Building and Environment 59 (2013) 436–447

Contents lists available at SciVerse ScienceDirect

Building and Environment

journal homepage: www.elsevier.com/locate/buildenv

Bioaerosol deposition in single and two-bed hospital rooms: A numerical and experimental study

M.-F. King*, C.J. Noakes, P.A. Sleigh, M.A. Camargo-Valero

Pathogen Control Engineering Institute, School of Civil Engineering, University of Leeds, Leeds LS2 9JT, UK

ARTICLE INFO

Article history:
Received 6 June 2012
Received in revised form
8 September 2012
Accepted 16 September 2012

Keywords:
Hospital acquired infection
Particle deposition
CFD
Turbulence models
Experiment
Bioaerosol

ABSTRACT

Aerial dispersion of pathogenic microorganisms and subsequent contamination of surfaces is well recognised as a potential transmission route for hospital acquired infection. Simulation approaches such as computational fluid dynamics (CFD) are increasingly used to model particle behaviour in indoor air and the results interpreted to infer infection risk. However there is little validation of such methods in the open literature. This paper considers the ability of CFD simulations to accurately predict spatial distributions of bioaerosol deposition in indoor environments and explores the influence that different room layouts have on deposition patterns. Spatial deposition of aerosolised *Staphylococcus aureus* was measured in an aerobiology test room arranged in three different layouts: an empty room, a single-bed and a two-bed hospital room. Comparison with CFD simulations using Lagrangian particle tracking demonstrates that a realistic prediction of spatial deposition is feasible, and that a Reynolds Stress (RSM) turbulence model yields significantly better results than the $k-\varepsilon$ RNG turbulence model used in most indoor air simulations. Results for all layouts demonstrate that small particle bioaerosols are deposited throughout a room with no clear correlation between relative surface concentration and distance from the source. However, a physical partition separating patients is shown to be effective at reducing cross-contamination of neighbouring patient zones.

1. Introduction

The risk of acquiring nosocomial infections is omnipresent in health-care facilities worldwide. Globally it is estimated that 1.4 million people are suffering from such an affliction at any one time [1]. In the USA for example, the National Nosocomial Infections Surveillance (NNIS) calculated that approximately 1.7 million patients were infected by Healthcare Associated Infections (HCAIs) and 99,000 attributable deaths were reported in 2002 [2]. The European counterpart (Hospital in Europe Link for Infection Control through Surveillance – HELICS) considers the figure of affected patients to be around 5 million in Europe [1]. Differences in benchmarking of surveillance data often make comparisons difficult on an international level however the significance of the problem is undisputed. While the transmission routes for some diseases are well documented, the precise mode of transmission is uncertain for many infections, particularly for those pathogens that cause HCAIs. Although it is highly likely that the majority of transmission occurs via a contact route [1], there is evidence suggesting that at least 20% of HCAIs potentially could have arisen from an environmental reservoir [3].

Deposition of pathogen laden bioaerosols has been highlighted as a potential mechanism for such environmental contamination. Several recent studies have demonstrated a strong correlation between hospital airborne microflora and contaminated surfaces [4,5]. Complementary studies have shown that the application of air cleaning technologies can reduce surface contamination [6]; while others have highlighted that environmental contamination, through the deposition on surfaces, cannot be underestimated in the contribution to fomite-based transmission [7–9]. That said, the fate of aerial pathogens in indoor environments is still poorly understood and constitutes an area of much controversy and challenging research. Conventional infection theory regards bioaerosol particles with a diameter below 5 μm (e.g., droplet nuclei) as remaining airborne and being controlled by ventilation, while particles with larger diameters (e.g., larger droplets from a sneeze, skin squama, etc) are cited as depositing out of the air within a 2 m radius of the source [10,11]. However the reality is not quite so clear cut. Smaller particles, while remaining airborne for longer, may still deposit out onto surfaces creating a possible contact transmission risk. While very large particles (>100 μm) will clearly deposit quickly, mid-range (5–100 μm) particles will be influenced by the air, initially

* Corresponding author. Tel.: +44 113 343 3292; fax: +44 113 343 2265.
E-mail address: bs07mfk@leeds.ac.uk (M.-F. King).

0360-1323/$ – see front matter © 2012 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.buildenv.2012.09.011

through evaporation and then subsequently by ventilation flow patterns [12,13]. As a result, the final destination of an airborne pathogen may be many metres away from its original source.

Understanding the role that ventilation airflow and ward design play in the dispersion and deposition of infectious bioaerosols is tantamount to assessing pathogen exposure risk. With the difficulties in aerosolising microorganisms in most experimental settings, many studies have turned to inert particle tracers [14,15] or computational fluid dynamics (CFD) models to infer bioaerosol behaviour in air and deposition onto surfaces [16]. As highlighted by Hathway et al. [17] direct comparison between CFD models and bioaerosol experiments is sparse. Wong et al. [18] undertook a small scale experimental/numerical comparison using bioaerosol deposition within a climatically controlled enclosure. They showed good comparison using the RNG $k-\varepsilon$ turbulence model and their results are encouraging at high grid densities, despite the many reservations held regarding eddy viscosity turbulence modelling. Hathway et al.'s study [17] is the only direct comparison between measured airborne concentrations and CFD simulations in a controlled room scale environment. While they also analysed and modelled deposition on the floor of an empty test room, only total deposition was considered and hence spatial variation is still uncharacterised. Lai and Chen [19,20] predicted deposition of particles sizes ranging from 0.01 to 10 μm with strong evidence supporting the claim that larger particles drop close to the source and do not remain suspended.

