Georgia Tech Quantitative Biosciences
Interdisciplinary Graduate Program

PH.D. PROGRAM IN QUANTITATIVE BIOSCIENCES

APPLY BY DECEMBER 1ST

QBIOS.GATECH.EDU

QBioS Research and Training: Spanning Molecules to Ecosystems

An Interdisciplinary Graduate Program

The Ph.D. in Quantitative Biosciences (QBioS) is a new interdisciplinary program at Georgia Tech, founded in 2015 and supported by a consortium of more than 50 program faculty from six home schools in the College of Sciences.

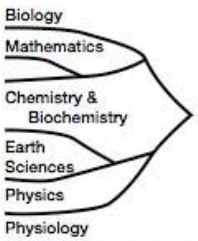
A Novel and Flexible Training Program

Featuring a flexible training program, including:
- Foundational courses in Quantitative Biosciences
- Rotations in modeling and/or experimental groups
- Selection of thesis advisor from all program faculty
- Rigorous and personalized quantitative training
- Five-year program of study from entrance to defense

Quantitative Biosciences integrates the physical, mathematical and biological sciences, enabling the discovery of scientific principles underlying the dynamics, structure, and function of living systems.

Home Schools

Biological Sciences, Chemistry & Biochemistry, Earth & Atmospheric Sciences, Mathematics, Physics, & Psychology

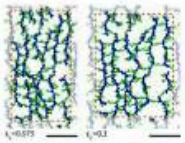


A Growing QBioS Student Community

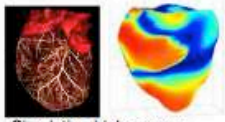
- **25 students in first three cohorts, in total.**
- **Feature diverse backgrounds and scientific training.**
- **Multiple award winners (including 2 NSF GRFP awardees).**
- **Students come from the USA (12), China (4), Mexico (3), Bosnia, France, India, Iran, Israel, and Romania.**

JOY PUTNEY
Inaugural cohort & NSF GRFP awardee (2017)

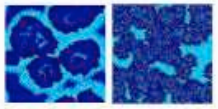
When I was considering graduate schools, the Quantitative Biosciences program at Georgia Tech stood out because of its interdisciplinary approach to the biological sciences. My campus visit impressed me with the quality of the faculty, both in terms of their world-class research and care for students. Both before and after I accepted the admission offer, it was clear that program faculty were invested in each individual student and wanted us all to succeed.



Bacterial cell wall structures in atom-scale simulations

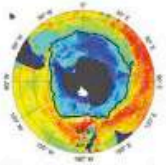


Simulating high energy shocks in a fibrillating heart



Clusters and waves of infections of microbes by viruses

Biomechanic principles of subsurface "swimming"



Biogeochemical dynamics resulting from the coupling of physical and biological processes

Contact Information:

Director of QBioS

Joshua S. Weitz, Professor
School of Biological Sciences
School of Physics
director@qbios.gatech.edu

Admissions

Lisa Redding
Academic Program Coordinator
QBioS Program
admissions@qbios.gatech.edu

QBioS is now accepting applications from students interested in integrating quatitative methods with bioscience research.

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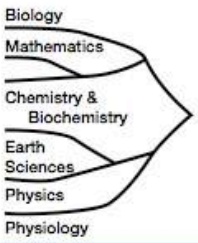

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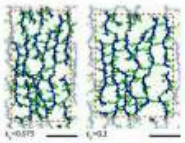

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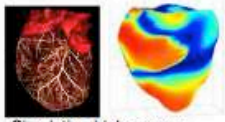

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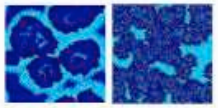

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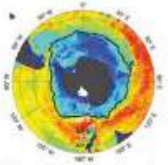

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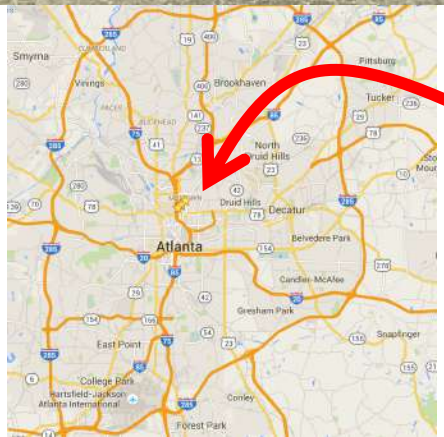
Cohorts, from 2016-2018 from bottom to top.


Detroit
Chicago
New York
PENNSYLVANIA
OHIO
ILLINOIS
INDIANA
Indianapolis
Washington
WEST VIRGINIA
VIRGINIA
KENTUCKY
Nashville
TENNESSEE
NORTH CAROLINA
Charlotte
Atlanta
SOUTH CAROLINA
MISSISSIPPI
ALABAMA
GEORGIA
Jacksonville
New Orleans
Orlando
FLORIDA
Miami


Smyrna
Vinings
Brookhaven
Pittsburg
Tucker
North Druid Hills
Druid Hills
Decatur
Atlanta
Belvedere Park
Candler-McAfee
Gresham Park
East Point
Snapfinger
College Park
Hartsfield-Jackson Atlanta International
Conley
Forest Park

ICTP-SAIFR
Summer Workshop
Mathematical Models of Evolution
Sao Paulo
Jan 21-26, 2019

Support:
ARO; NSF – DimBio;
NSF – Bio Oc; NSF PoLS
Simons Foundation - SCOPE,
Georgia Tech

VIRUS ECOLOGY AND EVOLUTION:

PRINCIPLES & APPLICATIONS OF VIRUS-MICROBE DYNAMICS

Joshua S. Weitz
School of Biological Sciences and School of Physics
Graduate Program in Quantitative Biosciences
Georgia Institute of Technology

Joshua S. Weitz, Georgia Tech, School of Biological Sciences & Physics
Email: jsweitz@gatech.edu, Twitter: @joshuasweitz
Web: http://ecotheory.biology.gatech.edu


DOS EQUIS
XX

	Replica																			
Experiment	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	10	18	125	10	14	27	3	17	17											
2	29	41	17	20	31	30	7	17												
3	30	10	40	45	183	12	173	23	57	51										
4	6	5	10	8	24	13	165	15	6	10										
5	1	0	3	0	0	5	0	5	0	6	107	0	0	0	1	0	0	64	0	35
6	1	0	0	7	0	303	0	0	3	48	1	4								
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MUTATIONS OF BACTERIA FROM VIRUS SENSITIVITY TO VIRUS RESISTANCE[1,2]

S. E. LURIA[3] AND M. DELBRÜCK

Indiana University, Bloomington, Indiana, and Vanderbilt University, Nashville, Tennessee

Received May 29, 1943

GENETICS 28: 491 November 1943

TABLE 2

The number of resistant bacteria in series of similar cultures.

EXPERIMENT NO.	1	10	11	15	16	17	21a	21b
Number of cultures	9	8	10	10	20	12	19	5
Volume of cultures, cc	10.0	10.0	10.0	10.0	.2*	.2*	.2	10.0
Volume of samples, cc	.05	.05	.05	.05	.08	.08	.05	.05
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5	14	31	183	24	0	0	8	13
6	27	30	12	13	5	303	1	
7	3	7	173	165	0	0	0	
8	17	17	23	15	5	0	1	
9	17		57	6	0	3	0	
10			51	10	6	48	15	
11					107	1	0	
12					0	4	0	
13					0		19	
14					0		0	
15					1		0	
16					0		17	
17					0		11	
18					64		0	
19					0		0	
20					35			

S.E. Luria

M. Delbruck

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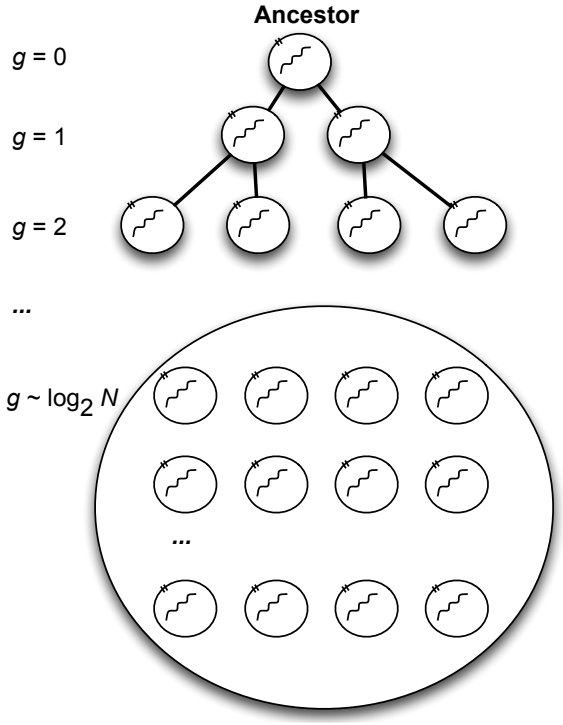
10			51	10	6	48	15	
11					107	1	0	
12					0	4	0	
13					0		19	
14					0		0	
15					1		0	
16					0		17	
17					0		11	
18					64		0	
19					0		0	
20					35			

S.E. Luria

M. Delbruck

Nobel Prize, 1969
(w/H. Chase)

A1. Growth of a bacterial population from a single ancestor



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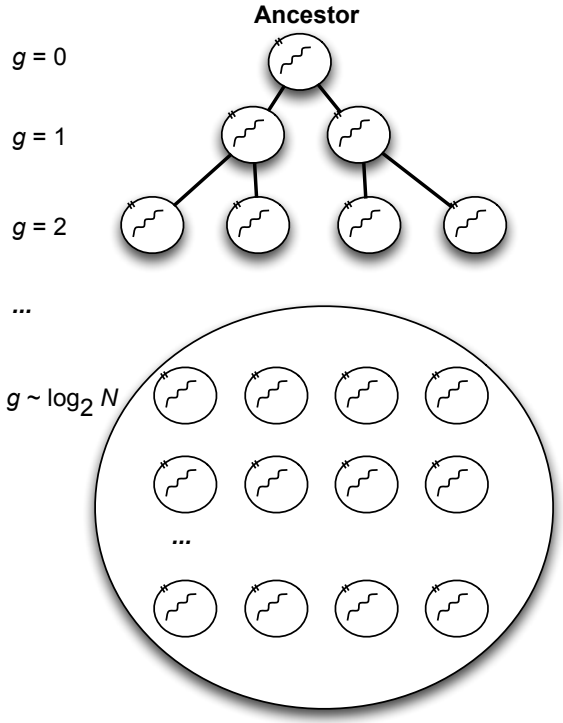

A2. Exposure of population to viruses

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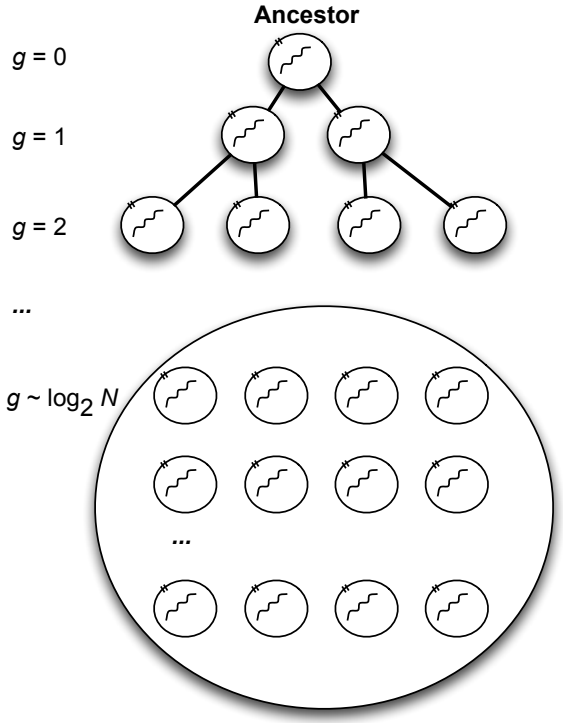

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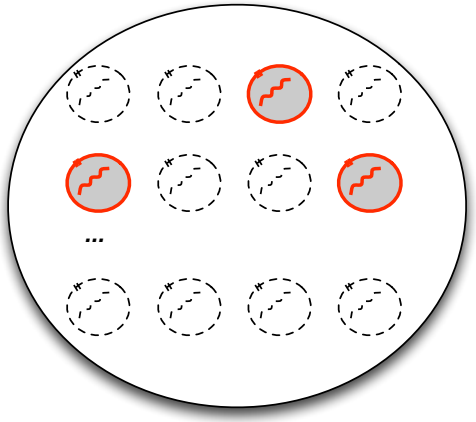
A3. Subpopulation of bacteria acquire resistance and survive viral infection.



