EFFECT OF PEDESTRIAN MOVEMENT ON THE SPREAD OF INFECTIOUS DISEASES DURING AIR TRAVEL: A MODELING STUDY

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ABSTRACT

This paper presents a formulation for an integrated model combining a social force based pedestrian movement including collision avoidance and a stochastic infection dynamics framework to evaluate the spread of infectious diseases in air transportation medium. We apply the multiscale model for two infectious diseases (1) Ebola and (2) SARS pathogens with different transmission mechanisms and compare the pattern of propagation during airplane boarding and deplaning at the airport gate. The objective of this analysis is to assess the influence of pedestrian movement on infection spread at an airport departure lounge.

INTRODUCTION

Air transportation medium and facilities are evolving exponentially to meet the necessity of connection, exchange and travel in an increasingly interconnected world. In addition to its many benefits, commercial air travel also enables rapid transmission of infectious diseases across the globe. Travelers are in close proximity to each other and are susceptible to infection spread during different phases of air travel. Pedestrian movement within airport is key to understanding and estimating the casual contacts between passengers at the airport and modeling the same can provide useful insight into disease spread.

Movement of passengers in airports is a special case of a more general problem of pedestrian movement. This problem has been addressed using several approaches such as particle dynamics or social force models [1, 2], models based on cellular automata [3], fluid flow models [4], and queuing based models [5]. Of these different approaches, social force models have specific advantages for studying passenger movement and contacts because each passenger is modeled individually and moves continuously which enables computing the individual trajectory contacts between pedestrians.

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2. Helbing, Dirk, Illés Farkas, and Tamas Vicsek. "Simulating dynamical features of escape panic." *Nature* 407, no. 6803 (2000): 487-490.

3. Burstedde, Carsten, Kai Klauck, Andreas Schadschneider, and Johannes Zittartz. "Simulation of pedestrian dynamics using a two-dimensional cellular automaton." *Physica A: Statistical Mechanics and its Applications* 295, no. 3 (2001): 507-525.

4. Henderson, L. F. "The statistics of crowd fluids." *nature* 229 (1971): 381-383.

5. Van Landeghem, Hendrik, and Annelies Beuselinck. "Reducing passenger boarding time in airplanes: A simulation based approach." *European Journal of Operational Research* 142, no. 2 (2002): 294-308.

Several studies use these generic approaches to study the pedestrian movement at airports especially from the viewpoint of airport operations and reduction of the turnaround time of airplanes at terminals. Schultz et al. (2006) model the intuitive behavior of airport travelers under emergency situation by a cellular automaton model.[9] In this model, the floor area is subdivided into small partitions where pedestrians may switch positions with neighboring spots based on a probabilistic distribution.[6] Several other investigators used agent-based models to model pedestrian motion in and passenger flow in airport terminals.[7,8] Other studies such as that by Lin et al. (2014) study the flow of pedestrians to their destinations by optimizing the guiding signs.[9] Pedestrian movement in airports is peculiar because it involves a series of nondiscretionary as well as discretionary activities. For example, prior to their scheduled flights, travelers fulfill the trip requirements starting from check-in, security and boarding. Once these processing steps are completed, they are often involved in individual or collective discretionary activities such as dining and shopping at the departure terminal.[10,11] The airport environment and building layout have a great influence on the passengers movements, choice and perception of activities preference over a set of alternatives.[9,12] This uncertainty creates additional challenges in modeling the pedestrian motion at airports.

Air travel brings together people from different geographic regions with different levels of vulnerability and receptivity due to variations in immunity, ethnic background, and intervention usage across geographic areas, consequently, airports and airplanes are potential, prime locations for infection spread.[13] During the Ebola epidemic

6. Schultz, M., S. Lehmann, and H. Fricke. "Pedestrian dynamics in airport terminals considering emergency cases." *Proceedings of Internation Council of the Aeronautical Sciences* (2006).

7. Ma, Wenbo. "Agent-based model of passenger flows in airport terminals." PhD diss., Queensland University of Technology, 2013.

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9. Lin, Yi-Hsin, and Ching-Fu Chen. "Passengers' shopping motivations and commercial activities at airports–The moderating effects of time pressure and impulse buying tendency." *Tourism Management* 36 (2013): 426-434.

10. Kraal, Ben, Vesna Popovic, and Philip J. Kirk. "Passengers in the airport: artefacts and activities." In *Proceedings of the 21st Annual Conference of the Australian Computer-Human Interaction Special Interest Group: Design: Open 24/7*, pp. 349-352. ACM, 2009.

11. Popovic, Vesna, Ben Kraal, and Philip J. Kirk. "Towards airport passenger experience models." In *Proceedings of 7th international conference on design & emotion*. 2010.

12. Kalakou, Sofia, Filipe Moura, and Valério Medeiros. "Analysis of airport configuration and passenger behaviour." In *Proceedings of the 10th International Space Syntax Symposium*. 2015.

