Introduction to Computational Science

6. Spatial Models - Meta Population Models

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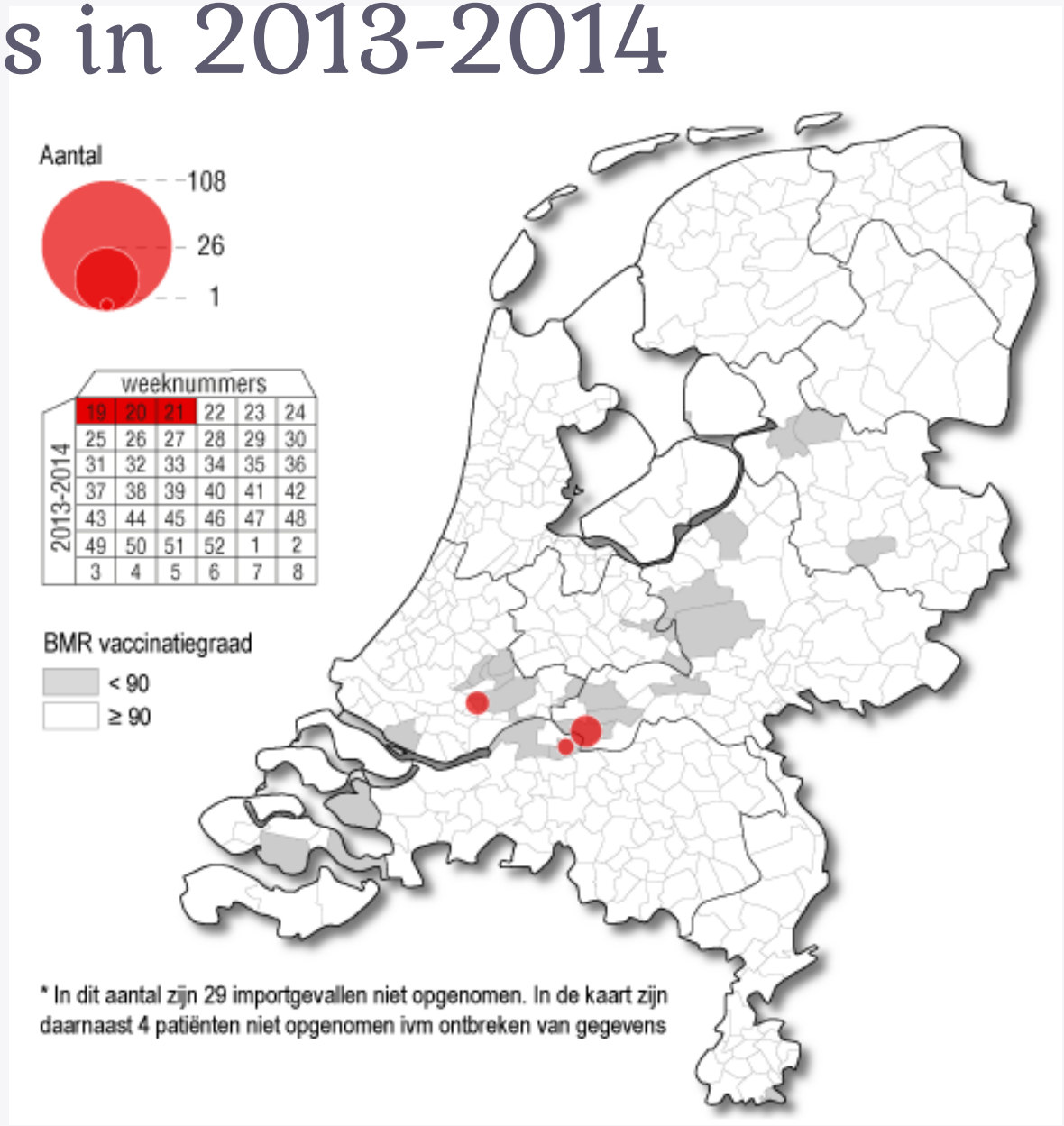
1. Spatial Model Introduction
2. Meta-population Models
3. Deterministic vs Stochastic Populations
4. Modelling Commuters
5. GleamViz – state of the art

6.1 Spatial Models Introduction

Remember: Example of a measles wave in the Netherlands in 2013-2014



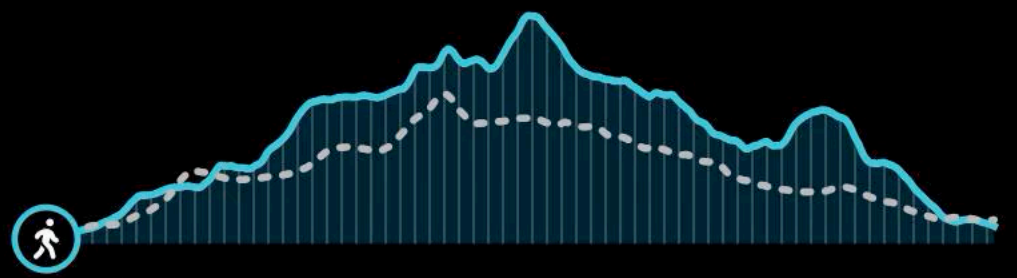
Source: https://www.volksgezondheidenzorg.info/onderwerp/ziekten-het-rijksvaccinatieprogramma/regionaal-internationaal/regionaal#node-mazelen-animatie-tijd

Donderdag 19 maart

06:00

Verplaatsingen ten opzichte van een reguliere donderdag

dat. mobility



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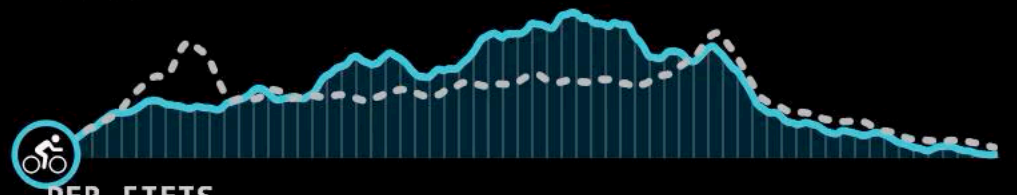

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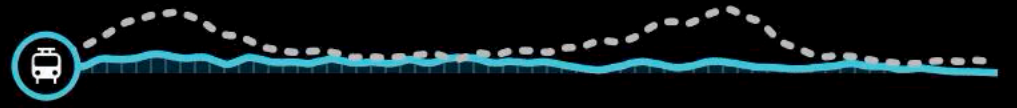

