

Disease Dynamics in a Dynamic Social Network

Claire Christensen¹, István Albert³,
Bryan Grenfell², and Réka Albert^{1,2}

¹Department of Physics and

²Center for Infectious Disease Dynamics,

³The Huck Institutes for the Life Sciences

The Pennsylvania State University, University Park, PA 16802, USA

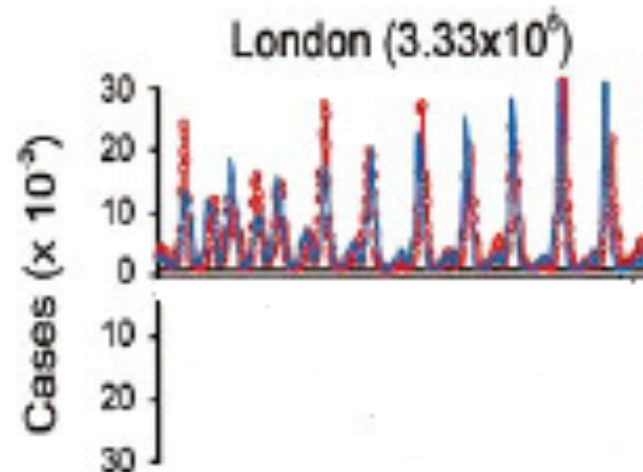
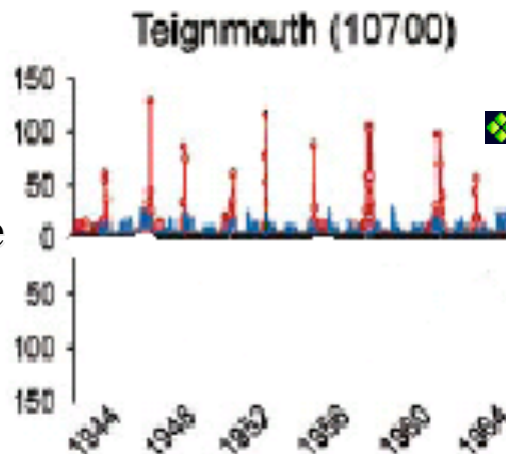
Social Structure and Disease Dynamics

What is it about the demographics and connectivity of an underlying *contact network* that creates the most prominent features of the landscape in which a disease travels?

Below threshold:
•chaotic epidemics
•intervening fadeouts

Above threshold:
•regular epidemics
•few fadeouts

The Classic
Example:
Population Size



Grenfell, B.T., et. al, *Ecological Monographs* 72 (2002): 185-202

How do *changes* to that landscape affect the dynamic behavior of the disease?

Classical Mathematical Modeling of Disease Dynamics

Basic model assumes *full-mixing* of susceptibles (S), infecteds (I), recovereds (R), such that rate of change of each group can be described by system of coupled differential equations:

$$\frac{dS}{dt} = -\beta S \frac{I}{N}$$

Contact-
transmission
parameter

$$\frac{dI}{dt} = \beta S \frac{I}{N} - \gamma I$$

Recovery rate

$$\frac{dR}{dt} = \gamma I$$

Model works well in large populations.
Variants of model account for some features
of small populations, but not all.

***In-silico* Simulation of Contact Networks and Disease Propagation: an Overview**

Why:

