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Behavioral response of population on transmissibility and saturation incidence of deadly pandemic through fractional order dynamical system

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A R T I C L E   I N F O

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- Fractional RK4
- Stability

A B S T R A C T

The world entered in another wave of the SARS-CoV-2 due to non-compliance of standard operating procedures appropriately, initiated by respective governments. Apparently, measures like using face masks and social distancing were not observed by populace that ultimately worsens the situation. The behavioral response of the population induces a change in the dynamical outcomes of the pandemic, which is documented in this paper for all intents and purposes. The innovative perception is executed through a compartmental model with the incorporation of fractional calculus and saturation incident rate. In the first instance, the epidemiological model is designed with proportional fractional definition considering the compartmental individuals of susceptible, social distancing, exposed, quarantined, infected, isolated and recovered populations. By virtue of proportional fractional derivative, effective dynamical outcomes of equilibrium states and basic reproduction number are successfully elaborated with memory effect. The expansion of this derivative greatly simplifies the model to integer order while remaining in the fractional context. Subsequently, the memory effects on the asymptotic profiles are demonstrated through various graphical plots and tabulated values. In addition, the inclusion of saturation incident rate further explains the transmissibility of infection for different behavior of susceptible individuals. Mathematically, the results are also validated through comparative analysis of values with the solutions attained from fractional fourth order Runge-Kutta method (FRK4).

Introduction

Healthcare researchers believe on mathematical simulations to make prompt measures and policies, and save the community from deadly viral infections. These models use epidemic systems through the provided data from clinicians and health workers for surveillance of the transmissibility of a disease. Different mathematical functions are being incorporated in epidemic models in order to design a model that considerably articulates the realistic behavior of a disease. Literature depicts many systems of epidemic models, which propose different rates and functions to prevent and control the spread of the infectious disease [1–3]. An incredible number of models have been formulated, investigated and functional, which improve our understanding and extrapolative skill about a diversity of transmissible disease qualitatively and quantitatively [4–7].

Calculus of non-integer orders have an extended and thrilling history beginning with communication by two scientist Leibniz and de L’Hospital. Equations with fractional order derivatives are controlling tool for unfolding processes with non-locality and memory. Some new definitions of fractional derivatives have a conventional of unusual things that contains violations of Leibniz rule for the derivative of product function and chain rule that define the composition of function. Some of these new operators and results are just formal non-integer calculus of the known conventional theories, often without any justification and motivation. There are different types of non-integer derivatives that are suggested by Liouville, Caputo and Reisz, that described the procedures of non-locality and memory [8]. Moreover, some of the definitions provide useful expansion that reduces the computational complexity of fractional order differential equations. For instance, Laplace expansion of Caputo fractional derivative [9], tempered fractional derivative [10] and proportional fractional derivative [11] etc., have added a great contribution in fractional calculus. The operators of fractional calculus...
have been broadly functional in many fields such as, engineering and sciences [11–14].

SARS-CoV-2, familiarized as coronavirus disease (COVID’19), being a communicable disease is a detriment to human-life in all respect. Carelessness in following the precautionary measures have caused the world to face another wave of this pandemic with more severity. People infected with the COVID’19 infection involve mild to moderate respiratory sickness and recuperate without needful special treatment in some cases, whereas some are at death’s door. As mathematical demonstrations play important role in the area of epidemiology, many models have been structured in this regard [15–17]. Keeping in view the behavioral change and crowding effect of populace that became one of the major reason of another wave of COVID’19, we assimilate the Holling type II function [18] in dynamics of susceptible and infective population. The incorporation of this function in the model is epidemiologically effective to discuss this novel scenario of COVID’19 transmission. It elaborates the overall effects that occur due to the behavioral change of susceptible from the crowding effect of the infective individuals, vice versa. In addition to this property, it prevents the unboundedness of the contact rate by choosing suitable parameters [19–24]. Therefore, the representation of incidence rate using Holling type II function is more reasonable than the bilinear incidence rate. Moreover, fractional order derivative is also considered to evaluate the historical effects in continuation with the future dynamics, innovatively. Here, we utilized proportional fractional order derivative to greatly simplify the fractional order system without losing the originality of the model [17,21]. The governing epidemiological model comprises of system of seven nonlinear fractional order differential equations for the dynamics among susceptible (S), social distancing (P), exposed (E), quarantined (Q), infected (I), isolated (M) and recovered (R) populations. The dynamical effect on the system due to the existence of social distancing population is an epidemiological modernity. Growth of susceptible and individuals with social distancing are contemplated with a threshold through the perception of logistic growth function [23–26], whereas growth in other compartments is due to the transmission of infection. This function intends to explain the per capita growth of population by limited resources in the environment. The pictorial representation in Fig. 1 defines the transmission of the deadly pandemic around the individuals in the considered compartments. Moreover, the validity of the attained numerical solutions are also further verified through a comparative study. This study is carried out with help of fractional fourth order Runge-Kutta method [27], which is a well-known numerical technique that can significantly generate numerical solutions with a choice of adequately small step size of iterations in fractional environment. Sequentially, the remaining sections of the paper comprise of; the detail deliberation of modelling in section 2, findings of basic reproduction number and equilibrium states are demonstrated in section 3, and the numerical discussions and conclusion are established in section 4 and 5, accordingly.

