confined bulges on the skin. These bulges are about 2-7 diameter that commonly appear on the back, neck, tail and legs following the onset of fever (Beard, 2016; Sevik & Dogan, 2017). Myiasis is increased by necrotic and ulcerative nodules (Beard, 2016). In rare cases, oedema of the legs and lameness were seen (Tuppurainen & Oura, 2012). LSDV can cause abortion, mastitis, and orchitis (Radostitis et al., 2006). Nodules, on the other hand, were not found in collapsed foetuses. (Sevik & Dogan, 2017). Respiratory abdominal swelling and congestion, along with papules everywhere in the GI tract and lungs, were discovered during the autopsy (Zeynalova et al., 2016). The snout, nasopharynx, laryngeal, windpipe, lip recesses, gums, browsing pad, digestive system, mammary glands, uterine tubes, testes & vagina may all be affected. Serious illness consequences included keratitis, diarrhoea, inflammation in the lungs, breast tissue infection, itching and pain (Tuppurainen et al., 2017; AlSalihi and Hassan, 2015).

Characteristic eosinophils intra-cytoplasmic inclusion bodies in corneocytes, vascular and lymphatic endothelial cells, phagocytic scavenger cells, and perivascular cells associated with spinosum cell ballooning can be found in histopathological examinations of skin nodules. Inflammatory cells such as macrophages, lymphocytes, and eosinophils have infiltrated affected areas' superficial dermal tissue. Furthermore, some patients may have widespread vasculitis and severe coagulative necrosis in the subcutaneous muscles.

Pathogenesis

Replication of the virus, viral load, rise in temperature, epidermal localization of the virus, and appearance of bulges all occur following LSDV infection (Constable et al., 2017). The following occurrences were observed experimentally after intradermal viral inoculation:

- Localized swelling of about 1-3 cm consisting of nodules 4-7 days post-infection (DPI) at the inoculation site.
- Viremia and dispersing of the viral content through the discharge from the oral and nasal cavity. This happens 6-18 DPI.
- The appearance of characterized bulges/nodules on the skin and regional lymphadenopathy, which occurs 7-19 DPI.
- 42 days after infection DPI: there is an occurrence of the LSDV (Coetzer, 2004)

There is a prevailing of lymphangitis and vasculitis in afflicted organs because of the viral replication within macrophages, endothelial cells, fibroblasts, and pericytes (Coetzer, 2004).

Epidemiology of LSDV

The appearance of the disease’s infection:

LSD is prevalent in Africa, mainly in the Sub-Sahara (Salihi, 2014). It expanded remarkably in the South-East areas of Europe, Russia, the Balkans, Kazakhstan, and Caucasus in 2012. Ground epidemics can range from severe and widespread infections with significant morbidity and death rates in a limited number of animals who are sick & no few/no fatalities documented. However, a more severe outbreak of LSDV occurred when the infection was first incorporated into the locality. However, it was demised after that because of the mass immunization procedures. Around 80% of the mortality rates surpassed at the times of epi-zootics while 20% in an indigenous region (Radostits et al., 2006).

Hosts and susceptibility

LSDV is mainly host-specific, except for some virus strains that can replicate in goats and sheep (domestic cattle and buffaloes). Despite this, there is no proof for

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the small ruminants to serve as the virus reservoir (Salihi, 2014; Tuppurainen, 2017). During LSD outbreaks, the field's most susceptible hosts were Asian water buffaloes and domestic cattle (Salihi, 2014). While their involvement is unknown, several wild species, including impala giraffe, are more susceptible to the infection resulting during exploratory investigations.

Origin of LSDV

Nodules that are created on the mucus membranes of the nose, mouth, eyes, abdomen, udder, and genitals, releasing sufficient viruses to serve as viral sources. Many scientifically afflicted sheep will become infectious and carriers of the virus; approximately 50 percent of the individuals exposed exhibit medical indications. LSD virus was found in saliva, sperm and skin nodules for about 11 days, 22 days and 33 days, respectively in deliberately exposed cattle, whereas it was not detected in the faeces or urine (Tuppurainen and Oura, 2012). Given that Capripox viruses are incredibly immune to chemical and biological circumstances, they can remain in lesions or scabs for extended periods and have a strong affinity for cutaneous tissues (Tageldin et al., 2014).

