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A Mathematical Model Explaining the Pathogenicity of the Pathogenic Strain of E.coli

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Abstract

The objective of this study is to establish a dynamical model for the spread of *Escherichia coli* within a community, while also identifying the key parameters influencing its propagation. Through an in-depth analysis of the dynamical system, two equilibrium points were discerned. Furthermore, the numerical solution of the system revealed the significant impact of various parameters on bacterial dissemination. These parameters encompassed the contact rates among healthy individuals, infected persons, and the bacteria itself. Additionally, the level of compliance with sanitation practices among infected individuals played a pivotal role. Gaining insights into the influence of these parameters holds substantial promise for effectively managing both bacterial spread and associated diseases.

Key words: Dynamical system, E.coli, contact rate, equilibrium point.
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1 Introduction

Escherichia coli, a Gram-negative bacterium belonging to the Enterobacteriaceae family, is the leading cause of urinary tract infections [1]. While most species of *E. coli* are non-pathogenic, it can act as an opportunistic pathogen, causing infections in individuals with weakened immune systems [2]. One particularly pathogenic strain of *E. coli* is *E. coli* 0157:H7, which produces a potent toxin that can lead to severe illness [3]. The most common symptoms of *E. coli* infection are diarrhea and abdominal pain but fever and vomiting are rare symptoms [4]. The infection begins after 2-5 days of exposure to the bacteria and the infected people can pass the infection to other individuals [5]. *E. coli*: 0157:H7 can be found in food, water, soil or surfaces contaminated with feces of the infected person [6]. In the biological field, many different research papers aimed to understand the mechanism of drugs in treatment. Because of multidrug resistance of *E. coli* and its role as a common cause for food poisoning, we try by using a mathematical model to understand the strategy of preventing *E. coli* infection spread and how bacterial concentration plays a role in development of an infection [7,8].

2 Formulation of Mathematical Modeling

Let S(t) denote the count of the susceptible population at time t, I(t) represent the number of individuals infected at time t, and T(t) represent the count of individuals under treatment at time t. Additionally, let B denote the concentration of bacteria in the environment at time t.

$$\begin{aligned}
\frac{dS}{dt} &= \pi - (1-\beta)(\Psi I + \sigma B)S + \rho\theta I + \alpha T - \gamma S \\
\frac{dI}{dt} &= (1-\beta)(\Psi I + \sigma B)S - (\theta + \gamma + \mu)I \\
\frac{dT}{dt} &= q\theta I - (\alpha + \gamma)T \\
\frac{dB}{dt} &= (1-k)\varphi I - (\mu_B + \gamma_B - \pi_B)B
\end{aligned} \tag{2.1}$$

where π and π_B is the natural birth and bacterial growth respectively. $\beta \in (0,1)$ is the homestead-isolation rate of susceptibility due to the media coverage. Ψ and σ are the probabilities of the interaction between susceptible individuals with the infected person and bacteria respectively. θ is the treatment rate of infected person. ρ represents infected individuals who do

not receive full regimen treatment while q represents individuals who get full treatment, with $\rho + q = 1$. γ and γ_B are the natural death rate of the infected person and bacteria respectively. α represents the transition rate from T class to S class. μ and μ_B represents the disease death rate and bacterial death rate with sanitation respectively. $(1-k)\varphi$, k is a level of adherence to sanitation by an infected individual, and φ is each individual's contribution to the pathogen community.

