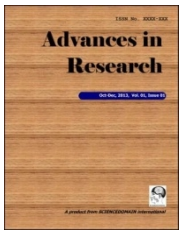

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Deterministic Mathematical Model for Dynamics of Water Borne Diseases

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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ABSTRACT

In this study a mathematical model is formulated using ordinary differential equations in order to understand the population dynamics of water borne diseases. The entire population is divided into five compartments depending upon their status. These compartments are – susceptible (S), exposed (E), symptomatically infected (I), recovered but carrying the infection asymptotically (R_C) and completely recovered (R). We have taken pathogen population into account as compartment (P). Here, two ways of getting infected are considered which are from person-to-person and from environment-to-person. i.e. a susceptible person can get infected either by coming directly in contact with the person having disease or by consuming contaminated food or water. A relation for the basic reproduction number is established. The analysis results show that the disease free equilibrium is locally asymptotically stable in $R_0<1$. Sensitivity analysis tells that the most important parameters are pathogen population and rate of transmission of disease from environment-to-person. Simulation is done using MATLAB. On the basis of sensitivity analysis and numerical simulation results we concluded that we need to cure an infected individual as soon as we identify the disease so that he would not contaminate environment for long.

Keywords: Water borne diseases; mathematical model; sensitivity analysis; simulation.

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1. INTRODUCTION

Water is very important for life. But if it is contaminated, it can be life-threatening as it is capable of carrying infection to a large population in a very small duration.

Water borne diseases are a big health issue in most parts of the world. In developed countries, these diseases are under control and are on the verge of elimination. But in developing countries, the diseases like typhoid, cholera, diarrhoea are a big challenge for the health departments [7-8]. Typhoid fever is caused by bacteria called *Salmonella typhi* (*S. typhi*) that infects only human beings. The symptoms of typhoid are high fever, headache, cough, rash, weakness, reduced White Blood Cell (WBC) counts and splenomegaly. The ultimate source of infection with these bacteria is a person having typhoid fever or carrier of *S. typhi*. Patients with disease excrete a large number of typhoid bacilli in faeces or urine, and enough bacteria may be present in their mucus, pus and in the vomit. A person having typhoid fever, excrete 10,00,000 *S. typhi* per gram of faeces and a large number of *bacilli* in urine [6]. These bacteria contaminate the environment and can survive for weeks in water, ice, dust and dried sewage. Diarrhoea is another water borne disease caused by *Escherichia coli*. In adults, this disease is mild and limited but fatal in small children. Main symptoms are watery loose motions accompanied by vomiting sometimes, that cause severe dehydration. Cholera is an acute diarrheal disease caused by bacteria known as *vibrio cholera*. It is a disease of small intestine. In this disease there is a rapid loss of body water and electrolytes from small intestine resulting in hypovolemic shock, hyper acidity and death (if not treated instantly). Humans are the only victims of these *bacilli* [6]. For more than one century, cholera is endemic in the delta of Ganges with annual epidemics.

There are various routes through which these pathogens enter a human body. They might be transmitted directly by consumption of contaminated water or indirectly by eating food that has come in contact with contaminated water, or by using a device (toothbrush etc.) that has contaminated water. They may also enter while taking a bath in contaminated water by penetrating skin, or through cut or wound on body [9].

Although a lot of measures are introduced by government to improve sanitation, new challenges have emerged. Many bacteria have developed resistance to water treatment and survive with chlorine also.

Epidemiological modelling helps us to understand the dynamics of these diseases so that we can trace the most important parameter(s) responsible for the disease spread [1]. Cvjetanovic et al. formulated a very detailed model for typhoid fever taking each minor stage into consideration. They analysed it for the effects of vaccination and sanitation and concluded that sanitation is more effective than vaccination for the control of disease spread [2]. Tien and Earn formulated two models – one SIR and the other with water compartment included into to the first and compared the analysis of two [3]. Robertson *et al.* extended the work of Tien and Earn and formulated a model for two separate groups of population and compared the disease dynamics in two groups [4]. Boots and Sasaki used a lattice model for modelling the disease dynamics on the basis of population density [5]. Codeco et al. formulated a stochastic model in order to see the impact of season on the waterborne diseases [6]. Hoeprich edited a book that is like an encyclopaedia of all infectious diseases which provide detailed biological information about each disease [7]. References [8-10] give information on the present scenario of these diseases as well as about water borne diseases. World Health Organisation website [11] and Centre for Disease Control website [12] are used to find out present epidemiological scenario and data for analysis.

