



University  
of Manitoba

## Vector-borne diseases MATH 8xyz – Lecture 10

Julien Arino  
Department of Mathematics @ University of Manitoba  
Maud Menten Institute @ PIMS  
[julien.arino@umanitoba.ca](mailto:julien.arino@umanitoba.ca)

Winter 20XX

The University of Manitoba campuses are located on original lands of Anishinaabeg, Ininew, Anisininew, Dakota and Dene peoples, and on the National Homeland of the Red River Métis.

We respect the Treaties that were made on these territories, we acknowledge the harms and mistakes of the past, and we dedicate ourselves to move forward in partnership with Indigenous communities in a spirit of Reconciliation and collaboration.

# Outline

Endemic SIRS-type models with demography

What if there's another guest at the party?

Last remarks



The background of the slide features a stylized world map with glowing yellow city lights, set against a light blue sky. Scattered across the sky are several 3D models of virus particles, which are green with red spikes and a red center.

Endemic SIRS-type models with demography

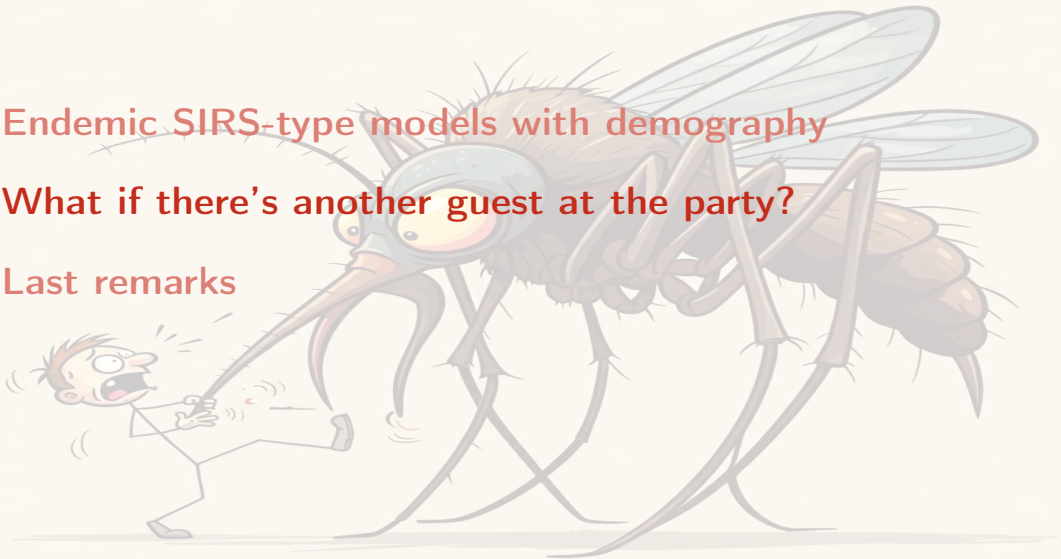
What if there's another guest at the party?

Last remarks

Endemic SIRS-type models with demography

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Last remarks



# What if there's another guest at the party?

Two Ross-Macdonald-type models

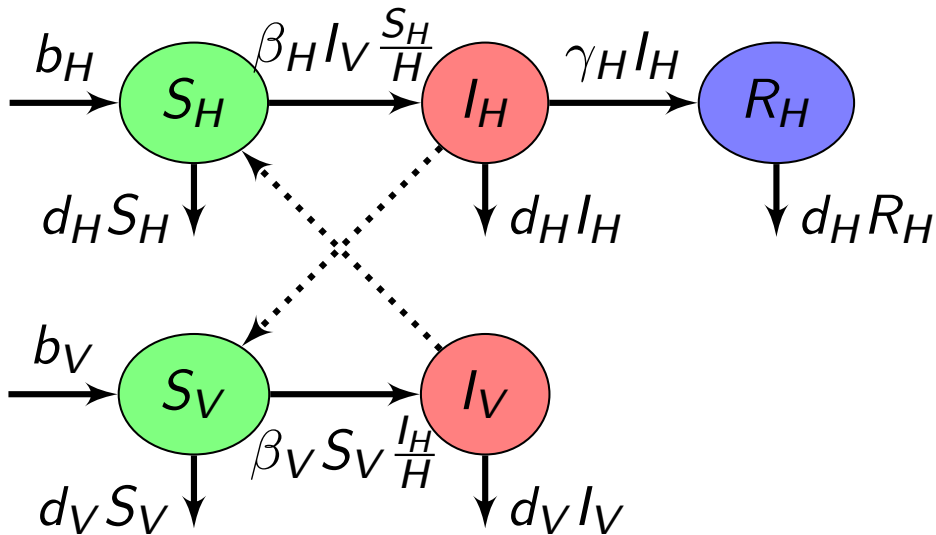
A little complexification of Ross-Macdonald



