AMERICAN UNIVERSITY OF BEIRUT

DETERMINANTS OF INDOOR AIR QUALITY IN HOSPITALS: IMPACT OF VENTILATION SYSTEMS WITH INDOOR - OUTDOOR CORRELATIONS AND HEALTH IMPLICATIONS

by
ASHRAF ZIAD CHAMSEDDINE

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Title: Determinants of Indoor Air Quality in Hospitals: Impact of ventilation systems with Indoor – Outdoor Correlations and Health Implications

Indoor air quality (IAQ) is highly affected by outdoor emission sources particularly in congested urban areas invariably associated with vehicle-induced emissions as well as construction and industrial emissions. Since people spend most of their time indoors, the effect of IAQ on personal exposure and human health is often more pronounced than outdoor air. On the other hand, IAQ in certain sensitive environments such as hospitals is a critical factor for its occupants that could negate the purpose of the visit if IAQ deteriorates. Hospitals act as specific indoor environments with highly vulnerable individuals potentially exposed to various harmful air contaminants exacerbating health risks. Hence, this study involved a seasonal exposure assessment of IAQ determinants including temperature (T), relative humidity (RH), carbon monoxide (CO), carbon dioxide (CO_2), sulfur dioxide (SO_2), nitrogen oxides (NO/NO_x), particulate matter (PM_{10} and $PM_{2.5}$), and total volatile organic compounds (TVOCs) in hospital environments with particular emphasis on indoor-outdoor (IO) correlations, effective ventilation modes and associated health implications. Then air quality indices (AQIs) were tested and compared using field data from twelve hospital zones and the most robust index was obtained when coupling IAQ indicators with the effect of thermal comfort. This study also assessed the presence of influenza and respiratory syncytial viruses (RSV) in air samples collected inside patient rooms and investigated the potential risk for transmission for healthcare professionals (HCPs) and visitors. The results indicated that while indoor and outdoor CO levels were below air quality standards, measured $PM_{2.5}$ and PM_{10} concentrations at several locations exceeded the standards by 2 to 3.5 fold. We generally recorded higher indoor PM levels during the warm season, particularly during regional desert storm events. The ingress of particles from the outdoor to indoor environment was evident with high correlations between indoor and outdoor $PM_{2.5}$ (r between 0.83 and 0.92) and PM_{10} (r between 0.74 and 0.86) levels, particularly during the warm season. We detected influenza viral RNA in 51% of the air samples collected from influenza patient rooms, indicating a potential risk for nosocomial transmission via the airborne route. The day of admission to hospital was significantly associated with virus detection in the air sample, with the majority of cases being detected from patient rooms one day after admission. Computational fluid dynamics (CFD) model results identified two major “hot zones” inside patient rooms where indoor occupants are at a higher risk of viral infection. This study finally concludes with implications of high PM exposure and a suggested management framework for limiting such exposure and mitigating hospital-aquired infections (HAIs) in a most vulnerable segment of the community.

CONTENTS

ILLUSTRATIONS

Figure Page

TABLES

Table Page

CHAPTER I

INTRODUCTION

A. IAQ and Exposure

Most air pollutants are encountered in the troposphere as a result of photochemical reactions, biomass burning, vehicle and industrial emissions that could be of natural or anthropogenic sources [1–3]. These pollutants are classified as primary (emitted directly into the atmosphere including mineral dust, and gaseous precursors such as SO_2, NO_x and NH_3) or secondary pollutants (formed through chemical reactions). Other pollutants featuring CO, elemental and organic carbon are emitted from automotive, industrial sources and incomplete combustion [3–5]. Once in the atmosphere, pollutants are subject to dispersion, condensation, coagulation, physicochemical transformations, thus forming secondary pollutants [1,5]. In addition to their significant impacts on regional and global climate change, pollutants are associated with adverse health and environmental impacts (Appendix A) [4,6,7]. Outdoor air carrying various air contaminants may affect indoor air quality (IAQ) where occupants spend most of their time [8–10]. Thus the knowledge of the influence of ambient/outdoor air pollution from various natural and anthropogenic sources on levels of indoor contaminants in closed environments is imperative for assessing potential health impacts by comparing IAQ measurements to health standards and guidelines.

The importance of IAQ stems from the fact that people spend up to 90% of their time indoors and hence the concern about the quality of the air that they breathe [7]. For some, most of their time is spent inside buildings where they live, or where they learn, whereas for others it is in the place where they work with ~ 40 to 60 hours/week.

Although the same air might be exchanged between indoors and outdoors, yet indoor air is different from ambient / outdoor air since contaminants levels, sources and dispersion mechanisms can be different indoors compared to outdoors. For some pollutants the concentration indoors may be lower than outdoors, while for others, the indoor concentrations may be significantly higher than ambient levels [10,11].

Common indoor air pollutants include particulate matter (TSP, PM_{10} and $PM_{2.5}$), ozone (O_3), Radon, poly aromatic hydrocarbons (PAHs), NO and NO_2, SO_2, CO as well as a wide range of volatile organic compounds (VOCs) and bioaerosols [7,11]. In the aggregate of health effects, particulate matter (PM) can cause cardiovascular and respiratory diseases because when coarse particles ($PM_{10-2.5}$) are inhaled, they reach the upper parts of the lungs, pharynx and trachea [7,12,13] whereas fine particulate matter ($PM_{2.5}$) with less than 2.5 µm in diameter are considered more dangerous because they penetrate deeply into the bronchi and alveoli regions of the lungs provoking lung cancer and respiratory diseases [12,13]. Carbon monoxide (CO) is an odorless and colorless gas that is generated by incomplete burning of carbon-based fuels and its exposure is dangerous to human health as it reduces the blood's ability to carry oxygen [14,15]. It has an affinity for the oxygen carrying sites on the hemoglobin in the blood of 210 times greater than oxygen [1,14], hence the higher the CO levels, the greater the displacement of oxygen occurs, and the more oxygen deficient the individual becomes. Initial symptoms associated with CO exposure include shortness of breath on mild exertion, mild headaches, listlessness, and nausea (see Appendix A) [14,15]. As exposure increases, the individual may experience severe headaches, mental confusion, dizziness,

nausea, rapid breathing, and fainting on mild exertion. Carbon dioxide (CO_2) is also an odorless and colorless product of carbon combustion. Indoors, where the primary CO_2 source is human metabolism and breathing, CO_2 levels are usually greater than outdoor levels. Other common indoor sources include gas-cooking appliances, space heaters, wood-burning appliances, and tobacco smoke as well as combustion by-products (automotive traffic), compressed CO_2 (fire extinguishers), dry ice, and aerosol propellants [1,16]. Exposure to high CO_2 levels (> 1000 ppm) is an important risk factor of sick building syndrome (SBS) [10,16]. Sulfur dioxide (SO_2) is generated in the atmosphere from coal-fired power plants, anthropogenic, mineral and volcanic sources [1,3]. It is corrosive and toxic and at high concentrations can cause life threatening pulmonary edema where coughing, shortness of breath, difficulty in breathing and tightness in the chest can be experienced [1,17]. Nitrogen oxides (NO_x) include nitrogen oxide (NO) and nitrogen dioxide (NO_2) that are the main nitrogen containing compounds emitted into the atmosphere (see Appendix A). Vehicular, industrial and combustion emissions are considered the major sources of NO_x and particularly NO_2 [1–3,11]. Nitrogen oxides may cause nausea, irritation in the eyes and nose, fluid forming in lungs and shortness of breath. At high levels, nitrogen oxides may lead also to swelling of the throat, long-term asthma, and respiratory diseases [1,2,11]. VOCs released from many housekeeping and maintenance products, building materials, industrial emissions, furnishings, equipment, pesticides and insecticides [1,18]. Food manufacturing and industrial operations are equally known to generate organic chemicals that include VOCs, some of which are toxic, mutagenic and carcinogenic and

can cause adverse health effects [1,7,18]. Typical symptoms of VOC exposure include headache, nausea, and irritation of the eyes, nose, and throat (Appendix A) [18,19].

In general, there is limited to a lack of information in the bioaerosols research field regarding what type of microorganisms or components may be found in different environments, how do they become aerosolized, and their impacts on human health and the environment. Bioaerosols are generated by sneezing, coughing, or vomiting and are important transmission route for infectious agents and include typical microorganisms of biological origin such as bacteria, archaea, fungi and viruses [20]. Studies have shown that the inhalation impact of bioaerosols components on human health depends on their concentration, infectivity, immunogenicity and particle size [20–23]. Bioaerosols can have significant adverse effects on human health as they may initiate an infection in the respiratory tract or other parts of the body through transmitting infectious microorganisms. People who are exposed to bioaerosols on a daily basis can develop allergic and chronic inflammatory responses as well as respiratory and cardiovascular diseases [21,24–26]. The upper and lower respiratory tracts can be both exposed to these bioaerosols. Typical examples of airborne respiratory diseases include tuberculosis, influenza, and legionellosis. Viruses such as influenza, respiratory syncytial virus (RSV), rhinovirus, adenovirus, and coronavirus can lead to infections in the upper respiratory tract [24,26,27]. On the other hand, bronchitis and pneumonia that are primarily caused by bacteria such as *Legionella spp*., *Streptococcus spp*., and *Haemophilus influenza* can adversely affect on the lower respiratory tract, which acts as a site for chronic diseases [28,29]. Bioaerosols have frequently been examined through

culture methods by using specific conditions and growth media [30–32]. However, only a small portion of the total bioaerosols in any environment is culturable because there are many biological components that cannot be determined through culture media, such as cellular fragments that are collected by air sampling or aerosolization processes [33,34]. The non-culturable microorganisms may be infectious, can cause inflammation and exacerbate existing respiratory diseases. Appendix B highlights the main non-culturable microorganisms and their health impacts that should be looked for in exposure and risk assessment studies. Despite their major impact on human health, the science of bioaerosols remains ambiguous and misunderstood in indoor environments, particularly in healthcare facilities and hospital settings. For critical indoor environments such as hospitals, information about viruses is still lacking and comprehensive knowledge and understanding of the total biological burden at these facilities remains incomplete.

The concentration levels of air contaminants to which indoor occupants are exposed can vary dramatically based on the types and sources of contaminants, proximity of sources, emission rates, the design, age and construction of the building, the ventilation systems and whether indoor air is frequently regulated and controlled. Thus in light of health effects, quantification and characterization of particulate matter (PM_{10} and $PM_{2.5}$), various gaseous contaminants (CO, CO_2, SO_2, NO_x and TVOC) and bioaerosols such as viruses became essential particularly when migration of air pollutants from outdoor spaces to the indoor environment is significant. The outdoor air quality has a major effect on pollution levels of indoor air in which occupants spend

most of their time [8,10,35]. Thus, a comprehensive understanding of the influence and impact of ambient air pollution from various natural and anthropogenic sources on the levels of air contaminants in indoor environments is vital and noteworthy not only for human health effects but also for exploring the migration of air pollutants and pathogens from outdoor spaces to the indoor environment. Therefore the characterization program involves the concurrent monitoring of ambient and indoor concentrations of PM_{10}, $PM_{2.5}$, CO, CO_2, SO_2, NO_x, TVOC and the assessment of bioaerosols to examine the impact of outdoor air on IAQ and investigate the potential risk for transmission of viruses for healthcare professionals (HCPs) and visitors in critical environments such as hospitals.

B. Hospital Buildings

Hospitals and healthcare facilities represent specific indoor environments with highly vulnerable individuals. Hospital buildings are constructed and designed to be occupied by medical and operational staff, patients, and visitors. The standards and requirements for hospitals must accommodate for the comfort of a wide range of occupants with an equally wide range of vulnerabilities [10,36–38]. Acceptable IAQ in hospitals can be obtained through the feedback of its occupants whereby an 80% satisfaction represents a good performance [9,39,40]. Hospitals should be rated as high performance buildings in terms of environmental and air quality to enhance and retain patients' healing process as well as working staff efficiency [41]. Moreover, hospital's primary function is centered on patient care and therefore the need to pay particular

attention to air quality inside hospitals cannot be compromised or overlooked. Several environmental indicators can be identified by occupants as contributing factors to the quality of air inside hospitals [9,35,42], however these factors would not be the same for all indoor occupants since different people have varying needs and expectations of the indoor environment. Although some studies have targeted the assessment of the influence of indoor environments on occupants and staff work efficiency in indoor environments [43–46], yet they have not examined the environmental and air quality in hospitals adequately. Life threatening infections could be acquired in hospitals as a result of poor IAQ, and patients are at risk of developing infections when hospitals' indoor environment is affected by various indoor and outdoor sources of air contaminants. The health-related problems created by air pollutants inside hospitals far outweighs the challenges posed by ambient air pollutants if not properly controlled and managed [10,35,47]. The presence of air contaminants indoors such as PM_{10} and $PM_{2.5}$, CO, SO_2, TVOC and bioaerosols may influence occupants' cognitive process, which also affects creative task performance as well as provoke pulmonary and cardiovascular diseases [13,35,47–49]. Medical treatments, medicines and cleaning solutions in hospitals can also affect IAQ, thus increasing the levels of TVOCs and particularly formaldehyde (HCHO) levels in the air [50,51]. Similarly, indoor activities may affect the PM level in the air[10,37,52]. Outdoor air plays an equally important role in polluting indoor environments, whereby exposure to CO and PM increases the risk of sick building syndrome (SBS) [52,53]. Studies have also indicated that exposure to

TVOCs and bioaerosols can increase the risk of allergic diseases, cause damage to nervous system and may cause cancer [19,30,35,51,54].

Indoor environmental quality (IEQ) has been drawing much attention due to its high impact on occupants of indoor environments. When designing a physical environment for indoor occupants; their response is invariably considered in defining indoor well-being, satisfaction, and comfort [55,56]. As such, studies on indoor environments considered IAQ and thermal comfort as two main parameters that determine indoor comfort level of building environments [55,57,58]. Hospital buildings on the other hand, include three different end users: medical employees, patients and visitors who have different perceptions and needs with regard to the environment in which they work or spend considerable time while waiting to return home. Since hospitals act as primary healthcare centers for patients to recover from illness, such indoor environments should demonstrate improved environmental quality that can reduce the recovery period of patients, enhance visitors comfort and increase staff productivity [55]. In hospitals, the time spent indoors depends on the health condition of patients, allocated time for visitors, and the working shifts of the staff rendering hospital environments special with a high occupancy ratio continuously. While schools and office buildings close for some days and remain unoccupied for certain periods of time, hospital buildings are always occupied as long as corresponding facilities are operational [9,56]. Therefore maintaining high standards that reflect the requirements and expectations of indoor occupants is a continuous concern for healthcare providers at hospitals.

C. Ventilation and Thermal Comfort

The design of heating, ventilating and air conditioning (HVAC) systems is a special field of engineering that is crucial for all types of buildings, including residential, commercial and primary and healthcare facilities such as hospitals. Health care facilities have distinct design criteria, where the HVAC requirements include regulating temperature (T), relative humidity (RH) and space pressurization, as well as filtration of supplied air, allowable recirculation of the air, and the effectiveness of air handling units system [59–61]. In addition, the HVAC systems of healthcare facilities and hospitals support a variety of medical functions, practices and ventilation systems that are critical to health and safety including infection control, environmental and IAQ control for specific medical functions, hazard control and life safety. The ventilation system is a major key determinant of IAQ in hospitals with two main types: natural and mechanical, both relied upon to control and remove air contaminants and hazardous chemicals [10,35,47]. In hospitals, mechanical ventilation systems can affect the transport and distribution of air contaminants with high ventilation rates diluting levels of airborne microbes [62,63]. Wu et al. [64] reported lower levels of bioaerosols in offices with air handling units (AHU) compared with fan cooling units (FCU). Zuraimi et al. [65] found higher indoor outdoor (IO) ratios of $PM_{2.5}$ in child care centers with natural ventilation compared with mechanical ventilation. However none of the studies seem to have addressed and associated the distribution of bioaerosols, chemical pollutants and IAQ in hospitals with various ventilation modes. Ventilation in hospitals

and similar healthcare facilities is critical for both patients and medical staff as it provides thermal comfort and maintains proper air exchange and IAQ [35,66]. Consideration must be equally given to mechanisms of filtering outdoor air transported indoors to maximize ventilation benefits and for developing IAQ guidelines for critical environments like hospitals. Best practices and the American Society of Heating, Refrigeration, and Air Conditioning Engineers (ASHRAE) design criteria for healthcare facilities are presented in Appendix C.

Thermal comfort in hospitals is of great importance in accomplishing safe and high standard indoor environmental conditions [44]. Temperature and relative humidity (RH) can activate or deactivate viruses and inhibit or increase the growth of bacteria as some airborne bacteria can survive and accumulate in a humid environment [44,46,67]. High temperatures may cause an increased out- gassing of toxins from building materials and low temperature can cause occupant discomfort including shivering, inattentiveness and muscular and joint tension; similarly, low RH increases the susceptibility for respiratory diseases and lead to other discomforting human effects such as drying nose, eyes, skin and throat irritation [46]. On the other hand, Murphy [67] has different guidelines for indoor temperature, humidity and air-change requirements for operation rooms, and indicated that medical staffs, namely surgeons expect a lower room temperature than those stated in the ASHRAE guidelines (see Appendix C). The need for lower air temperature, high relative humidity and the condensation risk in surgery rooms, which are the most critical working environments in hospitals, present a major concern [67]. Higher indoor temperatures might cause

discomfort and more favorable conditions for bacterial transmission from and to the patient [68]. Relative humidity levels have to be within the accepted levels; else higher levels of humidity can cause growth and transfer of bacteria as well as thermal discomfort, and low humidity can increase susceptibility to eye discomfort, irritation and respiratory diseases [46,68]. Although temperature and relative humidity criteria in indoor spaces of healthcare and hospital buildings are influenced and affected by infection control measures as well as thermal comfort [44,46,59,69,70], limited work has examined thermal comfort parameters in various indoor working spaces of hospitals other than operating rooms, as well as the effect of thermal comfort on productivity levels of staff and comfort conditions of patients and visitors at hospitals. While there is a considerable literature on ventilation and thermal comfort studies for indoor environments [40,57,58,71,72], hospitals received less attention. The interaction between indoor occupants and the physical environments at hospitals requires further examination to better understand the link between hospital buildings and IAQ as well as the thermal comfort of occupants in various hospital areas under different ventilation modes.

D. Research Questions and Objectives

Historically, public concern with IAQ has evolved as pollutants from various emission sources were identified in indoor environments at levels associated with adverse health effects [8,10,37,73,74]. Since people spend most of their time indoors, the effect of IAQ on personal exposure and human health became invariably more

pronounced than the outdoor air [7]. This is particularly true in certain sensitive environments such as hospitals, where IAQ is a critical factor for patients and its poor quality could negate the purpose of the visit if it deteriorates and exacerbate health risks [49,75].

While recent studies [62,76–81] have focused on air quality in various indoor environments, limited work examined IAQ in hospitals, often targeting a limited number of indicators (Table 1). Nardini et al. [82] measured $PM_{2.5}$ in two Italian hospitals in which medical offices, halls and waiting rooms were selected as monitoring areas, while Ostro et al. [83] estimated the risks of exposure to $PM_{2.5}$ in hospital admissions for respiratory diseases among children. Erdogan et al. [19] determined the levels of total volatile organic compounds (TVOC), CO and CO_2 concentrations in a hospital building in Istanbul. More recently, Jung et al. [35] examined the distribution of indoor air pollutants such as CO, CO_2, TVOC, $PM_{2.5}$ and PM_{10} in various working areas of hospitals in Taiwan, but didn't explore viruses in such healthcare environments. This research targets the assessment of IAQ determinants in hospitals located in congested urban areas with particular emphasis on indoor-outdoor (IO) correlations and associated health implications. For this purpose, a field- monitoring program was implemented at several hospitals of different settings (i.e. urban versus rural) and measured the concentrations of several air quality indicators commonly associated with indoor and outdoor emission sources including carbon monoxide (CO), carbon dioxide (CO_2), particulate matter PM_{10} and $PM_{2.5}$, sulfur dioxide (SO_2), nitric oxides (NO/NO_x) as well as total volatile organic compounds (TVOC). In addition, influenza and RSV viruses

and basic indoor thermal comfort variables such as temperature (T) and relative humidity (RH) were monitored concomitantly.

Table 1. Air quality indicators from previous studies and this research study

Study	Indicator									
	Bioaerosols*	**PM_{10}**	**$PM_{2.5}$**	**CO**	**CO_2**	**SO_2**	**NO/NO_x**	**TVOC**	**T**	**RH**
Nakata et al. [84]				×		×	×			
Nardini et al. [82]		×	×							
Ostro et al. [83]			×							
Erdogan et al. [19]				×	×			×		
Wan et al. [85]		×	×		×				×	×
Slezakova et al. [36]		×	×							
Jung et al. [35]		×	×	×	×			×		
This research study	×	×	×	×	×	×	×	×	×	×

*Including Infuenza and RSV viruses

Despite its complexity and importance, air pollution in indoor environments has been less thoroughly examined than the outdoor air. As people spend most of their time indoors, the quality of indoor air is a critical factor influencing human health [49,86]. This is in particular true for critical environments such as hospitals and healthcare centers, where there is a need for assessing IAQ to gain a better understanding of the comfort of indoor occupants and health risks associated with the exposure to bioaerosols and various air quality indicators including PM_{10}, $PM_{2.5}$, CO, CO_2, SO_2, NO/NO_x, and TVOC. Such studies are equally important for implementing informed decisions on IAQ management to reduce risks and help maintain a clean and healthy environment in hospitals. Previous studies have addressed a limited number of

IAQ influencing parameters with a lack of comprehensiveness towards understanding IAQ determinants in hospitals (Table 2).

Table 2. Determinants of IAQ from previous studies and this research study

		Influencing parameters											
Reference	**Indicators**	Indoor levels	Outdoor levels	I-O correlation	Age, type & setting	Seasonal variation	Spatial variation	Thermal comfort	Ventilation mode	CFD modeling	Laboratory Analysis [a]	Health risk	IAQ management
Nakata et al. [84]	CO, SO_2, NO_x	×											
Nardini et al. [82]	PM_{10}, $PM_{2.5}$	×	×										
Ostro et al. [83]	$PM_{2.5}$		×								×		
Erdogan et al. [19]	CO, CO_2, TVOC	×					×						
Wan et al. [85]	PM_{10}, $PM_{2.5}$, CO_2	×				×		×					
Slezakova et al. [36]	PM_{10}, $PM_{2.5}$	×									×	×	
Jung et al. [35]	PM_{10}, $PM_{2.5}$, CO, CO_2, TVOC	×	×	×			×		×				
This research study	Viruses, PM_{10}, $PM_{2.5}$, CO, CO_2, SO_2, NO_x, TVOC	×	×	×	×	×	×	×	×	×	×	×	×

[a] Includes PCR analaysis and culture methods.

This research study adds to the body of existing literature and attempts to fill in some gaps by evaluating the exposure to a wide range of air quality indicators (CO, CO_2, SO_2, NO/NO_x, TVOC, T, PM_{10}, $PM_{2.5}$, and viruses including Influenza and RSV). A first attempt at simulating different indicators was carried out with the aim to understand transport mechanism and spatial distribution within a hospital confines. Various influencing parameters that affect IAQ in hospitals were examined including the effect of outdoor concentrations, ventilation modes, temporal (seasonal) and spatial (different working areas) variability. The significance of the proposed research spreads

across several stakeholders including end-users (medical staff, visitors and patients), infection control and physical plant departments of hospitals, the public health sector, as well as local and regional environmental agencies. In broad terms, the design and implementation of appropriate intervention air quality management policies that will positively impact health effects exposure to various air quality contaminants in hospitals require answers to the following research questions:

- What are the main factors that affect IAQ in hospitals?
- What are the main substrains/types of influenza virus present at medical facilities?
- How do different working areas and ventilation modes affect IAQ in hospitals?
- How do seasonal and spatial attributes affect IAQ in hospitals?
- How do outdoor concentrations of pollutants affect indoor levels in hospitals?
- What would be the impact of outdoor air on IAQ and what are the potential outdoor and indoor sources that may contribute to high levels of indoor contaminants in hospitals?
- What are management alternatives to improve its present IAQ and future comfort performance in hospitals taking into account potential conflicting views and interests amongst stakeholders?

Indoor air pollution is likely to remain a critical health risk particularly in developing countries in the absence of successful intervention programs. Understanding how indoor exposure relates to outdoor concentrations in indoor environments is imperative for the assessment of policy interventions and decision making towards

alleviating potential adverse environmental and health impacts. Therefore, the objectives of this research include:

- Characterization of IAQ indicators at hospitals (T, RH, CO, CO_2, SO_2, NO/NO_x, TVOC, PM_{10}, $PM_{2.5}$, influenza and RSV viruses) under varied ventilation modes and different locations i.e. wards within hospital confines.
- Correlation of indoor-outdoor indicators to identify potential sources of indoor contaminants.
- Development of an IAQ index with consideration to both thermal comfort variables and air quality determinants in hospitals.
- Estimating human exposure risk to influenza and RSV viruses inside patient rooms of hospitals.
- Simulation of flow patterns and concentration profiles within hospital confines i.e. spatially, using Computational Fluid Dynamics (CFD) to gain a better understanding of contaminants spread as a function of the ventilation system operations.
- Defining a management framework towards improving IAQ in hospitals.

E. Research Innovations

The flow of chapters in this dissertation will be as follows: Chapter I – Introduction, Chapter II – Field sampling and experimental procedures, Chapter III – Seasonal variability and indoor-outdoor (IO) correlations, Chapter IV – Application and development of an indoor air quality index, Chapter V – Detection of viruses in patient

rooms, Chapter VI – CFD modeling, Chapter VII – Air quality management in hospitals, and finally Chapter VIII – Conclusions and furture work. No specific studies have adopted such a comprehensive and systematic approach for examining IAQ and thermal comfort in hospital environments. The data collected and results obtained are fundamental for designing better IAQ guidelines and management frameworks for indoor environments of great diversity and importance such as hospitals. Ensuring clean indoor air at hospitals is imperative with a need to increase awareness and knowledge of IAQ at such critical environments and its influence on health and comfort. Besides its comprehensiveness, the innovation of this research study includes:

- A comprehensive description of biological and chemical components of particles in critical environments such as hospitals by adopting new and novel sampling approaches with emphasis on indoor-outdoor (IO) correlations. The analytical methods will directly characterize the total genetic components of bioaerosols (i.e. viruses) using PCR, in addition to understanding their transmission routes inside patient rooms.
- The development of a new and robust indoor air quality index (RIAQI) to help in assessing IAQ and thermal comfort in hospitals. The composite index provides a risk assessment tool that hospital administration and public health practitioners can make use of to ensure safe conditions in critical indoor environments.
- The application of CFD to simulate the spatial distribution of air contaminants (such as $PM_{2.5}$) and air flow patterns carrying influenza and RSV viruses under

varied ventilation modes in hospitals towards improving the understanding of exposure risk within hospitals with corresponding health implications.

CHAPTER II

FIELD SAMPLING AND EXPERIMENTAL PROCEDURES

A. Study Area

We monitored air quality in two hospitals in mixed residential/commercial areas and a third in a rural setting. The first hospital (HOSP-A with 450 beds) was established in 1901 and is in Beirut in close proximity to three main arterial roads. The second hospital (HOSP-B with 50 beds) was established in 1982 and is approximately 200 meters from HOSP-A. The third hospital (HOSP-C with 35 beds) was established in 2010 and is in a rural area at an elevation of 1100 m, near a pine forest. In this research study, we measured the seasonal variations and concentration profiles of several air pollutants (CO, CO_2, SO_2, NO/NO_x, $PM_{2.5}$, PM_{10}, and TVOC) indoors and outdoors at the three hospitals in an effort to explore potential exposure under different ventilation modes with an emphasis on I/O correlations in such critical indoor environments. The overall monitoring and assessment framework that was adopted is depicted in Figure 1.

