

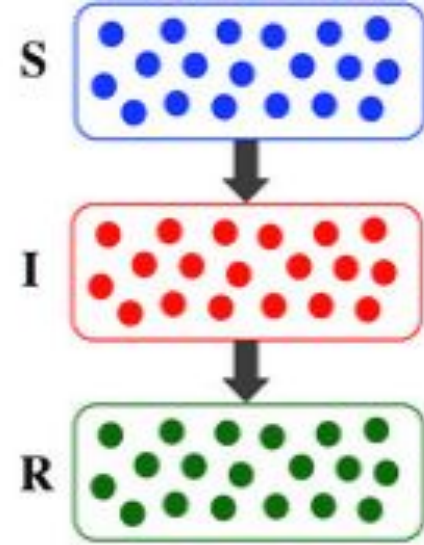
Compartmental Models in Epidemiology

NTD Workshop

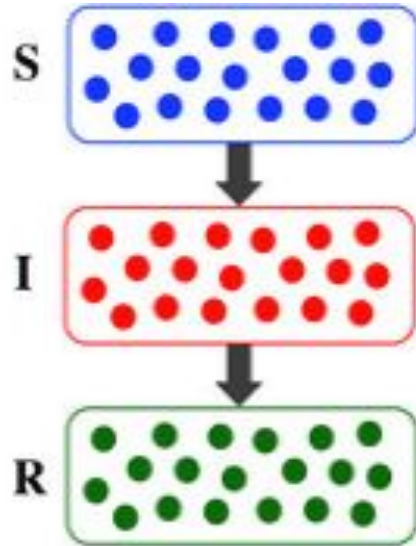
National Disease Modeling Consortium, IIT Bombay

