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THE IMPACT OF MEDIA COVERAGE ON THE TRANSMISSION DYNAMICS OF FOWL POX IN POULTRY

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Abstract

In this work, we have formulated a deterministic mathematical model of vector-host transmission dynamics of fowl pox in poultry, to investigate the impact of media coverage in the reduction and control of fowl pox in poultry. The compartmental model resulted in an ordinary differential equation that includes the effect of media coverage on reporting the numberof infections. Methods from dynamical system theory were employed in analysing the equilibrium stability of the model at both disease free equilibrium and endemic equilibrium. Appropriate conditions for the local asymptotic stability of both equilibrium points have been established. A threshold parameter R_0 (the basic reproductive ratio) was also derived analytically to discuss the local stability of the disease free equilibrium.

Key Words : *Media Coverage, Vector fowl pox, Basic reproductive ratio, Stability, Equilibrium state.*

1. Introduction

Infectious diseases are responsible for a quarter of all deaths in the world annually, the vast majority occurring in low and middle income countries [1]. Infectious diseases account for a vast majority of loss recorded in poultry farming. When an infectious disease appears and spreads in a region, farmers will do everything possible to control and prevent the disease spreading. One of the immediate measures to take is to educate people on the preventive knowledge of the disease through media coverage. It is a common sense that the more preventive knowledge the population has the better to prevent the spreading of the disease. Media coverage has great influence not only the farmer's behaviours but also on the formation and implementation of public intervention and control policies[9]. Media report plays a key role in the perception, management and even creation of crises [10].

The benefit of publicly reporting disease outbreaks seems obvious, and public health officials in the United States have a policy of regularly communicating with the news media about such incidents. But unfortunately, not all world governments choose to communicate effectively. The role of media coverage on disease outbreaks is thus crucial and should be given prominence in the study of infectious disease dynamics. Information dissemination influences the farmer's risk perception and shapes evolution of epidemics.

There have been mathematical modelling studies to investigate the impact of media coverage and psychology to the spread and control of infectious diseases in a given population or region.

[8] Develops a model to explore the impact of media coverage on the control of spreading of emerging or re-emerging infectious diseases in a given population. The model resulted in two equilibria:- a disease free equilibrium and a uniqueendemic equilibrium. Stability analysis of the model shows that the disease freeequilibrium is globally asymptotically stable if the reproduction number R_0i s lessthan unity, and the endemic equilibrium is globally asymptotically stable when itexists. Though the media coverage itself is not a determined fact to eradicate theinfection of the diseases, the analysis of model indicates to certain extent that themore media coverage/alert in a given population, the less number of individualswill be infected. Therefore, the media coverage is critical for educating people inunderstanding the possibility of being infected by the disease.

[11] Formulates a deterministic transmission and vaccination model to investigate the

effects of media coverage on the transmission dynamics of influenza. The resulted compartmental model includes the effect of media coverage on reporting the number of infections as well as the number of individuals successfully vaccinated. A threshold parameter (the basic reproductive ratio) was analytically derived and used to discuss the local stability of the disease-free steady state. The impact of costs that can be incurred, which include vaccination, education, implementation and campaigns on media coverage, were also investigated using optimal control theory. A simplified version of the model with pulse vaccination showed that the media can trigger a vaccinating panic if the vaccine is imperfect and simplified messages result in the vaccinated mixing with the infectives without regard to disease risk.

[9] Used a compartmental model to illustrate a possible mechanism for multiple outbreaks oreven sustained periodic oscillations of emerging infectious diseases due to the psychologicalimpact of the reported numbers of infectious and hospitalized individuals. This impact leadsto the change of avoidance and contact patterns at both individual and community levels, andincorporating this impact using a simple nonlinear incidence function into the model showed qualitative differences of the transmission dynamics.

