Spatial Spread of Infectious Diseases

Part I
Population-based approaches

Spatial Spread of Infectious Diseases

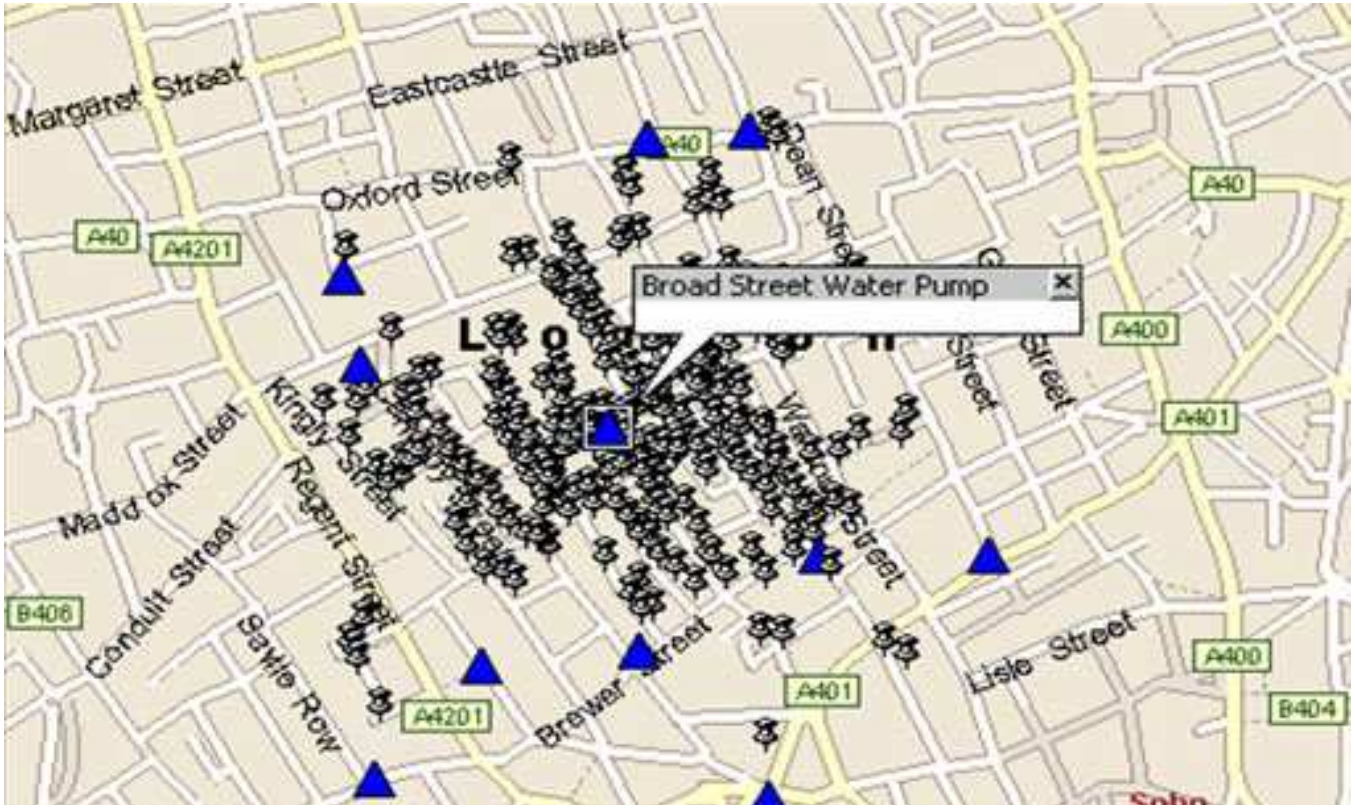
The distribution of populations across space and the patterns of interaction among groups influence how diseases spread in space and time.

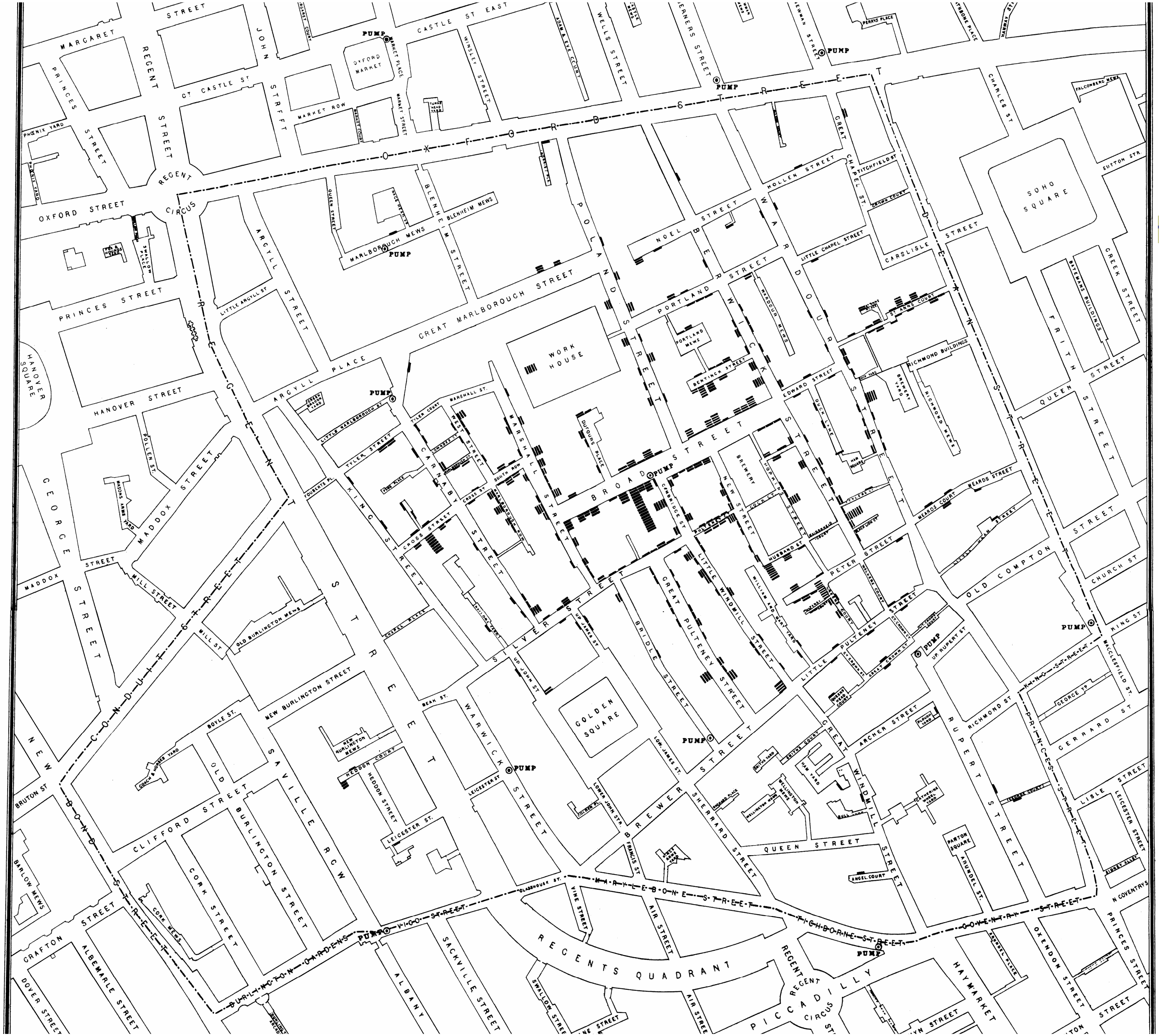
Spatial Spread of Infectious Diseases

John Snow (1854) tried to understand the origin of the cholera epidemic by analyzing the distribution of cases in London.

Spatial Spread of Infectious Diseases

Mapping the cases helped localize the disease and identifying the pattern of water use traced the infections to the source.




