

Chapter 5

Controlling negative diffusion in the presence of risk behavior changes

In Chapter 4, we analyzed intervention strategies assuming the behavior of each individual remains the same before and after taking interventions, which is not an accurate assumption in some real world scenarios. Previous studies have shown imperfect interventions and risk behavior changes can lead to perverse outcomes. Thus, in this chapter, we study how to control negative diffusion with the presence of risk behavior changes.

From the results in Chapter 4, we can see that Nash equilibrium may not exist even without risk behavior changes. Using game theory in the presence of risk behavior changes is going to be extremely difficult. In this chapter, we formulate a network-based model and use random graph techniques to understand how risk behavior change in conjunction with failure of prophylactic interventions can lead to perverse outcomes where “less (intervention) is more (effective)”. Our model captures the distinction between one- and two-sided risk behavior change. In one-sided situations (e.g. influenza/H1N1) it is sufficient for either individual in an interaction to exhibit risk behavior change whereas in two-sided situations (e.g. AIDS/HIV) it is necessary for both individuals in the interaction to exhibit risk behavior change, for a potential transmission of the disease. A central discovery is that the phenomenon of perversity occurs at differing levels of intervention coverage depending upon the “sidedness” of the interaction. Fur-

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thermore, again dependent on the “sidedness,” targeting highly connected nodes can be strictly worse than uniformly random interventions at the same level of coverage.

In Section 5.1, we formally define our model. In Section 5.2, we explain our first finding where less intervention can be more effective. In Section 5.3, we explain our second finding where targeted intervention strategy can be worse than random intervention strategy. Section 5.4 backs up our findings with comprehensive simulations. And we conclude this Chapter in Section 5.5.

5.1 Models

We obtain our results through both analytical techniques and simulations on a range of networks including preferential attachment networks [28] and large synthetic and real-world networks. For our analyses, we adopt the SIR model of epidemics defined on networks. Let $G = (V, E)$ denote an undirected social contact graph, where V denotes a set of people (referred to as nodes henceforth) and $(u, v) \in E$ denotes a contact between nodes u and v (see Figure 5.1(a) for an example). If node u becomes infectious, it will infect each of its susceptible neighbors independently with probability p (referred as *base transmissivity*). Each node in the graph is either vaccinated (e.g., nodes B or F in Figure 5.1(b)) or not (e.g., nodes A or C in Figure 5.1(b)). If a node u is not vaccinated, we label it as UV. The vaccine fails with probability p_f . If a node u 's vaccine fails, we label it as VF; otherwise, we label it VS. Both UV and VF nodes are susceptible. We assume that vaccine failure is a stochastic event and that (vaccinated) nodes do not know if (their own) vaccination succeeded or not. If a node with vaccine failure is infected then its risk behavior changes, i.e., it increases its contacts to some of its' neighbors, resulting in *boosted transmissivity* p_m - in the one-sided model a node infects all its susceptible neighbors with boosted transmissivity p_m , while in the two-sided model, it only infects those neighbors with boosted transmissivity p_m that have also had a failed vaccination. In the rest of the paper, we use p_v to denote the probability that a node is vaccinated, under a campaign of uniformly random vaccination.

The disease transmission process is thus defined by the tuple (p, p_m, p_f, p_v) in the following manner: every node is labeled with UV, VS, VF with probability $1 - p_v$, $p_v(1 - p_f)$, and $p_v p_f$, respectively. All nodes labeled VS are removed from the graph. Each edge (u, v) connecting two surviving nodes u and v , is “open” (or retained in the

graph, in the language of percolation, which corresponds to disease transmission on this edge), or “closed” (or removed from the graph), with some probability depending on the model - (i) in the one-sided model, edge (u, v) is open with probability p , if both u and v are labeled UV, and is open with probability p_m if one of u and v is labeled VF; (ii) in the two-sided model, edge (u, v) is open with probability p , unless both u and v are labeled VF. Following the well known correspondence between bond percolation and disease transmission, the connected component containing a specific node u is the (random) subset of nodes infected, if the disease starts at u . If the components resulting from one random instance of the above stochastic process are C_1, C_2, \dots, C_k , then $\sum_i |C_i|^2 / n$ denotes the expected outbreak size of the disease starting from a random initial node. In our analysis, we use this as a measure of *epidemic severity*.

5.2 Perversity and sidedness

We first report on our finding that both one-sided and two-sided behavior changes can lead to perverse outcomes (less vaccination is more effective) across a wide range of contact networks. One-sided behavior change leads to perverse outcomes at low levels of intervention, in which the epidemic severity increases with p_v , up to a point, as shown in Figure 5.2, 5.3, and 5.4. Two-sided behavior change leads to perverse outcomes at high levels of intervention, in which the epidemic severity starts increasing beyond a threshold value of p_v . We mathematically establish the phenomena of perversity and non-monotonicity for graphs generated according to the Erdős-Renyi model [108], denoted by $G(n, p)$, in which each edge between a pair of nodes is chosen independently with probability p . We prove rigorously that there exist p , p_m , and p_f , such that (i) in the one-sided model, it almost surely holds that the epidemic severity is $o(n)$ for both $p_v = 0$ and $p_v = 1$, yet $\Theta(n)$ for some p_v in $(0, 1)$; (ii) in the two-sided model, the epidemic severity is $\Theta(n)$ for both $p_v = 0$ and $p_v = 1$, yet $o(n)$ for some p_v in $(0, 1)$. This implies that there is a choice of parameters (which turns out to be quite broad), such that as the vaccinated fraction p_v is varied, the epidemic severity shows a non-monotone behavior.

