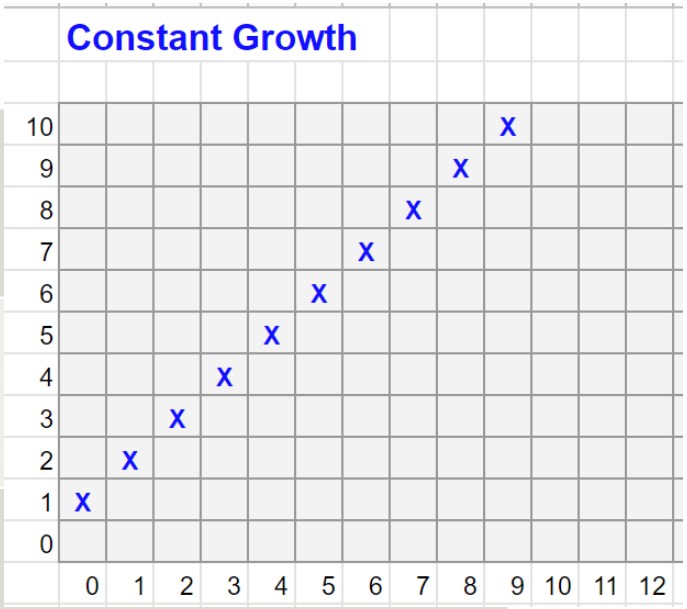
1

# From poker chips

to epidemic models

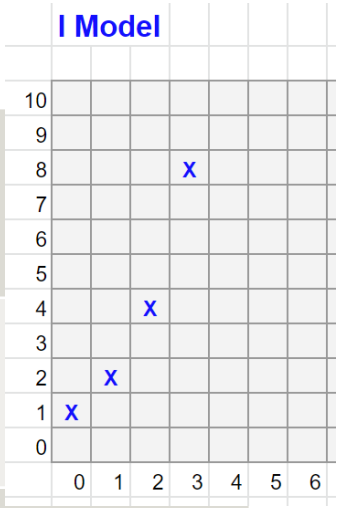
# Constant growth: Summary

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




***Was this process stochastic or deterministic?***

# I Model: Summary




| | |
|---|---|
| ***Population size*** | Infinite |
| ***Final epidemic size*** | Infinite |
| ***Prevalence curve*** | Exponential: *Now depends on prevalence at t* |
| ***Incidence curve*** | Exponential *(slope of prevalence curve, as before)* |

***Was this process stochastic or deterministic?***

# SI model

Each draw has 3 possible outcomes:

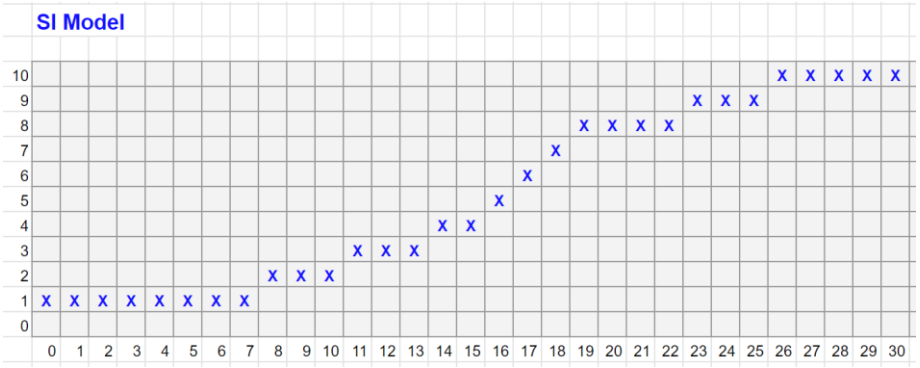
**SS:** concordant negative
**SI:** discordant
**II:** concordant positive

**Only SI creates incident infection**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 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 **SI** outcomes changes over time

- Low: at the start
- High: reaches its max when S=I,
- Low: decreases as S get depleted
- Bell shaped