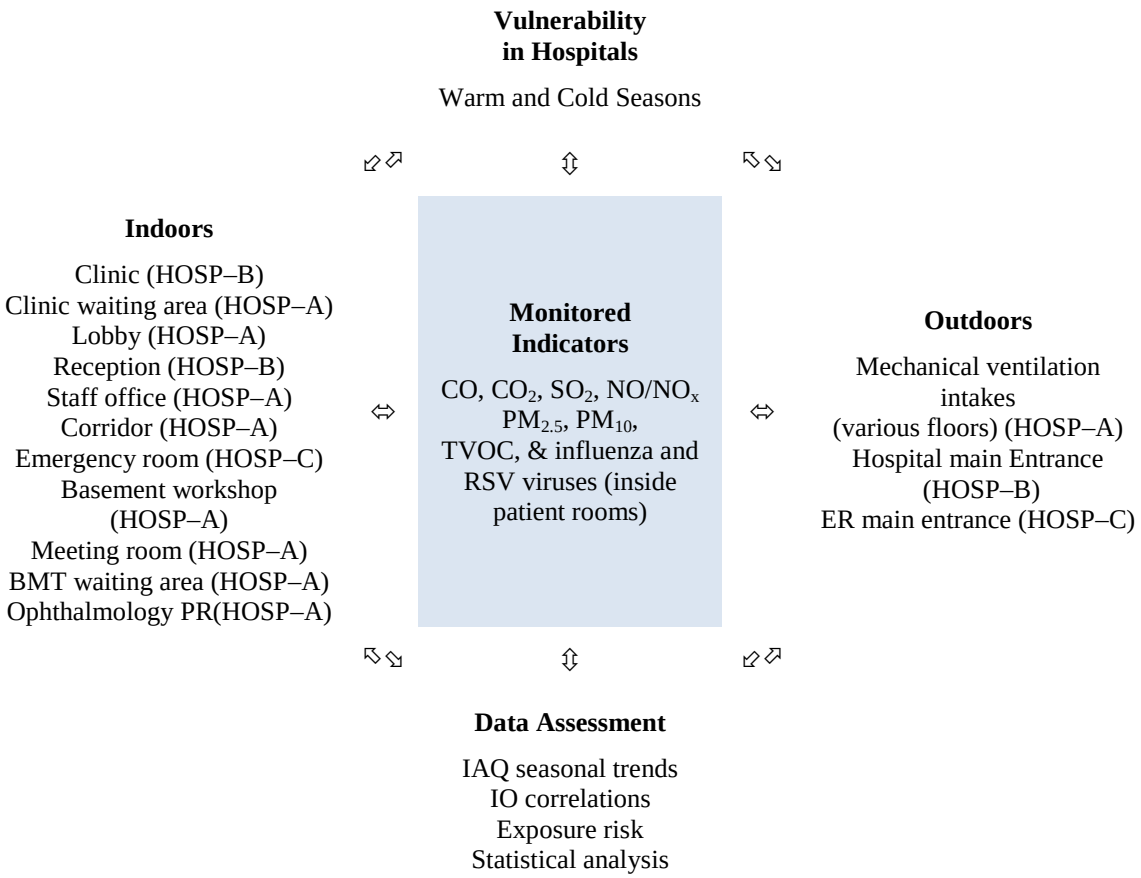

Figure 1. Adopted monitoring and assessment framework

We targeted a total of 12 working areas in the 3 hospitals including clinics, clinic waiting areas, lobbies, reception areas, staff offices, corridors, paediatric patient rooms (PPRs), emergency rooms (ERs), basement workshops, meeting rooms, bone marrow transplant (BMT) waiting areas, and ophthalmology patient rooms (OPRs). The total number of samples collected at each location varied at 18, 16, 21, 18, 15, 20, 6, 14, 16, 15, 12, and 16 samples, respectively. We conducted the monitoring during the periods March–April 2013, November–December 2014, February–April 2015, October–December 2015, February–April 2016, and October–December 2016. Concurrently with

IAQ sampling, we monitored the fresh air intake at each hospital. At HOSP-A, the fresh air intakes are in two separate buildings on the 8th floor facing a parking lot and a construction site and on the 11th floor facing the sea and a busy road. HOSP-B was not equipped with a mechanical ventilation system and as such we considered the first floor entrance as the fresh air intake. The entrance faced two busy streets and a construction site. Similarly, for HOSP-C, which was also not equipped with a mechanical ventilation system, we considered the ER main entrance as the fresh air intake. Note that both HOSP-B and HOPS-C had windows that allow natural ventilation, while HOSP-A did not have functional windows.

B. Equipment Selection and Monitoring Program

We simultaneously monitored indoor and outdoor levels of CO, CO_2, $PM_{2.5}$, PM_{10} and TVOC during daytime working hours (9:00 am–5:00 pm) and reported hourly average concentrations. We used two Langan air quality analyzers (Model L76x, San Francisco, CA, USA) to measure real-time CO and CO_2 concentrations (ppm). We measured PM_{10} and $PM_{2.5}$ (µg/m^3) using two DustTrak™ II Aerosol Monitors (Model 8532, TSI Corporation, Shoreview, US) equipped with a light-scattering laser photometer. Sulfur dioxide (SO_2) and nitrogen oxides (NO/NO_x) were also monitored using an E4500 Portable Emission Analyzer (E Instruments International, LLC 402 Middletown Blvd. Suite 216 Langhorne, PA 19047). We monitored TVOC levels (ppm) using two real-time PhoCheck Tiger photoionization detection (PID) instruments (Ion Science Ltd., The Way, Fowlmere, UK). Coriolis µ Biological Air Sampler (Bertin

Instruments, FRANCE) was used to collect the air samples at two locations within the patient's room. An ACCUBALANCE Model 8380 Air Capture Hood (TSI Corporation 500 Cardigan Road Shoreview, MN 55126) was used where possible to determine the airflow rate. At locations where the capture hood cannot be fitted, both TSI VELOCICALC® Air Velocity Meter Model 9535 (TSI Corporation 500 Cardigan Road Shoreview, MN 55126) and Model 9545 (TSI Corporation 500 Cardigan Road Shoreview, USA) were used to monitor temperature, relative humidity, air velocity and calculate corresponding air flow rates. Calibration tests were done on all the equipment and we collocated pairs of duplicate instruments at the beginning of each data collection campaign to ensure consistent readings. All monitoring equipment was positioned at the breathing zone (i.e. 1.5 m above ground level) and synchronized to the clock of a computer used for data acquisition. Appendix D provides a summary of equipment, specifications, and calibration tests for implementing the field monitoring program with Figure 2 depicting the overall sampling setup. We then compared our measurements to ambient and IAQ guidelines and standards (see Appendix E).

We also examined seasonal variations in air quality whereby samples collected during the months of March, April, and October were considered representative of the warm season that is affected by regional dust storms originating from the deserts of the Middle East and African Sahara. We characterized the impact of dust storms by assessing the 24-hour backward wind trajectories using HYSPLIT [87,88]. We considered the data collected during November, December, and February to be

representative of the cold season. Table 3 presents the seasonal number of indoor and outdoor samples collected at each hospital.

Figure 2. Setup adopted for field monitoring

Table 3. Number of indoor and outdoor samples collected at each hospital by season

Hospitals	*Warm*		*Cold*	
	Indoor	Outdoor [a]	Indoor	Outdoor [a]
HOSP-A [b]	83	83	54	54
HOSP-B	20	20	16	16
HOSP-C	8	8	6	6

[a] Outdoor (fresh air intakes) and indoor (working areas) monitoring were simultaneously conducted.
[b] Fifty one air samples were collected in patient rooms of HOSP-A during Influenza 2018 season for viral analysis.

C. Bioaerosol Sampling and Virology Analysis

Air sampling was conducted during the influenza season from January to March 2018 at a major tertiary care hospital (HOSP-A) located in Beirut area, Lebanon. Aerosol samples were collected from the rooms of patients with laboratory-confirmed influenza or RSV infections. Coriolis µ Biological Air Sampler was used to collect the air samples at two locations within the patient's room. One sample was taken 0.3 m apart from the patient simulating the distance to the patient's visitors or healthcare providers performing near bed procedures, while the other was 0.5 m away from the door and 2.2 m away from the patient's head (Figure 3), simulating the distance that physicians usually keep with influenza or RSV patients. For relatively smaller patient rooms, only one air sample was collected at 0.3 m to the subject. The air collected from patient rooms was aspirated for 10 minutes at a flow rate of 300 L/min and drawn into a collection tube containing 15 ml of sterile collection media. The air sampler was decontaminated with concentrated ethanol and air dried after each sample run to prevent potential carry-over contamination. Temperature and relative humidity were also monitored and recorded inside the patients' rooms using VELOCICALC ® Air Velocity Meter Model 9545 (TSI Corporation 500 Cardigan Road Shoreview, USA) to assess their effect on virus aerosolization. Patients' coughs and sneezes were counted during the monitoring program. Importantly, no aerosol-generating procedures such as bronchoscopy, intubation, open suction of airways, and nebulizer therapy were performed on patients during air sampling.

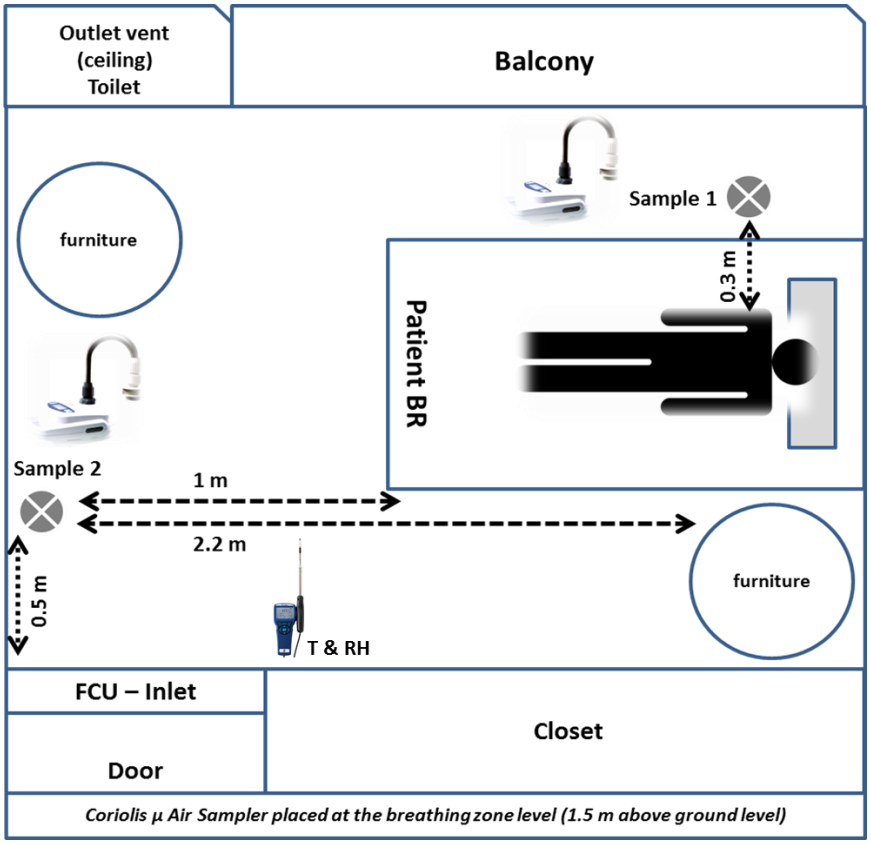

Figure 3. Field monitoring setup of bioaerosols inside patient room

The samples were immediately transported on ice to the laboratory where they were aliquot and stored at - 20 ^{o}C until further processing. RNA was extracted from a 1 mL sample using PureLink Viral RNA/DNA Mini Kit (Invitrogen) according to the manufacturer's specifications. The specimens were then screened by real-time PCR using primers and probes specific for influenza A, influenza B, and RSV as previously described [89]. One-Step RT-PCR AgPath-ID™ (Thermo Fischer Scientific) was used to amplify the target sequences on a CFX96 real-time PCR system-Bio-Rad. The detection and quantification of viable virus in the air samples was performed by using plaque assay as previously described [89].

D. CFD Modeling

Evidence has been reported on the close relation between the spread of aerosolized infectious agents with the air flow pattern and the ventilation modes indoors [90,91]. CFD is one of the most promising and reliable methods which could simulate and evaluate indoor environments. While the Lagrangian approach treats particles as a discrete phase and calculates and tracks the trajectory of each particle, the Eulerian method treats particles as a continuum and calculates concentrations in a control volume. The latter is widely used and known to be computationally reliable in predicting particle concentrations under steady-state conditions especially for fine particle sizes such as $PM_{2.5}$ [92–95]. In this research work, the Eulerian method was adopted to simulate air flow patterns and $PM_{2.5}$ (i.e. particles that act as carriers of microbes or bioaerosols including viruses and bacteria) concentrations in hospital wards. Measurements collected at the supply and exit locations of different sampling zones were used to define boundary conditions (BC) in the model based on the following assumptions:

- Sampled working areas of hospitals were considered with good air tightness, i.e. no air leakage effect.
- Indoor measured thermal comfort variables such as temperature and relative humidity were used as input parameters.
- Heat transfer was assumed as constant heat flux boundary condition.
- Interior wall and floor dimensions of sampled areas were considered as adiabatic boundary condition without temperature difference.

- Laminar and turbulent flows can be characterized and quantified using Reynolds Number (*Re*). If *Re* < 2000 – laminar flow, and *Re* > 4000, it is considered turbulent flow. When 2000 < *Re* < 4000, a transition region or critical region where the flow can be either laminar or turbulent is observed. In this case the indoor airflow in hospitals was considered a steady turbulent flow.
- Air inlet and outlet (air conditioning air supply inlet and exhaust outlet) were represented as grilles and dimensions of the vents were recorded and set as input parameters in the model.
- The $PM_{2.5}$ concentrations were converted from $\mu g/m^3$ to kg/kg for model simplification using the density of air (ρ = 1.225 kg/m^3).
- The RNG k - ε model was used to solve the species transport model, k - ε turbulence model, and the mass and momentum conservation equations (see Appendix F). A numerical grid was generated for the physical model to solve these conservations and scalar equations and to accelerate iterative convergence.

The advantage of using CFD modeling is to predict concentration profiles and simulate spatial distributions when difficult-to-measure concentrations are experienced and can be used further to assess the effect of ventilation modes on IAQ and air pollution levels inside hospitals. In this research work $PM_{2.5}$ concentrations and air flow patterns potentially carrying bioaerosols were simulated along the corridor and patient room of HOSP-A respectively, with an aim to examine the effect of different ventilation systems with different scenarios of air flow (Q) and air velocities (v) on IAQ and concentration levels of $PM_{2.5}$ and to define transmission routes and dispersal patterns of

viruses.

1. Hospital Corridor

The indoor airflow field and the spatial distribution of $PM_{2.5}$ concentrations were simulated along the corridor of one hospital (HOSP-A) using ANSYS Fluent 15.0, computational fluid dynamics (CFD) model. Measurements were collected at the supply and exit locations to define boundary conditions (BC) in the numerical model. The corridor has several air supplies (S) in its ceiling and an outlet exhaust (O) located at the end of the domain (Figure 4). The supplied airflow to the corridor is discharged through eight square ceiling diffusers each 0.22 × 0.22m, while the outlet exhaust dimensions is 0.61 × 0.61m. The corridor dimensions are 32.3m in length (*l*), 2.16m in width (*w*), and 2.40m in height (*h*).

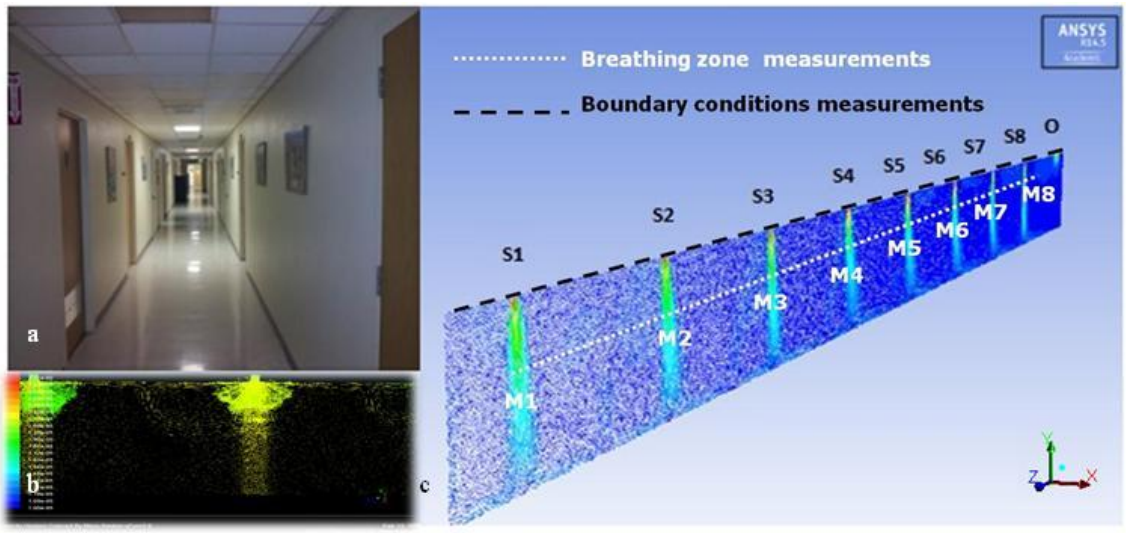

Figure 4. **a**) Hospital corridor, **b**) Diffuser model and **c**) ANSYS Fluent CFD model

A multiblock grid was generated to represent the physical domain and to accelerate iterative convergence. The corridor was assumed to have high air tightness and no air leakage [96]. The air supply flow (Q) and velocity (v) were simulated as mass flow carrying $PM_{2.5}$ and was measured by a TSI ACCUBALANCE® Air Capture Hood Model 8380 flow meter. The flow field belonged to Reynolds number (RNG) turbulence 3-D steady flow problem. The RNG k - ε model was used to solve the species transport model, the k - ε turbulence model, and the mass and momentum conservation equations (see Appendix F). As mentioned above, the $PM_{2.5}$ concentrations were converted from μg/m^3 to kg/kg for model simplification using the density of air (ρ = 1.225 kg/m^3).

2. Patient Room

The indoor airflow fields (Q) and air velocity (v) were simulated as mass flow carrying bioaerosols along the sampled patient room using ANSYS Fluent 15.0, computational fluid dynamics (CFD) model. The air supply flow (Q) and velocity (v) were measured by a TSI ACCUBALANCE® Air Capture Hood Model 8380 flow meter and VELOCICALC ® Air Velocity Meter Model 9545 (TSI Corporation 500 Cardigan Road Shoreview, MN 55126) and were reported as well by the hospital's maintenance and plant engineering department. Measurements of air flow (Q) were collected at the supply and exit locations to define boundary conditions (BC) in the numerical model. The supplied airflow to the patient's room is discharged through a fresh air inlet with an air supply of 50 cubic feet per minute (cfm) and a fan coil unit (FCU) that is installed in the ceiling above the main door and can discharge an air flow of 200 and 300 cfm

depending on the flow (Q) load operation and central conditioning system (Figure 5). Two common scenarios were simulated with 300 and 200 cfm corresponding to high and low air flow, respectively. Similarly, the flow field belonged to Reynolds number (RNG) turbulence 3-D steady flow where the RNG k-ε model was used to solve the k-ε turbulence model as well as the mass and momentum conservation equations (see Appendix F). The outlet exhaust is 0.25×0.22m and is located in the ceiling of the rest room, while the FCU inlet unit is 0.65×0.10m with a return grill unit of 0.45×0.45m and a fresh air intake inlet of 0.15×0.10m, all located in the ceiling above the main entrance of the patient room. The latter is 5.85 m in length (l), 3.60 m in width (w), and 2.80 m in height (h) (Figure 5). For a better CFD analysis of the modeling results, the patient room was divided into 4 quadrants (NW: Northwest includes the door of the patient room, NE: Northeast is the area facing the patient bed including the restroom, SE: Southeast includes the right-hand side of the patient and his/her bed, and SW: Southwest includes the left-hand side of the patient). Air samples were collected from

the SE and NW quadrants of the patient room as shown in Figure 5.

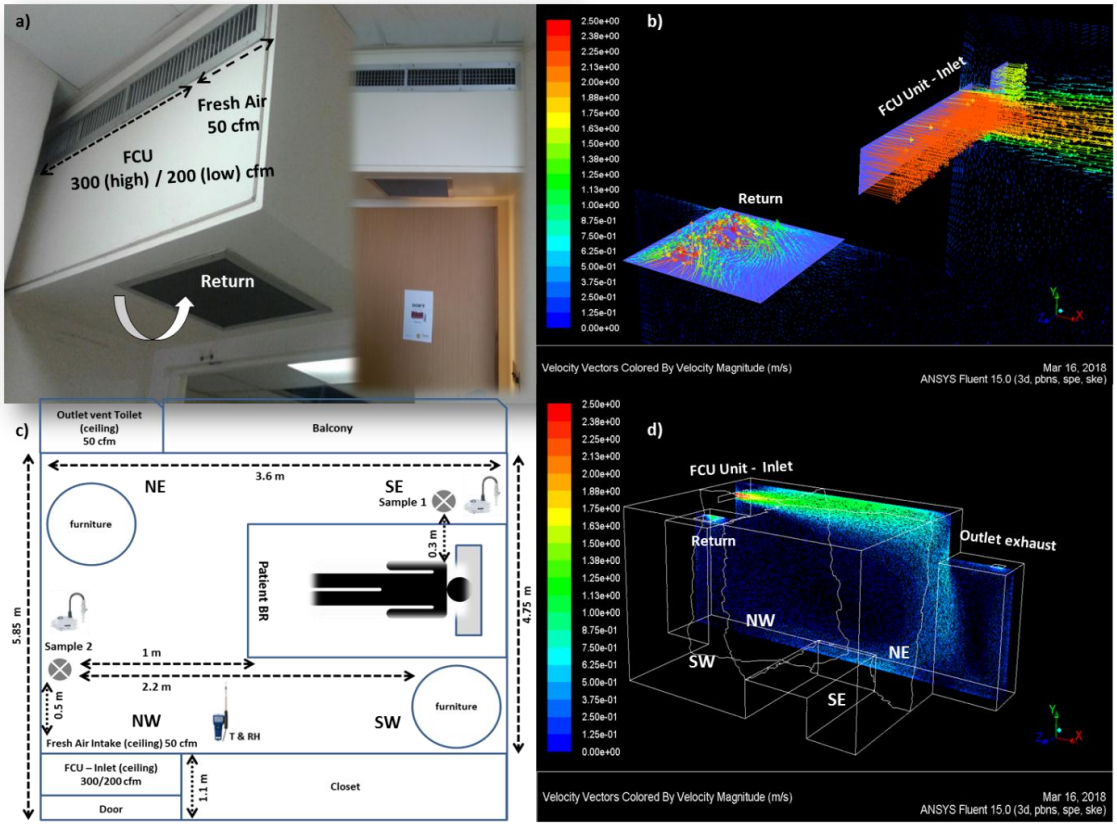

Figure 5. Field monitoring setup and CFD model of patient room **a**) FCU: Fan coil unit includes an inlet, fresh air intake and a return unit installed in the ceiling above the main door **b**) Diffuser model of the FCU unit **c**) Dimensions and sampling locations of patient room (Coriolis µ Biological Air Sampler was placed at 1.5m above ground i.e. breathing zone level) and **d**) CFD model revealing four quadrants inside patient room.

E. Air Quality Indices

Three commonly used indices (see Appendix G) were tested using five air quality indicators (PM_{10}, $PM_{2.5}$, CO, CO_2, and TVOC) to assess their applicability in transforming the data to a simple scale for evaluating IAQ in hospitals.

1. The air quality index (AQI) reflects how clean or unhealthy the air is and its associated health effects [97]. It is defined on six levels (see Appendix H) with 100 defined to protect public health and higher values reflecting a greater level of air pollution and health concerns [97].

2. The comprehensive air quality index (CAI), initially designed for the outdoors and further developed into the comprehensive indoor air quality index (CIAI) for confined environments such as subway stations. It can be applied to monitor the total amount of indoor air pollutants emitted inside buildings [81] and is classified into six groups (see Appendix I).

3. The maximum cumulative ratio (MCR) has been used to assess health impacts of exposure to individual or a mixture of air pollutants [80,98–101]. The MCR ratio is bounded by 1 and *n*, which is the number of contaminants in the mixture. It included PM_{10}, $PM_{2.5}$, CO, CO_2 and TVOC in this study. If MCR ratios are close to 1, this means that at least one contaminant is responsible for nearly all toxicity. Furthermore, exposures to a mixture of *n* substances of equal toxicities would result in an MCR ratio of *n* [80,99,100,102]. Appendix J provides a description on how the MCR can be used to classify the mixture exposures into four groups (Group I, II, IIIA and IIIB) according to the European Chemical Industry Council (*Conseil Européen des Fédérations de l'Industrie Chimique - Mixtures Industry Ad-Hoc team* – CEFIC-MIAT) decision tree with each group requiring a different risk management strategy [80,98–101].

In this research study, we also developed a new discomfort index (DI) and a robust indoor air quality index (RIAQI) with consideration to both thermal comfort and indoor air. Such indices offer a risk assessment tool that hospital administration and public health practitioners can make use of to ensure safe conditions in critical indoor environments. We have used a discomfort index (DI) as a function of temperature (T) and relative humidity (RH), to assess the thermal comfort within working areas [79]. A comfort zone was defined for each variable by setting a lower and an upper comfort bound. Within the comfort zone, the corresponding comfort value (S_{CV}) was set to 100. Beyond this zone the S_{CV} value decreased linearly at a rate defined by the upper and lower limits on comfort (CV_{ucl} and CV_{lcl}) as well as the upper and lower bounds defined for comfort (CV_{ucb} and CV_{lcb}). Beyond CV_{lcb} and CV_{ucb}, S_{CV} drops to zero. Accordingly, S_{CV} is defined by a trapezoidal function (Equations 1 and 2) as depicted in Figure 6. The DI is defined as the arithmetic mean of the two S_{CV} (Equation 3).

$$S_{CV} = 100 \frac{|CV_{i,mea} - CV_{i,lcb}|}{CV_{i,lcl} - CV_{i,lcb}} \quad \text{if } CV_{i,mea} < CV_{lcl} \tag{1}$$

$$S_{CV} = 100 \frac{|CV_{i,mea} - CV_{i,ucb}|}{CV_{i,ucb} - CV_{i,ucl}} \quad \text{if } CV_{i,mea} > CV_{ucl} \tag{2}$$

$$DI = \frac{1}{L} \sum_{i=1}^{L} S_{CVi} \tag{3}$$

Where, S_{CV} is the score value of the comfort variable (CV = T and RH) and L is the number of comfort variables considered (L = 2 in this case). The CV_{mea} is the measured mean comfort variable value, CV_{lcb} is the lower comfort bound (defined as 16

^{o}C for T and 25% for RH) and CV_{ucb} is the upper comfort bound (defined at 28 ^{o}C for T and 65% for RH). CV_{lcl} is the lower comfort level (19 ^{o}C for T and 35% for RH), CV_{ucl} is the upper comfort level (25 ^{o}C for T and 55% for RH) [59,61,79,103]. For RH, S_{CV} is set to 100 when 35 < CV_{mea} < 55. Similarly for T, when 19 < CV_{mea} < 25, the S_{CV} is set to 100 (Figure 6).

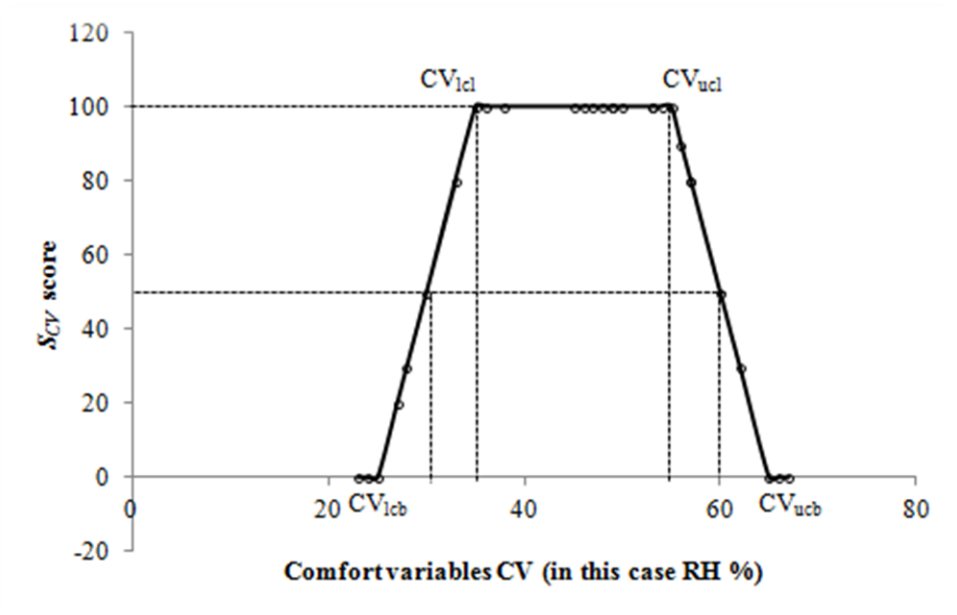

Figure 6. The score value (S_{CV}) plot vs comfort variables (in this case % RH) of DI function

The RIAQI was defined as the average of the quality indices / scores (S_i) for PM_{10}, $PM_{2.5}$, CO, CO_2, and TVOC. The S_i was defined by sigmoid curves as expressed in Equation 4 [104]. These functions penalize exceedances away from a guideline value.

$$S_i = \frac{100}{1 + ae^{-C_i b}} \tag{4}$$

$$RIAQI = \frac{\sum w_i \sum S_i}{n} \tag{5}$$

Where, w_i is the relative weight of each pollutant (in this study, all pollutants were assumed to have equal weights and $\sum w_i = 1$), S_i is the score value of each air pollutant (PM_{10}, $PM_{2.5}$, CO, CO_2, and TVOC), C_i is the average concentration measured for PM_{10}, $PM_{2.5}$, CO, CO_2, and TVOC, and s is the air pollution standard guideline value for each air pollutant (see Appendix E), n is the number of air pollutants analyzed which is 5 in this study (PM_{10}, $PM_{2.5}$, CO, CO_2, and TVOC), and a and b are empirically defined parameters and were derived based on the following assumptions: 1) a score of 98 will be assigned when measured concentrations of an air pollutant just meets the standard guideline; 2) a score of 100 is achieved at zero concentrations; and 3) indoor air pollutant concentrations that exceed their standards by two folds will get a score values (S_i) of 50 (Figure 7).

