

# OPTIMAL DISEASE MITIGATION WITH ENDOGENOUS NETWORK RESPONSE

Ozan Candogan<sup>a</sup>   Michael König<sup>b,c</sup>   Kieran Marray<sup>b,e</sup>   Frank Takes<sup>d,e</sup>

<sup>a</sup>University of Chicago

<sup>b</sup>Vrije Universiteit Amsterdam

<sup>c</sup>ETH Zurich

<sup>d</sup>Leiden Institute for Advanced Computer Science

<sup>e</sup>Statistics Netherlands

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## OVERVIEW

### **Motivation:**

Individuals often adjust connections in response to spread of disease (see e.g Gupta et al. 2020, Chowell & Nishiura 2014, McKusick et al. 1985) – contact structure co-evolves with spread of disease (cf. Acemoglu et al. 2024, Carnehl et al. 2023).

## OVERVIEW

### **Motivation:**

Individuals often adjust connections in response to spread of disease (see e.g Gupta et al. 2020, Chowell & Nishiura 2014, McKusick et al. 1985) – contact structure co-evolves with spread of disease (cf. Acemoglu et al. 2024, Carnehl et al. 2023).

### **Here:**

Introduce rewiring into standard SIR model of spread of disease on a network – susceptible individuals rewire links from infectious to prevent contracting disease.

Use model to formulate optimal lockdown problem with endogenous network structure – neglecting rewiring leads to suboptimally tight policy, and both initial network structure and rewiring interact to improve efficiency of policy.

Apply to second wave of Covid-19 in the Netherlands, building population-scale initial contact network for flu-like infectious diseases by combining registry data and contact surveys.

## RELATED LITERATURE

**Economics (theoretical)** Optimal policy (Acemoglu et al. 2024, 2021, Birge et al. 2022, Cianfanelli et al. 2021), ‘behavioural’ SIRs (Atkeson et al. 2021, Bisin & Moro 2022, Goolsbee & Syverson 2021, Aguirregabiria et al. 2021).

**Epidemiology/networks (theoretical)** SIS process on adaptive networks (Gross et al. 2006), simulations of adaptation in SIR models (Lagorio et al. 2011).

**Empirical literature** Covid-19 (e.g Gupta et al. 2021), Ebola (e.g Tuncer et al. 2018), SARS (e.g WHO 2006), HIV (e.g McKusick et al. 1985), 1918 flu pandemic (e.g Bootsma & Ferguson 2007).

## MODEL

$N$  individuals in continuous time  $t \in [0, T]$  on time-varying contact network  $G_t$ .

**Compartments:**  $S, I(H), R, D$  s.t  $S_t + I_t + R_t + D_t = 1$ .

$m(A, B)$  – number of links between compartment  $A$  and  $B$  divided by  $N$ .

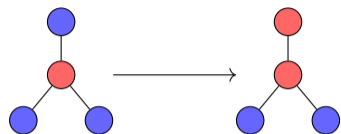
Infectious infect susceptible neighbours at rate  $\beta$ , recover at rate  $\gamma$ .

Add **rewiring** – susceptibles rewire links to infectious to another susceptible at rate  $w$  – prefer not to infect susceptible neighbours

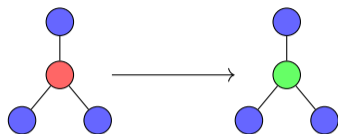
Reduced form representation of behaviour when individuals have a preference for certain level of socialisation and negative preference for socialising with the infectious/infecting others. [▶ Formal microfoundations](#)

**Vital dynamics** (for stability analysis) – natural birth/death rates  $\lambda, \mu$  link birth/death at rate  $\kappa, \nu$ .

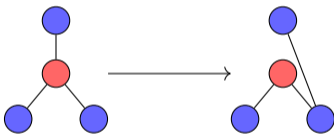
## EVENTS



(a) Infection, rate  $\beta$  per I-S link



(b) Recovery, rate  $\gamma$  per I node



(c) Rewiring, rate  $w$  per I-S link

**Figure:** Different events within our model

Mean-field dynamics of this jump process can be described with a set of ordinary differential equations (Kurz 1971) . [▶ Effect](#)

