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MODELS OF ECONOMIC AND SOCIAL SYSTEMS

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Modeling the impact of epidemic spread and lockdown on economy

M. Saade[a]

Peoples' Friendship University of Russia (RUDN University),
6 Miklukho-Maklaya st., Moscow, 117198, Russia

E-mail: [a] 1042225269@pfur.ru

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Epidemics severely destabilize economies by reducing productivity, weakening consumer spending, and overwhelming public infrastructure, often culminating in economic recessions. The COVID-19 pandemic underscored the critical role of nonpharmaceutical interventions, such as lockdowns, in containing infectious disease transmission. This study investigates how the progression of epidemics and the implementation of lockdown policies shape the economic well-being of populations. By integrating compartmental ordinary differential equation (ODE) models, the research analyzes the interplay between epidemic dynamics and economic outcomes, particularly focusing on how varying lockdown intensities influence both disease spread and population wealth. Findings reveal that epidemics inflict significant economic damage, but timely and stringent lockdowns can mitigate healthcare system overload by sharply reducing infection peaks and delaying the epidemic's trajectory. However, carefully timed lockdown relaxation is equally vital to prevent resurgent outbreaks. The study identifies key epidemiological thresholds—such as transmission rates, recovery rates, and the basic reproduction number ($\mathfrak{R}_0$) — that determine the effectiveness of lockdowns. Analytically, it pinpoints the optimal proportion of isolated individuals required to minimize total infections in scenarios where permanent immunity is assumed. Economically, the analysis quantifies lockdown impacts by tracking population wealth, demonstrating that economic outcomes depend heavily on the fraction of isolated individuals who remain economically productive. Higher proportions of productive individuals during lockdowns correlate with better wealth retention, even under fixed epidemic conditions. These insights equip policymakers with actionable frameworks to design balanced lockdown strategies that curb disease spread while safeguarding economic stability during future health crises.

Keywords: epidemic-economic ODE model, immunity waning, lockdown, wealth

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МОДЕЛИ ЭКОНОМИЧЕСКИХ И СОЦИАЛЬНЫХ СИСТЕМ

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Моделирование влияния распространения эпидемии и карантина на экономику

М. Сааде[a]

Российский университет дружбы народов (РУДН),
Россия, 117198, г. Москва, ул. Миклухо-Маклая, д. 6

E-mail: [a] 1042225269@pfur.ru

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Эпидемии серьезно дестабилизируют экономику, снижая производительность, ослабляя потребительскую активность и перегружая общественные ресурсы, что часто приводит к экономическим кризисам. Пандемия COVID-19 продемонстрировала ключевую роль нематериальных мер, таких как карантин, в сдерживании распространения инфекционных заболеваний. Данное исследование изучает, как развитие эпидемии и введение карантинных мер влияют на экономическое благополучие населения. С помощью компартментальных моделей на основе обыкновенных дифференциальных уравнений (ОДУ) анализируется взаимосвязь между динамикой заболевания и экономическими последствиями, особенно фокусируясь на том, как различные строгости карантина воздействуют как на распространение болезни, так и на благосостояние населения. Результаты показывают, что эпидемии наносят значительный экономический ущерб, однако своевременные и строгие карантинные меры могут снизить нагрузку на систему здравоохранения, резко уменьшая пик заражений и замедляя развитие эпидемии. Тем не менее, стратегически продуманное ослабление карантина не менее важно для предотвращения повторных вспышек. Исследование выявляет ключевые эпидемиологические пороговые значения, такие как скорость передачи, уровень выздоровления и базовое репродуктивное число ($\mathfrak{R}_0$), которые определяют эффективность карантина. Аналитически определяется оптимальная доля изолированных лиц, необходимая для минимизации общего числа заражений в условиях постоянного иммунитета. С экономической точки зрения, влияние карантина оценивается через динамику благосостояния населения: показано, что экономические последствия зависят от доли изолированных, но сохраняющих экономическую активность граждан. Чем выше эта доля, тем лучше сохраняется благосостояние даже при фиксированных эпидемиологических параметрах. Эти выводы предоставляют властям практические рекомендации для разработки сбалансированных карантинных стратегий, способных сдерживать распространение болезней и одновременно защищать экономическую стабильность в будущих кризисах.

Ключевые слова: эпидемия — экономическая модель, иммунитет ослабевает, карантин, богатство

Introduction

Mathematical modeling has become an essential tool in understanding and mitigating the impact of epidemics. In epidemiology, it has been motivated by periodically emerging large-scale epidemics such as HIV from the 1980s to the present [Fisher-Hoch, Hutwagner, 1995; Chintu, Athale, Patil, 1995], SARS in 2002–2003 [Anderson et al., 2004; Lam, Zhong, Tan, 2003], H5N1 influenza in 2005 [Chen et al., 2006; Kilpatrick et al., 2006], H1N1 in 2009 [Jain et al., 2009; Girard et al., 2010], and Ebola in 2014 [Briand et al., 2014; Kreuels et al., 2014]. The recent COVID-19 pandemic had a strong influence on public health, economy, and many other aspects of societal life.

Numerous epidemic models have been introduced, influenced by the Spanish influenza outbreak of 1918–1919 and the work of Kermack and McKendrick [Kapralov, Khanna, Sudan, 2014; Almeida, Qureshi, 2019]. Among these are multi-compartment models, which serve as the foundation for contemporary epidemiological study and offer important insights into the transmission of infectious diseases. Nowadays, their uses include deciphering past outbreaks, forecasting the course of present and future illnesses [Sharma, Volpert, Banerjee, 2020; Brauer et al., 2008; Keeling, Rohani, 2011], models with a nonlinear disease transmission rate [d'Onofrio, Banerjee, Manfredi, 2020; Sun et al., 2008], multi-patch models [Bichara, Iggidr, 2018; McCormack, Allen, 2007], multi-group models that take into account the impact of population heterogeneity [Elbasha, Gumel, 2021], and epidemic models that include vaccination and other control measures [Aniţa et al., 2021; Faniran et al., 2022]. To characterize the spatial distributions of susceptible and infected individuals, spatiotemporal models take into account the random mobility of individuals within the population [Ahmed et al., 2019; Filipe, Maule, 2004]. Monographs [Martcheva, 2015; Brauer, Castillo-Chavez, Feng, 2019] and review articles [Hethcote, 2000; Hurd, Kaneene, 1993].

Classical SIR-type models serve as the basis for the development of single and multi-strain epidemic models. At any given time t, they usually assume that the number of recoveries and deaths is proportionate to the number of infected persons. To overcome these constraints, delay differential equation (DDE) models have become a potent mathematical tool. By including explicit delays, DDE models are able to more accurately depict the temporal features of both illness progression and transmission. They have been used to investigate various infectious diseases, such as influenza (single and multi-strain) and COVID-19, which can be found in [Ghosh, Volpert, Banerjee, 2022b; Saade et al., 2023; Ghosh, Volpert, Banerjee, 2022a] for single-strain epidemic models and in [Saade et al., 2024a] for both the single-strain model and the two-strain model with the presence of cross-immunity. An epidemiological delay model that describes the interaction between two viral strains with the absence of cross-immunity is proposed in [Mozokhina et al., 2024]. Systems with distributed recovery and death rates have been introduced, and it has been demonstrated that DDE models offer an appropriate approximation of distributed recovery and death rate models [Ghosh, Volpert, Banerjee, 2022a]. A delay epidemic model with vaccination was examined in [Saade, Aniţa, Volpert, 2023]. Dynamics of delay epidemic models with periodic disease transmission rates for a single strain and double strains were investigated in [Saade et al., 2024b].

Globally, the COVID-19 epidemic has drastically changed communities, causing previously unheard-of public health issues and economic upheavals. A variety of measures were taken by governments in an effort to control the infection spread, with partial lockdowns becoming a common reaction. These policies, which entail selectively restricting certain activities while permitting others to function, have proven essential in striking a balance between the two demands of economic stability and public health [Varona, Gonzales, 2021].

Partial lockdowns have been shown to significantly affect the spread of epidemics. The authors of [Hsiang et al., 2020] showed that these steps can successfully lower the epidemic progression, which will slow down the pace of transmission. However, the application of these tactics is not

without financial repercussions. According to [Loayza, 2020], countries which implemented stringent lockdowns had notable drops in GDP, job losses and a rise in poverty levels. These results underline the urgent need for a comprehensive understanding of how partial lockdowns impact both health consequences and economic vitality.

An economic-demographic dynamical system was presented in [Zincenko, Petrovskii, Volpert, 2018] to illustrate scenarios which could eventually result in a significant population decline and/or deterioration of the economy. It is also demonstrated that even in the cases when the population may become extinct, it may still experience temporary growth.

The complex interplay between partial lockdown measures, the progression of epidemics, and economic outcomes has been explored in prior research, such as the epidemic-economic model proposed in [Mozokhina et al., 2024], which employed delay differential equations (DDEs) to capture time-lagged dynamics in disease transmission. In contrast, the ordinary differential equation (ODE) framework introduced in this study replaces these temporal delays with epidemiological transition rates to model shifts between the model compartments. By adopting this approach, the proposed model explicitly links public health outcomes to economic performance, focusing on short-term impacts such as immediate changes in infection rates, workforce availability, and economic productivity. This methodological shift simplifies the analysis of how real-time policy interventions — like lockdown intensity and duration — simultaneously influence both disease suppression and economic stability. The model's compartmental ODE framework allows for a granular examination of how adjustments in lockdown policies alter infection trajectories, healthcare burdens, and economic output over weeks to months. Ultimately, this work aims to generate actionable insights for policymakers, emphasizing strategies that balance effective disease containment with measures to mitigate economic downturns. By quantifying trade-offs between public health interventions and economic resilience, the study seeks to inform evidence-based policies that are not only effective in curbing epidemic spread but also economically sustainable, ensuring that societies can recover more robustly from future health crises.

This study begins by introducing an integrated epidemic-economic model, followed by a rigorous demonstration of the existence, uniqueness, positiveness, and boundedness of its solutions. Subsequent analysis identifies the system's equilibrium states and examines their stability properties. The work then explores how epidemic dynamics can destabilize economic systems, emphasizing the interplay between public health crises and socioeconomic disruption. Building on this foundation, the influence of partial lockdown measures on disease spread is investigated under the assumption of persistent immunity (i. e., no immunity waning). A comprehensive model incorporating both productive populations and wealth dynamics is subsequently developed, with numerical simulations illustrating how critical parameters shape epidemic trajectories and economic outcomes in scenarios where immunity remains permanent. Finally, the study concludes by synthesizing key findings and outlining potential directions for future research to refine strategies for balancing public health interventions with economic sustainability.

