WELCOME!

Network Modeling for Epidemics

Martina Morris, Ph.D.
Steven M. Goodreau, Ph.D.
Samuel M. Jenness, Ph.D.
Darcy Rao (UW Epi PhD program)

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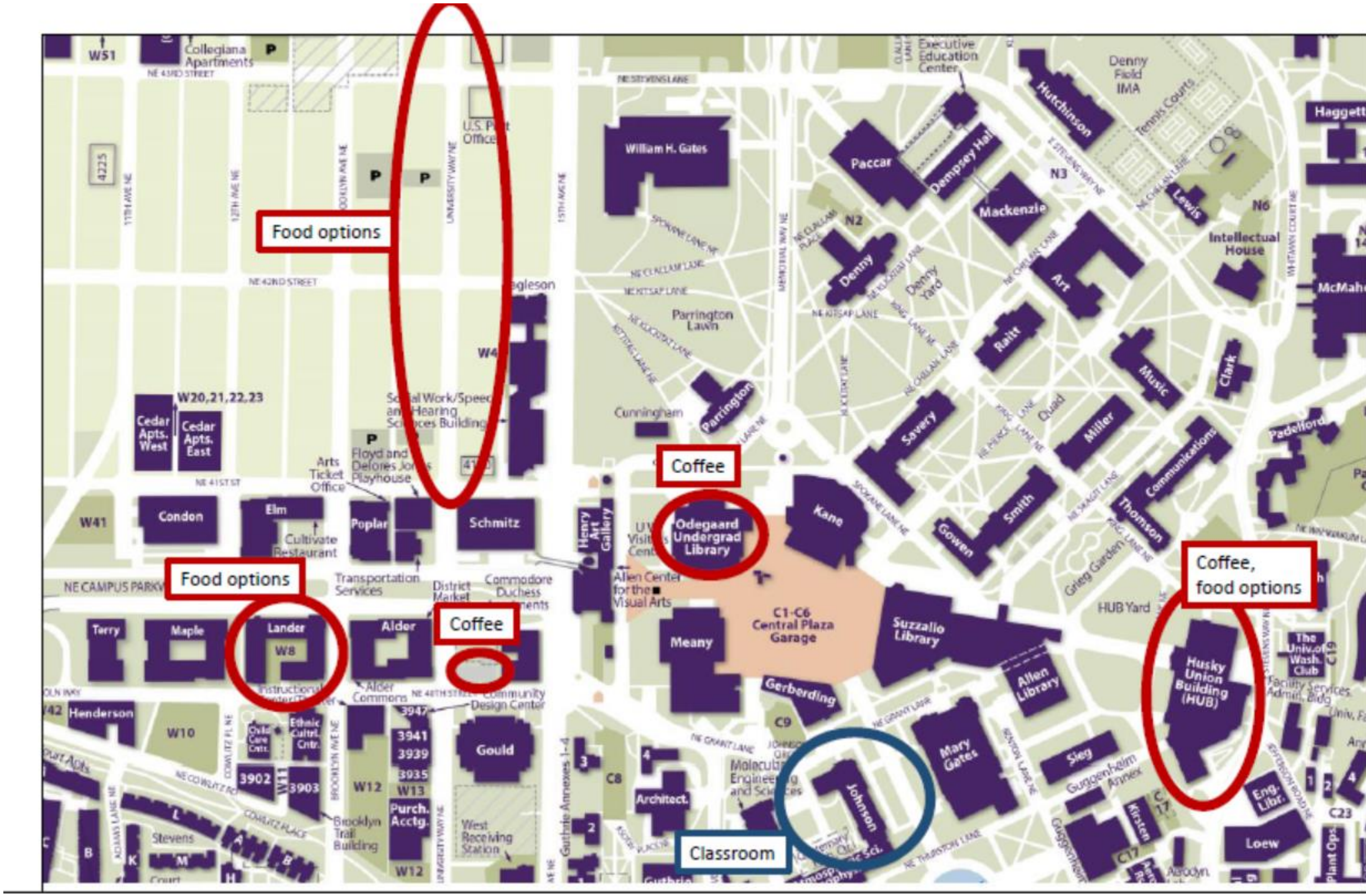
Food options
Food options
Coffee
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food options
Classroom
Odegaard Undergrad Library
Husky Union Building (HUB)
Johnson
Suzzallo Library
Lander
Schmitz

Objectives for the 1 week course

Gain intuition about epidemic dynamics

- Strengths and limitations of the different modeling frameworks

Understand the principles and methods of network analysis relevant to infectious disease epidemiology

- Descriptive network analysis
- Statistical network analysis with ERGMs and TERGMs
- Empirical study designs for networks

Develop the knowledge and software skills to run your own simple network transmission models.