13. Tatem, Andrew J., David J. Rogers, and S. I. Hay. "Global transport networks and infectious disease spread." *Advances in parasitology* 62 (2006): 293-343.

in 2014, models estimate that without travel restrictions, 7.17 infectious passengers per month would depart from the highly affected countries Liberia, Sierra-Leone and Guinea, to various destinations around the globe.[14] Transmission of Severe acute respiratory syndrome (SARS) virus via air travel has been recorded, in 2003, on three carries; Among 681 passengers, 23 tested positive for illness.[15] Several other diseases like tuberculosis, norovirus etc, have been transmitted through air travel.[16-19] In 1994, an infective with multidrug-resistant tuberculosis was onboard flights from Honolulu to Baltimore, passing by Chicago, transmitted the illness to passengers seated in the vicinity.[20] Three factors are known to influence the contagion spread: the infectivity of the index infectious, the flight duration and the number of contacts within the critical radius of infection.[20] The number of contacts is critically dependent on the pedestrian movement within airplanes and at airport lounges. Given the preponderance of infection spread through air travel, it is essential to identify air-travel related policies that can mitigate infection spread.

In this paper, we discuss the formulation of a multiscale model combining social-force based pedestrian movement with a population level stochastic infection transmission dynamics framework. We first formulate social force model for pedestrian movement incorporating collision avoidance and line forming. We then integrate this model with a stochastic susceptible-Infected infection transmission model. The model is then applied to study the infection

14. Bogoch, Isaac I., Maria I. Creatore, Martin S. Cetron, John S. Brownstein, Nicki Pesik, Jennifer Miniota, Theresa Tam et al. "Assessment of the potential for international dissemination of Ebola virus via commercial air travel during the 2014 west African outbreak." *The Lancet* 385, no. 9962 (2015): 29-35.

15. Olsen, Sonja J., Hsiao-Ling Chang, Terence Yung-Yan Cheung, Antony Fai-Yu Tang, Tamara L. Fisk, Steven Peng-Lim Ooi, Hung-Wei Kuo et al. "Transmission of the severe acute respiratory syndrome on aircraft." *New England Journal of Medicine* 349, no. 25 (2003): 2416-2422.

16. Moser, Michael R., Thomas R. Bender, Harold S. Margolis, Gary R. Noble, Alan P. Kendal, and Donald G. Ritter. "An outbreak of influenza aboard a commercial airliner." *American journal of epidemiology* 110, no. 1 (1979): 1-6.

17. Kenyon, Thomas A., Sarah E. Valway, and Ida M. Onorato. "Transmission of tuberculosis during a long airplane flight." *New England Journal of Medicine* (1996): 675-676.

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19. Widdowson, Marc-Alain, Roger Glass, Steve Monroe, R. Suzanne Beard, John W. Bateman, Perrianne Lurie, and Caroline Johnson. "Probable transmission of norovirus on an airplane." *Jama* 293, no. 15 (2005): 1855-1860.

20. Kenyon, Thomas A., Sarah E. Valway, Walter W. Ihle, Ida M. Onorato, and Kenneth G. Castro. "Transmission of multidrug-resistant Mycobacterium tuberculosis during a long airplane flight." *New England Journal of Medicine* 334, no. 15 (1996): 933-938.

transmission at the airport gate and within airplane for the transmission of Ebola and SARS infections through casual contacts.

MODEL FORMULATION

In our problem setting, we model the movement of pedestrian particles based on a force-field approach proposed by Helbing et al. (1995) which captures the actual interaction of pedestrians with their environment in real life situations.[1] While heading towards a designated destination, the behavior of an individual is influenced by his inclination to move effectively towards his targeted terminus. Stationary crowds or physical barriers obstructing the course of motion change the direction and reduce the speed of the pedestrians. In situations like boarding at an airport gate, we need to consider movement of pedestrians in a line, wherein the speed of pedestrian movement is heavily dependent upon speed of other pedestrians in front of them in a line.

Considering the self-propelled pedestrian P_i as a point mass m_i in a two dimensional space, the net resultant force $\vec{F_i}$ on the particle resulting in motion can be expressed by:

$$\vec{F_i} = \sum \vec{f_i} = \overrightarrow{f_i^{int}} + \overrightarrow{f_i^{ped}} = m_i \, \vec{a_i} \tag{1}$$

Where $\overrightarrow{f_i^{int}}$ is the intention force motivating the pedestrian to pursue his track despite the fact that a resulting opposing force $\overrightarrow{f_i^{ped}}$ is exerted by the surrounding to delay his locomotion. $\vec{a_i}$ is the acceleration vector of particle "i".

The force $\overrightarrow{f_i^{int}}$ in the motion direction $\hat{e_v}$ is the rate of change of momentum within a time interval (step) τ and is defined by:

$$\overrightarrow{f_i^{int}} = f_i^{int} \, \hat{e_v} = m_i\left(\frac{\overrightarrow{\Delta v}}{\tau}\right) = m_i \left(\frac{\overrightarrow{v_{0i}(t)} - \overrightarrow{v_i(t)}}{\tau}\right) = m_i \left(\frac{v_{0i}(t) - v_i(t)}{\tau}\right) \hat{e_v} \tag{2}$$

Here, $\overrightarrow{v_i(t)}$ designates the actual instantaneous velocity of pedestrian P_i and is characterized by its magnitude and its anticipated orientation. To predict collision avoidance, the expression of the desired velocity of navigation $\overrightarrow{v_{0i}(t)}$ depends on each individual in motion, his position in the crowd and the foremost direction of movement in the hallway of interest at every time step. Consider the cases of a walking passenger in a crowd. Before deciding the path to his

