

Modeling the Role of Environment and Asymptomatic Carriers in Infectious Disease Transmission

Olumuyiwa James Peter^{1,21} and Mayowa M. Ojo³, Ghaniyyat Bolanle Balogun⁴, Gbolahan Bolarin⁵, and Zainab O. Dere⁶

¹ Department of Mathematics, Saveetha School of Engineering, SIMATS, Saveetha University, Chennai, Tamil Nadu, 602105, India

² Department of Mathematical and Computer Sciences, University of Medical Sciences, Ondo City, Ondo State, Nigeria

³ Department of Mathematical Sciences, University of South Africa, Florida, South Africa

⁴ Department of Computer Science, University of Ilorin, Ilorin, Nigeria

⁵ Department of Mathematics, Federal University of Technology Minna, Niger State, Nigeria

⁶ Department of Mathematics, Florida State University, United States

ABSTRACT. Silent transmission by asymptomatic carriers and the persistence of pathogens in environmental reservoirs pose major challenges for controlling infectious diseases. These hidden transmission routes can sustain outbreaks even when symptomatic cases are effectively detected and managed. Motivated by these challenges, this study develops a deterministic compartmental model

¹Corresponding author: peterjames4real@gmail.com

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that incorporates asymptomatic carriers and environmental contamination into an extended SEIR-type framework. The model includes compartments for susceptible, exposed, asymptomatic infectious, symptomatic infectious, recovered individuals, and environmental pathogen concentration. Using qualitative analysis, we determine the disease-free and endemic equilibria and derive the basic reproduction number R_0 using the next-generation matrix method. Importantly, the reproduction number is decomposed into three epidemiologically meaningful components: R_C , R_I , and R_W , representing transmission driven by asymptomatic carriers, symptomatic individuals, and environmental reservoirs, respectively. This decomposition enables a clearer assessment of the relative contributions of distinct transmission pathways. Stability analysis shows that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$. Sensitivity analysis using Partial Rank Correlation Coefficients (PRCC) identifies key parameters influencing disease spread, highlighting the significant roles of asymptomatic transmission and environmental contamination. Numerical simulations further demonstrate how these hidden pathways can sustain outbreaks and increase disease burden. Overall, the results underscore the importance of integrated public health strategies that simultaneously address asymptomatic detection and environmental hygiene to effectively mitigate infectious disease transmission.

Keywords: Infectious disease, basic reproduction number, stability analysis, sensitivity analysis.

2000 Mathematics subject classification: 91A40, 34D23.

1. INTRODUCTION

The continued quest for knowledge regarding the determinants of infectious disease transmission is still a major focus of epidemiological studies. The traditional approaches to disease surveillance and control have mainly centered on detecting and containing infected individuals, as they are easily identified and are considered the primary cause of disease transmission during an epidemic [1]. However, recent findings have revealed that two less obvious routes of disease transmission are of primary importance in disease transmission and persistence. These routes are the asymptomatic disease transmission and disease transmission through the environment. The asymptomatic disease transmission occurs when people are infected with disease-causing organisms and still continue with their daily activities without displaying any signs of illness. This type of transmission is silent and often results in disease transmission within human populations without being detected. The disease transmission through the environment occurs through water, soil, air, and surfaces contaminated with disease-causing organisms. This type

of disease transmission is also silent and often acts as a reservoir for disease-causing organisms. The combination of these two routes of disease transmission results in a synergistic effect as human silent disease carriers are constantly replenishing disease transmission in the environment, and vice versa, even when human transmission is reduced or contained. These often-overlooked contributions provide significant problems for containment, especially in resource-limited or highly mobile populations [2, 3].

Outbreaks can be subtly caused by asymptomatic illnesses, in which people carry and spread diseases without showing any symptoms. These carriers might make up a sizable fraction of the infectious population for illnesses including hepatitis A, typhoid, and COVID-19 [2]. Studies using mathematical modeling have shown how asymptomatic people might not only prolong outbreaks but also compromise the efficacy of measures that only target symptomatic isolation and detection [3]. More inclusive modeling frameworks that accurately reflect their epidemiological impact are required because they are invisible to conventional public health systems.

In addition to silent carriers, environmental media such as water, soil, air, and fomites act as reservoirs and transmission vehicles for numerous diseases. Environmental transmission is especially important for enteric viruses, respiratory infections, and zoonotic diseases, as indirect exposure routes considerably contribute to disease burden [4, 5]. Persistent contamination in shared surroundings can prolong epidemics even after direct transmission pathways are closed.

The synergistic combination between asymptomatic carriers and environmental persistence can result in complicated and long-lasting transmission dynamics. For example, in zoonotic and livestock illnesses like foot-and-mouth disease virus (FMDV), models that incorporated carrier animals and nomadic herds indicated how such factors perpetuate endemicity despite management attempts [6], these findings emphasize the importance of including individual- and environmental-level factors into illness models.

Recent mathematical modeling studies show that outbreaks can persist in a quasi-endemic condition even with substantial symptom-based therapies [7, 8]. Pathogens in environmental substrates, such as soil, water, or fomites, can regenerate infection cycles without direct human contact [9, 10]. [7] found that asymptomatic infectious periods, which vary by pathogen, can account for up to 96% of transmission in some systems but are not well characterized in control efforts [7]. Harris et al. (2023) found that intermediate levels of asymptomatic transmission can increase epidemic deaths by sustaining long-duration outbreaks and

preventing quick exhaustion [8]. Global meta-analyses of SARS-CoV-2 suggest that household settings are hotspots for silent transmission, with asymptomatic rates lower than symptomatic but nonetheless significant in maintaining community spread [11, 12].

