The Impact of Vaccination on the Dynamics of Paratuberculosis Transmission in Guinea.

Abstract

Paratuberculosis, a chronic infectious disease that primarily affects ruminants, poses a major health and economic challenge for livestock systems in many developing countries,particularly in Guinea. This study analyzes the impact of vaccination on the transmission dynamics of this disease within herds, using a mathematical modeling approach inspired by epidemiological tools.

A compartmental model adapted to paratuberculosis is formulated to describe the evolution of susceptible, infected, and vaccinated animal populations. The basic reproductionnumber, denoted $\mathcal{R}_0$, is determined for the model without intervention, then compared to an effective reproduction number$\mathcal{R}_0^V$ that incorporates vaccination coverage and vaccine efficacy.

The results show that vaccination leads to a significant reduction in the disease's transmission potential, reflected in a decrease from$\mathcal{R}_0$ to $\mathcal{R}_0^V$. The analysis highlights the existence of a critical vaccination threshold beyond which the disease can no longer be sustained in theanimal population. However, the effectiveness of this strategy depends heavily on the level of vaccination coverage, husbandry conditions, and local epidemiological parameters.

Key words. Johne's disease, Mathematical model, Vaccination impact.

1. Introduction

Paratuberculosis, also known as Johne's disease, is a chronic infection of ruminants caused by Mycobacterium avium subsp. paratuberculosis (MAP). This disease is characterized by progressive granulomatous enteritis leading to chronic diarrhea, weight loss, and, in advanced cases, the death of the animal. It primarily affects cattle, sheep, and goats and is currently one of the most concerning infectious diseases for livestock worldwide [1, 2].

The pathogen is transmitted primarily via the fecal-oral route, particularly in young animals that ingest contaminated material from infected animals. Infected animals can excrete large quantities of bacteria in their feces, thereby contributing to environmental contamination and the spread of infection within herds [3]. This transmission dynamic promotes the establishment of a persistent infection that is difficult to eradicate.

From an economic standpoint, paratuberculosis poses a significant threat to livestock systems. It leads to reduced milk production, decreased fertility, and an increase in the rate of premature culling of infected animals. In several countries, the economic losses associated with this disease

are estimated at several million dollars annually in the cattle industry [4, 5]. In response to this problem, various control strategies have been proposed, including improved biosecurity measures, screening programs and the culling of infected animals, as well as vaccination.

Among these approaches, vaccination appears to be a promising tool for reducing the prevalence of the disease and limiting its progression to clinical forms. Several studies have shown that vaccination can reduce bacterial shedding and slow the spread of infection within herds [6, 7]. In West African countries, and particularly in Guinea, ruminant livestock farming plays a vital role in food security and the livelihoods of rural populations. However, epidemiological data onparatuberculosis in this region remain limited, and control strategies are still underdeveloped. In this context, assessing the potential impact of vaccination on the disease's transmission dynamics is an important scientific and public health issue. In Guinea, it primarily affects areas with high livestock densities, particularly Middle Guinea and Upper Guinea, where it is facilitated by transhumance and the movement of animals. The disease is potentially present throughout the country due to the persistence of the bacteria in the environment ([8]). Areas of intensive grazing (Middle Guinea) and areas of extensive livestock farming (Upper Guinea) are the most at risk, with increased risks of transmission via water sources and pastures ([8]). Screening tests and culling are the standard methods for controlling paratuberculosis in Guinea. Disease eradication programs, which focus on postmortem inspection of meat, intensive surveillance including farm visits, systematic screening of cattle through individual testing and the culling of infected animals as well as those that have been in contact with them, and the control of animal movements, have yielded very satisfactory results in terms of reducing or eliminating the disease in Guinea ([8]).

The aim of this article is therefore to examine the impact of vaccination on the dynamics of paratuberculosis in ruminant populations in Guinea. Using a mathematical transmission model, we analyze the effect of different vaccination strategies on the spread of infection and on reducing the prevalence of the disease in herds.

2. Model formulation

This section describes the mathematical model proposed for paratuberculosis infection, taking vaccination into account. The population is divided into four compartments: susceptible $S(t)$, asymptomatically infected $I_A(t)$, symptomatically infected $I_S(t)$, and vaccinated $V(t)$. Therefore, the total population size at time t is:

$$N(t) = S(t) + I_A(t) + I_S(t) + V(t).$$

We define $G(t)$ as the concentration of Mycobacterium avium present in the environment (feces and contaminated soil) to represent the influence of the environment on disease transmission.