1. Helbing, Dirk, and Peter Molnar. "Social force model for pedestrian dynamics." *Physical review E* 51, no. 5 (1995): 4282.

destination, the pedestrian scans his close surrounding within a radius (cutoff distance) labeled by δ_{cutoff} and is given by:

$$\delta_{cutoff} = \eta . \delta \tag{3}$$

Where η is a positive decimal number and δ is the minimum proximity distance at which two walking individuals can come close to each other before coming to halt. The instantaneous velocity vector is evaluated based on the two following cases:

Case 1 - Line forming: The pedestrian can move in the direction of motion assigned to the hallway where he is located at. Let $\widehat{e_1}$ and $\widehat{e_v}$ denote the unit vectors of directions attributed to the hallway and the pedestrian respectively. Since the pedestrian P_i is not impeded by any obstruction, $\widehat{e_1}$ is the same as $\widehat{e_v}$ as shown in Figure 1. Therefore, his desired speed at time t, $\overrightarrow{v_{0i}(t)}$, is obtained from the relation:

$$\overrightarrow{v_{0i}(t)} = v_{0i}(t).\widehat{e_v} = v_{0i}(t).\widehat{e_1} = (v_A + \gamma_i v_B)(1 - \frac{\delta}{\|\vec{r_i} - \vec{r_j}\|}) . \widehat{e_1} \tag{4}$$

The vector positions of pedestrian P_i and the stationary traveler P_j in his way are denoted by $\vec{r_i}$ and $\vec{r_j}$ respectively, and are issued from the origin of the coordinate system of the plane of motion. $(v_A + \gamma_i v_B)$ accounts for the ultimate desired speed ranging from v_A to $(v_A + v_B)$ adjusted for the upcoming obstructions within a distance δ. γ_i is a positive random variable less than unity attributed to pedestrian "i" considering the factors that can affect his mobility such as the age, sex, body type, health condition, etc.

In particular, when the inactive traveler P_j is distant from traveler P_i in such a way that the latter's motion is not affected ($\|\vec{r_i} - \vec{r_j}\| \gg \delta$) then, equation (4) reduces to:

$$\overrightarrow{v_{0i}(t)} = v_{0i}(t).\widehat{e_v} = v_{0i}(t).\widehat{e_1} = (v_A + \gamma_i v_B).\widehat{e_1} \tag{5}$$

In addition when a pedestrian joins a line his desired velocity and thereby the intention force ($\overrightarrow{f_i^{int}}$) reduces according to equation (4).

Case 2- Collision avoidance: The pedestrian is impeded by obstructions in his desired direction of motion as shown in Figure 2. Thus, a curved deviation of the trajectory is required. Here, the walking traveler has to steer in such a way that he keeps moving forward towards his destination, indeed he makes a lateral motion to escape from the stationary members in the horde. Then, the pedestrian's desired velocity is expressed by:

$$\overrightarrow{v_{0i}(t)} = v_{0i}(t).\widehat{e_{vi}} = (v_A + \gamma_i v_B)(1 - \frac{\delta}{\min(\|\vec{r_i} - \vec{r_j}\|, j \neq i)}) . \widehat{e_{vi}} \quad (6)$$

The expression of the velocity direction $\widehat{e_{vi}}$, in unit vector notation, is written as:

$$\widehat{e_v} = cos(\alpha_i)\hat{\imath} + \sin(\alpha_i)\hat{\jmath} \quad (7)$$

And α_i is an arbitrary angle alternating between θ_1 and θ_2 and is expressed by:

$$\alpha_i = \theta_1 + \delta_i . \theta_2 \ ; 0 < \delta_i < 1 \quad (8)$$

In the course of embarkation and deplaning, we have to insure impenetrability of the particles. This is achieved by the repulsive force $\overrightarrow{f_i^{ped}}$ and is obtained from the gradient of the repulsive term in Lennard-Jones' potential as follows:

$$\overrightarrow{f_i^{ped}} = \sum_{i \neq l} \vec{\nabla} [\, \epsilon \, (\frac{\sigma}{r_{il}})^{12}] \quad (9)$$

Where ϵ and σ are repulsive force field parameters ($\epsilon = 16, \sigma = 0.86m$) and r_{il} is the distance between the ith and the lth pedestrian. The second order ordinary differential equation (1) is solved by means of Nordsieck third order predictor-corrector integration method to compute the instantaneous displacement, speed and trajectory of every particle by extrapolating these entities at the next time step.

The subsequent step involves determining the rate and extent of propagation of the viral infection among the travelers. The study of epidemics informs about how a disease propagates and what are the suitable policies to suppress or inhibit its outspread. Therefore, the Susceptible-Infected (SI) dynamic model [21] of an epidemic is employed for the purpose. We assume a population of size N consisting of I(t) infected and S(t) susceptibles at time t. A susceptible becomes infected when coming into direct contact with an infected. However, the newly infected cannot be infective during the start of the incubation period of the illness (there is no second reproduction of the illness). At a time t, N, I(t) and S(t) are related by:

$$N = I(t) + S(t) \quad (10)$$

Moreover, the infection spread initiates due to the insertion of i_c^0 infectives initially (t_0= 0) at their "c" days of infection. Thus,

$$N = \sum_{c=1}^{d} i_c^0 + S(0) \quad (11)$$

21. Keeling, Matt J., and Pejman Rohani. *Modeling infectious diseases in humans and animals*. Princeton University Press, 2008.