It is becoming more widely acknowledged that environmental reservoirs, in addition to human hosts, play a significant role in endemicity. Environmental contamination alone can sustain disease around persistence thresholds, according to a 2025 compartmental model for soil-transmitted helminths that included animal-human-environment interaction [9]. Similarly, sanitation that targeted environmental *V. cholerae* reservoirs significantly reduced outbreak length and overall R_0 , surpassing measures that only addressed symptomatic human cases, according to a recent cholera transmission analysis [10].

Inspired by the above findings, we propose a deterministic compartmental model that explicitly accounts for both asymptomatic carriers and environmental transmission. The model builds on traditional SIR-type frameworks by including compartments for asymptomatic patients and an environmental reservoir. We use qualitative analysis and numerical simulations to evaluate how these parameters affect outbreak magnitude, basic reproduction number, and intervention strategy success. Our goal is to provide more detailed insights into the control of illnesses with silent transmission and chronic environmental pollution, which is an important step toward more effective public health policy and preparedness.

2. MODEL FORMULATION

We present an extended compartmental mathematical model to describe an infectious disease's transmission patterns within a human population, taking into consideration both direct and environmental transmission routes. The human population is classified into six mutually exclusive compartments: susceptible individuals $S(t)$, exposed individuals $E(t)$ who are infected but not yet infectious, asymptomatic infectious carriers $C(t)$, symptomatic infectious individuals $I(t)$, recovered individuals $R(t)$ who have developed immunity, and $W(t)$, which represents the concentration of pathogens in the environment. The entire human population at time t is calculated as

$$N(t) = S(t) + E(t) + C(t) + I(t) + R(t). \quad (2.1)$$

Individuals join the population at a constant recruitment rate Λ , which can include births or immigration. All individuals have a natural mortality rate μ , but infected individuals may die owing to disease

at additional rates μ_C and μ_I for asymptomatic and symptomatic individuals, respectively. Infection spreads through direct contact with infected individuals as well as exposure to contaminated environments. The force of infection, represented by $\lambda(t)$, comprises contributions from asymptomatic carriers, symptomatic individuals, and the environmental reservoir, and is given by

$$\lambda(t) = \alpha_c C + \alpha_I I + \alpha_W W, \quad (2.2)$$

where α_c , α_I , and α_W are the respective transmission rates.

The model is governed by the following system of ordinary differential equations. The susceptible population $S(t)$ decreases due to infection and natural death, and increases through recruitment:

$$\frac{dS}{dt} = \Lambda - (\alpha_c C + \alpha_I I + \alpha_W W)S - \mu S. \quad (2.3)$$

The exposed class $E(t)$ increases by infection and decreases by progression to the infectious stage at rate ϕ and natural death:

$$\frac{dE}{dt} = (\alpha_c C + \alpha_I I + \alpha_W W)S - \phi E - \mu E. \quad (2.4)$$

A proportion p of exposed individuals progress to become asymptomatic carriers $C(t)$, while the remaining fraction $1 - p$ become symptomatic. Carriers progress to symptomatic infected at a rate γ_C or die due to disease at rate μ_C :

$$\frac{dC}{dt} = p\phi E - (\gamma_C + \mu_C)C. \quad (2.5)$$

The symptomatic infected individuals $I(t)$ increase by the remaining exposed individuals and by progression from the carrier class, and decrease due to recovery at rate γ_I or disease-induced death at rate μ_I :

$$\frac{dI}{dt} = (1 - p)\phi E + \gamma_C C - (\gamma_I + \mu_I)I. \quad (2.6)$$

Recovered individuals $R(t)$ gain immunity through recovery from the symptomatic class and leave the class via natural death:

$$\frac{dR}{dt} = \gamma_I I - \mu R. \quad (2.7)$$

Finally, the pathogen concentration in the environment $W(t)$ increases through shedding by carriers and symptomatic individuals at rates ε_c and ε_I , respectively, and decays at a rate δ :

$$\frac{dW}{dt} = \varepsilon_c C + \varepsilon_I I - \delta W. \quad (2.8)$$

The model is therefore represented by the following system:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\alpha_c C + \alpha_I I + \alpha_W W)S - \mu S, \\
\frac{dE}{dt} &= (\alpha_c C + \alpha_I I + \alpha_W W)S - \phi E - \mu E, \\
\frac{dC}{dt} &= p\phi E - (\gamma_C + \mu_C)C, \\
\frac{dI}{dt} &= (1-p)\phi E + \gamma_C C - (\gamma_I + \mu_I)I, \\
\frac{dR}{dt} &= \gamma_I I - \mu R, \\
\frac{dW}{dt} &= \varepsilon_c C + \varepsilon_I I - \delta W.
\end{aligned} \tag{2.9}$$

3. MODEL ANALYSIS

This section presents the analytical investigation of the proposed infectious disease model. We focus on determining the feasible region for the model variables, the disease-free equilibrium (DFE), and the endemic equilibrium (EE). These equilibria provide important insights into the long-term behavior of the disease dynamics and conditions for disease eradication or persistence.

3.1. Feasible Region and Positivity of Solutions. The model describes population compartments that represent biological quantities and therefore must remain non-negative for all time. We define the feasible region

$$\Omega = \left\{ (S, E, C, I, R, W) \in \mathbb{R}_+^6 : N = S + E + C + I + R \leq \frac{\Lambda}{\mu} \right\}, \tag{3.1}$$

where $\mathbb{R}_+^6 = \{x \in \mathbb{R}^6 : x_i \geq 0, \forall i\}$.

