# **Analysis and Control of Epidemics on Time Dependent Adaptive Networks**

Ira B. Schwartz
US Naval Research Laboratory
Code 6792
Washington, DC

Luis Mier-y-Teran

JHU Bloomberg School of Public Health and NRL

Washington,DC

Leah B. Shaw
College of William and Mary
Williamsburg, VA

Brandon S. Lindley
NRL/Wagner Associates
Hampton, VA

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## Modeling Ebola Data

Camacho, A., et al., Potential for large outbreaks of Ebola virus disease. Epidemics (2014), http://dx.doi.org/10.1016/j.epidem.2014.09.003

**Table 1** Previously published estimates of basic reproduction number,  $R_0$ , for Ebola.

Location	Date	$R_0$	95% CI (if given)	Reference
DRC	1995	1.83		Chowell et al. (2004)
		3.65	3.05-4.33	Ferrari et al. (2005)
		2.7	1.9-2.8	Legrand et al. (2007)
		1.38		Lekone and Finkenstädt (2006)
		2.22	1.9-2.73	Ndanguza et al. (2013)
		1.93	1.74-2.78	White and Pagano (2008)
Uganda	2000/1	1.34		Chowell et al. (2004)
		1.79	1.52-2.30	Ferrari et al. (2005)
		2.7	2.5-4.1	Legrand et al. (2007)

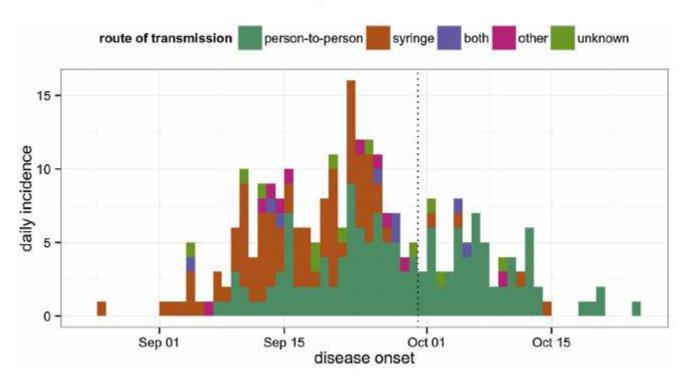
Caveat: Hospital-based infection played a substantial role in parameter fitting

Not possible to identify route of transmission nor human behavior.

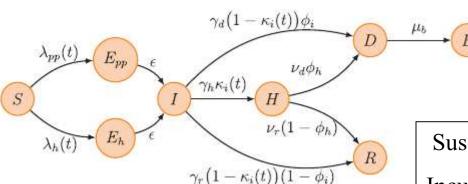
Revisited 1976 outbreak to reveal sources of transmission

## Daily Incidence for Zaire 1976





# Modeling Study of Ebola



$$\frac{dS}{dt} = -(\beta_i(t)I + \beta_h(t)H + \beta_d(t)D)\frac{S}{N}$$

$$\frac{dE_{pp}}{dt} = (\beta_i(t)I + \beta_d(t)D)\frac{S}{N} - \epsilon E_{pp}$$

$$\frac{dE_h}{dt} = \beta_h(t)H\frac{S}{N} - \epsilon E_h$$

$$\frac{dI}{dt} = \epsilon(E_{pp} + E_h) - \Gamma_i(t)I$$

$$\frac{dH}{dt} = \gamma_h \kappa_i(t)I - (\phi_h \nu_d + (1 - \phi_h)\nu_r)H$$

$$\frac{dD}{dt} = \gamma_d(1 - \kappa_i(t))\phi_iI + \nu_d\phi_hH - \mu_bD$$

$$\frac{dR}{dt} = \gamma_r(1 - \kappa_i(t))(1 - \phi_i)I + \nu_r(1 - \phi_h)H$$

$$\frac{dB}{dt} = \mu_bD$$

Susceptible to infection (S).

Incubation exposed class (E),

Infectious (I).

Assumed latent and incubation periods are equivalent.

Recovered state (R);

Remain infectious and go into hospital (H);

Or die and remain infectious (D) until buried (B).

