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Topics in epidemic spreading modeling

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Dissertation framing

- **Focus:**
  - **Influenza-like Illnesses**

    (airborne transmission, short infection cycle & short-term immunity).

  - **Vaccine hesitancy** through (age & contact) structured models .

  - **Spatial epidemics & mobility** through the metapopulation approach.

- **Methods:** Data-driven & computational mechanistic models.

- **Aims:** To bring insights into theoretical & real situations to

  i) Enhance understanding of propagation mechanisms

  ii) Assist public health policy-making.

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5) Closure.

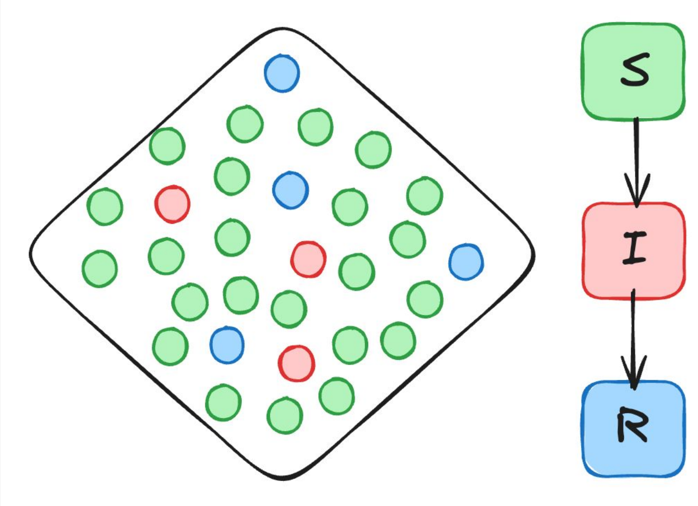
Framework

Elements of epidemic modeling: SIR model

Compartmental model

Single population

Well-mixing assumption



$$\mathcal{S} + \mathcal{I} \xrightarrow{\beta} \mathcal{I} + \mathcal{I},$$

$$\mathcal{I} \xrightarrow{\mu} \mathcal{R}.$$

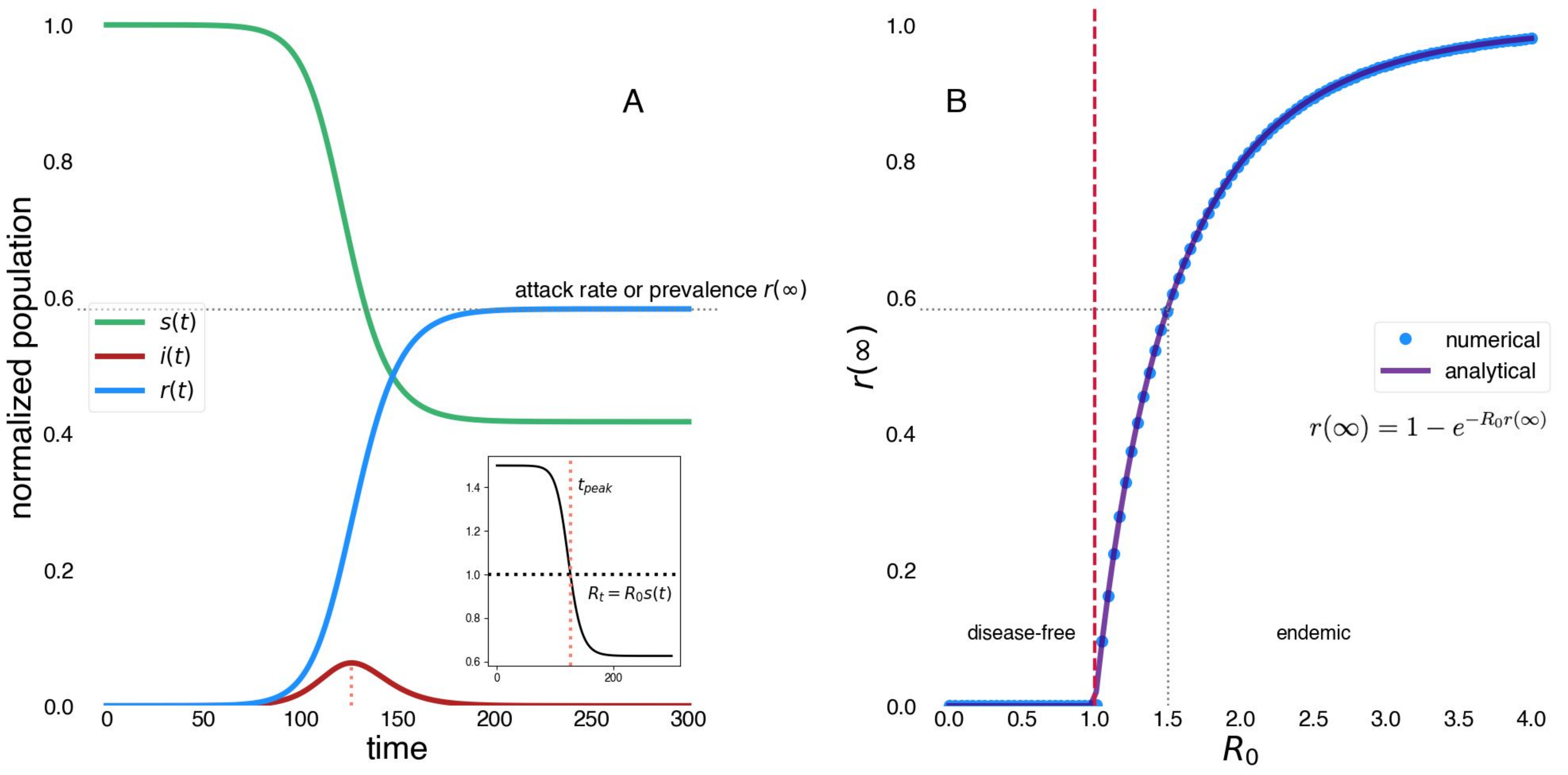
$$R_0 = \frac{\beta}{\mu}\langle k \rangle$$

$$\frac{ds(t)}{dt} = -\beta\langle k\rangle s(t)i(t),$$

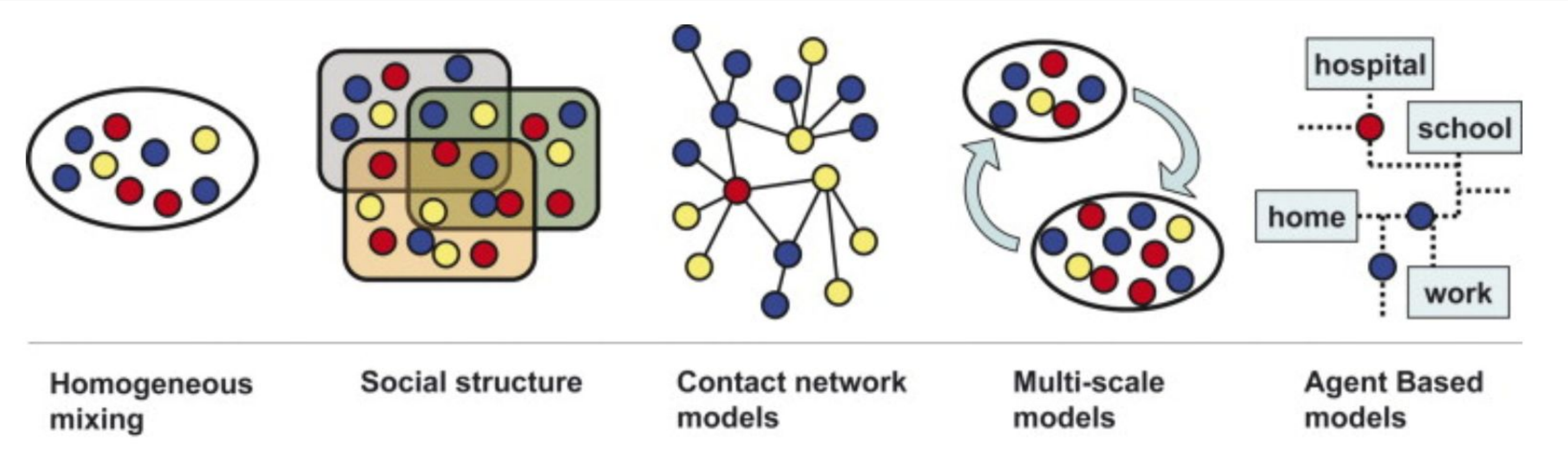
$$\frac{di(t)}{dt} = \beta\langle k\rangle s(t)i(t) - \mu i(t),$$

$$\frac{dr(t)}{dt} = \mu i(t).$$

SIR model main results



Extending the classical approach



Colizza et al. (2007): Epidemic modeling in complex realities

We have shifted **from homogeneous single-populations to heterogeneous structured systems**. This has been possible thanks to ongoing research efforts accompanied by increasing data availability and computational power.

Part I. Behavioral structured models

Work #1

de Miguel-Arribas *et al.*
BMC Infectious Diseases (2022) 22:511
https://doi.org/10.1186/s12879-022-07486-0

BMC Infectious Diseases

RESEARCH Open Access

Impact of vaccine hesitancy on secondary COVID-19 outbreaks in the US: an age-structured SIR model

Alfonso de Miguel-Arribas[1,2], Alberto Aleta[3*] and Yamir Moreno[1,2,3]

Abstract

Background: The COVID-19 outbreak has become the worst pandemic in at least a century. To fight this disease, a global effort led to the development of several vaccines at an unprecedented rate. There have been, however, several logistic issues with its deployment, from their production and transport, to the hesitancy of the population to be vaccinated. For different reasons, an important amount of individuals is reluctant to get the vaccine, something that hinders our ability to control and—eventually—eradicate the disease.

Materials and methods: Our aim is to explore the impact of vaccine hesitancy when highly transmissible SARS-CoV-2 variants of concern spread through a partially vaccinated population. To do so, we use age-stratified data from surveys on vaccination acceptance, together with age-contact matrices to inform an age-structured SIR model set in the US.

Results: Our results show that per every one percent decrease in vaccine hesitancy up to 45 deaths per million inhabitants could be averted. A closer inspection of the stratified infection rates also reveals the important role played by the youngest groups. The model captures the general trends of the Delta wave spreading in the US (July-October 2021) with a correlation coefficient of $\rho = 0.79$.

Conclusions: Our results shed light on the role that hesitancy plays on COVID-19 mortality and highlight the importance of increasing vaccine uptake in the population, specially among the eldest age groups.

Keywords: COVID-19, Mathematical modeling, Age-structured SIR, Vaccination, Hesitancy

Impact of vaccine hesitancy on secondary COVID-19 outbreaks in the US

A. de Miguel-Arribas, A. Aleta & Y. Moreno

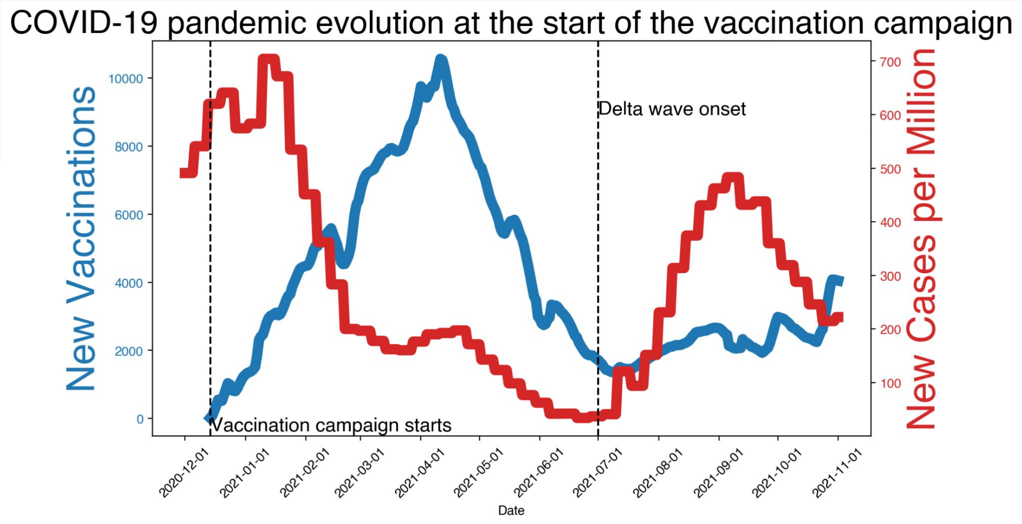
Background: Vaccine hesitancy

Vaccines arrived in late 2020 but…

Appendix A: Vaccine attitudes by state

If you were able to choose when to get a COVID-19 vaccine, would you get it…

State	Already vaccinated	As soon as possible	After at least some people I know	After most people I know	Would not get the COVID-19 vaccine	Error Margin	N
National	15	33	15	16	21	1	21459
AK	29	19	16	14	23	7	277
AL	13	30	13	20	24	6	375
AR	13	25	13	19	31	5	388
AZ	15	28	18	13	27	6	389
CA	11	32	24	18	15	5	507



- Social media & surveys showed growing concerns on vaccines safety & effectiveness
- This could be problematic to epidemic control if going back to normal…

Question [?]

Given the hesitancy levels in the US around COVID-19 vaccines, what epidemic impact can be expected in secondary outbreaks?

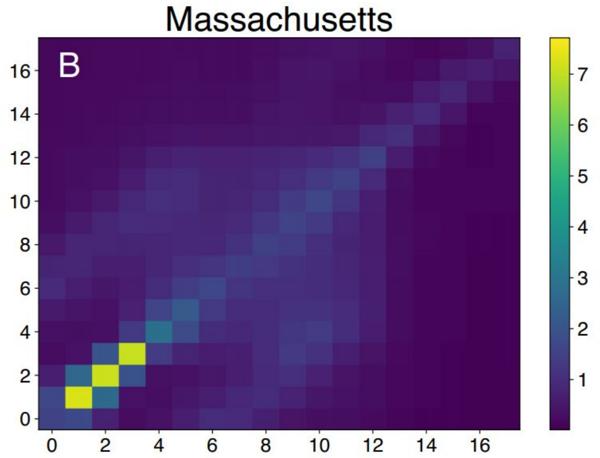
Age-structured SIR

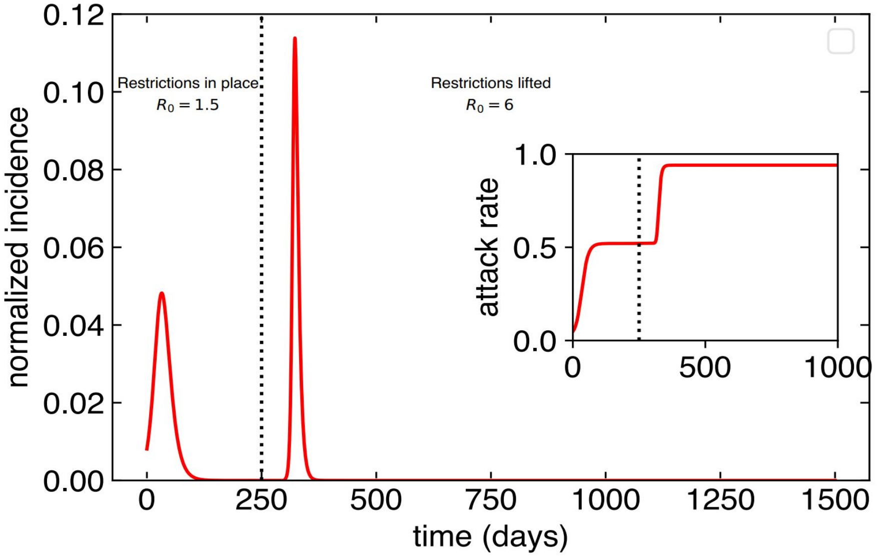
- SIR model + vaccination campaign.
- **Age-structure** feed with high-resolution contact matrices: 85 age groups (Mistry et al. 2020).
- **Vaccination campaign** feed with hesitancy data from all the states in the USA (Lazer et al. 2020).
- Underage not vaccinated.
- Proposed scenario:
  - Wave 1: Epidemic + vaccination
  - Wave 2: Unmitigated

$$\frac{dS_a}{dt} = -\lambda_a S_a,$$
$$\frac{dI_a}{dt} = \lambda_a S_a - \gamma I_a,$$
$$\frac{dR_a}{dt} = \gamma I_a,$$

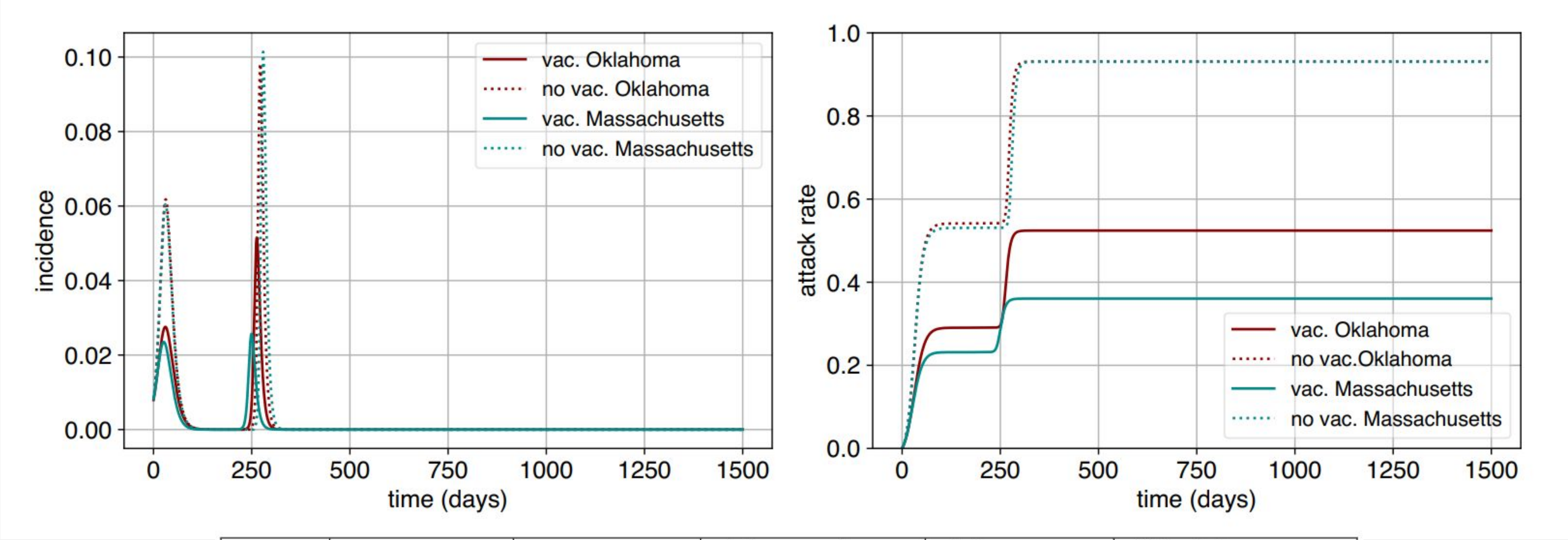
$$\lambda_a = \beta \chi_a \sum_{a'} M_{aa'} \frac{I_{a'}}{N_{a'}},$$

$$R_0 = \frac{\beta}{\gamma} \rho(\chi M),$$



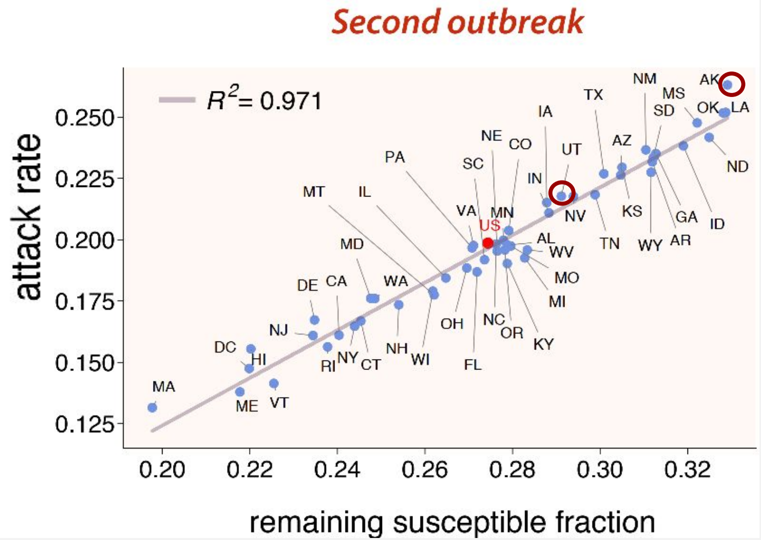


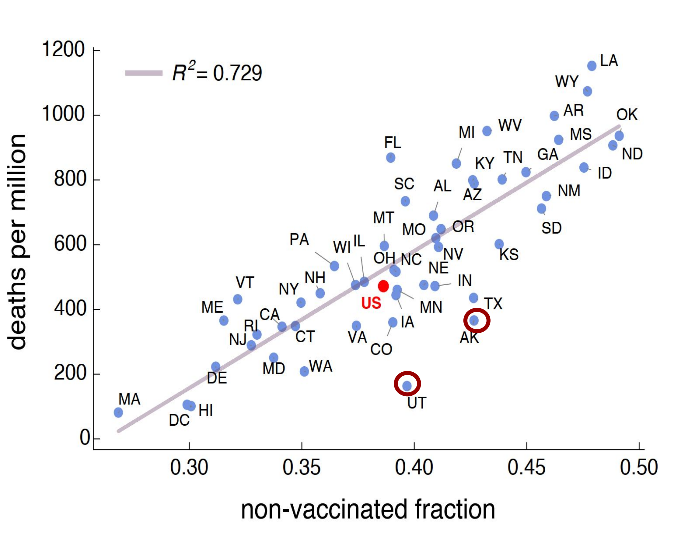
Results: Epidemic evolution in disparate states



State	Already vaccinated	As soon as possible	After at least some people I know	After most people I know	Would not get the COVID-19 vaccine
MA	13	47	18	13	9
OK	15	27	8	17	33

Results: Prevalence, deaths and hesitancy





- As expected, vaccine **hesitancy exacerbates the epidemic impact but**... Subtleties arise.

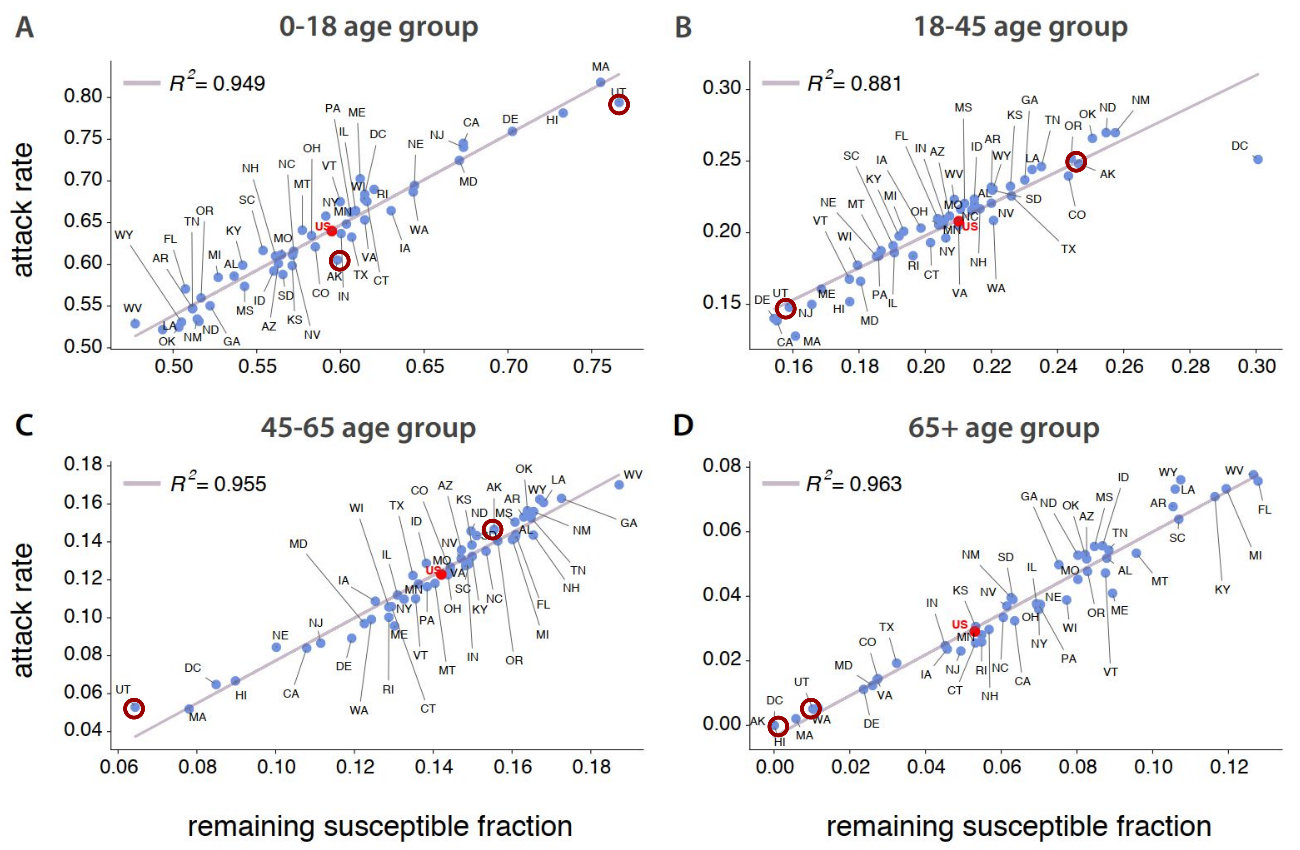
- AK highest attack in the 2nd wave, highest ‘RSF’ but ~23% hesitant fraction.
- UT, also high impact but ~15% hesitant fraction.
- But then, AK and UT have a rather low number of deaths per million.
- **Age structure** is **also** playing a **key role** in contagion dynamics and fatalities.

Results: Impact by age groups

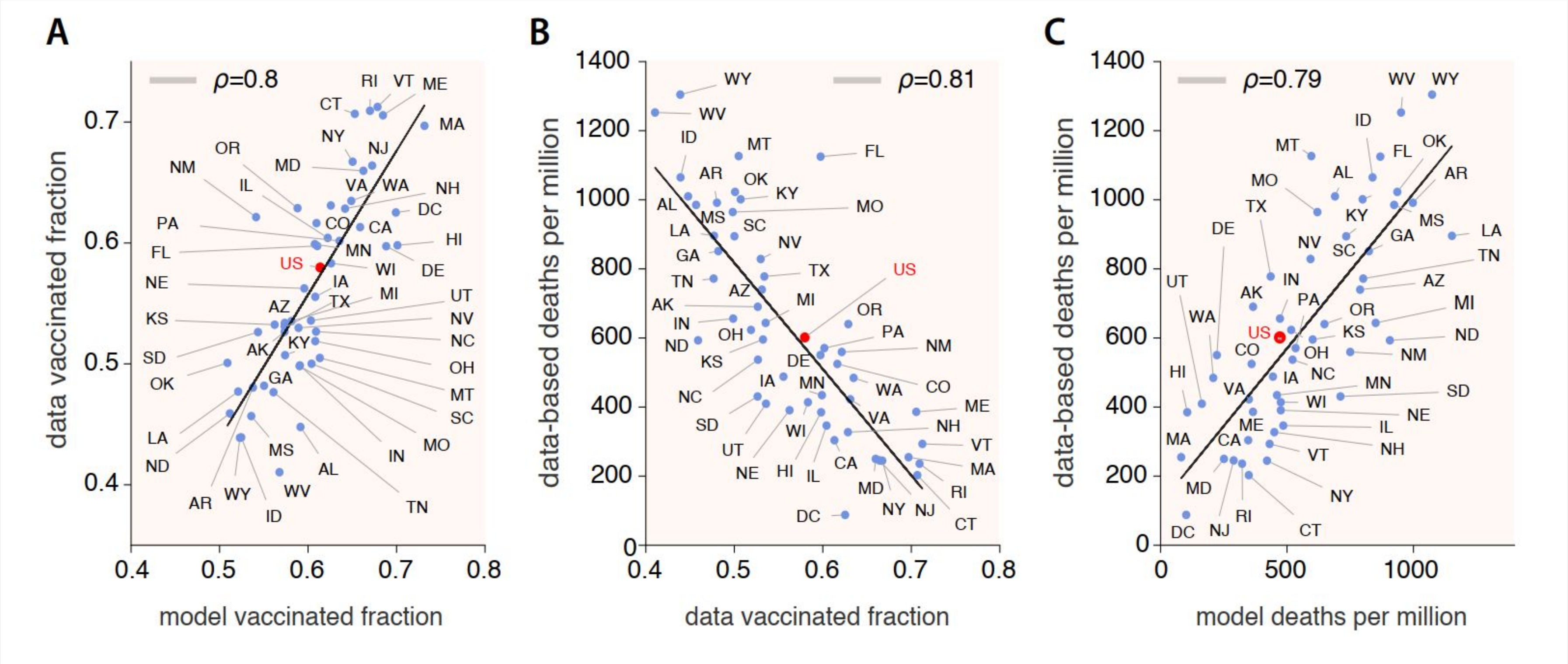
- RSFs are larger in the youngest age groups. Consequently, attack rates too.

- And rather small in the 65+ strata. Indeed, for AK and HI they are null. And very small for UT.

- This ultimately explains why these two states undergo large second outbreaks but limited deaths.



