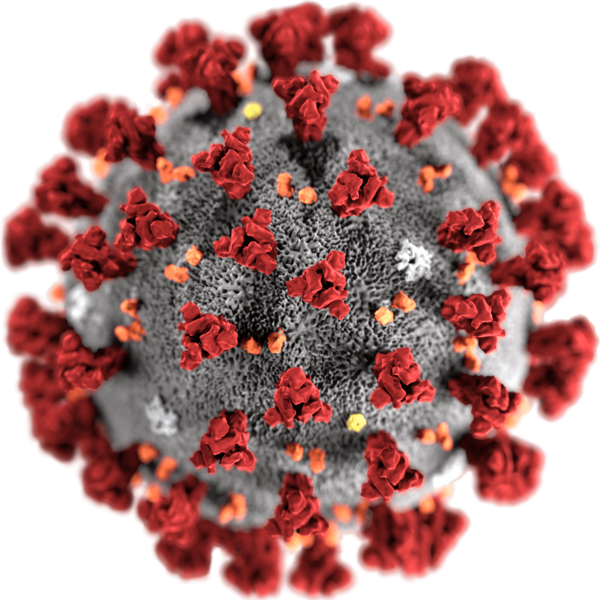
Class slides for Tuesday, November 3: Disease models

Matthew J. Salganik

COS 597E/SOC 555 Limits to prediction
Fall 2020, Princeton University

Vote

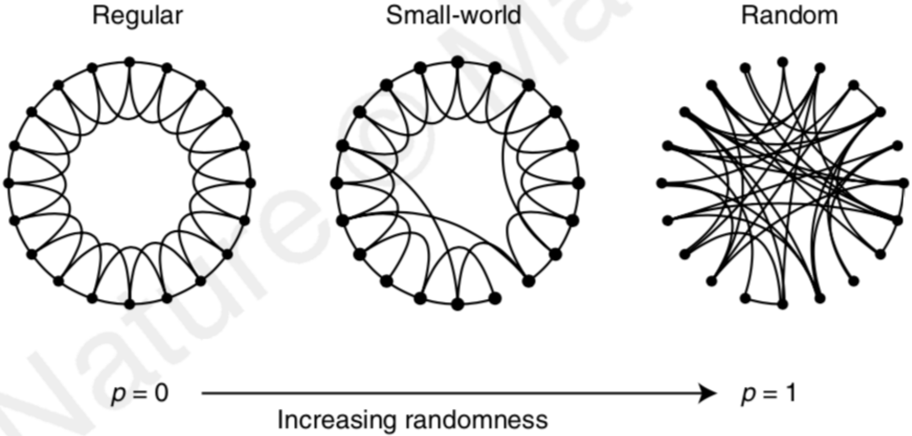


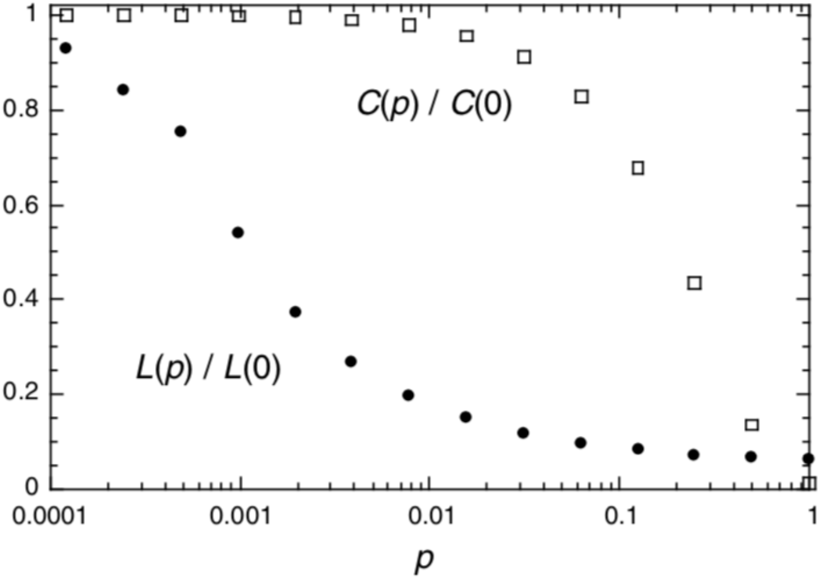
https://phil.cdc.gov/Details.aspx?pid=23312

