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Modeling the Impact of Vaccination on Newcastle Disease Dynamics in Caged Chickens

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Abstract

Newcastle disease continues to have a significant economic impact on farmers and food security. This study develops and analyzes a deterministic mathematical model to investigate the effect of vaccination on the transmission dynamics of Newcastle disease in caged chicken populations. The model is based on the Susceptible-Exposed-Infected-Vaccinated-Revaccinated-Recovered-Susceptible (SEIVrRS) framework, adapted to capture the unique characteristics of Newcastle disease transmission. The disease-free equilibrium of the model was computed, and the basic reproduction number for Newcastle disease was calculated using the next-generation matrix method. Both analytical results and numerical simulations show that frequent vaccinations increase the number of susceptible chickens by reducing the at-risk chicken population. Additionally, re-vaccination significantly enhances immunity, resulting in a higher number of recovered chickens. Sensitivity analysis indicates that the recruitment rate of chickens, the effective contact rate between susceptible and infectious chickens, and the natural death rate of chickens are the most sensitive parameters for targeting in disease control strategies. Therefore, the findings from this study can support farmers and food security practitioners in decision-making regarding Newcastle disease control strategies and emphasize their crucial role in poultry disease management.

Keywords: : Newcastle disease, Caged chicken, Vaccine, Dynamics, Revaccination.

2010 MSC: 34C60, 92D30, 93A30.

1. Introduction

Newcastle disease (NCD) is an acute and highly contagious viral infection affecting poultry and wild birds, causing mortality and decreased productivity, resulting in significant economic losses. In rural areas, 80% to 100% of exposed chickens might die from

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the disease [7, 21, 12, 23]. Newcastle Disease Virus (NDV) strains have been diagnosed in domestic poultry, including pigeons, ostriches, turkeys, caged pet birds, and racing and exhibition pigeons. However, the effects of the disease vary greatly between species, with chickens being the most sensitive and ducks and geese being the least sensitive [16]. The disease was first identified in Newcastle-Upon-Tyne, England, and Java, Indonesia, in 1926, where it adopted its name. However, there had been previous accounts of similar outbreaks, such as in 1896 in Scotland's Western Isles, causing the death of all chickens [7].

Newcastle disease is caused by the virulent strain of Avian *Paramyxovirus-1* (APMV-1). It occurs most frequently as a disease of Avians such as chicken, ducks, pigeons, guinea fowls, and wild birds. NCD remains a formidable threat to the poultry industry worldwide. To mitigate the devastating impact of this highly contagious and economically burdensome disease, vaccination has emerged as a crucial control strategy (Akele et al., [18]).

Chuma et al. [6] developed a mathematical model to assess the role of wild birds and the environment in the transmission of Newcastle disease among village chickens. The model assumed that the recruitment rate of village chickens and wild birds occurs through birth, that wild birds cannot recover once infected, and that the NCD virus is introduced into the environment through shedding by clinically infected village chickens. Numerical findings reveal that the basic reproduction number is significantly impacted by the contact rate among susceptible village chickens, wild birds, and the contaminated environment. Moreover, increasing the removal rate of the NCD virus from the environment reduces the disease transmission rate within the chicken population, emphasizing the significance of a contaminated environment in facilitating the spread of the NCD virus among village chickens.

Ijeoma et al. [15] incorporated comprehensive equations that account for various factors affecting the transmission of Newcastle disease, including host population dynamics and parameters. The study also integrated optimization strategies to pinpoint effective control measures. However, the research falls short in specifically focusing on real-life applications and validating the proposed model.

Hugo et al. [2] formulated an eco-epidemiological model to analyze the optimal control and cost-effectiveness of Newcastle disease (NCD). The model assumed that chicken growth follows a logistic function with intrinsic growth rate and carrying capacity, while the predation functional response of humans towards susceptible and infected chickens follows Michaelis-Menten kinetics and is modeled using Holling type-II functional response. Findings suggest that combining chicken vaccination with a human education campaign is the most effective approach. To minimize the spread of NCD, farmers are also advised to administer chicken vaccination accurately and in a timely manner.

Ijeoma et al. [15] formulated a mathematical model on the optimal control of Newcastle disease (NCD). The model assumes that productive birds become non-productive, and that birds are recruited by immigration or birth, while those that recover from infection move to the non-productive susceptible class. The results reveal that the control measure is effective. The study demonstrates that when vaccinations are administered effectively and the vaccines work efficiently, they significantly help control infections in birds. The simulation also reveals that even if vaccinations are not administered very frequently, they still reduce the number of birds with hidden infections, which ultimately boosts bird pro-

ductivity.

Mubamba et al. [11] used the retrospective and predictive time series model to study the behavior of Newcastle disease in the past and to predict its future trend to enhance the planning of control strategies. The findings commended that different control approaches are required to halt the progress of ND.

Shatanawi et al.[20] demonstrate the approximate solutions using their proposed method across different fractional orders. They note that the decline in the susceptible class occurs more quickly at smaller fractional orders and decelerates as the order nears its integer counterpart. The exposed class exhibits similar behavior. The infection rate rises, leading to a reduction in the exposed class, with the growth rate being faster at integer orders and slower at smaller ones. This rise in infection results in a reduction of the under-treatment class. The dynamic behavior of the recovered class also shows these trends.