2. MATHEMATICAL MODEL FOR WATER BORNE DISEASES

We have divided the entire population into five compartments. These compartments are namely: Susceptible (*S*), Exposed (*E*), Symptomatically infected (*I*), recovered but still carrying infection asymptotically (R_C) and completely Recovered (*R*). The sixth compartment is taken for the population of pathogen as (*P*). The disease may be transmitted either from person-to-person or from environment-to-person. The flow of population from one compartment to other is shown in Fig. 1 given below:

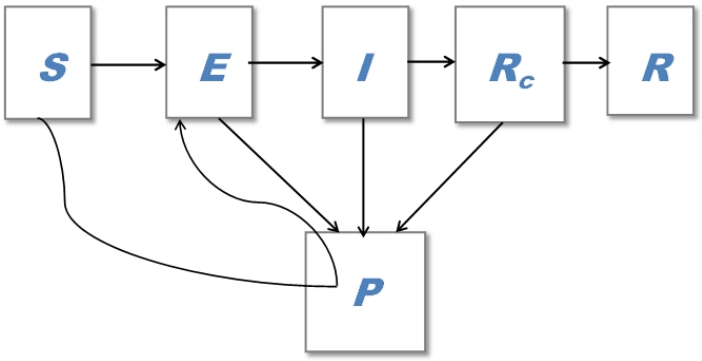

Fig. 1. Flow of population in various compartments

The parameters and state variables required for model formulation are given below:

State Variables

- *S*: Number of susceptible humans
- *E*: Number of humans having infection but no symptoms
- *I*: Symptomatically infected humans
- R_C: Recovered but still having infection without showing symptoms
- *R*: Completely recovered humans
- *P*: Pathogen population in water

Parameters

- *B*: Recruitment rate
- μ: Natural death rate
- δ: Disease induced death rate
- φ: Pathogen death rate (in food/water)
- β_1: Rate of transmission of disease from person (E / I / R_C) to person
- β_2: Rate of transmission of disease from water to person
- α_1: Rate of progression from exposed (E) class to infected (I) class
- α_2: Rate of progression from infected (I) class to recovered but carrier (R_C) class
- γ: Rate of progression from recovered but carrier (R_C) class to completely recovered (*R*) class
- *p*: Rate of shedding pathogen by an infected (E / I / R_C) individual into water

Using above variables and parameters, we formulate following set of ordinary differential equations:

$$\frac{dS}{dt} = B - \beta_1 \left(E + I + R_C \right) S - \beta_2 PS - \mu S \tag{1}$$

$$\frac{dE}{dt} = \beta_1 \left(E + I + R_C \right) S + \beta_2 PS - \left(\alpha_1 + \mu \right) E \tag{1}$$

$$\frac{dI}{dt} = \alpha_1 E - \left(\alpha_2 + \mu + \delta \right) I \tag{2}$$

$$\frac{dR_C}{dt} = \alpha_2 I - \left(\mu + \gamma \right) R_C \tag{3}$$

$$\frac{dR}{dt} = \gamma R_C - \mu R \tag{4}$$

$$\frac{dP}{dt} = p\left(E + I + R_C \right) - \phi P \tag{5}$$

with $N = S + E + I + R_C + R$ and $S > 0, E \geq 0, I > 0, R_C \geq 0, R \geq 0.$

Since, epidemic occurs for a short duration, therefore *R* does not appear in first four equations.

