Research on the Transmission of Disease in Airports and on Aircraft

DETAILS

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Research on the Transmission of Disease in Airports and on Aircraft

Summary of a Symposium

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Rapporteur

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This report has been reviewed by a group other than the authors according to the procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

This project was sponsored by the Airport Cooperative Research Program and the Transportation Research Board.

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Preface

In September 2009, about 100 people assembled in Washington, D.C., to participate in a symposium on research on the transmission of disease in airports and on aircraft. The symposium brought together individuals from the public sector (federal, state, and local agencies including public airports), private sector (airlines and consultants with expertise in various facets of airport emergency response), and research institutions to learn about current research and to consider ways to conduct and fund future research.

The symposium goals were to examine (*a*) the status of research on or related to the transmission of disease on aircraft and in airports, (*b*) the potential application of research results to the development of protocols and standards for managing communicable disease incidents in an aviation setting, and (*c*) areas where additional research is needed. To plan the event, TRB assembled a committee appointed by the National Research Council (NRC) to organize and develop the symposium program. The planning committee was chaired by Katherine B. Andrus, Air Transport Association of America, Inc.

The symposium program was designed to provide an opportunity for the aviation community to share data, models, and methods; discuss findings and preliminary conclusions of ongoing research; and identify gaps to inform future research projects. During the symposium, consecutive sessions were organized according to different approaches to research as identified by the planning committee. These approaches included case study investigations, theoretical modeling, and "bench science" experimental methods. A session discussing different approaches to policies and planning to minimize the spread of disease along with an open dialog among all attendees on candidate topics for future research was also conducted.

This summary report contains white papers, authored by the invited speakers to each session, that summarize the presentations they gave during the symposium. It includes a summary of the discussion of topics for future research. The planning committee was solely responsible for organizing the symposium, identifying topics, and choosing speakers. The responsibility for the published symposium summary rests with the symposium rapporteur and the institution.

This report has been reviewed in draft form by individuals chosen for their diverse perspectives and technical expertise in accordance with procedures approved by the NRC Report Review Committee. The purposes of this independent review are to provide candid and critical comments that will assist the institution in making the published report as sound as possible and to ensure that the report meets institutional standards for objectivity, evidence, and responsiveness to the project charge. The review comments and draft manuscript remain confidential to protect the integrity of the process.

TRB thanks the following individuals for their review of this report: Katherine B. Andrus, Air Transport Association of America, Inc.; Deborah C. McElroy, Airports Council International–North America; and Phyllis Kozarsky, Expert Consultant, Centers for Disease Control and Prevention. Although the reviewers provided many constructive comments and suggestions, they did not see the final draft of the report before its release. The review of this report was overseen by C. Michael Walton, Ernest H. Cockrell Centennial Chair in Engineering,

University of Texas at Austin. Appointed by NRC, he was responsible for ensuring that an independent examination of this report was carried out in accordance with institutional procedures and that all review comments were carefully considered.

The committee extends special thanks to the Airport Cooperative Research Program Oversight Committee for providing funding support for the workshop along with the vision and encouragement that made the event the success that it was.

Overview

Christine L. Gerencher, *Transportation Research Board*

On September 17–18, 2009, a diverse group representing academia, government, industry, and nonprofit organizations came together to share insights into the transmission of disease in airports and on aircraft. The symposium was the result of almost 8 months of planning and discussion by a committee chaired by Katherine B. Andrus, Air Transport Association of America, Inc., that included experts from the public sector (federal, state, and local agencies including public airports), private sector (airlines and consultants with expertise in various facets of airport emergency response), and research institutions. When planning began on the program, the committee knew it was an important topic but had no idea it would turn out to be so timely. The outbreak and rapid spread of the H1N1 influenza virus in April 2009 brought renewed attention to communicable diseases.

Although the H1N1 pandemic underscored the role that travel generally plays in the spread of disease, the planning committee decided to focus on the actual transmission of disease during air travel. The movement of infected people has always contributed to the spread of disease from one place to another, and air travel affects the pattern and rate of that spread. However, the committee determined there was enough interest in and uncertainty about the spread of disease within the aircraft and airport environment to justify devoting the symposium to that topic.

The symposium opened with an introductory session that laid the groundwork for a common understanding of how infectious disease is spread generally, how aircraft are ventilated, and how travel plays a role in spreading disease. After that session, three panels of leading researchers in their respective fields presented the science that underlies our current understanding of how pathogens may be transmitted in the specialized environment of the aircraft cabin and in airport facilities. The panels were organized by different approaches to research: case study investigations, theoretical modeling, and "bench science" experimental methods.

On Day 2, the focus shifted to the practices and policies that can be informed by science but too often are not. Whether the task is applying pesticides to aircraft in an effort to control vector-borne diseases, developing airline and airport sanitation measures, or imposing travel restrictions to stem the spread of a pandemic, more scientific evidence could help to determine the effectiveness of current practices, subjecting them to more rigorous analysis. In the concluding session, members of the audience joined the session moderators in identifying areas in which more research is needed to understand and mitigate the transmission of disease in air travel.

Over the course of the symposium, there were many opportunities for the exchange of ideas, and the resulting discussions illustrated the benefits of bringing together researchers from different disciplines along with potential consumers of that research. The different perspectives and expertise brought to bear on these issues identified some new paths to explore, as described in the tables provided in Session 6: Discussion of Topics for Future Research. Perhaps as important, the connections forged over a day and a half promise to lead to future collaborations that

will leverage available talent and resources and improve the aviation community's ability to gain a more complete scientific understanding of the topic.

The following papers are summaries of the presentations that were written and provided by the invited speakers to the symposium. These papers have not been peer reviewed and are intended only as written summaries of the research discussed in the presentations during the symposium. Not all speakers provided papers, so only those received are included in this document.

SESSION 1

Understanding How Disease Is Transmitted via Air Travel

Jeanne Yu, *Boeing Commercial Airplanes* (Presenter)
Ben S. Cooper, *United Kingdom Health Protection Agency* (Presenter)

The Aircraft Cabin Environment

Jeanne Yu (Presenter)

Travel is all about people moving! The overall travel experience includes many elements as a person moves from one location to another; we think about the travel experience in the context of a "door-to-door experience." Travelers can experience many environments, moving from ground transport to an airport to an airplane to another airport and to more ground transport before arriving at their final destination. To further our understanding of disease transmission at airports and on aircraft, it is important to recognize that the airplane flight is just one phase of the overall travel experience and that disease transmission can occur during all phases of the door-to-door experience.

This white paper describes the aircraft cabin environment part of the travel experience and how airplane systems work to provide the air you breathe in the aircraft cabin environment. This paper also addresses items that should be considered for aircraft cleaning and disinfection if a significant disease transmission event occurs.

Airplanes typically fly at 36,000 ft. To put this number in context, Mt. Everest is about 29,000 ft high. The environment is extreme at 36,000 ft:

- Very cold: −45°F (−43°C) to −85°F (−65°C);
- Very dry: less than 1% humidity;
- Very low pressure; and
- Naturally occurring ozone.

To sustain human life, advanced environmental control systems (ECSs) are needed. They control multiple important functions: cabin pressure, ventilation, temperature, anti-icing, and fire and smoke protection.

Aircraft ECS designs must meet FAA regulatory requirements for safety and health, such as cabin pressure (8,000 ft maximum) and ventilation (0.55 lb/min/person) and should not exceed threshold maximums for carbon monoxide, carbon dioxide, and ozone. The aircraft cabin environment also strives to meet objectives for comfort based on industry standards: Temperature (T) [65°F to 85°F, ΔT < 5°F within a temperature control zone, SAE Aerospace Recommended Practices (ARP) 85]=

- Rates of pressurization (climb. 500 ft/min; descent. 300 ft/min, SAE ARP 1270);
- Cabin air velocities (<60 ft/min, optimal 20 to 40 ft/min, SAE ARP 85);
- Aisle flow considerations for odor, temperature, ventilation mitigation; and
- Cabin air treatment (SAE ARP 85).

How is air provided to the aircraft cabin? In today's aircraft design, outside air at 36,000 ft continuously enters the engine. At this altitude, the air is very clean, dry, low in oxygen, and practically particle-free. The air is compressed in the engine compressors and then extracted upstream of the combustion process; it travels in high-pressure ducts along the wing to the wing box of the aircraft. Here the air can pass through a cata-

lytic ozone converter to remove the naturally occurring ozone at altitude. The air then travels to the air conditioning pack, which houses many components, such as its own compressor, turbine, and heat exchanger. Once the air is conditioned to the appropriate pressure and temperature, it goes to the mix manifold where it is mixed with highly filtered recirculated air in about a 50/50 ratio. Boeing aircraft use high-efficiency particulate air (HEPA) filters with an efficiency of 99.97% at a particle size of 0.3 micrometer (μm) in diameter. In Figure 1, the vertical axis shows filter efficiency, and the horizontal axis shows particle size. HEPA filters are ≥99% efficient over a particle size ranging from 0.003 to 10 μm, which encompasses a single virus and bacteria.

Air from the mix manifold is supplied to the cabin through the air distribution system via riser ducts to the overhead cabin region and then through downer ducts into air supply nozzles that introduce the air into the aircraft cabin. The ECS is fully automated and air distribution is set by aircraft design.

The ECS design goal for air supplied to the cabin is to generate a two-dimensional profile in a seat row to minimize drafts, temperature gradients, and odor migration. However, some three-dimensional aisle flow is inherent in the design and can be affected by movements such as galleys and occupants moving in the aisle. Air flows continuously into the cabin through the air distribution system and leaves the cabin through return air grilles that run the length of the cabin on both sides where the side wall meets the floor. The Harvard 1997 transportation study and other studies from 1987 to 1998 have measured the microbial level in different indoor environments. The measured levels of contaminants in aircraft cabin air are low compared with other indoor environments.

Air also flows continuously out of the airplane through the outflow valve. The outflow valve regulates outflow of air and thus cabin pressure. The cabin pressure system controls the cabin pressure so that as the airplane climbs to its maximum certification altitude (40,000 to 45,000 ft depending on airplane type), the cabin pressure climbs to about 8,000 ft. Airplanes do not usually fly at their maximum altitude; typically, they fly at an altitude of about 36,000 ft. The resulting aircraft cabin pressure is around 6,000 ft, which is similar to being in a tall building in Denver, Colorado.

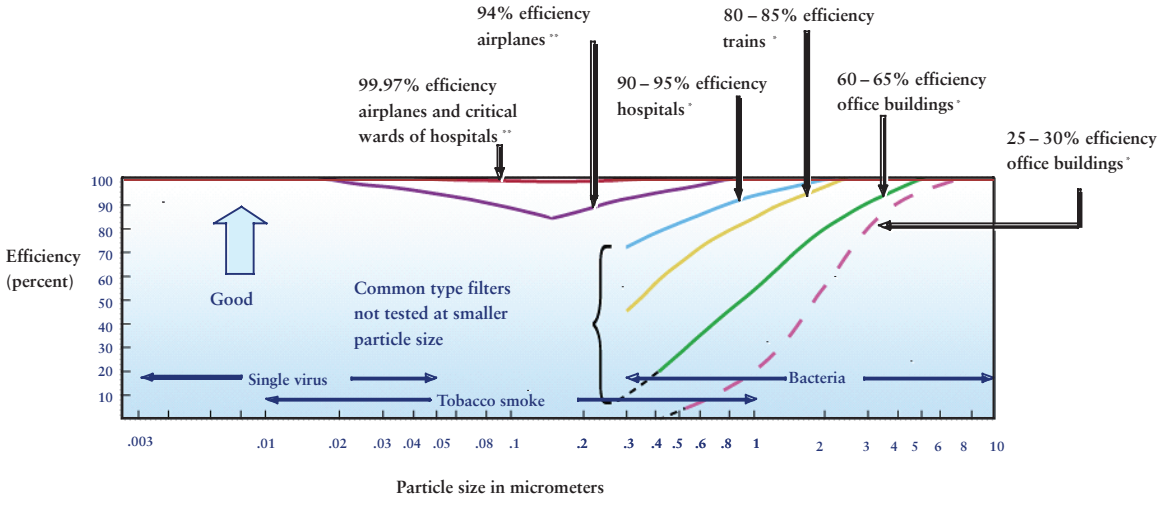
More detail and an animation showing how the air is provided to the cabin can be found at www.boeing.com/commercial/cabinair/.

ECSs are fully automated so that air flow rates to the cabin and to the flight deck are set by aircraft design. Flight decks on some aircraft receive a 50/50 ratio of outside-to-recirculated air and some receive all outside air depending on the requirements and challenges of the flight deck air distribution design: electronic cooling, high solar loading from windshields, and higher pressure required in the event of smoke or fire.

Pressurized cargo compartments can carry live animals. Depending on the model, systems to heat ventilate and air-condition cargo holds are standard or optional.

Boeing defers to appropriate authorities for disinfection of aircraft: the Centers for Disease Control and Prevention (CDC), the U.S. Environmental Protection Agency, and the United Nations World Health Organization (WHO)

- CDC recommendations for airlines: air travel industry;



* ASHRAE 52–76 ** (IEST) Filter type "B" VERV17

FIGURE 1 Comparative analysis of HEPA filters used in Boeing aircraft versus other applications.

- WHO: website and document, "Guide to Hygiene and Sanitation in Aviation;" and
- International Air Transport Association: website for "Health & Safety for Passengers and Crew."

Boeing also supports the following:

- Research and working with the U.S. Department of Agriculture Animal and Plant Health Inspection Service to develop consistent guidelines with all original equipment manufacturers on inspecting, cleaning, and disinfecting contaminated aircraft; and
- Airline event response with aircraft cleaning and disinfection guidelines, including an approved material-compatible cleaners list.

Aircraft cleaning and disinfection require substances that will not degrade aircraft materials. Boeing tests for material compatibility but does not test for substance efficacy against disease agents. Disinfection materials manufacturers and government agencies are responsible for efficacy testing.

Boeing outlines requirements in the following:

- Aircraft maintenance manuals that include safety instructions;
- Boeing document, "Cleaning Interiors of Commercial Aircraft;" and
- Boeing document, "Evaluation of Maintenance Materials."

Boeing research and collaboration are ongoing with academia and industry to further our understanding. We continue to work with the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) and industry collaboration to understand potential leverage points in ASHRAE's strategic research agenda being developed to address the role of heating, ventilation, and air conditioning systems in the spread of infectious disease.

We also are working toward maturing computational modeling capabilities. With Purdue University, we are developing model characterization of exhaled airflow from various modes of human respiration, including breathing, talking, and coughing. With the FAA Airliner Cabin Environment Research partners, we are studying additional modeling capabilities of moving bodies in the aircraft cabin.

In summary, travel is a phenomenon of people moving; the aircraft flight is one part of a traveler's door-to-door experience. Aircraft ECSs are fully automated and designed to meet unique requirements for passenger safety and comfort. Aircraft disinfection must take material compatibility issues into consideration. Further integrated collaborative research is needed.

Human Movement Patterns and the Spread of Infectious Diseases

Ben S. Cooper (Presenter)

Patterns of human movement are fundamental to the persistence, spatial distribution, and dynamics of human infectious diseases. Research aimed at teasing apart the complex relationship between human movement patterns and infectious disease dynamics has intensified in recent years, particularly since the 2002–2003 epidemic of coronavirus association with severe acute respiratory syndrome (SARS) and with concerns about a possible influenza H5N1 pandemic. However, the roots of this research go back much further.

One way to appreciate the role of travel in the spread of infectious disease is to consider what would happen if people did not move among communities. Research based on mathematical models in the 1950s and 1960s shows that without such movements immunizing infections such as measles would not be able to persist below a critical population size: in the troughs between epidemic peaks the numbers infected would fall to zero, and no further cases would occur without reintroduction from outside the community (*1, 2*). For measles, this critical population size was found to be about 300,000. The theory predicts that island populations below this size would be too small to sustain measles epidemics, and extended periods with no measles cases (until reintroduction of the virus) would be likely. Above this size, such stochastic fadeouts are unlikely and populations are large enough to maintain a continual presence of the pathogen. Later analysis of measles data from island populations has largely confirmed these predictions from mathematical models (*3*).

Such considerations apply not only to actual islands but also to inland islands: the cities, towns, and villages where we live. Over the last 20 years theoretical epidemiologists have extensively studied the spread of disease not just in a single population, but in metapopulations, or populations of populations coupled by travel links (*4*). In these cities and towns, population size plays a role similar to that observed on islands, although coupling (due to human movement) between population centers tends to be stronger. Large populations have a sufficient influx of people susceptible to infection (either through birth, as in the case of measles, or through loss of immunity) to maintain the pathogen throughout the year, typically resulting in a regular seasonal epidemic pattern (*5*). The smaller the population the more likely stochastic fadeout (epidemic extinction) is to occur. This situation is due to the relative size of the stochastic fluctuations being larger for smaller populations, and the chance of the number infected reaching zero and the epidemic ending is correspondingly greater. If these small populations are not

linked by travel to other population centers, transmission in these settings will end. Conversely, as coupling via transport networks strengthens, epidemics become more synchronized in the different population centers. Recent studies have shown how epidemic synchrony between different population centers can be explained by human movement patterns (*6*). At a more fundamental level, many human pathogens (including measles and influenza) are believed to have made the transition from their original animal hosts with the advent of agriculture, when humans began to change from living in small relatively isolated groupings of hunter gatherers to larger communities (*7*).

Air travel has an effect similar to that of any other means of human movement: by connecting geographically isolated populations, it allows disease to spread between them and enables pathogens to persist by reducing the chance of local stochastic fadeout. What makes air travel unique is its speed, which allows links between populations separated by large distances to be maintained for pathogens with short generation times. Using influenza (which has a generation time of about 3 days) as an example, before the advent of the steamship, a passenger traveling from Europe to America infected immediately before embarkation would have had virtually no chance of transporting the virus between continents. Had Columbus been latently infected with influenza when setting out in 1492 for his 70-day Atlantic crossing, about 23 generations of influenza transmission on his carrack would have been required for the epidemic to spread to the Americas. With a crew of 70 men, this feat would have been almost impossible. In contrast, smallpox, with a generation time of 15 days, would have required only four or five generations of transmission on the ship to cross continents, making intercontinental spread quite feasible.

With the advent of the steamer, Atlantic crossing times decreased to just a few days (a troop ship crossing the Atlantic in 1918 took about 7 days) and only about two generations of transmission were required to transmit influenza between continents, ensuring efficient global dissemination of the 20th century's first pandemic. Air travel now represents by far the most important means for the rapid global dissemination of human pathogens—partly because it is the predominant means of transporting people over large distances but also because the short transit times make it an extremely efficient means of ensuring that even pathogens with very short generation times can be transported over very large distances. These concerns led to work carried out at the United Kingdom's Health Protection Agency to determine whether practical measures could be taken to reduce this international spread in the event of a major pandemic with a virulent pathogen, particularly pandemic influenza.

First, we examined the potential role of airport entry screening. Entry screening of passengers with thermal imaging technology was used by a number of countries during the SARS epidemic and also by some during the 2009 H1N1 pandemic. A very simple analysis was able to show that, even if the sensitivity and specificity of the imaging technology used to detect symptomatic SARS or influenza infection were perfect (which is very far from being the case), the practice would have almost no value in protecting populations from influenza or SARS (*8*). This conclusion resulted from an elementary consideration of flight times and incubation periods for the two pathogens. Only 1% to 6% of passengers incubating SARS when boarding a plane would be expected to develop symptoms by the time they arrived in the United Kingdom (the higher percentage corresponding to the longer flight times), so almost all cases arriving in the United Kingdom would be missed, even with perfect screening. For influenza, which has a shorter incubation period, the corresponding range was 4% to 17%. The large number of passengers infected with influenza while traveling would mean that even if 17% could be detected and isolated, there would be no detectable impact on the epidemic in the destination country.

Given that entry screening had been shown not to be an effective strategy, we considered whether canceling flights from affected cities could significantly alter the pattern of global spread in an influenza pandemic (*9*). Although we did not expect flight cancelation to be able to stop the global spread of influenza (the virus spread around the world quite efficiently in 1918 without the help of air travel), an important question was whether global dissemination could be delayed sufficiently to allow time for the development and production of a vaccine that would protect against the pandemic virus (a process expected to take about 6 months). To address this question, we built on work started by Rvachev and colleagues working in the former Soviet Union in the 1960s (*10*). Rvachev had developed meta-population models to study the spatial dissemination of influenza. Originally, this work considered population centers linked by rail networks, but it was then extended by Rvachev and Longini to account for the global spread of influenza through the international aviation network (*11*). Our own work further extended these early efforts by recasting the deterministic global metapopulation models into a more realistic stochastic framework (which is important because at the beginning of the epidemic in each city, the numbers infected are small, stochastic effects are dominant, and the times of seeding new epidemics in each city are expected to show considerable chance variation). In contrast to earlier work, we paid particular attention to a careful parameterization of the model by comparing air travel and influenza data from the 1968–1969 pandemic. This comparison was impor-

tant for arriving at plausible values for the reproduction of pandemic influenza [before undertaking this work, no reliable estimates had been published, but estimates published concurrently with our analysis yielded results similar to those obtained with our model (*12*)]. This process also informed the modeling of seasonal variation in the transmission potential and differences in seasonal variation between tropical and temperate regions (all factors that could have important effects on model predictions). This work was the first to evaluate explicitly interventions that involved altering the international aviation network with the aim of slowing the global spread of pandemic influenza (Figure 2). We considered two possible control policies: first, we evaluated a policy that canceled a proportion, p, of all air travel from countries once they had experienced a certain number, q, of influenza cases (where both p and q were varied); second, we considered policies that did not involve canceling flights but that reduced local transmission rates in affected countries. Such interventions could include social distancing measures (such as closing schools and promoting hand hygiene) and antiviral treatment and prophylaxis (*13, 14*).

