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A MATHEMATICAL MODEL ON DENGUE DISEASE SPREAD WITH VACCINATION

Pradeep Porwal, Dinesh Kumar Verma*, V. H. Badshah
School of Studies in Mathematics,
Vikram University, Ujjain (M.P.), India
E-mail: dineshverma_51@yahoo.com

**Corresponding author.*

Abstract The present mathematical model deals with the study of dengue disease model. We start by formulating and analyzing the model with vaccinated human population. The resulting model equation were solved and analyzed. The disease free equilibrium of the system was established and analyzed for stability. Numerical results are also provided to justify the stability.

MSC: 93A30, 93D20, 92D30, 65L07
Keywords: Dengue disease model, Vaccination strategy, Stability analysis, Numerical analysis.

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1. Introduction

Dengue Fever (DF) and Dengue Hemorrhagic Fever (DHF), collectively known as dengue are mosquito-borne viral diseases that affect humans of all ages worldwide [3, 16, 14]. Transmitted to humans by the bite of an infected mosquito, dengue is caused by any one of four serotypes: DENV 1, DENV 2, DENV 3, and DENV 4 [3, 4, 9, 14]. After a human is bitten by a mosquito carrying the dengue virus, symptoms appear in 3-14 days (average 4-7 days) [16].

In recent decades the occurrence of the disease has grown-up significantly around the globe. Currently about 40% of the humanity are at the moment at risk from the disease. With human infectivity estimated at about 50100 million dengue cases globally each year by WHO. Only nine nations had experienced severe dengue epidemic before 1970. The disease is currently prevalent in more than 100 nations in Africa, the Americas, the Eastern Mediterranean, South-east Asia and the Western Pacific. Generally, the critically affected nations are the Americans, South-east Asia and the Western Pacific. Cases across these nations have surpasses 1.2 million in the year 2008 and more than 2.3 million in the year 2010 studied by Coller [5] and Clements. An approximate 500 000 humans with

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severe dengue need hospitalization every year, a huge percentage of whom are children. About 2.5% of infected humans die [7].

In the year 2013, 2.35 million cases of the disease were reported in the Americas only. Out of these cases 37687 were severe dengue. The number of cases is greater than ever as the disease stretches to new areas, but explosive epidemics are happening. The risk of a likely epidemic of the disease at the present exists in Europe and local spread of the disease was reported for the first time in France and Croatia in 2010 and imported cases were identified in three other European countries discussed by Schaffner [12].

In this paper, an epidemiology model for dengue disease is proposed by Rodrigues [10]. The model considered in this study offers some extensions to the dengue transmission model by Rodrigues [10]. This is done by incorporating the recruitment rate of humans and mosquitoes and the maturation rate of the infected larva to adult mosquitoes. It consists of six mutually-exclusive compartments involving the interactions between humans and mosquitoes. It is mathematically written as a system of ordinary differential equations. The model is designed to describe the dynamics of the disease transmission into a population and to perform the sensitivity analysis for the model parameters.

2. The Mathematical Model

The mathematical model is based on [11, 13]. The notation used in our mathematical model includes four epidemiological states for humans

$S_h(t)$: susceptible (individuals who can contract the disease),

$I_h(t)$: infected (individuals capable of transmitting the disease to others),

$R_h(t)$: resistant (individuals who have acquired immunity).

It is assumed that the total human population (N_h) is constant, $N_h = S_h(t)+I_h(t)+R_h(t)$. There are also three other state variables related to the female mosquitoes:

$A_v(t)$: aquatic phase (that includes the larva and pupa stages)

$S_m(t)$: susceptible (mosquitoes that are able to contract the disease)

$I_m(t)$: infected (mosquitoes capable of transmitting the disease to humans).

The parameters of the model are:

N_h = Total population

θ_h = Recruitment of humans

$b\beta_{mh}$ = Contact rate between infectious vectors and susceptible humans

$b\beta_{hm}$ = Contact rate between infectious hosts and susceptible mosquitoes

μ_h = Natural death rate for human

γ_h = Viremic period rate

Q_m = Recruitment of mosquitoes

γ_A = Maturation rate from larva to adult

μ_A = Natural Mortality rate of larva

C_v = Mortality rate due to using adult side

μ_v = Natural death rate for mosquitoes

P = Vaccinated susceptible human

α_h = Disease related death rate.