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Compartmental Models



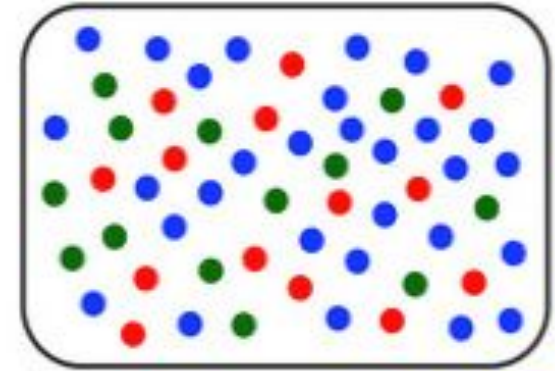
Classes of epidemic models



Compartmental Models

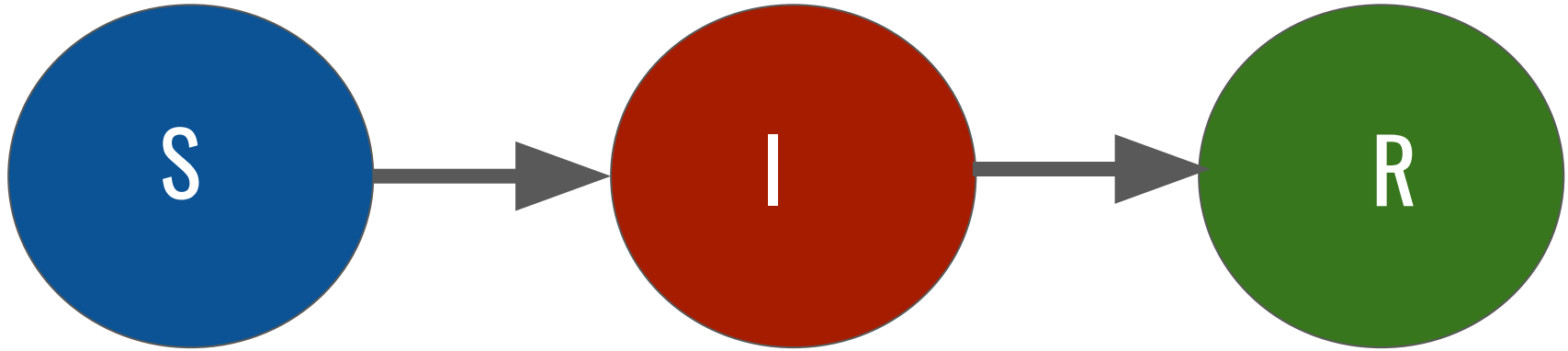


Spatial Models



Agent Based Models

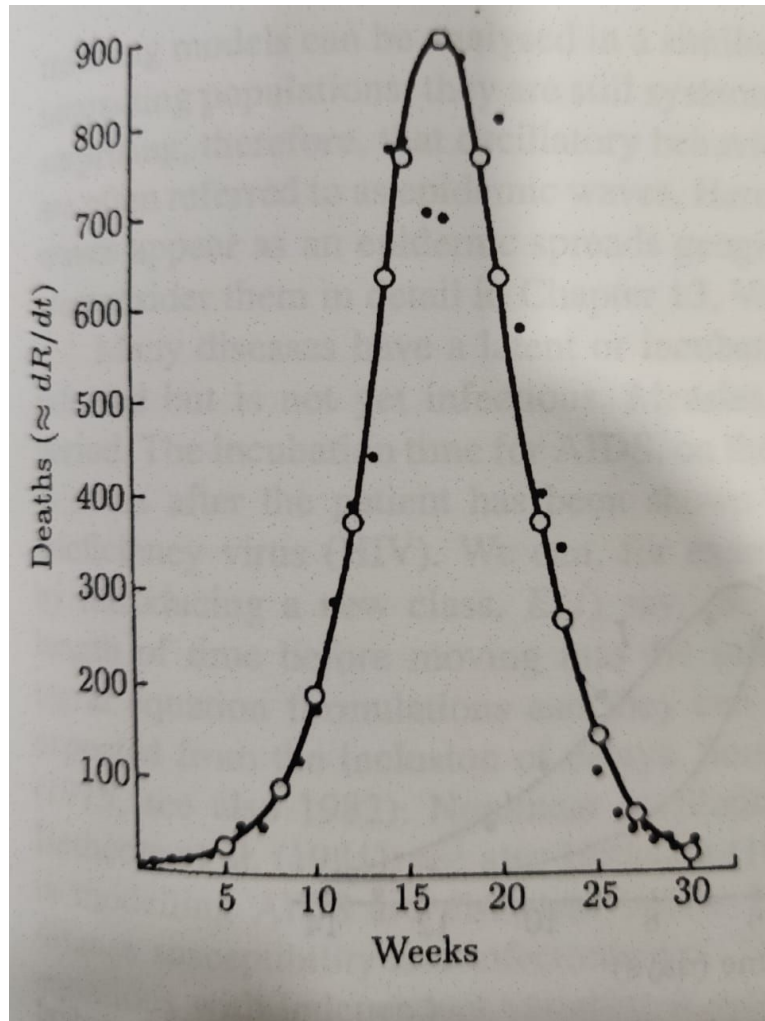
Kermack-McKendrick Epidemic Model



$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = +\beta SI - \gamma I$$

$$\frac{dR}{dt} = +\gamma I$$



General contact rates

Does the number of contacts per unit time depend on the population size?

Mass action incidence $c(N) = \beta N$

Standard incidence $c(N) = \beta$

Michaelis-Menten incidence $c(N) = \frac{\beta N}{1 + \lambda N}$

Power Law incidence $c(N) = \beta N^a$

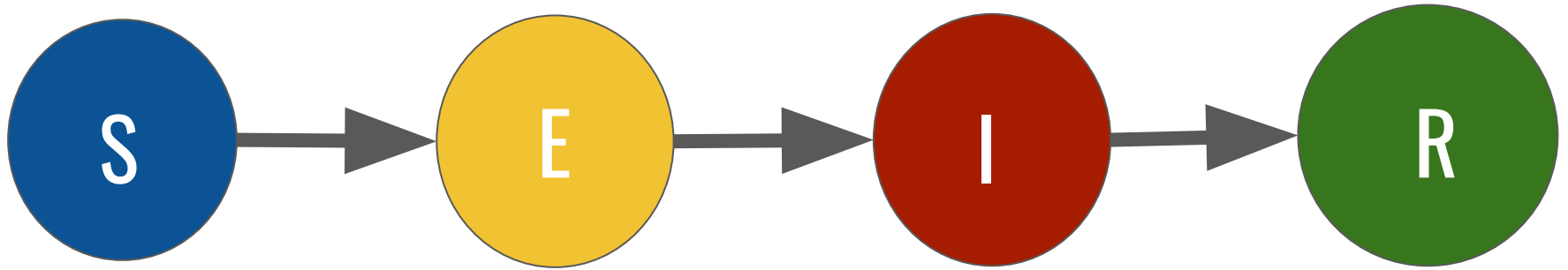
General contact rates

Does the number of contacts per unit time depend on the population size?