Theorem 39. *For the Erdős-Rényi random graph model $G(n, p)$, there exist p , p_m , and p_f , such that (i) in the one-sided model, it almost surely holds that the epidemic severity is $o(n)$ for both $p_v = 0$ and $p_v = 1$, yet $\Theta(n)$ for some p_v in $(0, 1)$; (ii) in the*

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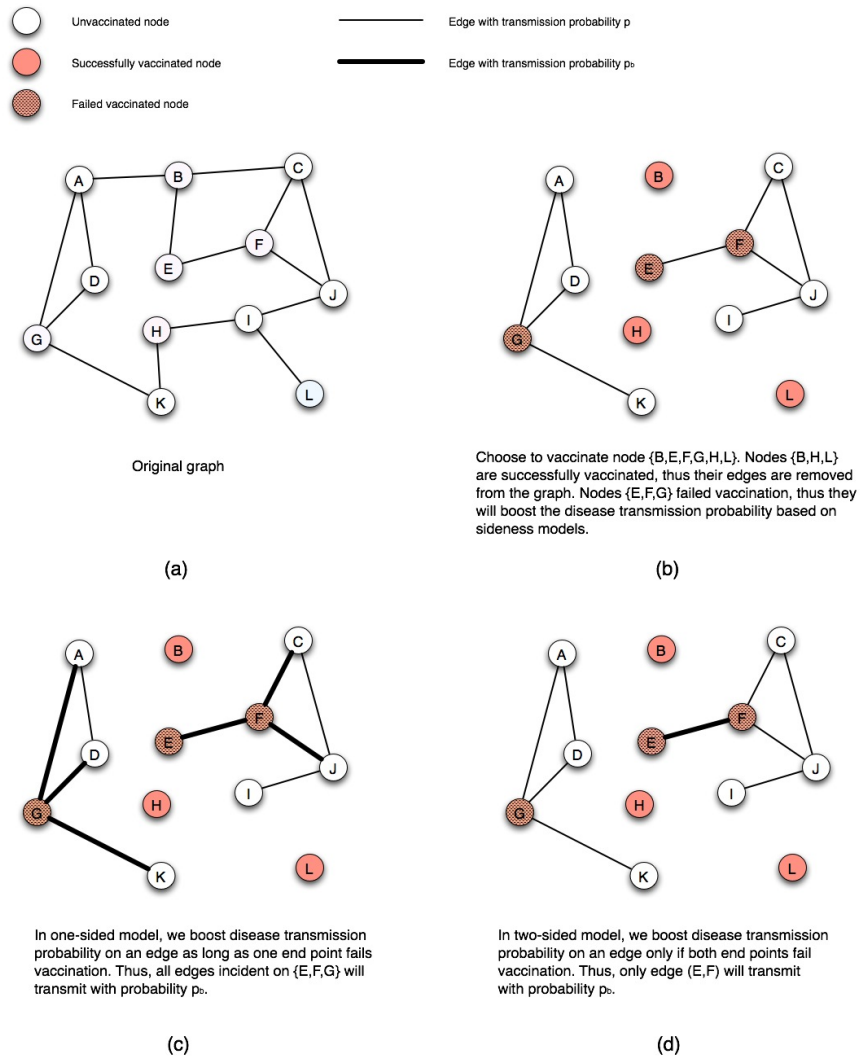


Figure 5.1: Sidedness of risk behavior change: the one-sided and two-sided models.

5.2 Perversity and sidedness

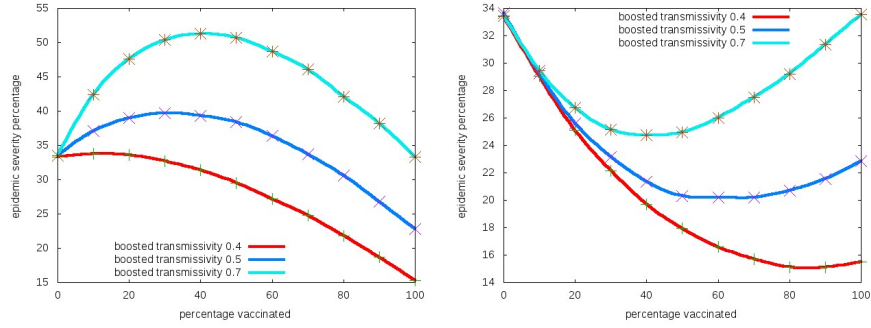


Figure 5.2: Epidemic severity with different boosted transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.25$ and $p_s = 0.35$.

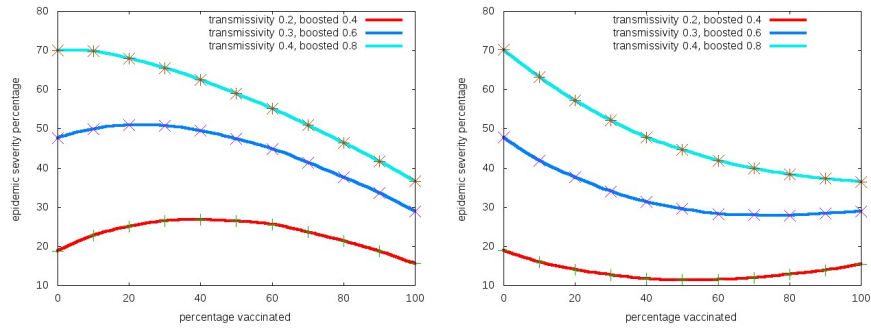


Figure 5.3: Epidemic severity with different transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p_s = 0.35$, and $p_m = 2p$.

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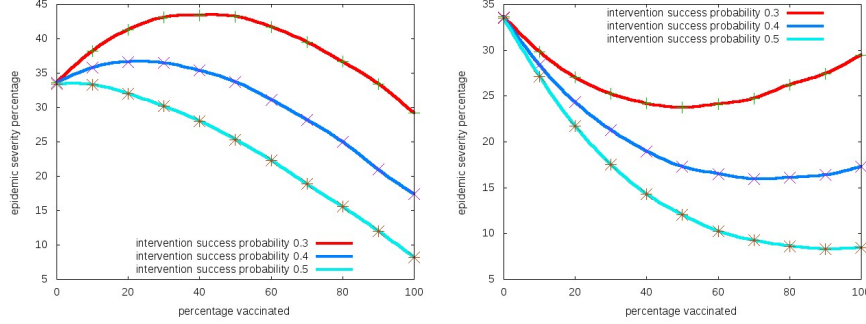


Figure 5.4: Epidemic severity with different intervention success probabilities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.25$, and $p_m = 0.5$.

two-sided model, the epidemic severity is $\Theta(n)$ for both $p_v = 0$ and $p_v = 1$, yet $o(n)$ for some p_v in $(0, 1)$.

