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A Study on Unified Role of Warning Signs and Signal-SIR Model Using DNN to Predict Epidemic Transmission

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ABSTRACT

This study extends the classical Susceptible-Infected-Recovered (SIR) model by integrating adaptive behaviors and policy interventions during epidemics through the Signal-SIR model. Here the susceptible population is divided into two groups: individuals who adhere to health regulations (AD strategy) and those who do not (NAD strategy). The model simulates the dynamic interaction between government signals and public behavior where it utilizes replicator dynamics to explore how health warnings influence population responses. It also introduces chicken game payoffs to analyze the redistribution of risks between compliant and non-compliant individuals. To optimize model parameters and explain time-varying dynamics, deep neural networks (DNNs) has been employed alongside Stochastic Gradient Descent. We establish a loss function that quantifies the discrepancies between observed data and model predictions. Simulation results indicate that enhanced adaptive behavior, driven by enhanced adherence to health regulations, significantly reduces the spread of infection. Therefore, it leads to lower infection peaks and higher recovery rates. This paper highlights the critical role of adaptive strategies in public health policy and provides a data-driven framework for effectively forecasting and managing epidemic dynamics.

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1 Introduction

Mankind has long been struggling with the tragic consequences of various infectious disease that not only threaten human lives but also impose an enormous economic burdens. Epidemic remains a constant menace to development of human civilization, starting from the historical outbreak of Black Death and the plague to contemporary challenges like the emergence of COVID-19 pandemic. Therefore, the prevention of globally transmitted infectious disease is treated as a major concern that needs immediate attention in the modern world [1, 2]. Researchers of all time have repeatedly employed various methods for modeling, predicting, and thereby investigating the root causes to control the rapid transmission of these epidemics. Due to the growing rate of international travel, investigations on epidemics are facing significant challenges in recent years. In this context, mathematical modeling has proven to be an extremely helpful tool that provides a strong theoretical

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foundation for executing relevant study. The practice of mathematical modeling in epidemiology originates from the seminal work presented by Kermack and McKendrick back in 1927 [3]. They introduced the classical SIS and SIR models, thereby shaped the concept of infectious disease thresholds. Relying on these classical epidemic models, a large plethora of works has been dedicated to explore the characteristics of epidemic propagation, where most of the existing studies considered the population being homogeneous and well-mixed. Thus, individual heterogeneity is often ignored in those models. Additionally, the epidemic modeling metaphor has been put in place to narrate a wide variety of phenomena. Information dissemination, cultural norms, and social behavior can conceptually be added to model a realistic contagion process [4]. Consequently, multiple attempts have been made to incorporate more practical factors into epidemic models. More precisely, ODE-based modeling approaches have been implemented to analyze the spread of various infectious diseases in populations exhibiting homogeneous mixing. In the course of time, researchers started acknowledging the concept of homogeneity, frequently used in modeling population structure, is incorrect. In reality, people who are socially proactive usually have more contacts with others than those always maintain less social engagements. Likewise neuronal system, interactions among individuals living in a society exhibit highly heterogeneous patterns. Therefore, it is necessary to take population topology into consideration while modeling and analyzing disease transmission behavior. To this end, complex social network particularly serves as a suitable tool for characterizing this heterogeneity [5–9].

Recent advances in network system dynamics have paved the way to conduct a proliferation of studies which are commonly based on numerous applications of complex networks in nonlinear science, sociology, biology, and other fields of information technology. Over the past few decades we observed a second golden age in epidemic modeling. Indeed, the real-world accuracy of the epidemic modeling has considerably been ameliorated by the incorporation of large-scale data sets and the decisive simulation of entire population down to the scale of single individuals. A bunch of recent studies has offered critical information regarding disease transmission model on complex networks [4, 10]. The theory of network topology has made a considerable progress over the past two decades that comes up with some unified framework to examine the transmission patterns of several epidemics. In particular, Pastor et al. revealed the fact that epidemic threshold of an SIS model tends to be 0 in scale-free (SF) networks, which is quite different from the traditional epidemic threshold theory and leading to a large volume of relevant studies [11,12]. Zhang et al. confirmed the existence of a unique global periodic solution if the basic reproduction number is greater than one while a scale-free SIS epidemic model is employed [13]. Bacear et al. developed a mathematical model that considers seasonality of the vector-borne population to estimate the basic reproduction number [14]. A significantly large variation in seasonality of a vector-borne population generates a periodic solution as demonstrated by London et al. [15]. Another interesting study pointed out the occurrence of a sequence of periodic-doubling bifurcations when the amplitude of seasonal variations increases considerably [16].

Epidemic compartmental models classified the entire population into several compartments to investigate the transmission dynamics of infectious diseases depending up the current health status, serving as an incredibly powerful tool for detecting, understanding, and combating outbreaks. Since the onset of COVID-19 pandemic, SIR compartmental model alongside its several variants have been the cutting-edge approach of investigating its transmission behavior. These epidemic compartmental models are formulated by a system of ordinary differential equations (ODEs), which are then distinguished by a set of parameters that are not known prior to the study and required to be identified from the relevant data set. These compartmental models thus require parameter estimation to fix the flow rate from one compartment to other. Yet, most of the previous studies assume constant values for the model parameters to minimize the complexity of epidemic modeling. Although numerous research efforts deeply focus on parameter estimation employing several well-known techniques, these methods still suffer from significant limitations which impede their diverse implementations. One such common limitation observed in parameter estimation is that the computational cost of numerical simulations rises exponentially with the complexity of parameters and models. Additionally, these parameter estimation methods are best suitable for time-constant cases that in turn fail to comprehend the sophisticated dynamics of infectious diseases over time in real-world contexts. Artificial intelligence(AI) based deep neural network (DNN) entailed with mathematical epidemiology plays a crucial role in comprehending the intricate dynamics of infectious diseases, providing a comprehensive framework for simulating diverse scenarios and thereby predicting the future trajectories of the epidemic propagation in real-world cases [17, 18]. Recently, deep learning has emerged as a new dimension of machine learning, and since then has been examined and deployed in a wide variety of research topics. Deep learning contains a series of machine learning algorithms delicately fed with inputs in the form of multi-layered

models. The models are typically neural networks comprising of varying degree of nonlinear operations.The machine learning algorithms used to learn from these deep neural networks by extracting distinct features and information [19]. The integrated impact of these mechanisms has proven its worth in analyzing and fighting against the transmission of COVID-19 epidemic [20]. Despite having its greater acceptability to data fitting as well as facilitating short-term prediction, it still has some weaknesses that can hinder its practical implications. First off, it is unable to find the disease transmission pattern and therefore, cannot suggest any reasonable predictions due to its inherent limitations. In fact, these AI-backed models heavily rely on the quality and quantity of the data set. At times, it might not be that much helpful given that the available data do not capture the reality. Therefore, exploring how to combine compartmental models with the AI tools can enhance their performance is a promising topic for contemporary research.