Theorem 2.1. *All solutions* $(S(t), I(t), T(t), B(t))$ *of model (2.1) are bounded.*

Proof.
We demonstrate the concept of population constraints. The overall size of the population remains constant as follows:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dT}{dt}$$

$$= \pi - \gamma S - \gamma I - \mu I - \gamma T$$

$$= \pi - \gamma(S + I + T) - \mu I$$

$$= \pi - \gamma N - \mu I$$

$$\leq \pi - \gamma N.$$

By the using of Gronwall theorem [9] we obtain

$$N(t) \leq \frac{\pi}{\gamma}(1 - exp(-\gamma t)) + N(0)\exp(-\gamma t)$$

. We have $N(t) \leq \frac{\pi}{\gamma}$, as $t \to \infty$.

$$\frac{dB}{dt} + (\mu_B + \gamma_B - \pi_B)B(t) = \varphi(1-k)I$$

.

$$B(t) = C_1 exp(-\mu_B + \gamma_B - \pi_B)t + \frac{(1-k)\Phi I}{\mu_B + \gamma_B - \pi_B} \leq \frac{(1-k)\Phi L}{\mu_B + \gamma_B - \pi_B}$$

. Hence, $B(t)$ and $N(t)$ are subject to bounds.

3 The Local Stability Analysis

3.1 The existence of disease-free equilibrium:

When there is no infection, the equilibrium point can be defined as a steady state solution and it has a unique solution. It is denoted as $E^0 = (S^0, 0, 0, 0)$, where $S^0 = \frac{\pi}{\gamma}$, and its local stability can be determined by examining the eigenvalues . The Jacobian matrix for the system can be described as follows:

$$J(E^\nu) = \begin{bmatrix} -(\tau_1+\gamma) & \rho\theta-\tau_2 & \alpha & -\tau_3 \\ \tau_1 & -\omega_1 & 0 & \tau_3 \\ 0 & q\theta & -(\alpha+\gamma) & 0 \\ 0 & \tau_4 & 0 & -\omega_B \end{bmatrix}$$

where $\tau_1 = (1-\beta)(\Psi I^\nu + \sigma B^\nu)$, $\tau_2 = (1-\beta)(\Psi S^\nu)$ $\tau_3 = (1-\beta)\sigma S^\nu$ $\tau_4 = (1-k)\varphi$, $\omega_B = (\mu_B + \gamma_B - \pi_B)$ and $\omega_1 = \theta + \gamma + \mu - \tau_2$

Theorem 3.1. *If ω_1 and ω_B are both positive, then the disease-free equilibrium point E^0 exhibits local asymptotic stability.*

Proof.
Considering the characteristics equation of the Jacobian matrix at the disease-free equilibrium point as $|J(E^0) - \lambda I| = 0$, and expanding the determinant by the first column, one of the eigenvalues is $\lambda = \gamma$. The remaining three eigenvalues can be obtained by expanding the determinant of the resulting 3×3 matrix, yielding the equation $\lambda^3 + k_1\lambda^2 + k_2\lambda + k_3 = 0$, where $k_1 = \alpha + \gamma + \omega_1 + \omega_B$.
$k_2 = (\alpha+\gamma)(\omega_1\omega_B + \omega_1\omega_B + \tau_3\tau_4)$.
$k_3 = (\alpha+\gamma)(\omega_1\omega_B + \tau_3\tau_4)$.
$k_1k_2 - k_3 = (\omega_1+\omega_B)(\alpha^2 + \tau_3\tau_4 + (\omega_1+\gamma) + (\omega_1+\gamma)) + \alpha(\omega_1+\omega_B+2\gamma)$.
We have $k_1 > 0, k_2 > 0, k_3 > 0$ and $k_1k_2 - k_3 > 0$. By using the well-known Routh-Hurwitz criterion [11]. Then all eigenvalues have negative real part, the proof is complete.

3.2 The existence of endemic equilibrium:

When the infection is present, the endemic equilibrium point exists and can be defined as the steady state solution ($I > 0$). These equilibrium points are characterized by the presence $E^1 = (S^1, I^1, T^1, B^1)$, where $S^1 = \frac{\pi+(\alpha d_2+\rho\theta)I^1}{\gamma+(1-\beta)(\Psi+\sigma d_1)I^1}$, $T^1 = d_1 I^1, B^1 = d_2 I^1$, while I^1 is a positive root for the following equation:

$$\xi(I^1)^2 + \xi_2 I^1 + \xi_3 = 0 \tag{3.2}$$

with $\xi_1 = (1-\beta)(\alpha d_1 + \theta)(\sigma d_2 + \Psi)$, $\xi_2 = (1-\beta)(\pi - (\theta + \mu + \gamma))(\sigma d_2 + \Psi)$ and $\xi_3 = -\gamma(\gamma + \mu + \theta)$, where $d_1 = \frac{q\theta}{\alpha+\gamma}$ and $d_2 = \frac{(1-k)\varphi}{\omega_B}$. Clearly, by using Descartes rule of signs [10], equation (3.2) has the unique positive root given by I^1 if and only if one of the following conditions hold: $\xi_2 > 0$ or $\xi_2 < 0$.

Theorem 3.2. *If ω_1 and ω_B are both positive and $\tau_3\tau_4 < \omega_1\omega_B$, then the equilibrium point E^1 of the dynamical system (2.1) is locally asymptotically stable.*

Proof.
Considering the characteristics equation $\mid J(E^1) - \lambda I \mid = 0$ of the Jacobian matrix, we have $\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4 = 0$, where $b_1 = \alpha + 2\gamma + \tau_1 + \omega_1 + \omega_B$.

$b_2 = \gamma^2 + \tau_1\tau_2 + \omega_1\omega_B - \tau_3\tau_4 + \tau_1\omega_1 + \tau_1\omega_B + \alpha(\gamma + \tau_1 + \omega_1 + \omega_B) + \gamma(\tau_1 + 2(\omega_1 + \omega_B))$.

$b_3 = \tau_1(\tau_2 + \omega_1)\omega_B + \gamma^2(\omega_1 + \omega_B) + \gamma(2(\omega_1\omega_B - \tau_3\tau_4) + \tau_1(\tau_2 + \omega_1 + \omega_B)) + \alpha(\omega_1\omega_B - \tau_3\tau_4 + \gamma(\omega_1 + \omega_B) + \tau_1(\tau_2 - \theta + \omega_1 + \omega_B))$.

$b_4 = \gamma(\gamma(\omega_1\omega_B - \tau_3\tau_4) + \tau_1((\tau_2 + \omega_1)\omega_B - \tau_3\tau_4)) + \alpha(\gamma(\omega_1\omega_B - \tau_3\tau_4) + \tau_1((\tau_2 - \theta + \omega_1)\omega_B - \tau_3\tau_4)$.

$b_1b_2 - b_3 = 2\gamma^3 + (\tau_1 + \omega_1)(\tau_1(\tau_2 + \omega_1) - \tau_3\tau_4) + ((\tau_2 + \omega_1)^2\omega_B - \tau_3\tau_4 + (\tau_1 + \omega_1)^2)\omega_B + (\tau_1 + \omega_1)\omega_B^2 + \alpha^2(\gamma + \tau_1 + \omega_1 + \omega_B) + \gamma^2(3\tau_1 + 4(\omega_1 + \omega_B)) + \alpha(3\gamma^2 + \tau_1^2 + (\omega_1 + \omega_B)^2 + 4\gamma(\tau_1 + \omega_1 + \omega_B) + \tau_1(\theta + 2(\omega_1 + \omega_B))) + \gamma(\tau_1^2 + 2(\omega_1 + \omega_B)^2 + \tau_1(\tau_2 + 4(\omega_1 + \omega_B)))$.