**Epidemiological modelling**

The epidemic models have been undergone with different mathematical modifications to design more realistic models. Since these equations deal with the study of the diseases on human population, therefore the basic biological postulations are retained. Here, we adapted the model in which a host population (N) is divided into seven compartments categorized as: susceptible (S), individuals following social distancing (P), individuals exposed to infected (E), exposed individuals quarantined (Q), infected individuals (I), infected individuals isolated (M) and individuals recovered from quarantine and isolation (R), per day. So the total population of the considered region can be expressed as,

![Fig. 1. Pictorial diagram of transmission of deadly infectious disease.](image-url)
\[ N(t) = S(t) + P(t) + E(t) + Q(t) + I(t) + M(t) + R(t) \] (1)

All individuals are equally possible to catch infection except those who are following social distancing even in susceptible. The growth of susceptible is defined with a maximum surviving capability in the available resources. The transmission process of disease is considered with the Holling-type II function, which describes the infection transmission rate together with the inhibitory effect. This term commonly known as saturation incidence function in epidemiology that intends to define the variation in infection force due to the crowding effect of infective. The exposed to infection with mild symptoms might get recovered after completing incubation period in quarantine. Beside, some may get infected that can transmit the disease to the others and either get recovered after an isolation period or die due to the infection. Moreover, the individuals once recovered are considered to gain immunity that they cannot be infected again and also if they join the individuals who are following social distancing measures. Different rate of treatments for exposed, quarantine and infected individuals is availed during the respective degree of infectiousness. Linked with the historical variations in the dynamical changes, the future interpretations is contemplated through the memory effect of proportional fractional order derivative. Based on aforementioned assumptions, the fractional order system of seven equations is thus designed as,

\[ \frac{p^\alpha D^\alpha_t S(t)}{S(t)} = r_S S(t) \left(1 - \frac{S(t)}{K_S}\right) - \frac{\beta S(t) I(t)}{1 + aI(t)} - (b + d_S) S(t) \]

\[ \frac{p^\alpha D^\alpha_t P(t)}{P(t)} = r_P P(t) \left(1 - \frac{P(t)}{K_P}\right) + \xi R(t) + b S(t) - d_P P(t) \]

\[ \frac{p^\alpha D^\alpha_t E(t)}{E(t)} = \frac{\beta S(t) I(t)}{1 + aI(t)} - (\gamma + d_E) E(t) \]

\[ \frac{p^\alpha D^\alpha_t Q(t)}{Q(t)} = \gamma E(t) - (\eta + \psi_D + d_Q) Q(t) \]

\[ \frac{p^\alpha D^\alpha_t I(t)}{I(t)} = \eta Q(t) - (\sigma + d_I) I(t) \]

\[ \frac{p^\alpha D^\alpha_t M(t)}{M(t)} = \alpha I(t) - (\psi_M + \rho) M(t) \]

\[ \frac{p^\alpha D^\alpha_t R(t)}{R(t)} = \psi_D Q(t) + \psi_M M(t) - (\xi + d_R) R(t) \]

With initial condition,
\[ S(0) = \delta_1, \quad P(0) = \delta_2, \quad E(0) = \delta_3, \quad Q(0) = \delta_4, \quad I(0) = \delta_5, \quad M(0) = \delta_6, \quad R(0) = \delta_7 \] (3)

where, \( \delta_i \geq 0 \) for \( i = 1, 2, ..., 7 \). All the variables and parameters presented in system (2) are defined in the Table 1. Special case of proportional fractional derivative defined in [11] is a lucrative operator that greatly simplifies the fractional order differential equations into integer order. The proportional fractional derivative of any continuous function \( h(t) \) expands to,

\[ \frac{p^\alpha D^\alpha_t h(t)}{h(t)} = g_0(\alpha, t) \dot{h}(t) + g_1(\alpha, t) h(t) \] (4)

where, \( g_0(\alpha, t) \neq 0 \) for \( \alpha \in (0,1] \), with \( \lim_{\alpha \to 0} g_0(\alpha, t) = 0 \) and \( \lim_{\alpha \to 1} g_0(\alpha, t) = 1 \). Moreover, \( g_1(\alpha, t) \neq 0 \) for \( \alpha \in (0,1] \), with \( \lim_{\alpha \to 0} g_1(\alpha, t) = 1 \) and \( \lim_{\alpha \to 1} g_1(\alpha, t) = 0 \).

**Table 1**

| Details of symbols of the SPEQIMR model (6). |
|---------------------------------------------|
| Symbols | Descriptions/Units | (Individuals/(individuals × day))(population in ‘000s & time in days) |
|---------|-------------------|------------------------------------------------------------------|
| \( r_S \) | Intrinsic growth rate of susceptible |
| \( k_S \) | Surviving capacity of susceptible |
| \( r_P \) | Intrinsic growth rate of social distancing individuals |
| \( k_P \) | Surviving capacity of social distancing individuals |
| \( b \) | Rate of susceptible following social distancing |
| \( \alpha \) | Saturation factor |
| \( \beta \) | Infection force |
| \( \eta \) | Rate of quarantine to infection |
| \( \gamma \) | Rate of exposed quarantined |
| \( \sigma \) | Rate of infected isolated |
| \( \rho \) | Death rate due to disease |
| \( \psi_D \) | Recovery rate after isolation |
| \( d_R \) | Natural death rate |

Let, \( g_0(\alpha, t) = \alpha \) a nd \( g_1(\alpha, t) = 1 - \alpha \), so Eq. (4) becomes

\[ \frac{p^\alpha D^\alpha_t h(t)}{h(t)} = \dot{h}(t) + (1 - \alpha) h(t) \] (5)

Thus, using the expansion (5) in model (2), we get the expression as,

\[ S(t) = \frac{1}{a} \left[ r_S S(t) \left(1 - \frac{S(t)}{K_S}\right) - \frac{\beta S(t) I(t)}{1 + aI(t)} - (b + d_S) S(t) - (1 - \alpha) S(t) \right] \]

\[ P(t) = \frac{1}{a} \left[ r_P P(t) \left(1 - \frac{P(t)}{K_P}\right) + \xi R(t) + b S(t) - d_P P(t) - (1 - \alpha) P(t) \right] \]

\[ E(t) = \frac{1}{a} \left[ \frac{\beta S(t) I(t)}{1 + aI(t)} - (\gamma + d_E) E(t) - (1 - \alpha) E(t) \right] \]

\[ Q(t) = \frac{1}{a} \left[ \gamma E(t) - (\eta + \psi_D + d_Q) Q(t) - (1 - \alpha) Q(t) \right] \]

\[ I(t) = \frac{1}{a} \left[ \eta Q(t) - (\sigma + d_I) I(t) - (1 - \alpha) I(t) \right] \]

\[ M(t) = \frac{1}{a} \left[ \alpha I(t) - (\psi_M + \rho) M(t) - (1 - \alpha) M(t) \right] \]

\[ R(t) = \frac{1}{a} \left[ \psi_D Q(t) + \psi_M M(t) - (\xi + d_R) R(t) - (1 - \alpha) R(t) \right] \]