## Disease dynamics

$$\frac{dS}{dt} = -\beta m(I, S)(t)$$

$$\frac{dI}{dt} = \beta m(I, S)(t) - \gamma I(t),$$

$$\frac{dR}{dt} = \delta_r H(t) + \gamma(I(t) - H(t)),$$

$$H = \iota I(t),$$

$$\frac{dD}{dt} = \delta_d H(t).$$

## Network dynamics

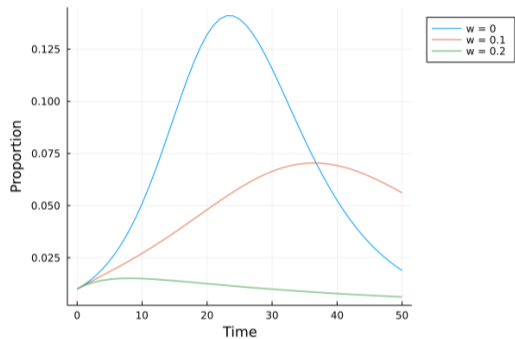
$$\frac{dm(S, S)}{dt} = w m(I, S)(t)$$

$$- 2\beta \frac{m(I, S)(t)m(S, S)(t)}{S(t)},$$

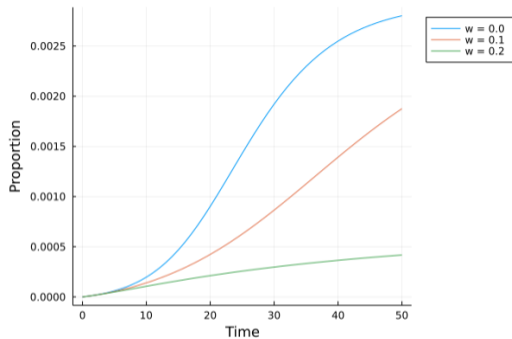
$$\frac{dm(I, S)}{dt} = \beta \frac{m(I, S)(t)}{S(t)} (2m(S, S)(t) - m(I, S)(t))$$

$$- (\gamma + w)m(I, S)(t).$$

**Figure:** Rewiring slows spread of disease and growth of deaths.



(a) Infections



(b) Deaths

**Notes:** Left panel shows paths of infections. Right panel shows time paths of deaths. Parameters are:  $\beta = 0.038$ ,  $\gamma = 0.2$ ,  $w \in \{0, 0.1, 0.2\}$ ,  $\delta^r = 0.19$ ,  $\delta^d = 0.01$ ,  $\nu = 0.076$ ,  $S(0) = 0.99$ ,  $I(0) = 0.01$ ,  $R(0) = 0.0$  and average degree  $\bar{d} = 10$ .



Can introduce **multiple groups**  $k \in \mathcal{K}$  and **group-group** lockdowns  $L_{kj} \in [0, 1]$ .

$$\frac{dS_k}{dt} = \sum_{j \in \mathcal{K}} \beta m(S_k, I_j)(t) L_{kj}(t)$$

$$\frac{dI_k}{dt} = \sum_{j \in \mathcal{K}} \beta m(S_k, I_j)(t) L_{kj}(t) - \gamma I_k(t),$$

$$\frac{dR_k}{dt} = \delta_{rk} H_k(t) + \gamma (I_k(t) - H_k(t))$$

$$\begin{aligned} \frac{dm(S_k, S_j)}{dt} &= w \frac{S_k(t)}{\sum_{k' \in \mathcal{K}} S_{k'}(t)} \sum_{k' \in \mathcal{K}} m(I_{k'}, S_k)(t) L_{k'k}(t) + w \frac{S_j(t)}{\sum_{k' \in \mathcal{K}} S_{k'}(t)} \sum_{k' \in \mathcal{K}} m(I_{k'}, S_j)(t) L_{k'j}(t) \\ &\quad - \frac{\sum_{k' \in \mathcal{K}} \beta m(I_{k'}, S_k)(t) L_{k'k}(t) m(S_k, S_j)(t)}{S_k(t)} - \frac{\sum_{k' \in \mathcal{K}} \beta m(I_{k'}, S_j)(t) L_{k'j}(t) m(S_j, S_k)(t)}{S_j(t)} \end{aligned}$$

$$\begin{aligned} \frac{dm(I_k, S_j)}{dt} &= \frac{1}{S_k(t)} \sum_{k' \in \mathcal{K}} \beta L_{k'k}(t) m(I_{k'}, S_k)(t) m(S_k, S_j)(t) \\ &\quad - \frac{1}{S_j(t)} \sum_{k' \in \mathcal{K}} \beta L_{k'j}(t) m(I_{k'}, S_j)(t) m(S_j, I_k)(t) - (\gamma + w) m(I_k, S_j)(t), \end{aligned}$$

## STABILITY ANALYSIS

Can use Jacobian of our model to study the stability of a disease-free steady state – what happens when we introduce "patient zero" given structure of connections between groups and when people rewire

### Theorem 1.

Let  $\Theta$  be a symmetric  $K \times K$  matrix with elements  $\frac{\beta L_{ij} m(S_i, S_j)}{\sqrt{S_i S_j}}$ . Then the disease-free equilibrium is stable if and only if  $\hat{\Theta} \preceq c \mathbf{I}$  where  $c = \gamma + w + \nu + 2\mu$ .

