

# WEEKLY EPIDEMIOLOGICAL REPORT

## A publication of the Epidemiology Unit Ministry of Health

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## 25th - 31st May 2013

### A. aegypti density and the risk of dengue virus transmission (Part I)

This is the first in a series of two articles on Aedes aegypti density and the risk of dengue-virus transmission

### Background

Dengue is a disease which is spread by Aedes mosquitoes (mainly Aedes aegypti) and the disease is mainly confined to the tropics. Vector control plays big role in controlling Dengue disease and this article aims to provide an insight to recently developed vector indices that can be used.

The relationship of A. aegypti indices to the diversity of dengue-related disease is essentially unknown. Inconsistent associations from researches are common and may in part be attributable to relatively small sample sizes and short durations of study. In a large, cohort-based prospective study determined that traditional indices for A. Aegypti densities are correlated with prevalence of human dengue infections, but are at best weakly correlated with the incidence.

Methodologies for the surveillance and control of A. aegypti are rooted in techniques that were developed for mosquito eradication in order to prevent yellow fever. In the 1950s and 1960s a hemispherewide campaign to eradicate A. aegypti was initiated in the New World . The programme was successful, eliminating the mosquito from most of Latin America. For a variety of reasons – including changes in political and public-health priorities, changes in human demographics, increases in human travel, mosquito resistance to insecticides and perhaps most importantly, the inability to sustain the funding and infrastructure requirements of eradication – reinfestation of cleared areas began in the 1970s.

The new goal of dengue prevention and control programmes became the "cost-effective utilization of limited resources to reduce vector populations to levels at which they are no longer of significant public-health importance". The implicit assumption of this approach is that a reduction in the adult A. aegypti populations will decrease risk of virus transmission. In fact, it could be interpreted as any reduction, no matter how small, will reduce disease. Although this recommendation makes intuitive sense, it is not precise enough to be applied in an operational context. How should public-health entomologists identify and then reduce mosquito populations to the level at which they are no longer significant? For control, the objective is to maintain A. aegypti populations below or close to minimal transmission thresholds (see below), slow the force of denguevirus transmission and reduce sequential infections with heterologous serotypes, which are positively associated with increased risk of severe disease.

#### Mosquito density and Dengue transmission

Malariologists have been more successful than dengue researchers in relating vector density to infection and disease. The entomological inoculation rate (EIR), which is defined as the number of sporozoiteinfected mosquitoes biting a person per unit of time, is a robust measure of entomological risk for transmission of malaria parasites. Unfortunately, dengue researchers do not have an entomological measure for predicting the risk of human infection and disease that is as effective as the EIR is for malaria risk predictions. Virus infection rates in A. aegypti are typically too low to base a surveillance programme on an EIR or its equivalent. This is especially true when one considers the currently available technology for collecting adult female A. aegypti and detecting virus in them. In addition, sterilizing immunity (i.e. immunity against the same sero type) that follows a dengue-virus infection, which is different from the non-sterilizing response associated with malaria, can lead to spatially and temporally explicit patterns of virus transmission. For example, the probability of transmission will be low in an area regardless of the magnitude of measures of entomological risk, if human herd immunity were high. Conversely, if herd immunity is low, relatively low population densities of A. aegypti could precipitate an epidemic.

Moreover, A. aegypti survive and efficiently transmit dengue virus even when their population densities are remarkably low. Efficient virus transmission at

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low vector densities has been attributed to this mosquito's propensity to imbibe blood meals almost exclusively from humans and to do so frequently (0.6 - 0.8 meals per day), something that increases their contact with human hosts and as a result enhances their opportunities for contracting or transmitting a virus infection.

#### Entomological assumptions of dengue control

The fundamental premise is that disease can be managed by reducing A. aegypti population densities. However, no well-controlled field studies have been carried out to define the dynamic relationships between A. aegypti density and human virus infections. Defining this relationship using indices is essential for a vector-control strategy, and these indices must be field tested and validated.