The Dengue epidemic can be modeled by the following nonlinear time varying state equations and generalize the result of Ali. T. M. et. Al [1]:

For Human Population:

$$\frac{dS_h}{dt} = \theta_h - \left(b\beta_{mh}\frac{I_m}{N_h} + \mu_h\right)S_h - pS_h, \tag{2.1}$$

$$\frac{dI_h}{dt} = b\beta_{mh}\frac{I_m}{N_h}S_h - \Big(\gamma_h + \mu_h + \alpha_h\Big)I_h, \tag{2.2}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h + pS_h. \tag{2.3}$$

For Vector Population:

$$\frac{dA_v}{dt} = Q_m\left(1 - \frac{A_v}{fN_h}\right)(S_m + I_m) - (\gamma_A + \mu_A + d_A)A_v, \tag{2.4}$$

$$\frac{dS_m}{dt} = \gamma_A A_v - \left(b\beta_{hm}\frac{I_h}{N_h}\mu_v + c_v\right)S_m \tag{2.5}$$

$$\frac{dI_s}{dt} = b\beta_{hm}\frac{I_h}{N_h}S_m - \Big(\mu_v + c_v\Big)I_m. \tag{2.6}$$

where $\gamma_h = b\beta_{mh}\frac{I_m}{N_h}$, $\gamma_v = b\beta_{hm}\frac{I_h}{N_h}$ represent force of infectious for human and Aedes, respectively.

3. Positivity of Solution

To verify the model (2.1) - (2.6) to be epidemiologically meaningful and well posed we have to prove that all state variables are positive for all $t \geq 0$.

Lemma 3.1. *Let* $\Big(S_h(0)\Big), \Big(I_h(0)\Big), \Big(R_h(0)\Big), \Big(A_v(0)\Big), \Big(M_s(0)\Big), \Big(M_i(0)\Big) \geq 0$. *Then the solution set* $\Big(S_h(t)\Big), \Big(I_h(t)\Big), \Big(R_h(t)\Big), \Big(A_v(t)\Big), \Big(M_s(t)\Big), \Big(M_i(t)\Big)$ *of the model (2.1)-(2.6), is positive for all* $t \geq 0$.

Proof. From the first equation of the system (2.1)-(2.6), we have

$$\frac{dS_h}{dt} = \theta_h - \left(b\beta_{mh}\frac{I_m}{N_h} + \mu_h\right)S_h - pS_h.$$

By substituting $\lambda = \Big(b\beta_{mh}\frac{I_m}{N_h}\Big)$, in equation (2.1) then

$$\frac{dS_h}{dt} = \theta_h - \Big(\lambda + \mu_h\Big)S_h - pS_h$$

Thus

$$\frac{dS_h}{dt} \geq -\Big(\lambda + \mu_h\Big)S_h - pS_h$$

$$\frac{dS_h}{dt} \geq \Big\{ -\Big(\lambda + \mu_h\Big) - p\Big\}S_h.$$

It then follows that

$$\frac{dS_h}{S_h} \geq \Big\{ -\Big(\lambda + \mu_h\Big) + p\Big\}dt.$$

By separating the variable and integrating, it gives

$S_h(t) \geq S_h(0)e^{-(\lambda+\mu_h+p)t} > 0$ if and only if $(\lambda + \mu_h) > 0$.

Similarly, we have

$$\frac{dI_h}{dt} = \lambda S_h - \Big(\gamma_h + \mu_h + \alpha_h\Big)I_h$$

or

$$\frac{dI_h}{dt} \geq -\Big(\gamma_h + \mu_h + \alpha_h\Big)I_h.$$

It then follows that

$$\frac{dI_h}{I_h} \geq -\Big(\gamma_h + \mu_h + \alpha_h\Big)dt.$$

On integrating, we have

$I_h(t) \geq I_h(0)e^{-(\gamma_h+\mu_h+\alpha_h)t} > 0$ if and only if $-(\gamma_h + \mu_h + \alpha_h) > 0$.