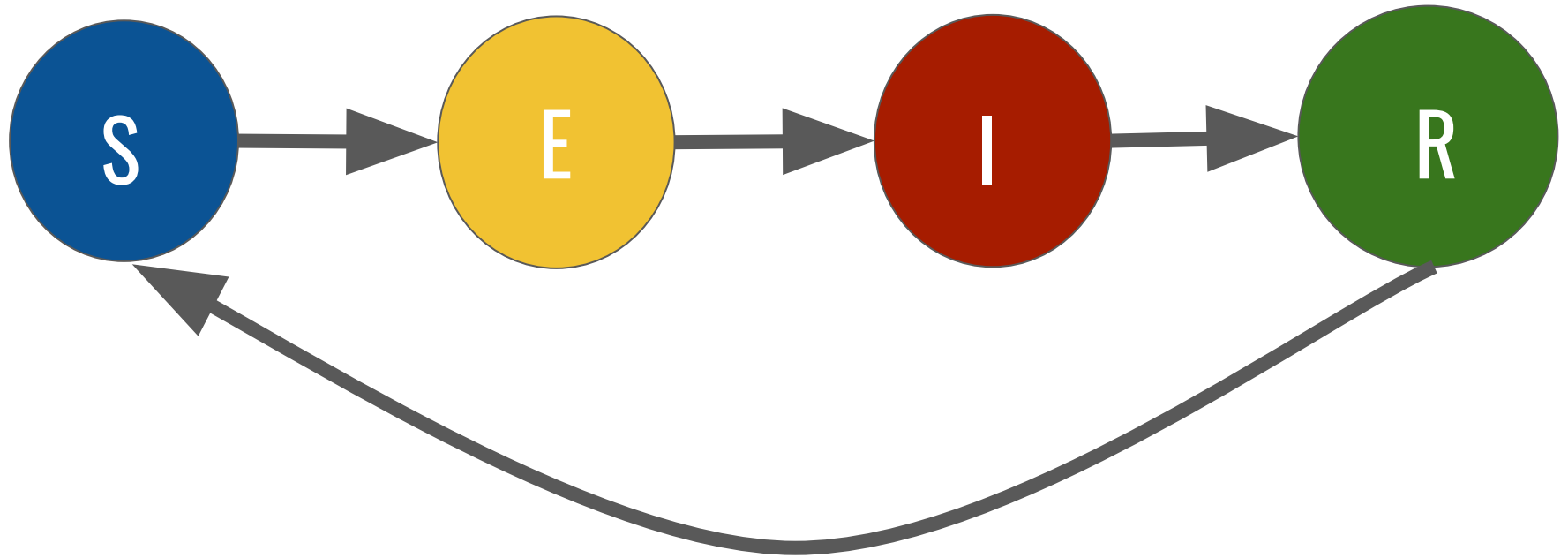
Standard incidence $c(N) = \beta$

$$\begin{aligned}\frac{dS}{dt} &= -\beta \frac{SI}{N} \\ \frac{dI}{dt} &= +\beta \frac{SI}{N} - \gamma I \\ \frac{dR}{dt} &= +\gamma I\end{aligned}$$

