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***Research paper***

# Mathematical Modeling of Epidemic Spread: Predicting and Controlling Infectious Diseases

**V. Pushparajesh [1] *, Jagtej Singh [2], Dr. Naresh Kaushik [3], Dr. Manoranjan Parhi [4], Harshit Singh [5], Dr. S. Usha Nandhini [6], Arpit Arora [7]**

*[1] Professor, Department of Electrical and Electronics Engineering, Faculty of Engineering and Technology, JAIN (Deemed-to-be University), Ramnagar District, Karnataka, India*
*[2] Chitkara Center for Research and Development, Chitkara University, Himachal Pradesh, India*
*[3] Assistant Professor, uGDX, ATLAS SkillTech University, Mumbai, India*
*[4] Professor, Center for Data Science, Siksha 'O' Anusandhan (Deemed to be University), Bhubaneswar, Odisha, India*
*[5] Assistant Professor, Maharishi School of Engineering & Technology, Maharishi University of Information Technology, Lucknow, India*
*[6] Assistant Professor, Department of Biotechnology, Sathyabama Institute of Science and Technology, Chennai, Tamil Nadu, India*
*[7] Center of Research Impact and Outcome, Chitkara University, Rajpura- 140417, Punjab, India*
**Corresponding author E-mail: v.pushparajesh@jainuniversity.ac.in*

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**Abstract**

Infectious diseases spread between individuals through direct or indirect contact. Microparasites, such as viruses and bacteria, and macroparasites, including flukes and helminths, are primarily responsible for causing a wide range of infectious diseases. Ecological and climatic changes increase the risk of pathogen emergence. Moreover, the evolution of pathogens makes it difficult to predict the spatio-temporal invasion of the diseases. HIV, smallpox, rabies, measles, dengue, and cholera are some of the diseases. For decades, these diseases have emerged: Zika, Ebola, Chikungunya, and others. Recently, these invaders have spread widely across the globe in just a few months. Infectious diseases can be a major economic problem for poor nations. Neglected tropical diseases lead to physical impairments and early deaths that hinder a nation's economic growth. Human-caused diseases harm both humans and animals, affecting economies and agriculture. Avian flu, foot-and-mouth disease, and viruses causing hemorrhage in fish all fall under this category of diseases. Because of significant financial damages in food production, farming industries, and the fisheries sectors annually. Furthermore, pathogens spread between species such as animals, birds, etc. Create the chance for humans to get sick.

***Keywords***: *Epidemic; Infection; Disease; Mathematical Model.*

## 1. Introduction

The term "communicable diseases" refers to illnesses that are brought on by infectious agents that bacteria or other microbes either directly or indirectly release into the environment. Multicellular parasites, viruses, bacteria, fungi, and protozoa are examples of infectious agents. In addition to direct contact, contaminated food, bodily fluids, objects, and airborne inhalation, infectious agents can also spread by vectors, as in the case of malaria. When infectious diseases, like the flu, may be easily spread by coming into contact with sick people or their secretions, they are deemed contagious.[1] [3]. Some infectious disease types with more specialized modes of infection, such as sexual or vector transmission, do not require Quarantine, or victim isolation, is frequently not necessary for certain infectious disease types that have more specialized mechanisms of infection, such as sexual or vector transmission, and are generally not contagious [2] [5]. The leading cause of death for people worldwide is infectious diseases [7]. They are accountable for over 13 million fatalities annually. In the next hour alone, a deadly disease will kill 1500 people. Most deaths from infectious diseases occur in developing countries, which have the least amount of funding for healthcare [11]. Approximately one-third of people in underdeveloped nations make less than $1 per day [15]. Understandably, fatal infectious diseases have been allowed to spread in the face of poverty and neglect [14]. Some of the world's poorest nations are currently suffering greatly due to global indifference and apathy. The developed nations are also at risk from the crisis if it is not addressed [4]. In Europe and other developed nations, old diseases like diphtheria and tuberculosis have rapidly spread. Because of climate change, infectious diseases continue to pose a threat to every nation. Therefore, adequate attention must be paid to the study and management of infectious diseases [10]. His model was predicated on the notion that density dependency affects measles incidence. It's interesting to note that in 1906, Hamer was the first to create differential equation models for malaria [12]. Among the many notable

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mathematicians who have influenced the theory of infectious disease modeling are Hudson, Martini, Lotka, Ross, who studied malaria in 1911, and others. These trailblazers created mathematical models that investigate the temporal spread of diseases using differential equations. [6].

