FUDMA Journal of Sciences (FJS)
ISSN online: 2616-1370
ISSN print: 2645 - 2944
Vol. 7 No. 1, February, 2023, pp 311 - 318
DOI: https://doi.org/10.33003/fjs-2023-0701-2066

DYNAMICS AND CONTROL OF BRUCELLOSIS IN HUMAN AND LIVESTOCK: PUBLIC HEALTH EDUCATION, TREATMENT AND VACCINATION

***[1]Mohammed Baba Abdullahi and [2]Amiru Sule**

[1]Department of Mathematics/ Statistics, School of Mathematics and Computing, Kampala International University, Kampala, Uganda.
[2] Department of Mathematical Sciences, Faculty of Science, Federal University Gusau, Zamfara State

*Corresponding authors' email: kutigibaba@gmail.com

ABSTRACT
Brucellosis is a multifaceted zoonotic infection with vital epidemiological, economic, and global health effect, principally for human and Livestock populations within developing nations. In this paper a dynamic model of livestock-to-human transmission of the disease is developed. Model investigation is carried out to obtain and establish the stability of the equilibrium points. The basic reproduction number $\Re_0$ is calculated and the conditions under which brucellosis can be cleared in the both populations are established. Then, optimal control approach to establish the required conditions for the optimality of the disease eradication or control are applied. Public health education for humans and vaccination for susceptible livestock and treatment for infected humans and livestock. Numerical simulations show the dynamics of disease transmission and the effect of the control strategies.

Keywords: Dynamics, Control, Brucellosis, Livestock, Public Health Education, Treatment, Vaccination

INTRODUCTION

Brucellosis is a zoonotic sex infection known as Mediterranean relax heat, wave heat, or waveform heat (Corbel, 2006). It is distinguished by its broad host range, tough infectivity, and effort in drastic treatment following infection (Akhvlediani et al 2017). It also seriously affects the economy, society, and public health. As a result, it is included in the World Organization for Animal Health as one of the transmissible illnesses that must be reported. It is recognized as a second type of animal disease in China (Akhvlediani et al 2017). The disease refers to zoonoses caused by gram-negative bacteria of the genus Brucella. The disease has a global distribution and affects both economically important domestic animals and a diverse spectrum of wild species (Godfroid et al 2011). Brucellaabortus, B. melitensis, and B. suis are the species with the greatest impact on domestic livestock productivity and human health (Godfroid et al 2011), and while they preferentially infect cattle, small ruminants, and swine, respectively, cross-infections may be significant in mixed husbandry systems or at the livestock-wildlife interface (Godfroid et al 2013). Despite being eradicated from cattle and small ruminants in a few developed countries, brucellosis remains widespread throughout most of the world (Moreno, 2014).

The condition affects over 500,000 people each year (Shevtsova et al 2016). It is usually transferred in animals by either direct contact between a susceptible and an infectious animal or indirectly, i.e., when a susceptible animal ingests contaminated forages or excrement containing large amounts of bacteria, which is generally emitted by infected animals (Zhang, 2014). However, in humans, the majority of infections are caused by direct or indirect contact with infected animals or the eating of raw animal products such as unpasteurized milk or cheese (Al-Tawfiq, & AbuKhamsin 2009). Because human-to-human transmission of the disease is extremely infrequent (Godfroid 2002), efficient control of brucellosis in animals can lead to the eventual management of human brucellosis. Some experts believe that by combining vaccination with test-and-slaughter programs, brucellosis in animals can be eradicated (Morris 2013).

Miscarriage in cows and orchitis in bulls are the most visible symptoms of brucellosis. Furthermore, it frequently causes arthritis in the knee and wrist (Corbel 1997). Direct contact with infected animals and indirect infection with bacteria in the environment are the routes of transmission (Ainseba, Benosman, and Magal 2010). It can also be broken into three parts. The first method of transmission is by skin contact, such as direct contact with droppings, vaginal secretions, and vaginal delivery content of diseased animals. It may also be exposed indirectly to the environment and objects contaminated by sick animals. The second method infections enter the body is through the digestive tract, such as through contaminated food, water, or milk. The respiratory tract is the final pathway of transmission (Brouwer et al 2017).

Many studies (Zinsstag et al, 2015, Zhou et al 2018, Hou et al, 2013, De Souza et al 2016, Ainseba, Benosman & Magal, 2010 & Abatih et al 2015) explored the disease's causes, complications, and other aspects. The present work exposes the advances in comparison to the previous literature in both human and livestock, as well as the infection of the environment by diseased human and animals. We also consider livestock in general, rather than splitting them as previous models have.

Model Development

A model of brucellosis control develops using the deterministic compartmental modeling approach. The whole livestock population at any given time t, designated by N_l , is divided into four classes: susceptible livestock S_l , exposed livestock E_l , infected livestock I_l, and recovered livestock R_l . Hence,

$$N_L = S_l + E_l + I_l + R_l \quad (1)$$

Susceptible Livestock: At a consistent rate Λ_l , susceptible livestock are recruited into the population (by births and livestock immigration). They decrease when susceptible livestock interact with infected animals or an infected environment at a rate $\beta(I_l + B)$ where β the force of infection is present. Natural death will occur at a rate $\mu$for the livestock. And those animals that have recovered will return to the susceptible at a rate δ_l . As a result,

$\frac{dS_l}{dt} = \Lambda_l + \delta_l R_l - (\beta(L_l + B) + \mu)S_l$ (2)

