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RETRACTED: AIRFLOW AND DOOR POSITION: A CASE STUDY ON AEROSOL DISPERSION WITHIN A GENERAL PATIENT AND AIRBORNE INFECTIOUS ISOLATION ROOM

K.R. Grosskopf, Ph.D.[a] **and Kelli Herstein**[b]

[a] Nebraska Hall 120, Durham School of Architectural Engineering and Construction, College of Engineering, University of Nebraska-Lincoln P.O. Box 880500 Lincoln, NE 68588-0500, USA kgrosskopf3@unl.edu +13524949591 FAX +14024724087 (corresponding author)

[b] Nebraska Hall 113, Durham School of Architectural Engineering and Construction, College of Engineering, University of Nebraska-Lincoln P.O. Box 880500 Lincoln, NE 68588-0500, USA kelliherstein@gmail.com

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ABSTRACT

An actual hospital was used to map the spatial dispersion of synthetic respiratory aerosols with respect to particle size, airflow, door position and personnel movement within a general patient room, an airborne infectious isolation room (AIIR) and corridor. Aerosols ≥1.0μm (most bacterial coliform and fungal spores) were found to be readily influenced by environmental conditions when compared to aerosols <1.0μm within both general patient and isolation rooms. Specifically, decay rates among particles ≥1.0μm were greater in the general patient room when compared to decay rates in the isolation room. In contrast, aerosols <1.0μm (viruses and some bacteria) appeared to disperse randomly and uniformly throughout both test rooms with significantly less regard to environmental conditions. Door motion and position were found to have a significant effect on room pressure relationships with adjacent spaces and subsequent aerosol containment in both general patient room and isolation anteroom. Results underscore the importance of not only maintaining proper environmental controls, but also diligence to proper entrance and egress procedures, source controls and use of personal protective equipment (PPE).

Subject Headings: airflow, hospitals, indoor air quality, particles, ventilation

INTRODUCTION

Infection via inhalation of pathogens (or pathogen-carrying particles) is termed airborne transmission. The efficacy of airborne transmission is influenced by many factors including the size, concentration, virulence, viability, and aerodynamic behavior of infectious particles. Infectious aerosols of greatest concern are those of respirable size (≤10.0μm), generated by both human and environmental sources, that have the capability of remaining airborne and viable (e.g.

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reproductive) for extended periods of time (Cole, et al., 1998). Infectious aerosols can be polymicrobial in nature, consisting of several species of bacteria, fungi and (or) viruses (Kowalski, 2007). Bacteria and fungi are typically present in colony-forming units (CFU) or spores larger than 3.0μm and can be effectively removed from the healthcare environment using filtration. However, filtration may be less effective at removing smaller airborne bacteria (<2.0 μm) and viruses (0.1-0.5μm) which are generally believed to be transmitted in dessicated respiratory droplets, or, droplet 'nuclei' (ASHRAE, 2003). As a result, ventilation air pressure relationships are considered one of the most effective methods to prevent the spread of airborne viruses (and other pathogens) within the health care environment.

Several organizations provide ventilation requirements for hospitals and clinics, including minimum air pressure relationships between function spaces. According to the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) Standard 170-2008, a general patient room must have a neutral pressure relationship to the corridor and other adjacent areas, a minimum of two changes of outdoor air (OA) per hour, six total air changes per hour (ACH), and, may re-circulate air only within individual rooms. An airborne infectious isolation room (AIIR) must have a negative pressure relationship to the corridor and other adjacent spaces, a minimum of two OA changes per hour, 12 total ACH, and, all exhaust air must be vented directly to the outdoors. The exhaust air volume in the AIIR must exceed the supply air volume by at least 1.4m^3/min to sustain a 2.5Pa negative air pressure difference and subsequent inward airflow between the isolation room and all other adjacent areas. AIIR anterooms must meet the same requirements as the isolation room, except the minimum number of total air changes is reduced to 10. Moreover, the anteroom should be positively pressurized with respect to the isolation room and negatively pressurized with respect to the corridor.

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The basis of such standards and the evidence supporting their effectiveness, however, is limited. Of 183 papers published worldwide from 1960-2005 with keywords or medical subject headings (MeSH) pertaining to airborne transmission of respiratory diseases, only 40 studies provided data on ventilation and airflows. Of these, only 10 studies were deemed by a panel of epidemiology and engineering experts as having conclusively demonstrated an association between airflow relationships and the transmission of infectious aerosols. Eight of the studies attempted to link index and secondary cases of measles, tuberculosis (TB), chickenpox, influenza and severe acute respiratory syndrome (SARS) using experimental or mathematical airflow studies. Retrospective airflow data was generally poor with little assurance it was representative of environmental conditions at the time of the outbreak. Few studies could eliminate other modes of transmission such as contact or droplet transmission, and collectively, data was insufficient to specify the minimum ventilation requirements to control the spread of airborne disease in any setting (Li et al., 2007). Subsequent studies using tracer gases or synthetic aerosols were comparatively brief and did not address the movement of people or the release of potentially infectious particles to adjoining healthcare spaces.

In response, the purpose of this case study is to observe the spatial dispersion of synthetic respiratory aerosols with respect to particle size, airflow, door position and personnel movement within a general patient room, an airborne infectious isolation room (AIIR) and corridor. These spaces were chosen for study given the potential for airborne disease transmission from infectious patients to large numbers of cohort patients and healthcare workers. While time and logitistical constraints preclude longitudinal study or laboratory control of the test environments, the results of this study, when compared to findings from other independent case studies, may provide evidence for effectiveness of ventilation standards and other environmental controls.

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LITERATURE REVIEW

Several studies from 1980-2005 have been able to demonstrate an association between airborne disease transmission and environmental factors such as airflow, door position, and personnel movement in both general patient and airborne infectious isolation rooms (AIIRs). Results of recent studies found that a single human cough generates ~10^4 particles with more than 70% in the respirable range of 10.0μm or less (Xie et al., 2006; Yang et al., 2007). Device generated aerosols produced by breathing assist equipment such as nebulizers, ventilators or oxygen masks are often much smaller in size (<5.0μm) and can more readily penetrate the lower respiratory tract, causing infection with a smaller dose (Bridges et al., 2003; Booth et al., 2005, Tellier, 2006-09; Brankston et al, 2007; Hui et al., 2007-09). Higher air change rates, particularly in AIIRs, can produce turbulent airflow which has been shown to significantly increase the horizontal and vertical migration of aerosols, particularly larger particles that would not otherwise move passively (Eames et al., 2009).

Airflow

A study of 1,289 healthcare workers (HCWs) in 17 Canadian hospitals found the risk of tuberculin conversion was 3.4 times higher in general patient rooms with <2.0 ACH when compared to patient rooms ≥2.0 ACH (Menzies, et al., 2000). In another study, a pediatric patient receiving respirator-assisted ventilation with VZV pneumonia was implicated in the transmission of secondary infections to 13 of 24 susceptible cohort patients. Retrospective airflow studies found that the index patient's room had no exhaust, resulting in a positive (outward) airflow-pressure relationship with respect to the corridor and adjacent spaces. Analysis of a nearby patient room having a 90% attack rate found the supply air system inoperable, resulting in a negative (inward) airflow-pressure relationship with respect to the

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index patient's room and corridor (Leclair et al., 1980). In a similar study, secondary VZV infection occurred in eight out of 36 susceptible patients despite isolation procedures. Tracer gas (SF_6) reached concentrations in the corridor as high as 10-15% of those inside the patient room, then 'halved' every ~6.0m in the adjacent corridor. Correspondingly, attack rates declined at roughly the same rate as the decline in SF_6 concentration (Gustafson et al., 1982).

More recently, research has shown a clear association between the infection patterns between index and secondary cases of SARS that could not be explained by the known limitations of either contact or droplet transmission. Retrospective airflow analyses of the Hong Kong outbreak found the supply air rate to be nearly 4 times the exhaust rate in the index patient room, resulting in a strong outflow of contaminated air to the corridor and adjacent rooms. Again, a direct correlation was observed between attack rates and bioaerosol concentrations simulated by tracer gas (CO_2) and computational models (Li et al., 2004; Yu et al., 2005).

Door Position

Although ventilation and directional airflow are clearly implicated in the transmission of airborne disease, door position, door motion and personnel movement were also likely contributors. As Leclair (1980) indicates, "activities" associated with the index patient's critical care "necessitated frequent and prolonged opening of the door to the room." Gustafson (1982) noted that tracer gas concentrations in rooms where secondary infections occurred averaged 50% of those in the corridor with entry door open despite 0.3-1.1m^3/min outward airflow. Another retrospective study of nosocomial transmission of VZV to 3 HCWs found NO_2 tracer gas concentrations in a nursing station equal to (or greater) than concentrations of NO_2 released through an open door from a nearby isolation room under 0.7m^3/min negative air pressure (Josephson and Gombert, 1988). In a subsequent study, a nurse was reported to have passed

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equipment through a doorway to other hospital staff attending to a VZV patient in isolation. Despite 3.0Pa negative air-flow-pressure, the nurse developed the same genotype of VZV, even though he did not enter the room (Tang et al., 2005).