Besides, mathematical modeling dovetailed with evolutionary game theory has become a popular approach in recent times to study the behavioral dynamics of recurring epidemics [21–23]. Identifying the transmission patterns and fixing the social responses are crucial attempts with a view to containing the epidemic propagation and guiding policymakers to make timely decisions [24]. Proper implementation of game-theoretic models help analyze the perceived risks and benefits of adopting a particular health intervention. A good number of previous works have employed the concept of imperfect vaccination and defense against contagion to portray the evolutionary consequences more prominently [25–27]. Compiling these fundamental concepts into a single framework brings incredible behavioral responses on health interventions. Numerous precursor studies have been dedicated to investigating human behaviors and transmission dynamics, relying on game-theoretic approaches [28]. A complete eradication of epidemics using health provisions is quite challenging or sometimes impossible without having additional incentives as suggested by previous studies [29]. To model human interactions, most of the researchers relied on either structured or unstructured population [30]. Meanwhile, a couple of contemporary studies illustrated defense against contagion with a reduced transmittance rate [10, 31, 32]. In reality, strategy adoption is heavily influenced by individual decision-making, and has a deep interaction with the spatial transmission pattern triggered by underlying mobility networks [33]. For successful implementation of health interventions, it is of utmost importance to notify infections much earlier to limit its further expansion [34]. Underlying networks, imitation dynamics, strategy selection, information propagation, public opinion, and different optimization techniques also play crucial roles in model prediction [35]. In broader sense, temporal population models having stochastic nature can illustrate intrinsic behaviors of epidemic propagation [36].

To this end, motivated by all these archetype studies, we propose a novel epidemic model equipped with DNN-based computational simulations that help such global efforts to understand the disease dynamics, to estimate the associated key transmission parameters as well as to make further improvements for controlling the rapid transmission of epidemics like COVID-19. Here, the epidemic spreading is studied using the Susceptible-Infected-Recovered (SIR) model. In response to the rapid virus propagation, social and political responses to such epidemics need to be dynamic. Majority of the earlier models assumed insignificant incubation periods, where susceptible people become infectious and get recovered. Compartmental epidemic models can take a realistic phenomenon like latent infection period into account as disease transmission largely depends on warning indicators. Health officials and policymakers can warn people regarding the occurrence of epidemics. Accordingly, individuals choose to follow the recommended health rules to avoid being infected. A chicken-type game class is employed to estimate payoffs coming from strategy pair. Replicator dynamics help study the illness status and warning indicators. The signal-SIR model loops pandemic announcements and infected population responses. It redistributes risk between those who follow rules and those who do not during the course of pandemic. Finally, combining the replicator equations with SIR dynamics this paper presents a complete theoretical investigation.

The remaining of this paper is outlined as follows. In section 2, we formulate the proposed framework of the epidemic game model coupled with DNN structure, governed by a system of differential equations. Section 3 conducts a variety of computational experiments. We present the simulation results based on the estimated parameters, and a detailed discussion is made on the obtained results focusing on the key aspects of evolutionary game outcomes. Finally, section 4 draws a holistic summary of the proposed scheme and highlights some of the crucial findings of the current study.

2 Model formulation

2.1 Mathematical structure of the Signal-SIR model

An epidemic can quickly spread through a population in which susceptible, infected, and cured individuals coexist. In this model, susceptible agents (S-agents) can adopt either an adherence (AD) or non-adherence (NAD) strategy. The AD strategy helps prevent infection. The policymaker's warnings and S-agents' awareness determine their choice of strategy. NAD agents are susceptible to infection by I agents, which may later recover and transition to the R category. To slow down the transmission of an epidemic, people could choose to follow the rule before each time step (for example, a day, week or month) without enforcement. This would be called a time-step update in which higher adherence to health standards evolves without coercion. Throughout the pandemic, policymakers should adjust epidemic signals in accordance with changing seasons. The propagation of an illness during an epidemic season i is modeled using time steps of length T, starting from $t = i$. Repeat $\Delta(t)$ until $t = i+1$. The pandemic scenario determines the policy cycle or time window for T. The current state of epidemic knowledge cannot be readily discarded by policymakers. Depending on how long the policymaker release window is, the S-population may be able to switch strategies whenever it likes.

There are three key reasons why the signal-SIR Model is considered the optimal approach to address the challenge of epidemic signal importation. Initially, it is imperative for the policymaker to effectively communicate precise indications in order to establish stability within the epidemic among the S-population, thereby promoting self-interested decision-making. Furthermore, the implementation of S-population strategies, such as AD or NAD tactics, will have an impact on the epidemic situation and may prompt policymakers to disseminate a new signal. Finally, the signal-SIR model solver can adjust its input parameters to achieve near-optimal policy efficiency. The problem of addressing the epidemic change is mapped to the optimal policy signal problem. The current state of the signal-SIR model can be represented by the tuples (S, I, AD, NAD, R), where S, I, and R denote the proportions of susceptible, infected, and recovered individuals, respectively. The input to the system can be obtained by standardizing the initial value of the model state. Here, $S + I + R = 1$ and $AD + NAD = S$. Also if x_i denotes each decision then $x_i \in \{\tau, \beta\}$ where, τ ={Severe, Common, Mild} and β ={emission, outbreak, epidemic, pandemic}. The government formulates a signal based on epidemic situations, selecting appropriate values of τ and β for each time period. The initial assumption of the model assumes that the AD/NAD strategies of the population are uniformly distributed within the S-population and that the infected population (I) accurately represents real-world active cases. Susceptible individuals exhibit an avoidance behavior proportion of $x(t)$. According to research in Ref. [37], the vaccination decision-making process follows a two-stage iterative framework. Initially, individuals decide whether to comply with health standards, and they reassess their adherence in subsequent periods (see Figure 2.1).

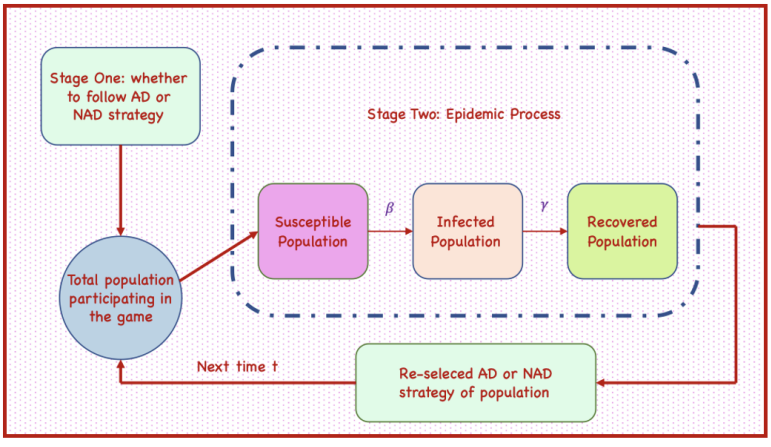


Figure 2.1: Health regulations are determined on a case-by-case basis. The right box provides an explanation of the process by which the epidemic spreads when the S-population with AD strategy either increases or decreases. The bottom section details the re-selected AD or NAD approach, which is established by consideration of the user's risk preferences.