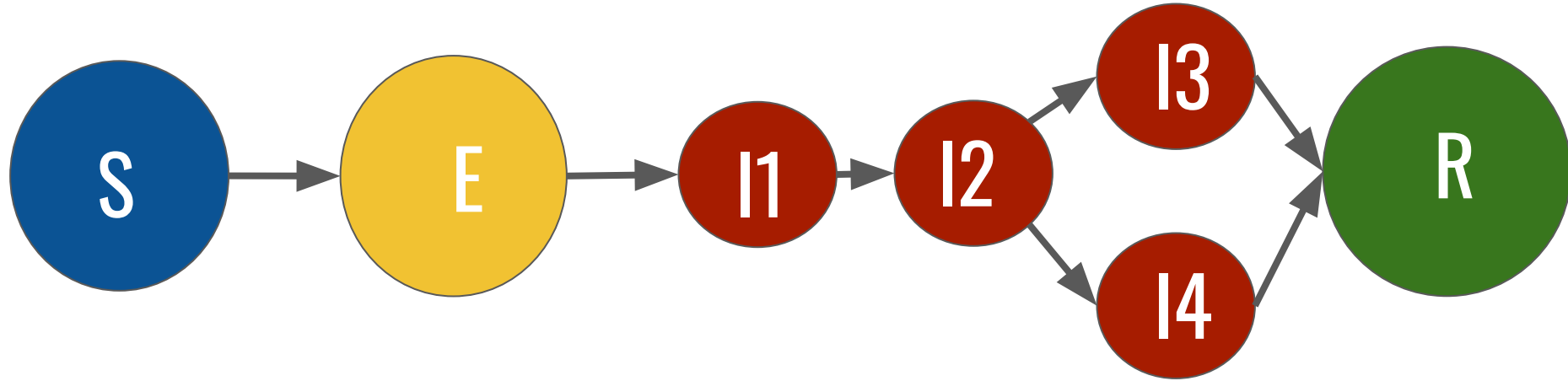
Generalisations of the SIR Model



Generalisations of the SIR Model



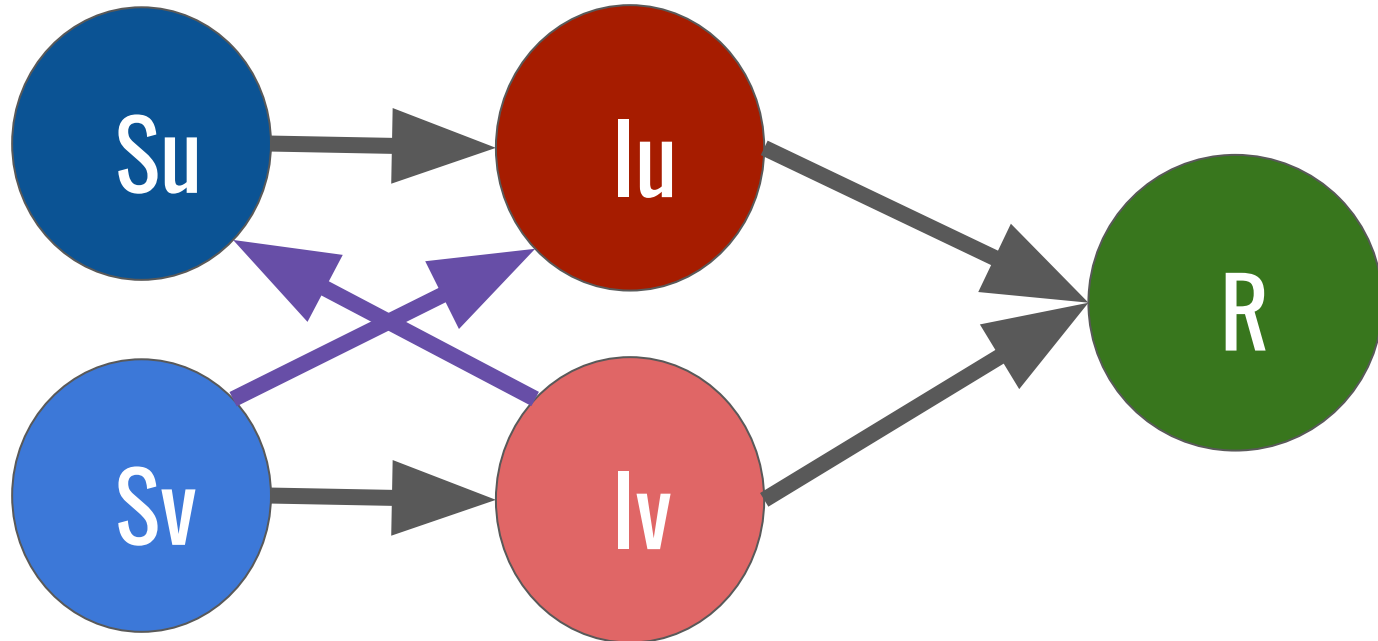
Generalisations of the SIR Model



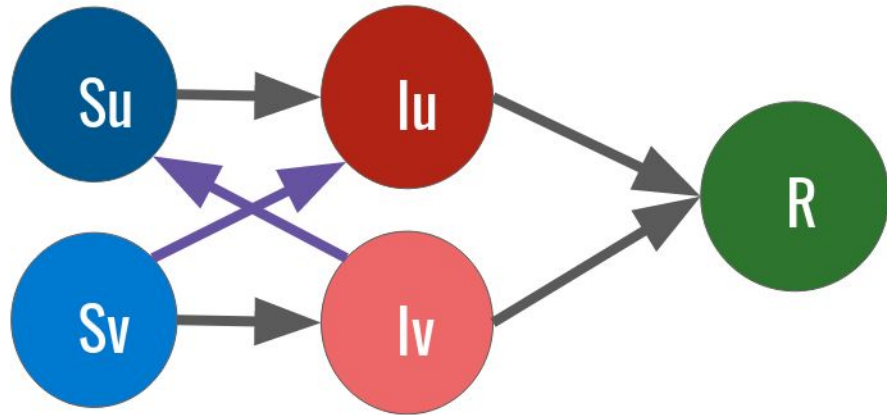
Vaccination Model

Vaccination against a disease can help in two ways:

- For vaccinated individuals, it can confer a reduced susceptibility to infections
- If a vaccinated individual does become infected, it can reduce their infectivity



Vaccination Model



$$\mathcal{R}_0 = \frac{a_u}{\alpha} + \frac{\delta \sigma a_v}{\alpha}$$

$$\begin{aligned}\frac{dS_U}{dt} &= -\beta S_U \left[\frac{I_U}{N_U} + \delta \frac{I_V}{N_V} \right] \\ \frac{dS_V}{dt} &= -\sigma \beta S_V \left[\frac{I_U}{N_U} + \delta \frac{I_V}{N_V} \right] \\ \frac{dI_U}{dt} &= +\beta S_U \left[\frac{I_U}{N_U} + \delta \frac{I_V}{N_V} \right] - \gamma I_U \\ \frac{dI_V}{dt} &= +\sigma \beta S_V \left[\frac{I_U}{N_U} + \delta \frac{I_V}{N_V} \right] - \gamma I_V \\ \frac{dR}{dt} &= +\gamma (I_U + I_V)\end{aligned}$$

Endemic Diseases

SIR models with birth and death

$$\begin{aligned}\frac{dS}{dt} &= +\Lambda(N) - \beta SI - \mu S \\ \frac{dI}{dt} &= +\beta SI - \gamma I - \mu I \\ \frac{dR}{dt} &= +\gamma I - \mu R\end{aligned}$$