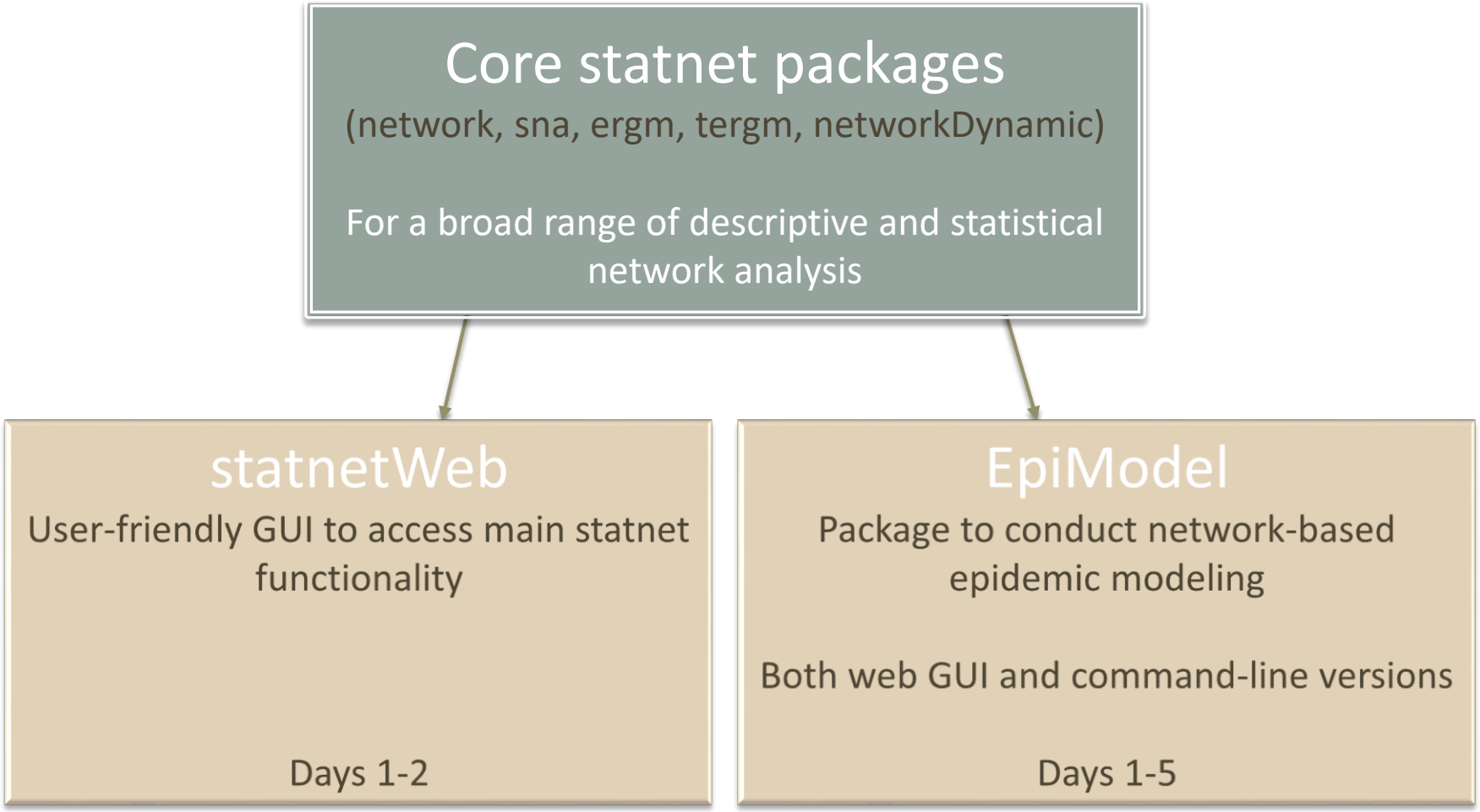
- Using R, statnet and the EpiModel package

