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Cysticercosis and Taeniasis in Humans, Pigs and Cattle: A Potential Extinction or Outbreak

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Abstract

Taeniasis and cysticercosis pose a health concern on both humans and animals, as well as the economy of livestock farmers in rural areas. This study examines cysticercosis and taeniasis transmission dynamics in human, pig and cattle populations. Both deterministic and continuous time Markov chain (CTMC) stochastic approaches are used. For deterministic and CTMC stochastic models, we used the next generation approach and the multitype branching process respectively to calculate the basic reproduction number and the stochastic threshold. The potential probability of cysticercosis and taeniasis extinction is computed through numerical simulations for the CTMC model using 10,000 sample paths and altering the initial values for classes that are infected. The findings demonstrate that when diseases' outbreak occur, the CTMC stochastic model's solutions resemble those of deterministic model quite closely. The findings also suggest that the likelihood of diseases' extinction is high if they develop from a small number of taenia eggs. If the infections, however, emerge from humans with cysticercosis, they will perish. If the infections arise from either infected beef and pork or humans with taeniasis, there is a significant diseases' outbreak in the human, pig and cattle populations. Therefore, at the beginning of a diseases' outbreak, management strategies that concentrate on reducing taeniasis-infected individuals and consumption of infectious beef and pork can help in regulating the transmission of the diseases in humans, pigs, and cattle.

Keywords: Taeniasis, Cysticercosis, Stochastic Threshold, Markov Chain, Multitype Branching Process, Basic Reproduction Number.

Introduction

Taeniasis is the intestinal infection caused by the adult tapeworms. Humans contract taeniasis when they eat inadequately cooked or raw pork or beef infected with *Taenia solium* or *Taenia saginata* tapeworm larval cysts. Cysticercosis refers to the infection of tissues or organs of humans or animals by larval form tapeworms (Symeonidou et al. 2018). These diseases are mostly found in rural areas where cattle and pigs are kept in a free ranging system (Flisser et al. 2006, WHO 2005). In the dynamics of taeniasis and cysticercosis, humans are the definitive hosts. Tapeworm eggs which contaminate water sources, fodder and pastures are released when individuals with taeniasis defecate in the fields (Dermauw et al. 2018). When cattle consume *T. saginata* tapeworms from the environment, they get cysticercosis. Pigs get cysticercosis when they feed on human feaces or *ingest T. solium* eggs from the environment (Symeonidou et al. 2018). Cysticercosis is acquired by humans when they consume *T. solium* eggs via drinking contaminated water, consumption of fruits and vegetables (Brutto 2013). When *T. solium* eggs are eaten, they hatch and progress into larvae that infiltrate the intestines and move into body tissues and organs, forming cysts (WHO 2005).

Whenever larval cysts invade the brain, an individual acquires neurocysticercosis that causes epilepsy worldwide (Mwasunda et al. 2021a).

Cysticercosis and taeniasis are globally distributed. However, the diseases are more endemic in Africa, Asia and Latin America (Symeonidou et al. 2018). These diseases affect human health and the livelihood of farmers in rural areas. Cysticercosis reduces market value for cattle and pigs by making beef and pork unsafe for human consumption (WHO 2005, Winskill et al. 2017). Worldwide, taeniasis and cysticercosis affect nearly 50 million people and approximately 50,000 human cysticercosis induced deaths occur every year (Aung and Spelman 2016). The World Health Organization has identified cysticercosis to be the main tropical neglected disease and is the major source of death due to food borne diseases (Mwasunda et al. 2021a). In Tanzania, porcine cysticercosis has been reported in northern, central and southern regions with the prevalence rates of 5.5-16.9%, 14.9% and 0.3-17.4% respectively (Trevisan et al. 2017). Human taeniasis and cysticercosis have also been reported in Tanzania (Kavishe et al. 2017, Mwanjali et al. 2013). For instance, in 2012 there were 17,853 epileptic cases and 212 deaths in the country. In the same year there were 183,927 porcine cysticercosis cases and cysticercosis economic burden was approximately US$ 7.9 million (Trevisan et al. 2017).

Mathematical modelling plays a significant role in understanding infectious disease transmission dynamics. Currently, few deterministic and statistical models have been formulated and rigously analyzed to study the dynamics of taeniasis and cysticercosis in human and pigs. These studies include Gonzalez et al. (2002), Braae et al. (2016), Kyvsgaard et al. (2007), Jose et al. (2018) Sanchez Torres et al. (2019), Winskill et al. (2017) and Mwasunda et al. (2021a, 2021b). Recently, the study by Mwasunda et al. (2022) has focused on studying the dynamics of *T. saginata* taeniasis and cysticercosis through deterministic and continuous time Markov chain (CTMC) stochastic models. However, the study did not capture the transmission dynamics of taeniasis and cysticercosis due to *T. solium* tapeworm. In this study, we formulate and analyze deterministic and continuous time Markov chain (CTMC) stochastic models for cysticercosis and taeniasis dynamics in pigs, cattle and humans due to *T. saginata* and *T. solium* tapeworms.

Deterministic Mathematical Model

Formulation of the Model

We take into account Mwasunda et al. (2021a) basic model for transmission dynamics of cysticercosis and taeniasis. Humans are divided into three groups: those who are susceptible to infection S_H, those with taeniasis I_{HT} and those who have cysticercosis I_{HC}. Pigs are classified into susceptible S_P and infected I_P pigs whereas cattle are grouped into S_C and I_C classes that represent susceptible and infected cattle respectively. The classes B_I and P_I stand for infectious beef and pork, respectively, while E_T denotes number of taenia eggs in the environment.

The recruitment of susceptible humans is considered to occur through birth at per capita rate ψ and decline when they consume inadequately cooked or raw infectious beef or pork at rates α_p and α_b respectively. Susceptible humans also decrease through ingestion of *T. solium* eggs at a rate θ. Cysticercosis infected humans replenish at a rate θ when susceptible humans consume *T. solium* eggs and decrease at a rate μ_d due to disease induced death. Taeniasis infected individuals grow at rates α_p and α_b as a consequence of susceptible humans to ingest pork and beef infected with tapeworm larval cysts respectively. The parameter β_T denotes the probability of susceptible humans to acquire taeniasis from raw or undercooked infected beef and pork. Human natural death is considered to occur at a rate μ_h. When individuals with taeniasis defecate in open spaces, the number of taenia eggs in the environment increases at a rate ν and diminishes at a rate μ_e as the eggs die naturally.

The per capita rates of susceptible cattle and pigs increase through birth at rates ϕ and Λ, respectively, and decline when they eat taenia eggs from the contaminated

environment at rates γ_C and γ_P, respectively. When slaughtered for consumption, susceptible cattle and pigs both decline at rates σ and ρ, respectively. Infected cattle and pigs grow at rates γ_C and γ_P respectively when susceptible cattle and pigs feed on the contaminated environment. The natural death is assumed to occur at the rates μ_C and μ_P, respectively, for all classes of cattle and pigs. When infected pigs and cattle are slaughtered for consumption, infectious beef and pork increase at the rates ω and η, respectively. The amount of infectious pork and beef is quantified in terms of number of infected cattle and pigs that are slaughtered for consumption. Humans who are susceptible to infection are considered to eat infectious beef and pork at rates of α_b and α_P, respectively. The proportions of infectious beef and pig that are not ingested by susceptible humans are represented by the parameters ϵ and δ respectively.

To formulate the basic model that governs cysticercosis and taeniasis in human, pig and cattle populations, we consider the following assumptions: Both cattle and pigs are kept in free range farming system, and we do not consider migration; Humans can be infected by either cysticercosis or taeniasis; pigs and cattle do not die due to cysticercosis infection; contact rates of pigs, humans and cattle with taenia eggs are density dependent; consumption rates of raw or inadequately cooked infected pork or beef are proportion to the quantity of pork or beef available. The basic model for cysticercosis and taeniasis transmission dynamics in human, pig and cattle populations is summarized in Figure 1 and model parameters are described in Table 1.

