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Global Climate Changes and International Trade and Travel: Effects on Human Health Outcomes

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Abbreviations

CFR	case fatality rate(s)
CNS	central nervous system
CSF	cerebrospinal fluid
DEN	dengue viruses (serotypes 1-4)
EM	eosinophilic meningitis
GNS	gnathostomiasis
ID(s)	infectious disease(s)
NAS	neuroangiostrongyliasis
SARS	severe acute respiratory syndrome

Introduction

The ultimate effects of global warming on rainfall, drought, and tropical cyclone activity will have far-reaching human health impacts, not only on weather-related infectious disease outbreaks but also on world food production and supplies, access to safe drinking water, and mass population relocations. A more frequent drought–monsoon cycle supports outbreaks of malaria transmitted by water surface ovipositing *Anopheles* species mosquitoes and dengue and Chikungunya fever outbreaks transmitted by container-breeding *Aedes* species mosquitoes. As nighttime temperatures increase and glaciers and permafrost retreat into the highlands, the geographic distribution ranges of mosquito and tick-borne diseases extend to higher altitudes and to new regions in formerly disease-free areas with competent insect vectors. This article will explore the influences of free trade and international travel on public health outcomes in a warming world with a global economy.

The Interrelationships of Climate Change, International Commerce, Free Trade, and International Travel on Observed Human Health Consequences

Many factors will influence the onset of emerging and reemerging infectious disease outbreaks including climatic and ecosystem changes, new vector–pathogen relationships, human health behaviors, and human host susceptibilities. Emerging infectious diseases may be defined as those that have recently appeared in human populations, have expanded their ranges of distribution, or threaten to increase their prevalences and distribution ranges in the near future. Today most emerging infectious diseases arise in the natural environment as zoonoses, such as Hantavirus pulmonary syndrome and severe acute respiratory syndrome (SARS); or adopt a competent, new insect vector, such as *Aedes albopictus*, a new mosquito vector for dengue and Chikungunya viruses (**Figure 1**). Reemerging infectious diseases may be defined as those whose pathogenesis, clinical manifestations, and treatment strategies are well known but have reemerged as public health threats, often with increased antimicrobial drug resistances, such as multidrug resistant tuberculosis and methicillin-resistant *Staphylococcus aureus*.

In addition to climatic, ecologic, and microbial factors, other significant factors that influence the emergence and reemergence of infectious diseases include international trade and air travel, the globalization of agriculture and food production, exotic eating habits, lifestyle, and residential choices. The worldwide spread of the Asian tiger mosquito, *A. albopictus*, by imported tire shipments on container ships from Southeast Asia has introduced a new secondary (to *Aedes aegypti*) vector for dengue fever into the tropical Americas and Chikungunya fever in India and Indian Ocean Islands (**Figure 1**). Accessible airline connections now permit infected individuals to travel anywhere in the world in less than 24 h, delivering human reservoirs of malaria, dengue, West Nile virus, and Chikungunya fever to new temperate areas for autochthonous or local transmission by new and adaptable mosquito vectors, often recent air or sea arrivals themselves.

West Nile virus was most likely imported to the United States in 1999 through air by the arrival in New York City of either an infected passenger or an infected *Culex* species mosquito from an endemic region of East Africa or the Middle East. By 2002, competent local *Culex* vectors had initially established a mobile reservoir for West Nile virus in wild birds in wet, warming ecosystems that began to move the virus rapidly across the United States from New York to the west coast. The initial wild animal reservoir for introduced West Nile virus in the United States was so specific that it targeted only birds of the family Corvidae, especially crows and jays. By 2005, West Nile virus infections were reported in other wild and domestic animals and humans across the continental United States and had caused over 4000 cases of meningoencephalitis with 263 deaths (case fatality rate

Figure 1 The female *Aedes albopictus*, or Asian tiger mosquito, has been disseminated in coastal temperate zones worldwide by global trade and has genetically adapted to become a competent new vector for dengue and Chikungunya viruses. Reproduced from CDC Image, available at http://www.dpd.cdc.gov/dpdx.

(CFR) = 6.6%). The major mosquito vectors of emerging and reemerging infectious diseases are featured in Table 1.

There were many historical examples of the international transport of human pathogens or their vectors by world exploration and trade that preceded any significant impact from recent climatic changes by centuries. Prominent examples included the introduction of syphilis in the Americas by European explorers, the spread of bubonic plague to Europe by Genoans returning from wars in the Middle East, and the introduction of yellow fever carriers and mosquito vectors in the Americas by West African slave traders. Some recent examples of the international transport of pathogens or their vectors include the expanded global distribution ranges of *Plasmodium falciparum* and *P. vivax* malaria and neuroangiostrongyliasis (NAS) caused by the rat lungworm, *Angiostrongylus cantonensis*.

International Air Travel and Airport Malaria

The most common reasons for malaria to occur in the industrialized nations of North America and Europe where malaria was once endemic are also related to international air travel and include airport malaria and imported malaria. Although similar subtropical ecosystems will support malaria reintroduced into formerly endemic regions such as the Southern United States and the Northern Mediterranean, the exact impact of warming temperatures and greater precipitation on expanded malaria distribution ranges worldwide remains uncertain. Nevertheless, malaria has become endemic at higher alpine altitudes in East Africa and in South America.