Learn how to extend EpiModel code for your own research applications

The lesson plan for the week

Day	Content
1	Epidemic models – overview of the range of methods available • Deterministic vs. Stochastic; Compartmental vs. Individual vs. Network Network analysis basics
2	Cross-sectional statistical network analysis • Exponential Random Graph Models (ERGMs) for static networks Dynamic statistical network analysis • Separable Temporal ERGMs (STERGMs) for dynamic nets
3	Simple disease transmission on dynamic networks • When network dynamics are independent of disease dynamics
4	Disease transmission on dynamic networks with feedback • When network and disease dynamics interact
5	Extending EpiModel • Exploring your research questions

Software: based on R



Objectives for today

Get an intuitive sense of epidemic modeling, including:

- Elements of an infectious disease transmission system
- Signature dynamics of classic systems: the SIR/S family
- The range of modeling methods, and differences between them
  - Traditional deterministic compartmental models (DCMs)
  - Stochastic individual-based (or “agent-based”) models
  - Stochastic network models

Learn to explore simple individual-based SIR/S models using the **EpiModel** web interface

Develop a basic familiarity with network concepts and analysis, using **statnetWeb**

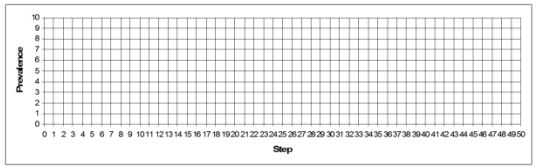
Starting with intuition:

Poker chip simulation

- **Blue chips** = susceptible
- **Red chips** = infected
- White chips = recovered

We will simulate and track the epidemic by hand

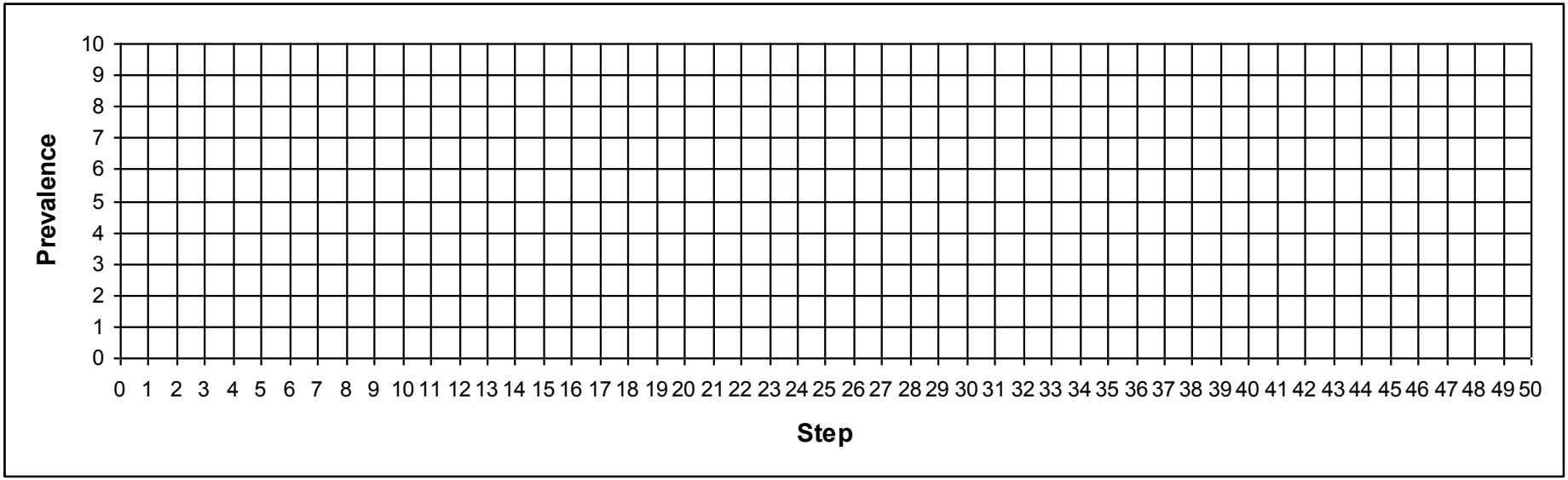
old school, analog style



Note:

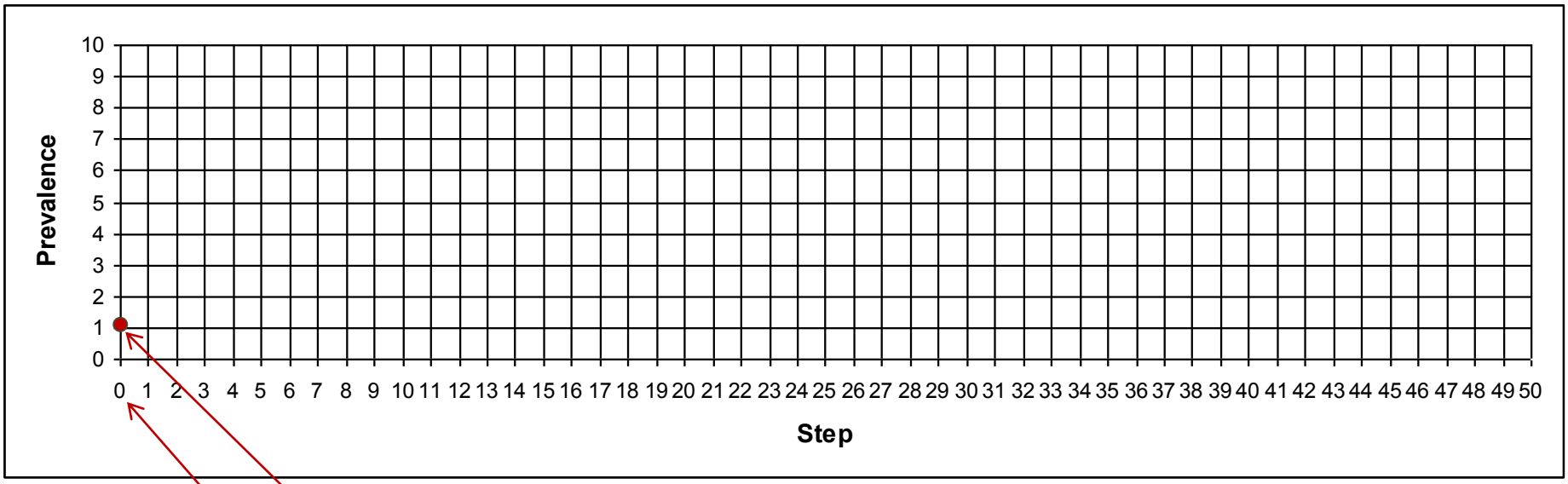
- We will simulate an individual-level process
  - Poker chips represent persons
    - Drawing poker chips from the bag represents the contact process
    - Replacing blue chips with red represents transmission
    - Replacing red chips with white represents recovery
- And record population-level outcomes
  - Prevalence: number of infecteds
  - Qualitative properties: extinction, persistence, equilibrium