An analysis of the door-opening motion indicates that the negative pressure relationship between an AIIR and adjacent spaces can be reversed if the door-opening motion is too rapid. Specifically, when the entry door to either the anteroom or isolation room is opened, a vortex of air appears to wrap around the leading edge of the door, allowing temporary spillage of potentially infectious air into the anteroom or corridor. The exchange volume of air produced by the door-opening motion is comparable to the swept volume of the door, or approximately $3m^3$. An added exchange volume of air can be produced by a person entering the room. A typical person with a forward projected area of $0.8m^2$ walking at 1m/s can generate a 'body wake' of approximately $4m^3$ (Tang et al., 2005). Together, a HCW opening a door and quickly exiting an AIIR can transport as much as 5-10 percent of the room volume to the corridor despite a 2.5Pa pressure difference (Eames et al., 2009).

If a temperature difference exists between the isolation room and corridor, colder (denser) air from the corridor may force warmer air from the isolation room upward into the breathing zone of unprotected HCWs entering the anteroom (vestibule), or, to persons nearby in the corridor (Tang et al., 2005). A cluster sample of 346 patients with acquired immunodeficiency syndrome (AIDS) found that 21 nosocomial tuberculosis infections occurred in a total of 16 patient rooms that were located two rooms or less away from index cases. In four of these rooms, inward airflow from the corridor to the patient room was observed at the bottom of the doorway while outward airflow from the patient room to the corridor was observed at the top of the doorway (Edlin et al., 1992).

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METHODS

A 37,510m^2 hospital was used to simulate the aerodynamic behavior of synthetic aerosols under various ventilation alignments in various function spaces. A total of two tests were conducted; one (1) each in a general patient room (22.5m^2) and an infectious isolation room (25.8m^2) located within a nursing ward (1,613.7m^2) on the 5th floor of an eight (8) story 'bed' tower (Figures 1-2, A1 and A3 Supplemental). The ward consisted of twenty-eight (28) general patient rooms and two (2) airborne infectious isolation rooms (AIIRs) as well as ancillary spaces.

Airflow Measurement

The ward was supplied with 168.8m^3/min of 100% outside air (as verified by duct traverse measurements) from a single air handling unit (AHU) providing conditioned air directly to the corridor, ancillary spaces and isolation rooms and indirectly to general patient rooms via re-circulating fan coil units. Exhaust air within the ward was removed by two (2) exhaust air risers serving other zones on other floors. Pressure mapping indicated that the 5th floor ward was positive in relation to the 4th floor (7.5Pa) and neutral in relation to the 6th floor. Alnor flow hood measurements (+/- 3% error) in the general patient test room indicated that supply air (7.4m^3/min) , return air (5.0m^3/min) and bathroom exhaust air (2.4m^3/min) were nearly balanced (Figure A2, Supplemental), producing 2.5 outside ACH, 7.7 total ACH, and, a neutral air pressure relationship with respect to the corridor in conformance with ASHRAE Standard 170-2008. Flow hood measurements in the AIIR test room indicated that exhaust air (6.2m^3/min) exceeded supply air (3.9m^3/min) by 2.3m^3/min, producing a 2.0Pa negative air pressure relationship with respect to the corridor (Figure A4, Supplemental). AIIR test room ventilation produced 3.5 outside ACH, and, 5.5 total ACH (roughly half the total airchange rate prescribed by ASHRAE Standard 170-2008 for an AIIR).

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The average temperature in both general patient and AIIR test rooms was 20.5^{o}C during testing. The average supply air temperature was 19.5^{o}C as there was little sensible load generated from within the vacated building or from the outside as outdoor temperatures were mild, ranging from 18.9-22.2^{o}C. Related, average relative humidity was high (>80%) as there was little effort to control latent loads during the decomissioning process occuring in other areas of the hospital. Barometric pressure remained near constant at 1,015mb and the average indoor air density was 1.18kg/m^{3}. Average wind speed was less than 2km/hr from the south-southwest (220^{o}) roughly parallel along the exterior (south) façade of the general patient room bed tower opposite the north facing AIIR.

The only significant heat source in either the general patient or AIIR test room was 80W of ceiling mounted fluorescent lighting used during the test. A thermal manikin simulating a human patient was not used. Although studies have shown the effects of body heat induced plumes on aerosols (Wan et al., 2007 and Chao et al., 2008), the effects of thermal flows due to people is not modelled in the experiments. In addition, spatial uniformity testing for the single zone ward was conducted using sulfur hexafluoride (SF_6) tracer gas decay method according to ASTM E741. The average SF_6 concentrations within the general patient room (137.5ppm) and AIIR (120.5ppm) was compared to the average SF_6 concentration within the zone (124.0ppm). Indoor temperature, relative humidity and air density were continuously recorded at a centrally located nursing station during the test. Outdoor wind speed and direction, precipitation, temperature, relative humidity and barometric pressure were recorded from two (2) meteorological stations placed outside windows immediately adjacent to the general patient room on the south tower façade, and, adjacent to the AIIR on the north façade. A third station was placed on the roof at the east end of the bed tower.

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Aerosol Generation

To simulate a respiratory aerosol, a synthetic aliphatic hydrocarbon (polyaliphaticolefin) approximately 84.7% of the density of water (at 20°C) was aerosolized at a rate of approximately 1.0g/min at 0.4L/s airflow rate (Wan et al., 2007; Chao et al., 2008), to generate a 0.5μm-10μm poly-disperse liquid aerosol (Figure A5, Supplemental). Polyaliphaticolefin (PAO) has flexible alkyl branching groups on every other carbon of its polymer chain, allowing it to remain a viscous, oily liquid well above (and below) normal room temperatures. As a result, the PAO aerosol did not readily desiccate and was more likely to maintain consistent aerodynamic behavior independent of fluctuating indoor temperature and humidity during testing.

As per the literature, the particle size distribution and production rate of respiratory aerosols varies widely depending on the individual, health condition and activity (e.g. breathing, talking, coughing, sneezing, etc.). As a result, no attempt was made to generate a specific particle size distribution or production rate within the size range of particles (0.5μm-10μm) used for this study. This size range however, is consistent with findings of other recent studies (Xie et al., 2006; Yang et al., 2007) which found that a human cough generates ~10^4 particles with a size range distribution of 0.62-15.9μm (71% <10.0μm) that are capable of being ejected 2m from the patient (10m/s initial velocity). For this study, the PAO aerosol was continuously injected at the approximate location of a patient's nose-mouth at rest (0.8m) using a NUCON SN-10 pneumatic aerosol generator. The aerosolization rate (0.4L/s) was roughly twice the ventilation rate of a healthy human at rest (0.7L per breath, 16-18 breaths per minute).

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Particle Measurement

Particle size measurements (particles/L) ranging from 0.5-10.0µm were collected using a NUCON F-1000-DD light scattering photometric aerosol detector (+/-2.5% error) at a total of ten (10) sampling locations (A_{1-5} and B_{1-5}) in each test room (Figures 1 and 2). Each sampling location consisted of three (3) sampling points at 0.6m, 1.2m and 1.8m above the floor (Figure 3). Air samples from each sampling point were drawn at 30 second intervals for a total of 30 minutes each. In addition, two (2) Lighthouse HH-3016-IAQ portable particle size counters were positioned in the center of the patient room bath (location of room exhaust) at a sampling height of 1.2m, and, in the corridor immediately outside the patient room above the entry door at a sampling height of 2.1m. Two additional portable particle size counters were positioned in the center of the AIIR anteroom at a sampling height of 1.2m, and, in the corridor immediately outside the anteroom room above the entry door at a sampling height of 2.1m. All sampling instrumentation was calibrated prior to testing using 2.5mg of PAO per m^3 of air as part of a procedure developed with guidance from ANSI 510 and 511, ASME AG-1 and ASHRAE 52.2.

Experimental Protocol

At the start of testing in the general patient room, the entry door was closed and the door to the bathroom was open. Concentrations of ambient airborne particles were then sampled for 30 minutes prior to PAO aerosol injection. Beginning at sampling locations A_1 and B_1, and ending at locations A_5 and B_5, a technician entered the test room once every 30 minutes to reposition sampling equipment. Each time the technician entered the test room, the entry door was opened and remained opened for approximately 30 seconds. Upon exiting the room, the technician closed the door. In addition to repositioning sampling equipment, this activity was intended to simulate the movement of a healthcare worker entering and exiting the room.

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At the start of testing in the AIIR, the entry doors to the anteroom and the isolation room were fully closed and the door to the bathroom was fully open. Following the same procedure as the general patient room test, background concentrations of airborne particles were sampled prior to aerosol injection and sampling equipment was repositioned every 30 minutes. Each time the technician entered the anteroom however, the technician opened and immediately closed the door from the corridor, paused for one minute to simulate donning personal protective equipment (PPE), then entered the isolation area. Upon exiting the room, the technician opened and immediately closed the door leading to the anteroom, paused for one minute in the anteroom to simulate the removal of PPE, then exited the anteroom by opening and immediately closing the corridor door.

Following the A_5 and B_5 sampling series in the general patient room, the entry door to the general patient room was opened and remained open for the remainder of testing. Thirty minutes later, the bathroom door was closed and remained closed for the remainder of testing. Following the A_5 and B_5 sampling series in the AIIR, the entry door from the anteroom to the isolation room was opened and remained open for the remainder of testing. Thirty minutes later, the door leading to the anteroom from the corridor was opened and remained open for the remainder of testing. For both general patient and isolation room tests, aerosol injection was terminated 30 minutes after the second door position change and samples collected for an additional 30 minutes to determine the time necessary to ventilate the test rooms to background levels. The intent of this test procedure (Tables 1 and 2) was to evaluate the effects of door position and personnel movement on aerosol dispersion. All doors were solid and opaque without ventilation louvers or fenestration.

