

Infectious Disease Transmission during Air Travel

Part II - Common Vehicle and Vector-Borne Transmission of Infectious Diseases during Air Travel

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Abstract

The combination of a mobile global population with the speed of air travel has made international air travel a competent vector of infectious disease dis-semination. Mathematical modeling has provided new insights into the mechanisms of in-flight disease transmission. The traditionally held tuberculosis or close-proximity model of in-flight disease transmission that side-to-side laminar airflow in air-cabins would restrict the spread of airborne pathogens to within 2 seat-rows has been disproven by the transmission of sudden acute respiratory syndrome and norovirus to passengers at greater distances from index cases. The cooperation of the global aviation industry and the public health community in the health education of the air-traveling public will be necessary to reduce risks of infectious disease transmission during air travel.

Introduction

The combination of a growing and globally mobile population with the convenience, popularity, and speed of air travel has now made international air travel a competent vector of infectious disease transmission and dissemination.¹⁻⁸ This review will describe the epidemiology and outcomes of common vehicle (food or water) and vector-borne infectious diseases transmitted during air travel and recommend strategies for their control and prevention.

Methods

In order to describe the physical features of the aircraft-cabin environment that support infectious disease transmission, the burden and outcomes of infectious diseases transmitted during commercial air travel, and the recommended strategies for infectious disease control and prevention, National Library of Medicine search engines were queried with several key words and subject headings including commercial aircraft, commercial airlines, air travel, infectious diseases, infectious disease transmission during air travel, and food-borne, waterborne, and vector-borne disease transmission.

Results

Common Vehicle Transmission of Infectious Diseases during Air Travel

Salmonellosis: Between 1947 and 1999, 41 in-flight food-borne infectious disease outbreaks were documented and resulted in 11 deaths.^{7,9-12}

Salmonellosis was the most commonly reported food-borne pathogen with 15 documented outbreaks, 4,000 cases, and 7 deaths.^{7,9}

Staphylococcal food poisoning: Between 1947 and 1999, 8 in-flight staphylococcal food poisoning out-breaks were documented with one death.^{7,9} One of the largest outbreaks of staphylococcal food poisoning affected 57% of passengers on an international commercial flight served a contaminated ham omelet in 1975.⁹

Cholera: The first documented in-flight outbreak of cholera occurred in 1972 on an international commercial flight from London to Sydney via Singapore.¹¹ There was one death among the 47 people who contracted cholera, which was associated with eating a cold appetizer.¹¹ During a cholera epidemic in South America in 1992, 75 passengers contracted cholera, which resulted in 10 hospitalizations and one death, during a flight from Buenos Aires to Los Angeles via Lima, Peru.¹² A food trace back investigation implicated a cold seafood dish prepared in a cholera-endemic South American country as the source of *Vibrio cholerae* transmission.¹² Fewer meals served in-flight, the use of prepackaged frozen meals in-flight, improved hygienic food handling by airline caterers, and frequent food inspection by sanitarians have resulted in fewer food-borne bacterial infectious disease outbreaks aboard commercial aircraft in recent years.⁷

Noroviruses: Today, noroviruses are recognized as the most common causes of acute gastroenteritis with diarrhea in adults, the second most common cause of diarrhea in children, and the most common cause of foodborne acute gastroenteritis outbreaks aboard cruise ships.^{13,14} Noroviruses are small, round, single-stranded RNA viruses (Caliciviridae) that are classified by their gene sequences into 3 genogroups (GI, GII, GIV), 25 genotypes, and many subgroups.¹⁵ In recent outbreaks, norovirus strains from genogroup II, genotype 4 (II.4) have been responsible for the majority of outbreaks in schools, hospitals, nursing homes, and on cruise ships.¹⁵ Humans are the only reservoir for noroviruses, which are highly infectious (infectious dose = 18-1,000 virions) and most commonly transmitted by the fecal-oral route via contaminated food or water.¹⁵ Noroviruses are also transmitted by close personal contact, aerosolized vomit droplets, and contact with fomites or environmentally contaminated surfaces, especially in lavatories.¹⁵ Viral shedding in feces precedes illness onset in 30% of cases, peaks by day 1-3 of illness, and may be prolonged in both healthy and, especially, in immunocompromised persons.¹⁵ After an incubation period of 10-51 hours, acute gastroenteritis begins with vomiting followed by fever, abdominal cramps, watery diarrhea, and constitutional symptoms including headache, chills, and myalgias.⁶¹ The illness resolves in 1-3 days, and fatalities are rare, but have been occurred among the elderly in nursing home outbreaks.¹⁵

