

GLOBAL-SCALE RELATIONSHIPS BETWEEN CLIMATE AND THE DENGUE FEVER VECTOR, *Aedes aegypti*

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Abstract. Considerable interest exists in the potential role climate may play in human health issues, especially regarding the effect of climate change on vector-borne disease. The *Aedes aegypti* mosquito, the principal vector for dengue, considered the most important vector-borne viral disease in the world, is particularly susceptible to climate variability and climatic change. Here we present a modeling analysis focusing on global-scale associations between climate and the development, potential distribution, and population dynamics of *Ae. aegypti*. We evaluate the model by comparing and contrasting model data with observed mosquito densities. There is good agreement between the observed and modeled global distribution of the mosquito; however, the model results suggest the potential for increased latitudinal distributions during warmer months. Seasonal fluctuations in mosquito abundance also compare well to observed data. Discrepancies possibly reflect the relatively low resolution of the climate data and model output and the inability of the model to account for local microclimate effects, especially in coastal areas. Future modeling efforts will involve study of interannual variability in mosquito dynamics.

1. Introduction

The relationships between climate and human health have recently captured much attention, both in the scientific community and the popular media (e.g., Environmental News Network, 1998; Epstein, 1994). Considering the effects of climatic variability and climate change upon the geographic distribution of disease vectors is a particular focus (e.g., Intergovernmental Panel on Climate Change, Chapter 18 Human Population Health, McMichael et al., 1996a). Specifically, several studies suggest projected climate changes may result in expanded geographic ranges for many mosquito-borne diseases, including malaria, yellow fever, and dengue (e.g., Jackson, 1995; Jetten and Focks, 1997; Longstreth and Wiseman, 1989; Martens, 1995; Martens et al., 1995, 1997; Monastersky, 1996; Patz et al., 1996, 1998; Reeves et al., 1994; Shope, 1991). Yet, the precise nature of these changes – the relationships between climate and disease vector ecology, and social, economic, and epidemiological factors involved in disease phenomena – remain unclear.

Socioeconomic factors, including adequate health care and sanitation, may affect the current geographic distribution and human incidence of many diseases more significantly than climate. Mosquitoes carrying malaria, yellow fever, and



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dengue, for example, have lived in the United States for centuries, but public education, mosquito control programs, piped water, and screened windows ensure epidemics remain an historical phenomenon (Gubler, 1998; Reiter, 1998). Accordingly, we must exercise care when examining the current geographic relationships between climate and disease occurrence.

Yet, the role of climate in disease ecology cannot be ignored. In particular, considering the fundamental mechanisms driving the relationships between climate and disease vector ecology is extremely useful. Only by examining the fundamental climatic and ecological components of disease phenomena can we differentiate between the biophysical and socioeconomic drivers of global disease patterns.

Climate may affect the *Aedes aegypti* mosquito, the principal vector for yellow fever and dengue, since increasing global temperatures and other associated climate changes may modify the mosquito's geographic range. In Colombia, for example, changes in the elevational distribution of *Ae. aegypti* have already been observed; previously limited to 1500 m, the mosquito has recently been found above 2200 m, presumably due to temperature increases (Suarez and Nelson, 1981). In Mexico, dengue cases have also been reported at previously unaffected higher altitudes (Herrera-Basto et al., 1992).

Several studies have already used global warming scenarios to examine the effects of climatic change on dengue transmission (Patz et al., 1998; Jetten and Focks, 1997; Martens et al., 1997). These models include some temperature-dependent calculations, such as adult mosquito survivorship and viral development. They also attempt to account for many temperature-independent, epidemiological factors, such as: the ability of an infected mosquito to infect a susceptible human and vice-versa, and the likelihood of infected persons to recover (Martens et al., 1997). Our research differs from this approach, as we focus our analysis specifically on the dengue vector, the *Ae. aegypti* mosquito.

Given the global distribution of *Ae. aegypti* and its presumed association with large-scale climatic events (Nicholls, 1993; Hales et al., 1996), we present an analysis of the global-scale relationships between climate and the ecology of the *Ae. aegypti* mosquito. In particular, our modeling analysis focuses on global-scale associations between climate and the development, potential distribution, and population density of *Ae. aegypti*. This kind of analysis provides a foundation for developing mechanistic models of the impact of climatic variability and climate change on disease ecology, epidemiological processes, and human health.

2. Dengue Fever and *Aedes aegypti*

The World Health Organization considers dengue to be the most important vector-borne viral disease, potentially affecting 2.5 billion people in tropical and subtropical countries throughout the world (WHO, 1999; Rigau-Pérez et al., 1998). Current estimates suggest that 50 to 100 million dengue cases occur annually, in

addition to 500,000 cases of the more serious, dengue hemorrhagic fever (Gubler, 1998; Pinheiro and Chuit, 1998; CDC website—<http://www.cdc.gov/travel/yellowbk/page107c.htm>).

Classical dengue fever, also known as breakbone fever, is characterized by headaches, fevers, sore muscles and joints, with occasional nausea/vomiting and rash; these symptoms may persist for several days. Dengue hemorrhagic fever (DHF), is a much more serious illness mainly affecting children and young adults. Symptoms include a sudden onset of fever and hemorrhagic manifestations that result in significant fluid loss and may lead to shock – dengue shock syndrome. Five percent of DHF cases are fatal (Gubler and Clark, 1995). A prior infection with one of the four dengue viruses results in a greater probability of contracting DHF. No vaccine currently exists for dengue.

The earliest reports of dengue fever epidemics date back to 1779–1780 in Asia, Africa and North America, indicating a widespread tropical distribution of *Ae. aegypti* during the past 200 years (Gubler and Clark, 1995). Since then, a global dengue pandemic, including the emergence of DHF, began in Southeast Asia after World War II. This pandemic intensified in the Americas during the 1980s with outbreaks in Caribbean and Latin American countries including Venezuela, Colombia, Brazil, French Guyana, Suriname, and Puerto Rico (Gubler and Clark, 1995). More recently, outbreaks of dengue have occurred in Brazil, Puerto Rico and in the western Pacific, including Vietnam, Fiji, Cambodia, Philippines, Malaysia, and Singapore (WHO, 1998).

The recent reemergence of dengue in the Americas has renewed current interest. During the 1950s and 1960s, the Pan American Health Organization (PAHO) directed *Aedes aegypti* eradication programs, eliminating the mosquito from most of Central and South America (Gubler and Clark, 1995). After the United States officially ended its eradication program in 1970, however, other nations discontinued their programs. Reinfestation of the mosquito in many countries resulted. Cuba, for example, experienced an unprecedented 344,000 cases of dengue and DHF in 1981 (PAHO, 1989).

A peridomestic (living in and around human dwellings), day-biting mosquito, *Aedes aegypti* feeds preferentially on human blood. It is also holometabolous, undergoing complete metamorphosis through egg, larval, pupal and adult stages. Climatic variables such as temperature and precipitation significantly influence the mosquito's development and survivorship (Bliss and Gill, 1933; Christophers, 1960; Focks et al., 1993a,b; Macfie, 1920; Rueda et al., 1990).