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RESULTS

Data from 'A' series sampling locations (A_1-A_5) at a sampling height of 1.8m were chosen as a 'baseline' to compare the spatial dispersion of the aerosol with respect to particle size, airflow, door position and personnel movement within a general patient room, an AIIR and corridor (Figures 4-7). This data was intended to represent the maximum breathing zone exposure of a healthcare worker standing within the airflow current between the 'patient' (aerosol injection point) and return/exhaust air vents. As shown, particle concentrations (particles/L) for all size groups increased significantly when the injection was started. Concentrations of particles <1.0µm in the general patient test room stabilized approximately 15 minutes into the test and remained relatively constant, regardless of time and distance from the aerosol injection point, for the remainder of the testing until the entry door was opened. Concentrations of particles <1.0µm in the isolation test room stabilized approximately 45 minutes into the test and remained relatively constant until the anteroom door was opened.

In contrast, concentrations of particles ≥1.0µm did not stabilize in either the general patient or isolation test rooms. After peaking at sampling locations A_2 and B_2, concentrations of particles ≥1.0µm in the general patient room *decreased* with respect to time (r^2=-0.92), and to a lesser extent, distance (r^2=-0.59) from the point of release. The highest rates of decay were observed at the 1.8m sample height and the lowest rates of decay were observed at the 0.6m sampling height (r^2=-0.97; Tables 3, A1-A4, Supplemental). Conversely, concentrations of particles ≥1.0µm in the isolation room *increased* with respect to time (r^2=0.69), and to a lesser extent, distance (r^2=0.47) from the point of release for both 'A' and 'B' series sampling locations. Analysis of the 'A' and 'B' series sample locations in the isolation test room indicates that nearly twice the volume of air was exhausted upward at more than 10-times the velocity than

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air supplied downward from ceiling mounted vents. Other possible explanations include 'mixing' from multiple ceiling mounted supply and exhaust air vents in the isolation test area, or, 2.3m^3/min of 'make-up' air infiltrating through the isolation room envelope at or near floor level (e.g. bottom of doors, partitions, fenestration, etc.).

Although higher rates of decay were observed at higher sampling heights in the general patient room (Table 3), higher concentrations of particles ≥1.0µm remained at higher sampling heights for 'A' series sampling locations in both the general patient and isolation rooms. In contrast, higher concentrations of particles ≥1.0µm were observed at *lower* sampling sampling heights for 'B' series sampling locations in both general patient and isolation test rooms. For both general patient and isolation test rooms, concentrations of 'A' series particles ≥1.0µm were greater, on average, than corresponding concentrations of ≥1.0µm particles at 'B' series sampling locations at the same sampling location and height. In other words, average concentrations of ≥1.0µm particles increased as sampling height increased within the airflow stream ('A' series) and decreased as sampling height increased outside of the airflow stream ('B' series). As a result, the observed spatial dispersion of aerosol in both general patient and isolation test rooms supports the premise that particles ≥1.0µm may be readily mobilized and suspended by airflow currents within the breathing zone between the supply air and 'patient' (aerosol injection point), and, the return/exhaust air ('A' series sampling locations).

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In contrast, particles ≥1.0µm appear to have a higher rate of gravitational settling within the 'static' airspace between the supply air and exterior wall ('B' series sampling locations). Consequently, the potential exposure to infectious aerosols ≥1.0µm appears greatest within the breathing zone between the patient and the return/exhaust air in both general patient and isolation test rooms. By comparison, the spatial dispersion of <1.0µm particles occurs rapidly and uniformly throughout both general patient and isolation test rooms and does not appear to differ significantly regardless of distance from the aerosol injection point, sampling height, or, differences in airflow (e.g. 'A' series vs. 'B' series). The effects of coarse fiber return air filters on aerosol concentration and distribution within the general patient test room were considered minimal as filters of this type (MERV<4) have a filtration efficiency of <20% for particles ≤10.0µm (ASHRAE, 2007). Since the isolation test room was supplied 100% ventilation air, there was no return air (e.g. recirculated air) and thus no in-room filtration.

The spatial dispersion of aerosol was also observed with respect to the position of test room doors, and, the movement of personnel into and out of the test rooms. Beginning at locations A_1 and B_1, a technician briefly entered the general patient and isolation test rooms once every 30-40 minutes to reposition sampling equipment. In each instance, the turbulence created by the door opening-motion appeared to allow the intermittent release of both <1.0µm and ≥1.0µm aerosols into the isolation anteroom (Figure 8), and, into the isolation and general patient room corridors (Figures 9 and 10), despite the presence of a negative air pressure relationship between the isolation room, anteroom and corridor, and a neutral air pressure relationship between the general patient room and corridor. The relatively small release of aerosols into the corridor did not appear to significantly affect the concentrations of either <1.0µm or ≥1.0µm particles within either the general patient or isolation test rooms.

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Following test series A_5 and B_5, the inner door between the isolation and anteroom was opened and remained open for the duration of testing. Immediately after opening the inner door, concentrations of both <1.0μm and ≥1.0μm particles increased significantly in the anteroom (Figure 8). Concentrations of both <1.0μm and ≥1.0μm particles also increased in the corridor (Figure 9), although only briefly. Correspondingly, concentrations of both <1.0μm and ≥1.0μm particles decreased in the isolation room, with the most rapid decline observed among ≥1.0μm particles at the B_5 sampling location furthest from the anteroom. As a result, data indicates the migration of aerosol from the isolation room to the anteroom caused by the opening of the inner door between the isolation room and anteroom.

Approximately 35 minutes after the inner door between the isolation and anteroom was opened, the outer door between the anteroom and corridor was opened and remained open for the duration of testing. Immediately after opening the outer door, concentrations of both <1.0μm and ≥1.0μm particles decreased rapidly in the anteroom (Figure 8). Concentrations of <1.0μm and ≥1.0μm particles also increased in the corridor (Figure 9), although (again) only briefly. Although concentrations of both <1.0μm and ≥1.0μm particles continued to decline to near background levels at sampling point B_5, concentrations of <1.0μm and ≥1.0μm particles at sampling point A_5 began to increase, presumably under the influence of infiltrating air from the corridor and two exhaust air vents in close proximity to the A_5 sampling location. Although the negative air pressure relationship between the isolation room and corridor restricted the escape of both <1.0μm and ≥1.0μm particles into the corridor, the potential exposure to infectious aerosols appears greatest in the anteroom when the inner door is left opened, particularly among healthcare workers donning or doffing PPE in the anteroom.

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Similarly, the entry door between the general patient room and corridor was opened and remained open following test series A_5 and B_5. Immediately after opening the entry door, concentrations of both <1.0μm and ≥1.0μm particles increased significantly in the corridor (Figure 10). Unlike the isolation test room, significant quantities of <1.0μm and ≥1.0μm particles continued to escape from the general patient room into the corridor through the opened entry door. Correspondingly, concentrations of <1.0μm particles decreased in the general patient room with the most rapid decline observed at the B_5 sampling location furthest from the corridor. Concentrations of ≥1.0μm particles also decreased in the general patient room at all sample locations except at A_5 1.2m and 1.8m sample heights (Figures 4 and 5). Simultaneously, concentrations of both <1.0μm and ≥1.0μm particles decreased in the general patient bathroom where the room exhaust fan was located (Figure 11). Together, this data suggests that particles in both size groups likely migrated from the general patient room into the hospital corridor, even though the air pressure difference between the patient room and corridor was found to be near neutral.

Approximately 30 minutes after the entry door to the corridor was opened, the general patient bathroom door was closed. Concentrations of ≥1.0μm particles increased rapidly at the A_5 sampling point located between the bathroom and corridor (Figures 4 and 5). Again, concentrations of both <1.0μm and ≥1.0μm particles increased outside of the entry door to the corridor (Figure 10) while declining rapidly in the bathroom (Figure 11). Increases in particles observed in the corridor appeared to 'mirror' proportional decreases in particles observed in the bathroom, suggesting that the closed bathroom door impinged exhaust air ventilation, causing the patient room to become positive relative to the hallway and allowing aerosols in both size groups to escape from the patient room into the corridor.

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Once the aerosol injection was terminated, both particle size groups in both general patient and isolation rooms decreased rapidly. In the isolation test room, concentrations of both <1.0μm and ≥1.0μm particles returned to background levels with 15-20 minutes (Figures 6-9). In the general patient test room, concentrations of ≥1.0μm particles returned to near background levels whereas <1.0μm particles remained above pre-test levels 30 minutes after the aerosol injection was terminated, particularly in the corridor (Figures 4-5 and 10-11). As a result, the effect of ventilation air on removing aerosols appears more significant among ≥1.0μm particles when compared to removal rates among particles <1.0μm in the general patient room.

