Approximation of stochastic models for epidemics on large multi-level graphs

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Motivation and goals

Epidemic spread depends on the underlying contact network $\rightarrow$ why focus on small contact structures (households, workplaces...)?

- **Clustering** affects epidemic outcomes, and the way clustering is achieved within the network matters (Volz et al., 2011)
- Some **control measures** (teleworking, school closures. . .) target specific types of contacts (Mendez-Brito et al., 2021)
- How contacts are included in the model changes predicted outcomes of control measures (Di Lauro et al., 2021)

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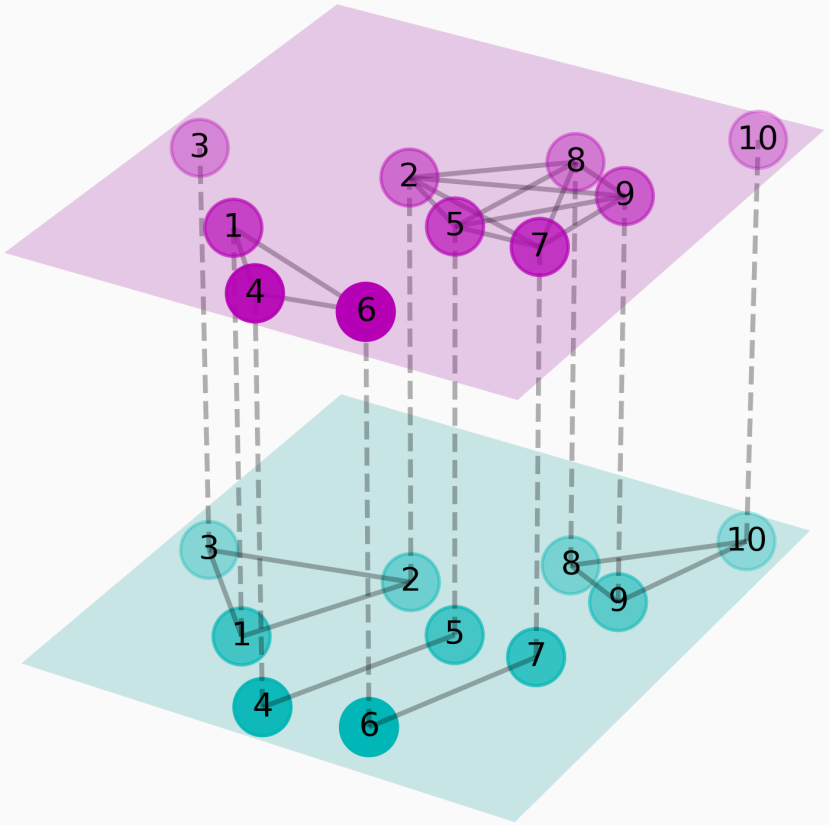
⇒ **Household-workplace model** (Ball and Neal, 2002; Pellis et al., 2009):

- **Multiscale** model (2 levels of mixing: global and local)
- Main mathematical difficulty: local level with **several contact structures**.

The household-workplace model

Local level of mixing $\rightarrow$ households and workplaces:

- Structure size distributions π^H and π^W, maximal size $n_{\max} < \infty$.
- Each individual is attributed to a household and workplace independently from one another and from other individuals.

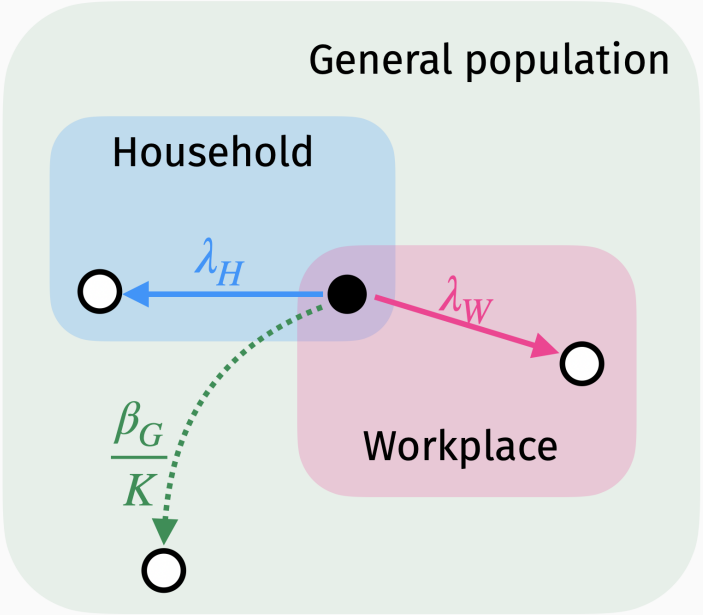


The household-workplace model

Modified SIR model $\rightarrow$ three ways of contamination in a population of size K:

- **General population**: total of S susceptible and I infected individuals $\rightarrow$ infections at rate $\frac{\beta_G}{K} SI$.
- **Within households or workplaces**: s susceptible and i infected members $\rightarrow$ infections at rate $\lambda_X si$ for $X \in \{H, W\}$.

Distribution ν of the duration of infectious periods.



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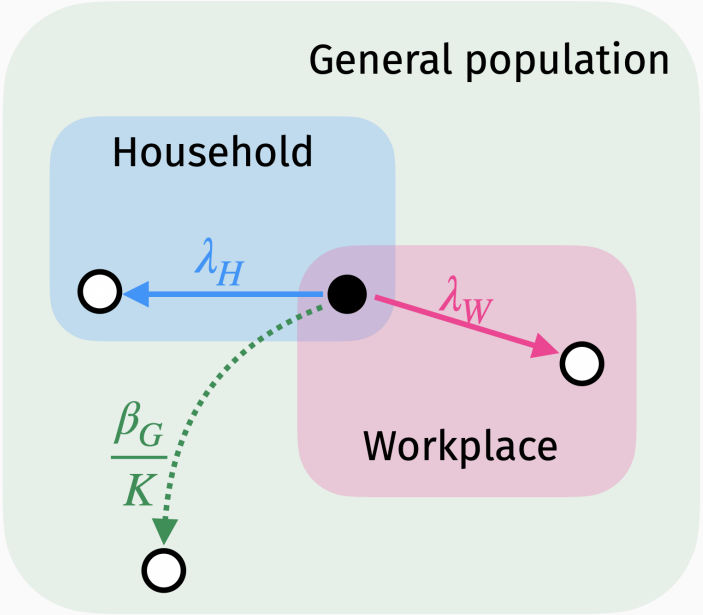


$\Rightarrow$ **Stochastic model of parameters**

$$(K, \underbrace{\pi^H, \pi^W}_{\text{social structure}}, \underbrace{\lambda_H, \lambda_W, \beta_G, \nu}_{\text{epidemic}}).$$

Talk outline

(I) Numerical exploration

- Impact of structure size distributions: teleworking strategies.
- Parsimonious model reduction.

« The epidemiological footprint of contact structures in models with two levels of mixing », V. Bansaye, F. Deslandes, M. Kubasch, E. Vergu (2023+)

(II) Large population limit

- Individual based model converges to deterministic limit.
- Asymptotically exact epidemic dynamics.

« Large population limit for a multilayer SIR model including households and workplaces », M. Kubasch (2023+)

(III) Sensitivity analysis

- Quantify model parameter impact on epidemic model outputs.
- Relax contact network assumptions.