Integrated epidemic-economic model development and analysis

We introduce a new mathematical model that combines epidemiological dynamics and economic balance. In this section, we fully analyze the mathematical aspects of the new model.

Model formulation

This study builds on the classical Susceptible-Infected-Recovered (SIR) model, a foundational epidemiological framework that tracks disease spread through transitions between susceptible, infected, and recovered populations. Besides the usual classes of susceptible individuals $S(t)$ and infectious individuals $I(t)$, we also introduce two more new classes. The first one is denoted by $R_n(t)$ and it represents the unproductive recovered individuals, that is, the individuals who have recovered from

the illness but are still not able to participate in their professional activity and wealth production, maybe due to lingering symptoms. The second new class is denoted by $R_p(t)$ and it describes the productively recovered individuals who have completely overcome the convalescence period and are able to return to their work. Susceptible individuals get infected when they come into contact with infectious individuals. Hence, new infections occur due to contact between susceptible and infectious individuals, that is $\frac{\beta}{N}S(t)I(t)$, where β is the transmission rate (the probability of infection per contact per unit of time). Infectious individuals recover at a rate ρ, defined as the inverse of the infectious period. The number of individuals transitioning from the class of infectious, $I(t)$, to the unproductive recovered class at time t is modeled by the term $\rho I(t)$. This represents the flow of people who have recovered from the infection but are no longer economically productive (e. g., due to long-term health consequences or workforce exclusion). Unproductive recovered individuals become productive with a rate μ signifying the inverse of the convalescence period (the period after which a recovered person who is not yet productive returns to produce wealth). The number of recovered individuals who have passed the convalescence period and return to the class of productive individuals is $\mu R_n(t)$. These individuals lose their immunity and return to the susceptible class at a rate γ representing waning immunity (the inverse of the immunity duration), are denoted by $\gamma R_p(t)$. Hence, the epidemic model is given by the following ordinary differential equations

$$\begin{aligned}
\frac{dS(t)}{dt} &= -\frac{\beta}{N}S(t)I(t) + \gamma R_p(t),\\
\frac{dI(t)}{dt} &= \frac{\beta}{N}S(t)I(t) - \rho I(t),\\
\frac{dR_n(t)}{dt} &= \rho I(t) - \mu R_n(t),\\
\frac{dR_p(t)}{dt} &= \mu R_n(t) - \gamma R_p(t).
\end{aligned}$$

It is assumed here that the total size of population is constant, and satisfies

$$S(t) + I(t) + R_n(t) + R_p(t) = N. \tag{1}$$

Since this work aims to study the influence of the epidemic on the economy, we introduce the class of productive individuals representing the people who contribute to the wealth production, denoted by $P(t)$. The number of productive individuals at time t is the sum of the number of susceptible individuals at time t and the number of productive recovered individuals who have passed the convalescence period at time t, that is,

$$P(t) = S(t) + R_p(t). \tag{2}$$

Hence, the rate of change of the productive class with respect to time is given by

$$\frac{dP(t)}{dt} = -\frac{\beta}{N}S(t)I(t) + \mu R_n(t).$$

The following equation for wealth $W(t)$ represents the economic part of our model:

$$\frac{dW(t)}{dt} = U(t) - C(t),$$

where the population's wealth production and consumption are denoted by $U(t)$ and $C(t)$, respectively:

$$U(t) = \alpha_1 \frac{P(t)}{P(t) + \alpha_2}\frac{W(t)}{W(t) + \alpha_3}, \tag{3}$$

$$C(t) = \alpha_4 W(t) + (\alpha_5 + \alpha_6 \frac{W(t)}{N})N. \tag{4}$$

Here, α_1 characterizes the rate of wealth production for large W, while parameters α_2, α_3 define the wealth production rate for small P and W. Regardless of population size, the parameter α_4 is associated with the rate of amortization and the depletion of wealth. The level of individual consumption is described by the expression $\alpha_5 + \alpha_6 \frac{W}{N}$, where the first term denotes a fundamental level of consumption that is independent of wealth and the second term denotes consumption that is dependent on wealth. These functions of production and consumption are inspired by the epidemic-economic model introduced in [Mozokhina et al., 2024]. The complete system of ordinary differential equations (ODE) for the epidemic–economic model becomes

$$\frac{dS(t)}{dt} = -\frac{\beta}{N} S(t) I(t) + \gamma R_p(t), \tag{5a}$$

$$\frac{dI(t)}{dt} = \frac{\beta}{N} S(t) I(t) - \rho I(t), \tag{5b}$$

$$\frac{dR_n(t)}{dt} = \rho I(t) - \mu R_n(t), \tag{5c}$$

$$\frac{dR_p(t)}{dt} = \mu R_n(t) - \gamma R_p(t), \tag{5d}$$

$$\frac{dP(t)}{dt} = -\frac{\beta}{N} S(t) I(t) + \mu R_n(t), \tag{5e}$$

$$\frac{dW(t)}{dt} = U(t) - C(t). \tag{5f}$$

It is appropriate to assume that $\mu > \gamma$. This means that the productivity rate is larger than the rate of waning immunity. In other words, the convalescence period is shorter than the duration of natural immunity, which is usually satisfied for respiratory viral infections. System (5) is considered with the following initial conditions:

$$S(0) = N - I(0) > 0, \quad I(0) > 0, \quad R_n(0) = 0, \quad R_p(0) = 0, \quad P(0) = S(0), \quad W(0) > 0. \tag{6}$$

All model simulations were performed using MATLAB software. The numerical values of the models' variables used in the simulations are estimated as follows. The transmission rate and recovery rate are chosen so that the basic reproduction number (the formula of basic reproduction number will be derived later in this paper) is equal to 3, since this value is suitable for most viral infectious diseases such as Covid-19 and influenza [Li et al., 2020; Biggerstaff et al., 2014]. The rate of deterioration of immunity is chosen based on the fact that the average duration of immunity against influenza is about 6 months [Couch, Kasel, 1983]. However, there are no sources available for the values of the economic parameters.

Figure 1 displays a direct simulation of model (5) for values of parameters that correspond to $\mathfrak{R}_0 = 3$. As can be observed in this figure, the endemic solution is oscillatory stable. It will be demonstrated that the previous observation holds for any value of $\mathfrak{R}_0$ larger than 1.

Existence, uniqueness and positivity of solution

Here, we study the existence and uniqueness for the solution of system (5), and positivity of the solution for Eqs. (5a)–(5e).

Lemma 1. *For any nonnegative initial condition* $(S(0), I(0), R_n(0), R_p(0), P(0), W(0))$ *which satisfies condition* (6)*, system* (5) *has a unique global solution. The solution of Eqs.* (5a)–(5e) *is positive and bounded.*

Proof. The proof of the existence and uniqueness of a local solution of system (5) follows directly from the Cauchy – Lipschitz theorem. To prove the positivity of the solution of Eqs. (5a)–(5e),

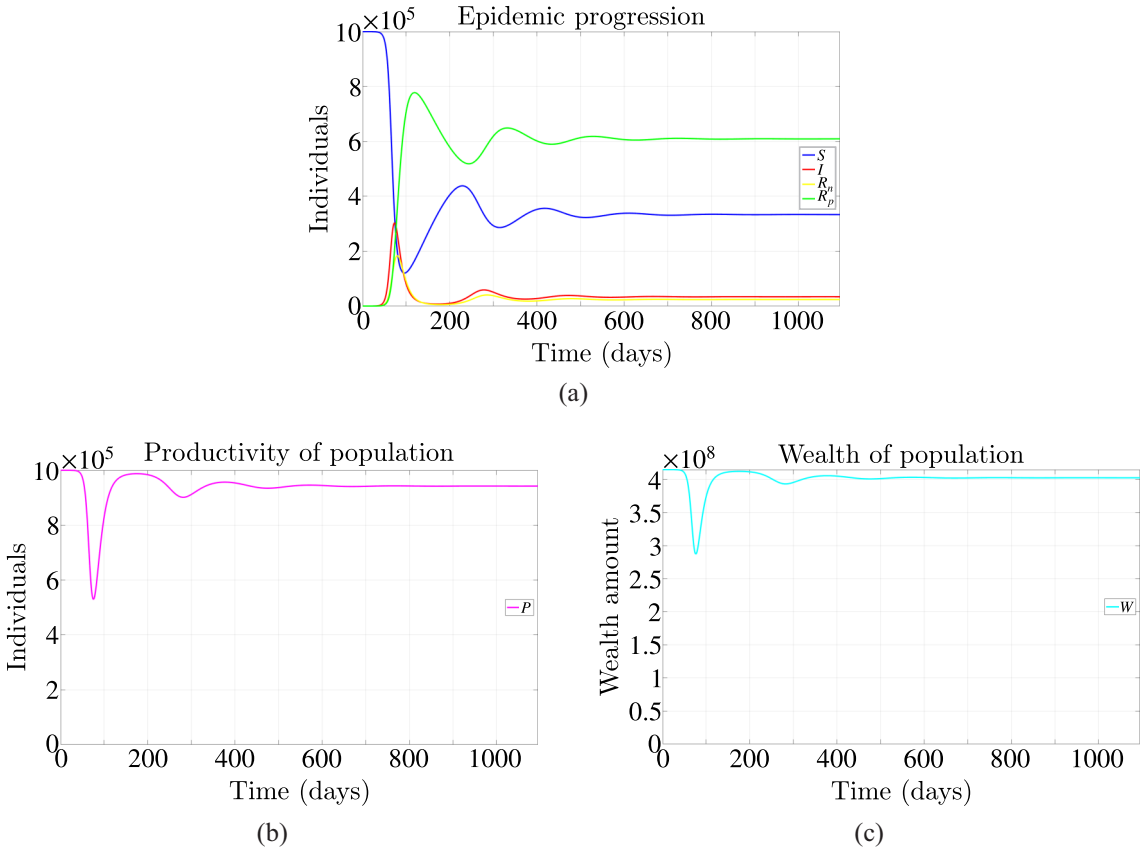


Figure 1. Simulation of model (5) for the initial conditions $N = 10^6$, $S(0) = N - 1 = P(0)$, $I(0) = 1$, $R_n(0) = 0$, $R_p(0) = 0$, $W(0) = 4.15 \cdot 10^8$, the epidemic parameters $\beta = 0.3$, $\rho = \frac{1}{10}$, $\gamma = \frac{1}{180}$, $\mu = \frac{1}{7}$, and economic parameters $a_1 = 10^9$, $a_2 = 10^6$, $a_3 = 10^6$, $a_4 = 0.5$, $a_5 = 0.7$, $a_6 = 0.7$. a) epidemic progression; b) productive individuals; c) wealth of population