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### Corollary 2.

Set  $K = 1, L = 1, \nu + 2\mu \approx 0$ . Then

$$\mathcal{R}_0 = \frac{\beta m(S, S)}{S(\gamma + w)}.$$

**Rewiring acts similarly to recovery**, reducing number of secondary infections.

## OPTIMAL POLICY

Imagine planner trying to target lockdowns across groups to keep disease-free equilibrium stable (prevent the disease from spreading/keep  $\mathcal{R}_0 < 1$ ).

Can set up a planner's problem as a semidefinite program (Boyd & Vandenberghe 2004) given our model

$$\begin{aligned} \min_{L_{kj}} \quad & C(\mathbf{L}) \\ \text{s.t.} \quad & \hat{\Theta}(\mathbf{L}) \preceq (\gamma + w)\mathbf{I} \\ & 0 \leq L_{kj} \leq 1 \\ & k, j = 1, \dots, K. \end{aligned}$$

Increasing **rewiring rate relaxes constraint**

As constraint is  $K \times K$ , can solve problem of which connections to lock down between many groups to lock down with standard software (3098 groups solves in approx 3-4 days in with Convex.jl and SCS)

## OPTIMAL POLICY

Set  $C(\mathbf{L}) = -\sum_{k,j} L_{kj} s_k$  – planner want to minimise total amount of individuals locked down.

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Can incorporate trade-offs from limited disease spread up to finite time (e.g up to vaccination roll-outs Acemoglu et al. 2021) by considering relaxations of stability of disease free equilibrium

$$\hat{\Theta}(\mathbf{L}) \preceq (\gamma + w)\mathbf{I} + \epsilon \implies \mathcal{R}_0 \leq 1 + \delta$$

Gives Pareto frontier of possible points policymaker can achieve given preferences without solving complicated non-convex optimisation problem as in Acemoglu et al. (2021).

Can also incorporate targeting different types of connections/ layers in the contact network (e.g school closures)

$$C(\mathbf{L}) = -\sum_{l \in \mathcal{L}} \sum_{k,j} L_{kj}^l s_k.$$

## ESTIMATION

Issue – how can we estimate rewiring rate when network dynamics are unobserved?

### Theorem 3.

Denote aggregate number of new cases  $C_t$ . Let the initial conditions be given by  $[S_k(0), I_k(0), R_k(0), m(S_{k'}, S_k)(0), m(I_{k'}, S_k)(0)]$  for  $k, k' \in \mathcal{K}$ . Then our system of ODEs defines a bijective mapping  $\theta \rightarrow \mathbb{R}_+^3$ :

$$(\beta, \gamma, w) \mapsto \left\{ \sum_{k \in \mathcal{K}} S_k(t), \sum_{k \in \mathcal{K}} I_k(t), C(t) \right\}_{t=0}^T,$$

and the parameters  $(\beta, \gamma, w) \in \theta$  are point identified given the initial conditions.

**Structural estimator:**  $\hat{\theta} = \arg\text{-max}(X_t - X(t, \theta))'W(X_t - X(t, \theta))$

$$X_t = \left[ \sum_{k \in \mathcal{K}} S_{kt}, \sum_{k \in \mathcal{K}} I_{kt}, C_t \right], \text{ and } X(t, \theta) = \left[ \sum_{k \in \mathcal{K}} S(t), \sum_{k \in \mathcal{K}} I(t), \beta \sum_{k, j \in \mathcal{K}} m(I_k, S_j)(t) \right]$$

## EMPIRICAL EXAMPLE

Study interplay of network-based targeting and endogeneity for optimal policy by estimating parameters and then solving different control problems.

Consider:

1. how targeting based on network structure between fine-grained groups compares to uniform lockdowns
2. how network endogeneity changes optimal targeted lockdowns
3. what are the trade-offs between deaths and economic damages when a planner targets based on the network, and how rewiring affects these trade offs.

**Setting** – second wave of Covid-19 in the Netherlands from 6th of August 2020 to 19th of November 2020, using data from National Institute of Public Health and Environment (RIVM).



## NETWORK DATA

Construct a **population-scale social network** from Statistics Netherlands registry data (van der Laan 2022, Bokányi et al. 2023)

17.6 million nodes, five **layers** of connections: household, family, work, school, neighbourhood.

