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Modelling the effect of temperature on transmission of dengue

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Abstract. The main entomological parameters involved in the rate of dengue virus transmission include the longevity of female mosquitoes, the time interval between bites and the extrinsic incubation period of the virus. Field and laboratory data provide estimates for these parameters, but their interactions with other factors (e.g. host population density and environmental parameters) make their integration into a transmission model quite complex. To estimate the impact of these parameters on transmission, we developed a model of virus transmission by a vector population which predicts the number of potentially infective bites under a range of temperatures and entomological parameters, including the daily survival rate of females, the interval between bites and the extrinsic incubation period. Results show that in a stable population, an increase in mosquito longevity disproportionately enhances the number of potential transmissions (e.g. by as much as five times when the survival rate rises from 0.80 to 0.95). Halving the length of the biting interval with a 10-°C rise in temperature increases the transmission rate by at least 2.4 times. Accordingly, the model can predict changes in dengue transmission associated with short-term variation in seasonal temperature and also with potentially long-lasting increases in global temperatures.

Key words. Bloodmeals, daily survival rate, dengue transmission, extrinsic incubation period, gonotrophic cycle, infective bites, model, temperature, vector.

Introduction

Dengue fever is an arboviral disease, caused by infection with one or more of the four dengue virus serotypes. Dengue virus is transmitted by *Aedes* sp. (Diptera: Culicidae) mosquitoes and restricted to tropical areas, where seasonal and geographical distributions are dependent on climate. Concerns regarding the impact of global warming on vector-borne diseases (Jetten & Focks, 1997; Russell, 1998) have intensified interest in the relationship between temperature and dengue fever incidence and include a focus on determining whether climate factors

alone can be used to indicate or predict variations in dengue incidence (World Health Organization, 2004). However, even if epidemiological surveys show that in endemic situations trends in incidence are generally driven by variations in seasonal climate, these changes in incidence depend on many parameters and the impact of temperature alone cannot be isolated easily from that of other climatic (e.g. rainfall, humidity) and non-climatic (e.g. herd immunity) factors (Watts *et al.*, 1987; Reiter, 1988; Focks *et al.*, 1993a; Kuno, 1995; Patz & Reisen, 2001).

Another approach to the evaluation of temperature effects is to focus on the change in the transmission capacity

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of a vector population subject to meteorological variation. In this study, we used a simple model to estimate the effect of temperature on the potential rate of transmission (number of infective bites) of a vector population with distinctive biological parameters, including: daily survival rate (DSR); duration of the gonotrophic cycle (GC; the time taken to digest a bloodmeal and for eggs to mature between two bloodmeals), and the length of the extrinsic incubation period (EIP; the period of time after an infectious bloodmeal before a female can efficiently transmit the virus, which includes virus replication, maturation and migration within the mosquito body to its salivary glands). Once the EIP has elapsed, females remain infective for the rest of their lives and are able to transmit the virus with each subsequent bite. Other values likely to modify transmission (e.g. probability of biting a human, distance to host, choice of different ages of hosts, etc.) are taken to be constant.

These biological parameters of a vector population reflect adaptation to the pathogen and exhibit geographical variation (Gubler & Rosen, 1976; Gubler *et al.*, 1979; Vazeille *et al.*, 1999). The DSR, GC duration and EIP in *Aedes aegypti* L., and the responsiveness of these variables to different temperatures, have been estimated through field and laboratory studies (Christophers, 1960; Sheppard *et al.*, 1969; Pant & Yasuno 1973; Watts *et al.*, 1987; Focks *et al.*, 1993a, 1993b). Although studies of the duration of the EIP in natural environments are rare, the rate of dengue virus multiplication has been experimentally measured through virus isolation or virus detection by polymerase chain reaction (PCR) from salivary glands, head, legs, midgut or full body and by transmission to monkeys (Watts *et al.*, 1987). We evaluate the effect on virus transmission of changes in the duration of the EIP caused by temperature changes.