OXFORD STREET
REGENT CIRCUS
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JOHN STREET
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NEWMAN STREET
CHARLES ST.
SOHO SQUARE
PRINCES STREET
HANOVER SQUARE
HANOVER STREET
GREAT MARLBOROUGH STREET
MARLBOROUGH MEWS
BLENHEIM STREET
BLENHEIM MEWS
ARGYLL STREET
ARGYLL PLACE
LITTLE ARGYLL ST
POLAND STREET
NOEL STREET
BERWICK STREET
WARDOUR STREET
DEAN STREET
FRITH STREET
CREEK STREET
HOLLEN STREET
CARLISLE STREET
PORTLAND STREET
PORTLAND MEWS
WORK HOUSE
BROAD STREET
MARSHALL STREET
CARNABY STREET
KING STREET
SILVER STREET
TYLER STREET
RICHMOND BUILDINGS
QUEEN STREET
MEARDS STREET
OLD COMPTON STREET
CHURCH ST
PETER STREET
HUSBAND ST
LITTLE PULTENEY STREET
GREAT PULTENEY STREET
LITTLE WINDMILL STREET
BRIDLE STREET
GOLDEN SQUARE
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MILL STREET
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OLD BURLINGTON MEWS
NEW BURLINGTON STREET
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SAVILLE ROW
CORK STREET
WARWICK STREET
BREWER STREET
SHERRARD STREET
GREAT WINDMILL STREET
ARCHER STREET
RUPERT STREET
RICHMOND ST
KING ST
PRINCES STREET
GERRARD ST
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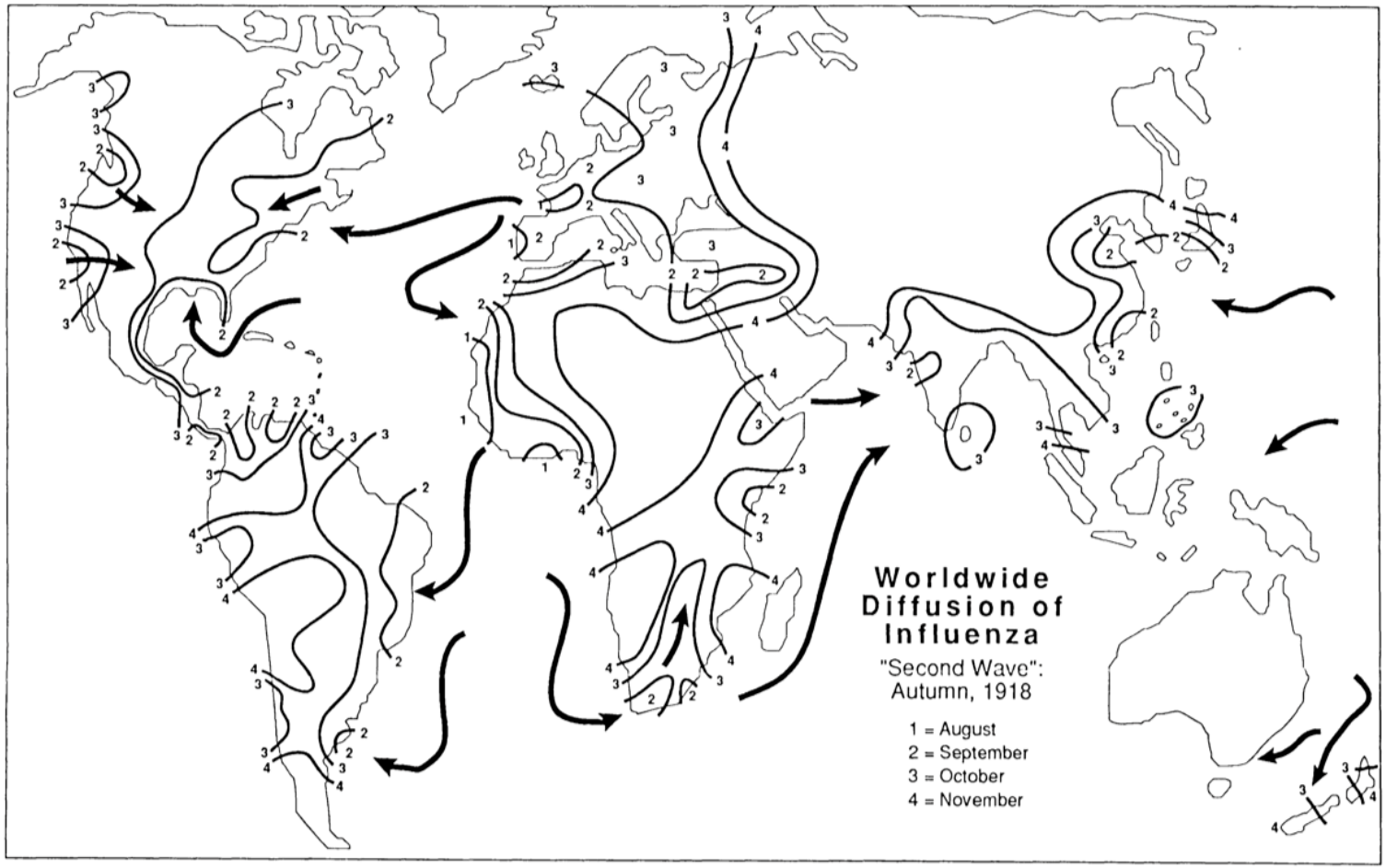
LEICESTER STREET
MARYLEBONE STREET
COVENTRY STREET
TICHBORNE STREET
REGENTS QUADRANT
REGENT CIRCUS
PICCADILLY
HAYMARKET
VIGO STREET
BURLINGTON GARDENS
SACKVILLE STREET
ALBANY
AIR STREET
VINE STREET
GRAFTON STREET
ALBEMARLE STREET
DOVER STREET
BARLOW MEWS
PARTON SQUARE
ANGEL COURT
PUMP