Collective dynamics of 'small-world' networks

Duncan J. Watts[*] & Steven H. Strogatz

Department of Theoretical and Applied Mechanics, Kimball Hall, Cornell University, Ithaca, New York 14853, USA

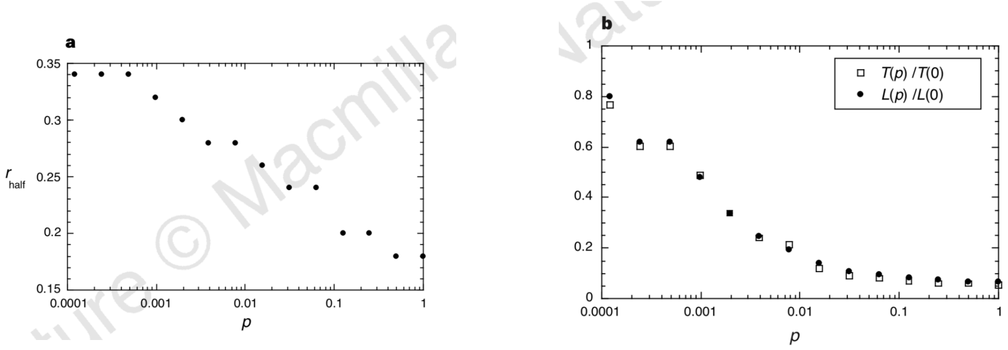

Regular
Small-world
Random
p = 0
Increasing randomness
p = 1



1
0.8
0.6
0.4
0.2
0
0.0001
0.001
0.01
0.1
1
p
C(p) / C(0)
L(p) / L(0)

Table 1 Empirical examples of small-world networks

	L_{actual}	L_{random}	C_{actual}	C_{random}
Film actors	3.65	2.99	0.79	0.00027
Power grid	18.7	12.4	0.080	0.005
C. elegans	2.65	2.25	0.28	0.05



Main result: Small change in network structure can make big difference for how infectious a disease needs to be to spread (a) and how quickly a highly infectious disease spreads (b)

Main take away for our class

- sometimes small changes in network structure can make a big difference in disease spread, but sometimes large changes can make no difference

Time evolution of predictability of epidemics on networks

Petter Holme[1, 2, 3, *] and Taro Takaguchi[4, 5]

[1] *Department of Energy Science, Sungkyunkwan University, Suwon 440–746, Korea*
[2] *Department of Physics, Umeå University, 90187 Umeå, Sweden*
[3] *Department of Sociology, Stockholm University, 10961 Stockholm, Sweden*
[4] *National Institute of Informatics, 2-1-2 Hitotsubashi, Chiyoda-ku, Tokyo, 101-8430, Japan*
[5] *JST, ERATO, Kawarabayashi Large Graph Project, 2-1-2 Hitotsubashi, Chiyoda-ku, Tokyo, 101-8430, Japan*

- SIR model on different networks

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- β probability of one person infecting another, ν rate of recovery (from I to R)