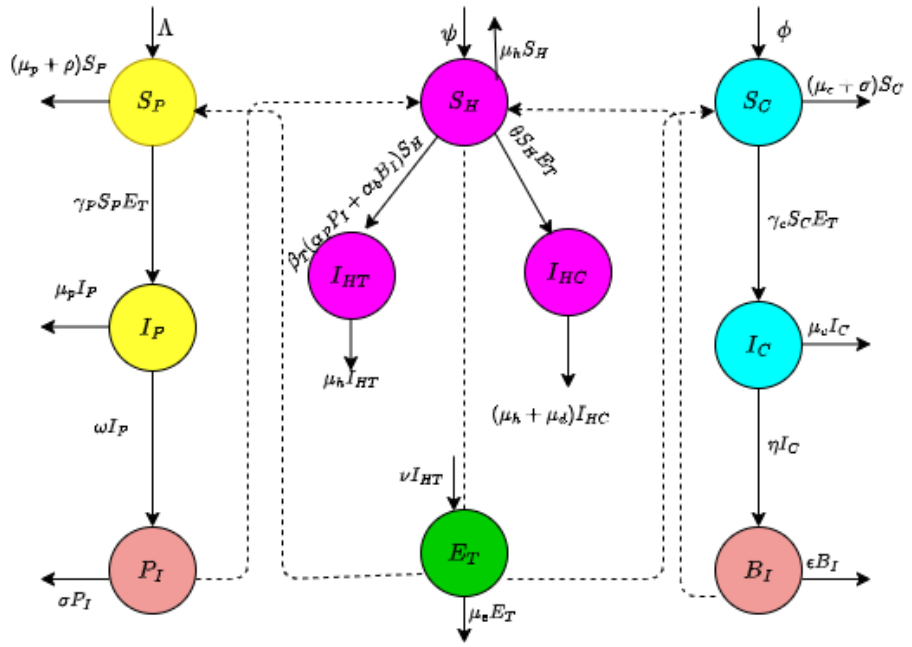

Figure 1: The Model Flow Diagram for Transmission Dynamics of Taeniasis and Cysticercosis in Human, Pig and Cattle Populations

The basic model for cysticercosis and taeniasis transmission dynamics is given by the system:

$$\begin{aligned}
\frac{dS_H}{dt} &= \psi - \beta_T(\alpha_b B_I + \alpha_P P_I)S_H - \theta E_T S_H - \mu_h S_H,\\
\frac{dI_{HT}}{dt} &= \beta_T(\alpha_b B_I + \alpha_P P_I)S_H - \mu_h I_{HT},\\
\frac{dI_{HC}}{dt} &= \theta E_T S_H - (\mu_d + \mu_h)I_{HC},\\
\frac{dS_P}{dt} &= \Lambda - \gamma_P E_T S_P - (\mu_P + \rho)S_P,\\
\frac{dI_P}{dt} &= \gamma_P E_T S_P - (\mu_P + \omega)I_P,\\
\frac{dP_I}{dt} &= \omega I_P - (\alpha_P + \delta)P_I,\\
\frac{dS_C}{dt} &= \phi - \gamma_C E_T S_C - (\mu_C + \sigma)S_C,\\
\frac{dI_C}{dt} &= \gamma_C E_T S_C - (\mu_C + \eta)I_C,\\
\frac{dB_I}{dt} &= \eta I_C - (\alpha_b + \epsilon)B_I,\\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T,
\end{aligned} \tag{1}$$

subject to initial conditions: $S_H(0) > 0; I_{HC}(0) \geq 0; I_{HT}(0) \geq 0; S_P(0) > 0; I_P(0) \geq 0;$ $S_C(0) > 0; I_C(0) \geq 0; B_I(0) \geq 0; P_I(0) \geq 0; E_T(0) \geq 0.$

Table 1: Model parameters' description (unit: per year)

Parameter	Description	Value	Source
ψ	Per capita human recruitment rate	300	Mwasunda et al. 2021a
μ_d	Human cysticercosis induced death rate	0.0141	Wang et al. 2013
μ_h	Per capita human natural death rate	0.0925	Wang et al. 2013
Λ	Per capita pig recruitment rate	150	Mwasunda et al. 2021a
α_P	Consumption rate of infectious pork	0.012	Mwasunda et al. 2021a
β_T	Probability of human to acquire taeniasis	0.093	Mwasunda et al. 2021a
γ_P	Pig infection rate with *T. Solium* eggs	0.01	Kyvsgaard et al. 2007
ω	Rate of slaughtering infected pigs	0.332	Kyvsgaard et al. 2007
ρ	Slaughtering rate of susceptible pigs	0.0252	Mwasunda et al. 2021a
μ_P	Per capita pig natural death rate	0.996	Winskill et al. 2017
δ	Decaying rate of infectious pork	0.358	Mwasunda et al. 2021a
ϕ	Per capita cattle recruitment rate	120	Mwasunda et al. 2021a
α_b	Consumption rate of infectious beef	0.023	Mwasunda et al. 2021a

γ_C	Cattle infection rate by T. saginata eggs	0.00625	Mwasunda et al. 2021a
σ	Slaughter rate of susceptible cattle	0.213	Mwasunda et al. 2021a
η	Rate of slaughtering infected cattle	0.0235	Mwasunda et al. 2021a
μ_C	Per capita cattle natural death rate	0.33	Kyvsgaard et al. 2007
θ	Human infection rate with cysticercosis	0.00523	Mwasunda et al. 2021a
ϵ	Decaying rate of infectious beef	0.225	Mwasunda et al. 2021a
ν	The rate of defecation by humans with taeniasis	0.150	Mwasunda et al. 2021a
μ_e	Per capita natural death rate for taenia eggs	10.42	Wang et al. 2013

Positivity and Boundedness of Solutions

To show whether the model solutions are well-posed, we need to show that the solutions are positive and bounded. In this section, we demonstrate that the solutions to model system model system (1) are bounded and positive.

Positivity of Model Solutions

Beginning with the equation for susceptible humans in the model system (1), we have:

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

Similarly, it can be shown that:

$I_{HT} \geq 0;\ I_{HC} \geq 0;\ S_P \geq 0;\ I_P \geq 0;\ P_I \geq 0;\ S_C \geq 0; I_C \geq 0;\ B_I \geq 0;\ E_T \geq 0, \forall t \geq 0.$

Therefore, all model solutions are non-negative for all t > 0.

Boundedness of Model Solutions

To demonstrate the boundedness of the model system (1), consider $H = S_H + I_{HT} + I_{HC}$, $P = S_P + I_P$, and $C = S_C + I_C$, respectively, representing the entire populations of humans, pigs, and cattle. After adding up all equations for human population in model system (1), we get:

$$\frac{dH}{dt} = \psi - \mu_d I_{HC} - \mu_h H, \tag{2}$$

$$\frac{dH}{dt} + \mu_h H \leq \psi.$$

Application of initial condition after integration gives:

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

where $H(0)$ is the initial total human population. In similar manner, it can be proved that pig and cattle populations are given by:

$$P(t) \leq \frac{\Lambda}{\mu_P} + \left(P(0) - \frac{\Lambda}{\mu_P}\right) e^{-\mu_P t} \quad \text{and} \quad C(t) \leq \frac{\phi}{\mu_C} + \left(C(0) - \frac{\phi}{\mu_C}\right) e^{-\mu_C t}, \tag{4}$$

respectively, where $P(0)$ and $C(0)$ are total initial populations for pigs and cattle respectively. For each population, two cases are considered to analyze (3) and (4):

$H(0) > \frac{\psi}{\mu_h}$, $(0) > \frac{\Lambda}{\mu_P}$, $C(0) > \frac{\phi}{\mu_C}$ and when $H(0) < \frac{\psi}{\mu_h}$, $P(0) < \frac{\Lambda}{\mu_P}$, $C(0) < \frac{\phi}{\mu_C}$.

For the two cases we obtain;

$$\begin{aligned} H(t) &\leq \Phi_t = max\left\{\frac{\psi}{\mu_h}, H(0)\right\}, \\ P(t) &\leq max\left\{\frac{\Lambda}{\mu_P}, P(0)\right\} \\ C(t) &\leq max\left\{\frac{\phi}{\mu_C}, C(0)\right\}. \end{aligned} \tag{5}$$

Since $H = S_H + I_{HT} + I_{HC} \leq \Phi_t$, then $I_{HT} \leq \Phi_t$. Taking into account the equation for taenia eggs in model system (1), we have:

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

Integration and application of initial condition gives:

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

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

Similarly, considering the equations for infected pork and beef, we obtain:

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

Showing that the model solutions go and do not leave the region:

$$\Omega = \left\{ \begin{matrix} (S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I) \in \mathbb{R}_+^{10} : 0 \leq H(t) \leq \Phi_t; 0 \leq P(t) \leq \Pi_t; \\ 0 \leq C(t) \leq \psi_t; 0 \leq E_T(t) \leq \Gamma_t; 0 \leq P_I(t) \leq \Theta_t; 0 \leq B_I(t) \leq \xi_t \end{matrix} \right\}.$$

Therefore, the solutions of the model system (1) are positive invariant throughout the region Ω. Solutions that begin at the boundary of Ω move into the region in infinite time. These results are summarized in Theorem 1.