Quantifying the contribution of the biological parameters investigated in this study to the vectorial capacity (VC) of a vector population, independently of other factors, will help us to understand the mechanisms of seasonal variations in dengue incidence. Moreover, estimating the impact of temperature on the potential rate of transmission will also elucidate how unusual seasonal cycles and global warming may affect the incidence of dengue and other arboviral diseases.

Materials and methods

A simulation model was used to investigate the effect on the transmission potential of a hypothetical population of dengue vectors of several biological parameters, including DSR, length of the GC and duration of the EIP, all of which are affected by temperature to some degree. Our model of dengue virus transmission used the standard range of observed values for the DSR (0.80–0.95) plus a maximum survival rate greater than normally observed to estimate the limits of possible future climate scenarios (Christophers, 1960; Sheppard *et al.*, 1969; Pant & Yasuno, 1973; Trpis & Hausermann, 1986; Focks *et al.*, 1993a, 1993b; Reiter, 1996).

In the basic model, 100% of a mosquito population (initially 100 females) is infected with dengue virus by the first

bloodmeal on day 0. These female mosquitoes continue to be infected for the remainder of their lives, to a maximum of day 45, corresponding to the maximum likely longevity of a female in the field (Christophers, 1960). The effects of mosquitoes becoming infected only in the second or third bloodmeal are also tested. A bloodmeal is defined as infectious if the female mosquito becomes infected and the parasite successfully multiplies (transmission from host to vector). Infective bites (IBs) are defined as those that can lead to infection of the host (e.g. of humans) after the first infectious bloodmeal is taken by the mosquito and the EIP is completed (transmission from vector to host). Here, we assume that the number of IBs reflects the transmission potential of a vector population. The number of surviving females is calculated each day. The numbers of bloodmeals and IBs are calculated at GC intervals, which is the estimated time interval (in days) between bloodmeals.

Female survival

The range of DSR values used by the model are values reported in published epidemiological studies (0.80–0.95; Focks *et al.*, 1993b). Daily survival rate was kept constant over the maximum 45-day lifetime of a female. The formula giving the number of surviving females at day n is:

$$\text{number of females at day } n = 100 \times \text{DSR}^n \quad (1)$$

Number of bloodmeals

The length of the GC in the model varies as integers from 2 to 8 days (in 1-day intervals) corresponding to the range observed in tropical areas (Pant & Yasuno, 1973; Haramis & Foster, 1990; Chadee *et al.*, 1993). The result is expressed as the sum of the number of bloodmeals (BM) taken in relation to each GC interval by surviving females:

$$\begin{aligned} \text{BM} = & \text{number of females at day 0} \\ & + \text{number of females at day GC} \\ & + \text{number of females at day } 2 \times \text{GC} \\ & + \dots + \text{number of females at day } m \times \text{GC}, \end{aligned}$$

where $m \times \text{GC} \leq 45 < (m + 1) \times \text{GC}$ [so there are no more females at day $(m + 1) \times \text{GC}$].

Hence,

$$\begin{aligned} \text{BM} &= 100 \times \text{DSR}^{0 \cdot \text{GC}} + 100 \times \text{DSR}^{1 \cdot \text{GC}} \\ &+ 100 \times \text{DSR}^{2 \cdot \text{GC}} + \dots + 100 \times \text{DSR}^{(45/\text{GC}) \cdot \text{GC}} \\ \text{BM} &= 100 \times \sum_{n=0}^{n=45/\text{GC}} \text{DSR}^{n \cdot \text{GC}} \\ &= 100 \times \frac{(1 - \text{DSR}^{(45/\text{GC}+1) \cdot \text{GC}})}{1 - \text{DSR}^{\text{GC}}} \quad (2) \end{aligned}$$

where integral values of $45/\text{GC}$ are used, as we consider only whole-day values.

