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Modelling campylobacteriosis dynamics: Impacts of contaminated animal products and environmental decontamination interventions

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ABSTRACT

Campylobacteriosis is responsible for approximately 500 million cases of illness globally each year. Globally, human campylobacteriosis infections and contaminated animal products cause an estimated loss of 8.6 and 12.6 billion US dollars annually, respectively. The disease is transmitted through consumption of contaminated foods and water, licking unsanitary hands and contact with infected hosts. As global demand for animal products like meat and milk continues to grow, the transmission of campylobacteriosis through these products has become a critical concern. This study aims at utilising mathematical modelling and analysis techniques to quantify the effects of contaminated animal products and environmental decontamination interventions on campylobacteriosis dynamics in host populations. A mathematical model as a system of ordinary differential equations is proposed with human and cattle populations and contaminated animal products. The next-generation matrix method is applied to compute the effective reproduction number $\mathcal{R}$ that describes disease persistence and extinction. The global stability of equilibria states is examined using the Lyapunov stability theory. The uncertainty and sensitivity of model parameters are examined using the Latin Hypercube Sampling and Partial Rank Correlation Coefficient methods. Model fitting and parameter estimations are performed using the least squares method alongside the human cases from January to August for the years 2017 to 2020 in the EU. The analysis indicates that the disease-free and endemic equilibria are globally asymptotically stable whenever $\mathcal{R} < 1$ and $\mathcal{R} > 1$, respectively. The numerical results show that the ingestion rates of contaminated animal products, shedding rates and the natural replication rates of *Campylobacter* jejuni bacteria are directly proportional to $\mathcal{R}$, while the environmental cleanliness and the decay rate of *Campylobacter* jejuni bacteria are inversely proportional to $\mathcal{R}$. In order to reduce the impact of contaminated animal products, the study recommends a couple of strategies for reducing shedding rates, killing bacteria, and vaccinating infected hosts.

Introduction

Campylobacteriosis is a zoonotic, food- and water-borne disease caused by *Campylobacter* jejuni bacteria [1]. It infects the intestines of humans and cattle [2]. In the human population, the disease spreads through the consumption of bacteria in food items, water, unsanitary hands, surfaces, home appliances, and other environmental settings, or through contact with infected

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humans, including breastfeeding. Infected humans experience fever, abdomen pain, and diarrhea one to three days after exposure to the bacteria [1,3]. The symptoms last for five to eight days [4]. Cattle contract campylobacteriosis through the faecal–oral route (consumption of contaminated grasses, licking the skins of infected animals and soil, breastfeeding, and drinking contaminated water), just like humans do. Infected cattle may experience fever, diarrhea, mucus, loss of appetite, and occasionally blood in the stool and abortion [1,2].

In an infectious phase, infected humans and cattle might infect susceptible ones and shed the bacteria in their faeces, which can contaminate the environment [1,5]. Contamination of water sources, glasses, vegetables, and fruits can result in the outbreaks, especially in areas where these are primary drinking water and food sources [3,6]. Additionally, contamination of swimming pools, rivers and lakes through run-off from sewage spills, agricultural lands, or improper waste disposal causes infection through exposure during recreational pursuits [7,8]. Furthermore, bacteria in the environment survive for extended periods under favourable environmental conditions like temperature and pH, increasing the risk of transmission [6,9].

Zoonotic infections, including campylobacteriosis, are responsible for an estimated 2.5 billion cases of illness and 2.7 million deaths globally each year [10]. As global demand for animal products like meat and milk continues to grow [11], the transmission of zoonotic diseases through these products has become a critical concern. Campylobacteriosis remains a major public health issue, posing significant risks to both humans and dairy cattle. In 2023, campylobacteriosis was the first most frequently reported zoonotic, food- and waterborne disease in humans [12]. The disease not only endangers health but also has economic consequences, as outbreaks can lead to restrictions or bans on the export of dairy cattle and products, limiting access to international markets and reducing potential foreign exchange earnings. Campylobacteriosis is responsible for approximately 500 million cases of illness each year, globally. An estimated 8.6 and 12.6 billion US dollars are lost globally each year due to human campylobacteriosis infections and contaminated animal products, respectively [13]. In 2023, campylobacteriosis accounted for 148,181 human cases, where 12,194 humans were hospitalised and 83,180 died in the European Union (EU). A cost of 2.4 billion euros is spent to control campylobacteriosis each year in the EU [12]. In the United States of America (USA), campylobacteriosis is the most common cause of diarrhea, resulting in approximately 1.5 million human cases, and 1.56 billion dollars are spent to control the disease annually [14,15]. Although campylobacteriosis is regarded as a serious public health issue in sub-Saharan Africa, Asia, and the Middle East, there is insufficient surveillance to determine the exact illness burden in these regions [1,16–18]. The sub-Saharan region's human prevalence is estimated to be between 8.5% and 14.1%. When looking at particular risk categories in sub-Saharan Africa, the prevalence rates for children under five years old are 50.6% [17], HIV patients are 45.0% [19], and diarrhea patients are 20.3% [20]. The overall prevalence of *Campylobacter* jejuni bacteria in the digestive tracts of animals, including cattle, was reported to exceed 80% in 2019 globally [21].

Mathematical modelling approaches have been used to simulate the spread of diseases through populations in order to quantify disease outcomes, including the burden of disease and the number of secondary cases arising from a single case, quantify the effects of epidemiological parameters, ecological and socio-economic factors, and assess the optimal allocation and utilisation of resources needed to control the spread of the diseases [6,9,22,23]. Owing to its global prevalence, rising incidence rates, unpredictable health risks, and the associated economic consequences, numerous mathematical models have been developed over the years to enhance the understanding of its transmission dynamics, severity, and potential control strategies.

Chuma and Ngailo [1] proposed a deterministic model to study the dynamics of campylobacteriosis disease among the human population. Their model incorporated the effects of public health education, treatment, and sanitation. The effective reproduction number was computed using the next generation matrix method. Important parameters that are responsible for disease transmission dynamics were identified through sensitivity analysis. Both local and global stability of equilibrium states were performed. Pontryagin's maximum principle was adopted to evaluate the best control strategy. Numerical findings showed that both treatment and improved hygiene practices through cumulative healthy public education campaigns are vital for effective control of campylobacteriosis in the human population. However, their study relies on assumed parameter values, which could produce wrong results. Furthermore, their study is isolated, as it considers only one population species, the attempt that cannot eliminate the *Campylobacter* jejuni bacteria life cycle in interacting animal hosts.

Parshotama [24] proposed a model of *Campylobacter* transmission in dairy herds. The model included the concentration of *Campylobacter* bacteria in the environment and infectious stages among the cattle population. The effective reproduction number was computed using the next generation matrix method. The fourth-order Runge–Kutta method was used to solve the system of ordinary differential equations. The dynamic behaviour of the model equations was assessed through the variation of epidemiological parameters. The threshold values of the basic reproduction number produced by the variation of the values of the epidemiological parameters against the basic reproduction number were used to assess the impact of epidemiological parameters on the infection cases. The results indicate that one infected cow would infect an average of 0.2 susceptible cattle, and the disease would become extinct in the community. Environmental factors like temperature, humidity, and sunlight were found to have an impact on the survival of environmental bacteria, and the transmission rates between cattle and the environment vary seasonally. However, their modelling attempt might not eliminate the *Campylobacter* bacteria life cycle in the host animals that interact.

A deterministic model for campylobacteriosis disease with optimal control was proposed by Osman et al. [2]. The nonstandard finite difference scheme was used to check the behaviour of the model solution. The stability analysis of the equilibrium points was checked using the eigenvalue techniques. The model was extended to the optimal control problem using prevention of susceptible humans and treatment of infected humans and animals strategies. The Pontryagin maximum principle was adopted to formulate necessary conditions for optimality. The Runge–Kutta fourth-order scheme was used to solve the state systems and adjoint equations under the transversality condition. The findings show that the most effective control strategy was treating infected animals and preventing susceptible humans. Their work neglected the contaminated environment, which is a sole accommodation

for *Campylobacter* bacteria. Additionally, the term 'animals' is too general. Animals differ in their shedding and consumption rates of *Campylobacter* bacteria. Putting all animals in one basket could result in wrong model predictions.

A model by Cousins et al. [25] was proposed to describe the transmission dynamics of campylobacteriosis among humans and house flies (Musca domestica) in Ontario, Canada. Their work aimed to ascertain whether the observed disease dynamics could be captured by a simple SIR compartmental framework. The parameters were estimated, and the model was fit to *Campylobacter* incidence in Ontario from 1 January to 31 December 2005 using R's optimising technique. The optimisation technique is based on Nelder–Mead, quasi-Newton and conjugate-gradient algorithms adopted from the existing literature. The model graphically appeared to have a good fit to the observed incidence data set. Latin hypercube sampling and partial rank correlation coefficient methods were adopted in the uncertainty and sensitivity analysis of model parameters. The analysis indicated that the model was most sensitive to the latent period, the environmental parameter and the fly death rate. The model was then used to estimate changes in campylobacteriosis incidence using anticipated changes in fly population size and fly activity under various climatic conditions. The results indicated that, when fly activity levels were at a 25% increase, the model predicted a 28.15% increase in incidence using the medium–low emissions scenario and a 30.20% increase using the high emissions scenario. Their study assumed that each susceptible human has an equal chance of coming into contact with contaminated environments, infected humans, and flies. This might not be the case in practice. The exclusion of other components, such as cattle population and animal products that are potential carriers of the *Campylobacter* bacteria, puts another shortcoming on their study.