Theorem 1: *The model solutions of the system (1) are positive invariant in the region Ω.*

Model Equilibria and Reproduction Number R_0

When there is no taeniasis and cysticercosis in pigs, cattle and humans we get the disease-free state E^0 given by:

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

The basic reproduction number R_0 is the expected number of new infections that could arise from the introduction of one infected individual in a fully susceptible population (Diekmann et al. 1990). The disease disappears in a population when $R_0 < 1$ and survives when $R_0 > 1$. We adopt the next generation matrix method to derive reproduction number R_0 (van den Driessche and Watmough 2002). Let the vectors for new infections F_i and transfer terms V_i be given by:

$$F_i = \begin{pmatrix} \beta_T(\alpha_b B_I + \alpha_P P_I)S_H \\ \theta E_T S_H \\ \gamma_P E_T S_P \\ 0 \\ \gamma_C E_T S_C \\ 0 \\ 0 \end{pmatrix}, V_i = \begin{pmatrix} \mu_h I_{HT} \\ (\mu_d + \mu_h)I_{HC} \\ (\omega + \mu_P)I_P \\ -\omega I_P + (\alpha_P + \delta)P_I \\ (\eta + \mu_C)I_C \\ -\eta I_C + (\alpha_b + \epsilon)B_I \\ -\nu I_{HT} + \mu_e E_T \end{pmatrix}. \quad (8)$$

We define matrices F and V by:

$$F = \frac{\partial F_i}{\partial X_j}(E^0), \qquad V = \frac{\partial V_i}{\partial X_J}(E^0), \quad (9)$$

where E^0 is the disease-free equilibrium. The largest eigenvalue of the matrix FV^{-1} is the basic reproduction number denoted by:

$$R_0 = \rho(FV^{-1}). \quad (10)$$

Thus from (9), we have:

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_T \alpha_P \psi}{\mu_h} & 0 & \frac{\beta_T \alpha_b \psi}{\mu_h} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\theta\psi}{\mu_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\Lambda\gamma_P}{\mu_P+\rho} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\phi\gamma_C}{\sigma+\mu_C} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and}$$

$$V = \begin{pmatrix} \mu_h & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \mu_d + \mu_h & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \omega + \mu_P & 0 & 0 & 0 & 0 \\ 0 & 0 & -\omega & \alpha_P + \delta & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta + \mu_C & 0 & 0 \\ 0 & 0 & 0 & 0 & -\eta & \alpha_b + \epsilon & 0 \\ -\nu & 0 & 0 & 0 & 0 & 0 & \mu_e \end{pmatrix}.$$

Applying the definition in equation (10), we obtain:

$$R_0 = \sqrt{R_{HP} + R_{HC}}, \quad (11)$$

where

$$R_{HP} = \frac{\beta_T \nu \alpha_P \gamma_P \omega \psi \Lambda}{\mu_h^2 \mu_e (\delta+\alpha_P)(\omega+\mu_P)(\mu_P+\rho)} \text{ and } R_{HC} = \frac{\beta_T \nu \alpha_b \gamma_C \eta \psi \phi}{\mu_h^2 \mu_e (\epsilon+\alpha_b)(\eta+\mu_C)(\mu_C+\sigma)}. \quad (12)$$

R_{HP} is the partial reproduction number that arises from interaction of *Taenia solium* eggs with susceptible human and pig populations whereas R_{HC} denotes the partial reproduction number due to interaction of *Taenia saginata* eggs with susceptible human and pig populations.

The Endemic Equilibrium (EE)

In the presence of cysticercosis and taeniasis in humans, pigs and cattle, we obtain the endemic equilibrium given by $E^* = (S_H^*, I_{HT}^*, I_{HC}^*, S_P^*, I_P^*, P_I^*, S_C^*, I_C^*, B_I^*, E_T^*)$ where:

$$S_H^* = \frac{\psi}{\beta_T F_0 E_T^* + \theta E_T^* + \mu_h},\ I_{HT}^* = \frac{\beta_T F_0 E_T^* S_H^*}{\mu_h},\qquad I_{HC}^* = \frac{\theta E_T^* S_H^*}{\mu_h + \mu_d},\qquad S_P^* = \frac{\Lambda}{\gamma_P E_T^* + \mu_P + \rho},$$

$$I_P^* = \frac{\gamma_P \Lambda E_T^*}{(\gamma_P E_T^* + \mu_P + \rho)(\omega + \mu_P)},\qquad P_I^* = \frac{\omega \gamma_P \Lambda E_T^*}{(\gamma_P E_T^* + \mu_P + \rho)(\omega + \mu_P)(\alpha_P + \delta)},$$

$$S_C^* = \frac{\phi}{\gamma_C E_T^* + \sigma + \mu_C}, I_C^* = \frac{\gamma_C \phi E_T^*}{(\gamma_C E_T^* + \sigma + \mu_C)(\eta + \mu_C)},$$

$$B_I^* = \frac{\eta \gamma_C \phi E_T^*}{(\gamma_C E_T^* + \sigma + \mu_C)(\eta + \mu_C)(\alpha_b + \epsilon)}.$$

To obtain the expression for E_T^*, the polynomial (13) is solved to obtain its positive real roots.

$$a_0 E_T^{*3} + a_1 E_T^{*2} + a_2 E_T^* + a_3 = 0, \tag{13}$$

where:

$$a_0 = 1 > 0, a_1 = \frac{\alpha_P \omega \Lambda \beta_T}{\theta(\delta + \alpha_P)(\mu_P + \omega)} + \frac{\alpha_b \eta \phi \beta_T}{\theta(\eta + \mu_C)(\alpha_P + \epsilon)} + \frac{\sigma + \mu_C}{\gamma_C} + \frac{\rho + \mu_P}{\gamma_P} + \frac{\mu_h}{\theta} > 0,$$

$$a_2 = \frac{(\sigma+\mu_C)(\rho+\mu_P)}{\gamma_C \gamma_P} + \frac{\mu_h(\sigma+\mu_C)}{\theta\gamma_C} + \frac{\mu_h(\rho+\mu_P)}{\theta\gamma_P} + \frac{\alpha_P \omega \Lambda \beta_T(\sigma+\mu_C)}{\theta\gamma_C(\delta+\alpha_P)(\omega+\mu_P)} + \frac{\alpha_b \eta \phi \beta_T(\rho+\mu_P)}{\theta\gamma_P(\eta+\mu_C)(\alpha_P+\epsilon)} - \left(\frac{\psi \nu \alpha_P \omega \Lambda \beta_T}{\theta \mu_h \mu_e (\delta+\alpha_P)(\mu_P+\omega)} + \frac{\nu \psi \alpha_b \eta \phi \beta_T}{\theta \mu_h \mu_e (\eta+\mu_C)(\alpha_P+\epsilon)}\right),$$

$$a_3 = \frac{\mu_h(\sigma+\mu_C)(\rho+\mu_P)(1+R_0)(1-R_0)}{\theta\gamma_C\gamma_P},\ F_0 = \frac{\alpha_P \omega \gamma_P \Lambda}{(\gamma_P E_T^* + \rho + \mu_P)(\delta+\alpha_P)(\omega+\mu_P)} + \frac{\alpha_b \eta \gamma_C \phi}{(\gamma_C E_T^* + \sigma + \mu_C)(\eta+\mu_C)(\alpha_b+\epsilon)}.$$

We apply the method in Okosun et al. (2016) to study the nature of roots for polynomial (13) when $R_0 < 1$ and present the results in Table 2.

Table 2: Number of Positive Real Roots for equation (13)

Cases	a_0	a_1	a_2	a_3	R_0	No. of Sign Change	No. of positive real Roots
1	+	+	+	+	$R_0 < 1$	0	0
2	+	+	+	−	$R_0 > 1$	1	1
3	+	+	−	+	$R_0 < 1$	2	0 *or* 2
4	+	+	−	−	$R_0 > 1$	1	1

Therefore when $R_0 > 1$, there exists only one endemic equilibrium in the model system (1). Theorem 2 summarizes the results.

Theorem 2: *The system (1) has only one endemic equilibrium when* $R_0 > 1$.

