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Modeling and analysis of taeniasis and cysticercosis transmission dynamics in humans, pigs and cattle

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Abstract

Taeniasis and cysticercosis pose a significant challenge to food safety and public health. Cysticercosis reduces the market value for pigs and cattle by making pork and beef unsafe for consumption. In this paper, a mathematical model for the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle is formulated and analyzed. The analysis shows that both the disease free equilibrium (DFE) and the endemic equilibrium (EE) exist. To study the dynamics of the diseases, we derived the basic reproduction number R_0 by next generation matrix method. When $R_0 < 1$, the DFE is globally asymptotically stable whereas when $R_0 > 1$ the EE is globally asymptotically stable. The normalized forward sensitivity index was used to determine sensitive parameters to the diseases. Humans' recruitment rate, probability of humans' infection with taeniasis and the defecation rate of taenia eggs by humans with taeniasis are the most positive sensitive parameters to diseases' transmission whereas the human natural death rate is the most negative sensitive parameter. However, it is biologically unethical and not practical to increase human natural mortality rate for disease control. In this case, other parameters with negative sensitivity indices such as death rate of taenia eggs and proportions of unconsumed infected beef and pork can be considered for disease control. Generally, to control the diseases, more efforts should be made directed to reducing the number of humans who have taeniasis and defecate in the open environment. Also meat inspection and indoor keeping of cattle and pigs should be emphasized.

Keywords: Taeniasis; Cysticercosis; Reproduction number; Equilibria; Stability analysis; Sensitivity analysis

1 Introduction

Taeniasis and cysticercosis are foodborne infections which are caused by the adult and larval form tapeworms, respectively [29]. Three tapeworm species that cause taeniasis in humans and cysticercosis in humans, pigs and cattle are *Taenia solium, Taenia saginata* and *Taenia asiatica* [36]. While *T. asiatica* is endemic only in Asia, other tapeworm species are distributed worldwide [35]. Taeniasis and cysticercosis are associated with poor hygiene and sanitation, open human defecation, free range farming system for pigs and cattle, and poverty [11, 35]. They affect mainly poor and marginalized communities who always have

close contact with pigs and cattle, and depend on pork and beef as their sources of food [30, 36].

Humans acquire taeniasis when they consume raw or undercooked beef or pork that contains the tapeworm larval cysts, which develop into adult tapeworms in the human intestine [36]. When humans who are infected with taeniasis defecate in the fields, the tapeworm eggs that are passed out with human faeces spread over the environment by several means, such as water, winds, animal feet and insects, and contaminate the soil, fodder, pastures and water sources [8]. Domestic cattle acquire cysticercosis when they feed on the contaminated environment while pigs acquire cysticercosis through direct consumption of human faeces or indirectly when they ingest taenia eggs from the contaminated environment [29]. When taenia eggs are consumed by cattle and pigs, they hatch, penetrate the intestinal wall and reach the blood circulation system, where they spread throughout the body tissues and organs such as heart, diaphragm, kidney, lungs, liver and tongue, and develop into cysticerci [13, 35]. Human cysticercosis is a result of consuming *T. solium* eggs from the contaminated environment via contaminated water, fruits and vegetables, or by putting contaminated fingers in the mouth [7]. Once the eggs are ingested, they hatch in the small intestine, and develop into larvae which penetrate the intestinal wall and migrate to various parts of the body such as eyes, muscles, skin and the central nervous system through the blood circulatory system, where they form larval cysts [35]. When the cysts reach and infect the brain, they cause neurocysticercosis which is the most severe form of tapeworm infection of the central nervous system and is the major cause of epilepsy worldwide [36].

Taeniasis and cysticercosis are endemic in many developing countries of Latin America, Africa and Asia [29]. The diseases threaten people's health and livelihood of subsistence farming communities, posing considerable challenges in food safety and reducing the market value of pigs and cattle by making pork and beef unsafe to eat [34, 35]. Globally, the diseases affect approximately 50 million people and nearly 50,000 people die annually due to cysticercosis [1]. In 2010, the World Health Organization (WHO) listed cysticercosis as a major neglected tropical disease and later in 2015, it was identified as a leading cause of deaths from foodborne diseases. In Tanzania, the disease has been reported to be a serious health problem whereby the parasite is spread in almost all regions [33]. Porcine cysticercosis has been reported to be highly endemic in southern, central and northern regions of Tanzania with the prevalence rate of 0.3–17.4%, 14.9% and 5.5–16.9%, respectively [21, 30]. Human infections have also been reported in the country [15, 23]. For example, in 2012 Tanzania had 17,853 cases and 212 deaths due to epilepsy while porcine cysticercosis cases were 18i and the economic burden for cysticercosis was estimated to be US$ 7.9 million [30].

The diagnosis of human taeniasis is done by examination of stool samples while human cysticercosis involves doing a biopsy of subcutaneous cysts, immunodiagnosis, radiography, computed tomography (CT) scan and magnetic resonance imaging (MRI). The diagnosis of neurocysticercosis requires both central nervous system imaging with CT brain scans or MRI and serological testing [35]. The diagnosis of cysticercosis in pigs and cattle involves meat inspection, serological tests and tongue inspection for pigs [34]. The treatment of human taeniasis and cysticercosis is through administration of prescribed medication of praziquantel, niclosamide, nitazoxanide or albendazole [35]. The treatment of neurocysticercosis may involve prolonged doses of albendazole and/or praziquantel with

supporting therapy such as anti-epileptic drugs, corticosteroids, and possibly surgery for some cases [35]. Intervention strategies in pigs and cattle involve using vaccines such as S3Pvac and TSOL18 for pigs and TSA-18 and TSA-9 for cattle [18, 19], and anthelmintic treatment with flubendazole, fenbendazole, oxfendazole, praziquantel and nitazoxanide [34, 35].

The use of mathematical models plays an important role in studying the transmission dynamics of infectious diseases and is very useful for deciding on the appropriate disease control strategies. Over the past two decades, various mathematical models have been formulated and analyzed to study the dynamics and control of parasitic foodborne diseases such as cholera, brucellosis and echinococcus [6, 20, 24, 28, 32, 37, 38]. In particular, some statistical and deterministic models with some stochastic elements that have been formulated and analyzed to study the transmission dynamics and control of taeniasis and cysticercosis in humans and pigs can be found in Gonzalez et al. [12], Kyvsgaard et al. [16], Braae et al. [2], Winskill et al. [34], José et al. [14] and Sánchez-Torreset al. [27]. The study done by Winskill et al. [34] has assumed that human taeniasis occurs when susceptible humans interact with pigs that are infected with cysticercosis something that is practically wrong. Moreover, previous studies did not include the cattle population to study the transmission dynamics of the taeniasis and cysticercosis. To get new insights on the diseases transmission dynamics, we formulate and analyze the mathematical model for the transmission dynamics of taeniasis and cysticercosis by including cattle population and incorporating compartments of infected pork and beef. The cattle population is included due to its significance in accelerating diseases through feeding on a contaminated environment which later affects human beings. Also, the market value of cattle is reduced and thus affecting the livelihood of subsistence farming communities and leading to food safety problems.

This paper is organised as follows: the deterministic model and analysis are presented in Sect. 2, and numerical analysis in Sect. 3. A summary and a conclusion are presented in Sect. 4.

2 Model formulation and analysis

2.1 Model formulation

The basic model for the dynamics of taeniasis and cysticercosis is formulated by modifying the work by Winskill et al. [34] that studied intervention strategies against *T. solium* cysticercosis, by including cattle population and incorporating compartments of infected pork and beef as described by Kyvsgaard et al. [16]. Taeniasis and cysticercosis use humans as the definitive hosts, and pigs and cattle as the intermediate hosts [8].

The model divides human population into S_H, I_{HT} and I_{HC} classes that represent susceptible humans, humans infected with taeniasis and humans infected with cysticercosis, respectively. The pig population is divided into S_P and I_P classes that represent susceptible pigs and pigs that are infected with cysticercosis, respectively, while the cattle population is divided into S_C and I_C, which represent susceptible cattle and cattle that are infected with cysticercosis, respectively. The compartments P_I and B_I represent infected pork and beef, respectively, and E_T represent the number of taenia eggs in the environment.

Susceptible humans are recruited through birth at per capita rate ψ and diminish through consumption of infected raw or insufficiently cooked pork or beef at rates α_p and α_b, respectively. They also diminish by acquiring cysticercosis through consumption

of *T. solium* eggs from the contaminated environment at a rate θ either in contaminated water, fruits, vegetables or by putting contaminated fingers in the mouth [7]. Humans with cysticercosis increase at a rate θ due to interaction of susceptible humans with *T. solium* eggs in the contaminated environment and they reduce due to disease induced death at rate μ_d. Humans with taeniasis increase at rates α_P and α_b when susceptible humans feed on infected pork and beef, respectively. The parameter β_T denotes the probability of susceptible humans acquiring taeniasis from an infected pork or beef, that is, the per capita rate at which susceptible individuals acquire infection. All human compartments suffer natural death rate μ_h. The number of taenia eggs in the environment grow as a result of open defecation by humans who are infected with taeniasis at a rate ν and decrease due to natural death at a rate μ_e.

