American Journal of Infection Control 44 (2016) S102-S108

Contents lists available at ScienceDirect

American Journal of Infection Control

journal homepage: www.ajicjournal.org

Global Perspective Article

Airborne spread of infectious agents in the indoor environment

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Key Words:
Respiratory droplet
indoor air
coughing
droplet dispersion
infection control
environmental ventilation

Background: Since the 2003 severe acute respiratory syndrome epidemic, scientific exploration of infection control is no longer restricted to microbiologists or medical scientists. Many studies have reported on the release, transport, and exposure of expiratory droplets because of respiratory activities. This review focuses on the airborne spread of infectious agents from mucus to mucus in the indoor environment and their spread as governed by airflows in the respiratory system, around people, and in buildings at different transport stages.

Methods: We critically review the literature on the release of respiratory droplets, their transport and dispersion in the indoor environment, and the ultimate exposure of a susceptible host, as influenced by airflows.

Results: These droplets or droplet nuclei are transported by expired airflows, which are sometimes affected by the human body plume and use of a face mask, as well as room airflow. Room airflow is affected by human activities such as walking and door opening, and some droplets are eventually captured by a susceptible individual because of his or her inspired flows; such exposure can eventually lead to long-range spread of airborne pathogens. Direct exposure to the expired fine droplets or droplet nuclei results in short-range airborne transmission. Deposition of droplets and direct personal exposure to expired large droplets can lead to the fomite route and the droplet-borne route, respectively.

Conclusions: We have shown the opportunities for infection control at different stages of the spread. We propose that the short-range airborne route may be important in close contact, and its control may be achieved by face masks for the source patients and use of personalized ventilation. Our discussion of the effect of thermal stratification and expiratory delivery of droplets leads to the suggestion that displacement ventilation may not be applicable to hospital rooms where respiratory infection is a concern.

Since the 2003 severe acute respiratory syndrome epidemic, the 2009 H1N1 influenza pandemic, and the 2014 Middle East respiratory syndrome epidemic, scientific exploration of infection control is no longer restricted to microbiologists or medical scientists. Fluid mechanics has played a role in understanding the mechanism of transmission and in developing engineering interventions; for example, the studies of airflow dynamics by Yu et al[1] provided plausible evidence of airborne transmission of severe acute respiratory syndrome. Airborne spread of infectious agents is directly relevant to the airborne route, and indirectly to the droplet-borne and fomite routes. Breathing, talking, sneezing, and coughing are major sources of some respiratory pathogens. Up to 40,000 droplets are expelled at a velocity of 100 m/s during a sneeze,[2] and a cough can generate approximately 3,000 droplet nuclei.[3] We now understand to some degree where and how respiratory droplets are formed and the pathogen content in each size of droplet. Turbulence and coherent structures in the airflow, mostly invisible, transport respiratory droplets between people. For example, vortex structures in coughing probably carry particles over long distances.[4] Our body's thermal plumes can bring fine droplet nuclei upward, and vortices generated during door opening and wakes behind walking individuals can transport contaminated air out of an isolation room. Turbulence generated by supply air jets causes mixing and dilution of room air. Understanding these airflows is crucial to minimizing spread of infectious agents and infection transmission.

Here, we review the release of respiratory droplets, their transport and dispersion in the indoor environment, and the ultimate

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Funding/Support: This research was funded by the Research Grants Council General Research Fund (HKU7142/12) and the Natural Science Foundation of China (51278440). Publication of this supplement is primarily supported by RB, Montvale, New Jersey, with additional support from MicroBioTest, a division of Microbac Laboratories, Inc., Sterling, Virginia. The City University of New York (CUNY) and the University of Ottawa, Ottawa, Canada, are academic sponsors. Editorial support was provided by Ashley O'Dunne, PhD; Shannon O'Sullivan, ELS; and Alanna Franchetti, ELS of Medergy (Yardley, PA), and funded by RB.

Conflicts of Interest: None to report.

0196-6553/
http://dx.doi.org/10.1016/j.ajic.2016.06.003

exposure of a susceptible host, as influenced by airflows. Microbial survival in the environment is beyond the scope of this article.

RELEASE, TRANSPORT, AND EXPOSURE

Release of droplets from mucus to mouth

If we understand the mechanism of where and how respiratory droplets are generated, we may have opportunities to suppress them at the source. Knowing the number and size of respiratory droplets is also crucial.

First, the content of infectious agents expelled by an infected person depends largely on the location within the respiratory tract where the droplets originate. Pathogenic microorganisms tend to be found in certain locations, particularly the tonsils and the larynx, and seldom at the front of the mouth.[5] Three droplet size distribution modes have been proposed: the bronchiolar fluid film burst mode, containing droplets produced during normal breathing ($d \leq 1$ μm); the laryngeal mode, most active during voicing and coughing ($d \geq 1$ μm); and the oral cavity mode, active during speech and coughing, producing droplets ≥100 μm.[6,7] The oral cavity is among the sources of expiratory droplets, especially larger ones[5,8,9]; the droplet formation mechanism in the oral cavity is shown in Figure 1. Large droplets from the trachea produced during coughing might not be released into the environment because they readily deposit within the head airways. Johnson et al[6] found that droplets ≥20 μm only originate from the oral cavity. Droplets generated during breathing may originate from both the upper and lower airways, but the latter seems to make the major contribution because of the film rupture mechanism.[12,13]