While there has been progress in understanding campylobacteriosis transmission dynamics and control through mathematical modelling approaches, significant gaps remain in considering two interactive hosts (humans and cattle), quantifying the risk of contaminated animal products and environment, and the assessment of environmental cleanliness thresholds in reducing campylobacteriosis cases. This study aims at utilising mathematical modelling and analysis techniques to quantify the effects of contaminated animal products on campylobacteriosis dynamics. This study also highlights the need for decontamination efforts through environmental interventions (cleanliness) in order to reduce campylobacteriosis cases.

A mechanistic mathematical model as a system of ordinary differential equations (ODEs) is proposed. A model considers human and cattle populations and the number of colony-forming units (CFU) of *Campylobacter* jejuni bacteria in animal products and the environment. Animal products include milk and meat, while the environment includes water sources (rivers, lakes, groundwater, and swimming pools); sewage systems; surfaces; home appliances; soil; vegetation; and other environmental components.

This study differentiates between animal products (milk and milk) and other foods derived from the natural environment, like fruits, salads and vegetables, to create a more realistic model of zoonotic disease transmission. Differentiation helps to quantify the zoonotic risk associated with the consumption of animal products and the microbial risk in fresh fruits, vegetables, and salads [9,23]. Accurate risk assessment facilitates effective communication with the public about the potential dangers of consuming contaminated products. Quantifying the risks allows public health policy makers to design targeted resources and control interventions.

The model groups all components in the natural environment in one compartment because they act as similar reservoirs for pathogens. This is attributed to several reasons, including exposure to the same contaminators and environmental conditions (temperature, water run-off, and pH) that influence the growth, survival, decay and spread of *Campylobacter* bacteria, contrary to animal products which act as separate reservoirs due to exposure to varying environmental conditions [6]. Furthermore, although water treatments and environmental cleanliness can be suggested, particularly for the contaminated environment, interventions like vaccination and organic acid disinfectants can be suggested to target the reduction of bacteria in animal products.

The adoption of a system of ODEs relies on its ability to capture the intricate non-linear complexity that underlies the dynamics of campylobacteriosis in interactive hosts [23]. Additionally, a proposed system of ODEs can be extended to other model types, such as stochastic models, through the addition of random variables. Furthermore, the results of a proposed model are easier to understand because the cause–effect relationships between the dependent and independent variables are clearly presented.

This work presents several novel features spanning the modelling, understanding, and control of campylobacteriosis. The study places a strong emphasis on meticulous model validation through data fitting and parameter estimation that guarantees the reliability of the model's prediction. Differentiating contaminated animal products and the natural environmental sources is essential for effective modelling, risk assessment, and management of zoonotic and water- and food-borne infections. A more accurate understanding of risk factors, transmission mechanisms, and the efficacy of alternative control methods is made possible by this difference. Through this study, setting intervention priorities can be made easier because the cause–effect relations between parameters, dependent variables and independent variables are clearly presented. The incorporation of environmental cleanliness as a mitigation strategy allows stakeholders to identify environmental cleanliness thresholds that can reduce campylobacteriosis cases.

The study will directly support the global Sustainable Development Goals (SDGs), including the African Union's Agenda 2063, which focuses on having citizens who are healthy and nourished. The research will help nations to achieve better public health outcomes by offering targeted and evidence-based strategies to lessen the prevalence of campylobacteriosis. The study will provide plans to improve food security and safety, which are essential to maintaining the population in good health and able to support economic growth. Furthermore, this study focuses on science, technology and innovation-driven development, one of the SDGs. The application of an innovative modelling approach to a critical health issue shows how innovatively this research may tackle challenging public health problems around the world. Furthermore, research findings will add to the existing body of knowledge, paving the way for future scientific research.

This paper is organised as follows: Section 1 contains the introduction. Specifically, the background of the disease, the motivation behind this study, and its novelty are discussed in this section. Section 2 contains the methods and results. In particular, a new mathematical model for campylobacteriosis transmission in humans and animals is developed. The effective reproduction number is computed and characterised in respect to disease persistence and extinction. The developed model is validated through data fitting

using the mean of human cases in the European Union (EU) from January to August in 2017 to 2020. The adoption of EU data is due to the availability of comprehensive, high-quality surveillance data, standardised across member states, which facilitates robust model validation. The model parameters are also estimated using the least squares algorithm alongside the EU data. Furthermore, the effects of contaminated animal products, environment, and environmental cleanliness are simulated alongside the variation in the effective reproduction number using contour plots. The discussions and conclusions are included in Section 3.

Methods and results

In this section, we develop and analyse a mechanistic mathematical model for the transmission dynamics of campylobacteriosis in human and cattle populations using a system of ordinary differential equations. In order to assess the impacts of contaminated animal products, environmental contamination, and environmental cleanliness and identify threshold values that increase infection cases, the effective reproduction number is simulated in response to the parameters that are responsible for the addition and removal of *Campylobacter* jejuni bacteria in the environment, animal products, and host populations.

Model formulation

The campylobacteriosis epidemic framework is made up of two host populations: humans and cattle. It also considers the colony-forming units (CFU) of *Campylobacter* jejuni bacteria in the environment and animal products. Moreover, we assume that (a) recruitments rates of susceptible humans and cattle through immigration and births are constants as these recruitment routes do not depend on the total populations in the community, (b) cattle are infected via bacterial ingestion from the contaminated environment and physical contact among diary cattle (licking the skins of infected cattle and breastfeeding), (c) humans are infected via bacterial ingestion from the contaminated environment, contaminated animal products and physical contact among humans, (d) exposed compartments are neglected because of short incubation period of campylobacteriosis in the host populations, (e) infected hosts shed the bacteria in the environment through excrements, (f) the environmental bacteria grow logistically following the available environmental resources, (g) contaminated animal products are harvested from infected cattle, and (h) the natural growth rate of bacteria in animal products is neglected because these products are immediately consumed by humans after being produced.

Throughout the paper, the human population is represented with the subscript h and cattle with d. The model apportions humans into three groups, namely, susceptible S_h, infectious I_h, and recovered R_h, and the cattle population into susceptible S_d, infectious I_d, and recovered R_d. Thus the total human and cattle populations are, respectively, $N_h = S_h + I_h + R_h$ and $N_d = S_d + I_d + R_d$.

Susceptible humans and cattle are recruited at the rates Λ_h and Λ_d, respectively. Susceptible humans become infected through the consumption of contaminated animal products (milk and meat), contaminated water, fruits, vegetables and salads, and licking unsanitary hands after touching dippers, contaminated surfaces and utensils at the rates β_h, β_m, or β_p. The parameters β_h, β_m, and β_p are the transmission rates from human-to-human, environment-to-human, and animal products-to-human, respectively.

On the other hand, susceptible cattle acquire the infection through breastfeeding, the consumption of contaminated water and grasses, and licking the skins of infected animals and soil at the rates θ_d or θ_m. The parameters θ_d and θ_m are, respectively, the transmission rates from cattle-to-cattle and environment-to-cattle. The infectious humans and cattle recover naturally at the rates of α_h and α_d or die due to the disease at the rates κ_h and κ_d, respectively. The recovered humans and cattle may lose immunity and become vulnerable again at the rates r_h and r_d, respectively. Humans and cattle may die naturally at the rates μ_h and μ_d, respectively.

The variable M is the CFUs of *Campylobacter* jejuni bacteria in the environment shed by the infectious humans and cattle at the rates of λ_h and λ_d, respectively. The CFUs of *Campylobacter* jejuni bacteria in environment M replicate at a rate of α_m in the environment with a carrying capacity of K. The CFUs of *Campylobacter* jejuni bacteria in environment M decrease through natural decay and environmental cleanliness at the rates of μ_m and ω, respectively.

The variable P stands for the contaminated animal products that increase at a rate of η through milking and slaughtering of infected cattle. Animal products P decay at a rate of μ_p. The descriptions of state variables and model parameters are presented in Table 1, while the flow diagram is represented in Fig. 1.

The spread of *Campylobacter* jejuni bacteria in the environment, animal products, and host populations, and the dynamics of campylobacteriosis among the host populations, are summarised as a system of ordinary differential Eqs. (1).

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + r_h R_h - (\beta_h I_h + \beta_m M + \beta_p P + \mu_h) S_h,\\
\frac{dI_h}{dt} &= (\beta_h I_h + \beta_m M + \beta_p P) S_h - (\alpha_h + \mu_h + \kappa_h) I_h,\\
\frac{dR_h}{dt} &= \alpha_h I_h - (r_h + \mu_h) R_h,\\
\frac{dS_d}{dt} &= \Lambda_d + r_d R_d - (\theta_d I_d + \theta_m M + \mu_d) S_d,\\
\frac{dI_d}{dt} &= (\theta_d I_d + \theta_m M) S_d - (\alpha_d + \mu_d + \kappa_d) I d,\\
\frac{dR_d}{dt} &= \alpha_d I_d - (r_d + \mu_d) R_d,\\
\frac{dM}{dt} &= \alpha_m M \left(1 - \frac{M}{K}\right) + \lambda_h I_h + \lambda_d I_d - (\mu_m + \omega) M,\\
\frac{dP}{dt} &= \eta I_d - \mu_p P,
\end{aligned}\right\} \tag{1}$$

Table 1
Description of state variables and model parameters.

