Research Article

Analytical Insights of the Effects of Non-Pharmaceutical Interventions on SARS-CoV-2 Dynamic with Application to West African Data

Kassifou Traore[1], Djideme F. Houenou[2], Romain Glèlè Kakaï[1*]

[1]Laboratory of Biomathematics and Forestry Estimations, University of Abomey-Calavi, Benin
[2]Laboratory of Biomathematics and Forestry Estimations, Department of Mathematic/Faculty of Sciences and Technics, University of Abomey-Calavi, Benin
E-mail: romain.glelekakai@fsa.uac.bj

Abstract: The COVID-19 pandemic was caused by the rapid spread of a new coronavirus (SARS-CoV-2) worldwide. COVID-19 pandemic mitigation measures caused significant social and economic disruption, especially in regions with weak economic and fragile healthcare systems like West Africa. Therefore, accurate knowledge of the impact of these measures on its dynamics is important in decision-making. In this study, we formulated and used a deterministic compartmental model, considering two sub-classes of susceptible individuals (S_1 and S_2), where S_1 is the population living around the epicenter of the epidemic and S_2 is the population living far from the epicenter of the epidemic. The aim was to (i) theoretically assess the impact of measures reducing the transmission rate of the disease (ψ) on the epidemic dynamics and (ii) analyze the impact of measures reducing the probability of contact between infected and susceptible individuals (detection and isolation rates, θ_{ap}, θ_s) on the epidemic dynamics. We determined the expressions of the basic and control reproduction numbers and studied the sensitivity and elasticity of the control reproduction number with respect to ψ, θ_{ap}, θ_s, and heterogeneity factor, k (S_1/S_2). Application to the COVID-19 first-wave data from West Africa revealed that the basic reproduction number was 1.85. Moreover, the results indicated that a 50% reduction in the transmission rate of COVID-19 or the detection and isolation of 10% of infected individuals per day should help to reach the peak of the epidemic. Furthermore, a 100% increase in the heterogeneity factor induces a 16% increase in the control reproduction number when $\theta_{ap} = 0.15$ and a 14% increase in the control reproduction number when $\theta_{ap} = 0.6$. These conclusions could help design control measures to curtail future epidemics.

Keywords: heterogeneous population, reproduction number, sensitivity and elasticity, transmission rate, type of control measures, mechanistic model

MSC: 92-10, 34H05, 37N25, 34-04

1. Introduction

The COVID-19 outbreak began with the first infected individual identified in Hubei Province, China, in late 2019. The disease is instigated by the new coronavirus (SARS-CoV-2) spreading across the world [1]. Humans can acquire the novel coronavirus when they come into contact with contaminated surfaces or from droplets released by infectious, symptomatic, presymptomatic, and asymptomatic individuals [2]. Mild to moderate infection symptoms of the disease include fever, cough, sore throat, nasal congestion, malaise, headache, muscle pain, and shortness of breath

(or tachypnea in children) [3]. In severe cases, the fever is associated with severe dyspnea, respiratory distress, and tachypnea [3]. The first case reported in West Africa was in Nigeria on February 27, 2020, two months after the first case was officially announced in China [4]. The highest disease burden in West Africa on June 27, 2020, was in Nigeria (about 23,298 cases and 554 deaths).

In general, in the absence of a vaccine or treatment available to control an epidemic, two categories of non-pharmaceutical interventions can be considered. The first one is related to measures that reduce the transmission rate of the disease, i.e., the proportion of contacts per day between susceptible and active infected individuals that induce contamination (probability of transmission of COVID-19 per contact). These measures include wearing face masks, hand washing with soap and water, and physical distancing. Empirical analyses revealed that they reduce the intensity of the peak of infection (the maximum number of new infections in a population) but lengthen its timing [5]. The second relates to measures aiming to prevent contact between susceptible and infected individuals (reducing the probability of contact). These measures include mainly systematic testing for identifying and isolating infected individuals and containment. Moreover, they bring the occurrence of the infection's peak closer but make it challenging to maintain the trend obtained [5].

Unfortunately, it is not easy to implement these basic public health measures effectively in some West African countries due to widespread poverty and poor investment in health care (staff, equipment, and infrastructure). Therefore, it is a great challenge for developing countries, especially those in the West African region, to scrutinize and determine other approaches and methods to control COVID-19. In the absence of vaccines, non-pharmaceutical interventions appear to be the most straightforward and affordable methods. Thus, optimal strategies are requested for implementing non-pharmaceutical interventions to control an epidemic before a vaccine can be developed. An optimal strategy in this sense involves weighing the relative costs of control and COVID-19 mortality to find an approach that minimizes these combined costs. Other optimal control analyses of COVID-19 are beginning to emerge [6-10]. The effect of non-pharmaceutical interventions on the spread of SARS-CoV-2 in West Africa has been studied by various authors [5, 11]. For example, using compartmental modeling of West African COVID-19 data, [5] showed that at least a 46% reduction in the transmission rate is required for disease elimination, but when combined with the detection and isolation of infected individuals, about a 29% reduction in the disease transmission rate is required for disease elimination.

Furthermore, in standard epidemic models, it is usually assumed that the population consists of homogeneous individuals who mix uniformly with one another [12]. This is a simplifying assumption that helps to make the mathematics tractable. Empirical evidence suggests that in real-world epidemics, there is often variability among individuals. It is therefore important to determine how different degrees of susceptibility in a population are likely to affect the conclusions drawn from the examination of standard epidemic models [12]. Heterogeneity exists in many aspects of disease transmission processes [13-16], for instance, gender heterogeneity (heterogeneous dynamics and behavior among groups for sexually transmitted diseases), age-related heterogeneity (heterogeneous infection among age groups), spatial heterogeneity (heterogeneous spatial distribution of host populations), etc. Some individuals have a high probability of contracting the disease (the population living around the epicenter of the disease), while others living far from the epicenter are less susceptible to being infected. The effects of control measures on the dynamics of COVID-19 in non-homogeneous populations have been considered in some studies. For example, using a numerical approach, [17] showed that the pandemic's peak time is different in Ethiopia's urban and rural populations. Varying the coverage of wearing masks in rural populations has no significant effect on the number of cases.

This study aims to analytically investigate the dynamics of an epidemic in a heterogeneous population, considering different types of non-pharmaceutical interventions with optimal control in West Africa. Specifically, we assess (i) the impact of measures that reduce the transmission rate of the epidemic, (ii) the impact of measures reducing the probability of contact between infected and susceptible individuals, and (iii) the impact of both types of measures on the dynamics of the epidemic.

2. Methods

In COVID-19 modeling, different types of models are used to understand SARS-CoV-2 transmission, such as deterministic models, stochastic models [18-25], agent-based models [26, 27], discrete models [28, 29], spatial models [30-32], network models [33, 34], etc. COVID-19 stochastic models are sometimes compartmental models with the

introduction of white noise, such as Gaussian noise or Brownian noise, to create randomness in order to find the condition of extinction and persistence on average of the disease and to prove the existence and uniqueness of the global positive solution of the model.

In this study, we used a deterministic compartmental model of the Susceptible, Exposed, Infectious, and Recovered (SEIR) framework, where the susceptible compartment is composed of populations living around the epicentre of the epidemic, S_1 (e.g., West African urban areas), and the population living far from the epicentre of the epidemic, S_2 (e.g., West African rural areas). Exposed people: E (infected but cannot transmit the virus), presymptomatic people: I_p (infected but cannot yet transmit the virus), asymptomatic people: I_a (infected without any symptoms and can transmit the virus), symptomatic people: I_s (infected with symptoms and can transmit the virus), detected infected individuals (I_D), and recovered (R). The natural immunity recovered is not permanent, and after a given period (six months [35]), they lost their immunity. At time t, one has $N(t) = S_1(t) + S_2(t) + E(t) + I_p(t) + I_a(t) + I_s(t) + I_D(t) + R(t)$. The schematic of the model is presented in Figure 1, and the description of model parameters is in Table C (see Appendix C).
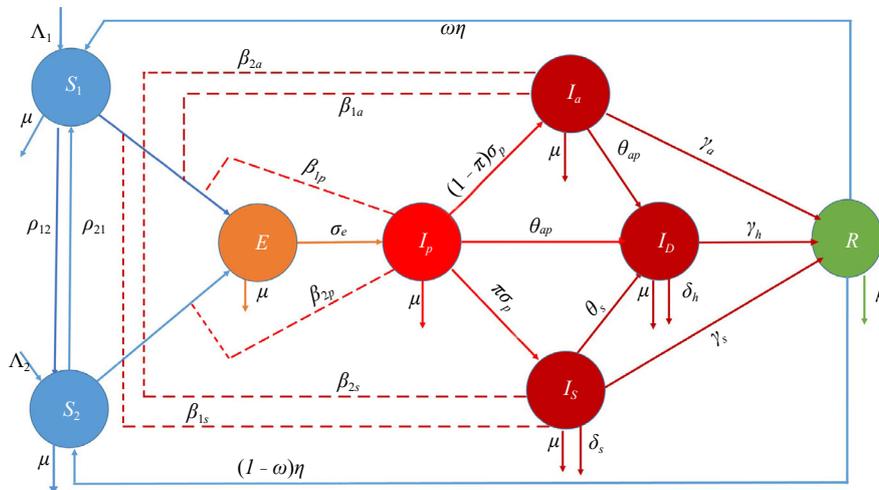

Figure 1. Flow diagram of the model depicting the movement of individuals between classes based on the disease. The susceptible population living around the epicentre of the epidemic (S_1), the susceptible population living far from the epicentre of the epidemic (S_2), exposed (E), presymptomatic (I_p), asymptomatic (I_a), symptomatic (I_s), detected infected individuals (I_D), and recovered (R). Definitions of the model parameters are provided in Table C (see Appendix C)

