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Commentary

Advances in developing a climate based dengue outbreak models in Dhaka, Bangladesh: challenges & opportunities

Dengue is a vector borne disease transmitted by *Aedes aegypti* and *Ae. albopictus*. The incidence of the disease is increasing worldwide and currently 40 per cent of the global population is at risk of the infection[1]. The number of cases in the WHO Southeast Asia region surged nearly 70 per cent from 152,448 in 2004 to 257,882 in 2009. There is no specific treatment of the disease and the only way to control the disease is through vector control, which includes removal or covering water harbouring containers, the use of insecticide treated bed nets and fogging with insecticides.

The risk of transmission varies in space and time and the dynamics of the disease is dependent on seasonal changes in weather and immunity. Transmission is particularly sensitive to rainfall, temperature and humidity, thus, dengue is a climate sensitive disease and in Asia the epidemics have been associated with the monsoon season[2].

There has been a need to develop early warning systems that can identify and quantify the risk of vector borne disease outbreaks such as dengue, malaria, Rift Valley fever, West Nile fever and more recently chikungunya. An early epidemic prediction tool is critical for evaluating the risk of an outbreak that enables an objective evaluation of the risk and evidence based decision making about interventions. Early identification of a climate risk allows early interventions and prevention of an epidemic instead of an epidemic management.

The development of early epidemic prediction models has been fraught with problems. Among these, the major problem is the availability of good quality epidemiological data. In addition, there is the issue of spatial representation of climate data. Only a few models have taken into consideration the spatial availability and stability of breeding habitats. It is commonly assumed that the total amount of rainfall has a direct relationship with the vector abundance and this may not be true. For example, the distribution of water containers in urban areas is dependent on the socio-economic development and availability of piped water supplies[3].

In many models, the biological significance of the meteorological data is not taken into consideration. For example, the populations of *Ae. aegypti* in Dhaka city, Bangladesh, do not increase until the rainfall threshold of 150 mm/month is exceeded. Subsequently, the use of data below this threshold in a regression model is biologically erroneous. It has been established that dengue epidemics are associated with rainfall in the range of 205-446 mm/month[3]. Likewise the development of *Ae. aegypti* does not occur below 17°C and may cease above 44°C. In contrast, the development of *Ae. albopictus* immature stages ceases below 10.4°C[4]. This suggests that the models must use different temperature thresholds for the two dengue vectors. It has been shown in the laboratory experiments that the *Ae. aegypti* only infected monkeys when the mosquitoes were maintained at >30°C[5]. Thus transmission may not have a statistical relation with temperate at <30°C[5]. The diurnal temperature range (DTR) has been shown to have a direct effect on the infectivity of *Ae. aegypti* with dengue virus. A high DTR range (20°C) reduced the probability of infection while a low range (10°C) increased the rate of infection[6]. One needs to consider that the relationship between temperature and the number of cases may be non-linear and this would affect a linear regression model.

These data suggest that regression models may indicate a poor association between the disease and temperature unless critical temperature thresholds are taken into place. The current work[7] indicates that

critical transmission for dengue in Dhaka city was only observed in the monsoon season but not in the pre- and post-monsoon periods. This observation explains the extremely low cases in the pre- and post-monsoon periods. A mean temperature of 31.9°C, rainfall of 349 mm humidity of 80 per cent and a low DTR were observed during the monsoon period but not in the pre- and post-monsoon periods. These factors lead to the climate model explaining 61 per cent on the variation in the dengue cases. In the case of malaria in the Western Kenya highlands, while climate conditions explained much of the temporal variation in cases it was not able to explain the spatial variations[8]. It was observed that sites with similar climates had an 8.5-fold difference in malaria prevalence. The explanation of this anomaly was found in the drainage quality of the different valley ecosystems. The poorly drained valley ecosystems had 3-fold greater numbers of vectors[8], and 8.5-fold greater malaria prevalence and 2-fold greater number of individual malaria antibody prevalence in the well drained valley ecosystems[9]. Thus environmental factors other than meteorological parameters could explain some of the variations in malaria prevalence.

