Modeling infectious diseases using agent-based models and population dynamics

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Introduction

- Computational modeling is a powerful tool for understanding disease outbreaks.
- Accurate models can be used to predict the implication of public health measures (i.e., forcast how effective a vaccine will be).

A real world scenario

During the 2009 H1N1 flu pandemic, researchers used models to allocate resources and to design effective public health strategies.

H1N1 2009 pandemic in numbers

In the United States, CDC[1][2] estimated

- Between 43 million and 89 million cases of H1N1.
- Between 195,000 and 403,000 H1N1-related hospitalizations.
- Between 8,870 and 18,300 2009 H1N1-related deaths

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[1]Centers for Disease Control and Prevention. Updated CDC Estimates of 2009 H1N1 Influenza Cases, Hospitalizations and Deaths in the United States, April 2009—April 10, 2010, 2010. [Online; accessed 6-May-2012]

[2]Centers for Disease Control and Prevention. Estimates of the Prevalence of Pandemic (H1N1) 2009, United States, April–July 2009, 2009. [Online; accessed 6-May-2012]

H1N1 2009 profile

- Fatality rates between 0.01 and 0.03%. Higher risks for people under 50. [3]
- Basic reproduction number R_0 estimated to be 1.75. [4] R_0 is the average number of secondary cases produced by a primary case. Diseases with $R_0 > 0$ can spread across a population.
- Infectious period usually last 4–6 days.
- 33% of the infectious individuals are asymptomatic.

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[3] L.J. Donaldson, P.D. Rutter, B.M. Ellis, F.E.C. Greaves, O.T. Mytton, R.G. Pebody, and I.E. Yardley. Mortality from pandemic A/H1N1 2009 influenza in England: public health surveillance study. *BMJ: British Medical Journal*, 339, 2009

[4] D. Balcan, H. Hu, B. Goncalves, P. Bajardi, C. Poletto, J. Ramasco, D. Paolotti, N. Perra, M. Tizzoni, W. Broeck, et al. Seasonal transmission potential and activity peaks of the new influenza A (H1N1): a Monte Carlo likelihood analysis based on human mobility. *BMC medicine*, 7(1):45, 2009

H1N1 profile table

Table: Best Estimates of the epidemiological parameters

Parameter	Best Estimate	Interval estimate
R_0	1.75	1.64 to 1.88
ν^{-1}	2.5	1.1 to 4.0
α^{-1}	1.1	1.1 to 2.5

Modeling an epidemic

Many parameters influence how a disease spreads.

- Sex, age, education distributions
- Transportation networks
- Vaccination campaigns, quarantines, and other public health measures
- Evolution
- Seasonal forcing
- Social structures
- etc.

Compartmental models in epidemiology

The SIR model[5] is a good and simple model for many infectious diseases.

- Represents a population in 3 distinct compartments at a particular time: susceptibles $S(t)$, infectious $I(t)$, and recovered $R(t)$.
- Rate of transitions of a population is modeled by differential equations.

[5] R.M. Anderson, R.M. May, et al. Population biology of infectious diseases: Part i. *Nature*, 280(5721):361, 1979

Basic SIR model

Basic SIR model

$$\frac{dS}{dt} = -\beta IS$$
$$\frac{dI}{dt} = \beta IS - \nu I$$
$$\frac{dR}{dt} = \nu I$$