Results: Comparison against Delta-wave data



Even though relatively simple, our model was capable of (retrospectively) capture some macroscopic features of the epidemic.

Conclusions

Take-home messages

- We contributed to quantify the impact of hesitancy on secondary outbreaks, showing that simple models can bring relevant and timely insights provided reliable, fine-grained data is at hand.

- Hesitancy clearly impacts health outcomes, but age-structure may play a subtle role in how this impact is distributed across the population.

Future considerations

- Availability of some more grain-fined data (state-basis, age-structured & logistics) for vaccination campaigns and hesitancy could improve results conserving model's simplicity.

Work #2

Epidemic spreading in contact networks coupled to a threshold-based opinion dynamics on vaccine uptake

A. de Miguel-Arribas, A. Aleta & Y. Moreno

Background: Vaccine hesitancy (strikes back)

(Unexploited) survey reply categories:

Appendix A: Vaccine attitudes by state

If you were able to choose when to get a COVID-19 vaccine, would you get it...

State	Already vaccinated	As soon as possible	After at least some people I know	After most people I know	Would not get the COVID-19 vaccine	Error Margin	N
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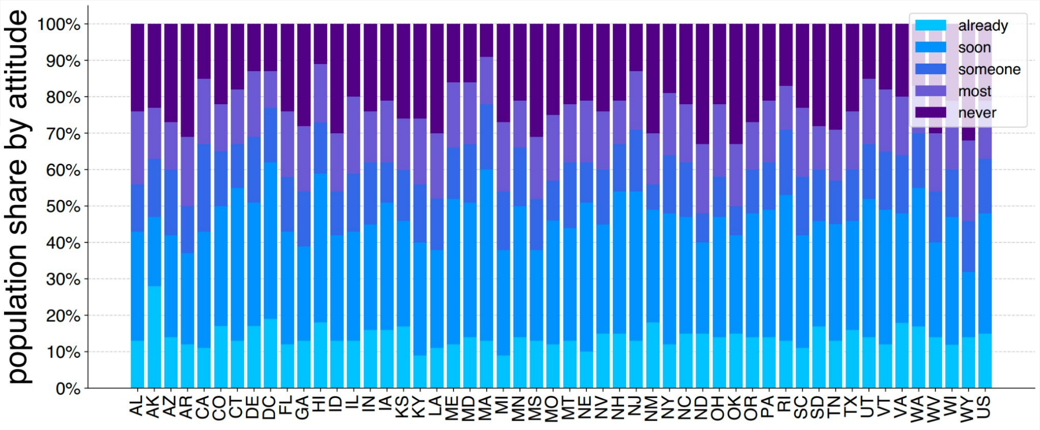


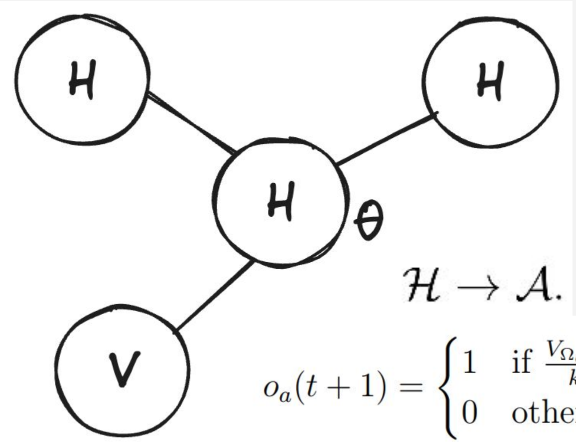
Threshold behavior + **social network** features: Better exploited on a networked population with explicit opinion dynamics.

Moreover: Coupled dynamics barely explored in the literature.

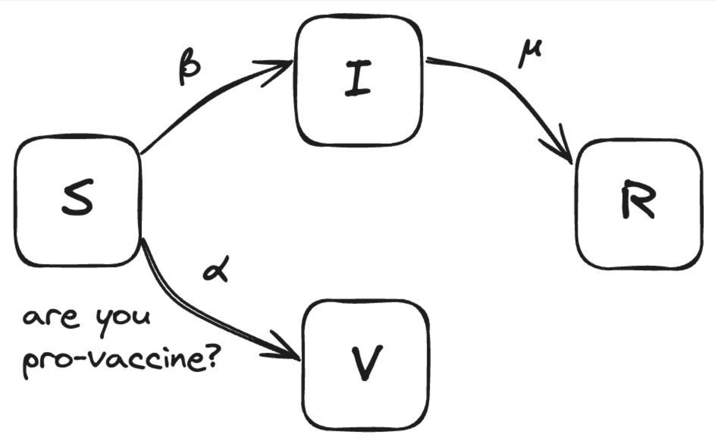
Question [?]

How a coupled opinion-vaccination dynamics impact the spreading of an epidemic in a networked population?

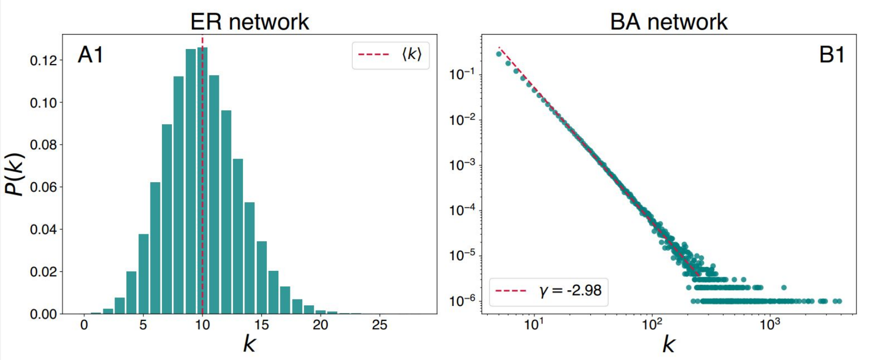
Epidemic + Vaccination + Opinion coupled dynamics



$$o_a(t+1) = \begin{cases} 1 & \text{if } \frac{V_{\Omega_a}(t)}{k_a} \geq \theta, \\ 0 & \text{otherwise.} \end{cases}$$

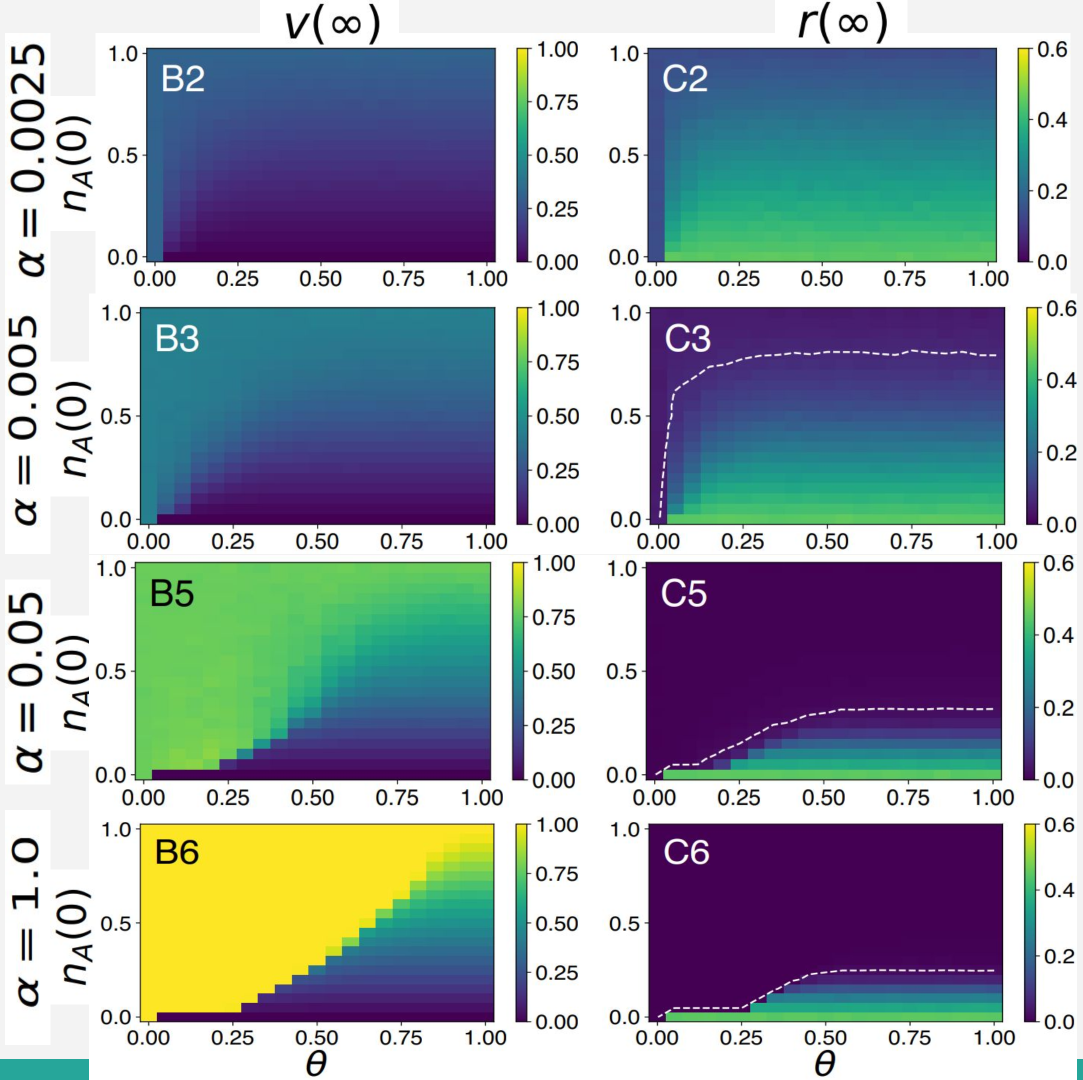


survey category	θ
already vaccinated	0
as soon as possible	0
after at least someone I know	$1/k_a$
after most people I know	0.5
would not get the [COVID-19] vaccine	1^+



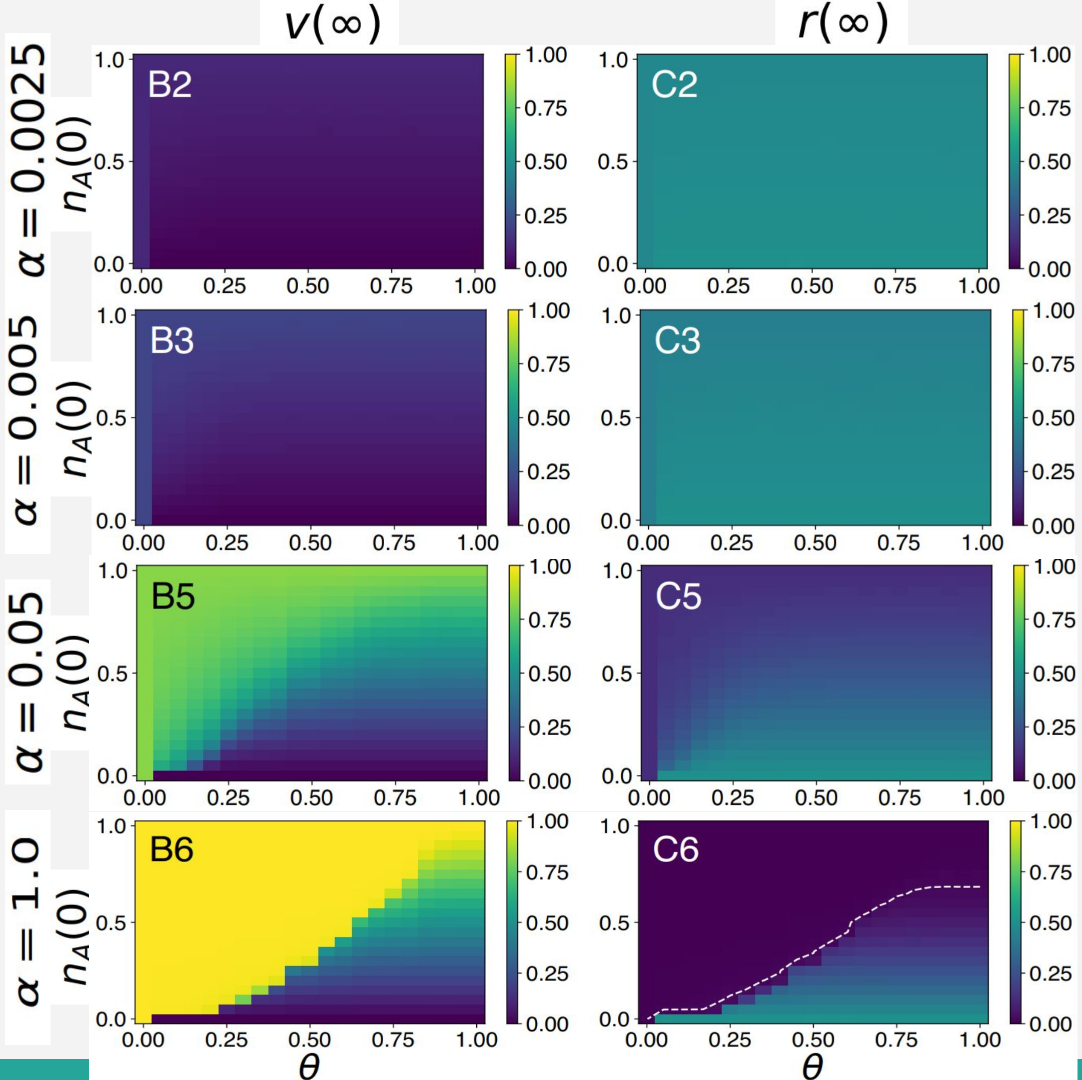
Results:
Homogeneous thresholds (ER networks)

- As α increases, cascading behavior triggered, increasing the VC & reducing prevalence.
- **ER networks:** **disease-free phase** emerges. Endemic phase relegated to very low pro-vaccine support $n_A(0)$ & medium-to-high activation threshold $\boldsymbol{\theta}$.
- Also, **diminishing returns** to vaccination observed beyond $\alpha = 0.05$.



Results: Homogeneous thresholds (BA networks)

- As α increases, cascading behavior triggered, increasing the VC & reducing prevalence.
- **ER networks: disease-free phase** emerges. Endemic phase relegated to very low pro-vaccine support $n_A(0)$ & medium-to-high activation threshold $\boldsymbol{\theta}$.
- Also, **diminishing returns** to vaccination observed beyond $\alpha = 0.05$.
- **BA networks:** disease-free phase requires **unrealistic vaccination efforts** to emerge.



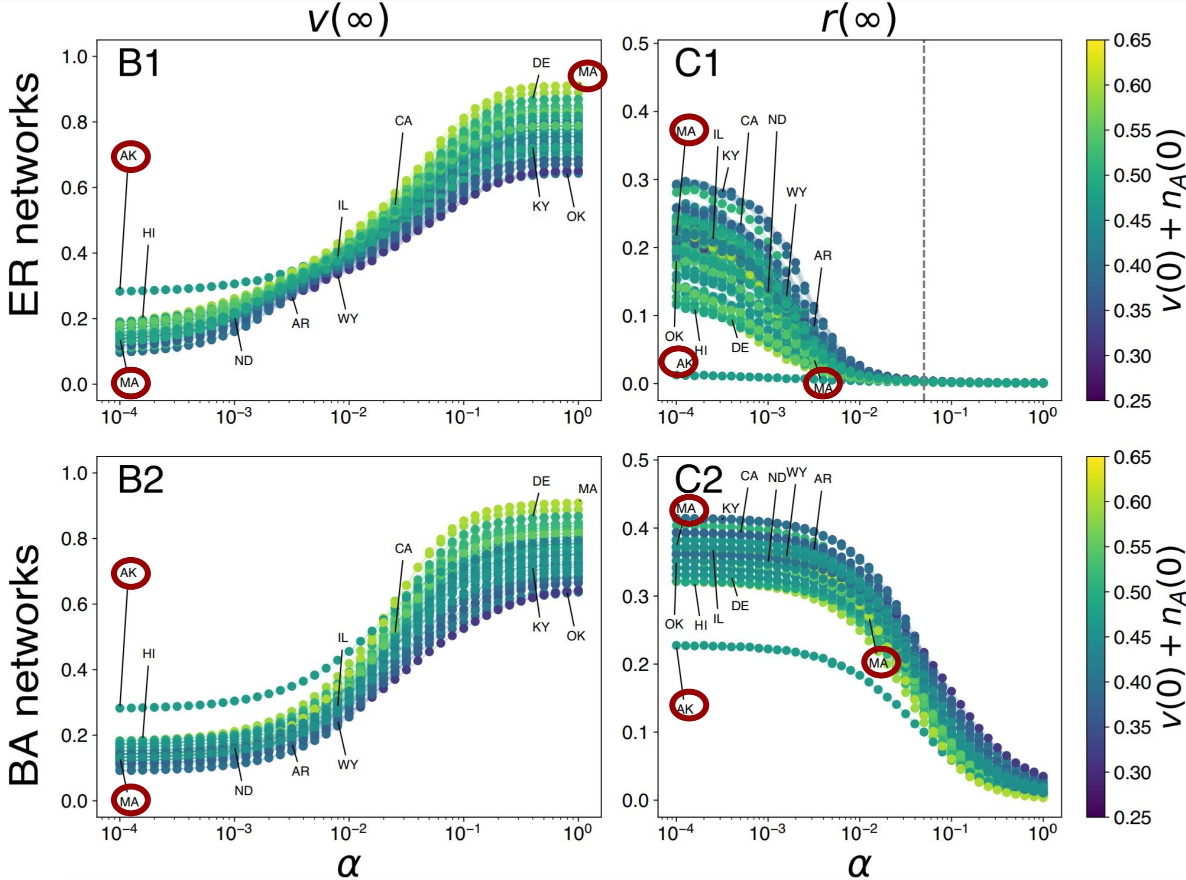
Results: Heterogeneous thresholds

- **Similar solution scenario** as in the homogeneous threshold $\boldsymbol{\theta}$ case.

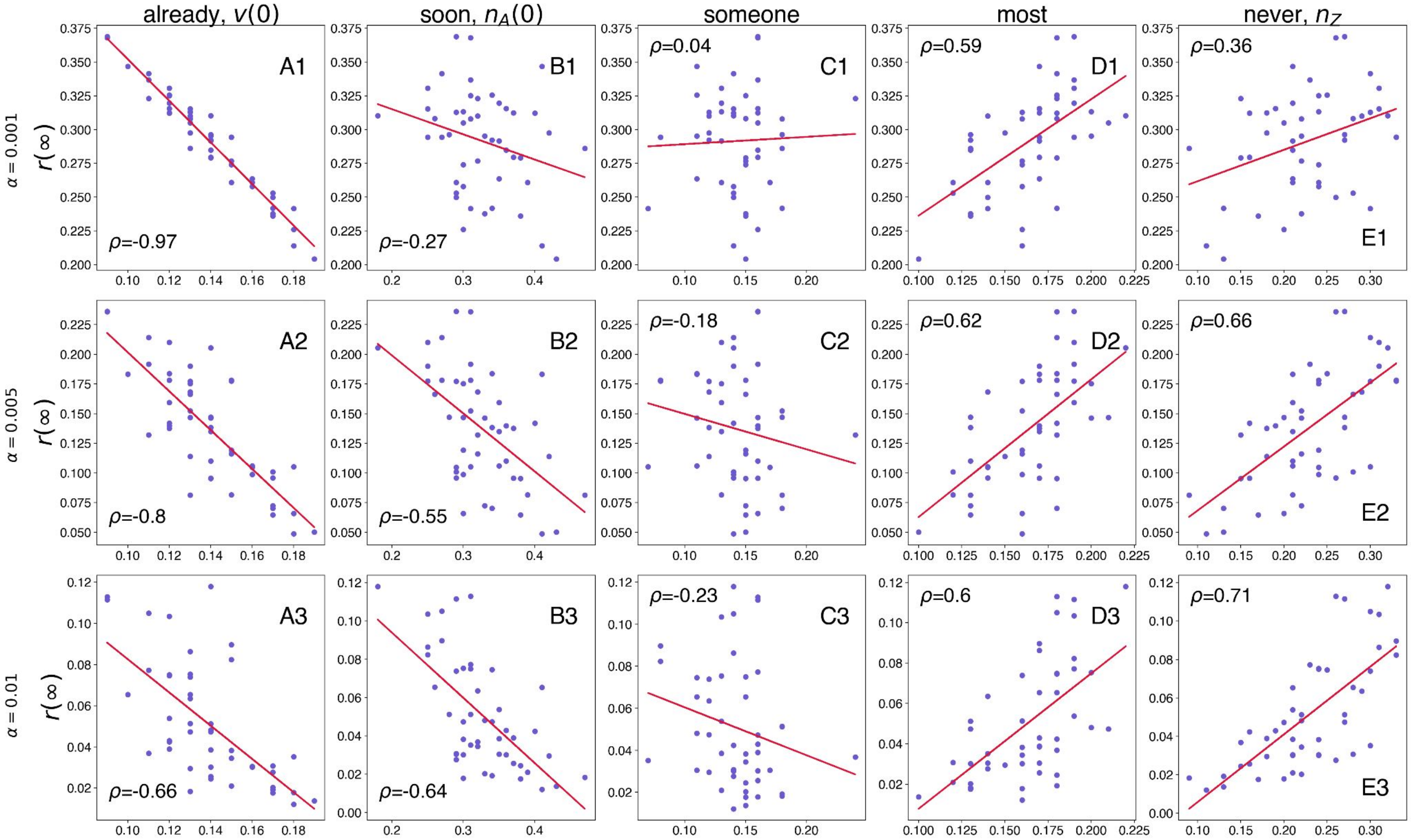
- Heterogeneity in attitudes for every state shows up in the curves' **non-monotonous changes**.

- **Example 1:** MA starts really bad (13% in 'already') but quickly ends on top (47% in 'soon').

- **Example 2:** AK starts strong already and shows no sizeable outbreaks all along.



Results: Prevalence vs attitudes correlations



'*Already v(0)*' play a key role in reducing prevalence, but explanatory power reduced as α grows.

'*Soon $n_A(0)$*' correlated negatively, explanatory power improves.

'*Never n_Z*' follows the same trend (correlating positively).

'*Most*' always shows some moderate correlation.

'*Someone*' is rather irrelevant.

Conclusions

Take-home messages

- Opinion-vaccination cascades can outperform disease spreading in ER networks. However, in BA networks, outbreaks are harder to mitigate even under high vaccination efforts.

- In a realistic setting, a diverse landscape of solutions may emerge. The influence of attitudes may strongly vary depending on the vaccination rate.

Future considerations

- Community structure & homophily in attitudes.

- Age structure (currently undergoing through an age-multilayer approach).

- More sophisticated decision-making (rational behavior, social media, etc.).

Part II. Metapopulation models

Work #3

www.nature.com/scientificreports

scientific reports

Check for updates

OPEN

Assessing the effectiveness of perimeter lockdowns as a response to epidemics at the urban scale

Alfonso de Miguel Arribas[1,2]✉, Alberto Aleta[1,2] & Yamir Moreno[1,2,3]

From September 2020 to May 2021 Madrid region (Spain) followed a rather unique non-pharmaceutical intervention (NPI) by establishing a strategy of perimeter lockdowns (PLs) that banned travels to and from areas satisfying certain epidemiological risk criteria. PLs were pursued to avoid harsher restrictions, but some studies have found that the particular implementation by Madrid authorities was rather ineffective. Based on Madrid's case, we devise a general, minimal framework to investigate the PLs effectiveness by using a data-driven metapopulation epidemiological model of a city, and explore under which circumstances the PLs could be a good NPI. The model is informed with real mobility data from Madrid to contextualize its results, but it can be generalized elsewhere. The lowest lockdown activation threshold Θ considered (14-day cumulative incidence rate of 20 cases per every 10^5 inhabitants) shows a prevalence reduction $> 20\%$ with respect to the scenario $\Theta = 10^3$, more akin to the case of Madrid, and assuming no further mitigation. Only the combination of $\Theta = 20$ and mobility reduction $> 90\%$ can avoid PLs for more than $> 20\%$ of the system. The combination of low Θ and strong local transmissibility reduction is key to minimize the impact, but the latter is harder to achieve given that we assume a situation with highly mitigated transmission, resembling the one observed during the second wave of COVID-19 in Madrid. Thus, we conclude that a generalized lockdown is hard to avoid under any realistic setting if only this strategy is applied.

Assessing the effectiveness of perimeter lockdowns as a response to epidemics at the urban scale

A. de Miguel-Arribas, A. Aleta & Y. Moreno

Background: Madrid's strategy

- **Epidemiological context**: Second wave of COVID-19 building up .

- **Socioeconomic context**: Avoid “economic ruin”.

- **Perimeter lockdowns**:

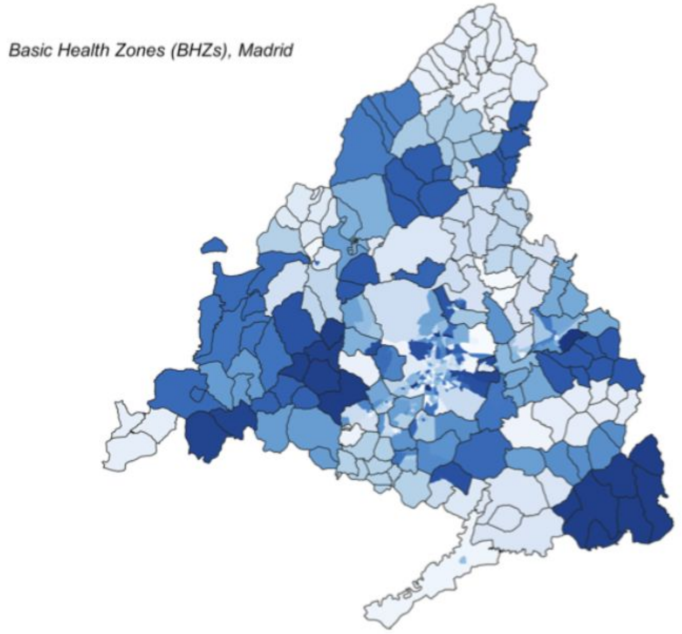
    - Spatial unit: Basic Health Zones (37 affected)

    - Time extension: 14 days or more.

    - First round: September 23, 2020.

    - **Conditions**:
        - 14-day CIR > 10^3 cases per 10^5 inhabitants.
        - Increasing trend.
        - Community spread observed.

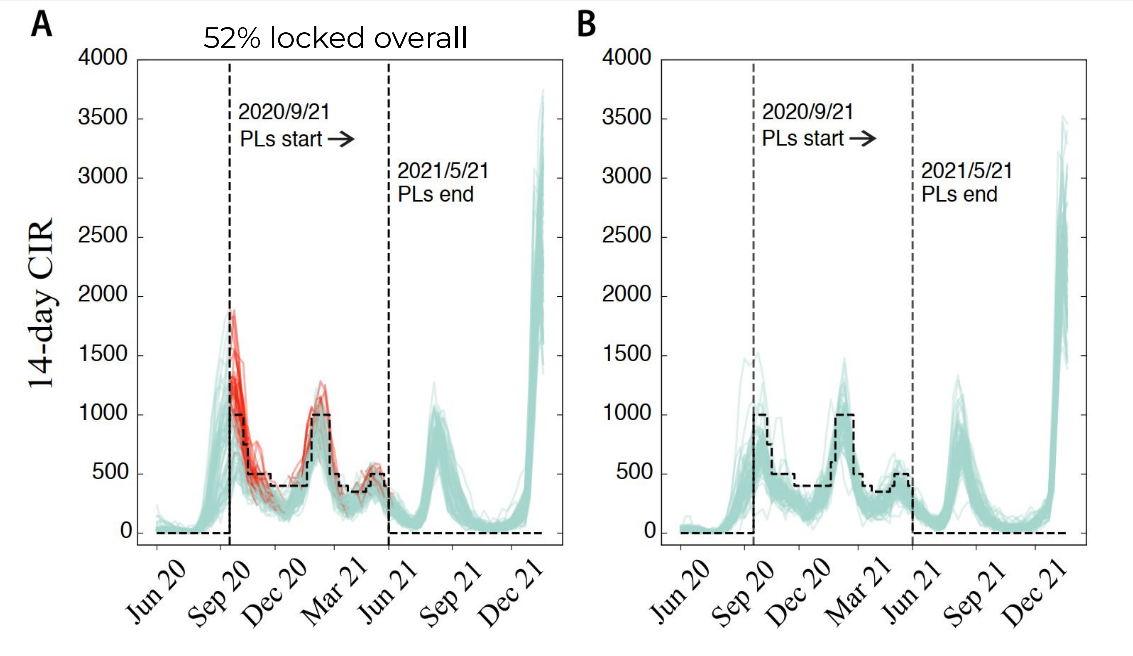
- Additionally:
General ‘social distancing’ measures in place...



from García-García et al. (2022)

https://doi.org/10.1186/s12889-022-12626-x

Material: Madrid epidemiological data



Observations from gathered data:

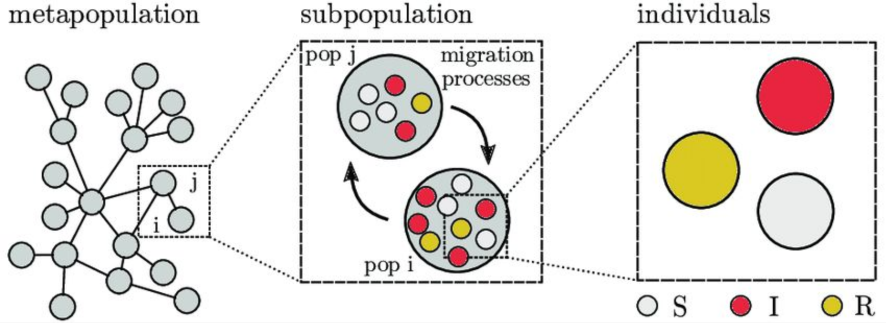
- Peak incidence & activation occurs at around the same time in the 1st round.
- Literature confirmed through formal statistical analyses that lockdowns had **no real effect**.
- **Threshold** criterion? Follows the epidemic waves and some BHZs were not locked.
- Highly **synchronized** time series.