Numerical exploration

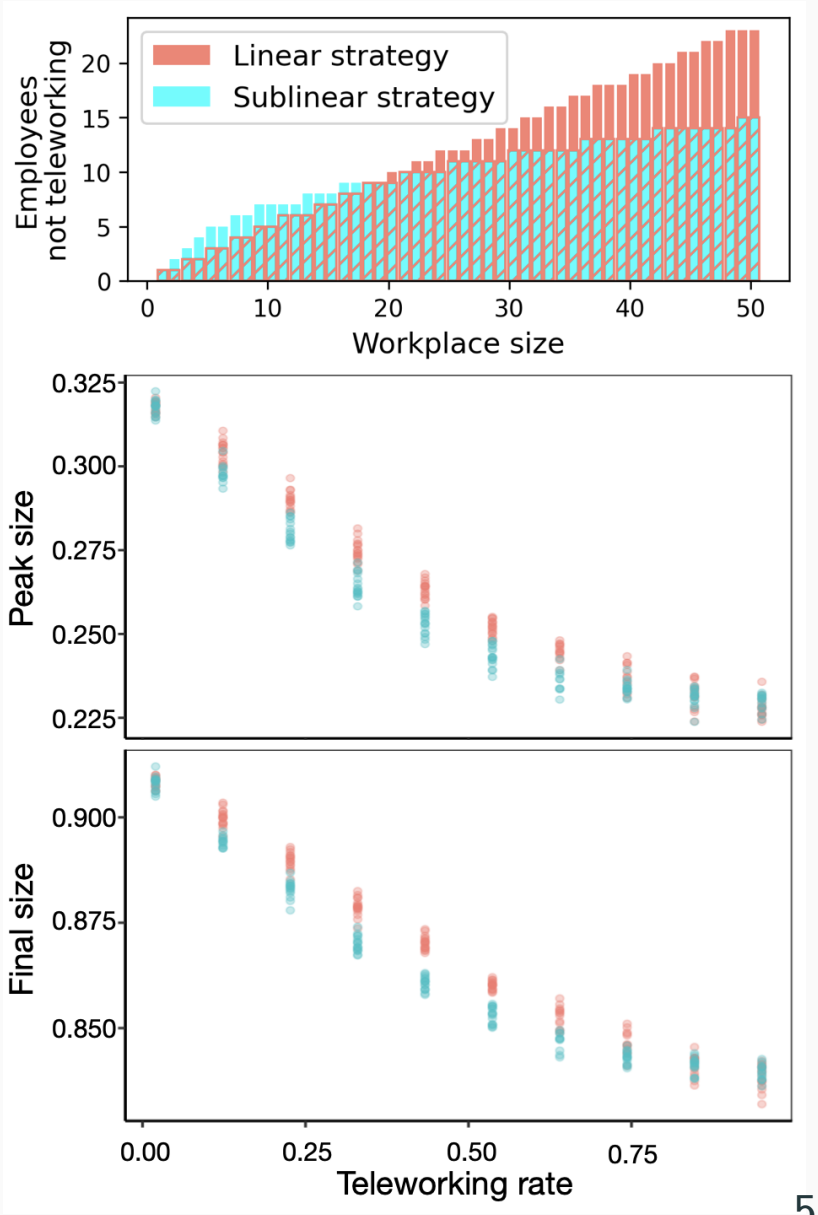
Comparison of teleworking strategies

Teleworking strategies: for a workplace of size k, the number of employees not teleworking proportional to

- $k \to$ **linear strategy**;
- $k^{\frac{1}{2}} \to$ **sublinear strategy**.

Simulations: COVID19-like setting, French structure size distributions.

$\Rightarrow$ **Better performance of the sublinear strategy.**



A parsimonious reduced model

Approximation by a **uniformly mixing SIR model**:

$$\begin{cases} S' = -\beta SI \\ I' = \beta SI - \gamma I \\ R' = \gamma I. \end{cases}$$

$\Rightarrow$ How to fit the parameters?

- Removal rate γ usually known (epidemiological expertise).
- Calibrate β using the exponential growth rate (Pellis et al., 2011), i.e. $\beta = r + \gamma$.

A parsimonious reduced model

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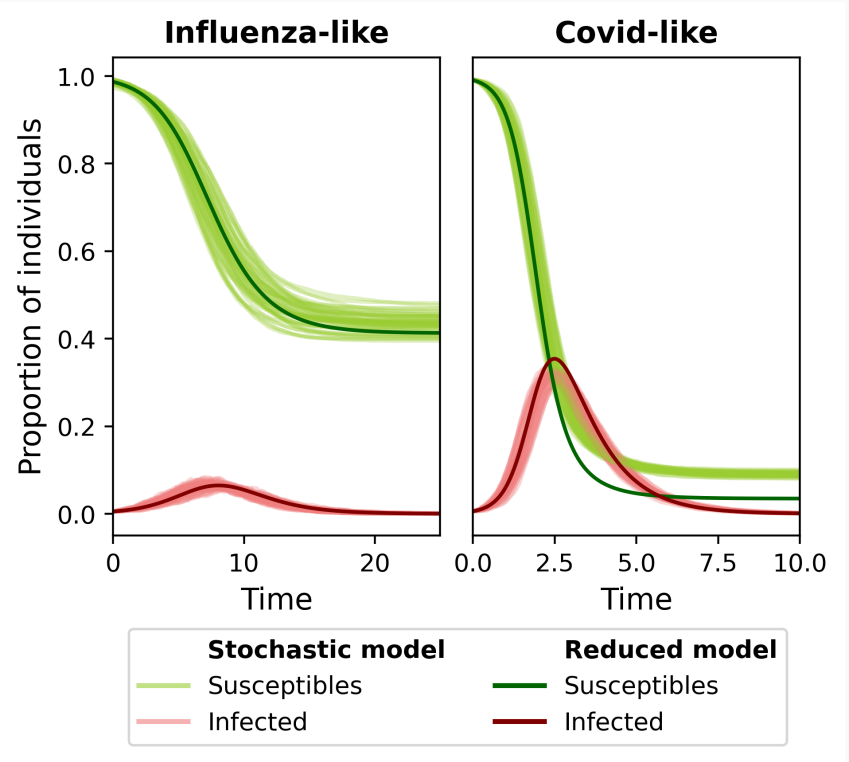
A parsimonious reduced model

Numerical evaluation:

- Satisfying results on epidemic peak and final size (error generally $< 5\%$).
- Growth rate: key parameter.
- Accuracy affected by epidemic intensity and proportions of infections per layer.

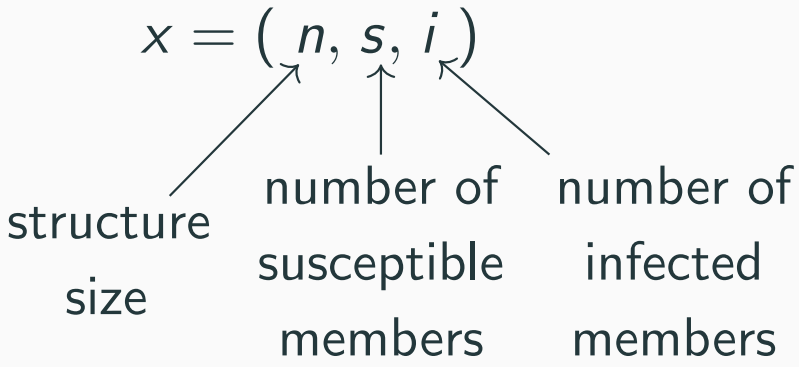


Room for improvement: Precision decreases over time.
No theoretical guarantees.

Large population limit

Introducing the structure types

Reduced models suggested in similar settings (House and Keeling, 2008; Volz et al., 2011) $\rightarrow$ epidemic at the level of structures characterised by a type x:



Difficulties:

- Infected individuals correlate the epidemic states of their household and workplace.
- Explore the random graph.

Introducing the structure types

$\Rightarrow$ **Solution:** keep track of each infected's remaining infectious period (similar in spirit to Ball, Sirl, et al., 2014).

$$x = (\, n,\, s,\, \tau \,)$$

- n: structure size
- s: number of susceptible members
- τ: remaining infectious periods

where $\tau = (\underbrace{\tau_1, \ldots, \tau_{n-s}}, \underbrace{0, \ldots, 0}) \in \mathbb{R}^{n_{\max}}$.

$\tau_k > 0 \rightarrow$ infectious at time t, else already recovered.

Null by default.

Introducing the structure types

Types evolve over time:

- Continuous decay of remaining infectious periods.
- Infection event: $(n, s, \tau) \to (n, s-1, \tau + \sigma e_{n-s+1})$ with σ sampled from ν and $(e_k)_{k \leq n_{\max}}$ the canonical basis of $\mathbb{R}^{n_{\max}}$.

