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Transmission dynamics of COVID-19 pandemic with combined effects of relapse, reinfection and environmental contribution: A modeling analysis

Salihu S. Musa, Abdullahi Yusuf, Shi Zhao, Zainab U. Abdullahi, Hammoda Abu-Odah, Farouk Tijjani Saad, Lukman Adamu, Daihai He

Department of Applied Mathematics, Hong Kong Polytechnic University, Hong Kong, China
Department of Mathematics, Kano University of Science and Technology, Wudil, Nigeria
Department of Computer Engineering, Biruni University, Istanbul, Turkey
Department of Mathematics, Science Faculty, Federal University Dutse, Jigawa, Nigeria
J. C. School of Public Health and Primary Care, Chinese University of Hong Kong, Hong Kong, China
Shenzhen Research Institute of Chinese University of Hong Kong, Shenzhen, China
Department of Biological Sciences, Federal University Dutse-Ma, Katsina, Nigeria
School of Nursing, Hong Kong Polytechnic University, Hong Kong, China
Nursing and Health Sciences Department, University College of Applied Sciences, Gaza, Palestine
Department of Mathematics, Yaauf Maitama Sale University, Kano, Nigeria
Department of Mathematical Sciences, University of Maiduguri, Nigeria

ABSTRACT

Reinfection and reactivation of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have recently raised public health pressing concerns in the fight against the current pandemic globally. In this study, we propose a new dynamic model to study the transmission of the coronavirus disease 2019 (COVID-19) pandemic. The model incorporates possible relapse, reinfection and environmental contribution to assess the combined effects on the overall transmission dynamics of SARS-CoV-2. The model’s local asymptotic stability is analyzed qualitatively. We derive the formula for the basic reproduction number ($R_0$) and final size epidemic relation, which are vital epidemiological quantities that are used to reveal disease transmission status and guide control strategies. Furthermore, the model is validated using the COVID-19 reported situations in Saudi Arabia. Moreover, sensitivity analysis is examined by implementing a partial rank correlation coefficient technique to obtain the ultimate rank model parameters to control or mitigate the pandemic effectively. Finally, we employ a standard Euler technique for numerical simulations of the model to elucidate the influence of some crucial parameters on the overall transmission dynamics. Our results highlight that contact rate, hospitalization rate, and reinfection rate are the fundamental parameters that need particular emphasis for the prevention, mitigation and control.

Introduction

Emerging and reemerging infectious disease pathogens remain a monumental problem to global public health and socioeconomic ripening [1,2]. These include coronaviruses and other globally dominated infectious diseases like vector-borne diseases, tuberculosis, sexually transmitted diseases, and meningitis. Coronaviruses are a group of encompassed RNA viruses that primarily circulate or persist among humans (and other mammals) and birds and cause respiratory, neurologic, enteric, and hepatic disease [1]. The coronavirus disease 2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). It emanated from China in December 2019 and was declared a global pandemic on March 11, 2020, by the World Health Organization (WHO) [3].

The COVID-19 pandemic is still in progress and endures in generating disastrous public health and socioeconomic misery worldwide [4–11]. By April 15, 2022, the disease had affected more than 220 countries and territories across all regions of the world, causing >500 million cases and >6 million deaths, respectively [12]. Apart from the SARS-COV-2, six other human coronaviruses are known to infect...
humans, including severe acute respiratory syndrome coronavirus and middle east respiratory syndrome coronavirus [4]. Coronaviruses are likely to reemerge periodically/seasonally in human population owing to recurring cross-species infections and cyclic spillover events [1]. This is plausibly due to the high prevalence and wide geo-distribution of coronaviruses, the enormous heterogeneity of the genetic and persistent recombination of genomes, and the increased human-to-animal interface activities [1].

The COVID-19 vaccines [13,14] coupled with nonpharmaceutical intervention (NPI) measures (e.g. social distancing, face mask use, quarantine, isolation, contact tracing, travel restrictions, school and border closures) effectively helps in suppressing the impact of the pandemic, especially in terms of severity and mortality cases [12,15–17].

