Ecology of parasites and infectious diseases

Predation

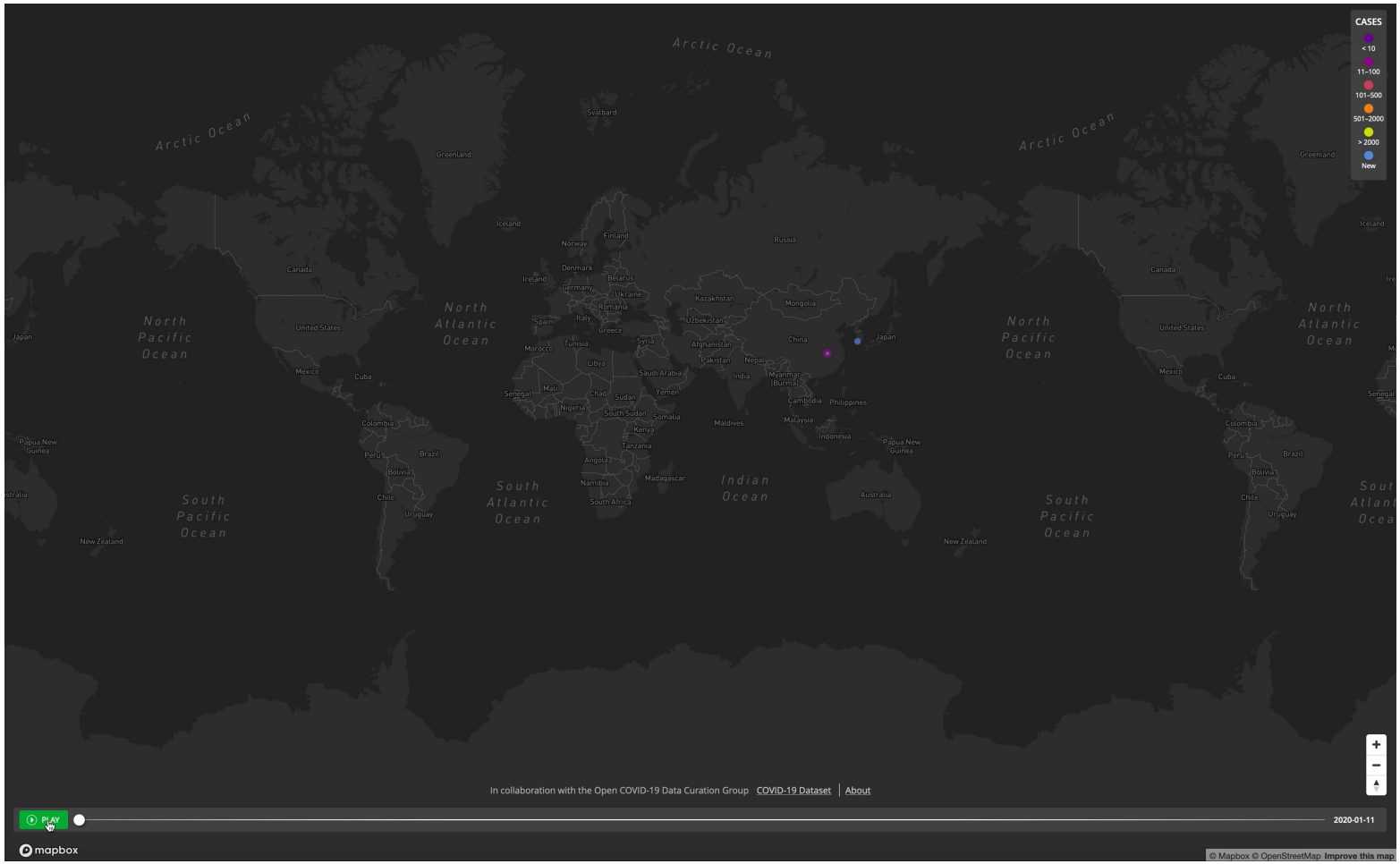
Parasitism

Competition

Mutualism

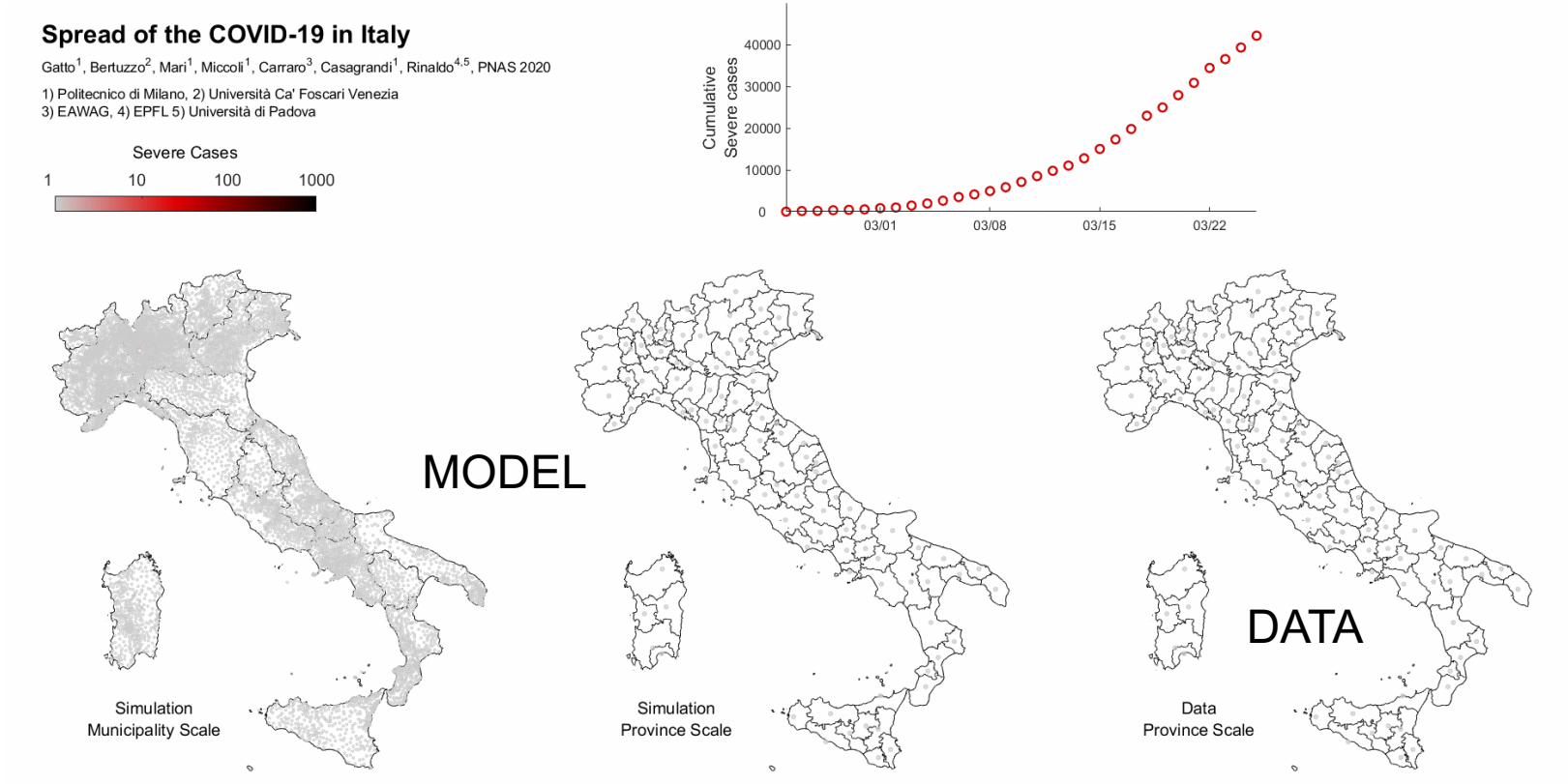
The spatial signature of COVID-19

in the world (Jan. 1st, 2020 / June 3, 2020)



The spatial signature of COVID-19

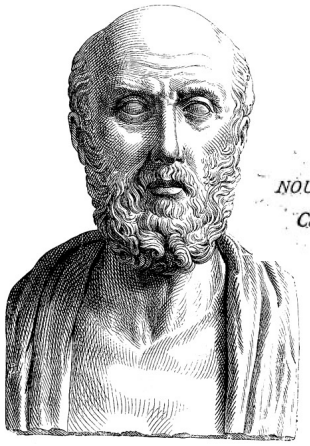
in Italy (Febr.25, 2020 / March 25, 2020)



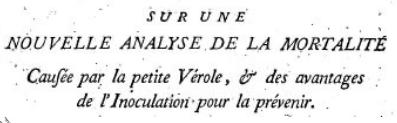
Marino Gatto et al. PNAS doi:10.1073/pnas.2004978117

PNAS

Ecology of human infectious diseases

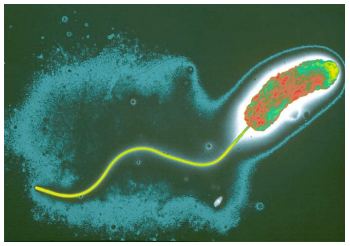


Hippocrates



Daniel Bernoulli

Filippo Pacini



Vibrio cholerae Pacini, 1854

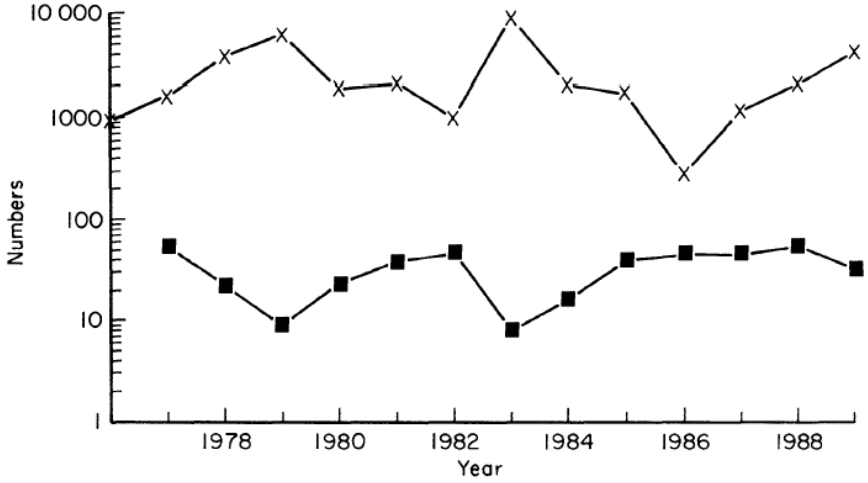
Giovanni Battista Grassi

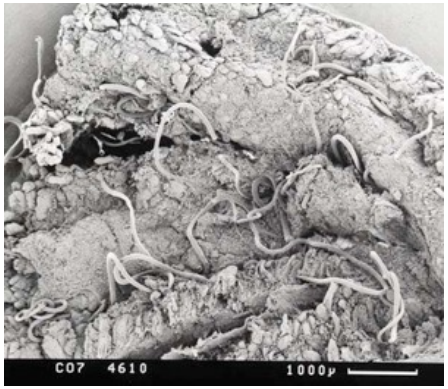
Ronald Ross

Anopheles stephensi
Malaria vector

Parasites and demographic regulation

Red grouse
(*Lagopus lagopus scoticus*)

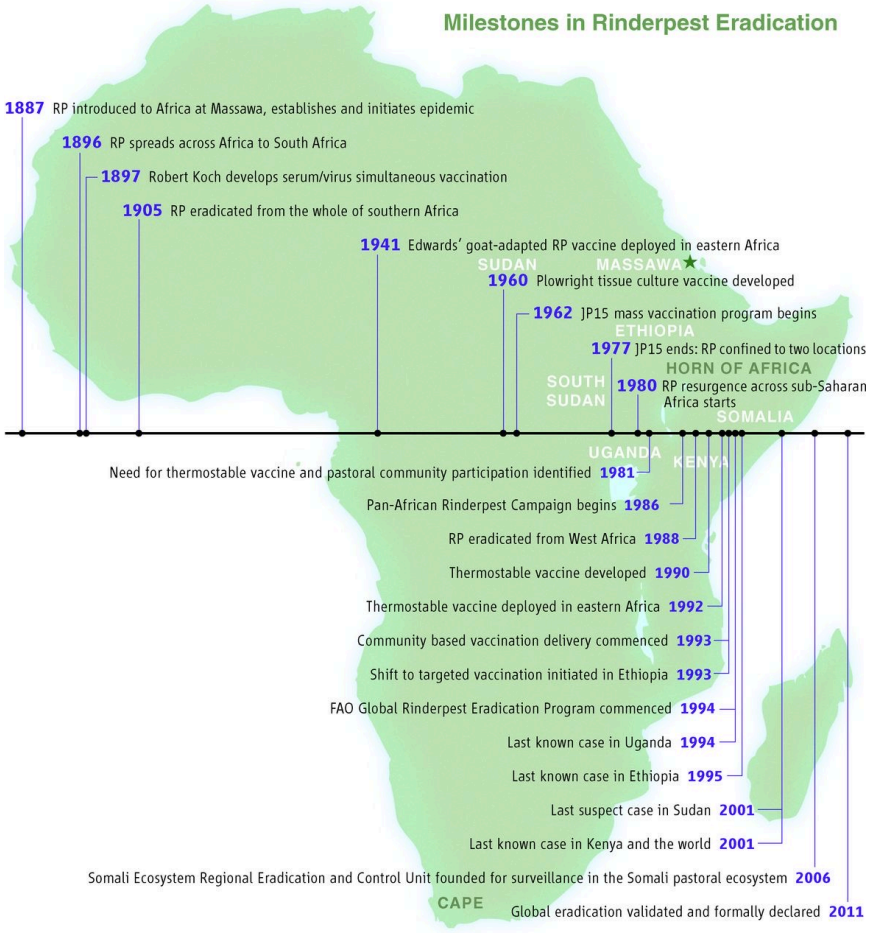




Trichostrongylus tenuis

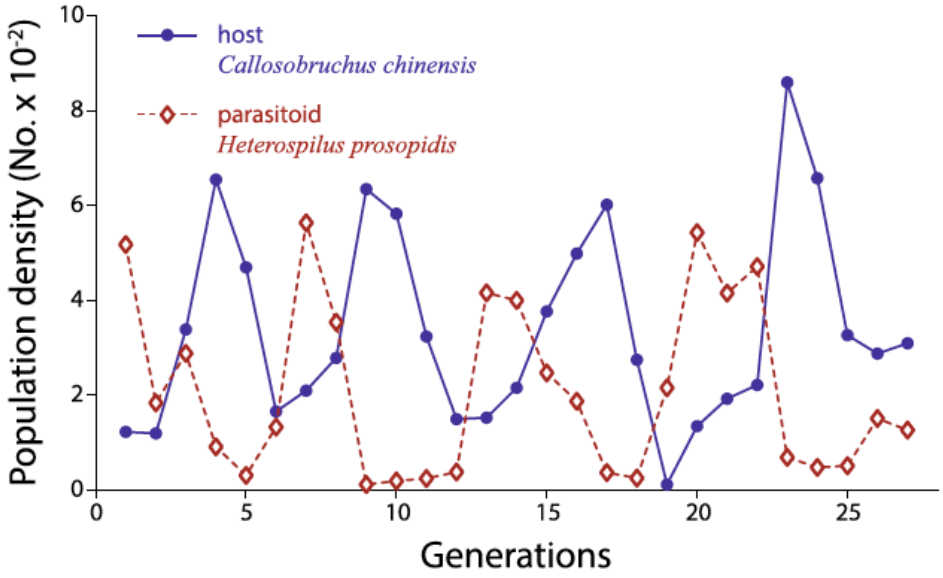
Fig. 8. Changes in numbers of grouse (hens breeding per km^2) (■) and mean parasite burden per grouse (×) over 14 years at Gunnerside. The methods used to collect the original data are described in Hudson, Newborn & Dobson (1992).

The *rinderpest* pandemic in western Africa



J C Mariner et al. Science 2012;337:1309-1312

Parasitoids



Pest beetle
Callosobruchus chinensis
Bean weevil

Parasitoid wasp
Heterospilus prosopidis

Parasitoids and biological control

Drosophila suzukii

- Invasive fruit fly
- Infests undamaged soft shelled fruit (cherry, berries etc) (Poyet et al., 2015)
- Large harvest losses in new habitat, including Trentino (De Ros et al., 2013; Wiman et al., 2014)

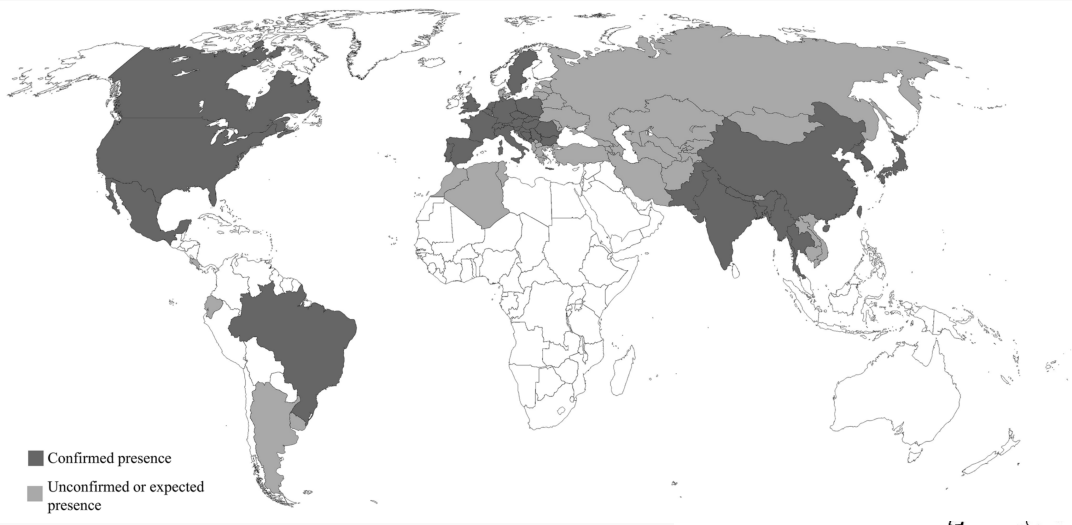
Serrated ovipositor

D. suzukii male

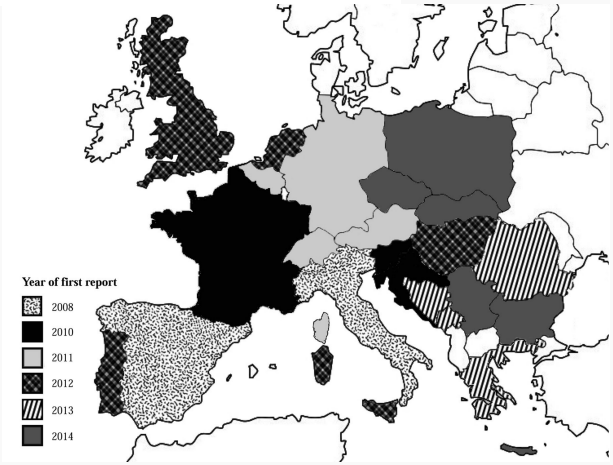
Damaged fruit

pictures: G. Arakelian/Dept. of Agriculture, Los Angeles County; Martin Hauser, California Department of Food and Agriculture; http://www.falw.vu.nl

Area of origin and invasion



- *Drosophila suzukii* native to east asia (Kanzawa, 1935)
- Arrived in 2008/2009 to Europe and America (Lee et al., 2011)
- Wide ecological range, rapid expansion (Rota-Stabelli et al., 2013)



maps: (Asplen et al., 2015)

Control

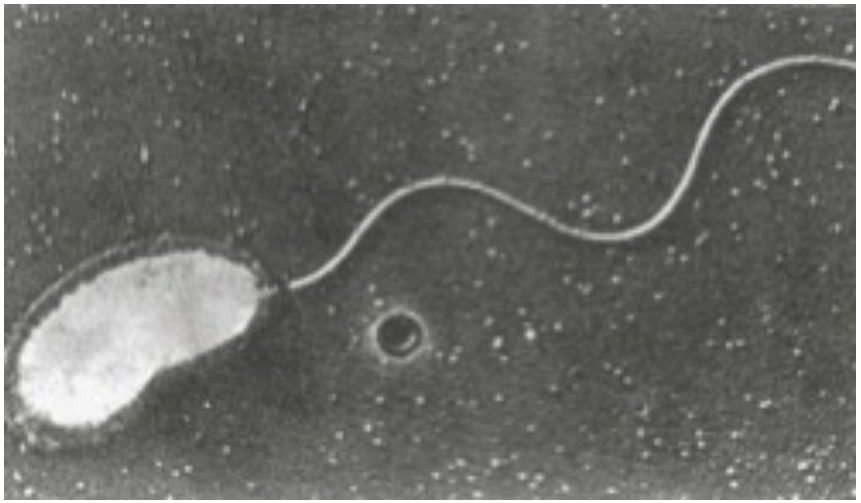
- Insecticides problematic (Cini et al., 2012)
- Possibly biological control with resident or introduced parasitoid (Rossi Stacconi et al., 2015; Daane et al., 2016)

Trichopria drosophilae (pupal parasitoid)

Leptopilina heterotoma (larval parasitoid)

pictures: http://www.bioplanet.eu; (Lue et al., 2016)

Micro and macroparasites



An example of microparasite, the bacterium *Vibrio cholerae*, the pathogenic agent of cholera (Pacini, 1854).

Short life time compared to host's
Dynamics can be neglected

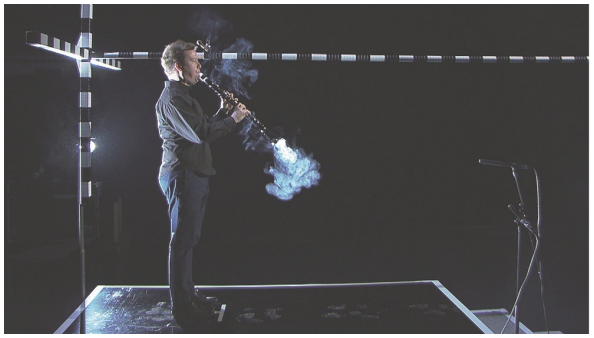
An example of macroparasite, the cestode worm *Taenia pisiformis*, dog tapeworm.

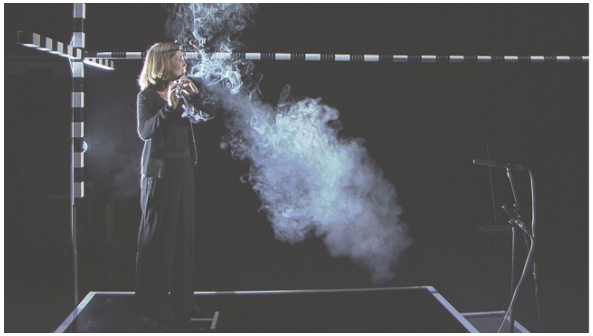
Life time comparable to host's
Dynamics cannot be neglected

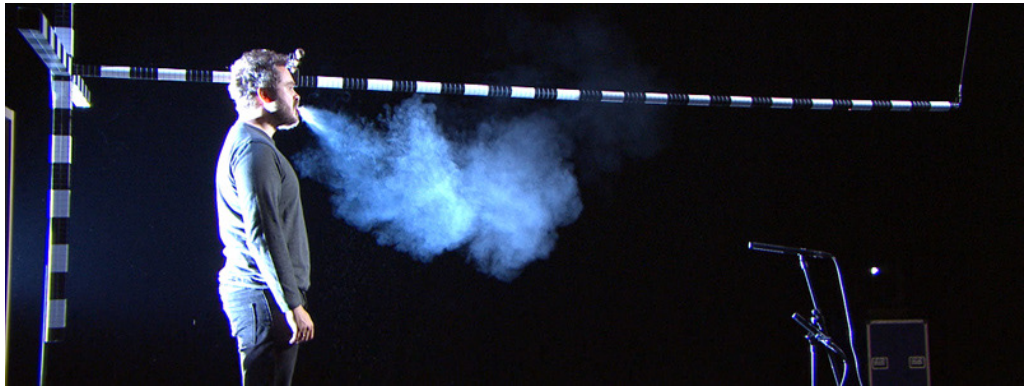
Transmission pathways of microparasitic diseases

Transmission mode	Description	Examples
Direct (airborne, sexual…)	Disease propagules are directly transmitted from one host to another via air or via physical contact (e.g. sexual intercourse)	Common cold, measles, syphilis, HIV/AIDS, rabies, influenza, SARS, COVID-19
Vector-borne	Propagules are transmitted from one host to another via a second host species, the vector (e.g. mosquito)	Malaria (anopheles mosquito), dengue (tiger mosquito), zika (Yellow Fever mosquito, *Aedes aegypti*)
Water-borne	Propagules are transmitted via contaminated water	Cholera, rotavirus
Environmental	Propagules are shed into the environment where they remain until another host acquires them. These pathogens differ from those responsible for direct transmission because of their longer permanence time in the external environment	Smallpox, anthrax, tetanus, Legionella
Vertical	Propagules are transmitted from mother to progeny via the milk or the body fluids	HIV/AIDS, hepatitis B and C

Aerosol emission



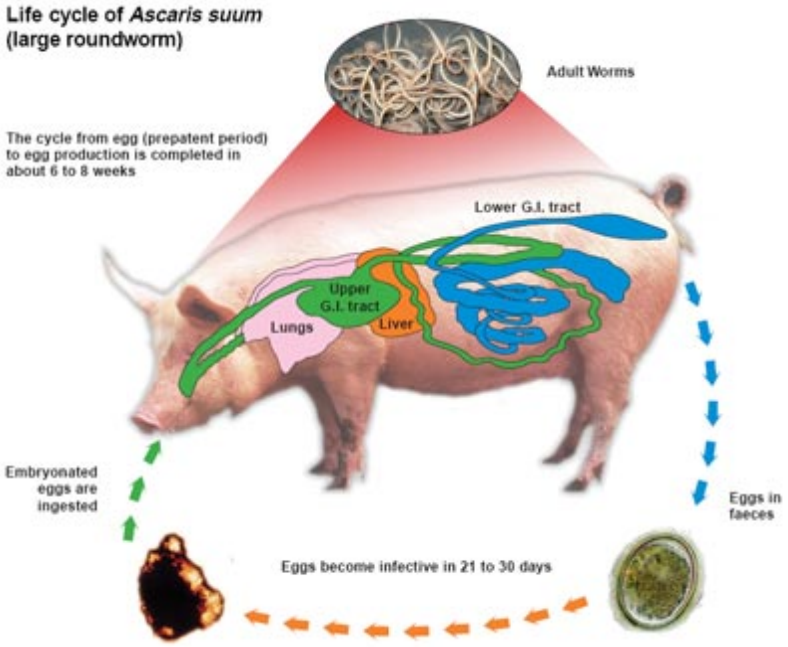




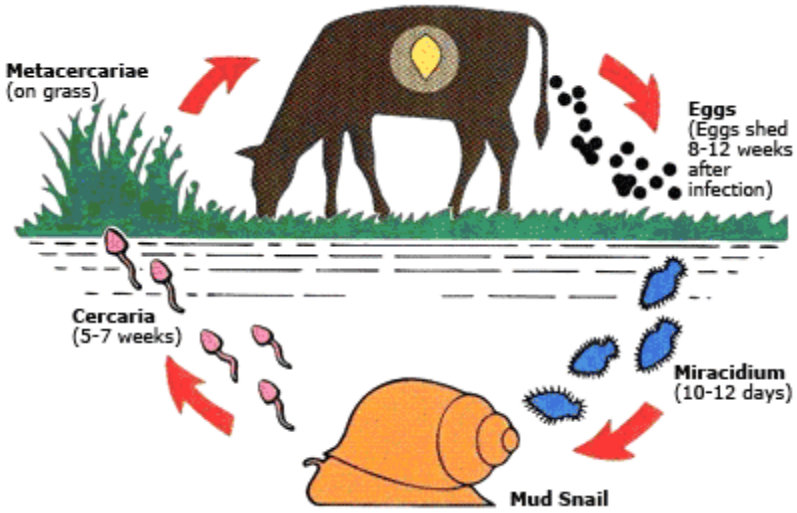
Transmission pathways of microparasitic diseases

Transmission mode	Description	Examples
Direct (airborne, sexual…)	Disease propagules are directly transmitted from one host to another via air or via physical contact (e.g. sexual intercourse)	Common cold, measles, syphilis, HIV/AIDS, rabies, influenza, SARS, COVID-19
Vector-borne	Propagules are transmitted from one host to another via a second host species, the vector (e.g. mosquito)	Malaria (anopheles mosquito), dengue (tiger mosquito), zika (Yellow Fever mosquito, *Aedes aegypti*)
Water-borne	Propagules are transmitted via contaminated water	Cholera, rotavirus
Environmental	Propagules are shed into the environment where they remain until another host acquires them. These pathogens differ from those responsible for direct transmission because of their longer permanence time in the external environment	Smallpox, anthrax, tetanus, Legionella
Vertical	Propagules are transmitted from mother to progeny via the milk or the body fluids	HIV/AIDS, hepatitis B and C

Life cycles in macroparasites.