Also we have

$$\frac{dA_v}{dt} = Q_m\left(1 - \frac{A_v}{fN_h}\right)(S_m + I_m) - \big(\gamma_A + \mu_A + d_A\big)A_v$$

or

$$\frac{dA_v}{dt} \geq -\big(\gamma_A + \mu_A + d_A\big)A_v.$$

Separating variables and integrating, we get

$$\frac{dA_v}{A_v} \geq -\big(\gamma_A + \mu_A + d_A\big)dt$$

or $A_v(t) \geq A_v(0)e^{-(\gamma_A+\mu_A+d_A)t} > 0$ if and only if $(\gamma_A + \mu_A + d_A) > 0$.

Hence we generalize for the other compartments of the model (2.1)-(2.6), get the same result. Then we can deduce that the state variables of the model (2.1)-(2.6) are all positive for all $t > 0$.

4. Existence of Equilibria

To obtain the equilibrium point set the right side of system (2.1)-(2.6) equal to zero. Let $E(S_h, I_h, R_h, A_v, S_m, I_s)$ be the equilibrium points of the system (2.1)-(2.6).

The disease free equilibrium points of the model (2.1)-(2.6) is obtained by equating the time derivative equals to zero, i.e.,

$$\theta_h - \left(b\beta_{mh}\frac{I_m}{N_h} + \mu_h\right)S_h - pS_h = 0 \tag{4.1}$$

$$b\beta_{mh}\frac{I_m}{N_h}S_h - \Big(\gamma_h + \mu_h + \alpha_h\Big)I_h = 0 \tag{4.2}$$

$$\gamma_h I_h - \mu_h R_h + pS_h = 0 \tag{4.3}$$

$$Q_m\left(1 - \frac{A_v}{fN_h}\right)(S_m + I_m) - (\gamma_A + \mu_A + d_A)A_v = 0 \tag{4.4}$$

$$\gamma_A A_v - \left(b\beta_{hm}\frac{I_h}{N_h}\mu_v + c_v\right)S_m = 0 \tag{4.5}$$

$$b\beta_{hm}\frac{I_h}{N_h}S_m - \Big(\mu_v + c_v\Big)I_m = 0 \tag{4.6}$$

We found that the disease free equilibrium points are:

$$E_0\Big(S_h, I_h, R_h, A_v, S_m, I_m\Big) = \left(\frac{\theta_h}{(\mu_h + p)}, 0, \frac{p\theta_h}{\mu_h(p + \mu_h)}, fN_h\left(1 - \frac{(\mu_v + c_v)(\gamma_A + \mu_A + d_A)}{Q_m\gamma_A}\right), \frac{\gamma_A fN_h}{(\mu_v + c_v)}(1 - \eta), 0\right)$$

where

$$\eta = \frac{(\mu_v + c_v)(\gamma_A + \mu_A + d_A)}{Q_m\gamma_A}$$

(ii) Existence of $E_1\Big(S_h^*, I_h^*, R_h^*, A_v^*, S_m^*, I_s^*\Big)$

The endemic equilibrium of the model (2.1)-(2.6) can be obtained by making right hand side equal to zero.

We found that the endemic equilibrium points are:

$$E_1\Big(a_h^*, I_h^*, R_h^*, A_v^*, S_m^*, I_m^*\Big) \neq 0,$$

where

$$S_h^* = \frac{\theta_h}{\left(b\beta_{mh}\frac{I_m}{N_h} + \mu_h\right) + p},\ I_h^* = \frac{b\beta_{mh}\frac{I_m}{N_h}S_h^*}{\left(\gamma_h + \mu_h + \alpha_h\right)},$$

$$R_h^* = \frac{\gamma_h I_h^* + pS_h^*}{\mu_h},\ A_v^* = \frac{Q_m\left(S_m^* + I_m^*\right)}{\left(\frac{Q_m\left(S_m^*+I_m^*\right)}{fN_h} + \left(\gamma_A + \mu_A + d_A\right)\right)},$$

$$S_m^* = \frac{\gamma_A A_v^*}{\left(b\beta_{hm}\frac{I_h^*}{N_h} + \mu_v + c_v\right)},\ I_m^* = \frac{b\beta_{hm}\frac{I_h^*}{N_h}S_m^*}{\left(\mu_v + c_v\right)}.$$