We give a brief sketch of our proof, which is based on recent results of Söderberg [120] and Bollobás et al [39] on heterogeneous random graphs. We refer the reader to supplementary information for details. Consider the model of heterogeneous random graphs denoted by $\mathcal{G}(N, K, \mathbf{r}, \mathbf{c})$, where (i) K is a positive integer, (ii) $\mathbf{r} = \{r_1, \dots, r_K\}$ is a probability vector, (iii) $\mathbf{c} = (c_{ij})$ is a $K \times K$ matrix, (iv) each node $j = 1, \dots, N$, is assigned a type $i \in \{1, \dots, K\}$ with probability r_i , and (v) each pair of nodes i, j are connected by an edge with probability $p(i, j) = c_{ij}/N$. Söderberg [120] and Bollobás et al. [39] established the following: (i) if the eigenvalues of the matrix $\{c_{ij}r_j\}$ are all less than 1, it is sub-critical (i.e., has no giant component), and (ii) if some eigenvalue is larger than 1, it is super-critical (i.e., has a giant component) with asymptotically $r_i(1 - f_i)N$ nodes of type i , where f_i satisfies the coupled set of equations: $f_i = \exp\left(\sum_j c_{ij}r_j(f_j - 1)\right)$. We show that if the contact network is generated by the Erdos-Renyi model $G(n, c/n)$, then the disease transmission process produces a heterogeneous random graph with the eigenvalue characteristic equation given by

$$-\lambda(\lambda^2 - (c(1 - p_v) + p_m c p_v p_f)\lambda + c^2(1 - p_v)p_m p_v p_f - c^2(1 - p_v)p_v p_f) = 0.$$

We show the existence of parameters p_v , p_f , p_m , and c such that the absolute value of every eigenvalue is smaller than 1.

We find the phenomenon of perversity exists in a broad class of graphs, and in order to formally prove its widespread occurrence, we consider *locally finite graphs*, which have been widely studied in percolation theory (e.g., see [38]). Locally finite graphs include infinite graphs in which each node has bounded degree. Using techniques from percolation theory, we prove that in every locally finite graph G , there exist p , p_m , and p_f , such that: (i) the epidemic severity is finite for both $p_v = 0$ and $p_v = 1$, yet infinite for some p_v in $(0, 1)$ in the one-sided model; (ii) the epidemic severity is infinite for both $p_v = 0$ and $p_v = 1$, yet finite for some p_v in $(0, 1)$ in the two-sided model. This result provides strong evidence of the universality of the phenomenon. As such it begs for a natural and intuitive explanation. Our best structural understanding at this point is that this is the consequence of two competing tensions – vaccine success that serves to contain the spread and risky behavior that, exacerbated by vaccine failure, serves to boost the contagion. In the one-sided situation since it is sufficient for infection spread to have just the one party in an interaction exhibiting risky behavior we see perversity manifesting itself at low levels of vaccination. Whereas, in the two-sided situation since it is necessary for both the interacting parties to exhibit risky behavior we see perversity manifesting itself only at high vaccination levels which is a prerequisite for a non-trivial fraction of parties with failed vaccines to exist.

Theorem 40. *For every locally-finite infinite graph G , there exist p , p_m , and p_f , such that: (i) the epidemic severity is finite for both $p_v = 0$ and $p_v = 1$, yet infinite for some p_v in $(0, 1)$ in the one-sided model; (ii) the epidemic severity is infinite for both $p_v = 0$ and $p_v = 1$, yet finite for some p_v in $(0, 1)$ in the two-sided model.*

The phenomenon of non-monotonicity and its dependence on sidedness that we have identified occurs across a wide range of network models.

5.2.1 Proof of Theorem 39

In this section, we give formal proofs of perversity and non-monotonicity in Erdős-Rényi random graphs. We have observed the non-monotonicity is pervasive in wide range of contact graphs, including scale-free graphs, Erdős-Rényi graphs, and other synthetic or real world graphs. Theorem 39 gives the rigorous proof of one-sided model and two-sided model for Erdős-Rényi random graphs.

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Lemma 41. *Given a complete graph as the contact network, for intervention with any success probability p_s , there exists parameter set p, p_m, p_v , such that there is non-monotonicity in two-sided risk behavior model.*

Proof. When nobody takes interventions, there are n nodes in the graph, and the disease transmission probability between each pair of nodes is $p = c/n$, where $c > 1$. By [60], there is a giant connected component with high probability (size of the connected component is $\Theta(n)$).

When everybody takes interventions, $p_s n + o(n)$ nodes will have successful interventions with high probability, and thus removed from the graph. The remaining $(1 - p_s)n + o(n)$ nodes will all exhibit risk behavior changes. Thus, the disease transmission probability between each pair of nodes is $p_m = c'/(1 - p_s)n$, where $c' > 1$. By [60], there is a giant connected component with high probability (size of the connected component is $\Theta(n)$).

Now we are going to show there exists a p_v , such that, if we apply interventions to each node independently with probability p_v , the epidemic severity will be $o(n)$ with high probability. Let A be the set of nodes that haven't taken interventions, B be the set of nodes that have taken interventions but failed, and C be the set of nodes that have taken interventions and succeeded. $r_A = 1 - p_v$ represents the probability of a random node being in set A , $r_B = p_v(1 - p_s)$ represents the probability of a random node being in set B , and $r_C = p_v p_s$ represents the probability of a random node being in set C . In the two-sided model, disease transmits with probability p_m between nodes in set C , and p otherwise. Set $a = c$ and $b = c'/(1 - p_s)$. Let

$$M = \begin{pmatrix} ar_A & ar_B & 0 \\ ar_A & br_B & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

This yields a model of inhomogeneous random graphs with 3 types of vertices (A , B , and C). By [120] Theorem 1, if all the eigenvalues of M are less than 1 in absolute value, then the size of the largest connected component is $o(n)$. Let λ be the eigenvalues of M .

$$\begin{aligned} \det(M - \lambda I) &= \det \begin{bmatrix} ar_A - \lambda & ar_B & 0 \\ ar_A & br_B - \lambda & 0 \\ 0 & 0 & -\lambda \end{bmatrix} \\ &= -\lambda((ar_A - \lambda)(br_B - \lambda) - a^2 r_A r_B) \\ &= -\lambda(\lambda^2 - (ar_A + br_B)\lambda + abr_A r_B - a^2 r_A r_B) \end{aligned}$$

Solving $\det(M - \lambda) = 0$, we have

$$\lambda_1 = \frac{(ar_A + br_B) + \sqrt{\Delta}}{2}, \lambda_2 = \frac{(ar_A + br_B) - \sqrt{\Delta}}{2}, \lambda_3 = 0$$

where $\Delta = (ar_A - br_B)^2 + 4a^2r_Ar_B$. Since $|\lambda_3| \leq |\lambda_2| \leq |\lambda_1|$, it is sufficient to show there exists a set of parameters that yields $|\lambda_1| < 1$. Set $c' = c$.