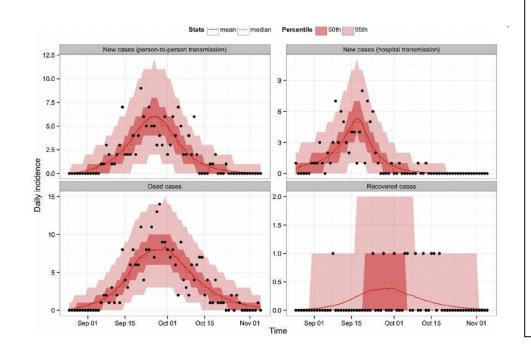
Following hospitalisation, infectious hosts also move either into the recovered or dead compartment.

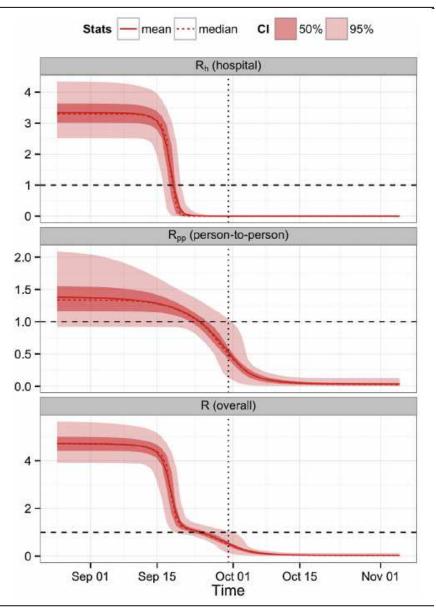
## **Temporal Reproductive**

## rates

**Table 3** Estimates of the basic reproduction number,  $R_0$ , split into different component transmission routes.

Parameter	Route of transmission	Estimates: median (95% CI)
R <sub>0h</sub>	Hospital via syringe	3.32 (2.53-4.34)
$R_{0pp}$	Person-to-person (in community and during funeral)	1.34(0.92-2.11)
$R_0$	Overall	4.71 (3.92-5.66)



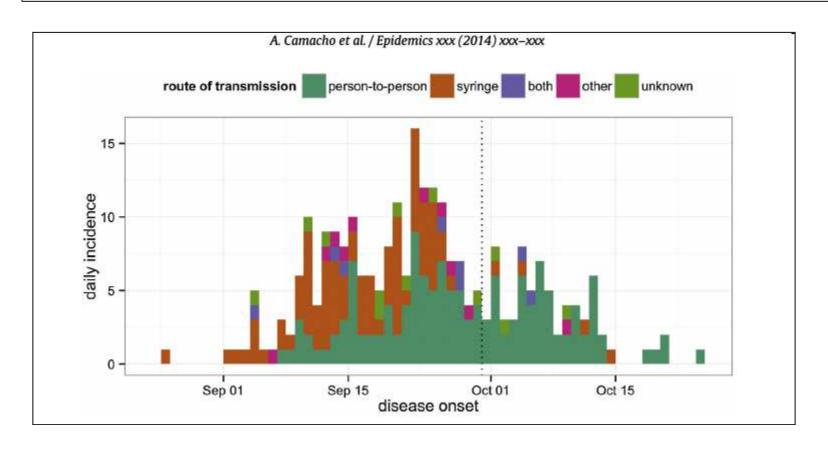


#### Conjectured Human Behavior

There was evidence that hospital and person-to-person transmission declined over the course of the 1976 outbreak.

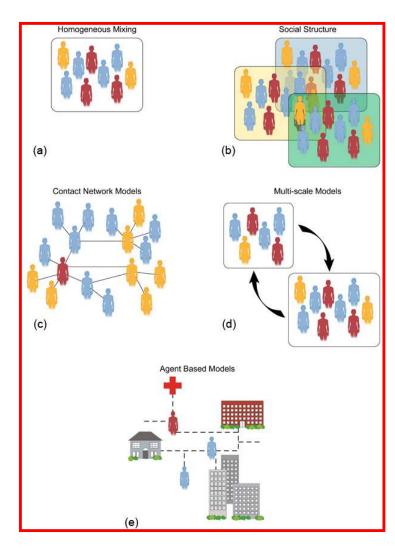
Epidemiological reports the community stopped coming to the outpatient department as they associated the epidemic with the Yambuku Mission Hospital, which eventually was closed on 30th September.