The exposed livestock: The population grows when susceptible livestock interact with diseased animals or the affected environment at a rate $\beta(I_l + B)$. The population of exposed livestock is reduced as exposed livestock develops to become infected livestock (at a rateβ_1) and via natural death (at a rateμ). Hence, yields

$\frac{dE_l}{dt} = \beta(I_l + B)S_l - (\beta_1 + \mu)E_l$ (3)

Infected livestock: are engendered as a result of exposed livestock getting infected at a rate β_1 . They perish due to both infection-related and natural mortality (at a rate $\mu_0 + \mu$) as well as recovery after therapy at a rate $(\tau + \gamma)$.The rate of change in the population of unhealthy livestock is provided by

$\frac{dI_l}{dt} = \beta_1 E_l - (\tau + \mu + \mu_0 + \gamma)I_l$ (4)

Recovered livestock: The recovered livestock population is made up of infected livestock that recovered after treatment (at a rateγ) and diminishes at a rate due to natural mortalityμ . As a result, the rate of change in population regained is given by:

$\frac{dR_l}{dt} = \gamma I_l - (\delta_l + \mu)R_l$ (5)

The Brucella in the environment at a time$t$, represented by N_b is specified by

$N_b = B$ (6)

The Brucella in the population is generated from both the exposed and the infected live stocks in the population at the$m(E_l + I_l)$, where $m$is the amount of brucella deposited in the environment. The brucella dies out naturally at a rate μ_2Thus

$\frac{dB}{dt} = m(E_l + I_l) - \mu_2 B$ (7)

At any time$a < 0 and b > 0$, the entire human population denoted by $N_t$is split into four sub groups consisting of the following classes: susceptible humanS_h, exposed humanE_h, infected human I_h and the recovered humanR_h. Hence,

$N_h = S_h + E_h + I_h + R_h$ (8)

Susceptible humans: The susceptible humans are increased via recruitment (by birth and immigration), into the population at a constant rate Λ_h. They diminished once the susceptible human interacts with either infected livestock or infected environment at a rate $\beta(I_l + B)$, where β is the force of infection. The livestock will experience natural death at a rateμ. And those humans that recovered will return to the susceptible at a rate δ_h.Thus gives

$\frac{dS_h}{dt} = \Lambda_h + \delta_h R_{lh} - (\beta(L_h + B) + \mu)S_h$ (9)

The exposed humans: The population of the exposed increases once the susceptible human interacts with either infected livestock or infected environment at a rate$\beta(I_l + B)$. The population of exposed is decreased; when exposed human progresses to become infected human at a rate$\beta_2$and through natural death (at a rateμ_1). Thus, yields

$\frac{dE_h}{dt} = \beta(I_l + B)S_h - (\beta_2 + \mu)E_l$ (10)

Infected humans: infected humans are generated due to exposed human becoming infected, this occurs at a rateβ_2. They decrease due to both death due to infection and natural death (at a rate$\mu_{10} + \mu_1$) and also decline to recovery after treatment at a rate$(\tau + \gamma)$.The rate of change of the population of infected human is given by

$\frac{dI_l}{dt} = \beta_1 E_l - (\tau + \mu + \mu_{10} + \gamma_1)I_l$ (11)

Recovered humans: The population of recovered human are generated by infected human that recovered after treatment (at a rateγ_1), and decreases due to natural death at rateμ. Thus, the rate of change of the population of recovered is given by

$\frac{dR_h}{dt} = \boldsymbol{\gamma_1 I_h - (\delta_h + \mu_1)R_h}$ (12)

The above description culminates into the following system of differential equations:

$$\frac{dS_l}{dt} = \Lambda_l + \delta_l R_l - (\beta(I_l + B) + \mu)S_l$$
$$\frac{dE_l}{dt} = \beta(I_l + B)S_l - (\beta_1 + \mu)E_l$$
$$\frac{dI_l}{dt} = \beta_1 E_l - (\tau + \mu + \mu_0 + \gamma)I_l$$
$$\frac{dR_l}{dt} = \gamma I_l - (\delta_l + \mu)R_l$$
$$\frac{dB}{dt} = m(E_l + I_l) - \mu_2 B \quad (13)$$
$$\frac{dS_h}{dt} = \Lambda_h + \delta_h R_{lh} - (\beta(I_h + B) + \mu)S_h$$
$$\frac{dE_h}{dt} = \beta(I_l + B)S_h - (\beta_2 + \mu)E_l$$
$$\frac{dI_l}{dt} = \beta_1 E_l - (\tau + \mu + \mu_{10} + \gamma_1)I_l$$
$$\boldsymbol{\frac{dR_h}{dt} = \gamma_1 I_h - (\delta_h + \mu_1)R_h}$$

Tables 1 and 2 summarize the corresponding model variables and parameters.