### 1.1. Problem statement

This study is organized in such a way as to bring an issue to the following two main research questions:

- Based on epidemiological research on amoebiasis, is it possible to design a mathematical model, derived from the theory of differential equations, that can serve as a tool for both quantitative and qualitative studies of the dynamic transmission of amoebiasis through a population?
- How do these designed mathematical models fit the literature review on amoebiasis in terms of the description of the pattern of the dynamics of amoebiasis spread?

## 2. Materials and Methods

To combat pandemics brought on by both newly emerging and re-emerging infectious diseases, several international initiatives have been undertaken throughout the years to create an extensive surveillance network. Scientists and researchers from a wide range of fields are actively working to evaluate these potentially urgent circumstances as soon as possible. Mathematical modeling plays a significant role in this process and is essential for forecasting, assessing, and creating control plans for possible outbreaks [13]. Researchers have recently turned their attention to mathematical modeling of viral diseases to better understand their dynamics. We can precisely forecast the possible factors for an epidemic to spread by using mathematical models. In the past, models have mostly focused on factors that were directly linked to the disease, such as the infectious period, the mechanism of transmission, and exposure to the disease.

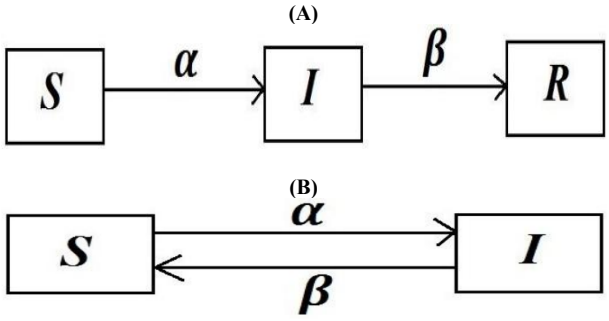



**Fig. 1:** Susceptible-Infected- Removed and Susceptible Model.

Zoonotic diseases are intriguing because they are caused by pathogens like bacteria, viruses, parasites, or fungi and have the amazing capacity to spread from animals to people and back again. Unfortunately, these diseases can lead to serious illness or even death in people, making them particularly concerning. Recently, we've seen a rise in zoonotic diseases, primarily due to the increasing interaction between humans and wildlife [8]. As farming expands and forests are cleared, humans and animals are increasingly sharing the same living spaces. Because of this overlap, people can contract diseases from diseased animals through bites, scratches, or even through their mucous, excrement, or saliva. Rabies, Dengue fever, Ebola, and West Nile are a few of the well-known zoonotic illnesses.

**Table 1:** Key Parameters for Amoebiasis Modeling

| Parameter | Value (Range) | Reference |
|---|---|---|
| Incubation Period | 1–4 weeks | [22] |
| Transmission Rate | 0.01–0.15 (variable by population) | [23] |
| Immune Decay Rate | 0.02–0.10 (based on immune response) | [24] |

Table 1 highlights the key parameters of mathematical modeling for amoebiasis, including the incubation period, transmission rate, and immune decay rate. These parameters are essential for studying the dynamics of infection and the transmission of Entamoeba histolytica in populations. Parameter values are based on the relevant literature on amoebiasis and its transmission.

The use of mathematical models to comprehend the transmission of infectious illnesses has been the subject of numerous studies. The primary pillars of epidemic modeling have been differential equation models, in which continuous functions describe the rates of entry and exit of susceptible, infected, and recovered populations. Computationally efficient and analytically solvable, these models are frequently based on the assumption of homogeneity within populations, which may not necessarily be an entirely accurate reflection of the complexity of the disease spread situation in the real world. This is especially true of diseases such as amoebiasis, in which transmission is influenced by environmental exposure (e.g., contaminated water) and human behavior (e.g., hygiene practices).

Alternatively, agent-based models (ABMs) have become a more versatile alternative, replacing traditional epidemic models. ABMs model the behavior of individual agents in a population, each with distinct behavior and personality. These models can incorporate heterogeneous interactions, including varying susceptibility and contact rates, and can model the impact of individual behavior (e.g., hand-washing, seeking medical treatment) on the dynamics of disease transmission. ABMs have found application especially in the study of such diseases as amoebiasis, where the environmental factors and individual behavior contribute much to the spread of the disease. As an example, it is easier to model the effects of public health interventions, such as water and sanitation, or behavior changes, with ABMs than it would be with traditional differential equation models that can ignore such granular dynamics.