Airport malaria is defined as the intercontinental transfer of malaria through the introduction of an infective anopheline mosquito vector into a nonendemic disease area with a changing ecosystem that supports the vector–pathogen relationship. However, imported malaria is defined as the intercontinental transfer of malaria by the movement of a parasitemic person to a nonendemic disease area with locally competent anopheline vectors in a welcoming ecosystem.

Airport malaria is acquired through the bite of an infected tropical anopheline mosquito within the vicinity of an international airport. The malaria-infected mosquito vector is a new arrival on an international flight from a malaria-endemic region. Climate change has now expanded the geographic distribution of malaria-endemic regions and extended the length of seasonal malaria transmission cycles in endemic regions, so more arrivals of malaria-carrying mosquitoes are anticipated.

How often do infected mosquitoes travel by air from tropical disease-endemic nations to capital cities in industrialized nations with disease-supporting warming ecosystems? In 1983, random searches of arriving airplanes at Gatwick Airport in London found that 12 of 67 airplanes from tropical countries contained mosquitoes, some of which were female *Anopheles* species capable of transmitting malaria. After the female *Anopheles* species mosquito leaves the aircraft, she may survive long enough, especially during temperate periods, to take a blood meal and transmit malaria pathogens, usually in the vicinity of an international airport. After one or more

Table 1 Mosquito vectors of medical importance

Mosquito subfamily	*Mosquito genera*	*Infectious disease transmission*	*Infectious disease (ID) distribution*	*Causative agents of IDs*	*Classification of causative agents*
Anophelinae (anophelines)	*Anopheles* spp.	Malaria	Africa, Asia, Central America, South America	*Plasmodium falciparum* *P. vivax* *P. ovale* *P. malariae*	Protozoan parasites
Anophelinae	*Anopheles* spp.	Bancroftian filariasis	Southeast Asia	*Wuchereria bancrofti*	Filarial worms causing lymphatic filariasis
		Brugian filariasis	Southeast Asia	*Brugia malayi*	
		Timor filariasis	Timor, Indonesia	*Brugia timori*	
Anophelinae	*Anopheles* spp.	O'nyong nyong fever	Africa	Alphavirus	Togaviridae
Culicinae (culicines)	*Aedes* spp.	Yellow fever	Africa, Latin America	Flavivirus	Flaviviridae
		Dengue fever	Africa, Asia, Latin America	Flaviviruses DEN 1-4	Flaviviridae
		Chikungunya fever	Africa, Asia	Alphavirus	Togaviridae
		Eastern equine encephalitis	Eastern, Southeastern United States	Alphavirus	Togaviridae
		Ross River fever	Australia, Papua New Guinea	Alphavirus	Togaviridae
		California encephalitis	Western United States	Bunyavirus	Bunyaviridae
		LaCrosse encephalitis	Midwestern United States	Bunyavirus	Bunyaviridae
		Rift Valley fever	Africa	Phlebovirus	Bunyaviridae
Culicinae	*Culex* spp.	Western equine encephalitis	Western United States, South America	Alphavirus	Togaviridae
		Venezuelan equine encephalitis	Central and South America	Alphavirus	Togaviridae
		Japanese encephalitis	Far East,	Flavivirus	Flaviviridae
			Southeast Asia, Indonesia	Flavivirus	Flaviviridae
		St. Louis encephalitis	United States	Flavivirus	Flaviviridae
		West Nile virus	Africa, Middle East, East Europe, North America	Flavivirus	Flaviviridae
Culicinae	*Mansonia* spp.	Brugian filariasis	Southeast Asia	*Brugia malayi*	Filarial worm causing lymphatic filariasis
Culicinae	*Psorophora* spp.	Venezuelan equine encephalitis	Central and South America	Alphavirus	Togaviridae
Culicinae	*Haemophagus* spp.	Yellow fever	Africa, South America	Flavivirus	Flaviviridae

blood meals, female mosquitoes seek a water surface to lay their eggs.

All female mosquitoes lay their eggs in standing water, either on the surface or just below. The anopheline vectors of malaria prefer to lay eggs in drainage ditches, marshy areas, and puddles. The culicine vectors of West Nile virus, dengue, and Chikungunya fever prefer to lay their eggs in containers that trap freshwater, such as flower pots and even discarded tires. Climate changes, particularly warming nighttime temperatures and increased precipitation, offer several selective advantages to all mosquito species including (1) a longer reproductive life and a prolonged breeding season, (2) opportunities for more blood meals during gestation, (3) plenty of standing water surfaces for egg laying, and (4) a faster egg hatch over days and not weeks.

As international air travel between malaria-endemic nations and malaria nonendemic nations increases, cases

of airport malaria have increased. In 1983, two cases of *P. falciparum* malaria were diagnosed in persons without histories of travel to malaria-endemic regions living 10 and 15 km from Gatwick Airport. Hot, humid weather in Britain may have facilitated the survival of imported, infected anopheline mosquitoes. During the summer of 1994, six cases of airport malaria were diagnosed in the vicinity of Charles de Gaulle Airport near Paris. Four of the patients were airport workers, infected at work, and the others were residents of Villeparisis, a small town approximately 7.5 km away from the airport. To reach Villeparisis, the infected anopheline mosquitoes were thought to have hitched a car ride with airport workers who lived next door to two of the patients.