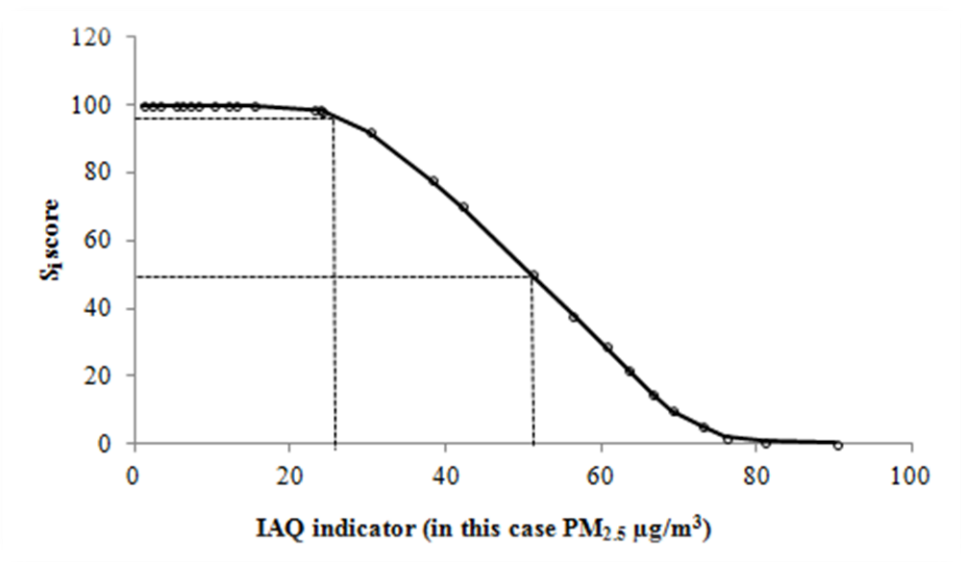

Figure 7. Score value (S_i) vs IAQ indicators (in this case $PM_{2.5}$) of RIAQI function

The RIAQI was mapped to four levels of health concerns (Table 4), with higher numerical values representing better quality. The RIAQI index proposed and developed in this research study is not aimed to replace the current indices, but can serve as an alternative approach for IAQ assessment in sensitive environments such as hospitals.

Table 4. Summary of the health concern levels for RIAQI

Classification	Range [a]	Description
Good	90 - 100	Air quality is considered satisfactory and acceptable.
Moderate	76 - 89	Air quality is acceptable; however, for some pollutants there may be a moderate health concern that may affect sensitive populations such as children and the elderly. This could be due to moderate / problematic air quality levels.
Unhealthy for Sensitive Groups	41 - 75	Members of sensitive groups such as children, elderly and people suffering from respiratory and heart diseases may experience health effects. This could be due to unhealthy and poor air quality levels.
Unhealthy / Hazardous	0 - 40	Health warnings of emergency conditions. The entire population is more likely to be affected. This could be due to poor IAQ and discomfort experienced due to unhealthy and hazardous air quality levels.

[a] The range of health concern was randomly selected based on relative scores of air contaminants.

F. Statistical Analysis

We computed the mean and standard deviation of the hourly concentrations during the warm and cold seasons and examined variations in the concentrations by one-way analysis of variance (one-way ANOVA) to assess the spatial differences among sampling sites at each hospital. We also conducted paired *t*-tests to assess statistically significant seasonal differences using a 95% confidence level (p – value = 0.05) at each sampling site. In addition, we computed the Pearson product-moment correlation coefficient (PCC) (r) to test for the linear association between the indoor and outdoor levels. As for bioaerosol samples collected inside patient rooms, contingency

tables were used to assess whether age, disease onset, measuring distance, and the occurrence of coughing/sneezing during sampling had a statistically significant effect on the percentage of positive Influenza A detected during sampling. As such, the Pearson Chi-squared (X_i^2) values with 95% confidence intervals (CI) (*p-value* < 0.05) were adopted to test for the independence of the positive Influenza-A cases from the defined categorical variables. For continuous variables, means and SDs were calculated, and statistical significance was assessed using *t*-tests and Wilcoxon rank sum tests (Mann-Whitney), with test-statistics associated with *p*-values < 0.05 considered to represent a significant effect of the predictor variable on the percentage of positive Influenza A detected during sampling. The statistical analyses and coding of air quality indices were performed using the software package R (version 3.4.3).

Finally, we evaluated exposure implications of the pollutants of concern and suggested a general framework for IAQ management in hospitals with the aim to involve and inform various stakeholders regarding the need to ensure safe and healthy IAQ in hospitals considering institutional and regulatory characteristics.

G. Ethical Approval

This study was approved by the Institutional Review Board (IRB) at the American University of Beirut (AUB) and the hospital's administration. All patients provided written informed consents prior to sample collection. Most importantly, the field sampling procedures performed did not replace or obstruct routine medical care procedures and institutional protocols of the hospital.

CHAPTER III

SEASONAL VARIABILITY AND INDOOR-OUTDOOR CORRELATIONS

A. Introduction

Air pollution in urban areas is associated with a range of health effects, both respiratory and cardiovascular [74,105,106], along with increased cancer risk [7,13,107]. The global burden of disease classifies air pollution as the fourth leading cause of mortality globally; this is mainly attributed to the joint effects of household and ambient air pollution [108,109]. Exposure may occur from multiple indoor and outdoor sources and is a characteristic of both human activities and the natural environment [11]. Pollution levels in ambient air can affect indoor air quality (IAQ), where most individuals spend the majority of their time [8,73,106,108,109]. Worldwide, concerns regarding poor IAQ have continuously increased as pollutants from various sources are identified indoors at concentrations exceeding health thresholds [74]. In addition, while indoor and outdoor levels are often correlated, the strength of their correlation varies between sites given that pollutant sources and levels, dispersion mechanisms, and personal exposures can vary among these environments [5]. In non-smoking environments, indoor concentrations have shown strong correlations with outdoor concentrations [35,37,75]. For some air pollutants such as CO, SO_2, NO_x, and PM_{10}, their outdoor concentrations tend to be higher than their indoor levels given that their

sources are primarily industrial, construction-related, or traffic-induced [7,19,74,110,111]. However, other air pollutants, such as VOCs, CO_2, and $PM_{2.5}$, tend to have higher indoor concentrations as compared to their ambient levels, given that these pollutants are typically associated with indoor activities [7,11,19,35,82,83,110,111]. The levels of air contaminants to which indoor occupants are exposed varies as a function of the type and source of the contaminant, proximity to a source, emission rate, design and age of the building, ventilation system, and enforcement of an IAQ monitoring plan [7,9,35,49,86,110–116]. While several studies have characterized and assessed IAQ in residential buildings, schools, and commercial and public buildings [74,76–81,110,112–116], limited studies have targeted such assessments in critical environments such as hospital buildings.

Hospitals represent a uniquely complex environment that differs from other commercial or residential buildings, given that the indoor occupants are at a higher risk of health symptoms such as eye irritation, headaches, coughs, colds, dizziness, asthma, and respiratory and cardiovascular diseases [49,86,117]. Many of these health concerns are reportedly associated with PM pollution, particularly $PM_{2.5}$ that penetrates deep into the bronchi and alveoli regions of the lungs [5,13,35,74,105,106,111]. Patients, medical staff, and visitors are also prone to nosocomial infections and occupational diseases [38,117]. Hospitals should be rated as high-performance buildings in terms of environmental and air quality to enhance staff efficiency and maintain patients' healing process [38,85,117,118]. Moreover, a hospital's primary function is patient care and therefore, the need to pay particular attention to air quality inside hospitals cannot be

compromised. Advanced medical treatments have helped to manage organ failure and cancer in patients; however, such treatments make the patients susceptible to common environmental microbes, including chemical and biological contaminants [38,85,117,118]. Furthermore, the hospital environment is replete with known hazards such as radiation, chemicals, and infectious agents that must be controlled. Some of these agents can become airborne, adversely affecting the IAQ of hospitals [38,85,117–119]. Environmental microbes can contaminate the patient care environment and complicate recovery if patients develop infections from common infectious agents. Patients can be put at risk by these airborne chemicals and pathogens while being treated. Therefore, consistent ventilation performance is necessary to ensure minimal exposure to infectious airborne microbes [38,85,117,118]. These airborne hazards should be recognized in critical settings of hospitals and their management involves clinical administration, engineering controls, and personal protective measures to ensure occupant safety. Ensuring clean indoor air within hospitals is thus imperative and requires a better understanding of how the ventilation systems, indoor occupants, type of medical activities, building materials, and spatial and seasonal variations affect indoor air pollution levels [9,19,35,38,117].

Previous IAQ studies in hospitals [9,19,82,83,85,112,118,120–122] have largely targeted few indicators concurrently and many have fallen short of exploring seasonal variations and indoor–outdoor correlations. Nardini et al. [82] measured $PM_{2.5}$ in two Italian hospitals in which medical offices, hallways, and waiting rooms were monitored. Erdogan et al. [19] determined the total volatile organic compound (TVOC),

CO, and CO_2 concentrations in a hospital building in Istanbul. Similarly, Jung et al. [35] examined the levels of several air quality indicators in several working hospital areas in Taiwan, but without accounting for seasonal variability. More recently, Baurès et al. [122] targeted the assessment of volatile and semi-volatile organic compounds as well as particulate concentrations in two French hospitals through a winter and summer campaign; however, they did not examine the indoor–outdoor (IO) correlations.

In this chapter, we attempted to quantify the seasonal variations in the IAQ of hospitals by monitoring temporal changes in various IAQ indicators (CO, CO_2, SO_2, NO/NO_x, $PM_{2.5}$, PM_{10}, and TVOC) across two seasons (warm vs. cold) while also tracking the association of the measured IAQ with the ambient levels. To our knowledge, no study has thus far adopted such a comprehensive and systematic approach for examining IAQ and establishing indoor – outdoor (I/O) correlations in hospital environments. Our data and results will help in adopting IAQ guidelines and designing management frameworks for indoor hospital environments.

B. Seasonal Trends

The indoor and outdoor concentrations of the various air pollutants recorded during the warm and cold seasons are summarized in Tables 5 and 6, respectively. We observed that differences between the mean indoor concentrations of CO, CO_2, $PM_{2.5}$, PM_{10} and TVOC across the sampling sites and over the two seasons were statistically significant ($p < 0.05$). Indoor CO levels during the cold season (Table 5) were lower than those recorded during the warm season across all sampling sites with all levels

below the U.S. Environmental Protection Agency National Ambient Air Quality Standards (USEPA NAAQS) i.e. 9 ppm for 8 hours and 35 ppm for 1 hour. In the multi-story hospitals (HOSP-A and HOSP-B), indoor CO levels (1.82 ± 0.09 ppm and 2.2 ± 0.35 ppm, respectively) at higher elevations (5th, 8th, and 10th floor) were slightly lower than those recorded at lower floors (i.e. on the ground at 3.49 ± 0.26 ppm and 1st floor at 2.76 ± 0.33 ppm). This is expected as CO levels on lower floors will primarily be a result of outdoor vehicle-induced emissions [14,19,123–125]. The clinic waiting area, lobby, staff office, corridor, pediatrics' room, and BMT waiting area were mechanically ventilated and at a greater distance from the main sources of pollution. When compared to concurrently measured outdoor CO concentrations (Table 6), most indoor levels were consistently lower (Table 5). In both urban hospitals (HOSP-A and HOSP-B), we observed that outdoor CO levels were below the USEPA NAAQS despite the proximity of the hospitals to CO sources such as vehicular emissions, tobacco smoke, and emissions from diesel generators that are commonly used throughout the city during periodic electrical outages.

With respect to CO_2, we measured indoor concentrations below the 1000 ppm American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) limit [59,69,103,126] (Table 5) and in general the measured levels were higher than outdoor levels irrespective of season (Table 6). This is attributed to human metabolism and breathing [14,111,120]. Similar to CO_2, measured TVOC concentrations were below the threshold level of 3 ppm [103,126,127], with higher

levels recorded indoors ($p < 0.05$) during both seasons (Table 5). TVOC levels indoors were associated with the use of cleaning chemicals and air fresheners [18,19,128].

In this study, concentrations of gaseous pollutants such as SO_2 and NO/NO_x were below detection limit in most samped areas and were also below their standards regardless of seasonal variation. However, the non-alarming low levels of SO_2, NO and NO_x were recorded only outdoors at floor 1 entrance of HOSP-B during the warm (SO_2: 0.11 ± 0.03 ppm; NO_x: 0.12 ± 0.03 ppm) and cold (SO_2: 0.10 ± 0.02 ppm; NO_x: 0.13 ± 0.02 ppm) seasons. Although not alarming, however the levels obtained for SO_2 and NO_x gaseous pollutants in this study might be attributed to the proximity of outdoor sampled locations (i.e. Floor 1 entrance) to some industrial activities, combustion sources such as vehicular emissions, and diesel power generators that operate in urban cities like Beirut for at least 3 to 6 hours / day when national power supply is interrupted. Similarly, other studies have also reported low levels of NO_x and SO_2 in critical environements such as medical facilities and schools [129–131]. Helmis et al. [131] measured air quality indicators including NO_x and SO_2 over a period of three months in a selected dentistry clinic where both NO_x and SO_2 concentrations remained at low levels and within their recommended limits for the whole experimental period [131]. Ayodele et al. [129] reported low levels of NO (0.03 – 0.21 ppm) and NO_2 (0 – 0.06 ppm) at a medical facility, while no concentration of SO_2 were detected at most sampled locations. Moreover, a study conducted by Yang et al. [130]reported a mean level of 0.017 ± 0.004 ppm of NO_2 inside classrooms and laboratories of urban elementary schools in Seoul, South Korea.

We observed that indoor $PM_{2.5}$ and PM_{10} concentrations were statistically different across sampling sites in each hospital ($p < 0.05$). Additionally, both concentrations showed strong seasonal variations ($p < 0.05$). During the warm season, indoor working areas including the clinic, clinic waiting area, lobby, reception, staff office, corridor, meeting room, and ophthalmology patient room had $PM_{2.5}$ levels that exceeded the World Health Organization (WHO) standards (25 µg/m^3) by 10.4 to 245 % (Table 5). High levels of PM_{10} were equally recorded at the clinic, reception and corridor, with concentrations exceeding the WHO standards (50 µg/m^3) by 88, 76, and 11%, respectively. Such high levels of $PM_{2.5}$ and PM_{10} during the warm season may be attributed to the high occupancy rate and human activities in these locations in addition to the occurrence of regional dust storms. During dust storm episodes, the average mass concentrations of $PM_{2.5}$ and PM_{10} increased by 68% and 56% at the clinic, 40% and 27% at the reception, and 112% and 56% at the corridor as compared to concentrations measured on non-dusty days during the cold season (Table 5). This is consistent with other studies in the Mediterranean region and China, where a significant increase in indoor PM_{10} and $PM_{2.5}$ concentrations is typically reported during dust storm events [132–138]. Although the levels of $PM_{2.5}$ and PM_{10} decreased during the cold season in the clinic ($PM_{2.5}$: 51.2 ± 8.20 µg/m^3; PM_{10}: 60.5 ± 11.5 µg/m^3) and the reception area ($PM_{2.5}$: 54.2 ± 14.3 µg/m^3; PM_{10}: 69.1 ± 7.50 µg/m^3), the measured concentrations still exceeded WHO standards. Other critical indoor environments such as the PPR, ER, workshop, and BMT waiting area showed lower concentrations of $PM_{2.5}$ and PM_{10} (Table 5) that were below the WHO standards [111,126,127]. Note that rainfall and W-

NW wind trajectories often accompanied the lowest PM_{10} and $PM_{2.5}$ levels during the cold season. These observations are consistent with other studies indicating that wet deposition reduces PM_{10} and $PM_{2.5}$ levels [139].

In addition, outdoor $PM_{2.5}$ and PM_{10} levels exceeded the WHO standards at most sampling sites at the two urban hospitals (HOSP-A and HOSP-B) (Table 6). During the warm season, $PM_{2.5}$ outdoor concentrations ranged from 39.9 ± 1.6 (11th Mechanical) to 93.9 ± 24.4 µg/m^3 (1st floor entrance), while PM_{10} varied from 44.6 ± 2.6 (11th Mechanical) to 104.5 ± 13.5 µg/m^3 (1st floor entrance). Lower levels of $PM_{2.5}$ and PM_{10} were recorded during the cold season (Table 6). The high outdoor $PM_{2.5}$ and PM_{10} levels can be attributed to the proximity of the sampling sites to on-road vehicular emissions and construction activities around urban HOSP-A and HOSP-B and dust storms during the warm season.

Table 5. Concentrations of indoor air quality indicators

					Mean ± Std. (warm season)					Mean ± Std. (cold season)				
ID	Location	HOSP	Ventilation Mode	Floor	CO ppm	CO_2 ppm	$PM_{2.5}$ µg/m^3	PM_{10} µg/m^3	TVOC ppm	CO ppm	CO_2 ppm	$PM_{2.5}$ µg/m^3	PM_{10} µg/m^3	TVOC ppm
1	Clinic	B	Natural	1st	3.49±0.26	731±33.5	86.2±10.5[a]	94.2±12.5[a]	BDL [b]	2.59±0.15	713±31.7	51.2±8.2	60.5±11.5	BDL [b]
2	Clinic waiting area	A	Mechanical	5th	1.82±0.09	453±21.8	40.9±8.6	48.9±9.4	0.17±0.02	0.90±0.14	431±6.90	27.2±6.8	35.5±9.7	BDL [b]
3	Lobby	A	Mechanical	10th	1.83±0.07	608±25.9	27.6±3.4	35.6±3.6	BDL [b]	1.63±0.02	624±43.7	25.6±1.2[c]	33.6±3.3[c]	BDL [b]
4	Reception	B	Natural	Ground	2.76±0.33	592±29.3	76.1±9.7 [a]	88.1±8.5 [a]	BDL [b]	2.71±0.20	568±16.7	54.2±14.3	69.1±7.5	BDL [b]
5	Staff office	A	Mechanical	7th	2.11±0.09	535±21.2	33.9±8.0	40.3±6.0	BDL [b]	1.75±0.02	536±8.80	21.5±1.1	33.3±6.2	BDL [b]
6	Corridor	A	Mechanical	8th	1.95±0.25	502±26.5	41.9±6.0[a]	55.7±5.2[a]	0.33±0.05	1.57±0.12	472±4.80	19.8±6.6	35.7±5.5	0.13±0.03
7	Pediatrics room	A	Mechanical	6th	1.41±0.06	418±22.2	13.0±0.8	25.2±2.8	BDL [b]	1.60±0.03	446±17.6	11.4±2.7[c]	27.2±2.9[c]	BDL [b]
8	Emergency room	C	Natural	Ground	1.58±0.54	392±16.9	24.8±4.4	30.8±5.4	BDL [b]	1.74±0.18	370±5.20	20.4±1.1[c]	28.8±4.5[c]	BDL [b]
9	Workshop	A	Mechanical	Basement	1.60±0.16	470±42.6	23.1±7.3	38.2±4.3	BDL [b]	2.11±0.09	457±55.7	19.8±3.8	37.5±3.3	BDL [b]
10	Meeting room	A	Mechanical	8th	2.20±0.35	588±28.1	36.8±4.0	42.8±4.0	0.18±0.03	1.26±0.02	553±8.40	18.7±9.7	32.2±4.1	0.20±0.02
11	BMT waiting area	A	Mechanical	8th	1.81±0.15	560±17.2	18.5±7.5	24.6±3.5	0.19±0.08	1.34±0.03	605±8.20	9.20±0.8[c]	26.6±3.6[c]	0.16±0.09
12	Ophthalmology PR	A	Mechanical	7th	2.12±0.07	481±9.50	29.0±1.9	44.2±2.9	0.23±0.07	2.08±0.04	493±8.00	19.1±7.7	36.2±3.5	0.14±0.01

[a] Dust storms were recorded during April 2013, April 2015, October 2015, and April 2016 and contributed to high concentrations of PM_{10} and $PM_{2.5}$ during the warm season.
[b] BDL: below the detection limit (5 ppb).
[c] Rainy days were recorded during December 2014, February 2015, November and December 2015, February 2016, and December 2016 of the cold season.

Table 6. Concentrations of outdoor air quality indicators

			Mean ± Std. (warm season)					Mean ± Std. (cold season)				
ID	Location [a]	HOSP	CO ppm	CO_2 ppm	$PM_{2.5}$ µg/m^3	PM_{10} µg/m^3	TVOC ppm	CO ppm	CO_2 ppm	$PM_{2.5}$ µg/m^3	PM_{10} µg/m^3	TVOC ppm
1	Floor 1 entrance	B	3.88±0.64	428±54.9	93.9±24.4[b]	104.5±13.5[b]	BDL [c]	2.71±0.57	467±76.0	52.9±26.1	66.8±14.5	BDL [c]
2	Floor 8 mechanical	A	1.06±0.16	391±7.7	70.6±6.2	92.3±7.4	0.07±0.02	1.19±0.16	397±5.00	42.3±6.90	54.3±4.7	BDL [c]
3	Floor 11 mechanical	A	2.46±0.25	521±15.3	39.9±1.6	44.6±2.6	BDL [c]	1.94±0.09	507±15.2	39.7±1.90[d]	42.1±2.6 [d]	BDL [c]
4	Floor 1 entrance	B	3.96±0.71	523±28.7	81.8±14.8[b]	96.2±12.5[b]	BDL [c]	2.27±0.20	490±18.0	65.3±18.5	76.1±11.5	BDL [c]
5	Floor 8 mechanical	A	1.34±0.15	431±5.5	46.3±6.2	52.3±3.0	BDL [c]	1.48±0.14	435±5.30	45.1±4.50	50.2±3.5	BDL [c]
6	Floor 8 mechanical	A	1.40±0.18	387±8.4	76.3±8.9[b]	83.7±9.2[b]	0.12±0.07	1.67±0.17	397±5.00	31.4±8.30	53.7±8.2	BDL [c]
7	Floor 11 mechanical	A	2.25±0.22	414±5.8	67.4±6.5	75.2±5.8	BDL [c]	1.66±0.13	418±8.70	38.1±1.20[d]	45.6±5.2[d]	BDL [c]
8	ER main entrance	C	1.90±0.28	292±2.5	31.5±6.7	36.8±3.4	BDL [c]	1.13±0.27	294±2.60	22.0±3.40[d]	34.8±4.4[d]	BDL [c]
9	Floor 8 mechanical	A	2.33±0.09	373±34.2	50.7±4.3	64.3±6.3	BDL [c]	2.05±0.10	350±10.8	30.9±8.10	48.2±6.9	BDL [c]
10	Floor 11 mechanical	A	1.41±0.54	397±7.5	40.2±1.2	52.3±3.5	0.05±0.02	1.20±0.10	433±2.90	21.3±2.10	42.8±3.1	0.06±0.02
11	Floor 11 mechanical	A	1.06±0.20	391±5.8	45.1±8.9	54.7±6.5	0.02±0.01	1.10±0.08	444±8.30	30.0±9.60[d]	41.7±4.5[d]	0.07±0.02
12	Floor 8 mechanical	A	1.34±0.15	357±11.4	46.1±9.7	63.2±3.9	0.03±0.02	1.27±0.10	373±10.9	31.6±6.00	53.5±5.9	0.09±0.03

[a] Outdoor and indoor measurements were concurrently conducted and outdoor sampling locations were selected to represent fresh air intakes into the indoor working areas.
[b] Dust storms were recorded during April 2013, April 2015, October 2015, and April 2016 and contributed to high concentrations of PM_{10} and $PM_{2.5}$ during the warm season.
[c] BDL: Below detection limit (5 ppb).
[d] Rainy days were recorded during December 2014, February 2015, November and December 2015, February 2016 ,and December 2016 of the cold season.

C. $PM_{2.5}/PM_{10}$ Ratios

Table 7 presents indoor and outdoor $PM_{2.5}/PM_{10}$ ratios during the warm and cold seasons. The $PM_{2.5}/PM_{10}$ ratio for indoor working areas varied between 0.52 (pediatrics room) and 0.92 (clinic) during the warm season, and from 0.35 (BMT waiting area) to 0.85 (clinic) during the cold season. Most working areas showed $PM_{2.5}/PM_{10}$ ratios > 0.5 during both seasons highlighting the predominance of $PM_{2.5}$. Likewise, and consistent with other studies [74,140,141], high $PM_{2.5}/PM_{10}$ ratios were recorded outdoors, where $PM_{2.5}$ constituted approximately 85–90%, and 79–86% of the total PM at the floor entrance during the warm and cold seasons, respectively. The results also showed that during dust storm episodes, the $PM_{2.5}/PM_{10}$ ratio increased from 0.79 to 0.90 at the 1st floor entrance, 0.58 to 0.91 at the 8th floor mechanical room intake, 0.85 to 0.92 at the clinic, and 0.55 to 0.75 at the corridor, suggesting a greater abundance of finer $PM_{2.5}$ as compared to coarse particles $PM_{10-2.5}$ during these storms. Unlike coarse particles that remain in the atmosphere for shorter periods (hours), fine particles ($PM_{2.5}$) can remain in the atmosphere for days or weeks [1,4,5,142]. Because of its longer-range transport over urban areas, $PM_{2.5}$ can react through adsorption and deposition with other anthropogenic and secondary pollutants, thus affecting human health and causing increased risk of respiratory and cardiovascular illness [6,133,134,143,144]. Note that the high $PM_{2.5}/PM_{10}$ ratios coincided with S-SE wind trajectories, which are linked to the movement of air masses from the deserts of the Arabian Peninsula.

Table 7. Indoor and outdoor $PM_{2.5}/PM_{10}$ ratios during the warm and cold seasons

					Indoor $PM_{2.5}/PM_{10}$		**Outdoor $PM_{2.5}/PM_{10}$**		
ID	Working area	HOSP	Ventilation Mode	Floor	*Warm*	*Cold*	Sampling location [a]	*Warm*	*Cold*
1	Clinic	B	Natural	1st	0.92 [b]	0.85	Floor 1 entrance	0.90 [b]	0.79
2	Clinic waiting area	A	Mechanical	5th	0.84	0.77	Floor 8 mechanical	0.76	0.78
3	Lobby	A	Mechanical	10th	0.78	0.76	Floor 11 mechanical	0.89	0.94
4	Reception	B	Natural	Ground	0.86 [b]	0.78	Floor 1 entrance	0.85 [b]	0.86
5	Staff office	A	Mechanical	7th	0.84	0.65	Floor 8 mechanical	0.89	0.90
6	Corridor	A	Mechanical	8th	0.75 [b]	0.55	Floor 8 mechanical	0.91 [b]	0.58
7	Pediatrics room	A	Mechanical	6th	0.52	0.42	Floor 11 mechanical	0.90	0.84
8	Emergency room	C	Natural	Ground	0.81	0.71	ER main entrance	0.86	0.63
9	Workshop	A	Mechanical	Basement	0.60	0.53	Floor 8 mechanical	0.79	0.64
10	Meeting room	A	Mechanical	8th	0.86	0.58	Floor 11 mechanical	0.77	0.50
11	BMT waiting area	A	Mechanical	8th	0.75	0.35	Floor 11 mechanical	0.82	0.72
12	Ophthalmology PR	A	Mechanical	7th	0.66	0.53	Floor 8 mechanical	0.73	0.59

[a] Concurrent outdoor and indoor monitoring with outdoor locations at fresh air intakes into indoor working areas
[b] Dust storms were recorded during April 2013, April 2015, October 2015, and April 2016 and contributed to high concentrations of PM_{10} and $PM_{2.5}$ during the warm season.

D. Indoor – Outdoor (IO) Correlations

Indoor levels of air pollutants are affected by both indoor sources and the infiltration of outdoor pollutants [74,120,145]. To assess the impact of outdoor air and the strength of indoor sources on IAQ during the warm and cold seasons, the average seasonal I/O ratios for CO, CO_2, $PM_{2.5}$, PM_{10}, and TVOC were calculated for various working areas with higher I/O CO ratios generally recorded during the warm season (Table 8).

Table 9 shows the Pearson correlation factor (r) between the indoor and outdoor CO levels for both the cold and warm seasons. Note that the CO levels showed correlations as high as 0.61 in the clinic during the warm season, suggesting significant migration of outdoor air into indoor environments, carrying vehicle-induced CO

emissions from congested roads [74,125,146]. The CO_2 indoor levels exceeded those recorded outdoors during both seasons and resulted in CO_2 I/O ratios > 1 (Table 8), suggesting that human metabolism and respiration contributed to the higher indoor levels of CO_2 [16]. Similarly, higher TVOC levels were recorded indoors during both seasons, consistent with other studies [18,128]. Higher TVOC I/O ratios were found during the warm season in several locations, reaching 3.6, 9.5, and 7.7 at the meeting room, BMT waiting area, and ophthalmology PR, respectively (Table 8). Such levels are mainly attributed to the frequent use of floor-cleaning chemicals and air fresheners, which are known sources of TVOCs [19,128]. While the average $PM_{2.5}$ and PM_{10} levels significantly varied between working areas ($p < 0.05$), most $PM_{2.5}$ and PM_{10} I/O ratios were < 1. In general, higher PM correlations were observed during the warm season as compared to those during the cold season (Table 9). A high correlation was recorded during the warm season between indoor and outdoor $PM_{2.5}$ and PM_{10} levels (Figure 8), suggesting that indoor environments are affected by outdoor sources including construction, vehicle-induced emissions, and/or dust storms.