With initial conditions same as defined in Eq. (3). Some essential theorems for the system (6), are discussed as follows:

**Theorem 1.** Let \( \Phi \in \mathbb{R}_+^7 \) be the set of all possible solutions of the system (6), such that

\[ \Phi = \left\{(S, P, E, Q, I, M, R) \in \mathbb{R}_+^7 ; N \in \left[ \frac{r_S}{K_S} + \frac{r_P}{K_P} \right] \right\} \] (7)

then it is uniformly bounded subset of \( \mathbb{R}_+^7 \).

**Proof.** Since,

\[ \frac{p^\alpha D^\alpha_t N(t)}{N(t)} = \frac{p^\alpha D^\alpha_t S(t)}{S(t)} + \frac{p^\alpha D^\alpha_t P(t)}{P(t)} + \frac{p^\alpha D^\alpha_t E(t)}{E(t)} + \frac{p^\alpha D^\alpha_t Q(t)}{Q(t)} + \frac{p^\alpha D^\alpha_t I(t)}{I(t)} + \frac{p^\alpha D^\alpha_t M(t)}{M(t)} + \frac{p^\alpha D^\alpha_t R(t)}{R(t)} \]

Expanding proportional fractional derivative, we get the expression as:
\[ N(t) = \frac{1}{a} \left( \dot{S}(t) + \dot{P}(t) + \dot{E}(t) + \dot{Q}(t) + \dot{I}(t) + \dot{M}(t) + \dot{R}(t) - (1 - \alpha)N(t) \right) \] (8)

using system (6) with some manipulation, Eq. (8) becomes,

\[ \dot{N}(t) = r_p G(t) - r_p P(t) - d_N N(t) \] (9)

for \(0 < \alpha \leq 1\), where \(d_N\) represent the total proportion of natural deaths i.e.,

\[ d_N N(t) = d_S S(t) + d_{SE} E(t) + d_{QI} Q(t) + d_P P(t) + d_R R(t) \] (10)

As, \(0 < \frac{r_p}{K_S} \leq 1\) and \(0 < \frac{r_p}{K_P} \leq 1\), the inequality (9) reduces to

\[ \dot{N}(t) \leq \frac{r_p}{K_S} - d_N N(t) \] (11)

After some manipulation, as \(t \to \infty\), we attain the boundedness relation as,

\[ N(t) \leq \frac{1}{d_N} \left( \frac{r_p}{K_S} + r_p \right) \] (12)

\[
B_1 = \begin{bmatrix}
    r_p - b - d_S & 0 & 0 & \ldots & \ldots & \ldots & 0 \\
    b & r_p - d_P & 0 & \ldots & \ldots & \ldots & \xi \\
    0 & 0 & -d_I - \gamma & \ldots & \ldots & \ldots & 0 \\
    \vdots & \vdots & \vdots & \ddots & \ddots & \ddots & \vdots \\
    0 & 0 & \gamma & -\gamma_0 - \eta & \ldots & \ldots & 0 \\
    \vdots & \vdots & \vdots & \vdots & \ddots & \ddots & \vdots \\
    0 & 0 & \ldots & \ldots & \ldots & -\rho - \psi_M & 0 \\
    0 & 0 & \ldots & \ldots & \ldots & \ldots & -d_R - \xi \\
\end{bmatrix}_{7 \times 7}
\]

Theorem 2. Let \(\Lambda(G(t)) : \mathbb{R}^7_{\text{in}} \to \mathbb{R}^7_{\text{out}}\) be the matrix of right hand side of system (6) with \(\Lambda(G(t))\) and \(\frac{\partial \Lambda(G(t))}{\partial G(t)}\) being continuous, such that

\[\|\Lambda(G(t))\| = \left\| \frac{1}{a} (B_1 B_2 G(t) + S(t) B_2 G(t) + P(t) B_2 G(t) + I(t) B_2 G(t) + (\alpha - 1) G(t)) \right\| \leq \frac{1}{a} \left( \|B_1\| + \|B_2\| + \|B_3\| + \|B_4\| + (\alpha - 1) \right) \|G(t)\| \]

\[\|\Lambda(G(t))\| \leq \frac{\omega}{|a|} |G(t)|, \forall G(t) \in \mathbb{R}^7_{\text{in}} \text{ and } 0 < a \leq 1 \] (13)

Then, for given initial conditions satisfying (2), there exists a unique, bounded and non-negative solution of the system (6) for all \(t \in [0, \infty)\).