Susceptible pigs are recruited at per capita rate Λ due to birth and reduce at a rate γ_p when they acquire cysticercosis from the contaminated environment. Similarly, susceptible cattle are recruited at per capita rate ϕ due to birth and diminish at a rate γ_c when they acquire cysticercosis from the contaminated environment. Both pigs and cattle are further slaughtered for consumption at rates ρ and σ, respectively. The infected pigs and cattle increase at rates γ_p and γ_c, respectively, as a consequence of susceptible pigs and cattle feeding on contaminated environment. All pigs and cattle classes suffer natural death at rates μ_p and μ_c, respectively. The infected pork increases when infected pigs are slaughtered at a rate ω and it decreases when consumed by humans at a rate α_p whereas infected beef increases when infected cattle are slaughtered at a rate η and decreases when consumed by humans at a rate α_b. The parameters δ and ϵ are the proportions of infected pork and beef unconsumed by susceptible humans.

In model formulation we consider the free range farming system for both pigs and cattle populations, and we do not consider immigration. We assume that humans can be infected by either taeniasis or cysticercosis; the number of taenia eggs consumed by humans, pigs and cattle has negligible effect on the total number of eggs in the environment and that infected humans, pigs and cattle cannot recover naturally without treatment. We further assume that pigs and cattle do not suffer disease induced mortality, that they become carriers for their life and that the rates at which susceptible humans consume infected raw or undercooked pork or beef depend on the amount of infected pork or beef that is present. Humans, pigs and cattle contact rates with taenia eggs in the environment are assumed to be density dependent. The model for the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle is summarized by using the flow diagram in Fig. 1.

The state variables and parameters are summarized in Tables 1 and 2, respectively.

Some parameter values in this paper are assumed within realistic ranges due to the fact that only little has been done on this area, the diseases being common in rural areas where there is inadequate or no meat inspection and the treatment is not readily available [22]. The model that describes the transmission dynamics of taeniasis and cysticercosis is governed by the following system of differential equations:

$$\frac{dS_H}{dt} = \psi - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H,$$

$$\frac{dI_{HT}}{dt} = \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \mu_h I_{HT},$$

$$\frac{dI_{HC}}{dt} = \theta S_H E_T - (\mu_h + \mu_d)I_{HC},$$

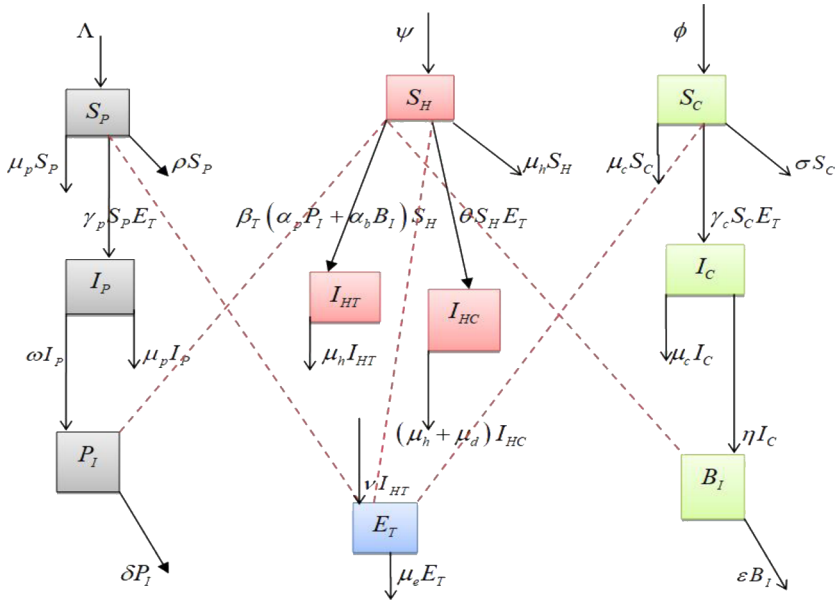

Figure 1 The model flow diagram

Table 1 Description of the state variables

Variable	Description	Variable	Description
S_H	Susceptible humans	P_I	Cysticercosis infected pork
I_{HT}	Humans infected with taeniasis	S_C	Susceptible cattle
I_{HC}	Humans infected with cysticercosis	I_C	Cattle infected with cysticercosis
S_P	Susceptible pigs	B_I	Cysticercosis infected beef
I_P	Pigs infected with cysticercosis	E_T	Taenia eggs in the environment

$$
\begin{aligned}
\frac{dS_P}{dt} &= \Lambda - \gamma_p S_P E_T - (\rho + \mu_p) S_P, \\
\frac{dI_P}{dt} &= \gamma_p S_P E_T - (\omega + \mu_p) I_P, \\
\frac{dP_I}{dt} &= \omega I_P - (\delta + \alpha_P) P_I, \\
\frac{dS_C}{dt} &= \phi - \gamma_c S_C E_T - (\sigma + \mu_c) S_C, \\
\frac{dI_C}{dt} &= \gamma_c S_C E_T - (\eta + \mu_c) I_C, \\
\frac{dB_I}{dt} &= \eta I_C - (\epsilon + \alpha_b) B_I, \\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T,
\end{aligned}
\tag{1}
$$

with initial conditions

$$
S_H(0) > 0; \quad I_{HT}(0) \geq 0; \quad I_{HC}(0) \geq 0; \quad S_P(0) > 0; \quad I_P(0) \geq 0;
$$

$$
P_I(0) \geq 0; \quad S_C(0) > 0; \quad I_C(0) \geq 0; \quad B_I(0) \geq 0 \quad \text{and} \quad E_T(0) \geq 0.
$$

Table 2 Parameters' description and their values (unit: yr^{-1})

Parameter	Description	Value	Source
ψ	Per capita recruitment rate of human population	2247	[37]
μ_h	Per capita natural death rate of humans	0.0141	[32]
μ_d	Human cysticercosis induced death rate	0.0925	[32]
Λ	Per capita recruitment rate of pig population	1450	Assumed
γ_p	*T. solium* eggs to pig transmission coefficient	0.01	[16]
α_p	Rate of eating raw/undercooked infected pork	0.012	Assumed
ρ	Harvesting rate of susceptible pigs	0.252	Assumed
ω	Slaughter rate of infected pigs	0.083×4	[16]
μ_p	Per capita natural death rate of pigs	0.083×12	[34]
δ	Proportion of unconsumed infected pork	0.358	Assumed
ϕ	Per capita recruitment rate of cattle population	750	Assumed
γ_c	*T. saginata* eggs to cattle transmission coefficient	0.00625	Assumed
α_b	Rate of eating raw/undercooked infected beef	0.023	Assumed
σ	Harvesting rate of susceptible cattle	0.213	Assumed
η	Slaughter rate of infected cattle	0.235	Assumed
μ_c	Per capita natural death rate of cattle	0.33	[32]
ϵ	Proportion of unconsumed infected beef	0.225	Assumed
β_T	Probability of humans getting taeniasis	0.093	Assumed
ν	Defecation rate by humans with taeniasis	0.150	Assumed
θ	*T. solium* eggs to human cysticercosis transmission coeffient	0.00523	Assumed
μ_e	Per capita death rate of taenia eggs	10.42	[32]

2.2 Positivity of solutions and invariant region

For the model system (1) to be biologically and epidemiologically meaningful, we need to show that the model solutions are positive and bounded.

2.2.1 Positivity of solutions

From the first equation in the model system (1) for susceptible humans, we have

$$\begin{aligned}
&\frac{dS_H}{dt} = \psi - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H,\\
&\frac{dS_H}{dt} \geq -\big(\beta_T(\alpha_p P_I + \alpha_b B_I) + \theta E_T + \mu_h\big)S_H,\\
&\frac{dS_H}{S_H} \geq -\big(\beta_T(\alpha_p P_I + \alpha_b B_I) + \theta E_T + \mu_h\big)dt,\\
&S_H(t) \geq S_H(0)e^{\int_0^t -(\beta_T(\alpha_p P_I+\alpha_b B_I)+\theta E_T+\mu_h)\,ds} \geq 0, \quad \forall t \geq 0.
\end{aligned}$$

Using the same approach, it can be shown that

$$\begin{aligned}
&I_{HT}(t) \geq 0; \quad I_{HC}(t) \geq 0; \quad S_P(t) \geq 0; \quad I_P(t) \geq 0; \quad P_I(t) \geq 0;\\
&S_C(t) \geq 0; \quad I_C(t) \geq 0; \quad B_I(t) \geq 0; \quad E_T(t) \geq 0, \quad \forall t \geq 0.
\end{aligned}$$

Therefore, all solutions of the model system (1) are positive for all $t \geq 0$.

