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Taenia solium taeniasis and cysticercosis: extinction or outbreak

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Abstract

Taenia solium taeniasis and cysticercosis are neglected zoonotic diseases that affect human health and economies of developing countries. In this work, we formulate and analyze deterministic and continuous time Markov chain (CTMC) stochastic models to determine parameters that drive *Taenia solium* taeniasis and cysticercosis and the likelihood of their extinction. The basic reproduction number R_0 is computed by the next generation matrix approach, sensitivity index of each parameter in R_0 is derived by the normalized forward sensitivity index and the likelihood of diseases' extinction is computed by the multitype branching process. The analysis shows that humans with *Taenia solium* taeniasis, infectious pork and *Taenia solium* eggs in the environment play an important role in the transmission of *Taenia solium* taeniasis and cysticercosis, and the model exhibits forward bifurcation at $R_0 = 1$. This implies that $R_0 < 1$ is a sufficient condition to eliminate *Taenia solium* taeniasis and cysticercosis. For CTMC model, analysis shows that the probability of *Taenia solium* taeniasis and cysticercosis extinction is high if the diseases emerge from humans with *Taenia solium* cysticercosis and there is an outbreak if the diseases emerge from either humans with *Taenia solium* taeniasis or infectious pork or *Taenia solium* eggs in the environment. To control *Taenia solium* taeniasis and cysticercosis, the intervention strategies should focus on improving hygiene and sanitation for reducing shedding rate of *Taenia solium* eggs in the environment, inspection of pork for reducing the rate of acquiring *Taenia solium* taeniasis and spraying of insecticides for killing *Taenia solium* eggs in the environment.

Keywords Taeniasis, Cysticercosis, Basic reproduction number, Sensitivity analysis, Multitype branching process, Stochastic model

Introduction

Taenia solium taeniasis and cysticercosis are neglected diseases that impose setback to health and economic sectors in developing countries (Mwanjali et al., 2013; Trevisan et al., 2018). The diseases are caused by *Taenia solium* tapeworm which infects both humans and pigs (Ngowi et al., 2007). The adult stage of *Taenia solium* tapeworm causes intestinal infection in humans called taeniasis and larval stage infects tissues in both humans and pigs causing cysticercosis (Gebrie and Engdaw, 2015; Mwanjali et al., 2013; Mwidunda et al., 2015; Schmidt et al., 2019). The diseases are mainly associated with poor sanitation and hygiene, pork consumption and free range pig keeping (Mwanjali et al., 2013; Trevisan et al., 2017). Developed countries are also vulnerable to the diseases due to global business, international tourism, and increased migration and refugees (Fernanda et al., 2010; José et al., 2018; Schantz et al., 1998; Sorvillo et al., 2011). Approximately 2.5 million people globally carry adult *Taenia solium* and at least 20 million are infected with cysticerci of *Taenia solium* (Pawlowski et al., 2005).

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In the dynamics of *Taenia solium* taeniasis and cysticercosis, pig is an intermediate host and human being is a definitive host (Gebrie and Engdaw, 2015; Maridadi et al., 2011; Trevisan et al., 2017). Interaction of human beings and pigs in the environment maintains the disease life cycle (Trevisan et al., 2017). Humans acquire *Taenia solium* taeniasis when they eat raw or prebaked pork contaminated with cysticerci (Dixon et al., 2019; Gebrie and Engdaw, 2015; Kyvsgaard et al., 2007; Mwanjali et al., 2013; Mwidunda et al., 2015; Schmidt et al., 2019; Shonyela et al., 2017; Yanagida et al., 2012). In the human intestine, cysticerci develop into adult tapeworms *Taenia solium* (Sorvillo et al., 2007) whose egg-bearing gravid proglottids pass into the environment during defecation (Canadian Paediatric Society, 2019; Gebrie and Engdaw, 2015) and live in the environment for a number of months (Gebrie and Engdaw, 2015). Adult tapeworm carrier can pass millions of eggs on a daily basis either freely in faeces (Schmidt et al., 2019) or as intact segments containing about 250, 000 eggs each (Gebrie and Engdaw, 2015). Pigs contract *Taenia solium* cysticercosis through direct consumption of human faeces containing *Taenia solium* eggs or through feeding on the contaminated environment (Mwidunda et al., 2015; Ngowi et al., 2007; Schmidt et al., 2019). The eggs develop oncospheres embryos which enter the intestinal wall and later disseminate to various tissues via bloodstream to form cysticerci (Garcia and Del Brutto, 2000; Sorvillo et al., 2011).

Human beings acquire *Taenia solium* cysticercosis through ingesting *Taenia solium* eggs in contaminated food or water (Gebrie and Engdaw, 2015; Mwanjali et al., 2013; Sorvillo et al., 2011; Trevisan et al., 2017; WHO, 2019). The ingested *Taenia solium* eggs hatch larvae in the intestine which later migrate to various tissues to form cysticerci (Canadian Paediatric Society, 2019). *Taenia solium* cysticercosis becomes critical when cysticerci migrate to the central nervous system. Infection of central nervous system by cysticerci leads to neurocysticercosis (Del Brutto et al., 2014; García et al., 2003; Mwidunda et al., 2015) which causes neurologic disease (Garcia et al. 2012; Sorvillo et al., 2011; White, 2000) with symptoms such as severe headache, epilepsy, seizure, sightlessness, mental interruption and even fatality (García et al., 2003; Garcia et al., 2016; José et al., 2018; Phiri et al., 2003; Trevisan et al., 2017). Neurocysticercosis is estimated to cause 50, 000 deaths annually (Boa et al., 2006; Garcia and Del Brutto, 2000). In Tanzania, 17, 853 epilepsy cases and 212 deaths due to neurocysticercosis were reported in 2012. It is estimated that 183, 927 pigs (which is 11.7% of total pigs in Tanzania) were affected with cysticercosis and their market value depreciated to half (Trevisan et al., 2017) or 60% of uninfected pig (Boa et al., 2006).

Though the impact of *Taenia solium* taeniasis and cysticercosis is felt among the communities in the developing countries, public knowledge on modes of transmission in many endemic areas is poor (Gebrie and Engdaw, 2015; Mwidunda et al., 2015; Ngowi et al., 2007; Trevisan et al., 2017). This is the main factor that makes human beings to take part in diseases' transmission (Mwidunda et al., 2015; Ngowi et al., 2007). Lack of knowledge for *Taenia solium* taeniasis and cysticercosis has led communities in developing world especially Africa to associate epilepsy (which is caused by neurocysticercosis) with witchcraft or demonic possession (Winkler et al., 2010). Studies conducted in Tanzania have shown that the communities' knowledge gap on the diseases and the modes of transmission is the problem that facilitate diseases' persistence. Ngowi et al. (2007) have shown that, communities' poor knowledge on modes of transmission and poor pig management practices are factors which influence people to behaviour that facilitate diseases' transmission. Other studies also found communities' knowledge gap (Maridadi et al., 2011; Mwidunda et al., 2015; Shonyela et al., 2017).

To bridge the knowledge gap, deterministic and continuous time Markov chain (CTMC) stochastic models are employed to study the transmission dynamics of *Taenia solium* taeniasis and cysticercosis. CTMC stochastic models are important tools for studying the dynamics of infectious diseases and determining the likelihood of diseases' extinction or outbreak. Deterministic models use the basic reproduction number R_0 to predict the disease dynamics and ignore uncertainties that can influence the disease dynamics. In deterministic approach, the disease clears when the basic reproduction number $R_0 < 1$ and persists when $R_0 > 1$. While deterministic modeling approach ignores uncertainties that influence the disease dynamics, its continuous time Markov chain modeling counterpart takes into account the uncertainties that are inherent to disease transmission and it has the likelihood for disease extinction. Unlike deterministic models, stochastic models can predict extinction of the disease even if the stochastic threshold is greater than unity. Most of mathematical models that are developed to study *Taenia solium* taeniasis and cysticercosis are deterministic in nature and ignore the influence of uncertainties that are inherent to the diseases' transmission.