Proof. Proving the uniqueness of the solution, the system (6) can be represented as:

\[ \dot{G}(t) = \Lambda(G(t)) \]

where,

\[ G(t) = [S(t) \ P(t) \ E(t) \ Q(t) \ I(t) \ M(t) \ R(t)]^T \] (14)

and

\[
\begin{bmatrix}
    r_p S \left( \frac{1}{K_S} - \frac{\beta SI}{1 + \alpha} \right) - (b + d_S) S - (1 - \alpha) S \\
    r_p P \left( \frac{1}{K_P} - \frac{\beta SI}{1 + \alpha} \right) + \xi R + bS - d_P P - (1 - \alpha) P \\
\end{bmatrix}
\]

\[ \Lambda(G(t)) = \frac{1}{a} \]

\[ B_2 = \left\lfloor \frac{r_p}{K_S} \ 0 \right\rfloor_{1 \times 7} \]

\[ B_3 = \left\lfloor r_p \ K_P \ 0 \right\rfloor_{1 \times 7} \]

\[ B_4 = \left\lfloor -\beta \ 0 \ \beta \ 0 \right\rfloor_{1 \times 7} \]

Taking norm on both side of Eq. (16),

\[ \|\Lambda(G(t))\| \leq \frac{\omega}{|a|} |G(t)|, \forall G(t) \in \mathbb{R}^7_{\text{in}} \text{ and } 0 < a \leq 1 \] (13)

Let \(\omega = \|B_1\| + \|B_2\| + \|B_3\| + \|B_4\|\), so for \(0 < a \leq 1\) the final statement is articulated as,

\[ \|\Lambda(G(t))\| \leq \frac{\omega}{|a|} |G(t)|, \forall G(t) \in \mathbb{R}^7_{\text{in}} \] (13)

Furthermore, using the positivity of initial conditions (2) and the first differential equation of system (6), to prove the non-negativity of the solutions as:

\[
\dot{S}(t) = \frac{1}{a} \left( r_p S \left( \frac{1}{K_S} - \frac{\beta SI}{1 + \alpha} \right) - bS - \frac{\beta SI}{1 + \alpha} \cdot d_S S - (1 - \alpha) S \right) \\
\geq - \frac{1}{a} (b + d_S + (1 - \alpha)) S \\
\]

thus, we get

\[ S(t) \geq S(t_0) e^{- \frac{1}{a} (b + d_S + (1 - \alpha)) t} \] (17)
Since $0 \leq e^{(b_1 + b_2 t)} e^{(1-\omega)} t \leq 1$ for $t > 0$, Eq. (17) yields,

\[ S(t) > 0 \]

Hence, the non-negativity of solution of $S(t)$ is proved. Correspondingly, in the similar manner, it can be concluded that the solutions of remaining equations of system (6) may also be non-negative with positivity of initial conditions.

**Epidemic thresholds**

In this section, the equilibria of the system (6) along with the basic reproduction number and its dynamical properties are discussed in particular. Let the constant solutions of system (6) be $\chi_0$, $\chi_1$, and $\chi_2$, as trivial, disease-free, and endemic equilibria, respectively. Suppose, $\bar{T}$ be the sub-model of system (6), expressed as:

\[
\frac{d\bar{T}}{dt} = \mathbf{X}(\bar{T}) - X(\bar{T})
\]

(18)

Such that the vector $\bar{T}$ includes the $E(t)$, $Q(t)$, $I(t)$ and $M(t)$ equations only, $\bar{T} = [E(t) \ Q(t) \ I(t) \ M(t)]^T$, $Z(\bar{T})$ defines new infections ingoing in these compartments, $Z(\bar{T}) = \begin{bmatrix} \beta S(t)(1+\alpha d(t)) & \eta \ E(t)(1+\alpha d(t)) & \sigma \ E(t)(1+\alpha d(t)) \\ \gamma \ E(t)(1+\alpha d(t)) & \eta \ Q(t)(1+\alpha d(t)) & \sigma \ Q(t)(1+\alpha d(t)) \end{bmatrix}$. $X(\bar{T})$ is further split down into $X^+ (\bar{T})$ and $X^-(\bar{T})$, containing all other outputs and inputs of these compartments, respectively. Since, no new infected individual enter the $Q(t)$ and $I(t)$ compartments, rather they transit from the $E(t)$ compartment into $Q(t)$ and then $I(t)$ compartments, therefore, the second and third element of $Z(\bar{T})$ are zero. On the other side,

\[
X(\bar{T}) = X^-(\bar{T}) = X^-(\bar{T})
\]

(19)

Taking Jacobian matrix of Eq. (18) at disease free equilibrium, $\chi_1 = (\frac{k_s(-1-b-d_s+r_s+a)}{r_s}), (\frac{k_p(-1-b+\rho + a)}{2r_p}, 0, 0, 0, 0)$ we get,

\[
\mathbf{J} \left[ \frac{d\bar{T}}{dt} \right] = \mathbf{Z} - \mathbf{X}
\]

\[
\begin{bmatrix}
0 & 0 & 0 & 0 & 0 \\
0 & \frac{\beta k_s(-1-b-d_s+r_s+a)}{r_s} & 0 & 0 & 0 \\
0 & 0 & \frac{\eta \ k_p(-1-b+\rho + a)}{2r_p} & 0 & 0 \\
0 & 0 & 0 & \frac{\sigma}{\alpha} & 0 \\
0 & 0 & 0 & 0 & \frac{1+\rho+\psi a}{\alpha}
\end{bmatrix}
\]

Using Eq. (19) and the eigenvalues with the largest magnitude of the next generation matrix $\mathbf{K} = \mathbf{ZX}^{-1}$, the required basic reproduction number is attained as,

\[
R_0 = \frac{k_s(-1-b-d_s+r_s+a)\beta \eta}{r_s \ (1+d_s-a+\gamma)(1+d_i-a+\sigma)(1+d_o-a+\eta+\psi a)}
\]

(20)

where $r_s > 1 - b - d_s + a$.

Furthermore, again constructing the Jacobian matrix $\mathbf{J}$, but for the complete system (6), the corresponding characteristic equation for the eigenvalues $\lambda$ is then expressed as,

\[
\mathbf{Q}(\lambda) = \text{Det}(\lambda I - \mathbf{J}) = 0
\]

(21)

**Theorem 3.** The trivial equilibrium solution, $\chi_0(0, 0, 0, 0, 0, 0) \in \Re^7$, of system (6), is asymptotically unstable, for all and $0 < \alpha \leq 1$.