Basic properties of the Brucellosis model

Positivity of Solutions

We suppose that all of the model's parameters (13) are positive and that the initial conditions for (13) are

$S_{l0}(0) > 0, E_{l0}(0) > 0, I_{l0}(0) > 0, R_{l0} > 0, B_0(0) > 0, S_{h0}(0) > 0, E_{h0}(0) > 0, I_{h0}(0) > 0, R_{00}(0) > 0$ (14)

Lemma 2.1 solutions of (13) and (14) satisfy

$S_{l0}(0) > 0, E_{l0}(0) > 0, I_{l0}(0) > 0, R_{l0} > 0, B_0(0) > 0, S_{h0}(0) > 0, E_{h0}(0) > 0, I_{h0}(0) > 0, R_{00}(0) > 0 for all t > 0$

Proof

From the first equation of the model (13) we have

$\frac{dS_l}{dt} = \Lambda_l + \delta_l R_l - (\beta(L_l + B) + \mu)S_l$

Note that on $t \in [0, \infty)$ we have

$S'_{l0} \geq -(\beta(L_l + B) + \mu)S_l$

So that $S_l(t) \geq \tilde{S}_l(t)$ where $\tilde{S}_l$ is the solution of $S'_{l0} \geq -(\beta(L_l + B) + \mu)S_l$

Satisfying $\tilde{S}_l(0) = S_l(0) > 0$. clearly $\tilde{S}_l(t) > 0$ for all $t > 0$.

Following the same argument, it can be shown that , $E_l(t) > 0, I_l(0) > 0, R_l(0) > 0, B(0) > 0, E_h(t) > 0, I_h(0) > 0, R_h(0) > 0$ for all$t > 0$. The proof complete

Invariant regions

The brucellosis model (13) will be analyzed in a biologically feasible region as follows.

The total population sizes $N_l(t), N_b(B) and N_h$ can be determined by

$N_l(t) = S_l(t) + E_l(t) + I_l(t) + R_l(t)$

$N_b(t) = B(t) and N_h = S_h(t) + E_h(t) + I_h(t) + R_h(t)$ or from the differential equations model

$\frac{dN_l(t)}{dt} = \frac{dS_l(t)}{dt} + \frac{dE_l(t)}{dt} + \frac{dI_l(t)}{dt} + \frac{dR_l(t)}{dt}$

$= \Lambda_l - \mu N_l - \mu_0 I_l,$ (15)

$\frac{dN_b(t)}{dt} = \frac{dB(t)}{dt}$

$= m(E_l + I_l) - \mu_2 B$ (16)

and

$\frac{dN_h}{dt} = \frac{dS_h(t)}{dt} + \frac{dE_h(t)}{dt} + \frac{dI_h(t)}{dt} + \frac{dR_h(t)}{dt}$

$= \Lambda_h - \mu_1 N_h - \mu_{10} I_h$ (17)

Assuming the disease does not kill, then$(\mu_0 = \mu_{10} = 0)$, thus

$\frac{dN_l(t)}{dt} = \Lambda_l - \mu N_l - \mu_0 I_l \leq \Lambda_l - \mu N_l$ (18)

Lemma 2.2. The model equation (13)has solutions which are contained in the feasible

$\Omega = \Omega_l \times \Omega_b \times \Omega_h$

Proof: Let $(S_l, E_l, I_l, R_l, B, S_h, E_h, I_h, R_h) \in R_+^9$ be any solution of the system with nonnegative initial conditions. Since

$\frac{dN_l(t)}{dt} \leq \Lambda_l - \mu N_l$ (19)

Hence, by the standard comparison theorem it can be shown that

$0 \leq N_l \leq \frac{\Lambda_l}{\mu}$, so that

$\Lambda_l - \mu N_l \geq K e^{-\mu t}$ where $K$is a constant (20)

Thus, all possible solutions of the Livestock population of the model equation (13)are in the region

$\Omega_l = \left\{(S_l, E_l, I_l, R_l) \in R_+^4 : N_l \leq \frac{\Lambda_l}{\mu}\right\}$ (21)

Similarly, all possible solutions of the human population of the model equation (13)are in the region

$\Omega_h = \left\{(S_h, E_h, I_h, R_{lh}) \in R_+^4 : N_h \leq \frac{\Lambda_h}{\mu_1}\right\}$ (22)

As well as all possible solutions of the Brucella population of the model equation (13)are in the region

$\Omega_b = \{(B) \in R_+^1 : N_b \leq \mu_2 B\}$ (23)

As a result, the feasible set for the model equation is given by

$\Omega = (S_l, E_l, I_l, R_l, B, S_h, E_h, I_h, R_h) \in R_+^9 : S_l, E_l, I_l, R_l, B, S_h, E_h, I_h, R_h \geq 0;$

$N_l \leq \frac{\Lambda_l}{\mu}, N_b \leq \mu_2 B, N_h \leq \frac{\Lambda_h}{\mu_1}$ (24)

Which is a positively invariant set under the model's flow. As a result, the model equation (13) is both epidemiologically significant and mathematically well posed in the domainΩ. In this domain, it is sufficient to consider the dynamics of the model's generated flow. Furthermore, the system follows the typical existence, uniqueness, and continuation of results.

Lemma 2.3 The region $\Omega = \Omega_l \times \Omega_b \times \Omega_h$ is positively – invariant for the basic model (2.1) with non- negative initial conditions in $\Re_+^9$

Analysis of Disease-Free Equilibrium (DFE) and Stability

The model (13) has a DFE, which is derived by setting the right-hand side of the equations in (13) to zero, which is illustrated by

$E_0(S_l^*, E_l^*, I_l^*, R_l^*, B^*, S_h^*, E_h^*, I_h^* R_h^*) = \left(\frac{\Lambda_l}{\mu}, 0,0,0,0, \frac{\Lambda_l}{\mu_1}, 0,0,0\right)$ (25)