$b_1b_2b_3 - b_3^2 - b_1^2b_4 = 2\gamma^5(\omega_1 + \omega_B) + \gamma^4(4(\omega_1 + \omega_B)^2 + \tau_1(2\tau_1 + 5(\omega_1 + \omega_B))) + \tau_1(\tau_2 + \omega_1)\omega_B(\tau_1^2(\tau_2 + \omega_1 + \omega_B) + (\omega_1 + \omega_B)(\omega_1\omega_B - \tau_3\tau_4) + \tau_1(-\tau_3\tau_4 + \tau_2\omega_1 + (\omega_1 + \omega_B)^2)) + \gamma^3(2(\omega_1 + \omega_B)(-2\tau_3\tau_4 + \omega_1^2 + 4\omega_1\omega_B + \omega_B^2) + \tau_1^2(3\tau_2 + 4(\omega_1 + \omega_B)) + \tau_1(6\tau_3\tau_4 + 8(\omega_1 + \omega_B)^2 + \tau_2(5\omega_1 + 3\omega_B))) + \alpha^3(\gamma^2(\omega_1 + \omega_B) + \tau_1^2(\tau_2 - \theta + \omega_1 + \omega_B) + (\omega_1 + \omega_B)(\omega_1\omega_B - \tau_3\tau_4) + \tau_1(\tau_3\tau_4 + \tau_2\omega_1 - \theta\omega_1 + (\omega_1 + \omega_B)^2) + \gamma((\omega_1 + \omega_B)^2 + \tau_1(\tau_2 - \theta + 2(\omega_1 + \omega_B)))) + \alpha^2(4\gamma^3(\omega_1 + \omega_B) + \tau_1^3(\tau_2 - \theta + \omega_1 + \omega_B) + (\omega_1 + \omega_B)^2(\omega_1\omega_B - \tau_3\tau_4) + \tau_1^2(3\tau_3\tau_4 + (\tau_2 - \theta + \omega_1)(\theta + 2\omega_1) + (\tau_2 + \theta + 4\omega_1)\omega_B + 2\omega_B^2) + \tau_1(\omega_1^2(\tau_2 - \theta + \omega_1) + \omega_1(\tau_2 + \theta + 4\omega_1)\omega_B + (\theta + 4\omega_1)\omega_B^2 + \omega_B^3 + \tau_3\tau_4(-\theta + 2(\omega_1 + \omega_B))) + \gamma^2(5(\omega_1 + \omega_B)^2 + \tau_1(4\tau_2 - 3\theta + 9(\omega_1 + \omega_B))) + \gamma((\omega_1 + \omega_B)(-4\tau_3\tau_4 + \omega_1^2 + 6\omega_1\omega_B - 4\tau_3\tau_4 + \omega_B^2) + \tau_1^2(5\tau_2 - 4\theta + 6(\omega_1 + \omega_B)) + \tau_1(6\tau_3\tau_4 + \theta(-3\omega_1 + \omega_B) + 7(\omega_1 + \omega_B)^2 + \tau_2(5\omega_1 + \omega_B)))) + \gamma(2(\omega_1 + \omega_B)(\omega_1\omega_B - \tau_3\tau_4)^2 + \tau_1^3(2\tau_3\tau_4 + (\tau_2 + \omega_1 + \omega_B)^2) + \tau_1^2(\tau_2^2(\omega_1 + \omega_B) + (\omega_1 + \omega_B)(\tau_3\tau_4\omega_1^2 + 6\omega_1\omega_B + \omega_B^2) + \tau_2(-3\tau_3\tau_4 + 2\omega_1^2 + 8\omega_1\omega_B + 3\omega_B^2)) + \tau_1(2\tau_3^2\tau_4^2 - \tau_3\tau_4(\omega_1^2 + 4\omega_1\omega_B + \omega_B^2 + \tau_2(3\omega_1 + \omega_B)) + \omega_B(4\omega_1(\omega_1 + \omega_B)^2 + \tau_2(4\omega_1^2 +$