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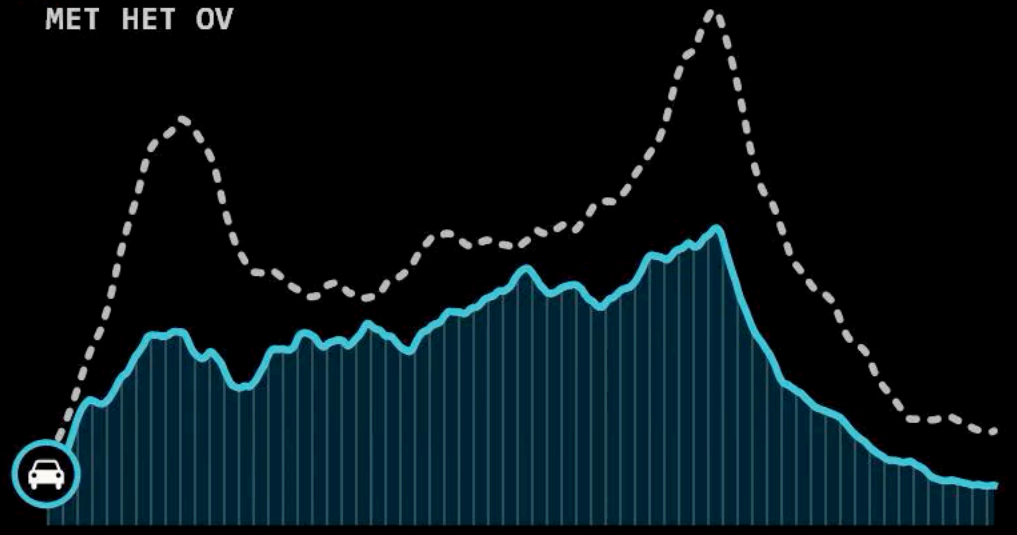

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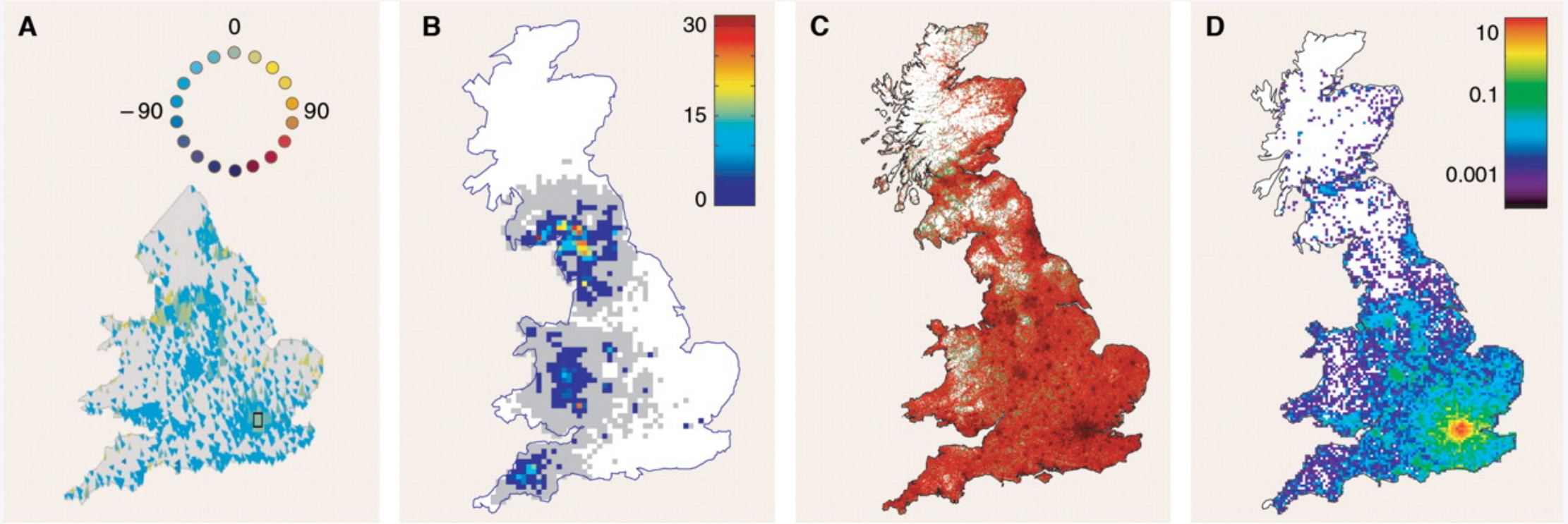
BRON: NEDERLANDS VERPLAATSINGS PANEL

The Spatial Component

- Capturing the spatial component
  - Transmission is a local process
  - Population is usually not evenly spread
  - Individuals move around between population centers
- To
  - Determine rate of spatial spread
  - Understand influence of heterogeneity of population density
  - Understand how to use this to optimize control measures

Patterns of disease transmission in the United Kingdom



Patterns of disease transmission in the United Kingdom. (**A**) Wavelet analysis of prevaccination measles epidemics in 954 locations in England and Wales [reproduced with permission from (*15*)] shows how London (black box) drove the epidemics in most of the country, with the exception of the Manchester-Liverpool urban concentration (upper left). (**B**) Simulations show the three high-risk areas [adapted with permission from (*21*)] for FMD in the United Kingdom in 2001. The key indicates the average number of cases in a 10-by-10–km square from 100 model realizations. (**C**) A novel strain of influenza [reproduced with permission from (*34*)] would spread rapidly though the United Kingdom during a global pandemic. Only 75 days after the arrival of the first cases from overseas, the intensity of red color shows the relative concentration of infectious individuals, and green indicates that the epidemic is already over in some small communities. (**D**) In contrast, even under a pessimistic transmission scenario, 75 days after 10 initial seeds became infectious with smallpox in London [adapted with permission from (*31*)], there would have been relatively few cases, and the degree of spatial correlation would still be striking [same key as in (B) but with 5-by-5–km squares.

Published by AAAS

Steven Riley Science 2007;316:1298-1301, Figure 1

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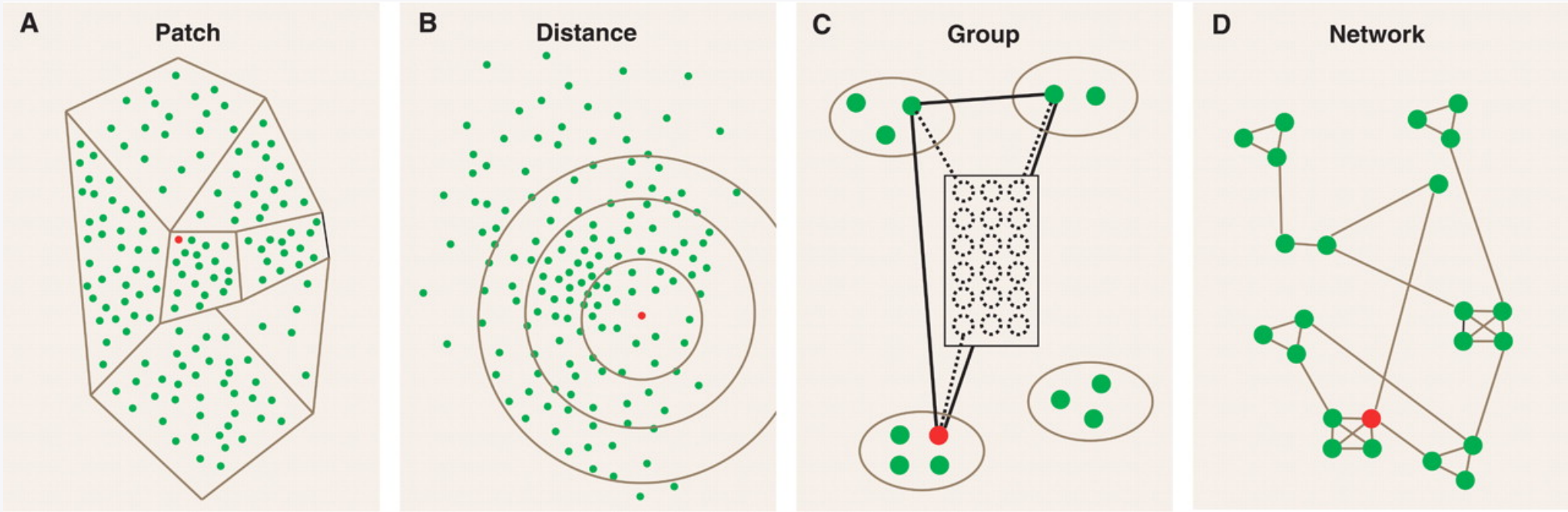
Four common abstractions for the spatial transmission of infectious diseases



