Computational Model for Pedestrian Movement and Infectious Diseases Spread During Air Travel

Pierrot Derjany, Sirish Namilae*
Embry-Riddle Aeronautical University, Daytona Beach, Florida

Anuj Mubayi
Arizona State University, Tempe, Arizona,

and
Ashok Srinivasan
Florida State University, Tallahassee, Florida

This paper presents an integrated computational framework combining a Molecular Dynamics (MD) based social force pedestrian movement model and a stochastic infection dynamics model to evaluate the spread of viral infectious diseases during air-transportation. We apply the multiscale model for three infectious (1) Ebola (2) Influenza (H1N1 strain) and (3) SARS pathogens with different transmission mechanisms and compare the pattern of propagation during an Airbus A320 carrier boarding and deplaning at an airport gate. The objective of this analysis is to assess the influence of pedestrian movement on infection spread during air travel.

I. Introduction

Air transportation medium and facilities are evolving exponentially to meet the necessity of connection, exchange and travel in an increasingly interconnected world. Despite its many benefits, commercial air travel enables rapid spread of infectious diseases across the globe [1-6]. Travelers are in close proximity to each other and are susceptible to infection transmission during different phases of air travel. Pedestrian movement within an airport is key to understanding and estimating the casual contacts between passengers and is a special case of a more general contact analysis. Modeling the scene at an airport provides sufficient useful insight into disease propagation.

Pedestrian motion has been addressed using several approaches such as particle dynamics or social force models [7,8], models based on cellular automata [9], fluid flow models [10], and queuing based models [11]. Of these different approaches, the social force model has superior advantages over the other methods to evaluate passenger movement and interaction because each passenger is modeled individually and moves continuously which enables computing the individual trajectories and contact patterns between pedestrians. These models utilize the same numerical framework as molecular dynamics simulations in materials science [12].

Several studies use these generic approaches to study the travelers movement especially from the viewpoint of airport operations and reduction of the turnaround time of airplanes at terminals. Schultz et al. [13] mimic the intuitive behavior of travelers under emergency situation by a cellular automaton model. In this model, the floor area of the airport is subdivided into small discrete partitions where pedestrians may switch positions to neighboring spots based on a probabilistic distribution [13]. Several other investigators also used agent-based models to simulate pedestrian motion in airport terminals [e.g. 14]. Other studies such as that by Lin et al. [15] investigate the flow of pedestrians to their destinations by optimizing the guiding signs.

Pedestrian movement in airports is peculiar because it involves a series of nondiscretionary as well as discretionary activities. For instance, 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 [16,17]. 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 [15,18]. 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 the variation in immunity, ethnic background, and intervention usage across geographic areas. Consequently, airports and airplanes are potential, prime sites for infection spread [19]. During the Ebola epidemic in

* Assistant Professor Aerospace Engineering, Corresponding Author – Email: namilaes@erau.edu

2014, analyses demonstrate 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 [20]. 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 [21]. Several other diseases like tuberculosis, norovirus etc, have been transmitted through air travel [22-25]. 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 [26]. The first recorded outbreak of Influenza aboard an aircraft occurred back in 1979 when passengers were sharing the same cabin along three hours with a broken ventilation system with an Influenza A strain infectious traveler. Within three days, 72% of the 54 passengers contracted the disease [22].

Three main factors are known to influence the infection spread: the contagion stage of the index infectious, the flight duration and the frequency of contacts between passengers within the critical radius of infection [26]. The environmental conditions such as the ventilation [28] as well as the temperature and humadity [29] can also play an important role in the virus lifetime survival. The number of contacts is critically dependent on the pedestrian flow within airplanes and in 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 our analysis, we have used social force based pedestrian dynamics formulation to estimate the number of contacts and evaluate the disease spread in an airplane and airport gate. We extend the social force model for pedestrian movement incorporating line forming and collision avoidance phenomena. In earlier studies, we have implemented a parameter sweep on massive parallel computers to determine the model parameters ranges [27, 30]. We have integrated the proposed formulation with a stochastic susceptible-Infected infection transmission model [31]. In this paper, the model is applied to study the infection transmission at the airport gate and within airplane for the transmission of Ebola, SARS and H1N1 Influenza pathogens through casual contacts.