Table 8. Average I/O ratios of air pollutants at sampled working areas

					I/O ratio (warm season)					I/O ratio (cold season)				
ID	Working area	HOSP	Ventilation Mode	Floor	CO ppm	CO_2 ppm	$PM_{2.5}$ µg/m^3	PM_{10} µg/m^3	TVOC ppm	CO ppm	CO_2 ppm	$PM_{2.5}$ µg/m^3	PM_{10} µg/m^3	TVOC ppm
1	Clinic	B	Natural	1st	0.90	**1.71**	0.92 [a]	0.90 [a]	BDL [b]	0.96	**1.53**	0.97	0.91	BDL [b]
2	Clinic waiting area	A	Mechanical	5th	**1.72**	**1.16**	0.58	0.53	**2.43**	0.76	**1.09**	0.64	0.65	BDL [b]
3	Lobby	A	Mechanical	10th	0.74	**1.17**	0.69	0.80	BDL [b]	0.84	**1.23**	0.64 [c]	0.80 [c]	BDL [b]
4	Reception	B	Natural	Ground	0.70	**1.13**	0.93 [a]	0.92 [a]	BDL [b]	**1.19**	**1.16**	0.83	0.91	BDL [b]
5	Staff office	A	Mechanical	7th	**1.57**	**1.24**	0.73	0.77	BDL [b]	**1.18**	**1.23**	0.48	0.66	BDL [b]
6	Corridor	A	Mechanical	8th	**1.39**	**1.30**	0.55 [a]	0.67 [a]	**2.75**	0.94	**1.19**	0.63	0.66	BDL [b]
7	Pediatrics room	A	Mechanical	6th	0.63	**1.01**	0.19	0.34	BDL [b]	0.96	**1.07**	0.30 [c]	0.60 [c]	BDL [b]
8	Emergency room	C	Natural	Ground	0.83	**1.34**	0.79	0.84	BDL [b]	**1.54**	**1.26**	0.93 [c]	0.83 [c]	BDL [b]
9	Workshop	A	Mechanical	Basement	0.69	**1.26**	0.46	0.59	BDL [b]	**1.03**	**1.31**	0.64	0.78	BDL [b]
10	Meeting room	A	Mechanical	8th	**1.56**	**1.48**	0.92	0.82	**3.60**	**1.05**	**1.28**	0.88	0.75	**3.33**
11	BMT waiting area	A	Mechanical	8th	**1.71**	**1.43**	0.41	0.45	**9.50**	**1.22**	**1.36**	0.31 [c]	0.64 [c]	**2.29**
12	Ophthalmology PR	A	Mechanical	7th	**1.58**	**1.35**	0.63	0.70	**7.67**	**1.64**	**1.32**	0.60	0.68	**1.56**

[a] Dust storms were recorded during April 2013, April 2015, October 2015, and April 2016 and contributed to the high concentrations of PM_{10} and $PM_{2.5}$ during the warm season.
[b] BDL: Below detection limit (5 ppb)
[c] Rainy days were recorded during December 2014, February 2015, November and December 2015, February 2016, and December 2016 of the cold season.

Table 9. Pearson correlation (r) between the indoor and outdoor levels for PM_{10}, $PM_{2.5}$, and CO at several working areas with a seasonal variation

		Outdoor [a]		
PM_{10} Indoor	Floor	Entrance Floor 1	Mechanical Floor 8	Mechanical Floor 11
Clinic [d]	1st	(**0.86**) [b] (0.68) [c]		
Reception [d]	Ground	(**0.74**) (0.62)		
Staff office [e]	7th	---	(**0.35**) (0.21)	
Corridor [e]	8th	---	(**0.43**) (0.39)	
Ophthalmology PR [e]	7th	---	(**0.25**) (0.16)	
Meeting room [e]	8th	---	---	(**0.46**) (0.31)
$PM_{2.5}$ Indoor	Floor	Entrance Floor 1	Mechanical Floor 8	Mechanical Floor 11
Clinic	1st	(**0.92**) (0.71)		
Reception	Ground	(**0.83**) (0.67)		
Staff office	7th	---	(**0.45**) (0.24)	
Corridor	8th	---	(**0.56**) (0.42)	
Ophthalmology PR	7th	---	(**0.23**) (0.21)	
Meeting room	8th	---	---	(**0.51**) (0.43)
CO Indoor	Floor	Entrance Floor 1	Mechanical Floor 8	Mechanical Floor 11
Clinic	1st	(**0.61**) (0.37)		
Reception	Ground	(**0.49**) (0.32)		
Staff office	7th	---	(**0.34**) (0.28)	
Corridor	8th	---	(**0.29**) (0.15)	
Ophthalmology PR	7th	---	(**0.24**) (0.26)	
Meeting room	8th	---	---	(**0.56**) (0.33)

[a] Sampling simultaneously occurred at outdoor locations with indoor working areas
[b] (***r***) in bold represents warm season correlations
[c] (*r*) represent cold season correlations
[d] The clinic and reception working areas of HOSP-B had natural ventilation.
[e] The staff office, corridor, ophthalmology PR and meeting room of HOSP-A had a mechanical ventilation system.

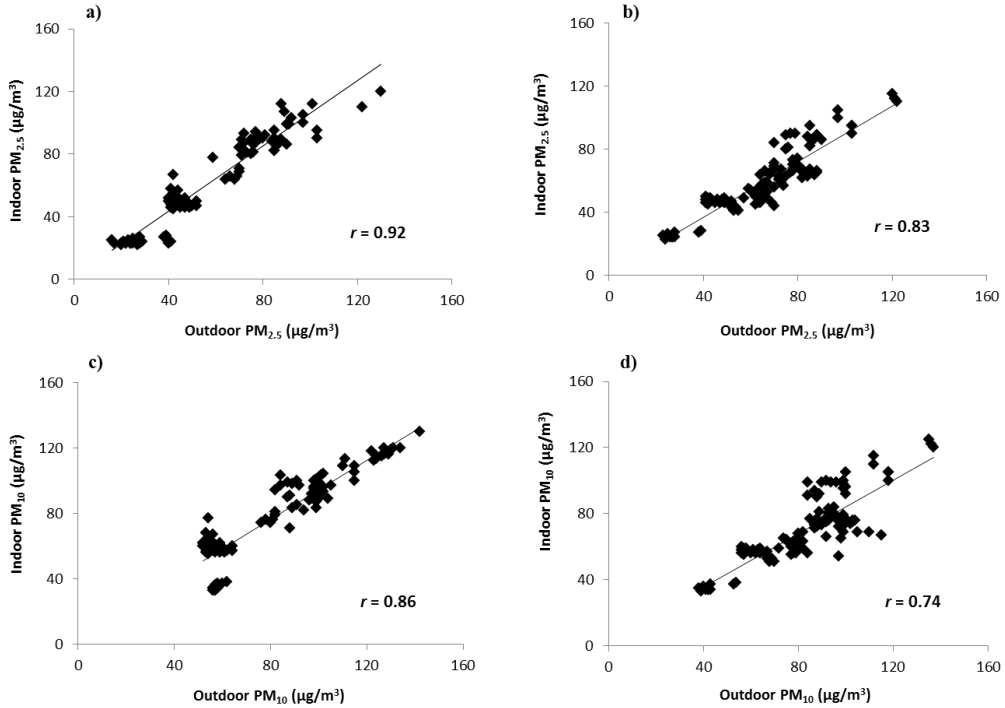

Figure 8. Indoor versus outdoor scatter plots for $PM_{2.5}$ **a)** clinic **b)** reception and PM_{10} **c)** clinic **d)** reception.

E. Implications of high PM concentrations on public health

Our results indicated relatively high PM_{10} and $PM_{2.5}$ levels within hospitals. These levels pose a concern for the health of patients, staff, and visitors. The chronic effects of exposure to such levels are of particular relevance to long-term patients and staff. In fact, various epidemiological studies have associated long-term exposure to PM_{10} and $PM_{2.5}$ with increased risk of daily mortality and morbidity [105,147–151]. Appendix K presents a summary of the short- and long-term mortality risk estimates given exposure to PM_{10} and $PM_{2.5}$.

Based on the PM measurements at the three hospitals, we estimated the toxicity potential (TP) associated with the measured concentrations. TP is expressed using Equation 6 [129,152]:

$$Toxicity\ Potential\ (TP) = \frac{C_p}{S_p} \quad (6)$$

Where, C_p is the measured concentration of the air pollutant and S_p is the standard threshold for the air pollutant (i.e. 25 and 50 µg/m^3 for $PM_{2.5}$ and PM_{10}, respectively). One limitation of this analysis is that we assumed the observed concentrations to be representative of a 24-hour exposure. Table 10 presents TP_{PM10} and $TP_{PM2.5}$ values during both the warm and cold seasons at the three hospitals illustrating various locations where TP values are greater than one, indicating a potential health concern.

Table 10. TP at sampled working areas with a seasonal variation

					TP [a]			
					Warm		***Cold***	
ID	Working area	HOSP	Ventilation Mode	Floor	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}
1	Clinic	B	Natural	1st	**3.45**	**1.88**	**2.05**	**1.21**
2	Clinic waiting area	A	Mechanical	5th	**1.64**	0.98	**1.09**	0.71
3	Lobby	A	Mechanical	10th	**1.10**	0.71	**1.02**	0.67
4	Reception	B	Natural	Ground	**3.04**	**1.76**	**2.17**	**1.38**
5	Staff office	A	Mechanical	7th	**1.36**	0.81	0.86	0.67
6	Corridor	A	Mechanical	8th	**1.68**	**1.11**	0.79	0.71
7	Pediatrics room	A	Mechanical	6th	0.52	0.50	0.46	0.54
8	Emergency room (ER)	C	Natural	Ground	0.99	0.62	0.82	0.58
9	Workshop	A	Mechanical	Basement	0.92	0.76	0.79	0.75
10	Meeting room	A	Mechanical	8th	**1.47**	0.86	0.75	0.64
11	BMT waiting area	A	Mechanical	8th	0.74	0.49	0.37	0.53
12	Ophthalmology PR	A	Mechanical	7th	**1.16**	0.88	0.76	0.72

[a] TP was calculated using Equation (6), where S_p is the WHO standard, i.e. 25 µg/m^3 and 50 µg/m^3 for $PM_{2.5}$ and PM_{10}, respectively. High TP values >1 are shown in bold.

CHAPTER IV

APPLICATION AND DEVELOPMENT OF AN INDOOR AIR QUALITY INDEX

A. Introduction

Indoor air quality (IAQ) is highly affected by outdoor emission sources particularly in congested urban areas, often associated with vehicle-induced emissions as well as construction and industrial emissions. Since people spend most of their time indoors the effect of IAQ on personal exposure and human health is invariably more pronounced than the effect of the outdoor air [7,10,37,122]. On the other hand, IAQ in certain sensitive environments such as hospitals is of critical concern for its occupants [75] with various IAQ and thermal comfort indicators commonly associated with respiratory diseases [12,86,105,142], as well as increased risk of allergies, intoxication, acute morbidity, and / or sick building syndrome (SBS) [7,10,53,153,154]. In this context, air quality indices (AQIs), which are mathematical formulations that translate measured air quality indicators into dimensionless variables, can be relied upon for better communication with the public [97,155–157]. Suitable indices include indicators that are continuously monitored and that have a clear impact on air quality. These indices should be objective, easy to use, flexible and sensitive to small changes in air quality [158,159]. In addition, proper categorization and description of risk levels corresponding to an index value are required to facilitate communication of the associated health risk [97,158–160].

Since the 1960s, several environmental quality indices (EQIs) have been developed and used for ambient air [159–161]. While the use of ambient air quality or pollution indices has become widespread [156,158–162], similar indices for indoor environments are limited. In this context, indoor risk assessment studies tend to rely on individual air quality indicators [163–166], which could underestimate the combined risk of a multi substance approach [80,98,167]. The latter remains a challenge for policy makers [80,164] with a lack of tools to assess the effects associated with potential exposure to multiple substances, particularly in critical indoor environments.

In this chapter, a comparative assessment between various indices is presented where we tested three existing indoor air quality (IAQ) indices for risk assessment exposure in hospitals using a mixture of air quality indicators (PM_{10}, $PM_{2.5}$, CO, CO_2, TVOC) with measurements collected from several hospitals over a three year period. Then a novel index was developed with consideration to thermal comfort indicators (T, RH) and air quality indicators and was tested as well and compared with existing indices to a) examine the extent to which each index can classify various hospital environments, b) analyze the consistency and repeatability of the calculated IAQ levels across indices in indoor environments of healthcare facilities, c) discuss the health implications of all indices under different pollution situations, and finally d) recommend the most robust index that can be used as a risk assessment tool towards safer conditions in critical indoor environments.

B. IAQ and Thermal Comfort Indicators

The air quality measurements were performed under real working conditions (9:00 Am – 5:00 PM) in hospitals (see Chapter II). Table 11 presents the overall average concentrations of indoor air quality indicators PM_{10}, $PM_{2.5}$ CO, CO_2, and TVOC as well as the thermal comfort variables values of T and RH measured throughout the monitoring program in various hospital zones. The data presented in Table 11 below will be used to assess the IAQ levels and health concerns of different working areas of hospitals.

Table 11. The overall average concentration of indoor pollutants and thermal comfort indicators of sampled areas

		Mean ± Std.						
Working area	Floor	PM_{10} (μg/m^3)	$PM_{2.5}$ (μg/m^3)	CO (ppm)	CO_2 (ppm)	TVOC (ppm)	Temp (°C)	RH (%)
Clinic	1st	77.4 ±12.1[a]	68.7 ± 9.6[a]	3.04 ± 0.21	722 ± 32.2	BDL[b]	21.8 ± 0.2	53 ± 1.4
Clinic WA	5th	42.2 ± 9.6	34.1 ± 7.6	1.36 ± 0.13	442 ± 14.3	0.17 ± 0.02	22.3 ± 0.4	57 ± 0.8
Lobby	10th	34.6 ± 3.5	26.6 ± 2.8	1.73 ± 0.06	616 ± 33.1	BDL	22.6 ± 0.3	54 ± 0.5
Reception	Ground	78.6 ± 7.8[a]	65.2 ± 13.8[a]	2.74 ± 0.28	580 ± 25.1	BDL	21.6 ± 0.3	58 ± 1.6
Staff office	7th	36.8 ± 6.1	27.7 ± 6.7	1.93 ± 0.06	536 ± 16.3	BDL	22.5 ± 0.2	53 ± 1.5
Corridor	8th	45.7 ± 5.4	30.9 ± 6.4	1.76 ± 0.21	487 ± 15.4	0.23 ± 0.04	21.8 ± 0.5	63 ± 1.7
Pediatrics PR	6th	26.2 ± 2.9	12.2 ± 2.0	1.51 ± 0.05	432 ± 21.3	BDL	22.3 ± 0.2	55 ± 0.9
ER	Ground	29.8 ± 4.9	22.6 ± 3.2	1.66 ± 0.41	381 ± 12.1	BDL	20.1 ± 0.1	63 ± 0.6
Workshop	Basement	37.9 ± 4.1	21.5 ± 6.5	1.86 ± 0.12	464 ± 47.3	BDL	19.5 ± 0.1	52 ± 1.5
Meeting room	8th	37.5 ± 4.0	27.8 ± 8.8	1.73 ± 0.24	571 ± 22.1	0.19 ± 0.03	22.3 ± 0.3	57 ± 1.2
BMT WA	8th	25.6 ± 3.6	13.9 ± 5.1	1.58 ± 0.08	583 ± 13.3	0.18 ± 0.07	20.6 ± 0.4	51 ± 1.5
Opthamology PR	7th	40.2 ± 3.3	24.1 ± 4.5	2.10 ± 0.05	487 ± 8.9	0.19 ± 0.06	22.5 ± 0.6	56 ± 0.8
p – Value[c]		< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	> 0.05	> 0.05

[a] Dust storms took place during sampling and contributed to the high concentrations of PM_{10} and $PM_{2.5}$.
[b] BDL: below the detection limit (5 ppb).
[c] One-way ANOVA (95 % CI) for different working areas (statistical significance was set at $p < 0.05$).

Studies revealed that the desirable indoor air temperature in hospital environments range between 20 and 24 °C according to international standards

[59,61,67,68,168,169]. However temperature can be sometimes slightly higher or lower when medical situations and patient comfort require such conditions [68,170]. It is important to mention that lower temperatures favors occupant discomfort including shivering, muscular and joint tension [44,46,68], while higher temperatures can increase out-gassing of toxins from building materials and provide a more favorable growing conditions for bacteria and their transport / migration mechanisms from and to the patients [44,46,68]. High relative humidity can activate viruses and promote bacterial growth, increase susceptibility to respiratory diseases and affect comfort safety and health of patients, visitors and medical personnel [44,46,68]. Low values of RH can increase blood coagulation, favors skin and throat drying and cause thermal discomfort. According to the international standards [59–61,169], it is recommended that levels of RH should range between 30 and 60 % (see Appendix C). Throughout the monitoring program of this research study, optimum temperatures were recorded in all working areas, while relatively higher RH values were recorded at the reception (58 ± 1.6%), the corridor (63 ± 1.7%) and the ER (63 ± 0.6%) that might activate viruses and bacterial growth, and hence affect the indoor environmental quality (IEQ) of such areas. However some studies have shown that RH can be greater than 60 % in some critical indoor spaces of hospitals and healthcare facilities to prevent the accumulation of static electricity at the emergency and operating / surgery rooms where volatile liquids and inflammable anesthetic gases are frequently used [44,68].

PM_{10}, $PM_{2.5}$, CO, CO_2, and TVOC varied significantly ($p < 0.05$) across the monitored areas. Indoor $PM_{2.5}$ and PM_{10} concentrations were significantly different

among sampling sites ($p < 0.05$). The clinic ($PM_{2.5}$: 68.7 ± 9.6 μg/m^3; PM_{10}: 77.4 ± 12.1 μg/m^3) and reception ($PM_{2.5}$: 65.2 ± 13.8 μg/m^3; PM_{10}: 78.6 ± 7.8 μg/m^3) areas exhibited high PM_{10} and $PM_{2.5}$ levels exceeding WHO standards of 25 μg/m^3 for $PM_{2.5}$ and 50 μg/m^3 for PM_{10} by 57 and 175 %, respectively (Table 11). Other critical indoor environments such as the pediatrics patient room ($PM_{2.5}$: 12.2 ± 2.0 μg/m^3; PM_{10}: 26.2 ± 2.9 μg/m^3), ER ($PM_{2.5}$: 22.6 ± 3.2 μg/m^3; PM_{10}: 29.8 ± 4.9 μg/m^3), workshop ($PM_{2.5}$: 21.5 ± 6.5 μg/m^3; PM_{10}: 37.9 ± 4.1 μg/m^3) and BMT waiting area ($PM_{2.5}$: 13.9 ± 5.1 μg/m^3; PM_{10}: 25.6 ± 3.6 μg/m^3) exhibited relatively low levels of particulate matter and were within the WHO standards. In contrast to previous studies done in hospital settings, Lomboy et al. [143] reported an average $PM_{2.5}$ of 30μg/m^3 in pediatrics patient room, almost three folds those recorded in this study, while Jung et al. [35] measured lower $PM_{2.5}$ concentrations at a hospital clinic area (17.2 ± 11.9 and 29 ± 22.1μg/m^3 for $PM_{2.5}$ and PM_{10}, respectively). Similarly, Nardini et al. [82] measured $PM_{2.5}$ at a high of 27 ± 10.6 to 107.1 ± 47.8 μg/m^3 (at areas with no smoking restrictions) and a low of 1.6 ± 0.9 to 14.8 ± 2.2 μg/m^3 (at areas with smoking restrictions).

At all sampling locations, average indoor levels of CO were lower than the NAQQS standards (9 ppm for 8 hours and 35 ppm for 1 hour) as shown in Table 11. Previous studies [19,35] have reported similar levels of CO in hospital environments (meeting rooms, lobbies, central laboratory and polyclinics) with an average of 2.7 ± 1.2 ppm. ASHRAE recommends a minimum indoor building ventilation rate of 10 L/s per person [59,61,168,169], which corresponds to an approximate steady state indoor concentration of 870 ppm for CO_2 [59]. The CO_2 levels are usually greater inside

buildings than outdoors, where CO_2 concentration is often used as an indicator to assess the efficiency of ventilation [59,126]. When CO_2 levels exceed 1000 ppm in indoor environments, it is advisable and recommended to enhance the air exchange by increasing ventilation rates [59,126]. In this case, indoor CO_2 concentrations were below the 1000 ppm threshold limit with maximum concentrations recorded at the clinic (722 ± 32.2 ppm) and lobby (616 ± 33.1 ppm) working areas which is consistent with other studies done recently where similar levels of CO_2 were reported at the clinic (655 ± 157 ppm) and lobby (643 ± 187 ppm) areas of hospitals [35].

While most working areas didn't show alarming levels of TVOC (Table 11), relatively higher concentrations of TVOC were recorded at the corridor (0.23 ± 0.04 ppm), meeting room (0.19 ± 0.03 ppm), BMT waiting area (0.18 ± 0.07 ppm), and Opthamology PR (0.19 ± 0.06 ppm). Although other studies [19,35] reported higher concentration of TVOC, with some levels twice as high at the clinic (0.42 ± 0.31 ppm), pharmacy department (0.71 ± 0.29 ppm), and lobby (0.52 ± 0.45 ppm) areas. In this study, the measured concentrations of TVOC were lower than standards and below the threshold level of 3 ppm [59,69,103,126].

C. Air Quality Indices and Health Concern Levels

Scores of air quality index (AQI) of working areas are presented in Table 12. Good air quality with AQI < 50 causing least risk was experienced at the pediatrics patient room, ER, basement workshop and BMT waiting area. The lobby, staff office, corridor, meeting room and opthamology patient room exhibited AQI values between

50 and 100 mainly due to high occupancy rate resulting in relatively greater levels of particulate matter (PM_{10} and $PM_{2.5}$) exceeding thresholds at some locations, and indicating moderate health concerns for individuals sensitive to air pollution. The clinic and reception areas experienced an unhealthy air quality for sensitive groups (such as children and elderly people) with AQI values of 107.1 and 101.2, respectively, mainly due to high levels of $PM_{2.5}$ and PM_{10} (clinic: $PM_{2.5}$: 68.7 ± 9.6 µg/m^3; PM_{10}: 77.4 ± 12.1 µg/m^3 and reception: $PM_{2.5}$: 65.2 ± 13.8 µg/m^3; PM_{10}: 78.6 ± 7.8 µg/m^3) exceeding guideline values by 2.75 and 1.57 folds respectively. High levels of $PM_{2.5}$ and PM_{10} were obtained due to the proximity of the clinic (1st floor) and reception (ground floor) sampled areas to on-road vehicular emissions and to construction activities. In addition to dust storms originating from Arabian deserts in Middle East and African Saharan desert in Africa occurred during sampling at these locations, which carried high levels of particulate matter (PM) [87] and increased indoor levels of $PM_{2.5}$ and PM_{10} at the clinic and reception working areas.

Table 12. AQI values and health concern levels of sampled working areas

ID	Working Areas	Floor	AQI values	Health Level [a]
1	Clinic	1st	107.1	Unhealthy for sensitive groups
2	Clinic waiting area	5th	57.1	Moderate
3	Lobby	10th	51.3	Moderate
4	Reception	Ground	101.2	Unhealthy for sensitive groups
5	Staff office	7th	51.9	Moderate
6	Corridor	8th	58.1	Moderate
7	Pediatrics PR	6th	32.2	Good
8	ER	Ground	41.3	Good
9	Workshop	Basement	45.7	Good
10	Meeting room	8th	53.7	Moderate
11	BMT WA	8th	37.6	Good
12	Opthamology PR	7th	51.0	Moderate

[a] Description of health concern levels of AQI is provided in Appendix H.

Table 13 presents the CIAI results for individual air pollutants and their corresponding integrated index (I_p) values at sampled working areas. Only one working area (i.e. pediatrics patient room) experienced a good IAQ with I_p value of 43.7. While most sampled working areas showed a moderate air quality with I_p values ranging from 59.0 (BMT waiting area) to 87.9 (clinic waiting area). Higher I_p values were recorded at the clinic (114.7) and reception (113) working areas indicating an unhealthy air quality for sensitive groups such as children and elderly people. CIAI scores of PM_{10} and $PM_{2.5}$ were greater than other pollutants which influenced the I_p results. Most I_p values corresponded to $PM_{2.5}$, thus reflecting the dominance of $PM_{2.5}$ pollution in most sampled locations, particularly at the clinic and reception working areas (Table 13).

Table 13. CIAI scores and health concern levels of sampled working areas

ID	Working Areas	Floor	CIAI values of air pollutants – Integrated Index						Health Level [b]
			PM_{10}	$PM_{2.5}$	CO	CO_2	TVOC	I*p*[a]	
1	Clinic	1st	97.4	114.7	30.4	72.7	NA[c]	114.7	Unhealthy for sensitive groups
2	Clinic waiting area	5th	62.2	87.9	13.6	44.2	5.7	87.9	Moderate
3	Lobby	10th	54.6	72.6	17.3	62.3	NA	72.6	Moderate
4	Reception	Ground	98.6	113.0	27.4	58.8	NA	113.0	Unhealthy for sensitive groups
5	Staff office	7th	56.8	74.9	19.3	54.4	NA	74.9	Moderate
6	Corridor	8th	65.7	81.3	17.6	48.7	7.7	81.3	Moderate
7	Pediatrics PR	6th	43.7	40.7	15.1	43.2	NA	43.7	Good
8	ER	Ground	49.7	64.5	16.6	38.1	NA	64.5	Moderate
9	Workshop	Basement	57.9	62.1	18.6	46.4	NA	62.1	Moderate
10	Meeting room	8th	57.5	75.0	17.3	57.8	6.3	75.0	Moderate
11	BMT WA	8th	42.7	46.2	15.8	59.0	5.8	59.0	Moderate
12	Opthamology PR	7th	60.2	67.4	21.0	48.7	6.2	67.4	Moderate

[a] The highest score is used as integrated index value (*Ip*) when calculating CIAI scores for each air pollutant, and if there are more than two indices in the "unhealthy for sensitive groups", the index with higher value receives more weightage [81].
[b] Description of concern levels of CIAI as suggested by *Environmental Protection Agency* (EPA) is provided in Appendix I.
[c] Not applicable

Table 14 presents the MCR values across the sampled locations. Some critical environments such as the pediatrics patient room, ER, basement workshop, BMT waiting area and ophthalmology patient room fell in the concern zone for the combined effect by several substances (Group IIIB), while the clinic, clinic waiting, lobby, reception, staff office, corridor and meeting room working areas revealed a single substance concern classification (Group I) (Table 14). As per MCR results, it is clear that none of the sampled areas of hospitals have shown low concern (Group II) or concern for combined effect dominated by one substance (Group IIIA). MCR computations have highlighted the importance of exposure to multi-pollutants as well as the the mixture effect of contaminants and not only individual pollutant [80,99,100,102]. A chemical-by-chemical assessment wouldn't have identified any air

quality concern for some working areas such as pediatrics PR, ER, basement workshop, BMT waiting area and ophthalmology PR since *max* $HQ_i < 1$ (Table 14). There was a significant decline ($r = -0.766$) of MCR as HI values increased across the sampled working areas (Figure 9). Group I sampled working areas such as the clinic, clinic waiting area, lobby, reception, staff office, corridor and meeting room working areas have shown large *HI* values and lower MCR scores. Furthermore, the highest values of *HI* were reported particularly at the clinic and reception working areas reaching a high of 5.35 and 5.06 respectively, as a result of high *HQ* values of PM_{10} and $PM_{2.5}$ obtained at the clinic ($HQ_{PM10} = 1.55$; $HQ_{PM2.5} = 2.75$) and reception areas ($HQ_{PM10} = 1.57$; $HQ_{PM2.5} = 2.61$) (Table 14). Finally, it is evident that indoor air of Group I working areas (i.e. the clinic and reception areas in particular) contained at least one substance or air contaminant that poses serious health risks (*HI* > 5 and MCR < 2) to indoor occupants of hospitals. Since MCR was found to be small relative to *n* in most of the sampled working areas, this indicated that the toxicity was in general driven by only few chemical pollutants, mainly PM_{10} and $PM_{2.5}$.

Table 14. MCR results and health concern levels of sampled working areas

ID	Working Areas	Floor	HQ values of air pollutants							Health Level [b]
			PM_{10}	$PM_{2.5}$	CO	CO_2	TVOC	HI [a]	MCR [a]	
1	Clinic	1st	1.55	2.75	0.34	0.72	NR	5.35	1.95	Group I
2	Clinic waiting area	5th	0.84	1.36	0.15	0.44	0.06	2.86	2.10	Group I
3	Lobby	10th	0.69	1.06	0.19	0.62	NR	2.56	2.41	Group I
4	Reception	Ground	1.57	2.61	0.30	0.58	NR	5.06	1.94	Group I
5	Staff office	7th	0.74	1.11	0.21	0.54	NR	2.59	2.34	Group I
6	Corridor	8th	0.91	1.23	0.20	0.49	0.08	2.91	2.36	Group I
7	Pediatrics PR	6th	0.52	0.49	0.17	0.43	NR	1.61	3.07	Group IIIB
8	ER	Ground	0.60	0.90	0.18	0.38	NR	2.07	2.28	Group IIIB
9	Workshop	Basement	0.76	0.86	0.21	0.46	NR	2.28	2.66	Group IIIB
10	Meeting room	8th	0.75	1.11	0.19	0.57	0.06	2.69	2.42	Group I
11	BMT WA	8th	0.51	0.55	0.18	0.58	0.06	1.88	3.23	Group IIIB
12	Opthamology PR	7th	0.80	0.96	0.23	0.49	0.06	2.55	2.65	Group IIIB

[a] See Appendix J for mathematical functions of *HI* and MCR.

[b] Health concern levels of MCR and group description is provided in Appendix J.