In this work, CTMC models are formulated and analyzed to study dynamics of *Taenia solium* taeniasis and cysticercosis. The multitype branching process is used to determine the likelihood for diseases' extinction. The organization of this article is as follows: we begin with deterministic model and its analysis followed by the stochastic model. Numerical analysis is presented before conclusion and recommendations.

Methods and Results

Deterministic model

Development and analysis

Taenia solium taeniasis and cysticercosis model considers human beings, pigs and *Taenia solium* eggs in the environment. Human population is divided into susceptible S_H, humans with *Taenia solium* taeniasis I_T, and humans with *Taenia solium* cysticercosis I_C. Pigs' population is also divided into susceptible pigs S_P and infected pigs I_P. The infectious pork and *Taenia solium* eggs are represented by M and P_a respectively.

Susceptible humans S_H are replenished at a rate Λ_H and contract *Taenia solium* taeniasis and cysticercosis at respective rates

$$\lambda_T = \alpha_H \beta_T M \text{ and } \lambda_C = r\beta_C P_a. \tag{1}$$

Humans with *Taenia solium* taeniasis I_T increase as susceptible individuals acquire *Taenia solium* taeniasis at a rate λ_T and humans with *Taenia solium* cysticercosis I_C increase when susceptible humans eat food or drink water which is contaminated with *Taenia solium* eggs at a rate λ_C. Humans with *Taenia solium* cysticercosis I_C suffer death due to disease at a rate α_C (Fernanda et al., 2010; Sorvillo et al., 2007) and all human classes suffer death due natural causes at a rate μ_H.

Susceptible pigs S_P are recruited at a constant rate Λ_P and diminish by contracting *Taenia solium* cysticercosis at a rate

$$\lambda_P = c_P \beta_P P_a, \tag{2}$$

and when they are harvested at a rate α_P. Pigs which are *Taenia solium* cysticercosis carriers I_P increase at a rate Λ_P and decrease as they are slaughtered at a rate γ_P. All pig classes are assumed to suffer natural mortality at a rate μ_P. Infectious pork M increase following slaughtering of infected pigs at rate γ_P. However, it decreases following consumption by susceptible humans at a rate α_H and decays at a rate α_m (α_m is the proportion of infectious pork that is not consumed by human beings). *Taenia solium* eggs P_a are replenished when humans with *Taenia solium* taeniasis defecate in the open environment at a rate γ_E, and diminish due to natural mortality at a rate μ_E.

The model assumes that: the number of *Taenia solium* eggs which are ingested to cause *Taenia solium* cysticercosis in humans and pigs is negligible thus has no significant impact on their total number in the environment (Winskill et al., 2017). For model symplicity, auto infection and individuals who have both *Taenia solium* taeniasis and cysticercosis are not considered. Transmission of *Taenia solium* taeniasis and cysticercosis is density dependent. Susceptible humans who acquire both *Taenia solium* taeniasis and cysticercosis are not considered. Susceptible pigs are harvested at a constant rate and all pigs are assumed to be kept under free range system. Table 1 described model parameters and interactions of variables are described in Fig. 1.

Using parameter values in Table 1 and state variables in Fig. 1, the model system for *Taenia solium* taeniasis and cysticercosis is given by:

$$\frac{dS_H}{dt} = \Lambda_H - (\lambda_C + \lambda_T + \mu_H)S_H, \tag{3a}$$

$$\frac{dI_C}{dt} = \lambda_C S_H - (\alpha_C + \mu_H)I_C, \tag{3b}$$

$$\frac{dI_T}{dt} = \lambda_T S_H - \mu_H I_T, \tag{3c}$$

$$\frac{dS_P}{dt} = \Lambda_P - \Lambda_P S_P - (\alpha_P + \mu_P)S_P, \tag{3d}$$

$$\frac{dI_P}{dt} = \Lambda_P S_P - (\gamma_P + \mu_P)I_P, \tag{3e}$$

$$\frac{dM}{dt} = \gamma_P I_P - (\alpha_H + \alpha_m)M, \tag{3f}$$

$$\frac{dP_a}{dt} = \gamma_E I_T - \mu_E P_a, \tag{3g}$$

$$S_H(0) > 0, I_C(0) \geq 0, I_T(0) \geq 0, S_P(0) > 0, I_P(0) \geq 0, M(0) \geq 0, P_a(0) \geq 0.$$

Positivity and boundedness of solutions

The positivity of model solutions and their bounds are proved by the following theorem:

Table 1 Parameters' description

Parameter	Description	Value	Source
Λ_H	Human recruitment rate	100	Mwasunda et al. 2022
Λ_P	Pigs' recruitment rate	1450	Mwasunda et al. 2021
μ_H	Human natural mortality rate	0.0141	Mwasunda et al. 2021
μ_P	Pigs' natural mortality rate	0.996	Mwasunda et al. 2021
μ_E	*Taenia solium* eggs natural mortality	0.95	Assumed
α_H	Rate at which human consume infectious pork	0.012	Mwasunda et al. 2021
α_m	Proportion of infectious pork that decays	0.3	Assumed
α_C	Human cysticercosis induced mortality	0.15	Assumed
α_P	Rate at which susceptible pigs are slaughtered	0.0252	Assumed
γ_P	Rate of producing cysts in infected pork	0.132	Assumed
γ_E	Rate at which infected human release *Taenia solium* eggs in the environment	0.35	Assumed
β_T	Probability of acquiring *Taenia solium* taeniasis per contact by human	0.011	Assumed
β_C	Probability of acquiring *Taenia solium* cysticercosis per contact by human	0.0123	Assumed
β_P	Probability of acquiring *Taenia solium* cysticercosis per contact by pig	0.00108	Assumed
r	Number of contacts a human makes with *Taenia solium* eggs in the environment	0.125	Assumed
c_P	Number of contacts a pig makes with *Taenia solium* eggs in the environment	2	Assumed

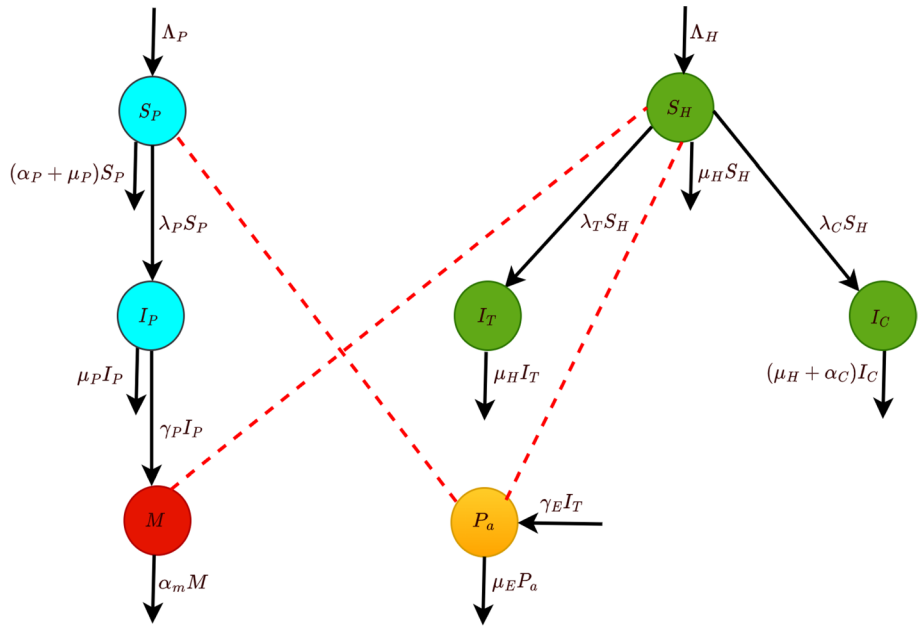

Fig. 1 Model flow chart for transmission dyamics of *Taenia solium* taeniasis and cysticercosis