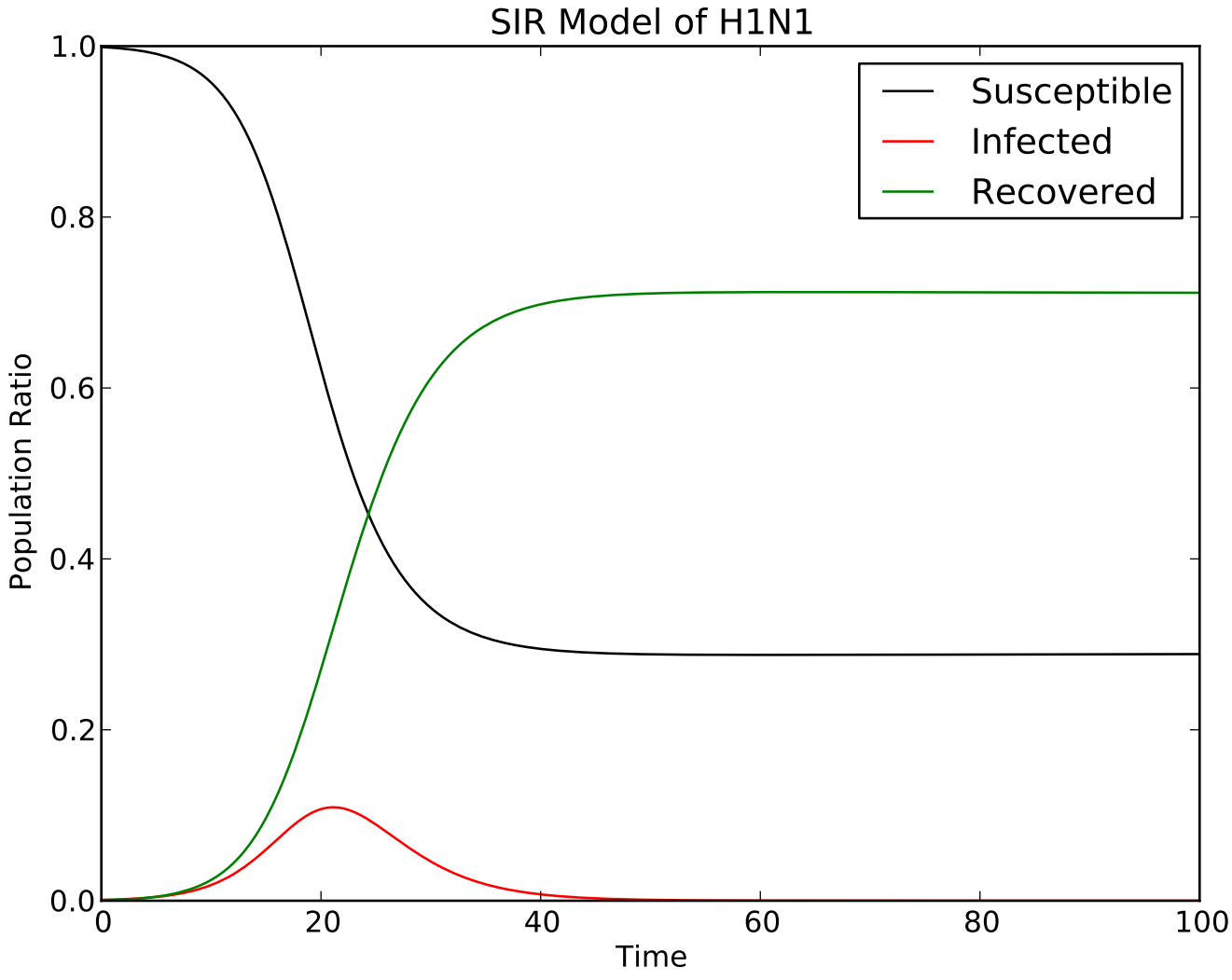
where the basic reproduction number R_0 is defined to be

$$R_0 = \frac{\beta}{\nu}$$

SIR model characteristics

- Continuous time model.
- Deterministic.
- Computationally cheap.
- Do not account for spatial distributions, assume uniform diffusion.

SIR Results


SIR Model of H1N1
Susceptible
Infected
Recovered
Population Ratio
Time
1.0
0.8
0.6
0.4
0.2
0.0
0
20
40
60
80
100

Agent-based modeling

- Model a system as a collection of autonomous decision-making entities called agents.[6]
- Agents may be capable of sophisticated behaviors.
- Capture emergent phenomena.
- Natural description of a system.
- Flexible.

[6] E. Bonabeau. Agent-based modeling: Methods and techniques for simulating human systems. *Proceedings of the National Academy of Sciences of the United States of America*, 99(Suppl 3):7280, 2002

Agent-based model characteristics

Typically,

- Discrete time model.
- Stochastic.
- Account for spatial distributions.
- Computationally expensive.

Simple agent-based model of an infectious disease

- We define the state of an agent as its position and velocity in the world and its compartment (i.e., susceptible, infectious, recovered).
- World is a continous 2d space
- Susceptible agents become infected if they move within the radius r of another infected agent.
- Infected agents recovers with probability γ.