Comparison with the local epidemic peaks from the 1968–1969 pandemic showed that the model, though relatively simple, was able to capture the timing of the global spread of that pandemic with a high degree of accuracy, although some cities, such as Tokyo (where the epidemic peaked more than a month later than predicted by the model), did show departures from the model that were not consistent with chance effects. This analysis also showed that, with contemporary air travel volumes (2002 data), the timing of the epidemic peaks in 1969 would have been expected to occur somewhat earlier, in some cases (for southern hemisphere cities) shifting to an earlier influenza epidemic season.

Results of the intervention analysis showed that restrictions on air travel from affected cities were likely to have little value in delaying epidemics unless almost all travel ceased almost as soon as epidemics were detected in each city (Figure 3). For example, if 90% of air travel from affected cities were canceled after the first 100 influenza cases, the arrival time of influenza in other cities typically would be delayed by only 2 or 3 weeks. Though these delays showed some sensitivity to the city where the pandemic first emerged and the timing of this event, in no case was the delay achieved close to the 6 months needed to develop and produce a vaccine. Even if 99% of journeys from affected cities could have been stopped, we found the delays in the timing of the epidemic peaks were

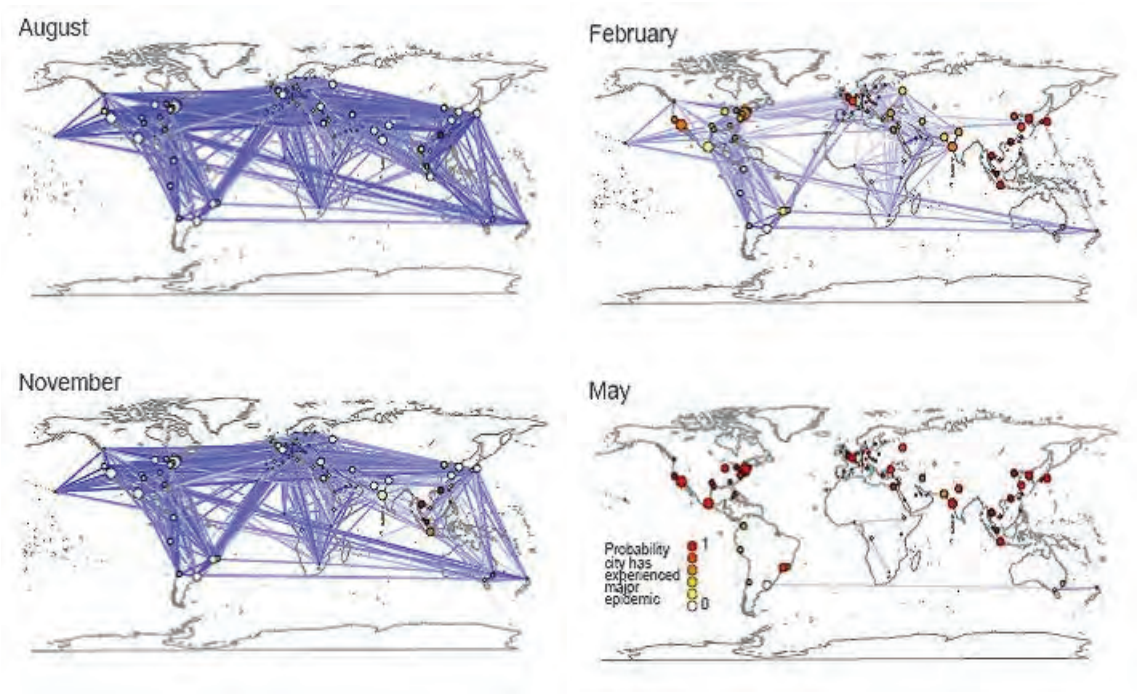


FIGURE 2 Global dissemination of a simulated influenza pandemic originating in Hong Kong at the beginning of June to 105 cities, under the assumption that 99.9% of air travel from affected cities is canceled after the first 100 cases in each affected city (and after 1,000 cases in Hong Kong). City shading indicates the probability that each city has experienced a significant epidemic (based on 100 stochastic simulations). Flights connecting cities are shown as blue lines when there is at least a 5% chance that they have not been suspended due to travel restrictions. [Figure adapted from Cooper et al. (*9*).]

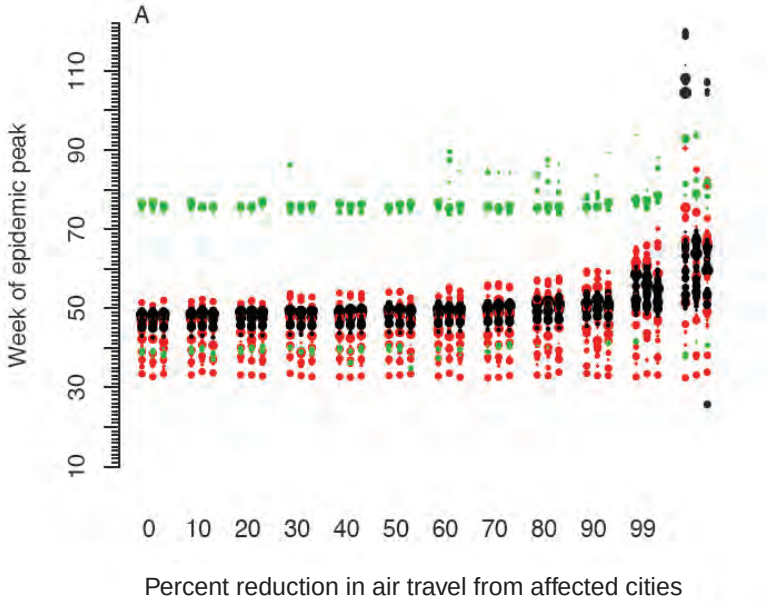


FIGURE 3 Impact of air travel restrictions on timing of epidemic peaks in the 105 cities shown in Figure 2 during a simulated influenza pandemic. Dots show timing of epidemic peaks in individual cities in the northern temperate zone (red), the tropics (black), and the southern temperate zone (green), where the area of each dot is proportional to the population size. Results from three stochastic simulation runs are shown for reductions in air travel between 0% (*far left*) and 99.9% (*far right*).

only 40 to 50 days, too short to have a significant practical benefit. Only if almost all travel from affected cities could be stopped almost as soon as influenza arrived was the intervention able to achieve delays likely to have a significant practical benefit in managing the pandemic. These results are somewhat counterintuitive but can be seen to be a function of the very short generation time of influenza, which results in a rapid initial rate of epidemic growth. If, at the beginning of the epidemic each case infected two other cases after 3 days, we would expect about 10 cases within 10 days of the first case and 100 within 20 days. Thus, even if travel from the city were reduced by a factor of 100 from Day 1, within about 3 weeks there would be the same number of people infected with influenza flying out as there would have been on Day 1 in the absence of any intervention.

In contrast, it was found that interventions to reduce local transmission were likely to be more effective at reducing the rate of global spread and less vulnerable to implementation delays. Nevertheless, under the most plausible scenarios, achievable delays were found to be small compared with the time needed to accumulate substantial vaccine stocks.

Other researchers, working with slightly different sets of assumptions, have reached similar conclusions about the limited role of air travel restrictions in controlling influenza pandemics (if the natural history parameters are similar to those for influenza strains we have seen before), and these results have directly informed both national and WHO recommendations for pandemic responses (*15–17*). While these conclusions have been challenged by a correlation found between a reduction in international travel to and from the United States after the terrorist attacks in September 2001 and the timing of the seasonal influenza peak in the United States the following winter (*18*), the modeling work shows that a direct causal relationship between the relatively modest reductions in air travel that year and the influenza epidemic timing is extremely unlikely (*19*). Notably, the timing of influenza peaks routinely shows considerable year-to-year variation that cannot be explained by changes in the number of international air travelers.

An obvious limitation of modeling studies evaluating the role of the aviation network in the international spread of human pathogens is the failure to account for other modes of travel. However, excluding such travel from global dissemination models will bias model findings in favor of interventions that restrict air travel; by ignoring land and sea travel, the models will overestimate the impact of air travel restrictions on epidemic spread. Thus, the finding that air travel restriction

will have limited value in controlling influenza pandemic spread should be informative to this simplifying assumption. Recently, the metapopulation modeling framework has been extended again to account for "multiscale mobility networks," accounting for both long-distance air travel links and shorter-distance commuting flows, which are an order of magnitude larger (*20*). Results of this analysis have shown that including such commuting flows has little effect on the pattern and rate of global spread of infectious diseases compared with those predicted by air traffic flows alone. The main difference found when including commuting flows in models is increased synchrony of epidemic timing in nearby subpopulations.

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SESSION 2

Practical Case-Response Approaches to Investigating the Spread of Disease in Airports and on Aircraft

Dan Fishbein, *Centers for Disease Control and Prevention* (Presenter)
Hannah L. Kirking, *Centers for Disease Control and Prevention*
Jennifer Cortes, *Centers for Disease Control and Prevention*
Sherry Burrer, *Centers for Disease Control and Prevention and New Hampshire Department of Health and Human Services*
Aron Hall, *Centers for Disease Control and Prevention*
Nicole J. Cohen, *Centers for Disease Control and Prevention*
Harvey Lipman, *Centers for Disease Control and Prevention*
Curi Kim, *Centers for Disease Control and Prevention*
Elizabeth R. Daly, *New Hampshire Department of Health and Human Services*
Itamar Grotto, *Israel Ministry of Health* (Presenter)
Shepherd Roee Singer
Emilia Anis

Norovirus Transmission on Aircraft

Dan Fishbein (Presenter), Hannah L. Kirking, Jennifer Cortes, Sherry Burrer, Aron Hall, Nicole J. Cohen, Harvey Lipman, Curi Kim, and Elizabeth R. Daly

An outbreak of gastroenteritis among members of a tour group on an airplane resulted in an emergency diversion. An investigation was conducted to determine the etiology of the outbreak, assess whether transmission occurred onboard the airplane, and describe risk factors for transmission. Case patients, defined as passengers or crew members with vomiting or diarrhea, were asked to submit stool samples for norovirus laboratory testing. Fifteen (41%) tour group members met the case definition, with most illnesses occurring before or during the flight. Seven (8%) passengers who were not tour group members met the case definition after the flight. Norovirus genogroup II was detected by reverse transcription–polymerase chain reaction (PCR) in stools from case patients in both groups. Multivariate logistic regression analysis showed that sitting in an aisle seat and sitting near any tour group member were associated with developing illness. Transmission of norovirus likely occurred during the flight, despite its short duration.

Swine Flu A/H1N1 Transmission via the Aviation Sector

Itamar Grotto (Presenter), Shepherd Roee Singer, and Emilia Anis

Pandemic influenza A/H1N1 2009 is now well established in all countries. While the northern hemisphere prepares to mitigate the effects of an anticipated "second wave," it is informative to look back at the early stages of the pandemic when containment was still a central strategy. This presentation describes the case of an Israeli traveler returning from Central America with influenza A/H1N1 2009 and considers the implications of in-flight transmission.

The first case of influenza A/H1N1 2009 was diagnosed in Israel on April 24, 2009, in a 26-year-old man who returned that day from Mexico. Israel was the sixth country in the world to confirm a case of the disease.

The first steps taken by the Israeli Ministry of Health were defined as the "containment phase." They included

mainly hospitalization and treating all patients with oseltamivir, adding swine flu to the list of notifiable diseases in Israel, and epidemiologic investigation of each case. The objectives of the investigation were to identify the possible source of infection as well as contact tracing. As for travelers, a special clinic was opened at Israel's only international airport, and travelers from Mexico were examined routinely and asked to stay in voluntary quarantine for 7 days and to go to an emergency room if they developed fever. The Israeli Ministry of Health recommended that people postpone travels to Mexico.

Case A

This case involves a 22-year-old Israeli woman who returned from Mexico through Madrid (May 2, 2009). On a flight from Madrid to Tel Aviv, she had fever, shivers, cough, sore throat, rhinorrhea, weakness, and headache. Upon landing, she did not report to the airport clinic but went directly to an emergency room, where she tested positive for influenza A/H1N1 2009 by using the PCR technique on her nasopharyngeal specimen.

The Ministry of Health control measures included a recommendation to all travelers on Case A's Madrid to Tel Aviv flight to stay at home for 7 days (voluntary quarantine) and to report to an emergency room immediately if they had influenza-like symptoms and fever. The recommendation was publicized in the Israeli media (television, radio, and Internet).

Case B

This case involves a 59-year-old Israeli woman who became ill in Israel on May 4, 2009. She had fever, cough, sneezing, and joint pain. She tested positive for influenza A/H1N1 2009 by PCR on May 5, 2009.

The epidemiologic investigation disclosed that the woman had left Israel traveling to Guatemala via Madrid on April 10, 2009. After touring Guatemala, she flew to Havana, Cuba, on April 22. Her return flight to Israel left Cuba on April 30 and she made a brief stopover in Madrid. After spending 9 h on May 1 in the city of Madrid and at various locations in the Madrid airport, including 90 min in the preflight waiting area, she boarded a 23:30 flight to Israel that arrived in Tel Aviv on the morning of May 2. On the flight from Madrid to Tel Aviv, she sat one row in front of Case A.

Outcome

Both women were hospitalized for 7 days with mild illness, were treated with oseltamivir, and fully recovered. No additional transmission from the two patients was identified (including Case A's boyfriend, who sat next to her during the flight).

Discussion

Case A was symptomatic during the flight and was therefore certainly infectious at that time. Given her close proximity to Case B, and the lack of any other purported sources of contagion, in-flight transmission is viewed as the most likely cause of the infection spreading to Case B. Contagion in Havana or Madrid or in the waiting rooms of the respective airports cannot be ruled out; however, no sustained community transmission was recorded in Cuba or Madrid at the time, and the epidemiologic investigation did not uncover any known contact with potentially infectious individuals in those settings.

Aircraft manufacturers have made great advances in cabin safety, and the risk of transmission of infectious disease aboard aircraft is very low. Cabin air systems in modern aircraft provide about 50% of the air from outside; the remainder is from recirculated air. Airflow is supplied at a rate of 20 to 30 air changes per hour. High-efficiency particulate air filters, similar to those used in hospital operating theatres and intensive care units, capture >99% of bacteria, fungi, and viruses (*1, 2*). However, no ventilation can completely prevent airborne transmission of infectious particles, particularly from passengers sitting in close proximity. Thus, despite the effectiveness of modern filtration systems, airline passengers remain at some risk of direct infection in the cabin as well as in preflight waiting areas and on shuttle buses.

Though rare, tuberculosis transmission has been documented (*3, 4*) and remains a long-standing concern among public health officials. More recently, five flights were associated with probable in-flight transmission of severe acute respiratory syndrome, affecting 37 people (*5, 6*). In-flight transmission of measles has been reported (*7*), as has influenza (*8–10*). However, Han and colleagues demonstrated a lack of airborne transmission during an outbreak of influenza A/H1N1 2009 among tour group members in China (*11*).

Conclusion

Airlines have undertaken a variety of measures over the years to minimize the risk of in-flight transmission of infectious agents. These measures cannot eliminate that risk entirely. Passengers should consult travel experts, ensure that they have completed recommended pretravel immunizations, and inquire about current health

guidelines for travelers. People who are unwell should always consult a doctor before traveling. There is a need for international guidelines to deal with medical and ethical issues related to pretravel screening and restrictions.

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SESSION 3

Theoretical Modeling Approaches to Investigating the Spread of Disease in Airports and on Aircraft

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Zhao Zhang
Byron Jones, *Kansas State University* (Presenter)
Joan B. Rose, *Michigan State University* (Presenter)
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Summarizing Exposure Patterns on Commercial Aircraft

James S. Bennett (Presenter), Jennifer L. Topmiller, Yuanhui Zhang, and Watts L. Dietrich

National Institute of Occupational Safety and Health (NIOSH) research into the aircraft cabin environment began with a request from the FAA to study health effects among aircraft crew. A review of previous studies showed that female flight attendants may be at increased risk of adverse reproductive outcomes (*1*). Exposure assessments and epidemiologic studies in the areas of radiation and cabin air-quality studies followed (*1–3*). Difficulties in conducting studies in the passenger aircraft cabin environment during flight led to the decision that further work be done using realistic cabin mock-ups and computational fluid dynamics (CFD) to understand the behavior of any air contaminants present.

The aircraft cabin environment is maintained during flight by the environmental control system (ECS). It is no small accomplishment to provide a safe atmosphere at cruise altitude—for example, 35,000 ft. In addition to pressurization, the ECS provides clean outside air to the cabin, which has a high-occupancy density compared with, for example, office buildings and classrooms. In newer aircraft, about 50% of the air supplied to the cabin has been recirculated and passed through a high-efficiency particulate air (HEPA) filter, with the remaining supply volume coming from the outside. The ECS is designed, as shown in Figure 1, to use the length of the cabin as a plenum, so that air is supplied and exhausted at a velocity that is constant with respect to the length of the plane. Also, the direction of flow out of the supply and into the exhaust slots is in the seat row direction, perpendicular to the aisle. The movement of air between seat rows is thus minimized in the ECS design concept.

While the airflow coming from the supply outlet can be considered two dimensional, the flow in the open space of the cabin is freer and somewhat turbulent, insofar as it is characterized by fluctuations in velocity (speed and direction). A flow can be deconstructed into its Reynold's averaged velocity components:

$$U(t) = \bar{U} + u(t) \tag{1}$$

where each instantaneous component, $U(t)$, is the sum of a time average and a fluctuation with a time average of

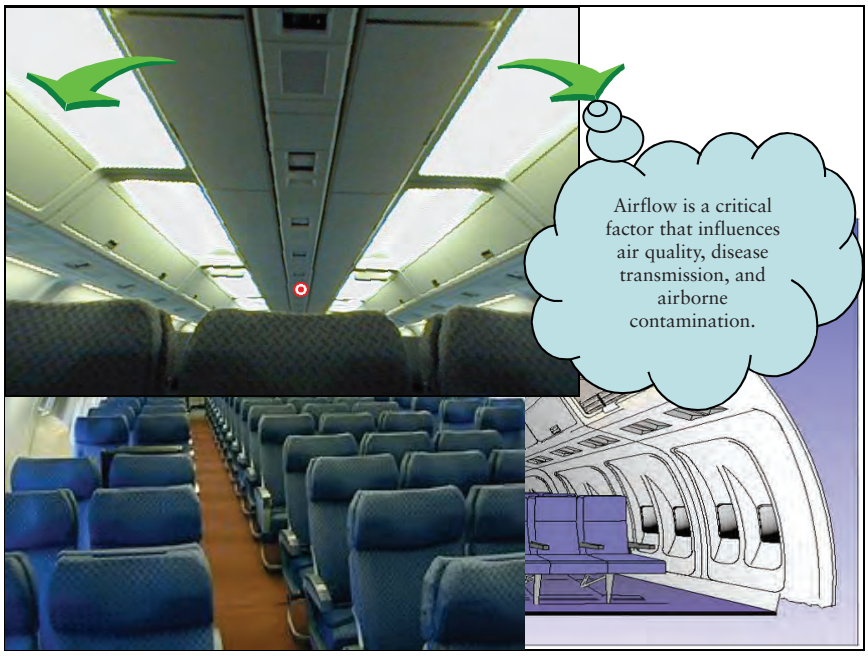


FIGURE 1 Aircraft environmental control system design concept attempts to minimize the movement of air between seat rows.

zero (*4*). Air contaminants, such as small droplets from an exhaled breath or a cough, are transported by the fluctuations, even though the average of the fluctuations is zero. The ECS, then, creates two competing processes, one that is intended and another that is perhaps impossible to avoid: (*a*) removal of potentially contaminated cabin air into the exhaust and replacement with clean air, and (*b*) movement of contaminants within cabin air by flow fluctuations. Fluctuations are present, even in the hypothetical absence of obstructions, moving bodies, and thermal plumes.

Airflow and contaminant transport research has taken place in collaboration with many expert partners (Figure 2). The data generated by collaborations have been flow fields measured by experiments with realistic mock-ups or calculated by using CFD. The flow fields have consisted of velocity, turbulence parameters, and either gas or aerosol contaminant concentration.

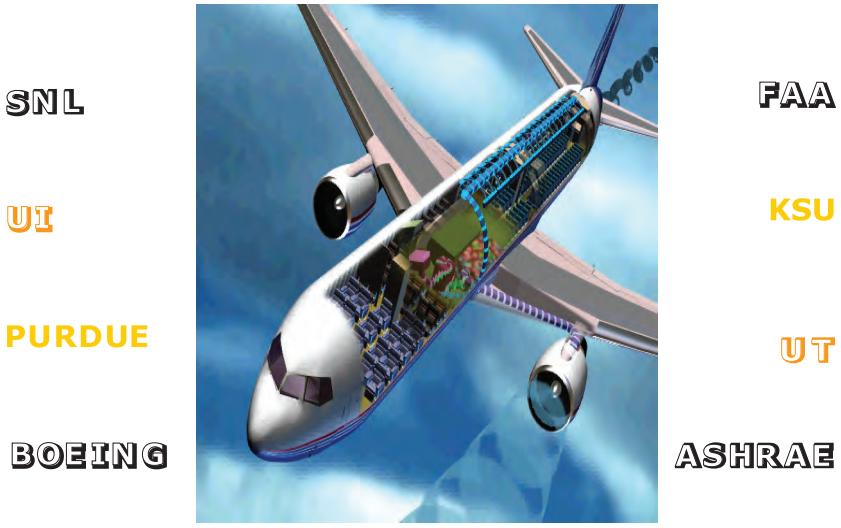


FIGURE 2 Aircraft Air Quality Partners: Sandia National Labs (SNL); University of Illinois (UI); Purdue University; Boeing Commercial Airplanes; Federal Aviation Administration (FAA); Kansas State University (KSU); University of Tennessee (UT); and American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE).