Theorem 1 *For non-negative initial conditions, the system* (3) *has non-negative solutions which are positively invariant and globally attracting in the region*

$$\Pi = \left\{(S_H, I_C, I_T, S_P, I_P, M, P_a) \in \mathbb{R}_+^7 : N_H \leq \frac{\Lambda_H}{\mu_H}, N_P \leq \frac{\Lambda_P}{\alpha_P+\mu_P}, M \leq \frac{\gamma_P \Lambda_P}{\alpha_P+\mu_P}, P_a \leq \frac{\gamma_E \Lambda_H}{\mu_H}\right\}, \tag{4}$$

where N_H *and* N_P *denote the total populations for humans and pigs respectively.*

Proof

To prove Theorem 1, we re-write the system (3) *in the form*

$$y'(t) = h(y) \tag{5}$$

where $y = (y_1, y_2, y_3, y_4, y_5, y_6, y_7) = (S_H, I_C, I_T, S_P, I_P, M, P_a)$ and $h(y) = \big(h_1(y), h_2(y), \cdots, h_7(y)\big)$ represent the rate of change for the corresponding state variables. In this form, the system (5) is;;

$$\frac{dy_1}{dt} = h_1 = \Lambda_H - r\beta_C y_1 y_7 - \alpha_H \beta_T y_1 y_6 - \mu_H y_1, \tag{6a}$$

$$\frac{dy_2}{dt} = h_2 = r\beta_C y_1 y_7 - (\alpha_C + \mu_H) y_2, \tag{6b}$$

$$\frac{dy_3}{dt} = h_3 = \alpha_H \beta_T y_1 y_6 - \mu_H y_3, \tag{6c}$$

$$\frac{dy_4}{dt} = h_4 = \Lambda_P - c_P \beta_P y_4 y_7 - (\alpha_P + \mu_P) y_4, \tag{6d}$$

$$\frac{dy_5}{dt} = h_5 = c_P \beta_P y_4 y_7 - (\gamma_P + \mu_P) y_5, \tag{6e}$$

$$\frac{dy_6}{dt} = h_6 = \gamma_P y_5 - (\alpha_H + \alpha_m) y_6, \tag{6f}$$

$$\frac{dy_7}{dt} = h_7 = \gamma_E y_3 - \mu_E y_7. \tag{6g}$$

We can see that for all $i = 1, \cdots, 7$, $h_i(y) \geq 0$ if $y \in [0, \infty)^7$ and $y_i = 0$. Since humans, pigs and *Taenia solium* eggs populations are non-negative, then the rate of change for each variable in system (5) is locally Lipschitz in Π and system (3) has a unique solution.

Now using total humans and pigs populations, we have:

$$\begin{aligned} \frac{dN_H}{dt} &= \Lambda_H - \mu_H N_H - \alpha_C y_2, \\ \frac{dN_P}{dt} &= \Lambda_P - \mu_P N_P - \alpha_P y_4 - \gamma_P y_5. \end{aligned} \tag{7}$$

Since $\frac{dN_H}{dt} \leq \Lambda_H - \mu_H N_H$ and $\frac{dN_P}{dt} \leq \Lambda_P - (\mu_P + \alpha_P) N_P$, then $\frac{dN_H}{dt} \leq 0$ if $N_H(t) \geq \frac{\Lambda_H}{\mu_H}$ and $\frac{dN_P}{dt} \leq 0$ if $N_P(t) \geq \frac{\Lambda_P}{\alpha_P + \mu_P}$. By standard comparison theorem, it is clear that: $N_H(t) \leq \frac{\Lambda_H}{\mu_H}$ when $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$ and $N_P(t) \leq \frac{\Lambda_P}{\alpha_P + \mu_P}$ when $N_P(0) \leq \frac{\Lambda_P}{\alpha_P + \mu_P}$.

Considering infectious pork and *Taenia solium* eggs, we can show that

$M(t) \leq \frac{\gamma_P \Lambda_P}{\alpha_P + \mu_P}$ when $M(0) \leq \frac{\gamma_P \Lambda_P}{\alpha_P + \mu_P}$ and $P_a(t) \leq \frac{\gamma_E \Lambda_H}{\mu_H}$ when $P_a(0) \leq \frac{\gamma_E \Lambda_H}{\mu_H}$.

Thus the region:

$$\Pi = \left\{ (S_H, I_C, I_T, S_P, I_P, M, P_a) \in \mathbb{R}_+^7 : N_H \leq \frac{\Lambda_H}{\mu_H}, N_P \leq \frac{\Lambda_P}{\alpha_P + \mu_P}, M \leq \frac{\gamma_P \Lambda_P}{\alpha_P + \mu_P}, P_a \leq \frac{\gamma_E \Lambda_H}{\mu_H} \right\}$$

is positive invariant and the solutions of the model system (3) enter the region in finite time and the flow generated can be considered for analysis.

Equilibrium states and basic reproduction number R_0

In the absence of *Taenia solium* taeniasis and cysticercosis, the system has disease free equilibrium:

$$T^0(S_H, I_C, I_T, S_P, I_P, M, P_a) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, \frac{\Lambda_P}{\mu_P + \alpha_P}, 0, 0, 0 \right). \tag{8}$$

To determine whether *Taenia solium* taeniasis and cysticercosis clear or persist, the basic reproduction number R_0 that measures the average new infections is computed by next generation matrix approach (Van den Driessche and Watmough, 2002; Winskill et al., 2017). *Taenia solium* taeniasis and cysticercosis clear when $R_0 < 1$ and persist when $R_0 > 1$ (Diekmann et al., 1990; Van den Driessche and Watmough, 2002). When $R_0 < 1$, the average new infections are less than one thus *Taenia solium* taeniasis and cysticercosis die out (Van den Driessche and Watmough, 2002) while when $R_0 > 1$, average new infections are greater than one therefore the diseases persist.

If we define the vectors for new infections and transition terms from model (3) by F_i and V_i respectively, then the basic reproduction number R_0 is given as the maximum eigenvalue of the next generation matrix FV^{-1} (Van den Driessche and Watmough, 2002), that is:

$$R_0 = \rho(FV^{-1}), \tag{9}$$

where

$$F = \frac{\partial F_i}{\partial X_j}(T^0), \text{ and } V = \frac{\partial V_i}{\partial X_j}(T^0).$$

From equation (9), the basic reproduction number R_0 is given by:

$$R_0 = \sqrt{\frac{\gamma_P \gamma_E \alpha_H c_P \beta_P \beta_T \Lambda_P \Lambda_H}{\mu_E \mu_H^2 (\alpha_P + \mu_P)(\gamma_P + \mu_P)(\alpha_m + \alpha_H)}}. \tag{10}$$

The basic reproduction number R_0 depends on the proportion at which infected pigs are slaughtered, the proportion of infectious pork that decays, the rate at which *Taenia solium* eggs are shed in the environment, the rates of acquiring *Taenia solium* taeniasis by humans and *Taenia solium* cysticercosis by pigs, the life expectancy for

human, pig and *Taenia solium* eggs, human initial population and the rate at which pigs are recruited. The relative impact of each parameter in the basic reproduction number R_0 is determined by sensitivity analysis.