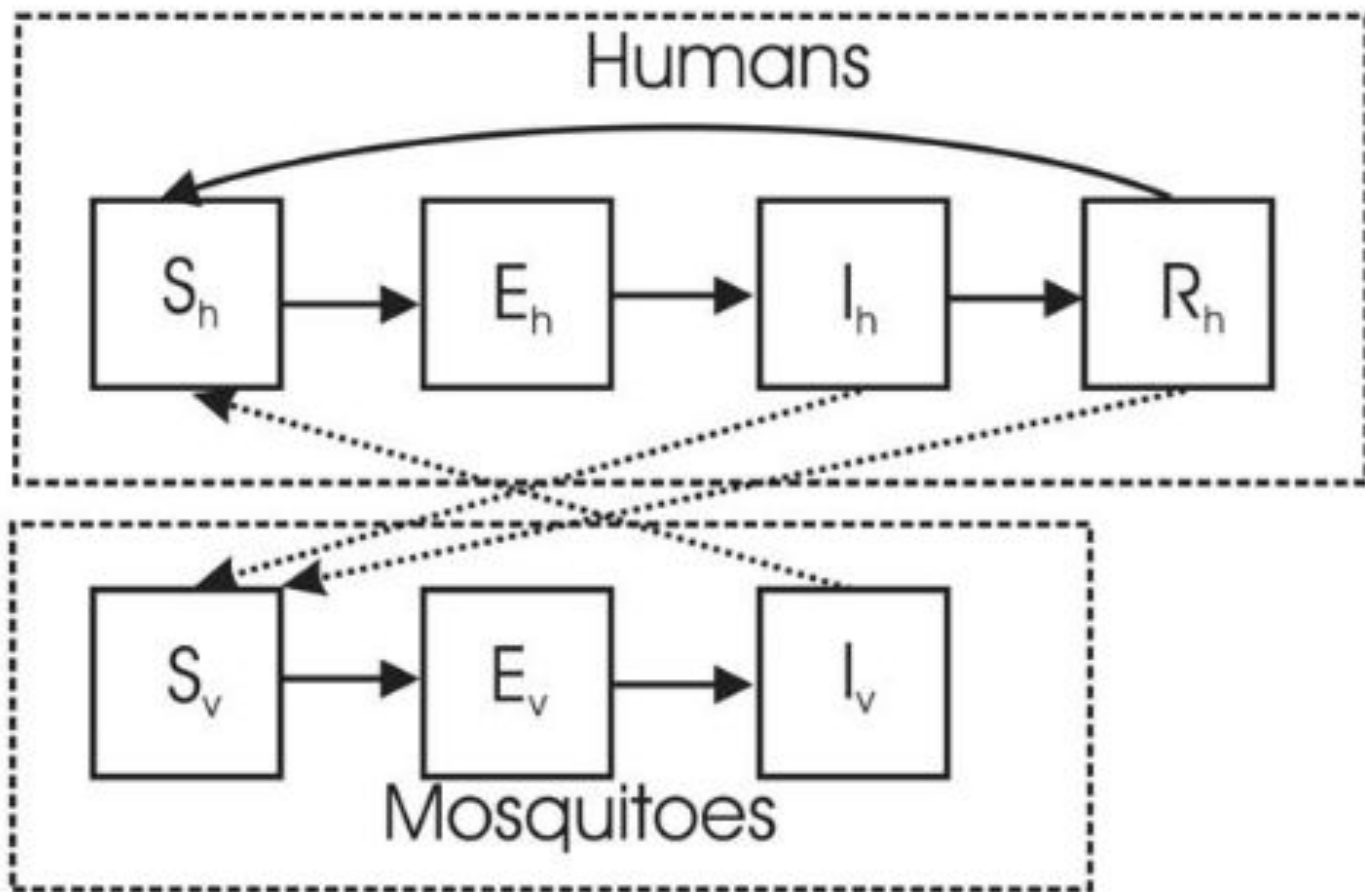
Vector-Borne Diseases

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**BIFURCATION ANALYSIS OF A MATHEMATICAL MODEL FOR
MALARIA TRANSMISSION***

NAKUL CHITNIS[†], J. M. CUSHING[‡], AND J. M. HYMAN[§]



S_h :	Number of susceptible humans
E_h :	Number of exposed humans
I_h :	Number of infectious humans
R_h :	Number of recovered (immune and asymptomatic, but slightly infectious) humans
S_v :	Number of susceptible mosquitoes
E_v :	Number of exposed mosquitoes
I_v :	Number of infectious mosquitoes
N_h :	Total human population
N_v :	Total mosquito population

$$(2.1a) \quad \frac{dS_h}{dt} = \Lambda_h + \psi_h N_h + \rho_h R_h - \lambda_h(t) S_h - f_h(N_h) S_h,$$

$$(2.1b) \quad \frac{dE_h}{dt} = \lambda_h(t) S_h - \nu_h E_h - f_h(N_h) E_h,$$

$$(2.1c) \quad \frac{dI_h}{dt} = \nu_h E_h - \gamma_h I_h - f_h(N_h) I_h - \delta_h I_h,$$

