# Network effects and behavioral feedback in epidemic models

## Abstract

The COVID-19 pandemic has highlighted two fundamental challenges in epidemic modeling: the need to account for population heterogeneity and the crucial role of behavioral adaptations. This thesis addresses both aspects by extending the classical SIR (Susceptible-Infected-Recovered) framework to settings that incorporate network effects and endogenous behavioral feedback. The overall aim is to understand how contact patterns and behavioral responses shape epidemic dynamics, and how such understanding can inform the design of effective mitigation strategies.

Adopting the classical SIR framework for its analytical tractability and generality, we first investigate a behavioral-feedback model within a homogeneous population, where the transmission rate evolves dynamically as a function of the epidemic state. In this setting, we establish a sufficient condition ensuring the unimodality of the infection curve and provide closed-form expressions for the infection peak and the final epidemic size, thus generalizing well-known results from classical SIR theory. We then extend the model to a network-based SIR framework with endogenous behavioral feedback. In this setting, the nodes of the network represent subpopulations that share similar characteristics, such as infectivity, susceptibility, activity levels and behavioral patterns. The interaction matrix, encoding the contact rates between groups, evolves dynamically in response to the epidemic state. We first analyze the asymptotic behavior of the system, deriving explicit invariants of motion for the case of constant interaction matrices, and show that when the matrix is constant and of rank-1, the limit equilibria can be characterized.

We then focus on the transient dynamics, introducing a weighted aggregate infection measure and proving its unimodality under suitable conditions, thereby generalizing classical SIR properties to networked settings. We study the infection curves at the single node level and show that they can undergo at most two changes in monotonicity before eventually decreasing. We further extend some of these results to a broader class of models with state-dependent interaction matrices, emphasizing how the interplay between network structure and feedback mechanisms can give rise to complex and heterogeneous epidemic trajectories.

Finally, we address an optimal control problem for behavioral-feedback SIR epidemic models, where interventions such as lockdowns are constrained by healthcare capacity. Through a geometric analysis of the system's state space, we derive optimal strategies under general monotonicity assumptions, showing that a filling-the-box policy minimizes cumulative costs. Under this optimal strategy, the infection is initially allowed to grow until the critical threshold is approached, at which point strong restrictions are applied and then gradually

relaxed. We also show that, when behavioral responses violate the monotonicity assumptions, more adaptive strategies may be required.

Overall, this work contributes a rigorous analytical foundation for understanding the interplay between behavior, network structure, and epidemic control, and it offers new tools for designing robust intervention strategies in the face of complex population dynamics.