Abstract
The most prevalent health issue is salmonella infection. Every year, between 200 million and 1.3 billion people are infected with Salmonella. Salmonella spreads by direct animal contact, food and drink, and very infrequently, person-to-person contact via the fecal-oral route. It is estimated that 94% of salmonellosis transmission occurs through food. Salmonella infection is, in fact, a bacterial infection. It is crucial to utilize mathematical modeling to describe how biological and biomedical systems behave dynamically. The SIR model of epidemics is the most often used mathematical model. Describing the patterns of Salmonella infectious disease in animal populations and the human population, we have formulated a SIRS epidemic model in our work. In significant part, the model was developed as a collection of ODEs based on the traits of transmission of infection. The occurrence of infection-free and endemic stable states was initially noted, and stability of the critical points of the model was later proven (infection-free and endemic). Following that, the fundamental reproduction number was calculated using the method of a next-generation matrix. Finally, the numerical simulations of this SIRS model have been completed. The analytical finding is explained using numerical simulations.

Keywords: Epidemic model, salmonella bacteria, equilibria, stability, sensitivity, numerical simulations

Introduction
Salmonella infection is a serious zoonotic illness that spreads from humans to other animals. Most patients get fever, diarrhea, and stomach cramps 12–72 hours after contracting salmonella. Immediately following a 4-7 day sickness, the majority of patients recover without therapy. But occasionally, the patient's diarrhea is so bad that they need to admit them to the hospital. Salmonella may be found in the intestines of people and other animals, such as birds. Salmonella is generally transferred to humans through animal feces-contaminated foods. Contaminated goods normally have a natural appearance and fragrance. Animal-based foods, for example meat, milk, poultry, or eggs, are frequently contaminated, but any item, consisting of veggies, can be polluted (P. Bouvet, 2002). As a result, Salmonella is regarded as a major public health concern across the world. There is little question that the mathematical model of Salmonella infection aids in the knowledge of disease transmission in specific environments as well as the prediction of outbreak behavior. Again, mathematical explanation manages the identification of critical states and the suggestion of consulted steps for decision-
makers to control disease transmission. The goal of this research is to use a SIRS epidemic model to understand the behavior of Salmonella infections in herds of animals and human population. Developing mathematical models and the best control strategies for infectious illnesses has been the subject of extensive research. In Biswas (2014) (see also Biswas et al., 2014), the use of mathematical modeling and an optimum control approach as the main instrument was used to independently study and assess the treatment of the deadliest infectious illnesses. We refer readers to see (Biswas et al., 2014; biswas et al., 2022; biswas et al., 2019; Das et al., 2021; Hasan et al., 2019; Hefferman et al., 2005; Khatun et al., 2020) for more details about mathematical modeling.

In this paper, we formulated a fresh mathematical framework considering the transmission behavior of Salmonella bacterial infection from animal to human by animal feces-contaminated foods.

Model Formulation

Consider that feeding animals will transmit the Salmonella illness from animal herds to the human population. We divided the total animal population into two groups. $S_a$ denote the appraisal of susceptible animals, $I_a$ denote the appraisal of infected animals. So, in this case $N_a = S_a + I_a$. Also, we divided the total human population into three groups. $S_h$ indicate the evaluation of vulnerable people, $I_h$ indicate the evaluation of infected people, and $R_h$ the evaluation of rescued persons. So, in this case $N_h = S_h + I_h + R_h$. According to our consideration the model diagram is given below:

The epidemic model may then be developed using the system of nonlinear ordinary differential equations shown below, which takes into account the entire situation:

$$
\begin{align*}
\frac{dS_a}{dt} &= \mu_a N_a - \frac{\beta_a S_a I_a}{N_a} - \mu_S S_a \\
\frac{dI_a}{dt} &= \frac{\beta_a S_a I_a}{N_a} - m I_a - \mu I_a \\
\frac{dS_h}{dt} &= \mu_h N_h - \frac{\beta_h S_h I_h}{N_h} - \mu_S S_h + \rho R_h \\
\frac{dI_h}{dt} &= \frac{\beta_h S_h I_h}{N_h} - \mu I_h - \gamma I_h \\
\frac{dR_h}{dt} &= \gamma I_h - \mu R_h - \rho I_h
\end{align*}
$$

Figure 1. SIRS model diagram of human and animal population.