Symbols	Description
S_h and S_d	Respectively, the number of humans and cattle that are not infected but are capable of contracting the disease.
I_h and I_d	Respectively, the number of humans and cattle that are infected and capable of transmitting the disease and shedding bacteria.
R_h and R_h	Respectively, the number of humans and cattle that are recovered from the disease.
M and P	Number of colony-forming units (CFUs) of *Campylobacter* jejuni bacteria in the environment and animal products, respectively.
Λ_h and Λ_d	Recruitment rates of susceptible humans and cattle, respectively.
λ_h and λ_d	Humans and cattle shedding rates, respectively.
r_h and r_d	Rates of losing immunity for recovered humans and cattle, respectively.
α_h and α_d	Recovery rates of infected humans and cattle, respectively.
μ_h and μ_d	Natural mortality rates of humans and cattle, respectively.
μ_m	The decay rate of *Campylobacter* jejuni bacteria in the environment.
μ_p	Decay rate of contaminated animal products.
κ_h and κ_d	Induced death rates for infected humans and cattle, respectively.
β_h and θ_d	Human-to-human and cattle-to-cattle transmission rates.
β_m and θ_m	Environment-to-human and environment-to-cattle transmission rates, respectively.
K	Environmental carrying capacity.
η	Harvesting rate of contaminated animal products.
β_p	Animal products-to-human transmission rate.
α_m	Environmental bacteria replication rate.
ω	Environmental cleanliness rate.

with the initial conditions:

$S_h(0) > 0,\ I_h(0) \geq 0,\ R_h(0) \geq 0,\ S_d(0) > 0,\ I_d(0) \geq 0,\ R_d \geq 0,\ M(0) \geq 0$ and $P(0) \geq 0$.

The invariant region, also known as the feasible region or a set of constraints within which the proposed model operates and remains valid, is denoted by

$$\Gamma = \left(S_h(t), I_h(t), R_h(t), S_d(t), I_d(t), R_d(t), M(t), P(t)\right) \in \mathfrak{R}^8_+.$$

Boundaries for the state variables, parameters, and assumptions of the proposed model are also established in this region, providing a substantial understanding of campylobacteriosis dynamics.

Characterisation of model's behaviour

This section examines the model's behaviour through the assessment of positivity and boundedness of the model solution, characterisation of the effective reproduction number, validation of the model through data fitting, stability analysis of equilibrium states, and the assessment of uncertainty and global sensitivity of model parameters against state variables.

Positivity of model solutions

Since the model (1) represents the populations of humans, cattle, and Campylobacter jejuni bacteria, it is crucial to verify that its solutions are positive and well-posed/bounded.

Theorem 1. *For any time $t \geq 0$, the model's solutions (1) are positive if their initial values are non-negative.*

Proof. Consider the ODE for susceptible humans S_h in the model system (1):

$$\frac{dS_h(t)}{dt} = \Lambda_h + r_h R_h - (\beta_h I_h(t) + \beta_m M(t) + \beta_p P(t) + \mu_h) S_h(t),$$

and observe that

$$\frac{dS_h(t)}{dt} \geq -(\beta_h I_h(t) + \beta_m M(t) + \beta_p P(t) + \mu_h) S_h(t). \tag{2}$$

By solving the differential inequality (2), we get

$$S_h(t) \geq S_h(0) e^{-\int_0^t (\beta_h I_h(t) + \beta_m M(t) + \beta_p P(t) + \mu_h) ds} \geq 0.$$

Following the same process, we get

$$I_h(t) \geq I_h(0) e^{-(\alpha_h + \mu_h + \kappa_h)t} \geq 0,$$
$$R_h(t) \geq R_h(0) e^{-(r_h + \mu_h)t} \geq 0,$$
$$S_d(t) \geq S_d(0) e^{-\int_0^t (\theta_d I_d + \theta_m M + \mu_d) ds} \geq 0,$$

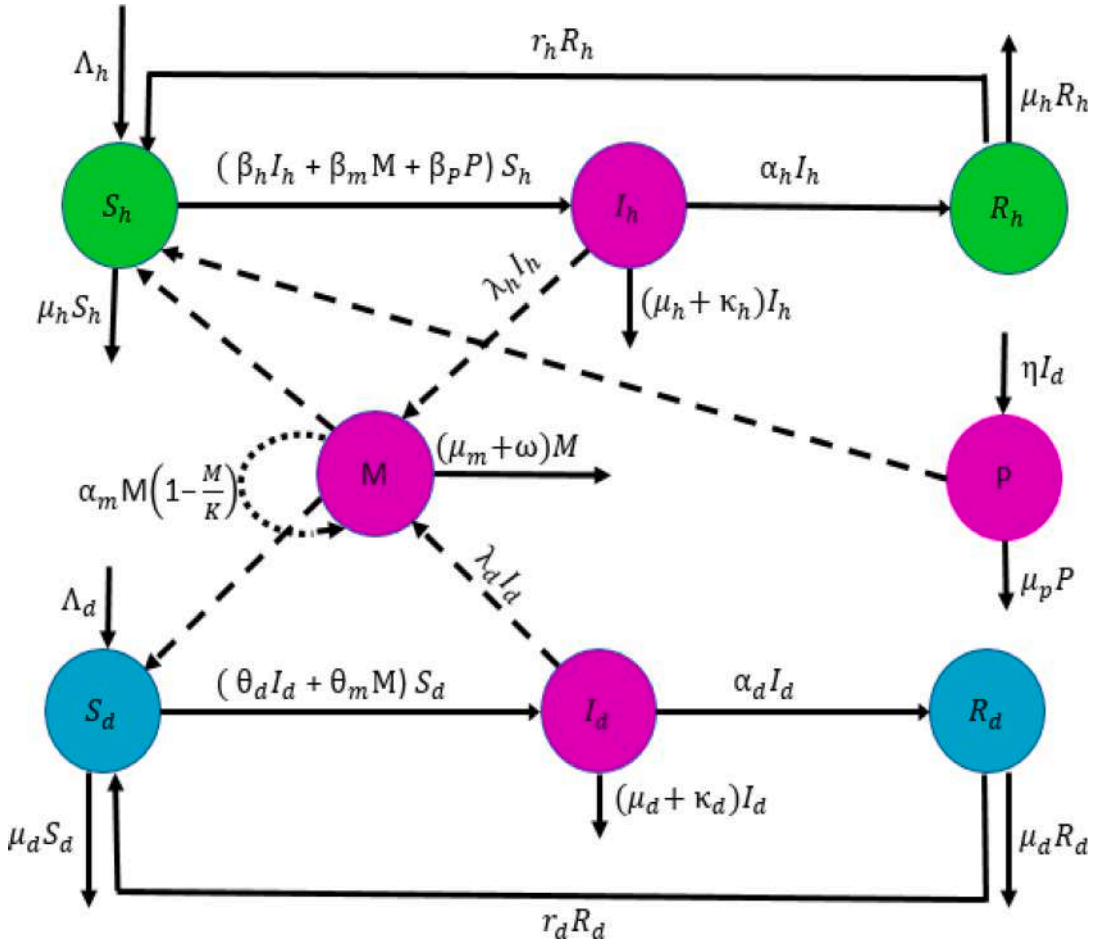

Fig. 1. The flow diagram for the transmission of *Campylobacter* jejuni bacteria and campylobacteriosis disease among human and dairy cattle populations.

$$I_d(t) \geq I_d(0)e^{-(\alpha_d+\mu_d+\kappa_d)t} \geq 0,$$
$$R_d(t) \geq R_d(0)e^{-(r_d+\mu_d)t} \geq 0,$$
$$M(t) \geq M(0)e^{-(\mu_m t+\omega)} \geq 0, \quad \text{and}$$
$$P(t) \geq P(0)e^{-\mu_p t} \geq 0,$$

meaning that all model solutions of the system (1) are positive at any time $t \geq 0$. □

Boundedness of the model

To characterise the boundedness of the model, we consider the total rate of change of human population:

$$N_h'(t) = \Lambda_h - \mu_h N_h(t) - \kappa_h I_h(t),$$

and note that

$$N_h'(t) \leq \Lambda_h - \mu_h N_h(t). \tag{3}$$

Using separation of variables method for the differential inequality (3) we obtain

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} + (N_h(0) - \frac{\Lambda_h}{\mu_h})e^{-\mu_h t}. \tag{4}$$

As $t \to \infty$, inequality (4) reduces to

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h}.$$

Because human population is non negative, we have

$$0 \le N_h(t) \le \frac{\Lambda_h}{\mu_h}.$$

When we follow the same steps for the population of cattle, we get $0 \le N_d(t) \le \frac{\Lambda_d}{\mu_d}$.

From $N_h(t) \le \frac{\Lambda_h}{\mu_h}$ and $N_d(t) \le \frac{\Lambda_d}{\mu_d}$ we get,

$I_h \le \frac{\Lambda_h}{\mu_h}$ and $I_d \le \frac{\Lambda_d}{\mu_d}$, respectively.

The differential inequality for the number of *Campylobacter* jejuni bacteria in animal products is solved without losing generality, and the result is

$$P(t) \le \frac{\eta \Lambda_d}{\mu_d \mu_p}.$$

Similarly, the rate of change in the number of *Campylobacter* jejuni bacteria in the contaminated environment produces

$$\frac{dM}{dt} \le \frac{\lambda_h \Lambda_h}{\mu_h} + \frac{\lambda_d \Lambda_d}{\mu_d} - (\mu_m - \alpha_m)M - \frac{\alpha_m}{K}M^2.$$

Consequently, there is a $\Pi > 0$ such that $\limsup_{t\to\infty} M(t) \le \Pi$. One can choose the constant Π as the unique positive root of the quadratic equation

$$\frac{\lambda_h \Lambda_h}{\mu_h} + \frac{\lambda_d \Lambda_d}{\mu_d} - (\mu_m - \alpha_m)y - \frac{\alpha_m}{K}y^2 = 0.$$

Consequently, the model's solution falls into the positive invariant region τ. Moreover, the model solutions that begin at the zone of the region $\Gamma \in \mathbb{R}^8_+$ enter that region in an infinite time. The solutions of the developed model are therefore bounded on the positive region $\Gamma \in \mathbb{R}^8_+$. Thus, the developed model in this instance is meaningful both mathematically and biologically, and we consider it for further analysis.