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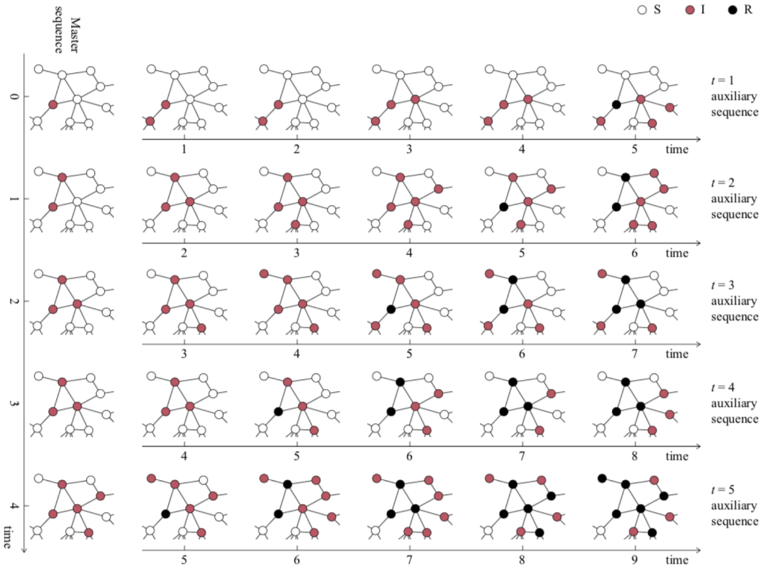
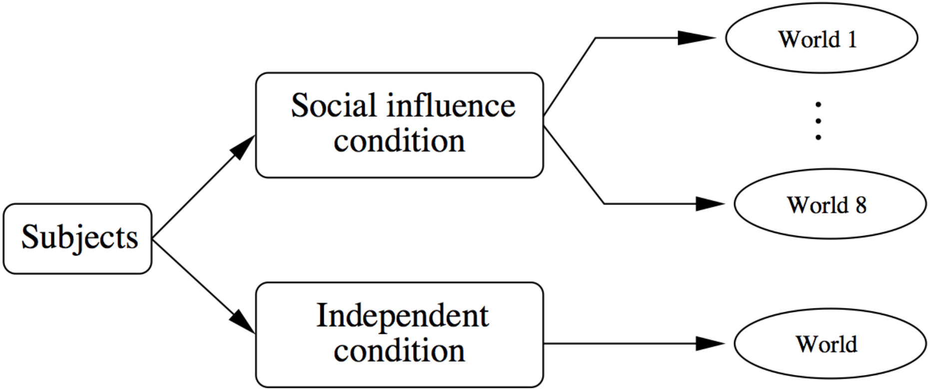
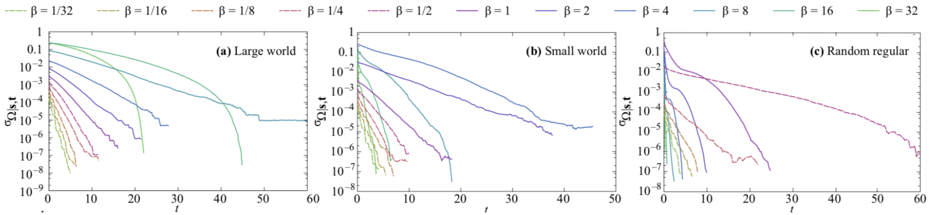
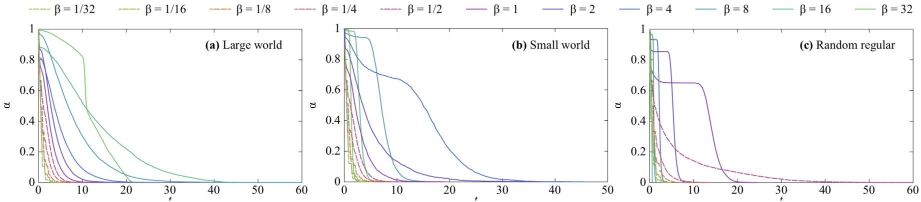
- β probability of one person infecting another, ν rate of recovery (from I to R)
- Assume that we have perfect information about the network and disease state of each person (they call this "limit of maximum information")


S
I
R
Master sequence
0
1
2
3
4
time
t = 1 auxiliary sequence
1 2 3 4 5 time
t = 2 auxiliary sequence
2 3 4 5 6 time
t = 3 auxiliary sequence
3 4 5 6 7 time
t = 4 auxiliary sequence
4 5 6 7 8 time
t = 5 auxiliary sequence
5 6 7 8 9 time

