

Environmentally Transmitted Pathogens

Models

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A few models of Cvjetanović

- A tetanus model

- A cholera model

A model of Capasso for ETP

A model for zoonotic transmission of waterborne disease

A few models of schistosomiasis

- A first model of Woolhouse

- A second model of Woolhouse – Latency

- A third model of Woolhouse – Heterogeneous contacts

- Spatial aspects – Cholera in Haiti

- ▶ Cvjetanović, Grab, Uemura & World Health Organization. Dynamics of acute bacterial diseases : epidemiological models and their application in public health. World Health Organization (1978)
- ▶ Briscoe. On the use of simple analytic mathematical models of communicable diseases. *International Journal of Epidemiology* **9**(3) (1980)

Models of (Branko) Cvjetanović are in discrete time and quite detailed on the epi side

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Tetanus

Bacterial infection caused by *Clostridium tetani*

Spores everywhere in the environment, including soil, dust, and manure

Spores develop into bacteria when they enter the body

Not spread P2P (so not a classic model)

Incubation period usually 3-21 days (average 8 days). Can range from 1 day to several months, depending on the kind of wound. Most cases occur within 14 days

1 to 2 in 10 cases are fatal

People of all ages need TETANUS VACCINES



DTaP

for young children

- ✓ 2, 4, and 6 months
- ✓ 15 through 18 months
- ✓ 4 through 6 years

Tdap

for preteens

- ✓ 11 through 12 years

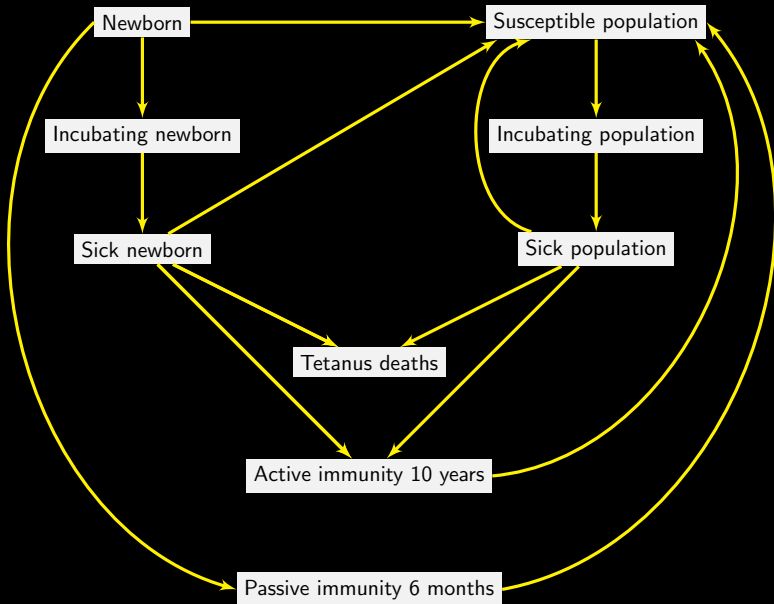
Td or Tdap