The variations in $S(t)$ depend on the recruitment of newborns into the population. We assume that at any time t, a constant number Λ of newborns is added to the population. The variation also depends on the vaccination of these newborns at a rate Π and on the additional vaccinationof susceptible individuals at a rate Π. Booster shots allow for the immunization of susceptible individuals who were not vaccinated at birth and for the reinforcement of immunity in those who have already received the vaccine. The susceptible population also varies due to

natural mortality, which occurs at a rate of μ. This reduces the number of susceptible individuals. The susceptible population decreases if susceptible individuals become infected. This is possible in only two ways: • directly, that is, through contact with a proportion of infected individuals. This is modeled by the infection rate:

$$\beta_1 \frac{I_A}{N},$$

where β_1 is the transmission rate between an infected individual and a susceptible individual.

• indirectly, that is, through contact with viruses in the environment. This phenomenon is modeled by the following saturation infection strength:

$$\beta_2 \frac{G}{G+K},$$

where β_2 is the transmission rate between a susceptible individual and viruses present in the environment. We define K as the amount of virus sufficient to give a 50% chance of becoming infected after contact with the viruses.

The infection rate is therefore given by:

$$\lambda(I_A, G) = \beta_1 \frac{I_A}{N} + \beta_2 \frac{G}{G+K}. \tag{2.1}$$

The variation of number of vaccinated individuals $V(t)$ depends on susceptible individuals who are vaccinated at birth and during additional vaccination campaigns at rates Π and θ, respectively. Vaccinated individuals become asymptomatic carriers at a rate of $q(1-v)\lambda(I_A,G)$. The various flows between the different states we are considering are illustrated in Figure (1). Consequently, the differential system obtained from this model is given by:

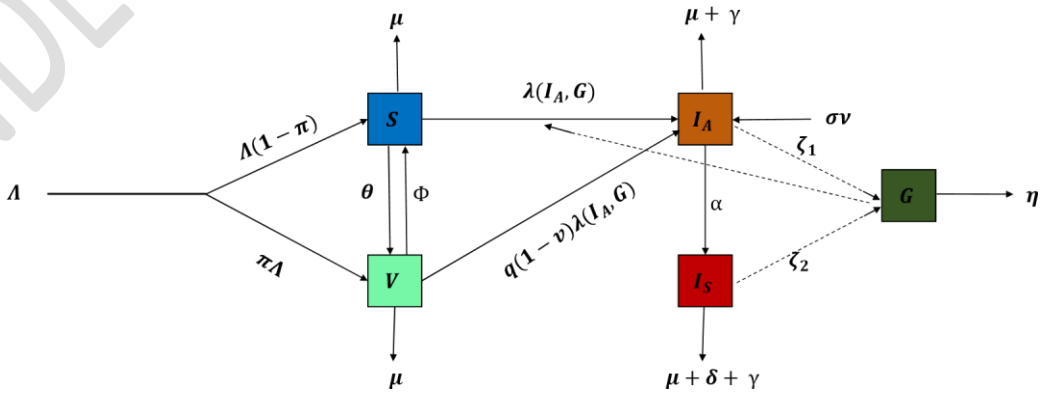

Figure 1: Diagram of the dynamics of paratuberculosis transmission with vaccination

$$\begin{cases} \dot{S} = (1-\Pi)\Lambda + \phi V - \lambda(I_A, G)S - (\theta + \mu)S, \\ \dot{V} = \Pi\Lambda + \theta S - \big(\mu + \phi + q(1-v)\lambda(I_A, G)\big)V, \\ \dot{I}_A = \big(S + q(1-v)V\big)\lambda(I_A, G) + \sigma\nu I_A - (\mu + \alpha + \gamma)I_A \\ \dot{I}_S = \alpha I_A - (\mu + \delta + \gamma)I_S, \\ \dot{G} = \zeta_1 I_A + \zeta_2 I_S - \eta G. \end{cases}, \quad (2.2)$$

3. Analytical results

3.1. General remarks

For model system (2.2) to be epidemiologically meaningful, it is important to prove that all its state variables are non-negative for all time t. We have the following result.

Lemma 3.1 *If the initial data* ($S(0), I_A(0), I_S(0), V(0), G(0)$) ≥ 0, *then the solutions* ($S(t), I_A(t), I_S(t), V(t), G(t)$) *of model system* (2.2) *are non-negative for all t* >0, *and the positive orthant* $\mathbb{R}^5_+$ *is positively invariant under the flow described by model system* (2.2). *Furthermore for any initial condition such that:*

$$N(0) \le \frac{\Lambda}{\mu - \sigma\nu} \text{ and } G(0) \le \frac{\Lambda(\zeta_1 + \zeta_2)}{\mu - \sigma\nu},$$

one has

$$N(t) \le \frac{\Lambda}{\mu - \sigma\nu} \text{ and } G(t) \le \frac{\Lambda(\zeta_1 + \zeta_2)}{\mu - \sigma\nu}, \ \forall\, t \ge 0,$$

with $\mu > \sigma\nu$.