Disease transmission

LSD has been observed over most of Sub-Saharan Africa during rhythmic raining cycles, because of the abundance of particular arthropods genera (Tageldin et al., 2014). A study of the risk factors associated with the development of LSD in Ethiopia found that a warm and humid agro-climate, which supports a large vector population, was associated with a higher incidence of LSD (Babiuk et al., 2008). LSDV, according to various sources, can be mechanically transmitted by various hematophagous arthropod vectors. The sickness is widespread, with 50-60% attack rates in areas with significant and low mosquito populations and 5-15% morbidity in arid conditions with few mechanical vectors (Tageldin et al., 2014, Gari et al., 2010). Due to high viral loads in skin lesions, transmission by mechanical means of certain poxvirus organisms by insect vectors like Stomoxys calcitrans could happen. Pervasive blood-feeding worms, for example, mosquitoes and sand-fly species, have been linked to large-scale LSD outbreaks (Weiss, 1968). *Stomoxys calcitrans* and *Biomyia fasciata* were found to have the LSD virus after being fed to sick cows (Chihota et al., 2001). Female mosquitos of the species Aedes aegypti have been reported to mechanically transfer LSDV from infected calves to susceptible cattle (Chihota et al., 2003). A vector that feeds regularly and swaps hosts between feedings is more likely to transfer LSDV mechanically (Chihota et al., 2001). The virus has also been found in blood, nasal discharge, lachrymal secretion, sperm, and saliva, all of which are assumed to be the main routes of LSD transmission (Honhold et al., 2011). Potential transmission channels for the LSD virus include consuming nursing cow milk and using contaminated bull

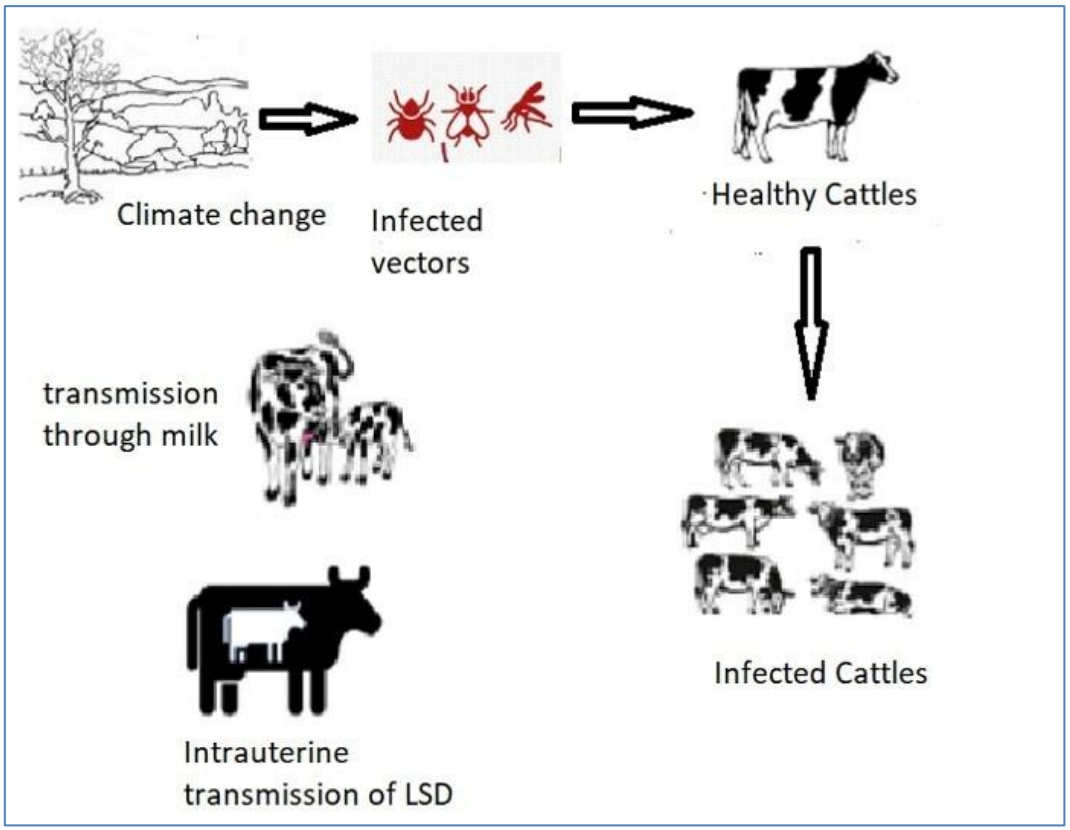

Figure 1. Routes of Lumpy Skin Disease transmission

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Table 1. Variables and parameter description

