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Review

Modeling the impact of global warming on vector-borne infections

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Abstract

Global warming will certainly affect the abundance and distribution of disease vectors. The effect of global warming, however, depends on the complex interaction between the human host population and the causative infectious agent. In this work we review some mathematical models that were proposed to study the impact of the increase in ambient temperature on the spread and gravity of some insect-transmitted diseases.

Keywords: Global warming; Vectors; Infections; Modeling

1. Introduction

Disagreement still remains about the extent to which recent warming in global temperatures deviates from normal climatic cycles [1]. In other words, the role of anthropogenic sources in the unequivocal warming of the Earth observed in the last decades is to be determined yet. However, few deny the fact that the global temperature has increased since around 1900 [2]. During this century, Earth's average surface temperature rises are likely to exceed the safe threshold of 2 °C above preindustrial average temperature [3]. Also undeniable is the fact that human activities are causing a net annual addition of 3 Gt of carbon to the atmosphere [4]. Among climatologists, in particular, there is now a general agreement that the main influence on the world's climate in the near future will be the warming effect of anthropogenic greenhouse gases [2].

Climate change due to greenhouse warming is not just an environmental issue but also a health issue [3] and it would have both direct and indirect effects upon human health [5,6]. The direct effects, via temperature change, thermal extremes and increased natural disasters, are easier to predict than are the various indirect and delayed effects [4]. Approximately 22,000 to 45,000 heat-related deaths occurred across Europe over two days in August 2003 [7,8], probably the hottest summer in Europe in over 500 years, with average temperatures 3.5 °C above normal [9]. Direct effects of climate change on human health ranges from cardiovascular mortality and respiratory illness due to heat-

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waves, to malnutrition from crop failures [10]. However, it is likely that the indirect effects, in particular the alteration in patterns of vector-borne infections, will outweigh the direct effects.

The increase in temperature will affect the spread and transmission rates of vector-borne infections. Temperature affects rate of pathogen maturation and replication within mosquitoes, the density of insects in a particular area, and increases the likelihood of infection [3]. Vector reproduction, parasite development cycle, and bite frequency generally rise with temperature; therefore malaria, tick-borne encephalitis, and dengue fever will become increasingly widespread.

Of particular concern is the expected shift in the 16 °C isotherm, which indicates the limit for reproduction and maintenance of anopheline mosquitoes, vectors of malaria [11]. The threat posed by malaria is especially worrisome. The disease is increasingly resistant to antimalarial drugs and is spreading out of control over large areas of South America and Asia. In addition to the new environmental conditions for the thriving of malaria vectors, the optimum temperature for the deadly *Plasmodium falciparum* parasite is 26 °C, which may soon be common in southern Europe and the United States. This may cause tremendous devastation because the emergence of *falciparum* malaria in an area where people have no natural immunity may lead to death rates as high as 50% [12].

The distribution of insects transmitting malaria, as well as other tropical infections, and their seasonal abundance are determined by the favorable temperature conditions and the presence of breeding places, which depend to a great extent on precipitation. With the increase in the average global temperature it is necessary to develop techniques to foresee and prevent outbreaks [11].

One of the first attempts at entomological forecasting was that of Gill [13], who in 1921 defined the areas where malarial epidemics are possible. On the basis of the temperature and humidity factors limiting the distribution of mosquitoes and necessary for the development of malaria parasite, Gill determined the areas where malarial transmission would be unlikely. Today, modern techniques for temperature and humidity measurement, such as remote sensing of meteorological data for the study of the distribution and abundance of vectors of disease [14], are widening the possibilities of entomological forecasting. The precise calculations, however, should be performed upon entomological knowledge that would correlate the factors determining the abundance and distribution of vectors and their life cycle [11].

Global warming will certainly affect the abundance and distribution of disease vectors [15]. The effect of global warming, however, depends on the complex interaction between the human host population and the causative infectious agent.

Some models suggest that vector-borne diseases will become more common as the Earth warms, although caution is needed in interpreting these predictions. Clearly, global warming will cause changes in the epidemiology of infectious diseases.

It should be mentioned that an increase in temperature might displace the current geographic distribution of those diseases and although transmission might get established in new areas, it might also disappear from current areas of transmission making predictions about the net balance in transmission rather uncertain [11].

For a comprehensive review on the impacts of global change on vector-borne diseases see [16].

2. Theoretical basis of vector-borne infections [17]

The central parameter related to the intensity of transmission of infections is the so-called basic reproduction number R_0 [17], defined by Macdonald [18] as the number of secondary infections produced by a single infective in an entirely susceptible population. Originally applied in the context of malaria, R_0 is a function of the vector population density as related to the host population, m, the average daily biting rate of the vector, a, the host susceptibility, b, the mosquito's susceptibility, c, the vector mortality rate, μ, the parasite extrinsic incubation period in days, n, and the parasitemia recovery rate, r, according to the (now) historical equation:

$$R_0 = \frac{ma^2bc\exp[-\mu n]}{r\mu} \tag{1}$$

(actually, Macdonald denoted R_0 as z_0 in his original paper). From the definition of the basic reproduction number it can be demonstrated that if R_0 is not greater than one, that is, when an index case (the first infective individual) is not able to generate at least one new infection, the disease dies out. Hence, in the original Macdonald analysis, R_0 coincides with the threshold for the infection persistence. For an interesting historical account of R_0, see [19].