Using the flowchart of the model framework (Figure 1), we obtain the following ordinary differential equations (ODEs) for the model:

$$\dot{S}_1 = \Lambda_1 + \omega\eta R + \rho_{21}S_2 - \left(\lambda_1 + \mu + \rho_{12}\right)S_1, \tag{1}$$

$$\dot{S}_2 = \Lambda_2 + (1-\omega)\eta R + \rho_{12}S_1 - \left(\lambda_2 + \mu + \rho_{21}\right)S_2, \tag{2}$$

$$\dot{E} = \lambda_2 S_2 + \lambda_1 S_1 - \left(\mu + \sigma_e\right)E, \tag{3}$$

$$\dot{I}_p = \sigma_e E - \left(\sigma_p + \mu + \theta_{ap}\right)I_p, \tag{4}$$

$$\dot{I}_a = (1-\pi)\sigma_p I_p - \left(\mu + \gamma_a + \theta_{ap}\right)I_a, \tag{5}$$

$$\dot{I}_s = \pi\sigma_p I_p - (\mu + \gamma_s + \delta_s + \theta_s)I_s, \tag{6}$$

$$\dot{I}_D = \theta_{ap}I_p + \theta_{ap}I_a + \theta_s I_s - (\mu + \delta_h + \gamma_h)I_D, \tag{7}$$

$$\dot{R} = \gamma_a I_a + \gamma_h I_D + \gamma_s I_s - (\mu + \eta)R. \tag{8}$$

where

$$\begin{cases} \lambda_1 & = & \dfrac{(1-\psi)(\beta_{1a}I_a + \beta_{1s}I_s + \beta_{1p}I_p)}{N - I_D}, \\[4mm] \lambda_2 & = & \dfrac{(1-\psi)(\beta_{2a}I_a + \beta_{2s}I_s + \beta_{2p}I_p)}{N - I_D}. \end{cases}$$

The parameter $\theta_{ap} \in [0,1]$ is the detection rate of presymptomatics/asymptomatics linked to testing effort. We assumed that

$$\theta_{ap} = \alpha\theta_s \text{ with } 0 < \alpha < 1.$$

The initial conditions are:

$$S_1(0) \geqslant 0, S_2(0) \geqslant 0, E(0) \geqslant 0, I_p(0) \geqslant 0, I_a(0) \geqslant 0, I_s(0) \geqslant 0, I_D(0) \geqslant 0, R(0) \geqslant 0.$$

3. Results
3.1 *Analytical results*
3.1.1 *Basic and control reproduction numbers*

In this section, we compute the control reproduction number, R_c, defined as the average number of secondary infections caused by an infectious individual among the susceptible population in the presence of non-pharmaceutical interventions (lockdown, quarantine, and isolation of detected infected) in the community. The control reproduction number is computed at disease-free equilibrium (DFE) using the next-generation matrix approach. We determine the DFE of the model (1)-(8) by setting: $\dot{S}_1 = \dot{S}_2 = 0$ and $E = I_p = I_a = I_s = I_D = R = 0$.

Thus, we get the system:

$$\begin{cases} \Lambda_1 + \rho_{21}S_2^0 - (\mu + \rho_{21})S_1^0 = 0, \\ \Lambda_2 + \rho_{12}S_1^0 - (\mu + \rho_{12})S_2^0 = 0. \end{cases} \tag{9}$$

Therefore, the DFE of the model is given by:

$$\left(S_1^0, S_2^0, E^0, I_p^0, I_s^0, I_a^0, I_D^0, R^0\right) = \left(\frac{\rho_{21}\Lambda_2 + (\mu + \rho_{21})\Lambda_1}{\mu(\mu + \rho_{12} + \rho_{21})}, \frac{\rho_{12}\Lambda_1 + (\mu + \rho_{12})\Lambda_2}{\mu(\mu + \rho_{12} + \rho_{21})}, 0, 0, 0, 0, 0, 0\right),$$

so that $N^0 = S_1^0 + S_2^0$.

To compute the control reproduction, we considered only the five infected classes in the model: the exposed E, presymptomatic I_p, asymptomatic I_a, symptomatic I_s, and detected I_D compartments. Equations (3) to (7) of the model (1)-(8) can be written as: $X' = (f - v)X$, where $X = (E, I_p, I_a, I_s, I_D)$.

$$f = \begin{pmatrix} \lambda_1 S_1 + \lambda_2 S_2 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, v = \begin{pmatrix} (\mu + \sigma_e)E \\ -\sigma_e E + (\mu + \sigma_p + \theta_{ap})I_p \\ -(1-\pi)\sigma_p I_p + (\mu + \gamma_a + \theta_{ap})I_a \\ -\pi\sigma_p I_p + (\mu + \delta_s + \theta_s + \gamma_s)I_s \\ -(\theta_{ap}I_p + \theta_{ap}I_a + \theta_s I_s) + (\mu + \delta_h + \gamma_h)I_D \end{pmatrix}. \tag{10}$$

The corresponding Jacobian matrices F and V evaluated at DFE are given below:

$$F = \begin{pmatrix} 0 & c & d & e & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{11}$$

$$V = \begin{pmatrix} \mu + \sigma_e & 0 & 0 & 0 & 0 \\ -\sigma_e & \mu + \sigma_p + \theta_{ap} & 0 & 0 & 0 \\ 0 & -(1-\pi)\sigma_p & \mu + \gamma_a + \theta_{ap} & 0 & 0 \\ 0 & -\pi\sigma_p & 0 & \mu + \delta_s + \dfrac{\theta_{ap}}{\alpha} + \gamma_s & 0 \\ 0 & -\theta_{ap} & -\theta_{ap} & -\dfrac{\theta_{ap}}{\alpha} & \mu + \delta_h + \gamma_h \end{pmatrix}, \tag{12}$$

where

$$c = \frac{(1-\psi)(\beta_{1p}S_1^0 + \beta_{2p}S_2^0)}{S_1^0 + S_2^0}, \quad d = \frac{(1-\psi)(\beta_{1a}S_1^0 + \beta_{2a}S_2^0)}{S_1^0 + S_2^0}, \quad e = \frac{(1-\psi)(\beta_{1s}S_1^0 + \beta_{2s}S_2^0)}{S_1^0 + S_2^0}. \tag{13}$$

Therefore, the matrix $F \times V^{-1}$ is given by:

$$F \times V^{-1} = \begin{pmatrix} f_1 & f_2 & \dfrac{d}{\mu + \gamma_a + \theta_{ap}} & \dfrac{e}{\mu + \delta_s + \dfrac{\theta_{ap}}{\alpha} + \gamma_s} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{14}$$

where

$$\begin{cases} f_1 = \dfrac{1}{(\mu + \sigma_e)} \left(\dfrac{c\sigma_e}{(\mu + \sigma_p + \theta_{ap})} + \dfrac{(1-\pi)d\sigma_p\sigma_e}{(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})} + \dfrac{\pi\sigma_p e\sigma_e}{(\mu + \sigma_p + \theta_{ap})y} \right), \\[4mm] f_2 = \dfrac{c}{\mu + \sigma_p + \theta_{ap}} + \dfrac{(1-\pi)d\sigma_p}{(\mu + \gamma_a + \theta_{ap})(\mu + \sigma_p + \theta_{ap})} + \dfrac{\pi e\sigma_p}{(\mu + \sigma_p + \theta_{ap})y}, \\[4mm] y = (\mu + \delta_s + \dfrac{\theta_{ap}}{\alpha} + \gamma_s). \end{cases} \qquad (15)$$

Hence, using $R_c = \rho(F \times V^1)$ (where $\rho(.)$ represents the spectral radius), we derived the following expression for the control reproduction number:

$$R_c = \frac{1}{(\mu + \sigma_e)} \left(\frac{c\sigma_e}{(\mu + \sigma_p + \theta_{ap})} + \frac{(1-\pi)d\sigma_p\sigma_e}{(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})} + \frac{\pi\sigma_p e\sigma_e}{(\mu + \sigma_p + \theta_{ap})y} \right). \qquad (16)$$

Let us write the control reproduction number as per contributions: $R_c = R_c^p + R_c^a + R_c^s$, where

$$R_c^p = \frac{c\sigma_e}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})}, R_c^a = \frac{(1-\pi)d\sigma_p\sigma_e}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})}, \text{and } R_c^s = \frac{\pi\sigma_p e\sigma_e}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})y}.$$

We observe that the control reproduction number, R_c, is the sum of the three components defined above: R_c^p represents the contribution of presymptomatic individuals; R_c^a stands for the contribution of asymptomatic individuals; and R_c^s is the contribution of symptomatic individuals in the overall reproduction number of the epidemic.

Let's consider a ratio, $k \left(k = \dfrac{S_1^0}{S_2^0} \right)$ of the sizes of the two susceptibility classes, and that can be interpreted as the relative importance of the susceptibility classes. If $k = 1$, $S_1^0 = S_2^0$ (fair allocation of individuals between the two susceptibility classes).

The basic reproduction number, R_0, of the model is obtained from R_c when all control measures are absent. Hence, the basic reproduction number, R_0, is given by

$$R_0 = \frac{1}{(\mu + \sigma_e)(\mu + \sigma_p)} \left(c_1\sigma_e + \frac{(1-\pi)d_1\sigma_p\sigma_e}{\mu + \gamma_a} + \frac{\pi\sigma_p e_1\sigma_e}{\mu + \delta_s + \gamma_s} \right), \qquad (17)$$

where $c_1 = \dfrac{\left(\beta_{1p}S_1^0 + \beta_{2p}S_2^0 \right)}{S_1^0 + S_2^0}$, $d_1 = \dfrac{\left(\beta_{1a}S_1^0 + \beta_{2a}S_2^0 \right)}{S_1^0 + S_2^0}$, and $e_1 = \dfrac{\left(\beta_{1s}S_1^0 + \beta_{2s}S_2^0 \right)}{S_1^0 + S_2^0}$.

3.1.2 Sensitivity and elasticity analyses

The sensitivity analysis studies how various sources of uncertainty in a mathematical model contribute to the model's overall uncertainty. This technique is used within specific boundaries that depend on one or more input variables [36]. The elasticity of a parameter with respect to a variable represents the proportion of the relative change of the parameter induced by a relative change in the variable. When the parameter is a differentiable function of the variable, the sensitivity index is defined using partial derivatives with respect to that variable [37, 38]. Therefore, the mathematical definition of the elasticity of a parameter y with respect to a variable p is given as follows:

$$\frac{\partial y}{\partial p} \times \frac{p}{y}. \qquad (18)$$

To evaluate the impact of control measures on the dynamic of COVID-19, we study the sensitivity of the control reproduction number, R_c with respect to ψ, θ_{ap}, and θ_s. We also study the sensitivity of the critical value of the percentage reduction of the transmission rate with respect to the detection rate of infected individuals.