(1)
Where the initial conditions are given below:

\[ S_0(0) = S_{00} \geq 0, \quad I_0(0) = I_{00} \geq 0, \quad S_h(0) = S_{h0} \geq 0, \quad I_h(0) = I_{h0} \geq 0, \quad R_h(0) = R_{h0} \geq 0. \]

In Table 1, the descriptions of the parameters are displayed.

Table 1. Explanation of the parameters of the model (1)

| Parameters | Description |
|------------|-------------|
| \( \mu_a \) | Birth and mortality rate of animal population |
| \( \beta_a \) | Transmission rate from susceptible to infected animal |
| \( m \) | Mortality rate of animal for infection |
| \( \mu_h \) | Birth and mortality rate of human population |
| \( \beta_h \) | Transmission rate of human by eating infected animal |
| \( \rho_h \) | Immunity loses rate of human |
| \( \gamma_h \) | Recovery rate of human |

**Model Analysis**

We examine the boundedness of the model, locate distinct equilibria (endemic equilibrium and disease-free points), determine the fundamental reproductive ratio, and carry out stability analysis at critical points.

**Equilibrium points and basic reproductive ratio**

The critical points of the model (1) are obtained by equating

\[ \frac{dS}{dt} = \frac{dI}{dt} = \frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dR_h}{dt} = 0. \]

We obtained two critical points. One is Disease-free Equilibrium Point

\( E_0(S_{00}, I_{00}, S_{h0}, I_{h0}, R_{h0}) = (N_a, 0, N_h, 0, 0) \)

And the second one is endemic equilibrium point \( E^*(S^*_a, I^*_a, S^*_h, I^*_h, R^*_h) \)

Here,

\[
S^*_a = \frac{N_a m + N_a \mu_a}{\beta_a}, \quad I^*_a = -\frac{\mu_h (N_a m - N_a \beta_a + N_a \mu_a)}{\beta_a (m + \mu_a)},
\]

\[
S^*_h = \frac{N_h \beta_h m \mu_a^2 + N_h \beta_h \mu_a \mu_h^2 + N_h \beta_h \gamma_h m}{\mu_h \rho_h + N_h \beta_h \mu_h \mu_h \rho_h}, \quad I^*_h = \frac{\mu_h + \rho_h - N_h \beta_h \mu_h^2 - N_a}{N_h \beta_h \mu_h + N_a N_h \beta_h m \mu_a},
\]

\[
R^*_h = \frac{N_a N_h \beta_h \gamma_h \mu_h^2 - N_a N_h \beta_h \beta - \gamma_h \mu_a^2}{N_a \beta_h m \mu_a \mu_h - N_a \beta_h m \mu_a \rho_h + N_h \beta_h m \mu_h \rho_h}.
\]
We obtained the basic reproduction numbers for this model \( R_{10} = \frac{\beta_a}{m + \mu_a} \) and \( R_{20} = \frac{\beta_h}{\gamma_h + \mu_h} \) applying the method of next generation matrix. And hence \( R_{10} > R_{20} \). So, \( R_0 = \frac{\beta_a}{m + \mu_a} \).

**Stability Test at \( E_0 \)**

**Theorem 1:** When basic reproductive ratio is less than 1 \( (R_0 < 1) \) then the \( E_0 \) point of the model (1) is locally asymptotically stable, when basic reproductive ratio is greater than 1 \( (R_0 > 1) \) then the \( E_0 \) point is unstable.

**Proof:** At the point of equilibrium (\( E_0 \) equilibrium point), the Jacobian of model (1) is

\[
\begin{pmatrix}
-\mu_a & -\beta_a & 0 & 0 & 0 \\
0 & \beta_a - m - \mu_a & 0 & 0 & 0 \\
0 & -\beta_h & -\mu_h & 0 & \rho_h \\
0 & \beta_h & 0 & -\gamma_h - \mu_h & 0 \\
0 & 0 & 0 & \gamma_h & -\mu_h - \rho_h
\end{pmatrix}
\]

We obtained the Eigen values are

\[\lambda_1 = -\mu_a, \lambda_2 = -\mu_h, \lambda_3 = -\gamma_h - \mu_h, \lambda_4 = -\mu_a - \rho_h, \lambda_5 = \beta_a - m - \mu_a = \left( m + \mu_h \right) \left( \frac{\beta_a}{m + \mu_a} - 1 \right) \]

Here, every eigenvalue is negative except \( \lambda_5 \).