5. Stability Analysis

To discuss the stability of the model (2.1) - (2.6),

(i) The Jacobian matrix of system (2.1)-(2.6) is given by

$$M(X) = \begin{bmatrix} \gamma_h - \mu_h - p & 0 & 0 & 0 & 0 & -b\beta_{mh}\frac{S_h}{N_h} \\ \gamma_h & -\left(\gamma_h + \mu_h + \alpha_h\right) & 0 & 0 & 0 & b\beta_{mh}\frac{S_h}{N_h} \\ p & \gamma_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & -\left(\gamma_A + \mu_A + \alpha_A\right) & 0 & 0 \\ 0 & -b\beta_{hm}\frac{S_m}{N_h} & 0 & 0 & -\left(\gamma_h + \mu_v + \alpha_v\right) & 0 \\ 0 & b\beta_{hm}\frac{S_m}{N_h} & 0 & 0 & b\beta_{mh}\frac{I_h}{N_h} & -\left(\mu_v + c_v\right) \end{bmatrix}$$

We found that the disease free equilibrium points:

$$E_1\left(S_h, I_h, R_h, A_v, S_m, I_s\right) = \left(\frac{\theta_h}{\mu_h + p}, 0, \frac{p\theta_h}{\mu_h(p + \mu_h)}, fN_h(1-\eta), \frac{\gamma_A fN_h(1-\eta)}{\mu_v + c_v}, 0\right),$$

where

$$\eta = \frac{\left(\mu_v + c_v\right)\left(\gamma_A + \mu_A + d_A\right)}{Q_m\gamma_A}.$$

(ii) The endemic equilibrium of the model (2.1)-(2.6), is given by

$$E_2\left(S_h^*, I_h^*, R_h^*, A_v^*, S_m^*, I_m^*\right) \neq 0.$$

The basic Reproduction number is obtained by Next generation method. Let F represent the rate of appearance of new infection in compartment i^{th} and V represent the rate of transfer of individual into compartment i^{th} of model (2.1)-(2.6), respectively, then these are given by

$$F = \begin{bmatrix} 0 & b\beta_{hm}\frac{S_m}{N_h} \\ b\beta_{mh}\frac{S_h}{N_h} & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \gamma_h + \mu_h + \alpha_h & 0 \\ 0 & \mu_v + c_v \end{bmatrix}.$$

To verify the model (2.1)-(2.6) to be epidemiologically meaningful and

$$f(X) = \begin{bmatrix} b\beta_{mh}\frac{I_m}{N_h}S_h \\ b\beta_{hm}\frac{I_h}{N_h}S_m \end{bmatrix}_{F_2}^{F_1} \text{ and } V(x) = \begin{bmatrix} \Big(\gamma_h + \mu_h + \alpha_h\Big)I_h \\ \Big(\mu_v + c_v\Big)I_m \end{bmatrix}_{V_2}^{V_1}$$

$$f = J_{f(x)} = \left[\frac{\partial F_i}{\partial X_j}(X_0)\right]$$

$$= \frac{\partial\Big(F_1, F_2\Big)}{\partial\Big(I_h, I_m\Big)}$$

$$= \begin{bmatrix} \frac{\partial F_1}{\partial I_h} & \frac{\partial F_2}{\partial I_h} \\ \frac{\partial F_1}{\partial I_m} & \frac{\partial F_2}{\partial I_m} \end{bmatrix}$$

$$F = \begin{bmatrix} 0 & b\beta_{hm}\frac{S_m}{N_h} \\ b\beta_{mh}\frac{S_h}{N_h} & 0 \end{bmatrix} \text{ and } v = J_{V(x)} = \left[\frac{\partial V_i}{\partial X_j}\Big(v_0\Big)\right]$$

$$= \frac{\partial\Big(V_1, V_2\Big)}{\partial\Big(I_h, I_m\Big)}$$

$$= \begin{bmatrix} \frac{\partial V_1}{\partial I_h} & \frac{\partial V_2}{\partial I_h} \\ \frac{\partial V_1}{\partial I_m} & \frac{\partial V_2}{\partial I_m} \end{bmatrix}$$

$$V = \begin{bmatrix} \gamma_h + \mu_h + \alpha_h & 0 \\ 0 & \mu_v + c_v \end{bmatrix}$$