$\Rightarrow$ Progressive discovery of the contact network:

- Upon an infection event, uncover the household and workplace of the newly infected.
- Update both structures' types using the same realization of σ.

The agent-based model for finite populations

Sequence $(\mathbf{G}^K)_{K\geq 1}$ of random contact networks for finite populations of size $K \geq 1$:

- The household and workplace of each individual are chosen independently from one another, and from other individuals.
- Almost sure convergence of the finite population structure size distributions to π^H and π^W.

$\Rightarrow$ The epidemic process depends on the sampled contact network: convergence result holds for almost every realization of $(\mathbf{G}^K)_{K\geq 1}$.

The agent-based model for finite populations

Sequence of realizations of the random contact network $\rightarrow$ population of size K with:

- K_H households $\rightarrow$ types $x_1^H(t), \ldots, x_{K_H}^H(t)$.
- K_W workplaces $\rightarrow$ types $x_1^W(t), \ldots, x_{K_W}^W(t)$.

Process of interest: associated normalised counting measure $\zeta^K = (\zeta^{H|K}, \zeta^{W|K})$, *i.e.* for $X \in \{H, W\}$ and $t \geq 0$,

$$\zeta_t^{X|K} = \frac{1}{K_X} \sum_{k=1}^{K_X} \delta_{x_k^X(t)}.$$

$\Rightarrow$ unique strong solution of a Poisson-driven SDE (Fournier and Méléard, 2004).

The agent-based model for finite populations

$\zeta^K = (\zeta^{H|K}, \zeta^{W|K})$ is the unique strong solution of the following equation: for $X \in \{H, W\}$,

$$\zeta_T^{X|K} = \frac{1}{K_X}\Big(\sum_{j=1}^{K_X} \delta_{\Psi(x_j^X(0),T,0)} + \sum_{Y\in\{G,H,W\}} \int_0^T \int_{U_Y} \mathcal{I}_Y(t-,u)\Delta_X(u,T,t)Q_Y(dt,du)\Big).$$

- $\Psi(x, T, t)$: deterministic flow of remaining infectious periods.
- Q_Y: Poisson Point process responsible for infection events.
- $\mathcal{I}_Y$: infection rate in layer Y.
- Δ_X: impact of the current infection.

Large population convergence

Theorem 1. Assume $\zeta_0^K \Rightarrow \eta_0 \in \mathfrak{M}_1$ (+ some technical condition). Then $(\zeta^K)_{K\geq 1}$ converges in law in $\mathbb{D}(\mathbb{R}_+, M_{P,1}(E))^2$ to $\eta = (\eta^H, \eta^W)$ defined as the unique solution of the following system of equations.
For any $X \in \{H, W\}$, $f \in \mathcal{C}_b^1(\mathbb{R}_+ \times E, \mathbb{R})$ and $T \geq 0$,

$$\langle \eta_T^X, f_T\rangle = \langle \eta_0^X, f_0\rangle + \int_0^T \langle \eta_t^X, \mathcal{A}f_t\rangle dt + \lambda_X \int_0^T \langle \eta_t^X, \mathbf{si}(f_t^{\mathcal{I}} - f_t)\rangle dt$$
$$+ \lambda_{\overline{X}} \int_0^T \frac{\langle \eta_t^{\overline{X}}, \mathbf{si}\rangle}{\langle \eta_t^{\overline{X}}, \mathbf{s}\rangle} \langle \eta_t^X, \mathbf{s}(f_t^{\mathcal{I}} - f_t)\rangle dt + \beta_G \int_0^T \frac{\langle \eta_t^H, \mathbf{i}\rangle}{\langle \eta_0^H, \mathbf{n}\rangle} \langle \eta_t^X, \mathbf{s}(f_t^{\mathcal{I}} - f_t)\rangle dt,$$

where

- $\mathbf{s}(x)$ = number of susceptibles in type x ($\mathbf{n}$ = size, $\mathbf{i}$ = infected).
- $f_t^{\mathcal{I}}(x) = \langle \nu, f_t(\mathrm{j}(x, \cdot))\rangle$ and $\mathcal{A}f_t(x) = \partial_t f(t,x) - \sum_{k=1}^{n-s} \partial_{\tau_k} f(t,x)$.

Large population convergence

Elements of proof: Tightness - Identification - Uniqueness

- State space E:

$$\{(n, s, \tau) \in [\![1, n_{\max}]\!] \times [\![0, n_{\max}]\!] \times \mathbb{R}^{n_{\max}} : s \leq n;\ \forall j > n - s, \tau_j = 0\}\,.$$

- Related to age-structured models (Wang, 1975; Tran, 2006).

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1. Tightness of $(\zeta^K)_{K\geq 1}$ in $\mathbb{D}(\mathbb{R}_+, (\mathcal{M}_F(E), w))^2 \to$ Main ingredients (Tran, 2014; Jourdain et al., 2012):
   - Tightness of $(\langle \zeta_\bullet^{X|K}, f\rangle)_{K\geq 1}$ for f in a large enough set, including $f = 1$.
   - Support of the mass of $\zeta^{X|K}$ must not escape to infinity over finite time intervals.
   - Limiting values $\in \mathcal{C}([0, T], (\mathcal{M}_F(E), w))^2$.

Large population convergence

Elements of proof: Tightness - Identification - Uniqueness

1. Tightness of $(\zeta^K)_{K\geq 1}$ in $\mathbb{D}(\mathbb{R}_+, (\mathcal{M}_F(E), w))^2$.
2. Identification: all limiting values are solution to the desired measure-valued equation.
   - Semimartingale decomposition of $\langle \zeta_T^{X|K}, f_T \rangle$.
   - Martingale part: quadratic variation $O(1/K)$ in expectation: vanishes as $K \to \infty$.
   - Bounded variation part $\Rightarrow$ limiting equation
     Convergence of $\langle \zeta_T^{X|K}, f \rangle$ for some discontinuous functions, e.g. $f(n, s, \tau) = \sum_{k=1}^{n-s} \mathbf{1}_{\{\tau_k > 0\}}$? $\to$ asymptotic absolute continuity.
3. Uniqueness of the solutions to the limiting equation.

Large population convergence

Remarks:

- Not limited to the Markovian case: ν is any absolutely continuous probability measure on $\mathbb{R}_+$.
- Associated to a system of non-linear, non-local transport equations.
- Rich limiting object: detailed information on infectious periods.
- Computational drawback: infinite dimension.

$\Rightarrow$ Finite-dimensional reduction based on a coarser population description ?

Finite-dimensional reduction

Let • $\nu = Exp(\gamma)$,

• $\eta_0 = \eta_{0,\varepsilon} =$ at time 0, remaining infectious periods of infected individuals are exponentially distributed.

$\Rightarrow$ Finite-dimensional reduction: dynamical system with variables

- s, i: proportion of susceptibles / infected in the population;
- $n^X_{(S,I)}$: proportion of structures of type X containing S susceptibles and I infected, for (S, I) such that $S + I \leq n_{\max}$, and $S \geq 2$ or $SI \geq 1$.

