Learning Epidemiology by Doing: The Empirical Implications of a Spatial-SIR Model with Behavioral Responses: External Appendix[§]

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A Appendix: Theoretical Structure of SIR and Spatial-SIR

In this appendix we construct the theoretical structure of SIR and Spatial-SIR as Markov chains processes. We present the structure of these models in discrete time first, for consistency with the simulation analysis in the text.

A.1 SIR

The society is populated by N individuals. Agents are ex-ante identical in terms of demographic characteristics. Let $\mathcal{S}$ denote the individual state-space. In the SIR model, the state-space is $\mathcal{S} = \{S, I, R\}$, indicating Susceptibles, Infected, and Recovered. Let $h_t^i \in \mathcal{S}$ denote the state of agent i at time t. Let $h_t = \frac{1}{N}[S_t, I_t, R_t] \in \Delta^{\mathcal{S}}$ denote the distribution of the population across the state-space.[1] The SIR model is represented by a Markov Chain:

$$prob(h_{t+1}^i = h' \mid h_t^i = h) = T_{h\,h'}(h_t)$$

where $T_{h\,h'}(h_t)$ is the generic element of a $\mathcal{S} \times \mathcal{S}$ double-stochastic (transition) matrix $T(h_t)$. The dependence of the transition matrix on h_t, the distribution of the population across the state-space (the aggregate state of the economy), is a mean-field property justified in this class of models by random matching in the population.

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[1]Abusing notation, we let we let the capital letters indicating a state also denote the fraction of the population in that state; and we let $\mathcal{S}$ denote both the set and its numerability.

More specifically, the matrix $T_{h\,h'}(h_t)$ is determined by the following transitions:

S $\longrightarrow$ I. A Susceptible agent becomes infected upon contact with an Infected, with probability πI.

A $\longrightarrow$ R. An agent Infected at t, at any future period, can Recover with probability ρ.

R Recovered is absorbing state of the dynamic process (agents entering this state never leave). This assumes Recoved agents are immune to infection.

The resulting dynamical system for the distribution of the population across the state-space, h_t, is the following,

$$h_{t+1} = T(h_t)h_t.$$

The dynamical system can be solved for in closed form, see e.g., Moll (2020), Neumeyer (2020).

A.2 Spatial-SIR

We now add a spatial dimension to the SIR model. We also expand the state space to better caputure several relevant aspects of the SARS-CoV-2 infection. Specifically, we split the I state into Asymptomatics and sYmptomatics, A and Y. We also add explicitly the state D, for Dead. Hence, $\mathcal{S} = \{S, A, Y, R, D\}$. We maintain the notation $h_t^i \in \mathcal{S}$ to denote the state of agent i at time t; and $h_t = \frac{1}{N}[S_t, A_t, Y_t, R_t, D_t] \in \Delta^{\mathcal{S}}$ to denote the distribution the N agents in the population across the state-space.

Agents are located in space, e.g., a lattice, which we call "the City." Agents are ex-ante identical in terms of demographic characteristics and symmetric in terms of location in space. A (Markov) transition process between states governs the dynamics of the system from the initial condition, at day $t = 0$. The spatial dimension maps the stochastic process into a local interaction model, a model in which agents' contacts are not the results of random matching but rather of local matching, with agents close in space (geographical distance as a metaphor for social distance). Let H_t denote the configuration of agent at time t, a vector $\left[h_t^1, h_t^2, \ldots, h_t^I\right]$; the set of all configuration is denoted $\mathcal{H}$. The local interaction model is characterized by

$$prob(h_{t+1}^i = h' \mid h_t^i = h) = T_{h\,h'}(H_t).$$

More specifically, the matrix $T_{h\,h'}(H_t)$ is determined by the following transitions:

S $\longrightarrow$A. Susceptible agents become infected upon contact with an Asymptomatic, with probability π.[2] A contact is defined to occur when agents are at a geographical

[2]Susceptible agents are not infected upon contact with a sYmptomatic agent; this is to capture the fact that sYmptomatic agents are either isolated at home or in the hospital. They movements in the City are vacuous.

distance in space $\leq p$.

A $\longrightarrow$ Y, R. An Asymptomatic agent infected at t, at any future period, can become sYmptomatic with probability ν, or can Recover with probability ρ.

Y $\longrightarrow$ R, D. An agent who has become sYmptomatic at t, at any future period, can Recover with probability ρ, or can Die with probability δ.