Hussain et al.[14] developed a stochastic epidemic model assessing the persistence of disease, the results reveal that R_0 is less than one when the noise levels are high implying that the disease does not persist otherwise the findings show that the disease persist only if the stochastic basic R_0 is greater than one. Chuma & Mwanga [10] developed a SEIV-structured eco-epidemiological mathematical model of the NCD in the backyard chicken. Bifurcation analysis of the model equilibrium points were done and the effects of vaccination on the disease dynamics assessed. The results show that the model undergoes forward bifurcation in the neighborhood $R_e = 1$, where the disease-free equilibrium point is locally asymptotically stable when $R_e < 1$ and unstable when $R_e > 1$. Also, the results reveal that the endemic equilibrium point is locally asymptotically stable when $R_e > 1$ and unstable when $R_e < 1$. The numerical evidence showed that the NCD can be controlled and eventually eradicated from the backyard chicken populations if and only if when $R_e < 1$. More interestingly, the results showed that the increase in vaccination rate leads to an increase in protective immunity which is crucial for successful control of NCD.

Annapragada et al. [1], developed a model on modeling the impact of Newcastle disease virus vaccinations on chicken production systems. The model focuses on changes in chicken populations over time from a vaccination intervention. The model assumes that the inflow and outflow parameters as known constants extracted from the longitudinal data. Also, the model assumes that vaccination is a known variable which is modeled for varying coverage levels. The results reveal that stabilization and population growth of chicken flocks are possible through moderate, consistent vaccination coverage. The model shows that vaccination coverage over 15 rounds (5 years) would enable population doubling. Also, the results indicate that in best-case scenarios the vaccination coverage needed is lower and may be unnecessary in some regions according to extreme best-case scenarios of chicken productivity. It is noteworthy that the increase in vaccination campaign leads to an increase in vaccination coverage rate, thus reducing the spread of Newcastle disease virus to the chicken population.

Daut et al. [5] formulated two mathematical models to explain the transmission of Newcastle Disease (ND) and how illegal harvest affects a population of parakeets. The first model describes a homogeneous population (model 1), while the second model (model 2) breaks down the population into different age groups. In model 1, all individuals experience the same infection transmission dynamics and harvest rates, making it simpler

to analyze mathematically compared to the more complex age-structured model 2. The findings indicate that ND could lead to significant disease-related deaths, possibly causing a population decline within two years. However, high rates of illegal harvest could reduce the severity of the outbreak. Model 2 showed moderate differences in disease dynamics compared to model 1, particularly in how the disease affects different age groups. Furthermore, the results suggest that while high rates of illegal harvest might reduce virus transmission and protect the population, the combined effects of intense harvest and disease-induced mortality could pose a threat to the survival of the population.

Abdo et al. [3] conducted a study on highly contagious disease spread through direct contact shows that vaccination programs are the most effective way to prevent measles. Backward bifurcation indicates that relying solely on basic reproduction numbers (R_0) can lead to incorrect predictions, as measles can still occur even when R_0 is below one. Despite the development of various models since then, several key issues remain to be addressed in future studies. These include the influence of IgG antibodies, which allow immunity to be passed from mothers to their offspring, and the significant impact of measles co-infection with other severe diseases like pneumonia and diarrhea.

Chuma and Mwangi [9] developed a mathematical model to study the stability analysis of equilibrium points of Newcastle disease in village chickens, considering the presence of a wild bird reservoir. The model assumes that the primary mode of Newcastle Disease Virus (NDV) transmission among hosts occurs through direct contact between infected and susceptible chickens, while secondary transmission routes involve susceptible chickens coming into contact with either contaminated environments or wild bird reservoirs. Additionally, the model assumes equal birth and death rates for village chickens. The results indicate that the disease-free equilibrium point is locally asymptotically stable when $R_e < 1$. By applying the Castillo-Chavez Theorem, it is demonstrated that the disease-free equilibrium point is globally asymptotically stable. Moreover, utilizing logarithmic functions and LaSalle's Theorem, the model identifies the globally asymptotically stable endemic equilibrium point when $R_e > 1$. Numerical simulations depict that implementing effective interventions decreases the frequency of Newcastle disease outbreaks in village chicken populations. Deterministic mathematical models are highly recommended for studying endemic diseases [34, 35]. Methods such as integer and fractional-order models play a crucial role in analyzing these diseases, as demonstrated by [13, 17, 22].

This study delves into the complex dynamics of NCD within caged chicken populations; with a particular focus on the inclusion of vaccination. Furthermore, the study is aimed at employing mathematical modeling techniques, to shed light on the interplay between disease transmission dynamics and the impact of vaccination strategies on the health and productivity of caged chicken. A thorough review of the related literature is provided below, laying the groundwork for a comprehensive understanding of the dynamics of Newcastle disease (NCD) in caged chicken populations, with a focus on the role of vaccines in disease control.

1.1. Contribution of the Study

This study formulates mathematical model that uniquely analyzes the effect of chicken vaccination on Newcastle disease (NCD) transmission among caged chicken populations. Distinct from earlier models, this study incorporates detailed NCD transmission dynamics

alongside vaccination strategies, presenting a novel method for assessing disease control measures. Furthermore, this research aims at providing practical insights for improving poultry health and productivity through effective vaccination programs, thereby supporting broader efforts to reduce the economic and health impacts of NCD on the poultry industry.