In addition to airport malaria transmitted by infected mosquito air travelers, many countries throughout the developed world are reporting an increasing number of cases of imported malaria because of the great increase in long-distance air travel by infected passengers. Malaria cases imported from Africa to the United Kingdom rose from 803 in 1987 to 1165 in 1993.

In 1998, an Italian woman was infected with malaria following a bite from a local, malaria-competent vector, *Anopheles labranchiae*. This species had been a common malaria vector in Italy until the country was declared malaria free in 1970. The local mosquito responsible for transmitting malaria was thought to have acquired the parasite after biting a parasitemic girl who had recently arrived in Italy from India. Airport malaria was ruled out in this case because of the great distance from the nearest international airport. This case illustrated the ease with which imported malaria may be reestablished in a formerly endemic nation with a warming climate, competent local anopheline vectors, and a humid and wet ecosystem that supports vector–host–parasite relationships.

In the United States, recent outbreaks of presumed local or autochthonous mosquito-borne malaria transmission have been reported in California, following the immigration of agricultural workers from malaria-endemic areas of Mexico. In 1986, a *P. vivax* malaria outbreak resulted in 28 cases, 26 in Mexican migrant workers, over a 3-month period. Epidemiological and microbiological investigations later confirmed secondary spread from infected immigrants to other immigrants and local residents transmitted by local malaria-competent, anopheline vectors.

Prevention and control strategies for airport and imported malaria should include early case definition, case confirmation, and treatment; strengthened vector surveillance to detect the potential for autochthonous transmission; and drainage of potential mosquito breeding and egg-laying surface water sites. Although the relationships among infected vector importation, index case immigration, reclaimed disease ecosystems, and malaria transmission are complex, future attempts to control and eradicate airport and imported malaria should be based on an understanding of disease transmission mechanisms and an appreciation that climate and ecosystem changes can support reemerging local mosquito-borne infectious diseases, especially malaria, dengue, Chikungunya fever, and West Nile virus (**Table 1**).

International Sea Trade and Imported Cholera in South America

In addition to commercial, business, and recreational international air travel, infectious diseases may also be transmitted by sea from endemic to nonendemic nations with warming ecosystems that will support host–pathogen relationships. Infected arthropod vectors, infected animal hosts, especially rodents, and even virulent microbes travel well at sea, especially in hot and humid ship cargo holds and in cargo containers. Recent outbreaks of cholera in Ecuador and Peru have followed increased shipping trade with Southeast Asia, where *Vibrio cholerae* is endemic in coastal estuaries. Traceback investigations have demonstrated that container vessels pump in contaminated saltwater ballast into their hulls in their homeports for smoother transoceanic sailing and then discharge the ballast before unloading in distant ports with warming estuaries. Such practices can effectively import cholera bacteria, marine viruses, and harmful algae to new, warming marine ecosystems and fisheries causing microbial and algal toxin contamination of shellfish beds and regional fisheries.

The Exotic Pet Trade, Exotic Cuisines, Adventure Travel, and Emerging Infectious Diseases in the Developed World

The helminthic infections that can cause eosinophilic meningitis (EM), neuroangiostrongyliasis (NAS) and gnathostomiasis (GNS), and Chagas disease, an arthropod-borne zoonosis, share many of the characteristics of emerging infectious diseases supported by free trade in a warming world. NAS is now endemic in Hawaii and some coastal US cities following the US importation of NAS by stowaway rodent hosts on cargo ships from China and Southeast Asia of the causative parasite, *A. cantonensis*, or rat lungworm. The intentional introduction of an intermediate *A. cantonensis* host, the giant African land snail (*Achatina fulica*), to control insect pests on US farmlands and to serve as exotic pets for home terrariums also imported the unwelcome parasites to US ecosystems by paratenic or transporting hosts.

Gnathostoma spinigerum-induced GNS has been recently recognized as an emerging imported disease in the United Kingdom. Since *G. spinigerum* is endemic in Central and South America, most notably in Mexico, GNS may soon become another emerging potential

cause of EM in North America, given the adventurous and exotic eating habits of North Americans abroad.

The reduviid insect vectors of Chagas disease have now moved from the rural areas of Latin America into cities and coastal resort areas frequented by international travelers. The vectors have also migrated northward into the temperate areas of the midsouthern United States and have established a *Trypanosoma cruzi* zoonosis among wild animals and some domestic animals kenneled outdoors as far north as Virginia.

Some Specific Examples of the Influences of Climate Change and International Commerce and Travel on Emerging and Reemerging Infectious Diseases

The Exotic Pet Trade and the Importation of Infectious Diseases: Monkeypox and Neuroangiostrongyliasis

In addition to *A. cantonensis*-infected African giant snails, the exotic pet trade has also imported monkeypox to the United States in pet rodents. In 2003, 53 cases of monkeypox were reported among three midwestern US states (Illinois, Indiana, and Wisconsin). Fortunately, there were no deaths, but 14 patients were hospitalized for supportive treatment, including a child with encephalitis. The monkeypox virus, a smallpox-like orthomyxovirus, was first isolated in the Congo River basin of West Africa in 1968. After an incubation period of 1–2 weeks, monkeypox is characterized by a prodrome of headache, fever, fatigue, and backache, followed by a characteristic rash. The monkeypox rash is similar to smallpox with evolving macules, vesicles, and pustules that crust over and heal within 14–21 days (**Figure 2**). Unlike smallpox, pronounced lymphadenopathy is usually present and complications may include pneumonia and encephalitis. Although person-to-person transmission may occur, infection is usually transmitted by contact with contaminated animals. The CFR ranges from 1% to 10%, with higher CFRs in young children.