3.1.2.1 Sensitivity and elasticity of R_c with respect to the percentage reduction in the transmission rate (ψ)

The sensitivity of R_c with respect to ψ is:

$$\frac{\partial R_c}{\partial \psi} = -\frac{1}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})}\left[\frac{\sigma_e[c_1(\mu + \gamma_a + \theta_{ap}) + (1 - \pi)\sigma_p\sigma_e d_1]}{(\mu + \gamma_a + \theta_{ap})} + \frac{\pi\sigma_p\sigma_e e_1}{y}\right] \tag{19}$$

where $c_1 = \dfrac{\left(\beta_{1p}S_1^0 + \beta_{2p}S_2^0\right)}{S_1^0 + S_2^0}$, $d_1 = \dfrac{\left(\beta_{1a}S_1^0 + \beta_{2a}S_2^0\right)}{S_1^0 + S_2^0}$, and $e_1 = \dfrac{\left(\beta_{1s}S_1^0 + \beta_{2s}S_2^0\right)}{S_1^0 + S_2^0}$.

For details, see (Appendix A.1).

From the equations (16) and (19), we get: $\dfrac{\partial R_c}{\partial \psi} = -\dfrac{R_c}{1 - \psi}$. Thus, the elasticity index is given by:

$$\frac{\partial R_c}{\partial \psi} \times \frac{\psi}{R_c} = -\frac{\psi}{1 - \psi}.$$

It is worth noticing that $\dfrac{\partial R_c}{\partial \psi} < 0$, therefore when ψ increases, R_c decreases.

3.1.2.2 Sensitivity and elasticity of R_c with respect to the detection rate of presymptomatic/asymptomatic (θ_{ap})

To evaluate the impact of detection and isolation of presymptomatic and asymptomatic individuals, we determine the sensitivity of R_c with respect to θ_{ap}, which is given by:

$$\frac{\partial R_c}{\partial \theta_{ap}} = -\frac{1}{(\mu + \sigma_e)\left(\mu + \sigma_p + \theta_{ap}\right)^2}\left[c\sigma_e + \frac{(1 - \pi)\sigma_p d\sigma_e(2\mu + \sigma_p + \gamma_a + 2\theta_{ap})}{\left(\mu + \gamma_a + \theta_{ap}\right)^2} + \frac{a}{\alpha y^2}\right] \tag{20}$$

where $a = \pi\sigma_p\sigma_e e[\alpha y + \theta_{ap} + \mu + \sigma_p]$. For more details, see (Appendix A.2).

$\dfrac{\partial R_c}{\partial \theta_{ap}} < 0$, an increase in θ_{ap} leads to a reduction in R_c. Hence, the detection and isolation of presymptomatic and asymptomatic individuals reduces the spread of the disease.

The elasticity of R_c with respect to θ_{ap} is:

$$\frac{\partial R_c}{\partial \theta_{ap}} \times \frac{\theta_{ap}}{R_c} = -\frac{\theta_{ap}\left[by^2 + (\mu + \gamma_a + \theta_{ap})^2\left(a + \alpha c\sigma_e y^2\right)\right]}{\alpha y\left(\mu + \gamma_a + \theta_{ap}\right)\left(\mu + \sigma_p + \theta_{ap}\right)E} \tag{21}$$

where $b = (1 - \pi)\alpha d\sigma_p\sigma_e(2\mu + \gamma_a + \sigma_p + 2\theta_{ap})$ and $E = \left[c\sigma_e(\mu + \gamma_a + \theta_{ap})y + (1 - \pi)d\sigma_p\sigma_e y + \pi\sigma_p e\sigma_e(\mu + \gamma_a + \theta_{ap})\right]$. For more details, see (Appendix B.1).

3.1.2.3 Sensitivity and elasticity of R_c with respect to θ_s

To evaluate the impact of detection and isolation of symptomatic individuals, we determined the sensitivity of R_c with respect to θ_s, which is given by:

$$\frac{\partial R_c}{\partial \theta_s} = -\frac{\alpha \sigma_e}{(\mu + \sigma_e)(\mu + \sigma_p + \alpha \theta_s)^2}\left[c + \frac{(1-\pi)\sigma_p d(2\mu + \sigma_p + \gamma_a + 2\alpha \theta_s)}{(\mu + \gamma_a + \alpha \theta_s)^2} + \frac{m}{\alpha y^2}\right] \tag{22}$$

where $m = \pi \sigma_p e\big(\mu + \sigma_p + \alpha(y + \theta_s)\big)$ and $y = \mu + \gamma_s + \delta_s + \theta_s$.

One easily derives that $\dfrac{\partial R_c}{\partial \theta_s} < 0$. Then, an increase in θ_s induces a reduction in R_c. Therefore, the detection and isolation of symptomatic individuals reduce the spread of the disease.

The elasticity of R_c with respect to θ_s is:

$$\frac{\partial R_c}{\partial \theta_s} \times \frac{\theta_s}{R_c} = -\frac{\theta_s\left[(\mu + \sigma_p + \alpha \theta_s)^2(c\alpha y^2 + m) + (1-\pi)\sigma_p d(2\mu + \sigma_p + \gamma_a + 2\alpha \theta_s)\alpha y^2\right]}{y(\mu + \sigma_p + \alpha \theta_s)(\mu + \gamma_a + \alpha \theta_s)\left[(\mu + \gamma_a + \alpha \theta_s)(cy + \pi \sigma_p e) + (1-\pi)d\sigma_p y\right]}. \tag{23}$$

3.1.2.4 Sensitivity of R_c with respect to ψ and θ_{ap}

The effect of measures reducing disease transmission and isolating infected individuals on epidemic dynamics can be analyzed considering the sensitivity of R_c with respect to ψ and θ_{ap}. This reflects more on the control strategies being applied in different countries where detection and isolation of presymptomatic and asymptomatic individuals are associated with social distancing measures. This sensitivity represents the gradient of R_c with respect to ψ and θ_{ap}:

$$J_{(\psi, \theta_{ap})}R_c = \left(\frac{\partial R_c}{\partial \psi}; \frac{\partial R_c}{\partial \theta_{ap}}\right).$$

where $\dfrac{\partial R_c}{\partial \psi}$ is given in the equation (19) and $\dfrac{\partial R_c}{\partial \theta_{ap}}$ in the equation (20).

We know that $\dfrac{\partial R_c}{\partial \psi} < 0$ and $\dfrac{\partial R_c}{\partial \theta_{ap}} < 0$; so if all composites of the gradient are negative, then if the control parameters ψ and θ_{ap} increase, the control reproduction number will decrease.

3.1.2.5 Sensitivity and elasticity of R_c with respect to heterogeneity factor k

We assessed the sensitivity of R_c with respect to k to analyze the impact of the heterogeneity of the population on the dynamics of the epidemic.

$$R_c = \frac{1}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})}\left[c\sigma_e + \frac{(1-\pi)d\sigma_p \sigma_e}{\mu + \gamma_a + \theta_{ap}} + \frac{\pi \sigma_p e \sigma_e}{y}\right] \tag{24}$$

where

$$c = \frac{(1-\psi)\big(\beta_{1p}S_1^0 + \beta_{2p}S_2^0\big)}{S_1^0 + S_2^0}; d = \frac{(1-\psi)\big(\beta_{1a}S_1^0 + \beta_{2a}S_2^0\big)}{S_1^0 + S_2^0}; e = \frac{(1-\psi)\big(\beta_{1s}S_1^0 + \beta_{2s}S_2^0\big)}{S_1^0 + S_2^0}. \tag{25}$$

Assuming that $S_1^0 = kS_2^0$, one has:

$$c = \frac{(1-\psi)(\beta_{1p}k + \beta_{2p})}{k+1}; d = \frac{(1-\psi)(\beta_{1a}k + \beta_{2a})}{k+1}; e = \frac{(1-\psi)(\beta_{1s}k + \beta_{2s})}{k+1}. \tag{26}$$

The sensitivity of R_c with respect to k is given by:

$$\frac{\partial R_c}{\partial k} = \frac{(1-\psi)\sigma_e}{(\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})(1+k)^2}\left[\beta_{1p} - \beta_{2p} + \frac{(1-\pi)\sigma_p(\beta_{1a} - \beta_{2a})}{\mu+\gamma_a+\theta_{ap}} + \frac{\pi\sigma_p(\beta_{1s} - \beta_{2s})}{y}\right]. \tag{27}$$

For details, see (Appendix A.3).

Since the population S_1 lives in the epicenter of the epidemic and the population S_2 lives far from the epicenter, $\beta_{1p} > \beta_{2p}, \beta_{1a} > \beta_{2a}$, and $\beta_{1s} > \beta_{2s}$, therefore, $\frac{\partial R_c}{\partial k} > 0$.

Hence, if the heterogeneity factor k increases, the control reproduction number, R_c, increases. However, an increase in k leads to an increase in S_1^0. Therefore, if the susceptible population S_1^0 living in the epidemic's epicenter increases, the disease's spread also increases. So, the decrease in the susceptible population S_1^0 leads to a reduction in R_c.

The elasticity of R_c with respect to k is:

$$\frac{\partial R_c}{\partial k}\frac{k}{R_c} = \frac{k\left[(\beta_{1p} - \beta_{2p})xy + (1-\pi)\sigma_p y(\beta_{1a} - \beta_{2a}) + \pi\sigma_p x(\beta_{1s} - \beta_{2s})\right]}{\left[c_2 xy + d_2(1-\pi)\sigma_p y + e_2\pi\sigma_p x\right](1+k)^2} \tag{28}$$

where $x = \mu+\gamma_a+\theta_{ap}, y = \mu+\gamma_s+\delta_s+\frac{\theta_{ap}}{\alpha}, c_2 = \frac{(\beta_{1p}k + \beta_{2p})}{k+1}, d_2 = \frac{(\beta_{1a}k + \beta_{2a})}{k+1}$, and $e_2 = \frac{(\beta_{1s}k + \beta_{2s})}{k+1}$. For more details, see (Appendix B.2).

3.1.2.6 Sensitivity of R_c with respect to k and ψ

Here, we evaluate the impact of the heterogeneity and the measures reducing the disease transmission by computing the gradient of R_c with respect to k and ψ.

$$J_{(\psi,k)}R_c = \left(\frac{\partial R_c}{\partial \psi}; \frac{\partial R_c}{\partial k}\right)$$

where $\frac{\partial R_c}{\partial \psi}$ is given in the equation (19) and $\frac{\partial R_c}{\partial k}$ in the equation (27).

$\frac{\partial R_c}{\partial \psi} < 0$ and $\frac{\partial R_c}{\partial k} > 0$, if k and ψ increase, the control reproduction number decreases in the direction of ψ and increases in the direction of k. Therefore, when the susceptible population S_1^0 living in the epicenter of the disease decreases and the percentage reduction in disease transmission increases, the spread of the disease will decrease.