DISCUSSION

This study used an actual hospital to simulate the aerodynamic behavior of human expiratory droplets via a synthetic aerosol. In general, particles ranging in size from 0.5μm to 10.0μm appeared to be influenced by airflows caused by the mechanical supply air system, door position and personnel movement. However, respirable aerosols <1.0μm were found to exhibit distinctly different aerodynamic behaviors when compared to aerosols ≥1.0μm, as did aerosols subject to different airflow conditions within a general patient room and AIIR. The most significant differences in aerosol behavior in relation to environment were observed among particles ≥1.0μm. Specifically, concentrations of particles ≥1.0μm in the general patient test room *decreased* with respect to time and distance, with the highest rate of decay occurring at the highest sampling heights. In contrast, concentrations of particles ≥1.0μm in the isolation test room *increased* with respect to time and distance, possibly caused by the volumetric dominance and high velocity, turbulent up-draft created by multiple exhaust air vents.

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When compared to particles ≥1.0μm outside of the directional airflow stream, particles ≥1.0μm in both general patient and isolation test rooms appeared to be readily mobilized and suspended within the breathing zone between the supply air and patient, and, return air/exhaust. By comparison, the spatial dispersion of <1.0μm particles occurred rapidly and uniformly throughout both test rooms with little or no change in particle concentration observed with respect to sampling location, sampling height or airflow.

Turbulence created by the door-opening motion appeared to allow the intermittent release of relatively small amounts of both <1.0μm and ≥1.0μm aerosols into the isolation anteroom, and into the isolation and general patient room corridors despite the presence of neutral to negative air pressure relationships between these adjacent spaces. This finding is consistent with several epidemiological studies (Leclair et al., 1980; Gustafson et al., 1982; Edlin et al., 1992; Tang et al., 2005; Eames et al., 2009) which found that door-opening motion may allow temporary spillage of infectious air into adjacent spaces despite the presence of inward airflow.

However, leaving the inner door to the anteroom and entry door to the general patient room open resulted in a significant and prolonged release of both <1.0μm and ≥1.0μm aerosols into the anteroom and corridor immediately outside of the general patient room despite the presence of neutral to negative airflow, similar to the Josephson and Gombert study (1988). Closing the bathroom door in the general patient room appeared to impinge the exhaust air, causing the room to be pressurized and release additional aerosol into the corridor. Similarly, exhaust air impingement (or insufficient exhaust air) was found to be the primary cause of airborne transmission in several other epidemiological studies (Anderson et al., 1985; Hutton et al., 1990; Li et al., 2004; Yu et al., 2005). In contrast, no prolonged release of aerosol between the isolation room and corridor was observed with respect to anteroom or entry door position.

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After testing, concentrations of ≥1.0µm particles returned to near background levels within 30 minutes (or less) in both general patient and isolation test rooms, as did <1.0µm particles in the isolation room. However, concentrations of <1.0µm particles remained above pre-test levels in the general patient room, particularly in the entryway to the corridor. This data, and the uniform dispersion of particles <1.0µm observed in both patient and isolation test rooms, suggests that particles <1.0µm may be less influenced by ventilation air than those ≥1.0µm.

In summary, the results of this study, when compared to findings from several other independent case studies, seem to indicate that healthcare ventilation standards such as ASHRAE 170-2008 are effective in limiting the release of aerosols 0.5-10.0µm between an infectious isolation room and adjacent corridor, even when both anteroom and entry doors are open. However, the potential for exposure in the isolation room appears greatest among unprotected healthcare workers in the anteroom when the inner door is open and outer door is closed. Related, the neutral air pressure relationship between a general patient room and corridor appears effective in limiting the release of aerosols into the corridor when the entry door is closed. When the entry door to the general patient room is opened, aerosols 0.5-10.0µm appear capable of escaping into the corridor. In patient rooms where the only source of exhaust ventilation is in the bathroom, the release of aerosol into the corridor can increase significantly when an unvented (e.g. 'solid') bathroom door is closed.

Furthermore, concentrations of aerosols ≥1.0µm were significantly higher in the breathing zone between the 'patient' (aerosol generator) and location of return and (or) exhaust air ventilation (e.g. the bathroom) in both general patient and isolation rooms. As a result, the potential for exposure in both general patient and isolation rooms appears least opposite the patient and return/exhaust air vents. Since aerosols <1.0µm were observed above background

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levels more than 30 minutes after testing, patient rooms may require an extended period of time (e.g. an hour or more) to ensure adequate removal of respiratory aerosols once a patient is discharged. Together, the data appears to support the effectiveness of not only proper engineering controls, but also attentiveness to procedures, particularly entrance and egress procedures and use of PPE within infectious isolation environments.

LIMITATIONS

The research presented herein was conducted in an actual healthcare setting using a synthetic (non-human) respiratory aerosol. Although attempts were made to control the test environment in conformance with consensus healthcare standards (e.g. ASHRAE 170-2008), many factors remained uncontrollable or unknown. General opinions have been made relating observed aerosol behavior to observed environmental conditions. However, no attempt has been made to suggest, recommend or specify changes in current ventilation standards to control the spread of airborne disease in any healthcare setting.

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FIGURE 1. Aerosol sampling locations (A_{1-5} and B_{1-5}) in general patient test room.

FIGURE 2. Aerosol sampling locations (A_{1-5} and B_{1-5}) in isolation patient test room.

FIGURE 3. Aerosol generator and particle sampling equipment used in general and isolation patient test rooms. Sampling locations A_1 and B_1 shown at 0.6m, 1.2m and 1.8m sampling heights, respectively.

FIGURE 4. Particle concentration relative to size (0.5-3.0μm), time and distance from aerosol injection point for 'A' series sampling locations at height of 1.8m in general patient test room.

FIGURE 5. Particle concentration relative to size (3.0-10.0μm), time and distance from aerosol injection point for 'A' series sampling locations at height of 1.8m in general patient test room.

FIGURE 6. Particle concentration relative to size (0.5-3.0μm), time and distance from aerosol injection point for 'A' series sampling locations at height of 1.8m in isolation test room.

FIGURE 7. Particle concentration relative to size (3.0-10.0μm), time and distance from aerosol injection point for 'A' series sampling locations at height of 1.8m in isolation test room.

FIGURE 8. Particle concentration relative to particle size (0.5-10.0μm) in the anteroom of the isolation test room.

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FIGURE 9. Particle concentration relative to particle size (0.5-10.0μm) in the corridor immediately outside of the isolation test room.

FIGURE 10. Particle concentration relative to particle size (0.5-10.0μm) in the corridor immediately outside of the general patient test room.

FIGURE 11. Particle concentration relative to particle size (0.5-10.0μm) in the bathroom of the general patient test room.

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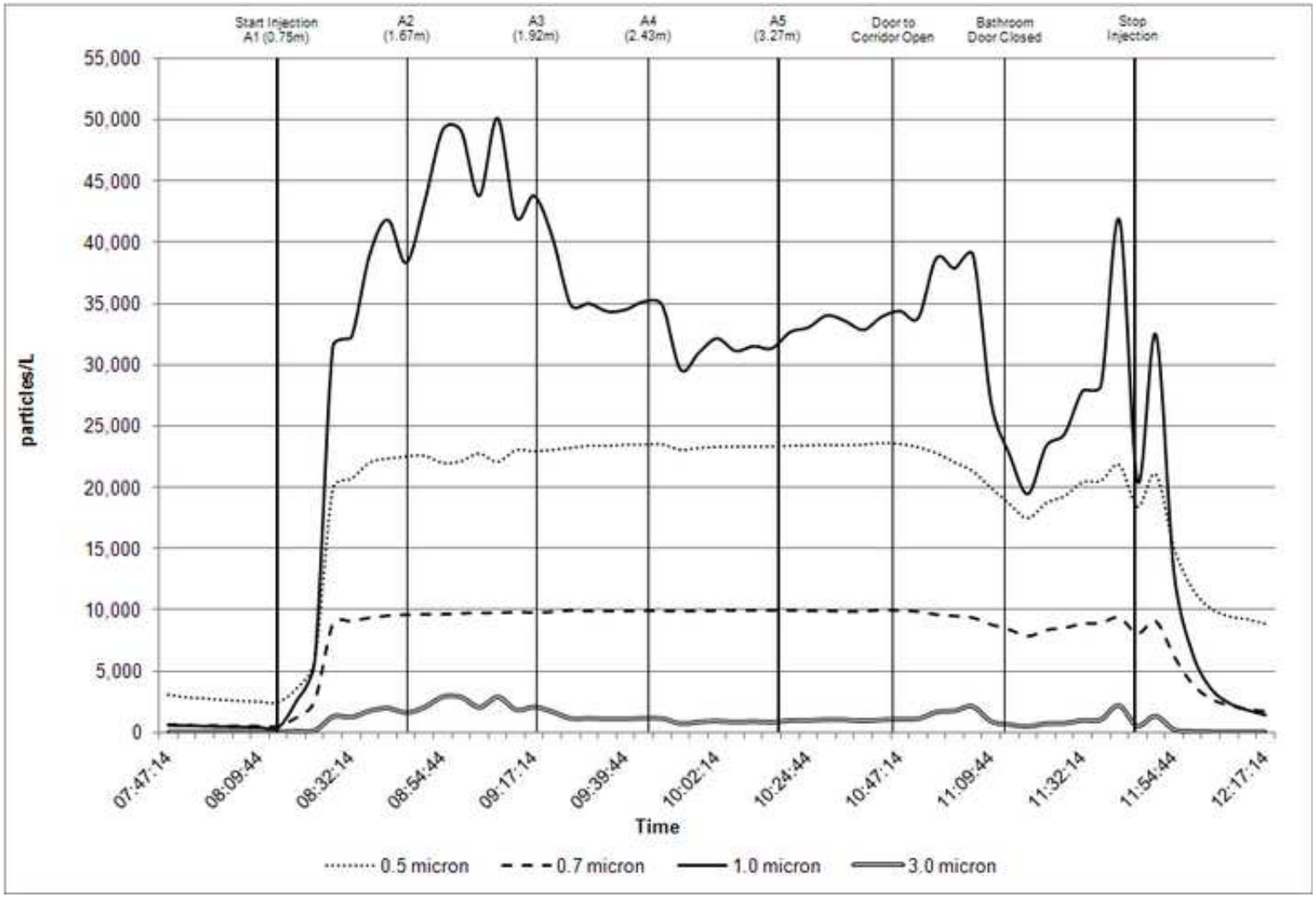