Sensitivity analysis

The relative impact of each parameter in the basic reproduction number R_0 is determined by normalized forward sensitivity index. If β_i is a parameter in the basic reproduction number R_0, then its relative impact is measured by sensitivity index given by:

$$\Upsilon_{\beta_i}^{R_0} = \frac{\partial R_0}{\partial \beta_i} \times \frac{\beta_i}{R_0}. \tag{11}$$

Using Eq. (11), the sensitivity indices for parameters in the basic reproduction number R_0 are summarized in Table 2. Analysis shows that, the average new infections will increase if pigs are recruited and acquire *Taenia solium* cysticercosis, humans acquire *Taenia solium* taeniasis, and when *Taenia solium* eggs are shed in the environment. However, the average new infections will diminish if natural mortality for pigs and *Taenia solium* eggs increases, a large proportion of infectious pork decays and susceptible pigs are highly harvested.

$$\alpha_H \beta_T M^* S_H^* - \mu_H I_T^* = 0, \tag{14}$$

$$\Lambda_P - c_P \beta_P P_a^* S_P^* - (\alpha_P + \mu_P) S_P^* = 0, \tag{15}$$

$$c_P \beta_P P_a^* S_P^* - (\gamma_P + \mu_P) I_P^* = 0, \tag{16}$$

$$\gamma_P I_P^* - (\alpha_H + \alpha_m) M^* = 0, \tag{17}$$

$$\gamma_E I_T^* - \mu_E P_a^* = 0. \tag{18}$$

Finding the equilibrium states, we solve for S_H^*, I_C^*, S_P^*, I_P^*, M^* and P_a^* in terms of I_T^* to obtain:

$$S_H^* = \frac{\mu_E \Lambda_H}{\gamma_E (r\beta_C + c_P \beta_P) I_T^* + \mu_E \mu_H}, \quad I_C^* = \frac{\Lambda_H r \beta_C \gamma_E I_T^*}{(\gamma_E (r\beta_C + c_P \beta_P) I_T^* + \mu_E \mu_H)(\alpha_C + \mu_H)},$$
$$S_P^* = \frac{\mu_E \Lambda_P}{c_P \beta_P \gamma_E I_T^* + \mu_E (\alpha_P + \mu_P)}, \quad I_P^* = \frac{c_P \beta_P \gamma_E \Lambda_P I_T^*}{(c_P \beta_P \gamma_E I_T^* + \mu_E (\alpha_P + \mu_P))(\gamma_P + \mu_P)},$$
$$M^* = \frac{\gamma_P \gamma_E c_P \beta_P \Lambda_P I_T^*}{(c_P \beta_P \gamma_E I_T^* + \mu_E (\alpha_P + \mu_P))(\gamma_P + \mu_P)(\alpha_H + \alpha_m)}, \quad P_a^* = \frac{\gamma_E I_T^*}{\mu_E}.$$

Substituting S_H^* and M^* in Eq. (14), we obtain the polynomial in I_T^* given by

$$I_T^* (A I_T^{*2} + B I_T^* + C) = 0, \tag{19}$$

where

$$A = \mu_H \gamma_E^2 c_P \beta_P (r\beta_C + c_P \beta_P)(\gamma_P + \mu_P)(\alpha_H + \alpha_m),$$
$$B = \mu_H \mu_E \gamma_E [(r\beta_C + c_P \beta_P)(\alpha_P + \mu_P) + \mu_H c_P \beta_P](\gamma_P + \mu_P)(\alpha_H + \alpha_m),$$
$$C = \mu_E^2 \mu_H^2 (\gamma_P + \mu_P)(\alpha_H + \alpha_m)(\alpha_P + \mu_P)(1 + R_0)(1 - R_0).$$

Analysis of equilibrium states

We obtain the equilibrium states by setting the right handside of model system (3) equal to zero and solve for the state variables. The model system (3) is therefore rewritten as:

$$\Lambda_H - r\beta_C P_a^* S_H^* - \alpha_H \beta_T M^* S_H^* - \mu_H S_H^* = 0, \tag{12}$$

$$r\beta_C P_a^* S_H^* - (\alpha_C + \mu_H) I_C^* = 0, \tag{13}$$

Table 2 Sensitivity indices

Parameter	Sensitivity index	Parameter	Sensitivity index
γ_E	+0.5000	γ_P	+0.2150
c_P	+0.5000	μ_H	-1.0000
β_P	+0.5000	μ_P	-0.6141
β_T	+0.5000	μ_E	-0.5000
Λ_P	+0.5000	α_m	-0.4808
Λ_H	+0.5000	α_P	-0.1010
α_H	+0.4808		

The polynomial (19) has two solutions which are $I_T^* = 0$ and $AI_T^{*2} + BI_T^* + C = 0$. The solution $I_T^* = 0$ represents the disease free equilibrium which is stable when $R_0 < 1$ and $AI_T^{*2} + BI_T^* + C = 0$ represents endemic equilibrium which is stable when $R_0 > 1$.

The number of roots for $AI_T^{*2} + BI_T^* + C = 0$ depends on the signs of A, B and C. Using the general formula, the roots that represent endemic equilibria are given by

$$I_T^* = \frac{-B \pm \sqrt{B^2 - 4AC}}{2A}. \tag{20}$$

The model system (3) has two endemic equilibria if $B^2 \geq 4AC$ and $R_0 > 1$. The endemic point $I_T^* = \frac{-B - \sqrt{B^2 - 4AC}}{2A}$ is not stable as we do not have negative infected pigs while the endemic point $I_T^* = \frac{-B + \sqrt{B^2 - 4AC}}{2A}$ is stable when $R_0 > 1$. The model system (3) undergoes forward bifurcation at $R_0 = 1$ as illustrated in Fig. 2. Therefore $R_0 < 1$ is a sufficient condition to eliminate *Taenia solium* taeniasis and cysticercosis.

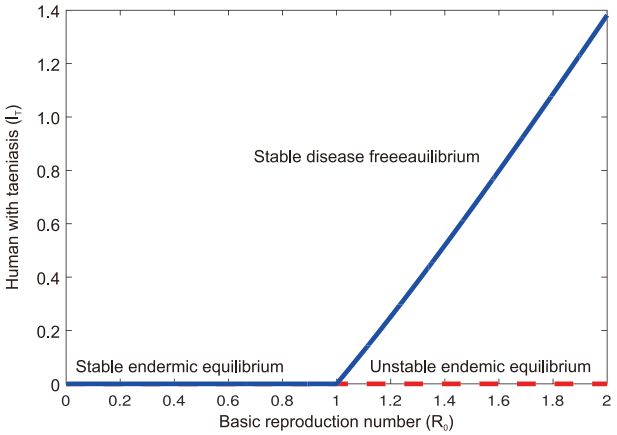

Fig. 2 Forward bifurcation

$$S_H(t), I_C(t), I_T(t), S_P(t), I_P(t), M(t), P_a(t) \in \{0, 1, 2, 3, \cdots, N\}, \quad (21)$$

where N is a positive integer that represents the maximum size of the population in the finite space (Allen, 2017).

Let

$$\{[S_H(t), I_C(t), I_T(t), S_P(t), I_P(t), M(t), P_a(t)]^T : t \in [0, \infty)\} \quad (22)$$

be the associated random vector for all discrete-valued random variables. The model is assumed to be time homogeneous and satisfies Markov chain property that the future state of the process at $t + \bigtriangleup t$ depends on the current state t, and time between events is exponentially distributed with parameter (Maliyoni, 2021; Maliyoni et al., 2017)

$$\begin{aligned}\Omega(\vec{Y}) = \Lambda_H + r\beta_C P_a S_H + \alpha_H \beta_T M S_H + \mu_H S_H + \alpha_C I_C + \mu_H I_C + \mu_H I_T + \Lambda_P \\ + c_P \beta_P P_a S_P + \alpha_P S_P + \mu_P S_P + \gamma_P I_P + \mu_P I_P + \alpha_H M + \alpha_M M + \gamma_E I_T + \mu_E P_a.\end{aligned} \quad (23)$$

Stochastic model

Disease dynamics are exposed to influences that cannot explicitly modeled (Ditlevsen and Samson, 2010). Modeling of such dynamics needs consideration of uncertainties which are inherent to diseases' transmission. CTMC stochastic models capture well uncertainties that are inherent to diseases' transmission by considering the movement of individuals from one epidemiological class to another as discrete (Lloyd et al., 2007; Maliyoni et al., 2017). In a discrete movement of individuals from one epidemiological class to another, there is a possibility of infectious individuals to die or recover before an outbreak occurs (Lahodny and Allen, 2013; Lloyd et al., 2007; Maliyoni, 2021; Maliyoni et al., 2017). This fact is not captured by deterministic models which rely on the basic reproduction number R_0 to conclude the extinction or persistence of the disease. The inability to take into account the possibility of an infectious individual to die or recover from the disease before an outbreak occurs shows that results from deterministic models need to be complemented with results from stochastic models. In this research, we develop CTMC model and apply multitype branching process to compute the probability of diseases' extinction.