It can be shown that Ω is positively invariant and attracting under the model flow, ensuring that solutions with non-negative initial conditions remain biologically meaningful and bounded.

3.2. Disease-Free Equilibrium (DFE). The disease-free equilibrium corresponds to the state where no infection exists in the population, i.e.,

$$E^* = C^* = I^* = W^* = 0.$$

At DFE, the population is at steady state with

$$S^* = \frac{\Lambda}{\mu}, \quad R^* = 0.$$

This equilibrium represents the scenario of a completely susceptible population with no pathogen present.

3.3. Endemic Equilibrium (EE). The endemic equilibrium represents a steady state where the disease persists in the population with nonzero levels of exposed, infectious, and environmental pathogen compartments. At EE, all compartments satisfy

$$(S^*, E^*, C^*, I^*, R^*, W^*) \in \Omega,$$

with

$$E^* > 0, \quad C^* > 0, \quad I^* > 0, \quad W^* > 0.$$

The explicit expressions for the endemic equilibrium are given by

$$\begin{aligned} S^* &= \frac{\phi + \mu}{K}, \\ E^* &= \frac{\frac{\Lambda}{\mu} - S^*}{Q}, \\ C^* &= \frac{p\phi}{\gamma_C + \mu_C} E^*, \\ I^* &= \frac{\phi}{\gamma_I + \mu_I} \left(1 - p + \frac{\gamma_C p}{\gamma_C + \mu_C} \right) E^*, \\ R^* &= \frac{\gamma_I}{\mu} I^*, \\ W^* &= \frac{\varepsilon_c C^* + \varepsilon_I I^*}{\delta}, \end{aligned} \tag{3.2}$$

where

$$K = \alpha_c \frac{p\phi}{\gamma_C + \mu_C} + \alpha_I \frac{\phi}{\gamma_I + \mu_I} \left(1 - p + \frac{\gamma_C p}{\gamma_C + \mu_C} \right) + \alpha_W \frac{1}{\delta} \left(\varepsilon_c \frac{p\phi}{\gamma_C + \mu_C} + \varepsilon_I \frac{\phi}{\gamma_I + \mu_I} \left(1 - p + \frac{\gamma_C p}{\gamma_C + \mu_C} \right) \right),$$

and

$$Q = 1 + \frac{p\phi}{\gamma_C + \mu_C} + \frac{\phi}{\gamma_I + \mu_I} \left(1 - p + \frac{\gamma_C p}{\gamma_C + \mu_C} \right) + \frac{\gamma_I}{\mu} \frac{\phi}{\gamma_I + \mu_I} \left(1 - p + \frac{\gamma_C p}{\gamma_C + \mu_C} \right).$$

These expressions characterize the endemic state where infection and environmental contamination persist over time.

3.4. Basic Reproduction Number $\mathcal{R}_0$. The basic reproduction number, $\mathcal{R}_0$, is a key epidemiological metric representing the expected number of secondary human infections generated by a typical infected individual introduced into a completely susceptible population. It serves as a threshold parameter for assessing the potential invasion of infection: if $\mathcal{R}_0 < 1$, the disease-free equilibrium is locally asymptotically stable, whereas if $\mathcal{R}_0 > 1$, the disease-free equilibrium is unstable and infection can invade the population.

In this model, transmission occurs through three pathways: direct transmission from asymptomatic carriers, direct transmission from symptomatic infectious individuals, and environmental transmission. Accordingly, the basic reproduction number can be decomposed as

$$\mathcal{R}_0 = \mathcal{R}_C + \mathcal{R}_I + \mathcal{R}_W,$$

where

- $\mathcal{R}_C$ represents the contribution from direct transmission by asymptomatic carriers;
- $\mathcal{R}_I$ represents the contribution from direct transmission by symptomatic infectious individuals; and
- $\mathcal{R}_W$ represents the contribution from environmental transmission.

Using the next-generation matrix approach [13], we consider the infected subsystem (E, C, I, W) , where W denotes the environmental pathogen concentration. Define

$$a = \phi + \mu, \quad b = \gamma_C + \mu_C, \quad c = \gamma_I + \mu_I,$$

and let the disease-free equilibrium be

$$E_0 = (S_0, 0, 0, 0, 0, 0), \quad S_0 = \frac{\Lambda}{\mu}.$$

1. **Asymptomatic Carriers.** A fraction p of exposed individuals enters the asymptomatic carrier class C at rate $p\phi$. Individuals leave this class through progression to the symptomatic class at rate γ_C or disease-induced mortality at rate μ_C . The contribution of direct transmission from asymptomatic carriers is therefore

$$\mathcal{R}_C = \frac{S_0 \alpha_c p \phi}{(\phi + \mu)(\gamma_C + \mu_C)} = \frac{\alpha_c p \phi \Lambda}{(\phi + \mu)(\gamma_C + \mu_C)\mu}.$$

2. **Symptomatic Infectious Individuals.** Symptomatic infectious individuals arise both directly from the exposed class, with fraction $1 - p$, and through progression from the asymptomatic carrier class at rate γ_C . Their removal rate is $\gamma_I + \mu_I$. Thus, the contribution of direct transmission from symptomatic infectious individuals is

$$\mathcal{R}_I = \frac{S_0 \alpha_I \phi}{(\phi + \mu)(\gamma_I + \mu_I)} \left(1 - p + \frac{p \gamma_C}{\gamma_C + \mu_C} \right).$$

Equivalently, in terms of the recruitment and natural mortality rates,

$$\mathcal{R}_I = \frac{\alpha_I \phi \Lambda}{(\phi + \mu)(\gamma_I + \mu_I)\mu} \left(1 - p + \frac{p \gamma_C}{\gamma_C + \mu_C} \right).$$