Parameter	Description	Values
S_c	Susceptible number of cattle who are liable to be infected with Lumpy virus at time.'t'	-
A_c	Asymptomatic infected number of cattle infected with Lumpy in the absence of any symptoms, however capable of infecting cattle and vector both at 't' time	-
I_c	Symptomatic infected no. of cattle infected with Lumpy and showing symptoms with the capability of spreading infection to vector and cattle both at 't' time	-
R_c	Recovered population that recovered either spontaneously or therapeutically from Lumpy infection	-
S_m	Susceptible number of mosquitoes that not yet infected with Lumpy but are capable of being infected by both asymptomatic and symptomatic infectious cattle at time.'t'	-
I_m	Number of infected pathogenic mosquitos with the ability to transmit Lumpy infection to vulnerable host (cattle) at 't' time.	-
Ω_c	recruitment terms for cattle populations (assumed susceptible)	0.03972
Ω_m	recruitment terms for mosquito populations (assumed susceptible)	0.96028
$\beta_1 b$, β_2 and β_3	Where $\beta_1 b$, β_2 and β_3 have been the probabilities of disease spread resulted because of I_m, I_c and A_c respectively, b represents the rate of a vector bite	β_1 =0.48014, b =0.96028, β_2 =0.03999, and β_3=0.47987
α_1 , α_2 and α_3	They represent the constants of positive saturation that help determine "the level at which the force of infection saturates" caused by I_m, I_c and A_c respectively.	α_1 =0.1, α_2 =0.5, and α_3=1.0
γ_c and σ_c	The population of the recovered cattle R_c is generated by the spontaneous recovery of both asymptomatic and symptomatic infectious individuals at rates, respectively.	γ_c =-0.4876 σ_c =-0.03998
θ	$0<\theta<1$ It represents the vulnerable cattle population that shifted to an asymptomatic population because of infection and $(1-\theta)$ the remaining cattle that shifted to a symptomatic population because of infection.	θ =-0.31991
μ_c and μ_m	The natural death rate of cattle and the natural death rate for both susceptible and infectious mosquito populations are represented by μ_c and μ_m	μ_c =-0.03984 μ_m =-1.44042
N_c	$N_c = S_c + A_c + I_c + R_c$	-
N_m	$N_m = S_m + I_m$	-

Source: Reddy *et al*., 2022; Mani *et al.*, 2022; Gupta *et al*., 2020; Calistri *et al*., 2020; Cohen *et al*., 2012.

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Table 2. The newest data on lumpy skin conditions show that Rajasthan and Punjab are among the worst-affected states in India, Money Control News (2022) and (Mani, 2022)

States	Cattle Susceptible (S_c)	Cattle infected with Lumpy without showing symptoms (A_c)	Cattle Infected (I_c)	Cattle Recovered (R_c)	Cattle deaths
Rajasthan	14381941	12981941	1399914	1335603	64311
Punjab	2500000	2325536	174464	156743	17721
Gujrat	6271274	6104306	166968	161111	5857
Himachal Pradesh	2400638	2311365	89273	84074	5199
Haryana	1932039	1821489	110550	107912	2638

sperm. This is due to the virus's ability to persist for extended periods in both milk and sperm.

Diagnosis of LSDV

Based on the disease's usual clinical symptoms, LSD is routinely diagnosed in the field. LSD should be examined clinically as soon as the prominent pampules on the skin appear, upon temperature rise, and when the lymph nodes swell. As a result, LSD's differential diagnosis depends upon various therapeutic manifestations. Additionally, sub-clinical and minor variants require rapid and consistent lab assessments for diagnostic purposes. Real-time polymerase chain reaction, also known as Rt PRC, has been considered the most common method for detecting LSD (Kholy et al., 2008). Other methods, like Serological diagnosis, have also been preferred as diagnostic approaches (Abdulqa et al., 2016).

Lumpy virus disease in India

First Lumpy Wave (2019-2020):

The initial epidemics occurred in Odisha in 2019, with more significant morbidity but extremely low fatality. The epidemic spread to adjacent states as well, including all southern states. However, the sickness was minor, and only a few cases of dermal farm disease were documented in 2019-2020 (Reddy et al., 2022).

Second Lumpy wave (2020-2021):

Several southern states, as well as other central regions, were hit, although the virus was a largely dermal farm and killed considerably more people in Maharashtra than in other affected areas, including MP and UP (Reddy et al., 2022).

Third Lumpy Wave (2021-2022):

The disease has become further contagious. During this wave, the "respiratory form of the disease was more prevalent, along with the dermal form", and the highest mortality was reported from states, particularly northern India, where there is a higher concentration of stary or owned cattle. Comorbidities in these stary and unowned animals, as well as in animal shelters, contributed to death, which was primarily related to management techniques (Reddy et al., 2022; Mani et al., 2022; Gupta et al., 2020; Calistri et al., 2020; Cohen et al., 2012).

Methodology

Very often, the differential equations arising in mathematical biosciences can be solved only with the help of computers. In some cases, the direct simulation of the biological situation on a computer without the intervention of a mathematical model may be necessary.

