ASSIGNMENT 3

JASON THOMAS

Note: He code for Q4 is on last page

A simple SI model for chlamydia bacteria infection in Australian koala populations is,

$$\frac{dS_f}{dt} = r(S_f + \alpha I_f) - rNS_f \left(\beta_{fm} \frac{S_f I_m}{N}\right)$$

$$\frac{dI_f}{dt} = \beta_{fm} \frac{S_f I_m}{N} - rNI_f - \mu I_f$$

$$\frac{dS_m}{dt} = r(S_f + \alpha I_f) - rNS_m - \beta_{mf} \frac{S_m I_f}{N}$$

$$\frac{dI_m}{dt} = \beta_{mf} \frac{S_m I_f}{N} - rNI_m - \mu I_m$$

the impact of chlorydia infection on female hoalas

where male and female koalas are indicated by subscripts m and f, and where

$$N = S_f + I_f + S_m + I_m \,,$$

is the total number of koalas per square kilometre. At the disease-free equilibrium we have N=1 and we assume that the sex-ratio is 1:1 such that $S_m = S_f = \frac{1}{2}$.

- QI D If we note the assumption that the disease is transmitted only via female-nale intercourse, then a multipost system is necessary to account for inability for female-temple and nale-nale transmission to occur.
 - 1) A multihost disease model will help represent biological reality, since chlorydia affects the ability of tende toolus to have offspring
 - 3 A multihost disease system has the added benefit that nodellers can use a next-generation-matrix, which is sometimes preferable to a system of DEs when there is little data available.
 - The above-nentoned NaM can be used to derive a system of DEs, and vice-versa. This allows researchers to check their results (by deriving the system of DEs by another nethod, this increases confidence).
 - 1) From the Nam, we can determine the Typer Reproduction Number and this is useful as:
 - a) a neasure of the required control effort $V_{i}=1-\frac{1}{T_{i}}$ b) a confirmation that R_{0} is correct, since $T_{i}<1\Rightarrow R_{0}<1$, $T_{i}>1\Rightarrow R_{0}>1$
 - On We require [0 km²] kū = Pr (koala j survives incutation).

 Kem 0] contact rate.

 infactions period.

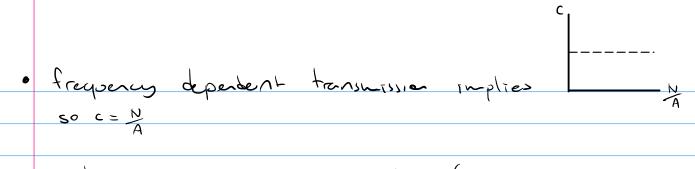
 Pr (transmission | contact)

let:

1/6 be the (average) rocubation period

1/4; be the (average) litespen of toola type;

Cis is the contact rate between seepes i and s



· in dI, rNIf-MIF= (rN-M)If (and similar for Im)

this looks similar to &I, and if I is exp. distributed then is the average inf. period. So, it is the average infections period

· Without a separate E comportment, probability of surviving incubation is assumed to be 1.

50 kj = 1. cj. v. inf. porod

= Bi. 1

ru-r

= B_{ij}

and
$$k = \begin{bmatrix}
0 & \beta_{12} \\
\beta_{21} & (N^* + \mu) \\
(N^* + \mu) & 0
\end{bmatrix}$$

Ro is equal to the dominant eigenvalue of k

A matrix has eigenales 1 (=> det (A-NI) = 0

$$\frac{\beta_{mf}}{\rho_{mh}} = \frac{(-\lambda)^{2} - \beta_{mf} \cdot \beta_{mf} \cdot (\frac{1}{\rho_{mh}})^{2}}{(-\lambda)^{2} - \beta_{mf} \cdot \beta_{mf} \cdot (\frac{1}{\rho_{mh}})^{2}} = 0$$

$$\frac{\beta_{fm}}{\rho_{mh}} = \frac{1}{\rho_{mf}} = \frac{1}{\rho_{mf}} \cdot \beta_{mf} \cdot (\frac{1}{\rho_{mh}})^{2} = 0$$