Finite-dimensional reduction

Theorem 2. Let $\varepsilon > 0$. Suppose that $\nu = Exp(\gamma)$, and that $\zeta_0^K \Rightarrow \eta_{0,\varepsilon}$. Then the functions of interest are characterized as the unique solution of:

$$\frac{d}{dt}s(t) = -(\tau_H(t) + \tau_W(t) + \beta_G i(t)s(t)),$$
$$\frac{d}{dt}i(t) = -\frac{d}{dt}s(t) - \gamma i(t),$$
$$\begin{aligned}\frac{d}{dt}n^X_{(S,I)}(t) = &-\left(\lambda_X SI + \tau_{\overline{X}}(t)\frac{S}{s(t)} + \beta_G i(t)S + \gamma I\right) n^X_{(S,I)}(t)\\ &+ \gamma(I+1)n^X_{(S,I+1)}(t)\mathbf{1}_{\{S+I<n_{\max}\}}\\ &+ \left(\lambda_X(S+1)(I-1) + \tau_{\overline{X}}(t)\frac{S+1}{s(t)} + \beta_G i(t)(S+1)\right) n^X_{(S+1,I-1)}(t)\mathbf{1}_{\{I\geq 1\}},\end{aligned}$$

where for $X \in \{H, W\}$ and $m_X = \sum_{k=1}^{n_{\max}} k\pi_k^X$,

$$\tau_X(t) = \frac{\lambda_X}{m_X}\sum_{(S,I)\in\mathbb{S}} SI\, n^X_{(S,I)}(t).$$

Finite-dimensional reduction

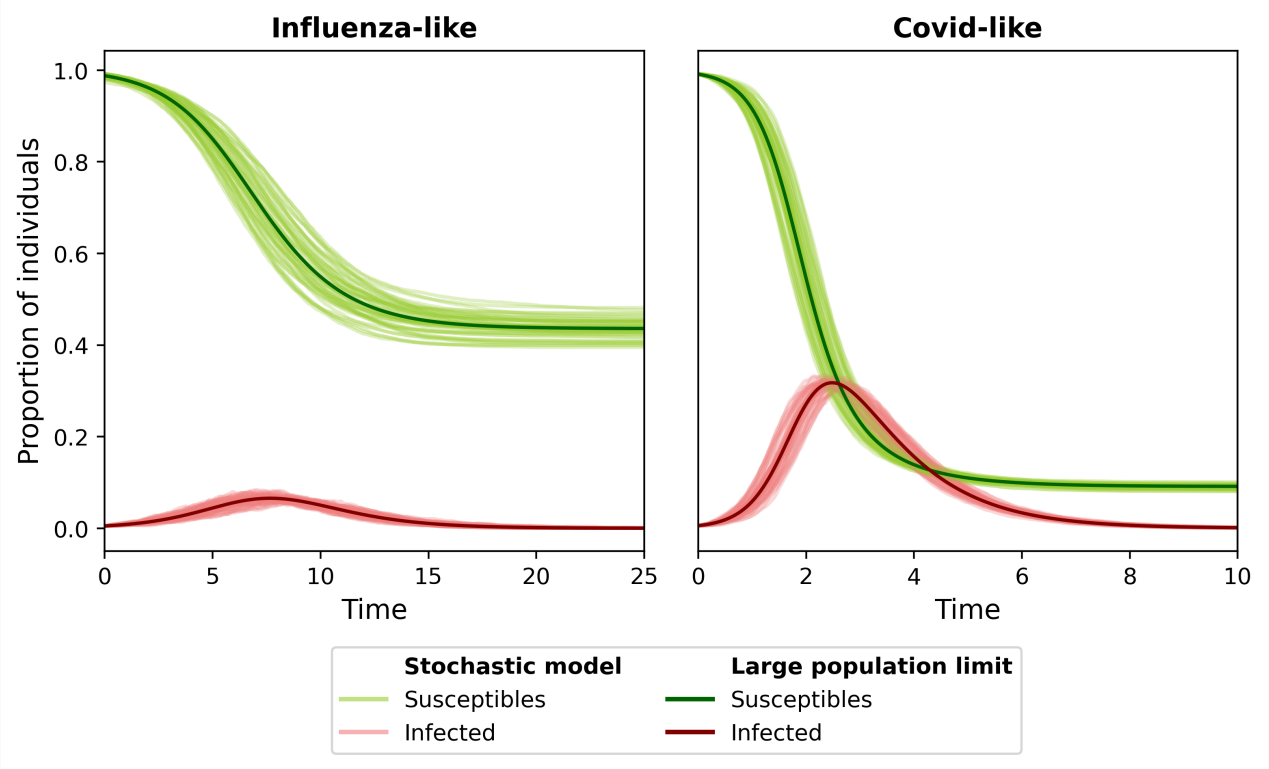
Elements of proof: integrating η^X over appropriate domains of the state space to recover the dynamics of s, i, $n^X_{(S,I)}$, for example:

$$i(t) = \frac{1}{n_H} \langle \eta_t^H, \sum_{k=1}^{\mathbf{n}(\cdot)-\mathbf{s}(\cdot)} \mathbf{1}_{\{\tau_k(\cdot)>0\}} \rangle.$$

Relies on the memory-less property of the exponential distribution $\rightarrow$ at each time, remaining infectious periods of infected belonging to the same structure are *i.i.d.* $Exp(\gamma)$.

Finite-dimensional reduction

Comparison to stochastic simulations (SSA) in a large population:



Computational cost: dynamical system pertinent for numerical explorations, despite its large dimension.

Sensitivity to epidemic parameters and contact network

Global sensitivity analysis

Quantify the impact of the model parameters on epidemic outcomes $\Rightarrow$ **global sensitivity analysis** using Sobol's decomposition of the variance.

General idea:

- Model parameters sampled independently from distributions $\rightarrow$ model outputs: random variables.
- **Main effect**: part of the output variance explained directly by one given parameter.
- **Total effect**: part of the output variance explained by a given parameter and its interaction with other parameters.

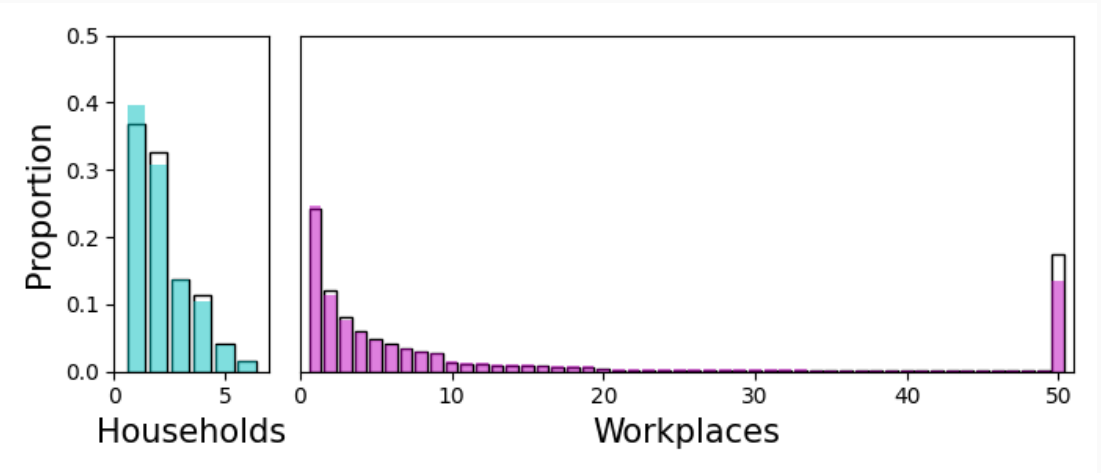
$\rightsquigarrow$ Experiment design?

Global sensitivity analysis

Structure size distributions:

- Perturbation of French household (X=H) and workplace (X=W) size distributions π^X_{Fr}.
- Mixture with beta-binomial distributions $b_{m,v}$ of mean m and variance v: $\pi^X = p_X \pi^X_{Fr} + (1 - p_X) b_{m,v}$
- **Sample independently** average m_X and variance v_X of π^X.

