

Ecological effects on arbovirus-mosquito cycles of transmission

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Mosquitoes transmit many viruses to a variety of hosts. Cycles of mosquito borne arbovirus transmission are the result of complex interactions between the mosquito, the arbovirus and the host that are influenced by genetic variations in a variety of traits in each that are all influenced by many environmental factors. R_0 , the basic reproduction number or mean number of individuals infected from a single infected individual, is a measure of mosquito borne arbovirus transmission.

Understanding the causes for the distribution of R_0 in any transmission cycle is a daunting challenge due to the lack of information on the genetic and environmental variances that influence R_0 . Information about the major factors influencing R_0 for specific transmission cycles is essential to develop efficient and effective strategies to reduce transmission in different cycles and locations.

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Introduction

There are ca. 1500 known arthropod-borne viruses (arboviruses). The majority are transmitted by one to several of the 100s of vector mosquito species among approximately 3500 species of mosquitoes worldwide [1].

Arbovirus-mosquito cycles of transmission are due to the interactions between the mosquito, the arbovirus, the vertebrate host and many abiotic and biotic factors encompassed in the arbovirus ‘episystem’ [2]. The complex interactions in an episystem are difficult to understand in different locations, and therefore information for one episystem is not generally useful in a different episystem or time. Ecological factors influence the biotic factors in the mosquito, arbovirus and host populations. This is the subject of ecological genetics that

studies how ecological factors influence organism population genetics and diversity. This paper provides examples of ecological factors that influence the mosquito’s biology that are important in arbovirus transmission, the complexity of these effects and the challenges they present in understanding mosquito-arbovirus transmission cycles.

Basic reproduction number (R_0)

R_0 and the Ross-MacDonald theory of mosquito-borne pathogen transmission provides a useful framework to assess transmission cycles. R_0 is the mean number of individuals infected by a single infected individual. There will be decreasing infections each generation until infections disappear when $R_0 < 1$. When $R_0 > 1$ there is more than one secondary infection so infections increase and persist as long as there are enough susceptible in the population to maintain transmission.

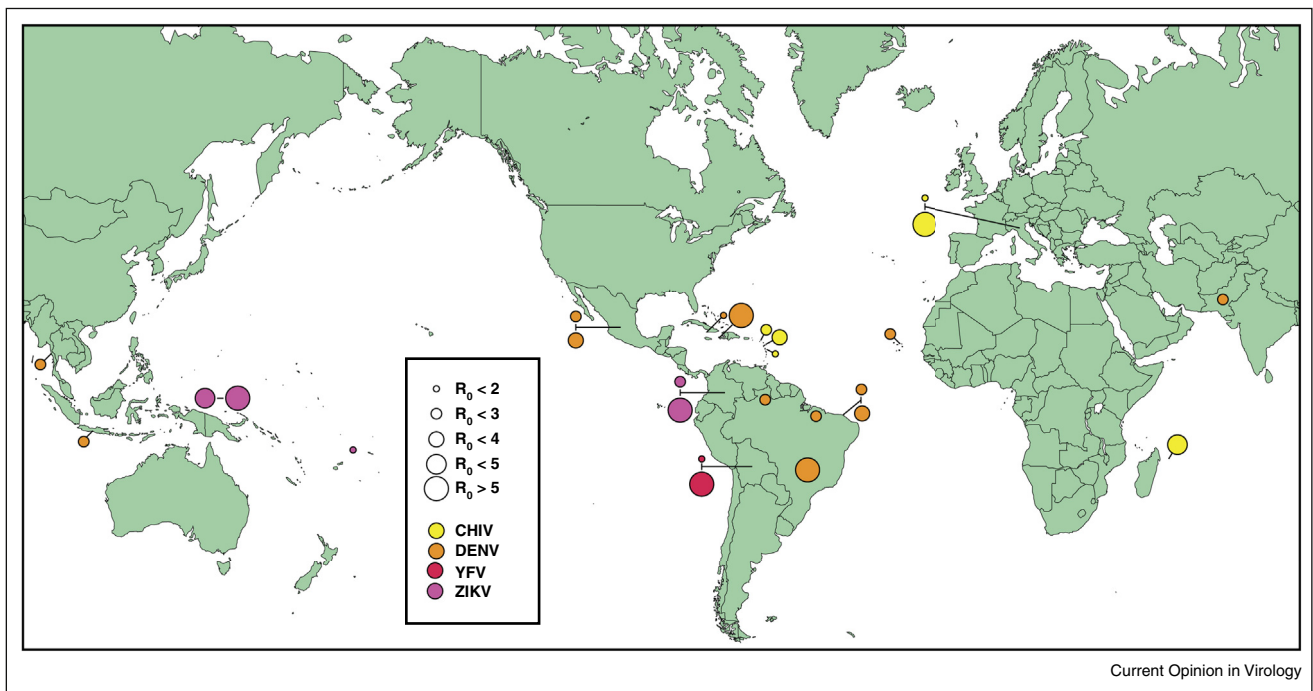
The development of R_0 and the Ross-MacDonald theory of mosquito-borne pathogen transmission are reviewed elsewhere [3^{**},4^{**}]. There have been many studies concerning R_0 , the Ross-MacDonald theory, methods to estimate R_0 and the challenges in providing such estimates. Between 1970 and 2010 325 publications provided 388 models for R_0 [5^{*}]. **Table 1** provides R_0 estimates for several *Aedes aegypti* and *Aedes albopictus* borne arbovirus epidemics. The estimates show great variation (**Figure 1**). It is not appropriate to compare R_0 between studies since different methods were used to estimate R_0 . There is variation within studies and several provide a range for R_0 for an outbreak (**Figure 1**) because of the range for some parameters in the estimation. **Table 1** also shows the percent of the human population that would need to be immunized to reduce $R_0 < 1$ and stop an epidemic. The actual reproduction rate (R) is R_0S where S is the proportion of susceptible individuals [17]. Since the immunized proportion (I) is $1 - S$, $R < 1$ requires $1 - (R_0/1)$. **Figure 2** shows the relationship between R_0 and I . Small changes in R_0 influence the immunity required to stop transmission. $R_0 = 2$ would require more than 50% I compared to 67% for $R_0 = 3$, with $R_0 > 5$ or more requiring $>80\%$ I . Risk maps with different R_0 in different locations have been proposed [18]. However estimating R_0 is difficult due to the patchiness of heterogeneities within each episystem [4] due to variations in contributing biotic and abiotic factors, some of which are illustrated in this paper. Despite difficulties in using R_0 to characterize and compare different episystems strategies seeking to reduce R_0 should address the biotic and abiotic components that most impact a particular episystem.