3. Model Description

To examine the effects of large-scale climatic conditions on this mosquito, we have developed a global-scale model driven by precipitation, temperature, relative humidity and solar radiation. We developed our model based on the CIMSIM mos-

quito model description of Focks et al. (1993a,b), which examines *Aedes aegypti* in relation to climatic variables at a specific site. Because we are working at a global scale, we have made some simplifications and changes to our model which differentiate it from CIMSiM. We refer the reader to Focks et al. (1993a,b) for a detailed reference and provide a very brief overview of our model, with most of the changes we have implemented, below. A more detailed discussion of some model components is presented in Appendices A and B.

Our modeling focuses on the global-scale relationships between climate and the development, population dynamics, and potential distribution of the *Aedes aegypti* mosquito. Our mosquito model runs on a daily timestep, across a 1 degree by 1 degree (~100 km per side) resolution global grid. Using cohorts to track the abundance (a function of survivorship), age, and development of the mosquito in its four life stages (specifically the egg, larval, pupal and adult stages (Figure 1)), the model simulates a life table of population structure.

Development Rates. Many entomological models use day-degree or temperature summation models that assume development rate is proportional to temperature. This suits modeling development within a limited temperature range. Sharpe and DeMichele (1977) developed an enzyme kinetics model appropriate for use over a significantly wider temperature range. In the model, a single rate-controlling enzyme determines development rates. Using the daily mean air temperature, a simplification of hourly water temperatures, we calculate daily development rates, for each cohort in its particular life stage (see Appendix A). Development accumulates each day and is complete when it reaches a specified level. In order to complete the larval stage, the larva must also reach a minimum weight. We calculate this using Gilpin and McClelland's (1979) differential equation, modified to account for the effect of temperature (Focks et al., 1993a). As a simplification at the global scale, we assume that food availability is not a limiting factor for larval weight gain and modify the equation accordingly (see Appendix B; Jetten and Focks, 1997). Fecundity (number of offspring) is a function of the adult female weight (see Appendix B).

Survival Rates. *Ae. aegypti* survival rates are temperature-dependent (Figures 1 and 2). Daily minimum and maximum temperatures, linearly approximated from the monthly values, determine survivorship rates. These rates are based on laboratory studies and anecdotal field observations, which could vary from what occurs in the field (Macfie, 1920; Bliss and Gill, 1933; Christophers, 1960). At temperature extremes we set the survival factor to 0.00 (instead of CIMSiM's 0.05), since these studies indicate that mosquitoes at any life stage are unlikely to survive extreme temperatures. Moisture also affects mosquito survival (Figure 3). Studies show eggs can survive desiccation for many months, but for hatching, water is necessary. We have simplified our model and assume that the mature egg will hatch if water is present (>10 mm) in the ovipositional container. Larvae require water for survival, but pupae do not, and vapor pressure deficit affects adult mortality (Christophers, 1960).

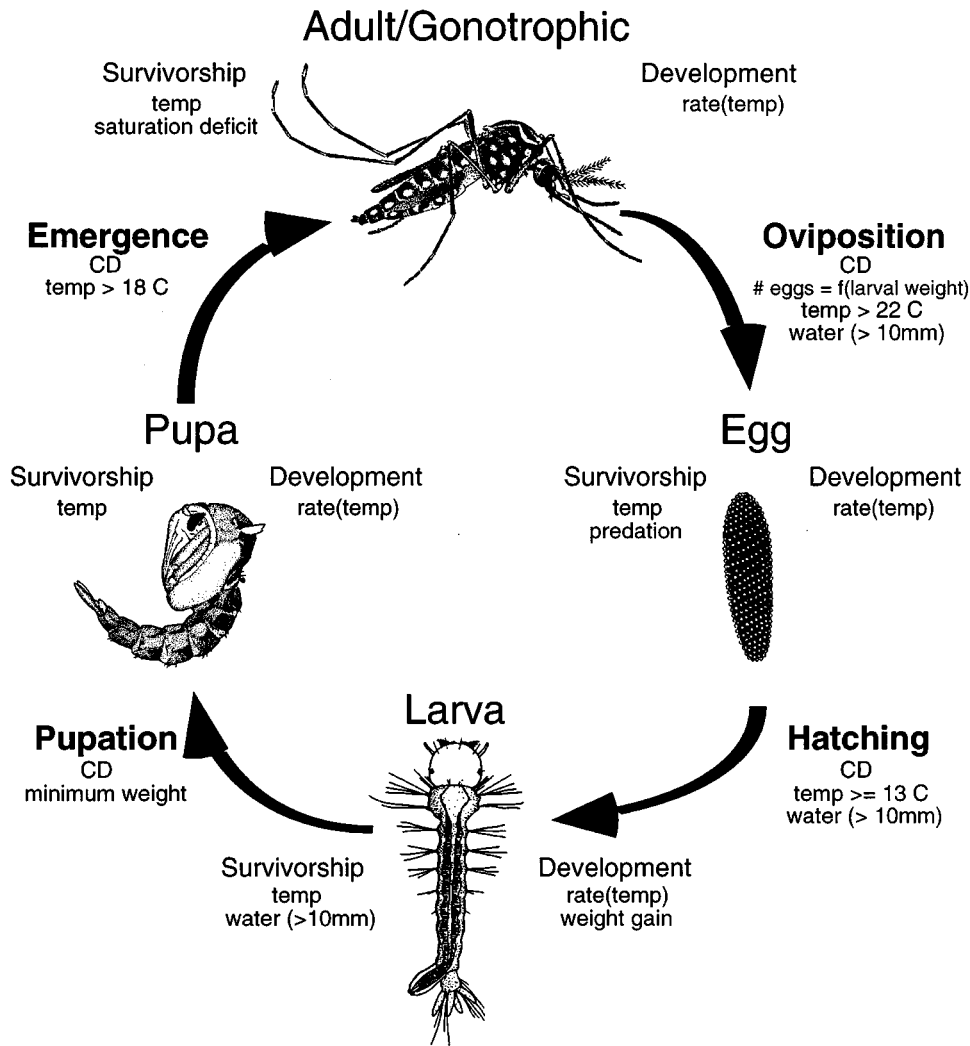


Figure 1. Each day the rate of development and survivorship for each cohort are calculated. Survivorship and development requirements are life stage-dependent. Metamorphosis is dependent on daily cumulative development (CD) and other life stage-specific factors. (Mosquito figures – Reprinted with permission from Samuel Richard Christophers, 1960, *Aedes Aegypti: Yellow Fever Mosquito*, ISBN 04638–6, Cambridge University Press, New York, p. 399, Fig. 49-I).

Climate Data. Global-scale, long-term average climate data (averaged between 1931–1960) for monthly-mean temperature, precipitation, and cloud cover (Leemans and Cramer, 1990; Cramer, personal communication) drive our model. Furthermore, we use long-term average (from 1958–1997) relative humidity data derived from the NCEP/NCAR reanalyzed meteorological dataset (Kalnay et al., 1996). Using a simple linear interpolation technique, the model converts the monthly-mean climatic data to quasi-daily values.