Traceback investigations of the US monkeypox outbreak revealed that all patients had had contact with pet animals: 51 patients had contacts with pet prairie dogs, 1 with a pet rabbit, and 1 with a pet imported giant Gambian rat, *Cricetomys gambianus*. The source of the monkeypox virus introduced into the United States was later identified as infected giant Gambian rats imported from Ghana to Texas and sold to an Illinois pet distributor, who housed the animals together before sale to pet shop owners and others. Although the monkeypox virus is endemic in West African river basins, including the Gambia and Congo, the humid river bottomland ecosystems of the Mississippi–Missouri–Ohio River basins of the central United States will support the transmission of the virus from imported rodents to domestic rodents, especially prairie dogs and squirrels, and to rabbits (**Figure 3**). The close contact between pet owners and their pets permitted the transmission of the zoonotic infection, for which there is no specific treatment, to humans. Regulations now prohibit the importation of African rodents into the United States, and sentinel monitoring systems of local rodents for monkeypox infections have now been initiated. Like the rat lungworm, the monkeypox virus may become an unwelcomed, but established, zoonosis in the United States

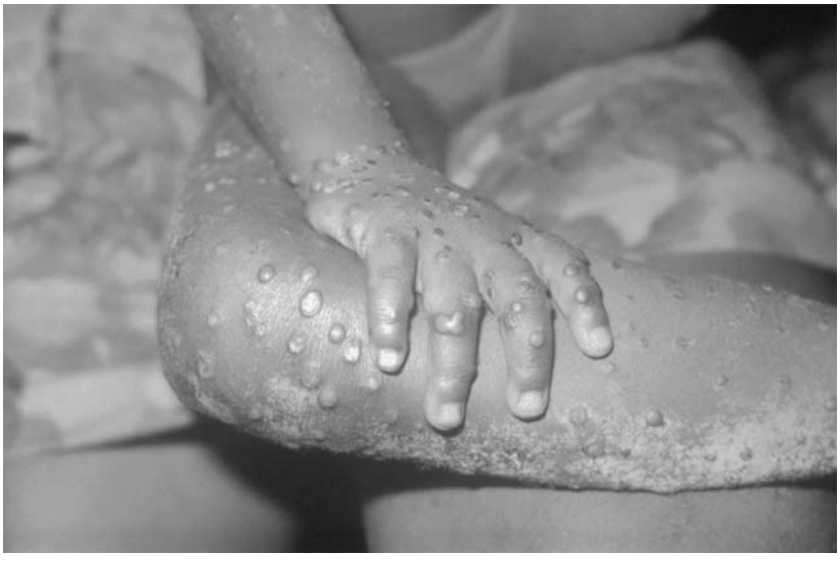

Figure 2 Close-up of monkeypox lesions on the arm and leg of a 4-year-old female. Reproduced from CDC Public Health Image Library (PHIL), Image Number 2329, available at http://www.cdc.gov.

Figure 3 An American prairie dog, genus *Cynomys*. In June 2003, monkeypox was reported among several people in the American midwest. Most of these people became ill after having contact with pet prairie dogs that had contracted the virus from imported Gambian giant rats in pet stores. This was the first outbreak of monkeypox, a viral disease endemic in West Africa, in the United States. Reproduced from CDC Public Health Image Library (PHIL), Image Number 4100, available at http://www.cdc.gov.

as a result of relaxed free trade regulations, especially of the exotic pet trade, and a welcoming, warming river-bottomland ecosystem.

International Commerce and the Exotic Pet Trade: *A. cantonensis* and Neuroangiostrongyliasis

A. cantonensis, the rat lungworm, was first described in China in 1935, living in the pulmonary arteries of rats. The first human infection was reported from Taiwan in 1945. The life cycle is complex and involves a rodent definitive host and an appropriate mollusk intermediate host, usually land snails or slugs (**Figure 4**). Adult worms mature in rat brains, enter the central circulation, and mate in the pulmonary arteries producing eggs. The eggs become first-stage larvae that penetrate pulmonary vessels to access the respiratory tree, where they are coughed up, swallowed, and excreted in feces. These second-stage larvae must be consumed by land snails or slugs to mature into infective third-stage larvae, be eaten by rodents, and maintain the parasite's life cycle. Man becomes a dead-end host by consuming raw intermediate mollusk hosts, or food items contaminated by their slime, or by consuming raw, crustacean (shrimp, crabs, fish, and frogs) transport, or paratenic, hosts that consumed infected mollusks. In man and paratenic hosts, the neurotropic larvae migrate to the CNS (neural larva migrans) seeking to mature into young adults as in rat brains but eventually die causing EM.