**Proof.** The eigenvalues of $\mathbf{J}$ at this equilibrium point are obtained as,

\[
\lambda_1 = \frac{-1-d_r+r_p+a}{\alpha} \quad \lambda_2 = \frac{-1-b-d_s+r_s+a}{\alpha} \quad \lambda_3 = \frac{-1-d_s+a-\gamma}{\alpha} \quad \lambda_4 = \frac{-1-d_w+a-\eta-\psi a}{\alpha} \quad \lambda_5 = \frac{-1+a-\rho-\psi a}{\alpha}
\]

Since $r_s > 1 - d_s + a$ and $r_s > 1 - b - d_s + a$, So it is clear that $\lambda_1, \lambda_2 > 0$, for $0 < \alpha \leq 1$. Thus, $\chi_0 \in \Re^7$ is unstable.

**Theorem 4.** Let $\chi_1 \in \Re^7$, be the disease free equilibrium solution of system (6), then it is asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$.

**Proof.** Solving $\mathbf{J}$ for $\chi_1 \in \Re^7$, we get the negative real eigenvalues,

\[
\lambda_1 = \frac{f_r}{\alpha} \quad \lambda_2 = \frac{-1-b-d_s+r_s+a}{\alpha} \quad \lambda_3 = \frac{-1+d_s+a-\gamma}{\alpha} \quad \lambda_4 = \frac{-1+b+\rho+\psi a-\alpha}{\alpha}
\]

where,
\[
 f_0 = \frac{b_k r_5 \left(-4b^2 k_r r_6 + k_r r_5 \left(-1 - d_k + r_k + a\right)^2 + 4b k_r r_5 \left(-1 - d_k + r_k + a\right) \right) \left(1 + d_k - a + \xi\right)^2 \left(1 - \alpha + \rho + \psi_\alpha\right)}{k_r r_5 \left(1 + d_k - a + \xi\right) \left(1 - \alpha + \rho + \psi_\alpha\right)}
\]

with the transcendental equation,

\[
 H_1 = \frac{1}{a G H_{r_5} \beta \gamma} \left( G H \left(4 b G r_5 + a k_r r_5 \left(-1 - d_k + r_k + a\right)^2 \beta \gamma\right) + \left(G(1 - R) + G_1 R_0\right) \right)
\]

where,

\[
 H = (1 + d_k - a + \xi) \left(1 - \alpha + \rho + \psi_\alpha\right)
\]

with the transcendental equation,

\[
 T(\lambda) = \lambda^3 + B_2 \lambda^2 + B_1 \lambda + B_0 (1 - R_0) = 0
\]

where,

\[
 B_0 = \frac{(1 + d_k - a + \gamma)(1 + d_2 - a + \sigma)(1 + d_3 - \alpha + \eta + \psi_\alpha)}{a^2}
\]

\[
 B_1 = \frac{(1 + d_2 - a + \sigma)(1 + d_3 - a + \eta + \psi_\alpha) + (1 + d_3 - a + \gamma)(2 + d_4 + d_5 - 2\alpha + \eta + \sigma + \psi_\alpha)}{a^2}
\]

\[
 B_2 = \frac{3 + d_2 + d_3 + d_4 - 3a + \gamma + \eta + \sigma + \psi_\alpha}{a}
\]

Since, \( f_0 > 0 \), iff

\[
 \left(k_r r_5 \left(-1 - d_k + r_k + a\right)^2 + 4b k_r r_5 \left(-1 - d_k + r_k + a\right) \right) < 4b^2 k_r r_5
\]

and \( r_k > -1 - b - d_k + a \), so it is clear that \( \lambda_i < 0 \), for \( i = 1, 2, 3, 4 \) and for all \( 0 < a \leq 1 \). Moreover, from Routh-Hurwitz condition [28,29] for cubic polynomial, if \( B_2 > 0, B_1 > 0 \) and \( B_0 (1 - R_0) > 0 \), then \( T(\lambda) > 0 \) for all non-negative real eigenvalues. Therefore, it can be clearly seen that \( B_2 > 0, B_1 > 0 \) and \( B_0 > 0 \), the only thing left to be proved is

\[
 T(\lambda) = \lambda^3 + C_3 \lambda^2 + C_1 \lambda + C_0 = 0
\]

where,

\[
 C_0 = \frac{1}{k_r a^2} \left( f_1 \gamma \eta (f_1 - f_2) + f_1 (1 + d_k - a + \gamma)(1 + d_2 - a + \sigma)(1 + d_3 - a + \alpha + \eta + \psi_\alpha) \right)
\]

that \( (1 - R_0) > 0 \). Hence, \( \lambda_1 \in \mathbb{R}_+^0 \) is asymptotically stable if \( R_0 < 1 \). If \( R_0 > 1 \), then \( (1 - R_0) < 0 \) implies \( T(0) < 0 \), which yields Eq. (22) to have a non-negative real part, thus it becomes unstable.

**Theorem 5.** If \( R_0 > 1 \), then the endemic equilibrium \( \chi_2(S', P', E', Q', \Gamma', M', R') \in \mathbb{R}_+^0 \) is asymptotically stable, and unstable if and only if, \( R_0 > 1 \).