The disease free equilibrium E_0

The disease-free equilibrium E_0 is a condition where campylobacteriosis infection does not persist in human and cattle populations. Upon setting the right-hand side of the system (1) to zero and setting $I_h = I_d = M = P = 0$, we obtain

$$E_0 = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_d}{\mu_d}, 0, 0, 0, 0\right). \tag{5}$$

From the technical perspective, E_0 is used to compute the effective reproduction number.

The effective reproduction number $\mathcal{R}$

The effective reproduction number $\mathcal{R}$ is a threshold quantity for the persistence and extinction of campylobacteriosis in a community. The effective reproduction number $\mathcal{R}$ is defined as the expected number of secondary cases produced by one infectious human or cattle during its infectious lifetime in a community implementing campylobacteriosis mitigation strategies, including environmental cleanliness.

Adopting the next-generation matrix procedures in [26–29], we compute the effective reproduction number of the model as follows (1). Let the infectious state variables of the system (1) be

$$\frac{d\mathbf{X}}{dt} = \mathcal{F}_i(x) - \mathcal{V}_i(x),$$

with

$$\mathcal{F}_i(x) = \begin{bmatrix} (\beta_h I_h + \beta_m M + \beta_p P)S_h \\ (\theta_d I_d + \theta_m M)S_d \\ 0 \\ 0 \end{bmatrix} \text{ and} \tag{6}$$

$$\mathcal{V}_i(x) = \begin{bmatrix} (\alpha_h + \mu_h + \kappa_h)I_h \\ (\alpha_d + \mu_d + \kappa_d)I_d \\ -\alpha_m M(1 - \frac{M}{K}) - \alpha_h I_h - \alpha_d I_d + (\mu_m + \omega)M \\ -\eta I_d + \mu_p P \end{bmatrix}. \tag{7}$$

The vectors $\mathcal{F}_i(x)$ and $\mathcal{V}_i(x)$ represent the generation of new infections in compartment i and the transfer of infections from compartment i to other compartments, respectively. The first entry $(\beta_h I_h + \beta_m M + \beta_p P)S_h$ in $\mathcal{F}_i(x)$ represents the number of new

infected humans generated as a result of infected humans-to-susceptible humans, contaminated environment-to-susceptible humans, and contaminated animal products-to-susceptible humans transmission, respectively, while the entry $(\theta_d I_d + \theta_m M)S_d$ represents dairy cattle infection cases resulting from the infected cattle-to-susceptible dairy cattle and contaminated environment-to-dairy cattle transmission, respectively. On the other hand, vector $\mathcal{V}_i(x)$ is composed of two sub-vectors $\mathcal{V}_i(x)^+$ and $\mathcal{V}_i(x)^-$, which represent the transfer of terms into and out of compartment i, respectively. Mathematically, the vector

$$\mathcal{V}_i(x) = \mathcal{V}_i(x)^- - \mathcal{V}_i(x)^+.$$

Expressing the entries in vectors $\mathcal{F}_i(x)$ and $\mathcal{V}_i(x)$ as the derivatives with respect to I_h, I_d, M and P, we obtain the Jacobian matrices evaluated at E_0, given by

$$F = \begin{bmatrix} \frac{\beta_h \Lambda_h}{\mu_h} & 0 & \frac{\beta_m \Lambda_h}{\mu_h} & \frac{\beta_p \Lambda_h}{\mu_h} \\ 0 & \frac{\theta_d \Lambda_d}{\mu_d} & \frac{\theta_m \Lambda_d}{\mu_d} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{ and} \tag{8}$$

$$V = \begin{bmatrix} \alpha_h + \mu_h + \kappa_h & 0 & 0 & 0 \\ 0 & \alpha_d + \mu_d + \kappa_d & 0 & 0 \\ -\lambda_h & -\lambda_d & \mu_m + \omega - \alpha_m & 0 \\ 0 & -\eta & 0 & \mu_p \end{bmatrix}. \tag{9}$$

The element f_{ij} of a 4×4 matrix F represents the rate at which contaminated settings, humans and cattle in infected state j give rise to or generate new infections in humans or cattle in infected state i in the linearised system.

The inverse V^{-1} of matrix V is

$$\begin{bmatrix} \frac{1}{\alpha_h + \mu_h + \kappa_h} & 0 & 0 & 0 \\ 0 & \frac{1}{\alpha_d + \mu_d + \kappa_d} & 0 & 0 \\ \frac{\lambda_h}{(\alpha_h + \mu_h + \kappa_h)(\mu_m + \omega - \alpha_m)} & \frac{\lambda_d}{(\alpha_d + \mu_d + \kappa_d)(\mu_m + \omega - \alpha_m)} & \frac{1}{\mu_m + \omega - \alpha_m} & 0 \\ 0 & \frac{\eta}{(\alpha_d + \mu_d + \kappa_d)\mu_p} & 0 & \frac{1}{\mu_p} \end{bmatrix}, \tag{10}$$

with an assumption that $\mu_m + \omega > \alpha_m$, for a non-negative matrix V^{-1}. The entries $\frac{1}{\alpha_h + \mu_h + \kappa_h}$ and $\frac{1}{\alpha_d + \mu_d + \kappa_d}$ of V^{-1}, are the average duration an infected human and cattle spends in an infectious condition, respectively. The entries $\frac{\lambda_h}{(\alpha_h + \mu_h + \kappa_h)(\mu_m + \omega - \alpha_m)}$ and $\frac{\lambda_d}{(\alpha_d + \mu_d + \kappa_d)(\mu_m + \omega - \alpha_m)}$ are the average life span for the *Campylobacter* jejuni bacteria shed by infected humans and cattle in the environment, respectively. The entry $\frac{1}{\mu_m + \omega - \alpha_m}$ is the average time, the *Campylobacter* jejuni bacteria in the environment spends during their life time. On the other hand, the entries $\frac{\eta}{(\alpha_d + \mu_d + \kappa_d)\mu_p}$ and $\frac{1}{\mu_p}$ are, respectively, the average life span for the *Campylobacter* jejuni bacteria in an infected cattle and contaminated environment.

The next generation matrix is given by

$$M = FV^{-1} = \begin{bmatrix} R_1 & R_2 & R_3 & R_4 \\ R_5 & R_6 & R_7 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

where

$$R_1 = \frac{\beta_h \Lambda_h}{\mu_h(\alpha_h + \mu_h + \kappa_h)} + \frac{\beta_m \Lambda_h \lambda_h}{\mu_h(\alpha_h + \mu_h + \kappa_h)(\mu_m + \omega - \alpha_m)},$$

$$R_2 = \frac{\beta_m \Lambda_h \lambda_d}{\mu_h(\alpha_d + \mu_d + \kappa_d)(\mu_m + \omega - \alpha_m)} + \frac{\beta_p \Lambda_h \eta}{\mu_h(\alpha_d + \mu_d + \kappa_d)\mu_p},$$

$$R_3 = \frac{\beta_m \Lambda_h}{\mu_h(\mu_m + \omega - \alpha_m)}, \quad R_4 = \frac{\beta_p \Lambda_h}{\mu_h \mu_p}, \quad R_5 = \frac{\theta_m \Lambda_d \lambda_h}{\mu_d(\alpha_h + \mu_h + \kappa_h)(\mu_m + \omega - \alpha_m)},$$

$$R_6 = \frac{\theta_d \Lambda_d}{\mu_d(\alpha_d + \mu_d + \kappa_d)} + \frac{\theta_m \Lambda_d \lambda_d}{\mu_d(\alpha_d + \mu_d + \kappa_d)(\mu_m + \omega - \alpha_m)}, \text{ and } R_7 = \frac{\theta_m \Lambda_d}{\mu_d(\mu_m + \omega - \alpha_m)}.$$

Since $\mathcal{R}$ is the spectral radius of M, then

$$\mathcal{R} = \frac{(R_1 + R_6) + \sqrt{(R_1 - R_6)^2 + 4R_2R_5}}{2}. \tag{11}$$

The term R_1 in (11) is the secondary number of infected humans produced by one infected human as a result of direct contact between susceptible and infected humans and consuming *Campylobacter* jejuni bacteria shed by an infected human in the environment. R_2 is the secondary number of infected humans produced by an infected cow as a result of consuming *Campylobacter* jejuni bacteria shed by an infected cow and consuming the *Campylobacter* jejuni bacteria found in the animal products. R_5 is the secondary number of infected cattle that is produced by consuming *Campylobacter* jejuni bacteria shed by the infected human, whereas R_6 is the secondary number of infected cattle produced by one infected cattle as a result of direct contact between susceptible and infected cattle and consuming *Campylobacter* jejuni bacteria shed by an infected cattle in the environment.

Global stability of disease-free equilibrium E_0

If the disease-free equilibrium E_0 remains in its state after a perturbation is introduced into the system, it is considered stable. If campylobacteriosis does not persist over an extended period of time, regardless of the size of the perturbation introduced into the system, the disease-free equilibrium E_0 is considered globally stable.

In this section, we utilise the Lyapunov stability approach [23] to assess whether a disease-free system reverts to its state after the introduction of infected humans, infected cattle and the bacteria. A positive definite function, known as a Lyapunov function, that satisfies stability conditions is adopted. A positive definite function produces positive values evaluated at the system's trajectories except at the disease-free equilibrium, where it produces zero value. Furthermore, its derivative along the system's trajectories must be non-positive, indicating a gradual reduction and convergence to the disease-free equilibrium.

