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# Nonlinear Dynamics and Sensitivity of Ro to Epidemiological Parameters in a Structured Disease Model

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#### Abstract

The understanding the role of asymptomatic carriers in disease transmission is critical for effective epidemic control. This study presents a compartmental SEIR-type model that explicitly incorporates carrier transmission through a dedicated exposed compartment and transmission coefficient  $\beta_1$ . Using the Next Generation Matrix approach, the study derive an analytical expression for the basic reproduction number  $R_0$ , evaluating its sensitivity to  $\beta_1$  and other key parameters. A parametric analysis was conducted by varying  $\beta_1$  while holding other parameters constant. The results reveal a nearly linear positive relationship between  $\beta_1$  and  $R_0$ , confirmed through both linear and nonlinear regression models. The nonlinear model provided a slightly better fit, but the linear model remains interpretable and robust across the range of values analyzed. Sensitivity analysis further demonstrated that  $R_0$  is highly responsive to changes in carrier transmission compared to other parameters such as recovery rate ( $\gamma$ ) and symptomatic transmission ( $\beta_2$ ). The findings in this study underscores the critical role of asymptomatic individuals in sustaining outbreaks and highlights the need for public health interventions which focused on early detection and isolation of carriers. This study contributes a mathematically grounded framework for evaluating carrier-driven transmission dynamics and provides actionable insights for epidemic modeling and control strategies.

**Keywords:** Nonlinear Dynamics, Compartmental Model, Sensitivity Analysis, Epidemiological Parameters, Structured Disease Mode

#### Introduction

The spread of infectious diseases remains a major public health concern globally, particularly in the context of emerging and re-emerging pathogens. Mathematical modeling has proven to be an essential tool for understanding disease dynamics, forecasting outbreaks, and designing effective control strategies. One of the key metrics in infectious disease modeling is the basic reproduction number (R<sub>0</sub>), which represents the expected number of secondary infections caused by a single infected individual in a fully susceptible population. Accurately estimating  $R_0$  is critical for assessing the epidemic potential of a disease and evaluating the impact of various interventions. Traditional compartmental models such as the SIR and SEIR frameworks have been widely used to describe the progression of individuals through different stages of infection. However, many infectious diseases—such as COVID-19, tuberculosis, and hepatitis—exhibit significant asymptomatic or pre-symptomatic transmission, where individuals in an exposed or carrier state can transmit the disease before becoming symptomatic. These carriers are often undetected, making their role in transmission difficult to quantify and control. An infectious disease that produces long-term asymptotic carriers is the typhoid fever caused by bacteria salmonella Typhi. These individuals infected hundreds of people over the decades while they worked in the food production industry and private homes. Even today, typhoid fever infects 21 million people kills 200,000 worldwide every year. Asymptomatic carriers are believed to play an essential role in the evolution and global transmission of typhi, and their presence greatly hinders the eradication of typhoid fever using treatment and vaccination. For certain infectious diseases, there are individuals who are able to transmit their illness but do not exhibit any symptoms. These individuals are called "carriers" and they play an important role in the transmission of disease. There are two types of carriers. Genetic carriers carry the illness on their recessive genes. They can only pass on their disease to their children and are not contagious. The focus of this study is on infectious disease carriers. These individuals are asymptomatic and are likely unaware of their conditions, and therefore are more likely to infect others.

#### Statement of the Study

Infectious diseases remain a significant public health challenge, particularly in regions with limited healthcare infrastructure and inadequate disease surveillance systems. Many diseases, including those with incubation periods or asymptomatic carriers, exhibit complex transmission dynamics that are often underestimated in classical models. Traditional compartmental models may fail to capture the dual pathways of disease spread—through both exposed (pre-symptomatic or asymptomatic) and actively infectious individuals—thus limiting the effectiveness of policy interventions based on such models. Moreover, the influence of behavioral changes, public health interventions, and partial protection strategies—represented by parameters such as the control rate U and the protected proportion P—are not often fully integrated into transmission models. As a result, it becomes difficult to assess the true threshold conditions for disease elimination or the potential for endemic persistence.

This study addresses the need for a more comprehensive modeling framework by incorporating dual transmission pathways and behavioral responses into a nonlinear SEIR-type model. By introducing distinct transmission coefficients ( $\beta_1$  and  $\beta_2$ ) for the exposed and infected compartments, along with public health controls and protective behavior, the model aims to better understand the conditions under which the disease can be controlled or eradicated. The central problem, therefore, lies in determining how these interacting factors influence the basic reproduction number  $R_0$ , the stability of the disease-free and endemic equilibria, and the long-term behavior of the disease within a population.