Second, 2 major mechanisms exist for droplet formation in the respiratory tract (Fig 1). One is the instability caused by the shear stress on the mucus-air interface that leads to the avalanche of mucus and droplet formation. The biphasic airway lining fluid has an overall thickness ranging from 5-100 μm. A critical air speed is required to initiate the instability, which varies according to mucus thickness, its viscoelastic properties, and surface tension at the mucus-air interface. Coughing is one mechanism for mucus clearance, during which air speed as high as 200 m/s can be attained[14] and interfacial shearing is peaked within the trachea.[15] This mechanism has traditionally been considered an exhalation process during coughing and sneezing; however, it was found to be also plausible around the first bifurcations during inhalation.[15,16] Recent studies include the effect of viscoelastic properties and surface tension on the onset of instabilities (eg, Vasudevan and Lange[17,18]) and the effect on the size distribution and volume concentration of bioaerosols produced.[19]

During normal tidal breathing, however, the shear force provided by the respiration airflow is not sufficient to induce instabilities. The mechanism for droplet formation during normal breathing relates to the reopening of collapsed terminal airways at the beginning of inspiration. Almstrand et al[20] examined the production of exhaled particles after varying degrees of airway closure. Concentrations of exhaled particles showed a 2- to 18-fold increase after exhalations to residual volume, compared with exhalations where no airway closure was shown. Malashenko et al[21] defined a critical capillary number ($Ca = \mu U/\sigma$, where μ is the dynamic viscosity of the liquid, U is the axial speed of the air-liquid meniscus propagation, and σ is the surface tension between the lining fluid and the air) above which droplets may be formed during normal breathing. In addition, experiments simulating the film droplet formation process showed that small fluid films generate droplets as efficiently as large films, and droplets may well be generated from films with diameters <1 mm (ie, the diameter of terminal bronchioles).[22]

Third, the reported number and size of released droplets vary significantly. In terms of the total mass of saliva, 1.1-6.7 mg of saliva were collected on a mask during a single cough, and 18.7 mg were collected while counting from 1-100.[9,23] There were 1-320 droplets per liter of exhaled air found for breathing, 24-23,600 found for coughing, and 4-600 found for speaking.[7,23-31]

Several factors account for the significant inconsistencies between the existing data (eg, individual differences, imperfect measurement techniques, and effect of evaporation). Humidity level is crucial in the measurement of droplet and droplet nuclei sizes.[29] Various measurement methods have been used, such as microscopic

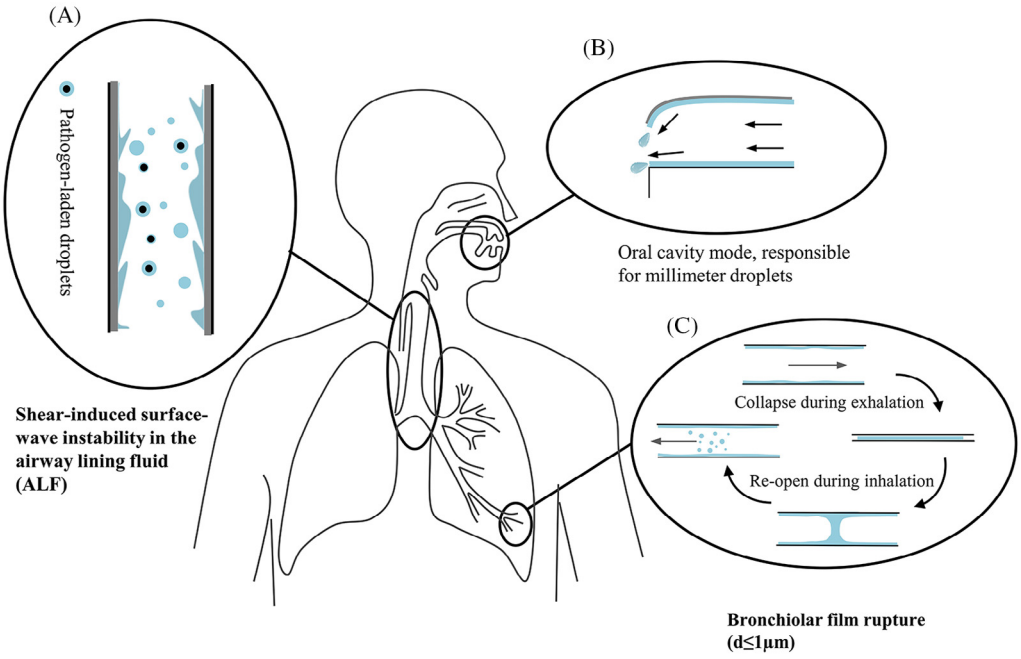

Fig 1. Schematic diagram revealing the origin and generation mechanism of respiratory droplets. (A) Instability of the airway lining fluid[10], (B) oral cavity model (drawn based on the atomization mechanism described in Morawska[5]), and (C) film rupture.[11]