A Continuous Time Markov Chain Model

Continuous time Markov chain (CTMC) stochastic models are important in disease modeling since they help to determine the chances of diseases' extinction or outbreak. Unlike deterministic models that assume continuous changes in state variables, CTMC stochastic models consider discrete number of state variables. A CTMC model is usually formulated based on its deterministic model's counterpart and the multitype branching process is adopted to find the likelihood of diseases' extinction or outbreak.

Formulation of CTMC Model

Assumptions, notations and parameters that were used in deterministic model are used to formulate a CTMC stochastic model. Allow $\vec{Z} = [S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I, E_T]^T$ as the random vector for all discrete random variables of the CTMC model. Table 3 summarizes the events and transition rates.

Table 3: Events and State Transitions

Event	Transition $\Delta\vec{Z}$	Rate of Transition
Recruitment of S_H	$(1,0,0,0,0,0,0,0,0,0)$	Λ_{H}
Human infection with infected pork	$(-1,1,0,0,0,0,0,0,0,0)$	$\alpha_P\beta_T P_I S_H$
Human infection with infected beef	$(-1,1,0,0,0,0,0,0,0,0)$	$\alpha_b\beta_T B_I S_H$
Human infection from environment	$(-1,1,0,0,0,0,0,0,0,0)$	$\theta E_T S_H$
Natural death of S_H	$(-1,0,0,0,0,0,0,0,0,0)$	$\mu_h S_H$
Natural death of I_{HT}	$(0,-1,0,0,0,0,0,0,0,0)$	$\mu_h I_{HT}$
Natural death of I_{HC}	$(0,0,-1,0,0,0,0,0,0,0)$	$\mu_h I_{HC}$
Disease induced death for I_{HC}	$(0,0,-1,0,0,0,0,0,0,0)$	$\mu_d I_{HC}$
Recruitment of S_P	$(0,0,0,1,0,0,0,0,0,0)$	Λ_P
Slaughter of S_P	$(0,0,0,-1,0,0,0,0,0,0)$	ρS_P
Infection of S_P	$(0,0,0,-1,1,0,0,0,0,0)$	$\gamma_P E_T S_P$
Natural death rate of S_P	$(0,0,0,-1,0,0,0,0,0,0)$	$\mu_P S_P$
Slaughter of I_P	$(0,0,0,0,-1,1,0,0,0,0)$	ωI_P
Natural death rate of I_P	$(0,0,0,0,-1,0,0,0,0,0)$	$\mu_P I_P$
Throwing of infected pork	$(0,0,00,0,,-1,0,0,0,0)$	δP_I
Consumption of infected pork	$(0,0,00,0,-1,0,0,0,0)$	αP_I
Recruitment of S_C	$(0,0,0,0,0,0,1,0,0,0)$	Λ_C
Slaughter of S_C	$(0,0,0,0,0,0,-1,0,0,0)$	σS_C
Infection of S_C	$(0,0,0,0,0,0,-1,1,0,0)$	$\gamma_c E_T S_C$
Natural death rate of S_C	$(0,0,0,0,0,0,-1,0,0,0)$	$\mu_b S_C$
Slaughter of I_C	$(0,0,0,0,0,0,0,-1,1,0)$	ηI_C
Natural death rate of I_C	$(0,0,0,0,0,0,0,-1,0,0)$	$\mu_b I_C$
Throwing of infected beef	$(0,0,0,0,0,0,0,0,-1,0)$	ϵB_I
Consumption of infected beef	$(0,0,0,0,0,0,0,0,-1,0)$	$\alpha_b B_I$
Shedding of E_T	$(0,0,0,0,0,0,0,0,0,1)$	νI_{TH}
Natural death of E_T	$(0,0,0,0,0,0,0,0,0,-1)$	$\mu_e E_T$

The value $-1, 0$ and 1 respectively represent, a decrease by 1, no change and increase by 1 in state variable from time t to $(t + \Delta t)$.

The CTMC stochastic model is homogeneous in time and fulfills the Markov property which explains that the future state of the process depends on the current state (Allen 2010, Maliyon et al. 2017). Using Markov assumptions, the time between events is exponentially distributed with parameter (Lahodny et al. 2015, Maliyoni et al. 2017):

$$\begin{aligned}\psi(\vec{Z}) = {} & \Lambda_H + \mu_h N_H + \Lambda_P + \mu_P N_P + \rho S_P + \omega I_P + (\delta + \alpha_p) P_I + \Lambda_C + \mu_b N_C \\ & + \nu I_{HT} + \sigma S_C + \eta I_C + (\epsilon + \alpha_b) B_I + \mu_e E_T + \beta(\alpha_p P_I + \alpha_b B_I) S_H \\ & + \gamma_p S_P E_T + \gamma_b S_C E_T. \end{aligned} \quad (14)$$

The Multitype Branching Process

The multitype branching process is used in this work to investigate the dynamics of taeniasis and cysticercosis close to the disease-free equilibrium (DFE) (Allen and Lahodny 2012, Allen and van de Drissche 2013, Maliyoni et al. 2017). This approach is also adopted to determine disease extinction or outbreak probabilities. If only few infectives exist at the beginning of the disease outbreak, then there is a possibility for the disease to grow exponentially or die (Allen 2017). Pigs, cattle and humans who are susceptible are regarded to be at DFE, that is $S_P^0 = \phi/(\sigma + \mu_b), S_C^0 = \Lambda/(\rho + \mu_p)$ and $S_H^0 = \psi/\mu_h$ (Lahodny and Allen 2013). Type i infectious hosts are considered to infect a susceptible

individual of another type leading to type j infectious individuals. Type i individuals who are infected are distinct from those produced by type i, I_i or type j,I_j, $j \neq i$ (Allen and van den Drisssche 2013, Maliyoni et al. 2017). The initial susceptible human, pig and cattle populations are considered to be adequately large, that is $S_H(0) \approx N_H(0) = S_H^0$, $S_P(0) \approx N_P(0) = S_P^0$ and $S_C(0) \approx N_C(0) = S_C^0$ where $N_P(0)$, $N_C(0)$ and $N_H(0)$ are the initial total populations for pigs, cattle and humans respectively. The offspring probability generating functions (pgfs) for infectious humans, cattle, taenia eggs, infectious beef and pork are defined which are then used to compute the probability of diseases' extinction or outbreak (Kyvsgaard et al. 2007).

Let Z_{ji} for $i, i = 1,2,\dots,n$ be the number of type j individuals produced by type i infective. The offspring probability generating function (pgf) $f_i : [0,1]^n \to [0,1]$ for individuals of type i if $I_i(0) = 1$ and $I_j(0) = 0, j = i$ is (Allen 2020, Lahodny and Allen, 2013, Maliyoni, 20202):

$$f_i(v_1, v_2, \dots, v_n) = \sum_{r_n=0}^{\infty} \dots \sum_{r_1=0}^{\infty} P_i(r_1, r_2, \dots, r_n) v_1^{r_1} \dots v_n^{r_n}, \tag{15}$$

where

$$P_i(r_1, r_2, \dots, r_n) = Prob\{Z_{1j} = r_1, Z_{2j} = r_2, \dots, Z_{nj} = r_n\} \tag{16}$$

is the probability that one infectious type i individual produces n_j infectious type j individuals. Equation (15) defines an $n \times n$ expectation matrix $\boldsymbol{M} = [m_{ji}]$ where m_{ji} is the expected number of type j individuals produced by type i infective. Matrix $\boldsymbol{M}$ elements are non-negative and they are obtained using the formula (Lahodny et al. 2015, Maliyoni 2020):

$$m_{ji} = \left.\frac{\partial f}{\partial v_j}\right|_{v=1} . \tag{17}$$

If the spectral radius $\rho(\boldsymbol{M}) \leq 1$, then the probability of disease extinction is one and if $\rho(\boldsymbol{M}) > 1$ then there exists a positive number less than one which indicates that there is disease persistence, that is:

$$P_0 = \lim_{t\to\infty} Prob\left\{\overrightarrow{I(t)} = \vec{0}\right\} = q_1^{i1} q_2^{i2} \dots q_n^{in} < 1, \tag{18}$$

where $(q_1, q_2, \dots, q_n)$ is the fixed point of the n offspring pgf, $f_i(q_1, q_2, \dots, q_n) = q_i$ and $0 < q_i < 1$ for $i = 1, 2, \dots, n$ (Kyvsgaard et al. 2007, Lahodny et al. 2015, Maliyoni et al. 2017). The value of q_i represents the probability of disease extinction for type i infectives and the probability of disease outbreak is given by (Maliyoni 2020, Maliyoni et al. 2017):

$$1 - P_0 = 1 - q q_1^{i1} q_2^{i2} \dots q_n^{in}. \tag{19}$$

CTMC Stochastic Threshold for the Epidemic Model

The multitype branching process is adopted to define the probability generating functions for all infectious classes. If there is only one human with taeniasis ($I_{HT}(0) = 1$) at the beginning of the diseases' outbreak and there are no other infectious classes, that is ($I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 0, I_C(0) = 0, B_I(0) = 0$ and $E_T(0) = 0$), the offspring pgf for humans with taeniasis I_{TH} is:

$$f_1(u_1, u_2, .., u_7) = \frac{\nu u_1 u_7 + \mu_h}{\nu + \mu_h} . \tag{20}$$

The expression $\nu/(\nu + \mu_h)$ represents the likelihood that the initial infectious human defecates a taenia egg in the environment, leading to one human with taeniasis and a taenia egg

in the environment. The likelihood that an initial human with taeniasis dies due to natural death leading to no infectious human with taeniasis is given by $\mu_h/(\nu + \mu_h)$.