Goal

Find a correspondence between the SIR model and this agent-based model.

Model correspondence

- This will allow us to interchange models as necessary.
- In practice, the parameters space of the agent-based model is difficult to define (e.g., how the rules of the agents fit in?).
- Or may not even exist, how do we map a deterministic world onto a stochastic world?

Simple parametrizations of the models

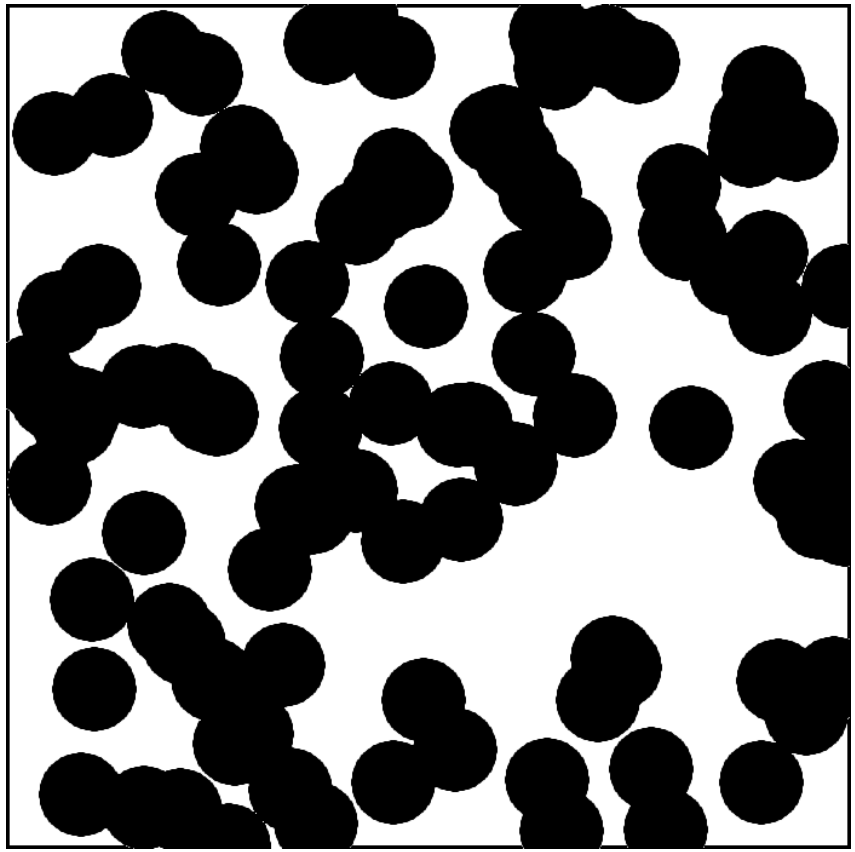
- For the agent-based model, we ignored the motion of the agents. So we only have the radius r and recovery probability γ as parameters.
- For the SIR model, we used infection rate β and recovery rate ν as parameters.

Finding the parameters

We found trivially that $\nu = \gamma$. But, finding β was harder. Measuring it experimentally was not really an option.

Finding the infection rate

What is the probability a point fall in the black region?



Finding the infection rate (1)

Assume an agent has a probability $\frac{a}{A}$ of being in the infectious area a of some agent in a world of area A, then the probability of *not* being in it is $\frac{A-a}{A}$.

Finding the infection rate (2)

So the probability of not being covered by k independent infectious agents is

$$\left(\frac{A-a}{A}\right)^k$$

Finding the infection rate (3)

And the probability of being covered by at least one of them is

$$1 - \left(\frac{A - a}{A} \right)^k$$

Finding the infection rate (4)

Our model, we assume $A = 1$ and we have $a = \pi r^2$. Let S_t be the number of susceptibles and I_t be the number of infected at time t, then the expected number of infected at time $t + 1$ is

$$I_{t+1} = I_t + S_t(1 - (1 - \pi r^2)^{I_t})$$

Finding the infection rate (5)