# SI Model: Summary

| | |
|---|---|
| ***Population size*** | FINITE: N = S + I |
| ***Final epidemic size*** | N (everyone) |
| ***Prevalence curve*** | Logistic *(slope = incidence)* |
| ***Incidence curve*** | For poker chips: p(incident case) is bell shaped<br>In general, the incidence curve is bell shaped<br>Depends on S(t) and I(t) |

***Was this process stochastic or deterministic?***

# SI Model: New concepts

1. Susceptible depletion

   As the pool of susceptibles shrinks

   - The probability of drawing an II pair rises
   - 'wasted' contacts (from the pathogen's perspective)

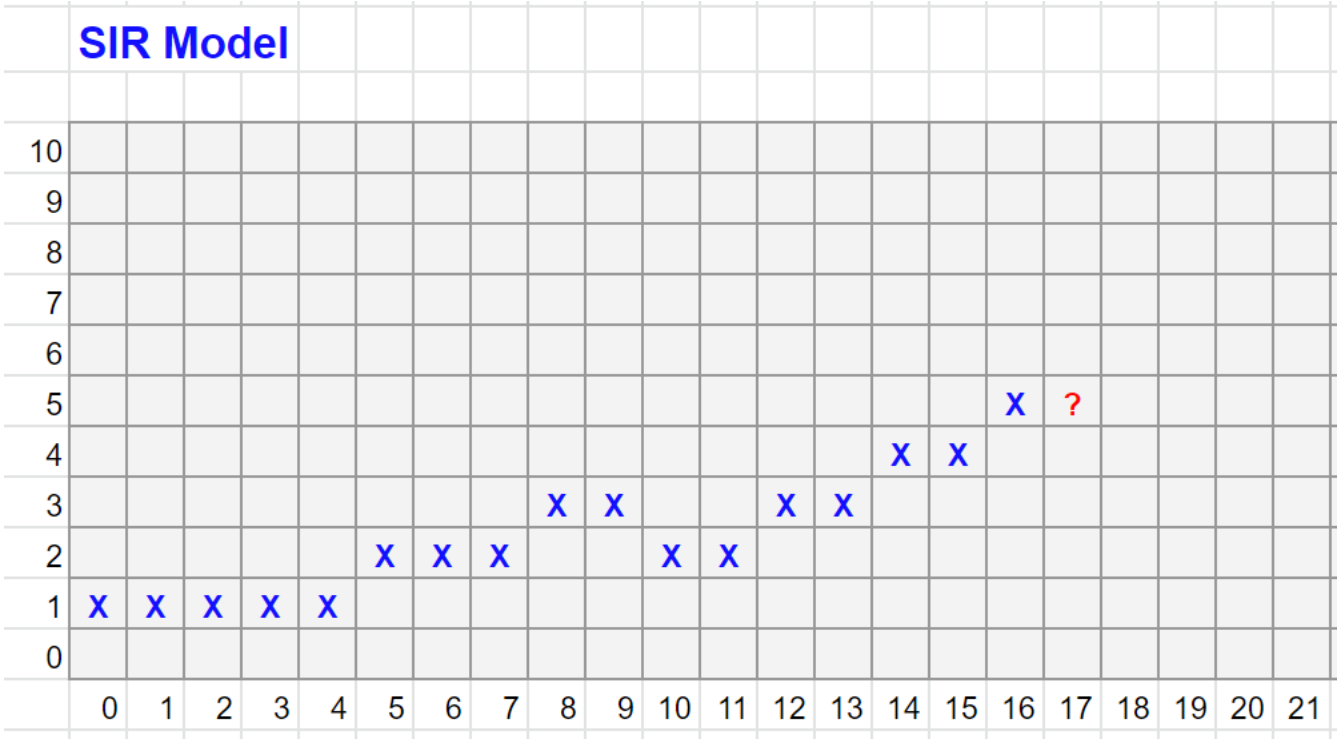
2. Equilibrium state

   Eventually the epidemic ends

   - Not because the infection dies out
   - But because there's no one left to infect