Four common abstractions for the spatial transmission of infectious diseases. Differences between these approaches are best understood in terms of the FOI, which is location-specific in spatially explicit models. Red dots represent infectious individuals. (**A**) For patch transmission, all members of the same patch (residents of a town, for example) receive the same FOI, which is a function of the distance from their home patch to other patches and of the prevalence of infection in all patches. (**B**) Distance transmission is explicitly individual-based. It is assumed that any given infectious individual can infect all susceptible individuals within range. .... (**C**) In a pure multigroup model, the FOI is determined entirely by group membership. For example, if an infectious individual shares a household with a susceptible individual (ovals), there is a high probability of transmission occurring between the two. However, if an infectious individual does not share a group with a particular susceptible individual, transmission between the two cannot occur. .... (**D**) Network transmission is similar to group transmission in that the FOI experienced by susceptible individuals is zero, unless they share an arc with an infectious individual. .... In general, computational requirements increase from (A) to (D). Patch models can be implemented effectively on a typical desktop computer because they do not explicitly represent individuals. For population sizes greater than 10 million, individual-based models have been implemented on clusters of large-memory personal computers (*26*, *31*, *34*). Detailed microsimulation models (*33*) have not yet been implemented at scales larger than a city.

Published by AAAS

Steven Riley Science 2007;316:1298-1301, Figure 2

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Types of Spatial Models

- Many options, still developing field

- We will be looking at
  1. Metapopulation models
     - Usually using patch transmission
     - Metapopulations usually modelled in a stochastic framework
  2. Individual based models
     - Using distance, group
     - Using stochastic simulation setups
     - Network Models or Agent-based Models

Other options (see Keeling and Rohani Ch. 7):

1. Lattice based approaches

   Special cases of above, mainly to study in abstract way fundamental processes of disease dynamics
2. Continuous PDE based models

Important concepts

1. Heterogeneity
   - Spatial differences in the fundamental forces
     - e.g. different social structures leading to different transmission rates
   - Emerging spatial differences
     - Arising from the dynamical processes, stochasticity, different movement patterns, etc.
2. Interaction
   - Due to movement of host between populations
   - Allows for spreading of a disease
   - Modelling interaction is key in including the spatial component
     - E.g. by adding additional force of infection due to infectious individuals in other locations
     - Typically, interaction between two populations should in some way decrease with distance d between them.
     - This is captured by a transmission kernel K
       - e.g. exponential $K \propto \exp(-Ad)$, Gaussian $K \propto \exp(-Ad^2)$, or power law $K \propto d^{-A}$.

Important concepts

3. Isolation
   - Isolated populations can be protected from infections
4. Local Extinction
   - Due to smaller subpopulation sizes in spatial models, local extinctions are common.
   - Interaction with larger subpopulations where the disease is endemic (e.g. large cities) then leads to new invasions of the pathogen
5. Scale
   - Scale of interactions
     - From metapopulations (but how large/small are they) down to individuals
     - More subtle in lattice based and PDE based models
   - Scale of the simulation
     - In real world problems this is clear, e.g. spreading of SARS in USA.
     - But in generic situations this is less clear. It should be large enough so that we can observe all the interesting dynamics.
       - but not too large ...

6.2 Meta Population Models

Metapopulations

- Most applicable to many human diseases
- Concept of metapopulation modelling
  1. Subdivide the population in distinct subpopulations
     - Cities, villages, or based on administrative boundaries (counties, districts, etc) leading to patches in which subpopulations live.
  2. Each subpopulation has independent dynamics
     - Deterministic, stochastic, with or without seasonal forcing, etc.
  3. and a limited form of interaction
     - Implementing some interaction kernel

Metapopulations in the SIR framework

- Assume n subpopulations, and (X_i, Y_i, Z_i) are the number of susceptible, infectious and recovered individuals in subpopulation i.
- Likewise, $N_i = X_i + Y_i + Z_i$ is to total number of individuals in subpopulation i and $N = \sum_{i=1}^{n} N_i$ is the total population size.
- We first model interaction purely due to an additional force of infection

$$\frac{dX_i}{dt} = \upsilon_i N_i - \lambda_i X_i - \mu_i X_i$$

$$\frac{dY_i}{dt} = \lambda_i X_i - \gamma_i Y_i - \mu_i Y_i$$

Force of infection

- Relation between force of infection in population i and the number of infectious individuals in population j.
  - Depends highly on assumed mechanism of transmission
  - In general