CFD simulations took place in collaboration with Boeing, Inc. (*5, 6*). At the University of Illinois, experiments in a five-row B767 mock-up delivered volumetric particle tracking velocimetry images of cabin flow seeded with helium bubbles and tracer gas (carbon dioxide) concentration fields generated by three source locations and three ventilation rates (*7–9*). Sandia National Labs provided a massively parallel computing platform for the Boeing–NIOSH CFD simulations, including large eddy simulation. Figure 3 provides snapshots of the Illinois, Boeing, and Sandia efforts. Sandia also provided advice and evaluation of the cabin airflow research and suggested that tracer gas experiments would be useful. Data for a real Boeing 747, including velocity and turbulence fields, were gathered by the University of Tennessee, at the FAA Aero-medical Research Institute. They also created detailed CFD simulations of the fluctuating cabin flow. NIOSH provided a review of the University of Tennessee report to the FAA.

Kansas State University (KSU) was a pioneer in aircraft cabin research. KSU, along with Purdue University, has continued to advance the field in part through the FAA Center-of-Excellence for Aircraft Cabin Environmental Research. KSU has a Boeing 767 mock-up with many seat rows and Purdue has done large-scale CFD simulations, including the wake effect of a moving body. Some collaborators, including KSU and Purdue, and NIOSH researchers were involved in research projects sponsored by the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) and development of an ASHRAE standard for aircraft cabin ventilation.

Much work has been done, yet the role of ventilation in controlling disease transmission in aircraft cabins remains opaque. There is consensus that the issue is complex because of the many variables involved. Figure 4 diagrams possible modes of transmission and variables discussed during the symposium.

In an effort to pull immediately useful information from the detailed, high-quality studies done to date, a simple model and a modeling framework are presented here. The general aircraft-cabin air-contaminant transport effect (GAATE) model seeks to build exposure–spatial relationships between contaminant sources and receptors, quantify the uncertainty, and provide a platform for incorporating future studies. To put this model in context,

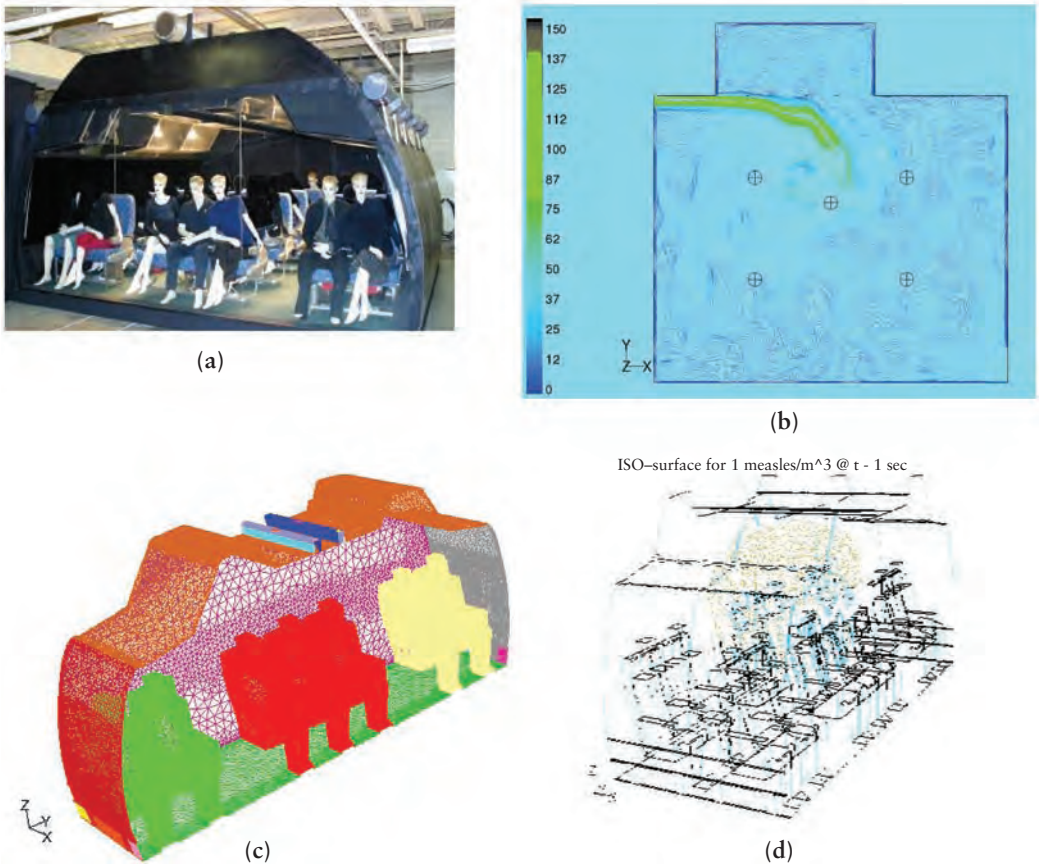


FIGURE 3 (*a*) Boeing 767 mock-up at the University of Illinois; (*b*) large eddy simulation CFD model of a velocity field conducted by Boeing, NIOSH, and Sandia; (*c*) unstructured mesh for a Reynolds-Averaged Navier–Stokes (RANS) CFD model of a Boeing 767, conducted by Boeing; and (*d*) time evolution of an aerosol cloud from a point source, using a RANS CFD model of a Boeing 767.

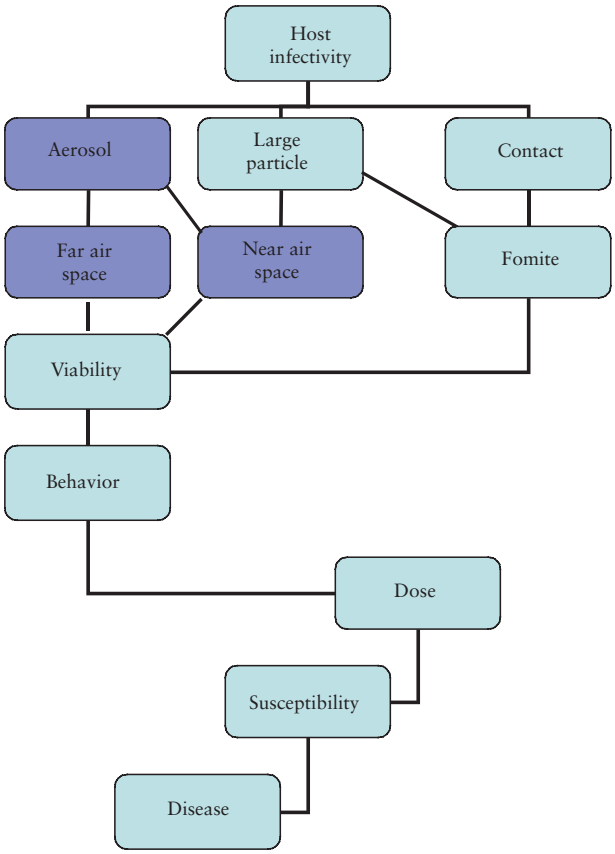


FIGURE 4 Aircraft cabin air quality research (blue highlight) in the context of disease pathways discussed at the symposium.

of the many variables presented in Figure 4, the GAATE model involves only the three variables indicated by blue boxes. Thus, it provides exposure information.

Knowledge of the infection risk to flight crews and passengers is needed to form a coherent response to an unfolding epidemic. An essential part of infection risk is exposure, and exposure may have an airborne component. The infection of flight attendants on Air China and Singapore Airlines with severe acute respiratory syndrome (SARS) in 2003 is evidence of the risk faced by these workers, who in some situations find themselves in the role of first responders. Moreover, the Association of Flight Attendants asked the FAA for protection from SARS. The goal of the GAATE model, then, is to provide useful information to authorities for addressing exposure incidents involving SARS, avian flu, H1N1, and other potentially lethal agents and to provide guidance to emergency response personnel.

Methods

The GAATE model can be thought of as a metamodel—that is, a model built from other models or studies. As such, the first step is solicitation of contaminant transport data for aircraft cabin environments from research partners. These data sets must be placed on a common footing and normalized to remove meaningless sources of variability. The large metadata set thus formed is amenable to statistical analysis. The model chosen currently is regression analysis, where the dependent variable is concentration gradient and the independent variable(s) describes location within the cabin.

Variables that must be normalized are mass emission rate of the source and air change rate of the cabin. Put another way, the ratio of these two terms is held constant. In the current study, this normalization was achieved by dividing the measured concentration at a given seat location by a reference concentration

$$C_{\mathrm{REF}} = \frac{C_{\mathrm{AVE}} + C_S}{2} \tag{2}$$

where C_{AVE} is the spatial average concentration over all measurement locations and C_S is the concentration measured nearest the source. As the cabin air is not well mixed, the inclusion of C_S helps to make C_{REF} more representative. The concentration variable used in the anal-

yses is then the ratio of the measured concentration to the reference concentration, C_{MEAS}/C_{REF},

$$C = \frac{C_{MEAS}}{C_{REF}} \tag{3}$$

Thus far, the GAATE model has been applied to a data set from the University of Illinois. Measurements of carbon dioxide as a tracer gas were taken in a five-row Boeing 767 mock-up. Data were generated over three air change rates and three source locations, in which the measured outcome was the concentration at each of 35 seat locations. The concentrations measured at 2-s intervals were time-averaged over 1,000 s after the system had stabilized. No exhaust air was recirculated, and the gaspers were off. These data sets reflect an isothermal scenario. A CFD simulation was performed for the same set of conditions. These results were not included in the GAATE model, because they did not fit the same regression equation as the experiments, which were considered more reliable. In principle, data generated by CFD are reasonable candidates.

The regression equation had the following general form:

$$Y_i = \beta_0 + \beta_1 X_i + \varepsilon_i \tag{4}$$

where

- Y_i = observed quantity (contaminant or pathogen concentration);
- β_0 and β_1 = y intercept and slope of regression line, respectively;
- X_i = independent random variable; and
- ε_i = residual for the ith observation.

Various functional forms were chosen to attempt a fit to the data by inspecting a plot of concentration versus distance from the source for all three source locations. Distinguishing between the seat letter coordinate direction and the row number coordinate direction did not provide a better fit than using the simple variable of distance, r.

Results

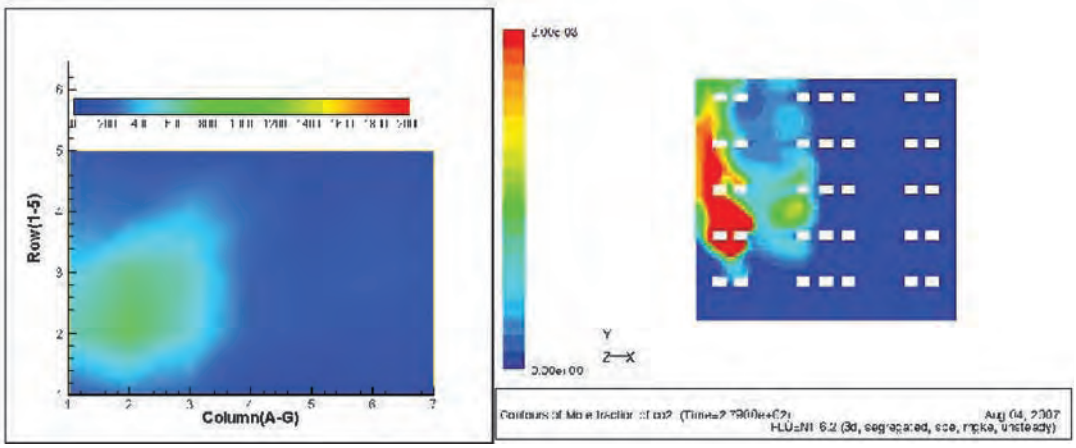
Figure 5 shows the contaminant dispersion pattern at time T for both the experiment and the simulation. The concentration pattern in the experiment resembles isotropic diffusion, while in the simulation the pattern is formed more by directional convection.

The specific form of Equation 4 that provided the best fit to the experimental tracer gas data was

$$C = \beta_0 + \beta_1 \ln\left(\frac{1}{r}\right) \tag{5}$$

The regression line shown in Figure 6 has an intercept, β_0, of 1.055 and a slope, β_1, of 0.493. With an R^2 value of 0.476, it can be said that 47.6% of the variability in the concentration data is explained by the regression model. While the regression passed the normality test (P = .141), it failed the constant variance test, which is not surprising given that the concentration is more variable near the source.

The analysis carries an uncertainty of 95%. This uncertainty applies in two different ways. β_0 and β_1 both have 95% confidence intervals ($0.9906 \leq \beta_0 \leq 1.1194$ and $0.4204 \leq \beta_1 \leq 0.5660$), and these intervals are not independent, which is why the blue confidence bands in



(a) (b)

FIGURE 5 Time slice of contaminant dispersion, source location, 2B: (*a*) measured and (*b*) simulated.

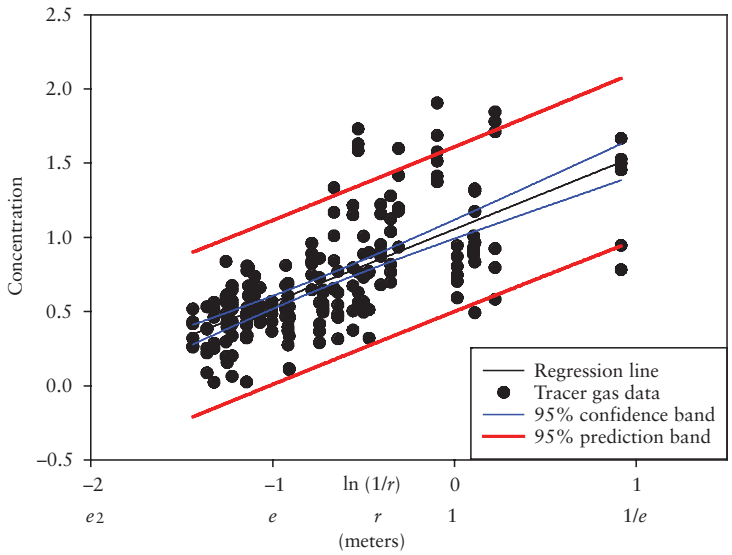


FIGURE 6 Regression analysis of (source distance, concentration) data pairs, with 95% confidence and prediction bands.

Figure 6 are curved. The red bands indicate uncertainty in prediction of the relation between C and $\ln(1/r)$ for any member of the population of r values. Put another way, the confidence band addresses the question of whether this regression line is the best one possible, while the prediction band addresses the value of this regression line as a predictive model.

Because the concentration variability is greater nearer the source, a two-segment linear regression (Figure 7) was also done to see if the fit could be improved. Both the slopes of the two lines and the breakpoint between them, r = 2.48 m, were determined in the regression. Thus, a physicality—the near-zone–far-zone distinction was identified by the statistical analysis. The freedom to adjust for this phenomenon increased the R^2 value from 0.476 to 0.502, only a small improvement. Here also, the analysis passed the normality test (P = .375) but failed the constant variance test. The near source behavior is perhaps not well described by any kind of model based on the isotropic assumption. However, performing the regression on only the far-field data— >2.48 m from the source—actually lowered the R^2 value. The benefit of more data points was apparently greater than the cost of the increased variance.

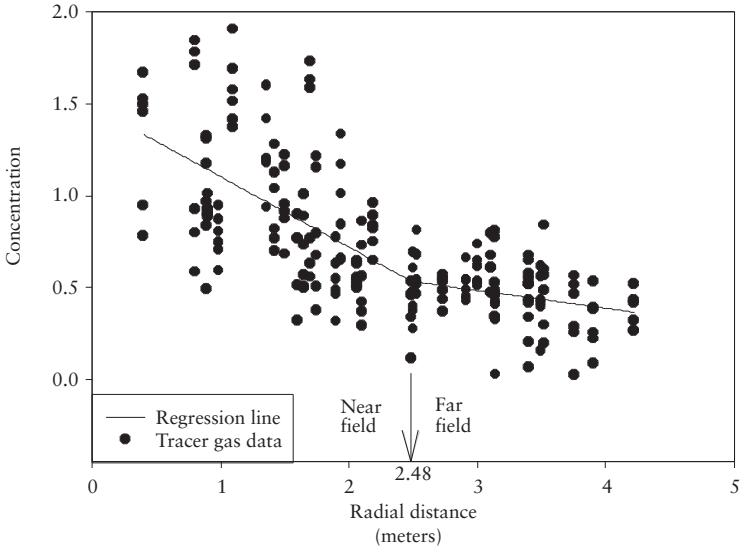


FIGURE 7 Two-segment regression, with breakpoint between near and far fields.

Discussion

Once a concentration–space relation is established, it can be applied in useful ways. With half the variability being explained by distance from the source, estimation using this simple model is widely applicable in the cabin environment, although the predictive power has quantifiable limitations. An interactive graphic tool was built using the idea that the relative exposure, taken here as the time average of normalized concentration, can be estimated for a source located anywhere in the Boeing 767 coach section. Figure 8 shows this idea actualized with a Visual Basic program. By clicking on any seat in the cabin diagram, the exposure is calculated for the rest of the 10-row field. The figure is an example of the resultant field from one source location.

An exposure map can be used to refine assumptions made about how far air contaminants such as small droplets travel in the cabin. Also, a case history and an exposure map may be used together to gauge infectivity by the airborne route. Moreover, if infectivity and exposure are both known, decisions about which passengers authorities should follow up with after a known exposure to a reportable disease are obvious.

Conclusion

The ability of the GAATE model to make a contribution in such situations depends on its predictive power. Improvements in accuracy may come from inclusion of additional data sets. The scalability inherent in this approach paves the way to study additional aircraft types. Exposure to small droplets and postevaporation nuclei, even at a source distance of several rows, is readily apparent. The airborne pathway should then be considered part of the matrix of possible disease transmission modes in aircraft cabins, unless the pathogen has been proven nonviable in air.

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

Advance Models for Predicting Contaminants and Infectious Disease Virus Transport in the Airliner Cabin Environment (Part 1)

Qingyan (Yan) Chen (Presenter), Sagnik Mazumdar, Michael W. Plesniak, Stephane Poussou, Paul E. Sojka, Tengfei Zhang, and Zhao Zhang

In 2003, SARS affected more than 8,000 patients and caused 774 deaths in 26 countries across five continents within months after its emergence in rural China (*10*). A more recent disease, H1N1 A flu, affected about 40,000 patients across 76 countries within 1.5 months after its

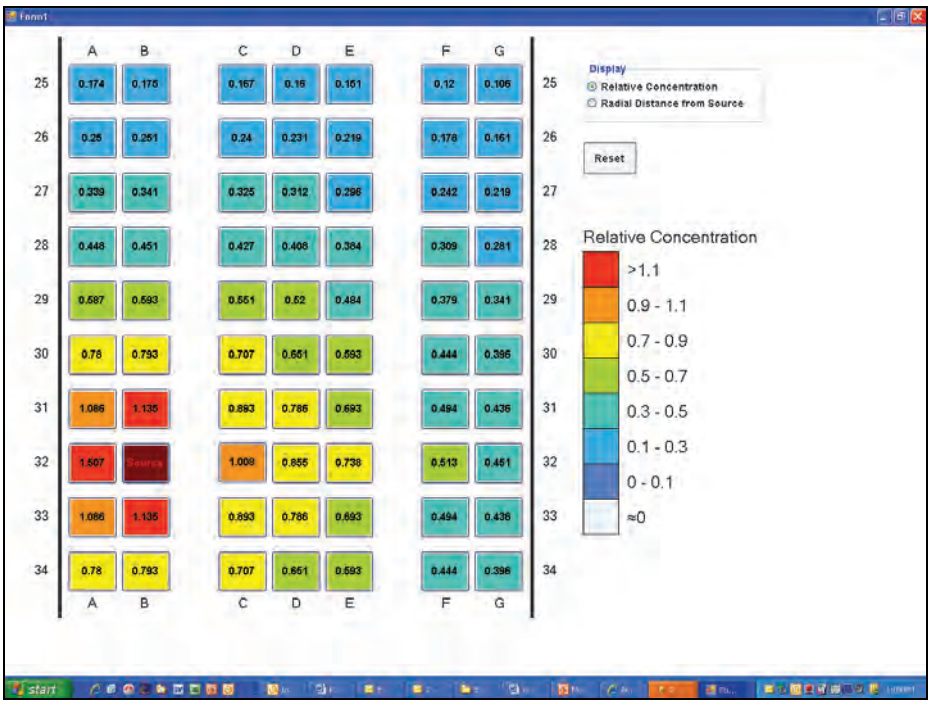


FIGURE 8 Example of use of the GAATE model interactive graphic: relative exposure to an air contaminant from a source in Seat 32B.

emergence (www.who.int/csr/disease/swineflu/updates/en/index.html). These cases illustrate the dramatic role of globalization and air travel in the dissemination of an emerging infectious disease. Other cases of airborne infectious diseases transmitted in airliners in recent years include tuberculosis, influenza, measles, and mumps.

CFD is a very attractive tool to study the transmission of airborne contaminants in an airliner cabin as it is inexpensive and flexible in changing thermofluid conditions inside the cabins compared with experimental measurements. The results presented here illustrate the potential of using CFD in modeling gaseous and particulate contaminant transport inside airliner cabins. CFD was also used to model the SARS transmission case in Air China Flight 112 from Hong Kong to Beijing in 2003 where a contagious passenger infected some 20 fellow passengers, as shown in Figure 9 (*11*). Some seated as far as seven rows from the contagious passenger were infected. The movement of passengers and crew members may play a role in transmission.

CFD Modeling

The commercial CFD software Fluent 6.2. (www.fluent.com) was used for the studies. The CFD model used a second-order upwind scheme and the SIMPLE algorithm. The renormalization group k-ε model was used to simulate the turbulent flow inside the cabin mock-ups.