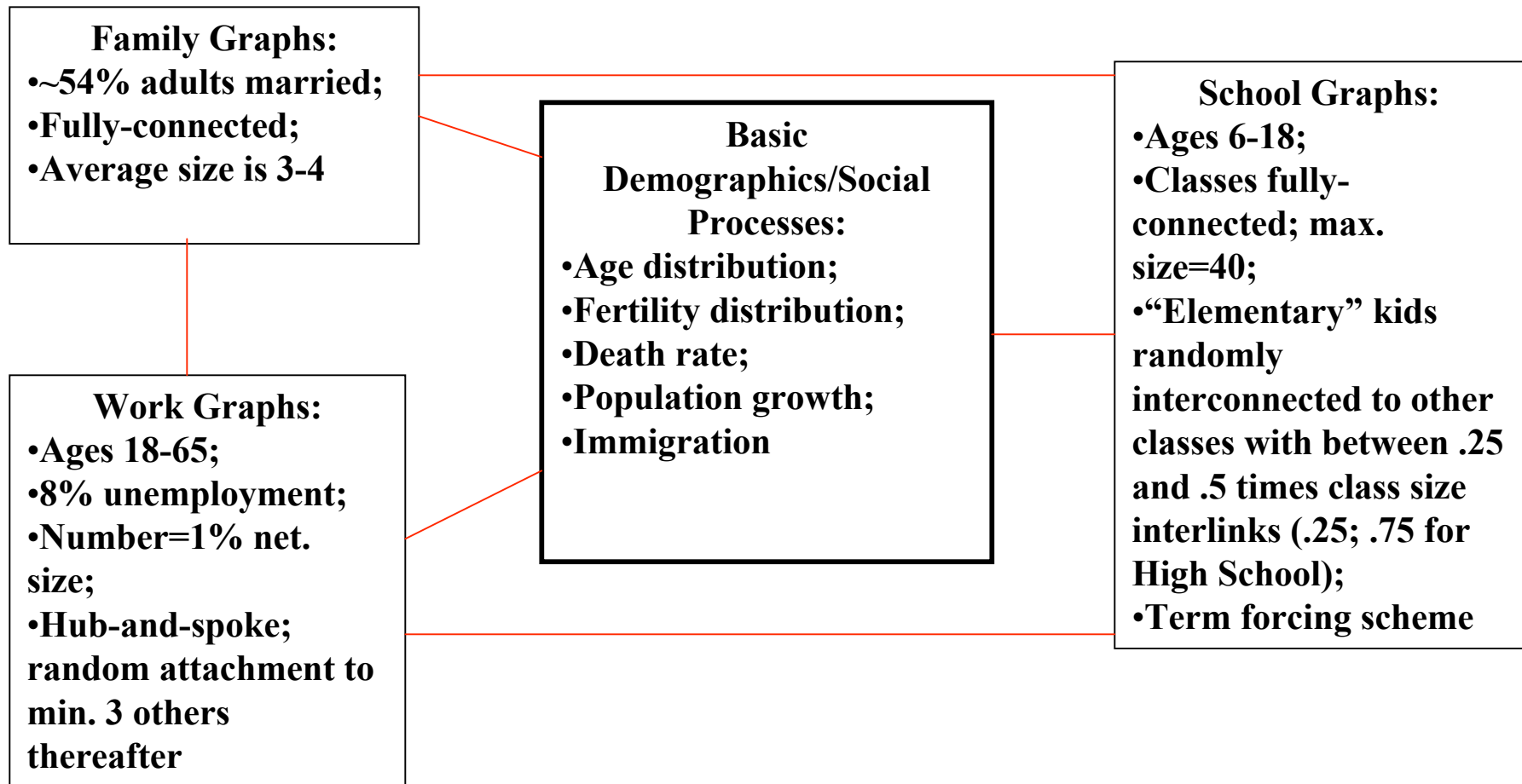
- Probe link between (changing) topology of contact network and disease dynamics;
- Incorporate statistical (social) data;
- Small size not a problem

How (first steps):

- Simulate social network (families+workplaces+schools+individuals);
- Statistical growth and change;
- Disease propagates (WAIFW) along edges of network

Dominant Social Processes/Features

(For more information, please visit the poster exhibit)



**All statistics adapted from:

1) *Vital Statistic of New York State (1990-2002)*, in ‘Information for a Healthy New York’, Health, N.Y.S.D. o,Ed.

