# The Irrelevance of Influencers: Information Diffusion with Re-Activation and Immunity Lasts Exponentially Long on Social Network Models

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## Abstract

Information diffusion models on networks are at the forefront of AI research. The dynamics of such models typically follow stochastic models from epidemiology, used to model not only infections but various phenomena, including the behavior of computer viruses and viral marketing campaigns. A core question in this setting is how to efficiently detect the most influential vertices in the host graph such that the infection survives the longest. In processes that incorporate re-infection of the vertices, such as the SIS process, theoretical studies identify parameter thresholds where the survival time of the process rapidly transitions from logarithmic to super-polynomial. These results contradict the intuition that the starting configuration is relevant, since the process will always either die out fast or survive almost indefinitely. A shortcoming of these results is that models incorporating short-term immunity (or creative advertisement fatigue) have not been subjected to such a theoretical analysis so far.

We reduce this gap in the literature by studying the SIRS process, a more realistic model, which besides re-infection additionally incorporates short-term immunity. On complex network models, we identify parameter regimes for which the process survives exponentially long, and we get a tight threshold for random graphs. Underlying these results is our main technical contribution, showing a threshold behavior for the survival time of the SIRS process on graphs with large expander subgraphs, such as social network models.

## 1 Introduction

Various phenomena at the forefront of AI research focus on information diffusion models on graphs, e.g, (Sun, Cautis, and Maniu 2023; Jiang, Ren, and Ferrara 2023; Liu et al. 2023; Sun et al. 2022; Razaque et al. 2022; Sharma et al. 2021). The underlying graph processes come from a plethora of contexts, such as, spread of infections (Pastor-Satorras et al. 2015; Leskovec et al. 2007a) and computer viruses (Berger et al. 2005; Borgs et al. 2010), social influence and the spread of ideas (Kempe, Kleinberg, and Tardos 2003), and viral marketing campaigns (Agarwal and Liu 2008). A central question in this area is to find influential vertices, the ones that maximize the spread of the process (Babay et al. 2022; Ohsaka et al. 2014; Kimura, Saito, and Motoda 2009).



The foundation for analyzing such processes goes back to epidemiology (see (Pastor-Satorras et al. 2015) for an extensive survey). Epidemiology models each vertex of the host network to be in one of various states, such as *infected* or *susceptible*, and transitions between these states occur based on certain events.

The underlying epidemic models considered in AI research are diverse. A large portion of research is based on the stochastic models where each vertex gets infected only once in this process (Kempe, Kleinberg, and Tardos 2003; Leskovec et al. 2007a; Babay et al. 2022). However, as argued by Kimura, Saito, and Motoda (2009) there exist situations where individuals get re-infected. This can happen for example with diseases that do not grant immunity such as tuberculosis and gonorrhea (Newman 2003) or with bloggers who can post repeated messages about the same topic (Leskovec et al. 2007b).

Phenomena with re-infection are captured well by the SIS epidemic model—a fundamental model in epidemiology. The SIS process is a continuous-time Markov chain where each vertex is either susceptible or infected. Each infected vertex becomes susceptible at a normalized rate of 1 and infects each of its susceptible neighbors independently at an *infection rate* $\lambda$ (see left-hand side of Figure 1). Note that this is a model of endemic disease and, therefore, it can happen that the infection will survive for an extremely long time for some parameter regimes with respect to the host graph $G$ and $\lambda$. Thus, one of the most basic questions one can ask about the SIS process is *how long* it takes until the infection dies out, known as the *survival time* (sometimes also referred to as the *extinction time*).

Due to its importance and its nice mathematical properties, the SIS process is well understood from a mathematical point of view on many relevant networks. On Erdős–Rényi graphs, Nam, Nguyen, and Sly (2022) show that the survival time of the SIS process exhibits a threshold behavior (from logarithmic to super-polynomial) with respect to $\lambda$. This is also the case for scale-free networks[1] (Berger et al. 2005; Borgs et al. 2010). More generally, Ganesh, Massoulié, and Towsley (2005) connect the survival time to the spectral radius and the isoperimetric constant of the host graph, which immediately

[1]Generated by the preferential-attachment model (Barabasi and Albert 1999).

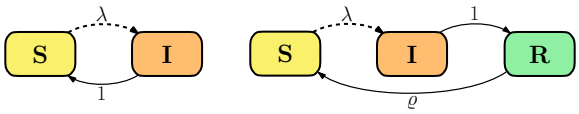


Figure 1: State transitions in the SIS model (left) and the SIRS model (right), with associated transition rates. The letters represent the states of being susceptible (S), infected (I), or recovered (R). Arrows with solid lines indicate that the transition is driven by one Poisson clock with the respective rate per vertex. The arrows with dashed lines between the susceptible and infected states indicate that there is one clock per edge. In the latter case, a susceptible vertex is infected if it has an infected neighbor and the clock that corresponds to their shared edge triggers.

translates to a variety of threshold values for simple graphs.

An interesting observation from the theoretical investigations of the SIS process is that, once beyond the threshold, the infection survives, in expectation, for a very long time in the network regardless of which vertices are initially infected. In most cases, even a single initially infected vertex is enough to result in a pandemic situation. These results showcase that the general structure of the network is far more important in the long survival of an infection than its starting state. This makes determining the relation between network structure and the survival time such an important topic.

While the SIS process is relevant, it makes the limiting assumption that once a vertex heals, it immediately becomes susceptible to re-infection. However, in various air-borne infections, such as the influenza or COVID-19, individuals become immune (or very resilient) to the infection for some period. This can also happen in the spread of computer viruses, when the infected servers get taken down for maintenance. Furthermore, studies have also shown that in digital marketing individuals may experience creative fatigue and ignore advertisements after a period of exposure (So, Kim, and Cohen 2017; Abrams and Vee 2007).