The shift in focus from mosquito eradication to control prompted a re-evaluation of A. aegypti surveillance techniques. Unfortunately, as mentioned before, associations between existing indices and dengue transmission have not proven to be satisfactorily predictive. This may be because the most commonly applied indices are based on easy to sample immature A. aegypti that do not transmit virus. Only adult females transmit virus, and because they do not enter standard mosquito traps, they are difficult to collect in the context of a geographically diverse surveillance programme. Development of new methodologies to collect adult A. aegypti, especially females, for surveillance purposes would be a most valuable contribution to dengue prevention.

#### Acceptable level of dengue risk

Defining an acceptable level of dengue risk will be a complex and dynamic process that will depend on the resources, public-health priorities and history of dengue in the country or region affected. A likely acceptable and overreaching goal will be the desire to prevent large, explosive epidemics. The objective will be to reduce the force of virus transmission. In order to understand transmission well enough to predict outcomes of interventions with reasonable certainty, considerably more needs to be learned about the relationship between transmission dynamics and severe disease. In practice, public-health officials will most often set goals based on the individual needs of their country or region. Goals will need to be dynamic; that is, they will need to fluctuate as virus transmission and successes in disease prevention rise and fall. So that, for example, goals could range from no deaths in a community to no hospitalizations to no children missing school with a dengue illness to specified reductions in any of these outcomes. It cannot be overemphasized that the goal will be to prevent disease, which varies in severity and is not always a consequence of infection; some dengue infections are asymptomatic.

A conceptual representation of the relationship between mosquito vector density and the risk of a person being infected with dengue is illustrated graphically in Figure 1. In this scenario there are two thresholds. The maximum threshold is a density above which additional mosquitoes will not increase the risk of human infection because the system is saturated. Conversely, at densities below the minimum threshold, the risk of infection does not decrease because there are too few mosquitoes to sustain transmission. Transmission has ceased or if virus is introduced, its basic reproductive rate is always less than 1 and it fails to persist. Between those two densities it is predicted that there is a functional relationship linking density and risk, such that reduction in mosquito density results in a corresponding decrease in infection risk. This relationship is not a single curve. Instead, it is a theoretically infinite series of differentshaped curves representing different circumstances and conditions. We expect that the shape of the curve, or the nature of the relationship between density and risk, will vary temporally and spatially depending on factors like human herd immunity, density of human hosts, characteristics of mosquito-human interaction, virus introductions into the system, virulence of virus strains and weather (e.g. temperature and relative humidity) that affect mosquito biology and mosquito-virus interactions.

One way to address these issues is through the application of quantitative models. A differential equation model of dengue transmission has been developed to estimate the basic reproductive rate of dengue and to evaluate the relative merits of different insecticide and source-reduction control strategies. Results from the model reinforce conventional thoughts about the role of herd immunity in

#### Figure I-Mosquito density and risk of dengue



dengue-transmission dynamics. As herd immunity increased, higher mosquito densities were needed to support dengue transmission. For example, at >80 immunity, no transmission occurred, even when mosquito densities were high (5 mosquitoes/person). Below 80% immunity, significant transmission could occur at increasingly lower vector densities. Thus, with herd immunity at 50%, about 40% of the human population can be infected at a density of 3 mosquitoes/ person, whereas with herd immunities at 10%, a similar number of cases can occur at less than 1.5 mosquitoes/person. But these conclusions require validation with field data.

During the early to mid-1990s two computer simulation models were developed and they can be used to estimate dengue entomological thresholds. First is the container-inhabiting mosquito simulation model, and it led to development of the dengue-transmission simulation model. The mosquito model is a habitat and weather driven accounting programme of the population dynamics of A. aegypti. The dengue model accounts for the dynamics of a human population driven by country and age-specific birth and death rates. As part of a validation of the dengue model, data from a 1978 dengue epidemic in Honduras were examined. During the epidemic, a positive correlation between A. aegypti indices and sero-prevalence of dengue antibody was observed, which suggests that low mosquito densities prevented dengue transmission in unaffected communities. Simulations with lower mosquito abundances indicated that this explanation was plausible.

