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ECONOMIC IMPACT OF EPIDEMICS: MATHEMATICAL MODEL AND DYNAMICAL ANALYSIS

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Abstract. The predicament of the economic health and human capital of any nation in the wake of an epidemic requires further studies to better handle both. Scientific research can assist in developing measures for managing an epidemic without generating substantial economic shocks because mitigation strategies, while helpful in controlling an outbreak, may also pose significant economic challenges. To this effect, we have constructed a six-compartment epidemic-economic model wherein the population is classified into four different classes: susceptible, vaccinated, infected, and recovered. The other two compartments are for mitigation level and economic development level. Existence of disease free and endemic equilibrium points, as well as the prerequisites for both their local and global stability have been attained. We have conducted a numerical simulation by using a feasible set of parameters to bolster our analytical findings. An analysis of the local sensitivity of the reproduction number provides insight into potential epidemic management strategies. According to our findings, if immunisations are given, less intensive mitigation measures can not only successfully lower the number of infectives but can also be beneficial to the economy. We have conducted a numerical simulation by using a feasible set of parameters to bolster our analytical findings.

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1. INTRODUCTION

Illness caused by the transfer of viruses and bacteria's from one human body to another via different channelling modes such as insect bites, airborne contamination, body fluids, blood products, or contact with infected surfaces is known as the contagious disease [1]. Examples of some such communicable diseases are Influenza, HIV, Ebola, Measles, etc. Finding the source of a sickness that has entered our society and treating the afflicted individuals medically to stop the illness from spreading are the first steps in combating any disease. An infectious disease is considered to be an epidemic disease when it outbursts and surfeits the anticipation of normal cases. There are also a few epidemic diseases which cannot be cured completely but with the proper care and treatment lives of infected can be prolonged for years. For example, HIV/AIDS which first surfaced in 1959 in Democratic Republic of Congo and it still continues to affect the global population with over 39.9 million cases documented as of 2023 [2]. The only way to hinder its spread is by taking the proactive steps. Seasonal influenza, commonly known as flu, is a serious respiratory illness that is omnipresent across the globe and is caused by influenza viruses. Sometimes, due to the severity of this illness may lead the infectives to get an immediate medical attention to recuperate from fever and other symptoms. On the other side, many people with flu can regain their health back on their own within a week if they get enough rest and drink lots of fluids. Different strains of influenza viruses are classified into four types: A, B, C, and D where Influenza A and B viruses are the source of seasonal outbreaks in human. Each year, over billion of cases are reported due to seasonal influenza with a total count of around $3-5$ million leading to a chaos in population and around 290000-650000 deaths yearly from respiratory disease. In underdeveloped nations, lower respiratory tract infections caused by influenza account for 99% of mortality in children under the age of five [3]. Ebola, originally known as Ebola hemorrhagic fever is a deadly disease that affects both human species and other primates. On an average mortality rate of Ebola patients is around 50 percent whereas the fatality rate during the prior outbreaks have varied from 25 to 90 percent, based on circumstances

and the reactions. Also, the first ever cases on Ebola virus was identified in 1976 which outburst simultaneously in Democratic Republic of the Congo and South Sudan. In outbreaks, the spread of Ebola has been managed with vaccinations against certain strains of the disease [4]. Another deadly communicable disease that existed for at-least 3000 years is smallpox which took away the lives of millions. Owing to the contributions of the physician Edward Jenner [5], and with the growth of science and medicine, smallpox has now been eradicated [6] from modern civilization.

Over the years, humans have faced different epidemic diseases, including COVID-19, which has been recognized as the pervasive disease of 21st century. COVID-19 was first discovered in Wuhan, China and the spread of COVID-19 sailed to every nation on earth. On March 11, 2020 WHO declared COVID-19 as global pandemic [7] and alerted the world about the incoming of the disease to prepare for the impending battle. With global population of 8.1 billion, around 704 million were impacted due to COVID-19, resulting in the loss of lives of seven million people which officially is stated by WHO [8] as of 2024. Implementing mitigation strategies like travel bans, mask-wearing requirements, social distancing, and quarantining affected individuals can helps to slow down the spread of COVID-19 [9, 10]. Since epidemics have a devastating effect on a nation's physical health, researchers must analyse the dynamics of such epidemic diseases and foresee the count of new cases to ensure that the health care systems are prepared to handle the influx of patients and treat those who are infected with the best available care.

1.1. Existing Literature and Motivation. Understanding the dynamics of an epidemic disease is crucial for identifying elements that contribute to infectious transmission. Mathematical modelling of transmissible diseases can shed light on the various factors that contribute to disease spread, allowing policymakers to devise suitable policies. In [11] authors made use of the compartmental modeling approach to understand disease dynamics. Their study focuses on apprehending the effects of the various factors influencing the spread of contagious diseases. This model operated under the presumption that models consisting of three compartments—the vulnerable individuals, the infected individuals, and the dead (or immune) individuals, can replicate the number of cases. Many researchers have since examined a number of mathematical models to comprehend the transmission of infectious diseases [12, 13, 14, 15, 16, 17, 18, 19]. The

study done in [14] explains the spread of the 2014 Ebola virus outbreak using the conventional SIR model. This study demonstrated how public health interventions might finally lower the effective reproductive number and put an end to the outbreak. Immunisations against the disease can be administered to people who are at risk for it. By encouraging hand washing and the use of hand sanitisers, one can reduce the transmissibility. The contact rate can be reduced by encouraging those who are sensitive to isolate. To create the best strategy for eradicating the Ebola virus disease, researchers in [16] modified the *SEIR* model and included hospital isolation, Ebola drugs, and vaccines. Researchers assessed the number of new cases in four important transmission pathways—community, household, unsafe funeral and hospital—using the regional spread of EVD and their transmission model learning. The authors concluded that methodical planning, efficient hospital isolation, and efficient EVD medication and immunisation are necessary for the eradication of Ebola. In [19] an HIV/AIDS epidemic model with treatment and time delay is investigated. Authors noted that delay induced Hopf bifurcations. A deterministic mathematical model is developed by the authors of [15] to study the dynamics of influenza transmission. The model is used to determine the threshold vaccination rate required for influenza control throughout the population. The authors suggested a deterministic SEIR model based on the clinical course of the disease, individual epidemiological status, and intervention strategies to assess the trend of the COVID-19 pandemic in China during the final phase of the outbreak [17]. Authors suggested the strategy for isolation and quarantine as well as increasing the country's rate of detection to control the spread.The impact of travel restrictions and human mobility on transmission and spatial dissemination of COVID-19 was examined by the authors in [18]. According to their qualitative and quantitative study, the travel ban may be able to stop a serious outbreak. The authors in their work [20] created a mathematical model that accounts for vaccination in susceptible and recovered groups in order to better understand how the COVID-19 outbreak spread. According to their findings, vaccination campaigns can effectively stop the COVID-19 virus from spreading, and expanding vaccination campaigns can aid in outbreak containment.

Because of the rise in mortality rates, the fear-induced behaviour, and the mitigation measures like travel restrictions and lockdowns, epidemics frequently generate economic disruptions. The

authors in [21] proclaimed that the epidemic disease impacts the nation's economy through various channels, which include tourism, transportation, agriculture, and health sectors. The impact of contagious disease results not only in loss of millions of lives but also weakens the economic growth of several nation. For instance, Asian flu pandemics that occurred in $1957-1958$ shook the world by taking away lives of around 1.5 million and loss in GDP by three percent in Australia, Canada, United Kingdom and in USA [22]. Apart from the negative impact on health, the Ebola pandemic caused an estimated U.S.$2.2 billion in losses to the GDP of Guinea, Sierra Leone, and Liberia in 2015 [23]. During COVID-19, non-essentials businesses closed during the US "stay-at-home" period, which ran from March to May 2020 [24]. As a result, the US saw a substantial spike in unemployment by the end of March 2020, with a 9.5 % rate [25, 24]. Global losses were estimated to be 5.5%–8.7% of global GDP in 2020 and 3.6%–6.3% of global GDP in 2021, respectively, compared to a no-COVID-19 baseline. Developing Asia's losses were estimated to be 6.0%–9.5% of regional GDP in 2020 and 3.6%–6.3% of regional GDP in 2021 [26]. Several studies were conducted to asses the impact of COVID-19 on economies [27, 28, 24, 29, 30, 31]. According to authors in [30], economic damage can be reduced without catastrophically increasing the number of hospitalised and deceased individuals when an extremely strict isolation policy for the senior population is combined with a very gradual removal of mandatory isolation for younger citizens once the outbreak is largely controlled. In [31], authors assessed the effects on health and economy of several age-based vaccine prioritisation schemes in the European Region of the World Health Organisation (WHO). The authors of [24] present a novel mathematical model to examine the COVID-19 outbreak and its economic ramifications, emphasising the interplay of disease transmission, pandemic containment, and economic expansion. Nevertheless, vaccination—a crucial element in epidemic control—has not been considered in their study [24]. Vaccination campaigns have the potential to be very effective in halting the spread of illness [32]. Though the vaccinations do not hold hundred percent success rate but it helps to prevent the individuals moving from susceptible to infected classes at least for some time or likely forever [33]. It becomes imperative to vaccinate those who are susceptible in order to boost community immunity and aid in infection management.

We expand on their approach [24] by incorporating vaccination into our own model and researching the subsequent issues:

- Effect of mitigation strategies on economic development level in a system with and without vaccination.
- Evaluating the impact of vaccination on the epidemic outbreak and its financial ramifications.
- Role of Vaccine efficacy on the disease control and its financial ramifications.

1.2. Organizational Structure. The paper is organised as follows: In Section 2, a mathematical model has been developed along with its assumptions; positivity and boundedness of the system (7) have been discussed in Subsection 2.1. Rigorous analysis of the system (7) has been studied in Section 3; disease free and endemic equilibrium points have been obtained in Subsection 3.1; the basic reproduction number has been derived in Subsection 3.2; subsection 3.3 and Subsection 3.4 deal with the local stability and global stability of equilibrium points, respectively . Analytical results have been verified through numerical simulation in Section 4. Lastly, discussion and conclusion have been provided in Section 5.