Since the emergence of the pandemic, several epidemiological (and clinical) investigations have been performed to examine the transmission dynamics of SARS-CoV-2, ranging from the disease’s clinical characteristics [1,5], estimation of reproduction number, exponential growth, patterns of the epidemic curve, and epidemic modeling [6,7,15,16,18–23]. Some studies have explored the COVID-19 serial interval [24–26], while some used fractional modeling approach to analyze the transmission of COVID-19 infection [8,9]. Furthermore, heterogeneity and super-spreading event have also been investigated [27,28]. In addition, the issue of reactivation [29–36], and reinfection [37–43] have also been studied to explore their impact on the overall transmission of SARS-CoV-2.

The scenario of COVID-19 relapse and reinfection has been reported recently [29–35,41–44]. That is, subsequent infection of SARS-CoV-2 by individual after recovery from previous episode of the disease (i.e., a COVID-19 patient can be certified recovered after satisfying the standard discharge criteria) [29,45]. Re-detectable positive (RP) SARS-CoV-2 can be obtained by using a reverse transcriptase-polymerase chain reaction (RT-PCR) test from a COVID-19 patient after recovery from a previous infection. Eventually, reinfection and reactivation of SARS-CoV-2 need hasty action by epidemiologists and public health practitioners to advise policymakers and public health authorities on how to control/alleviate the pandemic effectively. SARS-CoV-2 reactivation seems improbable in mildly infected outpatients, especially those with no risk factors for severe infection. A relapse (which is of no or less infectiousness) observed during the first eight weeks of sickness implies that retest or isolation is unneeded [46]. Reinfection of SARS-CoV-2 is typically defined as clinical recurrence of COVID-19 followed by a positive PCR test in ≥90 days (on average) after commencement of the primary infection; it usually has reduced infectiousness compared to new infection [39,46]. Moreover, after a symptom-free interval and re-positive PCR outcomes, the motley of clinical recurrence of SARS-CoV-2 infection has not been researched vastly and may be rare in outpatients. However, it could pose a severe threat to public health, thus, studying this issue is imperatively needed [39,40].

This paper proposes a new dynamic model for the transmission of the COVID-19 pandemic incorporating the combined effects of reactivation and reinfection, as well as considers environment’s contribution on the overall dynamics of the disease. According to previous studies [35,42,44], reactivation and reinfection significantly impact COVID-19 dynamics and evolution, which needs to be investigated broadly to uncover the strength of the disease severity and infectiousness, which mathematical modeling study has not explored extensively.

Following this introductory Section, the other component of the study is assembled as follows: The proposed model is designed in Section “Model Description”. Theoretical analysis and model fitting results are reported in Section “Analytical Results”. Numerical simulation results are also given in Section “Numerical Results”. Finally, brief conclusions of the study are presented in Section “Discussion and Conclusions”.

Model description

To analyze the transmission of COVID-19 through mathematical modeling, we, first of all, downloaded the time-series COVID-19 situations report for Saudi Arabia from the public domain of the WHO disease surveillance system (dashboard), accessible via https://covid19.who.int/ [12].

We designed a new classical model based on traditional SEIR-typed to analyze the transmission of COVID-19 pandemic with consideration of possible relapse/reactivation and reinfection, which has been reported previously [29–32,41,42,46,47]. Moreover, our model considers two different modes of transmission, i.e., human-to-human or direct transmission and environment-to-human or indirect transmission [48]. We did not include the zoonotic (animal-to-human) transmission route due to its less infectiousness, especially with the evolving herd immunity caused due to the COVID-19 vaccine or prior exposure to the disease [49–52].