Two different cabin geometries were used in this investigation to understand the effects of moving crew and passengers on contaminant transmission inside airliner cabins. Initial CFD studies were done with a section of a four-row, twin-aisle cabin model as shown in Figure 10*a*. The cabin section had 28 seats in four rows, representing a section of economy-class cabin. The cabin was fully occupied. The air entered through linear diffusers at the ceiling level and was exhausted through outlets placed in the side walls close to the floor. The airflow rate in the cabin was 10 L/s per passenger. Box-shaped manikins were used to represent passengers. A moving person was modeled as a rectangular box of height 1.7 m and was assumed to move along the aisle. To investigate the effects of a moving person on contaminant transport in the cabin, two scenarios were considered: one in which the person walked continuously from the front to the rear end of the cabin without stopping and the other with intermittent stops of 5 s at each row.

A second case used a 15-row, single-aisle cabin for studying SARS transmission in the flight from Hong Kong to Beijing in 2003 for Row 4 to 18 as shown in Figure 9. Figure 10*b* shows only one row of the cabin and the remaining rows are identical. The air entered the cabin through four linear diffusers: two placed at the ceiling above the aisle injected air downward and the other two at the side walls located below the storage bins injected air inward to the aisle. The total supply airflow rate of 10 L/s per passenger was distributed equally among the four inlets. The air was exhausted through outlets on the side walls close to the floor. The contagious passenger sat in Row 11 of the 15-row cabin. Two contaminant release scenarios were considered: one with a pulsed release for 30 s and the other with a continuous release. The body moved along the aisle from the rear end of the cabin and stopped seven rows in front of the contagious passenger.

The movement was simulated by using a combination of static and dynamic meshing schemes. For example, the computational domain of the four-row twin-aisle airliner cabin was modeled using two separate geometries: a section for the aisle with the moving body and the other section for the rest of the cabin, as shown in Figure 11. The meshes for the first section were dynamic; the remaining meshes were static. Hence, only 3.7% of the total meshes inside the domain were dynamic, which can reduce the computing costs for remeshing. The movement inside the 15-row, single-aisle model for the SARS transmission case was modeled similarly.

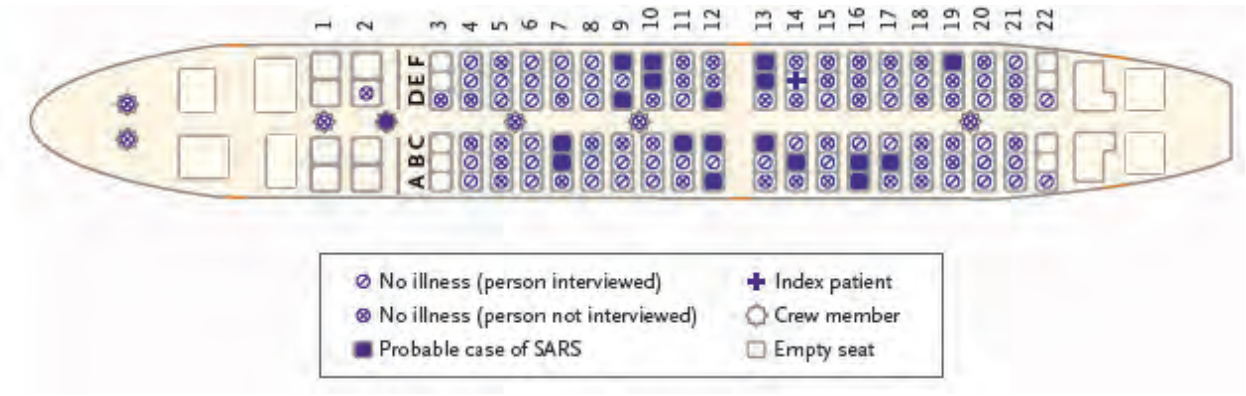


FIGURE 9 A contagious passenger with SARS virus infected some 20 passengers on the flight from Hong Kong to Beijing in 2003 (*11*).

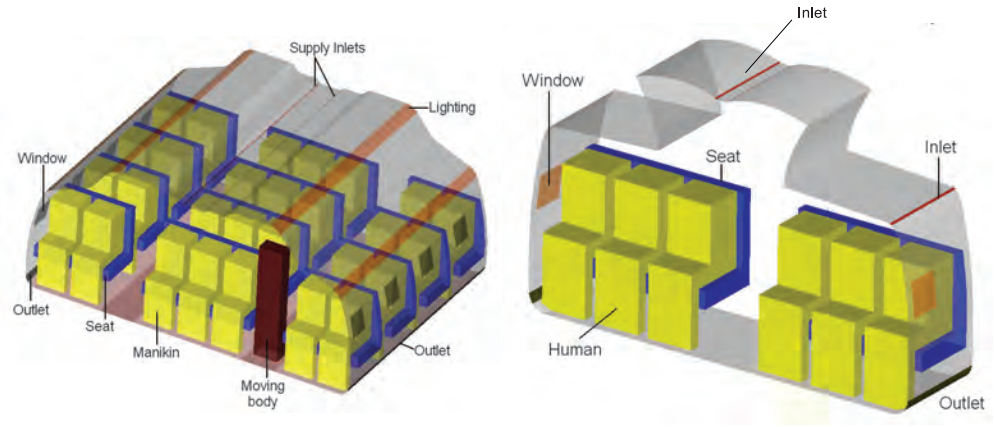


FIGURE 10 Two different cabins used in the study: (*a*) section of four-row, twin-aisle cabin, and (*b*) one-row model of the 15-row, single-aisle cabin.

CFD Modeling Results

Figure 12 shows the airflow pattern and airborne contaminant concentration at 1 m above the cabin floor as the body moved continuously from the front to the rear end of the cabin. The results were for a contaminant released from Passenger 2A seated in the right window seat on the second row. The results at t = 0 s show the initial steady-state air velocity and contaminant distribution before the body started moving. The airflow patterns illustrate that the flow disturbance created by the moving person was rather local. The impact of movement on airflow on the left half of the cabin was minimal. The moving body created a low pressure zone behind it and hence air was induced from the sides. The moving body also pushed the air at its front. Hence, the body could carry the contaminant behind to the rear of the cabin.

Figure 13 shows the effect of an intermittently moving body for the same contaminant source. The body stopped for 5 s in each row—that is, it stopped from 0.7 to 5.7 s in Row 2 and from 6.6 to 11.6 s in Row 3, which simulated a moving crew member who stopped at each row to provide service. The airflow pattern and contaminant concentration at 1 m above the cabin floor are shown at t = 0.7, 5.7, 6.6, and 11.6 s in the figure. The area near the contaminant source became heavily contaminated when the moving person stopped at Row 2, because it broke the near symmetric flow vortices at the cross section that aided in formation of the high-contaminant-concentration zone.

The intermittently moving body also enhanced the contaminant concentration level to passengers sitting near the aisle when it stopped at Row 3. When the moving person stopped, the highly contaminated air it carried at its back was pushed to the sides. Hence, the contaminant concentration can be higher than that with a continuously moving person.

The results from the four-row, twin-aisle cabin show a significant impact of a moving person on contaminant transport. Thus, this investigation used the method to study why the SARS virus could be transported as far as seven rows away in the Air China 117 flight from Hong Kong to Beijing in 2003. Figure 14 shows the contaminant distribution at the breathing level in the Air China cabin for a pulse contaminant release from the infected passenger, such as a cough. The high-concentration zone

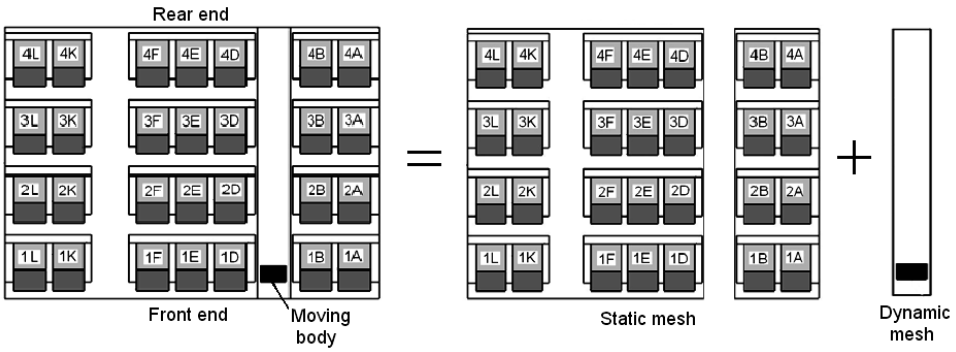


FIGURE 11 Mesh layout of the four-row, twin-aisle cabin section.

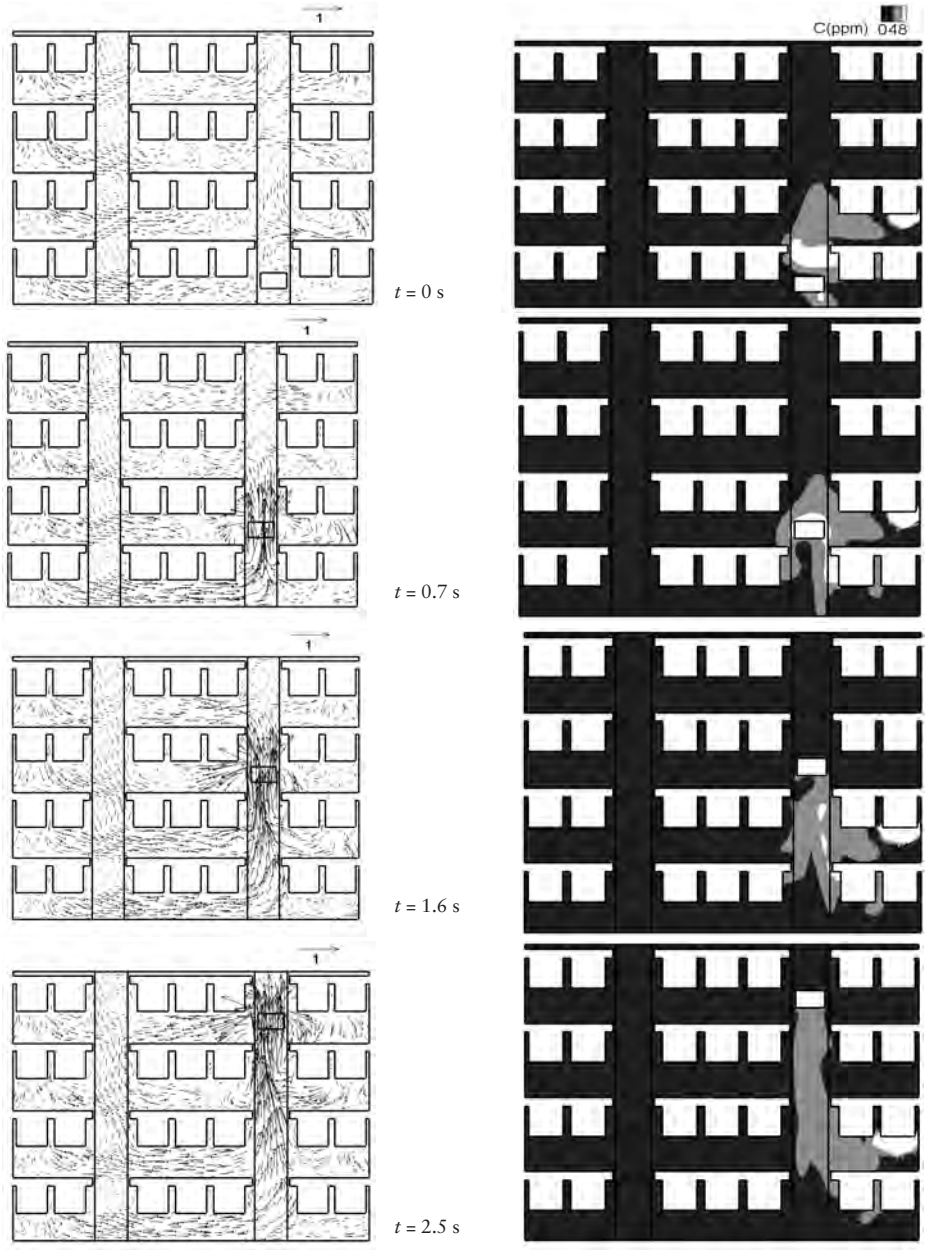


FIGURE 12 Velocity distribution and contaminant transport trends from the movement of a person in the four-row, twin-aisle cabin mock-up.

was initially within two rows of the infected passenger, which appears to be in good agreement with common sense because the flow in the longitudinal direction should be small. When a person moved along the aisle, the wake could carry the contaminant to seven rows in front of the infected passenger, where the body stopped its movement. The contaminant carried in the wake was then distributed to the passengers seated near the aisle. A similar phenomenon was observed for the scenario with a continuous contaminant release. The CFD results showed that body movement may have caused the transmission of SARS pathogen from the infected passenger to fellow passengers seated as far as seven rows away on the Air China flight from Hong Kong to Beijing in 2003.

Thus, CFD modeling appears to be a powerful and effective tool for predicting airborne contaminant transport in airliner cabins. Because CFD models use approximations, the predictions should always be validated with high-quality experimental data.

CFD Model Validation

It is expensive and time-consuming to conduct experimental measurements of airborne contaminant concen-

FIGURE 13 Velocity distribution and contaminant transport trends from an intermittently moving person in the four-row, twin-aisle cabin mock-up.

trations in a full-scale airliner cabin with passengers. Hence, this study used a 1/10th-scaled, water-based experimental test facility consisting of an upside-down cabin mockup as shown in Figure 15*a*. The cabin was made by a transparent semicircular pipe 45 cm in diameter and 2.44 m long. The mock-up, fully submerged in a water tank, was equivalent to a cabin with 28 rows of economy-class seats. The interior of the modeled cabin was empty so no seats and passengers were modeled. To simulate the ECS, water was injected through an overhead duct of the inlet diffuser assembly. To achieve a uniform inflow in the cabin, the water entered a settling chamber through 23 pipe fittings and was then supplied to the cabin through 48 elongated openings cut along the length, where a T-shaped diffuser diverted the fluid laterally to both sides of the cabin cross section. Water was extracted from two outlets located near the side walls of the cabin at floor level. To simulate a moving person, an automated mechanism placed above the experimental facility traversed the moving body (0.02 m thick × 0.05 m wide × 0.17 m tall) along the longitudinal direction of the cabin. Particle image velocimetry (PIV) was used to measure the velocity distribution inside the water tank. The camera and laser were positioned to capture cross-sectional and longitudinal flow images. The corresponding CFD model

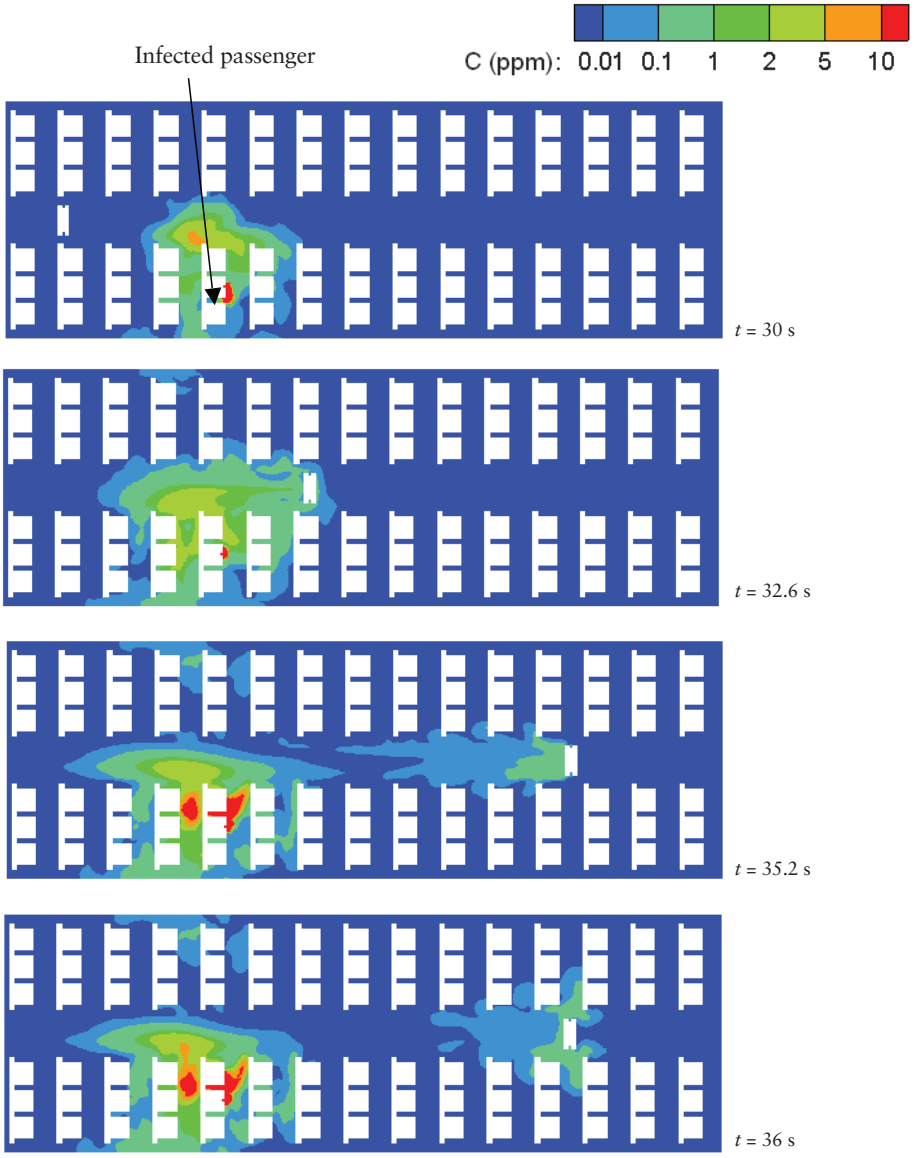
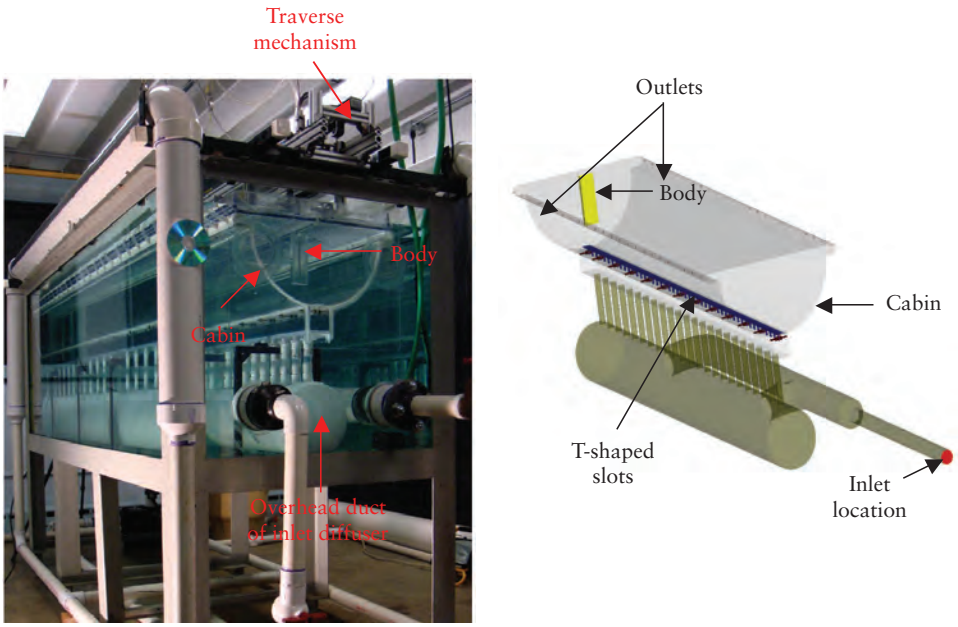


FIGURE 14 Contaminant transport process from a person's movement along the aisle with a pulse release of contaminant from the infected passenger in the single-aisle SARS transmission cabin mock-up.

was built for the water model as shown in Figure 15*b*. The model was constructed to simulate as close to the experimental model as possible. Thus, the inlet started at the water supplying pipe to eliminate the difficulties in specifying inlet conditions in the cabin.

Figure 16*a* shows the measured mean flow fields at Frames 4 and 7, which were acquired when the body moved 8.25 and 15.5 cm, respectively, past the laser sheet. A strong downwash in the wake of the moving body was observed, which is produced by the two symmetric eddies around the top corners. As the two eddies approached the cabin floor, they spread to the sides and dissipated. The disturbance created by the moving body diminished very rapidly after this process. Figure 16*b* shows the corresponding computed flow fields. Side-by-side comparison indicates that the CFD model was able to qualitatively predict the development of the two eddies. The predicted core size, flow pattern, and structure are in reasonable agreement with the experimental values, although noticeable differences exist with respect to vortex aspect ratio.