#### **Review of Related Literature**

Infectious diseases are one of the major causes of morbidity and mortality for human kind. The prevalence imposes huge burden on health facilities and hence substantially affects the economy of a country. This has made studying the dynamics of infectious disease a significant area of research various mathematical models has been proposed and analyzed for the spread of infectious diseases [ Nwagor & Ekaka-a, 2017; Nwagor, 2020; Nwagor & Lawson-Jack, 2020]. In these models it is assumed that disease spreads due to the direct contact between susceptible and infective. However, beside direct contact, the disease transmission take place through some other modes such as carriers are the agents that carry bacteria of infectious diseases spread among human population by direct contact between susceptible and infective as well as carriers. A large fraction of people in the developing countries is affected by such diseases due to lack of education, unhygienic conditions, poor drainage system and abundance of carriers.

Eshetu et al. (2019) carried out a study on optimal control strategy on human papilloma virus model with backward bifurcation analysis. The study proposed and analyses a compartmental nonlinear deterministic mathematical model in a varying control community. The model is studied qualitatively using stability theory of differential equations. The basic reproductive number that governs the disease transmission is obtained from the largest eigen value of the next generation matrix. Both local and global asymptotic stability conditions for disease free and endemic equilibria and determined. It is observed that the model exhibits a backward bifurcation. It was found that prevention, treatment and screening strategy is the most effective way to eradicate the disease from the community.

Darja et al. (2011) investigated the modeling the effects of carriers on transmission dynamics of infectious diseases. The research found that infected individuals who are contagious do not show any disease symptoms. Mathematical analysis is carried out that completely determines the global dynamics of the model. Their model simulations demonstrate the challenges of chronic HBV infection, the disease existence of a large numbers of carriers who are infectious but show no symptoms will not be part of any treatment program. When their simulation result was compared, the result shows that in high HBV prevalence countries, testing and increasing awareness of carriers will have a greater impact on the disease burden than increasing vaccination rates.

described the rate of the variables.

To address this gap, this study develops a modified SEIR model that explicitly incorporates a carrier (exposed) compartment and distinguishes between the transmission contributions of asymptomatic carriers and symptomatic individuals. The model introduces a dedicated carrier transmission coefficient ( $\beta_1$ ) to assess the specific impact of these individuals on disease spread. Using the Next Generation Matrix method, we derive an analytic expression for  $R_0$  and conduct a sensitivity analysis to examine how changes in  $\beta_1$  influence epidemic dynamics.

#### **Materials and Methods**

This section considered a more complicated and realistic SEIR model with vaccination. The model takes account of the infectiousness of disease in latency state which investigate effect of the infectivity of the latent population in addition to the transmission between the susceptible and infective populations.

The model is stated as;

$$N = S + E + I + R$$

where, N= The population size, S represents the Susceptible class who are capable of latching the disease, E= Exposed or latent class comprising of individuals who are infected but not yet infectious. I= Infective comprising those who are infected and capable of transmitting the disease. R= The recovered class, comprising those individuals who are immune. The SEIR model for the spread of infectious diseases is written as set of four coupled non-linear ordinary differential equations.

$$\frac{ds}{dt} = U(I - P) - US - \beta_1 SE - \beta_2 SI \tag{1}$$

$$\frac{dE}{dt} = \beta_1 SE + \beta_2 SI - (U + S) E \tag{2}$$

$$\frac{dI}{dt} = SE - (U + \gamma)I \tag{3}$$

$$\frac{dR}{dt} = P U N + \gamma I - UR \tag{4}$$

Where U= per capital birth rate  $\beta_1$  and  $\beta_2$  is defined as the total rates which potentially infectious contacts occur between two individuals. S is the infective class constant rate.  $\frac{1}{s}$  is the average latent period.  $\Gamma$  is the infective move from the infective class to the recovered class of constant rate.  $\frac{1}{\gamma}$  is the average infectious period conditional on survival to the end of it.