The total human population given by \( N(t) \) is partitioned into mutually exclusive classes of susceptible \( S(t) \), exposed \( E(t) \), asymptotically infected \( I_a(t) \), symptomatically infected (mild and severe) \( I_s(t) \), hospitalized (mild and severe) \( H(t) \) and recovered \( R(t) \) individuals. The
The proposed model is illustrated in Fig. 1, and the state variables and model parameters are given in Table 1. Note that all the parameters are assumed to be non-negative. The models’ systems are, thus, given by Eq. (1) below.

\[
\begin{align*}
\frac{ds}{dt} &= -\left(\beta(t)\left(\frac{m_i + j + h}{N}\right) + \mu_a\beta(t)\right) \frac{m}{m + k} \xi_s, \\
\frac{de}{dt} &= \left(\beta(t)\left(\frac{m_i + j + h}{N}\right) + \mu_a\beta(t)\right) \frac{m}{m + k} \xi_s \\
&\quad + \chi(t)\left(\frac{m_i + j + h}{N}\right) + \mu_a\beta(t)\right) \frac{m}{m + k} \xi_r. \\
\frac{dh}{dt} &= \delta_1 i + \delta_2 h. \\
\frac{dm}{dt} &= \delta_1 i + \delta_2 h. \\
\frac{dD}{dt} &= \delta_1 i + \delta_2 h.
\end{align*}
\]

Susceptible humans gain SAR-CoV-2 infection following effective community contact with an infected person or via the contaminated environment, which is time-dependent and is given by

\[
\xi(t) = \beta(t) \left(\frac{m_i + j + h}{N}\right) s + \mu_a\beta(t)\right) \frac{m}{m + k},
\]

where \(k\) represents the proportion of SARS-CoV-2 pathogens in the contaminated environment which can cause up to about 50% chance of infection. Infectious individual can contaminate the environment by shedding the virus pathogens at rates \(j_i\) and \(i\) respectively, while pathogens in the environment decay at a per capita rate \(\phi\). One of the novelties of the proposed model over prior studies is that the current model incorporates the combined effect of relapse, reinfection and environmental contribution with time-varying transmission rate to evaluate the overall transmission dynamics of SARS-CoV-2.

Following previous studies \([15,21,48,53,54]\), we consider that the contact rate \(\beta(t)\) is a time-dependent decreasing function (i.e., decrease with respect to time \(t\)), and is given by

\[
\beta(t) = \begin{cases} 
\beta_0, & t < t_0 \\
\beta_1 + (\beta_0 - \beta_1) \cdot [1 - a(t - t_0)], & t \geq t_0
\end{cases}
\]

where \(\beta_0\) is the contact rate at the initial time, \(\beta_1\) is the minimum contact rate under the current control strategies, and \(a\) represents the NPI compliance (which provides a measure of public health intervention improvement). For convenience, we normalize the parameter \(a\) to the interval \([0, 1]\), that is 0 \(\leq a \leq 1\), which has the following properties:

- \(a = 0\) representing total non-compliance of the NPI measures by the general public. This further shows that \(\beta(t)\) is the target control parameter that provides a measure of public health intervention improvement and should be reduced as much as possible;
- \(a = 1\) representing total compliance of NPI measures by the general public that is \(a = 100\%\), which is highly unlikely. This further implies that \(a = 1\) is equivalent to \(\beta(t) = \beta_1\), which indicates 100% of NPI measures, leading to minimal contact and thus minimal transmission rate.

For mathematical convenience, we set \(\beta(t) = \beta_0\) in most of results.
Basic reproduction number

The prevalence of SARS-CoV-2 can be evaluated using a threshold quantity, also known as the basic reproduction number, $R_0$, which is obtained following the next-generation matrix technique (see eqn (5)). It constitutes the number of secondary COVID cases that a typical primary case would cause (i.e., by an infected person or through contact with a pathogen-infested material) during the infectious period in a wholly susceptible population [54–61].