#### Source-

Aedes aegypti density and the risk of dengue-virus transmissionavailable from <u>http://edepot.wur.nl/136912</u>

Compiled by Dr. Madhava Gunasekera of the Epidemiology Unit

18th - 24th May 2013 (21st Week)

WRCD %	С**	31	0	15	26	31	31	47	17	0	50	75	0	25	80	36	71	42	15	23	42	29	24	36	33	6	77	30	
	т*	69	100	85	74	69	69	53	83	100	50	25	100	75	20	64	29	58	85	77	58	71	76	64	67	91	23	70	
maniasis	B	0	2	1	1	2	0	0	120	44	0	4	1	4	7	0	1	12	23	3	154	60	ю	Ŀ	8	0	1	456	
Leish	A	0	0	0	0	0	0	0	2	2	0	0	0	Ч	0	0	0	3	1	0	1	4	0	1	0	0	0	15	
gitis	8	25	49	34	2	13		23	11	28	27	9	4	19	2	2	7	2	59	10	48	∞	6	10	14	46	ъ	491	
Menin	A	1	3	1	0	0	0	0	1	0	1	1	1	0	0	0	0	0	1	1	2	0	1	0	0	1	1	16	
xodua	B	220	83	138	74	24	43	129	53	153	106	2	6	17	2	14	42	17	191	44	77	70	67	29	79	171	47	1901	
Chick	A	1	З	ю	2		0	4	1	ω	1	0	0		0	0	0	1	9	0	3	2	2	2	0	ω	4	43	
abies*	B	0	0	0	0	0	0	1	0	2	0	0	0	1	2	0	0	1	1	0	0	1	0	1	1	0	0	11	
Hu R	A	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	00	
Viral	B	35	98	10	23	21	11	9	63	66	6	0		0	0	∞	H	3	28	1	13	18	24	38	122	112	4	778	
	A	1	1	1	1	0	0	0	0	7	0	0	0	0	0	0	0	0	0	0	1	0	1	0	2	2	0	10	
Typhus	B	5	8	1	60	2	35	22	37	36	310	14	16	2	ъ	2	0	4	17	10	15	2	31	24	14	48	2	725	
	A	0	0	0	0	0	0	0	1	0	2	0	1	0	0	0	0	0	0	0	0	0	2	0	1	7	0	08	
ospiros	B	118	157	196	38	27	12	109	121	102	5	6	6	39	15	22	2	46	161	14	243	110	16	167	198	65	4	2010	
Lept	A	2	6	4			0	0	2	2	0	0	0	m	0	0	0	0	4		8	9	0		m	4	0	51	
poc	B	14	13	6	2	0	m	4	6	27	7	2	11	∞	m	2	0	1	5	35	2	0	2	18	15	4	25	231	
Ĕ	A	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	2	-	0	0		0	Э		0	60	
interic	8	50	17	35	6	9	4	2	7	10	222	۵	49	Ŀ	9	0	4	2	23	11	2	10	~	6	22	6	e	529	
Ei Ei	A	0	1	0	0	0	0	0	0	0	5	0		0	0	0	0	0	0	0	0	0	0		0	2	0	10	
cephali	B	11	8	11	9		2	∞	2	6	4	0	-	10	1	Μ	0	1	23	4	12	0		m	77	10	1	205	RCD).
En	A	1	1	0	0	0	0	0	0		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	5 03	ses (W
sentery	B	73	57	63	53	35	78	38	22	27	88	12	22	22	9	87	41	32	84	26	33	37	62	44	187	30	57	131(	e Disea
Dys	A	0	4	5	2	0	2	ω	0		2	0		0	0	4	0	0	2	0	1	0	m		ε	4		42	unicabl
ue Fever	B	3503	1523	709	737	197	105	321	150	241	433	25	53	40	99	339	64	137	1763	543	316	180	199	115	881	513	44	13599	of Comm
Deng	A	91	59	17	21		2	6	2	Ŀ	11	0		2		14	2		32	8	8		0		12	16	4	321	Returns
RDHS		Colombo	Gampaha	Kalutara	Kandy	Matale	NuwaraEliya	Galle	Hambantota	Matara	Jaffna	Kilinochchi	Mannar	Vavuniya	Mullaitivu	Batticaloa	Ampara	Trincomalee	Kurunegala	Puttalam	Anuradhapura	Polonnaruwa	Badulla	Monaragala	Ratnapura	Kegalle	Kalmune	SRI LANKA	Source: Weekly