D, R. Dead and Recovered are absorbing states of the dynamic process. As we noted, this assumes Recoved agents are immune to infection.

Abusing notation, a transition matrix $T(H_t)$ in the space of possible configurations $\mathcal{H}$ can be constructed from $T_{h\,h'}(H_t)$.[3] The resulting dynamical system for configurations H_t is

$$H_{t+1} = T(H_t)H_t.$$

But Spatial-SIR accounts for agents possibly coming into contact after moving randomly in space.[4] Let the operator P_t, mapping $H_t \in \mathcal{H}$ into $P_t \circ H_t \in \mathcal{H}$, represent a configuration after a random permutation of the position of the agents, indexed by i. Before transitioning from the state at t to the state at $t+1$ the agents' locations are permutated randomly. The local interaction model is characterized by

$$prob(h^i_{t+1} = h' \mid h^i_t = h) = T_{h\,h'}(P_t \circ H_t).$$

The resulting dynamical system for configurations H_t is:[5]

$$P_t \circ H_{t+1} = T(P_t \circ H_t)P_t \circ H_t. \tag{1}$$

The dynamical system is difficult to formally characterize, besides (possibly) an ergodicity result, with respect to initial conditions specifying, at day $t = 0$, a random allocation of agents on evenly spaced locations in the City, all of them Susceptible, excepts for $A_0 > 0$ agents who are exogenously infected Asymptomatics. All our simulations, for all parameter values and initial conditions, converge to a unique ergodic distribution over the state space $h_t = \frac{1}{N}[S_t, A_t, Y_t, R_t, D_t] \in \Delta^{\mathcal{S}}$.

[3] This is an ugly looking operation, but formally straighforward, as purely arithemetical.

[4] This is different from most mathematical literature on local interactions; see e.g., Kindermann and Snell (1980) and Liggett (2012).

[5] This representation is complicated in that the state space $\mathcal{H}$ is very large, and the permutation does not help. A simpler representation of $prob(h^i_{t+1} = h' \mid h^i_t = h)$ can be obtained as follows. Let I_t map locations $l \in \mathcal{L}$ into agents $i \in \mathcal{I}$. Assume at time $t = 0$ the map I_0 is an identity map so that the index i coincides with l. (This assumes, just for simplicity, that the numerability of agents is the same as that of locations.) Let $I_{t+1} = P \circ I_t$, $t \geq 0$. Fix an agent i and let l be the unique solution to $I_t(l) = i$. (As we constructed it, I_{t+1} is a byjection.) Let $NBHD_t(i) = \{i \in \mathcal{I} \mid i = I_t(l'),\ l - d \leq l' \leq l + d\}$. Then

$$prob(h^i_{t+1} = h' \mid h^i_t = h) = T_{h\,h'}([h^{i'}_t]_{i' \in NBHD(i)}).$$

A.3 SIR and Spatial-SIR in Continuous Time and Space

The SIR in continuous time model is the workhorse of the epidemiology literature, from Kermack and McKendrick (1927); see Hethcote (2000) for a thorough mathematical presentation. It is representated by the following system of differential equations:

$$\begin{aligned} \frac{dI}{dt} &= \beta S \frac{I}{N} - \rho I \\ \frac{dR}{dt} &= \rho I \\ N &= S + I + R \end{aligned}$$

The SIR has been extended to a continuous space s on some bounded domain Ω; see e.g., Chinviriyasit and Chinviriyasit (2010) and Wu et al. (2017). The Spatial-SIR is then represented by the following system of reaction-diffusion equations:

$$\begin{aligned} \frac{dI}{dt} - \alpha \Delta I &= \beta S \frac{I}{N} - \rho I \\ \frac{dR}{dt} \alpha \Delta I &= \rho I \\ N &= \int_\Omega (S + I + R) ds, \quad \text{for all } t \end{aligned}$$

where Δ is the Laplace operator, defined as the divergence ∇ of the gradient ∇, so that e.g., $\Delta I = \nabla^2 I$; and the boundary conditions, e.g.,

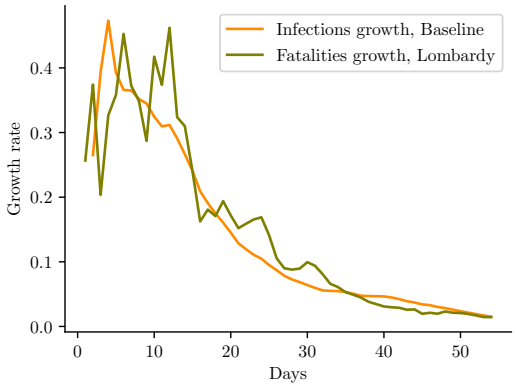
$$\partial_\eta S = \partial_\eta I = \partial_\eta R = 0$$

where η is the outward unit normal vector on the boundary of Ω, $\partial\Omega$.