$3\omega_1^2\omega_B + \omega_B^2)))) + \gamma^2(\tau_1^3(\tau_2 + \omega_1 + \omega_B) + 4(\omega_1 + \omega_B)^2(\omega_1\omega_B - \tau_3\tau_4) + \tau_1^2(\tau_2^2 + 7\tau_3\tau_4 + 5(\omega_1 + \omega_B)^2 + \tau_2(6\omega_1 + 5\omega_B)) + \tau_1(\tau_2(-2\tau_3\tau_4 + (3\omega_1 + \omega_B)(\omega_1 + 2\omega_B)) + (\omega_1 + \omega_B)(\tau_3\tau_4 + 3(\omega_1^2 + 4\omega_1\omega_B + \omega_B^2)))) + \alpha(5\gamma^4(\omega_1 + \omega_B) + (\omega_1 + \omega_B)(\tau_3\tau_4 - \omega_1\omega_B)^2 + \tau_1^3(2\tau_3\tau_4 + (\tau_2 + \omega_1)(\tau_2 - \theta + \omega_1)2(\tau_2 + \omega_1)\omega_B + \omega_B^2) + \tau_1^2(\tau_2^2\omega_1 + \omega_1^2(\omega_1 - \theta) + \omega_1(\theta + 4\omega_1)\omega_B + (\theta + 4\omega_1)\omega_B^2 + \omega_B^3 + \tau_2(-2\tau_3\tau_4 - \theta\omega_1 + 2\omega_1^2 + \theta - \omega_B + 4\omega_1\omega_B + \omega_B^2) + \tau_3\tau_4(\theta + 2(\omega_1 + \omega_B))) + \gamma^2(3(\omega_1 + \omega_B)(-2\tau_3\tau_4 + \omega_1^2 + 4\omega_1\omega_B + \omega_B^2) + \tau_1^2(7\tau_2 - 3\theta + 9(\omega_1 + \omega_B)) + \tau_1(11\tau_3\tau_4 + \theta(-3\omega_1 + \omega_B) + 14(\omega_1 + \omega_B)^2 + \tau^2(9\omega_1 + 4\omega_B))) + \gamma^3(8(\omega_1 + \omega_B)^2 + \tau_1(5\tau_2 - 2(\theta - 6(\omega_1 + \omega_B)))) + \gamma(4(\omega_1 + \omega_B)^2(-\tau_3\tau_4 + \omega_1\omega_B) + \tau_1^3(2\tau_2 - \theta + 2(\omega_1 + \omega_B)) + \tau_1^2(\tau_2^2 + 10\tau_3\tau_4 + \theta(-3\omega_1 + \omega_B) + 7(\omega_1 + \omega_B)^2 + \tau^2(8\omega_1 + 6\omega_B)) + \tau_1(2\theta(\omega_1^2 + \omega_1\omega_B + \omega_B^2) + 4(\omega_1 + \omega_B)(\omega_1^2 + 3\omega_1\omega_B + \omega_B^2) + \tau^2(-\tau_3\tau_4 + 4\omega_1^2 + 6\omega_1\omega_B + \omega_B^2) + \tau_3\tau_4(-2\theta + 5(\omega_1 + \omega_B)))) + \tau_1(\tau_3^2\tau_4^2 + \tau_3\tau_4(-\omega_1\omega_B + \theta(\omega_1 + \omega_B) - \tau^2(2\omega_1 + \omega_B)) + \omega_B(\tau^2\omega_1(2\omega_1 + \omega_B) + (\omega_1 + \omega_B)(\theta\omega_B + 2\omega_1(\omega_1 + \omega_B)))))).$

It is easy to observe that from condition $\omega_1 > 0$, $\omega_B > 0$ and $\tau_3\tau_4 < \omega_1\omega_B$ we have $b_1 > 0$, $b_2 > 0$, $b_3 > 0$ and $b_4 > 0$, $b_1b_2 > b_3$ and $b_1b_2b_3 > b_3^2 + b_1^2b_4$. By using the well-known Routh-Hurwitz criterion [11] , the proof is complete.

4 Numerical Analysis

Numerical solutions have an important role in verification of our finding of the model. We study the effect of one parameter on disease spread and with the other parameter fixed here, we study the contact rate of susceptible individuals with the infected one (Ψ), and the effect of the contact rate of susceptible individuals with the bacteria from the surrounding environment (σ). The other parameter is the compliance of sanitation of the infected person (k).