$$(2.1d) \quad \frac{dR_h}{dt} = \gamma_h I_h - \rho_h R_h - f_h(N_h) R_h,$$

$$(2.1e) \quad \frac{dS_v}{dt} = \psi_v N_v - \lambda_v(t) S_v - f_v(N_v) S_v,$$

$$(2.1f) \quad \frac{dE_v}{dt} = \lambda_v(t) S_v - \nu_v E_v - f_v(N_v) E_v,$$

$$(2.1g) \quad \frac{dI_v}{dt} = \nu_v E_v - f_v(N_v) I_v,$$

$$\frac{dN_h}{dt} = \Lambda_h + \psi_h N_h - f_h(N_h) N_h - \delta_h I_h,$$

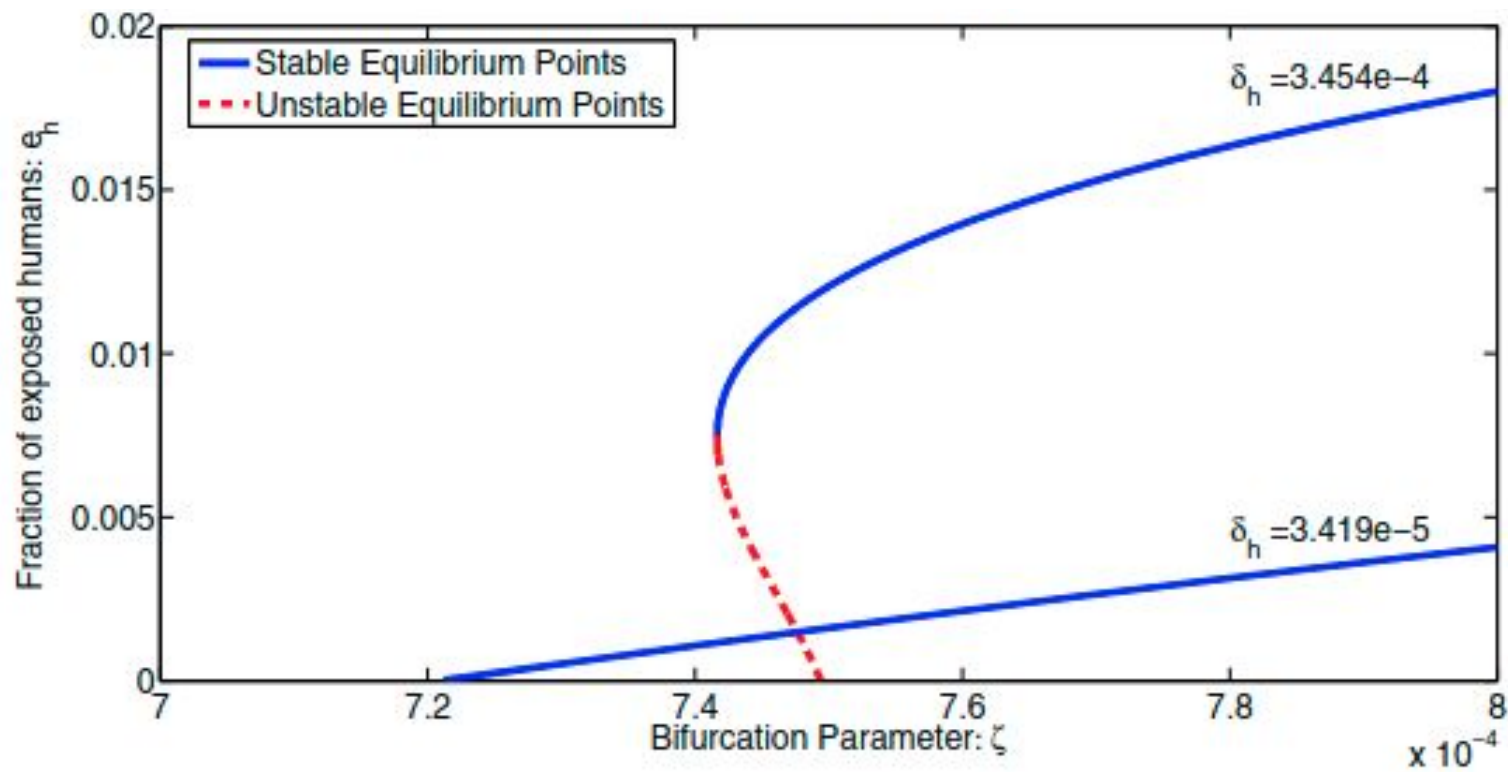
$$\frac{dN_v}{dt} = \psi_v N_v - f_v(N_v) N_v,$$

$$\lambda_h = b_h(N_h, N_v) \cdot \beta_{hv} \cdot \frac{I_v}{N_v} \quad \text{and} \quad \lambda_v = b_v(N_h, N_v) \cdot \left(\beta_{vh} \cdot \frac{I_h}{N_h} + \tilde{\beta}_{vh} \cdot \frac{R_h}{N_h} \right)$$

$$b = b(N_h, N_v) = \frac{\sigma_v N_v \sigma_h N_h}{\sigma_v N_v + \sigma_h N_h} = \frac{\sigma_v \sigma_h}{\sigma_v (N_v/N_h) + \sigma_h} N_v,$$

Λ_h :	Immigration rate of humans. Humans \times Time ⁻¹ .
ψ_h :	Per capita birth rate of humans. Time ⁻¹ .
ψ_v :	Per capita birth rate of mosquitoes. Time ⁻¹ .
σ_v :	Number of times one mosquito would want to bite humans per unit time, if humans were freely available. This is a function of the mosquito's gonotrophic cycle (the amount of time a mosquito requires to produce eggs) and its anthropophilic rate (its preference for human blood). Time ⁻¹ .
σ_h :	The maximum number of mosquito bites a human can have per unit time. This is a function of the human's exposed surface area. Time ⁻¹ .
β_{hv} :	Probability of transmission of infection from an infectious mosquito to a susceptible human, given that a contact between the two occurs. Dimensionless.
β_{vh} :	Probability of transmission of infection from an infectious human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless.
$\tilde{\beta}_{vh}$:	Probability of transmission of infection from a recovered (asymptomatic carrier) human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless.
ν_h :	Per capita rate of progression of humans from the exposed state to the infectious state. $1/\nu_h$ is the average duration of the latent period. Time ⁻¹ .
ν_v :	Per capita rate of progression of mosquitoes from the exposed state to the infectious state. $1/\nu_v$ is the average duration of the latent period. Time ⁻¹ .
γ_h :	Per capita recovery rate for humans from the infectious state to the recovered state. $1/\gamma_h$ is the average duration of the infectious period. Time ⁻¹ .
δ_h :	Per capita disease-induced death rate for humans. Time ⁻¹ .
ρ_h :	Per capita rate of loss of immunity for humans. $1/\rho_h$ is the average duration of the immune period. Time ⁻¹ .
μ_{1h} :	Density-independent part of the death (and emigration) rate for humans. Time ⁻¹ .
μ_{2h} :	Density-dependent part of the death (and emigration) rate for humans. Humans ⁻¹ \times Time ⁻¹ .
μ_{1v} :	Density-independent part of the death rate for mosquitoes. Time ⁻¹ .
μ_{2v} :	Density-dependent part of the death rate for mosquitoes. Mosquitoes ⁻¹ \times Time ⁻¹ .

$$\zeta = \frac{\sigma_v \sigma_h}{\sigma_v N_v^* + \sigma_h N_h^*}$$



Visceral leishmaniasis (VL) or Kala azar

Visceral Leishmaniasis in the Indian Subcontinent: Modelling Epidemiology and Control

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Abstract

Background: In the Indian subcontinent, about 200 million people are at risk of developing visceral leishmaniasis (VL). In 2005, the governments of India, Nepal and Bangladesh started the first regional VL elimination program with the aim to reduce the annual incidence to less than 1 per 10,000 by 2015. A mathematical model was developed to support this elimination program with basic quantifications of transmission, disease and intervention parameters. This model was used to predict the effects of different intervention strategies.