$$\begin{aligned} |\lambda_1| &= \frac{(ar_A + br_B) + \sqrt{(ar_A - br_B)^2 + 4a^2r_Ar_B}}{2} \\ &= \frac{c(1 - p_v) + c'p_v + \sqrt{(c(1 - p_v) - c'p_v)^2 + 4c^2p_v(1 - p_v)(1 - p_s)}}{2} \\ &= c \frac{(1 - p_v) + p_v + \sqrt{((1 - p_v) - p_v)^2 + 4p_v(1 - p_v)(1 - p_s)}}{2} \\ &= c \frac{1 + \sqrt{((1 - p_v) + p_v)^2 - 4p_s p_v(1 - p_v)}}{2} \\ &= c \frac{1 + \sqrt{1 - 4p_s p_v(1 - p_v)}}{2} \end{aligned}$$

When $0 < p_v < 1$, $\frac{1 + \sqrt{1 - 4p_s p_v(1 - p_v)}}{2}$ is a constant smaller than 1. We can find $c > 1$ that satisfies $c \frac{1 + \sqrt{1 - 4p_s p_v(1 - p_v)}}{2} < 1$. Thus, for intervention with success probability p_s , there exist parameters p_v , p , and p_m , such that the epidemic size is $o(n)$. This completes our proof of this lemma. \square

Lemma 42. *Given a complete graph as the contact network, for intervention with any success probability p_s , there exists parameter set p, p_m, p_v , such that there is non-monotonicity in one-sided risk behavior model.*

Proof. When nobody takes interventions, there are n nodes in the graph, and the disease transmission probability between each pair of nodes is $p = c/n$, where $c < 1$. By [60], the size of the largest connected component is $O(\log n)$ with high probability.

When everybody takes interventions, $p_s n + o(n)$ nodes will have successful interventions with high probability, and thus removed from the graph. The remaining $(1 - p_s)n + o(n)$ nodes will exhibit risk behavior changes. Thus, the disease transmission probability between each pair of nodes is $p_m = c' / (1 - p_s)n$, where $c' < 1$. By [60], the size of the largest connected component is $O(\log n)$ with high probability.

Now we are going to show there exists a p_v , such that, if we apply interventions to each node independently with probability p_v , the epidemic severity will be $\Theta(n)$ with high probability. Let A be the set of nodes that haven't taken interventions, B be the

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set of nodes that have taken interventions but failed, and C be the set of nodes that have taken interventions and succeeded. $r_A = 1 - p_v$ represents the probability of a random node being in set A , $r_B = p_v(1 - p_s)$ represents the probability of a random node being in set B , and $r_C = p_v p_s$ represents the probability of a random node being in set C . In the one-sided model, disease transmit with probability p between nodes in set A , and p_m otherwise. Set $a = c$ and $b = c'/(1 - p_s)$. Let

$$M = \begin{pmatrix} ar_A & br_B & 0 \\ br_A & br_B & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

This yields a model of inhomogeneous random graphs with 3 types of vertices (A , B , and C). By [120] Theorem 1, if some eigenvalue of M is larger than 1, then the size of the largest connected component is $\Theta(n)$. Let λ be the eigenvalues of M .

$$\begin{aligned} \det(M - \lambda I) &= \det \begin{bmatrix} ar_A - \lambda & br_B & 0 \\ br_A & br_B - \lambda & 0 \\ 0 & 0 & -\lambda \end{bmatrix} \\ &= -\lambda((ar_A - \lambda)(br_B - \lambda) - b^2 r_A r_B) \\ &= -\lambda(\lambda^2 - (ar_A + br_B)\lambda + abr_{ArB} - b^2 r_A r_B) \end{aligned}$$

Solving $\det(M - \lambda) = 0$, we have

$$\lambda_1 = \frac{(ar_A + br_B) + \sqrt{\Delta}}{2}, \lambda_2 = \frac{(ar_A + br_B) - \sqrt{\Delta}}{2}, \lambda_3 = 0$$

where $\Delta = (ar_A - br_B)^2 + 4b^2 r_A r_B$. Since $|\lambda_1| \geq |\lambda_2| \geq |\lambda_3|$, it is sufficient to show

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there exists a set of parameters that yields $|\lambda_1| > 1$. Let $c = c'$.

$$\begin{aligned}
 |\lambda_1| &= \frac{(ar_A + br_B) + \sqrt{(ar_A - br_B)^2 + 4b^2r_Ar_B}}{2} \\
 &= \frac{c(1 - p_v) + c'p_v + \sqrt{(c(1 - p_v) - c'p_v)^2 + 4c'^2\frac{p_v(1-p_v)}{1-p_s}}}{2} \\
 &= c \frac{(1 - p_v) + p_v + \sqrt{((1 - p_v) - p_v)^2 + 4\frac{p_v(1-p_v)}{1-p_s}}}{2} \\
 &= c \frac{1 + \sqrt{((1 - p_v) - p_v)^2 + 4p_v(1 - p_v) - 4p_v(1 - p_v) + 4\frac{p_v(1-p_v)}{1-p_s}}}{2} \\
 &= c \frac{1 + \sqrt{((1 - p_v) + p_v)^2 + 4p_v(1 - p_v) \left(\frac{1}{1-p_s} - 1\right)}}{2} \\
 &= c \frac{1 + \sqrt{1 + 4p_v(1 - p_v) \frac{p_s}{1-p_s}}}{2}
 \end{aligned}$$

When $0 < p_v < 1$, $\frac{1 + \sqrt{1 + 4p_v(1-p_v) \frac{p_s}{1-p_s}}}{2}$ is a constant greater than 1. We can find $c < 1$ that satisfies $c \frac{1 + \sqrt{1 + 4p_v(1-p_v) \frac{p_s}{1-p_s}}}{2} > 1$. Thus, for vaccination with success probability p_s , there exist parameters p_v , p , and p_m , such that the epidemic size is $\Theta(n)$. This completes our proof of this lemma. \square

Now we can show the proof of Theorem 39 as follows.

Proof of Theorem 39. We claim the disease transmission process on Erdős-Rényi random graph $G(n, p^*)$ with parameter set (p, p_m, p_s, p_v) is the same as the disease transmission process on a complete graph with parameter set (p^*p, p^*p_m, p_s, p_v) . It's simply because the edge between each pair of nodes ‘‘opens’’ with the same probability in both random processes. Thus, for any disease transmission process on Erdős-Rényi random graph, we can reduce it to the corresponding process on a complete graph. Then by Lemma 41 and 42 we can conclude the statement of this theorem holds. \square

5.3 Randomized vs. targeted vaccinations

We next report on our finding that targeted vaccination can be strictly worse than random vaccination for some level of vaccine coverage, and this phenomenon occurs both for one-sided as well as two-sided behavior change (as shown in Figure 5.5).