2) ‘U.S. Department of Labor Bureau of Labor Statistics’

3) Scardamalia, R., *The Face of New York– the Numbers* (2001)

Dominant Disease Processes/Features

(For more information, please visit the poster exhibit)

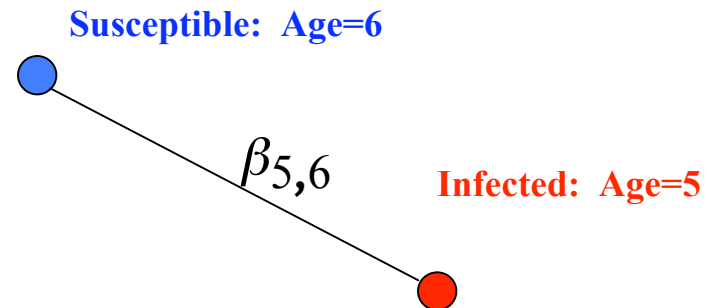
Immunity and Infection

- Initial immunity-by-age profile;
- Newborns lose maternally-acquired immunity;
- Disease spreads from infected to susceptible via edges, and with rates from WAIFW matrix

Maintaining Epidemics

- Sometimes necessary to “spark” population ($S \rightarrow I$ randomly);
- Sparking function of form:
 $P_{spark}(t) \sim \ln(N(t))$, where $N(t)$ is total population size

** All statistics adapted from:
Edmunds, W.J. *et al.*, *Epidemiology and Infection* 125 (2000): 635-650;
Scardamalia, R., *'The Face of New York-- The Numbers'* (2001);
Grenfell, B.T. and Anderson, R.M., *Proceedings of the Royal Society of London. Series B, Biological Sciences* 236 (1989): 213-252.



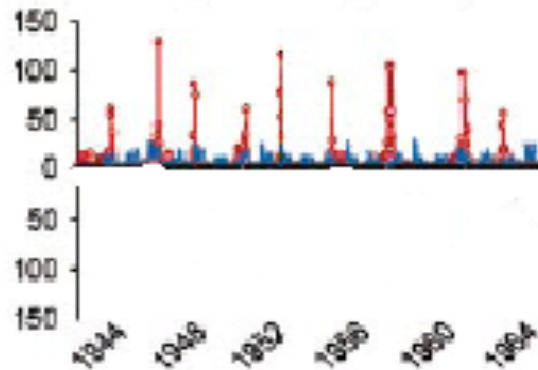
Prevaccine Measles

Comparison of infection profiles, interepidemic periods, and epidemic lengths

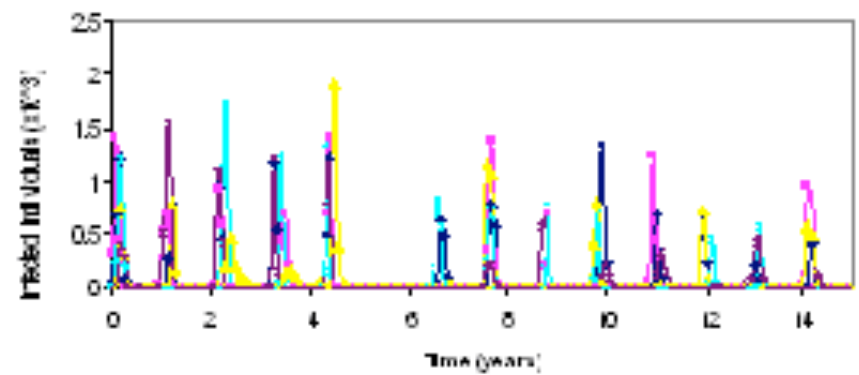
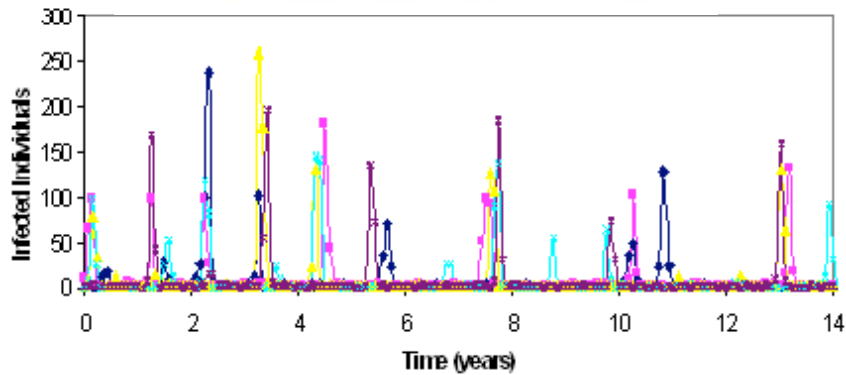
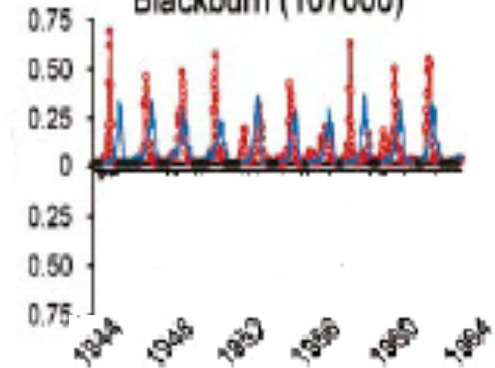
Data Set	$\langle I(t) \rangle$	$\langle T_{\text{inter}} \rangle$	$\langle \tau \rangle$
Teignmouth	~ 81	1-4 years	38 weeks
10,700 Network	75 ± 5	2.13 ± 1.15 years	40 ± 2 weeks