Prevalence Worksheet



We will use this to track the prevalence over time

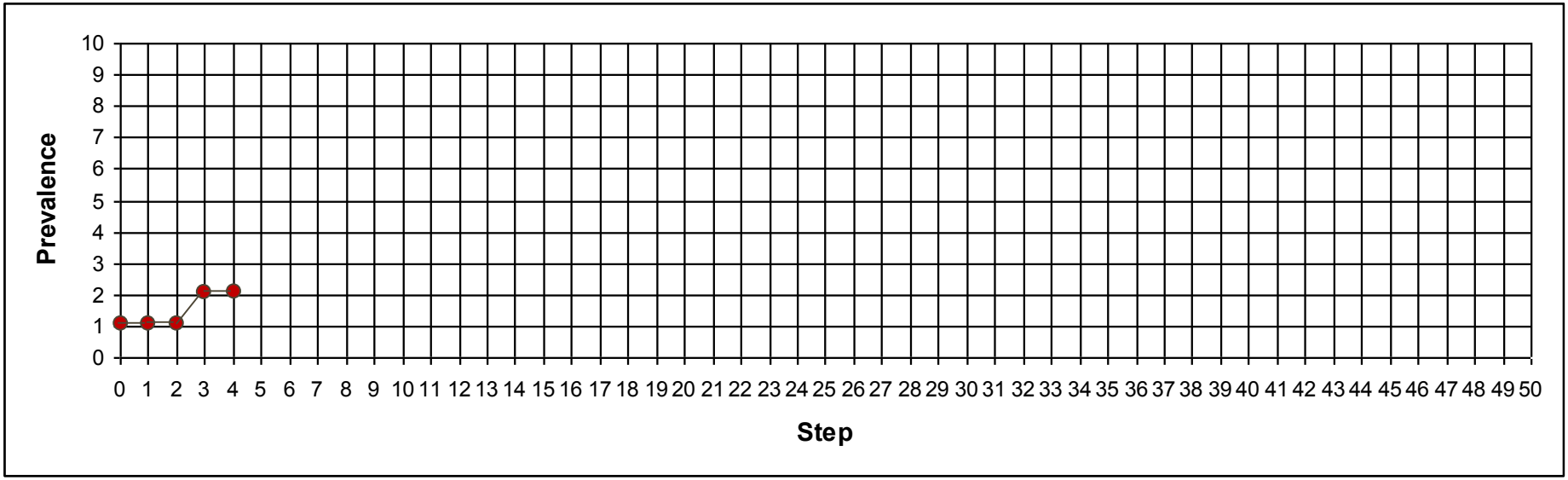
Prevalence Worksheet



We begin with one infected person

At time = 0, the start of the process

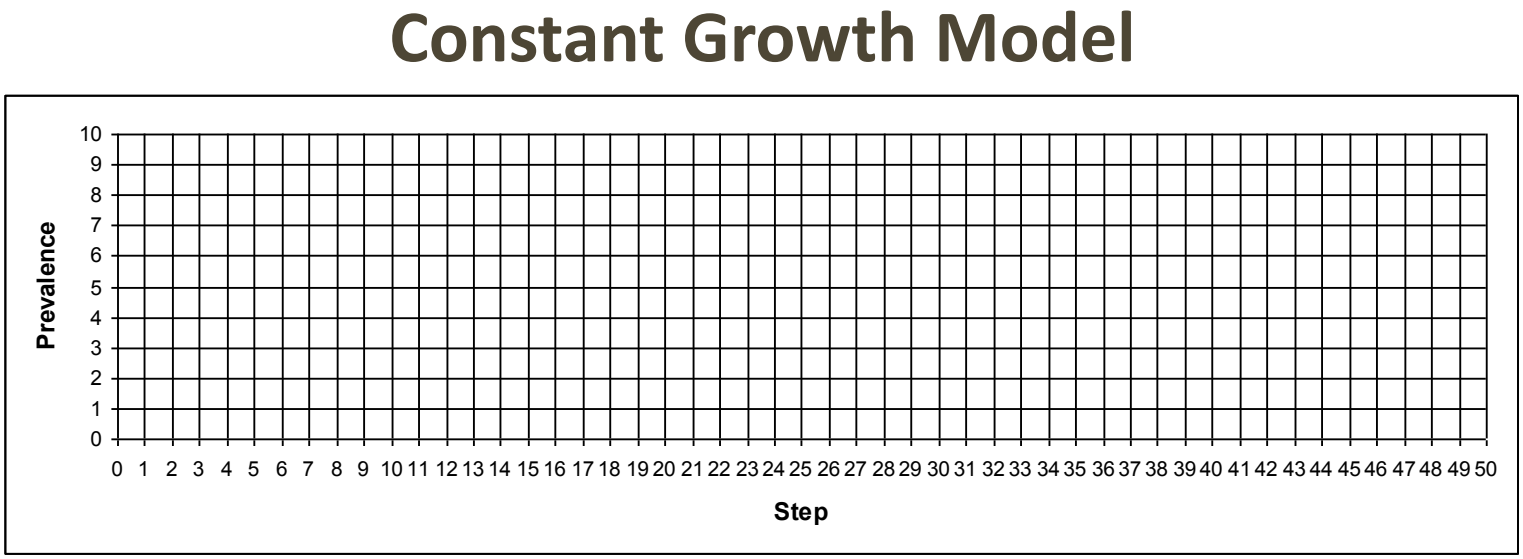
Prevalence Worksheet



At each subsequent time point we record the current number of infected persons (prevalence)

