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Modeling the Impact of Combined Effect of Vaccination, Testing, and Treatment on Epidemic Dynamics

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Abstract: This study introduces SVEIATRD compartmental model, exploring the interplay of vaccination, testing, treatment, in the context of individual decision-making in epidemic control. The model is comprehensively analyzed to derive reproduction numbers and maximum infections and deaths. The results of comprehensive simulations illustrate the intricate compromises associated with managing resource allocation regarding vaccine coverage, testing availability, and treatment capacity. Vaccination plays a crucial role in achieving herd immunity; however, the advantages diminish after certain coverage thresholds. Implementing widespread testing is crucial for early diagnosis and transmission control. Adopting treatment measures plays a crucial role in further limiting the spread of a particular disease or condition. However, the effectiveness of treatment adoption is hindered by the restrictions imposed by available resources.

Keywords: mathematical modeling; epidemiology; infectious disease; vaccination; testing; treatment

1. Introduction

The study presents an epidemiological model that expands upon the conventional susceptible-exposed-infected-recovered (SEIR) model by integrating factors related to vaccination, testing, and treatment. The primary objective of this study is to examine the potential impact of individuals' decision-making processes on the containment of epidemics, specifically concerning vaccination, testing, treatment rates, vaccine efficacy, and the development of immunity. This research endeavors to offer insights into the efficient management of epidemic transmission through informed decision-making by analyzing the interrelated dynamics.

Theoretical epidemic modelling predominantly uses compartmental models, with the SIR (susceptible-infected-recovered) model being widely recognized[1)]. The model has undergone expansion to incorporate many situations, interventions, and assumptions. Among the most prevalent extensions are the SEIR model[2)], along with its derivatives, including SVIS[3)], SEIQR[4)], SAVIER[5)], and SEAIHRD[6)]. The mathematical models of epidemic dynamics introduced by Kermack et al.[1)] and subsequent researchers[7-9)] have been very important in epidemiology. These models have been essential in studying various tactics to control and prevent the spread of infectious diseases. Their relevance has been particularly highlighted during the COVID-19 pandemic[10-11)]. Gaining a comprehensive understanding of the transmission dynamics of this disease is of utmost importance in efficiently implementing preventive measures and accurately forecasting the occurrence of outbreaks. Comprehending the transmission of this ailment is crucial for successfully implementing preventive measures and accurately forecasting epidemics. Many theoretical models have been postulated to replicate the dissemination of COVID-19 across various scenarios[12-15)]. Numerous health measures, including physical protection and individual and national initiatives, have been intensively investigated as strategies to mitigate the spread of the pandemic[16-21)].

Identifying and segregating individuals who have contracted the COVID-19 virus are widely acknowledged as crucial measures in managing the spread of the pandemic[22)]. The testing process plays a crucial role in identifying confirmed infections facilitating the implementation of suitable treatment strategies and prevention measures. As a result, implementing testing measures typically leads to a decrease in epidemic growth rates, as it encourages more individuals to pursue preventive measures and actively seek appropriate medical interventions. Participation in vaccination programs and adoption of self-defense measures during an epidemic are also influenced by individual behaviors and decisions[23-25)]. The contingent valuation method is crucial for evaluating individuals' monetary valuation, demand,

hesitancy, and acceptance of vaccination[26-30]. This assessment takes into account the socio-economic-demographic perspective, specifically considering vaccine effectiveness and the associated cost burden. Furthermore, it is imperative to consider the healthcare system's efficacy, capacity, and recovery rate in delivering treatment and mitigating disease transmission. This is important, as a dysfunctional system could worsen the epidemic[31]. Immunity can be acquired through several means, including the process of recovery, vaccination, or the administration of convalescent plasma transformation as a treatment[32]. The acquisition of immunity, whether through vaccination, therapeutic intervention, or natural infection, substantially influences the transmission of diseases.

To account for these characteristics, a novel epidemic model known as SVEIATRD (susceptible-vaccinated-exposed-diagnosed infected-undiagnosed infected-treated-immune-deceased) is introduced. This model integrates vaccination, testing, and treatment components into the conventional SEIR model. Extensive simulations have demonstrated that the testing rate significantly influences epidemic dynamics since individuals tend to forego seeking treatment if they have not received a diagnosis for the disease. Simultaneously monitoring test positivity rates and the basic reproduction number can be a valuable tool in evaluating and enhancing public health management and testing systems. This approach can contribute to a more comprehensive comprehension of epidemic dynamics. The present model considers the influence of self-awareness, specifically in the context of testing and vaccination, on many factors such as illness risk, rates of intervention (including vaccination, testing, and treatment), the effectiveness of these treatments, and the development of immunity via recovery.