Cruz Vargas de Leon,[13] in their work dealt with the global stability properties of two host-vector disease models using the Poicar_e-Bendixson Theorem and Second Method of Lyapunov. He constructed a Lyapunov function for each Vector-Host model and proved that the local and global stability are completelydetermined by the threshold parameter, R_0. He established that, if $R_0 < 1$, the disease-freeequilibrium point is globally asymptotically stable. Also if $R_0 > 1$, then, the uniqueendemic equilibrium point exists and is globally asymptotically stable inthe interior of the feasible region.

The study of the dynamics infectious diseases using mathematical models has proven to be a valuable tool to understand epidemiological patterns and processes, provided that the models are as close as possible to real life situations and based on biological knowledge. In this work, we shall formulate the mathematical model that incorporates media coverage which will enable us to investigate the control strategies and understand the complex dynamics of vector-host interactions that produce fowl pox infections in poultry.

2. Model Framework

2.1 Assumptions

1. The infection is transmitted by both the vector and the infected birds.
2. Birds that recover from one can become susceptible to the other strain.
3. Media report has positive impact on the transmission rate of the infection.
4. Mosquitoes are the primary reservoir and spreaders of the infection in poultry range[12].
5. The vector has logistic growth.

2.2 Parameters/Symbols

S	=	Susceptible birds
I	=	Infected birds
V	=	Vector (Mosquito)
Λ	=	recruitment term of the susceptible birds
α_1	=	infection rate of fowl pox in poultry
μ	=	the rate at which the infected birds recover and become susceptible
φ	=	natural death rate (it is the same for each sub-population)
γ	=	death due to infection
δ	=	death rate of the vector due to extra effort as a response to media coverage
$\frac{\alpha_2 I}{m_1+I}$	=	the measure of the effect of reduction in transmission rate as a result of media coverage
$\frac{\alpha_3 V}{m_1+V}$	=	the measure of the effect of reduction in transmission rate due to extra effort on the vector as a response of media coverage
m_1	=	the halfsaturation constant which reflects the impact of media coverage on thecontact- transmission rate
ρ	=	growth rate of the vector
K	=	the carrying capacity for the vector.

Our model describes the transmission dynamics of fowl pox infection based on two strains. Here we assume that birds which recover from one strain can become susceptible to the other strain. The susceptible population is increased by recruitment of birds either by birth or immigration and the recovered bird that become susceptible. This

population is reduced by infection and by natural death or emigration. The infected population is increased by infection of susceptible birds either by infected bird or the vector (mosquito). This population is diminished by natural death, death due to infection, and those that recover and become susceptible. The vector population assumes a logistic growth with K as the carrying capacity and ρ as the growth rate of the vector. This population is decreased by emigration or natural death and death due to extra effort as a response from media coverage. Media coverage is introduced into the system via saturated incidence functions $H(I) = \frac{\alpha_2 I}{m_1+I}$ and $g(V) = \frac{\alpha_2 V}{m_1+V}$.

2.3 Model Equations

The transmission model with media coverage is given by the following deterministic system of non linear ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= A - \left(\alpha_1 - \frac{\alpha_2 I}{m_1+I}\right) SI - \left(-\frac{\alpha_3 V}{m_1+V}\right) SV + \mu I - \varphi S \\ \frac{dI}{dt} &= \left(\alpha_1 - \frac{\alpha_2 I}{m_1+I}\right) SI + \left(\alpha_1 - \frac{\alpha_3 V}{m_1+V}\right) SV - \mu I - \varphi I - \gamma I \\ \frac{dV}{dt} &= \rho V\left(1 - \frac{V}{K}\right) - \varphi V - \delta V \end{aligned} \tag{3.1}$$

3. Stability of the Equilibrium States

The disease free equilibrium is given by

$$E_0 = (S^*, I^*, V^*) = \left[\frac{\Lambda\rho(\rho m_1 + P)}{(\alpha_1 P + \varphi\rho)(\rho m_1 + P) - \alpha_3 P^2},\ \ 0, \frac{K}{\rho}(\rho - \varphi - \delta)\right]$$

where $P = K(p - \varphi - \delta)$.