Spatial Spread of Infectious Diseases

Influenza 1918-1919 pandemic

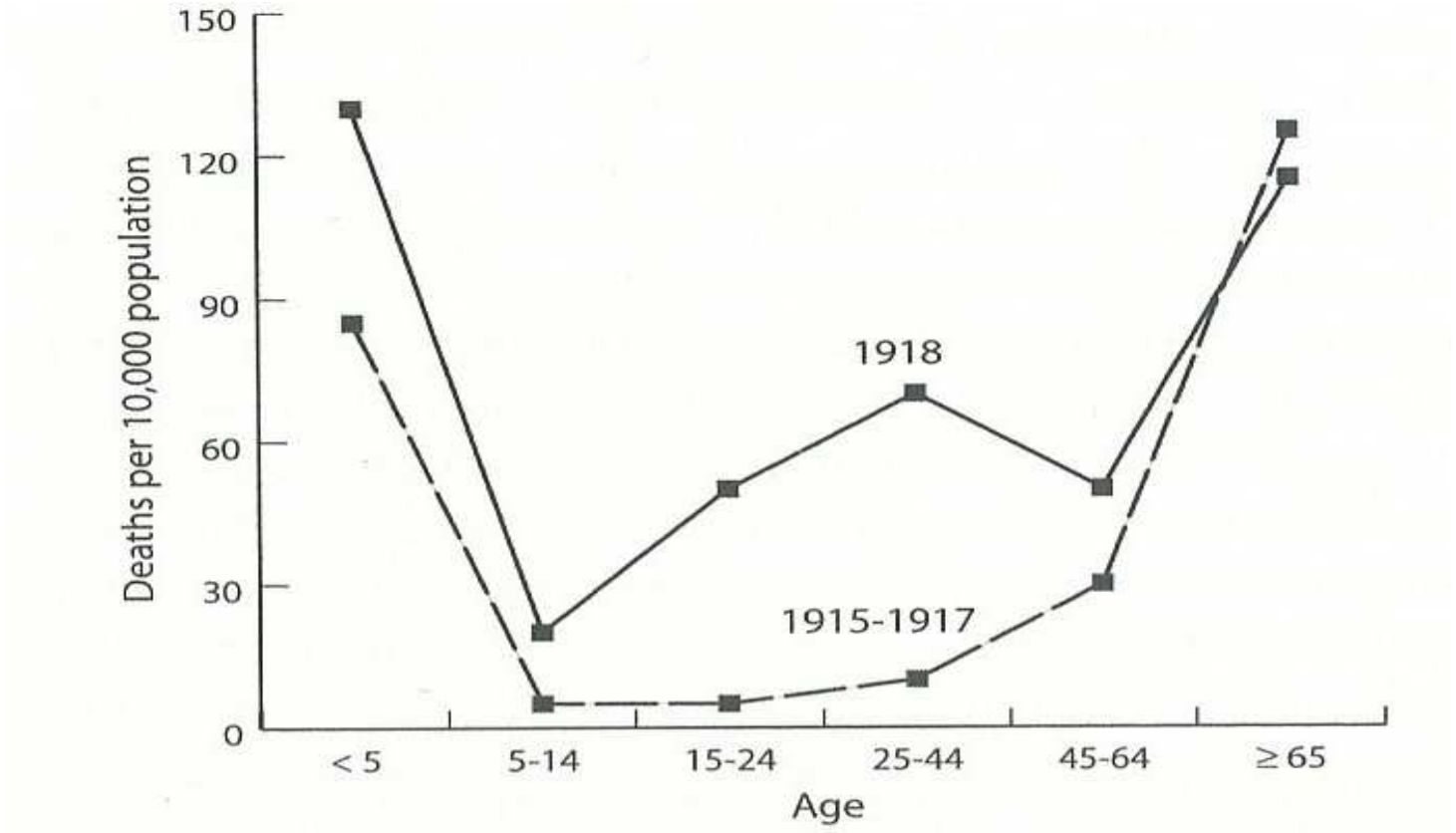
Spatial Spread of Infectious Diseases

Spatial spread of influenza for the 1918-1919 pandemic



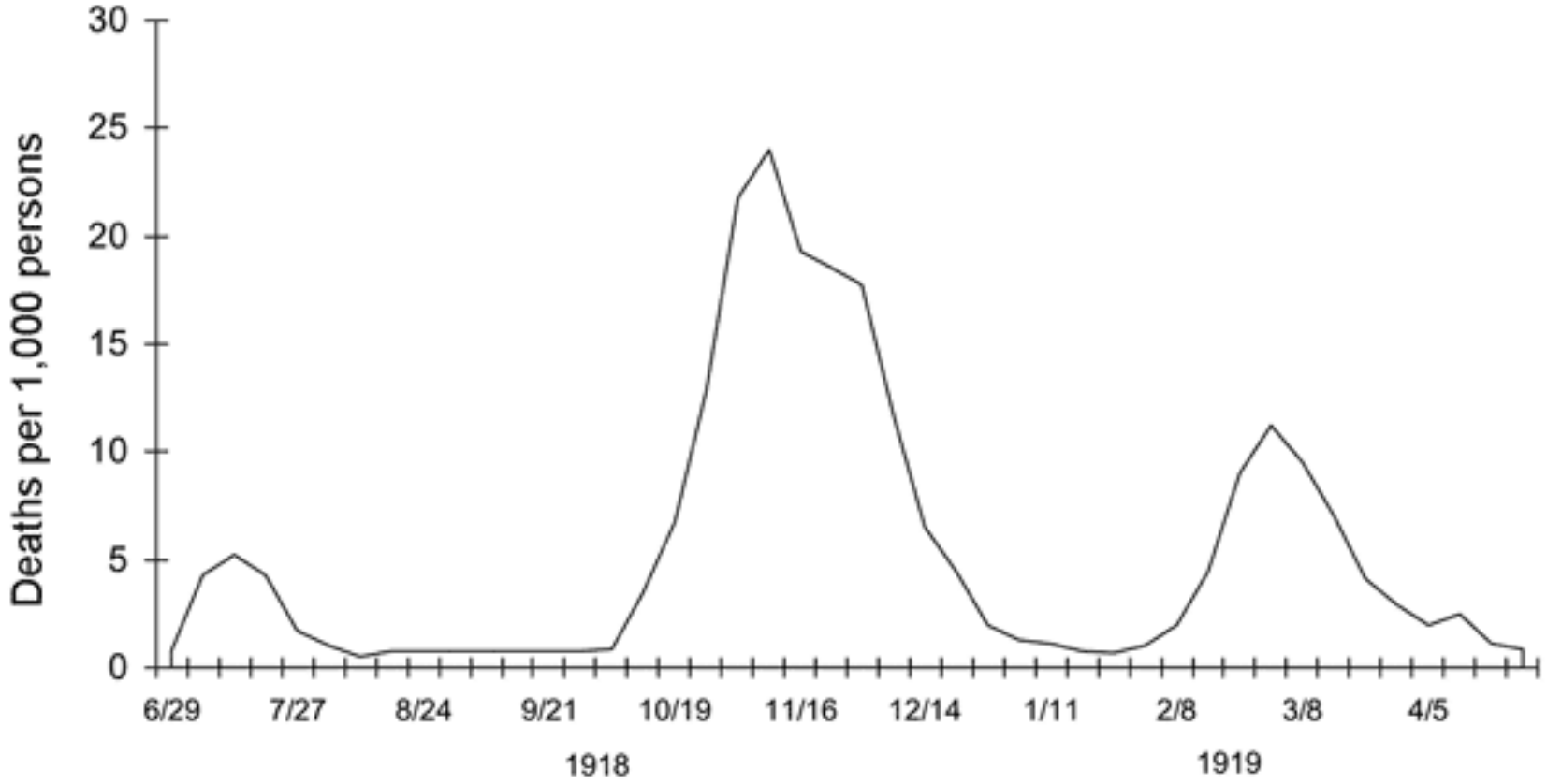
Spatial Spread of Infectious Diseases

Age distribution of influenza deaths for the 1918-1919 pandemic



Spatial Spread of Infectious Diseases

Three pandemic waves: weekly combined influenza and pneumonia mortality, United Kingdom, 1918–1919



Spatial Spread of Infectious Diseases

Well-mixed population: each individual is equally exposed

Structured population: members of one group are more likely to come into contact with each other than with members of other “distant” groups.

Spatial Spread of Infectious Diseases

Spatial structure:

- … affects the speed of the initial epidemic spread
- … is necessary when local interactions or local environment are important
- … is important for disease persistence.

Spatial Spread of Infectious Diseases

Spatial heterogeneity can:

- .. lead to repeated reintroductions
- .. prevent extinction of the disease
- .. enhance persistence at a regional level.

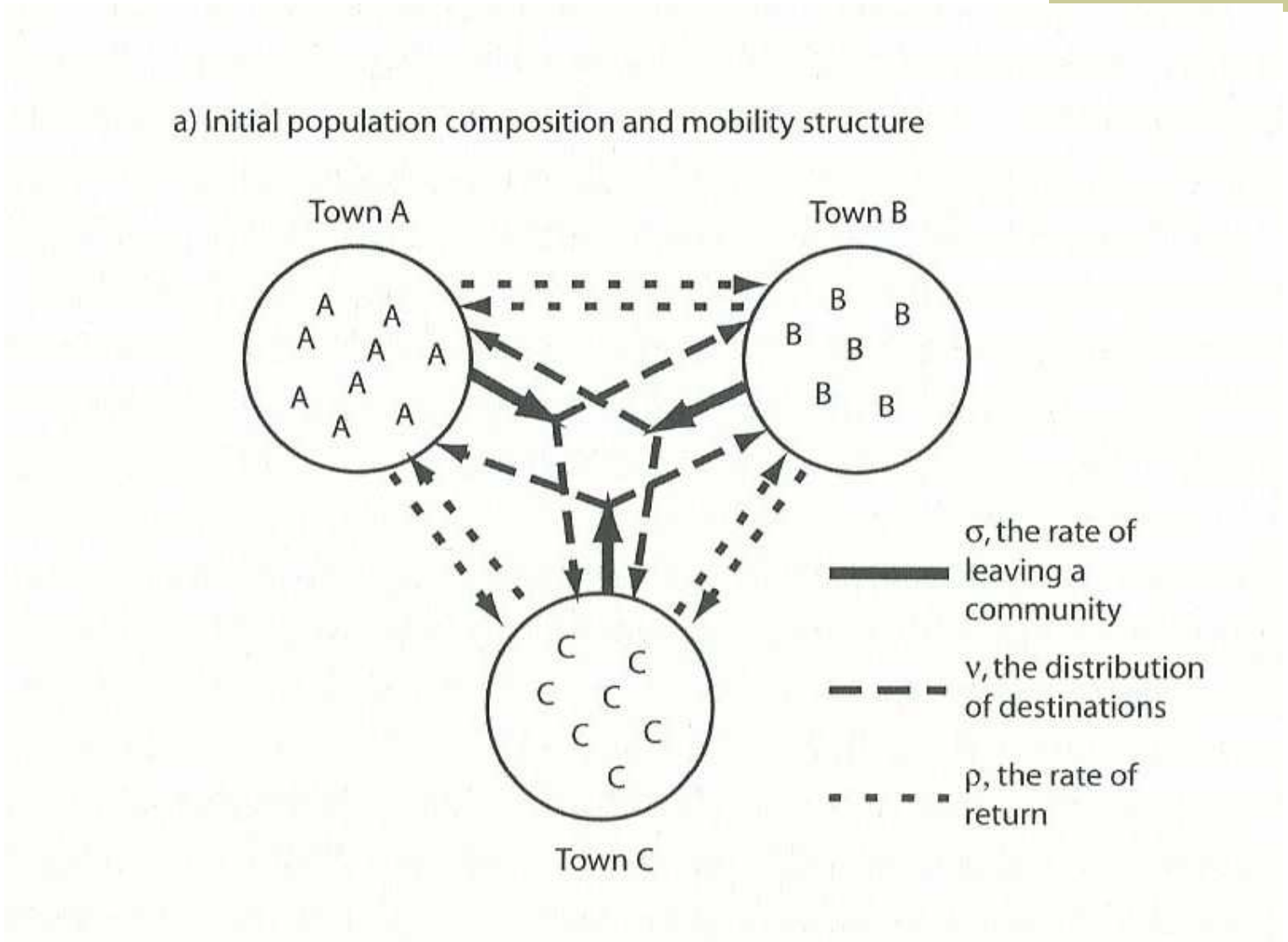
"Rescue" effects can appear if epidemics in different locations do not occur simultaneously (asynchrony).

Spatial Spread of Infectious Diseases

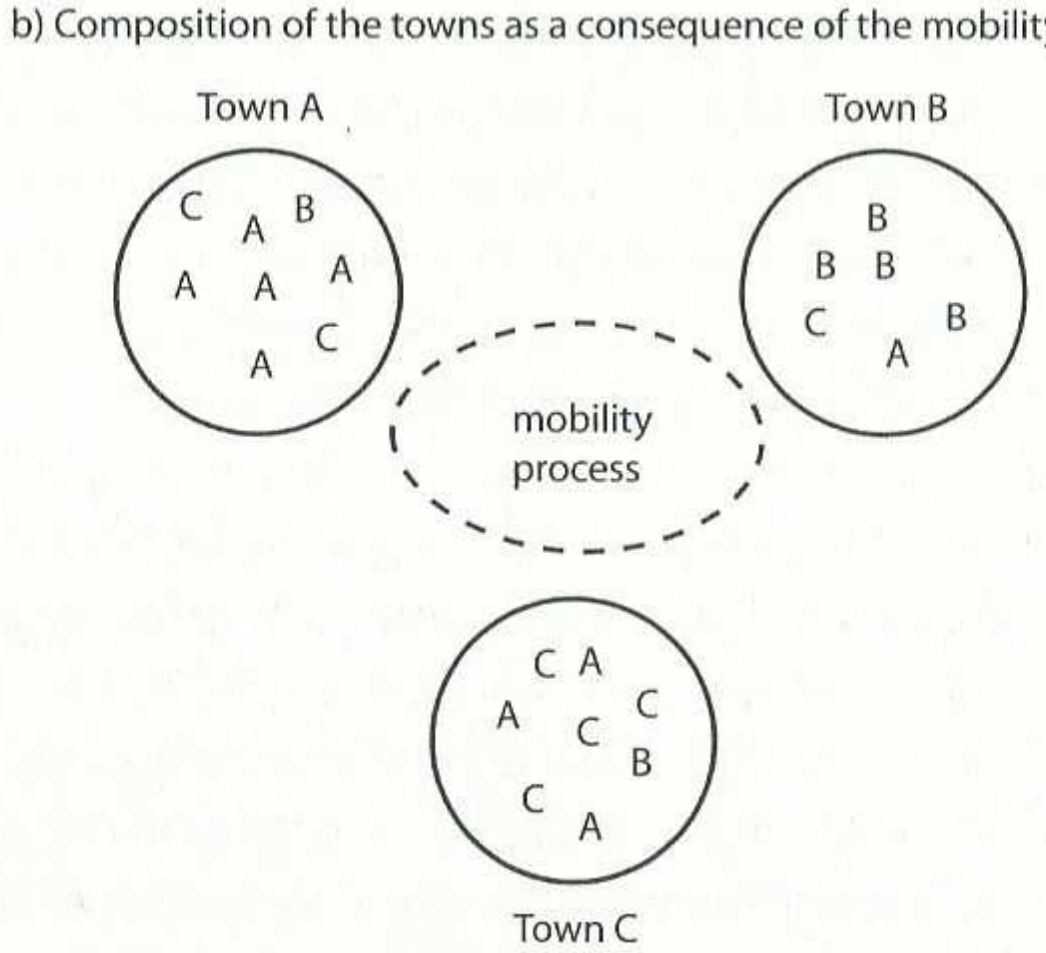
Spatial patterns in disease dynamics can be generated by:

- Asynchrony
- Age distribution of populations
- Seasonally varying transmission rates.