Phenomena that include temporary immunity are commonly modeled in epidemiology with the SIRS process, another continuous Markov chain where now each vertex is either susceptible, infected, or *recovered*. Each infected vertex becomes recovered at a normalized rate of 1 and infects each of its susceptible neighbors independently at an *infection rate* $\lambda$, while each recovered vertex becomes susceptible at a *deimmunization rate* $\varrho$ (see right-hand side of Figure 1). Thus, with a grain of salt, the SIS process can be viewed as a special case of the SIRS process in which recovered vertices turn immediately susceptible (that is, the deimmunization rate $\varrho$ is infinite).

Due to the relevance of the SIRS process, its survival time has been studied extensively in various aspects. This includes empirical results (Wang et al. 2017; Kuperman and Abramson 2001; Ferreira, Sander, and Pastor-Satorras 2016), mean-field approaches (Bancal and Pastor-Satorras 2010; Prakash et al. 2012), and results that consider deterministic variants of the process (Saif 2019) or generalized models (Prakash et al. 2012). However, surprisingly, to the best of our knowledge, no rigorous theoretical results for the SIRS process exist that do not directly follow from established results for the SIS process. That is, we are lacking deep mathematically rigorous insights that already exist for the SIS model.

### Main Contribution

We conduct the first rigorous, mathematical study of the expected survival time of the SIRS process on a large spectrum of graph classes, containing many models for social networks. Our results show that the network structure of the host graph is highly important whereas the set of initially infected vertices is not. Further, we uncover drastic differences between the SIS and the SIRS process but also large similarities.

Our key technical result (Theorem 5) shows that the expected survival time of the SIRS process on expander graphs is at least exponential in the number of vertices if the infection rate is greater than the inverse of the expander's average degree. Combining this with results for the SIS process that carry over to our setting (Ganesh, Massoulié, and Towsley 2005, Theorem 3.1) gives an almost tight threshold behavior of the SIRS process on expanders at $\lambda$. In other words, we pinpoint very precisely at which infection rate the survival time suddenly shifts from logarithmic to exponential.

In addition, we prove our result for expanders to carry over to supergraphs, which implies respective expected survival times for other well known graph classes, such as Erdős–Rényi graphs (Corollary 10) and complex networks exhibiting real-world properties (see, e.g., Corollary 14 for hyperbolic random graphs), which we discuss in Section 5.

Last, for star graphs, we prove an entirely different behavior of the SIRS process, further highlighting the immense importance of the network structure on the survival time. No matter the infection rate, the SIRS process on stars has at most a polynomial expected survival time (Theorem 1) if the deimmunization rate is constant. This is in strong contrast to the SIS process, where already very low infection rates result in a super-polynomial survival time (Ganesh, Massoulié, and Towsley 2005). Hence, being temporarily immune makes a drastic difference here.

Due to space limitations, for the complete proofs and all of the technical details, we refer the reader to the full version of the paper (Friedrich et al. 2022).

## 2 Preliminaries

The SIRS process is a continuous-time Markov chain on graphs in which the vertices change between different states, following events triggered by Poisson processes. We analyze how this process behaves asymptotically in the number of vertices $n$ of the graph. Especially, when we use big-O notation or refer to variables as constants, this is with respect to $n$. When we use big-O notation in a term of a relation, this means that there exists a function from the big-O expression such that the relation holds, for example, the equation $a = 2^{\Omega(n)}$ means there is a function $f \in \Omega(n)$ such that $a = 2^{f(n)}$. If not stated otherwise, all variables we consider may depend on $n$. Whenever we talk about Poisson processes, we refer to one-dimensional Poisson point processes that output a random subset of the non-negative real numbers.

We first define our infection models and some related terms that we use throughout the paper.

## Infection Processes

Let $G = (V, E)$ be a graph with vertex set $V$ and edge set $E$. Further, let $\lambda, \varrho \in \mathbb{R}_{>0}$. In the SIRS process, for each edge $e \in E$, we define a Poisson process $M_e$ with parameter $\lambda$, and for each vertex $v \in V$, we define the two Poisson processes $N_v$ with parameter 1 and $O_v$ with parameter $\varrho$. We refer to these processes as *clocks*, and when an event occurs in one of them, we say that the relevant clock *triggers*. We use $Z$ to denote the set of all of these clocks, that is, $Z = \big(\bigcup_{e\in E}\{M_e\}\big) \cup \big(\bigcup_{v\in V}\{N_v, O_v\}\big)$. Let $P$ denote the stochastic process in which all of the clocks in $Z$ evolve simultaneously and independently, starting at time 0. Note that almost surely there is no time point at which two clocks trigger at once. There are almost surely a countably infinite number of trigger times in $P$, which we index by the increasing sequence $\{\gamma_i\}_{i\in\mathbb{N}_{\geq 0}}$, where $\gamma_0 = 0$.