3. **Environmental Reservoir.** The environmental pathogen concentration W increases through shedding by asymptomatic carriers and symptomatic infectious individuals at rates ε_c and ε_I , respectively, and decreases through environmental pathogen decay at rate δ . Environmental transmission occurs at rate α_W . Accounting for shedding from both infectious classes, including progression from C to I , gives

$$\mathcal{R}_W = \frac{S_0 \alpha_W \phi}{(\phi + \mu) \delta} \left[\frac{\varepsilon_c p}{\gamma_C + \mu_C} + \frac{\varepsilon_I}{\gamma_I + \mu_I} \left(1 - p + \frac{p \gamma_C}{\gamma_C + \mu_C} \right) \right].$$

Summary. The basic reproduction number is obtained by summing the contributions from the three transmission pathways:

$$\mathcal{R}_0 = \mathcal{R}_C + \mathcal{R}_I + \mathcal{R}_W.$$

The decomposition identifies the contributions of direct transmission from asymptomatic carriers, direct transmission from symptomatic infectious individuals, and transmission through the environmental reservoir.

Next-Generation Matrix Representation. The next-generation matrix method separates the production of new human infections from transfers between infected states and other losses. With the infected-state ordering (E, C, I, W) , the matrix F , representing the production of new human infections, and the matrix V , representing transfers between infected states and losses, are

$$F = \begin{pmatrix} 0 & \alpha_c S_0 & \alpha_I S_0 & \alpha_W S_0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \phi + \mu & 0 & 0 & 0 \\ -p\phi & \gamma_C + \mu_C & 0 & 0 \\ -(1-p)\phi & -\gamma_C & \gamma_I + \mu_I & 0 \\ 0 & -\varepsilon_c & -\varepsilon_I & \delta \end{pmatrix}.$$

Here, new human infections enter the exposed class E , and hence only the first row of F is nonzero. The environmental pathogen concentration W is included in the infected subsystem because it contributes to the force of infection. The shedding terms are incorporated into V as transfers of pathogens from the infectious human compartments to the environmental reservoir, rather than as new human infections.

The next-generation matrix is FV^{-1} , and the basic reproduction number is its spectral radius:

$$\mathcal{R}_0 = \rho(FV^{-1}),$$

where $\rho(\cdot)$ denotes the spectral radius. For the model considered here, this expression yields the sum of the three pathway-specific contributions derived above.

3.5. Stability Analysis of the Disease-Free Equilibrium. The local stability of the disease-free equilibrium is determined by evaluating the Jacobian matrix of the system at the DFE. Using the state ordering (S, E, C, I, R, W) , the Jacobian matrix at the disease-free equilibrium is

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\alpha_c S_0 & -\alpha_I S_0 & 0 & -\alpha_W S_0 \\ 0 & -(\phi + \mu) & \alpha_c S_0 & \alpha_I S_0 & 0 & \alpha_W S_0 \\ 0 & p\phi & -(\gamma_C + \mu_C) & 0 & 0 & 0 \\ 0 & (1-p)\phi & \gamma_C & -(\gamma_I + \mu_I) & 0 & 0 \\ 0 & 0 & 0 & \gamma_I & -\mu & 0 \\ 0 & 0 & \varepsilon_c & \varepsilon_I & 0 & -\delta \end{pmatrix}, \quad (3.3)$$

where

$$S_0 = \frac{\Lambda}{\mu}.$$

The eigenvalues associated with the susceptible and recovered directions are both $-\mu$. The remaining four eigenvalues are determined by the subsystem involving the exposed class E , the asymptomatic carrier class C , the symptomatic infectious class I , and the environmental pathogen concentration W .

Assume that the model parameters are non-negative, that

$$\mu > 0, \quad \delta > 0, \quad \phi + \mu > 0, \quad \gamma_C + \mu_C > 0, \quad \gamma_I + \mu_I > 0.$$

Under these assumptions, the transition matrix V is nonsingular, and the next-generation matrix criterion can be applied to the infected subsystem.

Consequently, the disease-free equilibrium is locally asymptotically stable if

$$\mathcal{R}_0 < 1. \quad (3.4)$$

Conversely, if $\mathcal{R}_0 > 1$, the disease-free equilibrium is unstable, indicating that infection can invade the susceptible population. The threshold case $\mathcal{R}_0 = 1$ requires separate analysis and is not classified by these strict inequalities alone. ““

4. SENSITIVITY ANALYSIS

Sensitivity analysis is an important tool in mathematical epidemiology to identify the most influential parameters affecting the basic reproduction number R_0 . In this study, we employ the Partial Rank Correlation Coefficient (PRCC) method because it is well suited for nonlinear epidemiological models and can account for interactions among multiple parameters simultaneously. Unlike simple sensitivity indices that evaluate parameters independently, PRCC allows for the assessment of the

monotonic relationship between each parameter and the model output while controlling for the influence of other parameters.

By determining which parameters have the greatest impact, effective control strategies can be prioritized and optimized R_0 [14, 15, 16, 17, 18]. By determining which parameters have the greatest impact, effective control strategies can be prioritized and optimized. To understand the relative influence of model parameters on the key epidemiological quantities, we performed a Partial Rank Correlation Coefficient (PRCC) analysis for the reproduction numbers associated with asymptomatic carriers (R_C), symptomatic infectious individuals (R_I), environmental transmission (R_W), and the overall basic reproduction number (R_0).