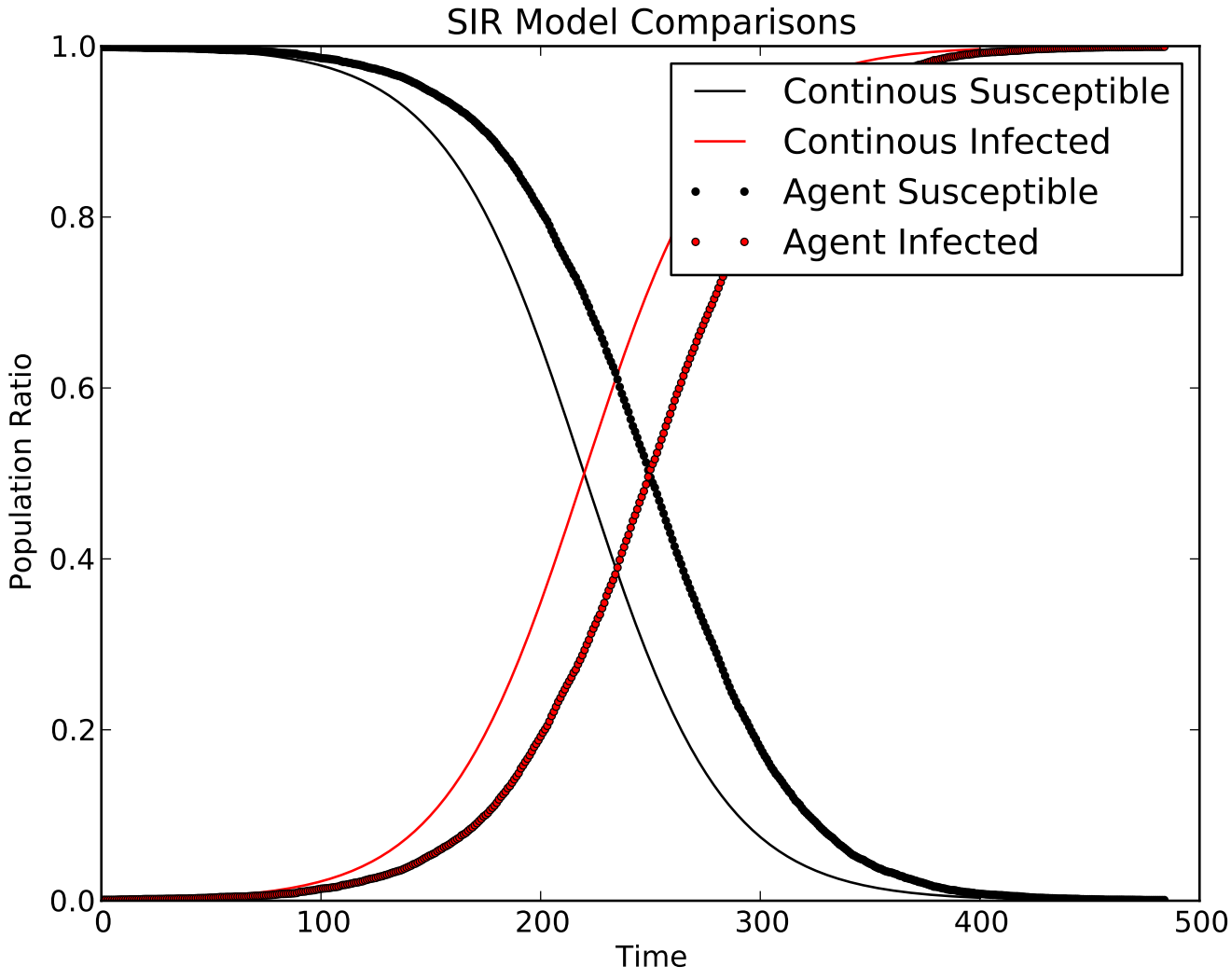
We took the Taylor expansion and made some resonable approximations to find