Theorem 2. *The disease-free equilibrium is globally asymptotically stable in the entire invariant domain Γ, if and only if $\mathcal{R} < 1$.*

Proof. Let $A = \begin{bmatrix} I_h(t) \\ I_d(t) \\ M(t) \\ P(t) \end{bmatrix}$. Motivated by Mhlanga [30] and Nyabadza et al. [31] we have the comparison principle, such that

$$\dot{A} \leq (F - V)A, \tag{12}$$

where matrices F and V are described in Eqs. (8) and (9), respectively, and the vector $\dot{A} = (I_h', I_d', M', P')^{\mathrm{T}}$ contains the rate of change of infected humans, infected cattle, and the CFUs of bacteria in the environment and animal products, respectively.

The off-diagonal elements of matrices F (8) and V^{-1} (10) are non-negative, implying that the two matrices are Metzler matrices. By the Perron–Frobenius theorem for Metzler matrices [32], $V^{-1}F$ has a dominant eigenvalue.

$$\mathcal{R} = \rho(V^{-1}F) = \rho(FV^{-1}),$$

corresponding to a Perron–Frobenius vector $\mathbf{b}$ whose entries are non-negative. Consequently,

$$\mathbf{b}^{\mathrm{T}}V^{-1}F = \mathcal{R}\mathbf{b}^{\mathrm{T}}. \tag{13}$$

We adopt a candidate Lyapunov function by Mhlanga [30], such that

$$\chi(t) = \mathbf{b}^{\mathrm{T}}V^{-1}A. \tag{14}$$

Differentiation of Eq. (14) with respect to time t, alongside the entries in vector $\dot{A}$ and the inequality (12), we obtain

$$\dot{\chi} = \mathbf{b}^{\mathrm{T}}V^{-1}\dot{A} \leq \mathbf{b}^{\mathrm{T}}V^{-1}(F - V)A = (\mathcal{R} - 1)\mathbf{b}^{\mathrm{T}}A, \text{ where } \mathbf{b}^{\mathrm{T}}A \geq 0.$$

For Lyapunov stability, $\dot{\chi} = (\mathcal{R} - 1)\mathbf{b}^{\mathrm{T}}A \leq 0$, and is attained only when $\mathcal{R} < 1$.

If $\dot{\chi} = 0$ for $\mathcal{R} < 1$, then $\mathbf{b}^{\mathrm{T}}A = 0$. Since $\mathbf{b}$ is a Perron–Frobenius vector, then $A = (I_h, I_d, M, P) = (0, 0, 0, 0)$. Substituting $I_h = I_d = M = P = 0$ into the steady state solutions of the system (1), we get $E_0 = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_d}{\mu_d}, 0, 0, 0, 0\right)$. For $\dot{\chi} < 0$, the infected and contaminated hosts $A = (I_h, I_d, M, P)$ lose their transmission energy; thus, the system converges to $(0, 0, 0, 0)$ as time t approaches infinity [6]. In general, when $\mathcal{R} < 1$, every trajectory in the domain Γ reverts to E_0 as time t approaches infinity. Therefore, the disease-free equilibrium E_0 is globally asymptotically stable whenever $\mathcal{R} < 1$. □

Endemic equilibrium E_1

The endemic equilibrium $E_1 = (S_h^*, I_h^*, R_h^*, S_d^*, I_d^*, R_d^*, M^*, P^*)$ of the system (1) occurs when campylobacteriosis persists in the community. During this state, the number of infected and recovered populations exist in the community. The entries of E_1 are the steady-state solutions of the system (1).

Theorem 3. *Endemic equilibrium E_1 is globally asymptotically stable whenever $\mathcal{R} > 1$.*

Proof. We adopt a Lyapunov function [33], given by

$$\begin{aligned} L &= \left[S_h - S_h^* + S_h^* \ln\left(\frac{S_h^*}{S_h}\right)\right] + \left[I_h - I_h^* + I_h^* \ln\left(\frac{I_h^*}{I_h}\right)\right] + \frac{r_h R_h^*}{\alpha_h I_h^*}\left[R_h - R_h^* + R_h^* \ln\left(\frac{R_h^*}{R_h}\right)\right] \\ &+ \left[S_d - S_d^* + S_d^* \ln\left(\frac{S_d^*}{S_d}\right)\right] + \left[I_d - I_d^* + I_d^* \ln\left(\frac{I_d^*}{I_d}\right)\right] + \frac{r_d R_d^*}{\alpha_d I_d^*}\left[R_d - R_d^* + R_d^* \ln\left(\frac{R_d^*}{R_d}\right)\right] \\ &+ \left[M - M^* + M^* \ln\left(\frac{M^*}{M}\right)\right] + \left[P - P^* + P^* \ln\left(\frac{P^*}{P}\right)\right]. \end{aligned} \tag{15}$$

Differentiating L with respect to time t, we obtain

$$\begin{aligned} \frac{dL}{dt} &= \left(1 - \frac{S_h^*}{S_h}\right)\frac{dS_h}{dt} + \left(1 - \frac{I_h^*}{I_h}\right)\frac{dI_h}{dt} + \frac{r_h R_h^*}{\alpha_h I_h^*}\left(1 - \frac{R_h^*}{R_h}\right)\frac{dR_h}{dt} + \left(1 - \frac{S_d^*}{S_d}\right)\frac{dS_d}{dt} \\ &+ \left(1 - \frac{I_d^*}{I_d}\right)\frac{dI_d}{dt} + \frac{r_d R_d^*}{\alpha_d I_d^*}\left(1 - \frac{R_d^*}{R_d}\right)\frac{dR_d}{dt} + \left(1 - \frac{M^*}{M}\right)\frac{dM}{dt} + \left(1 - \frac{P^*}{P}\right)\frac{dP}{dt}. \end{aligned} \tag{16}$$

From system (1) we let $\vartheta_h = \beta_h I_h + \beta_m M + \beta_p P$ and $\vartheta_d = \theta_d I_d + \theta_m M$.

At the steady state of the model system (1) we obtain

$$\begin{aligned} &\Lambda_h = (\vartheta_h^* + \mu_h)S_h^* - r_h R_h^*,\ \alpha_h + \mu_h + \kappa_h = \frac{\vartheta_h^* S_h^*}{I_h^*},\ r_h + \mu_h = \frac{\alpha_h I_h^*}{R_h^*},\ r_d + \mu_d = \frac{\alpha_d I_d^*}{R_d^*}, \\ &\Lambda_d = (\vartheta_d^* + \mu_d)S_d^* - r_d R_d^*,\ \mu_d + \omega = \alpha_m(1 - \frac{M^*}{K}) + \frac{\lambda_h I_h^*}{M^*} + \frac{\lambda_d I_d^*}{M^*},\ \mu_p = \frac{\eta I_d^*}{P^*}, \\ &\text{and}\ \ \alpha_d + \mu_d + \kappa_d = \frac{\vartheta_d^* S_d^*}{I_d^*}. \end{aligned} \tag{17}$$

Substitute the expressions (17) and the model system (1) into equation Eq. (16), we obtain

$$\begin{aligned} \frac{dL}{dt} &= -\left[\left(1 - \tfrac{1}{s}\right)^2 + \left(1 - \tfrac{1}{z}\right)^2 + \tfrac{\lambda_m}{K}(\tau M^* - M^*)^2\right] + \vartheta_h^* S_h^*\left[2 + y - \tfrac{1}{s} - n - \tfrac{ys}{n}\right] \\ &+ \vartheta_d^* S_d^*\left[2 + d - \tfrac{1}{z} - \phi - \tfrac{dz}{\phi}\right] + \lambda_h I_h^*\left[1 + n - \tau - \tfrac{n}{\tau}\right] + r_d R_d^*\left[\tfrac{1}{z} - \tfrac{h}{z} + \phi - \tfrac{\phi}{h}\right] \\ &+ r_h R_h^*\left[\tfrac{1}{s} - \tfrac{e}{s} + n - \tfrac{n}{e}\right], \end{aligned} \tag{18}$$

where

$$y = \frac{\vartheta_h}{\vartheta_h^*},\ d = \frac{\vartheta_d}{\vartheta_d^*},\ s = \frac{S_h}{S_h^*},\ n = \frac{I_h}{I_h^*},\ e = \frac{R_h}{R_h^*},\ z = \frac{S_d}{S_d^*},\ \phi = \frac{I_d}{I_d^*},\ h = \frac{R_d}{R_d^*},\ \tau = \frac{M}{M^*},\ \text{and}\ p = \frac{P}{P^*}.$$

Definition 1. If $x \in \mathfrak{R}_+$, then $1 - x \le -\ln x$.

It follows that,

$$\begin{aligned} 2 + y - \tfrac{1}{s} - n - \tfrac{ys}{n} &= (1 - \tfrac{1}{s}) + (1 - n) + (1 - \tfrac{ys}{n}) - (1 - y) \\ &\le -\ln\left(\tfrac{1}{s}\right) - \ln(n) - \ln\left(\tfrac{ys}{n}\right) + \ln(y) = 0. \end{aligned} \tag{19}$$

By the same procedures we obtain

$$2 + d - \frac{1}{z} - \phi - \frac{dz}{\phi} \le 0,\ 1 + n - \tau - \frac{n}{\tau} \le 0,\ \frac{1}{z} - \frac{h}{z} + \phi - \frac{\phi}{h} \le 0,\ \text{and}\ \frac{1}{s} - \frac{e}{s} + n - \frac{n}{e} \le 0.$$

Since $-\left[\left(1 - \frac{1}{s}\right)^2 + \left(1 - \frac{1}{z}\right)^2 + \frac{\lambda_m}{K}(\tau M^* - M^*)^2\right] \le 0$, then, $\frac{dL}{dt} \le 0$.