3.1.2.7 Sensitivity and elasticity analyses of the parameters at the peak time

The peak is reached if the control reproduction number, R_c, drops to one. We determine the expressions, ψ_c, of the control parameter ψ to attend the peak and determine the sensitivity of ψ_c with respect to θ_{ap}.

Hereafter, we derive the expression ψ_c of the parameter ψ from the control reproduction number by setting $R_c = 1$, which infers:

$$\frac{1-\psi_c}{\mu+\sigma_e}\left[\frac{c_1\sigma_e}{(\mu+\sigma_p+\theta_{ap})} + \frac{(1-\pi)d_1\sigma_p\sigma_e}{(\mu+\sigma_p+\theta_{ap})(\mu+\gamma_a+\theta_{ap})} + \frac{\pi\sigma_p e_1\sigma_e}{(\mu+\sigma_p+\theta_{ap})y}\right] = 1.$$

Therefore,

$$\psi_c = 1 - \frac{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})y}{c_1\sigma_e(\mu + \gamma_a + \theta_{ap})y + (1-\pi)d_1\sigma_p\sigma_e y + \pi\sigma_p e_1\sigma_e(\mu + \gamma_a + \theta_{ap})}. \tag{29}$$

If $\psi < \psi_c$, the peak will not be reached, and the disease won't be curtailed. If $\psi > \psi_c$, the control reproduction number becomes less than 1, and the epidemic will die out.

The sensitivity of ψ_c with respect to θ_{ap} helps to see how the critical value of the percentage reduction in disease transmission changes when the detection and isolation rate of presymptomatic/asymptomatic individuals changes. Its sensitivity is given by:

$$\frac{\partial \psi_c}{\partial \theta_{ap}} = -\frac{H}{\alpha\left[c_1\sigma_e(\mu + \gamma_a + \theta_{ap})y + (1-\pi)d_1\sigma_p\sigma_e y + \pi\sigma_p e_1\sigma_e(\mu + \gamma_a + \theta_{ap})\right]^2}, \tag{30}$$

where $H = (\mu + \sigma_e)\left[(\mu + \gamma_a + \theta_{ap})^2\left(\alpha c_1\sigma_e y^2 + \pi e_1\sigma_e\sigma_p(\mu + \sigma_p + \alpha y + \theta_{ap})\right) + B\right]$ and $B = (1-\pi)\alpha d_1\sigma_e\sigma_p y^2(2\mu + \sigma_p + \gamma_a + 2\theta_{ap})$. For details, see (Appendix A.4).

$\frac{\partial \psi_c}{\partial \theta_{ap}} < 0$. So, if θ_{ap} increases, ψ_c decreases at the peak time. Therefore, increasing the detection and isolation of presymptomatic/asymptomatic individuals helps decrease the critical value of the percentage reduction in disease transmission to reach the peak.

The elasticity of ψ_c with respect to θ_{ap} is:

$$\frac{\partial \psi_c}{\partial \theta_{ap}} \times \frac{\theta_{ap}}{\psi_c} = -\frac{H\theta_{ap}}{\alpha E_1\left[E_1 - (\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})y\right]} \tag{31}$$

with $E_1 = c_1\sigma_e(\mu + \gamma_a + \theta_{ap})y + (1-\pi)d_1\sigma_p\sigma_e y + \pi\sigma_p e_1\sigma_e(\mu + \gamma_a + \theta_{ap})$. For more details, see (Appendix B.3).

3.2 Numerical analyses

The implementation of basic public health control measures against COVID-19 spread in most West African countries started on March 15, 2020. Most of the parameters used in this study are those estimated in [5] using the first wave data from February 28, 2020, in West Africa, one day after the first case was detected, until June 26, 2020.

The detection rates are considered equal to 0.103 and 0.059 for symptomatic and asymptomatic, respectively [5]. By assuming $\theta_{ap} = \alpha\theta_s, \alpha \approx \frac{0.059}{0.103} \approx 0.5728$. In 2020, the urbanization ratio in West Africa was 47% [39], then $S_1^0 = 0.47 \times N^0$. Based on the latest United Nations estimates, the population of West Africa in 2020 is 399,386,502 [5], so $N^0 = 399,386,502$. Based on the statistics above, we have $S_1^0 \approx 187,711,656$ and $\_2^0 \approx 211,674,846$.

According to [5], the average incubation period is 5.1 days. Hence, $\frac{1}{\sigma_e} + \frac{1}{\sigma_p} \approx 5.1$. The latency period was taken equal 3 days [4]; thus, $\frac{1}{\sigma_e} \approx 3$, so $\sigma_e \approx 0.33$. Therefore, the average period of presymptomatic infected individuals to present symptoms or not $\left(\frac{1}{\sigma_p}\right)$ is the difference between the incubation period and the latency period. So, $\frac{1}{\sigma_p} \approx 5.1 - 3$, then $\sigma_p \approx 0.4$.

We considered that $\beta_{1p} \approx \beta_{1a}$ and $\beta_{2p} \approx \beta_{2a}$. According to [5], the disease transmission rates by individuals in the asymptomatic class and symptomatic class are $\beta_a = 0.373$ and $\beta_s = 0.197$ in West Africa, respectively. We are supposed to have $\beta_{1a} = 0.7 \times \beta_a$ and $\beta_{1s} = 0.7 \times \beta_s$, so $\beta_{1a} = 0.261$; $\beta_{2a} = 0.373 - 0.261 = 0.112$; $\beta_{1s} = 0.138$; and $\beta_{2s} = 0.059$.

The values of the fixed parameters of the model are provided in Table 1.

Table 1. Model parameter values were extracted from the literature

Parameters	Value	Confidence interval	Source	Unit
β_{1a}	0.261	–	Calculated from [5]	day^{-1}
β_{1s}	0.138	–	Calculated from [5]	day^{-1}
β_{1p}	0.261	–	Calculated from [5]	day^{-1}
β_{2a}	0.112	–	Calculated from [5]	day^{-1}
β_{2s}	0.059	–	Calculated from [5]	day^{-1}
β_{2p}	0.112	–	Calculated from [5]	day^{-1}
σ_e	0.33	–	Calculated from [40]	day^{-1}
σ_p	0.4	–	Calculated from [40, 41]	day^{-1}
γ_s	0.057	(0.053 – 0.060)	[5]	day^{-1}
δ_s	0.361	(0.315 – 0.406)	[5]	day^{-1}
γ_a	1/9.5	–	[5]	day^{-1}
γ_h	1/10	–	[5]	day^{-1}
δ_h	0.026	–	[5]	day^{-1}
$1 - \pi$	0.780	–	[5]	–
μ	1/23082.6	–	Calculated from [42]	day^{-1}
α	0.5728	–	Calculated from [5]	day^{-1}
η	1/180	–	[35]	day^{-1}
ω	0.47	–	[39]	–

3.2.1 *Investigating the impact of the percentage reduction in COVID-19 transmission on the control reproduction number in West Africa*

Figure 2(a) shows the decrease in the control reproduction number if the percentage reduction in disease transmission rate increases, whatever the value of the detection and isolation rate of infected individuals. The basic reproduction number, R_0, is 1.85. For $\theta_{ap} = 0$, the control reproduction number is 0.92 if $\psi = 0.5$, meaning that in the absence of detection and isolation of infected individuals, a 50% reduction in disease transmission is required to curtail the first wave of the epidemic. For θ_{ap} values greater than or equal to 0.1, the control reproduction number is less than one for any value of ψ. Therefore, if at least 10% of presymptomatic/asymptomatic individuals are detected and isolated per day, the first wave of the disease will be curtailed. From Figure 2(b), the elasticity of R_c with respect to ψ is -0.66 for $\psi = 0.40$. Hence, a 40% reduction in the transmission rate induces a relative change of 66% in the control reproduction number for any value of the detection and isolation rate of presymptomatic/asymptomatic individuals per day. The elasticity of R_c with respect to ψ is –1 for $\psi = 0.50$. Hence, a 50% reduction in the transmission rate induces a relative decrease of 100% in the control reproduction number for any detection and isolation rate value of presymptomatic/asymptomatic individuals per day. For $\psi = 0.8$, the elasticity of R_c with respect to ψ is –4. Therefore, an 80% reduction in the transmission rate induces a relative decrease of 400% in the control reproduction number for any value of the detection and isolation rate of presymptomatic/asymptomatic individuals per day.
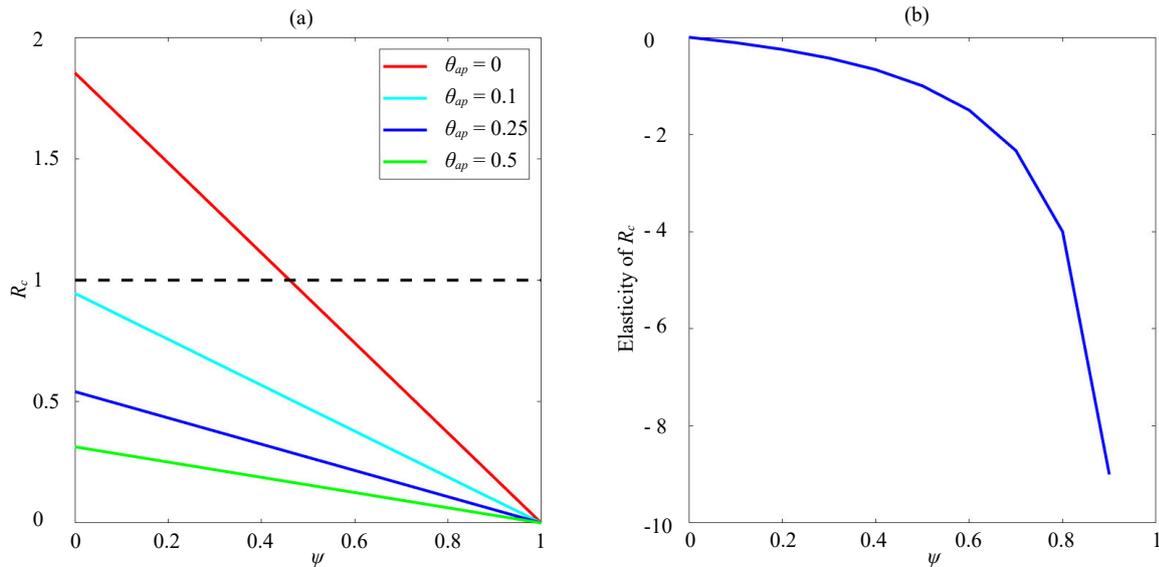
Figure 2. Impact of the percentage reduction in disease transmission on the control reproduction number. (a) Plot of control reproduction number R_c against the percentage reduction in disease transmission rate ψ for different values of the detection rate θ_{ap} of presymptomatic/asymptomatic individuals; (b) elasticity of R_c with respect to the percentage reduction in disease transmission rate ψ