Although the model primarily emphasizes epidemic dynamics, it is crucial to recognize the substantial amount of research that integrates social dynamics into epidemic modeling. The significance of incorporating human behavioral responses into disease models has been highlighted by significant advancements in the Vaccination Game (VG) and Intervention Game (IG) domains. These methodologies, frequently employing frameworks such as Bauch's Behavior model[33] or evolutionary game theory (EGT)[34], offer valuable insights into the influence of individual decision-making processes on epidemic outcomes[35-37].

The reason for our choice to exclusively examine epidemic dynamics in this research was driven by the intention to separate and examine the precise effects of vaccination, testing, and treatment interventions on the spread of the disease. By excluding social dynamics, we can better analyze the direct impact of these interventions on the spread of the epidemic. Nevertheless, we acknowledge that this method has constraints, as it fails to encompass the intricate interaction between human conduct and the spread of diseases. Possible future developments of this research could incorporate elements of social dynamics, enabling the essential parameters p, ρ, and η to fluctuate over time in response to changing individual attitudes and behaviors. This would offer a more extensive and accurate representation of the progression of an epidemic. Nevertheless, the existing model is an initial stage in comprehending the interaction among various intervention strategies, offering valuable insights that can guide public health policy and resource allocation choices.

2. Model and Methods

A compartmental model called SVEIATRD is developed, which aims to analyze the spread dynamics of infectious illnesses by incorporating the effects of vaccination, testing, and treatment as control strategies. The model consists of eight distinct stages, namely susceptible (S), vaccinated (V), exposed (E), diagnosed infected (I), undiagnosed infected (A), treated (T), recovered (R), and deceased (D). Individuals susceptible to the disease and who have not yet been affected can acquire the infection by encountering individuals who have been diagnosed with or have not been diagnosed with the infection. Nevertheless, an incubation phase precedes the manifestation of infection in individuals. The individuals throughout this period are commonly referred to as individuals who have been exposed. During the incubation period, individuals exposed to the virus carry it within their bodies but cannot transmit it to other vulnerable individuals.

Furthermore, susceptible individuals can potentially receive vaccination via a vaccination system that may have some imperfections. Individuals who have been vaccinated can develop immunity to the disease through the vaccination, or they may progress to a stage when they are exposed to the disease. However, the likelihood of transitioning to the exposed stage is significantly lower for vaccinated individuals than those susceptible to the disease. Following this, those who have contracted the illness can be classified into two distinct groups: those who have received a positive diagnosis through testing and those who remain undiagnosed due to either opting not to undergo testing or receiving inaccurate negative test results. Undiagnosed individuals are generally presumed to refrain from seeking treatment, differentiating them from diagnosed individuals who can explore medical assistance.

The compartmental model is depicted in Fig. 1. Individuals susceptible to a disease and those who have been vaccinated can be exposed to the disease when they come into contact with infected individuals. The transmission rates for susceptible individuals and vaccinated individuals are β and $(1-\eta)\beta$, respectively, where η denotes the effectiveness of the vaccination. Following a period of incubation lasting $1/\sigma$, a proportion p of the individuals who were exposed to the virus will test positive and subsequently be identified as

diagnosed infected individuals. Conversely, the remaining fraction of individuals, $1-p$, will not be diagnosed and will be classified as undiagnosed individuals.

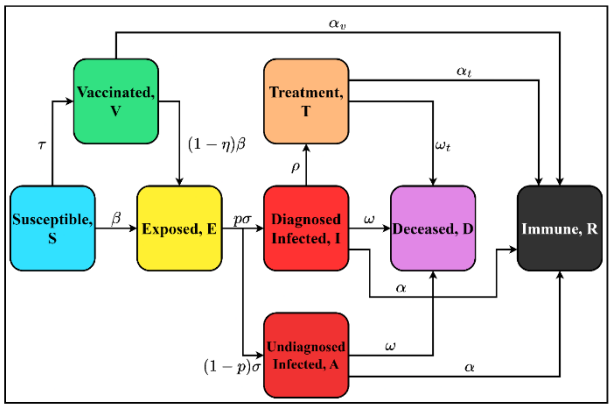


Fig. 1: Graphical representation of the compartmental SVEIARTD model

The parameter ρ represents the probability of individuals choosing to undergo treatment. A higher value of ρ signifies a greater likelihood for individuals to receive treatment. The parameter α_t signifies the rate at which individuals acquire immunity or achieve recovery during the course of treatment, whereas the parameter ω_t reflects the mortality rate associated with the treatment. Individuals who have received a vaccination acquire immunity as a result of the vaccination at a rate denoted as α_v.

Individuals who have received a diagnosis but choose not to pursue treatment have two possible outcomes: they may experience natural recovery at a rate represented by α, or they may encounter mortality with a chance denoted by ω. Individuals who remain undiagnosed and do not actively pursue treatment exhibit equivalent likelihoods of both recovery and fatality. The model's formulation is expressed by equations (1.1) to (1.8), where Λ represents the overall birth rate, and μ represents the per capita death rate. Table 1 is a compilation of thorough details regarding the model's parameters and their corresponding explanations.