A SIRS process $C = (C_t)_{t\in\mathbb{R}_{\geq 0}}$ has an underlying graph $G = (V, E)$, an infection rate $\lambda$, a deimmunization rate $\varrho$, and an initial partition of $V$ into susceptible, infected, and recovered vertices with the respective sets $S'_0$, $I'_0$, and $R'_0$. Note that we do not need to specify a healing rate as we normalized that to 1. At every time $t \in \mathbb{R}_{\geq 0}$, the configuration $C_t$ is a partition of $V$ into $S'_t$, $I'_t$, and $R'_t$. The configuration only changes at times in $P$. Let $i \in \mathbb{N}_{>0}$. We consider the following configuration transitions in $\gamma_i$:

- If for some $e = \{u, v\} \in E$ we have $\gamma_i \in M_e$, $u \in I'_{\gamma_{i-1}}$, and $v \in S'_{\gamma_{i-1}}$, then $S'_{\gamma_i} = S'_{\gamma_{i-1}} \setminus \{v\}$, $I'_{\gamma_i} = I'_{\gamma_{i-1}} \cup \{v\}$, and $R'_{\gamma_i} = R'_{\gamma_{i-1}}$. We say that $v$ *gets infected* at time point $\gamma_i$ by $u$.
- If for some $v \in V$ we have $\gamma_i \in N_v$ and $v \in I'_{\gamma_{i-1}}$ then $S'_{\gamma_i} = S'_{\gamma_{i-1}}$, $I'_{\gamma_i} = I'_{\gamma_{i-1}} \setminus \{v\}$ and $R'_{\gamma_i} = R'_{\gamma_{i-1}} \cup \{v\}$. We say that $v$ *recovers* at time point $\gamma_i$.
- If for some $v \in V$ we have $\gamma_i \in O_v$ and $v \in R'_{\gamma_{i-1}}$, then $S'_{\gamma_i} = S'_{\gamma_{i-1}} \cup \{v\}$, $I'_{\gamma_i} = I'_{\gamma_{i-1}}$ and $R'_{\gamma_i} = R'_{\gamma_{i-1}} \setminus \{v\}$. We say that $v$ *gets susceptible* at time point $\gamma_i$.

If none of the above three cases occurs, the configuration of $C$ at $\gamma_i$ is the same as the configuration of $C$ at $\gamma_{i-1}$. Note that at all times between $\gamma_{i-1}$ and $\gamma_i$, $C$ retains the same configuration as in $\gamma_{i-1}$.

In our proofs, we only consider the time points in $P$ at which the configuration changes. To this end, let $P' = \{\gamma_0\} \cup \{\gamma_i \mid i \in \mathbb{N}_{>0} \wedge C_{\gamma_i} \neq C_{\gamma_{i-1}}\}$. We index the times in $P'$ by the increasing sequence $\{\tau_i\}_{i\in\mathbb{N}}$. For all $i \in \mathbb{N}$, we call $\tau_i$ the $i$-th *step* of the process.

If at any point in time no vertex is infected, then from that point onward, no vertex is infected. We say that the infection *dies out* or *goes extinct* at the first (random) time $T$ with $I'_T = \emptyset$. We call $T$ the *survival time* of the SIRS process.

We only keep track of the number of vertices in each of the sets. To this end, we define for all $t \in \mathbb{R}_{\geq 0}$ the random variables $S_t = |S'_t|$, $I_t = |I'_t|$, and $R_t = |R'_t|$. These random variables change depending on the clocks in $P$. We say that an event *happens at a rate of* $r \in \mathbb{R}_{>0}$ if and only if the set of clocks that cause this event when they trigger has a sum of rates equal to $r$.

We define the *projection* $C'$ of $C$ onto a subgraph $G'$ of $G$ as the process on $G'$ such that, at each point in time, each vertex of $G'$ in $C'$ is in the same state as it is in $C$. When considering such a projection, we use $S_t$, $I_t$, and $R_t$ to only count the vertices of $C'$ in the corresponding state. Also $\{\tau_i\}_{i\in\mathbb{N}}$ only contains times at which the state of a vertex in $C'$ changes. The survival time of a projected process is the first point in time that the projected process has no infected vertices. Note that the survival time $T'$ of $C'$ is a lower bound for the survival time $T$ of $C$, as all infected vertices of $C'$ are also infected in $C$.

We use stochastic domination to transfer results from one random variable to another. We say that a random variable $(X_t)_{t\in\mathbb{R}}$ *dominates* another random variable $(Y_t)_{t\in\mathbb{R}}$ if and only if there exists a coupling $(X'_t, Y'_t)_{t\in\mathbb{R}}$ in a way such that for all $t \in \mathbb{R}_{\geq 0}$ we have $X'_t \geq Y'_t$.

# 3 Expected Survival Time on Stars

While stars might seem to be a mundane graph class to study, they proved to be an essential building block in key results for the survival time of the SIS process on far more complex networks, such as social networks (Berger et al. 2005; Borgs et al. 2010) and Erdős–Rényi graphs (Bhamidi et al. 2021). The reason is that a star of sufficiently high degree already lets the SIS process survive for a super-polynomial time for very low infection rates. In more detail, on stars with $n$ leaves, an infection rate of $\Omega\big(n^{-1/2+\varepsilon}\big)$ leads to a super-polynomial expected survival time. This immediately translates to a lower bound of the expected survival time for more complex graphs that contain a star of suitable size.

Due to this importance of stars for the SIS process, we explore whether this proof strategy applies to the SIRS process as well. It turns out that this is not the case, as the SIRS process behaves significantly differently on a star. The following result shows that the SIRS process only survives on stars for a polynomial time in expectation, no matter the infection rate.

**Theorem 1.** *Let $G$ be a star with $n \in \mathbb{N}_{>0}$ leaves, and let $C$ be a SIRS process on $G$ with infection rate $\lambda$ and with deimmunization rate $\varrho$. Let $T$ be the survival time of $C$. Then for sufficiently large $n$, it holds that $\mathbb{E}[T] \leq \big(\ln(n) + 2\big)(4n^\varrho + 1) \in \mathrm{O}(n^\varrho \ln(n))$.*

Note that this bound is independent of $\lambda$ and that it results in a polynomial expected survival time as long as $\varrho$ is at most constant with respect to $n$. Although we only prove an upper bound, our bound matches, up to a logarithmic factor, empirical investigations of the star (Ferreira, Sander, and Pastor-Satorras 2016), suggesting that our bound is almost tight. Note that these experimental results consider the infection rate $\lambda$ to be constant in terms of $n$, while our results apply for any $\lambda$. Our results also show a behavior similar to the deterministic variant of the process considered by Saif (2019).