See, e.g., Simoy & Aparicio, Ross-Macdonald models: Which one should we use?, *Acta Tropica* (2020)

Ross introduced the model in 1911. Later “tweaked” by Macdonald to include mosquito latency period

Here, I show a version in the paper cited, with some notation changed

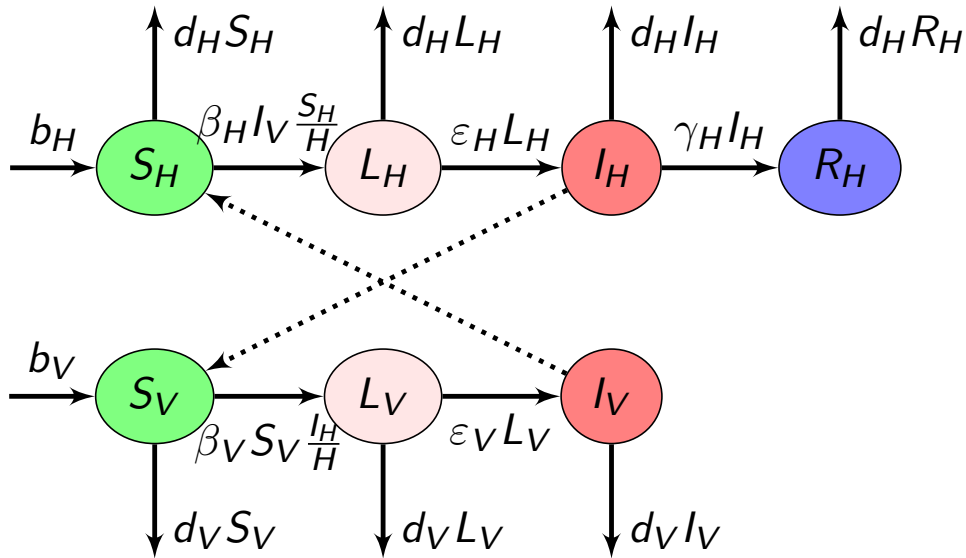


## Reproduction number

$$\mathcal{R}_0 = \frac{\beta_H \beta_V}{(\gamma_H + \gamma_V) d_V} \frac{V^*}{H^*} \quad (1)$$

where  $H^*$  and  $V^*$  are the total host and vector populations, respectively





## Reproduction number

$$\mathcal{R}_0 = \frac{\beta_H \beta_V}{(\gamma_H + \gamma_V) d_V} \frac{\varepsilon_V}{d_V + \varepsilon_V} \frac{\varepsilon_H}{d_H + \varepsilon_H} \frac{V^*}{H^*} \quad (2)$$

where  $H^*$  and  $V^*$  are the total host and vector populations, respectively

Here

$$f_X = \frac{\varepsilon_X}{d_X + \varepsilon_X}$$

are the fractions of latent individuals (of type  $X = \{V, H\}$ ) who survive the latency period

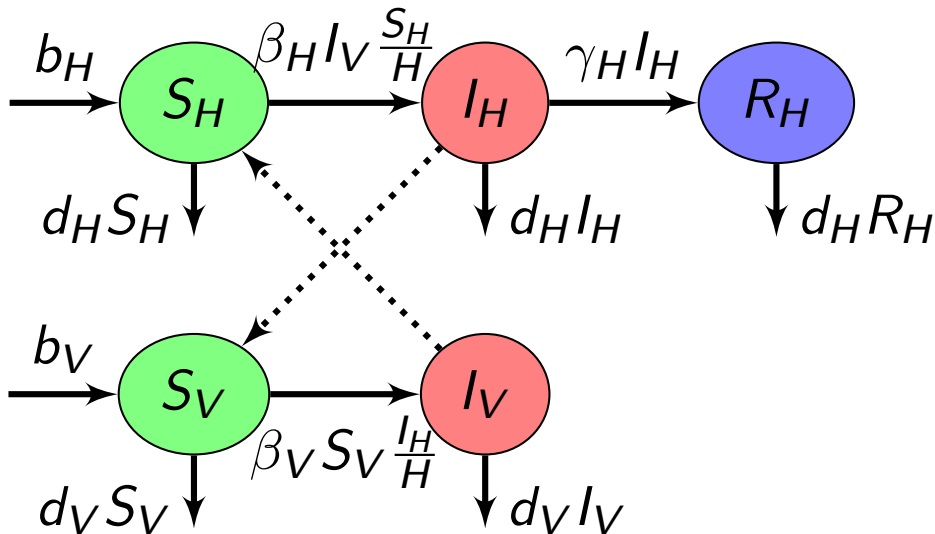
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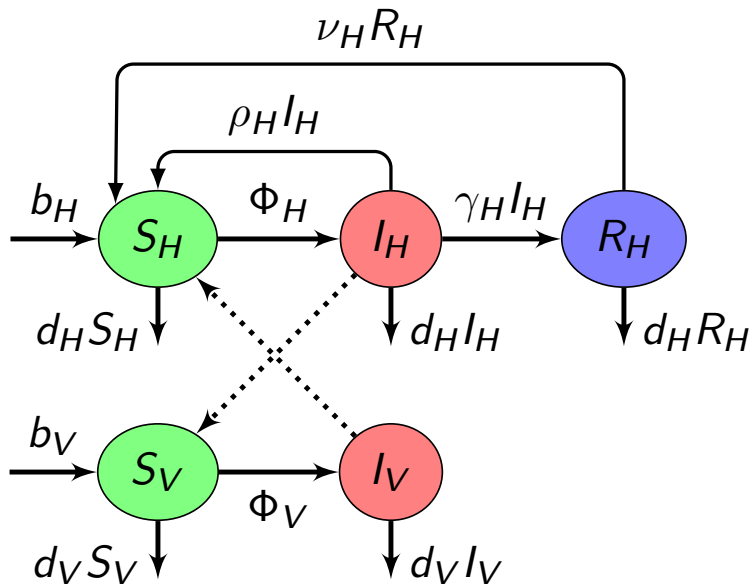
A little complexification of Ross-Macdonald