Methods and Findings: Parameters on the natural history of *Leishmania* infection were estimated based on a literature review and expert opinion or drawn from a community intervention trial (the KALANET project). The transmission dynamic of *Leishmania donovani* is rather slow, mainly due to its long incubation period and the potentially long persistence of parasites in infected humans. Cellular immunity as measured by the Leishmanin skin test (LST) lasts on average for roughly one year, and re-infection occurs in intervals of about two years, with variation not specified. The model suggests that transmission of *L. donovani* is predominantly maintained by asymptotically infected hosts. Only patients with symptomatic disease were eligible for treatment; thus, in contrast to vector control, the treatment of cases had almost no effect on the overall intensity of transmission.

Conclusions: Treatment of Kala-azar is necessary on the level of the individual patient but may have little effect on transmission of parasites. In contrast, vector control or exposure prophylaxis has the potential to efficiently reduce transmission of parasites. Based on these findings, control of VL should pay more attention to vector-related interventions. Cases of PKDL may appear after years and may initiate a new outbreak of disease; interventions should therefore be long enough, combined with an active case detection and include effective treatment.

PCR
DAT
LST

-/-
26%

+/-
10%

+/+
2%

-/+
12%

-/+
50%

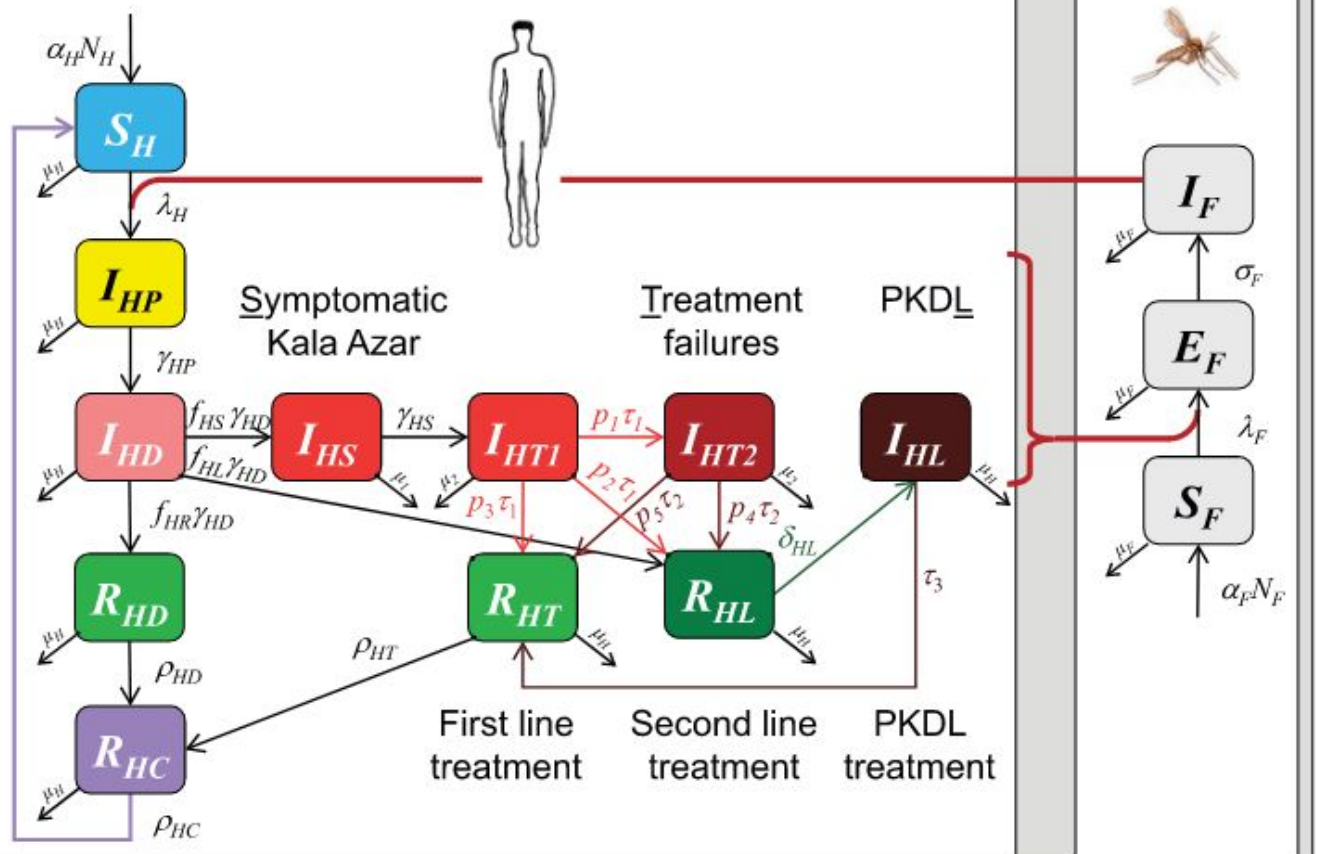


Table 4. Parameter combinations of treatment-related interventions.