We will now show that model (2.2) is mathematically and epidemiologically well-posed. In this respect, the following theorem is valid.

Theorem 3.2 *Model system* (2.2) *is a (dissipative) dynamical system in the positively invariant region:*

$$\Omega = \left\{ (S, I_A, I_S, G) \in \mathbb{R}^4_+, \ \ N(t) \le \frac{\Lambda}{\mu - \sigma\nu} \text{ and } G(t) \le \frac{\Lambda(\zeta_1 + \zeta_2)}{\eta(\mu - \sigma\nu)} \right\}.$$

Proof.

By setting $X(t) = (S(t), I_A(t), I_S(t), V(t), G(t))^T$ and

$$F(X(t)) = \begin{pmatrix} (1-\Pi)\Lambda + \phi V - \lambda(I_A, G)S - (\theta + \mu)S \\ \Pi\Lambda + \theta S - \big(\mu + \phi + q(1-v)\lambda(I_A, G)\big)V \\ \big(S + q(1-v)V\big)\lambda(I_A, G) + \sigma\nu I_A - (\mu + \alpha + \gamma)I_A \\ \alpha I_A - (\mu + \delta + \gamma)I_S \\ \zeta_1 I_A + \zeta_2 I_S - \eta G \end{pmatrix}$$

system (2.2) rewrite into the following Cauchy Problem:

$$\begin{cases} \dot{X}(t) &= F(X(t)) \\ X(t_0) &= X_0. \end{cases} \quad (3.1)$$

Since F is of class C^1, then F is locally lipschitzian. Therefore, by a classical results of the dynamical system, for any iniyial conditions (t_0,$S(0)$,$I_A(0)$,$I_S(0)$,$G(0)$) the Cauchy problem (3.1) admits a unique solution in $[0,T]$ with $T>0$.

This solution is always positive by Lemma (3.1). This solution globally exists in $\mathbb{R}^4_+$ if it is bounded.

Since the initial condition is any in Ω, then its invariance immediately follows.

By dividing the model system (2.2) into two parts, the ruminants population (i.e. $S(t)$, $I_A(t)$, $I_S(t)$) and the virus population in the environment (i.e. $G(t)$), and using the dynamics of the total ruminants population, we obtain:

$$\dot{N} = \Lambda - \mu N + \sigma\nu I_A - (\gamma + \delta) I_S, \leq \Lambda - (\mu - \sigma\nu) N.$$

Applying the Gronwall inequality to the above inequation yields

$$N(t) \leq \frac{\Lambda}{\mu - \sigma\nu} + \left(N(0) - \frac{\Lambda}{\mu - \sigma\nu} \right) \exp(-(\mu - \sigma\nu)t), \forall\, t \geq 0, \quad (3.2)$$

where $N(0)$ represents the initial value of $N(t)$. This implies that

$$0 \leq N(t) \leq \frac{\Lambda}{\mu - \sigma\nu} \quad \forall\, t \geq 0 \ \text{ if } \ N(0) \leq \frac{\Lambda}{\mu - \sigma\nu}.$$

Finally, using the fact that $I_A(t) \leq \frac{\Lambda}{\mu - \sigma\nu}$ and $I_S(t) \leq \frac{\Lambda}{\mu - \sigma\nu}$, the dynamics of the population of viruses satisfies

$$\dot{G} \leq \frac{\Lambda}{\mu - \sigma\nu} (\zeta_1 + \zeta_2) - \eta G. \quad (3.3)$$

Applying the Gronwall inequality to the above inequation we obtain

$$0 \leq G(t) \leq \frac{\Lambda}{\eta(\mu - \sigma\nu)} (\zeta_1 + \zeta_2) + \left(G(0) - \frac{\Lambda}{\eta(\mu - \sigma\nu)} (\zeta_1 + \zeta_2) \right) \exp(-\eta t),$$

where $G(0)$ represents the initial value of $G(t)$. It then follows that as $t \to \infty$, $G(t) \leq \frac{\Lambda(\zeta_1 + \zeta_2)}{\eta(\mu - \sigma\nu)}$,

whenever $G(0) \leq \frac{\Lambda(\zeta_1 + \zeta_2)}{\eta(\mu - \sigma\nu)}$. ■

Thus, Ω is a positively-invariant set under the flow described by model system (2.2). In this region, model system (2.2) is epidemiologically and mathematically well-posed. Now, the local existence

and the boundedness of a solution ensure the global existence (in $\mathbb{R}^4_+$) of a unique solution of any initial value problem of model system (2.2). Indeed, the right-hand side of model system (2.2) is a continuously differentiable map (C^1). Then, by the Cauchy Lipschitz theorem for any given solution $X_0 \in \mathbb{R}^4_+$, there exist a unique maximal solution, $\phi(t,X_0)$, to the Cauchy problem of the differential equation (2.2). This concludes the proof.