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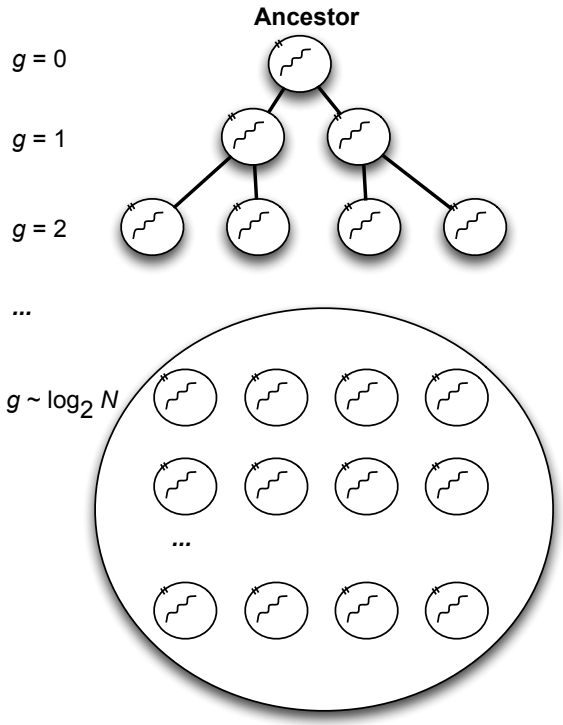

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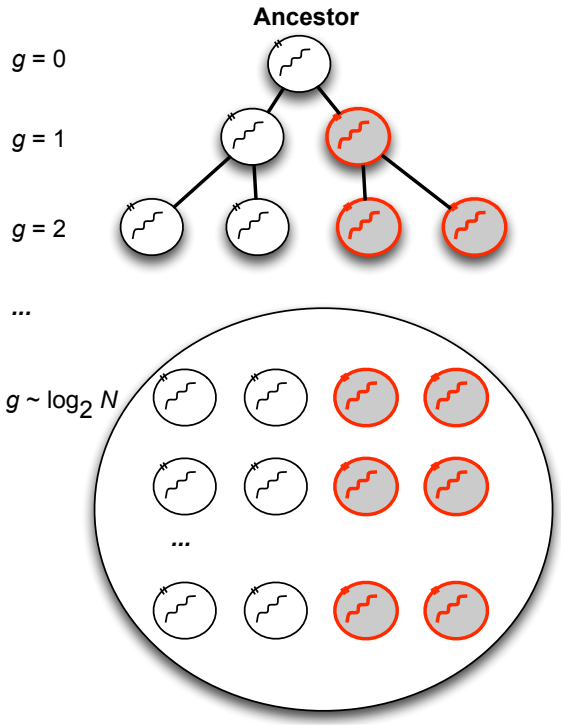

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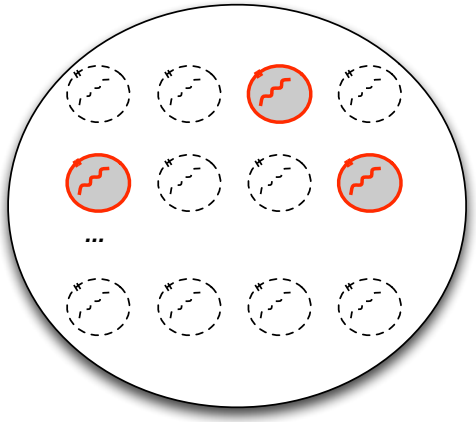

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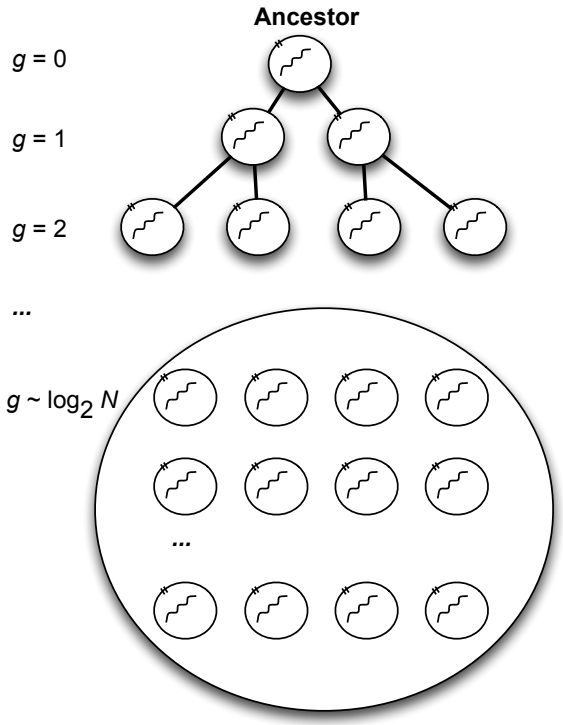

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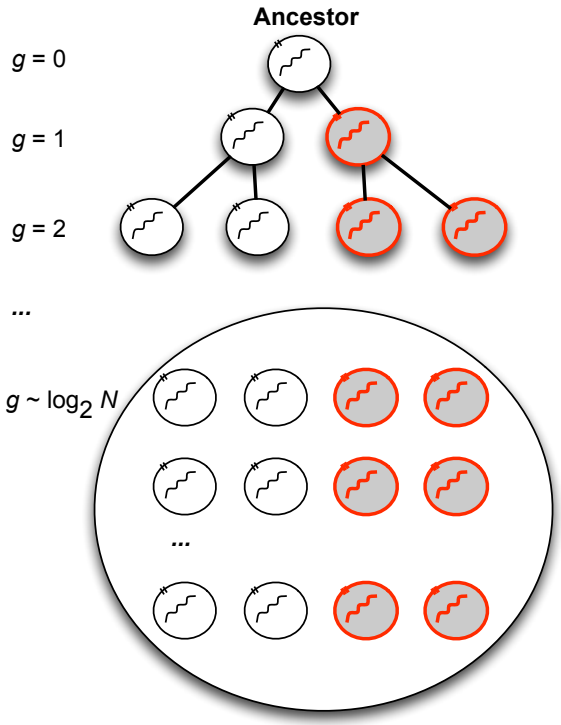

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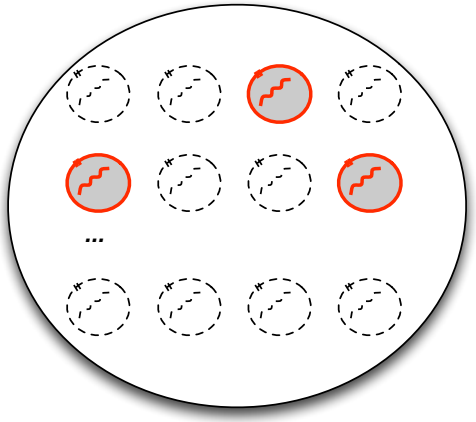

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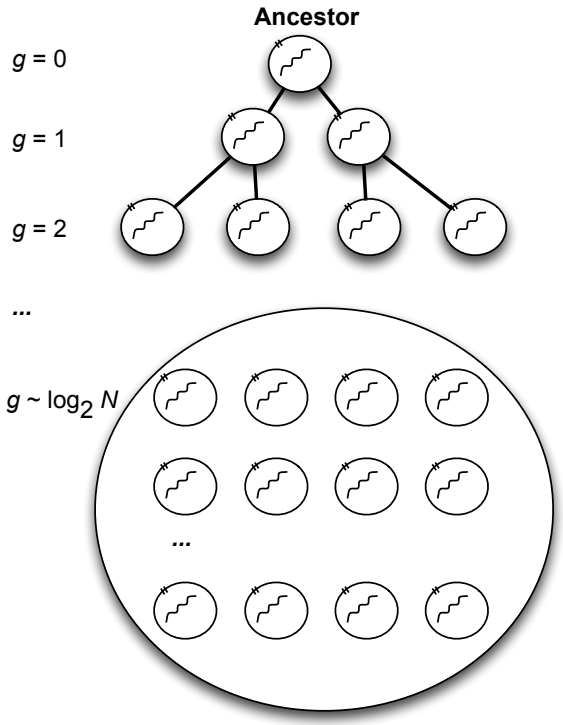

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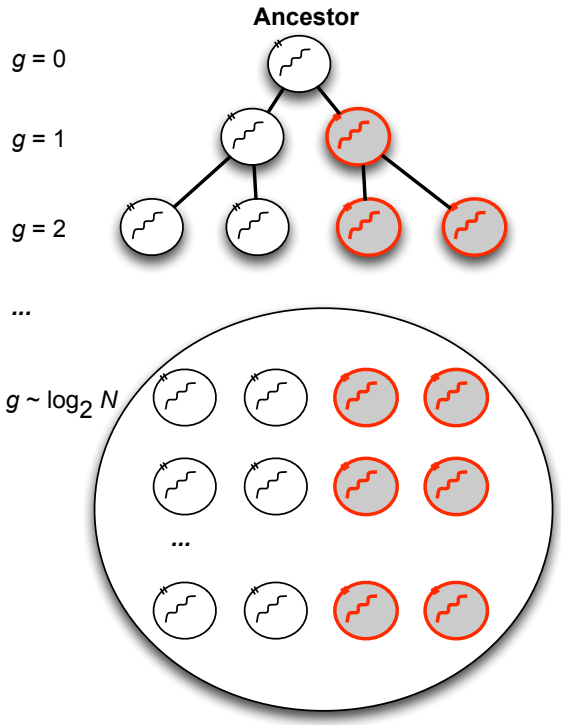

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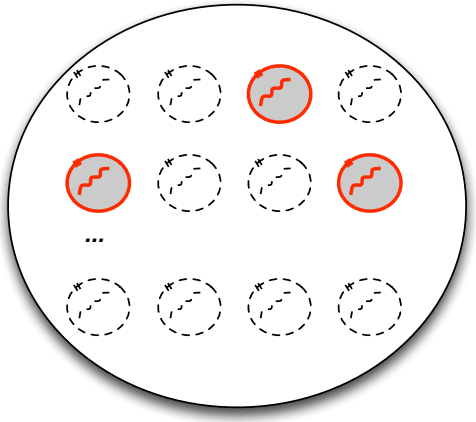
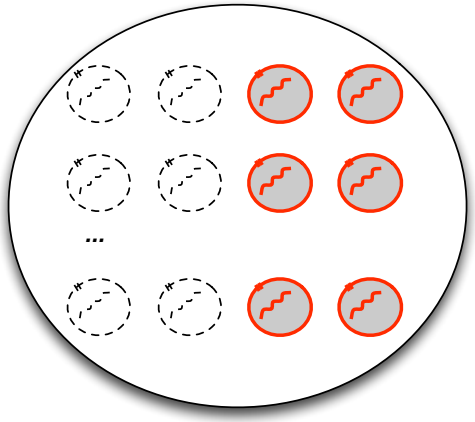


B3. Subset of resistant bacteria already present and survive viral infection.