Number of infective bites

The EIP refers to the time needed for the virus to reach the salivary glands and can range from 4 days to 24 days (Richardson *et al.*, 2006; Salazar *et al.*, 2007). However, transmission can only take place from the first bite after the EIP. We use an operational approach to the EIP, called EIP_{op} , by defining it in terms of the number of GCs (Fig. 1). Although the GC varies from 2 to 8 days, we generally restricted the values of EIP_{op} to the range of 2 GC to 4 GC and do not consider larger values for EIP_{op} , which could lead to unrealistic lengths of EIP. The potential number of IBs is considered to be equivalent to the sum of bloodmeals taken at GC intervals by surviving infective females (i.e. starting at the bloodmeal corresponding to the end of the EIP_{op} , or the first infective bite):

$$IB = 100 \times \sum_{n=EIP_{op}}^{n=45/GC} DSR^{n \times GC} = 100 \times \frac{DSR^{(EIP_{op} \times GC)} - DSR^{(45/GC+1) \times GC}}{1 - DSR^{GC}}. \quad (3)$$

The fact that feeding can be interrupted (i.e. a full bloodmeal can require several bites and as a result lead to several occasions for transmission) is not explicitly modelled. As a consequence, the IB, as defined here, leads to a minimal estimate of the transmission potential of a vector population.

Effect of delays in mosquito infection

Females may not obtain an infection from their first bloodmeal. The first IB is delayed, effectively, by the number of uninfected bloodmeals. In this case, to estimate the number of IBs, the sum in Eqn (3) starts at the numeric value of the number of bloodmeals before the mosquito obtains an infection + EIP_{op} . To determine the effect of not obtaining an infection from the first bloodmeal on the overall infective potential of the mosquito population, the results of a delay in infectious bloodmeals is compared with the results without a delay. The

value of the IB(delayed) : IB(no delay) ratio provides an estimate of the effect of a delayed IB for all mosquitoes on the overall transmission potential (IB) of the mosquito population: the greater the delay, the lower the transmission potential.

Effect of temperature variation

Based on data provided by laboratory and field studies of the effect of different temperatures on the length of the GC for individual mosquitoes (Christophers, 1960; Sheppard *et al.*, 1969; Pant & Yasuno 1973; Focks *et al.*, 1993a), the model assumes that the GC doubles for every decrease of 10°C in temperature. Assuming a homogenous effect of temperature, the GC increases by 1.07 ($= 2^{1/10}$) times per 1-°C decrease in temperature. The EIP is also known to be affected by temperature, but this relationship has rarely been quantified for dengue vectors (Watts *et al.*, 1987) and we have no precise knowledge about the relationship between the GC, EIP and temperature. Therefore, for the purposes of this study, the model assumed the relationship:

$$EIP_{op} = 2GC.$$

In this part of the study, we also assumed the DSR to be constant and equal to 0.90 as this value is the average of observed values found in the literature and is not affected by a wide range in temperature (20–34°C).

Results

Daily survival rate

The number of surviving females over time for the different values of the DSR is shown in Fig. 2. For a DSR of ≤ 0.90 , about 50% of females die before day 7 and most of them are thus unlikely to participate in virus transmission, given a GC of 2–8 days and an EIP of 4–24 days. The number of IBs increases non-linearly with increasing DSR, and decreasing GC and EIP (Fig. 3). A decrease in the DSR can strongly