Thus, the associated next-generation matrices, $F_i(i = 1, 2)$ representing the new infection terms and the transmission terms, respectively, and are given by

$$F_1 = \begin{bmatrix}
0 & \beta_1 \mu_a \left( \frac{\phi}{N_0} \right) & \beta_0 \left( \frac{\phi}{N_0} \right) & \beta_0 \mu_h \left( \frac{\phi}{N_0} \right) & \beta_0 \mu_h \left( \frac{\phi}{N_0} \right) \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
\end{bmatrix},$$

$$F_2 = \begin{bmatrix}
\sigma & 0 & 0 & 0 & 0 \\
-\beta \sigma & q_1 & 0 & 0 & 0 \\
-\omega \delta & q_2 & 0 & 0 & 0 \\
0 & -\sigma - q_1 \omega & \delta & 0 & 0 \\
0 & -q_1 \omega & \delta & \phi & 0 \\
\end{bmatrix}$$

with $q_1 = \psi_1 + \omega$, $q_2 = \psi_1 + \delta_1 + \tau$, $q_3 = \psi_1 + \delta_2$.

Thus, the basic (or control) reproduction number, $R_0$, are given by

$$R_0 = \rho^*(F_1F_2^{-1}) = R_1 + R_2,$$

where $\rho^*$ (spectral radius) is essentially the maximum eigenvalues of the next generation matrix, $F_1F_2^{-1}$, and $R_1$ and $R_2$ are respectively given by

$$R_1 = \left( \frac{\beta_0 \left( \frac{\phi}{N_0} \right) \left( q_1 + \sigma \right) \omega \mu_h \left( \frac{\phi}{N_0} \right) + \mu_h \left( \frac{\phi}{N_0} \right) \left( \frac{\phi}{N_0} \right) q_2 \omega \mu_h \left( \frac{\phi}{N_0} \right)}{q_1 q_2 \alpha} \right) \beta_0,$$

$$R_2 = \frac{\beta_0 \mu_h \left( \frac{\phi}{N_0} \right) q_1 \left( \tau \delta + \alpha q_2 \right) \omega \mu_h \left( \frac{\phi}{N_0} \right) + \mu_h \left( \frac{\phi}{N_0} \right) \left( \frac{\phi}{N_0} \right) q_2 \omega \mu_h \left( \frac{\phi}{N_0} \right)}{q_1 q_2 \alpha \phi}.$$

The quantities $R_1$ and $R_2$ given in (6) measure the contributions from human-to-human (via respiratory route) and environment-to-human (via the contaminated environment), respectively, to the overall infection rate at time $t$. Thus, the result below is in line with the results of Theorem 2 of [56].

Theorem 1. The DFE, $Y^0$, of the model (1), is locally-asymptotically stable (LAS) inside the biologically-feasible region, $\Omega$, if $R_0 < 1$, and unstable if $R_0 > 1$.

The basic reproduction number ($R_0$) is one of the most crucial epidemiological quantities that guide the control and prevention of emerging infectious diseases. Epidemiologically, the result established in Theorem 1 above insinuates that $R_0 < 1$ would not bring a large outbreak of COVID-19. Whereas for $R_0 > 1$, a severe outbreak could happen. Furthermore, the need for making $R_0 < 1$ for model (2) is only adequate but not mandatory for COVID-19 mitigation efforts. Hence SARS-COV-2 can be eradicated with time when $R_0 < 1$ and spreads when $R_0 > 1$. To achieve this, there is a need to monitor the contribution of human-to-human and environment-to-human transmission of COVID-19 in order to enhance mitigation processes. Therefore, strict containment measures are also necessary to prevent further transmission.

Final size epidemic relation

In this subsection, we obtained the final size epidemic relation for the COVID-19 pandemic by adopting previous technique [15,48,62]. With the expression of $R_0$ in Eq. (5), we consider constant contact rates, i.e., $\beta = \beta_0$ and $\beta = \beta_1$. The result to be computed here helps estimate the severity of the outbreak with respect to its final size relations used previously [62] and was recently adopted for COVID-19 models [15,48].