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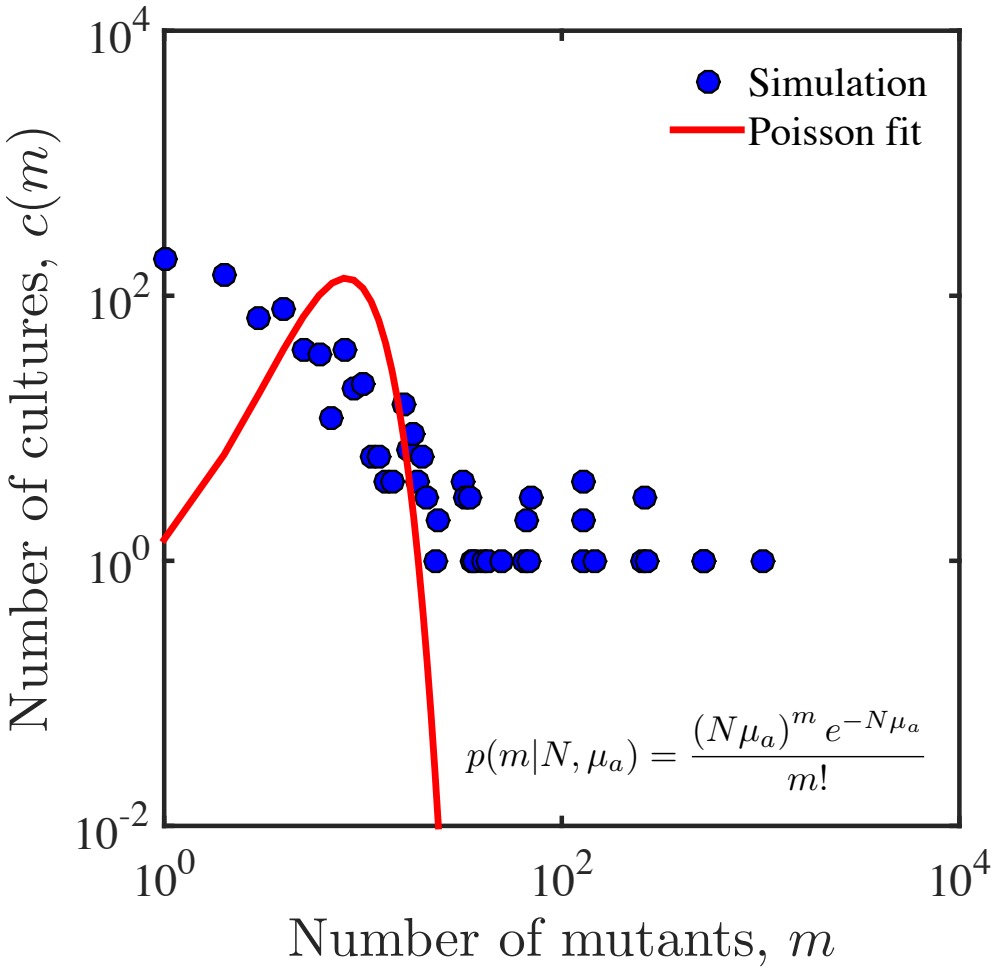

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8	38	28	35	107	13															

Viruses impose a strong selection pressure.

Host mutations that confer resistance are beneficial.

Hence, viruses induce host evolution.

But, what about the viruses?

MUTATIONS OF BACTERIAL VIRUSES AFFECTING THEIR HOST RANGE[1]

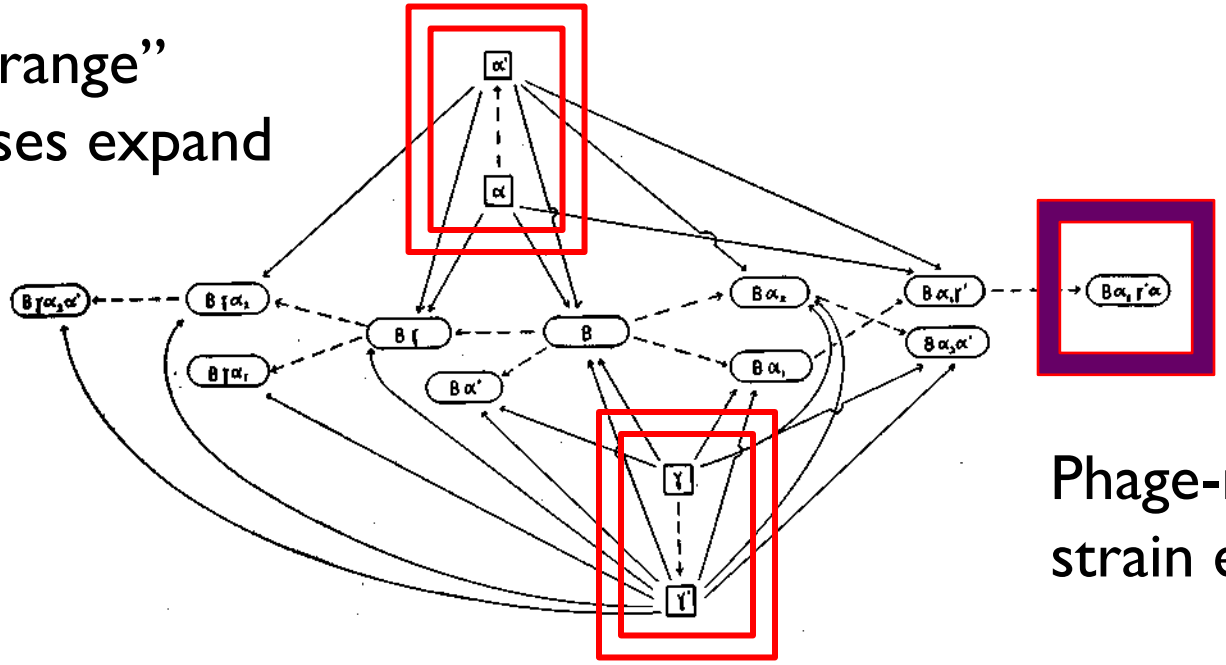
S. E. LURIA
Indiana University, Bloomington, Indiana

Received July 8, 1944

GENETICS 30: 84 Jan. 1945

S.E. Luria

"Host-range" of viruses expand



Phage-resistant host strain emerges

Phage → Infection

Bacteria - - - → Evolution

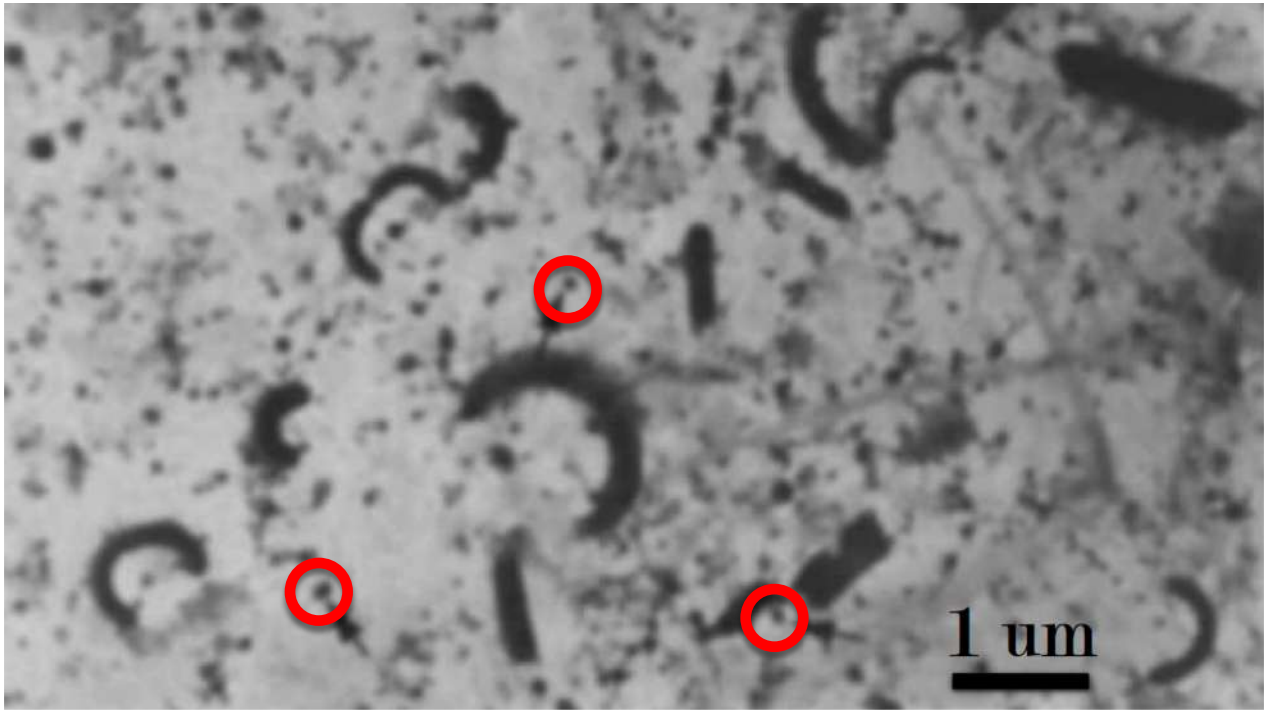
For decades, this dogma persisted…

"the coevolutionary potential of virulent phage is less than that of their bacterial hosts"

-Richard Lenski & Bruce Levin, Am. Nat. (1985)

until….