we integrate Eqs. (5b), (5c), (5d) and (5a), respectively, taking into account initial values as defined in Eq. (6). We get the following:

$$I(t) = I(0)e^{-\int_0^t \left(\rho - \frac{\beta}{N} S(\eta)\right) d\eta} > 0,$$

$$R_n(t) = R_n(0)e^{-\int_0^t \mu\, d\eta} + e^{-\int_0^t \mu\, d\eta} \int_0^t \rho I(\eta) e^{\int_0^t \mu\, d\eta}\, d\eta > 0,$$

$$R_p(t) = R_p(0)e^{-\int_0^t \gamma\, d\eta} + e^{-\int_0^t \gamma\, d\eta} \int_0^t \mu R_n(\eta) e^{\int_0^t \gamma\, d\eta}\, d\eta > 0,$$

and

$$S(t) = S(0)e^{-\int_0^t \frac{\beta}{N} I(\eta)\, d\eta} + e^{-\int_0^t \frac{\beta}{N} I(\eta)\, d\eta} \int_0^t \gamma R_p(\eta) e^{\int_0^t \frac{\beta}{N} I(\eta)\, d\eta}\, d\eta > 0.$$

From Eq. (2), one can demonstrate that $P(t) > 0$. Finally, from Eq. (1) it follows that the solution of Eqs. (5a)–(5e) is bounded. The lemma is proved. □

It remains to note that the wealth function W defined in Eq. (5f) may drop to negative values due to epidemic, as we will see in the section “Epidemic can destabilize the economy”.

Equilibrium solutions of the system

Disease-free equilibrium

The disease-free equilibrium is a steady-state solution where there are no infected individuals in the population, meaning $I(t) = 0$. At this equilibrium, we assume that there is no ongoing transmission of infection, and we look for equilibrium values $\left(S^*, I^*, R_n^*, R_p^*, P^*, W^*\right)$ that satisfy system (5) when

$$\frac{dS}{dt} = 0, \quad \frac{dI}{dt} = 0, \quad \frac{dR_n}{dt} = 0, \quad \frac{dR_p}{dt} = 0, \quad \frac{dP}{dt} = 0, \quad \frac{dW}{dt} = 0. \tag{7}$$

Setting $I(t) = 0$ in Eq. (7), we get

$$\left(S^*, I^*, R_n^*, R_p^*, P^*, W^*\right) = (N, 0, 0, 0, N, W^*). \tag{8}$$

W^* can be determined from the equation

$$aW^{*2} + bW^* + c = 0,$$

where

$$a = \alpha_4 + \alpha_6, \quad b = \alpha_3(\alpha_4 + \alpha_6) + \alpha_5 N - \frac{\alpha_1 N}{N + \alpha_2}, \quad c = \alpha_3\alpha_5 N.$$

It has the discriminant $\Delta_1 = \left(\alpha_3(\alpha_4 + \alpha_6) + \alpha_5 N - \frac{\alpha_1 N}{N+\alpha_2}\right)^2 - 4\alpha_3\alpha_5(\alpha_4 + \alpha_6)N$. We get the two solutions

$$W_1^* = \frac{\alpha_1 \frac{N}{N+\alpha_2} - \alpha_3(\alpha_4 + \alpha_6) - \alpha_5 N + \sqrt{\Delta_1}}{2(\alpha_4 + \alpha_6)}, \tag{9}$$

$$W_2^* = \frac{\alpha_1 \frac{N}{N+\alpha_2} - \alpha_3(\alpha_4 + \alpha_6) - \alpha_5 N - \sqrt{\Delta_1}}{2(\alpha_4 + \alpha_6)}. \tag{10}$$

Hence, there are two free equilibria given by

$$E_1^* = \left(N, 0, 0, 0, N, W_1^*\right), \quad E_2^* = \left(N, 0, 0, 0, N, W_2^*\right). \tag{11}$$

Basic reproduction number

The basic reproduction number is the average number of secondary infections caused by a single infected individual in a fully susceptible population with no prior immunity or interventions. To find the basic reproduction number $\Re_0$ for system (5), we use the next-generation matrix method. To apply the next-generation matrix method, we focus on the infected classes and analyze the transmission and transition terms. The infected classes here include $I(t)$, and we will assume $S \approx N$ at the disease-free equilibrium, that is, $S^* \approx N$. We identify the new infection terms $F(I)$ which represent the rate of new infections entering each infected class, and the transition terms $V(I)$ which represent the rate of transfer out of each infected class. For Eq. (5b), we have

$$F(I) = \frac{\beta}{N} S(t) I(t),$$

and the transition terms $V(I)$ include the recovery rate:

$$V(t) = \rho I(t).$$

The next-generation matrix $K = FV^{-1}$ constructed from the partial derivatives of F and V with respect to I, evaluated at the disease-free equilibrium, that is,

$$F = \left[\frac{\partial F}{\partial I}\right] = \frac{\beta}{N}S^* = \beta, \quad V = \left[\frac{\partial V}{\partial I}\right] = \rho,$$

is used to compute the basic reproduction number $\mathfrak{R}_0$. Hence, the basic reproduction number for system (5) is given by

$$\mathfrak{R}_0 = \frac{\beta}{\rho}. \tag{12}$$

Endemic equilibrium

The term "endemic equilibrium" in the context of an epidemic/economic system refers to a stable, long-term state where an infectious disease persists in a population at a constant level, and this persistence is analyzed in conjunction with economic factors. This equilibrium arises when the dynamics of disease transmission and recovery are balanced, and the economic impacts of the disease are also in a steady state. To find the endemic equilibrium of system (5), we solve Eq. (7) for each variable in terms of others to obtain

$$(S_*, I_*, R_{n*}, R_{p*}, P_*, W_*) = \left(\frac{\rho N}{\beta}, \frac{N\left(1-\frac{\rho}{\beta}\right)}{1+\frac{\rho}{\mu}+\frac{\rho}{\gamma}}, \frac{\rho}{\mu}I_*, \frac{\rho}{\gamma}I_*, S_* + R_{p*}, W_*\right). \tag{13}$$

We have two possible values for W_*, obtained by solving the equation

$$aW_*^2 + b'W_* + c = 0, \tag{14}$$

where

$$a = \alpha_4 + \alpha_6, \quad b' = \alpha_3(\alpha_4 + \alpha_6) + \alpha_5 N - \frac{\alpha_1 P_*}{P_* + \alpha_2}, \quad c = \alpha_3\alpha_5 N,$$

with the discriminant

$$\Delta_2 = \left(\alpha_3(\alpha_4 + \alpha_6) + \alpha_5 N - \frac{\alpha_1 P_*}{P_* + \alpha_2}\right)^2 - 4\alpha_3\alpha_5(\alpha_4 + \alpha_6)N.$$

These values are

$$W_{*1} = \frac{\alpha_1 \frac{P_*}{P_*+\alpha_2} - \alpha_3(\alpha_4 + \alpha_6) - \alpha_5 N + \sqrt{\Delta_2}}{2(\alpha_4 + \alpha_6)}, \tag{15}$$

$$W_{*2} = \frac{\alpha_1 \frac{P_*}{P_*+\alpha_2} - \alpha_3(\alpha_4 + \alpha_6) - \alpha_5 N - \sqrt{\Delta_2}}{2(\alpha_4 + \alpha_6)}. \tag{16}$$

Therefore, there are two endemic equilibria given by

$$E_{*1} = \left(\frac{\rho N}{\beta}, \frac{N\left(1-\frac{\rho}{\beta}\right)}{1+\frac{\rho}{\mu}+\frac{\rho}{\gamma}}, \frac{\rho}{\mu}I_*, \frac{\rho}{\gamma}I_*, S_* + R_{p*}, W_{*1}\right),$$

$$E_{*2} = \left(\frac{\rho N}{\beta}, \frac{N\left(1-\frac{\rho}{\beta}\right)}{1+\frac{\rho}{\mu}+\frac{\rho}{\gamma}}, \frac{\rho}{\mu}I_*, \frac{\rho}{\gamma}I_*, S_* + R_{p*}, W_{*2}\right).$$

When a system's parameter reaches a critical threshold, the dynamical system undergoes a significant change in behavior. We investigate $\mathfrak{R}_0$ as the model's critical threshold. Figure 2 illustrates the endemic

equilibrium solutions (S_*, I_*, R_{n*}, R_{p*}, P_*, W_*) in relation to the basic reproduction number $\mathfrak{R}_0$, demonstrating the stability of these equilibria when $\mathfrak{R}_0 > 1$. Increasing $\mathfrak{R}_0$ elevates disease severity and reduces the number of productive individuals, as reflected by the declining stationary value W_{*1} shown in Fig. 2, *b*, which contributes to economic decline. Conversely, the stationary value W_{*2} in Fig. 2, *c*, increases with $\mathfrak{R}_0$, indicating competing dynamics in wealth accumulation.