Parameter	Scenario									
	Default 1	2	3	4	5	6	7	8	9	10
Duration first-line treatment $1/\tau_1$ (days)	30	1	5	5	30	30	30	30	30	30
Duration second-line treatment $1/\tau_2$ (days)	30	1	5	5	30	30	30	30	30	30
Duration PKDL treatment $1/\tau_3$ (days)	180	1	30	180	180	180	180	180	180	180
Early case detection $1/\tau_{HS}$ (days)	1	1	1	1	42	90	365	1	1	1
Treatment fatality f_T (%)	5	0	5	5	5	5	5	0	5	5
Treated fraction leading to retention of KA p_1 (%)	5	0	5	5	5	5	5	5	0	5
Treated fraction leading to relapse into PKDL p_2 (%)	3	0	3	3	3	3	3	3	3	0

Ten different scenarios were considered for sensitivity analyses of the equilibrium solutions to the effects of seven treatment-related intervention parameters. doi:10.1371/journal.pntd.0001405.t004

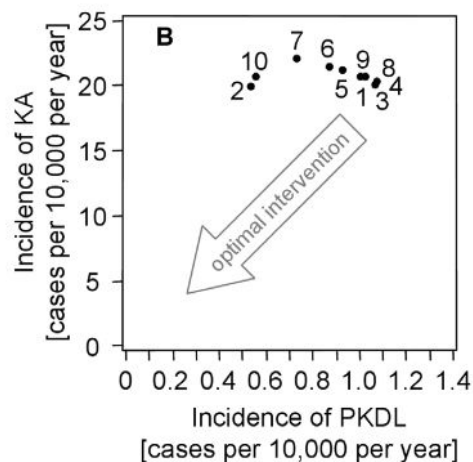
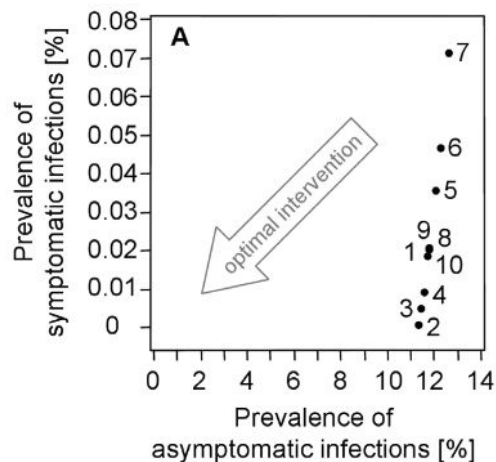
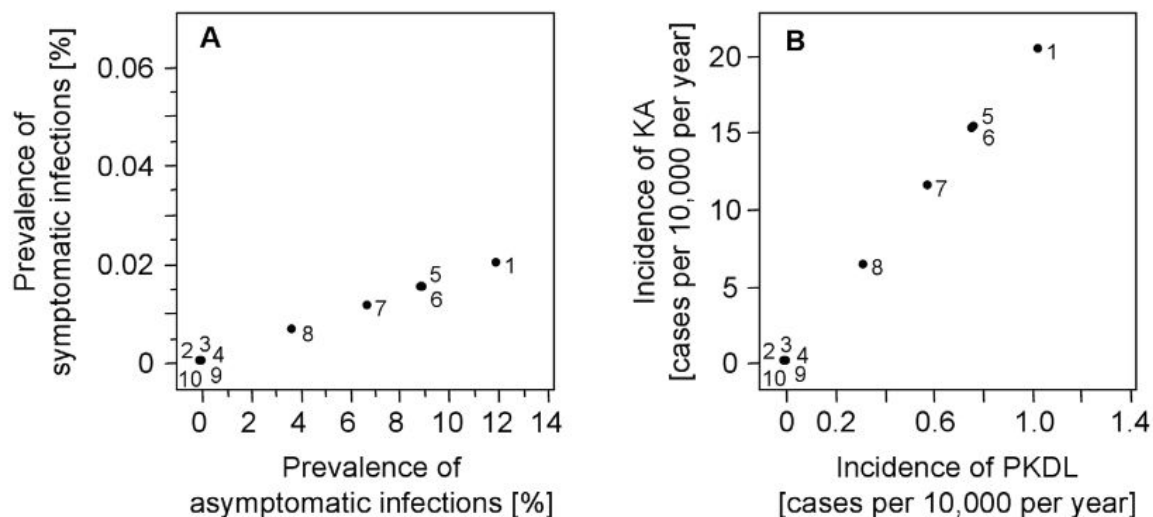


Table 5. Parameter combinations of vector-related interventions.

Parameter	Scenario									
	Default 1	2	3	4	5	6	7	8	9	10
No. of vectors (N_V) per $N_H = 100$ humans	527	100	527	527	300	527	527	300	300	527
Life expectancy of sand flies $1/\mu_F$ (days)	14	14	7	14	14	11	14	11	14	11
Feeding cycle duration $1/\beta$ (days)	4	4	4	8	4	4	6	4	6	6

Ten different scenarios were considered for sensitivity analyses of the equilibrium solutions to the effects of three vector-related intervention parameters.
doi:10.1371/journal.pntd.0001405.t005



Chapter 3

An Introduction to Stochastic Epidemic Models

Linda J.S. Allen

Lecture Notes in Mathematics

Fred Brauer
Pauline van den Driessche
Jianhong Wu (Eds.)

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