Reweight links by types of contact in POLYMOD contact surveys for flu-like infectious diseases to build population-level contact network.

Aggregate spatially by municipality/wijken to form 380/3098 groups – gives system of 289,940/19,204,502 non-linear differential equations.



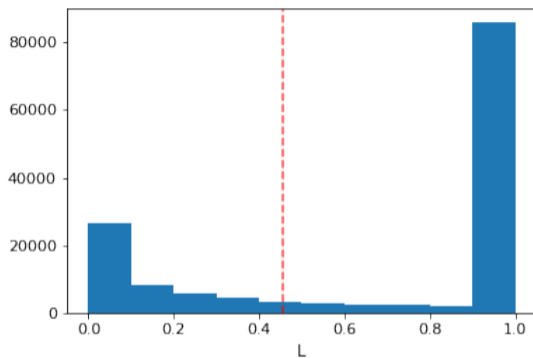
**Figure:** Contacts per individual per day for flu-like infectious diseases between municipalities in the Netherlands. Darker weights indicate more contacts. Connections containing more than 12000 links omitted for ease of reading, and to preserve privacy of individuals in small municipalities.

**Table:** Estimates of infection, recovery, and rewiring rates, second wave of Covid-19.

		With rewiring	Without rewiring
		(1)	(2)
Infection rate	$(\beta)$	0.0296*** (0.00579)	0.0207*** (0.000645)
Recovery rate	$(\gamma)$	0.191*** (0.00986)	0.174*** (0.00748)
Rewiring rate	$(w)$	0.0917* (0.0617)	

**Notes:** Covariance matrix estimated using standard GMM sandwich estimator with forward-mode autodifferentiation. Standard errors in parentheses. Significance levels: \*  $p < 0.10$ , \*\*  $p < 0.05$ , \*\*\*  $p < 0.01$ .

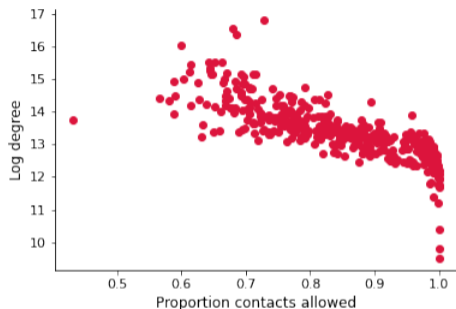
## OPTIMAL LOCKDOWNS



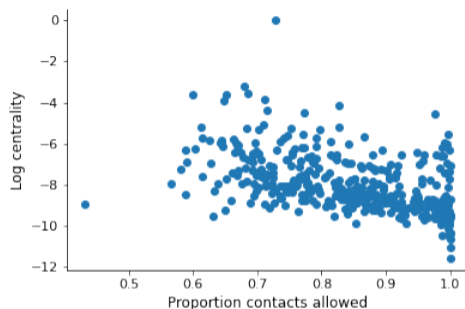
**Figure:** Targeted lockdowns by municipality-municipality pair for Covid-19 in the Netherlands with estimated parameters. The dashed line gives the optimal uniform lockdown level.

Targeting based on network increases efficiency – mean proportion contacts allowed 0.834 vs 0.454 without.

## OPTIMAL LOCKDOWNS



(a) Municipality degree



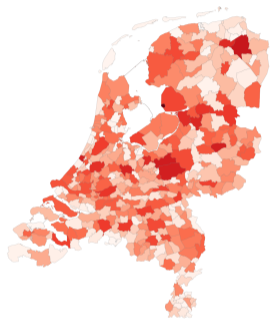
(b) Municipality eigenvector centrality

**Figure:** Relationship between municipality log degree/centrality and optimal lockdown levels.

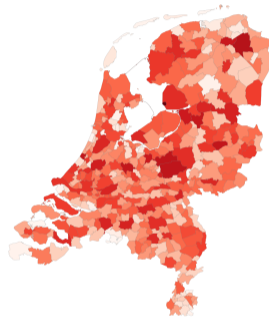
Allow fewer contacts the more connected/central (correlations  $-0.789, -0.478$ )

## OPTIMAL TARGETED LOCKDOWNS

(a) Proportion contacts allowed, rewiring.



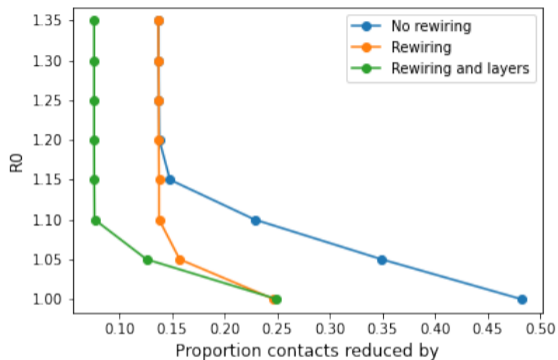
(b) Proportion contacts allowed, no rewiring.