Table 1: Explanation of the parameters in the model

Rate	Description
α	Recovery/immunity rate from infection
α_t	Recovery/immunity rate from treatment (signifies completely successful treatment)
α_v	Recovery/immunity rate from vaccination
β	Disease transmission rate
η	Vaccination efficiency
ω	Death rate from infection
ω_t	Death rate from treatment (signifies failed treatment)
p	Testing rate
ρ	Treatment rate
τ	Vaccination rate
σ	Incubation rate

$$S'(t) = \Lambda - \tau S(t) - \beta S(t)[I(t) + A(t)] + \gamma T(t) - \mu S(t) \quad (1.1)$$

$$V'(t) = \tau S(t) - (1-\eta)\beta V(t)[I(t) + A(t)] - \alpha_v V(t) - \mu V(t) \quad (1.2)$$

$$E'(t) = \beta[S(t) + (1-\eta)V(t)][I(t) + A(t)] - \sigma E(t) - \mu E(t) \quad (1.3)$$

$$I'(t) = p\sigma E(t) - \rho I(t) - \alpha I(t) - \omega I(t) - \mu I(t) \quad (1.4)$$

$$A'(t) = (1-p)\sigma E(t) - \alpha A(t) - \omega A(t) - \mu A(t) \quad (1.5)$$

$$T'(t) = -\gamma T(t) + \rho I(t) - \alpha_t T(t) - \omega_t T(t) - \mu T(t) \quad (1.6)$$

$$R'(t) = \alpha_v V(t) + \alpha I(t) + \alpha A(t) + \alpha_t T(t) - \mu R(t) \quad (1.7)$$

$$D'(t) = \omega I(t) + \omega A(t) + \omega_t T(t) - \mu D(t) \quad (1.8)$$

The proposed model is composed of a bilinear system that encompasses a total of eight differential equations. To comply with the principle of mass conservation, it is necessary for the overall change across all states to balance out and result in a net value of zero since

$$N(t) = S(t) + V(t) + E(t) + I(t) + A(t) + T(t) + R(t) + D(t) \quad (2)$$

To this end, it is assumed

$$\Lambda = \mu N(t) \quad (3)$$

3. Mathematical Analysis

3.1 Positivity and boundedness

For further analysis, we prove that the system is positive and well-bounded; that is, a non-negative value always bounds the fractions of all the states. Adding equations (1.1) through (1.8), we get

$$N'(t) = \Lambda - \mu N \quad (4)$$

Solving the differential equation (4) for N(t), it is obtained

$$N(t) = \Lambda/\mu + \left(N(0) - \frac{\Lambda}{\mu}\right)\exp(-\mu t) \quad (5)$$

Therefore, for all $t \geq 0$,

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \quad (6)$$

Consider any compartment X from the model. It is evident from equation (2) that $X(t) \leq N(t)$ for any $t \geq 0$. Additionally,

$$X'(t) \geq -\mu X(t) \quad (7)$$

Solving inequality (7) for $X(t)$, it is obtained

$$X(t) \geq X(0)\exp(-\mu t) \quad (8)$$

If $X(0) \geq 0$, then for all time $t \geq 0$,

$$0 \leq X(t) \leq N(t) \leq \frac{\Lambda}{\mu} \quad (9)$$

Therefore, equation (9) proves all the compartments' positivity and boundedness and the overall model.

3.2 Reproduction Number

The basic reproduction number of an infection is the expected number of cases directly generated by one case in a population where all individuals are susceptible to infection. We adopt the next-generation matrix approach[38] at the disease-free equilibrium (DFE) to calculate it. At DFE, the number of susceptible, vaccinated, and immune individuals is given by

$$(S^*, V^*, R^*) = \left(\frac{\Lambda}{\tau+\mu}, \frac{\tau\Lambda}{(\alpha_v+\mu)(\tau+\mu)}, \frac{(\alpha_v\tau\Lambda)}{\mu(\alpha_v+\mu)(\tau+\mu)}\right) \quad (10)$$

The value of R^* signifies the fraction of the population who obtained vaccination-induced immunity. In contrast, S^* and V^* values represent unvaccinated and vaccinated susceptible populations, respectively, at equilibrium. The DFE stands as

$$E_0 = (S^*, V^*, E^*, I^*, A^*, T^*, R^*, D^*) = \left(\frac{\Lambda}{\tau+\mu}, \frac{\tau\Lambda}{(\alpha_v+\mu)(\tau+\mu)}, 0,0,0,0, \frac{\alpha_v\tau\Lambda}{\mu(\alpha_v+\mu)(\tau+\mu)}, 0\right) \quad (11)$$

The matrices F of new infection terms and V of the remaining transfer terms associated with the model are given by

$$F = \begin{pmatrix} 0 & \beta s_0 & \beta s_0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (12a)$$

$$V = \begin{pmatrix} \sigma+\mu & 0 & 0 \\ -p\sigma & \rho+\alpha+\omega+\mu & 0 \\ -(1-p)\sigma & 0 & \alpha+\omega+\mu \end{pmatrix} \quad (12b)$$