The PRCC method assesses the monotonic relationship between each input parameter and the output reproduction number while controlling for the effects of all other parameters. This approach is particularly suitable for complex nonlinear models where parameters may interact and influence the outputs jointly. The parameters included in the sensitivity analysis were the proportion of exposed individuals who become asymptomatic carriers (p), the transmission rates from asymptomatic carriers (α_C) and symptomatic individuals (α_I), the environmental transmission rate (α_W), the decay rate of pathogens in the environment (δ), the shedding rates of pathogens by asymptomatic and symptomatic individuals (ε_c and ε_I , respectively), as well as other natural and disease progression rates. The analysis revealed that R_C exhibits a strong positive correlation with the asymptomatic carrier transmission rate α_C and the fraction p of exposed individuals progressing to this class. The high sensitivity to p (the asymptomatic fraction) suggests that even a small increase in “hidden” cases significantly expands the asymptomatic transmission pathway. This highlights the critical role asymptomatic carriers play in sustaining transmission, suggesting that interventions targeting asymptomatic spread could significantly reduce disease propagation. For R_I , the symptomatic transmission rate α_I showed the highest positive influence, with a moderate positive correlation with the fraction of individuals progressing to the symptomatic infectious class ($1 - p$). This indicates that controlling transmission from symptomatic individuals, through isolation or treatment, remains a vital strategy. Regarding the environmental transmission component R_W , it was strongly sensitive to the pathogen shedding rates ε_c and ε_I , the environmental transmission rate α_W , and inversely related to the pathogen decay rate δ . This underscores the importance of environmental hygiene and disinfection in controlling disease spread. Finally, since R_0 is the sum of all three

components, it showed sensitivity to all the aforementioned parameters, emphasizing that effective disease control requires addressing multiple transmission pathways simultaneously. These findings provide a quantitative basis for prioritizing public health interventions. Reducing transmission from asymptomatic carriers (e.g., through widespread testing and mask use), isolating and treating symptomatic individuals, and enhancing environmental sanitation (e.g., surface disinfection, ventilation) are all critical measures to effectively reduce the basic reproduction number and curb the spread of the infectious disease.

TABLE 1. Description of model parameters

Parameter	Definition	Value	Source
Λ	Recruitment rate into the population	90,000	[20]
μ	Natural death rate	0.0125 (i.e., 1/80)	[20]
α_c	Transmission rate from asymptomatic carriers	1.5	[20]
α_I	Transmission rate from symptomatic individuals	1.0	[20]
α_W	Transmission rate from environmental reservoir	1×10^{-4}	Estimated
ϕ	Rate at which exposed individuals become infectious	0.071–0.2	Estimated
p	Proportion of exposed individuals who become asymptomatic	0.3	[20]
γ_e	Progression rate from asymptomatic class to symptomatic class	0.1–0.5	[20]
γ_I	Recovery rate of symptomatic individuals	0.75	[20]
μ_c	Disease-induced death rate for asymptomatic carriers	0.005	Estimated
μ_I	Disease-induced death rate for symptomatic individuals	0.05	Estimated
ε_c	Shedding rate of pathogen from asymptomatic carriers to environment	0.5	Estimated
ε_I	Shedding rate of pathogen from symptomatic individuals to environment	0.5	Estimated
δ	Decay rate of pathogen in environment	0.7	[20]

““

5. NUMERICAL SIMULATION AND DISCUSSION

Numerical simulations are conducted to illustrate the dynamics of the model and to validate the analytical results. The simulations focus on the effects of the key model parameters on disease transmission and the relative contributions of the asymptomatic, symptomatic, and environmental transmission pathways. The dynamics of a disease are dependent on various factors, including the biological characteristics of the pathogen, the infectiousness period, and the human immune response ability. Infection is one of the most vulnerable things that has continually posed harm to the human population. This harm is increased by the potential dynamics of the symptomatic and asymptomatic nature of disease. Some diseases are known to be asymptomatic in nature, with the possibility of infectiousness, meaning that an individual can be infected with a disease without showing any symptoms but is capable of transmitting the disease to susceptible individuals in the population [7]. In addition, for diseases that are symptomatic in nature, there is a possibility that an individual may exhibit mild symptoms associated with the disease due to their immunity and, as a result, neglect the potential impact of disease transmission in the community.

Since asymptomatic carriers of infection are capable of transmitting disease, this presents a threat for a potential increase in the spread of diseases. In other words, when people are not aware that they are carriers of a disease and are capable of transmitting the infection, they will interact with other humans in the population without any precautions of disease transmission. For example, as reported in [7, 22], in the early period of the COVID-19 outbreak, the health practitioners and policymakers focused on the symptomatic people to define cases and strategies. In addition, testing and treatment priorities were provided for symptomatic people, which led to a missed opportunity for an earlier, better understanding of the transmission of the pathogen. However, it was believed that over half of the infections were either asymptomatic or pre-symptomatic, and during this period, over half of the transmission occurred.

Based on the potential influence of asymptomatic carrier in the abundance of disease in a given population, we perform a numerical simulation in this section to examine the role of environmental factor and asymptomatic carriers in infectious disease transmission. The developed model (2.9) is used with the parameter values provided in Table 1 for the simulations, and the baseline value used in each simulation is as presented in Table 1. For readers clarity, the parameter values are as provided in the work of [20]. Also, the selected initial population used

for the simulation is based on a hypothetical population size. As a result, the purpose of the simulation performed in this work is to examine the dynamics of the disease under different level of the progression rate of asymptomatic individuals to symptomatic, the transmission rate of asymptomatic carriers, and the transmission rate from environmental reservoirs.