$$\lambda_i = \beta_i \sum_{j=1}^{n} \rho_{ij} \frac{Y_j}{N_i}$$

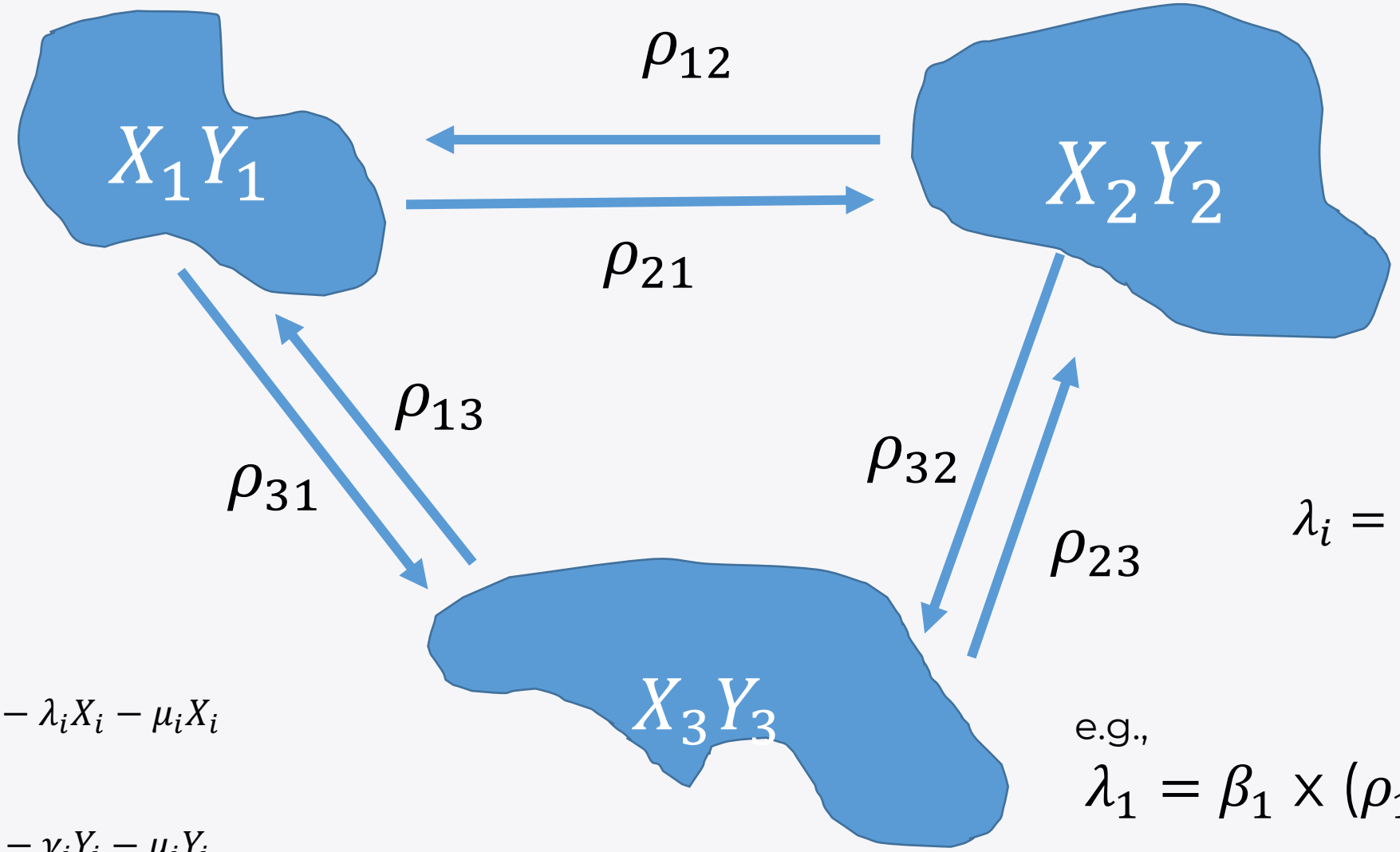
- So, a weighted sum over prevalence in all populations

(ps. note a little mistake equation in book)

- Strength of interaction matrix
- ρ_{ij} is the relative strength of transmission to population i from population j

- Important choice is the precise scaling with the population size.
- Here we assumed that transmission takes place in population i
  - E.g. due to a visit from an infectious individual from j to i
  - So, divide by N_i
- Vice versa,
  - if the model would be that the interaction is due to individual from i picking up an infection in j
  - Then divide by N_j

Metapopulation Schematic



$$\lambda_i = \beta_i \sum_{j=1}^{n} \rho_{ij} \frac{Y_j}{N_i}$$

$$\frac{dX_i}{dt} = v_i N_i - \lambda_i X_i - \mu_i X_i$$

$$\frac{dY_i}{dt} = \lambda_i X_i - \gamma_i Y_i - \mu_i Y_i$$

e.g.,

$$\lambda_1 = \beta_1 \times \left(\rho_{12} \frac{Y_2}{N_1} + \rho_{13} \frac{Y_3}{N_1}\right)$$

6.3 Deterministic vs Stochastic Populations

Deterministic vs. Stochastic subpopulations

- Consider two subpopulations, $n = 2$.
  - Both of equal size $N_1 = N_2 = N'$, simplifying the interaction terms
  - Ignore demography
  - Fully susceptible at start
  - Introduce infection in subpopulation 1
  - Only coupling from 1 to 2, specifically, assume $\rho_{ii} = 1$, $\rho_{12} = 0$, and ρ_{21} is small, to mimic a relative weak coupling
- Model subpopulation 1 with a deterministic model, and subpopulation 2 with either a deterministic or stochastic model.
  - What would be the differences in the dynamics of the spreading of the disease in subpopulation 2, as a function of the interaction strength ρ_{21}?

Population 2: deterministic

$$\frac{dY_2}{dt} = \beta_2 \rho_{21} \frac{Y_1}{N'} X_2 + \beta_2 \frac{Y_2}{N'} X_2 - \gamma_2 Y_2$$

Divide the equation by N' and consider early dynamics where $S_2 = \frac{X_2}{N'} \approx 1$, leading to

$$\frac{dI_2}{dt} = \beta_2 \rho_{21} I_1 + \beta_2 I_2 - \gamma_2 I_2$$

which can be solved as $I_2(t) = \int_0^t \beta_2 \rho_{21} I_1(s) \exp([\beta_2 - \gamma_2]s)\, ds$

We observe

1. The disease is present in population 2 immediate from the start of the epidemic in population 1
2. Early very small infections that arrive in population 2 trigger an exponential growth at rate $\beta_2 - \gamma_2$.

Population 2: stochastic

- Very different behavior
- The coupling between populations 2 and 1 will trigger events in 2 that lead to introduction of one or more infectious individuals in population 2.
  - However, that happens initially at a small rate
  - And the probability that the disease subsequently goes extinct is $\frac{1}{R_0}$
- Estimate the probability that an epidemic is triggered in population 2

$$P(\text{epidemic}) = \sum_{n=1}^{N'} P(\text{cases in 1 cause } n \text{ cases in 2}) \times P(n \text{ initial cases lead to epidemic})$$

$$= \sum_{n=1}^{N'} \exp\left(-\beta_2 \rho_{21} \int_0^\infty I_1(s)ds\right) \frac{\left(\beta_2 \rho_{21} \int_0^\infty I_1(s)ds\right)^n}{n!} \times \left[1 - \left(\frac{\gamma}{\beta_2}\right)^n\right]$$

Poisson statistics

Probability that invasion with *n* infecteds in population 2 leads to an epidemic

$$= \exp\left(-\beta_2 \rho_{21} \int_0^\infty I_1(s)ds\right)\left[\exp\left(\beta_2 \rho_{21} \int_0^\infty I_1(s)ds\right) - \exp\left(\gamma \rho_{21} \int_0^\infty I_1(s)ds\right)\right]$$

$$= 1 - \exp\left(-\beta_2 \rho_{21}\left[1 - \frac{\gamma}{\beta_2}\right]\int_0^\infty I_1(s)ds\right) < 1 - \exp\left(-\beta_2 \rho_{21}\left[1 - \frac{\gamma}{\beta_2}\right]\right)$$