The detailed proof of Theorem 1 can be found in the full version of the paper (Friedrich et al. 2022), however, we give a high-level overview of our proof in the following. The

analysis mainly relies on the method of investigating independent phases in which the center is not infected, bounding the probability of the infection process dying out during that time, as is common (Borgs et al. 2010; Berger et al. 2005). A phase lasts at most until all leaves triggered their recovery at least once, which occurs in expectation after a time of about $\ln(n)$. Thus, if the center just recovered, it needs to become susceptible more quickly than that bound, as otherwise all leaves are recovered. Since deimmunization triggers at rate $\varrho$, the probability that the center does not become susceptible in this time interval is about $\mathrm{e}^{-\varrho \ln n}$, resulting in a probability of about $n^{-\varrho}$ that the infection dies out. Since these phases are independent, the infection process survives, in expectation, about $n^{\varrho}$ of these trials, each lasting about $\ln(n)$ time in expectation. By Markov's inequality, this bound on the survival time also holds with high probability.

Note that the deimmunization rate and the state *recovered* are important for this argument to hold. Without this additional state, that is, in the SIS process, it is quite likely that the center becomes quickly reinfected before all leaves are not infected, which leads to an exponential expected survival time once $\lambda \geq n^{-1/2+\varepsilon}$ in this setting (Ganesh, Massoulié, and Towsley 2005), for all positive constants $\varepsilon$.

## 4 Expected Survival Time on Expanders

Expander graphs find numerous application in a broad range of domains (Hoory, Linial, and Wigderson 2006; Krivelevich 2019), but perhaps the most relevant for our setting is their usage in the design of reliable communication networks. There are many notions of how to define expander graphs. For our main theorem we use algebraic expanders in which all but one of the eigenvalues of the normalized Laplacian of the graph are very close to 1. These graphs have some nice properties that let us bound the number of edges between infected and susceptible vertices. Formally, let $G = (V, E)$ be a graph with $n$ vertices $\{v_i\}_{i=1}^{n}$, and let $L$ be its normalized Laplacian, which is defined for all $i, j \in [n]$ as

$$L_{i,j} = \begin{cases} 1 & \text{if } i = j, \\ -\frac{1}{\sqrt{\deg(v_i)\deg(v_j)}} & \text{if } v_i \text{ and } v_j \text{ are adjacent,} \\ 0 & \text{otherwise.} \end{cases}$$

Let $L$ have eigenvalues $\lambda_1 \leq ... \leq \lambda_n$. The *spectral expansion* of $L$ is defined as $\delta = \max_{i \geq 2} |1 - \lambda_i|$. We call $G$ an $(n, (1 \pm \varepsilon_d)d, \delta)$-expander if and only if it has $n$ vertices, a spectral expansion of $\delta$ and only vertices with degree between $(1 - \varepsilon_d)d$ and $(1 + \varepsilon_d)d$.

For two vertex sets $X, Y \subseteq V$, let $E(X, Y)$ denote the number of edges between $X$ and $Y$. For a vertex set $X$, let $\nu(X)$ denote the sum of the vertex degrees of all vertices in $X$ and let $\overline{X}$ denote the complement of $X$. Using this notation, we have the following theorem

**Theorem 2** ((Chung 1997, Theorem **5.2**)). *Let $G = (V, E)$ be a graph with spectral expansion $\delta$ and let $X, Y \subseteq V$. Then*

$$\left| |E(X, Y)| - \frac{\nu(X) \cdot \nu(Y)}{\nu(V)} \right| \leq \delta \frac{\sqrt{\nu(X)\nu(\overline{X})\nu(Y)\nu(\overline{Y})}}{\nu(V)}.$$

Applying Theorem 2 to expanders, we get the following two corollaries.

**Corollary 3.** *Let $G = (V, E)$ be a $(n, (1 \pm \varepsilon_d)d, \delta)$-expander, and let $X \subseteq V$. Then*

$$|E(X, \overline{X})| \geq (1 - \delta)(1 - 3\varepsilon_d)d\frac{|X| \cdot |\overline{X}|}{n}.$$

**Corollary 4.** *Let $G = (V, E)$ be a $(n, (1 \pm \varepsilon_d)d, \delta)$-expander, and let $X, Y \subseteq V$. If $\varepsilon_d \leq 1/5$, then*

$$\left| |E(X, Y)| - d\frac{|X| \cdot |Y|}{n} \right| \leq 4\varepsilon_d d\frac{|X| \cdot |Y|}{n} + 2\delta d\sqrt{|X| \cdot |Y|}.$$

Due to lack of space, the detailed proofs of Corollaries 3 and 4 are included in the full version of the paper (Friedrich et al. 2022).

As noted above, in contrast to stars, expanders feature many edges between arbitrary subsets of vertices. The key property we require for our results from $(n, (1 \pm \varepsilon_d)d, \delta)$-expanders is that the number of edges between any two sets $X$ and $Y$ of vertices is close to $\frac{d}{n}|X||Y|$ (see Corollaries 3 and 4).

Our results hold for any expander $G'$ that is a subgraph of a graph $G$ that hosts a SIRS process $C$. In order to derive such a result, we define the *projection* $C'$ of $C$ onto $G'$ to be the process on $G'$ such that, at each point in time, each vertex of $G'$ in $C'$ is in the same state as it is in $C$. The survival time of a projected process is the first point in time that the projected process has no infected vertices. Given these definitions, our main result follows.