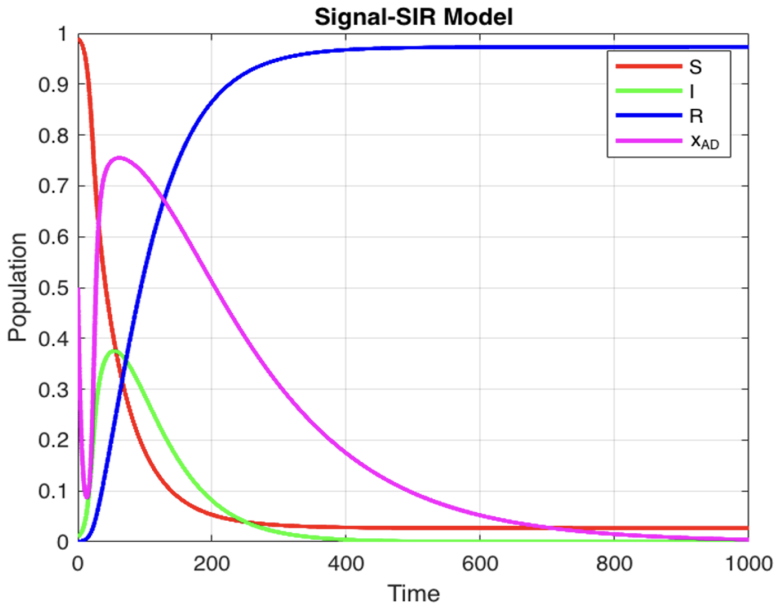


Figure 2.2: Time evolution of the Signal-SIR model.

The cost of any protective measures or income loss due to compliance with the AD strategy is denoted by c_{AD}. The widespread adoption of the AD strategy within the S-population plays a dominant role in controlling the epidemic during its second stage. To keep things simple, we will suppose that people are given the opportunity to participate in the decision-making process before an epidemic breaks out, and those who comply with health regulations (AD strategy) contribute to infection prevention. The dynamics of disease can be described using the SIR model, where the daily transmission is represented by the parameter β, and the recovery rate is denoted by γ. Initial proportions of susceptible, infected, and recovered individuals are denoted by S_0, I_0, and R_0, respectively. A risk payout matrix in a chicken game is used to determine the cost parameters, including c_{AD} and c_I, which fall within the range $(0, 1)$ [38]. The option μ represents the disease awareness of the warning indicator and is defined as $\mu = \frac{c_{AD}}{\alpha}$, where $0 < \mu < 1$. Additionally, the severity of epidemic spread is indicated by τ, which is related to the ratio of payoffs α to c_I within the range $0 < \tau \leq 1$. In this context, the government issues public health advisories in anticipation of potential outbreaks.

The payoff matrix for the proposed model can be shown in the following table:

Table 2.1: Payoff Matrix

	AD	NAD
AD	$-c_{AD}$	$-\alpha$
NAD	0	$-c_I$

The AD strategy fraction of the S-population is represented as x_{A_D}, while the NAD strategy fraction is given by $x_{NAD} = 1 - x_{A_D}$. The following items provide the well-mixed anticipated risk payoffs for the two different strategies:

$$p_{AD}(t) = S(t)x_{AD}(-c_{AD}) + S(t)(1 - x_{AD})(-\alpha) \tag{2.1}$$

$$p_{NAD}(t) = S(t)(1 - x_{AD})(-c_I) \tag{2.2}$$

Individuals with NAD who are at risk of contracting infections should take into account the affected component between the times $(t, t+1)$. According to Glaubitz et al. [39], this factor is expressed as $1 - e^{-\beta I(t)}$, where β represents the seasonal transmission rate. As a result, we may restate the expected value of risk for AD or NAD players as-

$$\pi_{AD}(t) = p_{AD} \tag{2.3}$$

$$\pi_{NAD}(t) = p_{NAD}(1 - e^{-\beta I(t)}) \tag{2.4}$$

Replicator dynamics yields the following equations:

$$\frac{dx_{AD}}{dt} = \bar{\omega} x_{AD}(t)(1 - x_{AD}(t))S(t)tanh(\kappa\Gamma(t)) \tag{2.5}$$

$$\Gamma(t) = -c_{AD}x_{AD}(t) + (1 - x_{AD}(t))(c_I(1 - e^{-\beta I(t)}) - \alpha) \tag{2.6}$$

where the responsiveness parameter $\bar{\omega}$ regulates the rate at which the health regulation behaviour is updated. the rationality parameter κ determines the likelihood of switching strategies in response to risk-reward perceptions. A higher κ indicates a greater tendency to switch strategies based on observed payoffs. For our simulations, we set $\kappa = 1$ and $\bar{\omega} = 0.5$. In game theory, the dynamics depend on perceived rather than actual risks and rewards [40]. The dynamics of our model are described by these ordinary differential equations (ODEs):

$$\frac{dS}{dt} = -\beta(1 - x_{AD}(t))S(t)I(t) \tag{2.7}$$

$$\frac{dI}{dt} = \beta(1 - x_{AD}(t))S(t)I(t) - \gamma I(t) \tag{2.8}$$

$$\frac{dR}{dt} = \gamma I(t) \tag{2.9}$$

$$\frac{dx_{AD}}{dt} = \bar{\omega} x_{AD}(t)(1 - x_{AD}(t))S(t)tanh(\kappa\Gamma(t)) \tag{2.10}$$

If an additional compartment is introduced, the equations take the following form:

$$\frac{dS}{dt} = -\beta(1 - x_{AD}(t))S(t)I(t) \tag{2.11}$$

$$\frac{dE}{dt} = \beta(1 - x_{AD}(t))S(t)I(t) - \sigma I(t) \tag{2.12}$$

$$\frac{dI}{dt} = \sigma E(t) - \gamma I(t) \tag{2.13}$$

$$\frac{dR}{dt} = \gamma I(t) \tag{2.14}$$

$$\frac{dx_{AD}}{dt} = \bar{\omega} x_{AD}(t)(1 - x_{AD}(t))S(t)tanh(\kappa\Gamma(t)) \tag{2.15}$$

Furthermore, the term $\Gamma(t)$ in the equations above is reformulated with $\delta(t)$ as:

$$\delta(t) = (-\mu x_{AD}(t) + (1 - x_{AD}(t)) \left(\frac{1}{\tau}(1 - e^{-\beta I(t)}) - 1 \right) \tag{2.16}$$

The parameter τ represents a strategic measure to prevent an epidemic, with policymakers adjusting it to increase player payoffs and encourage AD strategy adoption within the S-population. For instance, if the epidemic reaches a critical level, policymakers may reduce τ to enhance risk perception and promote preventive behavior. μ is also a helpful indicator for understanding psychological reactions in the AD population. A lower μ suggests that an AD player is more concerned about his opponent's NAD option and has an subjective belief that it poses a high risk if his opponent chooses NAD. It is evident that health regulations play a crucial role in controlling disease outbreaks. However, in many Western countries, social complexities make adherence voluntary rather than mandatory [41, 42]. Compliance in voluntary health programs is influenced by factors such as disease severity, transmission rates, and individual perceptions of risk. Furthermore, adherence to health regulations not only protects individuals but also helps safeguard their communities from potential infections [43].