**Figure:** Lightest color denotes the most contacts allowed and darkest denotes the fewest contacts allowed.

Rewiring reduces lockdown intensity (mean 7.78% more contacts with rewiring than without), mostly in moderately connected municipalities.

## PARETO FRONTIER



**Figure:** Pareto frontier representing trade-offs between epidemic spread and lockdown intensities faced by policymakers. Points represent relaxations  $\epsilon \in \{0, 0.05, \dots, 0.35\}$ .

Rewiring, and more fine-grained targeting allow planner to relax constraint much more efficiently than without rewiring.

## CONCLUSION

Introduce a model of spread of disease on a network when individuals adjust contacts in response to spread of disease.

Show how to use the model to estimate the rate at which individuals adjust contacts and optimal targeted lockdowns even as the number of groups become large.

Use the model to study how endogenous behaviour and heterogeneous contact structure interact to shape optimal policy in the context of Covid-19 in the Netherlands, constructing a population-scale contact network for flu-like infectious diseases from registry data to do so.



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Readjustment possibilities arrive at rate  $\rho$  per link (Blume 1993).

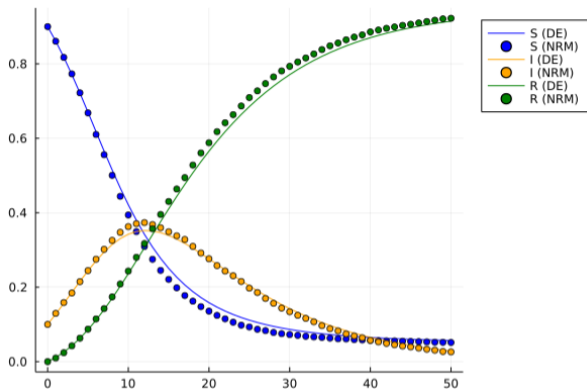
$\pi_S > 0$  denotes socialization utility,  $\eta > 0$  gives infection cost.

$$\pi_0 = \pi_S - \beta\eta + \varepsilon_0,$$

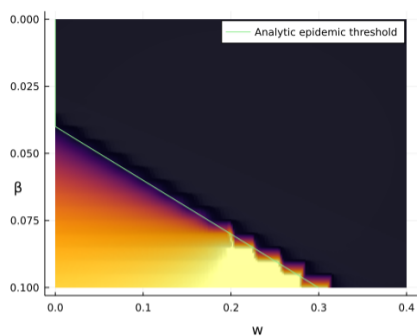
$$\pi_1 = \pi_S - c + \varepsilon_1.$$

Rewire if  $\pi_1 > \pi_0$ .

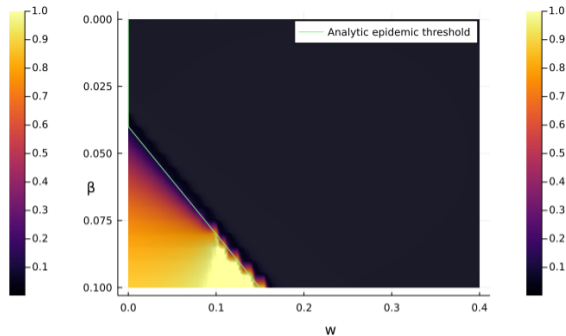
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**Figure:** Comparison of mean-field ODEs for the epidemic dynamics mean jump-process dynamics. DE denotes paths from system of ODEs. NRM denotes means from Monte-Carlo simulation of jump process using the next-reaction method.



(a)



(b)

**Figure:** The color gradient denotes the proportion of the population who have been infected with the disease at  $T = 100,000$  for a given pair  $\beta, w$  and fixed  $\gamma$ . Fixed parameters are:  $S(0) = 0.999$ ,  $I(0) = 0.001$ ,  $R(0) = 0$ ,  $q = 0$ ,  $v = 0$ ,  $T = 100,000$ . In the left panel, we set  $\bar{d} = 5$ , and  $\gamma = 0.2$ . In the right panel, we set  $\bar{d} = 2.5$ , and  $\gamma = 0.1$ . We use a grid  $\beta$  from 0 to 0.1 at intervals of 0.005, and a grid  $w$  from 0 to 0.4 at intervals of 0.005.