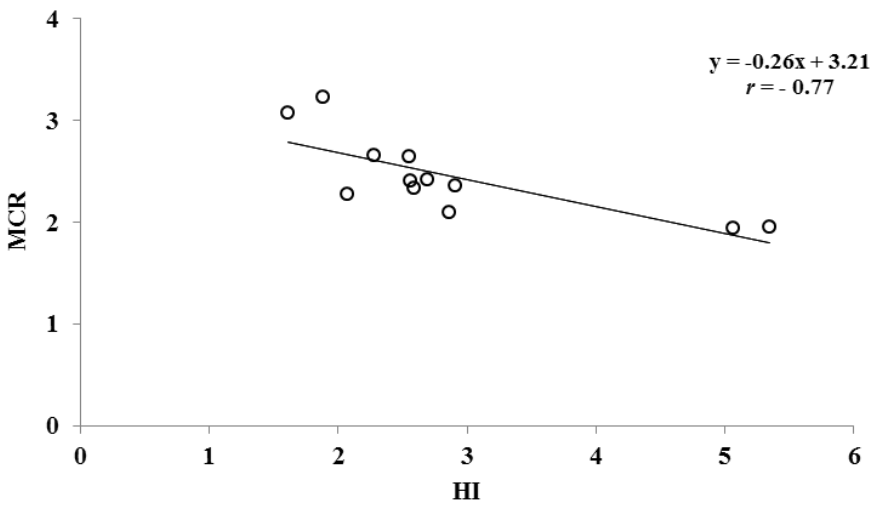

Figure 9. Scatter plot of MCR versus HI of sampled working areas

D. RIAQI Levels and Health Implications

Table 15 summarizes the results of newly developed indices DI and RIAQI. In general, most of the sampled areas inside hospitals have shown acceptable T and RH values that are within their standards, thus denoting high DI values with low discomfort and acceptable indoor environmental quality (IEQ) (Table 15). However due to relatively higher RH values recorded at the reception (58 ± 1.6%), corridor (63 ± 1.7%) and ER (63 ± 0.6%) working areas, lower DI scores with values of 85, 60 and 60 were obtained for those areas, respectively (Table 15). In most sampled areas of hospitals, the concentrations of air contaminants were below standards creating a satisfactory and "good" IAQ (Table 15). On the other hand, $PM_{2.5}$ levels exceeded the standards in some critical areas such as the clinic, clinic waiting area, reception and corridor reaching high concentrations of 68.7, 34.1, 65.2 and 30.9µg/m^3, respectively, which influenced the RIAQI results particularly at the clinic (RIAQI = 72) and the reception (RIAQI = 73) sampled areas. As a result, such areas had an "unhealthy for sensitive groups" air quality (Table 15) due to high levels of PM_{10} and $PM_{2.5}$ that are mainly attributed to the movement of indoor occupants, dusty conditions during sampling, and proximity of the sampling sites to construction activities and to on-road vehicular and generator emissions that are ubiquitous in urban settings as discussed earlier (Table 15).

Table 15. RIAQI scores and health concern levels of sampled working areas

ID	Working Areas	Floor	DI [a]	RIAQI [b]	Health Level [d]
1	Clinic	1st	100	72	Unhealthy for sensitive groups
2	Clinic working area	5th	90	98	Good
3	Lobby	10th	100	99	Good
4	Reception	Ground	85	73	Unhealthy for sensitive groups
5	Staff office	7th	100	99	Good
6	Corridor	8th	60	99	Good
7	Pediatrics patient room	6th	100	100	Good
8	Emergency room (ER)	Ground	60	99	Good
9	Basement workshop	Basement	100	99	Good
10	Meeting room	8th	90	99	Good
11	BMT WA	8th	100	100	Good
12	Opthamology PR	7th	95	99	Good

[a] DI is given in Equation (3)
[b] RIAQI is described in Equation (5)
[c] Health concern levels of RIAQI are defined in Table 4 (see Chapter II).

In this research study, the high concentrations of $PM_{2.5}$ and PM_{10} that were reported at the clinic and reception working areas had direct effect on RIAQI scores, thus categorized the air quality of clinic and reception working areas as “unhealthy for sensitive groups” with problematic implications for sensitive indoor occupants such as children and elderly people. It is noteworthy to mention that the clinic and reception working areas were frequently occupied with individuals of different age (children and adults) and various-function groups (patients, visitors and medical staff). Hence, indoor occupants of such areas were at higher risk of health related symptoms as well as respiratory and cardiovascular diseases that are mainly caused by PM pollution, and mostly by fine particulate matter $PM_{2.5}$ that penetrates deeply into the bronchi and alveoli regions of the lungs [5,13,74,106,111,119,171]. Furthermore, studies continue to find an increase in health risk with increasing PM_{10} and $PM_{2.5}$ exposure and have demonstrated that individuals with pre-existing diseases such as heart and pulmonary

diseases or myocardial infarction might have higher risk of acute exacerbation on days having high levels of particulate matter [106,119,151,172]. Based on these findings, the hospitals administration and infection preventionists (IPs) should take more rigorous and protective actions to limit the adverse effects of indoor air pollution and to promote and sustain a healthy indoor environment for sensitive groups such as children, and elderly with lung and heart diseases.

E. Comparative Assessment

Misclassifications of air quality levels in medical environments sometimes result in problematic protection recommendations affecting hospital infection control team and indoor occupants. We have found different health concern levels of air quality inside hospitals among the four indices (AQI, CIAI, MCR and RIAQI). Figure 10 presents the % frequency of IAQ levels of the tested air quality indices. According to AQI results, around 33 % of the sampled areas in hospitals experienced a good IAQ, while CIAI calculations have shown only 8 % of these areas had good IAQ (Figure 10). Moreover, 50 % of the tested areas by AQI experienced a moderate IAQ while around 75 % had a moderate IAQ as per CIAI computations. Both, AQI and CIAI indices have revealed that 17% of the sampled areas had an “unhealthy for sensitive groups” air quality. Although most of the sampled areas had a statisfactory IAQ, however RIAQI results revealed that 17 % of the sampled working areas had an “unhealthy for sensitive groups” air quality which included the clinic and reception working areas. On the other hand, results of MCR have shown significant variability and clear differences in IAQ of the sampled working areas (Figure 10). Around 58 % of the sampled areas had a single

substance concern (i.e. Group I), while 42 % were predominantly classified in Group IIIB (i.e. concern for combined effect by several substances) (Figure 10). In contrast to Group IIIB areas, Group I indoor environments had higher *HI* index (*HI* = 5.35 at clinic and *HI* = 5.06 at reception) and higher *max HQ* values (max HQi > 1) ranging between 2.56 and 5.35, and 0.75 and 2.75 respectively. This indicates a single substance concern primarly attributed to $PM_{2.5}$. In contrast to AQI and CIAI indices, MCR results have shown poor air quality levels due to multiple pollutants effect in most of the sampled locations. On the other hand AQI, CIAI and RIAQI results have reflected a single pollutant concern, namely attributed to $PM_{2.5}$ neglecting the health risks of other contaminants.

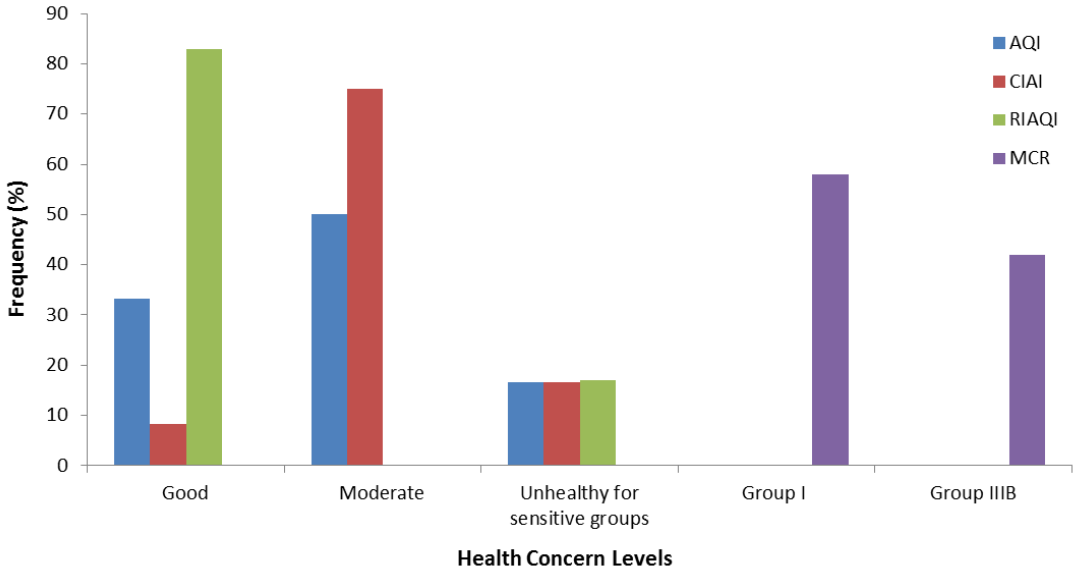

Figure 10. The percent (%) frequency and health concern levels of the air quality across the tested indices inside hospitals (See Appendix J for MCR group classifications: Group I and Group IIIB health concerns).

F. Limitations

The tested indices are arbitrary and differ in their classifications of air quality of hospital environments. Nevertheless, it is important to note the limitations since field data and measurements at hospitals were randomly based (i.e. air samples collected at each location varied with accessibility) and not taken on a daily basis, so the comparisons of effect are less direct than they would be with daily measurements of air contaminants. The AQI and CIAI indices do not take into account the combined effects of all pollutants while MCR does, and may have also underestimated the health risk of some environments associated with exposure to multi-pollutant indoor air pollution which MCR does not. Even though we may not have fully replicated the existing indices and have not shown consistent IAQ levels in some indoor environments of hospitals, however the existing indices used in this research study served as exploratory step towards developing an integrative indicator taking into consideration thermal comfort and IAQ in vulnerable environments such as hospitals. The limitations of the tested indices are presented in Table 16. Finally, it is noteworthy to mention that the developed RIAQI index is intended for use to quantify the state of the indoor environment with regards to chronic exposures and cannot be used when concentrations exceed health standards set for acute exposure. This is particularly true for CO, CO_2 and TVOC concentrations as some studies reported potential neurological damage at high levels [7,19,111,124]. In the future, relative weights of air contaminants will be further introduced to RIAQI based on experts in the field recommendations, with an aim to

improve this novel index and reveal the health risks of each contaminant in critical indoor environments.

Table 16. Existing air quality indices with corresponding limitations

Air quality index	Limitations	References
Air Quality Index (AQI)	− Similar importance is attached to all air quality indicators due to the absence of relative weights − Limited capacity to define risk intervals according to the indoor environment − Does not consider exposure response relationships − Does not consider thermal comfort data within the index	[97]
Comprehensive Indoor Air Quality Index (CIAI)	− The air quality indicators with the highest index value receives more weight and is used as the integrated index value (i.e. no mixture or average effect of contaminants is considered) − Biased to extreme or maximum index values that might result from potential outliers. − Does not consider exposure response relationships − Limited capacity to define risk intervals according to the indoor environment − Do not consider thermal comfort data within the index	[81]
Maximum Cumulative Ratio (MCR)	− Assumes that air pollutants may provoke the same endpoint (i.e. dose addition assumption) − Does not consider thermal comfort data within the index.	[80,99,100,102]

G. Recommendations

The developed (DI) and (RIAQI) indices provide a suitable risk management tool for evaluating thermal comfort and IAQ in critical environments such as hospitals. Given the limitations exhibited by the tested indices and presented before in Table 16, ultimately the new index RIAQI allows for the use of different air quality parameters

and for the establishment of different score functions by modifying the functional forms of the proposed score values. The RIAQI was developed and tested to allow use of different parameters, establishment of different objectives and guideline values, in addition to assessing mixture effects of air pollutants and can provide weights to air contaminants which considers the exposure – response relationships of pollutants through its mathematical functions. RIAQI is also capable of accounting for limited data and allows the user to set the assessment scale based on a set of easy-to-define metrics which renders it flexible for users. RIAQI can be best used to assess the sensitivity to small changes that affect the air quality and have shown consistent and reasonable score results as a function of the parameters' violations of each of the working areas sampled at the hospitals.

In closure, we recommend hospital administration, decision-makers and public health planners to use RIAQI as a tool to develop more stringent air quality regulations and ensure safe conditions in critical indoor environments. The use of such index can be extended to assess other indoor settings including residential and working environments such as apartments, schools and offices. Future improvement to the RIAQI will focus on integrating concentrations of additional indoor indicators such as bioaerosols including bacteria, fungi and molds which are of great significance in hospitals and healthcare facilities.

CHAPTER V

DETECTION OF VIRUSES IN PATIENT ROOMS

A. Introduction

Hospitals and healthcare facilities represent unique indoor environments with individuals at high risk of exposure such as medical staff or commonly known as healthcare providers (HCPs), patients and visitors. Since hospitals act as healthcare centres for critically ill patients to receive treatment and recover from illness, such environments must exhibit clean air and a healthy indoor environment that enhance their recovery, and maintains comfort and health of visitors' and staff [55]. The time spent indoors in the hospital depends on the health condition of the patient, allocated time for visitors, and the working shifts of the staff. Generally, hospital environments have an invariably continuous high occupancy. While commercial buildings may close on certain days and remain unoccupied for specific periods of time, hospital buildings are always occupied [9,173]. Thus, maintaining high standards that reflect the requirements and expectations of indoor occupants is a continuous concern for healthcare facilities. Pathogens in hospital environments can be transmitted through airborne particles, fomites, respiratory droplets and direct contact with bodily fluids [174,175]. At such sensitive environments, data on airborne viruses and the factors that promote their presence and potential spread within hospitals is limited. Therefore, understanding the

context of environmental contamination in hospital settings is essential to inform interventions and control the spread of hospital acquired infection.

Influenza is a highly contagious respiratory virus that occurs worldwide. Annually, 5–10% of adults and 20–30% of children are estimated to encounter an influenza infection resulting in up to 650,000 deaths globally [176,177]. The elderly and children under 5 years of age account for the majority of fatal cases [176,178–180]. There are four types of influenza virus (A – D) with A and B types being known to cause seasonal influenza outbreaks among humans [181,182]. Influenza A virus has numerous subtypes and a wide host range; they are of particular concern as they have historically caused several pandemics [183,184]. Seasonal influenza outbreaks are caused by influenza A/H1N1 and A/H3N2 subtypes, with other subtypes (e.g. H5N1 and H7N9) causing sporadic human infections [184,185]. Influenza viruses can be transmitted directly through airborne droplets among susceptible humans in crowded areas and community settings such as households, day-care centres, nursing homes and school classrooms, or indirectly through contact with contaminated hands, fomites, and various surfaces [177,180,186,187]. On the other hand, nosocomial transmission of influenza is a major concern because hospitalized patients might be more vulnerable to severe diseases if infected [179,180,188–191]. Influenza viruses can spread through respiratory droplets or aerosols in the air through different mechanical mechanisms such as sneezing, coughing, speaking and even exhaled breath of an infected person that generates particles with a geometric mean diameter of 13.5 µm (range 1-1000 µm) [29,179,192]. Observational and epidemiological studies have revealed that airborne

influenza transmission occurs among humans and can be contracted by inhaling small particles containing even low virus titer [192–194].

Respiratory syncytial virus (RSV) is another virus that is highly contagious and spreads through droplets [195–197]. RSV is a major cause of moderate to severe respiratory infection with a significant burden in terms of mortality particularly in children and the elderly [198–200]. In 2015, it was estimated that 33.1 million episodes of RSV resulted in about 3.2 million hospitalizations and around 60,000 in-hospital deaths among children younger than 5 years [199]. RSV is also associated with nosocomial infections since it is via direct contact or via large nasopharyngeal secretion droplets from infected individuals [195–197,199,201]. Vulnerable patients who are infected with RSV are likely to experience longer hospital stays with increased risk of morbidity and mortality [200,202,203].

Monitoring and control of microbiological contaminants including respiratory viruses in hospitals' air has become an integral part of prevention strategies against hospital-acquired infections [122,204]. Several international organizations such as the Institute of Medicine, the European Center for Disease and Control, the Centers for Disease Control and prevention (CDC) and the World Health Organization (WHO) highlighted the need for further research regarding influenza virus transmission routes [176,177,182,184,190,205–207]. In this context, large droplets were widely reported to play a predominant role in influenza transmission; as such, infection control guidelines recommends a minimum spacing of 1 m between patient beds and between the patients and the health care providers [191,208,209]. However, this guideline remains

controversial with more evidence needed to support the role of aerosols in influenza transmission. Despite their major impact on human health, the science of influenza and respiratory viruses remains ambiguous and misunderstood in critical indoor environments such as healthcare facilities. In order to better understand the potential for transmission of influenza and respiratory syncytial viruses within the hospital environment, we investigated the extent of airborne influenza in patient rooms. Air samples were collected at two locations within patient rooms to determine the risk of exposure in relation to distance from the patient. This is the main focus of this Chapter. In addition, air flow inside the patient's room was simulated using CFD with the aim of defining transmission routes and potential "hot zone" for transmission within the room. The latter is further discussed in Chapter VI.

B. Patients' Characteristics

Twenty-nine subjects/patients (11 males and 18 females, 0–90 years of age) admitted to inpatient care unit of the sampled hospital were screened for RSV and influenza virus. Patients were positive for influenza A virus; but none had influenza B. We had three RSV-positive patients however none of their collected air samples yielded positive results. Data of total air samples collected is presented below in Table 17. Emitters were defined as influenza virus-positive subjects based on PCR analysis with at least one virus positive air sample. On the other hand, influenza virus-negative patients were considered as non-emitters. Around 45 % of the males and 72 % of the females were positive for influenza A virus. Seventeen out of twenty-nine recruited patients were emitters; five were males and twelve were females (Table 18). Only one

child among the three recruited children was an emitter. Twelve out of the seventeen adult patients (18 – 64 years old) and four out of nine elderly (> 64 years old) patients were emitters (Table 18). Comparison of virus positivity (i.e. emitters) with age groups showed no statistical significance ($p > 0.05$).

Table 17. Summary of air quality data collected from patient rooms

ID #	Date	Time	Sample ID [a]	Age [b]	Sex [c]	rt - PCR	Emitters [e]	Day of admission	Coughing [f]	Sneezing [f]
1	24/1/2018	9:40 AM	AS1-A	1 m	M	(ND)[d]	No	2	0	0
	24/1/2018	9:55 AM	AS1-B	1 m[h]	M	(ND)	No	2	0	0
2	24/1/2018	10:55 AM	AS2	50 y	F	Influenza A[g]	Yes	3	2	1
3	30/1/2018	11:35 AM	AS3	4 m[h]	F	(ND)	No	3	0	0
4	7/2/2018	11:10 AM	AS4-A	8 m[h]	M	Influenza A	Yes	1	0	0
	7/2/2018	11:25 AM	AS4-B	8 m	M	Influenza A	Yes	1	0	0
5	7/2/2018	11:50 AM	AS5	38 y	F	Influenza A	Yes	2	3	1
6	7/2/2018	12:10 AM	AS6-A	40 y	F	Influenza A	Yes	1	1	0
	7/2/2018	12:25 AM	AS6-B	40 y	F	Influenza A	Yes	1	1	1
7	9/2/2018	9:00 AM	AS7 - A	45 y	M	Influenza A	Yes	1	1	1
	9/2/2018	9:15 AM	AS7 - B	45 y	M	Influenza A	Yes	1	1	0
8	9/2/2018	9:40 AM	AS8	31 y	F	Influenza A	Yes	1	1	0
9	9/2/2018	10:00 AM	AS9- A	23 y	M	Influenza A	Yes	1	2	1
	9/2/2018	10:15 AM	AS9- B	23 y	M	Influenza A	Yes	1	1	1
10	12/2/2018	10:30 AM	AS10 - A	51 y	F	Influenza A	Yes	1	2	1
	12/2/2018	10:45 AM	AS10 - B	51 y	F	Influenza A	Yes	1	1	1
11	12/2/2018	11:10 AM	AS11-A	38 y	F	Influenza A	Yes	1	1	1
	12/2/2018	11:25 AM	AS11-B	38 y	F	Influenza A	Yes	1	1	0
12	12/2/2018	11:40 AM	AS12	91 y	F	Influenza A	Yes	1	2	1
13	15/2/2018	9:25 AM	AS13- A	55 y	F	Influenza A	Yes	1	2	1
	15/2/2018	9:40 AM	AS13 - B	55 y	F	(ND)	No	1	0	0
14	15/2/2018	10:00 AM	AS14 - A	80 y	M	Influenza A	Yes	1	2	2
	15/2/2018	10:15 AM	AS14 - B	80 y	M	Influenza A	Yes	1	1	1
15	21/2/2018	10:15 AM	AS15 - A	70 y	F	Influenza A	Yes	1	1	2
	21/2/2018	10:30 AM	AS15 - B	70 y	F	Influenza A	Yes	1	1	1
16	21/2/2018	10:45 AM	AS16 - A	39 y	F	Influenza A	Yes	1	1	2

	21/2/2018	11:00 AM	AS16-B	39 y	F	Influenza A	Yes	1	1	0
17	21/2/2018	11:15 AM	AS17-A	31 y	F	Influenza A	Yes	2	1	0
	21/2/2018	11:25 AM	AS17-B	31 y	F	(ND)	No	2	1	0
18	21/2/2018	11:45 AM	AS18-A	53 y	F	(ND)	No	2	1	0
	21/2/2018	11:55 AM	AS18-B	53 y	F	(ND)	No	2	0	0
19	2/3/2018	9:00 AM	AS19-A	23 y	F	(ND)	No	2	1	0
	2/3/2018	9:10 AM	AS19-B	23 y	F	Influenza A	Yes	2	1	1
20	2/3/2018	9:25 AM	AS20-A	77 y	M	Influenza A	Yes	2	0	2
	2/3/2018	9:40 AM	AS20-B	77 y	M	(ND)	No	2	1	0
21	2/3/2018	9:55 AM	AS21-A	50 y	M	(ND)	No	2	1	1
	2/3/2018	10:05 AM	AS21-B	50 y	M	(ND)	No	2	0	0
22	2/3/2018	10:20 AM	AS22-A	18 y	F	(ND)	No	3	1	0
	2/3/2018	10:30 AM	AS22-B	18 y	F	(ND)	No	3	0	0
23	8/3/2018	10:55 AM	AS23-A	85 y	F	(ND)	No	2	1	1
	8/3/2018	11:10 AM	AS23-B	85 y	F	(ND)	No	2	0	1
24	8/3/2018	11:25 AM	AS24-A	45 y	F	(ND)	No	2	1	0
	8/3/2018	11:40 AM	AS24-B	45 y	F	(ND)	No	2	0	0
25	8/3/2018	11:55 AM	AS25-A	86 y	F	(ND)	No	3	1	1
	8/3/2018	12:10 AM	AS25-B	86 y	F	(ND)	No	3	1	0
26	12/3/2018	10:00 AM	AS26-A	70 y	M	(ND)	No	3	0	1
	12/3/2018	10:10 AM	AS26-B	70 y	M	(ND)	No	3	0	0
27	12/3/2018	10:25 AM	AS27-A	88 y	M	(ND)	No	3	1	1
	12/3/2018	10:40 AM	AS27-B	88 y	M	(ND)	No	3	0	1
28	12/3/2018	10:55 AM	AS28	70 y	M	(ND)	No	3	0	0
29	12/3/2018	11:10 AM	AS29	36 y	M	(ND)	No	3	0	0

[a] Two samples were collected from patient rooms, samples that were close to patient (~0.3m) were labelled as "A" while those collected far from patients (~2.2m).were labelled as "B".
[b] Age (m = months; y = years)
[c] Sex (M = male; F = Female)
[d] ND = Not determined.

[e] Emitters were defined as patients surrounded by influenza virus aerosols due to significant C*t* values obtained from rt-PCR analysis and significant coughing / sneezing activities. On the other hand, patients with no influenza virus detected were considered as non-emitters. All patients were on anti-viral medication treatment.
[f] Coughing and sneezing were counted and assessed by the study personnel during each sample collection.
[g] All Influenza positive cases were of Influenza A (H1N1) virus sub-strain.
[h] Only three RSV patients were recruited in this study and their air samples were collected on admission day 2, 3 and 1 respectively, but none has yielded positive results.

Table 18. Patients' characteristics

Age group (years)	Number / size	Males	Females	Virus positive cases	% Influenza A
Children (0 – < 18)	3	2	1	1	33
Adults (18 – 64)	17	4	13	12	71
Elderly (> 65)	9	5	4	4	44
Total (0 – 90)	29	11	18	17	59

Previous studies revealed that not all patients emit influenza virus in a similar way [190,206,207,210], hence the identification of emitters may improve the understanding of influenza virus transmission and provide a clear description of the spatial distribution of such viruses in healthcare settings, particularly inside patient rooms. In this study, we detected influenza viral RNA in 51% of the air samples collected from influenza patient rooms, indicating a potential risk for nosocomial transmission via the airborne route. However, none of the air samples from RSV infected patient rooms were positive. Nonetheless, we only had three patients with RSV precluding any conclusion as to whether influenza virus can be more stably aerosolized compared to RSV.

C. Virus Detection as a Function of Admission Date

Twenty-six out of 51 air samples (51%) tested positive for influenza A virus; 16 were close samples (0.30 m from the patients' head) while the remainder were far (2.2 m from patients' head) (Table 19). Noteworthy, none of the collected air samples yielded viable virus.

Table 19. Frequency of virus detection in proximity to the patient

Location	Number / size	Positive samples	% Influenza A
Close samples [a]	29	16	55
Far samples [b]	22	10	45
Total samples	51	26	51

[a] Air samples collected at 0.30 m distance close to patient's head.
[b] Air samples collected at 2.20 m distance away from patient's head and 0.50 m from room entrance.

Table 20 shows the frequency of influenza A virus-positive air samples as a function of admission date. A higher number of positive influenza A virus cases were obtained on day 1 of hospital admission compared to days 2 and 3, which were associated with a significant decline in viral detection. Around 95 % of the air samples collected on day 1 of admission were positive for influenza virus, while only 24 % and 8 % of the samples collected on days 2 and 3, respectively, were positive. Moreover, all of the close samples collected on day 1 of admission recovered influenza A virus and 90 % of the far samples were positive. In contrast, 33 % of the close samples and 13 % of the far samples were positive on day 2 of admission, and only 13 % of the close samples were positive and none of the far samples have shown any virus positivity on day 3 of admission (Table 20). Coughing and sneezing are two key contributors to airborne virus shedding [190,211–213]. The detection of influenza A virus RNA in 17 patient rooms was consistent with the presence of symptomatic influenza patients. Coughing and sneezing were treated as one variable and it was significantly associated with the detection of influenza virus in the air sample independent of the sampling locations ($p < 0.05$).

Table 20. Virus positivity with respect to the number of days elapsed since hospital admission

Day of admission	Air Samples [a]	Number / size	*rt*-PCR positive samples	% Influenza A
Day 1	Total	22	21	95
	Close [b]	12	12	100
	Far [c]	10	9	90
Day 2	Total	17	4	24
	Close	9	3	33
	Far	8	1	13
Day 3	Total	12	1	8
	Close	8	1	13
	Far	4	0	0

[a] Fifty-one air samples collected in total.
[b] Twenty-nine air samples collected at 0.3 m from patient head.
[c] Twenty-two air samples collected at 2.2 m from patient head and 0.50 m from room entrance

Influenza virus transmission is reported to occur primarily by large droplets traveling up to 1 m from the source [181,182,184]. Here in this research study, we have shown that HCPs and visitors could be at higher risk of influenza virus infection close to patients. However, albeit to a lesser extent, they could be still exposed to virus in the air at a distance up to 2.2 m away from patients with symptomatic influenza virus infection. This raises concerns beyond the current WHO and CDC recommendations (i.e. spacing of 1m) regarding the adequacy of protection to visitors and HCPs during routine care operations in hospitals and similar healthcare facilities.

Influenza virus RNA have reportedly been detected in the air at healthcare settings independent of patients or care activities, particularly in emergency and paediatrics departments [179,190,209]. There was no correlation found between sneezing and coughing and detection of influenza virus in the air samples. However, we have found a significant decline in influenza virus shedding and dispersal as longer time had elapsed from the day of admission to hospital. Previous studies have shown that the

majority influenza virus shedding occurs within the first two days of illness and that the time to shedding cessation is usually faster in adults compared to children [214,215]. Consistently, the highest detection rate of aerosolized influenza was detected within one day of hospital admission in our study and declined in subsequent days. Such findings are also in line with few other studies targeting environments different than hospital settings, such as college communities and multicentre areas which have shown negative association between the detection of aerosolized influenza A virus and the time elapsed since illness onset [29,192].This fast decline of airborne virus detection is also consistent with the fact that the majority of our patients were adults. However, we could not confirm the infectivity of the aerosolized virus using plaque assay. We cannot confirm whether this lack of infectious virus detection reflects inactivation of the virus in the air inside the patient room or due to negative effect of the collection method. Further studies are needed to more closely assess this.

Some limitations include the limited sample size which precludes analysis of more complex variables that might affect the presence of airborne viruses. Also, we included samples from symptomatic patient rooms only and in departments that were accessible for this type of work, thus excluding asymptomatic virus emitter and the emergence department, which might be a hub for dissemination of respiratory viruses.

CHAPTER VI

CFD MODELING

A. Introduction

Hospitals are special buildings that operate on a twenty-four hour basis where different groups of people occupy their indoor premises and distinct indoor sources of atmospheric pollutants are present including biological and chemical contaminants [36,49,189,202,216]. The indoor environment of hospitals is complex and different from other commercial or residential buildings. While IAQ at hospitals is affected by a multitude of indoor and outdoor sources, it has not been examined adequately and requires further attention using numerical analytical techniques.

Particulate matter (PM_{10} and $PM_{2.5}$), bioaerosols (viruses and bacteria) and gaseous compounds (CO, SO_2, NO_x and TVOC) are considered the main indoor air pollutants that may act as infectious agents through respiratory droplets [217,218]. Evidence has been reported on the close relation between the spread of aerosolized microbes with the air flow pattern and ventilation modes experienced indoors [35,63,219,220]. When the temporal and spatial distributions of costly or difficult-to-monitor IAQ variables are needed, mathematical modeling is relied upon with multi-zone modeling and Computer Fluid Dynamics (CFD) techniques being most promising in this context [90,91,221–223]. Such techniques have been widely applied in residential buildings and industrial facilities to simulate air quality and contaminant

dispersion [90,91,222,224–227] with limited work reported at hospitals [224,228,229]. In this chapter we discuss the mechanism of airflow associated with the distribution of $PM_{2.5}$ pollution levels within hospital's confines (i.e. corridor) and define transmission routes and potential "hot zone" for transmission of Influenza virus within the patient room.