Simple cycle of the large roundworm, a nematode



Life cycle of *Fasciolopsis buski*, a trematode, the agent responsible for fasciolopsiasis, an example of cycle with an intermediate host

Infectious diseases in history

Table 1. Emerging Infectious Diseases in History

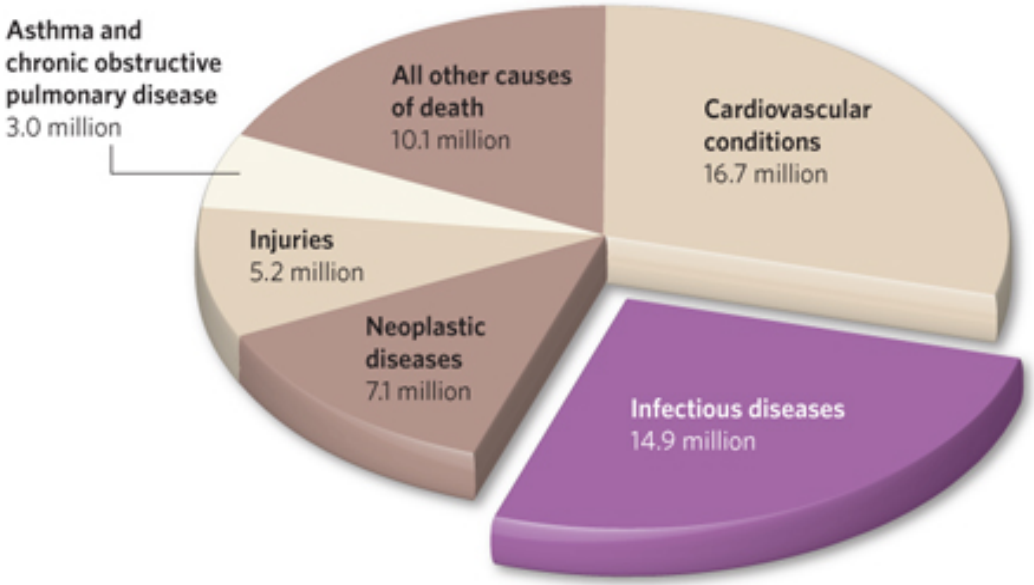
Year	Name	Deaths	Comments
430 BCE	"Plague of Athens"	~100,000	First identified trans-regional pandemic
541	Justinian plague (*Yersinia pestis*)	30–50 million	Pandemic; killed half of world population
1340s	"Black Death" (*Yersinia pestis*)	~50 million	Pandemic; killed at least a quarter of world population
1494	Syphilis (*Treponema pallidum*)	>50,000	Pandemic brought to Europe from the Americas
c. 1500	Tuberculosis	High millions	Ancient disease; became pandemic in Middle Ages
1520	*Hueyzahuatl* (*Variola major*)	3.5 million	Pandemic brought to New World by Europeans
1793–1798	"The American plague"	~25,000	Yellow fever terrorized colonial America
1832	2nd cholera pandemic (Paris)	18,402	Spread from India to Europe/Western Hemisphere
1918	"Spanish" influenza	~50 million	Led to additional pandemics in 1957, 1968, 2009
1976–2020	Ebola	15,258	First recognized in 1976; 29 regional epidemics to 2020
1981	Acute hemorrhagic conjunctivitis	rare deaths	First recognized in 1969; pandemic in 1981
1981	HIV/AIDS	~37 million	First recognized 1981; ongoing pandemic
2002	SARS	813	Near-pandemic
2009	H1N1 "swine flu"	284,000	5th influenza pandemic of century
2014	Chikungunya	uncommon	Pandemic, mosquito-borne
2015	Zika	~1,000?*	Pandemic, mosquito-borne

Selected important emerging and re-emerging infectious diseases of the past and present, 430 BCE–2020 CE. Mortality estimates are in most cases imprecise; see text.

*Zika mortality has not been fully established. Most deaths are fetal or related to outcomes of severe congenital infections.

D. M. Morens & A. S. Fauci, Cell, 182, September 3, 2020.

Main death causes in the world and the statistics of infectious diseases

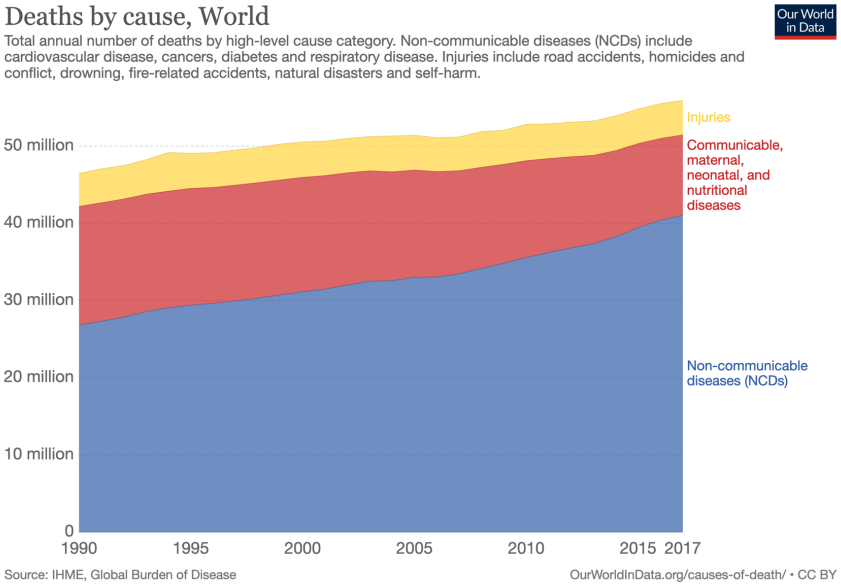


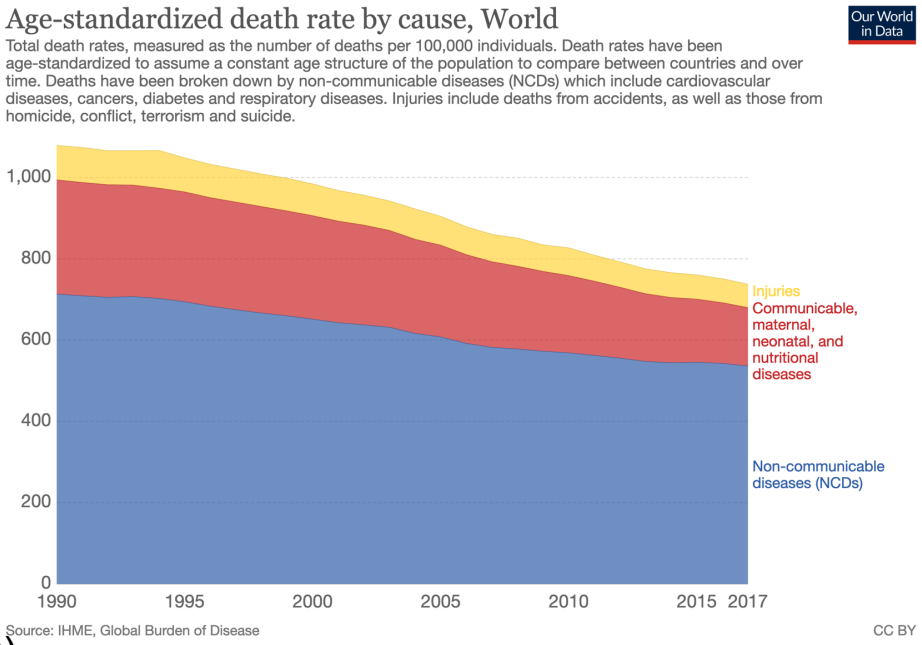
Infectious diseases	Annual deaths (millions)
Respiratory infections	3.96
HIV/AIDS	2.77
Diarrhoeal diseases	1.80
Tuberculosis	1.56
Vaccine-preventable childhood diseases	1.12
Malaria	1.27
STDs (other than HIV)	0.18
Meningitis	0.17
Hepatitis B and C	0.16
Tropical parasitic diseases	0.13
Dengue	0.02
Other infectious diseases	1.76

Morens, D., Folkers, G. & Fauci, A. The challenge of emerging and re-emerging infectious diseases. *Nature* **463**, 122 (2010). https://doi.org/10.1038/nature08554

Global deaths by cause: recent trends

In 2019 about 8 million deaths were caused by infectious diseases

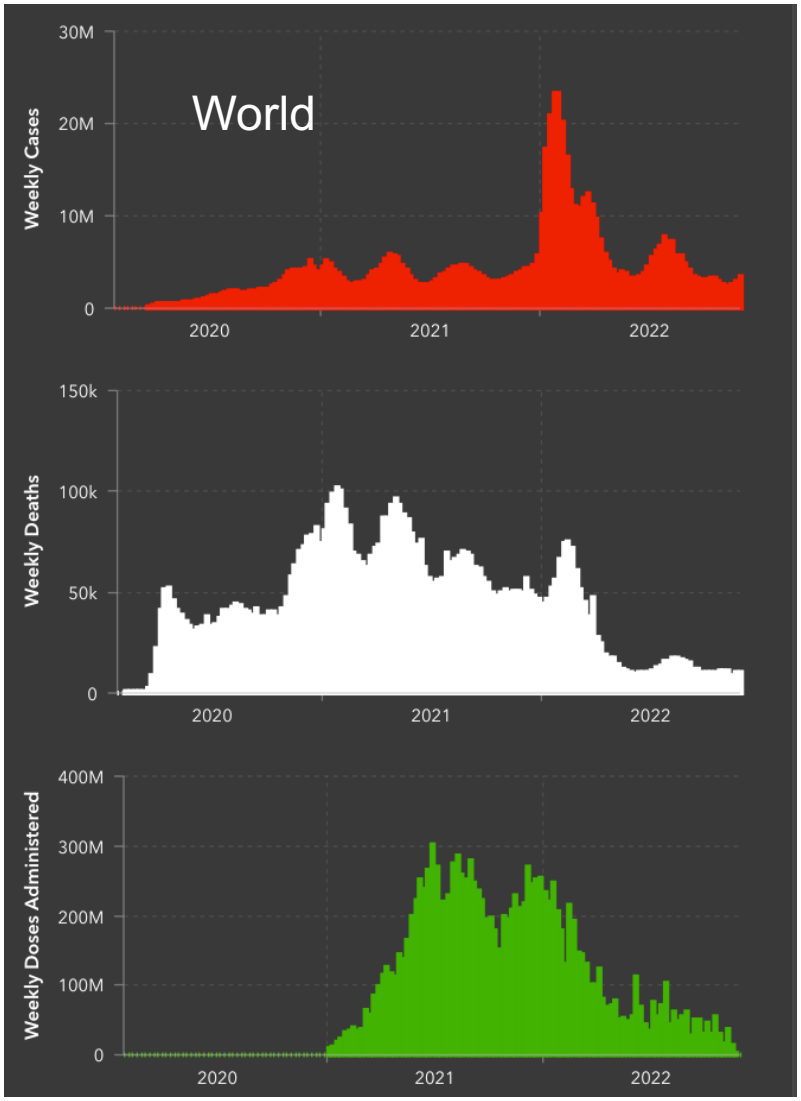


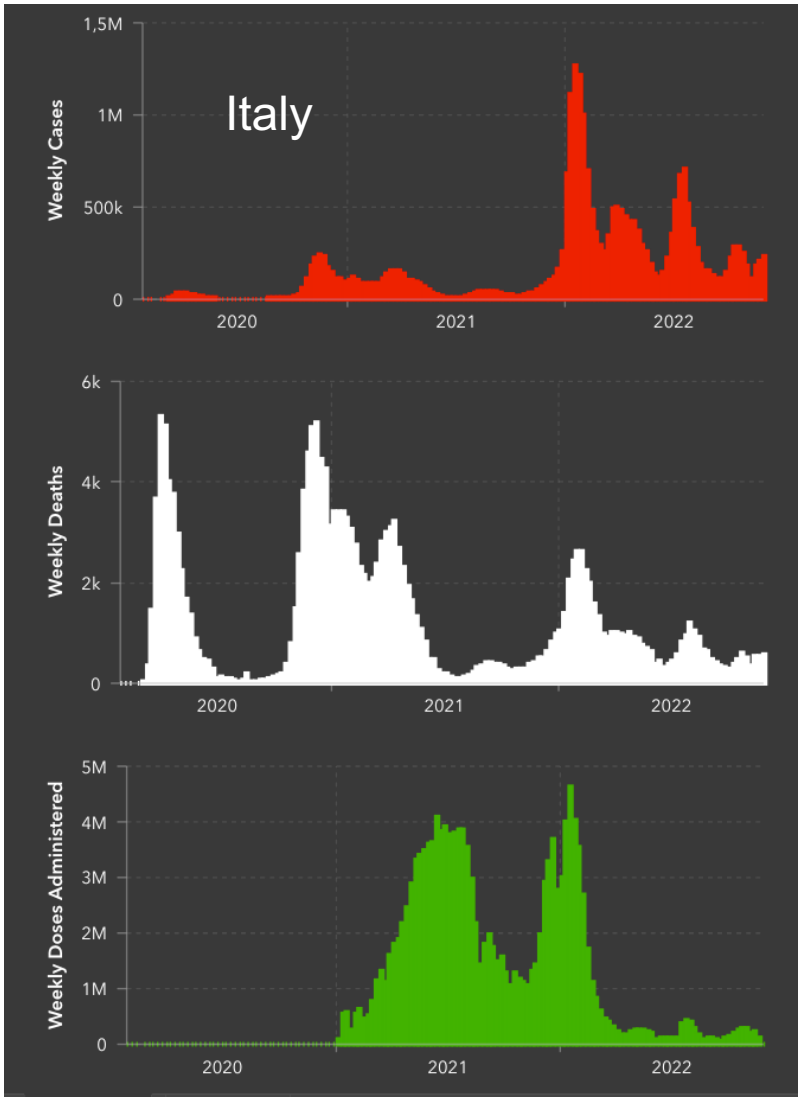


COVID-19: ~ 1.825 million deaths (in 2020)
~ 3.644 million deaths (in 2021)
> 6.631 million total deaths (as of Nov. 28, 2022)