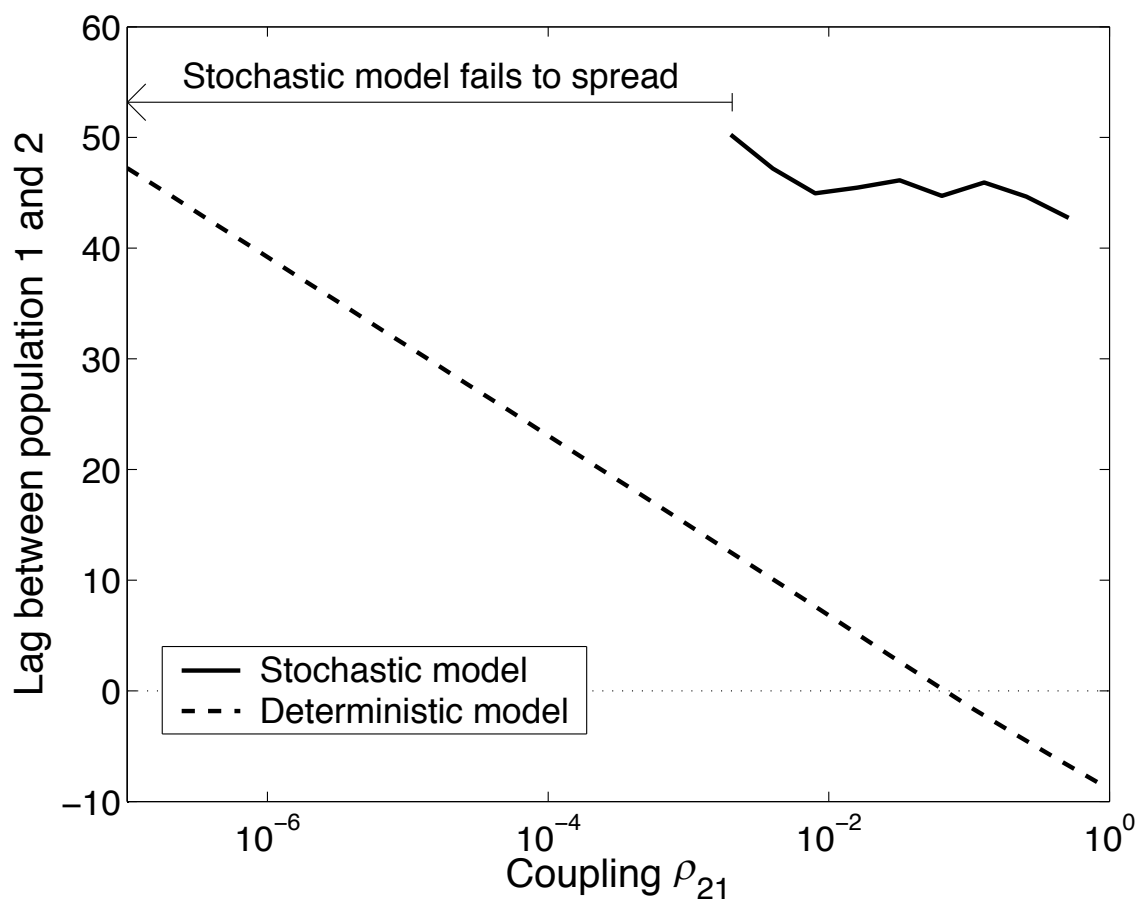
Population 2: stochastic

- Estimate the probability that an epidemic is triggered in population 2

$$P(\text{epidemic}) < 1 - \exp\left(-\beta_2 \rho_{21}\left[1 - \frac{\gamma}{\beta_2}\right]\right)$$

- We observe
  1. If the coupling ρ_{21} is small enough, there is a high probability that the disease will not spread in population 2
  2. For larger coupling ρ_{21} the pathogen can spread, but their can be a significant delay of spreading of the disease in population 2 with respect to the epidemic in population 1
  3. In stochastic metapopulation models the spread of infectious disease is slower than for deterministic ones.

Delay between population 1 and 2



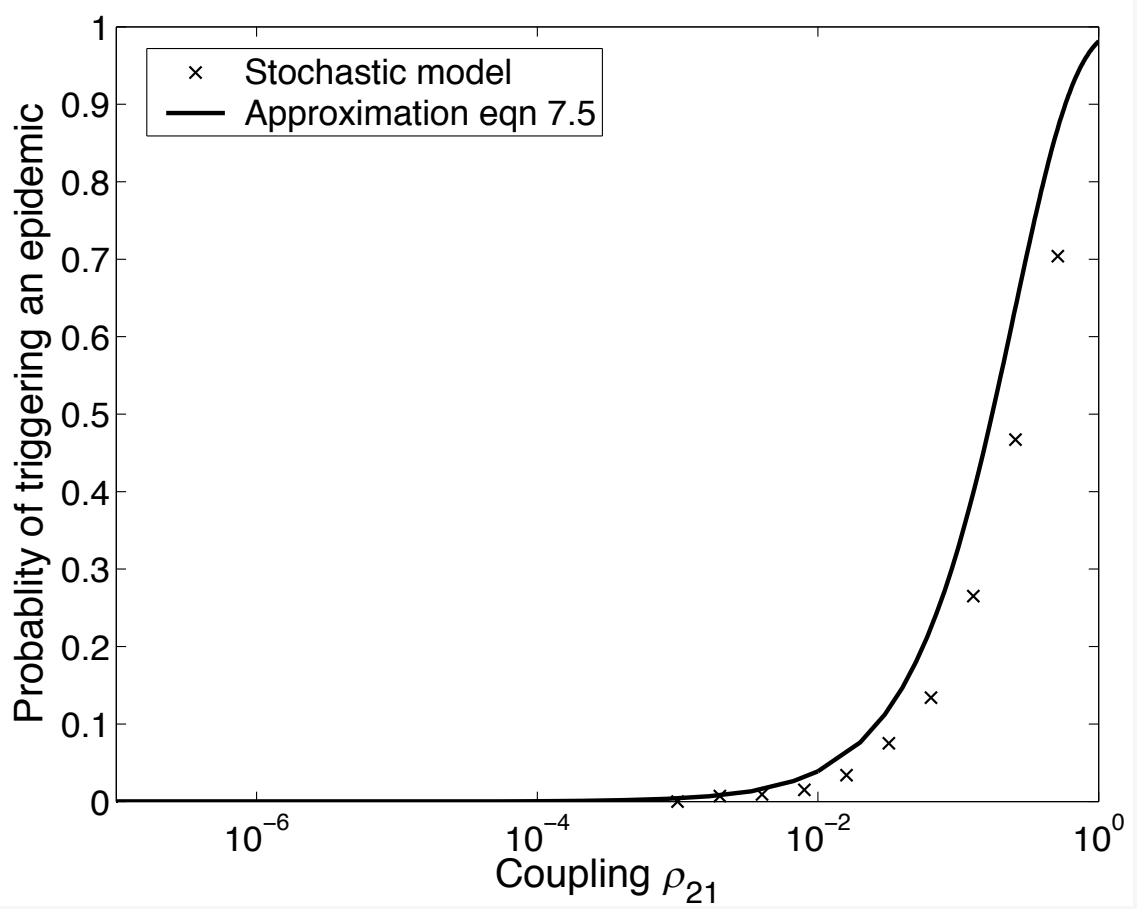


Figure 7.1 from Keeling and Rohani, For a metapopulation with just two populations we examine the effect of coupling ρ_{21} on the dynamics of the epidemic in population 2. Population 1 is modelled deterministically and is initialized with $I_1(0) = 10^{-5}$, $S_1(0) = 1 - I_1(0)$. The left-hand graph shows the delay between the peak of the epidemic in population 1 and the peak in population 2, where population 2 is modeled either stochastically (solid line) or deterministically (dashed line) and is initially disease-free. The right-hand graph is the probability that a major epidemic is triggered in the stochastic population (crosses) compared to the analytical approximation. ($\mu = \nu = 0$, $1/\gamma = 14$ days, $\beta = 0.3571$ per day $\Longrightarrow$ $R_0 = 5$, $\rho_{ii} = 1$, $\rho_{12} = 0$. Stochastic results are the average of 1,000 realizations, $N_2 = 10^5$).