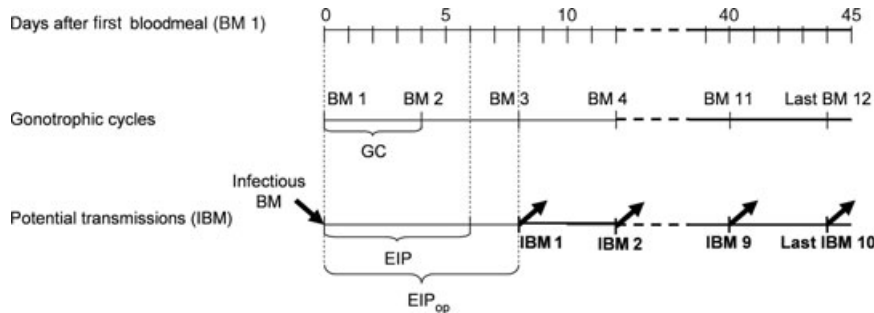


Fig. 1. Diagram of variables used in the model. In this example, the infectious bloodmeal (\searrow , transmits pathogen to the mosquito) is the first bloodmeal taken by the mosquito, the maximum longevity of a mosquito = 45 days, the gonotrophic cycle duration = 4 days, the extrinsic incubation period (EIP) = 6 days, and the operational EIP (EIP_{op}) = 8 days (i.e. = 2 GC). BM, bloodmeal; GC, gonotrophic cycle; IB, infective bite; IBM, infectious bloodmeal; (\nearrow , transmits pathogen to host); bold text indicates the period during which a mosquito can potentially transmit the dengue virus.

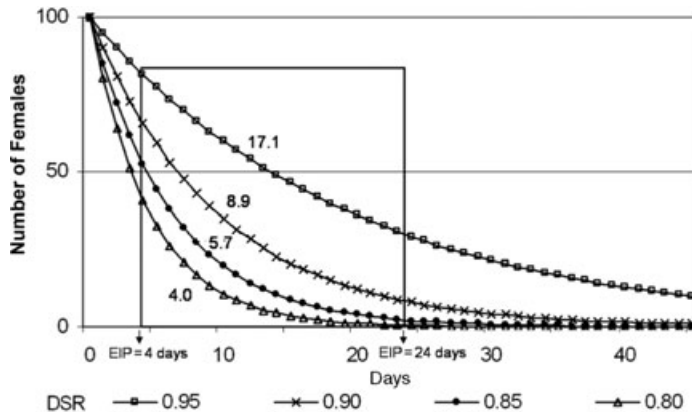


Fig. 2. Daily number of surviving females after the first bloodmeal. Number of survivors is calculated according to a constant daily survival rate (DSR) of 0.80, 0.85, 0.90 or 0.95. The square indicates the range in numbers of females out of 100 that reach the age at which they can transmit the virus to hosts, given extrinsic incubation periods (EIPs) of 4–24 days. Numbers by each curve give the life expectancy of the population in days.

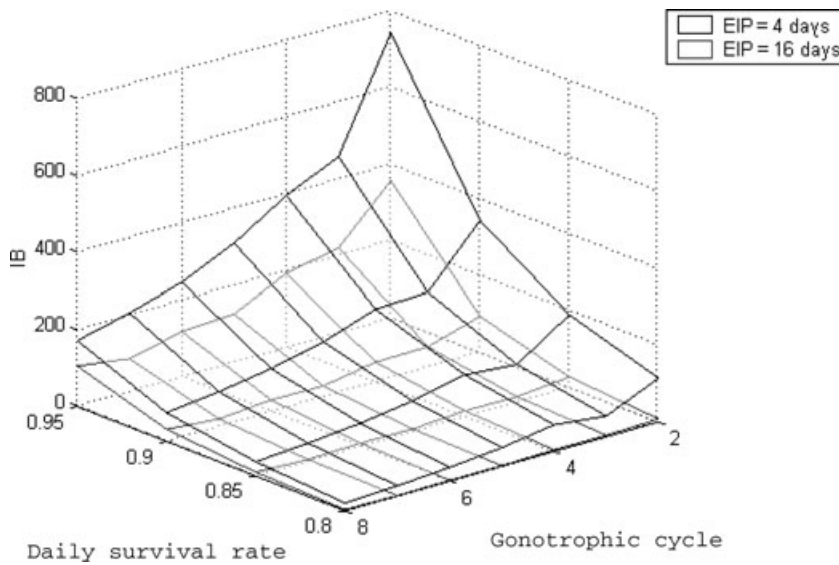


Fig. 3. Number of infective bites (IBs) taken by a virtual population. A virtual population initially of 100 females is followed under varying daily survival rates (DSR: 0.80, 0.85, 0.90, 0.95), gonotrophic cycle durations (GC: 2–8 days) and extrinsic incubation period durations (EIP = 4 days and 16 days).

