# Dynamics of Epidemic Phases in Adaptive SIS model on a Small-World Network

Antibar G. Angie, Gutiérrez H. Héctor
Physics Department
Universidad Nacional de Colombia

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**Abstract**

The spread of infectious diseases is a critical area of research in epidemiology, with models like the Susceptible-Infected-Susceptible (SIS) framework playing a central role in understanding disease dynamics. However, traditional SIS models assume static networks, which do not reflect real-world behaviors where individuals alter their connections in response to infection. This study focuses on an adaptive SIS model implemented on a small-world network, where susceptible nodes reconnect only with previously known susceptible nodes. Using Monte Carlo simulations, we explore how varying reconnection and infection rates impact the dynamics of disease spread, including the prevalence and lifespan of infection. Our results reveal how these parameters influence the transition between healthy and endemic phases, offering new insights into phase stability and network evolution.

***Keywords***— Complex Networks, Adaptive Dynamical Networks, adaptive SIS model, Epidemic Models, Small-World Network

## 1 Introduction

The spread of infectious diseases has long been a critical focus in epidemiology, where models such as the Susceptible-Infected-Susceptible (SIS) model have provided valuable frameworks for studying disease transmission [1]. These classical models typically assume static contact networks, where individuals' interactions remain constant throughout the course of an epidemic. However, in real-world scenarios, individuals frequently adapt their behavior to avoid contact with infected people, leading to dynamic changes in their social networks. This discrepancy between static assumptions and dynamic behavior has driven a shift towards adaptive network models, which incorporate the co-evolution of the network structure with disease dynamics, offering a more realistic representation of disease spread. [2]

The rewiring behavior has been shown to significantly raise the epidemic threshold, alter disease prevalence, and even lead to bistable states where endemic and healthy populations coexist [3]. Specifically, this contributes to a more comprehensive understanding of the mechanisms through which diseases propagate. Our work builds upon this existing body of research by exploring the specific influence of reconnection behaviours on epidemic dynamics. In our adaptive SIS model, susceptible nodes only reconnect with individuals they were previously linked to in the original network, but who are now also susceptible. This approach reflects real-world scenarios where individuals adjust their social contacts to minimise exposure while maintaining familiar relationships. It is important to note that this approach has not been explored in the existing literature. There are, however, some similar approaches in Song et al. (2019) with their link-reconnection strategies [4].

Building on the advances in adaptive networks and their application to epidemiological models, our work seeks to further explore these dynamics by focusing on several key objectives. Specifically, we aim to characterize the infection dynamics in our adaptive SIS model, examining how changes in reconnection and infection rates influence the overall behavior of the disease. By investigating how these parameters affect critical aspects such as the lifespan of the infection and the prevalence of infected individuals, we aim to deepen our understanding of epidemic processes in adaptive networks. Furthermore, we strive to evaluate the qualitative consistency of our model with existing adaptive SIS models in the literature, ensuring that our results align with established findings while offering new insights into phase stability and

transitions between endemic and healthy states.

## State of the Art

- *SIS Model*:

  The SIS (Susceptible-Infected-Susceptible) model, foundational in epidemiology, was first introduced by Kermack and McKendrick in 1927. This model represents diseases where recovery does not grant lasting immunity, capturing the cyclical infection dynamics[5]. The model was later expanded to consider network structures, a pivotal development by Pastor-Satorras and Vespignani in 2001. Their work on epidemic spreading in scale-free networks demonstrated that network topology significantly influences epidemic thresholds, with diseases potentially persisting in heterogeneous networks without a critical infection rate. [6]

- *Adaptive Dynamical Networks*:

  The concept of adaptive networks, where network topology evolves along with the state dynamics of its nodes, marked a significant advance in the understanding of complex systems. The pioneering work by Thilo Gross in 2006 introduced the idea of adaptive rewiring in networks, where susceptibles avoid contact with infected nodes by modifying their connections. This mechanism significantly alters the network structure and epidemic dynamics, introducing phenomena such as assortative degree correlation and bistability. [3]

  In 2008, Gross and Blasius published a comprehensive review titled "Adaptive Coevolutionary Networks" provided a comprehensive exploration of how networks and dynamics coevolve. They highlighted key examples across multiple fields where coevolutionary dynamics are present, emphasizing in the importance of feedback between the nodes states and the network structure. This work laid the foundation for numerous studies on coevolutionary networks across different domains. [7]