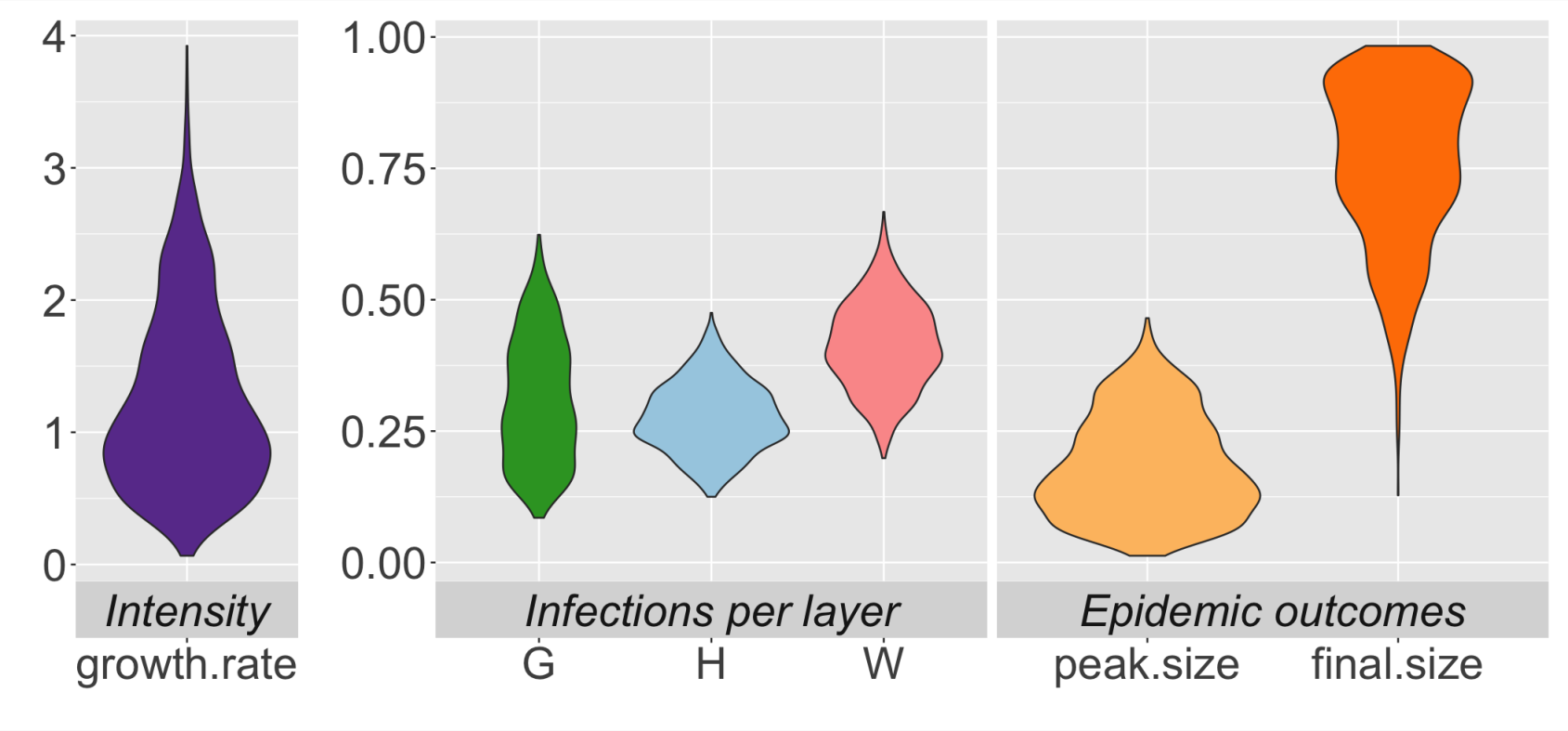
	$X = H$	$X = W$
p_X	$(0.75, 0.8)$	$(0.75, 0.8)$
m_X	$(2.13, 2.26)$	$(13.06, 16.05)$
v_X	$(1.53, 1.98)$	$(290, 339)$



Global sensitivity analysis

Epidemic parameters:

- Shifted β-distributions.
- **Relevant range of epidemic scenarios**.



Global sensitivity analysis

Results:

- Strong influence of β_G, with little interactions.
- Small fluctuations of size distributions: no impact.

Robustness to network variations

Our **contact network** relies on **simplifying assumptions**:

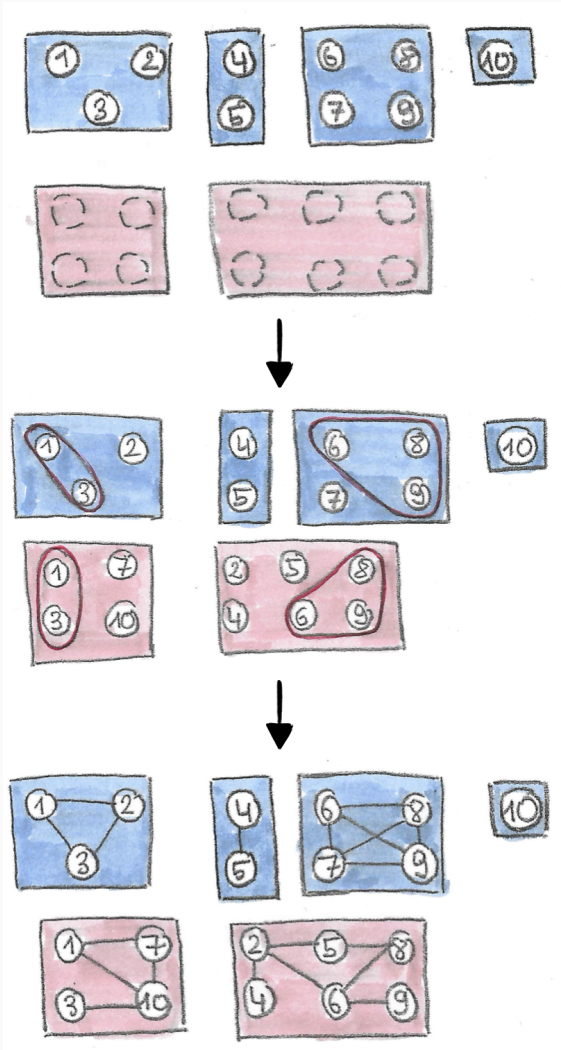
- Little overlap between households and workplaces?
  - → Life partners sharing a workplace (Wilson, 2015).
- Uniform mixing within structures?
  - → Average number of contacts per time unit does not grow linearly with structure size (Cauchemez et al., 2004).
  - → Workplaces likely not uniformly mixing (Contreras et al., 2022; Timpka et al., 2016).

⇒ What happens if these assumptions are relaxed?

Robustness to network variations

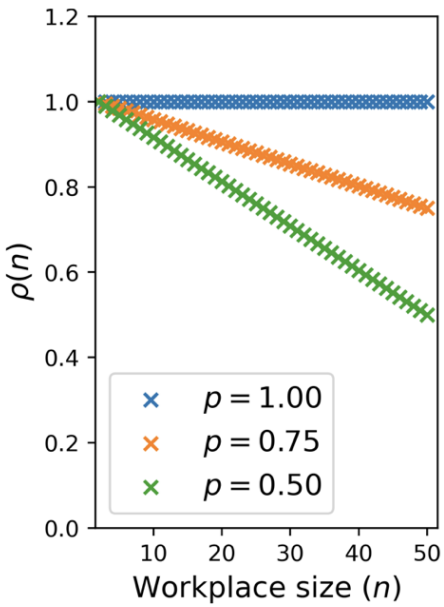
Generalized household-workplace model:

1. **Structure overlap:** Household of size n $\rightarrow \mathcal{B}(n, q)$ members work together.
2. **Workplace contact density:** Workplace of size $n \rightarrow$ Erdös-Rényi $G(n, \rho(n))$, where ρ is non-increasing.

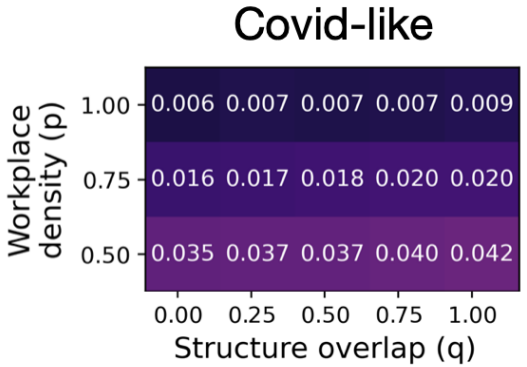


Robustness to network variations

Comparison of the generalized model and the large population limit of the household-workplace model.