2.2.2 Invariant region

To show that the model solutions are bounded, we let the total populations for humans, pigs and cattle be $H = S_H + I_{HT} + I_{HC}$, $T_P = S_P + I_P$ and $T_C = S_C + I_C$, respectively. By con-

sidering the human population, we have

$$\begin{aligned} &\frac{dH}{dt} = \psi - \mu_h H - \mu_d I_{HC}, \\ &\frac{dH}{dt} + \mu_h H \leq \psi. \end{aligned} \tag{2}$$

Integrating throughout, we obtain

$$H(t) \leq \frac{\psi}{\mu_h} + \left(H(0) - \frac{\psi}{\mu_h}\right)e^{-\mu_h t}, \tag{3}$$

where $H(0) = S_H(0) + I_{HT}(0) + I_{HC}(0)$. Following the same procedures, it can be shown that the populations for pigs and cattle are given by

$$T_P(t) \leq \frac{\Lambda}{\mu_p} + \left(T_P(0) - \frac{\Lambda}{\mu_p}\right)e^{-\mu_p t} \quad \text{and} \quad T_C(t) \leq \frac{\phi}{\mu_c} + \left(T_C(0) - \frac{\phi}{\mu_c}\right)e^{-\mu_c t}, \tag{4}$$

respectively, where $T_P(0) = S_P(0) + I_P(0)$ and $T_C(0) = S_C(0) + I_C(0)$. The analysis of the solutions (3) and (4) considers two cases: when $H(0) > \frac{\psi}{\mu_h}$, $T_P(0) > \frac{\Lambda}{\mu_p}$, $T_C(0) > \frac{\phi}{\mu_c}$ and when $H(0) < \frac{\psi}{\mu_h}$, $T_P(0) < \frac{\Lambda}{\mu_p}$, $T_C(0) < \frac{\phi}{\mu_c}$, which gives

$$\begin{aligned} &H(t) \leq \Phi_t = \max\left\{\frac{\psi}{\mu_h}, H(0)\right\}, \\ &T_P(t) \leq \Pi_t = \max\left\{\frac{\Lambda}{\mu_p}, T_P(0)\right\}, \\ &T_C(t) \leq \Psi_t = \max\left\{\frac{\phi}{\mu_c}, T_C(0)\right\}. \end{aligned} \tag{5}$$

Since $H(t) = S_H + I_{HT} + I_{HC} \leq \Phi_t$, it follows that $I_{HT} \leq \Phi_t$. We need to show that E_T is also bounded. By considering the last equation in model system (1), we have

$$\begin{aligned} &\frac{dE_T}{dt} = \nu I_{HT} - \mu_e E_T, \\ &\frac{dE_T}{dt} + \mu_e E_T = \nu I_{HT}, \\ &\frac{dE_T}{dt} + \mu_e E_T \leq \nu \Phi_t. \end{aligned} \tag{6}$$

Integrating throughout, we obtain

$$E_T(t) \leq \frac{\nu \Phi_t}{\mu_e} + \left(E_T(0) - \frac{\nu \Phi_t}{\mu_e}\right)e^{-\mu_e t}, \tag{7}$$

from which we get

$$E_T(t) \leq \Gamma_t = \max\left\{\frac{\nu \Phi_t}{\mu_e}, E_T(0)\right\}.$$

Using the same approach for the sixth and ninth equations in model system (1), we get

$$P_I(t) \leq \Theta_t = \max\left\{\frac{\omega\Pi_t}{(\alpha_b+\delta)}, P_I(0)\right\} \quad \text{and} \quad B_I(t) \leq \xi_t = \max\left\{\frac{\eta\Psi_t}{(\epsilon+\alpha_b)}, B_I(0)\right\}.$$

Therefore, all solutions of the model system (1) are positive invariant in the region

$$\begin{aligned}\Omega = \big\{&(S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I, E_T) \in \mathbb{R}_+^{10} : 0 \leq H(t) \leq \Phi_t; 0 \leq T_p(t) \leq \Pi_t;\\ &0 \leq T_C(t) \leq \Psi_t; 0 \leq E_T(t) \leq \Gamma_t; 0 \leq P_I(t) \leq \Theta_t; 0 \leq B_I(t) \leq \xi_t\big\}.\end{aligned}$$

All solutions which start at the boundary of region Ω converge to this region. The model system (1) is biologically and epidemiologically meaningful and therefore we can consider the flow generated by the model for analysis.

2.3 Equilibrium states and reproduction number R_0

2.3.1 The disease free equilibrium (E^0)

When there are no infections in humans, pigs and cattle populations, we obtain the disease free equilibrium E^0 which is given by

$$E^0 = \left(\frac{\psi}{\mu_h}, 0, 0, \frac{\Lambda}{(\rho+\mu_p)}, 0, 0, \frac{\phi}{(\sigma+\mu_c)}, 0, 0, 0\right).$$

2.3.2 The basic reproduction number R_0

The basic reproduction number R_0 is the expected number of secondary infections that may occur as a result of introducing one infected individual in a fully susceptible population [9]. When $R_0 < 1$, the disease clears whereas, when $R_0 > 1$, the disease persists within the population. In computing R_0, we adopt the next generation matrix method as used by Van den Driessche and Watmough [31]. Let $\mathcal{F}_i$ be the new infections in compartment i and $\mathcal{V}_i^+$ and $\mathcal{V}_i^-$ be the transfer terms in and out of the compartment i, respectively, then the infected classes can be written as

$$\frac{dx_i}{dt} = \mathcal{F}_i(x) - \mathcal{V}_i^+(x) - \mathcal{V}_i^-(x).$$

Using the next generation matrix method, we define $\mathcal{F}_i$ and $\mathcal{V}_i$ by

$$\mathcal{F}_i = \begin{pmatrix} \beta_T(\alpha_p P_I + \alpha_b B_I)S_H \\ \theta S_H E_T \\ \gamma_p S_P E_T \\ 0 \\ \gamma_c S_C E_T \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V}_i = \begin{pmatrix} \mu_h I_{HT} \\ (\mu_h+\mu_d)I_{HC} \\ (\omega+\mu_p)I_P \\ -\omega I_P + (\delta+\alpha_p)P_I \\ (\eta+\mu_c)I_C \\ -\eta I_C + (\epsilon+\alpha_b)B_I \\ -\nu I_{HT} + \mu_e E_T \end{pmatrix}. \tag{8}$$

The Jacobian of matrices $\mathcal{F}_i$ and $\mathcal{V}_i$ at the disease free equilibrium E^0 are given by

$$F = \frac{\partial \mathcal{F}_i}{\partial x_j}(E^0), \qquad V = \frac{\partial \mathcal{V}_i}{\partial x_j}(E^0). \tag{9}$$

The basic reproduction number R_0 is given by

$$R_0 = \rho\left(FV^{-1}\right). \tag{10}$$

Using the definitions in (9), we have

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_T \alpha_p \psi}{\mu_h} & 0 & \frac{\beta_T \alpha_b \psi}{\mu_h} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\theta\psi}{\mu_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_p \Lambda}{(\rho+\mu_p)} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_c \phi}{(\sigma+\mu_c)} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and}$$

$$V = \begin{pmatrix} \mu_h & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (\mu_h + \mu_d) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\omega + \mu_p) & 0 & 0 & 0 & 0 \\ 0 & 0 & -\omega & \delta + \alpha_p & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta + \mu_c & 0 & 0 \\ 0 & 0 & 0 & 0 & -\eta & (\epsilon + \alpha_b) & 0 \\ -\nu & 0 & 0 & 0 & 0 & 0 & \mu_e \end{pmatrix}.$$

The next generation matrix is given by

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_T \omega \alpha_p S_H^0}{C_1} & \frac{\beta_T \alpha_p S_H^0}{(\delta+\alpha_p)} & \frac{\beta_T \eta \alpha_b S_H^0}{C_2} & \frac{\beta_T \alpha_b S_H^0}{(\epsilon+\alpha_b)} & 0 \\ \frac{\nu\theta S_H^0}{\mu_e \mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\theta S_H^0}{\mu_e} \\ \frac{\nu\gamma_p S_P^0}{\mu_e \mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_p S_P^0}{\mu_e} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\nu\gamma_c S_C^0}{\mu_e \mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_c S_C^0}{\mu_e} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where $C_1 = (\omega + \mu_p)(\delta + \alpha_p)$, $C_2 = (\eta + \mu_c)(\epsilon + \alpha_b)$, $S_H^0 = \frac{\psi}{\mu_h}$, $S_P^0 = \frac{\Lambda}{(\rho+\mu_p)}$ and $S_C^0 = \frac{\phi}{(\sigma+\mu_c)}$. Using the definition in (10), the basic reproduction number R_0 is given by

$$R_0 = \sqrt{R_{HP} + R_{HC}}, \tag{11}$$

where

$$R_{HP} = \frac{\beta_T \nu \alpha_p \gamma_p \omega \psi \Lambda}{\mu_h^2 \mu_e (\omega + \mu_p)(\alpha_p + \delta)(\mu_p + \rho)} \quad \text{and}$$

$$R_{HC} = \frac{\beta_T \nu \alpha_b \gamma_c \eta \psi \phi}{\mu_h^2 \mu_e (\eta + \mu_c)(\alpha_b + \epsilon)(\mu_c + \sigma)}.$$