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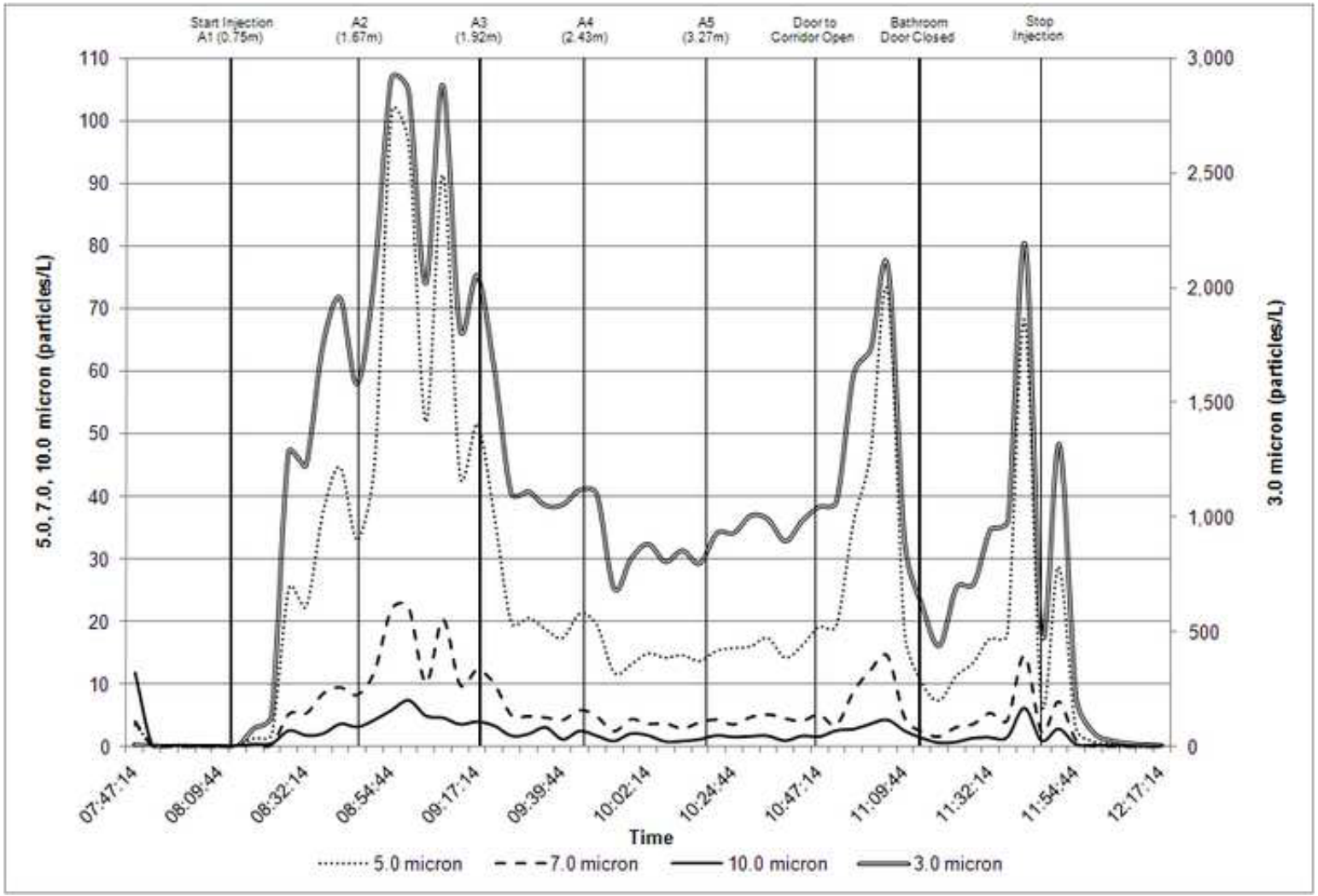

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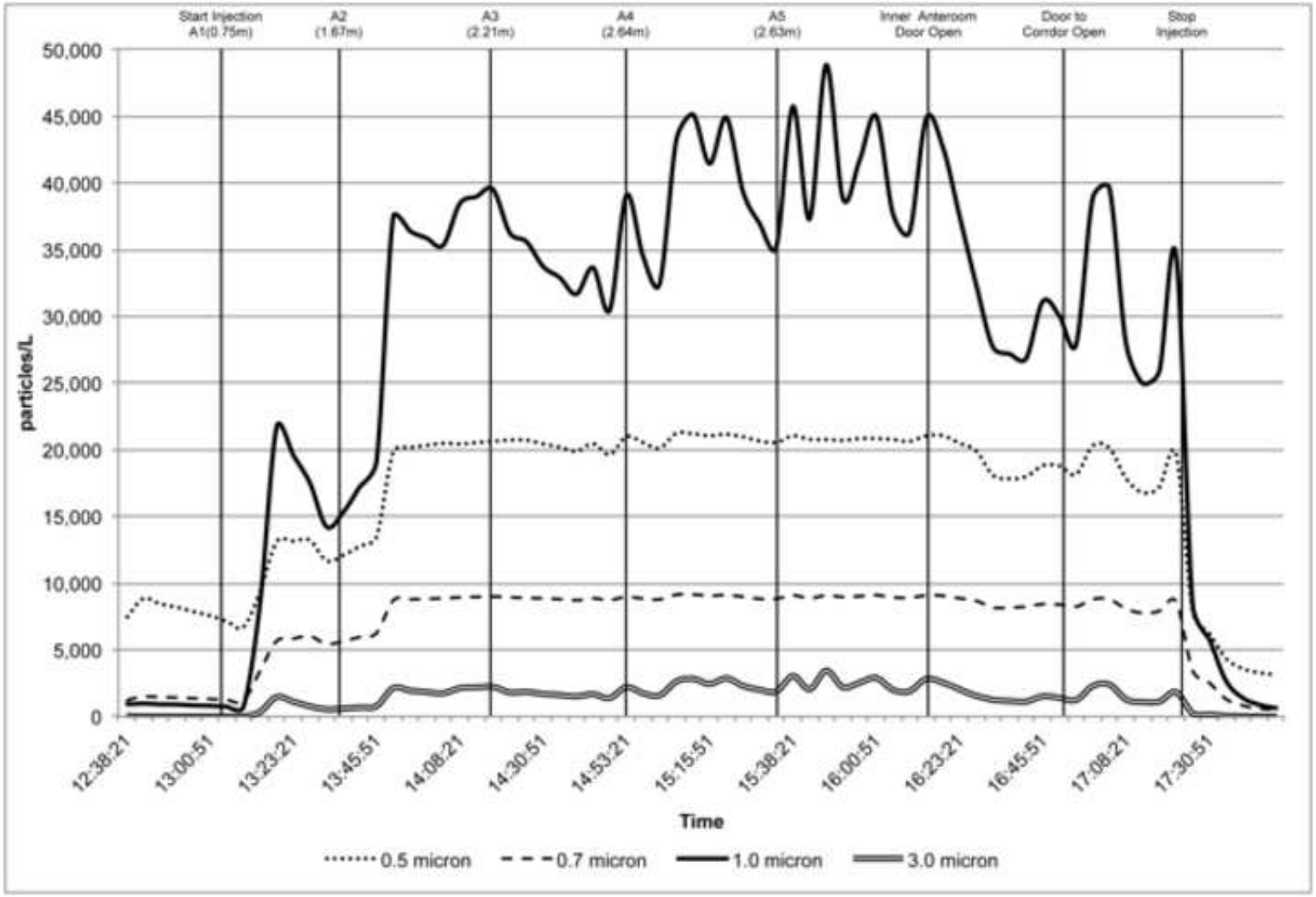