Data casts some doubts about the strategy and the implementation in itself...

Question [?]

Are perimeter lockdowns an effective strategy to contain an epidemic spreading at the urban scale?

Metapopulation with real mobility & lockdowns



Epidemic: Homogeneous-mixing SIR within Madrid's districts.

Mobility: Origin-Destination matrices with real Madrid data from pre-COVID-19 reference period.

$$D_{ij} = \begin{cases} \kappa \frac{M_{ij}}{\sum_j M_{ij}} & \text{if } i \neq j \\ 1 - \sum_{k \neq i} D_{ik} & \text{if } i = j \end{cases}$$

Lockdowns: 14-d CIR as surveillance variable.

- *Activation threshold ϴ.*
- *In-and-out mobility cut down.*
- Local transmission rate reduction fraction χ_i so that $\beta_i = \chi\, \beta$, where $\beta = R_0 T_I$. [with R_0=1.25 (< COVID-19's)]

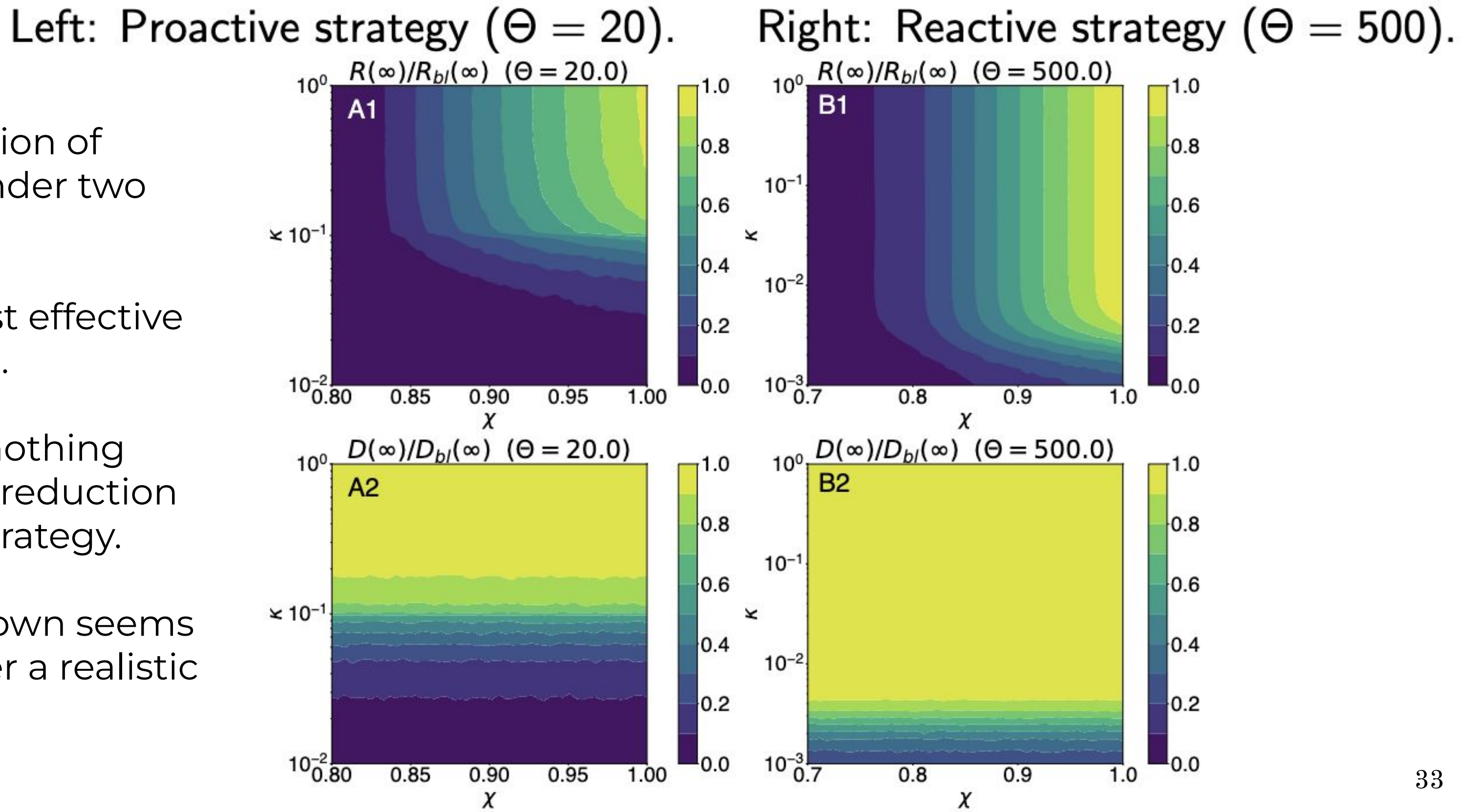
Results: Prevalence & district invasion

- Prevalence & fraction of locked districts under two strategies.

- Reducing χ is most effective (Hard to achieve?).

- Mobility κ? Does nothing until beyond 90% reduction in the proactive strategy.

- Full system lockdown seems unavoidable under a realistic scenario.



Conclusions

Take-home messages

- At the urban scale outbreak propagate fast and are highly synchronized.
- If the aim is to protect some parts of the system... Lockdowns should be activated unrealistically soon and tight
- Restricting mobility by itself does little to nothing.

Future considerations

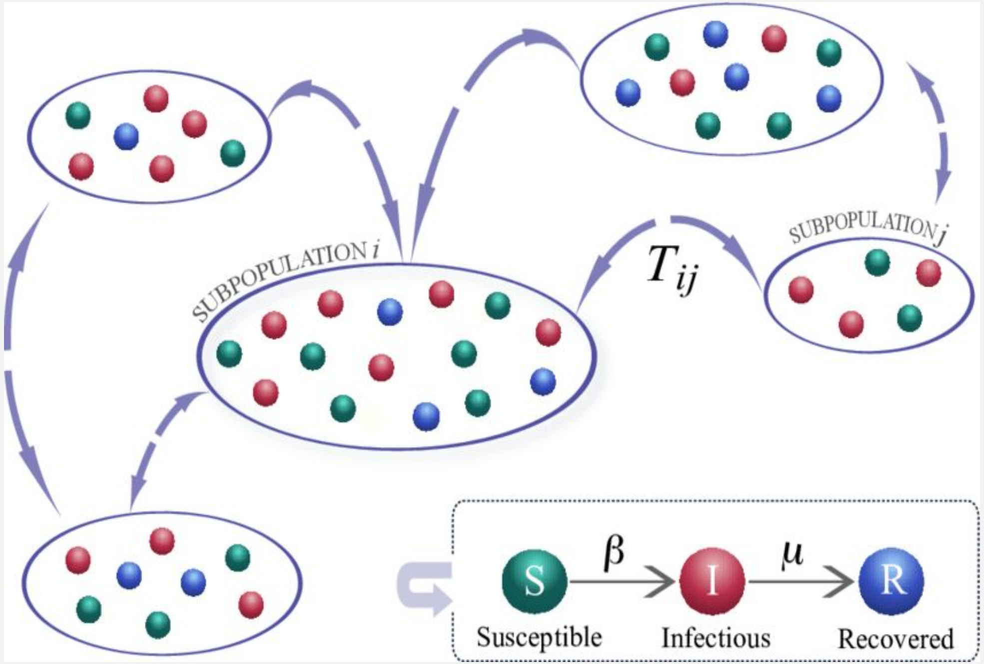
- Depart from homogeneous-mixing at district level.
- Improve spatial resolution.
- More realistic mobility models.

Work #4

Epidemic spreading in an urban environment under the d-EPR model of microscopic human mobility

A. de Miguel-Arribas, A. Aleta, Y. Moreno & E. Moro

Background: Metapopulation models



Ventura et al. (2022): Modeling the effects of social distancing on the large-scale spreading of diseases.

Mobility models (typically) assume:

- Markovian random walks.
- Indistinguishable agents.

Background: Advances in human mobility

- Last decade: Exploration and preferential return models.

- Analysis of human mobility datasets reveal two main types of behaviors:

EXPLORERS & RETURNERS

- Literature mentions the relevance of these discoveries to epidemics, but have not been thoroughly explored.

Vol 453|5 June 2008|doi:10.1038/nature06958 nature

LETTERS

Understanding individual human mobility patterns

Marta C. González[1], César A. Hidalgo[1,2] & Albert-László Barabási[1,2,3]

ARTICLES
PUBLISHED ONLINE: 12 SEPTEMBER 2010 | DOI: 10.1038/NPHYS1760
nature physics

Modelling the scaling properties of human mobility

Chaoming Song[1,2†], Tal Koren[1,2†], Pu Wang[1,2†] and Albert-László Barabási[1,2,3★]

nature COMMUNICATIONS

ARTICLE
Received 15 Dec 2014 | Accepted 24 Jul 2015 | Published 8 Sep 2015 DOI: 10.1038/ncomms9166 OPEN

Returners and explorers dichotomy in human mobility

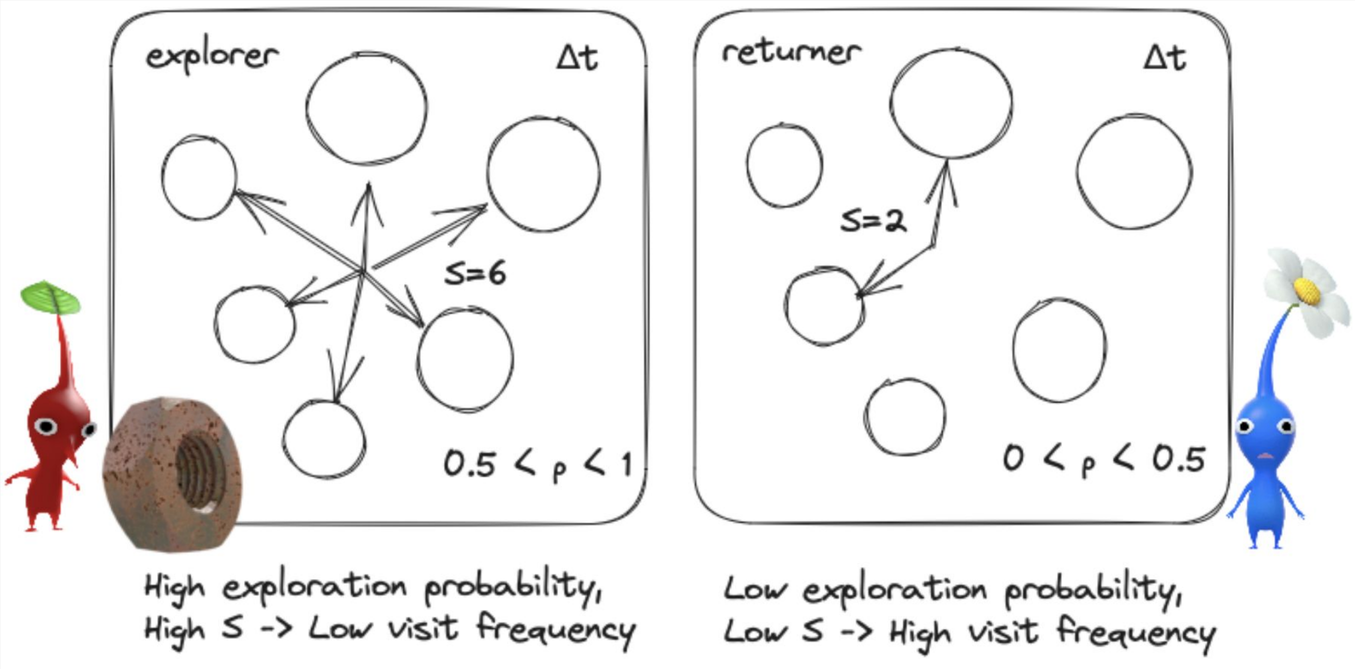
Luca Pappalardo[1,2,3,4], Filippo Simini[4,5,6], Salvatore Rinzivillo[1], Dino Pedreschi[1,2], Fosca Giannotti[1] & Albert-László Barabási[3,6,7]

The availability of massive digital traces of human whereabouts has offered a series of novel insights on the quantitative patterns characterizing human mobility. In particular, numerous recent studies have lead to an unexpected consensus: the considerable variability in the characteristic travelled distance of individuals coexists with a high degree of predictability of their future locations. Here we shed light on this surprising coexistence by systematically investigating the impact of recurrent mobility on the characteristic distance travelled by individuals. Using both mobile phone and GPS data, we discover the existence of two distinct classes of individuals: returners and explorers. As existing models of human mobility cannot explain the existence of these two classes, we develop more realistic models able to capture the empirical findings. Finally, we show that returners and explorers play a distinct quantifiable role in spreading phenomena and that a correlation exists between their mobility patterns and social interactions.

Question [?]

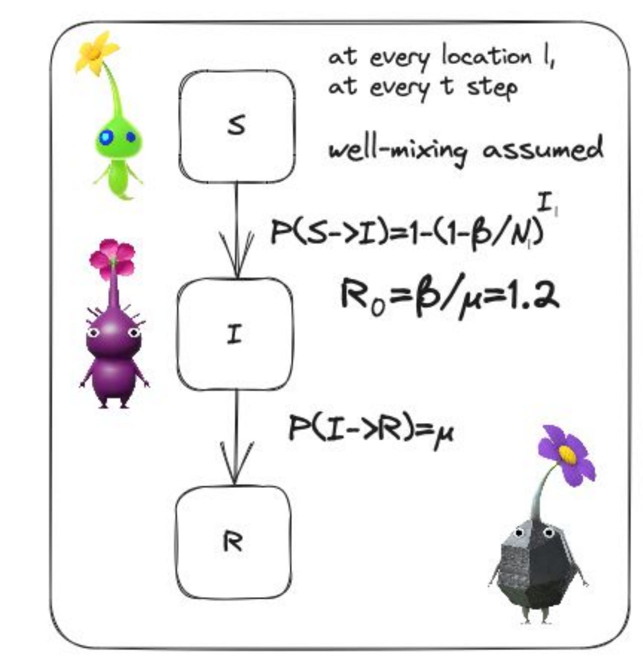
What is the role of explorers & returners in the spreading of epidemics?

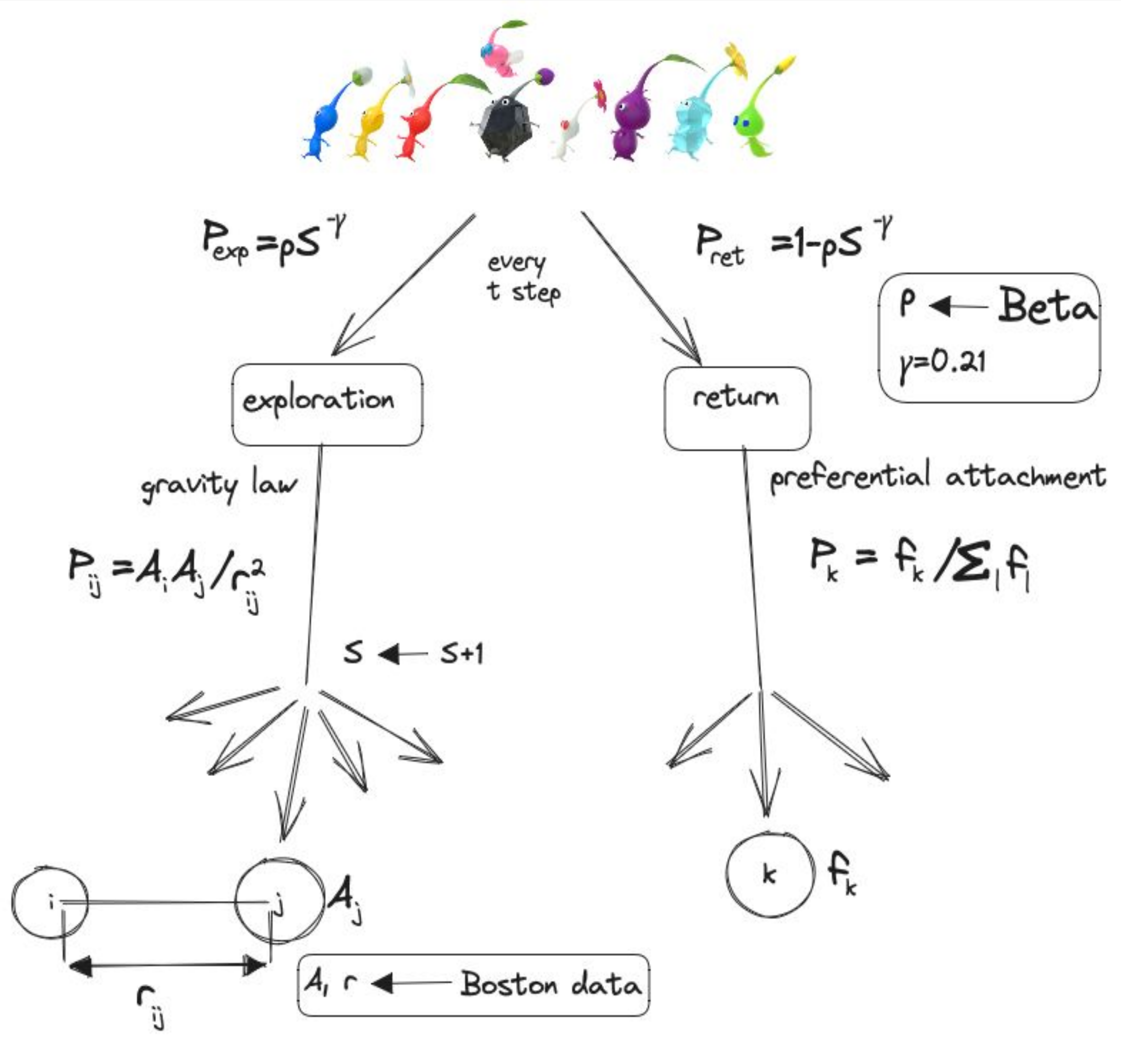
Explorer & returner profiles



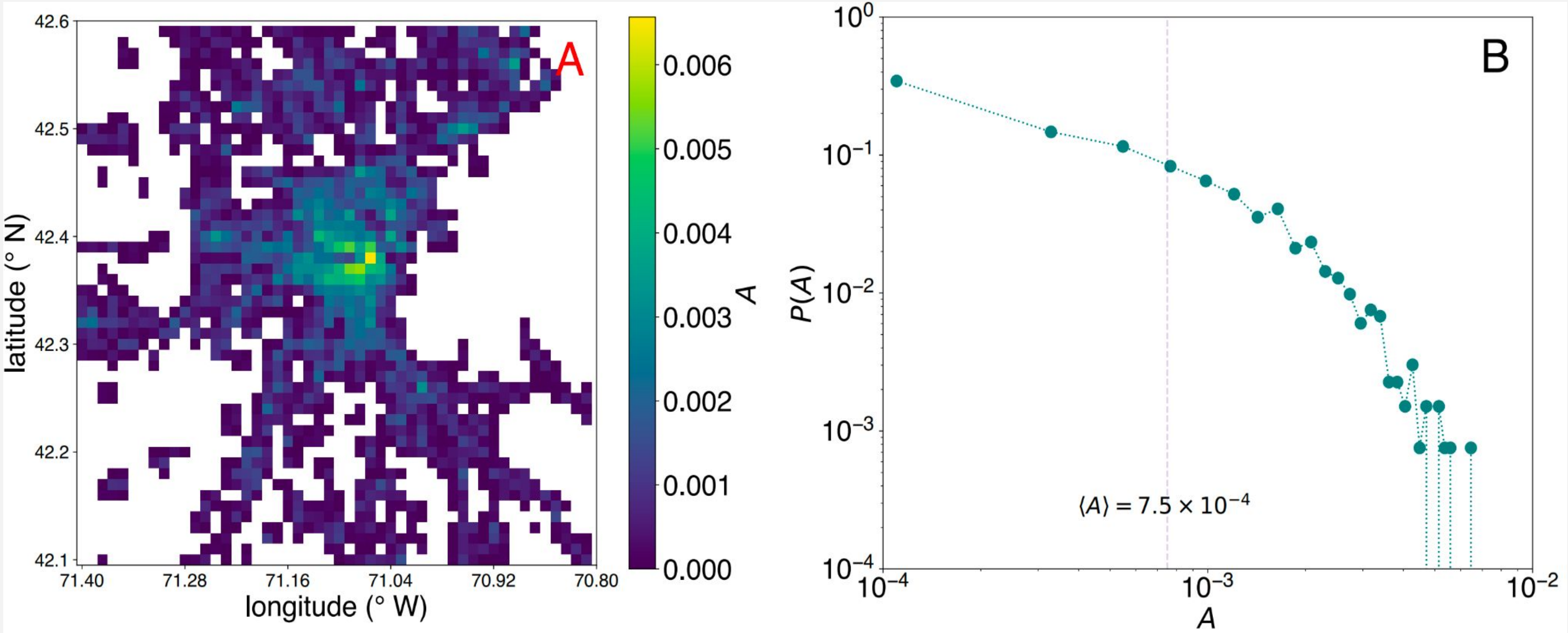
- Parameter ϱ is related to the ratio of distinct visited locations and total visits in a given Δt.
- Results analyzed in homogeneous populations (ϱ from a **Gaussian** distribution) and from a more heterogeneous one (ϱ sampled from a **Beta** distribution).

SIR model
+ d-EPR model





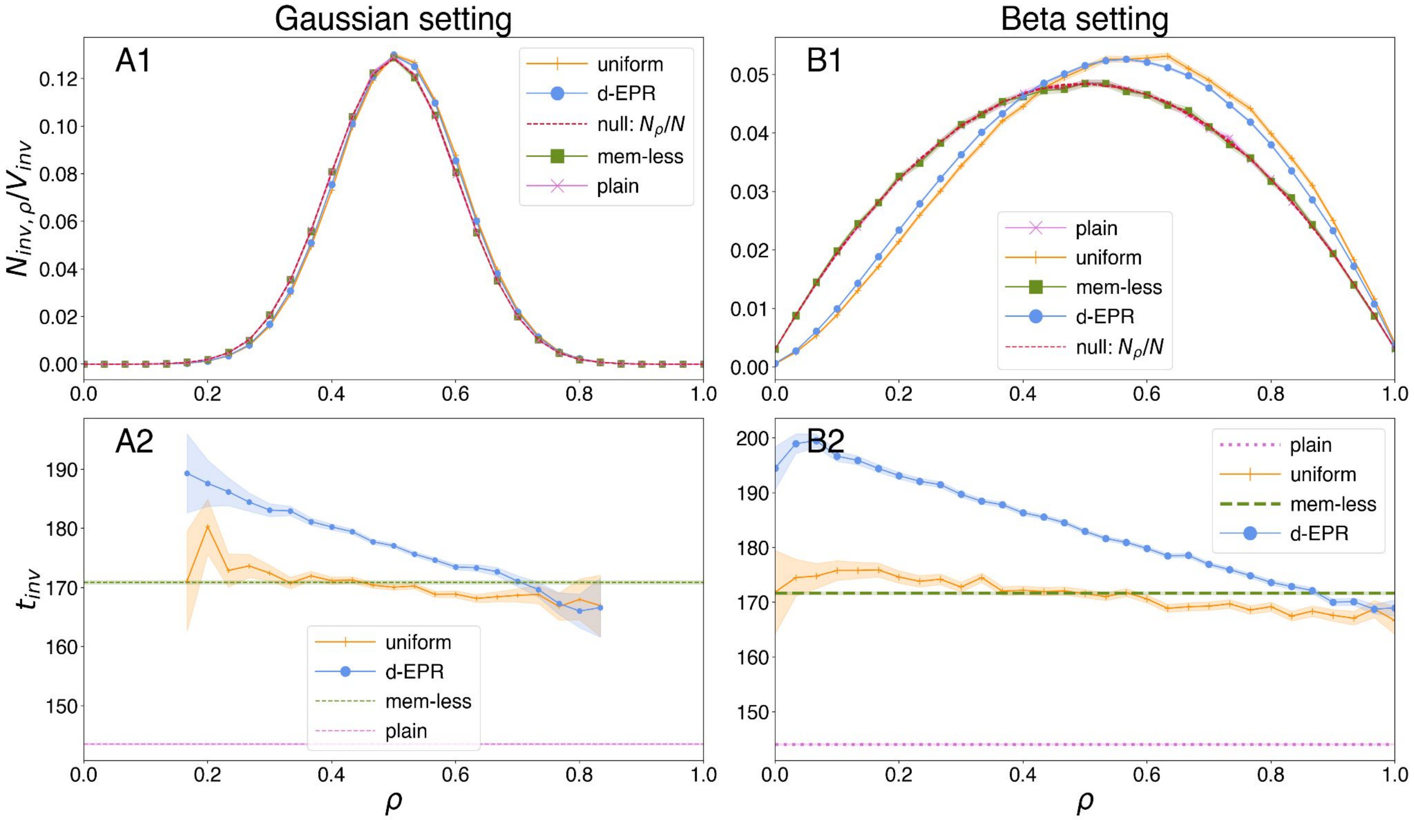
Spatial structure: Locations' attractiveness



Left: Field reconstruction from high-resolution individual anonymized trajectories from Cuebiq.
Right: Attractiveness distribution (log-log).

Effective system size V~1300 of 1km^2 each cell.

Disease invasion process: Explorers drive it



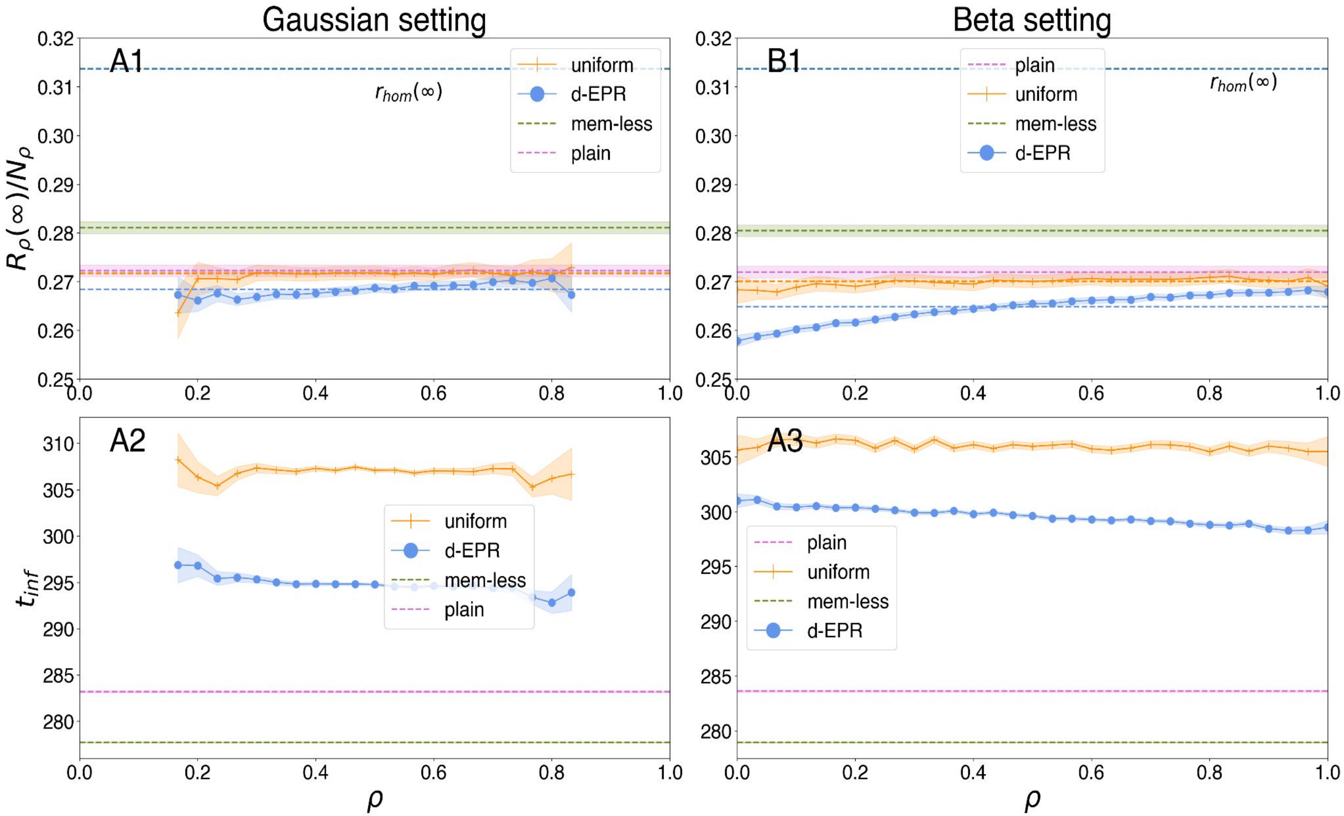
Invasion: secondary case for the 1st time in locations other than the epicenter.