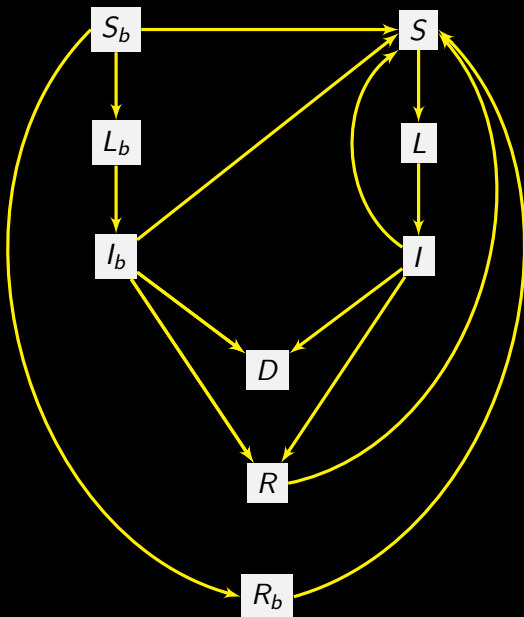
for adults

- ✓ Every 10 years

www.cdc.gov/tetanus







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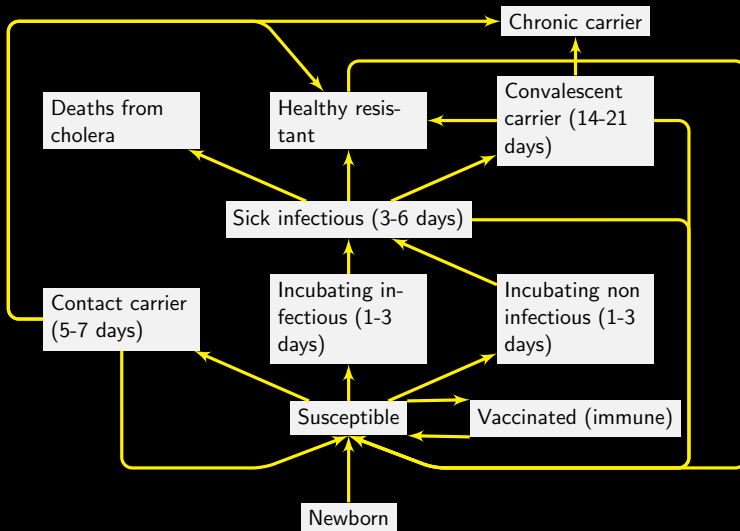
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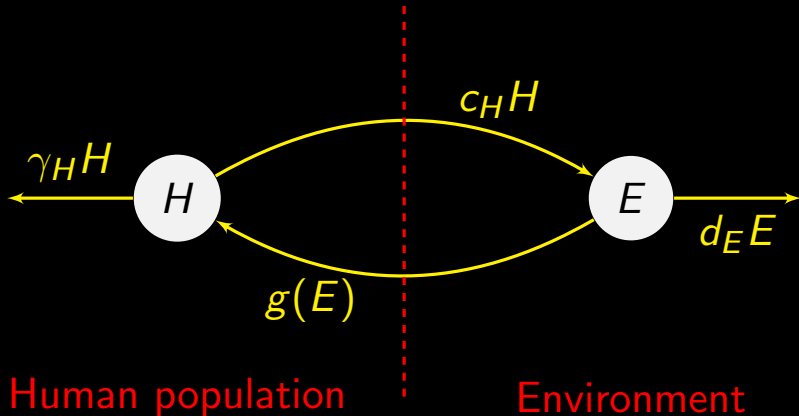
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A minimal model of V. Capasso



$1/\gamma_H$ mean infectious period, $1/d_E$ mean lifetime of the agent in the environment, c_H growth rate of the agent due to the human population, $g(E)$ “force of infection” (I would say “incidence”) of the agent on human population

Incidence function

$$g(E) = N\beta ph(E) \quad (1)$$

where

- ▶ N total human population
- ▶ β fraction of susceptible individuals in N
- ▶ p fraction exposed to contaminated environment per unit time (“probability per unit time to have a “snack” of contaminated food”)
- ▶ $h(E)$ probability for an exposed susceptible to get the infection

Typically, we would assume p and β independent of E and H and h to be saturating

To ensure (1) satisfies these conditions, we can assume

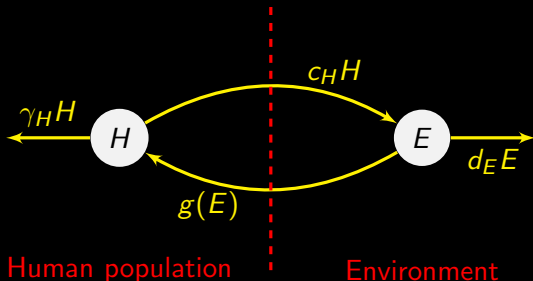
- ▶ $0 < g(e_1) < g(e_2)$ for $0 < e_1 < e_2$
- ▶ $g(0) = 0$
- ▶ $g''(z) < 0$ for all $z > 0$
- ▶ $0 < g'_+(0) < \infty$ (right derivative)
- ▶ $\lim_{z \rightarrow \infty} \frac{g(z)}{z} < \frac{d_E \gamma_H}{c_H}$

Of course, we also assume $d_E, c_H, \gamma_H > 0$

The model

$$E' = c_H H - d_E E \quad (2a)$$

$$H' = g(E) - \gamma_H H \quad (2b)$$



Pay attention to the flows..! E' does not have a $-g(E)$ and H' does not have $-c_H H$. Why?