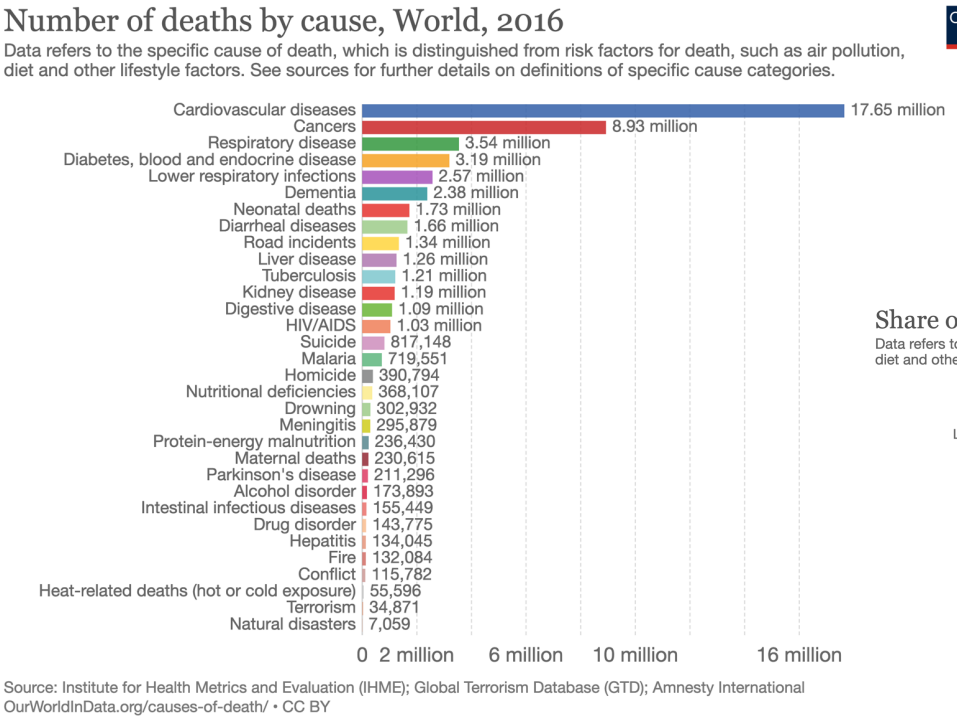
https://coronavirus.jhu.edu/map.html

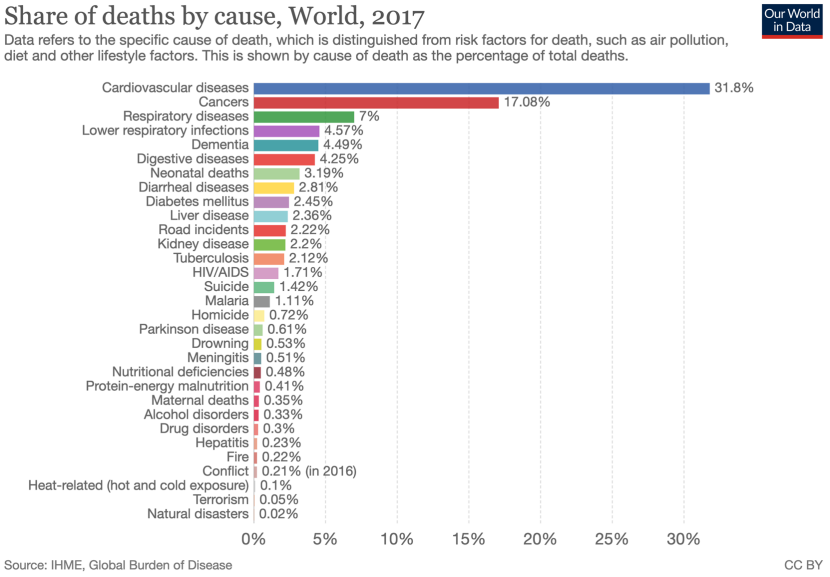
Situation as of November 28, 2022



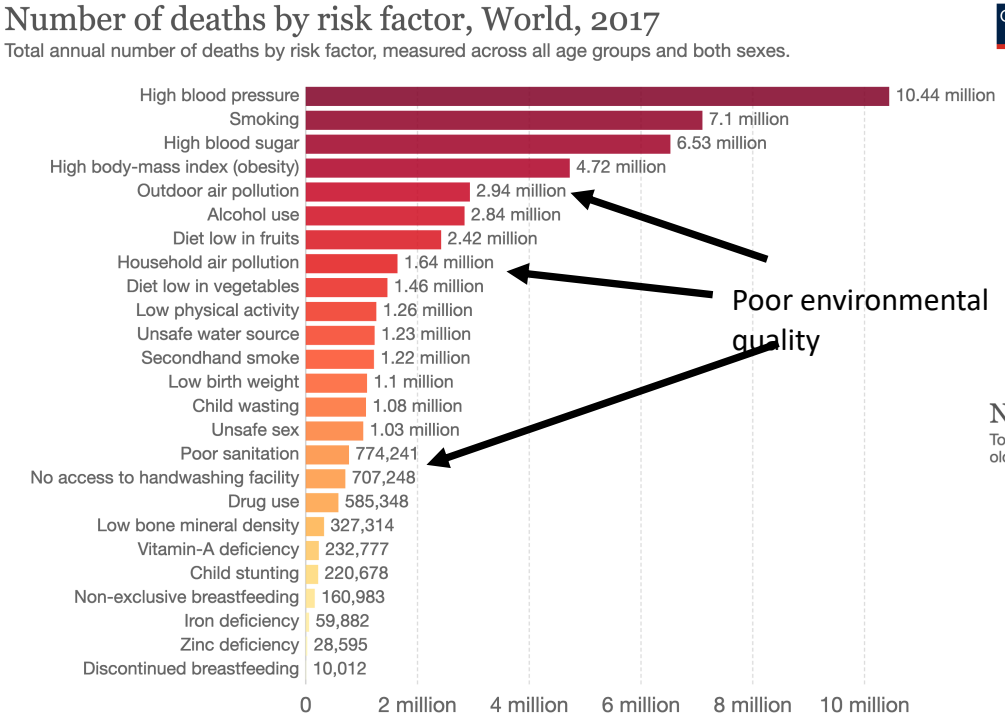


Global deaths by cause: the 2017 situation

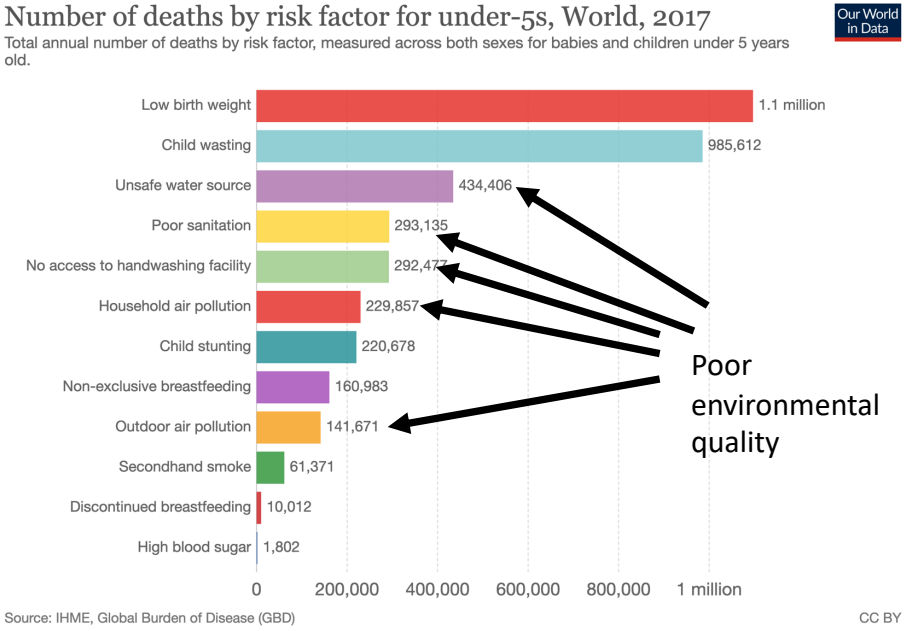




Cause vs. risk factors

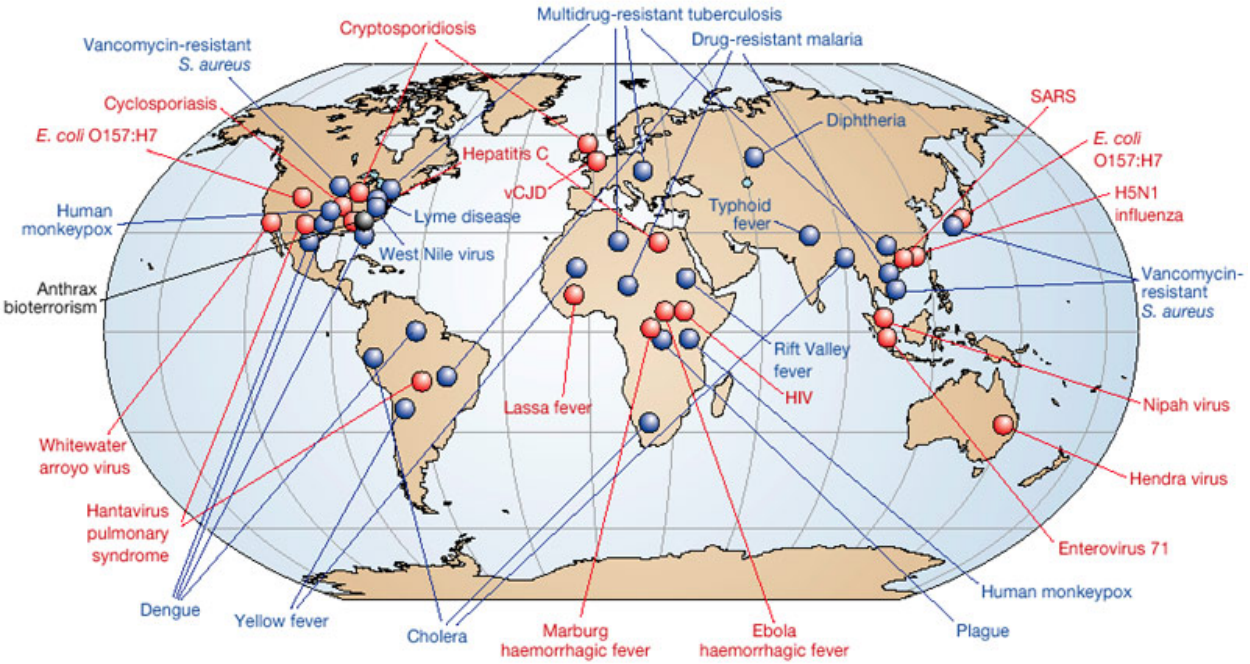


Poor environmental quality



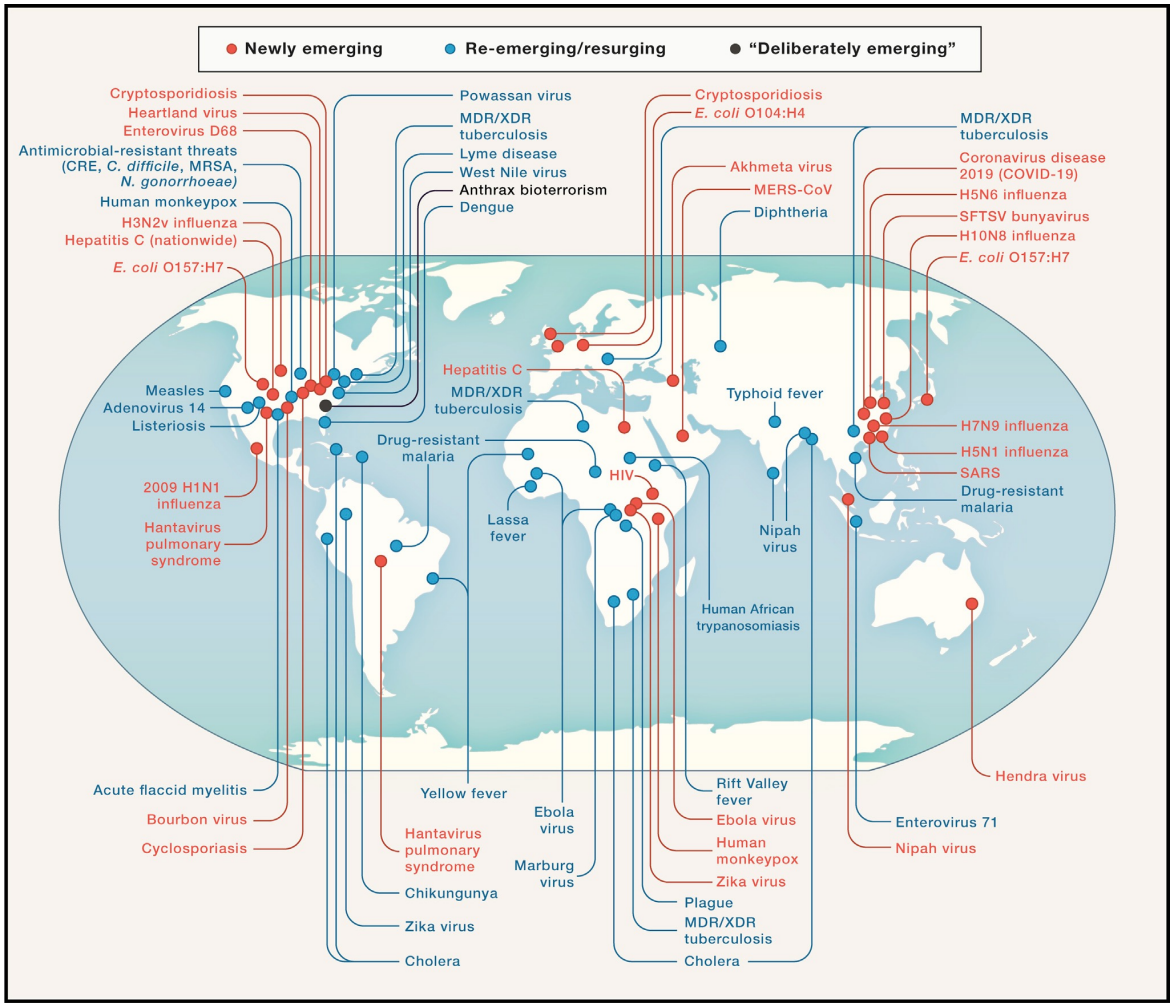
Poor environmental quality

Global map of emerging (red) e reemerging (blue) infectious diseases



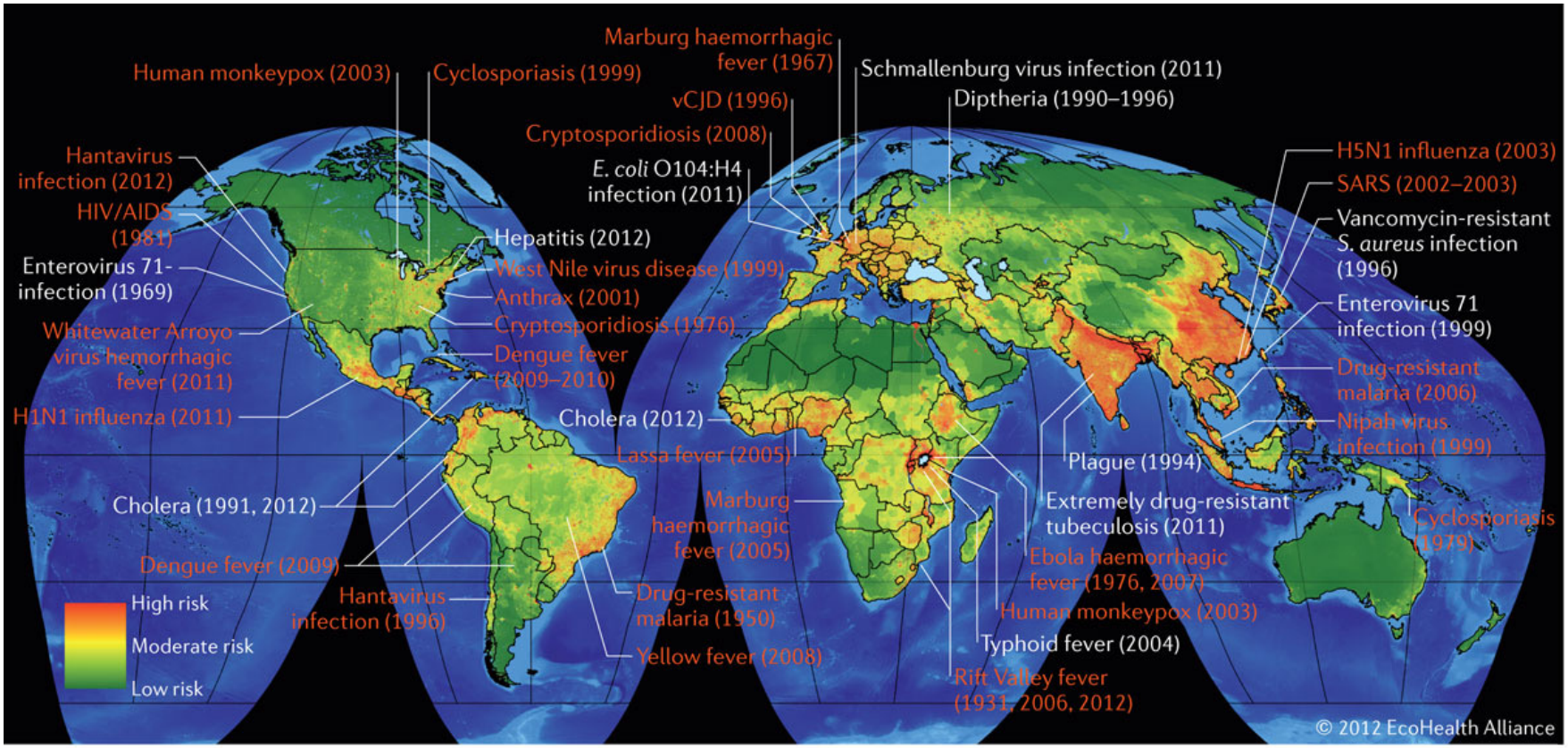
D. M. Morens, G.K. Folkers & A. S. Fauci, Nature, 2004; 430: 242-249

Global map updated to 2020



D. M. Morens & A. S. Fauci, Cell, 182, September 3, 2020.

Zoonoses (in red)

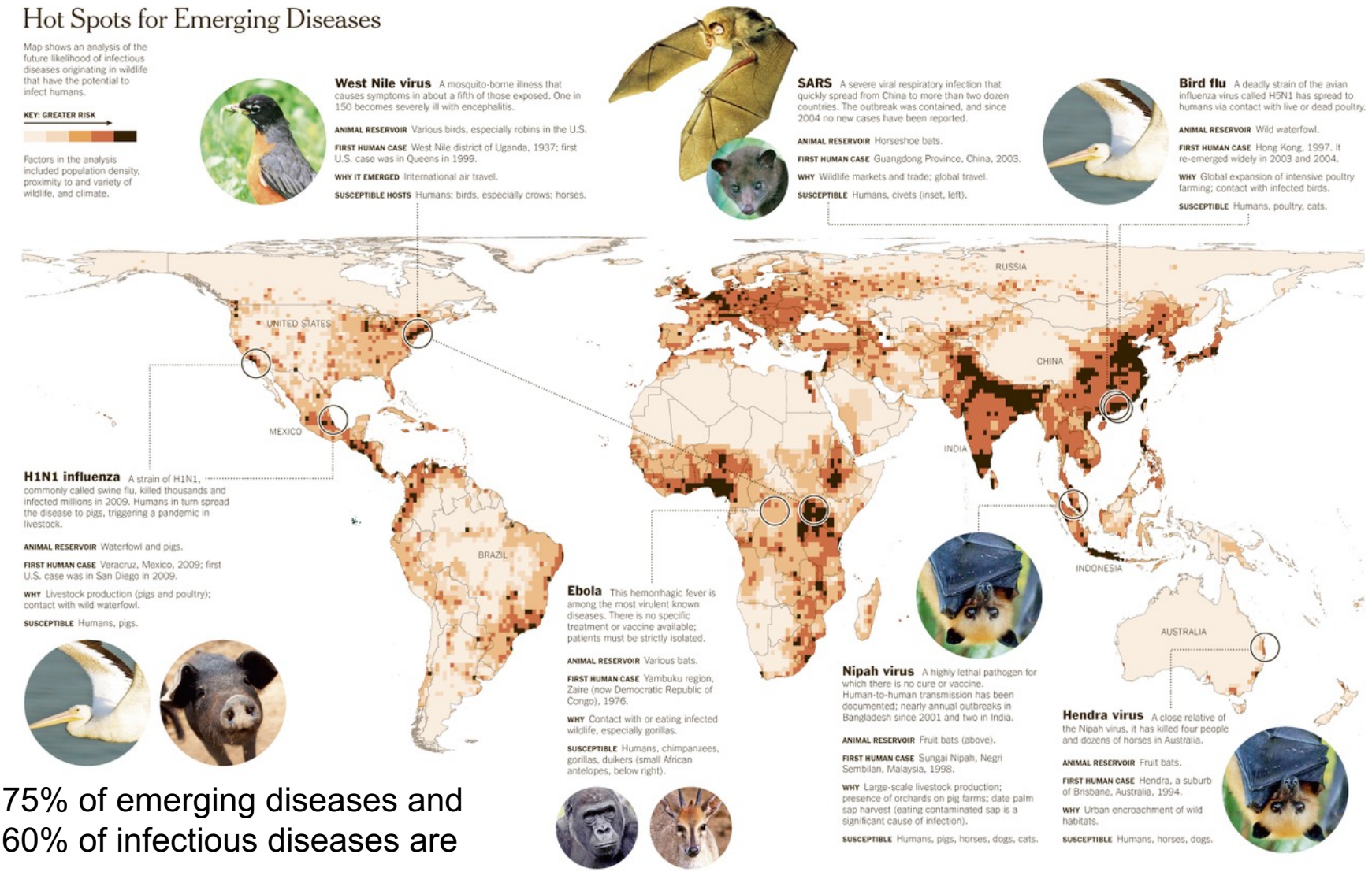


W. Ian Lipkin, Nature Reviews Microbiology 11, 133-141 (February 2013)

Nature Reviews | Microbiology

Main diseases

https://archive.nytimes.com/www.nytimes.com/imagepages/2012/07/15/opinion/15cover-grph.html



75% of emerging diseases and 60% of infectious diseases are zoonoses

Salyer SJ et al. Emerg Infect Dis. 2017;23(13). https://doi.org/10.3201/eid2313.170418

Global dynamics of emerging infectious diseases

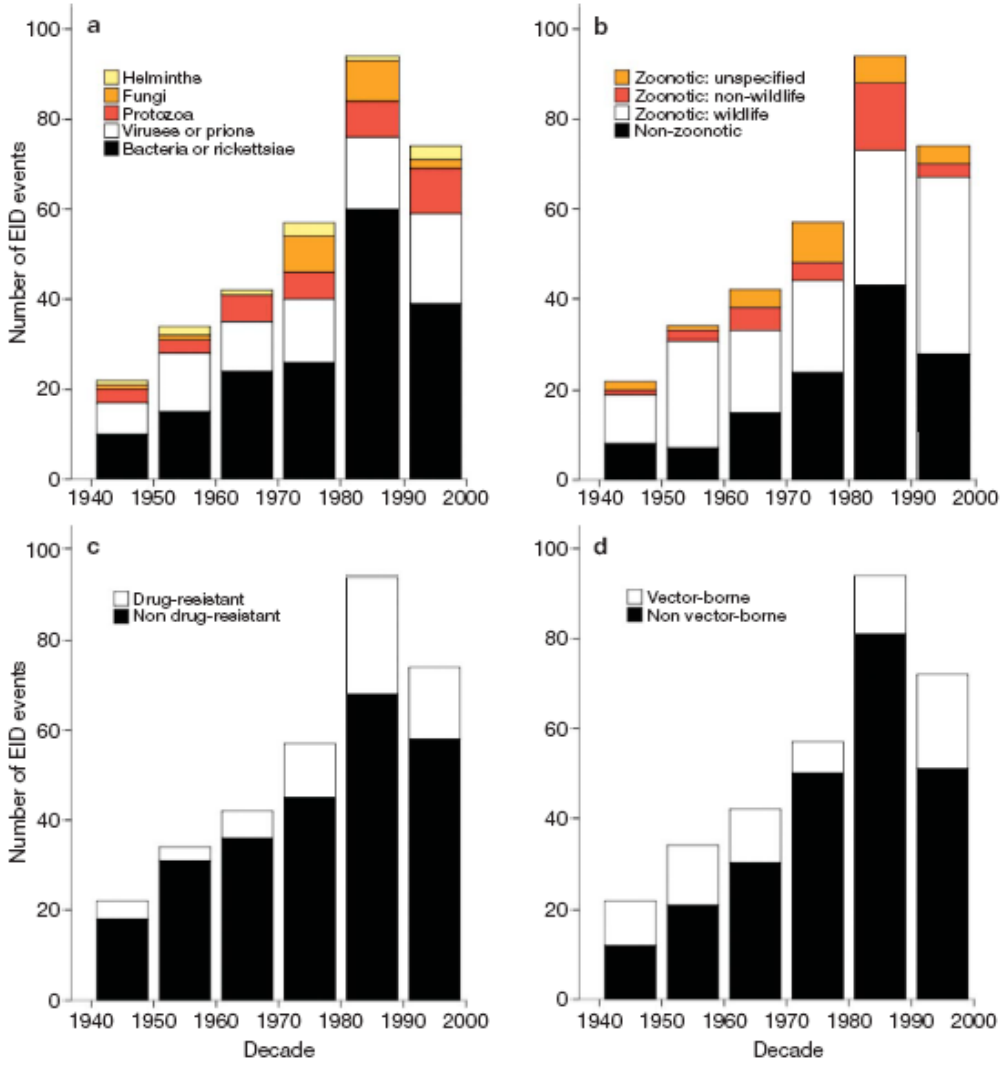
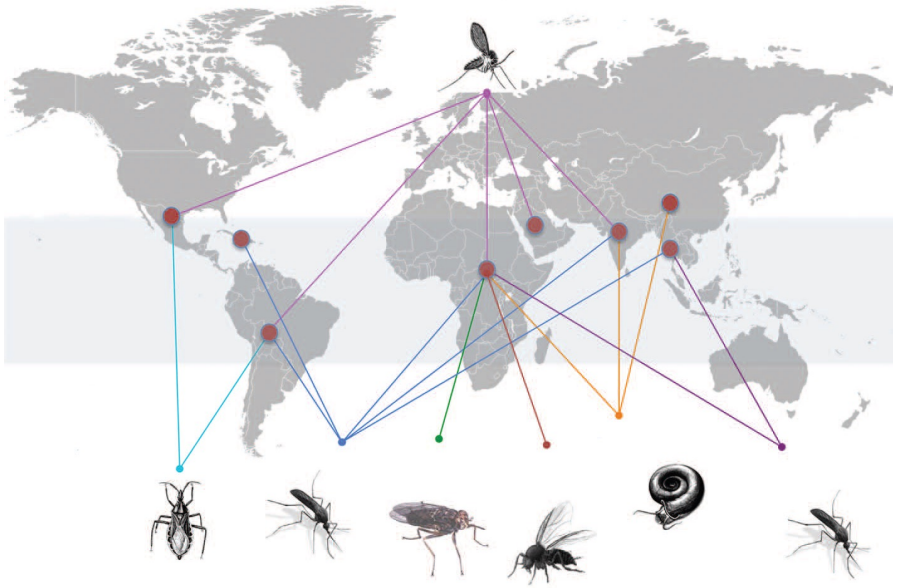


Figure 1 | Number of EID events per decade. EID events (defined as the temporal origin of an EID, represented by the original case or cluster of cases that represents a disease emerging in the human population—see Methods) are plotted with respect to **a**, pathogen type, **b**, transmission type, **c**, drug resistance and **d**, transmission mode (see keys for details).

K. E. Jones et al. Nature 2008; 451:990-994

Vector-borne diseases



Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae, Plasmodium ovale	Malaria	Anopheline mosquito	Africa, Asia, South America
Trypanosoma cruzi	Chagas disease	Kissing/ Assasin bugs (Triatomine bugs)	South America, Southern USA
Trypanosoma brucei	Sleeping sickness	Tsetse flies (*Glossina*)	East and west sub-Saharan Africa
Leishmania major, Leishmania mexica, Leishmania donovani, Leishmania infantum, Leishmania brasiliensis	Cutaneous and visceral Leishmaniasis	Sandflies (*Lutzomyia, Phlebotomus*)	
Schistosoma mansoni, Schistosoma japonicum	Bilharziosis	Freshwater snails, *Biomphalaria, Bulinus Oncomelania*	Africa, Asia, South America
Brugia malayi, Brugia timori, Wurcheraria bancrofti	Lymphatic filariasis		Africa, Asia
Onchocerca volvulus	River blindness	Blackflies (*Simulium*)	Sub Saharan Africa

Keith R. Matthews *Science* **331**, 1149 (2011)

West Nile Virus

Transmission season 2010 (latest update 20 November 2014)

Common mosquito

Area reporting cases in 2010

Area not reporting cases in 2010

Not included

Malta

Andorra

Monaco

San Marino

Liechtenstein

ECDC

West Nile Virus

Transmission season 2011 (latest update 20 November 2014)

Common mosquito

Area reporting cases in 2011

Area not reporting cases in 2011

Not included

Malta

Andorra

Monaco

San Marino

Liechtenstein

ECDC

West Nile Virus

Transmission season 2012 (latest update 20 November 2014)

Common mosquito

Area reporting cases in 2012

Area not reporting cases in 2012

Not included

Malta

Andorra

Monaco

San Marino

Liechtenstein

ECDC

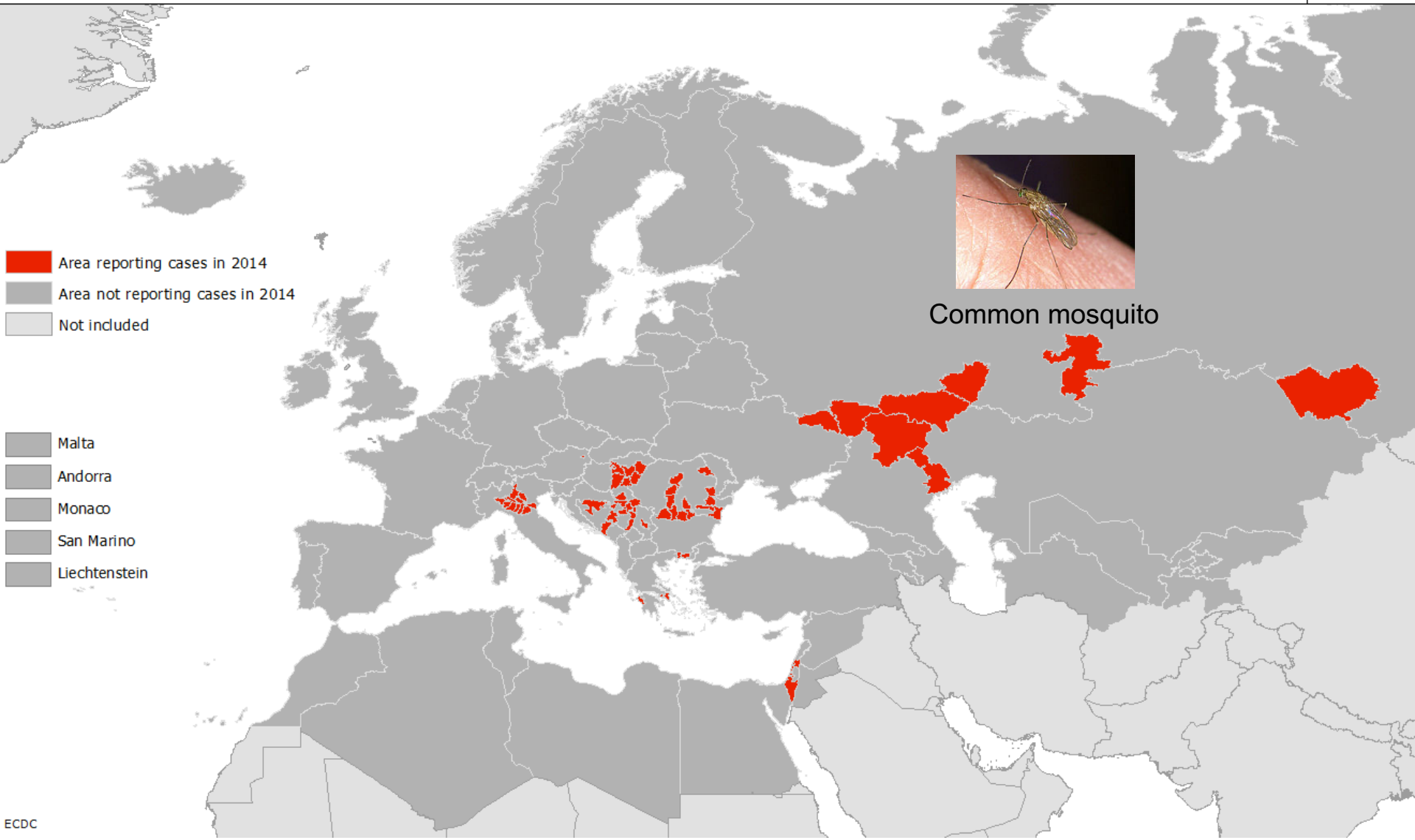
West Nile Virus

Transmission season 2013 (latest update 20 November 2014)

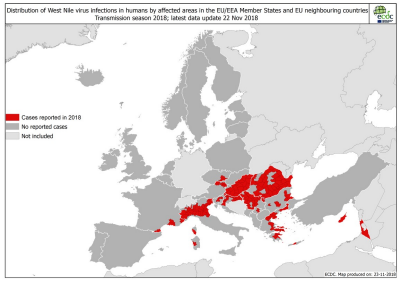
Common mosquito

West Nile Virus

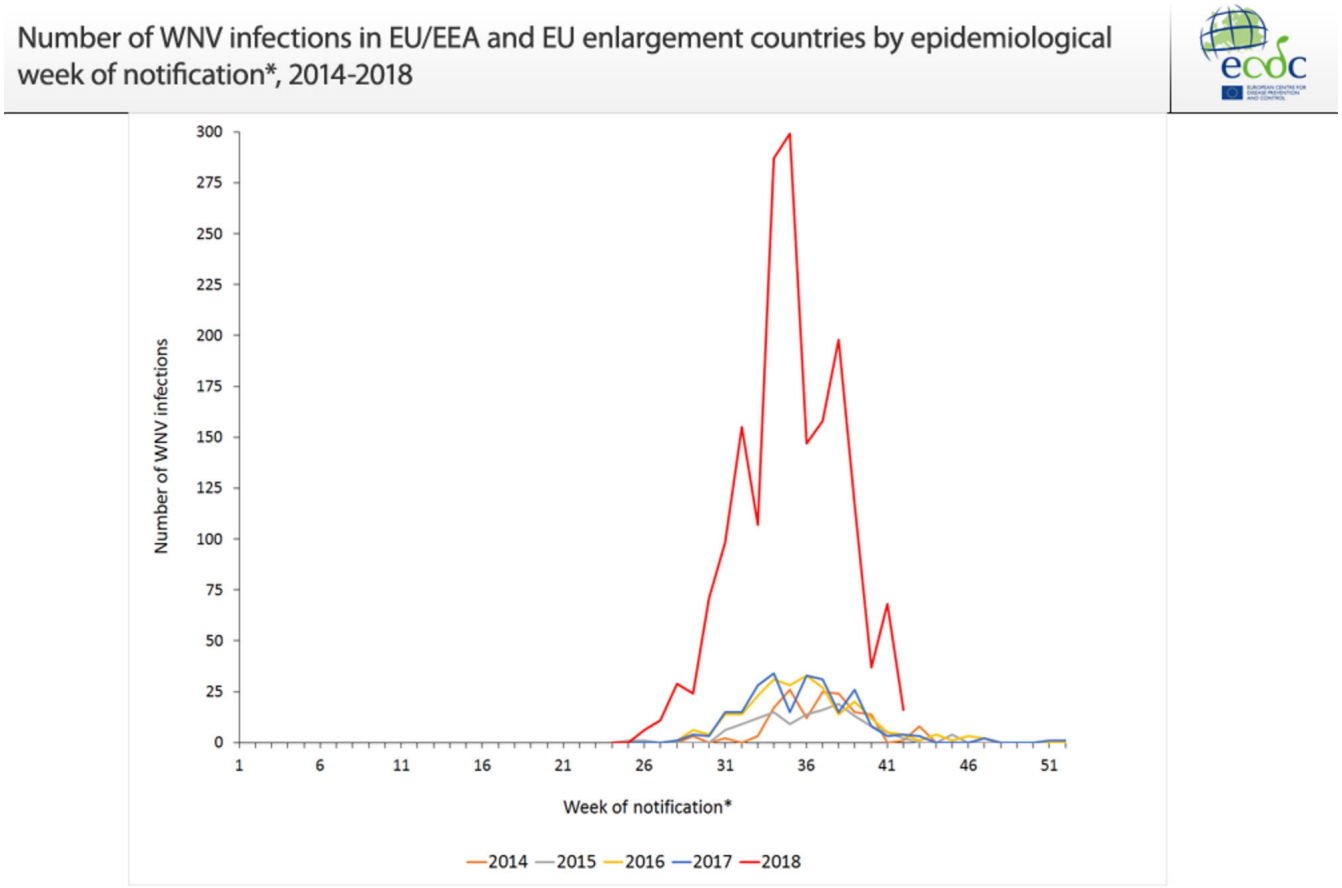
Transmission season 2014; latest update 20 November 2014



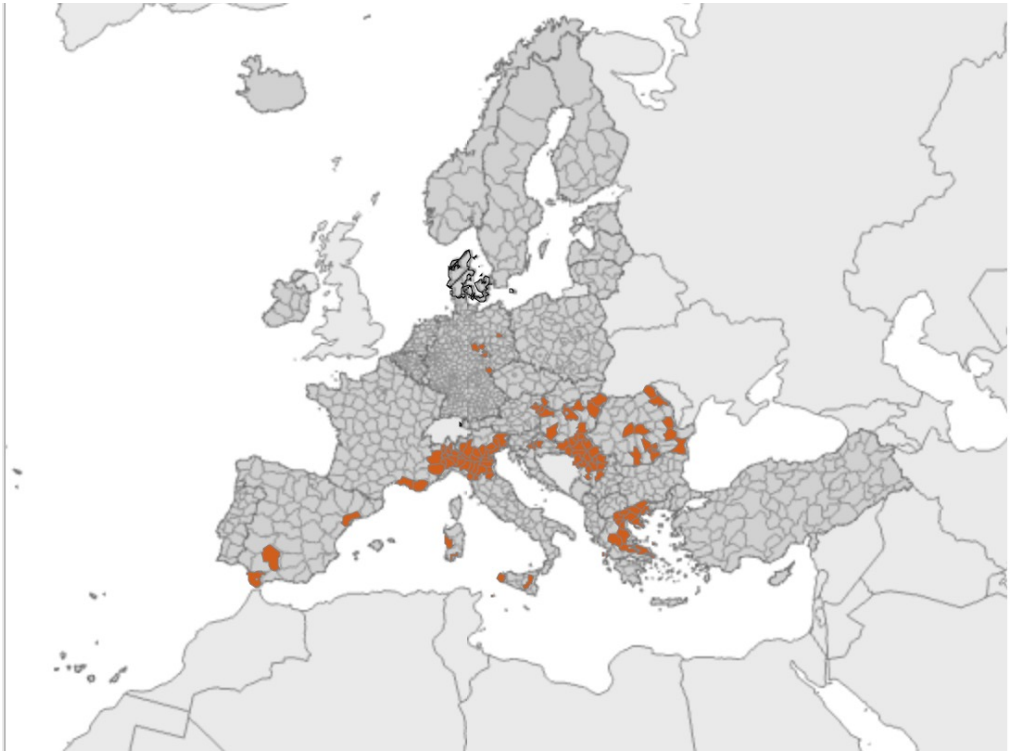
Common mosquito



West Nile Virus in 2018



West Nile Virus in 2022



965 human cases of WNV infection: in Italy (586), Greece (284), Romania (46), Hungary (14), Germany (11), Croatia (8), Austria (6), Spain (5), France (4) and Slovakia (1).
EU/EEA countries have reported 73 deaths:
in Italy (37), Greece (31) and Romania (5). EU-neighbouring countries have reported 226 human cases of WNV infection in Serbia (226) and 12 deaths in Serbia (12).