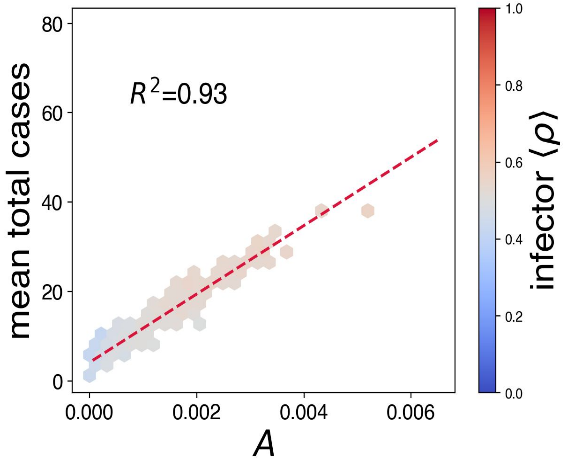
Who (per ϱ-profile)drives the invasion & **when** occurs?

Comparisons of hom. & het. populations & simplified versions of the d-EPR model.

Only the **d-EPR** shows **deviations** from the null case in invasion fraction & varying invasion times across ϱ-profiles.

Disease prevalence



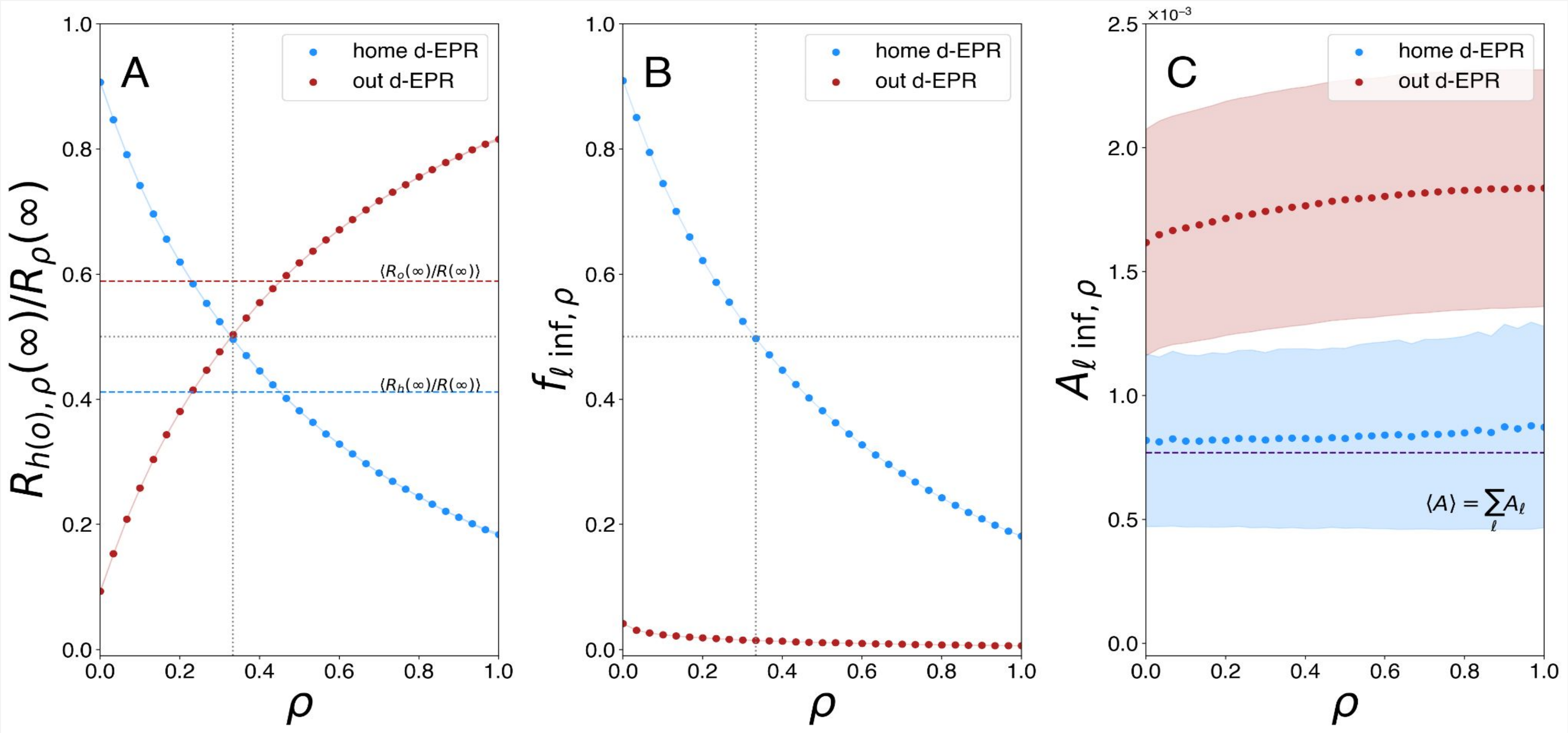


d-EPR: Explorers & returners deviate from the global average.

Infection times differ much less than invasion times (**high synchronization**)

Explorers tend to be infected in **most attractive locations** which accumulate **more cases**.

Origin of infection, recurrence & attractiveness

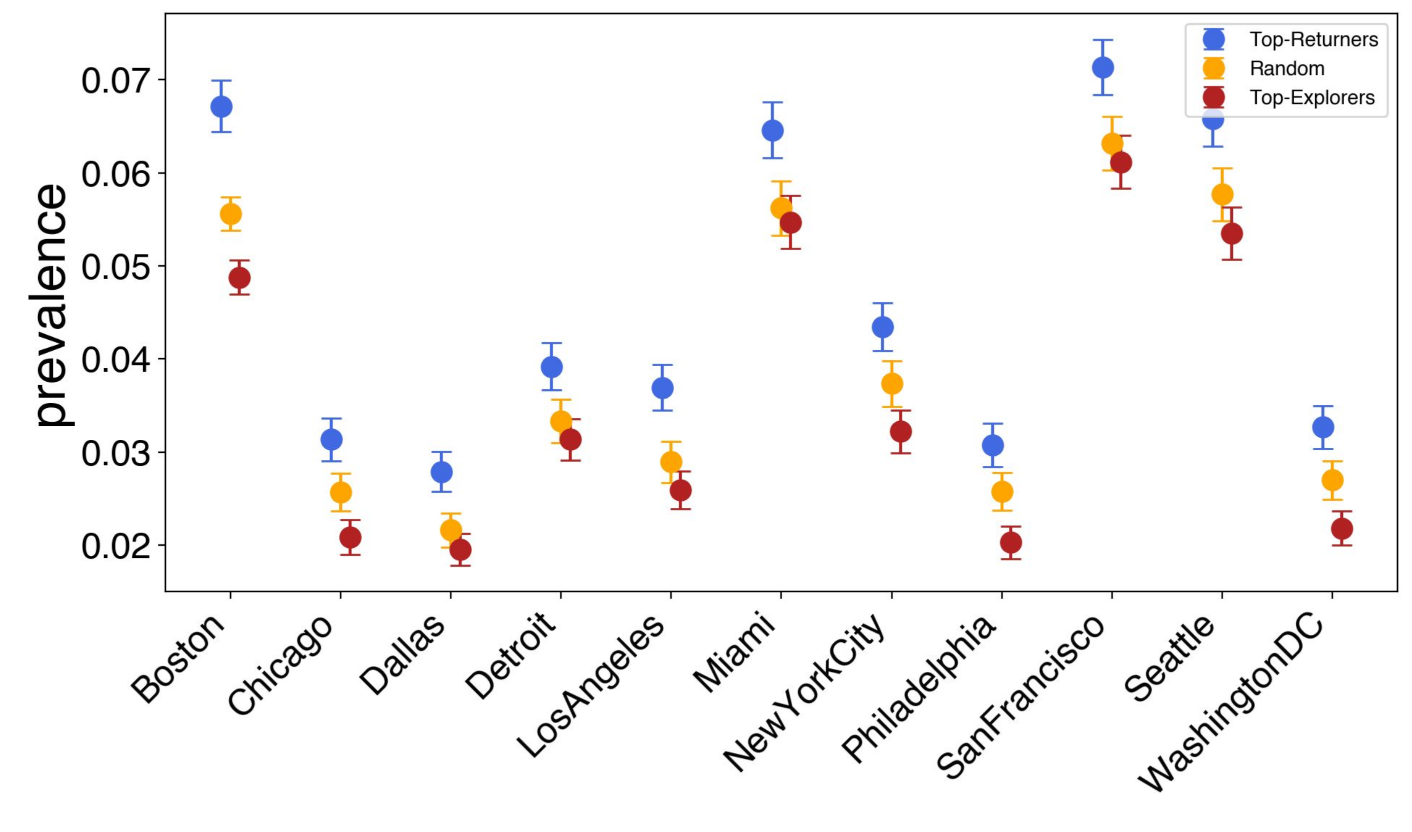


Majority of infections occur **outside** home location → Very **small recurrence** → Bad luck?
No! Agents were just wandering around **very attractive** locations (which concentrate more cases).

Targeted-mobility control policy

A fraction (10%) of the population is **immunized** based on their **mobility profile**.

Overall, **targeting exploration** brings lower global prevalence compared to the other strategies, across the urban systems analyzed.



Conclusions

Take-home messages

- Explorers (high ϱ-profile) deliver the disease across the system, they do it faster and are impacted more than returners.
- Even for medium-low ϱ-profiles, the majority of infections occur in high attractiveness locations.
- Targeted-mobility policies can have a positive impact in certain urban environments.

Future considerations

- More realistic extensions within EPR family (or other).
- Comparison to trajectories from real individuals.

Closure

Final remarks

- We have explored a variety of topics related to the modeling of epidemic spreading through the use of real data and mechanistic computational models.

- We have tried both to characterize real situations and understand fundamental mechanisms.

- Without resorting to highly sophisticated models, we can offer useful insights provided models are fed with reliable & quality data.

- Accounting for heterogeneity (age, behavior, contacts, mobility) is fundamental to provide a more nuanced picture of the phenomena and potentially apply more effective health policies.

Muchas gracias.

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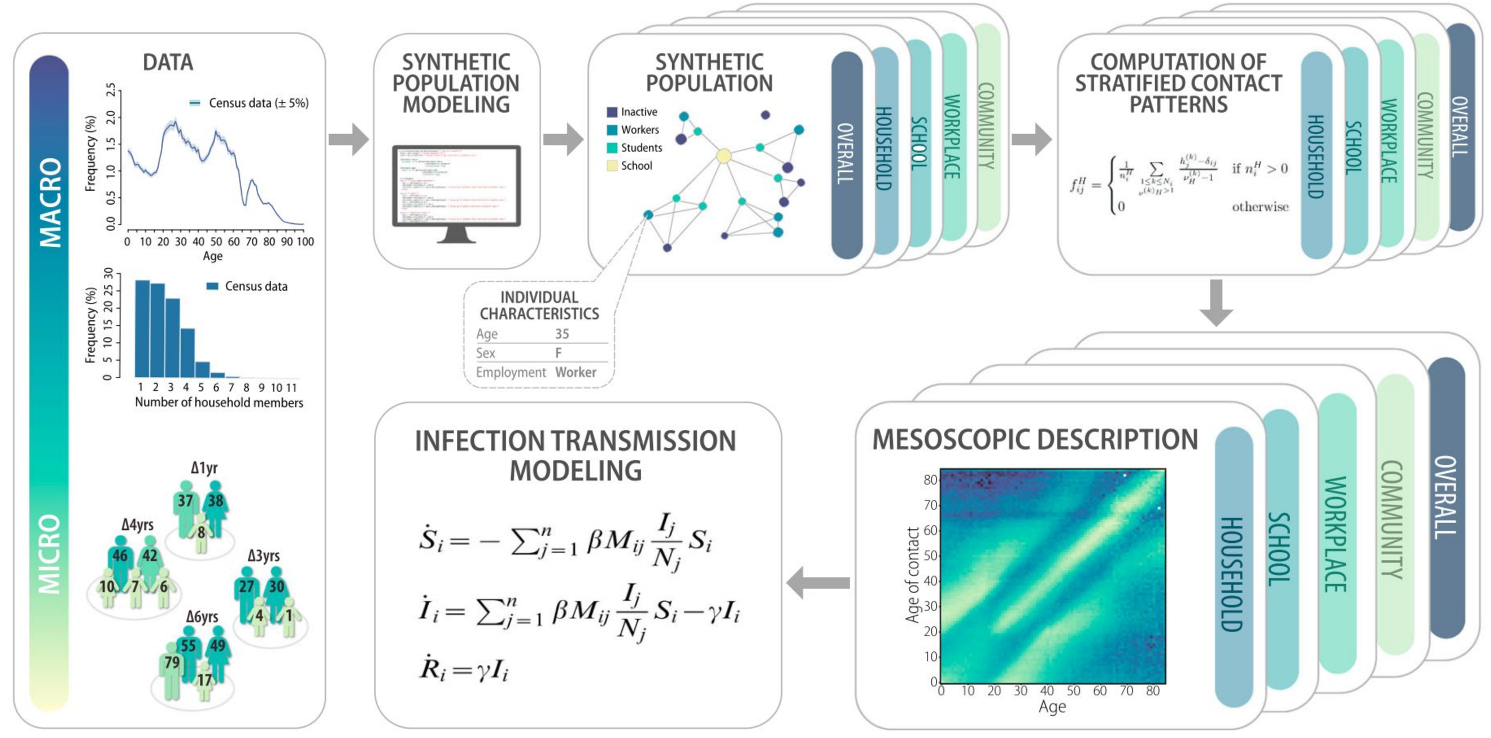
Cosnet

Supplementary Material

W#1: Hesitancy. Mistry et al. (2021)



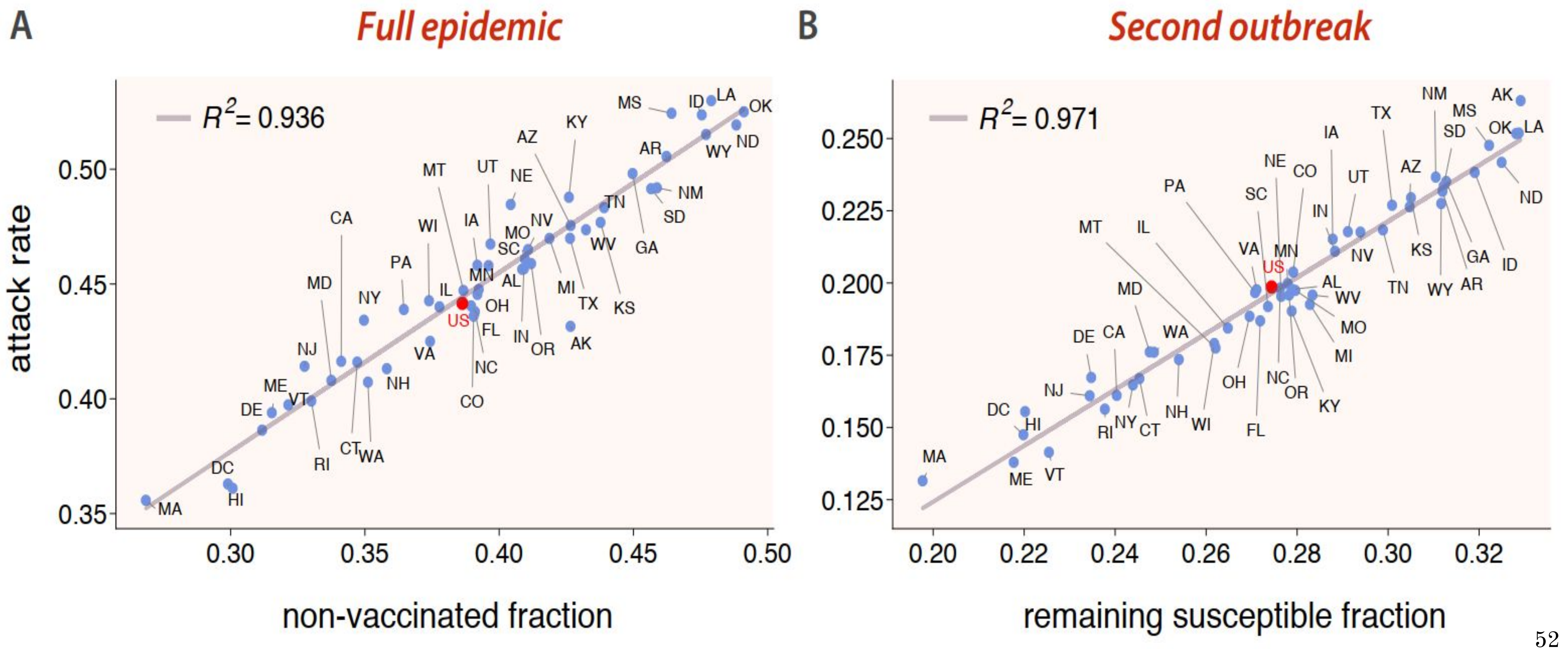
$$\dot{S}_i = -\sum_{j=1}^{n} \beta M_{ij} \frac{I_j}{N_j} S_i$$

$$\dot{I}_i = \sum_{j=1}^{n} \beta M_{ij} \frac{I_j}{N_j} S_i - \gamma I_i$$

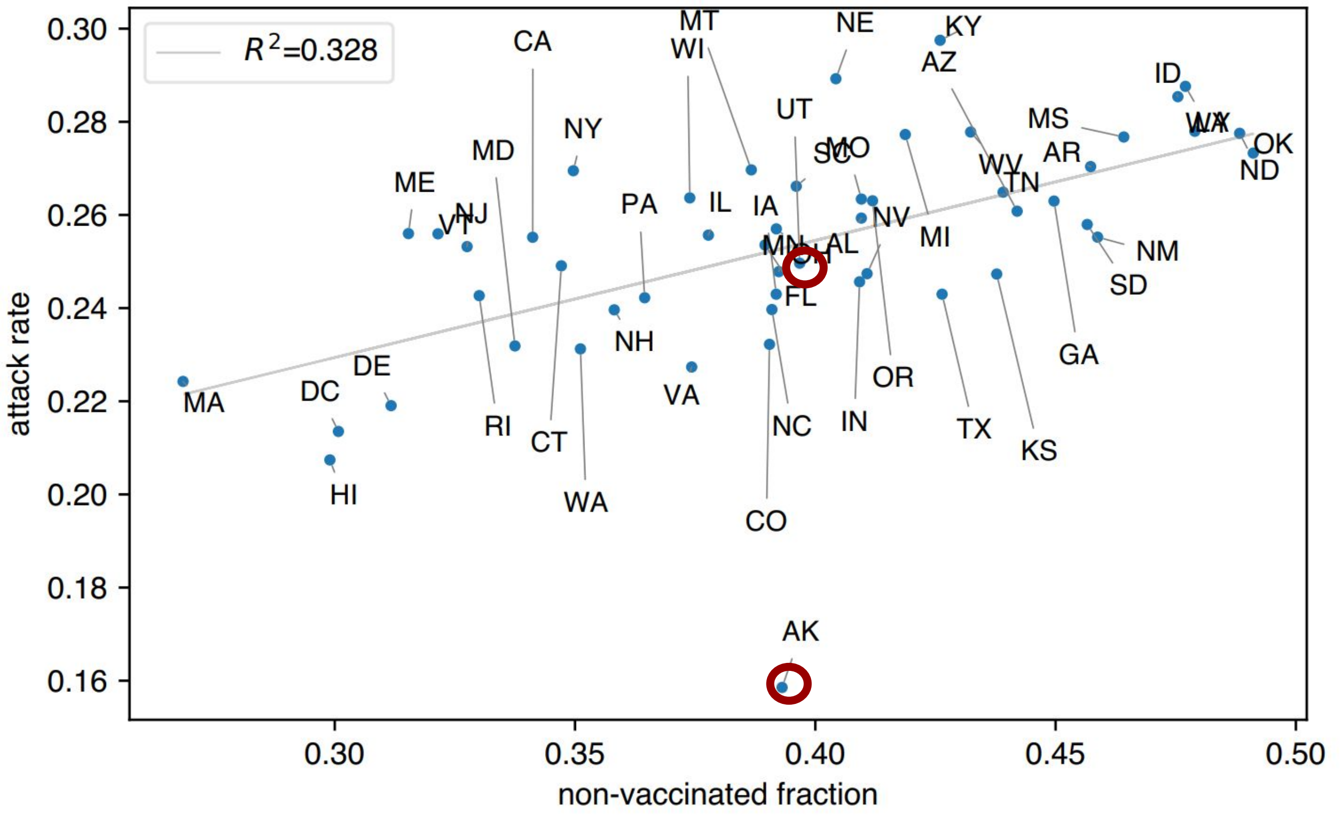
$$\dot{R}_i = \gamma I_i$$

Fig. 1 Modeling framework. Schematic representation of the workflow for modeling human-mixing patterns and infection transmission dynamics.

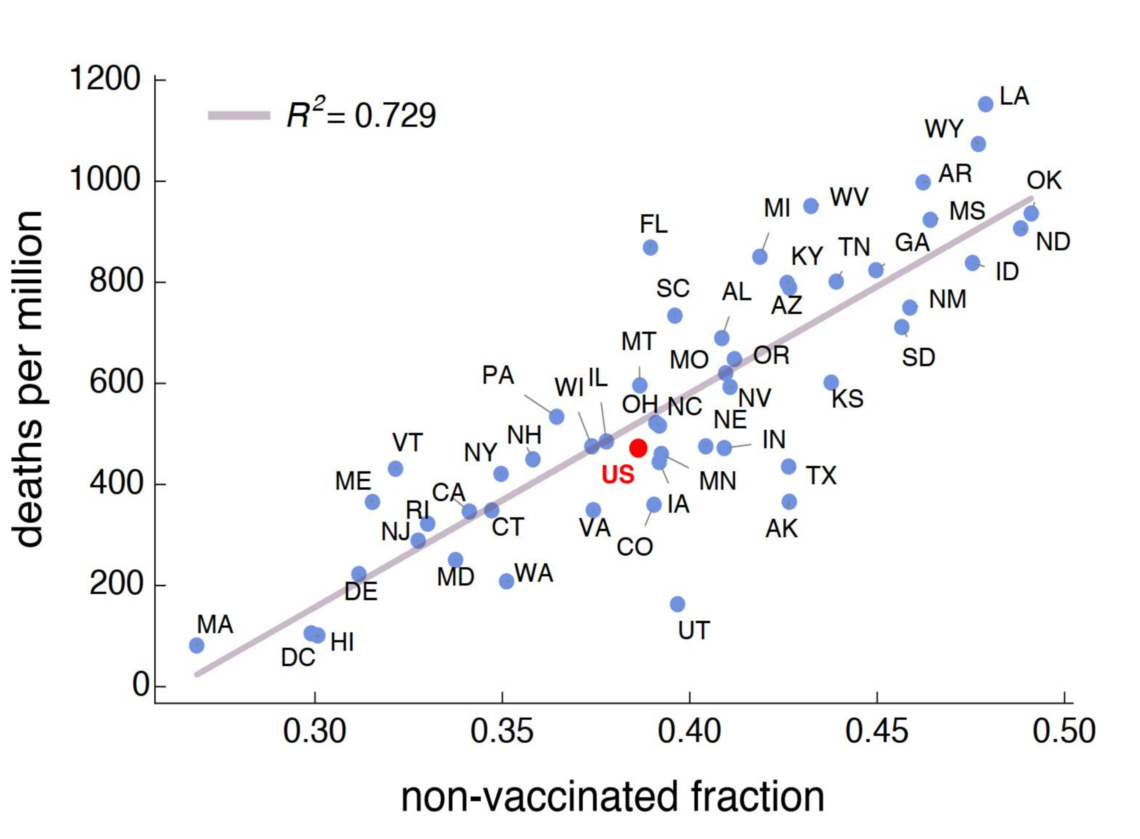
W#1: Hesitancy. Attack rate scatter plots



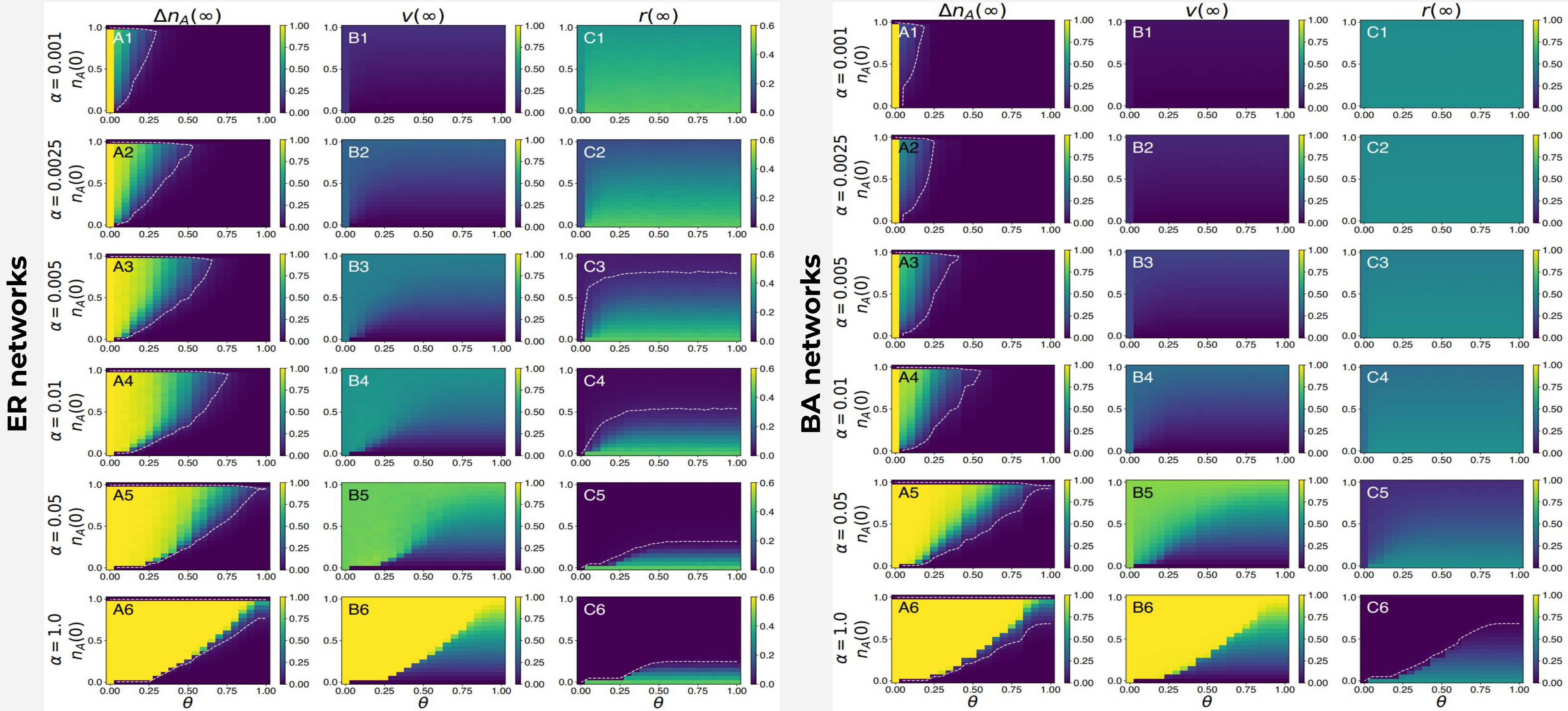
W#1: Hesitancy. Attack rate in 1st outbreak



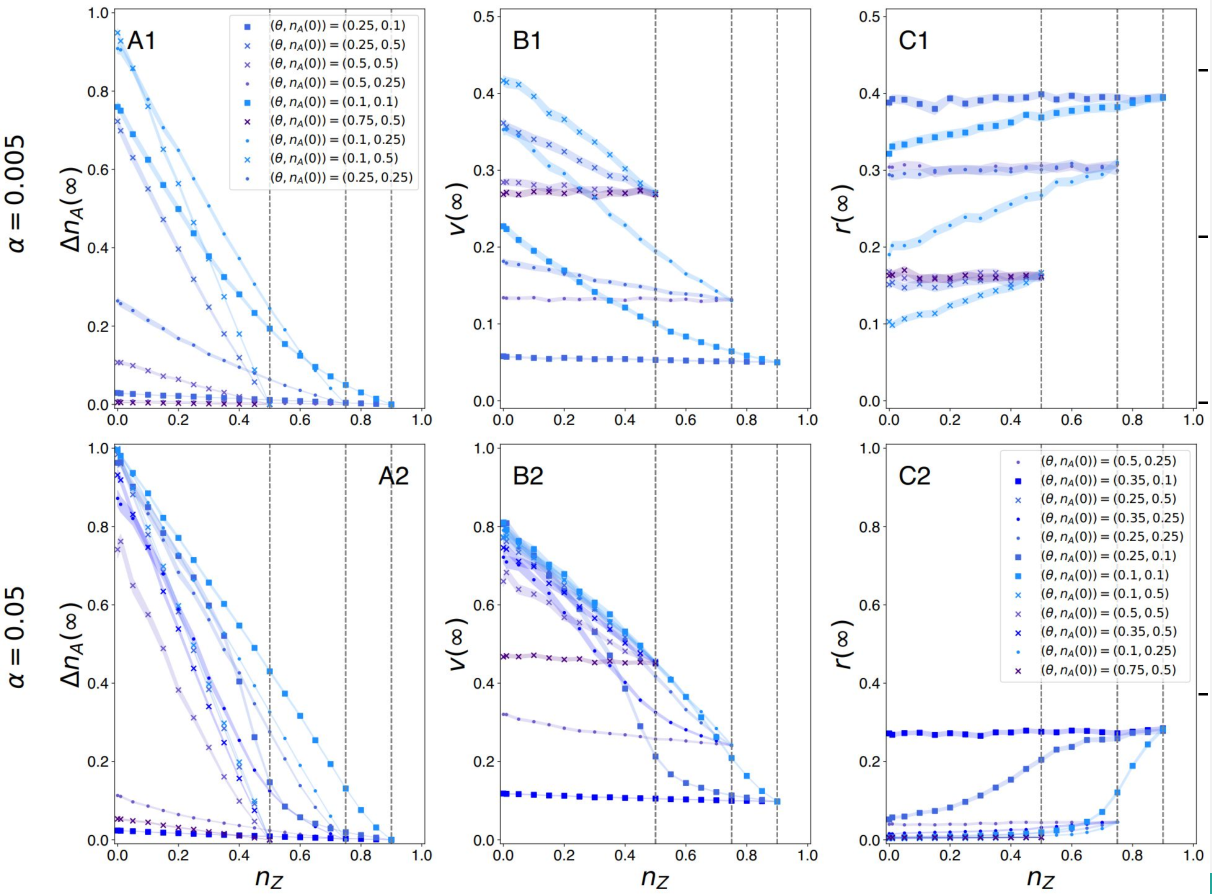
W#1: Hesitancy. Deaths during 2nd outbreak.