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In the literature it has been observed that targeting highly connected individuals for vaccination lead to better outcomes as opposed to random coverage [127, 55, 35]. Our finding adds nuance to the existing results when risky behavior is taken into account. This counterintuitive phenomenon can also be explained by the tug of war between successful vaccination and risky behavior. If the effect of risky behavior is dominant then one would expect that targeted vaccination ends up being worse than random coverage since it is the targeted high-degree individuals that are the most responsible for creating additional contagion. And, in fact the evidence supports this explanation in that we see targeted coverage being inferior to random coverage at low levels of vaccination in the one-sided case but at high levels in the two-sided case.

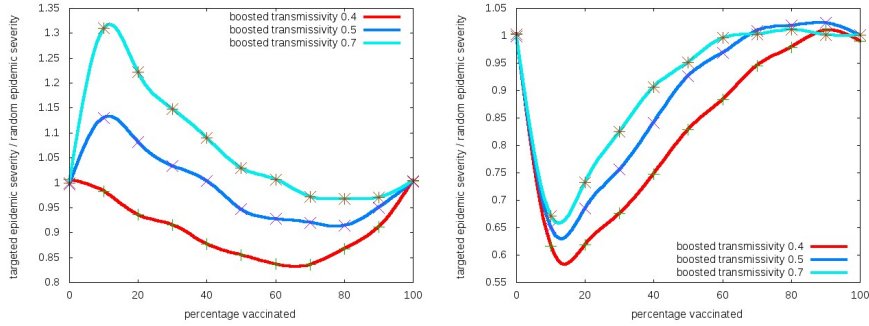


Figure 5.5: Epidemic severity comparison of random and targeted intervention strategies in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the ratio of the epidemic severity in targeted intervention strategy and the epidemic severity in random intervention strategy. $p = 0.25$, and $p_s = 0.35$.

5.4 Simulations

In order to validate our findings, we carried out comprehensive simulations over a wide range of networks, listed in Table 5.1.

The disease transmission is a random process, defined by the parameter set (p, p_m, p_f, p_v) . If a node u becomes infectious, it will infect each of its susceptible neighbors independently with probability p , referred as base transmissivity. Each node in the graph is either vaccinated or not. If a node u is not vaccinated, we label it as UV. If a node u 's

5.4 Simulations

Table 5.1: Descriptions of the networks used in the paper. For each network we show its type, name, number of nodes n and edges m .

	name	n	m	description
Human contact	con- NewRiverValley [29]	74,375	1,888,833	Synthetic human contact network for New River Valley county in Virginia.
Social communication	commu- Enron mail [87, 3]	36,691	367,666	Email communication network in a company.
Peer-to-peer network	Gnutella [116, 4]	10,876	39,994	Gnutella peer-to-peer file sharing network from August 2002
Random graphs	Preferential attachment [28]	100,000	300,000	Generated using Python NetworkX library.
	Erdős and Rényi [60]	100,000	5,000,000	

vaccine fails, we label it as VF. Otherwise, we label it VS. Both UV and VF nodes are susceptible. If a node with vaccine failure is infected then its risk behavior changes, resulting in boosted transmissivity p_m . In the one-sided model a node infects all its susceptible neighbors with boosted transmissivity p_m , while in the two-sided model it only infects those neighbors with boosted transmissivity p_m that have also had a failed vaccination. Parameter p_v denotes the probability that a node is vaccinated.

In our simulation, every node is labeled with UV, VS, VF with probability $1 - p_v$, $p_v(1 - p_f)$, and $p_v p_f$, respectively. All nodes labeled VS are removed from the graph. Each edge (u, v) connecting two surviving nodes u and v is “open”, which corresponds to disease transmission on this edge, or “close” with some probability depending on the model - (i) in the one-sided model, edge (u, v) is open with probability p if both u and v are labeled UV, and is open with probability p_m if one of u and v is labeled VF; (ii) in the two-sided model, edge (u, v) is open with probability p , unless both u and v are labeled VF. The closed edges are removed from the graph. In the residual graph, the connected component containing a specific node u is the (random) subset of nodes infected, if the disease starts at u . Let C_1, C_2, \dots, C_k be the resulting connected components, then $\sum_i |C_i|^2 / n$ denotes the expected outbreak size of the disease starting from a random initial node, which we referred as epidemic severity. Since the disease

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transmission is a random process, for a fixed parameter set we run the simulation for 10 iterations, and take the average value of the epidemic severity. We verified that the epidemic severity is tightly concentrated around the mean, thus the average value of the epidemic severity is a good measure.

We want to confirm our findings: (i) both one-sided and two-sided behavior changes can lead to perverse outcomes (less vaccination is more effective, more precisely, as the vaccinated fraction p_v is varied, the epidemic severity shows a non-monotone behavior); (ii) in both one-sided and two-sided behavior changes, targeted vaccination can be strictly worse than random vaccination for some level of vaccine coverage. For each graph, we run simulations over wide range of parameter set (p, p_m, p_f, p_v) , and generate the following 4 sets of plots to validate our findings.

- First set of plots shows how the change of boosted transmissivity will affect the perverse outcomes, as shown in Figure 5.6, 5.10, 5.14, and 5.18. The x -axis is p_v (percentage of vaccinated population) and the y -axis is the epidemic severity (expected percentage of nodes getting infected). We fix the base transmissivity p and the vaccination success probability p_s , then plot the curves for different boosted transmissivity.
- Second set of plots shows how the change of base transmissivity will affect the perverse outcomes, as shown in Figure 5.7, 5.11, 5.15, and 5.19. The x -axis is p_v and the y -axis is the epidemic severity. We fix the vaccination success probability p_s and keep the boosted transmissivity p_m twice the base transmissivity p (i.e. $p_m = 2p$), then plot the curves for different base transmissivity.
- Third set of plots shows how the change of vaccination success probability will affect the perverse outcomes, as shown in Figure 5.8, 5.12, 5.16, and 5.20. The x -axis is p_v and the y -axis is the epidemic severity. We fix the base transmissivity p and the boosted transmissivity p_m , then plot the curves for different vaccination success probability.
- Fourth set of plots shows the finding that targeted vaccination can be strictly worse than random vaccination, as shown in Figure 5.9, 5.13, 5.17, and 5.21. The x -axis is p_v and the y -axis is the ratio between the epidemic severity under targeted vaccination strategy and the epidemic severity under random vaccination

strategy. If y value is bigger than 1, it means targeted strategy is worse than random strategy. We fix the base transmissivity p and the vaccination success probability p_s , then plot the curves for different boosted transmissivity.