reduce the transmission potential of a vector population: when the DSR decreases from 0.95 to 0.80, the total number of IBs is reduced by a factor of 5.7 when the EIP = 4 days, to 73.9 when the EIP = 16 days, also depending on GC (Table 1). This effect is more marked the later the first IB occurs because a low survival rate means that fewer females survive long enough to make an IB. As a result, we observe a strong effect of the DSR for long EIPs (16 days), and for values of the GC that delay the first IB (e.g. if EIP = 16 days, for GC = 5 days and GC = 7 days, this implies a first IB at days 20 and 21, respectively). In addition, the shorter the GC, the more often the reduction in surviving mosquitoes is counted in the sum of infective bloodmeals, so the larger the effect of DSR on the cumulative sum of IBs at GC intervals. We thus observe large reductions in the number of IBs caused by decreases in the DSR for ‘moderate’ values of the GC.

Effect of a delay in mosquitoes obtaining an infection

Figure 4 shows the relationship between the decrease in transmission potential with increasing delays in mosquitoes

Table 1. Effect of a large decrease (from 0.95 to 0.80) in daily survival rate (DSR) on transmission potential, expressed as the factor by which the number of infective bites (IB) is reduced.

GC, days	Proportional decrease in IB with decrease in DSR from 0.95 to 0.80	
	EIP = 4 days	EIP = 16 days
2	6.5	45.4
3	8.5	59.3
4	5.7	40.2
5	6.3	72.7
6	6.9	48.3
7	7.7	73.9
8	8.5	31.2

The proportional decrease in the number of IBs resulting from a decrease in the DSR from 0.95 to 0.80 [$IB(DSR = 0.95) : IB(DSR = 0.80)$], for different lengths of GC (= 2–8 days), given EIPs of 4 or 16 days. Ratios are highly dependent on the EIP, with a large decrease in potential transmission caused by reduced DSR when the EIP is long.

IB, infective bite; DSR, daily survival rate; EIP, extrinsic incubation period; GC, gonotrophic cycle.