Considering the compound nature of amoebiasis transmission, which exerts both direct and indirect influences, agent-based modeling is a more nuanced methodology, as it considers the impact of individual-level behavior and local environmental factors on disease dissemination. With these factors included, it is possible to simulate community-level using the ABMs and gain important insights into disease control that would otherwise not be possible when simulating using differential equations.

Although the overall epidemic modeling strategies outlined above can be a useful tool in understanding the dynamics of the disease, the study of amoebiasis needs a more specific implementation of these strategies because of the specific transmission dynamics. One of the main health issues, particularly in the developing world where sanitation and access to clean water are minimal, is amoebiasis, an infection caused by the protozoan Entamoeba histolytica. In contrast to most infectious diseases, the transmission of amoebiasis depends considerably on both environmental factors (e.g., water contamination) and individual factors (e.g., personal hygiene and sanitation habits). It is in this complexity that amoebiasis presents a perfect case study, as the use of traditional and advanced epidemic modeling methods applies. The interaction of individual behavior, exposure to the environment, and the interventions aimed at the health of the population, including water sanitation, makes amoebiasis modeling of interest and difficulty. The ensuing sections will apply the epidemic modeling techniques mentioned above to the dynamics of amoebiasis, to understand their role in the spread of the condition and how specific interventions can help decrease transmission in infected groups.

## 3. Results and Discussion

In late 2002 and early 2003, severe acute respiratory syndrome, or SARS, was a major worldwide issue. Due in major part to worldwide travel, this extremely contagious disease spread swiftly, reaching over 28 countries. The World Health Organization (WHO) coordinated its response to the outbreak as the number of patients rose. During this time, researchers worked long hours to gather comprehensive data and conduct several studies. They isolated a new coronavirus from SARS patients and were able to identify the entire genomic sequence of SARS-CoV.

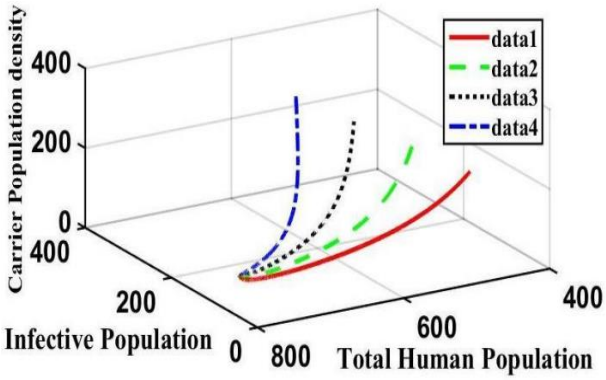



**Fig. 2:** Existence of Endemic Equilibrium Point.

The flowchart that goes along with the model is made up of rectangles, solid black arrows, dotted black arrows, plain shadowed circles, and one downward-pointing dotted arrow. The rectangles show different sections of the population, illustrating the various status levels of the disease, while the arrows indicate how people move from one compartment to another [9-10].

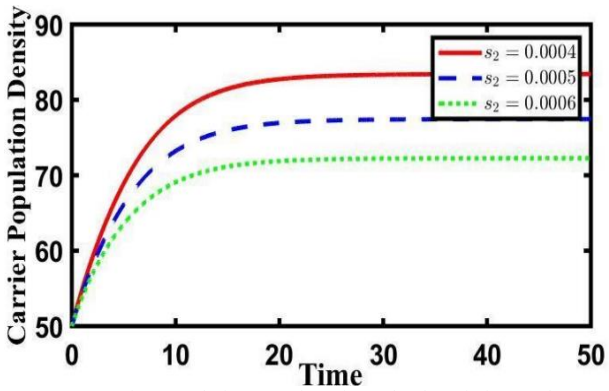



**Fig. 3:** Carrier Population Density-Time Plot for Distinct Values.

Figure 3: Provider Population Density-Time Plot, fantastic. This graphic illustrates how, given unique initial conditions, the provider population density changes over time. The exceptional values indicate the distinctive service densities of various intervals under exceptional conditions (e.g., specific transmission quotes or interventions of the public fitness). The plot assists in visualizing the results of modifications within the authentic population of companies on the long-term dynamics of the spread of the ailment.

You can find more details on the next page, where we've included the flow diagram or transfer diagram. The dead proportion of the population is represented symbolically by simple circles labeled D.