2. Establishment of Model with Its Assumption

We assume that the population affected by an outbreak of any disease is divided into four classes: susceptible (S), vaccinated (V), infected (I), and recovered (R). The population as a whole is given by $N(t) = S(t) + V(t) + I(t) + R(t)$ at time t. The model has been developed with the following presumptions:

- Recruitment of individuals due to immigration will increase the susceptible population at the rate of λ. Whereas, the susceptible class will decrease by either the natural mortality rate, vaccination rate, or disease transmission caused by the interaction of people in the susceptible class with the infected class. Therefore, the rate of change per unit time in susceptible class is as follows:

$$\frac{dS}{dt} = \lambda - \frac{\beta SI}{1+bM} - \mu S - pS. \tag{1}$$

- Vaccinated individuals increase due to the vaccination of susceptible individuals at the rate p. As the vaccination is not hundred percent effective, the vaccinated population is likely to get infected at a rate of $(1-\sigma)\frac{\beta I}{1+bM}$, where the effect of mitigation strategies is taken into account. Here, σ signifies the vaccine efficacy; if $\sigma = 1$, then the vaccine is effective, and on the other side, if $\sigma = 0$, then the vaccine is ineffective. Also, μ is the mortality rate at which the vaccinated people decrease due to natural deaths. Thus, the rate of change in the vaccinated population per unit time is as follows:

$$\frac{dV}{dt} = pS - (1-\sigma)\frac{\beta VI}{(1+bM)} - \mu V. \tag{2}$$

- The increase in the infective class is due to the interaction of susceptible and vaccinated people with infective. The transmission of vaccinated people to the class of infected people depends largely on vaccine efficacy. A drop in the infected class can be seen due to the recovery (μ_1) and natural mortality (μ) of the infected individuals. The equation for the rate of change in the infected class per unit time is formulated as:

$$\frac{dI}{dt} = \frac{\beta(S+(1-\sigma)V)I}{(1+bM)} - (\mu_1+\mu)I. \tag{3}$$

- The increase in the recovered class is due to the recovery of the infected population, and the decrease is a result of natural mortality at a rate of μ. Thus, the equation for the rate of change in the recovered class per unit time is formulated as:

$$\frac{dR}{dt} = \mu_1 I - \mu R. \tag{4}$$

- To control an epidemic, public health mitigation policies, such as personal and environmental hygienic practices, isolation and quarantine of known cases, contact tracing, social distancing, and closure of non-essential businesses and services [34], help in slowing the transmission of the virus and reducing the number of cases in communities [35]. Another variable, M, for mitigation level is incorporated into the model. A drop in mitigation level is due to the natural reduction of mitigation at the rate of p_1 and also to the decline of mitigation due to economic activities at the rate of f_0. Presumably, the number of infectives and the inefficacy of vaccines promote the degree of mitigation. Thus, below is the equation for the rate of change in mitigation level per unit time,

where the range of M is normalised so that the value of M lies between zero and one, where $M = 1$ denotes the upper limit of disease control and $M = 0$ the zero disease control.

$$\frac{dM}{dt} = \delta + mI + m(1-\sigma)V - p_1 M - f_0 K. \tag{5}$$

- Such mitigation strategies have an effect on economic development. In order to study the effect of mitigation strategies on economic development, another variable, K, is incorporated into the model. Economic development depends on the availability of the labor force, which constitutes the population of all the classes except the infected class, i.e., S, V, and R. Also, the rate of economic development per unit time decreases due to a natural reduction in economic development at the rate of d and a reduction in economic growth due to mitigation at the rate of g_0. Therefore, below is the equation for the rate of economic development per unit time, where we normalise K to lie between $0 \leq K \leq 1$, i.e., when $K = 1$, the highest possible degree of economic growth exists, and when $K = 0$, the lowest possible level of economic growth exists.

$$\frac{dK}{dt} = C_S S + C_V V + C_R R - dK - g_0 M. \tag{6}$$

Thus, below is the system of differential equation with explanations for each of the parameters listed in Table 2:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \lambda - \frac{\beta SI}{1+bM} - \mu S - pS, \\
\frac{dV}{dt} &= pS - (1-\sigma)\frac{\beta VI}{(1+bM)} - \mu V, \\
\frac{dI}{dt} &= \frac{\beta(S+(1-\sigma)V)I}{(1+bM)} - (\mu_1+\mu)I, \\
\frac{dR}{dt} &= \mu_1 I - \mu R, \\
\frac{dM}{dt} &= \delta + mI + m(1-\sigma)V - p_1 M - f_0 K, \\
\frac{dK}{dt} &= C_S S + C_V V + C_R R - g_0 M - dK,
\end{aligned}\right\} \tag{7}$$

with initial conditions as $S(0) > 0$, $V(0) \geq 0$, $I(0) \geq 0$ and $R(0) \geq 0$.

Parameter	Description
β	Transmission rate
λ	Recruitment rate of susceptible
σ	$0 \leq \sigma \leq 1$, Vaccine efficacy
μ_1	Recovery rate
μ	Natural mortality rate
m	Rate of mitigation rules
p_1	Natural mitigation diminution rate
f_0	Rate of mitigation decline brought on by economic activity
p	Vaccination rate
δ	Influx rate of disease mitigation
C_S	Rate of labour contribution from S
C_V	Rate of labour contribution from V
C_R	Rate of labour contribution from R
b	Implementation rate of disease mitigation
d	Natural reduction rate of economic development
g_0	Rate of economic growth decline brought on by mitigation

TABLE 1. Description of Parameters.

2.1. Preliminaries. In this subsection we obtain the conditions under which the solutions of the system (7) are positive and bounded.

From system (7), we attain

$$\dot{S}+\dot{V}+\dot{I}+\dot{R} = \lim_{t\to\infty} Sup(N(t)) \leq \frac{\lambda}{\mu}. \tag{8}$$

From the first four equations of the system (7), we have

$$\left.\frac{dS}{dt}\right|_{S=0} = \lambda > 0,\ \left.\frac{dV}{dt}\right|_{V=0} = pS > 0,\ \left.\frac{dI}{dt}\right|_{I=0} = 0,\ \left.\frac{dR}{dt}\right|_{R=0} = \mu_1 I \geq 0.$$

On the boundary planes, all of the rates are non-negative in this case. Because the vector field's orientation is inward on all of the bounding planes, if we begin anywhere inside $\mathbb{R}^4_+$, we will

stay there forever. As a result, it is assured that the solutions of first four equations of the model (7) are non-negative.

We now derive conditions that guarantee the non-negativeness of M and K. For $M \geq 0$, we need $\frac{dM}{dt} \geq 0$ when $M = 0$. This yields $\delta + mI + m(1-\sigma)V - f_0 K \geq 0$, which holds for all $I, V \geq 0$ and $K \leq 1$ if, $\delta - f_0 \geq 0$.

Hypothesis 1. $\delta - f_0 \geq 0$.

Adding the first two equations of system (7) yields,

$$\dot{S} + \dot{V} \geq \frac{\lambda \mu}{\mu^2 + \beta \lambda}. \tag{9}$$

Similarly, $K \geq 0$ if, $c_m \frac{\lambda \mu}{\mu^2 + \beta \lambda} - g_0 \geq 0$, where, $d_m = min(C_S,\, C_V,\, C_R)$.

Hypothesis 2. $d_m \frac{\lambda \mu}{\mu^2 + \beta \lambda} - g_0 \geq 0$.

To make sure that $M \leq 1$, we require $\frac{dM}{dt} \leq 0$ when $M = 1$, which results to $\delta + mI + m(1-\sigma)V - p_1 - f_0 K \leq 0$, and holds for all $I, V \leq \frac{\lambda}{\mu}$ and $K \geq 0$ if, $\delta + \frac{2\lambda m}{\mu} - \sigma m \frac{\lambda}{\mu} - p_1 \leq 0$.

Hypothesis 3. $\delta + \frac{2\lambda m}{\mu} - \sigma m \frac{\lambda}{\mu} - p_1 \leq 0$.

$K \leq 1$ can be ensured using condition (8), and $C_M \frac{\lambda}{\mu} - d \leq 0$, where, $C_M = max(C_S, C_V, C_R)$.

Hypothesis 4. $C_M \frac{\lambda}{\mu} - d \leq 0$.

Thus if hypotheses 1, 2, 3, and 4 holds, the biologically feasible and positively invariant set for the system (7) is as follows:-

$$\Gamma = \left\{ (S,V,I,R,M,K) \in \mathbb{R}^6_+ :\ 0 \leq S+V+I+R \leq \frac{\lambda}{\mu},\ 0 \leq M \leq 1,\ 0 \leq K \leq 1 \right\}.$$

3. Rigorous Analysis of the Model (7)

3.1. Equilibrium Points. In this subsection, for system (7), two equilibrium points- disease free and endemic equilibrium points are evaluated.

- **Disease free equilibrium point** For the system (7) the disease free equilibrium point $P_0 = (S_0, V_0, I_0, R_0, M_0, K_0)$ is:

$$\begin{aligned} S_0 &= \frac{\lambda}{(\mu+p)},\ V_0 = \frac{p\lambda}{\mu(\mu+p)},\ I_0 = 0,\ R_0 = 0, \\ M_0 &= \frac{f_0 d}{p_1 d - f_0 g_0}\left(\frac{\delta}{f_0} + \frac{m(1-\sigma)p\lambda}{f_0\mu(\mu+p)} - \frac{C_S\lambda}{d(\mu+p)} - \frac{C_V p\lambda}{d\mu(\mu+p)}\right), \\ K_0 &= \frac{g_0 p_1}{p_1 d - f_0 g_0}\left(-\frac{\delta}{p_1} - \frac{m(1-\sigma)p\lambda}{p_1\mu(\mu+p)} + \frac{C_S\lambda}{g_0(\mu+p)} + \frac{C_V p\lambda}{g_0\mu(\mu+p)}\right). \end{aligned}$$

- **Endemic equilibrium point** Now, we evaluate the endemic equilibrium point $P^* = (S^*, V^*, I^*, R^*, M^*, K^*)$ of system (7).

Adding equations (1), (2), (3), we get

$$S^* = \frac{\lambda - \mu V - (\mu_1 + \mu)I}{\mu}. \tag{10}$$

From (4), we get

$$R^* = \frac{\mu_1}{\mu} I^*. \tag{11}$$

From equation (6), we get

$$K^* = \frac{C_S S^* + C_V V^* + C_R R^* - g_0 M^*}{d}. \tag{12}$$

Next, substituting value of K^* from equation (12), S^* from equation (10), and R^* from equation (11) in equation (5), we get

$$M^* = Q_{11} + Q_{12} V + Q_{13} I, \tag{13}$$

where $Q_{11} = \dfrac{\mu d\delta - f_0 C_S\lambda}{\mu(p_1 d - f_0 g_0)},\ Q_{12} = \dfrac{\mu md(1-\sigma) - \mu f_0 C_V + \mu f_0 C_S}{\mu(p_1 d - f_0 g_0)},$

$$Q_{13} = \frac{\mu md - f_0 C_R \mu_1 + f_0 C_S(\mu_1 + \mu)}{\mu(p_1 d - f_0 g_0)}.$$

Substituting the value of S^* from equation (10) and value of M^* from (13) into equation (3), we get the V^* in the form of I^* as given below:

$$V^* = Q_{14} - Q_{15} I, \tag{14}$$

where, $Q_{14} = \dfrac{\beta\lambda - \mu(\mu_1+\mu)(1+bQ_{11})}{\beta\mu\sigma + b\mu(\mu+\mu_1)Q_{12}},\ Q_{15} = \dfrac{(\mu_1+\mu)(\beta + b\mu Q_{13})}{\beta\mu\sigma + b\mu(\mu+\mu_1)Q_{12}}.$

From equation (10) and (14), we get

$$S^* = Q_{16} + Q_{17} I, \tag{15}$$

where $Q_{16} = \dfrac{\lambda}{\mu} - Q_{14},\ Q_{17} = Q_{15} - \dfrac{\mu_1 + \mu}{\mu}$.