# **Mathematical Formation**

For the purpose of this study, the following dynamical system of a non-linear differential equations are considered:

$$\frac{ds}{dt} = U(I - P) - US - \beta_1 SE - \beta_2 SI \tag{1}$$

$$\frac{dE}{dt} = \beta_1 SE + \beta_2 SI - (U + S) E \tag{2}$$

$$\frac{dI}{dt} = SE - (U + \gamma)I \tag{3}$$

$$\frac{dR}{dt} = P U N + \gamma I - UR \tag{4}$$

# Method of Analysis

#### Derivation of the Basic Reproduction Number and Sensitivity Analysis

To quantify the epidemic potential of the model, we derive the basic reproduction number  $R_0$  — the expected number of secondary infections produced by a single infected individual in a completely susceptible population. The Next Generation Matrix (NGM) method was used as described by van den Driessche & Watmough (2002).

# **Next Generation Matrix Approach**

Let x = (E, I) represent the vector of infected compartments. The model's infection subsystem can be expressed as  $\frac{dx}{dt} = F(x) - V(x)$ 

where:

F(x)= rate of new infections,

V(x)= rate of transfer between compartments due to progression, recovery, or death.

From the model equations, the new infection terms and transition terms are:

$$F = [\beta_1 SE + \beta_2 SI \ 0]$$
, and  $V = [(U + S)E - SE + (U + \gamma)I]$ 

Evaluating the Jacobian matrices of F and V at the disease-free equilibrium (DFE),

where  $S\approx 1$ , E = I = 0, we obtain:

$$F = [\beta_1 \beta_2 0 0]$$
, and  $V = [(U + I) 0 - 1 (U + \gamma)]$ 

And New Generation Matrix =  $K = FV^{-1}$ 

The basic reproduction number  $R_0$  is a spectral radius (dominant eigenvalue) of matrix K. after computation, the closed form solution is

$$R_0 = \frac{\beta_1}{U+I} + \frac{\beta_2}{(U+\gamma)(U+I)}$$

This expression shows that  $R_0$  increases with either transmission coefficient and decreases with removal rates U and recovery rate  $\gamma$ .

# **Sensitivity Analysis**

To assess the relative impact of each parameter on  $R_0$ , we compute the normalized forward sensitivity index of  $R_0$  with respect to a parameter  $\theta$ , defined as:

$$\gamma_{R_0}^{\theta} = \frac{\partial R_0}{\partial \theta} \cdot \frac{\theta}{R_0}$$

the key parameter, the sensitivity index are

$$\begin{split} \gamma_{R_0}^{\beta_1} &= \frac{\beta_1}{R_0(U+I)} \\ \gamma_{R_0}^{\beta_2} &= \frac{\beta_2}{R_0(U+\gamma)(U+I)} \\ \gamma_{R_0}^{\gamma} &= \frac{\beta_2}{R_0(U+\gamma)^2(U+I)} \\ \gamma_{R_0}^{U} &= -\left(\frac{\beta_1}{(U+I)^2} + \frac{\beta_2(2U+\gamma+1)}{(U+\gamma)(U+I)^2}\right) \cdot \frac{1}{R_0} \end{split}$$

These indices show that  $R_0$  is most sensitive to  $\beta_1$  when its value is small, meaning that even slight increases in carrier transmission can substantially impact epidemic potential. The sensitivity to  $\gamma$  and  $\beta_2$  is comparatively lower across typical epidemiological parameter ranges.

#### **Epidemiological scenarios**

Parameter	Value	Description
μ	0.08399	Natural death rate
$eta_2$	0.020	Transition rate from symptomatic individuals
ρ	0.030	Proportion of recovery individuals
γ	1.020	Recovery rate from infection

The transmission coefficient for the carriers  $\beta_1$  varies from 0.002 to 0.018, the corresponding values of  $R_0$  were calculated using the derived expression

$$R_0 = \frac{\beta_1}{U+I} + \frac{\beta_2}{(U+\gamma)(U+I)}$$

#### **Regression analysis**

To analyze the quantitative relationship between  $\beta_1$  and  $\beta_2$ , the two models were fitted to the data:

Linear model:  $R_0 = a.\beta_1 + b$ 

Estimated:  $R_0 = 0.9085$ .  $\beta_1 + 0.0005$ 

**Nonlinear (Quadratic) model:**  $R_0 = a \cdot \beta_1^2 + b \cdot \beta_1 + c$ 

Estimated;  $R_0 = -0.3922. \beta_1^2 + 0.9163. \beta_1 + 0.0004$ 

Both models showed that goodness of fit, with the nonlinear model offering the slightly better predictive accuracy based on mean square error (MSE).