In Figure 1, we perform a simulation to examine the impact of different levels of the progression rate of asymptomatic individuals to symptomatic individuals on the infectious population. The progression from asymptomatic status to symptomatic status is attributable to the progression rate from asymptomatic to symptomatic infection is high due to the impact of diagnosis. Aggressive diagnostic screening, represented by a high $\gamma_c = 0.5$, effectively depletes the ‘silent’ infectious pool by transitioning individuals into the symptomatic class where they can be monitored (see Figure 1a). On the contrary, a reduction in the progression rate from asymptomatic to symptomatic infection is high (i.e. a low value of detection rate $\gamma_C=0.30$) will upsurge the population of asymptomatic infectious individuals. The impact of different levels of detection rate on the symptomatic infectious population is depicted in Figure 5. The result shows that when there is a lower progression rate from asymptomatic to symptomatic infection is high.” ($\gamma_C = 0.30$), the size of the symptomatic infectious population increases in the first 25th time interval, and then declines for the rest of the 25 time intervals. This dynamic can be explained by the fact that when there is an increase in the asymptomatic individuals due to low detection rate if infection, this will lead to an increase in the transmission rate of the disease, as the asymptomatic individuals would be unaware of their status and will not take any precautions about disease transmission. Furthermore, the later decline in the size of the symptomatic infectious population is attributed to the fact that efforts are placed on the treatment of symptomatic individuals to reduce disease burden in the population. This dynamic is also evident when there is a high progression rate from asymptomatic to symptomatic infection is high (i.e., $\gamma_c = 0.5$). The size of the symptomatic infectious population is reduced compared to when there is a low detection rate, because with a high detection rate, individuals are aware of their infection status and thus are more likely to take precautions. This awareness will reduce the transmission rate of the disease, thus resulting in a lower number of infected individuals in the populace.

In Figure 1, we perform a simulation to examine the impact of different levels of the progression rate of asymptomatic individuals to symptomatic individuals on the infectious population. The progression from

asymptomatic status to symptomatic status is attributable to the progression rate from asymptomatic to symptomatic infection is high due to the impact of diagnosis. A higher progression rate, represented by a high $\gamma_c = 0.50$, effectively depletes the ‘silent’ infectious pool by transitioning individuals into the symptomatic class where they can be monitored (see Figure 5). On the contrary, a reduction in the progression rate from asymptomatic to symptomatic infection is high (i.e. a low value of detection rate $\gamma_C=0.30$) will upsurge the population of asymptomatic infectious individuals. The impact of different levels of detection rate on the symptomatic infectious population is depicted in Figure 5. The result shows that when there is a lower progression rate from asymptomatic to symptomatic infection ($\gamma_C = 0.30$), the size of the symptomatic infectious population increases in the first 25th time interval, and then declines for the rest of the 25 time intervals. This dynamic can be explained by the fact that when there is an increase in the asymptomatic individuals due to low detection rate if infection, this will lead to an increase in the transmission rate of the disease, as the asymptomatic individuals would be unaware of their status and will not take any precautions about disease transmission. Furthermore, the later decline in the size of the symptomatic infectious population is attributed to the fact that efforts are placed on the treatment of symptomatic individuals to reduce disease burden in the population. This dynamic is also evident when progression rate from asymptomatic to symptomatic infection is high. (i.e., $\gamma_c = 0.50$). The size of the symptomatic infectious population is reduced compared to when there is a low detection rate, because with a high detection rate, individuals are aware of their infection status and thus are more likely to take precautions. This awareness will reduce the transmission rate of the disease, thus resulting in a lower number of infected individuals in the populace.

In Figure 2, we perform a simulation to examine the impact of different levels of the transmission rate from asymptomatic individuals on the infectious population. The transmission of infection from asymptomatic individuals is primarily due to their unawareness of disease status, leading to a lack of precaution in the spread of disease. The impact of different levels of the transmission rate from asymptomatic individuals on the asymptomatic and symptomatic infectious population is depicted in Figure 5 and Figure 5, respectively. The overall result shows that an increase in the transmission rate of infection from asymptomatic individuals will increase the burden of the disease in a given population over time. A higher transmission rate from asymptomatic carriers (α_C) effectively acts as a multiplier for the hidden infectious pool, increasing the rate at which undetected carriers transmit infection to susceptible

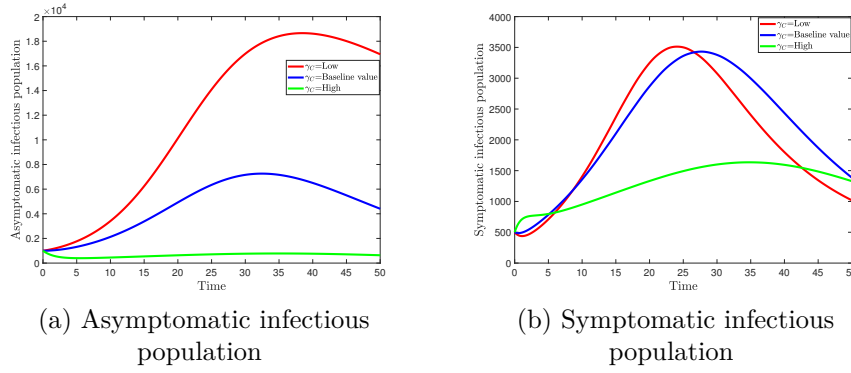


FIGURE 1. Simulation of model showing the impact of the progression rate of asymptomatic individuals to symptomatic at different levels (Low= $\gamma_c = 0.30$, High= $\gamma_c = 0.50$) on (a) asymptomatic infectious population; (b) symptomatic infectious population.

individuals. When this rate is high ($\alpha_c = 0.50$), the asymptomatic class rapidly generates new infections, leading to a surge in both asymptomatic and symptomatic infectious populations, as illustrated in Figure 5 and Figure 5.