The infectors subsystem is:

$$\frac{dI_1}{dx} = \beta_{21}^* \frac{S_2 I}{N^*} - cN^* I_2 - \mu I_2^*$$

Now Inewise at the DFE, where 5=N

$$\frac{dI}{dt} = \beta_{n}^{*} I_{2}^{*} - cN^{*}I^{*} - \mu I^{*} \qquad \text{in} \quad T$$

$$\frac{dT}{dt} = P_{21}^* T_1^* - (N^*T_1^* - \mu T_2^*)$$

So
$$T = \begin{bmatrix} 0 & \beta_{12} \\ \beta_{21} & 0 \end{bmatrix}$$
, $\Sigma = \begin{bmatrix} -rN^* - \mu & 0 \\ 0 & -rN^* - \mu \end{bmatrix}$, $\chi = \begin{bmatrix} I^* \\ I^* \end{bmatrix}$

$$\sum_{i=1}^{n} \frac{1}{\sqrt{N_{i}^{2}}} = \frac{1}{(-N_{i}^{2}-1)^{2}} \left[\frac{1}{\sqrt{N_{i}^{2}+1}} \right]$$

$$= \frac{1}{\sqrt{N_{i}^{2}+1}} \left[\frac{1}{\sqrt{N_{i}^{2}+1}} \right]$$

$$-T = \begin{bmatrix} 0 - \beta_{12} \\ -\beta_{21} & 0 \end{bmatrix}$$

$$K_{-} = -T \Sigma^{-1} = \begin{bmatrix} 0 - \beta_{12} \\ -\beta_{21} \end{bmatrix} \underbrace{\begin{bmatrix} -1 \\ rN^{+}rN \end{bmatrix}}_{(-rN^{*}-r)^{2}} \underbrace{\begin{bmatrix} -\beta_{11} \\ (-rN^{*}-r)^{2} \end{bmatrix}}_{(-rN^{*}-r)^{2}} \underbrace{\begin{bmatrix} -\beta_{11} \\ (-rN^{*}-r)^{2} \end{bmatrix}}_{(-rN^{*}-r)^{2}} \underbrace{\begin{bmatrix} -\beta_{11} \\ (-rN^{*}-r)^{2} \end{bmatrix}}_{(-rN^{*}-r)^{2}}$$

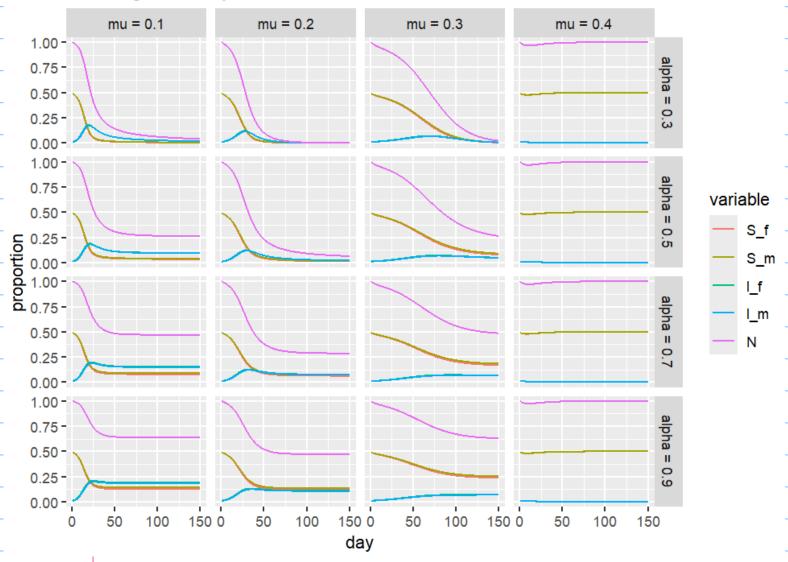
Since E here is just the 2x2 identity then $k=K_L$. The result K is almost the same as the Dam by epidentialwaical reasoning, but not quite.

Since the dragonal of t doesn't reflect buological reality (gues the assumption of transmission via hale-tende intercourse) then those terms are without meaning.

So, the result is the same as making the Nam via epidemiological reasoning.

Q4 This faceted chart shows how the system behaves given different values of a, m

For chlamydia affecting koalas with beta_mf = 1, beta_fm = 1.2, r = 0.2Assuming that initially 0.01 of males and 0.01 of females are infected



Doseration.

A lower a means that the disease affects
reproductive potential adversly. It igher values nears
the population can withstand the virus, as seen
in the chart above.

t behaves somewhat like a dampering component, if m is somewhat like a forcing component.

represents additional nortality de to intection

The effect on this system peaks at about $\mu = 0.2$. The interpretation is that it $\mu = 0.2$ then toolar die before they can infect others.

If $\mu = 0.1$, then the drop in population is sharper than at 0.3. That because fewer deaths leads to infectious toolar infecting nore toolar, rather than dying

Based on the chart, it seems these combination:

O.3 O.1 lead to extinction

O.3 O.3

O.5 O.2

Conduction

We can expect $\alpha < 0.3, \mu \simeq 0.2$ to lead to extinction