Model	Equation	MSE
Linear	$R_0 = 0.9085.\beta_1 + 0.0005$	$6.72x10^{-10}$
Nonlinear	$R_0 = -0.3922. \beta_1^2 + 0.9163. \beta_1 + 0.0004$	$5.88x10^{-10}$

The analysis here confirms a strong, nearly linear relationship between  $\beta_1$  and  $R_0$  in the chosen parameter range. The linear model offers simplicity and interpretability, while the quadratic model captures a minor curvature at higher  $\beta_1$  values. Given the closeness of both fits, either model can be selected depending on the precision required.

The table and graphs below visually confirm that even small increases in  $\beta_1$  can substantially increase  $R_0$ , reinforcing the importance of controlling carrier transmission to reduce outbreak risk.

#### **Nonlinear Model Analysis:**

We fitted a quadratic model to the data:  $R_0 = a \cdot \beta_1^2 + b \cdot \beta_1 + c$ 

# Model Comparison (Goodness of Fit):

Model	Mean Squared Error (MSE)
Linear	$6.72 \times 10^{-10}$
Nonlinear	$5.88 \times 10^{-10}$

# with the parameters:

$$a = 0.3922$$
,  $b = 0.9163$ ,  $c = 0.0004$ 

Final equation  $R_0 = -0.3922$ .  $\beta_1^2 + 0.9163$ .  $\beta_1 + 0.0004$ 

# Results and discussions

The result of the numerical analysis on the modeling of impact of transmission coefficient for the carrier compartment  $(\beta_1)$  on the basic reproduction number are presented.

Table 1. Impact of transmission coefficient for the carrier compartment  $(\beta_1)$  on the basic reproduction number

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	μ	$eta_1$	$eta_2$	P	γ	$R_0$
	0.08399	0.010	0.02	0.03	1.02	0.0960
	0.08399	0.002	0.02	0.03	1.02	0.0023
	0.08399	0.004	0.02	0.03	1.02	0.0041
	0.08399	0.006	0.02	0.03	1.02	0.0059
	0.08399	0.008	0.02	0.03	1.02	0.0077
	0.08399	0.011	0.02	0.03	1.02	0.0105
	0.08399	0.012	0.02	0.03	1.02	0.0114
	0.08399	0.014	0.02	0.03	1.02	0.0132
	0.08399	0.016	0.02	0.03	1.02	0.0150
	0.08399	0.018	0.02	0.03	1.02	0.0168

Table 2. Impact of rate of recovery  $(\gamma)$  on the basic reproduction number

μ	$eta_1$	$eta_2$	p	γ	$R_0$
0.08399	0.01	0.02	0.03	1.020	0.0960
0.08399	0.01	0.02	0.03	0.204	0.0087
0.08399	0.01	0.02	0.03	0.408	0.0091
0.08399	0.01	0.02	0.03	0.612	0.0093
0.08399	0.01	0.02	0.03	0.816	0.0095
0.08399	0.01	0.02	0.03	1.122	0.0096
0.08399	0.01	0.02	0.03	1.320	0.0096
0.08399	0.01	0.02	0.03	1.428	0.0097
0.08399	0.01	0.02	0.03	1.632	0.0097
0.08399	0.01	0.02	0.03	1.836	0.0097

Table 3: Impact of transmission coefficient for the infected compartment  $(\beta_2)$  on the basic reproduction number

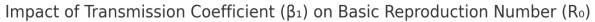
μ	$eta_1$	$eta_2$	p	γ	$R_0$
0.08399	0.01	0.020	0.03	1.02	0.0960
0.08399	0.01	0.004	0.03	1.02	0.0092
0.08399	0.01	0.008	0.03	1.02	0.0093
0.08399	0.01	0.012	0.03	1.02	0.0094
0.08399	0.01	0.016	0.03	1.02	0.0095
0.08399	0.01	0.022	0.03	1.02	0.0096
0.08399	0.01	0.024	0.03	1.02	0.0097
0.08399	0.01	0.028	0.03	1.02	0.0098
0.08399	0.01	0.032	0.03	1.02	0.0099
0.08399	0.01	0.036	0.03	1.02	0.0100