Continuous Time Markov Chain (CTMC) Model

Formulation of a CTMC model uses the notations for the state variables and parameters that were used in the deterministic model (3). The time is assumed to be continuous and the random variables S_H, I_C, I_T, S_P, I_P, M, P_a are discrete with a finite space

The time interval $\bigtriangleup t$ is sufficiently small (Allen, 2017) so that the transition probabilities of the process are within the interval [0, 1]. We further assume that at most one event takes place during the time interval $\bigtriangleup t$ (Maliyoni et al., 2017). The state transition rates for the continuous time Markov chain model are defined in Table 3. The positive, negative and zero values denote increase by one, decrease by one and no change respectively.

Table 3 State transitions and rates for CTMC mode

Description	Transition	Transition rate
Recruitment of human	$(1,0,0,0,0,0,0)^T$	Λ_H
Cysticercosis infection	$(-1,1,0,0,0,0,0)^T$	$r\beta_C P_a S_H$
Taeniasis infection	$(-1,0,1,0,0,0,0)^T$	$\alpha_H \beta_T M S_H$
Death of S_H	$(-1,0,0,0,0,0,0)^T$	$\mu_H S_H$
Death of I_C due to cysticercosis	$(0,-1,0,0,0,0,0)^T$	$\alpha_C I_C$
Death of I_C	$(0,-1,0,0,0,0,0)^T$	$\mu_H I_C$
Death of I_T	$(0,0,-1,0,0,0,0)^T$	$\mu_H I_T$
Recruitment of pigs	$(0,0,0,1,0,0,0)^T$	Λ_P
Infection of pigs	$(0,0,0,-1,1,0,0)^T$	$c_P \beta_P P_a S_P$
Harvesting of S_P	$(0,0,0,-1,0,0,0)^T$	$\alpha_P S_P$
Death of S_P	$(0,0,0,-1,0,0,0)^T$	$\mu_P S_P$
Slaughter of I_P	$(0,0,0,0,-1,1,0)^T$	$\gamma_P I_P$
Death of I_P	$(0,0,0,0,-1,0,0)^T$	$\mu_P I_P$
Eating of M by humans	$(0,0,0,0,0,-1,0)^T$	$\alpha_H M$
Throwing of M	$(0,0,0,0,0,-1,0)^T$	$\alpha_M M$
Release of P_a	$(0,0,0,0,0,0,1)^T$	$\gamma_E I_T$
Death of P_a	$(0,0,0,0,0,0,-1)^T$	$\mu_E P_a$

Multitype branching process and stochastic threshold

The multitype branching process is used to approximate non-linear CTMC model near the disease free equilibrium (Maliyoni et al., 2017). The theory of the multitype branching process is applied to infected classes I_C, I_T, I_P, M and P_a. S_H and S_P are assumed to be at disease free equilibrium i.e $S_H(0) \approx N_H(0)$ and $S_P(0) \approx N_P(0)$, where $N_H(0)$ and $N_P(0)$ are initial populations for humans and pigs respectively.

The infection of susceptible humans or pigs is referred to as a birth of an offspring and we say that, the infectious individual of type i, I_i has given birth to an infectious individual of type j, I_j. The number of offspring produced by an individual of type i is independent of the number of offspring produced by individuals of type j, $j \neq i$, and infectious individuals of type i have the same offspring probability generating function (pgf). With linearity of multitype branching process near disease free equilibrium and time-homogeneous with independent births and deaths, we can define offspring pgf for birth and death of the infectious humans and pigs, and use them to compute probability of disease extinction or major outbreak.

Let X_{ji} denote the offspring random variable of type j produced by infectious individuals of type i, then the offspring pgf associated with birth of infectious individuals of type i or death of infectious individuals I_j provided the process begins with one infectious individual of type j, $I_j = 1$, $I_i = 0$ for $i \neq j$ (Dorman et al., 2004; Pénisson, 2010) is;

$$g_i(x_1, x_2, \cdots, x_n) = \sum_{r_n=0}^{\infty} \cdots \sum_{r_1=0}^{\infty} P_i(r_1, \cdot, r_n) x_1^{r_1} \cdots x_n^{r_n}, \tag{24}$$

where

$$P_i(r_1, \cdot, r_n) = Prob\{X_{1j} = r_1, \cdots, X_{nj} = r_n\}$$

is the probability that one individual of type i gives birth to individual r_j. Using Eq. (24), we have $n \times n$ non negative expectation matrix $\mathbf{M} = [m_{ji}]$, where m_{ji} is the expected number of infectious offspring of type j generated by infectious individual of type i (Maliyoni et al., 2017) defined by

$$m_{ji} = \left.\frac{\partial f_i}{\partial x_j}\right|_{x=1} < \infty. \tag{25}$$

The probability of disease extinction or outbreak can be determined by finding the size of the spectral radius $\rho(\mathbf{M})$ of the matrix $\mathbf{M}$. When $\rho(\mathbf{M}) < 1$ then the probability of ultimate disease extinction is one, that is

$$\lim_{t \to \infty} \text{Prob}\{\mathbf{I}(t) = 0\} = 1. \tag{26}$$

We apply multitype branching process to define the offspring probability generating functions for all infected classes. If there is one human with *Taenia solium* cysticercosis $I_C(0) = 1$ and there are no infectives in other clasess $(I_T(0) = 0, I_P(0) = 0, M(0) = 0, P_a(0) = 0)$, then there is no chance for the diseases to spread. Therefore, the initially infected human can only die of *Taenia solium* cysticercosis or die naturally. Thus the offspring probability generating function is

$$f_1(u_1, u_2, u_3, u_4, u_5) = 1. \tag{27}$$

If there is one human with *Taenia solium* taeniasis $I_T(0) = 1$ and there are no infectives for other clasess $(I_C(0) = 0, I_P(0) = 0, M(0) = 0, P_a(0) = 0)$ then the offspring probability generating function is given by

$$f_2(u_1, u_2, u_3, u_4, u_5) = \frac{\gamma_E u_2 u_5 + \mu_H}{\gamma_E + \mu_H}. \tag{28}$$

The term $\gamma_E/(\gamma_E + \mu_H)$ is the probability that the initial human that is infected with *Taenia solium* taeniasis sheds *Taenia solium* eggs in the environment at a rate γ_E and does not die thus resulting into one human that is infected with *Taenia solium* taeniasis and one *Taenia solium* egg in the environment whereas the term $\mu_H/(\gamma_E + \mu_H)$ is the probability that the initial human that is infected with *Taenia solium* taeniasis dies before shedding *Taenia solium* eggs in the environment thus resulting into zero human with *Taenia solium* taeniasis and zero *Taenia solium* eggs in the environment.

When there is one infected pig $I_P(0) = 1$ and there are no infectives for other clasess $(I_C(0) = 0, I_T(0) = 0, M(0) = 0, P_a(0) = 0)$ then the offspring probability generating function is

$$f_3(u_1, u_2, u_3, u_4, u_5) = \frac{\gamma_P u_4 + \mu_P}{\gamma_P + \mu_P}. \tag{29}$$

The term $\gamma_P/(\gamma_P + \mu_P)$ defines the probability that the initial infected pig is slaughtered resulting into no infected pig and the presence of infectious pork whereas $\mu_P/(\gamma_P + \mu_P)$ is the probability that, the initial pig with *Taenia solium* cysticercosis dies before it is slaughtered thus resulting into zero infected pig and infectious pork.

If there is an infectious pork $M(0) = 1$ and there are no infectives for other classes $(I_C(0) = 0, I_T(0) = 0, I_P(0) = 0, P_a(0) = 0)$ then the offspring probability generating function is

$$f_4(u_1, u_2, u_3, u_4, u_5) = \frac{\alpha_H \beta_T S_H^0 u_2 u_4 + \alpha_m}{\alpha_H \beta_T S_H^0 + \alpha_m}. \tag{30}$$

The term $\alpha_H\beta_T/(\alpha_H\beta_T+\alpha_m)$ is the probability that a proportion of infectious pork is consumed by human resulting into one human with *Taenia solium* taeniasis and the presence of infectious pork whereas the term $\alpha_m/(\alpha_H\beta_T+\alpha_m)$ is the probability that the initial infectious pork decays resulting into no infectious pork and human with *Taenia solium* taeniasis.