Where d is the extent of the illness post onset of the symptoms at day one.

Let m be the total number of contacts per individual per time step and N the total population size. Assume the presence of a single infectious individual at c days of infection. The probability that this infective meets other individuals is m/N. denote by P_c the probability that a contact between a susceptible and an infective, whose age of infection is τ days, results in infection of the susceptible. Using the axiom of conditional probability:

$$\text{P(contact and infection)} = \text{P (infection/contact) . P (contact)} = P_c \cdot \frac{m}{N} \tag{12}$$

The number of susceptibles infected by this infective is binomially distributed with parameters n= S(t-1), the number of susceptibles exposed to the contagion at time t, and p = $P_c \cdot \frac{m}{N}$. In this situation, n is large and p is very small (below 0.1). Accordingly, the Poisson distribution can be used to approximate the binomial distribution with mean λ= n.p = S(t-1). $P_c \cdot \frac{m}{N}$.

For multiple infectious individuals at time t_0=0, the number of newly infected by an ith infective at time t, a discrete variable is a Poisson probability distribution, with mean m_i (t-1). p_c .[S_i (t-1)/N]. Therefore, the number of people infected at time t by all the infectives with an age of infection "c" is Poisson distributed with a mean$\sum_{i=1}^{i_c^0}[\, m_i(\mathrm{t}-1).p_c \,.(S_i\ (\mathrm{t}-1)/\mathrm{N})]$. Summing all over the values of c, we obtain:

$$\mathrm{I(t)} \sim \text{Poisson}\ (\textstyle\sum_{c=1}^{d}(\sum_{i=1}^{i_c^0}[\, m_i(\mathrm{t}-1).p_c \,.(S_i\ (\mathrm{t}-1)/\mathrm{N})]\,)) \tag{13}$$

Where m_i is the number of contact of susceptibles with the ith infectious traveler and p_c the infection transmission probability.

RESULTS AND DISCUSSION

During an epidemic outbreak, the prevalence of the disease in a large population relies on the ability of a pathogen to establish unrestrained reproductive infections. Consequently, disease control, suppression or prevention starts by determining the core of its initiation as well as the incidence, medium, range and probability of propagation. During the progression of illness, the variation of antigens in the blood serum can be captured, and it determines the severity of the patient's situation. In this study, we refer to observations of the evolution of the antibodies since the onset of the symptoms till recovery to generate what is referred as infectivity profile. The probability of infection

(p_c) has a major influence on the findings as it determines the total of newly infected passengers who were exposed to the contamination within a suitable environment of propagation. The simulations to this problematic have been conducted for both Ebola and SARS since these contagions were previously encountered in air travel. For Ebola, the infectivity profile in Figure 4 is acquired by the amount of RNA virus copies above the detection threshold in the blood serum.[22] The daily logarithmic amounts of RNA for fatal and non-fatal contagion are averaged along the 21 days of illness period, then divided by the total to obtain the probability of infection at a designated day. Also, the viral gene expression of the nucleocapsid (N) protein, detected at different rates along the evolution of SARS is indicative of the possibility of transmission (Figure 5).[23] This infectivity data is combined with the number of contacts between pedestrians generated using the pedestrian moment model to assess the extent of disease propagation among the travelers onboard.

The time evolution of pedestrian trajectories has been displayed for both ingress from a gate (Figure 6) and egress from an Airbus A320 carrier (Figure 7) respectively for comparison of outputs. During the enplaning, the trajectories of passengers, initially seated or standing in the departure lounge, heading to the passenger boarding bridge (PBB) and finding their assigned onboard seats, are modeled. In both scenarios, the instantaneous position and speed of each walking individual are obtained by solving equation (1) by means of a predictor-corrector numerical method. Many qualitative features of pedestrian movement are captured by the model. For instance, lane formation is observed in the hallways, in addition to reduced speed at bottlenecks where passengers from different seating zones merge and head to the airplane (Figure 6). Similar features are observed in egress when passengers walk out of their seats toward the aisle (Figure 7).

In real life the identity of infectious individual is not known beforehand, therefore all the possible permutations of a single infective are run to estimate the mean of newly infected susceptibles denoted by λ_i where i ranges from 1 to the total passenger capacity of the aircraft. Due to the stochastic nature of the problem, we assume

22. Towner, Jonathan S., Pierre E. Rollin, Daniel G. Bausch, Anthony Sanchez, Sharon M. Crary, Martin Vincent, William F. Lee et al. "Rapid diagnosis of Ebola hemorrhagic fever by reverse transcription-PCR in an outbreak setting and assessment of patient viral load as a predictor of outcome." *Journal of virology* 78, no. 8 (2004): 4330-4341.

23. Zhao, Guo-ping. "SARS molecular epidemiology: a Chinese fairy tale of controlling an emerging zoonotic disease in the genomics era." *Philosophical Transactions of the Royal Society of London B: Biological Sciences* 362, no. 1482 (2007): 1063-1081.

that the number of newly infected travelers by a single infectious chosen randomly among the airplane passengers is Poisson distributed with mean λ_i at every simulation. After performing all the simulations in parallel, the effective probability of means is calculated. Then, using the Bayes' theorem the probabilities are combined to generate the probability distributions in Figures (8, 9, 10 and 11).