Theorem 2. Suppose $N_\infty := \lim_{t \to \infty} N(t)$ and $S_\infty := s^0$. Let $x$ and $h$ represent respectively the column vector $x := (e, i_e, i_s, h, m)^T$ and the row vector $h := (0, \mu_e, 1, \mu_s, \mu_m N_\infty)$. Thus, we have:

1. Consider the functions $x(t), S(t)$ and $N(t)$ which satisfy the following properties

$$x_\infty := \lim_{t \to \infty} x(t) = 0 \quad \text{and} \quad \lim_{t \to \infty} S(t) = S_\infty > 0,$$

so that $0 < S_\infty \leq N_\infty < N_0$, where $S_\infty$ denote the final size epidemic to be computed.

2. The final epidemic size relation is given by

$$\ln \frac{S_\infty}{N_\infty} \geq R_0 \left( 1 - \frac{S_\infty}{S_0} \right) + \beta \left( \frac{1}{q_1 q_2} N_\infty \left( \frac{\phi}{N_0} + \frac{\phi}{N_0} \right) \right) \left( x(0) \right).$$

To obtain the final epidemic size given in (8), we assumed some initial conditions to be zero, i.e., $e(0) = i_e(0) = h(0) = m(0) = 0$ and $i_s(0) > 0$ [15,62]. So that, by determining the matrix $V^{-1}$, we can simply obtain the following lower bound for the final epidemic size relation (8):

$$\ln \frac{S_\infty}{N_\infty} \geq R_0 \left( 1 - \frac{S_\infty}{S_0} \right) + \beta \left( \frac{1}{q_1 q_2} \left( \frac{\phi}{N_0} + \frac{\phi}{N_0} \right) \right) \left( x(0) \right).$$

The term $\alpha := 1 - \frac{S_\infty}{S_0}$ represent the “infection attack rate”, which determines the proportion of the population severely infected by susceptible individuals, $S_\infty$, who scarps the infection during the outbreak [63]. The severity of the epidemic largely depends on the attack rate, which significantly increases the cumulative number of disease cases ($S_\infty - S_0$). Following [64], Theorem 2 indicates that, irrespective of the value of $R_0$, the epidemic will die out, not because of the pandemic fatigue only, but also due to the effectiveness of the interventions strategies.

Numerical results

Model fitting

In this subsection, we adopted similar approach as in [23,65,66] for model fitting/validation processes. We fitted the proposed model to the SARS-CoV-2 situations report for Saudi Arabia for the data from December 20, 2021, to March 20, 2022 (third wave), obtained from the public domain of the WHO dashboard for COVID-19 situations reports [12] (see Fig. 2). We employed chi-square distribution and estimated the following parameters: $\beta_0 = 1.026643$, $\gamma = 0.06297961$, $\gamma_1 = 0.5384781$, and $\gamma_2 = 0.1836414$. The initial conditions used for the fitting processes are: $s(0) = 15.000,000$; $e(0) = 30,000$; $i_e(0) = 104$; $i_s(0) = 80$; $h(0) = 45$; $r(0) = 0$; and $m(0) = 2000$. Using the estimated parameters and other parameters given in the Table 2, we computed the $R_0$ given in Eq. (5) as 3.672442. The estimation of the $R_0$ in the current study is consistent with previous results (see, for instance, [67] and the references therein).

Numerical simulations

In this part, numerical simulations were carried out to explain the theoretical results given in the previous sections as well as to gain deeper insight into the COVID-19 dynamic transmission behavior. To obtain numerical solutions to the proposed model, we employed the
consideration that must be examined for their growing or lowering are several significant factors for the chosen COVID-19 model under environment) as can be seen in Figs. 3 and 4. In addition, there $\nu$ (recovered individuals), and $\epsilon$ (exposed individuals), state variables of the model (1) namely $\nu$, $\epsilon$, $\sigma$, $\epsilon\nu$, $\beta$, $\delta$, and $\sigma\epsilon\nu$.