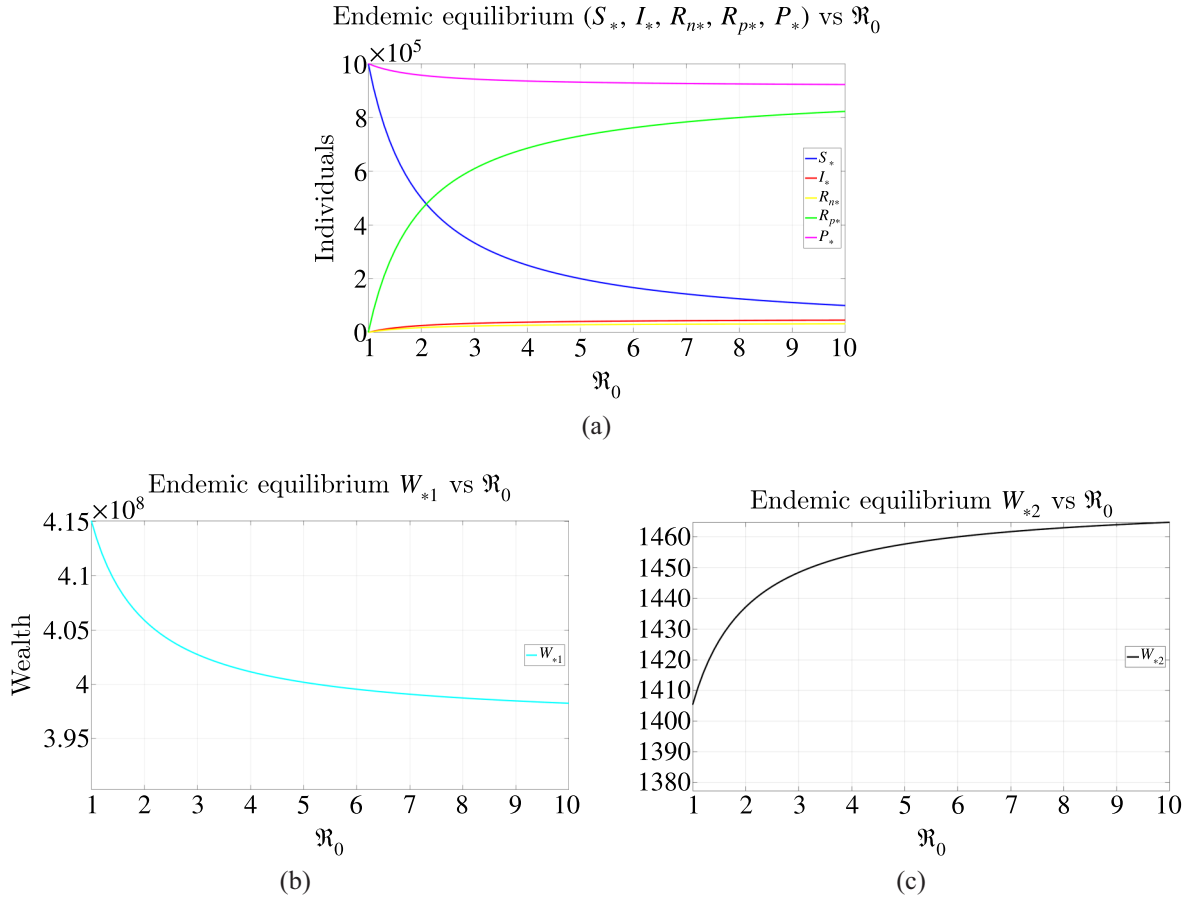


Figure 2. Forward bifurcation diagram of the endemic equilibrium of system (5) with respect to $\mathfrak{R}_0$ for the epidemic parameters $\rho = \frac{1}{10}$, $\gamma = \frac{1}{180}$, $\mu = \frac{1}{7}$, and the economic parameters $a_1 = 10^9$, $a_2 = 10^6$, $a_3 = 10^6$, $a_4 = = 0.5$, $a_5 = 0.7$, $a_6 = 0.7$. a) endemic equilibrium (S_*, I_*, R_{n*}, R_{p*}, P_*); b) endemic equilibrium W_{*1}; c) endemic equilibrium W_{*2}

Stability analysis

Local stability of the disease-free equilibrium

To determine local stability at the equilibrium point, we examine the eigenvalues of the system's Jacobian. In order to find the Jacobian matrix for this system of differential equations, let us denote the state vector as:

$$x = \begin{bmatrix} S & I & R_n & R_p & P & W \end{bmatrix}^T. \tag{17}$$

Then, the system can be written as $\frac{dx}{dt} = f(x)$, where $f(x)$ is the vector of right-hand side functions:

$$f(x) = \begin{bmatrix} -\frac{\beta}{N} S I + \gamma R_p & \frac{\beta}{N} S I - \rho I & \rho I - \mu R_n & \mu R_n - \gamma R_p & -\frac{\beta}{N} S I + \mu R_n & U - C \end{bmatrix}^T. \tag{18}$$

The Jacobian matrix J is defined as the matrix of partial derivatives of each component function of f with respect to each variable in x:

$$J = \frac{\partial f}{\partial x} = \begin{bmatrix} -\frac{\beta}{N}I & -\frac{\beta}{N}S & 0 & \gamma & 0 & 0 \\ \frac{\beta}{N}I & \frac{\beta}{N}S - \rho & 0 & 0 & 0 & 0 \\ 0 & \rho & -\mu & 0 & 0 & 0 \\ 0 & 0 & \mu & -\gamma & 0 & 0 \\ -\frac{\beta}{N}I & -\frac{\beta}{N}S & \mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\alpha_1\alpha_2 W}{(P+\alpha_2)^2(W+\alpha_3)} & \frac{\alpha_1\alpha_3 P}{(P+\alpha_2)(W+\alpha_3)^2} - (\alpha_4 + \alpha_6) \end{bmatrix}. \tag{19}$$

Theorem 1. *The disease-free equilibrium* $\widetilde{E}_0 = (N, 0, 0, 0, N)$ *corresponding to the epidemic part of system* (5) *is locally asymptotically stable if* $\mathfrak{R}_0 < 1$ *and unstable if* $\mathfrak{R}_0 > 1$.

Proof. The Jacobian matrix of the system at E_0 is given by

$$J_0 = \begin{bmatrix} 0 & -\beta & 0 & \gamma & 0 & 0 \\ 0 & \beta - \rho & 0 & 0 & 0 & 0 \\ 0 & \rho & -\mu & 0 & 0 & 0 \\ 0 & 0 & \mu & -\gamma & 0 & 0 \\ 0 & -\beta & \mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\alpha_1\alpha_2 W}{(P+\alpha_2)^2(W+\alpha_3)} & \frac{\alpha_1\alpha_3 P}{(P+\alpha_2)(W+\alpha_3)^2} - (\alpha_4 + \alpha_6) \end{bmatrix} \tag{20}$$

Let λ be an eigenvalue of J_0. The characteristic equation is obtained by solving:

$$\det(J_0 - \lambda I_{6\times 6}) = 0.$$

Thus, we obtain

$$\lambda^2(\lambda - \beta + \rho)(\lambda + \mu)(\lambda + \gamma)\left(\lambda - \frac{\alpha_1\alpha_3 P}{(P + \alpha_2)(W + \alpha_3)^2} + \alpha_4 + \alpha_6\right) = 0. \tag{21}$$

The characteristic equation has four nonzero real solutions given by

$$\lambda_1 = \beta - \rho, \quad \lambda_2 = -\mu, \quad \lambda_3 = -\gamma, \quad \lambda_4 = \frac{\alpha_1\alpha_3 P}{(P + \alpha_2)(W + \alpha_3)^2} - \alpha_4 - \alpha_6. \tag{22}$$

However, the characteristic equation corresponding to $\widetilde{E}_0$ is given by

$$(\lambda - \beta + \rho)(\lambda + \mu)(\lambda + \gamma) = 0, \tag{23}$$

and it has three nonzero real solutions given by λ_1, λ_2, λ_3. Both λ_2 and λ_3 are negative eigenvalues. Finally, note that λ_1 is positive only if $\beta > \rho > 0$, which means that $\mathfrak{R}_0 > 1$, and negative only if $0 < \beta < \rho$, which means that $\mathfrak{R}_0 < 1$. The theorem is proved. □

Note that the positive value of λ_4 determines the loss of stability of the stationary solution for the economic part of system (5).

Local stability of the endemic equilibrium

Theorem 2. *The endemic equilibrium* $\widetilde{E_*} = (S_*, I_*, R_{n*}, R_{p*}, P_*)$ *corresponding to the epidemic part of system* (5) *given in Eq.* (13) *is locally asymptotically stable if* $\Re_0 > 1$.

Proof. The Jacobian of system (5) at the endemic state E_* is given by Eq. (19). Then, the characteristic equation is obtained from

$$\det(J - \lambda I_{6\times 6}) = 0,$$

and it can be expressed as

$$\lambda^2\left(\lambda - \frac{\alpha_1\alpha_3 P_*}{(P_* + \alpha_2)(W_* + \alpha_3)^2} + \alpha_4 + \alpha_6\right)\left(\lambda^3 + \xi_1\lambda^2 + \xi_2\lambda + \xi_3\right) = 0, \tag{24}$$

where

$$\xi_1 = \frac{\beta(I_* - S_*)}{N} + \gamma + \mu + \rho, \tag{25}$$

$$\xi_2 = \frac{\beta\gamma(I_* - S_*)}{N} + \frac{\beta\mu(I_* - S_*)}{N} + \frac{\beta\rho I_*}{N} + \gamma\mu + \gamma\rho + \mu\rho, \tag{26}$$

$$\xi_3 = \frac{\beta\gamma\mu(I_* - S_*)}{N} + \frac{\beta\gamma\rho I_*}{N} + \frac{\beta\mu\rho I_*}{N} + \rho\gamma\mu. \tag{27}$$

Let us consider the equation

$$\lambda^3 + \xi_1\lambda^2 + \xi_2\lambda + \xi_3 = 0. \tag{28}$$

By the Routh – Hurwitz criterion, it follows that the endemic equilibrium $(S_*, I_*, R_{n*}, R_{p*}, P_*)$ is locally asymptotically stable if and only if

$$\xi_1 > 0, \quad \xi_2 > 0, \quad \xi_3 > 0,$$

and

$$\xi_1\xi_2 - \xi_3 > 0.$$

Substituting Eq. (13) into Eqs. (25)–(27), we obtain

$$\xi_1 = \frac{\beta - \rho}{1 + \frac{\rho}{\mu} + \frac{\rho}{\gamma}} + \gamma + \mu, \tag{29}$$

$$\xi_2 = \frac{(\rho + \gamma + \mu)(\beta - \rho)}{1 + \frac{\rho}{\mu} + \frac{\rho}{\gamma}} + \gamma\mu, \tag{30}$$

$$\xi_3 = \frac{\beta - \rho}{1 + \frac{\rho}{\mu} + \frac{\rho}{\gamma}}(\gamma\mu + \gamma\rho + \mu\rho). \tag{31}$$

Moreover, we have

$$\xi_1\xi_2 - \xi_3 = C^2(\rho + \gamma + \mu) + C(\gamma + \mu)^2 + (\gamma + \mu)\gamma\mu, \tag{32}$$

where

$$C = \frac{\beta - \rho}{1 + \frac{\rho}{\mu} + \frac{\rho}{\gamma}}. \tag{33}$$

If $\Re_0 > 1$, that is, $\beta > \rho$, and due to the positivity of all the parameters, then ξ_1, ξ_2 and ξ_3 are all positive. Additionally, $\xi_1\xi_2 - \xi_3 > 0$. Hence, by the Routh – Hurwitz criterion, it follows that the endemic equilibrium $\widetilde{E_*} = (S_*, I_*, R_{n*}, R_{p*}, P_*)$ is locally asymptotically stable. The theorem is proved. □

It remains to note that the positive real solution of the equation

$$\lambda = \frac{\alpha_1 \alpha_3 P_*}{(P_* + \alpha_2)(W_* + \alpha_3)^2} - (\alpha_4 + \alpha_6) \tag{34}$$

determines the loss of stability of the endemic equilibrium W_* for the economic part of system (5).