2.2 An overview of the Signal-SIR coupled with DNNs

Deep neural networks (DNNs) can be utilized to model the dynamics and parameters in the Signal-SIR system. In this context, the neural networks serve as data-driven approximations for the time-varying parameters

in the system, such as β and γ. These parameters will be represented as Θ, the set of learnable weights in the DNN [44–48]. To guide the DNN to learn the optimal parameters, a loss function is defined based on the difference between the observed data and the model's predictions. The loss combines multiple components for the Susceptible, Infected, Recovered, and Adherence populations:

$$\text{Loss} = w_s \cdot \text{Loss}_S + w_I \cdot \text{Loss}_I + w_R \cdot \text{Loss}_R + w_D \cdot \text{Loss}_R + w_{AD} \cdot \text{Loss}_{AD} + \lambda \left\|\Theta\right\|^2 \tag{2.17}$$

where $w_s \cdot \text{Loss}_S$, $w_I \cdot \text{Loss}_I$, $w_R \cdot \text{Loss}_R$ and $w_{AD} \cdot \text{Loss}_{AD}$ represent the squared error between the predicted and the observed values of $S(t)$, $I(t)$ and x_{AD} respectively. For instance, the loss for the susceptible population could be written as:

$$\text{Loss}_S = \frac{1}{N} \sum_{n=0}^{N-1} \left[S_{n+1} - S_n - f(S_n, I_n, \Theta(t_n))\right]^2 \tag{2.18}$$

Here, $\Theta = \{\theta_\beta, \theta_\gamma, \theta_\mu\}$ are time-varying parameters that have been learned by the DNN. Each part of the loss function measures how well the model predicts the dynamics of the epidemic under the current parametrization [42]. To find the optimal parameters Θ, we use optimization algorithms such as Stochastic Gradient Descent (SGD). The parameters are updated iteratively as:

$$\Theta^{(k+1)} = \Theta^k - \alpha_k \nabla_\Theta L(t; \Theta^k) \tag{2.19}$$

where α_k is the learning rate at the k-th iteration, and $L(t; \Theta^k)$ is the loss function at time t.

Algorithm for the DNN-based Signal-SIR Model

Below is the high-level algorithm for training the DNN-based Signal-SIR model.
Input: Simulated data for $S(t)$, $I(t)$, $R(t)$ and $x_{AD}(t)$, initial values for $\beta(t)$, $\gamma(t)$, and $\tau(t)$.

1. **Build the model:**
   - Use DNN to approximate the parameters $\beta(t)$, $\gamma(t)$, and $\tau(t)$, which are represented as $\Theta = \{\theta_\beta, \theta_\gamma, \theta_\tau\}$.
   - Solve the Signal-SIR model using the Runge-Kutta method.
   - Define the loss function.
2. **For each epoch in** `max_epoch`:
   - Compute the forward pass: Calculate $S(t)$, $I(t)$, $R(t)$, and $x_{AD}(t)$ using the current values of Θ.
   - Update Θ using gradient descent or other optimization techniques.

Output: Time-varying parameters $\beta(t)$, $\gamma(t)$, $\tau(t)$, and $x_{AD}(t)$.
Deep neural networks (DNNs) can be utilized to model the dynamics and parameters in the Signal-SIR system. In this context, the neural networks serve as data-driven approximations for the time-varying parameters in the system, such as β and γ. These parameters will be represented as Θ, the set of learnable weights in the DNN.

3 Result and discussion

After conducting an epidemiological study, health officials will be able to determine whether the pandemic virus poses a threat or not. Epidemic evaluations are performed based on the degrees of risk. The propagation is classified as emission, outbreak, epidemic, or pandemic (β=0.1, 0.5, 0.7, 0.9) is related to the infectious rate (see Ref. [49]). In order to make an epidemic announcement based on an epidemiological survey, the parameters α and c_I in the risk payoffs matrix are merged and presented. This helps ensure that our findings are as accurate as possible. In a similar fashion, the other value of μ will be determined by the matrix of risk payoffs. When the parameters are normalized, the signal-SIR epidemic model has been expressed with the constraints $S = S_0, I = I_0, R = R_0$, and $S_0 + I_0 + R_0 = 1$ at $t = 0$, with $S_0 = 0.99, I = 0.01$, and $R = 0$. It is anticipated that within a city, only certain individuals will be severely affected, while others may remain susceptible but eventually recover. Furthermore, we assume $x_{AD} = 0.5$, considering that the strategy selection

among vulnerable individuals follows a normal distribution of AD (adaptive) and NAD (non-adaptive) behaviors.

We incorporate evolutionary game behavior dynamics into the compartmental epidemic model. Figure 3.1 shows the percentage of affected people throughout time. Warning indications reduce the fraction of infected individuals in the model. In Figure 3.1, increasing τ spreads the virus. τ=1 suggests an outbreak under normal conditions.

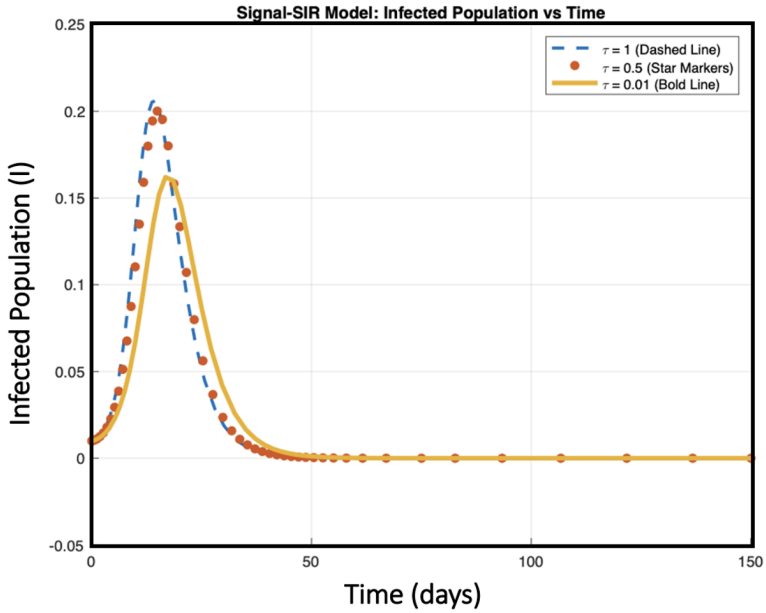
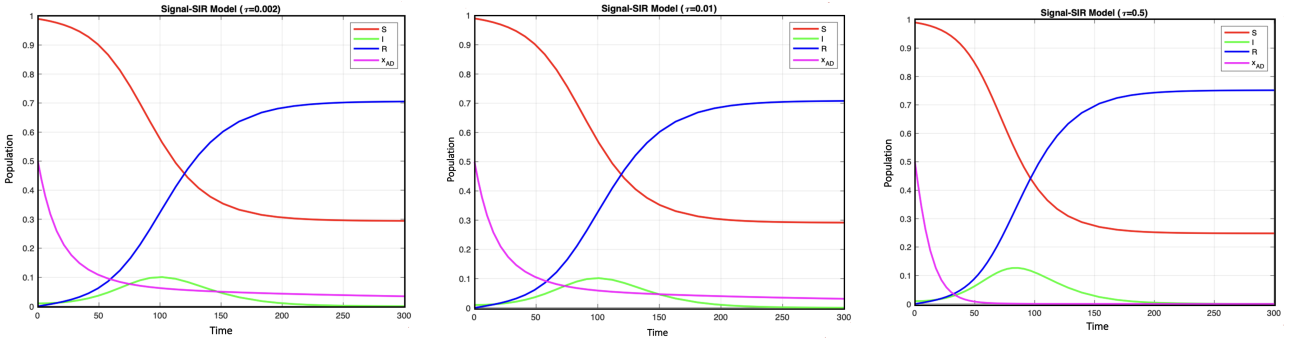


Figure 3.1: Time evolution of the Signal-SIR model.