Since 1991, there have been 5 reports of in-flight transmission of acute norovirus gastroenteritis aboard commercial airline flights.^{10,16-18} In a 1991 outbreak of acute gastroenteritis following commercial air travel, 30 case passengers had a Norwalk-like virus (now designated as norovirus genogroup I, genotype 1, [GI.1]) isolated from fecal samples.¹⁰ In 2005, Widdowson and co-investigators reported a probable acute norovirus gastroenteritis outbreak transmitted by 8 symptomatic flight attendants to passengers on an international flight.¹⁷

On November 2, 2006, a group of passengers with acute norovirus gastroenteritis contracted on an earlier riverboat cruise boarded a short (2.5 hours) commercial flight from St. Louis to Atlanta.^{16,18} Two (5%) of 42 possible secondary cases of norovirus gastroenteritis were confirmed by positive fecal serology.¹⁶ These 2 passengers were not seated in close proximity to the index case-patient and did not use the lavatory in-flight.¹⁶

On October 8, 2008, a California-based tour group boarded a commercial airline flight from Boston to Los Angeles that included several travellers already ill with acute gastroenteritis, later confirmed as norovirus genogroup II.¹⁹ Within 3 hours of departure, 6 tour group travelers experienced severe nausea and vomiting, resulting in an emergency diversion of the commercial flight to Chicago, Illinois.¹⁹ Of the 106 non-tour group passengers on the flight, 7

met the case definition for probable secondary cases, and, of these, one submitted a positive fecal sample for norovirus genogroup II, confirming in-flight transmission.¹⁹

The noroviruses have now replaced bacterial infections and enterotoxins as the most common causative agents of acute gastroenteritis transmitted during commercial air travel.

Noroviruses are highly infectious and may be transmitted not only by the fecal-oral route during food preparation by virus shedders, but also by aerosolization of vomitus (and possibly feces), by close personal contact, and by indirect contact with contaminated fomites and lavatory surfaces. Secondary cases of norovirus have been transmitted in flight at significant distances from symptomatic index case patients and to passengers not using on-board lavatories.¹⁶

Prevention and control strategies for norovirus transmission during aircraft outbreaks include excluding ill air crew workers from any food handling or service; avoiding the lavatory in flight; frequent hand washing and alcohol-based (> 62%) sanitizer use; avoiding close contact with and shaking hands with index cases; and promptly disinfecting lavatories and surfaces soiled by any secretions from index cases with dilute bleach solutions (1:50 to 1:10 dilutions of household 5.25% bleach) or other approved disinfectants; and hot cycle washing or disposal of any soiled linens or clothing.

Vector-borne Transmission of Infectious Diseases during Air Travel

Malaria: In 1983, random searches of airplanes arriving from tropical countries at London's Gatwick Airport found that 12 of 67 airplanes contained mosquitoes.²⁰ After a female mosquito leaves the aircraft, she may survive long enough, especially during the temperate periods, to take a blood meal and transmit pathogens, usually in the vicinity of an international airport (airport malaria). As international air travel between malaria-endemic nations and malaria non-endemic 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 km and 15 km from Gatwick Airport.²⁰ Hot, humid weather in Britain may have facilitated the survival of imported, infected anopheline mosquitoes.²⁰ During the summer of 1994, six cases of airport malaria were diagnosed in the vicinity of Charles de Gaulle Airport near Paris.²¹ Four of the patients were airport workers, infected at work, and the others were residents of Villeparisis, a suburb 7.5 km away from the airport.²¹

In addition to airport malaria transmitted by infected mosquitoes, many developed countries are reporting an increasing number of cases of imported malaria due to an increase in long-distance air travel by infected passengers. Annual malaria cases imported from Africa and Asia to the United Kingdom (UK) rose from 803 in 1987 to 1,165 in 1993.^{22,23} By 2006, 1,758 imported malaria cases were reported in the UK.^{22,23} From 1990 to 1998, the annual number of imported malaria cases in Italy increased by 100% due to the rising rates of immigration and international travel, with immigrants accounting for most of the cases currently.²⁴ In the US in 2005, a total of 1,528 cases of imported malaria were diagnosed, an increase of 15% over the prior year.²⁵ Today, imported malaria is the most common type of malaria in developed nations with over 30,000 cases reported annually.²⁵ Imported malaria remains the most common cause of fever in travelers returning from high-risk, malaria-endemic regions.²⁶