To the lab…

Record your outcomes on the



Constant growth model

INSTRUCTIONS: Start with 1 **red** chip (**red = I**)

For each round:

1. Add 1 more **red** chip
2. Update the current prevalence on the tracking worksheet
3. Repeat

Constant growth: Summary

Population size	Infinite
Final epidemic size	Infinite
Incidence curve	Flat: does not depend on prevalence
Prevalence curve	Linear: slope = incidence

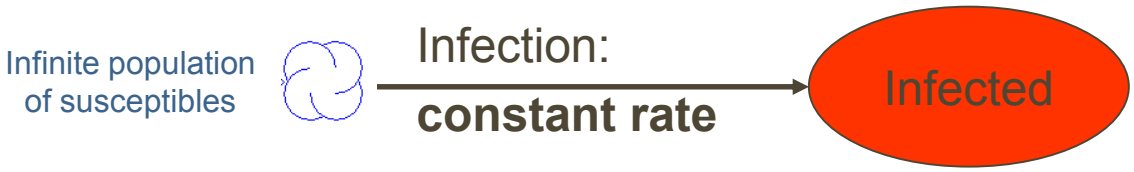
Was this process stochastic or deterministic?

Constant growth: Implications

Not like an infectious process, more like chronic disease

Each step is some unit of time (i.e. minute, hour, day, etc.)

- Only one state (infected)
- Only one transition (the infection process)



I: Infected model (proportional growth)

INSTRUCTIONS: Start with 1 **red** chip

For each round:

1. Add 1 more **red** chip *for each red chip already on the table*
2. Update the prevalence on the tracking worksheet to the current number of red chips on the table
3. Repeat

I Model: Summary

Population size	Infinite
Final epidemic size	Infinite
Incidence curve	Exponential: Depends on prevalence at t
Prevalence curve	Exponential (slope = incidence)

We'll get more specific this afternoon

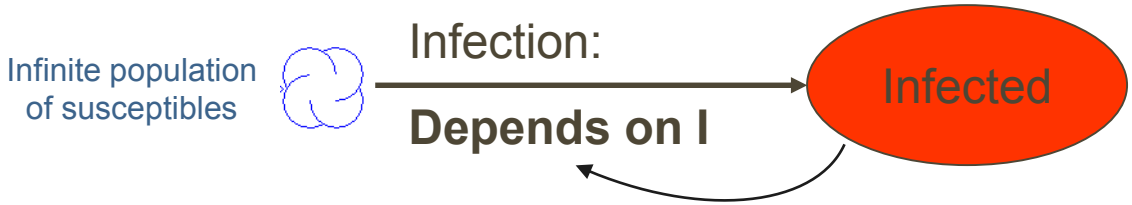
Was this process stochastic or deterministic?

I model: Components

The simplest true infection process

- Still only one state (infected)
- Still only one transition,

but now the incidence is determined by the prevalence



But … what if the population of susceptibles is *not* infinite?

SI: Susceptible-Infected model

INSTRUCTIONS: Now we will use the bag to represent the "population"

Prepare a bag with **1 red** chip and **9 blue** chips (10 total)

For each round: **S=blue**, **I=red**

1. **Pick two chips**
   - If the chips are the same color, no infection occurs.
     - Return both chips to bag, go to step (2)
   - If the chips are different colors, infection occurs
     - Replace **blue** chip with **red** chip and return to bag
2. **Update the prevalence on tracking sheet to # of red chips now in the bag**
3. **Are there any more blue chips in the bag?**
     - YES: Return to (1)
     - NO: Stop

SI model

Every draw has three possible outcomes:

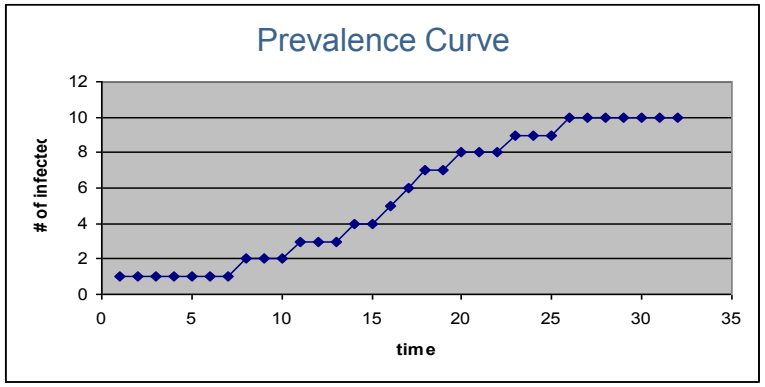
SS: concordant negative
SI: discordant
II: concordant positive

Only SI can create incidence

1.	SS	9.	SS	17.	SI	25.	II
2.	SS	10.	SS	18.	SI	26.	SI
3.	SS	11.	SI	19.	II	27.	II
4.	SS	12.	SS	20.	SI	28.	II
5.	SS	13.	SS	21.	SS	29.	II
6.	SS	14.	SI	22.	II	30.	II
7.	SS	15.	II	23.	SI	31.	II
8.	SI	16.	SI	24.	II	32.	II

The probability of each outcome changes as the process evolves.

- P(SI) low at the start
- reaches its max when S=I,
- decreases again as S get depleted.



SI Model: Summary

Population size	FINITE: N = S + I
Final epidemic size	N (everyone)
Incidence curve	Bell shaped: Depends on S and I
Prevalence curve	Logistic (slope = incidence)

Was this process stochastic or deterministic?

SI Model : New concepts

1: Stochasticity (of some model aspects, but not others)

The contact process is stochastic

The infection process is deterministic (why?)

2: Equilibrium outcome

Final prevalence at equilibrium: is *deterministic,* always = N

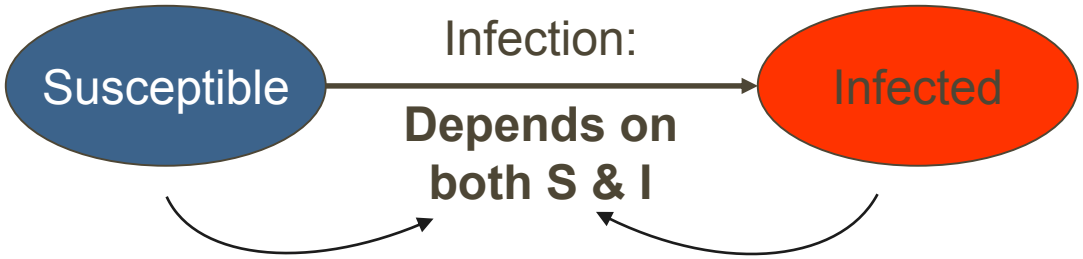
Time to equilibrium: is *stochastic*, because of the contact process

Range is open ended: [N-1, ∞]

If the infection rate/probability $\tau < 1$, it would depend on that too

SI model: Components

Two states, but still only one transition: infection



So … what does this model assume about the duration of infection?

Transition 2: Recovery

Consider other possible *states* and *transitions* in the system

- **Recovery with return to susceptibility** (e.g. common cold)

  This adds a new transition to the system: ***SIS***

- **Recovery with immunity** (e.g. measles)

  This adds a new transition and a new state to the system: ***SIR***

Both models have a new transition

- What does the transition from **I** → ℝ or **I** → **S** represent?
  - Not an infectious process
  - More like the constant rate we had before
  - Defined by the "duration of infection" D

- Let's see how this changes the process, with a final poker chip example of an SIR process
  - Now, we will need a new time tracker for **I** chips

DURATION TIMER

Days infected Case	0	1	2	3	4	5	6	7	8	9	10 Change state to R
1	X	X	X	X	X	X	X	X	X	X	X
2	X	X	X	X	X	X	X	X			
3											
4											
5											
6											
7											
8											
9											
10											

Take a **red** chip (any one is fine) and replace it with a **white** chip

SIR: Recovery with Immunity Example

INSTRUCTIONS:
Prepare a bag with **1 red** and **9 blue** chips; put **10 white chips** on the side.