5 Discussion

In our investigation, we delve into the impact of three distinct parameters on the spread of the disease, maintaining the remaining variables at a fixed value. To begin, we scrutinize the contact rate between susceptible individuals and those infected (Ψ). Referring to Figure (1-A), a noticeable trend emerges: when $\Psi = 0.15$, a high infection rate is evident; conversely, with $\Psi = 0.075$, the infection rate shows a marked decrease. This observation highlights the disease's tendency to dwindle as Ψ diminishes, assuming other parameters remain constant. To further validate our insights, we turn our attention to the contact rate between susceptible individuals and bacteria within the

surrounding environment (σ). As depicted in Figure (1-B), when $\sigma = 0.125$, a pronounced spike in infection rate is observed. Conversely, as σ decreases, the infection rate follows suit, emphasizing the importance of this parameter in influencing disease transmission dynamics. Exploring yet another factor, we assess the role of sanitation compliance (k) among infected individuals. This parameter encapsulates an individual's ability to propagate the disease within their community. Evidenced by Figure (1-C), a high infection rate is recorded when $k = 0$, indicating a lack of compliance. In contrast, as k increases, a corresponding decrease in the infection rate is witnessed. This underscores the significant impact of sanitation practices on curbing disease dissemination.

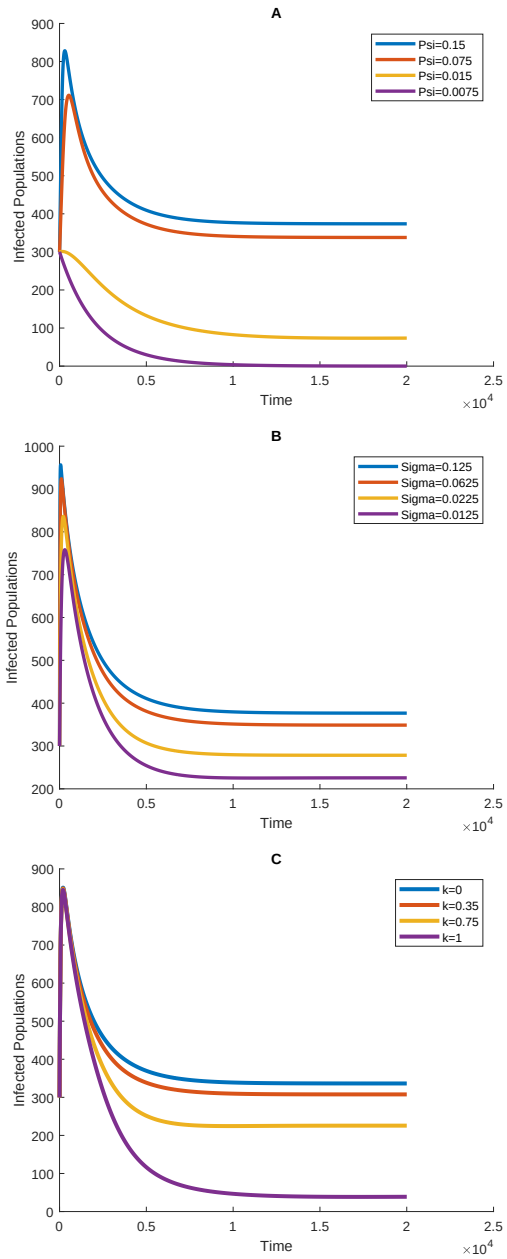

Figure 1: A: The impact of the contact rate between susceptible and infected individuals, B: The impact of the contact rate between susceptible individuals and bacteria, C: The influence of compliance with sanitation by infected individuals.

6 Conclusion

We can demonstrate that there are several factors WHICH affect the spread of each different disease and these factors differ from one disease to another and differ from one person to another. For further study, we can find a solution for other mathematical models studying the effect of bacterial concentration in surrounding environments and its concentration in infected persons and how this factor can affect the severity of disease.

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