As time went on the population became very "suspicious" and did not touch the corpses anymore, not even to bury them.



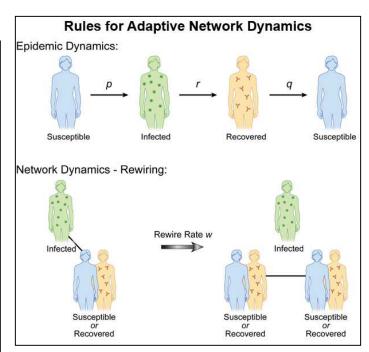
## **Background: Networks**

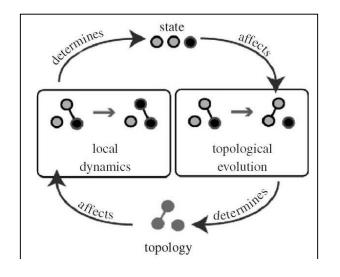
- Interconnected systems are often modeled as a network
- Structure of static networks has been well studied (Albert and Barabasi 2002, Newman 2003, plus gazillions of others)
- Some aspects of network dynamics have been studied (usually link dynamics or node dynamics)
  - Network growth-(Ben-Naim 2011)
  - Synchronization dynamics on nodes (Arenas et al. 2008)



#### **Adaptive Epidemic Networks**

- In real networks, both the nodes and links change in time – Dynamic networks
- Node dynamics affects network geometry Network geometry affects node dynamics Adaptive networks
- Feedback loop interaction
- Many applications
  - Human social networks
  - Fads, terrorist cell networks
  - Self-healing communications networks
  - Swarming of autonomous agents
  - Immune system networks
     Dimension/connection changing
  - Biological networks (e.g., food webs)
     Lobsters (Behringer, Nature 2006)





# Epidemics on Adaptive Social Networks

#### Epidemic dynamics: Avoidance Behavior

$$S \xrightarrow{p} I \xrightarrow{r} R \xrightarrow{q} S$$

Network dynamics—rewiring:

S: susceptible

I: infected

R: recovered

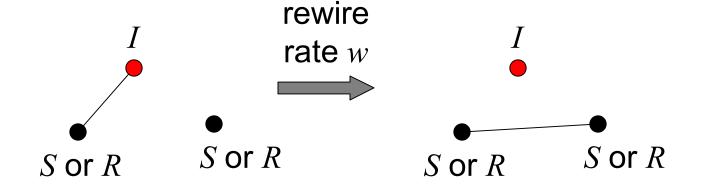
N<sub>AB</sub>: AB links

*p*: infection rate

r: recovery rate

q: resusceptibility rate

w: rewiring rate



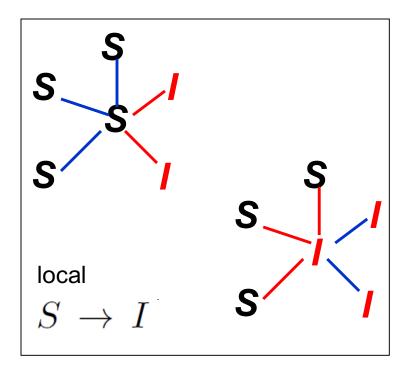
Run Monte Carlo simulation for  $N=10^4$  nodes,  $K=10^5$  links

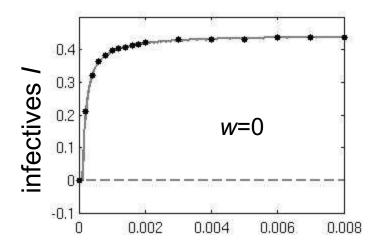
(Shaw and Schwartz PRE 77: 066101, 2008)