In a retrospective analysis of 380 imported malaria cases in Verona, Italy, over the 5-year period 2000-2004, 2008, Mascarello and coauthors reported that most cases occurred in

adults (337 adults vs. 43 children), in immigrants (n = 181, 48% of adults), in patients returning from Africa (n = 359, 94.5%), and in travelers returning from visiting friends and relatives (VFRs) in malaria-endemic regions (n = 154, 40.5%).²⁷

Most cases were caused by single *P. falciparum* infections (n = 292, 76.8%) with few mixed infections (n = 23, 6%).²⁷ The authors concluded that malaria in travelers returning to Verona from Africa was not uncommon and targeted certain high-risk travelers including adult expatriate immigrants visiting friends and relatives (VFRs), semi-immune children (recent immigrants), and non-immune children (expatriate or born in Italy).²⁷

In a similar retrospective analysis of 109 travellers with malaria returning to Basel, Switzerland, over the period 1994-2004, Thierfelder and co-investigators reported that *P. falciparum* was the most common causative parasite (84%); most infections were acquired in Africa in immigrants visiting friends and relatives (82%); and the mean incubation period was 4 days (range 0.5-31 days).²⁸ Following their descriptive analysis, the investigators conducted 3 comparative analyses with 2 prior studies of malaria in travelers returning to Basel during the periods 1970-1986 and 1987-1992.²⁸ The results of their comparative analyses included significant increases in the proportions of *P. falciparum* infections over 3 study periods (1970-1986, 49%; 1987-1992, 75%; 1994-2004, 88%) and significant increases ($p < 0.001$) in hospitalizations for *P. falciparum* malaria over the three decades studied.²⁸ The authors concluded that there was a significant trend towards more serious malaria infections with *P. falciparum* in immigrants returning to Basel after visiting friends and relatives in their malaria-endemic native homelands.²⁸

In 2008, Rodger and coauthors reported a cluster of six cases of *P. falciparum* malaria at a British airport among 30 students returning to the US after spending 2 months in East Africa in 2005.²³ Of the 6 patients, all were young (range = 19-22 years of age) and in prior excellent health; and 5 of the 6 exhibited features of acute cerebral malaria (disorientation, prostration) requiring urgent intensive care and therapy with intravenous quinine.²³ The authors commended alert UK-airport staff for recognizing the seriously ill travellers preparing to board a 9-hour second-leg flight to the US, and for rapidly evacuating the patients to the nearest healthcare facility for intensive care, without which the 5 cerebral malaria cases would likely have been fatal.²³

Although many developed nations do not have *P. falciparum* mosquito vectors as competent as *A. gambiae* in sub-Saharan Africa, southern Europe, the southern US, the Middle East, and Asia do have capable anopheline vectors for *P. falciparum* malaria; and most also have competent vectors for *P. vivax* malaria. The most disturbing recent trends in imported malaria today include the following: (1) an increasing proportion of *P. falciparum* infections capable of causing cerebral malaria and renal failure with the highest case fatality rates (CFRs); and (2) increasing immigration from malaria-endemic regions to malaria-free regions in developed nations creating a unique set of high-risk travellers, especially expatriates and their children returning from visiting friends and relatives in their malaria-endemic, native homelands.

Imported malaria cases are now increasing world-wide due to the ease and relatively low costs of international air travel to-and-from malaria-endemic regions. The world's malaria-endemic regions now have expanded distribution ranges for malaria transmission and longer mosquito vector breeding and feeding seasons due to global warming and increasing drought-monsoon cycles.^{25, 26}

Dengue: One third of the world's population lives in dengue-epidemic areas in over 100 countries.^{29,30} In 2002, Guzman and Kouri estimated that 100 million cases of dengue fever and 250,000 cases of dengue hemorrhagic fever (DHF) occurred annually.³⁰ Today, dengue prevalence has been increased by a greater frequency of dengue epidemics in Africa, Asia, Latin America, and the Caribbean.^{31,32} Imported dengue has now surpassed imported malaria as the most commonly reported cause of acute febrile illness in travelers returning from all of the world's tropical regions except Africa.^{33,34}