2. MATERIAL AND METHODS

2.1. Formulation of the Model

We develop a Susceptible-Exposed-Infected-Vaccinated-Revaccinated-Recovered-Susceptible (SEIVVrRS) model to study the transmission dynamics of ND, caged chicken are the targeted population. The model is constructed under several assumptions such as; principle mass action, the susceptible caged chicken can get infections by direct contact with infected chicken, vaccinated chicken will continue to receive vaccine after every 21 days, the presence of mortality within each compartment, considers constant recruitment of susceptible caged chicken, assumes that the disease is fatal (disease induced death), some infected chicken may go to recovery class naturally, the recovered chicken do not gains permanent immunity and the model does not consider the immigration of infected chicken. Table 1 presents the descriptions of parameters and variables used in model 2.1.

Table 1: Description of variables and parameters used in Model 2.1

Variable/Parameter	Description
S	Susceptible caged chicken population
E	Exposed caged chicken population
I	Infected caged chicken population
V	Vaccinated caged chicken population
V_r	Re-vaccinated caged chicken population
R	Recovered caged chicken population
λ	Recruitment rate of caged chicken
μ	Natural mortality rate of caged chicken
ϕ	Disease-induced death rate
α	Effective contact rate of caged chickens
σ	Rate at which exposed caged chicken show infection symptoms
δ	Natural recovery rate of caged chicken
η	Rate at which recovered chickens lose immunity
β	Vaccination rate
θ	Re-vaccination rate
ρ	Warning rate of vaccination

2.2. Model Flow diagram

Based on the interactions between caged chicken, model assumptions, definition of variables and parameters respectively, the dynamics of the Newcastle disease can be summarized in Figure 1 as follows:

2.3. Model Equations

The model equations represent the set of differential equations that describe the dynamics of the disease transmission, including changes in the populations of susceptible,

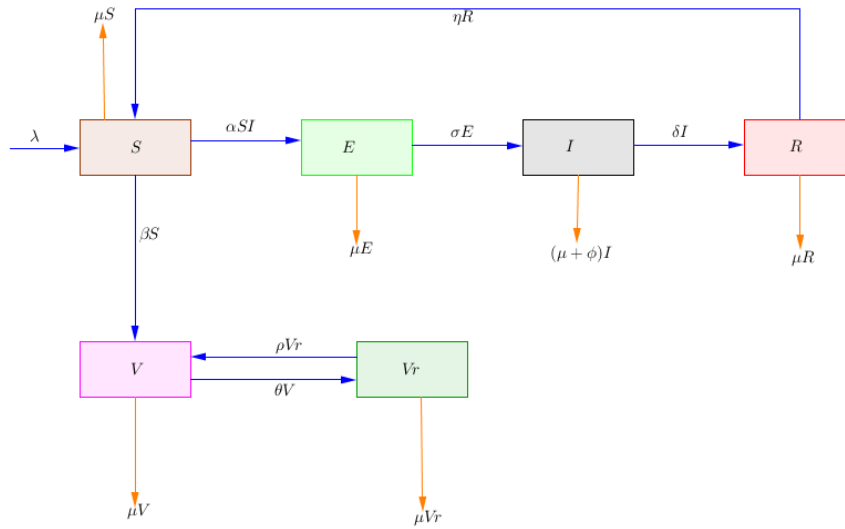


Figure 1: Compartment model diagram for the transmission dynamics of Newcastle disease in Caged chicken with inclusion of Vaccine.

vaccinated, exposed, infected, and recovered chickens over time. From Figure 1 we have the following linear differential equations:

$$\begin{cases} \frac{dS}{dt} = \lambda + \eta R - \alpha SI - (\mu + \beta)S \\ \frac{dE}{dt} = \alpha SI - (\mu + \sigma)E \\ \frac{dI}{dt} = \sigma E - (\mu + \phi + \delta)I \\ \frac{dR}{dt} = \delta I - (\eta + \mu)R \\ \frac{dV}{dt} = \beta S + \rho V_r - (\mu + \theta)V \\ \frac{dV_r}{dt} = \theta V - (\mu + \rho)V_r \end{cases} \quad (2.1)$$

with non-negative initial conditions;

$$S(0) > 0, \quad E(0) \geq 0, \quad I(0) \geq 0, \quad R(0) \geq 0, \quad V(0) \geq 0, \quad \text{and} \quad V_r(0) \geq 0$$

2.4. Basic properties of the model

2.4.1. Invariant region

This region defines the state space where the solutions of the differential equations describing the disease dynamics remain for all time $t \geq 0$ [32, 33]. Let the total population be given as $N = S + E + I + R + V + V_r$. It follows that:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dV}{dt} + \frac{dV_r}{dt}. \quad (2.2)$$

Substituting the equations of model system (2.1), into equation (2.2), we have:

$$\begin{aligned}\frac{dN}{dt} &= \lambda - \mu N - \phi I, \\ \implies \frac{dN}{dt} &\leq \lambda - \mu N, \\ \implies \frac{dN}{dt} + \mu N &\leq \lambda.\end{aligned}\tag{2.3}$$

Using the integration factor and the initial condition we obtain:

$$N(t) \leq \frac{\lambda}{\mu} + N(0)e^{-\mu t}.\tag{2.4}$$

Taking limit as $t \rightarrow 0$, we obtain:

$$N(t) \leq \frac{\lambda}{\mu}.\tag{2.5}$$

2.4.2. Positivity of Solution

Theorem 2.1 Let the initial value of variables of the model in the equation 2.1 be $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, $V(0) \geq 0$, and $V_r(0) \geq 0$, then the solution set $\{S(0) > 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, V(0) \geq 0, V_r(0) \geq 0\} \in \mathbb{R}_+^6$ is positive for all time.