B Appendix: Additional Figures

B.1 Baseline model calibration

Figure B.1: Growth rate of infections

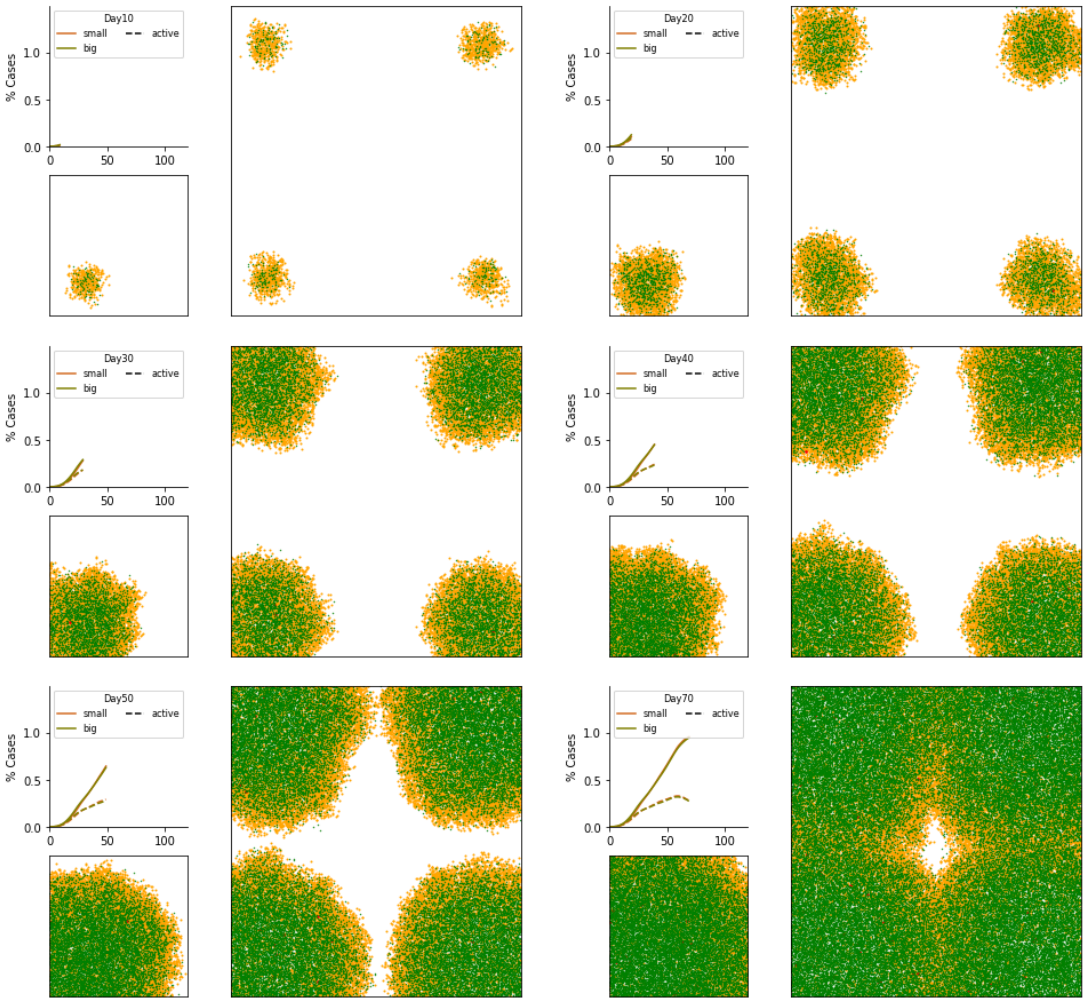


B.2 How the model scales

In Figure B.2 we compare the progression of the contagion at days 10, 20, 30, 40, 50, and 70, between the baseline city and a city with four times the population and the area (so that density is constant), and with four initial clusters of the same size as in the baseline located in symmetric locations. Each panel reports on the right the geographical location of infections in the bigger city, on the bottom left the geographical location of infections in the baseline (smaller) city, and on the top left the contagion rates.

