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Mathematical Modelling Of the Effect of Non-Drug Compliance on Transmission Dynamics of Typhoid.

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Abstract: *Typhoid fever is an infectious disease caused by salmonella typhi. We formulate a mathematical modeling of transmission dynamics of typhoid using non-linear ordinary differential equations, in order to understand how the disease is spread and to predict the future behaviour of the model. Epidemiological threshold, R_0, which is the condition for the disease spread is calculated. We obtain the disease-free equilibrium which is locally and globally stable respectively. The local stability of the endemic equilibrium of the model is also obtained.*

Keywords: *Non-linear ordinary differential equations, Typhoid fever, local stability, global stability.*

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I. Introduction

Typhoid fever is an infectious disease caused by the bacterial salmonella typhi. It is transmitted to human by taking the food or water contaminated by the urine or faeces of an infected human or carriers. Globally it is estimated that typhoid causes over 16 million cases of illness each year, resulting in over 600,000 deaths [3]. While improvements in water and sanitation led to the elimination of typhoid from most developed countries during the twentieth century, the global burden of typhoid fever has recently been estimated to be between 13.5 and 26.9 million episodes and 190,000 to 216,000 deaths annually. [1].

Getachew et al (2017) modelled an optimal control of typhoid fever disease with cost-effective strategies without considering the effect of non-drug compliance in the human population. In their model, they took into consideration sanitation, proper hygiene and vaccination as their control strategies.

Nthiiri et al. formulated a mathematical model based on system of ordinary differential equations to study the dynamics of typhoid fever incorporating protection against infection. They obtained the existence of steady states of the model and basic reproduction number. They carried out stability analysis of their model to determine the conditions that favour the spread of the disease in a given population. They also performed numerical simulation of their model, which showed that an increase in protection led to disease prevalence in a population.

Various papers have been written on mathematical modeling of typhoid recently without considering the effect of non-drug compliance on the spread of the disease. Non-drug compliant human are those who are given medication by their doctors but do not take it as prescribed.In view of the above, we formulate a deterministic model to investigate the effects of non-drug compliance on transmission dynamics of typhoid. The model also takes into consideration health education as a control strategy of non-drug compliant humans.

II. Model Formulation

The model sub-divides the total human population denoted by N_H, into subpopulations of susceptible human (S_H), Infected human (I_H), carriers (C_H), Non-drug Compliant Human (I_{NH}) and Recovered Human (R_H).

2.1 Assumptions of the Model

The following assumptions were made in order to formulate the equations of the model:

a) Some infectious human who take their drug as prescribed by their doctors get treated fully and move to the recovered compartment
b) Some infectious human who do not take their drug as prescribed by their doctors get treated partially and move to the non-drug compliant human compartment.

Hence, we have the following differential equations:

$$\frac{ds_H}{dt} = \wedge_H - \frac{\alpha C_H S_H}{N_H} + \varphi R_H - \mu_H S_H \quad \text{---------- (2.1)}$$

$$\frac{dI_H}{dt} = \frac{\alpha C_H S_H}{N_H} - \sigma I_H - {}_{\omega}\beta I_H - \tau I_H - \mu_H I_H \quad \text{---------- (2.2)}$$

$$\frac{dI_{NH}}{dt} = (1-\rho)\tau I_H - r\tau I_{NH} - \mu_N I_{NH} - \mu_N I_{NH} \quad \text{---------- (2.3)}$$

$\frac{d_{Gt}}{dt} = \omega\beta I_{H-} \Upsilon_{CH} - \sigma C_{H-} \mu_H C_H$ ---------- (2.4)

$\frac{dR_H}{dt} = \rho\tau I_{H-} \varphi R_H + r\tau I_{NH} + \Upsilon C_H - \mu_H R_H$ ---------- (2.5)

where r = education on drug use
β = rate of progression from infective to carrier
γ = recovery rate
φ = loss of immunity rate
α = rate of infection
r = typhoid fever-induced death
τ = drug
ω = fraction of symptomatic typhoid patients who become carriers

$\wedge_H$ = recruitment rate
μ_H = natural death rate
μ_N = death due to non-drug compliance
ρ = proportion of infected human with drug compliance
(1 - ρ) = proportion of infected human with non-drug compliance

III. Model Analysis

3.1 Local Stability of Disease-Free Equilibrium

It could be seen that the disease free equilibrium of the model is locally stable. In the absence of infection, the model reduces to

$\frac{dS_H}{dt} = \wedge_H + \varphi R_H - \mu_H S_H$ ---------- (3.1)

$\frac{dR_H}{dt} = - (\varphi + \mu_H) R_H$ ---------- (3.2)

The disease free equilibrium points of (3.1) – (3.2) is $E_0 = \left(\frac{\Lambda_H}{\mu_H}, 0\right)$

We next solve the system (3.1) – (3.2)
From (3.1),

$\frac{dR_H}{dt} = - (\varphi + \mu_H) R_H$

$R_H = Ae^{-(\varphi + \mu_H)t}.e^C = Ae^{-(\varphi + \mu_H)t}$

Then $R_H = R_H^0 Ae^{-(\varphi + \mu_H)t}$ where $R_H(0) = R_H^0$

Also, $S_H = \frac{\Lambda_H}{\mu_H} - R_H^0 Ae^{-(\varphi + \mu_H)t} + (S_H^0 - \frac{\Lambda_H}{\mu_H} + R_H^0)\, e^{-\mu_H t}$

where $S_H(0) = S_H^0$.

This shows that the recovered and susceptible population converge to their equilibrium solution $\left(\frac{\Lambda_H}{\mu_H}, 0\right)$ as t increases. Hence the disease-free equilibrium is locally stable.