In 2008, Schwartz and co-authors retrospectively examined the annual trends and seasonality of dengue cases in returning travelers reported to the International GeoSentinel Surveillance Network.³⁵ The authors reported that the incidence of dengue in returning travellers increased from 50 cases per 1,000 returned travellers with acute febrile illnesses in non-epidemic years to 159 cases per 1,000 ill travelers in epidemic years.³⁵ The incidence of dengue in returning travelers also demonstrated seasonality with region-specific peaks for Southeast Asia (June, September), South Central Asia (October), South America (March), and the Caribbean (August, October).³⁵ In a similar investigation in returning travelers with acute febrile illnesses reported to the CDC's Passive Dengue Surveillance System during the 10-year period, 1996-2005, Mohammed and co-investigators reported 1,196 suspected imported dengue cases, with 334 (28%) confirmed as laboratory-positive by both serologic and virologic techniques.³⁶ The authors also reported an overall increase in dengue incidence in returning travelers over the 10-year study period with the Caribbean, Mexico, Central America, and Asia as the most commonly visited regions.³⁶ There is now substantial epidemiologic evidence that imported dengue infections are increasing in travelers returning to the US and other temperate nations from travel experiences in popular vacation destinations in tropical, dengue-endemic regions of the Americas, the Caribbean, and Asia, where dengue epidemics are occurring with greater frequencies.^{6,35,36}

The spectrum of clinical illness in dengue infections may range from asymptomatic initial infections after a 5-day incubation period to subsequent infections complicated by DHF and dengue shock syndrome (DSS) with high CFRs. Viremia lasts 4-5 days throughout the acute febrile illness and recovery during which time the dengue virus may be transmitted by *Aedes* species mosquito bites or blood transfusions. Although the traditional mosquito vector of dengue (and yellow fever) is *Aedes aegypti*, the Asian tiger mosquito, *Aedes albopictus*, has recently emerged as a competent secondary vector of dengue (Figure 1). *Aedes albopictus* has now been distributed worldwide by the international tire trade, and has now been identified as the primary mosquito vector in recent dengue and chikungunya epidemics in India and the Indian Ocean islands.³⁷

Although most cases of dengue in developed nations are imported and rarely autochthonous, dengue could be transmitted by infected *Aedes* species mosquitoes either on aircraft or in airports.³⁷ In 1998, dengue fever virus infections were reported in a German couple returning to Germany from a vacation in Hawaii; and airport transmission by an *Aedes* species mosquito was suspected, rather than in-flight mosquito transmission.³⁸

Air-imported dengue will pose a much greater threat to the US and other developed nations than imported malaria for several reasons including: (1) There is a new secondary vector, *Aedes albopictus* that is now dispersed throughout the US (26 states) and worldwide.³⁷ (2) Although initial cases of dengue are often mild or subclinical, they are infectious and may be transmitted by mosquitoes and blood transfusions; and subsequent infections can be fatal.³⁷ (3) Unlike malaria, there are no chemoprophylactic or therapeutic treatment options for

dengue.³⁷ (4) Lastly, there is no vaccine for dengue prevention, and vaccine development has been hampered by multiple serotypes and inherent risks of vaccine-induced autologous cytokines responsible for DHF and DSS.³⁷

Chikungunya virus: Chikungunya, a viral disease characterized by fever and painful arthralgias, was first described in Tanganyika (now Tanzania) in 1952-53.^{39,40} The causative virus was later isolated and classified as a positive strand RNA alphavirus (Togaviridae).³⁹ In the Makonde language of East Africa, "chikungunya" means "that which bends up", a vivid description of the stooped and painful posture that may develop after the persisting arthritic symptoms that follow the acute febrile illness.⁴⁰ Chikungunya fever shares many of the same clinical and epidemiological characteristics as dengue fever including: (1) an acute febrile illness with high viremias, often complicated by coagulopathies and liver dysfunction; (2) arthropod-borne transmission by *Aedes* species mosquitoes, originally limited to *Aedes aegypti* in Africa and Asia, and now supported by a widely distributed new vector, *Aedes albopictus*; (3) a reservoir in humans with high viremias that supports both human-mosquito-human transmission and nosocomial human-to-human transmission; (4) a capacity for recurrent infections by multiple genotypes; (5) no specific chemoprophylactic or therapeutic treatment options, and no vaccine for primary prevention; and (6) increasing air importation by infected travelers returning from vacations in popular disease-endemic locations to developed countries with temperate climates, competent *Aedes* vectors, and no herd immunity—all of which will support autochthonous transmission.⁴⁰