Updated on 24/11/2023

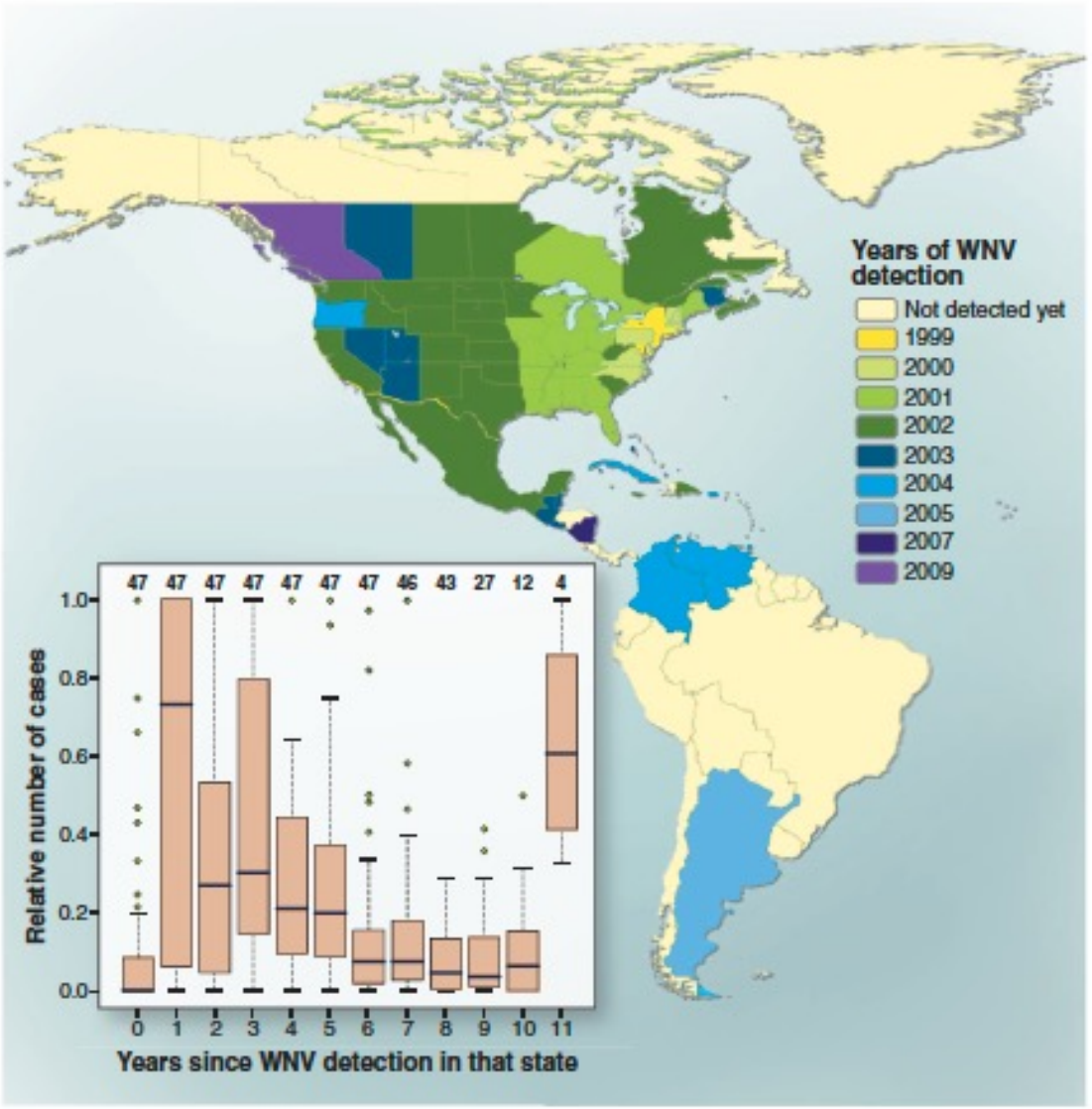
Since last week's update, and as of 22 November 2023, European Union (EU) and European Economic Area (EEA) countries reported 4 human cases of West Nile virus (WNV) infection. All cases were reported by France. EU-neighbouring countries reported no human cases of WNV infection.

Since the beginning of the 2023 transmission season and as of 22 November 2023, EU/EEA countries have reported 698 human cases of WNV infection in Italy (329), Greece (162, of which 1 with unknown place of infection), Romania (103), France (41), Hungary (29), Spain (17), Germany (6), Croatia (6) and Cyprus (5). EU/EEA countries have reported 64 deaths in Italy (26), Greece (23), Romania (12) and Spain (3). EU-neighbouring countries have reported 93 human cases of WNV infection in Serbia (91) and North Macedonia (2) and 2 deaths in Serbia.

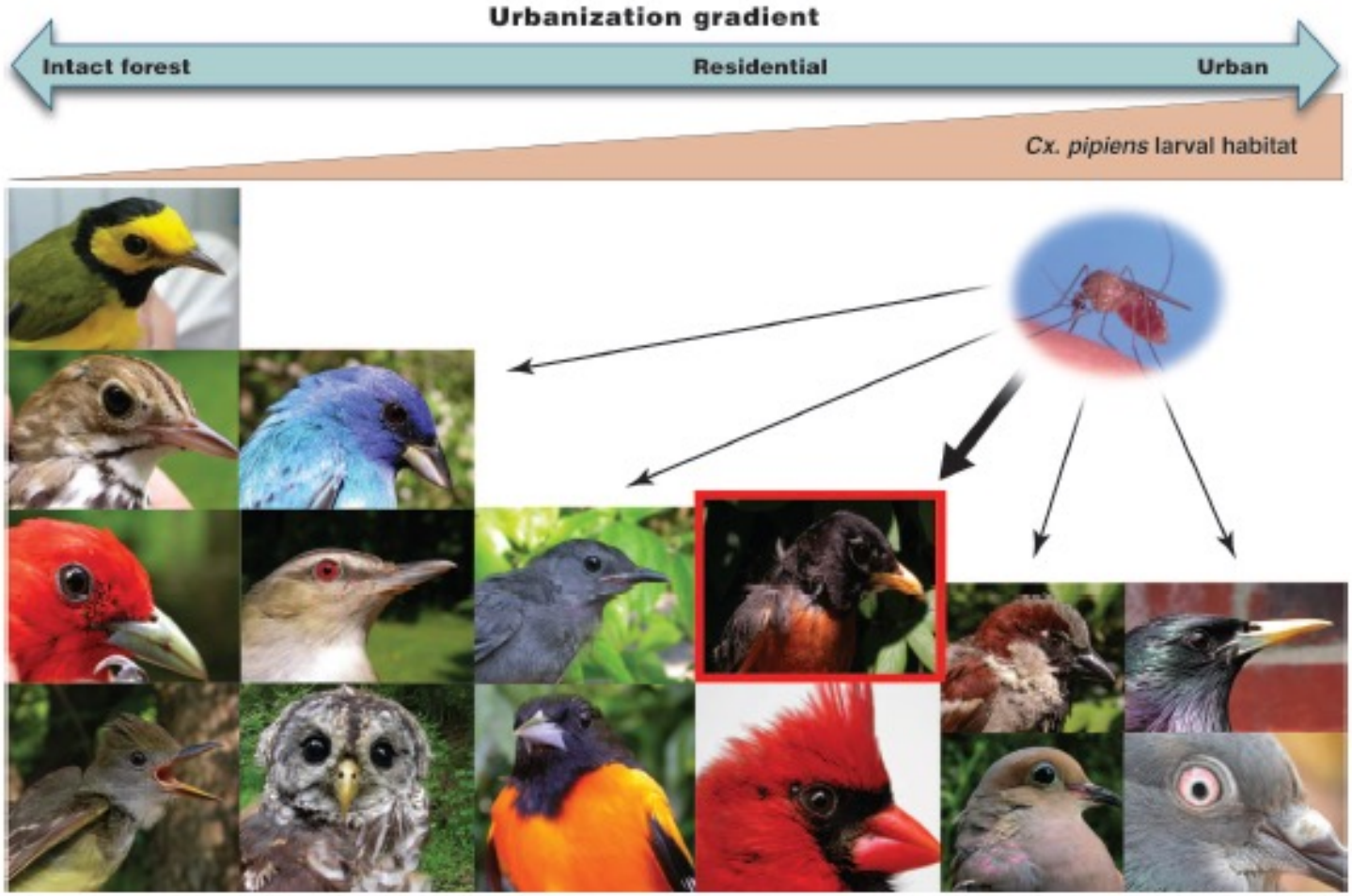
During the current transmission season, within the reporting countries, autochthonous human cases of WNV infection were reported from 140 different NUTS 3 or GAUL 1 regions, of which the following regions reported autochthonous human cases of WNV infection for the first time ever: Charente, Charente-Maritime, Gironde, Haute-Corse and Alpes-Maritimes in France, Sömmerda in Germany, Kastoria and Ioannina in Greece, Cosenza, Bari, Salerno, Lecce, Verbano-Cusio-Ossola, Taranto and Imperia in Italy, Gorj and Timiş in Romania, Cáceres, Huelva, Valencia/València, Barcelona and Toledo in Spain.

Since the beginning of the 2023 transmission season, 146 outbreaks among equids and 246 outbreaks among birds have been reported by EU/EEA countries. Outbreaks among equids have been reported by France (44), Spain (36), Hungary (26), Italy (23), Germany (14), Portugal (2) and Austria (1). Outbreaks among birds have been reported by Italy (195), Germany (19), Spain (19), Bulgaria (6), Hungary (3), France (2), Austria (1) and Greece (1).

West Nile Virus



WNV reservoirs



The host-parasite ecological continuum

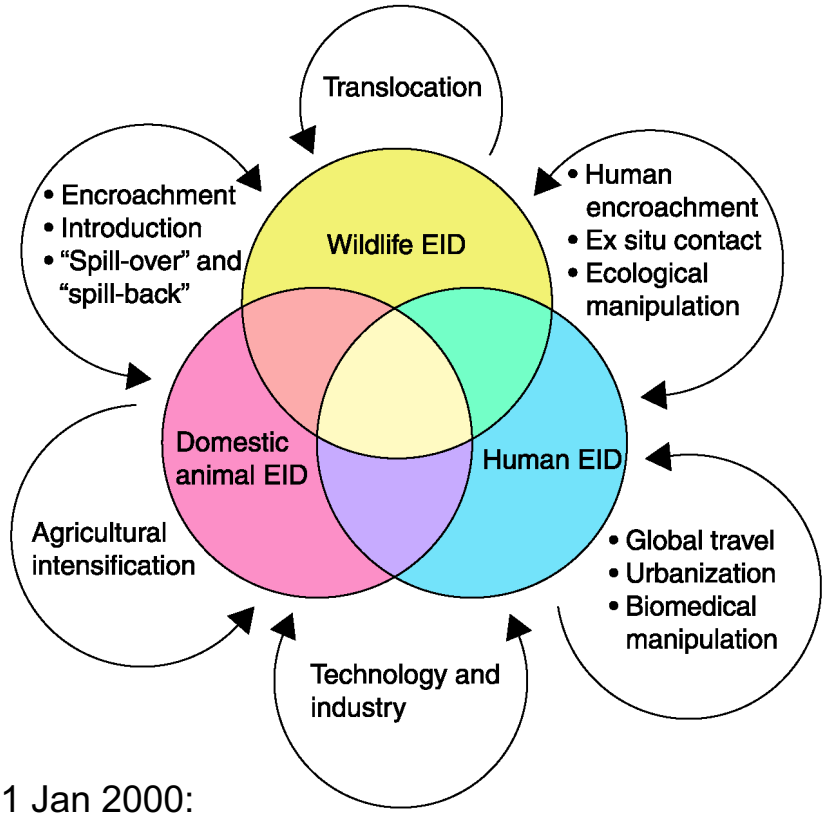
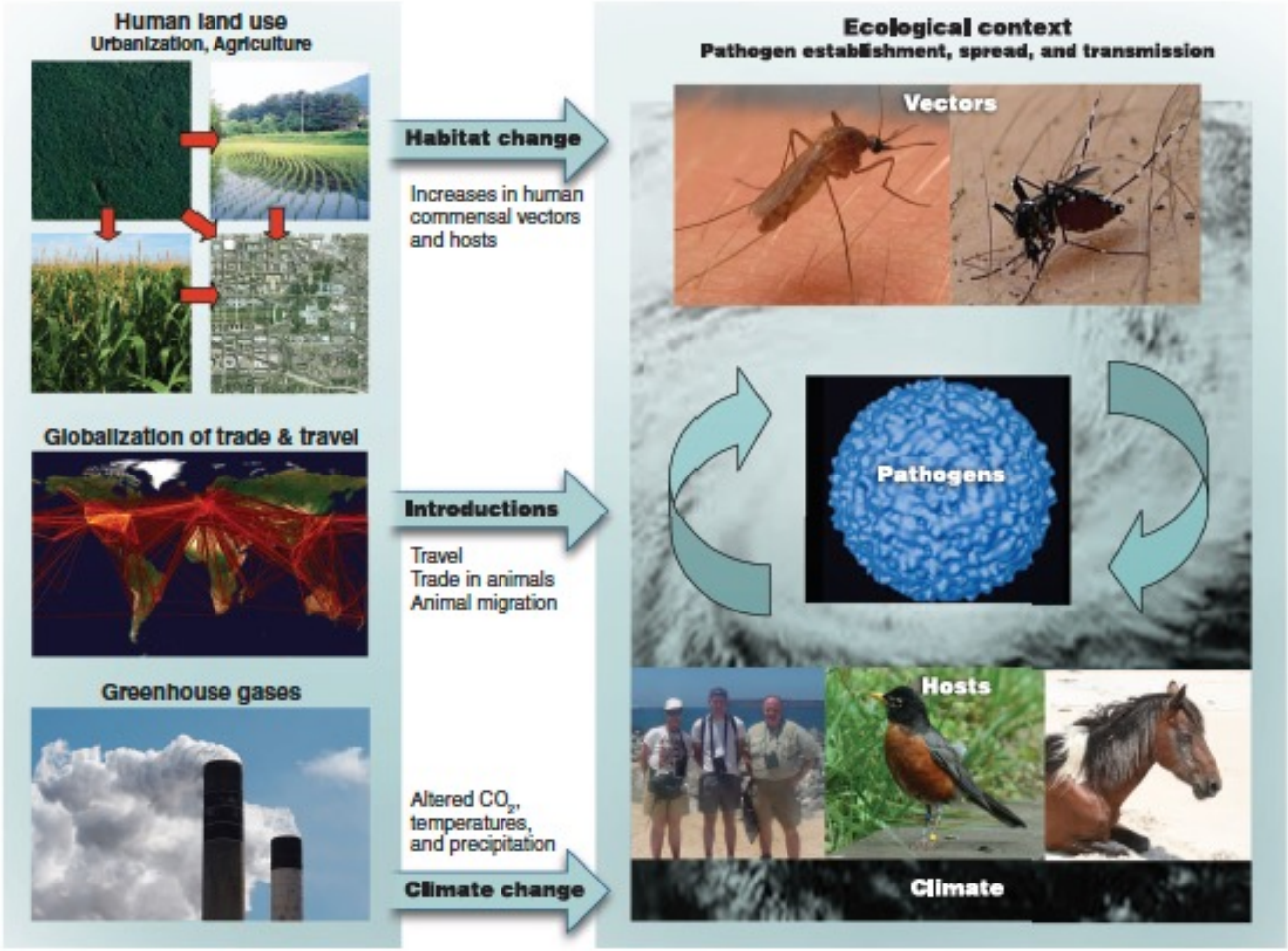


Fig. 1. The host-parasite ecological continuum (here parasites include viruses and parasitic prokaryotes). Most emerging diseases exist within a host and parasite continuum between wildlife, domestic animal, and human populations. Few diseases affect exclusively any one group, and the complex relations between host populations set the scene for disease emergence. Examples of EIDs that overlap these categories are canine distemper (domestic animals to wildlife), Lyme disease (wildlife to humans), cat scratch fever (domestic animals to humans) and rabies (all three categories). Arrows denote some of the key factors driving disease emergence.

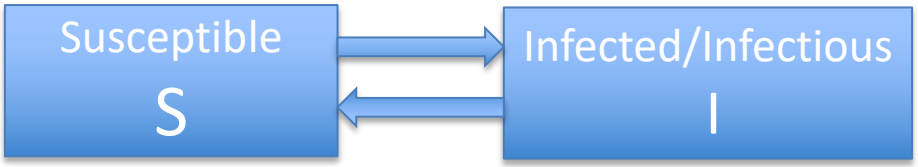
Science 21 Jan 2000:
Vol. 287, Issue 5452, pp. 443-449
DOI: 10.1126/science.287.5452.443

The *drivers* of change

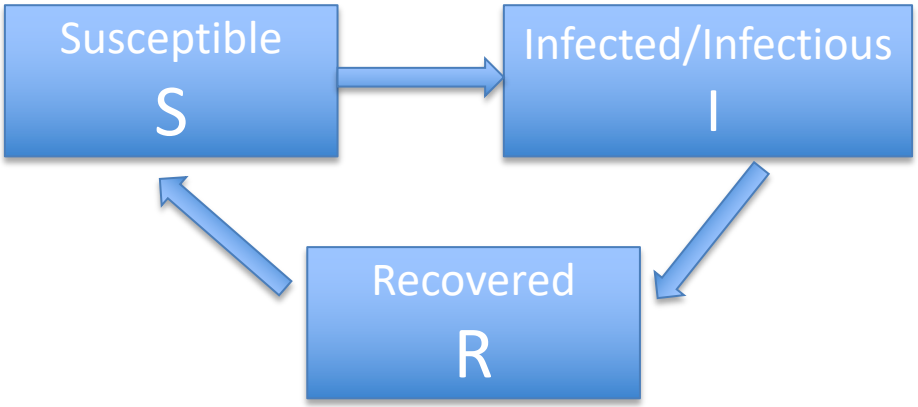


A. Marm Kilpatrick, Globalization, Land Use, and the Invasion of West Nile Virus, *Science* 334, 323 (2011)

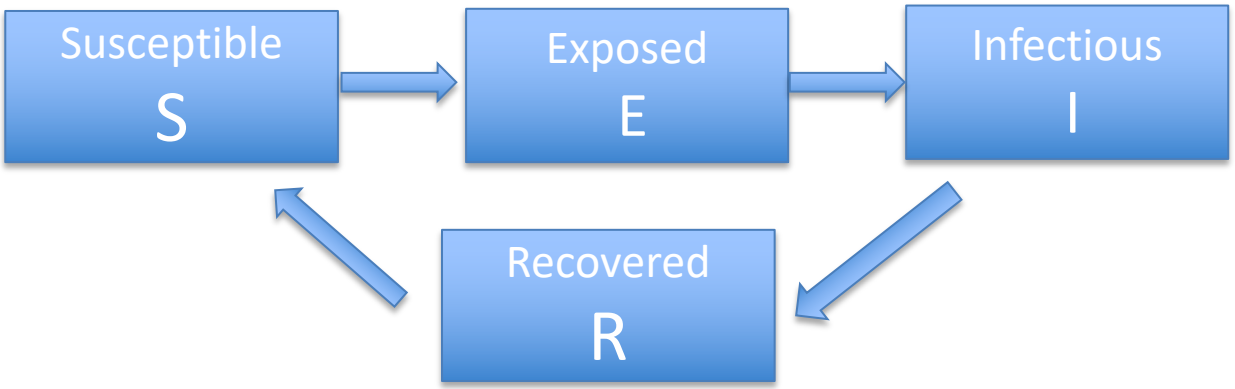
Microparasitic models



SI (SIS) models

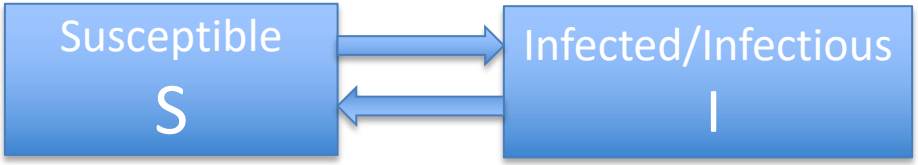


SIR (SIRS) models



SEIR (SEIRS) models

The SI model



$$\frac{dS}{dt} = \nu_S S + \nu_I I - \mu S - iS + \gamma I$$

$$\frac{dI}{dt} = iS - (\mu + \alpha + \gamma) I$$

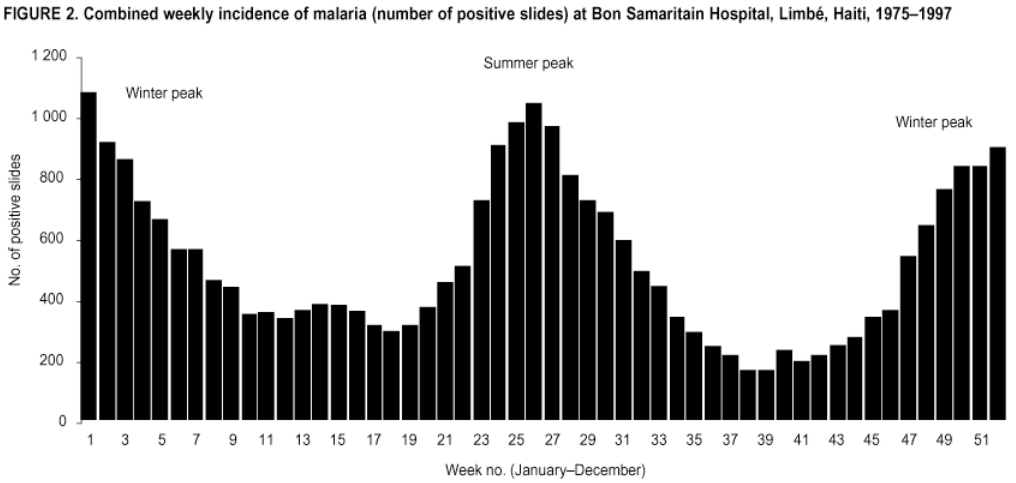
ν = birth-rate μ = natural death-rate

α = disease related death rate γ = recovery rate

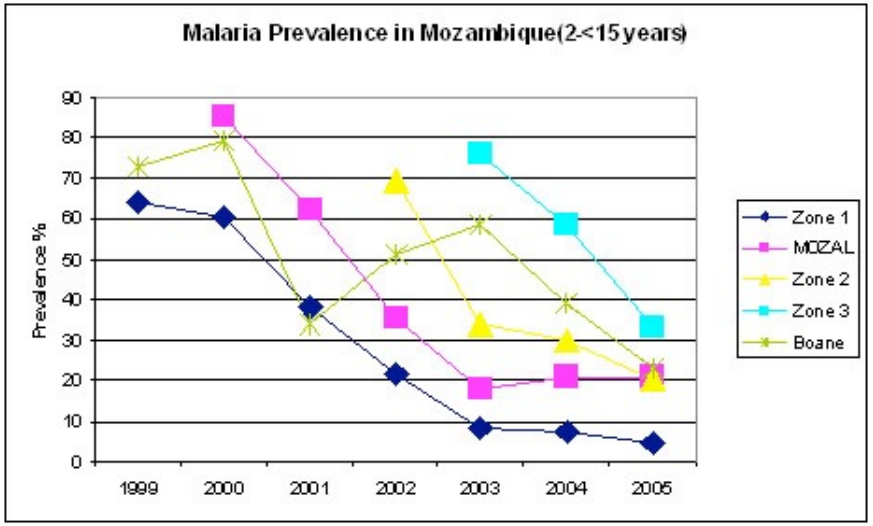
i = infection rate

Incidence and prevalence

Incidence = *iS* = flow of newly infected



Prevalence = *I/N* = *I*/(*S*+*I*) = Infected/Total



Density and frequency-dependent transmission

i = infection rate (probability per unit time susceptible gets infected) $\div$ $c(N) \times I/N \times$ constant probability becoming infected and infectious

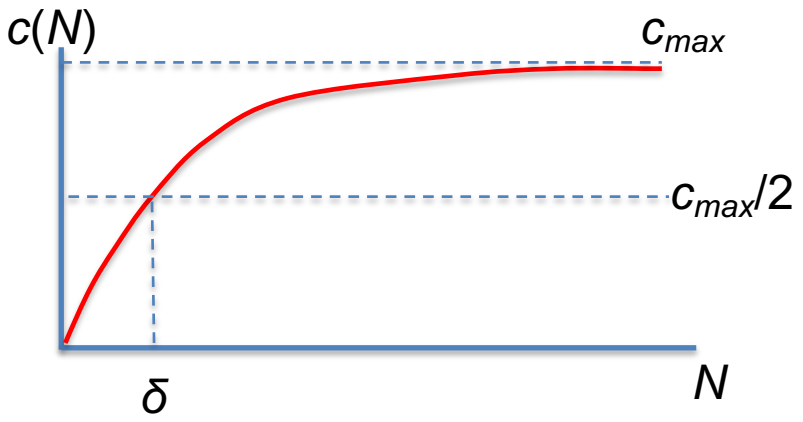
$c(N)$ = contact rate = No. contacts per unit time

- Density-dependence: $c(N) \div N$ (e.g. airborne transmission) → i is proportional to I (law of mass transmission)
- Frequency-dependence: $c(N)$ is constant (e.g. sexually transmitted diseases) → i is proportional to I/N, the prevalence

Both assumptions are unrealistic if considered for $0 \leq N < +\infty$

$c(N) \div N/(\delta + N)$

δ = half-saturation constant



Malthusian growth and DD transmission

$$\dot{S} = rS - \beta IS + \gamma I$$
$$\dot{I} = \beta IS - (\mu + \alpha + \gamma) I$$

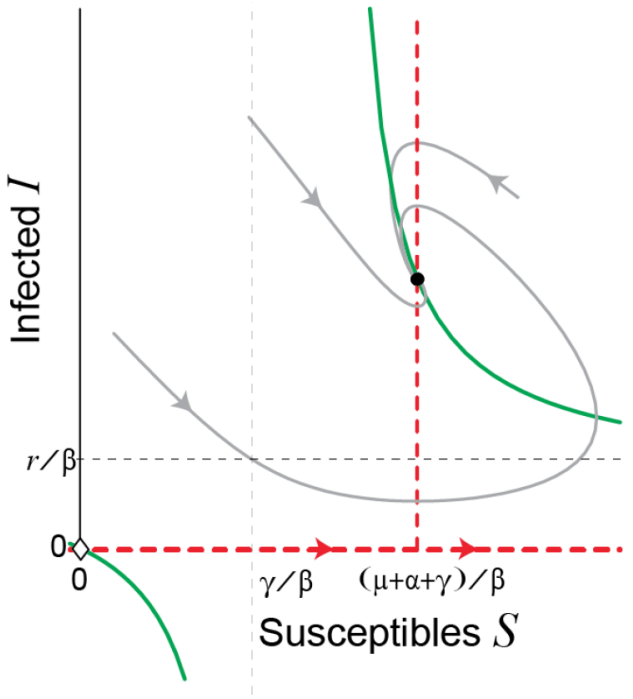
i is proportional to *I*

$r = \nu - \mu$

ν = birth-rate μ = natural death-rate
α = disease related death rate
γ = recovery rate
β = infection rate coefficient

If no infection $S = N$ and $N \div \exp(rt)$

Main message
DISEASE REGULATES THE POPULATION



Isoclines and equilibria

ISOCLINES

$dS/dt = 0 \rightarrow I = rS/(\beta S - \gamma)$

$dI/dt = 0 \rightarrow$ i) $I = 0$ ii) $S = (\mu+\alpha+\gamma)/\beta$

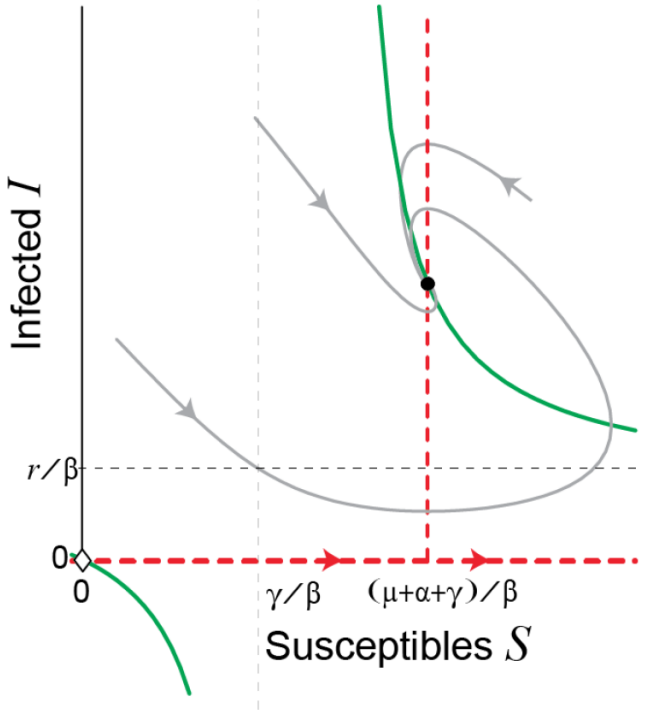
Two equilibria: $dS/dt = dI/dt = 0$

i) $\bar{S}_0 = 0, \bar{I}_0 = 0;$

ii) $\bar{S} = \frac{\mu+\alpha+\gamma}{\beta}, \bar{I} = \frac{r(\mu+\alpha+\gamma)}{\beta(\mu+\alpha)}$

$$\bar{N} = \bar{S} + \bar{I} = \frac{(\mu+\alpha+\gamma)(\nu_S+\alpha)}{\beta(\mu+\alpha)}$$

$$\frac{\bar{I}}{\bar{N}} = \frac{r}{\nu_S+\alpha}$$



← Prevalence decreases with α

Logistic growth and DD transmission

$$\dot{S} = rS\left(1 - \frac{S+I}{K}\right) - \beta IS + \gamma I$$
$$\dot{I} = \beta IS - (\mu + \alpha + \gamma)I$$