As utilized by (Mohammed, Yahya, and Farah (2015), the next-generation operator method can be used to determine the linear stability of brucellosis. The matrix F and V for the new infection terms and the remaining transfer terms, based on the model equation (13), are provided by

$$F := \begin{vmatrix} 0 & \frac{\beta\Lambda_l}{\mu} & \frac{\beta\Lambda_l}{\mu} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ m & m & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta\Lambda_h}{\mu_1} & 0 & \frac{\beta\Lambda_h}{\mu_1} \\ 0 & 0 & 0 & 0 & 0 \end{vmatrix}$$

$$V := \begin{vmatrix} \beta_1 + \mu & 0 & 0 & 0 & 0 \\ -\beta_1 & \tau + \mu + \mu_0 + \gamma & 0 & 0 & 0 \\ 0 & 0 & \mu_0 & 0 & 0 \\ 0 & 0 & 0 & \beta_2 + \mu_1 & 0 \\ 0 & 0 & 0 & -\beta_2 & \tau + \mu_1 + \mu_{10} + \gamma \end{vmatrix}$$

The threshold epidemiological of brucellosis, denoted by $\mathcal{R}_0 = \rho(FV^{-1})$, where ρdenotes the spectral radius, is given by

$\Re_0 = \frac{\beta \Lambda_h \beta_2}{\mu_1(\beta_2 + \mu_1)(\tau + \mu_1 + \mu_{10} + \gamma_1)}$ (26)

Theorem 1 The DFE of model equation (13), given (26), is locally asymptotically stable (LAS) if$\mathcal{R}_0 < 1, and$ unstable if $\mathscr{R}_0 > 1$.

The threshold quantity $\mathscr{R}_0$ is the threshold epidemiological of brucellosis. It is the expected average number of new infections produced by interaction with exposed and infected livestock and humans when introduced into a completely susceptible population.

Model for Optimal Control of brucellosis

In this section, the basic model (13) to include the possible interventions in order to reduce or limit the proliferation of exposed livestock or human and the sudden increase of the infected livestock and humans in a population was extended. The associated control model to derive optimal control with minimal cost was formulated. The control efforts $u_1, u_2 and u_3$ represent public health education for the susceptible humans, treatment for both infected livestock and human, and vaccination for recruitment rate of the livestock in the population respectively. The model equation (13) becomes

$$\begin{aligned}
\frac{dS_l}{dt} &= \Lambda_l(1-u_3) + \delta_l R_l - (\beta(L_l + B)(1-u_2) + \mu)S_l \\
\frac{dE_l}{dt} &= \beta(I_l + B)(1-u_2)S_l - (\beta_1 + \mu)E_l \\
\frac{dI_l}{dt} &= \beta_1 E_l - (\tau + \mu + \mu_0 + u_2 + \gamma)I_l \\
\frac{dR_l}{dt} &= \gamma I_l - (\delta_l + \mu)R_l \\
\frac{dB}{dt} &= m(E_l + I_l) - \mu_2 B \\
\frac{dS_h}{dt} &= \Lambda_h(1-u_1) + \delta_h R_{lh} - (\beta(L_h + B)(1-u_2) + \mu)S_h \\
\frac{dE_h}{dt} &= \beta(I_l + B)(1-u_2)S_h - (\beta_2 + \mu)E_l \\
\frac{dI_l}{dt} &= \beta_1 E_l - (\tau + \mu + \mu_{10} + u_2 + \gamma_1)I_l \\
\frac{dR_h}{dt} &= \boldsymbol{\gamma_1 I_h - (\delta_h + \mu_1) R_h}
\end{aligned}$$ (27)

The factor of $1 - u_1(t)$ is a control function representing public health education aimed at reducing infectivity in humans. The factor of $1 - u_2(t)$ is a control function representing treatment aimed at reducing infectivity in both human and livestock. The factor of $1 - u_3(t)$ is a control function representing vaccination aimed at reducing infectivity in livestock and the environment. Investigating the optimal control efforts that would be needed to control brucellosis in the society, an optimal control problem with the objective (cost) functional is given by

$J(u) = \int_0^T (A_1 E_l + A_2 I_l + A_3 E_h + A_4 I_h + B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2) dt$ (28)

Where T the final time and the coefficients $A_1, A_2, A_3, B_1, B_2, B_3$ are positive weights to balance the factors. The aim is to minimize the number of both exposed and infected livestock and humans, while minimizing the cost of controls $u_1(t), u_2(t), u_3(t)$. Thus, we seek an optimal control u_1^*, u_2^*, u_3^* such that

$J(u_1^*, u_2^*, u_2^*) = \min_{u_1, u_2, u_3} \{J(u_1, u_2, u_3) | u_1, u_2, u_3 \in u\}$

Where the control set is defined as

$U = \{(u_1^*, u_2^*, u_3^*) | u_i(t) is Lebesgues measurable, i = 1,2,3,0 \leq u_i(t) \leq 1, t \in [0,T]\}$. (29)

Subject to the system (8) and appropriate initial conditions. The basic framework of this problem is to characterize the optimal control.