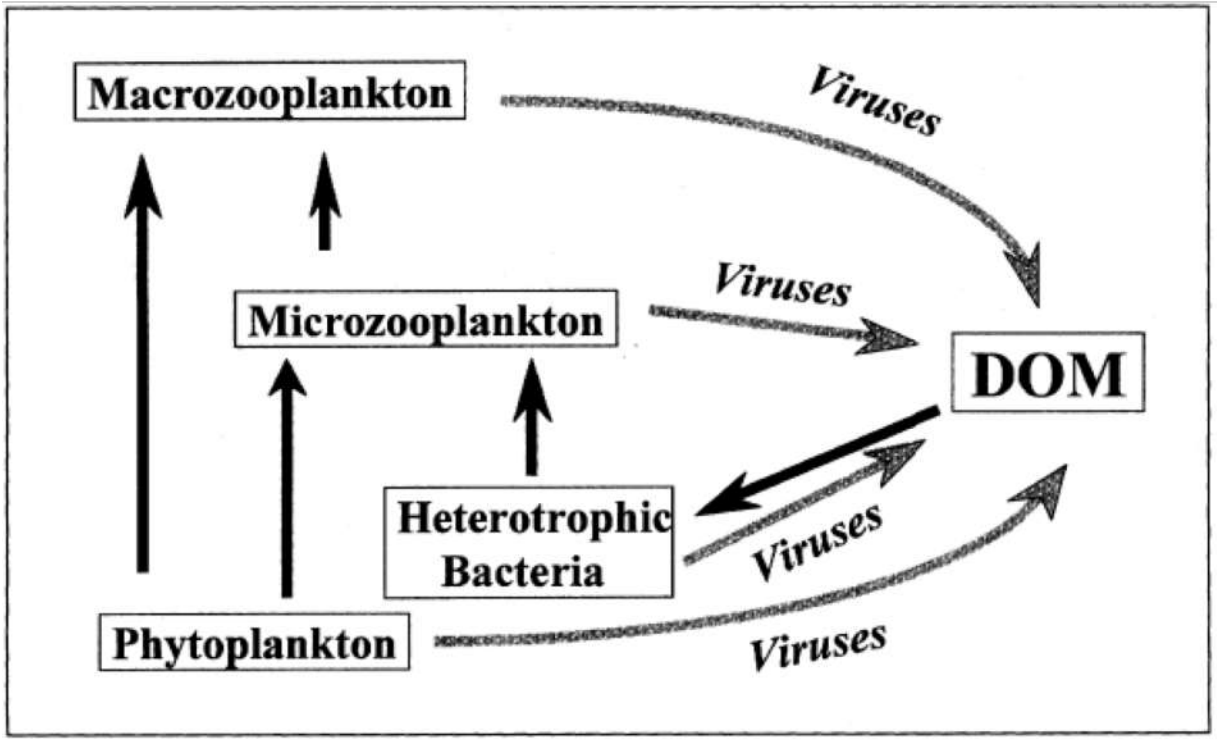
1989 - Numbers



Bergh et al., Nature 1989

"We have found up to 2.5 x 10^8 virus particles per ml in natural waters… 10^3-10^7 times higher than previous reports."

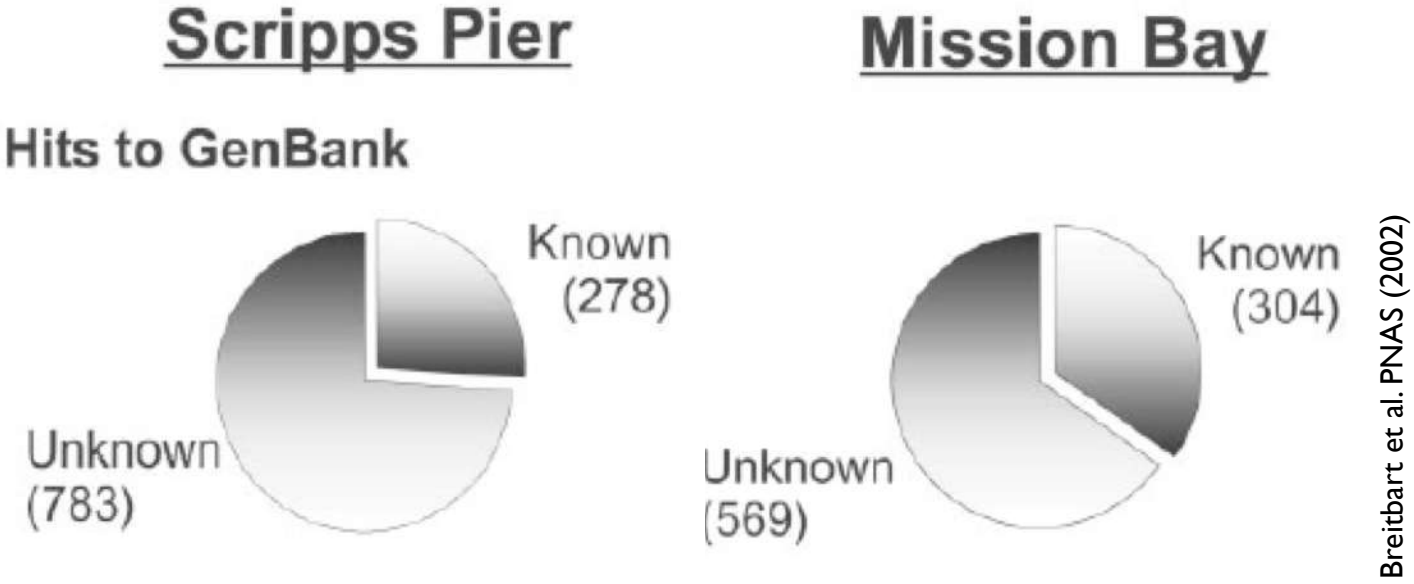
1999 - Functioning



Wilhelm & Suttle, BioScience (1999)
also, see Fuhrman, Nature (1999)

"Viruses divert the flow of carbon and nutrients… by destroying host cells and releasing the contents of these cells into the pool of DOM in the ocean."

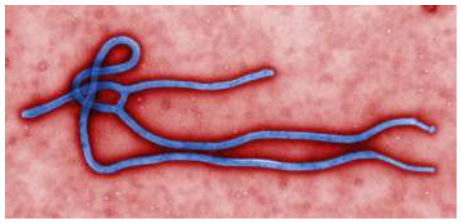
2002 - Diversity



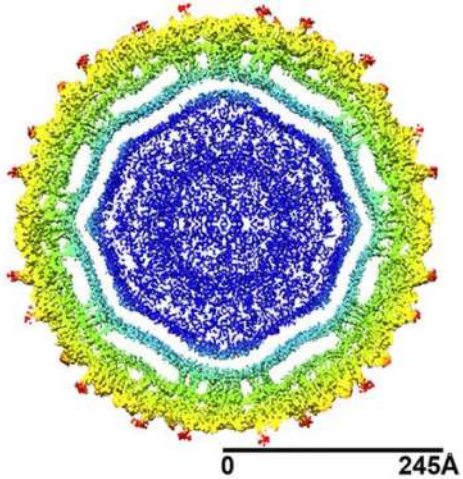
Breitbart et al. PNAS (2002)

"We report a genomic analysis of two uncultured marine viral communities. Over 65% of the sequences were not significantly similar to previously reported sequences, suggesting that much of the diversity is previously uncharacterized."

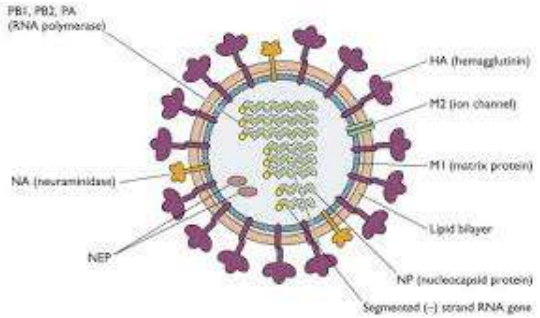
What We Talk About When We Talk About Viruses



Ebola Virus
Image source: CDC



Zika virus core
Sirohi et al. Science, 2016



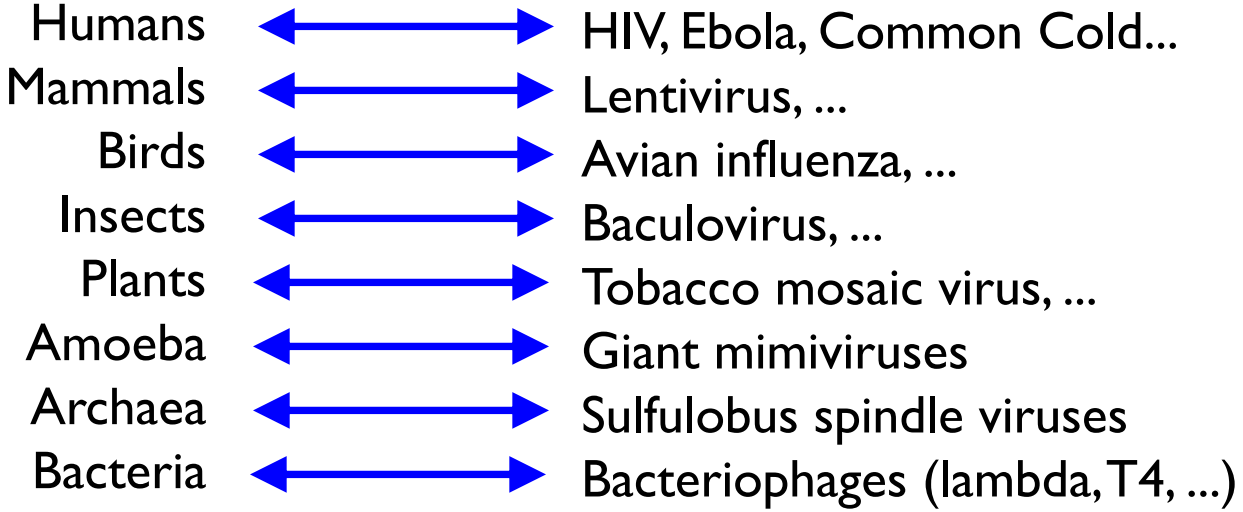
Influenza virus
virology.ws

John Moore, Getty Images
(Nature, 2014)