Recall this guy?



Let us add a few arrows



Arino, Ducrot & Zongo, A metapopulation model for malaria with transmission-blocking partial immunity in hosts, Journal of Mathematical Biology (2012)

Incidence functions take the form

$$\Phi_H = b_H(H, V)\sigma_{VH}\frac{I_V}{V}$$

and

$$\Phi_V = b_V(H, V)\left(\sigma_{HV}\frac{I_H}{H} + \hat{\sigma}_{HV}\frac{R_H}{H}\right)$$

where  $b_H$  and  $b_V$  are numbers per unit time of mosquito bites a human has and the number of humans a mosquito bites, respectively

## Parameters of the incidence function

- ▶  $\sigma_{HV}$  probability of transmission of the parasite (in gametocyte form) from an infectious human to a susceptible mosquito
- ▶  $\hat{\sigma}_{HV}$  probability of transmission of the parasite (in gametocyte form) from a semi-immune human to a susceptible mosquito
- ▶  $\sigma_{VH}$  probability of transmission of the parasite (in sporozoite form) from an infectious mosquito to a susceptible human

Additional parameter that can be factored in (all per unit time)

- ▶  $a_H$  maximum number of mosquito bites a human can receive
- ▶  $a_V$  number of times one mosquito would “want to” bite humans
- ▶  $a$  average number of bites given to humans by each mosquito

## People to read for malaria models (IMOBO)

See also the work of

- ▶ Gideon Ngwa at the University of Buea
- ▶ Nakul Chitnis at the Swiss Tropical and Public Health Institute

Many others...



## More complex models may be needed for malaria

Timing of processes is critical in malaria

Plasmodium life cycle in the mosquito is commensurate with mosquito lifetime

Need models that are able to account for that, because ODEs are not really good at this (see beginning of Stochastic systems lecture)

Mathematics becomes more complicated

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CONCLUSIONS



## To simplify or not to simplify?

- ▶ In the KMK epidemic model (??) and the SIRS endemic model (??), since the total population is constant or asymptotically constant, it is possible to omit one of the state variables since  $N^* = S + I + R$
- ▶ We often use  $R = N^* - S - I$
- ▶ This can greatly simplify some computations
- ▶ Whether to do it or not is a matter of preference


## To normalise or not to normalise?

- ▶ In the KMK epidemic model (??) and the SIRS endemic model (??), since the total population is constant or asymptotically constant, it is possible to normalise to  $N = 1$
- ▶ This can greatly simplify some computations
- ▶ However, I am not a big fan: it is important to always have the “sizes” of objects in mind
- ▶ If you do normalise, at least for a paper destined to mathematical biology, always do a “return to biology”, i.e., interpret your results in a biological light, which often implies to return to original values

## Where we are

- ▶ An *epidemic* SIR model (the KMK SIR) in which the presence or absence of an epidemic wave is characterised by the value of  $\mathcal{R}_0$
- ▶ The KMK SIR has explicit solutions (in some sense). **This is an exception!**
- ▶ An *endemic* SIRS model in which the threshold  $\mathcal{R}_0 = 1$  is such that, when  $\mathcal{R}_0 < 1$ , the disease goes extinct, whereas when  $\mathcal{R}_0 > 1$ , the disease becomes established in the population
- ▶ Some simple variations on these models
- ▶ A few models for vector-borne or water-borne diseases

# Bibliography I

-  J. Arino, A. Ducrot, and P. Zongo. A metapopulation model for malaria with transmission-blocking partial immunity in hosts. *Journal of Mathematical Biology*, 64(3):423–448, 2012.