Data Set	$\langle I(t) \rangle$	$\langle T_{\text{inter}} \rangle$	$\langle \tau \rangle$
Blackburn	~ 500	1-2 years	41 weeks
107,000 Network	450 ± 10	$1.47 \pm .6$ years	40 ± 2 weeks

Teignmouth (10700)

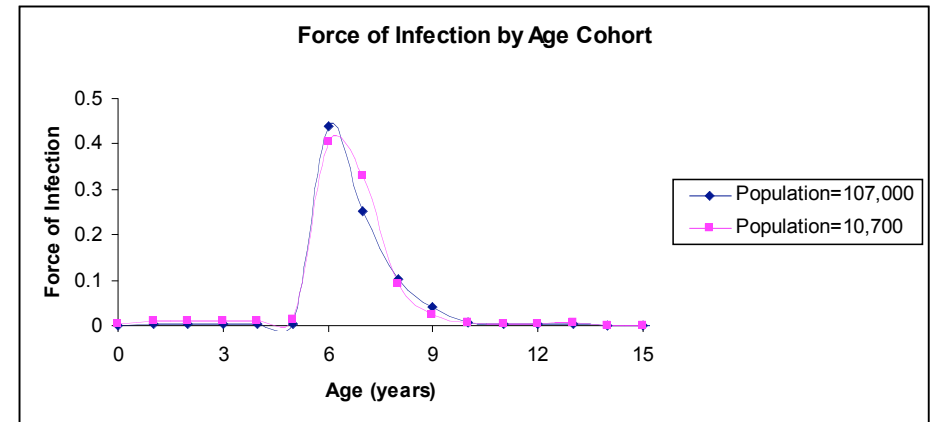
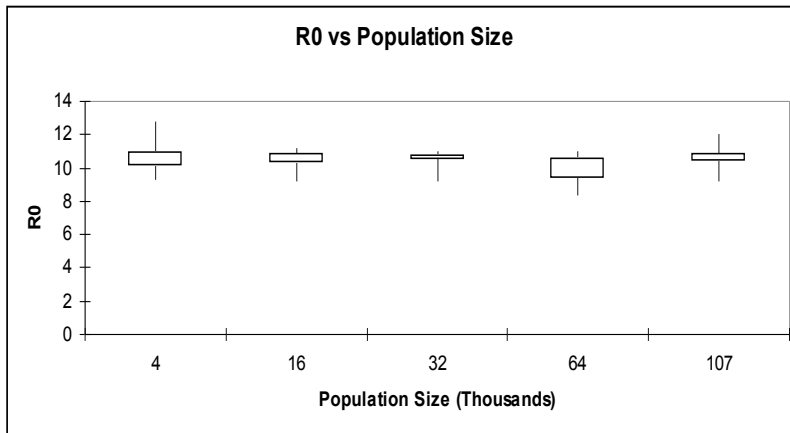