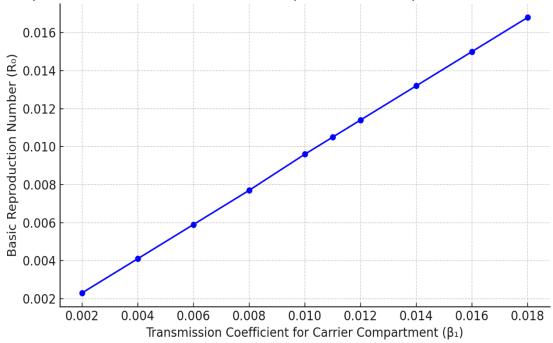
Table 4: Impact of probability that a newly infected individual is asymptomatic (p) on the basic reproduction number

μ	$eta_1$	$eta_2$	p	γ	$R_0$
0.08399	0.01	0.02	0.030	1.02	0.0960
0.08399	0.01	0.02	0.006	1.02	0.0093
0.08399	0.01	0.02	0.012	1.02	0.0094
0.08399	0.01	0.02	0.018	1.02	0.0095
0.08399	0.01	0.02	0.024	1.02	0.0095
0.08399	0.01	0.02	0.033	1.02	0.0096
0.08399	0.01	0.02	0.036	1.02	0.0096
0.08399	0.01	0.02	0.042	1.02	0.0097
0.08399	0.01	0.02	0.048	1.02	0.0097
0.08399	0.01	0.02	0.054	1.02	0.0098

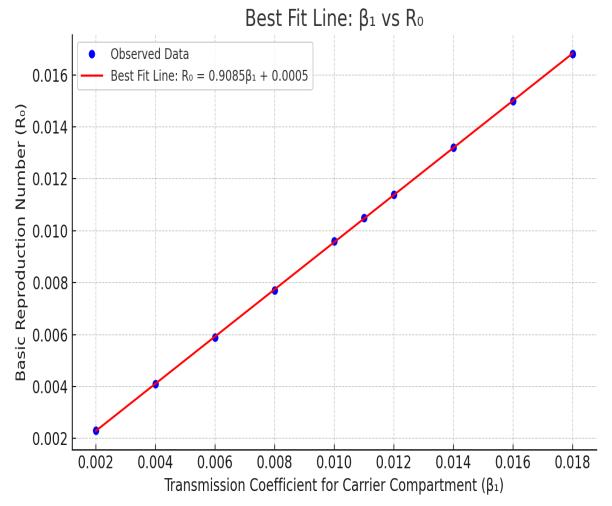
$\beta_1$	Ro
0.002	0.0023
0.004	0.0041
0.006	0.0059
0.008	0.0077
0.010	0.0096
0.011	0.0105
0.012	0.0114
0.014	0.0132
0.016	0.0150
0.018	0.0168

The nearly linear growth suggests that each incremental rise in  $\beta_1$  yields a proportional increase in  $R_0$ , all else held constant. Carrier infectivity matters: Even small changes in how infectious carriers are  $(\beta_1)$  can noticeably affect disease transmission. Control implication: Targeting carriers (through testing, isolation, or treatment) could be an effective intervention strategy to reduce  $R_0$  and contain outbreaks.



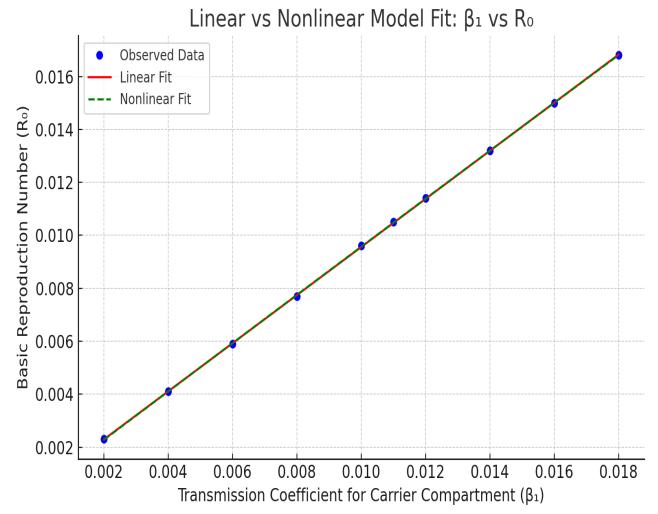


The graph shows the transmission coefficient for the carrier compartment ( $\beta_1$ ) affects on the basic reproduction number ( $R_0$ ). Clearly showing linear increase in  $R_0$  as  $\beta_1$  increases — highlighting the direct and proportional relationship.