By solving for the maximum eigenvalue of $K = FV^{-1}$ given by spectral radius $\rho(K)$, the basic reproduction number of the model R_0 is given by

$$R_0 = \rho(FV^{-1}) = \frac{p\sigma\beta s_0}{(\sigma+\mu)(\rho+\alpha+\omega+\mu)} + \frac{(1-p)\sigma\beta s_0}{(\sigma+\mu)(\alpha+\omega+\mu)} \quad (13)$$

For $R_0 \leq 1$, the disease-free equilibrium is globally stable. For $R_0 > 1$, two equilibria exist: the disease-free equilibrium, which is unstable, and a unique endemic equilibrium, which is globally stable whenever $I(0) + A(0) > 0$ [39]. The basic reproduction number can be subdivided into R_{sd}, R_{su}, R_{vd}, and R_{vu} for susceptible-diagnosed, susceptible-undiagnosed, vaccinated-diagnosed, and vaccinated-undiagnosed individuals, respectively.

$$R_{sd} = \frac{(p\sigma\beta S^*)}{(\sigma+\mu)(\rho+\alpha+\omega+\mu)} \quad (14a)$$

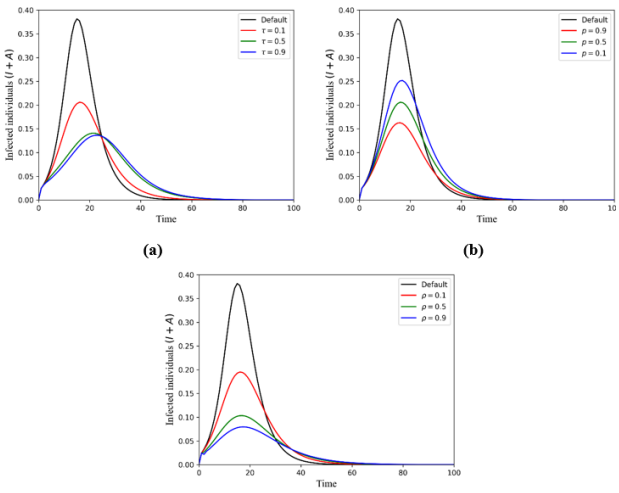
$$R_{su} = \frac{(1-p)\sigma\beta S^*}{(\sigma+\mu)(\alpha+\omega+\mu)} \quad (14b)$$

$$R_{vd} = \frac{p\sigma\beta(1-\eta)V^*}{(\sigma+\mu)(\rho+\alpha+\omega+\mu)} \quad (14c)$$

$$R_{vu} = \frac{(1-p)\sigma\beta(1-\eta)V^*}{(\sigma+\mu)(\alpha+\omega+\mu)} \quad (14d)$$

4. Results

The model under consideration simplifies to a conventional SEIRD framework when the parameters τ, p, and ρ are assigned specific values: $\tau = 0$, $p = 1$, and $\rho = 0$. The setup functions as the fundamental scenario illustrating the spread of disease without any measures to control it. Figure 2 presents a comparative analysis of several interventions, including vaccination, testing, and treatment, in relation to the default situation. Figure 2(a) demonstrates a decrease in the total number of infected individuals $(I + A)$ as the vaccination rate, denoted as τ, approaches unity. Significantly, the augmentation of the vaccination rate from $\tau = 0$ to $\tau = 0.1$ considerably enhances the containment of pathogen transmission. Increasing the value of τ to 0.5 results in further enhancements. However, the incremental improvement in the number of infected individuals between $\tau = 0.5$ and $\tau = 0.9$ is negligible. It is crucial to emphasize that as the value of τ increases, there is a decrease in the number of infected individuals, but at the same time, the duration of the infection period increases.



(a) (b) (c)

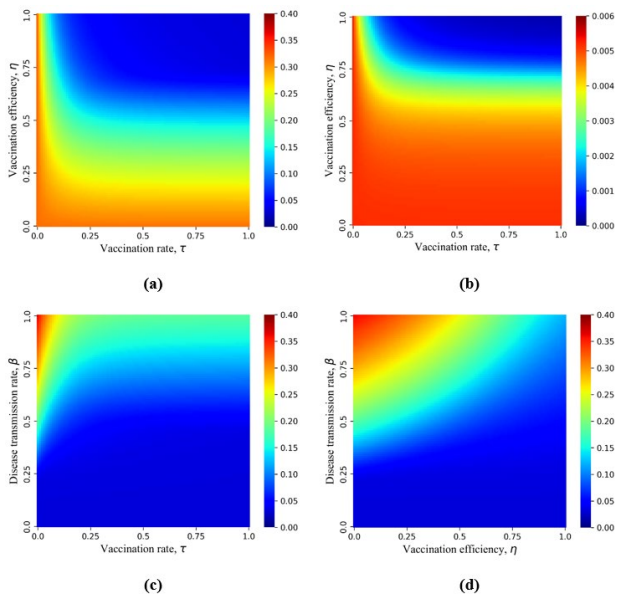
Fig. 2: Comparison of model dynamics with default cases under the variations of (a) vaccination rate τ, (b) testing rate p, and (c) treatment rate ρ. Values of other parameters are given in Table 2.