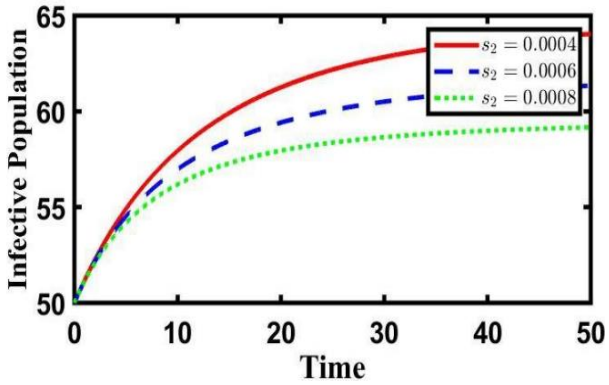



**Fig. 4:** Infective Population-Time Plot for Distinct Values.

To pinpoint the direct interactions between those who are susceptible and those who are infected, the dotted arrows illustrate how these infectious groups can contribute to the spread of amoebiasis.

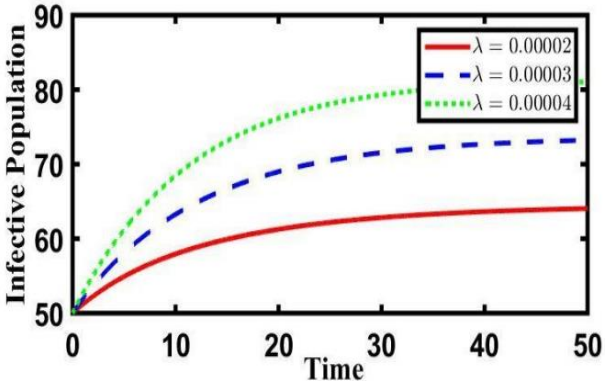



**Fig. 5:** Infective Population-Time Plot for Distinct Values of λ.

To accurately reflect the reality of contamination, it's important to consider that a susceptible person is more at risk when they meet infectious environments, like drinking water or eating contaminated food. From a modeler's perspective, the model should include a new state variable that indicates the presence of infection in these environments. Epidemic models for cholera serve as a great reference here, as both amoebiasis and cholera tend to spread from similar sources and follow comparable patterns of transmission.

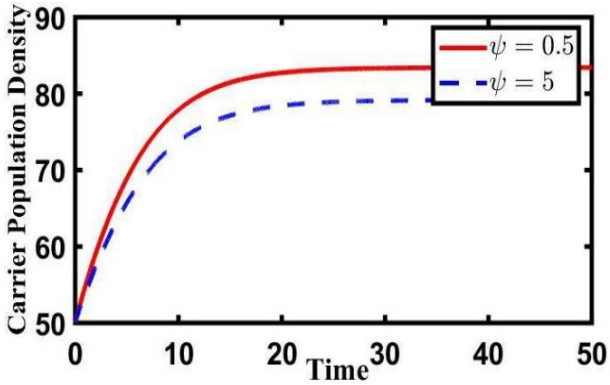



**Fig. 6:** Carrier Population Density-time Plot for Distinct Values of ψ.

Figure 6 shows the carrier population density plotted against time for each environment factor value. The image illustrates how variations in environmental factors, such as water contamination or hygienic conditions, affect the carrier population's density over time. The values are distinct, which means the carrier population reacts to various degrees of environmental exposure, giving information on the dissemination of the disease under different circumstances of public health.

In symptomatic amebiasis, the trophozoites make their way into the epithelial tissues, wreaking havoc on the protective mucus layer of the intestinal wall. This invasion leads to acute inflammation, which attracts neutrophils, plasma cells, macrophages, and lymphocytes, all of which contribute to tissue damage. Once the mucosa is compromised, it can result in tissue destruction, forming ulcers that cause diarrhea mixed with blood and a significant amount of mucus (Carrero et al. 2007).

It's important to note that amoebic liver abscess (ALA) tends to be more prevalent in older adults compared to younger individuals. The symptoms of ALA can vary, including abdominal pain, loss of appetite, and chest pain that radiates to the right upper quadrant and shoulder. In some cases, cough and jaundice may occur, lasting up to two weeks, particularly in severe instances of ALA.