Substituting the values of S^* from equation (15), V^* from equation (14), and R^* from equation (11) into equations (5) and (6), we get the values of M^* and K^* in terms of I^*, which are given as below:

$$M^* = Q_{18} + Q_{19} I, \tag{16}$$

where

$$\begin{aligned} Q_{18} &= \frac{d(\delta + m(1-\sigma)Q_{14}) - f_0(C_S Q_{16} + C_V Q_{14})}{p_1 d - f_0 g_0}, \\ Q_{19} &= \frac{d(m - m(1-\sigma)Q_{15}) - f_0\left(C_S Q_{17} - C_V Q_{15} + C_R \dfrac{\mu_1}{\mu}\right)}{p_1 d - f_0 g_0}. \end{aligned}$$

and

$$K^* = Q_{20} + Q_{21} I, \tag{17}$$

where

$$\begin{aligned} Q_{20} &= \frac{p_1(C_S Q_{16} + C_V Q_{14}) - g_0(\delta + m(1-\sigma)Q_{14})}{p_1 d - f_0 g_0}, \\ Q_{21} &= \frac{p_1\left(C_S Q_{17} - C_V Q_{15} + C_R \dfrac{\mu_1}{\mu}\right) - g_0(m - m(1-\sigma)Q_{15})}{p_1 d - f_0 g_0}. \end{aligned}$$

From equations (13),(14), (15), and (3), we get

$$I^* = \frac{\beta Q_{16} + \beta(1-\sigma)Q_{14} - (\mu_1 + \mu) - b(\mu_1 + \mu)Q_{18}}{\beta(1-\sigma)Q_{15} + b(\mu_1 + \mu)Q_{19} - \beta Q_{17}}.$$

3.2. Basic reproduction number (BRN). BRN is crucial in understanding the factors that contributes to the disease spread in epidemiology. To calculate the basic reproduction number, the next generation matrix method is used [36]. Therefore, basic reproduction number (R_{vac}) for the system (7) is:

$$\begin{aligned} R_{vac} &= \frac{\beta(S_0 + (1-\sigma)V_0)}{(1 + bM_0)(\mu_1 + \mu)}, \\ &= \frac{\beta\lambda(\mu + (1-\sigma)p)(p_1 d - f_0 g_0)}{(\mu + \mu_1)((p_1 d - f_0 g_0)\mu(\mu + p) + b(d\delta\mu(\mu + p) + dm(1-\sigma)p\lambda - C_S\mu\lambda f_0 - C_V f_0 p\lambda))}. \end{aligned}$$

3.3. Local stability of equilibrium points of the system (7).

Theorem 3.1. *If $p_1 d - f_0 g_0 > 0$ and $R_{vac} < 1$, then the disease free equilibrium point P_0 of the system (7) is locally asymptotically stable.*

Proof. System (7) may be written as $\dot{z} = g(z, R_{vac})$, where $z = [z_1, z_2, z_3, z_4, z_5, z_6]^T = [S, V, I, R, M, K]^T$ and $g = [g_1, g_2, g_3, g_4, g_5, g_6]^T = \dot{z}$.

The Jacobian matrix of system (7) for E_0 is given by:

$$\mathbf{D_x} g(P_0, R_{vac}) = \begin{bmatrix} -(\mu+p) & 0 & -\dfrac{\beta S_0}{1+bM_0} & 0 & 0 & 0 \\ p & -\mu & -\dfrac{(1-\sigma)\beta V_0}{1+bM_0} & 0 & 0 & 0 \\ 0 & 0 & \dfrac{\beta S_0 + \beta(1-\sigma)V_0}{1+bM_0} - (\mu+\mu_1) & 0 & 0 & 0 \\ 0 & 0 & \mu_1 & -\mu & 0 & 0 \\ 0 & m(1-\sigma) & m & 0 & -p_1 & -f_0 \\ C_S & C_V & 0 & C_R & -g_0 & -d \end{bmatrix}$$

Characteristic equation for disease free equilibrium point is as follows:

$$-[(\mu+p)+\Delta](\mu+\Delta)^2[\Delta^2+\Delta(p_1+d)+(p_1 d - f_0 g_0)][R_{vac}(\mu+\mu_1)-(\mu+\mu_1)-\Delta] = 0 \tag{18}$$

Eigenvalues for the above characteristic equation are:

$$-(\mu+p), -\mu, -\mu, \frac{-(p+d) \pm \sqrt{(p_1+d)^2 - 4(p_1 d - f_0 g_0)}}{2}, (R_{vac}-1)(\mu_1+\mu)$$

All the six eigenvalues are less than zero if and only if $p_1 d - f_0 g_0 > 0$ and $R_{vac} < 1$. □

Existence of Backward Bifurcation

In the case when $R_{vac} = 1$, one of the eigenvalues of the characteristic equation (18) vanishes, so we apply the center manifold theory to determine the bifurcation coefficients [12, 37]. In order to evaluate the bifurcation coefficients a, b, we evaluate the first and second order derivatives of g_i with respect to all the variables, S, V, I, R, M, K and R_{vac}. Except the below mentioned

second derivatives of g_i in system (7), rest all vanish at the disease free equilibrium point P_0:

$$\frac{\partial^2 g_1}{\partial S \partial I}(P_0,1) = \frac{-\beta}{1+bM_0},\ \frac{\partial^2 g_1}{\partial M \partial I}(P_0,1) = \frac{\beta S_0 b}{(1+bM_0)^2}, \frac{\partial^2 g_2}{\partial I \partial V}(P_0,1) = \frac{-(1-\sigma)\beta}{1+bM_0},$$

$$\frac{\partial^2 g_2}{\partial I \partial M}(P_0,1) = \frac{(1-\sigma)\beta b V_0}{(1+bM_0)^2},\ \frac{\partial^2 g_3}{\partial I \partial S}(P_0,1) = \frac{\beta}{1+bM_0}, \frac{\partial^2 g_3}{\partial V \partial I}(P_0,1) = \frac{(1-\sigma)\beta}{1+bM_0},$$

$$\frac{\partial^2 g_3}{\partial M \partial I}(P_0,1) = \frac{-\beta b}{(1+bM_0)^2}(S_0+(1-\sigma)V_0).$$

Corresponding first derivatives of f_i in (7) are listed as follows:

$$\begin{aligned}
\frac{\partial g_1}{\partial I}(P_0,1) &= \frac{-\beta S_0}{(1+bM_0)} = -(\mu+\mu_1)R_{vac} + R_{02}(\mu+\mu_1),\\
\frac{\partial g_2}{\partial I}(P_0,1) &= \frac{-\beta(1-\sigma)V_0}{(1+bM_0)} = -(\mu+\mu_1)R_{vac} + R_{01}(\mu+\mu_1),\\
\frac{\partial g_3}{\partial I}(P_0,1) &= \frac{\beta(S_0+(1-\sigma))V_0}{(1+bM_0)} - (\mu+\mu_1) = (\mu+\mu_1)R_{vac} - (\mu+\mu_1).
\end{aligned}$$

where, $R_{01} = \dfrac{\beta S_0}{(1+bM_0)(\mu_1+\mu)}$ and $R_{02} = \dfrac{\beta((1-\sigma)V_0)}{(1+bM_0)(\mu_1+\mu)}$.
Therefore,

$$\frac{\partial^2 g_1}{\partial I \partial R_{vac}}(P_0,1) = -(\mu+\mu_1),\ \frac{\partial^2 g_2}{\partial I \partial R_{vac}}(P_0,1) = -(\mu+\mu_1),\ \frac{\partial^2 g_3}{\partial I \partial R_{vac}}(P_0,1) = (\mu+\mu_1). \tag{19}$$

Let $\tilde{\xi}$ and $\tilde{\zeta}$ be such that $\tilde{\xi} D_z f(P_0,1) = 0$, $D_z f(P_0,1)\tilde{\zeta} = 0$ and $\tilde{\xi}.\tilde{\zeta} = 1$. We obtain $\upsilon = (\tilde{\xi}_1, \tilde{\xi}_2, \tilde{\xi}_3, \tilde{\xi}_4, \tilde{\xi}_5, \tilde{\xi}_6) = (0,0,1,0,0,0)$, and $\tilde{\zeta} = (\tilde{\zeta}_1, \tilde{\zeta}_2, \tilde{\zeta}_3, \tilde{\zeta}_4, \tilde{\zeta}_5, \tilde{\zeta}_6)$, where

$$\begin{aligned}
\tilde{\zeta}_1 &= \frac{-\beta S_0}{(1+bM_0)(\mu+p)},\ \tilde{\zeta}_2 = -\frac{p(\mu+\mu_1)}{\mu(\mu+p)} + \frac{-1}{\mu(\mu+p)}\frac{\beta(1-\sigma)V_0}{1+bM_0},\ \tilde{\zeta}_3 = 1,\ \tilde{\zeta}_4 = \frac{\mu_1}{\mu},\\
\tilde{\zeta}_5 &= \frac{1}{p_1 d - f_0 g_0}\left[md + (md(1-\sigma) - f_0 C_V)\left(-\frac{p(\mu+\mu_1)}{\mu(\mu+p)} + \frac{-1}{\mu(\mu+p)}\frac{\beta(1-\sigma)V_0}{1+bM_0}\right)\right.\\
&\quad \left. -\frac{f_0 C_R \mu_1}{\mu} + \frac{\beta f_0 C_S S_0}{(1+bM_0)(\mu+p)}\right],\\
\tilde{\zeta}_6 &= \frac{1}{f_0 g_0 - p_1 d}\left[mg_0 + (mg_0(1-\sigma) - p_1 C_V)\left(-\frac{p(\mu+\mu_1)}{\mu(\mu+p)} + \frac{-1}{\mu(\mu+p)}\frac{\beta(1-\sigma)V_0}{1+bM_0}\right)\right.\\
&\quad \left. -\frac{p_1 C_R \mu_1}{\mu} + \frac{\beta p_1 C_S S_0}{(1+bM_0)(\mu+p)}\right].
\end{aligned}$$

Now, the bifurcation coefficients are evaluated as follows:

$$\begin{aligned}
a &= \tilde{\zeta}_1 \frac{\beta}{1+bM_0} + \tilde{\zeta}_2 \frac{(1-\sigma)\beta}{1+bM_0} - \tilde{\zeta}_5 \frac{\beta b}{(1+bM_0)^2}(S_0+(1-\sigma)V_0),\\
b &= \mu+\mu_1 > 0.
\end{aligned}$$

Let $\varpi = \dfrac{f_0 C_R \mu_1}{\mu} - (md(1-\sigma) - f_0 C_V)\left(\dfrac{-p(\mu+\mu_1)}{\mu(\mu+p)} + \dfrac{-1}{\mu(\mu+p)}\dfrac{\beta(1-\sigma)V_0}{1+bM_0}\right) - md - \dfrac{\beta f_0 C_S S_0}{(1+bM_0)(\mu+p)}$.