The endemic equilibrium of the system is given by $E_1 = (S^*, I^*, V^*)$. It satisfies $S^* > 0, I^* > 0, V^* > 0$ and

$$S^* = \frac{(\mu + \varphi + \gamma)(\rho m_1 + K(\rho - \varphi - \delta)}{(h(I^*) + \alpha_1)(m_1\rho + K(\rho - \varphi - \delta)) - \alpha_3 K(\rho - \varphi - \delta)}$$

$$V^* = \frac{K}{\rho}(\rho - \varphi - \delta) \tag{3.2}$$

where $h(I^*) = \frac{\alpha_1(m_1+I^*)-\alpha_2 I^*}{m_1+I^*}$.

Substituting the above into the system at equilibrium yields the expression for I^* after

some rearrangement. That is,

$$(\varphi+\gamma)(\alpha_1-\alpha_2+h)I^{*2}+[(\varphi+\gamma)(\alpha_1 m_1+m_1 h)-\alpha_1+\alpha_2-h-\varphi(\mu+\varphi+\gamma)]$$
$$I^*-\Lambda(\alpha_1 m_1+m_1 h)-\varphi m_1(\mu+\varphi+\gamma) \tag{3.3}$$

where $h=\frac{\alpha_1 m_1\rho+(\alpha_1-\alpha_3)K(\rho-\varphi-\delta)}{m_1\rho+K(\rho-\varphi-\delta)}$, where I^* is the positive solution of the quadratic equation (3.3).

For illustration, suppose, there is no effect of media report on the number of infected birds. This implies that $\delta=\alpha_2=\alpha_3=0$.

Then the endemic equilibrium satisfies

$$\Lambda-\alpha_1 SI-\alpha_1 SV+\mu I-\varphi S=0$$
$$\alpha_1 SI+\alpha_1 SV-\mu I-\varphi I-\gamma I=0$$
$$\rho V\left(1-\frac{V}{K}\right)-\varphi V=0.$$

From here we have that

$$\begin{aligned} V^* &= \frac{K}{\rho}(\rho-\varphi) \\ S^* &= \frac{(\mu+\varphi+\gamma)I^*}{\alpha_1 I^*+\frac{K\alpha_1}{\rho}(\rho-\varphi)} \end{aligned}$$

where I^* is the positive solution of the quadratic equation.

$$\alpha_1(\varphi+\gamma)I^{*2}-\left[\alpha_2\Lambda+\mu\rho-(\varphi+\gamma)\left(\frac{K\alpha_1}{\rho}(\rho-\varphi)-\varphi\right)\right]I^*-\frac{K\Lambda\alpha_1}{\rho}(\rho-\varphi)=0.$$

That is,

$$I=\frac{\alpha_1\Lambda+\mu\rho-(\varphi+\gamma)\left(\frac{K\alpha_1}{\rho}(\rho-\varphi)-\varphi\right)+\sqrt{\left[\alpha_1\Lambda+\mu\rho-(\varphi+\gamma)\left(\frac{K\alpha_1}{\rho}(\rho-\varphi)-\varphi\right)\right]^2+4\alpha_1(\varphi+\gamma)}}{2\alpha_1(\varphi+\gamma)}.$$

3.1 Analysis of the Disease Free Equilibrium

When modelling infectious diseases, our aim is to find out whether or not the infection will invade the community. We therefore carry out equilibrium and stability analysis to have a better understanding of the dynamics of the disease.

To determine the behaviour of the population near the equilibrium solution, we need to compute the linearization of the system, which is obtained as the Jacobian matrix of the system. Here we seek to establish the local stability of the disease free equilibrium. From system of the equation in (3.1), the Jacobian matrix is follows:

$J =$

$$\begin{pmatrix} -\varphi - \left(\alpha_1 - \frac{\alpha_2 I^*}{m_1+I^*}\right) I^* - \left(\alpha_1 - \frac{\alpha_3 V^*}{m_1 V^*}\right) V^* & \mu & \alpha_S^* - \frac{(2m_1+V^*)\alpha_2 S^* V^*}{(m_1+V^*)^2} \\ \left(\alpha_1 - \frac{\alpha_2 I^*}{m_1+I^*}\right) I^* + \left(\alpha_1 - \frac{\alpha_3 V^*}{m_1+V^*}\right) V^* & -(\mu + vp + \gamma) & \alpha_1 S^* - \frac{(2m_1+V^*)\alpha_3 S^* V^*}{(m_1+V^*)^2} \\ 0 & 0 & \rho - \frac{2\rho V^*}{K} - (\varphi + \delta) \end{pmatrix}$$

The Jacobian matrix of the disease equilibrium

$$E_0 = \left[\frac{\Lambda\rho(\rho m_1 + P)}{(\alpha_1 P + \varphi\rho)(\rho m_1 + P) - \alpha_3 P^2}, 0, \frac{K}{\rho}(\rho - \varphi - \delta)\right]$$

is given thus

$$J_0 = \begin{pmatrix} -(\varphi + T) & \mu & \alpha_2 S^* - \frac{(2m_1+V^*)\alpha_3 S^* V^*}{(m_1+V^*)^2} \\ T & -(\mu + \varphi + \gamma) & \alpha_1 S^* - \frac{(2m_1+V^*)\alpha_3 S^* V^*}{(m_1+V^*)^2} \\ 0 & 0 & -(\rho - \varphi - \delta) \end{pmatrix}$$

where

$$T = \frac{\alpha_1 P(m_1\rho + P) - \alpha_3 P^2}{\rho(m_1\rho + P)}, \quad S^* = \frac{\Lambda\rho(\rho m_1 + P)}{(\alpha_1 P + \varphi\rho)(\rho m_1 + P) - \alpha_3 P^2}, \quad V^* = \frac{K}{\rho}(\rho - \varphi - \delta).$$

The eigenvalues of the Jacobian matrix J_0 are found to be $\xi_1 = (-(\rho - \varphi - \delta)$.

$$\xi_2 = -\frac{1}{2}(\mu + 2\varphi + \gamma + T) + \sqrt{(\mu + 2\varphi + \gamma + T)^2 - 4[(\mu + \varphi + \gamma)(\varphi + T) - \mu T]}$$

$$\xi_= -\frac{1}{2}(\mu + 2\varphi + \gamma + T) - \sqrt{(\mu + \varphi + \gamma + \varphi + T)^2 - 4[(\mu + \varphi + \gamma)(\varphi + T) - \mu T]}$$

ξ_2 has negative real part if $(\mu + \varphi + \gamma)(\varphi + T) < \mu T$.

If all of the roots of the characteristic equation obtained from the Jacobian of the linearization of a system of ordinary differential equations about equilibrium arenegative or have negative real parts then that equilibrium is said to be locallyasymptotically

stable. That is, small perturbations from the equilibrium die away andthe system returns to the equilibrium.Hence this system is locally asymptotically stableat this disease free equilibrium,since all the eigenvalues of the system at disease free equilibrium has negative real parts. Therefore any small infection introduced into the poultry will die out and the system returns to equilibrium.

3.2 Analysis of the Endemic Equilibrium

At the endemic equilibrium point, the infection has spread everywhere in the poultry. Hence there is no susceptible population, that is, $S = 0$.

From system of the equation in (3.1), the Jacobian matrix is follows: $J =$

$$\begin{pmatrix} -\varphi - \left(\alpha_1 - \frac{\alpha_2 I^*}{m_1+I^*}\right) I^* - \left(\alpha_1 - \frac{\alpha_3 V^*}{m_1 V^*}\right) V^* & \mu & \alpha_S^* - \frac{(2m_1+V^*)\alpha_3 S^* V^*}{(m_1+V^*)^2} \\ \left(\alpha_1 - \frac{\alpha_2 I^*}{m_1+I^*}\right) I^* + \left(\alpha_1 - \frac{\alpha_3 V^*}{m_1+V^*}\right) V^* & -(\mu + vp + \gamma) & \alpha_1 S^* - \frac{(2m_1+V^*)\alpha_3 S^* V^*}{(m_1+V^*)^2} \\ 0 & 0 & \rho - \frac{2\rho V^*}{K} - (\varphi + \delta) \end{pmatrix}$$