Proof. From the first equation of model system (2.1),

$$\begin{aligned}\frac{dS}{dt} &= \lambda + \eta R - \alpha SI - (\mu + \beta)S, \\ \implies \frac{dS}{dt} &> -(\alpha I + \mu + \beta)S.\end{aligned}\tag{2.6}$$

Separation of variables and integration using initial conditions, gives:

$$\begin{aligned}\frac{dS}{S} &> -(\alpha I + \mu + \beta)dt, \\ \implies \int_{S(0)}^{S(t)} \frac{dS}{S} &> -\int_0^t (\alpha I(s) + \mu + \beta)ds \\ \implies S(t) &> S(0)e^{-\int_0^t (\alpha I(s) + \mu + \beta)ds} > 0.\end{aligned}\tag{2.7}$$

Using the same approach, it can be shown that $E(t) \geq 0$, $I(t) \geq 0$, $R(t) \geq 0$, $V(t) \geq 0$, and $V_r(t) \geq 0$. \square

2.5. Existence of the Disease Free Equilibrium Point (DFE)

This defines the condition where the model predicts no presence of the disease within the chicken population. At this equilibrium point, the populations of infected and exposed chickens are zero, indicating that the disease has been eradicated or is not present. When there is no disease in the population, the disease free equilibrium E^0 , is given as: $E^0(S, E, I, R, V, V_r) = \left(\frac{\lambda}{\beta + \mu}, 0, 0, 0, \frac{\beta\lambda(\rho + \mu)}{\mu(\beta + \mu)(\theta + \rho + \mu)}, \frac{\beta\theta\lambda(\rho + \mu)}{\mu(\beta + \mu)(\theta + \rho + \mu)} \right)$.

2.5.1. The basic reproduction number R_0

The basic reproduction number is the number of new infections that may arise when one infected individual is introduced in a completely susceptible population [26, 27, 25]. The disease clears in a population when $R_0 < 1$ and persists when $R_0 > 1$. To find the basic reproduction number R_0 , we adopt the next generation matrix approach as used in the study conducted by Nyerere et al. [29, 30]. Let \mathcal{F}_i be the rate of appearance of new infection in compartment i and \mathcal{V}_i be the transfer of infections from one compartment i to another. Then, by differentiating \mathcal{F}_i and \mathcal{V}_i partially with respect to E and I at disease-free equilibrium point, E^0 , we get

$$F = \begin{pmatrix} 0 & \alpha\lambda \\ \mu & 0 \end{pmatrix} \quad (2.8)$$

$$V = \begin{pmatrix} (\mu + \sigma) & 0 \\ -\sigma & (\mu + \phi + \delta) \end{pmatrix}$$

Then,

$$V^{-1} = \begin{pmatrix} \frac{1}{(\mu + \sigma)} & 0 \\ \frac{\sigma}{(\mu + \sigma)(\mu + \phi + \delta)} & \frac{1}{(\mu + \phi + \delta)} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{\alpha\sigma\lambda}{\mu(\mu + \sigma)(\mu + \phi + \delta)} & \frac{\alpha\lambda}{(\mu + \sigma)(\mu + \phi + \delta)} \\ 0 & 0 \end{pmatrix}$$

Upon computation, the basic reproduction number (R_0) was found to be:

$$R_0 = \frac{\alpha\sigma\lambda}{\mu(\mu + \sigma)(\mu + \phi + \delta)}$$

2.5.2. Global stability of DFE

In this subsection, we conduct a global stability analysis of the HPV-free equilibrium point utilizing the methodology outlined by Castillo-Chavez et al. [28]. Let the Lyapunov function V be given as:

$$V = \frac{\sigma}{(\mu + \sigma)(\mu + \phi + \delta)} E + \frac{1}{\mu + \phi + \delta} I$$

$$\implies \frac{dV}{dt} = \frac{\sigma}{(\mu + \sigma)(\mu + \phi + \delta)} \frac{dE}{dt} + \frac{1}{\mu + \phi + \delta} \frac{dI}{dt}$$

$$\implies \frac{dV}{dt} = \frac{\alpha\sigma SI - (\mu + \sigma)(\mu + \phi + \delta)I}{(\mu + \sigma)(\mu + \phi + \delta)}$$

But, at disease free equilibrium, $S = \frac{\lambda}{\mu}$. Thus we have:

$$\frac{dV}{dt} = \left(\frac{\alpha\sigma\lambda - \mu(\mu + \sigma)(\mu + \phi + \delta)}{\mu(\mu + \sigma)(\mu + \phi + \delta)} \right) I, \quad (2.9)$$

$$\implies \frac{dV}{dt} = (R_0 - 1) I.$$

Thus we conclude that

Case I: the DFE is globally asymptotically stable if and only if $\frac{dV}{dt} < 0$ where, $R_0 < 1$ and $R_0 = 0$.