R_{HP} is the partial reproduction number due to interaction of humans and pigs whereas R_{HC} is the partial reproduction number due to interaction of humans and cattle. To give

the biological meaning for R_0, we rewrite R_{HP} and R_{HC} in the forms

$$\begin{aligned} R_{HP} &= \beta_T \gamma_p \frac{\psi}{\mu_h} \frac{\nu}{\mu_e} \frac{1}{\mu_h} \frac{\Lambda}{(\mu_p + \rho)} \frac{\omega}{(\omega + \mu_p)} \frac{\alpha_p}{(\alpha_p + \delta)}, \\ R_{HC} &= \beta_T \gamma_c \frac{\psi}{\mu_h} \frac{\nu}{\mu_e} \frac{1}{\mu_h} \frac{\phi}{(\mu_c + \sigma)} \frac{\eta}{(\eta + \mu_c)} \frac{\alpha_b}{(\alpha_b + \epsilon)}. \end{aligned} \tag{12}$$

The terms in (12) can be interpreted as follows: ν/μ_e is the density of taenia eggs released by humans with taeniasis, $1/\mu_h$ is the human life expectancy and β_T is the probability of humans to be infected with taeniasis due to consumption of raw or insufficiently cooked pork or beef infected with tapeworm larval cysts. The terms ψ/μ_h, $\Lambda/(\mu_p + \rho)$ and $\phi/(\mu_c + \sigma)$ are initial populations for susceptible humans, susceptible pigs and susceptible cattle, respectively; $1/(\mu_p + \rho)$ and $1/(\mu_c + \sigma)$ are the average times pigs and cattle spend in susceptible classes, respectively; $1/(\omega + \mu_p)$ and $1/(\eta + \mu_c)$ are the infectious periods of infected pigs and cattle, respectively; $1/(\alpha_p + \delta)$ and $1/(\alpha_b + \epsilon)$ are the average infectious period for infected pork and beef, respectively, whereas $\omega/(\omega + \mu_p)$ and $\eta/(\eta + \mu_c)$ are the proportions of infected pigs and cattle that are slaughtered for consumption, respectively. The terms $\alpha_p/(\alpha_p + \delta)$ and $\alpha_b/(\alpha_b + \epsilon)$ are the proportions of infected pork and beef that are eaten by susceptible humans, respectively, whereas γ_p and γ_c are the rates at which *T. solium* eggs and *T. sagnata* eggs are consumed by pigs and cattle, respectively, from the contaminated environment.

2.3.3 Sensitivity analysis

To determine how sensitive the model parameters are to the diseases' transmission, we adopt the normalized forward sensitivity index approach as used by Chitnis et al. [5]. If κ is a parameter in the basic reproduction number R_0, then the sensitivity index of R_0 with respect to κ is given by

$$\Gamma_{\kappa}^{R_0} = \frac{\partial R_0}{\partial \kappa} \times \frac{\kappa}{R_0}. \tag{13}$$

Using Eq. (13) and parameter values in Table 2, we obtain sensitivity indices for each parameter as shown in Table 3. The positive sign of sensitivity index indicates that an increase or decrease of parameter value while keeping other parameters constant increases or decreases the basic reproduction number R_0. The negative sign indicates that an increase or decrease of parameter value causes a decrease or increase in expected new average infection R_0. For instance, $\gamma_p = +0.1421$ means that an increase in γ_p by 10%, increases R_0 by 1.421% and hence the disease transmission; and $\delta = -0.1375$ means that an increase in δ by 20% causes a decrease in R_0 by 2.75% and thus decrease the disease transmission.

The most positive sensitive parameters in the model are human's recruitment (ψ), the probability of humans infection with taeniasis (β_T) and the defecation rate by humans who are infected with taeniasis (ν) whereas the most negative sensitive parameter is the natural mortality rate of humans (μ_h). However, since it is unethical and not practical to increase human natural mortality, other parameters with negative sensitivity indices such as death rate of taenia eggs and proportions of unconsumed infected beef and pork can be considered for disease control.

The sensitivity indices for all parameters in the basic reproduction number R_0 are plotted on a bar graph in Fig. 2.

Table 3 Sensitivity indices

Parameter	Sensitivity Index	Parameter	Sensitivity Index
Λ	+0.1421	ω	+0.1066
ψ	+0.3579	η	+0.2090
ϕ	+0.5000	ν	+0.5000
γ_p	+0.1421	δ	−0.1375
γ_c	+0.3579	ϵ	−0.3343
α_p	+0.1375	μ_p	−0.2200
α_b	+0.3343	μ_h	−1.0000
β_T	+0.5000	μ_c	−0.4266
σ	−0.1404	μ_e	−0.5000
ρ	−0.0287		

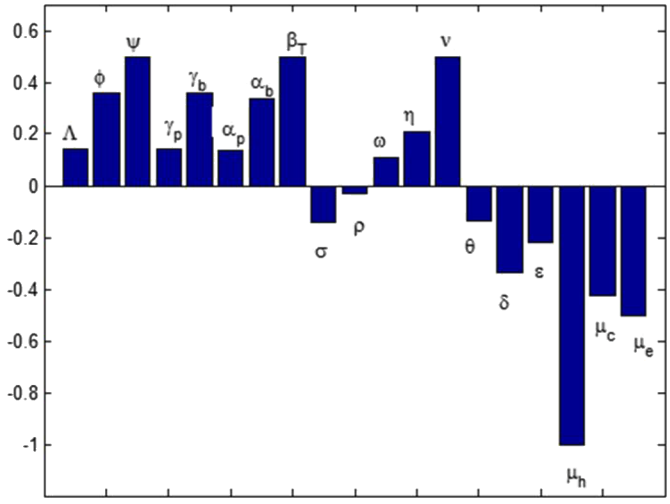

Figure 2 Graphical representation of sensitivity indices

2.3.4 The endemic equilibrium (E^*)

The endemic equilibrium is a point at which taeniasis and cysticercosis exist in humans, pigs and cattle populations. It is obtained by setting the derivatives in model system (1) equal to zero and solving for state variables. The endemic equilibrium $E^* = (S_H^*, I_{HT}^*, I_{HC}^*, S_P^*, I_P^*, P_I^*, S_C^*, I_C^*, B_I^*, E_T^*)$ is given by

$$S_H^* = \frac{\psi}{(\beta_T F_0 E_T^* + \theta E_T^* + \mu_h)}, \qquad I_{HT}^* = \frac{\beta_T F_0 E_T^* S_H^*}{\mu_h}, \qquad I_{HC}^* = \frac{\theta S_H^* E_T^*}{(\mu_h + \mu_d)},$$

$$S_P^* = \frac{\Lambda}{(\gamma_p E_T^* + \rho + \mu_p)}, \qquad I_P^* = \frac{\gamma_p \Lambda E_T^*}{(\gamma_p E_T^* + \rho + \mu_p)(\omega + \mu_p)},$$

$$P_I^* = \frac{\omega \gamma_p \Lambda E_T^*}{(\gamma_p E_T^* + \rho + \mu_p)(\omega + \mu_p)(\delta + \alpha_p)}, \qquad S_C^* = \frac{\phi}{(\gamma_c E_T^* + \sigma + \mu_c)},$$

$$I_C^* = \frac{\gamma_c \phi E_T^*}{(\gamma_c E_T^* + \sigma + \mu_c)(\eta + \mu_c)}, \qquad B_I^* = \frac{\eta \gamma_c \phi E_T^*}{(\gamma_c E_T^* + \sigma + \mu_c)(\eta + \mu_c)(\epsilon + \alpha_b)},$$

where

$$F_0 = \frac{\alpha_p \omega \gamma_p \Lambda}{(\gamma_p E_T^* + \rho + \mu_p)(\omega + \mu_p)(\alpha_p + \delta)} + \frac{\alpha_b \eta \gamma_c \phi}{(\gamma_c E_T^* + \sigma + \mu_c)(\eta + \mu_c)(\alpha_b + \epsilon)}.$$

Table 4 Number of possible positive real roots when $R_0 < 1$ and $R_0 > 1$

Cases	a_0	a_1	a_2	a_3	R_0	No. of Sign Change	No. of + ve Real Roots
1	+	+	+	+	$R_0 < 1$	0	0
2	+	+	+	−	$R_0 > 1$	1	1
3	+	+	−	+	$R_0 < 1$	2	0,2
4	+	+	−	−	$R_0 > 1$	1	1