3.2.2 *Assessing the impact of θ_{ap} and θ_s on the control reproduction number for different values of ψ in West Africa*

Figure 3(a) shows the decrease of the control reproduction number when the detection rate of presymptomatic/asymptomatic individuals increases, regardless of the percentage reduction in disease transmission rate. For $\psi = 0$, the control reproduction number is 0.94 if $\theta_{ap} = 0.1$, meaning that in cases of no reduction in disease transmission, at least 10% of presymptomatic/asymptomatic individuals must be detected and isolated per day to curtail the first wave of the epidemic. For $\psi = 0.1$, the control reproduction number is 0.85 if $\theta_{ap} = 0.1$. Thus, with a 10% reduction in disease transmission, at least 10% of presymptomatic/asymptomatic individuals must be detected and isolated daily to curtail the first wave of the epidemic. For $\psi = 0.25$, the control reproduction number is 0.94 if $\theta_{ap} = 0.05$. Therefore, with a 25% reduction in disease transmission, at least 5% of presymptomatic/asymptomatic individuals must be detected and isolated daily to curtail the first wave of the disease. For the value of ψ greater than or equal to 0.5, the control reproduction number is less than one for all values of θ_{ap}, meaning that at least a 50% reduction in disease transmission is required to curtail the first wave of the disease. Figure 3(b) shows that the elasticity of R_c with respect to the detection and isolation rate θ_{ap} decreases if θ_{ap} increases, whatever the value of the reduction in disease transmission. The elasticity of R_c is -0.78 if $\theta_{ap} = 0.2$. Then, a 20% increase in the detection of presymptomatic/asymptomatic individuals per day induces a relative decrease of 78% in the control reproduction number for any value of the reduction in disease transmission. The elasticity of R_c is -1.11 if $\theta_{ap} = 0.5$, meaning that a 50% increase in the detection of presymptomatic/asymptomatic individuals per day induces a relative decrease of 111% in the control reproduction number for any value of the reduction in disease transmission.
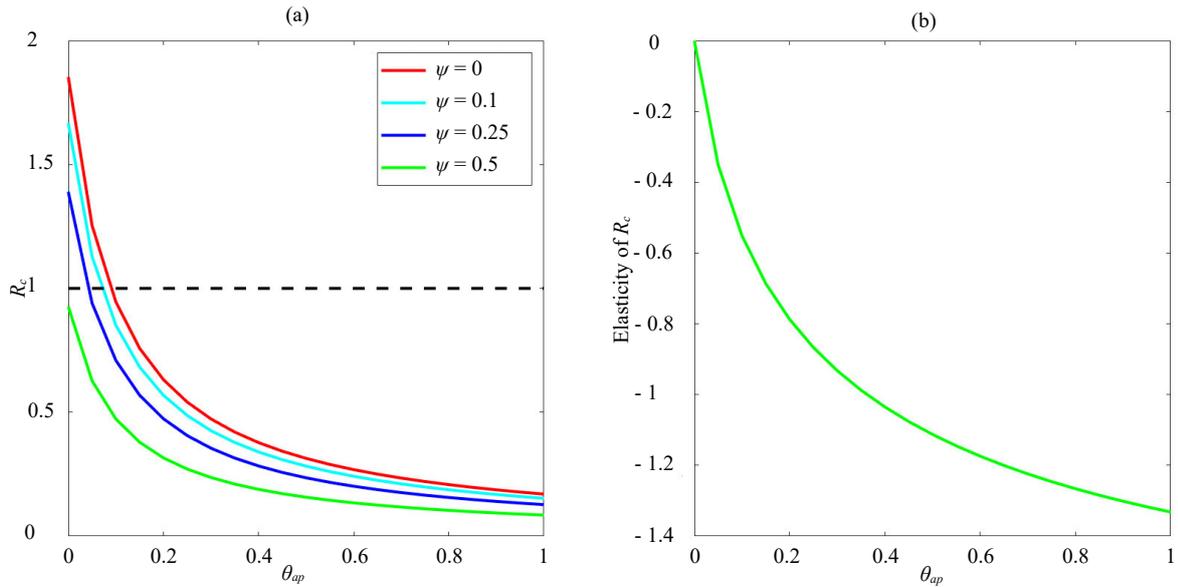
Figure 3. Impact of the detection and isolation of the infected individuals on the control reproduction number. (a) Plot of control reproduction number R_c against the detection rate of presymptomatic/asymptomatic individuals, θ_{ap} for different values percentage reduction in disease transmission rate ψ; (b) plot of elasticity of R_c with respect to the detection rate of presymptomatic/asymptomatic individuals, θ_{ap} against θ_{ap}

3.2.3 *Investigating the impact of the detection and isolation of the presymptomatic/asymptomatic (θ_{ap}) on the percentage reduction in disease transmission (ψ) to attend the peak*

Our result shows that the critical value of ψ, (ψ_c) is positive only for $\theta_{ap} \in [0, 0.09]$. We then focus our analysis on this interval $[0, 0.09]$. From Figure 4(a), we conclude that an increase in the detection and isolation rate of presymptomatic/ asymptomatic individuals leads to a decrease in the critical value of ψ. Figure 4(b) displays the relative reduction of the critical value ψ_c depending on θ_{ap}. The elasticity of ψ_c is -0.74 for $\theta_{ap} = 0.04$. Therefore, a 4% change in the detection and isolation of presymptomatic/asymptomatic individuals induces a relative decrease of 74% in the critical value, ψ_c.
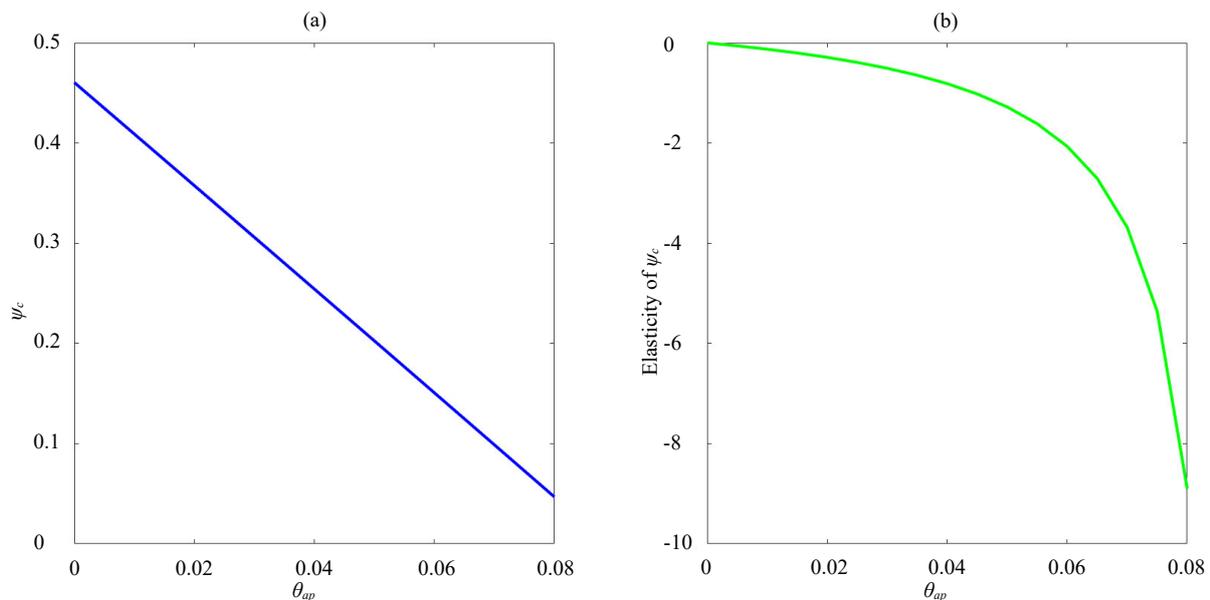

Figure 4. Impact of the detection of presymptomatic/asymptomatic individuals on the value of the percentage reduction in disease transmission to reach the peak. (a) Plot of the value of percentage reduction in disease transmission to attend the peak, ψ_c (critical value of ψ) against the detection rate of presymptomatic/asymptomatic individuals, θ_{ap}; (b) plot of elasticity of ψ_c with respect to θ_{ap} against θ_{ap}

3.2.4 *Assessing the impact of the heterogeneity factor on the control reproduction number for different values of detection and isolation rate of presymptomatic/asymptomatic individuals*

The result shows that the elasticity of R_c with respect to k increases for $\theta_{ap} \in [0, 0.15]$ and decreases for $\theta_{ap} \in [0.15, 1]$. Moreover, for $\theta_{ap} = 0.15$, the elasticity of R_c with respect to k is 0.16. Thus, a 100% change in heterogeneity factor induces a 16% relative change in the control reproduction number when $\theta_{ap} = 0.15$. For $\theta_{ap} = 0.6$, the elasticity of R_c with respect to k is 0.14. Thus, a 100% change in heterogeneity factor induces a relative change of 14% in the control reproduction number when $\theta_{ap} = 0.6$ (Figure 5).
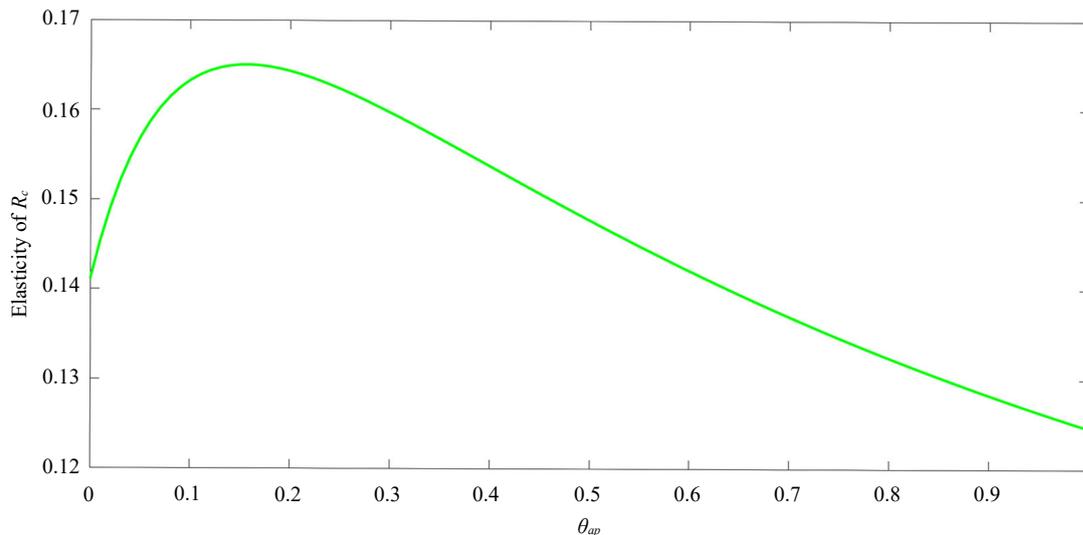