Figure 17*a* shows only a small area of the measured flow due to the limited image size captured by the PIV. The comparison between the measured and computed velocity in the midsection along the longitudinal direction in Figure 17 shows reasonable agreement between the two results. Flow recirculation due to flow separation could be observed from the results. However, the



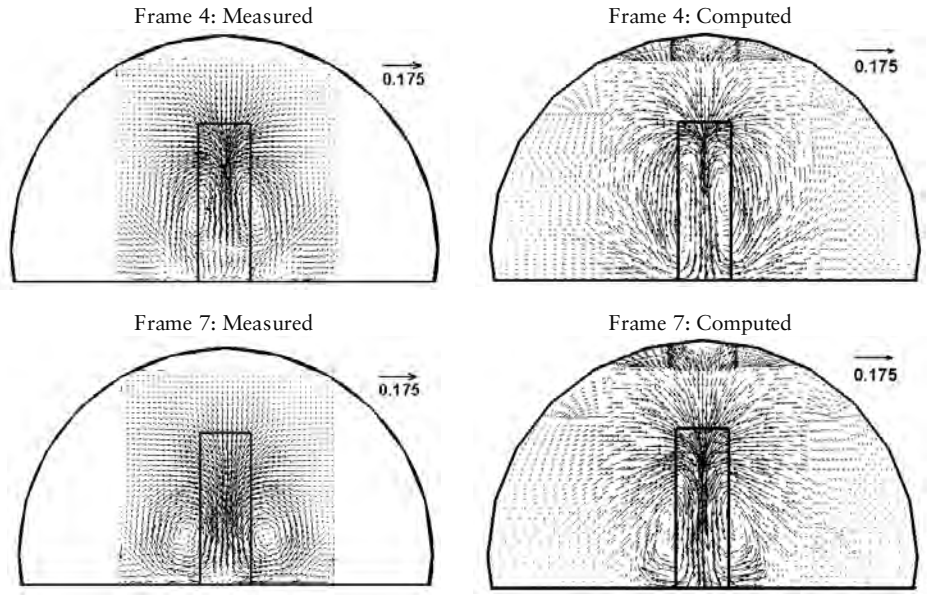
(a) (b)

FIGURE 15 (*a*) **Small-scale experimental test facility of the cabin mock-up, and** (*b*) **CFD model of the test facility.**

longitudinal flow computed behind the moving body is much stronger than that measured, with overprediction of longitudinal momentum transfer. This result may be due to less momentum transfer in lateral directions, resulting in vertically elongated eddy rings in the cabin cross section. Overall, the CFD model can capture the fundamental flow mechanisms found in such a simulated cabin.

Conclusions

CFD, a powerful tool for predicting the transport of airborne contaminants in airliner cabins, shows that the movement of a person could have a significant effect. The movement of a person may have resulted in the spread of SARS virus to passengers seated far from the contagious passenger on Air China Flight 112 from Hong Kong to



(a) (b)

FIGURE 16 (*a*) **Measured and** (*b*) **computed mean flow fields at Frames 4 and 7 from movement inside the small-scale cabin mock-up.**

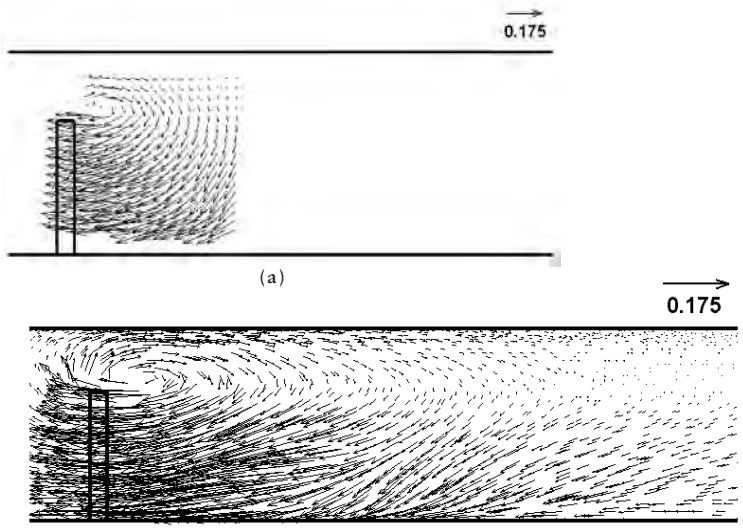


FIGURE 17 Velocity in the midsection along the longitudinal direction: (*a*) measurement and (*b*) computations.

Beijing in 2003. CFD results should always be validated with high-quality experimental data, as CFD models use many approximations. By using the measured velocity fields obtained from a small-scale, water cabin mock-up, CFD modeling can capture the fundamental flow features, although discrepancies exist between the measured and computed results.

Acknowledgments

This study was funded by the FAA Office of Aerospace Medicine through the National Air Transportation Center of Excellence for Research in the Intermodal Transport Environment under a cooperative agreement.

Although the FAA sponsored this project, it neither endorses nor rejects the findings of this research. This information is presented in the interest of invoking technical community comment on the results and conclusions of the research.

Advance Models for Predicting Contaminants and Infectious Disease Virus Transport in the Airliner Cabin Environment (Part 2)

Byron Jones (Presenter)

The results from three aircraft cabin contaminant dispersion studies are presented. These studies address dispersion of gaseous contaminants, solid particles, and bacteria in an aerosolized liquid. All the studies were conducted in an aircraft cabin mock-up. Each study was conducted with somewhat different goals. However, all the studies are intended to support the development and validation of mathematical and computational models of dispersion in aircraft cabins. An attempt is made here to compare the data from the three studies.

Cabin Mock-Up

The aircraft cabin mock-up facility (Figure 18) used in these studies is located in the Aircraft Cabin Environment Research Laboratory at KSU. It is based on the geometry of a Boeing 767 but is intended to represent a midsize wide-body aircraft in general. The mock-up cabin is 9.45 m (31 ft.) long with 11 rows of seats. The seat spacing is 825 mm (32.5 in.) per row and the seats are seven across in a 2-3-2 configuration. The air inlet diffusers are from a Boeing 767 aircraft as is the air distribution system that supplies the diffusers. The air supply design for this aircraft consists of two linear slot diffusers extending the length of the cabin near the center ceiling of the cabin, each blowing outward. The inlet airflow is uniform along the length of the cabin. The uniformity of this airflow was experimentally verified for both sides. Air exits the cabin through continuous floor-level exhausts on both sides of the cabin. The mock-up is equipped with coach seats from a Boeing 767 aircraft, and each seat is occupied by a thermal manikin with a heat output of 75 W. The manikins do not breathe or perspire. All inlet air is conditioned and passes through HEPA filters before entering the cabin. There is no recirculation. The total airflow rate to the cabin was 660 L/s (1,400 ft^3/min) for all data presented.

FIGURE 18 Aircraft cabin mock-up.

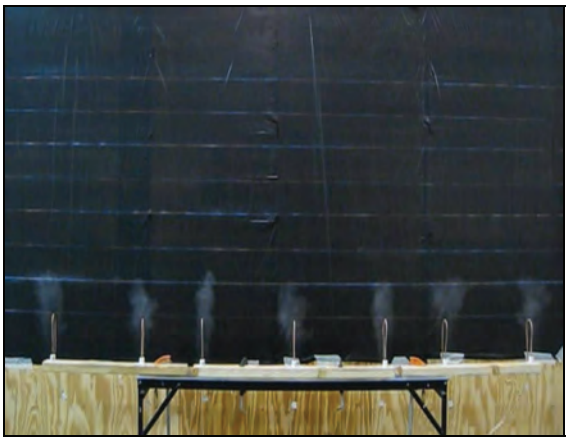


FIGURE 19 Solid particle injection.

Description of Experiments

The first set of experiments used carbon dioxide (CO_2) tracer gas to measure contaminant dispersion. The CO_2 tracer gas was mixed with helium to generate a mixture with a molecular weight equal to that of air. The tracer gas was at the same temperature as the cabin air when injected. As CO_2 is much denser than air, negative plume buoyancy gives distorted results if these measures are not taken to ensure neutral buoyancy. Calculations and experimental results show that turbulent diffusion is several orders of magnitude greater than molecular diffusion, so the molecular diffusion is expected to be a negligible consideration in these experiments. The tracer gas was injected continuously at low velocity through a vertical tube in the center of the right or left aisle at a height of 1.2 m (48 in.) as shown in Figure 18. The air was sampled through a seven-port sample tree that can be seen near the front of the cabin in Figure 18. All measurements reported are at a height of 1.5 m (60 in.). Air was sampled from one port at a time for a minimum of 30 min before proceeding to the next port. Once all ports were sampled, the entire tree was moved to the next location.

The second set of measurements used talcum powder as a representative solid particle contaminant. The peak number density for this powder occurred at approximately 1.5 μm and the data presented are for the total particle numbers between 0.5 and 5.0 μm. Injecting solid particles in a controlled manner without disrupting the cabin airflow is difficult. To accomplish this feat, a "puff generator" was developed. A measured amount of talcum powder was placed in a small cup. A small copper tube connected to a source of pressurized air was directed downward at the cup. The airflow was turned on and off quickly with a solenoid valve to generate a short but intense puff of air that aerosolized the talcum powder without generating a large airflow. Figure 19 shows seven of the devices being tested simultaneously. For the experiments, the injection occurred in Row 2 and was done simultaneously at all seven seats in the row. Particle concentration was measured with a TSI 3321 aerodynamic particle sizer (APS) with the instrument placed in the seat as shown in Figure 20. A straight tube was used to collect air samples at a height of 1.18 m (46.5 in.). Before the talcum powder was injected, the APSs were monitored to verify that the air was free of particles and the count rate was near zero. Data were then collected for 15 min after injection, at which time the counts had returned to near zero. The data reported here are the 15-min sums.

The third set of measurements used aerosolized *Lactococcus lactis* as a surrogate bacteria. The bacteria were aerosolized by using a handheld mister (Figure 21*a*) and the mist was released around head height of the seated "passengers." Collection plates were located on top of the seat backs as shown in Figure 21*b*. The collection plates were opened for 30 min for collection after the *L. lactis* was released. Additionally, air samples were taken at selected locations. The data presented here include only the collection plates. Controls were also run with no bacteria aerosolized to verify that near-zero counts

FIGURE 20 Solid particle measurements.

(a) (b)

FIGURE 21 (*a*) **Release of bacteria and** (*b*) **collection of bacteria.**

were obtained and thus all counts measured could be attributed to the aerosolized *L. lactis*.

Longitudinal Dispersion

These three sets of experiments were conducted with different objectives, and now an attempt is made to compare the results from all three studies. Figure 22 shows the seat and row numbers used to identify measurement locations.

For the tracer gas measurements, the tracer gas was injected at Row 6 and measurements were made along the entire centerline. For the solid particle measurements, the particles were injected at Row 2. One APS was located in Seat 3D for all experiments and was used as a reference. A second APS was placed, in turn, in each of the D seats for Rows 4 to 11. For the bacteria measurements, the aerosolized bacteria were sprayed along the front of the cabin, generally in the Row 1 area. Measurements were taken at each seat but, for the purpose of this presentation, only the data for the three center seats are reported.

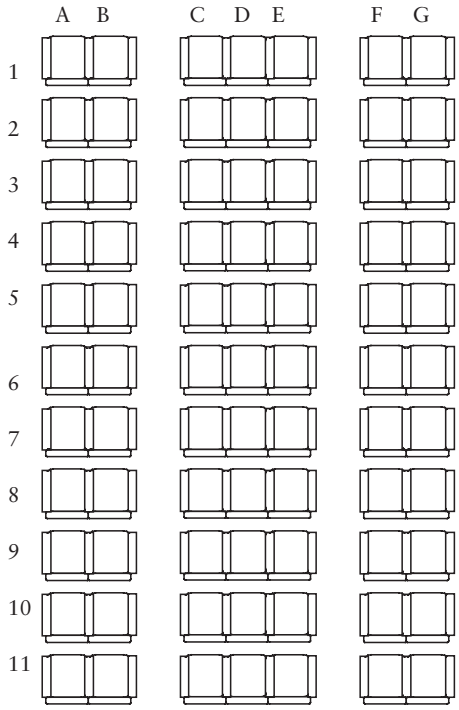


FIGURE 22 Row and seat identification for cabin mock-up.

Figure 23 presents the tracer gas data. The data were deduced as follows:

$$C_n = (c_m - c_o)/(V_t/V_v)$$

where

C_n = normalized concentration at a location (nondimensional);
c_m = measured concentration at a location [parts per million (ppm)];
c_o = concentration in the ventilation air supplied to the cabin (ppm);
V_t = volumetric flow of the tracer gas, CO_2 only (L/s, ft^3/min); and
V_v = volume flow rate of ventilation air (L/s, ft^3/min).

Data were collected at 178-mm (7-in.) intervals but are grouped by row for ease of comparison with the particle and bacteria data. The results are asymmetric, with the tracer gas in the right aisle tending to go rearward and the tracer gas in the left aisle tending to go forward. A clear drop-off with distance along the centerline is observed in both cases.

Figure 24 presents the particle measurement results. Each data point represents a separate experiment, and for each data point shown the total number of counts at that location was divided by the total number of counts at the reference APS in Seat 3D. Thus, the value at Row 3 is automatically 1 but is not shown. The drop-off with distance is similar to what was observed with the tracer gas.

Figure 25 shows the bacteria measurements results. Here the measurements for all three center seats for the row are averaged. The data are normalized based on the

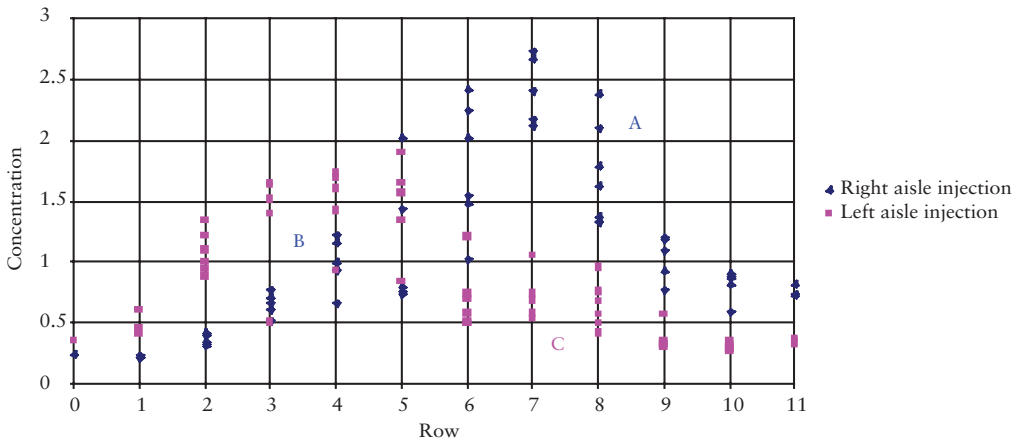


FIGURE 23 Tracer gas longitudinal dispersion data.

Row 3 data, similarly to the particle data. Again the drop-off with distance is similar to the tracer gas and particles.

Figure 26 presents all three sets of data on the same graph. The tracer gas data were divided into three groups identified as A, B, and C in Figure 23. Groups A and B (right aisle) were normalized by dividing by the average value at Row 7 and Group C was normalized by dividing by the average value at Row 5. Rows 5 and 7 then, effectively, became the equivalent of Row 3 for the particle and bacteria data sets and the drop-off with distance is plotted accordingly in Figure 26. Although there is quite a bit of scatter, especially for the tracer gas results, quantitatively similar trends are observed for all three data sets. Bacteria values appear to start high but drop off more rapidly with distance than the particles and tracer gas. The particles may drop off more quickly than the tracer gas.

Lateral Dispersion

The injection for the tracer gas and for the solid particles is the same as for the longitudinal dispersion. Tracer gas measurements were made from side to side for Rows 5 to 9. For the particles, measurements were made only for Rows 4 and 7. For the bacteria, releases were made at Seats 6B, 6D, and 6F and measurements were collected at all seats. Because of the differences in the experimental setup for each study, it is not possible to directly compare lateral dispersion results for the three data sets as was done for longitudinal dispersion. The results for each are presented in turn.

Figures 27 and 28 present the tracer gas results. The peak concentration is offset from the injection location in the lateral and longitudinal directions. The rearward shift for the right aisle injection and the forward shift for the left aisle injection are evident. There is a clear drop-off with distance across the aircraft at a given row that is similar to what was observed in the longitudinal direction but no direct comparison is made.

Figure 29 presents the solid particle results. As the injection was uniform across Row 2, this experiment does not give a direct measure of lateral dispersion. The distribution at Row 4 is very nonuniform. Much of the lateral nonuniformity has disappeared by the time the particles get back to Row 7, which should not be surprising.

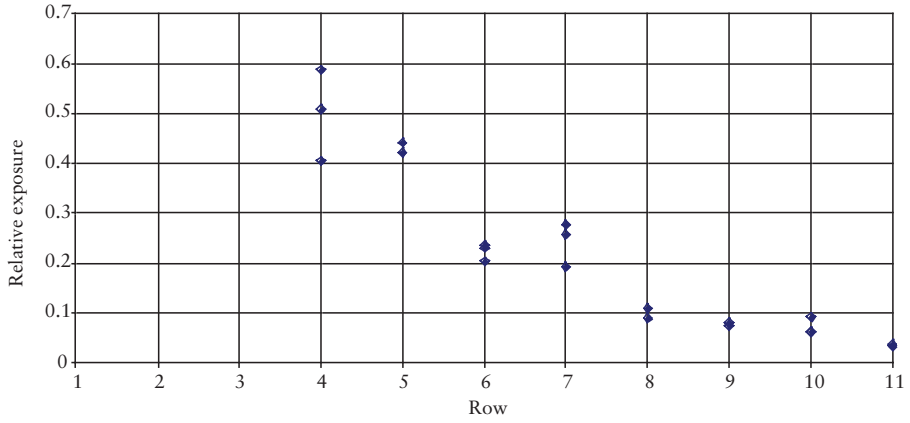


FIGURE 24 Solid particle longitudinal dispersion data (Row 3 level normalized to 1).

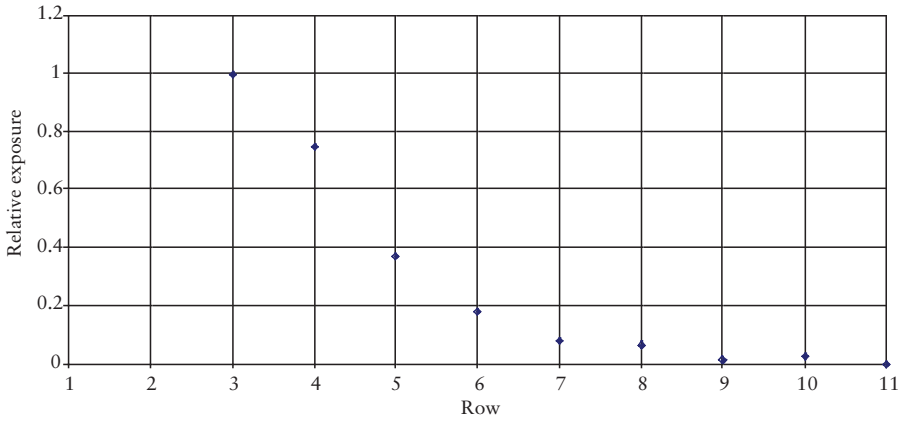


FIGURE 25 Bacteria longitudinal data (referenced to Row 3; average of Seats C, D, and E).

Figures 30, 31, and 32 present the bacteria results for the three different release locations. Counts above 400 are considered off scale for this method and are indicated as 400, as shown in Rows 4 and 5 in Figures 30 and 31 and in Rows 3, 4, and 5 in Figure 32. Although there are some local peculiarities (e.g., Row 3 for the Row 6D release), in general the dispersion patterns are pretty much as expected for the Row 6B and 6F releases, and the drop-off in counts across the cabin is clear and reasonably consistent in the region of the release. There was a tendency for forward movement at all three release locations. It is far more pronounced for the left-side release than for the right-side release, which is consistent with the right–left differences found with the tracer gas.

Discussion and Conclusions

It was observed, particularly with the longitudinal data, that the various forms of contaminants behave similarly with respect to dispersion. The relative bacteria concentrations appear to drop off more quickly with distance than those for the tracer gas and solid particles. There are at least two potential explanations. First, the bacteria may have a limited life span when airborne. Only viable bacteria are counted. Thus, in addition to being removed by ventilation as they disperse through the cabin, some of them may become nonviable before they reach the more distant parts of the cabin. It is also possible the collection plates preferentially collect larger droplets as they are oriented vertically and would catch falling droplets. The larger droplets may settle out of the airflow before they reach the more distant parts of the cabin. Nevertheless, these data combined give a reliable quantification of the far-field dispersion of contaminants and provide a basis for developing or validating dispersion models. The far field may be thought of as the region that is more than about two rows or seats in any direction from the point of release.

The data also give some insight into the behavior in the near field (two seats or fewer from the point of

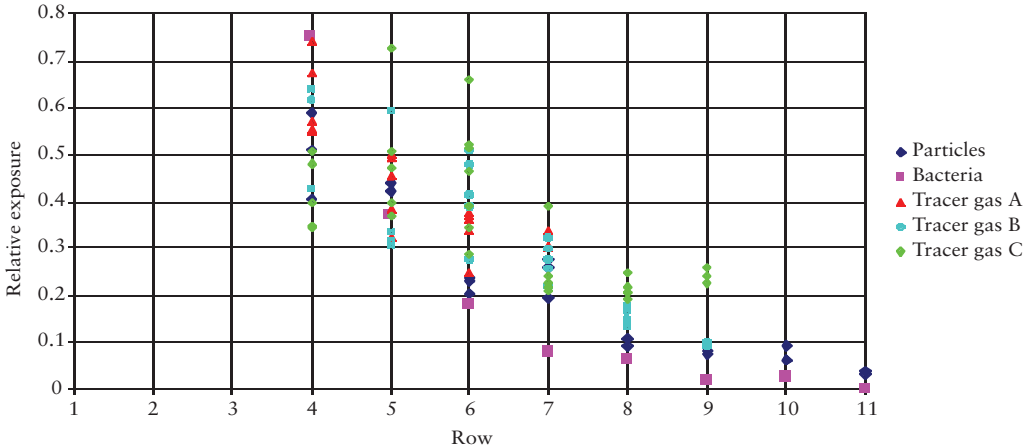


FIGURE 26 All longitudinal dispersion data combined (centerline dispersion).