**Theorem 5.** *Let $G$ be a graph, and let $G'$ be a subgraph of $G$ that is an $(n, (1 \pm \varepsilon_d)d, \delta)$-expander such that $d \to \infty$ and $\delta, \varepsilon_d \to 0$ as $n \to \infty$. Let $C$ be the SIRS process on $G$ with infection rate $\lambda$ and with constant deimmunization rate $\varrho$. Further, let $C$ start with at least one infected vertex in $G'$ and no recovered vertices in $G'$. Last, let $C'$ be the projection of $C$ onto $G'$, and let $T$ be the survival time of $C'$. If $\lambda \geq \frac{c}{d}$ for a constant $c \in \mathbb{R}_{>1}$, then for sufficiently large $n$, it holds that $\mathbb{E}[T] = 2^{\Omega(n)}$.*

We note that Theorem 5 is almost tight with respect to the range of $\lambda$. Ganesh, Massoulié, and Towsley (2005, Theorem 3.1) show that the survival time of the SIS process is at most logarithmic in $n$ when the spectral radius of a graph is less than $1/\lambda$. Note that the spectral radius of a graph is bounded from above by the maximum degree of the graph. This results in a logarithmic expected survival time of the process on $(n, (1 \pm \varepsilon_d)d, \delta)$-expanders when $\lambda \leq \frac{1-\varepsilon}{d}$, for some constant $\varepsilon$. Note that every SIRS process can be coupled to an SIS process on the same graph with the same infection rate such that at each point in time all infected vertices in the SIRS process are also infected in the SIS process. This is achieved by coupling all healing and infection clocks. Therefore, the expected survival time of the SIS process is an upper bound of the expected survival time of the SIRS process. Hence, the expected survival time of the SIRS process for $\lambda \leq \frac{1-\varepsilon}{d}$ is at most logarithmic in $n$ on $(n, (1 \pm \varepsilon_d)d, \delta)$-expanders.

Once again, due to space limitations, the technical details of the proof of Theorem 5 and all related lemmas are in the full version of the paper (Friedrich et al. 2022). What follows is a high-level explanation of our proof.

The proof of Theorem 5 consists of two main parts. First, we prove that a linear number of vertices in $G'$ becomes infected with polynomial probability. Then, we show that the number of infected vertices stays linear for an expected exponential amount of time. For both parts, we make use of potential functions, which map the configuration of the process to a single real number that allows us to quantify how likely the process is to die out. In order to get the result on the projection of the process, we use that the influence of $G \setminus G'$ only increases the rate at which vertices in $G'$ get infected. In the considered configurations, this rate increase only helps the process get into the desired region of the potential.

**First Part: Reaching a Linear Number of Infected Vertices** For the first part, our key lemma shows that the process reaches a configuration with at least $\varepsilon n$ infected vertices with probability at least $\frac{1}{n+2}$. To this end, let $I_{\tau_t}$ be the number of infected vertices after the $t$-th change of the configuration of the process.

**Lemma 6.** *Let $G$ be a graph, and let $G'$ be a subgraph of $G$ that is an $(n, (1 \pm \varepsilon_d)d, \delta)$-expander. Let $d \to \infty$ and $\delta, \varepsilon_d \to 0$ as $n \to \infty$. Let $C$ be a SIRS process on $G$ with infection rate $\lambda$ and with constant deimmunization rate $\varrho$. Further, let $C$ start with at least one infected vertex in $G'$ and no recovered vertices in $G'$. Consider the projection $C'$ of $C$ onto $G'$. If $\lambda \geq \frac{c}{d}$ for a constant $c \in \mathbb{R}_{>1}$, then there exists an $\varepsilon \in \mathbb{R}_{>0}$ such that for sufficiently large $n$, the probability that there exists a $t \in \mathbb{N}$ with $I_{\tau_t} \geq \varepsilon n$ is at least $\frac{1}{n+2}$.*

Note that if this event does not occur, then the infection might die out fast. As the probability of the infection dying out in the first step is roughly $1/2$, the event of Lemma 6 does not have a high enough probability to give us super-polynomial survival time with high probability. To obtain the probabilistic lower bound of Lemma 6, we use a fairly simple potential $H_t$ expressing the difference in the number of infected vertices minus $\varepsilon$ times the recovered vertices. We show that $H_t$ is a submartigale and then apply the optional-stopping theorem to $H_t$ to conclude the proof of Lemma 6.

**Second Part: Retaining a Linear Number of Infected Vertices for Exponential Time** For showing that the infection survives exponentially long once at least $\varepsilon n$ vertices have been infected, we define a more involved potential function $F_t$ than before, which increases when the number of infected vertices reduces. Our definition of $F_t$ is based on a Lyapunov function $f$ used by Korobeinikov and Wake (2002), which they utilize in order to derive results on the global stability of the SIRS process via mean-field theory. We briefly overview this approach before we explain how we adjust it to our setting. To this end, let $S_{\tau_t}$ and $I_{\tau_t}$ denote the number of susceptible and of infected vertices, respectively, of the $t$-th change of the configuration of the process.

Korobeinikov and Wake (2002) assume a fully mixed graph, which roughly corresponds to a clique for our process. In order to show global stability, the authors show a negative drift towards an equilibrium configuration with $I^*$ infected and $S^*$ susceptible vertices. To this end, they use an auxiliary function $f\colon \mathbb{R}^2_{>0} \to \mathbb{R}$ that satisfies for all $x, x^* \in \mathbb{R}_{>0}$ that $f(x^*, x) = x^*\left(\frac{x}{x^*} - \ln \frac{x}{x^*} - 1\right)$. For a fixed $x^*$, the function has a global minimum at $x^*$ and a derivative of $1 - \frac{x^*}{x}$, which is important for calculating the drift. They then define a Lyapunov function $F'(P_{\tau_t}, I_{\tau_t}) = f(P^*, P_{\tau_t}) + f(I^*, I_{\tau_t})$, where $P_{\tau_t} = S_{\tau_t} + \frac{\varrho}{\lambda}$ and $P^* = S^* + \frac{\varrho}{\lambda}$. Note that they use $P_{\tau_t}$ instead of $S_{\tau_t}$ in order for the drift not to be too large when $S_{\tau_t}$ is small. This function results in non-positive drift everywhere, which is enough for the setting of Korobeinikov and Wake (2002).