The current article[7] provides significant progress towards developing early prediction models for dengue epidemics. However, disease predictive models involve a multi-disciplinary approach because climate and weather alone cannot explain the variation in disease trends. A non-statistical method for the early prediction of malaria epidemics has been developed[10]. The changes in vectorial capacity was tested by simulating changes in entomological parameters such as daily survival rates, blood feeding interval and the extrinsic incubation period under different temperature scenarios. These simulations indicated that malaria transmission was indeed sensitive to increasing temperature. A process based model was then developed which involved the identification of critical transmission thresholds in temperature and rainfall. The lag between the monthly temperature and rainfall threshold was determined and programmed in Microsoft Excel® and a composite risk variable computed. The degree of departure from the thresholds is an indication of increasing risk[10]. Different ecosystems model variants have since been developed and their positive predictive power, sensitivity and specificity tested[11]. The models have performed very well in all aspects and are now available in an automated format for end users. In the majority of ecosystems anomalies in the maximum and minimum temperatures contributed to the mean temperature anomaly. However, in one site only the mean maximum temperature had a contribution to the anomalies. The quality of drainage in different ecosystems determined the mean monthly rainfall threshold of the ecosystem specific model[11]. It also determines the sensitivity of the ecosystem to epidemic outbreaks as a result of transmission stability and immunity.

The dengue model may be further improved by taking the process based approach. Before the model becomes acceptable to end users and policy makers, there are various processes that have to be undertaken including a participatory involvement of stakeholders and policy makers in its validation. There is also a need to demystify the statistics and mathematics of the models which quite often discourage health end users.

Adaptation to climate change will require the development of such tools to reduce the uncertainty involved in climate associated risks. The intensity and frequency of extreme climate events are expected to increase and this will be associated with significant health impacts. Early epidemic prediction models if used correctly, will allow early interventions that will minimize the risk of infections. In addition, there could be an economic benefit of intervening only when action is warranted.

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References

1. Mukhopadhyay S, Kuhn RJ, Rossmann MG. A structural perspective of the flavivirus life cycle. *Nat Rev Microbiol* 2005; *3* : 13-22.
2. Bhutta ZA, Ahmed T, Black RE, Cousens S, Dewey K, Giugliani E, *et al*. What works? Interventions for maternal and child undernutrition and survival. *Lancet* 2008; *37* : 417-40.
3. Ahmed TU, Rahman GM, Bashar K, Shamsuzzaman M, Samajpati S, Sultana S, *et al*. Seasonal prevalence of dengue vector mosquitoes in Dhaka City, Bangladesh. *Bangladesh J Zool* 2007; *35* : 205-12.
4. Delatte H, Gimonneau G, Triboire A, Fontenille D. Influence of temperature on immature development, survival, longevity, fecundity, and gonotrophic cycles of *Aedes albopictus*, vector of chikungunya and dengue in the Indian Ocean. *J Med Entomol* 2009; *46* : 33-41.
5. Watts DM, Burke DS, Harrison BA, Whitmire RE, Nisalak A. Effect of temperature on the vector efficiency of *Aedes aegypti* for dengue 2 virus. *Am J Trop Med Hyg* 1987; *36* : 143-52.

6. Lambrechts L, Paaijmans KP, Fansiri T, Carrington LB, Kramer LD, Thomas MB, *et al*. Impact of daily temperature fluctuations on dengue virus transmission by *Aedes aegypti*. *Proc Nat Acad of Sci* 2011; *108* : 7460.

7. Karim N, Munshi SU, Anwar N, Alam MS. Climatic factors influencing dengue cases in Dhaka city: a model for dengue prediction. *Indian J Med Res* 2012; *136* : 32-9.

8. Ototo EN, Githeko AK, Wanjala CL, Scott TW. Surveillance of vector populations and malaria transmission during the 2009/10 El Nino event in the western Kenya highlands: opportunities for early detection of malaria hyper-transmission. *Parasit Vectors* 2011; *4* : 144.

9. Wanjala CL, Waitumbi J, Zhou G, Githeko AK. Identification of malaria transmission and epidemic hotspots in the western Kenya highlands: its application to malaria epidemic prediction. *Parasit Vectors* 2011; *4* : 81.

10. Githeko AK, Ndegwa W. Predicting malaria epidemics in the Kenyan highlands using climate data: a tool for decision makers. *Global Change Human Health* 2001; *2* : 54-63.

11. Githeko AK, Ototo EN, Guiyun Y. Progress towards understanding the ecology and epidemiology of malaria in the western Kenya highlands: Opportunities and challenges for control under climate change risk. *Acta Trop* 2011; *121* : 19-25.