  Following this, in 2009, the book "Adaptive Networks: Theory, Models and Applications", edited by Thilo Gross and Hiroki Sayama, offered a comprehensive exploration of this burgeoning field. The book includes contributions from experts across multiple disciplines, covering topics such as real-world applications of adaptive networks, self-organization mechanisms, and the role of adaptive networks in epidemiology and social systems. Particularly notable is the section on Contact Processes and Epidemiology on Adaptive Networks, which delves into how adaptive rewiring can alter the course of epidemic outbreaks by changing the underlying network topology dynamically during the spread of disease. [8]

  Together, these works have laid the foundation for much of the modern research in adaptive network theory. Recently, the review by Berner et al. (2023) provided a synthesis of current developments in the field, emphasizing the broad applicability of adaptive dynamical networks in domains such as neuroscience, social systems, and epidemiology. They highlighted how modern adaptive network models can capture complex dynamics such as synchronization, multistability, and explosive transitions in epidemics, illustrating the versatility of these networks in modeling real-world phenomena. This review marks a significant step forward by integrating advancements in the mathematical tools used to analyze adaptive networks, making it a crucial reference for ongoing research in coevolutionary dynamics across various disciplines. [9]

- *Adaptive Networks in SIS Models*

  The application of adaptive network theory to epidemiological models began with the study of Gross in 2006, which remains a cornerstone in the field. This study demonstrated that adaptive rewiring could significantly raise the epidemic threshold by isolating infected individuals from susceptibles, leading to bistable states where both healthy and endemic states can coexist. This showed how changes in network topology, induced by the spread of disease, can create phenomena such as hysteresis and limit cycles, profoundly impacting epidemic dynamics. [3]

  Building on these foundational insights, Van Segbroeck et al. (2010) expanded the understanding of adaptive networks by investigating how dynamic contact reshaping affects disease infectiousness and spread. Their study revealed that healthy individuals, by severing links with infected contacts and maintaining connections with other healthy individuals, can significantly slow disease transmission. This dynamic reshaping of networks, driven by local information about individuals' health status, alters the effective infectiousness of a disease, making populations appear more resistant by adjusting the contact structure in real-time. [10]

  In recent years, the importance of simulation techniques in capturing these adaptive processes has grown. Tian Gan (2021), in their master's thesis, extended these concepts to continuous-time simulations of the SIS model. This work compared discrete-time and continuous-time simulations, concluding that continuous-time models, based on the Gillespie algorithm, more accurately capture the transitions between metastable states, such as disease-free, endemic, and bistable phases, particularly in adaptive networks. [1]

  Further work on the application of adaptive network theory to SIS models has considered the inclusion of weighted links. Song et al. (2019) introduced a model that accounted for

heterogeneous weighted networks. Their research demonstrated that the weights of connections play a crucial role in disease dynamics. [4]

Building on the advances in adaptive networks and their application to epidemiological models, our work seeks to further explore these dynamics by focusing on several key objectives. Specifically, we aim to characterize the infection dynamics in our adaptive SIS model, examining how changes in reconnection and infection rates influence the overall behavior of the disease. By investigating how these parameters affect critical aspects such as the lifespan of the infection and the prevalence of infected individuals, we can deepen our understanding of the epidemic processes within adaptive networks. Additionally, we strive to evaluate the qualitative consistency of our model with other adaptive SIS models in the literature, ensuring that our results align with established findings while offering potential new insights. Furthermore, we will observe the phases that emerge in our simulations, particularly focusing on endemic and healthy states, and compare the stability of these phases under varying reconnection and infection rates. This comparison will help elucidate how our model relates to and extends the current work on adaptive SIS models, particularly in terms of phase stability and transitions.

# 2 Theoretical Framework

## Complex Networks

Complex networks are mathematical structures, essential tools to model and analyze the interconnections present in various real-world systems. From biological ecosystems and social interactions to technological infrastructure and communication systems, networks offer a framework to understand how elements (nodes) interact with each other through connections (edges) giving rise to non-trivial behaviors. The structure of these networks plays a crucial role in shaping their function. For example, the topology of social networks affects the spread of information and disease. [11].