II. Model Formulation

In our problem setting, we model the movement of pedestrian particles based on a force-field approach proposed by Helbing et al. [7] which captures the actual interaction of pedestrians with their environment in real life situations. 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 alter the direction and diminish the speed of the pedestrians. In situations like boarding at an airport gate, we need to consider the movement of pedestrians in a line, wherein the speed of the pedestrian in motion is heavily dependent upon speed of other pedestrians in front of them in a queue.

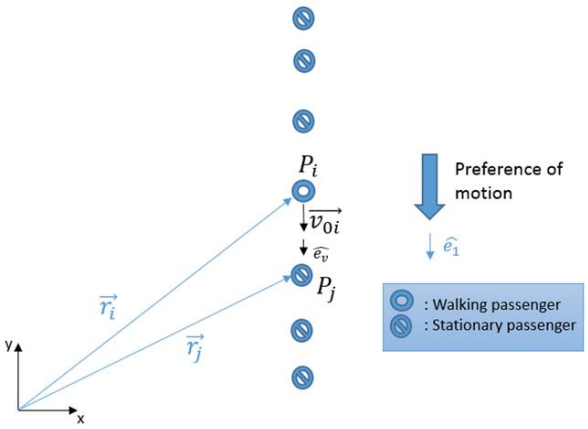


Figure 1. The distribution of the stationary crowd around the traveler Pi walking in the same direction of preference of the hallway.

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:

$$\vec{F_i} = \sum \vec{f_i} = \overrightarrow{f_i^{int}} + \overrightarrow{f_i^{ped}} = m_i \vec{a_i} \quad (1)$$

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 $\widehat{e_v}$ is the rate of change of momentum within a time interval (step) τ and is defined by:

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

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. 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, when a pedestrian joins a line his desired velocity and thereby the intention force $\overrightarrow{f_i^{int}}$ reduces according to the relation:

$$\overrightarrow{v_{0i}(t)} = v_{0i}(t).\widehat{e_v} = v_{0i}(t).\widehat{e_1} = (v_A + \gamma_i v_B)(1 - \frac{\delta}{\|\vec{r_i} - \vec{r_j}\|}) . \widehat{e_1} \quad (3)$$

The vector positions of pedestrian P_i and the adjacent forward 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 desired speed 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 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 (3) reduces to:

$$\overrightarrow{v_{0i}(t)} = v_{0i}(t).\widehat{e_v} = v_{0i}(t).\widehat{e_1} = (v_A + \gamma_i v_B).\widehat{e_1} \quad (4)$$

In the course of embarkation and deplaning, we have to insure impenetrability (collision avoidance) 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:

$$\overrightarrow{f_i^{ped}} = \sum_{i \neq l} \vec{\nabla} [\, \epsilon \, (\frac{\sigma}{r_{il}})^{12}] \quad (5)$$

Where ϵ and σ are repulsive force field parameters and r_{il} is the distance between the i^{th} and the l^{th} 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 extent of the viral infection propagation among the travelers. Viral infection spread is often characterized by the reproduction number, the total number of infected an infectious member reproduces in given time. Statistical-mathematical models have been used to predict the evolution of infectious diseases studied here. For instance, in different case studies about H1N1 outbreak, in 2009, in Israel [32] and Italy [33], time-dependent epidemic transmission models have been developed to obtain the reproduction number (R_0) of the virus. R_0 has also been estimated for Ebola virus during its outbreak in Africa in 2014 [34]. An uncertainty and sensitivity analysis of critical model parameters affecting R_0 has been conductedfor 2002-2003 SARS outbreak of, [35].

We use the Susceptible-Infected (SI) individual dynamic model [36] to estimate the number of infection spread during air travel. We assume a population of size N consisting of I(t) infected and S(t) susceptibles at time t. Thus,

$$N = I(t) + S(t) \quad (6)$$

A susceptible becomes infected when coming into direct contact with an infective. However, the newly infected cannot be infective at the start of the incubation period of the illness during air travel, therefore there is no second reproduction of the illness. Moreover, the infection spread initiates due to the insertion of i_c^0 infectives initially (t_0= 0) at their "c" days of infection and d is the extent of the illness post onset of the symptoms. Let "m_i" be the total number of contacts per infective individual "i" per time step and "N" the total population size. The probability that an infectious in the crowd meets other individuals is m_i/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 (infection transmission probability). In order to account for the demographic stochasticity and variations in susceptibility of the population, the number of newly infected individuals by an i^{th} infective at time t, a discrete variable, is Poisson distributed, with a 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 obtained by:

$$I(t) \sim \text{Poisson} \left(\sum_{c=1}^{d} \left(\sum_{i=1}^{i_c^0} [\, m_i(t-1).p_c \,.(S_i\,(t-1)/N)]\right)\right) \quad (7)$$