Figure 5. Plot of the elasticity of the control reproduction number, R_c, with respect to the factor heterogeneity $\left(\dfrac{S_1^0}{S_2^0} \right)$, against the detection and isolation rate of presymptomatic/asymptomatic individuals, θ_{ap} for $k = 0.89$

4. Discussion

The COVID-19 pandemic has become a great challenge to the health systems of many countries. Its rapid spread worldwide has sent a clear message to increase the readiness for a suitable health policy related to intervention strategies. Mathematical models have been useful in understanding the dynamics of disease and assessing the impact of non-pharmaceutical control measures on the dynamics of COVID-19 [5, 43-46]. This study developed a mathematical model to analytically evaluate the effects of non-pharmaceutical interventions on COVID-19 dynamics in West Africa. This study used an analytical approach to assess the impact of the measures reducing disease transmission and the detection and isolation of infected individuals on the dynamics of the epidemic, considering a heterogeneous population. The results show that measures reducing disease transmission, detection, and isolation of infected individuals reduce the spread of the disease. We used the numerical approach to verify the analytical results. The basic reproduction number $R_0 = 1.853$, similar to the value obtained by [5], can be explained by the effect of heterogeneity in the susceptible class. Therefore, the disease is still spreading ($R_0 > 1$) in West Africa with a high contribution of presymptomatic and asymptomatic individuals (80% [47]), according to the high values of the transmission rate for presymptomatic and asymptomatic individuals. Also, the individuals living around the epicentre of the epidemic (urban areas for West Africa) have a high transmission of the disease (90%) [48]. This study shows that in the case of no detection and isolation of presymptomatic and asymptomatic individuals, the high implementation of the measures reducing disease transmission (at least 50%) will significantly reduce the burden of COVID-19 and allow the control of the first wave of the disease. In the absence of measures reducing disease transmission, the high detection and isolation of infected individuals (at least 20%) will curtail the first wave of the epidemic. In addition, the study shows that the combination of two categories of non-pharmaceutical control measures will reduce the cost of implementation of control measures and can curtail the epidemic. Furthermore, it is important to

point out the role of vaccination in lowering the incidence of SARS-CoV-2 infections and COVID-19 hospital admission cases in Africa [49].

5. Conclusion

In this work, we evaluated the impact of control measures such as measures reducing the transmission rate of the epidemic (face mask wearing, hand washing with soap and water, and physical distancing) and measures that aim to prevent contact between susceptible (healthy) and infected individuals (systematic testing for identification and isolation of infected individuals) in the spread of COVID-19 in West Africa. The basic reproduction number is $R_0 = 1.853$, revealing that COVID-19 is contagious with a high contribution of presymptomatic and asymptomatic individuals. The results show that implementing measures to reduce disease transmission and detect and isolate presymptomatic and asymptomatic individuals through routine testing and isolation of positive individuals significantly reduces the disease burden. Furthermore, if measures to reduce the transmission of the disease are implemented, considering the heterogeneity factor, the spread of the disease will be reduced. For the safety of mankind, this study strongly suggests strict adherence to measures to reduce the transmission of COVID-19. Nevertheless, this study's results constitute the basis for more investigation of the effects of control measures on epidemic dynamics.

Conflict of interest

The authors declare no conflict of interest.

References

[1] Wong E, Ho KF, Wong SYS, Cheung AWL, Yeoh EK. Workplace safety and coronavirus disease (COVID-19) pandemic: Survey of employees. *Bulletin of the World Health Organization*. [Preprint] 2020.

[2] Bai Y, Yao L, Wei T, Tian F, Jin D-Y, Chen L, et al. Presumed asymptomatic carrier transmission of COVID-19. *JAMA*. 2020; 323(14): 1406-1407. Available from: https://doi.org/10.1001/jama.2020.2565.

[3] Lai C-C, Shih T-P, Ko W-C, Tang H-J, Hsueh P-R. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and coronavirus disease-2019 (COVID-19): The epidemic and the challenges. *International Journal of Antimicrobial Agents*. 2020; 55(3): 105924. Available from: https://doi.org/10.1016/j.ijantimicag.2020.105924.

[4] World Health Organization. *Coronavirus disease (COVID-19) technical guidance*. Available from: https://www.who.int/emergencies/diseases/novel-coronavirus-2019/technical-guidance [Accessed 4th March 2020].

[5] Taboe HB, Salako KV, Tison JM, Ngonghala CN, Kakaï RG. Predicting COVID-19 spread in the face of control measures in West Africa. *Mathematical Biosciences*. 2020; 328: 108431. Available from: https://doi.org/10.1016/j.mbs.2020.108431.

[6] Martinez-Alvarez M, Jarde A, Usuf E, Brotherton H, Bittaye M, Samateh AL, et al. COVID-19 pandemic in West Africa. *The Lancet Global Health*. 2020; 8(5): E631-E632. Available from: https://doi.org/10.1016/S2214-109X(20)30123-6.

[7] Djidjou-Demasse R, Michalakis Y, Choisy M, Sofonea MT, Alizon S. Optimal COVID-19 epidemic control until vaccine deployment. *medRxiv* [Preprint] 2020. Version 3. Available from: https://doi.org/10.1101/2020.04.02.20049189.

[8] Mallela A. Optimal control applied to a SEIR model of 2019-nCoV with social distancing. *medRxiv* [Preprint] 2020. Version 2. Available from: https://doi.org/10.1101/2020.04.10.20061069.

[9] Patterson-Lomba O. Optimal timing for social distancing during an epidemic. *medRxiv* [Preprint] 2020. Version 2. Available from: https://doi.org/10.1101/2020.03.30.20048132.

[10] Piguillem F, Shi L. Optimal COVID-19 quarantine and testing policies. *The Economic Journal*. 2022; 132(647): 2534-2562. Available from: https://doi.org/10.1093/ej/ueac026.

[11] Harling G, Gómez-Olivé FX, Tlouyamma J, Mutevedzi T, Kabudula CW, Mahlako R, et al. Protective behaviours and secondary harms from non-pharmaceutical interventions during the COVID-19 epidemic in South Africa: A

multisite prospective longitudinal study. *medRxiv* [Preprint] 2020. Version 1. Available from: https://doi.org/10.110
1/2020.11.12.20230136.

[12] Becker N, Marschner I, Gabriel J, Lefevre C, Picard P. The effect of heterogeneity on the spread of disease. In: Gabriel, JP, Lefèvre C, Picard P. (eds.) *Stochastic processes in epidemic theory*. Berlin, Heidelberg: Springer; 1990. p.90-103. Available from: https://doi.org/10.1007/978-3-662-10067-7_9.

[13] Andersson H, Britton T. Heterogeneity in epidemic models and its effect on the spread of infection. *Journal of Applied Probability*. 1998; 35(3): 651-661. Available from: https://doi.org/10.1239/jap/1032265213.

[14] Anderson RM, May RM. Spatial, temporal, and genetic heterogeneity in host populations and the design of immunization programmes. *Mathematical Medicine and Biology: A Journal of the IMA*. 1984; 1(3): 233-266. Available from: https://doi.org/10.1093/imammb/1.3.233.

[15] Hagenaars TJ, Donnelly CA, Ferguson NM. Spatial heterogeneity and the persistence of infectious diseases. *Journal of Theoretical Biology*. 2004; 229(3): 349-359. Available from: https://doi.org/10.1016/j.jtbi.2004.04.002.

[16] Lloyd AL, May RM. Spatial heterogeneity in epidemic models. *Journal of Theoretical Biology*. 1996; 179(1): 1-11. Available from: https://doi.org/10.1006/jtbi.1996.0042.

[17] Ejigu BA, Asfaw MD, Cavalerie L, Abebaw T, Nanyingi M, Baylis M. Assessing the impact of non-pharmaceutical interventions (NPI) on the dynamics of COVID-19: A mathematical modelling study of the case of Ethiopia. *PLoS ONE*. 2021; 16(11): e0259874. Available from: https://doi.org/10.1371/journal.pone.0259874.

[18] Niño-Torres D, Ríos-Gutiérrez A, Arunachalam V, Ohajunwa C, Seshaiyer P. Stochastic modeling, analysis, and simulation of the COVID-19 pandemic with explicit behavioral changes in Bogotá: A case study. *Infectious Disease Modelling*. 2022; 7(1): 199-211. Available from: https://doi.org/10.1016/j.idm.2021.12.008.

[19] Tesfay A, Saeed T, Zeb A, Tesfay D, Khalaf A, Brannan J. Dynamics of a stochastic COVID-19 epidemic model with jump-diffusion. *Advances in Difference Equations*. 2021; 2021: 228. Available from: https://doi.org/10.1186/s13662-021-03396-8.

[20] El Koufi A, El Koufi N. Stochastic differential equation model of COVID-19: Case study of Pakistan. *Results in Physics*. 2022; 34: 105218. Available from: https://doi.org/10.1016/j.rinp.2022.105218.

[21] Zhang Z, Zeb A, Hussain S, Alzahrani E. Dynamics of COVID-19 mathematical model with stochastic perturbation. *Advances in Difference Equations*. 2020; 2020: 451. Available from: https://doi.org/10.1186/s13662-020-02909-1.

[22] Sun J, Gao M, Jiang D. Threshold dynamics and the density function of the stochastic coronavirus epidemic model. *Fractal and Fractional*. 2022; 6(5): 245. Available from: https://doi.org/10.3390/fractalfract6050245.

[23] Lakhal M, Taki R, El Fatini M, El Guendouz T. Quarantine alone or in combination with treatment measures to control COVID-19. *The Journal of Analysis*. 2023; 31: 2347-2369. Available from: https://doi.org/10.1007/s41478-023-00569-4.