W#2: Results. Homogeneous thresholds



W#2: Results. Introduction of anti-vaccine agents



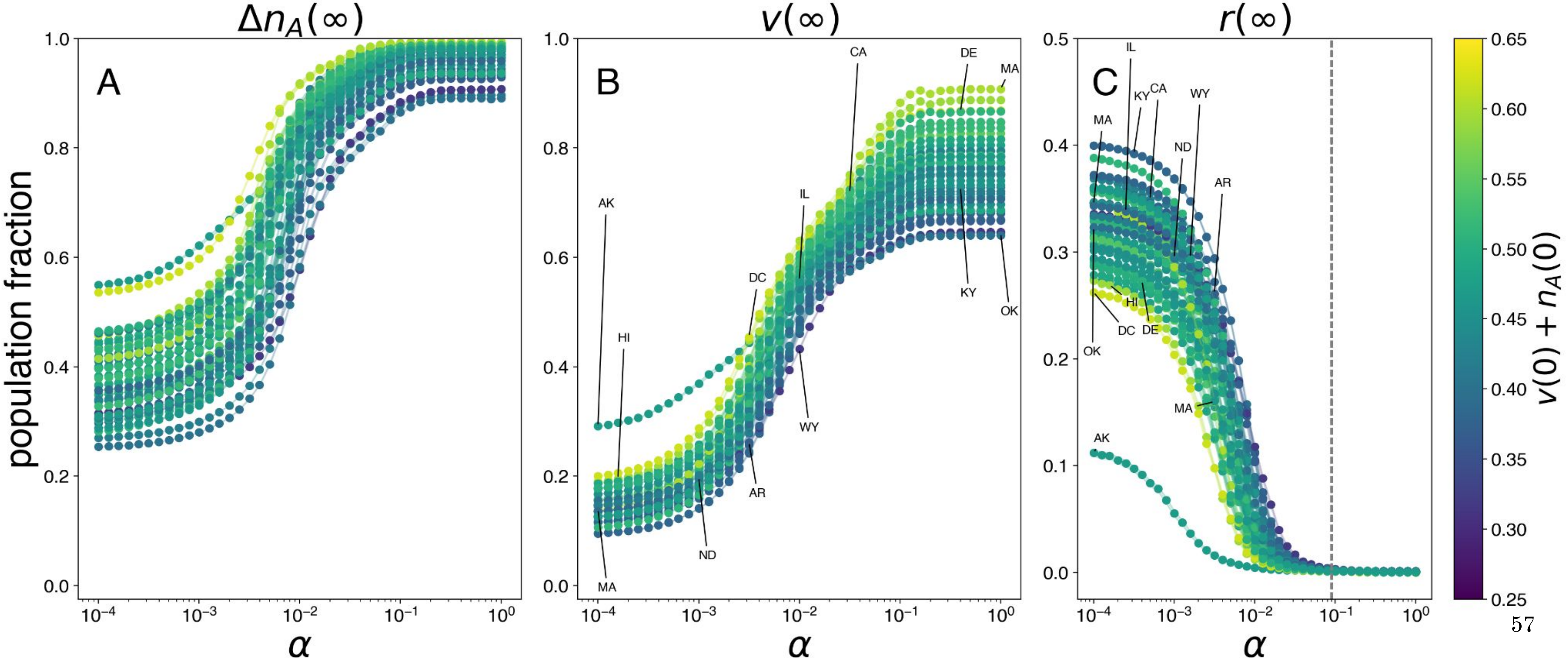
Situation 1: points where $\Delta n_A(\infty)=0$ at $n_Z=0$ are unaffected by subsequent increases in n_Z.

Situation 2: points where $\Delta n_A(\infty) \neq 0$ at $n_Z = 0$ show reduced VC, and increasing prevalence with rising n_Z.

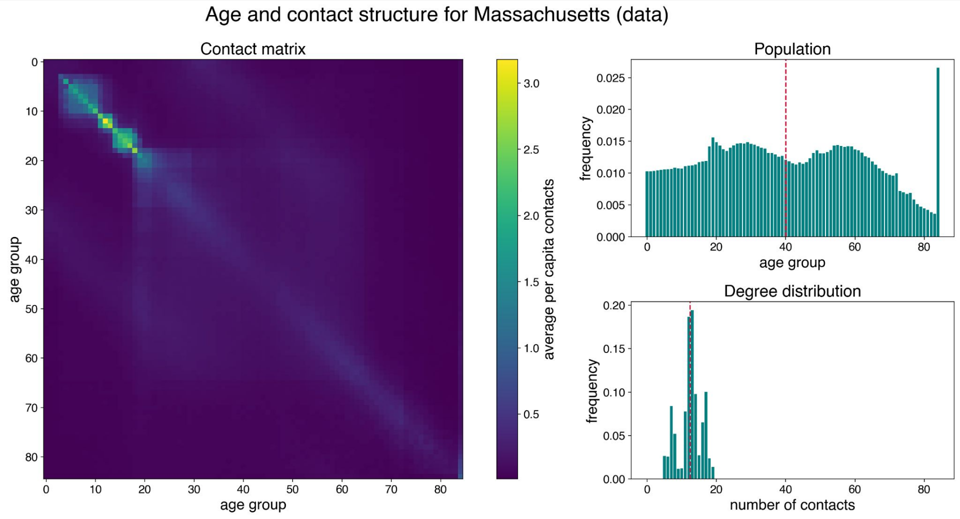
Within a given family of curves characterized by $n_A(0)$ value, those with lower θ values exhibit the most pronounced changes, suggesting that increasing n_Z effectively raises the system's activation threshold.

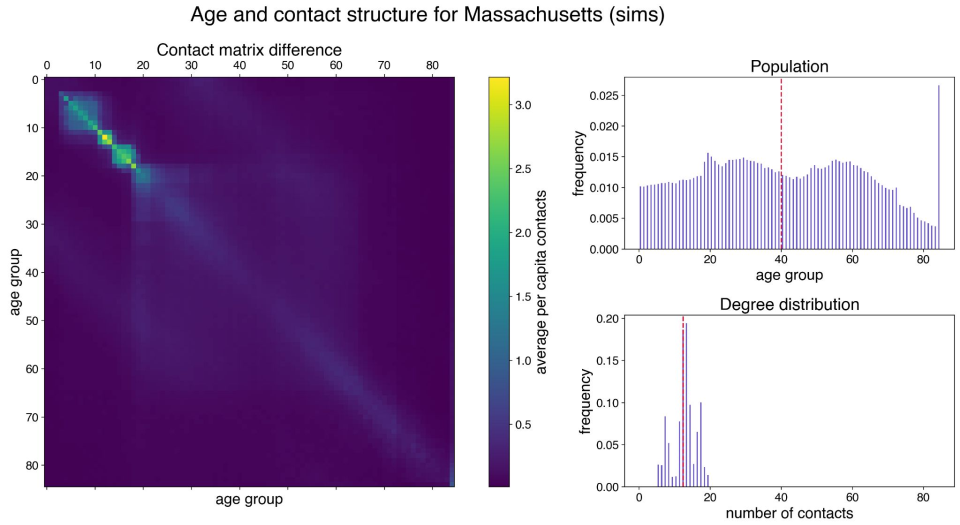
Convergence within a curve family occurs when the condition $n_A(0) + n_Z = 1$ is fulfilled.

W#2: Threshold. Vaccination curves (Data-driven-multilayer)

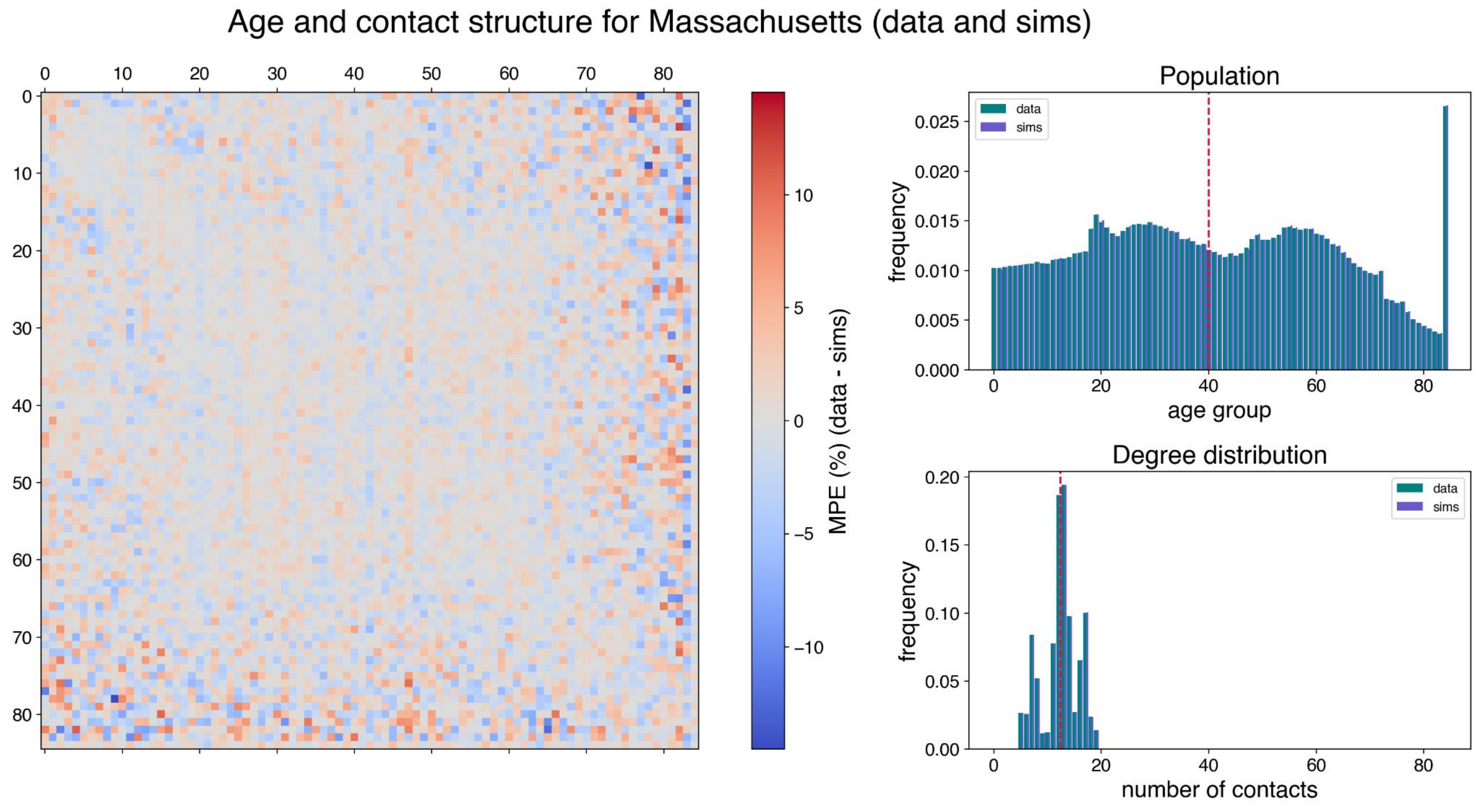


W#2: Threshold. Massachusetts (data & sims)

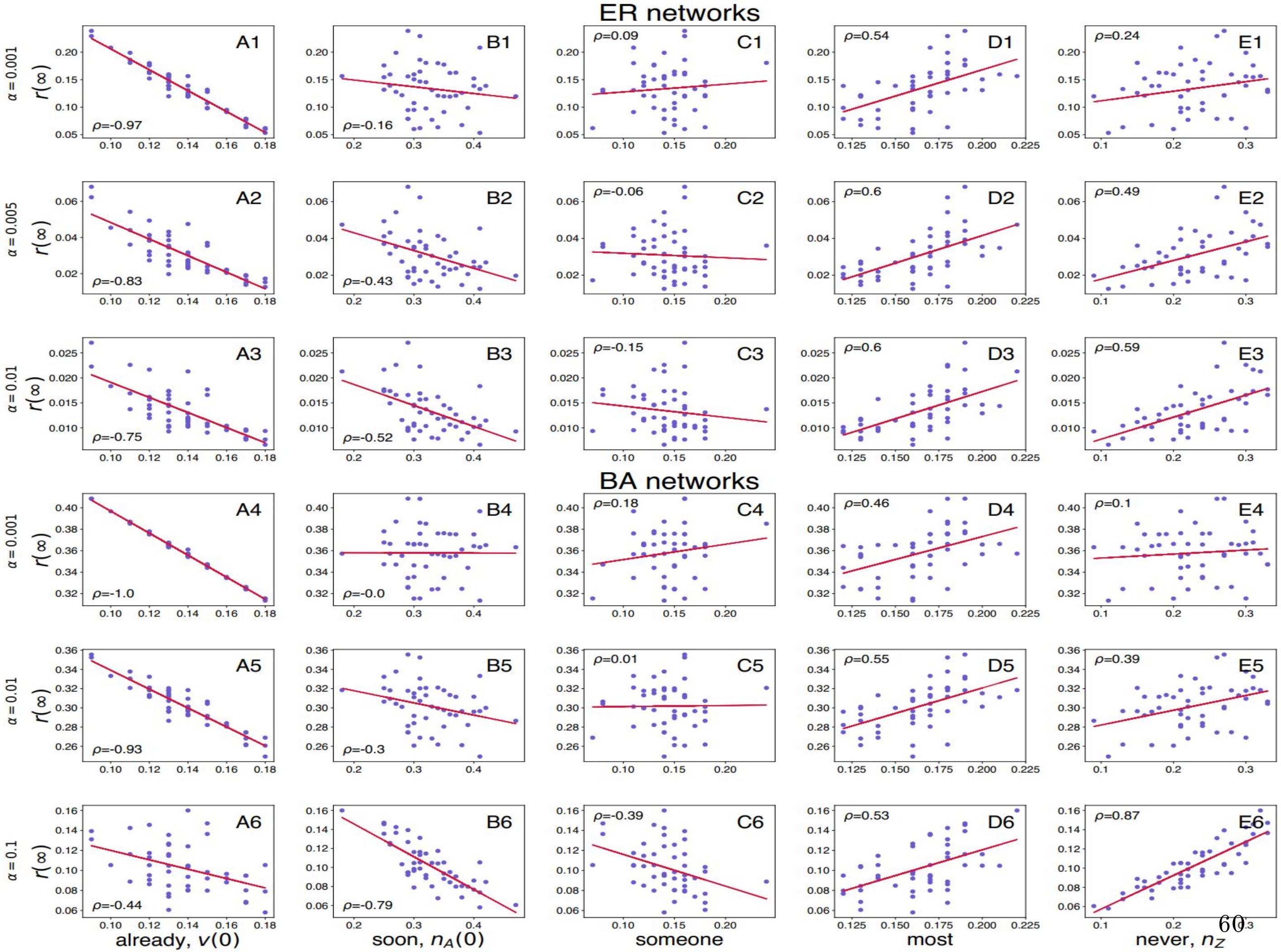




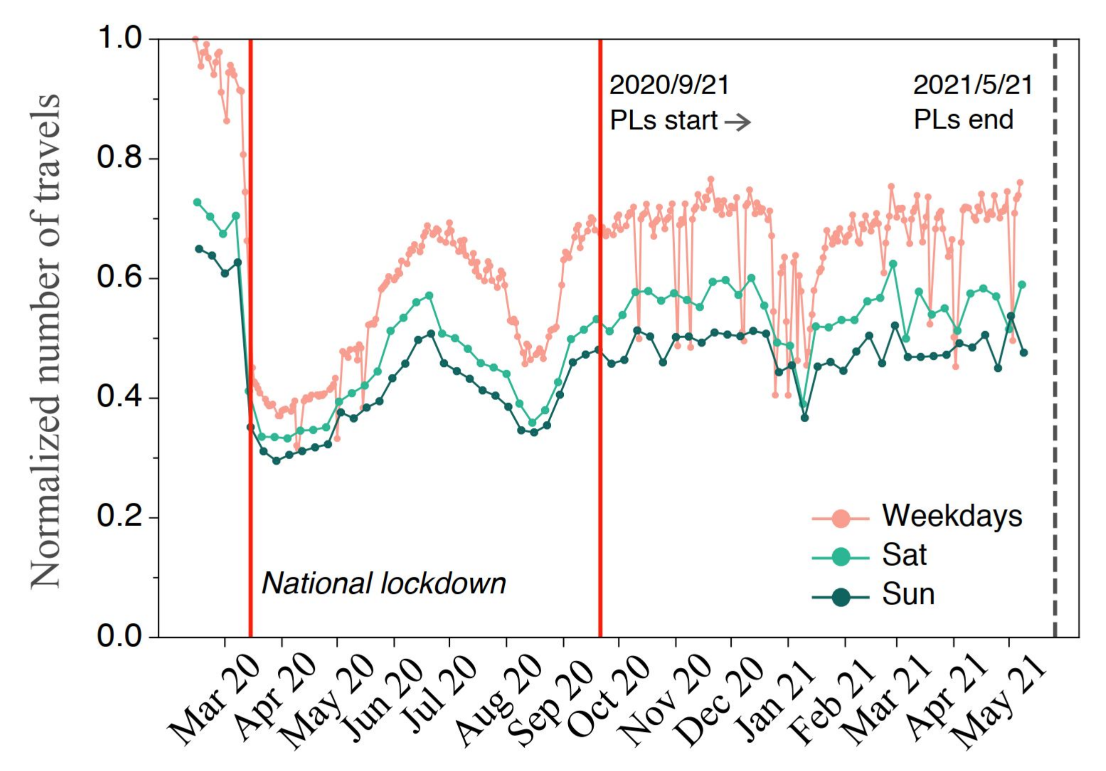
W#2: Threshold. Massachusetts (data & sims comp.)



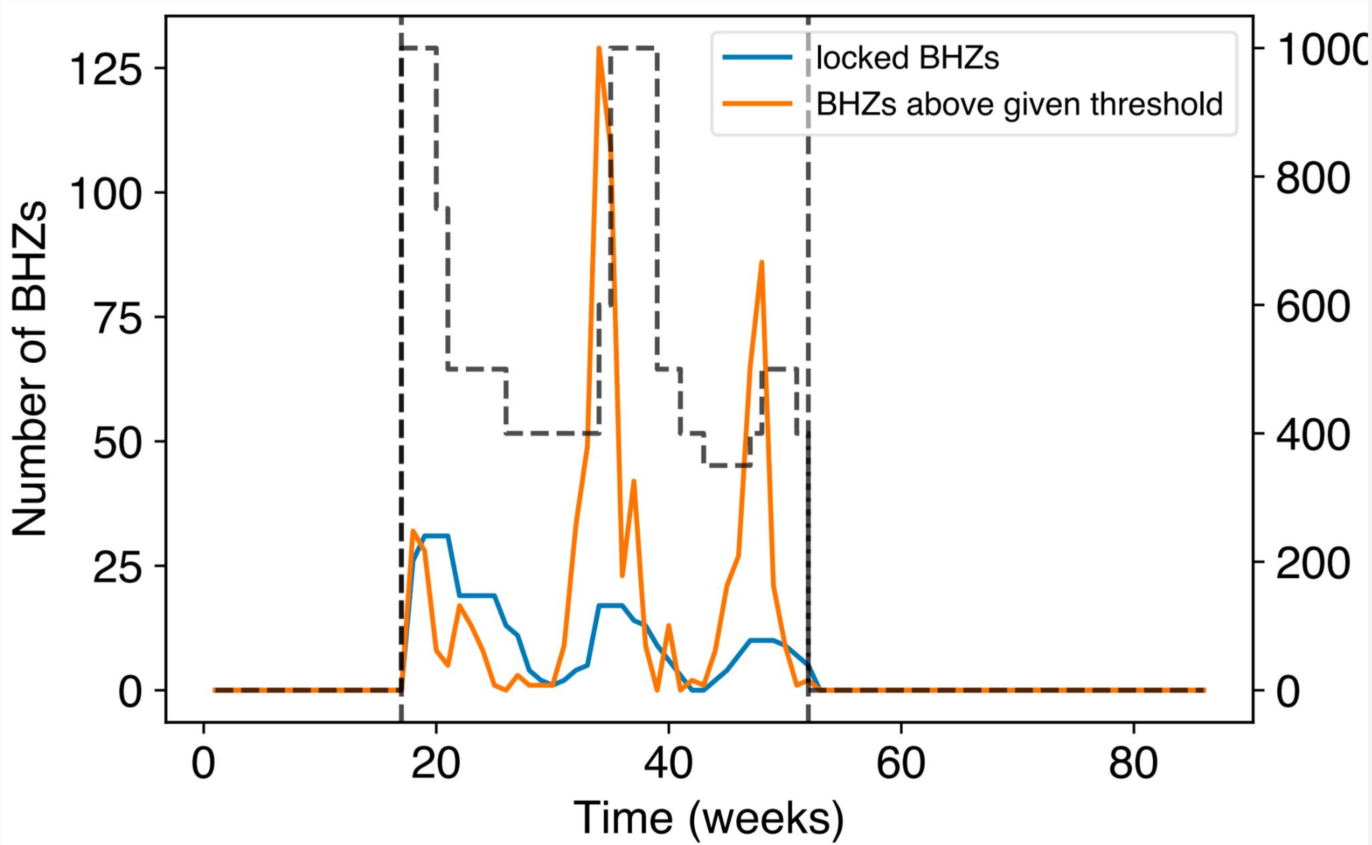
W#2: Threshold. Prevalence & attitudes



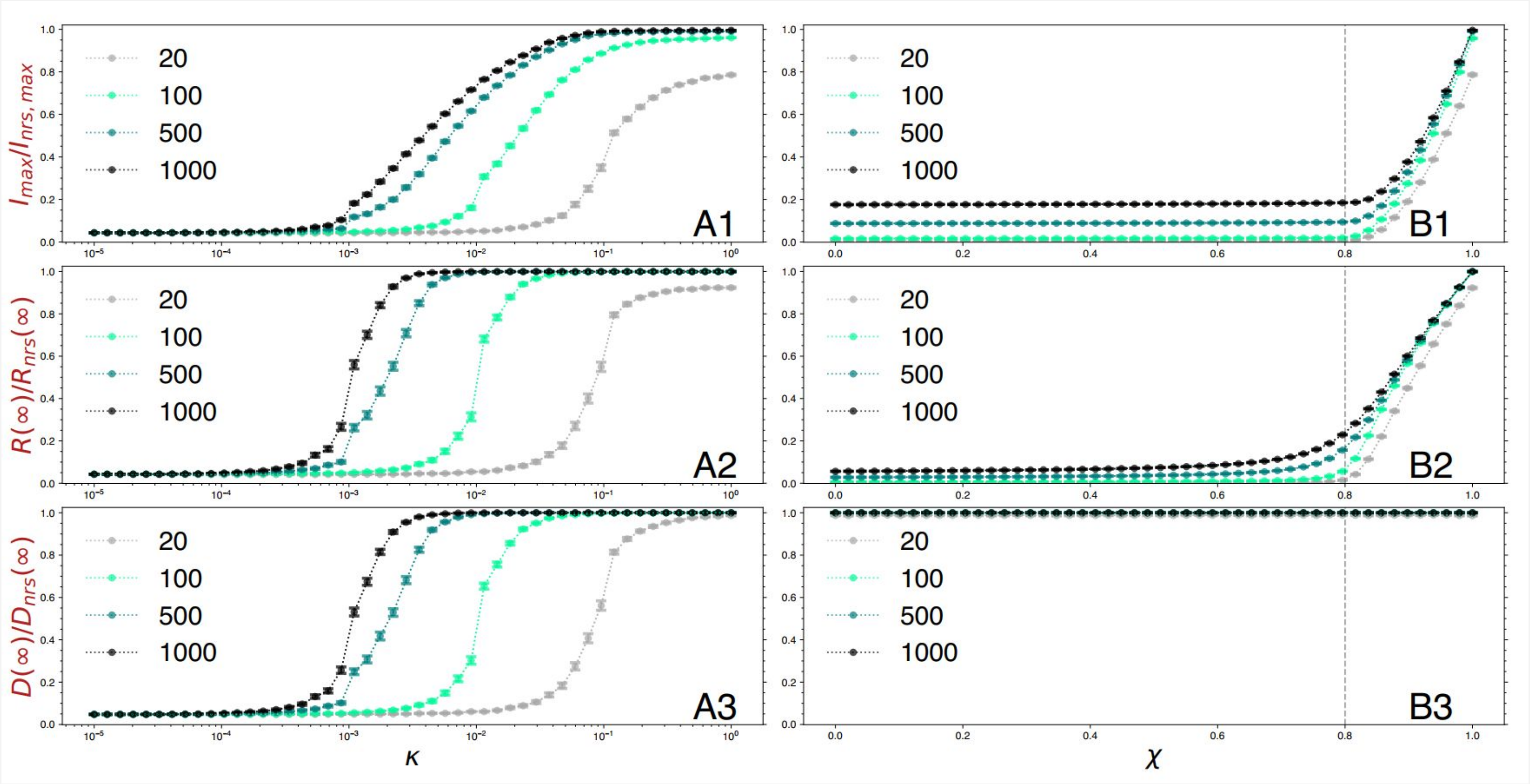
W#3: Madrid. General mobility levels



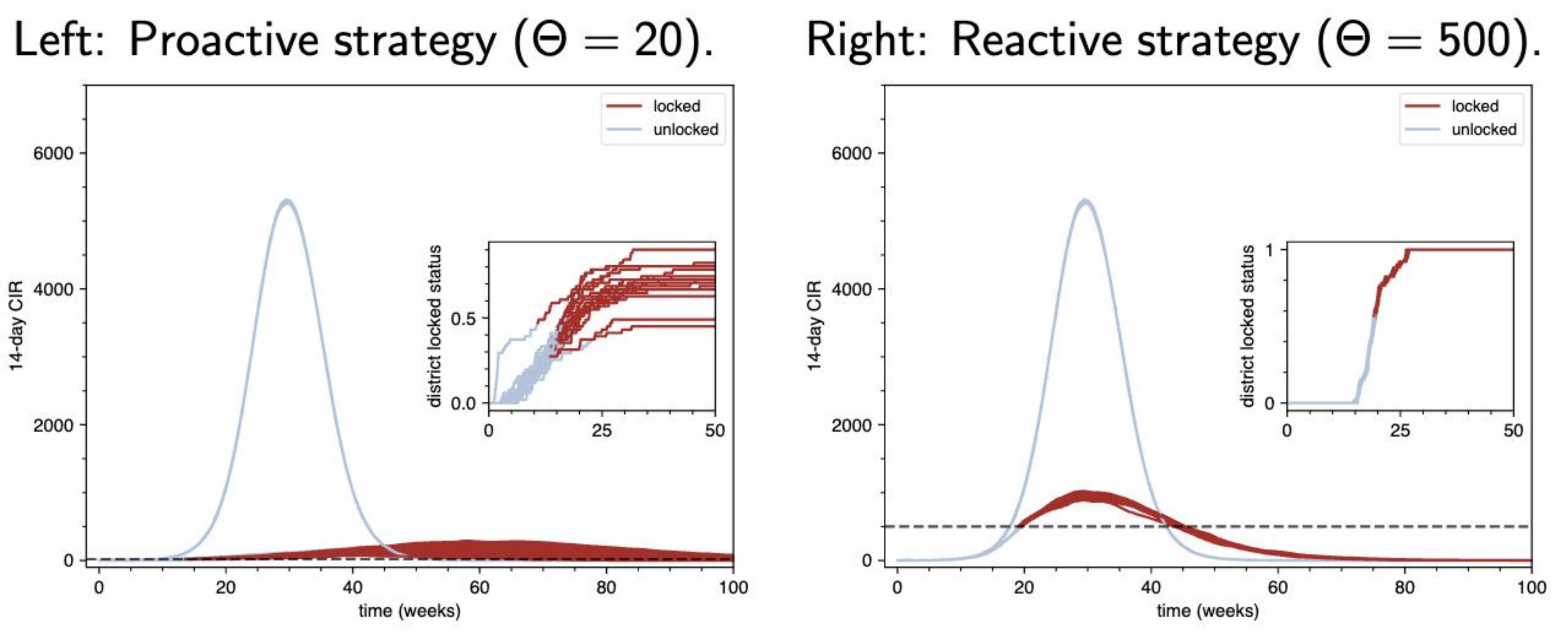
W#3: Madrid. BHZs under perimeter lockdown