A. cantonensis is enzootic throughout Southeast Asia, most Indian and Pacific Ocean islands, including the Hawaiian Islands, many Caribbean islands, and has even been reported in New Orleans, Louisiana. The global spread of the parasite resulted from international trade, parasite-infested rat stowaways disembarking container ships, and the intentional introduction of giant African land snails as biological controls and exotic pets. In 1988, *A. cantonensis* was isolated from 21% of Norway rats, *Rattus norvegicus*, trapped in New Orleans, Louisiana, between April 1986 and February 1987. In 1995, a case of *A. cantonensis*-induced EM was reported from New Orleans in an 11-year-old male who presented with a 1-week history of headache, stiff neck, low-grade fever, and myalgias. He admitted eating a raw snail from the street on a dare a few weeks earlier. A second presumed autochthonous case of *A. cantonensis* infection was reported from South Louisiana in 2006 in a 22-year-old man who presented with neck and backaches, myalgias, and paresthesias. He admitted having consumed, on a dare, two raw legs from a green tree frog, *Hylidae cinerea*, 9 days before symptom onset.

A. cantonensis was responsible for an outbreak of NAS in 12 US travelers returning from Jamaica to Chicago in 2000, who had consumed romaine lettuce. The lettuce food vector was actually imported to Jamaica from the United States and presumably contaminated somewhere in between with snails or slugs or their secretions containing infective *A. cantonensis* larvae.

In summary, there is now ample clinical, epidemiological, parasitological, and immunological evidence

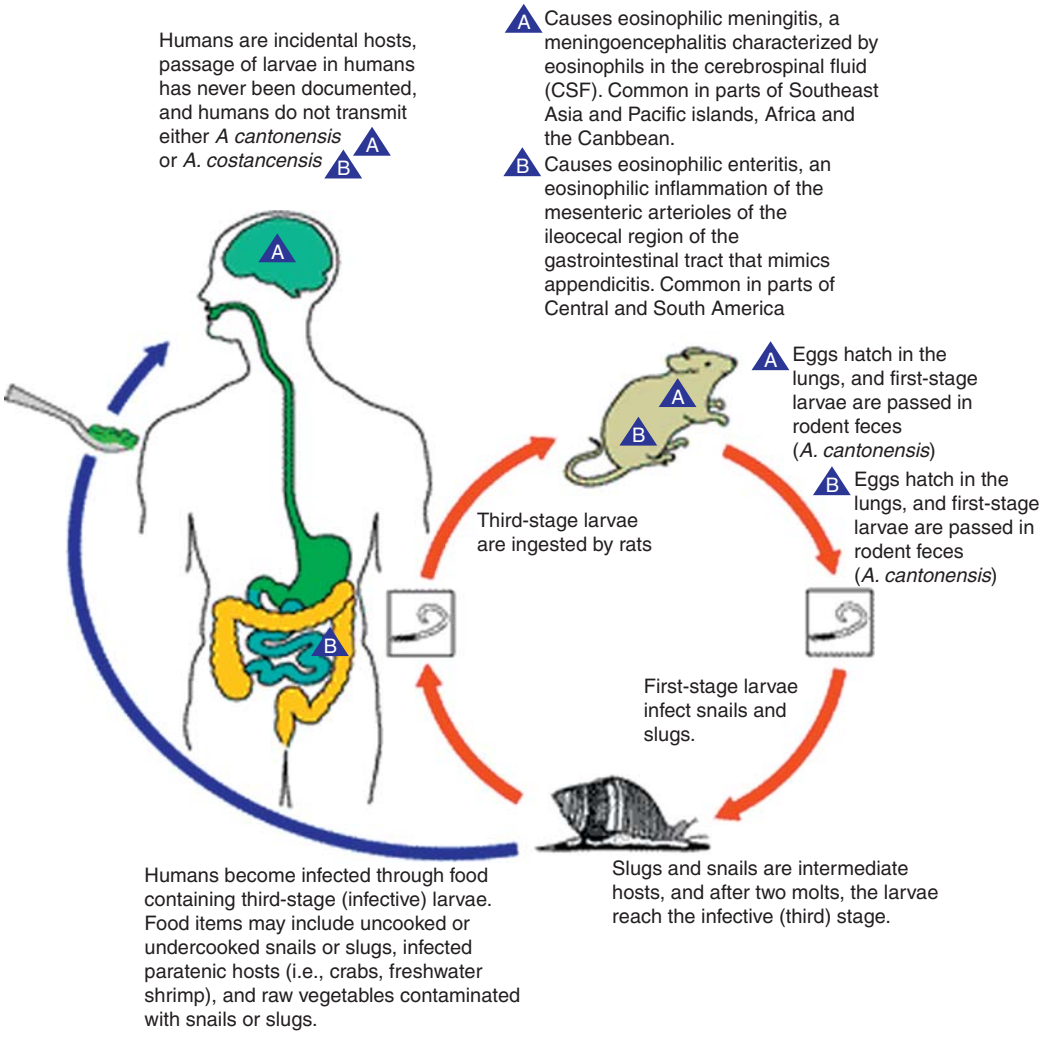

Figure 4 The life cycle of the rat lungworm, *Angiostrongylus cantonensis*, which causes eosinophilic meningitis (A), is compared to the life cycle of *Angiostrongylus costaricensis*, which causes eosinophilic enteritis (B). Reproduced from CDC Image, available at http://www.dpd.cdc.gov/dpdx.

that an *A. cantonensis* zoonosis has been established in the continental United States and in the Caribbean in rats, mollusks, and paratenic frog hosts as a direct result of international commerce. Although cases of NAS are rarely confirmed by the identification of *A. cantonensis* larvae or adults in the CNS, most cases can now be confirmed serologically and epidemiologically; the reported CFR in US cases is relatively low (5.0%); and most patients recover completely, even without specific antihelminthic treatment.

