Active risk aversion in SIS epidemics on networks

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Susceptible-Infected-Susceptible (SIS) model

$$\mathbf{S} \xrightarrow{\delta I} \mathbf{I}$$

Well-mixed population model $\ \dot{p} = -\delta p + (1-p)\,\beta p$

 $p \in [0,1]\,$ proportion of population that is infected

eta infection rate or contact rate, δ recovery rate

SIS model on a network



Recovery rate in population j

Contact with infected individuals from neighboring populations

 $A=(a_{jk})$ $\,$ Inter-population contact graph $\,\,a_{jk}\in\{0,1\}$

SIS model on a network

Transcritical bifurcation: IFE loses stability, unique globally stable EE appears



SIS model on a network

Transcritical bifurcation: IFE loses stability, unique globally stable EE appears



Motivation

Classic infection models do not account for **sociocultural phenomena** that can, in turn, affect infection spread:

- Spread of information and misinformation about infection rates
 - \odot Social media and traditional media
 - \odot Remember the COVID dashboards?
- Public health messaging and local policies
- In-group/out-group effects, social norms, peer pressure

Yang et al. "Sociocultural determinants of global mask-wearing behavior." *Proceedings of the National Academy of Sciences* (2022)



Some related works

Coupled epidemics and opinion dynamics

She et al. "On a networked SIS epidemic model with cooperative and antagonistic opinion dynamics." IEEE Transactions on Control of Network Systems (2022)

Risk avoidance with game-theoretic strategies

Ye et al. "Game-theoretic modeling of collective decision making during epidemics." Physical Review E (2021)

Satapathi et al. "Coupled evolutionary behavioral and disease dynamics under reinfection risk." IEEE Transactions on Control of Network Systems (2023).

Adaptive contact rate control policies for mitigating infection

Walsh et al. "Decentralised adaptive-gain control for eliminating epidemic spreading on networks." arXiv:2305.16658 (2023).



Our focus: risk perception

Assumption: populations assess infection risk through *local, dynamic* observations on a communication network

$$\tau_{s}\dot{p}_{sj} = -p_{sj} + \frac{1}{\hat{d}_{j}} \left(\sum_{k=1}^{N} \hat{a}_{jk} p_{k} \right)$$

Time scale
Perceived infection
risk for population j
$$\hat{d}_{j} = \sum_{k=1}^{N} \hat{a}_{jk}$$

Our focus: risk perception

Assumption: populations update their contact rate with others in response to their perception of infection risk



actSIS dynamics

Two networks over the same vertices: contact graph drives infection, communication graph drives risk perception

Key questions
$$\dot{p}_j = -\delta_j p_j + (1 - p_j) \bar{\beta} \alpha_j(p_{sj}) \sum_{k=1} a_{jk} p_k$$

How do the risk response strategies affect steady-state infection level at each node?

What is the role of the communication network structure in shaping the infection level on the graph?

$$\tau_s \dot{p}_{sj} = -p_{sj} + \frac{1}{\hat{d}_j} \left(\sum_{k=1}^N \hat{a}_{jk} p_k \right)$$

N

First result: transcritical bifurcation of IFE

Theorem (Local bifurcation of EE)

Assume A, \hat{A} are irreducible, define $D = \text{diag}(\delta_j)$. 1) The infection-free equilibrium (IFE) is locally exponentially stable for $\bar{\beta} < \bar{\beta}^*$

$$\bar{\beta}^* = \frac{1}{\rho(\operatorname{diag}\{(\alpha_1(0), \dots, \alpha_N(0))\}AD^{-1})}$$

and unstable for $\bar{\beta} > \bar{\beta}^*$.

Bifurcation point does **not** depend on communication graph, does depend on risk avoidance strategy.

First result: transcritical bifurcation of IFE

Theorem (Local bifurcation of EE)

Assume A, \hat{A} are irreducible, define $D = \text{diag}(\delta_j)$. 2) Let $\mathbf{v}^* \succ \mathbf{0}$, $\mathbf{w}^* \succ \mathbf{0}$ be the right and left null eigenvectors of $-D + \bar{\beta}^* \text{diag}\{(\alpha_1(0), \dots, \alpha_N(0))\}A$, and define

$$K_1 = \sum_{i=1}^N w_i^* \left((\hat{\Delta}^{-1} \hat{A} \mathbf{v}^*)_i \alpha_i'(0) (A \mathbf{v}^*)_i - 2v_i^* \alpha_i(0) (A \mathbf{v}^*)_i \right) \neq 0$$

At $\bar{\beta} = \bar{\beta}^*$, a branch of EE $\mathbf{p}^* \succ \mathbf{0}$, $\mathbf{p}_s^* = \hat{\Delta}^{-1} \hat{A} \mathbf{p}^* \succeq \mathbf{0}$ appear in a transcritical bifurcation along an invariant center manifold tangent to $\operatorname{span}(\bar{\mathbf{v}})$ at $(\mathbf{p}, \mathbf{p}_s, \bar{\beta}) = (\mathbf{0}, \mathbf{0}, \bar{\beta}^*)$, where $\bar{\mathbf{v}} = (\mathbf{v}^*, \hat{\Delta}^{-1} \hat{A} \mathbf{v}^*)$. If $K_1 < 0$, the EE appear for $\bar{\beta} > \bar{\beta}^*$ and are locally exponentially stable; if $K_1 > 0$, the EE appear for $\bar{\beta} < 0$ and are unstable.