Fitting results of the COVID-19 model (1) using the reported data in Saudi Arabia from December 20, 2021, to March 20, 2022 (third wave). In each panel, the sand color dots denote the observed COVID-19 cases, and the black lines represent the model prediction to the reported data. The result in the left panel shows the cumulative number of COVID-19 instances, and the result in the right panel shows the daily reported COVID-19 situations in Saudi Arabia. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 2

| Parameter | Value (Range) | Units/Remarks | Sources |
|-----------|--------------|---------------|---------|
| $\beta$  | 1.20266 (0.5–1.5) | day$^{-1}$ | Fitted |
| $\beta_1$ | 0.166 (0.002–0.3) | day$^{-1}$ | [48] |
| $\sigma$ | 0.005 (0–0.5) | day$^{-1}$ | [48] |
| $\nu$    | 0.5 (0–1) | day$^{-1}$ | [15,68] |
| $\mu$    | 0.45 (0–1) | day$^{-1}$ | [15,68] |
| $\mu_n$  | 2 × 10$^6$ (0–0.33) | (day/person$^{-1}$) | [48] |
| $k$      | 10$^{4}$ – 10$^{6}$ | per cells per ml | Estimated by [69,70] |
| $\alpha$ | 0.143 (0.05–0.275) | day$^{-1}$ | [71,72] |
| $\theta$ | 0.96834 (0–1) | day$^{-1}$ | [15,72] |
| $\tau$   | 0.13266 (0.001–0.5) | day$^{-1}$ | [22,72] |
| $\delta_1$ | 0.015 (0.01–0.05) | day$^{-1}$ | [22,73] |
| $\delta_2$ | 0.025 (0.01–0.05) | day$^{-1}$ | [22,73] |
| $\psi_1$ | 0.1 (1/14–1/5) | day$^{-1}$ | [15,72] |
| $\psi_2$ | 0.185 (1/30–1/3) | day$^{-1}$ | [15,72,74] |
| $\psi_3$ | 0.11624 (0–1) | day$^{-1}$ | [22,72] |
| $a_1$    | 0.002 (0–0.5) | per virus per person | [48] |
| $a_2$    | 0.002 (0–0.5) | per virus per person | [48] |
| $a_3$    | 0.001 (0–0.5) | per virus per person | [48] |
| $\phi$  | 0.85 (0–1) | day$^{-1}$ | [48] |
| $\zeta$ | 0.063 (0.001–1) | day$^{-1}$ | Fitted |
| $\epsilon_1$ | 0.5385 (0–1) | day$^{-1}$ | Fitted |
| $\epsilon_2$ | 0.1366 (0–1) | day$^{-1}$ | Fitted |
| $\omega$ | 0.85 (0.01–0.99) | day$^{-1}$ | [48,68] |

Euler framework to find the graphical results using parameter values from Table 2 [8–11,75–77]. The procedure is as follows: Suppose the initial condition, which is well-posed, is given below.

$$\frac{dy}{dt} = f(t, y) \text{ and } y(a) = \chi, \quad a \leq t \leq b, \quad (10)$$

a sequence of approximation point $(t, y) \approx (t, y(t))$ is formed by Euler method to exact solutions of ordinary differential equation by $t_{i+1} = t_i + h$ and $y_{i+1} = y_i + hf(t_i, y_i), i = 0, 1, \ldots, n - 1$, and $t_0 = a, y_0 = \chi, h = \frac{b - a}{n}$.