Spatial Spread of Infectious Diseases

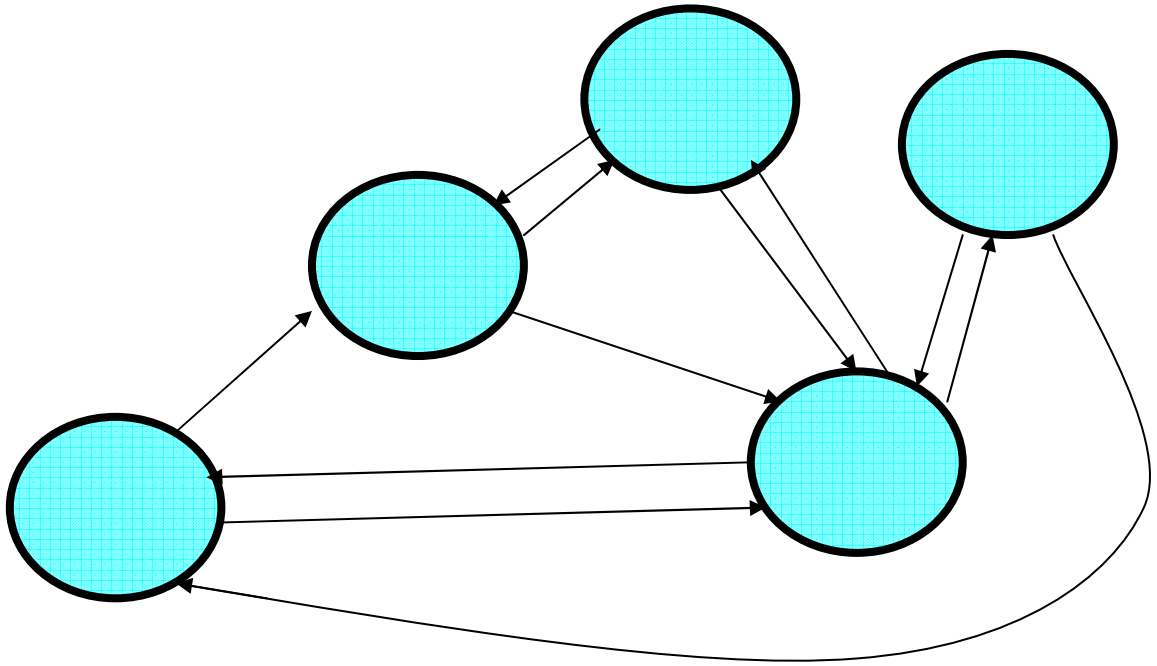


Spatial Spread of Infectious Diseases



Population-based approaches

- Metapopulation or patch models



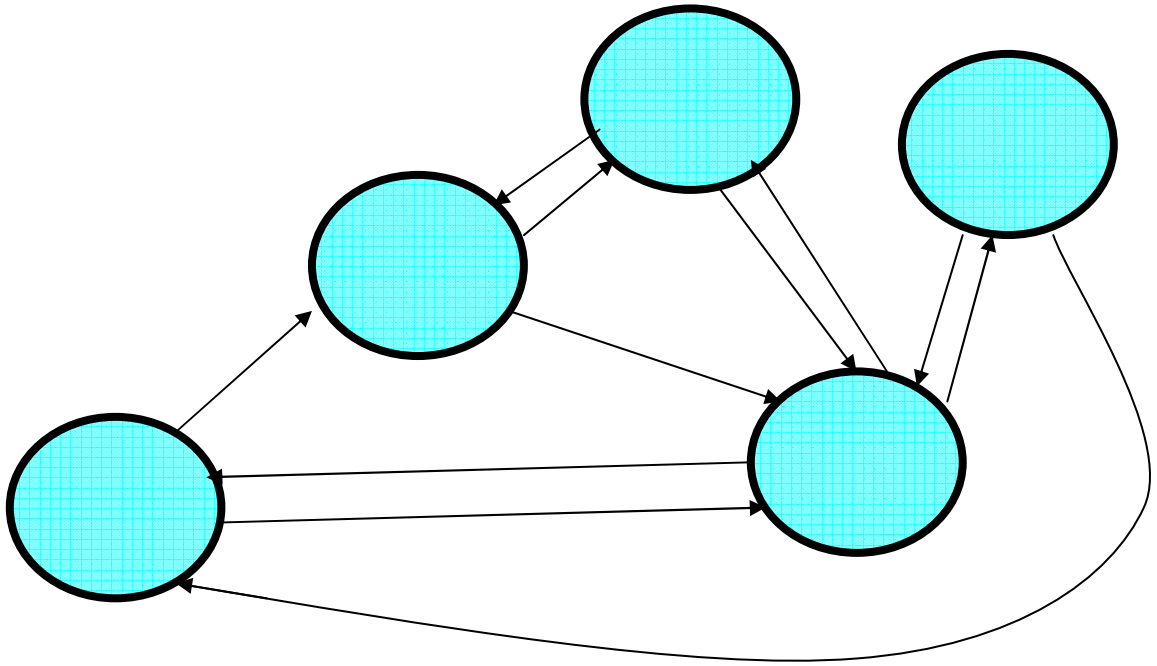
Population-based approaches

- Metapopulation or patch models
- Spatially continuous models

  Landscapes

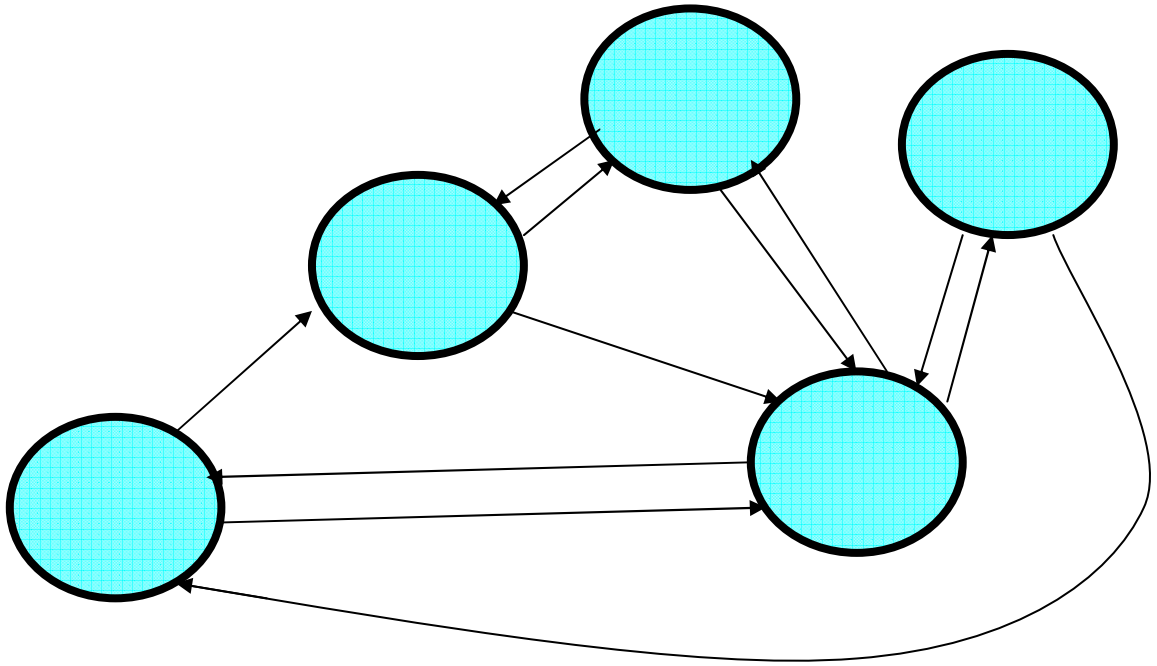
Metapopulation Models

Population is distributed into ***n*** spatially discrete groups linked to one another in some specified way.