If $\varpi < 0$, and $p_1 d - f_0 g_0 > 0$, then $a < 0$ and therefore, the system (7) does not experience backward bifurcation, it instead experiences forward bifurcation. Thus, we have the following theorem:

Theorem 3.2. *If $\varpi < 0$ and $p_1 d - f_0 g_0 > 0$, then the system (7) undergoes forward bifurcation.*

Theorem 3.3. *The endemic equilibrium point P^* of the system (7) is locally asymptotically stable if $|H_n^{'}| > 0$ for all $n = 1,2,3,4,5,6$, where $|H_n^{'}|$ are defined in (21).*

Proof. Jacobian matrix of the system (7) is at the endemic equilibrium point P^* is given by:

$$\begin{bmatrix} \mathscr{G}_{11} & 0 & \mathscr{G}_{12} & 0 & \mathscr{G}_{13} & 0 \\ p & \mathscr{G}_{21} & \mathscr{G}_{22} & 0 & \mathscr{G}_{23} & 0 \\ \mathscr{G}_{31} & \mathscr{G}_{32} & \mathscr{G}_{33} & 0 & \mathscr{G}_{34} & 0 \\ 0 & 0 & \mu_1 & -\mu & 0 & 0 \\ 0 & m(1-\sigma) & m & 0 & -p_1 & -f_0 \\ C_S & C_V & 0 & C_R & -g_0 & -d \end{bmatrix}$$

where $\mathscr{G}_{11} = \dfrac{-\beta I^*}{1+bM^*} - \mu - p$, $\mathscr{G}_{12} = \dfrac{-\beta S^*}{1+bM^*}$, $\mathscr{G}_{13} = \dfrac{b\beta S^* I^*}{(1+bM^*)^2}$,
$\mathscr{G}_{21} = -\mu - \dfrac{\beta(1-\sigma)I^*}{1+bM^*}$, $\mathscr{G}_{22} = \dfrac{-\beta(1-\sigma)V^*}{1+bM^*}$, $\mathscr{G}_{23} = \dfrac{b\beta(1-\sigma)V^* I^*}{(1+bM^*)^2}$,
$\mathscr{G}_{31} = \dfrac{\beta I^*}{1+bM^*}$, $\mathscr{G}_{32} = \dfrac{\beta(1-\sigma)I^*}{1+bM^*}$, $\mathscr{G}_{33} = \dfrac{\beta(S^*+(1-\sigma)V^*)}{1+bM^*} - (\mu+\mu_1) = 0$,
$\mathscr{G}_{34} = \dfrac{-b\beta(S^*+(1-\sigma)V^*)I^*}{(1+bM^*)^2}$.

The following below is the characteristic equation of the above jacobian matrix:

$$\Delta^6 + \mathscr{D}_5\Delta^5 + \mathscr{D}_4\Delta^4 + \mathscr{D}_3\Delta^3 + \mathscr{D}_2\Delta^2 + \mathscr{D}_1\Delta + \mathscr{D}_0 = 0, \tag{20}$$

where, the values of $\mathscr{D}_i$, $i = 0,1,2,3,4,5$ are given in Appendix A.

The negativity of the real parts of the roots of the equation (20) is guaranteed if $|H_n^{'}| > 0$ for

$n = 1, \ldots, 6$, where for each n, $|H_n^{'}|$ is of the form [38]:

$$H_n^{'} = \begin{bmatrix} \mathscr{D}_5 & 1 & 0 & 0 & 0 & 0 \\ \mathscr{D}_3 & \mathscr{D}_4 & \mathscr{D}_5 & 1 & 0 & 0 \\ \mathscr{D}_1 & \mathscr{D}_2 & \mathscr{D}_3 & \ldots & \ldots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & \ldots & \ldots & \ldots & \mathscr{D}_{6-n} \end{bmatrix} \tag{21}$$

□

3.4. Global Stability of equilibrium points of the system (7). Due to complexity of the system (7), we assume M to take any fixed value between 0 and 1.

Theorem 3.4. *Let $M = M^*$. When $R_{vac} < 1$, then the disease free equilibrium point P_0 of the reduced system from (7) is globally asymptotically stable.*

Proof. When $M = M^*$, then the third equation of system (7) implies that

$$\frac{dI}{dt} \leq \frac{\beta(S_0 + (1-\sigma)V_0)I}{(1+bM^*)} - (\mu_1 + \mu)I. \tag{22}$$

$$\text{Let } H_* = \frac{\beta(S_0 + (1-\sigma)V_0)}{(1+bM^*)}, G_* = (\mu_1 + \mu). \tag{23}$$

Then the system (22) yields $\frac{dI}{dt} \leq (H_* - G_*)I$.

There exists a positive left eigenvector u of the positive matrix $G_*^{-1}H_*$, with respect to the eigenvalue $R_0 = \rho(H_* {G_*}^{-1}) = \rho({G^{-1}}_* H_*)$, by the Perron-Frobenius theorem. Define Lyapunov function:

$$L = u^T G_*^{-1} I.$$

Note that $L \geq 0$ and $L = 0$ iff $I = 0$. Then,

$$L' = u^T G_*^{-1} \frac{dI}{dt} \leq u^T G_*^{-1}(G_* - H_*)I = (R_{vac} - 1)u^T I.$$

If $R_{vac} < 1$, then $L' \leq 0$. Furthermore, $L' = 0$ only if $I = 0$. Hence, by La Salle's Invariance Principle the disease free equilibrium point is globally asymtotically stable.

□

Theorem 3.5. *Let $M = M^*$. If the hypothesis 5 holds true, then the endemic equilibrium point P^* of the system (7) is globally asymptotically stable.*

Proof. Consider the following Lyapunov function:

$$F_1(S,V,I,R,K) = (S-S^*)^2 + (V-V^*)^2 + (I-I^*)^2 + (R-R^*)^2 + (K-K^*)^2. \tag{24}$$

It can be seen that $F_1(S^*,V^*,I^*,R^*,K^*) = 0$ and it is positive otherwise. Taking the derivative of the equation (24) along the solution of the system (7), we get

$$\begin{aligned}\frac{dF_1}{dt} &< -\left(\mu + \frac{p}{2} - \frac{\beta\lambda}{\mu(1+bM^*)} - \frac{C_S}{2}\right)(S-S^*)^2 - \left(\mu - \frac{\mu_1}{2} - \frac{C_R}{2}\right)(R-R^*)^2 \\ &- \left(\mu + \frac{\mu_1}{2} - \frac{2\beta\lambda}{\mu(1+bM^*)} - \frac{2(1-\sigma)\beta\lambda}{\mu(1+bM^*)}\right)(I-I^*)^2 \\ &- \left(\mu - \frac{p}{2} - \frac{(1-\sigma)\beta\lambda}{\mu(1+bM^*)} - \frac{C_V}{2}\right)(V-V^*)^2 \\ &- \left(d - \frac{C_S}{2} - \frac{C_V}{2} - \frac{C_R}{2}\right)(K-K^*)^2.\end{aligned}$$

Hypothesis 5. $\frac{\beta\lambda}{\mu(1+bM^*)} + \frac{C_S}{2} < \mu + \frac{p}{2}$, $\frac{\mu_1}{2} + \frac{C_R}{2} < \mu$, $\frac{C_S}{2} + \frac{C_V}{2} + \frac{C_R}{2} < d$, $\frac{2\beta\lambda}{\mu(1+bM^*)} + \frac{2(1-\sigma)\beta\lambda}{\mu(1+bM^*)} < \mu + \frac{\mu_1}{2}$, *and* $\frac{p}{2} + \frac{(1-\sigma)\beta\lambda}{\mu(1+bM^*)} + \frac{C_V}{2} < \mu$.

If hypothesis 5 holds true, then by LaSalles's Invariance Principle [39], the endemic equilibrium point P^* of the system (7) is globally asymptotically stable. □

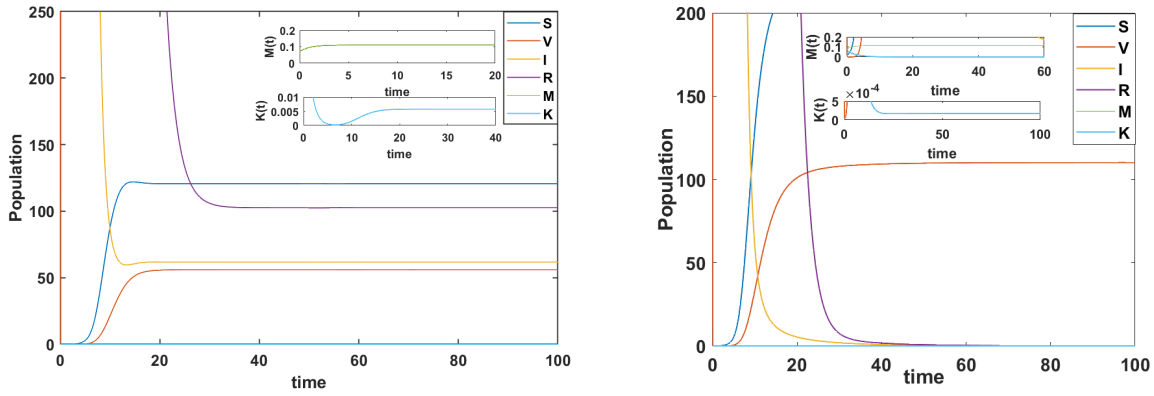
4. SIMULATION RESULTS

Numerical simulations are conducted in this section to verify the theoretical results and to gain a better understanding of the system dynamics. Table 2 provides the initial values of all the variables and values of all the parameters that were taken into consideration for disease free and endemic equilibrium points.

Parameter/ Variable	Disease Free Equilibrium Point	Endemic Equilibrium Point
β	$0.48 * 10^{-2}$	$0.98 * 10^{-2}$
λ	150	150
σ	0.98	0.98
μ_1	0.731	0.731
μ	$4.4 * 10^{-1}$	$4.4 * 10^{-1}$
m	$3.6 * 10^{-13}$	$3.6 * 10^{-13}$
p_1	0.99	0.99
f_0	$0.01 * 10^{-5}$	$0.01 * 10^{-7}$
p	$2.1 * 10^{-1}$	$2.1 * 10^{-1}$
δ	0.11	0.11
C_S	$1.08 * 10^{-10}$	$1.08 * 10^{-10}$
C_V	$1.02 * 10^{-6}$	$1.02 * 10^{-4}$
C_R	$1.98 * 10^{-8}$	$1.98 * 10^{-10}$
b	0.17	0.17
d	0.66	0.99
g_0	$0.13 * 10^{-6}$	$0.13 * 10^{-8}$
$S(0)$	1829000	1829000
$V(0)$	1042	1042
$I(0)$	1083	1083
$R(0)$	1051	1051
$M(0)$	0.07	0.07
$K(0)$	0.07	0.07

TABLE 2. Values of parameters and initial values of all the variable classes.