The progression of the infection is almost entirely symmetric, barring minor effects due to the randomness of people's locations and movement. The top-right chart in each panel shows that both the fraction of active and total cases is nearly identical between the two Cities.

Figure B.2: Rescaling a City



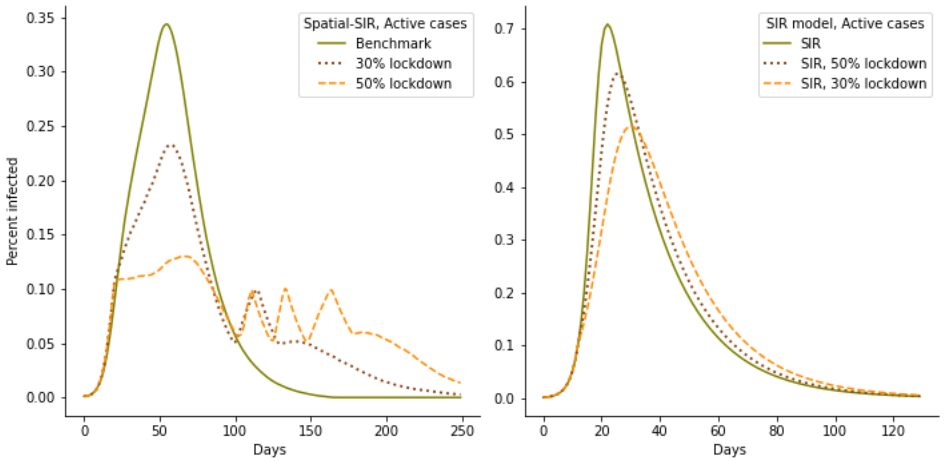
The figure illustrates the progression of infections in two cities. From the top-left panel proceeding right and down: day 10, 20, 30, 40, 50, and 70. The small city (bottom right square in each panel) is our baseline city. The large city (large square) is a city of four times the area, four times the population, and 4 times the initial cluster of infections, placed in simmetric positions geographically. The top-left chart in each punel illustrates the fraction of active cases and total cases at day t. Yellow dots are the the active cases, green dots are the recovered cases (susceptibles are omitted).

B.3 Lockdown policies

The role of local herd immunity appears very evident when comparing the effects of a lockdown policy (the typical Non-Pharmaceutical Intervention adopted in the SARS-CoC-2 epidemic) in SIR and in Spatial-SIR. Figure B.3 reports the dynamics

of active cases under lockdowns restricting the movements of 30% and 50% of the population. The lockdowns are imposed when the fraction of active cases reach 10% of the population and it is lifted when the fraction of active cases reaches 5%. The left panel reports results from Spatial-SIR, the right panel from SIR.

Figure B.3: Comparison of models with 30% and 50% lockdown policies



Left Panel: Spatial-SIR model, Right panel: SIR model. The lockdown is imposed when the fraction of active cases reaches 10% of the population, and lifted when the fraction returns to 5%

Lockdowns have a smooth effect on the dynamics of active cases in SIR (right panel), reducing the peak from 70% to 50% (for the 50% lockdown). Lifting the lockdown has minimal effects in SIR because, when active cases reach 5%, herd immunity is relatively far advanced. In Spatial-SIR, on the other hand, the lockdown sets local herd immunity immediately in action (especially so the 50% lockdown), dramatically reducing new cases (left panel). Cases however start surging as soon ad the lockdown is lifted, giving rise to the various waves/cycles (especially so for the 50% lockdown represented by the orange line).

In Figures B.4 (resp. B.5) we report, for both SIR and Spatial-SIR, additional outcomes of the epidemic dynamics under these a policies.