Adding equations from (1) to (4), we get

$$\frac{d}{dt}\left(S + E + I + R_C \right) = B - \mu\left(S + E + I + R_C \right) - \delta I - \gamma R_C$$

$$\Rightarrow \frac{d}{dt}\left(S + E + I + R_C \right) \leq B - \mu\left(S + E + I + R_C \right)$$

$$\Rightarrow \limsup_{t \to \infty} \left(S + E + I + R_C \right) \leq \frac{B}{\mu}$$

So, the feasible region for the system is

$$\Lambda = \left\{ \left(S, E, I, P, R_C \right) : S + E + I + R_C \leq \frac{B}{\mu}, S > 0, E \geq 0, I > 0, R_C \geq 0, P \geq 0 \right\}$$

Let there be an equilibrium point namely $X\left(\overline{S}, \overline{E}, \overline{I}, \overline{P}, \overline{R}_C \right)$ of the above system of equations. From the reduced system of equations, it is obvious that the system will have a disease free equilibrium at $X_0 = \left(\frac{B}{\mu}, 0, 0, 0, 0 \right)$.

Now, we establish a relation for basic reproduction number R_0.

R_0 = Number of secondary infections caused by an infected individual during its infectious period in a completely susceptible population.

= probability that an individual will survive until infectious *X* effective transmission rate *X* average duration of the infectious period.

$$\Rightarrow R_0 = \frac{B\left(\beta_2 p + \beta_1 \phi\right)\left(\alpha_1 + \alpha_2 + \mu + \delta\right)}{\phi\mu\left(\alpha_1 + \mu\right)\left(\alpha_2 + \mu + \delta\right)} \tag{6}$$

3. STABILITY OF DISEASE FREE EQUILIBRIUM

The Jacobian of the equations (1-4) and (6) can be written as

$$J(X_0) = \begin{bmatrix} -\mu & -\frac{\beta_1 B}{\mu} & -\frac{\beta_1 B}{\mu} & -\frac{\beta_2 B}{\mu} & -\frac{\beta_1 B}{\mu} \\ 0 & \frac{\beta_1 B}{\mu} - \left(\alpha_1 + \mu\right) & \frac{\beta_1 B}{\mu} & \frac{\beta_2 B}{\mu} & \frac{\beta_1 B}{\mu} \\ 0 & \alpha_1 & -\left(\alpha_2 + \mu + \delta\right) & 0 & 0 \\ 0 & p & p & -\phi & p \\ 0 & 0 & \alpha_2 & 0 & -\left(\mu + \gamma\right) \end{bmatrix} \tag{7}$$

The disease free equilibrium will be locally asymptotically stable if all the roots of (8) have negative real parts.

For this, it is sufficient to show that trace *(J)* < *0* and det*(J)* > *0* Clearly, by (8), trace *(J)* < *0*.

Now, for det (*J*) to be positive, on solving (8), we get

$$\frac{B\left(\beta_2 p + \beta_1 \phi\right)\left(\alpha_1 + \alpha_2 + \mu + \delta\right)}{\phi\mu\left(\alpha_1 + \mu\right)\left(\alpha_2 + \mu + \delta\right)} + \frac{\alpha_1 \alpha_2 B\left(\beta_2 p + \beta_1 \phi\right)}{\phi\mu\left(\alpha_1 + \mu\right)\left(\alpha_2 + \mu + \delta\right)\left(\mu + \gamma\right)} > 1$$

Using (7), we get

$$R_0 > \frac{1}{\left(1 + \frac{\alpha_1 \alpha_2}{\left(\alpha_1 + \alpha_2 + \mu + \delta\right)\left(\mu + \gamma\right)}\right)}$$

Here, right hand side of the above relation is always going to be less than or at most equal to 1. This implies that $R_0 < 1$.

Thus we conclude that the disease free equilibrium is locally asymptotically stable if $R_0 < 1$ otherwise unstable.

4. SENSITIVITY ANALYSIS

By this technique we check how much sensitive R_0 is to other parameters. This helps us find the right parameter(s) that need attention. For sensitivity analysis, we take the parameter values for typhoid fever. We used [1,6,7] to derive these values. They are given in Table 1.

Table 1. Values of different parameters for typhoid fever

Parameter	Value
B	2000
μ	0.0080
δ	0.0060
Φ	0.3333
β_1	0.1000
β_2	0.1000
α_1	1.0000
α_2	0.0476
γ	0.0111
p	100

Sensitivity indices are calculated for all those parameters which affect R_0. These indices are given in Table 2.