III. Results and Discussion

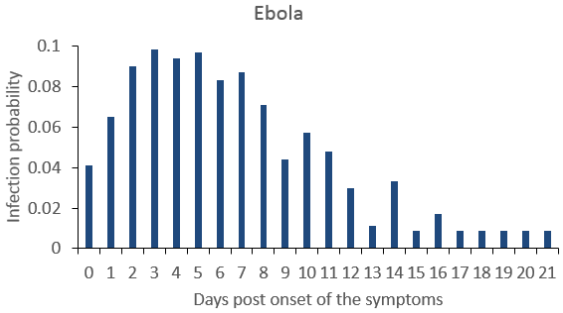


Figure 2. Infectivity profile along the days after clinical signs of Ebola infection.

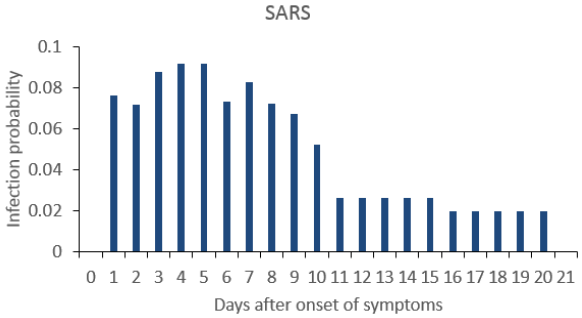


Figure 3. Infectivity profile along the days post onset of SARS symptoms.

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 within the incubation period of the virus 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 for propagation. We carry out simulations for Ebola, SARS and H1N1 Influenza viruses since these contagions were previously encountered in air travel. For Ebola, the infectivity profile is acquired by the amount of RNA (ribonucleic acid) virus copies above the detection threshold in the blood serum since the illness contraction [37]. 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 (Figure 2). For SARS pathogen, the viral gene expression of the nucleocapsid (N) protein (Figure 3), detected at different rates along the evolution of the virus from post onset of the symptoms till convalescence is indicative of the possibility of transmission [38]. For, Influenza H1N1, sometimes the viral shedding and RNA are not detectable (especially until 5-6 days of onset of symptoms) in positively tested patients [39]. The contraction of the influenza virus is also replicated in mamals. For instance, experimental investigations are conducted on pigs [40,41], mice [42] and ferrets [43] for a better observation and understanding of the virus. The H1N1 nasal, oral or ocular shedding has been detected by determining the relative equivalent unit (REU) from viral RNA level [44]. In our model, we assume that the transmission of Influenza disease occurs through aerosols expelled during coughing, sneezing or talking thus via nasal route. Therefore, the infectivity profile for H1N1 virus is obtained from measuring the evolution rate of REU in saliva from the first day of disease contraction. The infectivity profile is shown in Figure 4. The infectivity data for the three viruses under investigation is, then, combined with the number of contacts between pedestrians generated using the pedestrian movement 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 5) and egress from an Airbus A320 carrier (Figure 6) 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 and finding their assigned onboard seats, are modeled. In both scenarios, the instantaneous position and speed of each walking individual are obtained from solving equation (1) using a predictor-corrector numerical integration. 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 5). Similar features are observed in egress when passengers walk out of their seats toward the aisle (Figure 6).

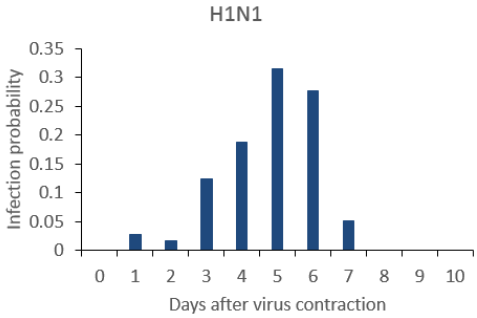


Figure 4. Infectivity profile along the days post infection with H1N1 virus.