# The SIℝ model

- This is where we stopped, about half way thru



# Quiz results

- Let’s see how you did

# SIℝ Model: Summary

| | |
|---|---|
| ***Population size*** | FINITE: N = S + I + ℝ |
| ***Final epidemic size*** | 0 : These infections always die out in a closed popn |
| ***Prevalence curve*** | Flat (if it dies out after the first case)<br>Non-monotonic otherwise<br>(roughly bell shaped in large populations)<br>*Slope = f(incidence and recovery)* |
| ***Incidence curve*** | For poker chips: p(incident case) is bell shaped<br>In general, the incidence curve is bell shaped<br>Depends on S(t), I(t) and R(t) |

***Was this process stochastic or deterministic?***

# SIℝ : New concepts

**1:** Extinction

Time to extinction of ***I*** : *stochastic in the poker chips (why?)*

Range is { D to D*N } (for our poker chips, 10 to 100)

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

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

Variable, but has a range for S of {0, N-1} and for R of {1, N-1}

Depends on cumulative number of infections before extinction.

# Note

- The prevalence of I may be non-monotonic
- But the prevalence of S is monotonic decreasing
  - Until it hits equilibrium
- And the prevalence of ℝ is monotonic increasing
  - Again, until it hits equilibrium

# On stochasticity …

- Did we track the time of individual infected chips?
  - Yes, … and no

    We replaced any one of the red chips in the bag with a white chip
  - What assumption does this represent?

    Homogeneity/exchangeability of individual cases, and “memoryless” survival function (exponential distribution of survival times)
- Was there any stochasticity in the recovery dynamics at the population level?
  - No. Exactly 10 days after each infection, you get a recovery

# Recovered/Immune cases

- Interfere with ongoing transmission
  - Lots of new contact pair possibilities that don't transmit
- As recovered/immune cases rise, incidence declines
  - This is related to the concept of "herd immunity"
    - Even if you're not immune, the immune cases around you give you protection.
  - And if we allow the probability of transmission to be < 1
    - Even if you contact an infected person, you may not get infected

# Zooming all the way back out

- What's an example of an infection that has recovery with immunity?
- Has that infection died out (the equilibrium state)?
- So, what's missing in our model?

# What about SIS

# 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 Model: Summary

| | |
|---|---|
| ***Population size*** | FINITE: N = S + I |
| ***Final epidemic size*** | Depends on the model you're using (!!!!) |
| ***Prevalence curve*** | Flat (if it dies out after the first case)<br>Non-monotonic otherwise<br>(for a long time, and not bell-shaped)<br>Slope = f(incidence and recovery) |
| ***Incidence curve*** | Like prevalence, flat 0 or non-monotonic |

# SIS : New concepts

1: **Susceptible replenishment**

S prevalence may also be non-monotonic now, which changes everything

**2:** Equilibrium prevalence in a closed (finite) population will depend on the type of model you're using

*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

# Formalizing these concepts

# Models have three basic components

- **Elements** – "actors" in the model
- **States** – attributes of system elements
- **Transitions** – rates of movement between states

All models have these same building blocks

# Model component 1: **Elements**

Elements can be

- Persons
- Animals
- Environmental reservoirs (water, soil)
- Etc.

*Example*: COVID transmission requires people

Is the pathogen a separate element?

- In some models, yes
- This week: no

# Model component 2: **States**

States – attributes of elements. For example:

- Infection-related (Susceptible, Infected, Recovered….)
- Demographic (sex, age….)
- Behavioral (level of sociality; occupation….)
- Clinical (tested or not; on treatment or not…)
- Geospatial (community; coordinates….)
- Etc. etc. etc.

*Example*: A very simple COVID model has three host states:

susceptible (S)

infected (I)

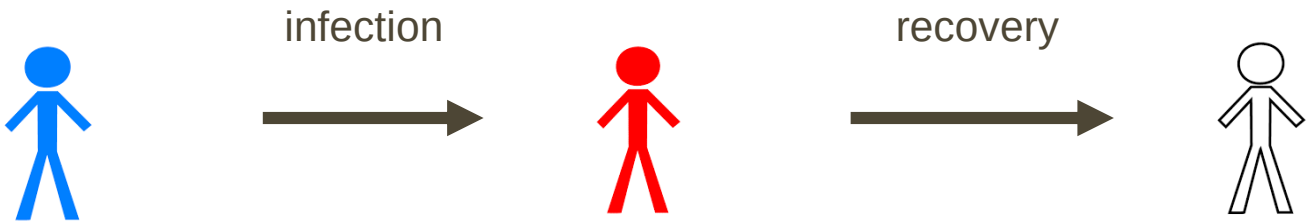
recovered w/ immunity (R)