The case when $\frac{dL}{dt} = 0$ implies the system attains its equilibrium state. This state is achieved when $\vartheta_h = \vartheta_h^*$, $\vartheta_d = \vartheta_d^*$, $S_h = S_h^*$, $I_h = I_h^*$, $R_h = R_h^*$, $S_d = S_d^*$, $I_d = I_d^*$, $R_d = R_d^*$, $M = M^*$, and $P = P^*$, or when $y = d = s = n = e = z = \phi = h = \tau = p = 1$. In this case, E_1 is observed to be the biggest compact invariant set in Γ. In the case when $\frac{dL}{dt} < 0$, it means that all solutions in the invariant region Γ converge to E_1 as time t approaches infinity. By La Salle's invariant principle [34], an endemic equilibrium E_1 exists when $\mathcal{R} > 1$ and by the Lyapunov stability, it is globally asymptotically stable. □

Subsection 1.1 in the supplementary material presents examples of numerical simulations of system (1) using the effective reproduction number $\mathcal{R}$ to support the analytical results in Theorems 2 and 3.

Model validation and parameter estimation

In this paper, we estimate model parameters using the least squares approach [23]. We used the mean of human cases from January to August for the years 2017 to 2020 in the EU [12] to estimate parameters and fit the model. The sum of the squared residuals, $SSR = \sum_{i=1}^{8}(M_i - Q_i)^2$, between the reported human cases M_i and model solutions Q_i is minimised using the Nelder–Mead simplex algorithm [35] to estimate the parameter values. A thorough overview of model fitting is given in Fig. 2, and the estimated parameter values are listed in Table 2.

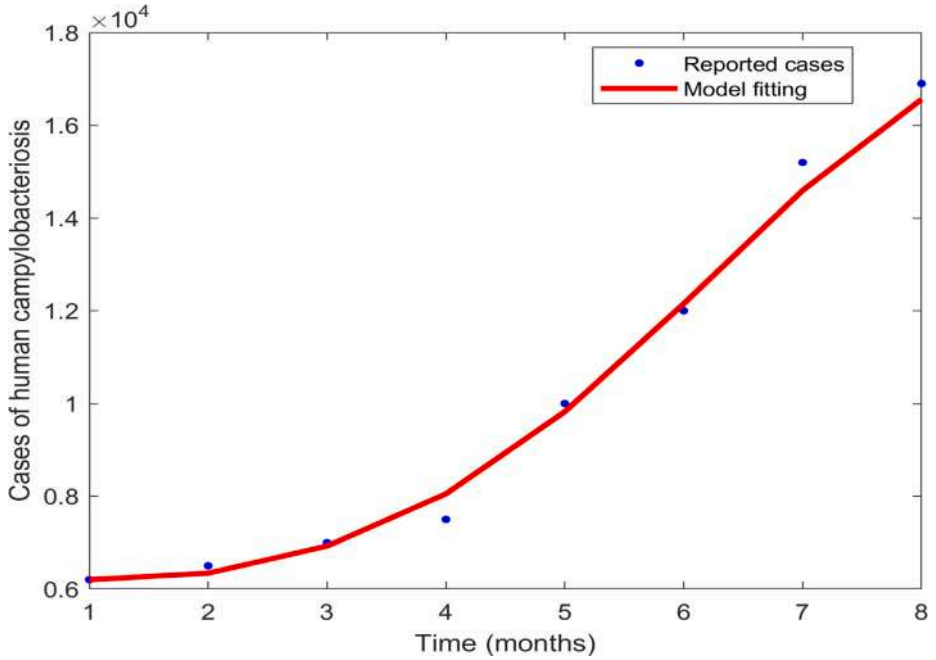

Fig. 2. Model fitting using the mean of human cases from January to August for the years 2017 to 2020 in the EU.

Table 2
Units of model parameters, baseline values and estimates for the model system (1).

Parameters	Units	Baseline value	Source	Estimates
Λ_h	humans d^{-1}	168	Assumed	167
Λ_d	cattle d^{-1}	30	Assumed	30
λ_h	(CFU/ml) human^{-1} d^{-1}	$(1-10^{10})$	[1,3]	999
λ_d	(CFU/ml) cow^{-1} d^{-1}	10^8	[1,3]	5.099×10^7
r_h	d^{-1}	0.001	[1,3]	0.0023
r_d	d^{-1}	0.007	[1,3]	0.007
α_h	d^{-1}	0.065	[1,3]	0.049
α_d	d^{-1}	0.05	[1,3]	0.017
μ_h	d^{-1}	$1/(70\times365)$	[1,3]	3.8138×10^{-5}
μ_d	d^{-1}	0.03	[1,3]	0.0096
μ_m	d^{-1}	0.14	[1,3]	0.0845
μ_p	d^{-1}	0.14	[1,3]	0.1399
η	cow^{-1} d^{-1}	0.64	[1,3]	0.6400
κ_h	d^{-1}	0.004	Assumed	0.0008
κ_d	d^{-1}	0.005	Assumed	0.0049
K	CFU/ml	10 000	Assumed	19 999
β_h	human^{-1} d^{-1}	2.1×10^{-5}	[1]	1.0110×10^{-5}
β_m	(CFU/ml)$^{-1}$ d^{-1}	$1.034177\times10^{(-13)}$	[1]	1.9003×10^{-13}
β_p	(CFU/ml)$^{-1}$ d^{-1}	$(0-0.2050)$	[1]	19107×10^{-10}
θ_m	(CFU/ml)$^{-1}$ d^{-1}	1.3×10^{-11}	[1]	1×10^{-11}
θ_d	(CFU/ml)$^{-1}$ d^{-1}	0.004	Assumed	9.379×10^{-4}
α_m	d^{-1}	0.0002	Assumed	1.8471×10^{-4}
ω	–	0.6	[1]	0.6

Uncertainty and sensitivity analysis

Parameters that influence the spread of diseases play a crucial role in how accurately mathematical models can forecast the behaviour of infectious diseases within populations [6]. However, uncertainties in these parameter values arise from factors such as insufficient data, variations in environmental conditions, disparities in immunological conditions among host populations, and the imposed assumptions when formulating a mathematical model [6,36]. Incorporating uncertainty analysis into disease modelling enhances the reliability of model predictions. Conversely, sensitivity analysis identifies parameters that significantly affect variability in model predictions [36]. The methods of Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficient (PRCC) are utilised to examine uncertainty and global sensitivity of model parameters. The LHS technique generates samples of model parameters, thus addressing parameter uncertainty by treating them as probabilistic parameters that are uniformly distributed across a parameter range. The Partial Rank Correlation Coefficient (PRCC) method is employed to determine and measure the strength

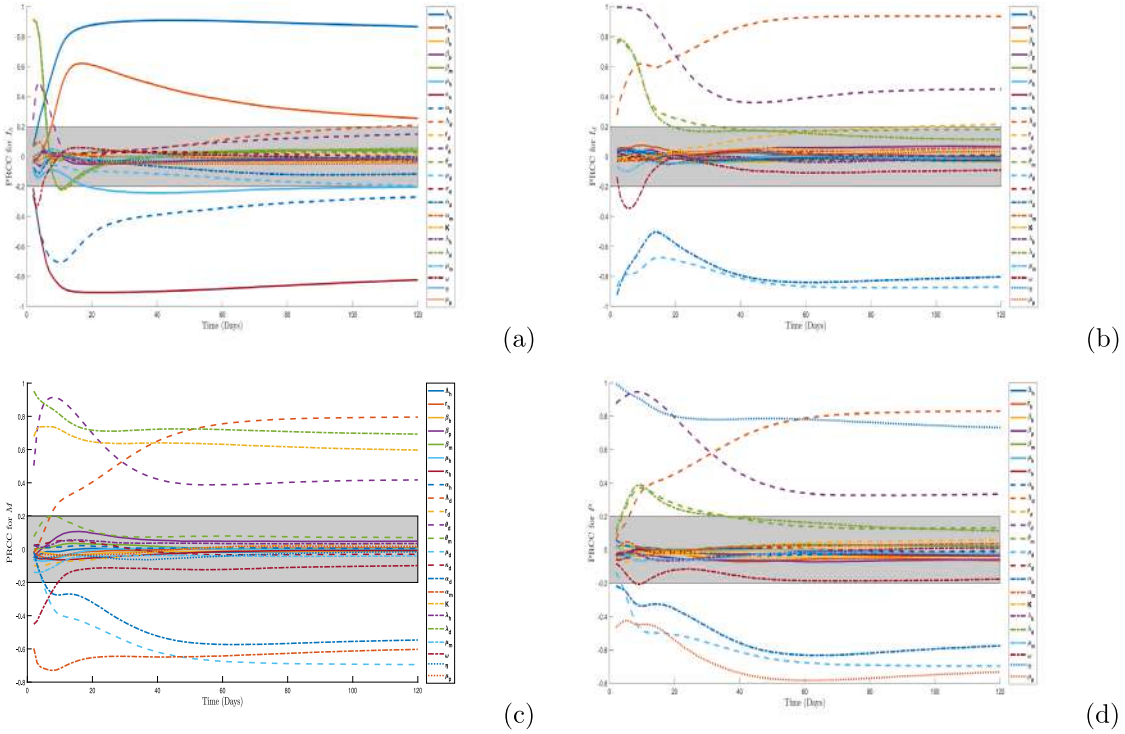

Fig. 3. The sensitivity analysis of the model outputs: (a) populations of infected humans, (b) infected cattle, (c) *Campylobacter* jejuni bacteria in the environment, and (d) *Campylobacter* jejuni bacteria in animal products.