Peak size



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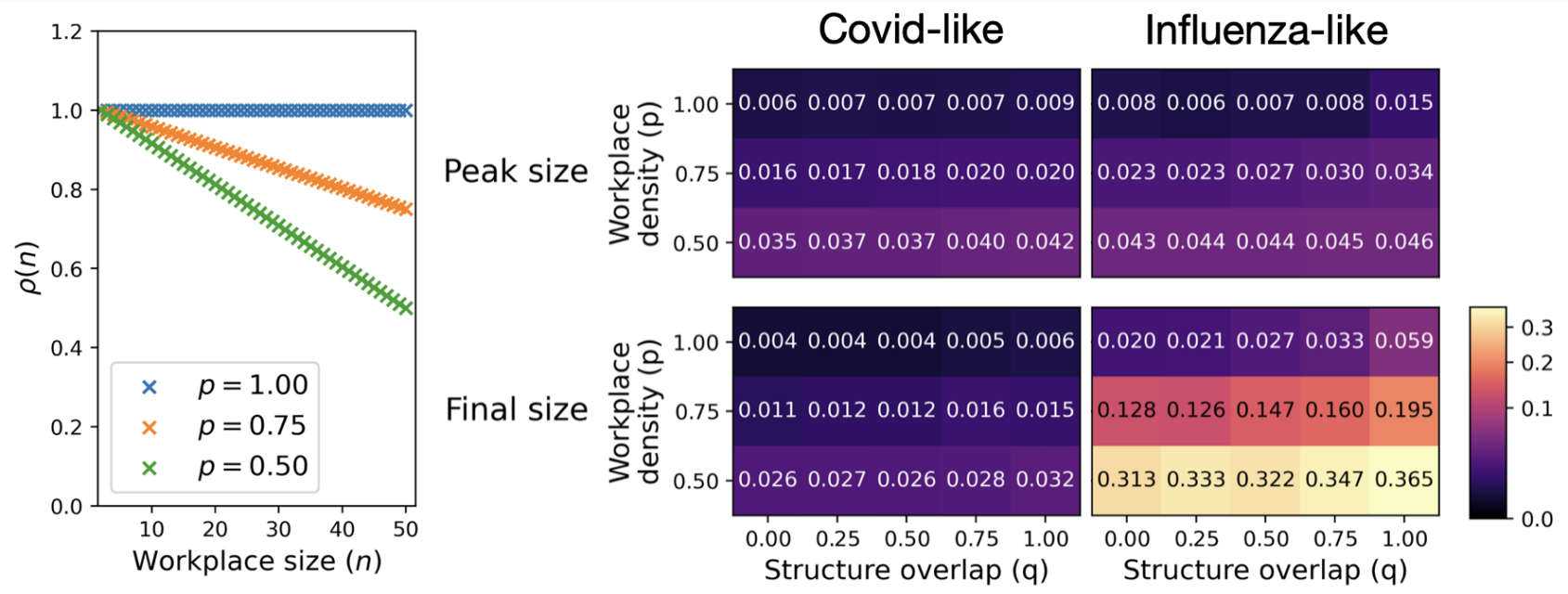

- Covid: higher growth rate, less local infections than influenza.
- Good approximation in most settings (error $\leq 5\%$).
- Influence of within-workplace density $p >$ structure overlap q.

Conclusion

Complements and perspectives

(I) Numerical exploration

- Impact of structure size distributions: teleworking strategies.
- Parsimonious model reduction.
- Control measures?

(II) Large population limit

- Individual based model converges to deterministic limit.
- Asymptotically exact epidemic dynamics.
- Gaussian fluctuations?

(III) Sensitivity analysis

- Quantify model parameter impact on epidemic model outputs.
- Relax contact network assumptions.
- Large perturbations of structure size distributions?
- Further investigation of within-structure networks?

(IV) Contamination chains

- Spinal constructions for density-dependent population processes.
- Application: contamination chains at endemic equilibrium?

Thank you for your attention

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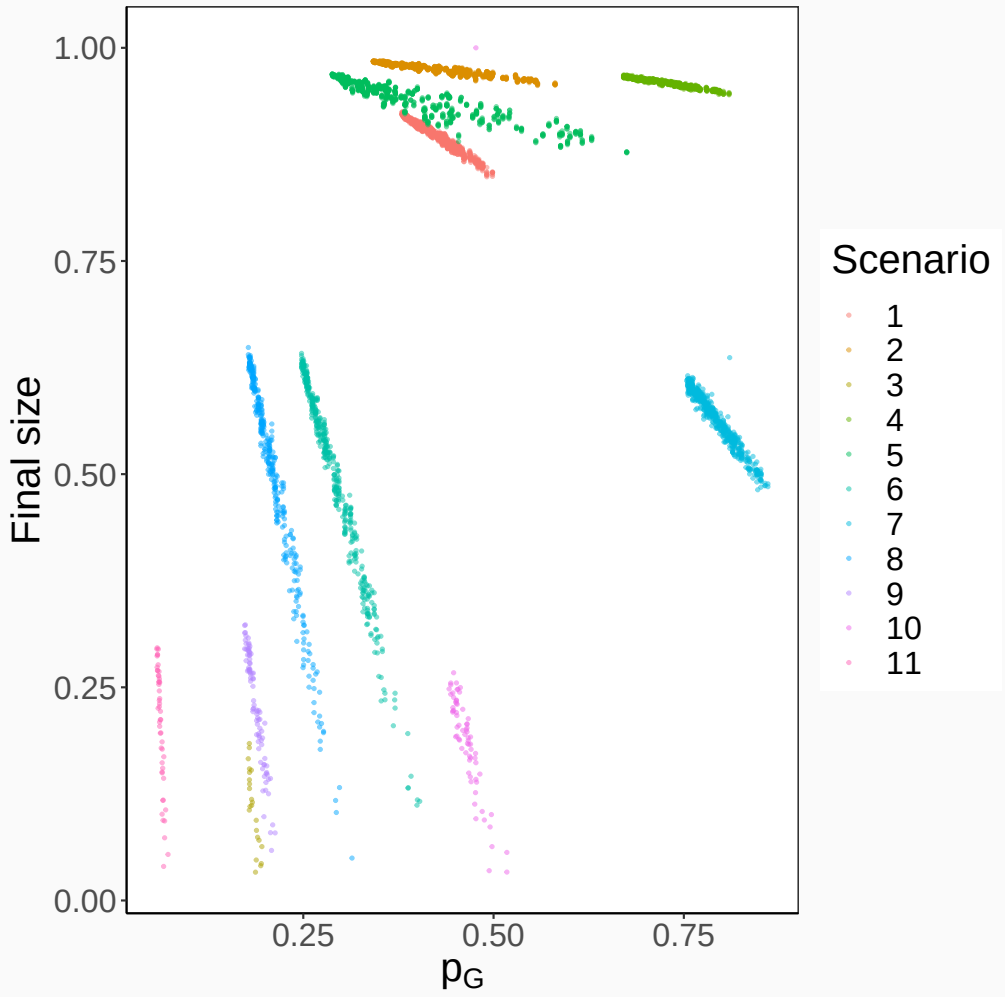
Variety of epidemic outcomes