So, if \( R_0 < 1 \) consequently, the \( E_0 \) equilibrium point of the model (1) is locally asymptotically stable and when \( R_0 > 1 \) then this point is unstable.

Hence Theorem 1 is proved.

**Stability test at endemic equilibrium**

**Theorem 2:** If basic reproductive ratio is greater than 1 \( (R_0 > 1) \) then this point of the model (1) is locally asymptotically stable and if \( R_0 < 1 \) then it is unstable.

**Proof:** At the endemic equilibrium point, the Jacobian of model (1) is
The characteristic equation of this Jacobian is

\[-(\lambda + \mu_a)(\lambda + \mu_h)(\gamma_h + \lambda + \mu_h)(\lambda + \mu_a + \rho_h)(\lambda - \beta_a + m + \mu_a) = 0\]

according to Routh-Hurwitz criteria if \( R_0 > 1 \), the real component of at least one of the eigenvalues is positive. As a result, the \( E^* \) is asymptotically stable locally if \( R_0 > 1 \) and otherwise unstable. As a result, Theorem 2 is established.

**Numerical Analysis**

In order to validate our analytic results, we ran numerical simulations of system (1) using the MATLAB ODE45-solver here. The epidemic model (1) may be solved by taking into account the beginning values as

\[ S_a(0) = 94, \quad I_a(0) = 40, \quad S_h(0) = 87, \quad I_h(0) = 18, \quad R_0(0) = 19 \]

and Table 2 contains all of the parameter values utilized for the numerical simulations. We executed simulations for fixed final duration 50 weeks. Figures 2-7 represent the solution trajectory and variation of different compartments.

**Table 2. Explanation and values of the parameters of the system (1)**

| Parameters | Description                                                | Values     |
|------------|-------------------------------------------------------------|------------|
| \( \mu_a \) | Birth and mortality rate of animal population                | 0.03223 week\(^{-1} \) |
| \( \beta_a \) | Transmission rate from susceptible to infected animal       | 0.38 week\(^{-1} \) |
| \( m \)    | Mortality rate of animal for infection                       | 0.003 week\(^{-1} \) |
| \( \mu_h \) | Birth and mortality rate of human population                | 0.00005 week\(^{-1} \) |
| \( \beta_h \) | Transmission rate of human by eating infected animal        | 0.75 week\(^{-1} \) |
| \( \rho_h \) | Immunity loses rate of human                                | 0.57 week\(^{-1} \) |
| \( \gamma_h \) | Recovery rate of human                                      | 0.32 week\(^{-1} \) |
Parvin, T. et al. (2022). Mathematical analysis of transmission dynamics and control of salmonella bacterial infection. *Khulna University Studies, Special Issue (ICSTEM4IR):* 845-854.

Figure 2. Solution trajectory of model (1).

Figure 3. Numerical solution of model (1).
Figure 4. Variations in infected humans for various human transmission rates from consuming diseased animals.

Figure 5. Animals that are affected vary depending on the transfer rate from susceptible to infected animals.
Figure 6. Variation in the number of infected people given various estimates of the ratio of infected to susceptible animals.

Figure 7. Human recovery varies with various recovery rate values.

From Figures 4-7 gives idea about control policy to minimize the transmission dynamics of salmonella bacterial infection.
Figure 8. A represents basic reproduction number depends on $\beta_a$ and $\mu_a$, B represents contour plot.

Conclusions

Intestinal bacteria can cause salmonellosis, often known as salmonella infection. Salmonella bacteria live in both human and animal intestines and are expelled through feces. Humans are often infected through polluted food or drink. Salmonella infections can affect persons who show no signs of illness. Most persons get diarrhea, fever, and stomach pain between 8 to 72 hours after exposure. Without treatment, the majority of healthy people recover within several days to a week. To explore the transmission of salmonella infection, a 5 compartmental coinfection framework has been put out in this research. Prior to doing the stability analysis of equilibria, we must establish the fundamental reproduction number. We discover that the endemic equilibrium point is stable if $R_0 > 1$ and the disease-free equilibrium point is unstable if $R_0 > 1$.

Acknowledgment

The first and second authors are supported by the M.Sc. (NST) fellowship, Session: 2021-2022. The Ministry of Science and Technology, Government of the People’s Republic of Bangladesh provided it. We sincerely thank the NST scholarship for providing financial assistance.

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