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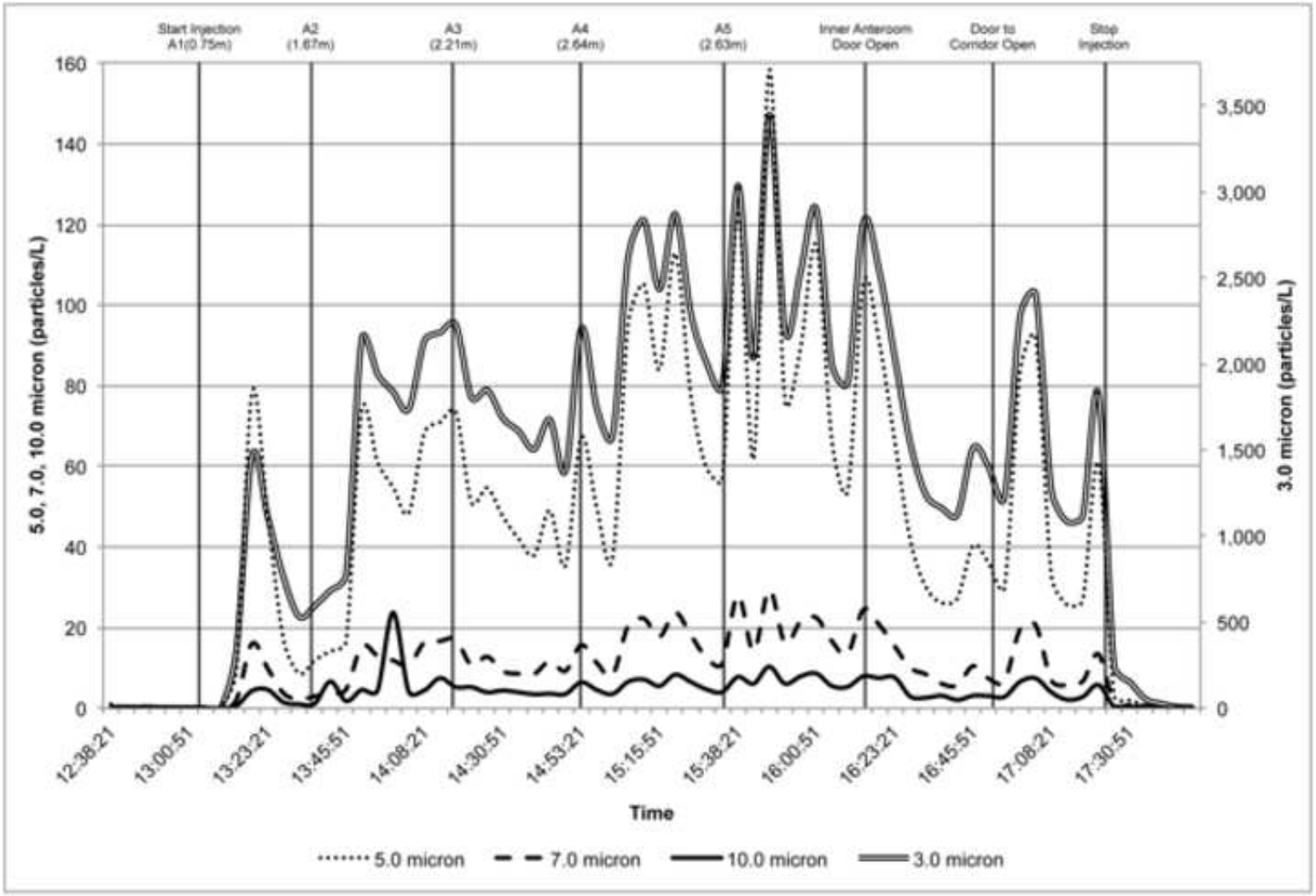

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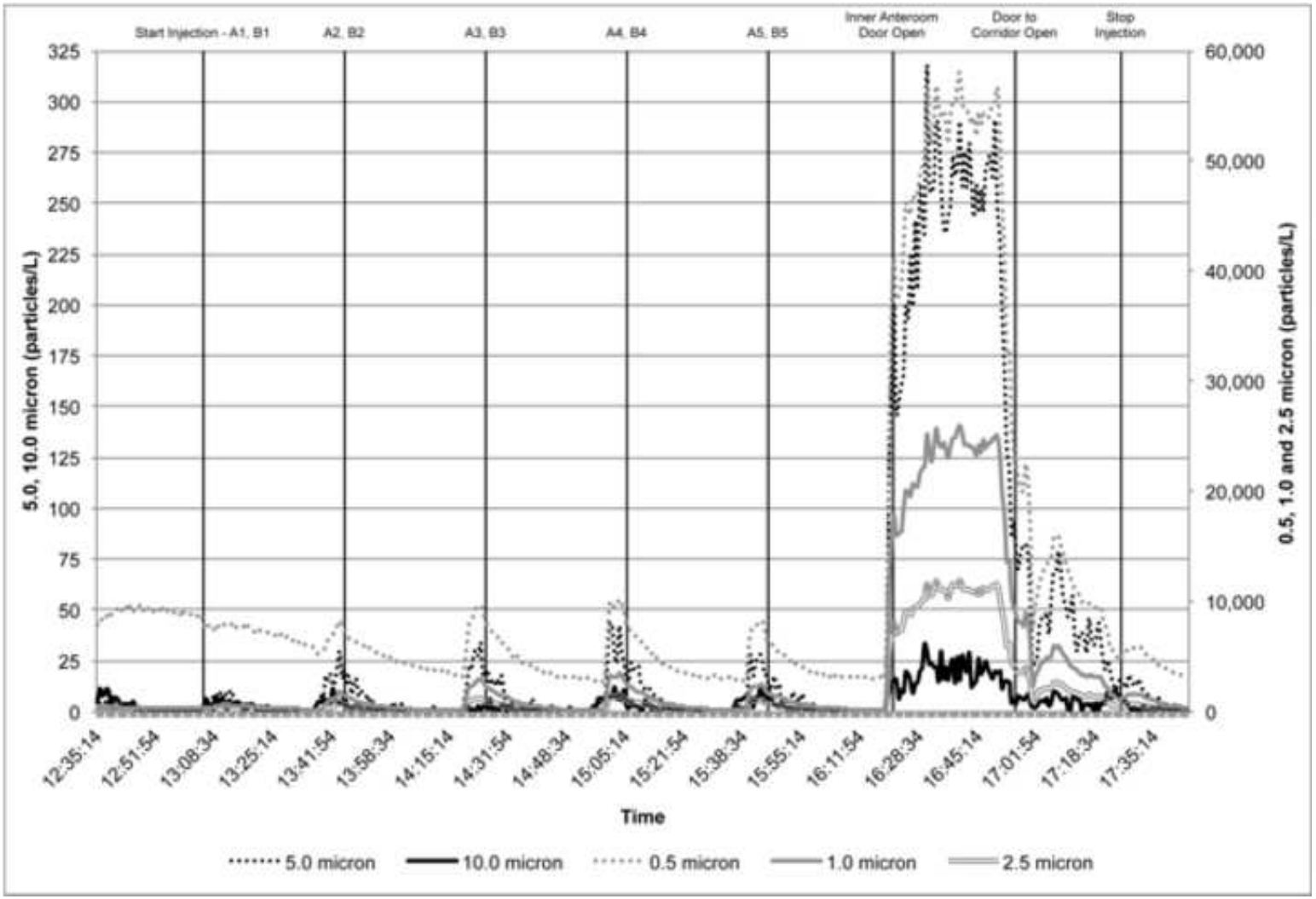

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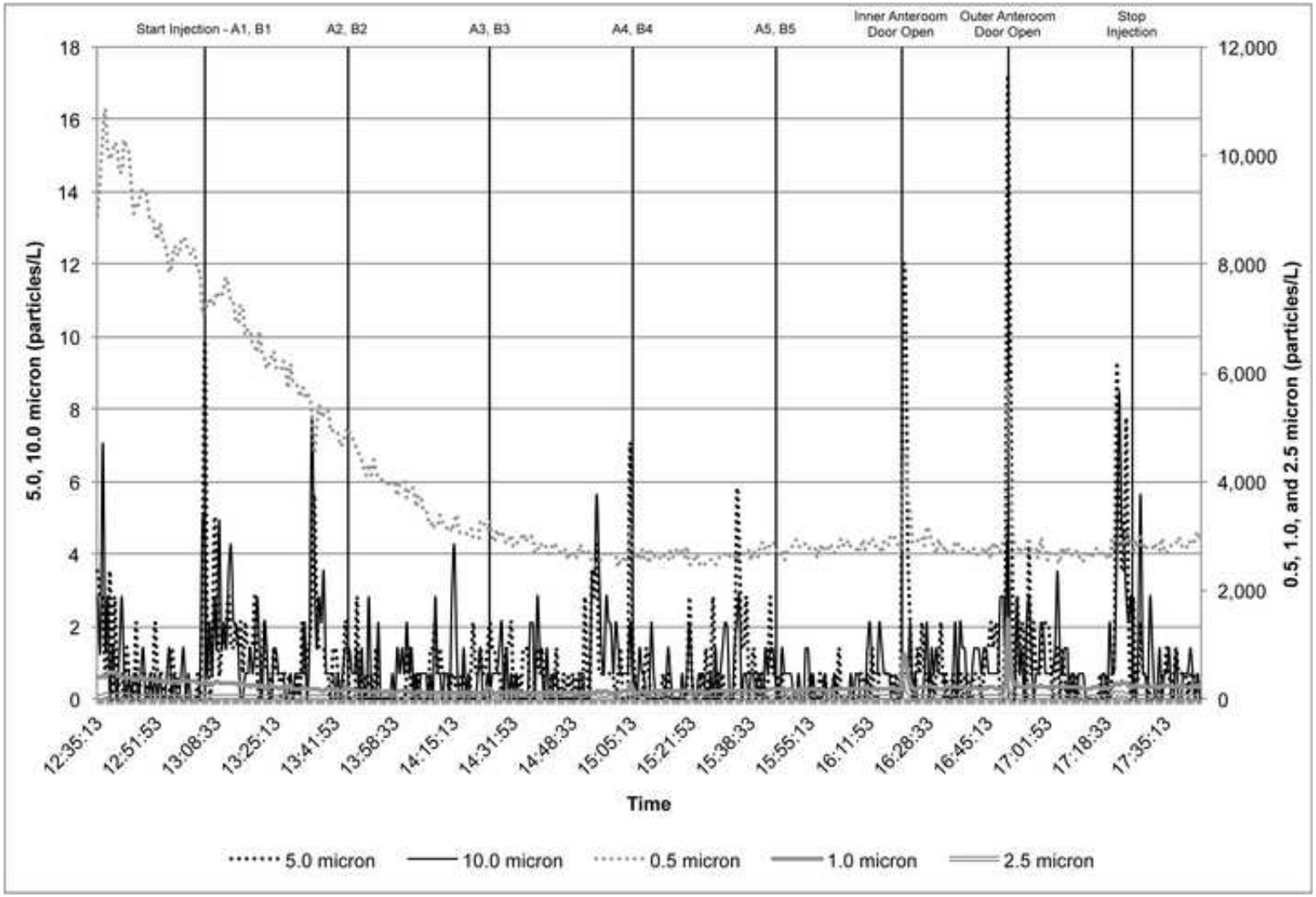