W#3: Madrid. Fixed chi=1 & kappa=1 curves

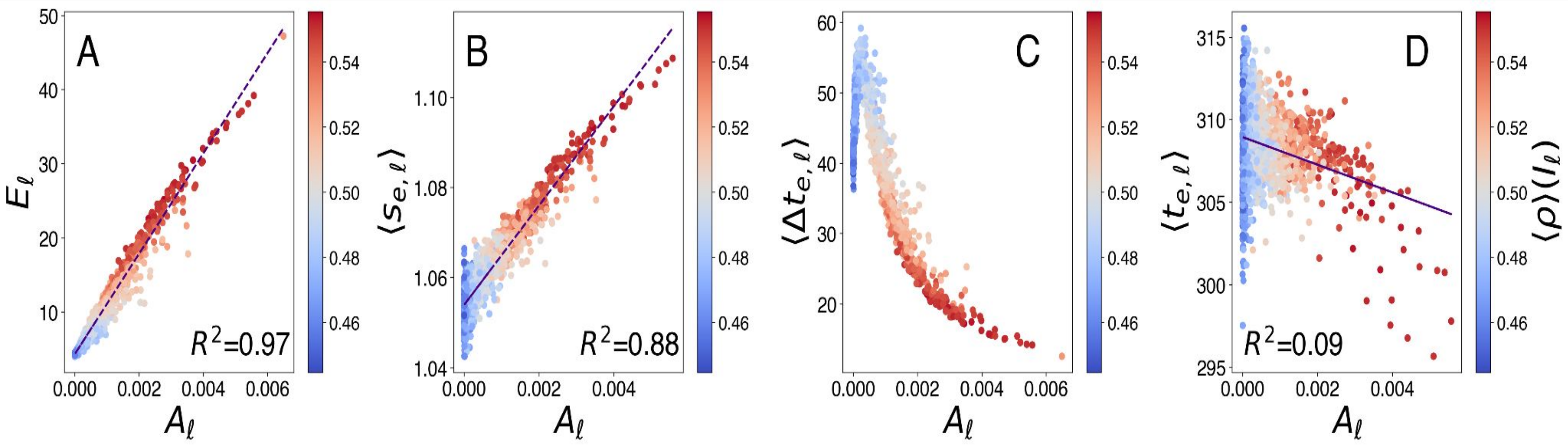


W#3: Madrid. Time series synchronization



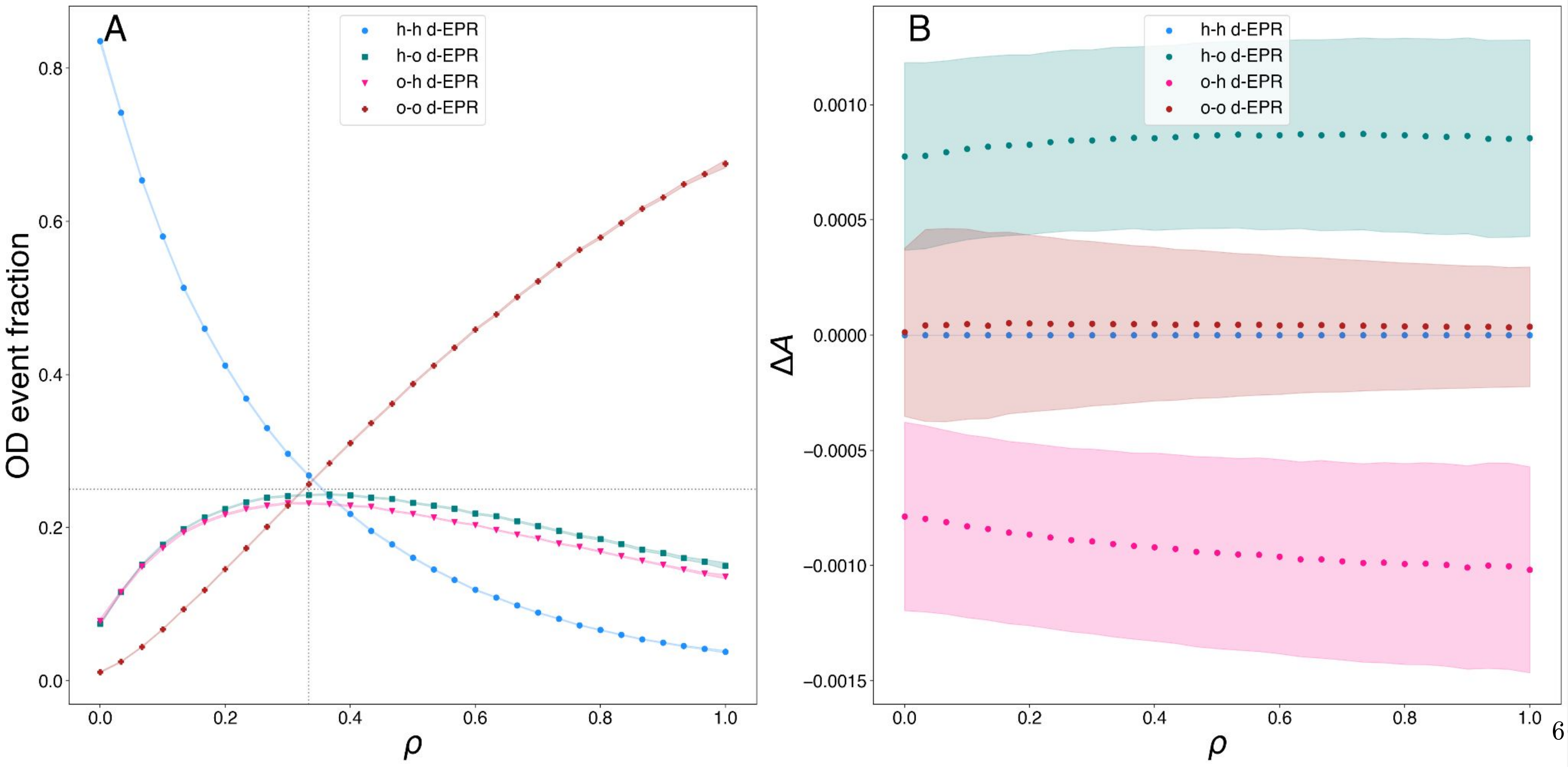
Doing something is better than nothing but... Local outbreaks are highly synchronized.

W#4: Explorers. Contagion events & attractiveness

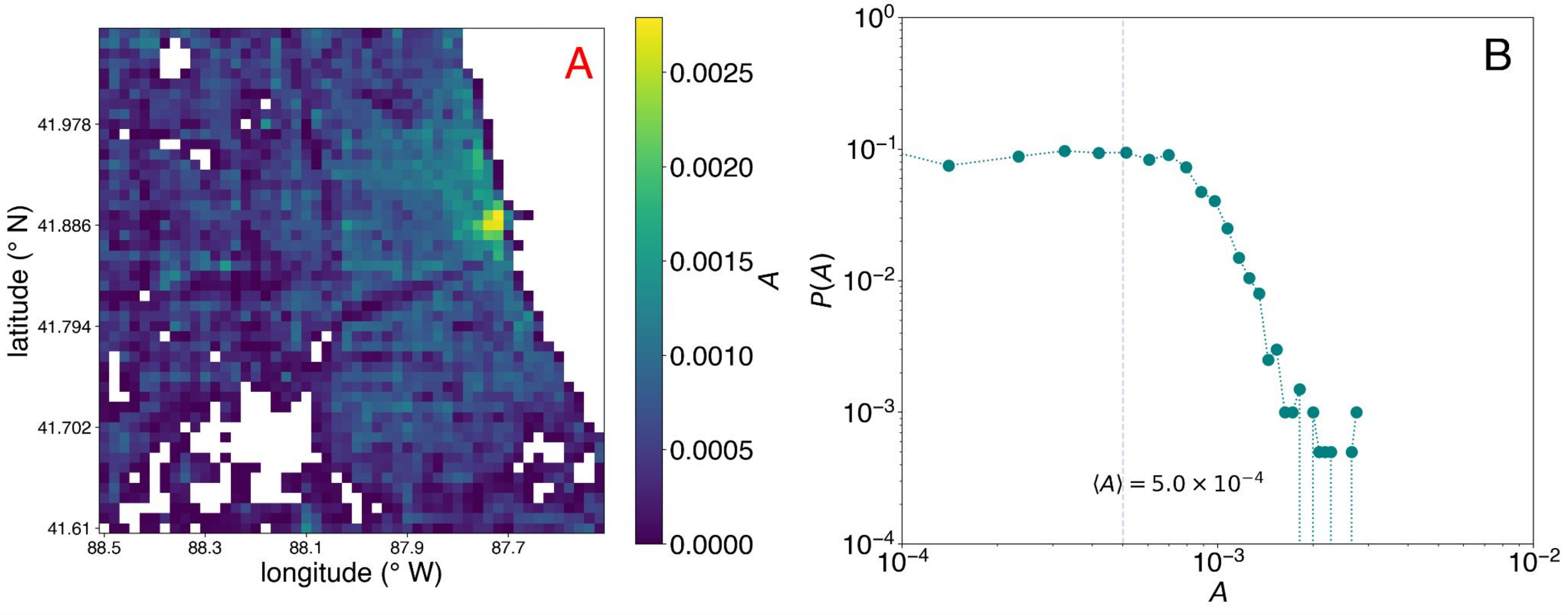


- [A & B] More attractiveness → more events, but size differences are not a thing. (?)
- [C] More attractiveness → Shorter inter-event times. But with also less attractiveness!
- Top A locations sustain the epidemic in time, bottom A locations show a short-lived outbreak
- [D] High synchronization

W#4: Explorers. Spreading the disease (from origin to destination)

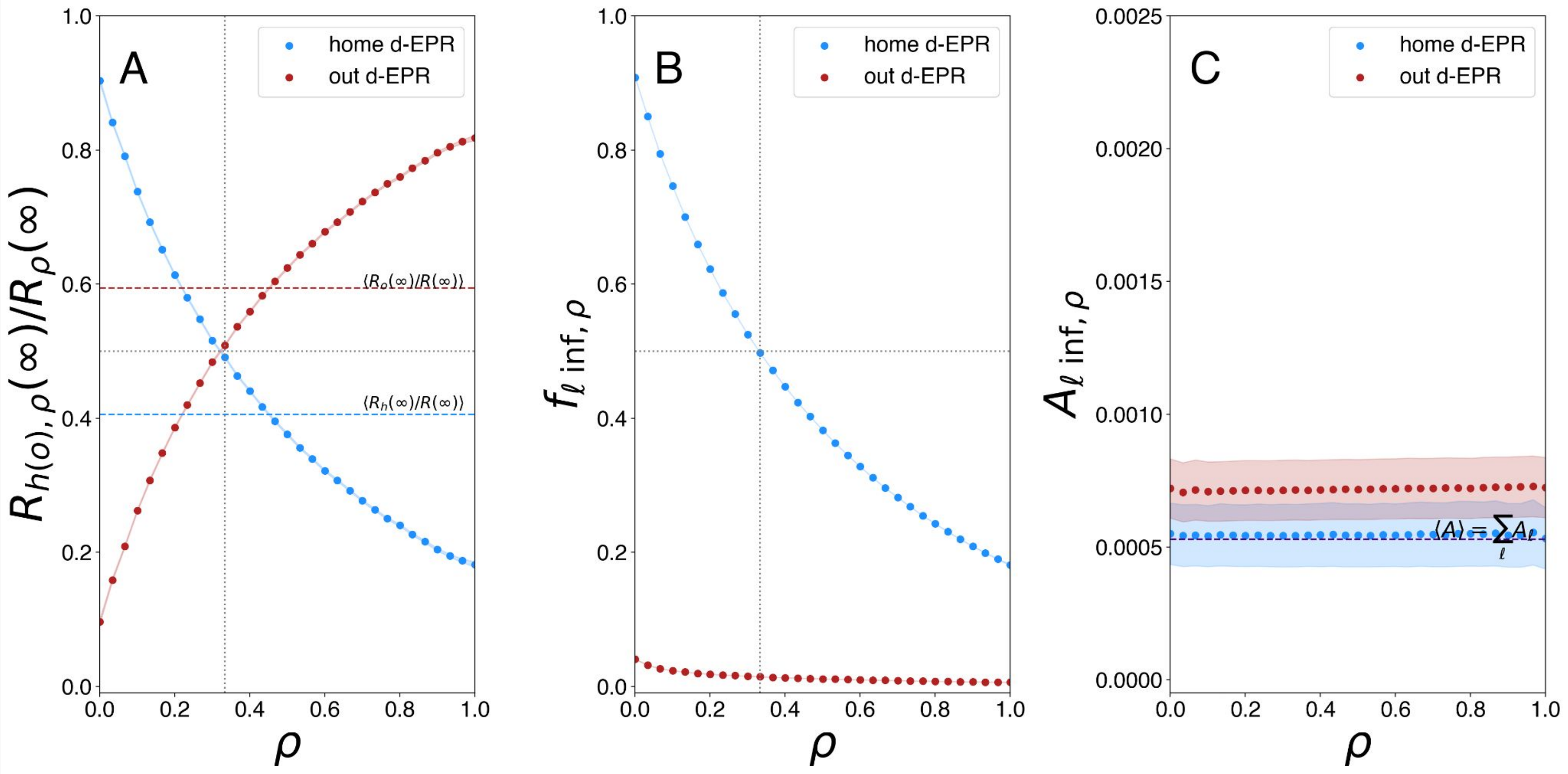


W#4: Chicago



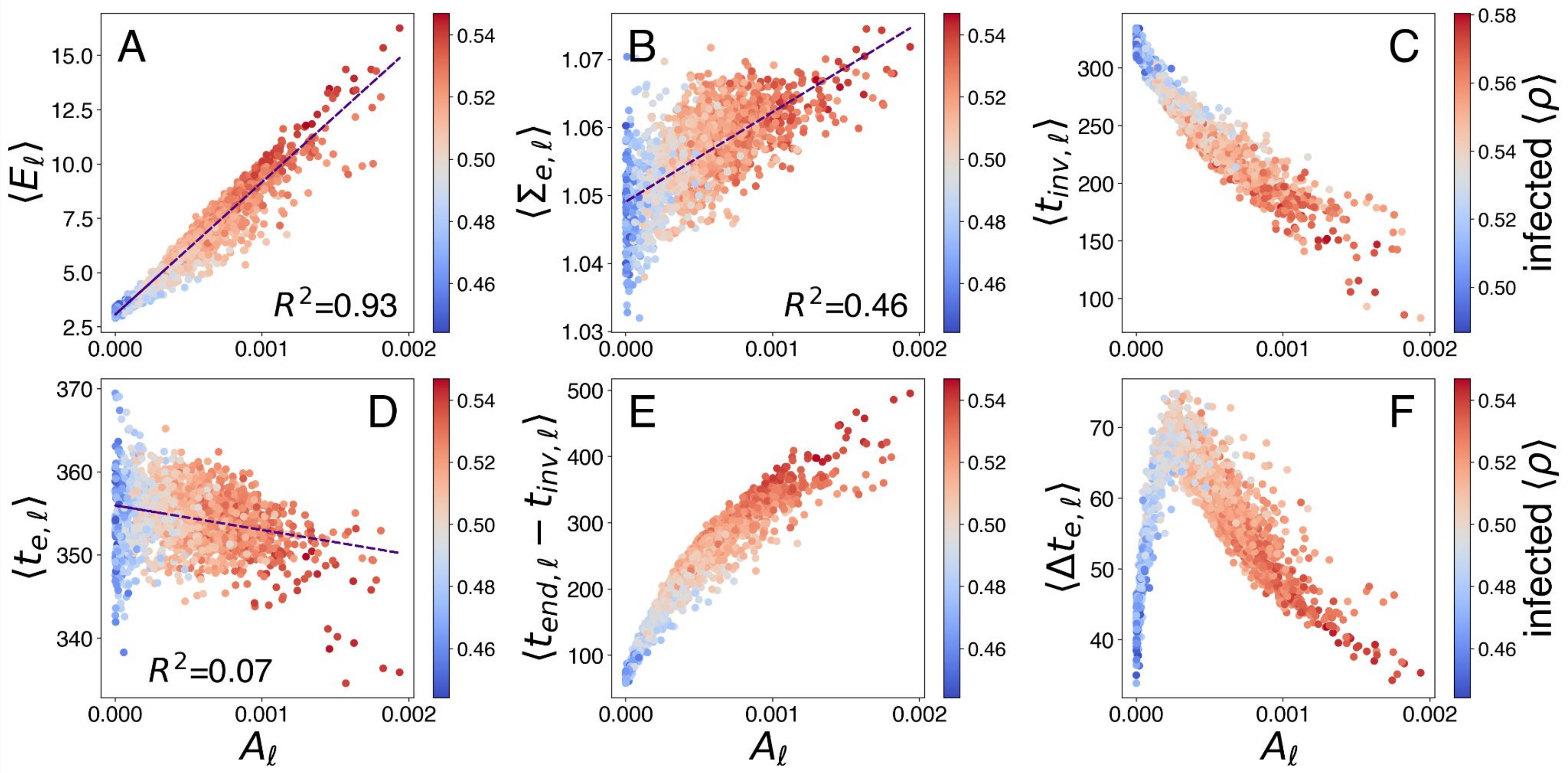
A
41.978
41.886
41.794
41.702
41.61
latitude (° N)
88.5
88.3
88.1
87.9
87.7
longitude (° W)
0.0025
0.0020
0.0015
0.0010
0.0005
0.0000
A
B
10^0
10^{-1}
10^{-2}
10^{-3}
10^{-4}
$P(A)$
$\langle A \rangle = 5.0 \times 10^{-4}$
10^{-4}
10^{-3}
10^{-2}
A

W#4: Chicago



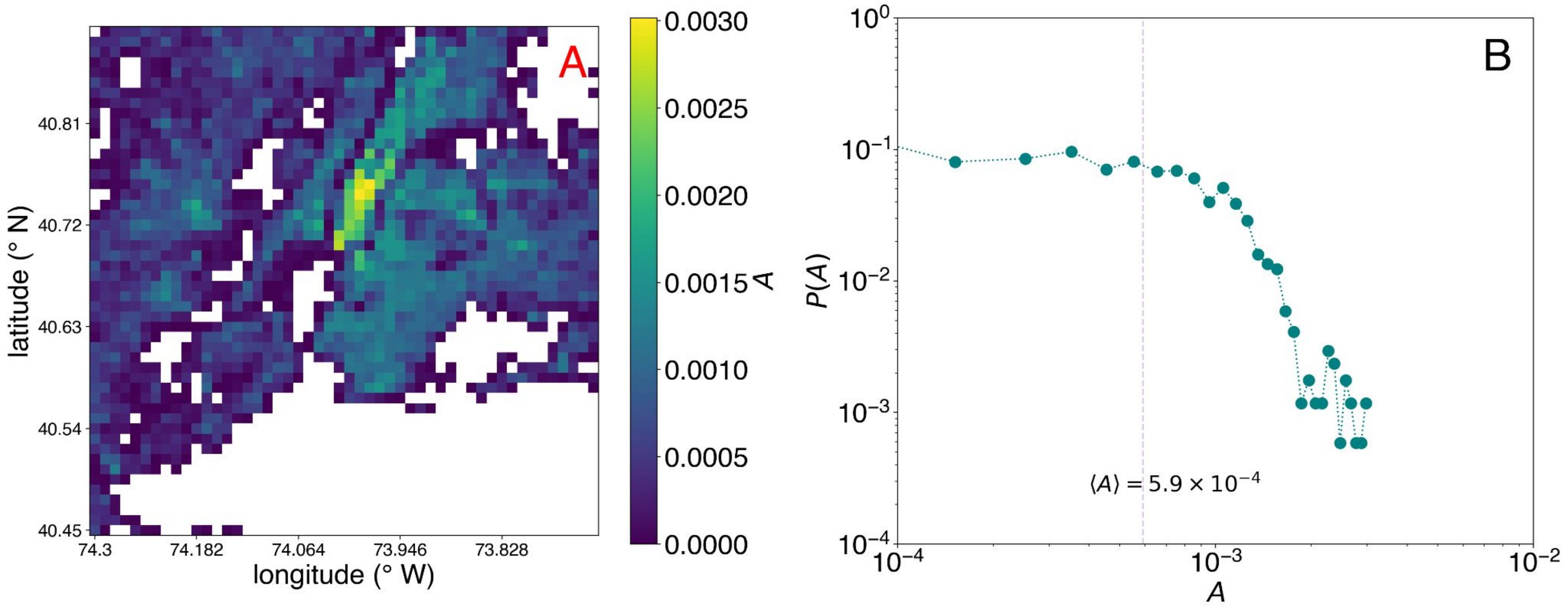
A
B
C
home d-EPR
out d-EPR
$\langle R_o(\infty)/R(\infty)\rangle$
$\langle R_h(\infty)/R(\infty)\rangle$
$R_{h(o),\rho}(\infty)/R_\rho(\infty)$
$f_{\ell\,\mathrm{inf},\rho}$
$A_{\ell\,\mathrm{inf},\rho}$
$\langle A\rangle = \sum_\ell A_\ell$
ρ

W#4: Chicago

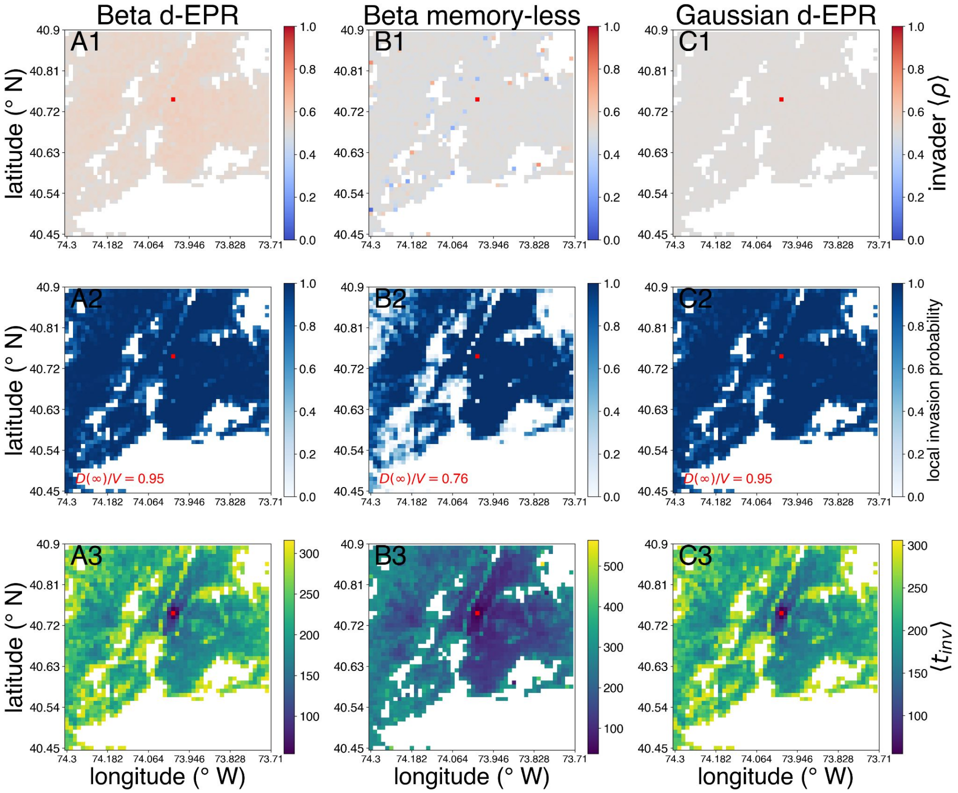


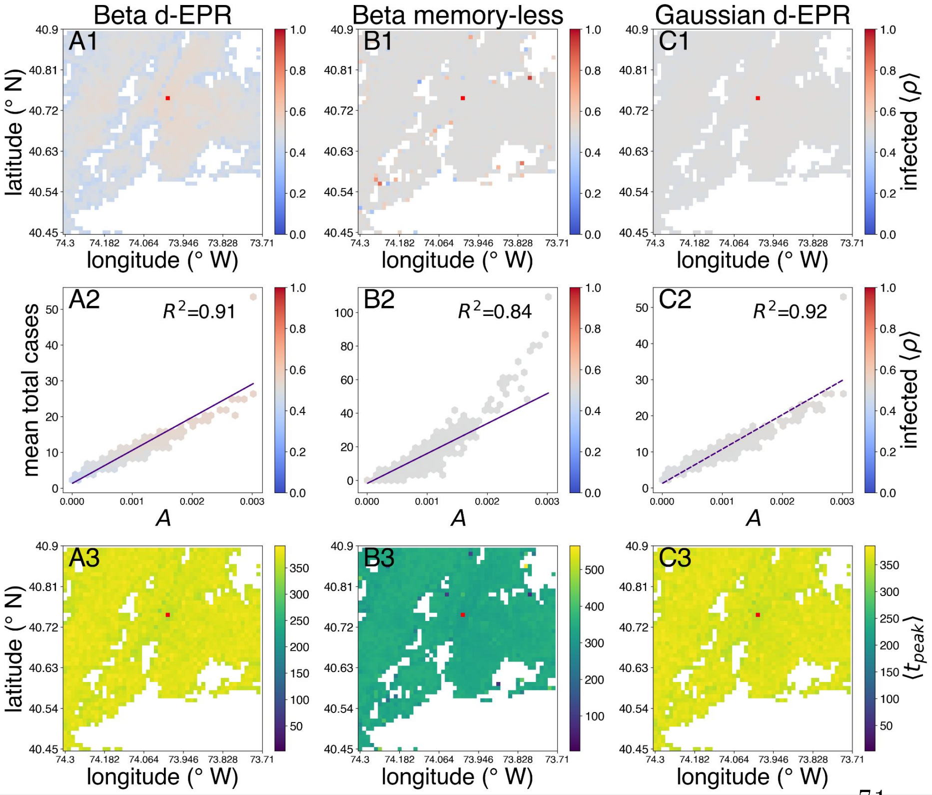
A
B
C
D
E
F
R^2=0.93
R^2=0.46
R^2=0.07
$\langle E_\ell \rangle$
$\langle \Sigma_{e,\ell} \rangle$
$\langle t_{inv,\ell} \rangle$
$\langle t_{e,\ell} \rangle$
$\langle t_{end,\ell} - t_{inv,\ell} \rangle$
$\langle \Delta t_{e,\ell} \rangle$
A_ℓ
infected $\langle \rho \rangle$