Deposition of bioaerosols also has implications for ward layout. Recommended bed spacing in multi-bed environments is often cited as being based on droplet transmission risk [21], and studies have recognised the relevance for pathogens such as *Staphylococcus aureus* as well as respiratory diseases [22]. Tracer gas and simulation studies have shown that ventilation design [13,23] and the presence of partitions between beds [23] influences airborne cross-infection risk between two patients. Several studies advocate the benefits of single patient rooms in reducing infection risk [24–26], although it is difficult to ascertain whether benefits can be directly attributed to room design or resulting change in nursing and hygiene practice. In reality many hospitals are constrained by their existing building stock and have a shortage of single rooms. There is currently little knowledge as to the importance of bioaerosol deposition in environmental contamination, so quantifying deposition in both single and multi-bed rooms is important for informing nursing practice and design.

This study uses a combined experimental and CFD modelling approach to evaluate the spatial distribution of bioaerosol deposition in a ventilated room. The primary objective of the study is to demonstrate, under a full-scale test environment, that CFD simulations are able to predict realistic deposition patterns for small diameter bioaerosol particles. The secondary objective of the study is to establish the influence of room layout on the spatial deposition of bioaerosols and the implications for infection control in a hospital context. The work builds on that of Hathway et al. [17] to carry out a direct comparison between the deposition pattern of a non-fastidious microorganism (*S. aureus*) nebulised into an aerobiology test room and the predicted deposition from CFD models incorporating Lagrangian particle tracking methodologies and two alternative turbulence models. The study then considers idealised single and two-bed hospital room scenarios to explore how the location of the source and room and ventilation layout influences the relative deposition on key surfaces in a patient environment.

2. Experimental methodology

2.1. Experimental set-up

Experiments were conducted in the environmentally controlled, negatively pressurized, aerobiology chamber at the University of Leeds. Dimensions are close to a hospital single room: 4.26 m (L) × 3.36 m (W) × 2.26 m (H). All walls are well insulated and considered adiabatic. External air was HEPA filtered before being conditioned by a humidifier and heater. This air was supplied to the chamber through a high level wall mounted diffuser as shown in Fig. 1.

Extraction of air was at a low-level, diagonally opposite; through a grille of the same design (Outlet). The ventilation rate in all experiments was 6 ACH, verified by using a balometer (Model PH721, TSI Incorporated, Shoreview, MN). Inlet air temperature (21.8 °C ± 1 °C) and humidity (60% ± 7%) were controlled throughout the experiments.

Prior to conducting bioaerosol experiments, air velocities in the empty room were measured using a hot wire comfort anemometer (Testo Ltd, Germany. Accuracy: ±0.01 m/s). Measurements were made on vertical lines at 5 selected locations in the room and at the supply air diffuser. The diffuser velocity profile (Fig. 1) was used to generate suitable boundary conditions for the CFD model, while the in-room measurements were for CFD validation purposes. Four main experimental scenarios were investigated as summarised in Table 1.

Empty room: The first experiment is similar to Hathway et al. [17], quantifying the spatial distribution of deposition in a similar manner to Wong et al. [18] but at a room-scale. Bioaerosol injection occurred at the geometric centre point in the room and no furniture or heat sources were present.
Single room: Experimental set-up number two replicates the situation within a single-bed, hospital room, where an infectious patient lies resting. A heated mannequin (DIN-man) is used to represent the heat source of the human. Particle collection is made on surfaces which mimic hospital furniture.
Double room: Scenarios three and four both present two heated mannequins, employed in a similar manner to Qian et al. [13]. Cross contamination of surfaces surrounding an infectious and a susceptible patient is examined by the collection of bioaerosols on adjacent surfaces. The effect of ventilation is investigated by reversing the location of susceptible and infectious source. The effect of a partition between the two beds is also included.

In scenarios 2–4, a quiescent patient was simulated by a DIN man (Deutsche Institut für Normung), a hollow aluminium cylinder (length 1 m by diameter 0.35 m) with an interior heat source. The heat source was created by a 100 W light bulb to represent the thermal emission of a resting adult human. Convective heat output from the skin is considered to be approximately 50%. Dimensions of the cylinder are however smaller than the average person but emit a similar heat flux. Infra-red thermal imaging of the DIN man shows the surface temperature in Fig. 2, which represents approximate body equivalents.

2.2. Bioaerosol generation

Staphylococci are spherical gram positive bacteria existing endogenously on most human skin squamae. With shedding of $\sim 10^6$ skin flakes per day, they are consequently abundant in many health-care settings [17,18]. *S. aureus* was chosen as the bacteriological agent given its ability to grow on general purpose media with ease. The *S. aureus* culture was incubated in nutrient broth (Oxoid, UK) for 24 h at 37 °C. Subsequent dilution tests showed the nebuliser concentration to be circa 10^{11} organisms per ml. A 10 ml aliquot of the pure culture was aseptically removed and suspended in 100 ml of sterile distilled water in a pre-autoclaved nebuliser. Sterile distilled water was the preferred suspension medium since it did not produce foaming of the suspension during nebulisation.