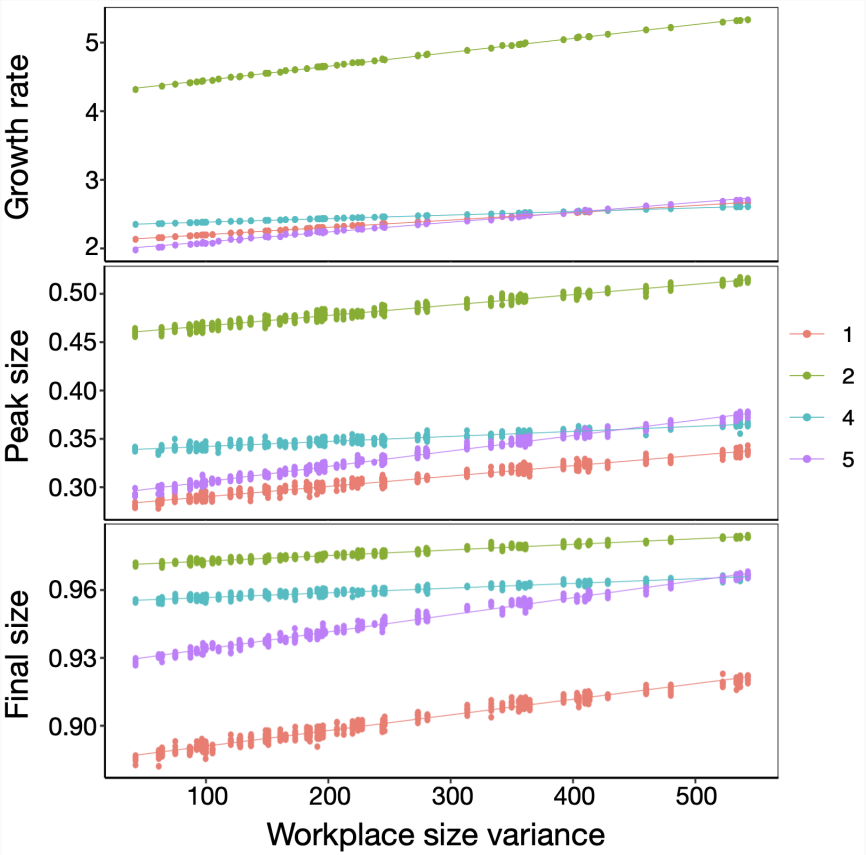
Scenarios for French size distributions:

	Intensity	Infections
1	+	
2	+++	
3	-	
4	+++	G
5	+++	W
6	+	
7	+	G
8	+	
9	-	H+W
10	-	
11	-	H+W



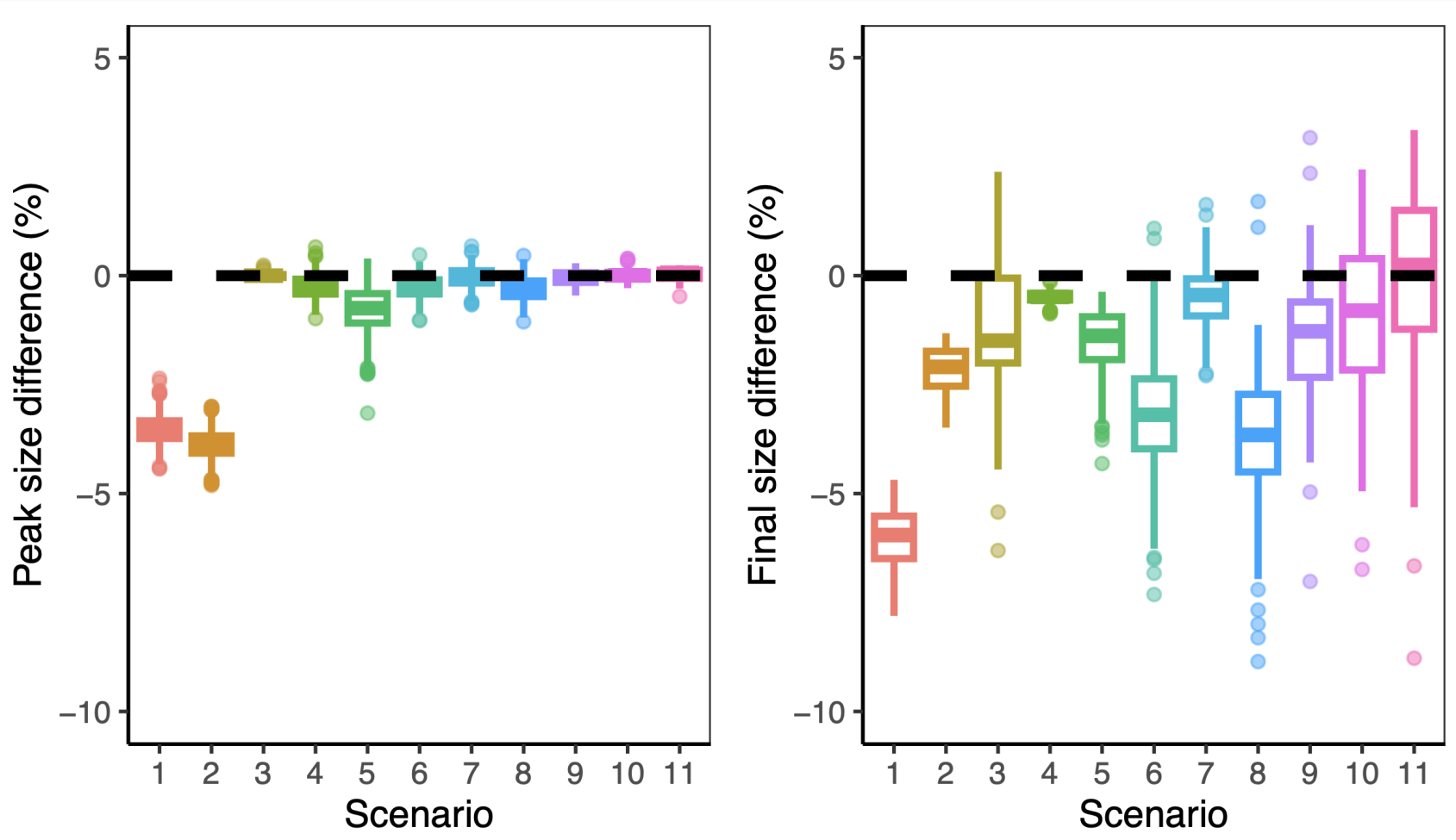
Epidemic impact at fixed average structure size

- Workplace size distributions with fixed average workplace size.
- Linear correlation of key epidemic characteristics and the size distribution variance: good proxy.



Approximation using a uniformly mixing SIR model

Comparison of simulation outputs and reduced model predictions:



Assumptions on $(\zeta_0^K)_{K\geq 1}$

- Same total number of infected within households and workplaces, etc.
- For any $X \in \{H, W\}$ and $T \geq 0$, suppose that:
  1. $$\lim_{N\to\infty} \sup_{K\geq 1} \mathbb{E}\left[\sup_{0\leq t\leq T} \frac{1}{K_X} \sum_{k=1}^{K_X} \sum_{i=1}^{n_{\max}} \mathbf{1}_{\left\{n_k^X - s_k^X(0)\geq i,\ |\tau_{k,i}^X(0)-t|\geq N\right\}}\right] = 0.$$
  2. For any $c \in \mathbb{R}$, for any $i \in [\![1, n_{\max}]\!]$,
  $$\lim_{\epsilon\to 0} \sup_{K\geq 1} \mathbb{E}\left[\frac{1}{K_X} \sum_{k=1}^{K_X} \mathbf{1}_{\left\{n_k^X - s_k^X(0)\geq i,\ |(\tau_{k,i}^X(0)-T)-c|\leq \epsilon\right\}}\right] = 0.$$

Associated PDE system

$$\partial_t \rho_{X,n,s}(t,\tau) - \sum_{k=1}^{n-s} \partial_{\tau_k} \rho_{X,n,s}(t,\tau) = -s(\lambda_X i(\tau) + \Lambda_X(t))\rho_{X,n,s}(t,\tau)$$