B. $PM_{2.5}$ Mass Fraction Simulations

Figure 11 depicts the concentration contours and velocity vectors of $PM_{2.5}$ mass fraction simulation results under Q = 70 and 120 cfm. The simulated results were compared to experimental field measurements (M_1 – M_8) for model validation (see Chapter II). The average relative errors with Q = 70cfm ranged between 1.71% at M_4 and 9.50% at M_6. While with Q = 120cfm, the average relative errors were between 1.30% at M_4 and 8.33% at M_6. This indicates that the model simulations agreed reasonably well with the experimental data, with relative errors falling below 10% [219].

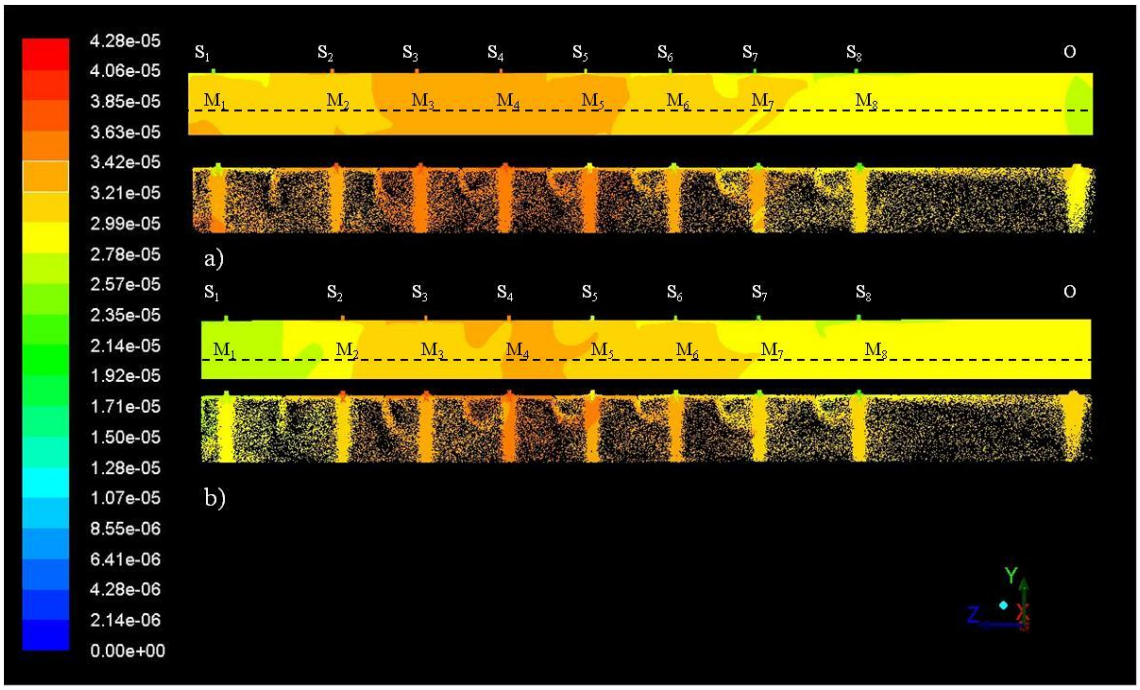

Figure 11. CFD simulations of $PM_{2.5}$ mass fraction (kg/kg air) at a hospital corridor using ANSYS Fluent: **a)** Q = 70cfm, **b)** Q = 120cfm. The color bar represents $PM_{2.5}$ concentrations in kg of $PM_{2.5}$/kg of air. The dashed horizontal line represents the breathing zone.

Mechanical ventilation systems are vital in ensuring proper air exchange rates as well as indoor air distribution that meet standards and requirements of energy savings, thermal comfort and well-being of indoor occupants [91]. They also can directly influence IAQ and have potential impacts on human health. The contours and velocity vectors (Figure 11) reveal that the $PM_{2.5}$ levels in the corridor were non-uniform in both scenarios (Q = 70 and 120 cfm) and exceeded the WHO standard. At Q = 70 cfm, the simulation results indicate that $PM_{2.5}$ levels accumulated in the middle of the corridor (between S_2 and S_6) with values ranging from 3.21×10^{-5} kg/kg (39.3 µg/m^3) to 3.85×10^{-5} kg/kg (47.2 µg/m^3) and spread over a third of the corridor length (~11m). When Q was raised to 120 cfm, the $PM_{2.5}$ levels spread over a tighter space of ~4m or

10% of the corridor length. Concurrently, the $PM_{2.5}$ levels dropped by 33% near the entrance of the corridor (between S_1 and S_2) suggesting that inadequate air exchange rates (Q) and air distribution can lead to indoor pollution stratification and accumulation of $PM_{2.5}$ that can become a health liability particularly that $PM_{2.5}$ may contain potential allergy carriers that can penetrate deeply into the lungs and cause respiratory diseases such as asthma [13,91,105,151]. Since $PM_{2.5}$ levels were significant in the corridor at Q = 70 cfm, it is imperative to either reduce the sources of $PM_{2.5}$ or increase the air exchange (ventilation) rate (i.e. Q = 120cfm) with more outlet exhausts to achieve a reasonable balance between IAQ and thermal comfort of patients, visitors and medical personnel in a hospital environment.

C. Air Flow Patterns Inside Patient Rooms

Evidence has been reported on the close relation between the spread of aerosolized infectious agents with the air flow pattern and the ventilation modes indoors [91]. CFD is one of the most promising and reliable methods which could simulate and evaluate indoor environments. Both the temperature and relative humidity values were within the accepted ASHRAE guidelines and thermal comfort standards of healthcare facilities (i.e. 30 – 60 % for RH, and 20 – 24 ^{o}C for T) [59,169,230]. The temperatures recorded inside patient rooms ranged from 21.5 to 23.5 ^{o}C with an average of 22.5 ± 0.60 ^{o} C, while the measured relative humidity values ranged from 40 to 45% with an average of 43.3 ± 1.34 %. There was no significant association between influenza virus positivity and the measured temperature ($p > 0.05$) or relative humidity values ($p > 0.05$). We performed CFD simulations to determine the potential routes and “hot zones”

of transmission within the patient rooms. Figure 12 presents the results of the CFD simulations of air velocity vectors colored by velocity magnitude at the plane of the patient's bed (Y = 0.9 m) and at the breathing zone level (Y = 1.5 m). Patient rooms displayed a non-uniform airflow where the air velocity ranged between 0.025 m/s and 0.500 m/s. Figure 13 shows the contour plots of the air velocity magnitude inside a patient room at the plane of the patient's bed (Y = 0.9 m) and breathing zone level (Y = 1.5 m). As can be seen from Figure 13, the model predicted air velocities at the plane of the patient's bed (Y = 0.9 m) that were largely comparable to those at breathing zone level (Y = 1.5 m), when the flow rate was increased from 200 to 300 cfm. At a flow rate of 300 cfm, the average air velocities at the breathing zone level in the NW, NE, SE and SW room quadrants were found to be 0.092, 0.333, 0.285, and 0.101 m/s respectively, while the average velocities recorded at the plane of the patient's bed were 0.167, 0.283, 0.275, and 0.101 m/s in the same four quadrants. The average velocities at Q = 200 cfm in the NW, NE, SE and SW quadrants were 0.108, 0.213, 0.187, and 0.083 m/s at Y = 0.9 m and 0.063, 0.250, 0.188, and 0.100 m/s at Y = 1.5 m, respectively. To further explore the air transport in the immediate vicinity of the patient, the air velocity vectors were examined at the plane of the patient head (i.e. Z = 3.2m) that extends into the SE and SW quadrants. These two locations are expected to have a higher occupancy rate by medical staff and visitors (Figure 14). At Q = 200 cfm, a maximum air velocity of 0.210 m/s and an average of 0.173 m/s were attained at the plane of the patient head (Z = 3.2m). On the other hand, higher velocities were evident with Q = 300 cfm recording a maximum velocity of 0.3 m/s and an average of 0.225 m/s at the Z = 3.2 m (Figure

14). This indicates that at Q = 200 cfm indoor occupants might be at higher risk of viral infection at the SW quadrant as compared to Q = 300 cfm scenario since bioaerosols (i.e. influenza virus) are carried at lower elevation (i.e. close to breathing zone level) from SE into SW quadrant of the room (Figure 14).

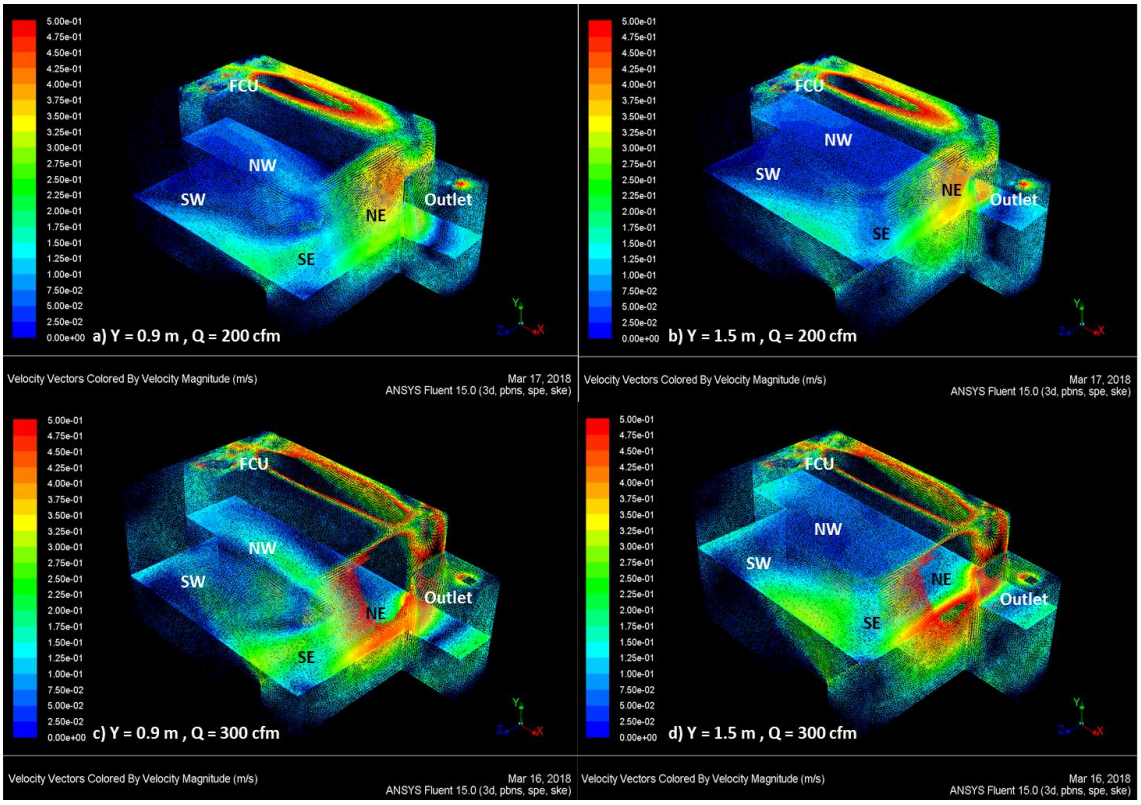

Figure 12. Air velocity vectors colored by velocity magnitude in patient room (side view) at different flows and planes, **a)** Y = 0.9 m and Q = 200cfm **b)** Y = 1.5 m and Q = 200 cfm **c)** Y = 0.9m and Q = 300 cfm and **d)** Y = 1.5 m and Q = 300 cfm. FCU = Fan coil unit includes an inlet, fresh air intake and a return unit. As shown in the CFD plots, the X-axis is oriented from NW/SW towards NE/SE region of the room, the Y-axis is pointing upwards from the floor towards the ceiling of the room and Z-axis is oriented from NW/NE towards SW/SE region of the room. Air samples were collected from the SE and NW quadrants of the patient room.

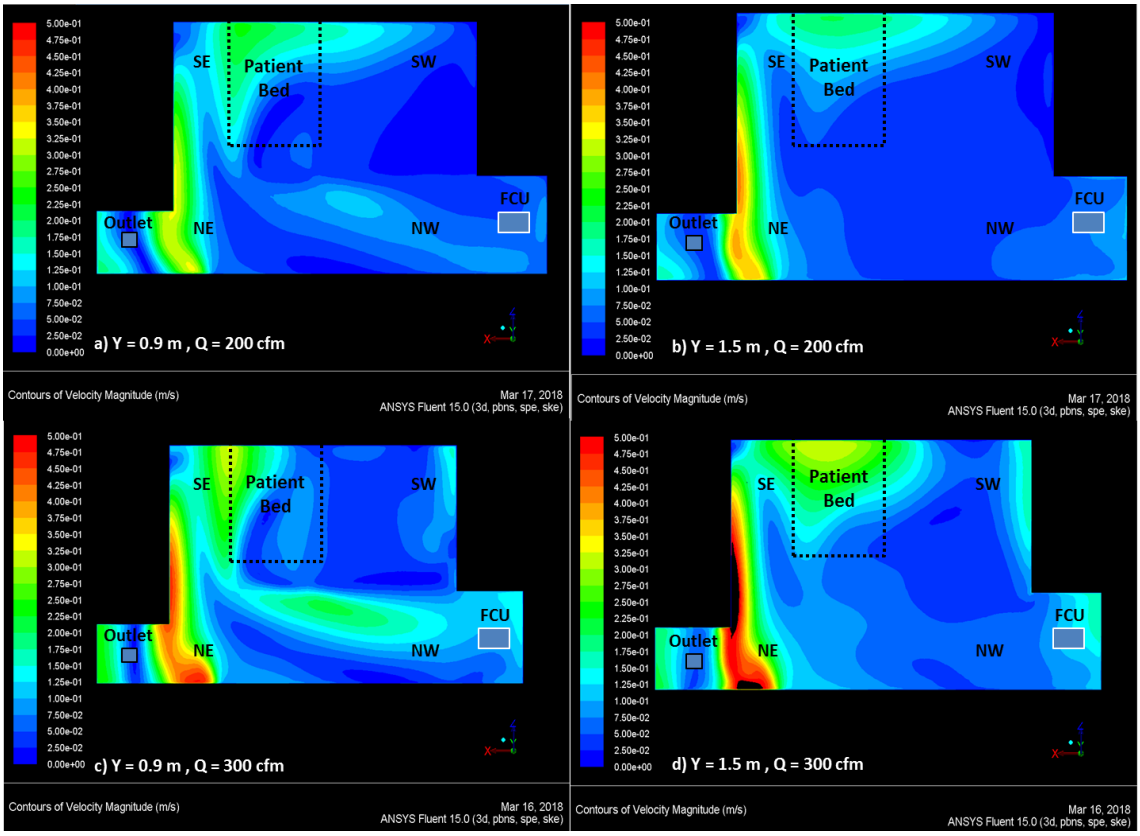

Figure 13. Contour plots of air velocity magnitude in patient room (top view) at different flows and planes, **a)** Y = 0.9 m and Q = 200cfm **b)** Y = 1.5 m and Q = 200 cfm **c)** Y = 0.9m and Q = 300 cfm and **d)** Y = 1.5 m and Q = 300 cfm. FCU = Fan coil unit includes an inlet, fresh air intake and a return unit. As shown in the CFD plots, the X-axis is oriented from NW/SW towards NE/SE region of the room, the Y-axis is pointing upwards from the floor towards the ceiling of the room and Z-axis is oriented from NW/NE towards SW/SE region of the room. Air samples were collected from the SE and NW quadrants of the patient room.

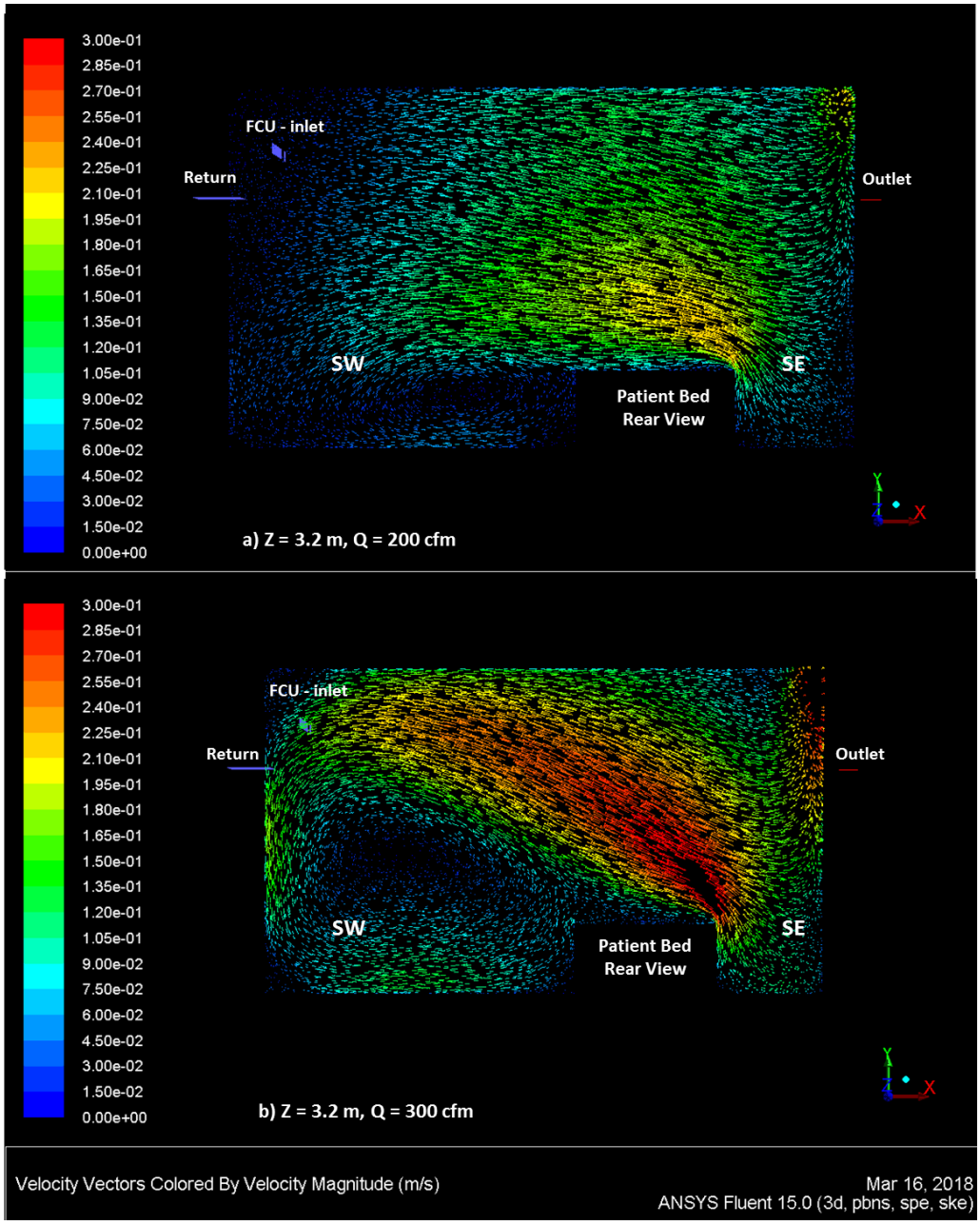

Figure 14. Air velocity vectors colored by velocity magnitude in patient room (rear view of patient room) displayed at the plane of the patient head (Z = 3.2 m) **a)** air flow *Q* = 200cfm and **b)** air flow *Q* = 300 cfm. The X-axis is oriented from NW/SW towards NE/SE region of the room, the Y-axis is pointing upwards from the floor towards the ceiling of the room and Z-axis is oriented from NW/NE towards SW/SE region of the room. Air sample was collected from the SE quadrant of the patient room.

Standards and guidelines (see Appendix C) specify temperature and humidity criteria in some hospital areas as a measure for infection control and thermal comfort [44,46,230]. Thermal comfort parameters such as temperature and humidity can either promote and prolong aerosolized viruses or inactivate viruses in healthcare buildings [44,191,231–233]. In this research study, the measured temperature and relative humidity inside patient rooms were within the ranges of comfort guidelines and thus were not significant predictors of virus aerosolization. As a variable parameter of thermal comfort conditions, indoor air movement can control or spread the infection in hospitals [35,63]. Therefore in such buildings, mechanical ventilation and air-handling units / systems should provide air movement patterns that minimize the spread of viral infection and contamination. The air velocities reached a maximum of 0.5 m/s in NE and SE quadrants of the patient room which is greater than threshold velocity of 0.1m/s needed to maintain an acceptable IAQ [44,46,168] . It is important to mention that in each sampling period, air samples were collected from the SE and NW quadrants of the patient room to minimize disturbance to the patients. Most importantly, air samples collected from mechanically ventilated patient rooms had no viral infections. However it is important to mention that the NE and SE regions of the patient room had higher velocities in both scenarios (Q = 200 and Q = 300 cfm) compared to NW and SW quadrants which could have enhanced aerosol transmission and air movement away from patient and into NW and SW zones of the room.

Evidently, the air movement was from the SE quadrant and towards the SW quadrant in both flow scenarios, however a sharper vertical slope was observed with Q = 300 cfm, thus carrying aerosols with influenza virus towards the SW quadrant of the room and at a higher elevation and above the breathing zone level. Thus, this further suggests that indoor occupants (HCPs and visitors) in the NW and SW quadrants might be at higher risk of influenza virus at Q =200 cfm as compared to high air flow scenario (i.e. Q = 300 cfm). Based on such findings, we recommend installing an additional outlet exhaust in the SW quadrant of the patient room to minimize risk and potential exposure. It is noteworthy to mention that all sampled patient rooms had similar HVAC design where the FCU unit is installed in the ceiling and above the main door. For such rooms, it would be safer for HCPs including nurses, doctors and medical employees to approach infected patients from the SE quadrant i.e. the right side of the patient's bed during routine care particularly when the mechanical ventilation system is on. Standard droplet precautions are also required such as lab-gowns, gloves and facemask and family members and visitors who spend considerable time with patients should abide also by the safety and droplet precautions and advised to stay more in the NE and SE zones of the room.

Considering our data, future studies should attempt, if feasible, to sample more locations within patient's room or at least includes the "hot zone" (i.e. the SW quadrant) to allow a more comprehensive and accurate risk assessment.

CHAPTER VII

AIR QUALITY MANAGEMENT IN HOSPITALS

A. Management Framework

Hospitals and healthcare facilities require a special and careful attention for ensuring a healthy air quality to protect indoor occupants and users including patients, healthcare workers (HCWs) and healthcare cleaners (HCCs) against chemical pollutants, hospital acquired infections (HAIs) and occupational diseases. Although infection control measures exist and are considered they are often inadequate because of concomitant construction problems and ingress of outdoor pollutants [52,118,234]. This is particularly true in metropolitan congested cities where hospitals located in urban environments are surrounded by industrial pollution and vehicular air emissions [52]. Reduced performance and increased symptoms of sick building syndrome (SBS) such as headache and fatigue have been widely associated with poor IAQ [40,49,172,235,236]. In this context, air quality management requires the consideration of a complex array of economic, technical, legal, infection control and political factors to ensure a healthy ambient and indoor environment (Figure 15). This management framework will be discussed in the following text.

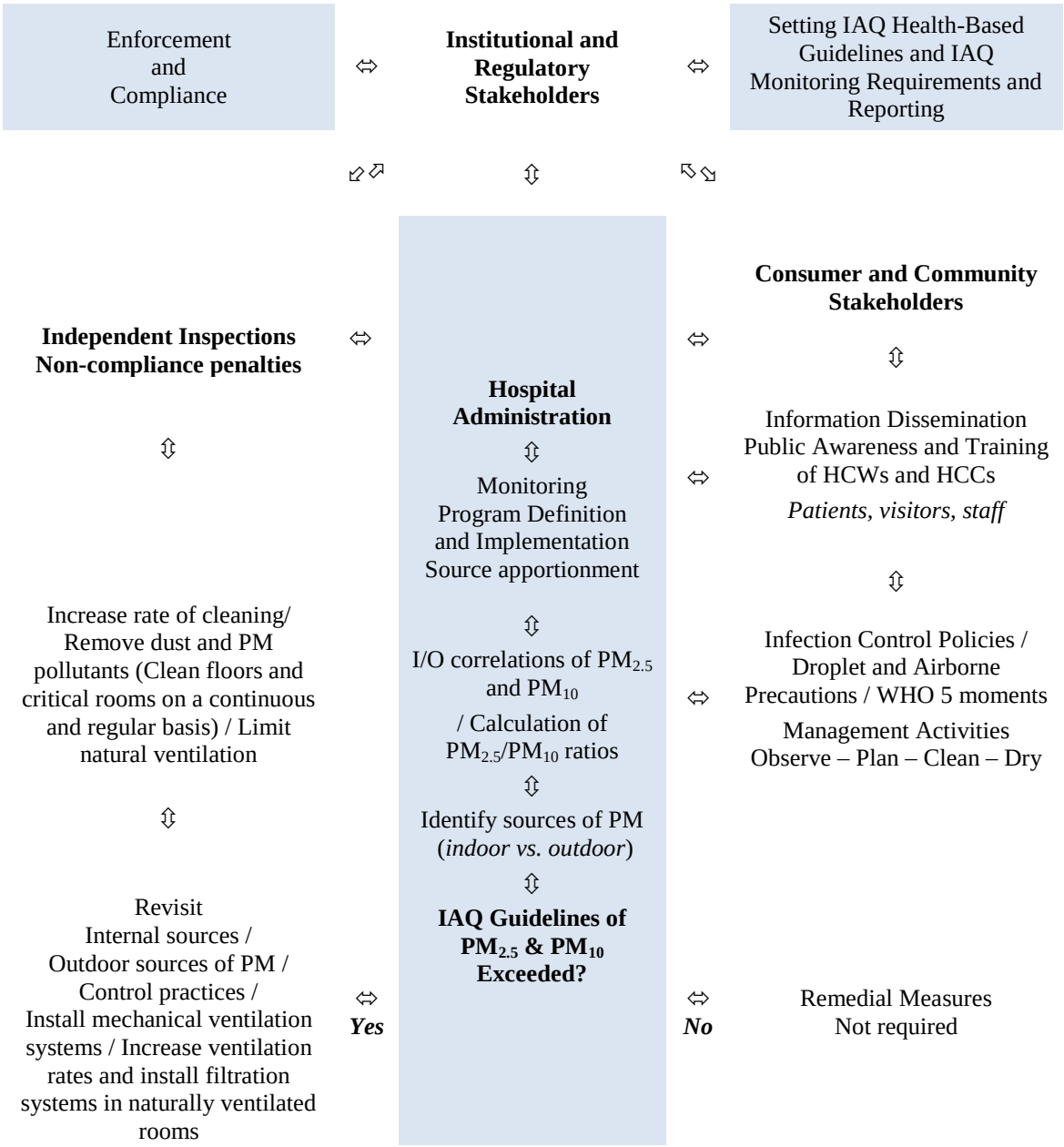

Figure 15. Air quality management framework

A hospital environment is naturally more sensitive to IAQ given the vulnerability of patients. In this study, both the clinic and reception working areas showed the highest exposure risk to PM_{10} and $PM_{2.5}$ particularly during dust storm

episodes of the warm season. Therefore the best approach to improve IAQ in hospitals is through three main steps: a) source control, b) dilution with outdoor air, and c) removal. Since outdoor air is mostly polluted, thus limiting and removing particles entering from the outdoor environment while also reducing indoor PM sources are crucial and necessary in enhancing the IAQ inside hospitals and healthcare facilities [7,9,35,82,111]. Properly designed and maintained central air conditioning and mechanical ventilation systems with adequate filtration are effective for this purpose [237–239]. While natural ventilation can improve air exchange rates, in polluted urban areas it has to be minimized to limit the exposure of patients, employees, and visitors alike to outdoor air pollution levels and traffic air emissions that often have significant levels of PM_{10} and $PM_{2.5}$.

Hospital-acquired infection represent a major public health concern worldwide, where pathogens in healthcare environments can be transmitted through airborne particles, fomites, respiratory droplets or direct contact with bodily fluids [52,174,175,228,240,241]. Despite the existence of the international infection control guidelines [175,242–244], an adequate level of basic infrastructure of such policies is still not available in many developing countires with low-income settings. In this study we detected Influenza A virus in half (~ 51 %) of the air samples collected inside patient rooms demonstrating the natural emission of influenza virus.Therefore preventing infection in healthcare settings requires serious and consistent application of infection control measures by healthcare workers (HCWs) and direct involvement of the infection control team. Maximizing coverage of seasonal influenza vaccine among vulnerable

groups and HCWs is necessary to limit the spread of infection by visitors or infected staff, as well as general education, training and raising awareness among medical staff is essential. In this chapter, we will discuss air quality management of hospitals and provide some infection control precautions and recommendations to improve IAQ and prevent nosocomial infections and transmission of diseases in hospitals and healthcare facilities.

B. Infection Control Precautions

Avoiding transmission of infections and diseases in healthcare settings can prevent considerable mortality, morbidity and healthcare costs. Infections are spread through one or more of three main routes: droplet, airborne and contact transmission. Droplets greater than 5 μm in size may be generated from the respiratory tract during coughing, sneezing or even talking [210,211,214,240,245]. If droplets from an infected person come into contact with the mucous membranes (i.e. mouth or nose) or surface of the eye of a recipient, they can transmit infection [210,211,214,245]. These droplets remain in the air for a short period and travel up to 1 to 2 m, so physical closeness is required for transmission [176,190,244]. As for airborne transmission, aerosol generating procedures (AGP) are considered to have a greater likelihood of producing aerosols compared to coughing for instance [176,190,244]. Aerosols are smaller than the droplets and can remain in the air for longer periods, hence potentially transmit infection by mucous membrane contact or inhalation [176,190,208,244]. Contact transmission may be direct where infectious agents can be inadvertently passed directly from an infected person to a recipient [176,246]. Indirect contact transmission takes

place when a recipient has contact with a contaminated object, such as furniture or object that an infected person may have coughed or sneezed on [176,246,247].