The most effective prevention and control strategies for NAS include (1) educating citizens and travelers in endemic areas that snails, slugs, freshwater fish and shrimp, frogs, and crabs must be cooked, not marinated or refrigerated, before being eaten; (2) washing all vegetables thoroughly before eating them uncooked; (3) washing hands thoroughly after handling pet African land snails or cleaning out their terrariums; (4) reducing and controlling the definitive host rodent populations with rodenticides; (5) reducing and controlling snail and slug paratenic host populations with molluscicides; and (6) policing and restricting the exotic pet trade.

Adventure Travel and Exotic Cuisine: *G. spinigerum* and Gnathostomiasis

Originally confined to Southeast Asia and Japan, GNS is acquired by eating raw or undercooked foods, infected with third-stage larvae of the roundworm, *G. spinigerum*. *G. spinigerum* is a common roundworm of wild and domestic cats, dogs, and other carnivores that coils within submucosal tumors in the stomach of definitive hosts, mates, and releases eggs in the host's feces. The eggs embryonate into first-stage larvae in fresh or brackish water ecosystems and are ingested by small crustacean intermediate hosts, which become prey for larger

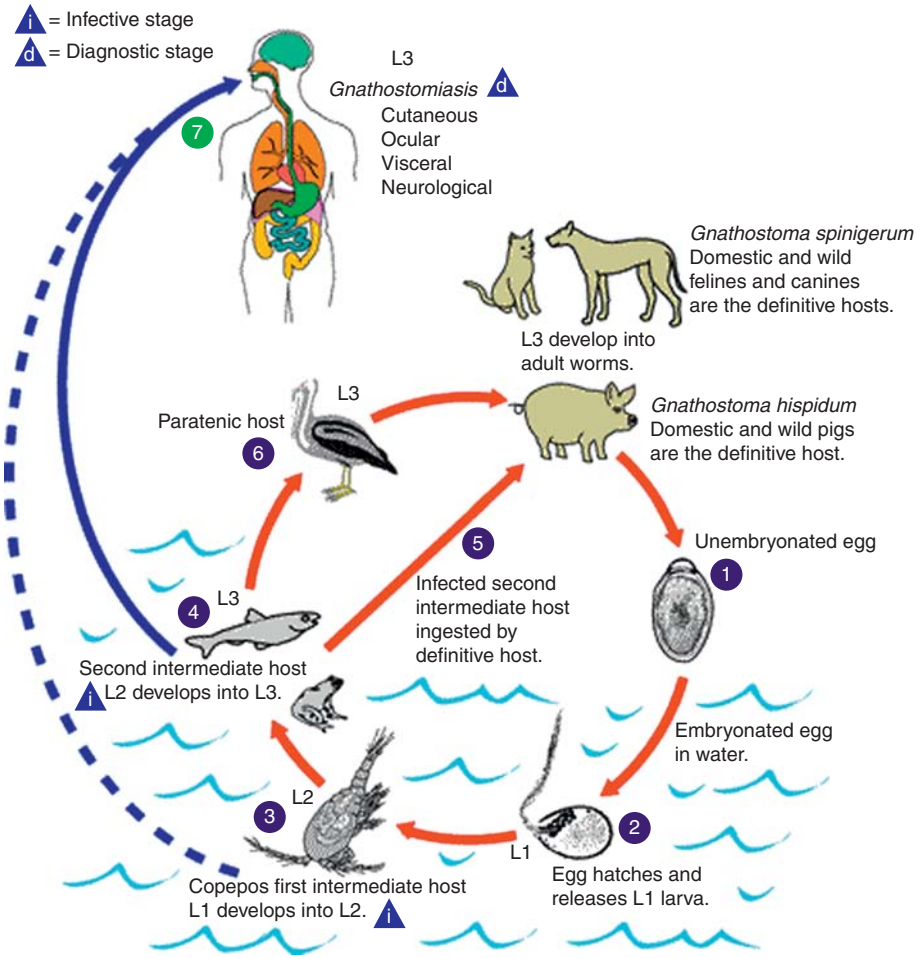

Figure 5 The life cycle of *Gnathostoma spinigerum*. Reproduced from CDC Image, available at http://www.dpd.cdc.gov/dpdx.

predators including fish, shrimp, crabs, crayfish, frogs, and snakes (Figure 5). The larvae mature into infective third-stage larvae in these transport or paratenic hosts, encyst in tissues, but do not develop into adults, unless the paratenic hosts are consumed by definitive carnivorous hosts. Once infective larvae are consumed by predators, they will mature into adults in the stomach and restart the parasite's life cycle (Figure 5). Since humans are not the natural definitive hosts, infective larvae consumed by humans in raw foods will not develop into adults but will penetrate the gastrointestinal tract and migrate hematogenously causing cutaneous or visceral larva migrans in any organ system.