Table 2: Default values of the parameters in the model

Parameter	Value
$S(0)$	0.9505
$E(0)$	0.048
$I(0)$	0.0005
$A(0)$	0.0005
$T(0)$	0.0002
$R(0)$	0.0003
α	0.1667
α_t	0.3333
α_v	0.0005
β	0.8333
η	0.5
ω	0.001
ω_t	0.0005
p	0.5
ρ	0.08
σ	0.5
τ	0.1
Λ	0.0001 for calculations related to R_0, 0 otherwise
μ	0.0001 for calculations related to R_0, 0 otherwise

Figure 2(b) examines various testing rates, where $p = 0.9$ and $p = 0.5$ represent 90% and 50% positivity rates for detecting infections, respectively. On the other hand, $p = 0.1$ indicates a significant proportion of undiagnosed cases. The examination of Fig. 2(b) indicates that the occurrence of infections is significantly reduced when there is widespread utilization of testing ($p = 0.9$). A discernible decrease in disease spread is noticed compared to the initial reference point. As the value of p lowers, there is a commensurate increase in the number of infected individuals. Concerning Figure 2(c), a value of $\rho = 0.1$ indicates a very small proportion of infected individuals choosing to pursue treatment, whereas a value of $\rho = 0.9$ indicates a significant treatment adoption. An increase in the parameter ρ is associated with decreased disease transmission. However, it is important to note that the magnitude of this effect diminishes as ρ increases, indicating diminishing marginal returns.

Figure 3 examines the efficacy of vaccination options within the scenario offered. Figures 3(a) and 3(b) illustrate the highest count of infected and deceased individuals, respectively, observed at different levels of vaccination rate (τ) and vaccination efficiency (η). The data highlights the significance of maintaining optimal levels of vaccination efficacy to successfully mitigate infection transmission. The containment of diseases continues to be challenging when the value of η is low, regardless of the degree of vaccine coverage. In contrast, increased values of η are associated with significantly reduced occurrences of infected and deceased individuals, especially when τ is at moderate to high levels. Figure 3(c) provides a comprehensive analysis of the relationship between the vaccination rate (τ) and disease transmission rate (β) in governing the upper limit of illnesses inside the model. When the value of β is low, it is seen that limited infection is prevalent across all values of τ. In situations where disease transmission rates are elevated, manipulating the parameter τ has been observed to lead to a reduction in infection rates. However, it is important to note that the effectiveness of this intervention lowers due to the fixed value of η within this particular context. In contrast, Fig. 3(d) demonstrates a significantly accelerated illness control as the efficacy of vaccination increases, particularly for large values of β.



(a) (b) (c) (d)

Fig. 3: Effectiveness of vaccination strategies: (a) Maximum number of infected individuals as a function of vaccination rate τ and vaccination efficiency η, (b) Maximum number of

deceased individuals as a function of vaccination rate τ and vaccination efficiency η, (c) Maximum number of infected individuals as a function of disease transmission rate β and vaccination rate τ, (d) Maximum number of infected individuals as a function of disease transmission rate β and vaccination efficiency η. Values of other parameters are given in Table 2.

Figure 4 emphasizes the crucial significance of testing inside the model context. Figure 4(a) depicts the relationship between the maximum number of infected individuals and two key variables: the testing rate (p) and the disease transmission rate (β). Figure 4(b) depicts the highest count of deceased individuals under identical settings. When the tendency for disease transmission decreases, the testing rate has minimal impact on the spread of the disease. However, when examining scenarios characterized by large values of β, it becomes increasingly imperative to possess a correspondingly high value of p. When the testing rate is elevated, there is an increase in the number of individuals who acquire a positive diagnosis for the disease. Consequently, there is also a rise in the number of individuals who are willing to undergo treatment such as hospitalization, quarantine, and other health interventions facilitate effective disease control.

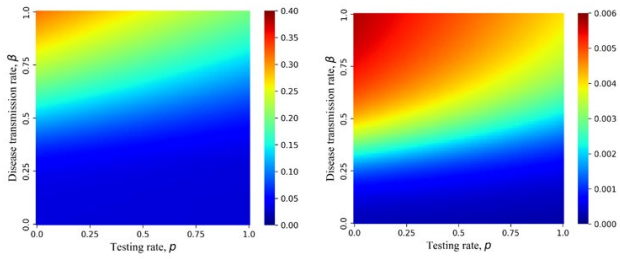


Fig. 4: Effect of testing and diagnosis on (a) number of infected individuals, (b) number of deceased individuals. Values of other parameters are given in Table 2.