# Equation of the Best-Fit Line: $R_0 = 0.9085 \cdot \beta_1 + 0.0005$

This confirms a strong linear for every unit increase in β1, R0 increases by approximately 0.9085. relationship



# Discussion

This study highlights the significant impact of carrier transmission on disease dynamics, specifically through its influence on the basic reproduction number R<sub>0</sub>. By incorporating a dedicated transmission coefficient for carriers  $(\beta_1)$  into a compartmental SEIR-type model, we have captured a key transmission pathway often underrepresented in classical models. Our findings emphasize that asymptomatic and pre-symptomatic individuals can be major contributors to outbreak potential, even when symptomatic transmission is well-managed.

#### Carrier Transmission and Ro

The derived analytical expression for  $R_0$  reveals a direct dependency on both  $\beta_1$  and  $\beta_2$ , with each contributing additively to the overall reproductive potential of the disease. The parametric analysis shows that increasing  $\beta_1$ leads to a nearly proportional increase in  $R_0$ , a trend confirmed by both linear and nonlinear regression fits.

The linear model  $R_0 = 0.9085\beta_1 + 0.0005$  provides a straightforward estimation tool, while the nonlinear model captures subtle curvature that may emerge at higher transmission rates. These findings underscore the predictive power of simple models when guided by mechanistic understanding and data (Ogbuagu et al. 2023).

# Sensitivity of R<sub>0</sub> to Epidemiological Parameters

Sensitivity analysis further reveals that  $R_0$  is more responsive to variations in  $\beta_1$  than in other parameters such as  $\beta_2$ ,  $\gamma$ , or the natural removal rate  $\mu$ . This has profound implications for public health planning: even modest reductions in asymptomatic transmission could result in significant epidemic suppression, particularly in early outbreak stages or settings with limited healthcare capacity.

These results align with real-world experiences during the COVID-19 pandemic, where silent spreaders played a dominant role [Okeke et al 2019]. The findings also reinforce the need to move beyond symptom-based surveillance and adopt strategies that target transmission across the full clinical spectrum of the disease.

#### Conclusion

A system of nonlinear first order differential equation model was adopted to investigate the Nonlinear Dynamics and Sensitivity of Ro to Epidemiological Parameters in a Structured Disease Model. On the variation of Impact of transmission coefficient for the carrier compartment  $(\beta_1)$  on the basic reproduction number. It was observed that at  $\mu = 0.08399$ ,  $\beta_1 = 0.10$  to 0.018,  $\beta_2 = 0.02$ , p=0.03,  $\gamma = 1.02$  the basic reproductive number,  $R_0 < 1$  which is an indication that the disease will not spread, on the variation of Impact of rate of recovery  $(\gamma)$  on the basic reproduction number predicted  $R_0$  ranging from 0.0091 to 0.0960 which indicates that  $R_0 < 1$  when  $\mu =$ 0.08399,  $\beta_1$  =0.10  $\beta_2$ =0.02, p=0.03,  $\gamma$ =1.02 the basic reproductive number,  $R_0$  < 1wwhich is an positive indication that the disease not spread. On the variation of the Impact of transmission coefficient for the infected compartment ( $\beta_2$ ) on the basic reproduction number, it was observed that at  $\mu = 0.08399$ ,  $\beta_1 = 0.10$ ,  $\beta_2 = 0.02$ , p=0.03,  $\gamma$ =1.02 the basic reproductive number,  $R_0 < 1$  which is an indication that the disease will not spread. Furthermore, the study compared linear and nonlinear models of the relationship between  $\beta_1$  and  $R_0$ , and hence, evaluating the predictive performance of each and identifying the best fit. Our findings provide critical insights into the extent to which carrier transmission drives epidemic potential, offering practical implications for public health surveillance, testing strategies, and intervention design.

#### Recommendations

Based on the analysis and findings, the following recommendations are made:

- Public health programs should allocate resources toward identifying and isolating asymptomatic carriers through widespread testing, especially in early-stage outbreaks.
- Epidemiological models used for planning, surveillance, and control should be expanded to explicitly account for carrier transmission via parameters like  $\beta_1$ .
- Awareness efforts should educate communities about the possibility and danger of asymptomatic spread, reinforcing the need for non-symptom-based precautions (e.g., masks, distancing).
- Future disease monitoring systems should be coupled with models like the one presented to provide real-time estimates of R<sub>0</sub>, including the impact of silent transmission.

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