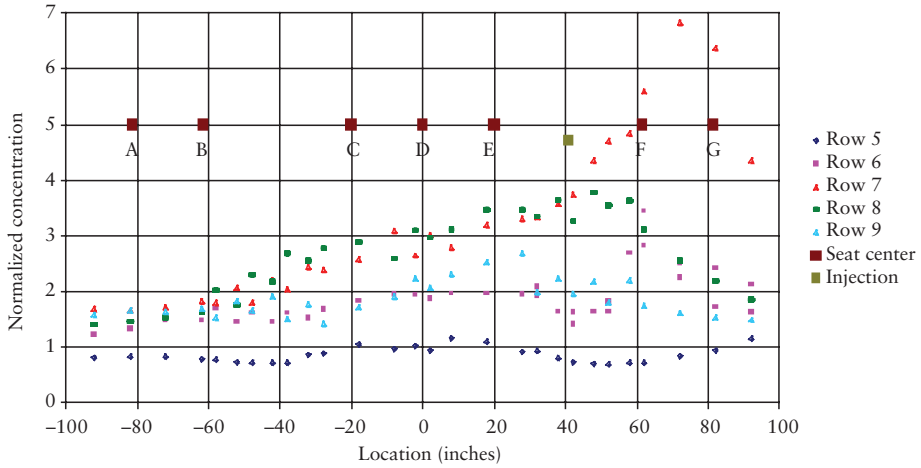


FIGURE 27 Tracer gas lateral dispersion data, right-aisle injection (Row 6).

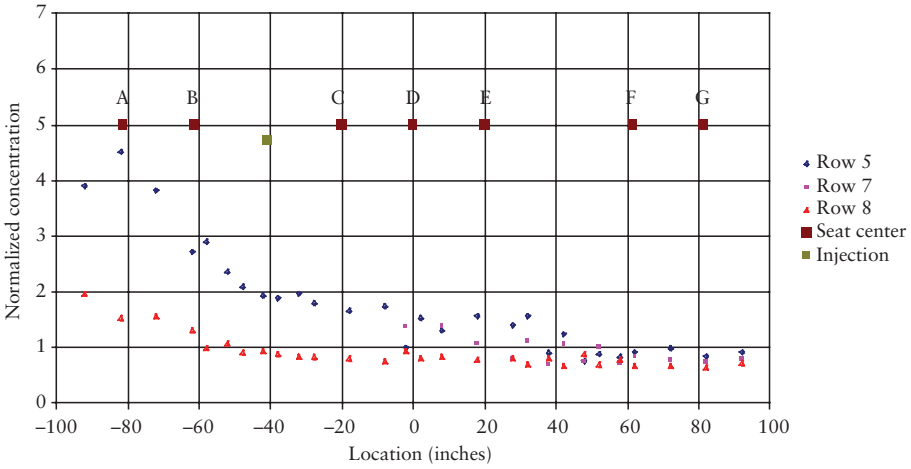


FIGURE 28 Tracer gas lateral dispersion data, left-aisle injection (Row 6).

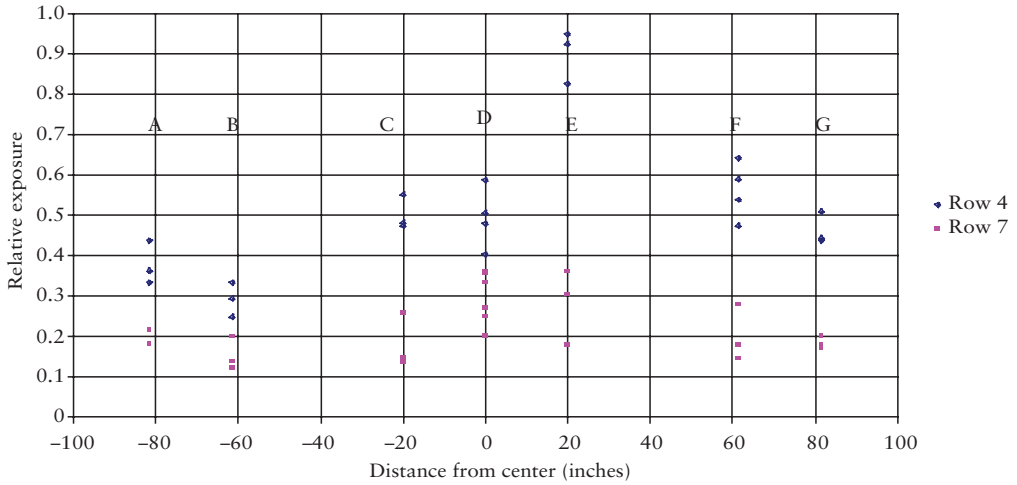


FIGURE 29 Solid particle lateral dispersion data (normalized to Seat 3D).

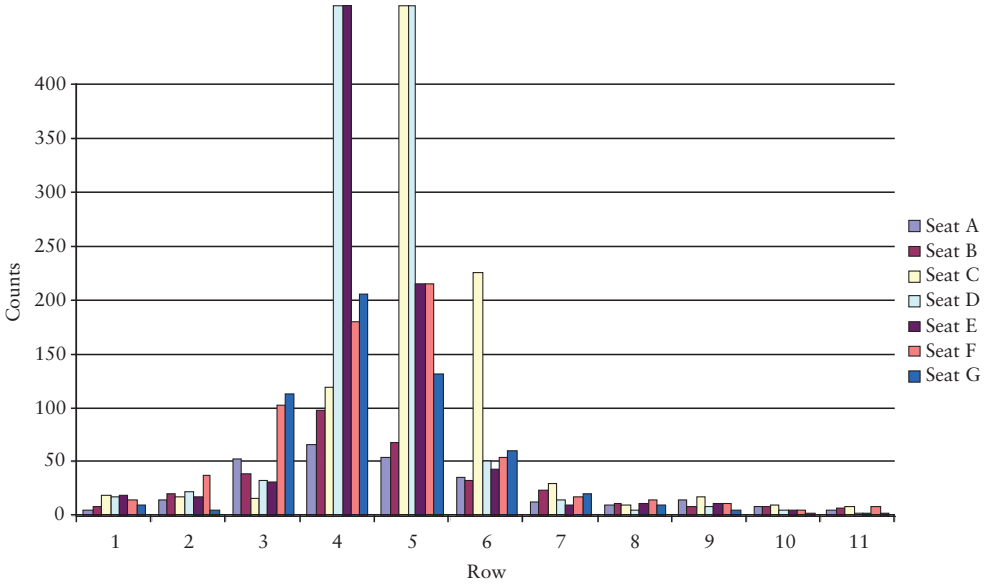


FIGURE 30 Bacteria lateral dispersion data, Seat 6D release.

release). In this region, the dispersion is dominated by local airflow patterns and concentrations are dominated by plumes of high concentration from the source or plumes of low concentration from the supply air. Evidence of large, three-dimensional flow structures is evident in all three data sets. Also, there is evidence that these structures are chaotic. For example, the tracer gas data in Figure 23 have poor repeatability in the vicinity of the injection, but they have good repeatability at locations well removed from the injection. This chaotic nature makes it difficult to model and predict concentrations in the near-field region.

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- Helmut Hirt, assistant professor of biology;
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- Francis Noonan, laboratory manager; and
- P. J. A. Priyadarshana, postdoctoral researcher.

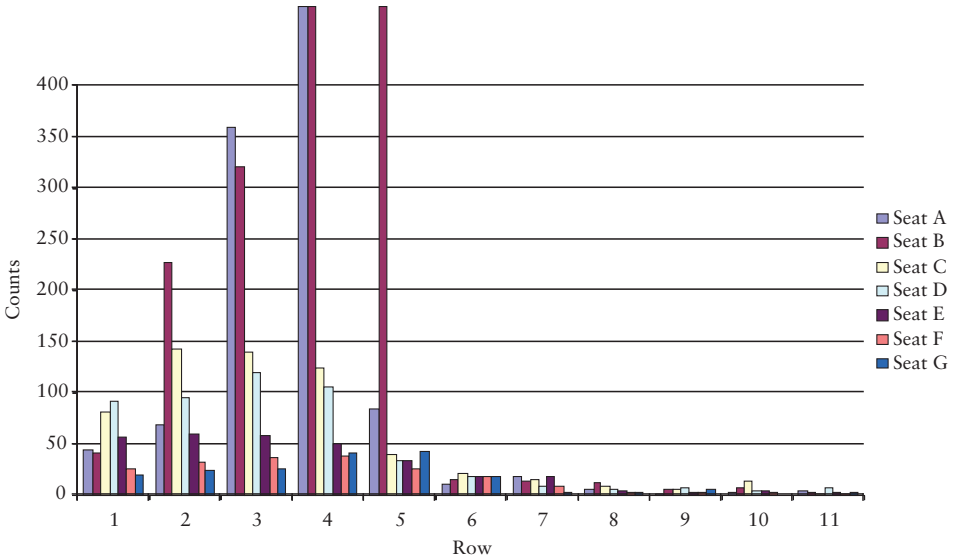

FIGURE 31 Bacteria lateral dispersion data, Seat 6B release.

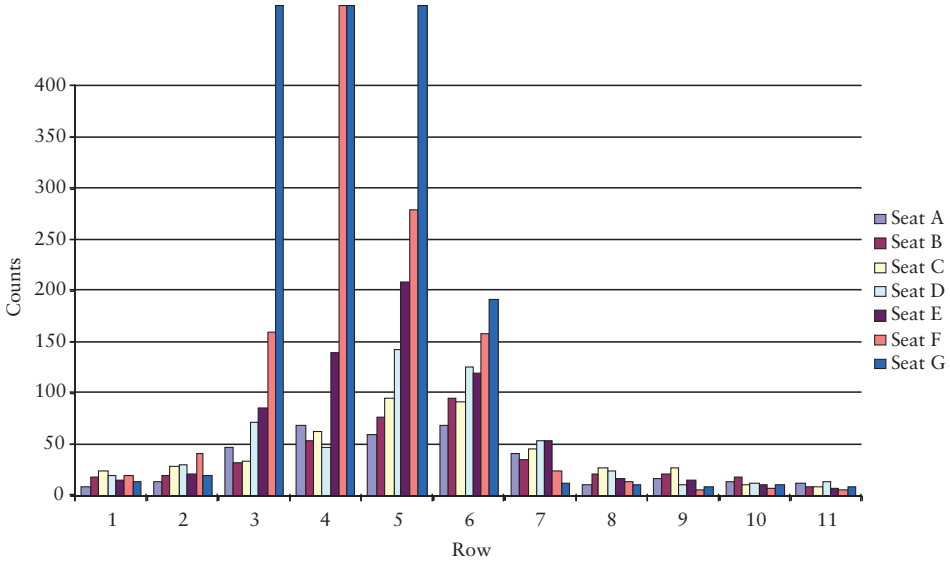

FIGURE 32 Bacteria lateral dispersion data, Seat 6F release.

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Although the FAA sponsored this project, it neither endorses nor rejects the findings of this research. This information is presented in the interest of invoking technical community comment on the results and conclusions of the research.

Characterizing the Risk of Tuberculosis Infection in Commercial Aircraft by Using Quantitative Microbial Risk Assessment

Joan B. Rose (Presenter) and Mark H. Weir

On May 12, 2007, a man infected with multidrug-resistant *Mycobacterium tuberculosis* (XDR-TB) traveled from Atlanta, Georgia, to Paris, France, and then from Prague, Czech Republic, to Montreal, Canada, on May 24, 2007. These flights lasted about 8 h each. The Centers for Disease Control and Prevention (CDC) attempted to address the risk of infection for the approximately 80 people who sat in the five rows surrounding the infected man during the flights. TB transmission to nearby passengers during a flight to Hawaii in 1994 had been previously reported (*12*); it has been acknowledged by the CDC that this risk is low (no estimate of how low has been given), but the consequence of infections could be high because of the rare, drug-resistant type of TB the patient had.

The combination of this individual's travel, global transmission of SARS, and now the potential for transmission via various new types of influenza strains (bird and A/H1N1) has reignited concern about the likelihood of disease transmission in commercial aircraft and the scientific uncertainties in addressing the risk. Several issues and questions are associated with transmission of disease during air travel:

1. Widespread geographic movement of infected individuals to new communities,
2. Spread of disease to fellow passengers during the flight,
3. The types of pathogens primarily associated with disease transmission on airplanes,
4. Understanding the level of risk associated with air flight, and
5. Implementing sound and effective policies to prevent disease transmission during air travel.

Quantitative microbial risk assessment (QMRA) is an approach that can be used to address these issues. QMRA as an integrated science is expanding and following a National Academies' approach (*13*) (Figure 33). It is now possible to address the hazards and model exposures and, via a dose–response relationship, characterize the risk to a greater confidence level than was previously possible (*14*).

The bounds of transmission for specific hazards have been in place, but the traditional understanding of transmission must be reexamined. Respiratory pathogens are transmitted by coughing or sneezing and enteric

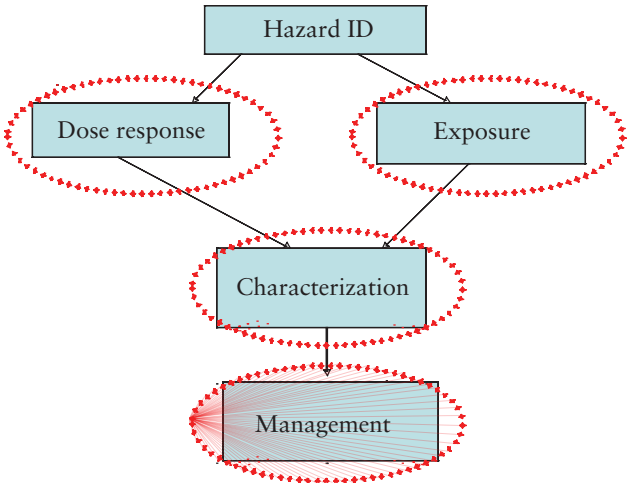

FIGURE 33 Microbial risk assessment framework.

pathogens are transmitted by the fecal–oral route, but it is important to acknowledge that these pathogens may also be transmitted via fomites and contaminated hands. In addition, the role of the contaminated environment needs to be further explored.

Infectious disease hazards enter an airplane environment through three major sources:

1. Infected individuals,
2. Contaminated food, and
3 Contaminated water.

It is known, through at least one small study that investigated sewage from airplanes, that infected passengers are traveling (*15*). This study found enteric viruses in 45% of the sewage on international flights.

Water on airplanes remains a potential source of pathogen risk. In the United States alone, there are 63 air carriers and 7,327 aircraft public water systems that will need to be monitored and potentially treated. The use of bottled water has reduced the risk from aircraft water systems, but safety depends then on the efficiency of bottling facilities and water safety programs in place in various countries. Food-borne disease onboard aircraft is being addressed through hazard analysis and critical control point programs for companies that prepare airline food. However, infected passengers, particularly those who are still excreting pathogens but may not have any major symptoms, are difficult to assess and control.

QMRA for TB Transmission on Airplanes

Jones and colleagues used the QMRA approach to address the TB scenario; a number of research needs and knowledge gaps needed to be addressed (*16*). The assumption of equal risk throughout the airliner cabin has been debunked; the transport was shown to be modeled by using Markov chains rather than the more complex and time-consuming CFD. This situation also showed the power of having a known dose–response model, which can give a risk level specific to XDR-TB. These advancements have allowed for a more complete picture of the risks to passengers and cabin crew members. Generally, risks ranged from 1/10,000 to 1/100,000 on flights in the United States but over an 8-h flight risk, estimates were 0.104 infection per 169 susceptible passengers, which is equivalent to 6.2 infections per 10,000 exposed susceptible persons.

The main drivers of the risk were shown to be coughing rate and active infection and numbers of bacilli.

Current understanding of the excretion of pathogens needs to be expanded, whereby much of the quantitative information typically has been gathered from peer-reviewed articles from the 1950s and 1960s. Further advancements in molecular methods may allow for more accurate determination of excretion rates. This information is very important to the risk assessment, as the level of pathogens to which a person is exposed depends on the amount of pathogen excreted from the infected individual. These levels can vary based on the medium being measured, such as sputum compared with saliva. Therefore, advancements in determining the level of secretion should include all fluids, which would be a concern for the exposure route, such as sputum, saliva, and coughing and sneezing overall for the respiratory route.

The current model, which has been used to model airflow indoors, is simplistic in important areas; even the more sophisticated CFD simulations keep all the people in the cabin stationary. No movement is allowed in the simulations, which can alter the results of the final destination of the pathogens. Also, most of the models that attempt to model the transport of pathogens and particulate matter in airliner cabins are based on indoor air models. Yet airliner cabins more closely resemble confined spaces than the traditional indoor air environments such as office building rooms and other rooms in buildings. A greater concern is the movement of cabin crew and passengers. This oversight can be critical to understanding the true risks posed by an infected passenger due to air current movements, which will be shifted from the baseline (which would be no one moving about in the cabin).

The other concern with modeling the indoor air environment is the viability question. Current indoor air models and those designed specifically for the interior cabin do not allow for including whether the pathogen is viable. This question is important, especially if the transport model is going to assist in designing sampling strategies. The viability question does not likely have a straightforward answer. This problem may go beyond the natural environmental survival of the pathogen and may be a function of the transport and means by

which the pathogen has been introduced to the indoor environment. Therefore, the viability questions should be answered by considering the scenario as well as the location where the transport will take place. This situation will allow for better overall risk estimates as well as risk-based surface sampling strategies after release of the pathogen.

The final research need recognized from this scenario surrounded the surface sampling and decontamination plans. These two concerns are connected. A primary concern is decontamination of the interior of an airliner. Ideally, the decontaminant would not pose a risk to damaging the electronics or structure of the airliner and cabin. Some decontaminants, such as chlorine dioxide, are aggressive oxidizers, which makes them good disinfectants but also damages multiple surfaces; residuals are not possible due to the human health risk as well as the chemistry of the disinfectant. In the case of TB, even XDR attachment of the pathogen to fomites is not a major concern as hand-to-self transmission is not known to occur. However, this issue is greater for other pathogens such as norovirus and methicillin-resistant *Staphylococcus aureus*.

The latter issue leads to the issue of surface sampling, which raises concerns about the target that should be monitored for (indicators versus pathogens) in addition to methods and detection limits. Sampling strategies should be established to determine whether the decontamination scheme has worked, the level of pathogens remaining is acceptable, and the risk posed is acceptable. Sampling is typically done by swabbing surfaces and using a rapid detection method such as a molecular tool. These methods are specific and rapid but would have to be tailored to the environment and the pathogen of interest.

Research to Inform Risk for the Airline Industry

There are two major research programs that would assist in building an improved understanding of the risks of disease transmission on aircraft.

- Characterization of hazards associated with traveling on airplanes: Surveillance of sewage, water, and key fomites (touched and nontouched) for contamination (using *Escherichia coli* and pathogen-specific quantitative polymerase chain reaction) on airplanes would allow one to examine quantitatively the numbers of passengers infected (sewage assessment) and evaluate water and surfaces addressing the key exposure pathways. This method would assist in developing adequate monitoring policies (of people, food, water, and surfaces) and disinfection.
- Assessment of risk and integration of air transport models with QMRA: While there are sophisticated particle—and in some cases microbial—transport models being developed in aircraft, they cannot adequately address risk as hazard-specific survival and dose–response have not been integrated with the partial assessment of exposure. Use of a QMRA framework would allow one to examine quantitative risks with a yardstick that would put the disease transmission during air travel into perspective (1/10,000, which has been deemed acceptable for drinking water).

Acknowledgments

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SESSION 4

Experimental "Bench Science" Approaches to Investigating the Spread of Disease in Airports and on Aircraft

James J. McDevitt, *Harvard School of Public Health*
Donald K. Milton, *University of Maryland School of Public Health*
Charles P. Gerba, *University of Arizona*

Interventions for Preventing the Transmission of Influenza Virus

James J. McDevitt and Donald K. Milton

In light of the 2009 H1N1 flu pandemic and threats of pandemic from highly virulent H5N1 avian flu, much attention has focused on influenza virus. However, according to a 2007 National Academy of Sciences report, *Preparing for an Influenza Pandemic: Personal Protective Equipment for Healthcare Workers*, our understanding of the transmission of influenza is woefully inadequate (*1*). It is important to elucidate the mode of transmission for infectious respiratory diseases, such as influenza, to develop and implement effective interventions to prevent transmission. There are three basic modes of transmission of respiratory viruses: direct contact with another person (e.g., kissing) or with large droplets expelled from the respiratory tract, indirect contact with inanimate objects contaminated with respiratory secretions, and inhalation of fine particles either directly released from the respiratory tract or resulting from the evaporation of large droplets. Recently published review articles addressing transmission of influenza virus reach vastly different conclusions about transmission of influenza virus. A review by Tellier largely concluded that influenza is transmitted by fine aerosols (*2*), while Brankston and colleagues generally concluded that influenza is transmitted directly by large droplets (*3*). Considering this uncertainty, our research efforts have focused on two areas of intervention that are likely to have a large impact on influenza transmission: decontamination of contaminated surfaces and the use of surgical masks as source control, nonpharmaceutical interventions.

Decontamination of Surfaces Contaminated with Influenza Virus

The goal of our surface decontamination research is to prevent the secondary spread of influenza virus via contaminated fomites. Research has demonstrated the presence and survival of influenza virus in many environments (*4*). Decontaminating small objects or occupied spaces is easily accomplished by applying disinfectants to surfaces. However, decontaminating large complex structures such as commercial aircraft, trains, and buses requires large amounts of time and effort, resulting in significant downtime for the use of that resource. While preventing secondary spread of influenza is our goal, decontamination needs to meet the following requirements: no (or minimal) harm to surfaces, electrical systems, and mechanical components; no harmful residues remaining after treatment; and fast turnaround time. Taking these requirements into consideration, we evaluated the following candidate decontamination methods: low-concentration vaporous hydrogen peroxide (VHP), triethylene glycol (TEG) vapor, and heat with moisture.