Blackburn (107000)



Grenfell, B.T., et. al, *Ecological Monographs* 72 (2002): 185-202

Basic Reproduction Number (R_0), Force of Infection, and the Influence of Contact Network Topology and Dynamics



Past explanation:

***Topology* of schools– contact network of most influential age cohort– is essentially the same, regardless of population size.**

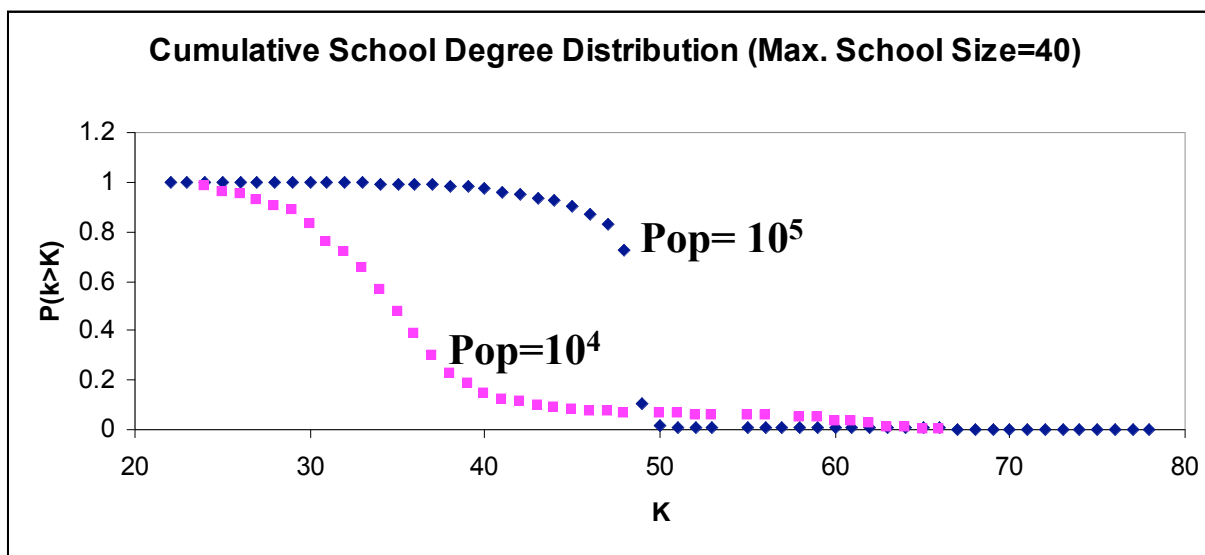
However...

Force of Infection

$$\lambda_i = \sum_{j=1}^n \beta_{i,j} \frac{I_j}{N}$$

School Network Topology is *NOT* Independent of Population Size

Why? *Class interlinking and abundance of full classes differ with population size.*



Different dynamic processes; same global characteristics!

Summary

- ***In-silico* simulation of contact networks and disease propagation has benefits of:**
 - A) Greater realism;***
 - B) Overcoming size/connectivity barriers;***
 - C) Window into interrelationship between (changing) network topology and disease dynamics at multiple scales of complexity and time***
- **Using measles as a test bed, we have:**
 - 1) Captured dominant features of observed measles dynamics in large *and* small populations;***
 - 2) Provided some insight into epidemiological trends by clearly synthesizing topological and disease dynamic features.***
- **We will extend the algorithm to other, less well-studied diseases and/or well-studied diseases in novel social environments.**

Thank You

Thank you to:

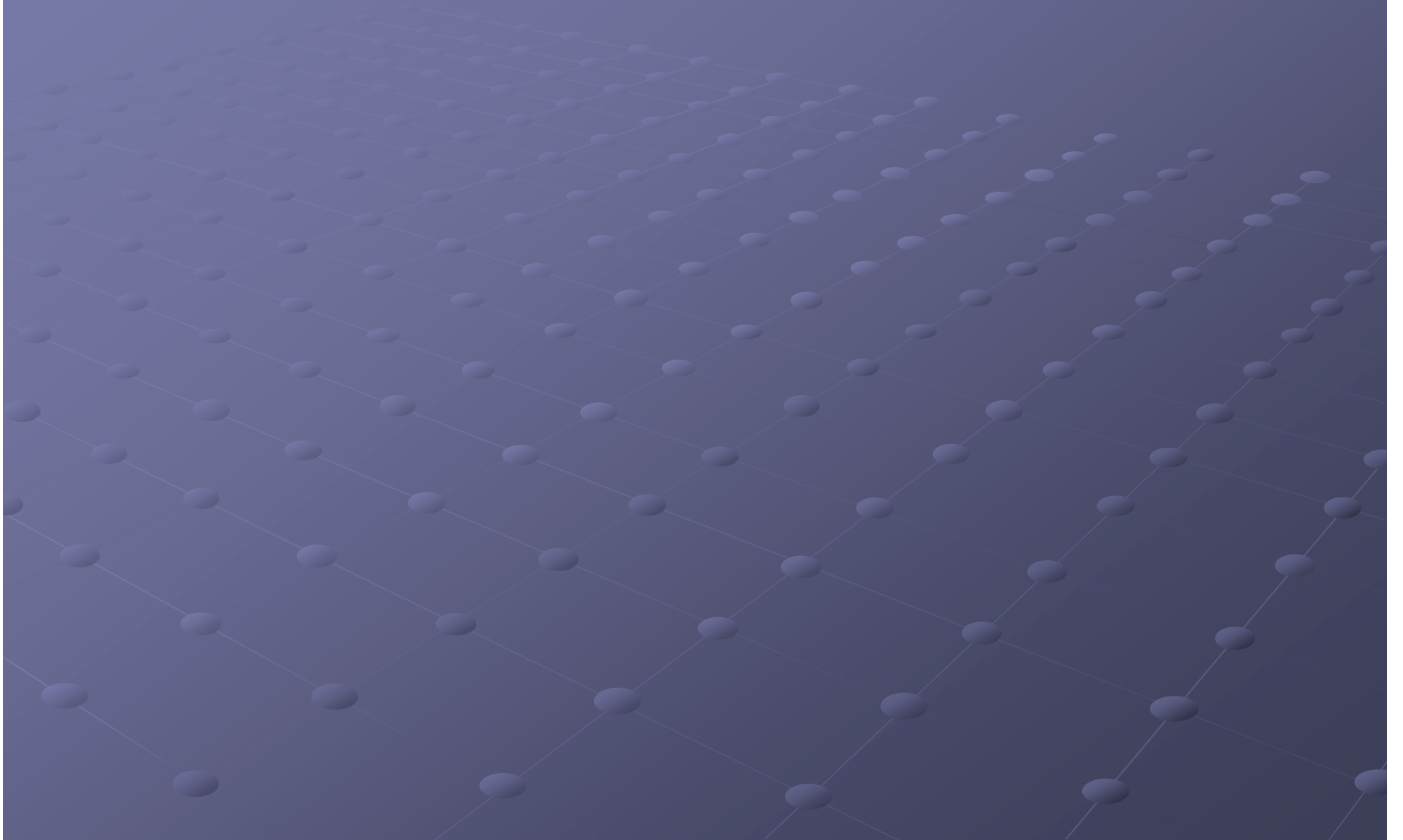
Dr. Réka Albert
Dr. Bryan Grenfell
Dr. István Albert