Case II: the DFE is globally unstable if and only if $\frac{dV}{dt} > 0$ where, $R_0 > 1$.

Case III: the DFE is neither globally asymptotically stable nor unstable if and only if $\frac{dV}{dt} = 0$ where, $R_0 = 1$ and $I = 0$.

2.5.3. The Effective Reproduction Number R_e

The effective reproduction number is the number of secondary cases that may occur when one infected individual is introduced in a susceptible population when some interventions are in place to control the disease [31]. The interventions are effective when $R_e < 1$ and ineffective when $R_e > 1$. In computing R_e , we adopt the next generation matrix approach as used in section 2.5.1. Use this approach it can be shown that the effective reproduction number is given as:

$$R_e = \frac{\alpha\sigma\lambda}{(\mu + \beta)(\mu + \sigma)(\mu + \phi + \delta)}.$$

2.6. Existence of the Endemic Equilibrium point

The endemic equilibrium is a point where the disease persist in the population. It is obtained by setting the derivative of the model system equal to zero and solving for the state variables. Thus, the endemic equilibrium is given as $E^* = (S^*, E^*, I^*, R^*, V^*, V_r^*)$ where:

$$\begin{aligned} S^* &= (\mu + \sigma) \frac{(\mu + \phi + \delta)}{\alpha\sigma} \\ E^* &= \frac{\lambda\sigma(\mu + \eta)(\mu + \phi + \delta) - (\mu + \eta)(\mu + \beta)(\mu + \delta)(\mu + \phi + \delta)^2}{\alpha\sigma(\mu + \sigma)(\mu + \eta)(\mu + \phi + \delta) - \nu\sigma^2\delta} \\ I^* &= \frac{\lambda\sigma - (\mu + \beta)(\mu + \delta)(\mu + \phi + \delta)}{\alpha(\mu + \sigma)(\mu + \phi + \delta) - \eta\sigma\delta} \\ R^* &= \frac{\lambda\sigma\delta - \delta(\mu + \beta)(\mu + \delta)(\mu + \phi + \delta)}{\alpha(\mu + \sigma)(\mu + \phi + \delta)(\mu + \eta) - \eta\sigma\delta} \\ V^* &= \frac{\beta(\mu + \rho)(\mu + \sigma)(\mu + \phi + \delta)}{\alpha\sigma(\mu + \theta)((\mu + \rho) - \alpha\sigma\theta\rho)} \\ V_r^* &= \frac{\beta\theta(\mu + \sigma)(\mu + \phi + \delta)}{\alpha\sigma(\mu + \theta)((\mu + \rho) - \alpha\sigma\theta\rho)} \end{aligned} \quad (2.10)$$

2.7. Sensitivity analysis of R_0

The sensitivity indices of the model parameters are calculated for determining parameters that have a major effect on basic reproduction number R_0 and should be targeted by strategies for intervention. The normalized forward sensitivity index method, as described by Chitnis *et al.*[4], is used to derive sensitivity indices. If Φ is a parameter in the basic reproduction number R_0 , then its sensitivity index is given by:

$$\Gamma_{\Phi}^{R_0} = \frac{\partial R_0}{\partial \Phi} \times \frac{\Phi}{R_0}.$$

When a parameter has a positive sensitivity index, such as the human birth rate with an index of +1.0000, its value increases in direct proportion to the initial transmission rate R_0 . Conversely, a negative sensitivity index, like the natural mortality rate with an index of -1.2000, indicates an inverse relationship with R_0 . Parameters with high sensitivity indices significantly impact disease transmission. However, certain factors, like natural mortality and human birth rates, remain unavoidable despite their high sensitivity indices [24].