Infection is greatest in its early stages. For example, the infectious period for influenza virus is thought to be from 1 day before the onset of symptoms until 3−5 days later [29,176,192]. Children, elderly and seriously ill people may remain infectious for a longer period, and action should be considered to minimise prolonged shedding of influenza virus by patients with risk factors. Evidence shows that influenza viruses can be transferred from surfaces such as glass or plastic to hands up to 24 hours after contamination takes place [210,213–215,217]; also from materials and objects influenza viruses may be transferred for up to 2 hours [210,213–215,217]. For that reason hygiene and environmental cleaning are important and necessary in helping to control infection and spread of influenza and respiratory viruses. Therefore standard infection prevention control precautions are required from all HCWs for the care of all patients and patients' environments, to prevent cross-transmission from recognised and unrecognised sources of infection. Below is a description of precautions [118,173,234,241,248] that can be taken to reduce the risk of transmitting respiratory and viral infections.

1. Droplet Precautions

These precautions are designed to minimise transmission of respiratory pathogens and influenza virus from infected patients via droplets to susceptible persons.

- Place patients in a single room. In case single rooms are in short supply, ensure patients are at least 1 to 2 meters apart from each other and draw

privacy curtains or screens between beds to minimize close contact between them.

- Display signage to control entry into isolation areas.
- Limit the movement of patients outside their room to those necessary for patient management. If patient movement is needed then the patient should wear a surgical face mask to minimize the dispersal of viral and respiratory secretions and reduce environmental contamination.
- If the patient is unable to wear a mask for any reason, then HCWs transporting or accompanying the patient who will be required to come within 2 m of the patient should wear face masks and careful hand hygiene should be observed.
- Use a disposable single use tissue to cover mouth and nose when coughing, sneezing, wiping or blowing nose. Then dispose tissues promptly in bin.
- Practice hand hygiene by washing hands with soap and water, and drying them thoroughly after coughing, sneezing or using tissues
- HCWs caring for patients with a suspected or confirmed influenza or any respiratory virus are advised to wear a surgical face mask and gloves when in close contact with the patient (i.e. within 2 m). Eye protection is also advisable where there is a risk of eye exposure to infectious sprays.

2. Airborne Precautions

Airborne precautions are designed to prevent transmission of infectious agents via particles which remain suspended in the air [49,118,241]. The below procedures are considered likely to generate aerosols capable of transmitting respiratory pathogens when undertaken on patients:

- Intubation and related procedures such as manual ventilation and open suctioning.
- Cardiopulmonary resuscitation.
- Bronchoscopy, surgery and dental procedures.
- Obtaining diagnostic nose and throat swabs
- Administration of medication via nebulisation

Only HCWs who are needed to undertake the AGP procedures should be present inside patient rooms. Disposable lab gowns, gloves, eye protection and a respirator should be worn by those undertaking these procedures and whoever is present in the same room. Also the number of visitors should be limited where possible and they should be made aware of the risks and be offered personal protective equipment (PPE) as recommended for staff.

3. Contact Precautions

Contact precautions are considered to prevent transmission of infection by contact with the patient or the patient's environment [234,243,247–249]. Hand hygiene is the most effective way to prevent transmission by direct contact. As a minimum, hand

hygiene must be performed at the WHO Five Moments [243,249] which includes the following:

- Before touching a patient
- Before a clean/aseptic procedure
- After exposure to body fluids
- After touching a patient
- After touching the patient's surroundings

All staff should wear lab gowns and gloves and should change them and perform hand hygiene between contacts with patients even when they are in the same room. Medical equipment should as far as possible be allocated to each individual patient and where reusable equipment cannot be dedicated to individual patients, these must be cleaned immediately after patient use and between each patient check-up. In addition, environmental measures should include the following:

- Individuals considered potentially infectious should be kept away from public / communal areas in healthcare settings.
- Continous testing for viral persistence as it may be helpful to ascertain whether isolation needs to be continued for infected patients.
- Ensure that patients rooms with infection are cleaned daily, and are prioritised for frequently touched surface cleaning such as over-bed tables, lockers, lavatory surfaces in patient bathrooms, door knobs and equipment in the immediate vicinity of the patient at least three times a day and immediately if visibly contaminated.

- It is essential that all frequently touched surfaces and all surfaces inside patient rooms should be decontaminated and cleaned after any AGP procedure.
- Keep the patient environment clean and clutter free.
- Use disposable cleaning materials of low VOC concentrations
- Ensure proper mechanical ventilation and adequate air filtration rates inside patient rooms.

In this research study we detected influenza A virus in 51 % of the air samples collected inside patient rooms and demonstrated the natural emission of influenza virus via aerosols. Therefore the above guidelines and infection control policies are recommended to reduce the risk of transmission of influenza and other respiratory viruses inside healthcare facilities. Adherence to these guidelines is above all important in higher risk settings and critical environment such as hospitals. These precautions should be utilized in combination with vaccination of at risk patients and HCWs, where available and appropriate, and should be accompanied as well by serious and productive management activities in hospitals which will be discussed in the next section.

C. Management Activities

Although in the last decades, the indoor air has attained a growing attention, however the legislation and regulations inside critical indoor environments such as hospitals and healthcare facilities are still insufficient [52,174,248,250]. The air quality management framework that is presented here in this study is aimed primarily to provide some guidelines for cleaning hospital environments and offer a practical

approach to alleviating the risk of nosocomial diseases or HAIs to reduce the risk from environmental contamination and improve IAQ in the most vulnerable segment of the community. This management protocol or framework must comprise two major activities; cleaning, and inspection and maintenance of ventilation systems.

1. Cleaning Activities

While keeping hospitals clean was originally regarded as necessary, it is now recognized that frequent cleaning can remove dust and particulate matter, and reduce the bioburden in the healthcare environment and associated risk of healthcare-acquired infection [173,251,252]. Evidence supporting cleaning has accumulated over the past decade [173,234,248,253], along with infection control and prevention activities which were discussed before in previous section. Cleaning activities rely heavily upon available resources and managerial support as they vary considerably even within the same hospital and health districts [248,254]. Healthcare cleaners (HCCs) receive little or no training for what they do and thus are in need of systematic aid to good practice with in-built risk assessment for themselves, as well as for medical staff and patients. The four-steps [173,244] of cleaning activities should include the following tasks:

a. <u>Step 1 – Observing</u>

The first task in cleaning process consists of a visual or observatory assessment. Every HCC should first inspect and observe the areas to be cleaned, then consider overall conditions and degree of visual contamination before beginning his/her cleaning task. Patients tend to touch high-risk sites containing dust particles or infection

without any hand hygiene reminders. For that reason, cleaners or medical janitors should always evaluate the cleanliness of a room. The overall impression allows a cleaner to initiate cleaning or not, guided by hospital clinical or nursing staff if necessary. Provided cleaning access is timely and appropriate, the decision to clean will be followed by step 2 that requires the cleaner to plan ahead and adjust the area in preparation for the cleaning process.

b. Step 2 – Planning

All HCCs should first abide by the infection control policies that were discussed before in previous section. Washing their hands appropriately with liquid soap and water and then cleaned with hand hygiene products is essential [243,247,249]. The cleaner should then put gloves and lab gowns on, and/or other PPE barriers in accordance with the infection control policy of the hospital. Thus, blinds or curtains should be opened or lights switched on in order to visualize and locate the areas that need to be cleaned. Strong smells and high temperature may allow the HCCs to open a window, however thermal comfort balance between natural ventilation, smell and temperature should be assessed in line with patient and hospitals' ward staff comfort. Cleaning staff should not handle any clinical items, and should call on assistance from clinical staff where necessary. Near-patient surfaces offer the greatest risk for contamination so cleaning should include all areas near the patient such as the bedside lockers and tables as well as potential hot zones of nosocomial infection. Working areas such as clinics, lobbies and waiting areas as well as offices of medical staff should also be readily cleaned. Once the room or bed space has been organized for access, bins

should be checked and emptied and visible rubbish on the floor and other surfaces must be removed. Final preparations include replacement of rubbish bags, soap, paper towels and rest room paper. Dirty linen and towels should be also removed and placed in appropriate containers. Cleaning fluids and detergents should be freshly prepared from in-use supplies with careful attention towards expiry date for chosen consumables to avoid any emission of toxic VOCs. Moreover medical equipment and printing machines inside hospital wards should always be clean and in a good state of repair. Lastly, sufficient clean water and fresh wipes should be readily available for HCCs with clear instructions on how to manage disposable and non-disposable items.

c. Step 3 – Cleaning

This task removes both dirt or dust particles and microorganisms from surfaces, floors and windows, thus reducing the amount of particulate matter and bioburden in hospitals. Cleaning should always precede disinfection because the presence of dust particles and dirt will hinder disinfectant activity as studies have shown before [255,256]. Some hospitals use detergents for routine cleaning while others choose products that either inactivate or kill living microorganisms [251,255,256]. Below are general principles generally accepted as good practice [173,252,257,258]:

- HCCs should have uni-directional flow in their cleaning process to avoid cross-contamination. Cleaners should start at the furthest end of the bed space working towards the exit and should clean from high to low not starting with the floor. The sites closest to patients should be cleaned first and priority should be for hand-touching sites such as bed

rails, bed control if electric bed and nurse call bell as these represent major zones for infection.

- Different wipes should be used for different hospital areas or wards and patient rooms. The wipes should be flat and unfolded to maximize the area cleaned and minimize the amount of hand contact. Most importantly cleaners should wipe in one direction without retracing area already cleaned and should wipe a large flat surface using an *S*-shaped pattern to avoid cross-contamination [259,260]. Finally after cleaning is over in each site, all used wipes should be disposed to eliminate the transfer of microbes.
- Detergents should be used only to remove dust and particulate matter while disinfectants are then used for killing microbes. Hence usage of detergents should always precede the disinfectants use [173,259,261].
- Cleaning fluids should be prepared, applied and discarded according to manufacturers' guidance and in adherence with hospital infection control policy.
- Ensure bathrooms are cleaned after the patient room beginning with the sink, then the shower or bath and finally the toilet. As with the near-patient environment, prioritise the hand-touch sites in the bathroom.
- Finally floor cleaning is the last task to complete.

d. Step 4 – Drying

The final task is drying by using clean paper towels or cloths. Drying of cleaning fluids such as detergents and disinfectants on surfaces and floors is also essential since the cleaning process is not complete until all surfaces are completely dry. Once the surfaces and floors are dried, then furniture (which should be mostly leather-based so they don't to act as sink for particles and other gaseous pollutants) and other objects can be repositioned, doors and windows adjusted, and signage removed [35,217,226,248]. Patient belongings should also be returned to the top of the locker or bed table, with the crowded site wiped over and similarly allowed to dry. When cleaners leave the patient area, they should apply hand disinfection if the cleaner has to fulfill further duties. In addition gloves and any other protective apparel or PPE barrier may be removed and hands washed and dried. As a final consideration cleaning activities should be done regularly and accurately, and any problems with cleaning should be reported to clinical staff in general and cleaning supervisors in particular.

2. Inspection and Maintenance of Ventilation Systems

Ventilation systems in buildings play a major role in providing adequate physical conditions and perceived air quality to indoor occupants and users through fresh air supply, heat removal, and pollutant dilutions [35,228,250]. In healthcare facilities, ventilation systems should prevent cross infection risks, harmful emissions, and spreading of pathogens and nosocomial diseases. A good design of the ventilation system can decrease the infection risks as studies have shown [262–264].

Thermohygrometric and physical parameters of healthcare facilities are defined by specific ASHRAE standards [59,61,169,250,265] that are presented below:

- Temperature in hospital wards should be between 21 and 24 ^{o}C (see Appendix C).
- Relative humidity in patient rooms, collective rooms and in hallways or corridors should have a range between 40 and 60% (see Appendix C).
- Air change be able to achieve and maintain the air quality and values should be 2AC/h for outdoor (air change rate minimum) and totally 6AC/h and can reach up to 12AC/h [59,61,169,250,265]. Air velocities should vary a minimum of 0.05 m/s and can reach up to 0.30m/s [59,61,169,250,265] to ensure pathogens-free environment. The outdoor air flow rates for hospitals, clinics, nursing areas range between 8.5×10^{-3} m^3/s and 11×10^{-3} m^3/s per person [59,61,169,250,265].
- On the outdoor side, the correct location of the outdoor air intakes and exhaust outlets of ventilation (HVAC) systems should include specific precautions since IAQ depends mostly on the supply [59,61,169,250,265]. Outdoor intakes should be localized not less than 9 m from cooling towers, ventilation exhaust outlets from hospital or adjacent buildings, combustion equipment stack exhaust outlets, plumbing vent stacks and medical-surgical vacuum systems, and other districts of hospitals that may collect vehicular exhaust and other toxic fumes [59,61,169,250,265].

- HCWs, particularly the maintenance team should change filtration systems on a regular basis to make sure the supply air is free of contaminants once inside the healthcare facilities.
- Finally proper and efficient ventilation combined with low-emission building materials can be key factors for an adequate IAQ and control of infections spread by air.

In closure, we recommend that hospital managers and health-care professionals or practitioners (HCPs) attempt to qualitatively and quantitatively evaluate indoor air of hospitals on a periodic basis, limit natural ventilation, use mechanical ventilation and air purification equipment in hospitals during design, maintenance and construction phases. Finally embedding such a systematic cleaning protocol in high performance buildings such as hospitals and applying the tasks presented above by medical employees (including HCWs and HCCs) and patients would practically help in improving IAQ and reducing HAIs.

CHAPTER VIII

CONCLUSIONS AND FUTURE WORK

In this research study, we examined seasonal variations in IAQ indicators (CO, CO_2, $PM_{2.5}$, PM_{10} and TVOC) in three hospitals with an emphasis on I/O correlations, different ventilation modes and associated potential exposure. The most significant indicators were $PM_{2.5}$ and PM_{10} with measured levels exceeding health standards by 2 to 3.5-folds, particularly during dust storm episodes of the warm season with an increase in $PM_{2.5}/PM_{10}$ ratios, suggesting direct association with the outdoor air and the abundance of finer particles ($PM_{2.5}$) in most sampled areas. The ingress of fine and coarse particles from outdoor into indoor environments of several areas was evident during both the cold and warm seasons with high correlations between indoor and outdoor $PM_{2.5}$ (r = 0.83 to 0.92) and PM_{10} (r = 0.74 to 0.86) recorded at several locations during the latter season. CO levels indoors were lower than those recorded outdoors and both remained within ranges below air quality standards and guidelines. In contrast, indoor concentrations of CO_2 and TVOCs exceeded outdoor levels during the warm and cold seasons with I/O ratios > 1 at all of the sampling locations. The application of various air quality indices revealed that hospital occupants, particularly sensitive individuals like elderly people and children, were at higher $PM_{2.5}$ exposure risk in some areas such as the clinic and reception working areas. While the indices concurred in classifying certain areas similarly, they differed in the classification of other areas. However the most robust index (RIAQI) was obtained when coupling IAQ

indicators with the concomitant effect of thermal comfort. This index can be used as a risk assessment tool towards safer conditions in hospitals and healthcare environments and can be extended to assess indoor residential and working environments such as apartments, schools and offices. Finally, this study has demonstrated the natural emission of the influenza virus via airborne route where 26 air samples from 17 patient rooms tested positive for influenza A virus. The day of hospital admission was significantly associated with virus detection in the air sample, with the majority of cases being detected from patient rooms one day after admission. In addition, two transmission "hot zones" were identified by CFD model inside patient rooms where indoor occupants are at a higher risk of viral infection.

The results of this study called for the adoption of remedial control measures and air quality management that aimed to improve the removal of small size PM from indoor air and reduce HAIs in the most critical segment of the community. This would require encouraging the use of mechanical ventilation instead of natural ventilation particularly in urban regions, while also upgrading the existing filtering capabilities of mechanical ventilation systems to increase their efficiency in terms of filtering PM and securing a healthy and safe indoor environment free of microbes. In the absence of effective interventions and management programs, IAQ is likely to remain a critical health risk particularly in developing countries. Understanding how indoor exposure relates to outdoor concentrations in critical environments is imperative for the assessment of policy intervention and decision making towards alleviating potential adverse health impacts for a most vulnerable segment of a community. Future work

should implement a longer term sampling and monitoring program to better assess exposure and health risk of air contaminants. Moreover, more work has to be completed assessing the composition of PM including organic and elemental analysis to identify potential sources of indoor $PM_{2.5}$ and PM_{10} with an emphasis on bioaerosols and biological burden of hospitals particularly bacteria, fungi and molds. Such studies can be helpful in risk assessments to further develop control measures towards improving IAQ in hospitals and protecting patients, medical employees, and visitors alike.

APPENDICES

Appendix A. Origin and health impacts of air pollutants

Pollutant	*Origin*	*Impact*	*Reference*
PM_{10}	Coarse particles are produced by mechanical processes such as erosion, grinding, coagulation (two or more small particles combine) or condensing (gas molecules condense into a solid particle). They are also rich in crustal matter such as Ca, Mg, Fe, Si that result from re-suspension of soils and surface dusts. They are also rich in sea salt (NaCl) and other naturally occurring earth constituents that deposit very close to their source area	PM_{10} can cause cardiovascular diseases, and has severe impacts on human respiratory system since when inhalation of coarse particles ($PM_{10-2.5}$) occur, they reach the upper parts of the lungs, pharynx and trachea. PM_{10} can also cause severe ecological effects and can lead to serious degradation of terrestrial and aquatic ecosystems through the process of scavenging and acid rain.	[1,5,111,142]
$PM_{2.5}$	Fine particles ($PM_{2.5}$) which is less than 2.5 µm in diameter can stay in the atmosphere for days or weeks. They are mainly formed from chemical reactions, such as condensation of low volatile organics. They also contain more soluble inorganic components such as sulfate (SO_4^{2-}) and ammonium (NH_4^+). Eddy diffusion and advection are the two major transport phenomena for fine particles. Moreover due to their small size, fine particles are moved by incorporation into cloud droplets then released by wet deposition and rain out.	Fine particulate matter ($PM_{2.5}$) is considered more dangerous and penetrates deeply into the bronchi and alveoli regions of the lungs provoking lung cancer and respiratory diseases.	[1,5,111,142]
CO	Carbon monoxide (CO) is an odorless and colorless gas that is generated by incomplete burning of carbon-based fuels and from tobacco smoke.	CO is dangerous to human health as it reduces the blood's ability to carry oxygen. It has an affinity for the oxygen carrying sites on the hemoglobin in the blood of 210 times greater than oxygen. As it displaces the oxygen, carbon monoxide prevents the distribution of the needed oxygen where the tissues become oxygen-deprived. Initial symptoms include shortness of breath on mild exertion, mild headaches, listlessness, and nausea (Lee et al. 2015; Stewart, 1975; Proctor and Hughes, 1978). As exposures increase, the individual may experience severe headaches, mental confusion, dizziness, nausea, rapid breathing, and fainting on mild exertion. Moreover, in pregnancy, the fetus may be susceptible to the effects of CO, suffering serious and even permanent damage to the central nervous system. In extreme cases, when CO concentrations reach 800 ppm and above, exposure	[1,5,111,116,142]

Pollutant	*Origin*	*Impact*	*Reference*
		may result in unconsciousness and death.	
CO_2	Carbon dioxide (CO_2) is also an odorless and colorless product of carbon combustion. Common indoor sources may also be human metabolism and breathing, gas cooking appliances, space heaters, wood-burning appliances, and tobacco smoke Other sources of carbon dioxide are combustion by-products such as automotive traffic, compressed carbon dioxide (e.g. fire extinguishers), dry ice, and aerosol propellants.	Exposure to high CO_2 levels i.e. > 1000 ppm is an important risk factor of sick building syndrome (SBS)	[1,5,16,111,142]
SO_2	SO_2 is generated in the atmosphere from coal-fired power plants, anthropogenic, mineral and volcanic sources. In addition, the oxidation of sulfur compounds such as dimethyl sulfide (DMS) and hydrogen sulfide (H_2S) that are emitted from the ocean and industrial sources respectively are a major source of SO_2 in the troposphere	Being a precursor for sulfuric acid, SO_2 contributes to acid rain and aerosol formation in the troposphere. It is also very corrosive and toxic and at high concentrations can cause life threatening pulmonary edema where coughing, shortness of breath, difficulty in breathing and tightness in the chest are mainly experienced.	[1,5,17,111,142]
NO/NO_2	Vehicular, industrial and combustion emissions are considered the major sources of nitrogen oxides and particularly NO_2. The heterogeneous reactions of nitrogen oxides have dramatic implications on the production of ozone in the troposphere (Finlayson Pitts and Pitts, 2000). Nitrogen oxides (NO_x) resulting from combustion processes of biomass and fossil fuel burning undergo heterogeneous reactions yielding particulate nitrate (NO_3), nitrous (HONO) and nitric acid (HNO_3). The reaction of NO_2 and O_3 would result in nitrate formation, while that of NO_2 and OH radicals yields nitric acid (HNO_3) in the atmosphere	NO_2 is certainly a dangerous pollutant and a component of acid rain. This gas has many undesirable health effects on humans, including asthma and other cardiovascular and respiratory diseases resulting in swelling of oxygen pathways in the human body.	[1,5,111,142,266]
VOC	Volatile organic compounds (VOCs) vaporize at room temperature and include all organic compounds with up to seventeen carbons in their	VOCs cause adverse health effects and Some of which are toxic, mutagenic and carcinogenic. The symptoms of VOCs may cause slight irritation	[1,5,18,111,142]

Pollutant	*Origin*	*Impact*	*Reference*
	molecular structure that have a boiling point up to 250°C. VOCs are released from many housekeeping and maintenance products, building materials, industrial emissions, furnishings, equipment, pesticides and insecticides. Food manufacturing and industrial operations have been known to generate organic chemicals that include VOCs.	including headache, nausea, and irritation of the eyes, nose, and throat. High toxicity levels eventually may lead to death.	

Appendix B. Non-culturable microorganisms of bioaerosols and their health impacts

Microorganism	*Description*	*Health-impact*	*References*
Archaea	Archaea are methanogens microorganisms constituting one of the three domains along with Eukarya and Bacteria. Archaea have never been cultured from air samples and are prokaryotes that share similar characteristics in their morphology and metabolism to bacteria. However different to bacteria, archaea have unique membrane lipids, a cell wall devoid of peptidoglycan, intrinsic capacity to resist antibiotics, and different ribosomal 16S DNA.	Archaea are resilient microorganisms that live in extreme environments and are mainly found in complex microbial communities such as the gut, and in swine and cow manure. Airborne archaea are also detected in high concentrations in bioaerosols of agricultural settings and wastewater treatment plants. Archaea can induce chronic inflammation in the lungs and lead to sensitization diseases.	[20,267]
Mycobacteria	Non-tuberculous mycobacteria (NTM) are mainly found in water related sources, soil and metal-working fluids. Naturally occurring Mycobacterium tuberculosis in aerosols can be viable but may not be culturable. Several studies have characterized airborne mycobacteria in indoor settings including hot tubs, therapy pools, dental clinics as well as hospitals, using molecular and culture-independent methods.	NTM are microorganisms that can cause lung diseases whether or not they are infectious. The mycobacterial cell components are known to cause inflammation in the lungs and it is known that exposure to airborne bioaerosols containing NTM can lead to hypersensitivity pneumonitis (HP). Moreover several species of NTM can induce inflammatory responses in mouse macrophage cells.	[32,165,267]
Gram-positive bacteria	Gram positive bacteria are anaerobes that are not often cultured. Molecular methods have revealed that fecal anaerobes are major constituents of bioaerosols in various environments such as agricultural settings.	They have potential impact on respiratory tract.	[268–270]
Viruses	Given their complex structures, viruses act as bioaerosol agents that are difficult to culture. Airborne viruses are markedly absent in culture media where there is no universal assay for viruses. However some studies have shown that there are some universal Polymerase Chain Reaction (PCR) assays for group of viruses that can amplify RNA. Other assays include aminidase assay as a potential broader marker for the presence of certain viruses.	Examples of airborne transmitted viral diseases are influenza A and B (flu), coronaviruss (common cold and severe acute respiratory syndrome), adenovirus (lung infections and common cold), norovirus (gastrointestinal illenesses), and morbillivirus (measles and mumps). Other viruses can also induce inflammatory response when present in an inactivated or non-replicating form (Respiratory Syncytial Virus – RSV).	[201,271–273]

Appendix C. ASHRAE recommendations and design criteria of working rooms in healthcare facilities

Unit	*Working Area*	*Pressure related to adjacent areas*	*Min air changes of outdoor air per hour (ACH)*	*Min total air changes per hour (ACH)*	*All air exhausted directly to outdoors*	*Air recirculated within room units*	*Relative humidity (RH - %)*	*Designed temperature (T – ^{o} C)*
SURGICAL & CRITICAL CARE	Operating room	Positive	5	25	---	No	30 – 60	20 – 23.9
	Surgical cystoscopy room	Positive	5	25	---	No	30 – 60	20 – 23.9
	Delivery room	Positive	5	25	---	No	30 – 60	20 – 23.9
	Recovery room	---	2	6	---	No	30 – 60	21.1 – 23.9
	Intensive care unit	---	2	6	---	No	30 – 60	21.1 – 23.9
	Newborn intensive care unit	---	2	6	---	No	30 – 60	22.2 – 25.6
	Treatment room	---	---	6	---	---	30 – 60	21.1 – 23.9
	Nursery suite	Positive	5	12	---	No	30 – 60	23.9 – 26.7
	Trauma room (crisis or shock)	---	3	15	---	No	30 – 60	21.1 – 23.9
	Trauma room (treatment)	Positive	2	6	---	No	30 – 60	21.1 – 23.9
	Anesthesia gas storage	Negative	---	8	Yes	---	---	---
	Endoscopy	Negative	2	6	---	No	30 – 60	20 – 22.8
	Bronchoscopy	Negative	2	12	Yes	No	30 – 60	20 – 22.8
	ER waiting areas	Negative	2	12	Yes	---	30 – 60	21.1 – 23.9
	Triage	Negative	2	12	Yes	---	---	21.1 – 23.9
	Radiology waiting rooms	Negative	2	12	Yes	---	---	21.1 – 23.9

	Class (A) operating room	Negative	3	15	---	No	30 – 60	21.1 – 23.9
NURSING	Patient room	---	2	6	---	---	30 – 60	21.1 – 23.9
	Toilet room	Negative	Optional	10	Yes	No	---	---
	Newborn nursery suite	---	2	6	---	No	30 – 60	22.2 – 25.6
	Protective environment room	Positive	2	12	---	No	---	21.1 – 23.9
	Airborne infection isolation room	Negative	2	12	Yes	No	---	21.1 – 23.9
	Isolation alcove or anteroom	Positive	2	10	Yes	No	---	---
	Labor/Delivery/recovery/postpartum (LDRP)	---	2	6	---	---	30 – 60	21.1 – 23.9
	Public corridor	Negative	2	2	---	---	---	---
	Patient corridor	---	2	4	---	---	---	---
	Admitting and waiting rooms	Negative	2	6	Yes	---	30 – 60	21.1 – 23.9
ANCILLARY	Laboratory, general	Negative	2	6	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, bacteriology	Negative	2	6	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, biochemistry	Positive	2	6	---	No	30 – 60	21.1 – 23.9
	Laboratory, cytology	Negative	2	6	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, glass washing	Negative	Optional	10	Yes	---	---	---
	Laboratory, histology	Negative	2	6	Yes	No	30 – 60	21.1 – 23.9
	Microbiology	Negative	---	6	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, nuclear medicine	Negative	2	6	Yes	No	30 – 60	21.1 – 23.9

	Laboratory, pathology	Negative	2	6	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, serology	Positive	2	6	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, sterilizing	Negative	Optional	10	Yes	No	30 – 60	21.1 – 23.9
	Laboratory, media transfer	Positive	2	4	---	No	30 – 60	21.1 – 23.9
	Autopsy room	Negative	2	12	Yes	No	---	---
	Non-refrigerated body holding room	Negative	Optional	10	Yes	No	---	21.1
	Pharmacy	Positive	2	4	---	---	30 – 60	21.1 – 23.9
	Radiology (X-ray)	Positive	3	15	---	No	30 – 60	21.1 – 23.9
DAIGNOSTIC & TREATMENT	Examination room	---	2	6	---	---	30 – 60	21.1 – 23.9
	Medication room	Positive	2	4	---	---	30 – 60	21.1 – 23.9
	Treatment room	---	2	6	---	---	30 – 60	21.1 – 23.9
	Physical therapy or hydrotherapy	Negative	2	6	---	---	30 – 60	22.2 – 26.7
	Soiled workroom	Negative	2	10	Yes	No	30 – 60	22.2 – 25.6
	Clean workroom	Positive	2	4	---	---	---	---
SERVICE	Food Preparation Center	---	2	10	Yes	No	---	---.
	Ware washing	Negative	Optional	10	Yes	No	---	---
	Dietary day storage	---	Optional	2	---	No	---	---

	Laundry, general	Negative	2	10	Yes	No	---	---
	Bathroom	Negative	Optional	10	Yes	No	---	22.2 – 25.6
	Janitor's room	Negative	Optional	10	Yes	No	---	---

References: [59–61,69,169,265]

(a) Where continuous directional control is not required, variations should be minimized, and in no case should a lack of directional control allow the spread of infection from one area to another. Boundaries between functional areas should have directional control. Design of the ventilation system shall provide air movement, which is generally from clean to less clean areas.
(b) The ventilation rates cover the ventilation for comfort, as well as for asepsis and odor control in areas of acute care hospitals that directly affect patient care.
(c) Total air changes indicated should be either supplied or, where required, exhausted. Number of air changes can be reduced when the rooms are unoccupied if pressure relationship is maintained and the number of air changes indicated is reestablished any time the space is being utilized. The air changes shown are minimum values, and higher values should be used when required to maintain room temperature and humidity conditions based on the cooling load of the space such as lights, equipment people exterior walls and windows.
(d) Recirculating HEPA filters used for infection control are acceptable. Gravity type heating or cooling units such as radiators or convectors shall not be used in operating rooms and other special care areas.
(e) For operating rooms, 100% outside air should be used only when codes require it and only if heat recovery devices are used.
(f) The term "trauma room" is a first aid room and/or emergency room used for general initial treatment of accident victims. The operating room within the trauma center that is routinely used for emergency surgery should be treated as an operating room.
(g) Refer to *ASHRAE Handbook – HVAC Applications* [230,265]for a discussion of design of toilet exhaust systems.
(h) The airborne infectious isolation rooms are those that might be used for infectious patients in the average community hospital. The rooms are negatively pressurized and some isolation rooms may have a separate anteroom.
(i) Protective environment rooms are used for immunosuppressed patients. Such rooms are positively pressurized to protect the patients. Anterooms are generally required and should be negatively pressurized with respect to the patient room.
(j) All air need not be exhausted if darkroom equipment has scavenging exhaust duct attached and meets ventilation standards regarding NIOSH, OSHA and local employee exposure limits.
(k) A non-refrigerated body-holding room is not applicable to facilities that do not perform autopsies on-site and use the space for short periods while waiting for the body to be transferred.
(l) Food preparation centers in hospitals should have an excess of air supply for positive pressurization when hoods are not in operation. The number of air changes may be reduced or varied for odor control when the space is not in use. Minimum total air changes per hour should be that required to provide proper makeup air to kitchen exhaust systems.
(m) Areas with contamination or odor problems shall be exhausted to the outside and not recirculated to other areas. Individual circumstances may require special consideration for air exhaust to the outside. Intensive care units in which patients with pulmonary infection are treated and rooms for burn patients are examples. To satisfy exhaust needs, replacement air from the outside is necessary. Minimum outside air quantities should remain constant while the system is in operation.
(n) The relative humidity ranges are the minimum and maximum limits where control is specifically needed. These limits are not intended to be independent of a space temperature. For example, the relative humidity is expected to be at higher end of the range when the temperature is also at the higher end, and vice versa. Some rooms may have a lower limit of the design relative humidity of 20% RH, and these rooms are considered "short-term stay" rooms and is evident that this is where the patient will not stay for long periods of time, and the exposure to relative humidity as low as 20% will have negligible effect on patient's care and well-being.