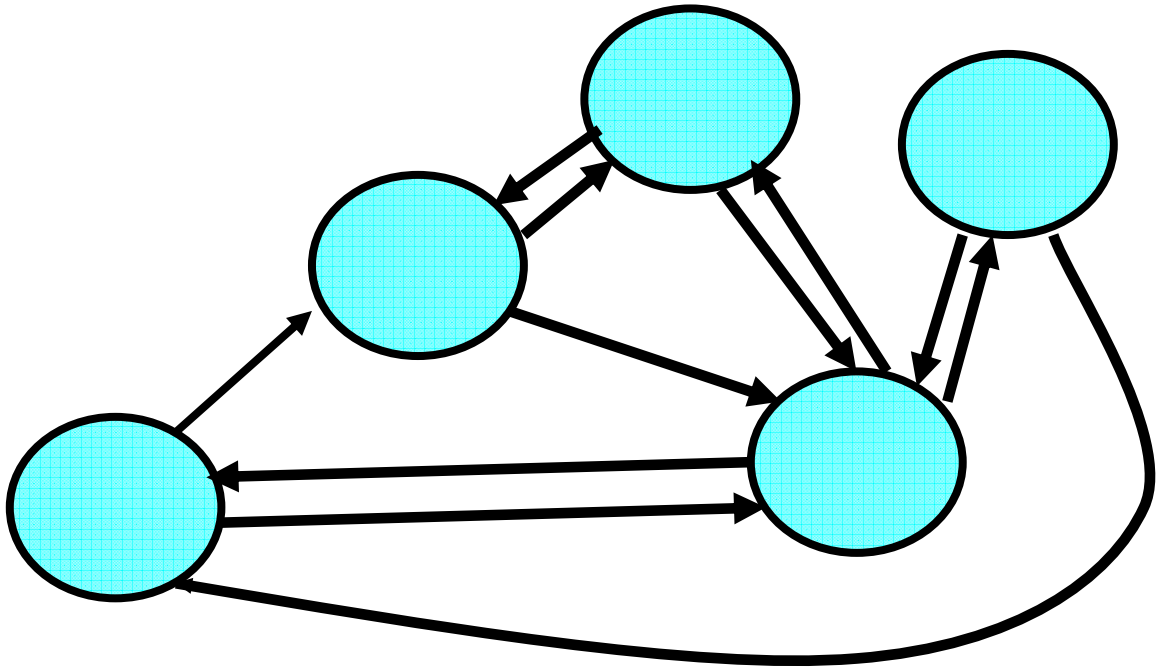
Metapopulation Models

Individuals in a group are assumed well-mixed



Metapopulation Models

Coupling terms represent how infection spreads among groups



Metapopulation Models

Spatial scale is represented through the choice of groups

Hierarchical transmission can be introduced

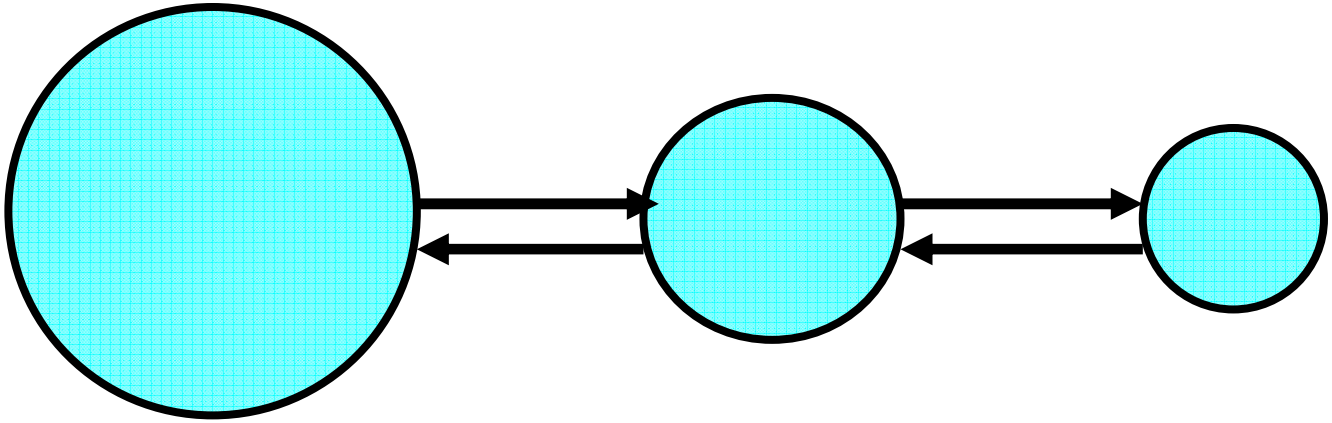
Metapopulation Models

Spatial scale is represented through the choice of groups

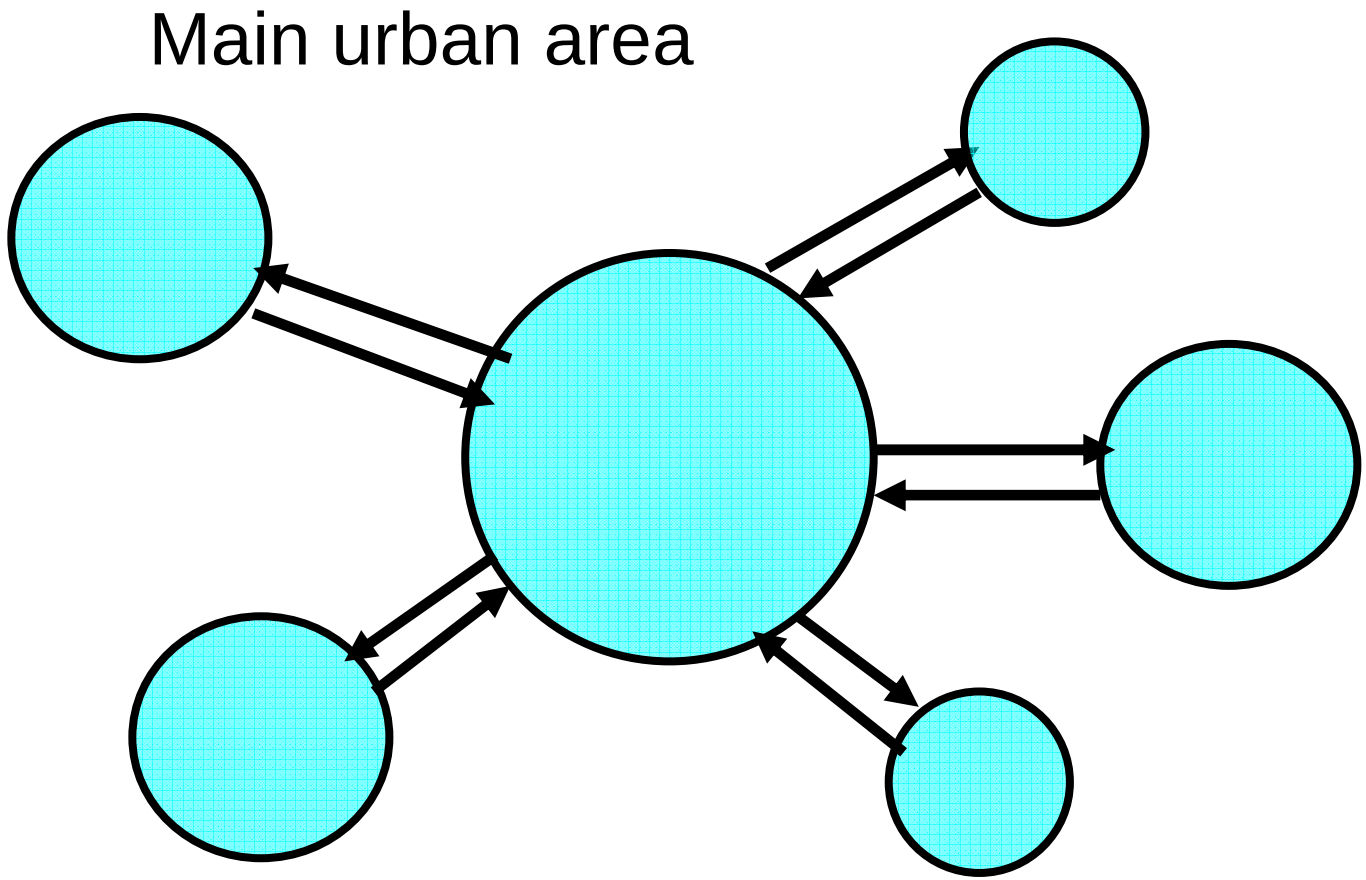
Hierarchical transmission can be introduced

Metapopulation Models

City – Town - Village

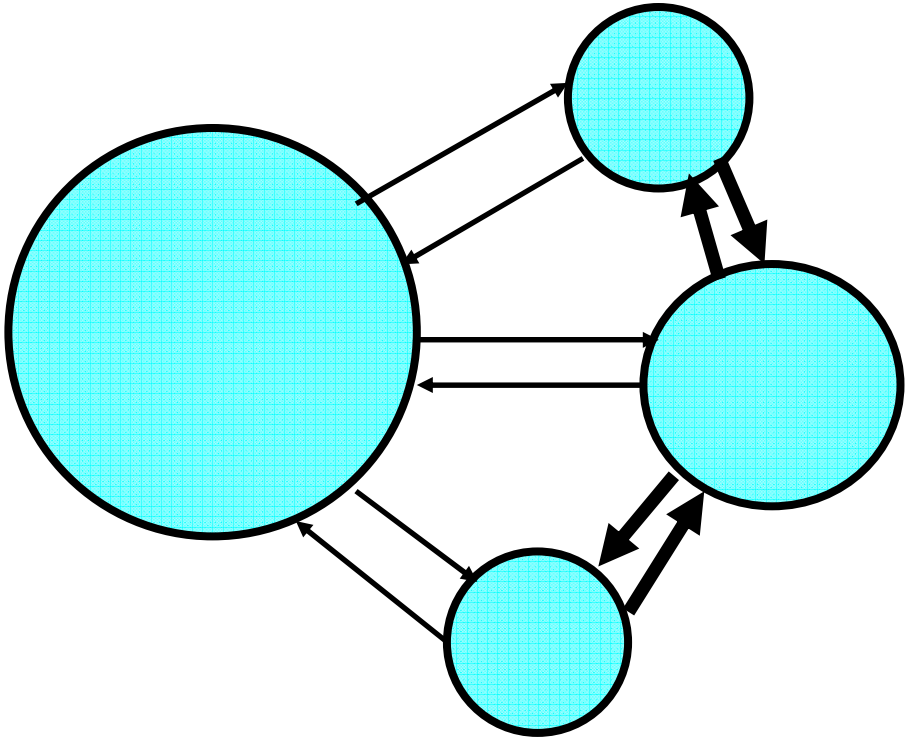


Metapopulation Models



Metapopulation Models

Sister towns



Metapopulation Models

For each subpopulation i the SIR model can be formulated as:

$$\frac{dS_i}{dt} = \nu_i N_i - \lambda_i S_i - \mu_i S_i$$

$$\frac{dI_i}{dt} = \lambda_i S_i - \gamma_i I_i - \mu_i I_i$$

where λ_i includes the transmission within population i and the coupling to other subpopulations.

Metapopulation Models

The force of infection λ_i can be expressed as:

$$\lambda_i = \beta_i \sum_j \rho_{ij} \frac{S_j}{N_i}$$

ρ_{ij} measures the relative strength of transmission from population j to population i.

Metapopulation Models

Plants or sessile hosts: spatial transmission is wind- or vector-borne

$$\lambda_i = \beta_i \sum_j \rho_{ij} I_j$$

Here ρ_{ij} is a decreasing function of the distance between subpopulations j and i.

Metapopulation Models

Plants or sessile hosts: spatial transmission is wind- or vector-borne.

R_0^i for infectious individuals in population i as the expected number of secondary cases generated in all subpopulations:

$$R_0^i = \sum_j \frac{\beta_j \rho_{ji}}{\gamma_i}$$

Transmission from ***i*** to ***j***

Metapopulation Models

Plants or sessile hosts: spatial transmission is wind- or vector-borne.

$$R_0^i = \sum_j \frac{\beta_j \rho_{ji}}{\gamma_i}$$

Dividing population j into two, k and l, implies that

$$\rho_{ji} = \rho_{ki} + \rho_{li}$$

So R_0^i does not change.