Takeaway so far

- Typical bifurcation: transcritical bifurcation of **stable** EE
- Sometimes: transcritical bifurcation of **unstable** EE, bistability
- Role of communication graph structure: so far unclear... does it matter?

Specialize model: regular graphs, homogeneous parameters, risk averters

$$\dot{p}_{j} = -\delta p_{j} + (1 - p_{j}) \,\bar{\beta} \alpha(p_{sj}) \sum_{k=1}^{N} a_{jk} p_{k} \qquad \tau_{s} \dot{p}_{sj} = -p_{sj} + \frac{1}{\hat{d}} \left(\sum_{k=1}^{N} \hat{a}_{jk} p_{k} \right)$$
$$\alpha'(p) < 0, \quad p \in (0, 1)$$

Bifurcating endemic equilibrium is uniform, refer to it as UEE

$$(\mathbf{p}^*, \mathbf{p}^*_s) = (p^* \mathbf{1}, p^* \mathbf{1})$$

Second result: risk aversion lowers UEE infection level compared to standard SIS



Communication graph does not seem to play a major role here either...

Next step: study stability of UEE

Recall: UEE is always stable and unique in standard SIS on a regular graph; is this true for actSIS?

$$J_{IFE} = \begin{pmatrix} -\delta I + \bar{\beta}\alpha(0)A & 0\\ \frac{1}{\tau_s}\hat{\Delta}^{-1}\hat{A} & -\frac{1}{\tau_s}I \end{pmatrix}$$

$$J_{UEE}(p^*) = \begin{pmatrix} -\delta \left(1 + \bar{\beta}dp^*\alpha(p^*)\right)I + \frac{\delta}{d}A & \delta \frac{\alpha'(p^*)}{\alpha(p^*)}p^*I \\ \frac{1}{\tau_s}\frac{1}{\hat{d}}\hat{A} & -\frac{1}{\tau_s}I \end{pmatrix}$$

Next step: study stability of UEE

$$J_{UEE}(p^*) = \begin{pmatrix} -\delta \left(1 + \bar{\beta} dp^* \alpha(p^*)\right) I + \frac{\delta}{d} A & \delta \frac{\alpha'(p^*)}{\alpha(p^*)} p^* I \\ \frac{1}{\tau_s} \frac{1}{\hat{d}} \hat{A} & -\frac{1}{\tau_s} I \end{pmatrix}$$
$$\mathcal{J}_{eff}(p^*) = -\delta \left(1 + \bar{\beta} dp^* \alpha(p^*)\right) I + \frac{\delta}{d} A + \frac{\delta}{\hat{d}} \frac{\alpha'(p^*)}{\alpha(p^*)} p^* \hat{A}$$

Combined effect of communication and contact graphs!

Stability of UEE

Theorem

Let A and \hat{A} correspond to connected regular graphs with degrees d and \hat{d} , respectively, and assume $\delta_j = \delta$, $\alpha_j = \alpha$ for all $j \in \mathcal{V}$. Define

$$g(p) = -\delta p + (1-p) \left(\lambda_{max} \left(\frac{1}{d} A + \frac{1}{\hat{d}} \frac{\alpha'(p)}{\alpha(p)} p \hat{A} \right) - 1 \right) \,.$$

If g(p) = 0 for some $p \in (0, 1)$, then there exists a critical value $\overline{\beta}_2 = \frac{\delta}{d(1-p_2)\alpha(p_2)} > \overline{\beta}^*$ where p_2 is the smallest $p \in (0, 1)$ for which g(p) = 0. If p_2 is not a unique solution to g(p) = 0 in (0, 1), the UEE $(\mathbf{p}^*, \mathbf{p}_s^*) = (p^*\mathbf{1}, p^*\mathbf{1})$ is locally exponentially stable whenever $\overline{\beta}^* < \overline{\beta} < \overline{\beta}_2$ and unstable for $\overline{\beta} \in (\overline{\beta}_2, \overline{\beta}_3)$ for some $\overline{\beta}_3 = \frac{\delta}{d(1-p_3)\alpha(p_3)}$, $p_3 > p_2$, $g(p_3) = 0$. If p_2 is a unique solution to g(p) = 0 in (0, 1), then the UEE is unstable for all $\overline{\beta} > \overline{\beta}_2$.

40 populations, d = 5, sparse contact graph

40 populations, d = 30, dense contact graph

Contact graph contributes little to the shape of g(p), communication graph has the dominant effect

Loss of stability = bifurcation of a $N = 26, d = 5, \hat{d} = 23$ stable heterogeneous state

Heterogeneity arises from a highly homogeneous model!

40 populations, d = 5, sparse contact graph

40 populations, d = 30, dense contact graph

Bonus simulation: mixed networks

Thoughts and future directions

- EE stability results hold beyond regular graphs, but are trickier to formally prove and interpret
- Shape and concavity of the risk response function matters a lot
- With directed networks, may sometimes get a Hopf bifurcation (oscillations); this has been found in other, more complex models
- Potential implication: more coverage/public awareness to suppress the secondary bifurcation → some populations benefit, but some actually increase infection!

Questions?

$$\begin{split} \dot{p}_{j} &= -\delta_{j}p_{j} + (1 - p_{j})\,\bar{\beta}\alpha_{j}(p_{sj})\sum_{k=1}^{N}a_{jk}p_{k} \\ \tau_{s}\dot{p}_{sj} &= -p_{sj} + \frac{1}{\hat{d}_{j}}\left(\sum_{k=1}^{N}\hat{a}_{jk}p_{k}\right) \end{split} \text{Infection}$$

Preprint: https://arxiv.org/abs/2311.02204