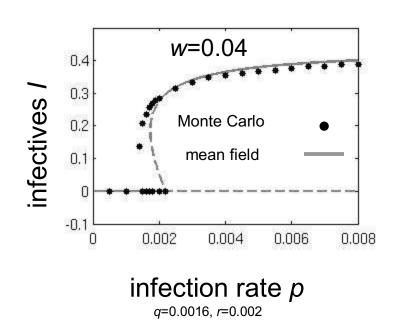
# **BifurcationStructure- Creation of New States**

(Erdos-Renyi graph)

- Rewiring leads to bistable behavior
  - Extinct and endemic states
- As rewiring rate w increases, larger infection rate p is needed for disease to persist



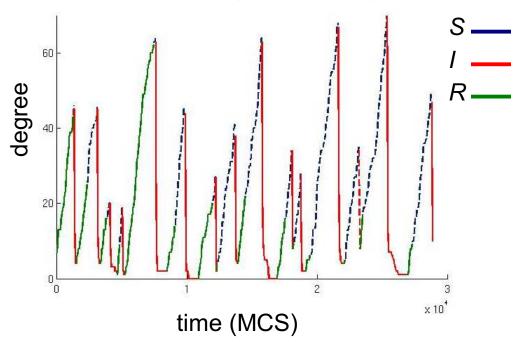




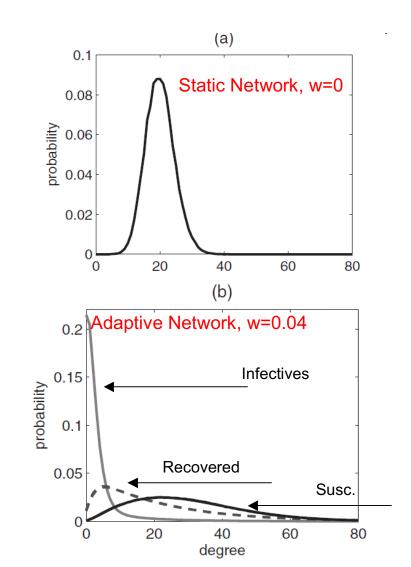
#### Network structure analysis-Degree Distribution

*q*=0.0016, *r*=0.002

Time series for degree of a single node:

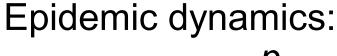


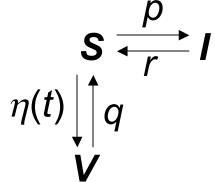
Node degree cycles in time *I* loses links *R* and *S* gain links



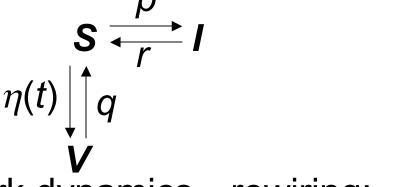
# Extinction Processes And Vaccination Control

#### Rules for Network Dynamics with Vaccination





Network dynamics—rewiring:



S: susceptible

*I*: infected

V: vaccinated

**p**: infection rate

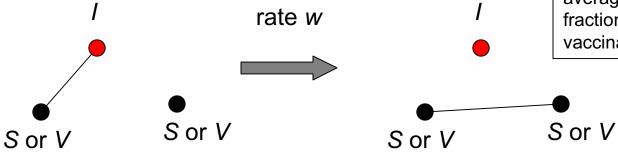
**r**: recovery rate

 $\eta(t)$ : vaccination rate

**q**: resusceptibility rate

w: rewiring rate

Vaccination rate is Poisson where events happen with average frequency  $\nu$  and a fraction A of susceptibles are vaccinated in each event.