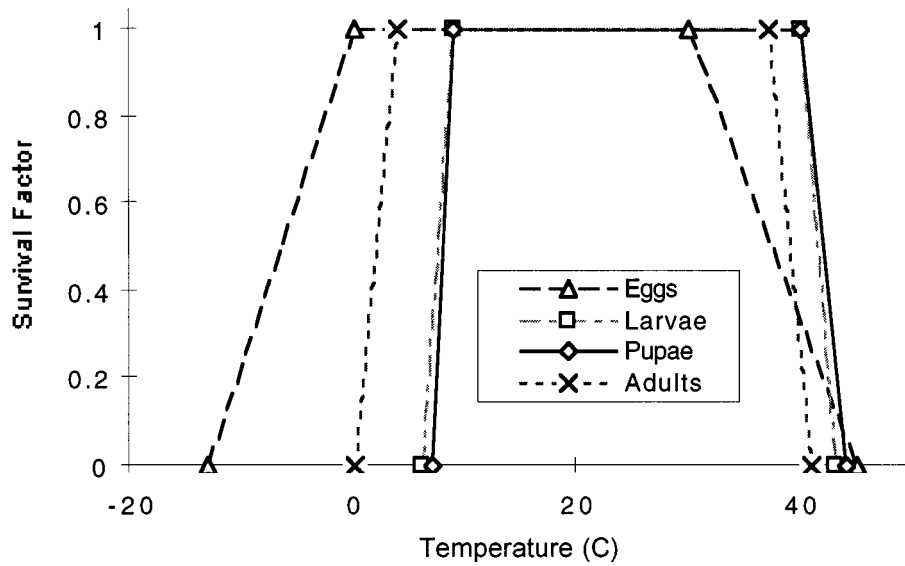


Figure 2. Temperature-dependent survivorship for the life stages of the *Aedes aegypti* mosquito.

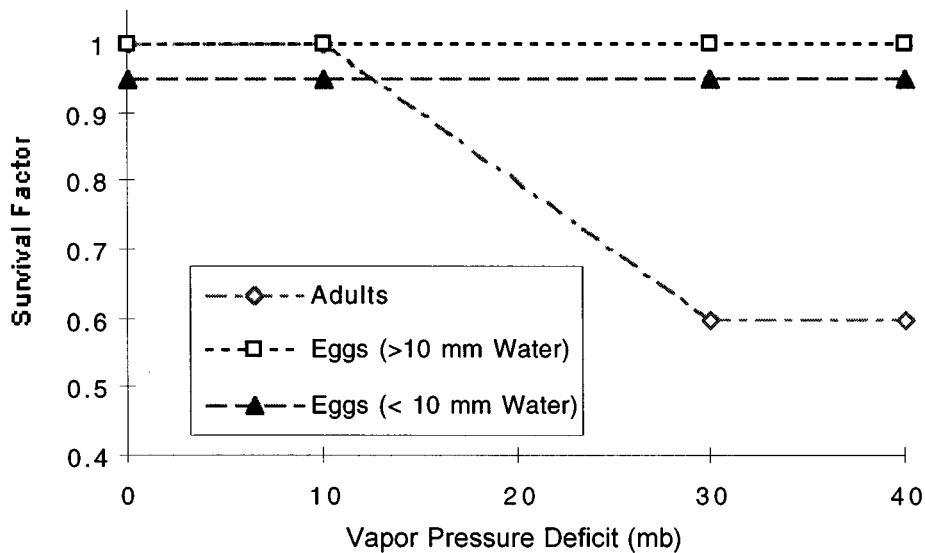


Figure 3. Mosquito-dependent survivorship for adults and eggs of the *Aedes aegypti* mosquito.

To account for water availability for oviposition (egg laying), egg hatching, and larval survivorship we use a hypothetical container in each grid cell, with a given depth of 20 cm. We chose this depth as an average depth between typical ovipositional containers, such as discarded bottles, tires and water storage drums. By excluding human population densities and socioeconomic factors that might determine the types and densities of appropriate ovipositional containers, we strictly

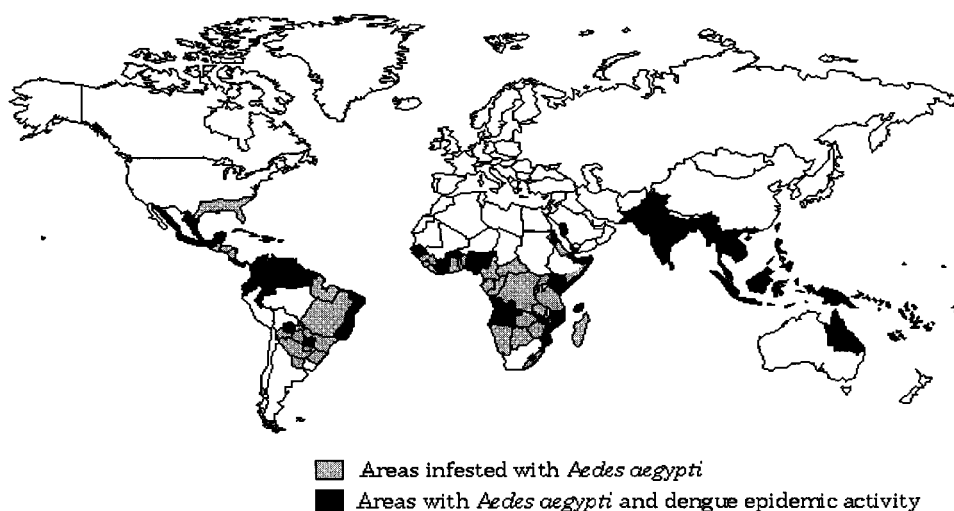


Figure 4. World Distribution of Dengue. Source: CDC web page: <http://www.cdc.gov/travel/yellowbk/figdeng.htm>

assess the effects of climate on mosquitoes. The number of mosquitoes in each grid cell is calculated per m^2 of container surface water.

We calculate the availability of water (in the hypothetical container) for mosquitoes, using a simple bucket model treatment of the surface water balance. Differences between precipitation and evaporation drive changes in water depth. Additional precipitation above the container's maximum depth (20 cm) runs off from the container. A simple energy balance approach (Prentice et al., 1993) simulates evaporation rates, where the net surface radiation load is estimated from empirical relationships for solar radiation and net infrared radiation (Linacre, 1986). The atmospheric vapor pressure deficit is calculated as a function of temperature and relative humidity.

4. Results

Geographic Distribution. One of the methods of evaluating this model is to compare and contrast the observed global distribution of the *Aedes aegypti* mosquito (Figure 4) to the modeled potential distribution of adults (Figure 5). Figure 5 shows the annual total number of mosquitoes, ranging from 0 to 10 adult females on our index; the index represents the density of mosquitoes per m^2 of container surface water. The modeled distributions correlate well with the actual distributions – mosquitoes do not appear in very dry or cold regions of the world. According to the model, however, the mosquitoes appear farther north in eastern and central North America and Asia (e.g., China) than the actual distribution and they appear in southern Europe. This suggests that if *Ae. aegypti* mosquitoes were introduced into