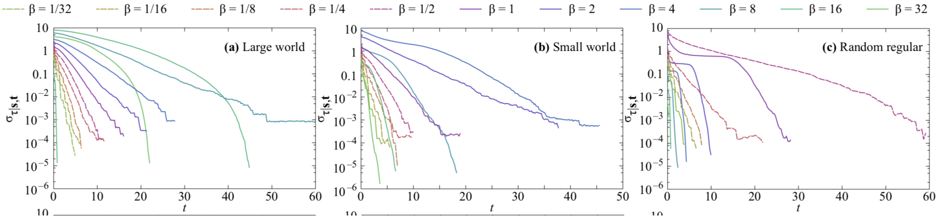

Subjects
Social influence condition
Independent condition
World 1
World 8
World



Main result: Unpredictability of outbreak size (Ω) goes down exponentially fast



Main result: Fraction of surviving outbreaks decreases over time. For large world, most infectious survive the longest, for small world and regular random, intermediate infectiousness survives the longest.



Main result: Unpredictability of extinction time (τ) goes down exponentially fast

2 main take aways

- using simulation it is possible to study how unpredictability changes over time

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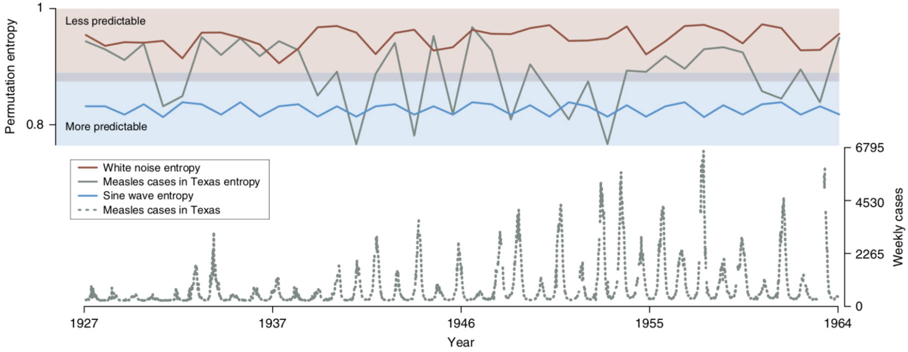
- using simulation it is possible to study how unpredictability changes over time
- outbreaks are most unpredictability right at the beginning and get much easier to predict over time in this toy setting. Think back to ex-ante vs peeking strategies. think forward to prediction vs surveillance.

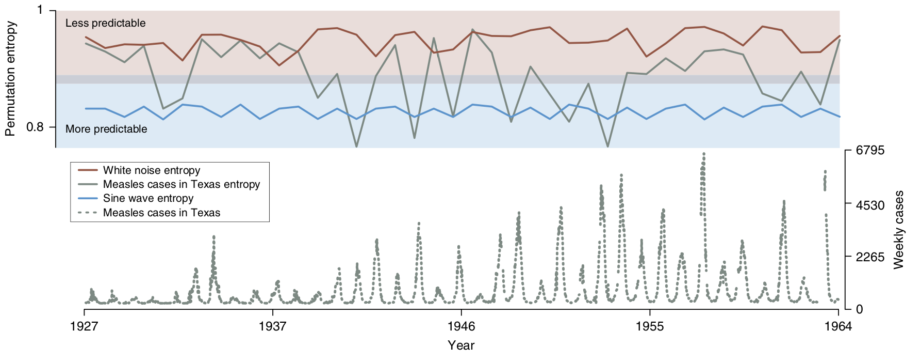
On the predictability of infectious disease outbreaks

Samuel V. Scarpino[1,2,3,4,5,6] & Giovanni Petri[6,7]

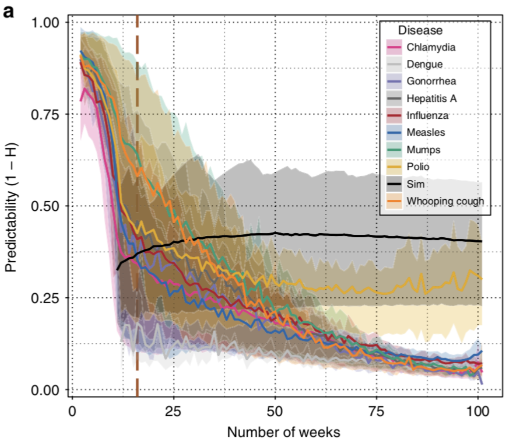
https://www.youtube.com/watch?v=LaTAq1NUTPE