6.4 Modelling Commuters

Modelling commuters

- Commuters live in one population, say j, but travel occasionally to another population, say i.
- Let's formulate an SIR framework including commuters
- X_{ij}, Y_{ij}, and N_{ij} are the number of susceptibles, infecteds, and total hosts currently in population i that live in population.

$$\frac{dX_{ii}}{dt} = v_{ii}N_{ii} - \beta_i X_{ii}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} - \sum_j l_{ji}X_{ii} + \sum_j r_{ji}X_{ji} - \mu_{ii}X_{ii}$$

$$\frac{dX_{ij}}{dt} = v_{ij}N_{ij} - \beta_i X_{ij}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} + l_{ij}X_{jj} - r_{ij}X_{ij} - \mu_{ij}X_{ij}$$

$$\frac{dY_{ii}}{dt} = \beta_i X_{ii}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} - \gamma Y_{ii} - \sum_j l_{ji}Y_{ii} + \sum_j r_{ji}Y_{ji} - \mu_{ii}Y_{ii}$$

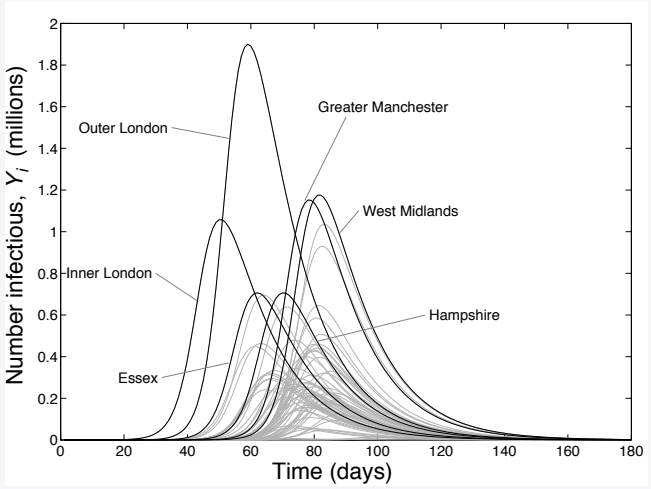
$$\frac{dY_{ij}}{dt} = \beta_i X_{ij}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} - \gamma Y_{ij} + l_{ij}Y_{jj} - r_{ij}Y_{ij} - \mu_{ij}Y_{ij}$$

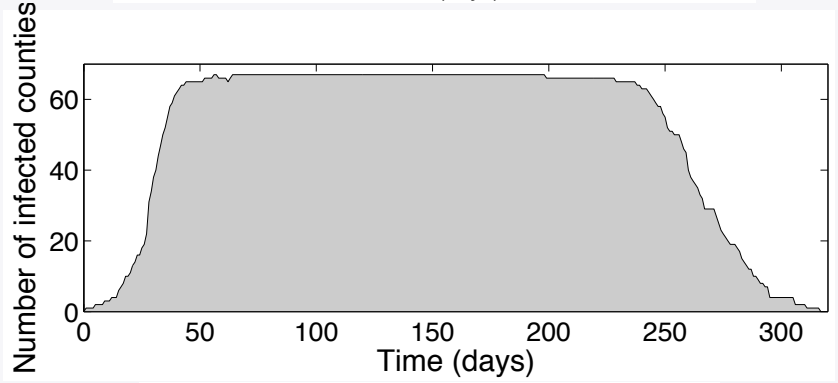
$$\frac{dN_{ii}}{dt} = v_{ii}N_{ii} - \sum_j l_{ji}N_{ii} + \sum_j r_{ji}N_{ji} - \mu_{ii}N_{ii}$$

$$\frac{dN_{ij}}{dt} = v_{ij}N_{ij} + l_{ij}N_{jj} - r_{ij}N_{ij} - \mu_{ij}N_{ij}$$

- l_{ij}: rate at which individuals leave their home j and commute to i
- r_{ij}: return rate, so from i back to home j
- l and r can be found from detailed data on populations, commuting, etc.
- We use frequency transmission with $\sum_j N_{ij}$ the total individuals in population i
- Full mechanistic description, but at expense of $3n^2$ equations for n populations
- Approximations exist (see 7.2.1.4 in book)

Spreading in 67 counties in England





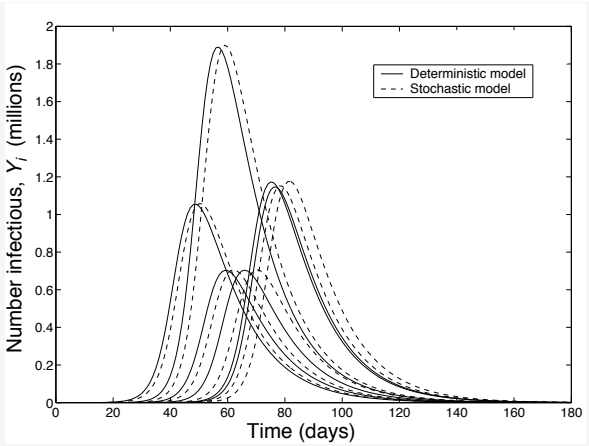


Figure 7.2 from Keeling and Rohani, Deterministic and stochastic results for an infection spread through the 67 counties of Great Britain. The epidemic is initialized with 10 cases in Inner London, and is spread by commuter movements. The population size and rate of commuting is taken from the 1991 census database, and all trips are considered to be of short duration, $1/r = 0.5$ days. The top figure shows the county level epidemics from a single stochastic iteration, with six counties highlighted. The middle graph shows the number of counties with infection from the same stochastic model. The bottom graph compares the deterministic solution (solid line) with the stochastic model (dashed line) for the six counties highlighted in the top graph. ($\mu = \nu = 0$, $1/\gamma = 14$ days, $\beta = 0.3571$ per day $\Longrightarrow R_0 = 5$).

- Some results not so surprising
  - First major epidemic in innner London
  - Next and largest peak in outer London
- Some maybe more surprising
  - E.g. late peak in greater Manchester
- Within 50 days infection reached all counties
  - After that time commuting plays a minor role
- Disease stays around until after 300 days
  - Despite very low levels
- In stochastic dynamics the epidemic is delayed.

