Ecology of Disease

Weekly theme: Climate

Lecture: Prediction modelling

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Why examine ecology?

Ecologists seek to understand:

- species richness
  (biodiversity)
- species abundance
  (populations/communities)
- species distribution
  (temporal, spatial)

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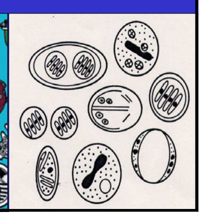
Problems with pathogens

Infectious micro-organisms have:

- diverse hosts (vertebrates, invertebrates)
- multiple infective stages (larvae-nymphs-adults)
- free-living stages (cysts-ova-eggs-larvae..)



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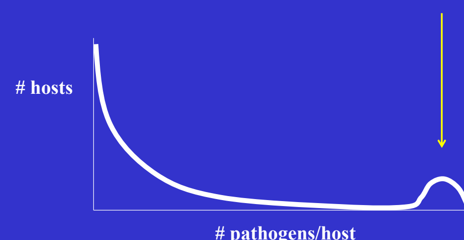
Pathogen Abundance

Degree of overdispersion determines disease

Many hosts have some pathogens → no disease
Some hosts have many pathogens → disease



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Factors affecting populations

INTERNAL environmental factors
e.g. host age, sex, behaviour, genetics, physiology, immune status

EXTERNAL environmental factors
e.g. temperature, humidity, photoperiod, salinity, hydrology, geology, flora, fauna, etc

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Epidemiology

Study of occurrence, spread and control of diseases

- Prevalence (no. infected) - point prevalence
  - period prevalence
- Incidence (change in prevalence over time)

exhibit longitudinal fluctuations (secular/periodic/seasonal)
influenced by many factors:

- demographic, socioeconomic, behavioural
- geographic, climatic

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Climate (= average weather)

Interaction of land, air, water
- temperature
- precipitation
- humidity
- solar radiation
- wind

We measure them, but can we predict them?

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Why model climate?

Seasonal forecasting
- vast agricultural opportunities (crops, pastures, animal production)
- several days/weeks/months warning
- good/bad seasons
- resource utilization/allocation
- disaster planning

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Mathematical models

Reductionist process to define:
- components parts
- interactions

Models well accepted in physical sciences, mathematics, engineering

Slow incorporation into biological sciences

> too many components and interactions

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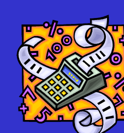
Purpose

Prediction models for:
- disease incidence
- disease distribution



Model impact of change in:
- environment (climate)
- activity (intervention)

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Climate forecasting

<u>Oceanic</u> flux

<u>El Nino</u> (EN) known since 1726
- occurs every 2-10 years (average 4)
- warm surface water in W equatorial Pacific (Peru)
- mortality of fish/guano birds crippling local economy
- slackening of westward trade winds
- droughts in Australia and Africa
- reduced Indian monsoon, cyclones in Polynesia
- counter-balanced by La Nina

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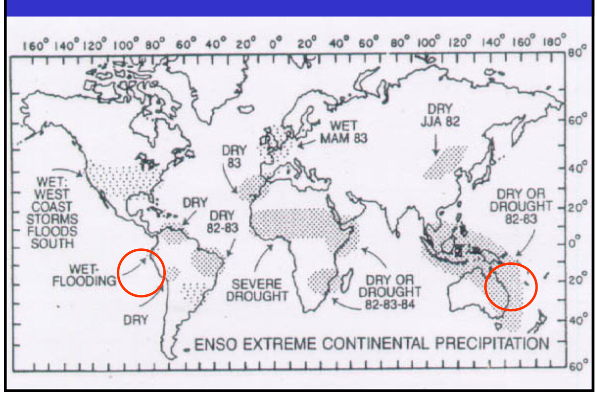
El Nino polarity



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Climate forecasting

Atmospheric flux

Southern Oscillation (SO) known since 1904

- occurs every 2-10 years (average 4)
- sea level atmospheric pressure swings between South America and India-Australia
- heavy rainfall in Pacific
- drought in India
- warm winters in Canada

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Climate forecasting

Oceanic-Atmospheric Correlation

El Nino-Southern Oscillation (ENSO)

- connection between EN and SO made in 1960's
- warming of Pacific over 1/4 circumference of earth
- correlation between:
  - sea surface temperature in equatorial Pacific (90-150°W, 5°S-5°N)
  - negative of SO index (sea level pressure difference between Tahiti and Darwin)

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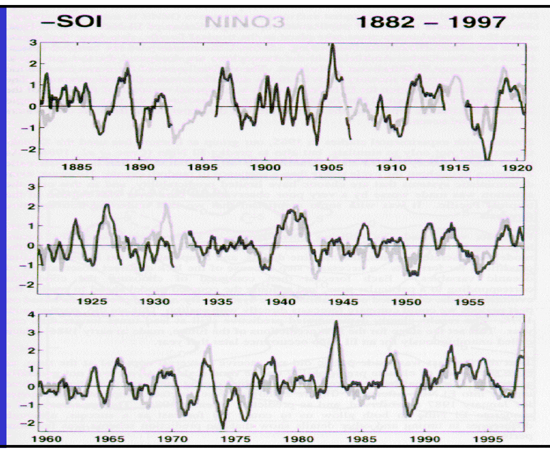

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ENSO

ENSO parameters included in models to predict distribution of:

- arthropod diseases (cattle tick, fly strike)
- vector-borne diseases (arbo viruses, tick fever)
- helminths (nematodes, trematodes)
- protozoa (enteric, tissues)
- water-borne diseases

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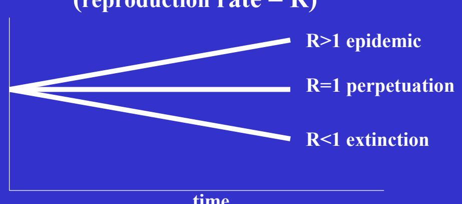
Modelling principles