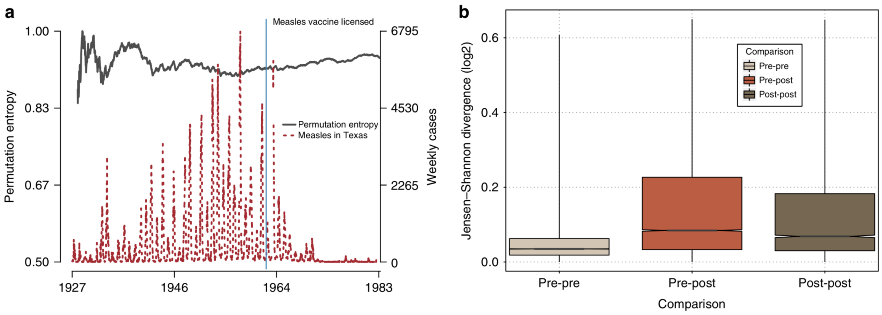
Is being correlated with forecast limits in other systems enough?



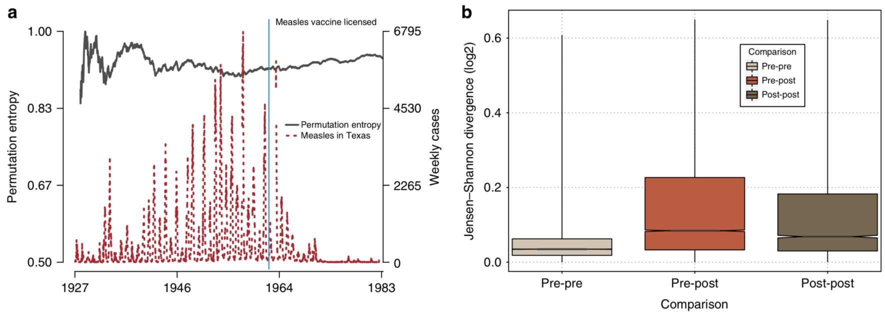
For the sake of this class, we will assume that these results are robust with respect to how one calculates permutation entropy



We find that all diseases show a clear decrease in predictability with increasing time series length , which implies that accumulating longer stretches of time-series data for a given disease does not translate into improved predictability. However, we also find strong



start of widespread vaccination. We consistently observe that predictability decreases after vaccination, again with significance determined by permutation test (Fig. 4a). We also find that the



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What information would you need to predict what will happen after a vaccine is introduced?

From these results, we can draw three conclusions. First, differences in the average reproductive number, coupled with heterogeneity in the number of secondary infections, can drive differences in predictability across diseases and outbreaks, which is related to results on predicting disease arrival time on networks[44] and to recurrent epidemics in hierarchical meta populations[45]. Second, the permutation entropy could provide a model-free approach for detecting epidemics, which is similar to a recent model-based approach based on bifurcation delays[46–48]. Finally, as outbreaks grow and transition to large-scale epidemics, they should become more predictable, which—as seen in Figs. 1 and 3—appears to be true for real-world diseases as well and agrees with earlier results on how permutation entropy relates to the predictability of nonlinear systems[32].

strate that outbreaks should be predictable. However, as outbreaks spread—and spatiotemporally separated waves become entangled with the substrate, human mobility, behavioral changes, pathogen evolution, etc.—the system is driven through a space of diverse model structures, driving down predictability despite increasing time-series lengths. Taken together, our results

Stepping back

- We can make precise statements about toy models (Holme and Takaguchi) or imprecise statements about real data (Scarpino and Petri). Ugh.

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- All these papers assume an isolated system.

Pandemics: spend on surveillance, not prediction

Measuring the present is more important than predicting the future, and it requires that we change our focus.

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- It is hard to learn about predictability in realistic systems
- Prediction might be less important than measurement

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