Figure 3.2 illustrates the effect of different τ values (0.002, 0.01, 0.5) on the progression of an epidemic within the Signal-SIR model, which includes Susceptible (S), Infected (I), Recovered (R), and Adaptive (X_{AD}) populations. Lower τ values (e.g., $\tau = 0.002$) result in slower transitions to adaptive behavior, leading to higher peaks in infections and slower containment of the epidemic. Conversely, higher τ values (e.g., $\tau = 0.5$) indicate stronger adherence to adaptive strategies, significantly reducing the peak of infections, increasing recovery rates, and suppressing the epidemic more effectively.



(a) $\tau = 0.002$ (b) $\tau = 0.01$ (c) $\tau = 0.5$

Figure 3.2: Different values of τ for $\omega = 0.25$, $\beta = 0.14$, $\gamma = 0.075$.

As expected, the lowest τ emergency sign works best. The final equilibrium epidemic size (FES), shown by $R(\infty)$ in Figure 3.3, is plotted against warning signs. According to the graphs, all of the models eventually settle into a steady state. In Figure 3.4, the evolution of infected individuals over time is presented for varying values of β (0.1, 0.3, 0.5, 0.7, 0.9) and τ (0.05, 0.1, 0.3). These values reflect the impact of adherence to health guidelines. In Figure 3.4a, low transmission rates result in a gradual increase in infections. For small τ (0.05),

growth is moderate, while larger τ values (0.1, 0.3) significantly flatten the curve, indicating that the number of infected individuals starts to become more constant throughout the time. In Figure 3.4b, as β increases, infection peaks become more pronounced, with higher τ values reducing the peak and flattening the curve, mitigating outbreak severity. In Figures 3.4c-3.4e, high transmission rates gradually lead to quick, sharp peaks in infections. Larger τ values demonstrate a flattening effect, but the disease spreads faster initially, especially when the value of τ is smaller. Overall, higher adherence to health guidelines (larger τ) particularly reduces peak infections and delays outbreak progression, especially at high transmission rates.

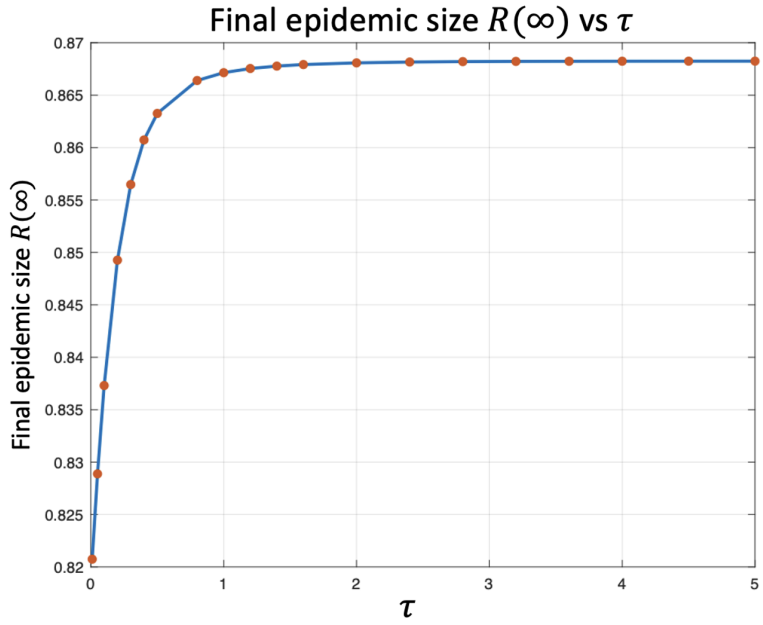
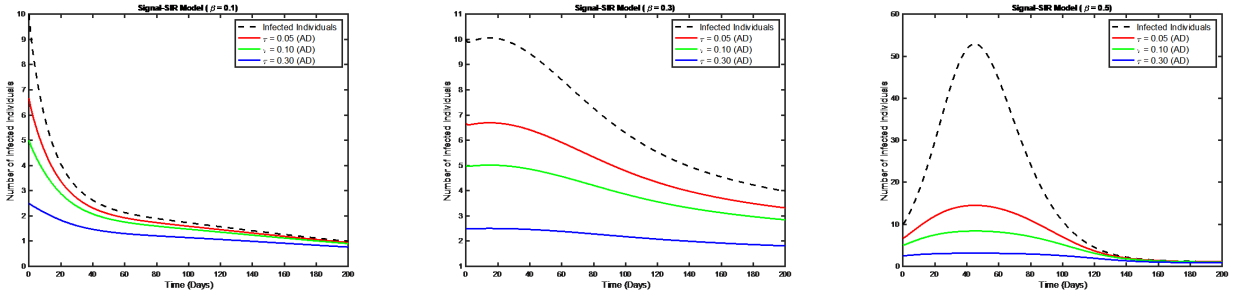
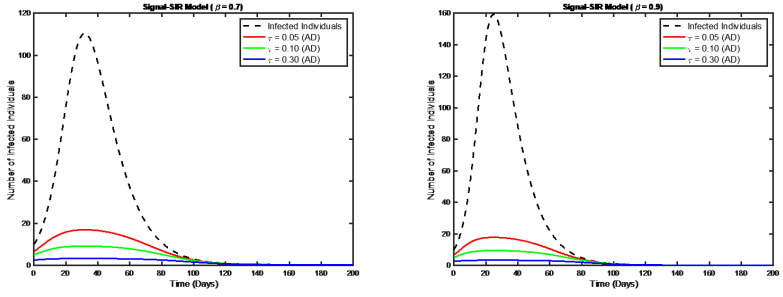


Figure 3.3: $R(\infty)$ vs warning signs. Results are shown for $\beta = 0.7, \gamma = 0.3$, and $\bar{\omega} = 0.8$.



(a) $\beta = 0.1$ (b) $\beta = 0.3$ (c) $\beta = 0.5$



(d) $\beta = 0.7$ (e) $\beta = 0.9$

Figure 3.4: Impact of β and τ on the number of infected individuals in the Signal-SIR model.