$$V^{-1} = \frac{Adj(v)}{|v|} = \frac{1}{(\gamma_h + \mu_h + \alpha_h)(\mu_v + c_v)} \begin{bmatrix} \mu_v + c_v & 0 \\ 0 & \gamma_h + \mu_h + \alpha_h \end{bmatrix}$$

$$FV^{-1} = \frac{1}{(\gamma_h+\mu_h+\alpha_h)(\mu_v+c_v)} \begin{bmatrix} 0 & b\beta_{hm}\frac{S_m}{N_h} \\ b\beta_{mh}\frac{S_h}{N_h} & 0 \end{bmatrix} \begin{bmatrix} \mu_v+c_v & 0 \\ 0 & \gamma_h+\mu_h+\alpha_h \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & b\beta_{hm}\frac{S_m}{N_h}(\gamma_h+\mu_h+\alpha_h) \\ b\beta_{mh}\frac{S_h}{N_h}(\mu_v+c_v) & 0 \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & \frac{b\beta_{hm}S_m}{N_h(\mu_v+c_v)} \\ \frac{b\beta_{mh}\frac{S_h}{N_h}}{N_h(\gamma_h+\mu_h+\alpha_h)} & 0 \end{bmatrix}$$

$$R_0 = \frac{1}{2}\left(trace(FV^{-1}) + \sqrt{tr(FV^{-1})^2 - 4det(FV^{-1})}\right)$$

$$R_0 = \frac{1}{2}\left(0 + \sqrt{0 + \frac{4b^2\beta_{hm}\beta_{mh}S_hS_m}{N_h^2(\gamma_h+\mu_h+\alpha_h)(\mu_v+c_v)}}\right)$$

$$R_0 = \left(\sqrt{\frac{b^2\beta_{hm}\beta_{mh}S_hS_m}{N_h^2(\mu_v+c_v)(\gamma_h+\mu_h+\alpha_h)}}\right)$$

Hence

$$R_0 = \left(\sqrt{\frac{b^2\beta_{hm}\beta_{mh}\theta_h\chi_A f(1-h_0)}{N_h(\mu_v+\delta)(\mu_v+c_v)(\gamma_h+\mu_h+\alpha_h)}}\right)$$

By applying theorem 2 of Van and Watmough [15], we get the following result.

Theorem 5.1. *The disease free equilibrium of the system (2.1)(2.6) is stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

6. Numerical Simulation And Conclusion

The graph was obtained using polymath software. The variables and parameters of the model as follows

Parameter	Interpretation	Value	Reference
N_h	Total population	$480*10^3$	[10]
θ_h	Recruitment rate of humans	30	[2]
b	Average daily biting(per day)	0.8	[10]
β_{mh}	Transmission probability from infected human to mosquito (per bite)	0.375	[10]
β_{mh}	Transmission probability from infected human to mosquito (per bite)	0.375	[10]
Q_m	Recruitment rate of mosquitoes	400	[6]
$\frac{1}{\mu_v}$	Average lifespan of adult mosquitoes (in days)	0.1	[10]
γ_A	Maturation rate from larvae to adult (per day)	0.08	[10]
$\frac{1}{\mu_A}$	Natural mortality of larvae (per day)	0.25	[10]
d_A	Mortality rate for the larva stage due the use of toxo-mosquito per day	16	[8]
C_v	Mortality rate of mosquitoes due to the use of adulticide	0.6	[10]
$\frac{1}{\mu_h}$	Average lifespan of humans (in days)	0.00003858	[10]
$\frac{1}{\gamma_h}$	Mean viremic period (in days)	0.33	[10]
f	Number of larvae per human	3	[150]
p	Vaccinated susceptible human	0.2	Modelled
α_h	Disease related death rate	0.3	Modelled

Table 6.1.

Figure 6.1 describes the typical behavior of both human and mosquito populations. It has shown that the number of both infected humans and mosquitoes is increased and reached maximum value between the 20^{th} and 30^{th} days.