Metapopulation Models

Plants or sessile hosts: spatial transmission is wind- or vector-borne.

$$R_0^i = \sum_j \frac{\beta_j \rho_{ji}}{\gamma_i}$$

However, **adding** more subpopulations (more hosts) increases R_0^i

More pathogens can be intercepted by additional hosts.

Metapopulation Models

Individuals migrate: the SIR-type model can be expressed as:

$$\frac{dS_i}{dt} = \nu_i - \beta_i S_i I_i - \mu_i S_i + \sum_j m_{ij} S_j - \sum_j m_{ji} S_i$$

$$\frac{dI_i}{dt} = \beta_i S_i I_i - \gamma_i I_i - \mu_i I_i + \sum_j m_{ij} I_j - \sum_j m_{ji} I_i$$

where m_{ij} is the migration rate from j to i

Metapopulation Models

Individuals migrate:

It is frequently assumed that $m_{ij}=m_{ji,}$, however this is not always true.

When $\beta_i = \beta \quad \text{and} \quad \gamma_i = \gamma \quad \forall i,$

$\text{and } N_i = N_j \text{ for } i \neq j$

then

$$R_0 = \frac{\beta N}{\gamma + \mu}$$

Independent of the coupling strength

Metapopulation Models

Commuters: live in population ***j*** but travel occasionally to population ***i***

S_{ij}, I_{ij}, N_{ij} represent the number of susceptibles, infected and total hosts currently in population ***i*** that live in population ***j*.**

Metapopulation Models

Commuters: live in population ***j*** but travel occasionally to population ***i***

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

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

Metapopulation Models

Commuters: live in population ***j*** but travel occasionally to population ***i***

$$\frac{dS_{ii}}{dt} = \nu_{ii} - \beta_i S_{ii} \frac{\sum_j I_{ij}}{\sum_j N_{ij}} - \sum_j l_{ji} S_{ii} + \sum_j r_{ji} S_{ji} - \mu_{ii} S_{ii}$$

$$\frac{dS_{ij}}{dt} = \nu_{ij} - \beta_i S_{ij} \frac{\sum_j I_{ij}}{\sum_j N_{ij}} + l_{ij} S_{jj} - r_{ij} S_{ij} - \mu_{ij} S_{ij}$$

Metapopulation Models

Commuters: live in population ***j*** but travel occasionally to population ***i***

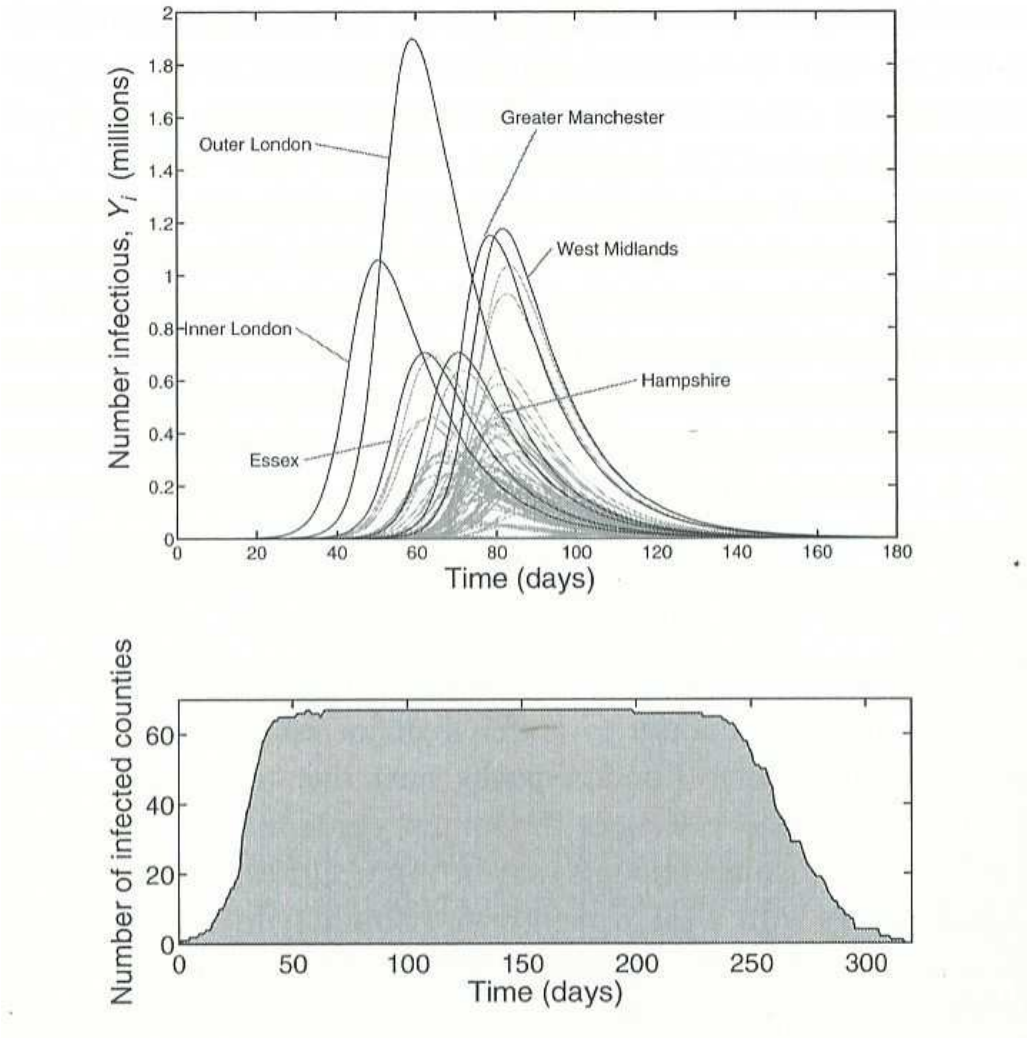
$$\frac{dI_{ii}}{dt} = \beta_i S_{ii} \frac{\sum_j I_{ij}}{\sum_j N_{ij}} - \gamma I_{ii} - \sum_j l_{ij} I_{ii} + \sum_j r_{ji} I_{ji} - \mu_{ii} I_{ii}$$

$$\frac{dI_{ij}}{dt} = \beta_i S_{ij} \frac{\sum_j I_{ij}}{\sum_j N_{ij}} - \gamma I_{ij} + l_{ij} I_{jj} - r_{ij} I_{ij} - \mu_{ij} I_{ij}$$

Metapopulation Models

Epidemics initiated with 10 cases in Inner London.

Data taken fron 1991 census database and all trips were short duration (r=2)



Metapopulation Models

Commuters: live in population ***j*** but travel occasionally to population ***i***

This gives a total of $3n^2$ equations for n populations.

Metapopulation Models

Commuter aproximations: simplify the model.

Keeling and Rohani take 2 populations of equal size and epidemiological characteristics and assume that commuter movements are very rapid.

In this case:

Coupling parameter

$$\lambda_i = \beta_i((1-\rho)I_i + \rho I_j)$$

Metapopulation Models

Commuter aproximations:

If q is the proportion of time that individuals spend in the other population, then

$$q = \frac{l_{ij}}{r_{ji} + l_{ij}} = \frac{l_{ji}}{r_{ij} + l_{ji}}$$

Metapopulation Models

Commuter aproximations:

The coupling parameter ρ can be defined as:

$$\rho = 2q(1-q)$$

because when either the susceptibles of one population or the infected of the other move (not both), there is a transfer of pathogen.

ρ is maximized when q=0.5

Metapopulation Models

Rapid commuter movements of individuals from their home subpopulation to another subpopulation and back are important in the spread of human diseases.

Hence, models need to include both the current location and the home location of individuals. When movements are of short duration this can be aproximated by simple coupling.

Here R_0 is independent of the coupling

Metapopulation Models

Coupling and Synchrony

The correlation between the disease dynamics in two subpopulation is generally a sigmoidal function of the interaction between them

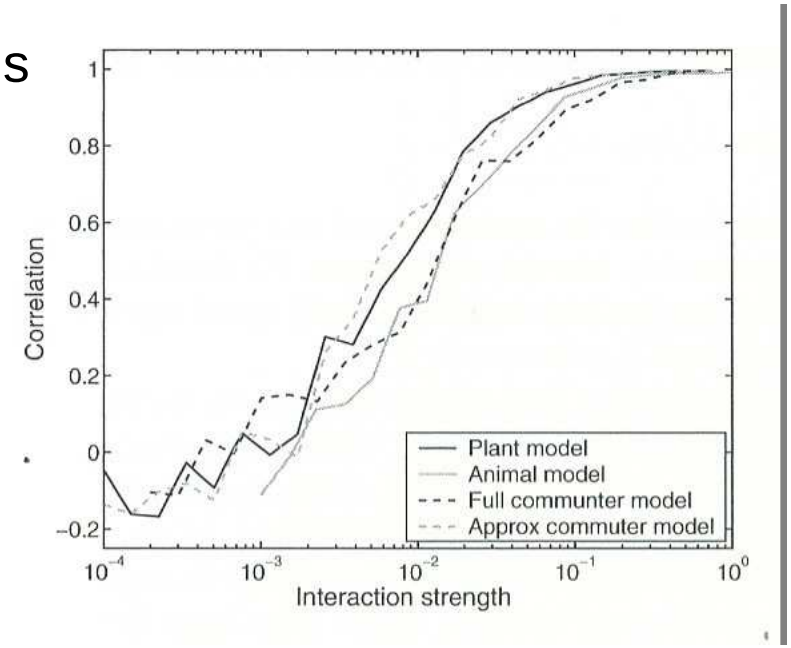
Stochastic models for 2 populations

$\gamma = 0.1 \quad \beta = 1$

$\mu = 10^{-4} \quad \nu = 1$

$N = 10^4$

$\varepsilon = 10^{-2}$ Rate of imports



Metapopulation Models

Coupling and Synchrony

The scalings between the interaction strengths are aprox.

$$\rho_{plants} \approx \frac{m}{\gamma + m} \approx 2\frac{l}{r+l}\left(1 - \frac{l}{r+l}\right) \approx \rho_{commuters}$$

Metapopulation Models

Extinction and Rescue Effects

Smaller populations will have fewer infected individuals and thus be more prone to stochastic effects.