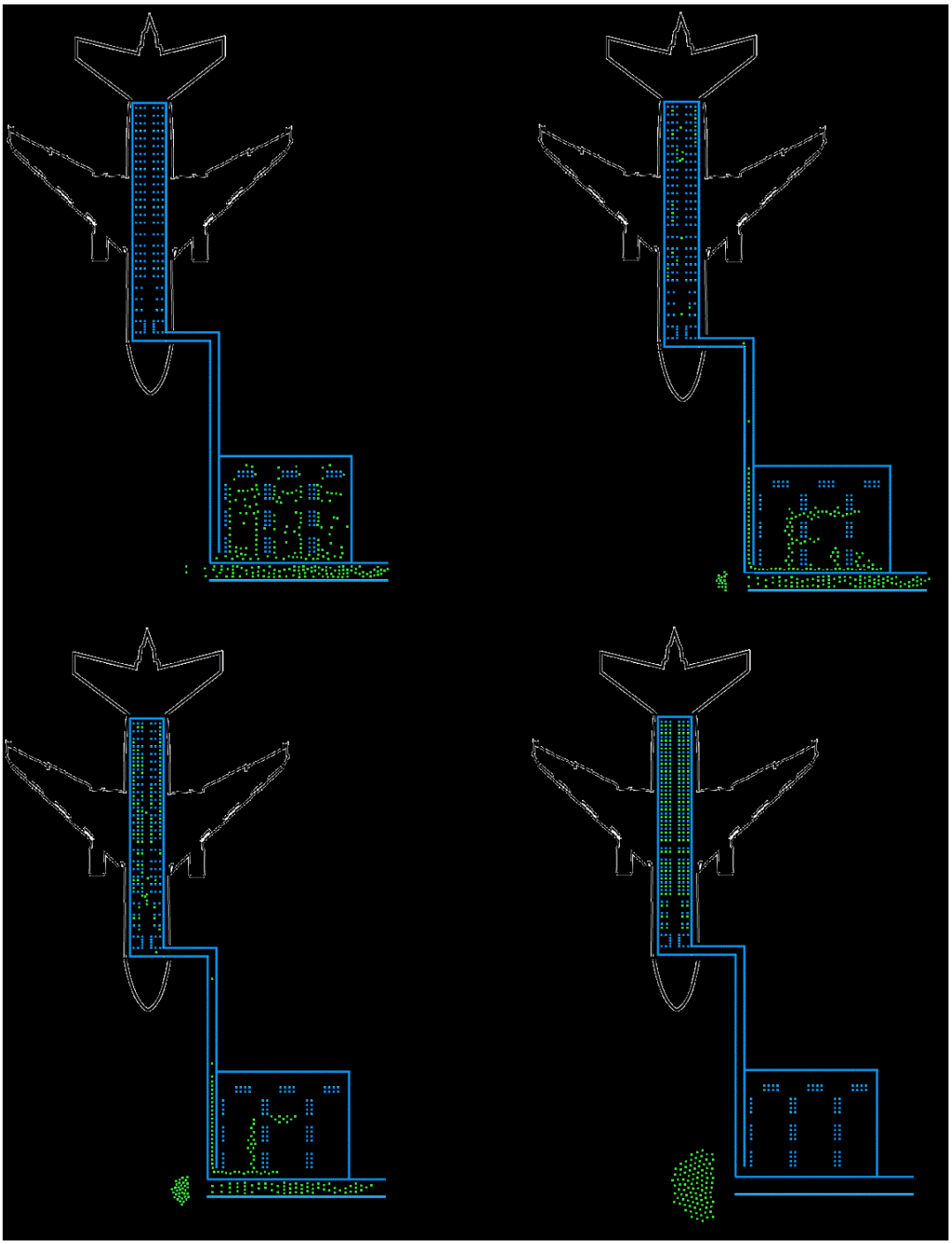


Figure 5. Simulation snapshot of an embarkation of an Airbus A320 from a departure lounge at different time steps.