Assuming realistic human behavior, the policymaker expects individuals to follow healthy rules and obtain higher-risk benefits over time, despite the costs associated with adaptive behavior. In an adverse epidemic scenario, it is imperative for policymakers to adjust their policies in order to decrease the value of τ. Alternatively, they may opt to regulate it to a higher value in order to create a more relaxed environment. The utilization of a reference value, denoted as τ^*, could potentially provide valuable insights for policy formulation within the context of game theory. In chicken game, an estimation for the value of τ^* can be obtained by equating π_{AD} to π_{NAD}. The given information is provided by the following equation:

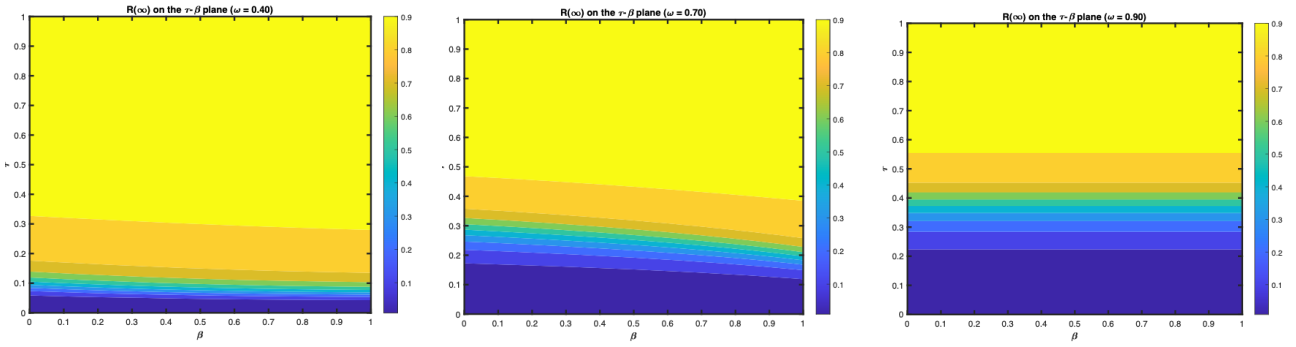
$$\tau^* = \frac{1 - e^{-\beta I(t)}}{1 + \mu \frac{x_{AD}}{x_{NAD}}} \tag{3.1}$$

When dealing with a severe illness, it is advantageous to have a higher ratio of x_{AD} to x_{NAD} in order to limit the spread of the epidemic to a specific extent, particularly when the value of τ^* is reduced. Moreover, Equation 3.1 can be expanded into an alternative mathematical expression in the way that follows:

$$\frac{x_{AD}}{x_{NAD}} = \frac{\frac{1-e^{-\beta I(t)}}{\tau} - 1}{1 - \omega} \tag{3.2}$$

It is evident that a larger portion of the S-population adopts the AD strategy when the value of ω increases.

Figure 3.5 portrays the $\tau - \beta$ phase diagram, showing the final ratio of recovered individuals, $R(\infty)$, for different levels of disease awareness parameter ω (0.4, 0.7, and 0.9). Each panel corresponds to a specific value of ω: (a) $\omega = 0.4$, (b) $\omega = 0.7$, and (c) $\omega = 0.9$. As shown, the final epidemic size (FES) increases significantly with higher transmission rates (β) and lower adaptive behavior (τ). For low ω (Figure 3.5a), adaptive strategies have limited impact, resulting in higher recovery ratios, which indirectly indicate more widespread infections. In contrast, with moderate ω (Figure 3.5b), adaptation slightly suppresses the FES. For large ω (Figure 3.5c), stronger reliance on adaptive behavior substantially reduces infection spread, as seen by the lower $R(\infty)$ values. These results demonstrate that increasing τ and ω enhances the effectiveness of adaptive strategies in controlling the epidemic. Policymakers can leverage these findings to promote public health interventions, emphasizing awareness campaigns and adaptive behavior to mitigate the outbreak's impact.



(a) $\omega = 0.4$ (b) $\omega = 0.7$ (c) $\omega = 0.9$

Figure 3.5: The $\tau - \beta$ phase diagram shows disease awareness parameter values of 0.4, 0.7, and 0.9.

The results presented in Figures 3.6 and 3.7 highlight the capability of the deep neural network (DNN) model in simulating and analyzing infection dynamics. Figure 3.6 demonstrates the DNN training process, showing a consistent decline in both Root Mean Square Error (RMSE) and the loss function over 500 iterations. The decreasing RMSE and loss values reflect the model's convergence toward an optimal solution, ensuring improved prediction accuracy. This trend also indicates the model's stability, as evidenced by the shrinking confidence intervals, which affirm its ability to generalize well to unseen scenarios. Figure 3.7 illustrates the predicted infection spread over 100 days for different values of transmission rate β and emission severity τ. For lower τ values (e.g., $\tau = 0.05$), higher β leads to rapid infection spread with sharp peaks, while lower β results in slower, flatter curves. As τ increases (e.g., $\tau = 0.30$), the infection dynamics become more controlled, with reduced peaks and delayed spread even for higher β. These findings emphasize the critical role of adherence to public health policies and the effectiveness of mitigation strategies in flattening infection curves and reducing

the epidemic's overall impact.

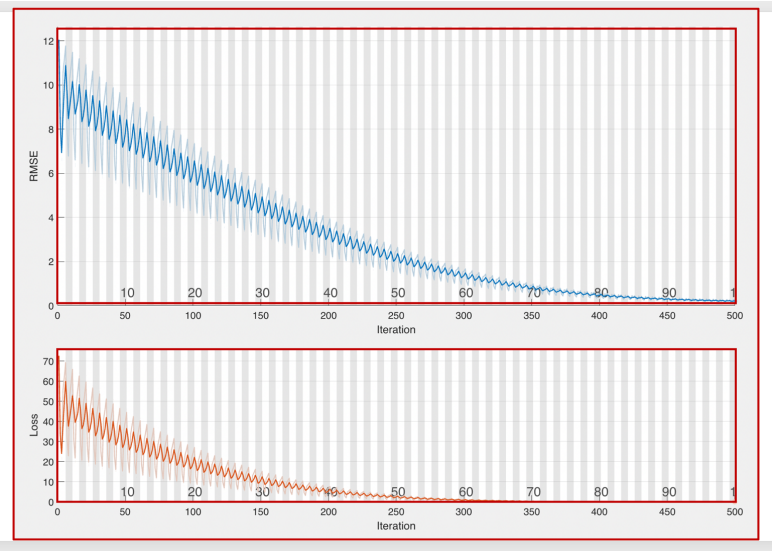
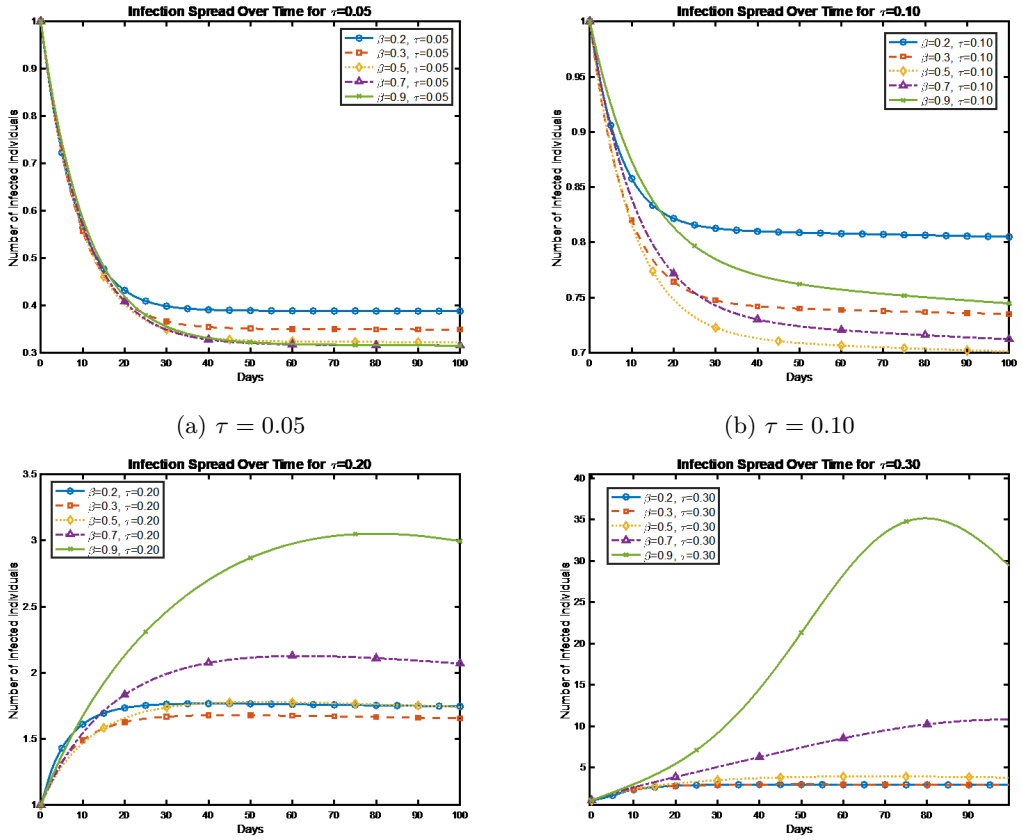