[24] Taki R, El Fatini M, El Khalifi M, Lakhal M, Wang K. Understanding death risks of COVID-19 under media awareness strategy: A stochastic approach. *The Journal of Analysis*. 2022; 30: 79-99. Available from: https://doi.org/10.1007/s41478-021-00331-8.

[25] Liu P, Huang L, Din A, Huang X. Impact of information and Lévy noise on stochastic COVID-19 epidemic model under real statistical data. *Journal of Biological Dynamics*. 2022; 16(1): 236-253. Available from: https://doi.org/10.1080/17513758.2022.2055172.

[26] Ciunkiewicz P, Brooke W, Rogers M, Yanushkevich S. Agent-based epidemiological modeling of COVID-19 in localized environments. *Computers in Biology and Medicine*. 2022; 144: 105396. Available from: https://doi.org/10.1016/j.compbiomed.2022.105396.

[27] Staffini A, Svensson AK, Chung U-I, Svensson T. An agent-based model of the local spread of SARS-CoV-2: Modeling study. *JMIR medical informatics*. 2021; 9(4): e24192. Available from: https://doi.org/10.2196/24192.

[28] Sitthiwirattham T, Zeb A, Chasreechai S, Eskandari Z, Tilioua M, Djilali S. Analysis of a discrete mathematical COVID-19 model. *Results in Physics*. 2021; 28: 104668. Available from: https://doi.org/10.1016/j.rinp.2021.104668.

[29] Singh RA, Lal R, Kotti RR. Time-discrete SIR model for COVID-19 in Fiji. *Epidemiology & Infection*. 2022; 150: e75. Available from: https://doi.org/10.1017/S0950268822000590.

[30] Guliyev H. Determining the spatial effects of COVID-19 using the spatial panel data model. *Spatial Statistics*. 2020; 38: 100443. Available from: https://doi.org/10.1016/j.spasta.2020.100443.

[31] Adekunle IA, Onanuga AT, Akinola OO, Ogunbanjo OW. Modelling spatial variations of coronavirus disease (COVID-19) in Africa. *Science of the Total Environment*. 2020; 729: 138998. Available from: https://doi.

org/10.1016/j.scitotenv.2020.138998.

[32] Rendana M, Idris WMR, Rahim SA. Spatial distribution of COVID-19 cases, epidemic spread rate, spatial pattern, and its correlation with meteorological factors during the first to the second waves. *Journal of Infection and Public Health*. 2021; 14(10): 1340-1348. Available from: https://doi.org/10.1016/j.jiph.2021.07.010.

[33] Karaivanov A. A social network model of COVID-19. *PLoS ONE*. 2020; 15(10): e0240878. Available from: https://doi.org/10.1371/journal.pone.0240878.

[34] Chang S, Pierson E, Koh PW, Gerardin J, Redbird B, Grusky D, et al. Mobility network models of COVID-19 explain inequities and inform reopening. *Nature*. 2021; 589(7840): 82-87. Available from: https://doi.org/10.1038/s41586-020-2923-3.

[35] Zuo J, Dowell AC, Pearce H, Verma K, Long HM, Begum J, et al. Robust SARS-CoV-2-specific T cell immunity is maintained at 6 months following primary infection. *Nature Immunology*. 2021; 22(5): 620-626. Available from: https://doi.org/10.1038/s41590-021-00902-8.

[36] Capristan FM. *Advances in flight safety analysis for commercial space transportation*. PhD thesis. Stanford University; 2016.

[37] Nadeem F, Zamir M, Tridane A, Khan Y. Modeling and control of zoonotic cutaneous leishmaniasis. *Punjab University Journal of Mathematics*. 2019; 51(2): 105-121.

[38] Mondal J, Samui P, Chatterjee AN. Optimal control strategies of non-pharmaceutical and pharmaceutical interventions for COVID-19 control. *Journal of Interdisciplinary Mathematics*. 2021; 24(1): 125-153. Available from: https://doi.org/10.1080/09720502.2020.1833459.

[39] Walther OJ. Urbanisation and demography in North and West Africa, 1950-2020. *West African Papers*. 2021; 33: 31. Available from: https://doi.org/10.1787/4fa52e9c-en.

[40] Bialek S, Gierke R, Hughes M, McNamara LA, Pilishvili T, Skoff T. Coronavirus disease 2019 in children—United States, February 12-April 2, 2020. *Morbidity and Mortality Weekly Report*. 2020; 69(14): 422.

[41] Lauer SA, Grantz KH, Bi Q, Jones FK, Zheng Q, Meredith HR, et al. The incubation period of coronavirus disease 2019 (COVID-19) from publicly reported confirmed cases: Estimation and application. *Annals of Internal Medicine*. 2020; 172(9): 577-582. Available from: https://doi.org/10.7326/M20-0504.

[42] Macrotrends. *Africa life expectancy 1950-2023*. Available from: https://www.macrotrends.net/countries/AFR/africa/life-expectancy [Accessed dd mm yy].

[43] Flaxman S, Mishra S, Gandy A, Unwin HJT, Mellan TA, Coupland H, et al. Estimating the effects of non-pharmaceutical interventions on COVID-19 in Europe. *Nature*. 2020; 584(7820): 257-261. Available from: https://doi.org/10.1038/s41586-020-2405-7.

[44] Ferguson N, Laydon D, Nedjati Gilani G, Imai N, Ainslie K, Baguelin M, et al. Report 9: Impact of non-pharmaceutical interventions (NPIs) to reduce COVID19 mortality and healthcare demand. *Imperial College London*. 2020. Available from: https://doi.org/10.25561/77482.

[45] Ngonghala CN, Iboi E, Eikenberry S, Scotch M, MacIntyre CR, Bonds MH, et al. Mathematical assessment of the impact of non-pharmaceutical interventions on curtailing the 2019 novel coronavirus. *Mathematical Biosciences*. 2020; 325: 108364. Available from: https://doi.org/10.1016/j.mbs.2020.108364.

[46] Lai S, Ruktanonchai NW, Zhou L, Prosper O, Luo W, Floyd JR, et al. Effect of non-pharmaceutical interventions to contain COVID-19 in China. *Nature*. 2020; 585(7825): 410-413. Available from: https://doi.org/10.1038/s41586-020-2293-x.

[47] Soy A. *Coronavirus en Afrique : Cinq raisons pour lesquelles le Covid-19 a été moins meurtrier qu'ailleurs*. BBC News Afrique. Available from: https://www.bbc.com/afrique/region-54464350 [Accessed 8th October 2022].

[48] Guterres A. *COVID-19 in an urban world. United Nation*. Available from: https://www.un.org/en/coronavirus/covid-19-urban-world [Accessed dd mm yy].

[49] Montcho Y, Nalwanga R, Azokpota P, Doumate JT, Lokonon BE, Salako VK, et al. Assessing the impact of vaccination on the dynamics of COVID-19 in Africa: A mathematical modeling study. *Vaccines*. 2023; 11(4): 857. Available from: https://doi.org/10.3390/vaccines11040857.

Appendix

A. Sensitivity analyses

A.1. The sensitivity of R_c with respect to ψ

$$\frac{\partial R_c}{\partial \psi} = \frac{1}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})}\left[\sigma_e \frac{\partial c}{\partial \psi} + \frac{(1-\pi)\sigma_e \sigma_p}{\mu + \gamma_a + \theta_{ap}} \times \frac{\partial d}{\partial \psi} + \frac{\pi \sigma_e \sigma_p}{y} \times \frac{\partial e}{\partial \psi}\right]. \tag{32}$$

where $\dfrac{\partial c}{\partial \psi} = -\dfrac{\beta_{1p}S_1^0 + \beta_{2p}S_2^0}{S_1^0 + S_2^0}, \dfrac{\partial d}{\partial \psi} = -\dfrac{\beta_{1a}S_1^0 + \beta_{2a}S_2^0}{S_1^0 + S_2^0}$, and $\dfrac{\partial e}{\partial \psi} = -\dfrac{\beta_{1s}S_1^0 + \beta_{2s}S_2^0}{S_1^0 + S_2^0}$.

Then,

$$\begin{cases} \dfrac{\partial c}{\partial \psi} = -c_1, \\[2mm] \dfrac{\partial d}{\partial \psi} = -d_1, \\[2mm] \dfrac{\partial e}{\partial \psi} = -e_1. \end{cases} \tag{33}$$

with

$$\begin{cases} c_1 = \dfrac{\beta_{1p}S_1^0 + \beta_{2p}S_2^0}{S_1^0 + S_2^0}, \\[3mm] d_1 = \dfrac{\beta_{1a}S_1^0 + \beta_{2a}S_2^0}{S_1^0 + S_2^0}, \\[3mm] e_1 = \dfrac{\beta_{1s}S_1^0 + \beta_{2s}S_2^0}{S_1^0 + S_2^0}. \end{cases} \tag{34}$$

Therefore,

$$\frac{\partial R_c}{\partial \psi} = \frac{-1}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})}\left[\sigma_e c_1 + \frac{(1-\pi)\sigma_e \sigma_p}{\mu + \gamma_a + \theta_{ap}}d_1 + \frac{\pi \sigma_e \sigma_p}{y}e_1\right]. \tag{35}$$

A.2. The sensitivity of R_c with respect to θ_{ap}

$$\frac{\partial R_c}{\partial \theta_{ap}} = \frac{\partial R_c^p}{\partial \theta_{ap}} + \frac{\partial R_c^a}{\partial \theta_{ap}} + \frac{\partial R_c^s}{\partial \theta_{ap}}. \tag{36}$$

where, $\dfrac{\partial R_c^p}{\partial \theta_{ap}} = -\dfrac{c\sigma_e}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})^2} \times \dfrac{\partial(\mu + \sigma_p + \theta_{ap})}{\partial \theta_{ap}}$, $\dfrac{\partial R_c^s}{\partial \theta_{ap}} = -\dfrac{\pi \sigma_e \sigma_p e}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})^2 y^2} \times \dfrac{\partial(\mu + \sigma_p + \theta_{ap})y}{\partial \theta_{ap}}$, with

$y = \mu + \delta_s + \dfrac{\theta_{ap}}{\alpha} + \gamma_s$ and

$$\frac{\partial R_c^a}{\partial \theta_{ap}} = -\frac{(1-\pi)\sigma_e \sigma_p d}{(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})^2(\mu + \gamma_a + \theta_{ap})^2} \times \frac{\partial(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})}{\partial \theta_{ap}}.$$

Then,

$$\frac{\partial R_c^p}{\partial \theta_{ap}} = -\frac{c\sigma_e}{(\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})^2}, \frac{\partial R_c^s}{\partial \theta_{ap}} = -\frac{\pi\sigma_e\sigma_p e\left[\mu+\sigma_p+\theta_{ap}+\alpha y\right]}{\alpha(\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})^2 y^2},$$

and

$$\frac{\partial R_c^a}{\partial \theta_{ap}} = -\frac{(1-\pi)\sigma_e\sigma_p d\left(2\mu+\sigma_p+\gamma_a+2\theta_{ap}\right)}{(\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})^2(\mu+\gamma_a+\theta_{ap})^2}.$$

After rearrangement, we get the equation (20).