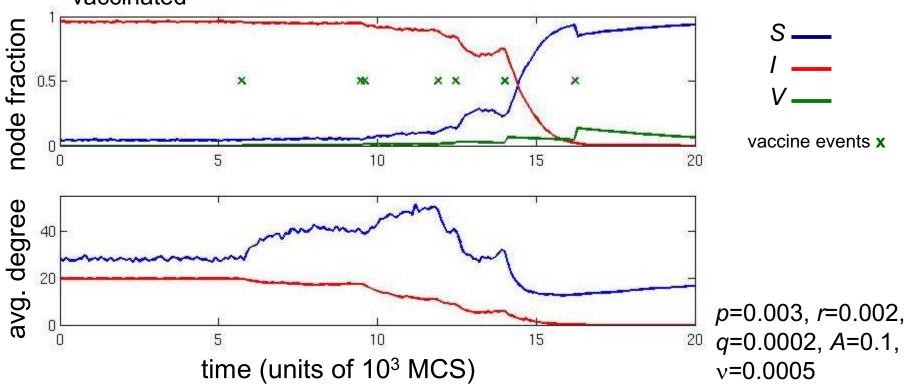
rewire

Run Monte Carlo simulation for **N**=10<sup>4</sup> nodes, **K**=10<sup>5</sup> links

L. B. Shaw and I. B. Schwartz, Phys. Rev. E (2010).

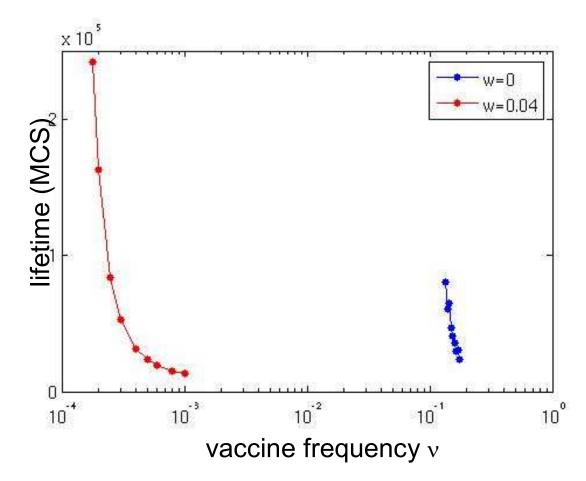
#### Effect of vaccination and rewiring on degree

- Vaccination occurs on susceptible nodes
- In the adaptive network, susceptible nodes have higher degree due to rewiring
- Vaccination of high degree nodes provides better protection (e.g., Pastor-Satorras and Vespignani PRE 65: 036104, 2002)
- In the static network, high degree nodes tend to be infected and are not vaccinated



# Controlling Adaptive Networks: Lifetime of Endemic State

- Poisson-distributed pulse vaccine control
- Compute lifetime of the infected state
- Average over 100 runs
- Rewiring in combination with vaccination significantly shortens the disease lifetime



#### **Summary**

- Developed a model for epidemics with adaptation to account for social interactions
- Developed and analyzed a corresponding mean field model, which captures many aspects of the full adaptive network system
- We identified regimes of onset thresholds, new state creation as functions of rewiring rates (adaptivity)-Possible oscillations

#### **List of Publications**

- Fluctuating epidemics on adaptive networks, LB Shaw, IB Schwartz, Physical Review E 77 (6), 066101 (2008)
- Noise induced dynamics in adaptive networks with applications to epidemiology, L. B. Shaw and I. B. Schwartz, in <u>Adaptive Networks</u>, <u>Understanding Complex Systems</u>, eds. T. Gross and H. Sayama, pp. 209-227 (2009).
- Enhanced vaccine control of epidemics in adaptive networks, L. B. Shaw and I. B. Schwartz, Phys. Rev. E 81: 046120 (2010).
- Rewiring for adaptation, I. B. Schwartz and L. B. Shaw, Physics 3: 17 (2010).
- Maximal sensitive dependence and the optimal path to epidemic extinction,
   E. Forgoston, S. Bianco, L. B. Shaw, and I. B. Schwartz, Bull. Math. Biol.
   73: 495-514 (2011).
- Converging towards the optimal path to extinction, I. B. Schwartz, E. Forgoston, S. Bianco, and L. B. Shaw, J. Roy. Soc. Interface doi: 10.1098/□rsif.2011.0159 (2011)
- Recruitment dynamics in adaptive social networks, MS Shkarayev, IB Schwartz, LB Shaw, J. Phys. A: Mathematical and Theoretical 46 (24), 245003 (2013)
- An iterative action minimizing method for computing optimal paths in stochastic dynamical systems, BS Lindley, IB Schwartz, Physica D: Nonlinear Phenomena 255, 22-30 (2013)