Coarse Graining the Commuter model

Fully detailed commuter model

$$\frac{dX_{ii}}{dt} = v_{ii}N_{ii} - \beta_i X_{ii}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} - \sum_j l_{ji}X_{ii} + \sum_j r_{ji}X_{ji} - \mu_{ii}X_{ii}$$
$$\frac{dX_{ij}}{dt} = v_{ij}N_{ij} - \beta_i X_{ij}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} + l_{ij}X_{jj} - r_{ij}X_{ij} - \mu_{ij}X_{ij}$$
$$\frac{dY_{ii}}{dt} = \beta_i X_{ii}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} - \gamma Y_{ii} - \sum_j l_{ji}Y_{ii} + \sum_j r_{ji}Y_{ji} - \mu_{ii}Y_{ii}$$
$$\frac{dY_{ij}}{dt} = \beta_i X_{ij}\frac{\sum_j Y_{ij}}{\sum_j N_{ij}} - \gamma Y_{ij} + l_{ij}Y_{jj} - r_{ij}Y_{ij} - \mu_{ij}Y_{ij}$$
$$\frac{dN_{ii}}{dt} = v_{ii}N_{ii} - \sum_j l_{ji}N_{ii} + \sum_j r_{ji}N_{ji} - \mu_{ii}N_{ii}$$
$$\frac{dN_{ij}}{dt} = v_{ij}N_{ij} + l_{ij}N_{jj} - r_{ij}N_{ij} - \mu_{ij}N_{ij}$$

?

Generic metapopulation model

$$\frac{dX_i}{dt} = v_i N_i - \left(\beta_i \sum_{j=1}^{n} \rho_{ij}\frac{Y_j}{N_i}\right)X_i - \mu_i X_i$$

$$\frac{dY_i}{dt} = \left(\beta_i \sum_{j=1}^{n} \rho_{ij}\frac{Y_j}{N_i}\right)X_i - \gamma_i Y_i - \mu_i Y_i$$

after a few simplifications and assumptions

$$\frac{dX_i}{dt} = \mu N - \beta\frac{X_i}{N}[(1-(n-1)\rho)Y_i + \rho\sum_{k\neq i}Y_k] - \mu X_i$$
$$\frac{dY_i}{dt} = \beta\frac{X_i}{N}[(1-(n-1)\rho)Y_i + \rho\sum_{k\neq i}Y_k] - \gamma Y_i - \mu Y_i$$

For all detailed derivations (study them!) see notes ‘Module 6 – Modelling Commuters I’.

Coarse Graining the Commuter model II

- 5 assumptions
  1. All parameters do not depend on i, j

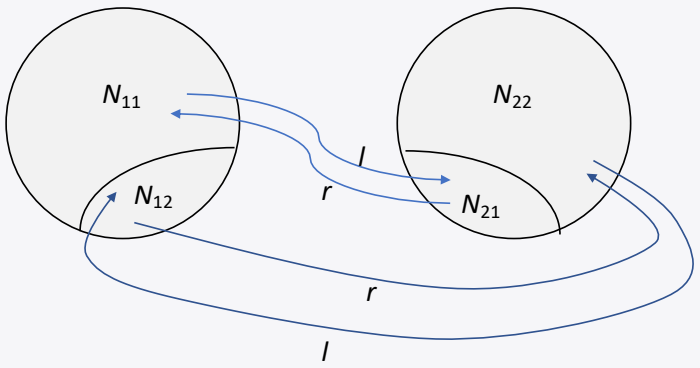
     $\Longrightarrow \upsilon, \mu, \beta, l, r$
  2. $\upsilon = \mu$
  3. All metapopulations of equal size

     $\Longrightarrow N_i = N \ \forall \ i.$
  4. Dynamics of commuting is fast, reaches equilibrium fast (and with $\alpha = \frac{l}{r}$)

     $$\Longrightarrow \begin{matrix} N_{ii} = \frac{1}{1+\alpha(n-1)} N \\ N_{ij} = \frac{\alpha}{1+\alpha(n-1)} N \end{matrix}$$
  5. Dynamics of commuting is much faster than dynamics of disease and demography

     $$\Longrightarrow \begin{matrix} X_{ij} = \frac{\alpha}{1+\alpha(n-1)} X_i \\ Y_{ij} = \frac{\alpha}{1+\alpha(n-1)} Y_i \end{matrix}$$

For $n = 2$



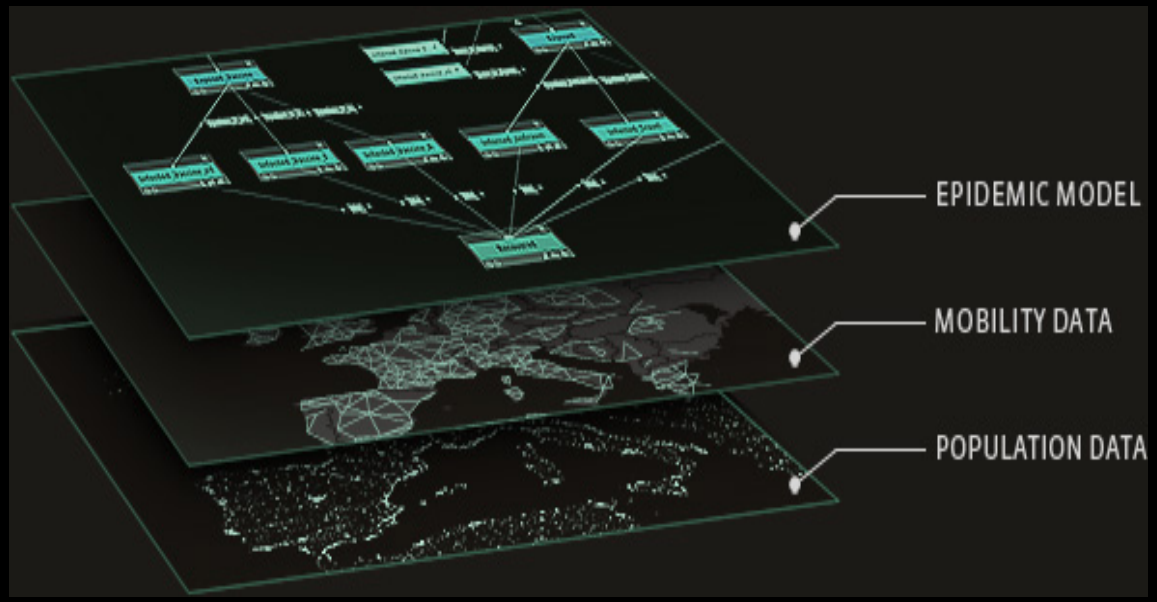
For all detailed derivations (study them!) see notes ‘Module 6 – Modelling Commuters I’. Also see ‘Module 6 – Modelling Commuters II’ for limits of ρ.