These plots (Figures 8-11) represent the probabilistic distribution of infected passengers who were closely exposed to Ebola and SARS viruses. These viral organisms are transmitted through direct contact or dispersion of particles exhaled from an infectious member by talking, coughing or sneezing, and remain sustained in the environment for a certain time before depositing and contaminating contiguous surfaces.[24,25] Mangili and Gendreau (2005) indicate large droplet and airborne mechanisms are possibly highest risk transmission mechanisms during air travel.[26] The transmission distance also depends on specific disease, for example, SARS has been transmitted by short range droplet based as well as longer range airborne mechanisms.[27,28] Primary mode of transmission for Ebola is through contact droplets [29], but studies with monkeys indicate possible transfer through aerosols [24,30]. The size of these particles as well as the environmental condition play an important role in contagion dispersion. Small particles dispersed in aerosols transmit over large distances, for example, experiments indicate micrometer sized aerosol clouds

24. Jones, R. M., and L. M. Brosseau. "Ebola virus transmission via contact and aerosol—a new paradigm." *Center for Infectious Disease Research and Policy, University of Minnesota, Minneapolis, MN. http://www. cidrap. umn. edu/news-perspective/2014/11/commentary-ebolavirus-transmission-contact-and-aerosol-new-paradigm* (2014).

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26. Mangili, Alexandra, and Mark A. Gendreau. "Transmission of infectious diseases during commercial air travel." *The Lancet* 365, no. 9463 (2005): 989-996.

27. Clark, Raymond P., and Mervyn L. de Calcina-Goff. "Some aspects of the airborne transmission of infection." *Journal of the Royal Society Interface* 6, no. Suppl 6 (2009): S767-S782.

28. Li, R. W. K., K. W. C. Leung, F. C. S. Sun, and L. P. Samaranayake. "Severe Acute Respiratory Syndrome (SARS) and the GDP. Part I: Epidemiology, virology, pathology and general health issues." *British dental journal* 197, no. 2 (2004): 77-80.

29. Centers for Disease Control and Prevention. "Review of human-to-human transmission of Ebola virus." *Atlanta, GA: CDC* (2014).

30. Jaax, N., P. Jahrling, Thomas Geisbert, J. Geisbert, K. Steele, K. McKee, D. Nagley, E. Johnson, G. Jaax, and C. Peters. "Transmission of Ebola virus (Zaire strain) to uninfected control monkeys in a biocontainment laboratory." *The Lancet* 346, no. 8991 (1995): 1669-1671.

generated during cough traveling over 2 m.[31,32] Smaller aerosols can be driven farther by ventilation or a freestream flowing from a high static pressure location to a lower pressure zone.[33] Based on primary modes of transmission, coarse droplets for Ebola and aerosol for SARS we assume a radius of infection of 1.2m (48 in) for Ebola and 2.1m (84 in) for SARS. Note that infectivity profile for both viruses are quite close in values and less than 0.1, the selection of radii of infection makes a noticeable difference in the number of contacts and transmission. In Figure 8, we consider an infectious passenger at his first day is onboard among the susceptible population. Ebola records a peak of 2 newly infected passengers exposed to the virus, whereas this number increases to 5 for SARS due to the wider range of infectivity. Shifting the infectivity to its highest (day 3 for Ebola and day 4 or 5 for SARS) in Figure 9, the means of the Poisson distribution increases by one unit for both plots compared to those of Figure 8.

Results for deplaning under similar conditions is shown in Figures 10 and 11. It can be noticed that the distribution of newly infected individuals behaves in the same way as that of Figures 8 and 9. However, the mean number of infected reduces to 1 and 2 respectively for Ebola and SARS. Egress phase is of a shorter period of time compared to boarding, therefore there are fewer contacts and lower number of infected.

The objective of the pedestrian movement methodology is to mimic the actual comportment of pedestrians in an airport terminal. In this paper, we simulated a boarding process at the departure lounge and deplaning from airplane. The study can be expanded further to include the motion of pedestrians at the moment of arrival, passing by check-in, security and boarding, while accounting for uncertainty due to discretionary activities prior to boarding. Such simulations can be used to suggest effective travel strategies to suppress infection spread for contact based diseases. In addition, the multiscale model is general and can be applied to any directly transmitted disease and can be used to develop policies that mitigate infection spread under different conditions.

31. Bourouiba, Lydia, Eline Dehandschoewercker, and John WM Bush. "Violent expiratory events: on coughing and sneezing." *Journal of Fluid Mechanics* 745 (2014): 537-563.

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SUMMARY

A mathematical method is formulated to characterize infection spread at airport gates and in airplanes combining the pedestrian movement and stochastic infection dynamics models. Using the model, we studied the infection transmission for two pathological contagions, Ebola and SARS during airplane boarding and deplaning. We discuss the effect of infection radius and transmission mechanism on the spread of infection.