\*T=Timeliness refers to returms received on or before 24th May, 2013 Total number of reporting units 329. Number of reporting units data provided for the current week: 234\*\* Completeness A = Cases reported during the current week. B = Cumulative cases for the year. Hu Rabies\*= Human Rabies

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## 25<sup>th</sup> - 31<sup>st</sup> May 2013

## Table 1: Vaccine-Preventable Diseases & AFP

## 25<sup>th</sup> – 31<sup>st</sup> May 2013

### 18th - 24th May 2013 (21st Week)

Disease			ľ	No. of Cas	ses by P	rovince	•	Number of cases during current	Number of cases during same	Total number of cases to date in	Total num- ber of cases to date in	Difference between the number of cases to date			
	W	С	S	N	E	NW	NC	U	Sab	week in 2013	week in 2012	2013	2012	in 2013 & 2012	
AFP*	00	01	00	00	00	00	00	00	01	02	01	31	35	- 11.4 %	
Diphtheria	00	00	00	00	00	00	00	00	00	-	-	-	-	-	
Mumps	02	01	00	03	00	02	03	01	02	14	07	678	1899	- 64.3 %	
Measles	25	13	08	00	00	00	00	00	00	46	00	497	20	+ 2385.0 %	
Rubella	00	00	00	00	00	00	00	00	00	00	-	11	-	-	
CRS**	00	00	00	00	00	00	00	00	00	00	-	05	-	-	
Tetanus	00	00	00	00	00	00	00	00	00	00	00	08	05	+ 60.0 %	
Neonatal Teta- nus	00	00	00	00	00	00	00	00	00	00	-	00	-	-	
Japanese En- cephalitis	02	01	00	00	00	00	00	00	00	03	-	209	-	-	
Whooping Cough	00	00	00	00	00	00	00	00	00	00	00	34	32	- 03.1 %	
Tuberculosis	132	08	09	15	00	60	00	03	04	231	170	3368	3572	+ 01.6 %	

#### Key to Table 1 & 2

Provinces: W: Western, C: Central, S: Southern, N: North, E: East, NC: North Central, NW: North Western, U: Uva, Sab: Sabaragamuwa.

RDHS Divisions: CB: Colombo, GM: Gampaha, KL: Kalutara, KD: Kandy, ML: Matale, NE: Nuwara Eliya, GL: Galle, HB: Hambantota, MT: Matara, JF: Jaffna,

KN: Killinochchi, MN: Mannar, VA: Vavuniya, MU: Mullaitivu, BT: Batticaloa, AM: Ampara, TR: Trincomalee, KM: Kalmunai, KR: Kurunegala, PU: Puttalam, AP: Anuradhapura, PO: Polonnaruwa, BD: Badulla, MO: Moneragala, RP: Ratnapura, KG: Kegalle.

Data Sources:

Weekly Return of Communicable Diseases: Diphtheria, Measles, Tetanus, Neonatal Tetanus, Whooping Cough, Chickenpox, Meningitis, Mumps., Rubella, CRS, Special Surveillance: AFP\* (Acute Flaccid Paralysis), Japanese Encephalitis

CRS\*\* =Congenital Rubella Syndrome

AFP and all clinically confirmed Vaccine Preventable Diseases except Tuberculosis and Mumps should be investigated by the MOH

**Dengue Prevention and Control Health Messages** 

Look for plants such as bamboo, bohemia, rampe and banana in your surroundings and maintain them free of water collection.

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Comments and contributions for publication in the WER Sri Lanka are welcome. However, the editor reserves the right to accept or reject items for publication. All correspondence should be mailed to The Editor, WER Sri Lanka, Epidemiological Unit, P.O. Box 1567, Colombo or sent by E-mail to chepid@sltnet.lk. Prior approval should be obtained from the Epidemiology Unit before publishing data in this publication

## **ON STATE SERVICE**

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