In particular, networks have been instrumental in studying phenomena such as epidemic spreading, where nodes can represent individuals, and edges represent the potential for disease transmission. Complex networks provide a deeper understanding of how diseases propagate in structured populations compared to simple homogeneous mixing models.

### Small-World Networks

One prominent type of network is the small-world network, characterized by a high local clustering of nodes and short average path lenghts between any two nodes. The small-world property is crucial in many real-world systems, such as biological or social networks, where individuals tend to form tightly connected groups, but are also linked to distant parts of the network through a few intermediary connections. This structure captures the essence of many phenomena, from the spread of information to the transmission of diseases. [12]

The most widely studied small-world model was introduced by Watts and Strogatz in 1998. Known as Watts-Strogatz graph, this model strikes a balance between regularity and randomness. It starts with a regular lattice where each node is connected to its nearest neighbors and gradually introduces randomness by rewiring each edge with a probability $p_{ws}$; this parameter controls the transition from a regular to a random network and plays a critical role in determining the network's structural properties. This unique combination of clustering and short paths makes the Watts-Strogatz model particularly suitable for simulating phenomena that rely on both local and global interactions, such as the spread of diseases, where infection can rapidly move through clustered groups while still being able to jump across distant parts of the network. [12]

## Adaptive Dynamical Networks

The significance of complex networks extends beyond static systems, giving rise to what are known as complex dynamical networks, the study of interconnected dynamical systems on temporally evolving networks has gained increasing importance in various fields. In this approximation, two main approaches are emphasized: temporal dynamical networks, where the network structure evolves over time based on a predetermined schedule, and adaptive dynamical networks, where the network structure coevolves with the dynamic state of the nodes [9]. Here, we focus on the second approach, adaptive dynamical networks.

Adaptive dynamical networks center on the co-evolution between the network's topology and the dynamics occurring within the network. Unlike traditional models that examine only the process propagating through the network, adaptive networks allow for a two-way interaction: the network's structure changes based on the dynamical states of the nodes, and these changes, in turn, influence the ongoing dynamics [2]. This co-evolutionary approach is crucial for modeling systems where both the topology (structure) and function are interdependent.

One key area where this approach has been applied is epidemiology, specifically through the adaptive SIS (aSIS) model. This model captures how both the spread of disease and the connectivity of the network co-evolve, since adaptive networks consider the feedback loop between the network topology (its structure) and the process through it, in this case, the spread of a contagion [13]. The ability of network connectivity to evolve based on infection dynamics allows for a more realistic approach to studying diseases, where individuals

can modify their connections (creation and breakage of links) depending on their susceptibility or infection status, specifically creating and breaking links [7].

## Epidemiology Context

The study of epidemics is crucial for public health, especially focused on understanding the phases of an epidemic, commonly divided into healthy, epidemic and endemic states, which can serve as the basis for effective policies and isolation strategies to control the spread of disease. Each phase represents a different level of disease prevalence within a population and requires different public health responses. In the healthy phase, the disease is largely absent, while in the epidemic phase, the infection spreads rapidly, often overwhelming healthcare systems. As an epidemic progresses, it may become endemic, meaning that the disease persists at a steady level within a population. Understanding these phases helps guide interventions like quarantine, isolation, and vaccination strategies to mitigate outbreaks. [14]

While various epidemiological models have been developed to capture different aspects of disease dynamics, here we focus on the SIS (Susceptible - Infected - Susceptible) model. Despite the availability of other models such as SIR, SIRS, and SEIS, the SIS model is particularly suited for infections where individuals do not gain lasting immunity after recovery. In the SIS framework, individuals can move back and forth between being susceptible and infected, making it a valuable tool for studying diseases that allow for reinfection, such as bacterial infections like gonorrhea or viral infections such as flu [15]. This simplicity enables a clearer exploration of the relation between dynamic and structure in the aSIS model, where connectivity between individuals evolves in response to the ongoing infection.

# 3 Adaptive SIS Model

The implemented model corresponds to an adaptive variant of the SIS model (Susceptible-Infected-Susceptible). In this approach, in addition to the two possible states of the nodes, the links between them can be activated or deactivated according to specific rules.