Figure B.4: Comparison of models with 30% lockdown

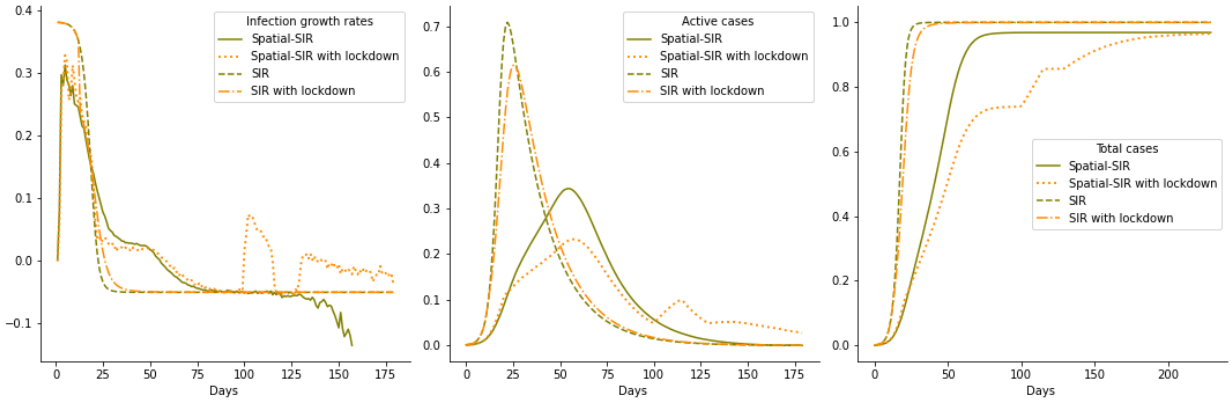
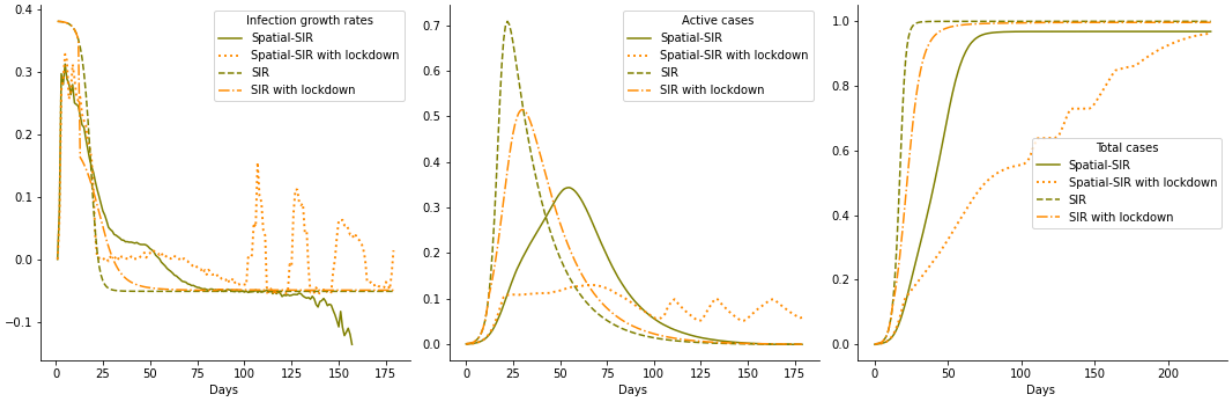


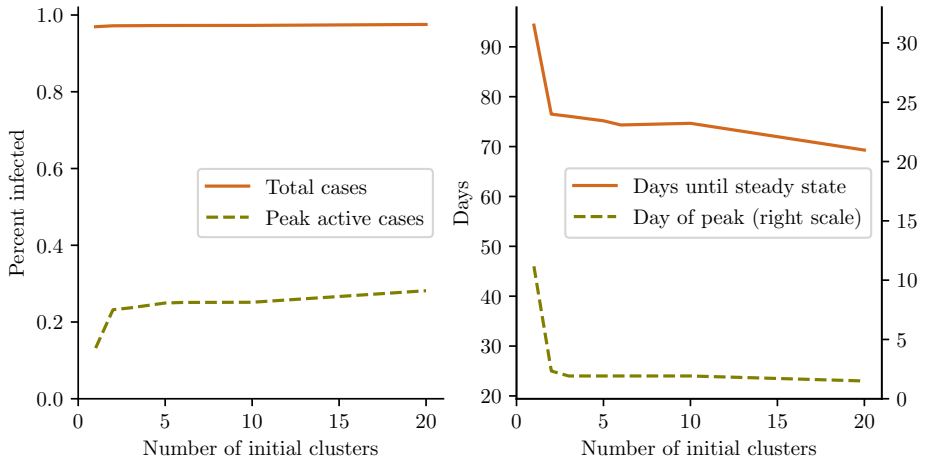
Figure B.5: Comparison of models with 50% lockdown



B.4 Number of clusters

In Figure B.6 we report the results of simulations varying the number of initial clusters from 0 to 20, in our otherwise baseline city, while keeping the number of initially infected agents constant. We observe that, with our calibrated parameters, the effect of increasing the number of initial clusters converges quite fast: when there are five or more initial clusters, increasing the number of initial clusters while keeping the number of initially infected the same, has no effect on the dynamics of the epidemics.