The subsequent decline in the infectious populations is driven by the transition and recovery processes embedded in the model. In particular, the diagnostic detection rate (γ_C) gradually transfers individuals from the asymptomatic class to the symptomatic class, where they can be monitored and treated, while the symptomatic recovery rate (γ_I) removes infected individuals from the transmission cycle. Together, these mechanisms reduce the effective infectious pool over time, resulting in the decline observed in the simulations. This includes the detection of disease status due to the diagnosis and treatment of individuals. The overall output of these controls is to reduce the burden of disease in the community, resulting to a lower number of infected people in the population. In Figure 2, it is also observed that when there is a low rate of disease transmission (i.e., $\alpha_C = 0.30$), the size of the infectious population is in the neighborhood of the initial population of the infectious people. This implies that there is a small increase in the number of infectious individuals over time. This result suggests that efforts should be made towards the reduction of disease transmission in the population to keep the size of infectious individuals low.

In Figure 3, we perform a simulation to examine the impact of different levels of the transmission rate from the environmental reservoir on

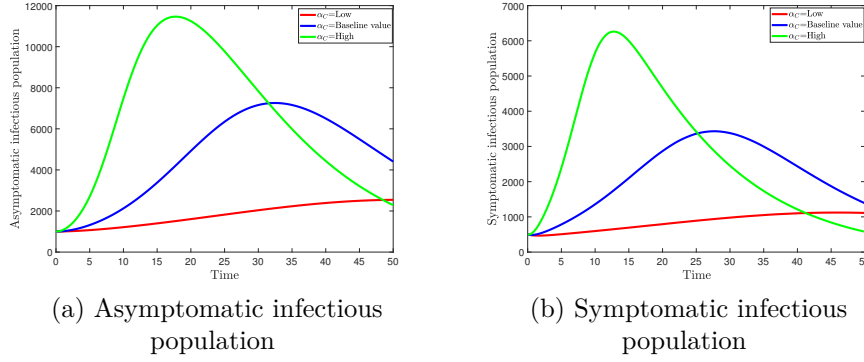


FIGURE 2. Simulation of model showing the impact of the transmission rate from asymptomatic individuals at different levels (Low= $\alpha_c = 0.30$, High= $\alpha_c = 1.5$) on (a) asymptomatic infectious population; (b) symptomatic infectious population.

the infectious population. The impact of different levels of the transmission rate from the environmental reservoir on the asymptomatic and symptomatic infectious population is depicted in Figure 5 and Figure 5, respectively. The overall result shows that an increase in the transmission rate from the environmental reservoir will increase the burden of the disease over time. A similar dynamic in Figure 2 is observed here in Figure 3. As seen in Figure 5 and Figure 5, there is a reduction in the size of the asymptomatic and symptomatic infectious populations after a period of time. In Figure 3, it is also observed that even when there is a lower rate of disease transmission from environmental reservoir (i.e., $\alpha_W = 1 \times 10^{-8}$). These results suggest that although complete elimination of environmental transmission is not strictly required to mitigate disease burden, aggressive sanitation measures are still necessary to substantially reduce the peak size of the infectious population. The simulations indicate the presence of a practical saturation point, beyond which further reductions in environmental transmission yield diminishing returns in terms of lowering infection levels. In other words, once environmental contamination has been reduced to a sufficiently low level, additional investments in sanitation may produce only marginal improvements. At this stage, the model suggests that shifting resources toward enhanced detection and monitoring of asymptomatic carriers may provide a more effective strategy for further reducing disease transmission. However, a reduction in the transmission rate from the environmental reservoir would lead to a reduction in the size of the infectious individuals in the population.

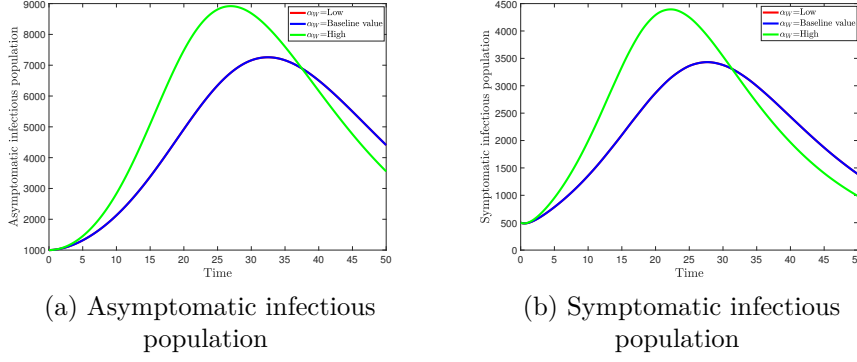


FIGURE 3. Simulation of model showing the impact of the transmission rate from the environmental reservoir at different levels ($\text{Low} = \alpha_W = 1 \times 10^{-8}$, $\text{High} = \alpha_W = 1 \times 10^{-6}$) on (a) asymptomatic infectious population; (b) symptomatic infectious population.