Mathematical Model

$$\frac{dS_c}{dt} = \Omega_c - \left(\frac{\beta_1 b I_m}{1+\alpha_1 I_m} + \frac{\beta_2 I_c}{1+\alpha_2 I_c} + \frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) S_c - \mu_c S_c \quad \text{.....(1)}$$

$$\frac{dA_c}{dt} = \theta\left(\frac{\beta_1 b I_m}{1+\alpha_1 I_m} + \frac{\beta_2 I_c}{1+\alpha_2 I_c} + \frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) S_c - (\gamma_c + \mu_c) A_c \quad \text{.....(2)}$$

$$\frac{dI_c}{dt} = (1-\theta)\left(\frac{\beta_1 b I_m}{1+\alpha_1 I_m} + \frac{\beta_2 I_c}{1+\alpha_2 I_c} + \frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) S_c - (\sigma_c + \mu_c) I_c \quad \text{.....(3)}$$

$$\frac{dR_c}{dt} = \gamma_c S_c + \sigma_c I_c - \mu_c R_c \quad \text{... (4)}$$

$$\frac{dS_m}{dt} = \Omega_m - \left(\frac{\beta_2 b I_c}{1+\alpha_2 I_c} + \frac{\beta_3 b A_c}{1+\alpha_3 A_c}\right) S_m - \mu_m S_m \quad \text{....(5)}$$

$$\frac{dI_m}{dt} = \left(\frac{\beta_2 b I_c}{1+\alpha_2 I_c} + \frac{\beta_3 b A_c}{1+\alpha_3 A_c}\right) S_m - \mu_m I_m \quad \text{....(6)}$$

Analysis

Stability analysis of model

The Jacobians of equation (1-6) of the infection disease is given by

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$$J=\begin{bmatrix} -\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}\right)-\mu_c & -\frac{\beta_1 S_c}{1+\alpha_1} & -\frac{\beta_2 S_c}{1+\alpha_2} & 0 & 0 & -\frac{\beta_1 bS_c}{1+\alpha_1} \\ \theta\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) & \frac{\theta\beta_1 S_c}{1+\alpha_1}-(\gamma_c+\mu_c) & \frac{\theta\beta_2 S_c}{1+\alpha_2} & 0 & 0 & \frac{\theta\beta_1 b}{1+\alpha_1} \\ (1-\theta)\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) & \frac{(1-\theta)\beta_1 S_c}{1+\alpha_1} & \frac{(1-\theta)\beta_2 S_c}{1+\alpha_2}-(\sigma_c+\mu_c) & 0 & 0 & \frac{(1-\theta)\beta_1 b}{1+\alpha_1} \\ \gamma_c & 0 & \sigma_c & 0 & 0 & 0 \\ 0 & -\frac{\beta_1 bS_m}{1+\alpha_1} & -\frac{\beta_2 bS_m}{1+\alpha_2} & 0 & -\left(\frac{\beta_2 bI_c}{1+\alpha_2 I_c}+\frac{\beta_3 bA_c}{1+\alpha_3 A_c}\right)-\mu_m & 0 \\ 0 & \frac{\beta_1 bS_m}{1+\alpha_1} & \frac{\beta_2 bS_m}{1+\alpha_2} & 0 & \left(\frac{\beta_2 bI_c}{1+\alpha_2 I_c}+\frac{\beta_3 bA_c}{1+\alpha_3 A_c}\right) & -\mu_m \end{bmatrix}$$

The characteristic equation for the governing equation is given by $|J-\lambda I|=0$.

$$|J-\lambda I|=\begin{bmatrix} -\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}\right)-\mu_c-\lambda & -\frac{\beta_1 S_c}{1+\alpha_1} & -\frac{\beta_2 S_c}{1+\alpha_2} & 0 & 0 & -\frac{\beta_1 bS_c}{1+\alpha_1} \\ \theta\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) & \frac{\theta\beta_1 S_c}{1+\alpha_1}-(\gamma_c+\mu_c)-\lambda & \frac{\theta\beta_2 S_c}{1+\alpha_2} & 0 & 0 & \frac{\theta\beta_1 b}{1+\alpha_1} \\ (1-\theta)\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}\right) & \frac{(1-\theta)\beta_1 S_c}{1+\alpha_1} & \frac{(1-\theta)\beta_2 S_c}{1+\alpha_2}-(\sigma_c+\mu_c)-\lambda & 0 & 0 & \frac{(1-\theta)\beta_1 b}{1+\alpha_1} \\ \gamma_c & 0 & \sigma_c & 0-\lambda & 0 & 0 \\ 0 & -\frac{\beta_1 bS_m}{1+\alpha_1} & -\frac{\beta_2 bS_m}{1+\alpha_2} & 0 & -\left(\frac{\beta_2 bI_c}{1+\alpha_2 I_c}+\frac{\beta_3 bA_c}{1+\alpha_3 A_c}\right)-\mu_m-\lambda & 0 \\ 0 & \frac{\beta_1 bS_m}{1+\alpha_1} & \frac{\beta_2 bS_m}{1+\alpha_2} & 0 & \left(\frac{\beta_2 bI_c}{1+\alpha_2 I_c}+\frac{\beta_3 bA_c}{1+\alpha_3 A_c}\right) & -\mu_m-\lambda \end{bmatrix}$$

The matrix gives Routh-Hurwitz's principle for the characteristic equation. By calculating numerical values, the results also do not satisfy the inequalities. So, the given system is not locally asymptotically stable.