3.2 Basic reproduction number and the local stability of the diseasefree equilibrium

The basic reproduction number is a threshold that determines when an infection can establish itself and persist in a new host population. The basic reproduction number is defined as the average number of secondary infections produced by an infected individual during its entire infectious period when introduced into a host population where everyone is susceptible. The technique described in Vanden Driessche and Watmough [9] involves determining the next-generation matrix,

which allows us to determine the number of cases produced by an infectious individual in a population at equilibrium without disease. The basic reproduction number is then the spectral radius of the next-generation matrix. We will therefore apply this technique to our model.

In the absence of infection, that is $I_A = I_S = 0$, that implies $G = 0$, the system (2.2) has a disease-free equilibrium (DFE) given by $E^0 = (S^0, V^0, 0, 0, 0)$ with

$$S^0 = \frac{\Lambda\,[\mu(1-\Pi)+\phi]}{\mu(\mu+\theta+\phi)} \quad \text{and} \quad V^0 = \frac{\Lambda(\mu\Pi+\theta)}{\mu(\mu+\theta+\phi)}.$$

The linear stability of E^0, without delays, can be established using the next generation operator, see e.g. Diekmann et al. 1990 and Driessche et al. 2002, on the system (2.2). The vectors F and V for the new infection terms and the remaining transfer terms are such that for $x = (I_A, I_S, G)'$, the populations in the disease compartments, we can write

$$\dot{x} = \mathrm{F}(x) - \mathrm{V}(x),$$

where

$$\mathcal{F}(x) = \begin{pmatrix} \big(S + q(1-v)V\big)\lambda(I_A, G) + \sigma\nu I_A \\ 0 \\ 0 \end{pmatrix} \quad \text{and}$$

$$\mathcal{V}(x) = \begin{pmatrix} (\mu+\alpha+\gamma)I_A \\ -\alpha I_A + (\mu+\delta+\gamma)I_S \\ -\zeta_1 I_A - \zeta_2 I_S + \eta G \end{pmatrix}.$$

Their Jacobian matrices evaluated at E^0 are given by

$$F = \begin{pmatrix} S^0\frac{\beta_1}{N} + q(1-v)\frac{\beta_1}{N}V^0 + \sigma\nu & 0 & \frac{\beta_2}{K}S^0 + \frac{\beta_2 q(1-v)}{K}V^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{and}$$

$$V = \begin{pmatrix} \mu+\alpha+\gamma & 0 & 0 \\ -\alpha & \mu+\delta+\gamma & 0 \\ -\zeta_1 & -\zeta_2 & \eta \end{pmatrix}.$$

Then, the basic reproduction number, $\mathcal{R}_0^V$, of model (2.2) without delays is defined, following Diekmann et al. 1990 [10] and Driessche et al. 2002 [9], as the spectral radius of the next generationmatrix, FV^{-1} and is

$$\mathcal{R}_0^V = \frac{K\eta(\mu+\delta+\gamma)(S^0\frac{\beta_1}{N} + q(1-v)V^0\frac{\beta_1}{N} + \sigma\nu) + (\beta_2 S^0 + \beta_2 q(1-v)V^0)(\alpha\zeta_2 + \zeta_1(\mu+\delta+\gamma))}{K\eta(\mu+\alpha+\gamma)(\mu+\delta+\gamma)}. \quad (3.4)$$

The threshold quantity $\mathcal{R}_0^V$ measures the average number of new infections generated by a single infective in a completely susceptible population. From [9, 10], we have the following result.

Lemma 3.3 *[9, 10, Theorem 2]. The DFE, E^0, of model (2.2) is locally asymptotically stable (LAS) If $\mathcal{R}_0^V < 1$ and unstable if $\mathcal{R}_0^V > 1$.*

The biological implication of the above lemma is that introducing a small number of infected individuals into a disease-free population in a state of equilibrium does not lead to an epidemic as long as $\mathcal{R}_0^V$ <1. Otherwise, an epidemic is possible. The following result is valid

Theorem 3.4 *If $\mathcal{R}_0^V < 1$, then the disease-free equilibrium E^0, is locally asymptotically stable in* Ω.