Extinction risk decreases exponentially as population size increases.

Metapopulation Models

Extinction and Rescue Effects

In a metapopulation subdivided in many small isolated populations with no interaction or coupling, the disease in each subpopulation will be driven extinct, leading to a swifter global eradication than if coupling is strong and the metapopulation is well mixed.

Metapopulation Models

Extinction and Rescue Effects

Suppose N is population size and rate of extinction is proportional to $exp(-\varepsilon N)$.

Then:

$$\text{Time to extinction} = \frac{k}{e^{-\varepsilon N}} = ke^{\varepsilon N}$$

Metapopulation Models

Extinction and Rescue Effects

Suppose N is divided into n small noninteracting subpopulations size, then for all populations to become disease-free:

$$\text{Time to extinction} = \frac{k}{ne^{-\varepsilon\frac{N}{n}}} + \frac{k}{(n-1)e^{-\varepsilon\frac{N}{n}}} + \ldots + \frac{k}{e^{-\varepsilon\frac{N}{n}}}$$

$$< ke^{\varepsilon\frac{N}{n}}(1+\log(n))$$

Metapopulation Models

Extinction and Rescue Effects

When subpopulations interact, the infection can be reintroduced into a disease-free subpopulation.

Long-term persistence can be observed because infections is constantly reinvading.

Rescue events

Metapopulation Models

Extinction and Rescue Effects

Rescue events are maximized when:

High rate of infection ⇐ Strong coupling

Asynchrony ⇐ Weak coupling

Persistence can be maximized at **intermediate** levels of coupling

Metapopulation Models

When interaction between the subpopulations is included, the level of local and global extinctions is an emergent property of the dynamics and cannot be easily predicted from the disease parameters

It is necessary to understand how population structure, disease dynamics, population size, infectious import rate, and coupling interact to determine disease persistence.

Metapopulation Models

Vaccination

- Reduces prevalence of infection
- Increases risk of extinction
- Reduces the effective interaction strength

→ Less synchrony in the disease dynamics

→ More effective rescue events

Metapopulation Models

Vaccination

Pulsed vaccination campaigns act to synchronize epidemics in coupled populations and may lead to an increase in global extinction rates.

Timing of vaccination pulses?

- Frequent pulses limit the buildup of susceptibles
- Infrequent pulses have a greater synchronizing action

Metapopulation Models

Vaccination

Two coupled populations with SIR-type infection.

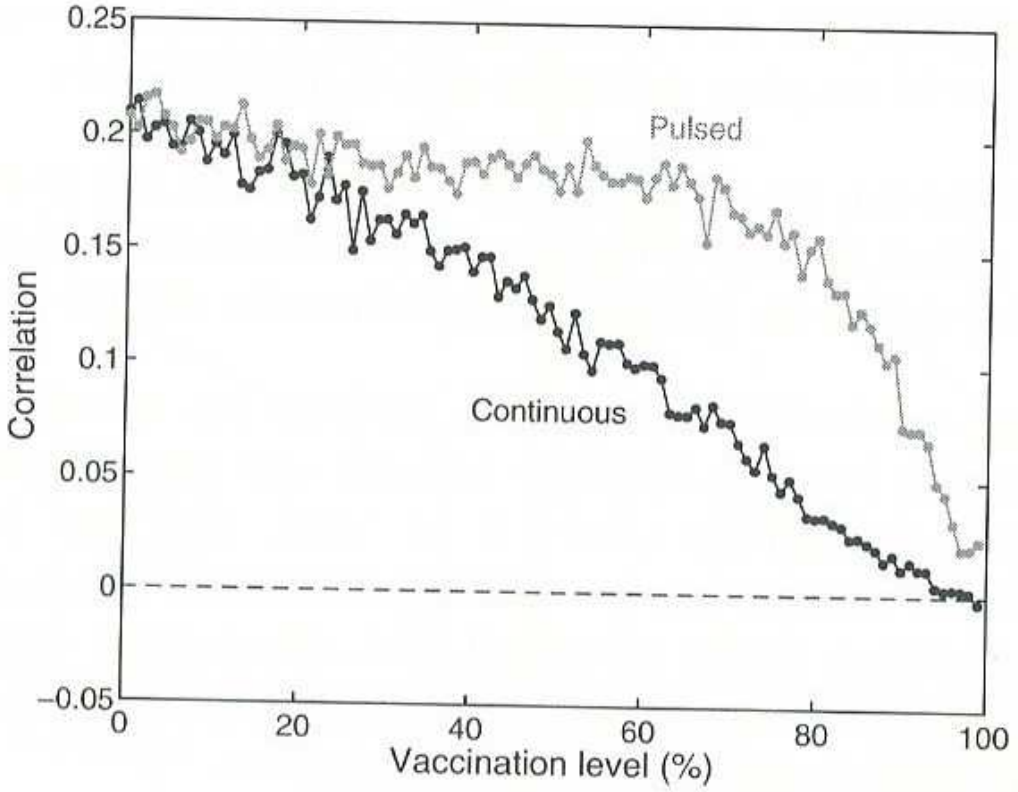
$$N = 10^5$$

$$\rho = 0.01$$

$$\frac{1}{\gamma} = 10d^{-1}$$

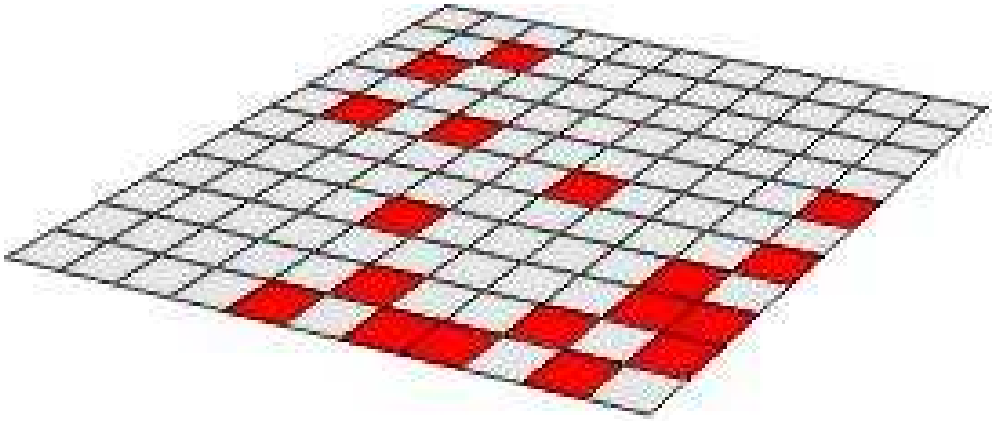
$$R_0 = 10$$

$$\delta = 5y^{-1}$$



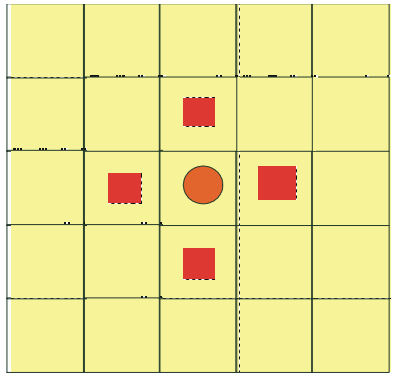
Lattice- or Grid-based Models

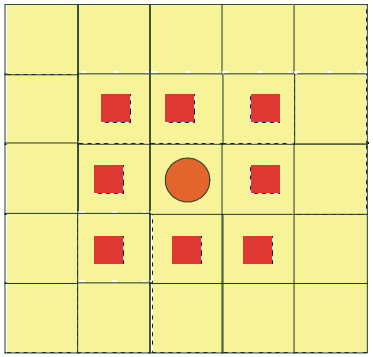
The spatial location of the hosts is considered important, but the population cannot be partitioned into discrete subpopulations



Individuals within a grid site are grouped into a subpopulation

Lattice- or Grid-based Models





Two-dimension square lattice

Higher dimensional lattices can be used to replicate more complex social structure.

Hexagonal grids have also been used.