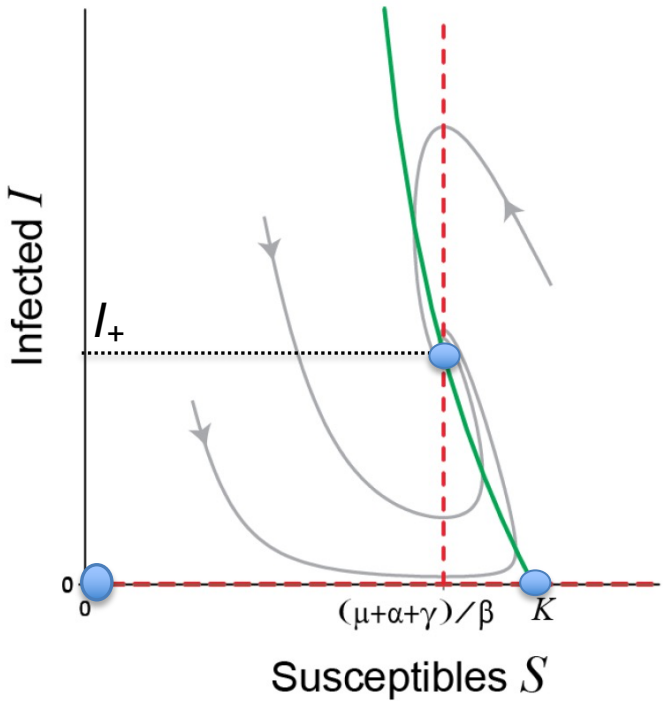
Hyp: μ = natural death-rate = constant
K = carrying capacity

ISOCLINES

$dS/dt = 0 \rightarrow I = \dfrac{rS(1 - {}^{S}/_{K})}{(\beta + {}^{r}/_{K})S - \gamma}$

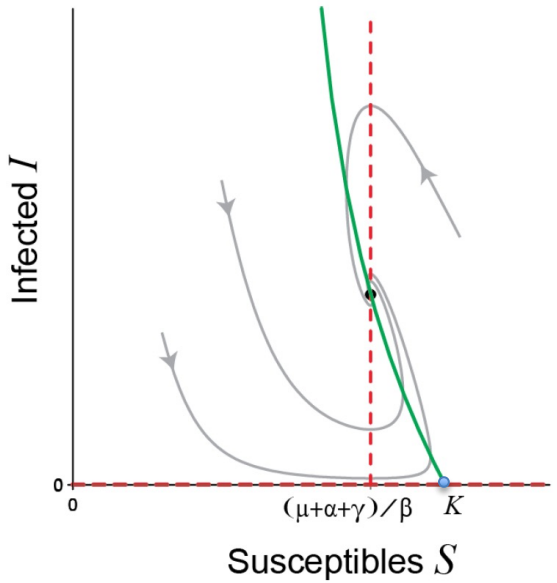
$dI/dt = 0 \rightarrow$ i) $I = 0$ ii) $S = (\mu+\alpha+\gamma)/\beta$

Equilibria: $dS/dt = dI/dt = 0$
3 possible equilibria
i) $S = 0$ $I = 0$ Trivial
ii) $S = K$ $I = 0$ DFE = disease-free equil.
iii) $S = (\mu+\alpha+\gamma)/\beta$ $I = I_+$



The basic reproduction number

$$\bar{I}_+ = \frac{rK(\mu+\alpha+\gamma)}{\beta K(\mu+\alpha)+r(\mu+\alpha+\gamma)}\left(1-\frac{\mu+\alpha+\gamma}{\beta K}\right) \qquad \frac{\bar{I}}{\bar{N}} = \frac{r}{\mu+\alpha+r}\frac{\beta K-(\mu+\alpha+\gamma)}{\beta K}$$



$\bar{I}_+$ is feasible > 0 only if

$K > (\mu+\alpha+\gamma)/\beta$

Equivalently

$$R_0 = \frac{\beta K}{\mu+\alpha+\gamma} > 1$$

$1/(\mu+\alpha+\gamma)$ = mean residence time in the infectious compartment

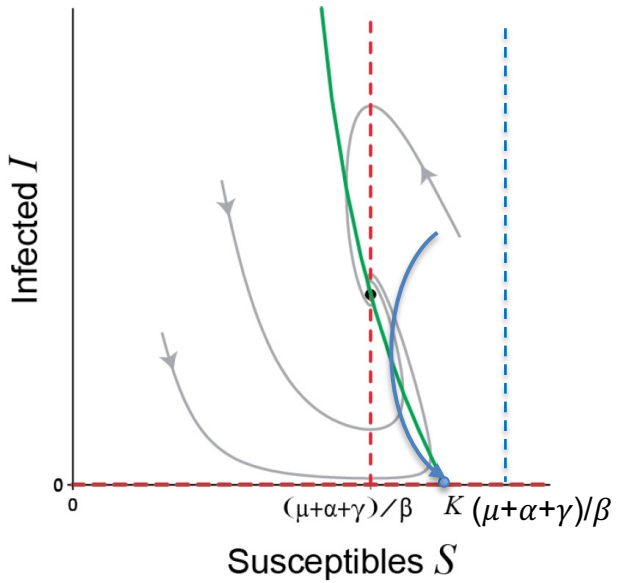
βK = No. of susceptibles infected per unit time in a disease free population

R_0 = average number of secondary infections caused by one primary infection in a healthy population at carrying capacity

If $R_0 < 1$ → DFE is stable, disease cannot become endemic

The basic reproduction number

$$\bar{I}_+ = \frac{rK(\mu+\alpha+\gamma)}{\beta K(\mu+\alpha)+r(\mu+\alpha+\gamma)}\left(1-\frac{\mu+\alpha+\gamma}{\beta K}\right) \qquad \frac{\bar{I}}{\bar{N}} = \frac{r}{\mu+\alpha+r}\frac{\beta K-(\mu+\alpha+\gamma)}{\beta K}$$



$\bar{I}_+$ is feasible > 0 only if

$K > (\mu+\alpha+\gamma)/\beta$

Equivalently

$$R_0 = \frac{\beta K}{\mu+\alpha+\gamma} > 1$$

$1/(\mu+\alpha+\gamma)$ = mean residence time in the infectious compartment

βK = No. of susceptibles infected per unit time in a disease free population

R_0 = average number of secondary infections caused by one primary infection in a healthy population at carrying capacity

If $R_0 < 1$ → DFE is stable, disease cannot become endemic

Logistic demography and saturating transmission

$$i = \beta \frac{N}{\delta + N} \frac{I}{N} = \beta \frac{I}{\delta + N}$$

$$\begin{aligned} \dot{S} &= rS\left(1 - \tfrac{S+I}{K}\right) - \beta \tfrac{IS}{\delta+S+I} + \gamma I \\ \dot{I} &= \beta \tfrac{IS}{\delta+S+I} - (\mu + \alpha + \gamma) I \end{aligned}$$

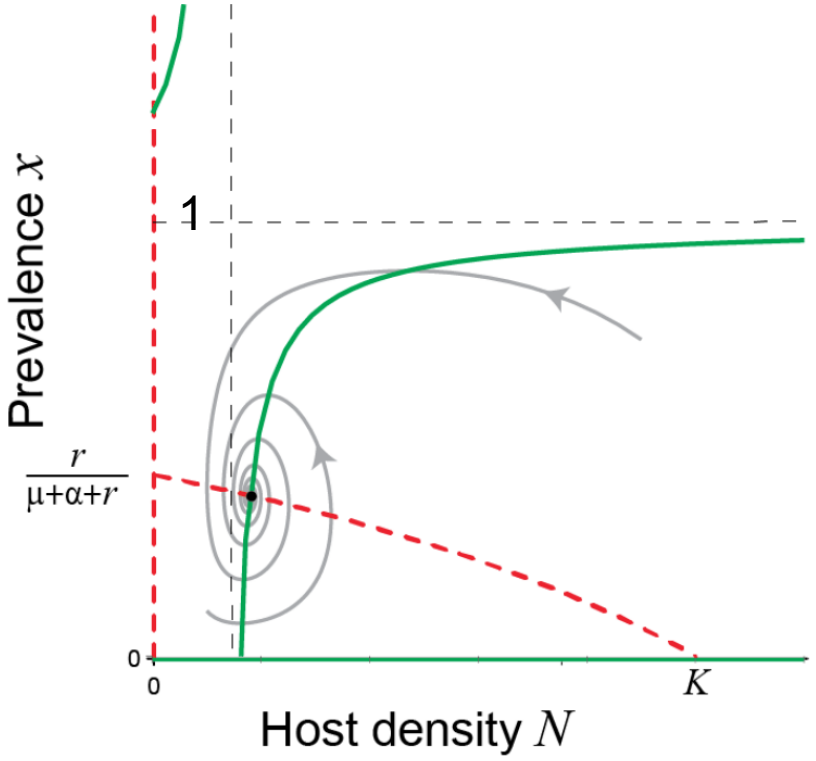
$N = S + I \quad$ prevalence $x = I/(S+I)$ Change variables

$$\begin{aligned} \dot{N} &= \left[r\left(1-x\right)\left(1 - \tfrac{N}{K}\right) - \left(\mu + \alpha\right) x\right] N \\ \dot{x} &= \left[\left(\beta \tfrac{N}{\delta+N} - r\left(1 - \tfrac{N}{K}\right) - \left(\mu + \alpha\right)\right)\left(1 - x\right) - \gamma\right] x \end{aligned}$$

Logistic demography and saturating transmission (isoclines and R_0)

$$\dot{N} = 0 \quad \Rightarrow \quad x = \frac{r\left(1-\frac{N}{K}\right)}{\mu+\alpha+r\left(1-\frac{N}{K}\right)}$$

$$\dot{x} = 0 \quad \Rightarrow \quad x = 1 - \frac{\gamma}{\beta\frac{N}{\delta+N} - r\left(1-\frac{N}{K}\right) - (\mu+\alpha)}$$



Contact rate

$c(N) \div N/(\delta + N)$

At $N=K$ must be < 1

$$\beta\frac{K}{\delta+K} - (\mu+\alpha) > \gamma$$

$$R_0 = \frac{\beta K}{(\delta+K)(\mu+\alpha+\gamma)}$$

R_0 = average number of secondary infections caused by one primary infection in a healthy population at carrying capacity

Frequency-dependent contact rate

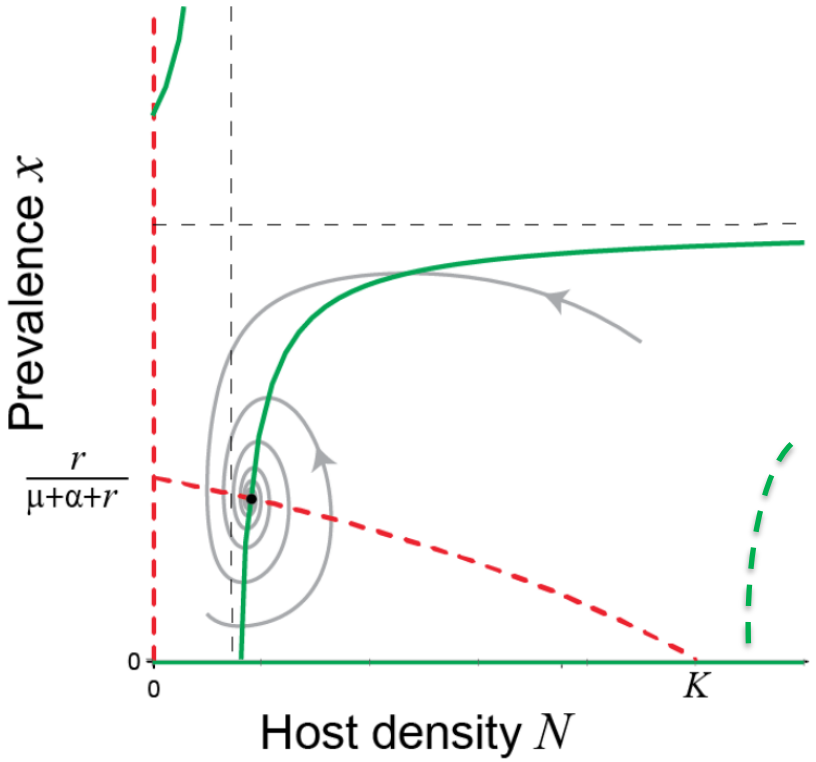
$\delta \rightarrow 0$

$$R_0 \cong \frac{\beta}{\mu+\alpha+\gamma}$$

Logistic demography and saturating transmission (isoclines and R_0)

$$\dot{N} = 0 \quad \Rightarrow \quad x = \frac{r\left(1-\frac{N}{K}\right)}{\mu+\alpha+r\left(1-\frac{N}{K}\right)}$$

$$\dot{x} = 0 \quad \Rightarrow \quad x = 1 - \frac{\gamma}{\beta\frac{N}{\delta+N} - r\left(1-\frac{N}{K}\right) - (\mu+\alpha)}$$



Contact rate
$c(N) \div N/(\delta + N)$

At $N=K$ must be < 1

$$\beta\frac{K}{\delta+K} - (\mu+\alpha) > \gamma$$

$$R_0 = \frac{\beta K}{(\delta+K)(\mu+\alpha+\gamma)}$$

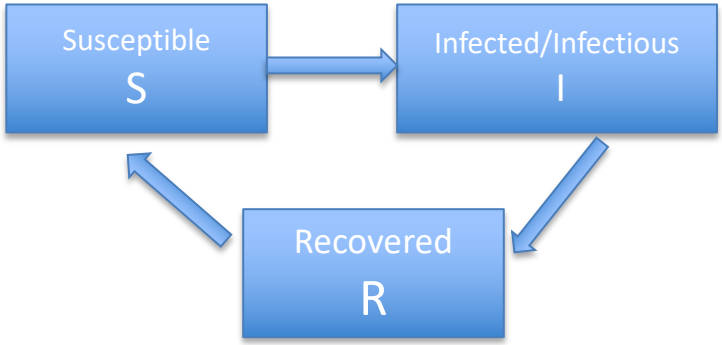
R_0 = average number of secondary infections caused by one primary infection in a healthy population at carrying capacity

Frequency-dependent contact rate
$\delta \rightarrow 0$

$$R_0 \cong \frac{\beta}{\mu+\alpha+\gamma}$$

Malthusian SIR model

$$\begin{aligned}\dot{S} &= \nu_S(S+R) + \nu_I I - \mu S - iS + \gamma R \\ \dot{I} &= iS - (\mu + \alpha + \rho)I \\ \dot{R} &= \rho I - (\mu + \gamma)R\end{aligned}$$



$$\begin{aligned}\dot{S} &= rS + \nu_S R - \beta SI + \gamma R \\ \dot{I} &= \beta SI - (\mu + \alpha + \rho)I \\ \dot{R} &= \rho I - (\mu + \gamma)R\end{aligned}$$

ρ = recovery rate γ = rate of immunity loss

Two possible equilibria: $dS/dt = dI/dt = dR/dt = 0$

i) Trivial $S=I=R=0$ (unstable)

ii) Non trivial (feasible if $S>0$, $I>0$, $R>0$)

Malthusian SIR model

$$\bar{X}_+ = \begin{bmatrix} \bar{S} &= \frac{\mu+\alpha+\rho}{\beta} \\ \bar{I} &= \frac{r(\mu+\alpha+\rho)(\mu+\gamma)}{\beta[(\mu+\alpha+\rho)(\mu+\gamma)-(\nu_S+\gamma)\rho]} \\ \bar{R} &= \frac{r\rho(\mu+\alpha+\rho)}{\beta[(\mu+\alpha+\rho)(\mu+\gamma)-(\nu_S+\gamma)\rho]} \end{bmatrix}$$

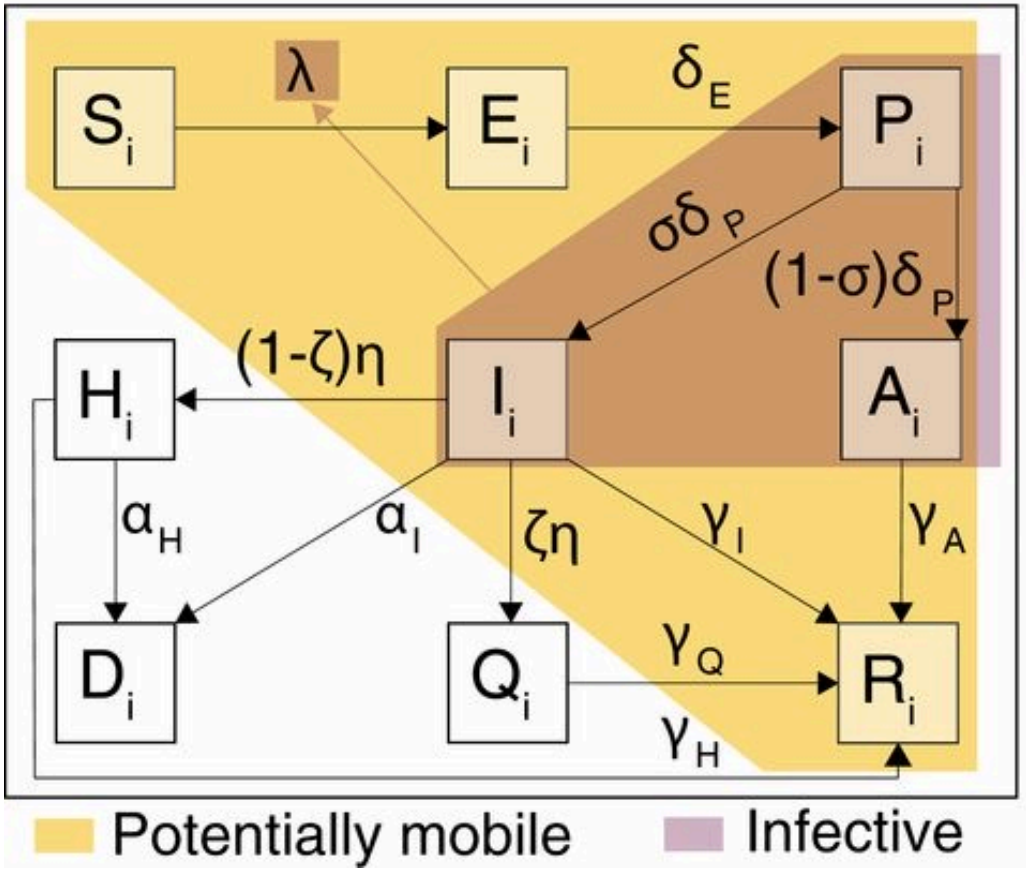
If these terms are positive equilibrium is feasible

Condition for feasibility

$$\rho < \frac{(\mu+\alpha)(\mu+\gamma)}{r}$$

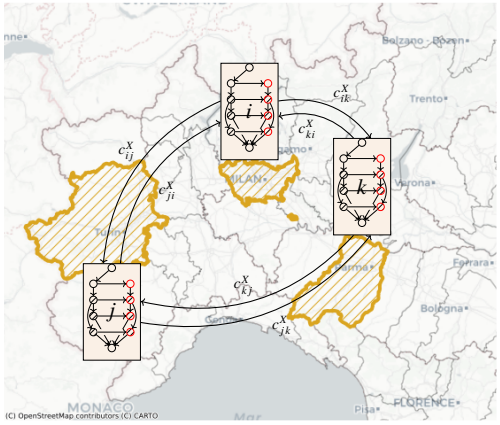
Main message
DISEASE REGULATES AN EXPONENTIALLY INCREASING POPULATION ONLY IF RATE OF RECOVERY IS NOT TOO LARGE

SEIR Models



S_i: susceptibles in site *i*
E_i: exposed in *i*
P_i: pre-symptomatic infectious in *i*
I_i: symptomatic infectious in *i*
A_i: asymptomatic/mildly symptomatic infectious in *i*

H_i , Q_i: Hospitalized, Quarantined and isolated in *i*
D_i , R_i: Deceased, Recovered in *i*



COVID-19 Model

Water-related diseases

Category	Transmission	Disease examples
Water-borne	Ingestion of water contaminated by human or animal faeces or urine containing pathogenic bacteria, viruses or parasites	Gastroenteritis, enteric hepatitis, amoebic & bacillary dysentery, cholera, leptospirosis, poliomyelitis, typhoid/paratyphoid fever
Water-washed	Skin, ear or eye contact with contaminated water & poor personal hygiene	Conjunctivitis, trachoma, intestinal helminth infections, leprosy, scabies
Water-aerosol disease	Inhalation of water aerosol containing pathogen	Legionellosis, phiesteria
Water-based	Parasitical worm infections (parasites found in intermediate organisms living in water)	Dracunculiasis, schistosomiasis, (tricho)bilharziasis
Water-related arthropod vector	Insect vectors breeding in water or biting near water	Dengue, lymphatic filariasis, malaria, onchocerciacis, trypanosomiasis, yellow fever

Key facts

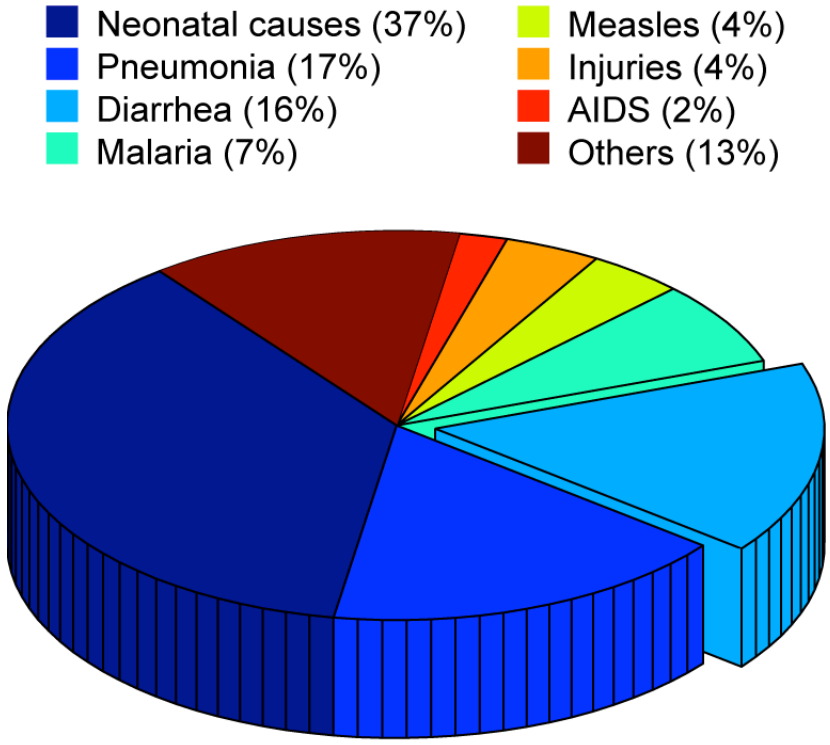
- Globally, at least 2 billion people use a drinking water source contaminated with faeces.
- Microbiologically contaminated drinking water can transmit diseases such as diarrhoea, cholera, dysentery, typhoid and polio and is estimated to cause 485 000 diarrhoeal deaths each year.
- More generally an estimated 829,000 deaths are WASH-attributable (water, sanitation and hygiene behaviours) and 49.8 million DALYs (disability-adjusted life years) occurred from diarrhoeal diseases in 2016, equivalent to 60% of all diarrhoeal deaths.
- In children under 5 years, 297,000 WASH-attributable diarrhoea deaths occurred, representing 5.3% of all deaths in this age group.

Prüss-Ustün A. et al. Int J Hyg Environ Health. 2019 Jun;222(5):765-777

Safer water, better health. 2019 update. Geneva: World Health Organization; 2019.

Waterborne diseases

- Waterborne diseases: pathogens transmitted when contaminated water (or food contaminated by water) is consumed.
- Pathogens include protozoa (e.g. *Entamoeba hystolitica*), bacteria (e.g. *Vibrio cholerae* and *Shigella dysenteriae*) and viruses (e.g. *Rotavirus gastroenteritis*).

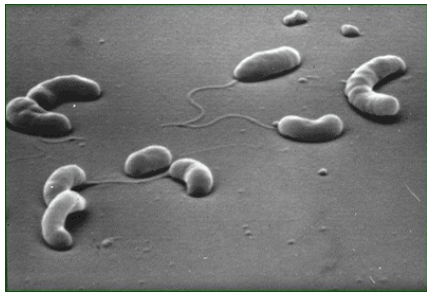


Leading causes of death among children under five years of age

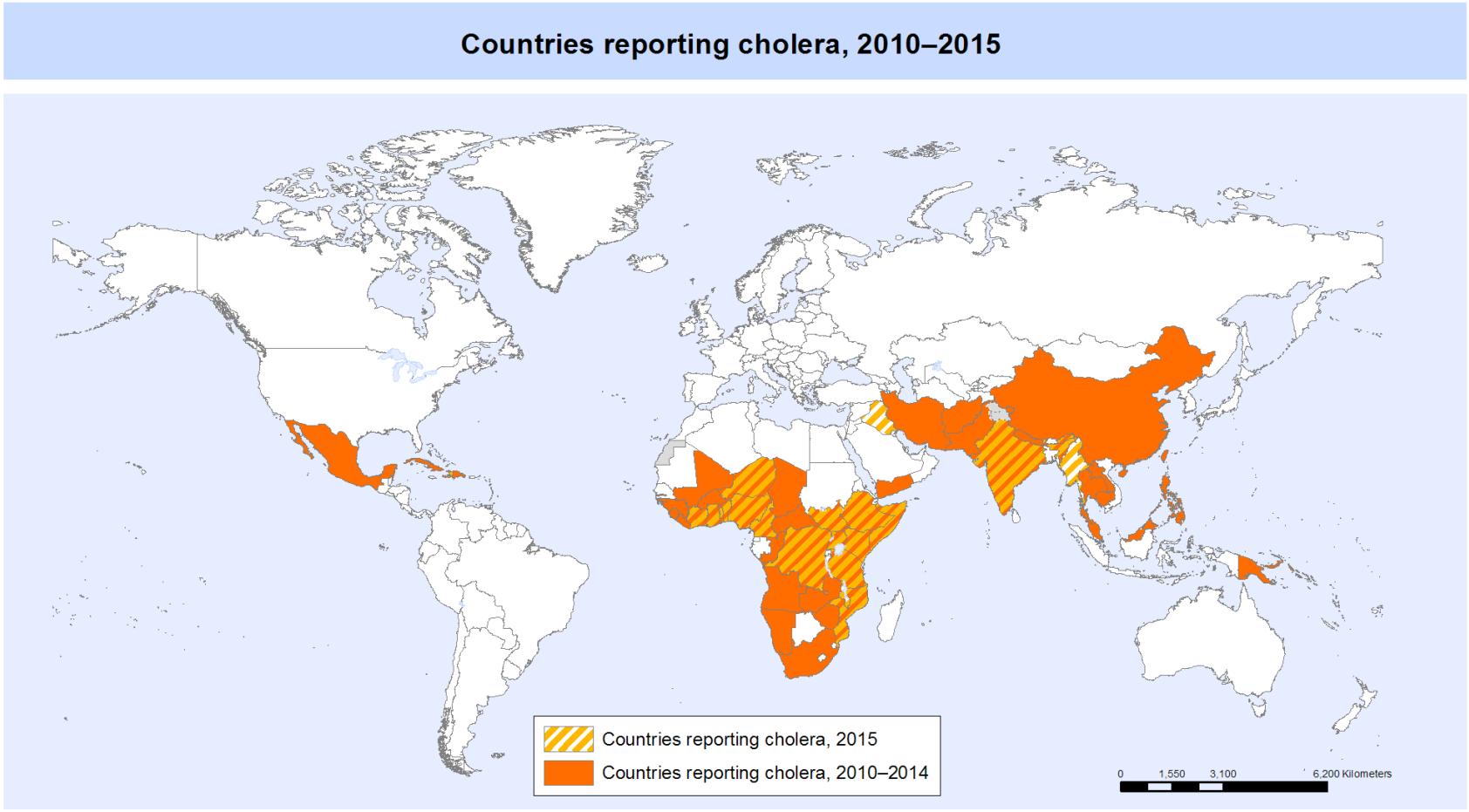
Cholera

- Infection of the small intestine caused by the bacterium *Vibrio cholerae* (Filippo Pacini, 1854).
- The bacterium produces a toxin, which can cause profuse diarrhea and death due to dehydration.
- About 200 serotypes, however only O1 ed O139 (1992, Bangladesh) are pathogenic to humans.
- Transmission is oro-fecal, via the ingestion of contaminated water or food. Usually the infecting inoculum is rather high (ca. 1 million bacteria).