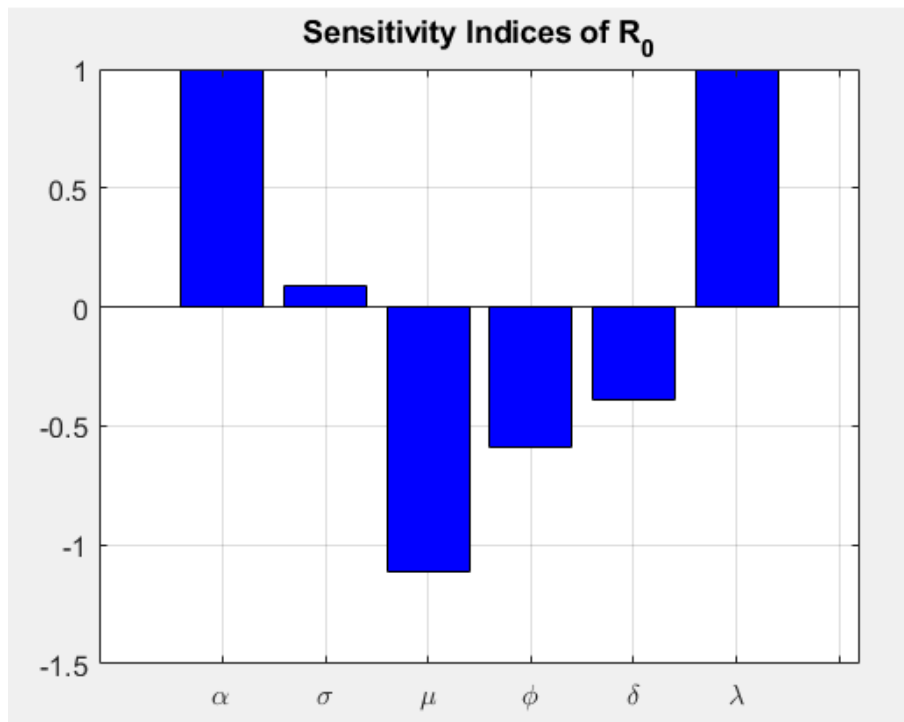


Figure 2: Sensitivity indices of R_0 with respect to the parameters.

Referring to Figure 2 the graph depicts that the most positive sensitive parameters are effective contact rate (α) and the recruitment rate (λ) whereas the most negative sensitive parameter is natural mortality rate (μ). Also, the graph indicates that the rate in which exposed caged chicken show sign of infections of Newcastle disease (σ) is the smallest positive sensitive parameter whereas the rate in which the caged chicken undergo natural recovery (δ) is the smallest negative sensitive parameter. Thus, parameters α and λ have positive influence on the basic reproduction number R_0 whereas μ has negative influence on the basic reproduction number R_0 . Furthermore, the index of parameter δ reveal that Newcastle disease is very dangerous and there is minimal probability of the affected caged chicken to undergo natural recovery. Therefore, based on these results, we recommend that vaccination and re-vaccination of infected individuals should be considered in controlling the transmission of Newcastle disease.

3. NUMERICAL SIMULATION OF THE MODEL

The Table 2 below presents the name of the parameter used in the model, the value of the parameter, and the source from which the value has been extracted

Table 2: Parameter values and their sources

Parameter	Parameter value	Source
λ	10	[36]
μ	0.002	[36]
ϕ	0.6	[2]
α	0.1	[2]
σ	0.2	Assumed
δ	0.4	[2]
η	0.05	Assumed
β	0.6	[2]
θ	0.2	Assumed
ρ	0.1	Assumed

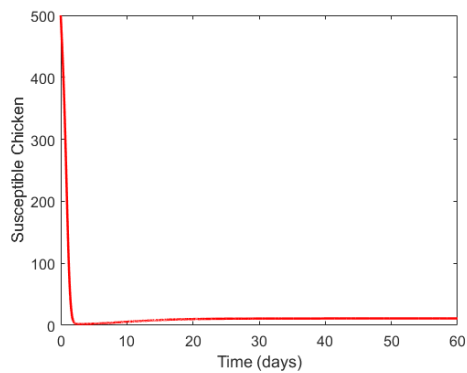
3.1. Model without interventions

From the Figure 3 below, the dynamics of chicken population exhibit clear trends. Initially, the susceptible chicken rapidly decline to zero within 2 to 3 days due to the absence of any control measure as depicted in Figure 3a. This scenario leads, the population of exposed chicken begins to rise quickly for the same approximately time. But eventually, some chicken transition to the infectious stage while others undergo natural death, leading to a decrease as shown in Figure 3b. Infected chicken also spend approximately 3 days to see an increase due to a higher progression rate, followed by a decline caused by natural and disease-induced deaths as illustrated in Figure 3c. The impact of strong immunity for some chicken is clearly observed in the curve representing recovered chicken. Initially spend 12 to 13 days to experience an increase, but over time, followed by gradual decrease and stabilization over time as shown in Figure 3d. Furthermore, the figure illustrates that the susceptible chicken begin to increase again approximately after 5 days as a result of increase of recovered chicken.

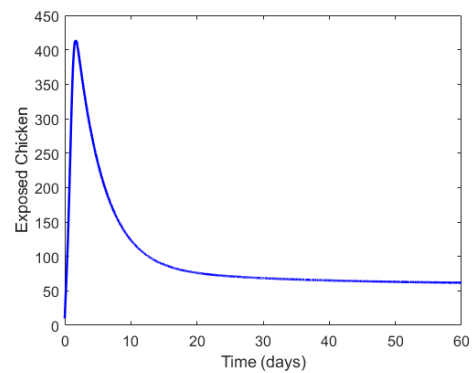
The observed trends in the chicken population dynamics can be biologically explained: The rapid decline of susceptible chickens within 2 to 3 days is due to the high transmission rate of the disease, leading to a sharp rise in exposed chickens. Some of these exposed chickens then progress to the infectious stage, initially increasing the number of infectious chickens before a decline due to natural and disease-induced deaths. Recovered chickens show an increase around 12 to 13 days as they mount an immune response, followed by a gradual decrease and stabilization due to natural deaths and long-term immunity. The rise in susceptible chickens after about 5 days is likely due to the natural reproduction and the re-entry of recovered chickens if their immunity wanes.