Total Annual Adult Female Density

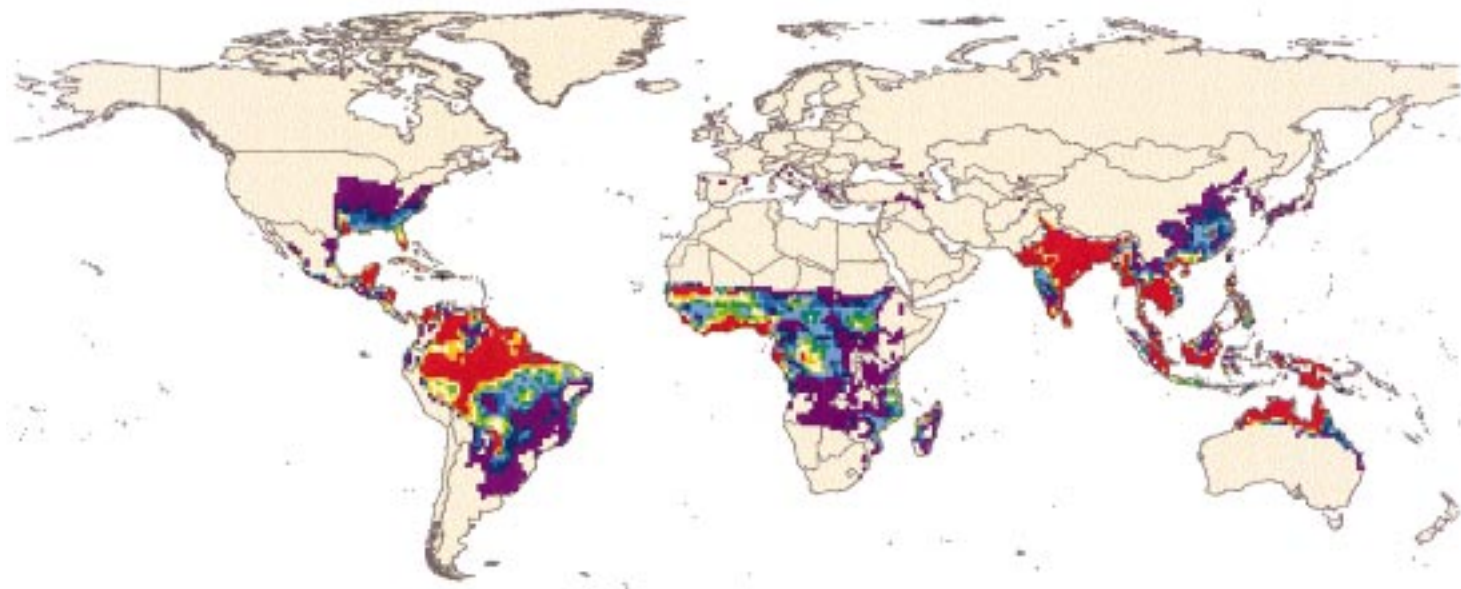


Figure 5. Total annual adult female density. The index represents the density of adult mosquitoes per m² of container surface water.

more northern latitudes during the summer, they could persist until cold weather arrived, excluding other socioeconomic factors affecting their persistence. It is also plausible that if the introduced mosquitoes were carrying dengue viruses, a dengue epidemic could occur. This scenario is not unprecedented: dengue epidemics occurred in port cities such as Philadelphia, Pennsylvania during the late 18th century (Shope, 1991) and Athens, Greece during the early part of this century (Halstead and Papaevangelou, 1980).

Though the modeled output does not indicate the presence of *Ae. aegypti* in desert regions of the world (e.g., Somalia, Eritrea, Namibia, Yemen, northeast Brazil, and parts of Saudi Arabia), their persistence in such conditions is observed (Figure 4). The lack of precipitation in these regions has probably limited the modeled mosquito densities. People living in dry regions tend to store water in and around their homes, providing ample breeding grounds for this domestic species (Shope, 1991). When we fix a minimum water depth in the model, mosquitoes persist throughout the hot and dry regions of the world.

Seasonal Variations. Accurately modeling the temporal dynamics of the population is an important objective of this study. Figures 6a–d display the modeled seasonal variations in density and distribution of the adult female mosquito for the months of January, April, July and October, respectively. The change in the mosquito's distribution during the year is apparent – as expected, during warmer months the mosquito's latitudinal range increases, as does the density.

To evaluate the model, we obtained observed mosquito densities from the literature, as well as an unpublished data set from the Centers for Disease Control (CDC). The CDC data set contains ovitrap data for various southeastern United States cities from 1981 to 1985 (Division of Vector-Borne Diseases, Centers for Disease Control, U.S. Public Health Service, Fort Collins, Colorado). The model outputs the total number of *Ae. aegypti* (e.g., eggs, larvae, pupae and adults) per m² of container surface water per month. We compare the relative seasonal densities of observed data to model output (Figures 7–10). When more than one year's worth of observations was present (e.g., CDC data and Galveston, Texas), and/or data from several sites in one city available (e.g., CDC data contains 15 sites per city), we calculated monthly averages and used standard deviations to estimate between-site or between-year variations. These averaged monthly observations are compared to the model's monthly output. To facilitate comparison, the observed and modeled data are displayed on different scales because the observed values are obtained from various indices (e.g., Breteau and House) and sizes of water-holding containers (e.g., 1-liter ovitraps, bottles, tires or cisterns), whereas the model's 'container' has a depth of 20 cm.

In general, there is good agreement between the observed CDC data and model output, with correlation coefficient values ranging from 0.73 for Brownsville, Texas (Figure 7a) to 0.91 for Jacksonville, Florida (Figure 7h). The model reasonably reflects the observed seasonal population fluctuations at these locations. However,

there is little correlation between the model output and observed data in Miami, Florida ($r = 0.34$) (Figure 7i).

Possible explanations for the low correlation between the observed and modeled Miami data may be the poor resolution of the climate data and model output (1 degree by 1 degree), and the inability of the model to account for local microclimate effects, especially in coastal areas. Some coastal cities and islands (e.g., Charleston, S.C., Key West, Florida, Puerto Rico) in the observed data sets do not exist on the 1-degree grid, because the coast line is distorted due to the low resolution. Another factor to consider in interpreting these results is the relatively large standard deviations (between-site variations) in the observed data and the difference in the number of observations between summer months and late fall months. For instance, in July, New Orleans and Miami both have 21 observations; in November, New Orleans has 12 observations, but Miami's total drops to only 1 observation.

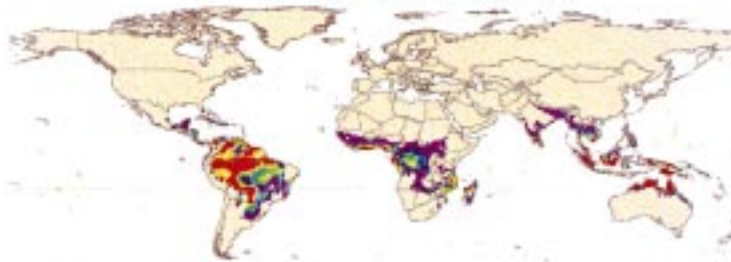
Ovitrap data collected in Galveston, Texas from 1977 to 1979 (Micks and Moon, 1980) were averaged and compared to the model output (between-year variation is indicated with error bars; Figures 9a–9c). The correlation coefficients vary from 0.37 (average number of eggs per ovitrap) to 0.71 (total eggs collected), depending on the index used. The observed data from these three years suggest a bimodal population distribution, but the model, run with average climate data, outputs a unimodal distribution.

Data collected from ovitraps in Trinidad in 1988 (Chadee, 1992) also suggest multiple peaks in *Aedes aegypti* abundances (Figures 10a,b). A weak correlation exists between these data and the model results, varying from 0.11 to -0.061 . The relatively small size of Trinidad compared to the model's resolution, as well as the lack of a distinct seasonal fluctuation in the mosquito densities, may account for this lack of correlation.