6.5 GleamViz

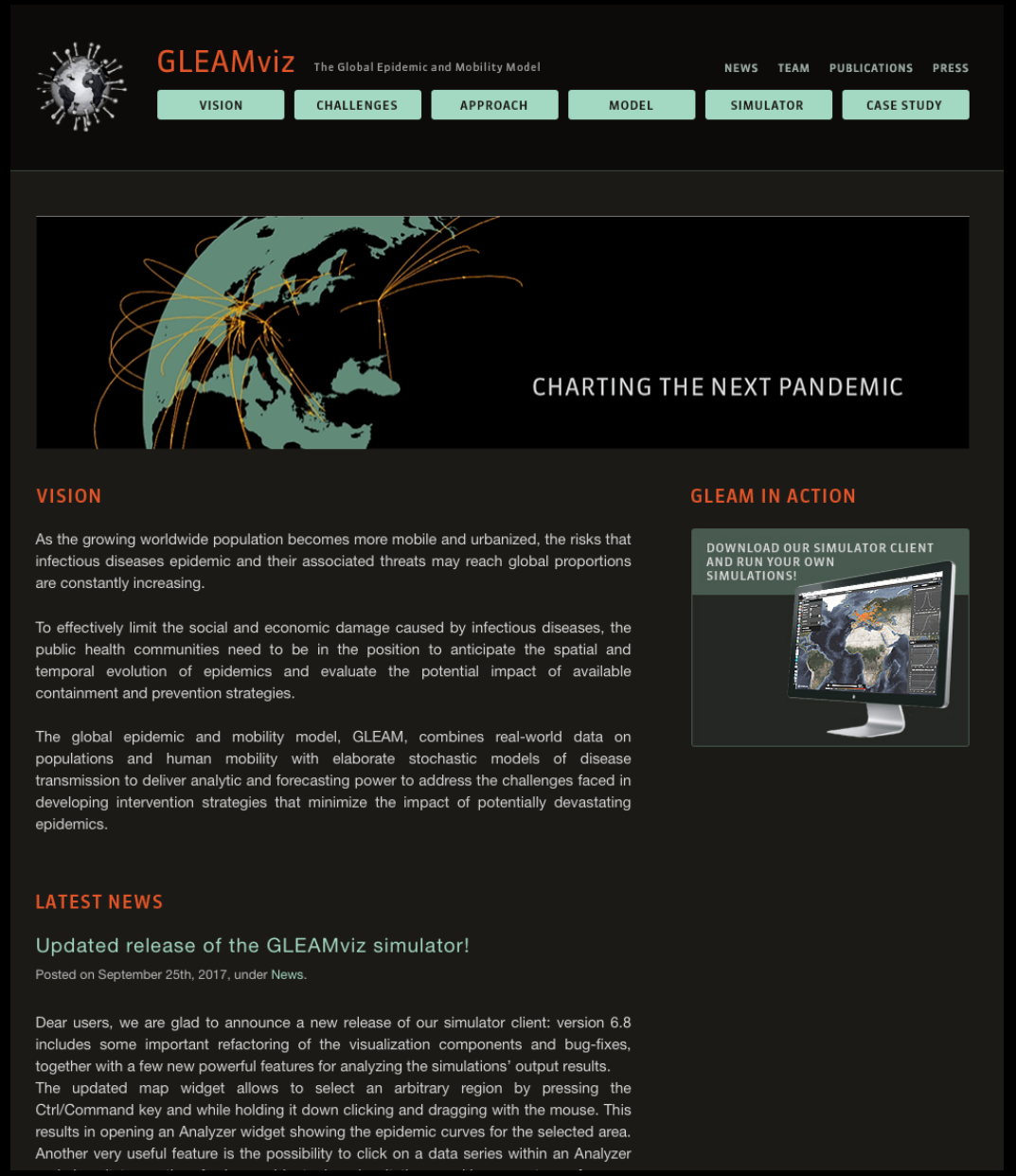
State of the art Metapopulation Model

The Global Epidemic and Mobility Model

http://www.gleamviz.org



- Structured metapopulation model
  - Subpopulation are population areas around major airports
  - Mobility data based on transportation data (e.g. from IATA)
  - Advanced disease models for each subpopulation



Relevant papers focusing on importance of airlines as major interaction

1. V. Colizza, A. Barrat, M. Barthélemy, A. Vespignani, *The Modeling of Global Epidemics: Stochastic Dynamics and Predictability*, Bulletin of Mathematical Biology (2006) 68: 1893–1921
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3. Vittoria Colizza, Alain Barrat, Marc Barthélemy, and Alessandro Vespignani, The role of the airline transportation network in the prediction and predictability of global epidemics, PNAS (2006) 103: 2015–2020
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- Available via blackboard, as background material. Good reading!

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RESEARCH ARTICLE

The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak

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Science 24 Apr 2020:
Vol. 368, Issue 6489, pp. 395-400
DOI: 10.1126/science.aba9757

Outbreak to pandemic

In response to global dispersion of severe acute respiratory syndrome–coronavirus 2 (SARS-CoV-2), quarantine measures have been implemented around the world. To understand how travel and quarantine influence the dynamics of the spread of this novel human virus, Chinazzi *et al.* applied a global metapopulation disease transmission model to epidemiological data from China. They concluded that the travel quarantine introduced in Wuhan on 23 January 2020 only delayed epidemic progression by 3 to 5 days within China, but international travel restrictions did help to slow spread elsewhere in the world until mid-February. Their results suggest that early detection, hand washing, self-isolation, and household quarantine will likely be more effective than travel restrictions at mitigating this pandemic.

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1
Welcome to EpiRisk
EpiRisk is a computational platform designed to allow a quick estimate of the probability of exporting infected individuals from sites affected by a disease outbreak to other areas in the world through the airline transportation network and the daily commuting patters. It also lets the user to explore the effects of potential restrictions applied to airline traffic and commuting flows.
Based on the number of infected individuals detected in one or more areas of the world, the platforms estimates two main quantities.
· Exported cases: the tool computes the probability P(n) of exporting a given number of cases n from the origin of the disease outbreak. In order to calculate the distribution P, the average time from exposure to symptoms onset and inability of traveling of infected individuals must be provided.
· Relative importation risk: for each location Y the platform evaluates the probability P(Y) that a single infected individual is traveling from the index areas to that specific destination Y. In other words, given the occurrence of one exported case, P(Y) is the probability that the disease carrier will appear in location Y, with respect to any other possible location.
By interacting with the map, the user can inspect the relative risk and the probability distribution of imported cases for single locations. In addition, the computed results are downloadable in commonly used data formats and as a high-resolution image of the risk map.
The airline transportation data used in the platform are based on origin-destination traffic flows from the OAG database that are aggregated at specific time and spatial scales by the GLEAM project. Commuting flows are derived by the analysis and modeling of data for more than 5,000,000 commuting patterns among 78,000 administrative regions in five continents. A manuscript detailing the algorithms devised to compute the estimates provided by the platform is under preparation.
Epirisk is a not-for profit platform: the results generated by the tool can be shared in compliance to the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International License.
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