(o) For indicated temperature ranges the systems shall be capable of maintaining the rooms at any point within the range during the normal operation. Use of lower temperature is acceptable when patient's comfort and medical conditions require those conditions.

(p) National Institute for Occupational safety and Health (NIOSH) Criteria Documents regarding "Occupational Exposure to Waste Anesthetic Gases and Vapors" [274] and "Control of Occupational Exposure to Nitrous Oxide" [274] indicate a need for both local exhaust scavenging systems and general ventilation of the areas in which the respective gases are utilized.

(q) Differential pressure between space and corridors shall be a minimum of 0.01 inch water gauge (2.5Pa). If monitoring device alarms are installed, allowances shall be made to prevent nuisance alarms

(r) Since some surgical procedures may require room temperatures that are outside the indicated range, hence operating room design conditions should be developed in consort with all users, surgeons, anesthesiologists and nursing staff. The required total air change rates are also a function of space temperature set-point, supply air temperature, sensible and latent load in the space.

(s) The first air room or emergency room used for initial treatment of accident victims can be ventilated as noted for the "treatment room". Treatment rooms used for bronchoscopy shall be treated as bronchoscopy rooms.

(t) In a recirculating, ventilation systems, HEPA filters can be used in lieu of exhausting the air from these spaces to the outside. In this case the return air shall be passed through HEPA filters before it is introduced into any other spaces.

(u) If exhausting the air from an airborne infection isolation room to the outside is not practical, then the air may be returned through HEPA filters to an air-handling system exclusively serving the isolation room.

(v) Total air changes per room for patient rooms and labor/delivery/recovery/postpartum rooms may be reduced to 4 when supplemental heating and cooling systems are used.

(w) The protective environment airflow design specifications protect patients from common environmental airborne infectious microbes such as *Aspergillus spores*. These special ventilation areas shall be designed to provide directed airflow from the cleanest patient area to less clean areas. These rooms shall be protected with HEPA filters at 99.70 % efficiency for 0.3 micron-sized particles in the supply airstream. Such interrupting filters protect patient rooms from maintenance-derived release of environmental microbes from the ventilation system components. Recirculation HEPA filters can be used to increase the equivalent room air exchanges. Constant volume airflow is required for consistent ventilation for the protected environment.

(x) The infectious disease isolation room described in these guidelines is to be used for isolating the airborne spread of infectious diseases, such as measles, varicella or tuberculosis. The design of airborne infection isolation (AII) rooms should include the provision for normal patient care during periods not requiring isolation precautions. Air may be circulated within individual isolation rooms if HEPA filters are used.

(y) When required appropriate hoods and exhaust devices are installed for the removal of noxious gases or chemical vapors.

(z) A simple visual method such as smoke trail, ball in tube, or flutterstrip can be used for verification of airflow direction. These devices will require a minimum differential air pressure to indicate airflow direction. In accordance with AIA 2001 guidelines [275], recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health care workers are likely to work, across the infectious source, and then to exhaust, so that the health care worker is not positioned between the infectious source and the exhaust location. Furthermore, the design of such systems should allow for easy access for scheduled preventative maintenance and cleaning.

Appendix D. Equipment Specifications for monitoring program implementation

Instrument	*Manufacturer*	*Parameter*	*Detection method*	*Specifications*
TSI Dustrak II Model 8532 [a] 	Model 8532, TSI Corporation, Shoreview, USA	PM_{10} & $PM_{2.5}$	90° light backscattering PM_{10} & $PM_{2.5}$ impactors	**Range**: 0.001 to 150 mg/m^3 **Resolution**: ±0.1% of reading or 0.001 mg/m^3
PhoCheck Tiger PID VOC [a] 	Ion Science Ltd, The Way, Fowlmere, UK	VOC	Photo ionization detection (PID)	**Range**: 1ppb – 20000 ppm **Response time**: 2s **Accuracy**: ± 5% display reading ± one digit Battery life up to 30 hrs
E4500 Portable Emissions Analyzer [a] 	E Instruments International, LLC 402 Middletown Blvd. Suite 216 Langhorne, PA 19047	O_2, CO, NO, NO_2, SO_2, C_xH_y (Hydrocarbons)	Electrochemical sensor detection	**Up to Four Gas Sensors**: O_2, CO, NO, NO_2, SO_2, C_xH_y Dilution Pump For CO **Auto-Range Measurements** Up to 50,000 ppm **Low NO_x Capable** with 0.1ppm resolution & high accuracy Gas Sensors are Pre-Calibrated & Field Replaceable **Full Color Graphic Display** Screen User Customized Display Screen & Print-Out Content **Automatic Data Saving** Efficiency, Excess Air, & CO_2 Calculations
Air Velocity Meters TSI 5725 & 9535& 9545	TSI Corporation 500 Cardigan	Air velocity (v), air flow (Q),	Anemometer	**Range:**0 to 4,000 ft/min (0 to 20 m/s) and 0 to 6,000 ft/min (0 to 30 m/s)

Instrument	*Manufacturer*	*Parameter*	*Detection method*	*Specifications*
	Road Shoreview, MN 55126	pressure (P), volume (V), relative humidity (RH), and temperature (T)		**Accuracy**: ±5% of reading or ±5 ft/min (±0.025 m/s), whichever is greater and ±3% of reading or ±3 ft/min (±0.015 m/s), whichever is greater **Resolution**: 1 ft/min (0.01 m/s)
TSI ACCUBALANCE® Air Capture Hood Model 8380 	TSI Corporation 500 Cardigan Road Shoreview, MN 55126	Air velocity (v), air flow (Q), pressure (P), volume (V), relative humidity (RH), and temperature (T)	Air capture hood, Flow conditioner micro-manometer	**Velocity:** 0.125 – 78 m/s ; Accuracy ±3% of reading ±7 ft/min (±0.04 m/s) at velocities >50 ft/min (>0.25 m/s) Resolution 1 ft/min (0.01 m/s) **Pressure:** Differential ±15 in. H_2O (±3735 Pa); 150 in. H_2O (37.5 kPa), Accuracy ±2% of reading ±0.0001 in.; Resolution: 0.00001 in. H_2O (0.001 Pa) static and differential; 0.01 in. Hg (1 mm Hg) absolute **Volume:** Range 25 to 2,500 ft^3/min (42 to 4250 m^3/h) ; **Accuracy** ±3% of reading ±7 ft^3/min (±12 m^3/h), **Resolution** 1 ft^3/min (1 m^3/h) **RH:** Range 5 to 95% RH temperature/RH probe; Accuracy ±3% RH; Resolution 0.1% RH **Temperature:** Sensor in base 40 to 140°F (4.4 to 60°C) **Accuracy**: ±0.5°F (±0.3°C) **Resolution:** 0.1°F (0.1°C)
Langan L76x [a, b]	2660 California Street, San Francisco, CA 94115 USA	CO	Electrochemical 3-electrode	**Range**: 0-200 ppm **Resolution**: 50 ppb **Response time t_{90}**: < 30 s at 20°C **Repeatability**: 1% of signal
		CO_2	Dual Beam Absorption Infrared	**Range**: 0-10000 ppm **Resolution**: 1 ppm **Response time** t90: < 60 s

Instrument	*Manufacturer*	*Parameter*	*Detection method*	*Specifications*
				**Accuracy**: ± 50 ppm or 5% of reading **Repeatability**: ± 20 ppm
		Temperature	Thermocouple	**Range**: -40 to 80°C **Resolution**: 0.1 °C Calibrated to 0.1 degrees Celsius
		Relative humidity	Ceramic based	**Range**: 5-95% **Resolution**: 0.1%; Calibrated to ± 2% over a 5-95% range
Coriolis µ Biological Air Sampler 	Bertin Instruments Parc d'activités du Pas du Lac 10 bis, avenue Ampère 78 180 Montigny-le-Bretonneux FRANCE	Airborne particles concentration in a liquid. Detects viruses, bacteria, molds, pollens, spores. Compatible with culture and molecular biology standard methods	Wet cyclonic technology, combined to a high air flow rate. Then samples collected will be subjected to laboratory assays and PCR analysis.	**Dimensions**: 22 × 33 × 36 cm **Weight**: 2,8 kg (with battery) **Air flow rate**: 100 to 300 L/min **Sampling time**: 1-10 min / up to 6 h **Liquid output volume**: 15mL **Collected particle sizes**: > 0.5 µm **Collection efficiency**: D50 <0.5µm **Autonomy on battery**: 1h (collection time)

[a] Although these equipment are factory calibrated, however calibration tests were conducted in the lab and at ambient air conditions, by placing every two identical equipment next to each other and recording the measured or recorded concentrations of pollutants. Then calibration curves were generated for identical equipment and an average error < 5 % was obtained. [b] As for the Langan L76x analyzers, point calibration tests were conducted where a known concentration of CO (40 and 50ppm) was measured by Langan L76x and an average error < 2 % was obtained. In addition, Field- test data collected using the TSI VELOCICALC® Air Velocity Meter Model 9535 were cross-compared and calibrated with values calculated by the ACCUBALANCE® Model 8380 2 Air Capture Hood and a high correlation value (r) of 0.997 was obtained.

Appendix E. Guidelines and standards of air quality indicators

Indicator	Average time	WHO standard	NAAQS / EPA standard	References
CO	8 hour	10 ppm	9 ppm	[59,61,103,111,265,276]
	1 hour	25 ppm	35 ppm	
	30 minutes	50 ppm	NR [d]	
CO_2	15 minutes	15,000 ppm [a]	30,000 ppm	[59,61,103,111,265,276]
	NR [d]	300–600 ppm[b] 1,000 ppm[c]	300–600 ppm 1,000 ppm	
	8 hour	5,000 ppm	5,000 ppm	
NO/NO_2	Annual	40µg/m^3 (0.02 ppm)	100 µg/m^3 (0.05 ppm)	[59,61,103,111,127,265,276]
	1 hour	200 µg/m^3 (0.1 ppm)	188 µg/m^3 (0.09 ppm)	
SO_2	Annual	35 µg/m^3 (0.012 ppm)	80 µg/m^3 (0.03 ppm)	[59,61,103,111,126,127,265,276]
	1 hour	0.133 ppm	196 µg/m^3 (0.07 ppm)	
	24 hour	350 µg/m^3	365 µg/m^3 (0.14 ppm)	
$PM_{2.5}$	Annual	10 µg/m^3	15 µg/m^3	[59,61,111,126,127,265,277]
	24 hour	25 µg/m^3	35 µg/m^3	
PM_{10}	Annual	20 µg/m^3	50 µg/m^3	[59,61,111,126,127,265,277]
	24 hour	50 µg/m^3	150 µg/m^3	
TVOC	NR [d]	300 µg/m^3 [g] (3 ppm)	200–300 µg/m^3 (3 ppm)	[59,61,111,126,127,265,277]

[a] Short-term exposure is an acceptable exposure over 15 minutes.
[b] For outdoors CO_2 standard limits should not exceed 700 ppm [59,265].
[c] For indoors CO_2 the standard limit is set at 1000 ppm [59,265].
[d] Not reported.
[e] Short-term exposure of 10 minutes to a high level of pollutant.
[f] Short- term exposure of 15 minutes to a high level of pollutant.
[g] The European Community target guideline of 300 µg/m^3, where no individual VOC should exceed 10% of the TVOC concentration. Guideline for building standard for State of Washington is 500 µg/m^3 [103].

Appendix F. CFD Governing Equations

Process	*Equation*	*Definition of variables*
Material Balance	$\frac{VdCi}{dt} = QC_0 + S - QC_i - kC_iV$ $\frac{dCi}{dt} + \left(\frac{Q}{V} + k\right)C_i = \frac{Q}{V}C_0 + \frac{S}{V}$ $C_i = \left\{C_0 - \tau\left(\frac{Q}{V}C_0 + \frac{S}{V}\right)\right\}exp^{\frac{-t}{\tau}} + \tau\left(\frac{Q}{V}C_0 + \frac{S}{V}\right)$ $C_i = \tau\left(\frac{Q}{V}C_0 + \frac{S}{V}\right)\left(1 - e^{\frac{-t}{\tau}}\right) + C_0\, e^{\frac{-t}{\tau}}$ $C_i = C_{i\,ss}\left(1 - e^{\frac{-t}{\tau}}\right) + C_0\, e^{\frac{-t}{\tau}}$ $C_{i\,ss} = \frac{\left(\frac{Q}{V}C_0 + \frac{S}{V}\right)}{\frac{Q}{V} + k}$	*V is the volume of the room (m^3)* *C_i is the indoor concentration of the air pollutant ($\mu g/m^3$)* *C_0 is the outdoor concentration of the air pollutant ($\mu g/m^3$)* *Q is the ventilation rate (m^3/hr)* *S is the source emission rate inside the room ($\mu g/hr$)* *k is the removal reaction rate constant (assumed to be first order, hr^{-1})* *τ is given by $(Q/V + k)^{-1}$ is the characteristic time or time constant of the system where C_0 here is the initial concentration in the sampled room at $t = 0$ and $C_{i\,ss}$ is the steady-state solution and $A = Q/V$ which is the air exchange rate (hr^{-1})*
Continuity	$\frac{\partial(\rho)}{\partial t} + \nabla.(\rho v) = 0$	*ρ is the density of the fluid (blood), [kg/m^3]* *v is the velocity of the fluid, [m/s]* t is the time, [s]
Momentum	$\frac{\partial(\rho \boldsymbol{v})}{\partial t} + \nabla.(\rho \boldsymbol{v}.\boldsymbol{v}) = \nabla.(\bar{\bar{\tau}}) + -\nabla p + \rho\vec{g} + \vec{\boldsymbol{B}}$ $\bar{\bar{\tau}} = \mu\left((\nabla.\boldsymbol{v} + \nabla.\boldsymbol{v}^{\boldsymbol{T}}) - \frac{2}{3}\nabla.\boldsymbol{v}\boldsymbol{I}\right)$	*p is the static pressure, [Pa]* *$\rho\vec{g}$ is the gravitational body force, [N/m^3]* *$\vec{B}$ represent the external body forces acting on the fluid, [N/m^3]* *$\bar{\bar{\tau}}$ is the stress tensor, [Pa]*
Energy	$\frac{\partial}{\partial t}\left(\sum_k \alpha_k \rho_k E_k\right) + \nabla.\left[\sum_k \alpha_k \boldsymbol{u}_k(\rho_k E_k + p)\right] = \nabla.\left[k_{eff}\nabla T\right] + S_h$	*k_{eff} is the effective conductivity [W/m K]* *k is the conductivity of the defined fluid, [W/m K]* *k_t is the turbulent thermal conductivity defined according to the*

Process	*Equation*	*Definition of variables*
	$k_{eff} = \sum \alpha_k (k + k_t)$ $E_k = h_k - \frac{p}{\rho_k} + \frac{{v_k}^2}{2}$ *The sensible enthalpy for ideal gases is defined as* h $h = \sum_j Y_j h_j$ *For incompressible flows the expression of the sensible enthalpy is given by:* $h = \sum_j Y_j h_j + \frac{p}{\rho}$ $h_j = \int_{T_{ref}}^{T} c_{p,j}\, dT$	*turbulence model implemented in the setup.* S_h *volumetric heat source* E *is the total energy of the fluid* *The sensible enthalpy for ideal gases is defined as* h $T_{ref} = 298.15\ K$ (Y_j) *is the mass fraction of species j* h_j *is the enthalpy of species j*
Turbulence	*The transport equations for* k *and* ε *in the realizable* $k - \varepsilon$ *turbulence model are given by* $\frac{\partial}{\partial t}(\rho k) + \frac{\partial}{\partial x_j}(\rho k u_j) = \frac{\partial}{\partial x_j}\left[\left(\mu + \frac{\mu_t}{\sigma_k}\right)\frac{\partial k}{\partial x_j}\right] + G_k + G_b - \rho\varepsilon - Y_M + S_k$ $\frac{\partial}{\partial t}(\rho\varepsilon) + \frac{\partial}{\partial x_j}(\rho\varepsilon u_j) = \frac{\partial}{\partial x_j}\left[\left(\mu + \frac{\mu_t}{\sigma_\varepsilon}\right)\frac{\partial \varepsilon}{\partial x_j}\right] + \rho C_1 S_\varepsilon - \rho C_2 \frac{\varepsilon^2}{k + \sqrt{\nu\varepsilon}} + C_{1\varepsilon}\frac{\varepsilon}{k} C_{3\varepsilon} G_b + S_\varepsilon$ $C_1 = max\left[0.43, \frac{\eta}{\eta + 5}\right], \quad \eta = S\frac{k}{\varepsilon}, \quad S = \sqrt{2S_{ij}S_{ij}}$ $G_k = h\rho\overline{\acute{u}_i \acute{u}_j}\frac{\partial u_j}{\partial x_i}$ $G_b = \beta g_i \frac{\mu_t}{Pr_t}\frac{\partial T}{\partial x_i}$ $\beta = -\frac{1}{\rho}\left(\frac{\partial \rho}{\partial T}\right)_p$ $C_{3\varepsilon} = tanh\left\lvert\frac{v}{u}\right\rvert$ $Y_M = 2\rho\varepsilon M_t^2$	G_k *is the generation of turbulence kinetic energy due to mean velocity gradients* G_b *is the generation of turbulence kinetic energy due to buoyancy* g_i *is the component of the gravitational vector in the* $i - th$ *direction, [m/s²]* Pr_t *is the turbulent Prandtl number for energy given by the default value* $(Pr_t = 0.85)$ β *is the coefficient of thermal expansion* v *is the velocity component that is parallel to the gravitational vector and* u *is the velocity component perpendicular to the gravitational vector.* Y_M *represents the contribution of the fluctuating dilatation in compressible turbulence to the overall dissipation rate computed* M_t *is the turbulent Mach number* a *is the speed of sound* C_2 *and* $C_{1\varepsilon}$ *are model constants.* σ_k *and* σ_ε *are the turbulent Prandtl numbers for* k *and* ε *respectively.* S_k *and* S_ε *are user-defined source terms*

Process	*Equation*	*Definition of variables*
	$M_t = \sqrt{\frac{k}{a^2}}$ $a = \sqrt{\gamma RT}$ $C_\mu = \frac{1}{A_0 + A_s \frac{kU^*}{\varepsilon}}$ $U^* = \sqrt{S_{ij}S_{ij} + \tilde{\Omega}_{ij}\tilde{\Omega}_{ij}}$ $\tilde{\Omega}_{ij} = \Omega_{ij} - 2\varepsilon_{ijk}\omega_k$ $\Omega_{ij} = \bar{\Omega}_{ij} - \varepsilon_{ijk}\omega_k$ $A_0 = 4.04, \quad A_s = \sqrt{6}cos\emptyset$ $\emptyset = \frac{1}{3}cos^{-1}(\sqrt{6}W), \; W = \frac{S_{ij}S_{jk}S_{ki}}{\tilde{S}^3}, \; \tilde{S} = \sqrt{S_{ij}S_{ij}}, \; S_{ij} = \frac{1}{2}\left(\frac{\partial u_j}{\partial x_i} + \frac{\partial u_i}{\partial x_j}\right)$ $C_{1\varepsilon} = 1.44, \quad C_2 = 1.9, \quad \sigma_k = 1.0, \quad \sigma_\varepsilon = 1.2$	$\bar{\Omega}_{ij}$ *is the mean rate of rotation tensor viewed in a rotating reference frame with the angular velocity* ω_k. A_0 *and* A_s *are the model constants* *The model constants including* $C_{1\varepsilon}$, C_2, σ_k *and* σ_ε *are set to ensure that the model predicts well certain canonical flows*
Species Transport Model	*The mass balance of specie (k) in a phase is given by the following mathematical model* $\frac{\partial \rho_{(k)} Y_{(k)}}{\partial t} + \nabla.\left(\rho_{(k)} u_{(k)} Y_{(k)}\right) = -\nabla.J_{(k)} + S_Y$	$Y_{(k)}$ *Is the mass fraction of specie (k)* $J_{(k)}$ *is the diffusion flux due to temperature and concentration gradients* S_Y *is the rate of species transport from phase to phase*

Reference: [278]

Appendix G. Summary of existing air quality indices

Air Quality Indices	Mathematical Equations	Description	References
Air quality index (AQI)	$AQI = \left\lfloor \frac{1}{n} \sum_{i}^{n} \frac{C_i}{C_s} \right\rfloor \times 100$	AQI is the average of the sum of the ratios of major pollutant concentrations C_i (in this case PM_{10}, $PM_{2.5}$, CO, CO_2 and TVOC) to their respective air quality standards C_s. The average is then multiplied by 100 to obtain the corresponding index.	[97]
Comprehensive indoor air quality index (CIAI)	$I_p = \frac{I_{HI} - I_{LO}}{BP_{HI} - BP_{LO}} \times (Cp - BP_{LO}) + I_{LO}$	I_P is the CIAI score of each air pollutant. C_P is the concentration of air pollutant. BP_{HI} and BP_{LO} are the upper and lower concentration bounds for a range of air pollutant. I_{HI} and I_{LO} are the mean indices corresponding to BP_{HI} and BP_{LO} which are the maximum and minimum indices of the range. After calculating CIAI scores for each air contaminant the highest score among them will be used as the integrated index value. If there are more than two indices with "unhealthy for sensitive groups", the index with higher value will receive more weightage	[81]
Maximum cumulative ratio (MCR)	$HQ_i = \frac{C_i}{RV_i}$ $HI = \sum_{i} HQ_i$ $MCR = \frac{HI}{\max HQi}$	*MCR* can be calculated using the hazard quotients *HQs* for each substance present in the mixture and the hazard index *HI* of the mixture. *Ci* is the concentration of the air pollutant to which an individual is exposed. RV_i is the health based reference or standard value of air pollutant *i* (expressed as a concentration). HQ_i is the hazard quotient of the individual's exposure to the air pollutant. *MCR* of the individual's exposure to the mixture is the ratio of hazard index *HI* of the mixture to the maximum of the hazard quotients of the individual components (*max* HQ_i).	[80,99,100,102]

Appendix H. AQI levels of health concern

Health Concern	Score	Description
Good	0 to 50	Air quality is considered satisfactory, and air pollution poses little or no risk
Moderate	51 to 100	Air quality is acceptable; however, for some pollutants there may be a moderate health concern for a very small number of people who are unusually sensitive to air pollution.
Unhealthy for sensitive groups	101 to 150	Members of sensitive groups such as people suffering from respiratory and heart diseases may experience health effects. The general public is not likely to be affected.
Unhealthy	151 to 200	Everyone may begin to experience health effects; members of sensitive groups may experience more serious health effects.
Very unhealthy	201 to 300	Health warnings of emergency conditions. The entire population is more likely to be affected.
Hazardous	301 to 500	Health alert: everyone may experience more serious health effects

***Reference*:** [97]

Appendix I. Comprehensive indoor air quality index (CIAI) classification for different air pollutants

Index	**A**		**B**		**C**		**D**		**E**		**F**	
Level	**Good**		**Moderate**		**Unhealthy for sensitive groups**		**Unhealthy**		**Very unhealthy**		**Hazardous**	
I_{LO}	0		51		101		151		201		301	
I_{HI}	50		100		150		200		300		500	
Conc. level	BP_{LO}	BP_{HI}	BP_{LO}	BP_{HI}	BP_{LO}	BP_{HI}	BP_{LO}	BP_{HI}	BP_{LO}	BP_{HI}	BP_{LO}	BP_{HI}
CO (ppm)	0	5	5.01	10	10.01	20	20.01	30	30.01	40	40.01	50
CO_2 (ppm)	0	500	501	1000	1001	1500	1501	2000	2001	3000	3001	5000
TVOC (ppm) [1]	0	1.0	1.06	3	3.1	4.5	4.6	6	6.1	8.86	> 8.86	
$PM_{2.5}$ (μg/m^3)	0	15	16	40	41	140	141	250	251	350	351	500
PM_{10} (μg/m^3)	0	30	31	80	81	120	121	200	201	300	301	600

Note: I_{LO} is the index breakpoint corresponding to BP_{LO}, I_{HI} is the index breakpoint corresponding to BP_{LO}, and BP_{LO} and BP_{HI} are the concentration breakpoints of each health level.

[1] There are no clear regulations for TVOC, however these pollutants are important to monitor as they can lodge deep inside lungs, cause irritation and discomfort, and increase neurotoxic effects [19,166]

References: [81,97]

Appendix J. Group classification of mixtures according to MCR and HI in the CEFIC-MIAT decision tree

Group	**Boundaries on MCR, HI and max HQ**	**Description**
Group I	*max HQ* > 1 (*HI* > MCR)	*Single substance concern*: mixtures containing at least one substance in a concentration that poses a health risk; the risk would have been identified also in a substance-by-substance assessment.
Group II	*HI* < 1	*Low concern*: mixtures of low concern with regard to individual substances and their combined effects.
Group IIIA	MCR < 2, *HI* > 1 and *max HQ* < 1	*Concern for combined effect dominated by one substance*: mixtures with low concern for the individual substances, but with concern for combined effects where one substance is responsible for most of the mixture's toxicity; further cumulative risk assessment is required; a substance-by-substance assessment would not have identified this mixture as of concern, since max HQi < 1.
Group IIIB	MCR > 2, *HI* >1 and *max HQ* < 1	*Concern for combined effect by several substances*: mixtures with low concern for the individual substances, but with concern for combined effects where several substances are responsible for the mixture's toxicity; further cumulative risk assessment is required; a substance-by-substance assessment would not have identified this mixture as of concern, since max HQi < 1.

References: [80,99,100,102]

Appendix K. Summary of mortality risk estimates associated with PM_{10} and $PM_{2.5}$ exposure

PM	Type	Health impact	Estimate (95 % confidence Interval)	References
PM_{10}	Daily	All cause	0.6 % (0.4–0.8 %) per 10 µg/m^3	[106,109,111]
	Daily	Respiratory	1.3 % (0.5–2.09 %) per 10 µg/m^3	[106,109,111]
	Daily	Cardiovascular	0.9 % (0.5–1.3 %) per 10 µg/m^3	[106,109,111]
	Daily	All cause	0.21 % (0.09–0.33 %) per 10 µg/m^3	HEI NMMAPS [a] [106,109,111]
	Daily	Cardiovascular	0.31 % (0.13–0.49 %) per 10 µg/m^3	HEI NMMAPS [a] [106,109,111]
$PM_{2.5}$	Long term	All cause	4 % (1–8 %) per 10 µg/m^3	ACS CPS II [b] [105,108,111,148,149]
	Long term	Cardiopulmonary	6 % (2–10 %) per 10 µg/m^3	ACS CPS II [b] [105,108,111,148,149]

[a] NMMAPS = National Morbidity, Mortality and Air Pollution Study.
[b] American Cancer Society Cancer Prevention Study II.

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