Source: CNN

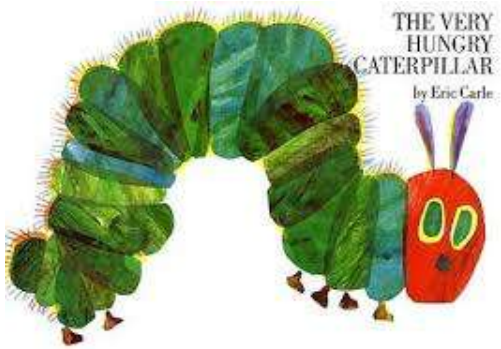
Source: CDC

And viruses infect organisms across the diversity of life

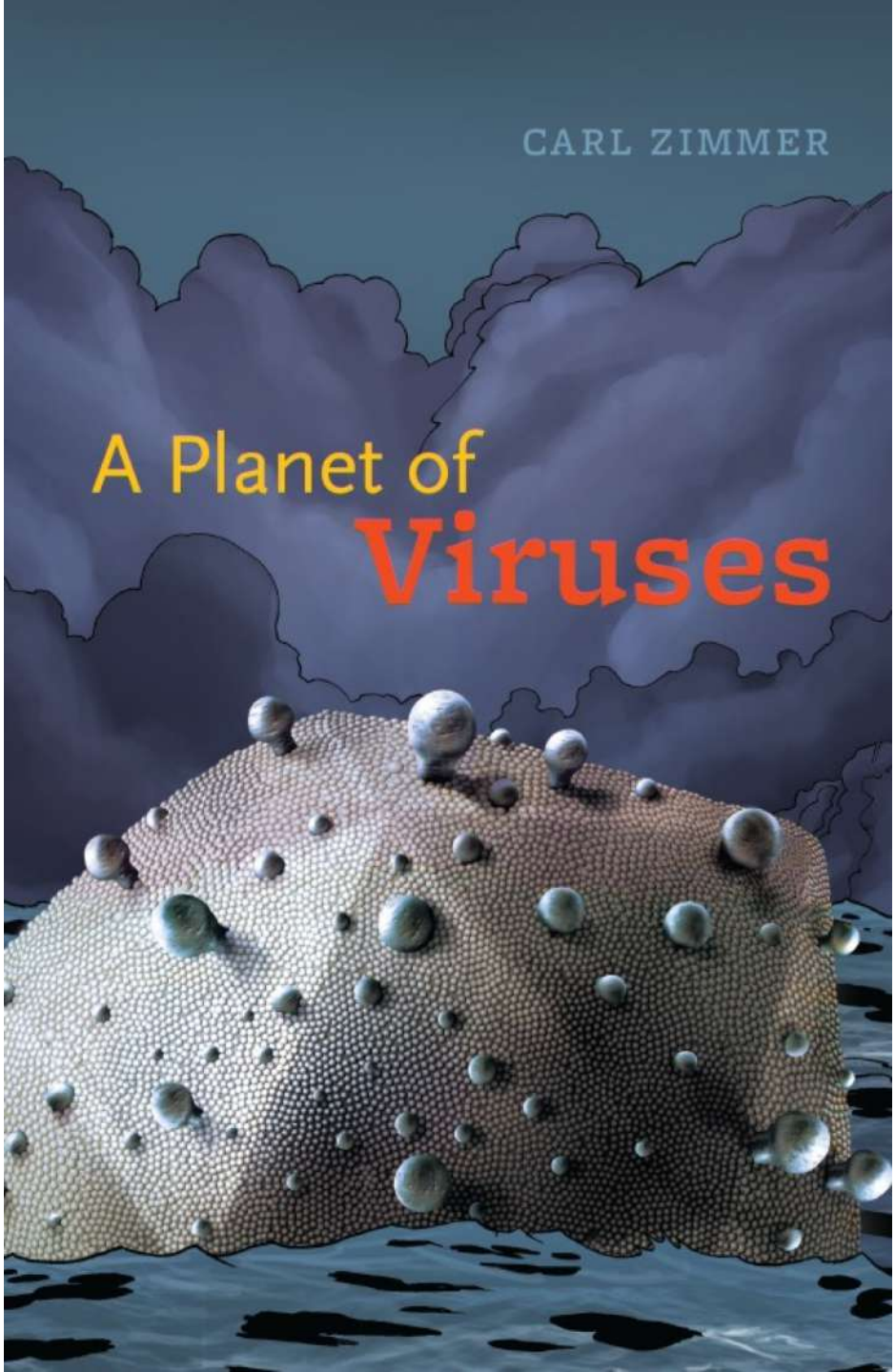


And viruses infect organisms across the diversity of life, sometimes strangely

Humans	⟷	HIV, Ebola, Common Cold...
Mammals	⟷	Lentivirus, ...
Birds	⟷	Avian influenza, ...
Insects	⟷	Baculovirus, ...
Plants	⟷	Tobacco mosaic virus, ...
Amoeba	⟷	Giant mimiviruses
Archaea	⟷	Sulfulobus spindle viruses
Bacteria	⟷	Bacteriophages (lambda, T4, ...)



Michael Grove, NPR, 2011

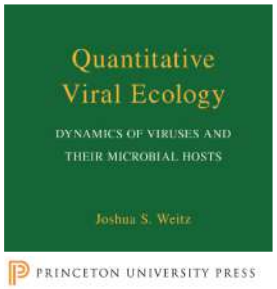

CARL ZIMMER
A Planet of
Viruses

Viral Ecology and Evolution
Lectures at the Interface

From Ecology to Evolution (Lectures 1-2)

Principles of eco-evolutionary dynamics: Monday Jan 20

Dynamics in complex communities: Wednesday Jan 22



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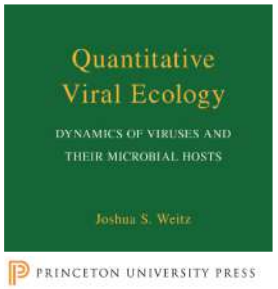
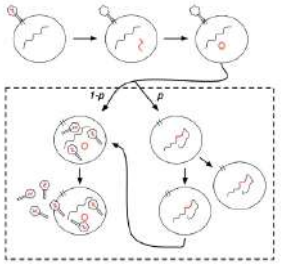


From Lysis to Latency (Lecture 3)

Friday Jan 25



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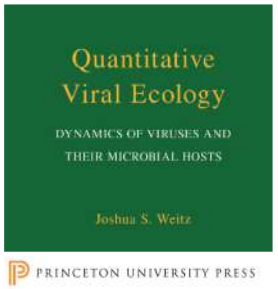

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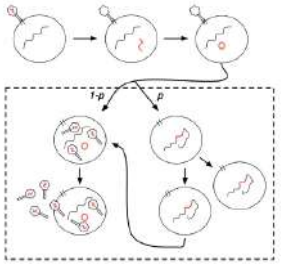
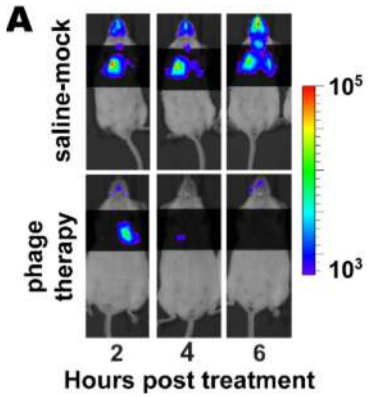


From Theory to Therapy (Lecture 4)

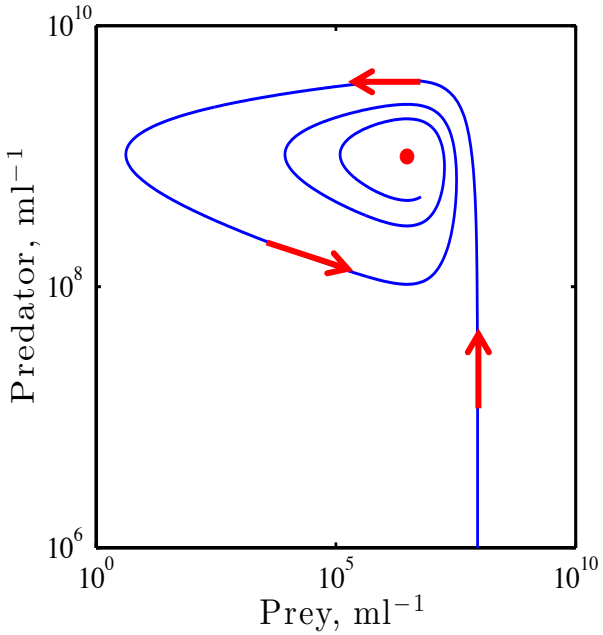
Saturday Jan 26



Throughout: theory and modeling motivated by fundamental eco-evolutionary challenges & real world applications.

Problems in Quantitative Viral Ecology
From Structure to Dynamics

- How does viral infection change microbial population dynamics?
- How does (co)evolutionary change alter viral-host population dynamics?
- What is the relationship between infection networks and host-viral dynamics in complex communities?



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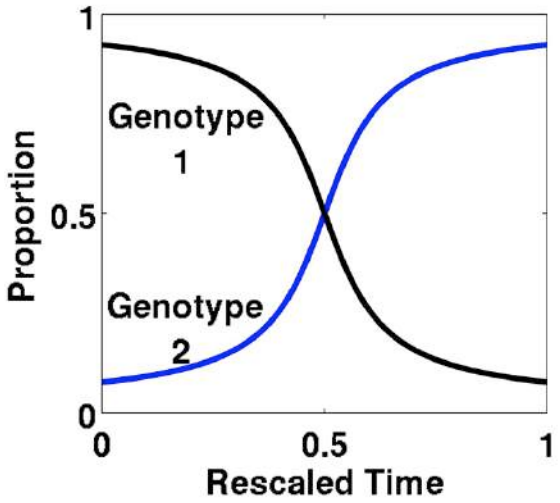

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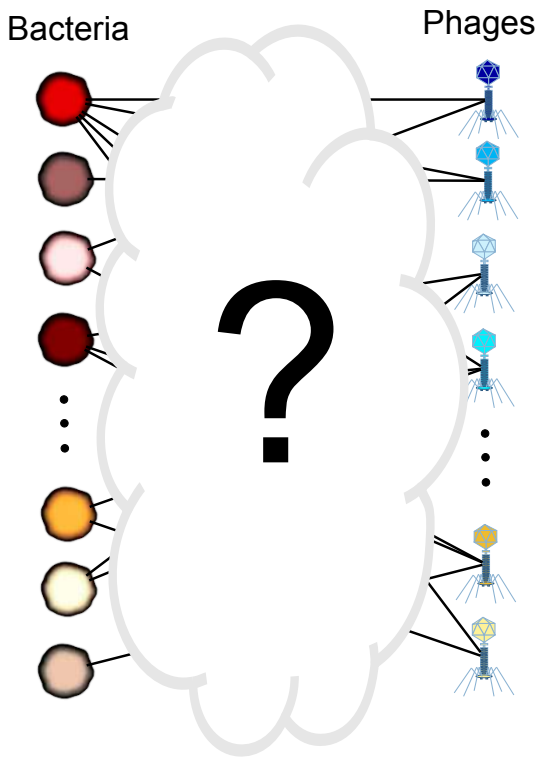


Part 1:

How does viral infection change microbial population dynamics?

Over 80 years ago, Volterra was convinced by his son-in-law, Umberto d'Ancona, to examine the fluctuations of the Adriatic fisheries

Vito Volterra

Alfred Lotka

Fluctuations in the Abundance of a Species considered Mathematically.[1]

By Prof. VITO VOLTERRA, For. Mem. R.S., President of the R. Accademia dei Lincei.

The first case I have considered is that of two associated species, of which one, finding sufficient food in its environment, would multiply indefinitely when left to itself, while the other would perish for lack of nourishment if left alone; but the second feeds upon the first, and so the two species can co-exist together.

NATURE [OCTOBER 16, 1926

In turn, Volterra & Lotka proposed a coupled pair of ODEs to describe predator-prey dynamics