Complex eigenvalues can indicate oscillatory behavior in the system. This is crucial for predicting how the disease might fluctuate over time, which can inform public health interventions and control strategies. Let the eigenvalue of the endemic equilibrium $\widetilde{E}_* = (S_*, I_*, R_{n*}, R_{p*}, P_*)$ corresponding to the epidemic part of system (5), given in Eq. (13), be a complex number $\lambda = x + iy$. Then substituting into Eq. (28), we get the following two equations:

$$x^3 - 3xy^2 + \xi_1 \left(x^2 - y^2\right) + \xi_2 x + \xi_3 = 0, \tag{35}$$

$$3x^2 y - y^3 + 2\xi_1 xy + \xi_2 y = 0, \tag{36}$$

where ξ_1, ξ_2 and ξ_3 are defined in Eqs. (29)–(31).

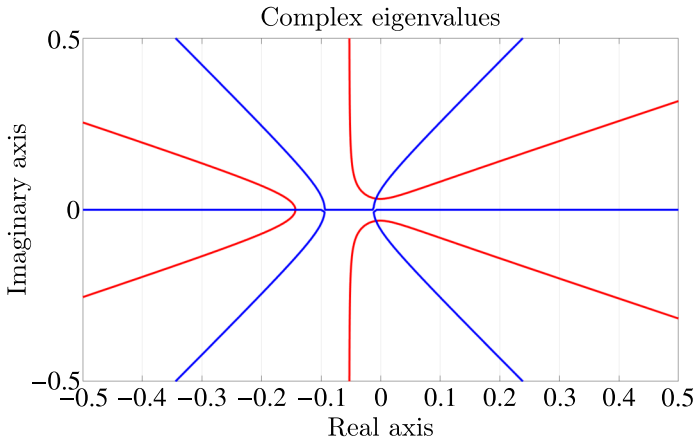


Figure 3. Solutions of Eq. (35) (red curve) and Eq. (36) (blue curve) for the values of parameters $\beta = 0.3$, $\rho = \frac{1}{10}$, $\gamma = \frac{1}{180}$, $\mu = \frac{1}{7}$. The intersection of these curves gives the eigenvalue of the Jacobian at the endemic equilibrium point $(S_*, I_*, R_{n*}, R_{p*}, P_*)$ related to the epidemiological part of system (5). All the eigenvalues are either negative real or complex with negative real part, determining the stability of endemic equilibrium (see Fig. 1, *a*, *b*)

Theorem 3. *For any value* $\mathfrak{R}_0 > 1$*, Eq.* (28) *does not have a purely imaginary solution for any positive values of the parameters* β*,* ρ*,* γ *and* μ*.*

Proof. Substituting $x = 0$ into Eq. (35), we have

$$y^2 = \frac{\xi_3}{\xi_1}.$$

Substituting $x = 0$ into Eq. (36), we get

$$y\left(-y^2 + \xi_2\right) = 0.$$

From the previous two equations, we obtain

$$\xi_3 = \xi_1 \xi_2.$$

This equality contradicts the fact that $\xi_1 \xi_2 > \xi_3$ concluded from Eq. (32) for $\mathfrak{R}_0 > 1$. The theorem is proved. □

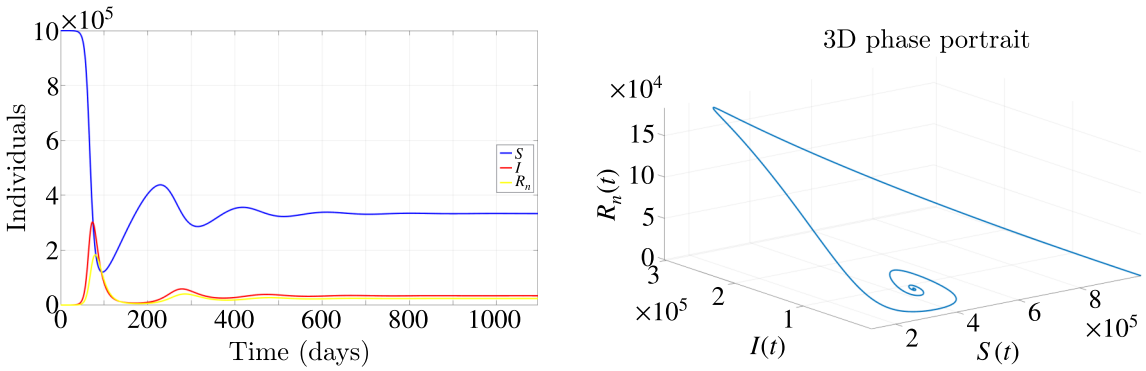
The fact that the complex eigenvalues of the endemic equilibrium regarding the epidemic part of system (5) always have a negative real part for any parameter values underscores the inherent stability of the epidemic part of that system. This property ensures that the disease dynamics will eventually stabilize, with any oscillations dampening over time. From a public health perspective, this stability provides a foundation for designing long-term control strategies and predicting the long-term behavior of infectious diseases. Figure 3 displays the eigenvalues corresponding to the endemic equilibrium of the epidemic part of system (5) for the initial conditions and parameters mentioned in Fig. 1 and emphasizes the stability of that equilibrium.

Using Eq. (1), the epidemiological part of system (5) can be reduced to the following system of differential equations:

$$\frac{dS(t)}{dt} = -\frac{\beta}{N}S(t)I(t) + \gamma(N - S(t) - I(t) - R_n(t)), \tag{37a}$$

$$\frac{dI(t)}{dt} = \frac{\beta}{N}S(t)I(t) - \rho I(t), \tag{37b}$$

$$\frac{dR_n(t)}{dt} = \rho I(t) - \mu R_n(t). \tag{37c}$$



(a) (b)

Figure 4. a) simulation of model (37) for the initial conditions $N = 10^6$, $S(0) = N - 1$, $I(0) = 1$, $R_n(0) = 0$, the epidemic parameters $\beta = 0.3$, $\rho = \frac{1}{10}$, $\gamma = \frac{1}{180}$, $\mu = \frac{1}{7}$; b) a 3D phase portrait, which represents the solution of system (37) for the previous values of parameters which correspond to $\mathfrak{R}_0 = 3$

Figure 4, *b* shows that the disease-free equilibrium $(S^*, I^*, R_n^*) = \left(10^6, 0, 0\right)$ is an unstable node, since the trajectory moves away from it. On the other hand, the endemic equilibrium $(S_*, I_*, R_{n*}) = (333\,212, 33\,856, 23\,705)$ is a stable spiral due to the motion of the trajectory towards the endemic equilibrium point.

The presented mathematical analysis verifies the model's adequacy and proves its suitability for studying the economic consequences of epidemic spread.

Modeling the socioeconomic outcome of infection spread and lockdown introduction

In this section, we illustrate the proposed model's applicability by studying socioeconomic parameters during uncontrolled epidemics and introduced lockdown measures. To simplify the analysis, we examine the special case with no immunity waning.

Epidemic can destabilize the economy

Consider the model without immunity waning, i. e., $\gamma = 0$ which is given by the following equations:

$$\frac{dS(t)}{dt} = -\frac{\beta}{N}S(t)I(t), \tag{38a}$$

$$\frac{dI(t)}{dt} = \frac{\beta}{N}S(t)I(t) - \rho I(t), \tag{38b}$$

$$\frac{dR_n(t)}{dt} = \rho I(t) - \mu R_n(t), \tag{38c}$$

$$\frac{dR_p(t)}{dt} = \mu R_n(t), \tag{38d}$$

$$\frac{dP(t)}{dt} = -\frac{\beta}{N}S(t)I(t) + \mu R_n(t), \tag{38e}$$

$$\frac{dW(t)}{dt} = U(t) - C(t), \tag{38f}$$

with initial conditions given in Eq. (6), where U and C are defined in Eq. (3) and Eq. (4), respectively. Note that model (38) is a special case of model (5), inheriting the same theorems, solutions, and stability properties.