Briefly, our experimental protocol consisted of applying liquid suspensions of influenza virus onto stainless steel coupons, allowing the suspension to dry, and exposing the coupons to the test environment. Control coupons, which were not exposed to the test environment, were used to calculate the magnitude of influenza virus

reduction. Test and control coupons were repeatedly rinsed and the rinse solution was assayed for influenza infectivity with a fluorescent focus reduction assay (*5*, *6*). Reductions of influenza were expressed as logarithmic (log) reductions. A log reduction of 1.0 is equivalent to a 10-fold decrease (90%) and a reduction of 2.0 is equivalent to a 100-fold decrease (99%), and so on.

Initial survival experiments were done at ambient conditions to determine the "natural," baseline decay rate for influenza virus. The number of log reductions versus time was generally linear, and our results showed 0.08 log reduction per hour. For VHP, generally, reduction marginally increased with increased exposure time or VHP concentration. At a VHP concentration of 10 parts per million (ppm), about 2 log reductions were observed after 2.5 min of exposure and the number increased to about 3.2 log reductions after 15 min (maximum exposure time measured). At a VHP concentration of 90 ppm (the highest concentration evaluated), about 3.2 log reductions were observed after 2.5 min of exposure and the number increased to about 4.7 log reductions after 15 min. VHP experiments were performed at about 25°C and 58% to 65% relative humidity (RH) (*7*). For TEG vapor concentrations ranging from 1.7 to 2.5 ppm, the number of log reductions versus time followed a linear relationship with a decontamination rate of 1.3 log reductions per hour. TEG vapor experiments were performed at 25°C to 29°C and 45% to 55% RH (*7*). Heat and RH experiments were carried out at 55°C, 60°C, and 65°C and 25%, 50%, and 75% RH. Surface inactivation of influenza virus increased with increasing temperature, RH, and exposure time. Greater than 5 log reductions of influenza virus on surfaces was achieved at temperatures of 60°C and 65°C, exposure times of 30 and 60 min, and RH of 50% and 75%. Our data also suggest that ambient humidity is a better predictor of surface inactivation than RH and allows for predicting survival by using two parameters instead of three, which greatly simplifies analysis and interpretation of virus survival data.

Surgical Masks as a Source Control for Influenza Transmission

We hypothesize that patients infected with influenza virus exhale infectious influenza virus aerosols. These aerosol particles are at their largest size and highest velocity as they exit the nose and mouth. Thus, surgical masks, which are normally considered inefficient for particle removal, may be able to capture a significant portion of these aerosols. The following specific aims were used to test these hypotheses: (*a*) measure number and size distribution of exhaled influenza viruses, and (*b*) measure the effect of wearing a surgical mask on the release of virus aerosol by patients. The research presented here was completed in two phases. The first phase was preliminary research to measure the output of influenza in infected patients, and the second phase consisted of measuring the utility of surgical masks to prevent the release of influenza virus aerosols from infected patients.

During Years 1 and 2 of the study, we used an Exhalair (Pulmatrix Inc., Lexington, Massachusetts) to collect exhaled breath from subjects infected with influenza virus within 3 days of the onset of symptoms. The Exhalair uses light scattering to measure particle number and size and a Teflon filter to collect particles for later analysis by quantitative reverse transcription–polymerase chain reaction (qRT-PCR) to measure influenza virus ribonucleic acid (RNA). During Year 1 of the study, carried out in Hong Kong, subjects wore a continuous positive airway pressure–type mask, breathed tidally for 3 min for the particle characterization phase, and breathed tidally for 15 min for the filter collection phase. The filter was washed and analyzed by qRT-PCR as described (*8*). Influenza status of each subject was confirmed by qRT-PCR of nasal swabs. Virus was detected in exhaled breath of four (33%) of the 12 studied patients, with generation rates ranging from <3.2 to 20 influenza RNA copies per minute. Particle count data showed that 87% of particles had an aerodynamic equivalent diameter <1 micrometer (μm_{AED}) and that fewer than 0.1% of particles were >5 μm_{AED}, which suggests that virus, detected by qRT-PCR, was present in fine particles generated during tidal breathing (*8*).

During Year 2 of the study, carried out at the University of Massachusetts, Lowell, testing similar to that for Year 1 was done, with the following exceptions: the particle counting phase was completed with a mouthpiece and nose clips rather than a continuous positive airway pressure mask, filter collection was performed for 30 min rather than 15 min, and an additional filter sample was collected while the subject was asked to cough 10 times. The filter was washed and analyzed by qRT-PCR as described. Influenza virus RNA was detectable in exhaled aerosols during tidal breathing but was more frequent in coughing than in tidal breathing. The generation rates were <3.2 to 20 RNA copies/min for tidal breathing and 0.1 to 419 RNA copies per cough.

During Year 3 of the study, also carried out at the University of Massachusetts, Lowell, surgical masks were evaluated as source control nonpharmaceutical interventions. During this study, we collected exhaled breath from influenza-positive subjects who were wearing ear-loop surgical masks for 30 min while tidal breathing and performing 10 voluntary coughs every 10 min. A second sample was collected from the same subject without a mask while tidal breathing and coughing as for the initial sample. Samples were collected with the G-II

exhaled breath air sampler (United States Provisional Patent Application No. 61/162,395). Briefly, subjects sat with their face directed into an obliquely truncated steel cone into which air was drawn by a vacuum pump at 160 L/min. The air then passed through a 5 μm_{AED} slit impactor with a Teflon collection substrate. The collection substrate was washed and analyzed by qRT-PCR as described (*9*). Forty-one subjects were tested and there was a significant reduction in the proportion of cases with detectable influenza in the samples collected while subjects were wearing surgical masks versus not wearing a mask for particles >5.0 μm_{AED}.

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The Role of Fomites in Transmission of Pathogens in Airports and on Airpcraft

Charles P. Gerba

Inanimate objects or fomites consist of porous and nonporous surfaces and objects that serve as vehicles for transmitting infectious diseases. During and after an illness, pathogenic microorganisms can be shed in large numbers in body excretions and secretions, including blood, feces, urine, saliva, and mucus. Fomites become contaminated with pathogens by direct contact with body fluids, contact with hands, contact with aerosols (large droplets), sneezing, coughing, vomiting, and contact when airborne organisms settle after resuspension from a contaminated surface. Once a fomite is contaminated, transfer of infectious microbes may readily occur between fomites and humans, or vice versa, and between two fomites (e.g., contaminated sponges used to wipe a surface).

Respiratory and enteric microorganisms can be readily transmitted when the hand becomes contaminated from contact with a fomite and the infectious microorganisms are transferred to a portal of entry (eyes, nose, mouth). Contact with the face is fairly frequent in children. Under 2 years of age, contact occurs about 81 times per hour. This number decreases to about 15.5 times per hour in adults (*1*). Factors controlling the probability of infection by this route include the frequency with which the fomite is contaminated, survival of the organism on the fomite, efficiency of transfer from the fomite to the hand, survival on the skin, and efficiency of transfer to the face. Viruses have a greater probability of being transmitted by fomites because of the greater probability of becoming infected with fewer organisms. For many enteric viruses, the dose to infect 50% of those exposed is only 1 to 100 viruses. Enteric bacteria may be excreted in numbers as great as 10^{10} per gram of feces and enteric viruses as much as 10^{11} per gram of feces.

Most bacteria causing respiratory and enteric infections usually survive only a few hours on dry surfaces, although enteric bacteria are capable of growing in moist environments (sponges, mops, cloths) in large numbers. Respiratory viruses such as influenza may survive from a matter of hours to several days on fomites (*2*). In contrast, enteric viruses, such as norovirus and hepatitis A virus, can live days to weeks on fomites. Survival of organisms on surfaces is related to the nature of the suspending fluid (longer survival in bodily fluids), temperature (longer survival at lower temperatures), RH (varies with the organism), and the nature of the surface (generally longer survival on porous surfaces).

The efficiency of transfer of organisms varies with the type of fomite and can vary from 0.01% to >50%. Generally, transfer is more efficient from hard nonporous surfaces such as stainless steel and porous surfaces

(cloth). Transfer from the hand to the face varies from 10% or more (*3*).

By knowing the degree of fomite contamination in an environment, survival, and transfer efficiencies, it is possible to model the probability of infection and the potential impact of interventions (*4*).

Surprisingly few studies have been done on the occurrence of respiratory and enteric pathogens on fomites in indoor environments. Such information is useful for the targeted use of cleaning and disinfecting efforts to reduce the risk of exposure. We have found that common high-touch areas and shared fomites become the most contaminated (*5*). Many other factors are important such as frequency with which an object or surface is cleaned or disinfected. For example, television remote controls and other types of electronic equipment tend to be more contaminated as they are seldom cleaned or disinfected (*6*). Also, cleaning tools (mops, cloths) can spread pathogenic organisms in an environment if disinfectants are not used.

When traveling, a common area we all share are public restrooms. Public restrooms have been implicated in outbreaks of *Salmonella*, *Shigella*, hepatitis A virus, and norovirus. We have found a greater frequency of enteric bacteria on fomites in airport and airplane restrooms than in hospital, fast-food restaurant, and office building restrooms. This finding probably reflects the high traffic in these restrooms. The most contaminated restrooms are in aircraft, probably because of the limited number of restrooms per passenger and the ease of using hand washing facilities (i.e., small sinks, water automatically shuts off). The common occurrence of enteric viruses in laboratory wastes collected from aircraft indicates that passengers are infected (*7*). In homes with persons infected with influenza, the virus can be isolated on more than half the fomites tested (phones, television remote controls) (*6*). Norovirus is also commonly isolated on fomites in schools and other public environments (*8*). Thus, individuals infected with these viruses can be expected to contaminate any environment they occupy. We have also detected fecal bacteria as well as norovirus on passenger trays, suggesting that these areas are not regularly cleaned or disinfected.

Risks are reduced from fomite transmission if proper hand washing, use of hand sanitizers, and disinfection of key areas are practiced. All these interventions have been shown to reduce risks of infection by 30% to 50% (*8*). Other potential interventions in aircraft and airports could include use of more persistent disinfectants, self-sanitizing surfaces, and surfaces that reduce transfer of microbes to the hands. To develop effective interventions, a better understanding of the occurrence of microbial contamination on fomites in aircraft and airports is needed. In this way, effective interventions can be developed and monitored for success.

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SESSION 5

Policies and Planning to Minimize the Spread of Disease

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Transmission Patterns of Mosquito-Borne Infectious Diseases During Air Travel: Passengers, Pathogens, and Public Health Implications

James H. Diaz (Presenter)

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, globalization of agriculture and food production, exotic eating habits, lifestyle, and residential choices. The worldwide spread of the Asian tiger mosquito, *Aedes 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, Bangladesh, and the Indian Ocean Islands, which are popular travel destination resorts (Figure 1).

Many models of climate change and vector–pathogen relationships now predict a significant expansion in potential malaria transmission cycles in the next few decades, with some studies predicting a 16% to 25% increase in person-months of exposure in malaria-endemic areas of Africa. 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.

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 vector for dengue fever and Chikungunya viruses. (Source: CDC Public Health Image Library, Image No. 4735.)

In 2008, Hochedez and coinvestigators in Paris reported their findings from a prospective study of 62 returning travelers who presented to their tropical diseases clinic with fever (above 38°C) and widespread rash over a 20-month period (*1*). The three main travel destinations were the Indian Ocean Islands (35%), Africa (21%), and Asia (18%). The three main tropical infectious disease diagnoses were Chikungunya (35%), dengue (26%), and African tick-bite fever (10%). Travel to the Indian Ocean Islands and South Africa was significantly

associated with Chikungunya and ATBF, respectively. The authors concluded that arthropod-borne infectious diseases presenting with fever and rash were not uncommon among returning travelers and that travelers returning from endemic areas should be rapidly screened for tropical infections, some of which could be fatal, such as dengue and malaria. The mosquito vectors of infectious diseases that may be imported by infected passengers are compared by geographic distribution ranges and infectious disease transmission in Table 1.

History Repeats Itself: Why Is Dengue Fever a 21st Century Public Health Threat?

Yellow fever outbreaks claimed tens of thousands of victims in coastal and inland U.S. seaports and throughout Latin America and the Caribbean until stopped by a live virus vaccine developed in the early 20th century. Dengue virus, like yellow fever, is a flavivirus but it comes from a larger family of dengue viruses and there is no effective vaccine for it. Dengue fever and, in particular, its complications from subsequent dengue infections with other dengue serotypes, such as dengue hemorrhagic fever (DHF) and dengue shock syndrome, may pose specific public health threats to the United States. Dengue is caused by four genetically related flaviviruses (DEN1 to -4); is transmitted by container-breeding, peridomestic *Aedes* species mosquitoes, preferentially *Aedes aegypti*; and can cause a spectrum of clinical manifestations ranging from asymptomatic initial infections to hemorrhagic fever with shock from microvascular plasma leakage.

Although an effective live vaccine is available for yellow fever (another flavivirus transmitted by *Aedes* mosquitoes), a dengue vaccine has proven very difficult to develop for several reasons: (*a*) the four dengue serotypes dictate a polyvalent vaccine, like the influenza vaccine; (*b*) a dengue vaccine must provide immunity against all four flaviviral serotypes at once by stimulating effective neutralizing antibodies; (*c*) the neutralizing antibodies must not cross-react and activate T cells, causing the cytokine reactions characteristic of DHF and DSS; and (*d*) multiple vaccinations every few years will likely be required to achieve long-lasting immunity against all four serotypes.

Dengue viruses are now endemic along the U.S.–Mexico border and have caused dengue fever outbreaks on both sides of the border and an autochthonous case of DHF in Brownsville, Texas, in 2005. Although yellow fever and dengue viruses historically have been confined to the tropics and transmitted by *Aedes aegypti*, a secondary *Aedes* vector, the Asian tiger mosquito *A. albopictus*, has now expanded its range globally in a warming ecosystem and is a competent vector of dengue viruses (Figure 1). The World Health Organization (WHO) considers dengue to be one of the world's most important reemerging infectious diseases, with 50 million to 100 million cases annually; 0.5 million hospitalizations, often requiring blood product transfusions; and 22,000 deaths annually, mostly in children. Even though the first dengue infection may be mild, the second could be lethal, even if it occurs years later. As there are no vaccines or specific drug treatments for dengue and because local *A. aegypti* and *A. albopictus* mosquitoes are capable of transmitting dengue in the United States, dengue poses a significant threat to the United States and a safe quadrivalent vaccine and better mosquito vector control along the U.S.–Mexico border are needed now.

TABLE 1 Mosquito Vectors of Infectious Diseases That May Be Imported by Infected Travelers or Vectors on Aircraft or in Airports

Mosquito Genera	Infectious Diseases Transmitted	Geographic Distribution Ranges	Causative Microbial Agents	Classification of Causative Agents
Anopheles spp.	Malaria	Africa, Asia, Central America, South America	*Plasmodium falciparum*, *P. vivax*, *P. ovale*, *P. malariae*	Protozoan parasites
Anopheles spp.	Bancroftian filariasis	Southeast Asia	*Wuchereria bancrofti*	Filarial worms causing lymphatic filariasis
	Brugian filariasis	Southeast Asia	*Brugia malayi*	
	Timorian filariasis	Timor, Indonesia	*Brugia timori*	
Anopheles spp.	O'nyong nyong fever	Africa	Alphavirus	Togaviruses
Aedes spp.	Yellow fever	Africa, Latin America	Flavivirus	Flaviviruses
	Dengue fever	Africa, Asia, Latin America	Flaviviruses DEN 1-4	Flaviviruses
	Chikungunya fever	Africa, Asia	Alphavirus	Togaviruses
	Eastern equine encephalitis	Eastern & Southeastern USA	Alphavirus	Togaviruses
	Ross River fever	Australia, Papua New Guinea	Alphavirus	Togaviruses
	California encephalitis	Western USA	Bunyavirus	Bunyaviruses
	LaCrosse encephalitis	Midwestern USA	Bunyavirus	Bunyaviruses
	Rift Valley fever	Africa	Phlebovirus	Bunyaviruses

Why We Could Not Stop the Spread of West Nile Virus Across the United States

Although dengue viruses are carried by mosquitoes or infected humans across the porous U.S.–Mexico border, West Nile virus was most likely imported to the United States in 1999 by international air travel. The West Nile virus arrived in New York City courtesy of an infected passenger or an infected *Culex* 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 fly 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 mostly 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 more than 4,000 cases of meningoencephalitis with 263 deaths [case fatality rate (CFR) = 6.6%].

Why Are Mosquitoes Such Competent Transmission Vectors of Infectious Diseases in an Era of Climatic Change?

Only female mosquitoes seek frequent blood meals for their developing eggs from preferred nearby hosts. All female mosquitoes lay their eggs in standing water, either on or just below the surface. 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, uncovered garbage cans, and even discarded tires. Climate changes, particularly warming nighttime temperatures and increased precipitation, offer selective advantages to all mosquito species, including (*a*) a longer reproductive life and a prolonged breeding season, (*b*) opportunities for more blood meals during gestation, (*c*) plenty of standing water surfaces for egg laying, and (*d*) a faster egg hatch over days and not weeks.

International Air Travel and Malaria

Malaria, a mosquito-transmitted parasitic disease, remains the most common cause of infectious disease deaths worldwide, followed by tuberculosis and AIDS. Although there are four *Plasmodium* protozoans capable of causing malaria in humans (*P. falciparum, P. malariae, P. ovale, P. vivax*), *P. falciparum* and relapsing *P. vivax* are the most common causative agents, with *P. falciparum* having a significantly higher CFR than *P. vivax*. According to the WHO's *World Malaria Report* (2005), 3.2 billion people live in malaria-endemic regions in 107 countries and territories, and there are between 350 million and 500 million cases worldwide per year, with 840,000 to 1.2 million deaths from malaria annually. Most malaria deaths occur in children under age 5 years, in pregnant women, and in nonimmune individuals, often travelers and expatriates returning to their malaria-endemic homelands to visit friends and relatives. About 60% of all cases of malaria worldwide and more than 80% of deaths from malaria worldwide occur in sub-Saharan Africa. Most malaria deaths worldwide are caused by *P. falciparum* transmitted by highly competent mosquito vectors, such as *Anopheles gambiae* in Africa, where transmission occurs year round.

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 in a warmer and wetter climate and include airport malaria and, more significantly, imported malaria. 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. The malaria-infected mosquito vector is a new arrival on an international flight from a malaria-endemic region. Airport malaria is transmitted by the bite of an infected tropical anopheline mosquito within the vicinity of an international airport, usually a few miles or even less. On the other hand, imported malaria is defined as the intercontinental transfer of malaria by the movement of a parasitemic person with malaria to a nonendemic disease area with locally competent anopheline vectors in a welcoming ecosystem. Climate change has now expanded the geographic distribution of malaria-endemic regions worldwide and extended the length of seasonal malaria transmission cycles in endemic regions, so more arrivals of malaria-carrying mosquitoes and malaria-infected travelers are anticipated. The greatest public health threats that imported malaria-infected mosquitoes and patients with malaria pose to nonmalarious regions include the reintroduction of *Plasmodium* species (especially *P. vivax* in the United States and Europe) into regions with competent anopheline vectors and the reestablishment of local or autochthonous malaria by local anopheline vectors.

Airport Malaria

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. After the female mosquito leaves the aircraft, she may survive long enough, especially during temperate periods, to take a blood meal and transmit pathogens, usually in the vicinity of an international airport. After one or more blood meals, female mosquitoes seek a water surface to lay their eggs.

As international air travel between malaria-endemic nations and malaria nonendemic nations increased, 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 about 7.5 km 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.

Imported Malaria

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 increase in long-distance air travel by infected passengers. Malaria cases imported from Africa to the United Kingdom (U.K.) rose from 803 in 1987 to 1,165 in 1993. By 2006, a total of 1,758 malaria cases were reported in the U.K. 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 currently accounting for most of the cases. In the United States in 2005, a total of 1,528 cases of imported malaria were diagnosed, an increase of 15% over the prior year. Today, imported malaria is the most common type of malaria in developed nations, with more than 10,000 cases reported annually; imported malaria remains the most common cause of fever in travelers returning from malaria-endemic regions.

In a retrospective analysis of 380 imported malaria cases in Verona, Italy, over the 5-year period 2000–2004 and 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 in malaria-endemic regions (n = 154, 40.5%) (*2*). Most cases were caused by single *P. falciparum* infections (n = 292, 76.8%), with few mixed *Plasmodium* infections (n = 23, 6%) (*2*). The authors concluded that malaria in travelers returning to Verona from Africa was not uncommon and targeted certain high-risk travelers, including adult expatriate immigrant travelers visiting friends and relatives, semi-immune children (recent immigrants), and nonimmune children (expatriates or born in Italy).

In a similar retrospective analysis of 109 travelers with malaria returning to Basel, Switzerland, over the period 1994–2004, Thierfelder and coinvestigators 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 to 31 days) (*3*). After their descriptive analysis, the investigators conducted three comparative analyses with two prior studies of malaria in travelers returning to Basel during the periods 1970–1986 and 1987–1992. The results of their comparative analyses included significant increases in the proportions of *P. falciparum* infections over three study periods (1970–1986, 49%; 1987–1992, 75%; 1994–2004, 88%) and significant increases ($P < .001$) in hospitalizations for *P. falciparum* malaria over the three decades studied. The authors concluded that there was a significant trend toward more serious malaria infections with *P. falciparum* in immigrants returning to Basel after visiting friends and relatives in their malaria-endemic native homelands.

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 United States after spending 2 months in East Africa in 2005 (*4*). Of the six patients, all were young (19 to 22 years of age) and in prior excellent health; five of the six exhibited features of acute cerebral malaria (disorientation, prostration) requiring urgent intensive care and therapy with intravenous quinine. The authors commended alert U.K. airport staff for recognizing the seriously ill travelers preparing to board a 9-h second-leg flight to the United States and for rapidly evacuating the patients to the nearest health care facility for intensive care, without which the five cerebral malaria cases would likely have been fatal.