3.3. Existence of the Endemic Equilibrium of Model (2.2)

In this section, we study the existence of equilibrium points other than the disease-free equilibrium, namely the limit equilibria and the possible internal equilibria. First, let us make a few useful remarks.

Suppose that an equilibrium is such that I_A = 0; then, by the fourth equationof the system (2.2)$I_S = 0$, and by the last equation, G = 0. Consequently, the equilibrium point in question is free of disease. Similarly, if an equilibrium is such that I_S = 0, then by the fourth equation of the system (2.2) I_A = 0 and by the last equation G = 0.Consequently, the equilibrium point in question is free of disease.Similarly, if an equilibrium is such that I_S = 0, then by the fourth equation of the system (2.2) I_A= 0 and by the last equation G = 0.Hence, the equilibrium point in question is disease-free.Conversely, if we assume that the animal population is disease-free, the free virus concentration$G$ = 0and by the last equation I_A = I_S = 0, once again, the corresponding equilibrium is diseasefree.Overall, the only limiting equilibrium point for the system (2.2) where the disease is absent in the animal population is the disease-free equilibrium. Consequently, we have proven the following result.

Lemma 3.5 *The system* (2.2) *has no limit equilibrium other than the disease-free equilibrium.*

This lemma is very important because it excludes the possibility that the complete model (2.2) could have non-trivial limit equilibria. This suggests that the complete model might have exactly one endemic equilibrium. This, together with the existence and uniqueness of an endemic equilibrium [11], motivates the following conjecture that we formulate.

Conjecture Suppose that $\mathcal{R}_0^V > 1$ for the system (2.2). Then there exists a unique endemic equilibrium.The stability of the endemic equilibrium will be illustrated numerically.

4. Impact of the vaccination on the transmission of Jone's disease

The analysis of disease dynamics is based on the basic reproduction number R_0, which measures the average number of new infections generated by a single infected individual in a fully susceptible population.

Vaccination reduces the proportion of susceptible animals and decreases the probability of disease transmission. This leads to a new reproduction number, called the reproduction numberwith vaccination$\mathcal{R}_0^V$.Studying R_0 and $\mathcal{R}_0^V$ makes it possible to:

- quantify the potential effectiveness of vaccination,
- determine the minimum vaccination threshold required to control the disease,
- evaluate different vaccination campaign scenarios,
- assist veterinary authorities in making health decisions tailored to the Guinean context.

Thus, analyzing the impact of vaccination by comparing R_0 and $\mathcal{R}_0^V$ is a relevant approach for understanding the dynamics of paratuberculosis in Guinea and for proposing effective control strategies on livestock farms.

Let's first study the model system (2.2) without vaccination. In the absence of vaccination, the model system (2.2) is as follows

$$\begin{cases} \dot{S} = \Lambda - \left(\beta_1 \frac{I_A}{N} + \beta_2 \frac{G}{G+K}\right) S - \mu S, \\ \dot{I}_A = \left(\beta_1 \frac{I_A}{N} + \beta_2 \frac{G}{G+K}\right) S + \sigma\nu I_A - (\mu+\alpha) I_A \\ \dot{I}_S = \alpha I_A - (\mu+\gamma+\delta) I_S, \\ \dot{G} = \zeta_1 I_A + \zeta_2 I_S - \eta G. \end{cases} , \quad (4.1)$$

In the absence of infection, that is $I_A = I_S = 0$, that implies $G = 0$, the system (2.2) has a disease-free equilibrium (DFE) given by $E^0 = (S^0,0,0,0)$ with$S^0 = \frac{\Lambda}{\mu}$.

The linear stability of E^0, without delays, can be established using the next generation operator, see e.g. Diekmann et al. 1990 and Driessche et al. 2002, on the system (2.2). The vectors F and V for the new infection terms and the remaining transfer terms are such that for $x = (I_A,I_S,G)'$, the populations in the disease compartments, we can write

$x^{\cdot}$ = F(x) −W(x),

where

$$\mathcal{F}(x) = \begin{pmatrix} \beta_1 \frac{I_A}{N} S + \sigma\nu I_A + \beta_2 \frac{G}{G+K} S \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{W}(x) = \begin{pmatrix} -(\mu+\alpha) I_A \\ \alpha I_A - (\mu+\delta+\gamma) I_S \\ \zeta_1 I_A + \zeta_2 I_S - \eta G \end{pmatrix}.$$