At the endemic equilibrium $E_e = (S^*, I^*, V^*) = \left(0, \frac{\Lambda}{\mu}, \frac{K}{\rho}(p - \varphi - \delta)\right)$ (the Jacobian matrix is as follows:

$$J_E = \begin{pmatrix} -\varphi - \delta_1 - \delta_2 & \mu & 0 \\ \delta_1 + \delta_2 & -(\mu + \varphi + \gamma) & 0 \\ 0 & 0 & -(\rho - \varphi - \delta) \end{pmatrix}$$

where the endemic equilibrium point is

$$E_e = (S^*, I^*, V^*) = \left(0, \frac{\Lambda}{\mu}, \frac{K}{\rho}(\rho - \varphi - \delta)\right).$$

The characteristic equation at the endemic equilibrium is

$$\{-(\rho - \varphi - \delta) - \lambda\}\{\lambda^2 + (\delta_1 + \beta + \delta_2)\lambda + (\beta - \mu)(\delta_1 + \delta_2)\} = 0$$

where

$$\delta_1 = \frac{\alpha_1 \Lambda \mu m_1 + (\alpha_1 - \alpha_2)\Lambda^2}{\mu(\mu m_1 + \Lambda)} \quad \delta_2 = \frac{\alpha_1 m_1 \rho + (\alpha_1 - \alpha_2)K^2(\rho - \varphi - \delta)^2}{m_1\rho^2 + K\rho(\rho - \varphi - \delta)}, \quad \beta = \mu + \delta + \gamma.$$

The eigenvalues of the endemic equilibrium is obtained as follows

$$\begin{aligned}
\lambda_1 &= -(\rho - \varphi - \delta), \\
& \lambda^2 + (\delta_1 + \beta + \delta_2)\lambda + (\delta + \gamma)(\delta_1 + \delta_2) = 0 \\
\lambda &= \frac{-(\delta_1 + \beta + \delta_2) \pm \sqrt{(\delta_1 + \beta + \delta_2)^2 - 4(\delta + \gamma)(\delta_1 + \delta_2)}}{2} \\
\lambda_2 &= \frac{-(\delta_1 + \beta + \delta_2) - \sqrt{(\delta_1 + \beta + \delta_2)^2 - 4(\delta + \gamma)(\delta_1 + \delta_2)}}{2} \\
\lambda_3 &= \frac{-(\delta_1 + \beta + \delta_2) + \sqrt{(\delta_1 + \beta + \delta_2)^2 - 4(\delta + \gamma)(\delta_1 + \delta_2)}}{2}
\end{aligned}$$

λ_3 has negative real part if$\delta_1 + \delta_2 < 0$.

Hence this system is locally asymptotically stableat the endemic equilibrium, since all the eigenvalues of the system at endemic equilibrium has negative real parts.

4. Analysis of the Stability Using Basic Reproductive Ratio

The concept of the basic reproductive ratio R_0 in epidemiology is fundamental as it serves as a threshold parameter that governs the spread of infectious disease in a population [2]. The basic reproductive ratio R_0 is defined as the expected number of secondary infections caused by an infective individual upon entering a totally susceptible population [3], [4], and [5]. The basic reproductive ratio is also defined as the spectral radius, that is, the dominant eigenvalue of the next-generation matrix [3]. This quantity is not only important in describing the infectious power but also, but can also supply information for controlling the spread of infection [6].

If $R_0 < 1$, the each infected individual in its entire period of infectivity will produce less than one infected individual on average. The disease free equilibrium is locally asymptotically stable. Thus the disease will be wiped out of the population. If $R_0 > 1$, then there is a cause for alarm as this implies that each infected individual in its entire infective period having contact with susceptible individual will produce more one infected individual on the average, which then will lead to the disease invading the susceptible population[2].