Typically, the most common foods containing infective larvae have included fish, shrimp, crab, crayfish, frog, snake, and chicken. However, most human cases have followed consumption of raw or citrus-marinated fish (*ceviche*) or shellfish. In 2003, GNS caused by *G. spinigerum* was first recognized as an emerging imported helminthic infection in the United Kingdom in a case series of 16 patients treated over a 12-month period. In this series, the median incubation period was 12 months; peripheral eosinophilia was present in 7 (44%) of the 16 patients, and was not a reliable screening tool; and cases presented with a myriad of symptoms ranging from migratory cutaneous swellings (also known as Yangtze edema in Asia, or nodular eosinophilic migratory panniculitis in the United States) to eosinophilic gastritis.

Today, GNS remains relatively common in southern China, Thailand, and Bangladesh; is becoming more common throughout Latin America and the Caribbean; and is most often described in the United States in Southeast Asian immigrants. A diagnosis of GNS should now be considered for all patients with a history of travel to endemic regions and migratory cutaneous swellings, eosinophilic gastritis, or a combination of cutaneous swellings with any manifestation of neural larva migrans, especially eosinophilic meningoencephalitis and migratory radicular pain or radiculomyelitis. Neural GNS has also caused radiculomyeloencephalitis and subarachnoid hemorrhage. Most fatal cases of GNS have been associated with neural larva migrans and EM, with eosinophils comprising over 50% of the cerebrospinal fluid (CSF) cell count.

Prevention and control strategies for GNS include (1) educating citizens and travelers in endemic areas that fish, shrimp, crayfish, frogs, crabs, chicken, and snakes must be cooked thoroughly first and not eaten raw, marinated, or refrigerated and (2) seeking medical care immediately for evaluation of persistent nonspecific gastrointestinal illnesses or migratory subcutaneous swellings.

Regional Warming in Northern Latitudes and Adventure Travel in the Americas: *T. cruzi* and Chagas Disease (American Trypanosomiasis)

Chagas disease, or American trypanosomiasis, is an arthropod-borne protozoan infectious disease endemic throughout most of the Americas, caused by the trypanosome, *T. cruzi*, and transmitted to man by reduviid, or kissing, bugs (Figure 6). The life cycle of *T. cruzi* is depicted in Figure 6.

Reduviid bugs, specifically the triatomines (Phylum Insecta, Order Hemiptera, Family Reduviidae, Subfamily Triatominae), transmit several strains of wild animal *T. cruzi* among many nonspecies-specific wild mammalian reservoir hosts throughout the Americas (Figure 7). There are also many competent species of reduviid vectors capable of transmitting zoonotic wild strains of *T. cruzi* to domestic animals and man throughout the Americas (Figure 7). Like malaria and West Nile virus, Chagas disease may also be transmitted congenitally and by blood transfusion and organ transplantation. Unlike malaria and West Nile virus, however, Chagas disease was recently found to be transmitted by the ingestion of infected triatomines, which poses special risks to international travelers who eat or drink unpasteurized foods made from palm oils or raw sugarcane.

In its 2003 World Health Report, the World Health Organization (WHO) noted that Chagas disease caused more deaths from parasitic disorders than any other parasitic disease in Latin America, and that *T. cruzi* was responsible for the third greatest number of parasitic infections in the world following malaria and schistosomiasis. Chagas disease is now the most common cause of myocarditis worldwide, and chagasic heart failure has become an increasingly common indication for heart transplantation in the Americas.

Since 1955, human cases of autochthonous, imported, transfusion, and organ transplant-related Chagas disease have been increasing in the United States as a result of