Although many developed nations, such as northern Europe and the United States, do not have as efficient mosquito vectors for *P. falciparum* malaria as *A. gambiae* in sub-Saharan Africa, many nonendemic nations in southern Europe, the Middle East, and Asia do have efficient vectors for *P. falciparum*, and most have competent vectors for *P. vivax*, including the United States and Europe. The most disturbing recent trends in imported malaria today include the following: (*a*) an increasing proportion of *P. falciparum* infections capable of causing cerebral malaria and renal failure with the highest

CFRs; and (*b*) increasing immigration from malaria-endemic regions to malaria-free regions in developed nations, creating a unique set of high-risk travelers, especially expatriates (semi-immunes) and their children (often nonimmune) returning from visiting friends and relatives in their malaria-endemic native homelands.

In summary, imported malaria cases are increasing worldwide because of the ease and relatively low costs of international air travel to malaria-endemic regions worldwide. The world's malaria-endemic regions now have expanded distribution ranges for malaria transmission and longer mosquito vector breeding–feeding seasons due to global warming and increasing drought–monsoon cycles.

Autochthonous (Locally Transmitted or Reintroduced) Malaria

In the United States, 21 outbreaks of presumed locally transmitted or autochthonous mosquito-borne malaria transmission have been reported since 1950, all caused by *P. vivax*. Most of these introduced malaria outbreaks (n = 14), occurred in southern California, primarily among migrant Mexican agricultural workers. In 1986, a *P. vivax* malaria outbreak resulted in 28 cases of the disease, 26 of which were in Mexican migrant workers, over a 3-month period. In 1988, another outbreak of locally transmitted *P. vivax* malaria occurred in San Diego County, California, and involved 30 patients, again mostly migrant farm workers, and represented the largest reported outbreak of autochthonous malaria in the United States since 1952. Epidemiologic and microbiologic investigations of these malaria outbreaks later confirmed secondary spread from infected immigrants to other immigrants and local residents transmitted by local malaria-competent anopheline vectors.

Conclusions

Competent mosquito vectors for dengue, yellow fever, and Chikungunya virus are now present in the United States, including *A. aegypti* in the southern United States and *A. albopictus* throughout the country, and are awaiting an opportunity to transmit these imported arboviral diseases locally from arriving infected airline travelers to nonimmune citizens nearby. In addition, anopheline species have demonstrated their capacity to transmit imported *P. vivax* malaria along the U.S.–Mexico border and to transmit more serious *P. falciparum* malaria from arriving infected airline travelers and nonimmune individuals in southern Europe.

Prevention and control strategies for the imported arboviral infectious diseases (Chikungunya virus, dengue, and West Nile virus) and for airport, imported, and autochthonous malaria should include early case definition, case confirmation, and treatment; strengthened vector surveillance to detect the potential for autochthonous or local 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 in nonendemic areas, especially malaria, dengue, Chikungunya, and West Nile virus.

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Airline Policies and Procedures to Minimize the Spread of Diseases

Rose M. Ong (Presenter)

Faced with the outbreak of severe acute respiratory syndrome (SARS) in 2003, airlines found that they were generally ill prepared to deal with infectious diseases with public health concerns. Since that time, especially for an Asian-based carrier such as Cathay Pacific, there have been a number of other "novel" communicable diseases, including avian influenza and most recently the pandemic A/H1N1 influenza epidemic. Air travel is frequently cited as being responsible for the rapid spread of communicable diseases on a worldwide basis.

Since 2003, significant progress has been made among various commercial airline stakeholders to collaborate to minimize the spread of communicable diseases onboard flights.

Airlines followed guidance issued by major international organizations such as the International Civil Aviation Organization (ICAO), WHO, U.S. Centers for Disease Control and Prevention, International Air Transport Association (IATA), and Airport Council International (ACI) as well as local organizations such as the Hong Kong Centre for Health Protection. Many initiatives have been introduced by these organizations to promote better alignment and collaboration among key stakeholders in managing infectious diseases in air travel.

Airlines engage in routine baseline activities to manage infectious diseases, which include educating and training frontline staff, crew fitness to fly, cabin air conditioning and ventilation, cabin hygiene and sanitation, in-flight catering hygiene, and preparedness drills conducted in conjunction with airport authorities. Emphasis was placed on the aircraft ventilation system; it introduces fresh air at a rate of 50%, which is mixed with recirculated air and filtered through high-efficiency particulate air filters, with a 99.9% efficiency rate of removal of airborne biological contaminants. The entire cabin air volume is exchanged every 2 to 3 min with laminar airflow patterns, which minimizes longitudinal air movement, lowering the risk of in-flight transmissions in a forward-and-aft direction. The aircraft is cleaned and disinfected in accordance with maintenance schedules.

Other actions are taken in response to specific infectious incidents, including activation of the in-flight medical management systems (e.g., cabin crew training, in-flight aeromedical telephonic support, medical equipment including personal protective equipment, bloodborne pathogen barriers) and contact tracing of crew and passengers as appropriate. Crew have specific protocols to follow when a passenger is suspected of having a communicable disease; the individual is given a mask to wear, relocated to the rear of the aircraft if appropriate and possible, assigned a toilet if appropriate, and given tissues or a disposal bag to use. One crew member should be assigned to look after the sick passenger. The crew will communicate with the telephonic medical advisory and, if appropriate and indicated, the pilot will notify the en route air traffic control, who will advise health authorities in the arrival port.

During an infectious disease outbreak, additional measures are taken, including screening temperatures of all crew before operating an aircraft, providing refresher training and safety reminders for all crew at crew depar-

ture lounges, stepped-up cleaning of aircraft cabin and equipment, and judicious use of masks by crew.

We also developed a series of business continuity plans taking into account the need to balance a positive cash flow position, protecting company brand and reputation while protecting the health and safety of passengers and employees.

The Practical Application of World Health Organization Travel Recommendations: Some Observations

Anthony D. B. Evans (Presenter)

On April 25, 2009, the WHO Emergency Committee [established in accordance with International Health Regulations (IHR-2005)] provided its view to Margaret Chan, Director General of WHO, that an influenza A/H1N1 outbreak represented a "public health emergency of international concern." On April 27, 2009, after the second meeting of the emergency committee, Chan raised the level of influenza pandemic alert from Phase 3 to Phase 4. At that time some additional announcements were made, including the following:

- Given the widespread presence of the virus, the director general considered that containing the outbreak was not feasible. The focus should be on mitigation measures.
- The director general recommended not closing borders and not restricting international travel.

This paper discusses the practical application of these recommendations by WHO (www.who.int/en/).

International Civil Aviation Organization

ICAO is a United Nations specialized agency that works to achieve a safe, secure, and sustainable development of civil aviation through cooperation among its member states (www.icao.int/). Its work is underpinned by the Convention on International Civil Aviation (signed in Chicago, Illinois, it is also known as the Chicago Convention) of which Article 14 states, in part "Each contracting State agrees to take effective measures to prevent the spread by means of air navigation of cholera, typhus (epidemic), smallpox, yellow fever, plague, and such other communicable diseases as the contracting States shall from time to time decide to designate, and to that end contracting States will keep in close consultation with the agencies concerned with the international regulations relating to sanitary measures applicable to aircraft."

Although written in 1944, upon establishment of ICAO, it remains relevant. It is because of this article that ICAO and the national regulatory authorities for aviation in each contracting state to the Chicago Convention undertake work on pandemic preparedness planning, in cooperation with WHO, IATA (www.iata.org/index.htm), ACI (www.airports.org/cda/aci_common/display/main/aci_content07.jsp?zn=aci&cp=1_665_2_), and other stakeholders.

Airport Screening

Although airport screening was not specifically mentioned in the announcement by Chan at the start of the outbreak, a document posted by WHO on May 1, 2009, entitled "No rationale for travel restrictions" clarified WHO's view by stating "Furthermore, although identifying the signs and symptoms of influenza in travelers can be an effective monitoring technique, it is not effective in reducing the spread of influenza as the virus can be transmitted from person to person before the onset of symptoms. Scientific research based on mathematical modeling indicates that restricting travel will be of limited or no benefit in stopping the spread of disease. Historical records of previous influenza pandemics, as well as experience with SARS, have validated this point."

Despite this advice from WHO, many states (countries) undertook, and continue to undertake, some form of screening at airports, including the use of thermal imaging to detect individuals with an elevated temperature. In addition, a few states quarantined travelers perceived to be at increased risk of incubating influenza. At the other end of the spectrum, some states have taken no action to identify possible cases.

This inconsistency of approach has two main disadvantages: travelers receive mixed messages from authoritative bodies, resulting in confusion about the actual risk, and those states undertaking screening use resources that might be more effectively used for some other purpose. The cost of screening is not trivial; for example, a thermal scanner may cost tens of thousands of dollars in addition to the cost of training personnel and operating the equipment. Medical staffs are required to assess further those individuals identified as having an elevated temperature.

As there appears to be little scientific justification for screening passengers at airports, it may be worthwhile exploring further the reasons why states apply such measures. There is some evidence that governments may wish to demonstrate to their citizens that action is being taken to reduce the risk of illness, or they may wish to reassure travelers or deter unwell individuals from flying. A survey of public health authorities could help to elucidate the reasons and form the basis for a more consistent approach.

Significant Interference with International Traffic

One aim of the WHO IHR-2005 is to provide a public health response to the international spread of disease, which avoids unnecessary interference with international traffic and trade. An important description in the IHR is therefore that of "significant interference." It is found in Article 43, where it is described as "refusal of entry or departure of international travelers, baggage, cargo, containers, conveyances, goods and the like, or their delay for more than 24 hours." In the aviation sector, delaying an aircraft's departure by more than a few minutes can disrupt operations and may be regarded as "significant interference" as far as an airline or its passengers and crew are concerned, even though it may not fall into the category of such interference according to the IHR. While aircraft delays for public health reasons may be justified, and unavoidable in certain circumstances, such delays can sometimes be imposed by a public health authority without full knowledge of the effects of such disruption on aircraft operations.

One reason for this situation is that much of the work of public health authorities is devoted to issues of national importance, and they may not be so focused on the international implications of their actions. On the other hand, an airline operating in 20 international airports may have to comply with many different public health requirements for documentation, screening, and reporting, all of which can cause inefficiencies and delay because they are not standardized. Airlines are therefore well aware of the potentially adverse effects of a lack of international public health harmonization. There may be good reasons for different public health responses from different states, but it appears that such differences often arise because of a lack of coordination between states rather than because of a difference in risk.

To minimize such differences, ICAO and WHO are working with the trade associations IATA and ACI as well as other organizations to try to improve harmonization of the public health response to diseases with pandemic potential.

WHO's Public Health Mandate

According to IHR-2005, its purpose and scope are "to prevent, protect against, control and provide a public health response to the international spread of disease in ways that are commensurate with and restricted to public health risks." WHO is therefore concerned with health risks that are relevant to the public health—that is, the health of communities. However, other health care providers may approach the question of risk from a different viewpoint. Occupational health physicians need to take account of the risks to the assets of their company or organization when advising about travel during an outbreak or pandemic. They need to consider risks to efficiency that are unrelated to public health risks, such as the chance that an employee may be stranded abroad (e.g., because of quarantine requirements or because of illness). Further, employees may prefer to delay or avoid travel in view of the perceived risk or because they do not wish to be away from home if illness affects their family when they are traveling.

In the same manner, a physician advising an individual patient about travel during an outbreak or pandemic may need to take account of specific circumstances that affect only that individual and that do not apply to the community as a whole.

The WHO message that travel restrictions and screening are not recommended is reassuring. However, when the other aspects are considered by health care providers, who have a different priority from that of public health, different messages about risk to individuals can contribute to the lack of a clear understanding about the risks involved.

Summary

IHR-2005 provide a solid basis for implementing proportionate measures that mitigate the risk to public health from influenza A (H1N1) by international travel. They permit flexibility to deal with the specific situation that has enabled WHO to provide consistent travel recommendations during the outbreak and subsequent pandemic of influenza A (H1N1). However, such recommendations have not been applied in a harmonized manner. Continued international communication and collaboration among public health authorities and between the public health and aviation sectors should help develop a more harmonized approach.

Two Recommendations for Further Research

1. Examine the motives of states in implementing screening and evaluate the outcomes of such screening.
2. Assess the effects of screening on the efficiency of aircraft and airport operations.

SESSION 6

Discussion of Topics for Future Research

The following tables are based on feedback received during discussion of topics for future research that occurred on Day 2 of the symposium. Audience members, speakers, moderators, and planning committee members participated in the open discussion. These tables capture and organize the research topics discussed into three main categories (foundational research, airport and aircraft research, and prevention and mitigation opportunities for air travel) and three pathogen transmission areas (source, transit, and receptor). The research topics are presented in no particular hierarchical order, and their inclusion here does not imply endorsement by the symposium participants, the planning committee, or TRB. Rather, they are summarized here in Tables 1, 2, and 3 as a record of the symposium discussion.

TABLE 1 Source of Pathogens[a]

Foundational Research[b]	Airport and Aircraft Research[c]	Prevention and Mitigation Opportunities for Air Travel[d]
Improve disease transport models by quantification of infectious particles and droplets from human exhalation.	Correlate symptomatic crew and passengers with pathogen concentrations in flight.	Develop methods to encourage travelers to self-report an illness.
Evaluate and understand differences between biowarfare (i.e., intentional release of pathogens) and naturally occurring infectious diseases.	Identify reasons why infectious people travel and assess the accuracy of their responses when asked by a public health official about their symptoms.	Develop protocols for screening at airports that optimize public health protection while minimizing operational impacts.
Determine what data elements can be gathered during outbreaks to support theoretical modeling studies.	Evaluate use of personal protective equipment by aircraft cabin crew and implications for safety-related functions.	Develop best practices for infection control in airport and aircraft settings.
Assess how modeling data can be used in survey design and investigative studies of outbreaks.	Identify barriers to good public hygiene practices by air travelers (e.g., limited access to hand-washing facilities).	
Assess the effectiveness of personal protective equipment in minimizing aerosol transmission of disease.	Evaluate effectiveness of exit screening and entry screening at airports.	
Evaluate human behavior related to using face masks to prevent the spread of disease.	Conduct real-time assessments of passenger screening efforts to identify operational impacts.	
Identify promotional techniques to improve passive and active public hygiene practices.		

[a] Infected person or other source of pathogens.
[b] Research needed to better understand infectious diseases in general and how they are spread.
[c] Research needed to better characterize disease transmission in airport and aircraft environments.
[d] Application of research to measures that may prevent or mitigate the spread of disease in the airport or aircraft environment.

TABLE 2 Transit of Pathogens[a]

Foundational Research[b]	Airport and Aircraft Research[c]	Prevention and Mitigation Opportunities for Air Travel[d]
Improve understanding of spore and virus survival rates, size of particles, dose of release, and infectivity of transported and deposited pathogens. Determine most important pathways for disease transmission. Identify bioaerosol markers that could help improve understanding of fate and transport of biological through combined biological and physical research efforts. Evaluate risk from sewage in watersheds through monitoring and measurement programs. Coupling of exposure modeling with quantitative microbial risk assessments to address specific science needs (e.g., relative risk). Assess quality of CDC surveillance data in their Quarantine Activity Reporting System. Conduct microbial background characterization of multiple modes of transmission (e.g., aerosol vs. fomite) to improve usefulness of biosensor systems. Assess the role of occupancy density and ventilation rate per person in airborne pathogen spread.	Measure fomites in all areas of the aircraft and airport environment and compare with other environments to assess relative risk. Evaluate the aircraft and airport environment during boarding and deplaning, when the aircraft is using auxiliary ventilation systems (e.g., gate-supplied air and power or auxiliary power units). Distinguish between the designed, within-row convective flow induced by the ECS and the between-row flow that occurs due to eddy action. Measure concentrations of airborne pathogens in aircraft cabins in actual flight. Improve characterization of microbial diversity and related risks on domestic and international aircraft. Assess safety of effective disinfection agents in the aircraft environment. Develop disease propagation models that integrate flight statistics, disease severity, and seasonality to assist in evaluating biosurveillance infrastructure. Evaluate the efficacy of disinfection efforts to prevent the spread of malaria and other insect- and vector-borne diseases. Evaluate detection and control strategies used by other industries that could transfer to airport and aircraft environment.	Develop passive control measures to mitigate fomite and airborne transmission of disease in aircraft and in airports. Identify effective measures to prevent the transport of potentially infected insects and other measures to reduce the risk of vector-borne diseases. Develop effective procedures and protocol for crew to manage infectious passengers.

[a] Movement of pathogens through space from an infected individual or other source of pathogens to a new receptor.
[b] Research needed to better understand infectious diseases in general and how they are spread.
[c] Research needed to better characterize disease transmission in airport and aircraft environments.
[d] Application of research to measures that may prevent or mitigate the spread of disease in the airport or aircraft environment.

TABLE 3 Receptor of Pathogens[a]

Foundational Research[b]	Airport and Aircraft Research[c]	Prevention and Mitigation Opportunities for Air Travel[d]
Distinguish between microbials that can be detected in the air or on surfaces and those that actually cause illness. Identify environmental and personal factors that make individuals more or less susceptible to infection (e.g., relative humidity, fatigue). Identify human behavior that contributes to or mitigates infection (e.g., touching face frequently).	Identify unique characteristics of the airport and aircraft environment or work practices that would make employees more susceptible to infection and implications for occupational health care providers. Evaluate relative infection risk in airports and on aircraft in comparison with other environments (offices, hospitals). Evaluate methods for reduction of the burden of illness in travelers.	Identify communication techniques and develop messages that clearly explain risks to personnel and the traveling public, and evaluate their effectiveness in reducing travel-related disease transmission. Identify disinfection measures that are broad spectrum, as safe as possible, environmentally benign, and compatible with materials used in the airport and aircraft environment.

[a] A susceptible individual who may be infected by a pathogen.
[b] Research needed to better understand infectious diseases in general and how they are spread.
[c] Research needed to better characterize disease transmission in airport and aircraft environments.
[d] Application of research to measures that may prevent or mitigate the spread of disease in the airport or aircraft environment.

APPENDIX A

Symposium Agenda

Research on the Transmission of Disease in Airports and on Aircraft: A Symposium

September 17–18, 2009
Washington, D.C.

Day 1—Starting at 8:30 a.m. and concluding at 5:00 p.m.

Welcome and Opening Remarks
Katherine Andrus, Air Transport Association, Symposium Planning Committee Chair
Christine Gerencher, Transportation Research Board

Session 1
Understanding How Disease Is Transmitted via Air Travel
Katherine Andrus, Air Transport Association, Moderator

- How Infectious Disease Spreads—*Michael Bell, Centers for Disease Control and Prevention*
- The Aircraft Cabin Environment—*Jeanne Yu, Boeing Commercial Airplanes*
- Human Movement Patterns and the Spread of Infectious Diseases—*Ben Cooper, U.K. Health Protection Agency*

Session 2
Practical Case-Response Approaches to Investigating the Spread of Disease in Airports and on Aircraft
John Neatherlin, Centers for Disease Control and Prevention, Moderator

- Norovirus Transmission on Aircraft—*Dan Fishbein, Centers for Disease Control and Prevention*
- Investigations of Tuberculosis on Aircraft—*Karen Marienau, Centers for Disease Control and Prevention*
- Swine Flu A/H1N1 Transmission Via the Aviation Sector—*Itamar Grotto, Israel Ministry of Health*

Session 3
Theoretical Modeling Approaches to Investigating the Spread of Disease in Airports and on Aircraft
Jennifer Topmiller, National Institute of Occupational Safety and Health, Moderator

- Summarizing Exposure Patterns on Commercial Aircraft—*James S. Bennett, National Institute of Occupational Safety and Health*
- Advance Models for Predicting Contaminants and Infectious Disease Virus Transport in the Airliner Cabin Environment—*Yan Chen, Purdue University, and Byron Jones, Kansas State University*
- Quantitative Microbial Risk Assessment—*Joan Rose, Center for Advanced Microbial Risk Assessment*

Session 4
Experimental "Bench Science" Approaches to Investigating the Spread of Disease in Airports and on Aircraft
Jack Spengler, Harvard School of Public Health, Moderator

- Airport-Related Biological and Chemical Transport of Infectious Diseases—*Richard Sextro, Lawrence Berkeley Labs*
- Disinfection and Production Rates of Viruses—*James McDevitt, Harvard School of Public Health, and Don Milton, University of Maryland*
- The Role of Fomites in the Transmission of Pathogens in Airports and on Aircraft—*Charles Gerba, University of Arizona*

End-of-Day Wrap-Up Discussion

Day 2—Starting at 8:30 a.m. and ending at 12:30 p.m.

Session 5
Policies and Planning to Minimize the Spread of Disease
Ben Cooper, U.K. Health Protection Agency, Moderator

- Transmission Patterns of Mosquito-Borne Infectious Diseases During Air Travel: Passengers, Pathogens, and Public Health Implications—*James Diaz, Louisiana State University Health Sciences Center*
- Airline Policies and Procedures to Minimize the Spread of Diseases—*Rose Ong, Cathay Pacific Airways*
- The Practical Application of the World Health Organization Travel Recommendations: Some Observations—*Tony Evans, International Civil Aviation Organization*

Session 6
Discussion of Topics for Future Research

Summary, Comments, Next Steps
Katherine Andrus, Air Transport Association, Symposium Planning Committee Chair
Christine Gerencher, Transportation Research Board

Symposium Conclusion

APPENDIX B

Reference Materials

NOTE: This information is not intended to be all-inclusive but is a synopsis of some references that may be informative and useful.

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