Figure 5 presents an analysis of the influence of treatment selection and effectiveness on the spread of the disease. Figure 5(a) depicts the relationship between the maximum number of infected individuals and two key factors: the disease transmission rate (β) and the treatment rate (ρ). The criticality of the issue arises when the value of β attains significant levels while ρ remains considerably lower in contrast. Most ρ values effectively mitigate the spread of diseases within the low to moderate β values range. Nevertheless, to effectively control the spread of the disease, it is imperative to maintain a treatment rate that ranges from moderate to high, especially while the transmission rate (β) continues to rise. A similar trend can be observed about the greatest number of deceased individuals, as depicted in Fig. 5(b). Reduced disease transmission results in decreased mortality rates across various degrees of treatment. In order to maintain regulated disease transmission, it is important to implement a treatment level that ranges from medium to high for intermediate β values. When β levels are high, there is a strong positive correlation between low values of ρ and a relatively high number of deaths, whereas moderate to high values of ρ are associated with a moderate number of deaths. Figure 5(c) displays the relationship between the maximum number of deceased individuals and the variables β and α_t, representing the infection rate and the rate of recovery or immunity from treatment, respectively. The variable α_t serves as a measure of treatment effectiveness within this particular situation. As depicted in the figure, the variable α_t demonstrates significantly reduced variability in its association with disease transmission. A decrease in β values is associated with a decrease in the number of deaths, regardless of the values of α_t.

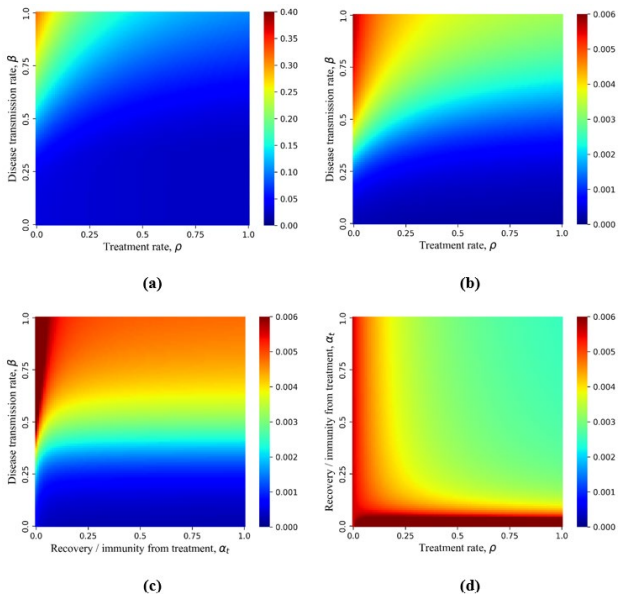
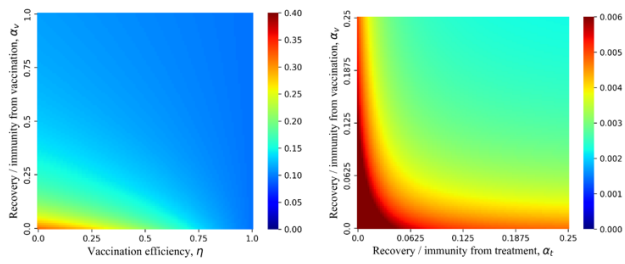


Fig. 5: Influence of treatment choice and efficacy: (a) Maximum number of infected individuals as a function of treatment rate ρ and disease transmission rate β, (b) Maximum number of deceased individuals as a function of treatment rate ρ and disease transmission rate β, (c) Maximum number of deceased individuals as a function of recovery rate from treatment α_t and disease transmission rate β, (d) Maximum number of deceased individuals as a function of treatment rate ρ and recovery rate from treatment α_t. Values of other parameters are given in Table 2.

In contrast, elevated β values are associated with an increased incidence of mortality across nearly all levels of α_t. The pattern is notably evident when examining values of β that are exceptionally high and values of α_t that are exceptionally low. Figure 5(d) illustrates the relationship between the variables ρ and α_t and their impact on the maximum number of deceased individuals. Adverse possibilities occur when either parameter takes on low levels. The issue is further aggravated when the value of α_t is low while ρ continues to climb. This scenario highlights a notable disparity between the inclination of individuals to pursue treatment and the sufficiency of the treatment resources that are accessible.

The disparity between the demand for treatment and the limited availability of medical resources has adverse consequences.