Mathematical Analysis

Since the model consisting (of equations from 1 to 6) observes cattle and vector populations, its every connected "parameters are non-negative". It is, therefore, necessary to demonstrate that each of the model's state variables are non-negative.

The system of equations is non-linear, where the non-linear forces of infection induced by infection, symptomatic and asymptomatic cattle are given respectively by noting that α_1 α_2 α_3 have been considered as constants of positive saturation which help in regulating "the level at which the force of infection saturates."

The choice of this approach is driven by the notion that the number of viable interactions (sexual or vector transmission) among vulnerable and infected people can reach saturation at an elevated pathogenic level because of infected people's chaos.

The solutions $S_c(t), A_c(t), I_c(t), R_c(t)$, $S_m(t), I_m(t)$ of lumpy skin disease model (1) with non-negative preliminary information $S_c(0), A_c(0), I_c(0), R_c(0), S_m(0), I_m(0)$ abide non-negative always when "$t>0$" writing the model's first equation as:

$$\frac{dS_c}{dt}+\left(\frac{\beta_1 bI_m}{1+\alpha_1 I_m}+\frac{\beta_2 I_c}{1+\alpha_2 I_c}+\frac{\beta_3 A_c}{1+\alpha_3 A_c}+\mu_c\right)S_c\geq 0 \quad \text{……..(7)}$$

So that

$$\frac{d}{dt}\left[S_c(t)\exp\left(\sum_0^t \frac{\beta_1 bI_m(\xi)}{1+\alpha_1 I_m(\xi)}+\frac{\beta_2 I_c(\xi)}{1+\alpha_2 I_c(\xi)}+\frac{\beta_3 A_c(\xi)}{1+\alpha_3 A_c(\xi)}+\mu_c\right)\right]\geq 0 \quad \text{…(8)}$$

Integrating equation (8)

$$S_c(t)\geq S_c(0)\exp\left[-\int_0^t \frac{\beta_1 bI_m(\xi)}{1+\alpha_1 I_m(\xi)}+\frac{\beta_2 I_c(\xi)}{1+\alpha_2 I_c(\xi)}+\frac{\beta_3 A_c(\xi)}{1+\alpha_3 A_c(\xi)}+\mu_c\right]\geq 0 \quad \text{..(9)}$$

Likewise, it has been conveyed about other certain variables, including $A_c(t), I_c(t), R_c(t), S_m(t), I_m(t)$ are non-negative for all t>0

Considering the biologically attainable region as described herein,

$$D=D_c\times D_m\subset R_+^{\,4}\times R_+^{\,2}, where$$

$$D_c=\left\{\left(S_c, A_c, I_c, R_c\right)\in R_+^{\,4}: N_c\leq\frac{\Lambda_c}{\mu_c}\right\}$$

$$D_m=\left\{\left(S_m, I_m\right)\in R_+^{\,2}: N_m\leq\frac{\Lambda_m}{\mu_m}\right\}$$

Now, it has been manifested here about the positive invariant nature of Region D.

"The rate of change of the total population is given by."

$$\frac{dN_c}{dt}=\Omega_c-\mu_c N_c$$

Which solving yields

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$N_c(t) = N_c(0)\,exp(-\mu_c(t)) + \frac{\Omega_c}{\mu_c}(1 - exp(-\mu_c(t))\ldots(10)$

A similar approach for the total vector population gives

$N_m(t) = N_m(0)\,exp(-\mu_m(t)) + \frac{\Omega_m}{\mu_m}(1 - exp(-\mu_m(t))\ \ldots..(11)$

It follows that $N_c \to \frac{\Omega_c}{\mu_c}\ N_m \to \frac{\Omega_m}{\mu_m}$ as $t \to \infty$ …..(12)

In particular $N_m \le \frac{\Omega_m}{\mu_m} \quad if \quad N_m(0) \le \frac{\Omega_m}{\mu_m}$, …..(13)

And $N_m \le \frac{\Omega_m}{\mu_m} \quad if \quad N_m(0) \le \frac{\Omega_m}{\mu_m}$ …….. (14)

Hence, "Region D" can be represented as a positive invariant region.