Existence of Control Problem

The term $A_1E_l, A_3I_l, A_3E_h, A_4I_h$ are the cost of infection while $B_1u_1^2, B_2u_2^2 and B_3u_1^2$ are the costs of public health education, treatment and vaccination respectively. The necessary conditions that an optimal control must satisfy come from the Pontryagin's Maximum Principle [20]. The principle converts (27-28) into a problem of minimizing point wise a Hamiltonian$H$, with respect to$u$.

$$H = A_1E_l + A_2I_l + A_3E_h + A_4I_h + B_1u_1^2 + B_2u_2^2 + B_3u_3^2 + \lambda_1(\Lambda_l(1-u_3) + \delta_l R_l - (\beta(L_l+B)(1-u_2)+\mu)S) + \lambda_2(\beta(I_l+B)(1-u_2)S_l - (\beta_1+\mu)E_l) + \lambda_3(\beta_1E_l - (\tau+\mu+\mu_0+u_2+\gamma)I_l) + \lambda_4(\gamma I_l - (\delta_l+\mu)R_l) + \lambda_5(m(E_l+I_l) - \mu_2B) + \lambda_6(\Lambda_h(1-u_1) + \delta_h R_{lh} - (\beta(L_h+B)(1-u_2)+\mu)S_h) + \lambda_7(\beta(I_l+B)(1-u_2)S_h - (\beta_2+\mu)E_l) + \lambda_8(\beta_1E_l - (\tau+\mu+\mu_{10}+u_2+\gamma_1)I_l) + \lambda_9(\gamma_1I_h - (\delta_h+\mu_1)R_h)$ (30)

Where $\lambda_i = 0, for i = 1,2,\ldots,9$ are adjoint variables or co-state variables.

Theorem 2 Given an optimal control u^* and solutions $(S_l^*, E_l^*, I_l^*, R_l^*, B, S_h^*, E_h^*, I_h^*, R_h^*)$ of the corresponding state system (27) that minimizes $J(u_1,u_2,u_3)$ over$U$. Then there exists adjoint variables $\lambda_i = 0, for i = 1,2,\ldots,9$satisfying

$$-\lambda_1 = \big((\beta I_l + \beta B)u_3 - \beta I_l - \beta B - \mu\big)S_l - \Lambda_l + \Lambda_l u_3 - \delta R_l$$
$$-\lambda_2 = (-1+u_3)(\beta I_l + \beta B)S_l + (\beta_1+\mu)E_l$$
$$-\lambda_3 = -\beta_1 E_l + (u_2+\tau+\mu+\mu_0+\gamma)I_l$$
$$-\lambda_4 = -\gamma I_l + (\delta+\mu)R_l$$
$$-\lambda_5 = -m(E_l+I_l) + \mu_2 B$$
$$-\lambda_6 = \Lambda_h(-1+u_1) - \delta_h R_h + (\beta I_l(1-u_2) + \beta B(1-u_2) + \mu_1)S_h$$
$$-\lambda_7 = (-1+u_2)(\beta I_l + \beta B)S_h + (\beta_1+\mu)E_h$$
$$-\lambda_8 = -\beta_2 E_l + (u_2+\tau+\mu+\mu_{10}+\gamma_1)I_h$$
$$-\lambda_9 = -\gamma_1 I_h + (\delta_h+\mu_1)R_h \quad (31)$$

$and with transversality conditions$

$$\lambda_i = 0, for i = 1,2,\ldots,9 \quad (32)$$

$and the controls u^* satisfy the optimality condition$

$$\begin{cases} u_1^* = max\left\{0, min\left(1, \frac{\lambda_6\Lambda_h}{2B_1}\right)\right\} \\ u_2^* = max\left\{0, min\left(1, \frac{-(\lambda_6-\lambda_7)(\beta I_l+\beta B)S_h + \lambda_8 I_h + \lambda_3 I_l}{2B_2}\right)\right\} \\ u_3^* = max\left\{0, min\left(1, \frac{-(\lambda_1-\lambda_2)(\beta I_l+\beta B)S_l + \lambda_1\Lambda_l}{2B_3}\right)\right\} \end{cases} \quad (33)$$

Proof The governing equations of the adjoints variables are obtained by differentiation of the Hamiltonian function, evaluated at the optimal control. And with transversality conditions

$$\lambda_i = 0, for i = 1,2,\ldots,9$$

On the interior of the control set, where $0 < u < 1$, we have Hence, it is obtained [20]

$$\begin{cases} u_1^* = max\left\{0, min\left(1, \frac{\lambda_6\Lambda_h}{2B_1}\right)\right\} \\ u_2^* = max\left\{0, min\left(1, \frac{-(\lambda_6-\lambda_7)(\beta I_l+\beta B)S_h + \lambda_8 I_h + \lambda_3 I_l}{2B_2}\right)\right\} \\ u_3^* = max\left\{0, min\left(1, \frac{-(\lambda_1-\lambda_2)(\beta I_l+\beta B)S_l + \lambda_1\Lambda_l}{2B_3}\right)\right\} \end{cases} \quad (33)$$

Numerical Simulations

In this section, the numerical simulations are presented to illustrate the analytical results. Next, we examine numerically the effect of the optimal control strategies on the spread of brucellosis *in* a population.

Strategy A: Optimal use of public health education.
Strategy B: Optimal use of treatment.
Strategy C: Optimal use of vaccination.