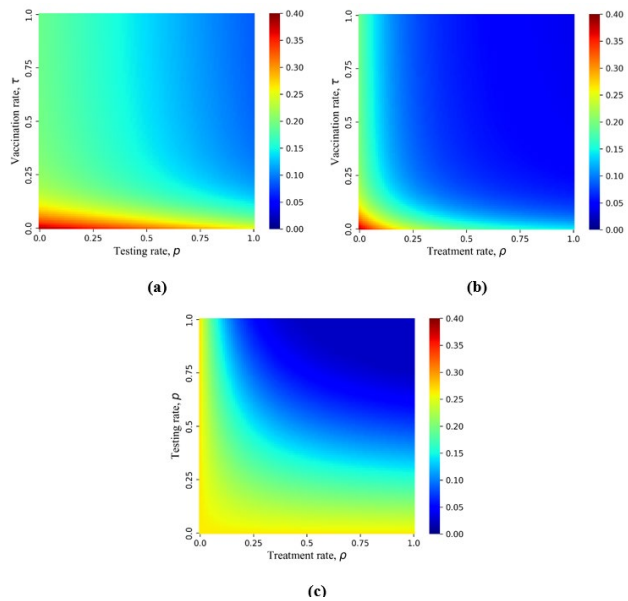
Figure 6 depicts the observed dynamics of the model, namely the interplay between various efficacy and recovery rates. Figure 6(a) examines the influence of vaccination efficiency (η) and vaccine-induced immunity (α_v) on the prevalence of infected people. An observable phenomenon of controlled infection transmission is seen when the values of either parameter are increased. At even moderate levels of α_v, the disease remains effectively managed throughout all values of τ. Infections may exhibit a modest increase in occurrence when α_v values are moderate and τ values are low. There is a significant rise in infection rates when both parameters reach much lower values. Figure 6(b) illustrates the relative impact of vaccine-induced immunity α_v and treatment-induced immunity α_t on the mortality rate. Both variables exhibit comparable functions in limiting the occurrence of fatalities. Lower values of any variable are linked to negative consequences. Relatively advantageous conditions are obtained when both variables exhibit moderate to high levels.



(a) (b)

Fig. 6: Modeling efficiency and recovery from different interventions: (a) Maximum number of infected individuals as a function of recovery rate from vaccination α_v and vaccination efficiency η, (b) Maximum number of deceased individuals as a function of recovery rate from treatment α_t and recovery rate from vaccination α_v. Values of other parameters are given in Table 2.

In Figure 7, we explore the impact of interventions and choice factors by graphing the number of infected individuals as a function of various intervention rates, namely the vaccination rate (τ) and testing rate (p). When the testing rate is high ($p = 0.9$), we continuously see low infection rates, irrespective of the vaccination rate. In the scenario where vaccination is not available ($\tau = 0$), installing comprehensive testing measures effectively decreases the occurrence of illnesses. Nevertheless, a discernible effect on the vaccination rate becomes apparent when the testing rate declines moderately ($p = 0.5$). The absence of vaccination is associated with a notable rise in the occurrence of illnesses, as indicated by τ values ranging from 0 to 0.3.



(a) (b)

(c)

Fig. 7: Effect of interventions and choice factors by plotting the maximum number of infected individuals as a function of (a) vaccination rate τ and testing rate p, (b) vaccination rate τ and treatment rate ρ, and (c) testing rate p and treatment rate ρ. Values of other parameters are given in Table 2.

In contrast, a higher vaccination rate is associated with consistently lower infection rates, as indicated by τ values ranging from 0.7 to 1. It is apparent that the influence of the testing rate surpasses that of the vaccination rate. The most notable reduction in infection rates is noticed while shifting from a moderate testing rate ($p = 0.5$) to a high testing rate ($p = 0.9$), as evidenced by the prominent blue color gradient. This statement underscores the importance of employing robust testing methodologies to mitigate the transmission of diseases by promptly identifying cases and facilitating timely medical intervention. Vaccination can offer supplementary safeguarding in situations where testing capabilities are constrained but to a diminished degree. The link between the maximum number of affected individuals and vaccination and treatment rates is illustrated in Fig. 7(b). In instances where the treatment rate is elevated ($\rho = 0.9$), the prevalence of infections remains comparatively low, irrespective of the level of vaccination.

On the contrary, the lack of vaccination does not adequately reduce the transmission of diseases in situations where the rate of treatment is high. As the treatment rate declines from $\rho = 0.5$ to $\rho = 0.1$, a noticeable correlation between the vaccination rate and the incidence of sickness becomes apparent. There is a correlation between a decline in vaccination rates and a notable rise in infection rates, which aligns with a decrease in the rate of treatment. Vaccination provides additional protection in situations where the availability of treatment is limited. However, the total impact of the treatment rate remains more substantial. The link between the maximum number of infected individuals and testing and treatment rates is depicted in Fig. 7(c). When the testing rate is high ($p = 0.9$), the number of infections

remains relatively low, regardless of the treatment rates. The crucial element in effectively managing infections in this particular scenario is the use of rigorous testing protocols.

Nevertheless, when exposed to a moderate level of testing with a chance of 0.5, the treatment rate has a discernible impact. As the treatment rate increases incrementally from 0 to 0.9, there is a gradual decline in the incidence of infections. However, it is important to acknowledge that the testing rate remains the most significant factor, particularly throughout the shift from moderate to high testing rates.