Therefore, it is adequate for studying the dynamical interactions in region D of the proposed model, where it could be considered as an epidemiological and numerically well-presented model.

Results and Discussion

The projection for Rajasthan's transmission pattern is based on the abovementioned methodology. When sick animals are around, the outbreak peaks and then exponentially drops. The impact of various measures on the outbreak of lumpy skin disease is being discussed.

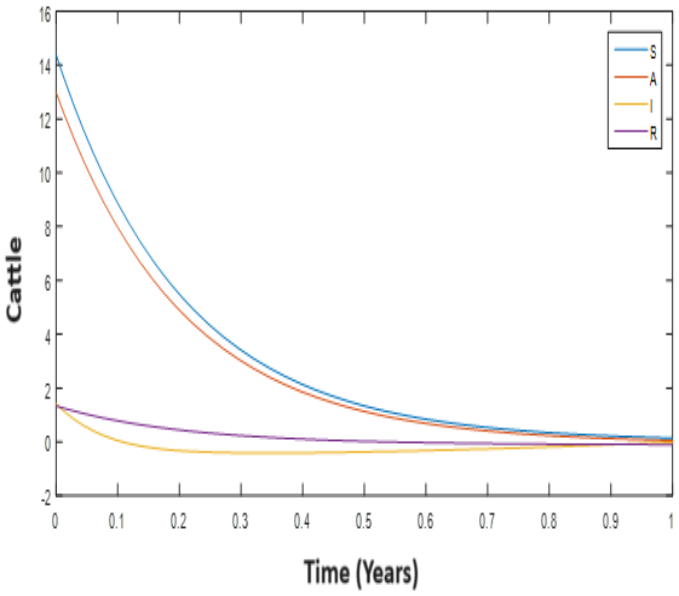

Figure 2. Simulated Curve (Cattel × 105 vs. time) to investigate Lumpy Skin Disease of cattle of Rajasthan where S= Susceptible; A= Asymptomatic; S=Symptomatic; and R=Recovered

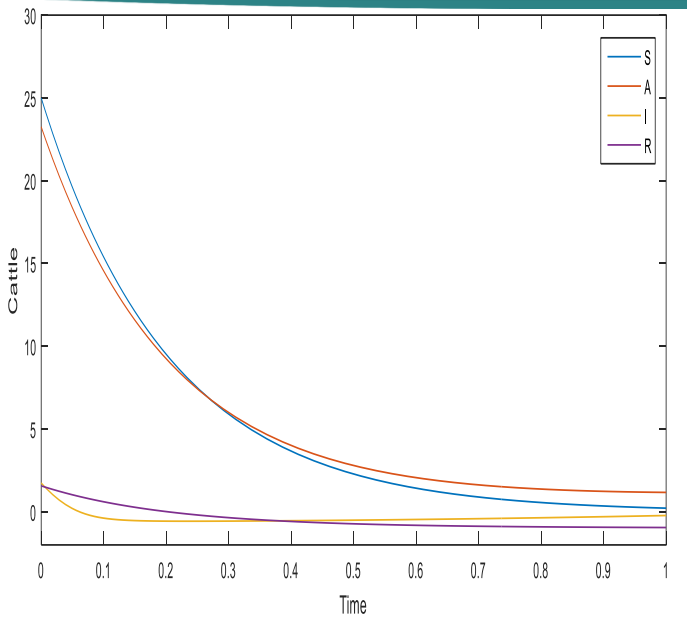

Figure 3. Simulated Curve (Cattel × 105 vs. time) to investigate Lumpy Skin Disease of cattle of Punjab where S= Susceptible; A= Asymptomatic; S=Symptomatic; and R=Recovered

Given the figure, the expectation for transmission design in Punjab. Through Lumpy skin disease's exponential decline, the outbreak reaches its peak with approximately infected cattle. The impact of specific actions on the feast of Lumpy Skin Disease is discussed.

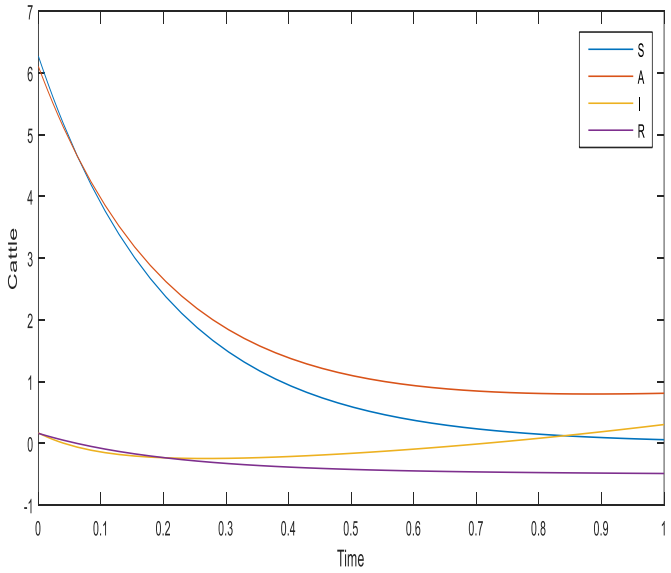

Figure 4. Simulated Curve (Cattel × 105 vs. time) to investigate Lumpy Skin Disease of cattle of Gujarat where S= Susceptible; A= Asymptomatic; S=Symptomatic; and R=Recovered