The stationary solution for wealth is determined by setting $g(W) = 0$, where

$$g(W) = a_1 \frac{W}{W + a_2}\frac{P_s}{P_s + a_3} - a_4 W - \left(a_5 + a_6\frac{W}{N}\right)N, \tag{39}$$

where P_s is the equilibrium value of the productive population, which is independent of wealth but depends on the number of productive recovered and susceptible individuals. Next, we have

$$g'(W) = \frac{\alpha_1\alpha_2 P_s}{(P_s + a_3)(W + a_2)^2} - \alpha_4 - \alpha_6 \ (= \lambda_4 \text{ from Eq. (22)}).$$

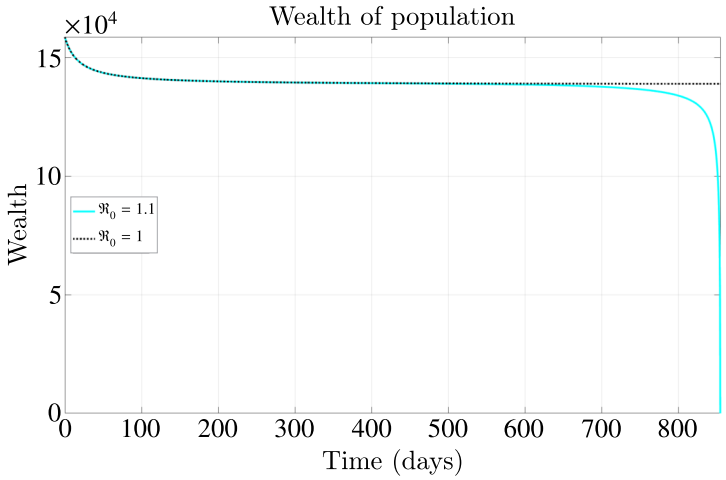


Figure 5. Evolution of wealth in numerical simulations of model (38) for initial conditions $N = 10^6$, $S(0) = N - I(0) = P(0)$, $I(0) = 1$, $R_n(0) = R_p(0) = 0$, $W(0) = 1.58 \cdot 10^5$, the rates $\rho = \frac{1}{10}$, $\mu = \frac{1}{7}$, and economic parameters $a_1 = 10^6$, $a_2 = 10^4$, $a_3 = 10^4$, $a_4 = 0.3$, $a_5 = 0.8611$, $a_6 = 0.15$. Wealth converges to a positive value for $\beta = 0.1$ ($\mathfrak{R}_0 = 1$) (black curve) or abruptly drops to negative values for $\beta = 0.11$ ($\mathfrak{R}_0 = 1.1$) (cyan curve)

When $\mathfrak{R}_0 < 1$, the disease cannot sustain an epidemic, as the infection-free equilibrium ($I = 0$) is stable. In this case, any small introduction of the disease will naturally result in its dying out over time without leading to widespread transmission. Hence, we have $P_s = N$, the equation $g(W) = 0$, or equivalently Eq. (14) which is a quadratic equation, can have two solutions given by Eqs. (15) and (16), one of which is stable (the one that satisfies $g'(W) < 0$), and defines the population's wealth. Recall that the stability of the solution of wealth part is derived from the eigenvalue λ_4 defined in Eq. (22) being less than 0. In the case of infection ($\mathfrak{R}_0 > 1$) where the endemic equilibrium is stable, the endemic solution for the productive population P_s is also stable. If it drops to a low enough value, then the equation $g(W) = 0$ or Eq. (14) does not have stationary solutions because $g(W)$ will become negative, and thus $\frac{dW}{dt} < 0$, which means that the wealth part will decrease to negative values (this implies that the determinant of the quadratic equation (14) is negative). The solution of Eq. (38f) with positive initial condition decreases and becomes negative. Negative wealth aligns with a debt-ridden economy. Nevertheless, since the model considered is not adapted to account for negative wealth, we ceased running further simulations at this point (Fig. 5). Interestingly, it should be noted that for weak infection, that is, when $\mathfrak{R}_0$ is slightly larger than 1, the level of wealth remains fairly constant over a long period of time before experiencing a sudden decline. Therefore, epidemics can induce economic instability, drive wealth down to negative levels, and disrupt the positive wealth equilibrium.

Influence of isolation on epidemic progression

In this section, we examine the case with permanent immunity. Isolating a portion of the population can affect the spread of the epidemic and reduce the number of infected people. We start our analysis of the influence of isolation and determine the optimal percentage of isolated people considering the scenario in which immunity does not wane. Hence, we model a partial lockdown in which a part of the population is isolated and cannot be infected. Isolation begins before the onset of the epidemic and ends after it has passed. Introducing a lockdown before the first disease case is detected, rather than after the epidemic has started, is a proactive strategy aimed at preventing or significantly mitigating the spread of an infectious disease. This approach can save lives, reduce the burden on healthcare systems, and minimize the size of the group of infected individuals. Thus, we impose a partial lockdown at time t_0 with duration T and a proportion of isolated population $k \in (0, 1)$ out of the total population N_0. Our goal is to determine the optimal percentage of isolated people which minimizes the size of infected individuals. Let us consider the following system of equations:

$$\frac{dS(t)}{dt} = -\frac{\beta}{N(t)} S(t) I(t), \tag{40a}$$

$$\frac{dI(t)}{dt} = \frac{\beta}{N(t)} S(t) I(t) - \rho I(t), \tag{40b}$$

$$\frac{dR_n(t)}{dt} = \rho I(t) - \mu R_n(t), \tag{40c}$$

$$\frac{dR_p(t)}{dt} = \mu R_n(t). \tag{40d}$$

Equation (40d) differs from Eq. (5d) because we assume that recovered individuals obtain permanent immunity against the disease and do not become susceptible any more. System (40) is considered with the initial conditions

$$S(0) = S_0 > 0, \quad I(0) = I_0 > 0, \quad R_n(0) = R_p(0) = 0, \tag{41}$$

where $S_0 + I_0 = N_0$. At the beginning of isolation at $t = t_0$, apart from the total population, the same part of the susceptible population is removed, and then returns at time $t_1 = t_0 + T$, as follows:

$$N(t_0) = (1 - k)N_0, \quad S(t_0) = S(t_0) - kN_0, \quad N(t_1) = N_0, \; S(t_1) = S(t_1) + kN_0. \tag{42}$$

Analytical estimate

Next, we evaluate the value of the percentage of isolated population k which determine the minimal total number of infected individuals. Let us set $R(t) = R_n(t) + R_p(t)$ denoting the number of all recovered individuals at time t, then $\frac{dR(t)}{dt} = \frac{dR_n(t)}{dt} + \frac{dR_p(t)}{dt}$ which gives

$$\frac{dR(t)}{dt} = \rho I(t). \tag{43}$$

Dividing Eq. (40a) by Eq. (43), and integrating from 0 to ∞, taking into account that $S_0 = (1-k)N_0$, and S_f is the final number of susceptible individuals, we obtain the following equation in $\phi = \frac{S_f}{(1-k)N_0}$:

$$\ln \phi = \mathfrak{R}_0(\phi - 1), \tag{44}$$

where $\mathfrak{R}_0 = \frac{\beta}{\rho}$ is the basic reproduction number. Equation (44) has a solution $\phi \in (0, 1)$ if $\mathfrak{R}_0 > 1$. When the lockdown is finished and isolated people return, the total number of susceptible individuals becomes $S_f + kN_0$ and the new value of the basic reproduction number is

$$\mathfrak{R}_0' = \frac{\beta}{\rho}\frac{S_f + kN_0}{N_0} = \mathfrak{R}_0((1-k)\phi + k).$$

We can find k from the condition $\mathfrak{R}_0' = 1$, which implies that the epidemic does not restart after the end of isolation:

$$k = \frac{\frac{1}{\mathfrak{R}_0} - \phi}{1 - \phi}. \tag{45}$$

The total number of infected individuals is given by the following formula:

$$I_{\text{total}} = (1-k)N_0 - S_f = (1-k)N_0(1-\phi). \tag{46}$$

For example, if $\mathfrak{R}_0 = 3$, then from Eq. (44) we obtain $\phi \approx 0.06$ and from Eq. (45) and Eq. (46) we have $k \approx 29\,\%$ and $\frac{I_{\text{total}}}{N} \approx 67\,\%$, respectively; on the other hand, without lockdown ($k = 0$) we obtain $\frac{I_{\text{total}}}{N} \approx 94\,\%$. Thus, isolation reduces the proportion of infected individuals by 27 %.

We note that for $\mathfrak{R}_0$ sufficiently large, we can use the approximation $\phi \ll 1$, and $k \approx \frac{1}{\mathfrak{R}_0}$. This simple formula gives a good approximation already for $\mathfrak{R}_0 = 3$.

Numerical simulations

Now, we numerically investigate the influence of isolation on the progression of the epidemic. Figure 6 displays examples of numerical simulations of system (40). The typical dynamics of epidemic breakout are seen in the situation without isolated population (panel a), where the number of susceptible individuals is declining and the number of recovered individuals is rising. We then consider the scenario where a portion of the population is isolated during the outbreak and then re-emerges once it is over. If the percentage of isolated people does not surpass some critical value, then the new basic reproduction number $\mathfrak{R}_0'$ is less than 1 and the epidemic does not recur (panel b). However, if the percentage of isolated people is high enough and $\mathfrak{R}_0' > 1$, then a second outbreak occurs (panel c) and the total number of infected people grows up. In the direct numerical solutions of Eq. (40) and Eq. (41), we calculated the total number of infected individuals. Figure 7 indicates that, in relation to the percentage of the isolated population, the total number of infected individuals in the model without immunity waning has a local minimum. For example, if $\rho = \frac{1}{10}$, then $\mathfrak{R}_0 = 3$, and the minimum optimal solution is given by the pair $\left(k, \frac{I_{\text{total}}}{N_0}\right) \approx (29\,\%, 67\,\%)$ (see Fig. 6, *b*), which corresponds to the analytical values obtained previously. However, if the percentage of isolated people is 60 % (as in Fig. 6, *c*), then