Existence of Disease free and Endemic Equilibrium points: If the values of parameters are as in the third column of Table 2, then from Figure 1a the existence of the endemic equilibrium point $(120.6256, 56.0589, 61.7068, 102.5175, 0.1111, 0.0057)$ of the system (7) and stability of all the classes can be inferred. Whereas, if the values of the parameters are taken as in the second column of Table 2, then from Figure 1b the existence of disease free equilibrium point $(230.7692, 110.1398, 0.0000, 0.0000, 0.1111, 0.0002)$ of system (7) is established. From Figure 1b, it can be inferred that the susceptible, vaccination, mitigation, and economic development stabilises in the longer run.

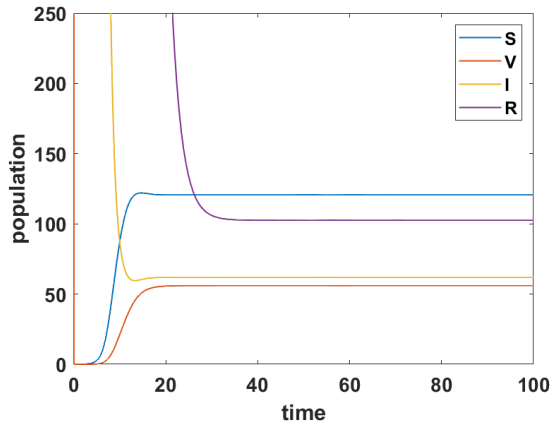


(A) Endemic equilibrium point.

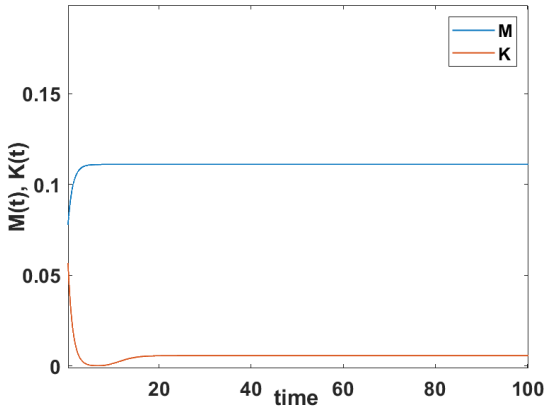
(B) Disease Free equilibrium point.

FIGURE 1. Solution of system (7).

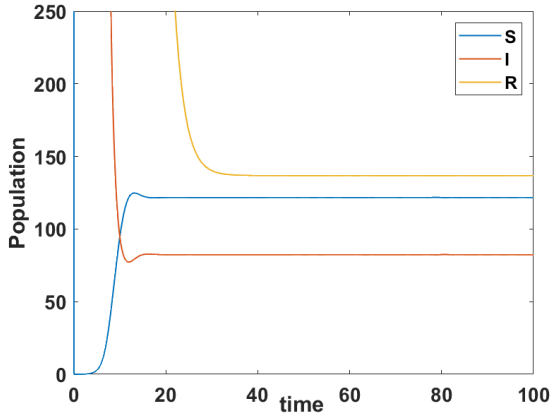
We examine the subsystem (*SIRMK*) of system (7), which does not take vaccination into account, and compare the results with those of system (7), in order to investigate the impact of vaccination on disease propagation. As can be seen from Figure 2c, the equilibrium level of the infected class (82.3496) rises when vaccination is not administered, as opposed to the situation where vaccination is taken into consideration (Figure 2a). This is because weakened immunity leads to a rise in the frequency of cases of people becoming infected after interacting with infected individuals when vaccinations are not available in society. As demonstrated in Figure 2d, in the absence of vaccination, there will also be a decline in financial growth in comparison with the case when vaccination is considered (Figure 2b). This reduction can be attributed to the increase in the number of individuals falling into the afflicted class as well as the enforcement of a lockdown-related market closure.



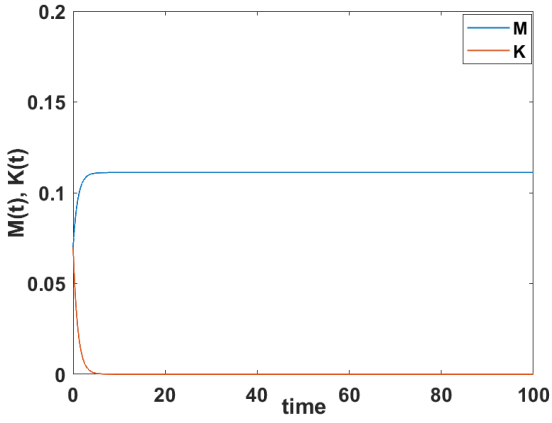
(A) Solution of S, V, I, and R classes of system (7).



(B) Solution of M, and K classes of system (7).



(C) Solution of S, I, and R classes of subsystem ($SIRMK$) of system (7), when vaccination is not considered.

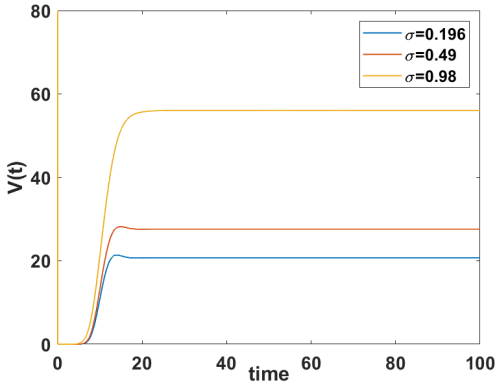


(D) Solution of M, and K classes of subsystem ($SIRMK$) of system (7), when vaccination is not considered.

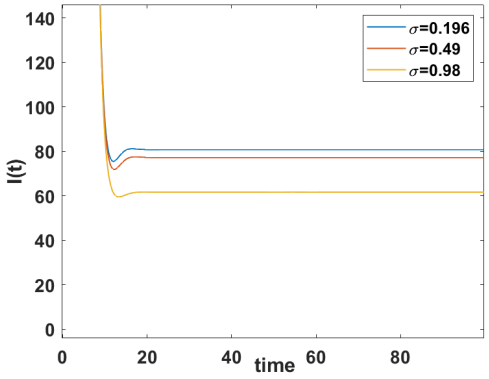
FIGURE 2. System (7) solution in two scenarios: with and without vaccination.

4.1. Effect of change in certain parameters on different classes of system (7). In order to measure how the model (7) variables change as the values of model (7) parameters $(\beta, \sigma, p, \delta)$ change, we run a thorough simulation. We use the simulation result as shown in Figure 2 as the basis scenario and the parameter values given in the third column of Table 2 as their baseline values.

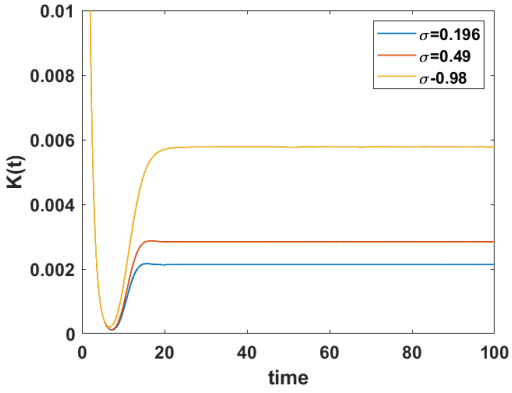
(i) We determine the impact of vaccine efficacy σ on different classes of the system (7). Figure 3 displays the curve of V, I and K by keeping the values for other parameters same as in the third column of Table 2. Reduction in the value of σ substantially effects the number of vaccinated (V), and infected (I) individuals. Decrease in σ also effects the economic development curve (K). It is evident from Figure 3a that the number of vaccinated individuals increases by increasing σ. On a contrary the count of infected decreases as σ increases (Figure 3b). Figure 3c clearly demonstrates that as σ increases, economic development increases too. This could be because fewer workers are lost from the labour market as more people become vaccinated and therefore more individuals are able to fight off the illness.



(A) Solution of V class of system (7).



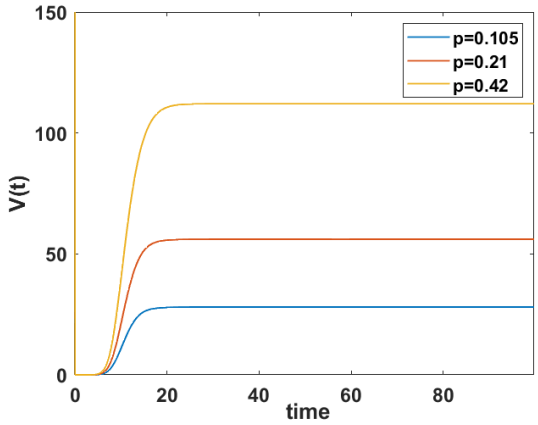
(B) Solution of I class of system (7).



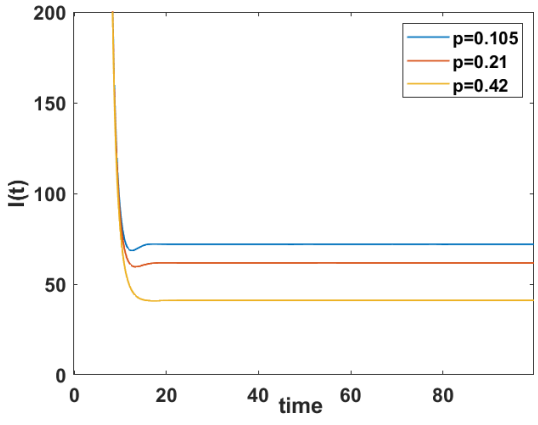
(C) Solution of K class of system (7).

FIGURE 3. Solution of system (7) for σ=0.196, 0.49, 0.98

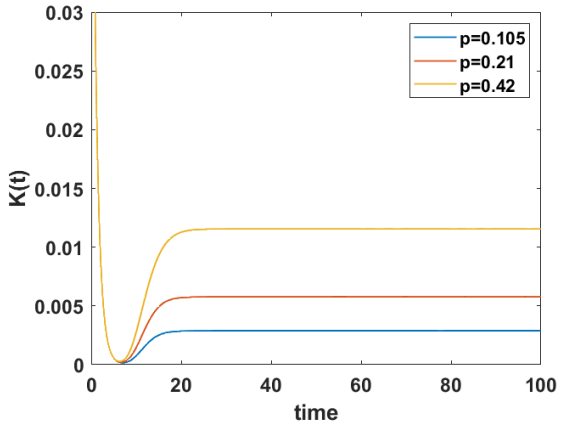
(ii) We analyse the impact of vaccination rate p on various classes of system (7). When p is increased two times its base value, it can be concluded that there is a significant increase in the equilibrium values of V and K, according to Figures 4a and 4c. The correlation between higher vaccination rates and economic growth could potentially stem from the fact that a lower labour shortage, as more individuals receive vaccinations, will lead to greater economic growth. On the other hand, Figure 4b depicts a decline in the equilibrium value of I as the value of p increases.