Table 1

R_0 estimates for selected *A. aegypti* or *A. albopictus arbovirus epidemics with the proportion (%) of the population needed with immunity to the arbovirus to provide $R_0 < 1$ [17]**

Epidemic	Country	Year	Arbovirus	R_0	Reference	% Immune
Belem	Brazil	1996	DENV	2.1	[6]	52%
Boa Vista	Brazil	1999	DENV	3.3	[6]	70%
Boa Vista	Brazil	2000	DENV	3	[6]	67%
Boa Vista	Brazil	2001	DENV	3.3	[6]	70%
Brasilia	Brazil	2001	DENV	103	[6]	99%
Brasilia	Brazil	2002	DENV	36	[6]	97%
Fortaleza	Brazil	2001	DENV	2	[6]	50%
Fortaleza	Brazil	2202	DENV	5.6	[6]	82%
Fortaleza	Brazil	2003	DENV	2	[6]	50%
Santiago	Cuba	1997/1998	DENV	2	[7]	50%
Port au Prince	Haiti	1996/1999	DENV	9	[7]	89%
Yogyakarta	Indonesia	1995	DENV	3–4	[7]	100%
Colima	Mexico	2002	DENV	2–3	[8]	50–67%
Cape Verde	Rep. Cape Verde	2009	DENV	2.4	[9]	58%
Pakistan	Pakistan	2011	DENV	3	[10]	67%
Rayong	Thailand	2010	DENV	3–4	[7]	100%
*Emilia-Romagna	Italy	2007	CHIKV	1.8–6	[11]	44–83%
Guadeloupe	Guadeloupe	2013	CHIKV	3.3	[12]	70%
Martinique	Martinique	2013	CHIKV	2.4	[12]	58%
Reunion	Reunion	2006	CHIKV	4	[13]	75%
St. Martin	St. Martin	2013	CHIKV	2.3	[12]	57%
Colombia	Colombia	2015–2016	ZIKV	2.2–14.8	[14]	55–93%
Fr. Polynesia	Fr. Polynesia	2013–2014	ZIKV	1.8–2.0	[15]	44–50%
Yap Island	Micronesia	2007	ZIKV	4.3–5.8	[15]	77–83%
Asuncion	Paraguay	2008	YFV	0.4–90	[16]	0–99%

Figure 1



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R_0 estimates for *A. aegypti* or *A. albopictus* epidemics of CHIKV, DENV, YFV and ZIKV (references in Table 1).

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