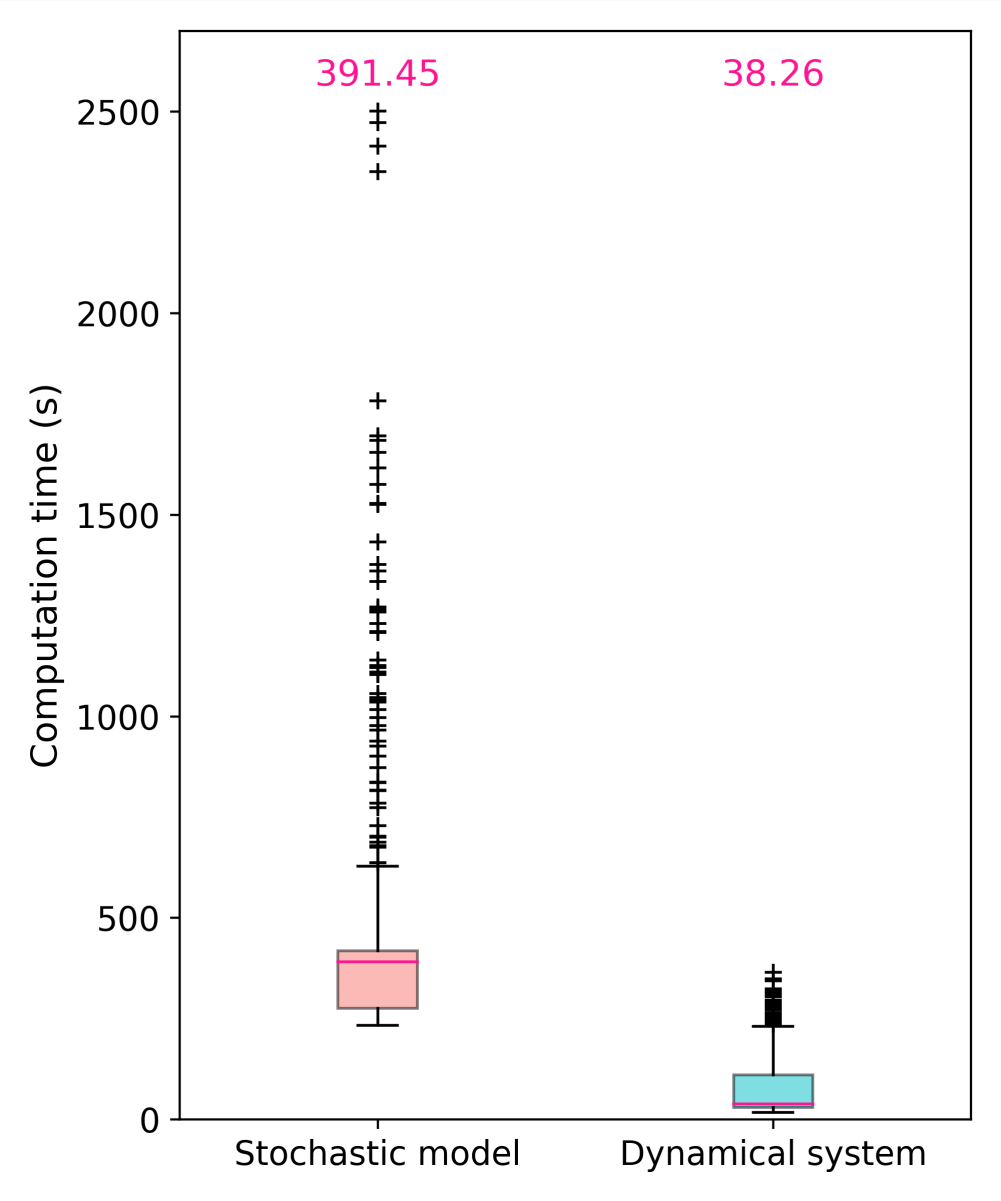
$$+\mathbf{1}_{\{s+1\leq n\}}(s+1)\left(\lambda_X i(\tau_{1,n-s-1}) + \Lambda_X(t)\right)\rho_{X,n,s+1}(t,\tau_{1,n-s-1})g_\nu(\tau_{n-s})$$

where $\Lambda_X(t) = \frac{\lambda_{\overline{X}}}{s_{\overline{X}}(t)} \sum_{n=1}^{n_{\max}} \sum_{s=0}^{n-1} \int_{\mathbb{R}^{n-s}} si(\tau)\rho_{\overline{X},n,s}(t,\tau)d\tau + \beta_G \frac{i_H(t)}{n_H}$,

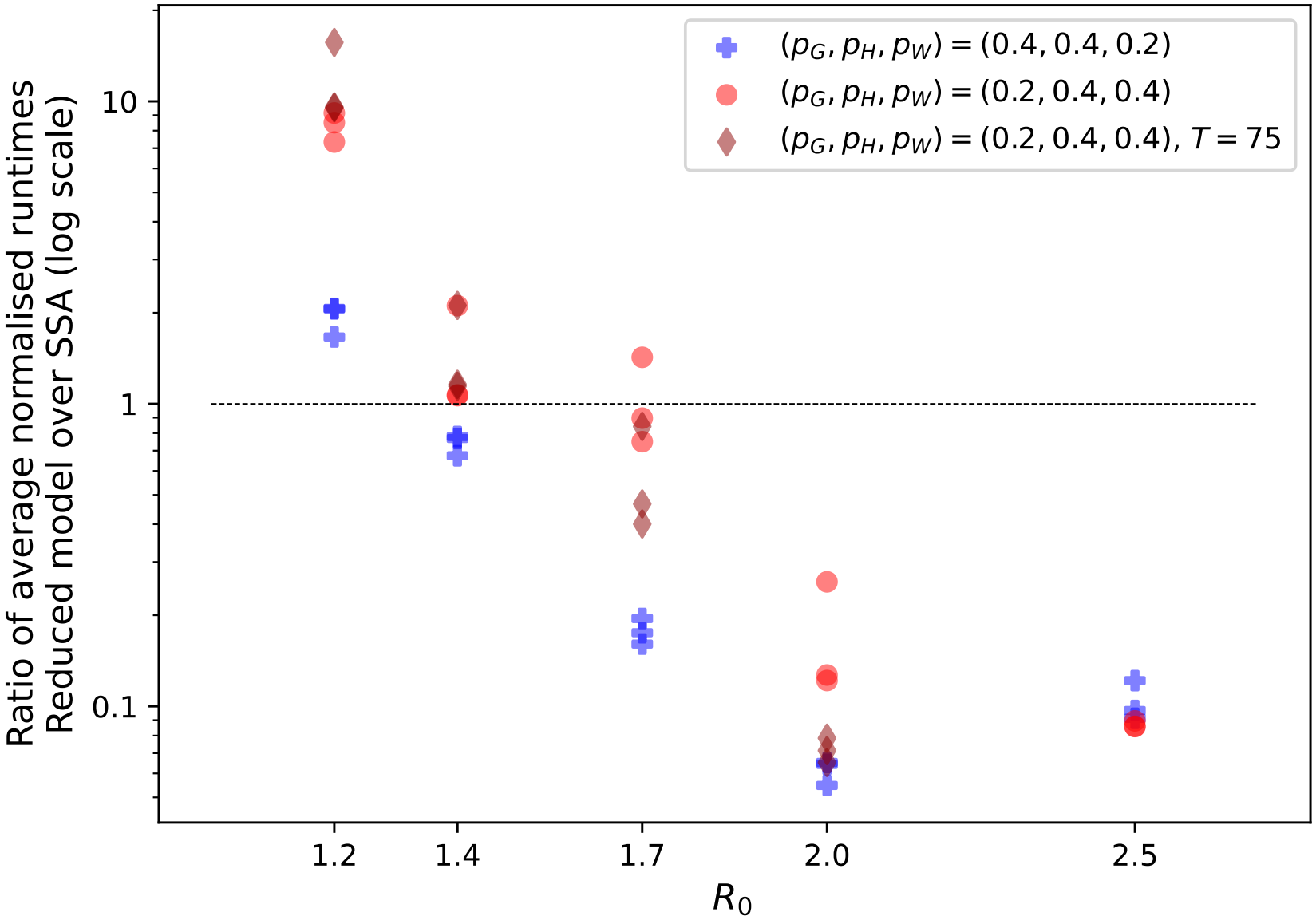
and $s_X(t) = \sum_{n=1}^{n_{\max}} \sum_{s=1}^{n} \|\rho_{X,n,s}\|_{L^1}$,

$$i_X(t) = \sum_{n=1}^{n_{\max}} \sum_{s=0}^{n-1} \int_{\mathbb{R}^{n-s}} i(\tau)\rho_{\overline{X},n,s}(t,\tau)d\tau.$$

Computational performance



Computational performance



Sensitivity analysis: reduction performance