(A) Solution of V class of system (7).



(B) Solution of I class of system (7).

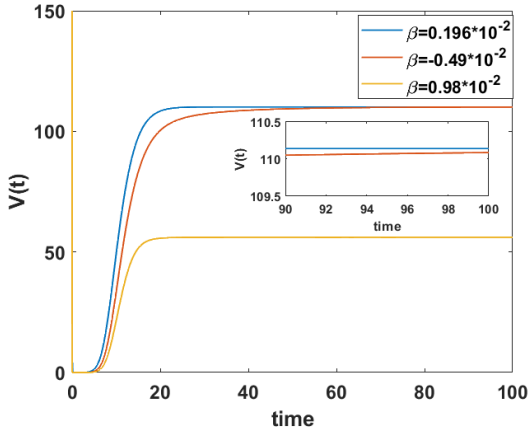


(C) Solution of K class of system (7).

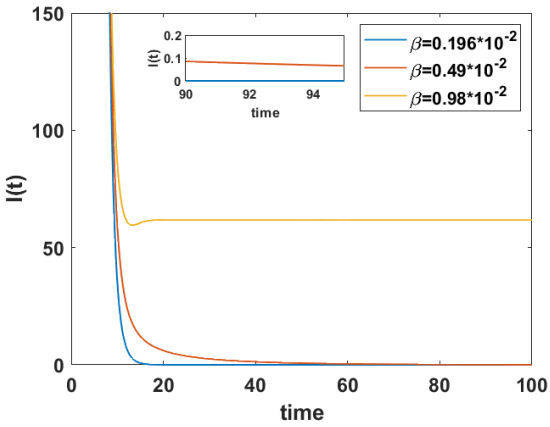
FIGURE 4. Solution of system (7) for $p = 0.105, 0.21, 0.42$.

(iii) The impact of transmission rate (β) on the different classes of system (7) is represented in Figure 5. With an increase in β, there is a decrease in the number of vaccinated

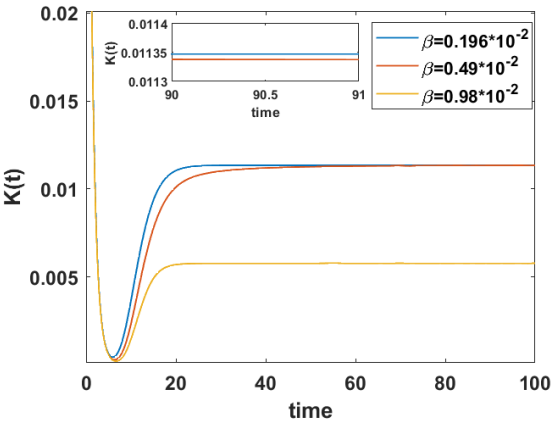
individuals (Figure 5a). Also, Figure 5c shows that the economic development decreases with an increase in β. This may be because when β increases, there is an increase in the number of infectives (Figure 5b), which perhaps leads to labor shortage.



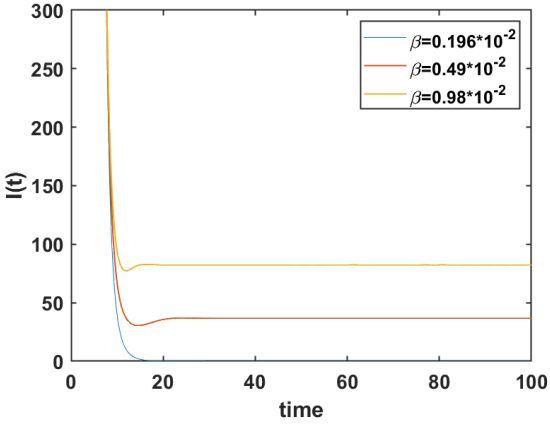
(A) Solution of V class of system (7).



(B) Solution of I class of system (7).



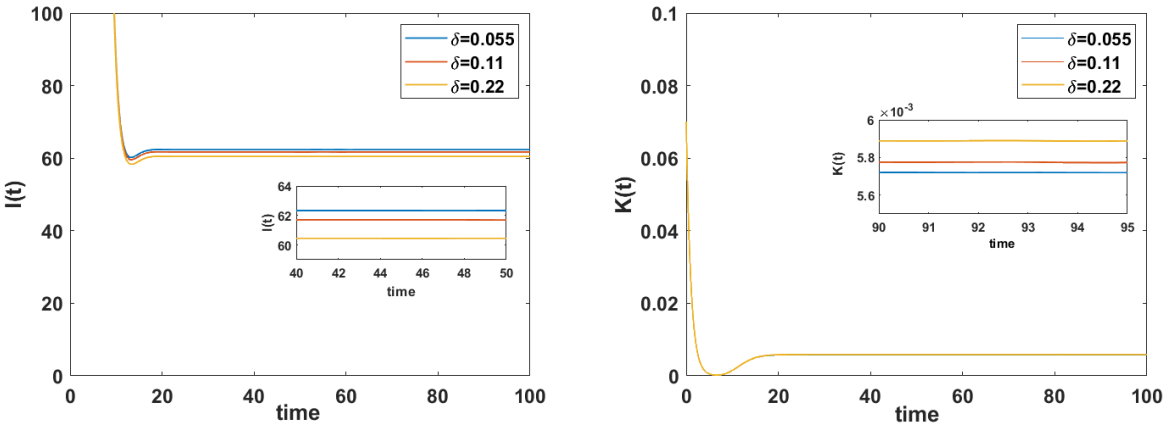
(C) Solution of K class of system (7).



(D) Solution of I class of ($SIRMK$)

FIGURE 5. Solution of system (7) for β=$0.196*10^{-2}$, $0.49*10^{-2}$, $0.98*10^{-2}$

(iv) As shown in Figures 5b and 5d that when parameter β increases then the number of infectives increase. This increase is significant in case there is no vaccination (Figure 5d) in comparison with the case when vaccination is considered (Figure 5b). Thus, it can be easily said that vaccination plays vital role in controlling the disease.



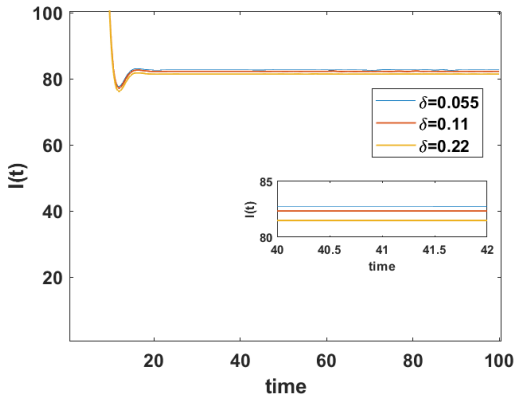
(A) Solution of I class of system (7). (B) Solution of K class of system (7).

FIGURE 6. Solution of system (7) for δ=0.055, 0.11, 0.22

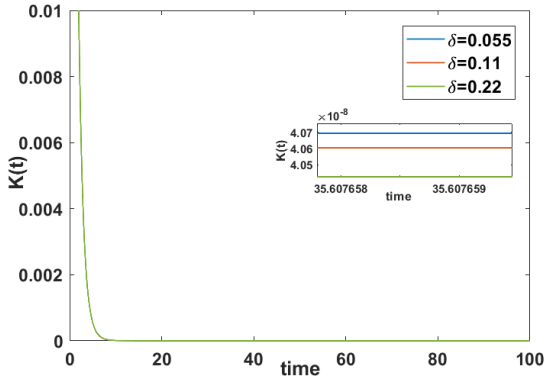
(v) In Figure 6 curves of I and K for the system (7) have been plotted with respect disease mitigation influx rate (δ). It can also be observed from Figure 6a that the count of infected decreases with an increase in influx rate of mitigation (δ). Figure 6b represents a slight growth in economic development level with an increase in δ.

(vi) We have plotted the solution of I and K of subsystem $SIRMK$ of system (7) with respect to the parameter δ in Figure 7. It can be seen that the economic development level K decreases with an increase in δ (Figure 7b). Also, there is a depreciation in the equilibrium levels of I with increase in the value of δ (Figure 7a).

Thus, it may be inferred from points (v) and (vi) that, in the event that vaccinations are administered, a certain amount of mitigation may actually be beneficial to the country's economic well-being. On the other hand, not providing immunisations can make even a little amount of mitigation perilous for the economy.



(A) Solution of I class of subsystem $(SIRMK)$ of system (7).



(B) Solution of K class of subsystem $(SIRMK)$ of system (7).

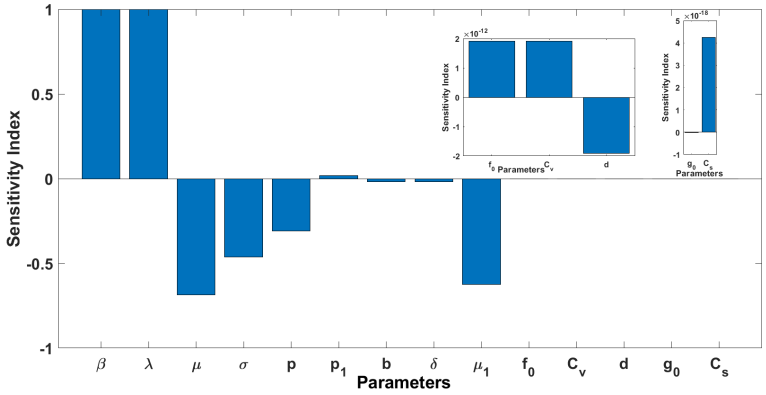
FIGURE 7. Solution of subsystem $(SIRMK)$ of system (7) for δ=0.055, 0.11, 0.22