Their Jacobian matrices evaluated at E^0 are given by

$$F = \begin{pmatrix} \frac{\beta_1 \Lambda}{N\mu} + \sigma\nu & 0 & \frac{\beta_2 \Lambda}{\mu K} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad W = \begin{pmatrix} \mu+\alpha & 0 & 0 \\ -\alpha & \mu+\delta+\gamma & 0 \\ -\zeta_1 & -\zeta_2 & \eta \end{pmatrix}.$$

Then, the basic reproduction number, $\mathcal{R}_0$, of model (2.2) without delays is defined, following Diekmann et al. 1990 and Driessche et al. 2002, as the spectral radius of the next generation matrix, FW^{-1} and is

$$\mathcal{R}_0 = \frac{\beta_1 \Lambda + N\mu\sigma\nu}{N\mu(\mu+\alpha)} + \frac{\beta_2 \Lambda(\zeta_2\alpha + \zeta_1(\mu+\delta+\gamma))}{\mu K \eta(\mu+\alpha)(\mu+\delta+\gamma)}. \tag{4.2}$$

The threshold quantity $\mathcal{R}_0$ measures the average number of new infections generated by a single infective in a completely susceptible population.

To better understand the impact of vaccination on the dynamics of paratuberculosis, we compare the model with vaccination to the model without vaccination. This comparison must be madeat the initial stage by comparing the two basic reproduction numbers $\mathcal{R}_0^V$ and $\mathcal{R}_0$ of the two models (2.2) and (4.1). This will allow us to analyze the spread of the disease at the very beginning of the epidemic. It will highlight the fundamental role of vaccination as a strategy for controlling paratuberculosis. By reducing the number of secondary cases, vaccination acts as a mechanism for regulating epidemic dynamics and can, under certain conditions, lead to the elimination of the disease in the population.

From (3.4) and (4.2) we obtain

$$\mathcal{R}_0^V = \left(\frac{(1-\Pi)\mu}{\mu+\theta} + \frac{q(1-v)(\theta+\Pi\Lambda\mu)}{\Lambda(\mu+\theta)} \right) \left(\mathcal{R}_0 - \frac{\sigma\nu}{\mu+\alpha} \right) \tag{4.3}$$

Thus, from equation (4.3), the comparison at the initial stage of the disease can be easily derived:

$$\mathcal{R}_0^V < \mathcal{R}_0. \tag{4.4}$$

This shows that, when vaccination is in place, the average number of infected herds is lower than the average number of infected herds when vaccination is not in place. This demonstrates that the disease spreads more slowly when vaccination is in place.

5. Numerical simulations

Now, let us give numerical illustrations of our stability results. To do so, we give some examples of parameters for system (2.2)

Parameters	Parameters values
Λ	50
μ	0.01
β_1	0.3
β_2	0.2
ν	0.05
ζ_1	0.03
ζ_2	0.04
σ	0.08
η	0.1
α	0.07
δ	0.01
γ	0.02
Ψ	0.05

Table 1: Parameters values

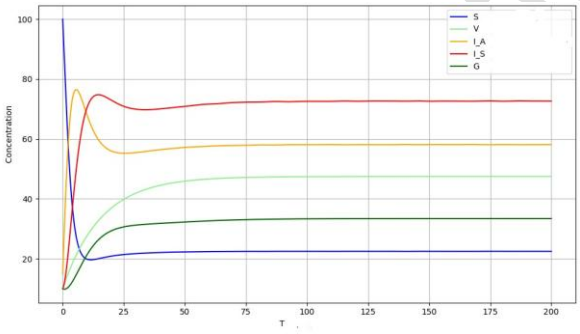

Figure 2: Trends with vaccination

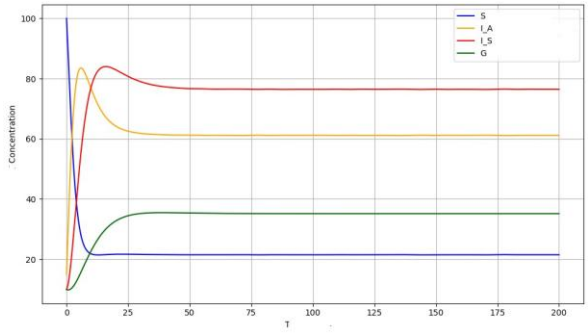

Figure 3: Trends with vaccination

We observe that vaccination is a way to reduce the spread of the disease within the herd. It acts simultaneously on several levels: it reduces asymptomatic reservoirs, decreases clinical cases,

