

Box 18-1 (continued)

to climate variation, so climate change may have a marked effect on its incidence and stability. Simulations with this model, using several different GCMs, predict a climate-induced increase in the incidence of annual malaria cases of approximately 50–80 million in response to a temperature rise of around 3°C by the year 2100, relative to an assumed approximate base of 500 million annual cases in a 2100 world without climate change (Martens *et al.*, 1995b).

Another recent attempt at aggregated global modeling (Martin and Lefebvre, 1995) has predicted that potential malaria transmission would spread to higher latitudes, while some currently stable endemic areas near the equator would become unstable, leading to reductions in population immunity levels.

Such models, despite their highly aggregated predictions and simplifying assumptions, provide indicative information about the likely impact of climate change on the potential transmission of vector-borne diseases (assuming that other relevant factors remain constant). There is a clear need for validation of these models and for incorporating more extensive detail into them. Meanwhile, this line of research has begun to elucidate the interdependent relationships among climate change, vector population dynamics, and human disease dynamics.

Recent evidence of the responsiveness of malaria incidence to local climate change comes from observations of marked increases in malaria incidence in Rwanda in 1987, when atypically hot and wet weather occurred (Loevinsohn, 1994), and annual fluctuations in falciparum malaria intensity in northeast Pakistan that correlated with annual temperature variations during the 1980s (Bouma *et al.*, 1994). Hence, it is a reasonable prediction that, in eastern Africa, a relatively small increase in winter temperature could extend the mosquito habitat and thus enable falciparum malaria to reach beyond the usual altitude limit of around 2,500 m to the large, malaria-free, urban highland populations, e.g., Nairobi in Kenya and Harare in Zimbabwe. Indeed, the monitoring of such populations around the world, currently just beyond the boundaries of stable endemic malaria, could provide early evidence of climate-related shifts in malaria distribution (Haines *et al.*, 1993).

18.3.1.2. African Trypanosomiasis

African trypanosomiasis, or “sleeping sickness,” is transmitted by tsetse flies. The disease is a serious health problem in tropical Africa, being generally fatal if untreated. Research in Kenya and Tanzania shows only a very small difference in mean temperature between areas where the vector, *Glossina morsitans*, does and does not occur. This indicates that a small change in temperature may significantly affect the limits of the vector’s distribution (Rogers and Packer, 1993).

18.3.1.3. American Trypanosomiasis (Chagas’ Disease)

American trypanosomiasis is transmitted by insects of the subfamily *Triatominae*. It is a major problem in Latin America, with 100 million people at risk and 18 million infected (WHO, 1995c). An estimated 15–20% of infected people develop clinical Chagas’ disease. Most of the triatomine vector species need a minimum temperature of 20°C for feeding and reproduction (Curto de Casas and Carcavallo, 1984), but at higher

temperatures (28–30°C) they feed more frequently, have a shortened life cycle, and an increased population density (Carcavallo and Martinez, 1972, 1985). At even higher temperatures, the most important vector species, *Triatoma infestans*, doubles its reproductive rate (Hack, 1955).

18.3.1.4. Schistosomiasis

Schistosomiasis is a water-based disease caused by five species of schistosomal flukes. Water snails act as the intermediate host (and, strictly speaking, are not active “vectors”). The infection has increased in worldwide prevalence since mid-century, perhaps largely because of the expansion of irrigation systems in hot climates, where viable snail host populations interact with infected humans (White *et al.*, 1972; Grosse, 1993; Hunter *et al.*, 1993).

Data from both the field and the laboratory indicate that temperature influences snail reproduction and growth, schistosome mortality, infectivity and development in the snail, and human-water contact (Martens *et al.*, 1995b). In Egypt, for example, water snails tend to lose their schistosome infections during winter, but if temperatures increase, snails may mediate schistosomiasis transmission throughout the year (Gillet, 1974; WHO, 1990). Predictive modeling indicates that a change in background temperatures may cause the infection to extend to currently unaffected regions. Fluctuations in temperature may also play an important role in optimizing conditions for the several life-cycle stages of schistosomiasis (Hairston, 1973).

18.3.1.5. Onchocerciasis (River Blindness)

Onchocerciasis, or “river blindness,” is a VBD affecting approximately 17.5 million people—some in Latin America, most in West Africa. The vector is a small blackfly of the genus *Simulium*, and the infectious agent is the larva of the *Onchocerca volvulus* parasite. This threadlike worm damages the skin, the