DOI: https://doi.org/10.52756/ijerr.2023.v33spl.004

The expectation for transmission design in Gujrat is shown in the figure. The outbreak of lumpy skin disease reaches its peak with approximately infected cattle despite its exponential decline. The effect of explicit activities on the banquet of Knotty Skin Sickness is the topic of conversation.

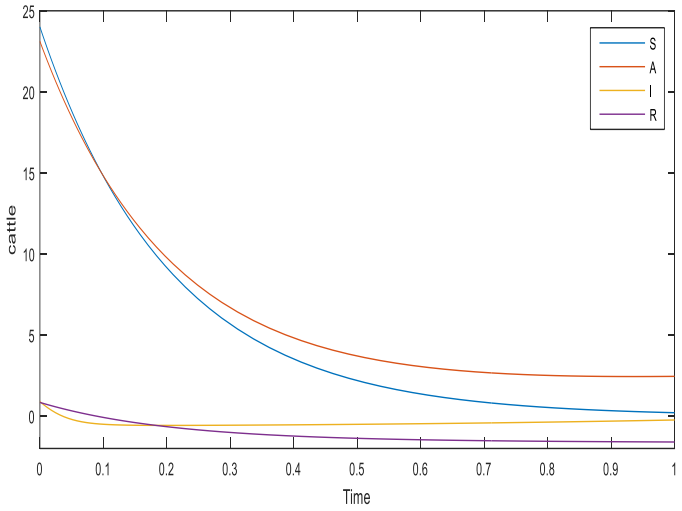

Figure 5. Simulated Curve (Cattel × 105 vs. time) to investigate Lumpy Skin Disease of cattle of Himachal Pradesh where S= Susceptible; A= Asymptomatic; S=Symptomatic; and R=Recovered

The simulation for transmission patterns in Himachal Pradesh is shown in Figure 5. The outbreak peaks with around infected cattle trough Lumpy Skin Disease declines exponentially. There is a discussion to judge the influence of specific actions on the feast of Lumpy Skin Disease.

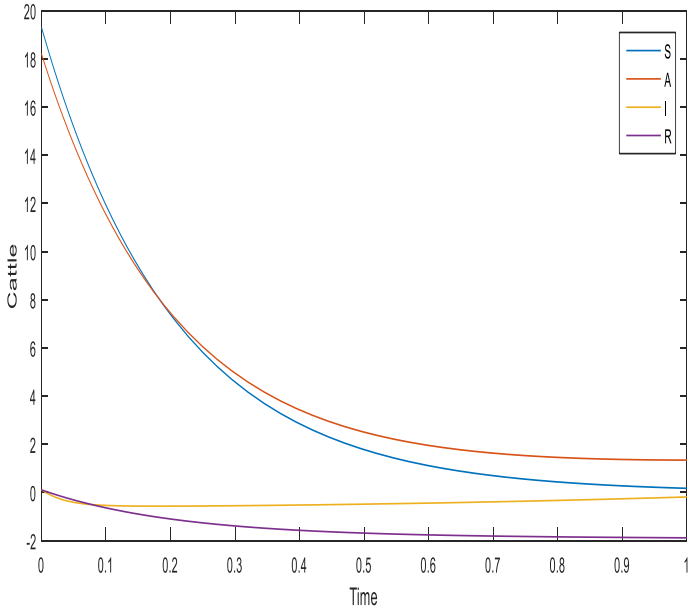

Figure 6. Simulated Curve (Cattel × 105 vs. time) to investigate Lumpy Skin Disease of cattle of Haryana where S= Susceptible; A= Asymptomatic; S=Symptomatic; and R=Recovered

The prognosis for the Haryana transmission pattern is based on the above graph. When sick animals are around, the epidemic peaks and then rapidly drops. The impact of various measures on the outbreak of lumpy skin disease is being discussed.