In order to capture real disease transmission through simulations, we find typical values of R_0 , the basic reproduction number, for many diseases such as influenza and HIV [65, 49, 125]. Then, we divide R_0 by the average degree of the graph, and use it as the base transmissivity p . For vaccination success probability, we use the efficacy for real vaccines [61, 70, 1].

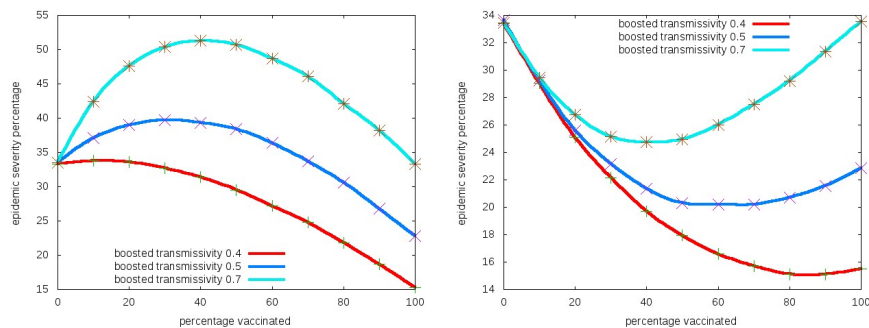


Figure 5.6: Epidemic severity with different boosted transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.25$ and $p_s = 0.35$.

5.5 Conclusion

In conclusion, risk behavior change in conjunction with failure of prophylactic interventions can have perverse non-monotone effects on the spread of diseases. This study has explicitly identified sidedness as an attribute of risk behavior change that needs to be taken into account in public policies for vaccinations and antiviral treatments. For one-sided risk behavior change, it is imperative to have sufficiently high levels of coverage, while two-sided situations require both high coverage as well as programs aimed at reducing risky behavior. Our results echo the central premise of Blower-McLean that the development of efficacious prophylactic treatments and increasing their coverage need to go hand in hand with behavioral intervention strategies. These issues need to

5. CONTROLLING NEGATIVE DIFFUSION IN THE PRESENCE OF RISK BEHAVIOR CHANGES

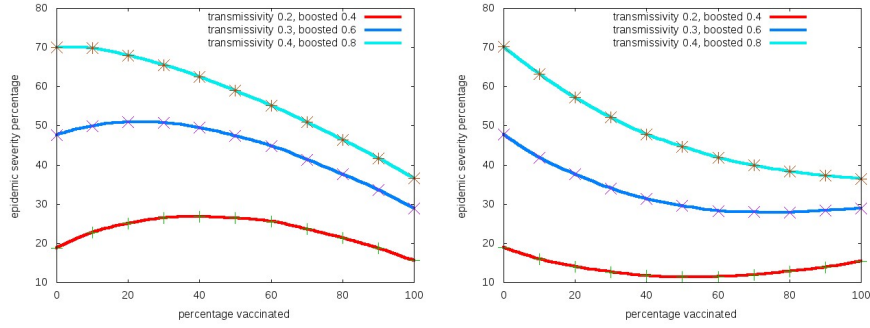


Figure 5.7: Epidemic severity with different transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p_s = 0.35$, and $p_m = 2p$.

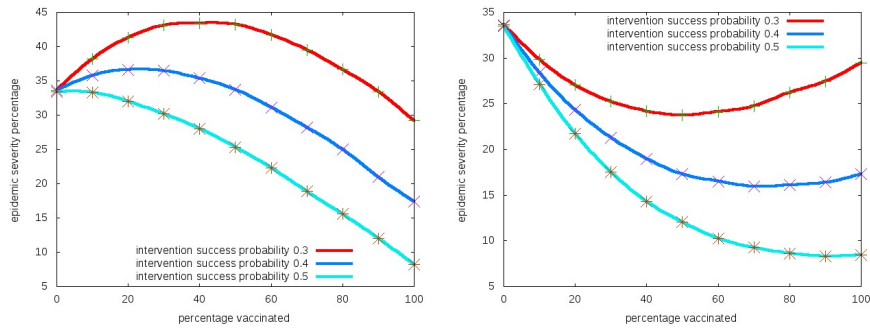


Figure 5.8: Epidemic severity with different intervention success probabilities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.25$, and $p_m = 0.5$.

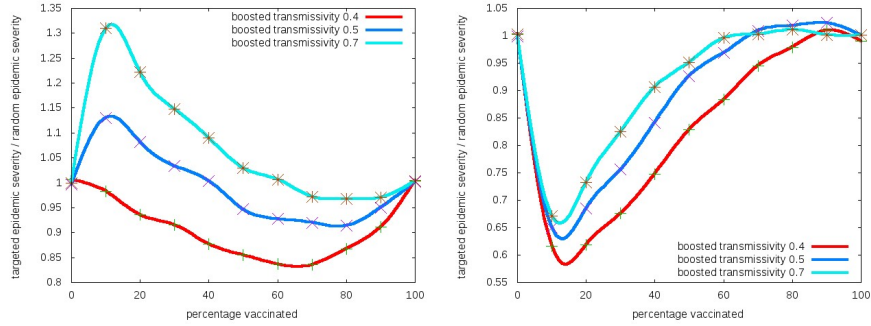


Figure 5.9: Epidemic severity comparison of random and targeted intervention strategies in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the ratio of the epidemic severity in targeted intervention strategy and the epidemic severity in random intervention strategy. $p = 0.25$, and $p_s = 0.35$.

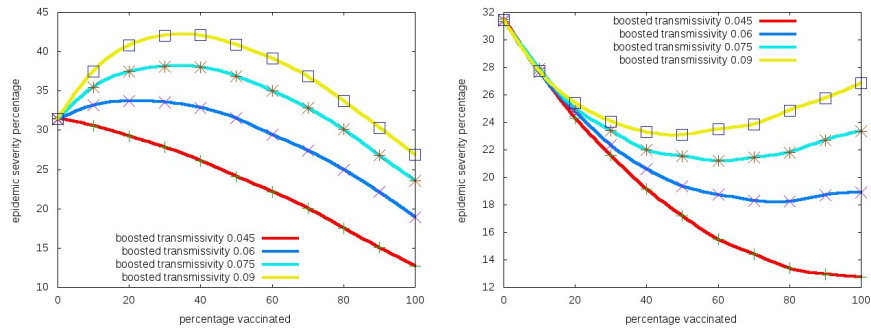


Figure 5.10: Epidemic severity with different boosted transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.03$ and $p_s = 0.35$.