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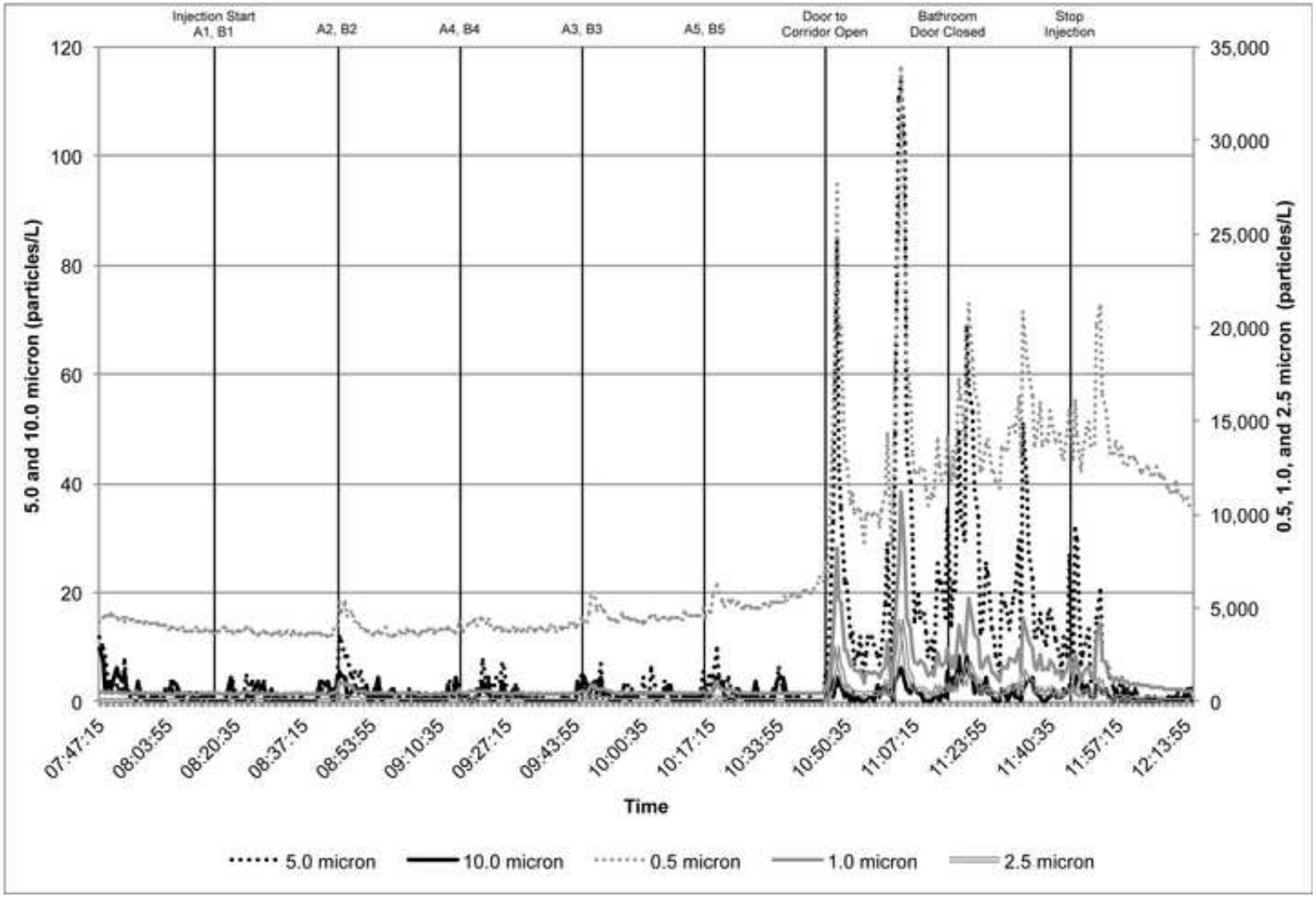

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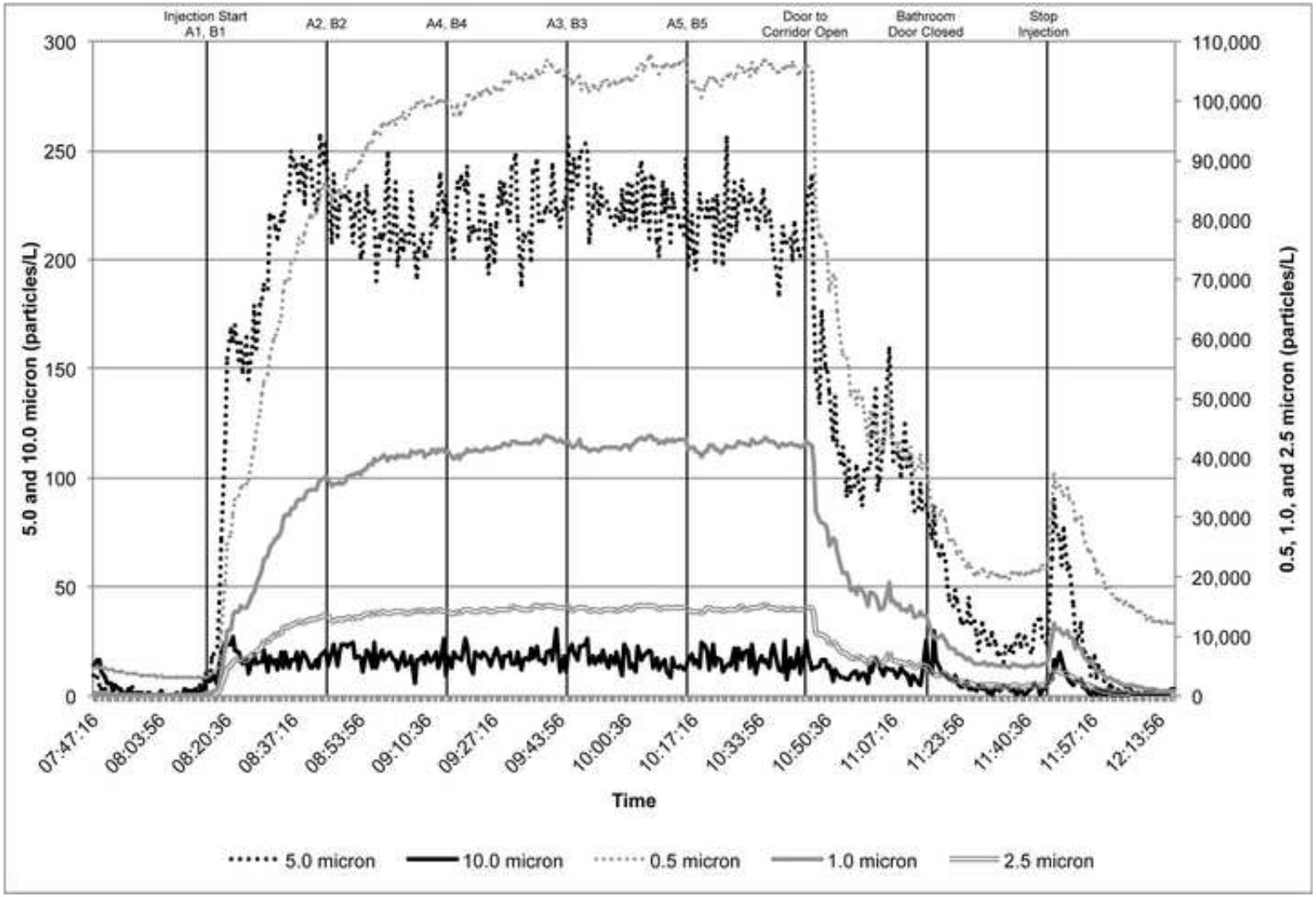