Dr. Anshuman Gupta
Dr. Jeff Nucciarone

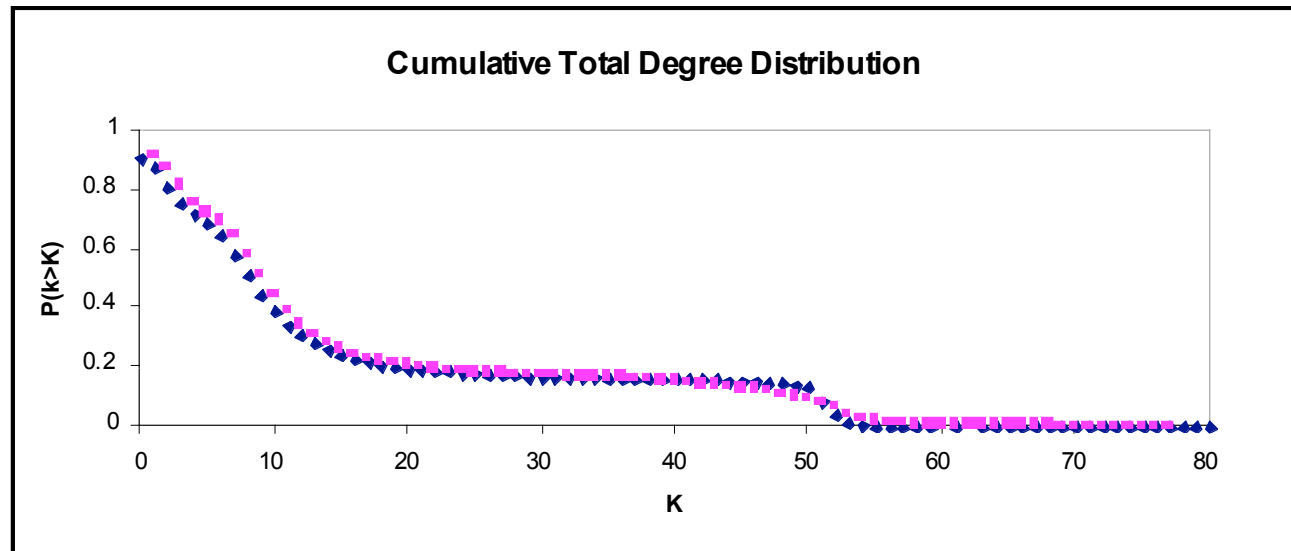
NIH MIDAS Cluster (Dr. Ganapathi Laxminarayana)

The organizers and sponsors of NetSci 2007

Extras

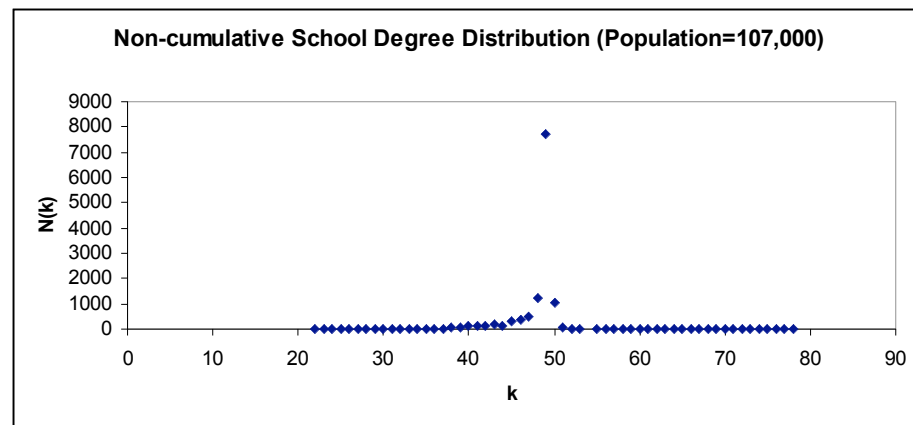
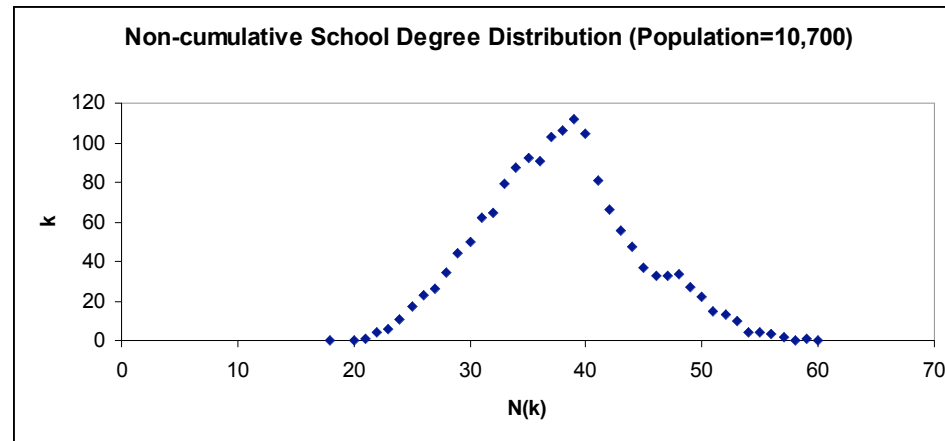