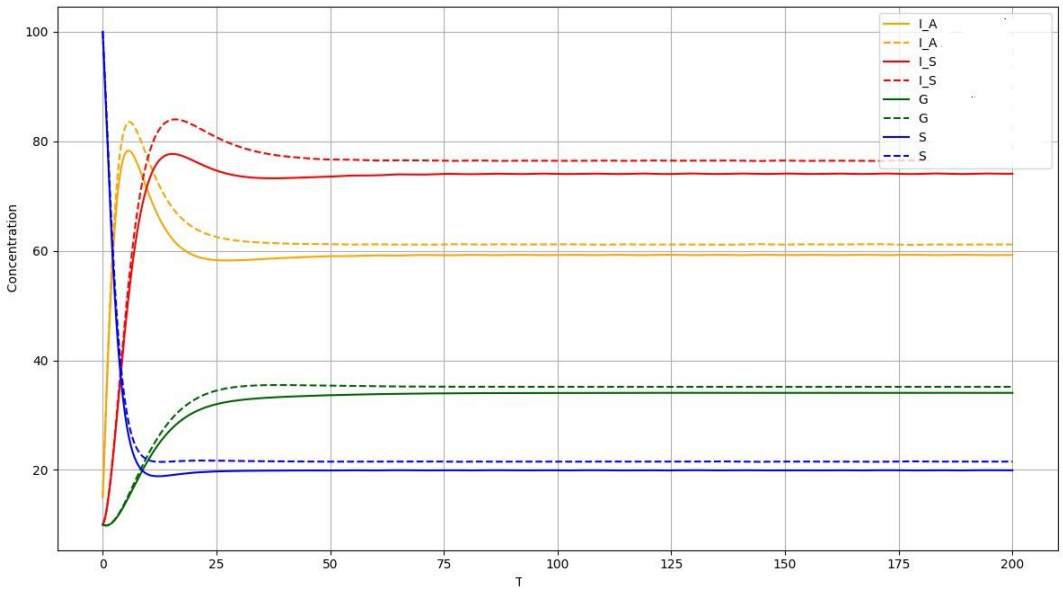

Figure 4: Comparison of paratuberculosis trends with and without vaccination

and, most importantly, minimizes environmental contamination, thereby helping to break the silent infection cycle that makes this disease so difficult to control. These results therefore highlight that, if vaccination is properly implemented, it can significantly reduce disease transmission. In this case,it becomes an effective and rational means of controlling the transmission of paratuberculosis.

After examining the impact of control strategies such as culling and vaccination on the dynamics of paratuberculosis, it appears necessary to pay particular attention to an often underestimated but crucial aspect: vertical transmission of the disease. This route of transmission, although silent, could compromise the effectiveness of control efforts if ignored. To assess its impact, we studied itsinfluence on the basic reproduction numbers, specifically$\mathcal{R}_0$(associated with the model without vaccination) and $\mathcal{R}_0^V$ (associated with the model with vaccination). The simulation results are presented in the following figure, which compares the values of the basic reproduction numbers in both cases. In the absence of vertical transmission, the values of $\mathcal{R}_0$and $\mathcal{R}_0^V$ remain relatively low, suggesting the possibility of natural extinction of the disease in the long term with controlmeasures in place. In contrast, even a moderate introduction of vertical transmission (σ = 0.05) leads to a significant increase in these indicators, particularly$\mathcal{R}_0^V$.