Beginning in 2004, there have been recurrent epidemic outbreaks of chikungunya in Africa and Asia, starting in Kenya (2004), spreading rapidly to the islands of the Indian Ocean (2005-2006), and to India (2005-2007).^{40,41} The initial mosquito vector of chikungunya in Africa (Kenya) and the Indian Ocean islands (Comoros) was *Aedes aegypti*, later replaced by *Aedes albopictus* as a primary vector in subsequent Indian Ocean island (Mauritius, Madagascar, Mayotte, Seychelles, Reunion) and Indian epidemics.^{40,41} By early 2005, air imported cases of chikungunya were being increasingly reported in Europe in travelers returning from vacations in Indian Ocean island resorts.⁴⁰ From April 1, 2005, to February 28, 2006, 307 imported cases of chikungunya, were confirmed in France.⁴² By 2006, imported cases were also confirmed in Germany, UK, Belgium, Switzerland, Norway, China, and the Czech Republic.⁴³ In March 2006, human-to-human transmission first occurred in France in a nurse caring for a patient with chikungunya and high viremia.^{40,41} During 2005-2006, the US CDC reported 12 cases of air imported chikungunya confirmed both serologically and virologically in travelers who returned to the US from regions known to be epidemic or endemic for chikungunya.⁴⁴ In recent chikungunya outbreaks in West Africa (Gabon, 2007) and India (Delhi, 2006), co-infections with chikungunya and dengue viruses were initially reported with *A. albopictus* identified as the principal vector for both viruses in the African outbreak.^{45,46} In 2008, Bonilauri and co-investigators isolated chikungunya viruses from *Aedes albopictus* mosquitoes collected in Italy and concluded that the virus was in active circulation in competent mosquito vectors in Italy and ready for autochthonous transmission.⁴⁷ Like the West Nile virus, the chikungunya virus has now been distributed by global air travel from its origins in Africa to developed nations in Europe with a competent mosquito vector, *A. albopictus*, another recent import; a welcoming temperate climate, and an absence of herd immunity.^{37,41,47}

The origin of the terms "airport malaria" and "disinsection" have been attributed to Dr. Norman G. Gratz (1925-1985), a medical entomologist and long-term Director of Vector Biology and Control at the World Health Organization (WHO), who wrote in 1999: "The

number of cases of 'airport malaria', plus cases of airport things we do not know about, demonstrate that aircraft disinfection (later 'disinsection') is often very necessary indeed."⁴⁸ The WHO issues International Health Regulations (IHR) periodically with recommendations for air-craft disinfection and disinsection (available at <http://www.who.int/csr/ihr/en> - last accessed Dec-ember 2010), which some vector-borne disease endemic nations enforce. The most commonly used insecticides which are sprayed, with or without prior announcement, on passengers and their belongings in the aircraft cabin are the synthetic pyrethroids, usually d-phenothrin or permethrin. The synthetic pyrethroids are considered the safest pesticides for human use; are found in most insect spray and aerosol repellants; are used to impregnate clothes, curtains, and fabrics; and may precipitate bronchospasm if sprayed in the face, especially in chemically sensitive or atopic persons.

Prevention and control strategies for the in flight transmission of vector-borne infectious diseases by infected arthropod vectors include combinations of the following: (1) personal protection of passengers and air crews on the ground with topically applied insect repellants containing 20-40% DEET (N, N-diethyl-3-methylbenzamide) or 7-20% picardin; (2) the use of pyrethroid-impregnated seat cushions or upholstered fabrics, pyrethroid-impregnated air cur-tains on cabin outer doors, between cabin compart-ments, and on outer exits of airport jet-ways and boarding ramps; and (3) the disinsection of aircraft with aerosolized pyrethroids as required by nations enforcing the WHO IHR.⁴⁹

Conclusions

Prepackaged frozen meals requiring less food handling in flight may have indeed reduced the fecal-oral transmitted outbreaks of enteric bacterial and entero-toxic acute gastroenteritis, but have not restricted out-breaks of noroviruses, which are highly pathogenic, environmentally stable, and can be aerosolized as droplet nuclei and spread by fomites and contact with contaminated surfaces. Despite the early warnings by Dr. Norman Gratz and others, vector-borne infectious diseases, such as West Nile virus and chikungunya, have now been effectively disseminated by air travel and have led to autochthonous transmission and clus-ter outbreaks in developed nations with competent local mosquito vectors.^{37,41-44,48} Lastly, human behavior can neither be predicted nor controlled, rendering mass pre-flight health screening for communicable diseases inefficient and impractical. Only the cooper-ation of the global aviation industry and the medical and public health communities in the health educa-tion of the air-traveling public will ameliorate, but never eliminate, the risks of infectious disease trans-mission during air travel.

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Figure 1.

An *Aedes albopictus* female is shown taking a human blood meal. *A. albopictus* is a new competent mosquito vector for both the chikungunya and dengue viruses that has now been disseminated globally by international trade and is poised for local transmission. Source: United States Centers for Disease Control and Prevention (CDC), Atlanta, Georgia USA.

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