Figure 3.6: DNN training progress. It illustrates a decrease in RMSE and loss over 500 iterations, which indicates improved prediction accuracy.



(a) $\tau = 0.05$ (b) $\tau = 0.10$

(c) $\tau = 0.20$ (d) $\tau = 0.30$

Figure 3.7: Percentage of infection spread over 100 days for varying β and τ (predicted value).

4 Conclusion

In this study, we have demonstrated that integrating warning mechanisms, modeled through chicken games, into the traditional Susceptible-Infected-Recovered (SIR) model results in more complex epidemic dynamics. By incorporating key parameters such as τ, ω, β, and γ, the S-population can be guided effectively, adapting their behavior based on the evolving epidemic situation. In the context of a severe outbreak, these adaptive strategies—supported by timely and clear signals—can significantly reduce the transmission of the virus. Simulation results validate the effectiveness of these measures, showing that they facilitate the redistribution of risk payoffs between affected and unaffected populations, which in turn encourages the adoption of various strategies at different stages of the epidemic. Our findings underscore the importance of incorporating adaptable and flexible policy responses into epidemic models, as they can help control the spread of infectious diseases. The Signal-SIR Model offers a unique perspective by combining adaptive behavior with traditional epidemic dynamics, revealing that public adherence to health guidelines and an understanding of the severity of the epidemic are crucial for controlling infection rates. The use of deep neural networks enhances the model's predictive capabilities, showing that higher adaptive behavior leads to lower infection rates and better recovery outcomes. Future research should focus on applying this model to real-world data, refining parameter estimations, and validating its effectiveness in guiding public health responses during future outbreaks.

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Authors' contribution

Ishrat Shoily conceptualized the model, carried out the numerical simulations to validate the proposed model, constructed the pictorial diagrams, and made the original draft. Muntasir Alam contributed in writing the draft, analyzed the outcomes, visualized the model outcomes, helped in reviewing and editing the manuscript, critically revised the manuscript, and supervised the entire project.

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Declaration of competing interest

The authors have no conflicts of interest to declare. We clarify that the submission is original work and is not under review at any other publication.

Data sharing

There is no available data regarding this study. We do not analyze or generate any datasets, because our work proceeds within a theoretical and mathematical approach.

Ethical approval

No consent is required to publish this manuscript. All authors have given approval for publication and agreed to be held accountable for the work performed herein.

References

[1] Muntasir Alam, Masaki Tanaka, and Jun Tanimoto. A game theoretic approach to discuss the positive secondary effect of vaccination scheme in an infinite and well-mixed population. *Chaos, Solitons & Fractals*, 125:201–213, 2019.

[2] Joel Henrique Ellwanger, Ana Beatriz Gorini da Veiga, Valéria de Lima Kaminski, Jacqueline María Valverde-Villegas, Abner Willian Quintino de Freitas, and José Artur Bogo Chies. Control and prevention of infectious diseases from a one health perspective. *Genetics and Molecular Biology*, 44(1 Suppl 1):e20200256, 2021.

[3] William Ogilvy Kermack and Anderson G McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772):700–721, 1927.

[4] Romualdo Pastor-Satorras, Claudio Castellano, Piet Van Mieghem, and Alessandro Vespignani. Epidemic processes in complex networks. *Reviews of modern physics*, 87(3):925–979, 2015.

[5] Hanqi Zhang, Zhongkui Sun, and Yuanyuan Liu. Impact of asymptomatic cases and human mobility on epidemic propagation in an sais network model. *Nonlinear Dynamics*, 112(8):6761–6775, 2024.

[6] Zhongkui Sun, Yuanyuan Liu, Xiaoli Yang, and Wei Xu. Control of epileptic activities in a cortex network of multiple coupled neural populations under electromagnetic induction. *Applied Mathematics and Mechanics*, 44(3):499–514, 2023.

[7] Tae Jin Lee, Masayuki Kakehashi, and Arni SR Srinivasa Rao. Network models in epidemiology. In *Handbook of Statistics*, volume 44, pages 235–256. Elsevier, 2021.

[8] Thomas Kovac, Tom Haber, Frank Van Reeth, and Niel Hens. Heterogeneous computing for epidemiological model fitting and simulation. *BMC bioinformatics*, 19:1–11, 2018.

[9] Martin A Nowak and Robert M May. The spatial dilemmas of evolution. *International Journal of bifurcation and chaos*, 3(01):35–78, 1993.

[10] Muntasir Alam, Yuki Ida, and Jun Tanimoto. Abrupt epidemic outbreak could be well tackled by multiple pre-emptive provisions-a game approach considering structured and unstructured populations. *Chaos, Solitons & Fractals*, 143:110584, 2021.

[11] Mengfeng Sun, Haifeng Zhang, Huiyan Kang, Guanghu Zhu, and Xinchu Fu. Epidemic spreading on adaptively weighted scale-free networks. *Journal of mathematical biology*, 74:1263–1298, 2017.

[12] Zoltán Dezső and Albert-László Barabási. Halting viruses in scale-free networks. *Physical Review E*, 65(5):055103, 2002.

[13] Lei Zhang, Maoxing Liu, Qiang Hou, Asma Azizi, and Yun Kang. Dynamics of an sis network model with a periodic infection rate. *Applied Mathematical Modelling*, 89:907–918, 2021.

[14] Nicolas Bacaër and Souad Guernaoui. The epidemic threshold of vector-borne diseases with seasonality: the case of cutaneous leishmaniasis in chichaoua, morocco. *Journal of mathematical biology*, 53(3):421–436, 2006.

[15] Wayne P London and James A Yorke. Recurrent outbreaks of measles, chickenpox and mumps: I. seasonal variation in contact rates. *American journal of epidemiology*, 98(6):453–468, 1973.

[16] Joan L Aron and Ira B Schwartz. Seasonality and period-doubling bifurcations in an epidemic model. *Journal of theoretical biology*, 110(4):665–679, 1984.

[17] JA Sánchez-Monroy, Javier Riascos-Ochoa, and Abel Bustos. Parameter estimation in the stochastic sir model via scaled geometric brownian motion. *Chaos, Solitons & Fractals*, 189:115626, 2024.

[18] Connor Shorten, Taghi M Khoshgoftaar, and Borko Furht. Deep learning applications for covid-19. *Journal of big Data*, 8(1):1–54, 2021.