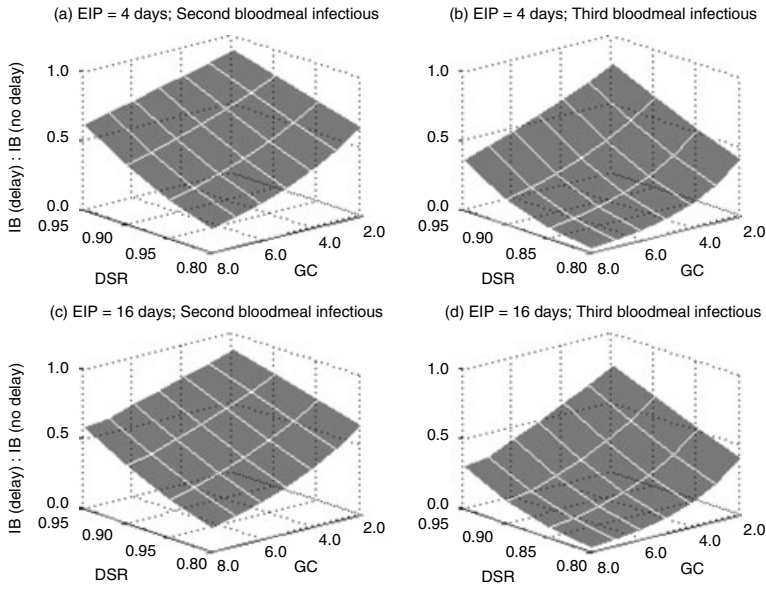


Fig. 4. Effect of a delay in the infectious bloodmeal; ratio of numbers of infective bites (IBs) with and without a delay in female mosquitoes obtaining an infection. A reduction in the average rate of transmission (number of IBs) is observed if the infectious bloodmeal is the second [IB(delay) in (a) and (c)] or the third [IB(delay) in (b) and (d)] compared with the first [IB(no delay)]. The figure shows the average IB(delay) : IB(no delay) ratio calculated for extrinsic incubation periods (EIP) of 4 days [(a) and (b)] or 16 days [(c) and (d)] and for different combinations of daily survival rate (DSR) and length of gonotrophic cycle (GC).

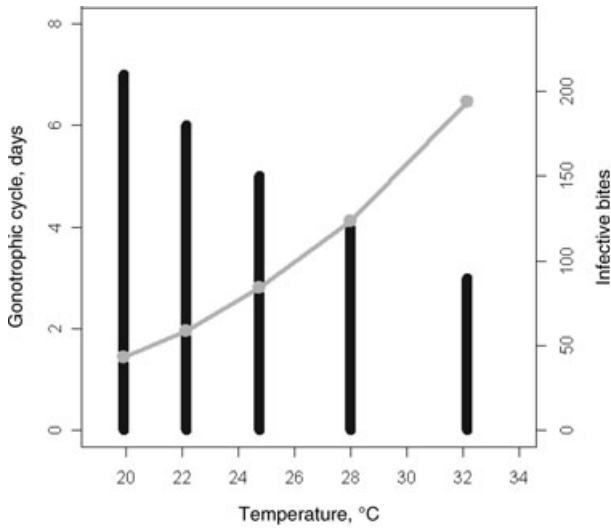


Fig. 5. Effect of changes in temperature on virus transmission. Changes in the duration of the gonotrophic cycle (GC; bar plots) caused by changes in temperature induce changes in the number of infective bites (IBs; grey line and circles) in a stable population of vectors, for DSR = 0.90 and EIP_{op} = 2 GC.

obtaining the first infectious bloodmeal and the other parameters, namely, DSR, GC and EIP. We observe that the length of the EIP has little effect on the IB(delayed) : IB(no delay) ratio; there is little difference between (a) and (c), or between (b) and (d). By contrast, this ratio is strongly dependent on the number of bloodmeals it takes for a mosquito to become infected; compare (a) and (b), or (c) and (d). Depending on these parameters, the number of IBs can be reduced by a factor of between 1.1 (second bloodmeal infectious, DSR = 0.95 and GC = 2 days) and 36.5 (third bloodmeal infectious, DSR = 0.80 and GC = 8 days).

Temperature effects

Integer values of the GC according to our underlying assumption concerning the dependence of GC length on

temperature are represented in Fig. 5. The grey curve represents the number of IBs calculated for each of the associated GC values, based on an EIP_{op} = 2 GC and a DSR = 0.90. An increase in temperature of 10°C reduces the GC by 50%, resulting in an increase in the mean number of IBs by a factor of 2.4–8.4. This factor is larger for lower values of the DSR and for greater GCs (i.e. at lower temperatures) (Table 2). The effect of a change in temperature of dT degrees on IBs ranges between $1.09(= 2.4^{1/10})^{dT}$ and $1.24(= 8.4^{1/10})^{dT}$ according to the values of DSR and GC between T and $T + dT$.

Discussion

The rate of circulation of virus transmitted by arthropod vectors is governed by the frequency of bites by infective vectors, which itself depends on the number and infection status of

Table 2. Ratio of the numbers of infective bites (IBs) for increased length of gonotrophic cycle (GC).