Construction of Basic Reproductive Number

The $v_{i,j}$ entry of the transition matrix V is the rate individuals in stage j progress to Stage i. The $f_{i,j}$ entry of the infection matrix F is the number of new infections at stage

j caused by contacts with diseased individuals in stage i. The $g_{i,j}$ entry of the next generation matrix $G = FV^{-1}$ is the expected number ofsecondary infections produced in compartment i by an index case initially in compartment j.

• G has a positive real eigenvalue R_0 which is at least as large in modulus as all other eigen- Values of G. This eigen value is the logical candidate for the basic reproduction number [7]. The linearity of the disease free equilibrium is governed by the basic reproductive ratio R_0. Using the next-generation matrix method, we have

$$F = \begin{pmatrix} \frac{\alpha_1\Lambda\rho(\rho m_1+P)}{(\alpha_1 P+\varphi\rho)(\rho m_1+P)-\alpha_3 P^2} & 0 & \frac{\Lambda(\alpha_1 P(\rho m_1+P)-\alpha_3 P^2)}{(\alpha_1 P+\varphi\rho)(\rho m_1+P)-\alpha_3 P^2} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \mu+\varphi+\gamma & 0 & 0 \\ \alpha_1 S^* & TV^* & TS^* \\ 0 & 0 & \varphi+\delta-\rho\left(1-\frac{V^*}{K}\right) \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu+\varphi+\gamma} & \frac{-(\alpha_1 S^*-\mu)}{(TV^*+\varphi)(\mu+\varphi+\gamma)} & 0 \\ 0 & \frac{1}{TV^*+\varphi} & 0 \\ 0 & \frac{-TS^*}{(TV^*+\varphi)\left(\varphi+\delta-\rho\left(1-\frac{V^*}{K}\right)\right)} & \frac{1}{\varphi+\delta-\rho\left(1-\frac{V^*}{K}\right)} \end{pmatrix}$$

$$G = \begin{pmatrix} \frac{\alpha_1 S^*}{\mu+\varphi+\gamma} & \frac{-\left(\varphi+\delta-\rho\left(1-\frac{V^*}{K}\right)\right)a_1 S^*(\alpha_1 S^*-\mu)}{(TV^*+\varphi)(\mu+\varphi+\gamma)\left(\varphi+\delta-\rho\left(1-\frac{V^*}{K}\right)\right)} & \frac{-TS^*}{\left(\varphi+\delta-\rho\left(1-\frac{V^*}{K}\right)\right)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The basic reproductive ratio is the spectral radius of G, which

$$R_0 = \frac{\alpha_1\Lambda\rho(\rho m_1+P)}{(\mu+\varphi+\gamma)(\alpha_1 P+\varphi\rho)(\rho m_1+P)-\alpha_3 P^2}.$$

And represent the number of secondary infection that each infectious bird is expected to produce, while its remains infectious when introduce the population of entirely susceptible birds. If $R_0 < 1$, that is the average number of secondary infection is less than one.

Therefore within a short while the infection will die out from the population. Hence at $R_0 < 1$, the disease free equilibrium is locally asymptotically stable.

5. Summary and Conclusion

It is true that provision of information influences individual risk perception and shapes the evolution of epidemics. Because of its sporadic nature andability to easily be carried from flock to flock, it is important that farmers remain vigilant in watching for its re-emergence.

In this work, we have formulated a deterministic mathematical model of vector-host transmission dynamics of fowl pox in poultry to investigate the impact of media coverage in the reduction and control of fowl pox in poultry. The population is divided into susceptible birds (S), infected birds (I) and the vector population (V). The compartmental model resulted in an ordinary differential equation that includes the effect of media coverage on reporting the numberof infections.

The method of dynamical system theory is employed in analysing the equilibrium stability of the model at both disease free equilibrium and endemic equilibrium. Appropriate conditions for the local asymptotic stability of both equilibrium points have been established. A threshold parameter R0 (the basic reproductive ratio) was also derived analytically to discuss the local stability of the disease free equilibrium.

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