We analyzed the respective numerical dynamic of each of the state variables of the model (1) namely $s$ (Susceptible individuals), $e$ (exposed individuals), $i_0$ (asymptotically infected individuals), $i_1$ (symptomatically infected individuals), $h$ (hospitalized individuals), $r$ (recovered individuals), and $m$ (infectious item on the contaminated environment) as can be seen in Figs. 3 and 4. In addition, there are several significant factors for the chosen COVID-19 model under consideration that must be examined for their growing or lowering values. In this regard, we have picked certain parameters due to their epidemiological impact on transmission such as $\beta, \sigma, \epsilon, \nu, \delta_2$. In Fig. 5(a) and (b), we show that raising the value of $\beta$ would slightly cause the infection to expand significantly, but increasing the rate of progression rate $\sigma$ leads the exposed population to rise immensely. Such manners exemplify the pandemic’s theoretical observations. For increasing and decreasing values of reactivation rates, Fig. 6(a) and (b), a slight increase or decrease in the disease’s reactivation rate from the asymptotically and symptomatically infection’s stage propels infectious individuals to the top, which is consistent with real phenomenon observed experimentally for the ongoing COVID-19 pandemic. Another critical parameter is the COVID-19 induced mortality rate, which was found to be changed with various proper values to evaluate how the model’s behavior changes; this is depicted in Fig. 7.

Sensitivity analysis

Following previous studies [55,78], we employed the partial rank correlation coefficient (PRCC) to investigate sensitivity analysis. The PRCC of the $R_0$ and the infection attack rate of the system (1) are evaluated. The sensitivity analysis results depicted in Fig. 8 reveal top-ranked parameter to be accentuated in controlling the SARS-CoV-2 pandemic is the contact/transmission rate ($\beta_0$), per capita rate of susceptible individuals who mingle with the environment per day ($\sigma_0$), and the proportion of SARS-CoV-2 pathogens present in the environment ($k$). Other top-ranked parameters apart from the ones mentioned include the rate of decay of the virus pathogen from the environment ($\phi$) and environmental contamination rates ($a_n (n = 1, 2, 3)$).

Discussion and conclusions

Since COVID-19 pandemic started in early 2020 [3,79], non-pharmaceutical interventions (NPIs) measures have been playing a pivotal role in halting the number of cases and deaths [80,81], despite the increasing rate of vaccination worldwide [13,14]. This paper proposed an SEIR-based epidemic model that incorporated direct and indirect transmission as the potential transmission pathways of COVID-19. The model also included possible relapse and reinfection of SARS-CoV-2 from a recovered patient who satisfied all the standard discharge procedures from the previous episode of the infection [31,42,44].

Some of the key epidemiological findings of the present study are outlined as follows:
Fig. 3. Simulation results of the COVID-19 pandemic model showing the dynamics behavior over the time period. Profiles of the variables $s$, $e$, $i_a$, and $i_s$ are portrayed in (a), (b), (c), and (d), respectively.

(i) The basic reproduction number, $R_0$, of the model was determined (see Eqs. (5) and (6)), which measures the potential transmission for an outbreak. It depends on the transmission coefficient and the average duration of infectiousness during the epidemic period. $R_0 < 1$ and $R_0 > 1$ indicates that the disease will die out and persists, respectively. Thus, the elimination of an infectious disease requires that $R_0$ must be reduced to one either by vaccination or via other intervention strategies, such as public awareness programs [82].

(ii) We analytically computed the final size of epidemic relations for the simplified model (i.e., by considering human-to-human transmission route only) to give an account of the rough epidemic size over the cause of the epidemics period [62,83]. The simplified version of the model was used in computing the final epidemic size relations due to the complexity of the full model and the fact that likely no significant changes could be detected even if the fundamental model was used.

(iii) The proposed model was fitted to the COVID-19 situation reports in Saudi Arabia for the third wave, i.e., for the data from December 20, 2021, to March 20, 2022. Our numerical results show that the fitting results nicely represented the actual situation and were adopted to estimate other crucial parameters of the model that are useful for control and mitigation strategy and could be used to predict future scenarios of the disease outbreak.

(iv) Furthermore, by using the $R_0$ and infection attack rates as response functions, we conducted the sensitivity analysis, which demonstrates the highest PRCCs ranked parameters (i.e., $\beta_0$, $k$, and $\mu_m$), which are relevant to environmental influence on the overall transmission of SARS-CoV-2. Hence, this study determines the parameters that should be targeted for effectual control of the pandemic. Other peak parameters with high ranked PRCCs (but not as high as the most sensitive ones) are the rate of decay of the pathogen from the environment ($\phi$) as well as environmental contamination rates ($a_n(n = 1, 2, 3))$.