Table 2. Sensitivity Indices for R_0

Parameter	Sign	Index
B	+	1
μ	-	1.1303
δ	-	0.0918
Φ	-	0.9967
β_1	+	0.0033
β_2	+	0.9967
α_1	-	0.0501
α_2	-	0.7279
p	+	0.9967

These sensitivity indices show that the parameters which are most responsible for the disease spread are pathogen population and environment –to-person transmission rate. The parameters which have negative effect on the disease spread are pathogen death rate and rate of progression from I class to R_C class.

5. NUMERICAL SIMULATION

In order to understand the trends of population dynamics, we take a sample population and values of the parameters and present them graphically. This way, it helps us to visualize the true scenario and guide us which segment of population needs the greatest attention. Here, we take a sample population of 25,000 and plot them using MATLAB. The results are shown in Fig. 2.

These results show that symptomatically infected population shoots up very fast and increases pathogen concentration faster. This tells us that we need to work intensely with such individuals the moment we identify the disease in terms of quick treatment, extra careful sanitation and hygiene so that they will not contaminate much water or food.

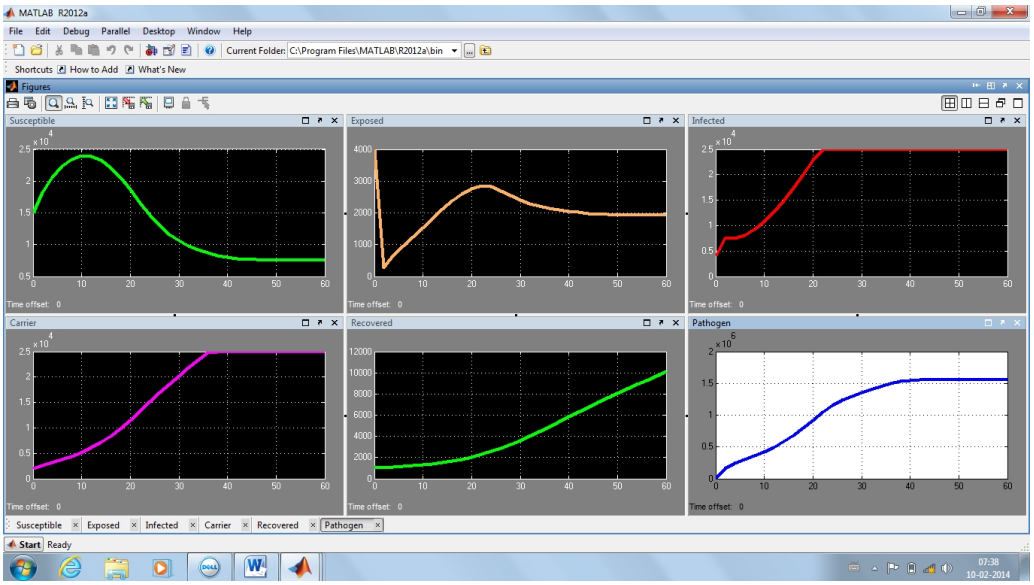

Fig. 2. Simulated population dynamics in various compartments

6. DISCUSSION

In this study, we formulated a model for water borne diseases using system of differential equations. We divided the human population into five compartments and took a sixth compartment for pathogen population. Relation for basic reproduction number, R_0, is established and disease free equilibrium is analysed. Analysis result shows that the disease free equilibrium is locally asymptotically stable if $R_0<1$. Sensitivity analysis is done using parametric values for typhoid. This tells us that the most crucial parameters which escalate disease spread are environment-to-person transmission rate and pathogen population. The parameters that have negative impact on the disease spread are pathogen death rate and the rate moving out from symptomatically infected class. This is also presented and supported by simulation results.

7. CONCLUSION

In this research work, we analysed the model analytically as well as numerically. Sensitivity analysis results indicate that contaminated environment is more responsible for the spread of these diseases. Simulation results (Fig. 2) show that population in infected (I) class increases very fast and parallel to it, pathogen population also shoots up. So, it concludes that we need to take special care of the individuals in this class by means of starting treatment as soon as we identify them, proper and extra careful sanitation and disinfection of patient's belongings, room and toilet etc. We also need to treat the water and drainage of the area where patient reside so that the disease bacteria will not contaminate the environment of that area.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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