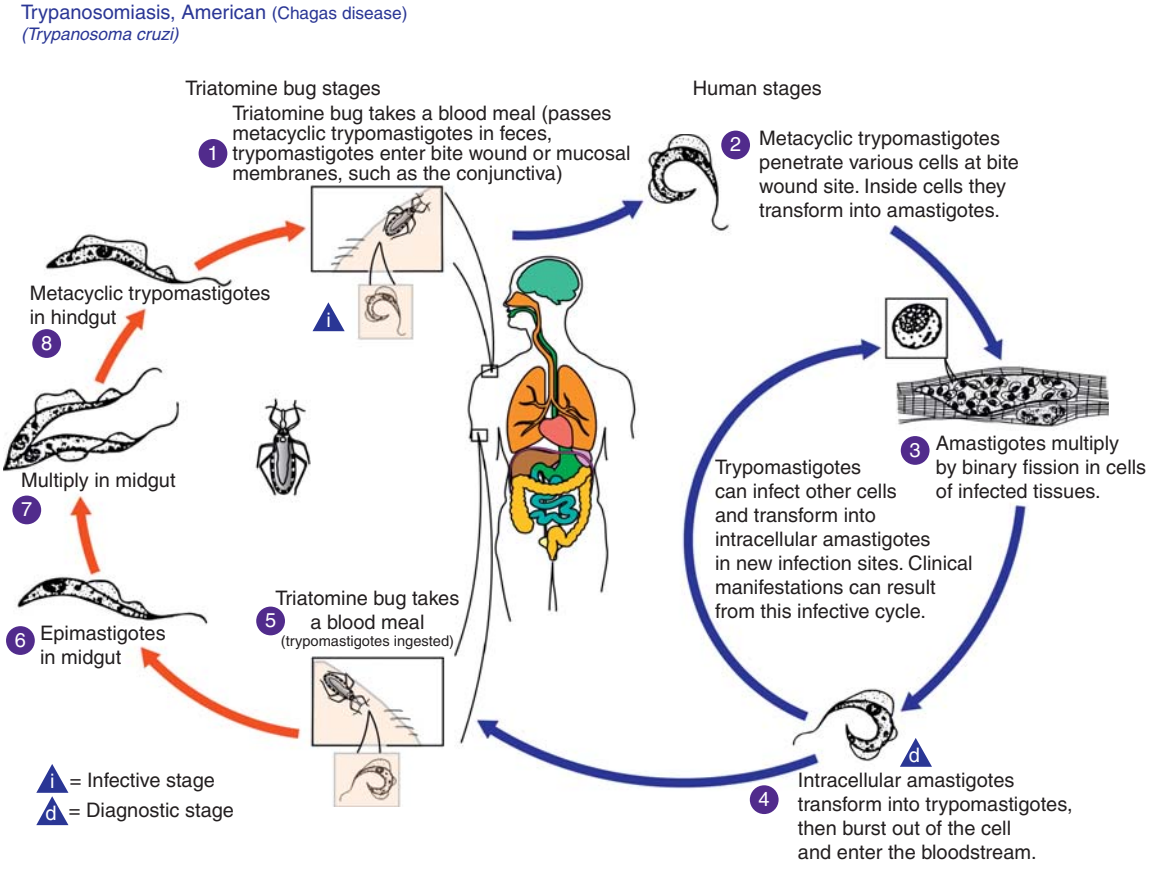

Figure 6 The life cycle of *Trypanosoma cruzi*, the causative protozoan parasite of Chagas disease, or American trypanosomiasis. Reproduced from CDC Image, available at http://www.dpd.cdc.gov/dpdx.

Figure 7 *Triatoma infestans*, a common reduviid vector of *Trypanosoma cruzi* among animals and humans in the Americas. Reproduced from CDC Image, available at http://www.dpd.cdc.gov/dpdx.

climatic, behavioral, and demographic factors. Global warming has significantly extended the distribution range for *T. cruzi*-infected reduviids north of latitude 46°. A migration of armadillos, opossums, nutrias, other rodents, and raccoons, into forested suburban areas has expanded domestic wild animal reservoirs for Chagas disease, especially in the southeastern United States; and outdoor-kenneled dogs are now the major domestic animal reservoirs of Chagas disease in the United States. An initial human migration of Latin Americans with asymptomatic Chagas disease, often acquired in childhood, from rural villages to Latin American cities and peri urban slums resulted in epidemics of urban transfusion and transplant-related Chagas disease in Latin America. Subsequent massive emigration of Latin Americans to US cities has resulted in expanded human reservoirs of *T. cruzi*-infected persons, estimated at over 200 000, now donating blood and organs to limited stockpiles.

Reduviid bites occur at nighttime and are either painless, possibly from combinations of salivary local anesthetics and anticoagulants, or associated with pruritus. The localized pruritus only serves to induce rubbing and scratching by sleepy victims, effectively dispersing infective trypomastigotes across bite-damaged epidermal surfaces or adjacent mucoepidemal junctions.

Since there is no vaccine to prevent Chagas disease and current chemotherapy is limited to only two drugs, most efficacious only in the earliest stages of acute or reactivated *T. cruzi* infections, the best preventive strategies for Chagas disease in travelers to the Americas should be directed at (1) the education of travelers to *T. cruzi*-endemic areas of the Americas in the transmission risks of Chagas disease; (2) a recommendation for sleeping under pyrethroid-impregnated insect nets, especially when staying overnight in thatched and mud-walled huts or unmortared cabins; and (3) a recommendation to travelers to drink only bottled, boiled, or pasteurized beverages; to avoid all local brews, especially those made from local palm trees and sugarcane; and to avoid chewing on unwashed sugarcane stems or palm hearts and avoid using unwashed sugarcane stems as swizzle sticks for beverages.

Conclusions and Recommendations

Several components of climate change, particularly warming temperatures and more frequent drought–rainy season cycles, have supported the success of new vector–pathogen relationships, as in airport and imported malaria transmission. Some insect vectors, particularly mosquitoes, have been given selective advantages by climate change, free trade, and air travel. Introduced pathogens from tropical regions, such as the monkeypox virus and the rat lungworm, have found new animal reservoirs in warming ecosystems north of the Equator.

How should humankind respond to climate change and its inevitable impact on biological systems and the quality and safety of human life? The United Nations, through its agencies and panels, such as the WHO and IPCC, has taken the lead in directing appropriate international responses that will include combinations of environmental, political, regulatory, socioeconomic, and public health measures. Such measures must include limiting anthropogenic greenhouse gases, fostering renewable energy resources, improving natural disaster forecasting, developing drought and disease-resistant food crops, recognizing the disease potential of introduced pathogens in a warming world, instituting sentinel monitoring for infectious diseases in animals and man, and developing primary prevention strategies for climate change-related infectious disease outbreaks and extreme weather events. Primary prevention strategies for emerging infectious diseases should include new vaccines for avian influenza, SARS, and West Nile virus. Primary injury prevention strategies for extreme weather events

should include early warning systems for heat waves, floods, tornadoes, tsunamis, and tropical cyclones.

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