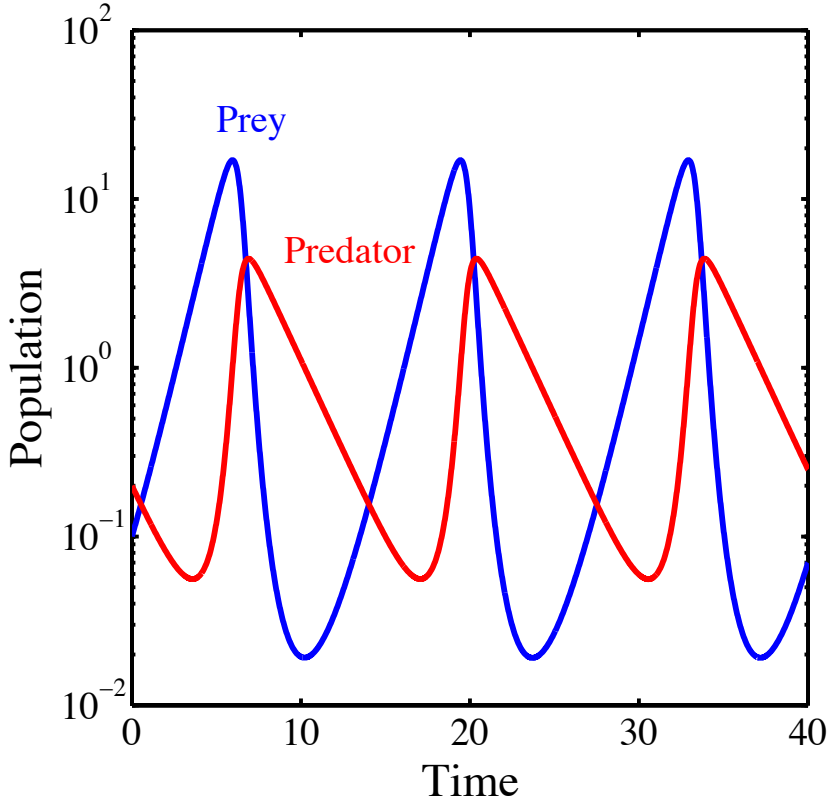
Model

$$\dot{N} = aN - bNP$$

$$\dot{P} = cNP - dP$$

N: prey abundance
P: predator abundance

Interactions:
Prey birth/death
Predation
Predator death



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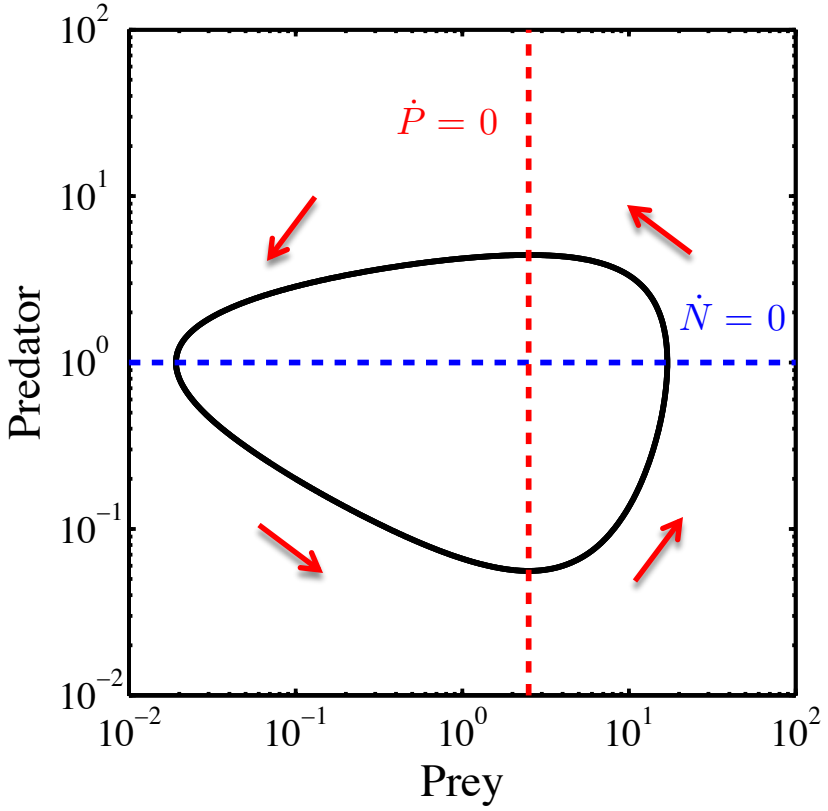

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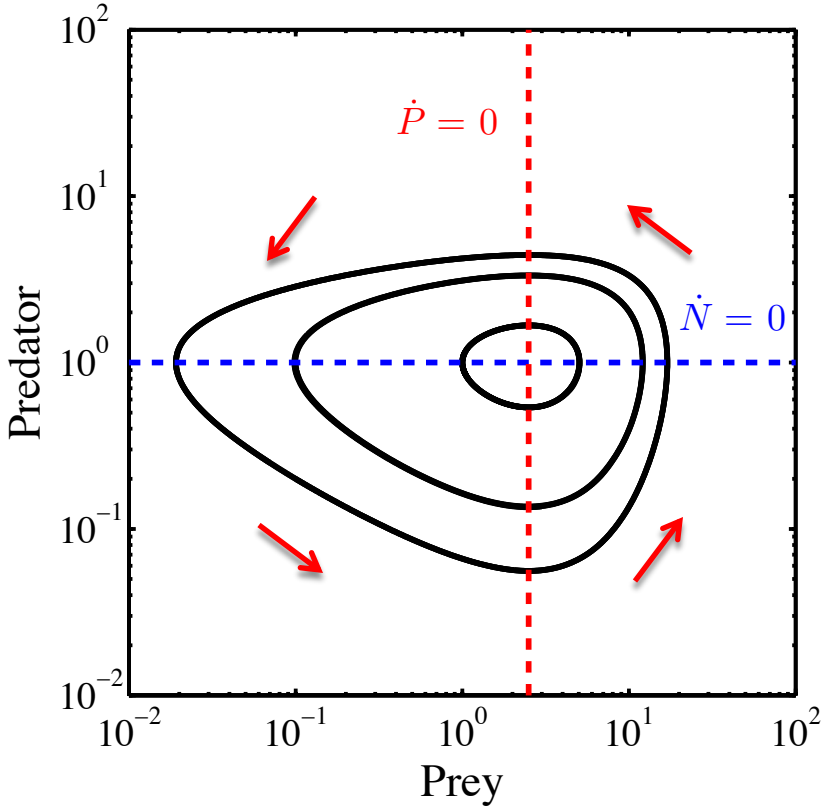
Predator death

Conservative system (not true limit cycles)

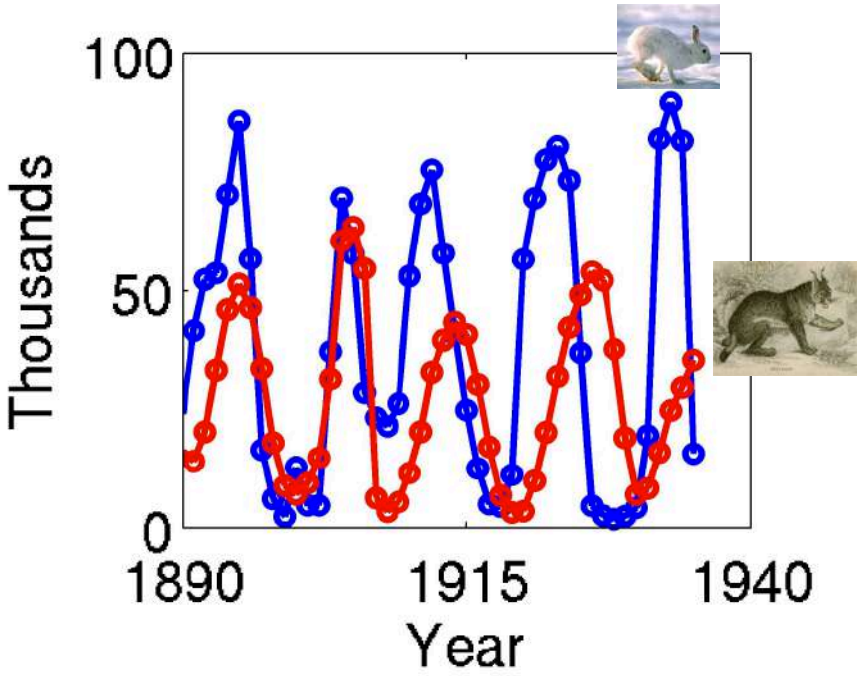
$$a \log P - bP + d \log N - cN = \text{const}$$



Later models included limit cycles, consistent with long-term observations

Features of limit cycles in predator-prey models

1. Prey peaks before predator peaks.
2. Predator oscillations are quarter-phase lagged behind prey oscillations
3. Hence, oscillations appear counter-clockwise in prey-predator phase plane



[Models include "handling time" of prey by predators and limited prey growth]

Levin et al, 1977

Vol. 111, No. 977 The American Naturalist January–February 1977

RESOURCE-LIMITED GROWTH, COMPETITION, AND PREDATION: A MODEL AND EXPERIMENTAL STUDIES WITH BACTERIA AND BACTERIOPHAGE

Bruce R. Levin, Frank M. Stewart, and Lin Chao

Zoology Department, University of Massachusetts, Amherst, Massachusetts 01002; and Mathematics Department, Brown University, Providence, Rhode Island 02912

"One Resource, One Prey, One Predator"

3. *One Resource, One Prey, One Predator*

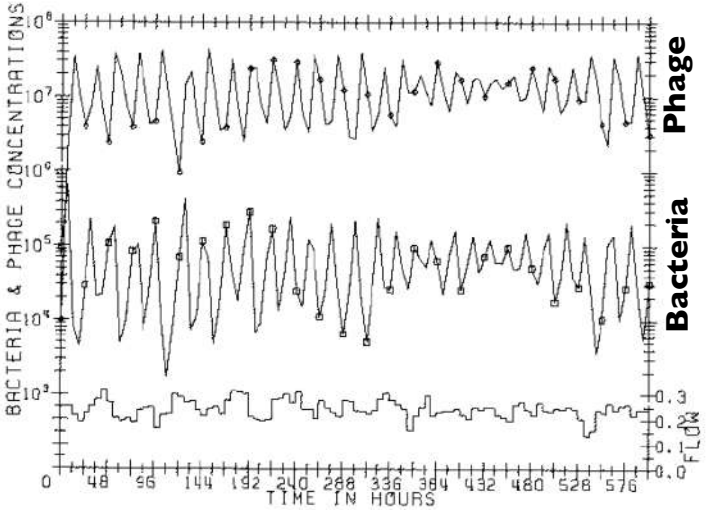
With one population at each trophic level the equilibrium conditions are:

$$\hat{r} + (\hat{n} + \hat{m})\phi(\hat{r})/\rho = C, \tag{9}$$

$$\phi(\hat{r})/e - \gamma\hat{p} = \rho, \tag{10}$$

$$\hat{m} = \gamma(1 - e^{-\rho t})\hat{n}\hat{p}/\rho, \tag{11}$$

$$(be^{-\rho t} - 1)\gamma\hat{n} = \rho. \tag{12}$$



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Idea:
Phage = Predators, Bacteria = Prey

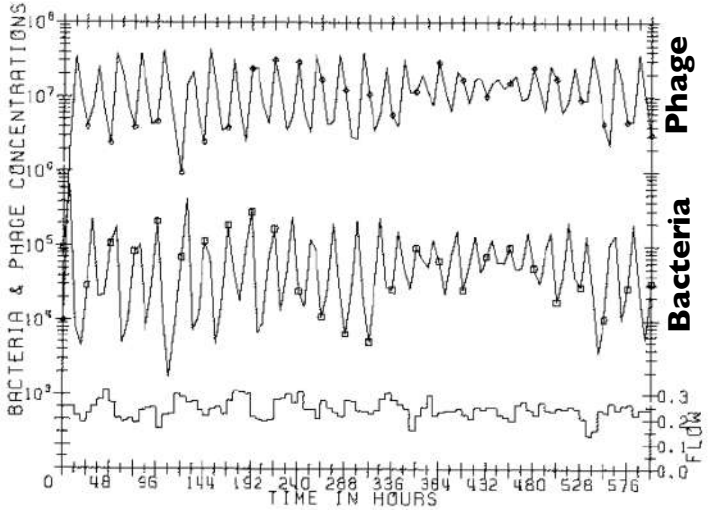
Attributed to:
Allan Campbell 1961 (Evolution):

"The simple predator. If a virulent phage and a susceptible bacterium are mixed in an open growth system, such as a chemostat…"

CONDITIONS FOR THE EXISTENCE OF BACTERIOPHAGE[1]

Allan Campbell
Department of Biology, University of Rochester

Received June 21, 1960



The Lotka-Volterra model is the basis for models of **viral-host** population dynamics