DSR	Proportional decrease in IB with increase in GC		
	GC = 2–4, days	3–6, days	4–8, days
0.80	4.0	5.8	8.4
0.85	3.3	4.3	5.6
0.90	2.8	3.3	3.9
0.95	2.4	2.6	3.0

IB(GC) : IB(2.GC) ratio of the numbers of infective bites for doubled GC, which can result from a decrease of 10°C in temperature. The lower the temperature, the longer the GC. We obtained larger ratios for lower DSRs and for longer GCs.

IB, infective bite; GC, gonotrophic cycle; DSR, daily survival rate.

the hosts. We studied the entomological component to roughly quantify the relationship between the transmission potential of a vector population and four physiological parameters: the DSR; the length of the period between two bloodmeals; the age at which the females are infected (delay before the infectious bloodmeal), and the length of the period between the infectious bloodmeal and transmission of the virus by a female (first infective bloodmeal). As temperature determines both the GC and the EIP, we elucidated the extent to which the transmission rate was governed by temperature.

In this study, we used computer simulations, which differ from the classical VC equation. This latter is usually expressed as (Macdonald, 1957):

$$VC = \frac{Ma^2p^x}{-\ln(p)}$$

where M is the man-biting rate or vector density in relation to man (here $M = 100$), a is the daily man-biting rate ($a = 1/GC$), p is the daily survival rate ($p = DSR$), and x is the duration of the sporogonic cycle ($x = EIP_{op} GC$). Note that in this equation, a^2 corresponds to the rate of infectious bloodmeals (mosquitoes' infections, at the rate a) multiplied by the rate of infective bloodmeals (potential humans' infections, at the rate a). In our study, in which all mosquitoes are assumed to become infected at the same (generally their first) bloodmeal, the rate of mosquitoes' infection (first a) is no longer relevant. With the parameter names we used throughout this study, the Macdonald equation would thus become:

$$VC = \frac{100 \cdot DSR^{EIP}}{-GC \cdot \ln(DSR)} \quad (4)$$

The main differences between our equation and the Macdonald model are that we consider that: (a) all females become infected at the same bloodmeal; (b) a female life cannot exceed 45 days, and (c) bloodmeals (and thus IBs) are reported at whole-day intervals, rather than at a continuous rate, as assumed by the classical equation, which uses the integral of bites performed per unit of time. In the classical equation the probability that a mosquito becomes infected is proportional to the biting frequency; however, in our model we assume that

every female mosquito becomes infected at the same bloodmeal, which enables us to disentangle the role of a delay in the infection of mosquitoes (Fig. 4) from the role of changes in the duration of the GC (Figs 3 and 5, Table 2), both of which are expressed through the biting frequency a in the classical equation. In agreement with the outcome of the classical equation, we find crucial effects of the DSR and the EIP on the number of IBs (Fig. 3, Table 1). However, for some ranges of parameter values, the results obtained with the Macdonald equation and ours may differ slightly. As the length of a mosquito's life is not given an upper limit in Macdonald's model, Eqn (4) would count bites performed by females after day 45. This would lead to an overestimate of the VC (equivalent to IBs), which would be larger for higher DSRs and longer EIPs. By contrast, in Macdonald's model the VC could be underestimated as a result of the assumption in Eqn (4) that the GC is based on a continuous biting rate, rather than the integer whole-day values used in our model, which is more consistent with the actual daily peak in biting activity of mosquitoes in the field. The magnitude of such an underestimate depends on the values of various parameters (results not shown). Lastly, our whole-day approach to the GC highlights the importance of considering the EIP_{op} , which depends on the EIP and on the GC, as IBs can only occur the day after the end of a GC and the completion of the EIP. This has important consequences for the results; for example, if females become infected by their first bloodmeal and the $EIP = 16$ days, the first IBs occur earlier for $GC = 8$ days (i.e. on day 16) than for $GC = 5$ days (on day 20). As a result, a decrease in DSR would have a larger effect for $GC = 5$ days (Table 1) because the induced reduction in the number of females is greater by day 20 than by day 16.