4.2. Sensitivity Analysis. Since R_{vac} is the most crucial tool in epidemiological modelling, therefore, to study the behaviour of model parameters affecting the dynamics of disease, we undertake the sensitivity analysis of the reproduction number (R_{vac}) with respect to the changes in parameters of the system (7), by using the below formula [40],

$$\rho_{R_{vac}}^{Y} = \frac{\partial R_{vac}}{\partial Y}\frac{Y}{R_{vac}}.$$

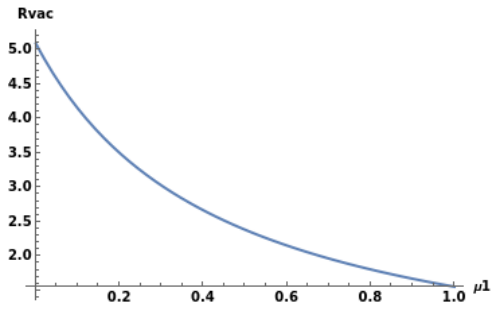
Sensitivity indices of the parameters of basic reproduction number R_{vac} are listed in Table 3 and its graphical representation is shown in form of bar graph in Figure 8. In Figure 8 for the parameters with the bar pointing above the x-axis, there will be an increase in the value of R_{vac} when the parameters' value increases, and for the bars pointing downwards below the x-axis, there will be a decrease in the value of R_{vac} with an increase in the value of such parameters. Figure 8 illustrates that while parameters $\sigma,\ p,\ \delta,\ b,\ \mu_1$ have negative influence on R_{vac}, the parameters β, λ and p_1 have positive effect on R_{vac}. Further, Figure 9 displays the nature of curve of R_{vac} with respect to changes in the parameters $\sigma,\ p,\ \delta,\ b,\ \mu_1,\ \beta,\ \lambda$ and p_1. From Figures 9a, 9b, 9c, 9d, and 9e it becomes evident that R_{vac} decreases with an increase in the value of $\mu_1,\ p,\ \sigma,\ \delta$, and b, respectively. Also, it is clear from Figures 9f and 9g that R_{vac} increases with an increase in the value of λ and β. Lastly, from Figure 9h, it can be observed that there

is a gradual increase in R_{vac} with an increase in p_1. As evident from Figures 9f and 9g that β and λ strongly influence the reproduction number R_{vac} and contribute to spreading the disease, therefore, it becomes crucial to reduce the value of these parameters. To reduce the value of λ, lockdowns can be imposed. And the value β can be reduced significantly by sensitizing people to wear masks, maintaining social distancing, and isolating the infected. Additionally, Figures 9c, 9d, and 9e demonstrate how strongly and negatively the parameters σ, δ, and b affect the reproduction number. Therefore, implementing mitigation strategies and providing timely vaccinations can help in reducing the disease spread. It is also equally important to note that we have no influence over some parameters and cannot alter them in order to lower down R_{vac} whereas understanding the dependence can nonetheless empower us to make proactive choices.

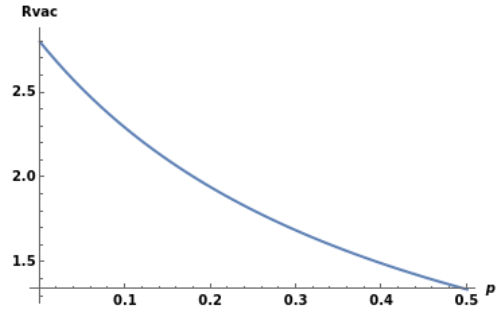


(A) Bar Graph

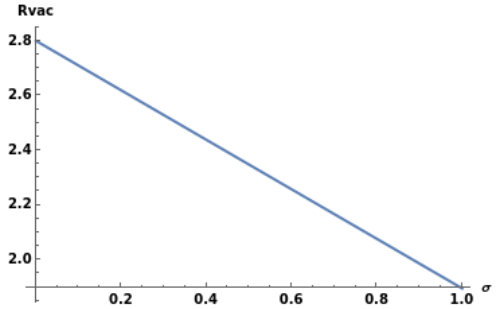
FIGURE 8. Sensitivity analysis with the bar graph



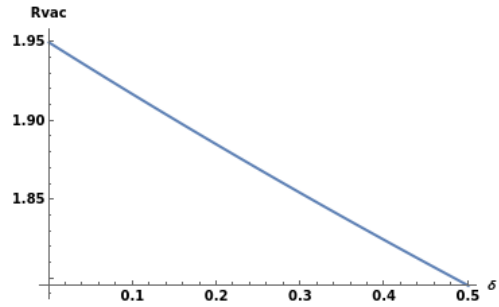
(A) R_{vac} and μ_1



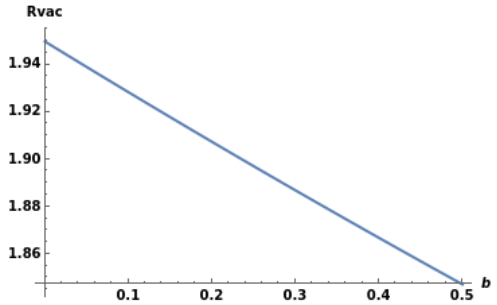
(B) R_{vac} and p



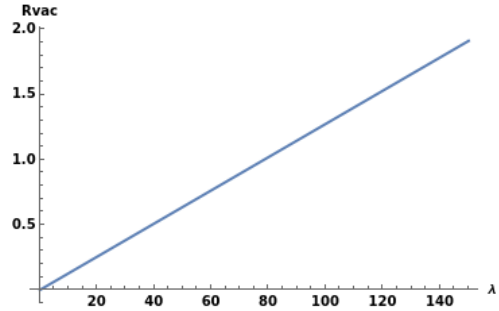
(C) R_{vac} and σ



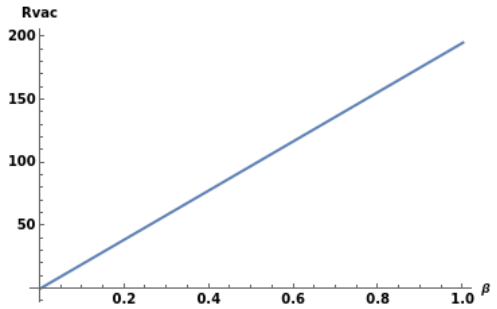
(D) R_{vac} and δ



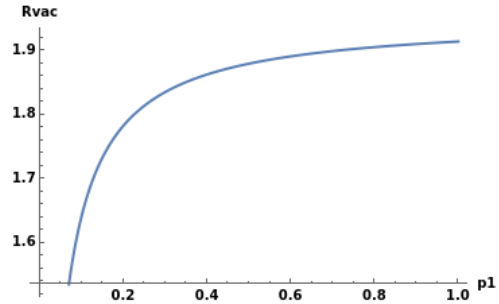
(E) R_{vac} and b



(F) R_{vac} and λ



(G) R_{vac} and β



(H) R_{vac} and p_1

FIGURE 9. Variations of R_{vac} with respect to the parameters μ_1, p, σ, δ, b, ,λ, β, p_1.

Parameter	Sensitivity
p	-0.3076322923003776
p_1	0.018538713193455878
μ_1	-0.624252775405636
σ	-0.4633048176431585
β	1.000
b	-0.01853871319345589
λ	1.0000
δ	-0.01853871319523472
μ	-0.6863782772862586
f_0	$1.912481999729719 * 10^{-12}$
C_v	$1.912477781511529 * 10^{-12}$
d	$-1.9124555624841357 * 10^{-12}$
g_0	$-2.458966141362373 * 10^{-20}$
C_s	$4.242807851420535 * 10^{-18}$

TABLE 3. Parameters Sensitivity for R_{vac}

5. Discussion and Conclusion

Epidemics are a global health problem because they have severe detrimental effects on public health and the economy. In this study, we have constructed and investigated an epidemic-economic *SVIRMK* model. We have obtained boundedness and the non-negativity of the solutions of the system (7). In addition, we evaluated the two equilibrium points of the system (7): the disease free equilibrium point $P_0 = (S_0, V_0, I_0, R_0, M_0, K_0)$ and the endemic equilibrium point $P^* = (S^*, V^*, I^*, R^*, M^*, K^*)$. We have derived the basic reproduction number and have discussed the stability of the disease free equilibrium point P_0 of the system (7). Also, it has been shown that if $R_{vac} < 1$, then the disease free equilibrium point P_0 of the reduced system (7) is globally asymptotically stable. Conditions for local and global stability of the endemic equilibrium point P^* of system (7) have been obtained. Numerical simulations have been performed using MATLAB software to validate our analytical results. We have examined the effects of certain parameters $(\beta, \sigma, p, \delta)$ on the model output. Our analysis shows that increase in the value of parameter β results in significant rise in the number of infectives when vaccination is not available (Figure 5d) compared to the case when vaccination is available (Figure 5b), demonstrating the importance of vaccination in disease control. Furthermore, we may deduce from Figures 6b and 7b that, in the event that vaccinations are administered, a certain degree of mitigation may actually be beneficial to the country's economy and reduce illness as well. However, even modest mitigation measures to manage the disease may be compromised if vaccinations are not administered.

APPENDIX A

$$\mathscr{D}_5 = -\mathscr{G}_{11} - \mathscr{G}_{21} + d + p_1 + \mu,$$

$$\begin{aligned}\mathscr{D}_4 &= \mathscr{G}_{11}\mathscr{G}_{21} - \mathscr{G}_{12}\mathscr{G}_{31} - \mathscr{G}_{22}\mathscr{G}_{32} - d\mathscr{G}_{11} - d\mathscr{G}_{21} - f_0 g_0 - m\mathscr{G}_{23} - m\mathscr{G}_{34} - \mathscr{G}_{11}p_1 - \mathscr{G}_{21}p_1 \\ &+ dp_1 - \mu\mathscr{G}_{11} - \mu\mathscr{G}_{21} + \mu d + \mu p_1 + m\sigma\mathscr{G}_{23},\end{aligned}$$

$$\begin{aligned}\mathscr{D}_3 &= \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31} + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32} + \mathscr{G}_{11}\mathscr{G}_{21}d - \mathscr{G}_{12}\mathscr{G}_{31}d - \mathscr{G}_{22}\mathscr{G}_{32}d + \mathscr{G}_{13}C_S f_0 + \mathscr{G}_{23}C_V f_0 \\ &+ \mathscr{G}_{11}f_0 g_0 + \mathscr{G}_{21}f_0 g_0 + \mathscr{G}_{11}\mathscr{G}_{23}m - \mathscr{G}_{13}\mathscr{G}_{31}m - \mathscr{G}_{23}\mathscr{G}_{32}m + \mathscr{G}_{11}\mathscr{G}_{34}m + \mathscr{G}_{21}\mathscr{G}_{34}m \\ &- \mathscr{G}_{22}\mathscr{G}_{34}m - \mathscr{G}_{23}dm - \mathscr{G}_{34}dm - \mathscr{G}_{12}\mathscr{G}_{32}p - \mathscr{G}_{13}mp + \mathscr{G}_{11}\mathscr{G}_{21}p_1 - \mathscr{G}_{12}\mathscr{G}_{31}p_1 - \mathscr{G}_{22}\mathscr{G}_{32}p_1 \\ &- \mathscr{G}_{11}dp_1 - \mathscr{G}_{21}dp_1 + \mathscr{G}_{11}\mathscr{G}_{21}\mu - \mathscr{G}_{12}\mathscr{G}_{31}\mu - \mathscr{G}_{22}\mathscr{G}_{32}\mu - \mathscr{G}_{11}d\mu - \mathscr{G}_{21}d\mu - f_0 g_0\mu - \mathscr{G}_{23}m\mu \\ &- \mathscr{G}_{34}m\mu - \mathscr{G}_{11}p_1\mu - \mathscr{G}_{21}p_1\mu + dp_1\mu - \mathscr{G}_{11}\mathscr{G}_{23}m\sigma + \mathscr{G}_{22}\mathscr{G}_{34}m\sigma + \mathscr{G}_{23}dm\sigma + \mathscr{G}_{13}mp\sigma \\ &+ \mathscr{G}_{23}m\mu\sigma,\end{aligned}$$