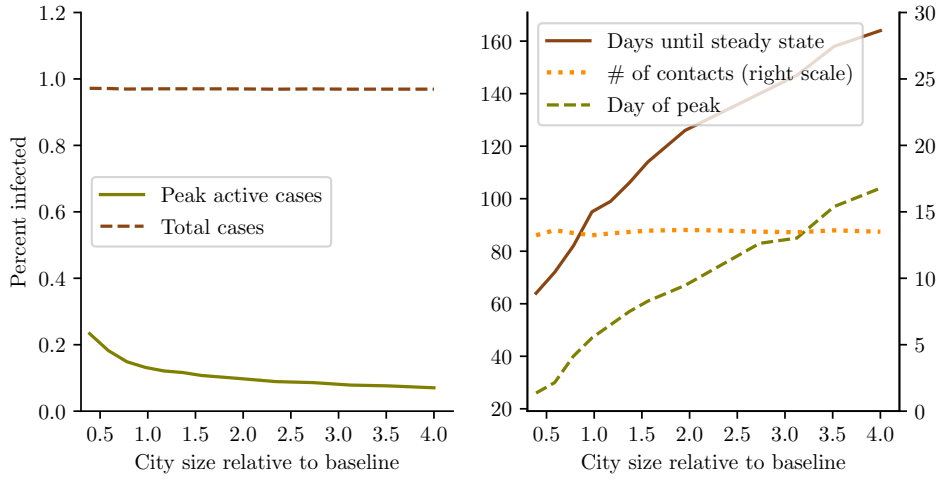
Figure B.6: The effect of the number of clusters



B.5 City size

In Figure B.7 we report the results of simulations varying the size of the City, in our otherwise baseline City, while keeping the size of the initial outbreak of the infection and City density constant. We observe that, with our calibrated parameters, the effect of increasing City size: the peak of active cases declines with size in a convex manner (less so the larger the city); the number of days it takes to reach the peak and the number of days to the stationary state (the end of the epidemic) both increases with size and do so with a slight concavity (less so the larger the city).

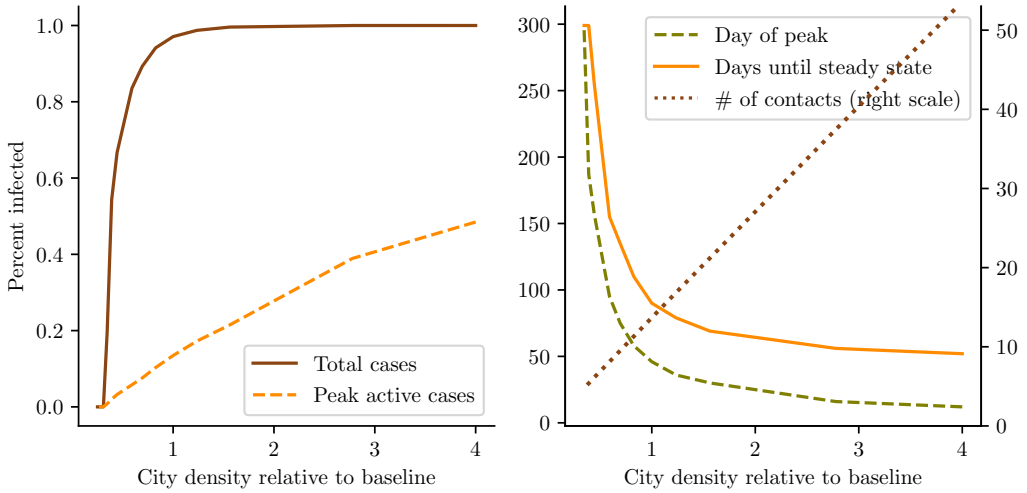
Figure B.7: City size comparisons



B.6 City density

In Figure B.8 we report the results of simulations varying City density, in our otherwise baseline City. We observe that, with our calibrated parameters: i) $(R^* + D^*)/N$ is increasing and concave in density; and the peak of $(A + Y)/N$ is also increasing and concave in density. In the right panel of Figure B.8 we see that the days it takes to reach the peak and the stationary state are decreasing and convex in density.

Figure B.8: The effect of varying city density with constant population



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