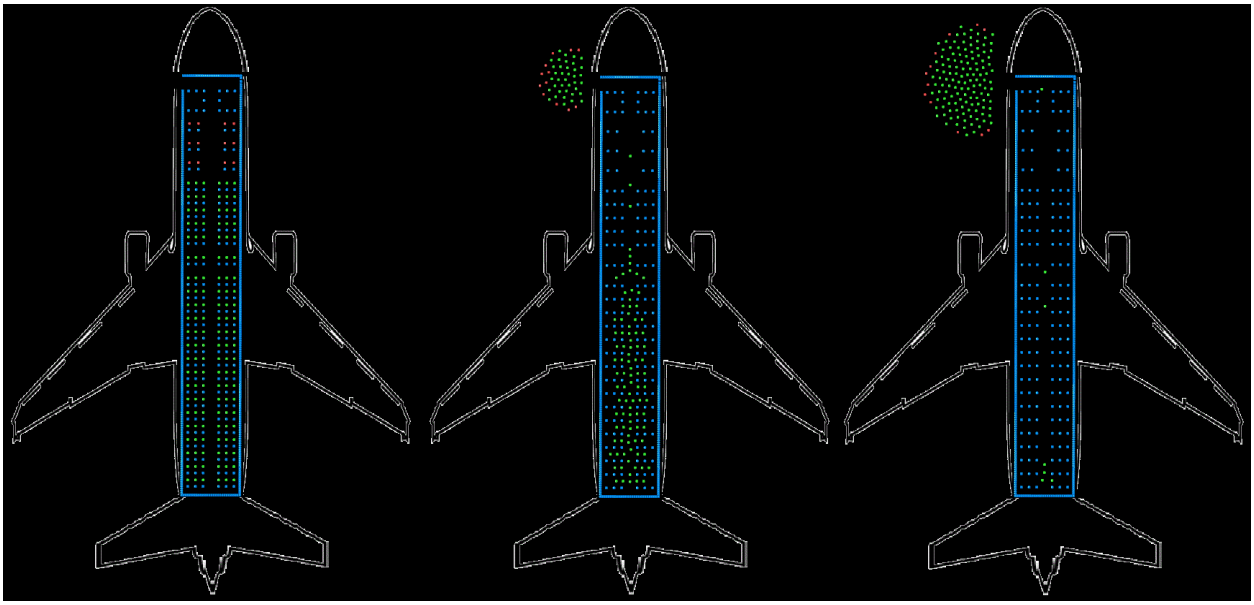


Figure 6. Simulation snapshot of Airbus A320 deplaning at different time steps.

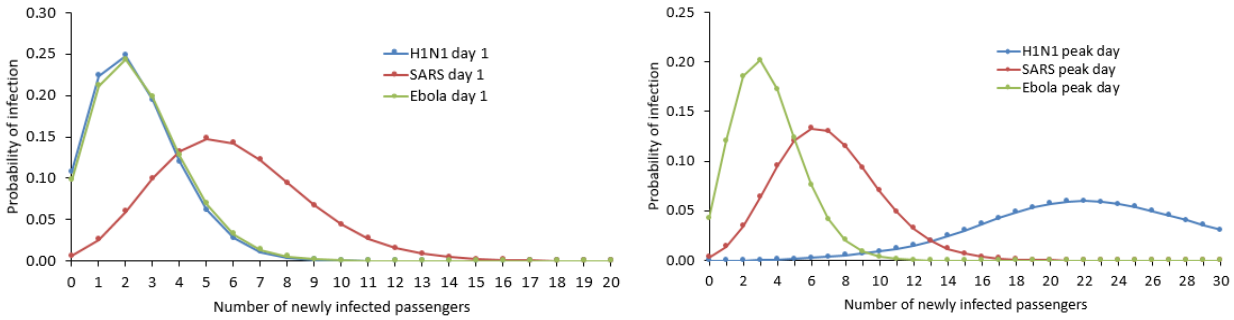
The identity of infectious individual is not known beforehand, therefore all the possible permutations of a single infective are run to estimate the mean of newly infected susceptibles denoted by λ_i where "i" ranges from 1 to the total passenger capacity of the aircraft. Due to the stochastic nature of the problem, we assume 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 shown in Figures 7 and 8.

These plots (Figures 7 and 8) represent the probabilistic distribution of infected passengers who were closely exposed to Ebola, Infulenza H1N1 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 [45,46]. Mangili and Gendreau [47] indicate large droplet and airborne mechanisms are possibly highest risk transmission mechanisms during air travel. 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 [48,49]. Primary mode of transmission for Ebola is through contact droplets [50], but studies with monkeys indicate possible transfer through aerosols [45,51]. Likewise, the influenza virus may be transmitted through coarse droplets or microscale bioaerosols being respired into the respiratory tract of a susceptible member [52]. There's a debate on the nature of transmission of Influenza virus. Wong and Yuen (2006) suggest that transmission occurs when the virus particles are suspended in air and inhaled by a susceptible individual or when that individual touches a contaminated surface with deposited droplets and then touches their eyes, nose or mouth [52].