3.2 Reproductive Number (R_0)

Reproductive number is calculated in order to know whether the disease dies out of the population or persists in the population. If $R_0 < 1$, then the disease dies out of the population, but if $R_0 > 1$, the disease persists in the population. Reproductive number is the number of secondary infectious humans that are infected by a primary infectious one in a susceptive population. But before we calculate R_0, we shall scale our model in terms of proportion of quantity rather than actual population. Hence we have the transformation;

$s_h = \frac{S_H}{N_H}$; $i_h = \frac{I_H}{N_H}$; $i_{nh} = \frac{I_{NH}}{N_H}$; $c_h = \frac{C_H}{N_H}$; $r_h = \frac{R_H}{N_H}$.

Differentiating the fraction with respect to time t gives

$\frac{d_{sh}}{dt} = \lambda_h(1 - s_h) - \alpha c_h s_h + \varphi r_h + \sigma s_h (c_h + i_h) + \mu_N s_h i_{nh}$ ---- (3.3)

$\frac{di_h}{dt} = \alpha c_h s_h - (\sigma + \omega\beta + \tau + \lambda_h) i_h + \sigma i^2{}_h + \sigma c_h i_h + \mu_N i_h i_{nh}$ ---- (3.4)

$\frac{di_{nh}}{dt} = (\tau - \rho\tau)\, i_h - (r\tau + \lambda_h) i_{nh} + \sigma i_{nh} (i_h + c_h)$ ---- (3.5)

$\frac{dr_h}{dt} = \rho\tau\, i_h - (\varphi + \lambda_h)\, r_h + r\tau i_{nh} + (\Upsilon + \sigma c_h) c_h + \sigma r_h i_h + \mu_N r_h i_{nh}$ ---- (3.6)

where E_0 is obtained by setting the right-hand side of (3.3) – (3.6) to zero. This implies that $E_0 = (1, 0, 0, 0)$.
The reproductive number is obtained by expressing (3.3) – (3.6) as the difference between the rate of new infection in each infected compartment F and the rate of transfer between each infected compartment G. Therefore, R_0 is the maximum eigenvalue of D given as $R_0 = \frac{\omega\alpha\beta}{A_T b_t}$ where

$A_T = \sigma + \omega\beta + \tau + \lambda_h$ and $bt = \gamma + \sigma + \lambda_h$.

3.3 Global Stability of disease-free equilibrium

Theorem1: The disease-free equilibrium $E_0 = (1,000)$ of (3.3) – (3.6) is globally stable if $R_0 \leq 1$ and unstable of $R_0 > 1$.

Proof: Consider the Lyapunov function

$L = \omega\beta i_h + A_T c_h$

$L^1 = \omega\beta \frac{di_h}{dt} + A_T \frac{d_{ch}}{dt}$

Further expression yields

$L^1 = A_T c_h (R_0 s_h - 1) - \frac{1}{bt} (A_T \sigma^2 c_h + A_T c_h \mu_N i_{nh} - \omega\beta\sigma i^2_h - A_T i_h \sigma c_h) \leq A_T c_h (R_t s_h - 1) \leq 0$ if $R_t \leq 1$

Hence, this shows that the disease – free equilibrium is globally stable.

3.4 Local Stability of endemic equilibrium

We shall employ the following theorem stated and proved by Mccluskey and Van Driessche [5] to demonstrate the local stability of endemic equilibrium E_1.

Theorem2: Let M be a 4 x 4 matrix. If tr(M), det(M) and det ($M^{(1)}$) are all negative, then all eigenvalues of M have negative real part.

Proof: We first compute the Jacobian matrix of (3.3) – (3.6). i.e.

$$J_E = \begin{bmatrix} r_1 & rs_{h-\emptyset\mu_N s_h - \emptyset} & -\alpha s_{h+rs_h-\emptyset} \\ \alpha c_h r_2 \ \mu_N i_h & & \alpha s_h \\ 0 & \tau - \rho\tau + \sigma i_{nh} & {}_{r_3}\sigma i_{nh} \\ 0 & \omega\beta + \sigma c_h & -\mu_{Nc_h r_4} \end{bmatrix}$$

where $r_1 = -(\lambda_h + \alpha\ c_h + \varphi - rc_h - ri_h - \mu_N i_{nh})$

$r_2 = -A_T + 2\sigma i_h + \sigma c_h + \mu_N i_{nh}$

$r_3 = -(r\tau + \lambda_h - \sigma i_h - \sigma c_h)$

$r_4 = -(\lambda_h - \sigma i_h + \Upsilon + \sigma + 2\sigma c_h + \mu_N i_{nh})$

From the Jacobian matrix, the first additive compound matrix employed in [Mouldowrey, (1990), L_1 et al. 1995)] is given by

[1]

$$J_E{}^{[1]} = \begin{bmatrix} -(A - \mu_N i_{nh}) & 0 & 0 & 0 \\ 0 & -(\beta - \mu_N i_{nh}) & 0 & 0 \\ 0 & 0 & -C & 0 \\ 0 & 0 & 0 & -(D + \mu_N i_{nh}) \end{bmatrix}$$

From the Jacobian matrix and the first additive compound matrix, we have

$\det(J_{E1}) = -(\lambda_h - \varphi\ c_h - \varphi i_{nh} + \varphi\ (1 - i_h) \left[-\frac{\alpha s_h c_h + \sigma \tau_h^2}{s_h c_h i_{nh}}\right]$

showing that the endemic equilibrium E_1 is locally stable.

IV. Conclusion

The formulated model predicted the reduction of the non-drug compliant humans as well as other disease classes by using education (r), as a control strategy. The effect of non-drug compliance on transmission of typhoid suggested that non-drug compliance may increase the spread of typhoid. Therefore, education control strategy (r) should be adopted for typhoid infection.

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