The model contemplates four fundamental processes: infection, recovery, distancing, and reconnection.

- Infection occurs with probability $p_{inf}$ in a susceptible node, provided that it is connected to an infected node.
- Recovery occurs in any infected node with probability $p_{recov}$.
- An infected node can choose to break the link to a susceptible node with probability $p_{break}$.
- Each susceptible node can reconnect with another susceptible node with a probability $p_{create}$. This can only occur between two initially connected susceptible nodes.

It is important to note that the distancing process, in which an infected node breaks the link with a susceptible node, is equivalent to a susceptible node deciding to disconnect an infected node. This interpretation is more consistent with an attitude of self-care on the part of the susceptible node, by avoiding possible infections.

## 3.1 Probabilities and effective rates

The dynamics of the model depend on the probabilities selected for each process. The probabilities associated with the node state reflect the particular characteristics of the infectious disease, while the probabilities related to the links represent the severity of the distancing strategies. In addition, higher probabilities generally indicate longer time intervals over which processes occur.

The comparison between $p_{inf}$ and $p_{recov}$, as well as between $p_{break}$ and $p_{create}$, defines intervals of interest where infection (or link-breaking) rates exceed or are exceeded by recovery (or reconnection) rates. Following Tian Gan's approach, effective infection $\tau$ and link-breaking $\omega$ rates are defined. [1]

$$\tau = \frac{p_{inf}}{p_{recov}}, \quad \omega = \frac{p_{break}}{p_{create}}$$

These effective rates are justified from the relation $p \approx \frac{dp}{dt}\Delta t$ for low probabilities. They are called rates because they approximate to a fraction between probability rates.

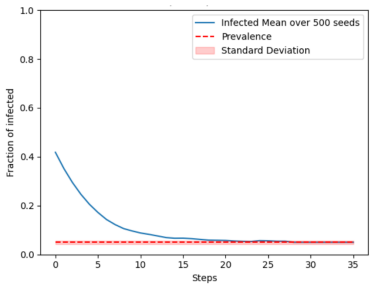
It is intended to study some global behavior for different $\tau$ and $\omega$. To do this, the probabilities $p_{recov}$ and $p_{create}$ are fixed in 0.2, and $p_{inf}$ and $p_{break}$ varies between 0 and 1, so $\tau$ and $\omega$ varies from 0 to 5.

## 3.2 Implementation

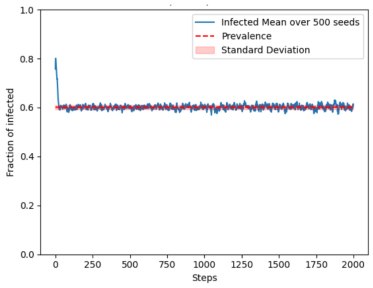
The classical SIS model is generally studied by means of first-order differential equations, using the mean-field approximation. This approach has been used by several authors, who have extended the analysis to adaptive models, incorporating additional terms that describe the adaptive rules, which increases the complexity of the analysis.

In this study, the model is implemented using a cellular automaton, in which all processes occur simultaneously. This feature can generate behaviors that, at first glance, seem counterintuitive. For example, a susceptible node may connect to a node that has just been infected or disconnect from a node that has just recovered. However, these events are plausible in real scenarios, especially when infection or recovery times are comparable to the times at which individuals perceive changes in the health status of their close contacts.

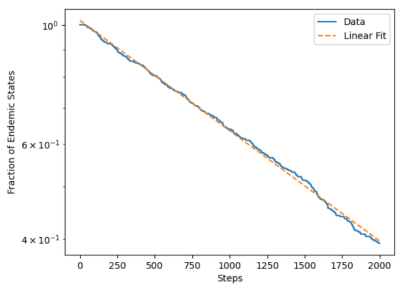
The automaton was run for 2000 steps, repeating 500 times over a Watts-Strogatz network with $N = 20$ nodes,




(a) Metaestable prevalence and mean infection for $\tau = 0.1$.




(b) Metaestable prevalence and mean infection for $\tau = 2.6$.




(c) Fraction of runs in endemic state vs time.