**Proof.** Solving \( J \) for the equilibrium point \( \chi_2(S', P', E', Q', \Gamma', M', R') \in \mathbb{R}_+^0 \), we get the negative real eigenvalues,

\[
 \lambda_i = \frac{H_i}{\alpha_i} = -1 + \frac{d_k + \xi - a}{\alpha} - \frac{1 + \rho + \psi_\alpha - a}{\alpha}
\]

\[
 f_i = \frac{(G_1 + G)R_0G_1 - (G(1 - R_0) + G - G_1 R_0)k_r \beta \gamma \eta}{a(G_1 + G)k_r R_0 \beta \gamma \eta}
\]

\[
 = \frac{2G^2}{aR_0 \beta \gamma \eta (G_1 + G)}
\]

\[
 \gamma = a k_r \beta \gamma \eta (-1 - b - d_k + r_k + a) - \frac{2G(1 - R_0) + 2G}{R_0}
\]

Since \( H_1 > 0 \), iff
and \( r_3 > -1 - b - d_6 + \sigma \), so it is clear that \( \lambda_i < 0 \), for \( i = 1, 2, 3 \), and for all \( 0 < \alpha \leq 1 \). From Routh-Hurwitz condition \( [28,29] \) for fourth order polynomial, if \( C_i > 0 \), for \( i = 0, 1, 2, 3 \), then \( T(\lambda) > 0 \) for all non-negative real eigenvalues. Therefore, it can be clearly seen that \( C_3 > 0, C_2 > 0, C_1 > 0 \) iff,

\[
(1 + d_k - \alpha + \gamma)(1 + d_{l1} + f_1 - \alpha + \sigma)(1 + d_i - \alpha + \eta + \psi_0) + f_i(1 + d_i - \alpha + \sigma)(2 + d_k + d_0 - 2a + \gamma + \eta + \psi_0)f_2 \gamma
\]

where \( f_1, f_2 > 0 \), iff \( G(1 - R_0) - G > G_1 R_0 \), whereas \( G, G_1 > 0 \), iff \( \sqrt{G^2 + k_0 \rho^2 \psi^2} \eta k_0 \beta^2 \gamma (k_0 \beta^2 \gamma + 2G(1 - R_0) + 2G) / R_0 > k_0 \beta^2 \gamma \). The only thing left to be proved is that \( (1 - R_0) < 0 \). Hence, \( \chi_2 \in \mathbb{R}^+ \) is asymptotically stable if \( R_0 > 1 \) and if \( R_0 < 1, (1 - R_0) > 0 \) implies \( T(0) < 0 \), which yields Eq. (23) to have a non-negative real part, thus it becomes unstable.

Numerical findings

Numerical investigations are carried out to validate the aforementioned analytical discussions of the model (6). Some fitted values of the parameters are considered for this purpose, which are mentioned in Table 2 as sets 1 and 2 along with the initial conditions,

\[
\begin{align*}
S(0) &= 100, \\
P(0) &= 0, \\
E(0) &= 0, \\
Q(0) &= 0, \\
I(0) &= 2, \\
R(0) &= 0 \\
\end{align*}
\]

These computations are conducted on Mathematica 12.0 software to reduce the workforce and produce well-defined graphical representations of the governing model. In addition, a comparative analysis is also added in the deliberation to study the effectiveness of the expansion of proportional fractional derivative.

Comparative analysis

The solutions attained by the virtue of special case of the expansion of proportional fractional derivative, are justified through a comparative analysis with the generated solutions of FRK4 algorithm. In [27], FRK4 algorithm is exemplified through programming codes of Mathematica software, which made it an easy user interface. Here, after re-designing the programming code according to the governing model and parameter values, in both sets of Table 2, comparison plots are attained. These graphs as shown in Figs. 2 and 3 represent the solutions of FRK4 method and expansion of PF together, for disease free and endemic states, respectively. In both aforementioned figures, the small octahedron depict the point-wise solutions of FRK4 method for \( h = 0.001 \), whereas straight lines are the solutions of PF expansion, at \( \alpha = 0.998 \) and \( t \in [0, 60] \). In addition, different color scheme of graphs represent the different compartments, as shown through graph legend. These pictorial solutions are in good agreement, which validate the accuracy of the solutions and efficiency of the special simplified case of proportional fraction derivative expansion.

Basic reproduction effect

One of the most important threshold of epidemiology, \( R_0 \), is studied through the Tables 3 and 4. These tables contain the computed values of \( R_0 \) for the model (6) with the parameters of Table 2 and initial conditions (26) at different values of \( \alpha \). Due to the existence of fractional index in\( R_0 \), as can be verified from Eq. (22), the values of \( R_0 \) are generated easily at various points of \( \alpha \). The values of \( S(t), P(t), E(t), Q(t), I(t), M(t) \) and \( R(t) \) along with \( R_0 \) obtained using set 1 of Table 1, are mentioned in Table 3. It can be seen clearly, that basic reproduction number remains less than 1 as \( \alpha \) approaches 1 for these values of parameters and validates the situation of disease free equilibrium points. So, the dynamical history also outlines the same behavior that the population remains disease free until the\( R_0 < 1 \). Unlike this, Table 4 defines the existence of disease in the population for the parameters of

| Parameters | Sets | 1 | 2 |
|-----------|-----|---|---|
| \( \rho \) | 0.7 | 3.781 | |
| \( r_s \) | 19 | 19 | |
| \( k_s \) | 6 | 2.2 | |
| \( r_p \) | 15 | 1.7 | |
| \( k_p \) | 6.5 | 6.5 | |
| \( a \) | 1 | 0.46 | |
| \( b \) | 1 | 1 | |
| \( \eta \) | 0.31 | 13.266 | |
| \( \gamma \) | 0.61 | 1.26 | |
| \( \sigma \) | 0.168 | 0.0714 | |
| \( \rho \) | 0.7 | 1.029 | |
| \( \psi_0 \) | 1.95 | 0.1 | |
| \( \psi_1 \) | 0.026 | 0.9 | |
| \( \delta_1 \) | 0.7 | 0.15 | |
| \( \delta_2 \) | 0.74 | 0.6 | |
| \( \delta_3 \) | 1 | 0.84 | |
| \( \delta_4 \) | 0.3 | 0.45 | |
| \( \delta_5 \) | 0.7 | 0.9 | |
| \( \gamma \) | 1 | 0.771 | |

Fig. 2. Comparative analysis of disease free equilibrium point \( \chi_2 \) with FRK4 method [27] and PF for set 1 of Table 2, \( h = 0.001 \) and \( \alpha = 0.998 \) at different values of \( t \).
set 2 at different values of $\alpha$. Throughout the memory till $\alpha$ approaches to 1, the basic reproduction number remains greater than 1. Consequently, it verifies the aforementioned analytical result, which states the stability of endemic equilibrium point when $R_0 > 1$.