of the correlation between model inputs and model outputs [37]. If X_i is a sampled input parameter, Z_i is the model output corresponding to X_i, and $\overline{X}$ is the mean of sampled input parameters, $X = (X_i)$, and $\overline{Z}$ is the mean of model outputs $Z = (Z_i)$ for $i = 1, 2, \ldots, N$, then the PRCC value r of X_i and Z_i is given by:

$$r = \frac{\mathrm{Cov}(X_i, Z_i)}{\sqrt{\mathrm{Var}(X_i)\mathrm{Var}(Z_i)}} = \frac{\sum_{i=1}^{N}(X_i - \overline{X})(Z_i - \overline{Z})}{\sqrt{\sum_{i=1}^{N}(X_i - \overline{X})^2 \sum_{i=1}^{N}(Z_i - \overline{Z})^2}}.$$

The Partial Rank Correlation Coefficients (PRCCs) range between −1 and +1. The magnitude of the PRCC value indicates the strength of the correlation, with values closer to −1 or 1 signifying a stronger relationship between X_i and Z_i. When the absolute value of PRCC is below 0.2, it reflects a weaker correlation [38]. A positive PRCC value suggests a direct relationship, meaning that as the input parameter increases, the output state variable also rises proportionally. Sensitivity indices that vary over time are computed across a defined time frame and displayed as a function of time. This approach is essential for evaluating the importance of input parameters in a model during specific intervals of its dynamical behaviour.

Sub-figure (a) in Fig. 3 shows the PRCCs between model parameters and infected humans. The results show that the human shedding rate Λ_h and the loss of immunity rate r_h are significantly positively correlated with infected humans I_h from the beginning of the outbreak to the end. These parameters increase campylobacteriosis cases in the human population as they increase. On the other hand, the induced disease rate κ_h and the recovery rate α_h are significantly negatively correlated with infected humans I_h from the beginning of the outbreak to the end. This means that they decrease campylobacteriosis cases whenever they increase. Additionally, the environment-to-human transmission rate β_m, cattle shedding rate λ_d, and cattle-to-cattle transmission rate θ_d are significantly positively correlated with infected humans I_h within the first 10 days, whereas the human natural death rate μ_h is strongly negatively correlated with I_h after the 20th day of the outbreak. Parameters with positive PRCC values should be reduced within the observed sensitivity period, while those with negative PRCC values should be increased in order to reduce the number of human cases.

In sub-figure (b) in Fig. 3, cattle-to-cattle transmission rate θ_d and cattle recruitment rate Λ_d, and cattle's shedding rate λ_d and the natural death rate of cattle μ_d, are, respectively, strongly positively and negatively correlated with infected cattle I_d throughout the outbreak. On the other hand, λ_d and θ_m are significantly positively correlated with I_d within the first 40 days of the outbreak, whereas the environmental cleanliness rate ω is negatively correlated with I_d within the first 10 days. The aforementioned parameters should be regulated accordingly in order to reduce the number of infected cattle.

In sub-figure (c) in Fig. 3, shedding rates λ_d, Λ_d, cattle-to-cattle transmission rate θ_d, and environment-to-cattle transmission rate θ_m are positively correlated with the CFU of *Campylobacter* jejuni bacteria in the environment M. However, the aforementioned

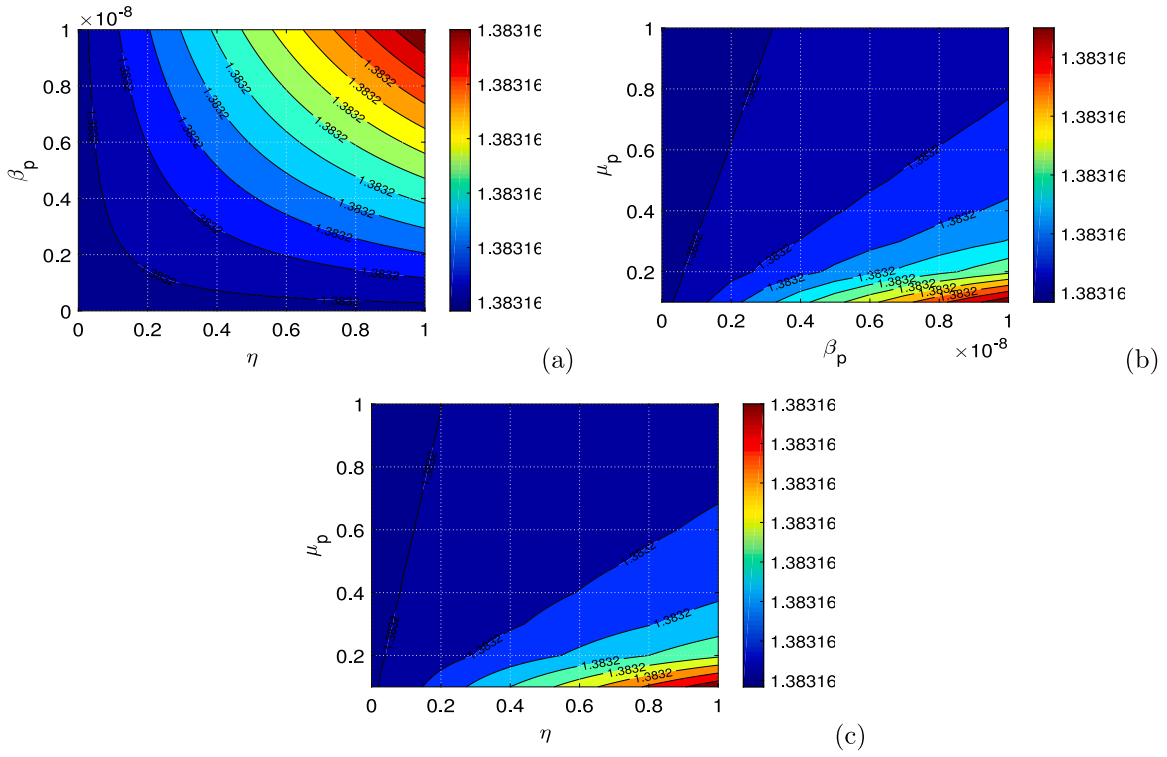

Fig. 4. The dynamics of $\mathcal{R}$ in relation to risk parameters associated with contaminated animal products.

parameters drive M throughout the entire period; θ_m drives M within the first 25 days of the outbreak. The aforementioned parameters should be reduced accordingly using appropriate mitigation strategies in order to reduce the CFU of *Campylobacter* jejuni bacteria in the environment and the associated infection cases. Conversely, cleanliness ω, decay rates of *Campylobacter* jejuni bacteria μ_d, μ_m, and the recovery rate of infected cattle α_d are inversely proportional to M throughout the entire period. This suggests that ω, μ_d, μ_m, and α_d should be increased proportionately in order to reduce the CFU of *Campylobacter* jejuni bacteria in the environment and the associated infection cases.

In sub-figure (d) in Fig. 3, the harvesting rate of contaminated animal products η, environmental shedding rates Λ_d, λ_d, and cattle-to-cattle transmission rate θ_d are directly proportional to the CFU of *Campylobacter* jejuni bacteria in animal products P. Out of these, η, Λ_d, and θ_d influence P for the entire dynamical period, whereas Λ_d does for the first 40 days. The aforementioned parameters should be reduced accordingly using appropriate control measures in order to reduce the CFU of *Campylobacter* jejuni bacteria in animal products. On the other hand, decay rates of *Campylobacter* jejuni bacteria μ_p, μ_d, and the recovery rate of infected cattle α_d are significantly negatively correlated with P. The aforementioned parameters should be increased using appropriate strategies in order to reduce the CFU of *Campylobacter* jejuni bacteria and the associated cases in humans.

We also assess the effect of parameters that are responsible for environmental contamination, harvesting rate of contaminated animal products, transmission of *Campylobacter* jejuni bacteria from contaminated environments and contaminated animal products to humans and cattle populations, and removal of *Campylobacter* jejuni bacteria in the environment and animal products by the aid of scatter plots (see subsection 1.2 in the supplementary material). Subsection 1.3 in the supplementary material examines the effects of environmental contamination on campylobacteriosis disease dynamics by means of contour plots. Knowing these impacts enables the creation and assessment of efficient intervention plans that target contaminants at its source and along the entire food production chain, thereby lowering exposure and disease incidence in humans and cattle.