A.3. *The sensitivity of R_c with respect to k*

$$\frac{\partial R_c}{\partial k} = \frac{1}{(\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})}\left[\sigma_e \times \frac{\partial c}{\partial k} + \frac{(1-\pi)\sigma_e\sigma_p}{\mu+\gamma_a+\theta_{ap}} \times \frac{\partial d}{\partial k} + \frac{\pi\sigma_e\sigma_p}{y} \times \frac{\partial e}{\partial k}\right]. \tag{37}$$

The $c = \frac{(1-\psi)(\beta_{1p}k+\beta_{2p})}{k+1}, d = \frac{(1-\psi)(\beta_{1a}k+\beta_{2a})}{k+1},$ and $c = \frac{(1-\psi)(\beta_{1s}k+\beta_{2s})}{k+1}.$

$$\begin{cases} \dfrac{\partial c}{\partial k} = \dfrac{(1-\psi)(\beta_{1p}-\beta_{2p})}{(k+1)^2}, \\[3mm] \dfrac{\partial d}{\partial k} = \dfrac{(1-\psi)(\beta_{1a}-\beta_{2a})}{(k+1)^2}, \\[3mm] \dfrac{\partial e}{\partial k} = \dfrac{(1-\psi)(\beta_{1s}-\beta_{2s})}{(k+1)^2}. \end{cases} \tag{38}$$

Therefore,

$$\frac{\partial R_c}{\partial k} = \frac{(1-\psi)\sigma_e}{(\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})(1+k)^2}\left[\beta_{1p}-\beta_{2p} + \frac{(1-\pi)\sigma_p(\beta_{1a}-\beta_{2a})}{\mu+\gamma_a+\theta_{ap}} + \frac{\pi\sigma_p(\beta_{1s}-\beta_{2s})}{y}\right]. \tag{39}$$

A.4. *The sensitivity of ψ_c with respect to θ_{ap}*

$$\psi_c = 1 - \frac{C}{E_1} \tag{40}$$

where $C = (\mu+\sigma_e)(\mu+\sigma_p+\theta_{ap})(\mu+\gamma_a+\theta_{ap})y$ and $E_1 = c_1\sigma_e(\mu+\gamma_a+\theta_{ap})y + (1-\pi)d_1\sigma_p\sigma_e y + \pi\sigma_p e_1\sigma_e(\mu+\gamma_a+\theta_{ap}).$

Therefore,

$$\frac{\partial \psi_c}{\partial \theta_{ap}} = -\frac{\dfrac{\partial C}{\partial \theta_{ap}} \times E_1 - C \times \dfrac{\partial E_1}{\partial \theta_{ap}}}{E_1^2}. \tag{41}$$

$\dfrac{\partial C}{\partial \theta_{ap}} \times E_1 - C \times \dfrac{\partial E_1}{\partial \theta_{ap}} = H,$ with $H = (\mu+\sigma_e)\left[(\mu+\gamma_a+\theta_{ap})^2\left(\alpha c_1\sigma_e y^2 + \pi e_1\sigma_e\sigma_p(\mu+\sigma_p+\alpha y+\theta_{ap})\right) + B\right]$ and $B = (1$

$$-\pi)\alpha d_1 \sigma_e \sigma_p y^2 (2\mu + \sigma_p + \gamma_a + 2\theta_{ap}).$$

Hence,

$$\frac{\partial \psi_c}{\partial \theta_{ap}} = -\frac{H}{\alpha E_1^2}. \tag{42}$$

B. Elasticity analyses

B.1. *Elasticity of R_c with respect to θ_{ap}*

One has

$$R_c = \frac{(\mu + \gamma_a + \theta_{ap})(c\sigma_e y + \pi\sigma_p e\sigma_e) + (1-\pi)d\sigma_p \sigma_e y}{y(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})} \tag{43}$$

and

$$\frac{\partial R_c}{\partial \theta_{ap}} = -\frac{(\alpha y^2 c\sigma_e + a)(\mu + \gamma_a + \theta_{ap})^2 + (1-\pi)\alpha y^2 \sigma_p d\sigma_e (2\mu + \sigma_p + \gamma_a + 2\theta_{ap})}{\alpha y^2 (\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})^2 (\mu + \gamma_a + \theta_{ap})^2}, \tag{44}$$

where $a = \pi\sigma_p \sigma_e e[\alpha y + \theta_{ap} + \mu + \sigma_p]$.

Therefore,

$$\frac{\partial R_c}{\partial \theta_{ap}} \times \frac{\theta_{ap}}{R_c} = -\frac{\theta_{ap}\left[by^2 + (\mu + \gamma_a + \theta_{ap})^2 (a + \alpha c\sigma_e y^2)\right]}{\alpha y(\mu + \gamma_a + \theta_{ap})(\mu + \sigma_p + \theta_{ap})E} \tag{45}$$

where $b = (1-\pi)\alpha d\sigma_p \sigma_e (2\mu + \gamma_a + \sigma_p + 2\theta_{ap})$ and $E = \left[c\sigma_e(\mu + \gamma_a + \theta_{ap})y + (1-\pi)d\sigma_p \sigma_e y + \pi\sigma_p e\sigma_e(\mu + \gamma_a + \theta_{ap})\right].$

B.2. *Elasticity of R_c with respect to k*

From equation (39), one have:

$$\frac{\partial R_c}{\partial k} = \frac{(1-\psi)\sigma_e\left[(\mu + \gamma_a + \theta_{ap})(\beta_{1p} - \beta_{2p} + \pi\sigma_p(\beta_{1s} - \beta_{2s})) + (1-\pi)\sigma_p y(\beta_{1a} - \beta_{2a})\right]}{y(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})(1+k)^2}. \tag{46}$$

Moreover,

$$R_c = \frac{(1-\psi)\sigma_e\left[(\mu + \gamma_a + \theta_{ap})(y(\beta_{1p}k + \beta_{2p}) + \pi\sigma_p(\beta_{1s}k + \beta_{2s})) + (1-\pi)\sigma_p y(\beta_{1a}k + \beta_{2a})\right]}{y(\mu + \sigma_e)(\mu + \sigma_p + \theta_{ap})(\mu + \gamma_a + \theta_{ap})(1+k)}. \tag{47}$$

Since,

$$\begin{cases} c = \dfrac{(1-\psi)(\beta_{1p}k + \beta_{2p})}{k+1}, \\ d = \dfrac{(1-\psi)(\beta_{1a}k + \beta_{2a})}{k+1}, \\ c = \dfrac{(1-\psi)(\beta_{1s}k + \beta_{2s})}{k+1}. \end{cases} \tag{48}$$

Therefore,

$$\frac{\partial R_c}{\partial k} \times \frac{k}{R_c} = \frac{k\left[\left(\mu + \gamma_a + \theta_{ap}\right)\left(\beta_{1p} - \beta_{2p} + \pi\sigma_p(\beta_{1s} - \beta_{2s})\right) + (1-\pi)\sigma_p y(\beta_{1a} - \beta_{2a})\right]}{(1+k)\left[\left(\mu + \gamma_a + \theta_{ap}\right)\left(y\left(\beta_{1p}k + \beta_{2p}\right) + \pi\sigma_p\left(\beta_{1s}k + \beta_{2s}\right)\right) + z\right]} \quad (49)$$

with $z = (1-\pi)\sigma_p y\left(\beta_{1a}k + \beta_{2a}\right)$.

B.3. *Elasticity of ψ_c with respect to θ_{ap}*

We know that $\dfrac{\partial \psi_c}{\partial \theta_{ap}} = -\dfrac{H}{\alpha E_1^2}$ and $\psi_c = \dfrac{E_1 - C}{E_1}$. Therefore,

$$\frac{\partial \psi_c}{\partial \theta_{ap}} \times \frac{\theta_{ap}}{\psi_c} = -\frac{\theta_{ap}H}{\alpha E_1(E_1 - C)}. \quad (50)$$

C. Table of the model parameters

Table C. Definition of the parameters of the model

Parameters	Description
Λ_1	Number of newborns and net migrations in S_1
Λ_2	Number of newborns and net migrations in S_2
μ	Natural death rate
ρ_{12}	Moving rate from S_1 to S_2
ρ_{21}	Moving rate from S_2 to S_1
ψ	Percentage reduction of the transmission rate due to control measures
θ_{ap}	Isolation rate of presymptomatic and asymptomatic individuals
θ_s	Isolation rate of symptomatic individuals
β_{1a}	Transmission rate of S_1 individuals by the asymptomatics, I_a
β_{1s}	Transmission rate of S_1 individuals by the symptomatics, I_s
β_{1p}	Transmission rate of S_1 individuals by the presymptomatics, I_p
β_{2a}	Transmission rate of S_2 individuals by the asymptomatics, I_a
β_{2s}	Transmission rate of S_2 individuals by the symptomatics, I_s
β_{2p}	Transmission rate of S_2 individuals by the presymptomatics, I_p
σ_p	Moving rate from presymptomatic to symptomatic compartments
σ_e	Moving rate from exposed, E to presymptomatics class, $I_p \left(\dfrac{1}{\sigma_e}\right.$ is the latent period$\left.\right)$
π	Proportion of Infectious presymptomatic individuals who develop symptoms
γ_a	Asymptomatic recovery $\left(\dfrac{1}{\gamma_a}\right.$ is the infectious period in asymptomatic class$\left.\right)$
γ_h	Recovery rate of detected $\left(\dfrac{1}{\gamma_h}\right.$ is the infectious period in the detected class$\left.\right)$
γ_s	Recovery rate of $\left(\dfrac{1}{\gamma_s}\right.$ is the infectious period in symptomatics class$\left.\right)$
δ_s	Disease-related death rate
δ_h	Disease-related death rate of detected individuals
η	Waning rate of recovery $\left(\dfrac{1}{\eta}\right.$ is the average time of immunity after recovery$\left.\right)$
ω	Proportion of recovered individuals who lost their immunity and moved to S_1