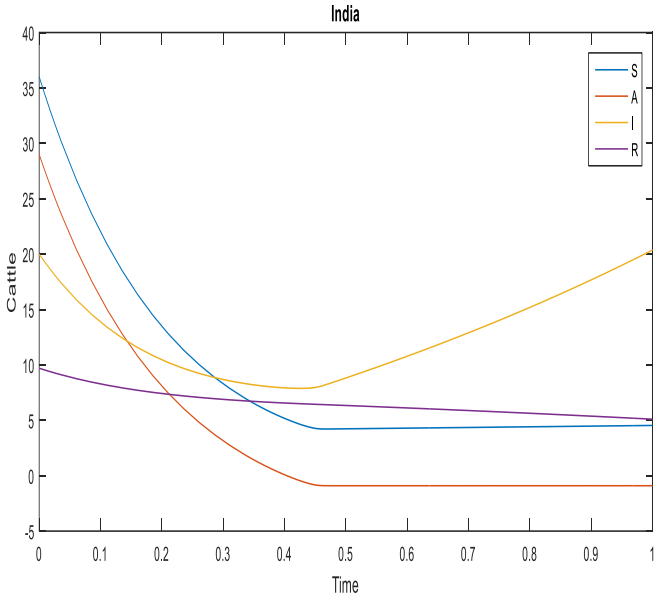

Figure 7. Simulated Curve (Cattel × 105 vs. time) to investigate Lumpy Skin Disease of cattle of India where S= Susceptible; A= Asymptomatic; S=Symptomatic; and R=Recovered

Based on the above figure, the prediction for transmission pattern in India. The outbreak peaks with around infected cattle through Lumpy Skin Disease declines exponentially. There is a discussion to judge the influence of specific actions on the feast of Lumpy Skin Disease.

Our computational technique for investigating the spread of Lumpy Skin illness (LSD) across India yields important insights into the dynamics of this illness and its potential impact on India's bovine herds. The simulated curves showed that the LSD epidemic peaks when ill animals are present, then rapidly declines on an exponential curve in each of the regions tested (Rajasthan, Punjab, Gujarat, Himachal Pradesh, Haryana, and India as a whole). The ramifications of these results for disease preventive and control efforts are substantial.

The Lumpy Skin Disease Virus (LSDV) poses a significant threat to the health of the cattle industries across Asia and Africa, particularly in India. Fever, lack of appetite, nasal discharge, salivation, swollen lymph nodes, reduced milk production, weight loss, and even mortality are just some of the clinical indications of the condition that can result in significant monetary losses. Large numbers of cattle have died or become sick as a result of LSDV outbreaks in the field.

The research shows that the impact of direct contact transmission of LSDV on viral replication during

DOI: https://doi.org/10.52756/ijerr.2023.v33spl.004

reported outbreaks appears to be small. This points to the importance of other causes, such as vector-borne transmission, in the propagation of the disease. To effectively develop control measures, an appreciation of these transmission dynamics is essential.

The positive invariant region (D) that we found is useful for studying the model's dynamics in discrete areas; this has important applications in epidemiological modelling. As a result, the dynamics of illness within a specific region may be represented in a way that is both simple and accurate, making for easier comprehension and analysis.

Our computational technique has elucidated LSD transmission patterns in Indian cattle, drawing attention to the necessity for focused management strategies to lessen the catastrophic effects of this illness. We can work towards lowering the burden of Lumpy Skin Disease in cow herds across India and beyond by learning more about how LSDV spreads and pinpointing places where treatments may be most successful.

Conclusion

Lumpy skin disease, produced by the lumpy skin disease virus, is one of the most severe health issues afflicting the cattle industries of most African and Asian nations. Clinical symptoms include fever, inappetence, nasal discharge, salivation and lachrymation, enlarged lymph nodes, a considerable decrease in milk production, loss of body weight, and death. Field outbreaks of Lumpy virus caused significant morbidity and mortality, and a lack of quantitative understanding of LSDV transmission has hampered disease prevention. A mathematical framework of non-linear equations was developed and tested to investigate the transmission of lumpy virus. Moreover, building a positively invariant area D is sufficient for investigating the dynamics of the model in region D, where the model may be considered an epidemiological model. To facilitate comprehension, a pictorial solution has also been provided. According to the findings of this investigation, direct contact transmission of LSDV had only a minimal influence on viral propagation during the outbreak reported. The findings presented here contribute to a deeper understanding of the factors influencing the spread of Lumpy Skin Disease, and they emphasize the importance of considering specific measures to mitigate its impact. With this knowledge, we can better inform disease control strategies and work towards reducing the burden of Lumpy Skin Disease in cattle populations in India and beyond.

Conflict of interest

The authors declare no conflict of interest.

Funding information

No Funding Agency provided any financial support.

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How to cite this Article:

Deepak Kumar, Bhanu Sharma and Pooja Khurana (2023). An exemplary computational approach to investigate lumpy skin disease in Indian cattle. *International Journal of Experimental Research and Review*. 33: 30-40.

DOI: https://doi.org/10.52756/ ijerr.2023.v33spl.004