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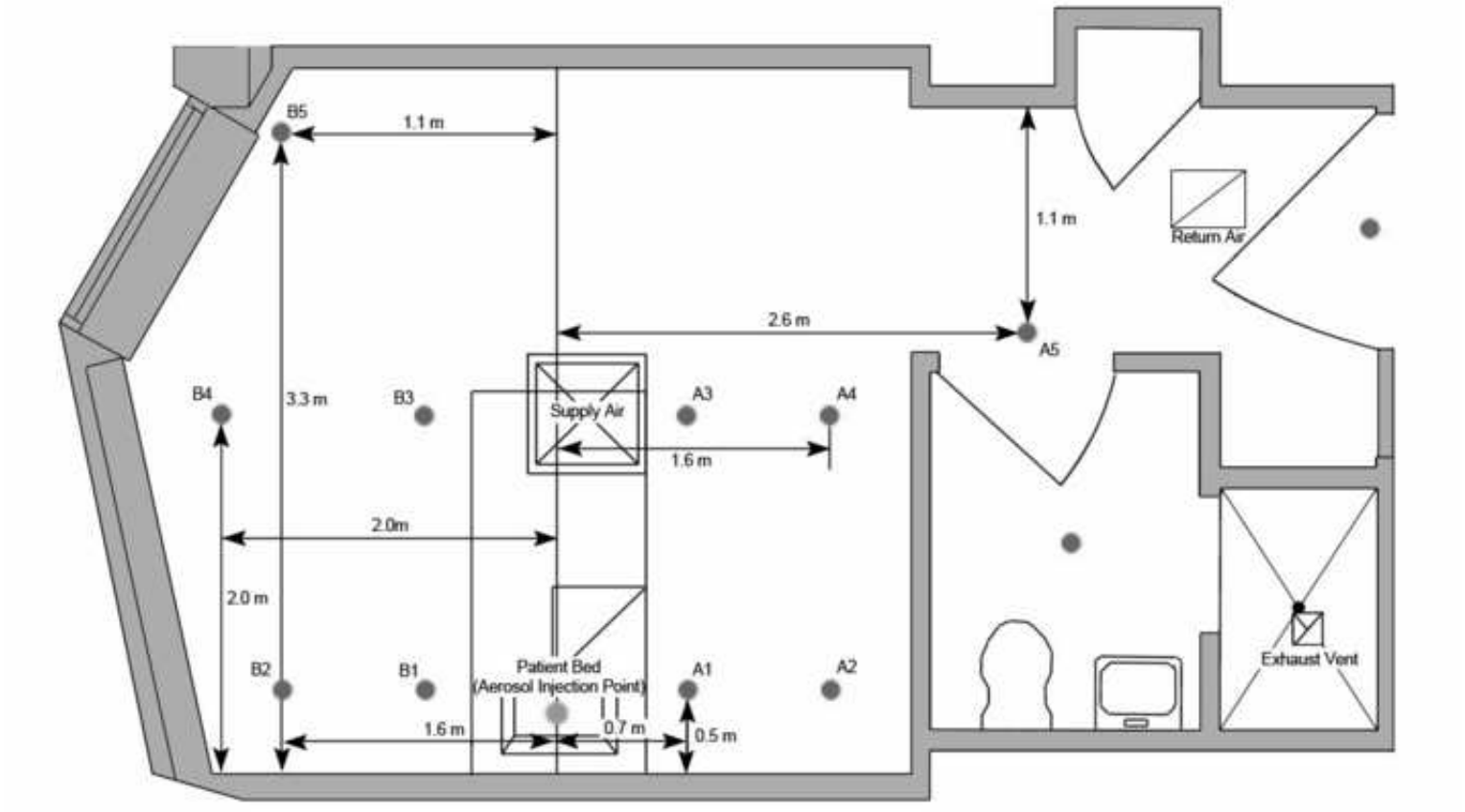

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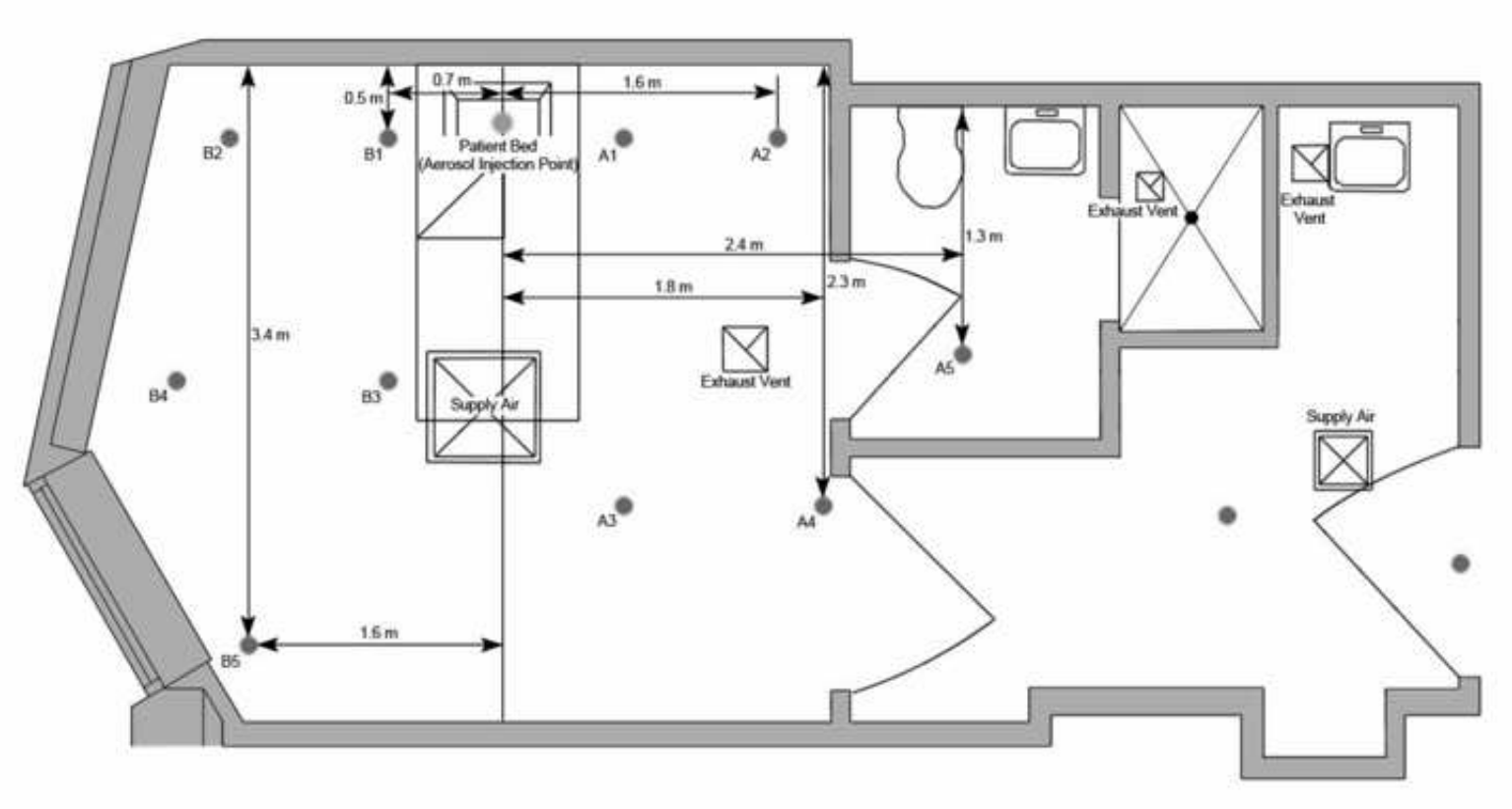

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TABLE 1. Summary of test procedure in general patient test room.

Duration	Instrument	Test Location	Description
0:00 - 0:30	NUCON F-1000-DD	A1, B1	Entry door closed, bathroom door open
0:30 - 1:00		A1, B1	Injection started
1:00 - 1:30		A2, B2	Position movement
1:30 - 2:00		A3, B3	Position movement
2:00 - 2:30		A4, B4	Position movement
2:30 - 3:00		A5, B5	Position movement
3:00 – 3:30		A5, B5	Entry door to corridor open
3:30 - 4:00		A5, B5	Bathroom door closed
4:00 - 4:30		A5, B5	Injection stopped, monitor to clear
0:00 - 4:30	Lighthouse HH-3016-IAQ	Static	In corridor, at top of entry door
0:00 - 4:30	Lighthouse HH-3016-IAQ	Static	In bathroom

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TABLE 2. Summary of test procedure in isolation patient test room.

Duration	Instrument	Test Location	Description
0:00 - 0:30	NUCON F-1000-DD	A1, B1	Entry door closed, anteroom door closed
0:30 - 1:00		A1, B1	Injection started
1:00 - 1:40		A2, B2	Position movement
1:40 – 2:20		A3, B3	Position movement
2:20 – 3:00		A4, B4	Position movement
3:00 - 3:40		A5, B5	Position movement
3:40 – 4:15		A5, B5	Anteroom door to isolation room open
4:15 - 4:45		A5, B5	Entry door to corridor open
4:45 - 5:15		A5, B5	Injection stopped, monitor to clear
0:00 - 5:15	Lighthouse HH-3016-IAQ	Static	In corridor, at top of entry door
0:00 - 5:15	Lighthouse HH-3016-IAQ	Static	In anteroom

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TABLE 3. Average change in particle concentration relative to particle size (1.0-10.0μm) and distance from aerosol injection point.

	General Patient Room		Isolation Patient Room	
Sample Height	Sample Locations A_2-A_5	Sample Locations B_2-B_5	Sample Locations A_2-A_5	Sample Locations B_2-B_5
0.6m	-2.7%	-3.4%	22.7%	21.6%
1.2m	-9.0%	-11.8%	4.5%	24.1%
1.8m	-12.5%	-13.9%	21.1%	31.1%