(v) Finally, we performed numerical simulations to portray the impact of the model’s parameters on the overall transmission dynamics. In particular, Figs. 3–4 showed the general dynamics behavior of SARS-CoV-2 dynamics in each sub-population and how they affect the overall transmission rate. In Fig. 5, we showed that increasing the value of $\beta$ would slightly increase the epidemic impacts significantly, thereby increasing the transmission rate. We also showed a slight increase or decrease in the disease’s activation rate from the asymptomatically and symptomatically infection stage propels the infectious population to the top; thus, it could not affect the overall transmission dynamics. Similarly, in Eq. (6)(a) and (b), increase in $v_1$ and $v_2$ likely increase the transmission. Moreover, a decrease in $\delta_2$ in Eq. (7) significantly helps suppress the transmission.
Fig. 4. Simulation results of the COVID-19 pandemic model showing the dynamics behavior over the time period. Profiles of the variables $h$, $r$, and $m$ are portrayed in (a), (b), and (c), respectively.

Fig. 5. Dynamic behavior of the proposed model with changing values of $\beta$ and $\sigma$ in (a) and (b), respectively.
In summary, this study designed an SEIR-typed model to qualitatively investigate the transmission dynamics of COVID-19 incorporating the relapse, reinfection and environmental transmission to get more profound knowledge of its transmission potential and provide suggestions for effective control. In addition, we obtained some crucial epidemiological parameters that need more emphasis for mitigation and control of the COVID-19 pandemic. Our results showed that $\beta_0$, $k$, and $\mu_m$ are the key parameters that should be prioritized to control the epidemics. Those parameters could help to maintain disease transmission at a low level with minimal socio-economic distraction and play an essential role, especially in the public health and policy-making sectors, in generating a continuous and imperishable plan to mitigate against the effects of the still ongoing pandemic.

Hitherto, several studies revealed that reactivation or reinfection causes less or no severe infection in vulnerable people; however, health workers and authorities should be prudent in declaring recovery for SARS-CoV-2 patients [39,40,44,84]. Thus, it is imperative to monitor recovered patients to prevent subsequent transmission.

The current study has some limitations. The proposed model is a time-dependent model, which makes it non-autonomous. Thus, analyzing it theoretically, particularly if more vital dynamics are incorporated (such as seasonality factors), will be challenging, requiring the design of a new mathematical theory and methodology. Further, characterizing bifurcation types for non-autonomous models is essential and will be considered in future work by extending the current study. Furthermore, the current study can be extended by employing an artificial neural network scheme to solve the model of the nonlinear dynamics for COVID-19 or by using a stochastic algorithm framework as proposed by previous studies [85–89], which would significantly help in prevention and mitigation strategies of emerging infectious diseases.
Salihu S. Musa: Conceptualization, Formal analysis, Writing – original draft, Writing – review & editing. Abdullahi Yusuf: Formal analysis, Writing – original draft, Writing – review & editing.
Zainab U. Abdullahi: Writing – original draft, Revision. Hammoda Abu-Odah: Writing – original draft, Writing – review & editing. Farouk Tijjani Saad: Writing – original draft, Writing – review & editing. Lukman Adamu: Writing – original draft, Writing – review & editing. Daihai He: Conceptualization, Formal analysis, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Availability of data and materials

The COVID-19 reported data used in this work were freely obtained from the public domains of the WHO dashboard via https://covid19.who.int/.

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Ethics approval and consent to participate

Since no individual data was used, neither ethical approval nor personnel consent was needed.

Fig. 8. The partial rank correlation coefficients (PRCC) of basic reproduction number ($R_0$) and infection attack rate versus the model’s parameters.

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