The potential function of Korobeinikov and Wake (2002) is not sufficient for our purposes, as its resulting drift is 0 for some configurations, whereas we require a constant negative drift in order to derive a rigorous lower bound for the expected survival time. Hence, we adjust the potential function of Korobeinikov and Wake (2002) such that it creates a region in the potential that has a sufficiently large negative drift. We note that we do not need negative drift everywhere but only in configurations with less than $\varepsilon n$ infected vertices. We achieve this by changing the target of susceptible vertices from the equilibrium point to $n$. Further, we use a slightly different shift in our setting to adjust for the base graphs being expanders instead of cliques. Letting $n' = n + P^* - S^*$, we define the potential

$$F_t = F(P_{\tau_t}, I_{\tau_t}) = f(n', P_{\tau_t}) + f(I^*, I_{\tau_t}).$$

By the definition of $f$, the potential $F_t$ has a global minimum for $n' = P_{\tau_t}$ and $I^* = I_{\tau_t}$, which roughly models the idealized (and impossible to reach) configuration of all vertices being susceptible while the number of infected vertices is as in the equilibrium.

For this new potential, we show that there is a region in which higher infection rates decrease the drift and, for a sufficiently high infection rate, the process is a strict super-martingale with a constant negative drift. This is formally stated in the following two lemmas. For a time $t$, $D_t$ refers to the expected change of the potential in the next step, so $D_t = \mathbb{E}[F_{t+1} - F_t]$. The rates $r_{ir,t}$ and $r_{rs,t}$ denote the rate at which vertices recover and lose their immunity respectively.

**Lemma 7.** *Let $G$ be a graph, and let $G'$ be a subgraph of $G$ that is an $(n, (1 \pm \varepsilon_d)d, \delta)$-expander. Let $C$ be a SIRS process on $G$ with infection rate $\lambda$ and with constant deimmunization rate $\varrho$. Consider the projection $C'$ of $C$ onto $G'$. Let $E(I, S)$ be the amount of edges between the infected and the susceptible vertices at time $t$, and let $r'_t = \frac{c}{d}E(I, S) + r_{ir,t} + r_{rs,t}$. If $\lambda \geq \frac{c}{d}$ for a constant $c \in \mathbb{R}_{>1}$, then there exists a constant $\varepsilon \in \mathbb{R}_{>0}$ such that, for all $t \in \mathbb{N}$ and sufficiently large $n$, if $2 \leq I_{\tau_t} \leq \varepsilon n$, then*

$$\begin{aligned} r'_t \cdot D_t \leq \frac{c}{d}E(I, S) \cdot (F(P_{\tau_t} - 1, I_{\tau_t} + 1) - F(P_{\tau_t}, I_{\tau_t})) \\ + r_{ir,t} \cdot (F(P_{\tau_t}, I_{\tau_t} - 1) - F(P_{\tau_t}, I_{\tau_t})) \\ + r_{rs,t} \cdot (F(P_{\tau_t} + 1, I_{\tau_t}) - F(P_{\tau_t}, I_{\tau_t})). \end{aligned}$$

**Lemma 8.** *Let $G$ be a graph, and let $G'$ be a subgraph of $G$ that is an $(n, (1 \pm \varepsilon_d)d, \delta)$-expander. Let $d \to \infty$ and*

*$\delta, \varepsilon_d \to 0$ as $n \to \infty$. Let $C$ be a SIRS process on $G$ with infection rate $\lambda$ and with constant deimmunization rate $\varrho$. Consider the projection $C'$ of $C$ onto $G'$. Let $t \in \mathbb{N}$ and $\varepsilon_0, \varepsilon \in (0,1)$ be sufficiently small constants. Assume that $\varepsilon_0 n \geq I_{\tau_t} \geq \varepsilon n$. If $\lambda \geq \frac{c}{d}$ for a constant $c \in \mathbb{R}_{>1}$, then there exists a constant $a \in \mathbb{R}_{>0}$ such that $D_t \leq -a$ for sufficiently large $n$.*

We use the expansion properties of the base graph that guarantee that the infected vertices always have enough susceptible neighbors such that new vertices get infected and the potential decreases in expectation. This allows us to apply a concentration bound by Oliveto and Witt (2011) for strict supermartingales, known as *negative-drift theorem*. The negative-drift theorem results in the lower exponential bound of the expected survival time.

## 5 Random Graphs and Complex Networks

The generality of Theorem 5 makes it applicable to a broad range of graph classes, as the only requirement is for the base graph to contain a large expander as a subgraph. We illustrate this by considering the SIRS process on Erdős–Rényi graphs as well as models of real-wold networks.

### Erdős–Rényi Graphs

The first random-graph model we are interested in is $G_{n,p}$ — the classical random-graph model of Erdős and Rényi (1959). The expansion properties of this model have been previously studied in literature.

**Theorem 9** ((Coja-Oghlan 2007, Theorem **1.2**)). *Let $G \sim G_{n,p}$ be an Erdős–Rényi graph with $(n-1)p \geq c_1 \ln(n)$ for a sufficiently large constant $c_1 \in \mathbb{R}_{>0}$. Then asymptotically almost surely, for the spectral expansion $\delta$ of the Laplacian of $G$ holds $\delta \in \mathrm{O}\big((p(n-1))^{-1/2}\big)$.*

By Chernoff bounds, it holds that the vertex degrees in Erdős–Rényi graphs are tightly distributed around the average degree $d$ if $d \in \omega(\ln n)$. Therefore, Erdős–Rényi graphs satisfy with high probability our definition of an $(n, (1 \pm \varepsilon_d)d, \delta)$-expander. Combining this with Theorem 5, we obtain the following threshold behavior.