W#4: New York City

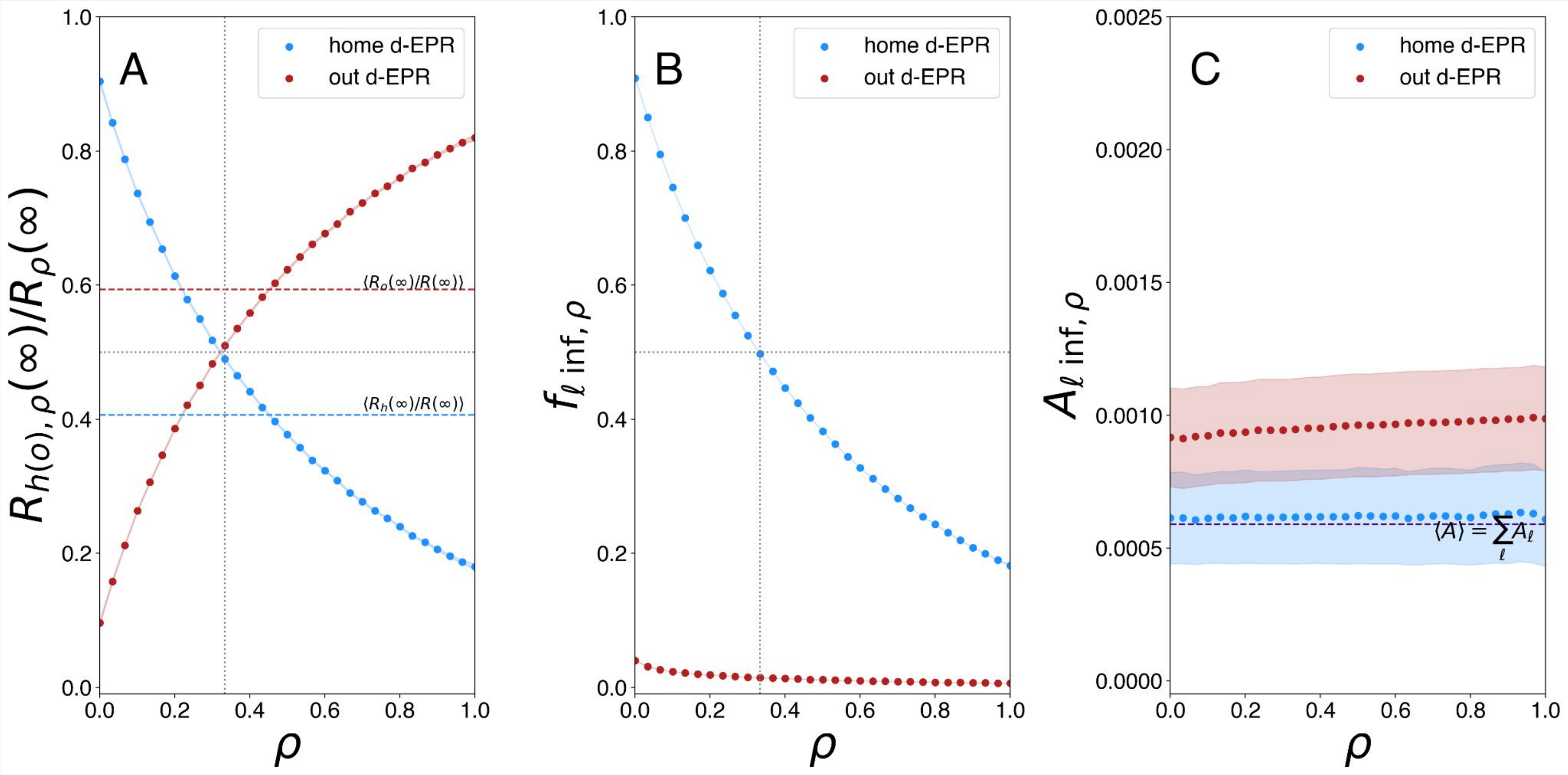


W#4: New York City invasion & infection maps

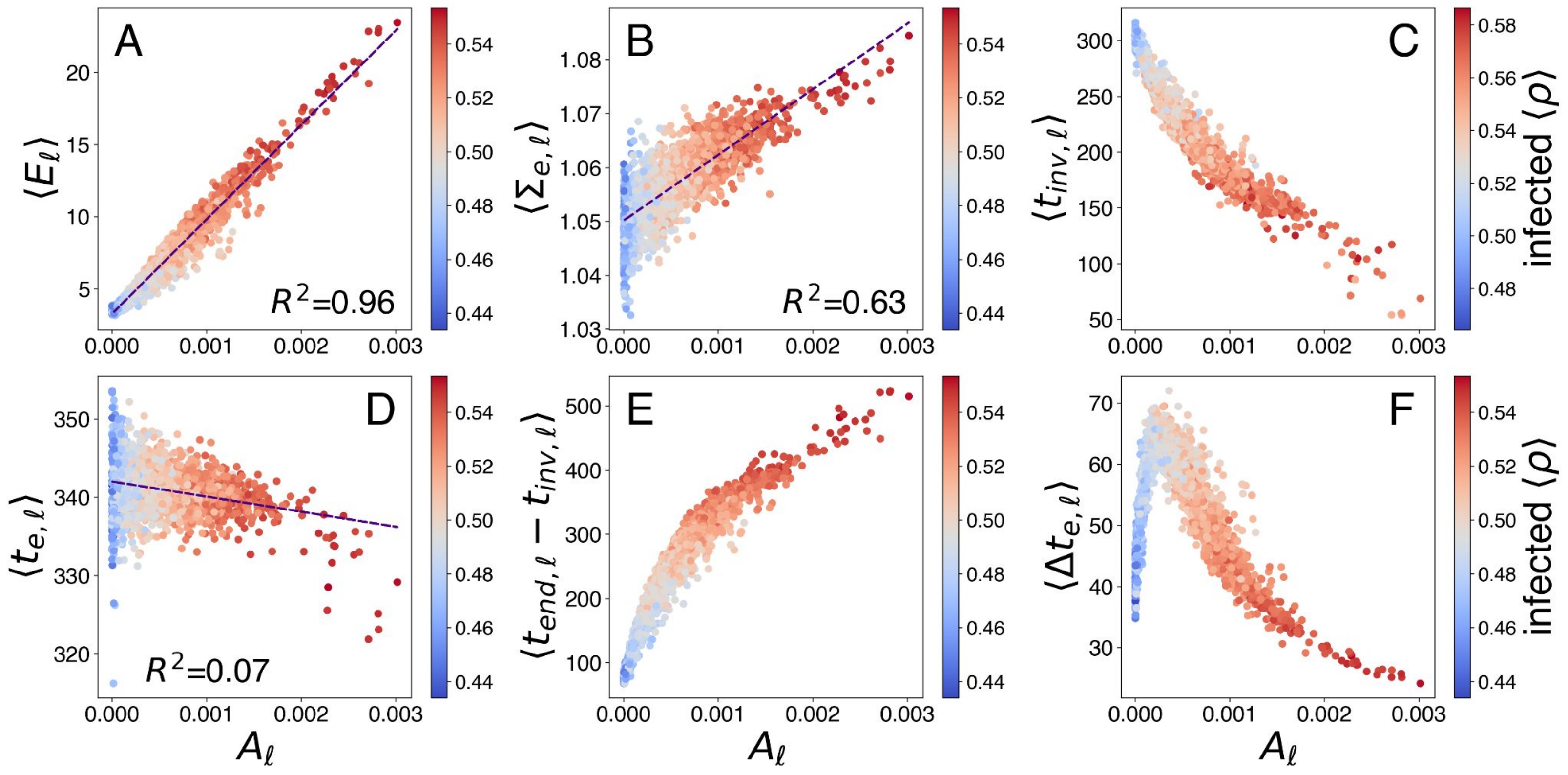




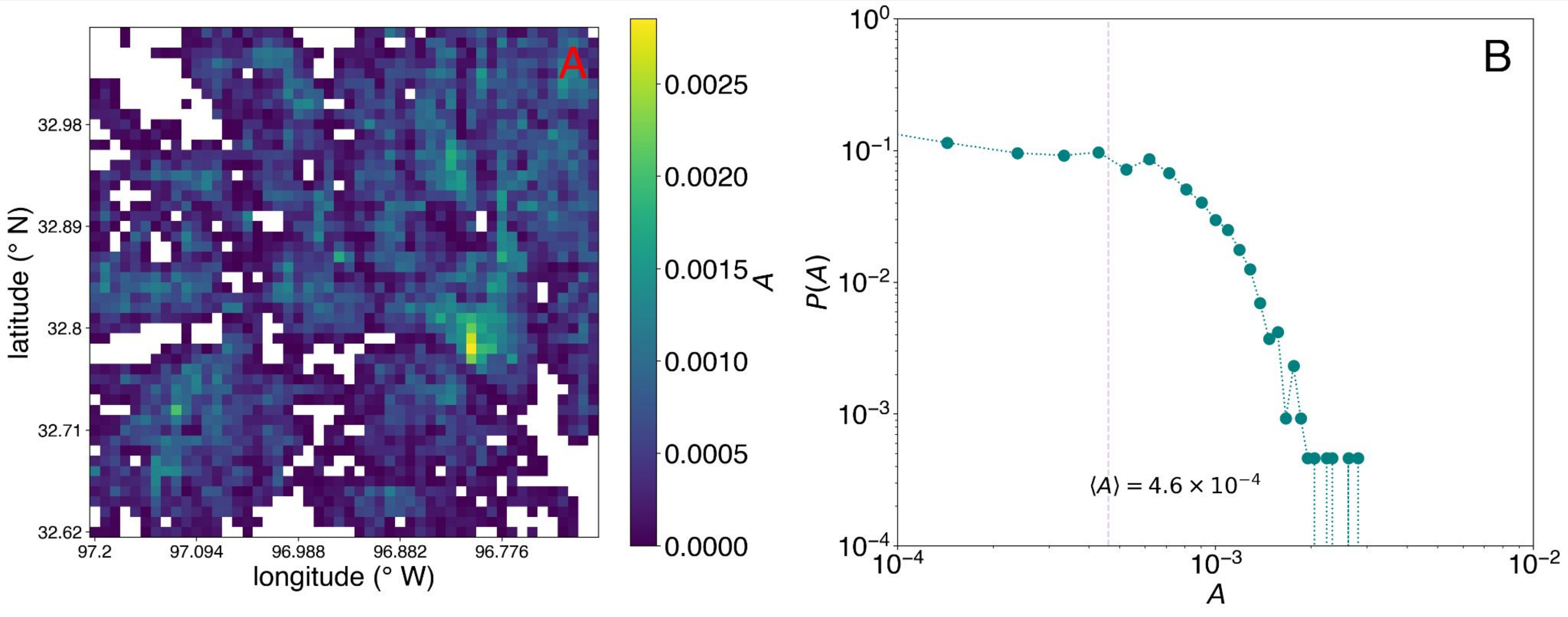
W#4: New York City



W#4: New York City

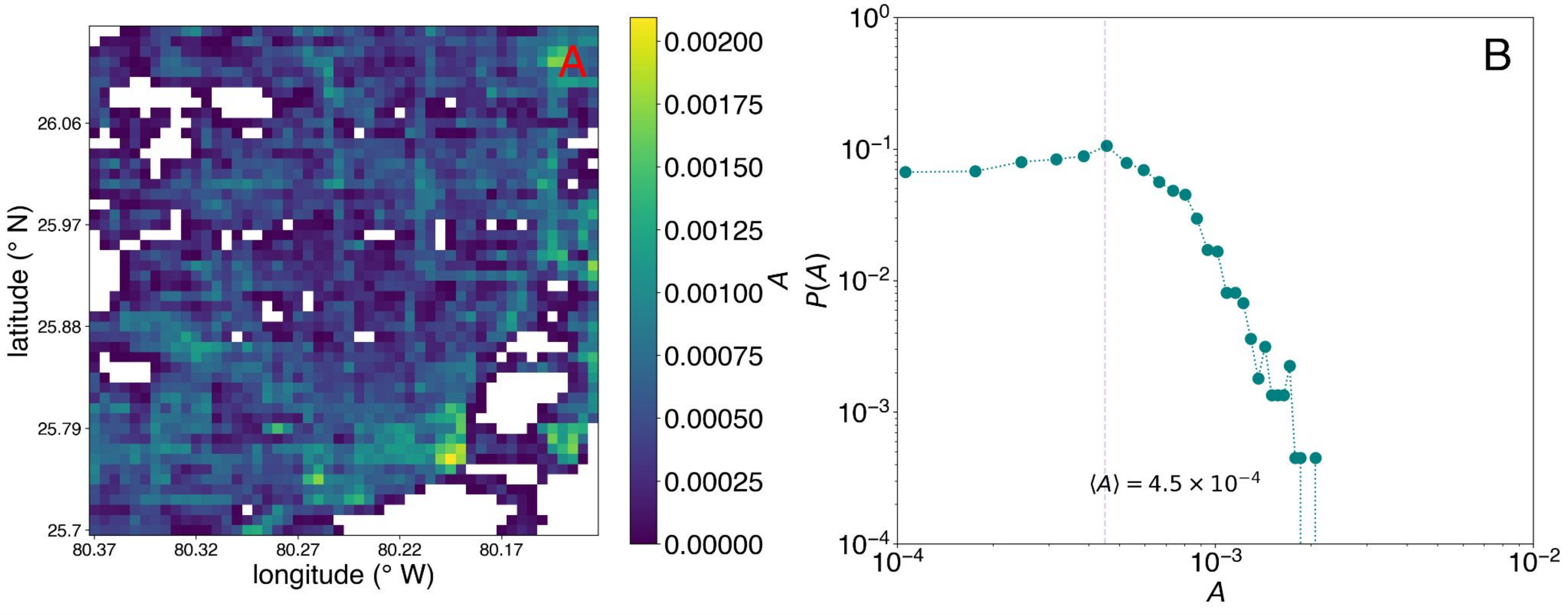


W#4: Dallas

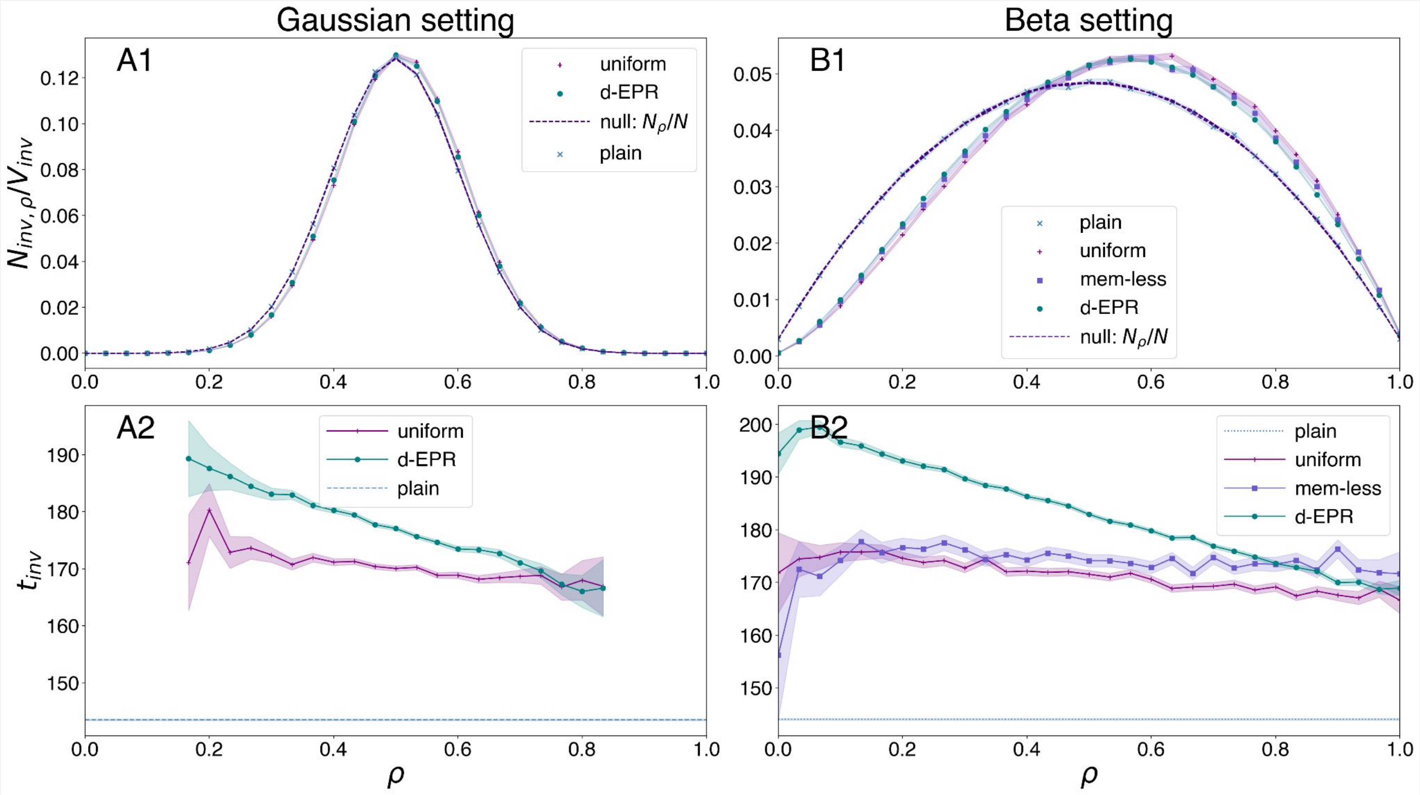


A
0.0025
0.0020
0.0015
0.0010
0.0005
0.0000
A
32.98
32.89
32.8
32.71
32.62
latitude (° N)
97.2
97.094
96.988
96.882
96.776
longitude (° W)
B
10^0
10^{-1}
10^{-2}
10^{-3}
10^{-4}
P(A)
$\langle A \rangle = 4.6 \times 10^{-4}$
10^{-4}
10^{-3}
10^{-2}
A

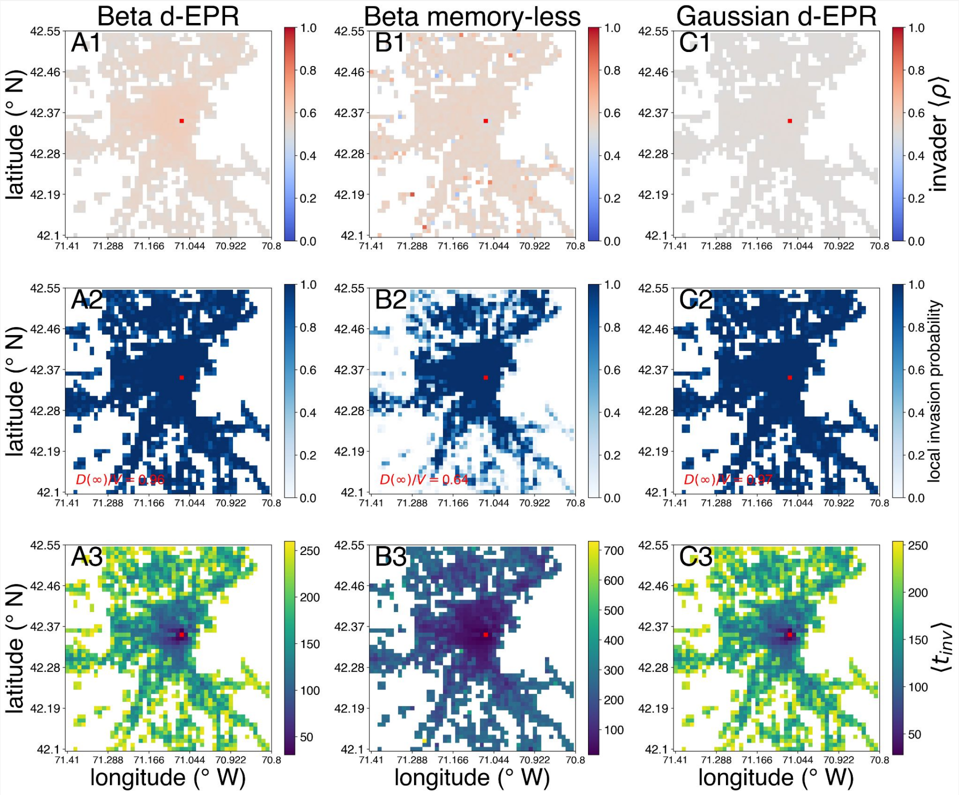
W#4: Miami


A
B
26.06
25.97
25.88
25.79
25.7
latitude (° N)
80.37
80.32
80.27
80.22
80.17
longitude (° W)
0.00200
0.00175
0.00150
0.00125
0.00100
0.00075
0.00050
0.00025
0.00000
A
10^0
10^{-1}
10^{-2}
10^{-3}
10^{-4}
$P(A)$
10^{-4}
10^{-3}
10^{-2}
A
$\langle A \rangle = 4.5 \times 10^{-4}$

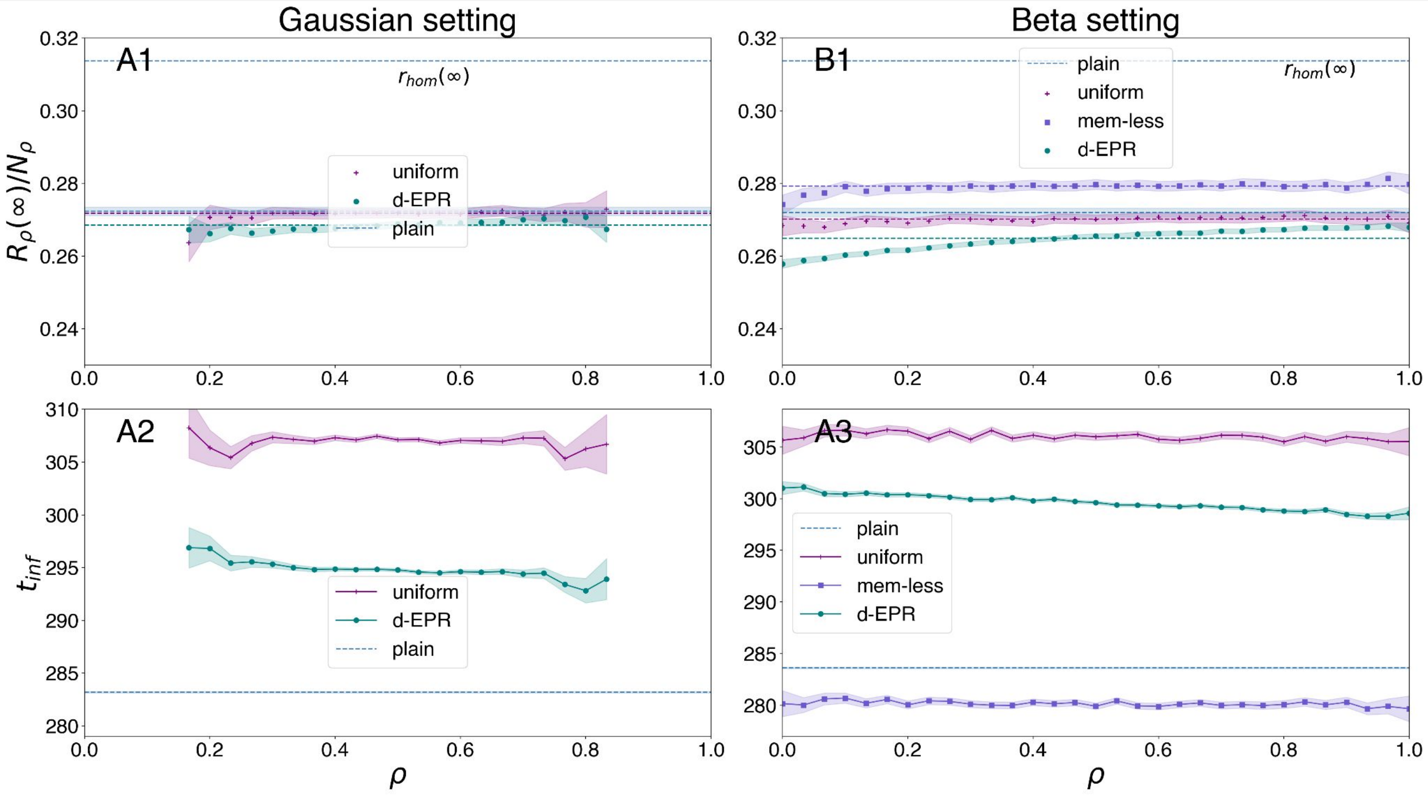
W#4: Boston (including heterogeneous mem-less)



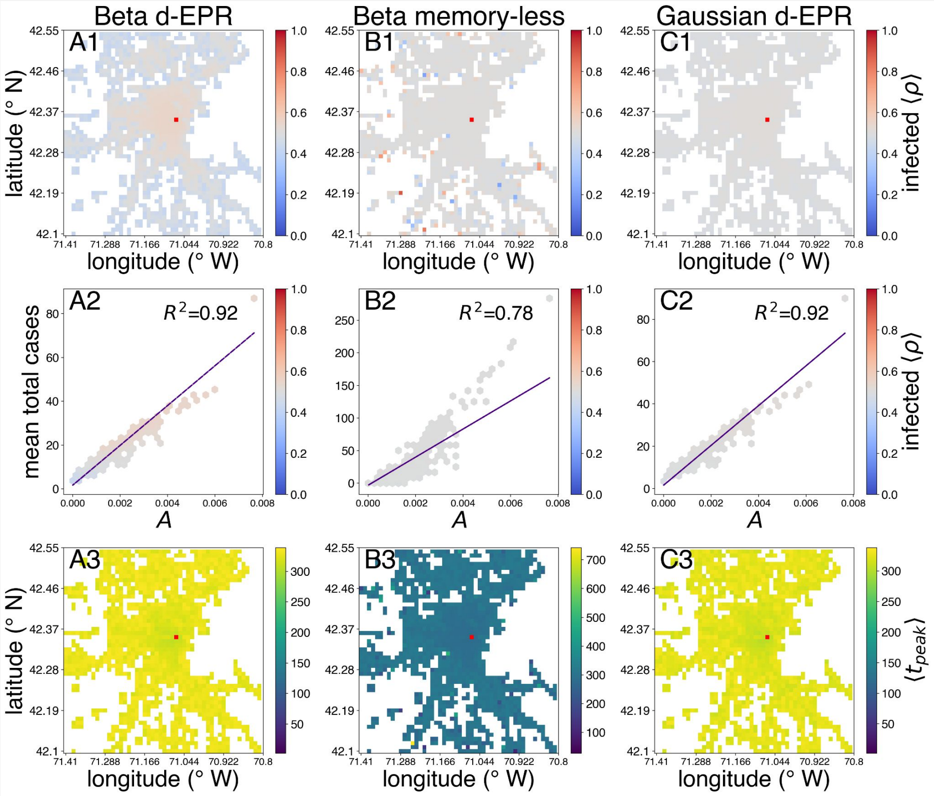
W#4: Boston (including heterogeneous mem-less)



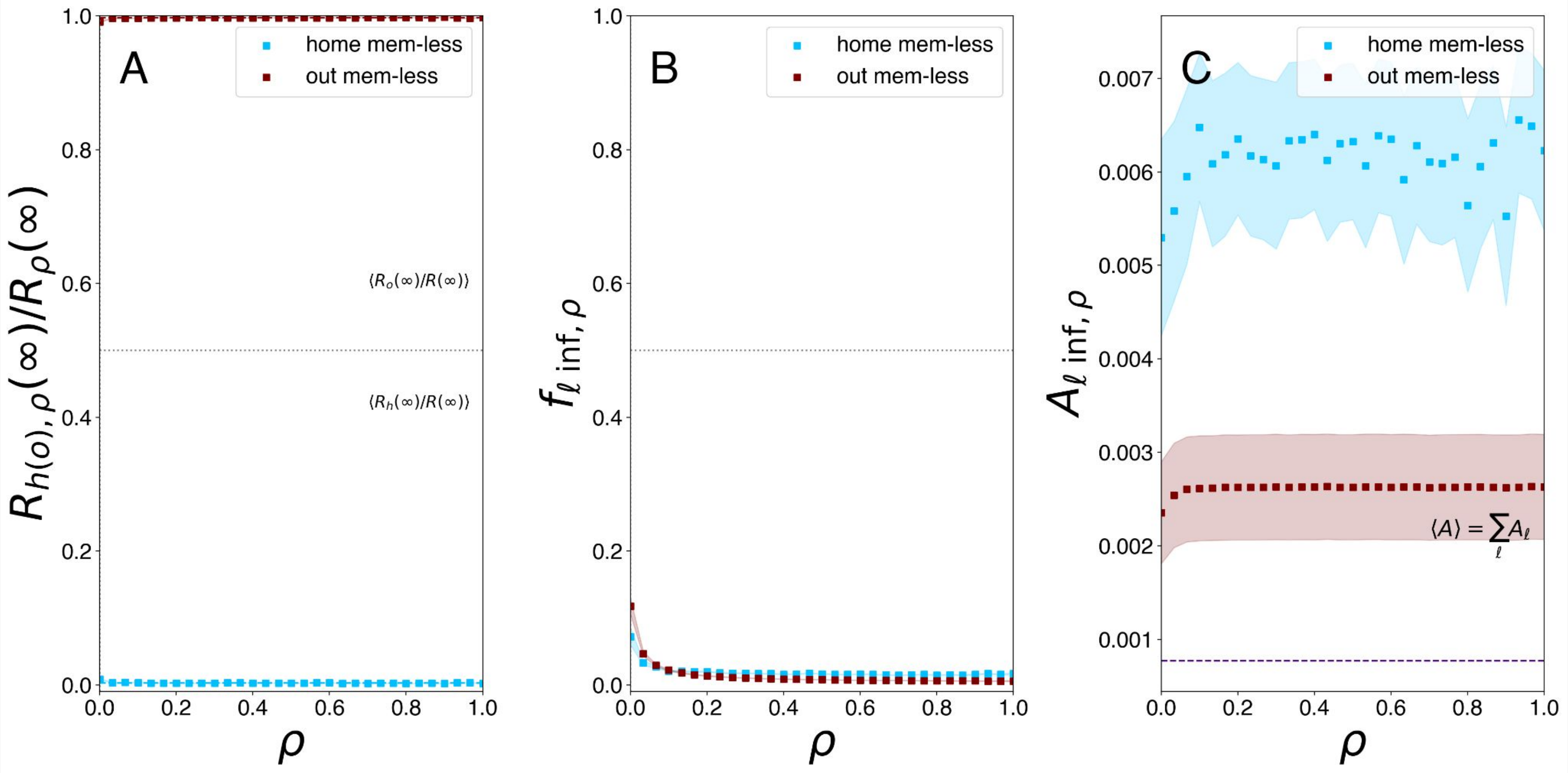
W#4: Boston (including heterogeneous mem-less)