To obtain E_T^*, we solve for the real roots of the polynomial:

$$a_0 {E_T^*}^3 + a_1 {E_T^*}^2 + a_2 E_T^* + a_3 = 0, \tag{14}$$

where

$$\begin{aligned}
a_0 &= 1 > 0,\\
a_1 &= \frac{\alpha_p \omega \Lambda \beta_T}{\theta(\mu_p+\omega)(\alpha_p+\delta)} + \frac{\alpha_b \eta \phi \beta_T}{\theta(\mu_c+\eta)(\alpha_p+\epsilon)} + \frac{(\mu_c+\sigma)}{\gamma_c} + \frac{(\mu_p+\rho)}{\gamma_p} + \frac{\mu_h}{\theta} > 0,\\
a_2 &= \frac{(\rho+\mu_p)(\sigma+\mu_c)}{\gamma_c\gamma_p} + \frac{\mu_h(\sigma+\mu_c)}{\theta\gamma_c} + \frac{\mu_h(\rho+\mu_p)}{\theta\gamma_p} + \frac{\alpha_p\omega\Lambda\beta_T(\sigma+\mu_c)}{\theta\gamma_c(\mu_p+\omega)(\alpha_p+\delta)}\\
&\quad + \frac{\alpha_b\eta\phi\beta_T(\rho+\mu_p)}{\theta\gamma_p(\mu_c+\eta)(\alpha_p+\epsilon)} - \left(\frac{\nu\psi\beta_T\alpha_p\omega\Lambda\gamma_c}{\mu_h\mu_e\gamma_c\theta(\mu_p+\omega)(\alpha_p+\delta)} + \frac{\nu\psi\beta_T\alpha_b\eta\phi\gamma_p}{\mu_h\mu_e\gamma_p\theta(\mu_c+\eta)(\alpha_p+\epsilon)}\right),\\
a_3 &= \frac{\mu_h(\sigma+\mu_c)(\rho+\mu_p)(1+R_0)(1-R_0)}{\theta\gamma_c\gamma_p}.
\end{aligned}$$

To analyze the possible number of positive real roots of polynomial (14) when $R_0 < 1$ and $R_0 > 1$, we adopt the approach in Okosun et al. [25]. Using this approach, the number of possible real roots when $R_0 < 1$ and $R_0 > 1$ are summarized in Table 4.

Therefore the model system (1) has a unique endemic equilibrium when $R_0 > 1$ as shown in cases 2 and 4. Hence we state the following theorem.

Theorem 1 *The model system* (1) *has a unique endemic equilibrium when the basic reproduction number* $R_0 > 1$.

2.4 Stability analysis of equilibrium states

2.4.1 The global stability of the disease free equilibrium (E^0)

Theorem 2 *The disease free equilibrium* (E^0) *of the model system* (1) *is globally asymptotically stable when* $R_0 < 1$.

Proof To analyze the global stability of the disease free equilibrium, we adopt the approach used in Castillo-Chavez et al. [4] and Dumont et al. [10]. Using this method, the system of differential equations (1) is written as

$$\begin{aligned}
\frac{dX_r}{dt} &= B(X_r - X_{\mathrm{DFE}}) + B_1 X_n,\\
\frac{dX_n}{dt} &= B_2 X_n,
\end{aligned} \tag{15}$$

where X_r and X_n are the non-transmitting and transmitting classes, respectively, X_{DFE} is the disease free equilibrium, whereas B, B_1 and B_2 are the matrices to be computed. Here,

we have

$$\begin{aligned} &X_r = (S_H, S_P, S_C)^T, \\ &X_{\mathrm{DFE}} = \left(\frac{\psi}{\mu_h}, \frac{\Lambda}{\mu_p + \rho}, \frac{\phi}{\mu_c + \sigma}\right)^T. \end{aligned} \tag{16}$$

Thus,

$$X_r - X_{\mathrm{DFE}} = \left(S_H - \frac{\psi}{\mu_h}, S_P - \frac{\Lambda}{\mu_p + \rho}, S_C - \frac{\phi}{\mu_c + \sigma}\right)^T. \tag{17}$$

Therefore from Eq. (15), we have

$$\begin{pmatrix} \psi - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H \\ \Lambda - \gamma_p S_P E_T - (\rho + \mu_p)S_P \\ \phi - \gamma_c S_C E_T - (\sigma + \mu_c)S_P \end{pmatrix} = B \begin{pmatrix} S_H - \frac{\psi}{\mu_h} \\ S_P - \frac{\Lambda}{\mu_p + \rho} \\ S_C - \frac{\phi}{\mu_c + \sigma} \end{pmatrix} + B_1 \begin{pmatrix} I_{HT} \\ I_{HC} \\ I_P \\ P_I \\ I_C \\ B_I \\ E_T \end{pmatrix} \tag{18}$$

and

$$\begin{pmatrix} \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \mu_h I_{HT} \\ \theta S_H E_T - (\mu_h + \mu_d)I_{HC} \\ \gamma_p S_P E_T - (\omega + \mu_p)I_P \\ \omega I_P - (\delta + \alpha_p)P_I \\ \gamma_c S_C E_T - (\eta + \mu_c)I_C \\ \eta I_C - (\epsilon + \alpha_b)B_I \\ \nu I_{HT} - \mu_e E_T \end{pmatrix} = B_2 \begin{pmatrix} I_{HT} \\ I_{HC} \\ I_P \\ P_I \\ I_C \\ B_I \\ E_T \end{pmatrix}. \tag{19}$$

From (18) and (19), the matrices B, B_1 and B_2 are given by

$$B = \begin{pmatrix} -\mu_h & 0 & 0 \\ 0 & -(\rho + \mu_p) & 0 \\ 0 & 0 & -(\sigma + \mu_c) \end{pmatrix},$$

$$B_1 = \begin{pmatrix} 0 & 0 & 0 & -\beta_T \alpha_p S_H & 0 & -\beta_T \alpha_b S_H & -\theta S_H \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_p S_P \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c S_C \end{pmatrix},$$

and

$$B_2 = \begin{pmatrix} -\mu_h & 0 & 0 & \beta_T\alpha_p S_H & 0 & \beta_T\alpha_b S_H & 0 \\ 0 & -(\mu_h+\mu_d) & 0 & 0 & 0 & 0 & \theta S_H \\ 0 & 0 & -(\omega+\mu_p) & 0 & 0 & 0 & \gamma_p S_P \\ 0 & 0 & \omega & -(\delta+\alpha_p) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\eta+\mu_c) & 0 & \gamma_c S_C \\ 0 & 0 & 0 & 0 & \eta & -(\epsilon+\alpha_b) & 0 \\ \nu & 0 & 0 & 0 & 0 & 0 & -\mu_e \end{pmatrix}.$$

It can be observed that matrix B has real and negative eigenvalues. Thus, the system

$$\frac{dX_r}{dt} = B(X_r - X_{\mathrm{DFE}}) + B_1 X_n \tag{20}$$

is globally asymptotically stable at X_{DFE}.

To prove the stability of B_2, we adopt the idea of stable Metzler matrix and apply the lemma in Dumont et al. [10]. A Metzler matrix is a matrix whose of-diagonal elements are non-negative, denoted by $B_2(i,j) \geq 0$, for all $i \neq j$. Thus, it can be observed that B_2 is a Metzler matrix. □

Lemma 1 *Let M be a square Metzler matrix written in block form*:

$$M = \begin{pmatrix} P & Q \\ R & S \end{pmatrix},$$

where P and S are square matrices. M is Metzler stable if and only if matrices P and $S - RP^{-1}Q$ are Metzler stable.