$$\begin{aligned}\mathscr{D}_2 &= \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}d + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}d - \mathscr{G}_{13}\mathscr{G}_{21}C_S f_0 + \mathscr{G}_{12}\mathscr{G}_{34}C_S f_0 - \mathscr{G}_{11}\mathscr{G}_{23}C_V f_0 + \mathscr{G}_{22}\mathscr{G}_{34}C_V f_0 \\ &- \mathscr{G}_{11}\mathscr{G}_{21}f_0 g_0 + \mathscr{G}_{12}\mathscr{G}_{31}f_0 g_0 + \mathscr{G}_{22}\mathscr{G}_{32}f_0 g_0 + \mathscr{G}_{13}\mathscr{G}_{21}\mathscr{G}_{31}m - \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}m + \mathscr{G}_{12}\mathscr{G}_{23}a\mathscr{G}_{31}m \\ &+ \mathscr{G}_{11}\mathscr{G}_{23}\mathscr{G}_{32}m - \mathscr{G}_{11}\mathscr{G}_{21}\mathscr{G}_{34}m + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}m + \mathscr{G}_{11}\mathscr{G}_{23}dm - \mathscr{G}_{13}\mathscr{G}_{31}dm - \mathscr{G}_{23}\mathscr{G}_{32}dm \\ &+ \mathscr{G}_{11}\mathscr{G}_{34}dm + \mathscr{G}_{21}\mathscr{G}_{34}dm - \mathscr{G}_{22}\mathscr{G}_{34}dm - \mathscr{G}_{12}\mathscr{G}_{32}dp + \mathscr{G}_{13}C_V f_0 p - \mathscr{G}_{13}\mathscr{G}_{32}mp - \mathscr{G}_{12}\mathscr{G}_{34}mp \\ &- \mathscr{G}_{13}dmp + \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}p_1 + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}p_1 + \mathscr{G}_{11}\mathscr{G}_{21}dp_1 - \mathscr{G}_{12}\mathscr{G}_{31}dp_1 - \mathscr{G}_{22}\mathscr{G}_{32}dp_1 - \\ &\mathscr{G}_{12}\mathscr{G}_{32}pp_1 + \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}\mu + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}\mu + \mathscr{G}_{11}\mathscr{G}_{21}d\mu - \mathscr{G}_{12}\mathscr{G}_{31}d\mu - \mathscr{G}_{22}\mathscr{G}_{32}d\mu + \\ &\mathscr{G}_{13}C_S f_0\mu + \mathscr{G}_{23}C_v f_0\mu + \mathscr{G}_{11}f_0 g_0\mu + \mathscr{G}_{21}f_0 g_0\mu + \mathscr{G}_{11}\mathscr{G}_{23}m\mu - \mathscr{G}_{13}\mathscr{G}_{31}m\mu - \mathscr{G}_{23}\mathscr{G}_{32}m\mu + \\ &\mathscr{G}_{11}\mathscr{G}_{34}m\mu + \mathscr{G}_{21}\mathscr{G}_{34}m\mu - \mathscr{G}_{22}\mathscr{G}_{34}m\mu - \mathscr{G}_{23}dm\mu - \mathscr{G}_{34}dm\mu - \mathscr{G}_{12}\mathscr{G}_{32}p\mu - \mathscr{G}_{13}mp\mu + \\ &\mathscr{G}_{11}\mathscr{G}_{21}p_1\mu - \mathscr{G}_{12}\mathscr{G}_{31}p_1\mu - \mathscr{G}_{22}\mathscr{G}_{32}p_1\mu - \mathscr{G}_{11}dp_1\mu - \mathscr{G}_{21}dp_1\mu + \mathscr{G}_{34}C_R f_0\mu_1 + \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}m\sigma \\ &- \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}m\sigma - \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}m\sigma - \mathscr{G}_{11}\mathscr{G}_{23}dm\sigma + \mathscr{G}_{22}\mathscr{G}_{34}dm\sigma + \mathscr{G}_{12}\mathscr{G}_{34}mp\sigma + \\ &\mathscr{G}_{13}dmp\sigma - \mathscr{G}_{11}\mathscr{G}_{23}m\mu\sigma + \mathscr{G}_{22}\mathscr{G}_{34}m\mu\sigma + \mathscr{G}_{23}dm\mu\sigma + \mathscr{G}_{13}mp\mu\sigma,\end{aligned}$$

$$\begin{aligned}\mathscr{D}_1 &= -\mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{32}C_S f_0 + \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{32}C_S f_0 - \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{34}C_S f_0 + \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}C_V f_0 - \\ &\mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}C_V f_0 - \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}C_V f_0 - \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}f_0 g_0 - \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}f_0 g_0 + \mathscr{G}_{13}\mathscr{G}_{21}\mathscr{G}_{31}dm \\ &- \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}dm + \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}dm + \mathscr{G}_{11}\mathscr{G}_{23}\mathscr{G}_{32}dm - \mathscr{G}_{11}\mathscr{G}_{21}\mathscr{G}_{34}dm + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}dm + \\ &\mathscr{G}_{12}\mathscr{G}_{34}C_V f_0 p + \mathscr{G}_{12}\mathscr{G}_{32}f_0 g_0 p - \mathscr{G}_{13}\mathscr{G}_{32}dmp - \mathscr{G}_{12}\mathscr{G}_{34}dmp + \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}dp_1 + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}dp_1 \\ &- \mathscr{G}_{12}\mathscr{G}_{32}dpp_1 + \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}d\mu + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}d\mu - \mathscr{G}_{13}\mathscr{G}_{21}C_S f_0\mu + \mathscr{G}_{12}\mathscr{G}_{34}C_S f_0\mu -\end{aligned}$$

$$
\begin{aligned}
&\mathscr{G}_{11}\mathscr{G}_{23}C_V f_0\mu + \mathscr{G}_{22}\mathscr{G}_{34}C_V f_0\mu - \mathscr{G}_{11}\mathscr{G}_{21}f_0 g_0\mu + \mathscr{G}_{12}\mathscr{G}_{31}f_0 g_0\mu + \mathscr{G}_{22}\mathscr{G}_{32}f_0 g_0\mu + \\
&\mathscr{G}_{13}\mathscr{G}_{21}\mathscr{G}_{31}m\mu - \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}m\mu + \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}m\mu + \mathscr{G}_{11}\mathscr{G}_{23}\mathscr{G}_{32}m\mu - \mathscr{G}_{11}\mathscr{G}_{21}\mathscr{G}_{34}m\mu + \\
&\mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}m\mu + \mathscr{G}_{11}\mathscr{G}_{23}dm\mu - \mathscr{G}_{13}\mathscr{G}_{31}dm\mu - \mathscr{G}_{23}\mathscr{G}_{32}dm\mu + \mathscr{G}_{11}\mathscr{G}_{34}dm\mu + \mathscr{G}_{21}\mathscr{G}_{34}dm\mu - \\
&\mathscr{G}_{22}\mathscr{G}_{34}dm\mu - \mathscr{G}_{12}\mathscr{G}_{32}dp\mu + \mathscr{G}_{13}C_V f_0 p\mu - \mathscr{G}_{13}\mathscr{G}_{32}mp\mu - \mathscr{G}_{12}\mathscr{G}_{34}mp\mu - \mathscr{G}_{13}dmp\mu + \\
&\mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}p_1\mu + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}p_1\mu + \mathscr{G}_{11}\mathscr{G}_{21}dp_1\mu - \mathscr{G}_{12}\mathscr{G}_{31}dp_1\mu - \mathscr{G}_{22}\mathscr{G}_{32}dp_1\mu - \mathscr{G}_{12}\mathscr{G}_{32}pp_1\mu \\
&+\mathscr{G}_{13}\mathscr{G}_{31}C_R f_0\mu_1 + \mathscr{G}_{23}\mathscr{G}_{32}C_R f_0\mu_1 - \mathscr{G}_{11}\mathscr{G}_{34}C_R f_0\mu_1 - \mathscr{G}_{21}\mathscr{G}_{34}C_R f_0\mu_1 + \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}dm\sigma \\
&-\mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}dm\sigma - \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}dm\sigma + \mathscr{G}_{12}\mathscr{G}_{34}dmp\sigma + \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}m\mu\sigma - \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}m\mu\sigma \\
&-\mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}m\mu\sigma - \mathscr{G}_{11}\mathscr{G}_{23}dm\mu\sigma + \mathscr{G}_{22}\mathscr{G}_{34}dm\mu\sigma + \mathscr{G}_{12}\mathscr{G}_{34}mp\mu\sigma + \mathscr{G}_{13}dmp\mu\sigma,
\end{aligned}
$$

$$
\begin{aligned}
\mathscr{D}_0 \quad &= -\mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{32}C_S f_0\mu + \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{32}C_S f_0\mu - \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{34}C_S f_0\mu + \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}C_v f_0\mu - \\
&\mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}C_V f_0\mu - \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}C_V f_0\mu - \mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}f_0 g_0\mu - \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}f_0 g_0\mu + \\
&\mathscr{G}_{13}\mathscr{G}_{21}\mathscr{G}_{31}dm\mu - \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}dm\mu + \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}dm\mu + \mathscr{G}_{11}\mathscr{G}_{23}\mathscr{G}_{32}dm\mu - \mathscr{G}_{11}\mathscr{G}_{21}\mathscr{G}_{34}dm\mu \\
&+\mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}dm\mu + \mathscr{G}_{12}\mathscr{G}_{34}C_V f_0 p\mu + \mathscr{G}_{12}\mathscr{G}_{32}f_0 g_0 p\mu - \mathscr{G}_{13}\mathscr{G}_{32}dmp\mu - \mathscr{G}_{12}\mathscr{G}_{34}dmp\mu + \\
&\mathscr{G}_{12}\mathscr{G}_{21}\mathscr{G}_{31}dp_1\mu + \mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{32}dp_1\mu - \mathscr{G}_{12}\mathscr{G}_{32}dpp_1\mu - \mathscr{G}_{13}\mathscr{G}_{21}\mathscr{G}_{31}C_R f_0\mu_1 - \mathscr{G}_{11}\mathscr{G}_{23}\mathscr{G}_{32}C_R f_0\mu_1 \\
&+\mathscr{G}_{11}\mathscr{G}_{21}\mathscr{G}_{34}C_R f_0\mu_1 + \mathscr{G}_{13}\mathscr{G}_{32}C_r f_0 p\mu_1 + \mathscr{G}_{13}\mathscr{G}_{22}\mathscr{G}_{31}dm\mu\sigma - \mathscr{G}_{12}\mathscr{G}_{23}\mathscr{G}_{31}dm\mu\sigma - \\
&\mathscr{G}_{11}\mathscr{G}_{22}\mathscr{G}_{34}dm\mu\sigma + \mathscr{G}_{12}\mathscr{G}_{34}dmp\mu\sigma.
\end{aligned}
$$

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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