Acknowledgements

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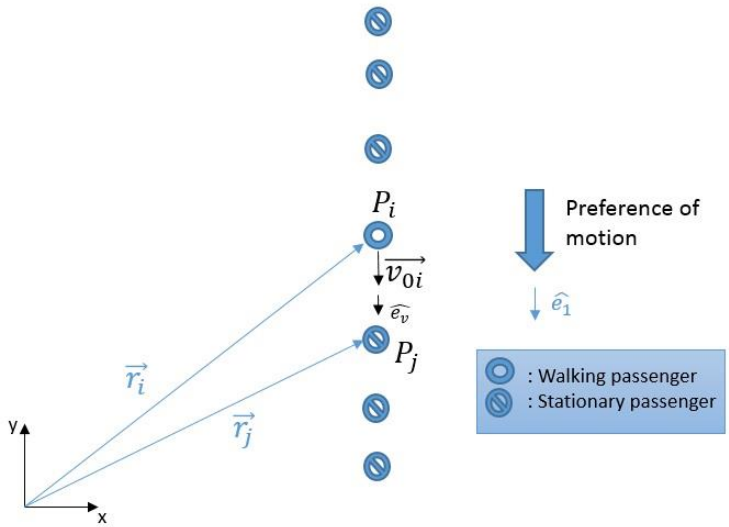

Figure 1. The distribution of the stationary crowd around the traveler P_i walking in the same direction of preference of the hallway.

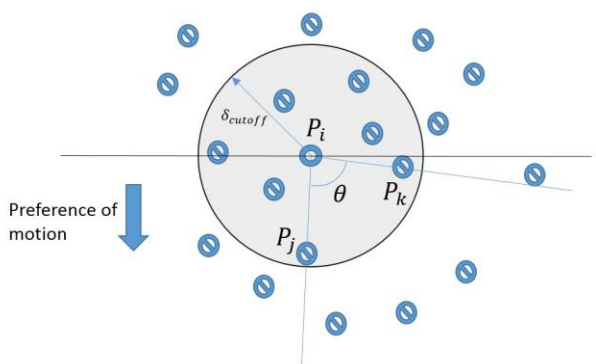

Figure 2. The distribution of the stationary crowd obstructing the traveler P_i from walking in the direction of preference of the hallway.

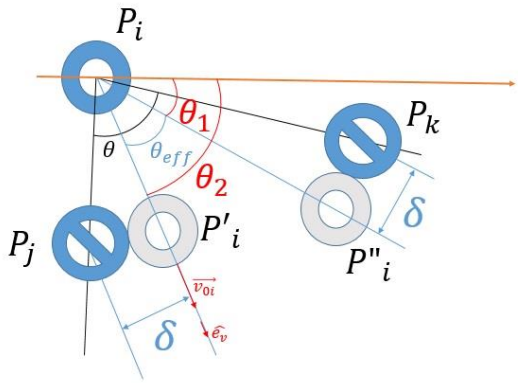

Figure 3. The deviation in direction of the desired velocity of pedestrian P_i in such a way to escape from the crowd between particles "j" and "k".

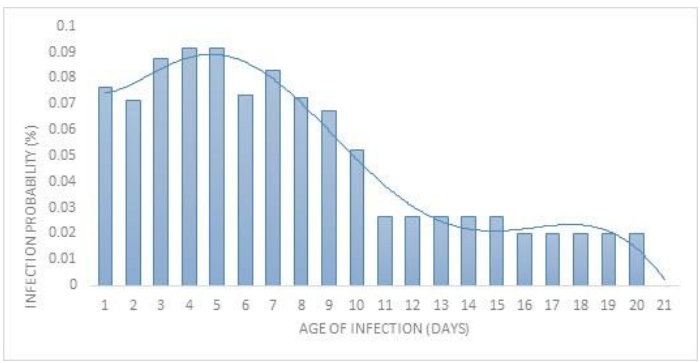

Figure 4. Infectivity profile along the days after inception of symptoms for SARS contagion

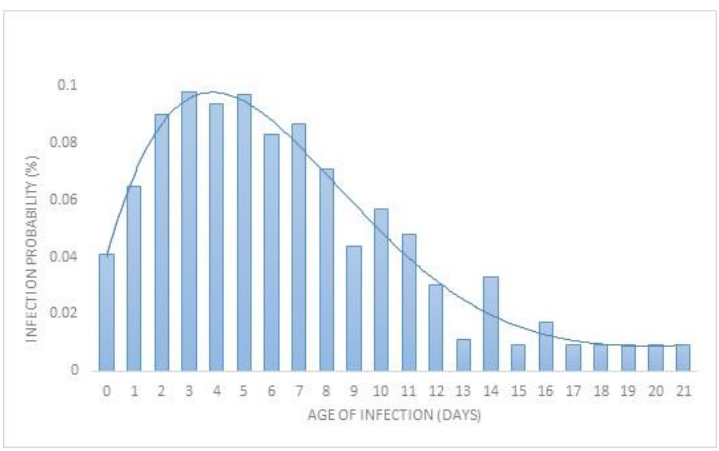

Figure 5. Infection probability distribution versus the days after onset of symptoms for Ebola virus