Lattice- or Grid-based Models

Commuter-like interactions

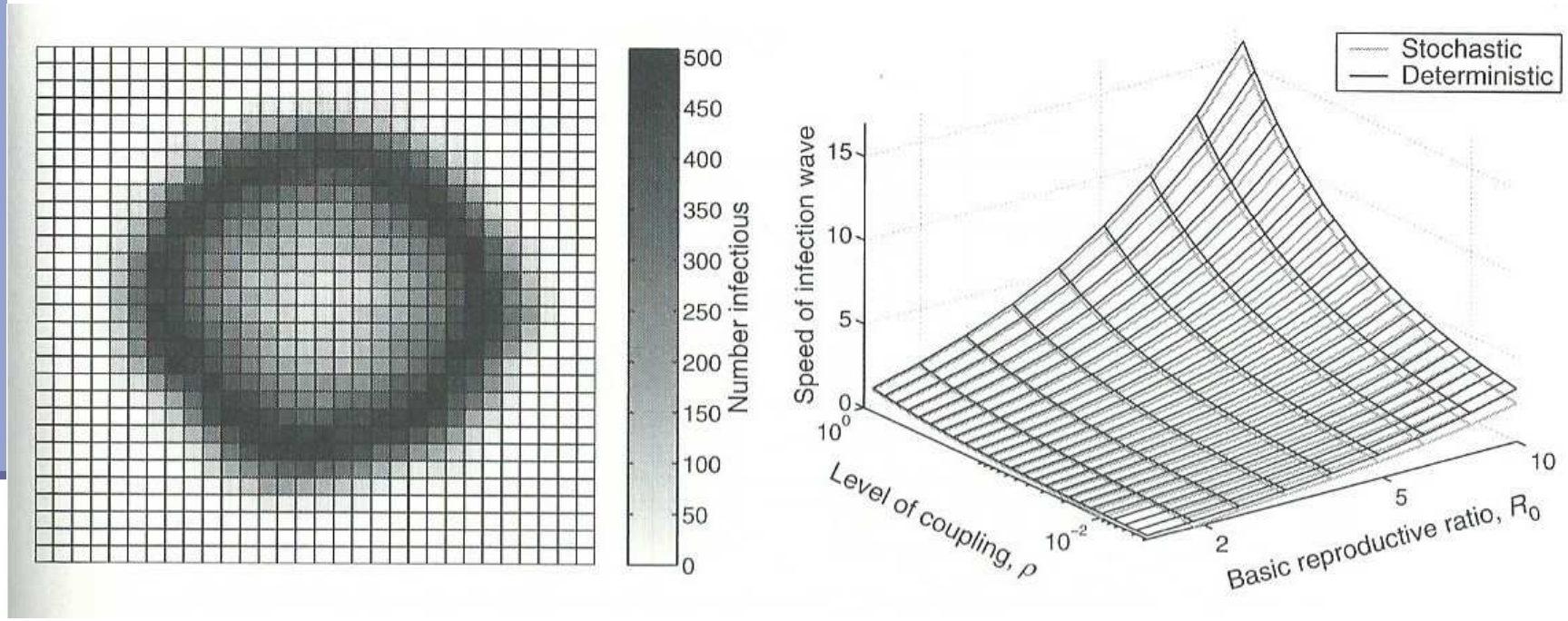
$$\frac{dS_i}{dt} = \nu - \beta S_i \frac{(1-\sum_j \rho_{ji}) I_i + \sum_j \rho_{ij} I_j}{(1-\sum_j \rho_{ji}) N_i + \sum_j \rho_{ij} N_j} - \mu S_i$$

$$\frac{dI_i}{dt} = \beta S_i \frac{(1-\sum_j \rho_{ji}) I_i + \sum_j \rho_{ij} I_j}{(1-\sum_j \rho_{ji}) N_i + \sum_j \rho_{ij} N_j} - \gamma I_i - \mu I_i$$

$$\rho_{ij} = \rho_{ji} = \begin{cases} \rho & \text{if } i \text{ and } j \text{ are neighbors} \\ 0 & \text{otherwise} \end{cases}$$

Lattice- or Grid-based Models

Commuter-like interactions



Lattice- or Grid-based Models

Coupled lattice models show a wave-like spread of infections across a homogeneous landscape.

However, the do not capture the expected individual level behavior

The grid size must be chosen with extreme care

Lattice- or Grid-based Models

Cellular Automata

A cell can have only a finite, usually small, number of population states.

Each cell is usually either empty or occupied by a susceptible, infectious or recovered individual. Interactions occur only with 4 or 8 neighboring lattice sites

Almost all cellular automata disease models are stochastic.

Lattice- or Grid-based Models

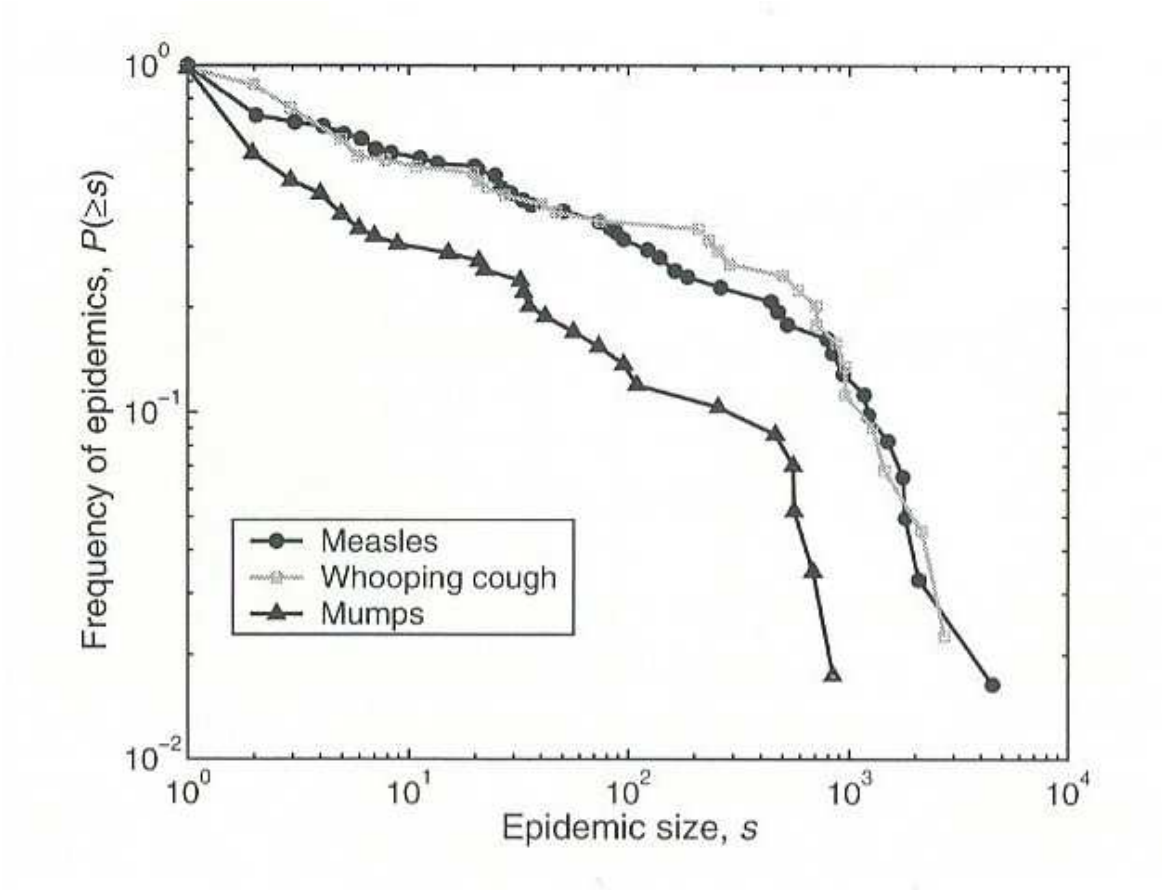
Cellular Automata

Cellular automata disease models are abtract models that do not incorporate realistic human behavior.

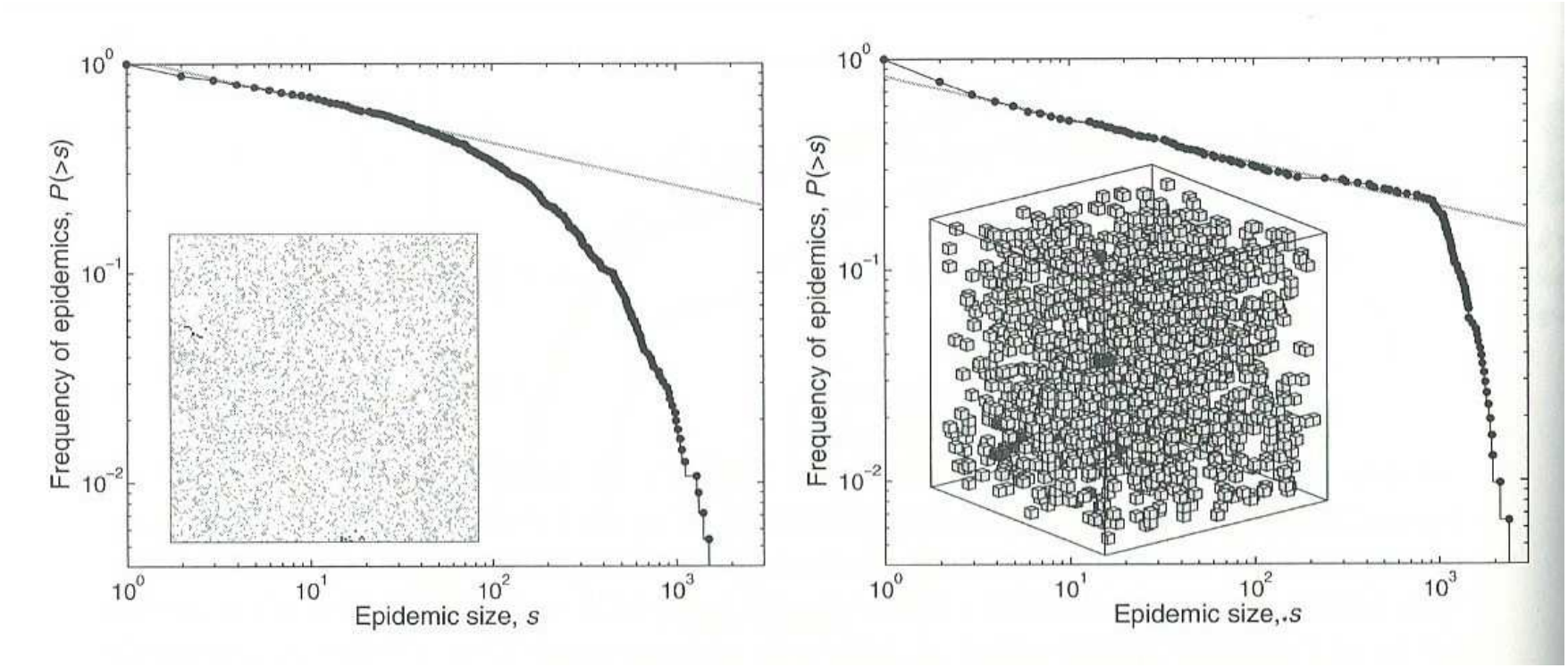
They are abstract tools that can be used for understanding the spatial dynamics of transmission but are not predictive models

Lattice- or Grid-based Models

Power Law



Lattice- or Grid-based Models



Questions and ….