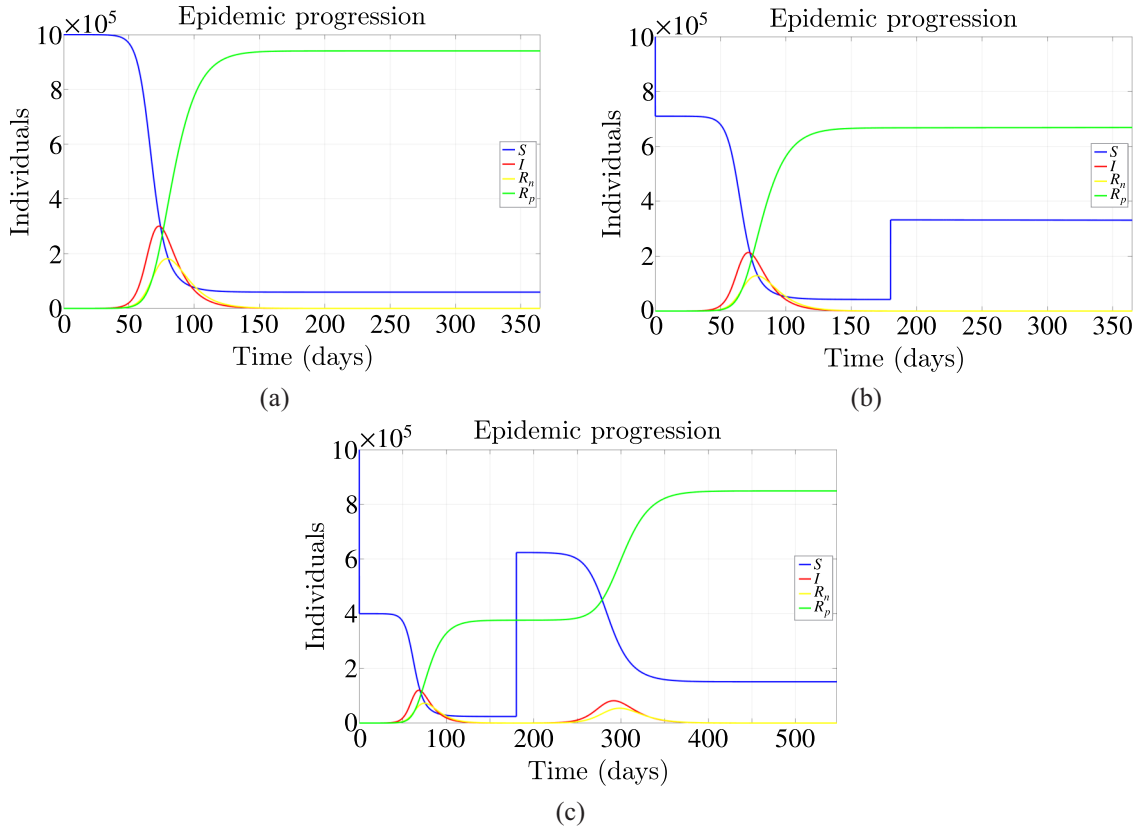


Figure 6. Susceptible, infected and recovered populations in numerical simulations of system (40) for initial conditions $N_0 = 10^6$, $S(0) = N_0 - 1$, $I(0) = 1$, $R_n(0) = R_p(0) = 0$ and parameters $\beta = 0.3$, $\rho = \frac{1}{10}$. a) without isolation; b) a part of the population is isolated before epidemic outbreak and returns afterward ($t_0 = 0$, $T = 180$, $k \approx 29\,\%$); c) $t_0 = 0$, $T = 180$, $k = 60\,\%$

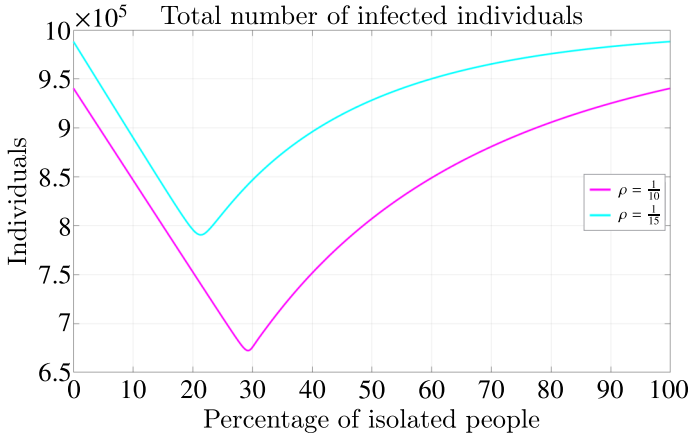


Figure 7. Dependence of the total number of infected individuals I_{total} on the percentage of isolated population in model (40) without immunity waning. The parameter values are $N_0 = 10^6$, $\beta = 0.3$, $t_0 = 0$, $T = 180$, $I(0) = 1$

the initial outbreak is minor, but there is a second outbreak after the end of isolation, which leads to a higher total number of infected individuals $\left(\frac{I_{\text{total}}}{N_0} \approx 85\,\%\right)$. The pair $\left(k, \frac{I_{\text{total}}}{N_0}\right) \approx (21\,\%, 79\,\%)$ represents the minimum optimal solution for $\rho = \frac{1}{15}$, or $\mathfrak{R}_0 = 4.5$. Sufficiently similar results are obtained using the analytical approximations $k \approx \frac{1}{\mathfrak{R}_0} \approx 22.2\,\%$, $\frac{I_{\text{total}}}{N_0} \approx 77.8\,\%$.

The influence of isolation on economy

We proceed to the analysis of the model with permanent immunity in addition to productive individuals and wealth, where we study the influence of isolation on the productive population and wealth. We obtain the following system of equations:

$$\frac{dS(t)}{dt} = -\frac{\beta}{N(t)}S(t)I(t), \tag{47a}$$

$$\frac{dI(t)}{dt} = \frac{\beta}{N(t)}S(t)I(t) - \rho I(t), \tag{47b}$$

$$\frac{dR_n(t)}{dt} = \rho I(t) - \mu R_n(t), \tag{47c}$$

$$\frac{dR_p(t)}{dt} = \mu R_n(t), \tag{47d}$$

$$\frac{dP(t)}{dt} = -\frac{\beta}{N(t)}S(t)I(t) + \mu R_n(t), \tag{47e}$$

$$\frac{dW(t)}{dt} = U(t) - C(t), \tag{47f}$$

where U and C are defined as

$$U(t) = \alpha_1 \frac{P(t)}{P(t) + \alpha_2} \frac{W(t)}{W(t) + \alpha_3}, \tag{48}$$

$$C(t) = \alpha_4 W(t) + (\alpha_5 + \alpha_6 \frac{W(t)}{N_0})N_0. \tag{49}$$

The model is considered with initial conditions

$$S(0) = N_0 - I(0) > 0, \quad I(0) > 0, \quad R_n(0) = 0, \quad R_p(0) = 0, \quad P(0) = S(0), \quad W(0) > 0. \tag{50}$$

In the case of lockdown, this model is completed by the following conditions:

$$\begin{gathered} N(t_0) = (1-k)N_0, \quad S(t_0) = S(t_0) - kN_0, \quad P(t_0) = P(t_0) - k\alpha N_0, \\ N(t_1) = N_0, \quad S(t_1) = S(t_1) + kN_0, \quad P(t_1) = P(t_1) + k\alpha N_0, \end{gathered} \tag{51}$$

which, respectively, determine the beginning of lockdown, its duration, and the proportion of isolated population where, $k \in (0, 1)$ and $\alpha \in [0, 1]$. Since isolated people can have different levels of productivity, we introduce the coefficient α for the proportion of isolated individuals who become unproductive during lockdown. Figure 8 displays the amount of wealth for different percentages of isolated people during lockdown, namely, 40 %, 65 % and 90 % also with different levels of productivity among these isolated people, namely, 0 %, 50 % and 100 %. In all of these plots, the amount of wealth shows a depression during the isolation period in proportion to the level of productivity among isolated people. Next, due to the secondary outbreak which occurs when the isolated people return to the susceptible compartment with a basic reproduction number larger than 1 after the lockdown, another dropping in the wealth amount happens in proportion to the percentage of isolated people before it stabilizes to the endemic equilibrium regardless of the proportion of isolated people. Figure 9 (magenta curve) shows that the amount of wealth during isolation is a decreasing function of the percentage of unproductive isolated individuals. However, if all the isolated people are productive, then the population wealth increases as the percentage of isolated people grows larger (cyan curve). If half of the isolated people are productive, then the amount of wealth is again a decreasing function

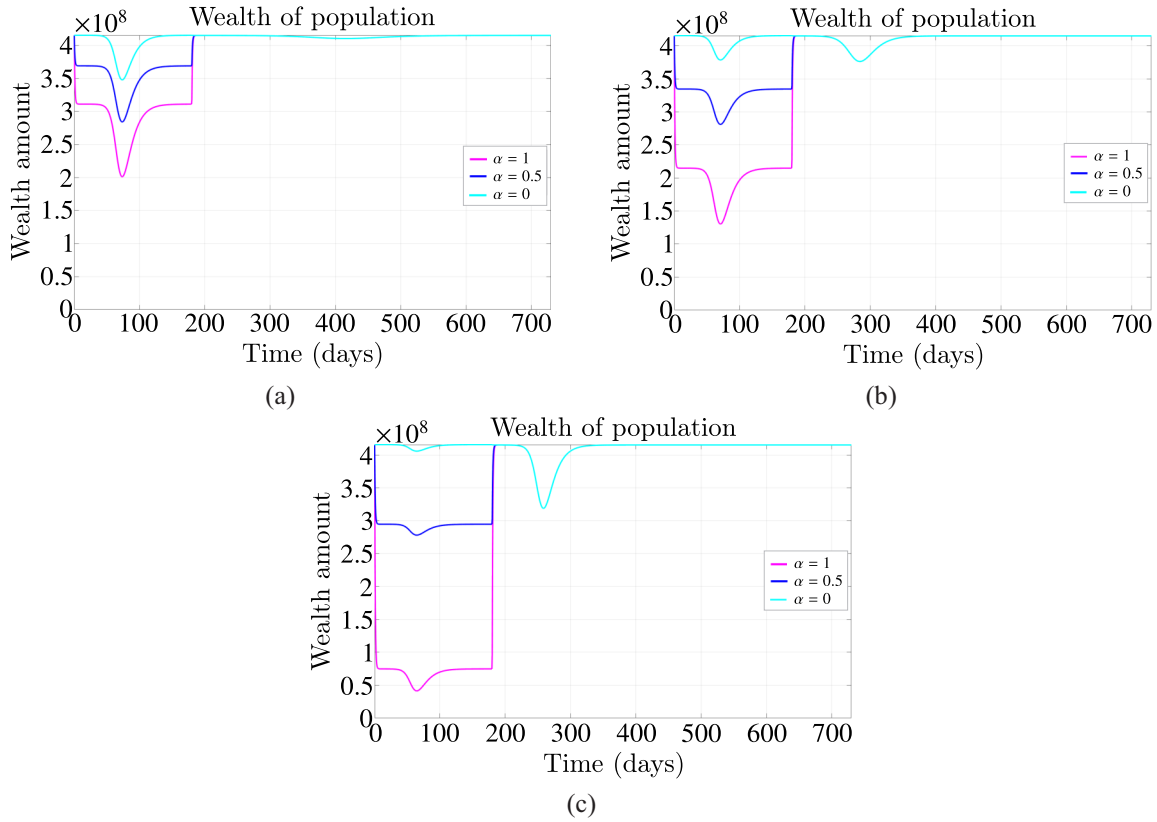


Figure 8. Simulations of model (47)–(51) with permanent immunity for the following values of parameters $N_0 = 10^6$, $\rho = \frac{1}{10}$, $\mu = \frac{1}{7}$, $t_0 = 0$, $T = 180$, $I(0) = 1$. a) $k = 40\,\%$; b) $k = 65\,\%$; c) $k = 90\,\%$