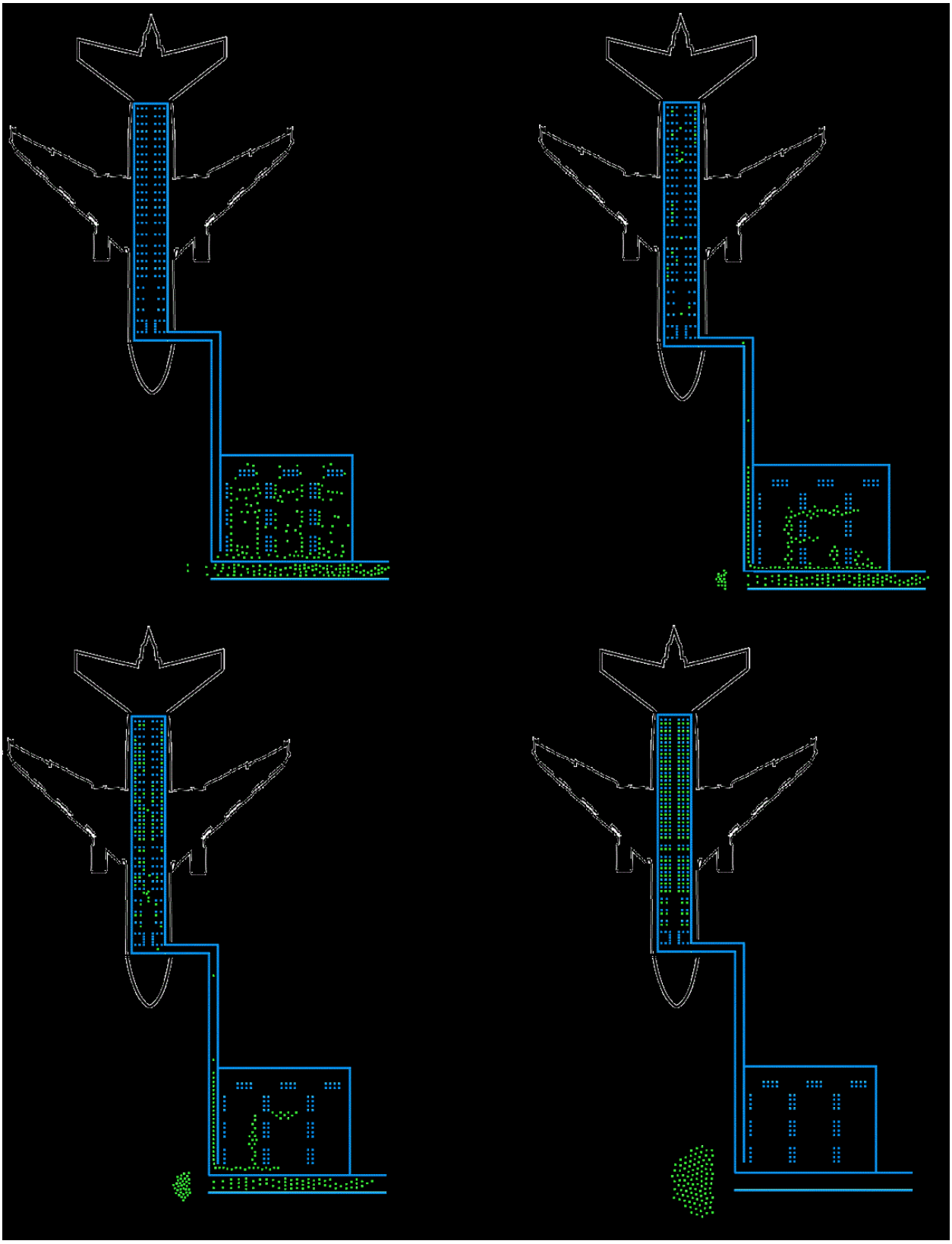

Figure 6. Simulation snapshot of an embarkation of an Airbus A320 from a departure lounge at different time steps