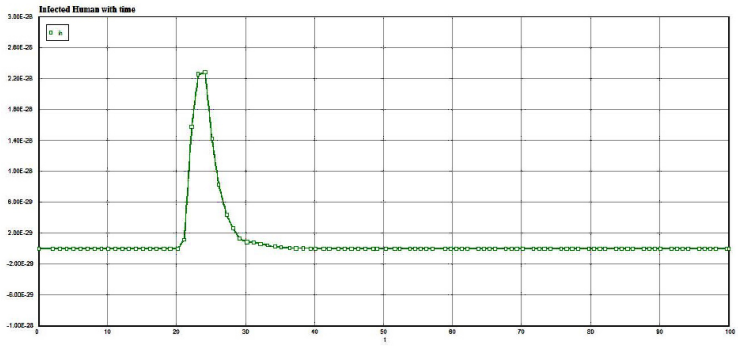

Figure 6.1.

In this chapter, we have studied an epidemic model with a fraction of vaccine is given 0 susceptible at rate p. The model has significance for the study of dynamics of dengue disease spread. The model is mathematically more general. The disease free equilibrium is stable if $R_0 < 1$ and unstable if $R_0 > 1$. It has been shown that the number of infected host and vector increased and has a peak between the 20^{th} and 30^{th} day. Also if the value of p is increases then the infected host, infected vector and the reproduction number to become small.

References

[1] T. M. Ali, A. A. Kamil and K. M. F. Abd, *Deterministic Mathematical Model of Dengue Disease Spread.*, Far East Journal of Mathematical Sciences, 96 (4) (2015); 419-436.

[2] C. Bowman, A. B. Gumel, P. V. D. Driessche, J. Wu and H. Zhu, *A Mathematical Model for Assessing Control Strategies Against West Nile Virus*, Bull. Math. Biology, 67 (5) (2005); 1107-1133.

[3] *Centers for Disease Control and Prevention*, http://www.cdc.gov/Dengue.

[4] *Classification, Medical Microbiology*, (1996); 213-261.

[5] B. A. Coller and D. E. Clements, *Dengue Vaccines: Progress and Challenges*, Curr. Opin. Immunol., 23 (3) (2011); 391-8.

[6] L. Esteva and C. Vargas, *Analysis of a Dengue Disease Transmission Model*, Math. Bio Sci., 150 (2) (1998); 131-151.

[7] J. Helmersson, *Mathematical Modeling of Dengue-Temperature Effect on Vectorial Capacity, Master Thesis*, Ume University (2012).

[8] C. Jones and E. Schreiber, *The Carnivores, Toxorhynchites*, Wing Beats, 5 (4) (1994); 4.

[9] K. J. Ryan, C. G. Ray and J. C. Sherris, *Sherris Medical Microbiology: an Introduction to Infectious Diseases*, McGraw-Hill Medical (2004).

[10] H. S. Rodrigues, M. T. T. Monteiro, D. F. M. Torres and A. Zinober, *Dengue Disease, Basic Reproduction Number and Control*, Inter. J. Computer Math. 89 (3) (2012); 334-346.

[11] H. S. Rodrigues, M. T. T. Monteiro and D. F. M. Torres, *Sensitivity Analysis in a Dengue Epidemiological Model, in Conference Papers in Mathematics*, Hindawi Publishing Corporation (2013).

[12] F. Schaffner, J. M. Medlock and W. Van Bortel, *Public Health Significance of Invasive Mosquitoes in Europe*, Clin. Microbiol. Infect., 19 (8) (2013); 685-92.

[13] S. Syafruddin and M. S. M. Noorani, *SEIR Model for Transmission of Dengue Fever in Selangor Malaysia*, Inter. J. Modern Physics: Conference Series, 9 (2012); 380-389.

[14] K. M. Tomashek, *Dengue fever (DF) and Dengue Hemorrhagic Fever (DHF)*, CDC Health Information for International Travel (2009).

[15] D. D. P. Van and J. Watmough, *Reproduction Numbers and Sub-threshold Endemic Equilibria for Compartmental Models of Disease Transmission*, Mathematical Biosciences, 180 (1-2) (2002); 2948.

[16] *World Health Organization*, http://www.who.int/topics/dengue/en.

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Science Laboratory Building, Faculty of Science
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126 Pracha Uthit Road, Bang Mod, Thung Khru, Bangkok, Thailand 10140
Website: http://tacs.kmutt.ac.th/
Email: tacs@kmutt.ac.th