Total Degree Distributions



Cumulative degree distribution for populations of size 10^5 (blue) and 10^4 (pink).

School Degree Distributions (non-cumulative)



Seasonal Network Dynamics and Disease Dynamics

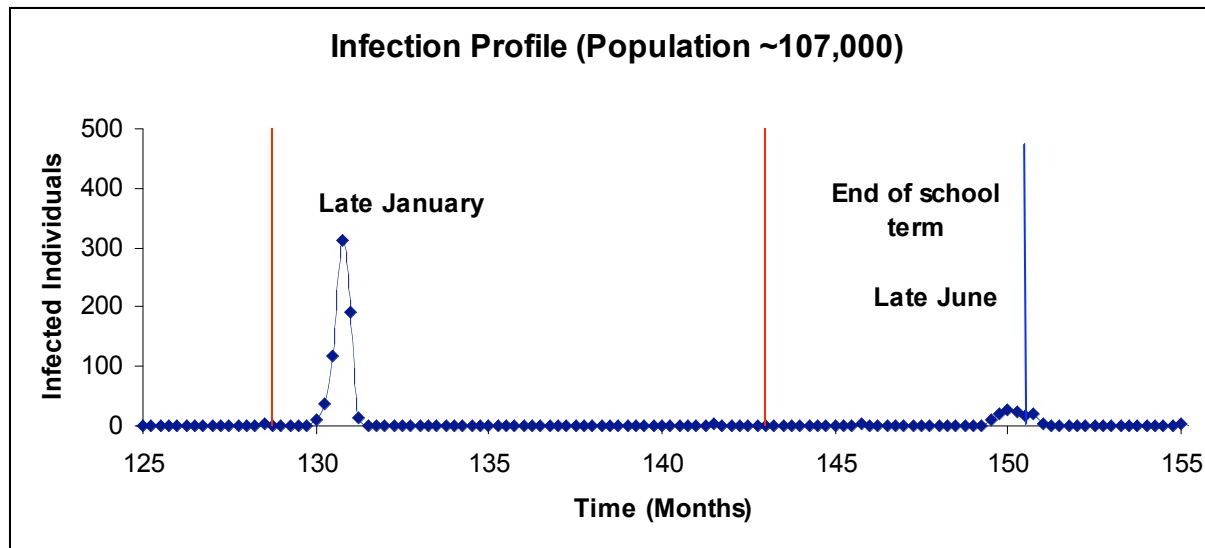
Recent work suggests:

Early-peaking epidemic at $year_t$

No epidemic at $year_{t+n}$

Seasonally-curtailed epidemic at $year_{t+n}$

Stone, L. *et al.*, *Nature* **446** (2007): 532-536



Evidence of this in our networks, as well.