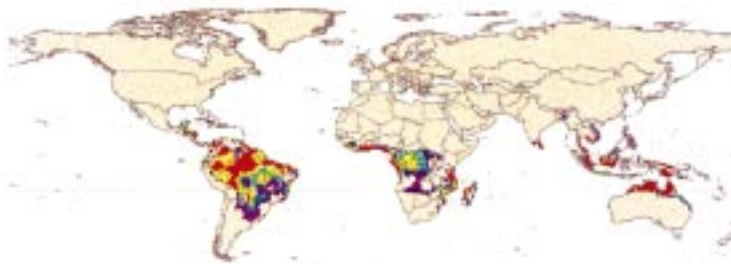
Because *Ae. aegypti* is peridomestic, conducting house-to-house surveys allows for evaluation of peridomestic breeding (Tun-Lin et al., 1996). Researchers use several different indices to measure densities, including the Breteau, House, and Larval Density Indices (Tun-Lin et al., 1996; Focks et al., 1981; Focks and Chadee, 1997; Beier et al., 1995). The Breteau Index sums the number of positive containers (the mosquito is present) per 100 houses; the House Index calculates the percentage of houses that are positive, and the Larval Density Index averages the number of immatures (larvae and pupae) per house. The correlations between the Breteau (Figure 8a) and House Indices (Figure 8b) and the modeled data for Mérida, Mexico (Winch et al., 1992) are good, being 0.85 and 0.88, respectively. The model underestimates the larval densities during the cooler months but predicts a small population peak during the early spring – an occurrence not evident in the observed data.

Figure 6 (facing page). Total adult female density for January, April, July and October, respectively. The index represents the density of adults mosquitoes per m^2 of container surface water.

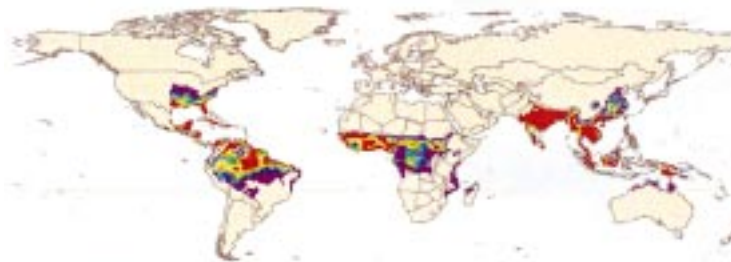
Total Adult Female Density



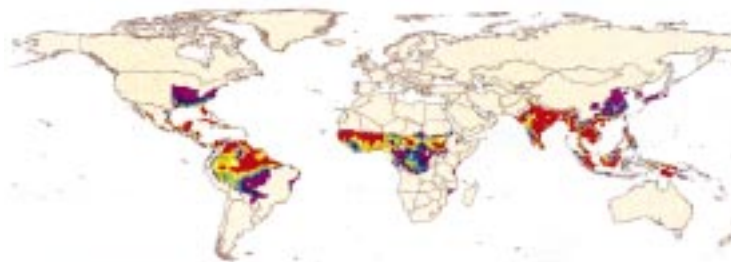
(a) January



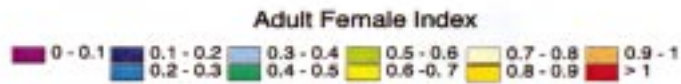
(b) April



(c) July



(d) October



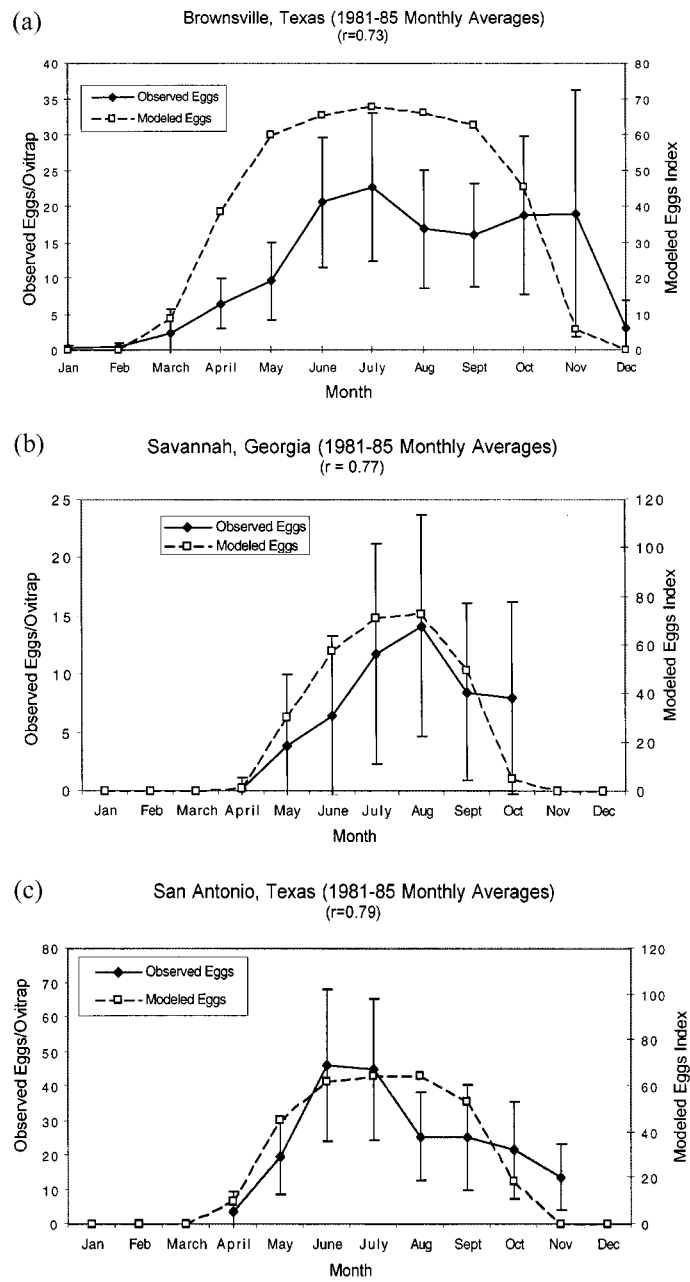


Figure 7a-c.