Filippo Pacini



Distribution of cholera in space and time

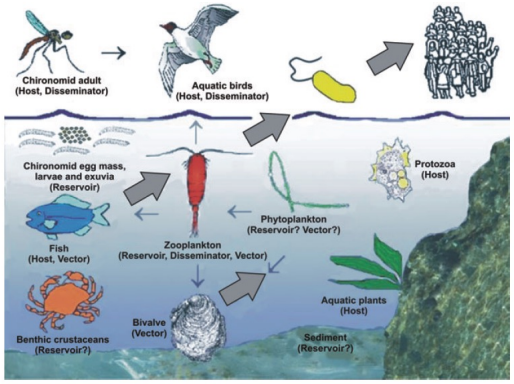


The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted and dashed lines on maps represent approximate border lines for which there may not yet be full agreement.

Data Source: World Health Organization
Map Production: Information Evidence and Research (IER)
World Health Organization

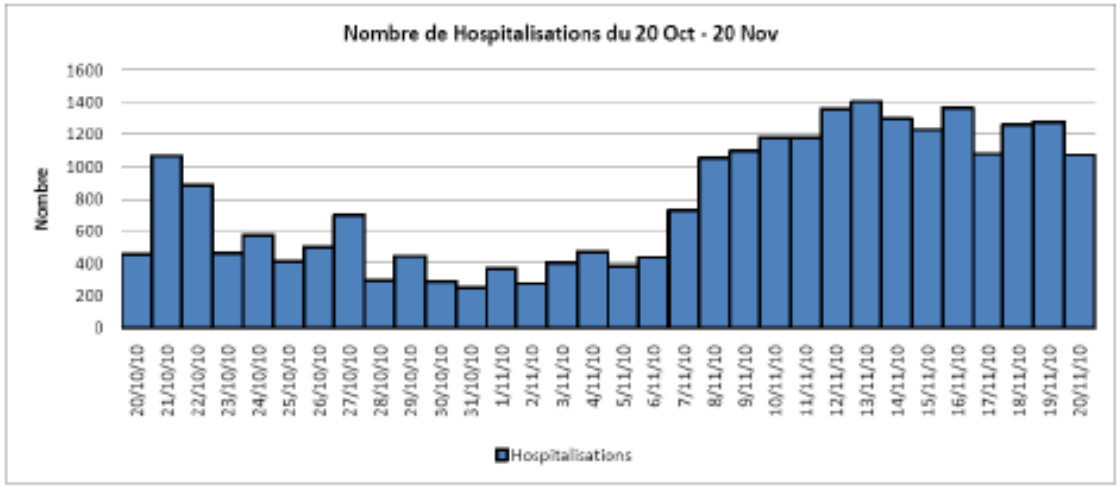
World Health Organization

Cholera characteristics

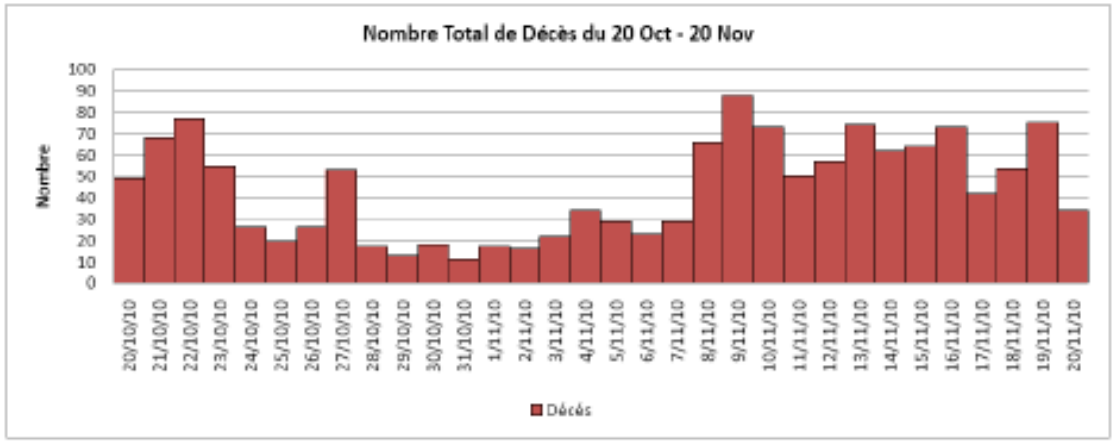


- Main reservoirs: water, humans
- Incubation period: 2 hours-5 days
- 75-85% of infectives are asymptomatic, but produce bacteria in fecal excretions for 7-14 days
- Within symptomatics (15-25%), only 20% develop acute symptoms with watery diarrhea and dehydration
- Recovery time: about 5 days
- Immunity:
  - acquired by infectives (symptomatic and asymptomatic)
  - not permanent, lasts a few years (1-5 ?)

Cholera outbreak in Haiti



Total number of hospitalized cases (20 October – 20 November)



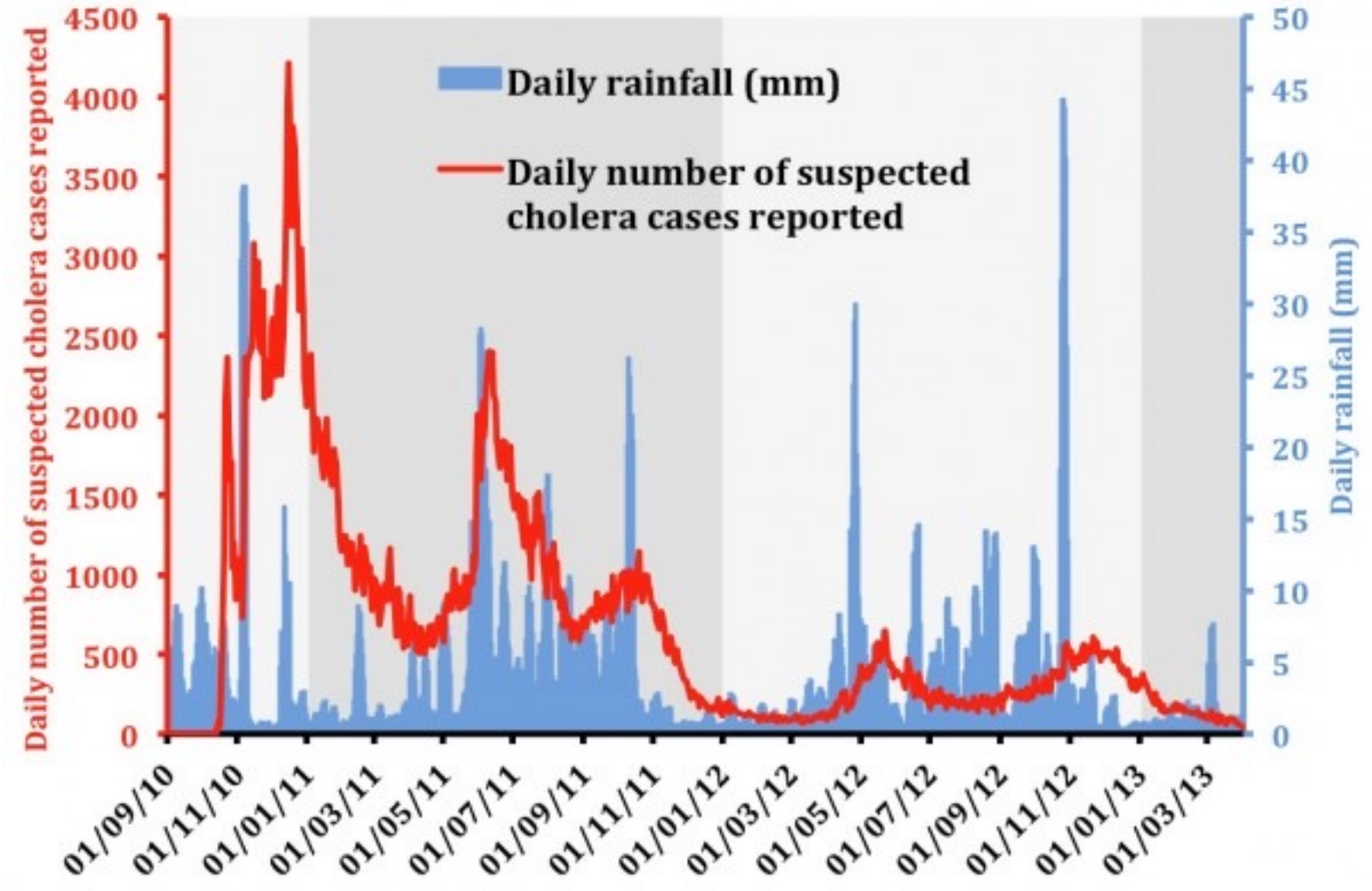
Total number of deaths (20 October – 20 November)

Latest bulletins show a steady increase in the number of hospitalizations

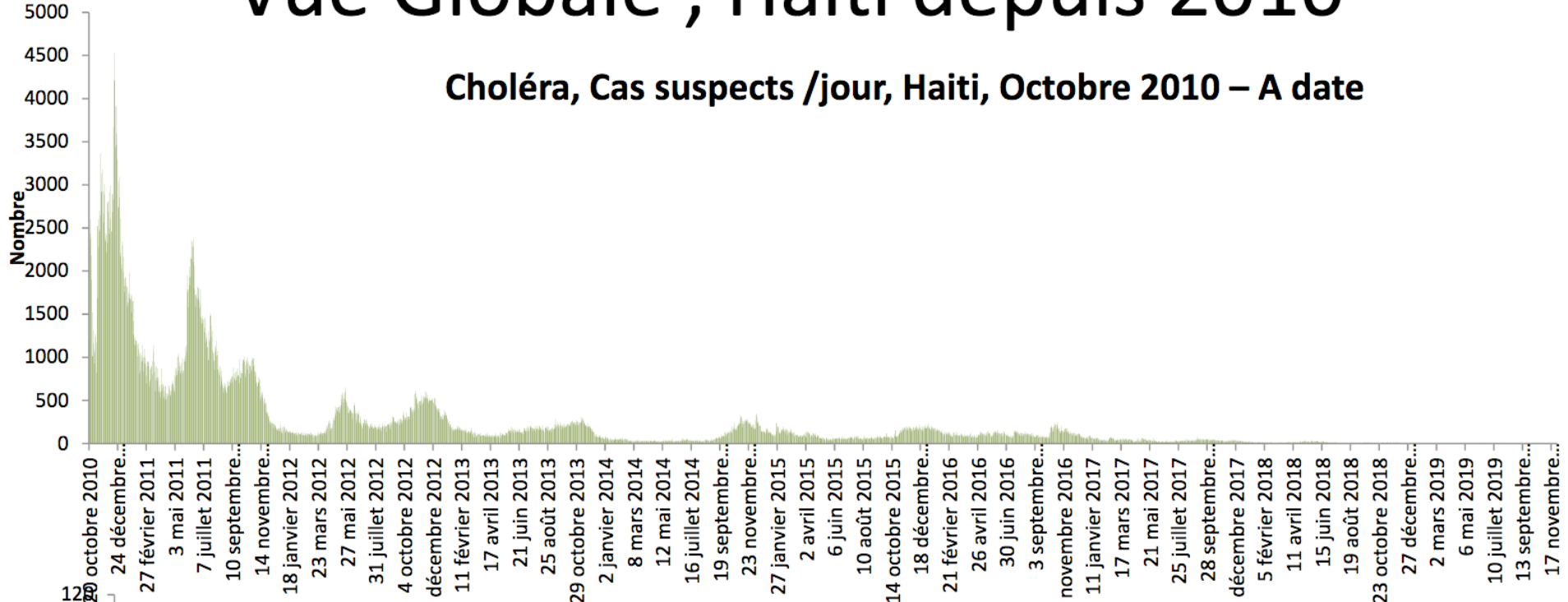
PAHO and UNICEF experts predict a peak for late December, even though the death rate is decaying

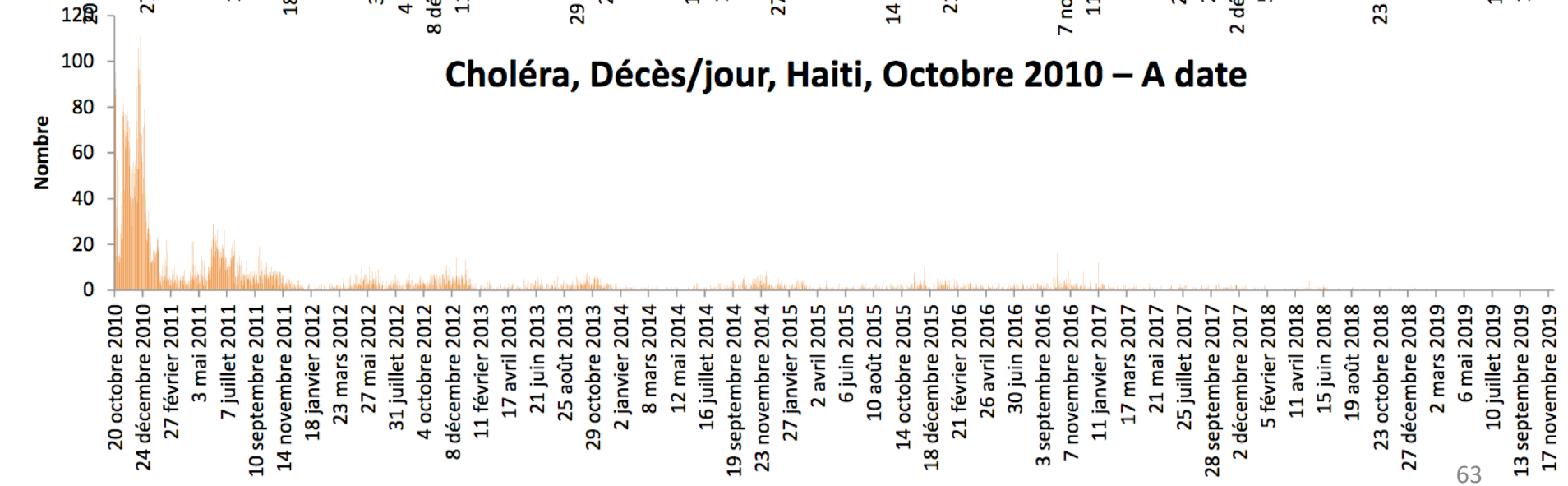
WHO predicts 2% of total Haitian population to be infected: 200000 ca.

The actual course of the disease



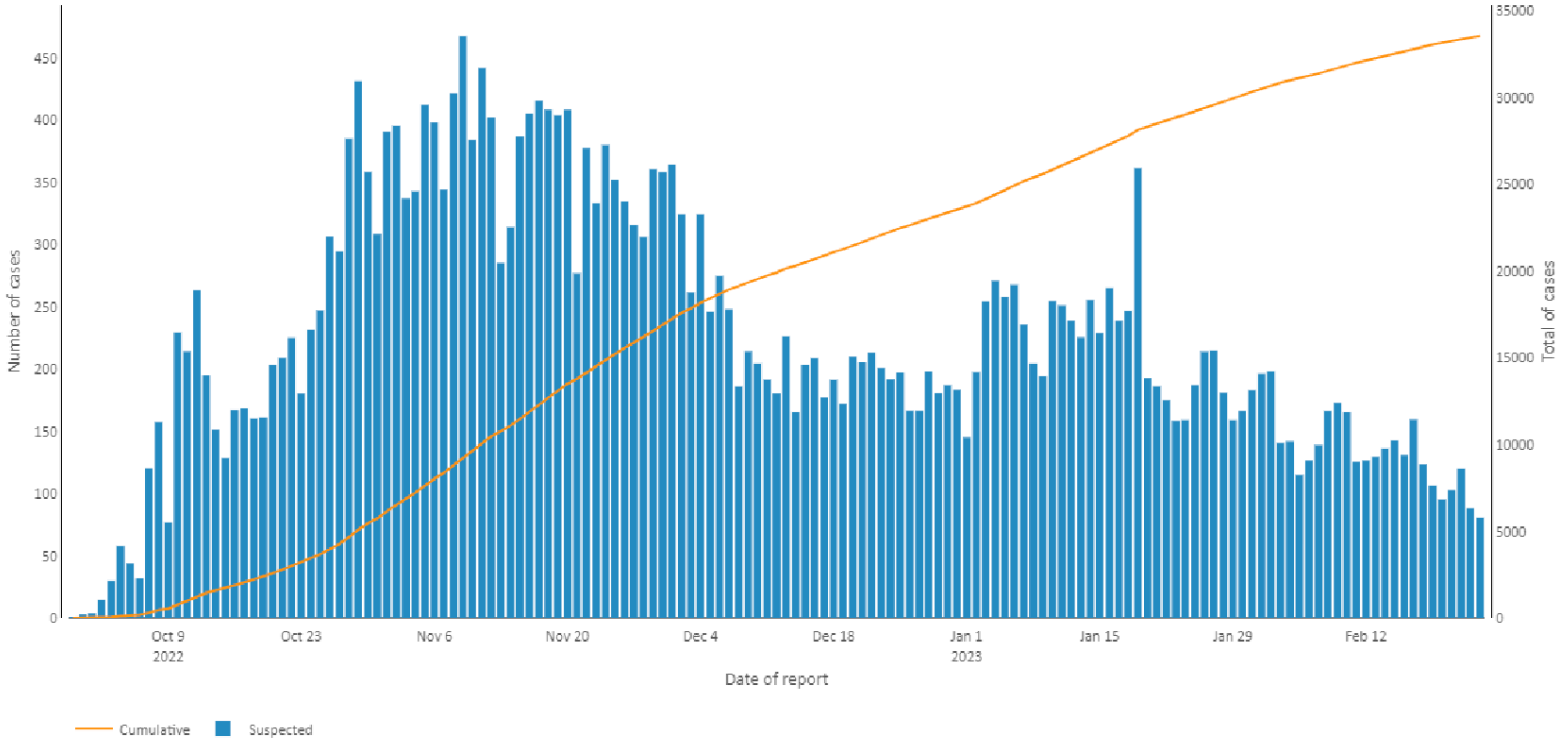
Vue Globale , Haïti depuis 2010





Recent resurgence

Figure 1. Daily distribution of suspected cases of cholera in Haiti from 29 September 2022 to 24 February 2023

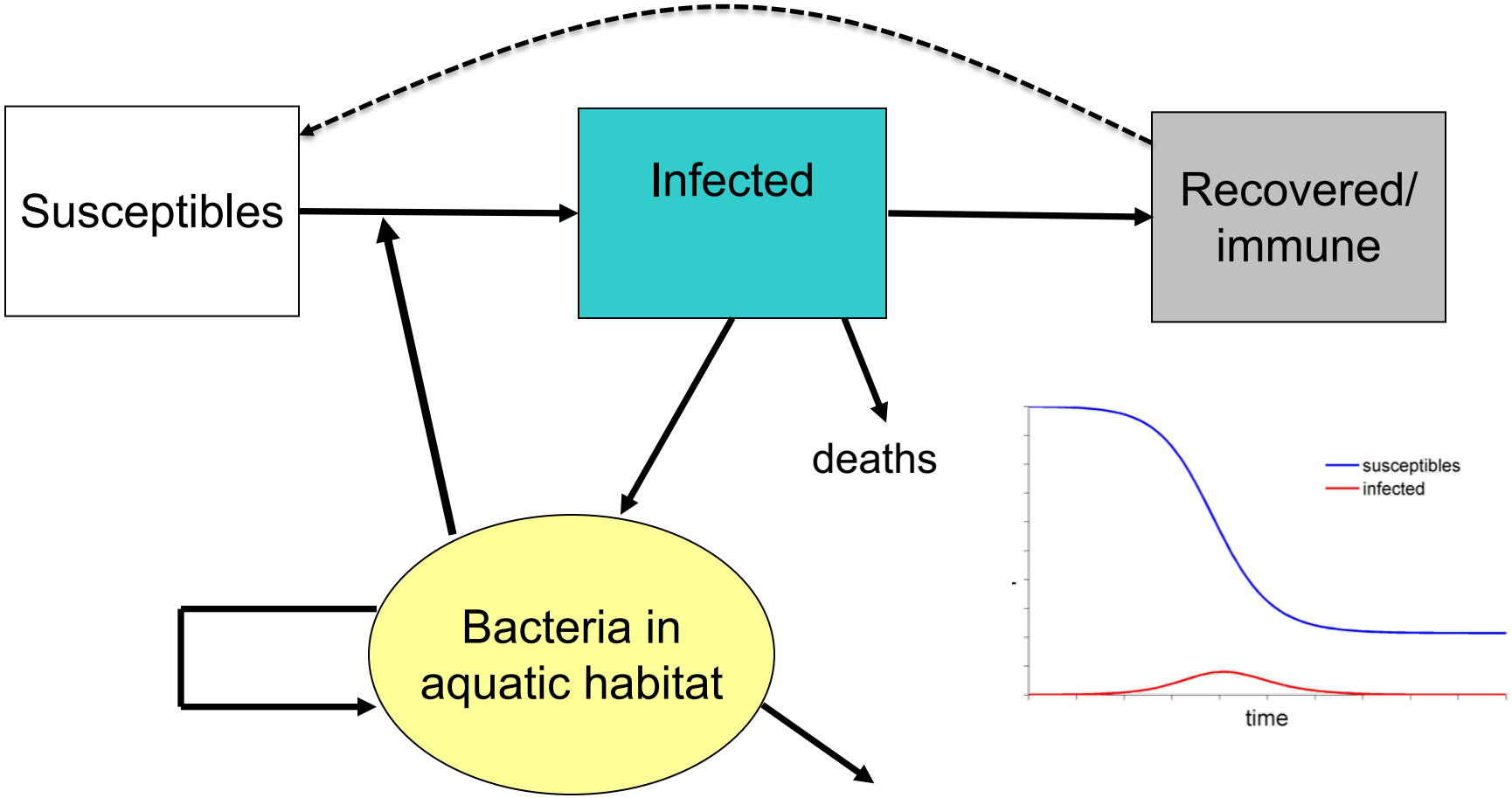


Source: Haïti Ministère de la Santé Publique et de la Population (MSPP). Data generated by PAHO/WHO.

The basic ecoepidemiological model

Capasso, V. & Paveri Fontana, S. 1979 Mathematical model for the 1973 cholera epidemic in the European Mediterranean region. *Rev. Epidemiol. Santé Publique* 27, 121–132.

Codeço, C. T., 2001 Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir. *BMC Infectious Diseases* 1, 1.



The basic water-borne disease model

$$\frac{dS}{dt} = \mu(H - S) - \beta BS$$ Susceptibles

$$\frac{dI}{dt} = \beta BS - (\mu + \alpha + \rho)I$$ Infected

$$\frac{dB}{dt} = \theta I - \delta B$$ Bacteria (concentration in water)

$\boldsymbol{\rho}$ = recovery rate $\boldsymbol{\theta}$ = contamination rate
$\boldsymbol{\delta}$ = bacteria mortality rate

Simplifying assumptions: permanent immunity, demography close to carrying capacity *H* (reservoir, constant flow μH of newborns)

The basic water-borne disease model

$$\bar{S} = \frac{\delta\phi'}{\beta\theta}, \quad \bar{I} = \frac{\mu}{\phi'}\left(H - \frac{\delta\phi'}{\beta\theta}\right), \quad \bar{B} = \frac{\mu\theta}{\phi'\delta}\left(H - \frac{\delta\phi'}{\beta\theta}\right)$$

$$\phi' = \mu + \alpha + \rho$$

Nontrivial equilibrium

Nontrivial equilibrium is feasible only if $H > \frac{\delta\phi'}{\beta\theta}$

$$R_0 = \frac{\beta\vartheta H}{(\mu+\alpha+\rho)\delta}$$

Basic reproduction number > 1
Water-borne disease can become endemic in the population

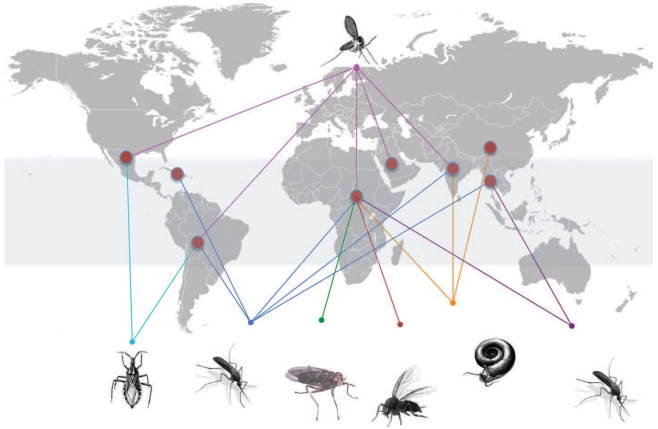
$1/(\mu+\alpha+\rho)$ = mean residence time in the infectious compartment
$1/\delta$ = mean residence time of bacteria in water
ϑ = bacteria excreted by one infectious per unit time
βH = No. of susceptibles infected per unit time per bacterium in a disease free population

Vector-borne diseases

Propagules are transmitted from one host to another via a second host species, the vector (e.g. mosquito)	**Malaria (anopheles mosquito), dengue (tiger mosquito), zika (Yellow Fever mosquito, *Aedes aegypti*)**

In 2021 malaria caused an estimated 247 million cases globally, and resulted in 619,000 deaths.
Dengue causes an estimated 96 million symptomatic cases and an estimated 40,000 deaths every year.
Some diseases are water-related