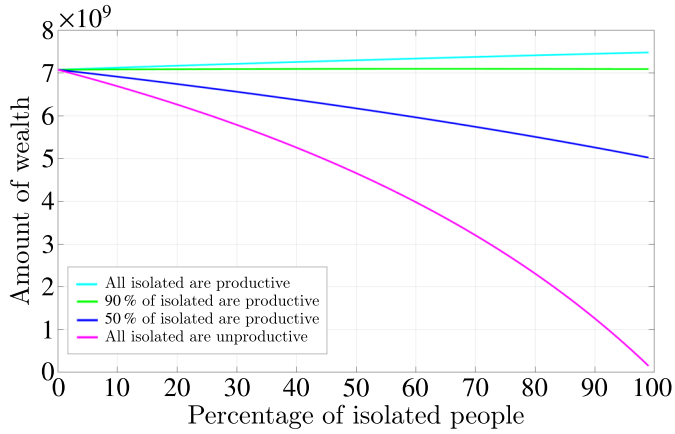


Figure 9. Dependence of the wealth amount during isolation $\rho \int_0^T W(\zeta)d\zeta$ on the percentage of isolated population in model (47)–(51) with permanent immunity. The parameter values are $N_0 = 10^6$, $\beta = 0.3$, $\rho = \frac{1}{10}$, $\mu = \frac{1}{7}$, $t_0 = 0$, $T = 180$, $I(0) = 1$

with respect to the percentage of isolated individuals (blue curve), but this decrease is weaker than in the case where all isolated people are unproductive. With 90 % of individuals among the isolated population being productive ($\alpha = 10\,\%$), the wealth remains approximately constant (green curve). Figure 10 tells us that if we increase the disease transmission rate and decrease the recovery rate, and consequently if we increase the basic reproduction number $\Re_0$, then the amount of wealth during

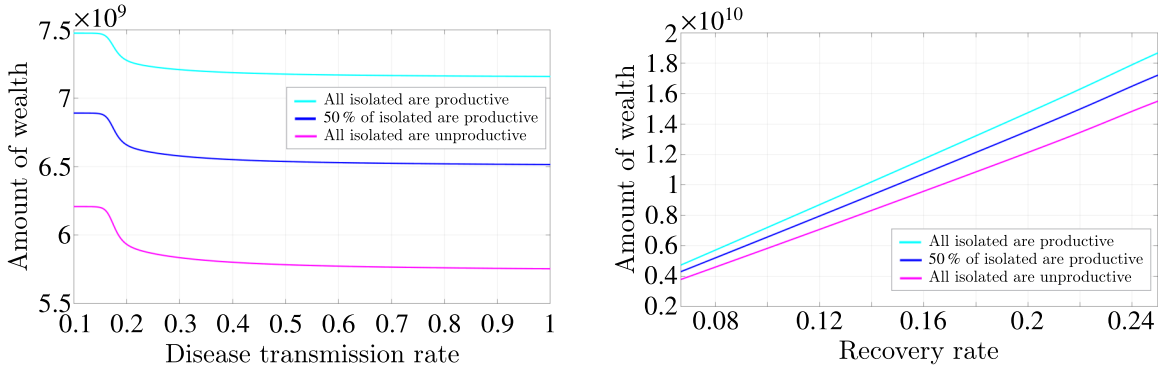


Figure 10. a) Dependence of the wealth amount during isolation $\rho \int_0^T W(\zeta)\, d\zeta$ on the disease transmission rate in model (47)–(51) with permanent immunity. The parameter values are $N_0 = 10^6$, $\rho = \frac{1}{10}$, $\mu = \frac{1}{7}$, $t_0 = 0$, $T = 180$, $k = 29\,\%$, $I(0) = 1$. b) Dependence of the wealth amount during isolation $\rho \int_0^T W(\zeta)\, d\zeta$ on the recovery rate in model (47)–(51) with permanent immunity. The parameter values are $N_0 = 10^6$, $\beta = 0.3$, $\mu = \frac{1}{7}$, $t_0 = 0$, $T = 180$, $k = 29\,\%$, $I(0) = 1$

isolation becomes smaller. Furthermore, as the percentage of productive individuals among the isolated people increases, the amount of wealth increases as well.

Discussion

In this work, an epidemiological-economic model that incorporates a system of ordinary differential equations featuring three rates is proposed. These rates correspond to recovery, immunity waning and productivity. Existence and uniqueness of its solution are proved along with the positiveness and boundedness regarding the solution corresponding to the epidemic parts of the ODE model, which is demonstrated as well. One of the goals of this work is to make a brief comparison with the insights obtained in the previous study [Mozokhina et al., 2024].

Stability analysis for both disease-free and endemic equilibria are investigated. In contrast to the corresponding model with time delays considered in [Mozokhina et al., 2024] where a positive stationary solution regarding the epidemic part of the system appears for a basic reproduction number larger than 1, then loses its stability and leads to periodic oscillations if the basic reproduction number exceeds some critical value, in the current work, the solution related to the epidemic parts of the proposed ODE model which refers to solution without infection in the population, loses its stability if $\mathfrak{R}_0 > 1$ but then oscillatory stabilizes to constant value reflecting the stability of endemic equilibrium point. The positive value of the eigenvalue λ_4 defined in (22) determines the loss of stability of both the disease-free and endemic equilibria of the economic part of system (5).

Isolation alters the wealth of the population, lessens the intensity of the disease outbreak, and has an impact on the epidemic and economic dynamics.

Determining the optimal percentage of isolated individuals is one of the work's primary outcomes. This best optimal choice is the maximum percentage of isolated people for whom the epidemic does not recur after the isolation ends, in the case of a single outbreak with no waning immunity. It is possible to calculate this optimal proportion analytically. For instance, the optimal proportion of isolated people is 29 % of the total population, which reduces the overall number of infected individuals by roughly 30 % if the basic reproduction number is 3. The analytical process of determining the optimal percentage of isolated people differs from that used in [Mozokhina et al., 2024]

because the formula of the basic reproduction number regarding each model is different. However, the numerical result of the ideal proportion of isolated individuals obtained in the present work is relatively close to that obtained in the previous work using the DDE model. Isolation can thereby lessen the strain on the economy and public health system. After the lockdown is over, a secondary epidemic outbreak happens if the percentage of isolated individuals is higher than the optimal level. As was the case with COVID-19 in China, this scenario can result in a significant number of infections and fatalities if it is not previously anticipated [Wikipedia, COVID-19].

Thus, while lockdown can be helpful, its duration and the percentage of isolated people should be adapted to each specific epidemic, otherwise the effects on the economy and public health may not be satisfactory.

We also study the influence of epidemics on the economy in the absence and in the presence of isolation. When recovered, individuals obtain permanent immunity against the disease. Wealth deteriorates as a result of lower numbers of productive individuals and increasing disease severity brought on by the increase in the basic reproduction number. Although enforcing partial lockdowns lessens the epidemic, the impact on the economy during isolation is contingent upon the productivity of the isolated people. Wealth increases during isolation if all isolated individuals are productive; nevertheless, as the productivity rate among the isolated population declines, the population's wealth declines. If 90 % of isolated people are productive, the level of wealth remains roughly constant. This is a crucial measure that describes how economically effective lockdowns are. This crucial number should be determined numerically for every unique situation, as we have yet to find an analytical formula for it.

Furthermore, this work demonstrates how epidemics can cause a sudden shift to negative wealth values, hence eliminating a positive wealth equilibrium. This occurs as a result of the decline in the proportion of productive people among the population. Because of the significant decline in the productive population, many epidemics have historically had significant economic and social repercussions [Wikipedia, Italian plague]. The effect of epidemics on the economy through the productive population is similar, even though this work does not take epidemic-induced mortality into account. By offering a thorough examination of the interaction between epidemiological dynamics and economic determinants, these findings clearly correspond to the research goal stated in the introduction. In particular, this analysis provides useful information on how policymakers might create more efficient lockdown plans that strike a balance between the demands of public health and economic viability during isolation. Future studies should concentrate on improving these models by adding more real-world data and broadening the scope of the analysis to encompass the impacts of vaccination and long-term immunity.

A pivotal outcome of this study is the construction of a mathematical model which integrates both epidemiological parameters and economic balance, accounts for factors critical to understanding the epidemic's economic impact such as long-term disease consequences and heterogeneity in population groups' economic participation. This model can be used for simulating economic costs in epidemic control, such as comparing different economic systems with varying parameter ratios, analyzing the economic impact of reduced recovery time and testing the model on real-world data from various countries.

This study has some limitations. First, ODE models do not simulate periodic outbreaks because the endemic equilibrium corresponding to the epidemic part of system (5) is always stable for $\mathfrak{R}_0 > 1$ in contrast to the DDE models where the endemic stationary solution related to the epidemic part is oscillatory unstable if $\mathfrak{R}_0$ exceeds some critical value $\mathfrak{R}_c > 1$ (see [Saade et al., 2024a; Saade, Aniţa, Volpert, 2023; Mozokhina et al., 2024]). As a result, one can conclude that DDE models are more appropriate in describing epidemic progression than ODE models, which fail to capture the periodic outbreaks which are a common phenomenon in the dynamics of several real-world diseases such as

influenza, measles, COVID-19, dengue fever, and many others. These oscillations arise due to the interplay between susceptible, infected, and recovered individuals, as well as factors like immunity, transmission rates, and population behavior. However, it would be interesting to understand how to describe them using ODE models. Furthermore, exposed parts, which can have some bearing on the population's economic situation, were not taken into account in this work. Moreover, in subsequent research, the modeling methods described in this paper might be used to examine data for different diseases in various nations using actual budget indicators. These and a few additional questions are intriguing open-ended questions for future research.

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