Table 1: Description of variables for brucellosis model

Parameter	Description
S_l	*Susceptible livestock*
E_l	Exposed livestock
I_l	Infected livestock
R_l	*Removed livestock*
B	*Brucella*
S_l	*Susceptible human*
E_l	Exposed human
I_l	Infected human
R_l	Recovered *human*

Table 2: Description of parameters for brucellosis model

Par.	Description	Value	Source
Λ_l	*Recruitment rate of livestock*	0.811	14
δ_l	*Loss of immunity after recovery livestock*	0	14
β	*Force of infection*	0.00025	15
μ	Natural death of livestock	0.6	15
β_1	Rate of progression from exposed to infected livestock	1	16
τ	Rate of treatment	Setting	Assumed
μ_0	Death due to infection of livestock	Setting	Assumed
γ	Recovery of livestock	*0.4*	*16*
m	Quantity of brucella generated	*15*	*16*
μ_2	Decay of brucella from the environment	*3.6*	*16*
Λ_h	*Recruitment rate of human*	*0.018*	*14*
δ_h	*Loss of immunity after recovery human*	0.8	14

μ_1	Natural death of human	0.003	14
β_2	Rate of progression from exposed to infected human	0.6	16
γ_1	Recovery of human	0.4	16
μ_{10}	Death due to infection of human	0.09	Assumed

Optimal application of public health education (u_1)

The utilization of public health education (u_1) is employed to optimize the objective functional J in this control method, while the control treatment (u_2) and vaccine (u_3) are set to zero. Figure 1 indicates a considerable difference in the S_h and R_h with optimal control strategy against S_h and R_h without control. Figure 1(a) shows that the susceptible humans reduce as a result of control techniques versus the increase in the uncontrolled situation. Figure 1(b) depicts a similar situation in the instance of a recovered individual.

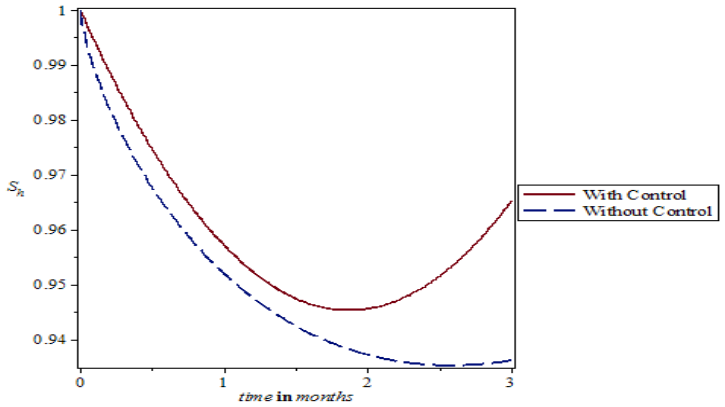

Figure 1(a)

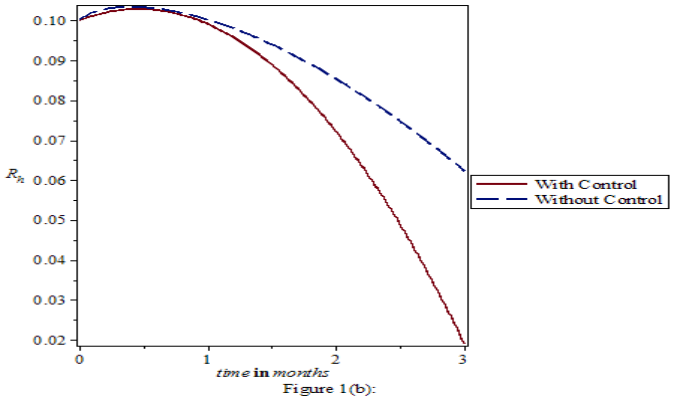

Figure 1(b):

Figure 1: Simulations of the brucellosis model demonstrate the influence of optimal public health education on brucellosis spread in humans.

Optimal application of treatment (u_2)

The optimal use of treatment (u_2) is employed to optimize the objective functional J in this control method, whereas the controls of public health education (u_1) and vaccination (u_3) are set to zero. Figure 2 indicates an important distinction in the $E_l, E_h, I_l and I_h$ optimal control technique over without control. Figure 2 shows how management techniques reduce the number of exposed and infected animals and humans while increasing in unmanaged conditions.