Proof Comparing the Metzler matrix B_2 with a square Metzler matrix M, the matrices P, Q, R and S are defined as

$$P = \begin{pmatrix} -\mu_h & 0 & 0 \\ 0 & -(\mu_h+\mu_d) & 0 \\ 0 & 0 & -(\omega+\mu_p) \end{pmatrix}, \quad Q = \begin{pmatrix} \beta_T\alpha_p S_H & 0 & \beta_T\alpha_b S_H & 0 \\ 0 & 0 & 0 & \theta S_H \\ 0 & 0 & 0 & \gamma_p S_P \end{pmatrix},$$

$$R = \begin{pmatrix} 0 & 0 & \omega \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ \nu & 0 & 0 \end{pmatrix}, \quad S = \begin{pmatrix} -(\delta+\alpha_p) & 0 & 0 & 0 \\ 0 & -(\eta+\mu_c) & 0 & \gamma_c S_C \\ 0 & \eta & -(\epsilon+\alpha_b) & 0 \\ 0 & 0 & 0 & -\mu_e \end{pmatrix}.$$

Clearly, P is a stable Metzler matrix. After some computations, we obtain

$$S - RP^{-1}Q = \begin{pmatrix} -(\delta+\alpha_p) & 0 & 0 & \frac{\omega\gamma_p S_P}{(\omega+\mu_p)} \\ 0 & -(\eta+\mu_c) & 0 & \gamma_c S_C \\ 0 & \eta & -(\epsilon+\alpha_b) & 0 \\ \frac{\nu\beta_T\alpha_p S_H}{\mu_h} & 0 & \frac{\nu\beta_T\alpha_b S_H}{\mu_h} & -\mu_e \end{pmatrix}.$$

Thus, $S - RP^{-1}Q$ is Metzler stable if $\mathrm{Det}(S - RP^{-1}Q) > 0$. That is,

$$\mathcal{W}(\delta + \alpha_p)\mu_e - \frac{\upsilon\beta_T S_H(\omega\alpha_p\gamma_p\mathcal{W}S_P + \eta\alpha_b\gamma_c(\delta + \alpha_b)(\omega + \mu_p)S_C)}{\mu_h(\omega + \mu_p)} > 0, \tag{21}$$

where $\mathcal{W} = (\epsilon + \alpha_c)(\eta + \mu_c)$. Substituting the values for S_H, S_P and S_C at disease free equilibrium into (21) and simplifying the expression, we obtain $1 - R_0^2 > 0$, where R_0 is given in (11). Therefore, the disease free equilibrium E^0 is globally asymptotically stable when $R_0 < 1$. □

2.4.2 *Global stability of the endemic equilibrium* (E^*)

Theorem 3 *The endemic equilibrium* (E^*) *for the model system* (1) *is globally asymptotically stable when* $R_0 > 1$.

Proof Since the endemic equilibrium exists if and only if $R_0 > 1$, we adopt the approach in Osman et al. [26] to prove global stability of endemic equilibrium E^*. Consider the nonlinear Lyapunov function

$$\begin{aligned}\mathcal{L} = {} & S_H^*\left(\frac{S_H}{S_H^*} - \ln\frac{S_H}{S_H^*}\right) + I_{HT}^*\left(\frac{I_{HT}}{I_{HT}^*} - \ln\frac{I_{HT}}{I_{HT}^*}\right) + I_{HC}^*\left(\frac{I_{HC}}{I_{HC}^*} - \ln\frac{I_{HC}}{I_{HC}^*}\right) \\ & + S_P^*\left(\frac{S_P}{S_P^*} - \ln\frac{S_P}{S_P^*}\right) + I_P^*\left(\frac{I_P}{I_P^*} - \ln\frac{I_P}{I_P^*}\right) + P_I^*\left(\frac{P_I}{P_I^*} - \ln\frac{P_I}{P_I^*}\right) + S_C^*\left(\frac{S_C}{S_C^*} - \ln\frac{S_C}{S_C^*}\right) \\ & + I_C^*\left(\frac{I_C}{I_C^*} - \ln\frac{I_C}{I_C^*}\right) + B_I^*\left(\frac{B_I}{B_I^*} - \ln\frac{B_I}{B_I^*}\right) + E_T^*\left(\frac{E_T}{E_T^*} - \ln\frac{E_T}{E_T^*}\right),\end{aligned} \tag{22}$$

The time derivative of the Lyapunov function $\mathcal{L}$ is

$$\begin{aligned}\frac{d\mathcal{L}}{dt} = {} & \left(1 - \frac{S_H^*}{S_H}\right)\frac{dS_H}{dt} + \left(1 - \frac{I_{HT}^*}{I_{HT}}\right)\frac{dI_{HT}}{dt} + \left(1 - \frac{I_{HC}^*}{I_{HC}}\right)\frac{dI_{HC}}{dt} + \left(1 - \frac{S_P^*}{S_P}\right)\frac{dS_P}{dt} \\ & + \left(1 - \frac{I_P^*}{I_P}\right)\frac{dI_P}{dt} + \left(1 - \frac{P_I^*}{P_I}\right)\frac{dP_I}{dt} + \left(1 - \frac{S_C^*}{S_C}\right)\frac{dS_C}{dt} + \left(1 - \frac{I_C^*}{I_C}\right)\frac{dI_C}{dt} \\ & + \left(1 - \frac{B_I^*}{B_I}\right)\frac{dB_I}{dt} + \left(1 - \frac{E_T^*}{E_T}\right)\frac{dE_T}{dt}.\end{aligned} \tag{23}$$

Substituting the equations of model system (1) into Eq. (23), we have

$$\begin{aligned}\frac{d\mathcal{L}}{dt} = {} & \left(1 - \frac{S_H^*}{S_H}\right)(\psi - \beta_T\alpha_p P_I S_H - \beta_T\alpha_b B_I S_H - \theta S_H E_T - \mu_h S_H) \\ & + \left(1 - \frac{I_{HT}^*}{I_{HT}}\right)(\beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \mu_h I_{HT}) \\ & + \left(1 - \frac{I_{HC}^*}{I_{HC}}\right)(\theta S_H E_T - (\mu_h + \mu_d)I_{HC}) \\ & + \left(1 - \frac{S_P^*}{S_P}\right)(\Lambda - \gamma_p S_P E_T - (\rho + \mu_p)S_P) + \left(1 - \frac{I_P^*}{I_P}\right)(\gamma_p S_P E_T - (\omega + \mu_p)I_P) \\ & + \left(1 - \frac{P_I^*}{P_I}\right)(\omega I_P - (\delta + \alpha_p)P_I) + \left(1 - \frac{S_C^*}{S_C}\right)(\phi - \gamma_c S_C E_T - (\sigma + \mu_c)S_C) \\ & + \left(1 - \frac{I_C^*}{I_C}\right)(\gamma_c S_C E_T - (\eta + \mu_c)I_C) + \left(1 - \frac{B_I^*}{B_I}\right)(\eta I_C - (\epsilon + \alpha_b)B_I)\end{aligned}$$

$$+\left(1-\frac{E_T^*}{E_T}\right)(\nu I_{HT}-\mu_e E_T). \tag{24}$$

Equation (24) can also be written as

$$\begin{aligned}\frac{d\mathcal{L}}{dt} &= \psi-\beta_T\alpha_p P_I S_H-\beta_T\alpha_b B_I S_H-\theta S_H E_T-\mu_h S_H\\ &\quad-\frac{\psi S_H^*}{S_H}+\beta_T\alpha_p P_I S_H^*+\beta_T\alpha_b B_I S_H^*+\theta E_T S_H^*+\mu_h S_H^*+\beta_T\alpha_p P_I S_H\\ &\quad+\beta_T\alpha_b B_I S_H-\mu_h I_{HT}-\frac{\beta_T\alpha_p P_I S_H I_{HT}^*}{I_{HT}}-\frac{\beta_T\alpha_b B_I S_H I_{HT}^*}{I_{HT}}+\mu_h I_{HT}^*+\theta S_H E_T\\ &\quad-(\mu_h+\mu_d)I_{HC}-\frac{\theta S_H E_T I_{HC}^*}{I_{HC}}+(\mu_h+\mu_d)I_{HC}^*+\Lambda-\gamma_p S_P E_T-(\rho+\mu_p)S_P\\ &\quad-\frac{\Lambda S_P^*}{S_P}+\gamma_p E_T S_P^*+(\rho+\mu_p)S_P^*+\gamma_p S_P E_T-(\omega+\mu_p)I_P-\frac{\gamma_p S_P E_T I_P^*}{I_P}\\ &\quad+(\omega+\mu_p)I_P^*+\omega I_P-(\delta+\alpha_p)P_I-\frac{\omega I_P P_I^*}{P_I}+(\delta+\alpha_p)P_I^*+\phi-\gamma_c S_C E_T\\ &\quad-(\sigma+\mu_c)S_C-\frac{\phi S_C^*}{S_C}+\gamma_c E_T S_C^*+(\sigma+\mu_c)S_C^*+\gamma_c S_C E_T-(\eta+\mu_c)I_C\\ &\quad-\frac{\gamma_c S_C E_T I_C^*}{I_C}+(\eta+\mu_c)I_C^*+\eta I_C-(\epsilon+\alpha_b)B_I-\frac{\eta I_C B_I^*}{B_I}+(\epsilon+\alpha_b)B_I^*+\nu I_{HT}\\ &\quad-\mu_e E_T-\frac{\nu I_{HT}E_T^*}{E_T}+\mu_e E_T^*.\end{aligned} \tag{25}$$