In essence, the significance of a high testing rate lies in its pivotal role in facilitating timely identification and intervention, hence mitigating the spread of infections. The presence of alternative therapies offers supplementary assistance in situations when testing capabilities are constrained. In contrast, vaccination acts as a protective measure when diagnostic and treatment resources are inadequate.

5. Discussion

The general study provides certain key insights across all analyses. One important insight is that the induction of immunity through vaccination plays a crucial role in forming herd immunity and lowering infection rates. Nevertheless, it is crucial to acknowledge that the marginal benefits derived from vaccination efforts begin to decline as the vaccination rate continues to increase. Once a specific threshold of vaccination rate is reached, the marginal increase in vaccination coverage does not result in a proportional decrease in the number of infections. This raises the question of whether investments in vaccine endeavors should be reassessed upon attaining this threshold. Another important takeaway is that the observed results regarding testing rates may seem contradictory at first, as without doing tests, the treatment rate becomes irrelevant. The importance of testing rates becomes evident only when the rate of treatment is also taken into active consideration. The results also highlight the importance of treatment adoption in individuals infected with the disease to manage disease transmission effectively. However, it is important to acknowledge an inherent constraint on the potential of greater treatment acceptance to reduce disease transmission effectively. The effectiveness of treatment diminishes gradually as the number of individuals choosing treatment increases. The observation above carries significant ramifications for policymakers and researchers in the field of public health as they develop measures to mitigate the spread of infectious illnesses.

According to the findings presented in Fig. 3, it can be observed that the most favorable outcomes are observed when certain circumstances are met. Specifically, these conditions include a low transmission rate β, as well as moderate to high rates of vaccination efficiency η and rate of vaccination τ. Under these particular conditions, the effectiveness of vaccination in reducing the transmission of the disease has been established. Even in situations where the likelihood of transmission is minimal, implementing a vaccination method with moderate efficacy can nevertheless be highly successful in controlling the spread of diseases. The vaccination rate, when varies from moderate to high, ensures that a substantial section of the population attains immunity, thereby collectively reducing the occurrence of diseases and fatalities.

Figure 5 illustrates the best situations under the combination of high values of treatment efficacy α_t and treatment rate ρ, along with a low disease transmission potential β, which leads to reduced death rates. On the contrary, when β is elevated, it becomes crucial to administer medication to reduce the likelihood of mortality. Moreover, an increased value of ρ can potentially offset the impact of a moderate α_t in cases when β is diminished. The matter of coverage is of utmost importance as it relates to the allocation of unequal treatment. When the value of α_t is somewhat high, specifically over 0.5, and the value of ρ is also high, there is a constant reduction in mortality rates, resulting in the maximum advantages.

Figure 6 illustrates the optimal circumstances, characterized by elevated α_t, α_v and η levels. In such scenarios, the effectiveness of vaccination is considerably enhanced, resulting in a noteworthy decrease in the prevalence of infections. The attainment of herd immunity is contingent upon the acquisition rate of immunity, which ensures that individuals are adequately protected against pathogens as they build immunity. The examination of Fig. 7 highlights the favorable circumstances in which parameters p, ρ, and τ exhibit elevated values. Significantly, a change in the p from 0.9 to 0.5 indicates a significant modification in infection dynamics, underscoring the necessity for heightened testing endeavors.

6. Conclusion

The present study presents a novel epidemic model, the SVEIATRD model, that integrates the dynamics of vaccination, testing, and treatment interventions. The model provides useful insights into the complex relationship between human decision-making and public health initiatives designed to reduce the transmission of diseases.

The analysis conducted emphasizes the crucial significance of vaccination in the establishment of herd immunity. However, it also highlights a point of saturation in the advantages of vaccination once a particular coverage rate is reached. The utilization of testing methods has been recognized as a crucial instrument for the timely identification of diseases and mitigating infection rates, surpassing the influence of treatment implementation. Nevertheless, the efficacy of increased diagnostic rates is limited by the scarcity of treatment

resources. The findings, as mentioned above, underscore the importance of upholding optimal immunization effectiveness, guaranteeing universal availability of testing, and offering sufficient treatment resources.

The findings of our study indicate that in situations characterized by heightened transmission capacity, the implementation of comprehensive testing protocols and treatment measures becomes crucial. On the other hand, in scenarios characterized by a low probability of transmission, treatments with modest effectiveness can effectively manage and contain epidemics. This study highlights the importance of acquiring immunity through either the process of healing from an infection or through vaccination.

Potential areas for future research could center on relevant evaluations of diseases, employing real-world data to validate and calibrate models. Additionally, the framework's adaptability enables the integration of intricate elements such as age distribution, social connections, geographical aspects, and resource limitations. In brief, our model provides practical insights that may be used to influence evidence-based policymaking regarding the allocation of resources in the face of competing tactics for controlling epidemics.

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