**Memory effect**

Another effective parameter in model (6) is the fractional index of the proportional fractional derivative. It enables to sketch the historical dynamical behaviour of the individuals in each compartment, before the occurrence of a complete change in the system. It can be further illustrated through the attained graphs for model (6), plotted in Figs. 4–17. These diagrams portray the stability of disease free and endemic equilibrium points at different stages of memory. The Figs. 4–10 show the plots of $S(t)$, $P(t)$, $E(t)$, $Q(t)$, $I(t)$, $M(t)$ and $R(t)$ for $\alpha \in [0.1, 1]$ and $t \in [0, 10]$ using the set 1. Evidently, the curves represent the stability of disease free equilibrium points, as it can be easily read that the populations of $E(t)$, $Q(t)$, $I(t)$, $M(t)$ and $R(t)$ become extinct after some time. On the other hand, susceptible and those individuals who are take precautionary measures will survive and thus disease will die out. Figs. 11–17 present plots of $S(t)$, $P(t)$, $E(t)$, $Q(t)$, $I(t)$, $M(t)$ and $R(t)$ for $\alpha \in [0.1, 1]$ and $t \in [0, 10]$ using the set 2. The curves represent the existence of individuals in all compartments that validate the stability of endemic equilibrium points and consequently, it indicate the persistence of diseases in the population. The historical behavior of these discussions are also depicted in figures through grayscale curves. The gradual rise of the curves from grayscale to a dark color explain the memory effect in the asymptotic profiles of $S(t)$, $P(t)$, $E(t)$, $Q(t)$, $I(t)$, $M(t)$ and $R(t)$. Hence, as value of $\alpha$ approaches 1, the level of the curves moves to the higher level, which clearly illustrate the realistic change in the compartments.

**Saturation incidence effect**

Model (6) also comprises the Holling Type II function, which plays an important role as saturation incidence function. It basically measures the dynamics of disease transmission in a crowded population of infectious individuals. The two major parameters that are involved in this function are $\beta$ and $a$, which illustrate the infection force and proportion of infectious individual who did not come to contact with susceptible, respectively. Subsequently, the effects of these parameters are portrayed in Figs. 18 and 19 for $t \in [0, 30]$. In these figures, the graphs are plotted for $E(t)$ at $\alpha = 0.99$ and for different cases of $\beta$ and $a$, while other parameters are kept same as defined in Table 2. Fig. 18 depicts different behavior of exposed individuals for these parameters for the disease free

### Table 3

| $\alpha$ | $R_0$ | $S(t)$ | $P(t)$ | $E(t)$ | $Q(t)$ | $I(t)$ | $M(t)$ | $R(t)$ |
|---------|-------|--------|--------|--------|--------|--------|--------|--------|
| 0.5     | 0.2141| 5.3052 | 6.3269 | 0       | 0       | 0       | 0       | 0       |
| 0.55    | 0.2388| 5.3210 | 6.3475 | 0       | 0       | 0       | 0       | 0       |
| 0.6     | 0.2675| 5.3368 | 6.3691 | 0       | 0       | 0       | 0       | 0       |
| 0.65    | 0.3010| 5.3526 | 6.3906 | 0       | 0       | 0       | 0       | 0       |
| 0.7     | 0.3366| 5.3680 | 6.4121 | 0       | 0       | 0       | 0       | 0       |
| 0.75    | 0.3668| 5.3842 | 6.4336 | 0       | 0       | 0       | 0       | 0       |
| 0.8     | 0.4223| 5.4000 | 6.4551 | 0       | 0       | 0       | 0       | 0       |
| 0.85    | 0.5090| 5.4157 | 6.4766 | 0       | 0       | 0       | 0       | 0       |
| 0.9     | 0.5901| 5.4315 | 6.4982 | 0       | 0       | 0       | 0       | 0       |
| 0.95    | 0.6897| 5.4473 | 6.5197 | 0       | 0       | 0       | 0       | 0       |
| 1.0     | 0.8136| 5.4631 | 6.5412 | 0       | 0       | 0       | 0       | 0       |