# Model component 3: **Transitions**

## Transitions – movement between states

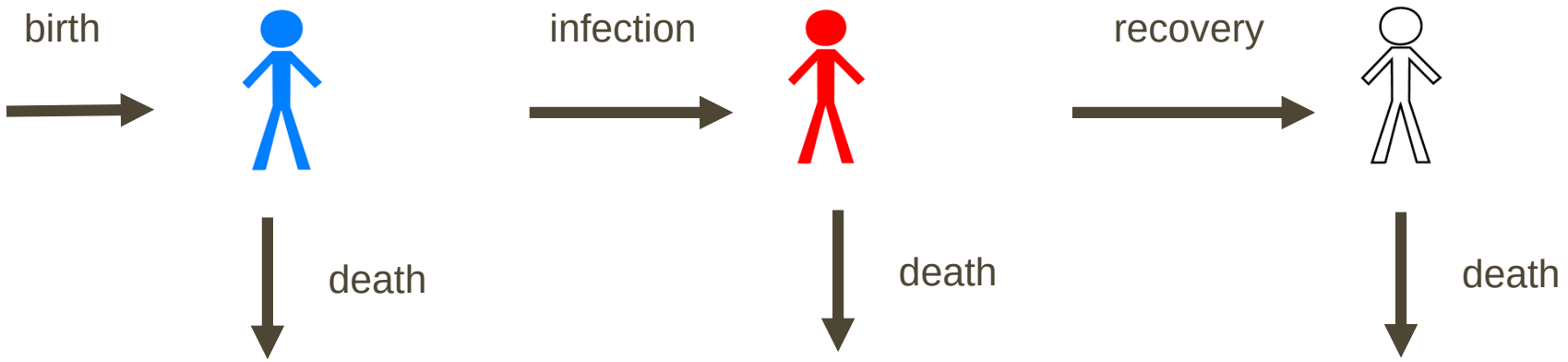
- **Deterministic**: fixed *rate* of transition between states.
  - Uses a population mean rate to govern flows
- **Stochastic**: *probability* that an element transitions between states.
  - Uses a draw from a probability distribution to govern flows

*Example*: A very simple COVID model has two transitions:



# Model component 3: **Transitions**

- For “open populations”
  - Can also represent entry, from outside the model
  - And exit, to outside the model



# Key transition: Infection

Many different modes of transmission:

| | |
|---|---|
| STD/HIV: | direct body fluid contact (sex, needles, MTC) |
| COVID, Measles, Influenza: | respiratory, air-borne |
| Diarrheal diseases: | fecal-oral |
| Malaria: | vector-borne (mosquitoes) |
| Schistosomiasis: | water and vector-borne |
| Cholera: | water and food-borne |

# Key transition:  Infection

- Focus on person-to-person transmission this week
  - It's a natural way to introduce network modeling
  - The personal contact network becomes the foundation of the transmission network
- But the concepts and methods here are general
  - You can represent contact networks of animals
  - Or multi-layer networks of persons and vectors
  - Or bipartite networks of persons and places
  - …

# Finally: Different modeling frameworks

- While all epidemic models share certain components
  - Elements, states, transitions
- There are different ways to represent the system as a mathematical model
  - Deterministic Compartmental Models (DCMs)
  - Stochastic Individual-based Contact Models (ICMs)
  - Stochastic Network Models
- The framework you choose has a big impact…

# 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, finite) |
| Draw out of bag | Transition (infection, stochastic) | Act |
| Draw blue and red * | | Discordant pair (SI) |
| Blue exchanged for red | | Transmission |
| * Blind draws out of bag | Model assumption | Random mixing |
| Red exchanged for white | Transition (recovery, deterministic) | Recovery with immunity |