**Corollary 10.** *Let $G \sim G_{n,p}$ be an Erdős–Rényi graph with $(n-1)p \in \omega(\ln n)$. Consider the SIRS process $C$ on $G$ with constant deimmunization rate $\varrho$, and let $T$ be the survival time of $C$ when the process starts with at least one infected vertex. If $\lambda \geq \frac{c}{d}$ for a constant $c \in \mathbb{R}_{>1}$, then $\mathbb{E}[T] = 2^{\Omega(n)}$ asymptotically almost surely with respect to $G$. If $\lambda \leq \frac{c}{d}$ for a constant $c \in (0,1)$, then $\mathbb{E}[T] \in \mathrm{O}(\log n)$ asymptotically almost surely with respect to $G$.*

Comparing Corollary 10 with the respective result for the SIS process (Ganesh, Massoulié, and Towsley 2005, Theorem 5.5) shows that the two processes, SIS and SIRS, behave similarly on Erdős–Rényi graphs.

### Complex Networks

A variety of random-graph models that exhibit properties found in real-world networks has appeared in network science (Boguna et al. 2021). Such network models provide a highly relevant structure for studying the survival time of the SIRS process. We focus our attention on three such complex network models that exhibit key properties required for applying Theorem 5. These models are Chung–Lu graphs (Aiello, Chung, and Lu 2001), hyperbolic random graphs (Krioukov et al. 2010), and geometric inhomogeneous random graphs (Keusch 2018).

A common characteristic shared by these three network models is that the degrees of the vertices follow a power-law distribution. The exponent of the power-law is controlled by a parameter $\gamma$. The interesting parameter range is $\gamma \in (2,3)$, as beyond this range, these models lose key properties present in real-world networks. When $\gamma \in (2,3)$, two key properties hold on all three models. The first one is that their diameter is at most poly-logarithmic with respect to the total number of vertices $n$.

**Theorem 11** ((Friedrich and Krohmer 2018, Theorem **1**)). *Let $G$ be a hyperbolic random graph with $n$ vertices that follows a power-law degree distribution with exponent $\gamma \in (2,3)$. Then the diameter of the giant component of $G$ is $\mathrm{O}\big((\log n)^{2/(3-\gamma)}\big)$ with probability $1 - \mathrm{O}\big(n^{-3/2}\big)$.*

The second property is that they contain a clique with polynomial size.

**Theorem 12** ((Friedrich and Krohmer 2015)). *Let $G$ be a hyperbolic random graph with $n$ vertices that follows a power-law degree distribution with exponent $\gamma \in (2,3)$. Then the size of the largest clique of $G$ is in $\Theta\big(n^{(3-\gamma)/2}\big)$ with high probability.*

Note that similar statements hold for Chung–Lu graphs (Chung and Lu 2003) and geometric inhomogeneous random graphs (Keusch 2018). We proceed with illustrating how Theorem 5 can be used to show a superpolynomial survival time on hyperbolic random graphs.

We first use the poly-logarithmic diameter to show that the infection reaches the largest clique with a sufficient probability when the process starts with at least one infected vertex.

**Lemma 13.** *Let $G$ be a hyperbolic random graph with $n$ vertices that follows a power-law degree distribution with exponent $\gamma \in (2,3)$, and let $C$ be an SIRS process on $G$ with infection rate $\lambda$ and with constant deimmunization rate $\varrho$. Further, let $C$ start with at least one infected vertex in the giant component and no recovered vertices in the giant component. If $\lambda \geq cn^{(\gamma-3)/2}$ for a constant $c \in \mathbb{R}_{>0}$, then the probability that the infection reaches a configuration in which a vertex in the largest clique is infected is at least $\exp\big(-(\ln n)^{3/(3-\gamma)}\big)$ for sufficiently large $n$.*

*Proof.* Let $v$ be a vertex that starts infected, and let $d$ be the shortest distance from $v$ to any vertex of the largest clique. Note that $d$ is bounded from above by the diameter of the giant component. Therefore, by Theorem 11, there exists a constant $a \in \mathbb{R}_{>0}$ such that for sufficiently large $n$ with a probability of at least $\frac{1}{2}$, it holds that $d \leq a(\ln n)^{2/(3-\gamma)}$.

For all $i \in \mathbb{N}$, let $E_i$ be the event that $C$ reaches a configuration with an infected vertex that has a distance of $i$ to the largest clique. Consider for all $i \in \mathbb{N}_{<d}$ the probability $\Pr[E_i \mid E_{i+1}]$. Each vertex with a distance of $i+1$ to the largest clique has a neighbor that has a distance of $i$

to the clique. With a probability of $\frac{\lambda}{1+\lambda}$, an infected vertex infects a specific neighbor before recovering. Therefore, $\Pr[E_i \mid E_{i+1}] \geq \frac{\lambda}{1+\lambda} \geq \frac{c}{2} n^{(\gamma-3)/(2)}$ for sufficiently large $n$.