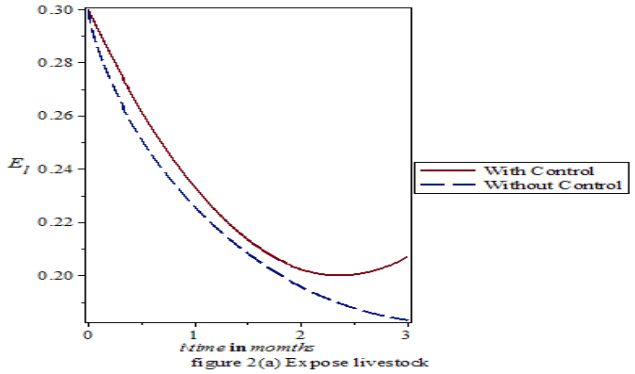

figure 2(a) Expose livestock

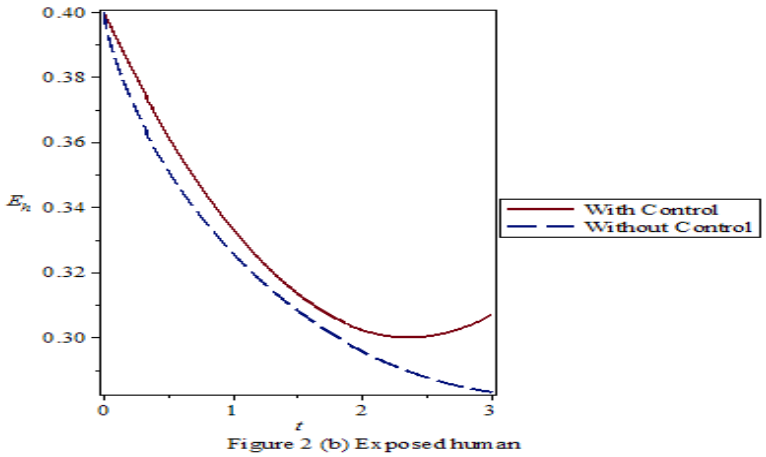

Figure 2 (b) Exposed human

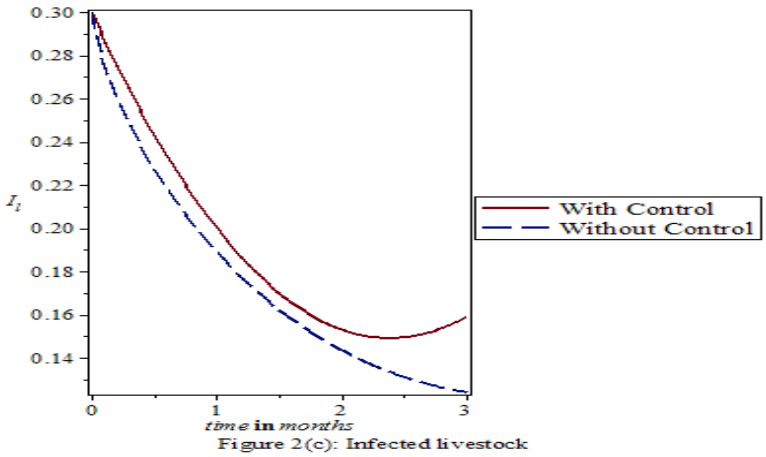

Figure 2(c): Infected livestock

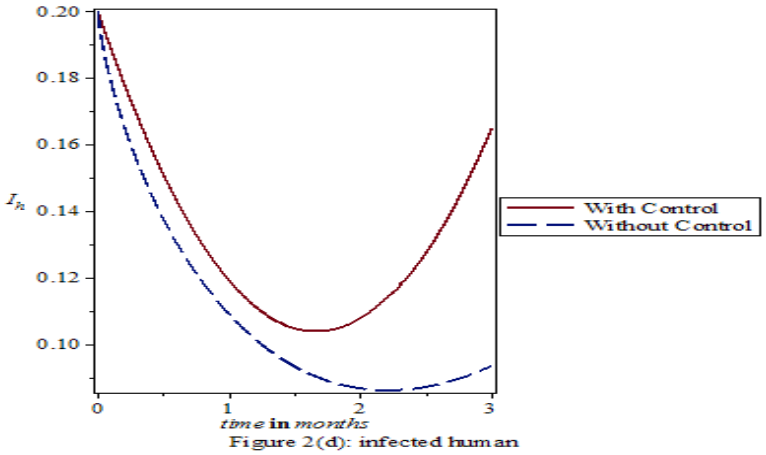

Figure 2(d): infected human

Figure 2: Simulations of the brucellosis model demonstrate the impact of optimal treatment utilization on the spread of brucellosis in humans and livestock.

Optimal application vaccination (u_3)

Vaccination (u_3) is employed to optimize the objective functional J in this control approach, whereas public health education $(\boldsymbol{u_1})$and treatment $(\boldsymbol{u_2})$are set to zero. Figure 3 indicates a considerable difference in the S_l $\rightleftarrows$ and $\rightleftarrows$ $R_l$optimal control method over those without control. Figure 3 shows that as a result of control techniques against the growth in uncontrolled cases of livestock, the susceptible and recovered cattle decrease.

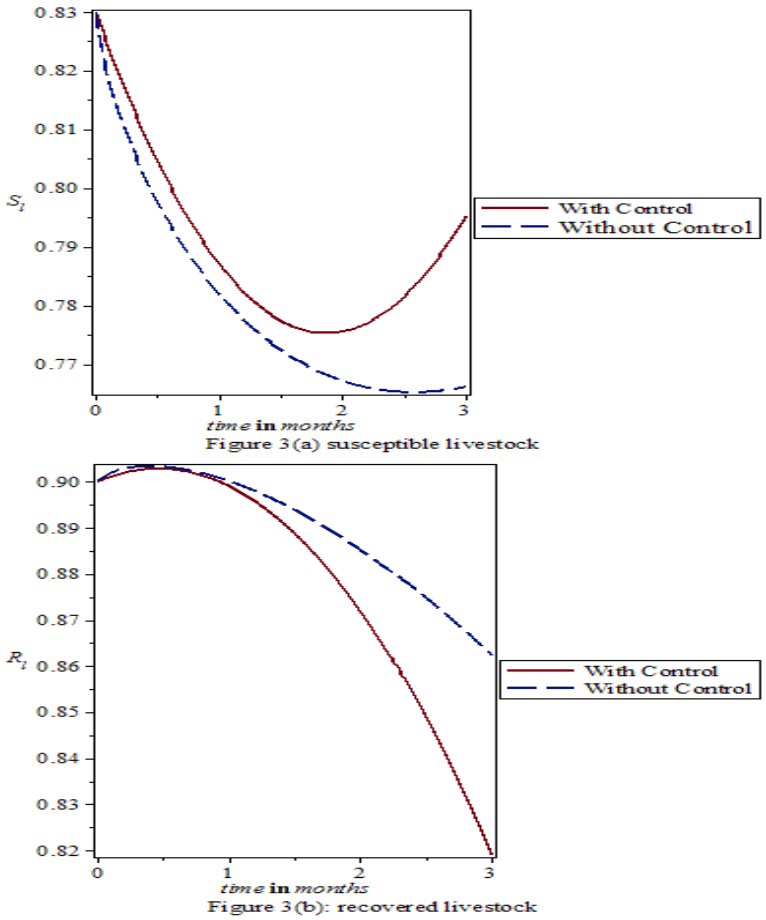

Figure 3: Simulations of the brucellosis model demonstrate the impact of appropriate vaccine use on the spread of brucellosis in livestock.