If initially there is one *Taenia solium* egg in the environment $P_a(0)=1$ and there are no infectives for other clasess $(I_C(0)=0, I_T(0)=0, I_P(0)=0, M(0)=0)$ then the offspring probability generating function is

$$f_5(u_1,u_2,u_3,u_4,u_5)=\frac{r\beta_C S_H^0 u_1u_5+c_P\beta_P S_P^0 u_3u_5+\mu_E}{r\beta_C S_H^0+c_P\beta_P S_P^0+\mu_E}. \quad (31)$$

The term $r\beta_C/(r\beta_C+c_P\beta_P+\mu_E)$ is the probability that the initial *Taenia solium* egg from the environment infect a susceptible human resulting into one human with *Taenia solium* cysticercosis and *Taenia solium* egg. The term $c_P\beta_P/(r\beta_C+c_P\beta_P+\mu_E)$ is the probability that the initial *Taenia solium* egg from the environment infect a susceptible pig resulting into one infected pig and *Taenia solium* egg whereas $\mu_E/(r\beta_C+c_P\beta_P+\mu_E)$ is the probability that the initial *Taenia solium* egg dies before it infects a human or pig resulting into zero infected human and pig.

The expectation matrix $\mathbb{M}$ is given by

$$\mathbb{M}=\begin{pmatrix} \frac{\gamma_E}{\gamma_E+\mu_H} & 0 & \frac{\alpha_H\beta_T S_H^0}{\alpha_H\beta_T S_H^0+\alpha_m} & 0 \\ 0 & 0 & 0 & \frac{c_P\beta_P S_P^0}{r\beta_C S_H^0+c_P\beta_P S_P^0+\mu_E} \\ 0 & \frac{\gamma_P}{\gamma_P+\mu_P} & 0 & 0 \\ \frac{\gamma_E}{\gamma_E+\mu_H} & 0 & 0 & 0 \end{pmatrix}. \quad (32)$$

The stochastic threshold is given by $\rho(\mathbf{M})$. The disease goes to entinction if $\rho(\mathbf{M})<1$. This fact is related to the basic reproduction R_0 that when $R_0<1$ the disease dies out in the population. While in deterministic models the disease persists when $R_0>1$, in CTMC stochastic models there are chances for the disease to die or persist even if $\rho(\mathbf{M})>1$ depending on the initial number of infectives in the population. Therefore if $\rho(\mathbf{M})>1$, there exists a fixed point $(q_1,q_2,q_3,q_4,q_5)\in(0,1)^5$ of the offspring generating functions (27)-(31) which is used to calculate the likelihood of disease extinction. The fixed point is obtained by setting $f_i(q_1,q_2,q_3,q_4,q_5)=q_i$ for $i=1,2,3,4,5$. That is:

$$\begin{aligned} q_1&=1,\\ q_2&=\frac{\mu_H}{(1-q_5)\gamma_E+\mu_H},\\ q_3&=\frac{\gamma_P q_4+\mu_P}{\gamma_P+\mu_P},\\ q_4&=\frac{((1-q_5)\gamma_E+\mu_H)\alpha_m\mu_H}{(\alpha_H\Lambda_H\beta_T+\alpha_m\mu_H)(1-q_5)\gamma_E+\alpha_m\mu_H^2},\\ q_5&=\frac{\mu_H\alpha_m(\gamma_E+\mu_H)+\gamma_E\alpha_H\Lambda_H\beta_T}{\mu_H^2(\alpha_m+\alpha_H)R_0^2+\gamma_E(\mu_H\alpha_m+\alpha_H\Lambda_H\beta_T)}. \end{aligned}$$

Thus, the likelihood of disease outbreak for type i infectives is

$$1-\mathbf{P_0}=1-q_1^{i1}q_2^{i2}q_3^{i3}q_4^{i4}q_5^{i5}, \quad (33)$$

where in for $n=1,2,3,4,5$ are the initial values for human with cysticercosis, human with taeniasis, infected pigs, infectious pork and *Taenia solium* eggs that were exposed in a susceptible population respectively.

Numerical Analysis

In this section, we study the dynamics of *Taenia solium* taeniasis and cysticercosis using parameter values in Table 1. Most of the parameter values are assumed due to the fact that, *Taenia solium* taeniasis and cysticercosis are neglected diseases which are not much studied and therefore, have no recorded data. We simulate the model system (3) and compare the results with continuous time Markov chain stochastic model.

The effects of *Taenia solium* taeniasis and cysticercosis in humans and pigs appear after five years. Susceptible humans decrease as they contract *Taenia solium* taeniasis and cysticercosis whereas humans with *Taenia solium* taeniasis and cysticercosis increase as shown in Fig. 3A. The steady state is attained after 20 years. The same trend is observed in pig population. The effects of *Taenia solium* cysticercosis in human and pig populations are observed in the first 20 years where steady state is attained as illustrated in Fig. 3B. The number of *Taenia solium* eggs in the environment increases when humans with *Taenia solium* taeniasis defecate in the open spaces while infectious pork increases when infected pigs are slaughtered as in Fig. 4.

Using 10000 sample paths, we concurrently simulate the deterministic and continuous time Markov chain models to compare their results. Susceptible humans and pigs are assumed to be at a disease free equilibrium.

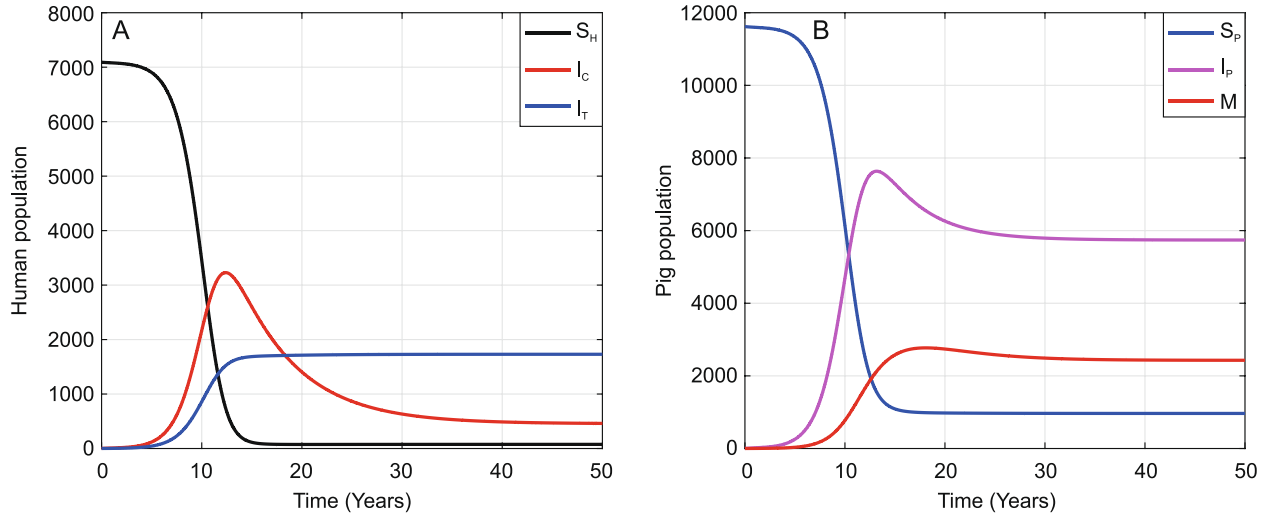

Fig. 3 Dynamics of *Taenia solium* taeniasis and cysticercosis in human and pig population. **A**. Human population; **B**. Pig population