Similarly, Eq. (25) can be written as

$$\frac{d\mathcal{L}}{dt}=\mathcal{J}-\mathcal{P}, \tag{26}$$

where

$$\begin{aligned}\mathcal{J} &= \psi+\beta_T\alpha_p P_I S_H^*+\beta_T\alpha_b B_I S_H^*+\theta E_T S_H^*+\mu_h S_H^*+\beta_T\alpha_p P_I S_H+\beta_T\alpha_b B_I S_H\\ &\quad+\mu_h I_{HT}^*+\theta S_H E_T+(\mu_h+\mu_d)I_{HC}^*+\Lambda+\gamma_p E_T S_P^*+(\rho+\mu_p)S_P^*+\gamma_p S_P E_T\\ &\quad+(\omega+\mu_p)I_P^*+\omega I_P+(\delta+\alpha_p)P_I^*+\phi+\gamma_c E_T S_C^*+(\sigma+\mu_c)S_C^*+\gamma_c S_C E_T\\ &\quad+(\eta+\mu_c)I_C^*+\eta I_C+(\epsilon+\alpha_b)B_I^*+\nu I_{HT}+\mu_e E_T^*,\\ \mathcal{P} &= \beta_T\alpha_p P_I S_H+\beta_T\alpha_b B_I S_H+\theta S_H E_T+\mu_h S_H+\frac{\psi S_H^*}{S_H}+\mu_h I_{HT}+\frac{\beta_T\alpha_p P_I S_H I_{HT}^*}{I_{HT}}\\ &\quad+\frac{\beta_T\alpha_b B_I S_H I_{HT}^*}{I_{HT}}+(\mu_h+\mu_d)I_{HC}+\frac{\theta S_H E_T I_{HC}^*}{I_{HC}}+\gamma_p S_P E_T+(\rho+\mu_p)S_P\\ &\quad+\frac{\Lambda S_P^*}{S_P}+(\omega+\mu_p)I_P+\frac{\gamma_p S_P E_T I_P^*}{I_P}+\frac{\omega I_P P_I^*}{P_I}+\gamma_c S_C E_T+(\sigma+\mu_c)S_C\\ &\quad+\frac{\phi S_C^*}{S_C}+(\delta+\alpha_p)P_I+(\eta+\mu_c)I_C+\frac{\gamma_c S_C E_T I_C^*}{I_C}+(\epsilon+\alpha_b)B_I+\frac{\eta I_C B_I^*}{B_I}\\ &\quad+\mu_e E_T+\frac{\nu I_{HT}E_T^*}{E_T}.\end{aligned}$$

It can be seen from Eq. (26) that if $\mathcal{J} < \mathcal{P}$ then $\frac{d\mathcal{L}}{dt} < 0$ and if $\Omega = \Omega^*$ then $\frac{d\mathcal{L}}{dt} = 0$. Thus, the largest invariant set in Ω is the endemic equilibrium. Hence from the LaSalle invariant principle [17], we can conclude that, as $t \to \infty$, the solution of the model system (1) approaches the endemic equilibrium when $R_0 > 1$. Therefore, the endemic equilibrium is globally asymptotically stable in the invariant set Ω if $\mathcal{J} < \mathcal{P}$. □

3 Numerical simulations

To understand well the dynamics of taeniasis and cysticercosis in humans, pigs and cattle, we simulate the model (1) using parameters from different literature and some are assumed as indicated in Table 2. To obtain initial conditions we consider a village with 5420 susceptible humans, 750 humans with taeniasis, 528 humans with cysticercosis, 1050 susceptible pigs, 620 infected pigs, 1250 susceptible cattle, 850 infected cattle and 1000 taenia eggs in the environment. We quantify pork and beef in terms of the number of infected pigs and cattle that are slaughtered for consumption. The Runge–Kutta order 4 numerical method is used to simulate the model in MATLAB software.

3.1 The fourth order Runge–Kutta method (RK4)

The 4th order Runge Kutta method (RK4) is a numerical method for solving a system of ordinary differential equations. The advantage of using RK4 over other numerical methods is that it is more accurate as it has high-order local truncation error $O(h^4)$ of the Taylor methods and eliminates the need of computing and evaluating the derivatives of a function. RK4 approximates the solution of the initial value system of the first order differential equation of the form

$$\frac{dy}{dt} = f\big(t, y(t)\big); \qquad y(t_0) = y_0.$$

The method uses the initial value of the function to start the algorithm:

$$k_1 = hf(t_i, y_i,), \qquad k_2 = hf\left(t_i + \frac{h}{2}, y_i + \frac{1}{2}k_1,\right), \qquad k_3 = hf\left(t_i + \frac{h}{2}, y_i + \frac{1}{2}k_2,\right),$$
$$k_4 = hf(t_{i+1}, y_i + k_3,), \qquad y_{i+1} = y_i + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4),$$

for each $i = 1, 2, 3, \ldots, N-1$, where $h > 0$ is the given step size and k_1, k_2, k_3, k_4 are constants that are used to eliminate the need for successive nesting in the second variable of $f(t, y)$ [3].

3.2 Model simulation

The infected cattle and pigs increase initially to their maximum in the first six months and later they decline and remain constant as illustrated in Fig. 3(a) and (b). The decline of infected cattle and pigs is in correspondence with the decline of taenia eggs in environment as shown in Fig. 3(d). This situation can happen if humans with taeniasis have toilets and hence do not shed taenia eggs in the environment. Susceptible cattle and pigs decrease to their lowest following infection by cysticercosis. However, they increase between the first and fourth year and thereafter remain constant.

In Fig. 3(c), susceptible humans decline rapidly in the first six months. However, at the end of the first year, susceptible humans increase gradually until the third year where they

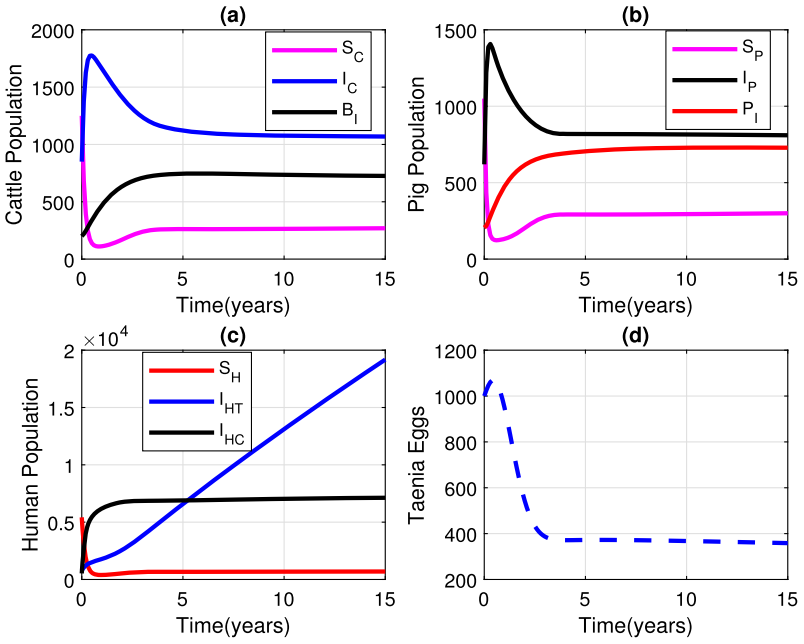

Figure 3 Dynamics of Humans, Pigs, Cattle and Taenia Eggs

remain constant. Humans with cysticercosis increase rapidly in the first six months and then with gradual increase until the second year where they remain constant. These trends are caused by variation of taenia eggs in the environment as depicted by Fig. 3(d) and consumption of raw or undercooked infected pork and beef. On the other hand, humans who are infected with taeniasis increase with time in the first two years and then increases linearly with time. This can happen if humans have habit of consuming raw or insufficiently cooked infected beef and pork.

3.3 Effect of varying the most sensitive parameters

In this subsection we present numerical simulation by considering the most sensitive parameters to observe how they affect disease transmission in humans, pigs and cattle. From the sensitivity analysis, the most positive sensitive parameters are the human recruitment rate (ψ), probability of humans to be infected with taeniasis (β_T) and the defection rate of humans with taeniasis (ν) whereas the most negative sensitive parameter is the natural mortality rate of humans (μ_h).

3.3.1 Effect of varying human recruitment rate (ψ)

The dynamics of taeniasis and cysticercosis shows that humans with taeniasis and cysticercosis, and infected cattle and pigs will increase in proportion to the human recruitment rate as illustrated in Fig. 4.