With a probability of at least $\frac{1}{2}$, it holds that $d \leq a(\ln n)^{2/(3-\gamma)}$. This yields for sufficiently large $n$ that

$$\begin{aligned}\Pr[E_0] &= \prod_{i=0}^{d-1} \Pr[E_i \mid E_{i+1}] \geq \prod_{i=0}^{d-1} \frac{c}{2} n^{\frac{\gamma-3}{2}} \\ &= \left(\frac{c}{2} n^{\frac{\gamma-3}{2}}\right)^d \geq \left(\frac{c}{2}\right)^d \left(n^{\frac{\gamma-3}{2}}\right)^{a(\ln n)^{\frac{2}{3-\gamma}}} \\ &= \mathrm{e}^{\frac{\gamma-3}{2} a (\ln n)^{\frac{5-\gamma}{3-\gamma}} + d \ln(c/2)} \geq \mathrm{e}^{-(\ln n)^{\frac{3}{3-\gamma}}}. \qquad \square\end{aligned}$$

When the infection reaches the largest clique of a hyperbolic random graph, Theorem 5 yields an exponential expected survival time for a sufficiently large infection rate.

**Corollary 14.** *Let $G$ be a hyperbolic random graph with $n$ vertices that follows a power-law degree distribution with exponent $\gamma \in (2,3)$, and let $C$ be the SIRS process on $G$ with infection rate $\lambda$ and with constant deimmunization rate $\varrho$. Further, let $C$ start with at least one infected vertex in the giant component and no recovered vertices, and let $T$ be the survival time of $C$. Then there exists a constant $c \in \mathbb{R}_{>0}$ such that if $\lambda \geq c n^{(\gamma-3)/2}$, then $\mathbb{E}[T] = 2^{\Omega\left(n^{(3-\gamma)/2}\right)}$.*

*Proof.* Let $k$ be the size of the largest clique of $G$. By Theorem 12, there exists a constant $a \in \mathbb{R}_{>0}$ such that with high probability it holds that $k \geq a n^{(3-\gamma)/2}$. Let $c = a^{-1} + 1$ such that with high probability it holds that $\lambda \geq \frac{1+a}{k}$. Let $E$ be the event that there exists a configuration in which a vertex in the largest clique of $G$ is infected. By Lemma 13, it holds that $\Pr[E] \geq \exp\left(-(\ln n)^{3/(3-\gamma)}\right)$ for sufficiently large $n$. Note that a clique with $k$ vertices is an $(k, (1 \pm k^{-1})k, (k-1)^{-1})$-expander. Hence, by Theorem 5, it holds that $\mathbb{E}[T \mid E] = 2^{\Omega(k)}$, as the infection survives that long on the clique alone after its first vertex gets infected.

By the law of total expectation and that with high probability $k \geq a n^{(3-\gamma)/2}$, we conclude

$$\begin{aligned}\mathbb{E}[T] &\geq \Pr[E] \cdot \mathbb{E}[T \mid E] \\ &\geq \mathrm{e}^{-(\ln n)^{\frac{3}{3-\gamma}}} \cdot 2^{\Omega\left(n^{(3-\gamma)/2}\right)} \\ &= 2^{\Omega\left(n^{(3-\gamma)/2}\right)}. \qquad \square\end{aligned}$$

Following this line of argumentation, similar statements can be proven for Chung–Lu graphs and geometric inhomogeneous random graphs for an appropriate choice of the respective parameters of these models.

## 6 Conclusions and Future Work

To the best of our knowledge, we provide the first mathematically rigorous analysis of the expected survival time of the SIRS process. Our results hold for a substantial amount of graph classes that have been considered in the last decades of research for similar processes, most notably the SIS process. Our main contribution shows for graphs with expander subgraphs an exponential survival time threshold, which covers a great amount of established graph classes, such as scale-free graphs, Erdős–Rényi graphs, and cliques. Our resulting threshold is almost tight for Erdős–Rényi graphs and gives a threshold for many social network models, like hyperbolic random graphs or geometric inhomogeneous random graphs. We complement our findings by showing that, for star graphs, the expected survival time of the SIRS process can never become super-polynomial, indicating that single vertices cannot be influential enough to lead to long survival on their own. This marks a significant difference compared to the previously studied SIS process.

Overall, our results provide deep insights into how the network structure impacts the expected survival time of the SIRS process. In contrast to the SIS model, it is not a single star that is important but good expansion instead. On stars, the SIRS process dies out quickly because once the center of the star (which is its only vertex with a high degree) is recovered and thus temporarily immune to re-infection, the infection has no way of spreading. During this time, many vertices can recover, thus drastically reducing the number of infected vertices. On graphs with sufficient expansion, this situation is entirely different. Due to the expansion, each vertex is sufficiently well connected in the network. Thus, even if a fair amount of its neighbors is recovered, there remain enough connections to other vertices that are either already infected or can easily be infected. This leads to an exponentially long survival of the SIRS process.

Although our results cover already a great range of interesting and important graph classes, this article is just the first step to understanding the SIRS process more thoroughly. Our analyses pose exciting new challenges for different scenarios, which we briefly delineate in the following.

Combined, our results for stars (Theorem 1) and expanders (Theorem 5) show that adding edges to a graph leads, eventually, from a polynomial expected survival time to an exponential one. However, it is not clear so far when this transition happens. An interesting next step is to look into connected stars instead of single stars. Connected stars appear as subgraphs in important real-world network models, most prominently, the preferential-attachment model (Barabasi and Albert 1999), but also in Chung–Lu graphs (Chung and Lu 2003), for which our initial results could be improved, motivating this research question.

A different extension of our results is to consider deimmunization rates that are dependent on the graph size. Comparing the behavior of the SIS and the SIRS process on stars suggests that an increased deimmunization rate leads to far longer expected survival times. Thus, an interesting question is whether the survival time exhibits a threshold behavior with respect to the deimmunization rate.

While the re-infection makes the topology much more important than the starting configuration, finding the best set of initially infected vertices to achieve the longest survival time is still an interesting question. Analyzing this question in the SIS or SIRS model could yield some new insights into influence maximization.

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