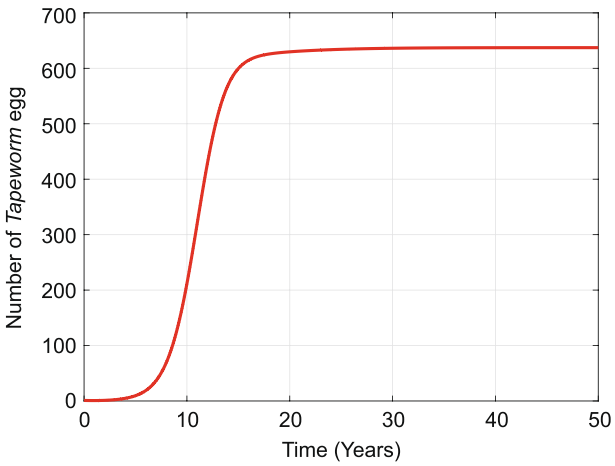

Fig. 4 Variation of *Taenia solium* egg number with time

Figures 4, 5 and 6 show that deterministic and stochastic results do not deviate significantly from each other and the number of *Taenia solium* taeniasis and cysticercosis cases are at the peak in the first 20 and 15 years respectively. From 15 years, susceptible humans and pigs decrease rapidly as depicted in Fig. 7. Generally, the results show that as *Taenia solium* taeniasis and cysticercosis invade human population, endemic level is attained in the first twenty years and stabilize thereafter. The solutions for both deterministic and CTMC stochastic models predict persistence of *Taenia solium* taeniasis and cysticercosis as illustrated in Figs. 4, 5 and 6. As long as there are infected pigs and *Taenia solium* eggs in the environment, *Taenia solium* taeniasis and cysticercosis will persist if interventions are not administered. To eradicate the diseases, efforts should focus on treating infected humans and pigs, pork inspection and improving hygiene and sanitation.

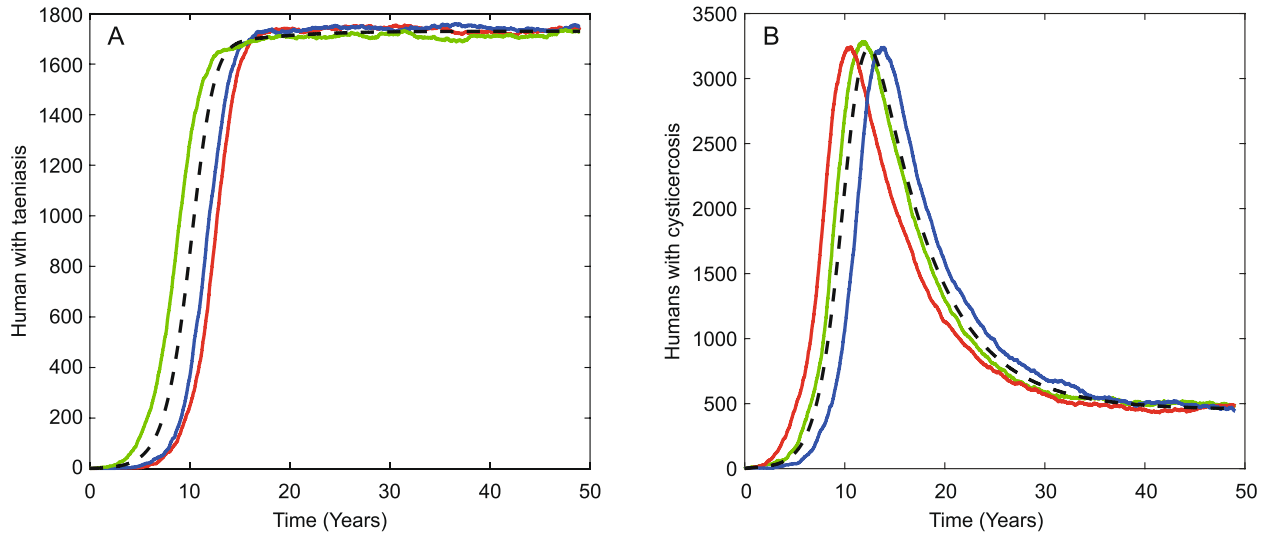

Fig. 5 Taeniasis and cysticercosis in human. **A**. Human with taeniasis; **B**. Human with cysticercosis

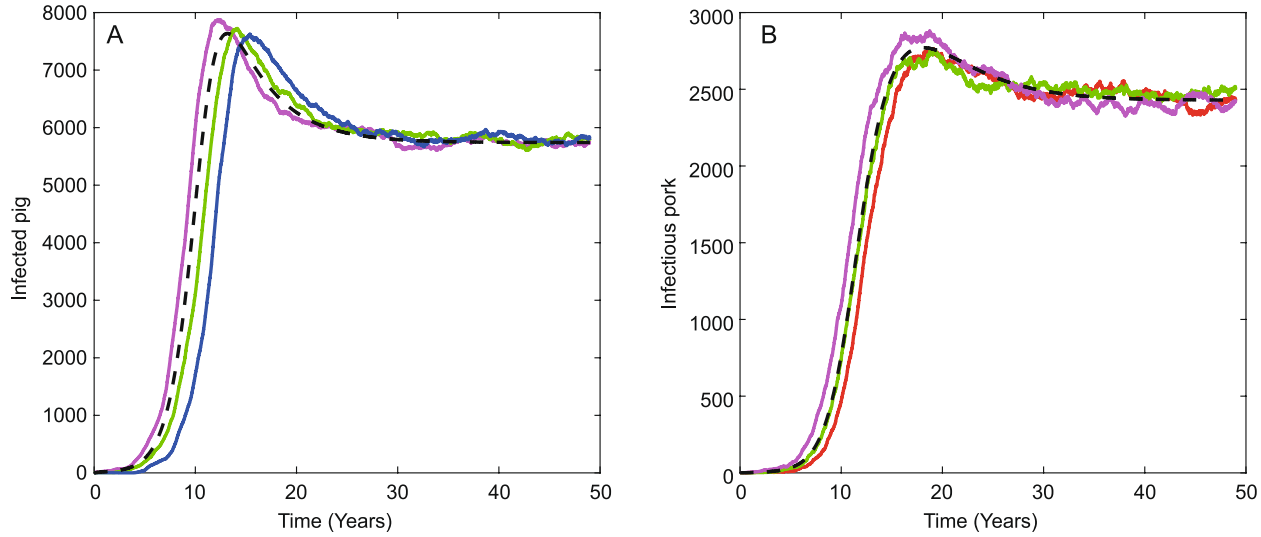

Fig. 6 Variation of infected pig and infectious pork over time. **A**. Infected pig; **B**. Infectious pork

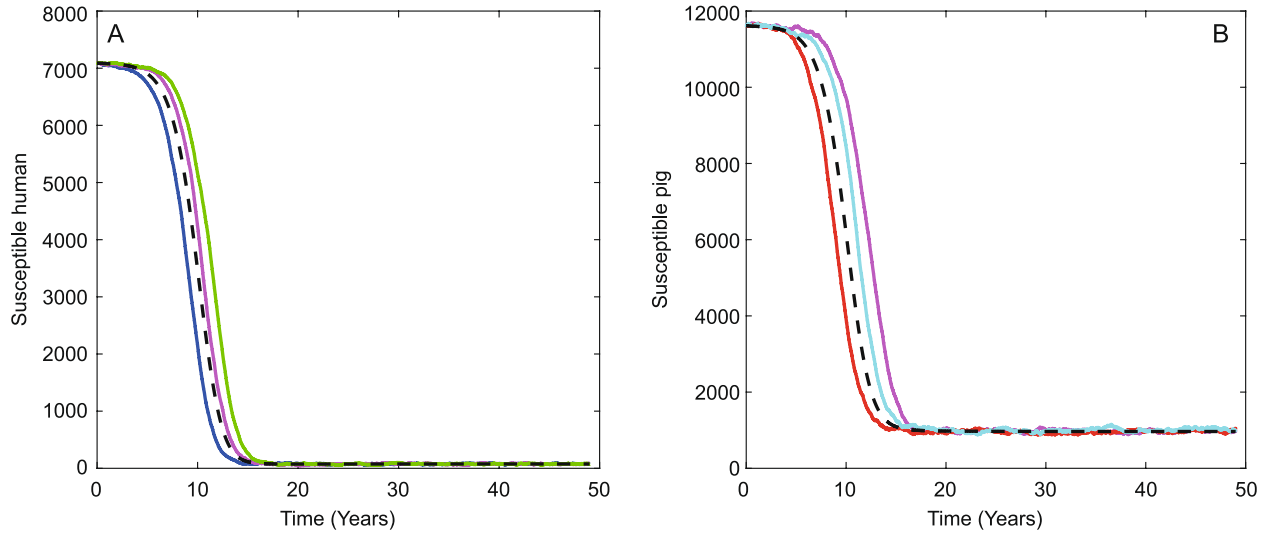

Fig. 7 Variation of susceptible human and pig. **A**. Human; **B**. Pig

Probability of *Taenia solium* taeniasis and cysticercosis Extinction or Outbreak