CONCLUSION

A deterministic mathematical model for brucellosis control was develops in this study. The model investigated how humans, livestock, and brucella deposited in the environment interacted. Furthermore, it is demonstrated that the model is locally asymptotically stable when $\Re_0 < 1$ and unstable when$\Re_0 > 1$. The numerical simulation results revealed that the optimum disease control strategies were a combination of the three control strategies. Using all of the controls, on the other hand, will incur additional costs. This is because strategy B has a major impact on brucellosis control. It can thus be argued that the employment of treatment (strategy B) is the most cost-effective option for controlling brucellosis.

REFERENCES

Abatih, E., Ron, L., Speybroeck, N., Williams, B., & Berkvens, D. (2015). Mathematical analysis of the transmission dynamics of brucellosis among bison. *Mathematical Methods in the Applied Sciences*, *38*(17), 3818-3832.

Ainseba, B. E., Benosman, C., & Magal, P. (2010). A model for ovine brucellosis incorporating direct and indirect transmission. *Journal of biological dynamics*, *4*(1), 2-11.

Akhvlediani, T., Bautista, C. T., Garuchava, N., Sanodze, L., Kokaia, N., Malania, L., ... & Trapaidze, N. (2017). Epidemiological and clinical features of brucellosis in the country of Georgia. *PLoS One*, *12*(1), e0170376.

Al-Tawfiq, J. A., & AbuKhamsin, A. (2009). A 24-year study of the epidemiology of human brucellosis in a health-care system in Eastern Saudi Arabia. *Journal of infection and public health*, *2*(2), 81-85.

Brouwer, A. F., Weir, M. H., Eisenberg, M. C., Meza, R., & Eisenberg, J. N. (2017). Dose-response relationships for environmentally mediated infectious disease transmission models. *PLoS computational biology*, *13*(4), e1005481.

Corbel, M. J. (1997). Brucellosis: an overview. *Emerging infectious diseases*, *3*(2), 213.

Corbel, M. J. Brucellosis in humans and animals: World Health Organization. 2006.

De Souza, V. A. F., Neto, J. S. F., Amaku, M., Dias, R. A., Telles, E. O., Grisi-Filho, J. H. H., ... & Ferreira, F. (2016). Mathematical modeling of bovine brucellosis control using the RB51 vaccine. *Semina: Ciências Agrárias*, *37*(5), 3767-3775.

Godfroid, J. (2002). Brucellosis in wildlife. *Revue Scientifique et Technique-Office international des épizooties*, *21*(1), 277-286.

Godfroid, J., Garin-Bastuji, B., Saegerman, C., & Blasco, J. M. (2013). Brucellosis in terrestrial wildlife. *Revue Scientifique et Technique. Office International des Epizooties*.

Godfroid, J., Scholz, H. C., Barbier, T., Nicolas, C., Wattiau, P., Fretin, D., ... & Letesson, J. J. (2011). Brucellosis at the animal/ecosystem/human interface at the beginning of the 21st century. *Preventive veterinary medicine*, *102*(2), 118-131.

Hou, Q., Sun, X., Zhang, J., Liu, Y., Wang, Y., & Jin, Z. (2013). Modeling the transmission dynamics of sheep brucellosis in Inner Mongolia Autonomous Region, China. *Mathematical biosciences*, *242*(1), 51-58.

Mohammed, B. A., Yahya A., & Farah A. A. (2015). A Mathematical Analysis of the Effect of Control of Plasmodium *Knowlesi* Malaria. Pak. J. Statist. Vol. 31(5), 483-514

Moreno, E. (2014). Retrospective and prospective perspectives on zoonotic brucellosis. *Frontiers in microbiology*, *5*, 213.

Morris Jr, J. G. (Ed.). (2013). *Foodborne infections and intoxications*. Academic Press.

Shevtsova, E., Shevtsov, A., Mukanov, K., Filipenko, M., Kamalova, D., Sytnik, I., ... & Ramanculov, E. (2016). Epidemiology of brucellosis and genetic diversity of Brucella abortus in Kazakhstan. *PLoS One*, *11*(12), e0167496.

Zhang, J., Sun, G. Q., Sun, X. D., Hou, Q., Li, M., Huang, B., ... & Jin, Z. (2014). Prediction and control of brucellosis transmission of dairy cattle in Zhejiang Province, China. *Plos one*, *9*(11), e108592.

Zhou, L., Fan, M., Hou, Q., Jin, Z., & Sun, X. (2018). Transmission dynamics and optimal control of brucellosis in Inner Mongolia of China. *Mathematical Biosciences & Engineering*, *15*(2), 543.

Zinsstag, J., Roth, F., Orkhon, D., Chimed-Ochir, G., Nansalmaa, M., Kolar, J., & Vounatsou, P. (2005). A model of animal–human brucellosis transmission in Mongolia. *Preventive veterinary medicine*, *69*(1-2), 77-95.

©2023 This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International license viewed via https://creativecommons.org/licenses/by/4.0/ which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is cited appropriately.