[19] Ying Xie, Linh Le, Yiyun Zhou, and Vijay V Raghavan. Deep learning for natural language processing. In *Handbook of statistics*, volume 38, pages 317–328. Elsevier, 2018.

[20] Long Nguyen, Maziar Raissi, and Padmanabhan Seshaiyer. Modeling, analysis and physics informed neural network approaches for studying the dynamics of covid-19 involving human-human and human-pathogen interaction. *Computational and Mathematical Biophysics*, 10(1):1–17, 2022.

[21] Jun Tanimoto. *Sociophysics approach to epidemics*, volume 23. Springer, 2021.

[22] Jun Tanimoto. Evolutionary games with sociophysics. *Evolutionary Economics*, 17, 2019.

[23] Jun Tanimoto. *Fundamentals of evolutionary game theory and its applications*. Springer, 2015.

[24] Matjaž Perc, Jillian J Jordan, David G Rand, Zhen Wang, Stefano Boccaletti, and Attila Szolnoki. Statistical physics of human cooperation. *Physics Reports*, 687:1–51, 2017.

[25] Kazuki Kuga and Jun Tanimoto. Which is more effective for suppressing an infectious disease: imperfect vaccination or defense against contagion? *Journal of Statistical Mechanics: Theory and Experiment*, 2018(2):023407, 2018.

[26] Muntasir Alam, Kazuki Kuga, and Jun Tanimoto. Three-strategy and four-strategy model of vaccination game introducing an intermediate protecting measure. *Applied Mathematics and Computation*, 346:408–422, 2019.

[27] Yoshiro Iwamura and Jun Tanimoto. Realistic decision-making processes in a vaccination game. *Physica A: Statistical Mechanics and its Applications*, 494:236–241, 2018.

[28] Umma Kulsum, Muntasir Alam, and Md Kamrujjaman. Modeling and investigating the dilemma of early and delayed vaccination driven by the dynamics of imitation and aspiration. *Chaos, Solitons & Fractals*, 178:114364, 2024.

[29] Ana Perisic and Chris T Bauch. Social contact networks and disease eradicability under voluntary vaccination. *PLoS computational biology*, 5(2):e1000280, 2009.

[30] Eriko Fukuda, Jun Tanimoto, and Mitsuhiro Akimoto. Influence of breaking the symmetry between disease transmission and information propagation networks on stepwise decisions concerning vaccination. *Chaos, Solitons & Fractals*, 80:47–55, 2015.

[31] Yuki Ida and Jun Tanimoto. Effect of noise-perturbing intermediate defense measures in voluntary vaccination games. *Chaos, Solitons & Fractals*, 106:337–341, 2018.

[32] Carlos P Roca, José A Cuesta, and Angel Sánchez. Evolutionary game theory: Temporal and spatial effects beyond replicator dynamics. *Physics of life reviews*, 6(4):208–249, 2009.

[33] Sheryl L Chang, Mahendra Piraveenan, and Mikhail Prokopenko. Impact of network assortativity on epidemic and vaccination behaviour. *Chaos, solitons & fractals*, 140:110143, 2020.

[34] Muntasir Alam and Jun Tanimoto. A game-theoretic modeling approach to comprehend the advantage of dynamic health interventions in limiting the transmission of multi-strain epidemics. *Journal of Applied Mathematics and Physics*, 10(12):3700–3748, 2022.

[35] Md Rajib Arefin, Yuichi Tatsukawa, and Jun Tanimoto. Evolution of cooperation under the coexistence of imitation and aspiration dynamics in structured populations. *Nonlinearity*, 36(5):2286, 2023.

[36] Vadim A Karatayev, Madhur Anand, and Chris T Bauch. Local lockdowns outperform global lockdown on the far side of the covid-19 epidemic curve. *Proceedings of the National Academy of Sciences*, 117(39):24575–24580, 2020.

[37] Hai-Feng Zhang and Zhen Wang. Suppressing epidemic spreading by imitating hub nodes' strategy. *IEEE Transactions on Circuits and Systems II: Express Briefs*, 67(10):1979–1983, 2019.

[38] Ilan Fischer, Shacked Avrashi, Tomer Oz, Rabab Fadul, Koral Gutman, Daniel Rubenstein, Gregory Kroliczak, Sebastian Goerg, and Andreas Glöckner. The behavioural challenge of the covid-19 pandemic: indirect measurements and personalized attitude changing treatments (impact). *Royal Society open science*, 7(8):201131, 2020.

[39] Alina Glaubitz and Feng Fu. Oscillatory dynamics in the dilemma of social distancing. *Proceedings of the Royal Society A*, 476(2243):20200686, 2020.

[40] Marco A Amaral, Marcelo M de Oliveira, and Marco A Javarone. An epidemiological model with voluntary quarantine strategies governed by evolutionary game dynamics. *Chaos, Solitons & Fractals*, 143:110616, 2021.

[41] KM Ariful Kabir and Jun Tanimoto. Evolutionary game theory modelling to represent the behavioural dynamics of economic shutdowns and shield immunity in the covid-19 pandemic. *Royal Society open science*, 7(9):201095, 2020.

[42] Min Zhou, Piao Long, Nan Kong, and Kathryn S Campy. Characterizing wuhan residents' mask-wearing intention at early stages of the covid-19 pandemic. *Patient Education and Counseling*, 104(8):1868–1877, 2021.

[43] Pedro D Manrique, Chen Xu, Pak Ming Hui, and Neil F Johnson. Atypical viral dynamics from transport through popular places. *Physical Review E*, 94(2):022304, 2016.

[44] Sifan Wang, Hanwen Wang, and Paris Perdikaris. On the eigenvector bias of fourier feature networks: From regression to solving multi-scale pdes with physics-informed neural networks. *Computer Methods in Applied Mechanics and Engineering*, 384:113938, 2021.

[45] Zhi-Qin John Xu, Yaoyu Zhang, Tao Luo, Yanyang Xiao, and Zheng Ma. Frequency principle: Fourier analysis sheds light on deep neural networks. *arXiv preprint arXiv:1901.06523*, 2019.

[46] Nasim Rahaman, Aristide Baratin, Devansh Arpit, Felix Draxler, Min Lin, Fred Hamprecht, Yoshua Bengio, and Aaron Courville. On the spectral bias of neural networks. In *International conference on machine learning*, pages 5301–5310. PMLR, 2019.

[47] Xi-An Li, Zhi-Qin John Xu, and Lei Zhang. Subspace decomposition based dnn algorithm for elliptic type multi-scale pdes. *Journal of Computational Physics*, 488:112242, 2023.

[48] Matthew Tancik, Pratul Srinivasan, Ben Mildenhall, Sara Fridovich-Keil, Nithin Raghavan, Utkarsh Singhal, Ravi Ramamoorthi, Jonathan Barron, and Ren Ng. Fourier features let networks learn high frequency functions in low dimensional domains. *Advances in neural information processing systems*, 33:7537–7547, 2020.

[49] Survival dynamical systems: individual-level survival analysis from population-level epidemic models, author=KhudaBukhsh, Wasiur R and Choi, Boseung and Kenah, Eben and Rempała, Grzegorz A. *Interface focus*, 10(1):20190048, 2020.