Since humans who are infected with cysticercosis do not play any role in the spread of the disease, therefore the offspring pgf for I_{HC} given that $I_{HT}(0) = 0, I_{HC}(0) = 1, I_P(0) = 0, P_I(0) = 0, I_C(0) = 0, B_I(0) = 0, E_T(0) = 0$ is:

$$f_2(u_1, u_2, \ldots, u_7) = 1. \tag{21}$$

The offspring pgf for I_P given that $I_{HT}(0) = 0, I_{HC}(0) = 0, I_P(0) = 1, P_I(0) = 0, I_C(0) = 0, B_I(0) = 0, E_T(0) = 0$ is:

$$f_3(u_1, u_2, \ldots, u_7) = \frac{\omega u_4 + \mu_P}{\omega + \mu_P}. \tag{22}$$

The term $\omega/(\omega + \mu_p)$ is the probability for the initial infected pig to be slaughtered for con sumption leading to the presence of infectious pork only whereas $\mu_p/(\omega + \mu_p)$ is the probability for the initial infected pig to die naturally resulting to no infected pigs and infectious pork.

The offspring pgf for P_I given that $I_{HT}(0) = 0, I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 1, I_C(0) = 0, B_I(0) = 0, E_T(0) = 0$ is:

$$f_4(u_1, u_2, \ldots, u_7) = \frac{\beta_T \alpha_p S_H^0 u_1 u_4 + \delta}{\beta_T \alpha_p S_H^0 + \delta}. \tag{23}$$

The term $\beta_T \alpha_p S_H^0/(\beta_T \alpha_p S_H^0 + \delta)$ is the probability that a proportion of initial infectious pork is consumed by a susceptible human whereas $\delta/(\beta_T \alpha_p S_H^0 + \delta)$ is the probability that the initial infectious pork is thrown following meat inspection leading to no infectious pork.

The offspring pgf for I_C given that $I_{HT}(0) = 0, I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 1, I_C(0) = 1, B_I(0) = 0, E_T(0) = 0$ is:

$$f_5(u_1, u_2, \ldots, u_7) = \frac{\eta\, u_6 + \mu_b}{\eta + \mu_b}. \tag{24}$$

The expression $\mu_b/(\eta + \mu_b)$ represents the chance that the initial infectious cattle may die before being slaughtered, leaving zero infectious cattle and infectious beef. The term $\eta/(\eta + \mu_b)$ represents the likelihood that the initial infectious cattle is slaughtered for consumption, resulting in the presence of infectious beef only.

The offspring pgf for B_I given that $I_{HT}(0) = 0, I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 1, I_C(0) = 1, B_I(0) = 1, E_T(0) = 0$ is:

$$f_6(u_1, u_2, \ldots, u_7) = \frac{\beta_T \alpha_b S_H^0 u_1 u_6 + \epsilon}{\beta_T \alpha_b S_H^0 + \epsilon}. \tag{25}$$

The term $\beta_T \alpha_b S_H^0/(\beta_T \alpha_b S_H^0 + \epsilon)$ denotes the probability that a proportion of initial infectious beef consumed by a susceptible human. The term $\epsilon/(\beta_T \alpha_b S_H^0 + \epsilon)$ is the probability that the initial infectious beef is thrown before consumption following meat inspection resulting to no infectious beef and thus no human with taeniasis.

The offspring pgf for E_T given that $I_{HT}(0) = 0, I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 1, I_C(0) = 1, B_I(0) = 0, E_T(0) = 1$ is:

$$f_7(u_1, u_2, \ldots, u_7) = \frac{\theta S_H^0 u_2 u_7 + \gamma_P S_P^0 u_3 u_7 + \gamma_b S_C^0 u_5 u_7 + \mu_e}{\theta S_H^0 + \gamma_P S_P^0 + \gamma_b S_C^0}. \tag{26}$$

The term $\theta S_H^0/(\theta S_H^0 + \gamma_P S_P^0 + \gamma_b S_C^0 + \mu_e)$ is the probability that the initial taenia egg infects a susceptible human resulting to one human with cysticercosis and a taenia egg. The term

$\gamma_P S_P^0/(\theta S_H^0 + \gamma_P S_P^0 + \gamma_b S_C^0 + \mu_e)$ represents the likelihood that a susceptible pig becomes infected by the initial taenia egg, resulting in one infected pig and one taenia egg. The expression $\gamma_b S_C^0 /(\gamma_P S_P^0 + \gamma_b S_C^0 + \mu_e)$ denotes the likelihood that a susceptible cattle will be infected by the initial taenia egg, producing one infectious cattle and one taenia egg, where as $\mu_e/(\theta S_H^0 + \gamma_P S_P^0 + \gamma_b S_C^0 + \mu_e)$ is the likelihood that a taenia egg will die naturally.

The expectation matrix M for the offspring pgfs is given by:

$$\boldsymbol{M} = \begin{pmatrix} \frac{\nu}{\nu+\mu_h} & 0 & 0 & \frac{\beta_T \alpha_P S_H^0}{\beta_T \alpha_P S_H^0 + \delta} & 0 & \frac{\beta_T \alpha_b S_H^0}{\beta_T \alpha_b S_H^0 + \epsilon} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\theta S_H^0}{J} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_P S_P^0}{J} \\ 0 & 0 & \frac{\omega}{\omega+\mu_h} & \frac{\beta_T \alpha_P S_H^0}{\beta_T \alpha_P S_H^0 + \delta} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_b S_C^0}{J} \\ 0 & 0 & 0 & 0 & \frac{\eta}{\eta+\mu_h} & \frac{\beta_T \alpha_b S_H^0}{\beta_T \alpha_b S_H^0 + \epsilon} & 0 \\ \frac{\nu}{\nu+\mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\theta S_H^0 + \gamma_P S_P^0 + \gamma_b S_C^0}{J} \end{pmatrix}$$

where $J = \theta S_H^0 + \theta S_H^0 + \theta S_H^0 + \mu_e$.

The spectral radius of the expectation matrix $\rho(\boldsymbol{M})$, serves as the stochastic threshold for disease extinction or outbreak in humans, pigs and cattle for the CTMC stochastic model. The stochastic threshold $\rho(\boldsymbol{M})$ for CTMC stochastic model and the basic reproduction number R_0 for deterministic model have a close relationship (Maliyoni et al. 2017). The disease dies in human, pigs and cattle if $\rho(\boldsymbol{M}) \leq 1$ or $R_0 \leq 1$. In deterministic models, the diseases persist in humans, pigs and cattle if $R_0 > 1$. However, this is not realistic because if there are only few infectives at the beginning of the disease outbreak, there is a possibility for these infectives to die, recover or be removed before transmitting the disease (Mwasunda et al. 2022). Therefore, in stochastic models, if $\rho(\boldsymbol{M}) > 1$ there is a possibility for disease outbreak or extinction depending on number of infectives that were initially available at the outset of the disease outbreak (Allen and van de Drisssche 2013, Lahodny and Allen 2013, Maliyoni 2020). Thus, if $\rho(\boldsymbol{M}) > 1$, there exist a fixed point $(q_1, q_2, q_3, q_4, q_5, q_6, q_7) \in (0,1)^7$ of the offspring generating functions (20)-(26) that is used in writing the probability of disease extinction (Allen and Lahodny 2012, Maliyoni et al. 2017). To get the fixed point, we set $f_i(q_1, q_2, q_3, q_4, q_5, q_6, q_7) = q_i \; for \; i = 1, 2, .., 7$. That is:

$$\begin{aligned} q_1 &= \tfrac{\nu q_1 q_7 + \mu_h}{\nu+\mu_h}, q_2 = 1, q_3 = \tfrac{\omega q_4 + \mu_P}{\omega+\mu_P}, \\ q_4 &= \frac{\beta_T \alpha_P S_H^0 q_1 q_4 + \delta}{\beta_T \alpha_P S_H^0 + \delta}, q_5 = \frac{\eta\, q_6 + \mu_b}{\eta + \mu_b}, \\ q_6 &= \frac{\beta_T \alpha_b S_H^0 q_1 q_6 \; + \; \epsilon}{\beta_T \alpha_b S_H^0 + \epsilon}, \\ q_7 &= \tfrac{\theta S_H^0 q_2 q_7 + \gamma_P S_P^0 q_3 q_7 + \gamma_b S_C^0 q_5 q_7 + \mu_e}{\theta S_H^0 + \gamma_P S_P^0 + \gamma_b S_C^0}. \end{aligned} \tag{27}$$

Due to non-linearity of probability generating functions (pgf) particularly the pgf in (27), q_i's are computed through numerical simulations.