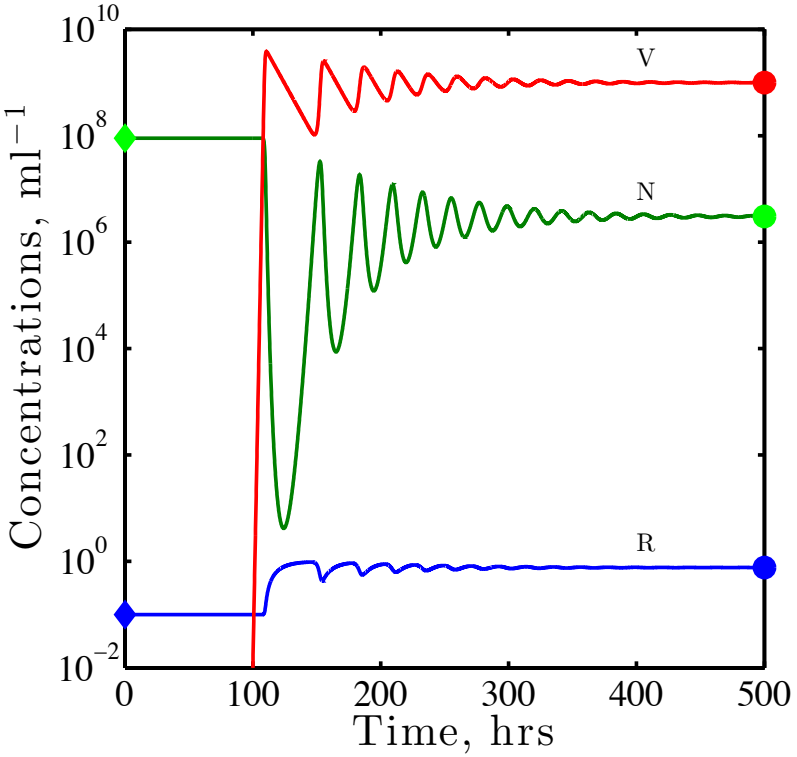
Dynamic model

$$\frac{dR}{dt} = -\omega(R - R_0) - \gamma RN$$
$$\frac{dN}{dt} = \epsilon\gamma RN - \phi NV - \omega N$$
$$\frac{dV}{dt} = \beta\phi NV - \phi NV - \omega V$$

Interactions:

Resource inflow/outflow
Host growth and outflow
Viral lysis and outflow

(note: original model included time delays)



Similar model proposed by Campbell (1961) Evolution 15: 153
& adapted to phage-bacteria chemostats by Levin et al. (1977) Am. Nat. 111:3

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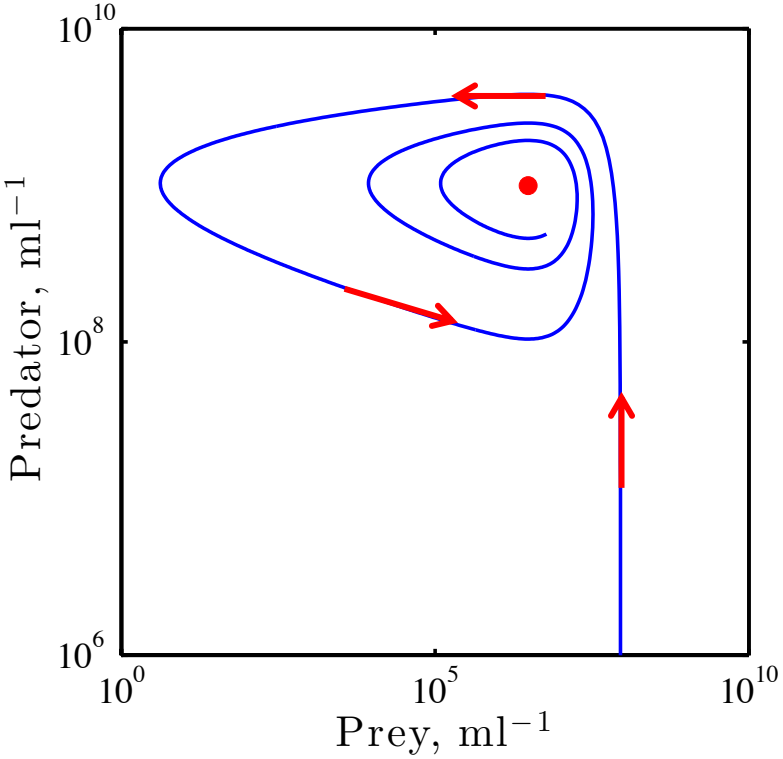

Again, counter-clockwise cycles

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The life of a bacterial virus (phage)

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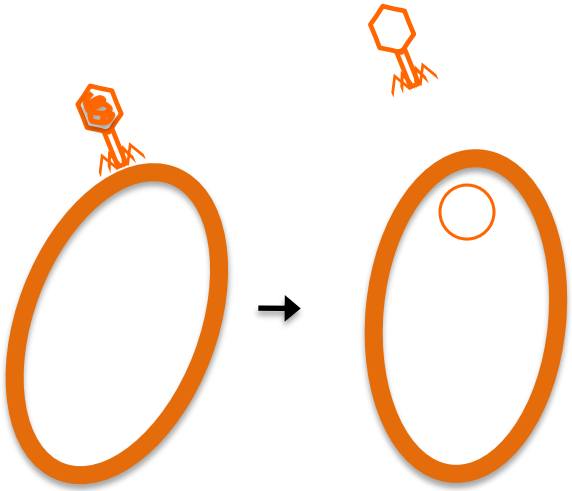

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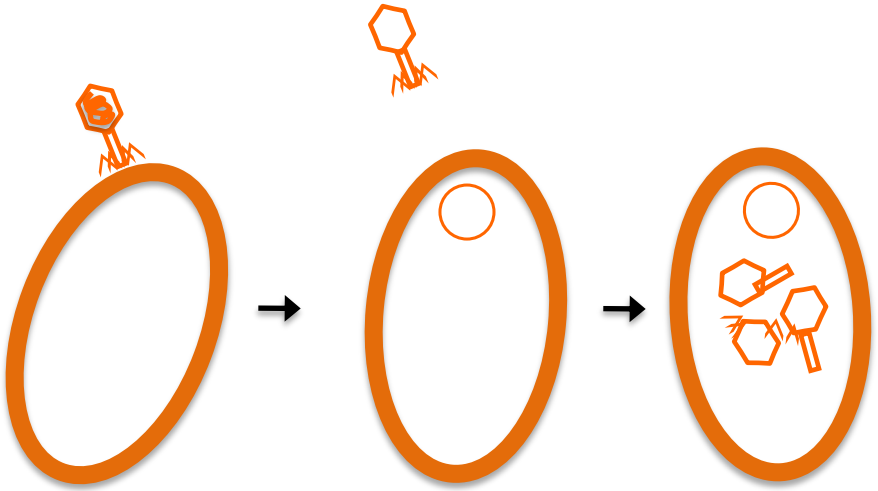

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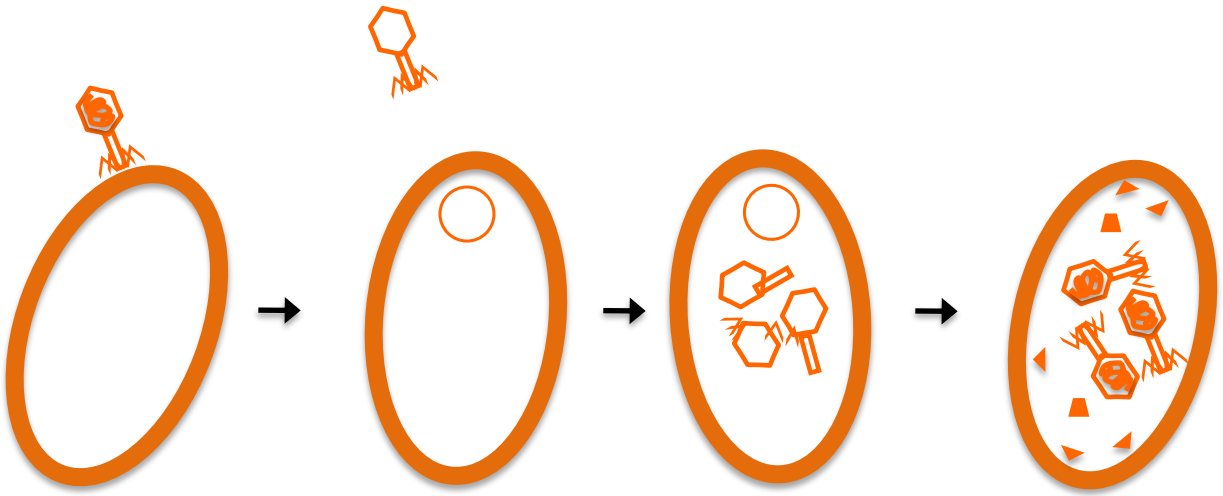

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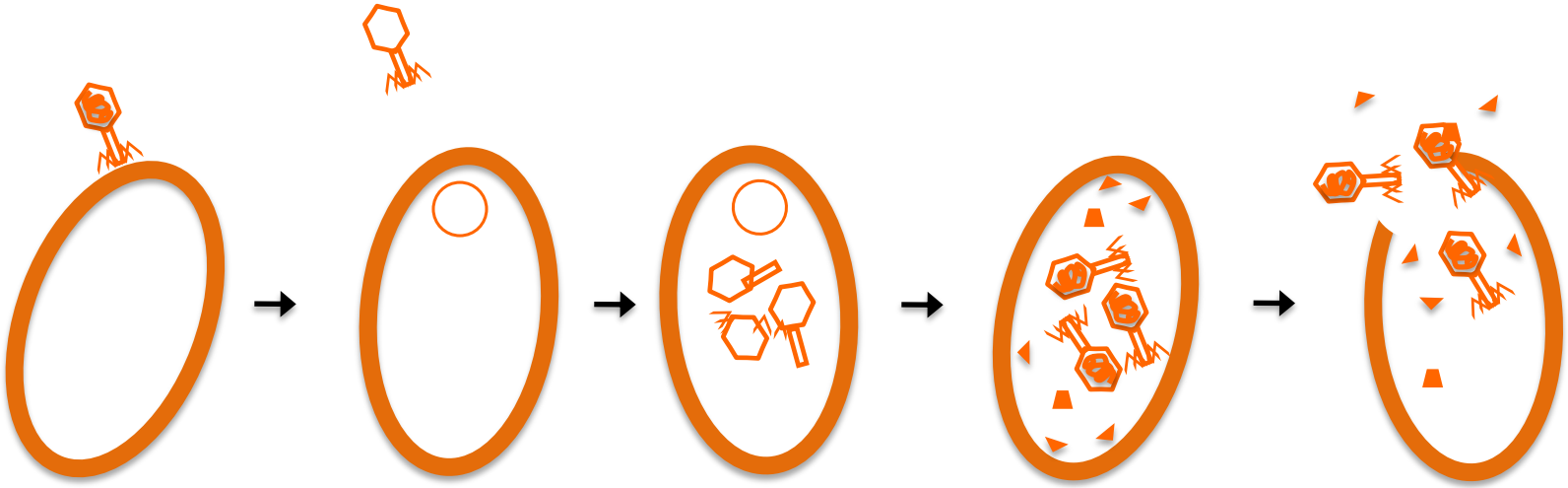