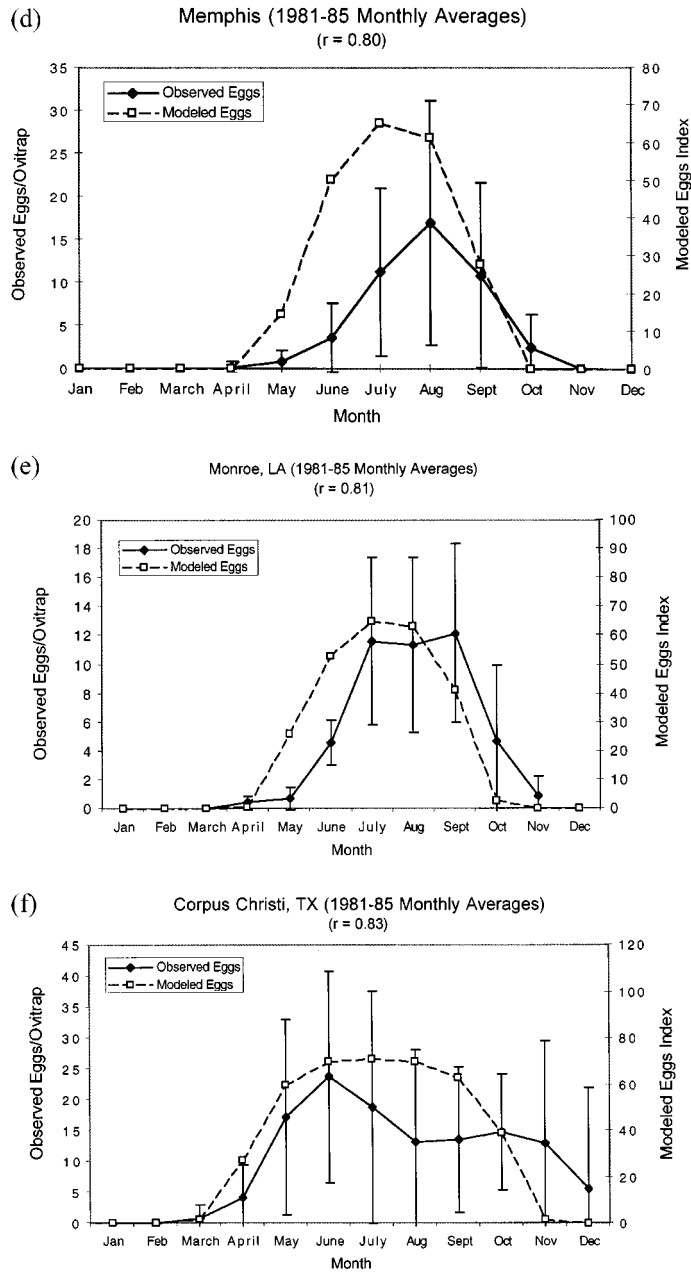


Figure 7d-f.

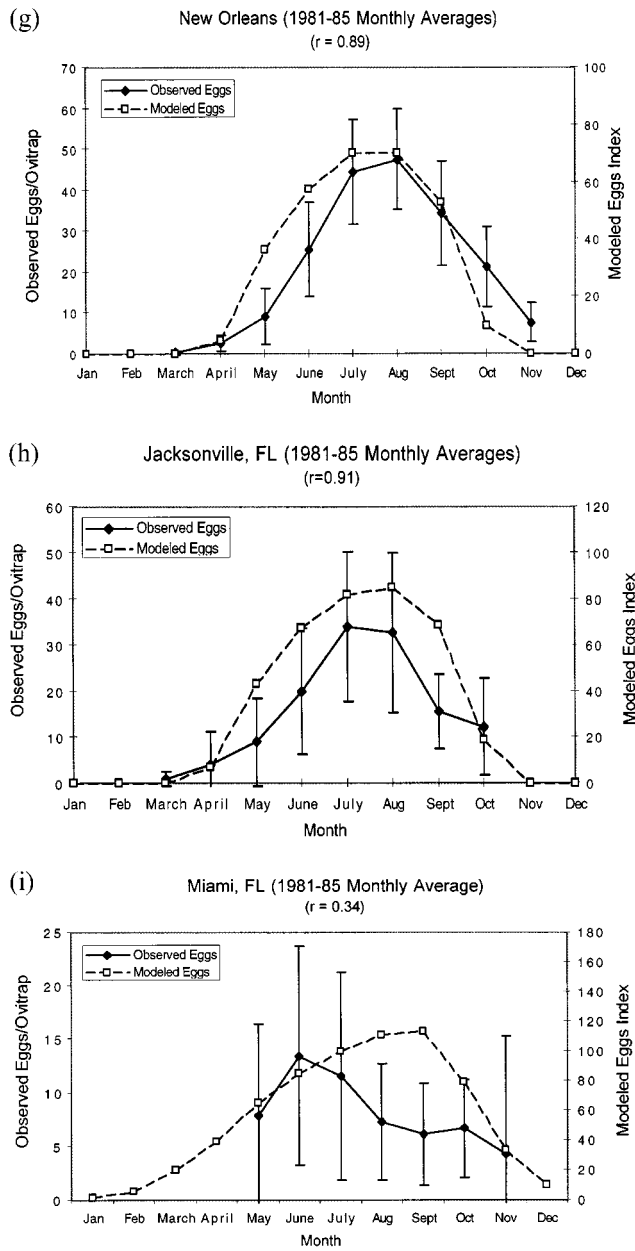


Figure 7g-i.

Figure 7. Modeled and observed (1981-1985 monthly averages) egg densities for Brownsville, Savannah, San Antonio, Memphis, Monroe, Corpus Christi, New Orleans, Jacksonville and Miami, respectively. The modeled egg index represents the density of mosquito eggs per m² of container surface water.

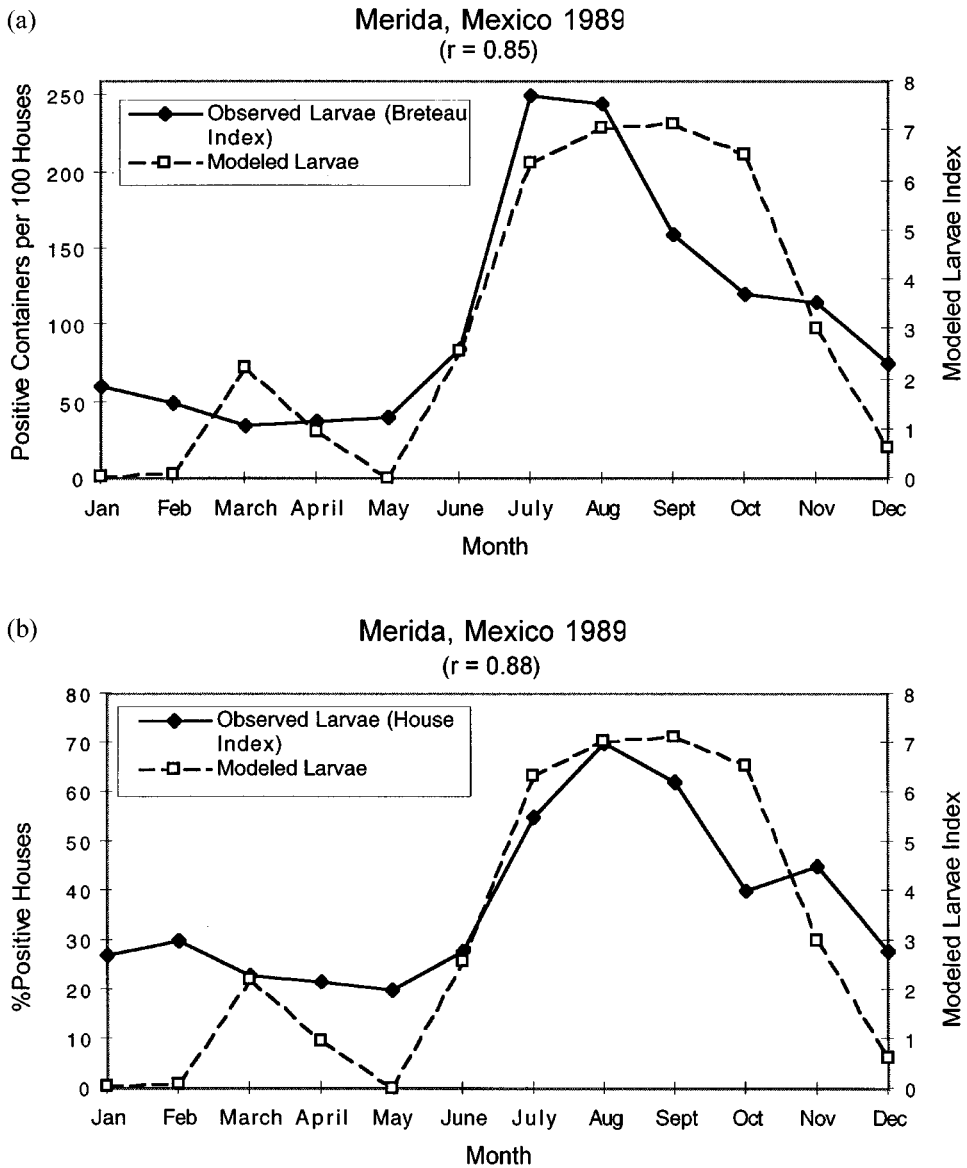


Figure 8. Modeled and observed (Breteau and House indices) larval densities for Mérida, Mexico (1989). The modeled larvae index represents the density of mosquito larvae per m^2 of container surface water.