Let

$$\mathcal{R}_0 = \frac{g'_+(0)c_H}{d_E\gamma_H} \quad (3)$$

Theorem 1

- ▶ If $0 < \mathcal{R}_0 < 1$, then (2) admits only the trivial equilibrium in the positive orthant, which is GAS
- ▶ If $\mathcal{R}_0 > 1$, then two EP exist: $(0, 0)$, which is unstable, and $z^* = (E^*, H^*)$ with $E^*, H^* > 0$, GAS in $\mathbb{R}_+^2 \setminus \{0, 0\}$

Adding a periodic component

Assume p in (1) takes the form

$$p(t) = p(t + \omega) > 0, \quad t \in \mathbb{R} \quad (4)$$

i.e., p has period ω . So we now consider the incidence

$$g(t, E) = p(t)h(E) \quad (5)$$

with h having the properties prescribed earlier. Letting

$$p_{\min} := \min_{0 \leq t \leq \omega} p(t), \quad p_{\max} := \max_{0 \leq t \leq \omega} p(t) \quad (6)$$

then we require that

$$\lim_{z \rightarrow \infty} \frac{g(z)}{z} < \frac{d_E \gamma_H}{c_H p_{\max}} \quad (7)$$

Let

$$\mathcal{R}_0^{\min} = \frac{cHP_{\min}h'_+(0)}{d_E\gamma_H}, \quad \mathcal{R}_0^{\max} = \frac{cHP_{\max}h'_+(0)}{d_E\gamma_H} \quad (8)$$

Theorem 2

- ▶ If $0 < \mathcal{R}_0^{\max} < 1$, then (2) with incidence (5) always goes to extinction
- ▶ If $\mathcal{R}_0^{\min} > 1$, then a unique nontrivial periodic endemic state exists for (2) with incidence (5)

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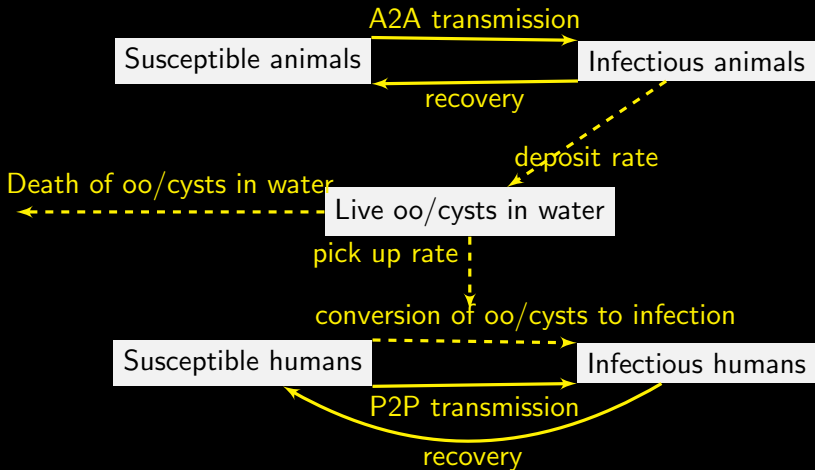
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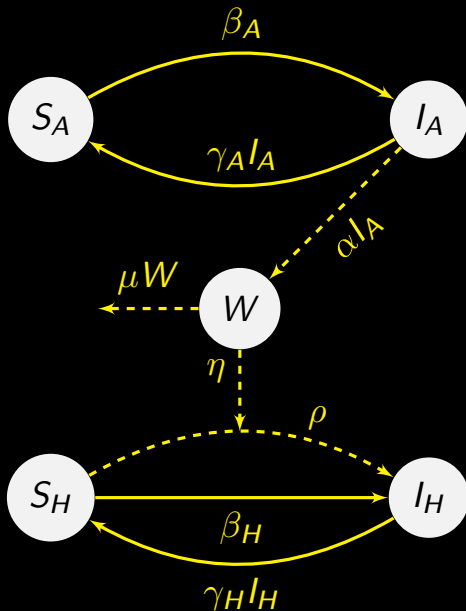
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Zoonotic transmission of waterborne disease

Waters, Hamilton, Sidhu, Sidhu, Dunbar. Zoonotic transmission of waterborne disease: a mathematical model. *Bull Math Biol* (2016)
Used for instance to model *Giardia* transmission from possums to humans