Effects of contaminated animal products

To investigate the effects of contaminated animal products on campylobacteriosis disease, $\mathcal{R}$ is simulated against the harvesting rate of contaminated animal products η, the consumption rate of contaminated animal products β_p, and the decay rate of contaminated animal products μ_p, as shown in sub-figures (a)–(c) in Fig. 4. The results indicate that η and β_p are directly proportional to $\mathcal{R}$, implying that increasing their associated rates proportionately increases the value of $\mathcal{R}$. The results suggest that the reduction of the contamination rate and the consumption rate of contaminated animal products could not reduce the reproduction number $\mathcal{R}$ below a unit. On the other hand, μ_p is inversely proportional to $\mathcal{R}$. The results in Fig. 4 indicate that increasing the decay rates of *Campylobacter* jejuni bacteria in animal products does not reduce $\mathcal{R}$ below a unit. Implying that strategies that aim at killing bacteria

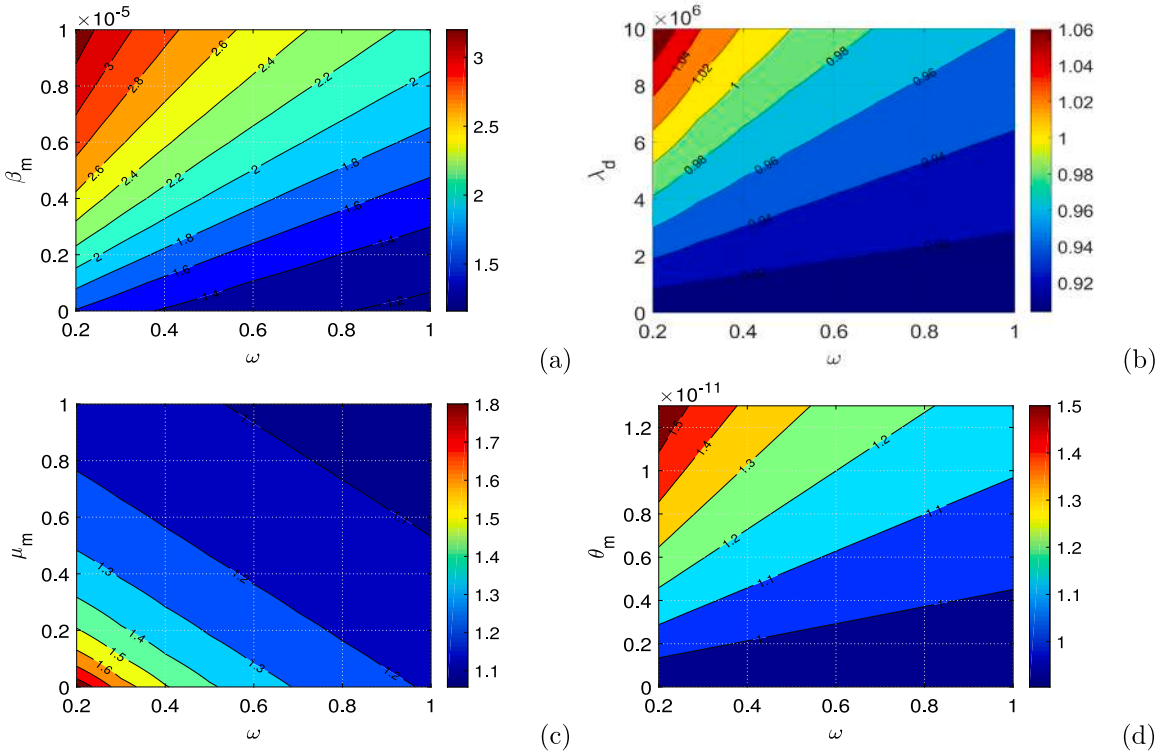

Fig. 5. The coupled effect of cleanliness and environmental risk parameters on the dynamics of $\mathcal{R}$.

in animal products are not sufficient to eliminate the disease when they are implemented alone. Generally, strategies that target other transmission routes other than contaminated animal products are needed in order to eliminate campylobacteriosis cases.

Effects of environmental cleanliness

Sub-figures (a)–(d) in Fig. 5 show how $\mathcal{R}$ varies in response to variation in environmental cleanliness rate ω alongside the parameters responsible for environmental contamination and environment-to-host transmission. The results highlight the effect of environmental cleanliness as a decontamination effort in the contaminated environment, underscoring the urgent need to increase its metrics in order to reduce the CFU of *Campylobacter* bacteria in the environment and the associated campylobacteriosis cases in humans and cattle.

The results in sub-figure (a) in Fig. 5 indicate that $\mathcal{R}$ decreases due to the increase and decrease in the environmental cleanliness and environment-to-host transmission rates, respectively. However, there is no cleanliness threshold that can reduce $\mathcal{R}$ below a unit, implying that cleanliness is not an effective strategy to be implemented alone in areas with environment-to-host transmission. This suggests that a couple of mitigation strategies are needed to reduce the number of infected cases.

The results in sub-figures (b) and (d) in Fig. 5 indicate that a threshold $\mathcal{R}$ below 1 is achieved when the shedding rate λ_d and environment-to-cattle transmission rate decrease below thresholds 5×10^6 and 0.1, respectively, for all cleanliness thresholds. The results in sub-figures (b) and (d) in Fig. 5 suggest that campylobacteriosis cases in humans and cattle can be sufficiently eliminated through reducing shedding rates and the implementation of control measures that reduce the environment-to-host transmission below the aforementioned thresholds.

The results in sub-figures (c) in Fig. 5 indicate that an increase in the cleanliness thresholds alongside the natural decay rates of the environmental bacteria does not reduce $\mathcal{R}$ below a unit. The results suggest that the strategies aimed at killing and washing away environmental bacteria are not sufficient to eliminate campylobacteriosis cases in humans and cattle populations when they are implemented alone. A couple of mitigation strategies that span from killing bacteria to reducing the environment-to-host transmissions and shedding rates are needed to control the disease.

Conclusions

In this paper, a mathematical model for the transmission dynamics of campylobacteriosis in human and cattle populations with different infectious statuses is formulated and analysed. The model contains CFU of *Campylobacter* jejuni bacteria in the contaminated environment and animal products. The inclusion of these components makes our study more comprehensive in eliminating the

campylobacteriosis life cycle in interacting populations compared to isolated studies that considered one population [1,25]. The model in this study is well posed since all model solutions are positive and bounded, making it mathematically and biologically valid. The effective reproduction number $\mathcal{R}$ was computed using the next generation matrix method. The global stability of equilibrium states was performed using the Lyapunov stability theory alongside the Lyapunov functions, Perron–Frobenius theorems, and the effective reproduction number. The model was validated, and parameters were estimated using the human cases in the EU.

The assessment of the impacts of contaminated animal products, environmental contamination, and the application of environmental cleanliness as a mitigation strategy makes this study unique compared to the existing studies [1,2,25]. This study has comprehensively examined and quantified the risk levels associated with environmental contamination and consumption of contaminated dairy products. The threshold values for the environmental cleanliness that can reduce campylobacteriosis cases were identified.

The results show that the campylobacteriosis cases among humans and cattle populations increase due to an increase in parameters responsible for environmental contamination and consumption of contaminated animal products and other foods from the contaminated environment. On the other hand, the disease decreases due to an increase in the parameters that are responsible for the removal of bacteria in the environment and animal products.

The results indicated that the environment-to-human transmission rate β_m, cattle shedding rate λ_d, and cattle-to-cattle transmission rate θ_d are significantly positively correlated with infected humans I_h within the first 10 days. The results suggest that the aforementioned parameters should be regulated within the first 10 days of the outbreak to reduce human cases.

The environment-to-cattle transmission rate θ_m and the cattle's shedding rate λ_d are strongly positively correlated with infected cattle I_d within the first 40 days of the outbreak and throughout the outbreak, respectively. Environmental cleanliness rate ω is negatively correlated with I_d within the first 10 days. The aforementioned parameters should be regulated accordingly in order to reduce the number of infection cases in the cattle population.

Shedding rates λ_d, Λ_d, cattle-to-cattle transmission rate θ_d, and environment-to-cattle transmission rate θ_m are positively correlated with the CFU of *Campylobacter* jejuni bacteria in the environment M. Shedding rate drives M throughout the entire period, while θ_m drives M during the first 25 days of the outbreak. The aforementioned parameters should be reduced within the observed sensitivity periods using appropriate intervention methods in order to reduce environmental contamination and the associated environmental bacteria and infection cases. In contrast, the results indicated that cleanliness ω, decay rates of *Campylobacter* jejuni bacteria μ_d, μ_m, and the recovery rate of infected cattle α_d are inversely proportional to M throughout the entire period. The results suggest that ω, μ_d, μ_m, and α_d should be increased proportionately in order to reduce the environmental bacteria and the associated infection cases.

The results show that reducing the environment-to-host and contaminated animal products-to-humans transmission can reduce the $\mathcal{R}$ below a unit. This suggests that in order to eliminate campylobacteriosis in humans and cattle populations, a couple of control strategies spanning from targeting the aforementioned transmission routes to killing bacteria in the environment and animal products are recommended. Furthermore, the results suggest that campylobacteriosis cases in humans and cattle can be sufficiently eliminated through reducing shedding rates and the implementation of control measures that reduce the environment-to-host transmission below the thresholds 5×10^6 and 0.1, respectively, for all cleanliness thresholds. Furthermore, the results suggest that the strategies aimed at killing and washing away environmental bacteria are not sufficient to eliminate campylobacteriosis cases in humans and cattle populations when they are implemented alone. A couple of mitigation strategies that span from killing bacteria to reducing the environment-to-host transmissions and shedding rates are needed to control the disease.

Since this study is not 100 per cent comprehensive, the proposed model might be modified by including more variables that influence campylobacteriosis dynamics. Environmental conditions and the accompanying seasonality trends have an impact on the development, decay, and survival rates of *Campylobacter* jejuni bacteria. An optimum control strategy is also required because the elimination of campylobacteriosis necessitates the application of multiple control measures associated with costly resources. Future research can also employ backward bifurcation theory to determine whether endemic and disease-free equilibria coexist, an attempt that will help public health policy-makers design an effective control intervention. A more reliable mathematical epidemiology model that might help describe the dynamics and control of campylobacteriosis could be developed by taking into consideration each of these factors.

CRediT authorship contribution statement

Herman Trazias: Conceptualisation, Formal analysis, Parameter estimation, Writing – original draft, Writing – review & editing. **Eva Lusekelo:** Conceptualisation, Methodology (analysis and simulation), Writing – original draft, Writing – review & editing, Submitting the final draft of the manuscript. **Kasim Sakran Abass:** Making insightful suggestions on Methodology, drafting, reviewing, and editing the manuscript.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Trazias Herman reports administrative support and writing assistance were provided by Mbeya University of Science and Technology, University of Dodoma and University of Kirkuk. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary material

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