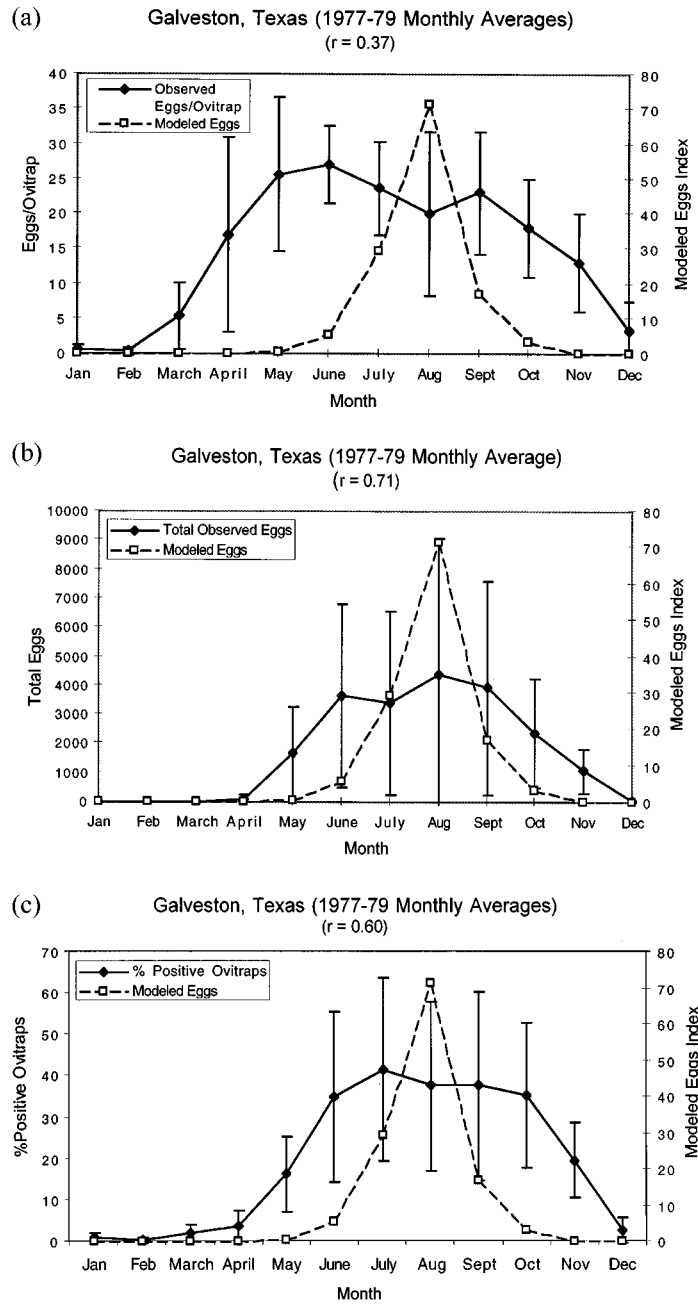


Figure 9. Modeled and observed (1977–1979 monthly averages) egg densities for Galveston, Texas. The modeled egg index represents the density of mosquito eggs per m² of container surface water.

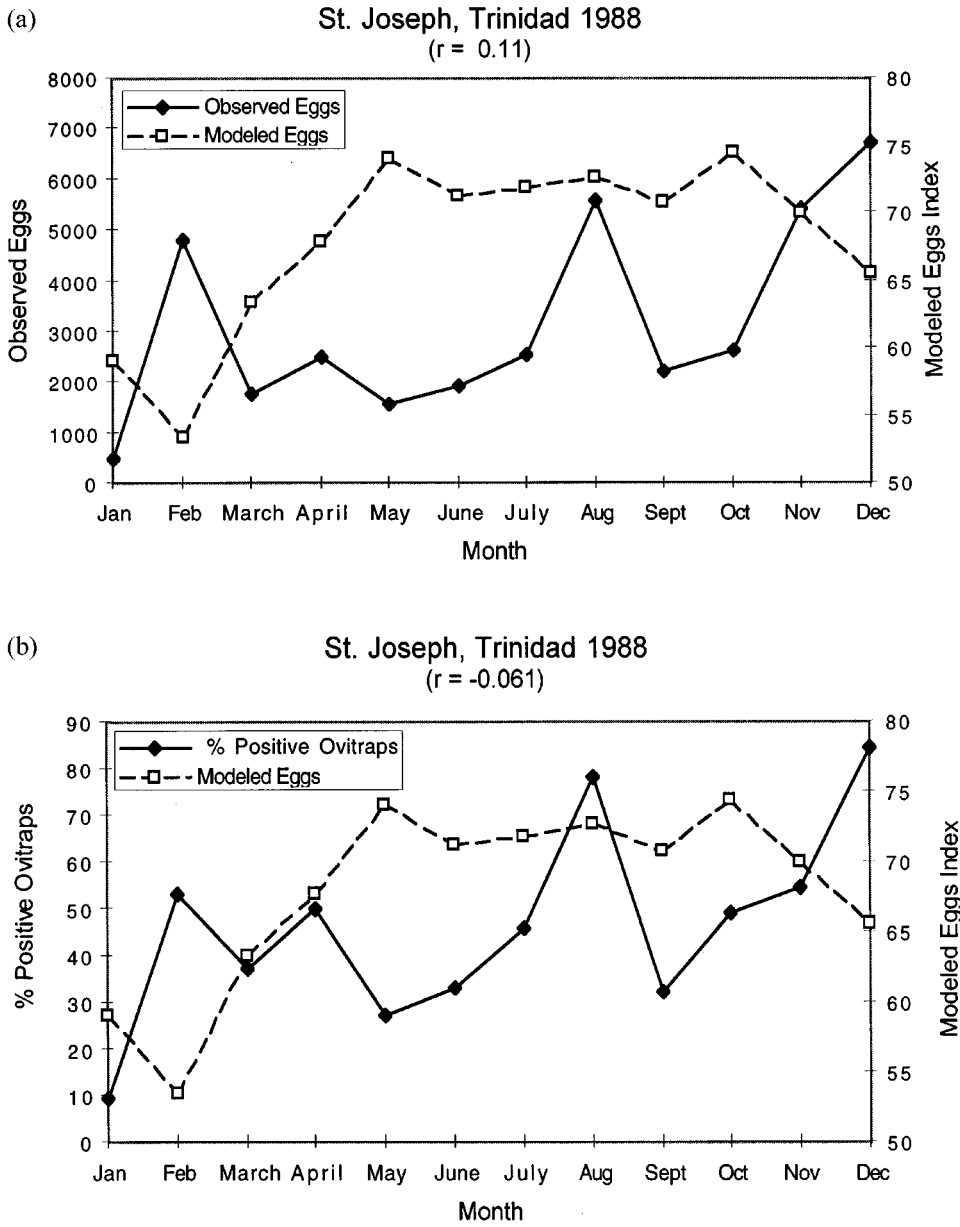


Figure 10. Modeled and observed egg densities for St. Joseph, Trinidad, 1988. The modeled egg index represents the density of mosquito eggs per m² of container surface water.