5. CONTROLLING NEGATIVE DIFFUSION IN THE PRESENCE OF RISK BEHAVIOR CHANGES

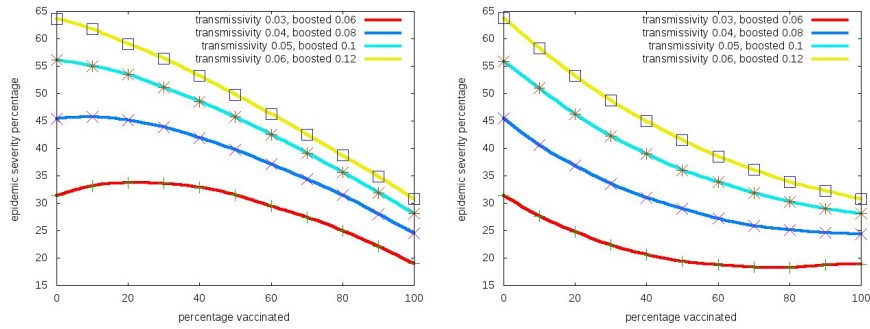


Figure 5.11: Epidemic severity with different transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p_s = 0.35$, and $p_m = 2p$.

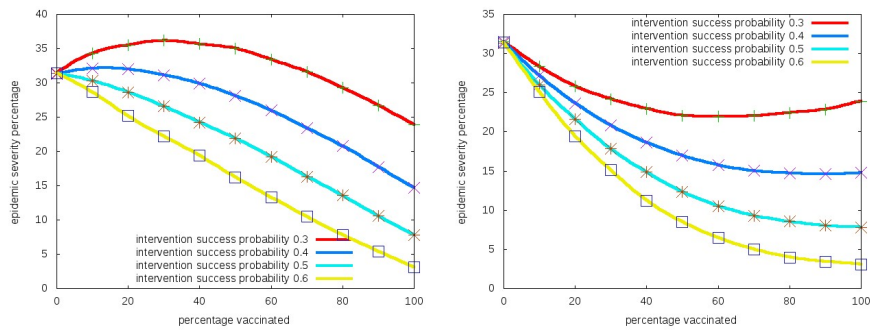


Figure 5.12: Epidemic severity with different intervention success probabilities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.03$, and $p_m = 0.06$.

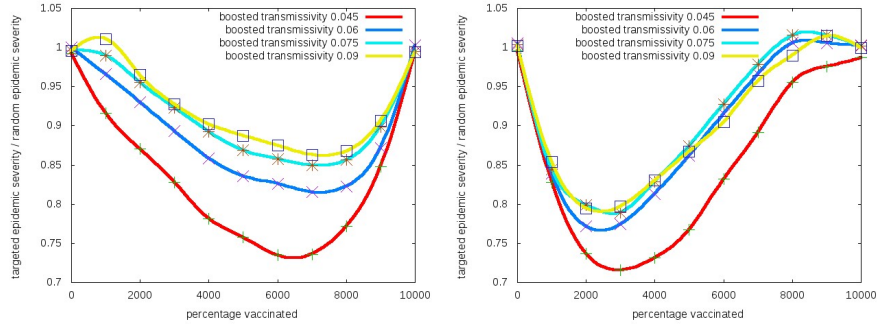


Figure 5.13: Epidemic severity comparison of random and targeted intervention strategies in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the ratio of the epidemic severity in targeted intervention strategy and the epidemic severity in random intervention strategy. $p = 0.03$, and $p_s = 0.35$.

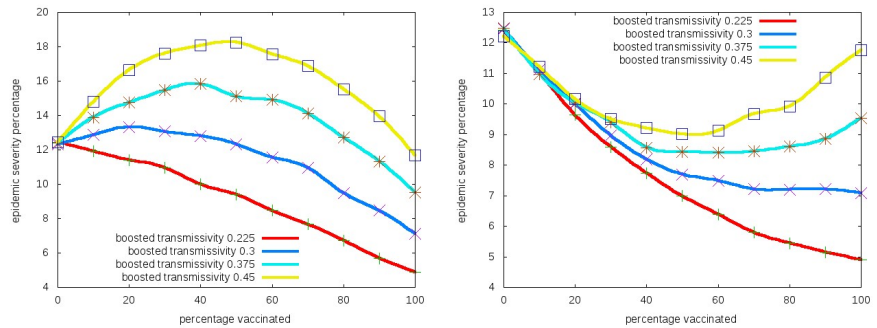


Figure 5.14: Epidemic severity with different boosted transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.15$ and $p_s = 0.35$.

5. CONTROLLING NEGATIVE DIFFUSION IN THE PRESENCE OF RISK BEHAVIOR CHANGES

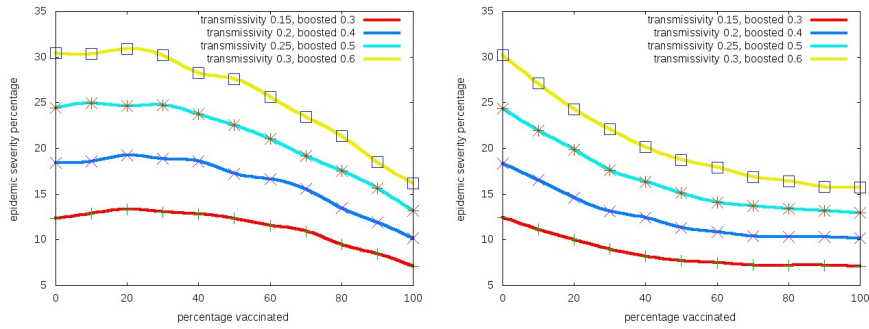


Figure 5.15: Epidemic severity with different transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p_s = 0.35$, and $p_m = 2p$.

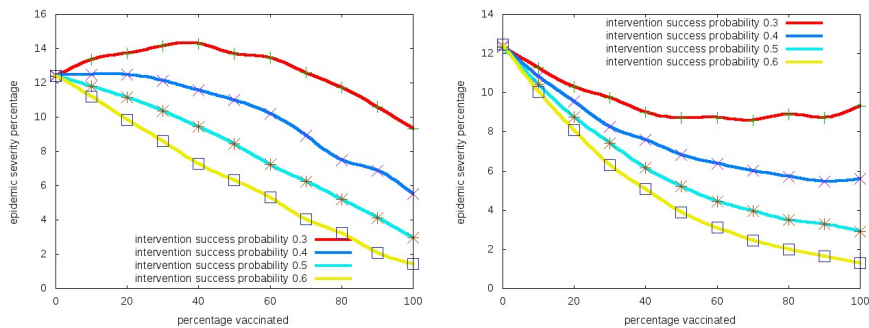


Figure 5.16: Epidemic severity with different intervention success probabilities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.15$, and $p_m = 0.3$.