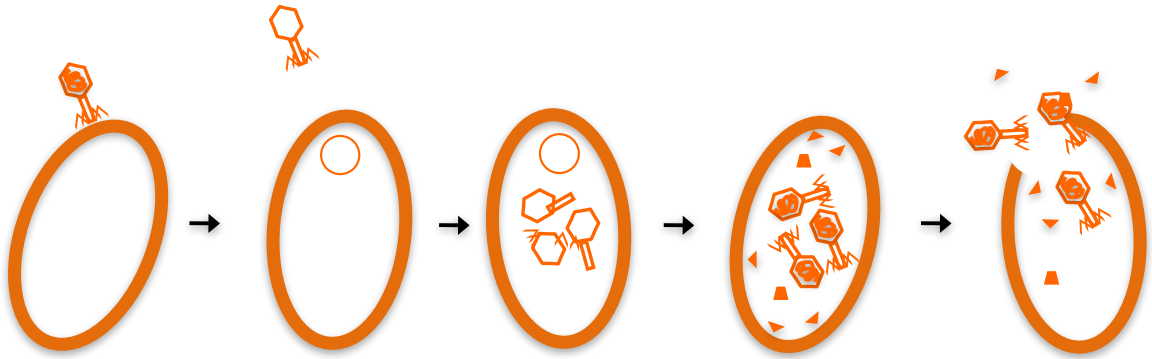

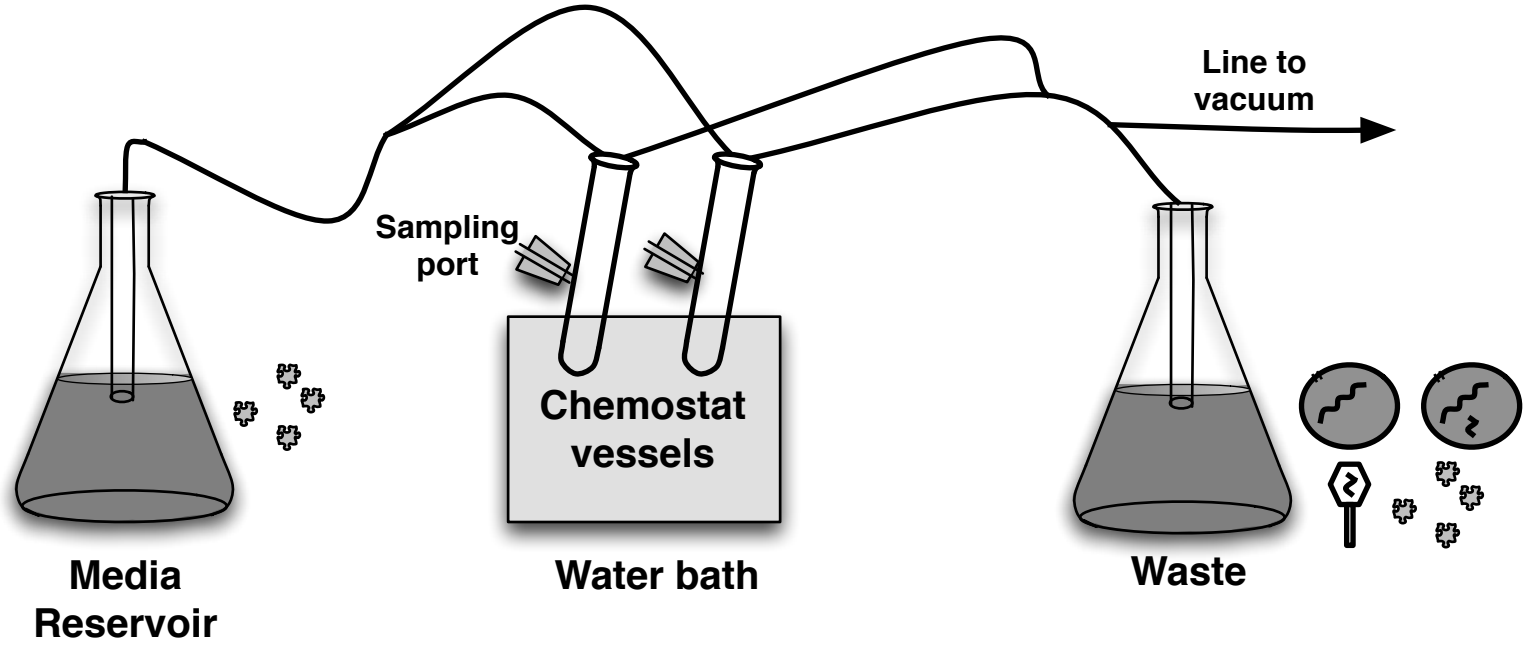
Line to
vacuum
Sampling
port
Chemostat
vessels
Media
Reservoir
Water bath
Waste

Counter-clockwise cycles have appeared robust given alternative viral-host models

1. Models with an infected class

$$\frac{dN}{dt} = rN\left(1 - \frac{N+I}{K}\right) - \phi NV - \omega N$$

$$\frac{dI}{dt} = \phi NV - \boxed{\eta I} - \omega I$$

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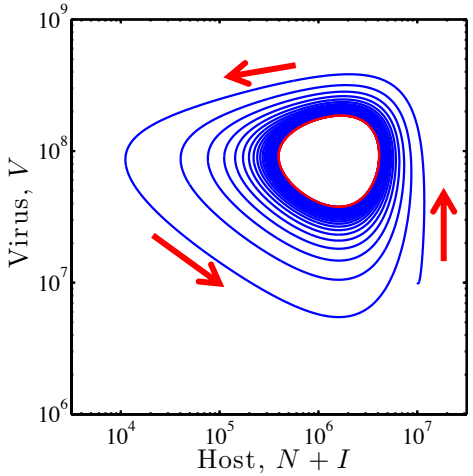

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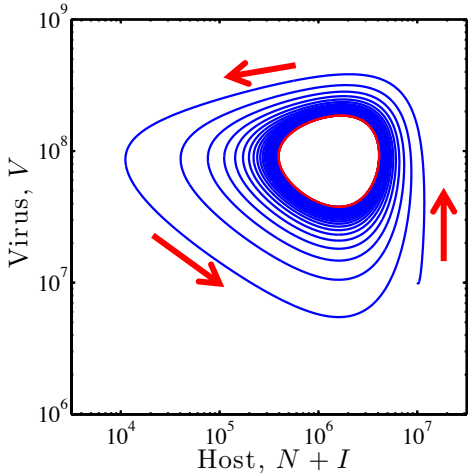

2. Models with an explicit delay between infection and lysis

$$\frac{dN}{dt} = rN\left(1 - \frac{N+I}{K}\right) - \phi NV - \omega N$$

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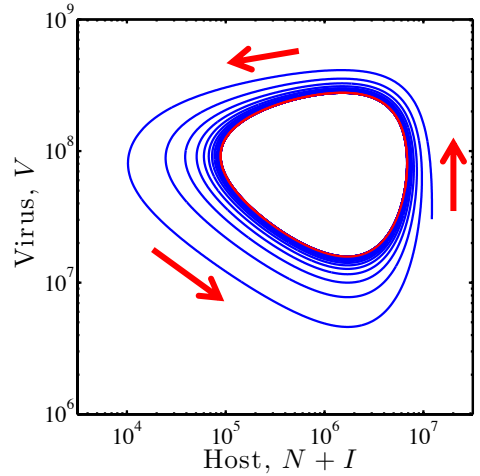

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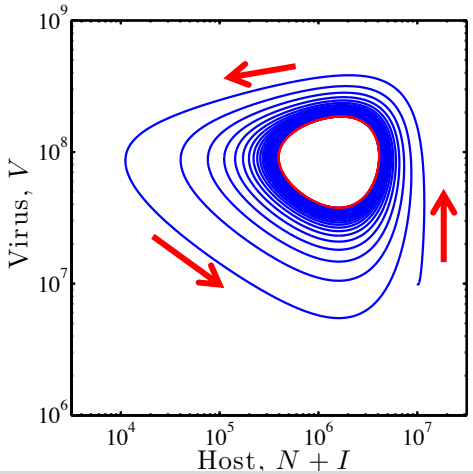


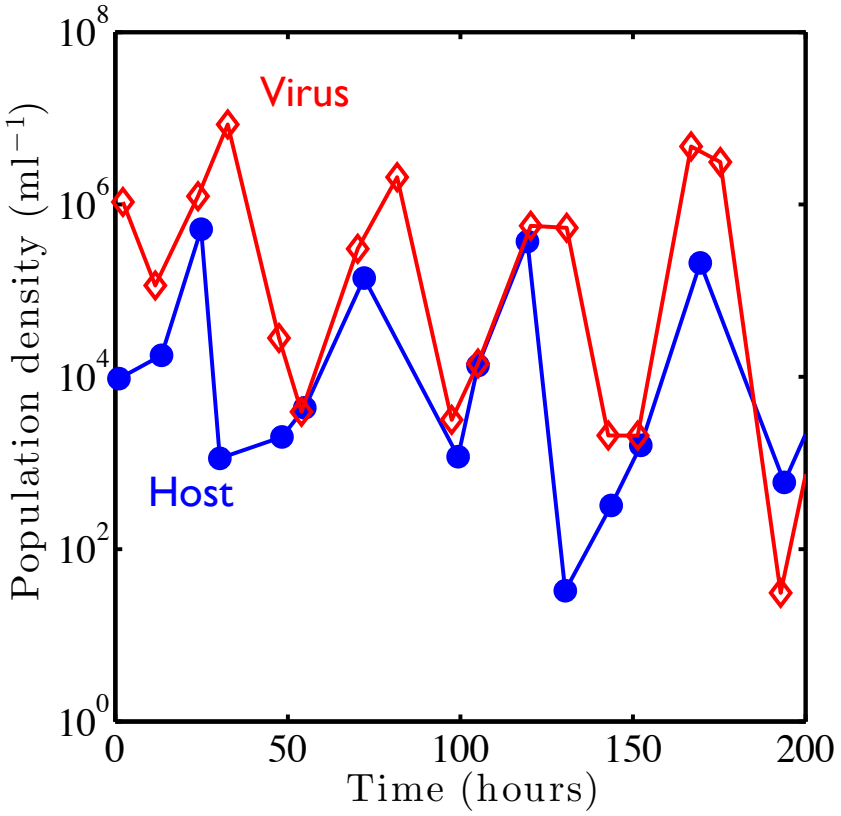
Mathematical Check-Point:

What life history traits enable viral invasion and persistence with their microbial hosts?

The same types of cycles can be observed in virus-host population dynamics

"Lotka-Volterra" like cycles between T4 and *E. coli* B

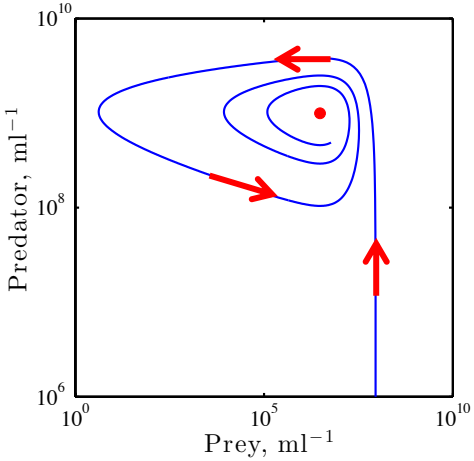
Data: Bohannan & Lenski, Ecology (1997)



Summary of Part 1

Take-home message:

Original models of viral-host dynamics presuppose a "simple" one virus, one host relationship.



In these models, viruses act like a predator, leading to cyclical dynamics in which viral peaks follow host peaks (leading to counter-clockwise cycles).

Invasion and persistence depends on both life history traits and environmental conditions - it is not inevitable.

However…

Evolution can rapidly change the number/relative abundance of viruses and hosts strains…