Figure 1: Average infected fraction, and the fraction of runs still endemic vs time, for 500 runs. For all, the effective breaking rate was $\omega = 1$ in a Watts-Strogats network with $N = 20$, $k = 4$, and $p_{WS} = 0.2$. (a) is in a healthy phase while (b) is in an epidemic one. The inverse of the coefficient in (c) is the Characteristic Lifetime, in this case is $T = 2117$ for $\tau = 2.6$.

an average degree $k = 4$ and a reconnection probability $p = 0.2$. In addition, the recovery probability ($p_{recov}$) and link breakage ($p_{break}$) were set to 0.2.

# 4 Analysis and Discussion

Figure 1 presents the average behavior of the fraction of infected nodes and the fraction of simulations in which infected nodes still exist at a given step. These plots highlight two main features of each model evolution: (i) the average fraction of infected nodes (prevalence) that remains constant over time and (ii) the time at which the epidemic is completely extinguished.

## About prevalence

The prevalence of infected was calculated as the time average of the fraction of infected individuals while they remain in a metastable state, as shown in Figures 1a and 1b. This approach allowed us to obtain the metastable prevalence of infected by averaging multiple simulations.

A remarkable behavior is the dependence of the metastable prevalence with the effective infection rate $\tau$, for different values of $\omega$. Figures 2a and 2b show that these curves start with a prevalence close to zero until they reach a point where they start to grow, and then stabilize in a straight line with a low slope. These two regions, where the prevalence remains almost constant with respect to $\tau$, correspond to the phases of the system.

The first region, called the healthy phase, is characterized by a rapid drop in prevalence to a value close to zero, as illustrated in Figure 1a. On the other hand, the region associated with larger values of $\tau$, where prevalence stabilizes, corresponds to the endemic phase. In this phase, the disease persists with a high average number of infected, and its lifetime is longer than in the healthy phase by several orders of magnitude. The intermediate points between these two phases can be interpreted as a phase transition; however, according to Gross [3], this region is called a bistable phase, since at some points in this transition both phases are supported under an approximate analytical solution of his model.

The value of $\tau$ at which the healthy phase ends is called the epidemic threshold. As seen in Figure 2, this threshold increases with $\omega$, but decreases with the average lattice degree $k$. This suggests that temporal distancing is effective not only in reducing the average infected prevalence, but also in reaching a healthy phase.

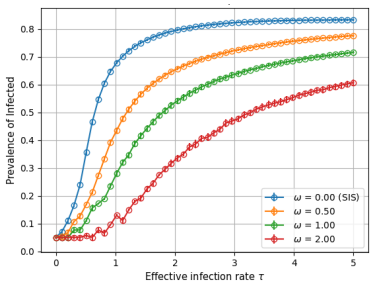
However, for diseases with higher transmission rates, the behavior is different. Figure 4b presents a heat map in $\tau - \omega$ space for $k = 8$. In this case, if the disease is highly infectious, distancing strategies may not be sufficiently effective. In these scenarios, it may be more appropriate to implement measures that reduce the degree of k, such as blocking certain transmission channels to control the spread of the disease.

## 4.1 About Lifetime

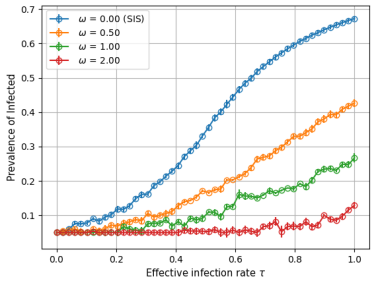
During each simulation run, it was observed that at some step all infected individuals recovered, eventually causing the network to return to its initial state. From this point, where the fraction of infected individuals is zero, is called the lifetime of the epidemic. This time varies between different simulations and can show considerable variability.

To illustrate this phenomenon, Figure 1c shows the fraction of endemic states, i.e. those in which the disease has not yet disappeared. The exponential decay of this quantity makes it possible to define a characteristic lifetime $T$, which is analogous to the average lifetime in other dynamical systems. This time is expressed by the following relation:

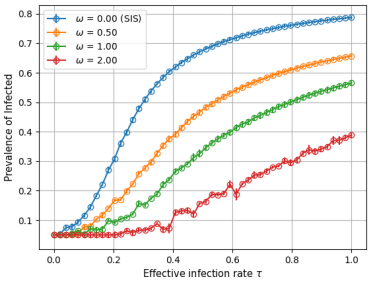
$$f_{endemic} \propto e^{-t/T},$$




(a) Prevalence vs $\tau$ $(0, 5)$, for four $\omega$. The mean degree is $k = 4$.