Aedes aegypti
Tiger mosquito



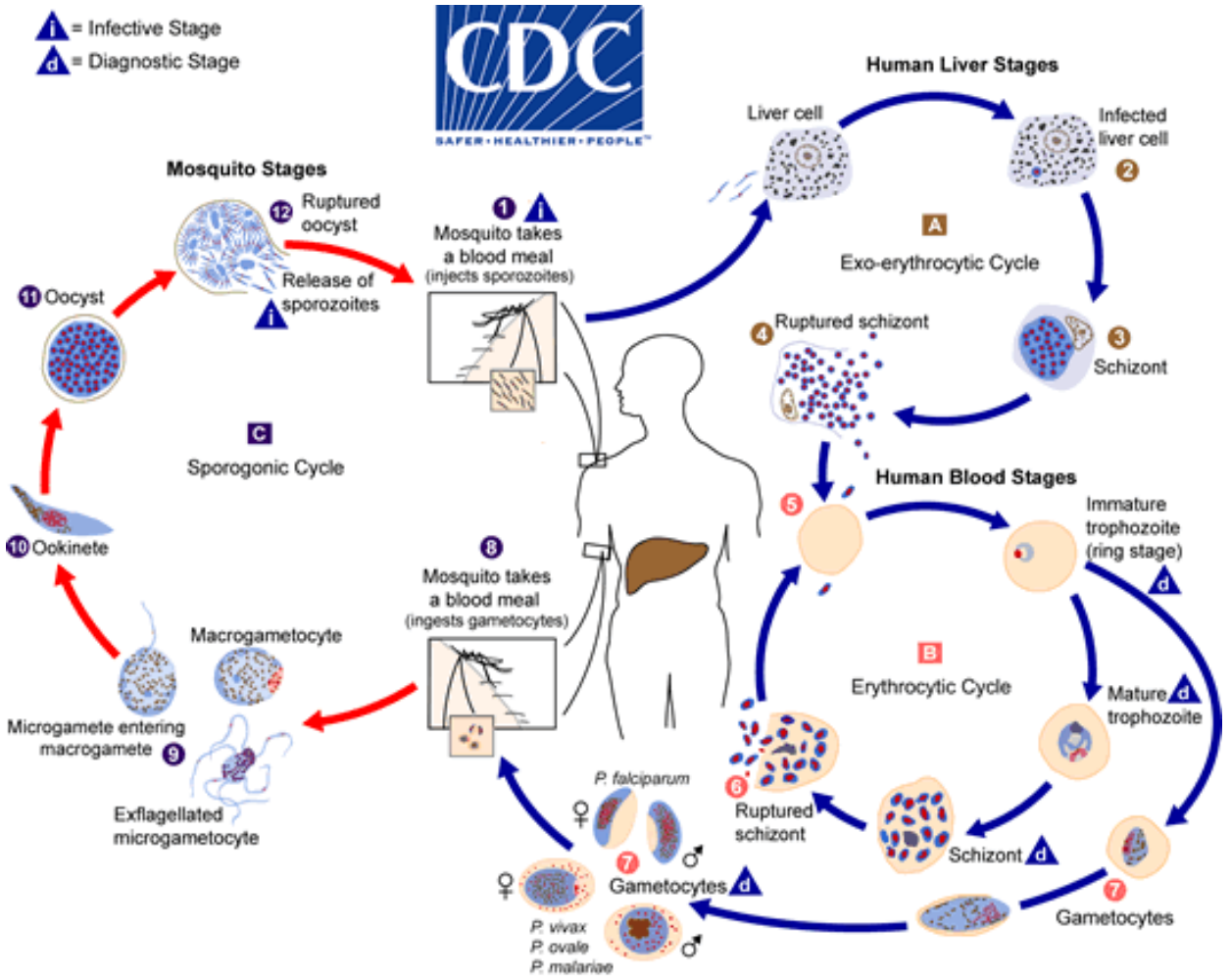
Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae, Plasmodium ovale	Malaria	Anopheline mosquito	Africa, Asia, South America
Trypanosoma cruzi	Chagas disease	Kissing/ Assasin bugs (Triatomine bugs)	South America, Southern USA
Trypanosoma brucei	Sleeping sickness	Tsetse flies (*Glossina*)	East and west sub-Saharan Africa
Leishmania major, Leishmania mexica, Leishmania donovani, Leishmania infantum, Leishmania brasiliensis	Cutaneous and visceral Leishmaniasis	Sandflies (*Lutzomyia, Phlebotomus*)	
Schistosoma mansoni, Schistosoma japonicum	Bilharziosis	Freshwater snails, *Biomphalaria, Bulinus Oncomelania*	Africa, Asia, South America
Brugia malayi, Brugia timori, Wurcheraria bancrofti	Lymphatic filariasis		Africa, Asia
Onchocerca volvulus	River blindness	Blackflies (*Simulium*)	Sub Saharan Africa

Keith R. Matthews *Science* **331**, 1149 (2011)

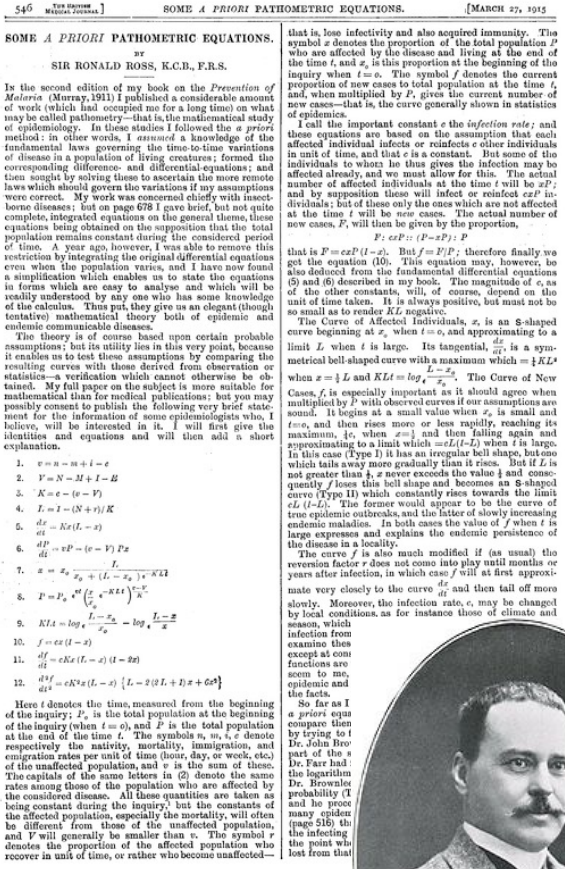
Malaria parasite life cycle

Plasmodium falciparum

Anopheles stephensi



Understanding malaria



546 [THE BRITISH MEDICAL JOURNAL] SOME *A PRIORI* PATHOMETRIC EQUATIONS. [MARCH 27, 1915

SOME *A PRIORI* PATHOMETRIC EQUATIONS.

BY

SIR RONALD ROSS, K.C.B., F.R.S.

Is the second edition of my book on the *Prevention of Malaria* (Murray, 1911) I published a considerable amount of work (which had occupied me for a long time) on what may be called pathometry—that is, the mathematical study of epidemiology. In these studies I followed the *a priori* method: in other words, I *assumed* a knowledge of the fundamental laws governing the time-to-time variations of disease in a population of living creatures; formed the corresponding difference- and differential-equations; and then sought by solving these to ascertain the more remote laws which should govern the variations if my assumptions were correct. My work was concerned chiefly with insect-borne diseases; but on page 678 I gave brief, but not quite complete, integrated equations on the general theme, these equations being obtained on the supposition that the total population remains constant during the considered period of time. A year ago, however, I was able to remove this restriction by integrating the original differential equations even when the population varies, and I have now found a simplification which enables us to state the equations in forms which are easy to analyse and which will be readily understood by any one who has some knowledge of the calculus. Thus put, they give us an elegant (though tentative) mathematical theory both of epidemic and endemic communicable diseases.

The theory is of course based upon certain probable assumptions; but its utility lies in this very point, because it enables us to test these assumptions by comparing the resulting curves with those derived from observation or statistics—a verification which cannot otherwise be obtained. My full paper on the subject is more suitable for mathematical than for medical publications; but you may possibly consent to publish the following very brief statement for the information of some epidemiologists who, I believe, will be interested in it. I will first give the identities and equations and will then add a short explanation.

1. $v = n - m + i - e$
2. $V = N - M + I - E$
3. $K = e - (v - V)$
4. $L = I - (N + r)/K$
5. $\frac{dx}{dt} = Kx(L - x)$
6. $\frac{dP}{dt} = vP - (v - V)Px$
7. $x = x_0 \frac{L}{x_0 + (L - x_0)\epsilon^{-KLt}}$
8. $P = P_0 \epsilon^{vt}\left(\frac{x}{x_0}\epsilon^{-KLt}\right)^{\frac{v-V}{K}}$
9. $KLt = \log_\epsilon \frac{L - x_0}{x_0} - \log_\epsilon \frac{L - x}{x}$
10. $f = cx(L - x)$
11. $\frac{df}{dt} = cKx(L - x)(L - 2x)$
12. $\frac{d^2f}{dt^2} = cK^2x(L - x)\{L - 2(2L + 1)x + 6x^2\}$

Here t denotes the time, measured from the beginning of the inquiry; P_0 is the total population at the beginning of the inquiry (when $t = o$), and P is the total population at the end of the time t. The symbols n, m, i, e denote respectively the nativity, mortality, immigration, and emigration rates per unit of time (hour, day, or week, etc.) of the unaffected population, and v is the sum of these. The capitals of the same letters in (2) denote the same rates among those of the population who are affected by the considered disease. All these quantities are taken as being constant during the inquiry, but the constants of the affected population, especially the mortality, will often be different from those of the unaffected population, and V will generally be smaller than v. The symbol r denotes the proportion of the affected population who recover in unit of time, or rather who become unaffected—that is, lose infectivity and also acquired immunity. The symbol x denotes the proportion of the total population P who are affected by the disease and living at the end of the time t, and x_0 is this proportion at the beginning of the inquiry when $t = o$. The symbol f denotes the current proportion of new cases to total population at the time t, and, when multiplied by P, gives the current number of new cases—that is, the curve generally shown in statistics of epidemics.

I call the important constant c the *infection rate;* and these equations are based on the assumption that each affected individual infects or reinfects c other individuals in unit of time, and that c is a constant. But some of the individuals to whom he thus gives the infection may be affected already, and we must allow for this. The actual number of affected individuals at the time t will be xP; and by supposition these will infect or reinfect cxP individuals; but of these only the ones which are not affected at the time t will be *new* cases. The actual number of new cases, F, will then be given by the proportion,

$$F : cxP :: (P - xP) : P$$

that is $F = cxP(I - x)$. But $f = F/P$; therefore finally we get the equation (10). This equation may, however, be also deduced from the fundamental differential equations (5) and (6) described in my book. The magnitude of c, as of the other constants, will, of course, depend on the unit of time taken. It is always positive, but must not be so small as to render KL negative.

The Curve of Affected Individuals, x, is an S-shaped curve beginning at x_0 when $t = o$, and approximating to a limit L when t is large. Its tangential, $\frac{dx}{dt}$, is a symmetrical bell-shaped curve with a maximum which $= \frac{1}{4}KL^2$ when $x = \frac{1}{2}L$ and $KLt = \log_\epsilon \frac{L - x_0}{x_0}$. The Curve of New Cases, f, is especially important as it should agree when multiplied by P with observed curves if our assumptions are sound. It begins at a small value when x_0 is small and $t = o$, and then rises more or less rapidly, reaching its maximum, $\frac{1}{4}c$, when $x = \frac{1}{2}$ and then falling again and approximating to a limit which $= cL(1 - L)$ when t is large. In this case (Type I) it has an irregular bell shape, but one which tails away more gradually than it rises. But if L is not greater than $\frac{1}{2}$, x never exceeds the value $\frac{1}{2}$ and consequently f loses this bell shape and becomes an S-shaped curve (Type II) which constantly rises towards the limit $cL(1 - L)$. The former would appear to be the curve of true epidemic outbreaks, and the latter of slowly increasing endemic maladies. In both cases the value of f when t is large expresses and explains the endemic persistence of the disease in a locality.

The curve f is also much modified if (as usual) the reversion factor r does not come into play until months or years after infection, in which case f will at first approximate very closely to the curve $\frac{dx}{dt}$ and then tail off more slowly. Moreover, the infection rate, c, may be changed by local conditions, as for instance those of climate and season, which

infection from
examine thes
except at com
functions are
seem to me,
epidemic and
the facts.

So far as I
a priori equa
compare then
by trying to t
Dr. John Bro
part of the s
Dr. Farr had
the logarithm
Dr. Brownlo
probability (T
and he proce
many epiden
(page 516) th
the infecting
the point wh
lost from that

Ronald Ross

Giovanni Battista Grassi

Anopheles stephensi

Malaria models

Table 4

Basic malaria models - (a) Ross Model, (b) Macdonald Model, and (c) Anderson-May Model, with corresponding basic reproductive number (R_0) and parameter descriptions

Models	R_0	Parameters and their values [12,36,40,44,56]
(a) **Ross model** [36] $\frac{dI_h}{dt} = a\,b\,m\,I_m\,(1 - I_h) - r\,I_h$ $\frac{dI_m}{dt} = a\,c\,I_h\,(1 - I_m) - \mu_2\,I_m$	$\frac{m a^2 b c}{r \mu_2}$	a : Man biting rate [0.01-0.5 day^{-1}] b : Proportion of bites that produce infection in human [0.2-0.5]
(b) **Macdonald model** [40] $\frac{dI_h}{dt} = a\,b\,m\,I_m(1 - I_h) - r\,I_h$ $\frac{dE_m}{dt} = a\,c\,I_h(1 - E_m - I_m)$ $- a\,c\,I_h(t - \tau_m)\,[1 - E_m(t - \tau_m) - I_m(t - \tau_m)]\,e^{-\mu_2 \tau_m} - \mu_2 E_m$ $\frac{dI_m}{dt} = a\,c\,I_h(t - \tau_m)\,[1 - E_m(t - \tau_m) - I_m(t - \tau_m)]\,e^{-\mu_2 \tau_m} - \mu_2 I_m$	$\frac{m a^2 b c}{r \mu_2} e^{-\mu_2 \tau_m}$	c : Proportion of bites by which one susceptible mosquito becomes infected [0.5] m : Ratio of number of female mosquitoes to that of humans [0.5-40] r : Average recovery rate of human [0.005-0.05 day^{-1}]
(c) **Anderson and May model** [12] $\frac{dE_h(t)}{dt} = a\,b\,m\,I_m(t)(1 - E_h(t) - I_h(t))$ $- abmI_m(t - \tau_h)\,[1 - E_h(t - \tau_h) - I_h(t - \tau_h)]\,e^{-(r+\mu_1)\tau_h}$ $- r\,E_h(t) - \mu_1 E_h(t)$ $\frac{dI_h(t)}{dt} = abmI_m(t - \tau_h)\,[1 - E_h(t - \tau_h) - I_h(t - \tau_h)]\,e^{-(r+\mu_1)\tau_h}$ $- r\,I_h(t) - \mu_1 I_h(t)$ $\frac{dE_m(t)}{dt} = a\,c\,I_h(t)\,[1 - E_m(t) - I_m(t)] - acI_h(t - \tau_m)[1 - E_m(t - \tau_m)$ $- I_m(t - \tau_m)]e^{-\mu_2 \tau_m} - \mu_2 E_m(t)$ $\frac{dI_m(t)}{dt} = acI_h(t - \tau_m)[1 - E_m(t - \tau_m)i$ $- I_m(t - \tau_m)]\,e^{-\mu_2 \tau_m} - \mu_2 I_m(t)$	$\frac{m a^2 b c}{r \mu_2} e^{-\mu_2 \tau_m} e^{-\mu_1 \tau_h}$	μ_1: Per capita rate of human mortality [0.017 year^{-1}] μ_2: Per capita rate of mosquito mortality [0.05-0.5 day^{-1}] τ_m: Latent period of mosquito [5-15 days] τ_h: Latent period of human [10-100 day]

Basic Ross model

$$\frac{dU}{dt} = \beta M(1-U) - \gamma U$$

U = prevalence of infected humans

$$\frac{dM}{dt} = \psi U(1-M) - \xi M$$

M = prevalence of infected mosquitoes (females)

β = mosquito-human transmission = *m a b*
γ = recovery of humans
ψ = human-mosquito transmission = *a c*
ξ = mortality of infected mosquitoes
m = No. female mosquitoes per human
a = No. bites per mosquito per unit time
b, c = probabilities of transmission per bite

$$R_0 = \frac{\beta\psi}{\gamma\xi} = \frac{ma^2bc}{\gamma\xi}$$

Basic reproduction number

$dU/dt = 0 \quad M = \gamma U/\beta(1-U)$

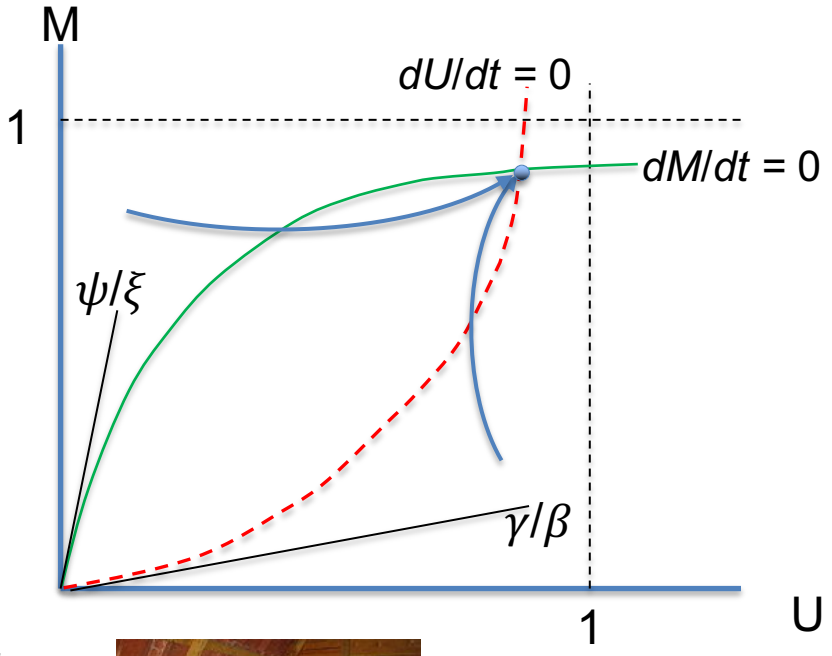
$dM/dt = 0 \quad M = \psi U/(\psi U+\xi)$

$$\bar{U} = \frac{\beta\psi - \gamma\xi}{\psi(\beta+\gamma)}, \qquad \bar{M} = \frac{\beta\psi - \gamma\xi}{\beta(\psi+\xi)}$$

Equil. feasible if $\beta\psi > \gamma\xi$, i.e. $\psi/\xi > \gamma/\beta$

$R_0 > 1$

$$R_0 = \frac{\beta\psi}{\gamma\xi} = \frac{ma^2bc}{\gamma\xi}$$



β = mosquito-human transmission = $m\ a\ b$

γ = recovery of humans

ψ = human-mosquito transmission = $a\ c$

ξ = mortality of infected mosquitoes

m = No. female mosquitoes per human

a = No. bites per mosquito per unit time

b, c = probabilities of transmission per bite

Bed nets

Basic reproduction number

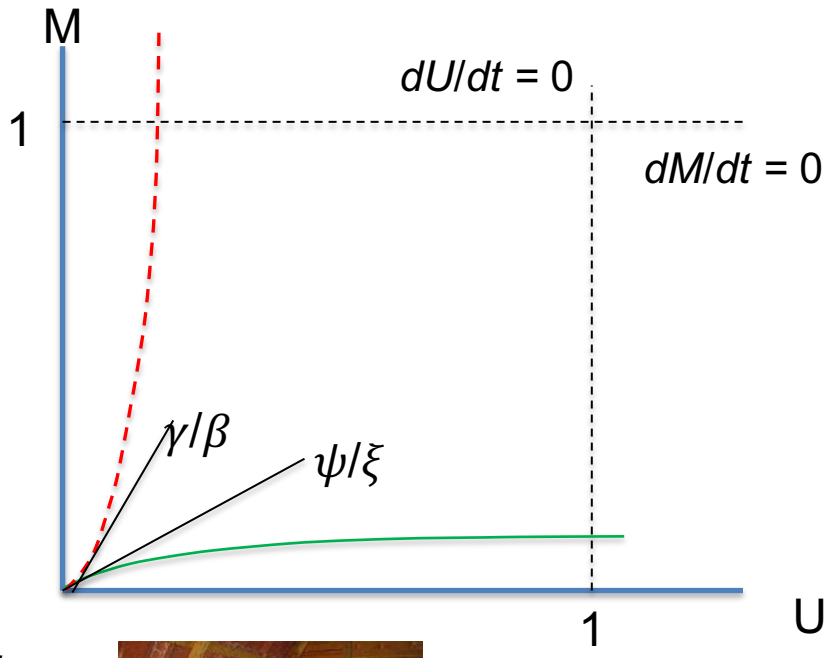
$dU/dt = 0 \quad M = \gamma U/\beta(1-U)$

$dM/dt = 0 \quad M = \psi U/(\psi U+\xi)$

$$\bar{U} = \frac{\beta\psi - \gamma\xi}{\psi(\beta+\gamma)}, \qquad \bar{M} = \frac{\beta\psi - \gamma\xi}{\beta(\psi+\xi)}$$

Equil. feasible if $\beta\psi > \gamma\xi$, i.e. $R_0 > 1$

$$R_0 = \frac{\beta\psi}{\gamma\xi} = \frac{ma^2bc}{\gamma\xi}$$



β = mosquito-human transmission = $m\ a\ b$

γ = recovery of humans

ψ = human-mosquito transmission = $a\ c$

ξ = mortality of infected mosquitoes

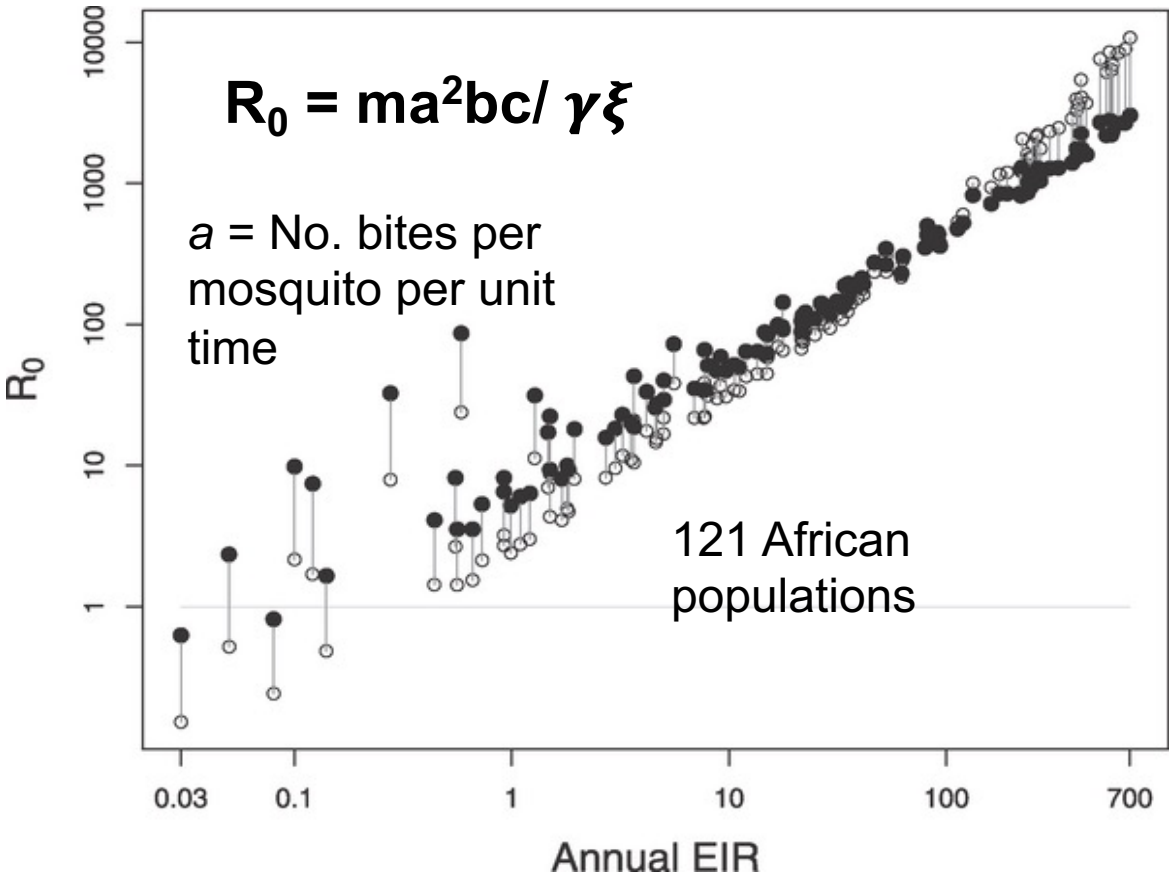
m = No. female mosquitoes per human

a = No. bites per mosquito per unit time

b, c = probabilities of transmission per bite

Bed nets

Figure 2. R0 Estimates for 121 African Populations

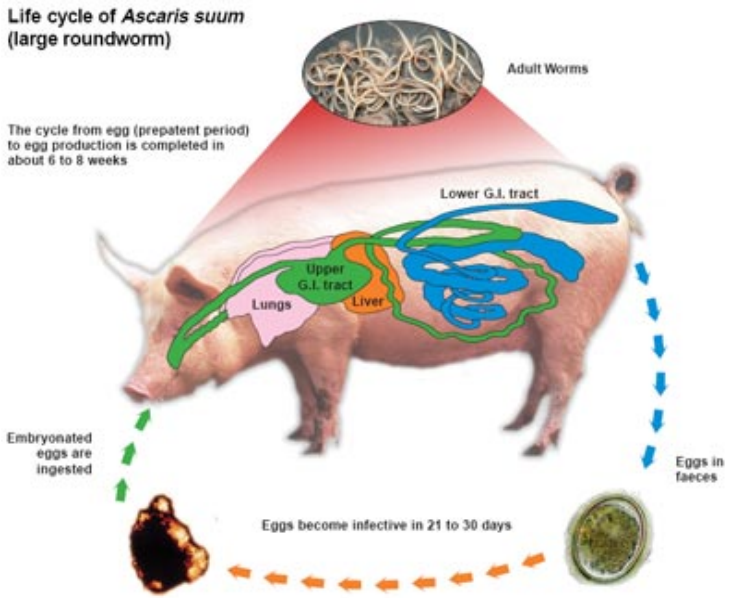


entomological inoculation rate (EIR) = average No. of infectious bites per person per year

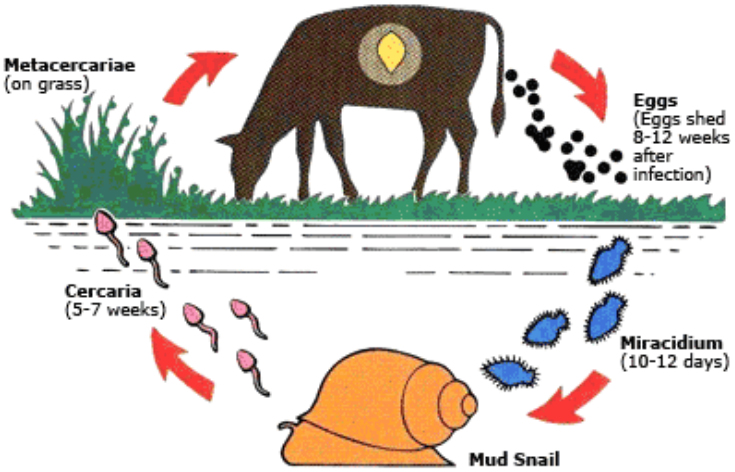
Smith DL, McKenzie FE, Snow RW, Hay SI (2007) Revisiting the Basic Reproductive Number for Malaria and Its Implications for Malaria Control. PLOS Biology 5(3): e42. doi:10.1371/journal.pbio.0050042
http://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.0050042

PLOS | BIOLOGY

Life cycles in macroparasites



Simple cycle of the large roundworm, a nematode



Life cycle of *Fasciolopsis buski*, a trematode, the agent responsible for fasciolopsiasis, an example of cycle with an intermediate host

Distribution of macroparasite burden

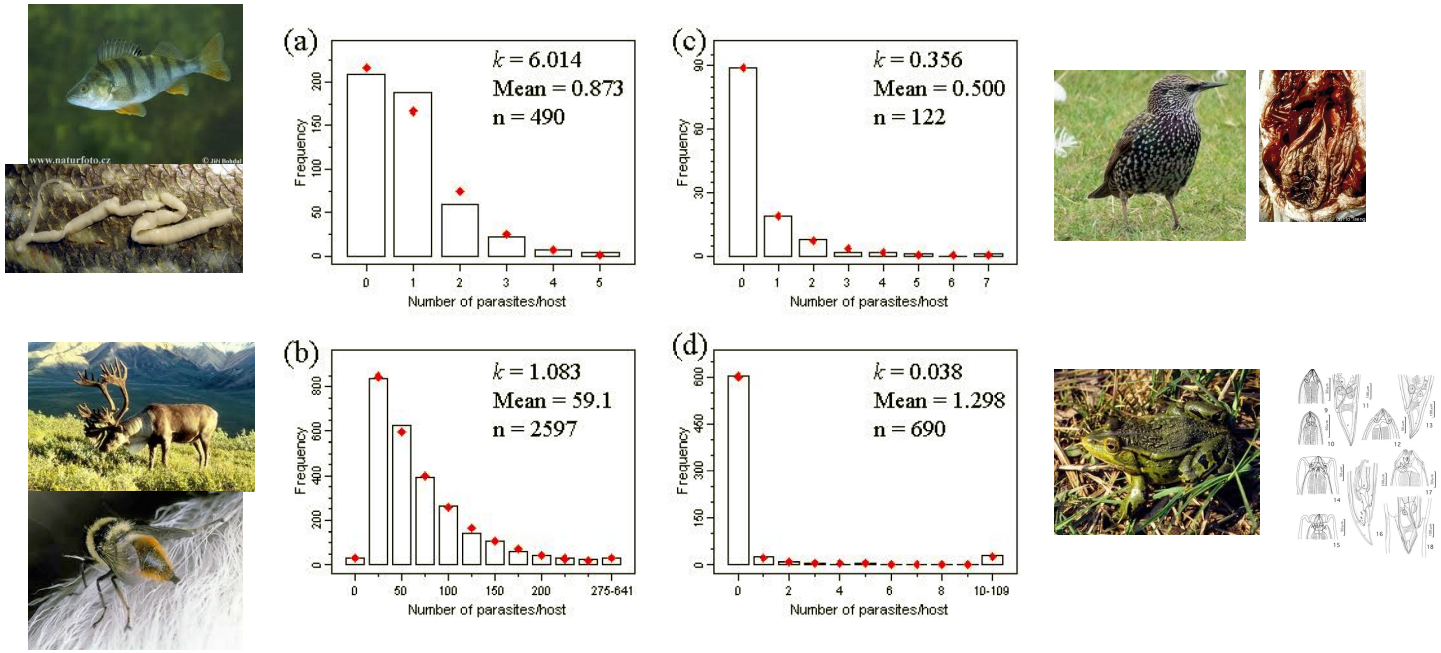


Figure 2.1. Observed parasite frequency distributions for four host-parasite interactions (after Shaw *et al.* 1998). In all cases, the bars represent the observed frequency distributions and the points are the fit of the negative binomial distribution. (a) host = perch *Perca fluviatilis*, parasite = tapeworm *Triaenophorus nodulosus*; (b) reindeer *Rangifer tarandus,* warble fly *Hypoderma tarandi*; (c) common starling *Sturnus vulgaris,* nematode *Porrocaecum ensicaudatum*; (d) pond frog *Rana nigromaculata,* nematode *Spiroxys japonica* (For reference sources see Shaw *et al.* 1998).