5. Conclusions

This study attempts to simulate the global-scale population dynamics of the *Aedes aegypti* mosquito, based solely on climatic conditions. However, it is clear that many socioeconomic factors affect the abundance and distribution of this significant disease vector. Local infrastructures that provide running water, (thus eliminating the need for water storage) as well as public education and mosquito eradication programs significantly affect *Ae. aegypti* populations. Yet, these model results support the view that a few climatic variables can explain much of the seasonal fluctuations in both geographic range and population abundance. Understanding the mechanistic basis for the relationships between climate and mosquito ecology is a necessary first step in elucidating the relationships between climate, disease ecology, epidemiology, and human health.

While the model appears to capture the basic climatic controls on the global-scale population dynamics of *Aedes aegypti*, there are several limitations to this approach. Using a fairly coarse 1-degree resolution grid, for example, appears to cause some problems in accurately modeling relatively small islands (e.g., Trinidad) and coastal areas (e.g., Miami and Galveston). However, the results from the more inland areas (e.g., Memphis, Jacksonville, and Mérida) suggest a reasonable ability to capture the seasonal variability of the mosquito population dynamics. We also recognize that we have simplified the potential breeding sites for the mosquitoes by designating a hypothetical 20 cm deep, 1 m² 'container' in each grid cell, likely affecting some of the results. Future work could incorporate other factors, such as population density and socioeconomic measures, to more accurately model potential ovipositional sites.

This study emphasizes the relationship between mosquito population dynamics and long-term average patterns of climate across the globe. Yet, strong interannual variations in climate may significantly affect disease vector ecology. Examining the impact of climatic variability on human-disease relationships is vital, especially in regions where a disease is already endemic.

A future application of this model could examine *Ae. aegypti* population densities during El Niño-Southern Oscillation (ENSO) events, versus non-ENSO years. Dengue, as well as other mosquito-borne diseases such as malaria and encephalitis, are thought to be strongly influenced by El Niño-Southern Oscillation events (Bouma et al., 1997; Bouma and van der Kaay, 1994; Hales et al., 1996; Nicholls, 1993). Understanding the relationship between past ENSO events and mosquito densities (as well as dengue epidemics) will provide clues to the behavior of present and future ENSO events.

TABLE I
Coefficients for the enzyme kinetics model of Sharpe and DeMichele (1977)
relating development rates to temperature

Stage or process	Parameters			
	$\rho(25^\circ\text{C})$ (h^{-1})	ΔH_A^\ddagger (cal mol^{-1})	ΔH_H (cal mol^{-1})	$T_{1/2H}$ (K)
Embryogenesis	0.01066	10,798.18	100,000.00	14,184.50
Larval development	0.00873	26,018.51	55,990.75	304.58
Pupal development	0.01610	14,931.94	-472,379.00	148.45
Gonotrophic cycle	0.00898	15,725.23	1,756,481.07	447.17

Appendix A

Using the daily mean air temperature, we calculate daily development rates ($r(T_t)$) for each cohort in its particular life stage. The enzyme kinetics model describes the rate of development by the equation:

$$r(T_t) = \frac{\rho(25C)(T_t/298) \exp[(\Delta H_A^\ddagger/R)((1/298) - (1/T_t))]}{1 + \exp[(\Delta H_H/R)((1/T_{1/2H}) - (1/T_t))]}.$$

Where, $r(T_t)$ is the rate of development (hour^{-1}) at temperature T (K) on day t . T , in our global model, is the mean daily air temperature. $\rho(25C)$ is the development rate (hour^{-1}) at 25°C assuming no inactivation of the rate-controlling enzyme. ΔH_A^\ddagger is the reaction's enthalpy of activation that is catalyzed by the enzyme (cal/mol); ΔH_H is the enthalpy change associated with high temperature inactivation of the enzyme (cal/mol); $T_{1/2H}$ is the temperature (K) at which 50% of the enzyme is inactivated from high temperature, and R is the universal gas constant ($1.987 \text{ cal/mol/deg}$). (Enthalpy is chemical energy referred to as the 'heat energy'; enthalpy is a system property that reflects its capacity to exchange heat with the surroundings. ΔH of a reaction is the difference in enthalpy between products and reactants (Brown and Rogers, 1987; Masterton and Hurley, 1989)).

We use the same equation parameters as used by Focks et al. (1993a) (Table I). These parameters were estimated from observed data using nonlinear regression and Marquardt's (1963) searching algorithm. An interactive parameterization process was used, with initial parameter estimates for $\rho(25C)$ (0.006 h^{-1}) and $T_{1/2H}$ (313 K) from McHugh and Olson (1982), and initial estimates for ΔH_A^\ddagger ($10,000 \text{ cal mol}^{-1}$) and ΔH_H ($100,000 \text{ cal mol}^{-1}$) taken from Schoolfield et al. (1981).

Development accumulates each day and is completed when the cumulative development (CD_t) is greater than 0.95 for the egg, larval and pupal stages, or when CD

$$CD_t = \sum_{t=0}^n r(T_t)$$

exceeds 1.00 for the first gonotrophic (maturation of reproductive organs that enables the female to lay eggs) cycle and increases by 0.58 for each additional gonotrophic cycle. The additional gonotrophic (or egg-laying to egg-laying) cycles are shorter, since the follicles are already at a more advanced stage of development than in newly emerged females (Macdonald, 1956).

Appendix B

In order to complete the larval stage, the larva must also reach a minimum weight. We calculate this using Gilpin and McClelland's (1979) differential equation, (shown below), modified to account for the effect of temperature (Focks et al., 1993a). At the global scale, we assume that food availability is not a limiting factor for larval weight gain and we modify the equation accordingly (Jetten and Focks, 1997).

$$\frac{dW(t)}{dt} = a * f(T_t)[W(t)^b - d_1 W(t)^{d_2}],$$

where $W(t)$ = dry weight of larva (mg) on day t ; a = assimilation rate of consumed food (30%); $f(T_t)$ = chronological basis of development as function of temperature; $W(t)^b$ = food exploitation rate as power of body weight ($b = 0.8$); $d_1 W(t)^{d_2}$ = metabolic requirements of the larva ($d_1 = 0.016$, $d_2 = 0.667$).

The minimum weight required for pupation when food is in excess is estimated as a function of temperature (Rueda et al., 1990). The average temperature for day t is T_t .

$$\text{Minimum weight} = 2.11 - 0.04 * T_t .$$

Fecundity (number of offspring) is a function of wet adult female weight based on observations by Bar-Zeev (1957) and Nayar and Sauerman (1975).

$$\text{Fecundity} = 46.5 * \text{wet adult female weight} ,$$

where the wet adult weight is calculated by multiplying the larval weight by 1.655 (Table 5 in Rueda et al., 1990).

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