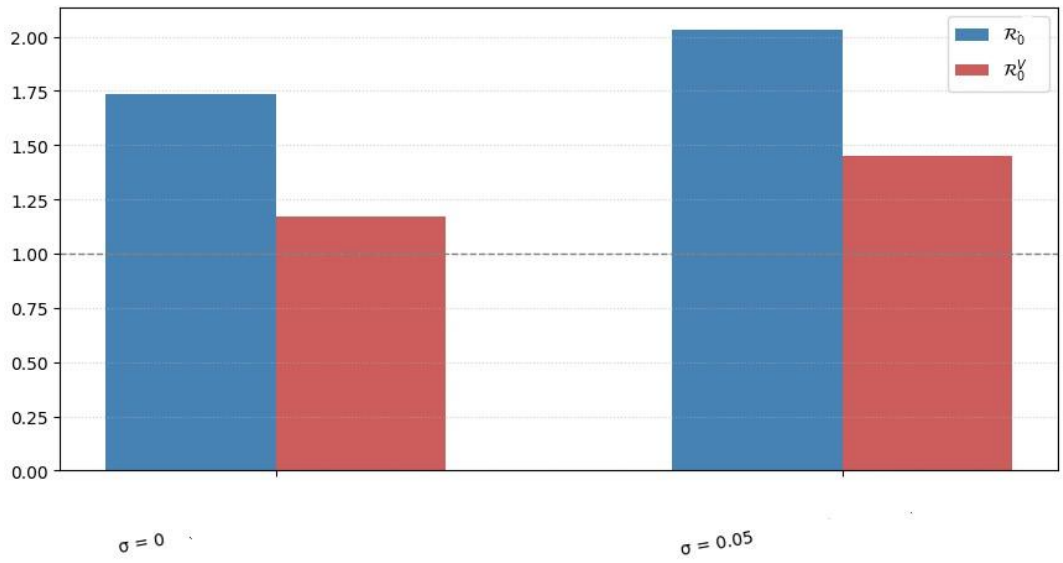

Figure 5: Impact of vertical transmission

Here, we simulate the effect of the vertical transmission rate σ on the vaccine-adjusted basic reproduction number $\mathcal{R}_0^V$. This simulation highlights a critical threshold for σ beyond which vaccination is no longer sufficient to control the disease.

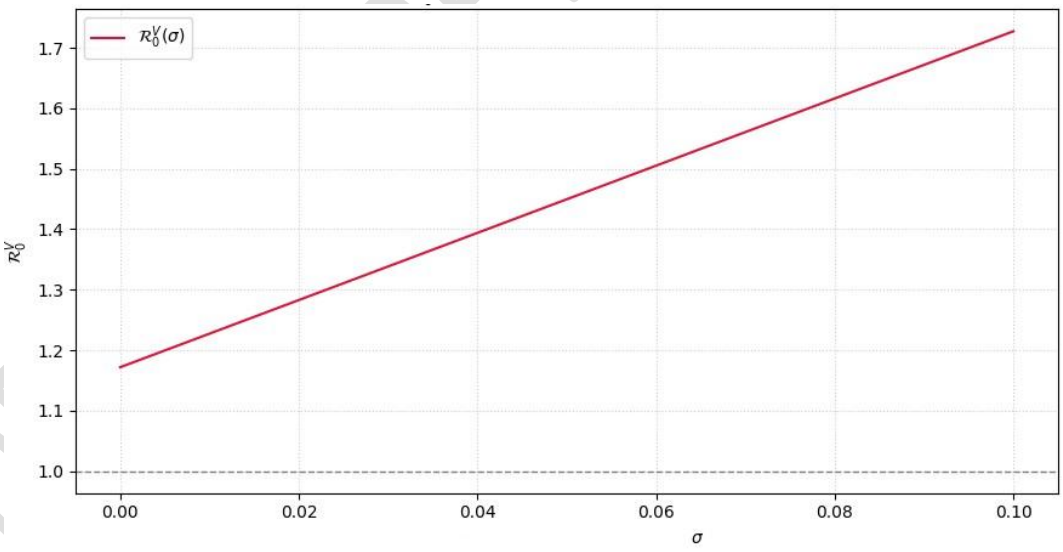

Figure 6: Impact of vertical transmission

Figure 6 shows that the basic reproduction number of the model with vaccination $\mathcal{R}_0^V$ is already greater than 1 in the absence of vertical transmission (σ = 0), indicating that the disease can persist in the population despite vaccination. This persistence is mainly due to horizontal transmission (direct and environmental).

In the presence of vertical transmission σ >0, the basic reproduction number increases. Thus,

vertical transmission constitutes an aggravating factor that must be taken into account in paratuberculosis control strategie.

These results suggest that vaccination must be supplemented by measures aimed at limiting both horizontal and vertical transmission in order to effectively control the disease.

6. Conclusion

A comparative study of reproduction numbers highlights the decisive influence of vaccination on the dynamics of paratuberculosis in Guinea. In the absence of vaccination, the basic reproduction number $\mathcal{R}_0$remains above 1, indicating active transmission and the persistence of infection within herds. Conversely, when vaccination is implemented, this basic reproduction number$\mathcal{R}_0^V$ decreases significantly, potentially approaching or even falling below the critical threshold of 1, provided thatvaccination coverage is adequate. This reduction indicates a slowdown in the spread of the disease and underscores the importance of vaccination as a control measure. However, the extent of this effect is largely determined by implementation methods, notably the level of commitment from livestock farmers, the frequency of vaccination campaigns, and their coordination with other health measures. As a result, vaccination has proven to be an effective tool, but it should not be considered a silver bullet, as its success depends on an integrated approach to disease management.

These findings open up significant opportunities for improving paratuberculosis control in Guinea. It would be worthwhile to conduct a more detailed assessment of reproduction rates in different livestock settings in order to identify the vaccination coverage thresholds needed to achieve sustainable control of the disease. Furthermore, the integration of more complex epidemiological models, taking into account livestock movements and interactions between herds, would allow for better anticipation of the large-scale impact of vaccination strategies.

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