$$I_{t+1} \approx I_t - \ln(1 - \pi r^2) I_t S_t$$

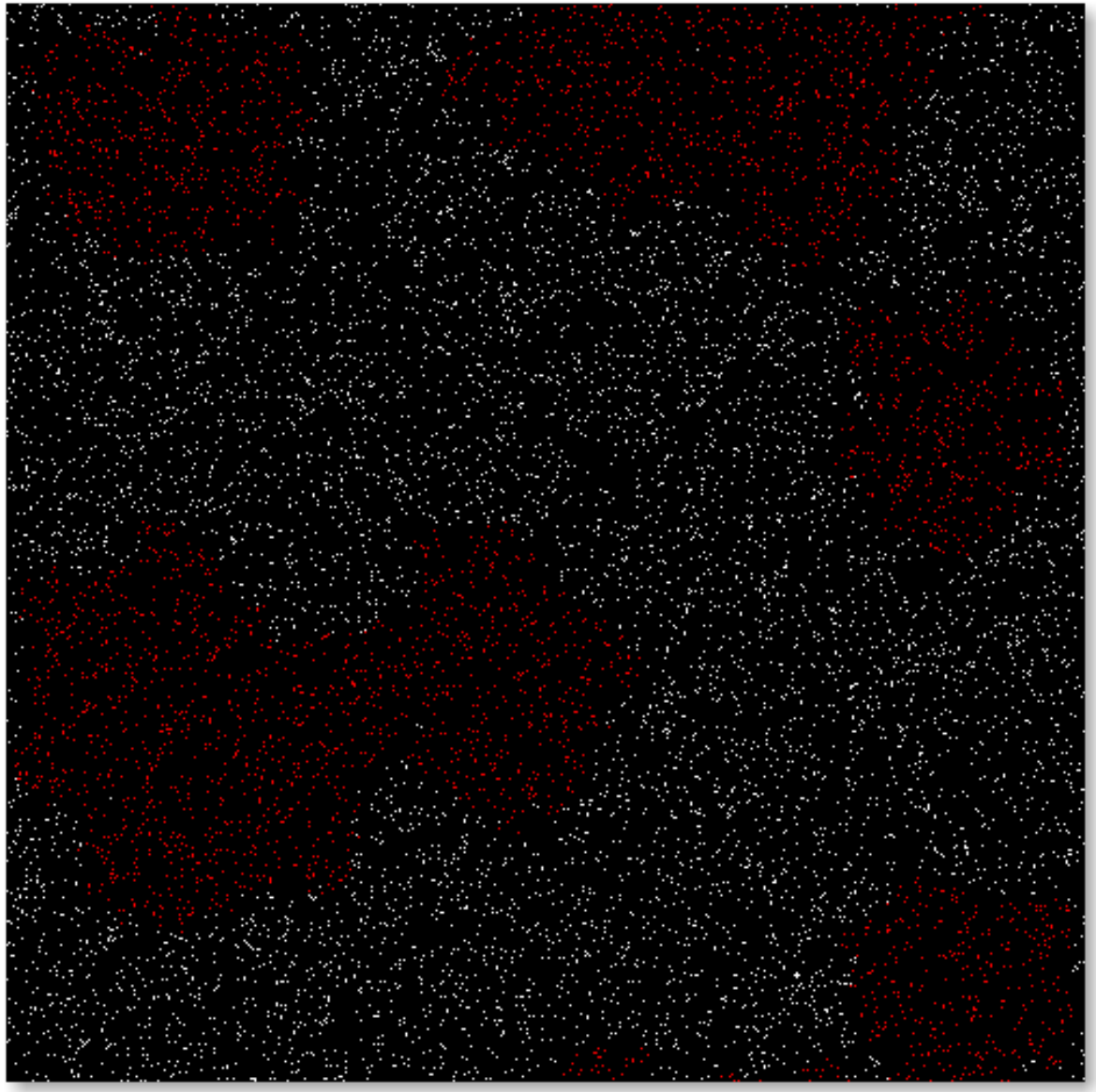
Therefore,

$$\beta \approx -\ln(1 - \pi r^2)$$

Comparison of infection rates with $r = 0.001$



Result with $r = 0.01$



Limits of our simple analysis

- Need PDEs and differential geometry to model forest fire dynamics analytically.

$$\frac{\partial \phi}{\partial t} = F\|\nabla \phi\|$$

- Show that very complex behaviours emergent even simple rules.

Conclusion

- Output of the system depends on the initial conditions.
- Limited forcasting capabilities for both models.
- Correspondence not obvious between agent-based model and populations.

Questions?