The full model

$$S'_A = -\beta_A S_A I_A + \gamma_A I_A \quad (9a)$$

$$I'_A = \beta_A S_A I_A - \gamma_A I_A \quad (9b)$$

$$W' = \alpha I_A - \eta W(S_H + I_H) - \mu W \quad (9c)$$

$$S'_H = -\rho \eta W S_H - \beta_H S_H I_H + \gamma_H I_H \quad (9d)$$

$$I'_H = \rho \eta W S_H + \beta_H S_H I_H - \gamma_H I_H \quad (9e)$$

Considered with $N_A = S_A + I_A$ and $N_H = S_H + I_H$ constant

Simplified model

Because N_A and N_H are constant, (9) can be simplified:

$$I'_A = \beta_A N_A I_A - \gamma_A I_A - \beta_A I_A^2 \quad (10a)$$

$$W' = \alpha I_A - \eta W N_H - \mu W \quad (10b)$$

$$I'_H = \rho \eta W (N_H - I_H) + \beta_H N_H I_H - \gamma_H I_H - \beta_H I_H^2 \quad (10c)$$

Three EP: DFE (0, 0, 0); endemic disease in humans because of H2H transmission; endemic in both H and A because of W

Three EP: DFE $(0, 0, 0)$; endemic disease in humans because of H2H transmission; endemic in both H and A because of W

Let

$$\mathcal{R}_{0A} = \frac{\beta_A}{\gamma_A} N_A \quad \text{and} \quad \mathcal{R}_{0H} = \frac{\beta_H}{\gamma_H} N_H \quad (11)$$

- ▶ DFE LAS if $\mathcal{R}_{0A} < 1$ and $\mathcal{R}_{0H} < 1$, unstable if $\mathcal{R}_{0A} > 1$ or $\mathcal{R}_{0H} > 1$
- ▶ If $\mathcal{R}_{0H} > 1$ and $\mathcal{R}_{0A} < 1$, (10) goes to EP with endemicity only in humans
- ▶ Endemic EP with both A and H requires $\mathcal{R}_{0A} > 1$ and $\mathcal{R}_{0H} < 1$

Note that proof is **not** global

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A model of Woolhouse

Woolhouse. On the application of mathematical models of schistosome transmission dynamics. I. Natural transmission. *Acta Tropica* **49**:241-270 (1991)

The model

Population of H individuals using a body of water containing N snails

i_H mean number of schistosomes per person and i_S the proportion of patent infections in snails (prevalence)

$$i'_H = \alpha N i_S - \gamma i_H \quad (12a)$$

$$i'_S = \beta H i_H (1 - i_S) - \mu_2 i_S \quad (12b)$$

- ▶ α number of schistosomes produced per person per infected snail per unit time
- ▶ $1/\gamma$ average life expectancy of a schistosome
- ▶ $1/\mu_2$ average life expectancy of an infected snail
- ▶ β transmission parameter

Let the basic reproductive rate for schistosomes be

$$\mathcal{R}_0 = \frac{\alpha N \beta H}{\gamma \mu_2} \quad (13)$$

(12) has two EP

- ▶ $(i_H^*, i_S^*) = (0, 0)$, LAS when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$
- ▶ $(i_H^*, i_S^*) = \left(\frac{\alpha N}{\gamma} - \frac{\mu_2}{\beta H}, 1 - \frac{1}{\mathcal{R}_0} \right)$, which only “exists” when $\mathcal{R}_0 > 1$ (and is LAS then)

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Extending the model

Interval between infection of a snail and onset of patency (release of cercariae) is *prepatent* or *latent* period

$$i'_H = \alpha N i_S - \gamma i_H \quad (14a)$$

$$\ell'_S = \beta H i_H (1 - \ell_S - i_S) - \sigma \ell_S - \mu_1 \ell_S \quad (14b)$$

$$i'_S = \sigma \ell_S - \mu_2 i_S \quad (14c)$$

- ▶ $1/\sigma$ average duration of prepatent period
- ▶ $f = \sigma/(\sigma + \mu_1)$ fraction of infected snails surviving prepatent period