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 generated during cough traveling over 2 m [53,54]. Smaller aerosols can be driven farther by ventilation or a freestream flowing from a high static pressure location to a lower pressure zone [55, 33]. Based on primary modes of transmission, coarse droplets for Ebola and aerosol for SARS and H1N1, we assume radii of infection of 1.2m (48 in) and 2.1m (84 in) respectively. Note that the infectivity profiles for both Ebola and SARS are quite close in values and less than 0.1, so the selection of radii of infection makes a noticeable difference in the number of contacts and transmission. For Influenza virus, the infectivity is at a higher rate compared to Ebola and SARS. This can be reflected by the reproduction number R_0. For instance, an infectious agent with SARS can reproduce 2-3 newly infected indivuduals, but this range increases considerably to an upper limit of 20 for H1N1 [55]. In Figure 7, we consider an

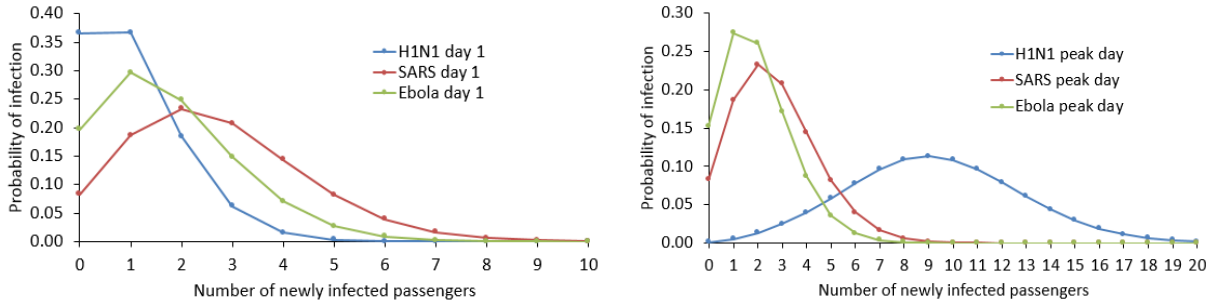
infectious passenger at his first day is onboard among the susceptible population. Ebola and H1N1 record a peak of 2 newly infected passengers exposed to the virus, whereas this number increases to 5 for SARS due to the wider range of infectivity. Shifting the infectivity to its highest (day 3 for Ebola, day 5 for H1N1 and day 4 or 5 for SARS), the means of the Poisson distribution increases by one unit for Ebola and SARS but expands tremendously for H1N1 since the infectivity reaches its peak of 30% at the fifth day of H1N1 infection.

We followed a similar approach for the deplaning strategies. We found that deplaning had a smaller impact on infection dynamics because of the lower number of new contacts and lower time of exposure during the comparatively faster process. From the results for deplaning under similar conditions, shown in Figure 8, It can be noticed that the distribution of newly infected individuals behaves in the same way as that of Figure 7. However, the mean number of infected reduces to 1 for H1N1 and Ebola and to 2 to SARS at day 1. Egress phase is of a shorter period of time compared to boarding, therefore, there are fewer contacts and lower number of infected.



(a) (b)

Figure 7. Infection profile at (a) the first and (b) peak days respectively post onset of symptoms during a random ingress to an Airbus A320 (144pax) for Ebola, Influenza H1N1 and SARS contagions.



(a) (b)

Figure 8. Infection profile at (a) the first and (b) peak days post onset of symptoms during deplaning from an Airbus A320 for Ebola, Influenza H1N1 and SARS contagions.

IV. Summary

In this paper, we simulated a boarding process at the departure lounge and deplaning from the airbus A320 airplane. The objective of the pedestrian movement methodology is to mimic the actual comportment of pedestrians in an airport terminal. We then evaluated the infectious disease spread for different viral contagions such as Ebola, SARS and Influenza H1N1. The study can be expanded to include different pedestrian movement patterns and air travel policies to determine if there is a reduction in infection spread by using specific boarding policies (e.g. reducing gate waiting time vs. spreading out passengers), while accounting for uncertainty due to discretionary activities prior to enplaning.

V. Acknowledgment

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