Numerical Simulations

To study the transmission dynamics of cysticercosis and taeniasis in human, pig and cattle populations, we first simulate the basic model system (1) and then with its corresponding CTMC stochastic model for comparison purposes using the parameter values in Table 1. We apply the initial conditions:

$$I_{HT}(0) = 1, I_{HC}(0) = 0, S_H(0) = 21{,}275, I_P(0) = 1, P_I(0) = 1, S_P(0) = 118,$$
$$I_C(0) = 1, B_I(0) = 1, S_C(0) = 220, E_T(0) = 2. \quad (28)$$

Basic Model Simulation

The basic model results in Figure 2 indicate that all susceptible humans, pigs and cattle becomes infected and thus decline with time whereby some proportions of these populations become uninfected. Infected classes for humans, pigs and cattle initially increase rapidly in the first 10 years and then slowly to settle at their steady states. These trends are in correspondence with the growth of taenia eggs in the environment that infects both susceptible humans, pigs and cattle; and the available infectious beef and pork that is consumed by susceptible humans. The trends signify that the taeniasis and cysticercosis will continue to persist in these population if measures are not taken to control the diseases.

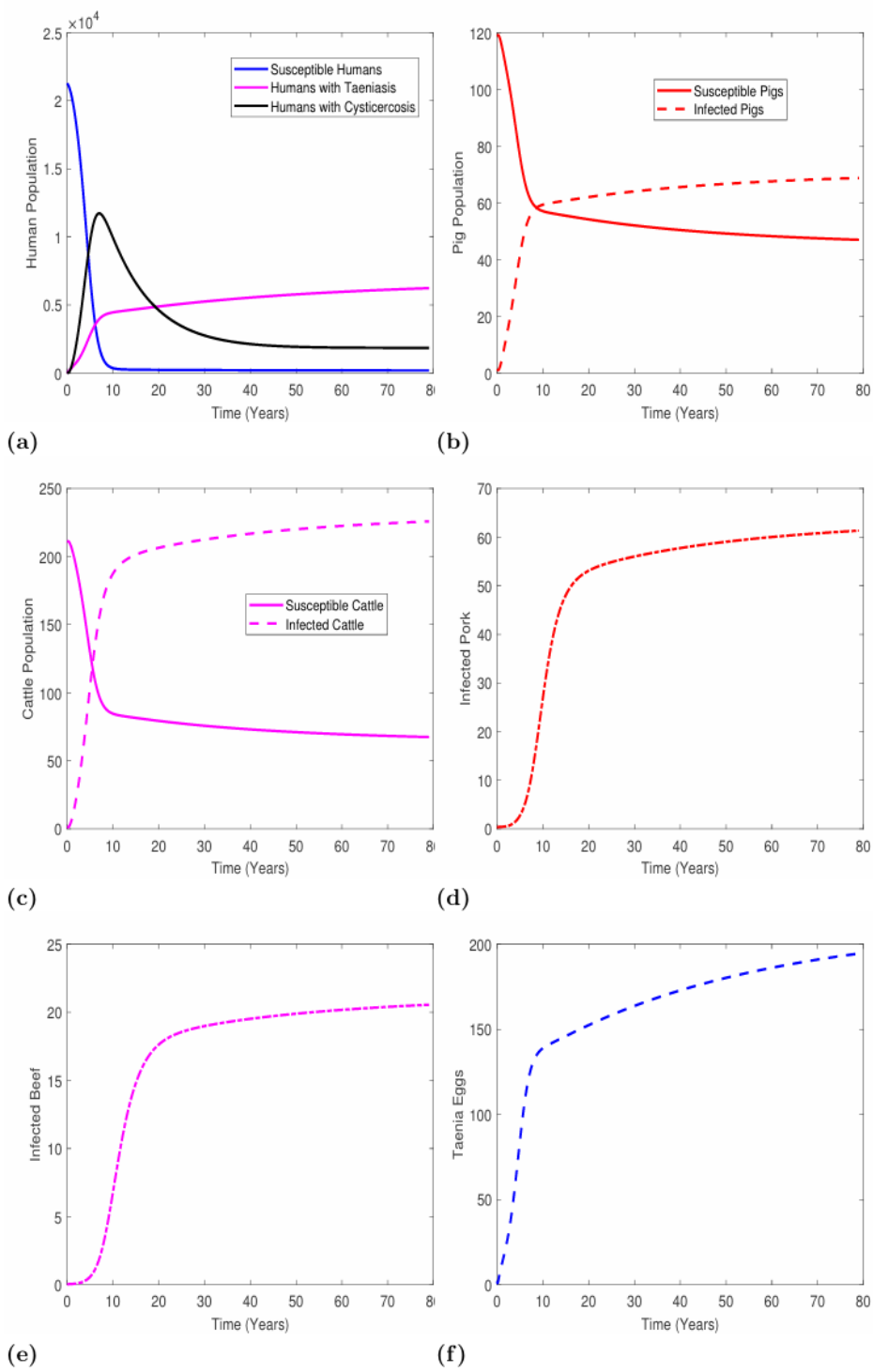

Figure 2: Dynamics of humans, pigs, cattle, pork, beef and taenia eggs for the deterministic model

CTCM Model Simulation

In this section, the deterministic model (1) is simulated with its corresponding CTMC stochastic model to study the dynamics of cysticercosis and taeniasis in pig, human and cattle populations. Simulation results are presented in Figures 3 and 4. The results show that CTMC model solutions are relatively close to the corresponding deterministic model solutions. CTMC model results fluctuates with the solutions of deterministic model.