The DSR and its outcome, the mean age of a population, are key factors in evaluating the transmission risk associated with a given vector population (Holmes & Birley, 1987) because older females are more likely to be infective and therefore more dangerous than younger ones. This effect is highlighted in our model as the number of females reaching the minimum age to be infective is significantly reduced when the DSR is lower (Fig. 3, Table 1). This could be reinforced by an increase in mortality rate with mosquito age, recently shown for *Ae. aegypti* by Styer *et al.* (2007) and Harrington *et al.* (2008).

We also show that the age at which females become infected is an important parameter, especially when the DSR is low (Fig. 4). Gonotrophic discordance, or the need for female mosquitoes to take a second bloodmeal before they lay their first eggs, could favour vector infection at an early age. In addition, this phenomenon could induce a delay between bloodmeals taken by females with and without discordance. This complexity could be included in future models. The probability that a female becomes infected through a bloodmeal is also a function of the number of hosts carrying the virus. Prevalences in host populations are highly variable. For example, there was a 30-fold increase in dengue incidence during the 1991–1992 Florida outbreak compared with the incidence reported over the previous 2 years in the same population (Morrison *et al.*, 1998). A lower incidence means that fewer females become infected and less frequently at the first bloodmeal. Moreover,

vertical transmission of dengue virus from females to their offspring (Rosen *et al.*, 1983), although it leads to the infection of a low proportion of adults (<1%), could be an important factor to consider: if the first bites of the female are potentially infective, the transmission potential of the mosquito population could be increased, particularly in the rainy season when a large proportion of the vector population is made up of quiescent eggs and emerging females may already be infected (Joshi *et al.*, 2003). Because of their potential role in transmission, the effects of host prevalence and vertical transmission deserve further investigation.

Gonotrophic cycle duration is also important because of its effect on the number of IBs performed by *Ae. aegypti* (Figs 3 and 5, Table 2). A similar range of variation in GC duration is observed for some other *Aedes* dengue vectors, such as 5 days in field studies of *Aedes albopictus* Skuse (Neto & Navarri-Silva, 2004) and 3–4 days for *Aedes polynesiensis* Marks (Lardeux & Cheffort, 2002). Gonotrophic cycles are generally longer in temperate climates, ranging from, for example, 1–2 weeks for *Aedes communis* (DeGeer) in California to 14 days for *Aedes triseriatus* (Say) in Ohio (U.S.A.), making the probability of transmission very low in temperate areas (Eldridge & Reeves, 1990; Haramis & Foster, 1990). However, as an increase in temperature decreases the GC, it is expected that global warming will increase the probability of virus transmission by these species (Jetten & Focks, 1997; WHO, 2004) and permit the colonization of new areas by the main dengue vectors, *Ae. aegypti* and *Ae. albopictus*, (Tatem *et al.*, 2006).

Our model considers only a very simple relationship between GC, EIP and temperature. However, this relationship may be more complex because survival may also be affected by an interaction between temperature and humidity. For example, low humidity may lead to a decrease in the DSR and flight capacity (Christophers, 1960). These complex relationships also induce large seasonal fluctuations in the size of vector populations; in Bangkok the size of an *Ae. aegypti* population of females varies five-fold between the dry-cool and rainy seasons (Sheppard *et al.*, 1969; Focks *et al.*, 1993b). Moreover, the respective roles played by temperature and humidity must be disentangled because they may have antagonistic effects. For example, a short-lasting increase in temperature during the dry-cool season in Thailand might reduce dengue incidence, which is already low in this season, because, without a change in humidity, mosquito mortality is likely to increase or activity levels decrease. Rainfall during the cold season may cause an increase in the number of mosquitoes, but not in incidence, because the low temperatures would lengthen the GC. In order to produce realistic predictions, it is crucial that the roles of these different factors are investigated separately in our forthcoming modelling framework.

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