Distribution of macroparasite burden

Binomial distribution
- Sequence of Bernoulli events (0 or 1)
- *p* = probability of 1's
- Probability of drawing *r* 1's in *n* trials

$$f(r;n,p)=\binom{n}{r}p^r(1-p)^{n-r}$$

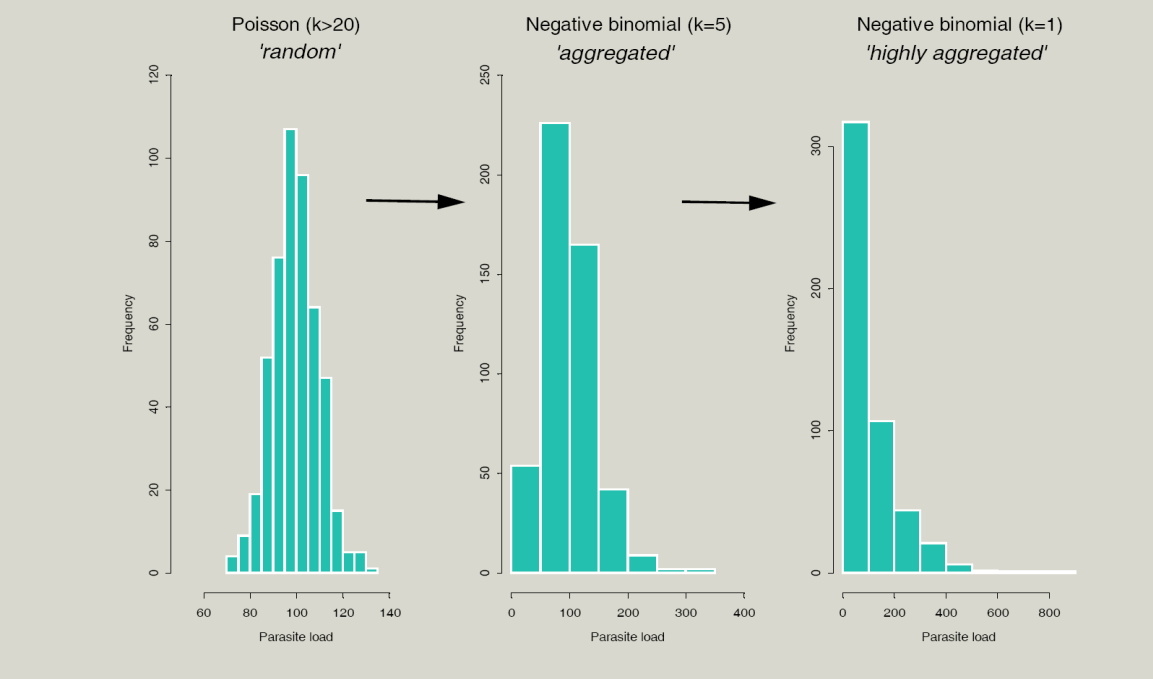
- Mean = E[*r*] = *np* Variance = *np*(1-*p*) ≤ Mean Underdispersion

Poisson distribution
- Let *p* −> 0, *n* −>∞ with *np* = λ; then

$$f(r;\lambda)=\frac{\lambda^r e^{-\lambda}}{r!}$$ Poisson distribution

- Mean = E[*r*] = *λ* Variance = *λ* = Mean

Negative binomial and *clumping*



•f(*i*;*k*,p): probability of the number *i* of successes in a sequence of independent and identically distributed Bernoulli trials (with parameter p) before a specified (non-random) number of failures (denoted *k*) occurs.

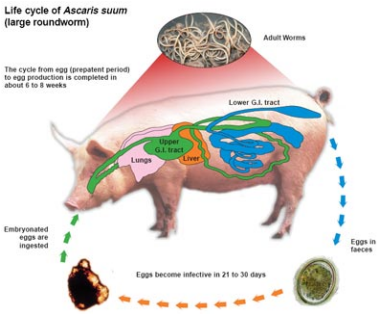
•Mean = p*k*/(1-p)

•Variance = Mean + Mean^2/*k* Overdispersion

•*k* = clumping parameter

The host-macroparasite model

H = host number (or density)
P = adult parasite number (or density)
L = number or density of parasite free-living stages (larvae)



$$\frac{dH}{dt} = (\nu - \mu)H - \sum_{i=0}^{\infty} \alpha i p_i H$$

$$\frac{dP}{dt} = \beta L H - mP - \sum_{i=0}^{\infty} (\mu + \alpha i) i p_i H$$

ν-μ = host birth (death) rate m = parasite natural death rate
α = additional mortality caused by 1 parasite
β = infection rate of hosts
p_i = proportion of hosts harboring *i* parasites

Calculating the parasite load

$$\sum_{i=0}^{\infty} \alpha i p_i H = \alpha H \sum_{i=0}^{\infty} i p_i \qquad \sum_{i=0}^{\infty} (\mu + \alpha i) i p_i H = \mu H \sum_{i=0}^{\infty} i p_i + \alpha H \sum_{i=0}^{\infty} i^2 p_i$$

p_i = proportion of hosts harboring *i* parasites
P = total number of parasites
H = total number of hosts
P/H = average parasite load

$$\frac{P}{H} = \sum_{i=0}^{\infty} i p_i = Mean$$

Assume distribution of parasites is negative binomial

$$\sum_{i=0}^{\infty} i^2 p_i = Mean^2 + Variance =$$

$$= Mean^2 + Mean + \frac{Mean^2}{k} = \frac{P}{H} + \frac{k+1}{k}\frac{P^2}{H^2}$$

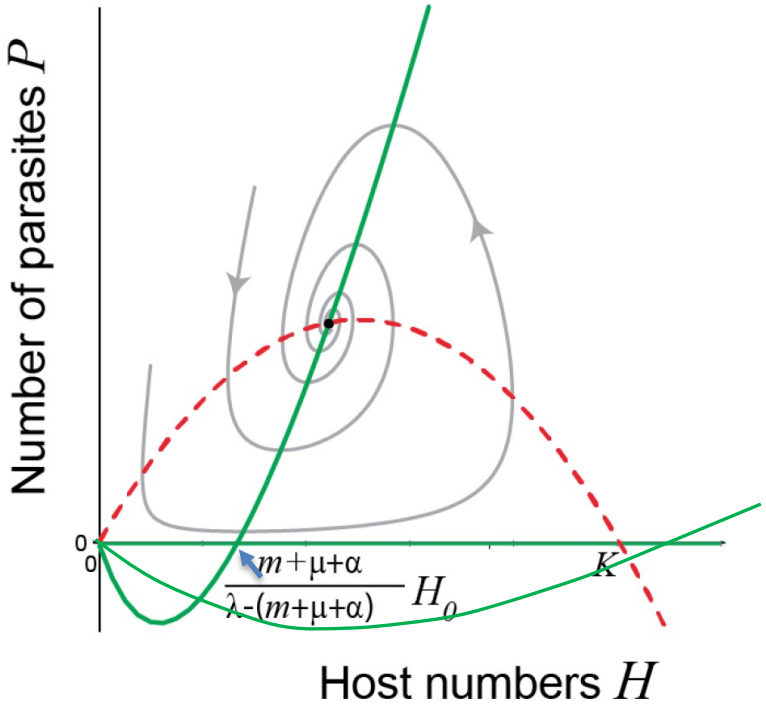
Anderson & May's model

Assume $L = \theta P/(H+H_0)$, logistic growth of the host and $\beta\theta = \lambda$

$$\frac{dH}{dt} = rH\left(1-\frac{H}{K}\right)-\alpha P$$

$$\frac{dP}{dt} = \frac{\lambda PH}{H+H_0}-(m+\mu+\alpha)P-\alpha\frac{k+1}{k}\frac{P^2}{H}$$

The basic reproduction number



$$R_0 = \frac{\lambda K}{H_0 + K} \frac{1}{m + \mu + \alpha}$$

R_0 = average number of secondary infections caused by one primary infections in a healthy population at carrying capacity = basic reproduction number

R_0 = 1 marks a transition

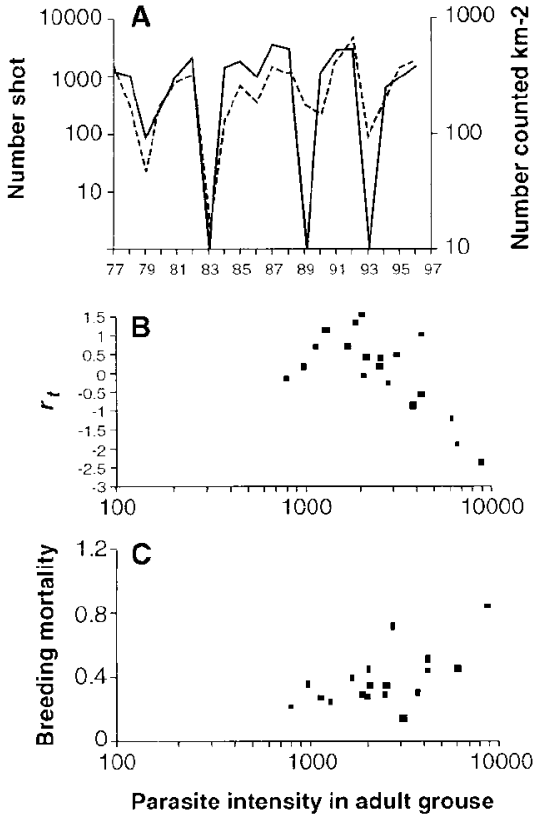
R_0 < 1 , DFE (H=*K*, P=*0*) is stable

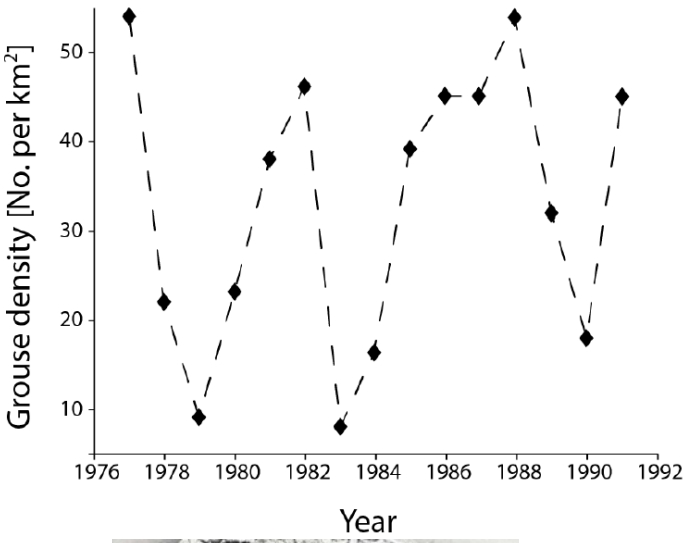
R_0 > 1 , endemic disease equilibrium is stable

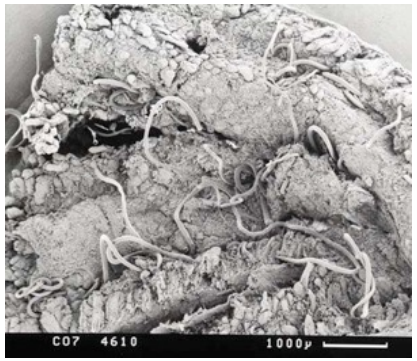
$$K > \frac{m + \mu + \alpha}{\lambda - (m + \mu + \alpha)} H_0$$

Parasites affecting reproductive success

Red grouse(*Lagopus lagopus scoticus*)

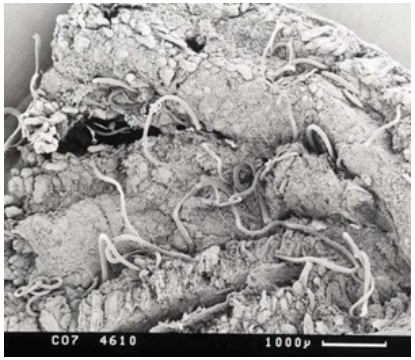




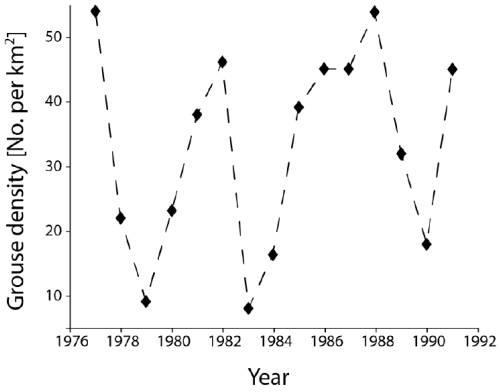


Trichostrongylus tenuis

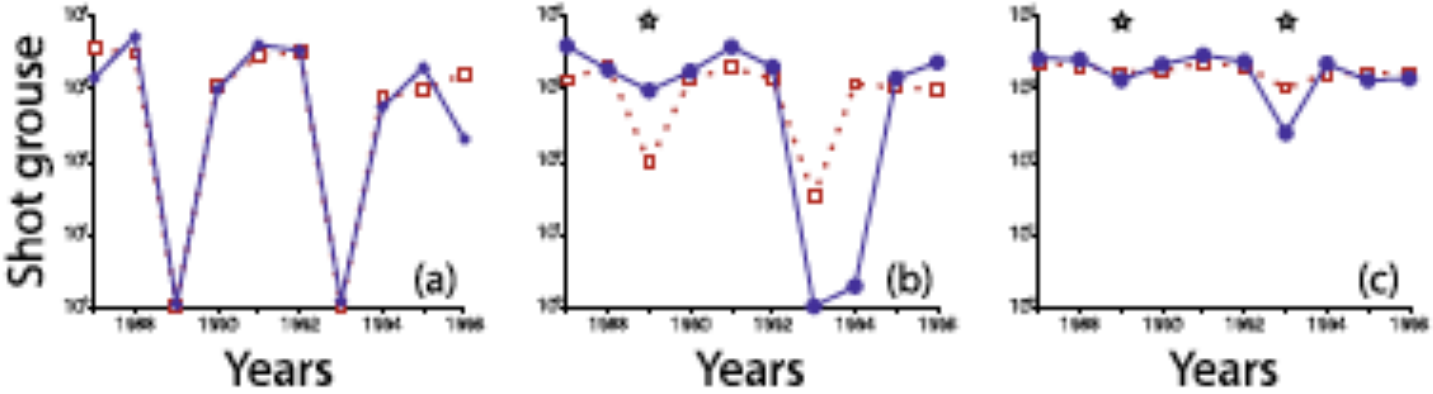
Parasites affecting reproductive success



Trichostrongylus tenuis



Untreated

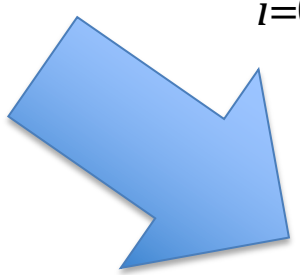


Treated

Parasites affecting fertility

$$\frac{dH}{dt} = (\nu - \sum_{i=0}^{\infty} \varepsilon i p_i - \mu) H$$

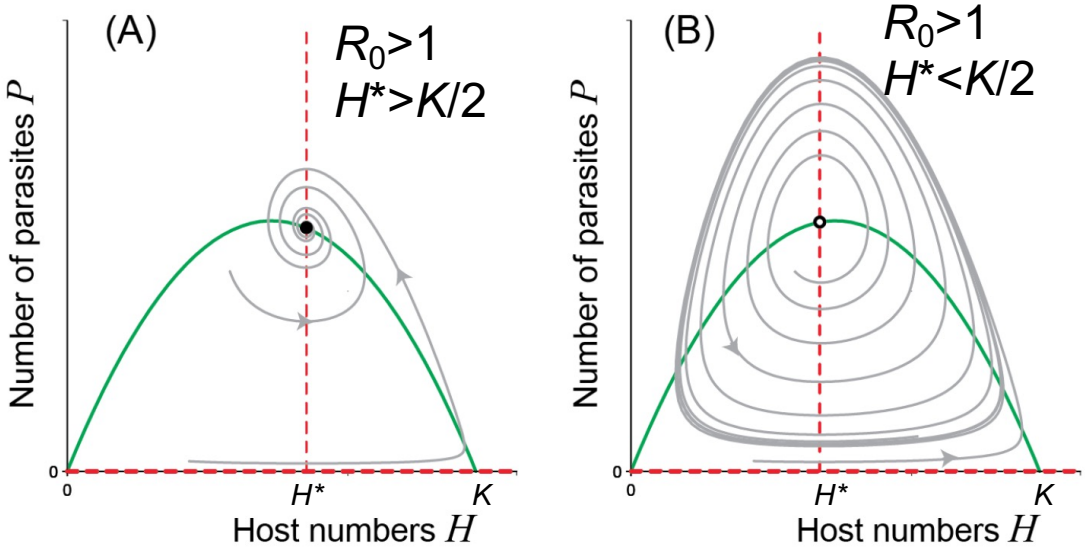
$$\frac{dP}{dt} = \beta L H - mP - \sum_{i=0}^{\infty} \mu i p_i H$$



$$\frac{dH}{dt} = rH\left(1 - \frac{H}{K}\right) - \varepsilon P$$

$$\frac{dP}{dt} = \frac{\lambda P H}{H + H_0} - (m + \mu) P$$

Permanent oscillations



$$R_0 = \frac{\lambda K}{H_0 + K} \frac{1}{m + \mu}$$

$$H^* = \frac{m + \mu}{\lambda - m - \mu} H_0$$

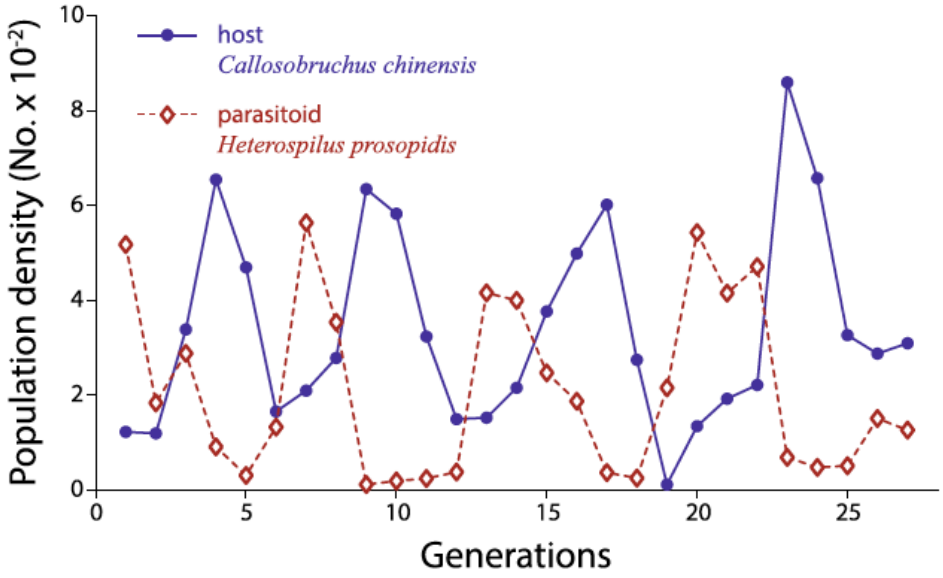
H^* does not depend on carrying capacity K

$R_0 = 1$ at $H^* = K$

$H^* < K/2 \rightarrow R_0 > 1$ and oscillations

For increasing parasite fertility $\lambda \rightarrow R_0$ increasing, H^* decreasing

Parasitoids



Pest beetle
Callosobruchus chinensis
Bean weevil

Parasitoid wasp
Heterospilus prosopidis

Parasitoids and biological control

Drosophila suzukii

- Invasive fruit fly
- Infests undamaged soft shelled fruit (cherry, berries etc) (Poyet et al., 2015)
- Large harvest losses in new habitat, including Trentino (De Ros et al., 2013; Wiman et al., 2014)

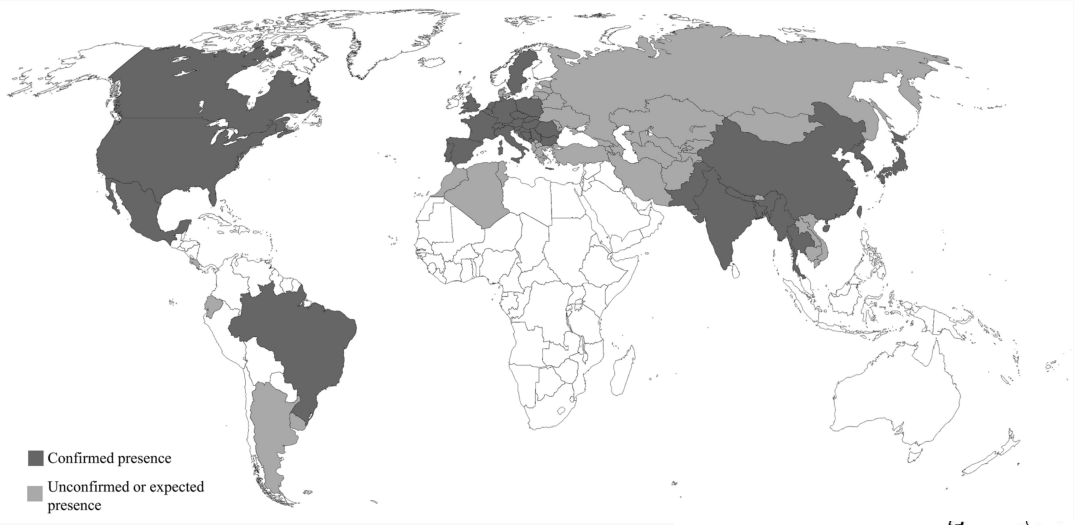
Serrated ovipositor

D. suzukii male

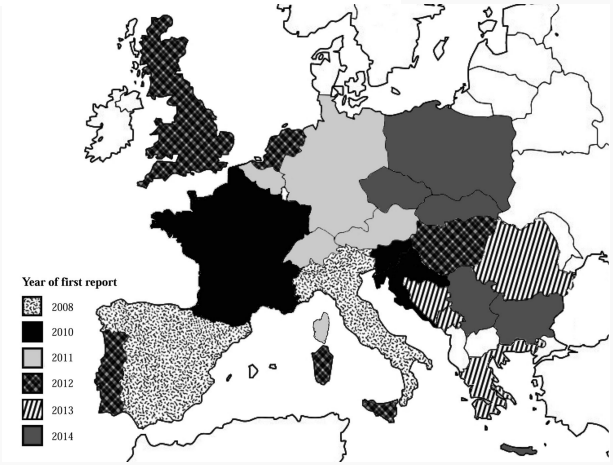
Damaged fruit

pictures: G. Arakelian/Dept. of Agriculture, Los Angeles County; Martin Hauser, California Department of Food and Agriculture; http://www.falw.vu.nl

Area of origin and invasion



- *Drosophila suzukii* native to east asia (Kanzawa, 1935)
- Arrived in 2008/2009 to Europe and America (Lee et al., 2011)
- Wide ecological range, rapid expansion (Rota-Stabelli et al., 2013)



maps: (Asplen et al., 2015)

Control

- Insecticides problematic (Cini et al., 2012)
- Possibly biological control with resident or introduced parasitoid (Rossi Stacconi et al., 2015; Daane et al., 2016)

Trichopria drosophilae (pupal parasitoid)

Leptopilina heterotoma (larval parasitoid)

pictures: http://www.bioplanet.eu; (Lue et al., 2016)

Nicholson & Bailey's host-parasitoid model (1935)

H_k = number of hosts in generation k
P_k = number of adult parasitoids in generation k
A_k = numbers of attacked hosts in generation k

$$H_{k+1} = \lambda(H_k - A_k)$$
$$P_{k+1} = \sigma A_k$$

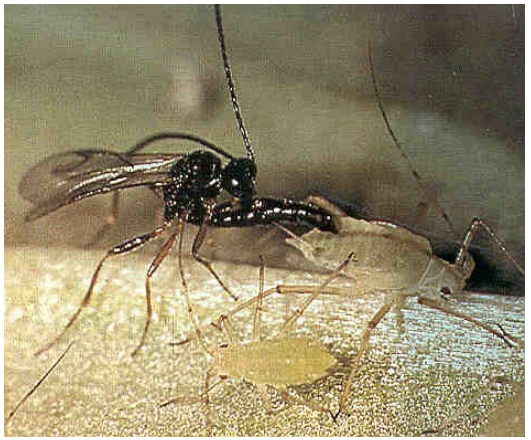
$$A_k = H_k p(T)$$

T = activity time period
p(*T*) = Prob. >1 encounter

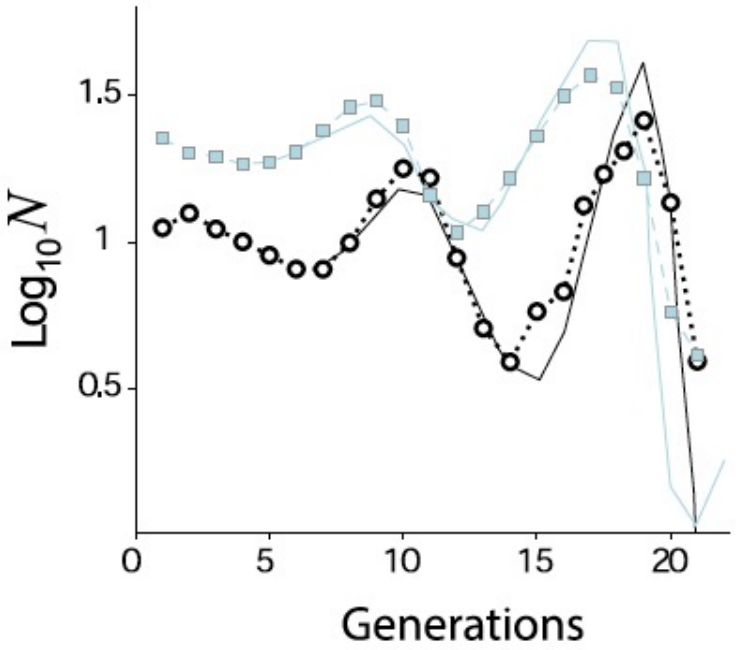
A.J. Nicholson

$$p(T) = 1 - p_0(T) = 1 - \exp(-\alpha T P_k)$$

$$H_{k+1} = \lambda H_k e^{-\beta P_k}$$
$$P_{k+1} = \sigma H_k (1 - e^{-\beta P_k}).$$



Encarsia formosa (black, ● Parasitoid) &
Trialeurodes vaporariorum (white, ■ Host)



$$\bar{H}_+ = \frac{\lambda}{\sigma\beta}\frac{\ln(\lambda)}{\lambda - 1} \qquad \bar{P}_+ = \frac{1}{\beta}\ln(\lambda)$$

Non-trivial equilibrium
UNSTABLE!