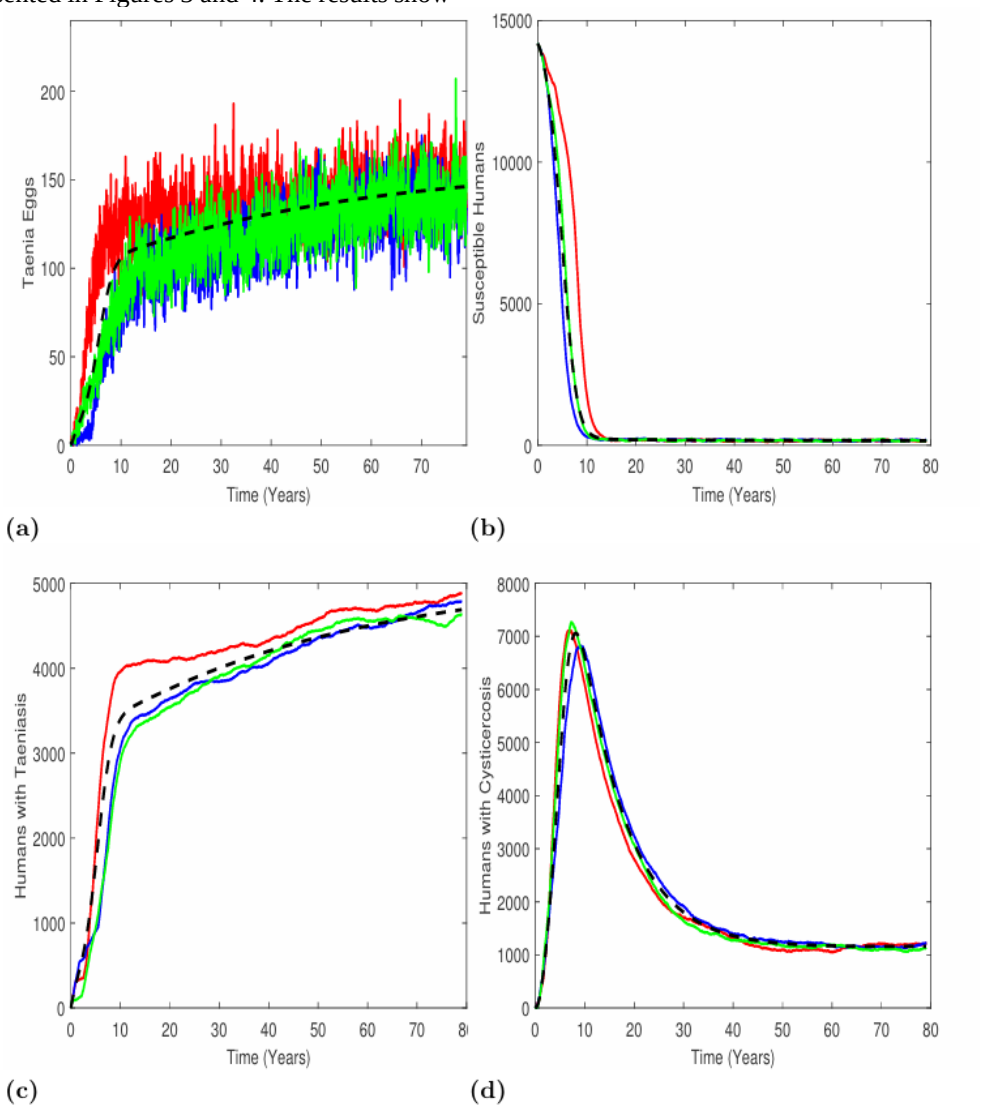

Figure 3: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for humans and taenia eggs in the environment

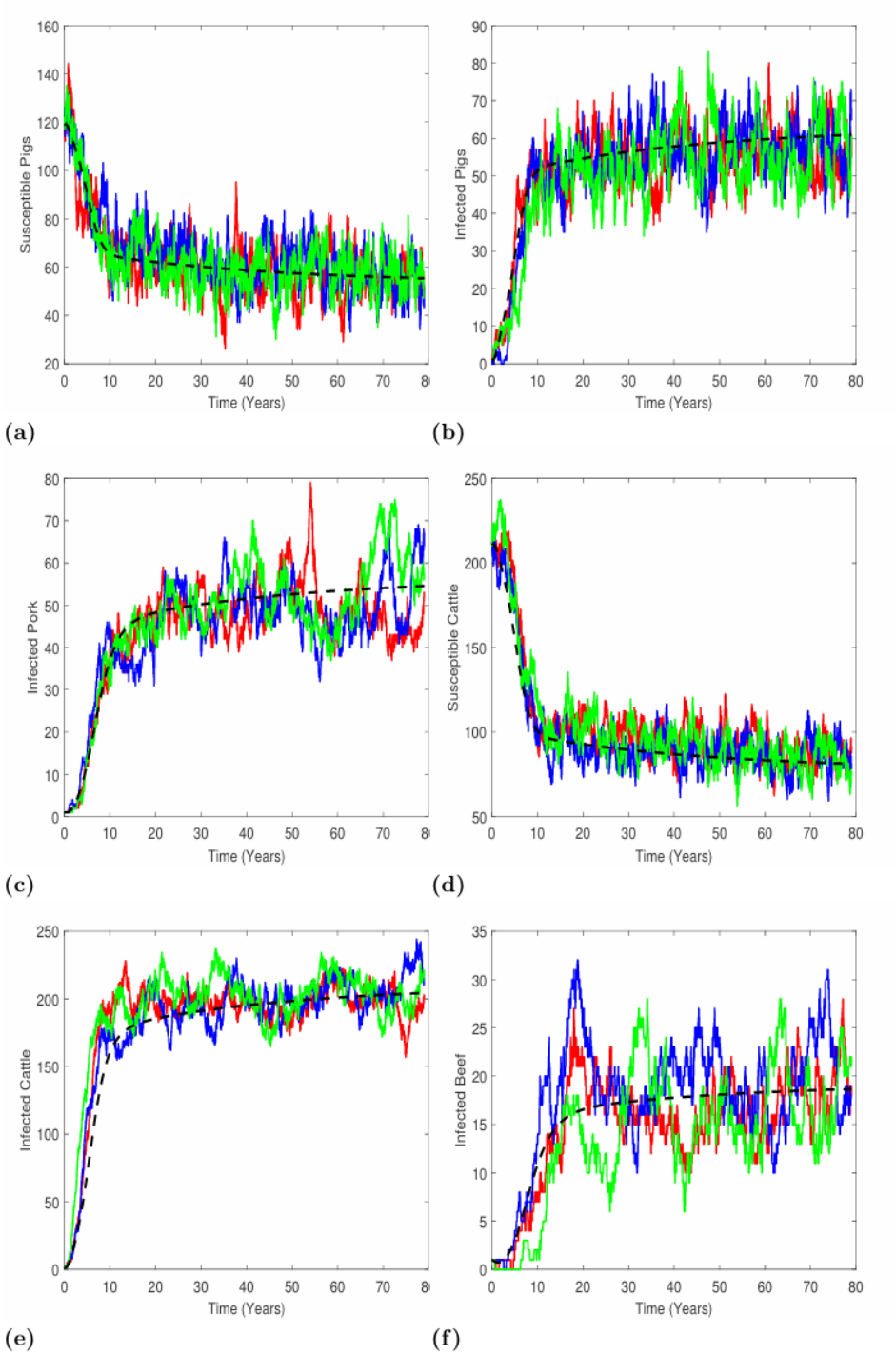

Figure 4: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for pigs, cattle, infected pork and beef

The likelihood of Outbreak or Extinction of Taeniasis and Cysticercosis

Using 10,000 sample paths of the CTMC stochastic model, the multitype branching process is used to calculate the approximate probability of disease extinction Pa. The approximation used in CTMC model simulation is the number of sample pathways that hit zero prior to the disease outbreak. The probabilities are calculated through varying the initial number of infectives. Depending on the initial number of infectives at the beginning of the disease outbreak, Figure 5 shows that some sample paths of the CTMC stochastic model hit zero, indicating the possibility of taeniasis and cysticercosis to clear in humans, pigs and cattle even if the stochastic threshold $\rho(\boldsymbol{M})$ is greater than one.

When $I_{HT}(0) = y_1 = 1, I_{HC}(0) = y_2 = 0, I_P(0) = y_3 = 1, P_I(0) = y_4 = 0, I_C(0) = y_5 = 1,\ B_I(0) = y_6 = 0, E_V(0) = y_7 = 1$ for instance, as seen in Figures 5 and 6, some sample trajectories of the CTMC model hit zero despite $R_0 = 19.4975 > 1$ and $\rho(\boldsymbol{M}) = 1.5877 > 1$. This indicates that the population following this sample path is rapidly absorbed and eventually approaches the disease-free equilibrium. On the other hand, when a significant number of infectious individuals is introduced into the susceptible population, the diseases will not eventually become extinct as seen in Figures 2 and 3 using initial conditions from equation (28).