### Table 4

| $\alpha$ | $R_0$ | $S(t)$ | $P(t)$ | $E(t)$ | $Q(t)$ | $I(t)$ | $M(t)$ | $R(t)$ |
|---------|-------|--------|--------|--------|--------|--------|--------|--------|
| 0.5     | 1.1410| 1.6011 | 3.9339 | 2.1690 | 0.1808 | 1.6300 | 0.0714 | 0.1116 |
| 0.55    | 1.2101| 1.5826 | 4.0641 | 2.3161 | 0.1937 | 1.8078 | 0.0817 | 0.1240 |
| 0.6     | 1.2855| 1.5643 | 4.1979 | 2.4651 | 0.2068 | 2.0099 | 0.0934 | 0.1375 |
| 0.65    | 1.3268| 1.5464 | 4.3351 | 2.6160 | 0.2202 | 2.2111 | 0.1067 | 0.1523 |
| 0.7     | 1.4584| 1.5287 | 4.4758 | 2.7691 | 0.2339 | 2.4407 | 0.1219 | 0.1686 |
| 0.75    | 1.5579| 1.5113 | 4.6198 | 2.9246 | 0.2478 | 2.6923 | 0.1394 | 0.1864 |
| 0.8     | 1.6678| 1.4942 | 4.7072 | 3.0828 | 0.2621 | 2.9690 | 0.1595 | 0.2063 |
| 0.85    | 1.7895| 1.4774 | 4.8179 | 3.2438 | 0.2768 | 3.2745 | 0.1828 | 0.2283 |
| 0.9     | 1.9248| 1.4610 | 5.0719 | 3.4081 | 0.2918 | 3.6131 | 0.2099 | 0.2520 |
| 0.95    | 2.0761| 1.4449 | 5.2294 | 3.5759 | 0.3072 | 3.9902 | 0.2416 | 0.2807 |
| 1.0     | 2.2458| 1.4292 | 5.3903 | 3.7476 | 0.3230 | 4.4121 | 0.2790 | 0.3121 |
Fig. 5. Memory profiles of \( P(t) \in X_1 \) of SPEQIMR model (6) for parameters mention in set 1 of Table 2, where the gray lines are for \( \alpha \in [0.1, 1] \) and bold blue line for \( \alpha = 1 \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 6. Memory profiles of \( E(t) \in X_1 \) of SPEQIMR model (6) for parameters mention in set 1 of Table 2, where the gray lines are for \( \alpha \in [0.1, 1] \) and bold blue line for \( \alpha = 1 \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 7. Memory profiles of \( Q(t) \in X_1 \) of SPEQIMR model (6) for parameters mention in set 1 of Table 2, where the gray lines are for \( \alpha \in [0.1, 1] \) and bold blue line for \( \alpha = 1 \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 8. Memory profiles of \( I(t) \in X_1 \) of SPEQIMR model (6) for parameters mention in set 1 of Table 2, where the gray lines are for \( \alpha \in [0.1, 1] \) and bold blue line for \( \alpha = 1 \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 9. Memory profiles of \( M(t) \in X_1 \) of SPEQIMR model (6) for parameters mention in set 1 of Table 2, where the gray lines are for \( \alpha \in [0.1, 1] \) and bold blue line for \( \alpha = 1 \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 10. Memory profiles of \( R(t) \in X_1 \) of SPEQIMR model (6) for parameters mention in set 1 of Table 2, where the gray lines are for \( \alpha \in [0.1, 1] \) and bold blue line for \( \alpha = 1 \). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Fig. 11. Memory profiles of $S(t) \in \chi_2$ of SPEQIMR model (6) for parameters mention in set 2 of Table 2, where the gray lines are for $\alpha \in [0, 1]$ and bold blue line for $\alpha = 1$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 12. Memory profiles of $P(t) \in \chi_2$ of SPEQIMR model (6) for parameters mention in set 2 of Table 2, where the gray lines are for $\alpha \in [0, 1]$ and bold blue line for $\alpha = 1$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 13. Memory profiles of $E(t) \in \chi_2$ of SPEQIMR model (6) for parameters mention in set 2 of Table 2, where the gray lines are for $\alpha \in [0, 1]$ and bold blue line for $\alpha = 1$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 14. Memory profiles of $Q(t) \in \chi_2$ of SPEQIMR model (6) for parameters mention in set 2 of Table 2, where the gray lines are for $\alpha \in [0, 1]$ and bold blue line for $\alpha = 1$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 15. Memory profiles of $I(t) \in \chi_2$ of SPEQIMR model (6) for parameters mention in set 2 of Table 2, where the gray lines are for $\alpha \in [0, 1]$ and bold blue line for $\alpha = 1$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 16. Memory profiles of $M(t) \in \chi_2$ of SPEQIMR model (6) for parameters mention in set 2 of Table 2, where the gray lines are for $\alpha \in [0, 1]$ and bold blue line for $\alpha = 1$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
The parameters such as infection force, rate of exposed, rate of quarantine, and isolation are effective in controlling the spread of the disease. Moreover, the effectiveness of social distancing to kill the transmissibility process of the disease is beneficial to control the infection before its outbreak. Conversely, the helpful effectiveness of these parameters is also found in the case where infection force is high i.e., \( \beta = 2 \) but less than the previous case if infection force is high and a larger proportion of infective is not in contact of susceptible i.e., at \( a = 2 \). In one-way or other, these parameters describe the effectiveness of social distancing to kill the transmigration of the deadly pandemic from the population in a realistic manner.

Conclusions

Consequently, from all analytical discussions and numerical illustrations through graphs and tables we may conclude effective investigations from the whole study as follows.

The governing compartmental model was designed with special simplified case of proportional fractional derivative, where its expansion reduced the fractional order system to an integer order system of differential equations. From the comparative analysis of the obtained solutions with the solutions of FRK4 method, the effectiveness of the expansion of proportional fractional derivative is validated. This expansion enables to involve the fractional index in the findings of each epidemic threshold, therefore each can be studied with the memory effect. Knowing the historical behavior of any dynamical model of any disease is beneficial to control the infection before its outbreak.

The designed model for the susceptible population: the parameters involved in this function deliberate different scenarios of the population at different level of infection force and contact proportion of infective. Moreover, the help in these parameters is also found in the case when infection force is high i.e., \( \beta = 1 \) and a larger proportion of infective is not in contact of susceptible i.e., at \( a = 2 \). In one-way or other, these parameters describe the effectiveness of social distancing to kill the transmigration of the deadly pandemic from the population in a realistic manner.

Epidemiological models play a vital role in managing the deadly pandemic from the population in a realistic manner.
diseases before its outbreak. Innovatively, designing such models is of great concern for health care researchers. In future, we aim to bring latest assumptions and techniques to make the study of such paradigm of equations effective and beneficial for health care system.

CRediT authorship contribution statement

Oyoon Abdul Razzaq: Conceptualization, Methodology, Writing - original draft, Writing - review & editing. Najeeb Alam Khan: Conceptualization, Writing - original draft, Writing - review & editing, Supervision. Muhammad Falzan: Methodology, Software, Investigation, Writing - original draft. Asmat Ara: Validation, Formal analysis, Investigation, Visualization. Saif Ullah: Validation, Formal analysis, Investigation, Visualization.

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.

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