3.2. Model with interventions

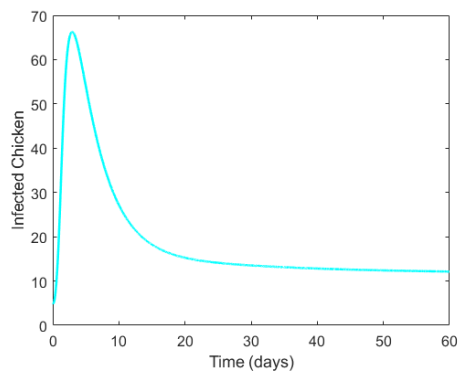
Referring Figure 4a below the results indicates that the susceptible chicken increase due to the effect of frequently vaccination. More interestingly the susceptible chicken curve undergoes steady state immediately after 25 days. Also, the experiments reveal that, caged chicken which are at high risks of being affected decreases due to the effect of continuous vaccination program. Hence the vaccination and revaccination programs



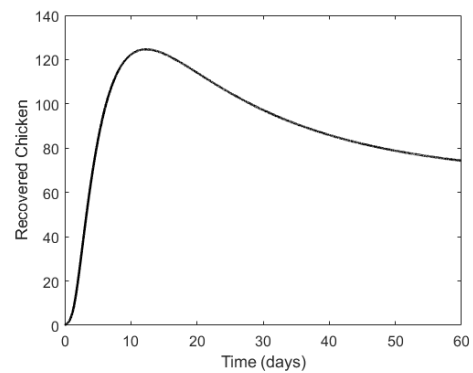
(a) Time against Susceptible chicken



(b) Time against Exposed chicken



(c) Time against Infected chicken



(d) Time against Recovered chicken

Figure 3: Model without intervention

have the greater contributions in reducing the spread of Newcastle disease. Also the graph depicts that the exposed chicken undergoes equilibrium immediately after 50 days as shown in Figure 4b. Referring to Figure 4c below the result proves that rate of infections of Newcastle disease decrease as the increase of vaccination and revaccination programs. More interestingly, the graph depicts that the infected caged chicken diminish immediately after the first phase of vaccination program. Due to effective vaccination and revaccination programs, the results show that the rate of infections goes to equilibrium immediately after 25 days of vaccination and revaccination programs. According to Figure 4d below the results reveal that the vaccination is less effective due to the fact that it takes so many days to reduce the spread of Newcastle disease. Also, the graph shows that the vaccinated chicken are less stable in comparison to revaccinated chicken.