The fixed point of multitype branching process is applied to compute the probability for *Taenia solium* taeniasis and cysticercosis extinction $\mathbf{P_0}$ when initial conditions are varied. Using a proportion of 10000 sample paths for CTMC stochastic model, we compute numerical probability for *Taenia solium* taeniasis and cysticercosis extinction $\mathbf{P_a}$ and compare $\mathbf{P_0}$ and $\mathbf{P_a}$ results. The results from Table 4 show that $\mathbf{P_a}$ is a good estimate of $\mathbf{P_0}$. The fixed point due to the offspring probability generating function in $(0, 1)^5$ is $(q_1, q_2, q_3, q_4, q_5) = (1, 0.0420, 0.5729, 0.2507, 0.0814)$. This reveals that, the probability for *Taenia solium* taeniasis and cysticercosis extinction is high if the diseases emerge from humans with *Taenia solium* cysticercosis or infected pigs. However, there is major outbreak for *Taenia solium* taeniasis and cysticercosis if the diseases emerge from either humans with taeniasis or infectious pork or *Taenia solium* eggs in the environment as shown in Fig. 8.

Conclusion

Taenia Solium taeniasis and cysticercosis are neglected diseases which affect poor communities in developing countries. Deterministic and CTMC stochastic models were formulated to study the dynamics of the diseases. The next generation matrix approach was used to compute the basic reproduction number R_0 and forward normalized sensitivity index is applied in deriving sensitivity indices to

Table 4 Probability of disease extinction

y_1	y_2	y_3	y_4	y_5	P_a	P_0
1	0	0	0	0	0.9999	1.0000
0	1	0	0	0	0.0412	0.0420
0	0	1	0	0	0.5729	0.5729
0	0	0	1	0	0.2554	0.2507
0	0	0	0	1	0.0814	0.0814
1	1	0	0	0	0.0416	0.0420
1	0	1	0	0	0.5716	0.5729
0	1	0	1	1	0.0008	0.0009
0	1	1	1	1	0.0005	0.0005
1	1	1	1	1	0.0005	0.0005

endemic equilibrium is globally asymptotically stable when $R_0 > 1$. Sensitivity results show that humans with taeniasis, infectious pork and *Taenia solium* eggs in the environment play an importan role in the persistence of *Taenia solium* taeniasis and cysticercosis. However, the natural mortality for pigs and *Taenia solium* eggs in the environment, and the rate at which infectious pork decays decrease the average new infections for *Taenia solium* taeniasis and cysticercosis.

The results for CTMC stochstic model reveal that there is a high likelihood of *Taenia solium* taeniasis and cysticercosis extinction if they emerge from either humans with *Taenia solium* cysticercosis or infected pigs. However, there is a major outbreak if the diseases emerge from humans with *Taenia solium* taeniasis or infectious

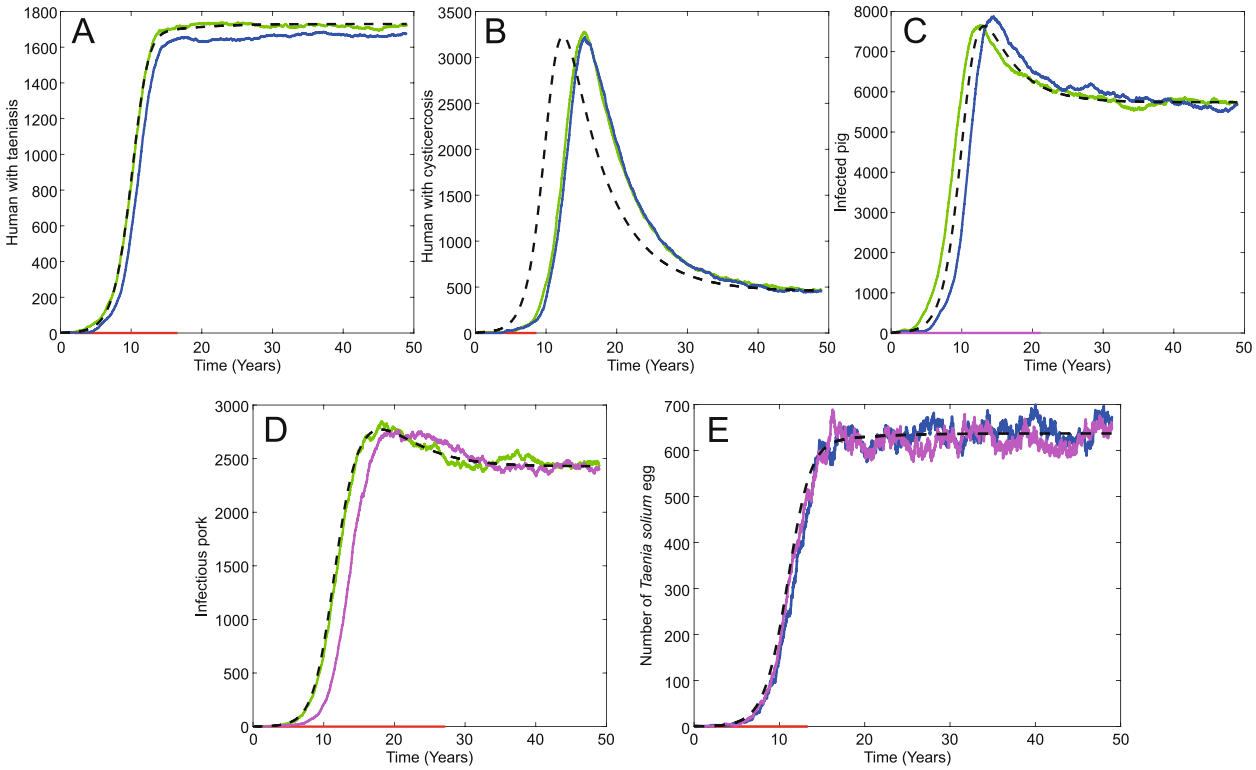

Fig. 8 Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for infected human, infected pig and infectious pork. **A**. Human with taeniasis; **B**. Human with cysticercosis; **C**. Infected pig; **D**. Infectious pork; **E**. Number of *Taenia solium* eggs

determine parameters that drive the diseases. The multitype branching process was applied to a CTMC stochastic model to determine the probability of diseases' extinction.

The analysis shows that the model undergoes forward bifurcation at $R_0 = 1$, implying that $R_0 < 1$ is a sufficient condition to eliminate *Taenia Solium* taeniasis and cysticercosis. Thus the disease free equilibrium is globally asymptotically stable when $R_0 < 1$ and pork or *Taenia solium* eggs in the environment. Simulation shows that the results for deterministic and CTMC stochastic models do not deviate from each other. Both models show that, humans with *Taenia solium* taeniasis, infectious pork and *Taenia solium* eggs in the environment play an important role in the persistence of *Taenia solium* taeniasis and cysticercosis. To effectively control *Taenia solium* taeniasis and cysticercosis, intervention

strategies should focus on treatment of infected humans and pigs, improving hygiene and sanitation, conduct pork inspection and spray of insecticides for killing *Taenia solium* eggs in the environment.

Abbreviations

CTMC	Continuous Time Markov Chain
pgf	probability generation function

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Authors' contributions
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Availability of data and materials
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Declarations

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Consent for publication
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