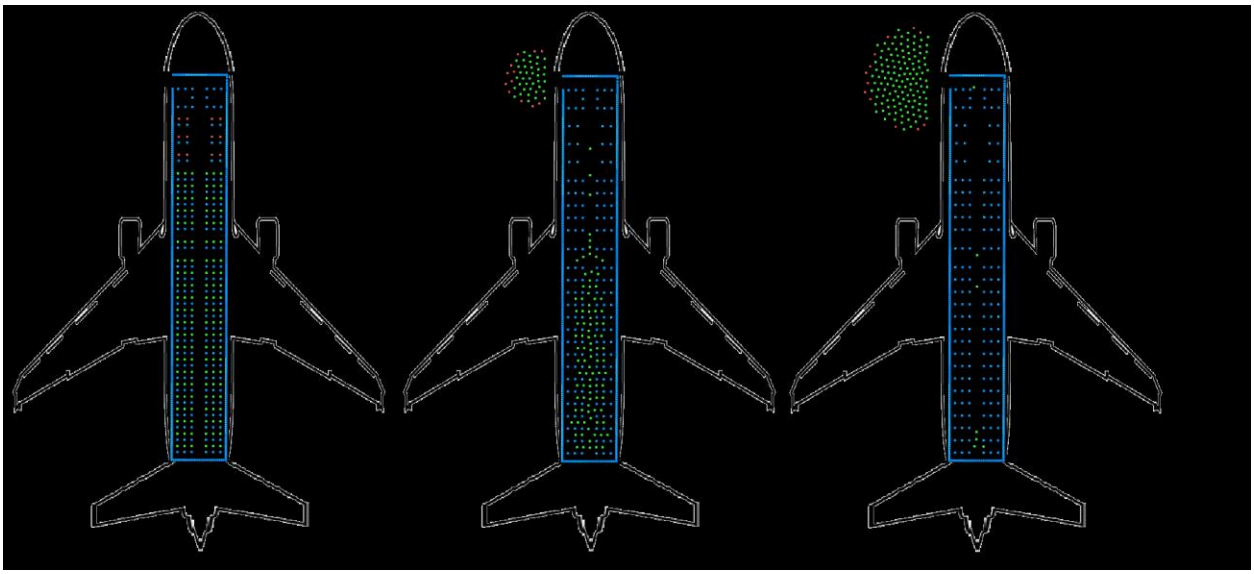

Figure 7. Simulation snapshot of Airbus A320 deplaning at different time steps

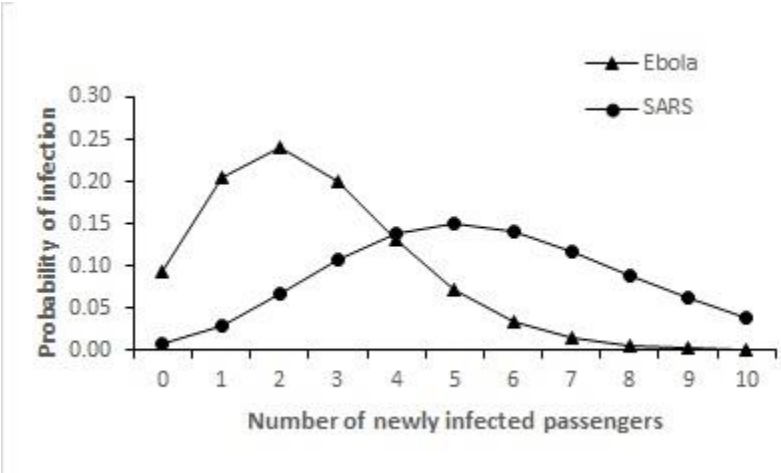

Figure 8. Infection profile at the first day post onset of symptoms during a random ingress for Ebola and SARS contagions

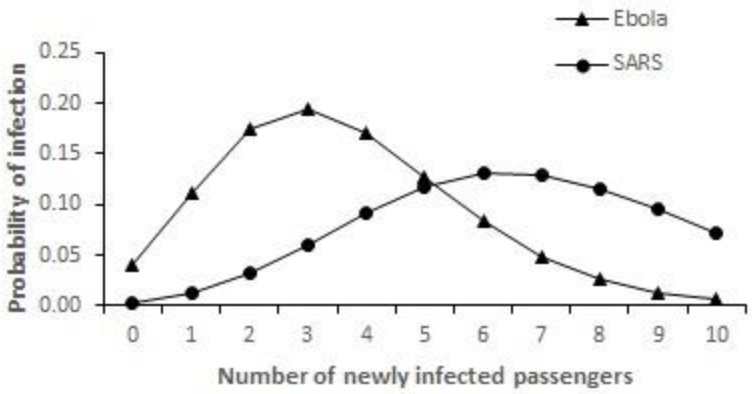

Figure 9. Infection profile at the peak day post onset of symptoms during a random ingress for Ebola and SARS contagions

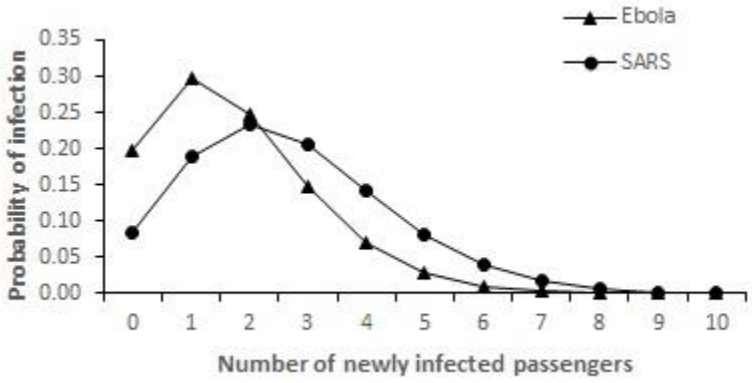

Figure 10. Infection profile at the first day post onset of symptoms during deplaning from an Airbus A320 for Ebola and SARS contagions

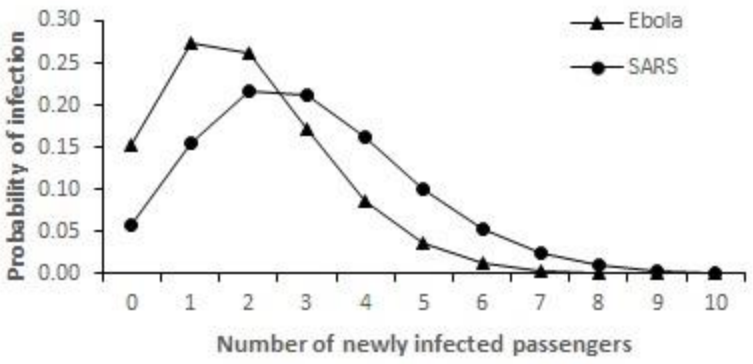

Figure 11. Infection profile at the peak day post onset of symptoms during deplaning from an Airbus A320 for Ebola and SARS contagions