3.3.2 Effect of varying defecation rate (ν)

Infected pigs and cattle, and humans who are infected with cysticercosis increase with time as a result of increase in defecation rate as depicted by Fig. 5(a), (b), (d). A different trend can be observed for humans who are infected with taeniasis in Fig. 5(c) where there is a

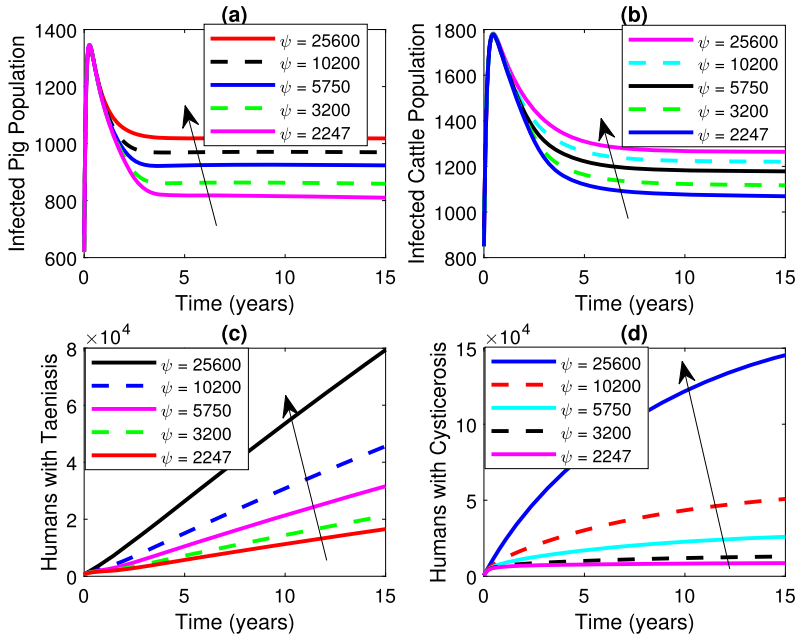

Figure 4 Impact of varying human recruitment rate on infected populations

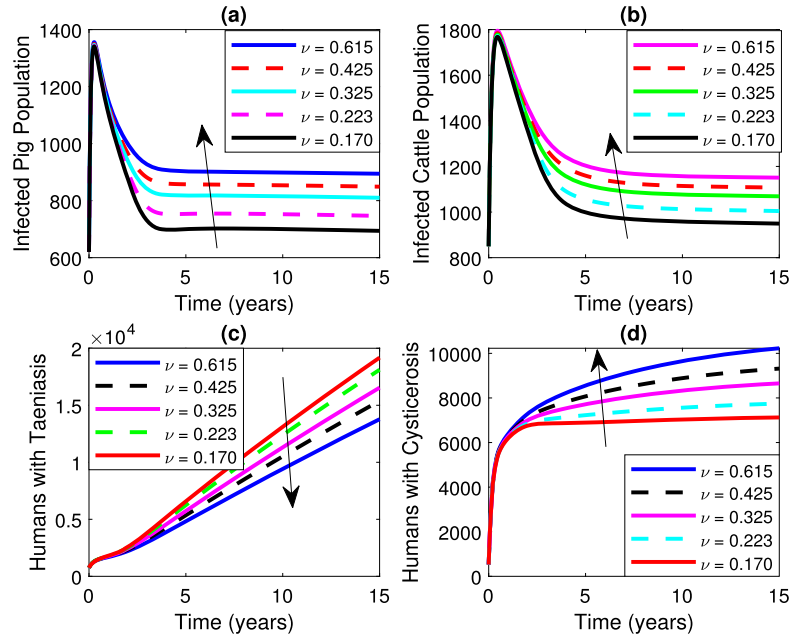

Figure 5 Impact of varying defecation rate on infected populations

decrease in cases of humans who are infected when the human defecation rate is increased. This is due to the fact that a high defecation rate can result in an expel of a high number of taenia eggs and so the possibility of recovery.

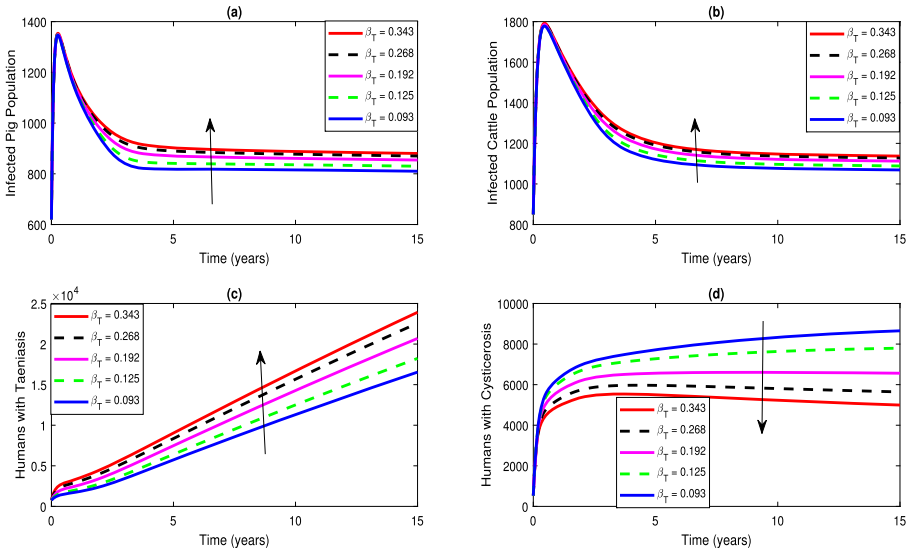

Figure 6 Impact of varying probability of infection on infected populations

3.3.3 Effect of varying probability of taeniasis infection (β_T)

In Fig. 6(a), (b), (c); the infected pigs, cattle and humans who are infected with taeniasis show an increase with time when the probability of taeniasis infection is increased. A different trend can be seen for humans who are infected with cysticercosis in Fig. 6(d), which shows the decrease in disease prevalence with increase in probability of taeniasis infection. This is because humans cysticercosis does not depend on the probability of human infection with taeniasis but on the rate at which humans consume *T. solium* eggs from the contaminated environment.

3.3.4 Effect of varying human natural death rate (μ_h)

In Fig. 7, the results show that an increase in human natural mortality rate has negative impact in disease prevalence for all infected sub-populations. This indicates that human beings play an important role in transmission of the two diseases.

4 Conclusion and recommendation

In this paper, a mathematical model for the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle is presented and analyzed. The model is well posed since the model solutions are positive and bounded. The disease free and endemic equilibria exist and their stability are investigated. The next generation approach is used to compute the basic reproduction number R_0. The analysis has shown that the disease free equilibrium is globally asymptotically stable when basic reproduction number $R_0 < 1$ while the endemic equilibrium is globally asymptotically stable when $R_0 > 1$. The effect of the most sensitive parameters in the diseases' transmission dynamics was assessed. Numerical results indicate that increasing human recruitment rate (ψ) leads to an increased disease prevalence whereas increasing human natural death rate (μ_h) reduces disease prevalence in all populations. On the other hand, increasing probability of human infection with taeniasis (β_T) increases the number of infected pigs, cattle and humans with taeniasis and reduces the number of cases for humans with cysticercosis. Similarly, increasing open hu-

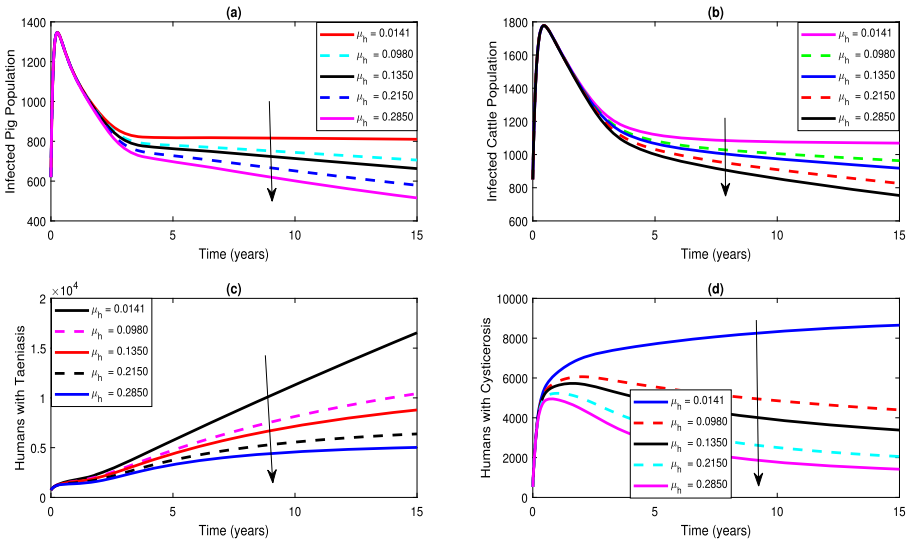

Figure 7 Impact of varying human natural death rate on infected populations

man defecation rate (ν) leads to an increased number of cases for infected pigs, cattle and humans with cysticercosis and decreases the number of cases for humans with taeniasis.

To control the diseases, the open human defecation rate should be reduced through use of toilets, especially in rural communities where pigs and cattle are kept under a free range system, treatment of infected individuals and proper cooking of pork and beef. To reduce the rate of transfer of infections from contaminated environment, we recommend pigs and cattle vaccination, indoor keeping of pigs and cattle as well as improvement in hygiene and sanitation. Moreover, infected pigs and cattle should be treated and meat inspection should be promoted to reduce the rate at which humans are infected with taeniasis.

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Competing interests
The authors declare that they have no competing interests.

Authors' contributions
JAM: Conceptualization, Model Formulation, Model Analysis and Drafting of the Manuscript; JII: Model Formulation and Supervision; DK: Supervision; and DK: Supervision. All authors read and approved the final manuscript.

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