Pathogens must reproduce themselves to survive (reproduction rate = R)



Basic (case) reproduction rate (Ro)
= average number of secondary infections resulting from primary case in susceptible population

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Ro estimation

BUT not all individuals in population are susceptible

effective reproductive rate (R) = Ro.S

where S = fraction of population susceptible

At equilibrium R = 1 therefore Ro = 1/S

can estimate S by serological surveys

if S is low (eg. 20% seronegative), Ro is high (1/0.2) to maintain equilibrium
if S is high (eg. 80% seronegative), Ro is low (1/0.8) to maintain equilibrium

Remember: Ro is a measure of transmission potential (determines the spread of infection)

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Ro manipulation

Objective of vaccination and other control programs is to reduce S (fraction susceptible) to reduce Ro (↓spread)

BUT spread dependent on population density

$Ro = \sigma.\beta.D$

where σ = density of susceptibles
β = transmission coefficient
D = average duration of infectivity

For infection to take hold, Ro must be >1
therefore, critical value for $\sigma_T = 1/\beta.D$

This is the target for mass vaccination programs
(must reduce the density of susceptibles below σ_T)

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BUT, nothing is IDEAL

Population varies in susceptibility

- proportion just infected (latent)
- proportion patent (infectious)
- proportion immune (resistant)

⇒ All influence Ro

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SEIR model (long-term immunity)

Identify four states in population	Movement shown by differential equations
S = susceptible	dS/dt
E = latently infected	dE/dt
I = infectious	dI/dt
R = resistant	dR/dt

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Relevant parameters

N= total population = S+E+I+R

b = probability of transmission

G = duration of latency
g = 1/G = rate of leaving latency

D = duration of infectious state
d = 1/D = rate of leaving infectious state

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SEIR model

S = susceptible	$dS/dt = -bSI$
E = latently infected	$dE/dt = bSI - gE$
I = infectious	$dI/dt = gE - dI$
R = resistant	$dR/dt = dI$

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Add vital dynamics

m = recruitment rate (birth rate/death rate)

S	$dS/dt = mN - bSI - mS$
E	$dE/dt = bSI - gE - mE$
I	$dI/dt = gE - dI - mI$
R	$dR/dt = dI - mR$

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Reproduction number

Assuming
- law of mass action (equal likelihood of encounter)
- no thresholds (no min # required to support epidemic)

$$Ro = bgN/(d+m)(g+m)$$

but duration of latent (1/g) and infectious state (1/d) usually very small compared to length of life (1/m), so

$$Ro = bN/d$$

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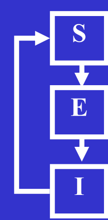
No R state for vectors

SEI vector model
no long-term immunity - short life span



$$dS/dt = -bSI + dI$$

$$dE/dt = bSI - gE$$

$$dI/dt = gE - dI$$

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Vector-borne diseases

must consider two interacting systems
e.g. malaria

<u>human host</u>	<u>mosquito vector</u>
- asexual reproduction	- sexual reproduction
- intermediate hosts	- definitive hosts
- persistent infection	- seasonal abundance
- no effective immunity (no R state)	- infected for life (no R state)

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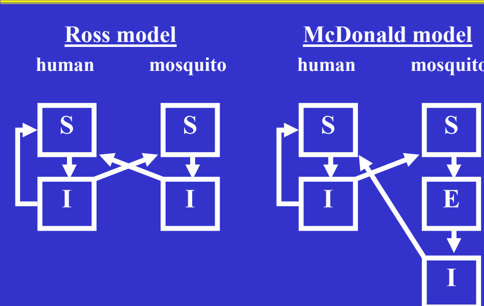
Combination models



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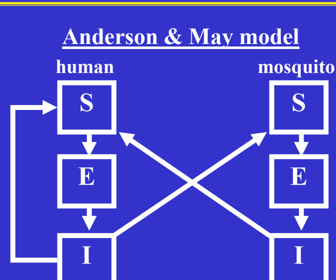
Combination models



$$Ro = \{ma^2bc[\exp(-p_h tau_h - p_m tau_m)]\}/p_g p_m$$

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Applications

Model effects of:
- treatment (chemotherapy, vaccination ..)
- control (insecticides, management ..)
- intervention (rural development, dams ..)
- migration (refugee influx, war, famine ..)
- urbanization (industry, demographics ..)
- natural phenomena (droughts, floods ..)
- climate change (global warming ..)

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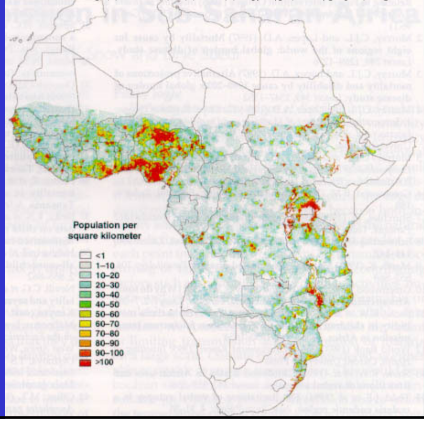
Africa malaria distribution



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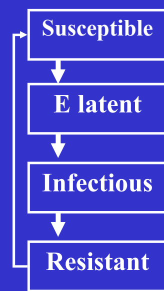
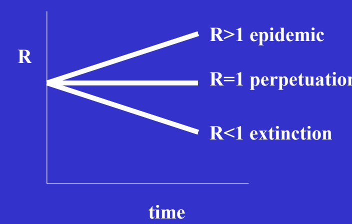
What's examinable?

Population states



Reproduction rate (Ro)



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