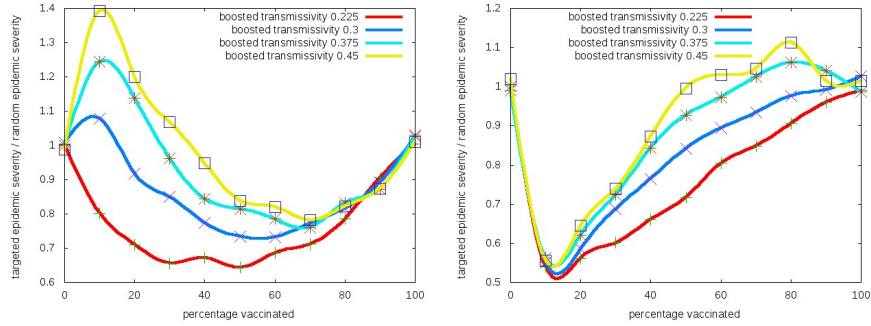


Figure 5.17: Epidemic severity comparison of random and targeted intervention strategies in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the ratio of the epidemic severity in targeted intervention strategy and the epidemic severity in random intervention strategy. $p = 0.15$, and $p_s = 0.35$.

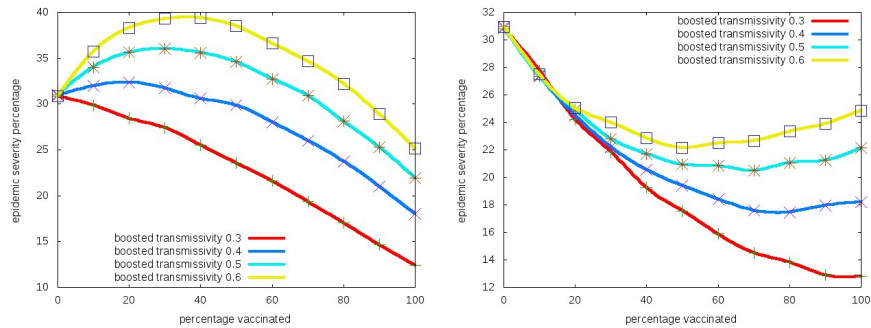


Figure 5.18: Epidemic severity with different boosted transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.2$ and $p_s = 0.35$.

5. CONTROLLING NEGATIVE DIFFUSION IN THE PRESENCE OF RISK BEHAVIOR CHANGES

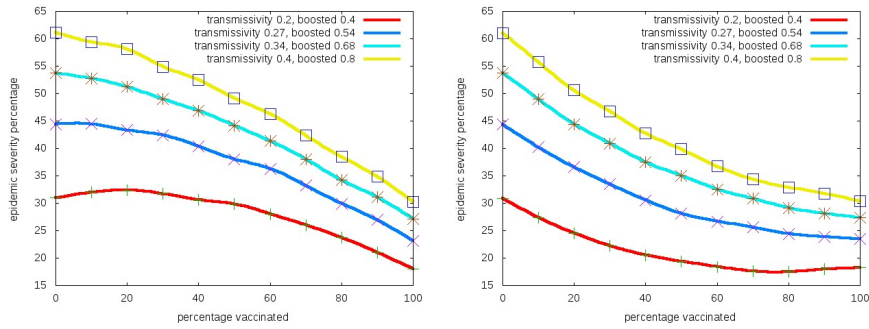


Figure 5.19: Epidemic severity with different transmissivities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p_s = 0.35$, and $p_m = 2p$.

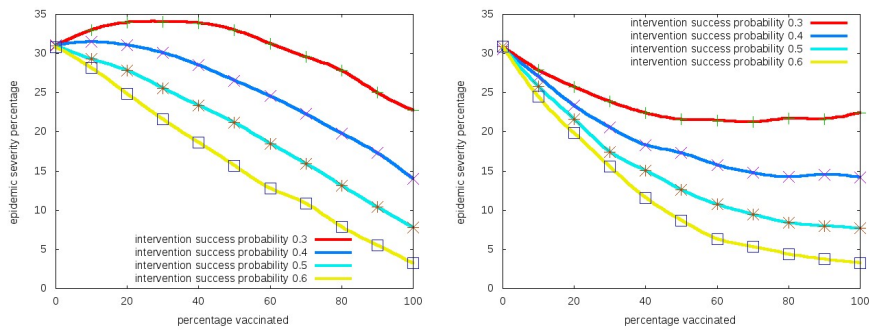


Figure 5.20: Epidemic severity with different intervention success probabilities in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the expected percentage of nodes getting infected. $p = 0.2$, and $p_m = 0.4$.

5.5 Conclusion

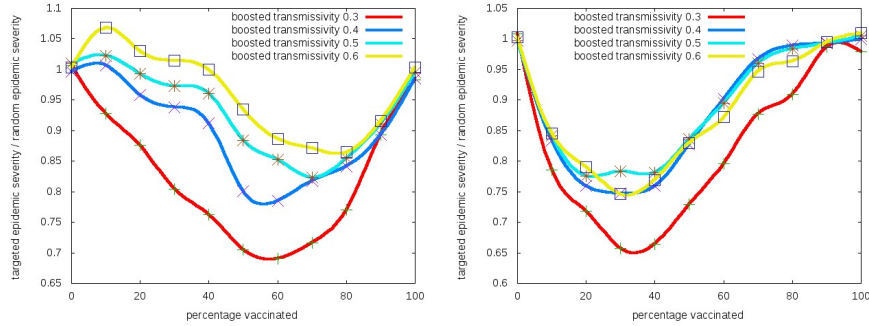


Figure 5.21: Epidemic severity comparison of random and targeted intervention strategies in one-sided (left) and two-sided (right) risk behavior models. x -axis is the percentage of nodes taking interventions, and y -axis is the ratio of the epidemic severity in targeted intervention strategy and the epidemic severity in random intervention strategy. $p = 0.2$, and $p_s = 0.35$.

be revisited in the context of new anti-retroviral treatments being considered for HIV [61].