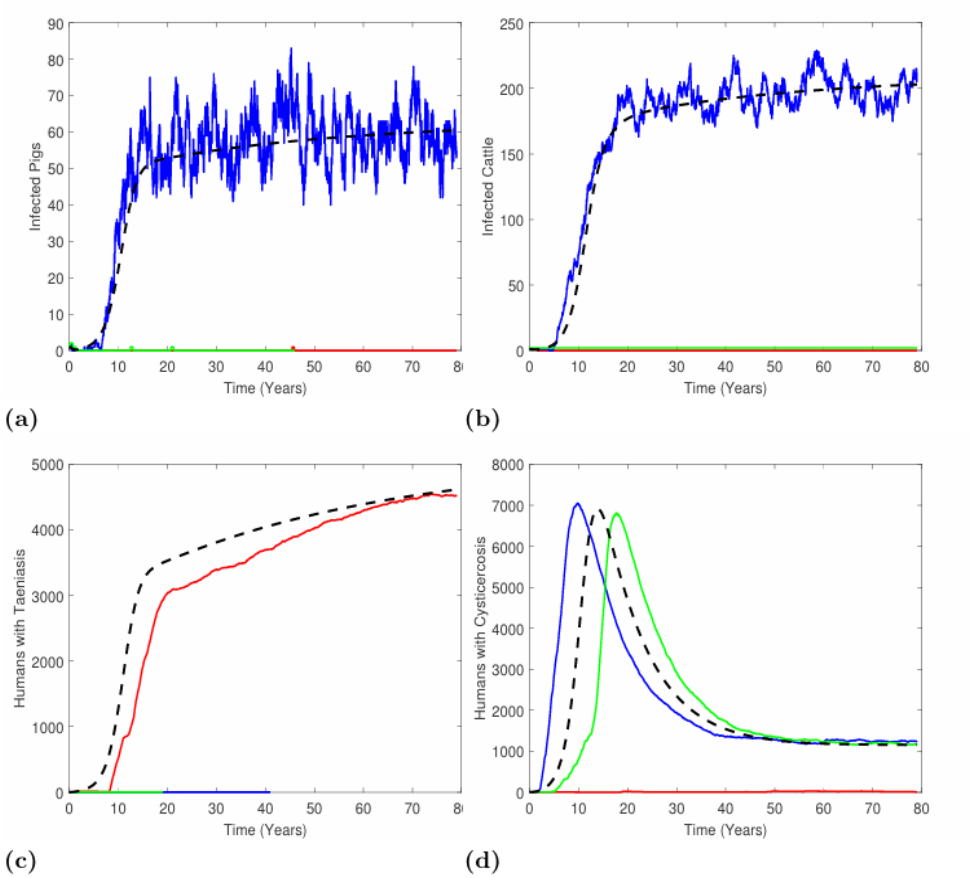

Figure 5: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for infected humans, pigs and cattle

Findings in Table 4 demonstrate that the dynamics of the disease are influenced by the initial number of infectious agents introduced into susceptible populations. The data also show that if the diseases originate from a small number of *Taenia* eggs, there is a very high possibility that they will eventually go to extinction. This is due to the fact that the initial number of taenia eggs will decay with time and exhaust as there are no individuals who are shedding eggs in the environment. If the diseases originate from humans who have cysticercosis, they are likely to automatically go to extinction because humans with cysticercosis are just dead-end hosts. There is a major outbreak of taeniasis and cysticercosis if the diseases emerge from humans with taeniasis or from infectious pork and beef. This is because humans with taeniasis will continue to shed eggs in the environment consequently affecting humans, pigs and cattle. Upon consumption of infectious pork and beef, repeats the cycle of the diseases. In all other cases, there is a chance of a significant disease outbreak in humans, pigs, and cattle where the probability of disease extinction is either very low or zero. These findings indicate that the best interventions for controlling cysticercosis and taeniasis in pigs, humans, and cattle focus on lowering the number of taeniasis-infected humans as well as infectious pork and beef at the onset of the disease outbreak.

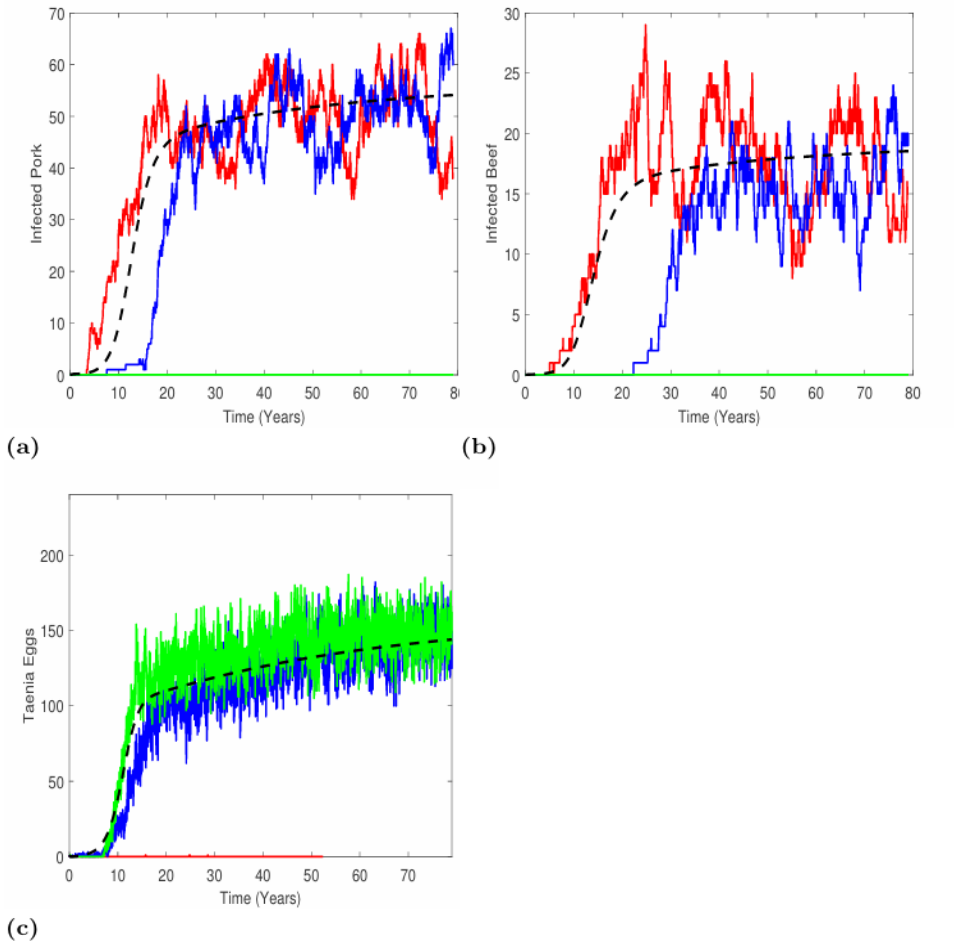

Figure 6: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for infected pork, beef and taenia eggs in the environment

Table 4: Probability of Disease Extinction

y_1	y_2	y_3	y_4	y_5	y_6	y_7	P_a
1	0	0	0	0	0	0	0.0008
0	1	0	0	0	0	0	1.0000
1	1	0	0	0	0	0	0.0013
0	0	0	0	0	0	1	0.5076
0	0	0	0	1	0	0	0.0241
0	0	0	0	0	1	0	0.0015
0	0	0	0	1	1	0	0.0000
0	0	1	0	0	0	0	0.0809
0	0	0	1	0	0	0	0.0170
0	0	1	1	0	0	0	0.0011
1	1	1	1	0	0	0	0.0000
1	1	0	0	1	1	0	0.0000
1	1	0	0	0	0	1	0.0003
0	0	0	0	1	1	1	0.0000
0	0	1	1	0	0	1	0.0008
0	0	1	1	1	1	0	0.0000
0	0	1	1	1	1	1	0.0000
1	1	1	1	1	1	1	0.0000

Conclusion

In this study, the continuous-time Markov chain (CTMC) stochastic model that corresponds to the deterministic model for the transmission dynamics of cysticercosis and taeniasis in human, pig, and cattle populations is studied and carefully analyzed. To ascertain whether the diseases may die or endure in humans, pigs, and cattle, the basic reproduction R_0 for the deterministic model is computed through the next generation matrix approach. The stochastic threshold $\rho(\boldsymbol{M})$ for the CTMC stochastic model is computed through the multitype branching process, which offers the prerequisites for the emergence or extinction of the diseases. In general, cysticercosis and taeniasis disappear in humans, pigs, and cattle if $R_0 < 1$ and $\rho(M) <1$ the diseases persist in humans, pigs, and cattle, whereas if $\rho(\boldsymbol{M}) > 1$ the diseases may spread or disappear depending on the initial number of infectious agents. Numerical results for probability of disease extinction P_a is computed from the multitype branching process using 10,000 sample paths. According to the findings, there is a good chance that taeniasis and cysticercosis will go-extinct if they develop from a number of taenia eggs, and they will also likely go extinct if they develop from cysticercosis-infected humans. There is a major outbreak of taeniasis and cysticercosis if the diseases emerge from humans with taeniasis or from infectious pork and beef. Therefore, to control the disease outbreak in humans, pigs, and cattle, interventions that focus on the control of humans with taeniasis and consumption of infectious beef and pork at the onset of the disease. Focus on the control of humans with taeniasis and consumption of infectious beef and pork at the onset of the disease outbreak are necessary.

Declaration of Competing Interests

The authors declare that they have no competing interests upon publication of this work.

Authors' Contribution

J.A. Mwasunda: Model Formulation, Analysis and Manuscript Drafting; J.I. Irunde: Model Development and Supervision.

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