The basic reproductive rate for schistosomes is now

$$\mathcal{R}_0 = f \frac{\alpha N \beta H}{\gamma \mu_2} \quad (15)$$

(14) has endemic EP

$$(i_H^*, i_S^*) = \left(\frac{\alpha N \sigma}{\gamma(\sigma + \mu_2)} - \frac{\mu_2(\sigma + \mu_1)}{\beta H(\sigma + \mu_2)}, \frac{\sigma}{\sigma + \mu_2} \left(1 - \frac{1}{\mathcal{R}_0} \right) \right)$$

Also has models

- ▶ where snails lose infectiousness (assumed to happen sometimes)
- ▶ with larval population dynamics
- ▶ single variable models
- ▶ human immigration and emigration
- ▶ reservoir hosts

Really worth a read

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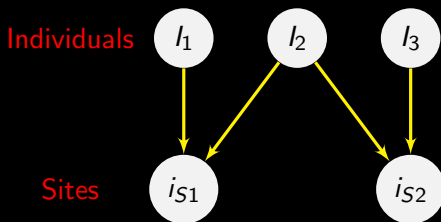
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Heterogeneities in contact rates

l_i the number of schistosomes in person $i = 1, \dots, H$ and i_{Sj} the proportion of patent infected snails in site $j = 1, \dots, L$ (L sites each supporting N snails)



l_i the number of schistosomes in person $i = 1, \dots, H$ and i_{Sj} the proportion of patent infected snails in site $j = 1, \dots, L$ (L sites each supporting N snails)

$$l'_i = \alpha \left(\sum_j \eta_{ij} N i_{Sj} \right) - \gamma l_i \quad (16a)$$

$$i'_{Sj} = \beta \left(\sum_i \eta_{ij} l_i \right) (1 - i_{Sj}) - \mu_2 i_{Sj} \quad (16b)$$

η_{ij} rate of water contact by individual i at site j

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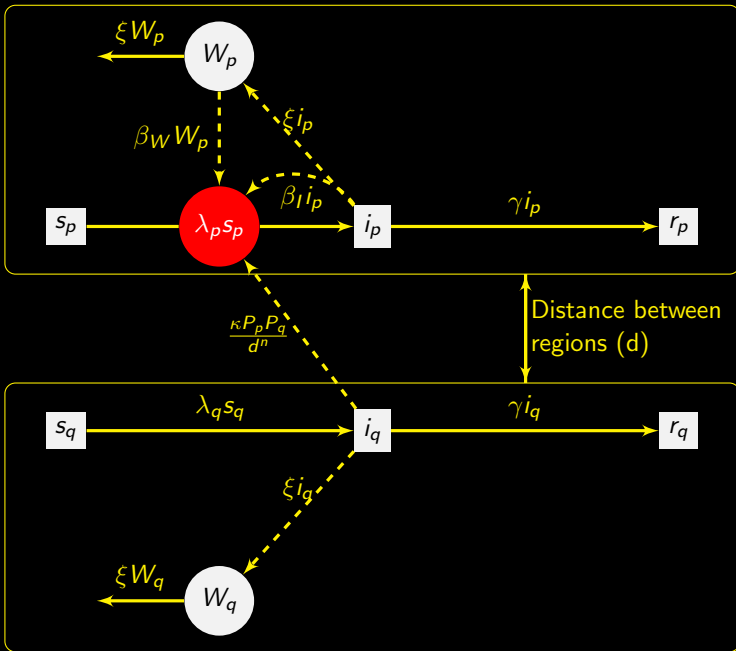
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Spatial aspects – Cholera in Haiti

Tuite, Tien, Eisenberg, Earn, Ma & Fisman. Cholera Epidemic in Haiti, 2010: Using a Transmission Model to Explain Spatial Spread of Disease and Identify Optimal Control Interventions. *Annals of Internal Medicine* **154**(9) (2011)



Metapopulation model with **implicit** movement

$$s'_p = \mu - \lambda_p s_p - \mu s_p \quad (17a)$$

$$i'_p = -\gamma i_p + \lambda_p s_p - \mu i_p \quad (17b)$$

$$r'_p = \gamma r_p - \mu r_p \quad (17c)$$

$$w'_p = \xi(i_p - w_p) \quad (17d)$$

with force of infection

$$\lambda_p = \beta_{i_p} i_p + \beta_{w_p} w_p + \sum_{q=1}^{10} \theta_{pq} i_q \quad (17e)$$

Influence of infection prevalence in q on incidence in p is gravity-type

$$\theta_{pq} = \kappa \frac{P_p P_q}{d^n}$$