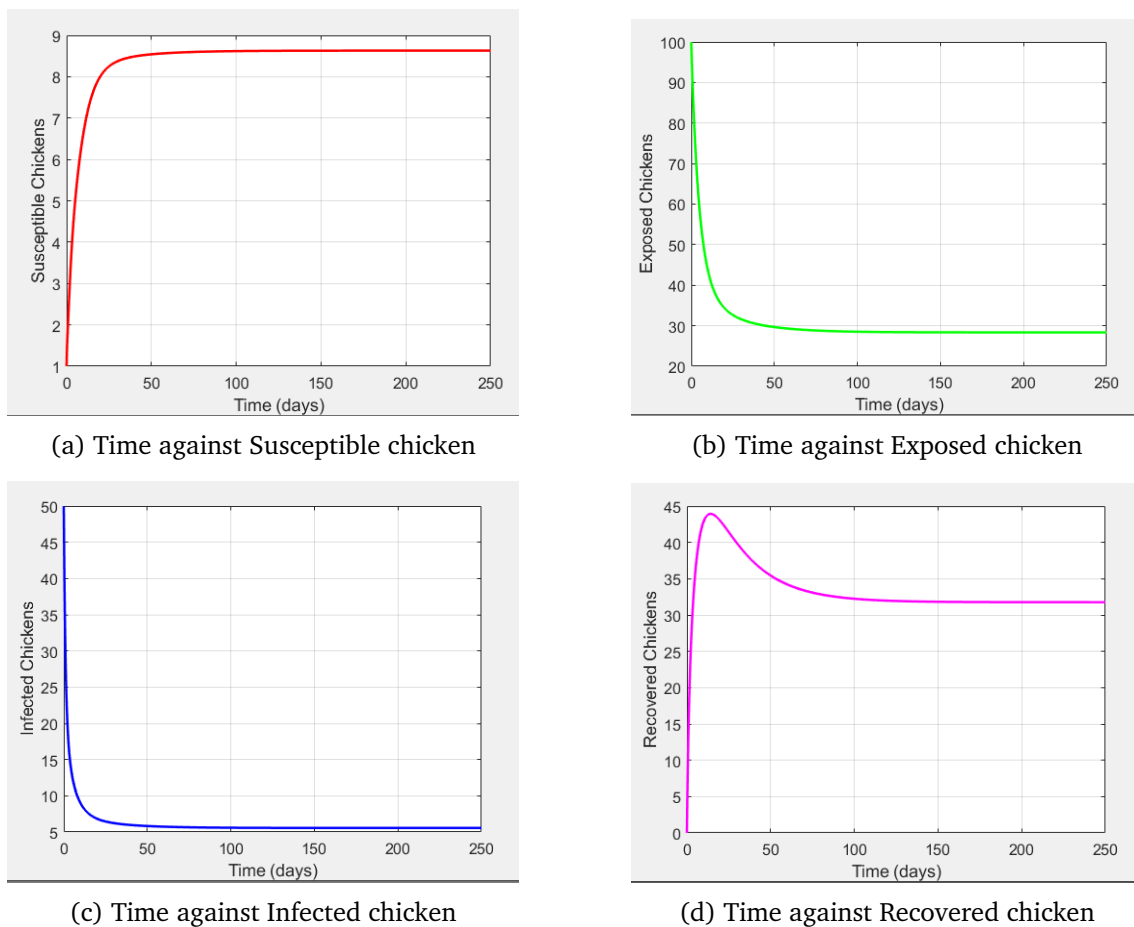


Figure 4: Model with intervention

According to Figure 5a below the results reveal that the revaccination is highly effective which leads to the increase of number of chicken with strong immunity reducing high risk of being affected with the Newcastle disease. Furthermore, the graph shows that the revaccinated chicken stabilizes immediately 200 days. Furthermore, the analysis shows that the recovered chicken increase as the increase of revaccination program as depicted

in Figure 5b. Due to effective vaccination and revaccination programs, the results depicts that recovered chicken undergoes steady state immediately after 100 days. Continuous vaccination and revaccination reduce the number of high-risk caged chickens, significantly curbing the spread of Newcastle disease.

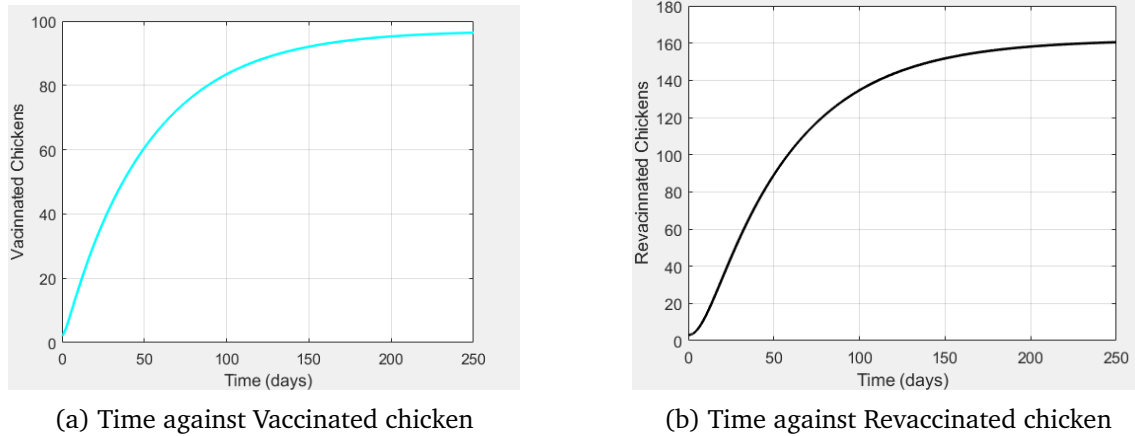


Figure 5: Effects of Vaccination and Revaccination

4. CONCLUSION AND RECOMMENDATIONS

4.1. Conclusion

The analysis of the model with intervention reveals several key findings regarding the dynamics of Newcastle disease transmission in chicken populations. Notably, frequent vaccination contributes to an initial increase in susceptible chicken, but the curve stabilizes after approximately 25 days, indicating the establishment of immunity within the population. Continuous vaccination programs demonstrate a significant reduction in the population of caged chicken at high risk, effectively reducing disease spread. Revaccination emerges as highly effective in enhancing chicken immunity and stabilizing population dynamics, particularly evident after 200 days. Additionally, the increase in recovered chicken over time, along with a decrease in infection rates, highlights the efficacy of vaccination and revaccination programs in controlling Newcastle disease transmission. Sensitivity analysis further underscores the importance of parameters influencing disease spread and recovery rates.

4.2. Recommendations

Based on these findings, it is recommended that vaccination and revaccination programs be prioritized in controlling the transmission of Newcastle disease among chicken populations. These interventions have demonstrated significant effectiveness in reducing disease spread, stabilizing population dynamics, and enhancing immunity against the disease. Continuous monitoring and implementation of vaccination and revaccination strategies are crucial for mitigating the impact of Newcastle disease and ensuring the health and well-being of poultry populations. Additionally, further research and analysis are warranted to explore additional parameters and factors that may influence disease

transmission dynamics and inform more targeted intervention strategies. By implementing these recommendations, stakeholders can effectively combat Newcastle disease and safeguard poultry populations from its detrimental effects.

4.3. Future work

Future research should investigate the prolonged effects of ongoing vaccination, refine vaccination timing and frequency, and perform in-depth sensitivity analyses to identify crucial factors affecting disease dynamics. Moreover, it should evaluate various vaccination approaches, adapt the model to different environments, include economic evaluations for cost-effectiveness, and examine how vaccination can be combined with other control measures to create comprehensive strategies for managing Newcastle disease.

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Data Availability

The information utilized to substantiate the conclusions in this research is provided within the article. In fact, data from other studies were employed for the simulations, with proper citation of the respective papers.

Conflicts of Interest

The authors declares no conflict of interest regarding the publication of this paper.

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