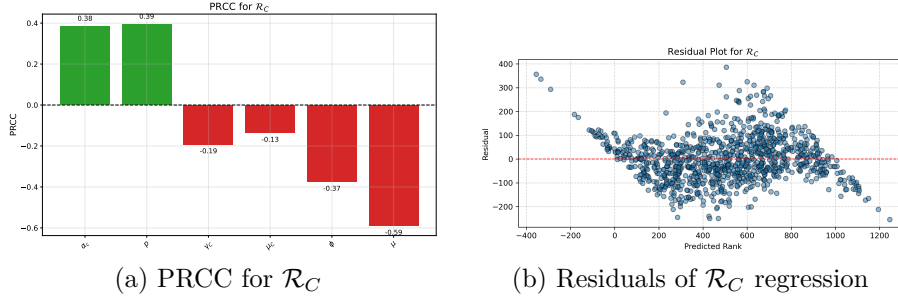


FIGURE 4. PRCC analysis and residual diagnostics for R_C .

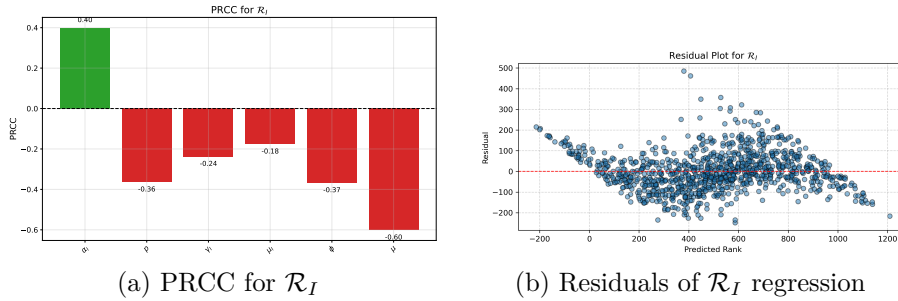
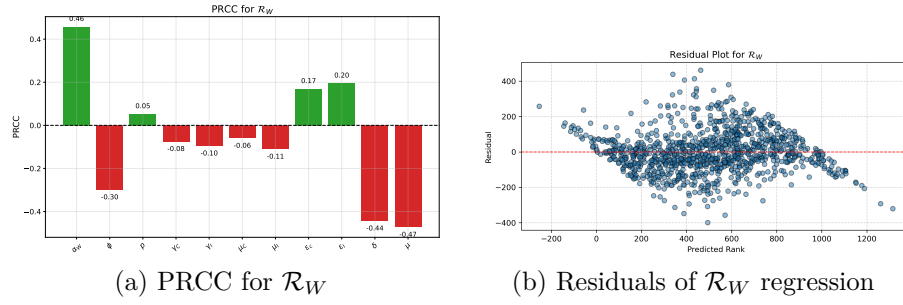
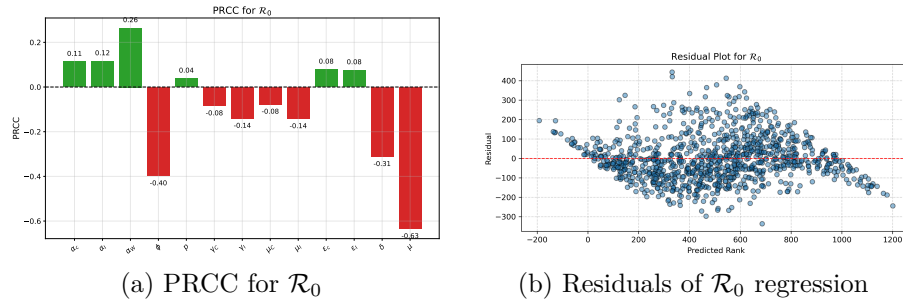


FIGURE 5. PRCC analysis and residual diagnostics for R_I .

FIGURE 6. PRCC analysis and residual diagnostics for $\mathcal{R}_W$.FIGURE 7. PRCC analysis and residual diagnostics for $\mathcal{R}_0$.

6. CONCLUSION

In this study, we developed a deterministic compartmental model to investigate the combined effects of asymptomatic carriers and environmental transmission on infectious disease dynamics. The model incorporated both direct and indirect transmission pathways and allowed us to quantify the relative contributions of asymptomatic individuals, symptomatic individuals, and environmental reservoirs to overall disease transmission.

The basic reproduction number was derived using the next-generation matrix approach and decomposed into three components corresponding to the three major transmission pathways. This decomposition provides a clearer understanding of how each pathway contributes to disease persistence. The stability analysis showed that the disease-free equilibrium is locally asymptotically stable when the basic reproduction number is less than unity, indicating that effective control measures can lead to disease elimination when transmission is sufficiently reduced.

Sensitivity analysis using the Partial Rank Correlation Coefficient method revealed that parameters associated with asymptomatic transmission and environmental contamination can have substantial effects

on the reproduction number. In particular, the transmission rate from asymptomatic carriers, the proportion of exposed individuals becoming asymptomatic, environmental transmission, and pathogen shedding rates were identified as important drivers of disease transmission. These findings highlight the importance of addressing transmission pathways that may remain hidden from conventional surveillance systems.

Numerical simulations further demonstrated that increases in the transmission rates associated with asymptomatic carriers and environmental reservoirs can increase the infectious population. Conversely, increasing the detection of asymptomatic individuals and reducing environmental transmission can substantially reduce disease burden. The results therefore suggest that relying solely on the identification and treatment of symptomatic individuals may not be sufficient for effective disease control.

Overall, the findings emphasize the need for integrated intervention strategies that combine early identification of asymptomatic carriers, appropriate management of symptomatic individuals, and effective environmental sanitation. Such combined strategies can reduce transmission through multiple pathways and improve the effectiveness of infectious disease control programs.

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DATA AVAILABILITY

Data used to support the findings of this study are included in the article. The authors used a set of parameter values whose sources are from the literature, as shown in Table 1.

CONFLICT OF INTEREST

There are no conflicts of interest to declare.

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