S=blue, I=red, R=white

1. **Pick two chips**
   - If the chips are not red and blue, no infection occurs.
     - Replace both chips in bag, go to step (2)
   - If the chips are red and blue, infection occurs
     - Replace blue chip with red chip and return to bag
2. **Update duration worksheet for BOTH new and pre-existing infections**
   - Increment each active row by 1 day
   - If any durations are at {CHANGE STATE}, take a red chip from the bag and replace it with a white chip
3. **Update prevalence worksheet with the # red chips now in the bag**
4. **Are there any more red chips in the bag?**
   - YES: Return to (1)
   - NO: Stop

SIℝ Model: Summary

Population size	FINITE: N = S + I + ℝ
Final epidemic size	0 : These infections always die out
Incidence curve	Bell shaped: Depends on S, I and ℝ
Prevalence curve	Bell shaped: (slope = f(incidence and ℝ))

Was this process stochastic or deterministic?

SIR: New concepts

1: Extinction

Time to extinction of ***I*** : *stochastic (why?)*

Range is { D to D*N }

If the infection rate/probability $\tau < 1$, it would depend on that too

2: Final prevalence of ***S*** and ***R***

Stochastic, but has a range(S,R) of { (0,N) to (N-1,1) }

Depends on cumulative number of infections before extinction.

SIS: Recovery with Susceptibility (on your own)

INSTRUCTIONS: Prepare a bag with 9 blue and 1 red chips (S=blue, I=red)

1. **Pick two chips**
   - If the chips are not red and blue, no infection occurs.
     - Replace both chips in bag, go to step (2)
   - If the chips are red and blue, infection occurs
     - Replace blue chip with red chip and return to bag

2. **Update duration worksheet for any new and pre-existing infections**
   - Increment each active row by 1 day
   - If any durations are at {CHANGE STATE}, take a red chip from the bag and *replace it with a blue chip*

3. **Update prevalence worksheet with the number of red chips currently in the bag**

4. **Are there any more red chips in the bag?**
   - YES: Return to (1)
   - NO: Stop

SIS: New concepts

1: Equilibrium prevalence in a closed (finite) population

In a deterministic model, equilibrium prevalence can range from [0, N]

If there is stochasticity, infection will always eventually die out.

Final prevalence of ***I*** is always 0, and ***S*** is always N

But it can take … forever to get there

And the probability of extinction at any time may be vanishingly small

2: Both S & I prevalence may be cyclical.

Model parameters

- These have been implicit in most of the discussion above, but we will foreground them in the next session

- Examples:
  - Contact is governed by a rate of acts per capita per time step (α)
  - Infection is governed by the probability of transmission per act (τ)
  - Recovery is governed by a rate or probability of recovery per time step (ρ)

- And these are sometimes combined to simplify equations
  - e.g.: $\beta = \tau\alpha$ to represent the overall infection rate

SUMMARY of MODELS

Transmission system	Rate of change in I = $\frac{\Delta I}{\Delta t}$	Prevalence signature	Assumptions
Non-infectious process for I	k	Constant linear growth	Infinite population of S, infinite D, no contact process
I	β**I**	Exponential growth	Infinite population of S, infinite D
SI	β**SI**	Logistic growth	Finite population, infinite D
SIR	(β**S** – ρ)**I**	Bell shaped	Finite population, finite D
SIS	(β**S** – ρ)**I**	Potentially cyclic	Finite population, finite D

* For models with infinite population, $\beta = \tau\alpha$ ("density dependent" transmission); for models with finite population $\beta = \tau\alpha/N$ ("frequency dependent" transmission)

Poker chips to epidemic modeling terminology

Poker chip component	Model component	Model Terminology
Poker chips	Elements	Individuals
Color	States	Individual disease status
Bag	Population	Population size (N, or infinite)
Draw out of bag	Transition (infection)	Act (governed by α)
Draw blue and red *		Discordant partnership (SI)
Blue exchanged for red		Transmission (governed by τ)
* Blind draws out of bag	Model assumption	Random mixing
Red exchanged for white	Transition (recovery)	Recovery with immunity (governed by recovery rate ρ via disease duration D)

Introductions

- Who are we?
- Who are you?

Course Website

http://statnet.github.io/nme/