(b) Prevalence vs $\tau$ $(0, 1)$, for four $\omega$. The mean degree is $k = 4$.



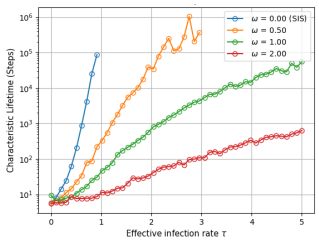

(c) Prevalence vs $\tau$ $(0, 1)$, for four $\omega$. The mean degree is $k = 8$.

Figure 2: Prevalence vs efective infection rate $\tau$, for different efective breaking rate. For all the figures, the simulation was run over 500 times in 2000 steps, in a Watts-Strogatz network with N=20 and $p_{WS} = 0.2$. Here, the healthy phase for $\tau s$ near to zero and the endemic phase for highest $\tau s$, the first one is characterized by lower and constant prevalences, while the other one for longer and almost constant.

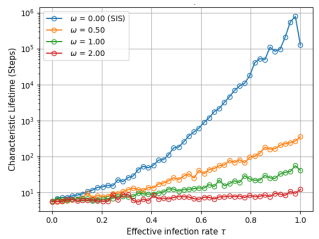
$T$ depends on both $\tau$ and $\omega$. Furthermore, this relationship indicates that the distribution of lifetimes follows an exponential distribution, since:

$$\begin{aligned} f_{endemic}(t) &= P(t_{LT} < t) \\ &= \sum_{t'=0}^{t} P(t_{LT} = t') \\ &\approx \int_0^t \rho_{LT}(t')dt' \end{aligned}$$

This implies that the distribution of lifetimes $\rho_{LT}$ is approximately the derivative of the fraction of endemic states.




(a) $\tau$ in the interval $(0, 5)$.




(b) $\tau$ in the interval $(0, 1)$.

Figure 3: Characteristic Lifetime vs efective infection rate $\tau$, for four different efective breaking rate. For all the figures, the simulation was run over 500 times in 2000 steps, in a Watts-Strogatz network with $N = 20$, $k = 4$ and $p_{WS} = 0.2$.

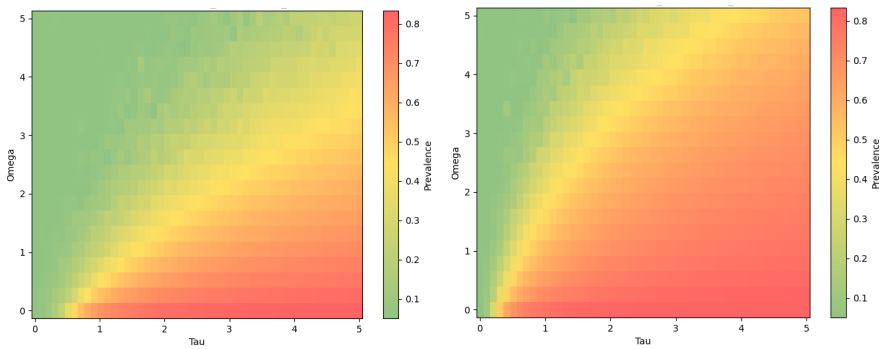
Figure 3 presents the characteristic lifetimes $T$ as a function of $\tau$ for different values of $\omega$. Unlike the prevalence plots, no obvious phase transitions are observed here. The behavior of the characteristic lifetime for different $\tau$ follows an approximately exponential pattern. Thus, although there are clear differences in prevalence that allow us to identify phases in the system, the lifetimes show a much more regular behavior.

## 4.2 About the topology

Figure 4 illustrates the heat maps for three Watts Strogatz reconnection probabilities. Figure 4c, with $p_{WS}$ equal to zero, corresponds to a ring network, 4d, with $p_{WS}$ equal to one, represents a random network, and 4a employs the fixed probability utilized in the analyses presented in this paper $p_{WS} = 0.2$. As illustrated in the graphs, the epidemic threshold is discernible in the color gradient. A transition from green to red prevalence signifies a shift in epidemic phases. The yellow prevalence represents a transition between these two phases, which can be identified as healthy and endemic, respectively. The epidemic threshold can be identified as the point at which the prevalence transitions from green to yellow.