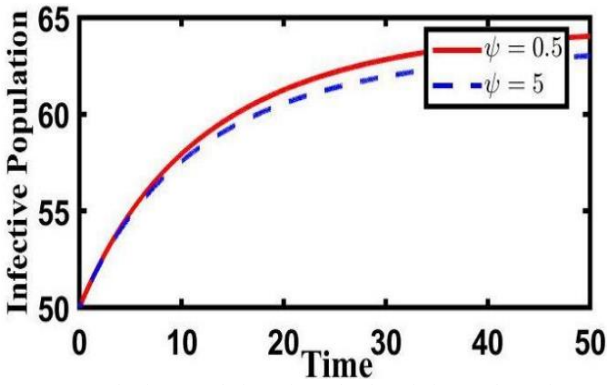



**Fig. 7:** Infective Population-Time Plot for Distinct Values of ψ.

Amoebiasis is particularly common in Sub-Saharan African countries. However, we don't have a clear picture of how widespread it is in this region, as there hasn't been much research done. That said, it's believed that the prevalence of amoebiasis is quite high in countries like Sudan, Côte d'Ivoire, Nigeria, Egypt, and South Africa.

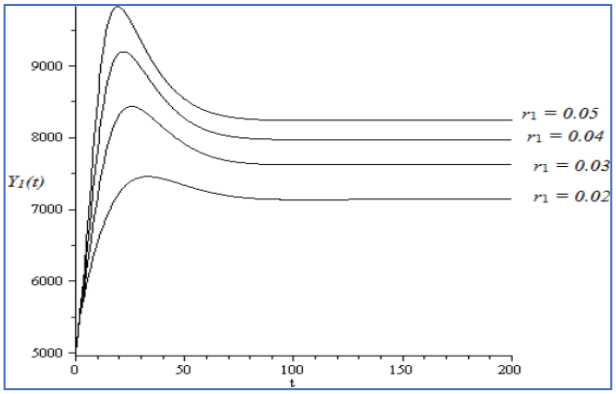



**Fig. 8:** Plot Between Different Values of Growth Rate Coefficient of Ecological Factors.

SARS was controlled in several ways, including the use of radio, television, newspapers, and posters to educate the public on ways to prevent the spread of the disease and the spraying of disinfectant in a variety of public areas, such as streets, shopping centers, airports, train and bus terminals, offices, classrooms, and even residential neighborhoods.

Whether at home, at a hospital, or on a school campus, everyone who had direct or indirect contact with probable SARS individuals was placed under quarantine. Both employees and students were subject to strict travel advisories. Every traveler had their body temperature taken, and nurses and physicians at the quarantine center examined those who failed the thermal imaging test. Many stock exchanges, cinemas, and internet cafes were temporarily closed as a precaution.

The existing model is useful but may be enhanced with delay different equations to help incorporate the incubation and immune decay characteristic of diseases such as amoebiasis, which are absent in the current model. This would enable a more realistic simulation of the temporal dynamics of the disease, particularly when delays in immune responses are considered and the time to the symptomatic period of the disease is taken into consideration. One possible use of this model might be in areas with inadequate healthcare facilities, like Sub-Saharan Africa, where early screening and treatment are important. As an example, a delay model may model the effect of delayed interventions, i.e., delay of water and sanitation or launch of targeted vaccination programs, on the distribution of amoebiasis with time. Additionally, partnerships with health agencies such as the World Health Organization (WHO) or the Centers for Disease Control and Prevention (CDC) can be utilized to test the model using real-life information on the endemic areas. The model could be refined by using real-time surveillance data, including reported cases of amoebiasis, data on water quality, and hygiene practices, to make the model more predictive.

## 4. Conclusion

It is best to think of the mathematical model developed to investigate the dynamics of amoebiasis transmission as a theoretical investigation. We now lack trustworthy health data, which limits its practical application. Currently, the data we do have globally comes from microscopy diagnosis, which only has a sensitivity rate of about 60%. This is largely due to the difficulty in distinguishing Entamoeba histolytica from other similar, non-pathogenic genera. Because of this, we can't really trust the results of fitting the model with such skewed data. The sensitivity index for the rate at which people are exposed to infectious stages seems quite low, suggesting that we should remove the exposed individuals from the other components of the epidemic model. Since the incubation duration is between one and four weeks, we suggest changing the model to incorporate delay differential equations that take into consideration the immunological decay period (ω) and the incubation period (σ) as delayed factors. We'll also combine the last two models from chapters five and six into a single epidemic model that integrates both direct and indirect transmission of the infection within the population.

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