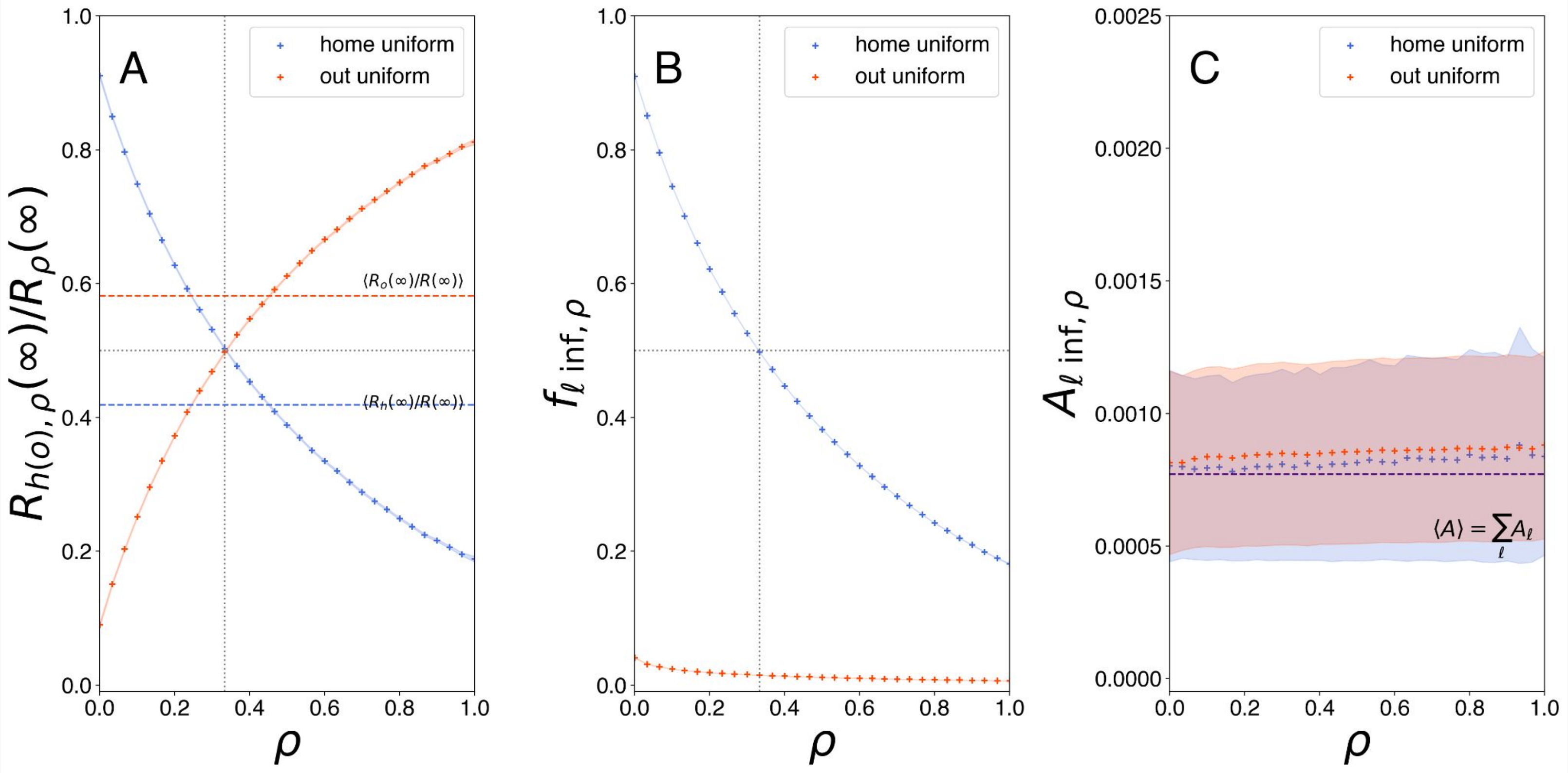
W#4: Boston (including heterogeneous mem-less)



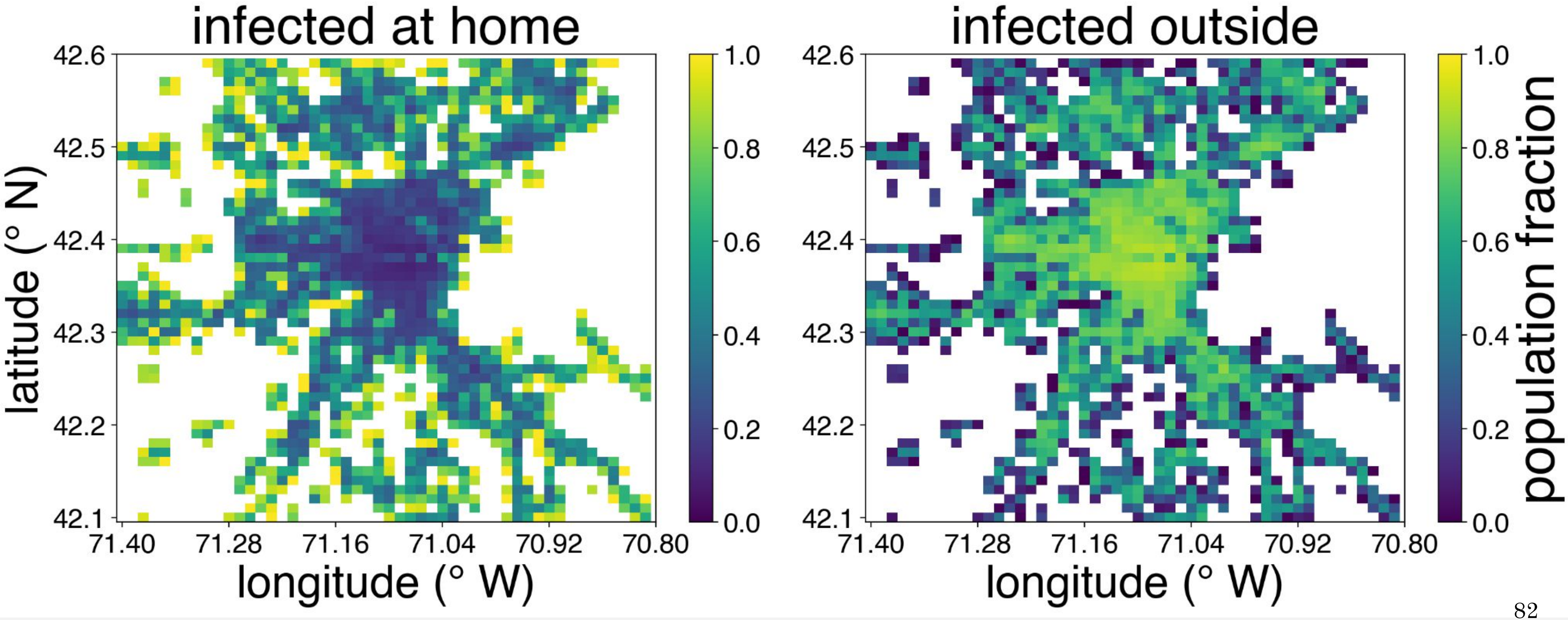
W#4: Boston (heterogeneous memory-less)



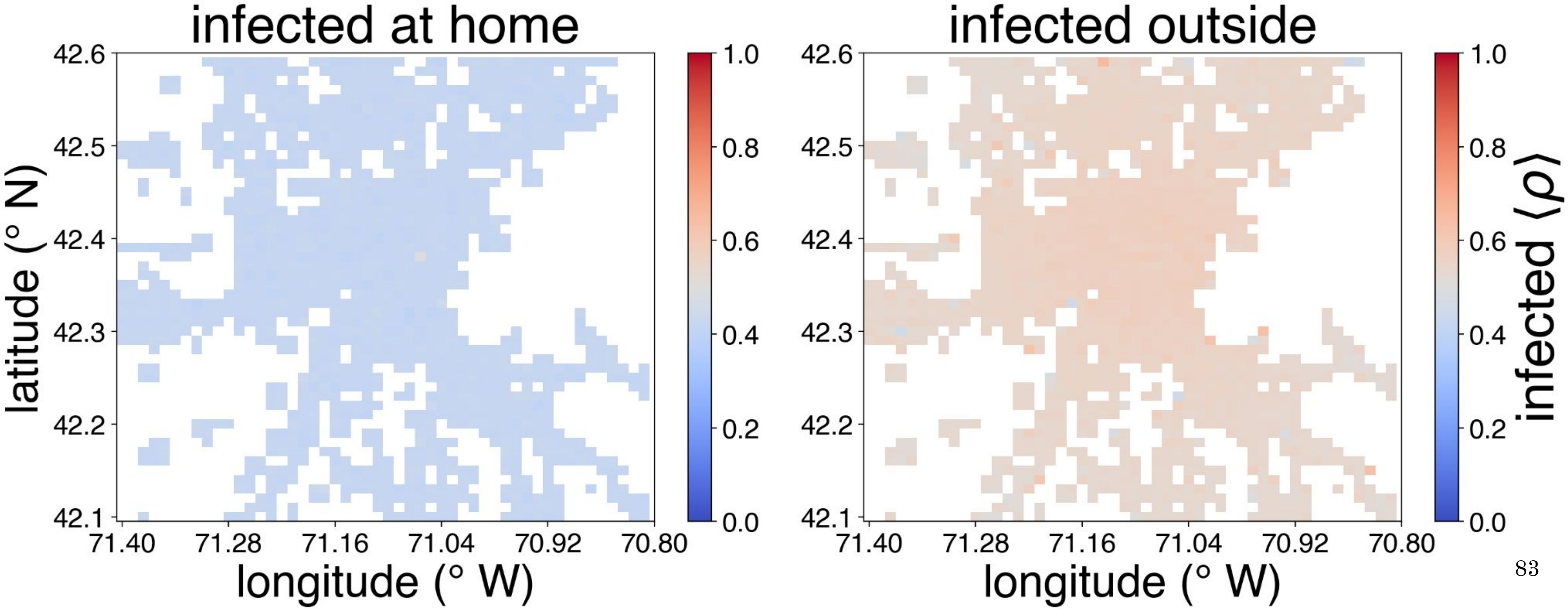
W#4: Boston (uniform)

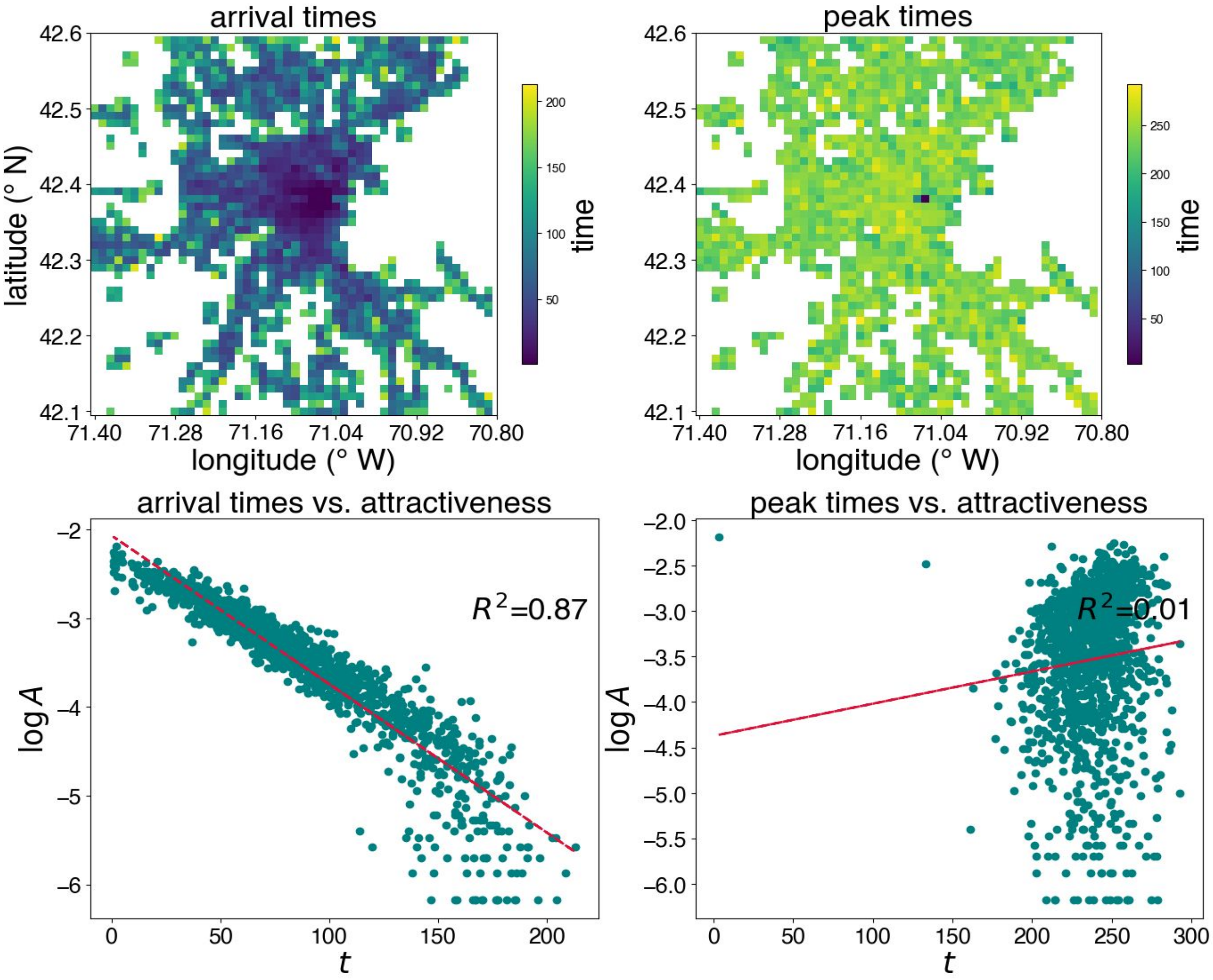


W#4: Explorers. Home/outside infection map



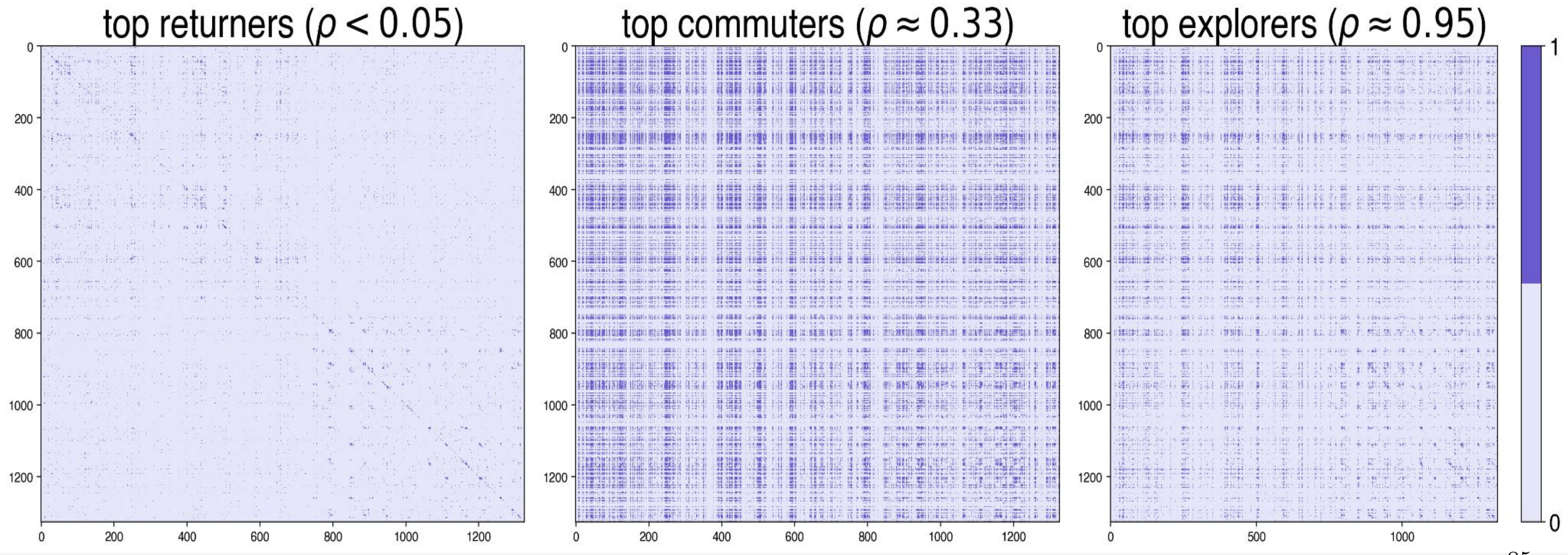
W#4: Home/outside infection map (II)



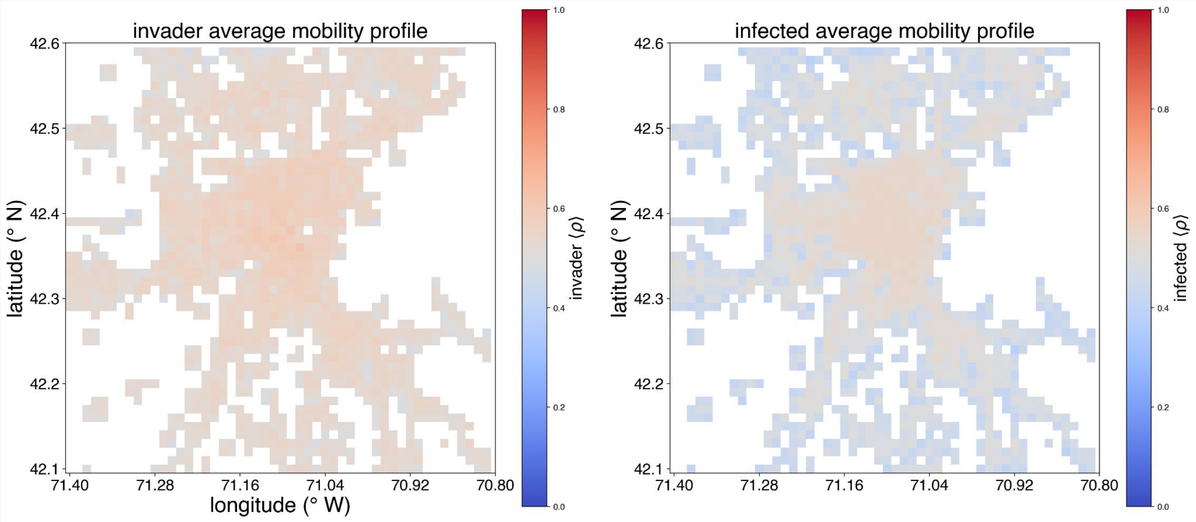


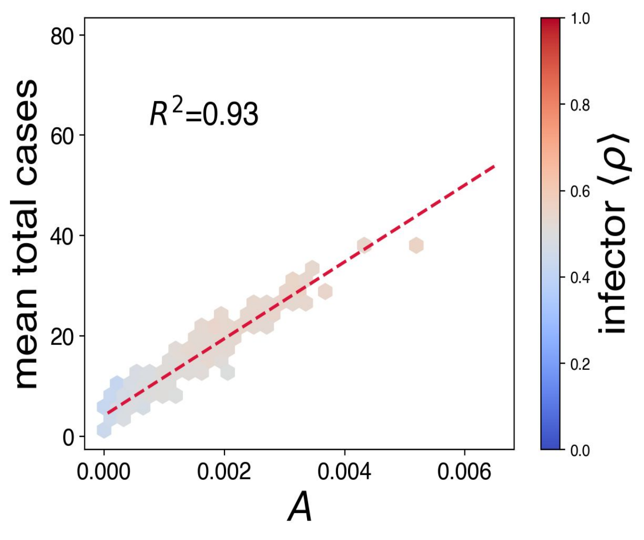
arrival times
peak times
latitude (° N)
longitude (° W)
time
arrival times vs. attractiveness
peak times vs. attractiveness
R^2=0.87
R^2=0.01
$\log A$
t

W#4: Explorers. Mobility adjacency matrices



Work #4: Explorers. What's the average invader/infected mobility profile per location?





Explorers absolutely dominate when bringing the disease to a new location.

In the most attractive locations, the typical infected tends to be an explorer (***ρ***>0.5).
In the least attractive locations, the typical infected tends to be a returner lower (***ρ***<0.5).

W#4: Explorers. Pappalardo et al. (2015)

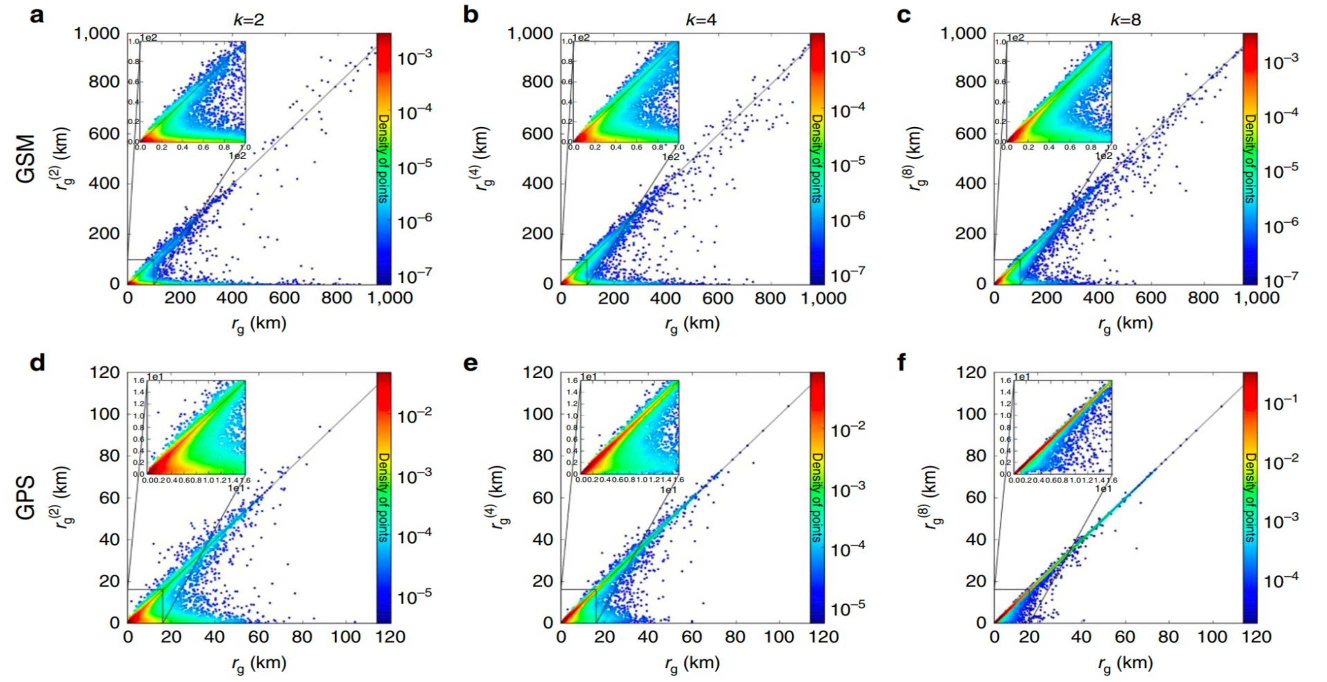


Figure 3 | The correlation between recurrent and overall mobility. The scatter plots represent the correlation between total r_g and $r_g^{(k)}$ for $k = 2, 4, 8$ in the GSM data set (**a–c**) and the GPS data set (**d–f**). Each point is coloured from blue to red, indicating the density of points in the corresponding region. Most of the points gather around the *x*-axis, the diagonal and the origin. The insets magnify the origin of the plot to [0, 100 km] for GSM and [0, 16 km] for GPS, demonstrating that the split emerges for smaller radii as well. As *k* increases explorers become returners. This transition is faster in the GPS case, consistent with the fact that the vehicle mobility represents a subset of trips and visited locations.

W#4: Explorers. Pappalardo et al. (2015)

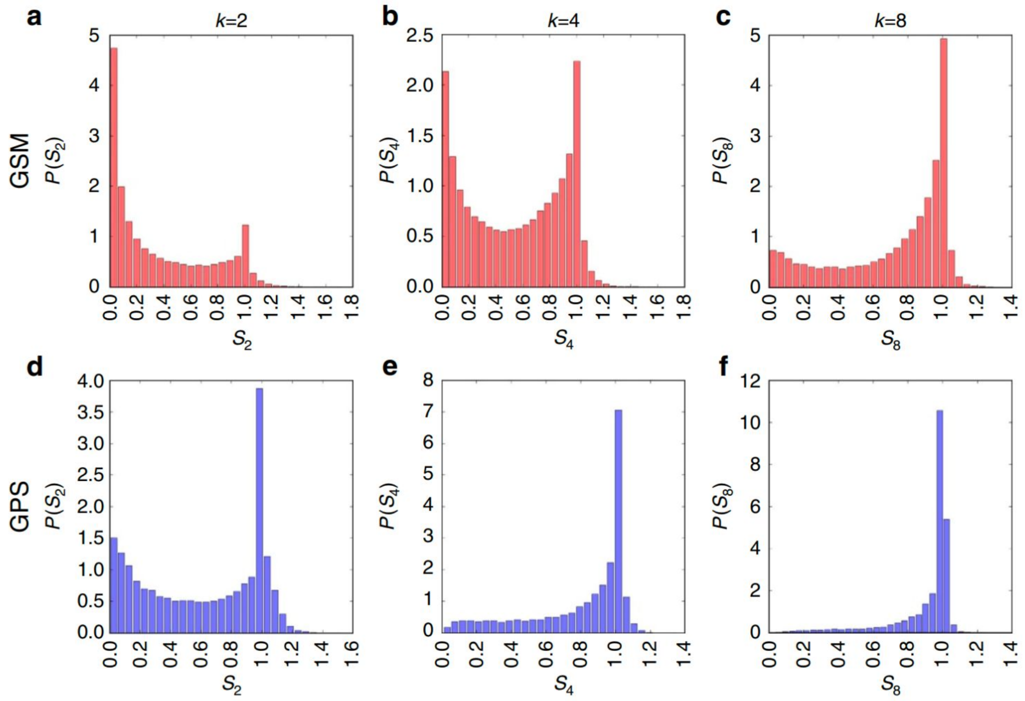


Figure 4 | The ratio between recurrent and overall mobility. The distribution $P(s_k)$ of the ratio $s_k = r_g^{(k)}/r_g$ measured on the GSM data for $k = 2, 4, 8$ (**a**–**c**). The peak at $s_k = 0$ corresponds to explorers, while the $s_k = 1$ peak corresponds to returners. For small k in the GSM data, k-explorers are more numerous than k-returners. As k increases the number of k-returners increases and overcomes the number of k-explorers. A balance in the population is reached at $k = 4$. (**d**–**f**) The $P(s_k)$ for the GPS data. We again observe two peaks, but the k-returners peak, $s_k = 1$, dominates for all $k \geq 2$.

$$r_g^{(k)} = \sqrt{\frac{1}{N_k}\sum_{i=1}^{k} n_i\left(\mathbf{r}_i - \mathbf{r}_{cm}^{(k)}\right)^2}$$

$$r_g = \sqrt{\frac{1}{N}\sum_{i\in L} n_i(\mathbf{r}_i - \mathbf{r}_{cm})^2},$$

W#4: Explorers. Pappalardo et al. (2015)

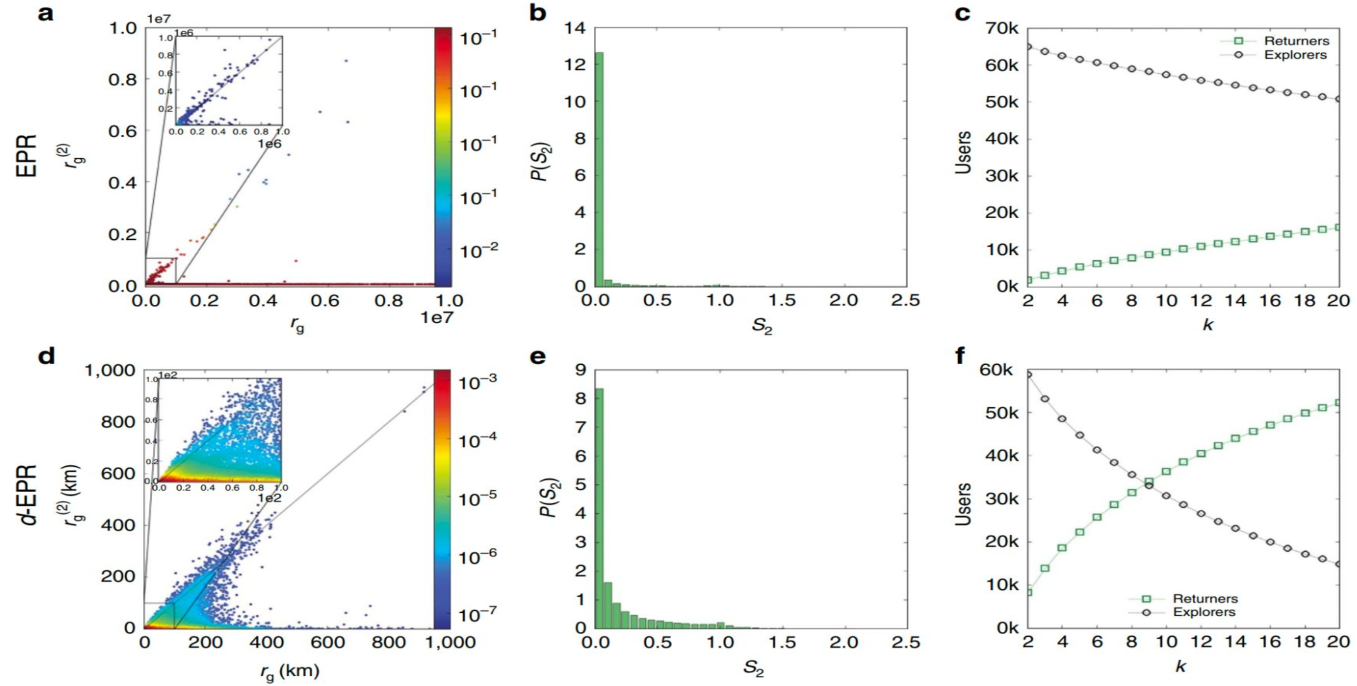


Figure 6 | EPR model predictions. (**a**,**b**) The prediction of the EPR model for $k=2$. We find that two-explorers dominate the population of synthetic individuals and the balance in the population is reached only for $k=60$, in contrast with $k=4$ in the empirical data. (**d**,**e**) The results of the d-EPR model for $k=2$. In this case, the two-explorers continue to dominate the population, although the balance is reached at lower values of $k=9$, coming closer to empirical data. The insets in **a**,**d** magnify the plot at smaller values of the radii of gyration. Plots (**c**,**f**) show how the number of k-returners and k-explorers changes with k for EPR model and d-EPR model, respectively.