Although the three networks display disparate clustering indices and mean path lengths, the three maps exhibit similar behaviors. A slight shift to the left is observed for $p_{WS} = 1$, though this change has no significant implications for the phases of the epidemic. The discrepancy in $\tau$ between $p_{WS} = 0$ and $p_{WS} = 1$ is practically inconsequential.

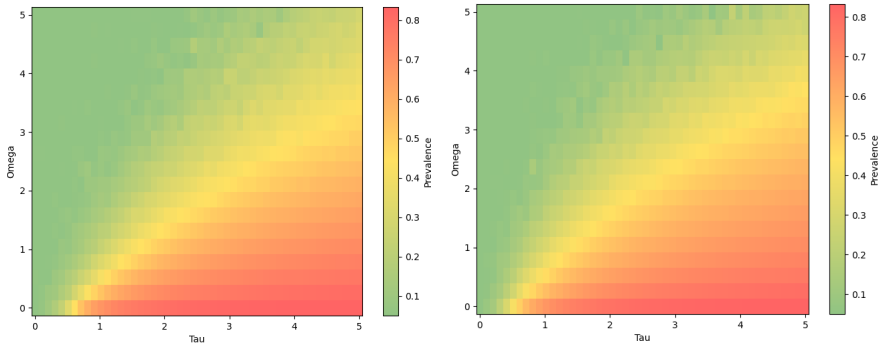
This apparent independence of small-world topology may be attributed to the tendency of nodes to become disconnected during adaptive evolution, which diminishes the influence of the base (or initial) network topology. Additionally, the diminished impact of small-world effects on smaller networks ($N = 20$) may contribute to this observation, for example, the difference in mean path length between regular and random networks is lower




(a) $k = 4$ and $p_{WS} = 0.2$.




(b) $k = 8$ and $p_{WS} = 0.2$.




(c) $p_{WS} = 0$ (regular network) and $k = 4$.




(d) $p_{WS} = 1$ (random network) and $k = 4$.

Figure 4: Heatmaps of the metastable prevalence of infected nodes for different configurations of $k$ and $p_{WS}$. There is a notable difference where $k$ is varied, but not when $p_{WS}$.

than 1.

# 5 Conclusions

Our adaptive SIS model successfully reproduces two distinct phases: healthy and endemic. These phases are clearly identified by the average number of infected individuals, with their prevalence dependent on the effective infection rate, $\tau$, link breakage rate, $\omega$, and the network's average degree, $k$. The interplay between these parameters dictates the transitions between the healthy and endemic states, and the dynamics within each phase.

In the healthy phase, the number of infected individuals remains low, and infections quickly die out. The prevalence of infected nodes shows little variation with respect to $\tau$ and $\omega$ until the system reaches a critical point, suggesting a phase transition related with the epidemic threshold. In contrast, the endemic phase is characterized by a stable, high prevalence of infections. Although minor fluctuations occur, the overall infection level remains relatively constant as $\tau$ and $\omega$ change, and epidemics persist for extended periods. Interestingly, the lifetime of epidemics within the endemic phase exhibits a stochastic behavior, following an exponentially decreasing distribution.

The analysis of the characteristic lifetime reveals that, while it correlates with $\tau$, it does not reflect the clear phase distinctions seen in infection prevalence. This exponential relationship suggests that while lifetime is not a decisive metric for identifying phase transitions, its study could shed light on the mechanisms influencing epidemic persistence and decay.

Furthermore, our results suggest that for small networks (such as the 20-node network studied), the phase boundaries are largely independent of the small-world topology. However, it is recommended for future research explore larger networks, where factors like clustering and the average path length between nodes may play a more significant role in phase transitions and epidemic spread.

This work contributes to the broader understanding of adaptive networks in epidemiology, particularly by showing how the reconnection of susceptible nodes to previously known susceptibles can impact both the stability and transitions between epidemic phases. Future studies may further refine these models, particularly by investigating how variations in network topology, larger network sizes, and different adaptive mechanisms influence epidemic dynamics.

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